Table 1. Initial demographic and clinical datas of patients with PsA

	PsA Patients (n=355)
Females, n (%)	227 (63,9)
Age of diagnosis, years*	34,6 (27-42)
CRP baseline*	6 mg/ L (3-15)
ESR baseline*	24 mm/h (10-38)
Smoking, n (%)	
Current	99 (28,5)
Never	192 (55,3)
Previous	56 (16,2)
HLA B27 positivity, n (%)	41 (26,4)
First biologic agent, n (%)	
-TNFi	332 (95,4)
Adalimumab	125 (37,6)
Etanercept	80 (24,1)
Golimumab	52 (15,6)
Certolizumab	44 (13,3)
Infliximab	31 (9,4)
- Other biologic agents	16 (4,6)
Secukinumab	13 (81,3)
Ustekinumab	3 (18,7)

* median (min-max)

Conclusion: The results of this study establish that more than half of patients with PsA can remain in their initial biologic treatment over a long term. It has been observed that the choice of biologic treatment did not effect the drug survival in PsA.

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SAT0424

OBESITY LINKS TO PSORIATIC ARTHRITIS (PSA) ACTIVITY, HIGHER PREVALENCE OF CARDIOMETABOLIC DISORDERS AND WORSE PATIENT REPORTED OUTCOMES (PROS): DATA FROM THE RUSSIAN PSA REGISTRY (RU-PSART)

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Background: PsA patients (pts) have an increased risk of cardiovascular (CV) and metabolic (Met) disorders due to the combination of inflammation and increased prevalence of traditional CV risk factors. Only limited data are available on Russian PsA pts. Data was collected from 25 rheumatology clinics of the Russian Federation

Objectives: to study, in clinical practice, the prevalence of obesity and its association with PsA activity, CV/Met comorbidities and PROs

Methods: 614 (M/F=331(54%)/283(46%)) PsA pts fulfilling the CASPAR criteria were included from the RU-PSART cohort. Mean age 45.2±0.52 yrs, PsA duration 5.7±0.27 yrs, PsO duration 15.71±0.56 yrs, DAPSA 28.79±0.75. At baseline (BL) PsA activity was evaluated by Tender Joint Count (TJC68), Swollen Joint Count (SJC66), PGA, physician global assessment by Visual Analog Scale (VAS), DAPSA; PROs according to PtGA VAS, PtPain VAS, HAQ, Work Productivity and Activity Index (WPAI) and Body Mass Index (BMI, kg/m²) were calculated. All pts were split into three groups by BMI (kg/m²): normal<25 (I group), overweight 25-30 (II group), obese>30 (III group). M±m, %, Me [Q25; Q75], χ² or Fischer's exact tests were performed

Results: At BL the BMI was 27.7±0.23 kg/m² with the following BMI categories: normal - 213 pts (34.7%), overweight - 214 pts (34.8%) and obese - 187 (30.5%). Increased BMI was observed in 65.3% of PsA pts. In all groups, comorbidities were found in 297 out of 614 pts (48%): arterial hypertension in 190 (64%), diabetes mellitus in 44 (14.8%), Met syndrome in 36 (12.1%), Coronary Heart Disease in 22 (7.4%). CV and Met disorders were significantly more common in obese pts with BMI>30 compared to overweight pts and to normal BMI pts; female prevailed (Table 1).

Table 1. CV/Met comorbidity in three groups, n (%).

	I group BMI<25 kg/m ²	II group BMI 25-30 kg/m ²	III group BMI>30 kg/m ²	p
Arterial hypertension	28(13.2)	55(25.7)	85(46.5)	p<0.0001*
Diabetes mellitus	0	15(7)	25(14)	p<0.0001**
Metabolic syndrome	0(0)	6(2.8)	23(12.8)	p<0.0001**
Coronary heart disease	2(0.9)	6(2.8)	10(5.6)	p<0.026***

*III vs I and II groups, ** III vs II, *** III vs I group

At BL pts with BMI>30 had higher PsA activity by DAPSA, HAQ, daily activity impairment by WPAI, PtPain compared to I and II groups (table 2).

Table 2. PsA activity and PROs based on BMI categories.

