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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Acknowledgements This study was supported by Medical Scientific Research Foundation of Guangdong Province(No. A2017551) and Natural Science Foundation of Guangdong Province (No.2017A030313526).

Disclosure of Interest None declared.

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

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10.1136/annrheumdis-2018-EWRR2019.145

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.

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

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10.1136/annrheumdis-2018-EWRR2019.146

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*.

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Acknowledgements This work was funded by Aide à la Recherche médicale de proximité (AIRE) and Aide à la Recherche Médicale Ondaine et Environs.

Disclosure of Interest G. Courbon: None declared, M. Rinaudo-Gaujous: None declared, V. BLASCO-BAQUE: None declared, I. Auger: None declared, R. CAIRE: None declared, L. Mijola: None declared, L. Vico: None declared, S. Paul : None declared, H. Marotte Grant/research support from: Pfizer, AbbVie, Nordic Pharma, BMS, Consultant for: Pfizer, AbbVie, Nordic Pharma, MSD, UCB, BMS, Novartis, Lilly, Roche Chugai, Janssen, Biogen, Biogaran, Sanofi.

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MOLECULAR AND CELLULAR BIOMARKERS THAT DISCRIMINATE JUVENILE IDIOPATHIC ARTHRITIS FROM SEPTIC ARTHRITIS

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10.1136/annrheumdis-2018-EWRR2019.147

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

Introduction Juvenile idiopathic arthritis (JIA) and septic arthritis (SA) are the most frequent cause of arthritis among children under the age of 16 years. JIA is an inflammatory disorder with articular inflammation that persists for more than 6 weeks, while SA is caused by bacterial infection in children joints. Although these diseases have different pathophysiological basis and involve different treatments and prognoses, they share clinical similarities.

Objectives To date, there are no markers sufficiently reliable to discriminate between these two forms of arthritis at the onset of the disease. Our goal is to identify diagnostic biomarkers capable of discriminating between JIA and SA. To that end, we focused on microRNAs (miRNAs) and myeloid cell subsets, both playing a major role in the immune system.

Methods We analyzed serum and synovial fluid (SF) samples from JIA or SA patients using a miRNAs Whole Transcriptome Assay associated with a next-generation sequencing detection technology to measure the expression level of 2083 miRNAs in a pilot study (oligoarticular JIA (oJIA): n=5; SA: n=3) and validated using RT-qPCR in a replicative study (oJIA: n=9; SA: n=9). In parallel, we performed a phenotypic characterization of peripheral blood (PB) and SF myeloid subpopulations using a flow cytometer (oJIA: n=9; SA: n=8).

Results Principal component analysis and hierarchical clustering characterizations did not show significant differences between serum miRNAs of oJIA and SA patients. However, we observed a distinct miRNA profile signature in SF between the two diseases with 16 upregulated miRNAs and 5 downregulated that perfectly discriminates oJIA and SA (p<0.01). In addition, phenotypic characterization revealed the existence of 5 myeloid cell subsets with distinct accumulation profile between the SF of oJIA and SA (p<0.05).

Conclusions In this study, we propose for the first time a SF-based miRNA signature as well as 5 myeloid cell subsets that discriminate between oJIA and SA and might be used as potential diagnosis markers in juvenile arthritis. Moreover, these markers give an insight into the mechanisms that are differentially altered between inflammatory and infectious arthritis.

Disclosure of Interest None declared.

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IMPACT OF DISEASE ACTIVITY, DURATION AND MUSCLE INVOLVEMENT ON CHANGES OF BODY COMPOSITION IN MYOSITIS PATIENTS

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10.1136/annrheumdis-2018-EWRR2019.148

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

Introduction Skeletal muscle, pulmonary and articular involvement in idiopathic inflammatory myopathies (IIM) limit the mobility/self-sufficiency of patients, and can have a negative impact on body composition.

Objectives To assess body composition and physical activity of IIM patients and healthy controls (HC).

Methods 54 patients with IIM (45 females; mean age 57.7; disease duration 5.8 years; PM, 22/DM, 25/IMNM, 7) and 54 age-/sex-matched HC (45 females, mean age 57.7) without rheumatic/tumor diseases were included. PM/DM patients fulfilled Bohan/Peter criteria for PM/DM. We assessed anthropometric parameters and body composition (by densitometry: iDXA Lunar, and by bioelectric impedance: BIA2000-M), and physical activity (by Human Activity Profile questionnaire, HAP). Routine biochemistry analysis was performed after 8 hours of fasting. Disease activity was evaluated by MITAX and MYOACT activity score, muscle involvement by manual muscle test (MMT-8) and functional index 2 (FI2).

Results Compared to HC, patients with IIM had significantly increased body fat% (BF%) but significantly decreased lean body mass (LBM), bone mineral density (BMD) and increased extracellular mass/body cell mass (ECM/BCM) ratio, which reflects worse muscle predispositions for physical activity and deteriorated nutritional status. Disease duration negatively correlated with BMD and LBM (assessed by BIA). Disease activity assessed by MITAX and MYOACT positively correlated with LBM (assessed by both BIA and DXA), as well as with basal metabolic rate (BMR), and fat free mass (FFM). CRP was positively associated with BF% (iDXA and BIA). Higher BF% (iDXA) was associated with worse physical endurance (FI2) and worse ability to perform physical activity (HAP). MMT-8 score negatively correlated with ECM/BCM ratio.

Conclusions We found significant negative changes in body composition of our IIM patients compared to healthy age-/sex-matched individuals, associated with their disease activity and duration, inflammatory status, skeletal muscle involvement, and physical activity. These data could reflect their impaired nutritional status and predispositions for physical exercise, aerobic fitness and performance.