AstraZeneca
What science can do

Investor science webcast: ERS 2017

Conference call and webcast for investors and analysts, Milan, Italy

13 September 2017

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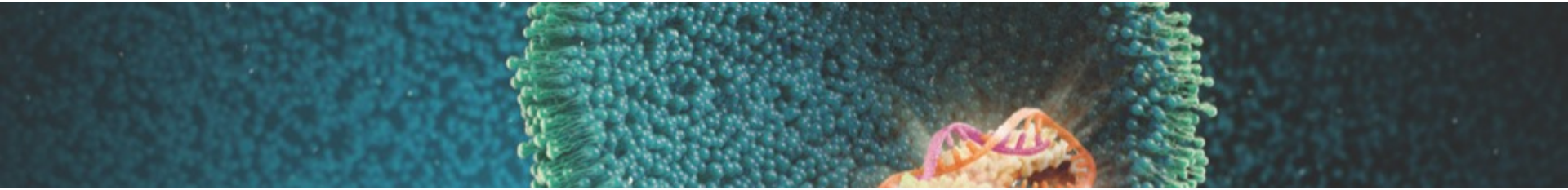
Presenters

Dr Colin Reisner
Chief Executive Officer, Pearl Therapeutics and Head of Clinical, Respiratory, Global Medicines Development, AstraZeneca

Dr Andrew Menzies-Gow
Consultant in Respiratory Medicine and Director, Lung Division, Royal Brompton Hospital, London, UK



Dr Jonathan Corren
David Geffen School of Medicine, UCLA and Principal Investigator of the PATHWAY trial

Tom Keith-Roach
Head of Respiratory, Global Product & Portfolio Strategy, AstraZeneca

Agenda

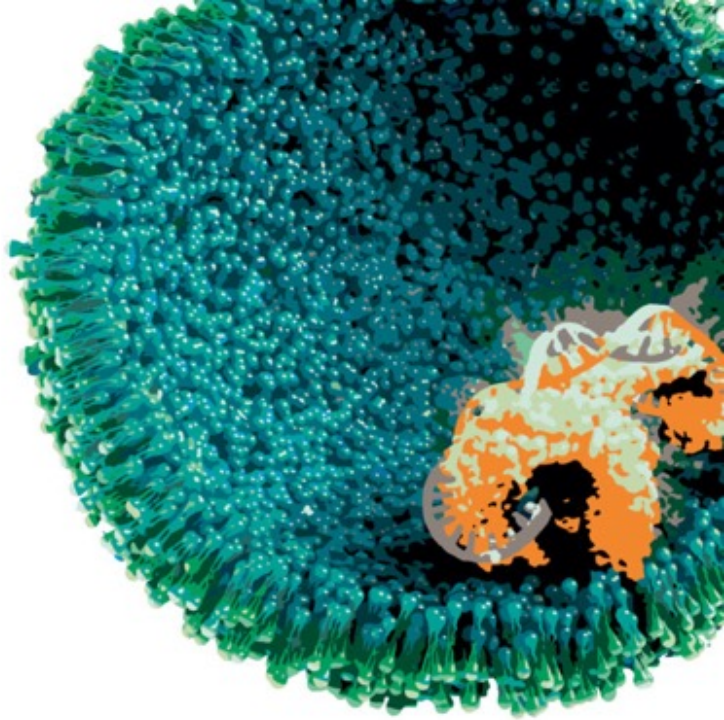
Introduction

Unmet medical need

Characterising responders to benralizumab

Tezepelumab Phase IIb PATHWAY

Commercial opportunity



Late-stage pipeline overview

Significant opportunities from lifecycle and potential new medicines

Oncology

- ***Lynparza***[1, 2] multiple cancers
- ***Tagrisso***[1, 2] lung cancer
- ***Imfinzi***[1, 2] multiple cancers
- **acalabrutinib**[2] blood cancers
- ***Imfinzi* + treme** multiple cancers
- **moxetumomab** leukaemia
- **selumetinib** thyroid cancer
- **savolitinib** kidney cancer

Cardiovascular & Metabolic Diseases

- **ZS-9**[2] hyperkalaemia
- **roxadustat**[2] anaemia

Other

- **anifrolumab** lupus
- **lanabecestat** Alzheimer's disease

Respiratory

- **benralizumab**[1, 2] severe, uncontrolled asthma / COPD
- **tralokinumab** severe, uncontrolled asthma
- **PT010** COPD / asthma

Phase IIb data at ERS 2017:

- **tezepelumab** severe asthma

1. Lifecycle development programme.
2. Under regulatory review.

Status as of 13 September 2017.

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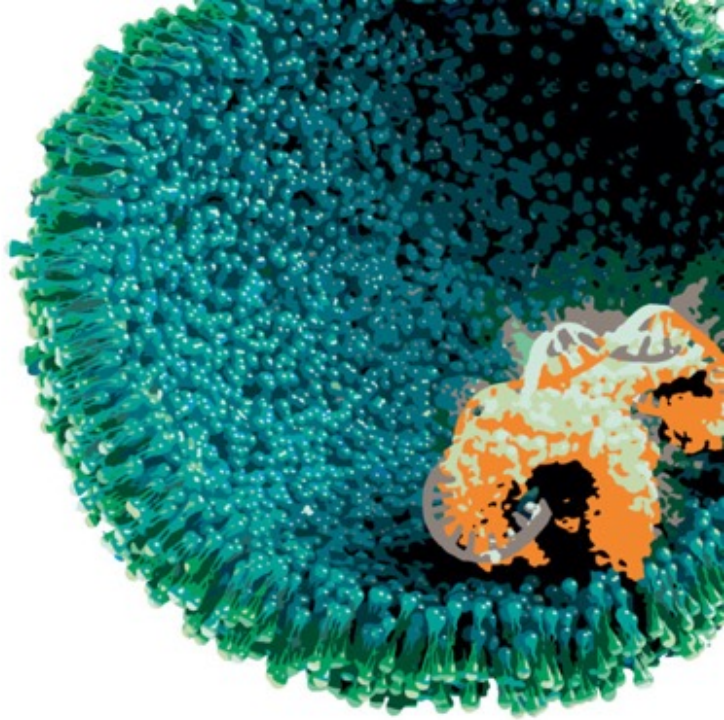


Asthma that is inadequately controlled by high-dose ICS-based therapy represents a significant healthcare burden

Asthma varies in disease severity[1,2,3]

315 million people
suffer from asthma worldwide

~1 in 10 people
with asthma have severe asthma, requiring:

High-dose ICS-based therapy

Other asthma medications

Linked to poor outcomes and medical emergencies[4,5]

In patients with uncontrolled asthma:

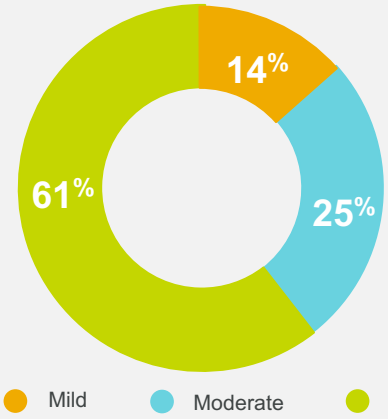
91% have normal daily activities impacted at least once per week

In patients with uncontrolled severe asthma*:

Higher risk of death and **10X** higher risk of hospital stays

Severe asthma accounts for majority of asthma costs[6]

Share (%) of total direct cost of asthma for different levels of severity



* Compares severe uncontrolled asthma with severe controlled asthma

ICS, inhaled corticosteroids

1. Chung KF et al. Eur Respir J. 2014 Feb;43(2):343-73. 2. To T et al. BioMed Central Public Health. 2012: 12(204). 3. Hekking PPW et al. J Allergy Clin Immun. 2015:135(4):896-902. 4. Price D et al. NPJ Prim Care Respir Med 2014; 12; 24: 14009. 5. Fernandes AG et al, J Bras Pneumol. 2014; 40(4): 364-372. 6. Sadatsafavi M et al. Can Respir J 2010; 17: 74-80

Glucocorticoids-associated side effects increased in severe asthma with high OCS use

Cross-section of OPCRD database and BTS difficult asthma registry

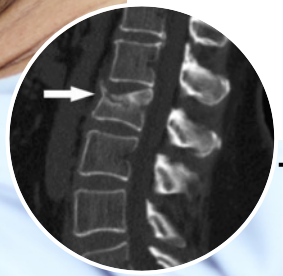
Severe asthma (GINA 5 ≥ 4 OCS burst) vs. mild/moderate asthma

Cataracts
OR 1.89, p<0.001

Gastrointestinal
OR 3.99, p<0.001

Osteoporosis
OR 5.23, p<0.001

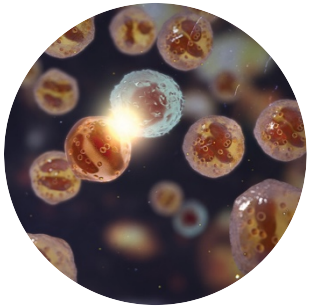
Diabetes
OR 1.46, p<0.01



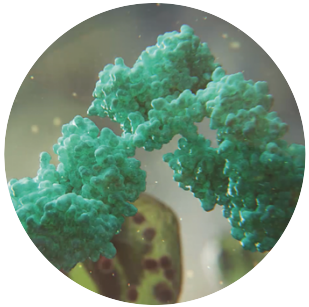
Spinal compression related to systemic corticosteroid use[2]

1. Sweeney J, et al. Thorax 2016;71:339–346.
2. Miller J. Radiology Rounds. 2009;7:1-4.
OR = odds ratio, OCS = oral corticosteroids, BTS = British Thoracic Society Severe Asthma Registry.

