

Early and Sustained Efficacy of Depemokimab in Type 2 Asthma: A Pooled Analysis of the SWIFT-1/-2 Studies



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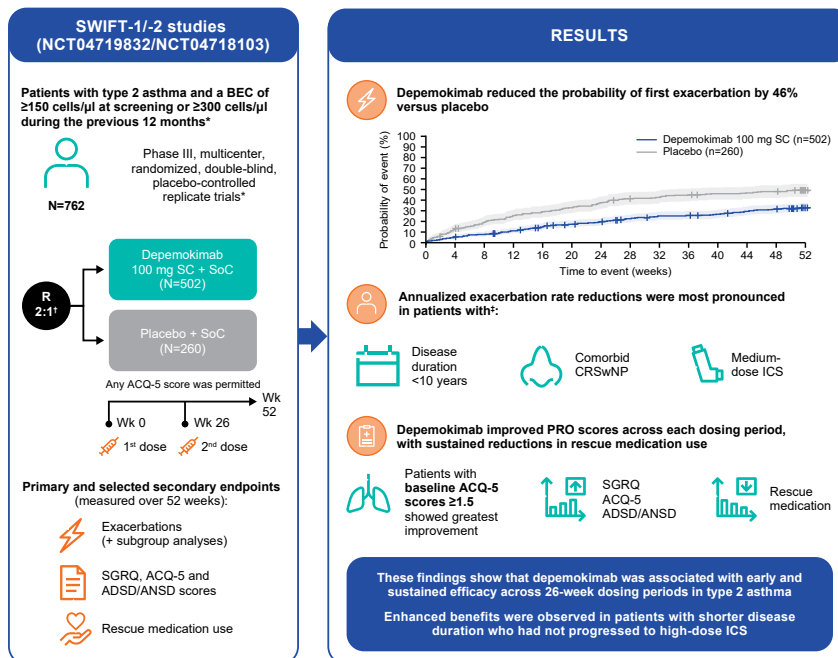
What is already known about this topic? In the phase III SWIFT-1/-2 trials, twice-yearly depemokimab significantly reduced annualized exacerbation rates by 54% versus placebo, and improved patient-reported outcomes over 52 weeks in patients with type 2 asthma.

What does this article add to our knowledge? Rapid improvements in exacerbation risk and health-related quality of life with depemokimab were each sustained across 26-week intervals, particularly in patients with shorter disease duration, those receiving medium-dose ICS, or those with comorbid chronic rhinosinusitis with nasal polyps at baseline.

How does this study impact current management guidelines? These findings suggest that initiation of twice-yearly depemokimab in patients with shorter disease duration may offer early and sustained efficacy with reduced dosing frequency, potentially reducing treatment burden and informing future asthma management guideline updates.

VISUAL SUMMARY

Early and Sustained Efficacy of Depemokimab in Type 2 Asthma: A Pooled Analysis of the SWIFT-1/-2 Studies Wechsler et al, *J Allergy Clin Immunol: In Pract* (2026)



*Eligible patients were ≥ 12 years of age with a physician diagnosis of asthma for ≥ 2 years, a BEC of ≥ 150 cells/ μ l at screening or ≥ 300 cells/ μ l during the previous 12 months, had regular treatment with medium-to-high-dose ICS in the previous 12 months, plus current treatment with ≥ 1 additional controller for ≥ 3 months and had a history of ≥ 2 exacerbations requiring SCS in the previous 12 months. †Depemokimab:placebo. ‡See manuscript for full list of assessed subgroups. ACQ-5, Asthma Control Questionnaire-5; ADSD, Asthma Daytime Symptom Diary; ANSQ, Asthma Nighttime Symptom Diary; BEC, blood eosinophil count; CRSwNP, chronic rhinosinusitis with nasal polyps; ICS, inhaled corticosteroid; PRO, patient-reported outcome; R, randomized; SC, subcutaneous; SCS, systemic corticosteroids; SGRQ, St George's Respiratory Questionnaire; SoC, standard of care; Wk, week. These analyses of the SWIFT trials (GSK ID [ClinicalTrials.gov ID]: 206713 [NCT04719832]/213744 [NCT04718103]) were funded by GSK.

Abbreviations used

ACQ- Asthma Control Questionnaire
 ADSD- Asthma Daytime Symptom Diary
 ANSD- Asthma Nighttime Symptom Diary
 BEC- Blood eosinophil count
 CRSwNP- Chronic rhinosinusitis with nasal polyps
 HRQoL- Health-related quality of life
 ICS- Inhaled corticosteroids
 OCS- Oral corticosteroid
 SGRQ- St George's Respiratory Questionnaire

BACKGROUND: Depemokimab, the first ultra-long-acting biologic with enhanced IL-5 binding affinity, high inhibition potency, and extended half-life, enables twice-yearly dosing in patients with asthma. In phase III SWIFT-1/-2 trials, depemokimab significantly reduced the annualized exacerbation rate by 54% versus placebo with sustained suppression of inflammation assessed by blood eosinophil count in type 2 asthma.

OBJECTIVE: To evaluate the onset and duration of depemokimab efficacy across each 26-week dosing period in the pooled SWIFT-1/-2 population and identify patient characteristics associated with enhanced clinical response.

METHODS: Patients with type 2 asthma, independent of Asthma Control Questionnaire-5 (ACQ-5) status, were randomized 2:1 to receive depemokimab 100 mg subcutaneously or placebo every 26 weeks for 52 weeks. Prespecified end points included time to first exacerbation, St George's Respiratory

Questionnaire total score, ACQ-5 score, Asthma Nighttime Symptom Diary weekly score, Asthma Daytime Symptom Diary weekly score, and rescue medication use over time. We conducted *post hoc* subgroup analyses by baseline characteristics. **RESULTS:** Depemokimab reduced the probability of first exacerbation over 52 weeks versus placebo by 46% (hazard ratio = 0.54; 95% CI, 0.43-0.69), with effects from week 4 sustained across both dosing periods. Annualized exacerbation rate reductions were most pronounced in patients with asthma disease duration less than 10 years, comorbid chronic rhinosinusitis with nasal polyps, and medium-dose inhaled corticosteroids (ICS) at baseline. Across each dosing period, depemokimab also improved St George's Respiratory Questionnaire, ACQ-5, Asthma Nighttime Symptom Diary, and Asthma Daytime Symptom Diary scores, particularly in patients with baseline ACQ-5 scores of 1.5 or greater, with sustained improvements in rescue medication.

CONCLUSION: Early and sustained efficacy with depemokimab was observed across 26-week dosing periods in type 2 asthma, with enhanced benefits in patients with shorter disease duration who had not progressed to high-dose ICS. © 2026 The Authors. Published by Elsevier Inc. on behalf of the American Academy of Allergy, Asthma & Immunology. This is an open access article under the CC BY license (<http://creativecommons.org/licenses/by/4.0/>). (J Allergy Clin Immunol Pract 2026;14:1351-62)

Key words: Asthma control; Asthma exacerbation; Anti-IL-5 antibody; Biologics; Dosing period; Patient-reported outcomes; Quality of life; Rescue medication; Sustained efficacy; Symptoms

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INTRODUCTION

Patients with uncontrolled asthma experience unpredictable exacerbations that significantly impair health-related quality of life (HRQoL).^{1,2} Type 2 inflammation has a central role in the pathophysiology of asthma, contributing to airway inflammation, mucus hypersecretion, and airway mucus plugging, which in turn are associated with persistent symptoms and an increased risk of exacerbations.²⁻⁴ Whereas many patients with type 2 asthma respond to inhaled corticosteroids (ICS), a substantial proportion continue to experience poor asthma control, frequent exacerbations, and impaired HRQoL, often necessitating use of oral corticosteroids (OCS) as either rescue bursts or maintenance therapy.⁵

For these patients, biologic therapies targeting key type 2 inflammatory pathways, such as IL-5, offer a more personalized approach. IL-5 is a core cytokine in type 2 inflammation with broad multidirectional effects on immune cells and structural cells, known for promoting eosinophil maturation, survival, activation, and recruitment, while also influencing mast cells, basophils, epithelial cells, fibroblasts, and other airway-resident cells.^{2,6-10} These broad effects underscore the role of IL-5 in sustaining type 2 inflammation and airway dysfunction, reinforcing its importance as a therapeutic target in type 2 asthma.

