Visionary Innovation

September 2026

Safe Harbor Statements

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Registrational programs for neuro-ophthalmic and ophthalmic indications target multi-billion-dollar market opportunities

- Global biopharma Nasdaq-listed company with **two registrational-stage candidates** targeting a market opportunity of over **$25B**
  - **Licaminlimab (Precision Med. DED):** First genotype-based dev. for **dry eye disease**, in **PREDICT-1** trial with topline results anticipated around year-end 2026
  - **Privosegtor (ON & NAION):** Potentially First-in-class **neuroprotective candidate,** in the **PIONEER** program for **optic neuritis** with **FDA Breakthrough Therapy** and **EMA PRIority MEdicines (PRIME) designations**, and for non-arteritic anterior ischemic optic neuropathy with plans to expand into acute MS relapse
- **Financials:** Strong balance sheet, no debt, and current cash runway into **2H 2029,** excluding a CHF100m loan facility

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Upcoming Value-Driving Milestones Across Our Targeted Registrational Programs

	Candidate	Phase 1	Phase 2	Phase 3	Upcoming Anticipated Value Catalysts
OPHTHALMOLOGY	**Licaminlimab** **Genotype-based development program**	Dry Eye Disease			**PREDICT-1** TLR in **around year-end 2026**
NEURO-OPHTHALMOLOGY	**Privosegtor** **Neuroprotective candidate**	Optic Neuritis — **Breakthrough Therapy and PRIME designations, SPA**			**PIONEER-1** enrollment completion in **2027** **PIONEER-2** trial initiation in **Q4 2026**
		NAION			**PIONEER-3** trial initiation in **Q4 2026**
		Acute MS relapses			Cross-reference optic neuritis IND for **new IND submission** in MS relapses in **Q4 2026**

IND: investigational new drug, MS: multiple sclerosis, NAION: non-arteritic anterior ischemic optic neuropathy, PRIME: priority medicine, SPA: special protocol assessment, TLR: topline results.
Privosegtor and Licaminlimab are investigational drugs, their safety or efficacy has not been established, and they have not received regulatory approval for commercial use in any country.

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Licaminlimab

Dry Eye Disease

Licaminlimab is a Novel Anti-TNFα Eye Drop for Ocular Inflammation with Clinically Proven MoA

Topical Biologic Candidate

Licaminlimab is an **anti-TNFα antibody fragment** specifically formulated for **topical** delivery

Clinically proven MoA

Anti-inflammation and **anti-apoptosis** MoA approved as systemic treatment for ocular disease and with **transformative** impact in other areas

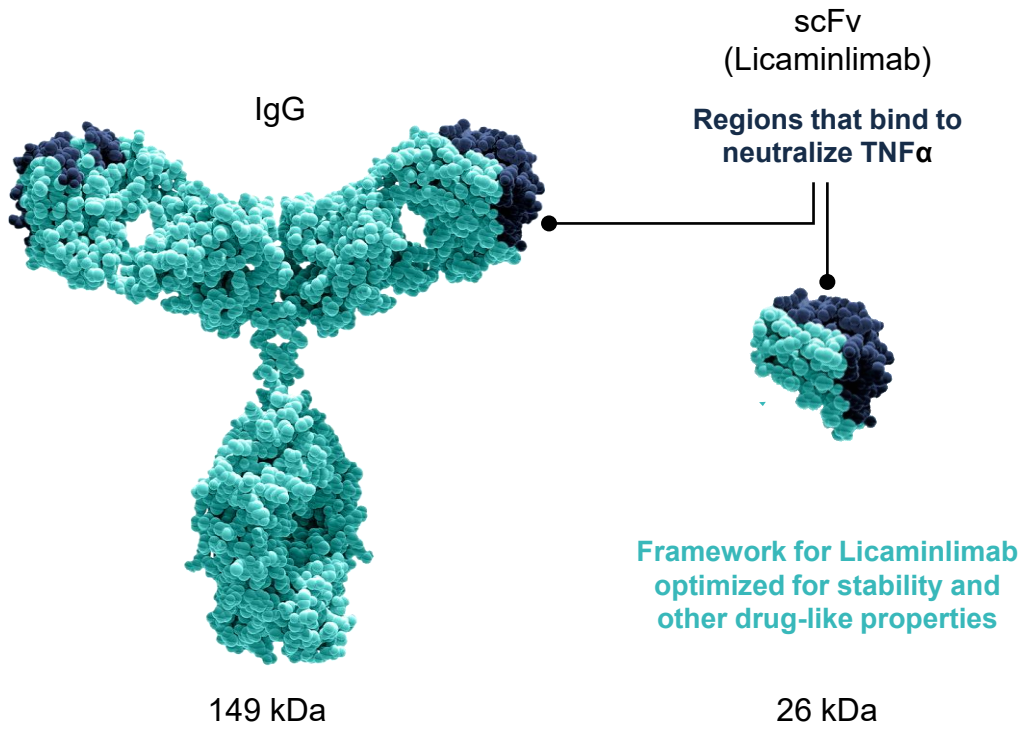
Enhanced ocular penetration

Lower molecular weight, **enhanced ocular penetration** and **higher concentration**

Proprietary genetic biomarker

Associated with **Licaminlimab** response highlights opportunity to drive **precision medicine** in DED

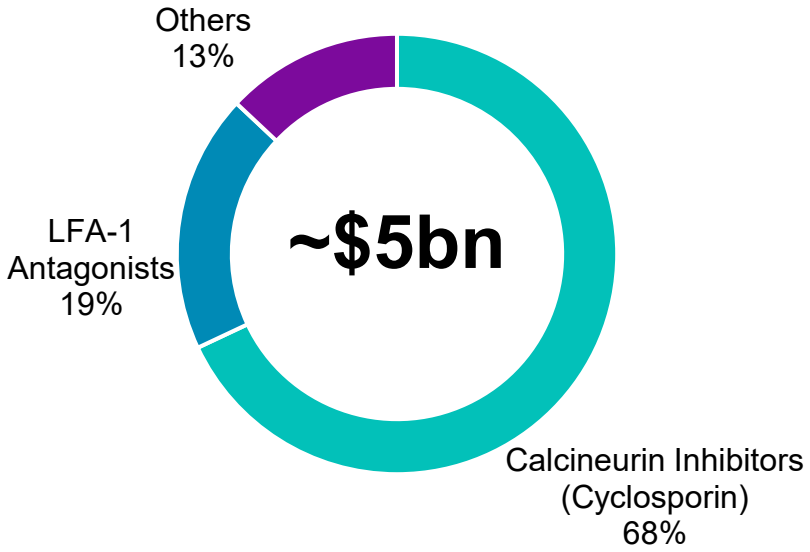
Innovative Antibody Fragment Technology



DED: dry eye disease; MoA: mechanism of action.

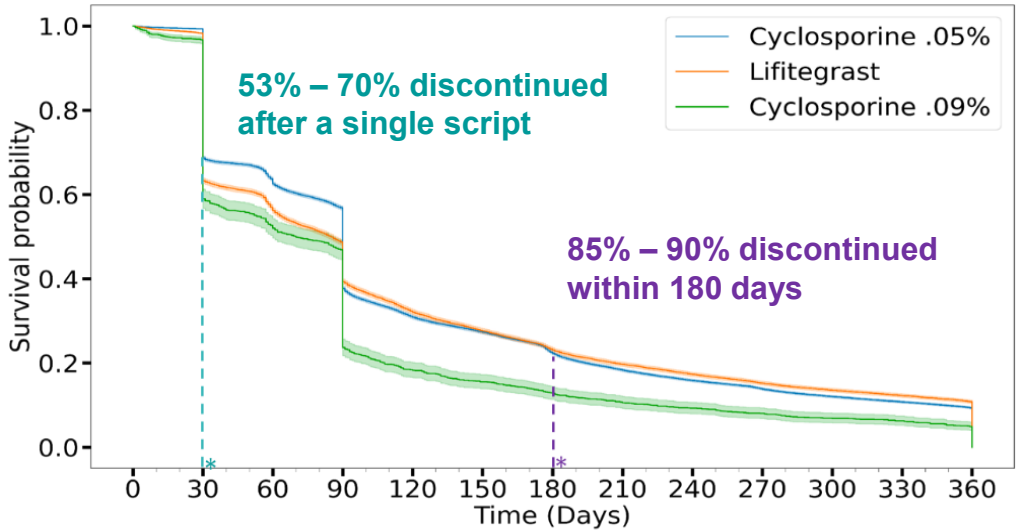
Large Unsatisfied Market with Only 13% of Patients Experiencing Lasting Relief After 12 Months with Current Treatments[1]

2024 Dry Eye Rx drug market in G7 countries[2] and U.S. Rx split[3]



Driven by trial and error with significant unmet needs

Discontinuation & switching is commonplace in DED[4]



Establishing Licaminlimab as a precision-based approach, in a market with high failure and discontinuation rates, is expected to provide a clear option for physicians and payors for TNFR1 genotype patients

1. Health Union Community Editorial Team. 2021 In America Survey Findings: Living With Chronic Dry Eye. Chronic Dry Eye. 2021. https://chronicdryeye.net/infographic/in-america-findings.
2. DRG Dry Eye Disease Landscape and Forecast 2020 (market value in 2024)
3. IQVIA DED report, data on file. Prescriptions volume in DED March 2024 for split per drug class
4. Mbagwu M, et al. Characterization of Discontinuation and Switching Patterns of Dry Eye Disease Medications Using Linked EHR Registry and Claims Data. Presented at: ASCRS Annual Meeting 2024 https://ophthalmology360.com/study-finds-high-discontinuation-rate-of-dry-eye-medications/

Licaminlimab: Three Positive DED Phase 2 Trials Completed with Consistent Results and Potential for Precision Medicine Approach

Phase 2 Randomized Controlled Studies in DED

- **DED#1 Symptoms**
  85 patients Phase 2 PoC
- **DED#2 Symptoms**
  134 patients Phase 2 PoC
- **DED#3 (RELIEF) Signs**
  122 patients Phase 2b

Consistent positive results across studies and unique genotype-based development opportunity

01 Meaningful and rapid treatment effect in signs and symptoms

02 More pronounced treatment effect in TNFR1 genotype positive (5X in signs and 7X in symptoms)

03 Well-tolerated, drop comfort like artificial tears

PREDICT genotype-based registrational trial initiated in Q4 2025 in active site recruitment phase with topline results anticipated around year-end 2026

DED#1 and DED#3: Data on file
DED #2: Sheetle L, et al. *Clin. Ophthalmol.* 2022; 16:2167-2177

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DED#2: Licaminlimab (OCS-02) Phase 2a Trial Evaluating Symptoms in DED

Phase 2a study design

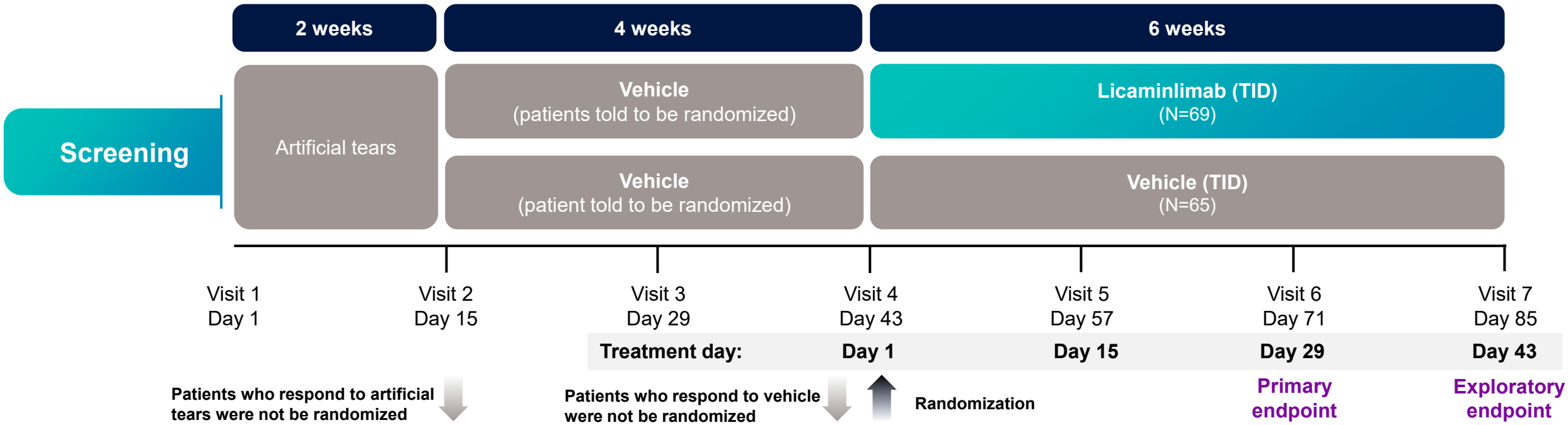
- Randomized, double-blind, vehicle-controlled, 6-week multi-center trial
- 134 participants

Efficacy endpoints

- **Primary endpoint:** Change from baseline in global ocular discomfort score* at Day 29
- **Secondary endpoint:** % of high responders**

