



# Inhaled anti-TSLP antibody fragment, ecleralimab, blocks responses to allergen in mild asthma

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**Ecleralimab significantly attenuated allergen-induced airway responses and inflammation in subjects with mild asthma. It was generally safe and well tolerated, suggesting anti-TSLP may be a promising, new therapeutic class for inhaled asthma treatment.** <https://bit.ly/3U294EA>

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## Abstract

**Background** Thymic stromal lymphopoietin (TSLP) is a key upstream regulator driving allergic inflammatory responses. We evaluated the efficacy and safety of ecleralimab, a potent inhaled neutralising antibody fragment against human TSLP, using allergen inhalation challenge (AIC) in subjects with mild atopic asthma.

**Methods** This was a 12-week, randomised, double-blind, placebo-controlled, parallel-design, multicentre allergen bronchoprovocation study conducted at 10 centres across Canada and Germany. Subjects aged 18–60 years with stable mild atopic asthma were randomised (1:1) to receive 4 mg once-daily inhaled ecleralimab or placebo. Primary end-points were the allergen-induced change in forced expiratory volume in 1 s ( $FEV_{1}$ ) during the late asthmatic response (LAR) measured by area under the curve ( $AUC_{3-7h}$ ) and maximum percentage decrease (LAR%) on day 84, and the safety of ecleralimab. Allergen-induced early asthmatic response (EAR), sputum eosinophils and fractional exhaled nitric oxide ( $F_{ENO}$ ) were secondary and exploratory end-points.

**Results** 28 subjects were randomised to ecleralimab (n=15) or placebo (n=13). On day 84, ecleralimab significantly attenuated LAR  $AUC_{3-7h}$  by 64% (p=0.008), LAR% by 48% (p=0.029), and allergen-induced sputum eosinophils by 64% at 7 h (p=0.011) and by 52% at 24 h (p=0.047) post-challenge. Ecleralimab also numerically reduced EAR  $AUC_{0-2h}$  (p=0.097) and EAR% (p=0.105).  $F_{ENO}$  levels were significantly reduced from baseline throughout the study (p<0.05), except at 24 h post-allergen (day 43 and day 85). Overall, ecleralimab was safe and well tolerated.

**Conclusion** Ecleralimab significantly attenuated allergen-induced bronchoconstriction and airway inflammation, and was safe in subjects with mild atopic asthma.

## Introduction

Allergic asthma is characterised by eosinophilic inflammation and evidence of atopy, where T-helper type 2 immune pathway elements play an important role in the development and maintenance of airway





The study was approved by the independent ethics committees at participating centres in accordance with the International Conference on Harmonisation Guidelines for Good Clinical Practice and the Declaration of Helsinki. All subjects provided written informed consent to participate in the study.

### Subjects

Male and female subjects aged 18–60 years with stable mild atopic asthma requiring no asthma medications except short-acting  $\beta_2$ -agonists less than twice weekly for treatment of symptoms were recruited. Subjects were required to have forced expiratory volume in 1 s ( $FEV_1$ )  $\geq 70\%$  predicted [20], methacholine provocation concentration causing a 20% decrease in  $FEV_1$  ( $PC_{20}$ )  $\leq 16 \text{ mg}\cdot\text{mL}^{-1}$  during the screening methacholine inhalation challenge (MIC), positive skin prick test to common aeroallergens, and positive early asthmatic response (EAR; at least 20% decrease in  $FEV_1$  within 2 h of allergen inhalation) and positive late asthmatic response (LAR; at least 15% decrease in  $FEV_1$  between 3 and 7 h after inhalation) to a common inhaled allergen during the screening AIC.

Subjects were excluded if they required hospitalisation or emergency department treatment for asthma within the last 6 months, or if they had a history of clinically significant comorbidities or clinically significant chronic lung disease other than mild atopic asthma.

