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Clinical Trial

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Durability of response to icotrokinra in adults with moderate-to-severe plaque psoriasis: 1-year results from the phase III, placebo- and active comparator-controlled ICONIC-ADVANCE 1 and ICONIC-ADVANCE 2 trials

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Abstract

Background **Icotrokinra, a targeted oral peptide, precisely blocks the interleukin-23 receptor. In two phase III moderate-to-severe plaque psoriasis trials, ICONIC-ADVANCE 1 (NCT06143878) and ICONIC-ADVANCE 2 (NCT06220604), icotrokinra provided significantly higher skin clearance rates vs. placebo at week 16 and deucravacitinib at weeks 16 and 24.**

Objectives **To evaluate the clinician- and patient-reported outcomes (PROs) for participants who received icotrokinra in the ICONIC-ADVANCE 1 and ADVANCE 2 phase III clinical trials.**

Methods **ICONIC-ADVANCE 1 and 2 randomized (2 : 1 : 2 and 4 : 1 : 4, respectively) adults with moderate-to-severe plaque psoriasis to icotrokinra 200 mg, placebo (transition to icotrokinra at week 16) or deucravacitinib 6 mg once daily (transition to icotrokinra at week 24). Outcomes assessed through week 52 included proportions of participants achieving Investigator Global Assessment (IGA) 0/1 (with ≥ 2-grade improvement from baseline), Psoriasis Area and Severity Index (PASI) 90, IGA 0, PASI 100, scalp-specific IGA (ss-IGA) 0/1, several well-established PROs, and adverse events (AEs).**

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Results **ICONIC-ADVANCE 1 and 2 randomized 1505 participants (774 and 731, respectively). Skin clearance rates among participants randomized to icotrokinra increased through week 24 and were durable through week 52, with approximately 70–75% exhibiting clear/almost clear skin (IGA 0/1, PASI 90) and approximately 50% demonstrating completely clear skin (IGA 0, PASI 100) during weeks 24–52. Among participants achieving clear/almost clear skin at week 16, around 85–90% maintained response at week 52. Response rates for ss-IGA 0/1 and PROs were also durable through week 52. Participants who transitioned from placebo or deucravacitinib to icotrokinra exhibited increasing response rates that were comparable to participants randomized to icotrokinra through week 52. The AE profile was similar to placebo through week 16 and showed lower AE rates than deucravacitinib through week 24; no safety signals were observed through week 52.**

Conclusions **Once-daily icotrokinra provided robust, durable skin clearance and symptom improvement, with no safety signals, through week 52 in adults with moderate-to-severe plaque psoriasis. Participants transitioning from placebo or deucravacitinib to icotrokinra achieved similar increased response rates to participants who were randomized to icotrokinra through week 52. Our findings support icotrokinra as a systemic therapy with long-term robust disease control and a favourable safety profile.**

Lay summary

Psoriasis is an inflammatory skin condition, which causes thick, scaly skin plaques that may itch or hurt. Psoriasis is caused by high levels of certain immune system signalling molecules. One such molecule is interleukin (IL)-23. Injected medicines that block IL-23 help to treat moderate-to-severe plaque psoriasis.

Icotrokinra is a tablet taken by mouth that precisely blocks the IL-23 receptor. This approach aims to provide the benefits of highly efficacious IL-23-targeted therapy in a convenient form. ICONIC-ADVANCE 1 and 2 are two global clinical trials that studied icotrokinra. Adults with moderate-to-severe plaque psoriasis received icotrokinra, a placebo (for 16 weeks) or deucravacitinib (for 24 weeks). After 16 or 24 weeks, all trial participants received icotrokinra throughout 52 weeks. More participants receiving icotrokinra from the start achieved clear or almost clear skin compared with participants who received placebo at 16 weeks, or participants who received deucravacitinib at 16 and 24 weeks. The skin clearance rates remained high throughout 52 weeks. Many participants who received icotrokinra reported better quality of life. They also reported improvements in their psoriasis symptoms, including itch. By week 52, participants who changed from placebo or deucravacitinib to icotrokinra showed similar improvements to those who received icotrokinra from the beginning. The rates of side-effects were similar for participants who received icotrokinra and placebo at week 16. These participants reported fewer side-effects than those treated with deucravacitinib at week 24. No safety concerns with icotrokinra arose through 52 weeks.

Icotrokinra may offer a convenient treatment option for people with moderate-to-severe plaque psoriasis. It has the advantage of being provided in a once-daily pill, with a favourable safety profile.

What is already known about this topic?

- Subcutaneous interleukin (IL)-23-targeting biologics provide high skin clearance rates in moderate-to-severe plaque psoriasis.
- Icotrokinra, a targeted oral peptide that precisely blocks the IL-23 receptor and inhibits IL-23 pathway signalling, demonstrated significantly higher psoriasis-related response rates vs. placebo at week 16 and deucravacitinib at weeks 16 and 24 in phase III trials.
- Icotrokinra is approved in the USA for adults and paediatric patients (≥ 12 years, ≥ 40 kg) with moderate-to-severe plaque psoriasis.

What does this study add?

- Through 52 weeks, once-daily icotrokinra provided durable psoriatic skin clearance and improvements in patient-reported symptoms, including itch, and health-related quality of life.
- By week 52, participants who transitioned to icotrokinra from placebo or deucravacitinib at week 16 or 24, respectively, exhibited psoriasis-related response rates comparable to those achieved with continuous icotrokinra treatment.
- Icotrokinra exhibited a favourable safety profile, consistent with previous studies, with no safety signals through week 52.

Psoriasis is a chronic, immune-mediated inflammatory disease predominantly affecting the skin.[1] Interleukin (IL)-23 is a key regulator of the pathogenic T-cell activation underlying psoriasis.[2] Biologic therapies targeting the IL-23/T helper (Th)17 axis have demonstrated substantial, durable clinical benefits across multiple randomized trials, with high rates of near-complete and complete skin clearance.[2–6] However, these and other biologic therapies require subcutaneous or intravenous administration, which may be undesirable for many individuals.[7] Other systemic psoriasis therapies, including the tyrosine kinase (TYK)2 inhibitor deucravacitinib and the phosphodiesterase-4 inhibitor apremilast, offer oral dosing but have more modest efficacy than biologics and can be associated with untoward adverse events (AEs).[8,9]

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Icotrokinra is the first and only targeted oral peptide that precisely blocks the IL-23 receptor (IL-23R) and inhibits IL-23 pathway signalling and downstream cytokine production.[10,11] Icotrokinra is approved by the US Food and Drug Administration for the treatment of moderate-to-severe plaque psoriasis in adults and paediatric patients (≥ 12 years, ≥ 40 kg).[12] In phase III studies, icotrokinra provided significantly higher skin clearance rates than placebo and a safety profile similar to placebo in adults and adolescents with moderate-to-severe plaque psoriasis (ICONIC-LEAD; NCT06095115)[13] and psoriasis involving high-impact sites (ICONIC-TOTAL; NCT06095102).[14]

In the phase III ICONIC-ADVANCE 1 (NCT06143878; ADVANCE 1) and ICONIC-ADVANCE 2 (NCT06220604; ADVANCE 2) studies, adults with moderate-to-severe plaque psoriasis achieved superior responses with icotrokinra vs. placebo and deucravacitinib for all coprimary and key secondary endpoints through weeks 16 and 24, with AE rates similar to placebo and lower than deucravacitinib.[15] Here, we report clinician-reported, patient-reported and safety outcomes through week 52 across both studies.

