

# Upadacitinib in patients with psoriatic arthritis and inadequate response to biologics: 3-year results from the open-label extension of the randomised controlled phase 3 SELECT-PsA 2 study

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## Abstract

### Objective

To assess the long-term safety and efficacy of upadacitinib in patients with psoriatic arthritis (PsA) and an inadequate response (IR) to biologic disease-modifying anti-rheumatic drugs (bDMARDs) who completed up to 152 weeks of treatment in the SELECT-PsA 2 study (ClinicalTrials.gov: NCT03104374).

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### Methods

Patients were randomised to receive blinded upadacitinib 15 or 30 mg once daily (QD), or placebo for 24 weeks followed by upadacitinib 15 or 30 mg QD. After 56 weeks, patients were eligible to enter an open-label extension (OLE) in which they continued their assigned dose of upadacitinib. Efficacy and safety were assessed through 152 weeks. A subanalysis of patients with IR to tumour necrosis factor inhibitors (TNFis) was also conducted.

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### Results

In total, 450 patients entered the OLE and 358 completed 152 weeks of treatment. Improvements in efficacy outcomes observed at week 56, including the proportion of patients achieving: 20/50/70% improvement in American College of Rheumatology criteria, minimal disease activity, and 75/90/100% improvement in Psoriasis Area and Severity Index, were maintained through week 152. Efficacy outcomes in the TNFi-IR subgroup were similar to those reported in the overall population. Upadacitinib was well tolerated throughout long-term treatment, with no cumulative adverse effects observed through 152 weeks.

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### Conclusion

Efficacy of upadacitinib was maintained up to 152 weeks of treatment in this highly treatment-refractory population of patients with PsA. The long-term safety profile of upadacitinib 15 mg was consistent with its known safety profile across indications; no new safety signals were identified.

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### Key words

upadacitinib, psoriatic arthritis, safety, efficacy

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#### Data availability

AbbVie is committed to responsible data sharing regarding the clinical trials we sponsor. This includes access to anonymised, individual, and trial-level data (analysis datasets), as well as other information (e.g. protocols, clinical study reports, or analysis plans), provided the trials are not part of an ongoing or planned regulatory submission.

This includes requests for clinical trial data for unlicensed products and indications. These clinical trial data can be requested by any qualified researchers who engage in rigorous, independent scientific research, and will be provided following review and approval of a research proposal and statistical analysis plan, and execution of a Data Sharing Agreement.

Data requests can be submitted at any time after approval in the US and Europe and after acceptance of this manuscript for publication. The data will be accessible for 12 months, with possible extensions considered. For more information on the process, or to submit a request, visit: <https://www.abbvieclinicaltrials.com/hcp/data-sharing>.

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**Competing interests:** see page 2296.

#### Introduction

Psoriatic arthritis (PsA) is a chronic, immune-mediated, inflammatory, musculoskeletal disease characterised by pain and stiffness due to inflammation of the peripheral and axial joints, enthesitis, and dactylitis, and is often accompanied by skin and nail psoriasis (1, 2). Several pharmacologic therapies are available for patients with PsA, including conventional synthetic disease-modifying anti-rheumatic drugs (csDMARDs), as well as more advanced therapies, including biologic DMARDs (bDMARDs), such as tumour necrosis factor inhibitors (TNFis), and targeted synthetic DMARDs, such as apremilast and Janus kinase (JAK) inhibitors (3).

However, despite the wide availability of treatment options, some patients still fail to achieve treatment targets, representing an unmet need. Data on the subpopulation of patients in whom prior bDMARD treatment has failed are currently limited, particularly among highly treatment-refractory patients who have previously experienced treatment failure with >1 bDMARD (4, 5). Unlike most advanced therapies for PsA, which are administered via injection, the reversible JAK inhibitor upadacitinib is taken orally. In addition, upadacitinib is engineered for increased selectivity for JAK1 over JAK2, JAK3, and tyrosine kinase 2 (6). Treatment with upadacitinib is approved for several immunologic conditions, including rheumatoid arthritis (RA), PsA, axial spondyloarthritis (non-radiographic axial spondyloarthritis and ankylosing spondylitis [AS]), atopic dermatitis (AD), and ulcerative colitis (7, 8)). In the randomised, double-blind, placebo-controlled SELECT-PsA 2 study, the efficacy and safety of upadacitinib 15 and 30 mg once daily (QD) were assessed in patients with PsA and inadequate response or intolerance to bDMARDs (bDMARD-IR) (9). After 24 weeks, upadacitinib was significantly more effective than placebo in improving the signs and symptoms of PsA (9). Patients randomised to placebo were then switched to double-blind upadacitinib 15 or 30 mg QD, and efficacy was maintained through 56 weeks, with no new safety signals (10). Here we present efficacy and safety data from patients

who completed up to 152 weeks of upadacitinib treatment in SELECT-PsA 2, including a subanalysis of patients with prior exposure to TNFi (TNFi-IR).

#### Methods

##### Study design

In SELECT-PsA 2 (ClinicalTrials.gov: NCT03104374; start date May 1, 2017, data cut-off May 5, 2022), patients were initially randomised to receive upadacitinib 15 or 30 mg QD, or placebo for 24 weeks followed by upadacitinib 15 or 30 mg QD. Blinding was maintained until week 56, at which time patients were eligible to enter an open-label extension (OLE) in which they continued their assigned upadacitinib dose (10). Following approval of the 15 mg QD dose, the protocol was amended and all patients receiving upadacitinib 30 mg QD in the OLE were switched to the approved dose, which occurred at different visits across patients, with the earliest switch occurring at week 116.

Background therapy was permitted throughout the study with stable doses of non-steroidal anti-inflammatory drugs, corticosteroids (up to 10 mg/day prednisone or equivalent), or  $\leq 2$  non-biologic DMARDs, except for the combination of methotrexate and leflunomide (9). Patients who did not achieve a reduction of  $\geq 20\%$  from baseline in tender joint count in 68 joints (TJC68) and swollen joint count in 66 joints (SJC66) at weeks 12 and 16 were required to initiate or adjust background medication at week 16. After week 36, all patients were permitted to initiate or adjust background therapy, but any patient who did not achieve a  $\geq 20\%$  reduction in baseline TJC68 and/or SJC66 at two consecutive visits was required to discontinue study medication.

The study was conducted according to the International Conference on Harmonisation guidelines and the Declaration of Helsinki. The trial protocol was approved by independent ethics committees and institutional review boards. The full study design of SELECT-PsA 2 has been published previously (9).

