Only 12% had moderate and 6% had severe arthritis. Similarly, only 10% had moderate and 7% had severe psoriasis. During and following pregnancy, about half of all patients reported no change in either joint or skin activity during and follow pregnancy, with fairly equal numbers reporting improved and worsened arthritis. On the other hand, 42% had improved psoriasis during pregnancy compared to 6% with worsened. There did not appear to be a significant postpartum flare.

Conclusions: Our analysis found that among women with PsA who have tried to become pregnant, 36% experienced infertility, primarily due to PCOS. Compared to pregnancies occurring before the diagnosis of PsA, pregnancies after PsA diagnosis had a lower frequency of live births and a higher frequency of preterm births. Overall, in this cohort of women with mostly mild disease during pregnancy, arthritis pain and psoriasis activity did not appear to substantially worsen during pregnancy.

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SAT0467 ALL 10-YEAR CARDIOVASCULAR RISK SCORES ARE SIMILAR IN PATIENTS WITH PSORIATIC ARTHRITIS AND PSORIASIS

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Background: Psoriatic arthritis (PsA) and Psoriasis (PsO) are associated with higher cardiovascular (CV) risk with higher risk in patients with a severe disease phenotype^{1,2}. Long-term CV risk is evaluated using several methods for the general population.

Objectives: The aim of the study was to compare CV risk factors and 10-year CV risk scores between patients with PsA and those with PsO only.

Methods: PsA patients fulfilling the CASPAR criteria and PsO with disease duration <10 years were enrolled consecutively from Rheumatology and Dermatology clinics. Fasting bloods were obtained for glucose, insulin and lipids. Patients underwent thorough physical examination, joint and skin assessments and completed questionnaires on health and quality of life. Four different CV risk scores were calculated: (1) Framingham Coronary risk score (FCS); (2) American College of Cardiology and American Heart Association (ACC/AHA) 10-year atherosclerotic cardiovascular disease (ASCVD) algorithm; (3) Systematic Coronary Risk Evaluation (SCORE) algorithm; and (4) QRISK2 (2016).

Results: 232 patients (100 PsA and 132 PsO) were recruited with mean age 52.4 (±10.5) for PsA and 39.7 (±14.4) for PsO. Mean disease duration for PsA was 17.9 (±10) years. There were significantly more patients with hypertension (HTN) and metabolic syndrome in PsA group, and the patients taking DMARDs and/or biologics treatment were also higher compared to PsO. Fasting glucose, insulin, lipids, BMI and waist/hip ratio did not show significant differences. The mean FCS, ASCVD, SCORE and QRISK2 were significantly higher in PsA as compared to PsO (7.6±7.0% vs. 4.7±6.1%, P=0.0002; 8.4±8.2% vs. 4.5±5.8%, P<0.0001; 2.1±2.6% vs. 1.1±1.8%, P=0.0002; 11.2±9.9% vs. 5.2±6.4%, P<0.0001, respectively). However, after adjusting for age and sex, all CV risk scores were similar (P=0.21). Multiple regression analysis revealed that waist/hip ratio significantly correlated with all CV risk scores in both PsA and PsO.

Conclusions: We found similar 10-year risk of CVD in PsA and PsO patients using FCS, ASCVD, SCORE and QRISK2. Waist/hip ratio had significant effect on all CV risk scores suggesting that central obesity should be managed appropriately to reduce long-term CVD in psoriatic disease.

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SAT0468 PRESENCE OF POOR PROGNOSTIC FACTORS MAY PREDICT RESPONSE TO ABATACEPT IN PATIENTS WITH ACTIVE PSORIATIC ARTHRITIS: RESULTS FROM A POST HOC ANALYSIS FROM A PHASE III STUDY

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Background: Abatacept, a selective T-cell co-stimulation modulator, significantly increased ACR20 response and had an overall beneficial effect on musculoskeletal symptoms in patients (pts) with active psoriatic arthritis (PsA) in the Phase III Active pSoriaTic aRthritis RAndomizEd triAl (ASTRAEA, NCT01860976).¹ Factors that may predict responses to abatacept were explored in this *post hoc* analysis.

Objectives: To evaluate the relationship between baseline characteristics and abatacept response in a *post hoc* analysis of ASTRAEA.