Patients and methods

Study design and oversight

ADVANCE 1 and ADVANCE 2 are global, phase III, randomized, multicentre, double-blind, placebo- and active comparator-controlled trials in adults with moderate-to-severe plaque psoriasis. Study design details have been published.[15] Local or central ethics committees approved each study protocol (File S1; see Supporting Information), the trials were conducted according to the Declaration of Helsinki and Good Clinical Practice, and all participants provided written informed consent.

Participants

Both trials employed the same enrolment criteria. Eligible individuals were adults (≥ 18 years) with plaque psoriasis for ≥ 26 weeks at screening; had body surface area involvement ≥ 10%, Psoriasis Area and Severity Index (PASI) ≥ 12, Investigator Global Assessment (IGA) ≥ 3; and were candidates for phototherapy and/or systemic therapy. Those with nonplaque psoriasis, previous suboptimal response, or AEs related to IL-23-targeting agents or TYK2 inhibitors were excluded. Full eligibility criteria are described in the protocol.[15]

Randomization, masking and treatments

ADVANCE 1 (2 : 1 : 2) and ADVANCE 2 (4 : 1 : 4) randomized participants to once-daily icotrokinra 200 mg, placebo or deucravacitinib 6 mg. The trials included a placebo-controlled period through week 16 and an active comparator-controlled period through week 24; thereafter, all participants received icotrokinra. The ongoing 3-year studies will conclude with final assessments at week 156 and safety follow-up at week 160. Findings through week 52 are reported here.

Endpoints and assessments

Coprimary endpoints in both studies were the proportion of participants achieving IGA 0/1[16] [IGA score of 0 or 1 (clear or minimal) with ≥ 2-grade improvement from baseline] and PASI 90[17] (≥ 90% improvement from baseline) responses at week 16 for icotrokinra vs. placebo.[15] Secondary endpoints, at different timepoints through week 16 vs. placebo and through week 24 vs. deucravacitinib, included the proportion of participants achieving IGA 0/1, PASI 90, IGA 0, PASI 100, scalp-specific IGA (ss-IGA) 0/1[18] [ss-IGA score of 0 or 1 (absent or very mild) with ≥ 2 grade improvement from baseline; among participants with baseline score ≥ 2], clinically meaningful improvement (CMI) in participant-reported itch using Psoriasis Symptoms and Signs Diary (PSSD)[19] (among participants with baseline score ≥ 4), participant-reported PSSD symptom score 0[20,21] (among participants with baseline score > 0) and participant-reported Dermatology Life Quality Index (DLQI) 0/1[22] (among participants with baseline score > 1).[15] Percentage improvement in absolute PASI scores over time, all clinician-reported and patient-reported outcomes assessed after week 24 and all pooled analyses were exploratory. Safety assessments included documentation of pooled AEs and serious AEs from both studies to provide a larger sample size to detect potentially uncommon AEs.

Statistical analyses

Statistical analyses through week 24 have been described previously.[15] As outcomes assessed after week 24 were exploratory, no formal hypothesis testing was performed.

Binary endpoints were in general analysed using Cochran–Mantel–Haenszel tests stratified by baseline weight (≤ 90 vs. > 90 kg) and geographic region (Europe, North and South America, Asia-Pacific). Percentage improvement in PASI was analysed using a mixed-effects model for repeated measures with fixed effects for treatment, visit, treatment-by-visit interaction, geographic region, baseline weight, baseline PASI and baseline PASI-by-visit interaction.

Participants who discontinued owing to lack of efficacy, psoriasis worsening or use of prohibited psoriasis treatment were counted as nonresponders from that point onwards for binary endpoints or had their baseline value carried forward for continuous endpoints (Table S2; see Supporting Information). If participants discontinued treatment for other reasons, observed data were used in the analysis. Remaining missing data were imputed as nonresponse for binary endpoints and not imputed for continuous endpoints.[15]

Efficacy analyses included all randomized participants, with the exception of eight ADVANCE 2 participants who were excluded owing to noncompliance with Good Clinical Practice at a study site. Safety analyses included all randomized participants who received at least one dose of the study drug. AE and exposure-adjusted AE rates [number of participants with AEs/total participant-years (PYs) at risk × 100] were reported.

Results

Participants

ADVANCE 1 and ADVANCE 2 randomized 1505 participants (774 and 731, respectively). Most participants randomized to icotrokinra continued treatment through week 52, with similar overall discontinuation rates across studies [60 of 735 (8%) and 55 of 697 (8%), respectively], primarily owing to participant withdrawal (Figure 1). Discontinuations resulting from AEs were infrequent and comparable across trials (2% each).

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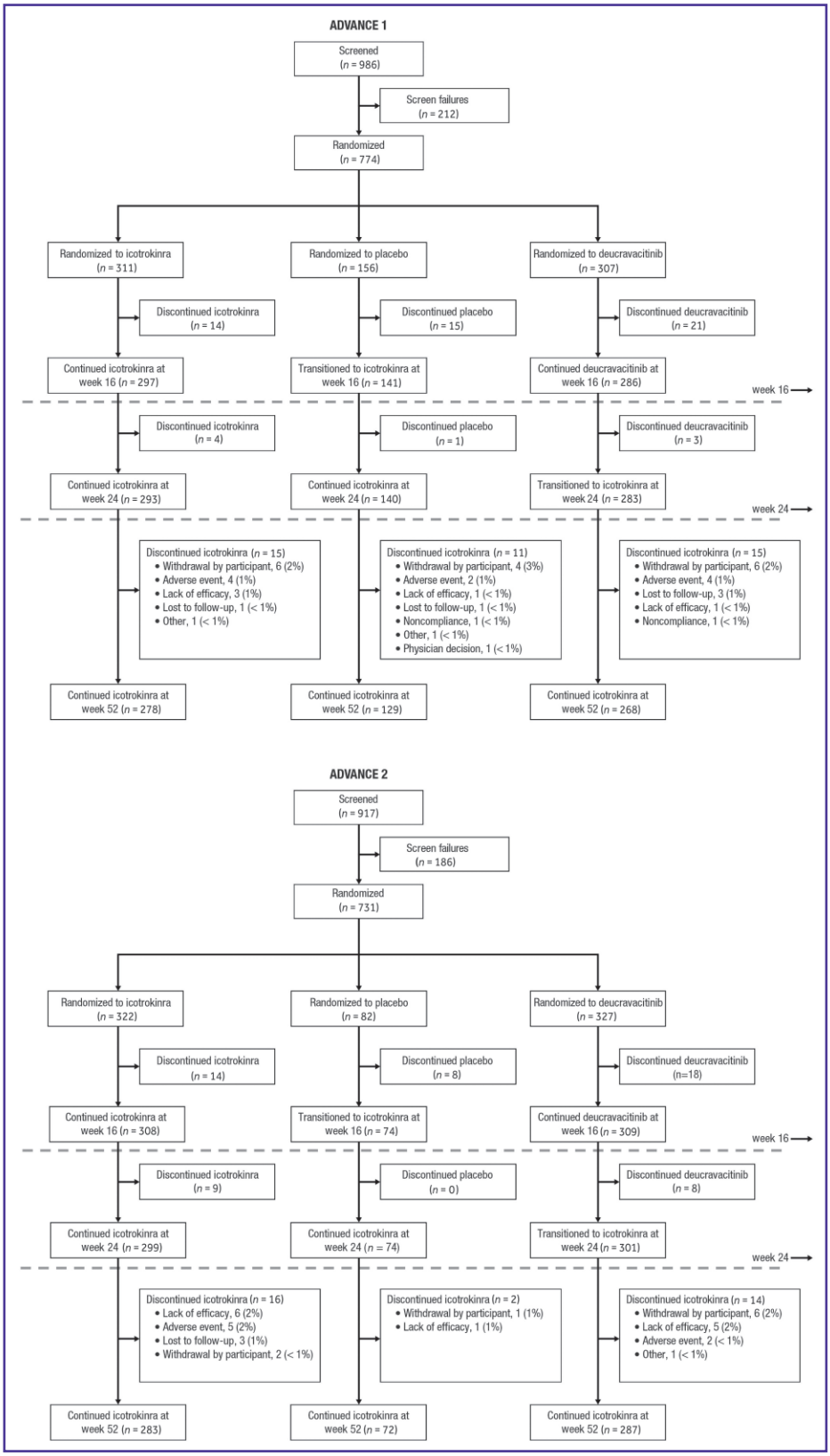