Targeted biologics have transformed the treatment of asthma by reducing exacerbation rates and improving asthma control, HRQoL and lung function while decreasing the need for OCS.¹¹⁻¹³ Furthermore, 20% to 83% of patients achieve clinical remission with biologic therapy depending on the definition used, patient characteristics, and the duration of treatment.¹⁴⁻¹⁶ Previous evidence suggests that early intervention with biologics in asthma may result in improved outcomes by preventing irreversible lung function decline and minimizing the impact of adverse effects associated with corticosteroid use, although data are limited.¹⁷ However, biologic underuse, delayed initiation, nonadherence, and nonoptimal persistence remain common challenges in clinical practice.¹⁸⁻²⁰ Dosing frequency may be one of the barriers to overcoming these challenges, because shorter dosing intervals have been associated with worse adherence to biologics,¹⁹ and many patients and physicians prefer less frequent dosing.²¹ Consequently, biologics with a reduced dosing frequency that also provide sustained suppression of inflammation may improve adherence and help patients achieve long-term treatment goals, such as clinical remission.^{19,21,22}

Depemokimab is the first ultra-long-acting biologic with enhanced IL-5 binding affinity, high potency, and an extended half-life, enabling twice-yearly dosing and sustained suppression of type 2 inflammation in patients with type 2 asthma.^{23,24} In the phase III SWIFT-1/-2 trials, depemokimab significantly reduced asthma exacerbations by 54% in patients with type 2 asthma, characterized by blood eosinophil count (BEC).²⁵ There was also a rapid reduction in BEC after administration of depemokimab that was maintained over the 52-week treatment period, reflecting sustained suppression of the underlying inflammatory pathway.²⁵ Improvements were also observed in patient-reported outcomes, including HRQoL, asthma control, and lung function,²⁵ supporting the potential for broader clinical benefit. However, variability in baseline characteristics may have limited the ability to detect statistically significant differences in these measures within the whole trial population.²⁵

Because depemokimab is the first ultra-long-acting biologic, it is important to demonstrate that its clinical benefits are consistently sustained across the full 6-month dosing period with no decline in

efficacy. Additionally, further insights into the baseline characteristics of patients in the SWIFT-1/-2 trials may help identify those most likely to benefit from depemokimab: for instance, whether earlier treatment initiation (as indicated by shorter disease duration and lower maintenance therapy dose) is associated with better outcomes. Therefore, we carried out analyses of the SWIFT-1/-2 trials to evaluate the onset and duration of depemokimab efficacy on exacerbations and patient-reported outcomes across each 26-week dosing period in the pooled SWIFT-1/-2 population and to assess outcomes at week 52 stratified by baseline characteristics.

METHODS

SWIFT-1 and SWIFT-2 study design

SWIFT-1 and SWIFT-2 were phase III, multicenter, randomized, double-blind, placebo-controlled replicate trials conducted from February 4, 2021 to April 11, 2024. Detailed methods are published elsewhere.²⁵ In brief, patients were randomized 2:1 to receive depemokimab 100 mg subcutaneously or placebo at weeks 0 and 26, in addition to standard of care. Rescue medication (albuterol/salbutamol metered dose inhalers) was permitted throughout the study.

Eligible patients were aged 12 years or older with a physician diagnosis of asthma for 2 years or more and a BEC of 150 cells/mL or greater at screening or 300 cells/mL or greater during the previous 12 months, and had had regular treatment with medium- to high-dose ICS in the previous 12 months, plus current treatment with one or more additional controllers for 3 months or longer. All had a history of two or more exacerbations requiring systemic corticosteroids in the previous 12 months. Any score on the Asthma Control Questionnaire-5 (ACQ-5) was acceptable for enrollment. Patients were excluded if they were receiving a biologic therapy as part of current maintenance therapy, anti-IL-5 antibody therapy in the previous 12 months, or other antibody therapy (eg, omalizumab or dupilumab) within 130 days before visit 1 (to allow for sufficient washout time before the start of the study). We collected no data on biologic-naïve or biologic-experienced status, or on previous biologic type (if applicable).

Both trials were conducted in accordance with ethical principles derived from international guidelines, including the Declaration of Helsinki, the Council for International Organizations of Medical Sciences, and the International Council on Harmonisation Good Clinical Practice guidelines, as well as all applicable laws and regulations. The protocols were approved by an institutional review board or ethics committee at each site, and all patients provided informed consent.

End points and assessments

Here we report the time to the first exacerbation (defined as worsening of asthma leading to the use of systemic corticosteroids or at least a doubling in the dose for 3 days or more in patients who were receiving OCS, hospitalization, or an emergency department visit; a prespecified “other” end point in the original SWIFT trials) in the overall pooled SWIFT-1/-2 population. We analyzed the occurrence of exacerbations by study day (individual exacerbation pattern) and 13-week intervals (both *post hoc*). We also conducted subgroup analyses to evaluate the annualized rate of exacerbations by these baseline patient characteristics: ACQ-5 score (<1.5 and ≥1.5, *post hoc*), age at asthma onset (<18 and ≥18 years, *post hoc*), season (spring, summer, autumn, and winter, *post hoc*), BEC (<150, ≥150 to <300, and ≥300 cells/μL, prespecified), exacerbations in the previous year (≤2 and >2, *post hoc*), asthma duration by quartile (<10, 10–17, 18–33, and ≥34 years, prespecified), ICS level (medium- or high-dose, prespecified), and past or current chronic rhinosinusitis with nasal polyps (CRSwNP) (prespecified).

We also report the following prespecified secondary end points assessed in the overall pooled SWIFT-1/-2 population: change from baseline during the 52-week period in St George's Respiratory Questionnaire (SGRQ) total score, ACQ-5 score, and Asthma Nighttime Symptom Diary (ANS) and Asthma Daytime Symptom Diary (ADSD) weekly scores. The ADS/ANS were qualified by the US Food and Drug Administration within its Clinical Outcome Assessment Qualification Program in 2019, and the qualification statement supports these diaries as measures of asthma symptoms in drug development.²⁶ These secondary analyses specified earlier were also carried out by baseline ACQ-5 score (ie, <1.5 and ≥1.5, *post hoc*) to assess the impact of baseline symptom severity on these outcomes because the SWIFT-1/-2 trials did not require patients to be symptomatic at baseline.

Finally, we report the change from baseline in rescue medication use during the 52-week period assessed in the overall pooled SWIFT-1/-2 population (prespecified "other" end point). No data were recorded on adherence to background inhaler therapy during the SWIFT-1 and SWIFT-2 studies; however, patients with a known lack of adherence to controller medications or the lack of ability to follow physician recommendations were not eligible for inclusion in these studies.

