Global Science.
One Purpose.

48-week Phase 3 Clinical Trial Data from China for Telitacicept in Primary Sjögren's Disease

October 28, 2025

VOR

Forward Looking Statement

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Agenda

1.	Opening Remarks & Data Highlights	**Jean-Paul Kress, M.D.**, Chairman & Chief Executive Officer
2.	Sjögren's Disease – A Major Unmet Need	**Ronald van Vollenhoven, M.D., Ph.D.,** Professor of Rheumatology at Amsterdam University Medical Center
3.	Primary Sjögren's Disease China Phase 3 Results	**Qing Zuraw, M.D., M.P.H., M.B.A.**, Chief Development Officer
4.	Competitive Landscape & Commercial Opportunity	**Dallan Murray**, Chief Commercial Officer
5.	Closing Remarks	**Jean-Paul Kress, M.D.**, Chairman & Chief Executive Officer

AANEM | Redefining Durability in gMG with Telitacicept

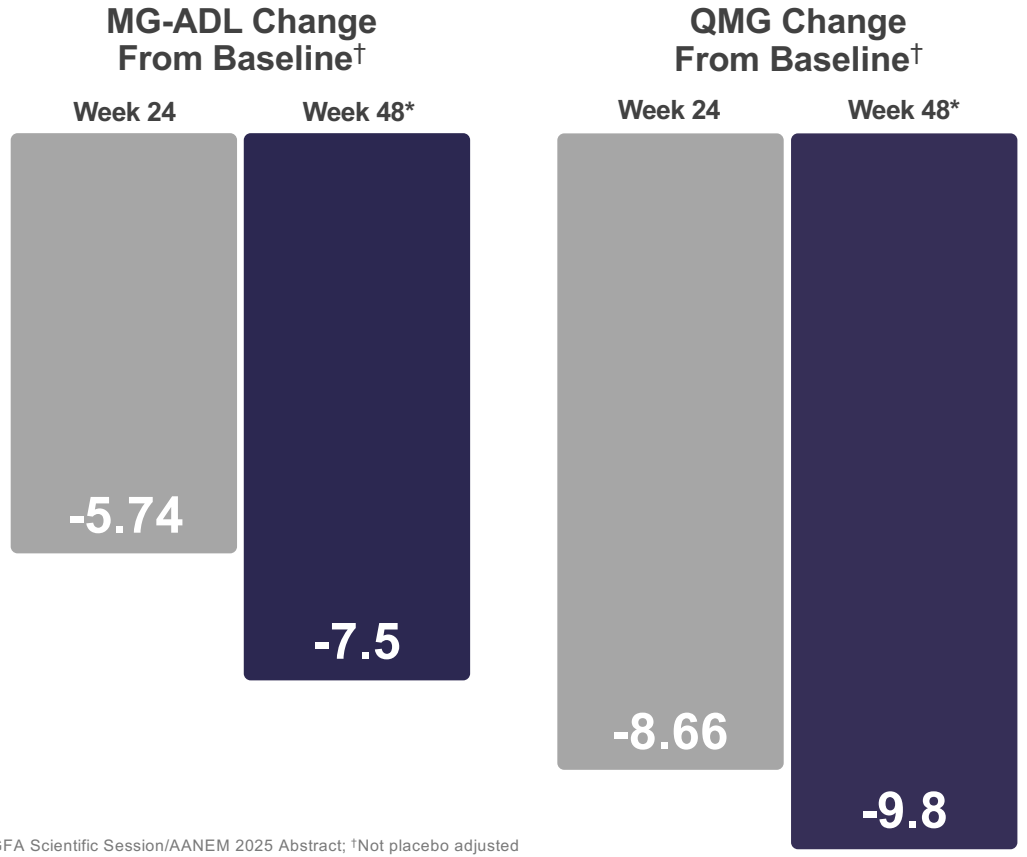
48-week OLE results oral presentation on October 29, 2025 at 10:50AM PT

Deep, sustained duration of response over time underscores BAFF/APRIL inhibition

Significant MG-ADL and QMG score reduction experienced by placebo crossover patients

Consistent safety and tolerability over one year of therapy

Opportunity to become the first disease modifying therapy in gMG



gMG, generalized myasthenia gravis; *MGFA Scientific Session/AANEM 2025 Abstract; †Not placebo adjusted

ACR | Potential to Redefine Treatment in pSD

Telitacicept showed statistically significant and clinically meaningful improvement in pSD

A True Signal, No Noise

- No DMARDs, no steroids

Clinically Meaningful, Statistically Clear

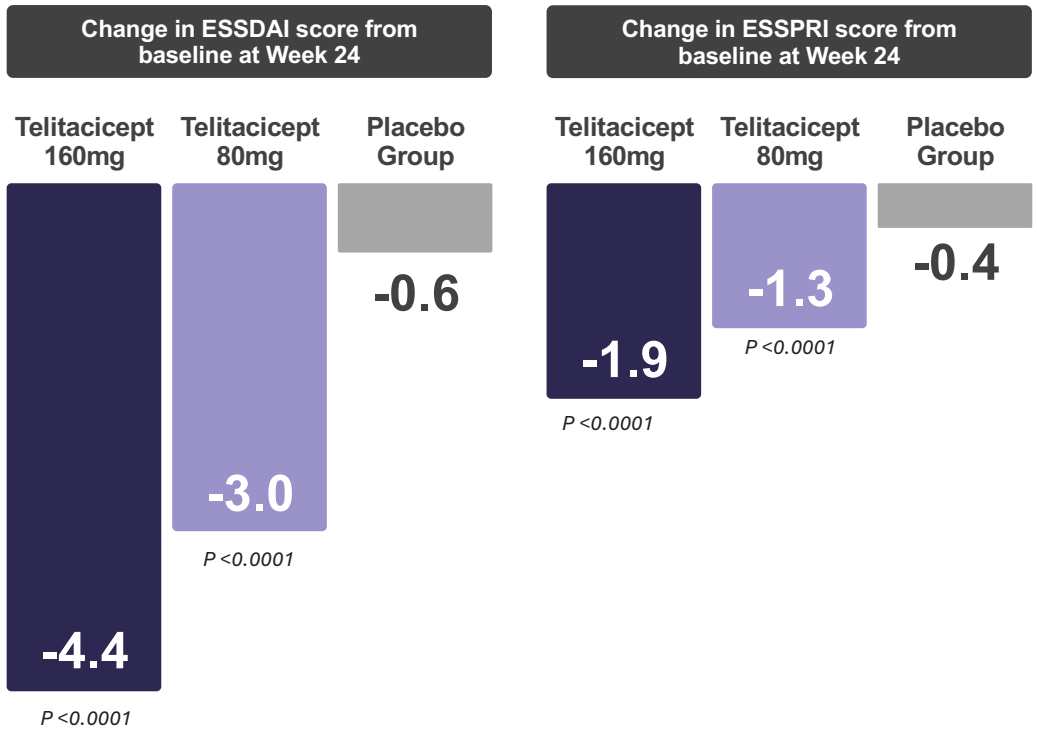
- Robust, dose-dependent improvements across physician- and patient-assessed outcomes

Depth and Durability Across Domains

- Improvement of systemic activity, symptoms, and function

Consistent Safety Profile

- No new safety signals. No opportunistic infection reported.

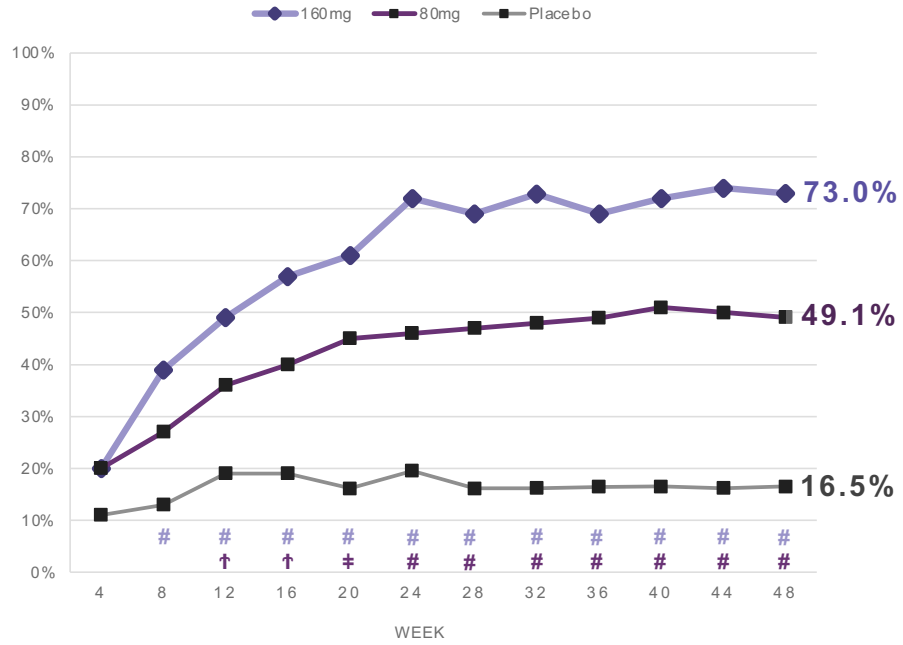


PSD, primary Sjogren's disease; The analysis of change from baseline in ESSDAI and ESSPRI score over Weeks 0-24 was based on the estimate population (EP). The MMRM method was used and missing data were not imputed. The analysis of change from baseline in ESSDAI and ESSPRI score over Weeks 0-48 was based on the estimate population (EP). The post-switching data for the two telitacicept groups and the placebo group were handled with the LOCF method, i.e. imputing all the post-switching values with the most recent pre-switching results.

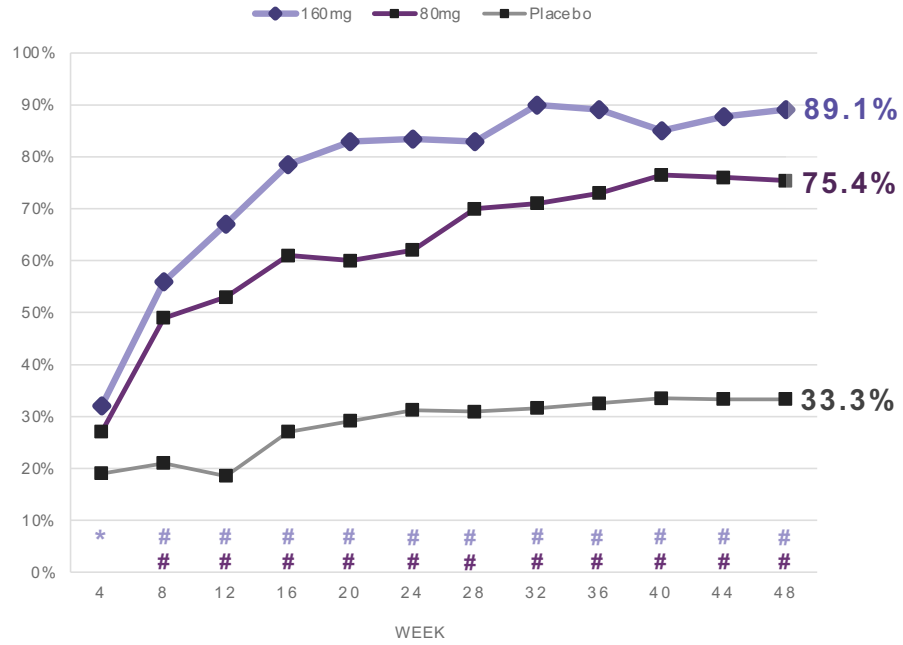
Early, Broad Symptom Improvements Observed with Telitacicept

Nearly 90% of patients report improvement as physicians confirm disease control in 3 out of 4 patients

PROPORTION OF PARTICIPANTS WITH ≥3-POINT REDUCTION FROM BASELINE IN ESSDAI SCORE OVER TIME⁺‡



PROPORTION OF PARTICIPANTS WITH ≥1-POINT OR ≥15% REDUCTION FROM BASELINE IN ESSPRI SCORE OVER TIME⁺‡



(* *P*<0.05, † *P*<0.01, ‡ *P*<0.001, # *P*<0.0001)

Placebo*: Participants randomized to the placebo group. ⁺The analysis of change from baseline in ESSDAI and ESSPRI score over Weeks 0-24 was based on the estimate population (EP). The MMRM method was used and missing data were not imputed. ‡The analysis of change from baseline in ESSDAI and ESSPRI score over Weeks 0-48 was based on the estimate population (EP). The post-switching data for the two telitacicept groups and the placebo group were handled with the LOCF method, i.e. imputing all the post-switching values with the most recent pre-switching results.

