NOVARTIS | Reimagining Medicine

Novartis Immunology Pipeline Event

October 30, 2025

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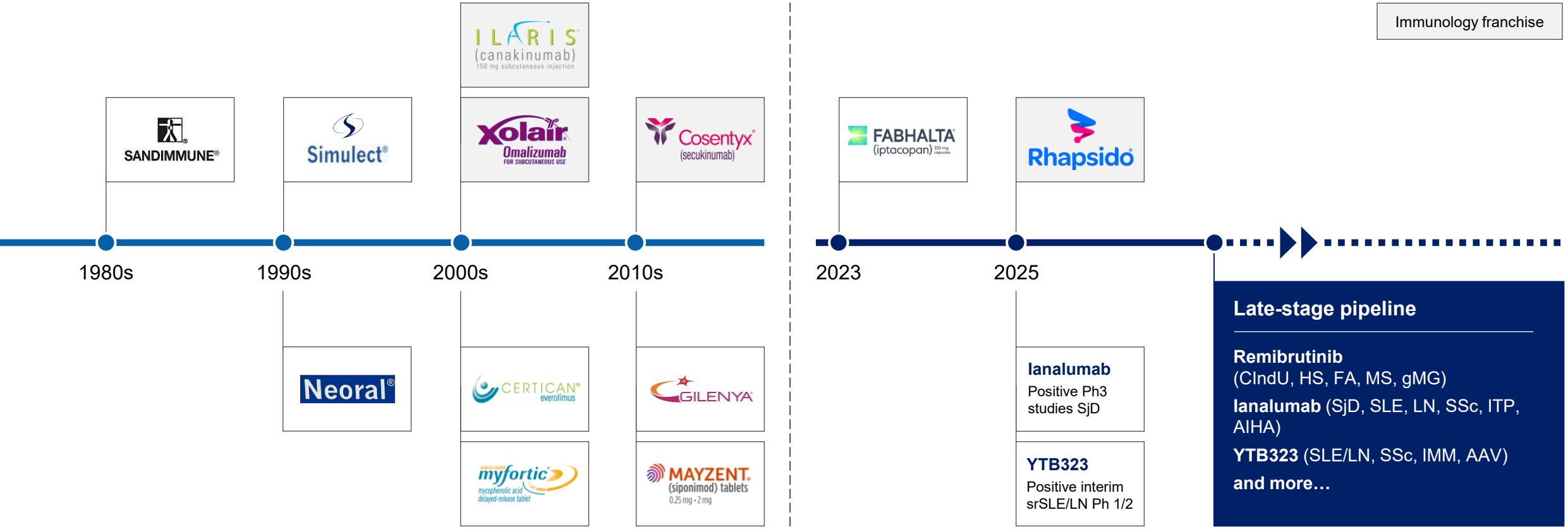
Participants

Shreeram Aradhye, MD
President, Development
and Chief Medical Officer

Victor Bulto
President, US

Angelika Jahreis, MD PhD
Global Head, Immunology
Development Unit

Novartis has a long legacy in immunology



Remibrutinib has only been approved in the US with expected ex-US approvals in 2026.

Immunological conditions present a large and growing burden on patients and society[1,2]

Immunological conditions affect

>10%

of the global population[3-4]

Daily life impact for patients[9-11]

- Chronic, painful and progressive nature of immunological conditions places significant physical and psychological burden on patients[10-12]
- Impact is often underestimated, leading to delays in diagnoses[12-16]

Major financial and socioeconomic burden

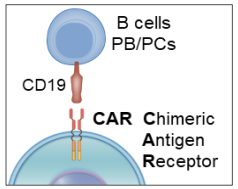
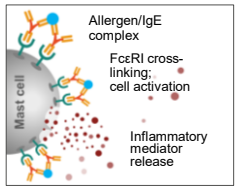
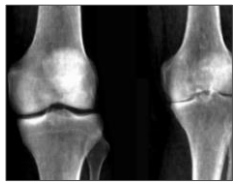
- Autoimmune diseases alone drive >USD 100bn in annual US healthcare costs[17-18]

Rising demand for innovation

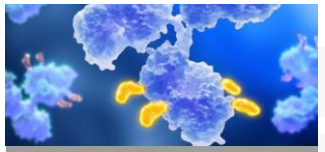
- Treatments considered effective today often only work for a subset of patients, even among those who exhibit similar symptoms[5]
- Availability of new therapies expected to drive >10% global annual market growth[6-8]

See page 51 for references (footnotes 1-18).

Within immunology, we are building a deep portfolio around selected core disease areas

Core areas		Indications
Immuno-Dermatology		PsO, HS, CSU, CIndU, AtD T-cell driven skin diseases
Systemic autoimmunity		SjD, SLE, LN, SSc, IIM, AAV, RA
Allergies		CSU, CIndU, Food Allergy
Arthritides		Spondylitis/Spondyloarthritis Osteoarthritis

Enabled by classic and new modalities

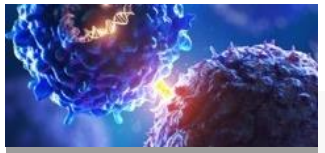


Biologics

- mAbs
- Bi/Trispecifics

Orals

- LMW monotherapies
- Combination therapies
- Peptides



Novel modalities

- CAR-T

Complementing internal innovation with external assets to deliver on our immunology ambitions

Recent deals to expand into new and high value target spaces

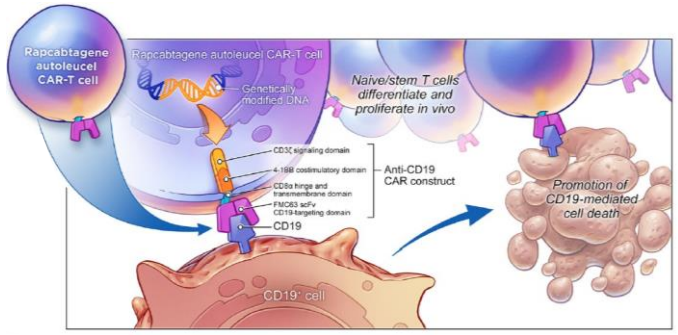
Calypso biotech	IFMDUE	Monte Rosa Therapeutics	Kyorin
anti-IL15 mAb	**STING antagonist**	**VAV1 degrader**	**MRGPRX2 inhibitor**
GIA632: Anti-IL-15 for CD8 T-cell-driven skin diseases, including AtD (PhII FPFV 2025)	**IFM-32531:** Brain penetrant covalent STING inhibitor with FiC potential (PhI)	**MRT-6160:** Highly specific and potent non-canonical CRBN-based molecular glue degrader (PhI)	**KRP-M223**: Potent and specific MRGPRX2 inhibitor blocking non-IgE mediated mast-cell degranulation

Advancing “pipeline-in-a-pill” products across modalities

YTB323

CAR-T therapy for deep B cell depletion

Lead indication: srSLE/LN



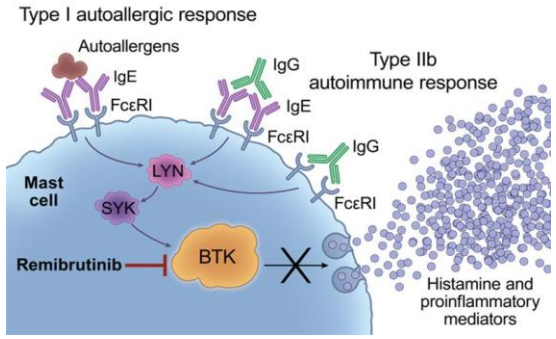
LCM

SSc, IIM, AAV, RA, SjD, rMS, pMS, gMG

Rhapsido

Small molecule, selective BTK inhibitor

Lead indication: CSU



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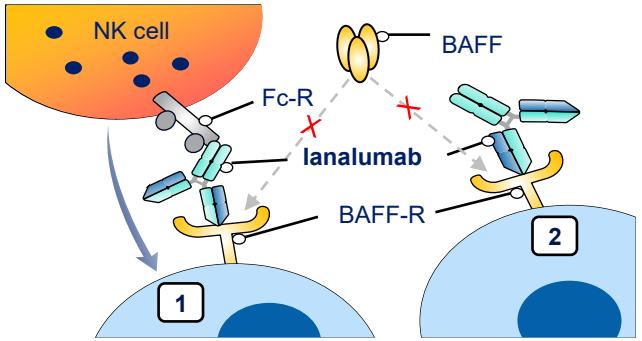
mAb, dual MOA for enhanced B cell targeting

Lead indication: SjD

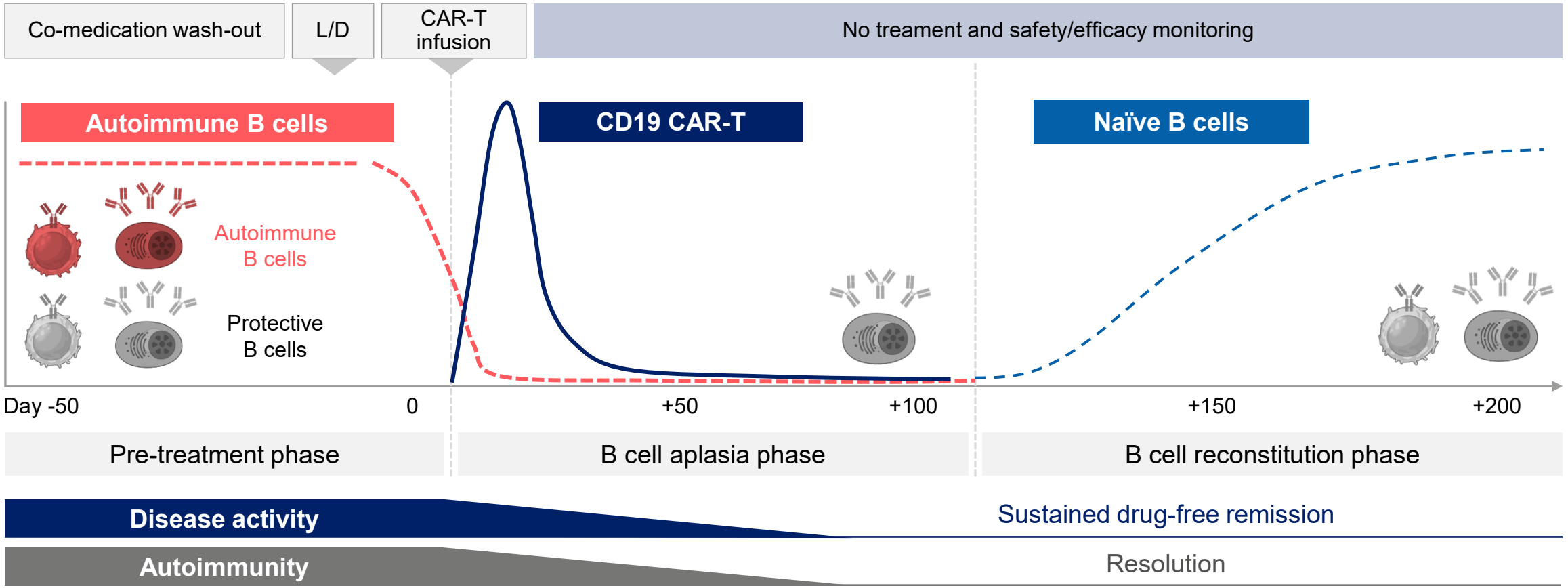


LCM

SLE, LN, SSc, ITP, wAIHA

Investing in broad development programs with high conviction and alignment across RDC continuum

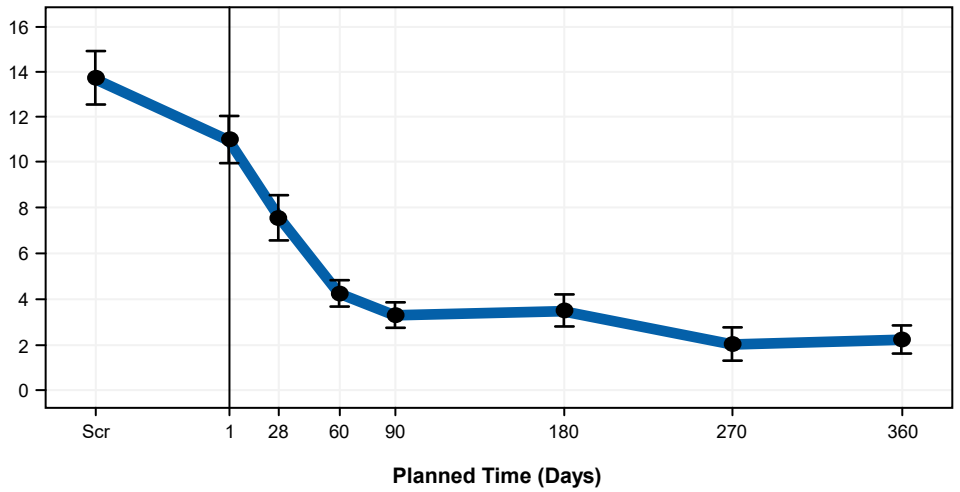
Effective depletion of the B cell compartment via CD19 CAR-T therapy may reset pathologic autoimmunity



Adapted from Schett G et al Lancet 2023.

Positive Ph1/2 study[1] with YTB323[2] in SLE prompted four pivotal trials across autoimmune diseases, with first readout expected ≥2027

SLEDAI-2K total score over time, mean (SE)[3]



Early and sustained improvement of overall disease activity in patients with srSLE (n=21) with up to 12 months follow-up

Safety in line with CAR-T therapy experience

Selected projects	Pre-clinical	Phase I/II	Phase II	Phase III
srSLE/LN[4]				
srSLE/LN[4, 5]				
SSc[5]				
IIM[5]				
AAV[5]				
RA/SjD[4]				
rMS				
pMS				
gMG				

Disease area: Rheumatology | Neuroscience

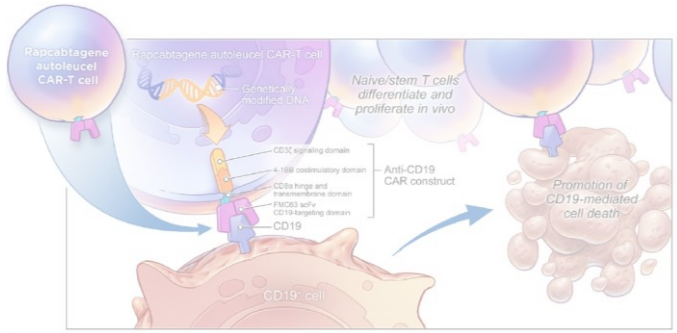
See page 52 for references (footnotes 1-5).

