

**Results:** The mean age of our population was 49.17±11.21 years (age 24–78). The disease average duration was 7.44±2.12 years (4 months - 29 years), 82.71% of RA were women and 17.29% were men. Seropositive RA were 80.24%, and 71% of RA have anti CCP positive antibody. Univariate analysis of the presence of anti-CCP antibodies in conjunction with HLA DRB1 and DQB1 was performed. Carriership of HLA DR\*0301, 0401 and 1501 were significantly associated with the presence of anti-CCP antibodies ( $p < 0.0001$ ). Four DRB1 0401 carriers were homozygotes with three out of them having anti-CCP antibodies. Carriership of HLA DQB1\*0201, 0301, 0302,0501 and 0601 was associated with the presence of anti-CCP antibodies and so was HLA-DQB1\*0401, but with a less significant association.

**Conclusions:** Although no formal conclusions on causality can be drawn from this association study, these findings suggest that anti-CCP antibodies are associated with different phenotypes; which suggest that various pathogenetic mechanisms underlie the positivity for anti-CCP in RA.

**Disclosure of Interest:** None declared  
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### AB0281 CHARACTERIZATION OF CHANGES IN LYMPHOCYTE SUBSETS IN BARICITINIB-TREATED PATIENTS WITH EARLY, DMARD NAÏVE, RHEUMATOID ARTHRITIS IN A PHASE 3 STUDY

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**Background:** In RA-BEGIN (NCT01711359), baricitinib (bari), an oral Janus Kinase (JAK)1/JAK2 inhibitor, improved signs and symptoms of moderately to severely active RA in patients (pts) who had received no or limited prior csDMARD and no prior bDMARD therapy.<sup>1</sup>

**Objectives:** To analyse changes in absolute lymphocyte count (ALC) and cell subsets (LCS) in RA-BEGIN.

**Methods:** Pts (N=588) were randomised 4:3:4 (MTX up to 20mg QW, bari 4mg QD, bari 4mg+MTX) for 52 Wks. T and B cells plus subsets and natural killer (NK) cells were quantified by flow cytometry at baseline (BL) and Wks 4, 12, and 32.

**Results:** At BL, low cell counts were observed in 4.3%, 8.2%, 5.3%, 18.3%, and 19.7% of pts for ALC, CD3+, CD8+, B (CD19+), and NK cells. In the MTX group, slight declines in mean counts were observed for all cell types at post-BL visits (Table). For bari and bari+MTX, cell counts increased for all cell types at Wks 4 and 12, with, except for B cells, a return towards BL, or slightly below, at Wk 32. Changes in other T and B cell subsets generally reflected these patterns (data not shown). Treatment emergent (TE) low NK cell counts occurred in 11.6%, 13.4%, and 20.5% of pts for MTX, bari, and bari+MTX; TE low CD8+ cell counts occurred in 5.2%, 3.2%, and 8.3% of pts. Overall serious infection (SI) rates were 3.8%, 3.8%, and 2.3% for all pts in MTX, bari, and bari+MTX; rates were 6.8% (4 of 59 pts), 4.8% (2 of 42), and 2.8% (2 of 72) for pts with  $\geq 1$  low post-BL NK cell count and 15.8% (3 of 19), 25.0% (2 of 8), and 5.3% (1 of 19) for pts with  $\geq 1$  low post-BL CD8+ cell count. Herpes zoster (HZ) rates were 1.0% 2.5%, and 2.3% for all pts in MTX, bari, and bari+MTX; rates were 3.4% (2 of 59 pts), 4.8% (2 of 42), and 4.2% (3 of 72) for pts with  $\geq 1$  low post-BL NK cell count and 10.5% (2 of 19), 12.5% (1 of 8), and 5.3% (1 of 19) for pts with  $\geq 1$  low post-BL CD8+ cell count.

