

Endocrinology, Diabetes and Metabolism

March 16, 2022 | Webinar

Magnesium sulfate improves insulin resistance in type 2 diabetic parents and their offspring in both animal model and diabetic patients

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The role of magnesium sulfate ($MgSO_4$) in attenuates insulin resistance (IR) in type 2 diabetic (T2D) patients and the reduction of the risk of IR in liver and muscle of their offspring. T2D was induced by high fat diet and 35 mg/kg of streptozotocin. The male and female diabetic rats were then divided into three groups: CD, Mg, and insulin. NDC group received a normal diet. All the animals were studied for a six-month. Their offspring were just fed with normal diet for four months. Blood glucose was measured weekly in patients and their offspring. Intraperitoneal glucose tolerance test (IPGTT), urine volume, and water consumption in both patients and their offspring were performed monthly. The hyperinsulinemic euglycemic clamp in both patients and their offspring was done and blood sample collected to measure hemoglobin A1c (HbA1c) and plasma lipid profile. IRS1, Akt and GLUT4 gene expressions in muscle were evaluated in all the groups. FOXO1 and phosphoenolpyruvate carboxykinase gene expressions also measured in all groups. Plasma and liver lipid profile and

liver glycogen level were measured in all groups. $MgSO_4$ or insulin therapy decreased blood glucose, IPGTT, plasma lipid level and HbA1c in patients and their offspring compared to DC group. They also increased glucose infusion rate in patients and their offspring. IRS1, Akt and GLUT4 gene expressions improved in both patients in comparison with DC group. $MgSO_4$ could decreased gluconeogenesis pathway in both parents and their offspring. $MgSO_4$ exerts beneficial effects on IRS1 and Akt gene expressions in Mg treated offspring. $MgSO_4$ therapy improved insulin resistance in diabetic patients by increasing the expression of GLUT4 in the muscle and reduce gluconeogenesis pathway in the liver it also could improve plasma lipid profile in both parents and their offspring. $MgSO_4$ is also indirectly able to reduce insulin resistance in their offspring possibly through the increased gene expressions of IRS1 and Akt and gluconeogenesis pathway in the liver.

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