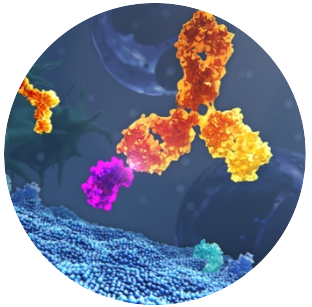
A biologics portfolio that follows the science



Benralizumab is an anti-eosinophil monoclonal antibody that targets the IL-5 receptor, thereby inducing direct and near complete depletion of eosinophils via antibody-dependent cell mediated cytotoxicity



Tralokinumab is an anti-IL-13 monoclonal antibody that blocks binding and signalling of IL-13 to IL-13 receptors



Tezepelumab is a potential first in class monoclonal antibody that specifically binds to TSLP, an upstream epithelial master switch that drives multiple downstream inflammatory pathways

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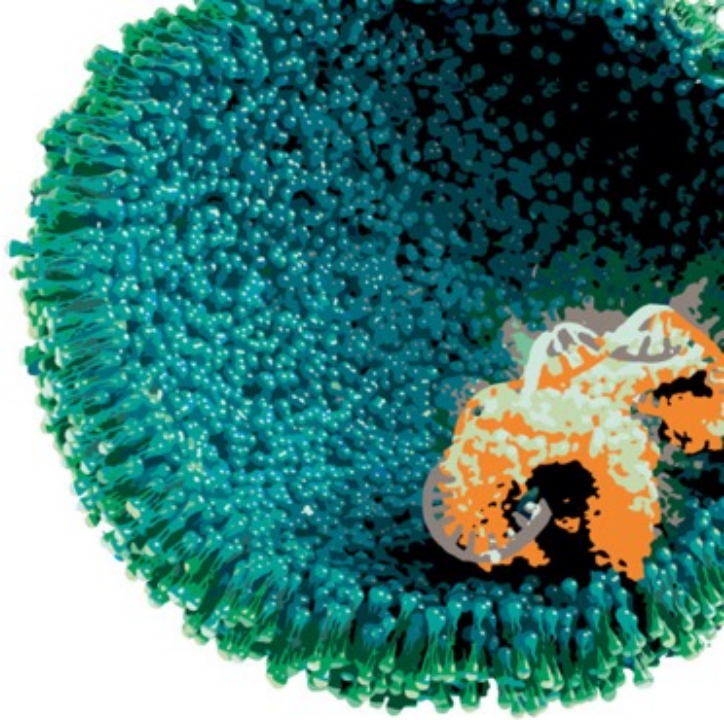

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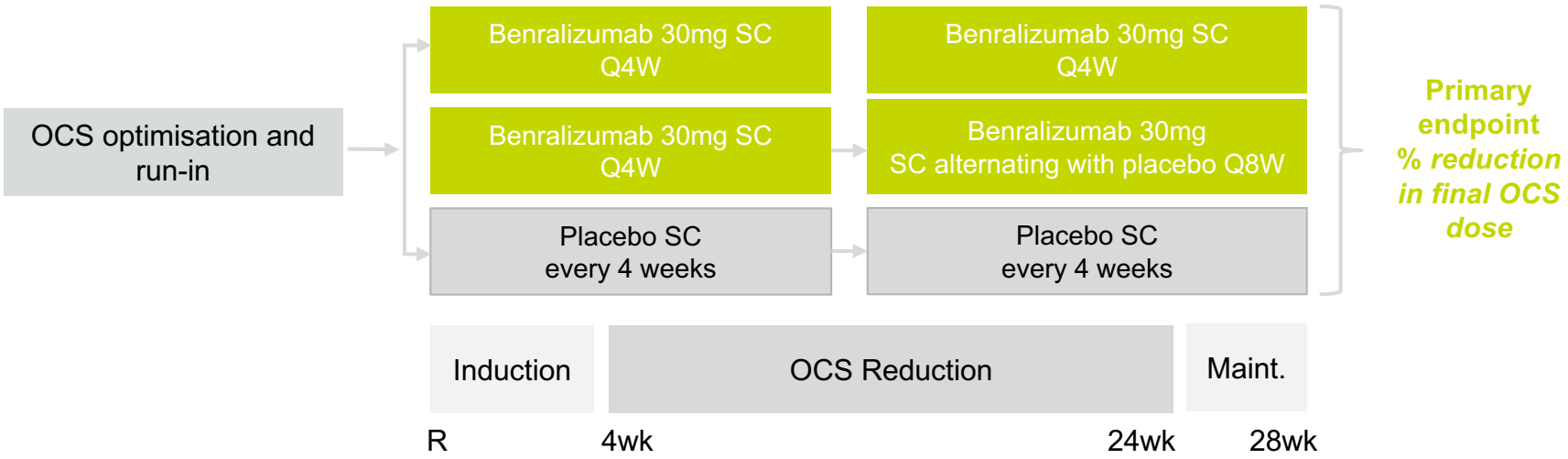
What science can do

Benralizumab for uncontrolled, severe asthma (ZONDA, SIROCCO and CALIMA)

ZONDA: OCS sparing trial in adult OCS-dependent asthma patients



Key inclusion: High dose ICS LABA + Chronic OCS requirement, age ≥ 12

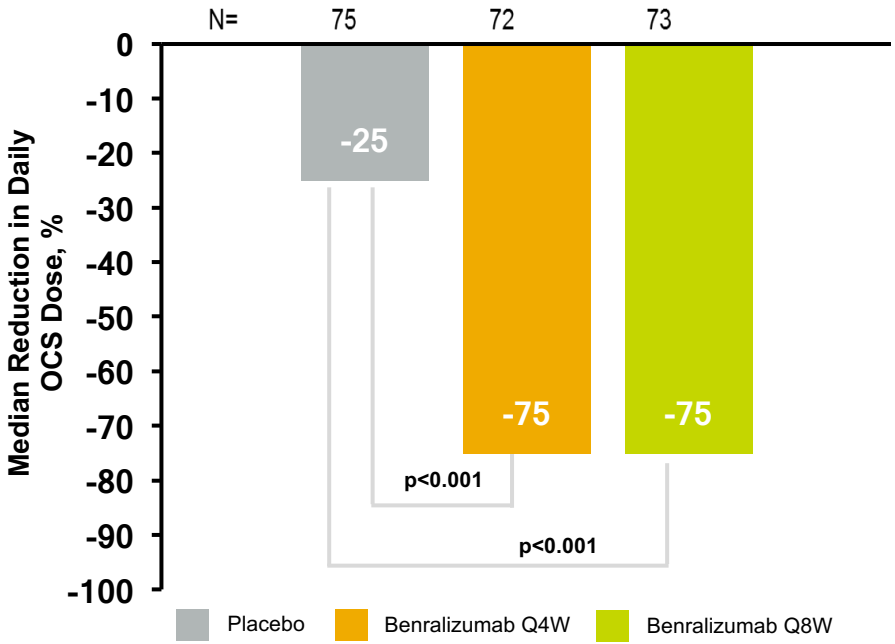
- ≥1 historical EXAC, FEV_1 < 80% pred
- Reversible to BD
- EOS ≥150 cells/µL

EOT = end of treatment; IP = investigational product; LABA = long-acting beta-agonist; OCS = oral corticosteroid; SC = subcutaneous; Tx = treatment; V = visit.

Nair P et al New Eng J Med 2017

ZONDA: Benralizumab significantly reduced final OCS doses at week 28 while maintaining asthma control vs. placebo (full analysis set)

Primary Analysis



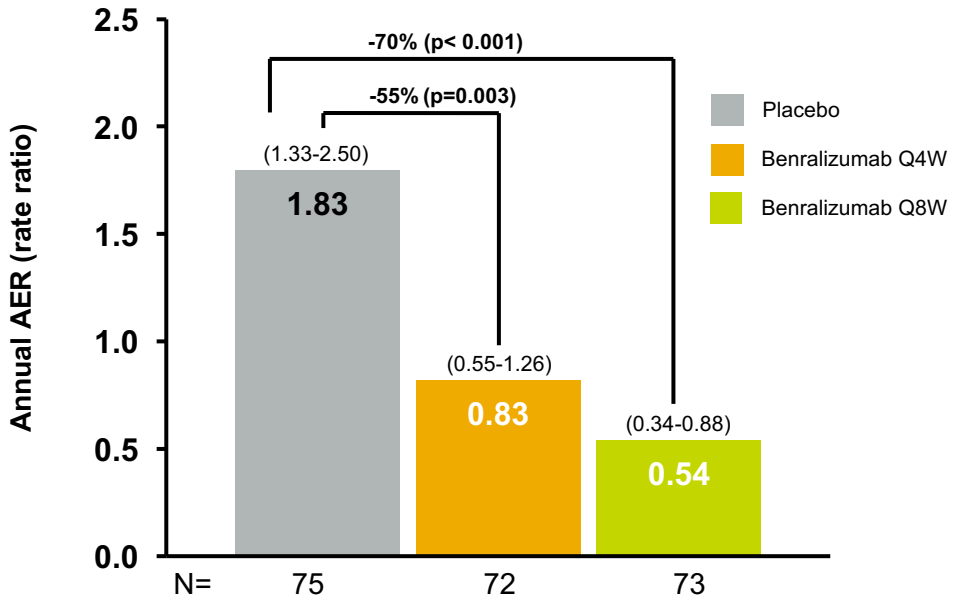
Categorical Analysis

Reduction in Final OCS Dose, n (%)	Placebo N=75	Benralizumab 30mg Q4W, N=72	Benralizumab 30mg Q8W, N=73
≥90%	9 (12)	24 (33)	27 (37)
≥75%	15 (20)	38 (53)	37 (51)
≥50%	28 (37)	48 (67)	48 (66)
>0%	40 (53)	55 (76)	58 (79)
No change or any increase in OCS dose	35 (47)	17 (24)	15 (21)
OR (95 %CI)	—	**4.09 (2.22 – 7.57)**	**4.12 (2.22 – 7.63)**
p	—	<0.001	<0.001

- The odds of a reduction in final OCS daily dose was 4X greater with benra vs. placebo
- median baseline OCS was 10 mg/d in all groups

CI = confidence interval; OCS = oral corticosteroid; OR = odds ratio; Q4W = every 4 weeks; Q8W = every 8 weeks.
Nair P et al New Eng J Med 2017

ZONDA: Benralizumab significantly reduced annualised asthma exacerbation rate, while reducing OCS doses at Week 28



Exacerbation definition (at least 1)[2]

- A temporary bolus/burst of systemic corticosteroids[a]
- An emergency room visit due to asthma that required systemic corticosteroids, or
- An inpatient hospitalisation due to asthma

Values above bars represent 95% CI.