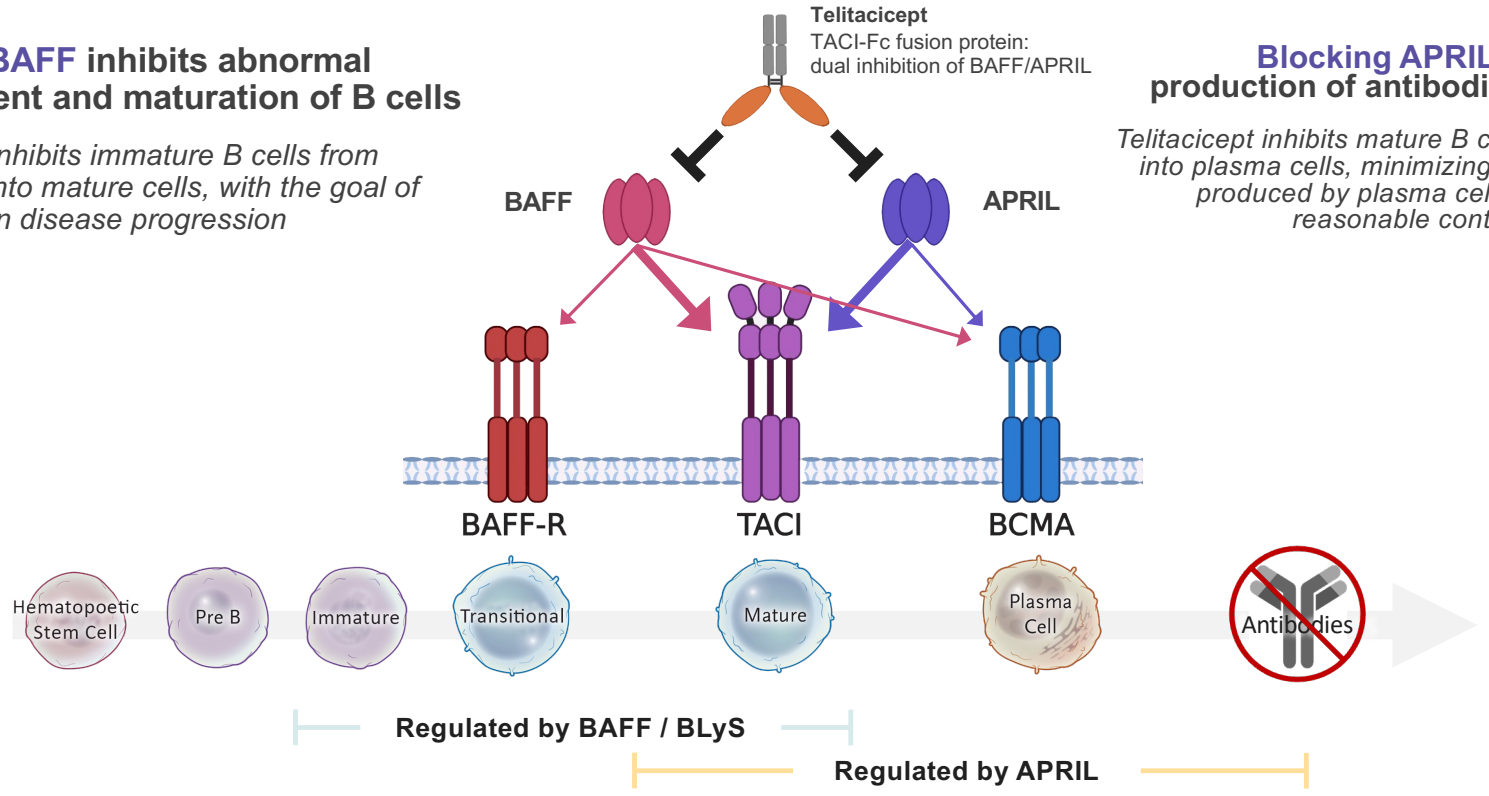
RemeGen-sponsored trial

Potential Disease Modification Through Upstream and Downstream Control

Dual BAFF/APRIL blockade is designed to stop B cell survival and plasma cell antibody production

Blocking BAFF inhibits abnormal development and maturation of B cells

Telitacicept inhibits immature B cells from developing into mature cells, with the goal of slowing down disease progression



Blocking APRIL inhibits abnormal production of antibodies by plasma cells

Telitacicept inhibits mature B cells from differentiating into plasma cells, minimizing antibodies abnormally produced by plasma cells and contributing to a reasonable control of disease activities

doi.org/10.3390/cancers12041045

BAFF, B-cell activating factor; BLyS, B Lymphocyte Stimulator; APRIL, a proliferation inducing ligand

Harnessing Native TACI Biology for Optimal Binding

Preserving receptor integrity enhances binding strength and supports a favorable safety profile

Natural Design Advantage

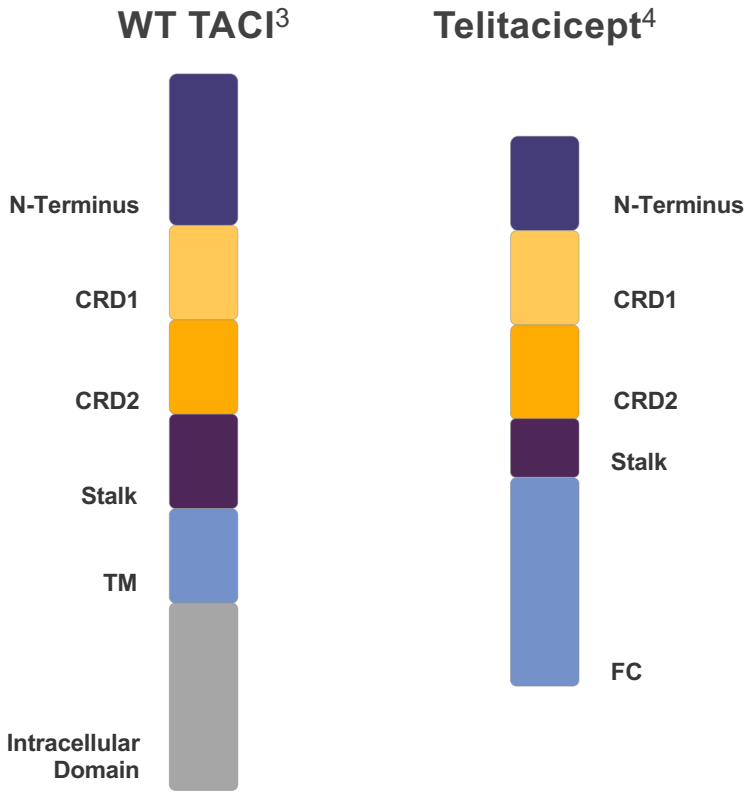
Mimics full native TACI structure (N-terminus + CRD1/2)

Native Structure Enables Optimal Binding

N-Terminus + CRD1/2 preservation targets more natural ligand engagement with BAFF and APRIL[1,2]

Safety Differentiation

Wild-type mimicry designed for cleaner tolerability vs truncated designs



1. DOI 10.1074/jbc.M411714200; 2. DOI 10.3389/fimmu.2018.02125; 3. WO2002094852A2; 4. Internal Telitacicept IP

Telitacicept: Redefining What Success Looks Like In Autoimmune Disease

Clinically meaningful results in every late-stage program with consistent efficacy and safety

1

3 Commercial Approvals in China

2021 - Systemic Lupus Erythematosus (SLE)†

2024 - Rheumatoid Arthritis (RA)

2025 – Generalized Myasthenia Gravis (gMG)

2

2 BLA Submissions in China

Primary Sjogren's Disease (pSD, Filed 2025)

*IgA Nephropathy (IgAN, Filed 2025)**

3

Best-In-Disease Leadership

Unique Dual BAFF/APRIL Inhibition Drives Superior Clinical Benefit in SLE, pSD, gMG in China

4

10s of Thousands of Patients Treated in China

Proven real-world impact at commercial scale

5

Favorable Safety Profile

No burdensome vaccination

No B-cell depletion-related SAEs

Mild to moderate AEs

6

Consistent Tolerability

Safety profile confirmed in ~1,800 patients across 6+ indications in China

AE rates comparable to placebo

†Conditional Approval, Full Approval in 2023; *Accelerated Approval in China

Amsterdam UMC
Universitair Medische Centra

Sjögren's disease – a major unmet need

Ronald van Vollenhoven

Department of Rheumatology and Clinical Immunology

Disclosures

- Research Support (institutional grants): AstraZeneca, BMS, Cabaletta, Novartis, Remegen
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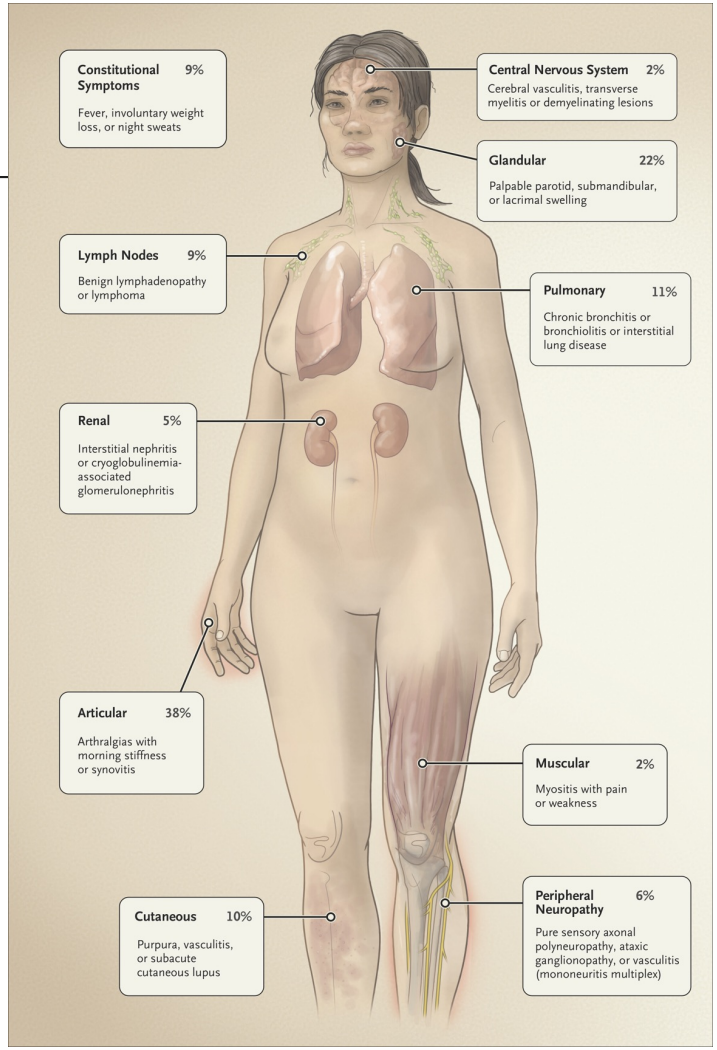
- Consultancy and/or speaker: AbbVie, AstraZeneca, Biogen, BMS, Cabaletta, Galapagos, GSK, Janssen, Kyowakirin, Pfizer, RemeGen, Sanofi, UCB, Vor Bio

Sjögren's Disease

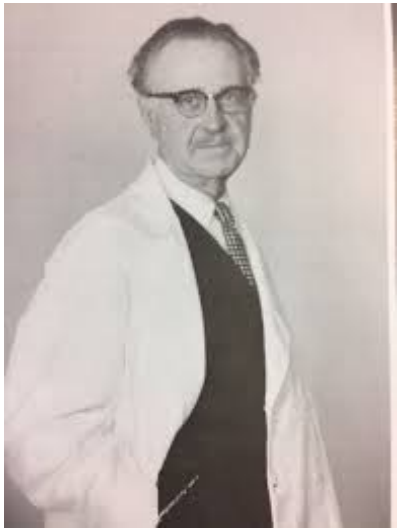
A multi-organ systemic autoimmune disease

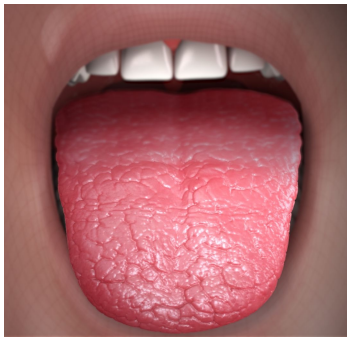


Xavier Mariette, M.D., Ph.D., and Lindsey A. Criswell, M.D., M.P.H., D.Sc.; Published March 7, 2018; N Engl J Med 2018;378:931-939; DOI: 10.1056/NEJMcp1702514; VOL. 378 NO. 10; 2018

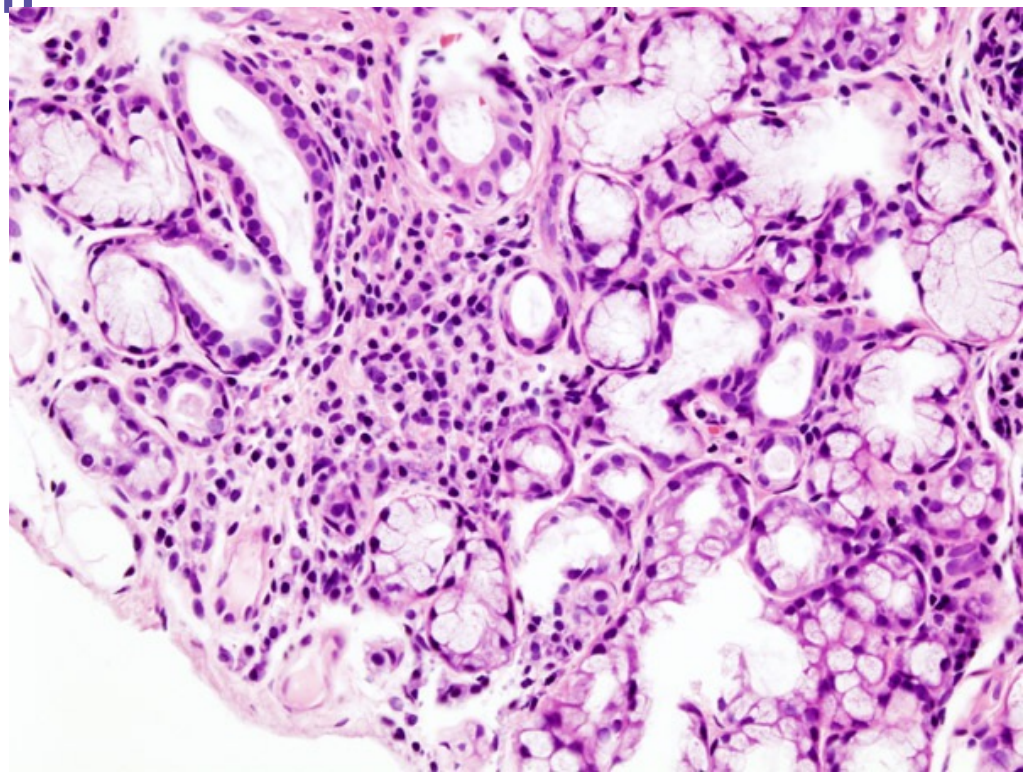
Sjögren's Disease (Sjögren's syndrome)