With Rhapsido® FDA approval and ianalumab positive data in SjD, we have derisked two assets with multi-blockbuster potential

YTB323

CAR-T therapy for deep B cell depletion

Lead indication: srSLE/LN



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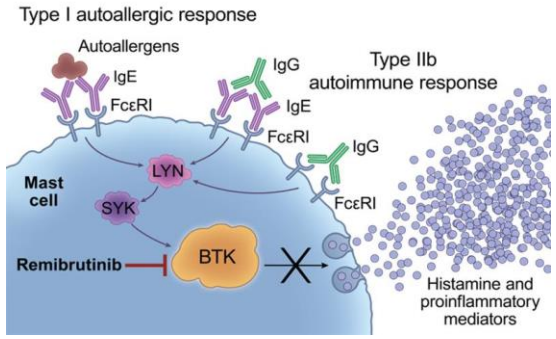

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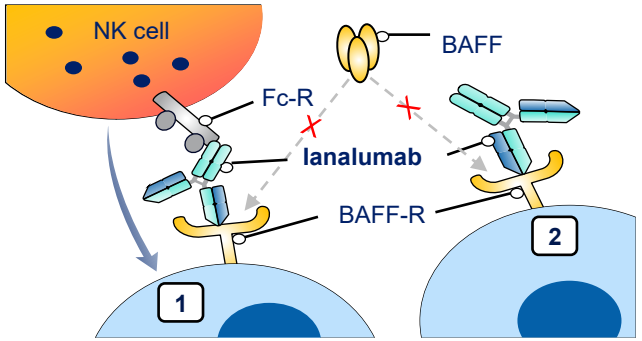

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SLE, LN, SSc, ITP, wAIHA

Investing in broad development programs with high conviction and alignment across RDC continuum

Rhapsido® approved by FDA as the only oral, targeted BTK inhibitor for CSU with a clean label[1]

Broad indication[1]

Indicated for the treatment of chronic spontaneous urticaria (CSU) in adult patients who remain symptomatic despite H1 antihistamine treatment

Clean safety[1]

- **NO boxed warning**
- **NO contraindications**
- **NO required routine lab monitoring**

Oral administration[1]

25mg tablet twice daily, with or without food

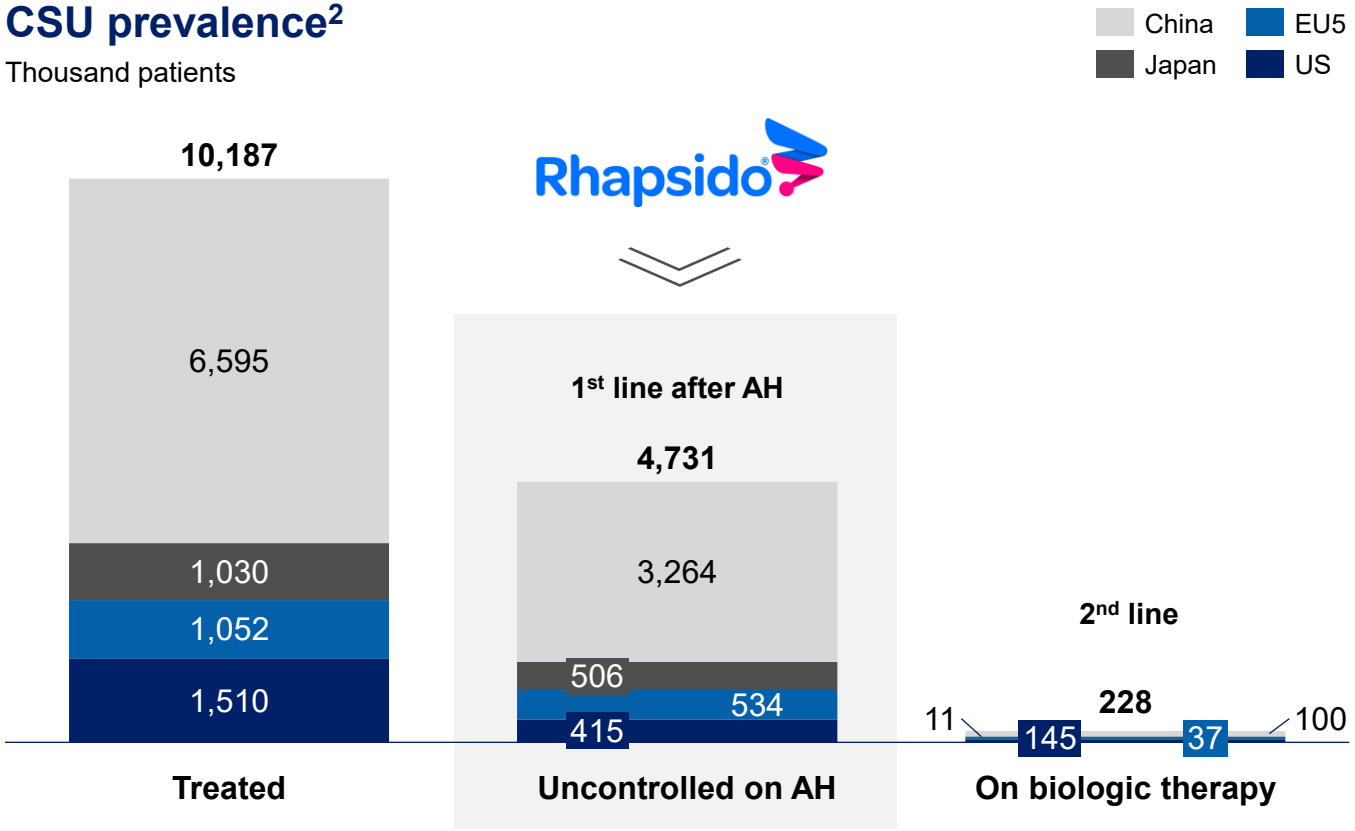
Ex-US: EU, CN and JP submissions completed

See page 52 for references (footnote 1).

Positioned to address a long-standing treatment gap after antihistamine failure and before biologics; CSU market opportunity 1/2 the size of PsO[1]

CSU prevalence[2]

Thousand patients



CSU patient experience

- Systemic debilitating mast cell-driven autoimmune disease, characterized by red, swollen and itchy hives[3]
- 60% CSU patients experience mental health disorders, mainly depression and anxiety[4]
- QoL impairment comparable to PsO[5] and AD[5]; disrupted sleep reported as one the most burdensome impacts[6]
- ~1/5 patients report having to take time away from work[7]

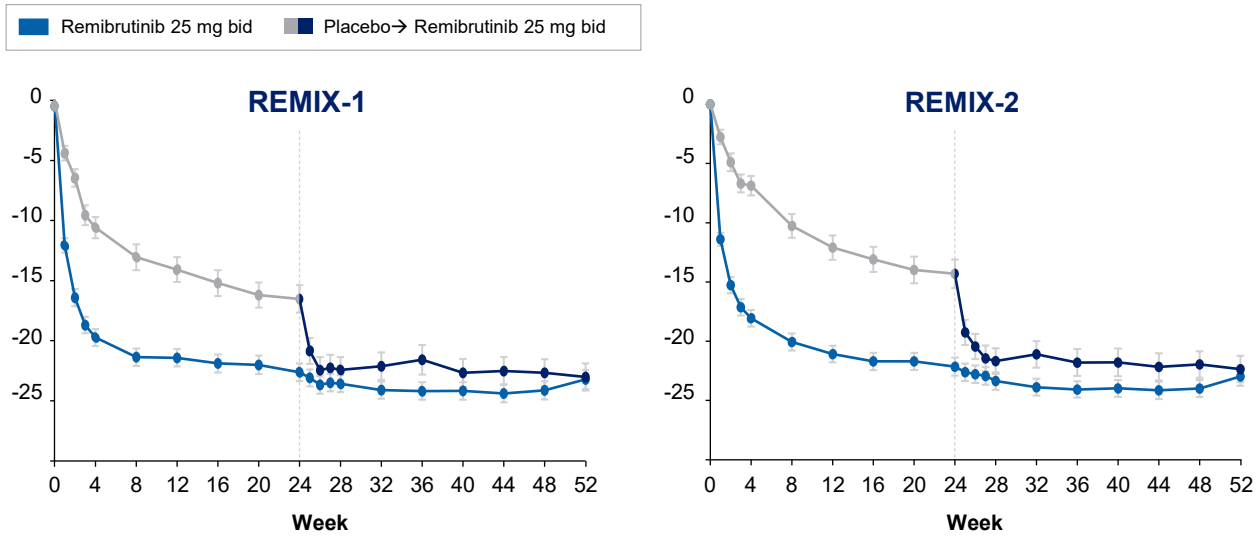
Achieving symptom control as quickly as possible to improve QoL is an important treatment goal for CSU

See page 52 for references (footnotes 1-7). Treated refers to adults with antihistamines and biologics. Uncontrolled despite treatment with H1, H2 antihistamines incl. dose escalation, with treatment at the specialist level. Excludes patients with rare blood disorders, patients taking anticoagulants due to bleeding risk, and other co-morbidities.

Rhapsido® has demonstrated long-term safety and efficacy in CSU, with a fast onset of action

Phase III REMIX studies[1,2]

Change from baseline in UAS7 (mean ± SE)



- Meaningful improvement in **symptom control across all measures**[3], with **results observed as early as Week 1** in post-hoc analyses
- **Favorable safety profile**[4] including balanced LFTs

What we've heard

"Remibrutinib represents a **new way of treating CSU**. By blocking the activity of BTK, remibrutinib stops a key pathway of the immune response in CSU. This is an exciting new option that has the **potential to help a broad range of patients get fast relief.**"

Dr. Mark Lebwohl, MD (Dermatologist)

"The approval of remibrutinib is an **important development in CSU care**. It **quickly reduces symptoms**, offering patients **control of the hives and itching that they experience** on a daily basis."

Dr. Giselle Mosnaim, MD, MS (Allergist)

See page 52 for references (footnotes 1-4).

Phase IIIb US HTH study vs. dupilumab evaluating speed of symptom control, of critical importance to patients

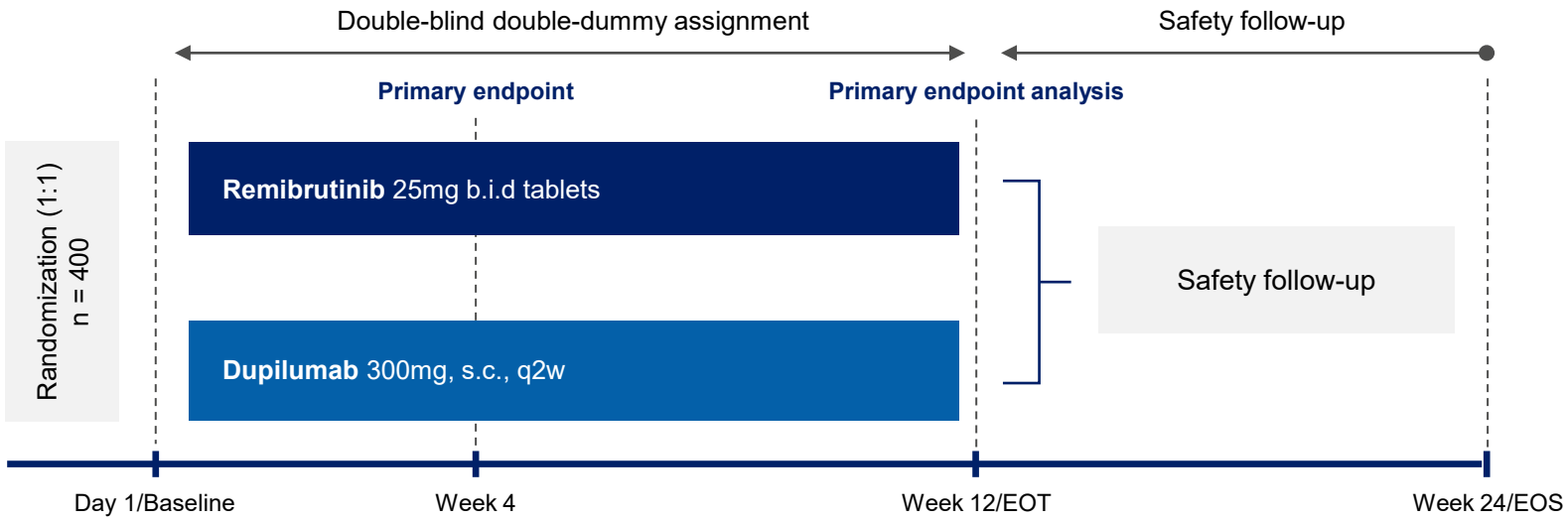
Objective

- Assess the **superiority of Rhapsido vs. dupilumab in CSU** inadequately controlled by H1-antihistamines (AH)
- Primary endpoint: **UAS7** change from baseline at **Week 4**

Status

- Multi-site, US-based study
- Currently recruiting

RECLAIM study design

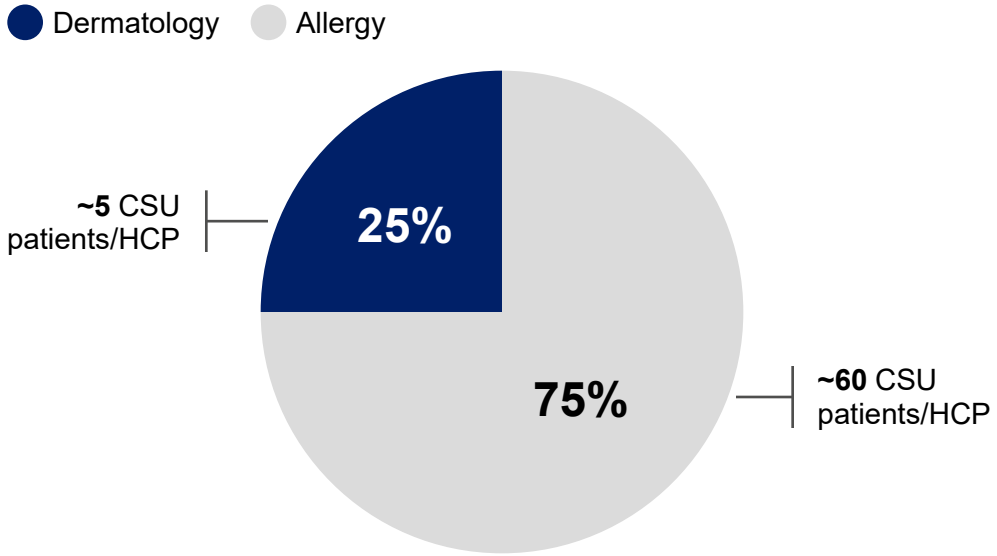


Population: Adult CSU patients inadequately controlled by 2nd gen. H1-AHs
Background: 2nd gen. H1-AHs allowed as background and rescue therapy