[a]OCS burst should be at a dose at least one level higher than the current titration step

AER = asthma exacerbation rate; CI = confidence interval; OCS = oral corticosteroid dose; Q4W = every 4 weeks; Q8W = every 8 weeks.

1. Nair P et al. Supplementary appendix. *N Engl J Med*. 2017; 2. Nair P et al New Eng J Med 2017

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HEART+LUNG Health
Strong beats. Clear breaths. Full lives.

Characterising responders (enhanced efficacy) to benralizumab for severe asthma: pooled analysis of the SIROCCO and CALIMA studies

J. Mark FitzGerald[1], Eugene R. Bleecker[2], Andrew Menzies-Gow[3], James G. Zangrilli[4], Paul Metcalfe[5], Ian Hirsch[4], Paul Newbold[6], Mitchell Goldman[4]

[1]Centre for Heart and Lung Health, The Lung Centre, Vancouver General Hospital, UBC Institute for Heart and Lung Health, Vancouver, BC, Canada
[2]Center for Applied Genetics and Genomics Medicine, University of Arizona School of Medicine, Tucson, AZ, United States
[3]Department of Asthma and Allergy, Royal Brompton Hospital, London, United Kingdom
[4]AstraZeneca, Gaithersburg, MD, United States
[5]AstraZeneca, Cambridge, United Kingdom
[6]MedImmune LLC, Gaithersburg, MD, United States

Objectives

To determine the relationship between benralizumab's clinical efficacy versus baseline blood eosinophil counts and exacerbation history

To identify other intrinsic and/or extrinsic factors that might influence benralizumab's efficacy

Methods

- Post hoc analysis of pooled results for SIROCCO and CALIMA exacerbation studies for age ≥12 years with asthma uncontrolled using high-dosage ICS/LABA

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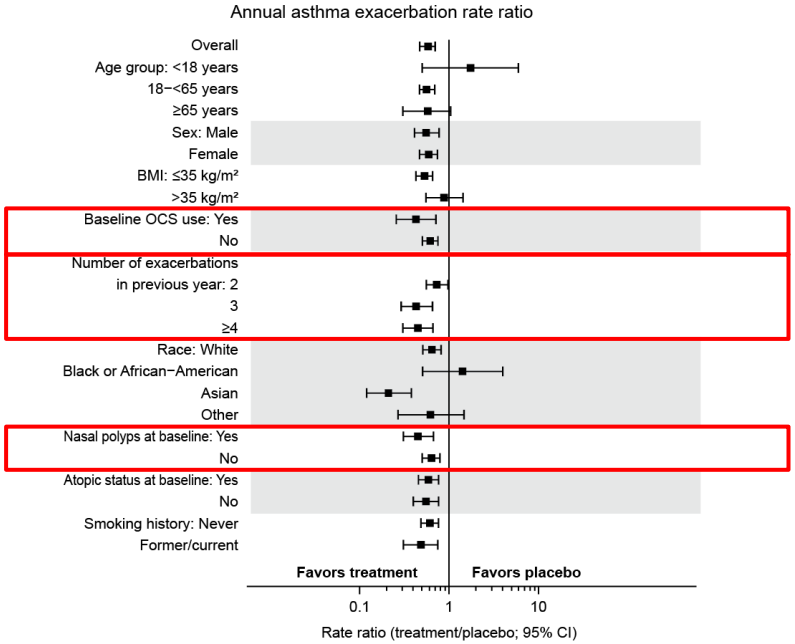
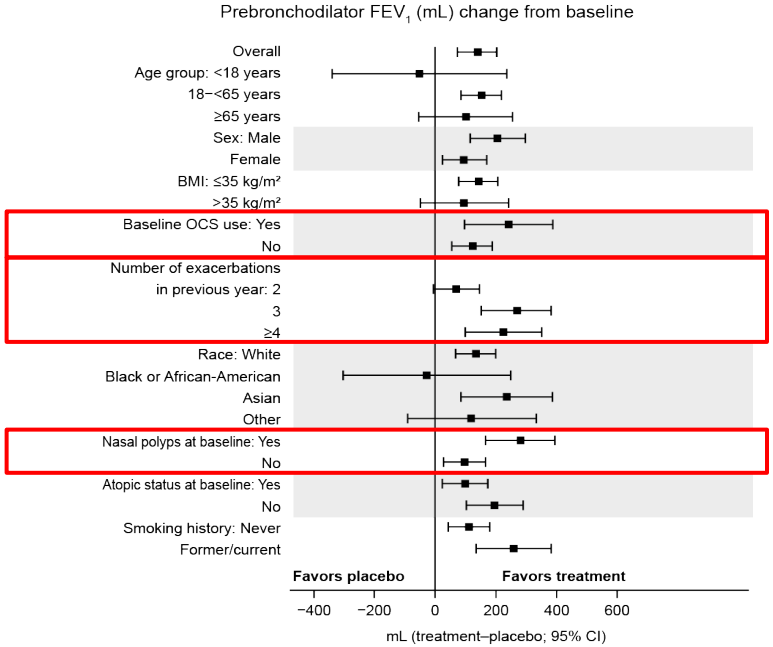
Subgroup analysis of benralizumab Q8W treatment AER and FEV_1 response (baseline blood eosinophils ≥300 cells/µL; full analysis set, pooled)





Larger AER reductions and FEV_1 improvements were associated with characteristic features of an eosinophilic phenotype including, exacerbation history, OCS usage, and history of nasal polyps

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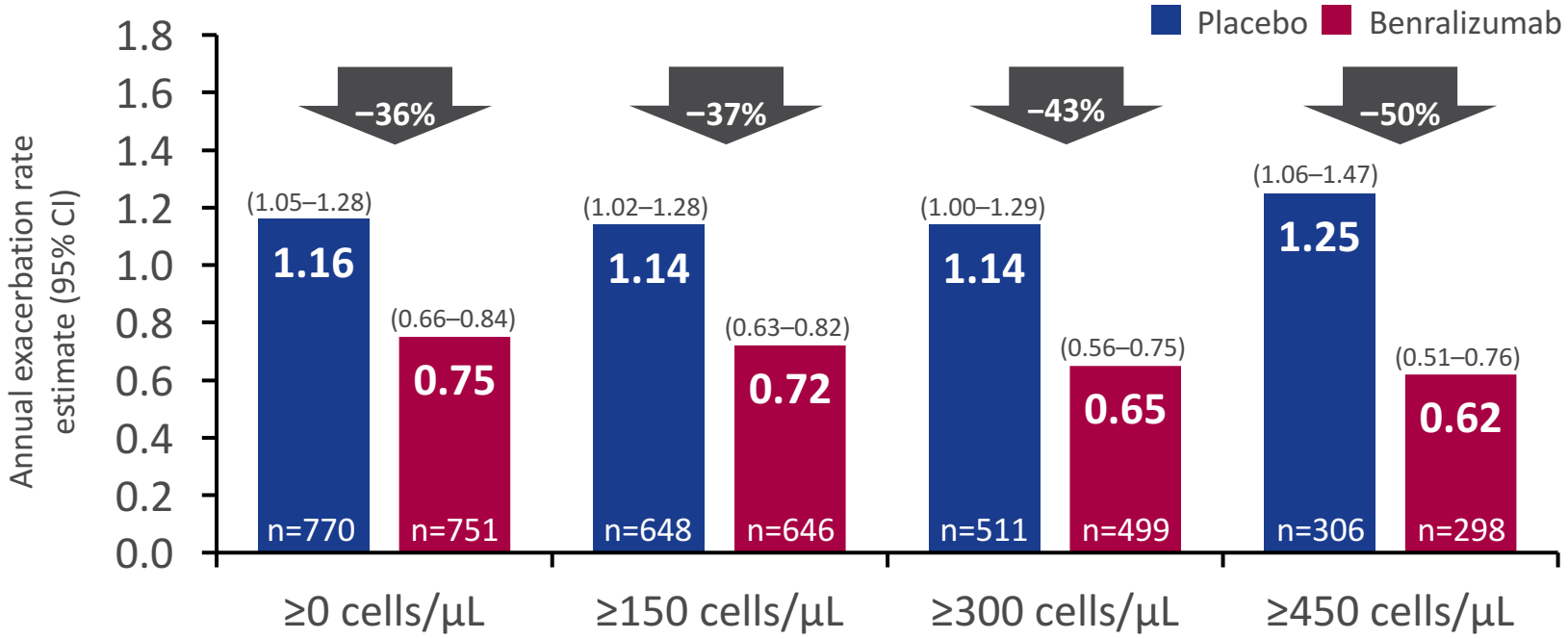
Annual asthma exacerbation rate reduction with benralizumab Q8W by eosinophil ranges (full analysis set, pooled)



Estimates calculated by a negative binomial model with adjustment for treatment, study code, region, oral corticosteroid use, and prior exacerbations.
The estimates were weighted to account for the 2:1 randomization ratio of patients with baseline blood eosinophil counts ≥300 cells/µL or <300 cells/µL.
CI: confidence interval; Q8W: every 8 weeks (first three doses every 4 weeks); $p < 0.0001$ for every EOS cut-off.