- Incidence 3-11/100,000
- Prevalence 0.5%
- Female-male 9:1
- 2 peaks: 20-30 and menopause
- Autoantibodies:
  - SSA = anti-Ro
  - SSB = anti-La
- ACR-EULAR classification criteria





Pathophysiology: chronic lymphocytic inflammatorion



https://en.wikipedia.org/wiki/Sj%C3%B6gren%27s_disease

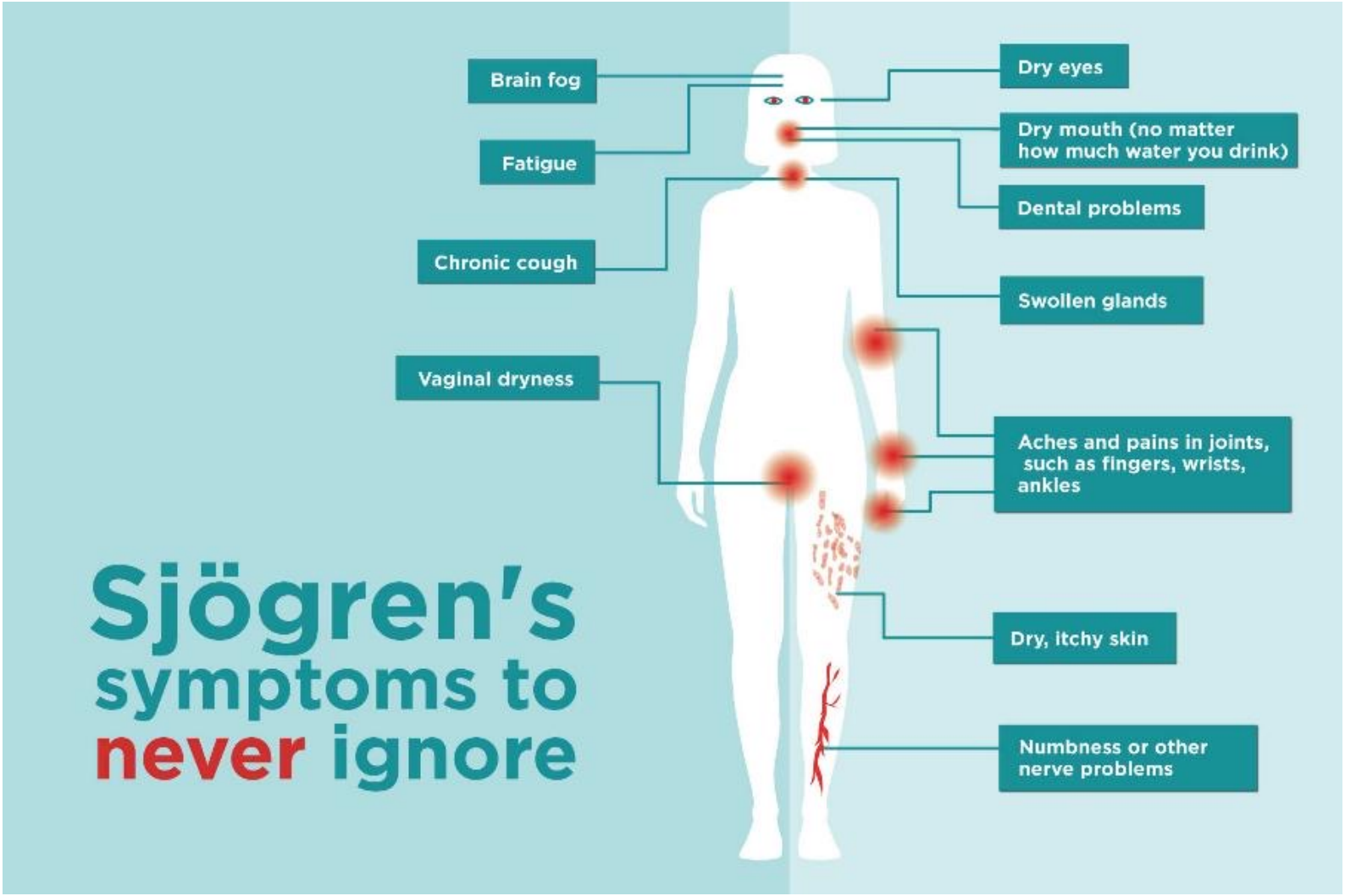
Diagnosis ≠ classification

ACR-EULAR 2016 Classification Criteria for Sjögren's		
Item #	*Item to be Scored*	*Weight*
1	Labial salivary gland with focal lymphocytic sialadenitis and focus score of $1 foci/4 mm	3
2	Anti-SSA/Ro positive	3
3	Ocular Staining Score ≥5 (or van Bijsterveld score ≥4) in at least 1 eye	1
4	Schirmer's test ≤5 mm/5 minutes in at least 1 eye	1
5	Unstimulated whole saliva flow rate #0.1 ml/minute	1

Serological features	pSS (%)	SLE (%)
Antinuclear antibodies[2,3,26]	70	99
Anti-Ro (SSA)[2,3,26]	50–90	30–40
Anti-La (SSA)[2,3,26]	25–60	10–15
Anti-dsDNA[2–4]	–	40–70
Anti-Sm[2–4]	–	20–40
Anti-ribosomal P protein[2–4]	–	13–20
Rheumatoid factor[2,3,26]	36–74	15–30
Cryoglobulins[26,27,35]	7–20	48.8
Antiphospholipids[2–4,39]	16	40
Anti-c2M3PR[38]	62.2	7.1
Low C3 complement fraction[27,35]	13.4	41.3
Low C4 complement fraction[27,35]	14.4	48.8

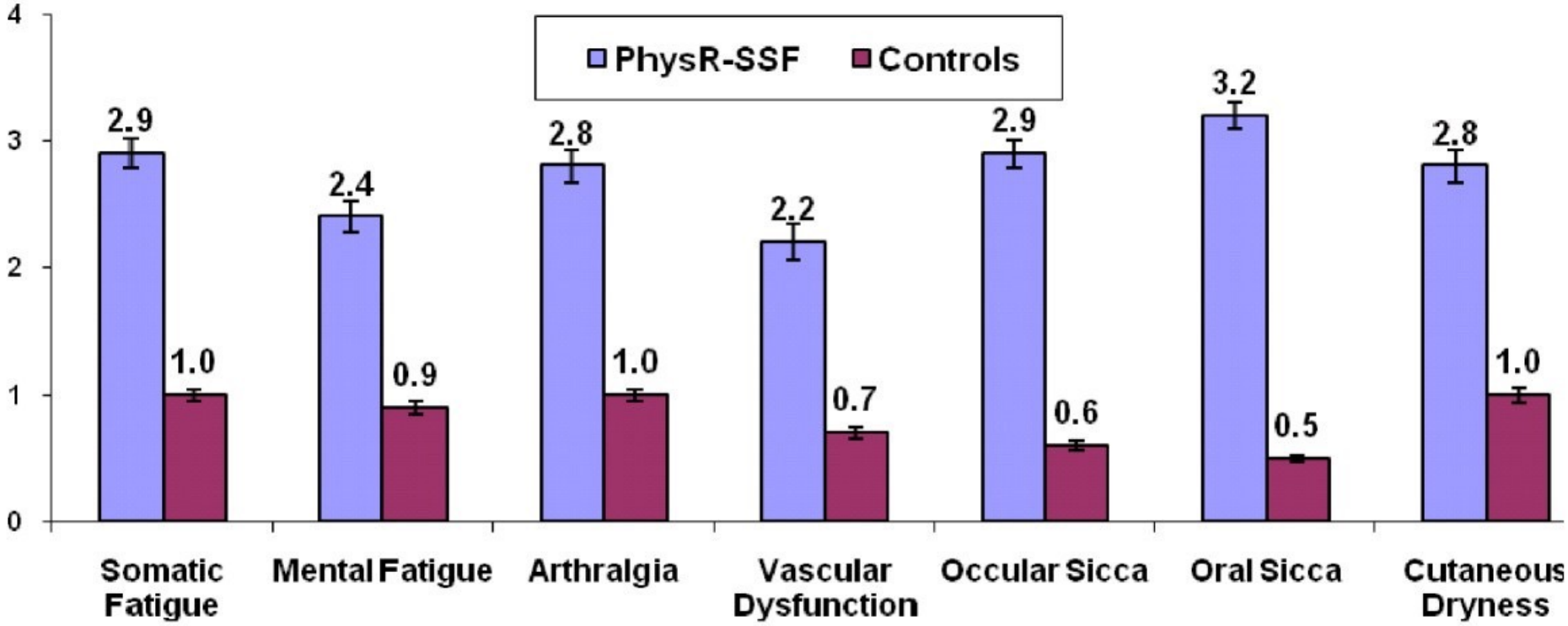
Sjögren's Disease (Sjögren's syndrome)

- Primary
- Secondary → Overlap
  - RA, lupus, SSc etc.
- Glandular and extraglandular
- "Autoimmune exocrinopathy"
- "Autoimmune epithelitis"



https://creakyjoints.org/living-with-arthritis/symptoms/sjogrens-syndrome-symptoms/

Multiple domains are affected

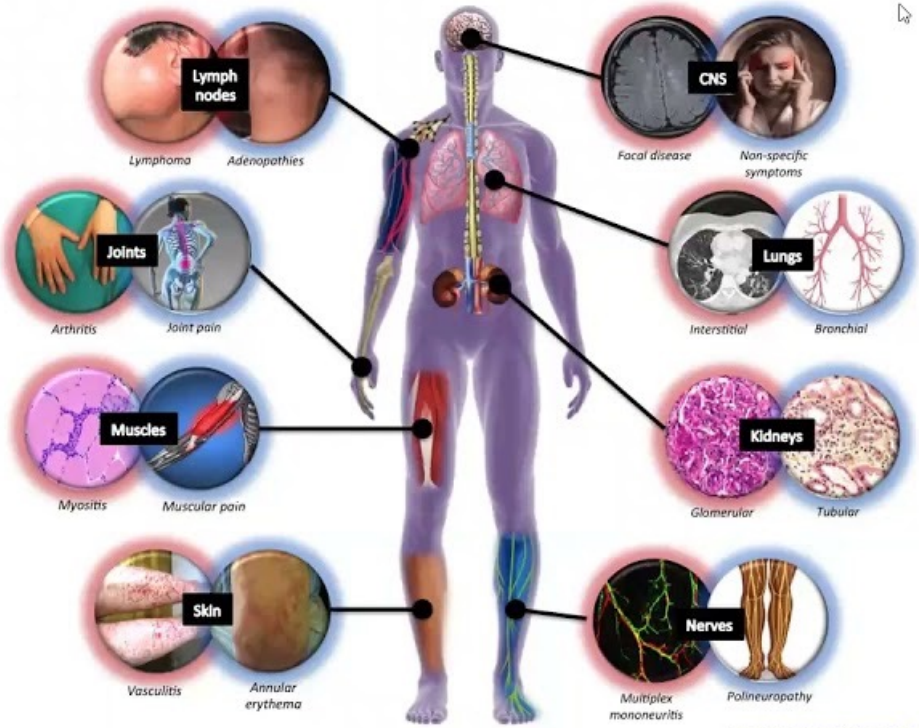


Note: Mean scores and Standard Errors for PROFAD-SSI domains

Outcome measures in pSS

Clinical outcomes

- EULAR Sjögren's Syndrome Disease Activity Index (ESSDAI)