Figure 1 Participant disposition in ADVANCE 1 and ADVANCE 2.

Baseline demographic and clinical characteristics

Baseline characteristics were balanced across treatment groups and consistent across studies.[15] In the pooled population, the mean age was 46.4 years (SD 13.5); most participants were male (68%), White (78%) and not Hispanic-Latinx (84%) (Table S1; see Supporting Information). At baseline, mean psoriasis duration was 17.4 years (SD 12.6), mean PASI was 20.0 (SD 7.1), and IGA-rated psoriasis severity was moderate in 80% of participants and

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severe in 20%. Overall, 72% had received prior systemic therapy and 26% had received prior biologic therapy.

Clinician-reported outcomes

Clear/almost clear skin

At week 16, icotrokinra met both coprimary endpoints in each study. Significantly higher proportions of participants treated with icotrokinra vs. participants treated with placebo achieved IGA 0/1 and PASI 90 (Figure 2a, b).[15] Separation between the icotrokinra and placebo groups was evident by week 8 for PASI 90. Response rates for both IGA 0/1 and PASI 90 continued to increase or were maintained from weeks 16 to 24. Icotrokinra also met all key secondary endpoints vs. deucravacitinib, with significantly higher proportions of participants achieving IGA 0/1 and PASI 90 at weeks 16 and 24.[15]

Among participants randomized to icotrokinra, IGA 0/1 response rates were durable from week 24 [ADVANCE 1: 74% (230 of 311); ADVANCE 2: 69% (220 of 320)] through week 52 [74% (230 of 311); 73% (233 of 320)] (Figure 2a). Findings were consistent for PASI 90 (Figure 2b). Among participants achieving IGA 0/1 and PASI 90 at week 16, responses were maintained at week 52 by 88% (187 of 213) and 90% (154 of 171), respectively, in ADVANCE 1, and 84% (190 of 227) and 87% (160 of 184), respectively, in ADVANCE 2.