	I group BMI<25 kg/m ²	II group BMI 25-30 kg/m ²	III group BMI>30 kg/m ²	p
DAPSA	22.7 [14.7; 35.8]	24.9 [13.5; 38.5]	27 [17.2; 44.4]	p<0,05*
HAQ	0.9 [0.5; 1.3]	0.9 [0.5; 1.3]	1 [0.8; 1.6]	p<0,05*
Daily activity impairment	0.3 [0; 0.5]	0.3 [0; 0.5]	0.4 [0; 0.6]	p<0,05*
PtPain, mm VAS	50 [30; 66.5]	50 [30; 68]	50 [40; 70]	p<0,05**

*p<0.05 III vs I and II groups, ** p<0.05 III vs II group

Conclusion: In clinical practice, BMI increase was found in the majority of PsA pts. Obesity was associated with higher PsA activity, more prevalence of CV/ Met disorders and worse PROs. Obesity requires a change of the pts' lifestyle, nutrition correction and a right choice of therapy

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SAT0425

NOVEL COMPUTER-ASSISTED METHODOLOGY FOR QUANTITATIVE ASSESSMENT OF MRI TREATMENT RESPONSES TO APREMILAST IN PATIENTS WITH PSORIATIC ARTHRITIS

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Background: Response to treatment in psoriatic arthritis (PsA) can be captured using the OMERACT PsA Magnetic Resonance Imaging Score (PsAMRIS). While reliable and valid, PsAMRIS interpretation requires a trained reader to assess inflammatory lesions such as synovitis and flexor tenosynovitis on a discrete scale ranging from 0 to 3, which might not have sufficient sensitivity to capture early and subtle changes in inflammation in small cohorts.

Objectives: To propose a novel computer-assisted imaging quantitative methodology to assess early response to treatment on a continuous scale and compare its results with those of PsAMRIS.

Methods: Patients with active PsA in the hand and wrist were treated with apremilast 30mg twice daily after a 5-day titration period. A total of 29 patients underwent MRI scans at baseline and months 3, 6, and 12. Images were scored for synovitis using the PsAMRIS interpreted by an experienced reader and were read in blinded sequences. Images for 13 patients with involvement of the wrist and metacarpophalangeal (MCP) joints and MRI available at baseline, 3 months, and 6 months were further processed using a novel computer-assisted imaging quantitative methodology. Images were scored concurrently, with the reader blinded to the order of visits. An experienced reader pre-defined regions of interest (ROIs) around the wrist, MCP joints (MCP-2 to MCP-5), and flexor and extensor tendons of the fingers and wrist (as applicable) with adjacent blood vessels and possible artifacts excluded from ROIs. From these ROIs, the normalized volume of inflammation (NormI) was calculated in each joint and tendon. This was done by automatically counting the pixels that were enhanced above the intensity level of a muscle. Each enhanced pixel was given a weight corresponding to the degree of enhancement, allowing differentiation of areas of residual inflammation and high perfusion. This method has been validated, tested, and implemented in the CE/FDA510-cleared software package Dynamika (IAG, Image Analysis Group). PsAMRIS responses were compared with those of the computer-assisted imaging quantitative methodology at baseline, 3 months, and 6 months. A heat map of normalized intensities was produced, highlighting areas of perfusion higher than that of healthy muscle. Changes from baseline were tested for significance using a *t*-test. Patients with non-missing data were included in the final statistical analysis.

Results: The generated NormI map highlighted a reduction in wrist inflammation activity after 3 and 6 months of treatment with apremilast. In all cases, a downward trend in inflammatory activity in the wrist and MCP joints was observed at 3 months, indicating a reduction following treatment with apremilast (Figures 1 and 2). Similar improvements were observed in tenosynovitis (Figures 1 and 2).

Conclusion: In this pilot assessment, apremilast was associated with improvements in synovitis and tenosynovitis over a period of 6 months using PsAMRIS. Assessment of images using NormI, a methodology allowing quantification of inflammatory activity within a joint or tendon, demonstrated the same trends over 6 months. Further studies are planned to determine the sensitivity of this novel computer-assisted imaging quantitative methodology relative to that of PsAMRIS and whether it could be used to provide early indications of treatment response in small cohorts of patients.

