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POSTER PRESENTATIONS

P-0194

IFN-γ produced in infection down-regulates PPAR-γ to modulate adipose tissue biology

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Adipose tissue is a major lipid storage organ which releases and distributes lipids to maintain energy homeostasis. In context of metabolic disease, adipose tissue was shown to closely interact with the immune system as obesity drives inflammation in this organ which alters local and systemic regulation of metabolism. However, how immune cells interact with adipocytes in context of viral infection is largely unknown. Here, we investigated the impact of virus-induced activation of the immune system on adipose tissue metabolism and the underlying benefit of these changes to the organism. In an *in vitro* model of adipocyte differentiation, we could show that the pro-inflammatory cytokine IFN-γ significantly reduces cellular lipid content. High-throughput transcriptome analysis of these cells demonstrated that IFN-γ mediates down-regulation of PPAR-γ, a master regulator of adipocyte tissue metabolism, as well as many of its downstream targets, causing a net efflux of nutrients. Infection of mice with cytomegalovirus induced a striking reduction of adipocyte cell size and induced a change in the transcriptional profile of these cells corresponding with an IFN-γ imprint. Accordingly, infection caused a systemic increase of adipose tissue derived nutrients, such as free fatty acids in circulation. Importantly, our results indicate that these nutrients promote the acute lymphocyte response to viral infection. Our findings suggest that cytokines produced in response to viral infection can modulate adipocyte and systemic metabolism to benefit the immune response to infectious disease.

Keywords: Cytokines and mediators, metabolic control of immune responses, nutrients, viral infections

P-0205

Glycan-dependent signalling routes and transcriptional programs in human dendritic cells after triggering the C-type lectin receptor MGL

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C-type lectin receptors on dendritic cells can shape the immune response to bacteria, viruses and tumour cells. Signalling through the macrophage galactose-type lectin (MGL) reduces the glycolytic capacity of monocyte-derived dendritic cells (moDCs) and increases their IL-10 and TNFα production, which stimulates a Th2 and Tr1 response. Recently, several MGL ligands were discovered to induce different conformations of the MGL carbohydrate-recognition domain but the effect on the moDC maturation and functionality was only investigated for two of these MGL ligands. We will investigate the transcriptional programs that are induced in response to five different MGL ligands, which we coupled to dendrimers to increase their polyvalency. Furthermore, we will determine which cytokines, co-stimulatory and inhibitory receptors, and signalling molecules are associated with the transcriptional programs triggered by the five MGL ligands. Up to now, MGL stimulation resulted in a higher CD11c and CD14 co-expression by moDCs. The five MGL ligands also displayed a different capacity to increase IL-10 secretion. In the future, MGL-mediated ligand-specific transcriptional programs could be exploited to manipulate immune responses in human DCs.

Keywords: Cell signalling, dendritic cells, molecular immunology

P-0206

Novel aryl hydrocarbon receptor antagonist promotes macrophage pro-inflammatory phenotype

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Aryl hydrocarbon receptor (AhR) is a ligand-activated transcription factor that responds to various aromatic compounds, both endogenous such as kynurenine and exogenous such as natural plant flavonoids, polyphenolics and indoles. AhR has been recently identified as the regulator of immune cells function. The activation of AhR generally leads to the attenuation of the immune response, while its inhibition promotes the opposite effects. In this study we have selected several plant-derived indol derivatives and tested them for their AhR ligand activity. A potent AhR antagonist was identified (code C46) and further evaluated on mouse peritoneal macrophages for its ability to modulate macrophage function. Macrophages were exposed *in vitro* to compound C46 in concentrations ranging from 250 ng/ml to 1000 ng/ml for 48 h. Flow cytometry analysis showed that C46 significantly and dose-dependently up-regulated the proportion of M1 macrophages (F4/80+CD40+). Interestingly, C46 influenced only M1 macrophages, as the proportion of M2 (F4/80+CD206+) remained stable upon the exposure to C46. In addition, C46 increased the cytocidal function of macrophages by increasing the content of nitric oxide as determined by DAF-FM staining. Similarly to *in vitro* effects, intraperitoneal C46 administration up-regulated the proportion of M1 macrophages in the peritoneum, 72 h after the treatment. In conclusion, blocking of AhR pathway by C46 potentiates pro-inflammatory function of macrophages and it may represent a promising approach for future testing in animal models of cancer.

Keywords: Biology of the immune system, immune regulation and therapy, macrophage, molecular immunology

P-0218

Generation and intial characterization of a SARM epitope-tagged mouse

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Sterile alpha and HEAT/Armadillo motif–containing (SARM) protein is a member of Toll/IL-1R (TIR) domain protein family and is highly conserved in evolution with a role in innate immunity in diverse organisms. A major recent discovery was that the TIR domain of SARM actually has NADase enzymatic activity which is required for SARM-dependent neuronal cell death. Although most research to date on SARM relates to its function in axon degeneration, SARM likely has non-neuronal functions too, and we showed that SARM is a regulator of inflammasome responses and pyroptosis in murine macrophages. Studies of non-neuronal SARM are hampered by the fact that immunodetection of the protein outside the brain is challenging. To address this, here using CRISPR technology we generated a mouse expressing epitope-tagged SARM whereby the protein contains a triple-Flag tag and Strep-tag II on its C-terminus (SARM-FS). We confirmed this SARM-FS mouse shows the same level of *Sarm1* mRNA expression compared to the wild-type mouse and also discovered that the mouse expresses SARM protein not only in brains but also in various tissues such as the liver, spleen and kidney, albeit at a lower level than in the brain. Furthermore, we established immortalised BMDMs isolated from the SARM-FS mouse and confirmed SARM protein expression in these cells. These data suggest that SARM has roles in various tissues apart from the brain and pave the way for further work to examine the broader contribution of the SARM TIR domain and NADase activity to normal physiology and the immune response.

Keywords: Animal models, macrophage, molecular immunology