in the 'Complement activation'. GO analysis of the cellular components demonstrated that most proteins were annotated as exosome, vesicle and micro-particle. KEGG pathway analysis revealed proteins were significantly enriched in 'Complement and coagulation cascades', 'Staphylococcus aureus infection', 'Systemic lupus erythematosus' and 'PI3K-Akt signaling pathway'. ELISA demonstrated that both EVs-derived SAA1 and serum SAA1 were more abundant in AS group, showed a consistent with the Label-free-LC-MS/MS data.

Conclusions Differentially expressed proteins were found in serum-derived EVs of AS patients, and the functional analysis indicated that they may potentially involved in immune response.

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Disclosure of Interest None declared.

P163

EFFECT OF HIGH FAT DIET ON SYSTEMIC AND LOCAL INFLAMMATION IN THE CONTEXT OF OSTEOARTHRITIS AND RHEUMATOID ARTHRITIS MOUSE MODELS

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Career situation of first and presenting author Student for a master or a PhD.

Introduction Adipocytokines are bioactive factors mainly produced by adipose tissue. Adipokines such as leptin, adiponectin and visfatin exert various important effects on energy homeostasis as well as immune responses.

Objectives This work investigates the influence of obesity on systemic and local inflammation and adipokine expression in rheumatoid arthritis (RA) and osteoarthritis (OA) mouse models at different time points.

Methods The OA model (DMM, destabilization of the medial meniscus) was performed in C57Bl/6 mice fed with HFD (high fat diet) or ND (normal diet) prior to OA induction. In RA (CIA, collagen induced arthritis), DBA/1Rj mice were fed with the same diets prior to CIA induction. Mice were sacrificed to collect histological as well as serological data at 4, 6 and 8 weeks (DMM model) or at 4, 5.5 and 7 weeks (CIA model) after arthritis induction. Histological scoring for arthritis induction as well as assessment of a clinical score for the CIA model was performed. To evaluate the effects of HFD on adipokine levels, adiponectin, leptin and visfatin serum concentrations were measured together with the inflammation markers CRP and IL-6. Diet-induced systemic changes were analyzed using a fatty liver score and evaluation of crown-like structures (CLS) in adipose tissue.

Results Induction of OA/RA could be successfully combined with obesity induced by HFD, shown by the histological destruction as well as increased fatty liver score and body-weight. In DMM model, the number of CLS were

significantly higher in the HFD group (0.2 ± 0.16 , $n=7$) compared to the ND group (5.2 ± 0.98 , $n=8$) but could not be observed in DBA/1Rj mice. However, CIA induction increased the number of CLS in HFD and especially in ND animals. Furthermore, CIA and DMM induction decreased systemic leptin levels significantly. Interestingly, even HFD-induced leptin was greatly reduced in CIA animals regarding leptin levels. Additionally, CIA induction increased significantly CRP levels in DBA/1Rj mice illustrating the inflammatory properties of CIA vs the DMM model.

Conclusions The data show that OA is deteriorated by HFD, similar to the human organism. In the CIA model, HFD or ND mice showed no significant difference in histological CIA scoring. Based on the observed high numbers of CLS in CIA animals with ND and the strong reduction of leptin levels in CIA animals with HFD, we could also support the idea that CIA onset and severity are mainly obesity independent while OA (DMM) appears to be influenced by obesity.

Disclosure of Interest None declared.

P164

PORPHYROMONAS GINGIVALIS EXPERIMENTALLY INDUCES PERIODONTITIS AND AN ANTI-CCP2-ASSOCIATED ARTHRITIS IN THE RAT

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Career situation of first and presenting author Post-doctoral fellow.

Introduction Association between periodontal disease (PD) and rheumatoid arthritis (RA) has been extensively described, but direct evidence of causal involvement of PD in RA is missing.

Objectives We investigated the priming role of oral *Porphyromonas gingivalis* (*P.gingivalis*) in PD and subsequent RA and we assessed biomarkers of bone resorption and arthritis development in rats.

Methods Lewis rats were orally exposed to either *P.gingivalis*, *Prevotella intermedia* or control gel for one month, and then followed during 8 months. The onset and development of PD was assessed by serology, gingivitis severity, and micro-computed tomography (μ CT). We investigated arthritis development using circulating pro-inflammatory markers, anti-cyclic citrullinated peptide (CCP), anti-citrullinated protein antibody (ACPA), ankle histology, and μ CT.

Results PD was only observed in the *P.gingivalis* treated rats, as early as one month post-exposure. Joint and systemic inflammation were detected only in the *P.gingivalis* group after 4 and 8 months. At 8 months, inflammatory cell infiltrate was observed in ankle joints, and paralleled cortical erosions and overall cortical bone reduction. Furthermore, anti-CCP2 correlated with local and systemic bone loss.

Conclusions In our long term study, PD induced by oral exposure to *P.gingivalis* triggered seropositive arthritis, with systemic inflammation and bone erosions. This is the first *in vivo* demonstration of arthritis induced by oral priming with *P.gingivalis*.