Figure 1. NormI Mean Intensity Values at Baseline, 3 Months, and 6 Months

Figure 2. PsAMRIS Mean Values at Baseline, 3 Months, and 6 Months

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Glenmark, Novartis, Pfizer, UCB (scientific advisor),. Speakers bureau: Eli Lilly, Esate, Novartis, Pfizer, UCB, Mark Hinton: None declared, Eser Sanverdi Employee of: Image Analysis Group – employment, Romies Hagoug Employee of: Image Analysis Group – employment, Christopher Sabin Employee of: Image Analysis Group – employment, Priscila Nakasato Employee of: Amgen Inc. – employment; Celgene Corporation – employment at the time of study conduct, Benoit Guerette Employee of: Amgen Inc. – employment; Celgene Corporation – employment at the time of study conduct, Olga Kubassova Shareholder of: IAG, Image Analysis Group, Consultant of: Novartis, Takeda, Lilly, Employee of: IAG, Image Analysis Group

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SAT0426

CAN BIOLOGICS “PREVENT” THE DEVELOPMENT OF PSORIATIC ARTHRITIS IN PSORIASIS PATIENTS? DATA FROM A LARGE UNIVERSITY HOSPITAL COHORT IN ARGENTINA

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Background: As psoriasis (Pso) commonly precedes psoriatic arthritis (PsA), an important unanswered question is whether treatment of Pso might influence the development of PsA in patients with psoriasis

Objectives: The objective of this study was to analyze the incidence of PsA in a large cohort of patients with PsO according to different treatments, with the hypothesis that treatment with biologics might prevent the development of PsA.

Methods: Patients with PsO without PsA followed at a University Hospital were included in this retrospective cohort study. Data was obtained from the Hospital Electronic Medical Record (EMR). Patients were classified according to their treatment in topics group (topic and phototherapy), conventional DMARDs (cDMARDs) group (Methotrexate (MTX) and cyclosporine (Cyc)), and biologic DMARDs group (bDMARDs) (TNFi, IL17i, and IL12-23i). Patients contributed time since beginning of the corresponding treatment until diagnosis of PsA, lost of follow up, end of treatment or end of study. Incident cases of PsA were attributed to one treatment if developed during the administration of that treatment and up to 6 months after its discontinuation if no other treatment was started. Incident cases that developed more than one year after discontinuation of treatment were disregarded (3 cases). Incidence rate was calculated for the whole population and for each one of the treatment groups and compared with chi2test, and rate ratios were calculated as well. A multivariable logistic model for the development of PsA was analyzed by treatment groups, adjusting by other variables.

	Topics, n= 599	cDMARDs, n=106	bDMARDs, n=92	Total group, n=797
Mean age at PsO diagnosis (SD)	43.6 (20.6)	46.2 (20.7)	35.1 (18.9)	42.9 (20.6)
Female, n (%)	314 (52)	53 (50)	36 (39)	403 (50.5)
Plaque PsO, n/N (%)	514/578 (89)	93/103 (90)	85/90 (94)	692/771 (90)
BMI, n; Mean (SD)	498; 27.6 (5.4)	80; 28.5 (5.6)	91; 30 (7)	659; 28 (5.7)
N Developed PsA (%)	68 (8.5)	3 (0.38)	1 (0.13)	72 (9)
Follow up (patient/years)	9153	316	548	10017
Incidence rate/1000 Patient/years (95% CI)	7.4 (5.8-9.4)	9.5 (1.9-27.5)	1.81 (0.04-10)	7.2 (5.6-9)
Median years between PsO and PsA (IQR)	10 (2-21)	Patient 1: 23 Patient 2: 11.7 Patient 3: 24.7	Patient (1): 9.6	10.5 (2-20.8)

Results: 797 patients, contributed a total of 10017 patient/years. Patient's characteristics are shown in table 1. 599 (75%) patients were treated only with topics or phototherapy, 106 (13%) with cDMARDs (81% MTX and 19% Cyc) and 92 (11.5%) with biologics (TNFi: 64: etanercept: 44, adalimumab:23, infliximab:6; IL17i: 43: 14 Ixekizumab, 29 Secukinumab; IL12-23i: (Ustekinumab) 16; some patients received more than one biologic). During follow-up 72 patients developed PsA (68 under topics; 3 under cDMARDs (2 MTX and 1 Cyc) and 1 under biologics (1 Secukinumab): Global incidence rate: 7.2 per 1000 patient/years (table 1). Although numerically the incidence of PsA in PsO patients treated with biologics was lower, the difference was not statistically significant. In Cox regression analysis, after adjusting by sex, age, and BMI, treatment with biologics was significantly associated with a reduced risk of developing PsA: Hazard ratio (95% CI): 0.1 (0.013 – 0.7); p= 0.021.

Conclusion: Treatment with biologics in patients with PsO seemed to reduce the risk of PsA and preventing its development in this retrospective single center cohort.

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