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#### POSTER PRESENTATIONS

#### P-0951

#### The populations of peripheral blood T-lymphocytes at different stages of differentiation in patients with Parkinson's disease

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Parkinson's disease (PD) is one of the most common neurodegenerative diseases. The mechanisms of PD development are largely associated with the processes of chronic inflammation in the brain tissue (neuroinflammation). The development of neuroinflammation involves the central nervous system's resident immune cells and the cells of the peripheral immune system migrating to the brain. We examined 31 PD patients, 33 old-aged healthy donors (OHD), 30 young healthy donors (YHD). Immunophenotyping was performed using flow cytometry. Was analyzed the populations of T-lymphocytes (CD3+) at different stages of differentiation: replicative senescence (CD56-CD57+), Naïve (CCR7+CD45RA+), Central memory (CM) (CCR7+ CD45RA-), terminally differentiated effector memory (TEMRA) (CCR7- CD45RA+), effector memory (EM) (CCR7- CD45RA-). The proportion of T-lymphocytes (CD3+CD56-) expressing the CD57 marker was lower in the PD group than in the OHD group (8.7 and 13.1, p = 0.02). The proportion of these cells in the group of the YHD was significantly lower than in the group of PD and OHD. There were no significant differences in the naïve, CM, EM, TEMRA populations between patients with PD and OHD in the population of T-lymphocytes (CD3+) at different stages of differentiation. This study demonstrates that the peripheral immune profile in PD is not typical for older donors. We found that there is no replicative senescence of T-cells (CD3+CD56-CD57+). However, in the group of PD and OHD in the population of T-lymphocytes (CD3+) at different stages of differentiation, no differences were found.

Keywords: Ageing, biomarkers, immune senescence, memory

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#### P-0952

#### Severity of allergic rhinitis symptoms is associated with ceruloplasmin levels and a deficiency of microbial strains that sequester iron

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Since allergy is associated with iron deficiency, we sought to determine whether iron and microbial parameters correlate with the clinical response of allergic rhinitis subjects during nasal provocation. Female allergic subjects donated blood, and stool samples before they underwent a graded nasal provocation (n=38) with birch or grass pollen extract. Total nasal symptom scores (TNSS), visual analogue scale (VAS) and weight of nasal fluids were recorded. Complete blood cell counts and iron metabolism markers were determined. Stool samples were subjected to 16S rRNA amplicon sequencing. Serum hepcidin was assessed by ELISA. LegendPlex assay analysis was utilized to define levels of IgE, ceruloplasmin, lipocalin2 and cytokines in nasal fluids. Trace elements in serum and aqueous stool extracts were determined via inductively coupled plasma-mass spectrometry. Nasal and serum ceruloplasmin was the sole protein marker that positively correlated with all assessed symptom parameters (TNSS, VAS and nasal fluid weight), VAS scores in addition correlated with serum transferrin, haptoglobin and nasal IgE-levels. A positive correlation to symptoms was confirmed for serum copper, whereas gut iron and cobalt showed an inverse relationship to clinical symptoms. Particularly members of the order Bacteriodales and members of the genus Ruminococcus seems protective against TNSS and correlate well with the presence of gut iron. For the first time, we show that essential parameters in iron homeostasis, such as the copper-containing ferroxidase ceruloplasmin, correlate with the allergic outcome. Additionally, commensal bacteria sequestering iron seem to play a beneficial role against hay fever in adults.

Keywords: Allergic disorders, drugs for immune modulation, microbiome and environmental factors

#### P-0953

#### Overexpression of galectin 3 in pancreatic beta cells amplifies beta cell apoptosis and islet inflammation in type 2 diabetes in mice

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During obesity hematopoetic cells-derived galectin 3 induces insulin resistence. While the role of galectin 3 expressed in islet invading immune cells in both type of diabetes has been studied, the importance of expression of this molecule on the target pancreatic beta cells is not defined. We have used 10-12 weeks old C57/BL6 male mice (WT) and C57/ BL6 mice with transgenically enhanced Gal-3 expression in pancreatic β cells (TG). Obesity was induced with 16 weeks high fat diet regime. Pancreatic beta cells were tested for susceptibility to apoptosis induced by non-esterified fatty acids and cytokines as well as parameters of oxidative stress. The overexpression of galectin 3 increases beta cells apoptosis in HFD conditions and increases the percentage of proinflammatory F4/80+ macrophages in islets that express galectin 3 and TLR4. In isolated islets, we have shown that galectin 3 overexpression increases cytokine and palmitate-triggered beta cells apoptosis and also increases NO2- induced oxidative stress of beta cells. Also, in pancreatic lymph nodes, macrophages were shifted towards proinflammatory TNF-α producing phenotype. By complementary approach *in vivo* and *in vitro*, we have shown that galectin 3 overexpression facilitates beta cell damage, enhances cytokine and palmitate-triggered beta cells apoptosis and also increases NO2- induced oxidative stress in beta cells. Further, the results suggest that increased expression of galectin 3 in the pancreatic beta cells affects the metabolism of glucose and glycoregulation in mice on HFD, affecting the fasting glycemic values, as well as glycemia affer glucose loading.

Keywords: Animal models, cytokines and mediators, diabetes, effector molecules