discontinuation. These data suggest that the development of ADA is a frequent cause of secondary inefficacy in our RA pts

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.6371

SAT0169 MAINTENANCE AND IMPROVEMENT IN CLINICAL EFFICACY BETWEEN WEEK 12 AND 24 IN PATIENTS WITH RHEUMATOID ARTHRITIS TREATED WITH SB4 OR REFERENCE ETANERCEPT

P. Emery¹, J. Vencovsky², E. Keystone³, J. Ghil⁴, S.Y. Cheong⁴, E.E. Hong⁴.
¹Leeds Institute of Rheumatic and Musculoskeletal Medicine, Leeds, United Kingdom; ²Institute of Rheumatology, Prague, Czech Republic; ³Mount Sinai Hospital, Toronto, Canada; ⁴Samsung Bioepis Co., Ltd., Incheon, Korea, Republic Of

Background: SB4 is approved by the European Commission as a biosimilar of the reference etanercept (ETN).

Objectives: To evaluate the maintenance and improvement in clinical efficacy between week 12 and 24 in patients with rheumatoid arthritis (RA) treated with SB4 or ETN from a post-hoc analysis of phase III results.

Methods: Patients with RA were randomised to receive 50 mg/week of either SB4 or ETN with background methotrexate. American College of Rheumatology (ACR) and European League Against Rheumatism (EULAR) responses were compared at week 12 and week 24. At week 12, patients with available assessment results were categorised as ACR responders or ACR non-responders and EULAR responders (patients with moderate or good EULAR response) or EULAR non-responders. The same was assessed for week 24 and patients with missing data at week 24 were regarded as non-responders.

Results: A total of 551 patients (283 patients from SB4 and 268 patients from ETN) completed 24 weeks of the study. In both treatment groups, efficacy was well maintained between week 12 and week 24. Among patients who were ACR20, 50, or 70 responders at week 12, 90.8% vs. 91.4%, 80.5% vs. 80.6%, and 74.5 vs. 77.5% of patients from SB4 and ETN, respectively, maintained their responses at week 24. Likewise, EULAR response was maintained by 93.1% vs. 92.6% of patients who had a good or moderate response at week 12. (Table). The improvement in ACR responses between week 12 and 24 was comparable between SB4 and ETN group (Table). In SB4 and ETN, respectively, 42.1% vs. 50.5% of 12-week ACR20 non-responders became ACR20 responders at week 24. Similarly, 20.9% vs. 21.9% of 12-week ACR50 non-responders became ACR50 responders and 13.0% vs. 11.4% of 12-week ACR70 non-responders became ACR70 responders. The improvement in EULAR responses was also comparable between SB4 and ETN. 43.2% vs. 52.2% of 12-week EULAR non-responders in SB4 and ETN, respectively, became EULAR responders at week 24.

Table. Maintenance and improvement of efficacy between week 12 and week 24

		Week 24 responders	
		SB4 50 mg n/n' (%)	ETN 50 mg n/n' (%)
ACR20	Responder at week 12	179/196 (91.3%)	162/175 (92.6%)
	Non-responder at week 12	41/97 (42.3%)	51/105 (48.6%)
ACR50	Responder at week 12	91/114 (79.8%)	75/93 (80.6%)
	Non-responder at week 12	37/179 (20.7%)	41/187 (21.9%)
ACR70	Responder at week 12	38/51 (74.5%)	31/40 (77.5%)
	Non-responder at week 12	31/242 (12.8%)	28/240 (11.7%)
EULAR response*	Responder at week 12	231/246 (93.9%)	215/231 (93.1%)
	Non-responder at week 12	19/46 (41.3%)	24/49 (49.0%)

n, number of responders; n', number of responders or non-responders at week 12

*Patients with moderate or good EULAR response were regarded as responders.

Conclusions: Efficacy of SB4 and ETN was well maintained and the maintenance rate was comparable between week 12 and week 24. In addition, a similar and considerable proportion of patients in SB4 and ETN who did not achieve a clinical response at week 12 reached clinical response at week 24. These results suggest that etanercept non-responders at week 12 may benefit from continuing treatment up to 24 weeks.

Disclosure of Interest: P. Emery Grant/research support from: Abbvie, BMS, Pfizer, USB, MSD, Novartis, Lilly, Consultant for: Samsung Bioepis Co., Ltd., J. Vencovsky Consultant for: Samsung Bioepis Co., Ltd., Biogen, E. Keystone Grant/research support from: Abbott, Amgen, AstraZeneca, BMS, Janssen, Lilly, Novartis, Pfizer, Consultant for: Abbott, AstraZeneca, Biotest, BMS, Genentech, Janssen, Lilly, Merck, Pfizer, Samsung Bioepis, Speakers bureau: Abbott, AstraZeneca, BMS Canada, Janssen, J. Ghil Employee of: Samsung Bioepis Co., Ltd., S. Y. Cheong Employee of: Samsung Bioepis Co., Ltd., E. Hong Employee of: Samsung Bioepis Co., Ltd.

