Results: The following features proved to be the most informative: male sex ($p = 0.300$), presence of HLA B27 ($p = 0.107$), mild or high DAS ($p = 0.098$), skin lesion severity BSA>3% ($p = 0.118$), and CRP > 5 mg/L (0.038). Sensitivity of model 79.0%, specificity of model 57.7%. Area under ROC curve 0.756; 95% CI (0.642-0.869).

Conclusion: It is a combination of features – male sex, HLA-B27 positivity, mild or high activity of peripheral arthritis according to DAS, CRP > 5 mg/L, and BSA> 3% – that constitutes a clinical predictor for the development of axial involvement in early psoriatic arthritis.

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FRI0342

POST-EFFORT SYNOVIAL AND ENTHESIC RESPONSE IN PATIENTS WITH PSORIATIC ARTHRITIS IN REMISSION OF LOW DISEASE ACTIVITY

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Background: Synovial inflammation is part of the pathophysiological process of PsA although it is considered secondary to enthesitis. It is of interest to assess the synovial joint, peritendinous and enthesic response of patients with PsA in remission under controlled mechanical stress.

Objectives: Our aim is to present our observational experience in patients with APso in remission or low activity of the disease exposed to controlled manual physical efforts adapted to the own grasping capacity of each subject.

Methods: Before-after study of a consecutive cohort of patients with PsA (CASPAR criteria), of at least two years evolution and DAPSA₁₄ at present. Patients with positive rheumatoid factor, patients with exclusively axial forms and patients on biological therapy at the beginning of the study were excluded. All patients underwent controlled basal ultrasound and post-dynamometric exercise (CAMRY EH101-17) of the dominant hand which included the carpus, MCFs, IFPs and IFDs of the 2nd to 5th fingers. The ultrasound findings were scored according to EULAR recommendations in grey scale (GS) and power Doppler (PD) for synovitis, enthesitis and tenosynovitis (maximum scores 71 and 87, respectively). For statistical analysis, comparisons were made with the results of their baseline and post-exercise ultrasound scores between subjects diagnosed with PsA and controls. The Student's T test was used for related and unrelated data according to correspondence.

Results: Nineteen patients and controls were included, of which 73.7% were male. Mean age: 42.2 SD 6.6 and 42.21 SD 8.28, respectively. Basal DAPSA among patients: 7.26 SD 4.53. Mean traction strength of patients and controls: 23.8 SD 7.3 and 26.1 SD 6.9 kg, respectively ($P=0.336$). In the group of patients, the post exercise DAPSA had a mean of 7.52 SD 4.62 ($P=0.021$, with respect to the basal DAPSA). The mean total GS score in the patient group was 3.94 SD 2.36 and 7.31 SD 3.3, pre- and post-exercise, respectively ($P<0.001$). The mean total score in the PD study was 0.73 SD 0.73 and 2.57 SD 1.16, respectively ($P<0.001$). In patients with PsA there were no detectable enthesic changes. In the control group, no ultrasound changes were statistically significant, although the score for tenosynovitis ranged from 0.1 SD 0.31 to 0.42 SD 0.6 ($P=0.055$).

Conclusion: Patients with APso in clinical remission or low disease activity develop ultrasound changes after controlled exercise. These changes are appreciable in the joint synovium and tendon but not at an enthesic level. Although these changes are also detectable in a healthy population, their prevalence is much lower. Our interpretation is that control of the disease correlates with an absence of enthesic inflammatory activity although synovial susceptibility remains less evident. This reinforces the idea that PsA is an inflammatory enthesitis with associated arthritis and not its opposite.

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FRI0343

EFFICACY AND SAFETY OF FILGOTINIB, A SELECTIVE JANUS KINASE 1 INHIBITOR, IN PATIENTS WITH ACTIVE PSORIATIC ARTHRITIS: SUBGROUP ANALYSES FROM A RANDOMIZED, PLACEBO-CONTROLLED, PHASE 2 TRIAL (EQUATOR)

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Background: Treatment with the oral selective Janus kinase 1 inhibitor filgotinib was associated with rapid and significant improvements in multiple domains of active psoriatic arthritis versus placebo in the 16-week Phase 2, multicenter, double-blind, randomized EQUATOR trial (NCT03101670).¹ A significantly greater proportion of patients receiving filgotinib, versus placebo, achieved the primary endpoint of 20% improvement in American College of Rheumatology (ACR) 20 response at Week 16 (80% vs 33%, respectively).¹

Objectives: The aim of this predefined analysis was to evaluate the consistency of the response to filgotinib across predefined relevant subpopulations participating in the EQUATOR trial.

Methods: In EQUATOR, patients with active psoriatic arthritis were treated with filgotinib 200 mg (n=65) or placebo (n=66) once daily for 16 weeks. Key clinical endpoints, including ACR20 and ACR50 (50% improvement) response rates, Psoriatic Arthritis Disease Activity Score (PASDAS), and Disease Activity Index for Psoriatic Arthritis (DAPSA) were evaluated according to the following baseline characteristics: sex, body mass index, disease duration, baseline disease severity, concurrent use of disease-modifying antirheumatic drug(s), and prior exposure to tumor necrosis factor inhibitor(s). For PASDAS and DAPSA scores, statistical analysis of changes from baseline was performed using analysis of covariance with factors for treatment, randomization stratification, subgroup, and an interaction between treatment and subgroup. Least-squares (LS) mean difference between treatment arms and the corresponding 95% confidence intervals (CI) were calculated. For ACR20 and ACR50 response rates, statistical analysis used the point estimate and corresponding 95% CI, based on the Newcombe method.