### Randomisation and masking

Randomisation utilised interactive response technology (IRT) that assigned a randomisation number to the subject, which linked the subject to a treatment arm and specified a unique medication number to be dispensed. Randomisation codes, data on treatment and dosage allocation were tracked using IRT. Eligible subjects were randomly assigned to ecleralimab or placebo in Cohort 1 (1:1) and Cohort 2 (2:1).

Treatment allocations were concealed using identical packaging, labelling and schedule of administration and odour, and by the assignment of unblinded staff to assist subjects with investigational drug administration at study visits. No emergency unblinding took place during the study.

### Procedures

Ecleralimab is formulated as a PulmoSol engineered powder in hard capsules and delivered to the lungs via a Breezhaler dry powder inhaler device, all provided by Novartis (Basel, Switzerland). Based on clinical and nonclinical safety data, a starting dose of 4 mg administered once daily for 12 weeks was expected to provide adequate pulmonary exposure to assess pharmacodynamic effects against allergen-induced airway responses. Each randomised subject received a single inhaled dose of ecleralimab or placebo on day 1 at the investigational site. Subsequent daily dosing began on day 3 provided no safety concerns were identified in the 48 h following the initial dose. Dosing was self-administered during the morning at home or at the study site during scheduled visits up to and including day 84. MIC and AIC testing commenced at least 1 h after dosing. Compliance to study treatment was recorded by the subject in a diary and assessed at each visit using blister pack counts and the diary.

13 visits were scheduled during the 12-week treatment period, including two allergen challenge triads (MIC–AIC–MIC) at days 41–43 and days 83–85. Safety, pharmacokinetics and pharmacodynamics assessments were also performed. To enter the treatment period, subjects needed to demonstrate return to baseline with  $FEV_1 \geq 70\%$  predicted, and  $FEV_1$  and forced vital capacity were to be within 10% and the methacholine  $PC_{20}$  not more than 1 doubling concentration lower than the values measured at day –15 during screening.

MIC was conducted during screening (to qualify subjects for the study), pre-treatment on day 1, and 24 h pre-AIC and 24 h post-AIC, as previously described [21], until a 20% decrease in  $FEV_1$  occurred. The methacholine  $PC_{20}$  was calculated from the log concentration *versus* response curve.

AIC was conducted during screening and again at day 42 and day 84 as previously reported (see supplementary material) [22]. At screening, doubling concentrations of commercially available aero-allergen extracts (table 1) were inhaled at 12-min intervals until a  $\geq 20\%$  decrease in  $FEV_1$  was reached.  $FEV_1$  was then measured at regular intervals for 7 h to identify subjects with positive LAR. Allergen concentrations administered on days 42 and 84 were the same as those administered at screening. EAR and LAR were reported as time-adjusted area of percentage decrease in  $FEV_1$  (EAR  $AUC_{0-2h}$  and LAR  $AUC_{3-7h}$ ), maximum percentage decrease in  $FEV_1$  (EAR% and LAR%) and minimum  $FEV_1$  (EAR<sub>min</sub> and LAR<sub>min</sub>).

TABLE 1 Demographics and baseline characteristics (safety set)

	Ecleralimab 4 mg (n=15)	Placebo (n=13)
Age (years)	34.1±10.9	34.1±12.6
Female	8 (53)	9 (69)
Body mass index (kg·m <sup>-2</sup> )	26.8±3.9	23.7±2.3
FEV <sub>1</sub> (L)	3.4±0.6	3.3±0.5
FEV <sub>1</sub> (% pred)	91.0±10.6	92.1±15.5
Methacholine PC <sub>20</sub> (mg·mL <sup>-1</sup> ) <sup>#</sup>	1.41 (272)	1.37 (136)
F <sub>ENO</sub> (ppb) <sup>#</sup>	44.02 (89)	32.20 (72)
Blood eosinophils (×10 <sup>9</sup> L <sup>-1</sup> ) <sup>#</sup>	0.23 (91)	0.16 (80)
Sputum eosinophils (%) <sup>#</sup>	1.97 (101)	2.19 (135)
<b>Inhaled allergen</b>		
Animal dander	7 (46.6)	7 (53.8)
House dust mite	3 (20.0)	0 (0)
Grass	1 (6.7)	1 (7.7)
Tree	3 (20.0)	5 (38.5)
Crops	1 (6.7)	0 (0)