Safety Assessments

- Ophthalmic evaluation & adverse events



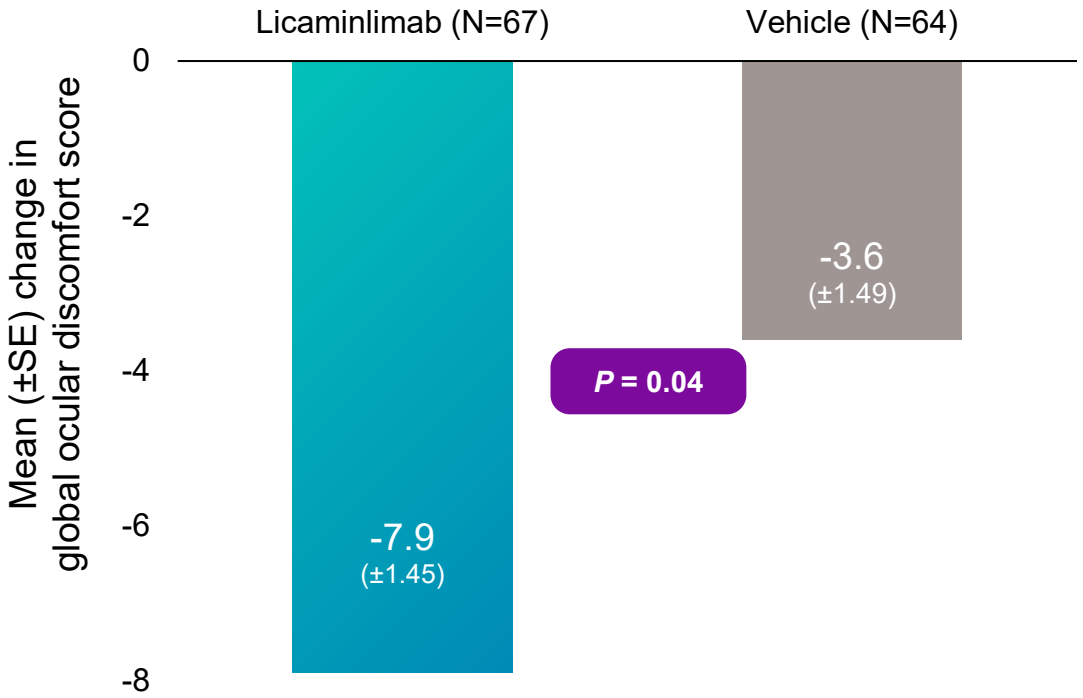
*Change from baseline in global ocular discomfort score based on the Symptom Assessment iN Dry Eye (SANDE) questionnaire. ** patients with improvement in global ocular discomfort score >20) DED: dry eye disease.

Shettle L, et al. Clin. Ophthalmol. 2022;16:2167-2177.

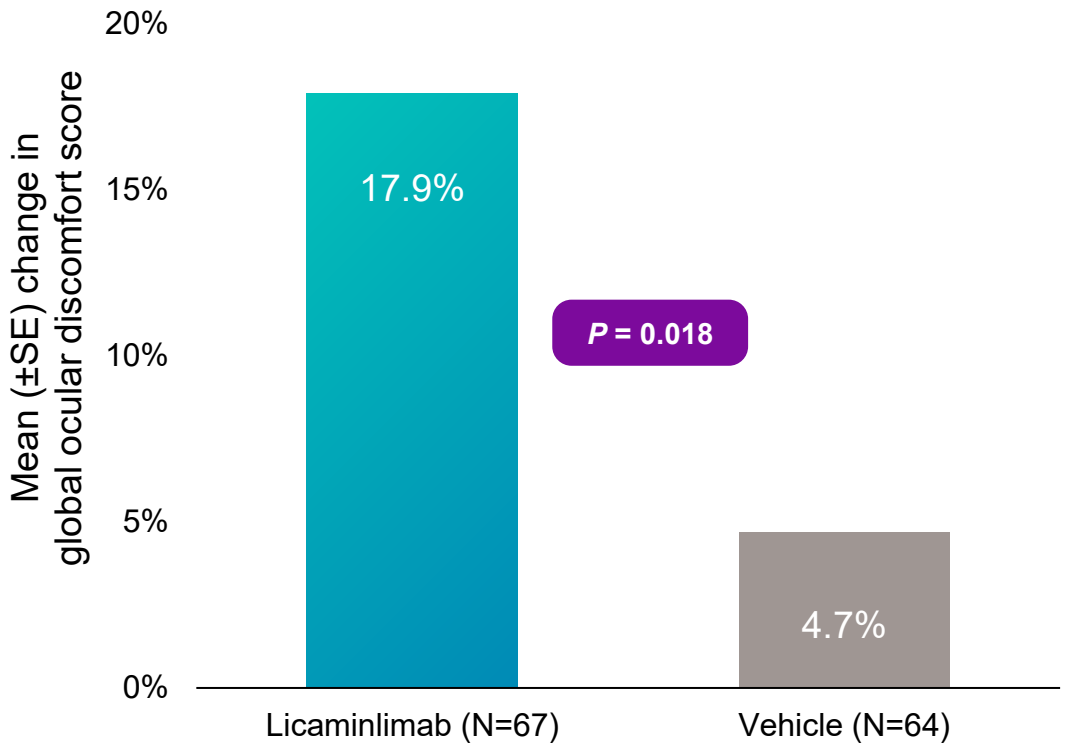
Licaminlimab (OCS-02) Phase 2a Trial Evaluating Symptoms in DED

Statistically significant reduction in ocular discomfort and greater percentage of high responders with Licaminlimab (OCS-02) vs. vehicle

Primary Endpoint - Mean Change In Global Ocular Discomfort Score* At Day 29



Secondary endpoint – Percentage of high responder patients at Day 29



*Change from baseline in global ocular discomfort score based on the Symptom Assessment iN Dry Eye (SANDE) questionnaire.
DED: dry eye disease.
Shettle L, et al. Clin. Ophthalmol. 2022;16:2167-2177.

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DED#3: RELIEF Phase 2b in Signs of DED

Designed to identify the most relevant endpoint in signs and assess TNFR1 genetic biomarker in signs in DED

Phase 2b study design

- Randomized, masked, vehicle-controlled study
- Multi-center, 10-week trial stratification based on TNFR1-related genotype ~20% patients

Primary Objective

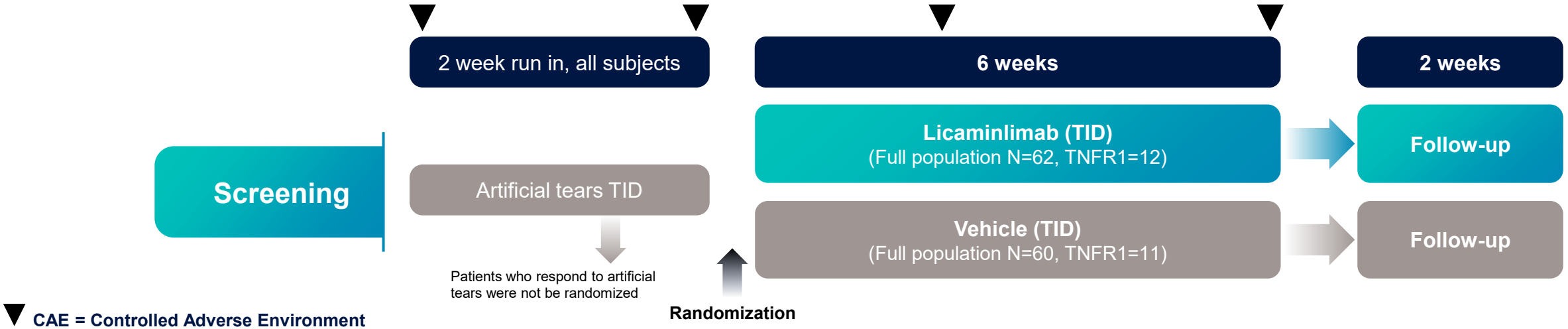
- Evaluate efficacy and safety in subjects with signs of dry eye disease

Endpoints

- Corneal / Conjunctival Staining, Redness and Schirmer's

Patient Population

- Moderate to severe corneal staining
- Susceptible to corneal damage of the inferior region under stressed conditions
- Decreased tear production (Schirmer's test at baseline)



Licaminlimab (OCS-02) Effect on Inferior Corneal Staining

Meaningful treatment effect in the full population and more pronounced in TNFR1-related genotype group

	Pre- to Post-CAE change from baseline at Day 43 Difference in means of OCS-02 vs Vehicle; (CI)*			
Efficacy Measures (accepted by regulators)	**Full Population Licaminlimab (n=62); Vehicle (N=60)**	**TNFR1 Genotype Licaminlimab (n=12); Vehicle (N=11)**	**Treatment Effect Favors Licaminlimab over Vehicle in Full population**	**Treatment Effect Favors Licaminlimab over Vehicle more pronounced in TNFR1 Genotype Group**
Inferior Corneal Staining	**-0.12** (-0.378, 0.134)	**-0.59** (-1.165, -0.017)	✓	✓ ✓

*90% CI for Difference in Means based on the t-distribution; sample t-test: directional nominal p-value

- Corneal staining is reflective of inflammation and apoptosis which play crucial roles in DED
- Corneal staining, mainly the inferior part (given its exposure), is also the most commonly assessed sign in clinical practice as it can affect quality of vision

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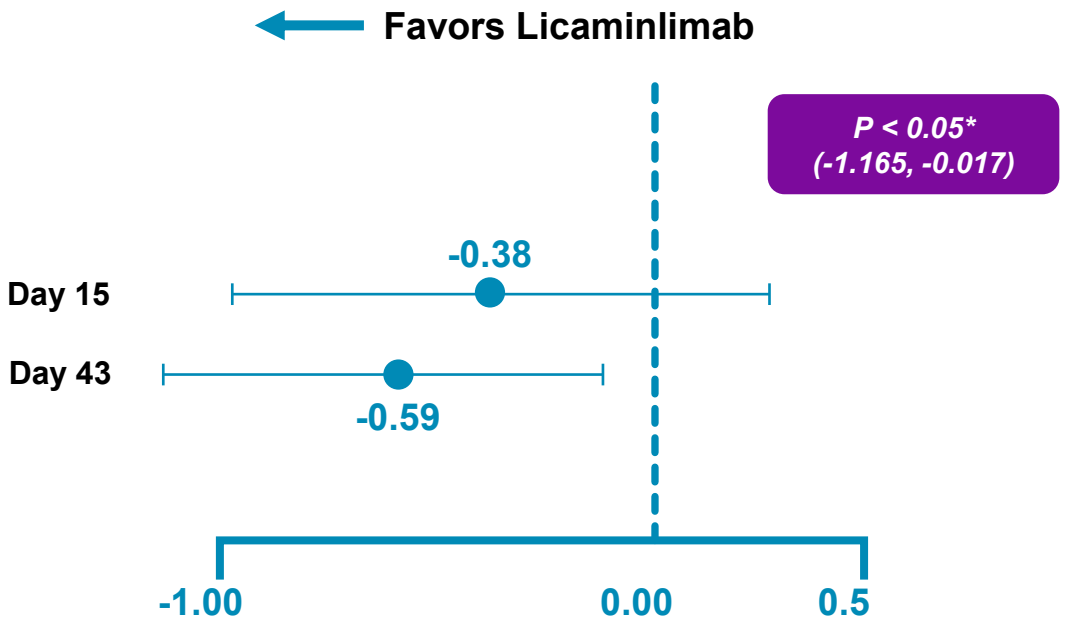
Licaminlimab (OCS-02) in Phase 2b: Effect on Inferior Corneal Staining in TNFR1 Genetic Biomarker Population

Mean change from baseline (Pre- to Post-CAE)

Visit	Licaminlimab (N = 12)	Vehicle (N = 11)	Difference (90% CI)
Baseline	1.46	1.23	
Day 15	-0.29	+0.09	**-0.38** (-1.012, 0.247)
Day 43	-0.50	+0.09	**-0.59** (-1.165, -0.017)



90% CI for Difference in Means based on the t-distribution; sample t-test: 1-sided directional nominal p-value

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Opportunity for Highly Differentiated Product Profile

Licaminlimab (OCS-02) has potential to address key unmet needs and transform the treatment paradigm of DED

Unmet Needs in DED[1]	Licaminlimab (OCS-02)
New MoA targeting both signs and symptoms	✓ Meaningful treatment effect in both signs and symptoms with a potential disease-modifying TNFα inhibitor
Rapid onset of action	✓ Symptoms and signs improvement seen as early as 2 weeks
Good tolerability and drop comfort	✓ Mild and transient AEs reported with drop comfort consistent with artificial tears
Ability to predict treatment response	✓ Treatment effect in TNFR1 genotype group was 5-fold higher in signs and 7-fold higher in symptoms

1. DRG Dry Eye Disease Landscape and Forecast 2020

Licaminlimab First Genotype-based Development to Drive Precision Medicine in DED

Registration Program Started with PREDICT-1 Trial in Q4 2025

Phase 2/3 Study Design

- Randomized, multicenter, double-masked, vehicle-controlled, 6-week study
- N= ~160 patients, 1:1 randomization

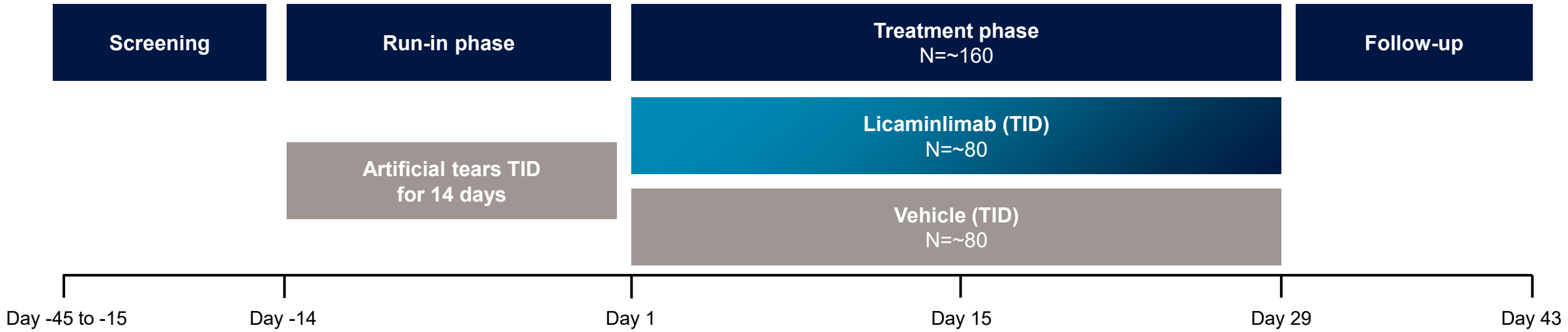
Key Efficacy Endpoints

Primary endpoint:
Global ocular discomfort score* at Day 29 in patients with TNFR1 genotype positive

