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PROPARGYLGLYCINE AND HYDROGEN SULFIDE EFFECTS ON PLASMA GALECTIN-3 IN STREPTOZOTOCIN-INDUCED DIABETIC RATS

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Background and aims: The majority of diabetic patients are suffering from heart and kidney disease. Galectin-3 belongs to mediators of cardiac and renal fibrogenesis, while hydrogen sulfide (H2S), metabolite of sulfur-containing amino acids, is considering to have antifibrogenic effects. However, interaction between H2S system and galectin-3 associated with diabetes and its complications is undefined.

This study aimed to determine influence of H2S metabolism modulators (NaHS, propargylglycine) on plasma galectin -3 in rats with streptozotocin diabetes.

Methods: 32 healthy male rats (180-250 g) were divided into 4 groups. To induct diabetes, rats (group 2-4) were injected with streptozotocin (STZ, 40 mg/kg,/i.p., 0.1 M citrate buffer (pH 4.5). Rats (group 3-4) were administered NaHS (3 mg/kg/i.p.) or propargylglycine (50 mg/kg/i.p.) once per day starting from 3 to 28 day after streptozotocin injection. Rats of group 1 (control) were administered the equivalent volumes of 0.9% NaCl. Plasma galectin-3 was measured by ELISA. Rats' hearts and kidneys were sempled for determination of H2S by reaction with N,N-Dimethyl-p-phenylenediamine.

Results: Plasma galectin-3 in STZ-diabetic rats (group 2) was significantly higher than in controls (10.7 ± 0.98 ng/ml vs 3.02 ± 2.68 ng/ml, $p < 0.01$). While, H2S in myocardium and kidney of this rats (group 2) was decreased (in 1.37 - 1.48 times, $p < 0.05$). Administration of H2S donor (NaHS) resulted in significant decrease of plasma galectin-3 (1.28 times, $p < 0.05$), but propargylglycine administration (inhibitor of H2S -synthesising enzyme - cystathionine gamma-lyase) caused significant increase (in 1.56 times, $p<0.05$) of plasma galectin-3 (group 3-4). Increased plasma galectin-3 correlated with decreased H2S in myocardium and kidney of STZ-diabetic rats ($r = 0.62; 0.59$, $p < 0.05$).

Conclusions: Plasma galsectin-3 is increased in setting of STZ-diabetes in rats. Inhibition of H2S endogenous production could apparently enhance this effect, when NaHS administration led to reduction in plasma galectin-3. Thus, H2S system can be integrated in pathogenesis of diabetic complications through modulation of proinflammatory and profibrogenic mediator galectin-3.