Data presented as mean±SD or n (%), unless otherwise stated. #: geometric mean (geometric coefficient of variation %). BMI: body mass index; FEV<sub>1</sub>: forced expiratory volume in 1 s; PC<sub>20</sub>: provocative concentration causing a 20% decrease in FEV<sub>1</sub>; F<sub>ENO</sub>: fractional exhaled nitric oxide.

Sputum was induced before the start of treatment on day 1, and during each allergen challenge triad at 24 h pre-allergen and at 7 and 24 h post-allergen, and processed using a method modified from PIZZICHINI *et al.* [23].

Fractional exhaled nitric oxide (F<sub>ENO</sub>) was sampled before each MIC, beginning on day -1 and also at 7 h post-allergen (prior to 7 h spirometry), using a Niox VERO (Aerocrine, Stockholm, Sweden) F<sub>ENO</sub> testing device. Peripheral blood eosinophils were also assessed.

Safety assessments included physical examinations, systems review, open-ended health inquiry, ECGs, vital signs, haematology, blood chemistry, urinalysis, and monitoring of adverse events (AEs) and serious AEs (SAEs). Subjects completed an end-of-treatment period visit on day 85 and entered a 4-week follow-up period.

### Outcomes

The primary end-points of this study included the allergen-induced LAR on day 84 assessed by LAR AUC<sub>3-7h</sub> and LAR%, and the safety and tolerability of daily ecleralimab administration assessed by AEs and SAEs.

Secondary end-points included day 42 and day 84 EAR AUC<sub>0-2h</sub>, EAR%, EAR<sub>min</sub> and LAR<sub>min</sub>, and day 42 LAR AUC<sub>3-7h</sub> and LAR%.

Exploratory end-points included sputum eosinophils, F<sub>ENO</sub>, methacholine PC<sub>20</sub> and blood eosinophil levels.

### Statistical analysis

The sample size was calculated considering 80% power to detect an absolute difference either of 5% between ecleralimab and placebo in LAR AUC<sub>3-7h</sub> at day 84 (assuming that the standard deviation was 7.7) or to detect an absolute difference of 8% between ecleralimab and placebo in LAR% (assuming that the standard deviation was 12.7) using a Hochberg procedure with an overall family-wise controlled type 1 error rate of 10% and assuming a correlation coefficient of 0.8 between the two end-points.

Two unblinded interim analyses (IA) were conducted. IA1 included 20 subjects from Cohort 1 (n=10 in ecleralimab and n=10 in placebo) to confirm the sample size assumptions while assessing the variability in time-adjusted area of percentage decrease in FEV<sub>1</sub> AUC<sub>3-7h</sub> and maximum percentage decrease in FEV<sub>1</sub> following the AIC in this population. These data were not intended to change the conduct of this study and were not used to stop the study at this time, but could have informed a change in sample size. No pause in enrolment, pending the data from IA1, was required. IA2 included 28 subjects from Cohorts 1 and 2 (n=15

in ecleralimab and  $n=13$  in placebo), and was conducted when 26 subjects had completed day 84. This analysis showed that there were no safety issues, and that the variability for AUC and maximum percentage decrease was slightly less than assumed (the correlation between these was 0.95). The study was terminated when 27 subjects completed the study. As the study met the primary end-point with the 4-mg dose, a decision was made to cancel the planned dose escalation phase.

