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OP0222 SECUKINUMAB PROVIDES SUSTAINED IMPROVEMENTS IN THE SIGNS AND SYMPTOMS OF ACTIVE PSORIATIC ARTHRITIS: 104 WEEKS RESULTS FROM A PHASE 3 TRIAL, FUTURE 2

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Background: Secukinumab significantly improved the signs and symptoms of psoriatic arthritis (PsA) over 52 weeks (wks) in FUTURE 2 study (NCT01752634).[1,2]
Objectives: To present longer-term (104 wks) efficacy and safety data of secukinumab from FUTURE 2 study.
Methods: Overall, 397 patients (pts) with active PsA were randomised to secukinumab (300, 150, or 75 mg) or placebo at baseline, Wks 1, 2, 3, and 4, and every 4 wks thereafter. Assessments at Wk 104 are from pts originally randomised to secukinumab and included ACR20/50/70, PASI 75/90, DAS28-CRP, SF-36 PCS, HAQ-DI, dactylitis, and enthesitis. Multiple imputation was used for analysis of binary variables and mixed-model repeated measures for continuous variables. Analyses stratified by anti-TNFα status (naïve/inadequate response or intolerance to these agents) were prespecified and are reported as observed. Safety analysis included all pts who received ≥1 dose of secukinumab.
Results: In total, 86/100 (86.0%), 76/100 (76.0%) and 65/99 (65.7%) pts in the secukinumab 300, 150, and 75 mg groups respectively completed 104 wks. Sustained clinical improvements were observed through Wk 104 with secukinumab across all clinically important domains of PsA (Table). Responses were sustained through Wk 104 regardless of anti-TNFα status. Over the entire treatment period (mean [±SD] exposure to secukinumab of 709±210.99 days), the exposure adjusted incidence rates for serious infections/infestations, candida infections, inflammatory bowel disease and malignant/unspecified tumors with secukinumab were 1.6, 2.3, 0.5 and 1.3, respectively.

Table 1. Summary of Efficacy Results at Wk 104

Variable*	Secukinumab		
	300 mg s.c. (N=100)	150 mg s.c. (N=100)	75 mg s.c. (N=99)
ACR20	69.4	64.4	50.3
ACR50	50.6	36.0	28.2
ACR70	33.1	23.1	14.9
[a]PASI 75	79.5	73.3	58.4
[a]PASI 90	69.6	52.5	33.7
SF-36 PCS, LS mean change from BL (SE)	6.8 (0.85)	5.0 (0.87)	4.1 (0.91)
DAS28-CRP, LS mean change from BL (SE)	?1.9 (0.12)	?1.7 (0.12)	?1.5 (0.13)
HAQ-DI, LS mean change from BL (SE)	?0.58 (0.05)	?0.48 (0.06)	?0.27 (0.06)
[b]Resolution of enthesitis	71.5	61.8	68.4
[c]Resolution of dactylitis	79.9	78.0	88.6

*% responders unless otherwise specified. [a]Assessed in pts with psoriasis affecting ≥3% body surface area at BL (300 mg: n=41; 150 mg: n=58; 75 mg: n=50). [b]Assessed in pts (n=56 [300 mg], 64 [150 mg] and 68 [75 mg]) with this symptom at BL. [c]Assessed in pts (n=46 [300 mg], 32 [150 mg] and 33 [75 mg]) with this symptom at BL. BL, baseline; DAS28-CRP, 28-joint disease activity score using C-reactive protein; HAQ-DI, health assessment questionnaire-disability index; LS, least squares; N, number of pts randomised; PASI, psoriasis area and severity index; SE, standard error; SF-36 PCS, short form-36 physical component summary.

Conclusions: Secukinumab 300 and 150 mg provided sustained improvements in signs and symptoms and multiple clinical domains of active PsA through 2 years of therapy. Secukinumab was well tolerated, with a safety profile consistent with that reported previously.
References:
[1] McInnes IB, et al. Lancet 2015;386:1137–46.
[2] McInnes IB, et al. Ann Rheum Dis. 2015;74:352–3.
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OP0223 ABATACEPT IN THE TREATMENT OF ACTIVE PSORIATIC ARTHRITIS: 1-YEAR RESULTS FROM A PHASE III STUDY

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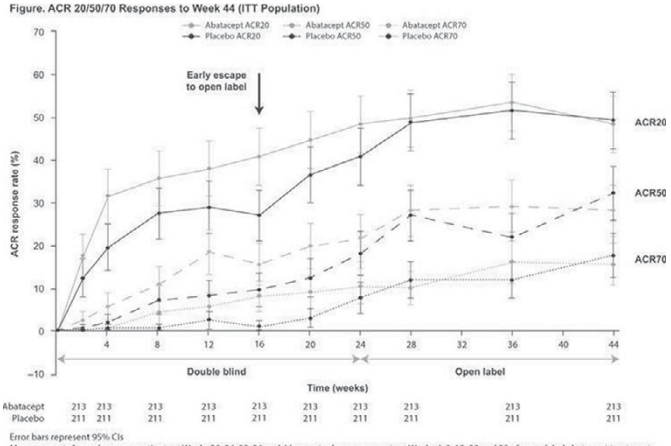
Background: In the Phase III ASTRAEA trial (NCT01860976), abatacept (ABA), a selective T-cell co-stimulation modulator, significantly increased ACR20 response (primary endpoint; PE) and had an overall beneficial effect vs placebo (PBO) on musculoskeletal symptoms in patients (pts) with active psoriatic arthritis (PsA) at 24 weeks (W).[1]
Objectives: To analyse 1-year results from ASTRAEA.
Methods: Pts with active disease (≥3 swollen and ≥3 tender joints), ≥2 cm target lesion of plaque psoriasis and inadequate response/intolerance to ≥1 non-biologic DMARD were randomized (1:1) to SC ABA 125 mg weekly or PBO for 24W, followed by open-label (OL) SC ABA up to 52W. Randomization was stratified by MTX use, prior TNF inhibitor (TNFi) use and skin involvement ≥3% of body surface area. Pts without ≥20% improvement in joint counts at W16 were switched to OL ABA (early escape; EE) for 28W (total study time: 44W). Pre-specified exploratory endpoints included: ACR20/50/70 responses at W44; adjusted mean changes from baseline (BL) in DAS28 (CRP; *post hoc* analysis) and HAQ-DI at W44 and PsA-modified total Sharp/van der Heijde score (SHS) at W44 (EE pts)/W52 (non-EE pts); complete resolution of BL enthesitis and dactylitis at W44 (EE pts)/W52 (non-EE pts); and Psoriasis Area and Severity Index (PASI) 50/75 responses at W44. Analyses used the ITT population with non-responder imputation for missing values and actual data at each time point for all pts (denominator at each time point equal to number of pts in ITT population). All missing responses were imputed as non-responders, except if the missing value was between 2 visits for which the pt was a responder. In that case the missing value was imputed as a responder.
Results: Of 424 pts enrolled, 213 received ABA and 211 PBO. Most (>60%) pts had received prior TNFis. Of pts in the ABA and PBO groups, 76 (36%) and 89 (42%) were EE, 12 (6%) and 24 (11%) discontinued by PE of W24;

Figure. ACR 20/50/70 Responses to Week 44 (ITT Population)



Error bars represent 95% CIs
Measurements for early escape patients at Weeks 20, 24, 28, 36 and 44 are actual measurements at Weeks 4, 8, 12, 20 and 28 of open-label abatacept treatment
At all time points, if there were missing data, patients were imputed as non-responders, unless data were missing between two time points at which the patient had a response, in which case response was imputed