Next steps: Readout expected in 2027

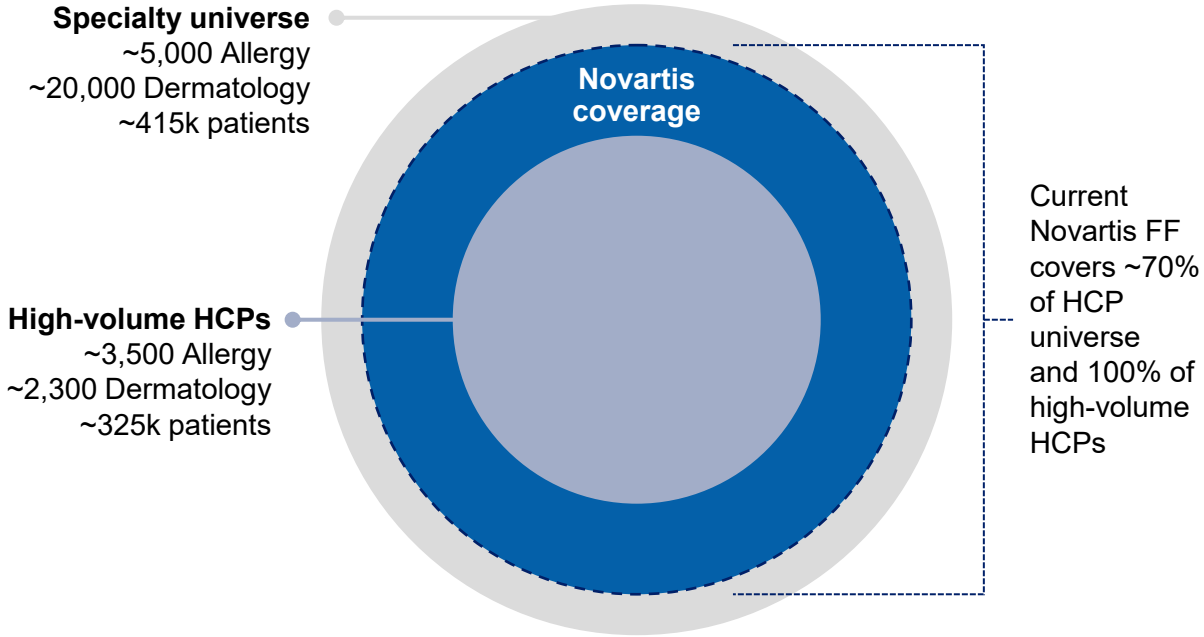
For the US launch, we expect initial uptake from Allergists, followed by Dermatologists – both specialties we know well

Split of target CSU patients by specialty[1]



We expect **Rhapsido to evolve the specialist landscape**, bringing more CSU care into dermatology over time.

Deploying our field force in CSU[1]



See page 53 for references (footnote 1).

Early US launch success factors

1

Engage early prescribers

Target **high-prescribing allergists and dermatologists** who treat ~80%[1] of CSU patients after AH failure

2

Prioritize Rhapsido-ready patients

Focus on **~415k CSU patients uncontrolled on antihistamines**[1] driving **early positive experiences**

~20k patients identified[2] and activated in pre-launch activities

3

Support patient access

Provide a simplified experience with **robust bridge program, sampling**, and rapid coverage expansion

Expect fast uptake once access is established, positioning Rhapsido as the 1L treatment option after AH failure

See page 53 for references (footnotes 1-2).

Leveraging commercial capabilities honed over the last 3 years

1

Customer engagement

Transformed field model, improving customer engagement effectiveness across the portfolio by **+20% for three consecutive years**[1]

2

Patient support

Developed **industry-leading bridge support** to accelerate onboarding

Fully owned Patient Support Program model, resulting in **3-5 days to dispense** on average[2]

3

Market access

Secured **>70% access to label within 6 months** for recent launches[3]

~30 days average[4] conversion from free to paid drug

Compounding capabilities through multiple launches across therapeutic areas

See page 53 for references (footnotes 1-4).

Rhapsido® CSU launch provides foundation for future indication expansion

Indication	Phase I	Phase II	Phase III	Status
CSU				**Launched in US**
CIndU				Readout 2026
HS				Readout 2028
FA				PhII positive, PhIII preparation
MS				Readout 2026
gMG				Readout 2028

Future launches leverage existing infrastructure and capabilities

CIndU
Complete overlap with CSU

HS
Complete overlap Cosentyx HS

FA
Builds on CSU Allergy footprint

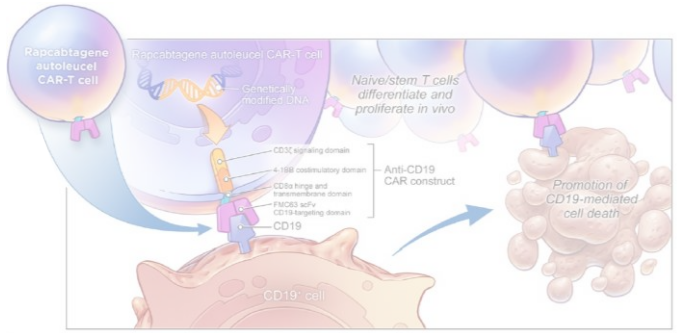
MS & gMG
Builds on Neuroscience footprint

With Rhapsido® approval and ianalumab positive data in SjD, we have derisked two assets with multi-blockbuster potential

YTB323

CAR-T therapy for deep B cell depletion

Lead indication: srSLE/LN



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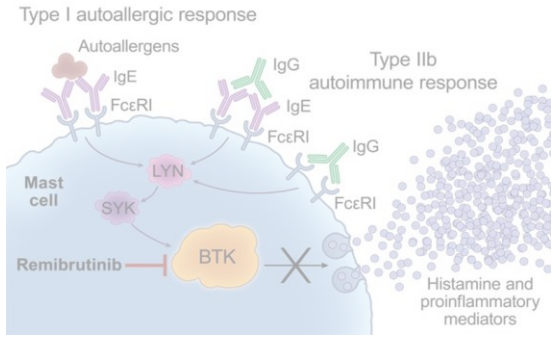

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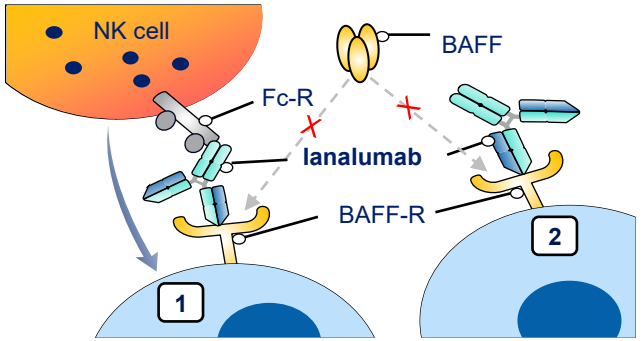

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Investing in broad development programs with high conviction and alignment across RDC continuum

Sjögren's is a severe, systemic, and complex autoimmune disease – far beyond dryness

Heterogenous B cell mediated disease

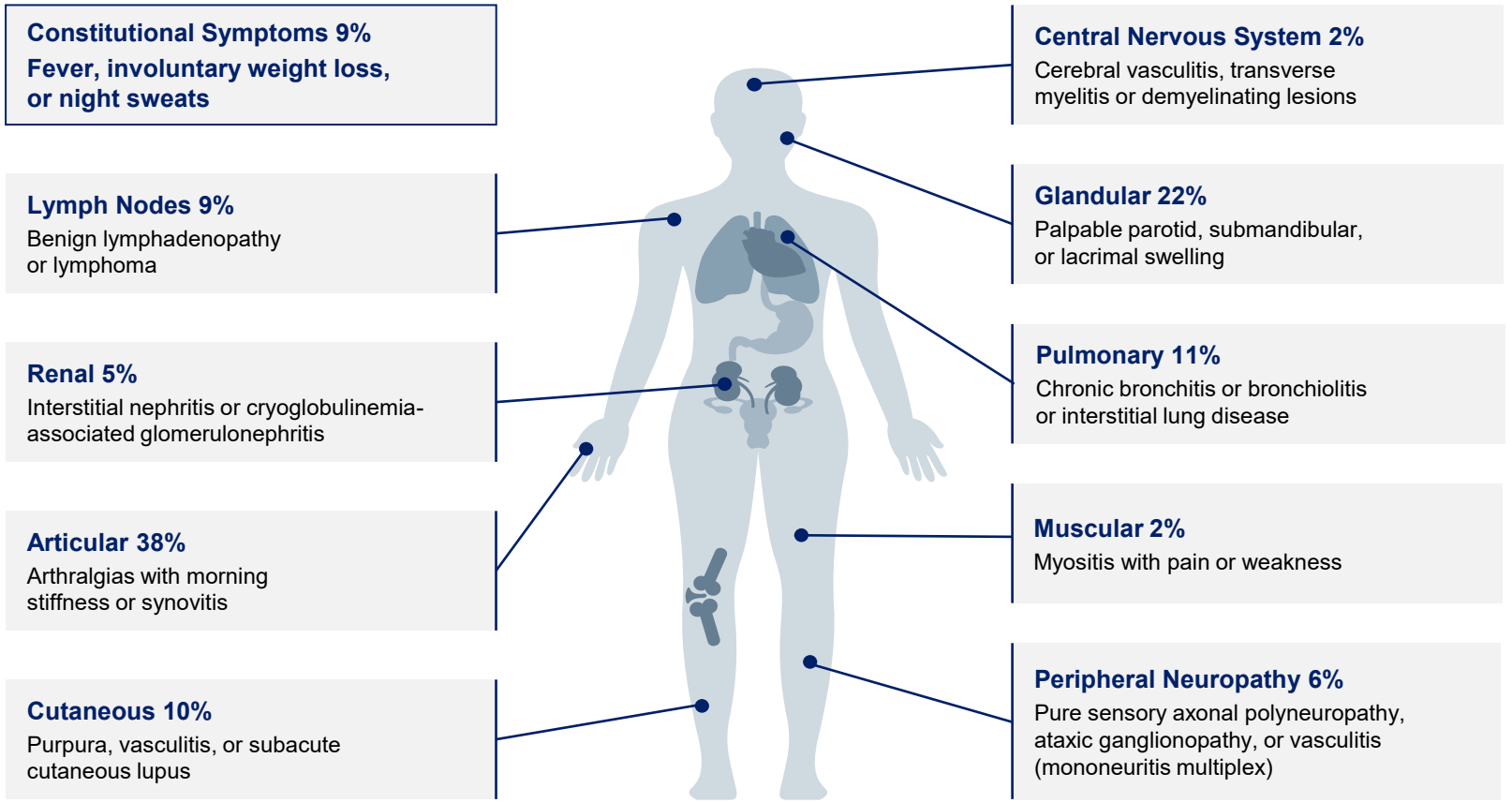
Debilitating eye and mouth dryness, fatigue and joint pain[1]

30-40% suffer potentially irreversible organ and system damage[1]

Reduced quality of life metrics comparable to RA or SLE[1]

Increased mortality including a 20-40x lifetime risk of lymphoma[2]

Percentage of patients with organ manifesations[1]



See page 53 for references (footnotes 1-2).

A lengthy, multi-physician path is common before reaching a SjD diagnosis[1]

Pre-diagnosis (~2–10 years) | **Diagnosis (1-4 months)**

Symptom presentation

Variable presentation, common symptoms and systemic manifestations can wax and wane[2]

Overlapping symptoms with other autoimmune or chronic conditions

Systemic nature often missed: early care splintered across settings (PCP, dental, ophthalmology)

Referral pathways

Very low disease awareness among patients and HCPs outside of Rheumatology

Patients typically see multiple physicians before being referred to rheumatologist

Diagnostic confirmation

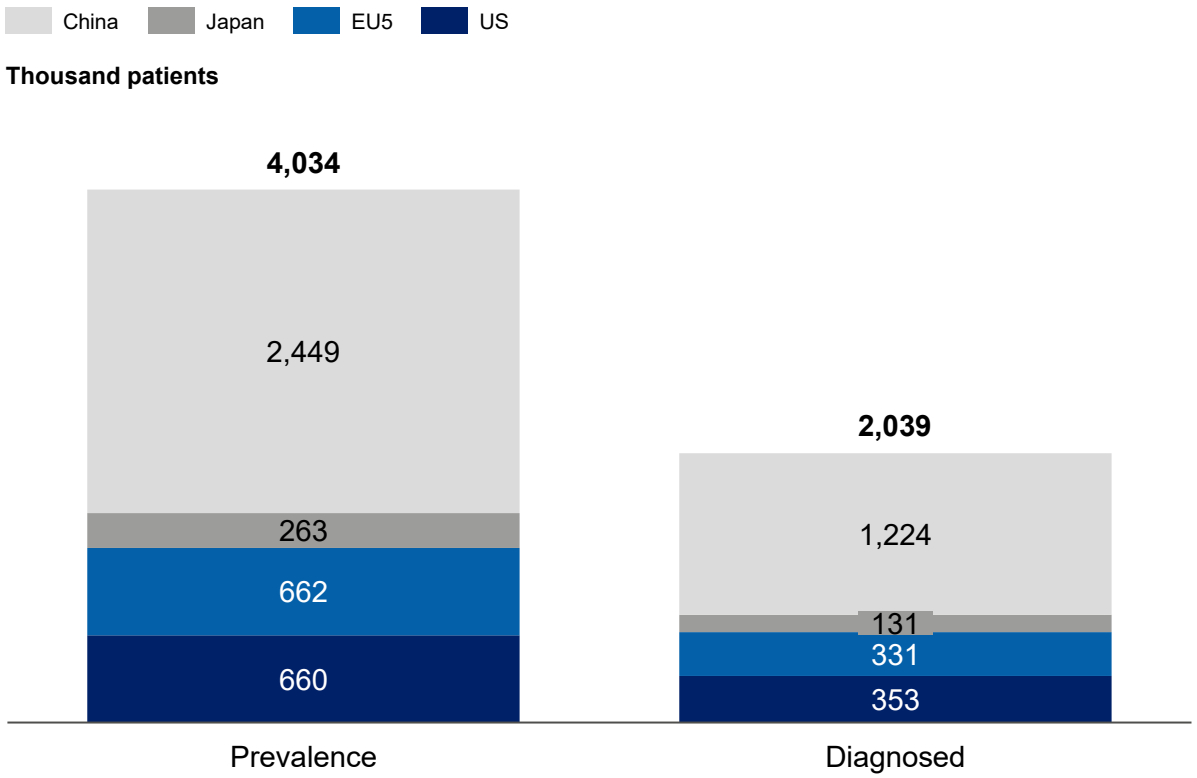
No single conclusive serologic test, which can delay time to a confirmed diagnosis

Requires a combination of tests and clinical symptom evaluations to arrive at SjD diagnosis

See page 53 for references (footnotes 1-2).

