equality. These evaluations were performed in pts randomized to UPA from baseline (BL; continuous UPA, assessed at wk 14) and those who were randomized to PBO and switched to UPA upon entry in the OLE (PBO to UPA; re-baselined at wk 14 and assessed at wk 32, representing 18 wks of UPA exposure).

Results: At wk 14, for the continuous UPA group, 16 pts (19%) achieved ASAS PR. At wk 32, following 18 wks of UPA exposure for the PBO-to-UPA group, 28 pts (33%) achieved ASAS PR. Among both groups (continuous UPA and PBO-to-UPA), improvements were seen across all core components (Figure 1). Of the 44 total pts who achieved ASAS PR, 91% achieved either ASDAS ID or LDA. The majority of patients who achieved ASAS PR achieved ASDAS ID in the continuous UPA and PBO-to-UPA groups: 11/16 (69%) and 16/28 (57%), respectively. For the continuous UPA group, the remaining 5 pts who achieved ASAS PR also achieved ASDAS LDA (Table 1). ASAS PR was associated with ASDAS categories in the following manner: the highest rate of ASAS PR was achieved for ASDAS ID followed by ASDAS LDA followed by ASDAS HDA/VHDA. The cutoff of 1.3 (the upper threshold for ASDAS ID) was a better discrimination threshold for ASAS PR than the cutoff of 2.1 (the upper threshold for ASDAS LDA).

Conclusion: Nineteen percent of pts receiving UPA from BL achieved ASAS PR after 14 wks of treatment, with similar results seen in pts who were originally randomized to PBO and switched to UPA at wk 14. A consistent improvement was seen across all core components of ASAS among those who achieved ASAS PR with UPA treatment. The achievement of ASAS PR was most closely associated with the achievement of ASDAS ID, providing further clarity on the reduction of disease activity in AS pts treated with UPA.

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Figure. Core ASAS (A) and ASDAS (B) Components Among Patients Who Achieved ASAS PR

*Inflammation based on mean of BASDAI question 5 and 6; peripheral pain/swelling based on BASDAI question 3; Back/neck/hip pain based on BASDAI question 2; duration of morning stiffness based on BASDAI question 6.
ASAS, Assessment of SpondyloArthritis International Society response criteria; ASDAS, AS Disease Activity Score; PBO, placebo; PR, partial remission; UPA, upadacitinib.

Table 1. Association Between ASAS PR and ASDAS Clinical Thresholds (ID/LDA/HDA or VHDA)

	ASDAS ID (<1.3)	ASDAS LDA (1.3 to <2.1)	ASDAS HDA or VHDA (≥2.1)
Continuous UPA Group	n=15	n=31	n=39
ASAS PR Responders (n=16)	11	5	0
ASAS PR Non-responders (n=69)	4	26	39
PBO to UPA Group	n=25	n=35	n=25
ASAS PR Responders (n=28)	16	8	4
ASAS PR Non-responders (n=57)	9	27	21

$P < 0.001$ for association of ASAS PR with the ordered ASDAS categories of ID-LDA-HDA, for both Continuous UPA Group and PBO to UPA Group. P -value calculated from Cochran-Armitage trend test for association of ordinal categories. ASAS, Assessment of SpondyloArthritis International Society response criteria; ASDAS, AS Disease Activity Score; HDA, high disease activity; ID, inactive disease; LDA, low disease activity; PBO, placebo; PR, partial remission; UPA, upadacitinib; VHDA, very high disease activity.

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POS0906

PREVALENCE OF SLEEP DISTURBANCE IN PATIENTS WITH ANKYLOSING SPONDYLITIS WITHIN THE AUSTRALIAN CLINICAL SETTING (ASLEEP STUDY): A REAL-WORLD OBSERVATIONAL STUDY USING THE OPAL DATASET

K. Tymms^{1,2}, B. Butcher^{3,4}, T. Sletten⁵, T. Smith², C. Osullivan², G. Littlejohn^{2,6}, R. Sadler⁷, R. Tronberg⁷, H. Griffiths^{2,8}. ¹Canberra Rheumatology, Rheumatology, Canberra, Australia; ²OPAL Rheumatology, Ltd., Sydney, Australia; ³WriteSource Medical, Pty Ltd, Lane Cove, Australia; ⁴UNSW Sydney, Sydney, Australia; ⁵Turner Institute for Brain and Mental Health, Monash University, Clayton, Australia; ⁶Dept of Medicine, Monash University, Clayton, Australia; ⁷Novartis Australia, Rheumatology, Macquarie Park, Australia; ⁸Barwon Rheumatology Services, Rheumatology, Geelong, Australia

Background: Sleep disorders are more prevalent in patients with ankylosing spondylitis (AS) compared to the general population. Sleep disturbance in AS, in addition to pain and fatigue, can lead to impaired physical function and reduced quality of life.

Objectives: The primary objective was to determine the prevalence of sleep disturbance in patients with AS in a real-world Australian cohort using the Insomnia Severity Index (ISI) and Multivariate Apnoea Prediction Index (MAPI). ISI score of ≥ 15 is considered clinical insomnia. MAPI values below 0.05 are suggestive of clinical apnoea.