Key secondary endpoint:
Global ocular discomfort score at Day 29 in all patients

Study Population

- TNFR1 genotype: ~2/3 positive
- Diagnosis of DED of at least 6 months
- Global ocular discomfort score of ≥ 60



*Global discomfort score is a composite of discomfort frequency and severity as assessed by a visual analog scale.
DED: dry eye disease. FDA: Food and Drug Administration. TID: Three times a day. TNFR1: Tumor necrosis factor receptor 1

Privosegtor

Optic Neuritis

Privosegtor Product Overview

A potential first-in-class neuroprotective therapy for the optic nerve and CNS

Privosegtor

Novel Small Molecule

Peptoid-based small molecule with a differentiated profile enabling broad CNS & retinal access

Crosses Blood-brain and Retinal Barriers

Privosegtor penetrates both the blood-brain barrier and retinal barrier, reaching the site of neuronal injury where it matters

Prevents Neuronal Apoptosis

Promotes neuro-axonal survival by protecting neurons from apoptosis — validated across apoptosis, oxidative stress and inflammation injury models

Neuroprotective Platform

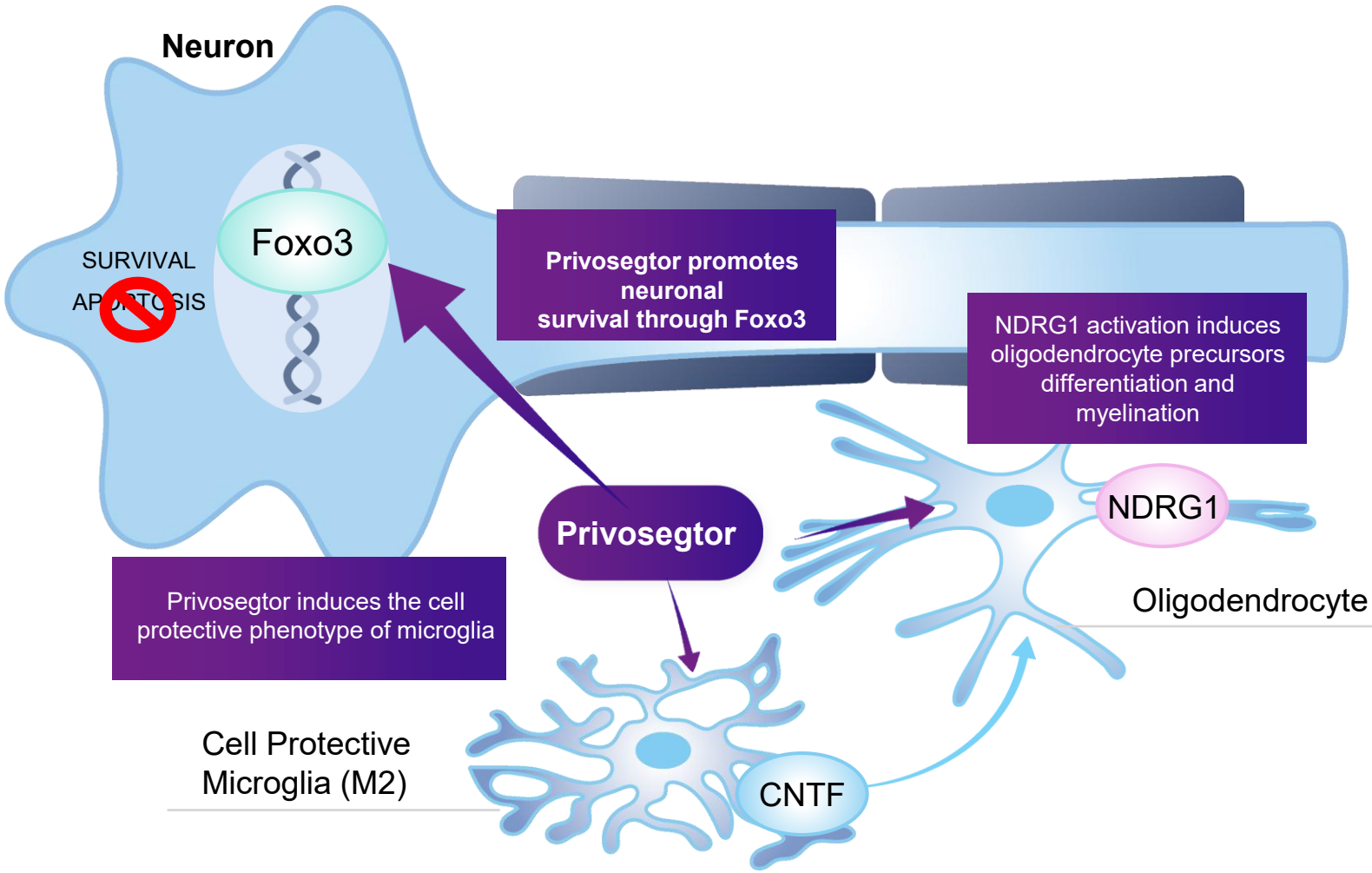
Potential to expand across acute CNS injury — from optic neuritis and NAION to acute MS relapses and other neurodegenerative conditions

Neuroprotection Matters

Once retinal ganglion cells and their axons die, the loss is permanent. Privosegtor was selected by high-throughput screening for its capacity to promote neuro-axonal survival, and protects neurons across apoptosis, oxidative stress and inflammation models.

Privosegtor Is a Novel Neuroprotective Candidate with Broad Potential for Neuro-axonal Diseases

- Peptoid small molecule that **crosses blood brain** and **retinal barriers**
- Developed using a hybrid drug discovery approach that combined rational medicinal chemistry design with phenotypic drug selection for its unique ability to **promote neuro-axonal survival,** validated across multiple in vitro injury models: **apoptosis, oxidation, and inflammation**
- Confirmed **neuro-axonal survival** in vivo in glaucoma, MS, and optic neuritis models
- FDA **Breakthrough Therapy Designation** and EMA **PRIority MEdicines (PRIME)** designations granted for optic neuritis



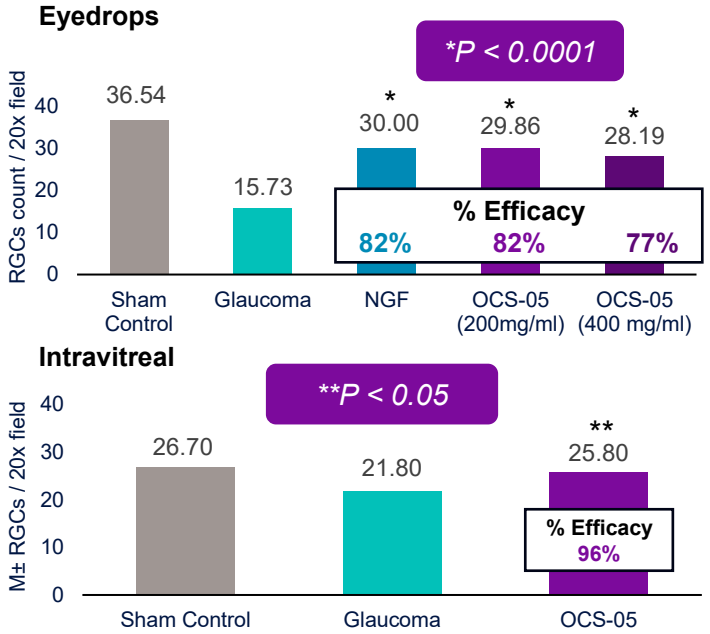
CNTF: ciliary neurotrophic factor; GSK3b: glycogen synthase kinase-3 beta; MoA: mechanism of action; NDRG1: N-myc downstream regulated; MS: multiple sclerosis; SGK: serum glucocorticoid kinase

Privosegtor | Pre-clinical Evidence of Neuroprotective Activity (1/2)

Compelling data showing prevention of RGC damage in glaucoma, acute optic neuritis and functional improvement in EAE models

OCS-05, IVT and topical, shown to **prevent RGC damage** (the key element in Glaucoma vision loss)

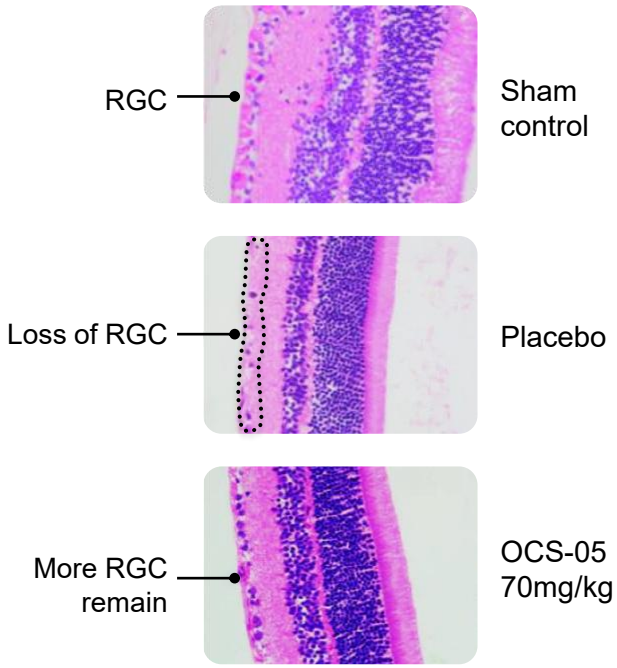
OCS – 05 | H&E for RGC density at week 6[1]



High-pressure Glaucoma rat model of neurodegeneration without inflammation

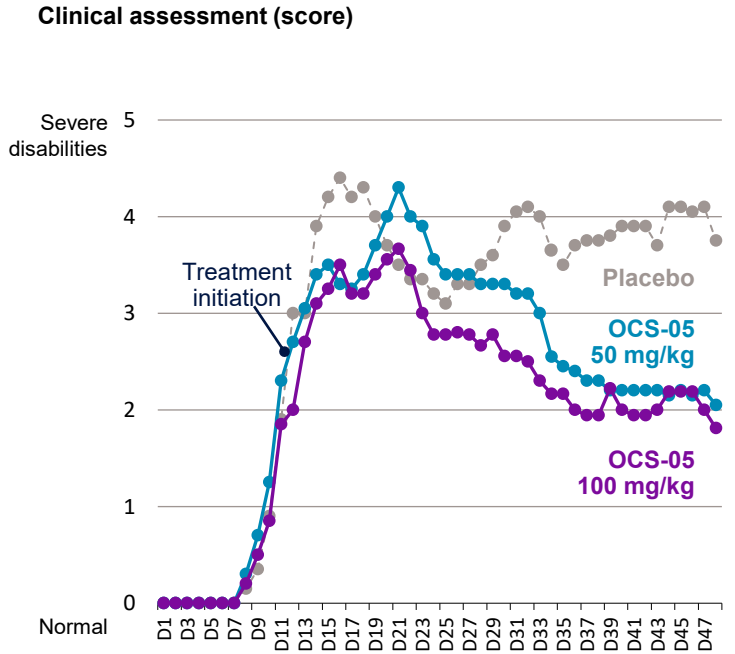
Acute optic neuritis model (5-day treatment, assessment at day 6)

Visual of RGC Protection



OCS-05 shown to promote **improvement of mobility** in experimental autoimmune encephalomyelitis (EAE) model

OCS – 05 | Model of autoimmune AON and MS



Experimental Autoimmune Encephalomyelitis model in mice

EAE: experimental autoimmune encephalomyelitis; H&E: hematoxylin and eosin staining; MS: multiple sclerosis; RGC: retinal ganglion cell; IVT: intravitreal; NGF; nerve growth factor.

