



Figure 1 Mean V from baseline in DAS28-ESR (OLE population; as observed). **Conclusions:** Switching from adalimumab (40 mg q2w) to sarilumab monotherapy (200 mg q2w) improved the signs and symptoms of RA to a similar level as continuous sarilumab treatment, and was associated with a lower HAQ-DI score, which may have resulted in numerical differences in ACR responses between the two groups.

Acknowledgements: Study funding and medical writing support (Vicki Cronin, Adelphi) provided by Sanofi and Regeneron Pharmaceuticals, Inc.

Disclosure of Interest: G. R. Burmester Grant/research support from: AbbVie, Pfizer, UCB, Roche, Consultant for: AbbVie, Lilly, Merck Sharpe & Dohme, Pfizer, Sanofi, Roche, UCB, Speakers bureau: AbbVie, Lilly, Merck Sharpe & Dohme, Pfizer, Sanofi, Roche, UCB, G. St John Shareholder of: Regeneron, Employee of: Regeneron, M. Iglesias-Rodriguez Employee of: Sanofi, C.-C. Hu Shareholder of: Sanofi, Consultant for: Astellas, Employee of: Sanofi, Quintiles, T. Raskina: None declared, H. Amital Grant/research support from: Pfizer, Abbvie, Yansen, Consultant for: Pfizer, Merck Sharpe & Dohme, Speakers bureau: Pfizer, Merck Sharpe & Dohme, Yansen, Sanofi, Bristol-Myers Squibb, A. Gomez Centeno Grant/research support from: Boehringer Ingelheim, Celltrion, Galapagos-Gilead, Lilly, Novartis, Pfizer, Roche, Sanofi, UCB, YL Biologics, Consultant for: Abbvie, Biogen, Bristol-Myers Squibb, Celgene, Gebro, Hospira, Lilly, Merck Sharpe & Dohme, Pfizer, Roche, Rubio, Sandoz, Sanofi, Speakers bureau: Abbvie, Bristol-Myers Squibb, Gebro, Janssen, Lilly, Menarini, Merck Sharpe & Dohme, Pfizer, Roche, Rubio, UCB, Sanofi, A. Rubbert-Roth Consultant for: Abbvie, Bristol-Myers Squibb, Chugai, Roche, Merck Sharpe & Dohme, Pfizer, Lilly, Hexal/Novartis, Janssen, Sanofi, Speakers bureau: Roche, Chugai, Sanofi, Lilly
DOI: 10.1136/annrheumdis-2018-eular.1372

the analysis. Treatment response was evaluated as achievement of “BASDAI50” or “ASDAS Clinically important improvement (CII)” at the 3-months’ and 6 months’ visits. Clinical and demographic parameters were compared between current/never and current/previous smoker groups. Demographic and descriptive data are presented by medians/interquartile ranges (IQRs). Groups were compared by non-parametric tests (χ^2 , Kruskal Wallis and Mann Whitney tests). Kaplan Meier plots, Cox and logistic regression analyses were calculated for treatment adherence and treatment response.

Results: Among 561 AS patients included in the study, 506 (90%) had known smoking status (37% current, 35% never, 17% previous smokers). The median follow-up time was 1.9 years (IQR 0.85–3.5) and disease duration was 3.1 years (0.6–7.7). At baseline, current smokers were younger (34, IQR 29–41) compared with never (38, IQR 30–46 $p=0.007$) and previous smokers (42, IQR 34–49 $p<0.001$). Current smokers had male predominance ($n=148$, 43.9%; $n=85$, 25.2%); lower erythrocyte sedimentation rate (28 mm/h (13–42); 34 mm/h, (20–49) and higher change in BASMI (40, IQR 10–57.5; 10, IQR 4–30) compared with never smokers (all $p<0.005$). HLA status, body mass index, CRP, baseline disease indexes (BASDAI, BASFI, BASMI, HAQ, ASDAS) and treatment response was not found to be different between current and never smoker patients in our population (table 1). In multivariate analysis, male (OR:1.98; 95% CI (1.39–2.82), $p<0.01$), HLA positive (OR:1.54; 95%CI (1.08–2.18), $p=0.016$) and active DMARD user (OR:1.84; (95%CI 1.12–3.01) $p=0.015$) patients had better treatment response and treatment adherence (HR:1.93; 95% CI (1.36–2.73); HR:1.60; 95% CI (1.13–2.27); HR:1.80; 95% CI (1.10–2.95) all $p<0.005$) but smoking status were not significant ($p>0.05$).