	MTX (N=210)				Bari 4 mg (N=159)				Bari 4 mg + MTX (N=215)			
	Week: 0	4	12	32	0	4	12	32	0	4	12	32
ALC:	1890	-110*	-110*	-80	1920	310***	380***	-20	1940	410***	220***	-100*
CD3+:	1384	-108***	-98.5***	-102.5***	1369	148***	207***	-78*	1403.5	210***	90**	-150***
CD8+:	435	-36**	-45***	-48***	422	52***	69***	-34**	435	80.5***	27*	-63***
CD19+:	261	-17.5	-10	-13	258	103***	113***	51***	269	119***	101***	46***
NK:	218	-11	-25**	-23**	247	74***	49***	-24**	234	64***	14	-44***

Data are mean (Wk 0) and LSM  $\Delta$  from BL (Wks 4, 12, 32), last observation carried forward. \* $p < 0.05$ , \*\* $p \leq 0.01$ , \*\*\* $p \leq 0.001$  within grp comparison, LSM  $\Delta$  from BL. Reference ranges (cells/ $\mu$ L): ALC=800–4280; CD3+=603–2990; CD8+=125–1312; CD19+=107–698; NK=95–64.

**Conclusions:** Low B and NK cell counts were common at BL, and post-BL changes within normality occurred in all treatment groups. Compared to MTX, bari was not associated with an increase in the % of pts with low NK or CD8+ cell counts while bari+MTX did show an increase in the % of pts with a low NK cell count. Changes appear distinct for LCS suggesting different mechanisms may underscore the effect of JAK inhibition. Whether low NK or CD8+ cell counts predispose to increased risk for SI or HZ was difficult to assess due to few pts with low counts experiencing these events.

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Genentech, GSK, Janssen, Pfizer, Merck, Regeneron, Roche, Sanofi-Aventis, UCB, Consultant for: Abbvie, Akros, Amgen, Bristol-Myers Squibb, Celgene, Eli Lilly and Company, Genentech, GSK, Janssen, Pfizer, Sanofi-Aventis, UCB, M. Schiff Consultant for: Abbvie, BMS, Eli Lilly and Company, Johnson & Johnson, Speakers bureau: Abbvie, M. Issa Employee of: Eli Lilly and Company, W. Macias Employee of: Eli Lilly and Company, T. Rooney Employee of: Eli Lilly and Company, S. Zuckerman Employee of: Eli Lilly and Company, D. Schlichting Employee of: Eli Lilly and Company, I. McInnes Grant/research support from: Eli Lilly and Company, Abbvie, Pfizer, Novartis, Roche, Janssen, Consultant for: Eli Lilly and Company, Abbvie, Pfizer, Novartis, Roche, Janssen  
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### AB0282 RHEUMATOID ARTHRITIS PATIENTS ACHIEVED BETTER QUALITY OF LIFE THAN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS AT SUSTAINED REMISSION: THE IMPACT OF DISEASE DIAGNOSIS ON HEALTH-RELATED QUALITY OF LIFE OUTCOMES

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**Background:** Systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA) impact the health related quality of life (HRQoL) of the patients. The 36 item Medical Outcome Study Short-Form survey (SF-36) assesses HRQoL and allows comparison of outcomes among different conditions. Whether remission represents similar status in terms of QoL in RA and SLE patients is unknown. In 2004 and 1999, respectively, recent-onset RA and SLE cohorts were initiated in a referral center for rheumatic diseases in México City; the SF-36 was applied beginning from enrollment.

**Objectives:** To compare the SF-36v2 scores between patients from both cohorts who achieved for the first time sustained remission (SR) and to define the role of disease diagnosis as associated to SF-36v2 normative data in SR patients.

**Methods:** First SR was considered when RA and SLE patients achieved at least 12 months of continuous follow-up with either SLE disease activity index 2000 update =0 or Disease Activity Score (28 joints)  $\leq 2.4$ , respectively. Up to December 2015, updated data from 172 RA patients and 211 SLE patients with at least one year of follow-up were reviewed. In the SLE cohort, SF-36 was incorporated to routine assessments from 2005 onwards, meanwhile in the RA cohort it was applied since the beginning of enrollment. The SF-36v2 licensee re-scored the SF-36 used in the SLE cohort. In all the cases, Spanish versions were used and scoring was adjusted by gender and age. SF-36v2 scores were available for the totality of SR assessments. Logistic regression models were used to investigate factors associated with normative SF-36v2. Written informed consent was obtained from all patients.

**Results:** Cohorts were integrated primarily by middle-aged females (89%), with recent-onset disease (5.3±3.2 months); at inclusion, RA patients were older and lesser educated; follow-up was longer in SLE patients (10.6±2.9 vs. 7.5±3.2 years,  $p \leq 0.001$ ) and a higher number of them died (15% vs. 2%,  $p \leq 0.001$ ).

