**Background:** Clinically suspect arthralgia (CSA) can precede development of clinically evident inflammatory arthritis (IA). Autoantibody, C-reactive protein (CRP), and subclinical inflammation are known predictors, but risk estimation remains insufficiently accurate. Especially CRP has a small effect size and is inadequately reflective of inflammation that can be measured systemically. RNA expression in whole blood of patients with rheumatoid arthritis (RA) have shown differences compared to healthy individuals. Therefore, we hypothesized that differences in RNA expression can be found between CSA patients that do and do not progress to IA.

**Objectives:** This study assessed whole blood RNA expression levels of inflammatory and immune genes as potential biomarkers for prediction of IA-development in patients with arthralgia.

**Methods:** Between April 2012-March 2015, 234 patients were consecutively included in the Leiden CSA-cohort. Follow-up ended when patients developed clinically apparent IA (determined at physical examination), or else after 2-years. RNA expression in whole blood, at the moment of inclusion, was determined for 135 genes of the innate and adaptive immune system by dual color Reverse-Transcription Multiplex Ligation-dependent Probe Amplification (dCRT-MLPA) profiling. Cox proportional hazard models were used to associate time-to-event with gene expression level at inclusion, while adjusting for age, gender, and assay plate (model 1). The false discovery rate was used to correct for multiple testing. Genes with significantly different expression were subsequently studied for reproducibility by qPCR, and mutual independence in their association with IA-development. For the latter, we employed a forward selection strategy, starting with the most significantly associated gene and iteratively adding more genes. Resulting mutually independent genes were further investigated for their added predictive value over known risk factors, CRP, ACAP, and subclinical joint inflammation.

**Results:** 21% of CSA-patients developed IA after mean 3.6 months (IQ9:16; 10.7) follow-up. After correction for multiple testing, six genes were significantly associated with IA-development (model 1), namely IFN-γ, PHEX, IGF-1, IL7R, CD19, or CCR7 (ordered by significance). For all six genes, a lower expression at inclusion was associated with an increased risk of IA-development. IFN-γ was only weakly expressed in peripheral blood, hampering the technical reproducibility between MLPA and qPCR results, and was excluded for further analyses. PHEX and IGF-1 were highly correlated (R² 0.97) and only IGF-1, but not PHEX, was included in further analyses. Of the remaining significant genes (IGF-1, IL7R, CD19, CCR7), an independent association with IA-development was observed for IGF-1 and IL7R, but not for CD19 or CCR7. qPCR data of IL7R correlated with IA-development. For the latter, we employed a forward selection strategy, time to development of RA was predicted by the following questions: pain moving from joint to joint, having moderate or severe swelling in joints, feeling ≥1 days fatigue per month and feeling stiffness in joints of one and both feet (table 1).

**Disclosure of Interests:** None declared.

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