

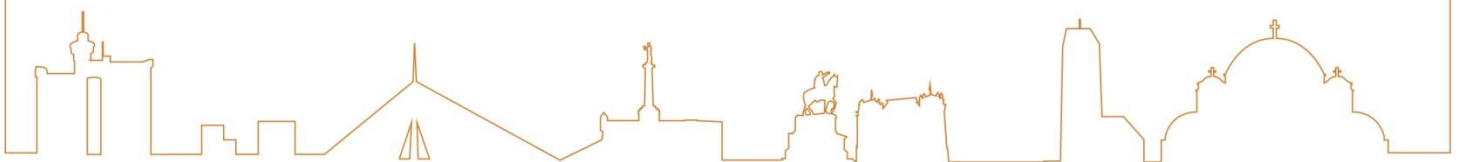
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GLUCOCORTICOID-MEDIATED EFFECTS OF MIF DEFICIENCY AND FRUCTOSE-ENRICHED DIET ON LIPID METABOLISM IN THE MOUSE INTRA-ABDOMINAL ADIPOSE TISSUE

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Introduction: The macrophage migration inhibitory factor (MIF) is a pro-inflammatory cytokine involved in metabolic inflammation and regulation of glucocorticoid action. Low-grade inflammation in adipose tissue is associated with obesity and dyslipidemia and may be caused by fructose-enriched diet. We hypothesized that the effects of MIF deficiency on lipid metabolism in adipose tissue, after high fructose consumption, could be mediated by glucocorticoids (GCs) as potent regulators of energy metabolism.

Methods: We analyzed the effects of 9-week 20% fructose-enriched diet on energy intake, body mass, intra-abdominal adipose tissue mass and histology in MIF wild type (WT) and MIF deficient (MIF^{-/-}) C57Bl/6J mice. Expression of key transcriptional regulators involved in adipogenesis and lipogenesis, peroxisome-proliferator-activated receptor γ (PPAR γ) and sterol regulatory element-binding protein-1 (SREBP-1), was also assessed. Glucocorticoid signaling was characterized by prereceptor metabolism, glucocorticoid receptor (GR) protein level and phosphorylation, and expression of GC-target genes involved in lipogenesis.

Results: Both WT and MIF^{-/-} mice on fructose diet had increased energy intake, but the elevation of adipose tissue mass and enlargement of adipocytes were observed only in fructose-fed MIF^{-/-} mice. Elevated GR protein level and its activating Ser²²⁰ phosphorylation, enhanced glucocorticoid prereceptor metabolism, an increase in PPAR γ and SREBP-1 levels and induced expression of all examined lipogenic genes were also observed in MIF^{-/-} mice on fructose diet.

Conclusion: The results show that only under fructose caloric overload MIF deficiency results in lipogenesis and adipocyte hypertrophy and that this effect might be mediated by enhanced glucocorticoid signalling in intra-abdominal adipose tissue.

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