Special Issue

Molecular Mechanisms at the Basis of Systemic Complications of Glycogen Metabolism Disorder

Message from the Guest Editor

This Special Issue is focused on molecular mechanisms involved in the pathogenesis of systemic complications of glycogen metabolism disorder. Glycogen storage disease type I (GSDI) is the most frequent form of glycogen metabolism disorder. GSDI is an inborn error of carbohydrate metabolism caused by mutations of either the G6PC gene (GSDIa) or the SLC37A4 gene (GSDIb). Glucose 6-phosphate (G6P) accumulates in endoplasmic reticulum and cytosol in GSD1a and GSD1b, respectively. In GSDIa, the G6P excess in the liver ER has been associated with hyperlipidemia and therefore mitochondrial disfunction and increasing 11□HSD1 activity. Interestingly, recent studies demonstrated that 11□HSD1 activity is increased in GSDIa and reduced in GSDIb. If hyperlipidemia and increasing 11□HSD1 activity may contribute to the development of insulin resistance and metabolic syndrome (MS) in GSD1a, GSD1b patients appear to be protected. This Special Issue is going to focus on molecular mechanisms involved in mitochondrial dysfunction, de novo lipogenesis and cortisol production to increase the scientific community's interest in this disease.

Guest Editor

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The International Journal of Molecular Sciences (*IJMS*, ISSN 1422-0067) is an open access journal, which was established in 2000. The journal aims to provide a forum for scholarly research on a range of topics, including biochemistry, molecular and cell biology, molecular biophysics, molecular medicine, and all aspects of molecular research in chemistry. *IJMS* publishes both original research and review articles, and regularly publishes special issues to highlight advances at the cutting edge of research. We invite you to read recent articles published in *IJMS* and consider publishing your next paper with us.

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