REFERENCES:

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Table 1. Absolute counts of T-cell subpopulations at baseline, after 6 and 12 m of TCZ therapy

<table>
<thead>
<tr>
<th>Cell Type</th>
<th>Donors</th>
<th>Baseline</th>
<th>6m</th>
<th>12m</th>
</tr>
</thead>
<tbody>
<tr>
<td>T (CD3+)</td>
<td>1.3 (1.2-1.8)</td>
<td>1.2 (0.9; 1.7)</td>
<td>1.1 (0.7; 1.4)</td>
<td>1.0 (0.7; 1.5)</td>
</tr>
<tr>
<td>Th (CD3+CD4+</td>
<td>0.9 (0.7-1.2)</td>
<td>0.8 (0.6; 1.0)</td>
<td>0.7 (0.5; 1.0)</td>
<td>0.6 (0.4; 1.1)</td>
</tr>
<tr>
<td>Tc (CD3+CD8+)</td>
<td>0.4 (0.3-0.5)</td>
<td>0.4 (0.2-0.5)</td>
<td>0.3 (0.2; 0.4*)</td>
<td>0.3 (0.2; 0.5)</td>
</tr>
<tr>
<td>T (CD3+)</td>
<td>2.5 (1.9-3.1)</td>
<td>2.3 (1.4; 3.0)</td>
<td>2.3 (1.5; 3.0)</td>
<td>2.1 (1.4; 4.0)</td>
</tr>
<tr>
<td>NK (CD3-CD56+)</td>
<td>0.2 (0.2-0.5)</td>
<td>0.2 (0.1; 0.2)</td>
<td>0.2 (0.1; 0.3)</td>
<td>0.2 (0.1; 0.3)</td>
</tr>
</tbody>
</table>

Note: *- p<0.05 between pts at baseline and after 12 m of TCZ therapy; **- p=0.02 between pts at baseline and after 6 m of TCZ therapy; ***- p<0.05 between donors and pts after 12 m of TCZ therapy.

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POS623 CYTOKINE PRODUCTION BY BLOOD LYMPHOCYTES DEFINES A PROFILE ASSOCIATED WITH NON-REMISION IN PATIENTS WITH RHEUMATOID ARTHRITIS TREATED WITH TNF INHIBITORS

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Background: In clinical practice no more than 50% of the patients treated with TNF inhibitors (TNFi) achieve remission (REM). Previous investigations suggested that peripheral blood mononuclear cells (PBMC) may be markers associated with the TNFi treatment success.

Objectives: This study aims to analyse the intracellular cytokine production by PBMC and its association with REM achievement after 6 months (m) of TNFi treatment in patients with RA.

Methods: This was a prospective study including 62 patients with RA starting the 1st TNFi. PBMC were isolated from patients at baseline and after 6 m of treatment with TNFi and cryopreserved until studied. In vitro stimulation and intracellular cytokine production by PBMC was performed as follow: in the presence of 2µg/mL brefeldin and 2µmol/L monensin monocytes were stimulated with 50ng/mL LPS during 4h whereas lymphocytes were stimulated with 50ng/mL phorbol 12-myristate 13-acetate and 750ng/mL ionicycin for 4h at 37ºC. To identify IL10-producing B cells, PBMC were pre-incubated with 3µg/mL anti-CD19, then with 2µg/mL anti-CD16 and 2µg/mL anti-CD7 and a 24h stimulation. Intracellular cytokine production (TNFα, IL6, GM-CSF, IL10) by the different cell subsets (monocytes, CD4+ and CD8+ T cells, naïve and memory B cells) was analysed by flow-cytometry. Clinical activity at baseline and after 6m was assessed by DAS28-ESR. REM was defined as DAS28<2.6 at 6m. The association between cytokine production by each PBMC subset and REM was assessed through univariable and multivariable logistic regression models. Receiving operating curve (ROC) analysis was used to select the optimal ratio of cytokine production associated with REM status.

Results: After 6m of TNFi treatment, 30 (48%) patients achieved REM. No significant differences between REM and non-REM groups were observed for patients' characteristics at baseline except for DAS28, which was lower in the REM group (non-REM: 5.4±0.9; REM: 4.3±0.9; p<0.0001) (Table 1). Therefore, further analysis were adjusted by baseline DAS28. A lower ratio between calculated with the IL10 and TNFα production by B cells and by CD4+ T cells (IL10/B TNFα CD4) at 6m was found for non-REM patients (non-REM: 0.31 vs REM: 0.54; p=0.007). Based on a ROC analysis, we found that a (IL10/B TNFα CD4)<0.54 at 6m was