gender-matched patients with RA. The results were analysed with respect to clinical and laboratory parameters such as data on GC treatment (frequency, duration defined as start of treatment until timepoint of measurement, actual and cumulative dose), csDMARD and bDMARD (including as well tsDMARDs) therapy, serological parameters (Vitamin D, alkaline phosphatase, calcium, inflammatory markers and rheumatoid factor) and functional status (e.g. Health Assessment Questionnaire (HAQ), sporting activities). Statistical analyses were performed descriptively using mean and standard deviation, t-tests for metric variables, and chi-square tests for nominal variables. For subgroup analyses with less than 30 patients per group, tests for non-normally distributed data were used due to the lower test power.

Results: RA patients showed significantly lower means of bone density values (minimal T-score, $p=0.03$) than PsA patients leading to a higher frequency of osteopenic bone densities ($p<0.005$). However, no differences in the frequency of osteoporotic bone densities could be detected. PsA patients reported a significantly longer disease duration and a higher current GC dosage. In contrast, the frequency of current GC intake was higher in RA patients. Although the calcium intake was higher in the RA group, neither blood levels of calcium and vitamin D, nor the cumulative GC dose (GCCD) or duration of GC therapy could indicate a causal relationship for the differences observed in bone density values between the two groups. The frequency of csDMARD therapy did not differ significantly between PsA and RA patients while the frequency of bDMARD therapy was higher in the PsA group ($p=0.04$).

Conclusion: The lower bone density in RA patients seems not to be fully explained by higher GCCD, disease duration or higher levels of inflammation. However, RA patients had a higher frequency of current GC intake. Additionally, differences in bone density between the two groups could be related to the higher number of bDMARD therapies in PsA patients, but further investigations like multivariate analyses with higher numbers of patients are necessary. Furthermore there is more need for research on possible molecular and genetic factors in PsA, which are protecting from low bone density.

Disclosure of Interests: Desiree Freier: None declared, Edgar Wiebe: None declared, Robert Biesen: None declared, Thomas Buttgerit: None declared, Sandra Hermann: None declared, Timo Gaber: None declared, Frank Buttgerit Grant/research support from: Amgen, BMS, Celgene, Generic Assays, GSK, Hexal, Horizon, Lilly, medac, Mundipharma, Novartis, Pfizer, Roche, and Sanofi. DOI: 10.1136/annrheumdis-2020-eular.3447

AB0768

TREATMENT WITH TOFACITINIB IN REFRACTORY PSORIATIC ARTHRITIS. MULTICENTER STUDY OF 87 PATIENTS IN CLINICAL PRACTICE

E. Galindez¹, D. Prieto-Peña², J. L. Martín-Varillas², B. Joven-Ibáñez², O. Rusinovich², R. Almodovar², J. J. Alegre-Sancho², L. Mendez Díaz², A. Sellas-Fernández², A. Martínez-Ferrer², R. García de Vicuña², C. Ventín-Rodríguez², J. Ramírez², M. Moreno², M. J. Moreno², M. D. C. Castro Villegas², A. Crespo Golmar², N. Palmou-Fontana², F. Ortiz Sanjuan², X. E. Larco Rojas², A. J. Mas², C. Y. Soletto², I. Gorostiza¹, M. A. González-Gay², R. Blanco². ¹HU Basurto, Bilbao, Spain; ²NHS, Madrid, Spain

Background: Tofacitinib (TOFA) is the first JAKi approved for psoriatic arthritis (PsA) in Europe (July 2018). TOFA has shown efficacy in refractory patients to anti-TNF in Randomized Clinical Trials (RCT) (Gladman D. *NEJM* 2017; 377: 1525-36).

Objectives: To assess efficacy and safety of TOFA in clinical practice (CP). To compare the profile of CP with RCT

Methods: Study of 87 patients of CP with PsA treated with TOFA; Results are expressed as percentage, mean±SD or median [IRQ].

Results: 87 patients (28♀/59♂), mean age of 52.8±11.4 years (Table 1). Pattern of joint involvement was: peripheral (n=60), axial (1) and mixed (26). Presented also enthesitis (49.4%), nail involvement (30.2%) and dactylitis (31%).

Prior TOFA, most patients (80%) received oral prednisone, synthetic immunosuppressants (mean 2.3±0.9) and biological therapy (BT) (3.6±1.9): etanercept (n=58), adalimumab (54), infliximab (31), golimumab (37), certolizumab (30), secukinumab (54), ustekinumab (39) and ixekizumab (2). Apremilast was used in 17.

After a mean follow-up of 12.3±9.3 years from PsA diagnosis, TOFA was started (5mg/12h). In 48 (55.2%) TOFA was used in combined therapy: methotrexate (30) and leflunomide (15). In the remaining 39, monotherapy was prescribed. Patients of CP compared with RCT have a longer duration of PsA, worst functional disability (HAQ) and received a higher proportion of corticosteroids and BT (anti-TNF and non-anti-TNF) (Table 1).

Patients improved in activity indexes (PASI, DAS28, DAPSA) and laboratory test (table 2). Minor side effects were reported in 21 patients (gastrointestinal symptoms), and TOFA was discontinued in 29 due to inefficiency mainly.

Conclusion: Patients of CP had a longer evolution and received a greater number of biologics than those of RCT. TOFA as in RCT seems effective, rapid and relatively safe for refractory PsA.

