MECHANISMS OF ATHEROGENIC DYSLIPIDEMIA UNDER THE METABOLIC SYNDROME IN HAMSTERS OF DIFFERENT AGE

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Metabolic syndrome is the complex of hormonal and metabolic disorders that increase the risk of type 2 diabetes mellitus and cardiovascular system diseases. It's known the prevalence of metabolic syndrome in the population increases with age and is highest among the elderly. However mechanisms of atherogenic dyslipidemia and age-related differences in lipid metabolism changes under metabolic syndrome are not fully understood.

The goal: to investigate the mechanisms of atherogenic dyslipidemia under the metabolic syndrome in hamsters of different age.

Material and methods

Experiments were planned to develop a diet-induced MS in Golden Syrian hamsters of different age (4 weeks and 20 weeks at the beginning of the experiment), which were kept in a standard vivarium conditions. Animals were fed a standard normal diet (intact group), and during 5 weeks high-calorie diet that contained 29% of fats (predominantly saturated) with fructose addition – 1 g daily per 100 g body weight (metabolic syndrome groups). Blood samples were taken after decapitation in necessary terms and prepared according to individual procedures.

Experiments were carried out according to the “European Convention for the Protection of Vertebrate Animals used for Experimental and other Scientific Purposes” (Strasbourg, 1985). Statistical analysis were performed using nonparametric van der Waerden criterion.

Triacylglycerol (TAG) content was determined by enzymatic assay (“KONE”, Finland). Free and esterified cholesterol (CE) were determined by the help of enzymatic assays (“Boehringer Mannheim GmbH diagnostica”, Germany). Lipoprotein fractions (very low density lipoproteins (VLDL); low density lipoproteins (LDL) and high density lipoproteins (HDL)) were determined by the help of electroforesis. Total LDL and apoB-containing lipoproteins (apoB-LP) in blood serum were determined by by gradient gel electrophoresis.

Results

Changes in blood hormone levels observed under the MS led to a shift in the lipolysis/lipogenesis balance and were accompanied by the excessive production of the free fatty acid. Indeed, the free fatty acid level was increased by approximately 40% in male experimental animals independently of age (table 1).

Atherogenic dyslipidemia develops independently of age in males fed high-calorie diet (table 1). As it can be seen from the data presented, increased serum total lipids level in animals is mediated by the increase of apoB-LP level because the HDL content did not change. Herewith, serum TAG level rose by 47% and 30% relative to intact groups, in young and adult animals respectively (table 1).

Table 1.

Some lipid metabolism parameters in blood serum of male Syrian hamsters with the experimental metabolic syndrome

Age	Group	Parameter		
		Triacylglycerols, g/L	ApoB-containing lipoproteins, g/L	Free fatty acid,
4 weeks	Intact	1.06±0.07	4.72±0.23	1.02±0.07
	Metabolic syndrome	1.56±0.09*	6.68±0.15*	1.44±0.29*
20 weeks	Intact	1.57±0.22	5.66±0.34	1.64±0.16
	Metabolic syndrome	2.00±0.13*	6.68±0.21*	2.29±0.25*

Each group was composed of six animals. Mean±S.D. * – p□0.05 vs the same age intact group.

Conclusion

The blood TAG content increase under metabolic syndrome is considered as the key factor in the atherogenic dyslipidemia formation. A clear correlation between hypertriacylglycerolemia and ApoB-containing lipoproteins accumulation in the blood plasma demonstrated in numerous experimental and clinical studies.

Based on these data, we can suppose that lipolysis activation and free fatty acids accumulation in the blood leads to morphological changes of lipoproteins that secreted by the liver under the metabolic syndrome development.