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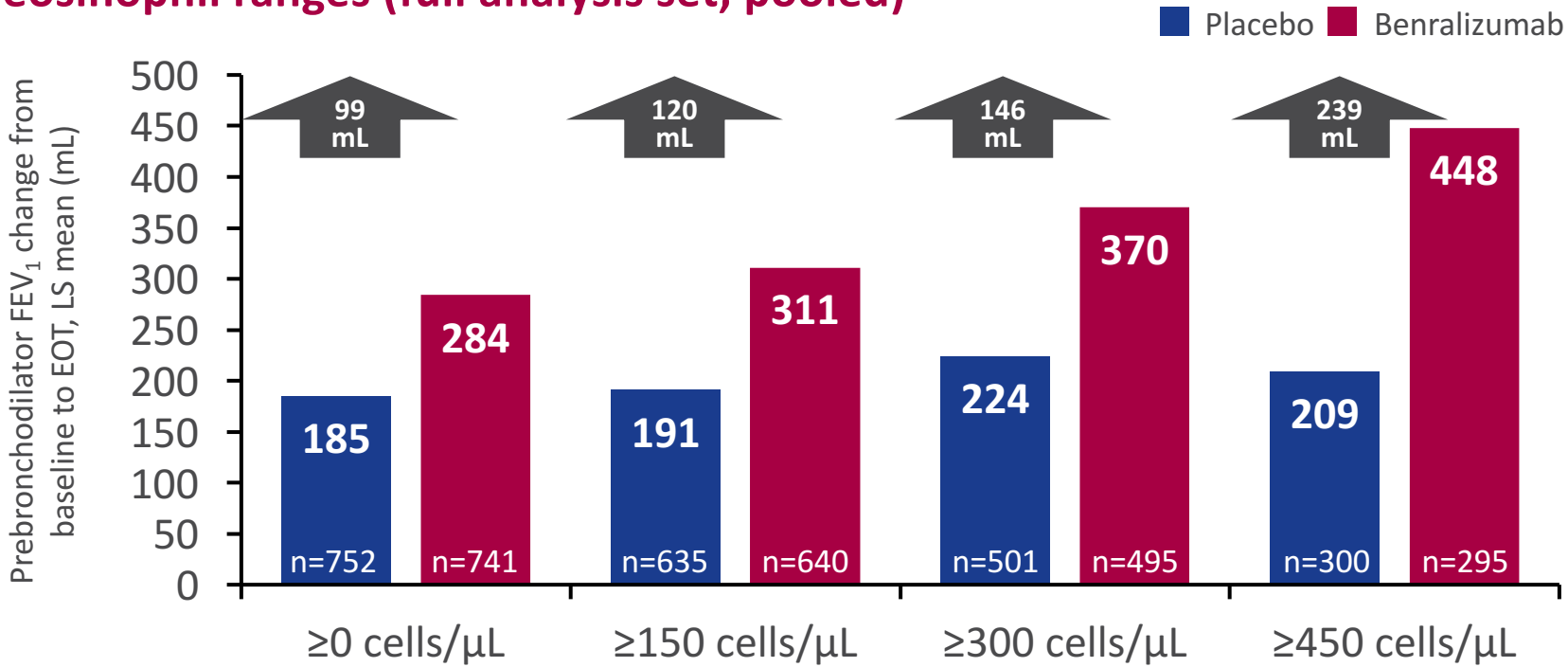
Prebronchodilator FEV_1 increase with benralizumab Q8W by eosinophil ranges (full analysis set, pooled)



Estimates calculated using a mixed-effects model for repeated measures analysis with adjustment for treatment, study, baseline value, region, oral corticosteroid use at randomization, visit, and visit × treatment. EOT visit at Week 48 (SIROCCO) or 56 (CALIMA). Estimates weighted to account for the 2:1 randomization ratio of patients with baseline blood eosinophil counts ≥300 or <300 cells/µL. CI: confidence interval; EOT: end of treatment; FEV_1: forced expiratory volume in 1 s; LS: least squares; Q4W: every 4 weeks; Q8W: every 8 weeks (first 3 doses Q4W); $p < 0.0001$ for every EOS cut-off

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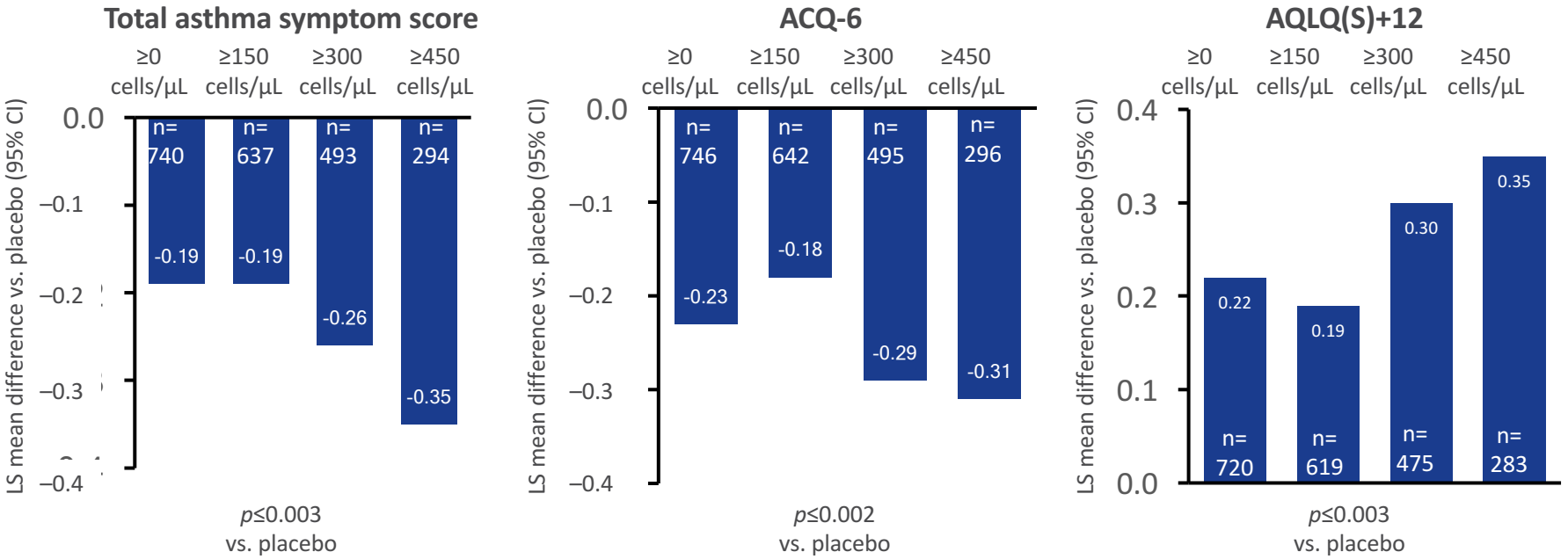
Greater improvement in symptoms and asthma-related quality of life with benralizumab Q8W with increasing baseline blood eosinophil counts



Data are LS mean difference (95% CI). Estimates calculated by a mixed-effects model for repeated measures analysis with adjustment for treatment, study code, baseline value, region, oral corticosteroid use at time of randomization, visit, and visit × treatment. The estimates were weighted to account for the 2:1 randomization ratio of patients with baseline blood eosinophil counts ≥300 cells/µL or <300 cells/µL. EOT visit at Week 48 (SIROCCO) or 56 (CALIMA). ACQ-6: Asthma Control Questionnaire 6; AQLQ(S)+12: Asthma Quality of Life Questionnaire (standardized) for 12 years and older; CI: confidence interval; EOT: end of treatment; LS: least squares; Q8W: every 8 weeks (first three doses every 4 weeks).

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Greater benralizumab Q8W efficacy for patients with more frequent exacerbation history

Blood eosinophil counts ≥300 cells/μL	Exacerbations in previous year	
	2	≥3
Annual exacerbation rate[a]	n=308	n=198
Rate ratio vs. placebo	0.73 (0.55 to 0.95)	0.45 (0.34 to 0.60)
P-value vs. placebo	0.019	<0.001
Prebronchodilator FEV_1 (mL)[b]	n=305	n=197
LS mean difference vs. placebo	70 (−8 to 149)	252 (148 to 357)
P-value vs. placebo	0.080	<0.001
Total asthma symptom score[b]	n=304	n=196
LS mean difference vs. placebo	−0.18 (−0.36 to −0.00)	−0.36 (−0.57 to −0.14)
P-value vs. placebo	0.049	0.001

[a]Data are rate ratio (95% CI). Estimates were calculated by using a negative binomial model, with adjustment for treatment, study code, region, oral corticosteroid use at time of randomization, and prior exacerbations. Total follow-up time is defined as the time from randomization up to and including EOT visit at Week 48 (SIROCCO) or 56 (CALIMA) or last contact if the patient is lost to follow up. [b]Data are LS mean difference (95% CI). Prebronchodilator FEV_1 and total asthma symptom score change are from baseline to end of treatment (SIROCCO: Week 48; CALIMA: Week 56). Estimates calculated by a mixed-effects model for repeated measures analysis with adjustment for treatment, study code, baseline value, region, oral corticosteroid use at time of randomization, visit, and visit × treatment. CI: confidence interval; EOT: end of treatment; FEV_1: forced expiratory volume in 1 s; LS: least squares; Q4W: every 4 weeks; Q8W: every 8 weeks (first three doses Q4W).

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Conclusions

- In combination with high-dosage ICS/LABA, benralizumab provides additional benefit for patients with severe, uncontrolled asthma across the spectrum of baseline blood eosinophil counts
- Improvements were greater for patients with increased baseline blood eosinophil counts
- Enhanced efficacy was observed also for patients with ≥3 exacerbations/year as opposed to those with fewer exacerbations/year
- Certain clinical features consistent with the eosinophilic asthma phenotype such as maintenance OCS use and nasal polyps were also associated with enhanced efficacy
- Identifying clinical characteristics and predictive markers associated with increased benralizumab efficacy will be important for identifying patients who will benefit most from this agent

ICS: inhaled corticosteroids; LABA: long-acting β_2-agonists.