Table 1. Baseline features

	CLINICAL TRIAL Gladman N=131	CLINICAL PRACTICE N=87
Age, years (mean±SD)	49.5±12.3	52.8±11.4
Sex, n (%)	67M/64F (51/49)	59M/28F (68/32.2)
Duration PsA, years (mean±SD)	9.6±7.6	12.3±9.3
HAQ-DI	1.3±0.7	1.4±0.7 (n=26)
Swollen joint count, mean±SD	12.1±10.6	5.7±5.8
Painful joint count, mean±SD	20.5±13.0	8.0±6.6
Elevated CRP, n (%)	85 (65)	55 (63.2)
PASI score, median [IQR]	7.6 [0.6-32.2]	9.0 [4.2-15]
Oral glucocorticoid, n (%)	37(28)	44(50.5)
Concomitant synthetic DMARDs, n (%)		
- Methotrexate	98 (75)	30 (34.4)
- Leflunomide	12 (9)	15 (17.2)
- Sulfasalazine	21 (16)	6 (6.9)
- Others	2 (2)	
N. of previous TNF inhibitors, mean±SD	1.7±1.0	2.4±1.4
Previous use of other biological no anti-TNF, n (%)	11 (8)	68 (78.2)

Table 2. Improvement at 1st, 6th and 12th month

	Baseline n=87	1st month n=77	6th month n=52	12th month n=20
Nail involvement, n (%)	17 (19.5)			
Improvement, n (%)		5 (35.7)	6 (60)	5 (83.3)
Enthesitis, n (%)	28 (32.2)			
Improvement, n (%)		8 (47.1)	10 (58.8)	3 (50)
Dactylitis, n (%)	16 (18.4)			
Improvement, n (%)		9 (69.2)	6 (85.7)	0 (0)
CRP mg/dl, median [IQR]	1.9 [0.3-5]	0.5 [0.1-2.2]	0.5 [0.3-1.2]	0.4 [0.4-3.7]
p (vs baseline)		0.004	0.005	0.66
DAS28, median [IQR]	4.8 [4.1-5.40]	3.7 [2.8-4.6]	2.8 [2.2-3.8]	2.9 [2.2-3.7]
p (vs baseline)		<0.001	<0.001	<0.001
DAPSA, median [IQR]	28 [18.41-34.05]	15.5 [10.1-25.7]	9 [6.07-15]	4.3 [2.4-8]
p (vs baseline)		<0.001	<0.001	<0.001
PASI, median [IQR]	5 [1-14]	1.4 [0-7]	0 [0-4]	0.05 [0-2.7]
p (vs baseline)		0.192	0.105	0.300

Disclosure of Interests: E. Galindez: None declared, D. Prieto-Peña: None declared, José Luis Martín-Varillas Grant/research support from: AbbVie, Pfizer, Janssen and Celgene, Speakers bureau: Pfizer and Lilly, Beatriz Joven-Ibáñez Speakers bureau: AbbVie, Celgene, Janssen, Merck Sharp & Dohme, Novartis, Pfizer, Olga Rusinovich: None declared, RAQUEL ALMODOVAR Speakers bureau: AbbVie, Celgene, Janssen, Lilly, Novartis, Pfizer.

, Juanjo J Alegre-Sancho Consultant of: UCB, Roche, Sanofi, Boehringer, Celltrion, Paid instructor for: GSK, Speakers bureau: MSD, GSK, Lilly, Sanofi, Roche, UCB, Actelion, Pfizer, AbbVie, Novartis, LARA MENDEZ DIAZ: None declared, Agusti Sellas-Fernández Speakers bureau: Abbott, Lilly, Celgene, Pfizer, Schering-Plough, Janssen, Novartis, and Nordic Pharma, A Martínez-Ferrer: None declared, Rosario García de Vicuña Grant/research support from: BMS, Lilly, MSD, Novartis, Roche, Consultant of: AbbVie, Biogen, BMS, Celltrion, Gebro, Lilly, Mylan, Pfizer, Sandoz, Sanofi, Paid instructor for: Lilly, Speakers bureau: BMS, Lilly, Pfizer, Sandoz, Sanofi, Clara Ventín-Rodríguez: None declared, Julio Ramírez: None declared, Manuel Moreno: None declared, Maria Jose Moreno: None declared, María del Carmen Castro Villegas: None declared, Antia Crespo Golmar: None declared, Natalia Palmou-Fontana: None declared, FRANCISCO ORTIZ SANJUAN: None declared, Ximena Elizabeth Larco Rojas: None declared, Antonio Juan Mas: None declared, Christian Y Soletto: None declared, Iñigo Gorostiza: None declared, Miguel A González-Gay Grant/research support from: Pfizer, AbbVie, MSD, Speakers bureau: Pfizer, AbbVie, MSD, Ricardo Blanco Grant/research support from: AbbVie, MSD, and Roche, Speakers bureau: AbbVie, Pfizer, Roche, Bristol-Myers, Janssen, and MSD

DOI: 10.1136/annrheumdis-2020-eular.2903

AB0769

THE IMPACT OF BODY MASS INDEX ON DISEASE ACTIVITY AND ENTESITIS IN PSORIATIC ARTHRITIS

S. Ganhão¹, B. M. Fernandes¹, S. Garcia¹, F. Pinheiro¹, M. Rato¹, E. Mariz¹, M. Bernardes^{1,2}, L. Costa¹. ¹Centro Hospitalar e Universitário de São João, Rheumatology, Oporto, Portugal; ²Faculty of Medicine of Oporto's University, Oporto, Portugal

Background: Overweight/obesity has increased exponentially in the last decades, becoming a huge Public Health problem. Moreover, an increase in adipose tissue is associated with an increased production of several proinflammatory cytokines and acute phase reactants. Higher BMI has been related with new bone formation including syndesmophytes and enthesophytes. In fact, besides