between stop of SB4 and re-start of ETA was 1^{1-1} days. Baseline characteristics of back-switchers vs the rest of the switch population were similar (all $p>0.05$). Among back-switchers who stopped SB4 due to LOE, PGA increased during SB4 treatment, whereas CRP and SJC were largely unchanged (table 1). At the date of censoring, 104/120 back-switchers (87%) were still treated with originator ETA (median treatment duration 236 (155–302) days).

Abstract FRI0103 – Table 1. Description of ETA-SB4-ETA back-switchers (n=120)

	RA, n=80	PsA, n=20	AxSpA, n=20
Characteristics upon start of SB4, n=120			
Female, %	73	55	35
Age, years	59 (52-70)	45 (36-56)	43 (38-56)
SJC, n	0 (0-1)	0 (0-0)	-
CRP, mg/L	3 (1-8)	1 (1-5)	3 (1-6)
PGA, mm	27 (12-54)	25 (13-63)	23 (13-44)
Characteristics upon re-start of ETA in pts who back-switched due to LOE, n=62			
Pt number, n	38	11	13
SJC, n	2 (0-5)	0 (0-2)	-
CRP, mg/L	3 (2-11)	3 (2-7)	4 (1-6)
PGA, mm	64 (50-76)	78 (18-90)	42 (35-63)
Delta-values among pts who stopped due to LOE and back-switched, n=62**			
Delta-SJC, n	1 (0-4)	0 (0-0)	-
Delta-CRP, mg/L	0 (-1-5)	1 (0-2)	0 (0-4)
Delta-PGA, mm	30 (12-52)	15 (7-77)	25 (19-35)
Pts stopped due to AE, n=47: Arthralgia 1 pt, bladder dysfunction 1, blurred vision 1, diarrhea 4, dizziness 2, dyspnea 2, erectile dysfunction 1, hair loss 1, headache/migraine 4, hyperhidrosis 2, hypertension 1, hypotension 1, infections 2, leg cramps 1, local injection problems 3, myalgia 1, nausea 2, neuropathies 1, psoriasis/pustulosis 1, rash/itching 9, not stated 21 (total =62 events)			
Numbers are medians (IQR) unless otherwise stated, *DAS28<2.6 (RA, PsA), ASDAS<1.3 (AxSpA)			
** Disease activity at time of re-start ETA minus SB4 start			
Abbreviations: AE: adverse event, CRP: C-reactive protein, ETA: originator etanercept, LOE: lack of effect, PGA: patient global score, SB4: biosimilar etanercept, SJC: swollen joint count			

Conclusions: In a nationwide cohort of 1621 arthritis patients that were switched from ETA to SB4, 7% switched back to ETA. Back-switchers had no distinct clinical or disease characteristics upon start of SB4. AEs prior to back-switching were largely unspecific. In pts who withdrew SB4 due to LOE, PGA had increased. Reasons for back-switching appeared to be predominantly subjective rather than objective (nocebo effect). Originator drug was still available (25 mg or 50 mg powder-solution), which may have encouraged back-switching.

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FRI0104

PREDICTORS FOR THE RESPONSE TO ANTI-TNF THERAPY AND THE IMPACT OF SMOKING IN RHEUMATOID ARTHRITIS- RESULTS ON A GROUP OF PATIENTS IN ROMANIA

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Background: 20%–40% of the patients are declared nonresponders to at least one of the biologic agents.

Objectives: to test the possible predictive role of RF type IgM and IgA, anti-CCP, anti-MCV, 14–3–3eta protein and COMP on a group of patients treated with anti-TNFα agents. We also followed the evolution of serum titres of these biomarkers under biologic therapy and the impact of smoking regarding response to treatment.

Methods: prospective and observational study including 64 patients followed 12 months with active RA, uncontrolled by csDMARDs. Clinical assessment was performed at 0, 6 and 12 months according to ACR criteria and evaluation of treatment response according to EULAR criteria (good/moderate/nonresponder).

Results: Following baseline immunological parameters titres and the response at 6 months, tests for identifying differences between the groups showed that lower titres of both RF isotypes, anti-CCP, 14–3–3eta protein and COMP had predictive value on achieving a good EULAR response at 6 months. Grouping patients in 2 categories (responders/nonresponders), just 14–3–3eta protein and anti-CCP maintained their predictive value for the response at 6 months.

For the visit at 12 months, lower baseline titres for RF type IgM (92.93±120.22 U/ml, $p=0.010$) and IgA (49.96±98.08 U/ml, $p=0.002$) had predictive value for achieving a good response at 12 months. We didn't obtain other informations grouping patients in 2 categories.

The status pretreatment influenced the good response for COMP at 6 months ($p=0.001$) and RF IgA at 12 months ($p=0.004$).

Using multivariate logistic regression methods we obtained a statistical model for predicting the response at 6 months including normal values for 14–3–3eta protein, anti-CCP and COMP (Hosmer and Lemeshow according test $\chi^2=5.795$, $p=0.670 \geq 0.05$ with a predictive response accuracy of 89.1%).

Smoking had a negative impact on the EULAR response as well as on 2 groups response at 6 months of treatment ($p=0.017$).

	6 Months response nonresponders	6 Months response moderate	6 Months response good
Smoking	2/7 (28.6%)	29/38 (76.3%)	17/19 (89.5%)
No	0/6 (0.0%)	3/38 (7.9%)	1/19 (5.3%)
Past	5/7 (71.4%)	6/38 (15.8%)	1/19 (5.3%)
Yes			

0.046 (Likelihood Ratio corrected Bonferroni)

Following the evolution of serum levels, we noticed a reduction for all biomarkers tested, statistically significant at 6 and/or 12 months from baseline.