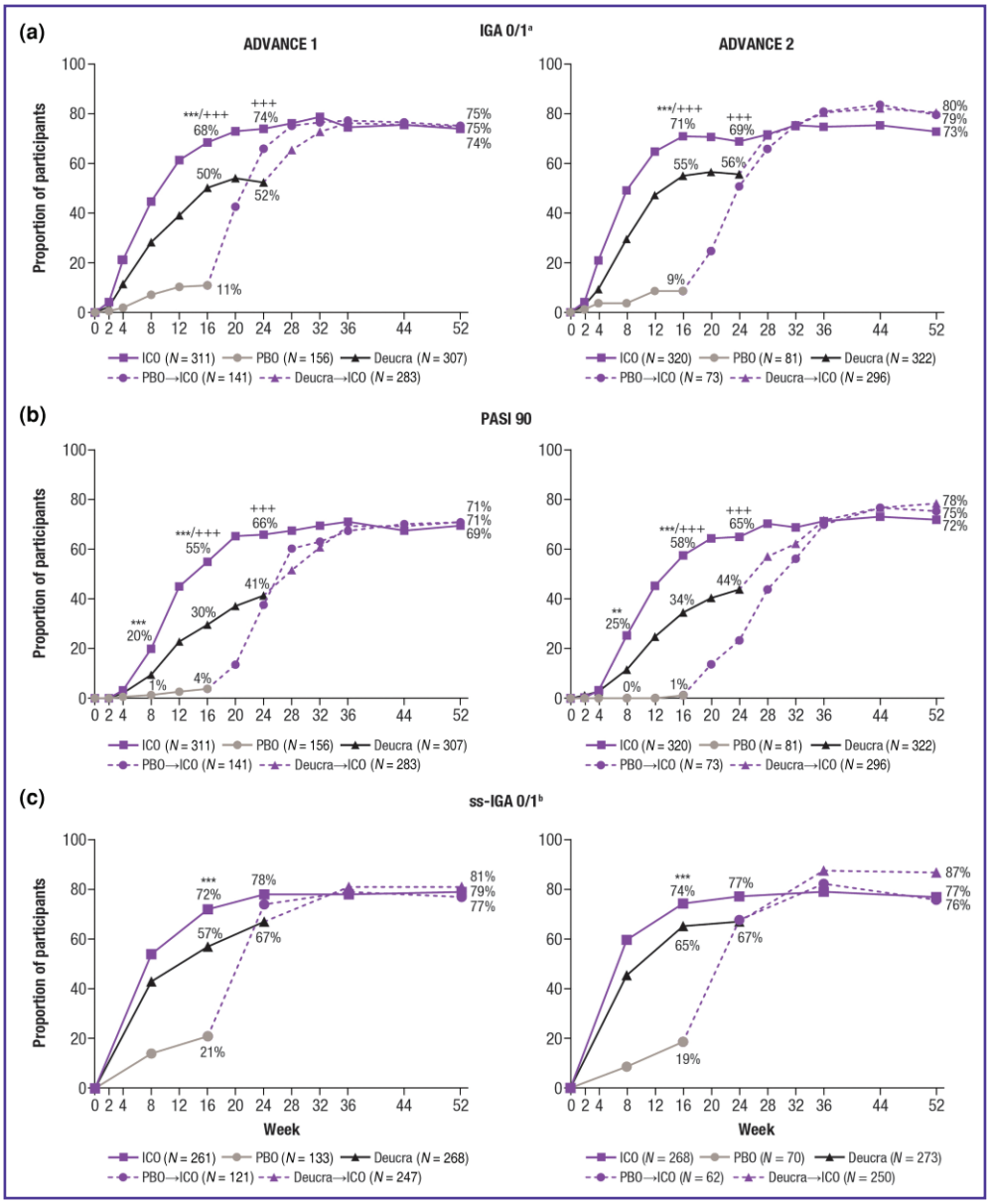


Figure 2 Proportions of participants achieving IGA 0/1[a], PASI 90 and ss-IGA 0/1[b] responses by treatment group through week 52. Deucra, deucravacitinib; ICO, icotrokinra; IGA, Investigator Global Assessment; PASI, Psoriasis Area and Severity Index; PASI 90, ≥ 90% improvement in PASI score from baseline; PBO, placebo; ss-IGA, scalp-specific IGA. ADVANCE 2 enrolled 731 participants, 723 of whom were evaluable for efficacy. Detailed information on the missing data for each outcome is provided in Table S2 (see Supporting Information). Multiplicity-adjusted $^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$ vs. placebo; multiplicity-adjusted $+P < 0.05$, $++P < 0.01$, $+++P < 0.001$ vs. Deucra. Outcomes assessed after week 24 were exploratory; no formal hypothesis testing was performed. [a]Defined as an IGA score of 0 or 1 (clear or minimal) with ≥ 2-grade improvement from baseline. [b]Defined as an ss-IGA score of 0 or 1 (absent or very mild) with ≥ 2 grade improvement from baseline, among participants with baseline score ≥ 2.

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In participants who transitioned from placebo to icotrokinra at week 16 (placebo → icotrokinra) and from deucravacitinib to icotrokinra at week 24 (deucravacitinib → icotrokinra), rates of achieving clear or almost clear skin increased after starting icotrokinra and were similar to those in participants randomized to icotrokinra at week 52. IGA 0/1 and PASI 90 response rates increased by approximately 60–70 percentage points from weeks 16 to 52 in the placebo → icotrokinra groups and by approximately 20–30 percentage points from weeks 24 to 52 in the deucravacitinib → icotrokinra groups.

When pooling ADVANCE 1 and 2 data, 73% (463 of 631) of participants randomized to icotrokinra, 77% (164 of 214) of participants in the placebo → icotrokinra group, and 78% (450 of 579) of participants in the deucravacitinib → icotrokinra group achieved IGA 0/1 at week 52; 71% (446 of 631), 72% (155 of 214) and 75% (433 of 579) achieved PASI 90 at week 52, respectively (Figure S1; see Supporting Information).

Completely clear skin

Significantly higher proportions of participants randomized to icotrokinra vs. participants randomized to placebo achieved complete skin clearance (IGA 0, PASI 100) at week 16, and response rates continued to increase through week 24.[15] Approximately twofold more participants randomized to icotrokinra vs. participants randomized to deucravacitinib achieved completely clear skin at weeks 16 and 24 (Figure 3).

Among participants randomized to icotrokinra, IGA 0 response rates were durable from week 24 [ADVANCE 1: 48% (150 of 311); ADVANCE 2: 40% (128 of 320)] through week 52 [52% (163 of 311); 51% (162 of 320)] (Figure 3a). Findings were consistent for PASI 100 (Figure 3b).

Participants randomized to placebo and those randomized to deucravacitinib in each study had increasing rates of achieving completely clear skin (IGA 0, PASI 100) after starting icotrokinra, approximating those in participants randomized to icotrokinra by week 52. Rates of complete skin clearance increased by approximately 40–50 percentage points from weeks 16 to 52 in the placebo → icotrokinra groups and by approximately 25–35 percentage points from weeks 24 to 52 in the deucravacitinib → icotrokinra groups (Figure 3).

In pooled analyses, 52% (325 of 631) of participants randomized to icotrokinra, 53% (114 of 214) of participants in the placebo → icotrokinra group, and 51% (296 of 579) of participants in the deucravacitinib → icotrokinra group achieved IGA 0 at week 52; 48% (306 of 631), 48% (103 of 214) and 47% (273 of 579) achieved PASI 100, respectively (Figure S1).

Percentage improvement from baseline in Psoriasis Area and Severity Index

In pooled analyses, participants randomized to icotrokinra achieved greater mean percentage improvement in PASI (exploratory endpoint) vs. placebo at week 16 (84% vs. 27%). Both participants who were randomized to icotrokinra and participants in the

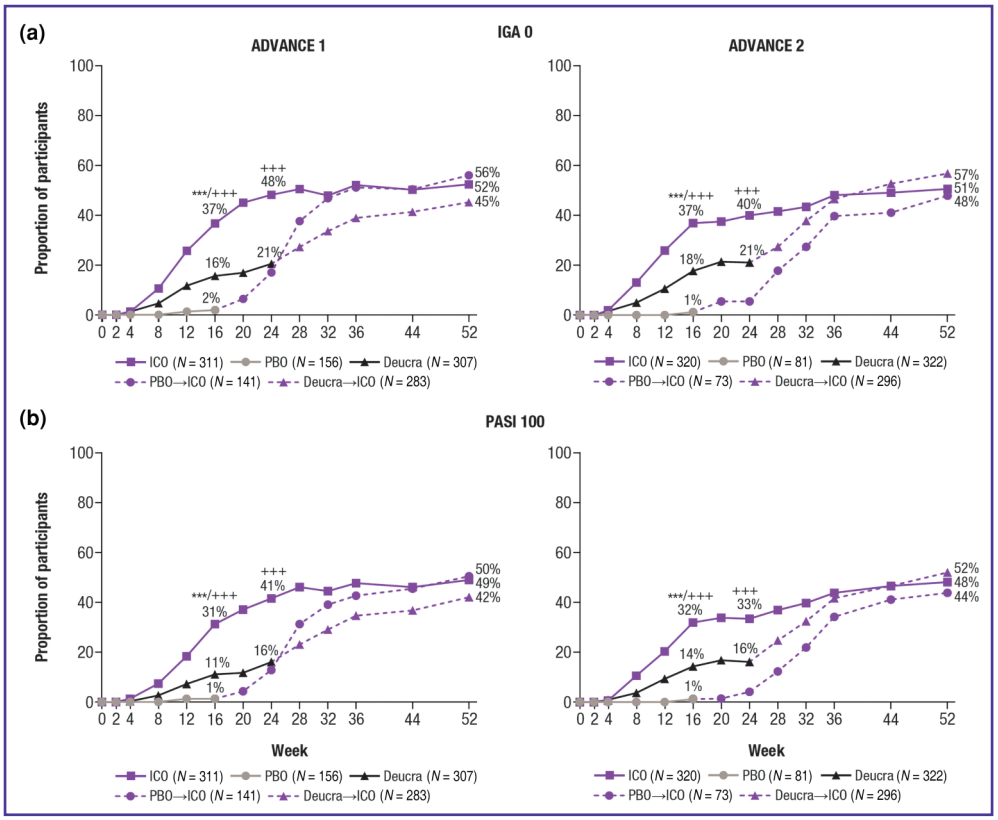


Figure 3 Proportions of participants achieving IGA 0 and PASI 100 responses by treatment group through week 52. Deucra, deucravacitinib; ICO, icotrokinra; IGA, Investigator Global Assessment; PASI, Psoriasis Area and Severity Index; PASI 100, 100% improvement in PASI score from baseline; PBO, placebo. ADVANCE 2 enrolled 731 participants, 723 of whom were evaluable for efficacy. Detailed information on the missing data for each outcome is provided in Table S2 (see Supporting Information). Multiplicity-adjusted $^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$ vs. placebo; multiplicity-adjusted $+P < 0.05$, $++P < 0.01$, $+++P < 0.001$ vs. Deucra. Outcomes assessed after week 24 were exploratory; no formal hypothesis testing was performed.

