Characteristics	AS; n=108	PsA; n=239
Age (Mean±SD)	46.9±12.7	53.4±11.9
Male (%)	62.0	42.7
CRP (mg/L) (Mean±SD)	14.8±22.5	11.8±18.0
Previous NSAID exposure (%)	61.1	24.7
Previous cDMARD exposure (%)	39.8	72.8
Previous bDMARD exposure (%)	68.5	72.8
Lack of efficacy of prev. bDMARD (%)	82.4	85.1

other studies in the phase III program of secukinumab[1,2]. Major difference is represented by the high number of biological-experienced patients and comorbidities. Potential differences between these real world results and previously obtained phase III results will have to be discussed to assess their impact on patients.

References:
[1] McInnes IB et al. Lancet. 2015;386(9999):1137–46.
[2] Baeten D et al. Lancet. 2013;382(9906):1705–13.

Disclosure of Interest: U. Kiltz Grant/research support from: AbbVie, Chugai, MSD, Novartis, Pfizer, Roche, UCB, Consultant for: AbbVie, Chugai, MSD, Novartis, Pfizer, Roche, UCB, C. Legeler: None declared, M. Maier-Peuschel Employee of: Novartis, J. Veit Employee of: Novartis, C. Mann Employee of: Novartis, H.-P. Tony Grant/research support from: AbbVie, Astra-Zeneca, BMS, Chugai, Janssen, Lilly, MSD, Novartis, Pfizer, Roche, Sanofi, Consultant for: AbbVie, Astra-Zeneca, BMS, Chugai, Janssen, Lilly, MSD, Novartis, Pfizer, Roche, Sanofi

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THU0396 EFFICACY OF SWITCHING BDMARDS IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS: RESULTS FROM A SYSTEMATIC LITERATURE REVIEW

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Background: bDMARDs (TNF or IL-17 inhibitors) have been shown to be efficacious in patients with axial spondyloarhritis (axSpA). However, approximately 30–50% of patients who receive a first bDMARD do not respond well. Current practice in these patients is switching to another bDMARD but the scientific evidence for this attitude is sparse.

Objectives: To evaluate the efficacy of switching bDMARDs in patients with axSpA.

Methods: A systematic literature review until February 2016 was performed using Medline, EMBASE and Cochrane databases. Furthermore, abstracts from the previous EULAR and ACR meetings were reviewed. The research question was formulated according to the PICOS method: Population (axSpA patients); Intervention (bDMARD); Outcome (clinical response); and Setting (longitudinal studies with follow-up ≥12 weeks of follow up including data from ≥50 patients). Data was extracted using a form developed for this specific purpose. The quality of the studies was assessed based on CEBM Oxford. Clinical response in patients who switched to a second bDMARD was determined and compared with the one achieved after receiving the first bDMARD (a TNFi in all cases). Results are shown as median (range) and relative frequencies (%).

Results: In total, 7 studies out of 1506 retrieved citations were included. All studies included patients with ankylosing spondylitis (AS). The study design was prospective observational (n=3), retrospective observational (n=2), open-label trial (n=1) and post-hoc analysis from two RCTs (n=1). The level of evidence for all the studies was 4. In these studies, a total of in 4678 patients received a first bDMARD and 1198 patients switched to a second bDMARD (a TNFi in all cases except in 51 patients that switched to secukinumab). Baseline characteristics of patients included in the studies were: 41 (38–44) years old, 67% (64–74) males, 78% (74–89) HLA-B27+ and BASDAI before switching 6.2 (5.3–6.5). The most frequent reason to switch bDMARD was inefficacy, followed by intolerance/adverse events. Median (range) time to assess response after switching was 6 (3–12) months. The criteria to define clinical response were heterogeneous. BASDAI50 was employed in four studies and the percentage of patients who achieved this response after the first and the second bDMARD for each study was: (63% vs 41%), (50% vs 28%), (54% vs 37%), (72% vs 56%), respectively. The response for the other three studies was based on different definitions, being as follows: BASDAI <4 (83% vs 78%), ASAS20 response (67% vs 48%) and retention rate after one year (65% vs 60%). The reason to switch bDMARD (intolerance or inefficacy) was not found as a significant predictor of treatment response in most of the studies. In addition, two studies reported data (n=137 and 11 patients) to evaluate the efficacy of switching to a third bDMARD (TNFi in both cases). The percentage of patients who responded (BASDAI50) to the third TNFi was 30% and 52%, respectively.