##### Patients

The SELECT-PsA 2 study enrolled adults (age  $\geq 18$  years) with active

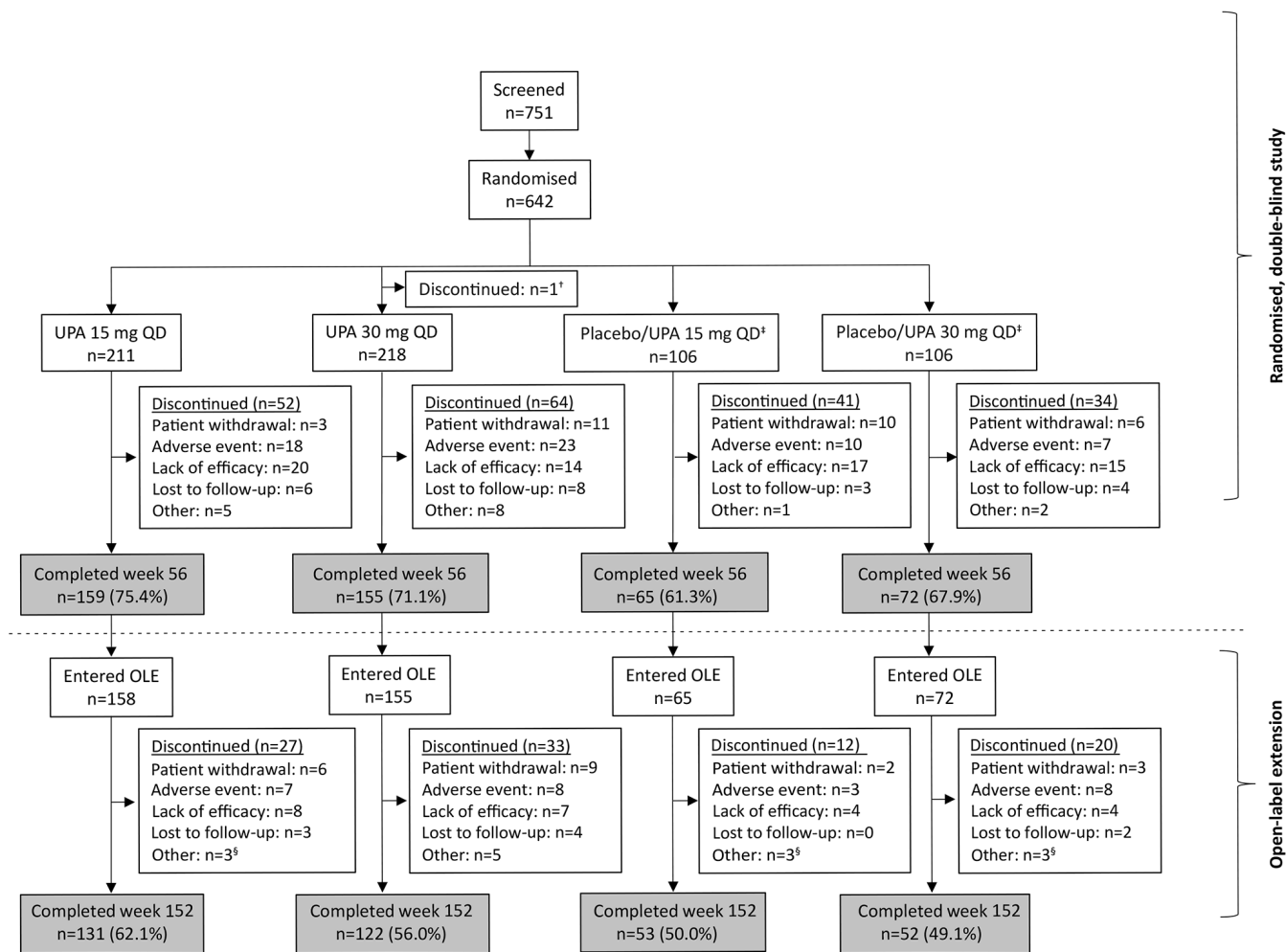


Fig. 1. Patient disposition over 152 weeks, based on study treatment status.

†One patient randomised to UPA 30 mg QD did not receive study medication. ‡Patients randomised to placebo switched to their assigned dose of UPA after 24 weeks. §One patient discontinued due to COVID-19 infection. OLE: open-label extension; QD: once daily; UPA: upadacitinib.

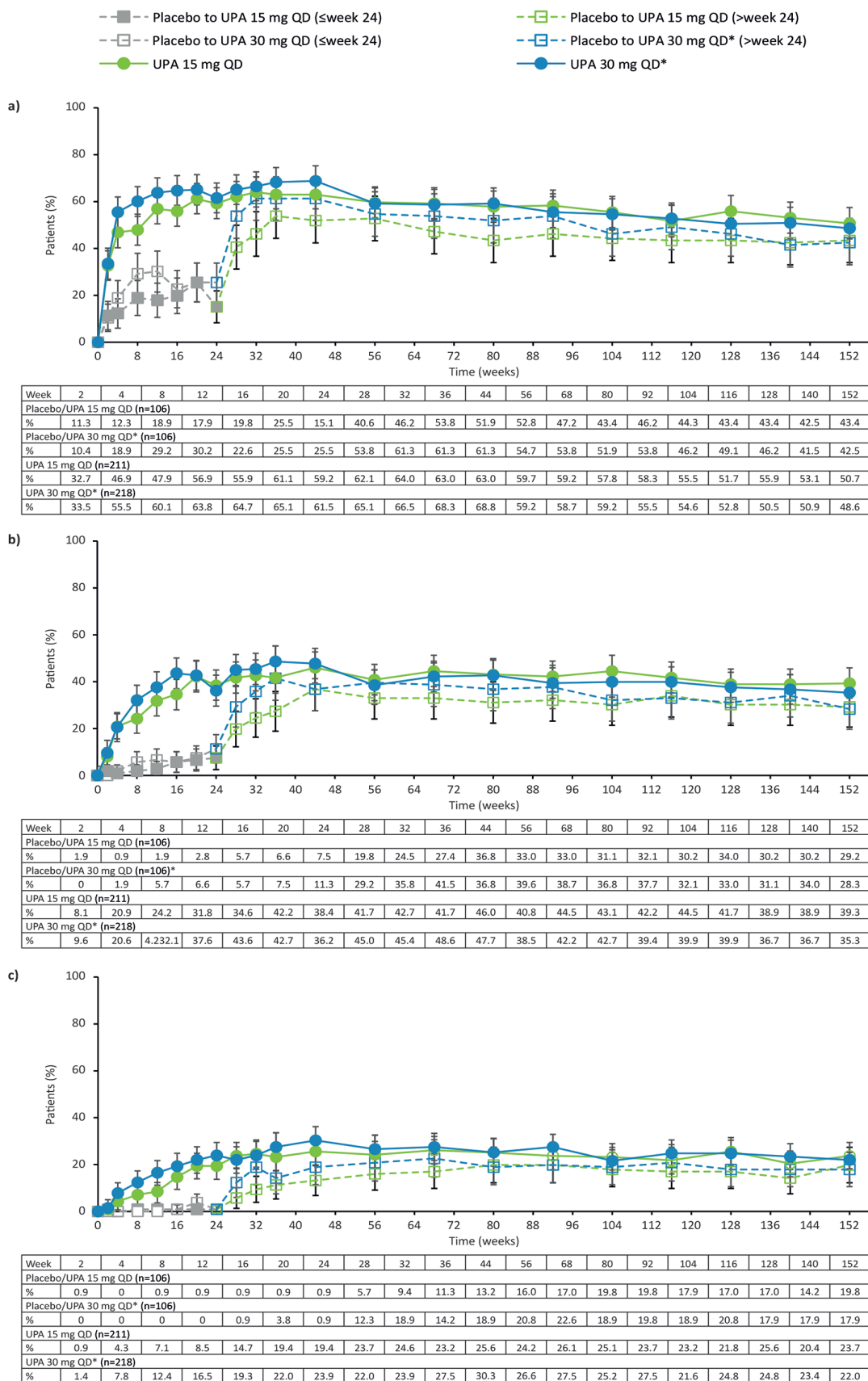
PsA who had plaque psoriasis (active or documented history), SJC66 and TJC68  $\geq 3$  at screening and at baseline, and inadequate response or intolerance to  $\geq 1$  bDMARD (9). Patients who had previously received a JAK inhibitor were excluded. Patients with a history of fibromyalgia were excluded, unless there was documentation of change in diagnosis to PsA, or documentation that the prior diagnosis of fibromyalgia was made incorrectly. Full inclusion and exclusion criteria for SELECT-PsA 2 have been published previously (9). All patients provided written, informed consent before any study procedures were performed.