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placebo → icotrokinra group demonstrated a 91% mean percentage improvement in PASI at week 52 (Figure S2; see Supporting Information).

Scalp-specific psoriasis clearance (in participants with baseline scalp-specific Investigator Global Assessment score ≥ 2)

Greater proportions of participants randomized to icotrokinra vs. placebo achieved absent or very mild scalp psoriasis (ss-IGA 0/1) at week 16 (Figure 2c).[15] Icotrokinra response separated from placebo as early as week 8, with increased response rates through week 24.

Icotrokinra treatment provided durable ss-IGA 0/1 response rates from week 24 [ADVANCE 1: 78% (203 of 261); ADVANCE 2: 77% (207 of 268)] to week 52 [79% (206 of 261); 77% (206 of 268)] (Figure 2c). In each study, the proportions of participants achieving absent or very mild scalp psoriasis after starting icotrokinra at week 16 or 24 increased by approximately 60 percentage points in the placebo → icotrokinra groups and by around 20 percentage points in the deucravacitinib → icotrokinra groups, which approximated the response rates of participants randomized to icotrokinra by week 52.

Patient-reported outcomes

Through week 16 or 24, higher proportions of participants randomized to icotrokinra vs. placebo or deucravacitinib, respectively, achieved CMI in PSSD itch, resolution of psoriasis symptoms, and no impact of psoriasis on their health-related quality of life (HRQoL) (Figure 4).[15]

Clinically meaningful improvement in Psoriasis Symptoms and Signs Diary itch (in participants with baseline score ≥ 4)

The proportions of participants randomized to icotrokinra reporting CMI in PSSD itch continued to increase through week 24 [ADVANCE 1: 63% (157 of 251); ADVANCE 2: 61% (156 of 256)] and were durable through week 52 [73% (184 of 251); 75% (193 of 256)] (Figure 4a). Within each study, the proportion of participants reporting CMI in PSSD itch after starting icotrokinra at week 16 or 24 increased by approximately 60 percentage points in the placebo → icotrokinra groups and by around 20–30 percentage points in the deucravacitinib → icotrokinra groups, which approximated the response rates among participants randomized to icotrokinra by week 52.

In pooled analyses, 74% (377 of 507) of participants randomized to icotrokinra, 76% (122 of 160) of participants in the placebo → icotrokinra group, and 83% (364 of 441) of participants in the deucravacitinib → icotrokinra group reported CMI in PSSD itch at week 52 (Figure S1).

Psoriasis Symptoms and Signs Diary symptom score 0 (in participants with baseline score > 0)

The proportions of participants randomized to icotrokinra reporting complete resolution of psoriasis symptoms continued to increase from week 24 [ADVANCE 1: 29% (82 of 286); ADVANCE 2: 22% (64 of 296)] to week 52 [45% (115 of 256); 39% (88 of 227)] (Figure 4b). Within each study, the proportion of participants reporting a PSSD symptom score of 0 after starting icotrokinra at week 16 or 24 increased by approximately 30–40 percentage points in the placebo → icotrokinra groups and by around 20–30 percentage points in the deucravacitinib → icotrokinra groups, which approximated the response rates among participants randomized to icotrokinra by week 52.

In pooled analyses, 42% (203 of 483) of participants randomized to icotrokinra, 41% (70 of 170) of participants in the placebo → icotrokinra group, and 42% (181 of 430) of participants in the deucravacitinib → icotrokinra group reported a PSSD symptom score of 0 at week 52 (Figure S1).

Dermatology Life Quality Index 0/1 (in participants with baseline score > 1)

The proportions of participants randomized to icotrokinra reporting no impact of psoriasis on their HRQoL was durable from week 24 [ADVANCE 1: 66% (200 of 304); ADVANCE 2: 59% (187 of 317)] to week 52 [70% (212 of 304); 64% (204 of 317)] (Figure 4c). Within each study, the proportion of participants reporting DLQI 0/1 after starting icotrokinra at week 16 or 24 increased by approximately 55–60 percentage points in the placebo → icotrokinra groups and by around 20–25 percentage points in the deucravacitinib → icotrokinra groups, approximating the response rates among participants randomized to icotrokinra by week 52.

Safety

Across both trials, 1431 participants received at least one dose of icotrokinra, with 1057 PYs of follow-up (Table 1). Safety analyses included 632 participants randomized to icotrokinra, 215 participants in the placebo → icotrokinra group and 584 participants in the deucravacitinib → icotrokinra group through week 52.

Safety findings through week 24 have been previously reported.[15] In pooled analyses through week 16, the exposure-adjusted AE and infection rates were lower with icotrokinra than placebo (230 vs. 316; 87 vs. 130 per 100 PYs, respectively; Table 1). Through week 24, the exposure-adjusted AE and infection rates were also lower with icotrokinra than with deucravacitinib (207 vs. 269; 82 vs. 119 per 100 PYs, respectively).

Through week 52, exposure-adjusted AE rates were consistent across the group randomized to icotrokinra, and the placebo → icotrokinra and deucravacitinib → icotrokinra groups (167, 186 and 165 per 100 PYs, respectively) (Table 1). The most common AEs (≥ 5% in any group) from week 0 through week 16 or 24[15] remained the most frequent through week 52 (nasopharyngitis 12–17%, upper respiratory tract infection 7–12%, headache 2–6%), with the addition of influenza (3–7%). Exposure-adjusted serious AE rates through week 52 were 5–6 per 100 PYs, serious infections ≤ 1 per 100 PYs, and AEs leading to treatment discontinuation 1–4 per 100 PYs across all participants treated with icotrokinra.

No cases of active tuberculosis or opportunistic infection occurred through week 52. Rates of gastrointestinal AEs were similar between the icotrokinra and placebo groups through week 16; rates did not increase through week 52 with icotrokinra. No clinically meaningful laboratory changes were observed. Three deaths occurred through week 52; one (< 1%) in the placebo → icotrokinra group (toxicity to various agents) and two (< 1%) in the icotrokinra group (haematemesis, myocardial infarction; narrative details are provided in File S1).