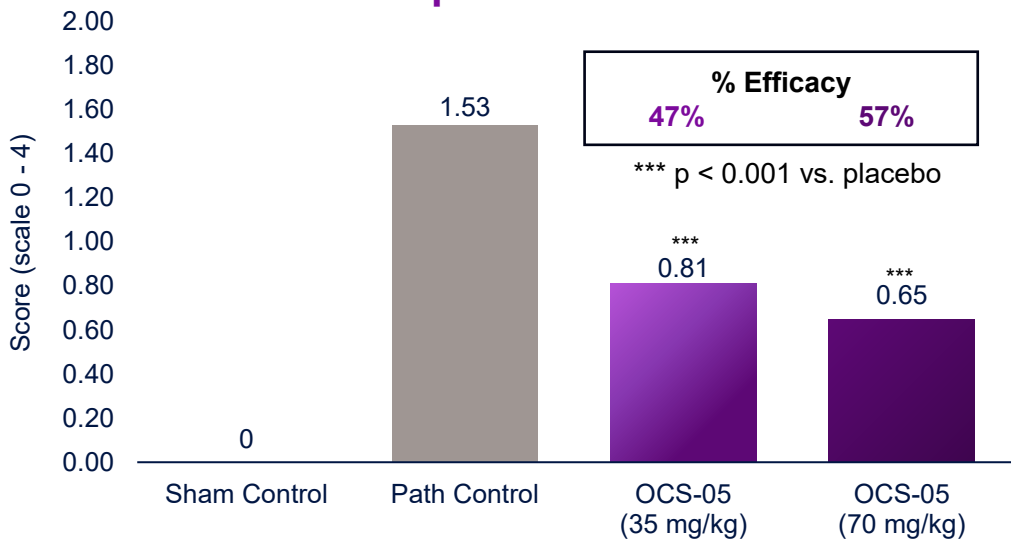
1. Villoslada P. et al. Neurotherapeutics. 2019; 16(3):808-827

Privosegtor | Pre-clinical Evidence of Neuroprotective Activity (2/2)

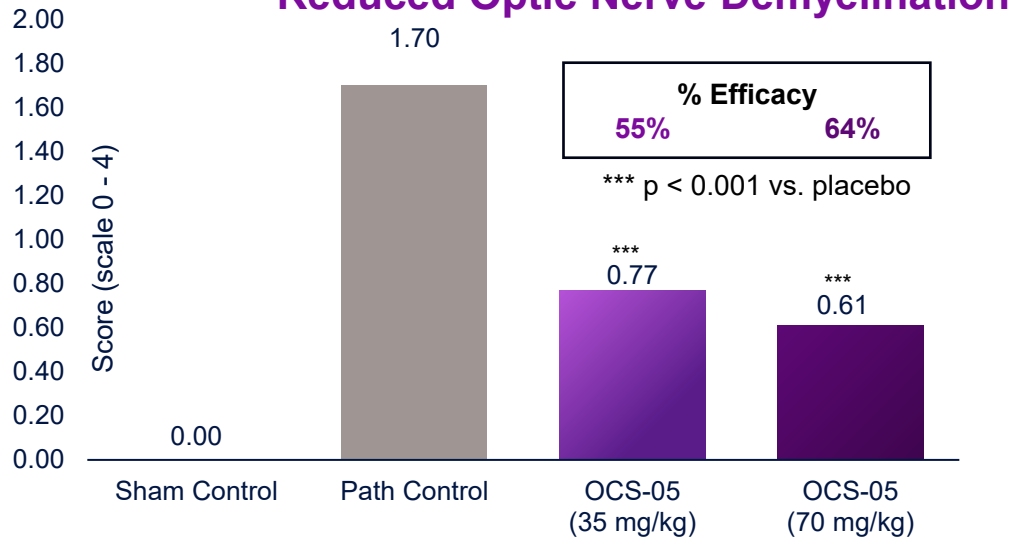
Promotes axonal sparing and reduces demyelination in model of acute optic neuritis

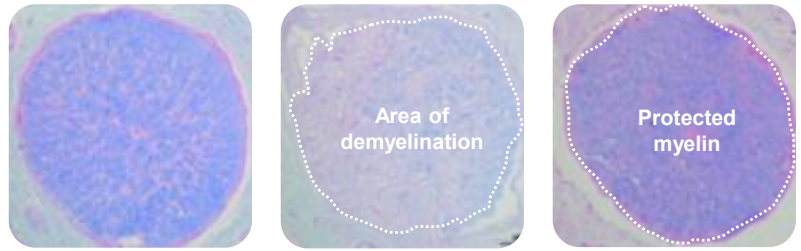
Assessment after 5-days of treatment[1]

Reduced Optic Nerve Axonal Loss



Reduced Optic Nerve Demyelination





Lysolecithin induced demyelinating model in rat (model of acute optic neuritis)

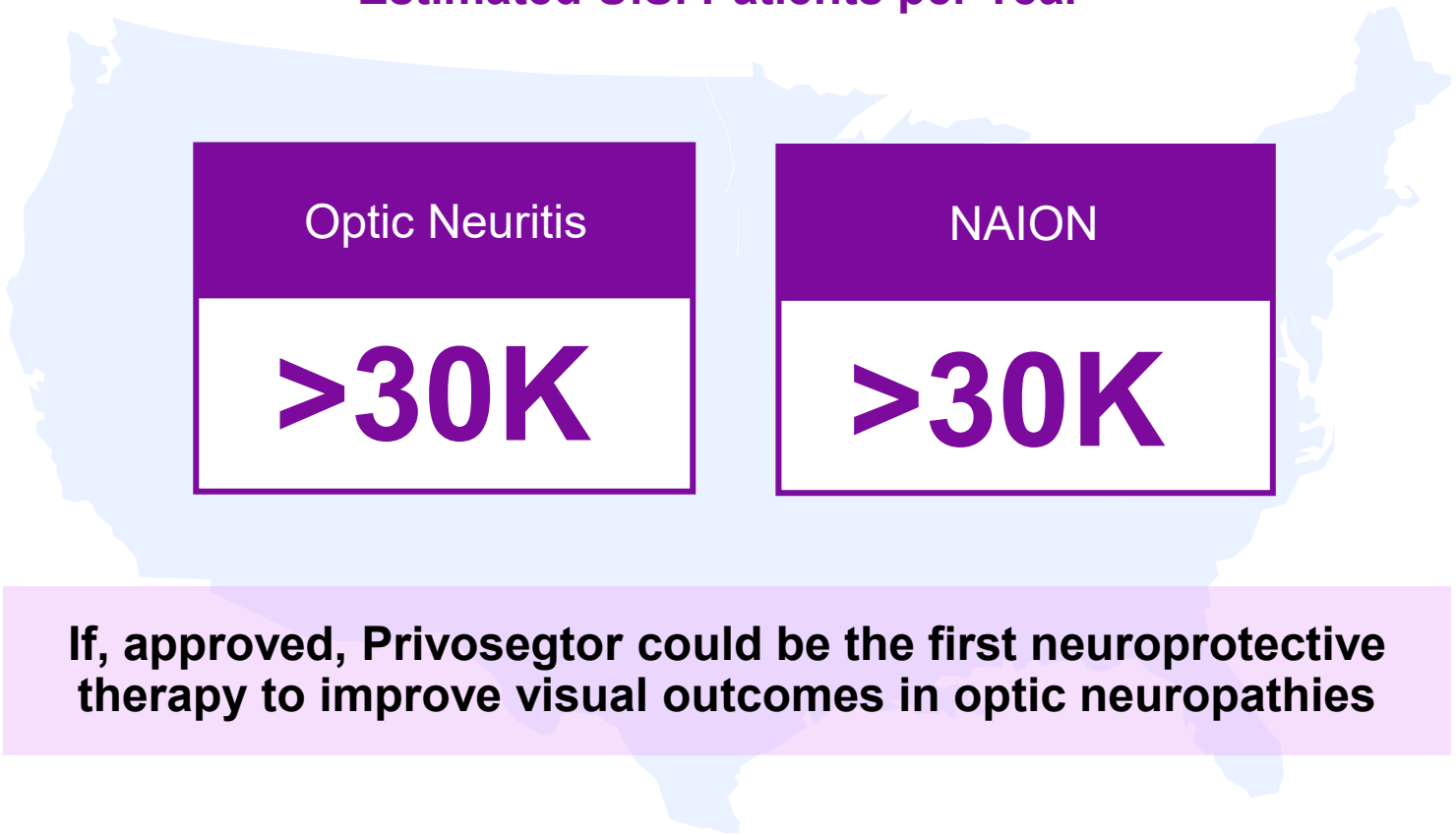
*** p < 0.001 vs. placebo

1. Villoslada P, et al. *Neurotherapeutics*. 2019;16(3):808-827.

Opportunity to Change Standard of Care for Optic Neuropathies

Two Optic Neuropathies Under Same IND

Estimated U.S. Patients per Year[1,2]



Optic Neuritis	NAION
>30K	>30K

If, approved, Privosegtor could be the first neuroprotective therapy to improve visual outcomes in optic neuropathies

Estimated U.S. Market Potential

>$7B

- Rare diseases without approved therapies often under-diagnosed
- Price analogs: $100k-$400k per treatment[3,4]
- Highly concentrated: ~450 neuro-ophthalmologists in U.S.[5]

1. Weidong Gu et al. (2023) Incidence of Optic Neuritis and the Associated Risk of Multiple Sclerosis for Service Members of U.S. Armed Forces, Military Medicine, vol. 188, March/April 2023
2. Incidence of nonarteritic anterior ischemic optic neuropathy – PubMed and Incidence of nonarteritic anterior ischemic optic neuropathy: increased risk among diabetic patients – PMC and discussions with experts
3. https://www.medicalmex.com/oxervate-cenegermin-bkbj/ Oxervate pricing in U.S. $96k-$120k
4. https://iovs.arvojournals.org/article.aspx?articleid=2783085 Tepezza pricing in U.S. $386k
5. Active members of the American Academy of Ophthalmology, self-reported subspecialty, EyeNet Media Kit 2025

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Optic Neuritis, an Acute Inflammation of the Optic Nerve which Can Lead to Permanent Visual Impairment

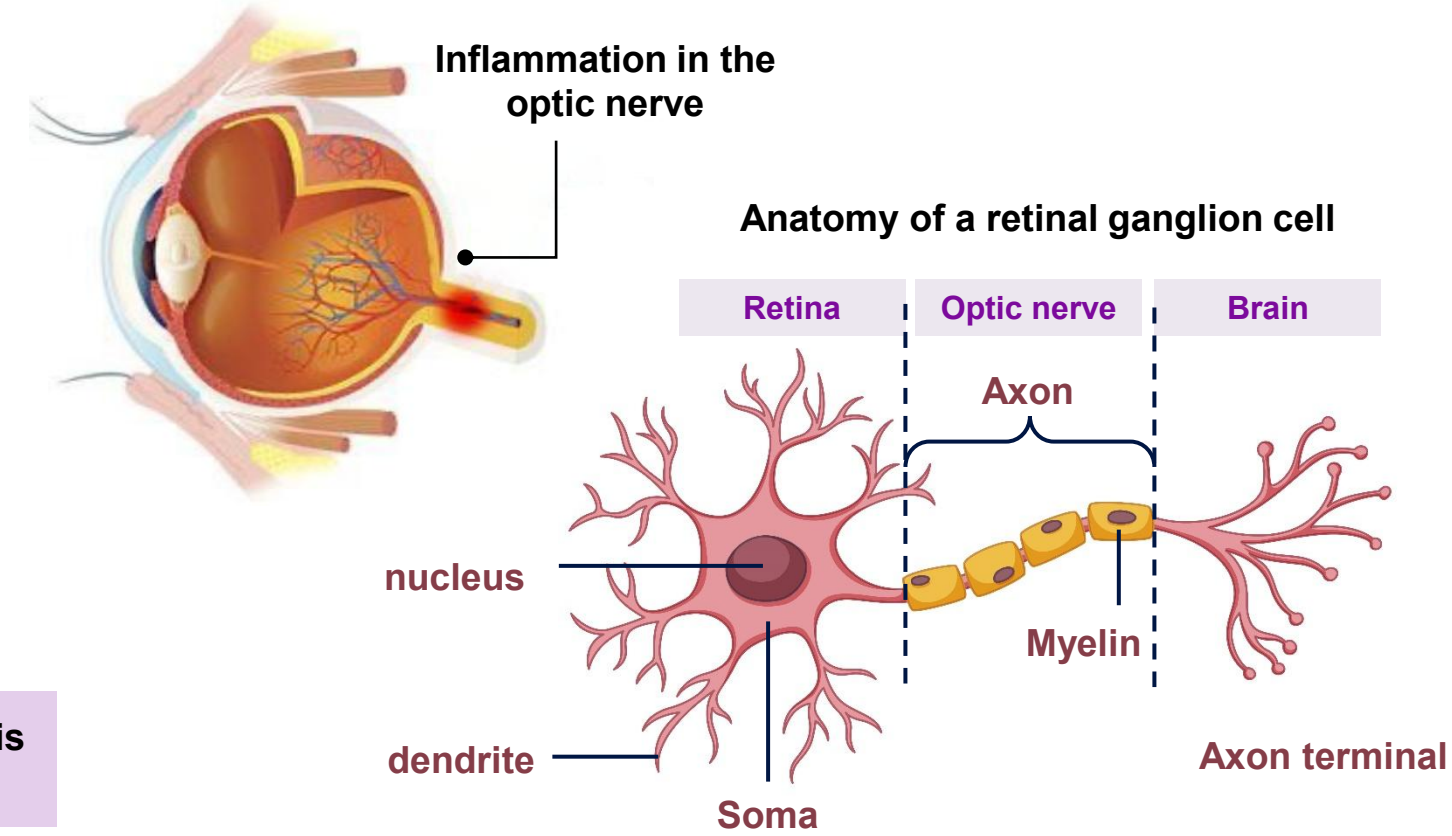
Orphan indication with ~ 65k patients per year (US/EU)[1,2]

Acute inflammation of the optic nerve impacting retinal ganglion cells and leading to vision loss

- Type of neuropathy causing vision loss and pain, and **can lead to permanent visual impairment**

- **Inflammation** affects the signals through the **optic nerve**, which connects the eyes and the brain
- **Mainly affecting young women** with an average onset at age 32[3]

Direct link with chronic conditions like **multiple sclerosis (MS)** and other autoimmune diseases



1. Weidong Gu et al. (2023) Incidence of Optic Neuritis and the Associated Risk of Multiple Sclerosis for Service Members of U.S. Armed Forces, Military Medicine, vol. 188, March/April 2023
2. Martínez-Lapiscina EH, et al. (2014): Is the incidence of optic neuritis rising? Evidence from an epidemiological study in Barcelona (Spain) 2008-2012. *J Neurol*. 2014 Apr; 261(4): 759-767.
3. Guier CP, Kaur K, Stokkermans TJ. Optic Neuritis. January 2025. StatPearls. https://www.ncbi.nlm.nih.gov/books/NBK557853

