



**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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MULTIDISCIPLINARY APPROACHES**

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**Dr Mirjana Mihailović, director of the Institute for Biological Research "Siniša
Stanković" - National Institute of Republic of Serbia, University of Belgrade**

Dr Nada Pejnović, president of the Immunological Society of Serbia

EDITORS

Tamara Saksida

Suzana Stanisavljević

Đorđe Miljković

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Poster presentation

INFLAMMATION AND INSULIN SENSITIVITY IN THE LIVER OF
FRUCTOSE-FED *Mif* DEFICIENT MICE

Ljupka Gligorovska¹, Ana Teofilović¹, Nataša Veličković¹, Danijela Vojnović
Milutinović¹, Sanja Kovačević¹, Gordana Matic¹ and Ana Djordjević¹

¹*Institute for Biological Research "Siniša Stanković"- National Institute of
Republic of Serbia, University of Belgrade, Serbia*

Introduction: The macrophage migration inhibitory factor (MIF) is a key pro-inflammatory mediator involved in the regulation of energy metabolism and metabolic inflammation in the liver. Fructose overconsumption has been previously associated with development of low-grade inflammation characterized by elevated production of pro-inflammatory cytokines and activation of mitogen-activated protein kinase (MAPK) signaling pathway. The inflammatory response can disrupt insulin signaling and genetic deletion of *Mif* may contribute to the development of systemic insulin resistance, as well. The aim: The aim of the present study was to elucidate combined effects of *Mif* deficiency and fructose-enriched diet on metabolic inflammation and insulin sensitivity in the liver of male mice. Methods: Wild type (WT) and *Mif* deficient (*MIF*^{-/-}) C57Bl/6J mice were used to analyze the effects of 9-week 20% fructose-enriched diet on indicators of insulin sensitivity and markers of metabolic inflammation (tumor necrosis factor α (TNF α), interleukin (IL)-1 β and IL-6). Deregulation of Akt signaling pathway was used as hallmark of hepatic insulin resistance. Also, the protein levels of extracellular signal-regulated kinase (ERK), c-Jun N-terminal kinase 1 (JNK) and p38 were analyzed. Results: *Mif* deficient animals exhibited elevated expression of IL-1 β and IL-6 in the liver, regardless of the diet regime, while hepatic TNF α was unchanged in all animals. On the other hand, both total and phosphorylated ERK and JNK protein levels were decreased in all fructose-fed mice. In the same animals, impaired hepatic insulin signaling, revealed by decreased pAkt and total Akt protein levels, was observed. Conclusion: Although, *Mif* deficiency led to upregulation of pro-inflammatory cytokines, fructose diet did not aggravate this effect. On the other hand, insulin signalling was diminished by fructose feeding independently of *Mif* deficiency.