clinical research collaborations and 69% basic research collaborations. The majority of young specialists would be interested in online (84%) and/or 1–2 days (86%) common courses including cases discussion (80%) and training workshops (84%), as well as webinars recorded with several specialists on a specific disease (96%). Respondents were a bit less enthusiastic with developing collaboration through social media (facebook 61%, twitter 58%) but interested in common apps (71%).

Conclusions: This collaborative initiative highlighted wishes from young specialists for developing 1) regular local multidisciplinary meetings to discuss complex patients 2) clinical research collaboration with combined grants and 3) multidisciplinary online projects such as common courses, webinars and apps.

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The changing therapeutic landscape of PsA

OP0305

DISEASE INTERCEPTION IN PSORIASIS PATIENTS WITH SUBCLINICAL JOINT INFLAMMATION BY INTERLEUKIN 17 INHIBITION WITH SECUKINUMAB – DATA FROM A PROSPECTIVE OPEN LABEL STUDY

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Background: Musculoskeletal changes precede the onset of psoriatic arthritis (PsA). A subset of psoriasis patients is characterised by arthralgia as well as inflammatory changes in the joints visible by MRI assessment. These patients have a high risk to progress into PsA.

Objectives: To test the concept of a very early intervention in PsA we exposed psoriasis patients with subclinical joint inflammation to the anti-interleukin (IL)–17A antibody secukinumab. We hypothesised that IL-17A inhibition disrupted the early link between skin and joint disease in psoriasis.

Methods: Psoriasis (but not PsA) patients were included in the open prospective 24 weeks 'Interception in Very Early PsA' (IVEPSA) study. To fulfil the inclusion criteria patients had to have a PASI score greater than 6 or nail or scalp involvement as well as inflammatory or erosive changes in MRI or high-resolution peripheral quantitative computed tomography (HRpQCT) at baseline. Patients received treatment with secukinumab 300 mg sc. for 24 weeks. MRI scans and HRpQCT of the dominant hand were performed at baseline and at 24 weeks. MRI was scored according to PsAMRIS. HRpQCT evaluated for erosions and enthesiophytes.

Results: 20 patients (median age 49.5 years (IQR 42.8, 59), 70% males) with a median disease duration of 14 years (IQR 5, 20), were included into the study. At baseline, 85% reported arthralgia assessed by a Visual Analogue Scale (VAS) and 40% had tender joints on examination (TJC78). 83.3% had at least one inflammatory lesion in the MRI, 66.7% synovitis, 55.6% tendinitis/enthesitis, 27.8% osteitis and 16.7% periarticular inflammation. Erosions were present in 72.2% and 58.8% in the MRI and HRpQCT, respectively, while enthesiophytes were found in 33.3% and 41.2%. One patient was discontinued early due to lack of improvement (wk12) and one patient was unable to perform the follow-up MRI. Psoriatic skin disease (total PASI and BSA) significantly improved (both $p < 0.05$) and also arthralgia (VAS pain, tender joint count) significantly declined after secukinumab treatment (both $p < 0.05$). Total PsAMRIS score and synovitis subscore significantly improved at week 24 ($p = 0.005$ and $p = 0.008$, respectively). Importantly, improvement in total PsAMRIS score significantly correlated with the improvement in arthralgia ($p < 0.05$). Finally, neither erosions nor enthesiophytes in MRI and HRpQCT progressed during the 24 weeks of treatment. There was no new safety signal in the study.

Conclusions: IL-17 inhibition by secukinumab over 24 weeks led to resolution of inflammation and no progression of bone changes in the joints in psoriasis patients with subclinical peripheral joint involvement. These data suggest that very early disease interception in PsA is a feasible approach. IVEPSA also provides the guide for further very early interventions in PsA providing concepts for imaging-based identification and the sensitivity to change subclinical inflammation through biological disease modifying anti-rheumatic drug therapy.

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Disclosure of Interest: None declared

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OP0306

SUBCUTANEOUS SECUKINUMAB INHIBITS RADIOGRAPHIC PROGRESSION IN PSORIATIC ARTHRITIS: ANALYSIS BY PRIOR ANTI-TNF THERAPY AND CONCOMITANT METHOTREXATE USE

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Background: Psoriatic arthritis (PsA) is associated with joint inflammation, characterised by synovitis, presence of erosions, joint space narrowing (JSN) and new bone formation leading to structural damage, increased disability and reduced quality of life. Secukinumab (SEC) provided significant and rapid clinical efficacy, and inhibition of radiographic progression in PsA patients (pts) in the FUTURE 5 study.¹

Objectives: To assess the effect of subcutaneous (sc) SEC on radiographic progression by prior anti-TNF therapy or concomitant methotrexate (MTX) use in the FUTURE 5 study.

