# IMMUNOLOGY AT THE CONFLUENCE OF MULTIDISCIPLINARY APPROACHES

# ABSTRACT BOOK

**Institute for Biological Research "Siniša Stanković" National Institute of Republic of Serbia**
**University of Belgrade**

**Immunological Society of Serbia**

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Saturday, December 7th Session: METAB INFLAMM

Short oral presentation

## IS METAFLAMMATION A USUAL SUSPECT FOR FRUCTOSE-INDUCED METABOLIC DISTURBANCES?

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Fructose overconsumption, especially in the form of sweetened beverages, has been linked to development of obesity, insulin resistance, dyslipidemia and type 2 diabetes. In rodents, high-fructose diet leads to hypertriglyceridemia, ectopic fat deposition and insulin resistance. Metabolically triggered inflammation (metaflammation) is now recognized as a link between nutrient signals and insulin resistance and considered as usual suspect for metabolic disturbances. Metaflammation usually evolve from visceral adipose tissue, progresses to liver and brain strucures, and results in peripheral insulin resistance, lipid accumulation and oxidative stress. Hence, the aim of our study was to investigate metaflammation as a trigger for fructose-induced metabolic disturbances. Experiments were performed on male Wistar rats fed with different concentrations of liquid fructose (10, 20 and 60%) during 9 weeks. Physiological and biochemical parameters, hepatic and brain inflammation, indicators of peripheral and systemic insulin resistance, as well as hepatic lipogenesis and oxidative stress were examined. The results demonstrated that fructose-enriched diet generally led to increased proinflamatory cytokines in the liver, hippocampus and hypothalamus, and to stimulated activation of proinflamatory kinases NFκB and JNK, while it did not change the expression of inflammasome component NLRP3, toll-like receptor 4 or anti-inflammatory cytokines in the liver. The observed metabolic inflammation was accompanied with impaired glucose tolerance after 10 and 20% fructose-enriched diet, while decreased hepatic insulin sensitivity, hypetriglyceridemia and increased expression of hepatic lipogenic genes were observed after all fructose diets. The treatment of fructose-fed rats with chronic unpredictable stress annulled the effects of fructose on hepatic and hypothalamic inflammation and glucose tolerance, but did not alter fructose-induced effects on lipogenesis and insulin signaling. The results suggest that fructose-induced metaflammation and systemic insulin resistance are closely interconnected, while the link between inflammation and other metabolic disturbances could still be a matter of debate.