SjD is the second largest Rheumatology market after RA, and represents significant unmet need

Epidemiology[1]



Demographics[2]

90% of people with SjD are women

Typically diagnosed between 30-50 years

Current treatment options[3]

Fail to address the wide range of symptoms

Reliance on off-label therapies for systemic disease

Increasing diagnoses

Prevalence likely underestimated due to diagnostic complexity and overlap with other AID[4]

Diagnosed population expected to grow with approval of targeted therapy

See page 54 for references (footnotes 1-4). Prevalence assumes primary Sjogrens patients only. Source: Kantar Health, DRG, Evaluate Pharma, US demand study, Novartis. Data for year 2025. Epidemiology numbers include patients without access.

ESSDAI is the gold standard to assess disease activity in SjD trials

Composition and application in patient care[1]

12 organ-specific domains used to stratify disease activity

Scoring system with low disease activity <5, moderate 5-13, high ≥14

<40% of Rheumatologists have ever utilized ESSDAI in clinical practice[2]

Utilization in clinical trials[3]

Validated endpoint and enrollment criterion in pivotal trials

Used alongside complementary instruments to capture full disease impact:

- PROs (fatigue, dryness, pain),
- global assessments (patient and physician) and
- clinical tests (gland function assessments and serological test)

	Domain (score)	Weighting
Clinical Domains	Constitutional (0-2)	3
	Lymphadenopathy (0-3)	4
	Glandular (0-2)	2
	Articular (0-3)	2
	Cutaneous (0-3)	3
	Pulmonary (0-3)	5
	Renal (0-3)	5
	Muscular (0-3)	6
	Peripheral nervous system (0-3)	5
	Central nervous system (0-3)	5
Laboratory Domains	Hematological (0-3)	2
	Biological (0-2)	1
	Score Total	**0-123**

See page 54 for references (footnotes 1-3).

Systemic disease activity as measured by ESSDAI is related to long-term outcomes and decreased mortality

Higher ESSDAI scores

- Increased risk of damage accrual[1] and adverse outcomes including development of lymphoma[2], interstitial lung disease or CV events[3]
- Linked to poorer quality of life, reduced work productivity, and greater socioeconomic burden[4]

Lower ESSDAI scores

- Sustained ESSDAI reduction associated with preservation of glandular function over time[5]
- Low disease activity in related AIDs (RA and SLE) are well established as improving long-term outcomes and mortality[6,7]

Consistent, clinically relevant and durable reductions in disease activity have the potential to prevent progression of both local and systemic complications

See page 54 for references (footnotes 1-7).

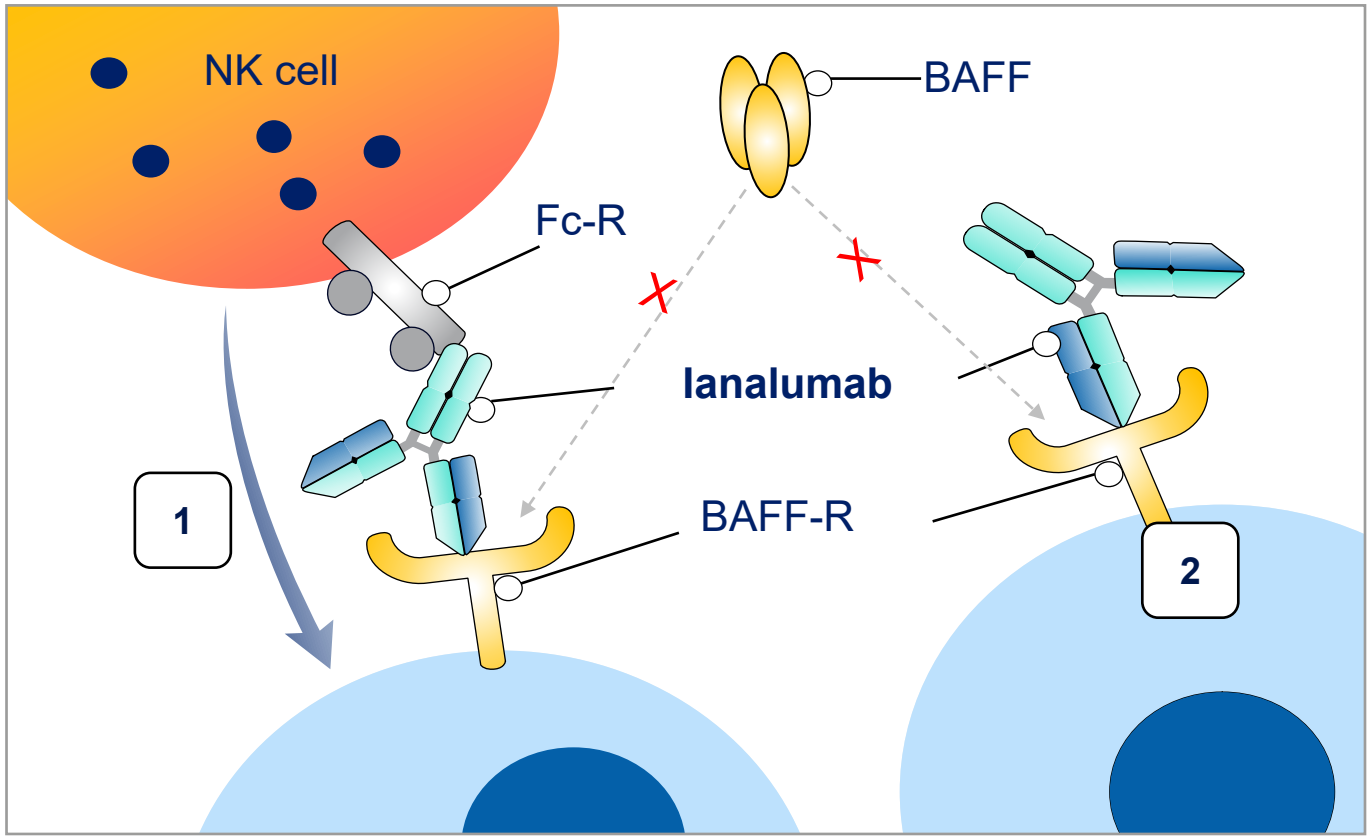
Ianalumab is an afucosylated, fully human, IgG1 mAb targeting B cells through a novel dual mechanism of action

Dual MoA binding to BAFF-R[1]

01	Enhanced depletion of B cells via ADCC
02	Inhibition of B cell activation and survival via BAFF-R blockade[2,3]

Relevance in SjD

B cell hyperactivity and dysregulated BAFF/BAFF-R signaling are hallmarks of SjD pathogenesis[2-4]



See page 55 for references (footnotes 1-4).

Ianalumab achieves deep B-cell depletion in target tissue

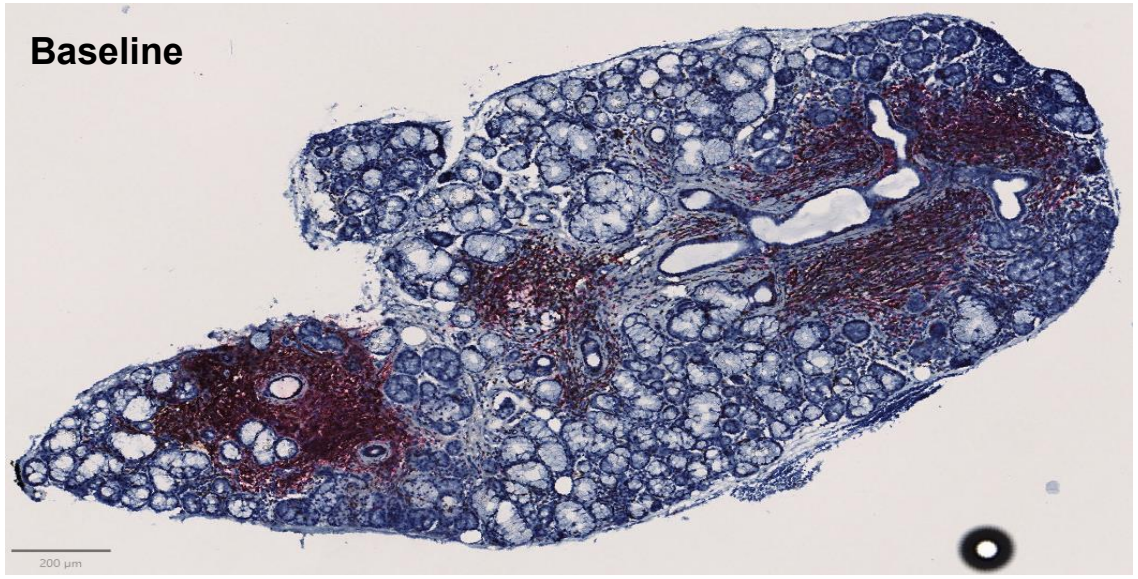
Phase II mechanistic study[1]

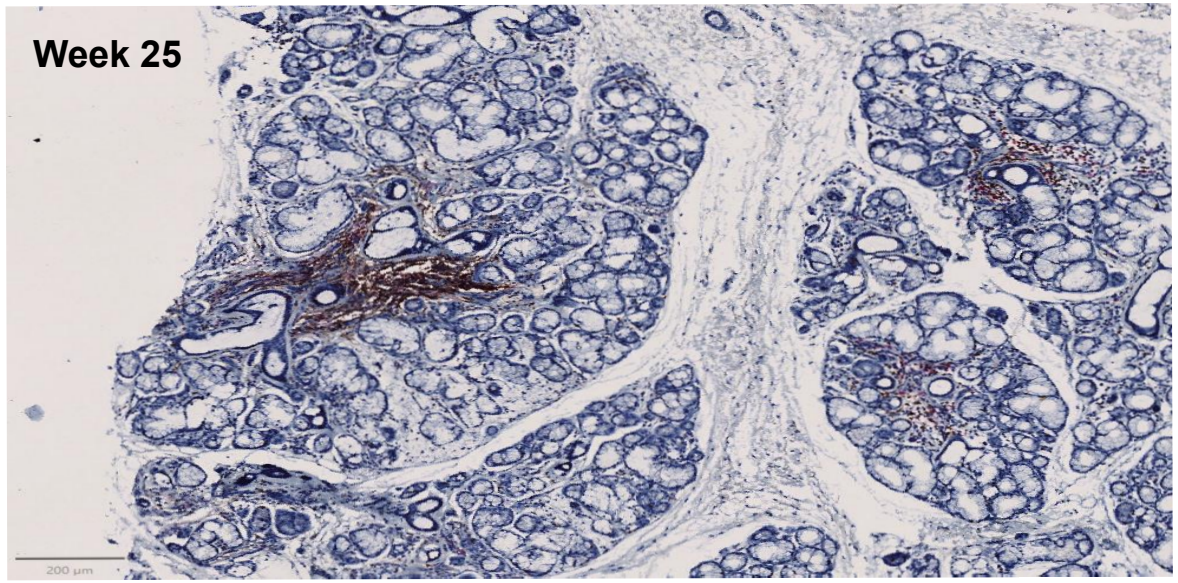
Ianalumab QM reduces lymphocytic infiltrate in salivary gland tissue at Week 25

99% reduction in circulating B cells

84% reduction in salivary gland B cell density

Analysis of labial salivary gland biopsy CD3 / CD20





See page 55 for references (footnote 1).

Phase III NEPTUNUS-1 and NEPTUNUS-2 study designs[1]

Primary endpoint

- ESSDAI change from baseline at Week 48, ianalumab vs. placebo

Key secondary endpoints

- ESSDAI response (≥5 point reduction from baseline)
- ESSDAI low systemic disease activity (ESSDAI score <5)
- Physican (PhGA) and patient reported outcomes (PaGA, ESSPRI, SSSD), stimulated salivary flow (sSF)
- Safety and tolerability up to Week 52

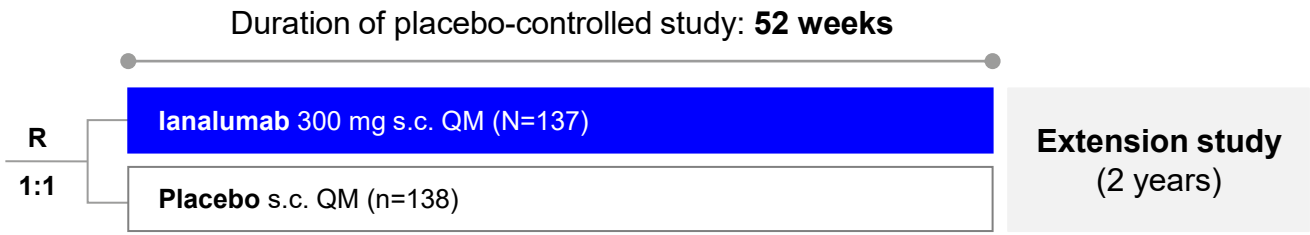
Pooled analysis

- Ianalumab QM data was pooled in a pre-specified statistical analysis plan

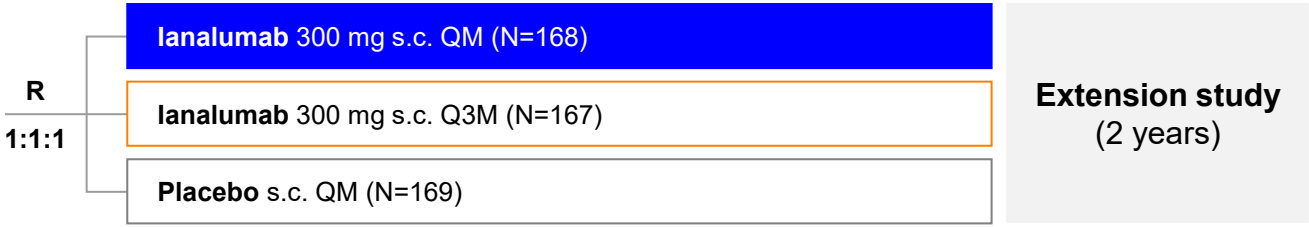
Baseline characteristics

- Generally balanced between treatment groups

NEPTUNUS 1 study design



NEPTUNUS 2 study design

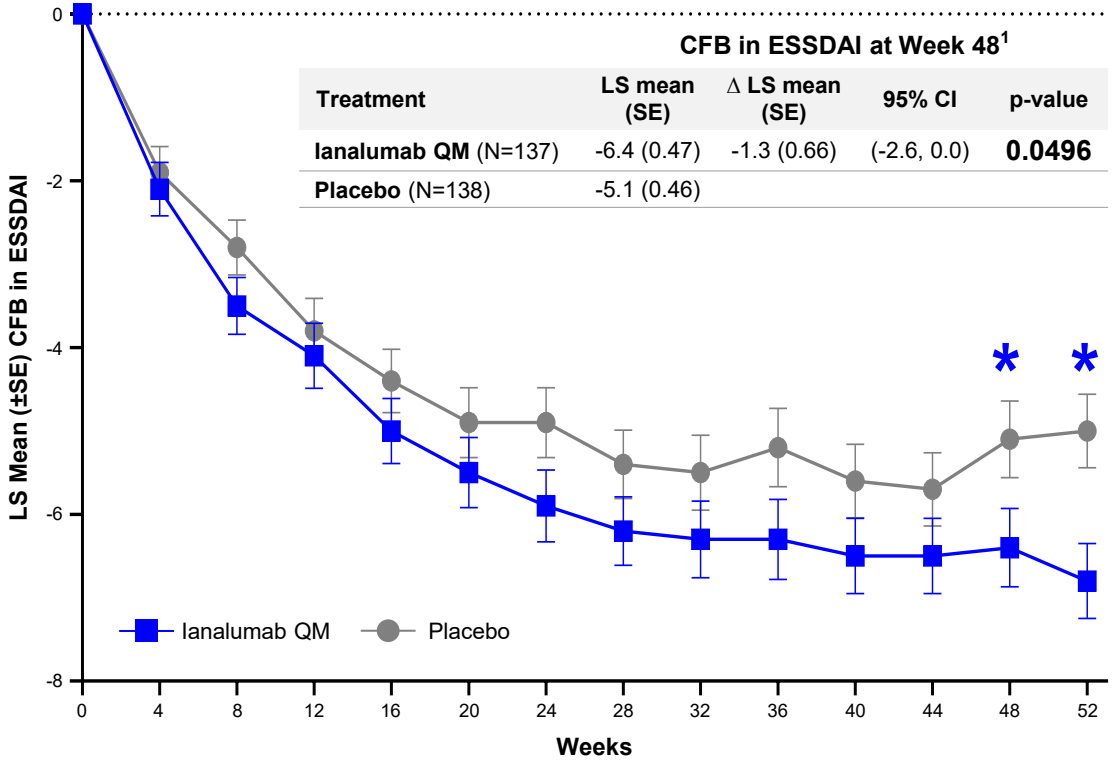


Patient population: Adult SjD patients with moderate to high disease activity (ESSDAI ≥ 5). All arms could continue concomitant background therapy at investigator's discretion.