The efficacy variables were analysed using the efficacy set, which included all subjects who received any study drug and experienced no protocol deviations with relevant impact on efficacy end-points. The two primary end-points, LAR AUC<sub>3–7h</sub> and LAR%, compared with placebo were analysed separately using an ANCOVA model for repeated measures. The model included treatment and visit as independent variables, treatment by visit interaction term, and baseline by visit interaction term where baseline values for time-adjusted area of percentage decrease in FEV<sub>1</sub> AUC<sub>3–7h</sub> and maximum percentage decrease in FEV<sub>1</sub> are measured from the AIC at day –14 during the screening period. Model-based estimates of mean (least square estimates) were reported for ecleralimab and placebo groups on day 42 and day 84 along with the treatment difference, two-sided 90% confidence interval for treatment difference and one-sided p-value.

The secondary efficacy end-points were listed by treatment, subjects and visit/time.

EAR AUC<sub>0–2h</sub>, EAR%, EAR<sub>min</sub> and LAR<sub>min</sub> were also analysed using an ANCOVA model for repeated measures. Baseline values for all secondary efficacy end-points were measured from the screening AIC at day –14.

The exploratory efficacy end-points were listed by treatment, subjects and visit/time, and descriptive statistics were provided by treatment and visit/time, as needed. Methacholine PC<sub>20</sub> and  $F_{ENO}$  data were log transformed prior to analysis. Comparisons of ecleralimab with placebo on change from baseline at day 41 and day 83 and allergen-induced shift (change from pre-challenge (day 42/43 to day 41 and day 84/85 to day 83) at 7 and 24 h post-challenge) were carried out using an ANCOVA model for repeated measures for sputum eosinophils,  $F_{ENO}$  and methacholine PC<sub>20</sub> (24 h post-challenge). Blood eosinophil levels were compared on change from baseline at day 43 and day 71.

The safety analysis set included all subjects who received any study drug. The primary variable for the primary safety end-point was the occurrence of AEs/SAEs. The numbers and percentages of subjects with AEs by maximum severity of AEs were tabulated by body system and MedDRA preferred term with a breakdown by treatment. All the analyses were performed using SAS version 9.4 (SAS Institute, Cary, NC, USA).

## Results

### *Subject disposition and demographics*

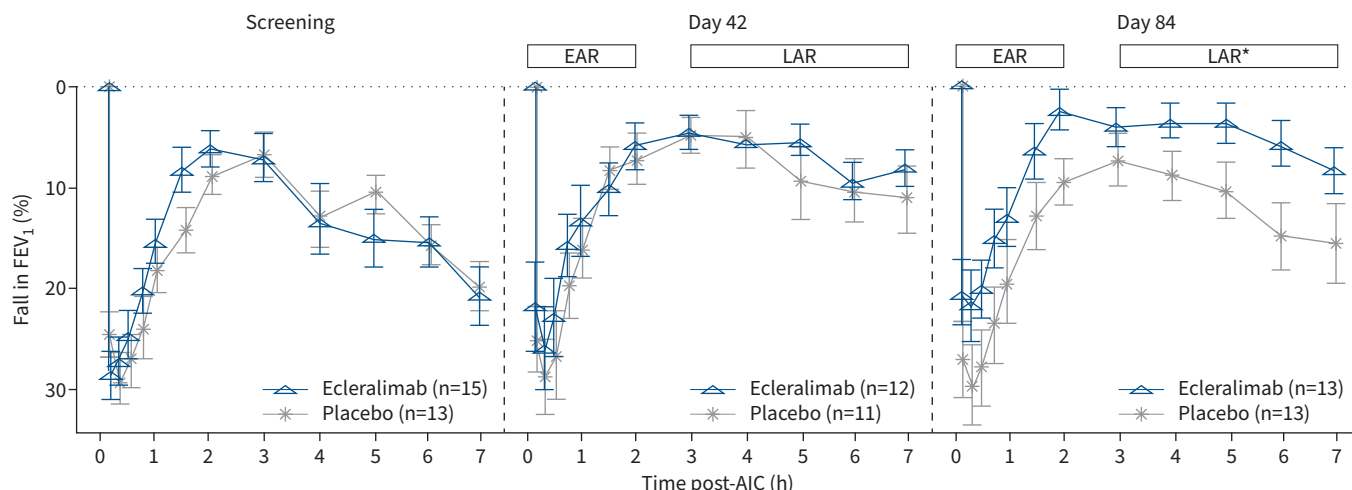
A total of 28 subjects were enrolled in the study (15 received ecleralimab and 13 received placebo) and 27 subjects (96.4%) completed the study; one subject in the ecleralimab group was discontinued from the study due to unstable asthma prior to day 84 allergen challenge (supplementary figure S1).