Conclusions: In patients with AS who do not respond to a first TNFi, switching to another bDMARD (either a TNFi or secukinumab) is efficacious in a considerable number of patients (30–50%). However, the clinical response after receiving a second bDMARD is lower to the one experienced after the first TNFi. Published data for switching to a third bDMARD is very limited.

Disclosure of Interest: None declared

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THU0397 SECUKINUMAB PROVIDES SUSTAINED IMPROVEMENTS IN THE SIGNS AND SYMPTOMS OF ACTIVE ANKYLOSING SPONDYLITIS: 3-YEAR RESULTS FROM A PHASE 3 EXTENSION TRIAL (MEASURE 1)

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Background: Rapid and sustained improvements in the signs and symptoms of ankylosing spondylitis (AS) have been reported with secukinumab, a fully human anti–IL-17A mAb, over the first 2 years (yrs) in the Phase 3 MEASURE 1 trial.[1,2]

Objectives: To report efficacy and safety of secukinumab through 3 yrs in an extension trial (NCT01863732) to the core MEASURE 1 trial.

Methods: After the 2-yr core trial, patients (pts) receiving secukinumab 150 or 75mg s.c. were invited to enter a 3-yr extension trial. Efficacy results at Week (Wk) 156 are reported for pts who were originally randomised to secukinumab. Assessments at Wk 156 included ASAS20/40, BASDAI, BASDAI50, SF-36 PCS, ASAS partial remission (ASAS PR) and ASDAS-CRP. Binary and continuous variables used multiple imputation and MMRM estimates, respectively. Analyses by anti-TNF use (naïve/intolerant to or inadequate response [IR]) was pre-specified and reported as observed. Safety analyses included all pts who received ≥1 dose of secukinumab.

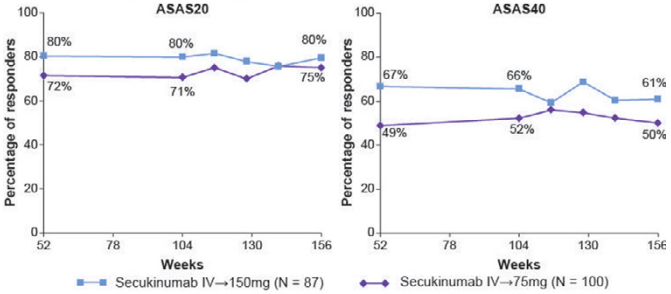
Results: A total of 290/371 pts (78%) completed the 2-yr core trial. Of these, 274 pts entered the extension trial, with 260 completing 156 wks (83/87 pts [95%] in IV→150mg; 95/100 pts [95%] in IV→75mg; 82/87 [94%] pts in placebo→secukinumab). At Wk 156, clinical improvements were sustained across all endpoints (Table, Figure). Similar trends were observed regardless of prior anti-TNF use (Table). Across the treatment period (secukinumab exposure [mean±SD]: 964.3±372.1 days), exposure-adjusted incidence rate with secukinumab for serious infections, Crohn's disease and malignant/unspecified tumours was 1.1, 0.5 and 0.5 per 100 pt-yrs, respectively.

Table 1. Summary of 156-wk efficacy results

	Observed data		Missing data considered[a]	
	Secukinumab IV→150mg (N=87)	Secukinumab IV→75mg (N=100)	Secukinumab IV→150mg (N=87)	Secukinumab IV→75mg (N=100)
ASAS, % response				
ASAS20/40	80/62[b]	76/50[b]	80/61	75/50
ASAS PR	27[b]	14[b]	27	14
BASDAI				
Baseline, mean±SD	6.1±1.5	6.0±1.5	6.1±1.5[#]	6.0±1.5[#]
Mean change from baseline[c]	–3.3±2.4[b]	–3.0±1.7[b]	–3.1±0.2	–2.9±0.2
	Secukinumab IV→150mg		Secukinumab IV→75mg	
Analysis by anti-TNF status[d]				
Anti-TNF-naïve[e]	N=70		N=76	
ASAS20/40, % response	80/61		76/48	
Anti-TNF-IR[e]	N=17		N=24	
ASAS20/40, % response	81/63		74/57	