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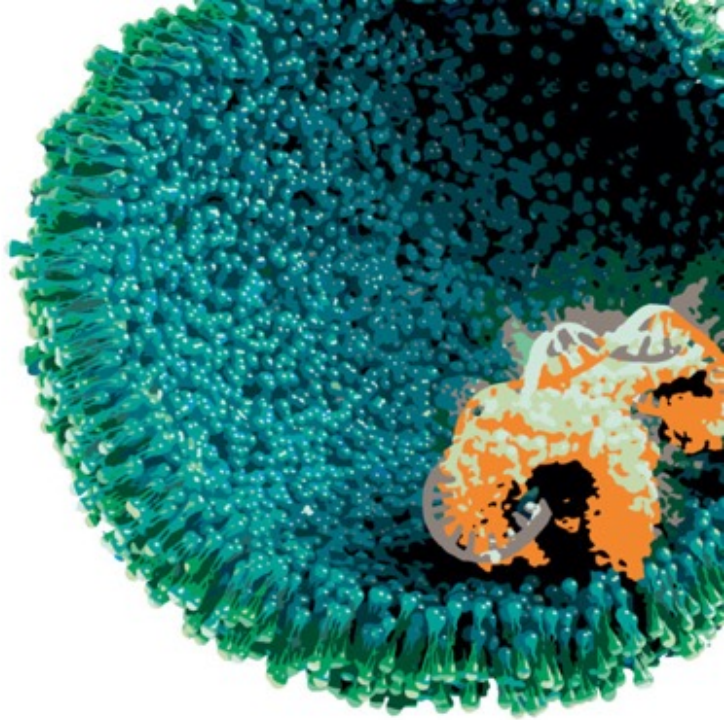


OA3189

Efficacy and safety of tezepelumab in adults with severe asthma: A randomised Phase II Study

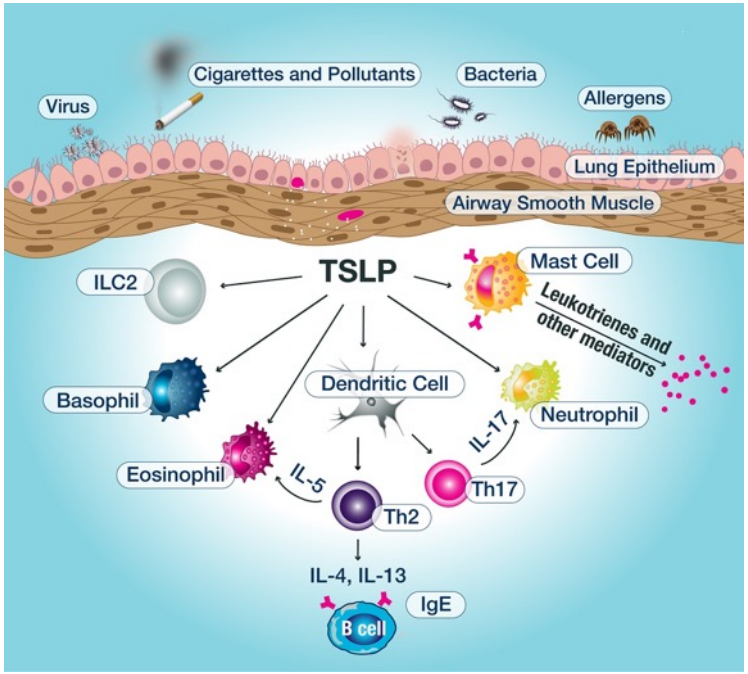
Jonathan Corren, M.D.,[1] Jane R. Parnes, M.D.,[2] Liangwei Wang, Ph.D.,[3] May Mo, M.S.,[2] Stephanie L. Roseti, A.P.N., M.S.N.,[3] Janet M. Griffiths, Ph.D.,[3] René van der Merwe, M.B.Ch.B.[4]

[1]David Geffen School of Medicine at UCLA, Los Angeles, CA, USA, [2]Amgen Inc., Thousand Oaks, CA, USA, [3]MedImmune, Gaithersburg, MD, USA, [4]MedImmune, Cambridge, UK

ERS 2017, 9–13 September, Milan, Italy
12 September 2017

Functions of TSLP

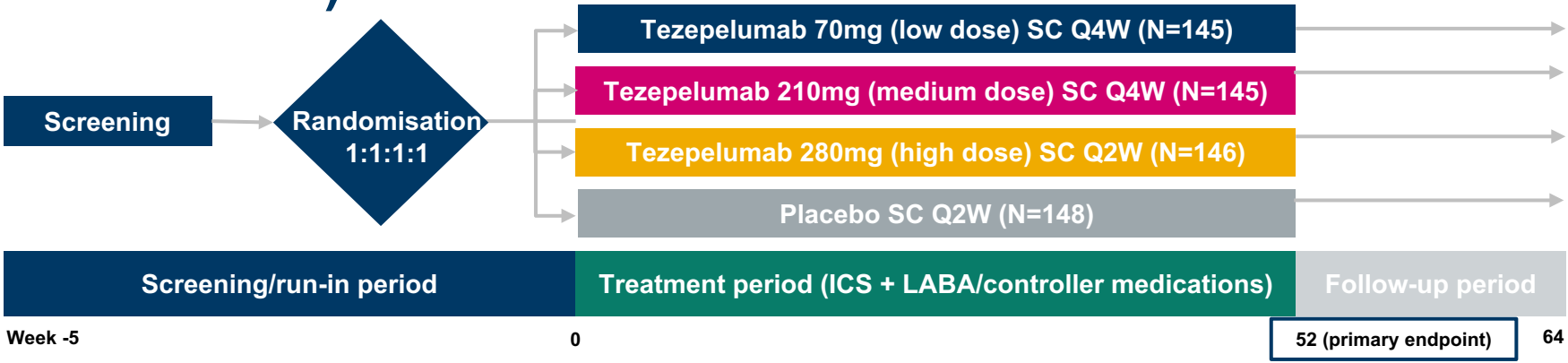
- TSLP is an epithelial-derived cytokine central to the regulation of type 2 immunity[1–4]
- TSLP expression is increased in the airways of patients with asthma, and correlates with Th2 cytokine and chemokine expression, and disease severity[5–7]
- Tezepelumab (AMG 157/MEDI9929) is an investigational human IgG2 mAb that binds to TSLP, inhibiting its interaction with the TSLP receptor complex[8]



ILC2, type 2 innate lymphoid cell; TSLP, thymic stromal lymphopoietin; IgG2, immunoglobulin G_2; mAb, monoclonal antibody

1. Ziegler and Artis. *Nat Immunol* 2010;11:289–293. **2.** Soumelis, et al. *Nat Immunol* 2002;3:673–680. **3.** Allakhverdi, et al. *J Exp Med* 2007;204:253–258. **4.** Ziegler, et al. *Adv Pharmacol* 2013;66:129–155. **5.** Shikotra, et al. *J Allergy Clin Immunol* 2012;129:104-11 e1-9. **6.** Ying, et al. *J Immunol* 2005;174:8183–8190. **7.** Ying, O'Connor B, et al. *J Immunol* 2008;181:2790–2798. **8.** Gauvreau GM, et al. N Engl J Med 2014;370:2102–2110.

PATHWAY Phase 2b placebo-controlled trial design (NCT02054130)



Patient population

- Non-smokers, aged 18–75 years
- Asthma uncontrolled despite treatment with medium- or high-dose ICS* plus LABA (GINA 2012 guidelines[1])
- ≥2 asthma exacerbations that led to systemic glucocorticoid treatment, or >1 severe exacerbation that led to hospitalisation (12 months before trial entry)

Stratification

- Location: Japan or rest of world
- Blood eosinophil count (≥250 or <250 cells/μL)
- ICS dose: medium or high (GINA 2012 guidelines[1])

*Medium dose: 250–500 μg/day fluticasone DPI or equivalent; high dose: >500 μg/day fluticasone DPI or equivalent
DPI, dry-powder inhaler; Q2W, every 2 weeks; Q4W, every 4 weeks; SC, subcutaneous

1. Global Initiative for Asthma. Global Strategy for Asthma Management and Prevention, 2012. Available from http://ginasthma.org/.

Baseline patient characteristics

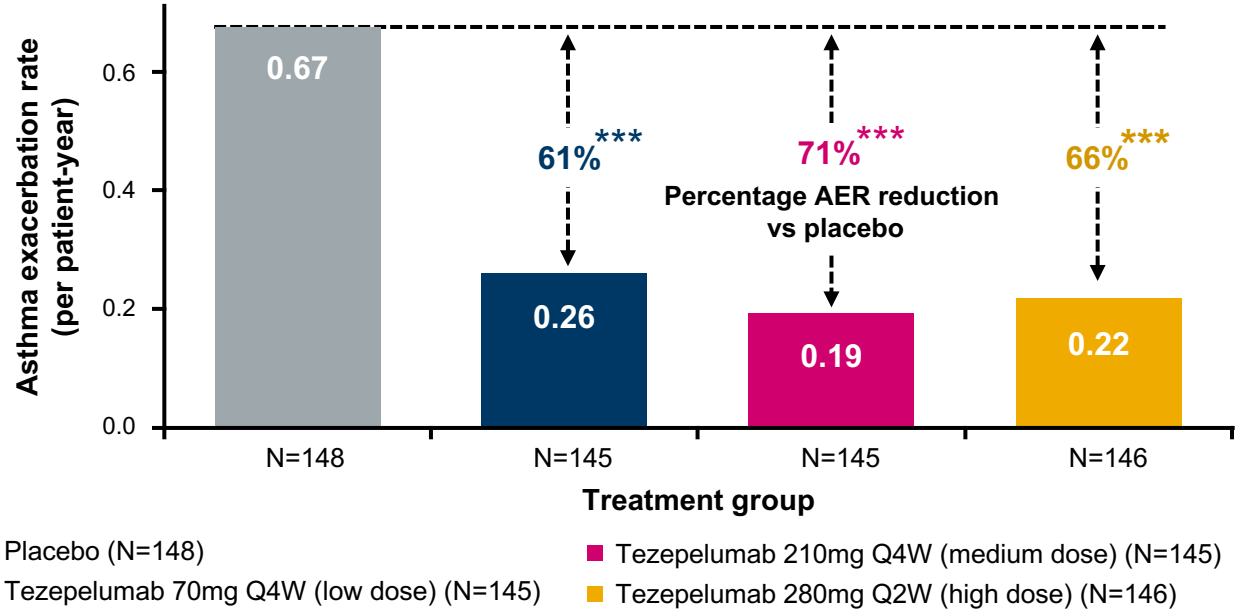
Baseline demographics were similar between treatment groups (ITT population)		Placebo (N=148)	Low dose (70 mg Q4W) (N=145)	Medium dose (210 mg Q4W) (N=145)	High dose (280 mg Q2W) (N=146)
Age, years	Mean (SD)	52.2 (11.5)	50.6 (12.4)	52.6 (12.5)	50.1 (12.2)
Male	n (%)	48 (32.4)	50 (34.5)	54 (37.2)	53 (36.3)
Caucasian	n (%)	133 (89.9)	138 (95.2)	136 (93.8)	129 (88.4)

