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ORIGINAL ARTICLE

EVALUATION OF THE EFFECTS OF N-ACETYLCYSTEINE ON SERUM GLUCOSE, LIPID PROFILE, AND BODY WEIGHT IN RATS WITH FRUCTOSE-INDUCED METABOLIC SYNDROME

Abstract

Background: Overconsumption of fructose may cause metabolic syndrome (MetS). MetS pathogenesis is caused by oxidative stress, cellular malfunction, and systemic inflammation caused by hereditary and environmental factors. N-acetylcysteine (NAC) has become associated with the phrase "antioxidant." Most researchers use and test NAC with the goal of preventing or reducing oxidative stress.

Aim: To determine the positive effects of NAC on blood glucose, lipid profile, and body weight in fructose-induced metabolic syndrome in albino rats.

Materials and Methods: Forty male albino rats, 10-12 weeks old, were haphazardly divided into five groups of identical size. Group I (negative control) received tap water for 12 weeks. Group II (positive control) received a 60% w/w fructose solution (60% FS) instead of tap water for 12 weeks. Group III (NAC) received tap water and an intra-peritoneal (IP) injection of NAC (150 mg/kg/day) for 12 weeks. Group IV (protection) co-administered 60% FS orally and NAC IP injection (150 mg/kg/day) for 12 weeks. Group V (treatment) received 60% FS for 8 weeks followed by 4 weeks of drinking tap water with NAC IP injection (150 mg/kg/day). Blood samples were taken at weeks 0, 8, and 12 and were tested for serum glucose and lipid profile. All animals of each group were weighted at weeks 0, 8 and 12 of the study.

Results: Concerning serum glucose, group II showed increased glycaemia at week 8 and further elevation during week 12. Group III displayed normal glycaemia at weeks 8 and 12. In group IV, glycaemia showed elevation at week 8 followed by almost complete restoration at week 12. In group V, there was an increased glycaemia at week 8 followed by a partial restoration at week 12. Regarding lipid profile parameters, group II demonstrated a deterioration during week 8 and more worsening during week 12. There were no significant changes in group III's parameters during weeks 8 and 12. Group IV displayed a worsening in lipid profile during week 8 followed by a nearly complete improvement during week 12. During week 8, group V deteriorated, followed by a partial recovery during week 12. Concerning body weight, group II showed a weight gain at week 8 and further elevation during week 12. Group III displayed normal glycaemia at weeks 8 and 12. In group IV, glycaemia showed elevation at week 8 followed by almost complete restoration at week 12.