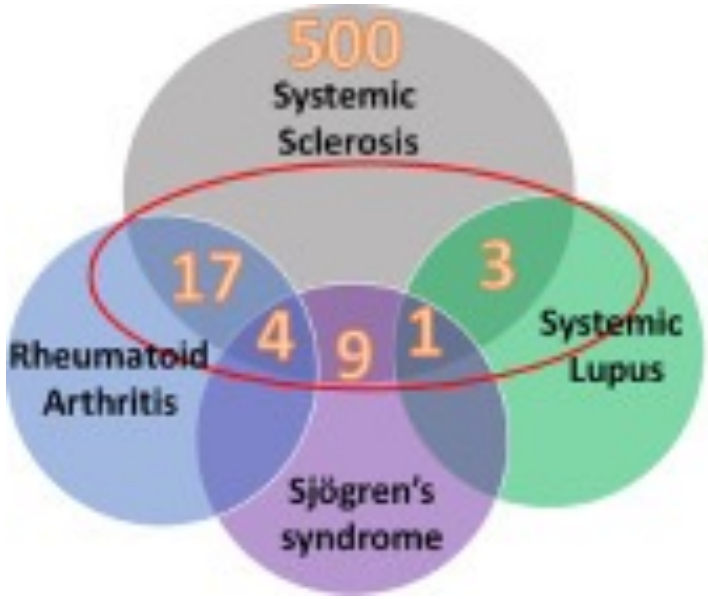


ESSDAI: EULAR Sjogren's syndrome disease activity index

Domain	Weight	Characteristics of the domain	Activity levels**
1. Constitutional	3	Fever, night sweats, weight loss	0-2
2. Lymphadenopathy and lymphoma	4	Swollen lymph nodes, splenomegaly, current B-cell proliferative malignancy	0-3
3. Glandular	2	Swollen salivary and/or lacrimal glands	0-2
4. Articular	2	Arthralgias with morning stiffness, or synovitis among 28 joints	0-3
5. Cutaneous	3	Erythema multiforme, cutaneous, including urticarial, vasculitis, or purpura, or subacute cutaneous lupus, ulcers related to vasculitis	0-3
6. Pulmonary	5	A persistent cough, bronchial involvement, or radiological evidence of ILD	0-3
7. Renal	5	Tubular acidosis, glomerular involvement with proteinuria >0,5 g/L, or haematuria, or renal failure, or histological evidence of glomerulonephritis, interstitial nephritis, or cryoglobulinemia-related renal involvement	0-3
8. Muscular	6	Active myositis proven by abnormal EMG or biopsy with or without weakness or elevated creatine kinase	0-3
9. Peripheral nervous system	5	Evidence of active peripheral nerve involvement proven by nerve-conductive studies, trigeminal neuralgia, or cranial peripheral nerve involvement	0-3
10. Central nervous system	5	Cranial nerve involvement, optic neuritis, multiple sclerosis-like syndrome with pure sensory, or cognitive impairment, or motor deficit, cerebral vasculitis with cerebrovascular accident, seizures, transverse myelitis, lymphocytic meningitis	0-2
11. Haematological	2	Cytopenia of autoimmune origin with neutropenia, anaemia, thrombocytopenia or lymphopenia	0-3
12. Biological	1	Clonal component, cryoglobulinemia, or hypocomplementemia, or hypergammaglobulinemia with IgG>15 g/L, or recent onset of hypogammaglobulinemia (IgG<5 g/L)	0-2

*Adapted from Seror *et al.* 2010 (21).
**0=no activity, 1=low activity, 2=moderate activity, 3=high activity.
ILD=interstitial lung disease; EMG=electromyogram.

Overlap among the systemic autoimmune diseases



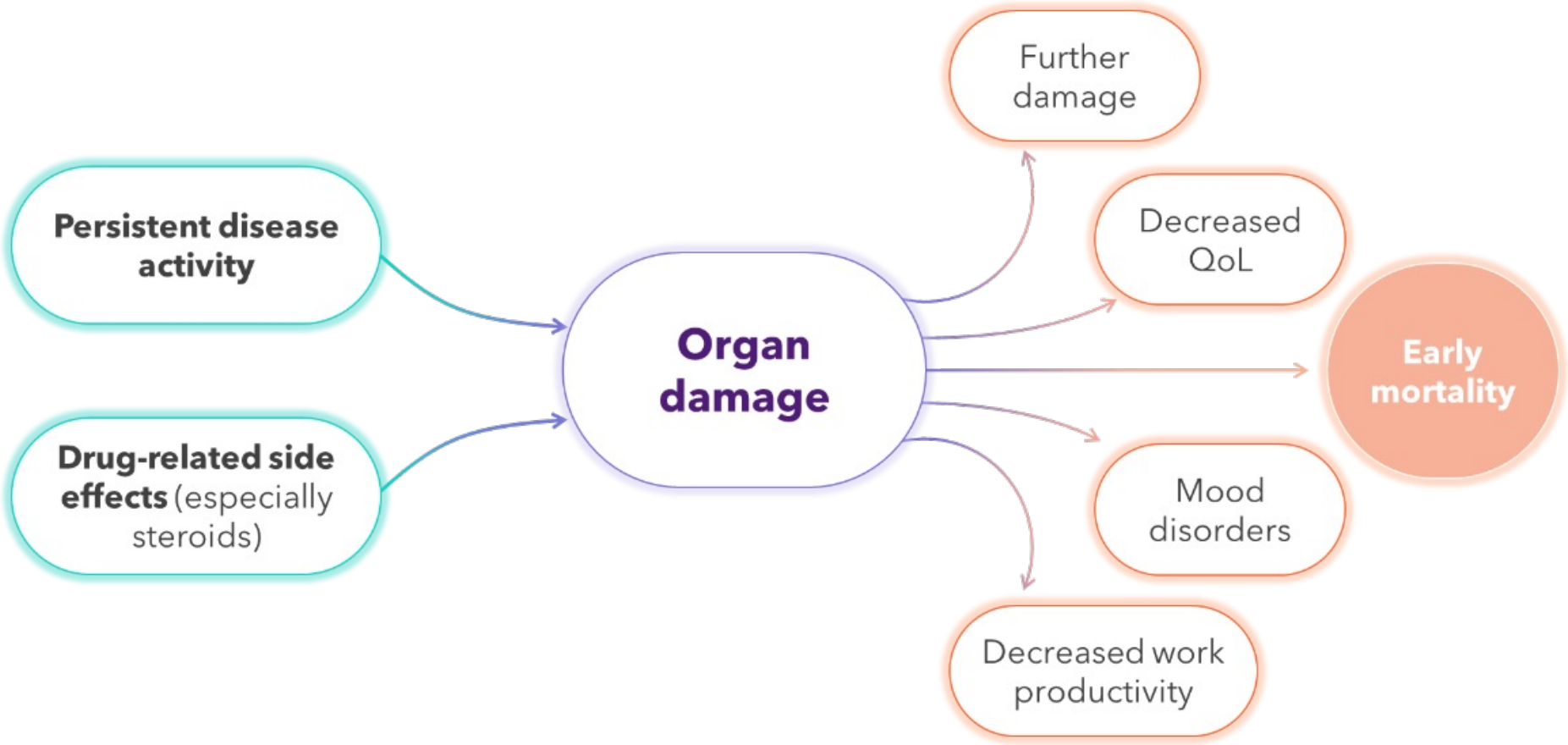
OVERLAP SYNDROMES

- → > 6% of Systemic Sclerosis
- → More corticosteroid prescription
- → Increased mortality in Sjögren's syndrome/Systemic Sclerosis overlap

Screening for overlap should be encouraged

SLE: systemic lupus erythematosus (erythematodes); “lupus”

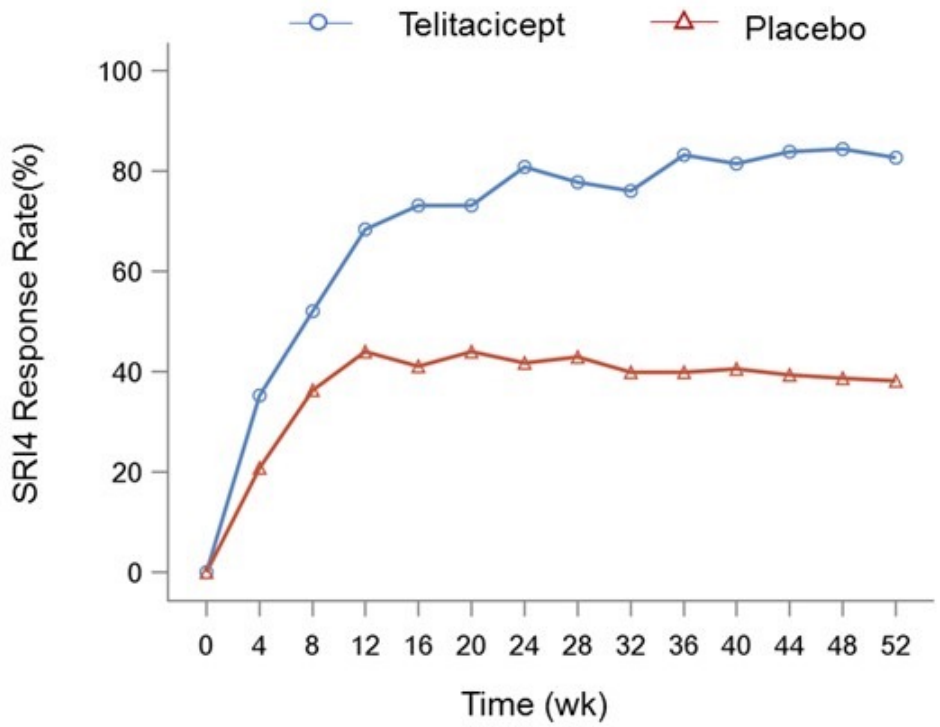
Leading causes and consequences of organ damage in SLE



QoL = quality of life.

Adapted from Doria A, et al. Autoimmun Rev 2014;13:770–777.

Telitacicept: an effective new therapy for SLE



Van Vollenhoven et al, NEJM, 2025

Conclusions

- Sjogren's disease:
  - an important chronic autoimmune disease
  - major burden on patients' quality of life
  - limited treatment options
- New treatments are emerging for Sjogren based on pathophysiology, including those that target B-cells
- For lupus, a closely related disease, recent data support the efficacy and safety of the Blys/APRIL antagonist telitacicept

Primary Sjögren's Disease China Phase 3 Results

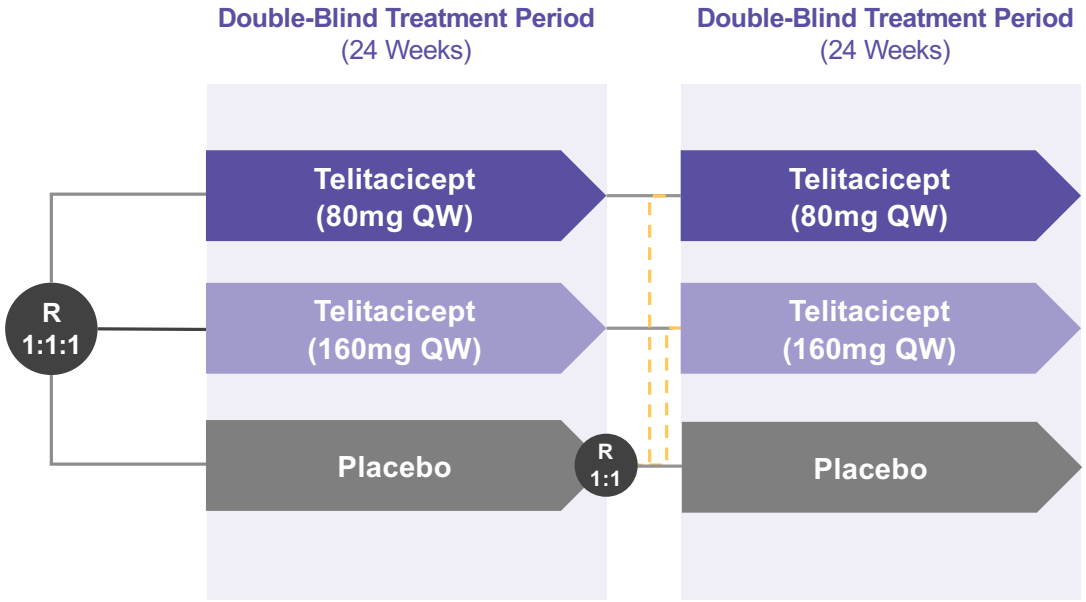
Qing Zuraw, M.D., M.P.H., M.B.A., Chief Development Officer

Phase 3 Trial in Primary Sjögren's Disease Completed in China

Potential best-in-disease profile in China; randomized, double-blind, placebo-controlled study