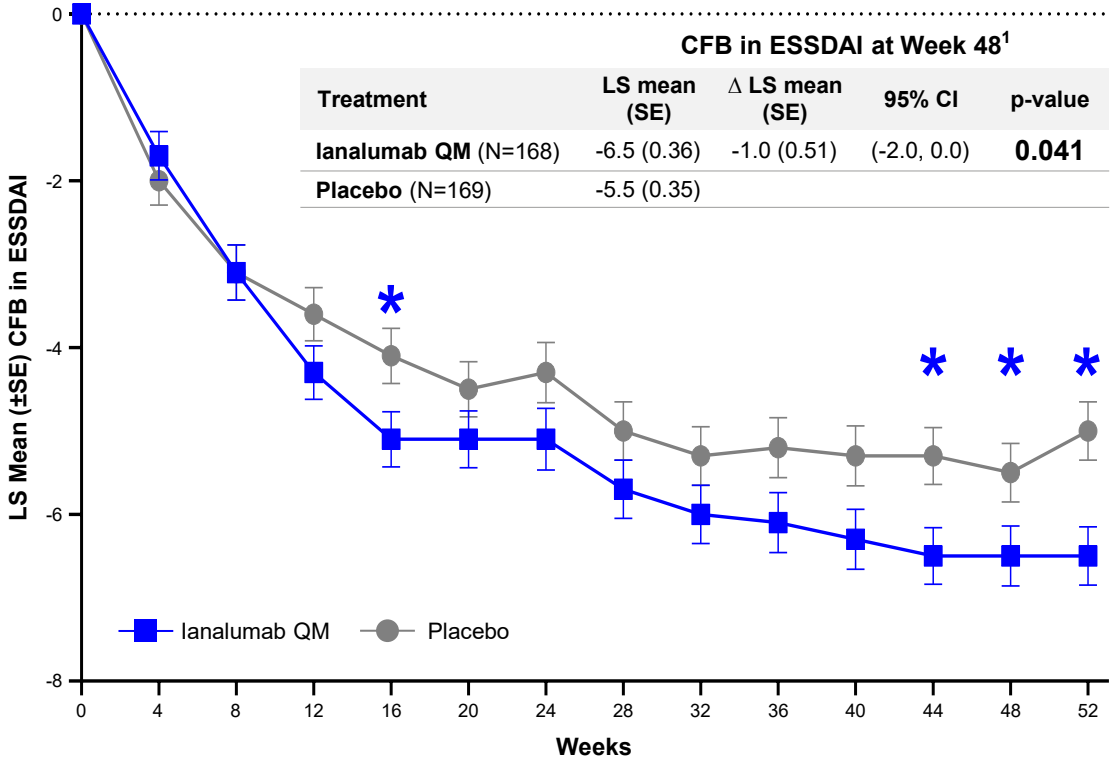
See page 55 for references (footnote 1).

Ianalumab QM demonstrated statistically significant improvement in ESSDAI in both NEPTUNUS studies[1]

NEPTUNUS-1



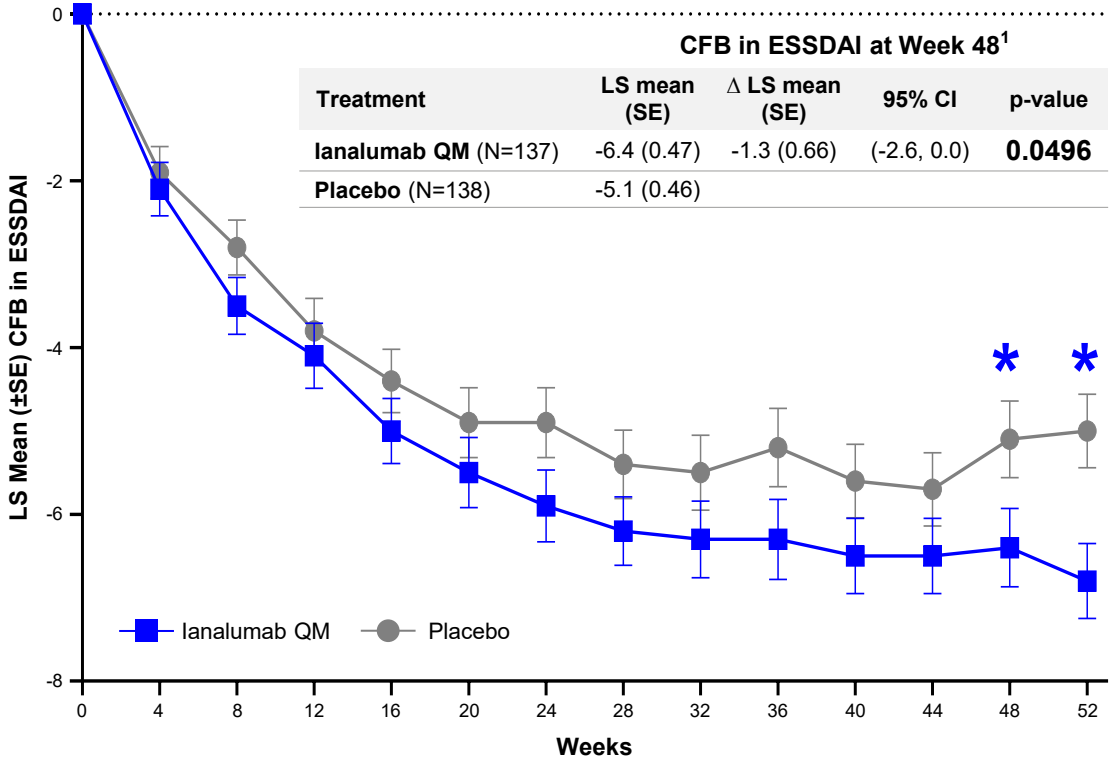
NEPTUNUS-2



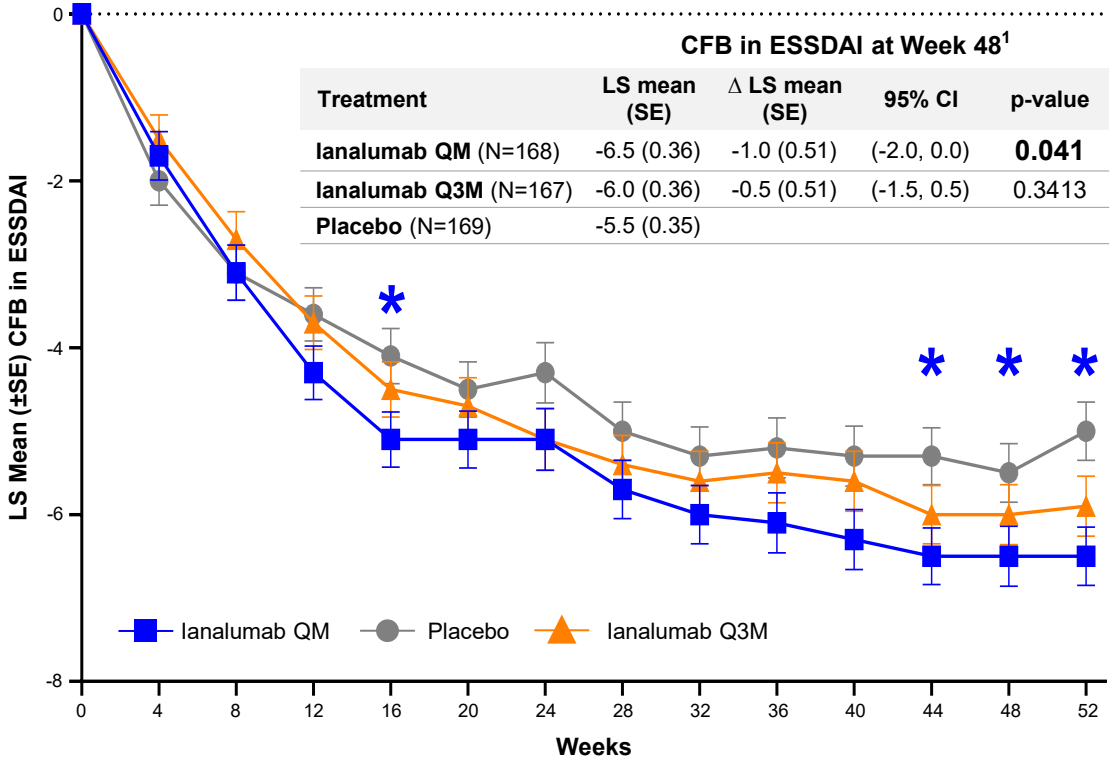
See page 55 for references (footnote 1). *p<0.05. 1. ANCOVA model with study treatment, ESSDAI strata, and region as factors and baseline score as covariate. N, number of patients in each treatment group of the specified analysis set; n, number of patients with evaluable data.

Ianalumab QM demonstrated statistically significant improvement in ESSDAI in both NEPTUNUS studies[1]

NEPTUNUS-1



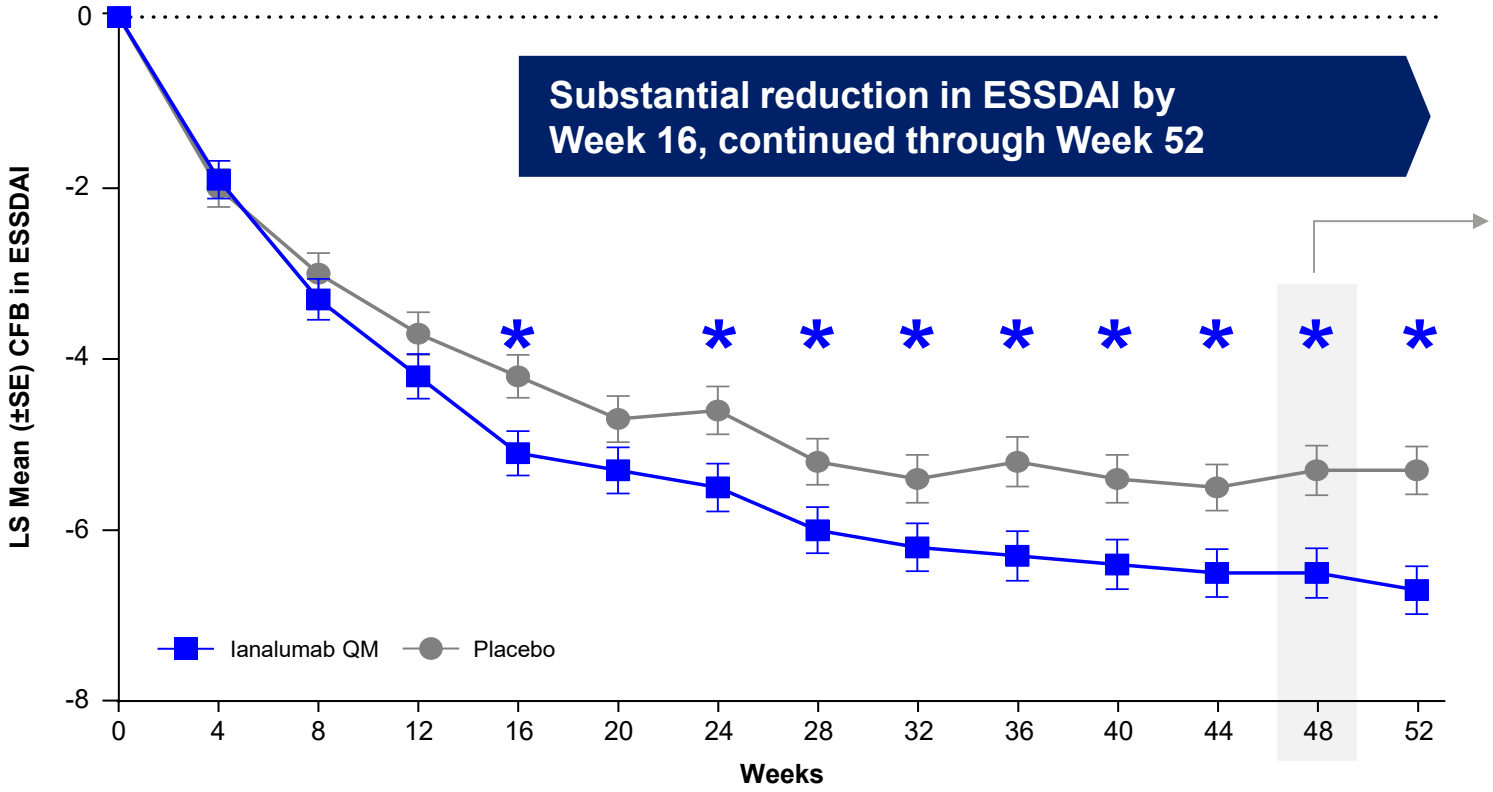
NEPTUNUS-2



See page 55 for references (footnote 1). *p<0.05. 1. ANCOVA model with study treatment, ESSDAI strata, and region as factors and baseline score as covariate. N, number of patients in each treatment group of the specified analysis set; n, number of patients with evaluable data.

