

ImmunoTools IT-Box-Cy55M-Award 2013



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The immunomodulatory effect of Galectin-3 on adipose tissue derived natural helper cells in diet-induced obesity and diabetes

We are involved in investigation of adipose tissue inflammation during obesity that underlies metabolic dysfunctions in type 2 diabetes. We have recently reported that body weight, amount of visceral adipose tissue, fasting blood glucose were significantly increased in Galectin-3 deficient mice compared with wild-type mice fed a high-fat diet. Visceral adipose tissue from obese Gal-3 deficient mice contained increased percentages of T and NKT lymphocytes with Th1-phenotype and pro-inflammatory CD11c⁺CD206⁺ macrophages while reduced CD4⁺CD25⁺FoxP3⁺ T regs accompanied with a marked infiltration of macrophage/dendritic lineage cells, higher NLRP3 inflammasome and IL-1 β expression in pancreatic islets (*Pejnovic N, Pantic J et al. Galectin-3 Deficiency Accelerates High-Fat Diet-Induced Obesity and Amplifies Inflammation in Adipose Tissue and Pancreatic Islets. Diabetes 2013 Epub ahead of print DOI: 10.2337/db12-0222*)

Further investigation will focus on the role of innate lymphoid cells in obesity-induced chronic inflammation in Galectin-3 deficient mice. The recent publication (*Moro K. Et al. Innate production of TH2 cytokines by adipose tissue-associated c-Kit⁺Sca-1⁺ lymphoid cells. Nature 2010; 463:540-546*) has revealed a new type of innate lymphocytes present in a novel lymphoid structure associated with adipose tissue. We will isolate natural helper cells from adipose tissue and propagate them in vitro by using recombinant cytokines such as IL-33, IL-25 or IL-23. Also, the immunomodulatory effects of the cytokines produced by various subpopulations of innate lymphoid cells such as IL-13, IL-5, IL-17 and IL-22 on adipose tissue macrophages, Tregs and myeloid-derived suppressor cells will be explored.

ImmunoTools IT-Box-Cy55M for Jelena M. Pantic

includes 55 recombinant mouse cytokines

rm EGF, rm Eotaxin / CCL11, rm FGF-a / FGF-1, rm FGF-b / FGF-2, rm FGF-8, rm Flt3L / CD135, rm G-CSF, rm GM-CSF, rm GRO-a / CXCL1, rm GRO-b / CXCL2, rm IFN γ , rm IL-1 α , rm IL-1 β , rm IL-2, rm IL-3, rm IL-4, rm IL-5, rm IL-6, rm IL-7, rm IL-9, rm IL-10, rm IL-11, rm IL-13, rm IL-15, rm IL-16, rm IL-17A, rm IL-17C, rm IL-17F, rm IL-19, rm IL-20, rm IL-21, rm IL-22, rm IL-25 / IL-17E, rm IL-27, rm IL-31, rm IL-33, rm IP-10 / CXCL10, rm LIF, rm MCP1 / CCL2, rm M-CSF, rm MIP-1 α / CCL3, rm MIP-1 β / CCL4, rm MIP3 α / CCL20, rm MIP3 β / CCL19, rm NGF- β , rm PDGF-AA, rm PDGF-BB, rm RANTES / CCL5, rm sCD40L / CD154, rm SCF, rm SDF-1 α / CXCL12a, rm SDF-1 β / CXCL12b, rm TNF α , rm TPO, rm VEGF

[DETAILS](#)