Statistical analysis

Data from the full analysis set of SWIFT-1/-2 were pooled for this analysis. We analyzed the time to first exacerbation using a Cox's proportional hazards model with covariates for treatment group, exacerbation history (two, three, and four or more events), baseline ICS dose (medium and high), baseline prebronchodilator percent predicted FEV₁, geographic region, and study (SWIFT-1 or SWIFT-2). Mean changes from baseline in SGRQ total score, ACQ-5 score, and ANSD and ADS weekly scores at each time point over the 52-week

treatment period were analyzed using mixed-models repeated measures with covariates of treatment group, baseline ICS dose (medium or high), exacerbation history (two, three, and four or more events), geographic region, baseline score for variable assessed, baseline pre-bronchodilator percent predicted FEV₁, study (SWIFT-1 or SWIFT-2), visit, visit by baseline score for variable assessed, and visit by treatment group. Models were fit with an unstructured variance-covariance matrix. We calculated rescue medication as the mean daily number of occasions of use over a 2-week period. Analyses were conducted using SAS Data Science (version 3.6, SAS Institute Inc, Cary, NC).

RESULTS

Patient populations

Overall, 762 eligible patients were included in this pooled SWIFT-1/-2 analysis (n = 502 in the depemokimab group and n = 260 in the placebo group). Notably, one patient with a history of zero exacerbations was included. Baseline patient characteristics of the pooled SWIFT-1/-2 population were generally balanced across treatment groups (Table I); mean disease duration ranged from 22 to 24 years (min-max range, 2 to 78 years) across treatment groups. Mean (SD) exacerbations in the previous 12 months was 2.4 (1.59) in the depemokimab group and 2.4 (1.15) in the placebo group. Overall, 5% of patients were receiving maintenance OCS at baseline (4% to 7% across treatment groups), and around half of patients (42% to 47%) were receiving medium-dose ICS (Table I).

Exacerbations

Treatment with depemokimab significantly increased the time to first asthma exacerbation, reducing the probability of having an exacerbation over 52 weeks by 46% compared with placebo (32% vs 49%; hazard ratio = 0.54; 95% CI, 0.43-0.69). The difference

TABLE I. Baseline patient characteristics for the total pooled SWIFT-1/-2 population

Characteristic	Pooled SWIFT-1/-2 population		
	Total (n = 762)	Depemokimab 100 mg subcutaneous (n = 502)	Placebo (n = 260)
Female, n (%)	464 (61)	304 (61)	160 (62)
Age, y (mean [SD])	53.3 (15.23)	53.8 (14.94)	52.4 (15.77)
Race, n (%)			
Asian	133 (17)	90 (18)	43 (17)
Black	36 (5)	22 (4)	14 (5)
Other*	5 (<1)	2 (<1)	3 (1)
White	588 (77)	388 (77)	200 (77)
Disease duration, y (mean [SD])	23.4 (17.45)	24.0 (17.54)	22.0 (17.22)
Exacerbations† in previous 12 mo, n (mean [SD])	2.4 (1.46)	2.4 (1.59)	2.4 (1.15)
St George's Respiratory Questionnaire total score (mean [SD])‡	44.42 (19.708)	44.73 (19.651)	43.82 (19.842)
Asthma Control Questionnaire-5 score, mean (mean [SD])‡	2.22 (1.083)	2.21 (1.097)	2.24 (1.059)
Prebronchodilator FEV ₁ , L (mean [SD])‡	1.841 (0.6848)	1.852 (0.6771)	1.819 (0.7003)
Blood eosinophil count, cells/μL, geometric mean (SD Log)‡	318 (0.8355)	318 (0.8410)	319 (0.8263)
Total IgE, U/mL, geometric mean (SD Log)‡	161.88 (1.485)	151.12 (1.492)	184.74 (1.465)
Baseline ICS dose level, n (%)			
Medium	333 (44)	212 (42)	121 (47)
High	429 (56)	290 (58)	139 (53)
Maintenance OCS use, n (%)	40 (5)	21 (4)	19 (7)
Maintenance OCS mean daily dose, mg (SD)	7.0 (3.8)	6.2 (2.7)	7.9 (4.6)

OCS, oral corticosteroid.

*Includes American Indian or Alaska Native, Native Hawaiian or other Pacific Islander, and mixed race.

†Worsening of asthma leading to the use of systemic corticosteroid or at least a doubling in the dose for ≥3 d in patients who were receiving OCS, hospitalization, or an emergency department visit.

‡We assessed the St George's Respiratory Questionnaire total score, Asthma Control Questionnaire-5 score, FEV₁, blood eosinophil count, and IgE in 744, 744, 742, 762, and 754 patients, respectively.