A higher proportion of patients achieved SR sooner in the recent-onset RA cohort than in the SLE cohort: 58% vs. 30.6% of the patients, after 30.8±23.9 vs. 59.4±37.5 months, respectively,  $p < 0.001$ . At SR, RA patients achieved better scores in 6 out of 8 SF-36v2 domains and in the physical health component summary (PHCS) compared with SLE patients; also, a greater proportion of RA patients achieved norms in five domains and in the PHCS; SLE patients achieved more frequent roles (physical and emotional) norms and scored higher mental health component summary than their counterpart. Finally, at SR RA patients had greater improvement in the majority of SF-36v2 domains and both summary components, despite having worse SF-36v2 scores at baseline evaluation. In SR patients from both cohorts, age ( $\beta$ : 1.06, 95% CI: 1.02–1.1,  $p = 0.03$ ) and SLE diagnosis ( $\beta$ : 9.64, 95% CI: 3.61–25.75,  $p \leq 0.001$ ) were predictors of not achieving normative PHCS.

**Conclusions:** RA patients who achieved SR had better HRQoL than their SLE counterparts. Age and SLE diagnosis were associated with not achieving HRQoL norms in SR patients.

**Disclosure of Interest:** None declared

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### AB0283 ONE-YEAR FOLLOW-UP OF QUALITY OF LIFE IN RHEUMATOID ARTHRITIS PATIENTS FROM BULGARIAN POPULATION TREATED WITH CSDMARDS AND BDMARDS

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**Background:** Rheumatoid arthritis is a chronic systemic disabling condition associated with pathology mainly of the peripheral joints. Quality of Life (QoL) encompasses the impact of the disease and its treatment on the ability of the patient to fulfill/satisfy his or her needs. Assessing QoL in RA is an attempt to ensure the concern of patients that this important aspect of their daily lives will be measured.

**Objectives:** To evaluate the change of QoL of Bulgarian patients with RA after 6 months to 1-year of treatment with csDMARDs and bDMARDs by using two

generic questionnaires (HAQ-DI and SF-36) and a disease-specific measurement RAQoL.

**Methods:** A total of 220 patients with a mean age  $55.05 \pm 10.63$ SD meeting the 1987 ACR classification criteria for RA were included in the study. The mean disease duration was  $9.97 \pm 5.78$ SD. Patients were stratified according to treatment regimens into 2 age-matched treatment groups: 96 on csDMARDs and 124 on bDMARD therapy. Subjects with significant comorbidity, infectious disease, congestive heart failure (NYHA class III or IV), malignant hypertension, psychiatric illness, a history of lymphoproliferative disease or neoplasia were excluded from the study. All participants completed the HAQ-DI, SF-36v2TM and RAQoL at baseline, at months 6 and 12 thereafter. The scores of the three instruments were calculated via licensed calculator. Comparison was performed by analysis variance ANOVA.

**Results:** At baseline the mean scores of HAQ-DI and RAQoL did not differ greatly among patients on csDMARDs and bDMARDs ( $1.29 \pm 0.78$ SD vs  $1.13 \pm 0.54$ SD,  $p=0.063$ ;  $16.31 \pm 8.26$ SD vs  $15.03 \pm 7.13$ SD,  $p=0.219$ , respectively). However, the mean physical component summary score of SF-36 was significantly higher in bDMARDs compared with csDMARDs ( $32.98 \pm 5.97$  vs  $31.05 \pm 7.82$ ,  $p=0.039$ ), while in the mental component of this scoring system not such a difference was found ( $p=0.983$ ). After 6 months subjects treated with bDMARD showed a significant decreasing of the means of the HAQ-DI and RAQoL, as opposed to the other treatment group ( $0.86 \pm 0.55$ SD vs  $1.17 \pm 0.76$ SD,  $p<0.001$ ;  $10.98 \pm 6.53$ SD vs  $14.55 \pm 7.96$ SD,  $p<0.001$  respectively). Similar results were obtained for both physical and mental component summary scores of SF-36 ( $39.49 \pm 6.43$ SD vs  $33.48 \pm 8.04$ SD,  $p<0.001$ ;  $43.69 \pm 7.99$ SD vs  $39.66 \pm 10.19$ SD,  $p=0.001$  respectively). At month 12 a significant improvement of QoL, measured by the three assessment tools was registered in patient receiving bDMARDs compared with the csDMARD treatment group ( $p<0.001$ ).