Ianalumab QM showed rapid and sustained reduction in disease activity compared to placebo in the pooled analysis[1]

Pooled NEPTUNUS data



CFB in ESSDAI at Week 48

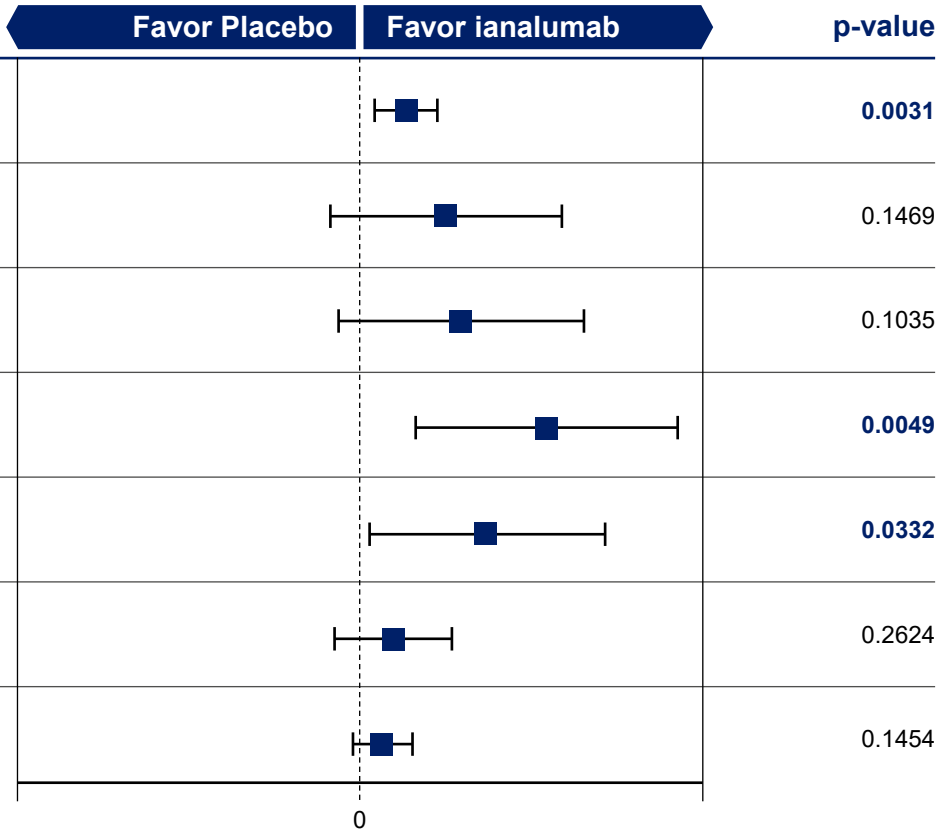
Treatment	LS mean (SE)	Δ LS mean (SE)	95% CI	p-value
Ianalumab QM (N=305)	-6.5 (0.29)	-1.2 (0.41)	(-2.0, -0.4)	0.0031
Placebo (N=307)	-5.3 (0.29)			

See page 55 for references (footnote 1). *p<0.05. 1. ANCOVA model with study treatment, ESSDAI strata, and region as factors and baseline score as covariate. N, number of patients in each treatment group of the specified analysis set; n, number of patients with evaluable data.

Ianalumab QM showed consistent improvement in continuous secondary endpoints over placebo at Week 48[1]

Pooled NEPTUNUS data

Endpoint: Treatment group	LS mean	Difference in LS mean (95% CI)	p-value
ESSDAI: Total score			
Ianalumab QM (N=305)	-6.5	-1.2 (-2.0, -0.4)	**0.0031**
Placebo (N=307)	-5.3		
SSSD: 4-item unweighted summary score			
Ianalumab QM (N=305)	-1.52	-0.23 (-0.53, 0.08)	0.1469
Placebo (N=307)	-1.29		
ESSPRI: Total score			
Ianalumab QM (N=305)	-1.73	-0.26 (-0.58, 0.05)	0.1035
Placebo (N=307)	-1.47		
PaGA: Visual analog scale (0-100)			
Ianalumab QM (N=305)	-13.5	-4.9 (-8.3, -1.5)	**0.0049**
Placebo (N=307)	-8.7		
PhGA: Visual analog scale (0-100)			
Ianalumab QM (N=305)	-29.2	-3.3 (-6.4, -0.3)	**0.0332**
Placebo (N=307)	-25.9		
FACIT-F: Total score			
Ianalumab QM (N=305)	7.6	0.9 (-0.7, 2.4)	0.2624
Placebo (N=307)	6.8		
Whole salivary flow rate (mL/min): Stimulated			
Ianalumab QM (N=305)	0.121	0.059 (-0.020, 0.137)	0.1454
Placebo (N=307)	0.063		

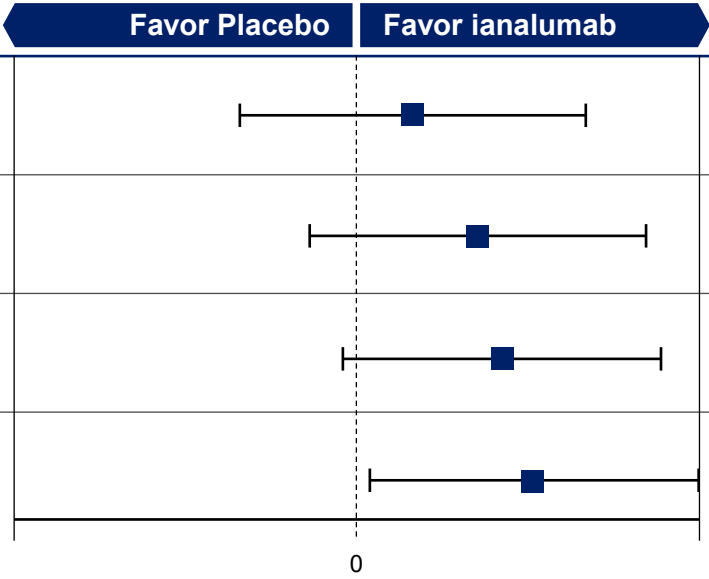


See page 55 for references (footnote 1). For presentation purpose, the lines of LS mean difference (95% CI) are multiplied by -1 for ESSDAI, PHGA and PAGA; lines for stimulated salivary flow rates are scaled up with multiplying by 10; and multiplied by -10 for ESSPRI and SSSD. N, number of patients in each treatment group of the specified analysis set

Ianalumab QM showed consistent numerical improvement in binary secondary endpoints over placebo at Week 48[1]

Pooled NEPTUNUS data

	Endpoint: Treatment group	**Estimated response proportion** (%)	**Estimated difference (95% CI)**	p-value
1	**SSSD response** Ianalumab QM (N=279) Placebo (N=276)	 38.5 35.9	 2.6 (-5.5, 10.6)	 0.5335
2	**ESSPRI response** Ianalumab QM (N=280) Placebo (N=271)	 42.5 36.9	 5.6 (-2.3, 13.5)	 0.1650
	ESSDAI >= 5 points reduction Ianalumab QM (N=305) Placebo (N=307)	 59.2 52.4	 6.8 (-0.7, 14.2)	 0.0748
	ESSDAI < 5 Ianalumab QM (N=305) Placebo (N=307)	 53.4 45.1	 8.3 (0.6, 15.9)	 **0.0342**



Response definitions

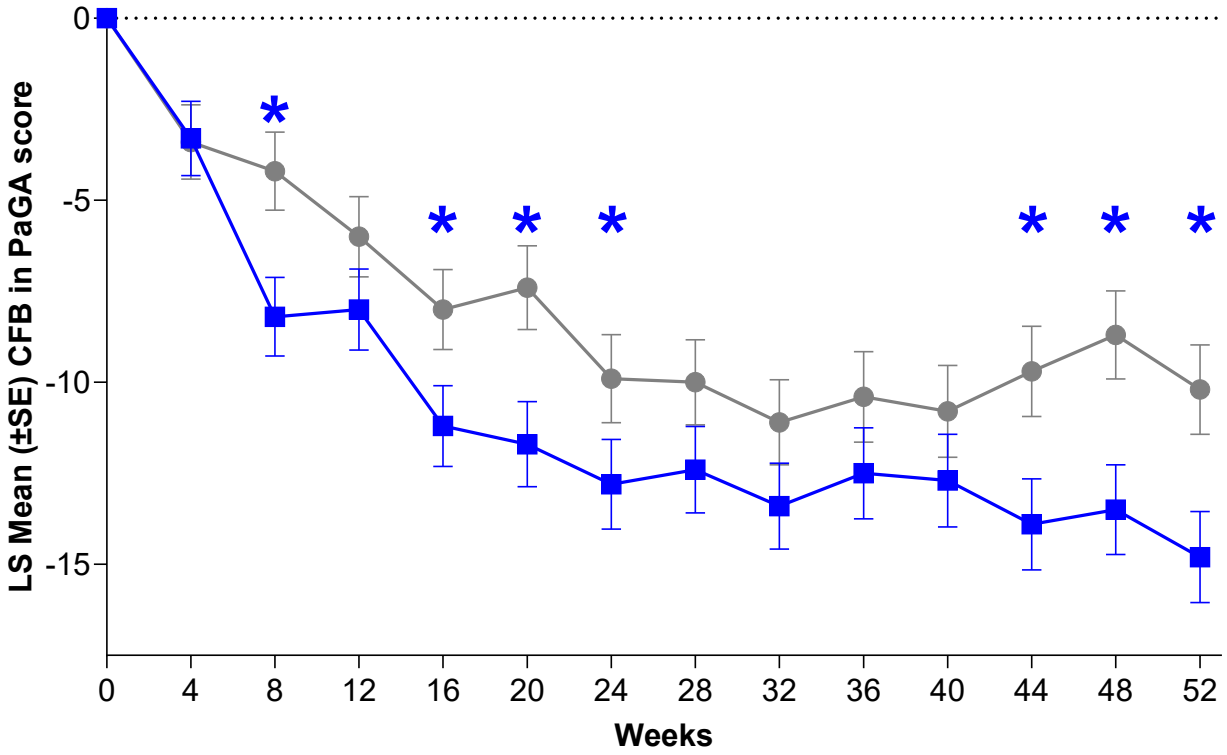
1 | **SSSD:** ≥2-point reduction in patients with baseline ≥3

2 | **ESSPRI:** ≥ 2.3-point reduction in patients with baseline ≥3

See page 55 for references (footnote 1). N, number of patients in each treatment group of the specified analysis set

Ianalumab QM delivered fast and sustained symptom relief (as measured by PaGA) as early as Week 8 and up to Week 52

Pooled NEPTUNUS data



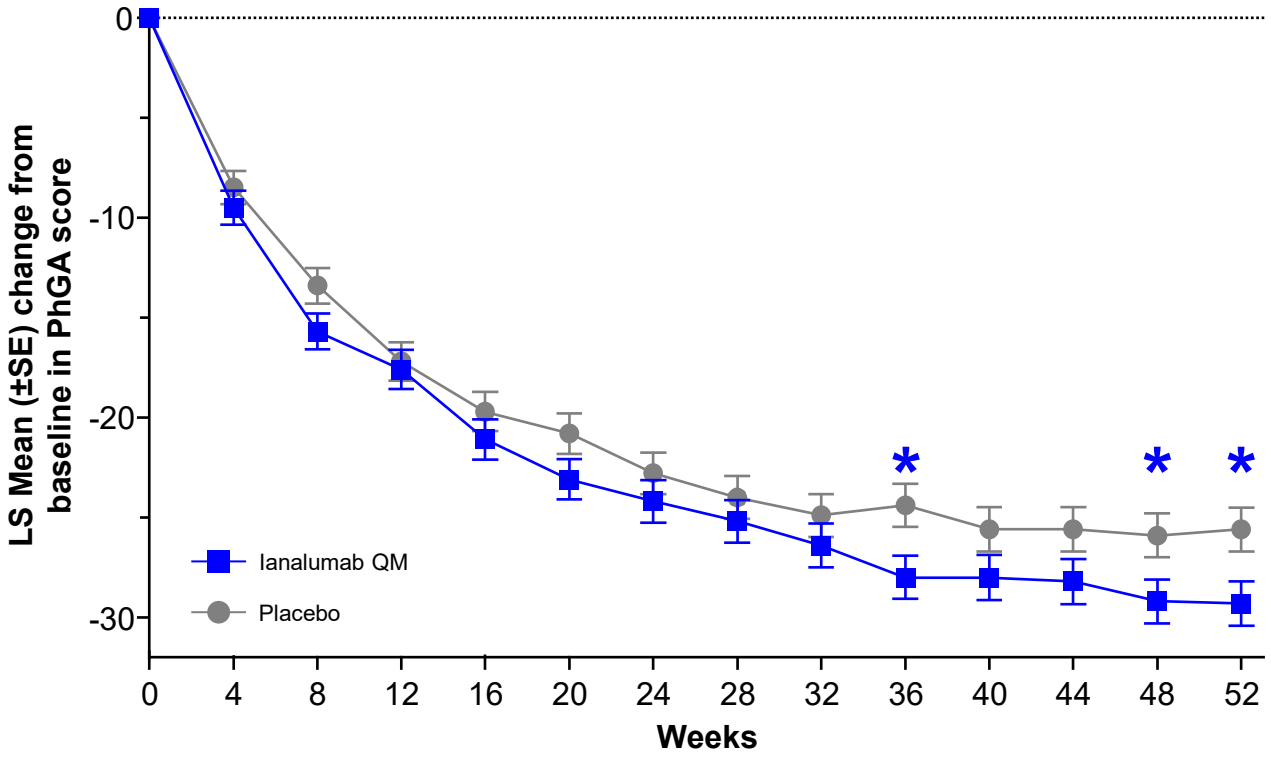
CFB in PaGA at Week 48

Treatment	LS mean (SE)	Δ LS mean (SE)	95% CI	p-value
Ianalumab QM (N=305)	-13.5 (1.23)	-4.9 (1.73)	(-8.3, -1.5)	**0.0049**
Placebo (N=307)	-8.7 (1.21)			

See page 55 for references (footnote 1). *Indicates significant treatment effect observed with nominal p-value <0.05. N, number of patients in each treatment group of the specified analysis set; n, number of patients with evaluable data.