#### Outcome measures

The primary endpoint of the SELECT-PsA 2 study was the proportion of

patients who achieved 20% improvement in American College of Rheumatology criteria (ACR20) at week 12 (9). Efficacy outcomes assessed in the OLE included: ACR20/50/70; minimal disease activity (MDA); 75/90/100% improvement in Psoriasis Area and Severity Index (PASI 75/90/100) in patients with affected body surface area  $\geq 3\%$  at baseline; static Investigator's Global Assessment (sIGA) score 0/1 and improvement of  $\geq 2$  points from baseline; change from baseline in: Physician's Global Assessment of Disease Activity (PhGA), SJC66, TJC68, and high-sensitivity C-reactive protein (hsCRP); and resolution of dactylitis and enthesitis (Leeds Dactylitis Index [LDI] =0 and Leeds Enthesitis Index [LEI] =0 in patients with baseline LDI >0 and LEI >0, respectively).

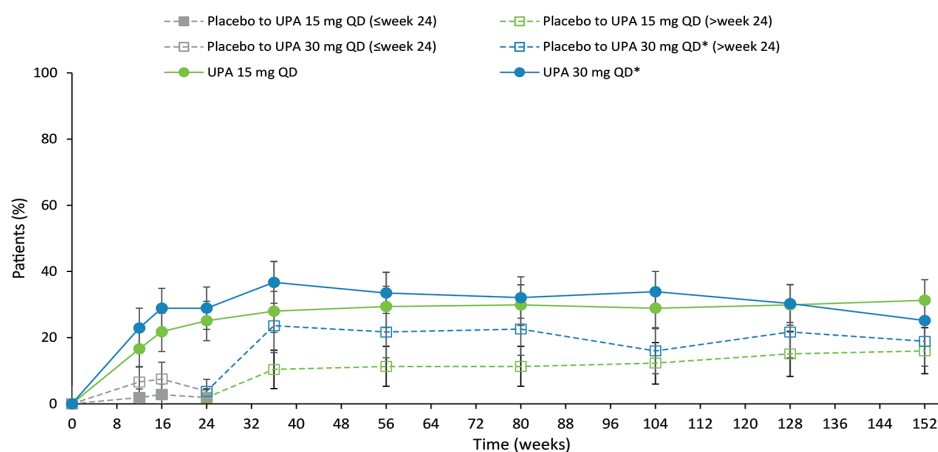
In addition, several patient-reported outcomes (PROs) were assessed, including change from baseline in: Health Assessment Questionnaire-Disability Index (HAQ-DI), Functional Assessment of Chronic Illness Therapy-Fatigue (FACIT-F); 36-item Short-Form questionnaire Physical and Mental Component Summaries, Self-Assessment of Psoriasis Symptoms, Work Productivity and Activity Impairment overall work impairment score, Patient's Global Assessment, patient's assessment of pain based on a 0–10 numeric rating scale, and morning stiffness (mean score of Bath Ankylosing Spondylitis Disease Activity Index [BASDAI] questions 5 and 6). In patients with investigator-determined axial PsA at baseline, changes in BASDAI and Ankylosing Spondylitis Disease Activity Score (ASDAS),



**Fig. 2.** Proportion of patients achieving a) ACR20, b) ACR50, and c) ACR70 over 152 weeks (NRI).  
 \*All patients receiving UPA 30 mg QD were switched to UPA 15 mg QD during the open-label extension. The switch occurred at different visits across patients, with the earliest switch occurring at the week 116 visit.  
 ACR20/50/70: 20/50/70% improvement in American College of Rheumatology criteria; NRI: non-responder imputation; QD: once daily; UPA: upadacitinib.

**Fig. 3.** Proportion of patients achieving minimal disease activity over 152 weeks (NRI).

\*All patients receiving UPA 30 mg QD were switched to UPA 15 mg QD during the open-label extension. The switch occurred at different visits across patients, with the earliest switch occurring at the week 116 visit. NRI: non-responder imputation; QD: once daily; UPA: upadacitinib.



Week	12	16	24	36	56	80	104	128	152
Placebo/UPA 15 mg QD (n=106)									
%	1.9	2.8	1.9	10.4	11.3	11.3	12.3	15.1	16.0
Placebo/UPA 30 mg QD* (n=106)									
%	6.6	7.5	3.8	23.6	21.7	22.6	16.0	21.7	18.9
UPA 15 mg QD (n=211)									
%	16.6	21.8	25.1	28.0	29.4	29.9	28.9	29.9	31.3
UPA 30 mg QD* (n=218)									
%	22.9	28.9	28.9	36.7	33.5	32.1	33.9	30.3	25.2

and proportions of patients achieving 50% improvement in BASDAI score (BASDAI 50) at week 152, were also assessed.

Adverse events (AEs) were reported throughout the OLE and coded per the Medical Dictionary for Regulatory Activities version 24.1. Major adverse cardiovascular events (MACE) and venous thromboembolic events (VTE) were adjudicated by an independent external cardiovascular adjudication committee, and gastrointestinal perforation events were adjudicated by an internal gastrointestinal perforation adjudication committee. Both committees were blinded to treatment assignment.

*Statistical analysis*

No formal statistical comparisons between groups were conducted for long-term efficacy outcomes. Efficacy and safety analyses were conducted on the full analysis set, including all randomised patients who received ≥1 dose of study drug. Efficacy and safety data are also presented for the TNFi-IR subgroup of patients.

For binary endpoints, data are presented using non-responder imputation (NRI) and as-observed (AO). NRI was used for missing data and study drug discontinuation. Patients who were rescued at week 16 were considered non-responders for MDA, resolution

of enthesitis, and resolution of dactylitis. For continuous endpoints, AO data at all visits were analysed using mixed effect model for repeated measures (MMRM), including treatment, visit, treatment-by-visit interaction, and current DMARD use (yes/no) as fixed factors, with least squares means and 95% confidence intervals (CIs) for each randomised treatment group sequence. Patients’ discontinuation status was also included in the models, and Toeplitz or Variance Components covariance structure was used (11).