[a]Missing data of binary variables were imputed and for continuous variables MMRM estimates are shown. [b]Evaluable data available in n=86 and n=98 pts in the secukinumab IV→150mg and IV→75mg groups, respectively. [c]Least squares mean±SE for MMRM estimates and mean±SD for observed data. [d]Observed data. [e]Evaluable data available in n=70 and 75 pts (naïve) and n=16 and 23 pts (IR) in the secukinumab IV→150mg and IV→75mg groups, respectively. [#]Observed data provided for reference. IV, pts received secukinumab 10mg/kg i.v. loading at baseline, Wks 2 and 4; N, number of pts in the extension trial.

Figure. ASAS20 and ASAS40 responses through Week 156



Data presented after multiple imputation through Week 156. ASAS20 and ASAS40 responses through Week 104 have been reported previously.[2] N, number of patients in the extension trial

Conclusions: Secukinumab provided sustained efficacy in signs/symptoms and physical function in pts with active AS over 3 yrs. Secukinumab was well tolerated with a favorable safety profile consistent with that reported previously.[1,2]

References:
[1] Baeten D, et al. N Engl J Med 2015;373:2534–48.
[2] Braun J, et al. Ann Rheum Dis 2016;doi: 10.1136/annrheumdis-2016-209730.

Disclosure of Interest: X. Baraliakos Grant/research support from: AbbVie, BMS, Celgene, Chugai, Merck, Novartis, Pfizer, UCB, Werfen, Consultant for: AbbVie, BMS, Celgene, Chugai, Merck, Novartis, Pfizer, UCB, Werfen, Speakers bureau: AbbVie, BMS, Celgene, Chugai, Merck, Novartis, Pfizer, UCB, Werfen, A. Kivitz Consultant for: AbbVie, Pfizer, Genentech, UCB, Celgene, Speakers bureau: Celgene, Pfizer, Genentech, A. Deodhar Grant/research support from: Eli Lilly, Janssen, Novartis, Pfizer, UCB, Consultant for: Eli Lilly, Janssen, Novartis, Pfizer, UCB, J. Braun Grant/research support from: Abbvie (Abbott), Amgen, BMS, Boehringer, Celgene, Celltrion, Centocor, Chugai, EBEWE Pharma, Medac, MSD (Schering-Plough), Mundipharma, Novartis, Pfizer (Wyeth), Roche, Sanofi-Aventis, UCB, Consultant for: Abbvie (Abbott), Amgen, BMS, Boehringer, Celgene, Celltrion, Centocor, Chugai, EBEWE Pharma, Medac, MSD (Schering-Plough), Mundipharma, Novartis, Pfizer (Wyeth), Roche, Sanofi-Aventis, UCB, Speakers bureau: Abbvie (Abbott), Amgen, BMS, Boehringer, Celgene, Celltrion, Centocor, Chugai, EBEWE Pharma, Medac, MSD (Schering-Plough), Mundipharma, Novartis, Pfizer (Wyeth), Roche, Sanofi-Aventis, UCB, J. Wei Grant/research support from: BMS, Janssen, Pfizer, Sanofi-Aventis, Novartis, Consultant for: Pfizer, Celgene, Chugai, UCB Pharma, TSH Taiwan, Speakers bureau: AbbVie, BMS, Chugai, Janssen, Pfizer, E. Delicha Employee of: Novartis, Z. Talloczy Shareholder of: Novartis, Employee of: Novartis, B. Porter Shareholder of: Novartis, Employee of: Novartis

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THU0398 SECUKINUMAB SUSTAINS INDIVIDUAL CLINICAL RESPONSES OVER TIME IN PATIENTS WITH ACTIVE ANKYLOSING SPONDYLITIS: 2-YEAR RESULTS FROM A PHASE 3 TRIAL, MEASURE 2