Discussion

Icotrokinra, a targeted oral peptide that precisely blocks the IL-23R and inhibits IL-23 pathway signalling, provided durable

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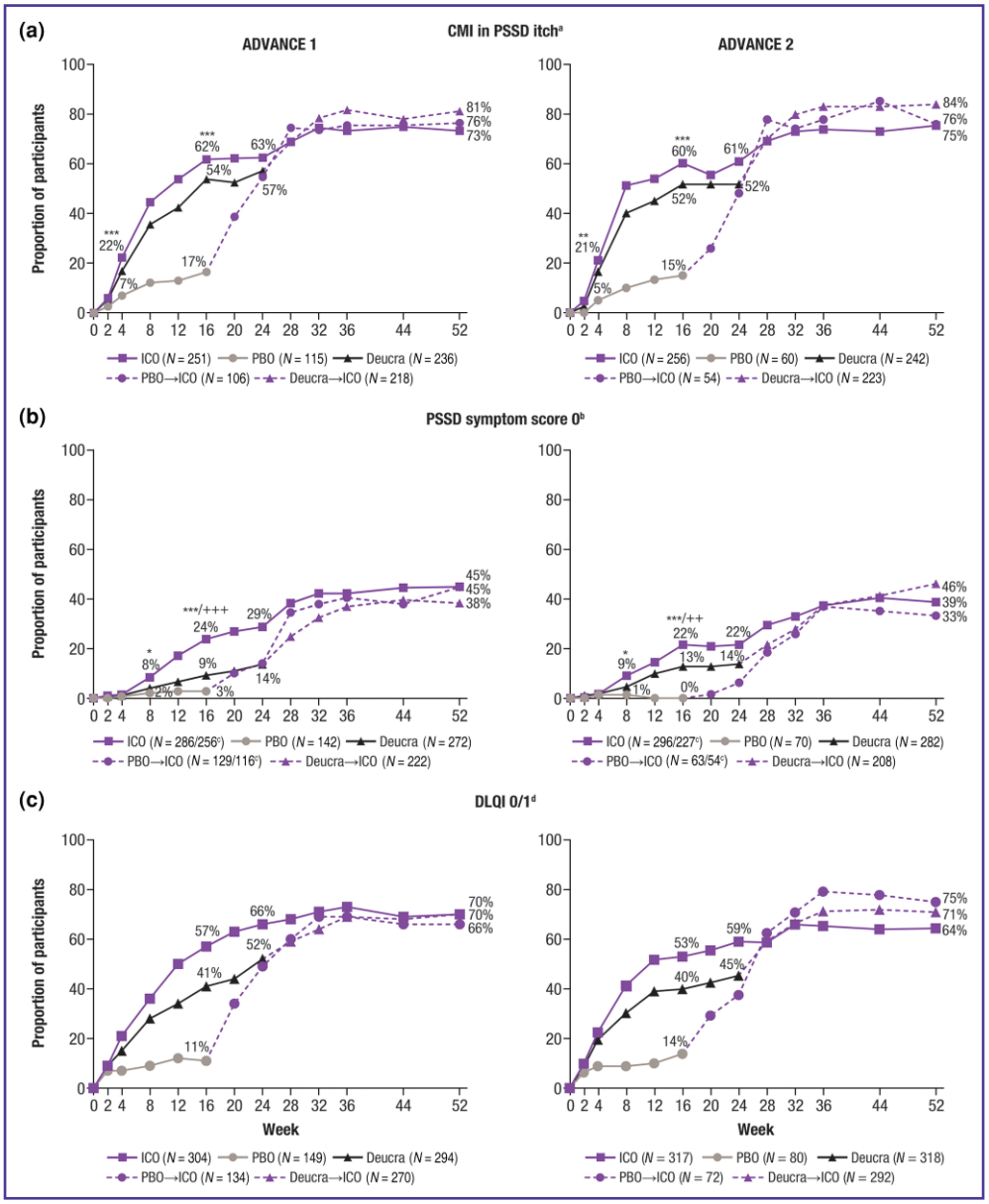


Figure 4 Patient-reported outcomes by treatment group through week 52. CMI, clinically meaningful improvement (≥ 4-point improvement from baseline); Deucra, deucravacitinib; DLQI, Dermatology Life Quality Index; ICO, icotrokinra; PBO, placebo; PSSD, Psoriasis Symptoms and Signs Diary. ADVANCE 2 enrolled 731 participants, 723 of whom were evaluable for efficacy. Detailed information on the missing data for each outcome is provided in Table S2 (see Supporting Information). Multiplicity-adjusted $*P < 0.05$, $**P < 0.01$, $***P < 0.001$ vs. placebo; multiplicity-adjusted $+P < 0.05$, $++P < 0.01$, $+++P < 0.001$ vs. Deucra. Outcomes assessed after week 24 were exploratory; no formal hypothesis testing was performed. [a]Among participants with a baseline PSSD symptom score > 0. [b]Among participants with a baseline PSSD itch score ≥ 4. [c]*N* after week 24. [d]Among participants with a baseline DLQI score >1.

disease control across multiple clinician-reported and patient-reported outcomes through week 52 in the phase III ADVANCE 1 and 2 trials, which involved adults with moderate-to-severe psoriasis. Among participants randomized to icotrokinra, approximately 70–75% achieved clear or almost clear skin and around 50% achieved completely clear skin during weeks 24 through 52. Approximately 85–90% of those with clear or almost clear skin at week 16 maintained this high-level response through week 52. In pooled analyses, participants randomized to icotrokinra demonstrated a 91% mean percentage improvement in PASI at week 52.

Scalp psoriasis, similar to other high-impact sites (fingernails, hands/feet, genitals), can be difficult to treat.[23] Icotrokinra provided robust and durable clearance of scalp psoriasis (ss-IGA 0/1) through week 52 (76–87% across groups), consistent with ICONIC-TOTAL and ICONIC-LEAD results (66–72% at weeks 16–52).[13,14,24] These rates are within the range of response rates observed for biologics targeting IL-23 and higher than those reported for other oral therapies.[4,25–29]

In parallel with sustained clinical responses, icotrokinra was associated with durable improvements in psoriasis symptoms and HRQoL through week 52. On average at baseline, participants experienced substantial disease impact, with a mean PSSD itch score of 6.4 (score 10 indicates worst itch imaginable), PSSD score of 51.7 (indicating high symptom burden) and DLQI score of 12.5

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Table 1 Overall summary of treatment-emergent AEs through week 16, week 24 and week 52

	Placebo-controlled (through week 16)		Active comparator-controlled (through week 24)		Through week 52		
Combined ADVANCE 1 and ADVANCE 2[a]	**Placebo (*N* = 237)**	**Icotrokinra (*N* = 632)**	**Icotrokinra (*N* = 632)**	**Deucravacitinib (*N* = 634)**	**Placebo → icotrokinra (*N* = 215)[b]**	**Icotrokinra (*N* = 632)**	**Deucravacitinib → icotrokinra (*N* = 584)[c]**
Mean weeks per total PYs of follow-up	15.5/70.4	15.9/192.6	23.6/285.3	23.3/283.0	35.5/146.3	49.5/599.1	27.8/311.3
Any AE	137 (58)	308 (49)	368 (58)	417 (66)	148 (69)	466 (74)	317 (54)
Incidence per 100 PYs (95% CI)	316 (257–364)	230 (205–256)	207 (186–229)	269 (243–295)	186 (168–239)	167 (152–182)	165 (146–183)
Serious AE	4 (2)	14 (2)	18 (3)	20 (3)	8 (4)	31 (5)	17 (3)
Incidence per 100 PYs (95% CI)	6 (< 1–9)	7 (3–11)	6 (3–9)	7 (4–10)	6 (1–9)	5 (3–7)	6 (3–8)
AE leading to discontinuation	12 (5)	14 (2)	17 (3)	19 (3)	2 (1)	24 (4)	6 (1)
Incidence per 100 PYs (95% CI)	17 (7–25)	7 (3–11)	6 (3–9)	7 (4–10)	1 (0–3)	4 (2–6)	2 (< 1–3)
Infection	74 (31)	146 (23)	193 (31)	254 (40)	101 (47)	310 (49)	199 (34)
Incidence per 100 PYs (95% CI)	130 (96–154)	87 (72–100)	82 (70–93)	119 (104–134)	94 (80–121)	75 (66–83)	82 (70–92)
Serious infection	1 (< 1)	1 (< 1)	3 (< 1)	5 (1)	0	6 (1)	4 (1)
Incidence per 100 PYs (95% CI)	1 (0–9)	1 (0–3)	1 (< 1–3)	2 (< 1–4)	0 (0–4)	1 (< 1–2)	1 (< 1–4)
Gastrointestinal AE	14 (6)	45 (7)	55 (9)	82 (13)	15 (7)	83 (13)	37 (6)
Incidence per 100 PYs (95% CI)	21 (11–36)	24 (17–32)	20 (15–26)	31 (25–38)	11 (5–15)	15 (12–18)	12 (8–16)
Malignancy	1 (< 1)	3 (< 1)	3 (< 1)	2 (< 1)	0	4 (1)	2 (< 1)
Incidence per 100 PYs (95% CI)	1 (0–9)	2 (< 1–5)	1 (< 1–3)	1 (< 1–3)	0 (0–4)	1 (< 1–2)	1 (< 1–2)

