cardiovascular changes were assessed by Cardiac Magnetic Resonance Imaging (CMR).

Methods PBMCs samples from 60 RA patients (mean (90% CI)) age 62.4 (60.2, 64.6)y.o, sex F/M 42/18, disease duration 153.3 (121.3, 185.2) mths and n=18 healthy controls (HC); age 42 (37.8,46.0) y, F/M 12/6 were obtained. RA patients were stratified based on CMR into: no CVD (RA CMRneg n=13) and abnormal CMR (RA CMRpos n=26), and RA with clinical CVD (no CMR, defined as history of cerebrovascular disease or ischaemic heart disease) (RA CVD n=21). qPCR of ISGs was performed using TaqMan Gene Expression Assays on Biomark (Fluidigm). Factor scores were created using the set of 51 genes measured in 78 subjects.

Results All routinely used biomarkers were associated with RA diagnosis (ACPA, RF, TJC, SJC, CRP, DAS28). Total cholesterol and LDL levels were higher in RA than in HC group (p=0.051 and p=0.015). Three IFN-1 scores were present in the dataset (IFN Score 1, 2 and 3) and were composed of 19, 21 and 7 genes respectively. IFN-1 score 2 showed significant differences between HC and RA CMRpos groups (mean difference and 90% CI: 0.52 (0.09, 0.96)) and HC and RA CVD (0.57 (0.10, 1.03)) as well as all RA patients with CVD (CMRpos and RA CVD) ((0.54 (0.16, 0.93); p<0.05 for all comparisons). IFN-1 Score 3 showed trend for a difference between RA CMRneg and CMRpos patients (0.37 (0.03, 0.70); p=0.071). There was no statistical difference between HC and RA CMRneg groups.

Conclusions An IFN-1 score 2 emerged as a possible factor characterising progression along a CVD continuum in RA patients, from no CVD to subclinical and clinical CVD; also distinguishing between HC and RA with subclinical and clinical CVD. These results warrant further evaluation in a larger cohort to confirm the findings.

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P124 ALTERED CD4+ T CELL DNA METHYLATION IN EARLY RHEUMATOID ARTHRITIS

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Introduction Over 100 genetic variants associated with rheumatoid arthritis (RA) to date explain a relatively small proportion of its heritability. Epigenetic factors, including variation in DNA methylation, may in part account for this, impacting gene expression at a cellular level.

Objectives Seeking insights into disease pathogenesis and discriminatory biomarkers of clinical value, we undertook an epigenome-wide association study (EWAS) of DNA methylation in CD4 +T cells of untreated RA and control patients attending a UK early arthritis clinic.

Methods A cohort of 44 treatment-naive RA patients were recruited from the Newcastle Early Arthritis Clinic (NEAC) along with a comparator cohort of 53 matched disease controls with arthritis. CD4 +T cells were isolated from fresh peripheral blood by positive selection, and DNA/RNA extracted from lysates. DNA methylation was quantified on the Illumina Human MethylationEPIC array, and gene expression measured using the Illumina 12HT BeadChip. Following methylation data pre-processing, functional normalisation and probe filtering, 715,279 CpGs were considered. Batch effects were corrected using the ComBat function and a moderated T-statistic identified differentially methylated positions (DMPs) between RA patients and disease controls. Differentially methylated regions (DMRs; clusters of DMPs) were also sought using the DMRcate function. Linear Discriminant Analysis (LDA) combined with a Stepwise Forward Selection (SFS) procedure was used to identify the most discriminatory CpGs for downstream independent validation.

Results Between RA and non-RA patients we identified a total of 263 DMPs in CD4 +T cells, of which 159 were hypomethylated in RA patients (adjusted p-value<0.05, $\Delta\beta$>5%), as well as 14 DMRs. One DMR in particular, comprising 4 intronic CpG sites in the *CD151* gene that were hypomethylated in RA patient CD4 +T cells, was notable for the strong association between *CD151* expression and methylation. This suggests a mechanism whereby altered DNA methylation during early disease determines pathogenically relevant gene expression. We assessed the ability of our differentially-methylated CpG sites to discriminate patients by diagnosis through evaluating the performance of a classification algorithm. LDA classification based on CpGs selected by SFS achieved an area under the receiver operating characteristic (ROC) curve of 0.82 (95% confidence interval: 0.80–0.85).

Conclusions The CD4 +T cell methylation landscape in age- and sex-matched early inflammatory arthritis patients has unique features in early RA, indicating a possible role for DNA methylation in conferring susceptibility to this condition. A further application of these data may be in predicting outcome in undifferentiated arthritis, though further work is required to explore this.

Disclosure of interest None declared

P125 CHONDROCYTES DERIVED FROM MESENCHYMAL STEM CELLS DIFFERENTIATED IN THE PRESENCE OF PLASMA-DERIVED EXTRACELLULAR VESICLES FROM OSTEOARTHRITIC PATIENTS EXPRESS DISEASE-RELATED GENES

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Introduction Osteoarthritis (OA) is an age-related musculoskeletal disease and the most common form of arthritis characterised by low grade synovial inflammation and articular cartilage degeneration. OA has many risk factors such as genetic predisposition, age and gender, but also joint specific (e.g. trauma) and systemic (obesity, hormones). Also many different cell types have been implicated in the pathogenesis of OA and interplay between these cells contribute to the dynamic events