Conclusion: In our trial of elderly rheumatoid arthritis patients, patients appeared to be mostly adherent according to conventional capsule counts. Results from adherence caps were highly discrepant with the capsule counts, with patterns suggesting patients did not use the bottle for daily dispensing, despite specific advice to do so.

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FRI0582

GM-CSF PRODUCED BY CD4+ T CELLS AS A MARKER OF CLINICAL REMISSION IN PATIENTS WITH RHEUMATOID ARTHRITIS TREATED WITH TNF INHIBITORS

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Background: According to the EULAR recommendations, the therapeutic target in patients with RA should be remission (REM). However, no more than 50% of the patients treated with TNF inhibitors (TNFi) attains this outcome. Previous investigations suggested the peripheral blood mononuclear cells (PBMC) as markers associated with the TNFi treatment success^{1,2}. Granulocyte-monocyte colony-stimulating factor (GM-CSF) plays a relevant role in the pathogenesis of rheumatoid arthritis (RA) because it promotes the macrophage differentiation, survival and activation³.

Objectives: To analyse the intracellular cytokine production by PBMC and its association with REM attainment after 6 months (m) of TNFi treatment in patients with RA.

Methods: This was a prospective bi-center pilot study including 36 patients with RA. PBMC were isolated from patients at baseline and after 6m of treatment with TNFi and cryopreserved until studied. Intracellular cytokine production by PBMC was stimulated in the presence of 2µg/mL brefeldin as follow: monocytes were stimulated with 20ng/mL LPS during 4h; and simultaneously lymphocytes were stimulated with 50ng/mL phorbol 12-myristate 13-acetate (PMA) and 750ng/mL ionomycin during 4h at 37°C. To identify IL-10-producing B cells, PBMC were pre-incubated with 3µg/mL of CpG oligonucleotide during 20h at 37°C prior to stimulation in presence of 2µmol/L monensin. 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. REM was defined as DAS28≤2.6 at 6m. The association between REM and the change in cytokine production (Δ, 6m-0m) by each PBMC subset was analysed through univariable and multivariable logistic regression models.

Results: Seventy-eight percent of the patients were female. After 6m of TNFi treatment, 47% patients attained REM. Univariable analyses was performed to investigate the association between REM and the baseline variables. Male sex (OR: 12.6; 95% CI: 1.35-117.57; p=0.03) and having lower baseline DAS28 (OR: 0.4; 95% CI: 0.19-0.85; p=0.02) were independently associated with attaining REM after 6m of TNFi. In the multivariable analysis, only being male (OR: 19.7; 95% CI: 1.4-273.9; p=0.03) remained independently associated with REM after 6m of treatment. Therefore, further analyses were adjusted by sex. Decreased production of GM-CSF by CD4+ T cells percentage was found after 6m of TNFi treatment in REM patients (0m: 6.07%; 6m: 3.87%; p=0.007) while no-REM patients did not show differences with the baseline (0m: 3.70%; 6m: 3.75%; p=0.9). The decrease was significantly associated with attaining REM (OR: 0.56; 95% CI: 0.33-0.95; p:

0.03). No significant association was found between any other analysed intracellular cytokine produced by the different PBMC subsets and REM.

Conclusion: GM-CSF intracellular production by CD4+ T cells was significantly decreased by TNFi treatment only in patients who attained REM. Therefore, our results suggest that GM-CSF production by CD4+ T cells may be a useful marker of REM to TNFi in RA.

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Figure 1: Association between the change in intracellular cytokine production (Δ, 6m-0m) by each PBMC subset and REM. Adjusted logistic regression analyses were performed for each cytokine.

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FRI0583

VALIDATION OF THE MODIFIED FATIGUE IMPACT SCALE IN DANISH PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: Patients with systemic lupus erythematosus (SLE) experience significant fatigue, a debilitating symptom associated with reduced quality of life. A simple, reliable multidimensional method for assessing fatigue has not yet been validated for Danish patients with SLE.