Methods: Pts were randomized (1:1) to SC abatacept 125 mg weekly or placebo for 24 weeks in this trial. Pts without >20% improvement in joint counts at Week 16 were switched to open-label abatacept (early escape). ACR20 response rate in pts stratified by baseline variables was investigated in a multivariate analysis and odds ratios (ORs) generated to identify differences in response. Using a cut-off of OR 1.2, indicating pt subgroups in whom abatacept appeared to have a meaningful treatment benefit, baseline variables were further investigated in a univariate analysis and estimated differences calculated.

Results: Of 424 pts enrolled, 213 received abatacept and 211 placebo. In abatacept-treated pts, the multivariate model showed a difference in ACR20 response (OR >1.2) for baseline CRP (>upper limit of normal [ULN] vs ≤ULN; OR 1.346 [95% CI 0.668, 2.712]), DAS28 (CRP) (>5.1 vs ≤5.1; 1.489 [0.782, 2.836]), dactylitis (>0 vs 0; 1.372 [0.708, 2.659]), and median baseline erosions (≥3 vs <3; 1.924 [1.032, 3.587]). In placebo-treated pts, the OR was >1.2 for dactylitis only (1.406 [0.619, 3.193]). These factors, which have been identified previously as indicating poor prognosis in PsA, were balanced between treatment arms at baseline. In the univariate model by poor prognostic factors, the differences in ACR20 response rates with abatacept treatment vs placebo in distinct subgroups were numerically greater in pts who were positive for these prognostic factors at baseline than in those who were not (Figure).

Figure. Univariate Analysis of ACR20 Response Rate by Poor Prognostic Factors at Week 24

ULN=upper limit of normal

Conclusions: These findings identified subgroups of pts with PsA with certain baseline characteristics in whom abatacept is most likely to be effective. The predictive factors identified are aligned with poor prognostic factors in the EULAR and Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) guidelines,^{2,3} and may indicate pts with the highest unmet medical need.

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SAT0469 INTEGRATED EFFICACY ANALYSIS OF TOFACITINIB, AN ORAL JANUS KINASE INHIBITOR, IN PATIENTS WITH ACTIVE PSORIATIC ARTHRITIS

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Background: Tofacitinib is an oral Janus kinase inhibitor under investigation for treatment of psoriatic arthritis (PsA).

Objectives: To determine efficacy based on pooled data from 2 pivotal Phase 3 studies of tofacitinib in patients (pts) with active PsA.

Methods: Data were pooled from 2 placebo (PBO)-controlled, double-blind, multicentre, global Phase 3 studies (OPAL Broaden [N=422; 12 months; NCT01877668]; OPAL Beyond [N=394; 6 months; NCT01882439]). Pts had active PsA and either inadequate response (IR) to ≥ 1 conventional synthetic disease-modifying antirheumatic drug (csDMARD) and were tumour necrosis factor inhibitor (TNFi)-naïve (OPAL Broaden), or had IR to ≥ 1 TNFi (OPAL Beyond). Pts were randomised to tofacitinib 5 mg twice daily (BID), 10 mg BID, adalimumab 40 mg subcutaneous injection once every 2 weeks (OPAL Broaden only) or PBO, in addition to a single, stable background csDMARD. PBO pts advanced to tofacitinib 5 mg or 10 mg BID at Month (M)3. Endpoints included ACR20/50/70 response rates ($\geq 20\%$, $\geq 50\%$ and $\geq 70\%$ improvement), change from baseline (Δ) in HAQ-DI (Health Assessment Questionnaire-Disability Index), HAQ-DI response (decrease from baseline [BL] of ≥ 0.35), PASI75 ($\geq 75\%$ improvement from BL in Psoriasis Area and Severity Index), Δ LEI (Leeds Enthesitis Index) and enthesitis absence, Δ DSS (Dactylitis Severity Score) and dactylitis absence and Δ DLQI (Dermatology Life Quality Index). Pooled data only included tofacitinib 5 mg and 10 mg BID (to M6) and PBO (to M3). Significance was declared for $p \leq 0.05$ without correction for multiplicity.