Successful Phase 2 ACUITY Trial Investigated Safety and Efficacy of Privosegtor in Optic Neuritis

Study Design

- Randomized, double-masked, placebo-controlled study
- Multi-center, 6-month trial with 36 patients randomized (mITT: 33)

Key Endpoints

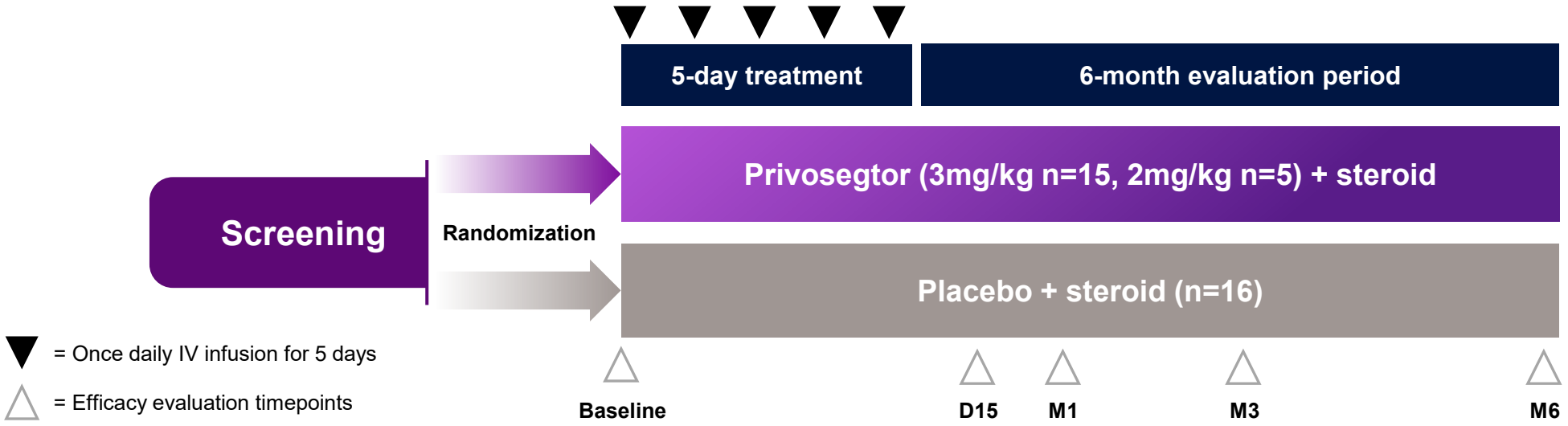
Primary: Safety

Secondary:

- Function: LCVA
- Anatomy: GCIPL and RNFL thickness
- Biology: Neurofilaments

Study Population

- Unilateral optic neuritis with a demyelinating origin
- Onset of visual loss symptoms in the last 12 days before randomization



mITT: Modified Intent to Treat; LCVA: low contrast visual acuity; GCIPL: ganglion cell plus inner plexiform layer; RNFL: retinal nerve fiber layer; D: day; M: month

https://clinicaltrials.gov/study/NCT04762017

Patient Demographics and Baseline Characteristics

	Privosegtor + steroid 3 mg/kg/day (N = 15)	Placebo + steroid (N = 14)
Age, mean (SD), years	33.7 (9.8)	32.7 (10.3)
Female, n (%)	9 (60.0)	10 (71.4)
GCIPL thickness, mean (SD), µm	89.3 (8.3)	84.3 (13.8)
RNFL thickness, mean (SD), µm	104.6 (13.1)	115.5 (54.1)
HCVA, mean (SD), ETDRS	54.1 (34.5)	42.6 (34.5)
LCVA, mean (SD), ETDRS	19.4 (22.3)	17.8 (24.3)
Visual Field Mean Deviation, mean (SD), dB	-14.1 (11.9)	-14.5 (12.5)
Time since first visual loss symptoms at date of first dose, mean (SD), days	9.5 (2.7)	9.6 (2.5)
Multiple sclerosis at baseline, n (%)	10 (66.7)	9 (64.3)
Disease Modifying Therapies n (%)	10 (66.7)	9 (64.3)

GCIPL, ganglion cell plus inner plexiform layer; HCVA, high contrast visual acuity; LCVA, low contrast visual acuity; RNFL, retinal nerve fiber layer;
Data, analysis and conclusions are preliminary, and subject to change as full analysis is ongoing.

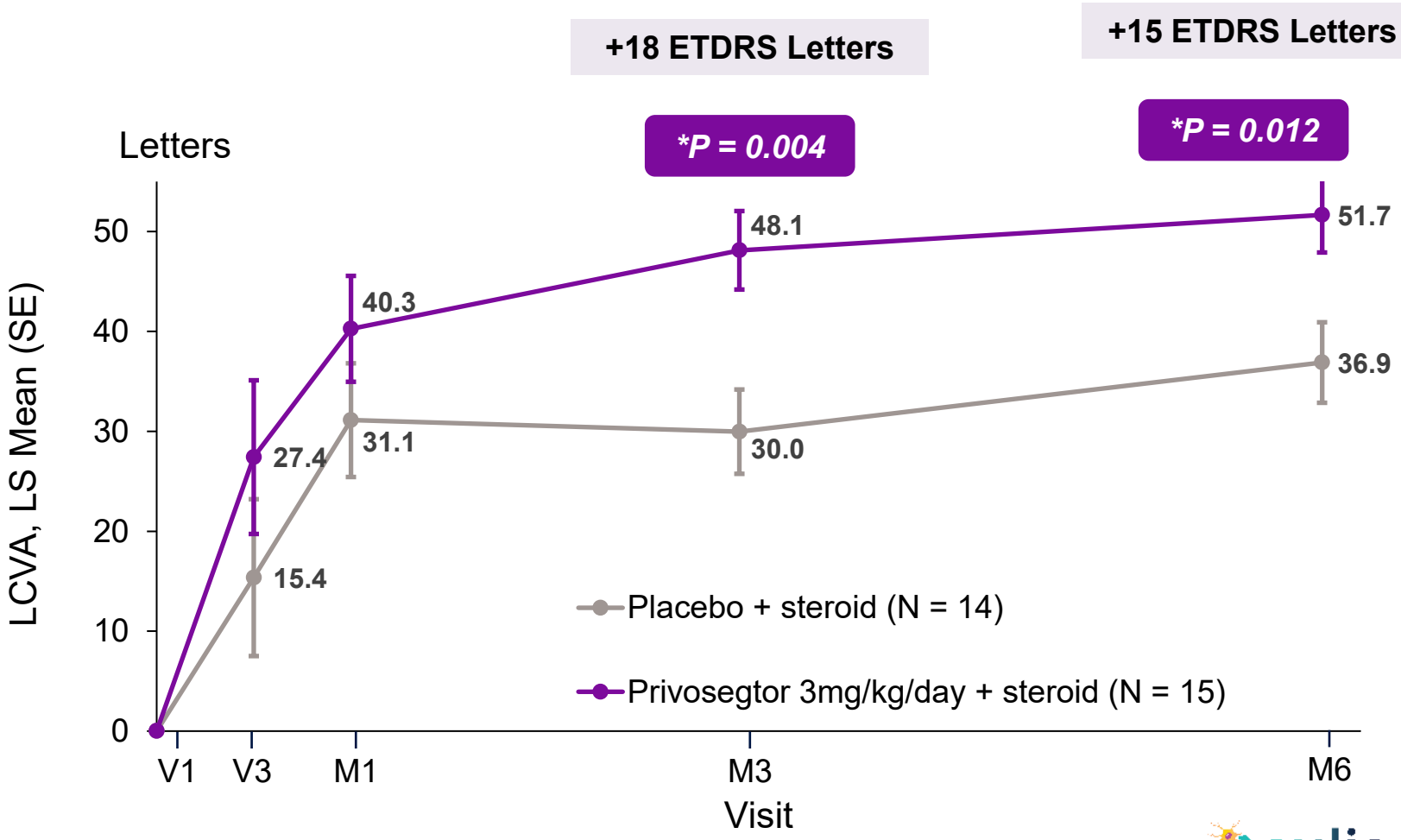
Patients in the Privosegtor 3mg/kg/day Arm Achieved Clinically Meaningful and Sustained Improvement in Visual Function

FUNCTION

LCVA: Low-contrast visual acuity is often affected in patients with ON

LCVA

2.5% ETDRS LCVA in the Affected Eye: MMRM, LS Mean Change From Baseline, mITT

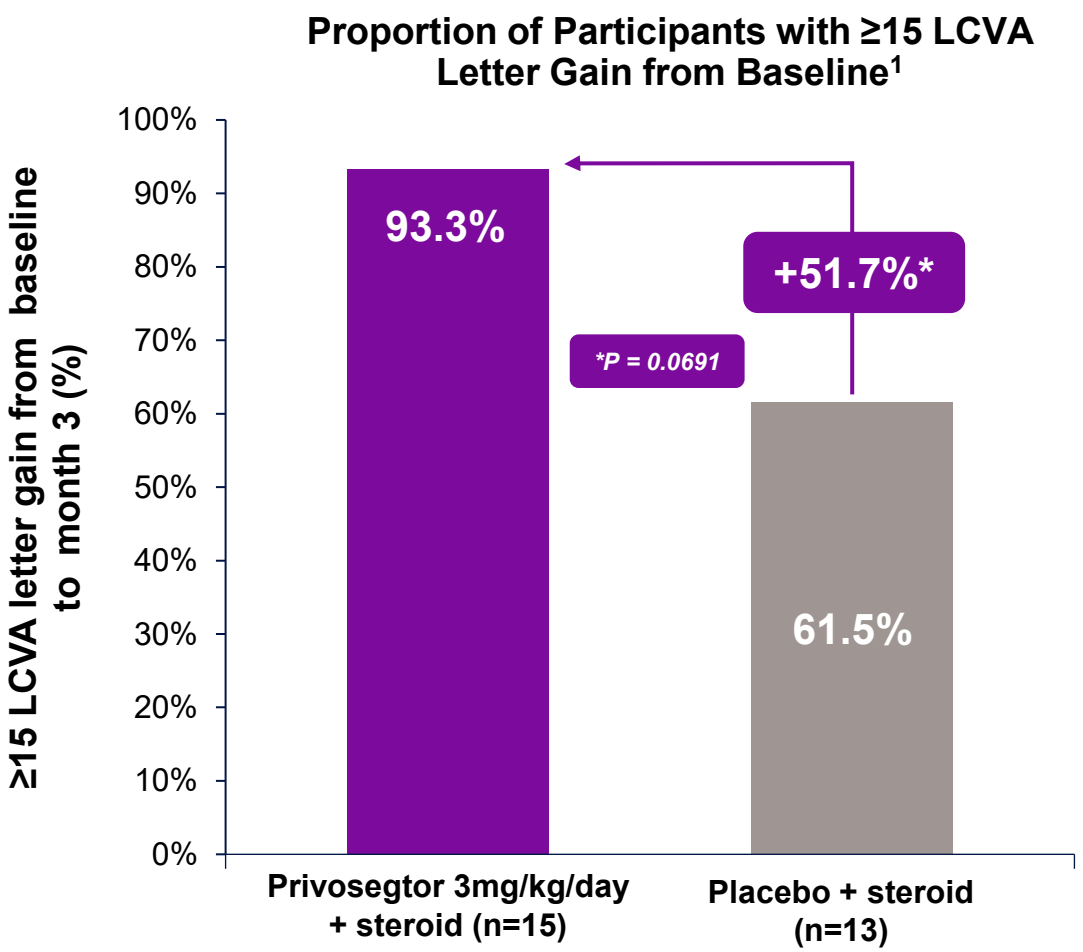


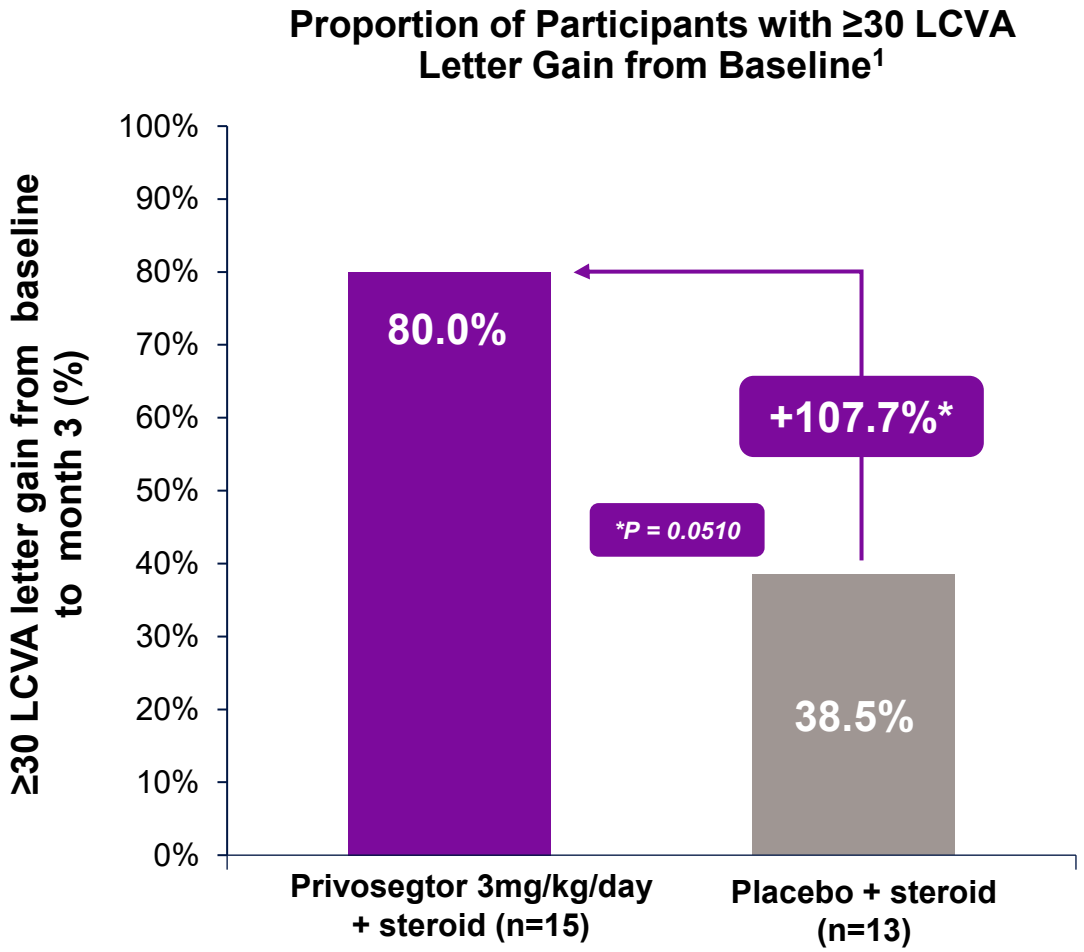
1. https://pubmed.ncbi.nlm.nih.gov/28206829/

ETDRS: Early Treatment Diabetic Retinopathy Study; LCVA: low contrast visual acuity

*Mixed Model for Repeated Measures (MMRM), Least-Squares Mean Change from Baseline: (2-sided nominal p-value), mITT population (affected eye).