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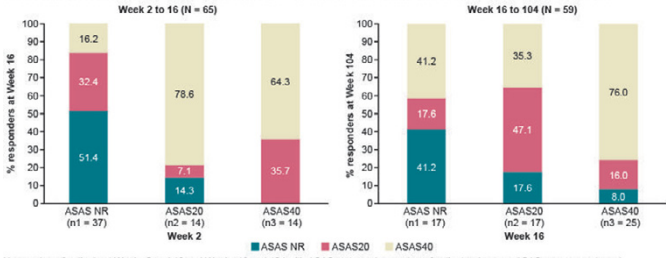
Background: The assessment of achieving, maintaining or improving clinical response to biologics in ankylosing spondylitis (AS) is a part of treat-to-target recommendations aimed at optimising treatment goals.[1]
Objectives: To evaluate patient (pt)-level secukinumab data and assess the likelihood of achieving, maintaining or improving an Assessment of SpondyloArthritis international Society (ASAS) response from Week (Wk) 2 (early response) to Wk 16 (primary endpoint) and from Wk 16 to Wk 52 or 104 (sustained effect) in pts with active AS from the MEASURE 2 trial.[2,3]
Methods: This is a *post-hoc* analysis of AS pts originally randomised to secukinumab 150mg (approved dose) who completed the 16-wk double-blind treatment period, followed by long-term uncontrolled treatment. Shift analyses on ASAS response between Wks 2 and 16 and Wks 16 and 52 or 104 were performed on subgroups of secukinumab 150mg treated pts categorised by their highest ASAS criteria response at the earlier time point (ASAS non-responder [ASAS NR], ASAS20 responder, ASAS40 responder) and evaluating whether this response was improved, sustained, or worsened at the later time point, based on observed analysis.
Results: Overall, 65, 61 and 59 pts treated with secukinumab 150mg had available data to determine ASAS responses for shift analyses from Wk 2 to 16 and Wk 16 to 52 or 104, respectively. At baseline, mean age was 41.9±12.5 years, mean time since diagnosis was 7.0±8.2 years and mean Bath Ankylosing Spondylitis Disease Activity Index score was 6.6±1.5. Approximately half of the ASAS NR pts at Wk 2 or 16 subsequently developed an ASAS 20 or 40 response at the later time point of Wk 16 or 52, respectively. A total of 79% pts improved their response from ASAS20 to ASAS40 at Wk 16 (Wk 2 to 16) and another 44% pts improved their response from ASAS20 to ASAS40 from Wk 16 to 52. A majority (64% and 84%) of ASAS40 responders at Wk 2 or 16 maintained this response at Wk 16 or 52, respectively. Similar trends were observed in responses from Wk 16 to 104 (Figure).
Conclusions: In this *post-hoc* pt-level analysis, the majority of secukinumab 150mg treated pts maintained or improved their ASAS responses over time, consistent with the sustainability of group-level ASAS responses reported previously.[2,3] In particular, the majority of pts who achieved either an ASAS20 or ASAS40 response at Wk 2 or 16 maintained or improved their response at Wks 16, 52 or 104, respectively.
References:
[1] Smolen JS, et al. Ann Rheum Dis 2014;73:6–16.
[2] Baeten D, et al. N Engl J Med 2015;373:2534–48.
[3] Marzo-Ortega H, et al. Ann Rheum Dis 2016;75:812–3.

Figure. Shift analyses on ASAS responses in secukinumab 150mg from Week 2 to 16 and 16 to 104



N = number of patients at Weeks 2 and 16 and Weeks 16 and 104 with ASAS status; n1 = number of patients who were ASAS non-responders at Week 2 or 16; n2 or n3 = number of patients who achieved ASAS20 or ASAS40 at Week 2 or 16; Percentages were calculated by considering n1 to n3 as denominator at Weeks 16 and 104. Patients with multiple responses are counted once at Week 2 and counted once in best response at Week 16 or 104.

Disclosure of Interest: X. Baraliakos Grant/research support from: AbbVie, BMS, Celgene, Chugai, Merck, Novartis, Pfizer, UCB, Werfen, Consultant for: AbbVie, BMS, Celgene, Chugai, Merck, Novartis, Pfizer, UCB, Werfen, Speakers bureau: AbbVie, BMS, Celgene, Chugai, Merck, Novartis, Pfizer, UCB, Werfen, M. Schiff Consultant for: Abbvie, BMS, Lilly, J&J, Speakers bureau: Abbvie, K. Pavelka Speakers bureau: MSD, AbbVie, Roche, UCB, Amgen, Hospira, Egis, Pfizer, Medac, BMS, A. Widmer Shareholder of: Novartis, Employee of: Novartis, B. Porter Shareholder of: Novartis, Employee of: Novartis, C. Gaillez Shareholder of: Novartis, BMS, Employee of: Novartis