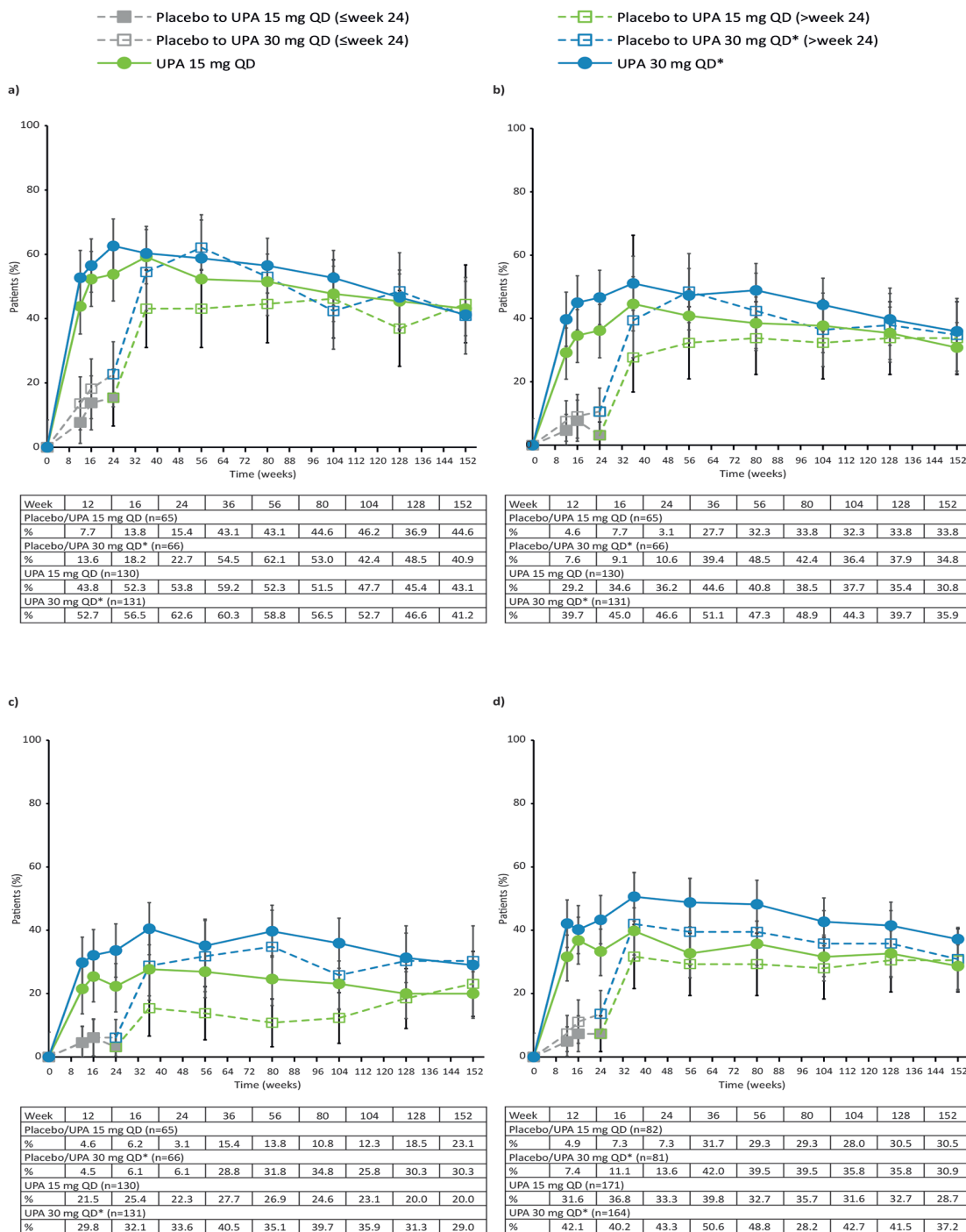
Safety was analysed based on the dose of upadacitinib received or, in the case of placebo patients, to which dose they were switched at week 24. The exception to this was patients who received upadacitinib 30 mg QD but were later switched to 15 mg QD. In these patients, AEs occurring during treatment with upadacitinib 30 mg QD were included in the overall 30 mg QD group, while those occurring after switching to upadacitinib 15 mg QD were analysed separately and were not included in the overall 15 mg QD group. Treatment-emergent AEs (TEAEs) are summarised for events occurring after the first dose of upadacitinib and ≤30 days after discontinuing study medication. Exposure-adjusted event rates (EAERs) are summarised as events per 100 patient-years (PY), with 95% CIs

calculated based on the Poisson distribution. Deaths are reported including treatment-emergent and non-treatment-emergent deaths.

**Results**

*Patients*

In total, 641 patients were randomised and received at least one dose of study treatment (upadacitinib 15 mg, n=211; upadacitinib 30 mg, n=218, placebo/upadacitinib 15 mg, n=106; placebo/upadacitinib 30 mg, n=106), of whom 451 (70.4%) completed 56 weeks of double-blind treatment (Fig. 1) (9, 10). Baseline demographics of the study population have been published previously (9). Mean duration since PsA diagnosis was 10.1 years across treatment arms, with mean baseline SJC66 of 12.1, TJC68 of 24.8, and PASI of 10.2. Overall, 66.9% of patients had enthesitis (LEI >0) and 26.4% had dactylitis (LDI >0). Over half of patients had a prior inadequate response to one bDMARD (n=391; 61.0%), while 116 patients (18.1%) had an inadequate response to two bDMARDs and 83 (12.9%) had an inadequate response to ≥3 bDMARDs. The remaining 51 patients (8.0%) were intolerant to prior bDMARDs and did not have a prior lack of efficacy. Overall, 501 patients (78.2%) had previously received TNFi, among whom 311 (48.5%) had



**Fig. 4.** Proportion of patients achieving a) PASI 75<sup>†</sup>, b) PASI 90<sup>‡</sup>, and c) PASI 100<sup>‡</sup>, or d) sIGA 0/1 and ≥2-point improvement from baseline, over 152 weeks (NRI). <sup>†</sup>PASI response rate assessed in patients with psoriasis body surface area ≥3% at baseline. \*All patients receiving UPA 30 mg QD were switched to UPA 15 mg QD during the open-label extension. The switch occurred at different visits across patients, with the earliest switch occurring at the week 116 visit. NRI: non-responder imputation; PASI 75/90/100: 75/90/100% reduction in Psoriasis Area and Severity Index; QD: once daily; sIGA: static Investigator Global Assessment of Psoriasis; UPA: upadacitinib.

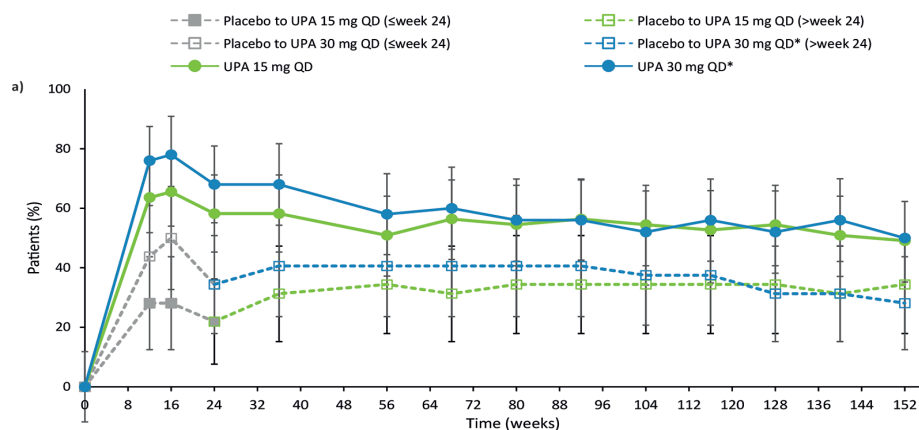
received one prior TNFi, 119 (18.6%) had received two prior TNFis, and 71 (11.1%) had received ≥3 prior TNFis. At baseline, 296 patients (46.2%) were receiving concomitant non-biologic DMARDs, including 222 (34.6%) receiving methotrexate alone, 18 (2.8%)

receiving methotrexate with another DMARD, and 56 (8.7%) receiving DMARDs other than methotrexate (9). Of the 451 patients who completed the double-blind period, 450 entered the OLE (upadacitinib 15 mg, n=158; upadacitinib 30 mg, n=155, placebo/