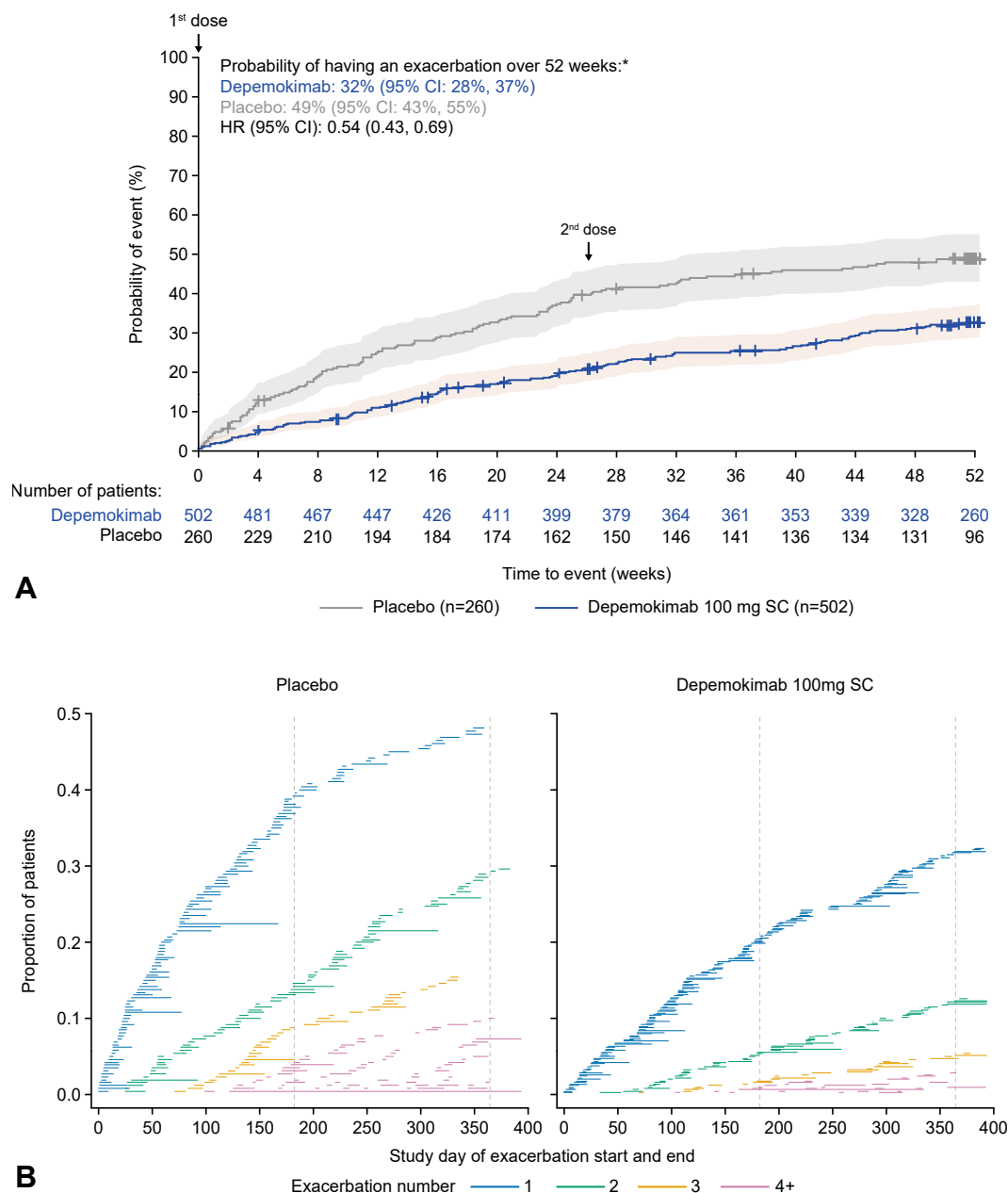


FIGURE 1. Time to first exacerbation (**A**) and *post hoc* analysis of individual exacerbations patterns by study day (**B**) over 52 weeks (pooled SWIFT-1/-2 population, n = 762). (**A**) Shaded area represents 95% CI; crosses indicate censoring of patients. (**B**) Vertical lines at days 182 and 364 represent the end of each dosing period. The y-axis was scaled using the total number of patients in each treatment group. HR, hazard ratio; SC, subcutaneous.

in exacerbation risk between depemokimab and placebo became apparent as early as 4 weeks after the first dose, with no evidence of waning of response over 52 weeks (Figure 1, A). In the analysis of individual exacerbation patterns by study day, a lower cumulative proportion of patients experienced exacerbations in the depemokimab group compared with placebo across the 52-week study period. Most patients had only one or two exacerbations, which occurred less frequently and were more evenly distributed over time (Figure 1, B). A lower proportion of patients receiving depemokimab experienced four or more exacerbations compared with patients taking a placebo (2.8% vs 10%, respectively). The

distribution of exacerbations indicates a delayed onset of events with depemokimab compared with placebo (Figure 1, A and B). Additionally, patients receiving depemokimab consistently achieved a greater reduction in the annualized rate of exacerbations than did patients taking a placebo when analyzed by 13-week intervals (percent reduction range, 33% to 61%) (see Table E1 in this article's Online Repository at www.jaci-inpractice.org). Reductions in the annualized rate of exacerbations were consistent across ACQ-5 subgroups, age of asthma onset subgroups, seasonal subgroups, and subgroups by BEC at baseline (Figure 2). The greatest reductions in the annualized rate of exacerbations were observed with

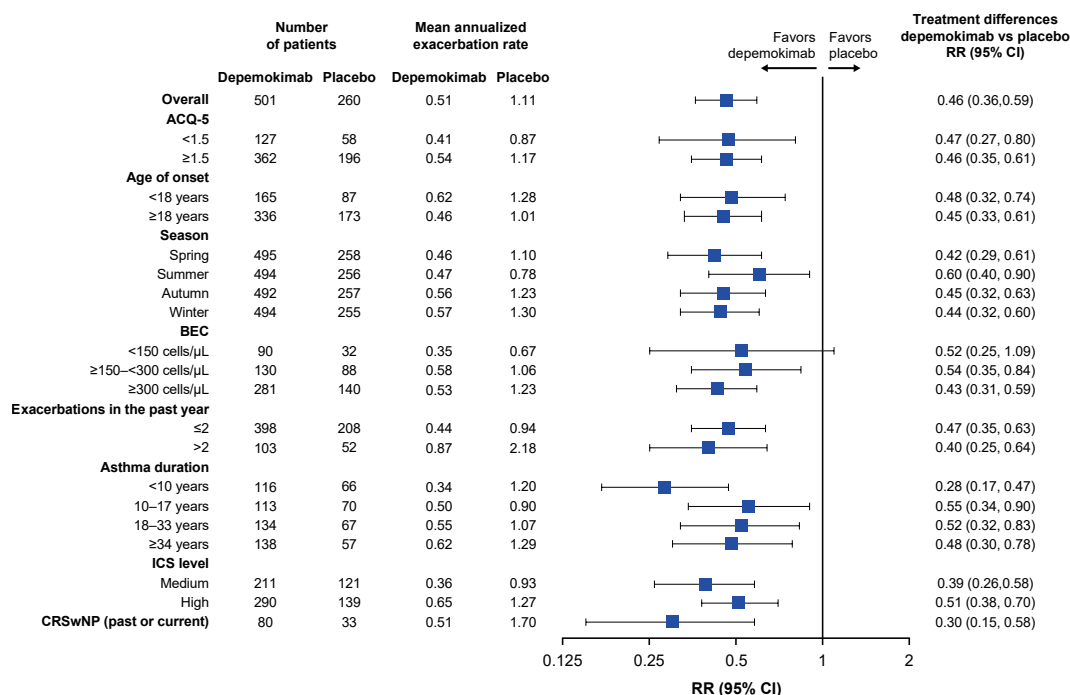


FIGURE 2. Annualized rate of exacerbations by baseline patient characteristics (pooled SWIFT-1/-2 population, $n = 762$). *ACQ-5*, Asthma Control Questionnaire-5; *BEC*, blood eosinophil count; *CRSwNP*, chronic rhinosinusitis with nasal polyps; *RR*, rate ratio.

depemokimab versus placebo in patients with the shortest disease duration (ie, <10 years; 72% rate reduction), in those with past or current CRSwNP (70% rate reduction), and in those receiving medium-dose ICS at baseline (64% rate reduction) (Figure 2).

St George's Respiratory Questionnaire total score

In the overall SWIFT-1/-2 pooled population, we observed reductions in SGRQ total scores from baseline in both the depemokimab and placebo groups, with greater reductions in the depemokimab group as early as week 4 (depemokimab vs placebo difference, -2.77 ; 95% CI, -5.02 to -0.51) and maintained through to week 52 (-2.88 ; 95% CI, -5.43 to -0.32) (Figure 3, A). We observed a difference in SGRQ total score reduction between ACQ-5 subgroups over 52 weeks, in which symptomatic patients (ie, with a baseline ACQ-5 score of ≥ 1.5) experienced greater reductions with depemokimab versus placebo relative to non-symptomatic patients (ie, with a baseline ACQ-5 score of < 1.5). These differences were observed from week 4 and were sustained across each 26-week dosing period (Figure 3, B and C).

Asthma Control Questionnaire-5 score

In the overall SWIFT-1/-2 pooled population, we observed improvements from baseline in least-squares mean ACQ-5 scores in both treatment groups that were sustained across each 26-week dosing period, with differences seen from week 4 (depemokimab vs placebo difference, -0.25 ; 95% CI, -0.39 to -0.12). At week 52, the treatment difference between depemokimab and placebo groups was -0.08 (95% CI, -0.24 to 0.07) (Figure 4, A). Over 52 weeks, similar to the trend seen in SGRQ, patients with a baseline ACQ-5 score of 1.5 or greater experienced a trend for greater reductions in ACQ-5 scores with depemokimab versus placebo than did those with baseline ACQ-5 scores less than 1.5 (Figure 4, B and C).