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THU0399 TAPERING THERAPY OF TNF-INHIBITOR FOR MRI CHANGES IN SPONDYLOARTHRITIS

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Background: TNF-inhibitors could significantly improve disease activity of SpA patients, however, there is still no answer to the effect of prolonged the interval of TNF-inhibitors on MRI changes.
Objectives: The aim of the study was to investigate whether prolonged the interval of TNF-inhibitor injection could maintain SpA at low disease activity and improve imaging changes of sacroiliac joint.
Methods: A total of 98 SpA patients were included and 67 of them received TNF-i with or without conventional DMARDs. TNF-i included Etanercept, Infliximab and Adalimumab. The full dosage treatment was defined as patients received Etanercept 50 mg per week, Infliximab 4 mg/kg at 0, 2, 6 week and Adalimumab 40 mg every two weeks. The dose of Etanercept was gradually reduced to 50 mg every two weeks, 50 mg every three weeks and then 50 mg per month. The infusion of Infliximab was reduced to every 8 weeks, every 12 weeks and then every 16 weeks. The interval of Adalimumub injection was changed from 3 weeks to 4 weeks and then to two months. After full dose treatment in the first 3 months, patients who administrated TNF-i were evaluated every 3–6 months. According to laboratory tests including ESR, CRP and IgA levels, BASDAI, BASFI, ASDAS results and sacroiliac joint SPARCC scores, the interval of TNF-i treatment was prolonged gradually. Fat metaplasia, bone erosion, sclerosis and ankylosis changes on MRI were compared between baseline, 4–6 months and 1–2 years.
Results: After 3 months of treatment, inflammatory indexes, BASDI, BASFI, ASDAS and SPARCC scores were significantly lower than baseline ($P<0.05$). After 4–6 months of treatment, ESR, CRP and IgA levels were greatly lower than before (8 (3,25) *vs.* 2 (1,3) mm/h, 4.97 (1.83,14.5) *vs.* 1.71 (1.24,2.82) mg/l, 2.96±1.34 *vs.* 2.20±1.01mg/l, Table 1, $P<0.01$). Compared to baseline, significant reduction of BASDAI and BASFI score was observed in TNF-inhibitor

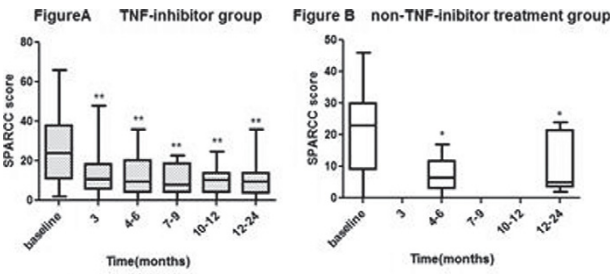

Figure 1 SPARCC score changes in TNF-inhibitor(A) and non-TNF-inhibitor(B) treatment group

Abstract THU0399 – Table 1. ESR, CRP and IgA changes before and after treatment

		Baseline	3 months	4–6 months	7–9 months	9–12 months	12–24 months
ESR (mm/h)	TNF-inhibitor	8 (3,25)	2 (1,3.75)**	2 (1,3)**	7 (2,9)	2.93 (2,6)	5.05 (2,17.75)
	non-TNF-inhibitor	10 (5,36)	–	2 (2,8)	–	–	4 (0.5,10.5)
CRP (mg/L)	TNF-inhibitor	4.97 (1.83,14.5)	1.54 (1.03,3.02)*	1.71 (1.24,2.82)**	4.89 (1.95,8.75)	2.01 (1.25,4.37)	4.21 (1.0,10.83)
	non-TNF-inhibitor	6.85 (3.11,16.05)	–	1.95 (1.59,5.34)	–	–	4.72 (1.8,7.43)
IgA (mg/L)	TNF-inhibitor	2.96±1.34	1.88±1.38	2.20±1.01**	1.92±1.63	1.78±1.32	2.45±1.63
	non-TNF-inhibitor	3.47±1.63	–	1.95±1.35	–	1.79±2.53	1.47±1.48

*P<0.05, **P<0.01.