The subject demographics and baseline characteristics were similar between the groups (table 1).

The two treatment groups developed similar EAR and LAR at the screening allergen challenge. Three subjects ( $n=2$  in ecleralimab and  $n=1$  in placebo) were excluded from the LAR and EAR analyses in the first challenge (day 42), with one ecleralimab subject also excluded in the second challenge (day 84) due to incorrect dosing of allergen; however, a sensitivity analyses including the data demonstrated no change from the reported results. These subjects were not excluded from the methacholine PC<sub>20</sub> and  $F_{ENO}$  analyses.

### *Early and late asthmatic responses*

On day 84, ecleralimab significantly attenuated LAR AUC<sub>3–7h</sub>, being 4.2% in the ecleralimab group and 11.38% in the placebo group, representing a 64% reduction (treatment difference ( $\Delta$ ):  $-7.18\%$  (90% CI  $-11.92$ – $-2.44\%$ );  $p=0.008$ ). Similarly, ecleralimab significantly attenuated LAR%, being 9.3% in the ecleralimab group and 17.7% in the placebo group, demonstrating a 48% reduction ( $\Delta$ :  $-8.42\%$  (90% CI  $-15.66$ – $-1.18\%$ );  $p=0.029$ ). In addition, LAR<sub>min</sub> was significantly higher in the ecleralimab group compared with the placebo group, being 2.96 versus 2.69 L, respectively, on day 84 ( $\Delta$ : 0.27 L (90% CI  $-0.00$ – $0.55$  L);  $p=0.050$ ). Small, nonsignificant reductions in LAR responses were found at day 42 for LAR AUC<sub>3–7h</sub> ( $\Delta$ :  $-2.90\%$  (90% CI  $-7.43$ – $1.62\%$ );  $p=0.141$ ), LAR% ( $\Delta$ :  $-2.64\%$  (90% CI  $-9.02$ – $3.74\%$ );  $p=0.243$ ) and LAR<sub>min</sub> ( $\Delta$ : 0.09 L (90% CI  $-0.19$ – $0.37$  L);  $p=0.293$ ) (figure 2).

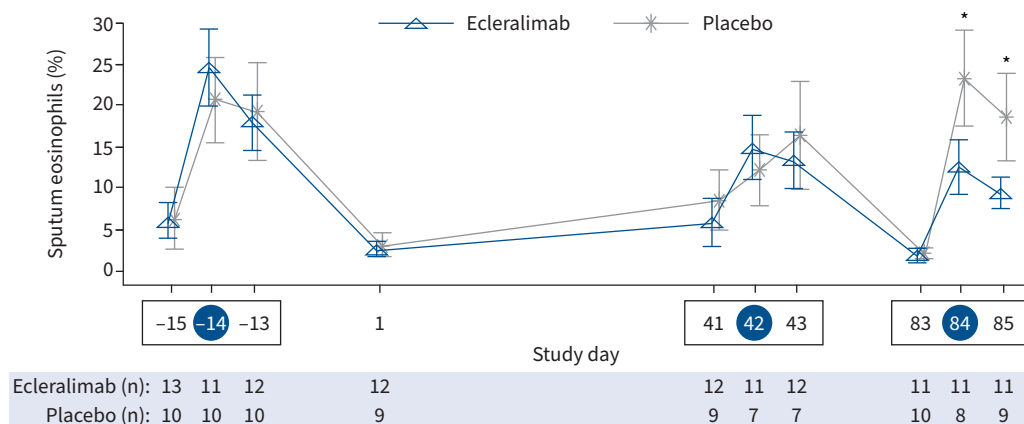


**FIGURE 2** Effect of ecleralimab on allergen-induced early asthmatic response (EAR) and late asthmatic response (LAR) after days 42 and 84 of treatment. Data are presented as mean±se. FEV<sub>1</sub>: forced expiratory volume in 1 s; AIC: allergen inhalation challenge. \*: p<0.05 significant difference between ecleralimab and placebo.