	Smoking status				p ^a	p ^b	Smoking status unknown	Smoking status				p ^a	p ^b	Smoking status
	Current	Never	Previous	n				Current	Never	Previous	n			
Number, n (%)	209 (37)	199 (35.5)	98 (17.5)	55 (10)	0.007	<0.001	38 (29-46)	245 (43.5)	209 (37.5)	116 (20.5)	0.007	0.63	30 (14.4)	
Age, median (IQR), years	34 (29-41)	38 (30-46)	42 (34-49)	40 (34-49)	0.13	0.48	0.1 (0-46)	45 (34.5-46)	46 (34.5-46)	52 (34.5-46)	0.9	0.3	60 (34.5)	
Disease duration, median (IQR), years	3.51 (0.5-9.36)	3.55 (0.5-9.36)	3.45 (0.5-9.36)	4.05 (0.6-8.2)	0.13	0.48	0.1 (0-46)	26 (13-25)	25 (13-25)	25 (13-25)	0.47	0.4	48 (25.7-43)	
Women, n (%)	61 (27.2)	114 (56.3)	22 (9.8)	28 (8.3)	<0.001	0.37	27 (12.1)	40 (28.5)	39 (28.5)	50 (28.5)	0.11	0.8	30 (20.5-15)	
Men, n (%)	148 (43.9)	85 (25.2)	76 (22.6)	28 (8.3)				105 (46.5)	70 (31.5)	66 (38)			30 (20.5-15)	
HLA positive, n (%)	84 (37.2)	117 (58.8)	14 (23.8)	23 (4.2)	0.4	0.23	0.4	12 (5.3)	17 (12.5)	14 (8)	0.3	0.3	15 (10.5)	
Body Mass Index, kg/m ² , median (IQR)	25.05 (22.8-28.4)	26.6 (23.3-29.5)	25.8 (22.3-29.5)	25.4 (25.4-25.4)	0.19	0.84	25.4 (25.4-25.4)	25.05 (22.8-28.4)	26.6 (23.3-29.5)	25.8 (22.3-29.5)	0.11	0.8	30 (20.5-15)	
TNFi drug type, n (%)	209 (37.3)	199 (35.5)	98 (17.5)	55 (10)	0.4	0.37	55 (9.8)	209 (37.3)	199 (35.5)	98 (17.5)	0.4	0.37	21 (14.9)	
Adalimumab	49 (34.8)	54 (28.3)	17 (12.1)	13 (9)			13 (9)	49 (34.8)	54 (28.3)	17 (12.1)			11 (8.3)	
Etanercept	60 (41.4)	45 (23.1)	27 (18.6)	13 (9)			13 (9)	60 (41.4)	45 (23.1)	27 (18.6)			11 (8.3)	
Infliximab	48 (36.3)	39 (26.3)	35 (26.3)	11 (8.3)			11 (8.3)	48 (36.3)	39 (26.3)	35 (26.3)			11 (8.3)	
Goldimab	38 (37.6)	42 (41.8)	14 (13.9)	7 (6.9)			7 (6.9)	38 (37.6)	42 (41.8)	14 (13.9)			7 (6.9)	
Certolizumab	14 (14.1)	15 (16.1)	1 (1.2)	2 (2.1)			2 (2.1)	14 (14.1)	15 (16.1)	1 (1.2)			2 (2.1)	
Methotrexate use, n (%)	12 (38.7)	7 (22.6)	7 (22.6)	0 (0)	0.28	0.87	5 (16.1)	12 (38.7)	7 (22.6)	7 (22.6)	0.28	0.87	5 (16.1)	
DMARD use, n (%)	12 (21.1)	22 (38.6)	13 (22.8)	0 (0)	0.05	0.58	12 (21.1)	12 (21.1)	22 (38.6)	13 (22.8)	0.05	0.58	12 (21.1)	
Methotrexate	6 (33.3)	9 (50)	3 (16.7)	0 (0)			0 (0)	6 (33.3)	9 (50)	3 (16.7)			0 (0)	
Sulfasalazine	5 (27.8)	7 (38.9)	4 (21.1)	0 (0)			0 (0)	5 (27.8)	7 (38.9)	4 (21.1)			0 (0)	
Protoninone	1 (5.6)	2 (11.1)	1 (5.6)	0 (0)			0 (0)	1 (5.6)	2 (11.1)	1 (5.6)			0 (0)	
Hydroxychloroquine	0 (0)	1 (5.6)	1 (5.6)	0 (0)			0 (0)	0 (0)	1 (5.6)	1 (5.6)			0 (0)	
Azathioprine	0 (0)	2 (11.1)	2 (11.1)	0 (0)			0 (0)	0 (0)	2 (11.1)	2 (11.1)			0 (0)	

