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CoMBoS2

Content

WELCOME SPEECH

Professor Dušanka **Savić-Pavićević**
President of the Serbian Society
for Molecular Biology

Dr. Melita **Vidaković**
President of the Steering Committee
of the Serbian Society for Molecular Biology

Dear colleagues and friends,

On behalf of the Serbian Society for Molecular Biology (MolBioS), we warmly welcome you to Belgrade for the Second Congress of Molecular Biologists of Serbia (CoMBoS2).

The congress is gathering almost 250 participants from 13 countries (Sweden, United Kingdom, Italy, Switzerland, USA, Australia, Hungary, Czech Republic, Romania, Montenegro, Croatia, Bosnia and Herzegovina, and Serbia).

The program covers various fields of Molecular Biology, including Molecular Biomedicine, Molecular Biotechnology and Molecular Cell Biology, and consists of plenary and invited lectures, the MolBioS award winner lecture, poster sessions and the project corner. Special attention is paid to students and young scientists through the MolBioS Student Session, flash presentations and workshops on state-of-the-art molecular biology methods.

We wish you to be inspired by exciting and outstanding lectures given by renowned scientists and experts, exchange ideas, find opportunities for new collaborations, and have good fun.

WELCOME TO

CoMBoS2

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MODULATION OF HEPATIC LIPID METABOLISM IN OBESITY-RESISTANT MICE ON A HIGH-FAT DIET

Miloš Vratarić,[1] Ana Teofilović,[1] Danijela Vojnović Milutinović,[1] Nataša Veličković,[1] Biljana Bursać,[1] Ljupka Gligorovska,[1] Bojana Mićić,[1] Mirna Jovanović[1] and Ana Djordjevic[1]

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Introduction: High-fat diet primarily leads to obesity but it can also lead to obesity resistant (OR) phenotype with various metabolic complications. Liver plays central role in modulating lipid metabolism in response to dyslipidemia induced by adipose tissue hypertrophy. The aim of this study was to define key regulatory points *that adjust lipid metabolism in the liver of OR mice on high-fat diet (HFD).*

Methods: Male C57BL/6J mice were divided into two groups: control group on normal diet (10 kcal% fat, D12450J, Research Diets, USA) and HFD group (60 kcal% fat, D12492, Research Diets, USA). After 14 weeks, mice on HFD were classified as obese or OR based on 30% difference in body weight gain compared with controls. Liver sections were analyzed histologically, while alterations in hepatic lipid metabolism were assessed by qPCR and Western blot.

Results: Although HFD restricted hepatic de novo lipogenesis, increased influx of free fatty acids (FFA) led to accumulation of lipid droplets in the liver of obese mice. In OR mice, liver morphology was restored, as was expression of insulin sensitive sterol regulatory element-binding protein 1c (SREBP-1c). Level of FFA transporter CD36 was reduced, whereas higher expression of diacylglycerol acyltransferase 2 limited lipotoxicity in OR compared with obese mice. FFA β-oxidation remained unchanged in both HFD groups.

Conclusion: Lower FFA input and reduced lipid storage and lipotoxicity in the liver of OR mice suggest that dyslipidemic complications associated with obesity could be ameliorated by targeted modulation of expression of FFA transporters and regulators of lipid droplet formation.

Key words: obesity, obesity resistance, lipid metabolism, liver

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