A numerical attenuation of the EAR was observed in subjects treated with ecleralimab at day 42 and day 84, but was not statistically different compared with placebo (supplementary table S1).

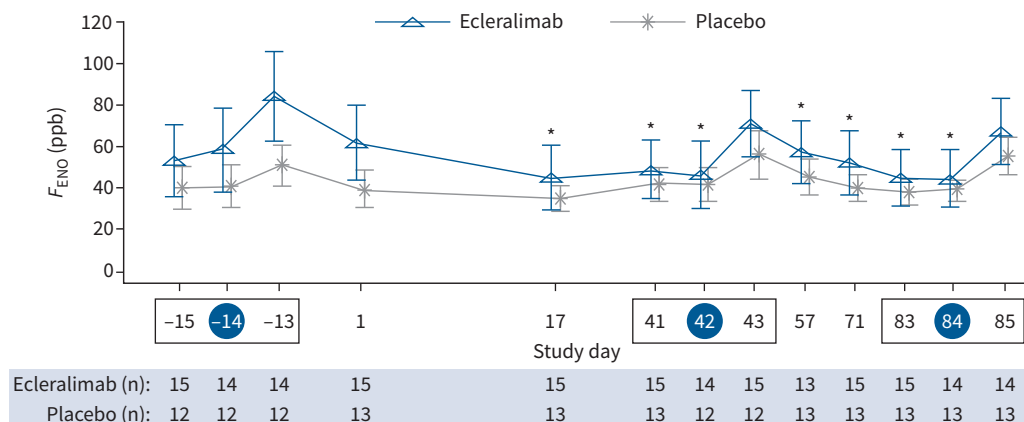
**Allergen-induced airway inflammation**

Ecleralimab significantly attenuated allergen-induced sputum eosinophils at 7 h (8.44% ecleralimab versus 23.19% placebo; Δ: -14.75% (90% CI -24.85– -4.65%); p=0.011) and 24 h (8.12% ecleralimab versus 16.84% placebo; Δ: -8.72% (90% CI -17.29– -0.15%); p=0.047) after day 84 AIC, corresponding to a 64% and 52% reduction, respectively, compared with placebo (figure 3). Compared with placebo there was no effect of ecleralimab on pre-allergen sputum eosinophil levels on day 41 (2.46% ecleralimab versus 6.14% placebo; Δ: -3.68% (90% CI -12.19–4.83%); p=0.231) or day 83 (-1.88% ecleralimab versus -1.32% placebo; Δ: -0.56% (90% CI -2.16–1.05%); p=0.27). There was a significant improvement from baseline in F<sub>ENO</sub> by day 17 with ecleralimab compared with placebo (ratio to baseline: 0.73 versus 0.93) corresponding to a 27% versus 7% reduction (Δ: 0.78 (90% CI 0.63–0.97); p=0.031). Over the 12-week treatment period there were significant reductions in F<sub>ENO</sub> (ppb) with ecleralimab (p<0.05), except at day 43 and day 85 at 24 h post-allergen (figure 4). There was no effect of ecleralimab on allergen-induced shift in F<sub>ENO</sub> after either AIC.



**FIGURE 3** Effect of ecleralimab on sputum eosinophils before and after allergen challenge over 85 days. A blue circle around the study day indicates allergen inhalation challenge (AIC) and a box indicates an allergen challenge triad. Data are presented as mean±se. \*: p<0.05 significant difference between ecleralimab and placebo.





