years; mean RA duration, 7.7 years; and 82.7% seropositive –similar between treatment groups. Baseline CS was less frequent in the mono vs combo group (41.1% vs 51.2%); however, the mean prednisone equivalent daily dose was similar (6.6 vs 6.5 mg/day, respectively). Pts who continued TCZ to week 24 based on Kaplan-Meier estimates (95% CI) were 79.3% (74.7%>83.2%) for mono and 85.6% (83.7%>87.3%) for combo. DAS28 scores decreased comparably from baseline to week 24 in both groups (mean change: mono −3.40 and combo −3.46), with no significant difference between groups (P =0.61). Results were similar for the Clinical Disease Activity Index (CDAI, mean change by week 24: −23.5 and −23.8, with no significant difference between groups: P=0.42). The proportion of pts who achieved DAS28 or CDAI-based remission, low disease activity or ACR20/50/70/90 responses was similar between groups (Figure 1). In all, 18.2% of pts withdrew; 6.4% did so for safety reasons (mono 9.1%, combo 5.8%). AE rates were similar between groups (Table). Serious AE (SAE) rates were 14.6/100 PY (mono: 22.8/100 PY, combo: 12.8/100 PY). Serious infection and infestation rates were 3.6/100 PY (mono: 4.0/100 PY, combo: 3.5/100 PY) – similar between groups. Six deaths occurred (0.64/100 PY), 1 in the monotherapy group (0.57/100 PY) and 5 in the combination (0.65/100 PY) group.

Figure 1: DAS28 and CDAI Disease Activity and ACR Responses*

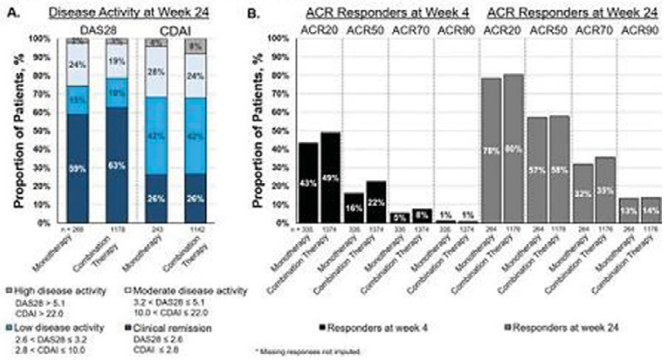

Table: Summary of Relevant Adverse Events

		Total Population (N = 1804; 943.3 PY)	Monotherapy (n = 353; 175.7 PY)	Combination Therapy (n = 1451; 767.6 PY)
Adverse events	Total patients with ≥ 1 AE, n (%)	1508 (83.6%)	282 (79.9%)	1226 (84.5%)
	Rate of AEs per 100 PY	622.4	622.1	622.5
Serious adverse events	Total patients with ≥ 1 SAE, n (%)	105 (5.8%)	29 (8.2%)	76 (5.2%)
	Rate of SAEs per 100 PY	14.6	22.8	12.8
Serious infections and infestations	Total patients with ≥ 1 SAE, n (%)	27 (1.5%)	6 (1.7%)	21 (1.4%)
	Rate of SAEs per 100 PY	3.6	4.0	3.5
Withdrawals due to insufficient therapeutic response	Total patients with ≥ 1 reason, n (%)	40 (2.2%)	9 (2.5%)	31 (2.1%)
Withdrawals due to safety reasons*	Patients, n (%)	116 (6.4%)	32 (9.1%)	84 (5.8%)
Deaths	Number of deaths, n (%)	6 (0.3%)	1 (0.3%)†	5 (0.3%)‡
	Rate of deaths per 100 PY	0.64	0.57	0.65

AE, adverse events; SAE, serious adverse events; PY, patient-year.
* Deaths, anaphylaxis and hypersensitivity reactions not included.
† Coronary artery disease.
‡ Myocardial infarction, pneumonia, pulmonary fibrosis, sepsis, septic shock.
Note: TOZURA trial numbers: NCT01995201, NCT02046603, NCT02011334, NCT02031471, NCT02001987, NCT01941095, NCT01941940, NCT02046616, NCT01988012, NCT01987479, NCT01951170.

Conclusions: TCZ-SC demonstrated convincing and comparable efficacy as mono- and combination therapy in pts with RA as was previously observed with TCZ-IV. The safety profile of TCZ-SC is consistent with the known safety profile of TCZ as monotherapy and in combination with csDMARDs.

Acknowledgements: Funded by F. Hoffmann-La. Roche, Ltd.

Disclosure of Interest: E. Choy Grant/research support from: F. Hoffmann-La Roche, Chugai Pharmaceutical, Consultant for: F. Hoffmann-La Roche, Chugai Pharmaceutical, Speakers bureau: F. Hoffmann-La Roche, Chugai Pharmaceutical, R. Caporali Consultant for: AbbVie, Bristol-Myers Squibb, F. Hoffmann-La Roche, UCB and MSD Pharmaceuticals, R. Xavier Consultant for: F. Hoffmann-La Roche, Pfizer, Janssen Pharmaceuticals, AbbVie, Eli Lilly and Company, Speakers bureau: F. Hoffmann-La Roche, Pfizer, Janssen Pharmaceuticals, Abbvie, Eli Lilly and Company, B. Fautrel: None declared, R. Sanmarti: None declared, C. Bernasconi Employee of: F. Hoffmann-La Roche (contractor), A. Pethö-Schramm Employee of: F. Hoffmann-La Roche