Asthma Nighttime Symptom Diary and Asthma Daytime Symptom Diary

In the overall SWIFT-1/-2 pooled population, we observed greater reductions in both the weekly ANSD and ADSD symptom scores with depemokimab versus placebo from week 2 (depemokimab vs placebo difference, -0.21 ; 95% CI, -0.38 to -0.03 ; and depemokimab vs placebo difference, -0.17 ; 95% CI, -0.33 to -0.01 , respectively). Changes from baseline in ANSD and ADSD scores were maintained across each 26-week dosing period in the depemokimab group (differences vs placebo at week 52: ANSD, -0.16 ; 95% CI, -0.40 to 0.08]; ADSD, -0.12 ; 95% CI, -0.34 to 0.09) (Figures 5, A and 6, A). We observed greater reductions in ANSD and ADSD weekly scores in patients with a baseline ACQ-5 score of 1.5 or greater compared with those with a score of less than 1.5. The treatment difference versus placebo emerged as early as week 2 and was sustained across each 26-week dosing period (Figures 5, B and C, and 6, B and C).

Rescue medication use

In the overall SWIFT-1/-2 pooled population, a reduction in mean daily rescue medication use (2-week mean) from baseline with depemokimab versus placebo was evident from weeks 3 to 4 (Figure 7). This reduction was maintained across each 26-week dosing period through to week 52, with consistently lower use in the depemokimab group compared with placebo (Figure 7). However, there were substantial variations in rescue medication use among patients in both treatment groups, and by week 52 the differences between depemokimab and placebo were less evident.

DISCUSSION

This pooled analysis of the SWIFT-1/-2 population provides insight into the clinical profile and potential impact of twice-yearly

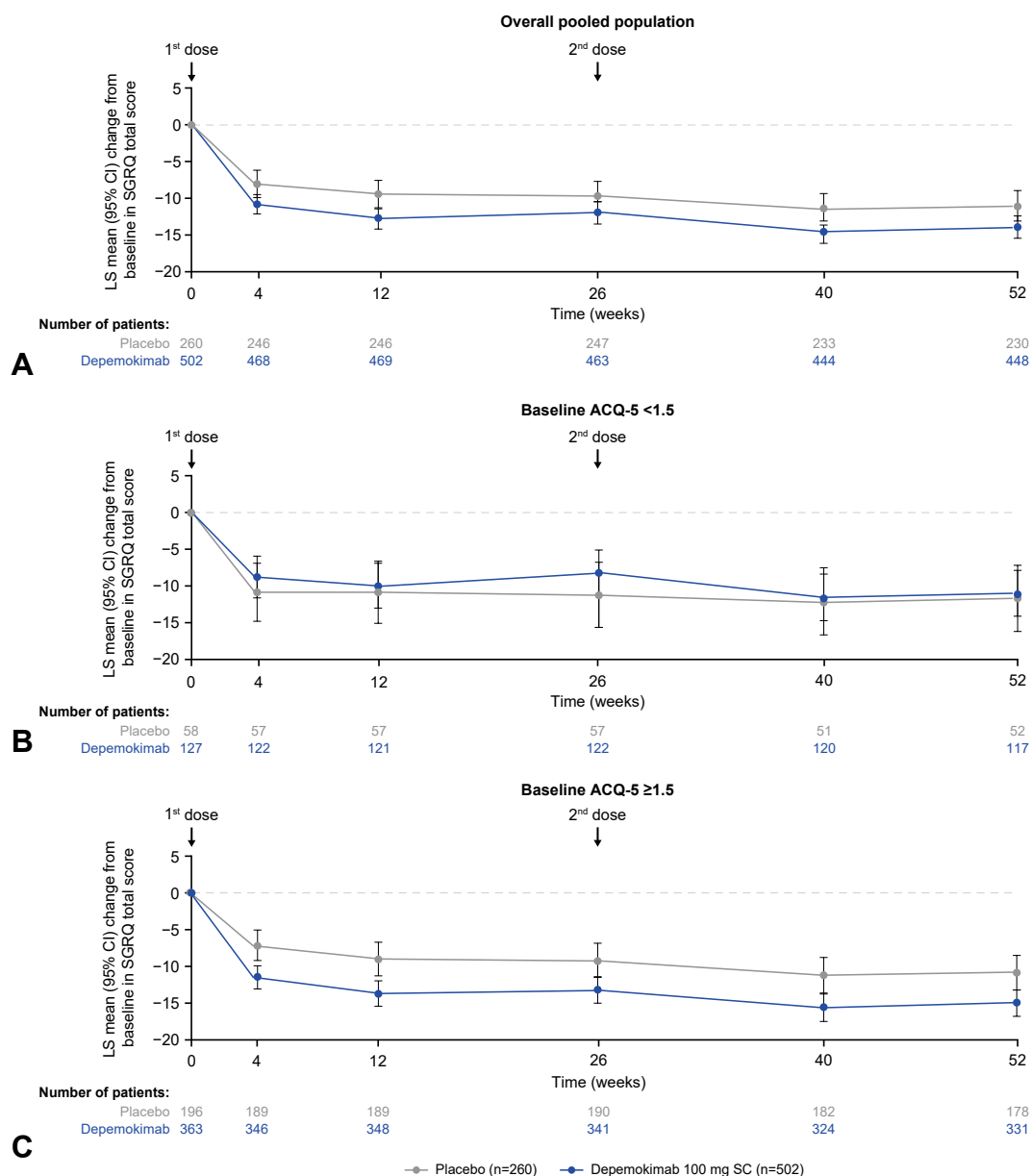


FIGURE 3. Change from baseline to week 52 in St George's Respiratory Questionnaire (SGRQ) total score in overall pooled population (A) and by baseline Asthma Control Questionnaire-5 (ACQ-5) less than 1.5 (B) and 1.5 or greater (C) scores (pooled SWIFT-1/-2 population, n = 762). SGRQ = 0 to 100; higher scores indicate worse function. LS, least squares; SC, subcutaneous.

treatment with depemokimab, the first ultra-long-acting respiratory biologic, in patients with type 2 asthma characterized by BEC. The study allowed for an evaluation of the onset and maintenance of treatment effects over time, as well as patterns of response across baseline patient characteristics subgroups, forming the basis for a broader discussion of its potential role in asthma management.