**FIGURE 4** Effect of ecleralimab on fractional exhaled nitric oxide ( $F_{ENO}$ ) change from baseline compared with placebo over 85 days. A blue circle around the study day indicates allergen inhalation challenge (AIC) and a box indicates an allergen challenge triad. Data are presented as mean $\pm$ SE. \*:  $p < 0.05$  significant difference in change from baseline in ecleralimab versus change from baseline in placebo.

**Airway hyperresponsiveness**

There was a numerical increase in the methacholine  $PC_{20}$  on day 41 and day 83 in ecleralimab compared with placebo. Ecleralimab was better than placebo by 0.49 doubling doses on day 41 ( $\Delta$ : 0.49 (90% CI -0.39-1.38) doubling doses;  $p=0.173$ ) and by 0.18 doubling dose on day 83 ( $\Delta$ : 0.18 (90% CI -0.54-0.90) doubling doses;  $p=0.335$ ). There was no effect of ecleralimab on allergen-induced changes in methacholine  $PC_{20}$  measured on days 43 (day 43 to day 41) and 85 (day 85 to day 83). Moreover, there was no difference in peripheral blood eosinophil counts in subjects receiving ecleralimab compared with those receiving placebo.

**Safety**

A total of 22 subjects (78.6%) reported 93 treatment-emergent AEs: 51 AEs were reported in 10 subjects (66.7%) who received ecleralimab and 42 AEs were reported in 12 subjects (92.3%) who received placebo. The majority of AEs (75.0%) in both treatment groups were assessed as mild (table 2). The number, incidence and severity of treatment-emergent AEs were comparable between the treatment groups. The most frequent AEs were headache (25.0%), nasopharyngitis (17.9%) and oropharyngeal pain (17.9%) (supplementary table S2). No severe or serious AEs or deaths were reported. No subject discontinued study drug due to an AE. No clinically relevant changes were noted in the haematology, chemistry, urinalysis, ECG and spirometry results.

**Discussion**

Treatment with ecleralimab, the first inhaled anti-TSLP molecule, was shown to be safe and well tolerated in subjects with mild atopic asthma. Ecleralimab significantly attenuated the allergen-induced LAR on day

	Ecleralimab 4 mg (n=15)		Placebo (n=13)	
	$n_E$	$n_S$ (%)	$n_E$	$n_S$ (%)
Total AEs	51	10 (66.7)	42	12 (92.3)
AEs of mild intensity	40	10 (66.7)	30	11 (84.6)
AEs of moderate intensity	11	6 (40.0)	12	8 (61.5)
AEs of severe intensity	0		0	
Study drug-related AEs	4	4 (26.7)	0	
Serious AEs	0		0	
AEs leading to discontinuation of study treatment	0		0	
Study drug-related AEs leading to discontinuation of study treatment	0		0	

Percentage is based on the number of subjects. Only AEs occurring at or after first drug intake are included.  $n_E$ : number of AEs in the category;  $n_S$ : number of subjects with at least one AE in the category.

84, similar to that by *i.v.* anti-TSLP monoclonal antibody tezepelumab in a similar study [14]. Ecleralimab also reduced  $F_{\text{ENO}}$  after 17 days and allergen-induced sputum eosinophil levels after 3 months of dosing.

Emerging evidence suggests targeting TSLP may provide clinical benefit in asthma [15, 16] by reducing the inflammatory response to allergens [14]. In subjects with asthma, an increase in TSLP protein levels has been observed in both lung tissue and bronchoalveolar lavage fluid, and TSLP levels correlate with disease severity [11]. TSLP is thought to cause airway and blood eosinophilia among subjects with allergic asthma by activating airway dendritic cells and by polarising naïve T-cells to T-helper type 2 cells to produce pro-inflammatory cytokines, including interleukin (IL)-5 and IL-13 [24]. The significant inhibition of allergen-induced sputum eosinophils with ecleralimab on days 84 and 85 demonstrates that inhaled anti-TSLP  $F_{\text{ab}}$  reduces allergen-induced airway inflammation in subjects with allergic asthma. There was no change in peripheral blood eosinophil counts in subjects receiving ecleralimab, which was expected due to the inhaled delivery.

