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COMBINED S-ADENOSYLMETHIONINE AND GLUTATHIONE TREATMENT ATTENUATES PANCREATIC STEATOSIS IN A DIET- AND STRESS-INDUCED RAT MODEL OF METABOLIC DYSFUNCTION

Background. Pancreatic steatosis reflects metabolic dysfunction associated with insulin resistance, inflammation and remodeling. **Aim.** The aim was to determine whether S-adenosylmethionine/glutathione corrects pancreatic morphology and metabolic disturbances following experimental steatosis. **Methods.** Male white rats were allocated into three groups of 8 animals each: control, 60-day model and correction groups. The model combined a high-fat diet, 10% fructose solution and chronic spatial restriction. After modeling, the correction group received S-adenosylmethionine plus glutathione at 0.2 g/kg/day for 30 days. The pancreatic morphology, carbohydrate/lipid metabolism, transforming growth factor beta 1 (TGF- β 1) and fecal short-chain fatty acids (SCFAs) were assessed. The data are presented as mean $\pm$ SEM. **Results.** In the model, pancreatic steatosis was verified in 8/8 rats (100%) and graded qualitatively as mild/moderate by interlobular and intralobular adipocyte accumulation, acinar vacuolization, inflammation and early fibrosis. Initial acinar atrophy and fibrosis were found in 3/8 animals (37.5%). After correction, minimal steatosis persisted in 7/8 animals (87.5%) and was absent in 1/8 animal (12.5%); fibrosis and inflammatory infiltration were not detected, and acinar-islet architecture was preserved. Glucose decreased from 9.4 ± 0.83 to 6.8 ± 0.52 mmol/L ($p < 0.05$), insulin from 42.8 ± 10.4 to 16.7 ± 1.6 μ U/mL, and homeostatic model assessment of insulin resistance (HOMA-IR) from 18.9 ± 4.9 to 4.95 ± 1.21 ($p < 0.05$ vs model), but remained above control (1.8 ± 0.66 ; $p < 0.05$). TGF- β 1 decreased from 2591.8 ± 136.6 to 2302.8 ± 152.1 pg/mL, but exceeded control (1176.2 ± 257.3 pg/mL; $p < 0.05$). Low-density lipoprotein cholesterol (LDL-C) and the atherogenic coefficient remained increased versus control ($p < 0.05$). Among SCFAs, only butyric acid significantly decreased in the model versus control, ($p < 0.05$). **Conclusions.** S-adenosylmethionine/glutathione promoted morphological regression of pancreatic steatosis, reduced fat infiltration, eliminated histological fibrosis and inflammation, and preserved acinar-islet architecture. Normalization was partial, as residual HOMA-IR and TGF- β 1 elevation indicated persistent metabolic and profibrotic activation. **Funding.** "To study the clinical, biochemical, and structural features of pancreatic steatosis in servicemen of the Armed Forces of Ukraine with metabolic dysfunction-associated steatotic liver disease and to improve its treatment" (No. 0125U000190).

Keywords: pancreatic, steatosis, S-adenosylmethionine, glutathione, HOMA-IR, TGF- β 1, SCFAs.

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