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T1 – Immunometabolism

T1:PO.046

Acute hyperglycemia increases monocyte and t-lymphocyte content in subcutaneous adipose tissue of healthy obese women

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Background/Objectives: Hyperglycemia represents one of possible mediators for activation of immune system. It may contribute to worsening of inflammatory state of adipose tissue associated with obesity and thus accelerate the development of metabolic syndrome. The aim of our study was to investigate the effect of a short-term hyperglycemia (HG) on the phenotype and relative content of immune cells in subcutaneous abdominal adipose tissue (SAAT) in obese women without metabolic complications.
Subjects/Methods: Three-hour HG clamp combined with infusion of octreotide (to block insulin secretion) and control investigations with infusion of either octreotide or saline were performed in three groups of obese women (n=10 per group). Before and at the end of the interventions, samples of SAAT were obtained by needle biopsy. The relative content of immune cells in SAAT was determined by flow cytometry. Gene expression analysis of immunity-related markers (chemokines/cytokines, markers of macrophages and lymphocytes, Toll like receptors) in SAAT was performed by quantitative real-time PCR.
Results: In SAAT, HG induced an increase in the content of CD206 negative monocytes/macrophages ($4.9 \pm 0.5\%$ vs.$5.6 \pm 0.5\%$. $p < 0.05$) and T lymphocytes (T helper and T cytotoxic lymphocytes; $3.4 \pm 0.3\%$ vs. $5.6 \pm 0.5\%$ and $1.9 \pm 0.2\%$ vs. $3.1 \pm 0.2\%$ respectively, $p < 0.01$). Further, HG promoted an increase of mRNA levels of immune response markers (CCL2, TLR4, TNFα) and lymphocyte markers (CD3g, CD4, CD8a, TBX21, GATA3, FoxP3) in SAAT ($p < 0.05$). Under both control infusions, none of these changes were observed.
Conclusions: Acute HG increased the content of monocytes and lymphocytes in SAAT of healthy obese women. This result suggests that the short-term HG can modulate an immune status of AT in obese subjects.

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T1:PO.047

Role of interleukin 15 in liver fat accumulation in patients with concomitant obesity

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Background: non-alcoholic fatty liver disease (NAFLD) and obesity are associated with low grade inflammation. Experimental data suggest crucial role of interleukin 15 (IL-15) in NAFLD development. Y. Cepero-Donates et al. showed that exceeded secretion of IL-15 promotes fat accumulation in mice. The aim was to investigate IL-15 concentration in patients with NAFLD associated with obesity depending on steatosis degree. The study included 32 patients with NAFLD associated with obesity, 31 normal weight patients with NAFLD and 26 healthy volunteers.
Methods: NAFLD was diagnosed by abdominal ultrasound examination. Steatosis degree was assessed using hepatorenal index (HRI). Obesity was measured using body mass index (BMI) and waist circumference (WC). Concentration of IL-15 were measured using enzyme-linked immunosorbent assay kit. The results showed that IL-15 concentration was significantly increased in patients with NAFLD comparing to control group. Concentrations of IL-15 observed in NAFLD patients with concomitant obesity were significantly higher than those in NAFLD patients with normal weight ($p < 0,05$). In all NAFLD patients IL-15 correlated with HRI supporting its role in hepatic fat accumulation. Furthermore, in patients with NAFLD there was significant correlations of IL-15 concentration with BMI ($p < 0,05$) and WC ($p < 0,05$).
Conclusions: patients with NAFLD and concomitant obesity might have more significant proinflamatory status that could be caused by adipose tissue dysfunction and abnormal adipocytokines synthesis. Exceeded IL-15 observed in NAFLD patients with obesity supports its role in liver lipid accumulation found in experimental studies. Further investigations are needed to explore correlations of IL-15 with histological findings in NAFLD obese patients.

T1:PO.048

Effect of diet and cd36 deficiency on murine mesenteric lymph nodes

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Introduction: Mesenteric lymph nodes (MLN) of diet-induced obese mice are smaller than those of lean mice because of atrophy of lymphoic cells. Cluster of differentiation (CD)36 contributes to high fat diet induced obesity (DIO) and is expressed on lymphocytes.
Methods: Five weeks old male wild-type (WT) and CD36 deficient (CD36-/-) mice were kept on standard fat diet (SFD, lean) or on high fat diet (HFD, obese) for 15 weeks. Macrophage content and lymphocyte populations of MLN were analyzed by FACS.
Results: DIO, as compared to SFD feeding, was associated with significantly smaller MLN in WT mice, but not in CD36-/- mice. MLN of obese versus lean mice showed lower prevalence of cytotoxic T cells (TC; CD8+), helper T cells (TH; CD4+), activated T cells (Tact; CD4+CD25+) and regulatory T cells (Treg; CD4+FoxP3+CD25+). These differences were not observed between MLN of obese and lean CD36-/- mice. Treg cell content in MLN of WT, but not CD36-/-, mice correlated positively with MLN size. MLN macrophage content was lower for lean than obese WT mice, but was significantly higher for lean as compared to obese CD36-/- mice. Overall, MLN of obese CD36-/- mice contained significantly fewer macrophages as compared to obese WT mice. Analysis of apoptotic markers in MLN revealed lower expression of annexin V and caspase 3 (pro-apoptotic) in MLN of obese CD36-/- versus WT mice, and higher expression of annexin V and FasL for lean CD36-/- versus lean WT mice. Caspase 8, Caspase 9, Bax, AIFm2, Bcl2 and XIAP were not affected by either diet or genotype, thus not supporting differences in apoptosis.
Conclusion: DIO in WT mice, as compared to lean mice, is associated with smaller MLN, lower prevalence of TC, TH, Tact and Treg cells. CD36-/- mice appear to be protected against these changes.

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T1:PO.049

Nesfatin-1 levels are significantly increased after weight loss induced by bariatric surgery

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Background and aims: Nesfatin-1 was recognized as a central novel anorexigenic modulator of food intake and body weight.Thus, nesfatin-1 might be a target to influence hunger and satiety. The peripheral mode