Baseline clinical characteristics were similar between treatment groups (ITT population)	Placebo (N=148)	Low dose (70 mg Q4W) (N=145)	Medium dose (210 mg Q4W) (N=145)	High dose (280 mg Q2W) (N=146)
Pre-BD FEV_1 (L)/ FEV_1 % predicted, mean (SD)	1.83 (0.58)/ 60.4 (13.6)	1.91 (0.66)/ 60.7 (13.5)	1.83 (0.58)/ 59.2 (12.4)	1.86 (0.60)/ 59.3 (11.8)
Mean ACQ-6,* mean (SD)	2.66 (0.67)	2.76 (0.80)	2.71 (0.81)	2.63 (0.75)
Overall AQLQ(S)+12,† mean (SD)	4.06 (0.86)	4.14 (0.94)	4.19 (0.90)	4.09 (0.90)
Daily ICS dose (µg), median (min, max)	500 (250, 2500)	500 (250, 3000)	500 (200, 2400)	500 (250, 2000)
Eosinophil count (cells/µl), mean (SD)	366 (323)	345 (284)	359 (347)	378 (423)
Total serum IgE (IU/ml), mean (SD)	447 (1232)	314 (870)	464 (1366)	344 (579)
FE_{NO} (ppb), mean (SD)	36.3 (38.9)	34.5 (46.9)	30.4 (29.4)	32.6 (33.9)

ITT, intention-to-treat; SD, standard deviation
*Mean ACQ-6 score: ≤0.75 = well controlled; >0.75 and <1.5 = partly controlled; ≥1.5 = uncontrolled
†Mean AQLQ score: 7 = no impairment; 1 = severe impairment

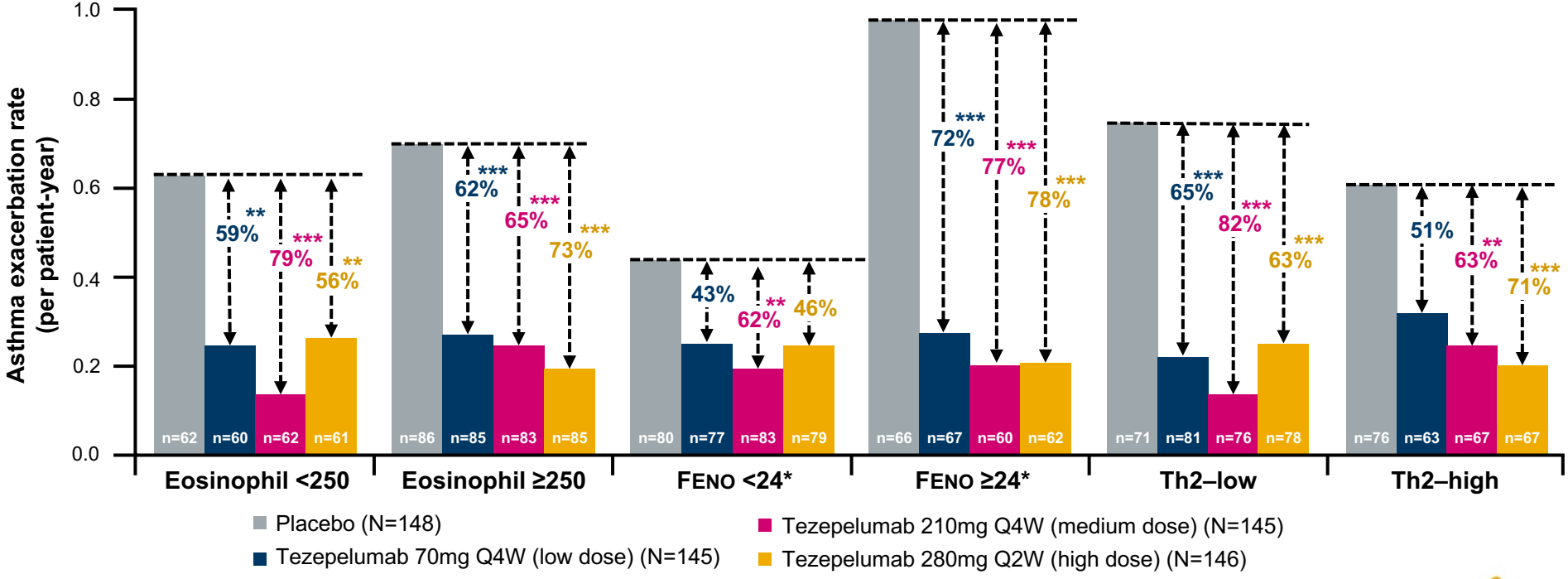
Tezepelumab treatment reduced the annualised AER vs. placebo at Week 52

Significant reduction in annualised AER for all tezepelumab treatment groups compared with placebo; P<0.001



***P<0.001, compared with placebo group. Sequential testing approach was used to adjust for the multiplicity caused by the multiple dose-placebo comparisons. The hierarchy was tezepelumab 280 mg, 210 mg, and 70 mg vs placebo

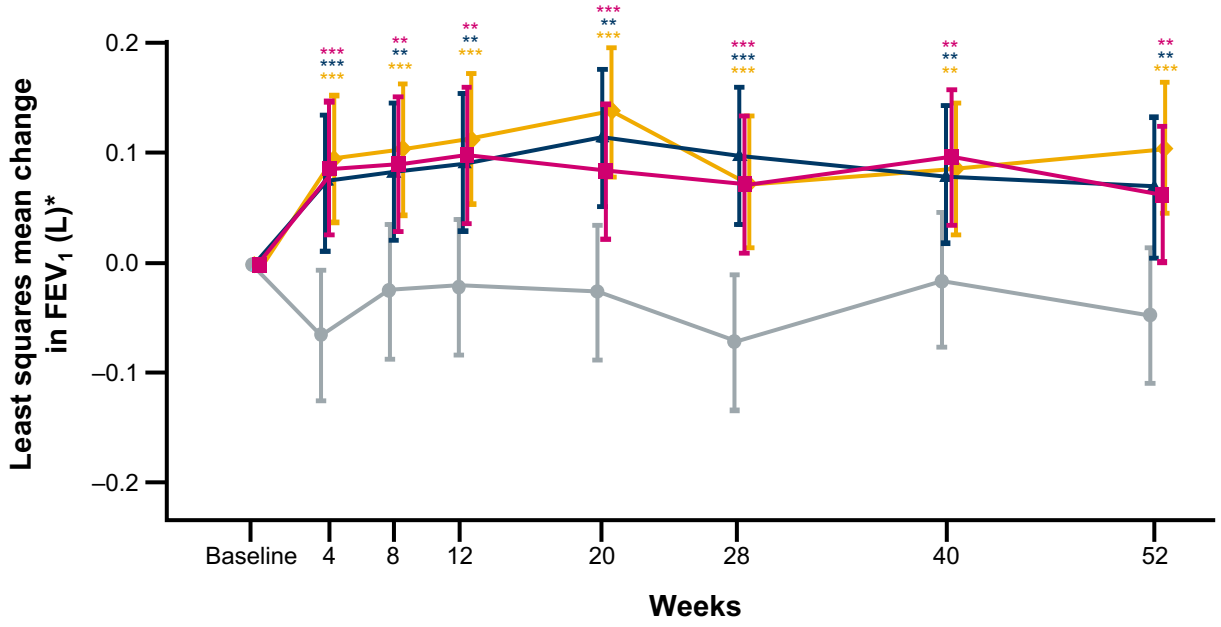
Tezepelumab treatment reduced annualised AER vs. placebo at Week 52 irrespective of baseline biomarker status



Nominal two-sided P-values: **P<0.05, ***P≤0.01 compared with placebo group

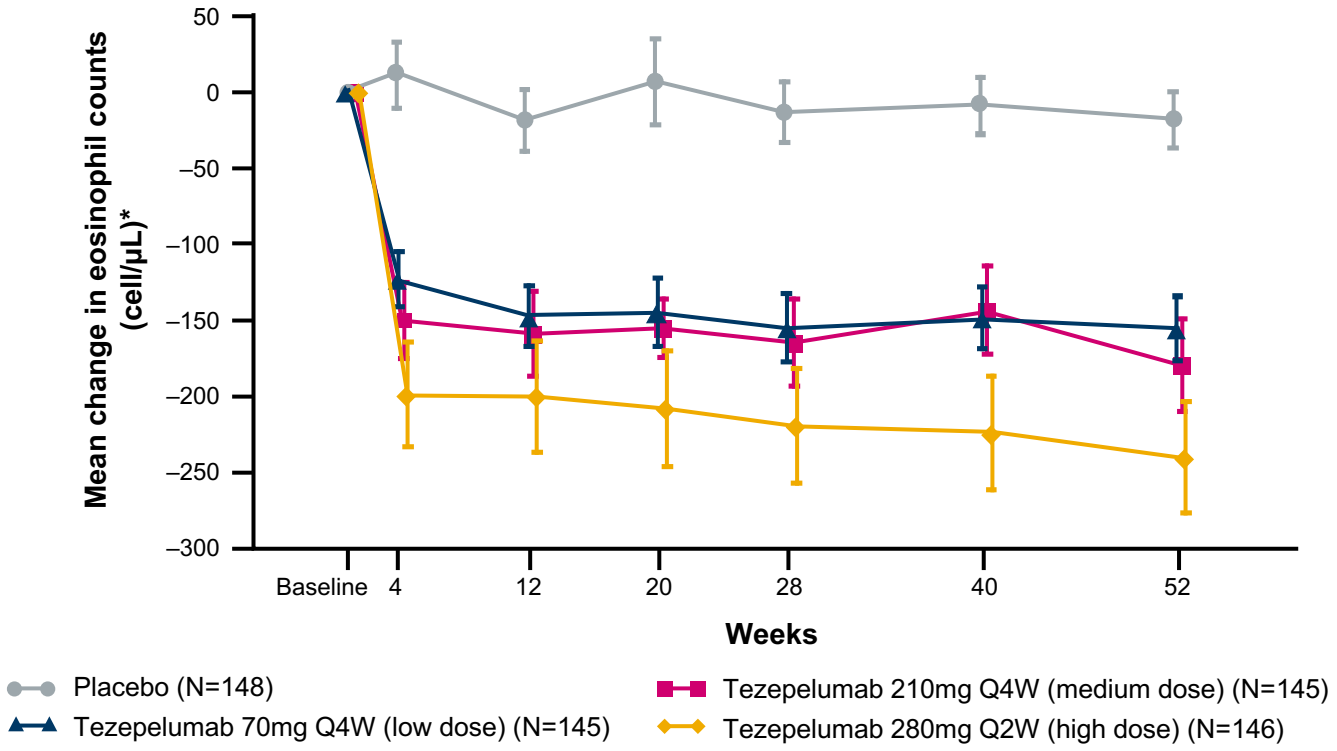
*Clinically meaningful cutoff for the FE_{NO} subpopulation analysis: 24 ppb

