

# Special Issue

## Metabolic Diseases Regulators

### Message from the Guest Editor

Metabolic syndrome is a group of metabolic abnormalities centered around excessive visceral adiposity that involves diseases in target organs, mainly cardiovascular system, skeletal muscle, pancreas and liver. Diseases associated with metabolic syndrome are associated with a high level of morbidity and mortality. Treating organ diseases that are induced by metabolic syndrome is cumbersome. Therefore, identifying common pathogenic mechanisms and specific factors is the focus of multidisciplinary approaches, the aim of which is to translate this knowledge into prevention and therapy. This Special Issue is focused on unfolding the role of novel regulators that are involved in the development of the metabolic syndrome and related organ diseases. The scope is to reveal potential therapeutic targets that are common for metabolic diseases.

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### Guest Editor

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### Deadline for manuscript submissions

closed (30 November 2024)



# Biomedicines

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### Message from the Editor-in-Chief

*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.

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### Editor-in-Chief

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