Results: For pooled data, pts were predominantly white (94.2%) and female (55.4%) with ≥ 5 peripheral swollen or tender joints (98.0%), enthesitis (LEI) > 0 ; 67.5%), dactylitis (DSS) > 0 ; 52.5%), psoriatic body surface area $\geq 3\%$ (67.7%) and CRP levels above the upper limit of normal (> 2.87 mg/L; 62.5%) at BL. Mean age was 49.1 years and PsA duration was 8.0 years. Significant improvements vs PBO at M3 were observed for peripheral arthritis and physical function endpoints for tofacitinib 5 mg and 10 mg BID: ACR20, ACR50, ACR70, Δ HAQ-DI (least squares mean [LSM]), and HAQ-DI response (Table 1). Significant improvements in psoriasis, enthesitis and dactylitis endpoints vs PBO were also observed for tofacitinib 5 mg and 10 mg BID at M3: PASI75, Δ LEI, enthesitis absence based on LEI, Δ DSS (LSM), dactylitis absence based on DSS and Δ DLQI (Table 1). Efficacy was maintained or further improved at M6.

Table 1. Efficacy endpoints at Month 3 and Month 6; pooled data from OPAL Broaden and OPAL Beyond (Full Analysis Set*)

	Placebo (N=236)	Month 3		Month 6 ^b	
		Tofacitinib 5 mg BID (N=236)	Tofacitinib 10 mg BID (N=236)	Tofacitinib 5 mg BID (N=238)	Tofacitinib 10 mg BID (N=236)
ACR20, n (%)	66 (28.0)	119 (50.0)***	125 (53.0)***	141 (59.2)	135 (57.2)
ACR50, n (%)	29 (12.3)	69 (29.0)***	79 (33.5)***	91 (38.2)	87 (36.9)
ACR70, n (%)	18 (7.6)	40 (16.8)*	34 (14.4)*	47 (19.8)	52 (22.0)
Δ HAQ-DI, LSM (SE)	-0.16 (0.03)	-0.38 (0.03)***	-0.38 (0.03)***	-0.45 (0.03)	-0.41 (0.04)
[N1/N2]	[219/235]	[227/236]	[223/236]	[222/236]	[212/236]
HAQ-DI response, n/N3 (%)	61/210 (29.1)	109/212***	101/215***	-	-
		(51.4)	(47.0)		
PASI75, n/N3 (%)	24/168 (14.3)	52/162 (32.1)***	66/151 (43.7)***	65/162 (40.1)	79/151 (52.3)
Δ LEI, LSM (SE)	-0.5 (0.2)	-1.2 (0.1)*	-1.4 (0.2)***	-1.5 (0.2)	-1.6 (0.2)
[N1/N2]	[145/156]	[149/156]	[149/160]	[149/156]	[145/160]
Enthesitis absence rate (LEI), n/N3 (%)	34/158 (21.5)	58/158* (36.7)	58/163* (35.6)	75/163 (45.7)	71/163 (43.6)
Δ DSS, LSM (SE)	-2.5 (0.7)	-4.6 (0.6)*	-5.8 (0.6)**	-6.0 (0.7)	-6.4 (0.7)
[N1/N2]	[110/119]	[122/125]	[118/124]	[119/125]	[114/124]
Dactylitis absence rate (DSS), n/N3 (%)	37/121 (30.6)	55/127* (43.3)	69/125*** (55.2)	71/127 (55.9)	76/125 (60.8)
Δ DLQI, LSM (SE)	-2.1 (0.3)	-4.1 (0.3)***	-5.5 (0.3)***	-4.4 (0.3)	-5.7 (0.4)
[N1/N2]	[219/233]	[226/234]	[222/234]	[222/234]	[211/234]

Nominal * $p \leq 0.05$; ** $p \leq 0.001$; *** $p \leq 0.0001$ vs placebo at M3

*All randomised patients who received ≥ 1 dose of study medication; ^bp values not calculated at M6 as the placebo-controlled period ended at M3; ^cprimary endpoint in each individual study at M3; ^damong patients with baseline HAQ-DI ≥ 0.35 (analysis at M6 not performed); ^eamong patients with baseline BSA $\geq 3\%$ and baseline PASI > 0 ; ^famong patients with baseline score > 0

Large sample approximation to the difference in binomial proportions was used to analyse ACR20/50/70, HAQ-DI response, enthesitis absence, dactylitis absence and PASI75; missing response considered as non-response. HAQ-DI, Δ LEI, Δ DSS, and Δ DLQI were analysed with a mixed model for repeated measures without imputation for missing values