Tezepelumab treatment increased pre-BD FEV_1 vs. placebo at Week 52



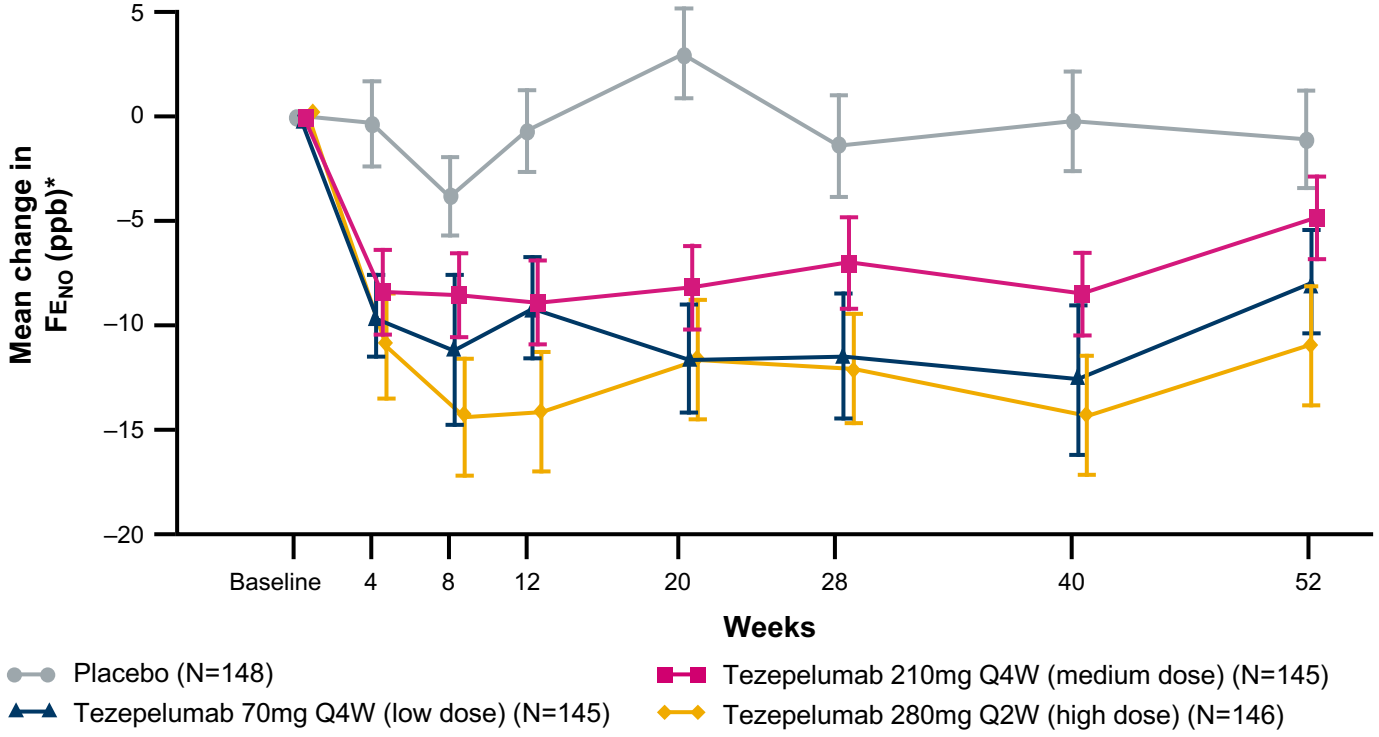
Nominal two-sided P values: **P<0.05, ***P<0.01 compared with placebo group
*Least squares mean and SE of the observed data were plotted over time. Approximately 9% of data were missing at Week 52
SE, standard error

Tezepelumab treatment reduced blood eosinophils



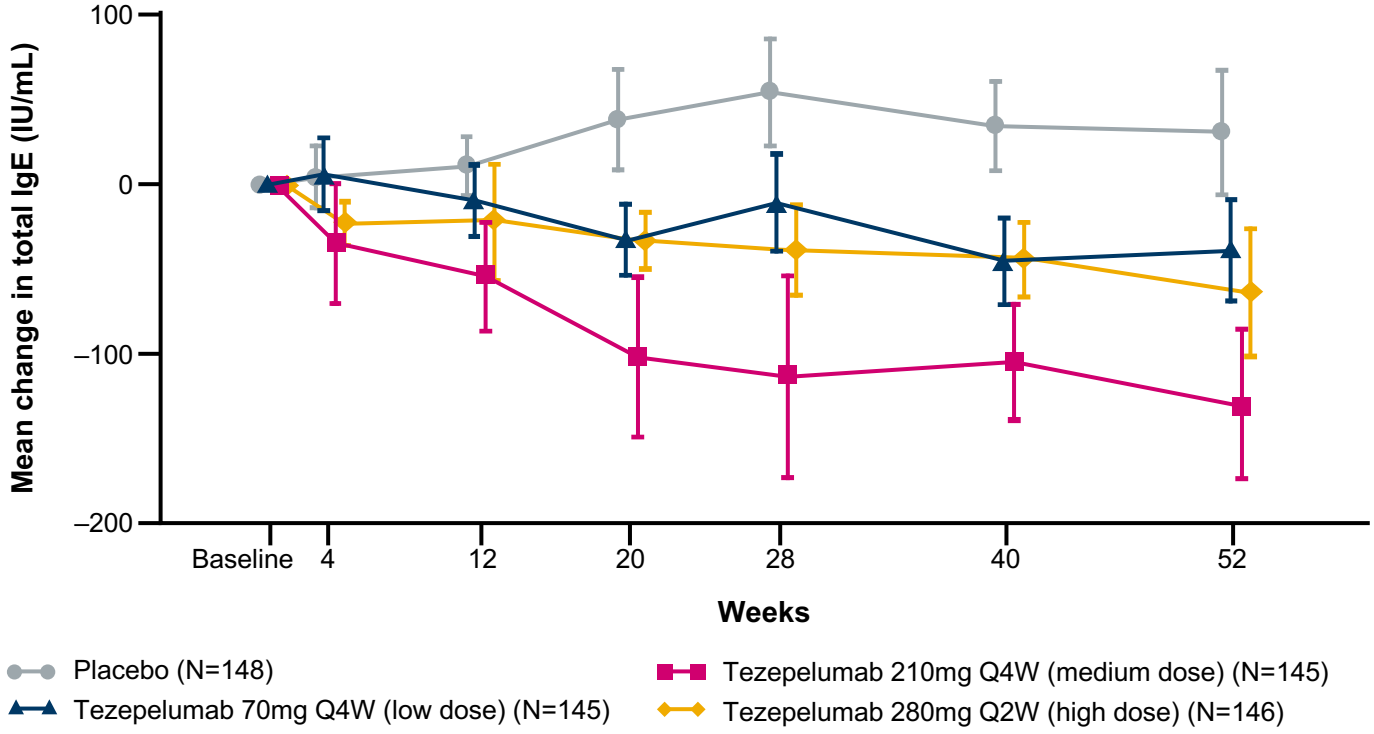
*Mean and SE of the observed data were plotted over time. Approximately 13% data were missing at Week 52

Tezepelumab treatment reduced FENO



*Mean and SE of the observed data were plotted over time. Approximately 27% data were missing at Week 52

Tezepelumab treatment reduced total serum IgE



*Mean and SE of the observed data were plotted over time. Approximately 9% data were missing at Week 52

Safety summary

Patients* with	Placebo (N=148)	Low dose (70 mg Q4W) (N=145)	Medium dose (210 mg Q4W) (N=145)	High dose (280 mg Q2W) (N=146)
At least one AE, %	62.2	66.2	64.8	61.6
At least one AE of ≥Grade 3–5 severity, %†	18.9	17.9	20.0	14.4
Death (Grade 5 severity†), n	0	1	0	0
At least one serious‡ AE, %	12.2	11.7	9.0	12.3
At least one serious‡ and/or ≥Grade 3–5 severity† AE, %	23	22.1	22.1	19.9
At least one AE leading to discontinuation of investigational product, %	0.7	0	1.4	2.1

*Patients were counted once for each category regardless of the number of events; †Grade 3: severe, Grade 4: life threatening, Grade 5: fatal
‡Serious adverse event criteria: death, life threatening, required inpatient hospitalisation, prolongation of existing hospitalisation, persistent or significant disability/incapacity, important medical event, congenital anomaly/birth defects (in the offspring of the patient)

Conclusions

- Tezepelumab treatment reduced blood eosinophil, FENO and total IgE counts, indicating that **tezepelumab has important effects on IL-4, IL-5 and IL-13 pathways**
  - Inhibition of TSLP appears to have broader physiological effects than the targeting of individual Th2 cytokines

- These data provide clinical evidence that **inhibition of TSLP reduces annualised AER irrespective of baseline biomarker status** (high/low eosinophil count, FENO ≥ or <24 ppb, Th2 high/low)
  - Inhibition of TSLP may also benefit patients with non-Th2 inflammation