Methods: Routinely collected, de-identified clinical data were sourced from the OPAL dataset. Patients aged between 18 and 95 years with a diagnosis of AS and who had completed at least one ISI or MAPI questionnaire between Jan-2019 and Sept-2020 were included. ISI and MAPI questionnaires were emailed to patients using OPAL's electronic patient reported outcome (ePRO) delivery method or completed in the clinic using a smart device and returned to the patient's file using a QR code. Disease activity was assessed using BASDAI collected at the same time as the sleep questionnaires. Age, sex and duration of symptoms were used to propensity match patients in the IL-17ai and TNFi group in a 1:2 ratio.

Results: 495 of the 5,323 patients identified with AS completed a questionnaire and were included in the analysis (n=395 TNFi, n=48 IL-17ai (secukinumab), n=52 other therapies). 142 were included in the propensity score matched population (n = 94 TNFi, and n = 48 IL-17ai). In the overall population the mean (SD) age was 48.3 (13.6), 55.4% were males, the mean (SD) BMI was 30.1 (19.6) at the index date and 4.8% reported depression. 51.7% had an optimal disease control (BASDAI <4). The mean (SD) ISI score was 8.6 (6.2). 48.1% reported no clinical significant insomnia, 32.7% reported subthreshold insomnia, 16% reported clinical insomnia (moderate severity) and 3.2% reported clinical insomnia (severe). The mean (SD) MAPI score was 0.4 (0.3). 292 patients (59.0%) had low risk of clinical apnoea, 134 patients (27.1%) had high risk of clinical apnoea and 69 patients (13.9%) had not completed the MAPI questionnaire. In the propensity score matched population, the TNFi and IL-17ai groups had mean (SD) ISI scores of 9.1 (6.6) and 8.9 (5.9) at index, respectively (p = 0.83) and mean (SD) MAPI scores of 0.3 (0.2) and 0.4 (0.3) at index, respectively (p=0.046), however a higher percentage of overweight and obese patients were identified in the IL-17ai treatment group. Ordered logistic regression analysis of the relationship between demographics and ISI in the matched population found that patients with BASDAI ≥ 4 were seven times more likely to experience greater sleep disturbance (OR 7.29, 95%CI 2.37 to 22.46, p=0.001) than those with BASDAI <4.

Conclusion: In this real-world AS cohort, poor disease control was associated with sleep disturbance, despite bDMARD therapy. Little difference was observed between TNFi and IL-17ai treatment. Screening for sleep disturbance and fatigue in routine clinical care may provide a more holistic view of the burden of this disease.

Table 1. Patient characteristics and outcome scores in the propensity score matched population at index.

	TNFI (n=94)	IL-17ai (n=48)	p value
BMI category Underweight	2 (2%)	1 (2%)	0.094
Normal weight	33 (35%)	8 (17%)	
Overweight	24 (26%)	16 (33%)	
Obese	27 (29%)	21 (44%)	
Missing	8 (9%)	2 (4%)	
Duration of treatment (months), mean (SD)	61.0 (156.7)	23.7 (20.1)	0.18
BASDAI <4	43 (46%)	23 (48%)	0.89
BASDAI >4	16 (17%)	8 (17%)	
Missing	35 (37%)	17 (35%)	
ISI score, mean (SD)	9.1 (6.6)	8.9 (5.9)	0.83
ISI category (n %)			0.83
No clinically significant insomnia	44 (57%)	24 (50%)	
Subthreshold insomnia	30 (32%)	15 (31%)	
Clinical insomnia (moderate)	15 (16%)	8 (17%)	
Clinical insomnia (severe)	5 (5%)	1 (2%)	
MAPI score, mean (SD)	0.3 (0.2)	0.4 (0.3)	0.046
MAPI, high apnoea risk Yes	17 (18%)	16 (33%)	0.051
No	62 (66%)	26 (54%)	
Missing	15 (16%)	6 (12%)	

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POS0907

EFFECT OF UPADACITINIB ON REDUCING PAIN IN PATIENTS WITH ACTIVE ANKYLOSING SPONDYLITIS AND INADEQUATE RESPONSE TO NONSTEROIDAL ANTI-INFLAMMATORY DRUGS

A. Deodhar¹, X. Baraliakos², I. McInnes³, K. De Vlam⁴, L. Bessette⁵, A. Maniccia⁶, R. Lippe⁷, C. Saffore⁶, T. Gao⁶, I. H. Song⁶, A. Ostor⁸. ¹Oregon Health & Science University, Rheumatology, Portland, United States of America; ²Ruhr-University Bochum, Rheumatology, Bochum, Germany; ³University of Glasgow, Rheumatology, Glasgow, United Kingdom; ⁴University Hospitals Leuven, Rheumatology, Leuven, Belgium; ⁵Laval University, Rheumatology, Québec City, Canada; ⁶AbbVie Inc., Rheumatology, North Chicago, United States of America; ⁷AbbVie Deutschland GmbH & Co. KG, Rheumatology, Wiesbaden, Germany; ⁸Cabrini Hospital, Monash University & Emeritus Research, Melbourne, Australia