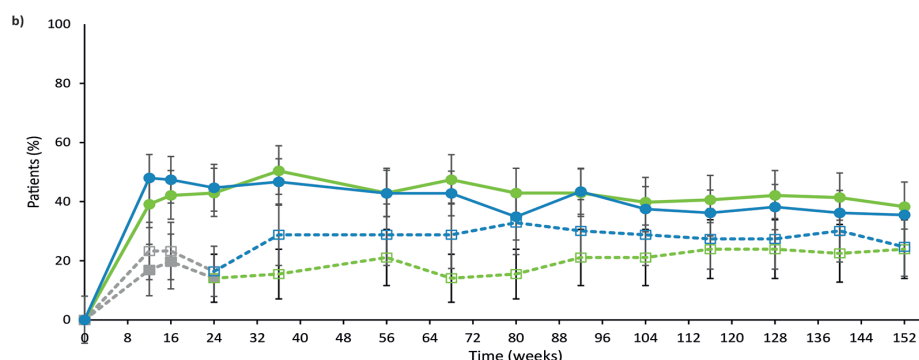
upadacitinib 15 mg, n=65; placebo/upadacitinib 30 mg, n=72) (Fig. 1). In total, 62.1% (n=131), 56.0% (n=122), 50.0% (n=53), and 49.1% (n=52) patients, respectively, completed 152 weeks of treatment. The most common primary reasons for discontinuation

**Fig. 5.** Proportion of patients achieving a) resolution of dactylitis (LDI = 0<sup>†</sup>) and b) resolution of enthesitis (LEI = 0<sup>‡</sup>) over 152 weeks (NRI).

<sup>†</sup>For patients with baseline LDI >0.  
<sup>‡</sup>For patients with baseline LEI >0.  
 \*All patients receiving UPA 30 mg QD were switched to UPA 15 mg QD during the open-label extension. The switch occurred at different visits across patients, with the earliest switch occurring at the week 116 visit.  
 LDI: Leeds Dactylitis Index; LEI: Leeds Enthesitis Index; NRI: non-responder imputation; QD: once daily; UPA: upadacitinib.



Week	12	16	24	36	56	68	80	92	104	116	128	140	152
Placebo/UPA 15 mg QD (n=32)													
%	28.1	28.1	21.9	31.3	34.4	31.3	34.4	34.4	34.4	34.4	34.4	31.3	34.4
Placebo/UPA 30 mg QD* (n=32)													
%	43.8	50.0	34.4	40.6	40.6	40.6	40.6	40.6	37.5	37.5	31.3	31.3	28.1
UPA 15 mg QD (n=55)													
%	63.6	65.5	58.2	58.2	50.9	56.4	54.5	56.4	54.5	52.7	54.5	50.9	49.1
UPA 30 mg QD* (n=50)													
%	76.0	78.0	68.0	68.0	58.0	60.0	56.0	56.0	52.0	56.0	52.0	56.0	50.0



Week	12	16	24	36	56	68	80	92	104	116	128	140	152
Placebo/UPA 15 mg QD (n=71)													
%	16.9	19.7	14.1	15.5	21.1	14.1	15.5	21.1	21.1	23.9	23.9	22.5	23.9
Placebo/UPA 30 mg QD* (n=73)													
%	23.3	23.3	16.4	28.8	28.8	28.8	32.9	30.1	28.8	27.4	27.4	30.1	24.7
UPA 15 mg QD (n=133)													
%	39.1	42.1	42.9	50.4	42.9	47.4	42.9	42.9	39.8	40.6	42.1	41.4	38.3
UPA 30 mg QD* (n=152)													
%	48.0	47.4	44.7	46.7	42.8	42.8	34.9	43.4	37.5	36.2	38.2	36.2	35.5

during the OLE were AEs, lack of efficacy, and patient withdrawal (4.1%, 3.6%, and 3.1%, respectively, across treatment groups).

**Efficacy**

During the randomised, double-blind portion of SELECT-PsA 2, the proportions of ACR20/50/70 responders increased rapidly from baseline (Fig. 2; Supplementary Fig. S1). Responses previously observed at week 56 (10) were maintained through to week 152 in the OLE, with ACR20 achieved by 50.7%, 48.6%, 43.4%, and 42.5% of patients in the upadacitinib 15 mg QD, upadacitinib 30 mg QD, placebo/upadacitinib 15 mg QD, and placebo/upadacitinib 30 mg QD arms, respectively

(NRI; Fig. 2a); as expected, higher response rates were observed in AO data (80.6%, 78.6%, 87.0%, and 79.3%, respectively; Suppl. Fig. S1a). Similar trends were observed for ACR50/70 (Fig. 2b-c; Suppl. Fig. 1b-c).

In the randomised, double-blind portion of the study, MDA was achieved by more patients at week 56 in the initial active treatment arms compared with the initial placebo arms (Fig. 3; Suppl. Fig. S2). Week 56 responses were maintained through to week 152, with the highest proportion of MDA responders (31.3% [NRI] and 44.4% [AO]) observed in the continuous UPA 15 mg arm at week 152 (Fig. 3; Suppl. Fig. S2). The condition of patients' psoriasis, assessed in terms of PASI 75/90/100,

also improved from baseline to week 56 in the initial double-blind portion of the study, with maintenance of responses through to week 152 in the OLE (Fig. 4a-c; Suppl. Fig. S3a-c). Similar results were observed in terms of sIGA 0/1 and ≥2-point improvement (Fig. 4d; Suppl. Fig. S3d). Of patients with dactylitis at baseline (LDI >0), 49.1% and 50.0% of patients in the upadacitinib 15 and 30 mg QD arms, respectively, achieved resolution of dactylitis (LDI = 0) at week 152, while these proportions were 34.4% and 28.1% in the respective placebo to upadacitinib arms (NRI; Fig. 5a). In the AO data, 94.4% of patients in the upadacitinib 15 mg QD arm and 100.0% in the three other arms achieved resolution of dac-