Methods: Adults (n=996) with active PsA, stratified by prior anti-TNF therapy (naïve and inadequate response/intolerance [IR]) were randomised 2:2:2:3 to sc SEC 300 mg with loading dose (LD), 150 mg LD, 150 mg no LD, or placebo (PBO) at baseline (BL), Wks 1, 2, 3, 4, and every 4 wks thereafter. At Wk 16, PBO non-responders were switched to SEC 300 or 150 mg. Concomitant MTX (≤ 25 mg/week) was allowed. Radiographic progression (mean change in van der Heijde-modified total Sharp score for PsA [vdH-mTSS] and its components: erosion and joint space narrowing [JSN] scores from BL to Wk 24) was based on hand/wrist/foot X-rays obtained at BL, Wks 16 (non-responders) assessed by two blinded readers (plus an adjudicator if required). Average scores were used. Statistical analyses used linear extrapolation at Wk 24 for all PBO non-responders and for all other pts with missing Wk 24 X-rays.

Results: At BL, 30% pts were anti-TNF-IR and 50% were on concomitant MTX. Radiographic progression was significantly inhibited at Wk 24 in the overall population with SEC vs PBO; mean change from BL in vdH-mTSS was 0.08 (300mg; $p < 0.01$), 0.17 (150mg; $p < 0.05$), -0.09 (150mg no LD; $P < 0.01$) vs 0.50 (PBO). Lower radiographic progression (vdH-mTSS, erosion and JSN scores) was observed with SEC vs PBO regardless of prior anti-TNF therapy or concomitant MTX use (table 1).

Abstract OP0306 – Table 1. Radiographic findings (mean change in scores from BL to Wk 24)

	Radiographic findings (mean change in scores from BL to Wk 24)			
	SEC 300mg		SEC 150mg	
	LD	LD	No LD	PBO
Anti-TNF-naïve	n=152	n=149	n=151	n=214
vdH-mTSS	0.01 [§]	0.12 [‡]	-0.25 [‡]	0.48
Erosion	0.03 [§]	0.09 [‡]	0.01 [‡]	0.36
JSN	-0.02	0.03	-0.25 [‡]	0.12
Anti-TNF-IR	n=65	n=64	n=59	n=82
vdH-mTSS	0.23	0.29	0.31	0.54
Erosion	0.10	0.19	0.27	0.30
JSN	0.13	0.10	0.04	0.24
Without concomitant MTX	n=107	n=106	n=95	n=151
vdH-mTSS	0.06 [‡]	0.16	-0.17 [‡]	0.46
Erosion	0.07 [‡]	0.13	-0.09 [§]	0.41
JSN	-0.01	0.03	-0.07	0.05
With concomitant MTX	n=110	n=107	n=115	n=145
vdH-mTSS	0.10 [‡]	0.18 [‡]	-0.03	0.54
Erosion	0.04 [‡]	0.11	0.22	0.27
JSN	0.06	0.07	-0.25 [‡]	0.26

[‡] $P < 0.0001$; [§] $P < 0.001$; [‡] $P < 0.01$; [‡] $P < 0.05$ un-adjusted P-values vs. PBO

Conclusions: Subcutaneous secukinumab 300 mg with loading dose, and 150 mg with and without loading dose, inhibited radiographic progression in patients with active PsA. Low rates of radiographic progression were observed regardless of previous anti-TNF therapy or concomitant MTX use.

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[1]] Mease PJ, et al. *Arthritis Rheumatol* 2017;69(suppl 10).

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OP0307 EFFICACY AND SAFETY OF RISANKIZUMAB, A SELECTIVE IL-23P19 INHIBITOR, IN PATIENTS WITH ACTIVE PSORIATIC ARTHRITIS OVER 24 WEEKS: RESULTS FROM A PHASE 2 TRIAL

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Background: Interleukin-23 (IL-23), a key regulator of multiple effector cytokines, has been implicated in the pathogenesis of psoriatic lesions, synovitis, enthesitis, and bone erosion. Risankizumab (RZB) is a humanised IgG1 monoclonal antibody that binds to p19 subunit of IL-23, selectively inhibiting this critical cytokine.