In this pooled analysis, we observed a rapid and sustained reduction in exacerbation rates with no evidence of waning during the 26-week dosing schedule with depemokimab, decreasing the risk of experiencing an exacerbation by 46% versus placebo over the 52-week treatment period, and producing a considerable delay in time to first exacerbation, with similar cumulative incidence observed across each 26-week dosing period. Reductions in exacerbation rates were consistent across ACQ-5 scores (<1.5 vs ≥1.5), age at asthma

onset (<18 vs 18 years), and seasonal (spring, summer, autumn, and winter) subgroups, suggesting a consistent effect on exacerbations independent of baseline symptom severity, age at onset of asthma disease, and seasonal variations. Reductions in exacerbation rates were also consistent across baseline BEC subgroups. However, because study eligibility required all patients to have evidence of blood eosinophilia (≥150 cells/μL at screening or ≥300 cells/μL during the previous 12 months) as a biomarker of type 2 inflammation, the ability to detect a relationship between baseline BEC and treatment response may have been limited. Approximately 80% of the SWIFT-1/-2 population had had just two exacerbations in the year before study entry (despite the inclusion criterion of two or more exacerbations), and subgroup analyses showed that these patients experienced reductions in exacerbation rates with

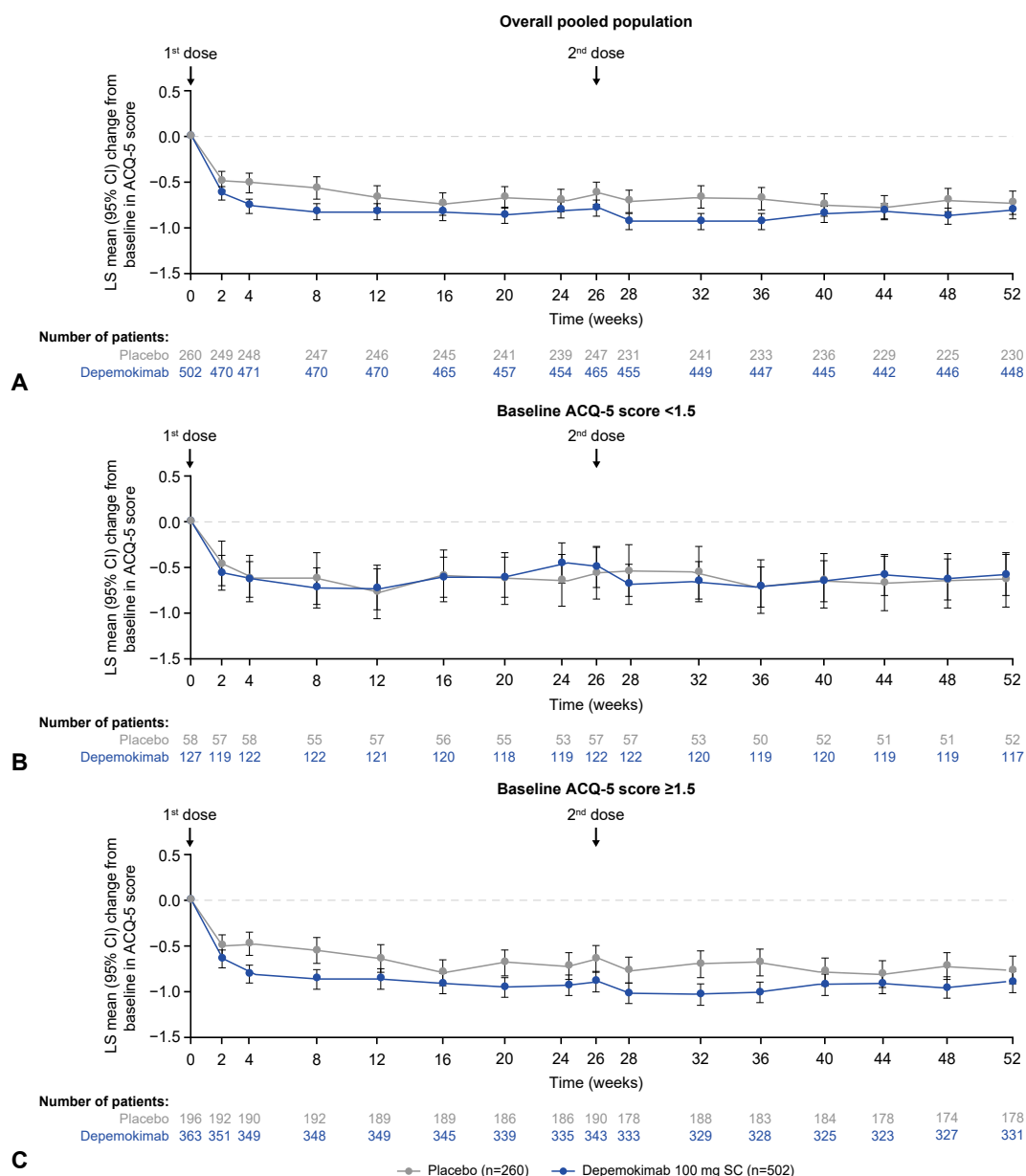


FIGURE 4. Change from baseline to week 52 in Asthma Control Questionnaire-5 (ACQ-5) score in the overall pooled population (**A**) and by baseline ACQ-5 less than 1.5 (**B**) and 1.5 or greater (**C**) scores (pooled SWIFT-1/-2 population, $n = 762$). *LS*, least squares.

depemokimab versus placebo that were comparable to those of the overall population. This finding suggests that timely intervention, rather than waiting until patients experience three or more exacerbations, may provide meaningful clinical benefit. At week 52, the greatest reductions in exacerbation rates with depemokimab versus placebo were observed in patients with a shorter asthma duration (ie, <10 years since diagnosis), a history of comorbid CRSwNP, or medium-dose ICS, suggesting that these factors may be important predictors of treatment response. The enhanced benefits observed in patients with shorter disease duration and those receiving medium-dose ICS add to the growing body of evidence to support the potential opportunity to initiate biologics before disease progression and escalation to high-dose ICS therapy.^{17,28-33}

Most clinical trials of biologic therapies for asthma have required a baseline ACQ-5 of greater than 1.5, so there are limited data on

exacerbation reduction in patients with better symptom control. The results of the SWIFT-1/-2 data analysis suggest that patients with better symptom control (ACQ-5 <1.5) had exacerbation reductions with depemokimab that were comparable to those with higher baseline symptom burden before treatment, indicating that the benefit of treatment extends to patients with well-controlled symptoms, thus dissociating the biology of exacerbation risk from that of symptom generation, which may be multifactorial as previously suggested.^{34,35} These data also align with previous evidence showing that patients with either poorly or well-controlled symptoms may remain at risk of future exacerbations, particularly in the context of elevated biomarkers, such as BEC and FeNO, or a history of exacerbations.³⁴

Nonsignificant reductions in the SGRQ total score with depemokimab versus placebo at week 52 in the pooled population were

