FRUCTOSE-RICH DIET INDUCES LIVER DAMAGE IN MURINE MODEL

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INTRODUCTION: The nonalcoholic fatty liver disease (NAFLD) is characterized by the accumulation of triacylglycerols (TG) in hepatocytes (steatosis) and it is called as the first hit, the most prominent alteration in NAFLD. The consumption of fructose has expanded worldwide, in addition to nonalcoholic fatty liver disease (NAFLD), in parallel with obesity and high fructose diet consumption.

OBJECTIVES: To evaluate the effects of fructose-rich diet on liver enzymes and lipid profile in the serum, liver and feces.

METHODS: 20 male Fischer rats were separated into 2 groups: C (control) was fed with AIN-93M diet and F (fructose) was fed with fructose-rich diet (60%) during 18 weeks. The extraction of liver and fecal lipids was described by FOLCH et al. (1957). The biochemical assays were done by commercial kits. Histological hepatic tissue analysis used hematoxylin and eosin (H&E) method. Data were analyzed by Student t-test. Differences were considered significant when p < 0.05.

RESULTS: The animals showed no difference in weight gain (C:171,3±30,13; F:153,5±19,46), food intake (g/day) (C:19,81±1,252; F:19,50±0,7926) and fecal excretion (g/week) (C:5,240±0,6443; F:5,099±0,8441). The F group showed increased liver weight, triglycerides and glucose levels, as well as alkaline phosphatase activity. In the liver, the triglycerides levels were higher in the fructose group. The fecal fat percentage of fructose group was increased, and also the triglyceride levels. The hepatic changes were examined on hepatic tissue and showed fructose-rich-diet-induced severe steatosis with microvesicular and macrovesicular steatosis. The fructose group showed greater field of fat droplets and more droplets when analyzed cell / field. Thus, fructose group have a reduced number of nuclei.

CONCLUSION: The alterations suggest that the fructose-rich diet induces NAFLD, confirmed by histological damage. In addition, fructose-rich diet increased triglycerides excretion in feces. Therefore, this model can be useful for future studies on the mechanisms related to excessive intake of fructose.

KEYWORD: fructose-rich diet, hepatic tissue damage, nonalcoholic fatty liver disease.

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