Objectives: To report the efficacy and safety of different doses of RZB in patients (pts) with active psoriatic arthritis (PsA) over 24 weeks.

Methods: In this double-blind, parallel-design, dose-ranging Phase 2 study, pts with active PsA (stratified by prior TNFi use and concurrent MTX use) were randomised in a 2:2:2:1:2 ratio to receive RZB (150 mg at weeks [Wks] 0, 4, 8, 12, and 16 [Arm 1], 150 mg at Wks 0, 4, and 16 [Arm 2], 150 mg at Wks 0 and 12 [Arm 3], 75 mg single dose at Wk 0 [Arm 4]) or matching placebo (PBO, Arm 5). Pts completing Wk 24 visit had an option to enter a separate open-label extension (OLE) study; pts not entering the OLE were followed until Wk 32. Efficacy assessments included ACR20/50/70, PASI, minimal disease activity (MDA), DAS28(CRP), dactylitis count, SPARCC enthesitis index, pain-VAS, HAQ-DI, and mTSS scores.

Results: Of the 185 pts who received the study drug, 173 (93.5%) completed 16 Wks of treatment and 145 (78.4%) entered OLE at Wk 24. The primary endpoint of ACR20 response at Wk 16 was achieved by pts in each of the RZB arms.¹ At Wk 24, ACR20/50/70 responses were significantly higher in pts receiving RZB (pooled across all RZB arms) compared with PBO (table 1). PASI75/90/100 responses at Wk 24 were significantly higher in RZB-treated pts compared with PBO. At Wk 24, RZB-treated pts achieved significantly higher MDA responses as well as greater improvements in DAS28(CRP) and Pain-VAS. Improvements in HAQ-DI and enthesitis from BL were numerically greater in RZB arms. At Wk 24, RZB-treated pts (pooled across all RZB arms) showed significant improvement from BL in mTSS compared with PBO. Treatment-emergent adverse events (TEAEs), collected up to Wk 32, were comparable across treatment arms (table 2); the most common TEAE was infection. There were no deaths or cases of tuberculosis in RZB-treated pts; 2 adjudicated major adverse cardiovascular events were reported in RZB arms.

Abstract OP0307 – Table 1. Summary of efficacy results at week 24*

Endpoints	Risankizumab (RZB)					Placebo
	Arm 1 N=42	Arm 2 N=42	Arm 3 N=39	Arm 4 N=20	Arms 1-4 N=143	Arm 5 N=42
ACR20 (%)	42.9	47.6	59.0**	40.0	48.3*	31.0
ACR50 (%)	19.0	16.7	33.3**	20.0	22.4**	7.1
ACR70 (%)	11.9	11.9	15.4*	15.0	13.3**	2.4
PASI 75 (%) ^b	68.8***	70.0***	69.6***	55.6*	67.6***	14.3
PASI 90 (%) ^b	60.0***	52.9**	47.6**	55.6**	53.2***	10.0
PASI 100 (%) ^b	46.7**	35.3**	28.6*	44.4*	37.1***	5.0
MDA (%)	19.0	28.6**	25.6*	30.0*	25.2***	7.1
ΔDAS28(CRP) ^c	-1.1	-1.4**	-1.6***	-1.7***	-1.4***	-0.6
ΔDactylitis Count ^d	-1.3	-2.3	-3.6	-3.5	-2.5	-2.6
ΔSPARCC Enthesitis Index ^{e,d}	-1.3	-2.7*	-1.6	-3.4*	-2.0	-1.1
ΔPain-VAS ^f	-6.9	-11.2	-19.1**	-6.4	-11.5*	-3.2
ΔHAQ-DI ^g	-0.19	-0.11	-0.21	-0.13	-0.16	-0.09
ΔmTSS ^h	-0.3	0.2	-0.5*	-0.2	-0.2*	0.6

Arm 1, 150 mg RZB at weeks 0, 4, 8, 12, and 16; Arm 2, 150 RZB at weeks 0, 4, and 16; PBO at weeks 8 and 12; Arm 3, 150 mg RZB at weeks 0 and 12; PBO at weeks 4, 8, and 12; Arm 4, 75 mg RZB at week 0; PBO at weeks 4, 8, 12, and 16; Arm 5, PBO at weeks 0, 4, 8, 12, and 16.

P-values for comparison versus placebo: ***, P < 0.001; **, P < 0.01; *, P < 0.05.

