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

Guest Editor:

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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 hyperlipidemia and therefore mitochondrial disfunction and increasing 11βHSD1 activity. Interestingly, recent studies demonstrated that 11βHSD1 activity is increased in GSDIa and reduced in GSDIb. If hyperlipidemia and increasing 11β 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.













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Editor-in-Chief

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Message from the Editor-in-Chief

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