adjusting for covariates [HR 1.96 (95% CI: 1.18, 3.24) $p=0.01$]. Disability (SF-36 physical component), fatigue (FACIT-F) and mental health (SF-36 MHI) predicted flare in univariate models (figure 1). In multivariate analyses, only MHI score remained a statistically significant independent predictor of flare [HR per 10 units 0.74 (95% CI: 0.60, 0.93) $p=0.01$].

Conclusions: Baseline DAS28 and mental health predict flare events in patients in LDA who taper their anti-TNF agents. Other psychological and functional states, measured by patient-reported outcome do predict flare events although the effect size is small and does not persist when adjusting for known predictors. Based on these findings, an assessment of functional and mental health states should be considered prior to biologic tapering.

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THU0116 LIPID PROFILE AND CARDIOVASCULAR RISK IN SUBJECTS AT RISK FOR DEVELOPING RHEUMATOID ARTHRITIS

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Background: Rheumatoid arthritis (RA) is a systemic inflammatory autoimmune disease associated with an increased cardiovascular (CV) risk that is already present at the time of diagnosis. However, it is unclear at what point in the period before diagnosis of RA the CV risk increases. Therefore, we assessed the 10 year risk of CV morbidity and mortality in a cohort of subjects at risk for RA and analysed associations with anti-citrullinated protein antibody (ACPA) status and arthritis development.

Methods: In a cohort of 594 consecutive arthralgia patients with positivity for rheumatoid factor (RF) and/or ACPA, demographics, medical history, medication use and comorbidities were assessed. Lipid profile was determined and blood pressure was measured in a subset of patients. The 10 year CV risk score according to the Dutch CV risk management guideline (Dutch Systematic Coronary Risk Evaluations (SCORE)) was calculated for patients of whom data were complete.

Results: ACPA positive subjects ($n=382$) were younger (mean age 48.6 vs 51.6, $p=0.003$), more often smokers (31.8% vs 23.9%, $p=0.045$) and had lower cholesterol (mean level 5.2 vs 5.5, $p=0.001$) and HDL (mean level 1.1 vs 1.2, $p=0.004$) than ACPA negative subjects. Subjects who developed arthritis ($n=205$) had lower cholesterol (mean level 5.2 vs 5.4, $p=0.008$) and HDL levels (mean level 1.0 vs 1.2, $p=0.001$) and a higher TC/HDL ratio (median ratio 5.2 vs 4.8, $p=0.047$) compared to subjects who did not develop arthritis. Lower cholesterol (OR 1.24, CI 1.04–1.48), lower HDL (OR 2.01, CI 1.29–3.13) and higher TC/HDL ratio (OR 1.07, CI 1.00–1.138) predicted arthritis development.

The Dutch SCORE was calculated in 177 subjects (median 2, IQR 1–9). 76.4% had a low risk (SCORE <10%), 12.5% a medium risk (SCORE 10–<20%) and 11.4% had a high 10 year risk (SCORE >20%) of cardiovascular morbidity and mortality. The score was not associated with ACPA status or arthritis development.

Conclusions: Dyslipidemia as known in RA patients with active disease was also present in seropositive arthralgia patients at risk for RA, and predicted development of RA. However, arthralgia subjects who developed arthritis did not have a higher CV risk score than those who did not develop arthritis. Also, despite differences in lipid profile and smoking, the CV risk score does not differ between ACPA positive and ACPA negative subjects at risk for RA. Overall, differences in lipid profile predict development of RA but were too small to have an effect on the 10 year risk of CV morbidity and mortality as calculated by the Dutch CV risk score.

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THU0117 CHEMOKINE CCL18 ENRICHED IN SYNOVIAL FLUID IS INVOLVED IN JOINT DESTRUCTION THROUGH PROMOTING MIGRATION OF FIBROBLAST-LIKE SYNOVIOCYTES IN RHEUMATOID ARTHRITIS

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Background: CC chemokine ligand 18 (CCL18) which is either constitutively expressed or induced in monocytes/macrophages and dendritic cells has been reported to be highly expressed in peripheral blood and synovial fluid of rheumatoid arthritis (RA) patients compared with healthy controls, indicating the possible role of CCL18 in the development and pathogenesis of RA.

Objectives: To explore the association of serum and synovial fluid CCL18 with clinical and radiographic outcome in RA patients and its potential effect on RA fibroblast-like synoviocytes (FLS).

Methods: Consecutive patients with active RA (DAS28-CRP>2.6) were recruited. Synovial fluid was collected from inflamed joints if available. Demographic and clinical data were collected according to the 2017 EULAR recommendation. Serum and synovial fluid CCL18 was detected by ELISA. RA-FLS was cultured *in vitro* with RA synovial fluid and neutralising antibody to CCL18. Migration/invasion ability was analysed by Transwell assay.

Results: (1) Among 83 RA patients, age (median and IQR, similarly hereinafter) was 50 (41–58) years old and 65 patients (78%) were female, with median disease duration 36 (12–102) months, median DAS28-CRP 5.0 (4.6–6.1). (2) Serum CCL18 was 107 (80–126) ng/mL, which was significantly higher than healthy controls [$n=25$, 51 (29–70) ng/mL, $p<0.001$, figure 1A]. Serum CCL18 correlated slightly but significantly with CRP ($r=0.385$, $p<0.001$), ESR ($r=0.239$, $p=0.03$), PrGA ($r=0.249$, $p=0.03$), DAS28-CRP ($r=0.368$, $p=0.001$), DAS28-ESR ($r=0.336$, $p=0.003$), SDAI ($r=0.360$, $p=0.001$), CDAI ($r=0.328$, $p=0.004$) and HAQ ($r=0.325$, $p=0.004$). (3) Synovial fluid CCL18 of 31 patients was 719 (415–1271) ng/mL, which was significantly higher than corresponding serum level (Paired test, $p<0.001$, figure 1A). Among them, 13 patients who were treated according to treat to target strategy received X-ray assessment of hand/wrist both at baseline and month 12. Six patients who had one-year radiographic progression (a change of the Sharp/van der Heijde modified sharp score ≥ 0.5 units) showed higher synovial fluid CCL18 than other 7 patients without radiographic progression [1481 (1244–2034) ng/mL vs. 458 (405–681) ng/mL, $p=0.004$, figure 1B). (4) When incubated with RA synovial fluid, the migration ability of RA-FLS was significantly increased; but this effect was inhibited by neutralising antibody to CCL18 (figure 1C).

Abstract THU0117 – Figure 1. Chemokine CCL18 which was enriched in RA synovial fluid promoted migration of fibroblast-like synoviocytes in Rheumatoid Arthritis (RA), but was inhibited by neutralising antibody to CCL18. A: Comparison of RA synovial fluid CCL18 with serum CCL18 of RA patients and health controls. B: Comparison of RA synovial fluid CCL18 between patients with and without one-year radiographic progression. C: Transwell assay, crystal violet staining for RA-FLS (purple), with original magnification of $\times 100$.

** $p<0.001$, *** $p<0.0001$