380*

Adults With pSD



Primary Endpoint

- Change from baseline in ESSDAI at 24 weeks

Secondary Endpoints

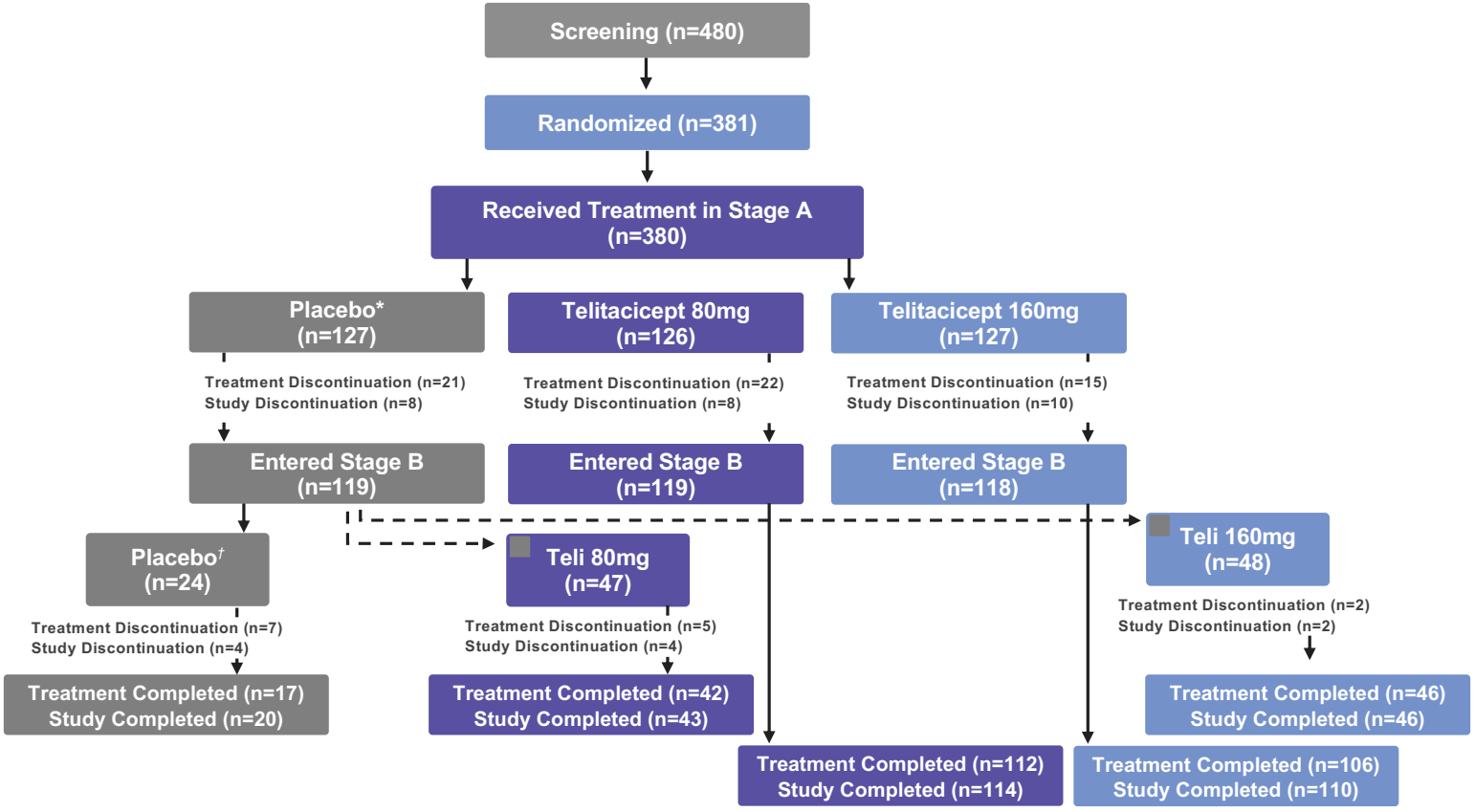
- Change from baseline in ESSDAI at 12 weeks
- Changes from baseline in ESSPRI, PGA, PaGA, SF-36 and MFI-20 at 12 and 24 weeks

*Estimated. ESSDAI, EULAR Sjögren's syndrome disease activity index; ESSPRI, EULAR Sjögren's Syndrome Patient Reported Index; MFI, multidimensional fatigue inventory; MFI-20, multidimensional fatigue inventory; PGA, physician's global assessment; PaGA, patient's global assessment; SF-36, 36-item short-form; pSD, primary Sjögren's disease; QW, per week.

RemeGen-sponsored trial

Patient Disposition

Strong study execution across 79 sites in China



*Participants randomized to the placebo group; †Participants who did not switch treatment throughout the trial; Teli, telitacicept

RemeGen-sponsored trial

Baseline Characteristics

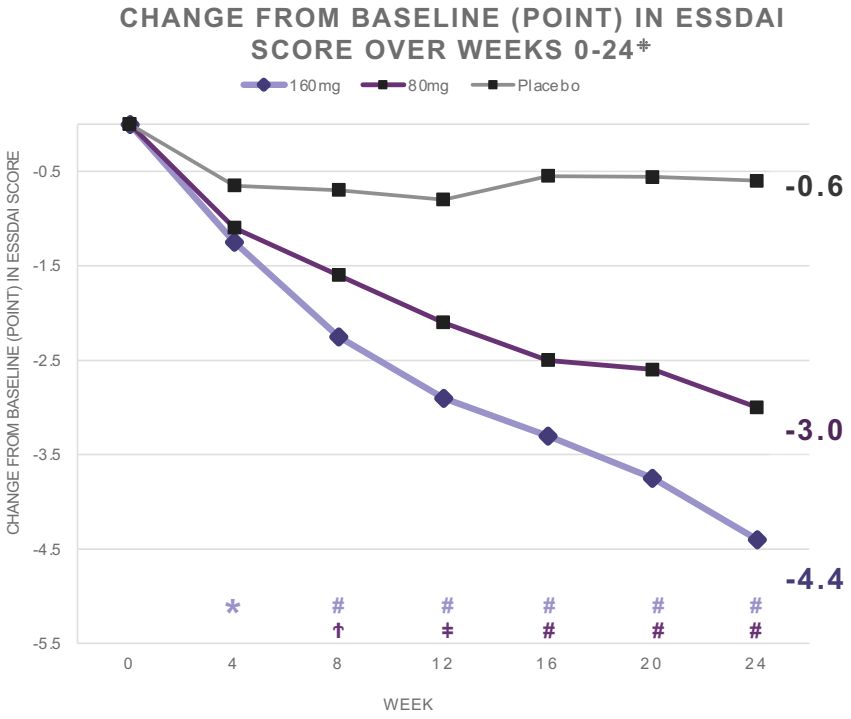
Well-balanced, representative patient population across treatment arms

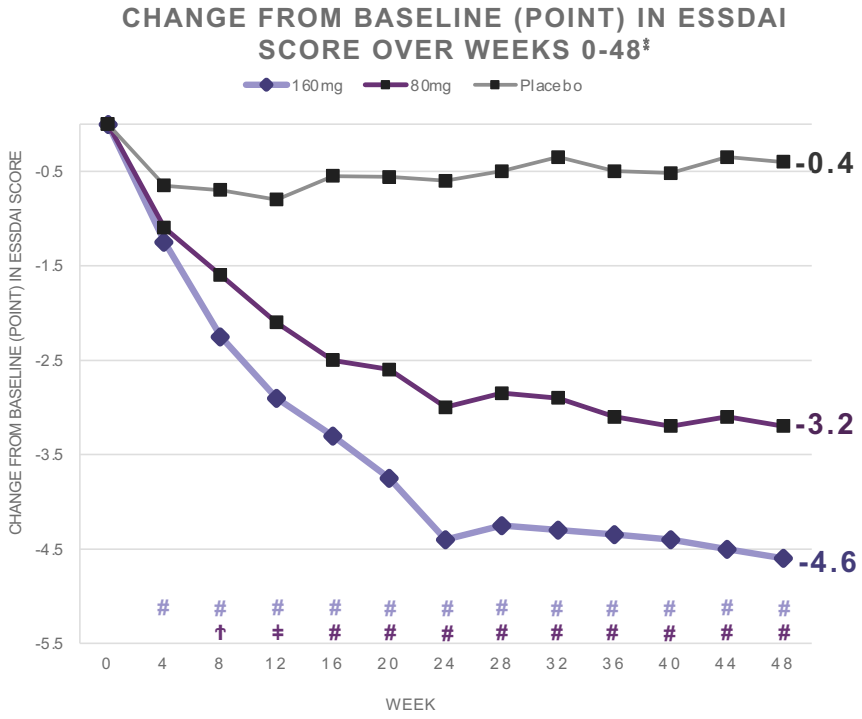
	Telitacicept 160mg (n=127)	Telitacicept 80mg (n=127)	Placebo (n=127)	Total (n=381)
Age (yr), Mean (SD)	45.9 (12.29)	44.6 (12.06)	47.3 (12.75)	46.0 (12.39)
Body Weight (kg), Mean (SD)	55.89 (9.04)	56.88 (8.71)	56.99 (11.08)	56.58 (9.65)
BMI (kg/m^2), Mean (SD)	22.03 (3.16)	22.31 (3.19)	22.24 (3.58)	22.19 (3.31)
Sex, n (%), Female	124 (97.6)	124 (97.6)	123 (96.9)	371 (97.4)
pSD Duration (mon), Mean (SD)	21.388 (36.32)	25.831 (46.90)	19.930 (39.57)	22.383 (41.14)
ESSDAI Score, Mean (SD)	10.0 (3.77)	9.8 (3.52)	10.2 (4.17)	10.0 (3.82)
ESSDAI ≥10 points, n (%)	63 (49.6)	61 (48.0)	63 (49.6)	187 (49.1)
ESSPRI Score, Mean (SD)	5.07 (1.60)	4.91 (1.72)	5.08 (1.76)	5.02 (1.69)
MFI-20 Total Score, Mean (SD)	56.8 (12.08)	56.8 (12.50)	58.1 (13.07)	57.2 (12.54)
Baseline Hydroxychloroquine Use, n (%)	87 (68.5)	97 (76.4)	99 (78.0)	283 (74.3)
IgG (g/L), Mean (SD)	21.459 (7.43)	22.429 (7.95)	22.297 (7.21)	22.062 (7.53)
IgA (g/L), Mean (SD)	3.435 (1.63)	3.637 (1.86)	3.154 (1.68)	3.409 (1.73)
IgM (g/L), Mean (SD)	1.458 (0.64)	1.372 (0.92)	1.274 (0.70)	1.368 (0.77)
CD19$^+$ B Cell (cells/µL), Mean (SD)	209.412 (129.10)	183.993 (109.52)	266.902 (726.52)	220.102 (430.96)

SD: standard deviation; BMI: body mass index; Mon, months; ESSDAI, EULAR Sjögren's syndrome disease activity index; ESSPRI, EULAR Sjögren's Syndrome Patient Reported Index; MFI-20, multidimensional fatigue inventory; RemeGen-sponsored trial

Deep, Consistent ESSDAI Reduction Through 48 Weeks

7x greater improvement means fewer active symptoms and broader systemic relief for patients





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RemeGen-sponsored trial

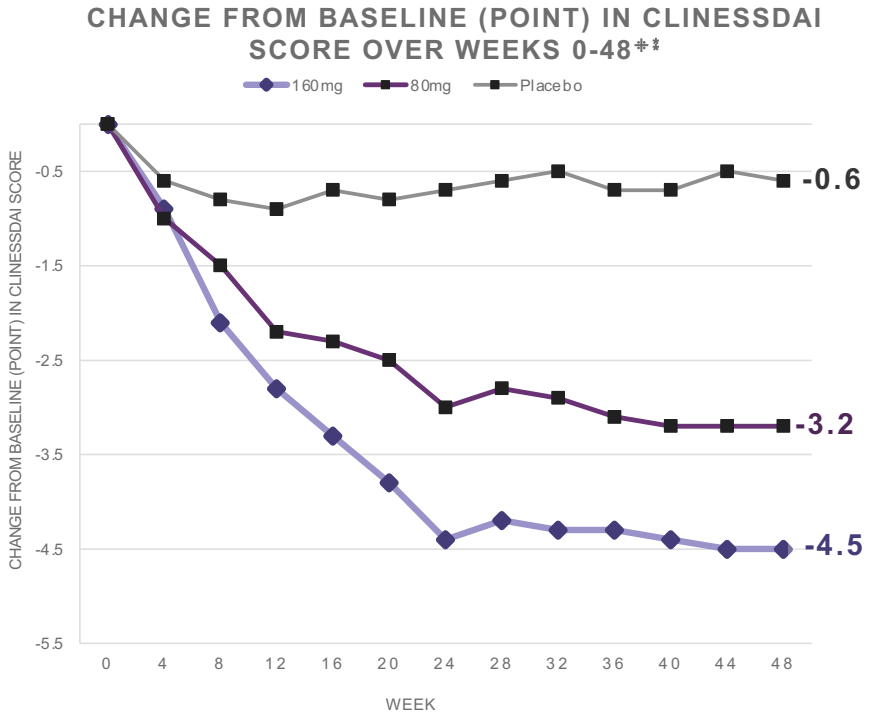
Deep, Consistent ClinESSDAI Reduction Through 48 Weeks

Sustained improvement in clinical benefit beyond serologic change

ESSDAI and ClinESSDAI remain nearly identical at week 48

-4.6 vs -4.5

ClinESSDAI excludes biological domain (IgG, complement), representing a more sensitive measure of pure clinical disease activity



ClinESSDAI, clinical ESSDAI; Placebo*: Participants randomized to the placebo group. ⁺The analysis of change from baseline in ClinESSDAI score over Weeks 0-24 was based on the estimate population (EP). The MMRM method was used and missing data were not imputed. ‡The analysis of change from baseline in ClinESSDAI score over Weeks 0-48 was based on the estimate population (EP). The post-switching data for the two telitacicept groups and the placebo group were handled with the LOCF method, i.e. imputing all the post-switching values with the most recent pre-switching results.