The modest but statistically significant reduction of  $F_{\text{ENO}}$  levels suggests there is a constitutive release of TSLP from the airways of these subjects under baseline conditions [25, 26]. Furthermore, the reduction in allergen-induced eosinophils by ecleralimab indicates that TSLP, which is critical for maintaining eosinophilic airway inflammation, is effectively targeted with this inhaled formulation.

Methacholine challenge is a diagnostic test to evaluate the presence/severity of AHR consistent with a diagnosis of asthma including asthma control. While systemic anti-TSLP therapy has been reported to reduce baseline and allergen-induced eosinophilia in blood and airways in subjects, resulting in some improvements in AHR [14, 27], inhaled ecleralimab numerically improved baseline methacholine  $PC_{20}$  at days 41 and 83 by 0.49 and 0.18 doubling doses, respectively. In this same population, tezepelumab improved baseline methacholine  $PC_{20}$  at days 41 and 83 by 1.24 and 0.66 doubling doses, respectively [14]. Inhaled corticosteroid treatment over approximately the same period of time is reported to shift methacholine  $PC_{20}$  by 0.4–1.4 doubling doses [28]. Neither ecleralimab nor tezepelumab improved allergen-induced changes in methacholine  $PC_{20}$ .

Ecleralimab inhaled at a dose of 4 mg once daily had no effect on EAR at day 42 and 84 allergen challenges, and no effect on LAR or sputum eosinophils at day 42 allergen challenge. The main reason for no effect on LAR at day 42 is due to higher than expected variability in two placebo outliers with significant improvement; when excluded, the LAR profiles look more similar to day 84. In contrast, in a separate study, treatment with systemic anti-TSLP therapy (700 mg *i.v.* every 4 weeks for 12 weeks) significantly inhibited EAR, LAR and sputum eosinophils at day 42 and 84 allergen challenges [14]. Collectively the results suggest at the doses studied there may be additional and/or more rapid blockade of cellular targets with systemic therapy. A higher dose of ecleralimab may have improved efficacy on these primary and secondary end-points; however, this was not studied because the dose was not escalated.

Ecleralimab was generally safe and well tolerated by the mild asthmatic subjects enrolled in this study. The study was terminated to proceed to development in severe asthmatic subjects, the intended population, rather than obtaining more data in mild asthmatic subjects and the decision for early termination was not based on any safety concerns regarding ecleralimab.

Limitations include the fact that the study was not powered to detect changes in secondary end-points such as methacholine  $PC_{20}$ . We did observe a 0.49 doubling dose improvement in AHR; however, this change did not meet statistical significance. Although LAR at day 42 was numerically improved by ecleralimab, the lack of statistical significance is likely due to a lower sample size (only 12 received ecleralimab and 11 received placebo) and influenced by a marked placebo effect; the magnitude of LAR at day 42 AIC is considerably smaller than that at day -14 in the placebo group. No information on dose response could be obtained because of early termination of the study.

Ecleralimab, an inhaled  $F_{\text{ab}}$ , reduced both allergen-induced late-phase bronchoconstriction and sputum eosinophilia, and was well tolerated. These findings demonstrate that ecleralimab is efficacious for attenuating allergen-induced responses in atopic asthmatic subjects.

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This study is registered at ClinicalTrials.gov with identifier number NCT03138811. Data sharing: Novartis is committed to sharing with qualified external researchers, access to patient-level data and supporting clinical documents from eligible studies. These requests are reviewed and approved by an independent review panel on the basis of scientific merit. All data provided are anonymised to respect the privacy of subjects who have participated in the trial in line with applicable laws and regulations. Result summaries have been posted on the Novartis clinical trial database and other online public databases. More information on Novartis' position on access to clinical trial results and patient-level data is available at: [www.novartis.com/our-science/clinical-trials/clinical-trial-information-disclosure](http://www.novartis.com/our-science/clinical-trials/clinical-trial-information-disclosure)

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