Results: 75 pts with chest CT scans were included in the analysis. Of these, 38.7% (n=29) were non-ILD RA and 61.3% (n=46) had some manifestation of RA-ILD. Of the RA-ILD cohort, 63.0% (n=29) and 37.0% (n=17) were classified as subclinical and clinically evident RA-ILD, respectively. At the time of chest CT scan, RA-ILD (vs non-ILD RA) pts were older with longer disease duration, a greater proportion were male and had higher anti-citrullinated protein antibody and RF titres (Table 1). In terms of RA treatments, a significantly greater proportion of RA-ILD (vs non-ILD RA) pts were on corticosteroids (CS; 47.8% vs 20.7%) and a significantly greater proportion of clinically evident RA-ILD (vs non-ILD RA) were on non-TNF inhibitor (TNFi) biologics. RA-ILD (vs non-ILD RA) pts had numerically higher joint disease activity and modified HAQ score at baseline (Table 1). However, the change in joint disease activity and PROs in RA-ILD pts was numerically greater vs non-ILD RA pts. In a multivariable analysis, RA-ILD status did not impact change in joint disease activity, but baseline joint disease activity was significantly associated with reduction in joint disease activity at 12 months (Table 2).

Conclusion: RA-ILD pts compared with non-ILD RA pts in clinical practice are more likely to be managed with CS therapy and non-TNFi bDMARDs. The improvement in joint disease activity was similar between RA-ILD and non-ILD RA pts. The analysis was limited by small sample size; further studies with larger pt numbers are required to confirm the findings.

Table 2. Multivariable analysis of change in disease activity (CDAI)

Parameter	Estimate	95% CI
Intercept	0.8	(-12.5, 14.0)
No ILD (vs ILD)	-3.9	(-12.8, 5.1)
RA duration, years	-0.1	(-0.5, 0.3)
Baseline CDAI score*	0.4	(0.0, 0.7)
No CS (vs CS)	-3.6	(-13.5, 6.2)
*p<0.05 vs no ILD CS=corticosteroids; ILD=interstitial lung disease		

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SAT0073

STAT3/STAT5 BALANCE AS A BIOMARKER IN RA: CTLA4-IG AND T CELL DIFFERENTIATION THROUGH STAT SIGNALING

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Background: Regulatory T cells (Treg) play suppressive functions and are modulated by Abatacept (CTLA4-Ig). Limited data are available on CTLA4-Ig effect on Treg population in Rheumatoid Arthritis (RA).

Objectives: The aim of the study was to analyze if STAT3/STAT5 expression in CD4+ T cells in peripheral blood (PB) of RA patients at baseline predicts response to Abatacept treatment (after 12 months follow-up).

Methods: Early RA (ERA) and long-standing RA (LS-RA) patients with conventional DMARDs insufficient response were enrolled in this observational, investigative, monocentric, non-randomized, no profit study, and treated with CTLA4-Ig in combination with methotrexate. Each enrolled RA patients underwent peripheral blood sampling and CD4+ cells isolation using magnetic micro-beads at baseline and after 6-12 months follow-up. Flow cytometric analysis (FACS) for CD4 positive cells phenotype was performed to assess T-regulatory cells (Treg) as CD4+/CD25+/CD127- and CD4+/CD25+/Foxp3+, respectively. STAT3/STAT5 gene expression on CD4+ cells was performed by RT-PCR for each enrolled patient at every time-point follow-up. Low disease activity (LDA) and disease remission (DAS) achievement were assessed at 6 and 12 months follow-up (FU), respectively.

Results: A total of 35 patients were enrolled in the study (16 ERA and 19 LS-RA, respectively). At baseline, ERA and LS-RA did not differ based on clinical parameters. Eight (22.9%) withdrew from the study because of treatment failure (n=6), severe infection (n=1) and death (n=1). LDA or DAS remission within twelve months follow-up were achieved in 28/34 (82.4%) and 16/34 (47.1%) patients, respectively, without any significant difference among ERA and LS-RA. There were no significant differences in the demographic and clinical characteristics of RA patients at study based on LDA or DAS remission status achievement within 12 months FU, even stratifying patients based on disease duration. FACS analysis showed CD4+/CD25+/CD127- and CD4+/CD25+/Foxp3+ cells decrease during CTLA4-Ig treatment (p=0.01 and p=0.02, respectively after 12 months FU), despite disease duration. RT-PCR revealed that PB CD4+ cells of RA patients achieving LDA, but not DAS remission, after CTLA4-Ig treatment have significantly lower endogenous expression of STAT3 and STAT5 compared to RA patients not achieving this outcome (p=0.03 and p<0.001, respectively). Moreover, baseline STAT3/STAT5 ratio in PB CD4+ cells of RA patients directly correlates with Treg cells percentages (CD4+/CD25+/CD127- cells (%): R=0.518, p=0.03; CD4+/CD25+/Foxp3+ cells (%): R=0.549, p=0.02, respectively). Finally, baseline STAT3/STAT5 expression ratio on CD4+ cells > 0.93 (obtained by ROC analysis: AUC:0.754+/-0.100; Sensitivity 75.0%, Specificity: 80.0%) arose as baseline predictor factor of LDA achievement in RA patients treated with CTLA4-Ig [OR(95%CI): 12.0 (1.98-72.89)].

Conclusion: STAT3/STAT5 expression ratio in T cells at baseline identify RA patients better responding to CTLA4-Ig, which decreases Treg cells.

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SAT0074

DAS-28 DISEASE ACTIVITY DEFINES THE TNFR1/2 CO-EXPRESSION PROFILE IN RHEUMATOID ARTHRITIS PATIENTS

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Background: TNF-alpha act as main proinflammatory cytokine in rheumatoid arthritis (RA) immune processes. However, TNF-alpha activity and functions may be regulated not only by soluble receptors (which act as decoys) but also by number, density, and co-expression of its membrane-bound receptors type 1 and 2 (TNFR1 and TNFR2).

Objectives: To analyze the TNFR1/2 co-expression profile in RA patient with different disease activity in comparison to healthy donors (HD).

Methods: PBMC were analyzed from 46 HD and 64 patients with RA using flow cytometry. Patients were divided according to the DAS-28 index into groups with high (n=22, 34.4%), moderate (n=30, 46.9%) and low (n=12, 18.8%) disease activity. Co-expression of TNFR1/2 was evaluated as percentage of cell with different receptors. Number of receptors of each type per cell was counted using QuantiBrite PE beads (BD, USA). The following populations were analyzed: total monocyte pool; common pool of B lymphocytes; common pool of T lymphocytes; cytotoxic T cells (CD8+), T helper cells (CD4+), activated (CD25+) cells among CD8+ and CD4+, T memory (CD45RO+) and naïve T cells