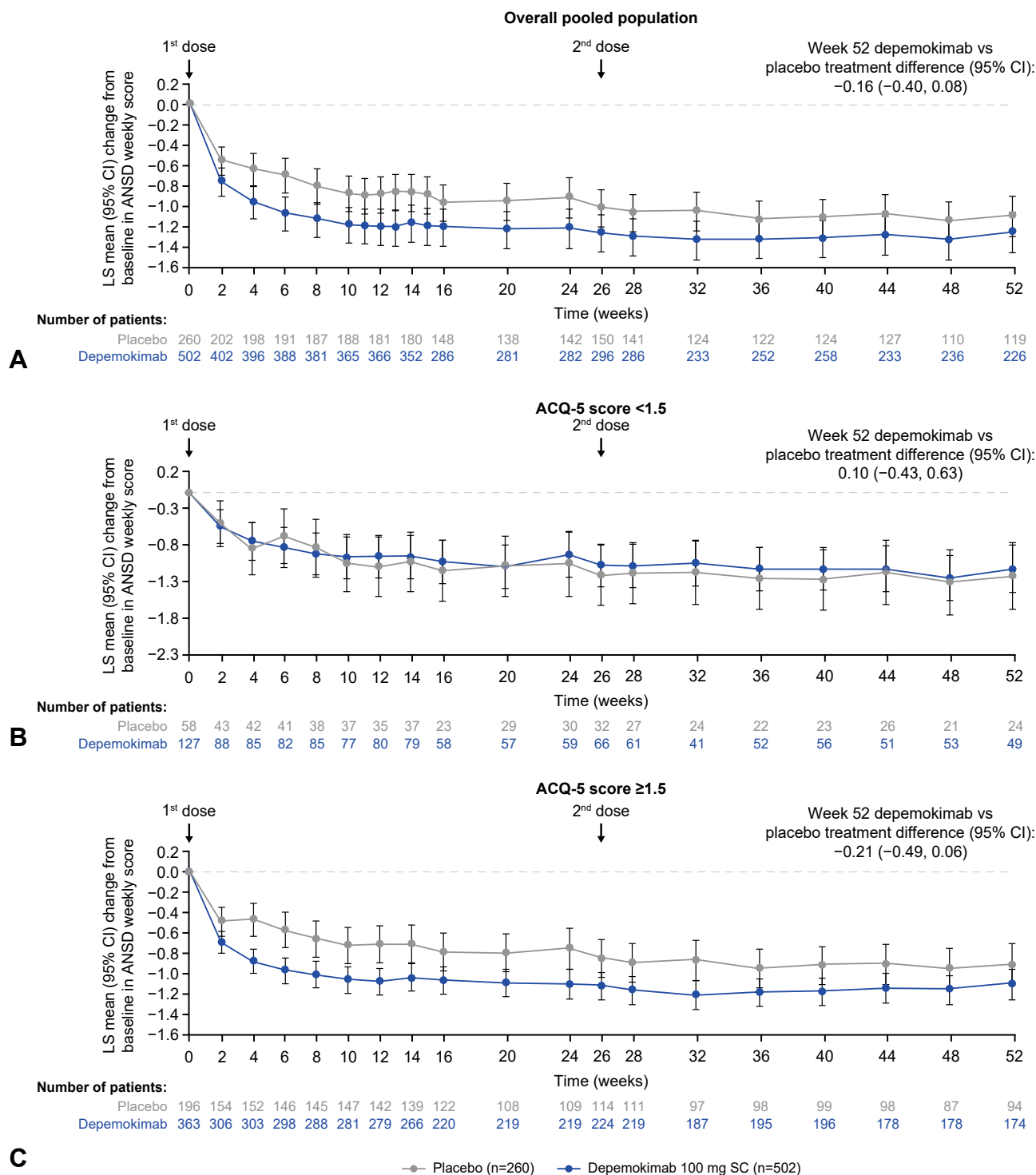


FIGURE 5. Change from baseline to week 52 in Asthma Nighttime Symptom Diary (ANSND) weekly scores in overall population (**A**) and by baseline Asthma Control Questionnaire-5 (ACQ-5) less than 1.5 (**B**) and 1.5 or greater (**C**) scores (pooled SWIFT-1/-2 population, $n = 762$). Score range = 0 to 10, with higher score indicating worse symptoms; within-patients minimal clinically important difference of -1.5 .²⁷ LS, least squares; SC, subcutaneous.

previously reported,²⁵ but the current analysis shows that these reductions were rapid in onset and sustained across each 26-week dosing period. Although reductions in ACQ-5 scores from baseline with depemokimab were also rapid in onset and sustained across each

26-week dosing period, the treatment difference versus placebo was nominal. This could be partly due to the lack of requirement for patients to have poor control (defined by the ACQ-5 score) to enter the SWIFT-1/-2 studies; some patients may have had limited

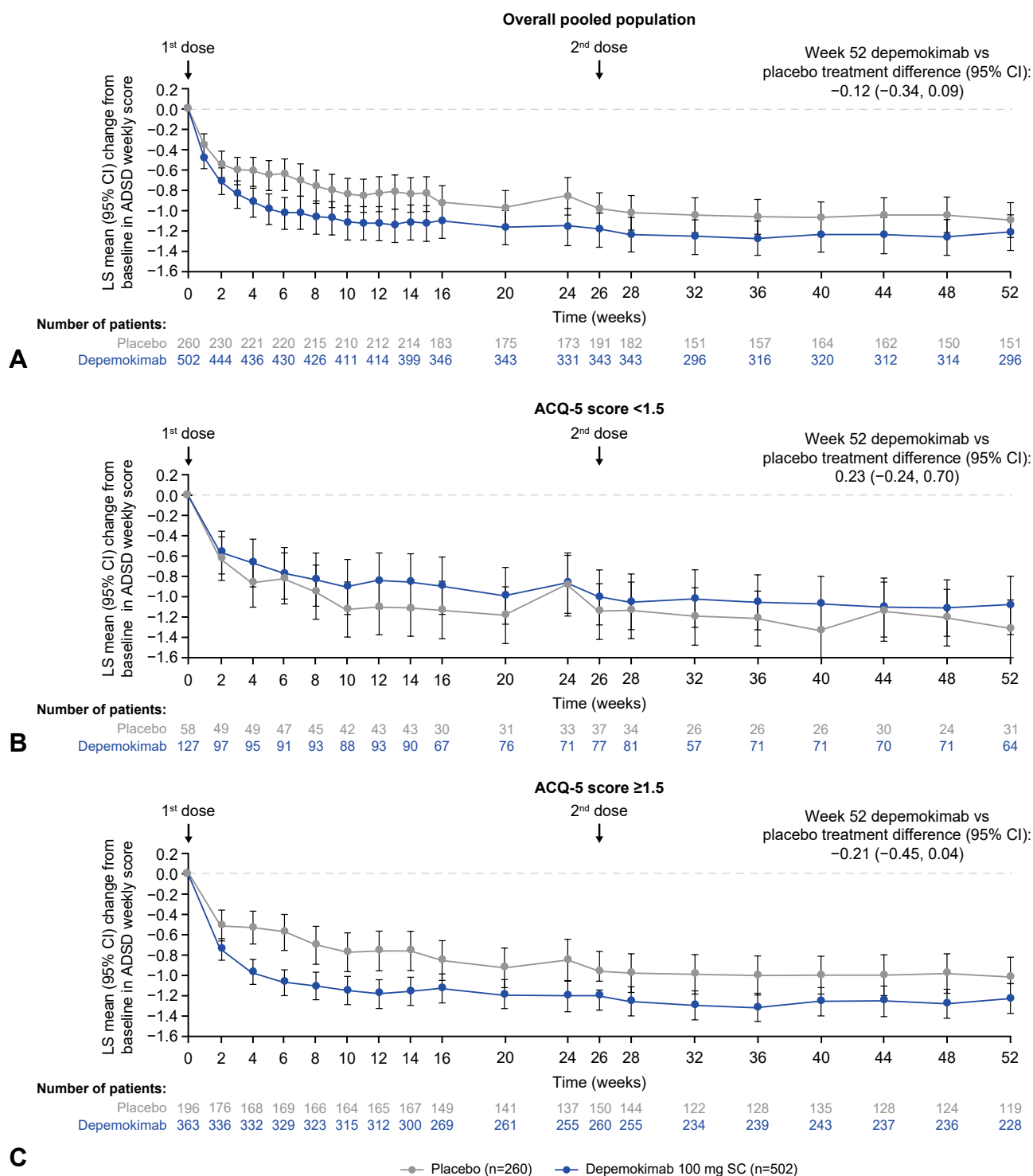


FIGURE 6. Change from baseline to week 52 in Asthma Daytime Symptom Diary (ADSD) weekly scores in overall population (**A**) and by baseline Asthma Control Questionnaire-5 (ACQ-5) less than 1.5 (**B**) and 1.5 or greater (**C**) scores (pooled SWIFT-1/-2 population, $n = 762$). Score range = 0 to 10, with higher score indicating worse symptoms; within-patients minimal clinically important difference of -1.2 .²⁷ LS, least squares; SC, subcutaneous.

