provide important insights into divergent and shared mechanistic features of RA and serve as a template for future studies to guide drug target discovery by synovial molecular signatures and de-sign stratified approaches for patients with RA.

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Figure 1 Identification of RA patients using non-negative matrix factorization, functional enrichment, and immune cell infiltration in the differentially expressed genes set. (A-D) The consensus map of NMF clustering results in the total RNA sequencing dataset. Patients were divided into cluster 1-5 according to the expression profiles of 576 DEGs. Heatmap of five clusters defined by the cluster-based genes expression. (E-F) Gene Ontology (GO) terms and KEGG pathway related to common cluster-based gene signatures. (G-H) Cell subset enrichment score according to RA subgroup. (I-J) Distribution of the five RA subgroups by clinical phenotypes (the positivity of ACPA). (ns, not significant; * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$; **** $P < 0.0001$)

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OP0185

RADIOGRAPHIC PROGRESSION DESPITE PERSISTENT LDA OR REMISSION IS INFLUENCED BY CURRENT SMOKING RATHER THAN THE RESPECTIVE DAS 28 LEVEL, RESULTS OF THE SWISS RHEUMATOID ARTHRITIS REGISTER (SCQM)

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Background: The therapeutic aim for rheumatoid arthritis (RA) is to control disease activity and prevent radiographic progression. Various clinical scores are utilized to describe disease activity in RA patients. The DAS28 score can define states of low disease activity (LDA) and remission. Despite achieving LDA or remission, radiographic progression may nevertheless occur. However, the rates and frequency of this occurrence have not been analyzed in detail.

Objectives: To describe the frequency and rate of radiographic progression in patients with persistent LDA or remission.

Methods: Analysis of RA patients from the SCQM cohort. Persistent LDA or remission were defined as DAS 28 ≤ 3.2 or < 2.6 respectively, at two subsequent follow up time points in the database. We included patients with at least two sets of radiographs within these intervals of LDA and/or remission. Radiographic progression was measured with the Raitingen-score (range 0-190), which describes joint erosions numerically. Repair was defined as an improvement in the Raitingen score > 5 points/year and progression as > 2 or > 5 points change in the Raitingen score within one year.

Results: Among 10'141 RA patients, 4'342 episodes of remission occurred in 3'927 patients with 1'776 sets of X rays available within these episodes. Similarly, 8'136 episodes of LDA in 6'765 patients and 2'358 sets of X rays were present within these intervals. For patients in LDA or remission, rates of repair were 5.5% and 4.8%, respectively, while for radiographic progression > 5 points in the Raitingen score/year were 10.3% in both groups and for > 2 points change of Raitingen score/year were 27.7 and 25.4%, respectively.

No differences for demographic factors or measures of disease activity, rheumatoid factor or ACPA were found comparing patients with radiographic progression or non-progression despite LDA or remission at the beginning of the episode of LDA and/or remission.

Interestingly, 42.9% of patients in LDA with progression of > 5 points in the Raitingen score/year were current smokers vs 29.4% among the non-progressors ($X^2 = 6.55$, $p = 0.01$). This significant difference vanished when the cut-off for radiographic progression was set at > 2 points yearly change in Raitingen score or in patients in remission.

Conclusion: Radiographic progression despite LDA or remission are more frequent than expected. No differences in radiographic progression were found comparing LDA and remission suggesting that the goal of LDA is appropriate. Smoking seems to be an independent risk factor for radiographic progression despite LDA. Why the effect of smoking could was not demonstrated in patients in remission, remains unclear.

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OP0186

CHANGES IN CIRCULATING B CELL LEVELS AND IMMUNOPHENOTYPE ARE ASSOCIATED WITH DEVELOPMENT OF ARTHRITIS FOLLOWING TREATMENT WITH CHECKPOINT INHIBITORS

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