More Patients Achieved ≥15 and ≥30 ETDRS LCVA Letter Improvement with Privosegtor 3 mg/kg/day vs Placebo at Month 3 (Post Hoc Analysis)

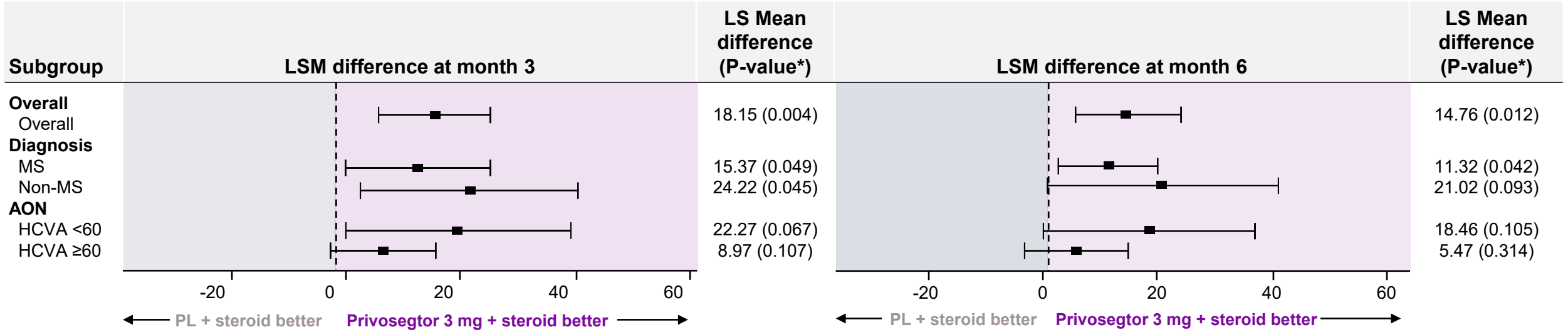




ETDRS (Early Treatment Diabetic Retinopathy Study); LCVA, low contrast visual acuity; mITT population (affected eye); 2.5% ETDRS LCVA in the affected eye
*Relative Difference
1. Data presented at ARVO 2026

Privosegtor Arm Showed a Robust LCVA Improvement Across all Subgroups and Maintained through Month 6

LCVA letters subgroup analyses of Privosegtor 3mg + steroid vs placebo + steroid

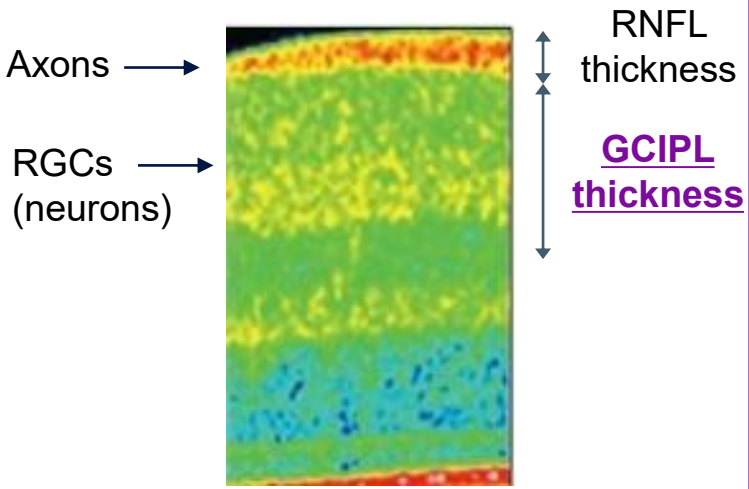


*2-sided nominal p-value based on LSM difference.
AON, acute optic neuritis; HCVA, high-contrast visual activity; LCVA, low-contrast visual acuity; LSM, least square mean; MS, multiple sclerosis; PL, placebo

Functional Improvement Correlated with Significant Preservation of Neurons in the Retina (RGCs)

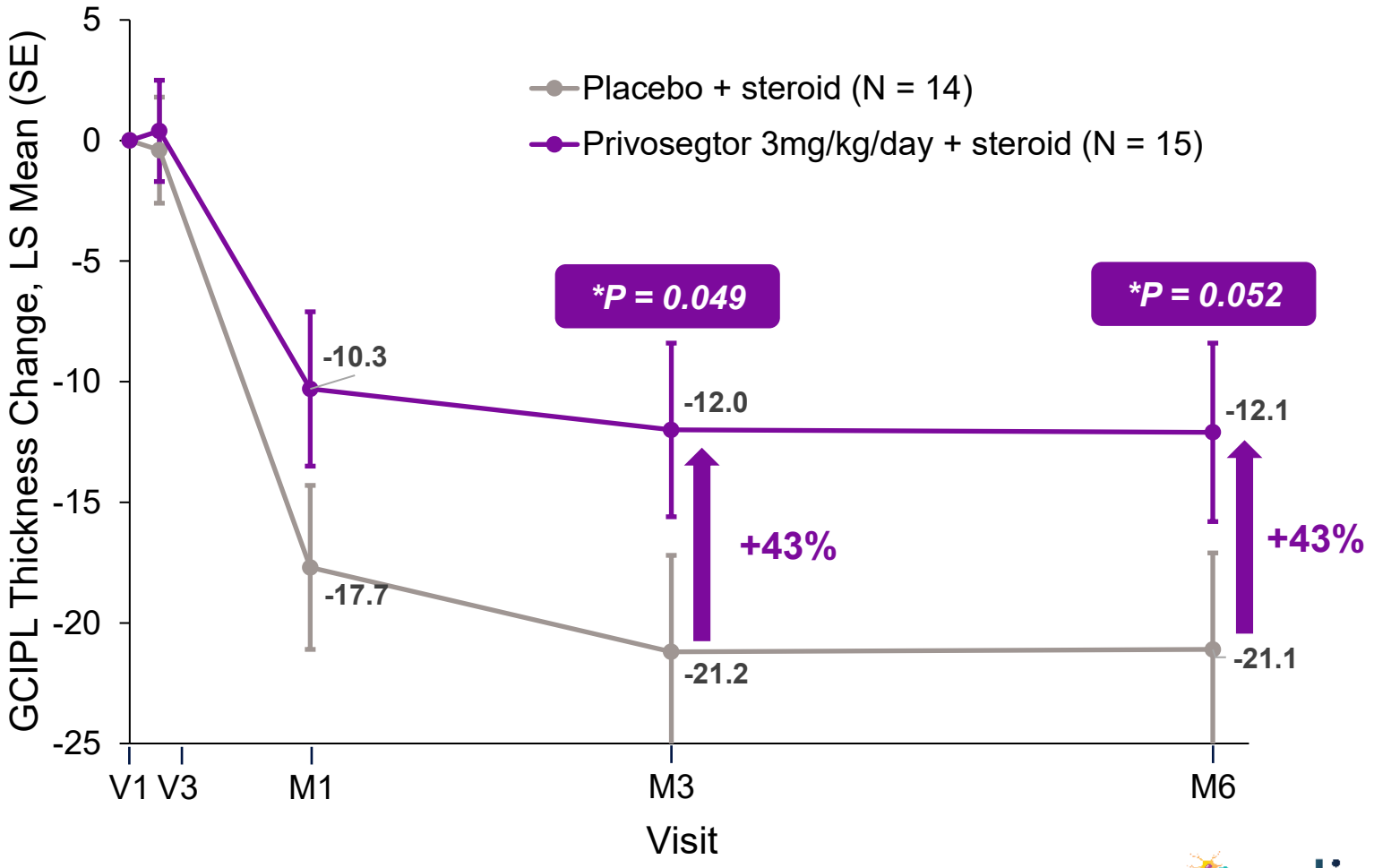
ANATOMY

GCIPL and RNFL: layers of the retina measured by OCT to monitor nerve damage



Decrease in GCIPL predicts poor LCVA, VF and CVA[1]

GCIPL Thickness in the Affected Eye: MMRM, LS Mean Change From Baseline, mITT



1. Gabilondo et al. Ann Neurol. 2015 Mar;77(3):517-28.

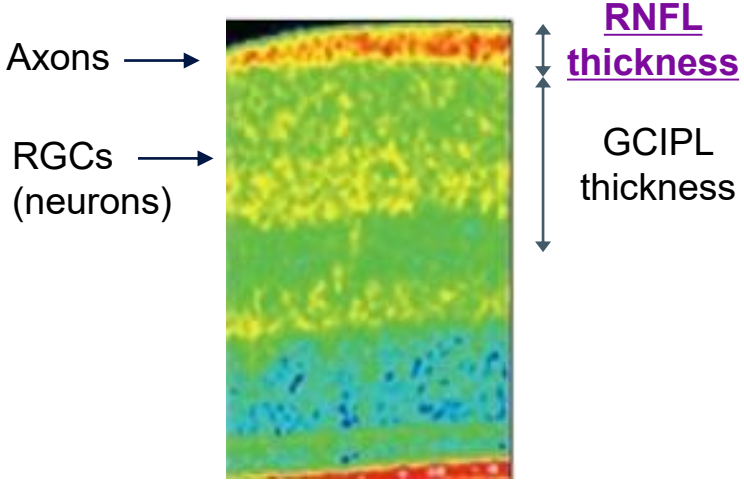
GCIPL; ganglion cell plus inner plexiform layer. RNFL: retinal nerve fiber layer. OCT: optical coherence tomography. LCVA: low-contrast visual acuity, VF: visual field, CVA: color visual acuity.

*Mixed Model for Repeated Measures (MMRM); Least-Squares Mean Change from Baseline: (1-sided directional nominal p-value), mITT population (affected eye)

Functional Improvement Correlated also with Significant Preservation of Axons (RNFL Thickness)