*Categorical endpoints are based on NRI analyses using Cochran-Mantel-Haenszel test; continuous variables are based on MMRM analyses and mTSS is based on ANCOVA. *PASI responses calculated only in patients with psoriasis affecting ≥3% BSA at baseline; Arm 1, N=16; Arm 2, N=20; Arm 3, N=23; Arm 4, N=9; Arm 5, N=21. *LS Mean for change from baseline are presented. ^bDactylitis count and SPARCC enthesitis index assessed only in patients with active dactylitis (Arm 1, N=9; Arm 2, N=11; Arm 3, N=11; Arm 4, N=6; Arm 5, N=8) and enthesitis (Arm 1, N=26; Arm 2, N=24; Arm 3, N=26; Arm 4, N=11; Arm 5, N=26) at baseline, respectively.

ACR20/50/70 = 20/50/70% improvement in American college of rheumatology score; ANCOVA = analysis of co-variance; BSA = body surface area; DAS28(CRP) = 28-joint disease activity score based on C-reactive protein; Δ = change from baseline to week 24; HAQ-DI = health assessment questionnaire-disability index; MDA = minimal disease activity; MMRM = mixed effect model; repeat measurement; mTSS = modified total Sharp score; NRI = non-responder imputation; PASI75/90/100 = 75/90/100% improvement in psoriasis area and severity index; PBO = placebo; RZB = risankizumab; SPARCC = Spondyloarthritis Research Consortium of Canada; VAS=visual analog scale.

Abstract OP0307 – Table 2. Overview of treatment-emergent adverse events (TEAEs) over 32 weeks

TEAEs, n (%)	Risankizumab (RZB)					Placebo
	Arm 1 N=42	Arm 2 N=42	Arm 3 N=39	Arm 4 N=20	Arms 1-4 N=143	Arm 5 N=42
Any AE	27 (64.3)	22 (52.4)	27 (69.2)	13 (65.0)	31 (73.8)	8 (19.0)
Drug related AEs ^a	8 (19.0)	7 (16.7)	8 (20.5)	4 (20.0)	8 (19.0)	0
Serious AE ^b	3 (7.1)	0	2 (5.1)	3 (15.0)	2 (4.8)	0
Drug related serious AE ^b	1 (2.4)	0	2 (5.1)	1 (5.0)	0	0
Severe AE	3 (7.1)	0	2 (5.1)	1 (5.0)	4 (9.5)	0
AE leading to drug discontinuation	3 (7.1)	0	0	1 (5.0)	2 (4.8)	0
Infection ^c	16 (38.1)	13 (31.0)	16 (41.0)	6 (30.0)	12 (28.6)	0
Serious infection	0	0	0	2 (10.0) ^d	0	0
Serious hypersensitivity	1 (2.4) ^e	0	0	0	0	0
Adjudicated MACE	1 (2.4) ^f	0	0	1 (5.0) ^g	0	0
Malignancy	0	0	0	1 (5.0) ^h	0	0
Depression	0	0	2 (5.1)	0	0	0

Arm 1, 150 mg RZB at weeks 0, 4, 8, 12, and 16; Arm 2, 150 RZB at weeks 0, 4, and 16; PBO at weeks 8 and 12; Arm 3, 150 mg RZB at weeks 0 and 12; PBO at weeks 4, 8, and 12; Arm 4, 75 mg RZB at week 0; PBO at weeks 4, 8, 12, and 16; Arm 5, PBO at weeks 0, 4, 8, 12, and 16.

^aInvestigator assessed AE as possibly related to study drug. ^bNo serious adverse event type was reported in more than one patient.

^cThe most frequently reported AE was upper respiratory tract infection. ^d1 patient with urinary tract infection, influenza, and sepsis. ^e1 patient with gastroenteritis and pyelonephritis. ^fAn event of investigator reported anaphylactic reaction occurred after the 1st injection of risankizumab. The sponsor assessed the event details as not consistent with IgE-mediated anaphylaxis. ^g1 patient with ischemic stroke. ^h1 patient with type 1 myocardial infarction. ⁱ1 patient with ovarian cancer.

AE = adverse events; IgE = immunoglobulin E; MACE = major adverse cardiovascular event; PBO = placebo; RZB = risankizumab; TEAE = treatment-emergent adverse event.

Conclusions: Pts with active PsA treated with RZB maintained improvement in joint and skin symptoms through 24 wks. RZB-treated pts (pooled across all RZB arms) showed evidence for inhibition of radiographic progression. RZB was well-tolerated with no new or unexpected safety findings.

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