analysis (PCA) was applied on the BioQC enrichment scores to (i) identify potential clusters of samples and to (ii) identify the gene signatures responsible for the clustering. In addition, associations between cadherin 11 gene expression and gene signatures were tested.

**Results** In both studies, cadherin 11 was higher expressed in RA patients compared to healthy controls. PCA performed on gene enrichment scores showed RA patients clustered apart from the healthy controls. Moreover, fibroblast, macrophage, lymphocyte and osteoclast gene signatures were significantly enriched in the RA patients compared to healthy controls in both studies. Unexpectedly, an adipokine-related signature was significantly enriched in RA patients from one transcriptomics dataset (GSE77298), while only showing a trend in the second set (GSE7037). In addition, in both studies Cadherin 11 was associated positively with fibroblast and lymphocyte signatures and negatively with adipokines-related signature.

**Conclusions** The identified cadherin 11 related gene signatures expand our knowledge on cadherin 11 biology in human RA and may serve as potential biomarkers for RA studies in the future.

**REFERENCES**

Disclosure of interest K. Hatje Employee of: Roche, T. Kam-Thong Employee of: Roche, D. Hartl Employee of: Roche, G. Duchateau-Nguyen Employee of: Roche

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**PO42 INVESTIGATING IL-6 INTRACELLULAR SIGNALLING IN PERIPHERAL BLOOD CELL SUBSETS IN PATIENTS AT EARLY AND LATER STAGES OF RHEUMATOID ARTHRITIS (RA)**

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**Introduction** Rheumatoid arthritis (RA) is a chronic, inflammatory arthritis that evolves along an immunological and inflammatory disease continuum. The era of targeted biological therapies has been transformative; however, a significant unmet need is the effective tailoring of therapy to deliver optimal treatment responses. In addition, the concept of a window of opportunity is well-recognised whereby early commencement of treatment confers improved outcomes compared to delayed treatment. The importance of pro-inflammatory cytokines TNF and IL-6 in particular, is well recognised; but high, homogeneous response in early RA (ERA) compared to later RA remains unexplained.

**Objectives** The present project focuses on measuring the phosphorylation of STAT3 (p-STAT3) levels as an indication of the activation of IL-6/JAK-STAT signalling pathway at different disease stages (early and established/late). The main aim is to evaluate the variation in cell-subset IL-6 signalling and its association with response to treatment which included IL-6 targeted therapy (Tocilizumab-TCZ) as well as other bDMARD.

**Methods** Phosphorylation of IL-6/JAK-STAT key transcription factor STAT3 (p-STAT3) was measured using multiparameter phosphoflow cytometry (phosflow) in T-, B- cells and monocytes isolated from peripheral blood of RA patients. Patients cohorts represented groups at different stages of RA: Treatment-naive Early RA (ERA group) n=20. Later RA group