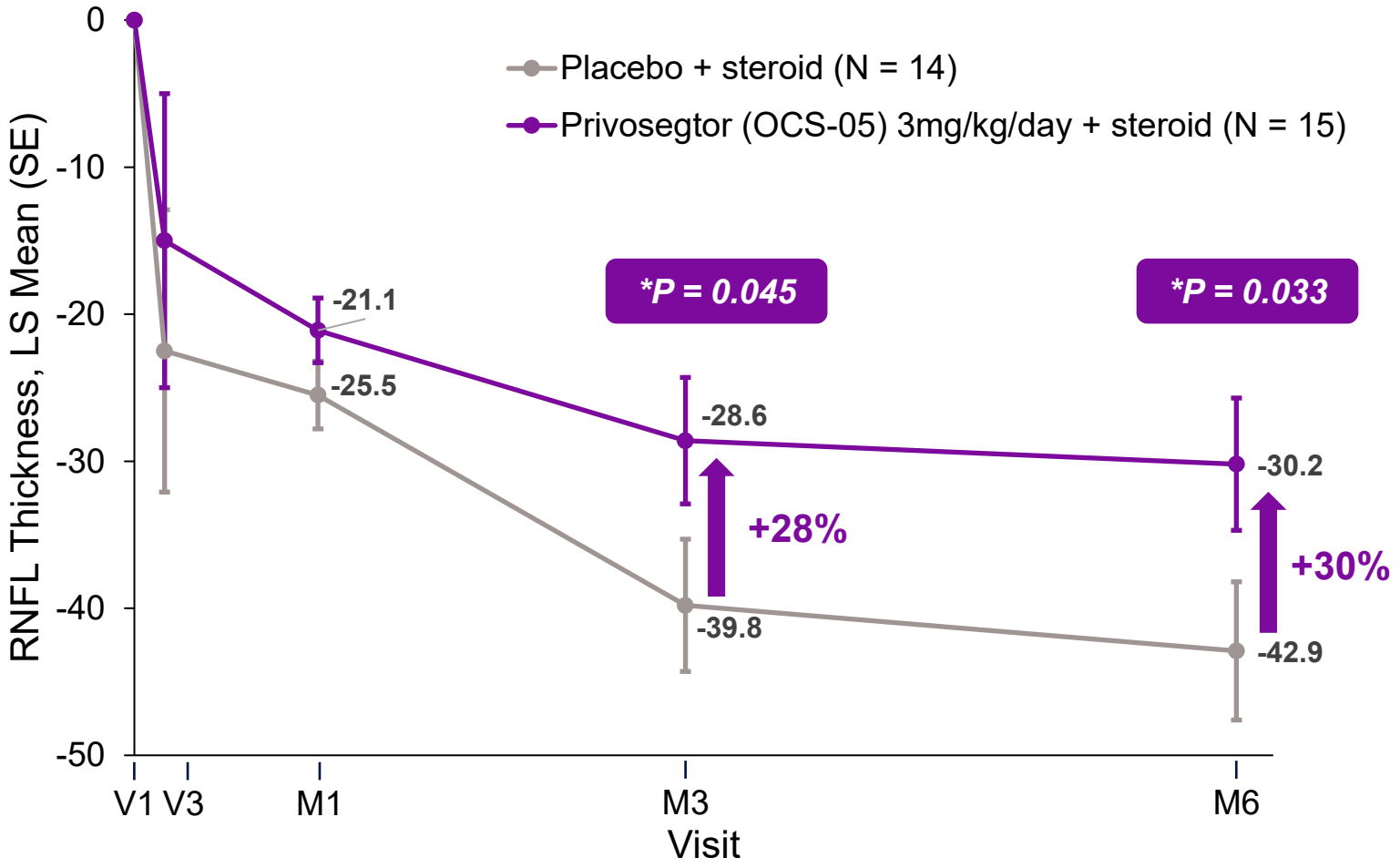
ANATOMY

GCIPL and RNFL: layers of the retina measured by OCT to monitor nerve damage



RNFL indicates damage or loss of retinal ganglion cell axons

RNFL Thickness in the Affected Eye: MMRM, LS Mean Change from Baseline, mITT



GCIPL; ganglion cell plus inner plexiform layer. RNFL: retinal nerve fiber layer. OCT: optical coherence tomography.
*Mixed Model for Repeated Measures (MMRM); Least-Squares Mean Change from Baseline: (1-sided directional nominal p-value), mITT population (affected eye).

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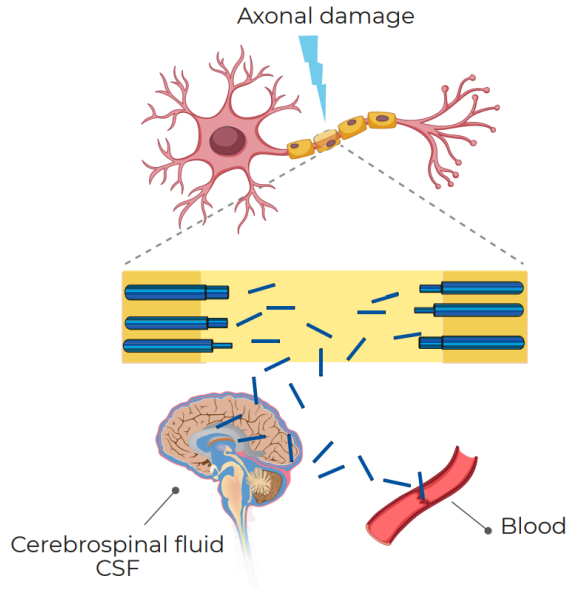
Neuroprotective Benefits with Privosegtor Also Observed in Biological Sign of Neuronal and Axonal Death

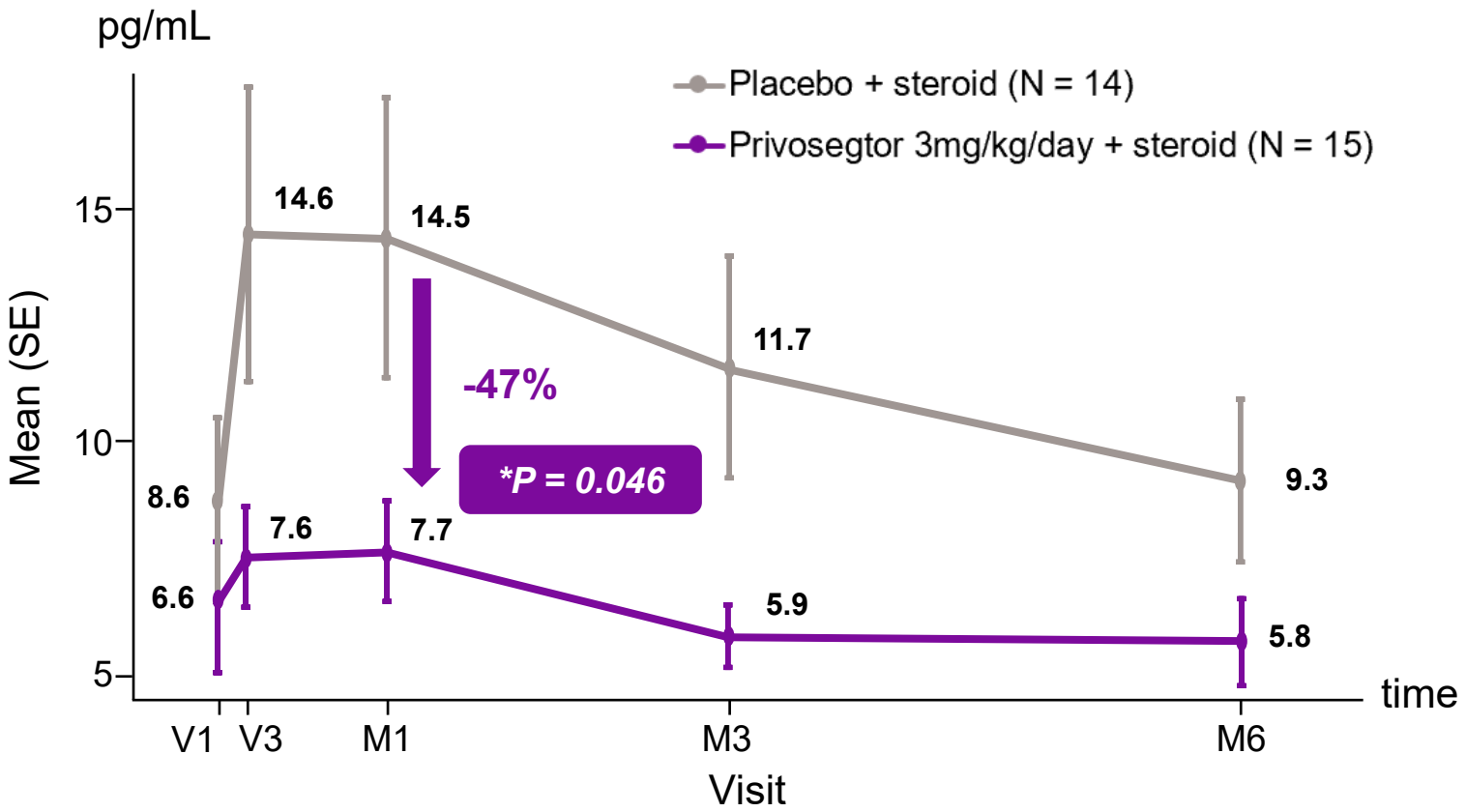
BIOLOGY

Neurofilaments: Released into CSF and blood as a result of axonal and neuronal death[1]



Biomarker for regulatory approval of neurodegenerative disease[2]

Mean Neurofilaments Over Time, mITT



*Mixed Model for Repeated Measures (MMRM); Least-Squares Mean Change from Baseline: (2-sided nominal p-value), mITT population (affected eye).

1. Gafson AR and al. Neurofilaments: neurobiological foundations for biomarker applications. Brain. 2020 Jul 1;143(7):1975-1998.
2. Stern S and al. Trends in clinical studies evaluating neurofilament light chain as a biomarker. Biomark Med. 2025 Sep;19(17):813-823.

Safety Profile Reported in ACUITY Phase 2 Trial Showed No AEs Leading to Drug Withdrawal or Study Discontinuation

- No AEs leading to drug withdrawal or study discontinuation
- No drug-related serious adverse events (SAEs)

Event, n (%)	Privosegtor + steroid			Placebo + steroid (N = 14)
	2 mg/kg/day (N = 4)	3 mg/kg/day (N = 15)	Pooled (N = 19)	
At least one TEAE *Related to study treatment*	4 (100.0%) *4 (100.0%)*	12 (80.0%) *6 (40.0%)*	16 (84.2%) *10 (52.6%)*	14 (100.0%) *6 (42.9%)*
At least one grade ≥2 TEAE *Related to study drug*	2 (50.0%) *0*	9 (60.0%) *2 (13.3%)*	11 (57.9%) *2 (10.5%)*	6 (42.9%) *0*
At least one serious TEAE *Related to study drug*	0 *0*	1 (6.7%) *0*	1 (5.3%) *0*	1 (7.1%) *0*
At least one SAE leading to death	0	0	0	0
At least one TEAE leading to a dose reduction	0	0	0	0
At least one TEAE leading to a dose interruption	0	0	0	0
At least one TEAE leading to a drug withdrawn	0	0	0	0
At least one TEAE leading to premature discontinuation of the study	0	0	0	0

SAE, serious adverse event; TEAE, treatment emergent adverse event.
Two (2) unrelated SAEs: Hospitalization due to MS relapse (Privosegtor (OCS-05 + steroid) and due to myelitis (placebo + steroid)

PIONEER-1 Registrational Trial Aligned with FDA under Special Protocol Assessment

Privosegtor has potential to be first neuroprotective medicine in Optic Neuritis

Study Design

- Randomized, multicenter, double-masked, placebo-controlled study
- Primary analysis at Month 3; Safety follow-up through Month 12
- N: ~210, 2:1 randomization
- Privosegtor 3mg/kg/day + SoC (IV Methylprednisolone) vs. placebo + SoC

Key Efficacy Endpoints

Primary:

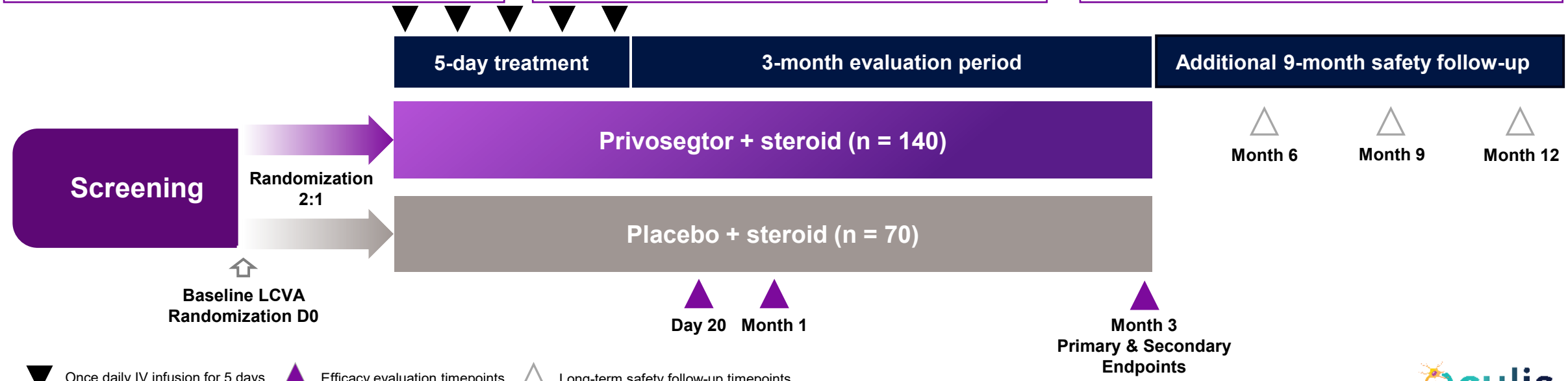
- Proportion (%) of ≥15 letter gainers at Month 3

Secondary:

- Proportion (%) of ≥30 letter gainers at Month 3
- LCVA mean change from baseline at Month 3
- GCIPL mean change from baseline at Month 3
- sNfL mean change from baseline at Month 3

Study Population

- All comers: including MS and non-MS optic neuritis
- Privosegtor treatment within 12 days from the first onset of symptoms