Δ , change from baseline; ACR20/50/70, American College of Rheumatology $\geq 20/50/70\%$ improvement; BID twice daily; BSA, body surface area; DLQI, Dermatology Life Quality Index; DSS, Dactylitis Severity Score; HAQ-DI, Health Assessment Questionnaire-Disability Index; LEI, Leeds Enthesitis Index; LSM, least squares mean; M, Month; N, number of patients in the Full Analysis Set; n, number of patients with response; N1, number of patients evaluable at a visit; N2, number of patients included in the model; N3, number of patients evaluable at a visit (denominator); PASI, Psoriasis Area and Severity Index; PASI75, $\geq 75\%$ improvement from baseline PASI; SE, standard error

Conclusions: In a pooled analysis of csDMARD-IR/TNFi-naïve and TNFi-IR pts,

Abstract SAT0470 – Table 1. Summary of efficacy results at Week 156

Variables	Secukinumab IV $\rightarrow$ 150 mg (n=161)	Secukinumab IV $\rightarrow$ 75 mg (n=147)
ACR 20/50/70 ^a , % responders	77/55/33	65/39/26
PASI 75 ^{a,b} , % responders	76	59
DAS28-CRP ^c , mean change (SD)	-1.9 (1.3)	-1.9 (1.5)
HAQ-DI ^c , mean change (SD)	-0.43 (0.6)	-0.42 (0.6)
Resolution of dactylitis ^{a,d} (%)	88.1	86.8
Resolution of enthesitis ^{a,d} (%)	76.7	74.8

Analysis by anti-TNFi status^c

	Anti-TNFi-naïve		Anti-TNFi-IR	
	Secukinumab IV $\rightarrow$ 150 mg (n=120)	Secukinumab IV $\rightarrow$ 75 mg (n=110)	Secukinumab IV $\rightarrow$ 150 mg (n=41)	Secukinumab IV $\rightarrow$ 75 mg (n=37)
ACR20/50/70	81/63/39	67/43/28	62/36/18	56/28/19
PASI 75	76	60	75	56

^aMultiple imputation (missing binary variables). ^bAnalysis performed in pts with psoriasis $\geq 3\%$ body surface area at time of randomisation (n=89 and n=82 in secukinumab IV $\rightarrow$ 150 and IV $\rightarrow$ 75 mg, respectively). ^cObserved data. ^dData from the pts with these symptoms at BL (dactylitis, n=83 in secukinumab IV $\rightarrow$ 150 mg and n=77 in secukinumab IV $\rightarrow$ 75 mg and enthesitis, n=99 in secukinumab IV $\rightarrow$ 150 mg and n=91 in secukinumab IV $\rightarrow$ 75 mg). n, number of pts in the extension study.

tofacitinib 5 mg and 10 mg BID were superior to PBO at M3 across four PsA disease domains: peripheral arthritis, psoriasis, enthesitis and dactylitis.

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SAT0470 SECUKINUMAB PROVIDES SUSTAINED IMPROVEMENTS IN THE SIGNS AND SYMPTOMS OF ACTIVE PSORIATIC ARTHRITIS: 3-YEAR EFFICACY AND SAFETY RESULTS FROM PHASE 3 FUTURE 1 TRIAL

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Background: Secukinumab significantly improved the signs and symptoms of psoriatic arthritis (PsA) over 2 years (yrs) in the FUTURE 1 study.¹

Objectives: To report efficacy and safety of secukinumab through 3 yrs in an extension of the FUTURE 1 study (NCT01892436).

Methods: After 2-yr core trial, patients (pts) receiving secukinumab 150/75mg s.c. entered the 3-yr extension phase. Efficacy results at Week (Wk) 156 are presented for those pts originally randomised to secukinumab (n=308) and included ACR20/50/70, PASI 75, DAS28-CRP, SF-36 PCS, HAQ-DI, dactylitis and enthesitis. Multiple imputation was used for analysis of binary variables while continuous variables are reported as observed. Analyses by anti-TNF status (naïve/inadequate response) were prespecified and reported as observed. Safety analysis included all pts (n=587) who received ≥ 1 dose of secukinumab.

Results: Overall, 457 of the original 606 pts entered the extension study of which 435 (95.2%) pts completed 156 wks (151 [93.8%] pts in IV $\rightarrow$ 150 mg group; 142 [96.6%] in IV $\rightarrow$ 75 mg group; 142 [95.3%] in PBO $\rightarrow$ secukinumab groups). Sustained clinical improvements through Wk 156 were observed across all endpoints and were seen regardless of prior anti-TNF use (Table 1). Over