RemeGen-sponsored trial

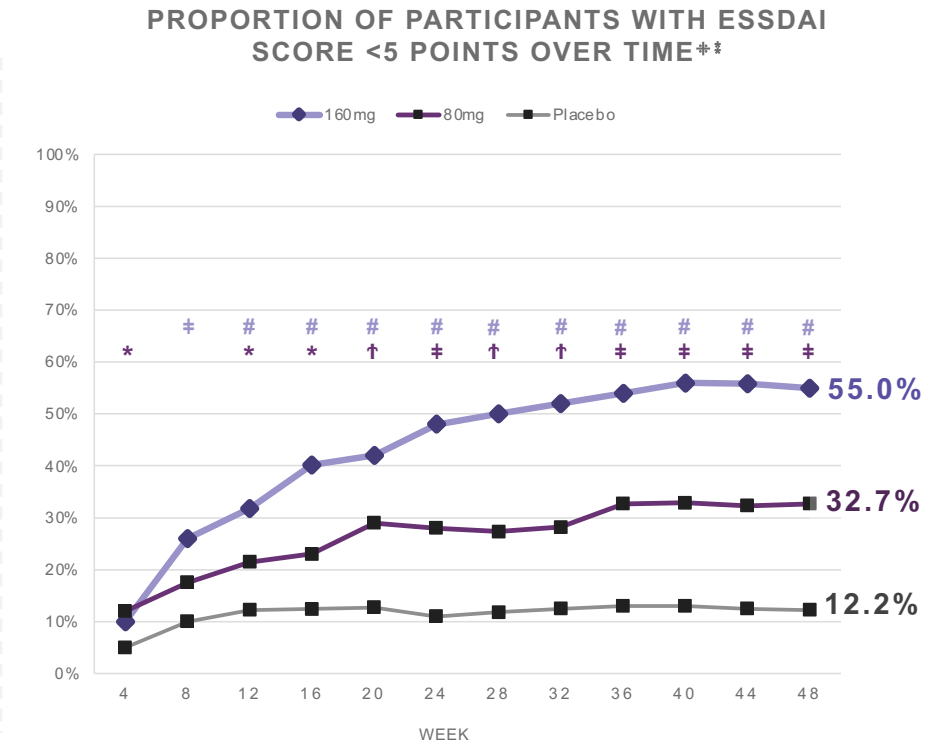
More Than Half of Patients Achieve Low Disease Activity

Nearly 5x more patients on 160mg vs placebo achieved this threshold

Low Disease Activity (ESSDAI <5) Represents Minimal Systemic Involvement

55% vs 12%

Consistent improvement sustained through 48 weeks, indicating durable immune stabilization

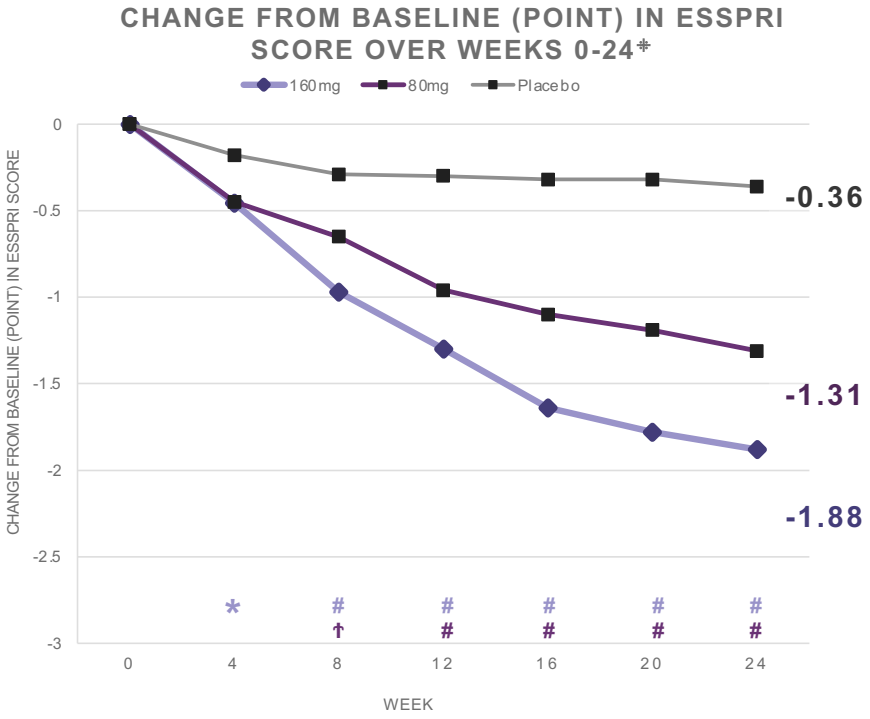


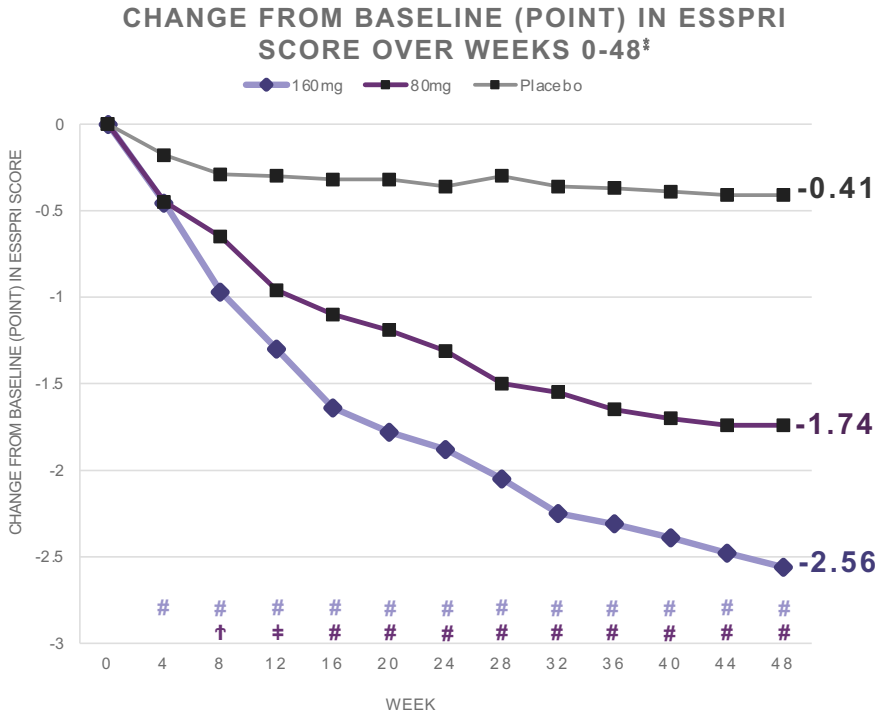
(* *P*<0.05, † *P*<0.01, ‡ *P*<0.001, # *P*<0.0001)

Placebo*: Participants randomized to the placebo group. ⁺The analysis of change from baseline in ESSDAI score over Weeks 0-24 was based on the estimate population (EP). The MMRM method was used and missing data were not imputed. ‡The analysis of change from baseline in ESSDAI score over Weeks 0-48 was based on the estimate population (EP). The post-switching data for the two telitacicept groups and the placebo group were handled with the LOCF method, i.e. imputing all the post-switching values with the most recent pre-switching results.

Sustained Improvement in ESSPRI Through 48 Weeks

Reduction in patient-reported fatigue, pain, and dryness by ~2.6 points at one year





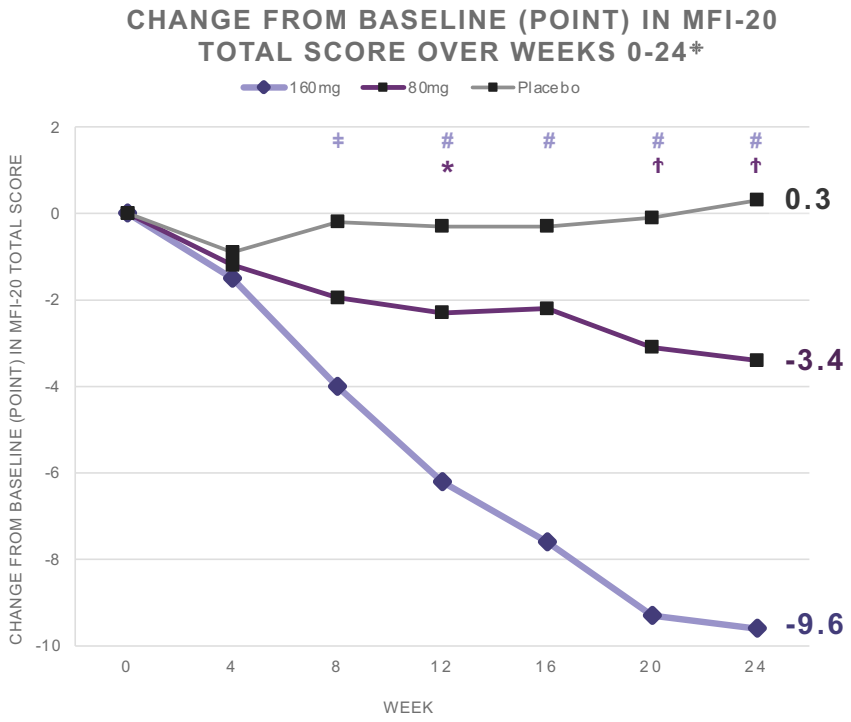
(* *P*<0.05, † *P*<0.01, ‡ *P*<0.001, # *P*<0.0001)

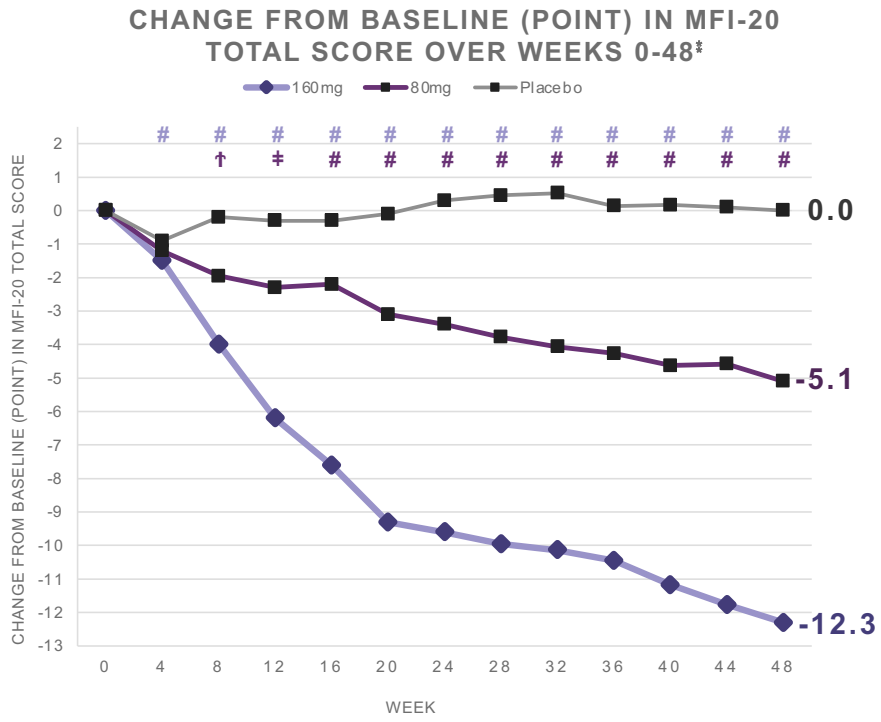
Placebo*: Participants randomized to the placebo group. ⁜The analysis of change from baseline in ESSPRI score over Weeks 0-24 was based on the estimate population (EP). The MMRM method was used and missing data were not imputed. ⁑The analysis of change from baseline in ESSPRI score over Weeks 0-48 was based on the estimate population (EP). The post-switching data for the two telitacicept groups and the placebo group were handled with the LOCF method, i.e. imputing all the post-switching values with the most recent pre-switching results.

RemeGen-sponsored trial

Telitacicept Produced Clinically Meaningful Improvement in Fatigue

12-point improvement over 48 weeks to a critical daily symptom for patients





(* *P*<0.05, † *P*<0.01, ‡ *P*<0.001, # *P*<0.0001)

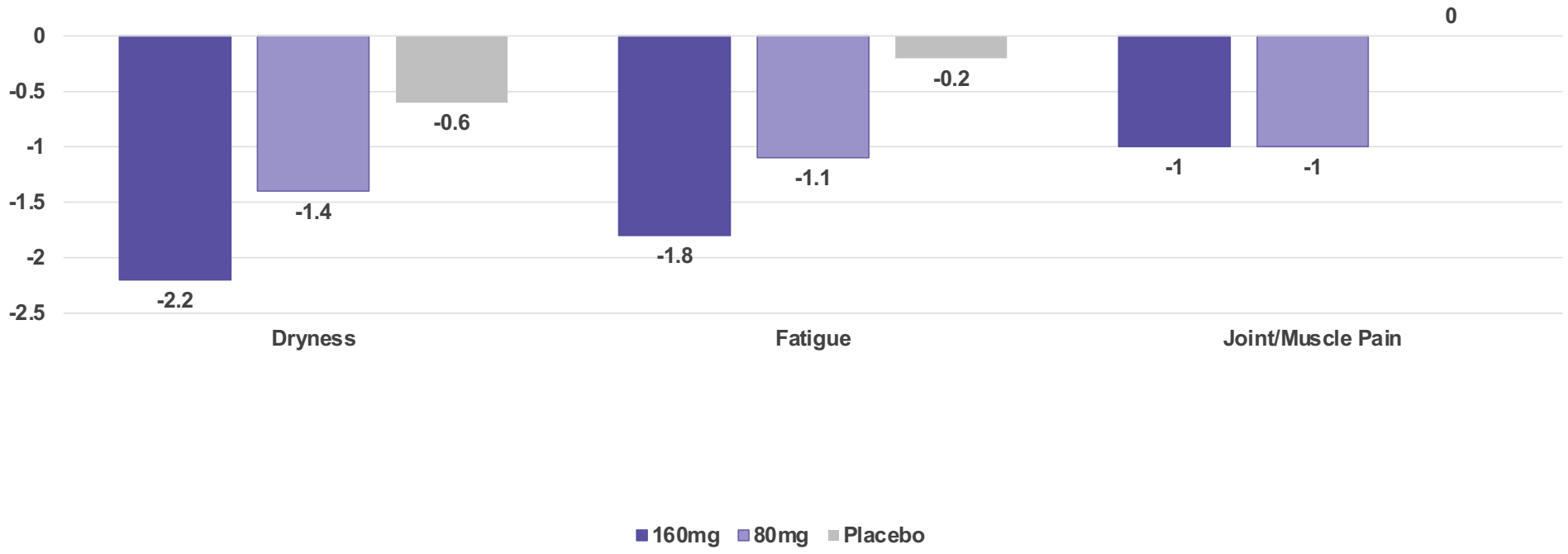
Placebo*: Participants randomized to the placebo group. ⁜The analysis of change from baseline in MFI-20 score over Weeks 0-24 was based on the estimate population (EP). The MMRM method was used and missing data were not imputed. ⁑The analysis of change from baseline in MFI-20 score over Weeks 0-48 was based on the estimate population (EP). The post-switching data for the two telitacicept groups and the placebo group were handled with the LOCF method, i.e. imputing all the post-switching values with the most recent pre-switching results.