Conclusions: In this study of TNFi-treated AS patients in clinical practice, smoking was not found to be associated with disease activity, treatment response and treatment adherence.

Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2018-eular.6586

SAT0184 THE EFFECT OF SMOKING ON RESPONSE TO TUMOR NECROSIS FACTOR-ALPHA INHIBITOR TREATMENT IN ANKYLOSING SPONDYLITIS PATIENTS: RESULTS FROM THE TURKBIO REGISTRY

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Background: Although there is good evidence that smoking has a dose-dependent impact on structural damage progression in ankylosing spondylitis (AS) the evidence is poor for its impact on disease activity, physical mobility, life quality and treatment response.

Objectives: We aimed to investigate the impact of smoking on disease activity, treatment adherence and treatment response in Turkish patients with AS treated with their first tumour necrosis factor-alpha inhibitor (TNFi) therapy in a real-life cohort.

Methods: 561 patients fulfilling the modified New York criteria for AS and treated with their first TNFi therapy since 2011 from 8 centers in Turkey were included in

SAT0185 CERTOLIZUMAB PEGOL SERUM LEVELS ≥20 MG/L ARE ASSOCIATED WITH IMPROVEMENT IN DAS28 IN RHEUMATOID ARTHRITIS PATIENTS. DATA FROM THE NOR-DMARD STUDY

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Background: Measurement of serum drug levels can help clinicians tailor treatment with TNF-inhibitors. An association between certolizumab pegol (CP) serum levels and treatment response in rheumatoid arthritis (RA) patients (pts) has previously been demonstrated in a prospective observational study (1). These results need to be confirmed in other studies, with particular focus on finding an optimal therapeutic serum level for CP.

Objectives: To examine the association between serum CP drug levels and treatment response in RA pts and to identify a therapeutic target level.

Methods: Patients with a clinical diagnosis of RA starting standard treatment with CP included in the NOR-DMARD registry with biobank sample at 3 months follow-up, were included in the present analyses. Serum drug levels (non-trough) were analysed with an in-house immunofluorometric assay automated on the AutoDELTA immunoassay platform. We studied association between serum CP level and ΔDAS28 and EULAR good/moderate response at 3 months by multi-variable linear and logistic regression analyses, respectively, adjusting for age, sex and prior bDMARD (Y/N).

Results: In 91 included patients, median serum drug level at 3 months follow up was 34.7 mg/L (17.6–44.6). Response data were available in 81/91 patients. Serum CP level ≥20 mg/L was associated with greater improvement in DAS28 at