Results: Sixty patients (92%) in the filgotinib group and 64 (97%) in the placebo group completed the study. The total number of patients in each subpopulation ranged from 18 to 104 (Figure 1). Differences in the proportions of patients achieving ACR20 consistently favored filgotinib, compared with placebo, across all subgroups (Figure 1); all differences reached statistical significance. Similarly, differences in the proportions of ACR50 responders and LS mean treatment differences for PASDAS and DAPSA consistently favored filgotinib, reaching statistical significance in most subgroups. No clinically relevant differences in the effect of filgotinib were observed across subgroups. Filgotinib was generally well tolerated and no new safety signals were identified.

Figure 1. Effect of filgotinib on ACR20 response across subgroups.

ACR20, 20% improvement in American College of Rheumatology response criteria; BMI, body mass index; DMARD, disease-modifying antirheumatic drug; PASDAS, Psoriatic Arthritis Disease Activity Score; TNF, tumor necrosis factor

Conclusion: In the 16-week EQUATOR trial, the effects of filgotinib on key efficacy endpoints were generally consistent across a range of subgroups based on patient, disease, and treatment characteristics.

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[1] Mease P, et al. *Lancet* 2018;392:2367–77.

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FRI0344

THE LONG-TERM EFFECT OF TREATING PSORIATIC ARTHRITIS WITH THE JANUS KINASE 1-SELECTIVE INHIBITOR FILGOTINIB ON LIPID PROFILES: AN ANALYSIS OF THE EQUATOR AND EQUATOR2 TRIALS

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Background: Cardiovascular (CV) comorbidities are common in psoriatic arthritis (PsA); patients are at high risk for major adverse cardiovascular events (MACE).¹ In the Phase 2, double-blind, randomized EQUATOR trial, significant improvements across multiple PsA domains were observed with the oral selective Janus kinase (JAK) 1 inhibitor filgotinib compared with placebo.² Inhibition of JAK signal transducer and activator of transcription signaling is associated with raised serum lipids.³

Objectives: To evaluate the effects of filgotinib on the lipid profile of PsA patients and determine if those with higher MACE risk show similar changes in lipid profile compared with the overall population.

Methods: In EQUATOR, 131 patients with active PsA received filgotinib 200 mg (n=65) or placebo (n=66) once daily for 16 weeks. Patients completing EQUATOR could enter the ongoing EQUATOR2 open-label extension (OLE; NCT03320876), in which patients receive filgotinib 200 mg for up to 148 weeks. Effects of filgotinib on total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and TC/HDL-C ratio at OLE Week 52 (68 weeks after EQUATOR initiation) were analyzed. In a post hoc analysis, patients were classified into subgroups according to presence/absence of obesity (baseline body mass index [BMI]; ≥ 30 vs <30 kg/m², respectively), diabetes mellitus, arterial hypertension ($\geq 130/80$ mmHg), hyperlipidemia, and metabolic syndrome. Changes in lipid levels were explored graphically.

Results: 124 patients (95%) completed EQUATOR; 122 (93%) enrolled in the OLE. Of these, 11 patients (9%) discontinued treatment by OLE Week 52. Median (range) exposure to filgotinib was 66.0 (0.4–104.1) weeks. In the OLE, TC, LDL-C, and HDL-C levels increased versus baseline with filgotinib, resulting in a decreased TC/HDL-C ratio. Changes in lipid levels were consistent irrespective of presence of obesity (n=56; **Fig**), diabetes (n=53), arterial hypertension (n=80), hyperlipidemia (n=108), or metabolic syndrome (n=36); baseline lipid values were greater in the higher risk groups. In patients who were assigned placebo in the randomized controlled trial (RCT), HDL-C increased on switching to

filgotinib in the OLE (in a manner similar to that seen in filgotinib-treated patients during the RCT), and remained elevated compared with baseline (**Fig**). Triglyceride levels remained stable throughout, across all subgroups. Seventeen patients (13%) were taking lipid-lowering drugs (LLDs) prior to the start of the trial (and continued to do so); the effect of filgotinib on the lipid profile in these patients was similar to that in the overall population. During the RCT phase, another six patients in the filgotinib group and one in the placebo group began taking LLDs.

Conclusion: In patients exposed to filgotinib for ≥ 52 weeks, the effects on lipid profile were consistent regardless of baseline CV risk. Lipid changes included an elevation in TC and HDL-C, with a decrease in TC/HDL-C ratio.

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Figure 1. Change from baseline in lipid profile in non-obese and obese patients

BL, baseline; BMI, body mass index; FIL, filgotinib; HDL-C, high-density lipoprotein cholesterol; OLE, open-label extension; PBO, placebo; TC, total cholesterol; W, Week

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