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Metabolic Syndrome in Basic Research: How to Study It

Editorial

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The number of diabetic patients is increasing worldwide. It is estimated that between 2010–2030, the number of adult diabetic patients will increase by 69% and 20% in developing and developed countries, respectively [1]. Additionally, one-third of adults have metabolic syndrome in United States [2], defined as a syndrome that includes insulin resistance, hypertension, abdominal obesity, hypertriglyceridemia, and low HDLcholesterol, and is caused by interactions between genetic and environment components. The components of metabolic syndrome may lead to other pathologies. For example, hyperglycemia and hypertension are the main causes of chronic kidney disease. It is a challenge in the field of basic research where variables are often isolated for better analysis. Researchers have attempted to develop a model that includes all the aspects of metabolic syndrome. One of them is a high-fructose intake (20-60%) diet that mimics most of the associated pathologies observed in this syndrome, such as elevated blood pressure, hypertriglyceridemia, insulin resistance, and hyperuricemia. [3, 4]. In the United States (1909-1997), a correlation between the prevalence of diabetes and corn syrup but not protein and fat intake [5] was observed. High-fructose corn syrups contain 55–90% fructose, which is commonly found in soft drinks, beverages, and breakfast cereals.

The mechanism proposed for the correlation between fructose intake and metabolic syndrome signs may involve uric acid synthesis and hepatic fat accumulation. The fructose in the diet is absorbed in the intestine by the Glut5 transporter and is distributed mainly to the liver, kidney, and adipose tissue, but it does not stimulate insulin release. Fructose is phosphorylated by fructokinase into fructose 1-phosphate. The latter is then metabolized by aldolase B and other enzymes, generating glyceraldehyde and dihydroxyacetone, glycerol-phosphate, and then triglycerides in the hepatocytes [6]. Additionally, fructose phosphorylation causes a decrease in intracellular phosphate and ATP depletion. Adenosine mono-phosphate is generated and broken down by adenosine mono-phosphate deaminase, resulting in the generation of inosine mono-phosphate and eventually uric acid [7].

A correlation between nonalcoholic fat liver disease (NAFLD) and the prediction of cardiovascular and renal events has been reported in a study that suggested that NAFLD is an early predictor of cardiovascular and kidney diseases [8]. This abnormality was also present in a fructose-induced metabolic syndrome model. A 10% glucose intake for 14 weeks induced endogenous generation of fructose and liver fat accumulation induced by glucose [9].

Uric acid production induced by fructose intake was correlated with hypertension, which was prevented by allopurinol treatment [10]. Additionally, fructose intake and uric acid production are also correlated with kidney disease. A high fructose intake in rats induced kidney hypertrophy and tubular proliferation; furthermore, in a remnant kidney model, fructose intake exacerbated proteinuria, decreased kidney function, and accelerated glomerulosclerosis [11].

Although the high fructose intake experimental model seems to mimic most of the signs of metabolic syndrome, some questions remain to be answered. The high incidence of metabolic syndrome in countries lacking high-fructose diets such as Germany, Poland, Greece, Portugal, and Egypt is unclear. Furthermore, the importance of a high-lipid diet in this experimental model should be addressed.

An experimental model wherein two or three variables are concomitantly observed (e.g. hypertension and high-fructose diet, high-fat and high-fructose diet, or hyperglycemia and high-fructose diet) may help analyze the contribution of each component to this syndrome.

References

[1]. J. E. Shaw, R. A. Sicree, and P. Z. Zimmet, "Global estimates of the prevalence of diabetes for 2010 and 2030.," *Diabetes Res. Clin. Pract.*, vol. 87, no. 1, pp. 4–14, 2010.
[2]. E. S. Ford, "Prevalence of the metabolic syndrome defined by the International Diabetes Federation among adults in the U.S.," *Diabetes Care*, vol. 28, no. 11, pp. 2745–2749, 2005.
[3]. R. J. Johnson, T. Nakagawa, L. G. Sanchez-Lozada, M. Shafiu, S. Sundaram, M. Le, T. Ishimoto, Y. Y. Sautin, and M. a Lanaspa, "Sugar, uric Acid, and the etiology of diabetes and obesity." *Diabetes*, vol. 62, no. 10, pp. 3307–15, 2013.
[4]. R. J. Johnson, P. Stenvinkel, S. L. Martin, A. Jani, L. G. Sánchez-Lozada, J.

O. Hill, and M. A. Lanaspa, "Redefining metabolic syndrome as a fat storage condition based on studies of comparative physiology.," *Obesity (Silver Spring).*, vol. 21, no. 4, pp. 659–64, 2013.

[5]. L. S. Gross, L. Li, E. S. Ford, and S. Liu, "Increased consumption of refined carbohydrates and the epidemic of type 2 diabetes in the United States: an ecologic assessment." *Am. J. Clin. Nutr.*, vol. 79, no. 5, pp. 774–779, 2004.

[6]. H. Basciano, L. Federico, and K. Adeli, "Fructose, insulin resistance, and metabolic dyslipidemia," *Nutr. Metab. (Lond).*, vol. 2, p. 5, 2005.

[7]. L. G. Sánchez-Lozada, M. a Lanaspa, M. Cristóbal-García, F. García-Arroyo, V. Soto, D. Cruz-Robles, T. Nakagawa, M. a Yu, D.-H. Kang, and R. J. Johnson, "Uric Acid-Induced Endothelial Dysfunction Is Associated with Mitochondrial Alterations and Decreased Intracellular ATP Concentrations.," *Nephron. Exp. Nephrol.*, vol. 121, no. 3–4, pp. e71–e78, 2012.

[8]. A. A. El Azeem HA, Khalek el-SA, El-Akabawy H, Naeim H, Khalik HA, "Association between nonalcoholic fatty liver disease and the incidence of cardiovascular and renal events," *J Saudi Hear. Assoc, vol.* 25, no. 4, pp. 239–246, 2013.

[9]. M. A. Lanaspa, L. G. Sanchez-Lozada, C. Cicerchi, N. Li, C. A. Roncal-Jimenez, T. Ishimoto, M. Le, G. E. Garcia, J. B. Thomas, C. J. Rivard, A. Andres-Hernando, B. Hunter, G. Schreiner, B. Rodriguez-Iturbe, Y. Y. Sautin, and R. J. Johnson, "Uric Acid Stimulates Fructokinase and Accelerates Fructose Metabolism in the Development of Fatty Liver," *PLoS ONE, vol.* 7, no. 10. p. e47948, 2012.

[10]. S. E. Perez-Pozo, J. Schold, T. Nakagawa, L. G. Sánchez-Lozada, R. J. Johnson, and J. L. Lillo, "Excessive fructose intake induces the features of metabolic syndrome in healthy adult men: role of uric acid in the hypertensive response.," *Int. J. Obes. (Lond).*, vol. 34, no. 3, pp. 454–461, 2010.

[11]. M. S. Gersch, W. Mu, P. Cirillo, S. Reungjui, L. Zhang, C. Roncal, Y. Y. Sautin, R. J. Johnson, and T. Nakagawa, "Fructose, but not dextrose, accelerates the progression of chronic kidney disease.," *Am. J. Physiol. Renal Physiol.*, vol. 293, no. 4, pp. F1256–F1261, 2007