Abstract FRI0104 – Table 1. Description of ETA-SB4-ETA back-switchers (n=120)

	Nonresponder	Moderate response	Good response	p value	Nonresponder	Responder	p value
No.	7	38	19		7	57	
RF type	132.35±99.602	157.22±131.47	51.36±95.359	0.016	132.35±99.602	121.93	0.571
Ig M						±129.92	
RF type	122.81±99.876	102.08±128.33	22.45±61.256	0.033	122.81±99.87	75.54	0.307
Ig A						±116.282	
Anti-MCV	74.04±47.951	80.06±149.543	33.77±113.069	0.459	74.04±47.951	64.63	0.860
14–3–3 eta protein	0.99±0.888	0.28±0.469	0.51±0.580	0.045	0.99±0.888	±139.174	
Anti-CCP	146.16±41.688	113.65±50.448	60.82±26.331	0.000	146.16±41.68	0.36±0.515	0.040
COMP	1042.2±181.71	1032.8±188.67	746.04±130.09	0.000	1042.2±181.717	937.27	0.028
						±218.10	0.227

Conclusions: In the future a version using multiple biomarkers could increase accuracy for identifying pretreatment patients who will respond to anti-TNF therapy. Smoking has a negative impact on the response to biologic treatment.

Disclosure of Interest: None declared

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FRI0105 CONCOMITANT USE OF CORTICOSTEROIDS AT THE BASELINE DOES NOT AFFECT THE DRUG SURVIVAL OF ABATACEPT IN RHEUMATOID ARTHRITIS

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Background: Rheumatoid arthritis (RA) is a chronic inflammatory disease leading to deformities and disabilities. In the treatment of RA glucocorticoids are selected sometimes to relieve symptoms and to increase compliance for treatment.

Objectives: The purpose of our study is to investigate whether concomitant glucocorticoid treatment at the baseline affects drug survival for abatacept treatment in RA.

Methods: Data on patient characteristics, diagnosis, previous treatment and outcomes have been collected since 2011 in Turkish Biologic (TURKBIO) Registry. By the end of December 2017, 338 RA patients, received abatacept from the TURKBIO registry, were included in the analysis. Patients were divided into groups according to the use of glucocorticoid when abatacept therapy was started. Demographic and clinical data including age, sex, disease type, disease duration, and previous or current treatment with DMARDs and biological drug durations are recorded in the database. Kaplan-Meier survival analysis was performed to estimate the drug survival. Subgroups were compared by log-rank.

Results: There were no significant differences in age, gender, seropositivity, tender and swollen joint counts at baseline in the study groups. The disease duration was higher in the glucocorticoid users ($p=0.001$). Abatacept was the first choice bDMARDs in the 44.5% of glucocorticoid users while it was 68.6% in the glucocorticoid non-users ($p<0.001$). In addition to abatacept, use of sDMARDs were 96.7% and 53.8% in the glucocorticoid users and non-users, respectively. Baseline VAS-pain and ESR were higher in the glucocorticoid non-users ($p=0.047$, $p=0.009$, respectively), but other baseline parameters were similar in glucocorticoid users and non-users. There was no difference between groups in terms of drug survival rates for abatacept (figure 1).

Abstract FRI0105 – Table 1. Clinical and laboratory characteristics

	Glucocorticoid non-users (n=156)	Glucocorticoid users (n=182)	p
Age, year	56 (47–64)	57 (47–63)	0.827
Gender (Females), n (%)	135 (86.5)	151 (82.9)	0.390
Disease duration, years	10 (5–15)	13 (8–18)	0.001
1 st choice bDMARDs is abatacept, %	68.6%	44.5%	<0.001
Concomitant sDMARD usage, %	53.8%	96.7%	<0.001
RF positivity, n (%)	68.1	65.8	0.735
CCP positivity, n (%)	61.8	66.7	0.485
Baseline ESR, mm/h	38 (24–55)	28 (18–44)	0.009
12th month ESR, mm/h	33 (20–50)	27 (16–39)	0.155
Baseline CRP, mg/dl	12 (5–21)	12 (4.89–30.9)	0.870
12th month CRP, mg/dl	4 (3–8)	9 (3–19)	0.020
Baseline DAS28-CRP	4.95 (4.2–5.4)	4.8 (3.9–5.35)	0.123
12th month DAS28-CRP	2.05 (1.7–2.9)	2.6 (2.1–3.9)	0.002
Baseline CDAI	22 (13.8–28.3)	19.8 (13.2–27)	0.278
12th month CDAI	3.2 (2–7.95)	6.3 (3–10.7)	0.061

Abstract FRI0105 – Figure 1. Drug survival curve in glucocorticoid user and non-user patients

Conclusions: When abatacept treatment started, concomitant use of glucocorticoid at the baseline could not significantly alter drug survival for abatacept in the RA.

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FRI0106 IMPACT OF BIOLOGICAL AND TARGETED SYNTHETIC DMARDs ON WORK IN PATIENTS WITH CHRONIC INFLAMMATORY ARTHRITIS : A META ANALYSIS OF RANDOMISED CONTROLLED TRIALS AND CONTROLLED COHORTS

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Background: The addition of biological (b) and new targeted synthetic (ts) DMARDs agents in chronic inflammatory arthritis (CIAs) therapeutic strategies