RemeGen-sponsored trial

BAFF/APRIL MOA Associated With Improvement Across All ESSPRI Domains

Meaningful improvement across dryness, fatigue, and pain – symptoms that define daily patient life

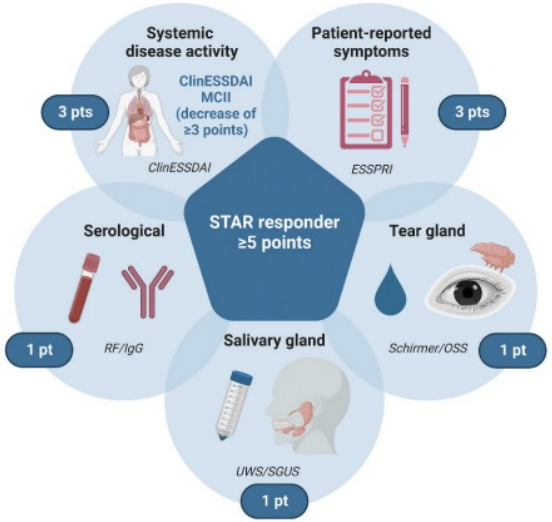
CHANGE FROM BASELINE IN ESSPRI SCORE BY DOMAIN AT 24 WEEKS



MOA, mechanism of action

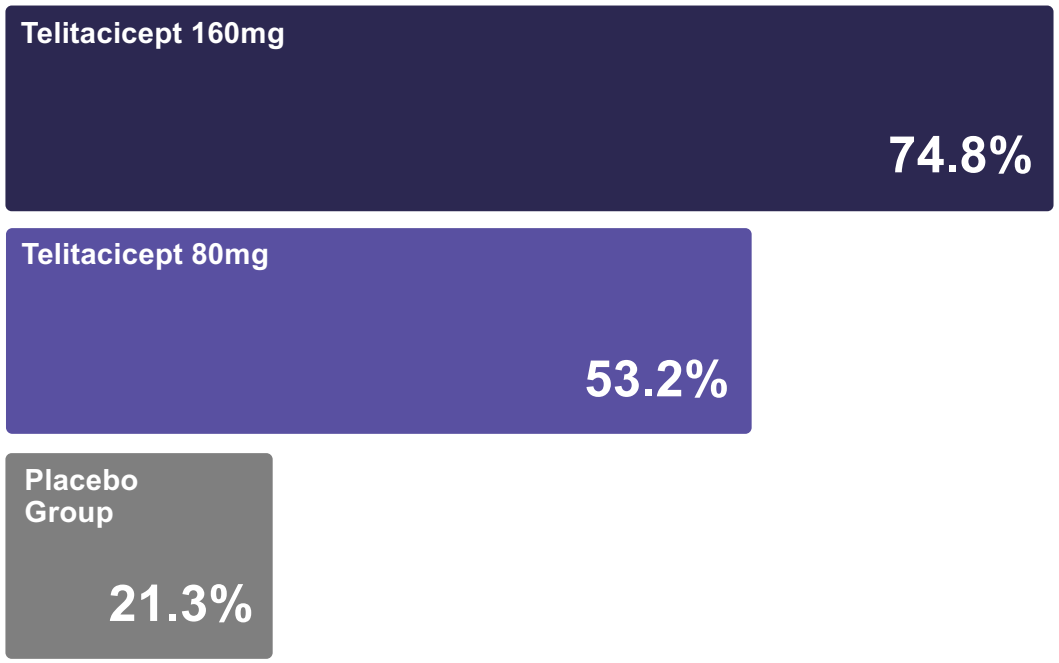
STAR: Exploratory Endpoint Demonstrates Multi-Domain Improvement

Nearly 3 in 4 patients achieved ≥5 point response



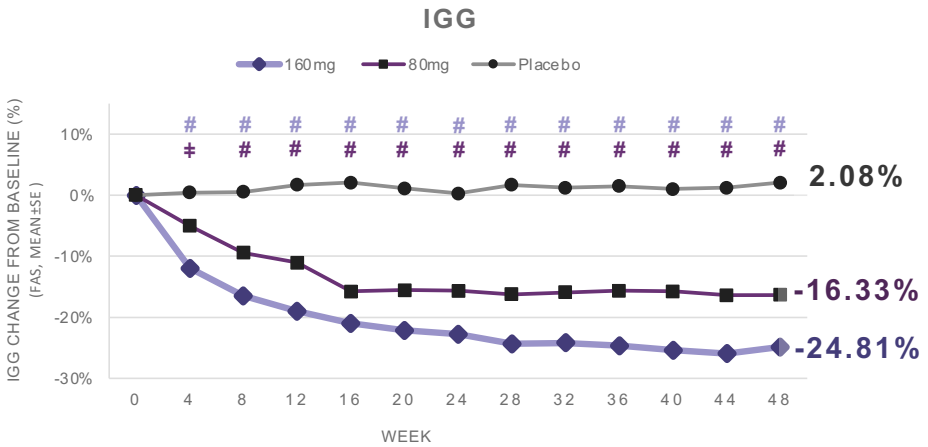
- STAR integrates systemic disease activity (ClinESSDAI), symptoms (ESSPRI), and glandular function (Schirmer's / salivary flow)
- Systemic disease activity and patient-reported symptoms are considered as major items (3 points per item) and the rest as minor items (1 point per item)
- Patients are classified as STAR responders when they reach ≥ 5 of 9 points

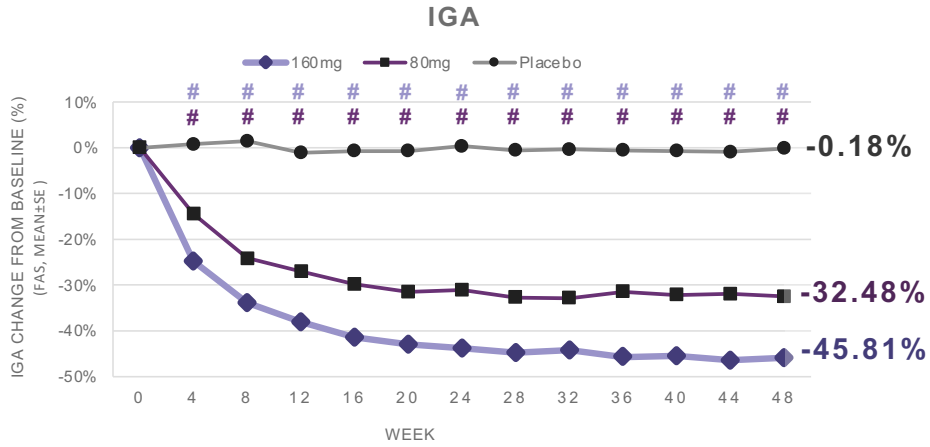
STAR Responders (%) at Week 24

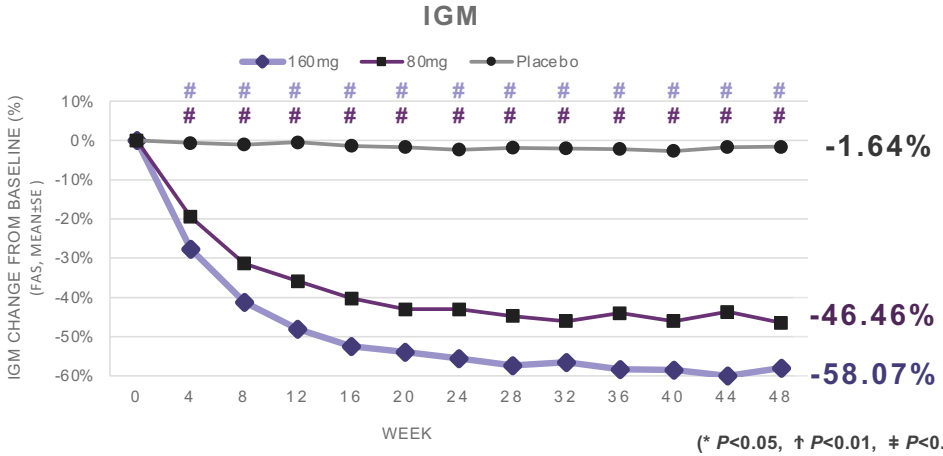


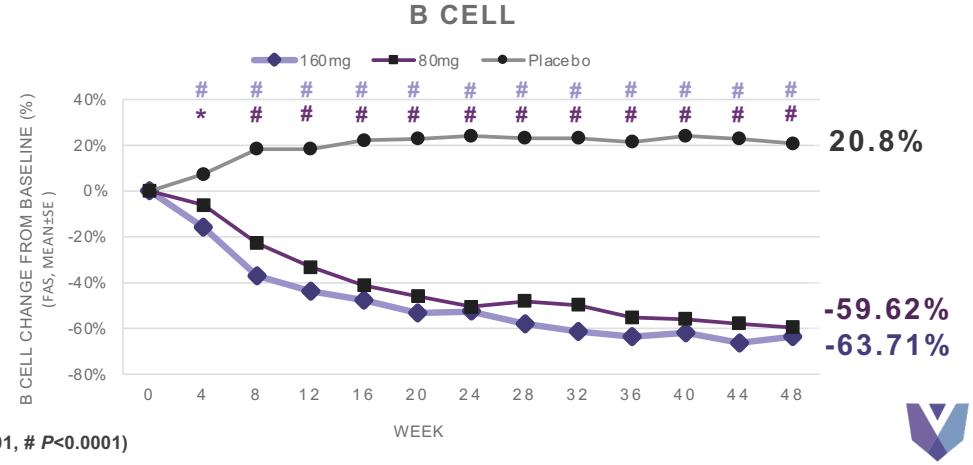
Placebo*: Participants randomized to the placebo group. A STAR responder was defined as a participant with a total STAR score of ≥5 points. Participants with missing scores in any domain (except those with a total STAR score of ≥5 points despite missing scores in some domains) were imputed as "non-responders". The responder proportion analysis for Weeks 0-24 was based on estimate population (EP). The stratum-adjusted between-group difference was tested with the CMH method. The responder proportion analysis for Weeks 28-48 was based on the estimate population (EP). The stratum-adjusted between-group difference was tested with the CMH method. The post-switching data for the two telitacicept groups and the placebo group were handled with the LOCF method, i.e. imputing all the post-switching values with the most recent pre-switching results.

Consistent Reduction in IgG, IgA, IgM, and B Cells









(* *P*<0.05, † *P*<0.01, ‡ *P*<0.001, # *P*<0.0001)

RemeGen-sponsored trial

Favorable Safety Profile

Consistent with data from clinical trials in SLE, RA, gMG, and IgAN, and post-marketing data

	Telitacicept 160mg (N=127)	Telitacicept 80 mg (N=126)	Placebo Group (N=127)
TEAE, n(%)	122 (96.1)	119 (94.4)	112 (88.2)
TRAE, n(%)	107 (84.3)	106 (84.1)	74 (58.3)
TESAE, n(%)	11 (8.7)	14 (11.1)	10 (7.9)
TRSAE, n(%)	2 (1.6)	5 (4.0)	4 (3.1)
Severe TEAE, n(%)	3 (2.4)	5 (4.0)	3 (2.4)
Severe TRAE, n(%)	0	1 (0.8)	1 (0.8)
Death, n(%)	0(0)	0(0)	0(0)
Common TEAE (incidence ≥10% in any group)			
Upper respiratory tract infections, n(%)	80 (63.0)	85 (67.5)	74 (58.3)
Urinary tract infection, n(%)	8 (6.3)	15 (11.9)	7 (5.5)
Cough, n(%)	11 (8.7)	14 (11.1)	8 (6.3)
Hepatic function abnormal, n(%)	7 (5.5)	16 (12.7)	7 (5.5)
Injection site reaction, n(%)	53 (41.7)	51 (40.5)	5 (3.9)
Pyrexia, n(%)	4 (3.1)	14 (11.1)	4 (3.1)

