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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:

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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.

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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 Adalimumab 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