Ianalumab QM demonstrated a greater improvement in physician's assessment of disease burden (PhGA)[1]

Pooled NEPTUNUS data



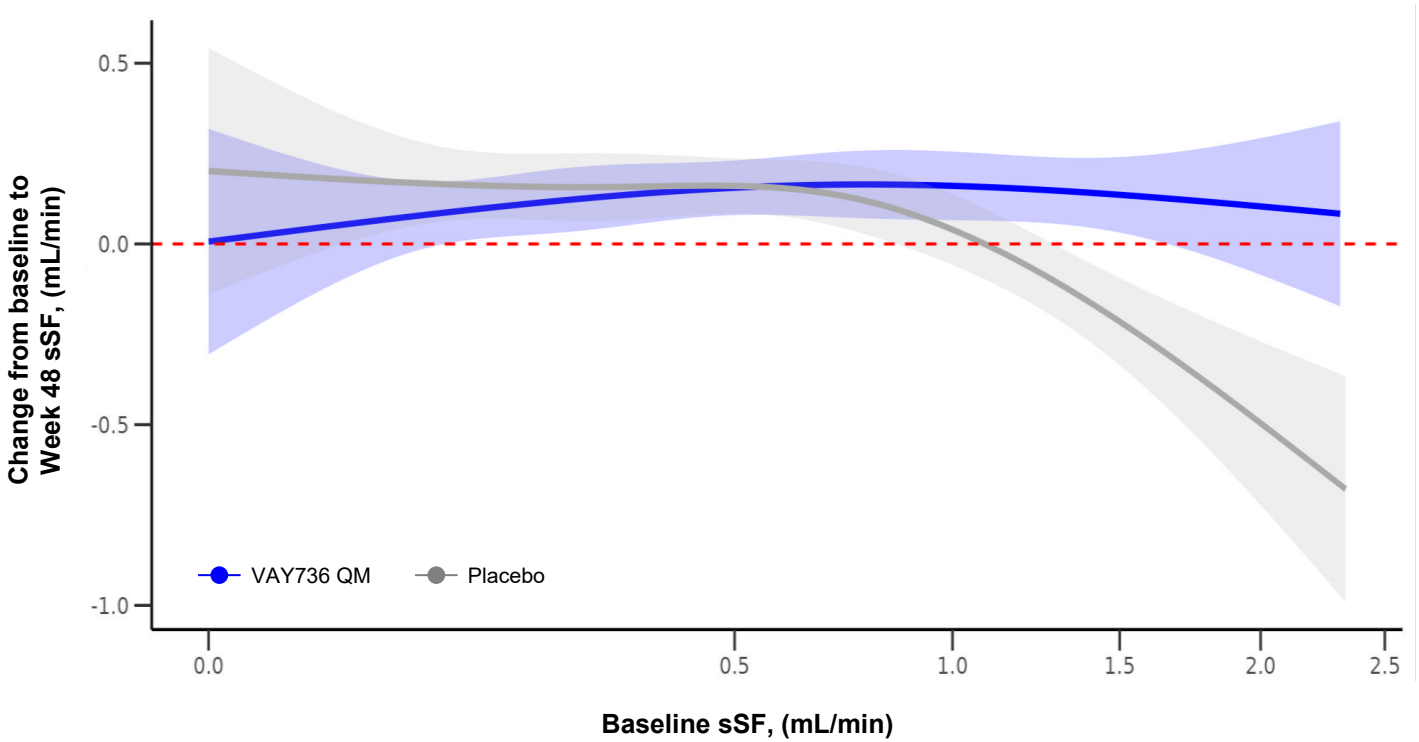
CFB in PhGA at Week 48

Treatment	LS mean (SE)	Δ LS mean (SE)	95% CI	p-value
Ianalumab QM (N=305)	-29.2 (1.10)	-3.3 (1.55)	(-6.4, -0.3)	**0.0332**
Placebo (N=307)	-25.9 (1.09)			

See page 55 for references (footnote 1). *Indicates significant treatment effect observed with nominal p-value <0.05. N, number of patients in each treatment group of the specified analysis set; n, number of patients with evaluable data.

Ianalumab QM preserved salivary function and prevented disease progression on the salivary gland[1]

Pooled NEPTUNUS data: Week 48 change in sSF



Ianalumab preserved salivary function

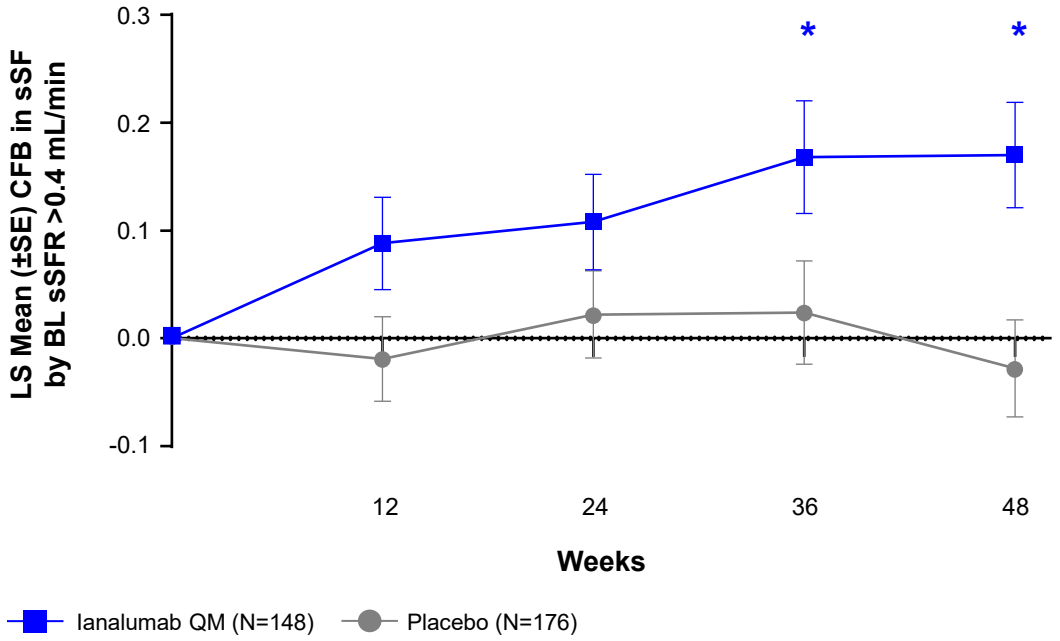
- Ianalumab maintained stimulated salivary flow (sSF) at Week 48 regardless of baseline rates
- Placebo showed declines in patients with functioning salivary glands at baseline; minimal change seen in those with reduced function

See page 55 for references (footnote 1).

Patients with sSF >0.4 mL/min at baseline showed improvements in oral dryness (SSSD) with ianalumab in a post hoc pooled analysis[1]

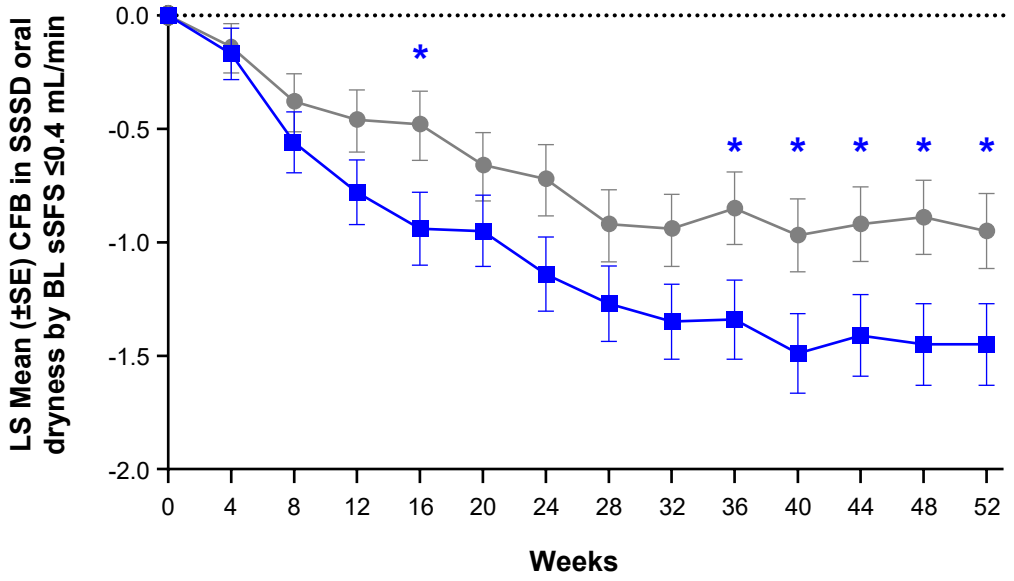
Flow rate

Patients with baseline **sSF >0.4 mL/min**



Oral dryness (SSSD)

Patients with baseline **sSF >0.4 mL/min**



See page 55 for references (footnote 1). *Indicates a significant treatment effect observed with a nominal p-value of <0.05; #[0.4 mL/min = median of pooled BL data].

Ianalumab showed a favorable safety profile with AEs and SAEs comparable to placebo[1]

	NEPTUNUS-1		NEPTUNUS-2		
Category[2]#	**Ianalumab QM (N=137), n (%)**	**Placebo (N=138), n (%)**	**Ianalumab QM (N=168), n (%)**	**Ianalumab Q3M (N=167), n (%)**	**Placebo (N=169) n (%)**
AEs	116 (84.7)	111 (80.4)	146 (86.9)	145 (86.8)	145 (85.8)
AEs related to study treatment	62 (45.3)	48 (34.8)	90 (53.6)	82 (49.1)	69 (40.8)
AEs leading to treatment discontinuation	5 (3.6)	5 (3.6)	14 (8.3)	11 (6.6)	6 (3.6)
SAEs	**5 (3.6)**	**12 (8.7)**	**16 (9.5)**	**13 (7.8)**	**18 (10.7)**
Death	**0**	**1 (0.7)**	**0**	**0**	**0**
Infections	**78 (56.9)**	**81 (58.7)**	**92 (54.8)**	**98 (58.7)**	**113 (66.9)**
Serious infections	3 (2.2)	1 (0.7)	5 (3.0)	5 (3.0)	8 (4.7)
Opportunistic infections[2]	-	-	1 (0.6)	1 (0.6)	1 (0.6)
Malignant neoplasms					
Waldenstrom's macroglobulinemia (PT)	0	1 (0.7)	-	-	-
Tubular breast carcinoma (PT)	-	-	1 (0.6)	0	0
Adrenal neoplasm (PT)	-	-	0	1 (0.6)	0
Intraductal proliferative breast lesion (PT)	-	-	0	1 (0.6)	0
Squamous cell carcinoma (PT)	-	-	0	1 (0.6)	0

See page 55 for references (footnotes 1-3). N, total number of patients per group; n, number of patients with an event.

Ianalumab demonstrated clinically meaningful benefit in SjD

Results summary: NEPTUNUS 1 & 2

First-ever successful Phase III studies in SjD

- Statistically significant ESSDAI improvement in both NEPTUNUS trials
- Rapid, sustained disease activity reduction vs. placebo in the pooled analysis

Consistent improvements in secondary endpoints

- More patients achieved low ESSDAI disease activity
- Reduced patient-assessed disease burden (PaGA)
- Reduced physician-assessed disease burden (PhGA)
- Numerical improvements in dryness, pain, fatigue (SSSD, ESSPRI)

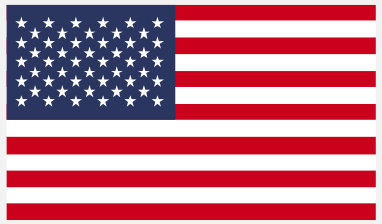
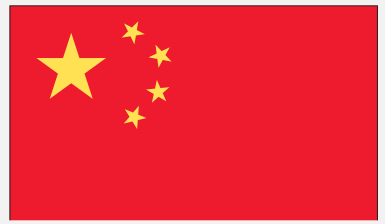
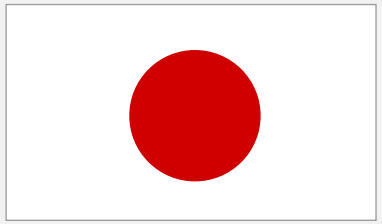
Impact on symptoms of importance to patients

- Preserved salivary function and prevented disease progression on salivary gland
- Improved oral dryness (SSSD) in patients with baseline saliva production (post hoc analysis)

Favorable safety profile

- AEs and SAEs comparable to placebo

Global regulatory submissions H1 2026

	 US	 EU	 China	 Japan
Submission	Q1 2026	Q1 2026	Q1 2026	Q2 2026
	FDA Fast Track designation received 2016			

Building on NEPTUNUS studies, launching evidence generation plan to further demonstrate systemic and symptomatic relief

Selected elements of evidence generation strategy

NEPTUNUS extension study[1] to assess real-world effectiveness and safety

Initiated 2025

Exploring future studies to assess ianalumab's impact across diverse SjD populations

Under consideration

Comprehensive characterization of ianalumab efficacy and safety across disease activity, control, flares, endpoints and subpopulations[2]

Publications planned in 2026

See page 56 for references (footnotes 1-2).

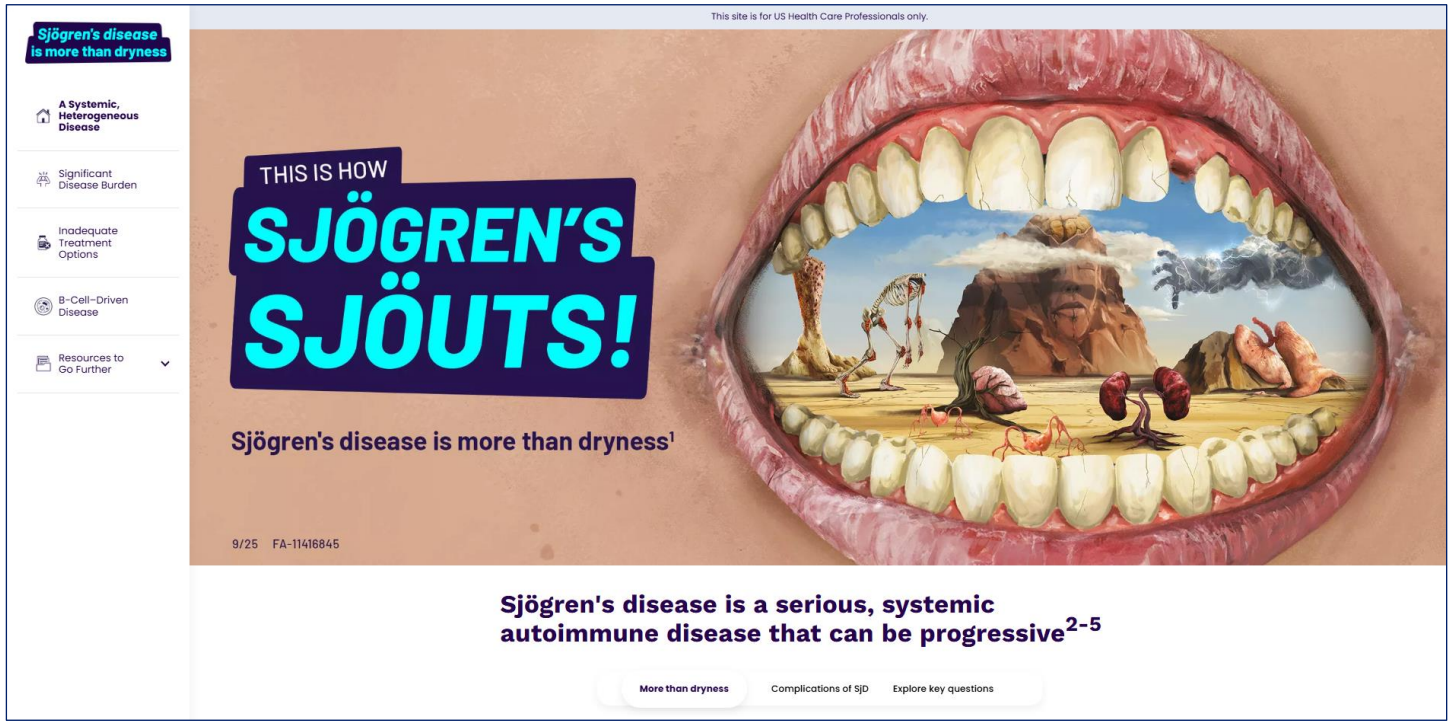
Launching disease state education to increase recognition of SjD as a serious, systemic, autoimmune disease[1]

Context

- Lack of approved therapies
- Low familiarity with clinical endpoints

Opportunity

- Expand understanding of systemic nature and burden of SjD
- Provide framework for physicians to identify moderate to severe patients
- Engage and empower SjD patients



See page 56 for references (footnote 1).