- The overall incidence of AEs was similar across treatment and placebo groups

Agenda

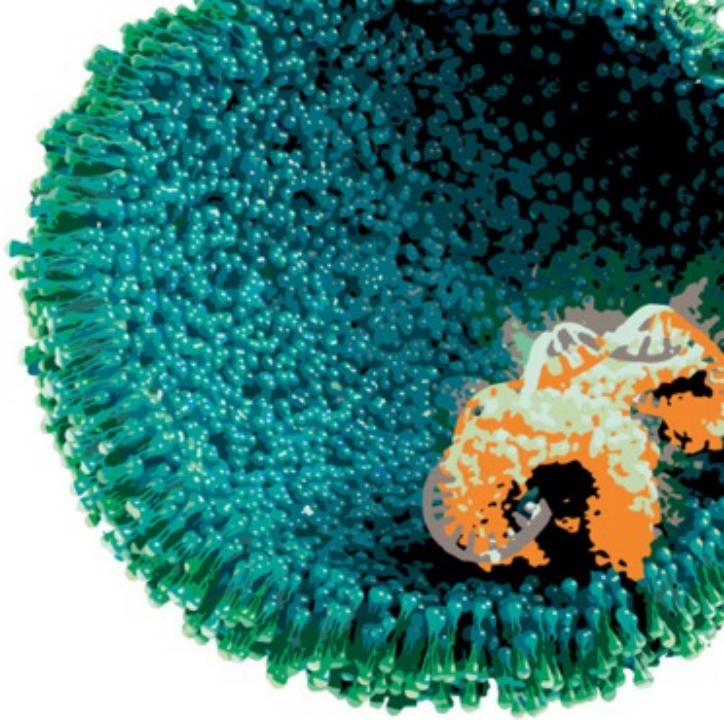

Introduction


Unmet medical need


Characterising responders to benralizumab


Tezepelumab Phase IIb PATHWAY


Commercial opportunity



How does benralizumab compare to currently-approved biologics and those in late-stage development?

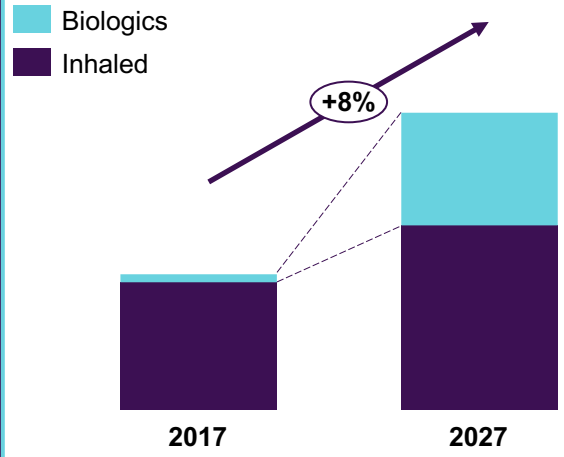
How do you see tezepelumab fitting into or potentially changing the treatment paradigm?

What factors will enable more asthma patients to benefit from biologic medicines in the future?

Respiratory - strategy

Therapy area with potential for biologics leadership

Drivers of market growth



Strength in inhaled

Backbone of care

Established medicines
Symbicort, *Pulmicort*, *Bevespi*, *Duaklir*, *Daliresp*/*Daxas*

New paradigms
PRN *Symbicort*, PT027, PT010, PT009, Aerosphere platform

Next generation
iSGRM, MABA, abediterol, iENAC

Leading biologics portfolio

Transforming outcomes

Benralizumab
Direct, rapid and near-complete depletion of eosinophils

Tralokinumab
Blocks binding and signalling of IL-13 to IL-13 receptors

Tezepelumab
First-in-class targeting thymic stromal lymphopoietin (TSLP), an upstream driver of airway inflammation

Disease modification

Early intervention


Epithelium

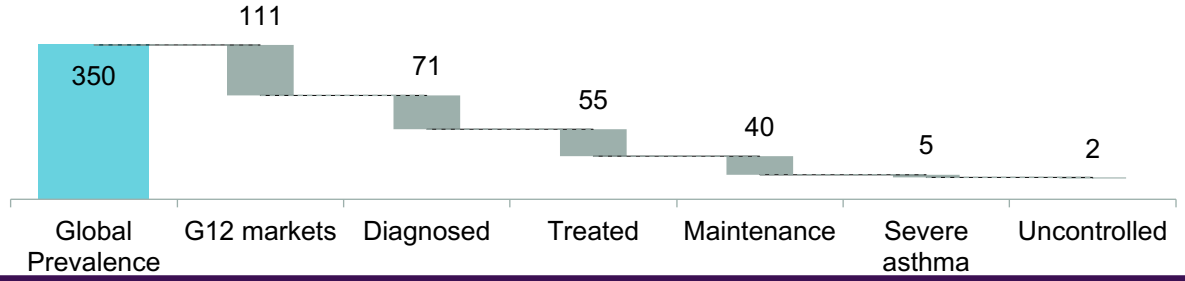

Immunity


Regeneration

Source: External market research and internal estimates.

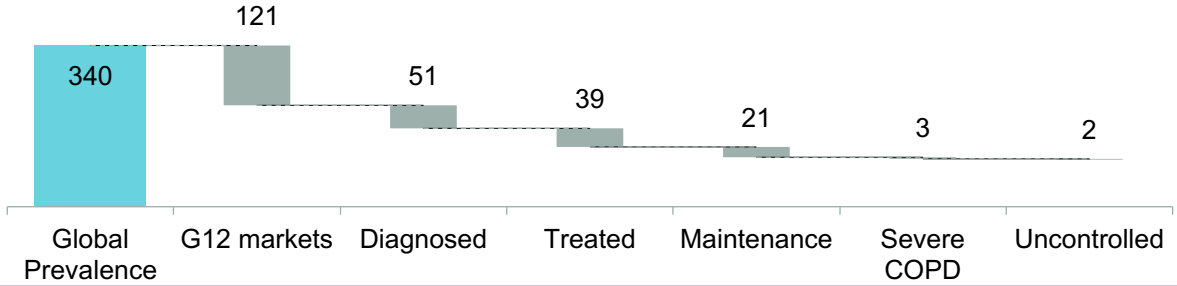
High economic burden and a clear unmet need will continue to drive the severe asthma and COPD market

Asthma, 2015, G12 Countries[1, 2]



Asthma, rates[1]	2015	2017	2025
Prevalence	350	360	↑
Diagnosis	64%	65%	↑
Treatment	77%	78%	↑
Biologic	8%	10%	↑

COPD, 2015, G12 Countries[1, 2]



COPD, rates[1]	2015	2017	2025
Prevalence	340	357	↑
Diagnosis	42%	43%	↑
Treatment	78%	79%	↑
Biologic	0%	0%	↑

1. AstraZeneca analysis supported by Decision Resources, IMS MIDAS and IMS longitudinal data and other specific country sources.
2. Markets include: US, EU5 (United Kingdom, Germany, Italy, France, Spain), Japan, China, Canada, Australia, Brazil and Russia.

Benralizumab: Potential precision medicine for eosinophilic asthma

Strong Phase III clinical profile

- Annual exacerbation rate (AERR) reduction up to 51%
- In OCS-dependent patients 70% AERR and 75% median OCS dose reduction
- Improved lung function after the first dose, improved symptoms, QoL
- Q8w dosing, pre-filled syringe, injection site reactions not different from placebo

Clear patient phenotype in clinical practice

- Blood eosinophils ≥ 300 cells/μL
- Frequent exacerbator ≥3 exacerbations/year
- Chronic OCS
- Nasal polyps

Next steps

- Awaiting regulatory decision US (H2 2017), EU and Japan (H1 2018)
- Phase III VOYAGER (COPD) programme 2018
- Life-cycle management programme, which including home administration

Tezepelumab: First-in-class/first-in-disease treatment that blocks TSLP - an upstream driver of inflammation in asthma

Best-in-disease Phase II efficacy

- Reduced exacerbation rates 61% - 71%
- Consistent across sub-populations with and without T2 inflammation
- Improvements in lung function, asthma control and quality of life
- Unprecedented reductions of key T2 biomarkers: blood eosinophils, FE_{NO} and IgE

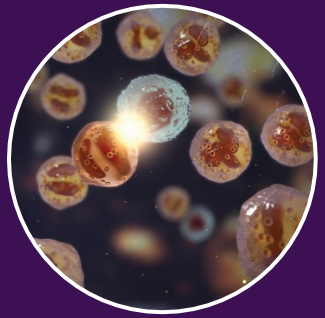
Broadest potential role in clinical practice

- Best in disease T2 profile
- Extended to non-T2 patients ineligible for other biologics
- Potential default first line choice, paradigm simplification

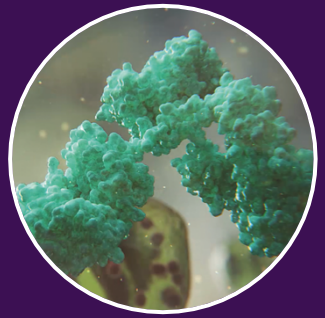
Next steps

- Evaluating Phase III plans in severe asthma
- Lifecycle opportunities

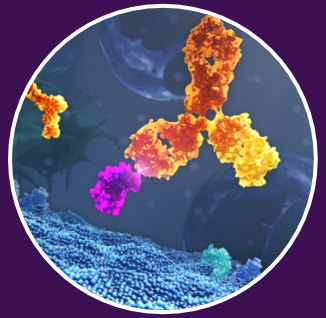
A biologics portfolio that follows the science



Benralizumab is an anti-eosinophil monoclonal antibody that targets the IL-5 receptor, thereby inducing direct and near complete depletion of eosinophils via antibody-dependent cell mediated cytotoxicity



Tralokinumab is an anti-IL-13 monoclonal antibody that blocks binding and signalling of IL-13 to IL-13 receptors



Tezepelumab is a potential first in class monoclonal antibody that specifically binds to TSLP, an upstream epithelial master switch that drives multiple downstream inflammatory pathways.

Q&A

Use of AstraZeneca webcast, conference call and presentation slides

AstraZeneca
What science can do

Investor science webcast: ERS 2017

Conference call and webcast for investors and analysts, Milan, Italy

13 September 2017