AE, adverse event; CI, confidence interval; PYs, participant-years. Data are provided as *n* (%) unless otherwise noted. Incidence per 100 PYs: number of participants with AEs/total PYs at risk × 100. CI based on study-size adjusted Wald statistics, except malignancy, which was based on the Wald statistic with continuity correction. [a]Safety analysis set included all randomized and treated participants [ADVANCE 1 and 2: placebo, 155 and 82 (placebo → icotrokinra, 141 and 74); icotrokinra, 310 and 322; deucravacitinib, 307 and 327 (deucravacitinib → icotrokinra, 283 and 301)]. [b]Includes data after week 16 for participants randomized to placebo who transitioned to receive icotrokinra. [c]Includes data after week 24 for participants randomized to deucravacitinib who transitioned to receive icotrokinra.

(indicating very large effect on daily life). By week 24, 61–63% of participants randomized to icotrokinra achieved CMI in itch, 22–29% reported complete psoriasis symptom resolution, and 59–66% indicated no impact of psoriasis on their HRQoL; these improvements continued to increase to 73–75%, 39–45% and 64–70% by week 52, respectively. These findings demonstrate that long-term icotrokinra treatment not only provides durable skin clearance, but also delivers meaningful, sustained benefits in symptoms and HRQoL.

Previously reported ADVANCE 1 and 2 findings through week 24, in which icotrokinra met all coprimary and key secondary endpoints vs. placebo and deucravacitinib,[15] are supported by novel findings through week 52 from participants who transitioned from placebo or deucravacitinib to icotrokinra at weeks 16 or 24, respectively. Participants who transitioned from placebo or deucravacitinib achieved clear or almost clear skin (71–80%), completely clear skin (42–57%), CMI in itch (76–84%), psoriasis symptom resolution (33–46%) and no impact of psoriasis on daily life (66–75%), comparable to those treated continuously with icotrokinra through week 52. This evidence highlights that icotrokinra may provide benefits to patients previously treated with another oral psoriasis therapy.

Safety findings of icotrokinra through week 52 aligned with previously reported phase II/III data, showing a similar AE profile vs. placebo and numerically lower AE and infection rates vs. deucravacitinib.[11,13–15,30,31], Exposure-adjusted AE rates with icotrokinra remained stable over time, and no new safety signals were observed through week 52. Taken together, these results support icotrokinra as a durable and efficacious systemic therapy option with a favourable safety profile for patients with moderate-to-severe plaque psoriasis.

Strengths of these studies include the large sample size, global enrolment, and evaluation of deucravacitinib transition, allowing assessment of both clinician-reported and patient-reported outcomes for icotrokinra following transition from deucravacitinib. One limitation was that only adults were included in these studies; however, results from adolescents enrolled in ICONIC-LEAD and ICONIC-TOTAL provide supportive evidence for this population.[13,14,24,31]

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Missing data were handled using nonresponder imputation, which may underestimate response rates; however, the amount of missing data was low (Table S2). Here, a 52-week assessment period is reported; however, ongoing longer-term extensions across four phase III trials will further characterize the durability of icotrokinra response through 2–3 years, and a head-to-head study (ICONIC-ASCEND; NCT06934226) is under way to compare icotrokinra response with a biologic (ustekinumab).

From a broad therapeutic perspective, these data, together with previous findings,[11,13–15,24,30,31] highlight the importance of durable efficacy and safety with this first and only targeted oral peptide that precisely blocks the IL-23R. Icotrokinra was recently approved for the treatment of moderate-to-severe plaque psoriasis in the USA.[12] IL-23-targeting biologics have set a high bar for sustained skin clearance; results from the ADVANCE studies suggest that precise inhibition of IL-23 pathway signalling with icotrokinra can achieve similarly robust, persistent outcomes with a favourable safety profile, while offering the convenience of a once-daily oral pill.

In conclusion, through 52 weeks of ADVANCE 1 and 2, once-daily icotrokinra demonstrated robust, durable disease control in moderate-to-severe plaque psoriasis, with a high proportion of participants achieving completely clear skin and reporting symptom resolution and no psoriasis impact on HRQoL. Through week 52, participants who transitioned from placebo or deucravacitinib to icotrokinra achieved outcomes comparable to participants randomized to icotrokinra from baseline. Icotrokinra had an AE profile similar to placebo through week 16, lower AE rates than deucravacitinib through week 24 and no safety signals through 52 weeks. Together with prior data,[11,13–15,24,30,31] these findings support the potential of icotrokinra for sustained, comprehensive disease control for a broad psoriasis population with a once-daily pill.

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Author contributions

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Conflicts of interest

L.S.G. served as an investigator, advisor and/or speaker for AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Galderma, Johnson & Johnson, Leo, Pfizer, Regeneron, Sanofi and Takeda. **A.W.A.** served as a speaker, consultant, and/or investigator for AbbVie, Amgen, Arcutis, Bristol Myers Squibb, Dermavant Sciences, Eli Lilly, Galderma, Incyte, Johnson & Johnson, Leo, Novartis, Pfizer, Regeneron, Sanofi, Takeda and UCB. **J.S.** served as a speaker, consultant, advisory board member and/or investigator for AbbVie, Amgen, Arcutis, ASLAN, Bristol Myers Squibb, Coval Biopharma, Dermavant, Eli Lilly, Johnson & Johnson, KoBioLabs, National Psoriasis Foundation, Novartis, Pfizer, Regeneron/Sanofi and UCB. **R.B.V.** received grants/research support and speakers bureau/honoraria from AbbVie, Alumis, Amgen, Arcutis, Bausch Health, Boehringer Ingelheim, Bristol Myers Squibb, Celltrion, Dermavant, Dermira, DICE, Eli Lilly, Galderma, Incyte, JAMP Pharma, Johnson & Johnson, Leo, Meiji, Nimbus Therapeutics, Novartis, Organon, Orka Therapeutics, Pfizer, Sandoz, Sanofi, Sun Pharma, Takeda, UCB and Zai Lab. **Á.G.C.** served as a consultant for and/or received grants/other payments from AbbVie, Almirall, Amgen, Apogee Therapeutics, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, CeraVe, Eli Lilly, Innovaderm Research, Johnson & Johnson, Leo, L'Oréal, Novartis and Organon. **M.H.** served as an advisory board member, consultant, speaker and/or investigator for and/or received honoraria and/or grants from AbbVie, Almirall, Biofrontera, Bristol Myers Squibb, Dermapharm, Dermasence, Forward Pharma, Eli Lilly, Galderma, Incyte, Johnson & Johnson, Kiniksa Pharmaceuticals, Leo, L'Oréal, Merck Sharp & Dohme, Nektar Therapeutics, Novartis, Pierre Fabre, Pfizer, Sanofi, Schering-Plough, Smerud Medical Research, Stallergenes Greer, UCB and Viatris. **S.T.G.** served as an investigator, consultant and/or speaker for AbbVie, Acelyrin, Allergan, Almirall, Alumis, Amgen, Apogee, Arcutis, ASLAN, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, Dermavant, Dermira, Dow Pharmaceutical Sciences, Dr Reddy's Laboratories, Eli Lilly, Galderma, GSK, Idera Pharmaceuticals, Incyte, Johnson & Johnson, Leo, Medicis, Merck, MoonLake, Novartis, Organon, Pfizer, Regeneron, Sanofi, Sun Pharma, Takeda, Timber Pharmaceuticals, UCB and Zura Bio. **H.C.-H.H.** served as a consultant and/or

