





PROGRAM & BOOK OF ABSTRACTS



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AND VISCERAL ADIPOSE TISSUE OF FEMALE RATS

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Introduction: Increased fructose consumption, mainly through sweetened beverages, coincides with growing rate of obesity, women being more prone than men. Chronic low-grade inflammation has been implicated in the pathogenesis of obesity-related disorders including metabolic syndrome and insulin resistance.

The aim: We investigated whether fructose overconsumption causes inflammation in the visceral adipose tissue (VAT) and hypothalamus of female rats contributing to development of obesity and insulin resistance. Methods: Using qPCR and Western blot, we examined the effects of 9-week fructose-enriched diet on inflammatory status, insulin and leptin signaling in the VAT and hypothalamus, as well as on the expression of orexigenic and anorexigenic neuropeptides in the hypothalamus.

Results: Fructose-fed rats had increased nuclear accumulation of nuclear factor κB (NF- κB) and elevated expression of pro-inflammatory cytokines (IL-1 β , IL6, and TNF α), as well as increased protein level of macrophage-specific marker F4/80 in the VAT. In the

same tissue, fructose overconsumption reduced protein content and stimulatory phosphorylation of Akt kinase, while increasing inhibitory phosphorylation of insulin receptor substrate-1 (IRS-1). There were no changes in VAT mass, nor in inflammatory markers, insulin and leptin signaling (leptin receptor and SOCS3 expression) and appetite regulation (NPY, AgRP, POMC and CART) in the hypothalamus.

Conclusions: The results suggest that fructose overconsumption causes alterations in pro-inflammatory markers and reduces insulin signaling in the VAT of female rats. These alterations could be one of the first consequences of fructose overconsumption, since they were detected in the absence of obesity, and hypothalamic inflammation and insulin and leptin resistance.

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