Methods: Pts with baseline PASI and BSA available from ADEPT, a phase 3, randomized, double-blind, PBO-controlled trial of ADA 40 mg every other week, were included in this post hoc analysis.2 Pts were sub-grouped based on baseline PsO severity (severe PsO: PASI >12 OR BSA >10%; mild-to-moderate PsO: PASI <12 AND BSA <10%), relative to overall study population. Clinical outcomes at wk 24 were assessed by attainment of minimal disease activity (MDA), remission and low disease activity (LDA) by disease activity index for psoriatic arthritis (DAPSA) and modified total Sharp score (mTSS)7 functional and structural progression were assessed by % of pts experiencing HAQ-DI normative values (<0.25) and improvement from baseline in mTSS <0.5 in the modified total Sharp score (mTSS) respectively. A logistic model with treatment group baseline PASI/BSA and PASI/BSA by treatment interaction was fit for each outcome variable. P-values for interaction term and baseline BSA/PASI were calculated. Non-responder imputation was used for missing clinical and functional endpoints. 75th percentiles were imputed for missing radiographic data. Treatment-emergent adverse events (TEAEs) were monitored throughout.

Results: Of the 163 pts enrolled in the study with baseline PASI and BSA, only 23% (n=37; ADA: 17; PBO: 20) were classified as having severe PsO and had similar characteristics to those with mild-to-moderate PsO, except for numerically higher physician's global assessment, dactylitis, and enthesitis scores. Following 24 wks of treatment, 41% of ADA-treated pts achieved MDA and 45% achieved DAPSA LDA or better, a finding consistent irrespective of baseline PsO severity (Table). 72% of ADA-treated pts with baseline PsO did not exhibit any structural progression by mTSS <0.5 through 24 wks as compared to 55% receiving PBO. Logistic regression analyses confirmed limited role that PsO severity related to BMI progression. It is important to evaluate BMD in these patients exposed to tobacco. Observed greater progression of BMD in patients with PsA and to investigate the factors related to these changes.

Methods: We included PsA patients with peripheral joint involvement. They were treated at the Doctor Peset University Hospital and a 3-year follow-up was carried out.

All patients underwent a bone densitometry and history of fracture was collected by conventional radiology at baseline and at 3 years. Phosphocalcic metabolism was also analyzed. We collected demographic and clinical variables [disease duration, DAS28, body mass index (BMI), smoking]. We considered 25OHD as deficient if <20 ng/ml, low bone mass as T ≤-1 SD and osteoporosis as scale T ≤-2.5 DE or Z ≤-2 SD. Statistical analysis were performed with SPSS 22.0 program.

Results: We included 91 patients (67.2% women), with a mean age of 56.4 (SD 1.2) years. The mean of disease duration was 104.9 (SD 11.8) months. The BMI mean was 27.6 (SD 0.5) and 36.3% of patients were smokers. 25OHD mean was 27.7 (SD 1.3) ng/ml and 33.3% of patients had a deficient 25OHD. More than half of the patients (53.8%) had low bone mass and 14.3% had osteoporosis, measured by densitometry. Twelve patients had history of fractures [6 distal forearm, 5 vertebral fractures (VF) and 1 of femur]. Thirteen patients received antiresorptive treatment [9 bisphosphonates, 3 denosumab and 1 selective estrogen receptor modulator (SERM)], and 39.6% of patients received 25OHD supplements. We did not observe any significant correlation between the 25OHD values and the activity parameters of the PsA. However, we observed lower levels of 25OHD in obese patients. At 3 years, the BMD remained stable in the majority of patients (68.1%) receiving antiresorptive treatment, and it worsened in 2 patients despite treatment with bisphosphonates. We registered new fractures in 7 patients (4 VF, 1 lumbar fracture and two patients had more than one fracture). New fractures were not correlated with age, 25OHD deficiency or previous osteoporotic treatment. We observed greater progression of BMD in patients exposed to tobacco.