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investigator and/or speaker for AbbVie, Amgen, Arcutis, ASLAN, Bausch Health, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, Cutanea, Dermavant, Dermira, DS Biopharma, Eli Lilly, Evelo Biosciences, Galderma, GSK, Incyte, Johnson & Johnson, Leo, MedImmune, Merck, Mirimar, Novartis, Organon, Oruka Therapeutics, Pfizer, Regeneron, Roche, Sanofi and UCB. **D.F.M.** served as an investigator and/or advisor for Almirall, Arcutis, Argenx, ASLAN, Bristol Myers Squibb, Eli Lilly, Evelo, Galderma, GSK, Incyte, Johnson & Johnson, Kiniksa, Leo, Nectar, Novartis, Nurix Therapeutics, Inc., Ono Pharmaceutical, Pfizer, Pierre Fabre, Principia, RAPT Therapeutics, Inc., Regeneron, Roche, Sanofi and UCB; was a creator and/or cocreator of Bullous Pemphigoid Disease Area Index, Mucous Membrane Pemphigoid Disease Area Index, Pemphigus Disease Area Index and their definitions, in addition to the Autoimmune Bullous Disease Quality of Life and Treatment of Autoimmune Bullous Disease Quality of Life instruments; was an author of the textbook *Blistering Diseases* and has contributed to UptoDate on the topic of bullous pemphigoid management. **E.J.S.** served as a speaker, consultant, advisory board member and/or investigator for AbbVie, Acelyrin, Alphyn Biologics, Alumis, Amgen, Apogee Therapeutics, Arcutis, ASLAN, Boehringer Ingelheim, Bristol Myers Squibb, Celldex Therapeutics, CorEvitas, Dermavant/Organon, DermBiont, Inc., Eli Lilly, Galderma, Genzyme, Incyte, Johnson & Johnson, Leo, MoonLake, Novartis, Pfizer, Regeneron, Sanofi, Sun Pharma, TARGET PharmaSolutions, Timber Pharmaceuticals, UCB and Zura Bio. **L. Kircik** served as a speaker, consultant, advisory board member and/or clinical study investigator for Abbott Laboratories, AbbVie, Allergan, Almirall, Amgen, Arcutis, Biogen, Boehringer Ingelheim, Breckinridge Pharmaceutical, Bristol Myers Squibb, Celgene, Cellceutix, Centocor Inc., Cipher Pharmaceuticals Inc., Combinatrix, Connetics Corporation, Coria, Dermavant, Dermira, Dow Pharmaceutical Sciences Inc., Dr. Reddy's Laboratories, Eli Lilly, Galderma, Genentech, GSK, Idera Pharmaceuticals, Johnson & Johnson, Leo, Maruho, Medicis Pharmaceutical Corp, Merck, Nimbus Therapeutics, Novartis, Pfizer, PharmaDerm, Promius Pharma, Serono, Stiefel Laboratories Inc., Sun Pharma, Taro Pharmaceutical, UCB, Valeant Pharmaceuticals Intl, Ventyx Biosciences and XenoPort. **M.A.** served as a consultant, speaker and/or investigator for AbbVie, Almirall, Amgen, Biogen, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, Centocor Inc., Eli Lilly, Galderma, Hexal, Incyte, Johnson & Johnson, Klinge, Leo, medac Pharma, Merck Sharp & Dohme, Mylan, Novartis, Pfizer, Sandoz, Takeda, UCB and Viatris. **M.S.** served as an investigator and/or advisor and/or speaker for AbbVie, Alumis, Amgen, Arcutis, Bausch Health, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Leo, Novartis, Sun Pharma, Takeda and UCB. **S.G.** served as an advisor and/or received speaker's honoraria and/or received grants and/or participated in clinical trials for AbbVie, Acelyrin, Adimmune, Affibody Medical AB, Akari Therapeutics, Almirall Hermal GmbH, Alumis, Amgen, Apogee Therapeutics, Argenx BV, Aristea Therapeutics, AstraZeneca, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, Celltrion, Dermira, Eli Lilly, Galderma, Hexal AG, Incyte, Johnson & Johnson, Klinge, Kymab, Leo, Medac Pharma, MoonLake, Neubourg Skin Care GmbH, Novartis, Oruka Therapeutics, Pfizer, Pierre Fabre, Principia, Regeneron, Sandoz, Sanofi, Santis Pharma and UCB. **O.R.-S., B.E.E., J.C., K.C., L. Kephart, S.L.** and **Y.-W.Y.** are employees of Johnson & Johnson and may own stock/stock options in Johnson & Johnson. **R.B.** served as an advisory board member, consultant, speaker and/or investigator for and received honoraria and/or grants from AbbVie, Alumis, Amgen, AnaptysBio, Inc., Arcutis, Bristol Myers Squibb/Celgene, Dermavant, Eli Lilly, Johnson & Johnson, Leo, Nimbus Therapeutics, Oruka Therapeutics, Takeda, UCB, Ventyx Biosciences, Vyne Therapeutics, Xencor, Zai Lab and Zura Bio; and is an employee and shareholder of Innovaderm Research.

Data availability

The individual participant data collected for the study will not be made available. The data sharing policy of Johnson & Johnson is available at https://innovativemedicine.jnj.com/our-innovation/clinical-trials/transparency. As noted on this site, requests for access to the study data can be submitted through the Yale Open Data Access Project site at https://yoda.yale.edu.

Ethics statement

Independent ethics committees or institutional review boards (list of institutional review boards and ethics committees provided in File S1; see Supporting Information) approved the study protocol, informed consent form(s) and recruitment materials before patient enrolment. The studies were conducted in compliance with Good Clinical Practice, the International Council for Harmonisation Guideline E6, the Declaration of Helsinki and applicable regulatory requirements.

Patient consent

Not applicable.

Supporting information

Additional Supporting Information may be found in the online version of this article at the publisher's website.

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