**Table I.** Continuous efficacy endpoints at week 152 (MMRM<sup>†</sup> on as-observed data).

Parameter	UPA 15 mg QD		UPA 30 mg QD*		Placebo/upadacitinib 15 mg QD		Placebo/upadacitinib 30 mg QD*	
	n <sup>‡</sup>	LSM change from baseline (95% CI)	n <sup>‡</sup>	LSM change from baseline (95% CI)	n <sup>‡</sup>	LSM change from baseline (95% CI)	n <sup>‡</sup>	LSM change from baseline (95% CI)
PhGA (0–10 NRS)	210	-4.5 (-4.7, -4.2)	217	-4.4 (-4.7, -4.2)	103	-4.1 (-4.6, -3.7)	105	-4.3 (-4.7, -3.9)
SJC66	210	-10.4 (-10.7, -10.0)	217	-10.4 (-10.8, -10.0)	103	-10.3 (-10.8, -9.7)	105	-9.9 (-10.4, -9.3)
TJC68	210	-18.7 (-20.1, -17.3)	217	-17.5 (-18.9, -16.1)	103	-18.8 (-21.0, -16.6)	105	-18.3 (-20.3, -16.2)
hsCRP, mg/L	210	-5.5 (-6.9, -4.1)	217	-7.4 (-8.8, -6.0)	103	-5.7 (-7.9, -3.5)	105	-5.2 (-7.3, -3.0)
HAQ-DI score	207	-0.37 (-0.46, -0.29)	217	-0.45 (-0.54, -0.37)	103	-0.46 (-0.59, -0.33)	103	-0.30 (-0.43, -0.17)
FACIT-F score	206	+5.5 (3.9, 7.1)	213	+7.0 (5.4, 8.6)	96	+7.8 (5.3, 10.3)	101	+5.3 (2.9, 7.6)
SF-36 PCS	206	+6.79 (5.47, 8.11)	213	+7.58 (6.26, 8.91)	97	+6.28 (4.23, 8.33)	101	+4.76 (2.80, 6.73)
SF-36 MCS	206	+2.48 (1.06, 3.89)	213	+3.27 (1.84, 4.70)	97	+3.44 (1.22, 5.66)	101	+3.03 (0.91, 5.15)
SAPS score	200	-26.3 (-29.6, -23.0)	211	-26.6 (-30.0, -23.3)	95	-23.4 (-28.6, -18.1)	100	-27.8 (-32.8, -22.8)
WPAI overall work impairment	117	-13.99 (-20.70, -7.27)	116	-18.52 (-25.16, -11.88)	49	-9.06 (-19.88, 1.77)	42	-2.90 (-14.58, 8.79)
PtGA (0-10 NRS)	207	-2.9 (-3.3, -2.6)	217	-2.9 (-3.2, -2.5)	103	-2.3 (-2.9, -1.7)	103	-2.5 (-3.1, -1.9)
Pain (0-10 NRS)	207	-2.7 (-3.0, -2.3)	217	-2.5 (-2.8, -2.1)	103	-2.3 (-2.9, -1.8)	103	-2.5 (-3.0, -1.9)
Morning stiffness <sup>§</sup>	206	-2.48 (-2.84, -2.13)	213	-2.42 (-2.78, -2.06)	96	-2.53 (-3.09, -1.98)	101	-2.50 (-3.03, -1.97)
ASDAS <sup>§</sup>	73	-1.36 (-1.60, -1.12)	68	-1.14 (-1.39, -0.89)	35	-1.01 (-1.37, -0.65)	34	-1.17 (-1.52, -0.82)
BASDAI <sup>§</sup>	73	-2.56 (-3.07, -2.05)	68	-2.00 (-2.53, -1.46)	35	-2.32 (-3.11, -1.53)	34	-2.49 (-3.24, -1.73)
LDI <sup>**</sup>	54	-104.3 (-105.6, -103.0)	50	-104.3 (-105.7, -102.9)	31	-104.6 (-106.4, -102.7)	31	-105.7 (-107.5, -103.8)
LEI <sup>††</sup>	131	-2.1 (-2.4, -1.9)	149	-2.0 (-2.3, -1.8)	68	-2.4 (-2.8, -2.1)	70	-2.1 (-2.5, -1.8)

<sup>†</sup>MMRM analysis includes treatment, visit, treatment-by-visit interaction, and current DMARD use (yes/no) as fixed factors and baseline value as covariate. Patient's discontinuation status is also included in the model. Unstructured covariance structure is used. Data as observed at all visits are included in the model.

\*All patients receiving upadacitinib 30 mg QD were switched to upadacitinib 15 mg QD during the open-label extension. The switch occurred at different visits across patients, with the earliest switch occurring at the week 116 visit. <sup>‡</sup>Number of unique patients contributing to MMRM model estimates: patients with at least one available change from baseline value and no missing data for the factors and covariates in the model. <sup>§</sup>Mean of BASDAI questions 5 and 6.

<sup>§</sup>Among patients with psoriatic spondylitis at baseline, as determined by the investigator. <sup>\*\*</sup>For patients with baseline LDI >0. <sup>††</sup>For patients with baseline LEI >0. ASDAS: Ankylosing Spondylitis Disease Activity Score; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; CI: confidence interval; DMARD: disease-modifying anti-rheumatic drug; FACIT-F: Functional Assessment of Chronic Illness Therapy - Fatigue; HAQ-DI: Health Assessment Questionnaire - Disability Index; hsCRP: high-sensitivity C-reactive protein; LDI: Leeds Dactylitis Index; LEI: Leeds Enthesitis Index; LSM: least squares mean; MCS: mental component summary; MMRM: mixed effect model for repeated measures; NRS: Numeric Rating Scale; PCS: physical component summary; PhGA: Physician's Global Assessment; PtGA: Patient's Global Assessment; QD: once daily; SAPS: Self-Assessment of Psoriasis Symptoms; SF-36: 36-item Short-Form questionnaire; SJC66: swollen joint count in 66 joints; TJC68: tender joint count in 68 joints; UPA: upadacitinib; WPAI: Work Productivity and Activity Impairment.

tylitis at 152 weeks (Suppl. Fig. S4a). Of patients with enthesitis at baseline (LEI >0), 38.3% and 35.5% of patients in the upadacitinib 15 and 30 mg QD arms, respectively, achieved resolution of enthesitis at week 152, compared with 23.9% and 24.7% of those in the respective placebo arms (NRI; Fig. 5b). In the AO data, the proportion of patients achieving resolution of enthesitis ranged between 61.9 and 76.3% across treatment arms (Suppl. Fig. S4b).

Improvements from baseline in continuous efficacy measures (PhGA, SJC66, TJC68, and hsCRP) were seen at week 152 in all treatment groups (Table I). These were accompanied by improvements in several PROs relating to disease status and symptoms, quality of life, fatigue, and work impairment (Table I). In general, the magnitude of improvement at week 152 was similar across the four treatment arms. For example, least squares mean reductions

(improvements) from baseline in HAQ-DI score were -0.37, -0.45, -0.46, and -0.30 in the upadacitinib 15 mg QD, upadacitinib 30 mg QD, placebo/upadacitinib 15 mg QD, and placebo/upadacitinib 30 mg QD arms, respectively (AO/MMRM). Similarly, least squares mean improvements in BASDAI were -2.56, -2.00, -2.32, and -2.49, respectively, and in ASDAS were -1.36, -1.14, -1.01, and -1.17, respectively, in the upadacitinib 15 mg QD, upadacitinib 30 mg QD, placebo/upadacitinib 15 mg QD, and placebo/upadacitinib 30 mg QD arms (AO/MMRM; Table I). BASDAI 50 response rates at 152 weeks were 34.2%, 20.6%, 20.0%, and 25.7% (NRI) and 63.0%, 37.5%, 50.0%, and 47.4% (AO), respectively.