RemeGen-sponsored trial

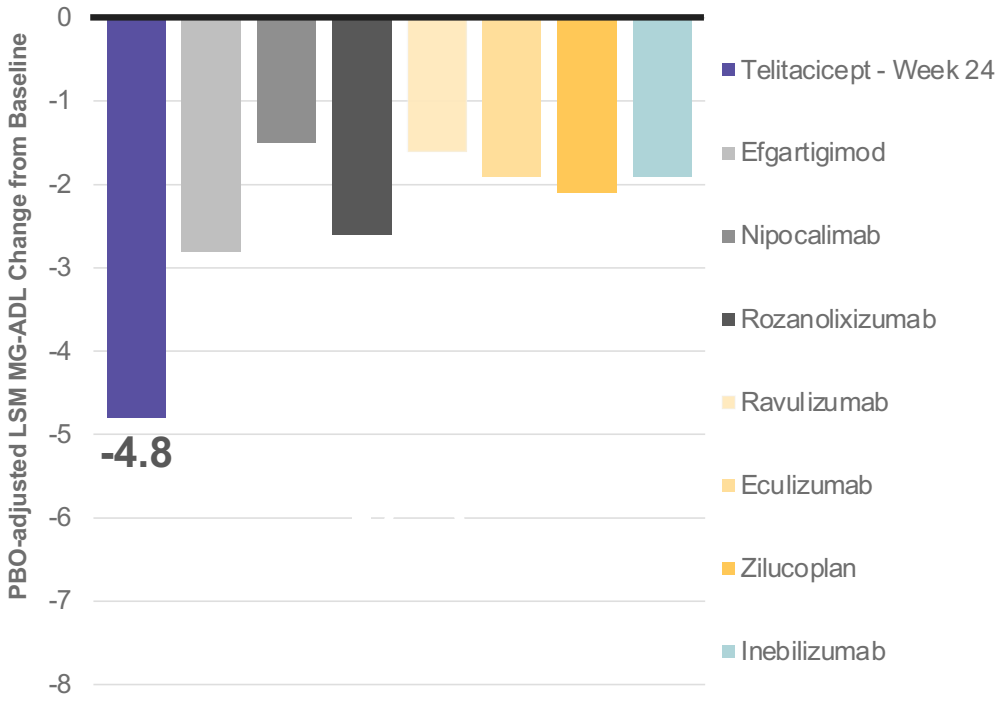
TEAE, treatment emergent adverse event; TRAE, treatment related adverse event; TESAE, treatment emergent serious adverse event; TRSAE, treatment related serious adverse event; SLE, systemic lupus erythematosus; RA, rheumatoid arthritis; gMG, generalized myasthenia gravis; IgAN, IgA nephropathy

pSD Commercial Opportunity

Dallan Murray, Chief Commercial Officer

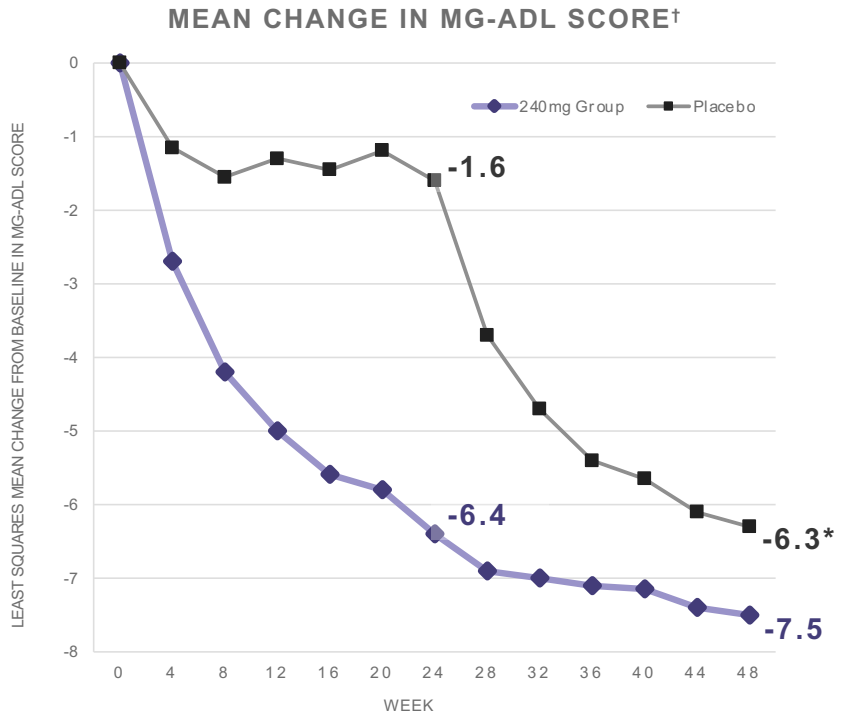
Telitacicept: Potential Best-In-Disease Efficacy Globally

Statistically significant and clinically meaningful improvement in MG-ADL score



Based on historical clinical data; not a head-to-head trial

Efgartigimod - ADAPT; Nipocalimab - Vivacity-MG3; Rozanolixizumab – MycarinG; Ravulizumab – CHAMPION-MG; Eculizumab – REGAIN; Zilucoplan – RAISE; Inebilizumab - MINT

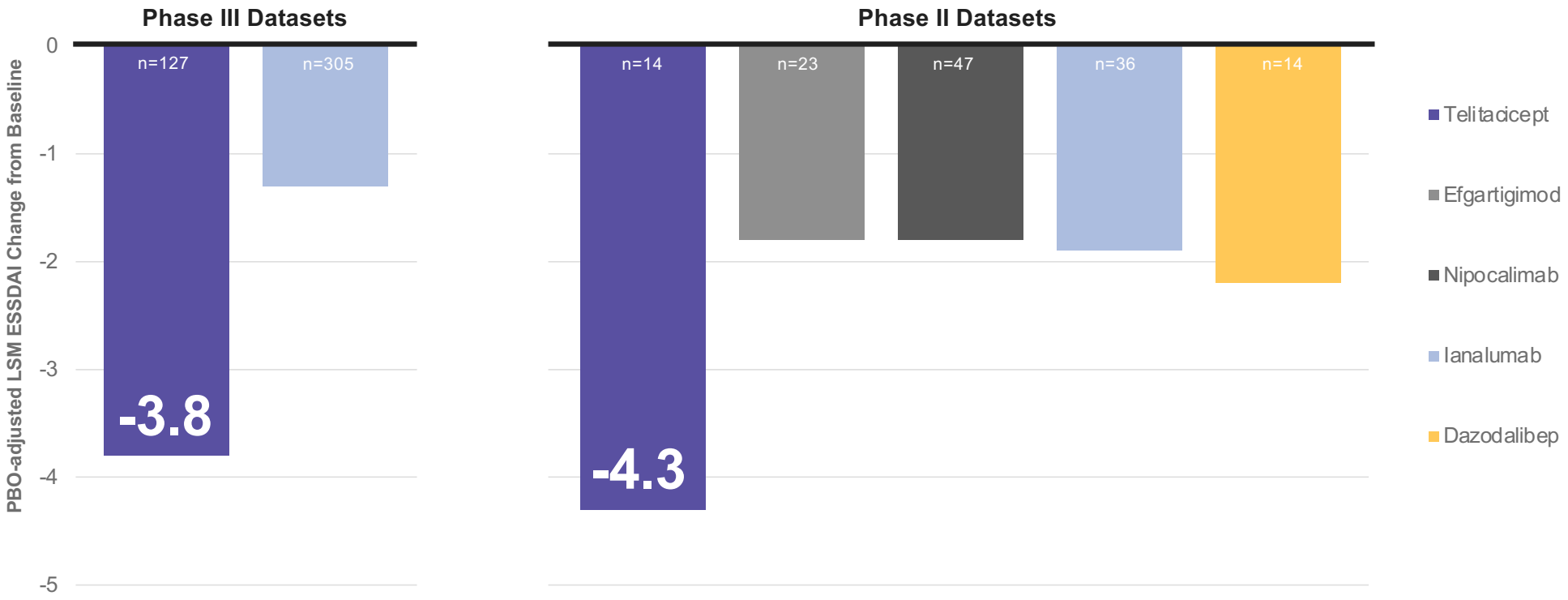


* Telitacicept arm continue with the same treatment, Placebo arm switched to Telitacicept during OLE period. The efficacy analysis was based on descriptive statistical analysis of the actual data in the full analysis set (FAS), and missing data were not filled.

RemeGen-sponsored trial

Telitacicept: Potential Best-In-Disease Efficacy Globally

Statistically significant and clinically meaningful improvement in ESSDAI



Based on historical clinical data; not a head-to-head trial

Efgartigimod - RHO; Nipocalimab - Bowman 2022, Lancet; Ianalumab - St. Clair 2024, Nature and Grader-Beck 2025 ACR; Dazodalibep - Xu 2024 Rheumatology

ESSDAI, EULAR Sjögren's syndrome disease activity index; mAb, monoclonal antibody

RemeGen-sponsored trial

Sjögren's: One of the Most Common Systemic Autoimmune Diseases Globally

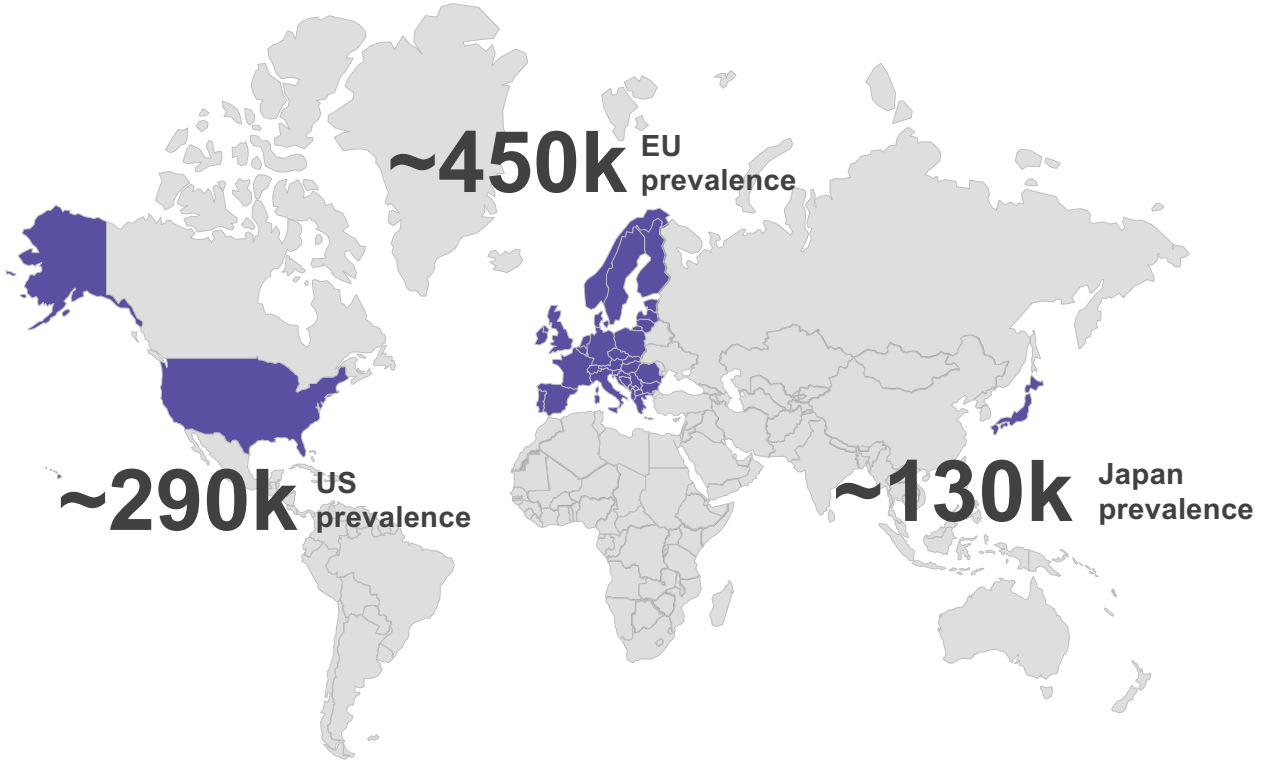
~870,000 diagnosed Sjögren's patients across key markets[1]

Favorable Growth Drivers

- Rising diagnosis rates from better awareness and testing

Underpenetrated Market

- No approved disease-modifying systemic therapies
- Patients managed with symptomatic treatments



1. Vor metanalysis and estimates; excludes Chinese market

Telitacicept Checks Every Box — Ideal Profile to Transform Sjögren's Disease

Expert Insights	Imperative	What Telitacicept Has Delivered
"I think ESSDAI is brilliant" *"At the minimum of two and preferably a three-point improvement in the ESSDAI index"*	**Prove Efficacy on ESSDAI** Show statistically significant ≥3-point reduction in ESSDAI	Phase III primary ESSDAI endpoint met with >3-point reduction vs placebo.
"I think just safety data is very important – probably most important"	**Must be Safe** Deliver a clean, non-cytotoxic B-cell approach; avoid infection or PML risk	Favorable safety profile — no PML, minimal infections through BAFF/APRIL modulation to regulate, not eradicate, B cells
"We need longer-term safety and real-world data"	**Long-Term Safety** Provide multi-year tolerability data supporting chronic use	Proven long term safety with 10s of thousands of commercially treated patients and ~1,800 patients treated in clinical trials
"I can't think of anything better than B cell targets and trying to make them safer"	**Deliver Disease Modification** Show modulation of underlying B-cell biology that stabilizes disease course, not just symptoms	Dual BAFF/APRIL blockade restores B-cell balance and supports long-term disease modification.

Telitacicept
Redefining Success In Autoimmune Disease

01

Telitacicept
The Most Advanced BAFF/APRIL Inhibitor Globally

Validated mechanism of action

Proven pipeline-in-a-product

Proof of concept in 5+ indications

02

Myasthenia Gravis
The Beachhead Indication

Significant MG-ADL and QMG score reduction at week 48

Opportunity to become the first disease modifying therapy in gMG

03

Primary Sjögren's Disease
The First Expansion Opportunity

Clinically meaningful, statistically clear physician- and patient-reported outcomes

Depth and durability across key domains

Favorable, consistent safety profile

Thank You.

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