For the US launch, we expect initial adoption from Rheumatologists who are current biologic users, a segment we know well

Focus at launch[1]

Early adopters

Early biologic users

Proactive, use off-label biologics early to prevent disease progression

~15% of HCPs

~35% of SjD patients

Later biologic users

Reactive, rely on frameworks to identify biologic-ready patients

~20% of HCPs

~35% of SjD patients

Later adopters

Symptom-focused HCPs

Focused on symptom relief, typically do not use off-label biologics

~65% of HCPs

~30% of SjD patients

Disease state education, patient activation and ianalumab approval expected to shift more symptom-focused HCPs into biologic users

>90% overlap between Cosentyx/Ilaris field force and Rheumatologists treating SjD; ~100% coverage of early adopters[1]

See page 56 for references (footnote 1).

With increasing awareness of SjD, opportunity to engage and empower moderate to severe patients with organ involvement

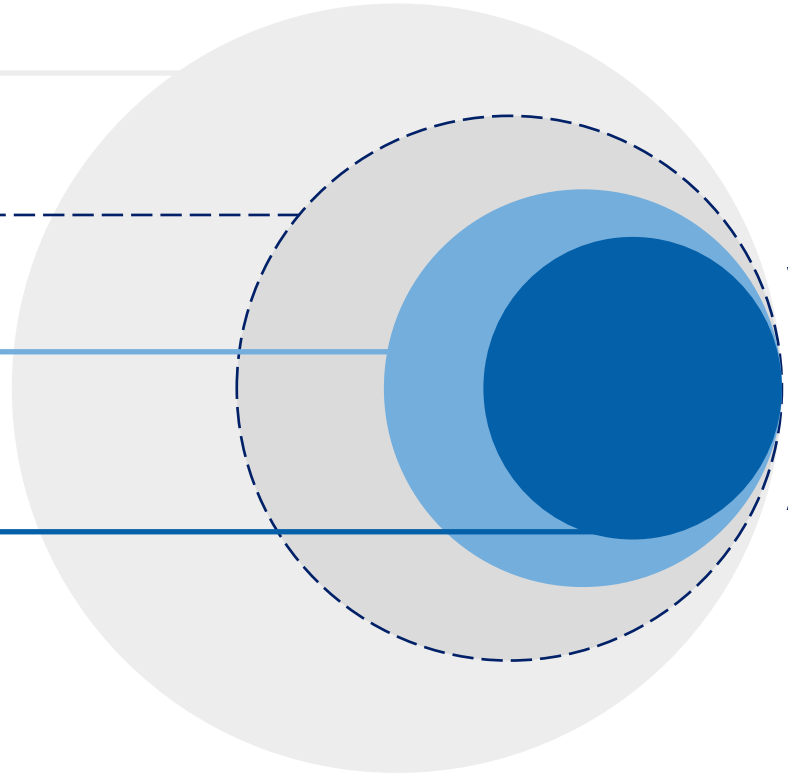
US SjD prevalence

Overall prevalence[1]:
~0.25% = ~660k patients

Diagnosed patients[2]:
~350k patients

Under Rheumatologist care[2]:
~175k patients

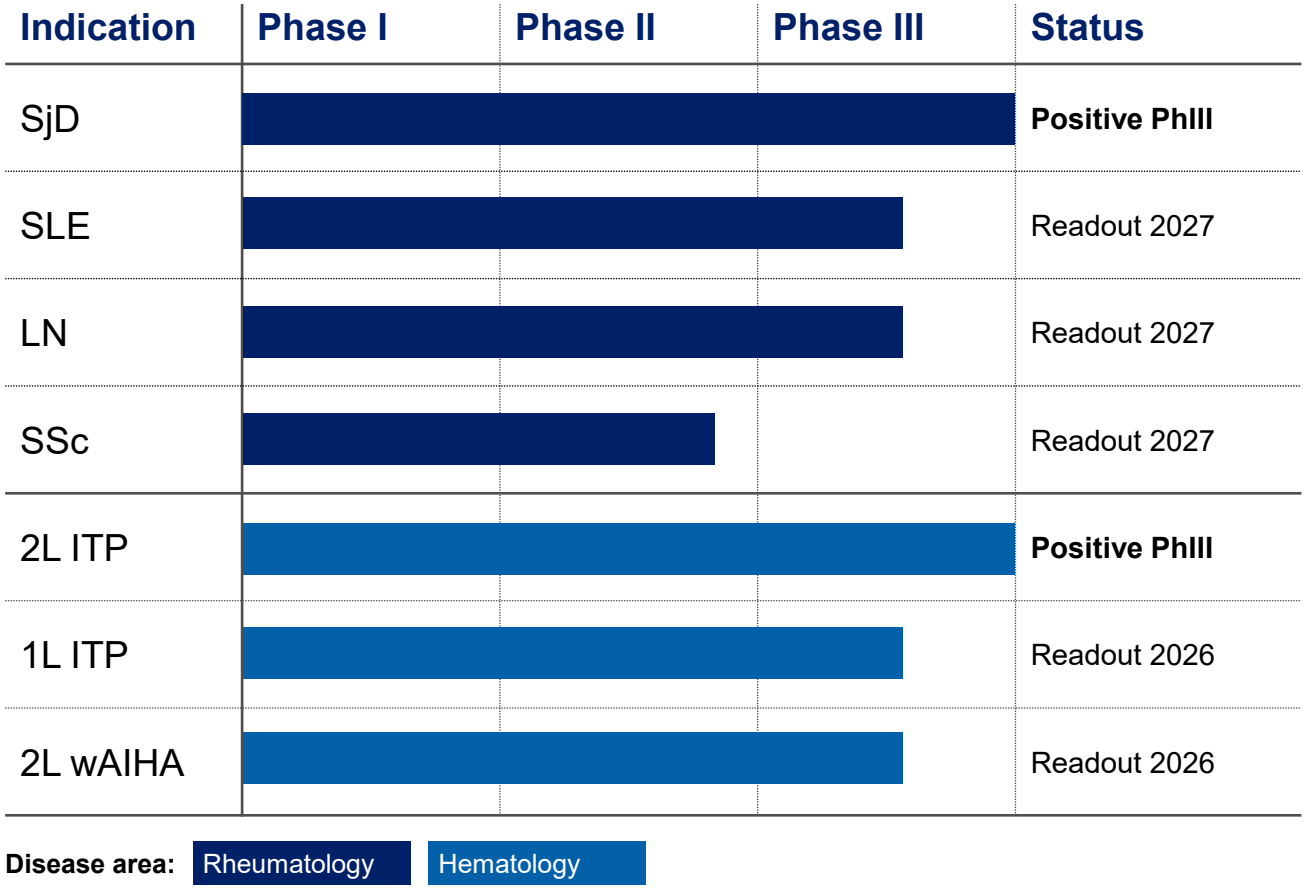
With organ / system involvement[2]:
~100k-115k patients



Targeting 30-40% of diagnosed SjD patients with organ / system involvement[3] (~55-65% under Rheumatologist care)

See page 56 for references (footnotes 1-3).

Positive Phase III study in SjD, a highly heterogenous disease, increases confidence in other B cell-driven diseases



Future launches leverage existing infrastructure and capabilities

SLE
Builds on Cosentyx Rheumatology experience

LN
Builds on Rheumatology and Nephrology

SSc
High overlap with Rheumatology

ITP & wAIHA
Builds on Promacta, Hematology footprint

Key takeaways

Broad and deep immunology pipeline	Rhapsido poised for strong CSU launch	Ianalumab demonstrated meaningful benefit in SjD	Positive SjD data de-risks ianalumab LCM	Compounding commercial capabilities
With multiple late-stage assets targeting areas of high unmet need	As first oral option post-antihistamine failure; multiple LCM readouts starting next year	Consistent across studies, over time, and across patient- and physician-reported outcomes	Across B cell diseases, supporting multi-blockbuster potential	To drive launch excellence and maximize pipeline value

Appendix

Baseline characteristics were generally balanced between treatment groups

Characteristics	NEPTUNUS-1 (N=275)		NEPTUNUS-2 (N=504)		
	Ianalumab QM N=137	Placebo N=138	Ianalumab QM N=168	Ianalumab Q3M N=167	Placebo N=169
Age (years)[1]	48.8 (11.7)	48.0 (13.8)	49.6 (11.4)	50.4 (12.7)	50.9 (12.8)
<65 years, n (%)	125 (91.2)	126 (91.3)	149 (88.7)	145 (86.8)	143 (84.6)
Female, n (%)	127 (92.7)	127 (92.0)	158 (94.0)	159 (95.2)	161 (95.3)
ESSDAI[1]	12.7 (6.81)	12.6 (6.73)	11.7 (5.8)	11.5 (6.2)	12.1 (5.7)
ESSDAI >13, n (%)	51 (37.2)	48 (34.8)	45 (26.8)	46 (27.5)	47 (27.8)
ESSPRI[1]	6.40 (2.0)	6.01 (2.2)	6.29 (2.1)	6.21 (2.2)	6.26 (2.2)
ESSPRI ≥5, n (%)	108 (78.8)	95 (68.8)	126 (75.0)	123 (73.7)	130 (76.9)
sSF (mL/min) [1]	0.62 (0.6)	0.65 (0.5)	0.62 (0.6)	0.56 (0.7)	0.70 (0.7)
Positive Anti-Ro/SSA status, n (%)	125 (91.2)	132 (95.7)	160 (95.2)	161 (96.4)	158 (93.5)
Any DMARDs use, n (%)	85 (62.0)	94 (68.1)	109 (64.9)	100 (59.9)	117 (69.2)
HCQ	65 (47.4)	78 (56.5)	95 (56.5)	82 (49.1)	103 (60.9)
MTX	29 (21.2)	22 (15.9)	22 (13.1)	21 (12.6)	26 (15.4)
AZA	4 (2.9)	14 (10.1)	9 (5.4)	14 (8.4)	10 (5.9)
Systemic CS usage, n (%)	43 (31.4)	47 (34.1)	43 (25.6)	42 (25.1)	44 (26.0)
≥5 mg/day	35 (25.5)	40 (29.0)	40 (23.8)	39 (23.4)	33 (19.5)

1. Values are mean (SD), unless otherwise specified. AZA, azathioprine; CS, corticosteroids; DMARDs, disease-modifying antirheumatic drugs; ESSDAI, EULAR Sjögren's Syndrome Disease Activity Index; ESSPRI, EULAR Sjögren's Syndrome Patient Reported Index; EULAR, European Alliance of Associations for Rheumatology; HCQ, hydroxychloroquine; MTX, methotrexate; QM, monthly; Q3M, every 3 months; sSF, stimulated whole-salivary flow.

Abbreviations

Abbreviation	Full Form
AAV	Adeno-Associated Virus
ACR	American College of Rheumatology
AD	Atopic Dermatitis
ADCC	Antibody-Dependent Cellular Cytotoxicity
AE	Adverse Events
AH	Antihistamine
AID	Autoimmune Inflammatory Disease
AtD	Atopic Dermatitis
BL	Baseline
CI	Confidence Interval
CIndU	Chronic Inducible Urticaria
CSU	Chronic Spontaneous Urticaria
CV	Cardiovascular
ESSDAI	EULAR Sjögren's Syndrome Disease Activity Index
ESSPRI	EULAR Sjögren's Syndrome Patient Reported Index
EULAR	European Alliance of Associations for Rheumatology
FA	Food Allergy
FF	Field Force
FiC	First-in-Class
gMG	Generalized Myasthenia Gravis
HCP	Health Care Provider
HS	Hidradenitis Suppurativa
IgE	Immunoglobulin E
IIM	Idiopathic Inflammatory Myopathy
ITP	Immune Thrombocytopenia
L/D	Lymphodepletion
LN	Lupus Nephritis
LS	Least Squares
LFT	Liver Function Test
mAb	Monoclonal Antibody
MoA	Mechanism of Action
MS	Multiple sclerosis
PaGA	Patient Global Assessment
PCP	Primary Care Physician
PhGA	Physician Global Assessment

Abbreviation	Full Form
pMS	Progressive Multiple Sclerosis
PRO	Patient-Reported Outcome
PsO	Psoriasis
PT	Preferred Term
Q3M	Every Three Months
QoL	Quality of Life
QM	Monthly
RA	Rheumatoid arthritis
rMS	Relapsing Multiple Sclerosis
SAE	Serious Adverse Event
SE	Standard Error
SjD	Sjögren’s Disease
SLE	Systemic Lupus Erythematosus
srSLE	Refractory Systemic Lupus Erythematosus
SSc	Systemic Sclerosis
sSF	Stimulated Whole-Salivary Flow
SSSD	Sjögren's Syndrome Symptom Diary
wAIHA	Warm Autoimmune Hemolytic Anemia

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3 Mean line with standard error bars.
4 Basket study design. Patients have a single disease rather than comorbid conditions.
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1 Internal Novartis analysis leveraging multiple data sources, including IQVIA claims (LAAD, Xponent, DDD), Komodo claims.

Slide 17

1 Internal Novartis analysis leveraging multiple data sources, including IQVIA claims (LAAD, Xponent, DDD), Komodo claims.

2 CSU patient sign-ups through disease state consumer-facing website.

Slide 18

1 Novartis data on file

2 3-5 days are examples from Kisqali and Kesimpta (paid and bridge)

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