DOI: 10.1136/annrheumdis-2017-eular.1615

SAT0200 BIOLOGIC THERAPY RETENTION IN RHEUMATOID ARTHRITIS (RA) PATIENTS (PTS) ACCORDING TO THE MOSCOW ARTHRITIS REGISTRY (MAR)

G. Lukina[1], E. Koltsova[2], E. Schmidt[3], E. Zhilyaev[4]. *[1]V.A. Nasonova Research Institute of Rheumatology, Moscow Clinical Scientific Center; [2]Research Institute of the Organization of health and healthcare management; [3]City Clinical Hospital 1 named after N.I. Pirogov; [4]Russian Medical Academy of continuing professional education, CJSC "European Medical Center", Moscow, Russian Federation*

Background: The use of biologics significantly improved results of the therapy of RA pts who did not achieve the target disease activity level on traditional DMARDS treatment. However the biologic therapy is in many cases withdrawn due to inefficacy or side effects (seldom because of sustained remission). Retention on treatment is a good integral index of efficacy and safety of biologics used in the real clinical practice.

Objectives: To assess the treatment survival of various biologics in RA pts in the real clinical practice.

Methods: Patients from MAR with RA receiving biologics were enrolled. Cases with missed results were excluded. A Cox proportional hazards regression model was used to determinethe predictors of the treatment discontinuation risk. Comparison of biologics retention rates for different biologicswas performed by means of Kaplan-Meier survival curves. Bonferroniadjustment was applied because of multiplicity of comparisons.

Results: 306 RA pts (mean age 54,5 years, mean age of disease onset 39,6 years, 86,5% women, 18% smokers, RF-positive 83,7%) were included in the study and 394 treatment courses (263 retrospective and 131 prospective) were analyzed. It was shown that significant independent predictors of discontinuation risk were: the biologic drug, the sequence number of the biologic drug in the patient and the age of RA onset. Risk of withdrawal was minimal by the use of the first biologic and increased by administration of the next ones. It also increased in pts with late onset of RA. Mass body index, age of the patient and the dose of methotrexate did not show significant correlations. Abatacept (ABA) demonstrated significant superiority over adalimumab (ADA) ($p<0.001$), infliximab (INF) ($p<0.001$), rituximab (RTM) (p=0.004) and etanercept (ETA) (p=0.035) when they was used as the first biologic drug. The treatment survival of tocilizumab was significantly higher compared to INF (p=0.02). As a second-line biological therapy ADA was maintained significantly longer than the INF (p=0.048).

Conclusions: Results of the real clinical practice trial show the significant differences in the retention rates of some biologics. It is reasonable to take these differences into consideration by the planning of the biologic treatment of RA pts.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.3657

SAT0201 ABATACEPT BUT NOT TNF INHIBITORS BLOCK AUTOANTIBODY-MEDIATED CYTOKINE PRODUCTION BY MONOCYTES

A. Bozec[1], Y. Luo[2], C. Engdahl[1], C. Figueiredo[3], H. Bang[4], G. Schett[1]. *[1]University of Erlangen-Nuremberg, Erlangen, Germany; [2]University of Chengdu, Chengdu, China; [3]Universidade de São Paulo, Sao Paulo, Brazil; [4]Orgentec Inc., Mainz, Germany*

Background: The anti-inflammatory effect of abatacept (CTLA4-Ig) is most pronounced in patients with high-titer autoantibodies (including anti-citrullinated protein antibodies, ACPA, and rheumatoid factor, RF) even exceeding the effect of TNF inhibitors (TNFi)[1]. Considering that autoantibodies trigger inflammatory cytokine production by monocytes[2] and that abatacept bind to monocytes influencing their functional state[3] we hypothesized that abatacept, in contrast to TNFi, may effectively inhibit the production of several different cytokines by ACPA-or RF-challenged monocytes.

Objectives: (i) To test whether abatacept inhibits the production of TNFa, IL-1b, IL-6 and IL-8 by monocytes exposed to ACPA or RF, (ii) to compare these effects of abatacept with those of TNFi and (iii) to investigate whether the effect of abatacept on cytokine production is based on IDO induction in monocytes.

Methods: CD68+ monocytes were isolated from peripheral blood and stimulated with MCSF for 24 hours before exposing them to random IgG alone (negative control), 10mg/mL purified anti-citrullinated vimentin antibodies (ACPA), 10mg/mL RF or LPS (positive control) in cell culture plates coated with citrullinated vimentin (to allow ACPA immune complex formation). ACPA and RF stimulation was done in the presence or absence of abatacept or TNF-antibody (adalimumab) with or without IDO inhibitor 1-MT. Supernatants were analyzed for four key pro-inflammatory cytokines TNFa, IL-1b, IL-6 and IL-8 by cytokine array (R&D Proteome Profiler) after 24h.

Results: Exposure to ACPA or RF dramatically induced the production of TNFa (20 fold and 27-fold, respectively) IL-1b (each 4-fold), IL-6 (12-fold and 11-fold, respectively) IL-8 (43-fold and 30-fold, respectively) in human monocytes. Abatacept significantly inhibited this up-regulation of inflammatory cytokine production with TNFa reduced by 79%, IL-1b by 74%, IL-6 by 88% and IL-8 by 83%. In contrast, TNFi did not influence autoantibody-induced production of IL-1b, IL-6 and IL-8. Inhibition of IDO by 1-MT reversed the effect of abatacept and unlocked cytokine production in the presence of ACPA and RF.

Conclusions: These data show that abatacept interferes with autoantibody mediated cytokine production by induction of IDO. The fact that several different