potential for additional symptom improvements. Indeed, patients with uncontrolled asthma at baseline (ACQ-5 score of ≥ 1.5) had numerically greater reductions in SGRQ and ACQ-5 scores, as well as in ANSD and ADSD scores, over the 52-week study than those with

a score less than 1.5. Additionally, a high substantial placebo response in ACQ-5 score (-0.73 points at week 52) may have contributed to the small response size observed. High placebo response may also explain why the reductions with depemokimab in ANSD, ADSD,

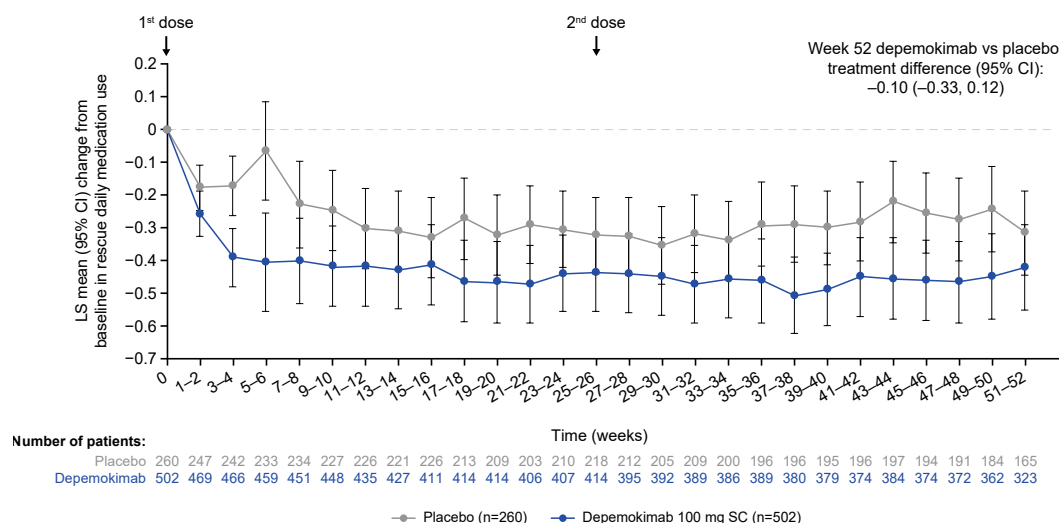


FIGURE 7. Change from baseline in mean daily rescue medication use through to week 52 (pooled SWIFT-1/-2 population, $n = 762$). LS, least squares; SC, subcutaneous.

and rescue medication use observed in the overall pooled SWIFT-1/-2 population were less pronounced relative to placebo.

This pooled analysis strengthens the primary findings of the individual SWIFT-1/-2 trials by increasing statistical power. Furthermore, it demonstrates the evolution of biologic use in asthma over time, showing efficacy in patients with type 2 asthma who had relatively low numbers of exacerbations in the year before the start of the study ($\sim 80\%$ of patients had two exacerbations during this period), low maintenance OCS use (5%), and high levels of medium-dose (rather than high-dose) ICS use (44%) compared with patients involved in the early randomized controlled trials of anti-IL-5 therapy.^{36,37} Nonetheless, there are some limitations. For example, the relatively milder asthma population may have affected the power to detect differences between depemokimab and placebo. It is also possible that the comparable reduction in exacerbation rate observed in patients with two or fewer exacerbations at baseline (compared with patients with more than two exacerbations at baseline) may be due at least partly to their higher degree of health at baseline (correlating with more pliable lung function and a greater ability to recover from illness rapidly, resulting in fewer exacerbations). Most of the subgroup analyses were conducted *post hoc*, and no adjustments were made for multiplicity in the subgroup analyses. Therefore, these results should be interpreted with caution. Additionally, for some end points (eg, ANSD and ADSD) and baseline subgroups (eg, BEC < 150 cells/ μ L), the number of patients with available data at later time points was relatively small, limiting the ability to detect meaningful differences or trends over time. A substantial subjective placebo response, particularly in patient-reported outcomes, also made it harder to distinguish between depemokimab and placebo for ACQ-5, ANSD, ADSD, and rescue medication use measures.

As noted previously, no data were recorded on adherence to background inhaler therapy during the SWIFT-1 and SWIFT-2 studies. Although patients with a known lack of adherence to controller medications or a lack of ability to follow physician recommendations were not eligible for inclusion in these studies, it is still possible that low or varying adherence to background inhaler therapy may have affected asthma control in some patients. Also, no data were collected on biologic-naïve or biologic-experienced

status, or (for the latter group) on previous biologic type. Therefore, no conclusions can be drawn regarding whether depemokimab initiation is beneficial in biologic-naïve patients or in patients who previously had not responded to other biologic therapies. Also, it is possible that patients may have been enrolled who had previously responded to anti-IL-5 therapy (> 12 months before initiating depemokimab), and this may have contributed to the positive results reported here. In addition, disease duration was defined broadly (eg, from diagnosis or symptom onset), which may introduce some imprecision. Finally, FeNO was not measured even though it commonly is used as a biomarker of type 2 inflammation, although the close correlation between FeNO and sputum eosinophils in asthma^{38,39} and the close relationship between serum IL-5 and other type 2 cytokines such as IL-4 and IL-3^{40,41} support blood eosinophils alone as a good biomarker of type 2 inflammation.⁴² Despite these limitations, the consistency of findings across multiple end points and subgroups supports the potential benefit of depemokimab in patients with type 2 asthma.

We observed durable clinical benefits with twice-yearly depemokimab over 52 weeks, considerably delaying the time to first exacerbation and enhancing HRQoL. Improvements were rapid, and efficacy was sustained across each 26-week dosing period with no evidence of waning. Reductions in exacerbation rates were independent of baseline asthma control, with the greatest benefits seen in patients with a disease duration of less than 10 years, those with comorbid CRSwNP, and those receiving medium-dose ICS. Trends toward sustained improvements in asthma control, symptoms, and rescue medication use were also observed, particularly in patients who were symptomatic at baseline. These results highlight the opportunity for biologic initiation in patients with shorter disease duration who had not progressed to high-dose ICS, with durable treatment responses that can be achieved with depemokimab.

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A plain language summary of this article is included as an Appendix (available in this article's Online Repository at www.jaci-inpractice.org).

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TABLE E1. Annualized rate of exacerbations by 13-wk intervals (pooled SWIFT-1/2 population, n = 762)

	1-13 wk		14-26 wk		27-39 wk		≥40 wk	
	Depemokimab 100 mg SC	Placebo	Depemokimab 100 mg SC	Placebo	Depemokimab 100 mg SC	Placebo	Depemokimab 100 mg SC	Placebo
n	501	260	497	257	486	255	477	247
Annualized exacerbation rate (95% CI)	0.46 (0.36-0.60)	1.15 (0.91-1.47)	0.55 (0.42-0.70)	1.17 (0.90-1.51)	0.46 (0.34-0.61)	1.19 (0.90-1.57)	0.58 (0.45-0.75)	0.87 (0.65-1.17)
Depemokimab vs placebo rate ratio (95% CI)	0.40 (0.29-0.56)		0.47 (0.33-0.66)		0.39 (0.26-0.57)		0.67 (0.46-0.97)	

SC, subcutaneous.