МЕДИЦИНСКИ УНИВЕРСИТЕТ - ВАРНА
„Проф. д-р Параскев Стоянов“
Ул.“Марин Дринов“ 55, Варна 9002, България
Тел. : 052/ 65 00 57, Факс: 052/ 65 00 19
e-mail: uni@mu-varna.bg, www.mu-varna.bg

MEDICAL UNIVERSITY - VARNA
“Prof. Dr. Paraskev Stoyanov”
55, Marin Drinov Str., 9002 Varna, Bulgaria
Tel.: +359 52/ 65 00 57, Fax: + 359 52/ 65 00 19
e-mail: uni@mu-varna.bg, www.mu-varna.bg

Fund “Nauka” Project № 11007 Resume – Competition-Based Session 2011:

“A study of the relation between steatosis, nonalcoholic apoptosis and liver damage, induced by high fructose diet”

Project leader: Prof. Ganka Yordanova Bekyarova, MD, PhD, DSc

The aim of this project was to study the relationship between oxidative stress, inflammatory response and apoptosis as key factors in the development non -alcoholic fatty liver induced by high fructose diet (HFD) and the effect of S-adenosylmethionine (SAMe) and allopurinol (All). The studies were performed on male Wistar rats divided into 3 groups: control, fructose fed (35%,16 weeks), fructose fed and treated with SAMe and All. The results showed dislipidemia, hyperglycemia, increased body weight, malondialdehyde (MDA) (lipid peroxidation markers) levels, reduced content of glutathione and antioxidant enzymes such as glutathione peroxidase (GPx) and increased heme-oxygenase-1(HO-1) activity in the liver of HFD group.

The results showed increased level of pro-apototic Bax protein and decreased levels of anti-apoptotic Bcl-2 protein and Beclin1 in liver of HFD group. Increased levels of pro-inflammatory mediators and TNF-α, IL-6, CRP in the liver were established, too. HFD group showed microvesicular steatosis and liver dysfunction. SEM and Allopurinol inhibited the elevation of MDA, TNF-α, IL-6, CRP and Bax levels, prevented the decrease in glutathione, Bcl 2 protein levels and GPx activity and augmented the increase in expression of HO-1.

In conclusion, HFD diet causes obesity, microvesicular steatosis, oxidative stress, inflammation, mitochondrial dysfunction and apoptosis in the liver. Treatment with SAMe and All reduces dyslipidemia, steatosis, inhibit oxidative stress, inflammation and apoptosis in hepatocytes involved in liver damage and progressive development of non-alcoholic fatty liver disease. The presented data are in accordance with the scientific hypothesis of the project for the relationship between steatosis, oxidative stress, inflammation and apoptosis in the pathogenesis of non-alcoholic fatty liver disease (component of metabolic syndrome) and its progression.

Results of this project were presented at:

- 32nd Balkan Medical Week, 21-23 September 2012. Nis, Serbia;
- The International Liver Congress, Amsterdam, The Netherlands, April 24-28, 2013;
- 21st European Congress on Obesity, May 28th - 31st 2014, Sofia, BG;
- 33nd Balkan Medical Week, 8-10 October 2014, Bucharest, Romania;