Short Communication
Volume 10, Issue 2, 2021

Journal of Metabolic Syndrome

Open Access

3rd International Conference and Exhibition on Obesity & Weight Management

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Abstract

Metabolic Syndrome, a pathological condition affecting approximately one-third of the USA population, is characterized by obesity, hepatic steatosis, insulin resistance, and hypertension among other medical conditions, and represents the single most common condition predisposing to diabetes and hypertension. Within this contest, liver steatosis is considered the hepatic representation of the disease and is currently classified as NAFLD. Hypomagnesaemia has been consistently observed in association with metabolic syndrome, and recent data from our laboratory suggest low Mg2+ level in the circulation and within the hepatocytes promotes dysmetabolic conditions typical of the metabolic syndrome. Rats exposed for 2 weeks to a 40% Mg2+ deficient diet present a 2-3 fold increase in intrahepatic triglyceride content associated with a marked increase in numerous genes involved in fatty acid synthesis including IRS2, SREBP1c, PPARγ, FAS, LCAD, HMG-CoA reductase and HMG-CoA synthethase. PPAR-α, instead remained unaltered The upregulation of IRS2 contrasted the downregulation of IRS- 1 and pat, leading to decreased glucose accumulation, and attenuated insulin signaling. As a result of this altered signaling Fox-01 remained active and resulted in the upregulation of the gluconeogenic genes PEPCK and F, 16 BPase. Additionally, the reticular hexose 6-phosphate dehydrogenase (H6PD) and the coupled 11β-hydroxysteroid dehydrogenase-1 (11-β-HSD1) were both 3-4 fold upregulated in gene and protein expression, resulting in the significant production of intraluminal NADPH and cortisol levels. Taken together, our results provide
compelling evidence that reduced extracellular Mg2+ level precedes and promotes metabolic syndrome onset in that: 1) liver metabolism undergoes a switch from glucose-based to fatty acidbased synthesis with minimal changes in catabolism, resulting in the increased deposition of intrahepatic triglycerides; 2) increased cortisol production, and 3) reduced insulin responsiveness.

This work is partly presented at 3rd International Conference and Exhibition on Obesity & Weight Management

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