Objectives: The primary objective was to study the internal consistency, test-retest reliability, and construct validity (convergent and discriminant validity) of the multidimensional Modified Fatigue Impact Scale (MFIS) in patients with SLE. The secondary objective was to investigate the contribution of disease activity and organ damage to fatigue.

Methods: Data from the "Bio and Genome Bank Study in Centre for SLE and Vasculitis" obtained through routine visits were used. Fatigue was assessed using the MFIS and Short Form 36 (SF36). Internal consistency of the MFIS was assessed with Cronbach's alpha (α). Test-retest reliability was evaluated using the intraclass correlation coefficient (ICC). Construct validity was studied using Spearman's rank correlation coefficient (r_s) and Principal Component Analysis (PCA) between MFIS and SF36 vitality (VT-SF36) and mental health (MH-SF36) subscales. Association between MFIS and disease activity and organ damage was estimated with Spearman's rank correlation coefficient.

Results: The study included 30 patients with SLE. Internal consistency of the MFIS was excellent with Cronbach's $\alpha = 0.97$ for the complete scale. Excellent test-retest reliability was found with ICC = 0.95 (95% confidence interval: 0.88–0.98, $p < 0.05$). Construct validity was confirmed by Spearman's correlation (VT-SF36: $r_s = -0.73$, $p < 0.001$ (Fig. 1). MH-SF36: $r_s = -0.74$, $p < 0.001$ (Fig. 2)) and PCA with explained variance from the first two principal components (PC) (VT-SF36: PC1 = 60.2%, PC2 = 8.5%. MH-SF36: PC1 = 58.5%, PC2 = 7.4%). No significant correlation was found between the MFIS and SLEDAI ($r_s = 0.04$, $p = 0.84$) or SLICC Damage Index ($r_s = 0.32$, $p = 0.08$).

Figure 1. Scatter plot of the Modified Fatigue Impact Scale (MFIS) and the Short Form 36 vitality (VT-SF36) subscale.

Figure 2. Scatter plot of the Modified Fatigue Impact Scale (MFIS) and the Short Form 36 mental health (MH-SF36) subscale.

Conclusion: The present study found the multidimensional assessment of fatigue with MFIS to be a reliable and valid instrument in SLE. The MFIS might provide more detailed information about fatigue in future studies. In agreement with some previous studies we found no association between fatigue and SLEDAI or SLICC which raises

questions about the cause of this symptom. Further and larger studies are needed to investigate if any association between fatigue and disease components exist.

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FRI0584 SCREENING AND IDENTIFICATION OF BIOMARKERS IN MYOSITIS

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Background: Myositis, including dermatomyositis and polymyositis, is autoimmune disorders that is characterized by muscle degeneration in the proximal extremities, with the complications of weakness of muscles, interstitial lung disease and vascular lesions, even leading to death in an acute progressive process^[1,2]. However, the molecular mechanisms of myositis are rarely understood.

Objectives: Identify the candidate genes in myositis.

Methods: Microarray datasets GSE128470, GSE48280 and GSE39454 were extracted from the Gene Expression Omnibus (GEO) database. The differentially expressed genes (DEGs) and function enrichment analyses were conducted. The protein-protein interaction network and the analyses of hub genes were performed with STRING and Cytoscape.

Results: There were 98 DEGs, of which the function and pathways enrichment analyses showed defense response, immune response, response to virus, inflammatory response, response to wounding, cell adhesion, cell proliferation, cell death and macromolecule metabolic process. 20 hub genes were identified, of which 7 including IRF9 TRIM22 MX2 IFITM1 IFI6 IFI44 IFI44L had not been reported in the literature, related to the response to virus, immune response, transcription from RNA polymerase II promoter, cell apoptosis, cell death. The verification analysis about the 7 genes in GSE128314 showed significant differences in myositis.

Conclusion: In conclusion, DEGs and hub genes identified in our study showed the potential molecular mechanisms in myositis, providing the helpful targets for diagnosis and clinical strategy of myositis.

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Figure 1. The protein-protein interaction network of 20 hub genes

Figure 2. 7 genes in GSE128314 showed significant differences in myositis