DOI: 10.1136/annrheumdis-2017-eular.3344

SAT0170 B-CELL PHENOTYPE AND IGD-CD27- MEMORY B CELLS ARE AFFECTED BY TNF-INHIBITORS AND TOCILIZUMAB TREATMENT IN RHEUMATOID ARTHRITIS

R.A. Moura¹, C. Quaresma¹, A.R. Vieira¹, M.J. Gonçalves^{1,2}, J. Polido-Pereira^{1,2}, V.C. Romão^{1,2}, N. Martins², H. Canhão^{1,2}, J.E. Fonseca^{1,2}.
¹Rheumatology Research Unit, Instituto de Medicina Molecular; ²Rheumatology Department, Centro Hospitalar de Lisboa Norte, EPE, Hospital de Santa Maria, Lisbon, Portugal

Background: The use of TNF-inhibitors and/or the IL-6 receptor antagonist, tocilizumab, in rheumatoid arthritis (RA) have pleiotropic effects that also involve circulating B-cells.

Objectives: The main goal of this study was to assess the effect of TNF-inhibitors and tocilizumab on B-cell phenotype and gene expression in RA.

Methods: Blood samples were collected from untreated early RA (ERA) patients (<1 year of disease duration), established RA patients under methotrexate treatment, established RA patients before and after treatment with TNF-inhibitors and tocilizumab, and healthy donors. B-cell subpopulations were characterized by flow cytometry and B-cell gene expression was analyzed by real-time PCR on isolated B-cells. Serum levels of BAFF, CXCL13 and sCD23 were determined by ELISA.

Results: The frequency of total CD19+ B-cells in circulation was similar between controls and all RA groups, irrespective of treatment, but double negative (DN) IgD-CD27- memory B-cells were significantly increased in ERA and established RA when compared to controls. Treatment with TNF-inhibitors and tocilizumab restored the frequency of IgD-CD27- B-cells to normal levels, but did not affect other B-cell subpopulations. TACI, CD95, CD5, HLA-DR and TLR9 expression on B-cells significantly increased after treatment with either TNF-inhibitors and/or tocilizumab, but no significant changes were observed in BAFF-R, BCMA, CD69, CD86, CXCR5, CD23, CD38 and IgM expression on B-cells when comparing baseline with post-treatment follow-ups. Alterations in B-cell gene expression of BAFF-R, TACI, TLR9, FcγRIIB, BCL-2, BLIMP-1 and β2M were found in ERA and established RA patients, but no significant differences were observed after TNF-inhibitors and tocilizumab treatment when comparing baseline and follow-ups. Serum levels of CXCL13, sCD23 and BAFF were not significantly affected by treatment with TNF-inhibitors and tocilizumab.

Conclusions: In RA, treatment with either TNF-inhibitors or tocilizumab affects B-cell phenotype and the frequency of memory B-cell subpopulations in peripheral blood, particularly DN (IgD-CD27-) B-cells, but not B-cell gene expression or serum levels of CXCL13, sCD23 and BAFF, when comparing baseline with post-treatment follow up. Overall, our results may suggest that TNF-inhibitors and tocilizumab inhibit B-cell trafficking towards inflammatory sites, thus supporting activated B-cell recirculation from tissues through blood and lymphatic systems.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.3011

SAT0171 ABP 501 BIOSIMILAR TO ADALIMUMAB: FINAL SAFETY, IMMUNOGENICITY, AND EFFICACY RESULTS FROM AN OPEN-LABEL EXTENSION STUDY

S. Cohen¹, J.L. Pablos², H. Wang³, G.A. Müller⁴, A. Kivitz⁵, A. Matsumoto⁶, E. Krishnan³. ¹Metroplex Clinical Research Center, Dallas, United States; ²Instituto de Investigación Hospital, Madrid, Spain; ³Amgen, Thousand Oaks, United States; ⁴Abteilung für Nephrologie und Rheumatologie, Göttingen, Germany; ⁵Altoona Center for Clinical Research, Duncansville; ⁶Arthritis and Rheumatism Associates, Wheaton, United States

Background: ABP 501 has been approved by the US FDA as the first biosimilar to the fully human recombinant monoclonal antibody, adalimumab. Totality of evidence to date suggests that ABP 501 is highly similar to adalimumab. Subjects receiving either ABP 501 or adalimumab in the active-controlled, comparative, pivotal phase 3 study in rheumatoid arthritis (parent study) continued on to this open-label extension (OLE) study if they had completed the final week 26 visit of that study.

Objectives: To describe the safety, immunogenicity, and efficacy outcomes of ABP 501 in the OLE study.

Methods: Subjects who completed the parent study were screened and were included if they met the eligibility requirements. All subjects included in the OLE were treated with ABP 501 40 mg subcutaneously every other week for 68 weeks followed by disease assessments at week 70 and the follow-up safety assessment at week 72 (or early termination). Data were summarized descriptively and no inferential analyses were performed.

Results: Of the 467 subjects enrolled in the OLE study, 466 were treated with ABP 501. Of these, 237 transitioned from the adalimumab arm of the parent study; 412/467 completed the study. Demographics and disease characteristics were balanced between subjects who transitioned from adalimumab and those who continued on ABP 501 from the parent study. Overall, the incidence of treatment-emergent adverse events (TEAEs) was 63.7% (297/466) and that of grade ≥3 TEAEs was 9.0% (42/466); incidence of TEAEs leading to discontinuation of investigational product was 3.6% (17/466). TEAEs with incidence ≥5% were nasopharyngitis (9.2%), upper respiratory tract infection (8.6%), bronchitis (6.4%), rheumatoid arthritis (6.2%), hypertension (4.7%), and pharyngitis (4.1%). The incidence of serious adverse events was 9.9% (46/466). Most common