Background: Pain is a debilitating symptom of ankylosing spondylitis (AS) and negatively impacts patient (pt) lives. Upadacitinib (UPA), a Janus kinase (JAK) inhibitor engineered for increased selectivity for JAK1 over JAK2, JAK3, and tyrosine kinase 2, ¹ showed significant efficacy vs placebo (PBO) in the randomized phase 2/3 SELECT-AXIS 1 study in active AS.²

Objectives: To evaluate the efficacy of UPA on multiple pain assessments through 64 weeks (wks).

Methods: SELECT-AXIS 1 (NCT03178487) enrolled adults with active AS, who had an inadequate response, intolerance or contraindications to ≥ 2 NSAIDs, were biologic DMARD naive and met the modified New-York Criteria. Pts were randomized 1:1 to UPA 15 mg once daily (QD, n=93) or PBO (n=94) for 14 wks (Period 1), followed by open-label UPA 15 mg QD during 90-wk extension (Period 2); reported here are data through wk 64. Pain endpoints included the proportion of pts achieving $\geq 30\%$ / $\geq 50\%$ / $\geq 70\%$ reduction in Pt's Global Assessment (PGA) of pain on a 0–10 numeric rating scale (NRS), minimal clinically important difference (MCID, defined as ≥ 1 point reduction or $\geq 15\%$ reduction from baseline [BL]) in PGA of pain, and much better improvement (MBI, defined as ≥ 2 point reduction and $\geq 33\%$ reduction from BL) in PGA of pain. In addition, mean change from baseline in PGA of pain, BASDAI questions 2 (neck/back/hip pain) and 3 (peripheral pain/swelling), and pt's assessment of back pain and nocturnal back pain NRS scores (NRS 0–10) were assessed. Non-responder imputation (binary endpoints) and mixed-effects model for repeated measurements (continuous endpoints) were used

for missing data/dropouts in Period 1; as-observed analysis was used for Period 2.

Results: A significantly higher proportion of pts receiving UPA vs PBO achieved reductions in all PGA of pain assessments as early as wk 2 that was sustained at all time points in Period 1; the only exception was $\geq 70\%$ reduction in PGA of pain that was significant at wk 4 and sustained thereafter (Figure 1). For $\geq 30\%$ / $\geq 50\%$ / $\geq 70\%$ reduction and MBI, the response rate increased over time with UPA; the difference for UPA vs PBO also continued to increase over time for $\geq 50\%$ and $\geq 70\%$ reduction endpoints. For MCID, an increase from BL to wk 2 was observed and plateaued thereafter. The mean change from BL in PGA of pain, BASDAI Q2, back pain, and nocturnal back pain NRS scores were significantly greater for UPA vs PBO at all time points in Period 1; BASDAI Q3 was significant at wk 8 and 14 (Figure 1). The effect of UPA on pain reduction was sustained through wk 64. PBO pts who switched to open-label UPA at wk 14 generally reached the same level of pain reduction as those initially randomized to UPA (Figure 1).

Conclusion: In pts with active AS and an inadequate response/contraindication to NSAIDs, a greater proportion of patients treated with UPA achieved rapid, significant, and clinically meaningful reductions in pain vs PBO through 14 wks across multiple pain assessments. The reductions in pain were sustained over time, and pts who switched from PBO to UPA reached the same level of improvement as the continuous UPA group.

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Figure. Proportion of Patients Achieving Pain Endpoints ($\geq 30\%$ / $\geq 50\%$ / $\geq 70\%$ Reduction from Baseline and MBI or MCID in PGA of Pain) and Mean Change from Baseline in PGA of Pain, BASDAI Q2 and Q3, Back Pain, and Nocturnal Back Pain

AO, as observed; MBI, much better improvement; MCID, minimal clinically important difference; MMRM, mixed-effects model for repeated measurements; NRI, non-responder imputation; NRS, numeric rating scale; PGA, patient's global assessment; UPA, upadacitinib. Dashed line: all patients randomized to placebo received open-label UPA starting from week 14. UPA vs PBO comparison was calculated using Cochran-Mantel-Haenszel test adjusting for stratification factor of high-sensitivity CRP level; nominal P values: ***P<0.001, **P<0.01, *P<0.05. Patient's Global Assessment of Pain based on: "How much pain have you had because of your condition during the last week?" BASDAI Q2 based on a question: "How would you describe the overall level of AS neck, back or hip pain you have had?" BASDAI Q3 based on a question: "How would you describe the overall level of pain/swelling in joints other than neck, back or hips, you have had?" Patient's assessment of back pain based on question: "What is the amount of back pain that you experienced at any time during the last week?" Patient's assessment of nocturnal back pain based on a question: "What is amount of back pain at night that you experienced during the last week?"

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