Table 1. Baseline predictors of reduction of disease activity at 12 months from start of abatacept. Linear regression.

Variables	Bivariate			Adjusted for baseline DAS28			Multivariate		
	B	95% CI	P-value	B	95% CI	P-value	B	95% CI	P-value
Male sex	0.39	[0.13, 0.64]	0.003	0.43	[0.20, 0.66]	<0.001	0.42	[0.19, 0.64]	<0.001
Age (per SD)	0.10	[-0.004, 0.21]	0.058	0.044	[-0.051, 0.14]	0.37	N/A		
RA duration (per SD)	-0.14	[-0.25, -0.037]	0.008	-0.11	[-0.21, -0.016]	0.022	-0.030	[-0.13, 0.065]	0.54
HAQ (per SD)	0.12	[0.007, 0.23]	0.037	-0.24	[-0.35, -0.13]	<0.001	N/A		
VAS pain (per SD)	0.26	[0.15, 0.37]	<0.001	-0.058	[-0.17, 0.054]	0.31			
MTX ¹	0.20	[-0.013, 0.41]	0.066	0.17	[-0.018, 0.36]	0.076	0.11	[-0.076, 0.29]	0.25
Prednisolone ¹	-0.19	[-0.41, 0.017]	0.072	-0.17	[-0.36, 0.024]	0.087	-0.14	[-0.33, 0.043]	0.13
bDMARDs ² (per SD)	-0.30	[-0.40, -0.20]	<0.001	-0.32	[-0.41, -0.23]	<0.001	-0.31	[-0.40, -0.21]	<0.001

B=beta coefficient; N/A=not applicable; SD=standard deviation; CI=confidence interval. Bold text indicates significant associations. DAS28, Disease activity Score of 28 joints; RA, rheumatoid arthritis; HAQ, Health Assessment Questionnaire; VAS, visual analogue scale; MTX, methotrexate; bDMARD, biologic disease-modifying antirheumatic drug. ¹Current treatment ²Number of previous bDMARDs

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POS0630 COMPARISON OF THE EFFICACY AND SAFETY OF ORIGINAL AND BIOSIMILAR ADALIMUMAB MOLECULES IN CHILDHOOD RHEUMATIC DISEASES

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Background: The TNF- α inhibitor adalimumab is a biological disease modifying anti-rheumatic drug (bDMARD) that has been used in different rheumatic diseases with a resistant course. ABP-501 is a biosimilar product (BP) of adalimumab, recently approved by the FDA and EMA. To our knowledge, there is no study assess the efficacy and safety of these two molecules on pediatric patients.

Objectives: We aimed to compare the efficacy and safety of the original and biosimilar adalimumab (ABP-501) molecules in childhood rheumatic diseases.

Methods: This non-interventional, retrospective, single-centre analysis carried out in Umraniye Training and Research Hospital, Pediatric Rheumatology Clinic, Istanbul, Turkey. The study group consisted of patients who were followed due to chronic rheumatic disease between January 1, 2016 and June 1, 2020, and received reference or biosimilar adalimumab therapy for at least three months. Demographic and clinical data of patients were collected at baseline, 3rd, 6th, and 12th months of treatment. Disease activity assessment was made with JADAS-27 in JIA patients, with SUN criteria in uveitis patients, and with Behçet's Disease Activity Index in BD patients. Efficacy and safety of treatments were compared between reference and biosimilar adalimumab groups.

Results: A total of 89 patients (65 with original and 24 with biosimilar molecule) treated with adalimumab, were included in the study. There were 45 female and 44 male in the study, and the median age at the initiation of the adalimumab was 166 months (min-max: 36-231). Of the 89 patients evaluated, the primary diagnoses of 62 were juvenile idiopathic arthritis, 13 were idiopathic uveitis, eight were Behçet's disease, three were Blau syndrome, two were chronic recurrent multifocal osteomyelitis and one was Vogt-Koyanagi-Harada syndrome. 63 of the patients were biologic-naïve, and 13 were switched from etanercept, 11 from infliximab, and two from other bDMARDs. The median exposure time of adalimumab was 16 months (min-max: 3-70) in RP and 14.5 months (min-max: 3-23) in BP. All patients had active disease before treatment. In the group treated with RP, inactive disease was achieved in 60%, 76.6% and 87.2% of the patients at the 3rd, 6th and 12th months, respectively. Also, inactive disease was achieved in 62.5%, 78.2% and 78.2% of the patients at the 3rd, 6th and 12th months in the group treated with BP, respectively. There was no statistically significant difference in efficacy between the groups at the 3rd, 6th and 12th months ($p=0.83$, 0.07 and 0.32). Serious adverse events were seen in one patient in each groups (lymphoma in RP group, tuberculous meningitis in BP group). Non-serious adverse events were observed in eight patients (12.3%) in the RP group and in two patients (8.3%) in the BP group, without statistically significant difference between groups ($p=0.86$).