**Conclusions:** Patient treated with bDMARDs showed better results for QoL than those on therapy with csDMARDs within a period of 12 months of treatment. Current management strategies should focus on improving the symptoms of activity and maintaining physical function in order to increase QoL in patients with RA.

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**AB0284 ASSESSMENT OF DISEASE ACTIVITY BY DAS28-CRP, CDAI, SDAI AND RESPONSE TO TREATMENT WITH CSDMARDs AND bDMARDs AFTER ONE-YEAR FOLLOW-UP IN RHEUMATOID ARTHRITIS PATIENTS FROM BULGARIAN POPULATION**

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**Background:** The assessment of disease activity is an essential component in the selection of therapeutic approach for the prevention of disability of patients with RA.

**Objectives:** The current study was conducted to evaluate the disease activity in patients on csDMARDs and bDMARDs after 6 months to 1-year of treatment and to determine whether the benefits of different therapies were sustained over time.

**Methods:** For the purpose of the study were selected 220 patients with a mean age  $55.05 \pm 10.63$ SD years, meeting the 1987 ACR classification criteria for RA. Patients were stratified according to treatment regimens into 2 age-matched treatment groups: 96 on csDMARDs and 124 on bDMARD therapy. Patient's assessment of disease related pain, global health and physician assessment of global health was made by visual analogue scale (VAS) – 100mm. Disease activity was the primary outcome domain. Independent joint assessor evaluate 28 joints on baseline, 6th and 12th month of the follow-up period. C-reactive protein (CRP) was used to measure the inflammation process. DAS28-CRP, CDAI and SDAI were calculated according to the standard formulas. Comparison was performed by analysis variance ANOVA.

**Results:** On baseline, patients on bDMARDs had a significantly higher mean time-averaged 28-joint disease activity score ( $5.03 \pm 0.84$ SD vs  $4.35 \pm 1.20$ SD,  $p<0.001$ ), CDAI ( $25.06 \pm 7.32$ SD vs  $20.83 \pm 10.53$ SD,  $p<0.001$ ) and SDAI ( $28.27 \pm 8.74$ SD vs  $23.19 \pm 11.89$ SD,  $p<0.001$ ) compared to those on csDMARDs. On the 6th month in both groups (bDMARDs and csDMARDs) we found significant decrease in mean DAS28 ( $p<0.001$ ,  $p<0.001$ ), although no significant difference in disease activity between the groups was measured by this indicator ( $3.75 \pm 2.49$ SD vs  $3.90 \pm 1.10$ SD,  $p=0.566$ ). Patients on bDMARDs had significantly lower disease activity compared to those on csDMARDs after 6th and 12th month of treatment assessed by CDAI ( $13.43 \pm 4.98$ SD vs  $16.81 \pm 9.94$ SD,  $p=0.001$ ;  $8.65 \pm 4.53$ SD vs  $15.86 \pm 10.02$ SD,  $p<0.001$ ), and SDAI ( $14.63 \pm 5.42$ SD vs  $18.38 \pm 10.49$ SD,  $p<0.001$ ;  $9.39 \pm 4.92$ SD vs  $16.79 \pm 10.5$ SD,  $p<0.001$ ). Unlike results reported by DAS28-CRP which showed no change between the 6th and 12th month in patients receiving csDMARDs ( $3.90 \pm 1.10$ SD,  $3.82 \pm 1.12$ SD,  $p=0.156$ ) we observed a statistically significant difference in all three time intervals (0,6th,12th month) of the follow up period regarding to CDAI and SDAI.

**Conclusions:** After a year prospective follow-up, therapy with biologic DMARDs results in sustained suppression – minimal disease activity assessed by DAS28-CRP, CDAI and SDAI, compared to patients receiving DMARDs who had moderate disease activity according to these tools. The therapy with bDMARDs was superior to csDMARDs therapy for suppressing disease activity (assessed by DAS28-CRP,

CDAI and SDAI) of rheumatoid arthritis (RA) on 6th and 12th month of the follow-up period.

