Methods Human adipose-derived stem cells (hASCs) were encapsulated via a micromolding method. We first manufactured polydimethylsiloxane chips containing 1600 micromolds with a circular shape, 150 μm in diameter and 100 μm in depth. For cell encapsulation, a solution of 2% alginate (w/v) containing 3 million of hASCs per mL polymer was deposited onto the chips, loaded in the moulds either by sedimentation or centrifugation and crosslinked using an agarose gel charged with CaCl_2 . The number of encapsulated cells was evaluated by a CyQUANT assay immediately and 24 hour after encapsulation. The impact of encapsulation on metabolic activity was determined by a presto blue assay 24 hours after encapsulation and 24 hour after their injection through a 26G needle.

Results We successfully obtained cylindrical alginate microparticles presenting a diameter of $103 \pm 0.7 \mu\text{m}$. Using cell quantification, we determined that the centrifugation method allowed the encapsulation of $30\,333 (\pm 5552)$ cells within one chip versus $6056 (\pm 2862)$ by sedimentation. Cell number and metabolic activity remained stable for 24 hours after encapsulation. We also demonstrated that injection through a 26G needle had no impact on the viability of encapsulated cells.

Conclusions Our results show that micromolding allows hASCs encapsulation into alginate particles injectable through a 26 G needle without impacting cell viability. Future work will focus on evaluating *in vitro* long-term encapsulated cell survival and functionality. In case of success, we will then consider intra-articular injection in an animal model of osteoarthritis.

Disclosure of interest None declared

P112 TARGETING T-CELL TRAFFICKING IN A MURINE MODEL OF SJÖGREN'S SYNDROME

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Introduction Salivary glands of primary Sjögren's syndrome (pSS) are characterised by complex leukocyte infiltration organised into tertiary lymphoid structures (TLS). The mechanisms regulating leukocyte trafficking into inflamed salivary glands are poorly described, but dysregulated T-cell recruitment during inflammation is believed to contribute to disease onset and chronicity. We recently described a homeostatic pathway in which a B cell-derived peptide (PEPITEM), secreted in response to adiponectin, regulates T-cell trafficking during inflammation via sphingosine 1 phosphate activity on endothelial cells.¹ Loss of this pathway by downregulation of adiponectin receptor on circulating B cells has been demonstrated in type 1 diabetes and rheumatoid arthritis, suggesting a potential role for PEPITEM in the pathogenesis of autoimmune diseases and indicating a role for adiponectin receptor as biomarker in autoimmune diseases.¹

Objectives We aimed to investigate the efficacy of PEPITEM as an inhibitor of T-cell trafficking in an inducible animal model of salivary gland inflammation that mimics the histological features of pSS and to investigate the potential translatability of this pathway in patients with pSS.

Methods Submandibular salivary glands of C57BL/6 mice were intra-ductally cannulated with luciferase-encoding replication-deficient adenovirus to induce TLS formation as previously described.² Mice were administered daily either with PBS or PEPITEM by intraperitoneal injection from day 0, and their salivary glands dissected at day 5 post cannulation. T-cell infiltration into salivary glands was assessed using a combination of flow cytometry, immunofluorescence and qRT-PCR.

Results B cells in sera from cannulated animals express lower levels of both adiponectin receptors 1 and 2 in comparison with non-inflamed control mice. In cannulated animals treated with PEPITEM, histological analysis of salivary glands revealed fewer, as well as less aggregated, infiltrating T cells. Both CD4⁺ and CD8⁺ numbers were significantly lower in the salivary glands of PEPITEM-treated animals. Furthermore, administration of PEPITEM also decreased mRNA transcripts for lymphotoxin beta, IL-7, lymphoid chemokines (CCL19 and CXCL13) and T cell chemokine receptor CCR7, cytokines and chemokines known to regulate ectopic lymphoidogenesis in pSS. Human samples of pSS are currently being assessed to validate the relevance of this pathway in pSS.

Conclusions These results demonstrate that administration of exogenous PEPITEM can reduce T-cell influx into salivary glands. This may represent a rescue of the homeostatic regulation of leukocyte trafficking, which is disrupted in inflammation. Our work suggests that PEPITEM should be considered to address the therapeutic needs in chronic inflammatory conditions and that the detection of decreased levels of adiponectin receptor could be used as biomarkers in pSS.

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Disclosure of interest None declared

P113 ARGINASE I AND THE METABOLIC CONTROL OF OSTEOCLASTOGENESIS

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Introduction Osteoclasts are giant, multi-nucleated cells that derive from the monocyte-macrophage lineage and are regulators of bone turnover. Availability and catabolism of L-Arginine have been implicated with immune cell biology, skewing inflammatory responses within myeloid cells in a pro- or anti-inflammatory manner.

Objectives While the role of L-Arginine within certain myeloid lineages such as macrophages is well appreciated, its role within osteoclasts is relatively unknown. We therefore aim to investigate L-Arginine metabolism in the context of osteoclastogenesis.

Methods We analysed osteoclastogenesis of C57BL/6J wildtype bone marrow cells *in vitro* in the presence and absence of recombinant Arginase 1 (recArg1). This approach was complemented via qPCR analysis of relevant osteoclast marker genes and an extracellular flux assay. We further investigated the