Conclusion: No significant difference was observed between the biosimilar adalimumab ABP-501 and RP adalimumab in terms of efficacy and safety.

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POS0631 COMPARATIVE EFFICACY OF COMBINATION THERAPY WITH BIOLOGIC OR TARGET SYNTHETIC DRUGS FOR RHEUMATOID ARTHRITIS: A BAYESIAN NETWORK META-ANALYSIS

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Background: Biologic agents and small molecules have shown long term benefit when added in patients with active RA non-responders to conventional DMARDs treatment (1). In head-to-head trials only adalimumab was compared to other drugs in combination with methotrexate, with some evidence of superiority but no data on multiple comparisons have been reported (2). The availability of biosimilar agents led in clinical practice to prefer mainly the cheaper one, so the choice of the most effective treatment remains a clinical unmet need (3).

Objectives: To assess the relative efficacy of different therapeutic strategies for achieving ACR50 response at 24 weeks of treatment in patients with active RA, based on direct and indirect evidence.

Methods: We performed systematic reviews of MEDLINE, EMBASE, and Cochrane Library databases, searching for all published phase 3 Randomised Controlled Trials (RCTs) comparing adalimumab originator to its biosimilars, abatacept, baricitinib, certolizumab pegol, tofacitinib or upadacitinib, combined to MTX, in patients with active RA inadequate responders to previous conventional DMARDs. American College of Rheumatology (ACR) 50% response at 24 weeks of treatment had to be evaluated both in adalimumab branch and in examined drug branch. Bayesian fixed-effect network meta-analysis was performed to combine the direct and indirect evidence using the WinBUGS 1.4 software (MRC Biostatistics Unit, Cambridge, UK).

Results: Eleven RCTs evaluating 6'004 patients were included in the analysis, namely originator (1) and biosimilars (2) adalimumab, abatacept (3), baricitinib (4), certolizumab pegol (5), tofacitinib (6) and upadacitinib (7). Convergence was reached at n.100'000 iterations. Upadacitinib seems to be more effective than both originator and biosimilar adalimumab in achieving ACR 50 (OR 1.65 95% CI 1.25-2.14 and OR 1.22 95%CI 1.10-2.18; see Figure 1). Similarly, tofacitinib was more effective of originator adalimumab (OR 1.25 95%CI 1.01-155). Upadacitinib was ranked first among treatments with a probability of being the agent more likely to induce ACR 50 response of 86.3%. In this regard tofacitinib had a probability of 4.8%, hence it was ranked second among treatments.

Figure 1. Caterpillar plot OR for ACR50 at 24 weeks (originator [1] and biosimilars [2] adalimumab; abatacept [3]; baricitinib [4]; certolizumab pegol [5]; tofacitinib [6]; upadacitinib [7]).

Conclusion: Although patients with active RA and inadequate response to MTX have different therapeutic combination of biologics or small molecules options, the best relative efficacy in terms of ACR50 response after 24 weeks of treatment is for upadacitinib 15 mg/day.

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POS0632

THE LONGITUDINAL ASSOCIATIONS OF METHOTREXATE AND BIOLOGIC USE ON HOSPITAL ADMISSION FOR RHEUMATOID ARTHRITIS PATIENTS IN WESTERN AUSTRALIA POPULATION (1995- 2014)

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Background: Rheumatoid arthritis (RA) carries a substantial burden for patients and society in terms of morbidity, enduring disability, and costs [1]. The Australian Pharmaceutical Benefits Scheme (PBS) has subsidised biological disease-modifying anti-rheumatic drugs (B-DMARDs) since 2003 [2].

Objectives: We examined the impact of B-DMARDs availability on RA hospitalisation rate in the Western Australia (WA) population pre- and post- B-DMARDs introduction to the PBS (1995-2002 and 2003-2014).

Methods: Population PBS dispensing data for WA of DMARD were obtained and converted to defined daily doses (DDD)/1000 population/day using the WA population census. RA inpatient records were extracted from the WA Hospital Morbidity Data Collection using ICD-9 codes 714 and ICD-10 codes M05.00-M06.99). Principal component analysis (PCA) was applied to determine the relationship between DMARDs use and RA hospital admission rates.