Conclusion: More than half of patients with PsA have a low BMD (53.8%) and 33.3% deficient levels of 25OHD. Our study confirms results found in general population: classic risk factors, such as smoking, are related to BMI progression. It is important to evaluate BMD in these patients and intervene early. Disclosure of Interests: None declared DOI: 10.1136/annrheumdis-2019-eular.5861

## Table 1

<table>
<thead>
<tr>
<th>Outcome</th>
<th>ADA (n=32)</th>
<th>PBO (n=24)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serous Fractures</td>
<td>2 (6.3%)</td>
<td>3 (12.5%)</td>
<td>0.60</td>
</tr>
<tr>
<td>Femur Fractures</td>
<td>1 (3.1%)</td>
<td>2 (8.3%)</td>
<td>0.37</td>
</tr>
<tr>
<td>Vertebral Fractures</td>
<td>1 (3.1%)</td>
<td>2 (8.3%)</td>
<td>0.37</td>
</tr>
</tbody>
</table>

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Disclosure of Interests: Josep F. Merola Consultant for: Biogen IDEC, Abbvie, Amgen, Eli Lilly and Company, Novartis, Pfizer, Janssen, UCB, Samumed, Celgene, Sanofi Regeneron, Merck, and GSK, Laura C Coates Grant/research support from: Abbvie, Celgene, Lilly, Novartis and Pfizer, Consultant for: Abbvie, Amgen, BMS, Celgene, Gallapagos, Gilead Sciences Inc, Janssen, Lilly, Novartis, Pfizer, Prothera Corp and UCB, Elizabeth Lesser Shareholder of: Abbvie Inc, Employee of: Abbvie Inc, Wendell Valdecantos Shareholder of: Abbvie Inc, Employee of: Abbvie Inc, Yanna Song Shareholder of: Abbvie, Employee of: Abbvie Inc, Philip J Mease Grant/research support from: Abbvie, Amgen, BMS, Celgene, Janssen, Lilly, Novartis, Pfizer, SUN and UCB, Consultant for: Abbvie, Amgen, BMS, Galilead Sciences, Inc, Janssien, Lilly, Novartis, Pfizer, SUN and UCB, Speakers bureau: AbbVie, Amgen, BMS, Celgene, Genentech, Janssen, Lilly, Novartis, Pfizer and UCB.


## EVOlution OF Bone MINERAL DensiTy IN Patients WITH PsoriACtic ArThropATHy

### Background

Some studies indicate that psoriatic arthritis (PsA) patients have increased bone loss due to chronic inflammation, but data in the Spanish population is scarce.

### Objectives

To study the evolution over time of bone mineral density (BMD) in patients with PsA and to investigate the factors related to these changes.

### Methods

We included PsA patients with peripheral joint involvement. They were treated at the Doctor Peset University Hospital and a 3-year follow-up was carried out.

All patients underwent a bone densitometry and history of fracture was collected by conventional radiology at baseline and at 3 years. Phosphocalcic metabolism was also analyzed. We collected demographic and clinical variables [disease duration, DAS28, body mass index (BMI), smoking]. We considered 25OHD as deficient if <20 ng/ml, low bone mass as T ≤-1 SD and osteoporosis as scale T ≤-2.5 DE or Z ≤-2 SD. Statistical analysis were performed with SPSS 22.0 program.

### Results

We included 91 patients (67.2% women), with a mean age of 56.4 (SD 1.2) years. The mean of disease duration was 104.9 (SD 11.8) months. The BMI mean was 27.6 (SD 0.5) and 36.3% of patients were smokers. 25OHD mean was 27.7 (SD 1.3) ng/ml and 33.3% of patients had a deficient 25OHD. More than half of the patients (53.8%) had low bone mass and 14.3% had osteoporosis, measured by densitometry. Twelve patients had history of fractures [6 distal forearm, 5 vertebral fractures (VF) and 1 of femur]. Thirteen patients received antiresorptive treatment [9 bisphosphonates, 3 denosumab and 1 selective estrogen receptor modulator (SERMI)], and 39.6% of patients received 25OHD supplements. We did not observe any significant correlation between the 25OHD values and the activity parameters of the PsA. However, we observed lower levels of 25OHD in obese patients. At 3 years, the BMD remained stable in the majority of patients (68.1%) receiving antiresorptive treatment, and it worsened in 2 patients despite treatment with bisphosphonates. We registered new fractures in 7 patients (4 VF, 1 lumbar fracture and two patients had more than one fracture). New fractures were not correlated with age, 25OHD deficiency or previous osteoporotic treatment. We observed greater progression of BMD in patients exposed to tobacco.

### Conclusion

More than half of patients with PsA have a low BMD (53.8%) and 33.3% deficient levels of 25OHD. Our study confirms results found in general population: classic risk factors, such as smoking, are related to BMI progression. It is important to evaluate BMD in these patients and intervene early.