**Disclosure of Interest:** None declared

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**AB0285 CLINICAL SIGNIFICANCE OF GLUCOCORTICOID-INDUCED TUMOR NECROSIS FACTOR RECEPTOR RELATED PROTEIN LIGAND (GITRL) IN RHEUMATOID ARTHRITIS: RELATION TO DISEASE ACTIVITY AND TREATMENT OUTCOME**

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**Background:** Glucocorticoid-induced tumor necrosis factor receptor related protein (GITR) is a member of the tumor necrosis factor receptor superfamily that is activated by its specific ligand (GITRL). GITR is mainly expressed in immature and mature T cells especially regulatory (Treg) cells (CD4+ CD25+) and effector T cells (CD25-) [1]. GITRL is mainly expressed in endothelial cells, dendritic cells, macrophages and B cells but not in T cells. GITR-GITRL system is known to have important regulatory role on inflammatory response and immune reactivity.

**Objectives:** This study aimed to measure serum and synovial fluid (SF) levels of GITRL in patients with recent onset rheumatoid arthritis (RA) before and after initiation of therapy and to evaluate the relationship between GITRL and RA clinical and laboratory characteristics, disease activity and response to therapy.

**Methods:** We measured GITRL in the serum (n=48) and SF samples (n=21) from 48 recent onset RA patients and in the serum from 20 healthy control (n=20) at baseline and 6 months after initiation therapy with non-biological disease modifying anti-rheumatic drugs (DMARDs). In the patients Disease activity was calculated by the 28 joint counts (DAS28) and musculoskeletal ultrasound examination (MSUS) was performed at baseline and after 6 months using a 12-joint score (bilateral elbow, wrist, 2nd metacarpophalangeal (MCP), 3rd MCP, knee, ankle) [2]; immunoglobulin-M rheumatoid factor (IgM-RF) titre, anti-cyclic citrullinated peptide (anti-CCP) antibodies titre and C-reactive protein (CRP) levels were measured and the health assessment questionnaire (HAQ) score were recorded.

**Results:** Serum and SF GITRL levels were highly significantly increased in RA ( $39.38 \pm 16.78$  ng/mL and  $30.6 \pm 16.79$  ng/mL respectively) compared to serum level in the healthy controls ( $10.3 \pm 5.46$  ng/mL) ( $p<0.001$ ). In RA patients, baseline serum and SF levels of GITRL significantly correlated with DAS28 ( $r=0.52$  and  $0.56$  respectively,  $p<0.05$ ), anti-CCP titres ( $r=0.46$  and  $0.51$  respectively,  $p<0.05$ ), grey scale (GS) ( $r=0.5$  and  $0.52$  respectively,  $p<0.05$ ) and power Doppler (PD) ( $r=0.65$  and  $0.68$  respectively,  $p<0.001$ ) synovitis scores. Also, serum and SF levels of GITRL at 6 months follow up significantly correlated with the DAS28 ( $r=0.42$  and  $0.48$  respectively,  $p<0.05$ ), GS score ( $r=0.46$  and  $0.51$  respectively,  $p<0.05$ ), PD signal ( $r=0.43$  and  $0.45$  respectively,  $p<0.05$ ). Logistic regression analysis showed that baseline serum levels of GITRL were predictive of follow up DAS 28 and PD synovitis score ( $p=0.009$  and  $0.03$  respectively).

**Conclusions:** Rheumatoid arthritis patients have significantly increased serum and synovial levels of GITRL that remarkably correlated with the DAS28 and MSUS parameters of inflammations suggesting that it could be a useful marker to reflect RA disease activity. GITRL could be a useful biomarker to predict treatment outcome in RA patients.

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**Disclosure of Interest:** None declared

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**AB0286 14 CASES STUDY OF MACROPHAGE ACTIVATION SYNDROME (MAS) IN SYSTEMIC ONSET JUVENILE IDIOPATHIC ARTHRITIS**

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**Background:** Macrophage activation syndrome (MAS) is a severe, potentially life-threatening syndrome.

**Objectives:** we aim to review the precipitating events, clinical features, treatment, and outcome of macrophage activation syndrome (MAS). Part patients were analysed the Polymorphisms of Perforin A91V (NCBI:SNP rs35947132) using special primers by polymerase chain reaction (PCR).

**Methods:** Retrospective review of cases of MAS from a prospectively collected database of children with autoimmune diseases from 2003 to 2008.

**Results:** Fourteen patients (nine boys) were considered to have evidence of MAS. The primary diagnosis was systemic onset juvenile idiopathic arthritis, with age ranged from 5 months to 12 years. No medication was identified as trigger. Eleven had infections prior to MAS, specific infectious agents were identified in four. High grade fever, new onset hepatosplenomegaly, lymphadenopathy, dysfunction