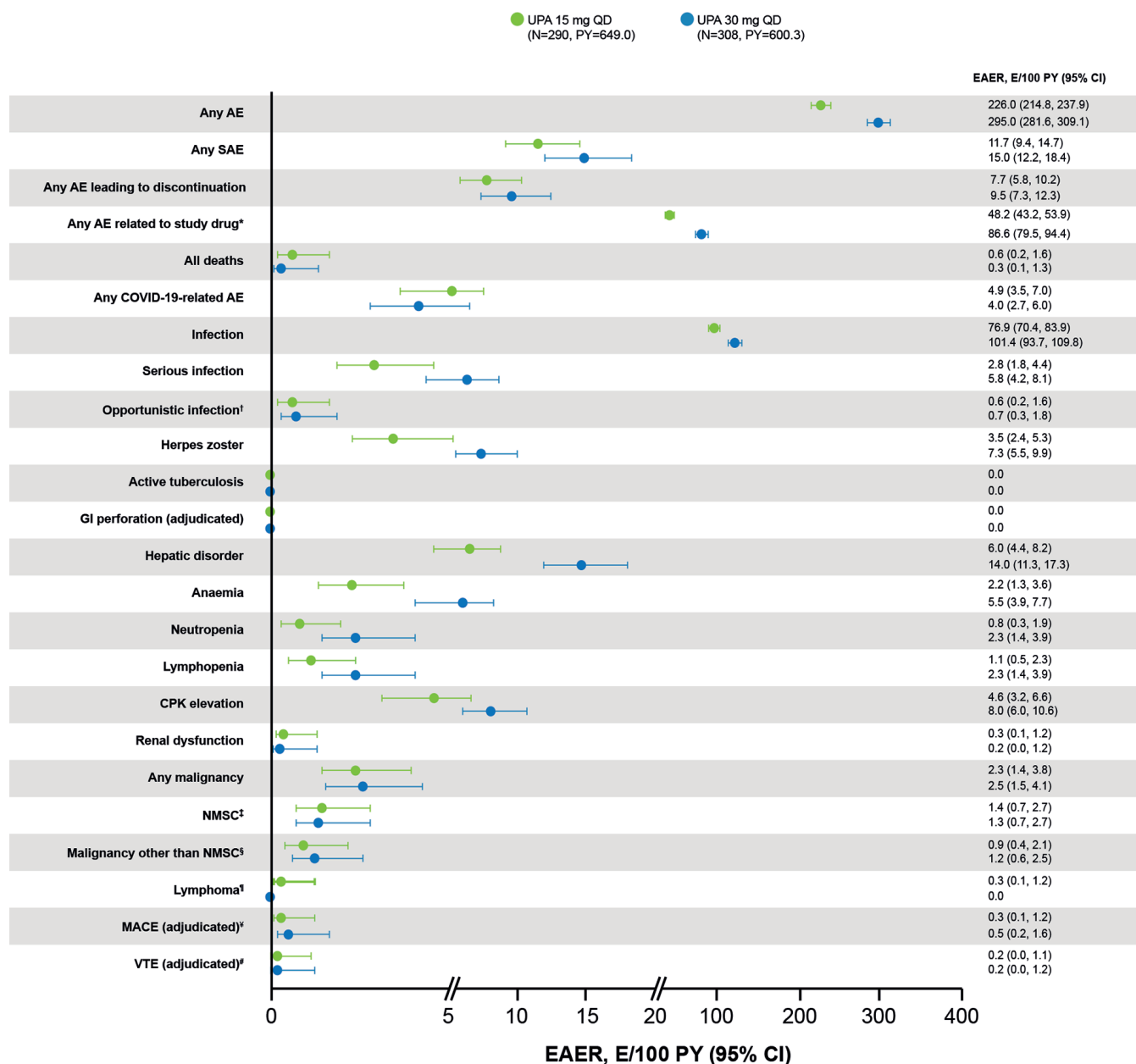
In the TNFi-IR subgroup of patients, the efficacy of upadacitinib was similar to that in the overall population (Suppl. Fig. S5-8; Suppl. Table S1). In the upadacitinib 15 mg QD, upadacitinib

30 mg QD, placebo/upadacitinib 15 mg QD, and placebo/upadacitinib 30 mg QD arms, ACR20 was achieved by 48.4%, 47.4%, 38.3%, and 41.9% (NRI) and by 80.0%, 77.7%, 94.3%, and 81.8% (AO) of patients, respectively; similar trends were observed for ACR50/70 and PASI 75/90/100. Similar to the overall population, the highest proportion of MDA responders was observed in the upadacitinib 15 mg QD arm in both the NRI (30.4%) and AO (46.2%) analyses. BASDAI 50 response rates at 152 weeks in this subgroup were 28.6%, 17.9%, 21.2%, and 25.8% (NRI) and 58.3%, 34.4%, 61.5%, and 50.0% (AO), respectively.

### Safety

Overall exposure to upadacitinib in the study was 649.0 and 600.3 PY with upadacitinib 15 and 30 mg QD, respectively. The overall EAER for any TEAE was higher for upadacitinib 30 mg QD (295





**Fig. 6.** Treatment-emergent adverse events in patients treated with UPA 15 or 30 mg.

\*AEs with a reasonable possibility of being related to the study drug, in the opinion of the investigator. †Excluding tuberculosis and herpes zoster.

‡Six cases of basal cell carcinoma and three cases of squamous cell carcinoma in the UPA 15 mg QD group (including one patient who had one event of basal cell carcinoma and two events of squamous cell carcinoma); four cases of basal cell carcinoma and four cases of squamous cell carcinoma in the UPA 30 mg QD group (including one patient who had two events of basal cell carcinoma and three events of squamous cell carcinoma).

§Two cases of prostate cancer and one case each of malignant melanoma (stage III), malignant neoplasm (not specified), ovarian cancer, and rectal cancer in the UPA 15 mg QD group; one case each of basosquamous carcinoma, endometrial cancer, intraductal papillary breast neoplasm, malignant melanoma, oropharyngeal squamous cell carcinoma, ovarian cancer, and rectal adenocarcinoma in the UPA 30 mg group (including one patient with both ovarian and endometrial cancer); one case of prostate cancer in a patient who switched from UPA 30 mg QD to 15 mg QD.

¶Two cases of abnormal lymphocyte morphology in the UPA 15 mg QD group are shown as this preferred term is included in the Malignant Lymphoma Standardised MedDRA Queries. These non-SAEs were based on single laboratory results and were not confirmed to be lymphomas.

‡‡Defined as cardiovascular death, non-fatal myocardial infarction, and non-fatal stroke. ¶¶Includes deep vein thrombosis and pulmonary embolism.

AE: adverse event; CI: confidence interval; CPK: creatine phosphokinase; E: events; EAER: exposure-adjusted event rate; GI: gastrointestinal; MACE: major adverse cardiovascular events; MedDRA: Medical Dictionary for Regulatory Activities; NMSC: non-melanoma skin cancer; PY: patient-years; QD: once daily; SAE: serious adverse event; UPA: upadacitinib; VTE: venous thromboembolic events.

events/100 PY) than for the approved 15 mg QD dosage (226 events/100 PY; Fig. 6). Rates of serious AEs (SAEs), AEs leading to discontinuation, AEs

considered by the investigator to be related to the study drug, and several AEs of special interest (including creatine phosphokinase [CPK] elevation,

anaemia, and hepatic disorder) were also higher with 30 versus 15 mg QD (Fig. 6). During the 152-week study period, a total of six deaths were reported,

all of which were treatment-emergent and four occurred after week 56. Four of the deaths were in patients receiving upadacitinib 15 mg QD, with one case each of unspecified cancer, COVID-19, myocardial infarction, and death due to unknown cause. Of the two deaths in patients receiving upadacitinib 30 mg QD, one patient's causes were reported as acute respiratory distress syndrome and right pneumothorax per the death certificate, and as pancytopenia per investigator (which occurred during the 56-week double-blind period (10)); the second patient's cause of death was reported as COVID-19 pneumonia.