Results: There was a total of 17,125 patients who had 50,353 admissions with a diagnostic code for RA during the study period. DMARD use for RA rose from 1.45 to 3.19 DDD/1000 population/day over 1995-2014 (Figure 1). In 1995-2002, the number of RA admissions fell from 7.9 to 2.6 per 1000 hospital separations, then dropped further from 2.9 to 1.9 per 1000 hospital separations in 2003-2014. Based on PCA analysis, conventional DMARDs (methotrexate) and B-DMARDs dispensing had an inverse association with hospital admissions for RA.

Conclusion: The increased availability of conventional and biological DMARDs for RA was associated with a significant decline in hospital admissions for RA patients in WA.

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Figure 1. The hospital separations and total drugs use patterns of RA in 1995-2014 in Western Australia.

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POS0633

DURATION OF STARTING bDMARDs ARE ALMOST 3 TIMES LONGER IN RA PATIENTS THAN PsA PATIENTS: HUR-BIO REAL LIFE RESULTS

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Background: Before using biological DMARDs, EULAR suggests the use of synthetic DMARDs (especially methotrexate) for RA and PsA [1-2].

Objectives: It was aimed to evaluate the differences of disease duration and csDMARDs till first bDMARD in RA and PsA patients.

Methods: HUR-BIO (Hacettepe University Biologic Registry) is a prospective, single center database of biological treatments since 2005 and to date 2070 RA and 520 PsA patients have been recorded. Demographic, clinical and laboratory data before bDMARDs of the patients were noted. When investigating the differences between groups, the effects of gender, age and disease duration were adjusted using two-way ANOVA and ANCOVA tests. The selection was made for the gender, age and for indifference of the relevant groups by using propensity score matching.

Results: We included 481 RA, and 482 PsA age and gender matched patients in the study. Age, gender and disease duration information were given in the Table 1. 72.8% of the RA patients were RF or anti-CCP positive. Overall, 56.3, 100% of the RA, and PsA patients first biologic therapies were anti-TNFs, respectively. All RA patients started with csDMARDs before bDMARD treatments, whereas 450 of 482 (93.4%) PsA patients. Methotrexate was the anchor csDMARD for both diseases. RA patients more frequently used all csDMARDs included methotrexate, leflunomide, sulphasalazine hydroxychloroquine and corticosteroids as well. Median disease duration till bDMARD treatments in RA and PsA patients were 55 and 18.5 months respectively (p<0.001) (Table 1).

Table 1. Demographic characteristics and csDMARDs before first bDMARD

	RA (n=481)	PsA (n=482)	P value
Female, n (%)	319 (66.3)	332 (68.9)	0.218
Age, years (mean±SD)	48.2 ± 13.5	47.4 ± 12.2	0.332
Disease duration, years*	10 (6-16)	7 (3-12)	0.000
Symptom duration before diagnosis, years*	0 (0-1)	1 (0-4)	0.000*
The period of time between diagnosis and bDMARD initiation, months*	55 (24-115)	18.5 (8-58)	0.000*
The period of time between symptoms and bDMARD initiation, months*	70 (35-151)	48 (20-124)	0.000*
Methotrexate	Ever n (%)	400 (83.3)	373 (77.5)
	Just before bDMARD initiation n (%)	251 (52.2)	230 (47.7)
			0.093
Hydroxychloroquine sulfate	Ever n (%)	292 (60.8)	170 (35.3)
	Just before bDMARD initiation n (%)	262 (54.5)	99 (20.5)
			0.000*
Leflunomide	Ever n (%)	237 (49.4)	129 (26.8)
	Just before bDMARD initiation n (%)	160 (33.3)	96 (19.9)
			0.000*
Sulphasalazine	Ever n (%)	353 (73.5)	265 (55.1)
	Just before bDMARD initiation n (%)	156 (32.4)	146 (30.3)
			0.259*
Corticosteroids	Ever n (%)	419 (87.3)	281 (58.4)
	Just before bDMARD initiation n (%)	335 (69.6)	187 (38.8)
			0.000*

*Median (IQR)

Conclusion: According to HUR-BIO real life data, for inflammatory arthritis patients who started bDMARDs, the periods of time between diagnosis and bDMARDs were more reasonable (18 months) in PsA patients than RA patient's periods which were approximately three times longer. RA patients were used much more and longer duration of csDMARDs. This explicit distinction may be explained by synthetic DMARDs on activity differences between the RA and PsA.

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