The most frequently reported AEs were infections, including urinary tract infection, nasopharyngitis, and upper respiratory tract infection (Suppl. Table S2). EAERs of serious infections were 2.8 and 5.8 events/100 PY in the upadacitinib 15 and 30 mg QD groups, respectively (Fig. 6), with the most frequently occurring serious infections being pneumonia (0.6 and 1.0 events/100 PY, respectively), COVID-19 (0.8 and 0 events/100 PY, respectively), and COVID-19 pneumonia (0 and 0.7 events/100 PY, respectively). Herpes zoster was more frequent with upadacitinib 30 *versus* 15 mg QD (7.3 and 3.5 events/100 PY, respectively), while opportunistic infection rates were similar between dose groups (0.6 and 0.7 events/100 PY, respectively; Fig. 6).

In regard to malignancy, 15 events were reported in the upadacitinib 15 mg QD group (EAER, 2.3/100 PY), of which eight events occurred across three patients, and two cases were of abnormal lymphocyte morphology that resolved by the next visit and were not confirmed as lymphomas. Likewise, 15 malignancy events were reported in the upadacitinib 30 mg QD group (2.5/100 PY; Fig. 6). Additional details on the malignancy events are provided in the supplementary material. For adjudicated MACE, two (0.3/100 PY) and three (0.5/100 PY) events were reported in the upadacitinib 15 and 30 mg QD groups, respectively, and there was one (0.2/100 PY) event of adjudicated VTE in each group (Fig. 6). In patients who initially received 30 mg QD but were switched to 15 mg QD, the overall exposure to upadacitinib in

the study was 46.2 PY, and the overall EAER for TEAEs was 155.8 events/100 PY. No cases of herpes zoster, opportunistic infection, or adjudicated MACE or VTE were reported in this subgroup (Suppl. Table S3).

In the TNFi-IR subgroup of patients, the overall exposure to upadacitinib was 462.2 and 473.4 PY in the upadacitinib 15 and 30 mg QD groups, respectively. The safety profile of upadacitinib in the TNFi-IR subgroup was similar to that in the overall population, with higher EAERs for AEs, SAEs, study drug-related AEs, and infections with upadacitinib 30 *versus* 15 mg QD (Suppl. Fig. S9). Event rates of malignancies, and adjudicated MACE and VTE, were similar between the two doses.

### Discussion

Results from the SELECT-PsA 2 study demonstrate the safety and efficacy of upadacitinib 15 and 30 mg QD through 152 weeks in bDMARD-IR patients with PsA. Of note, this study included a highly treatment-refractory population: 92% and 31% of patients had an inadequate response to  $\geq 1$  and  $\geq 2$  prior bDMARDs (including TNFi and/or IL-17 inhibitors), respectively, and more patients had high TJC68/SJC66 scores and enthesitis/dactylitis at baseline compared with previous studies (4, 5). Improvements previously seen across efficacy endpoints assessing musculoskeletal (including axial) and skin outcomes, PROs, and disease control (MDA) at week 56 (10) were maintained through week 152, particularly in the more stringent outcomes, with no major differences between the 15 and 30 mg QD dosages. Importantly, MDA was achieved by up to 44.4% of patients (AO data). In addition, the safety profile of upadacitinib over 152 weeks was similar to that seen in the first 56 weeks, with no new safety signals. As reported previously (10), rates of known JAKi-related AEs, including serious infections, herpes zoster, and CPK elevations, were higher with upadacitinib 30 *versus* 15 mg QD, while rates of malignancies, adjudicated MACE, and VTE were similar between the two dosages. Notably, the efficacy and safety profiles of upadaci-

tinib in patients with a prior inadequate response to  $\geq 1$  TNFi were similar to those in the overall population.

The long-term safety data reported here are consistent with those presented in the recent integrated safety analysis of the SELECT-PsA 1 and 2 studies comparing pooled data from patients treated with upadacitinib 15 mg QD, upadacitinib 30 mg QD, or adalimumab 40 mg every other week (12). While SELECT-PsA 2 enrolled bDMARD-IR patients, those in the SELECT-PsA 1 study had an inadequate response to non-biologic DMARDs. Results from the integrated analysis showed that upper respiratory tract infection, nasopharyngitis, and CPK elevations were the most common AEs with upadacitinib, while rates of malignancies, MACE, VTE, and deaths were similar across treatment groups. Similar to the present analysis, serious infection, anaemia, and CPK elevations were more frequent with upadacitinib 30 *versus* 15 mg QD (12).

Safety data from the current study are consistent with the known safety profile of upadacitinib across immunologic conditions (9, 13). For example, rates and types of AEs in the current analysis were generally similar to those observed through 5 years in the SELECT-BEYOND study of upadacitinib in bDMARD-IR patients with RA (14), and through 2 years in the SELECT-AXIS 1 study of upadacitinib in bDMARD-naïve patients with AS (15). As in the current study, rates of SAEs, AEs leading to discontinuation, serious infections, herpes zoster, and CPK elevation were higher with upadacitinib 30 *versus* 15 mg QD in SELECT-BEYOND (14). Of note, most cases of herpes zoster were non-serious, similar to previously reported data in patients with chronic inflammatory conditions (16).

The maintenance of clinical and functional improvements observed through 3 years in the current study is consistent with that previously reported through 2 years for this patient population (17). Long-term efficacy of upadacitinib has likewise been demonstrated through 2 years in non-bDMARD-IR patients with PsA (SELECT-PsA 1; (18)), and through 5 years in bDMARD-IR patients with RA (SELECT-BEYOND

(14)), as well as in other immunologic indications. Of note, although radiographic and MRI assessments were not performed in this study, improvements were observed in BASDAI and ASDAS outcomes in patients with psoriatic spondylitis at baseline.

One of the main limitations of this study, as with all studies incorporating OLEs, is the lack of a placebo arm after the initial placebo-controlled phase. In addition, caution is advised regarding the generalisability of the aforementioned findings to populations that may have been underrepresented in the present study (e.g. black or African American patients comprised 2.7% of our study population yet comprise a higher percentage in the real world (19)), which is part of a larger issue regarding frequent underrepresentation of minorities in clinical trials (20).

### Conclusions

In summary, long-term data from the SELECT-PsA 2 study show that the efficacy of upadacitinib observed after 56 weeks was maintained through 152 weeks in a treatment-refractory bDMARD-IR population of patients with PsA. The safety profile of upadacitinib 15 mg QD observed through 152 weeks was consistent with its known safety profile across indications. These 3-year results from SELECT-PsA 2 help to address the need for long-term data in patients with PsA and support the favourable benefit-risk profile of upadacitinib in these patients.

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