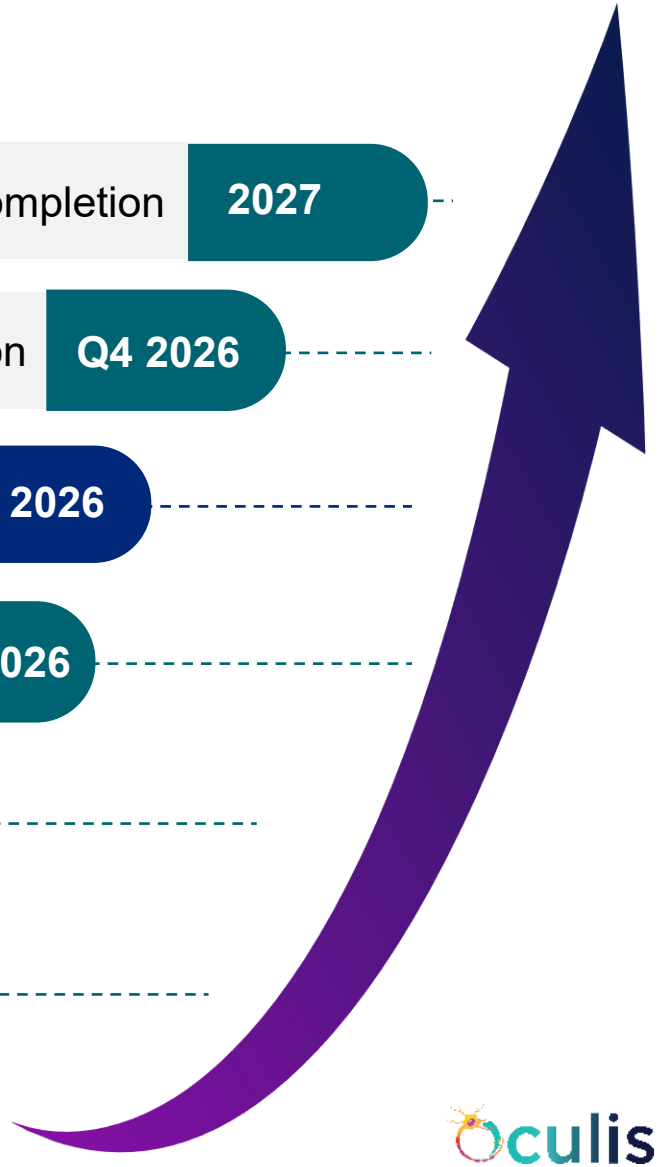
IV: intravenous, GCIPL: ganglion cell-inner plexiform layer, LCVA: low-contrast visual acuity, MS: multiple sclerosis, PL: placebo, SoC: standard of care, sNfL: Serum Neurofilaments

Privosegtor Driving Multiple Anticipated Milestones in Next 18 Months

PIONEER

Three Registrational trials in optic neuritis and NAION

- **PIONEER-1 in optic neuritis** enrollment completion — **2027**
- **PIONEER-2** in ON and **PIONEER-3** in NAION trials initiation — **Q4 2026**
- **Acute MS relapses IND submission** planned — **Q4 2026**
- ✓ **First Patient Treated in PIONEER-1** in ON — **Aug. 2026**
- ✓ **Special Protocol Assessment** agreement with FDA — **Q2 2026**
- ✓ **Breakthrough Therapy** and **PRIME designations** granted — **Q1 2026**



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Privosegtor

Acute Multiple Sclerosis (MS) Relapses

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Optic Neuritis a Solid PoC in MS and Beyond

Phase 2 ACUITY trial established Privosegtor's neuroprotective potential in acute optic neuritis, *the same biology underpins every acute MS relapse*

PoC Phase 2 ACUITY Trial

Acute optic neuritis · Privosegtor 3 mg/kg IV daily × 5 days, added to IV methylprednisolone

VISION
+18 letters
Mean 2.5% LCVA gain vs placebo, Month 3

RGC PRESERVATION
−43%
Less GCIPL thinning vs placebo (9.2 µm, M3 & M6)

AXONAL PROTECTION
↓ sNfL
Lower blood neurofilament — marker of axonal injury

Same Biology

Optic neuritis is a typical MS relapse — a well-established pathophysiological model generalizable to other relapse types

LCVA	GCIPL on OCT	Plasma NfL
→ T25FW / 6MW	→ MRI lesion vol. & atrophy	→ Plasma NfL

Same Development Path, Aligned with FDA

- **FDA pre-IND (Division of Neurology):** existing IND data may be cross-referenced — no additional preclinical work required
- **Agency supported** the development strategy, a 3-month primary endpoint, and the same 3 mg/kg × 5-day regimen as PIONEER-1
- **IND submission planned Q4 2026,** followed by a Phase 2/3 program covering optic neuritis (PIONEER-2) and ambulatory relapses

~170,000 U.S. acute MS relapses per year — no approved neuroprotective therapy

Anchor indication: optic neuritis is registrational today — PIONEER-1 under an FDA Special Protocol Assessment · Breakthrough Therapy (FDA) · PRIME (EMA) · Orphan Drug (FDA & EMA)

Privosegtor is investigational and not approved in any country; its safety and efficacy have not been established. Relapse volume derived as ARR 0.2 × ~850,000 U.S. MS prevalence (Hauser SL, et al. N Engl J Med. 2020;383:546-557, Bigaut K, et al. J Neurol. 2022 Jun;269(6):3295-3300).
Source: Oculis Phase 2 ACUITY data; Oculis press release, 3 August 2026.

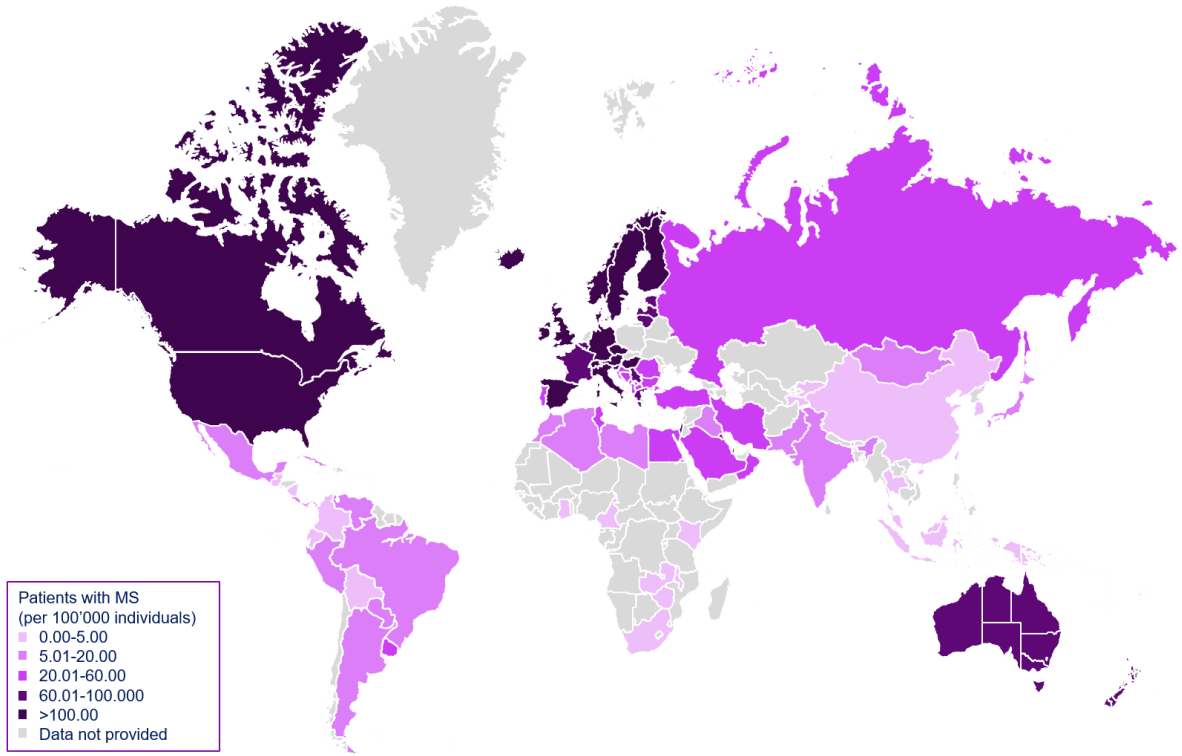
MS is a Major Cause of Neurological Disability in Young Adults and Affects ~850,000 Individuals in the United States Alone[1-5]

MS at a Glance

MS is an autoimmune-mediated neurodegenerative disease of the central nervous system characterized by inflammatory demyelination with axonal transection[3]

- Every **5 minutes** someone is diagnosed with MS in the world[1]
- Typically presents in **young adults**, with a mean age at diagnosis of 32 years[2]
- Can lead to **physical disability**, **cognitive impairment**, and decreased quality of life[3]

Worldwide Prevalence of MS[2]

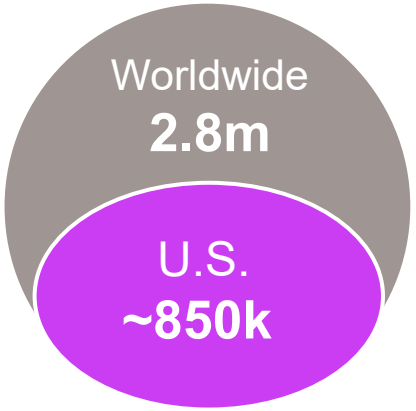


MS: Multiple Sclerosis

1. Walton C, et al. Multi Scler. 2020; 26(14):1816-1821; 2. Multiple Sclerosis International Federation – Atlas of MS – 3rd Edition; 3. McGinley MP, et al. JAMA. 2021; 325:765–779. 4. Wallin et al., 2019, The prevalence of MS in the United States: A population-based estimate using health claims data – PubMed. 5. MS National Society

MS is One of the Most Common Causes of Neurological Disability Affecting Young Adults[1]

High prevalence of multiple sclerosis worldwide[2-5]

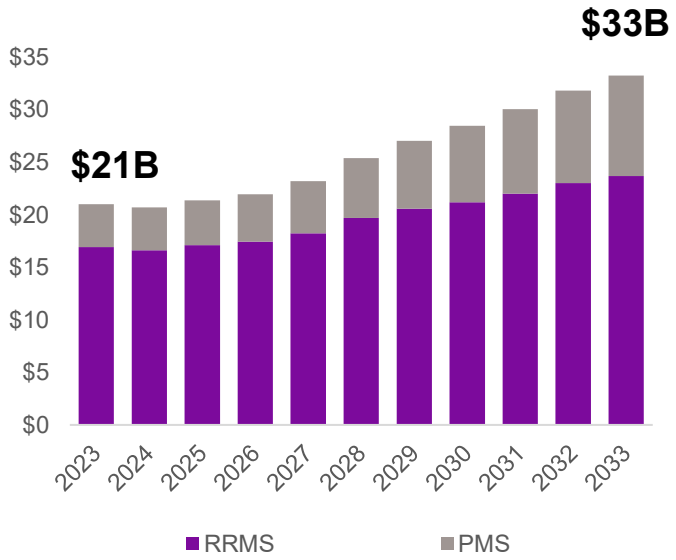


Characterized by relapses or attacks on the central nervous system (CNS), leading to inflammation, demyelination, and neurodegeneration

Main phenotypes:
Relapsing-Remitting and Progressive Multiple Sclerosis (RRMS or PMS)

Large and growing market driven by immuno-modulators

Estimated Multiple Sclerosis Market Size in G7 countries in billions (US, EU5 and JP)[6]



Existing therapies reduce relapse frequency however relapse-associated worsening remains

Affects function in cognitive, emotional, motor, sensory, or visual areas

Driven by a person's immune system attacking their brain, spinal cord and optic nerves

1. Jakimovski D. et Al. Multiple Sclerosis, The Lancet, 2023; 403, 183-202, 2. MS National Society, used for WW prevalence of 2.8m, and combined with references 2 to 4 to calculate an avg prevalence in US Prevalence of Multiple Sclerosis | National MS Society, 3. Wallin et al., 2019, The prevalence of MS in the United States: A population-based estimate using health claims data – PubMed, 4. McGinley et al., 2021, Diagnosis and Treatment of Multiple Sclerosis: A Review – PubMed, 5. Multiple Sclerosis International Federation – Atlas of MS – 3rd Edition, 6. MS Disease Landscape and Forecast Report 2024. Clarivate

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Whether Inaugural or Recurrent, Acute MS Relapses Share the Same Inflammatory Demyelinating Pathophysiology

Acute MS Relapses[1-3]

Characterized by focal or multifocal **inflammatory demyelination leading to neuronal damage**, lasting >24h. Clinical presentation is heterogeneous and depends on the location of demyelinating lesions.

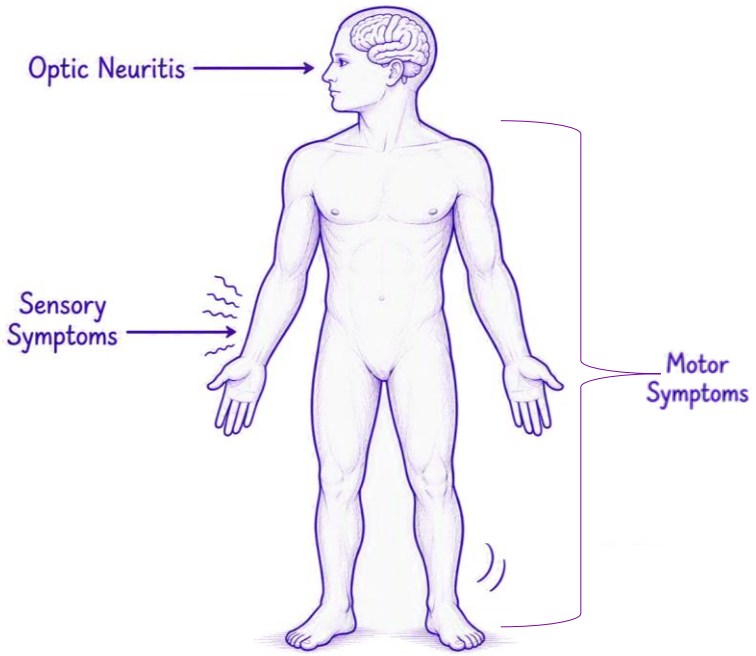
Inaugural Relapse[1]

- **Optic Neuritis ~25%**
- **Motor Symptoms ~35%**
- **Sensory Symptoms ~40%**

Recurrent Relapse

Relapses tend to recur within **previously affected** neurological **domains**[3]

Over the course of MS, **optic neuritis** affects **~70%** of patients[4] and **motor symptoms** affect **nearly all**[1]



MS: Multiple Sclerosis; CIS: Clinically Isolated Syndrome; RRMS: Relapsing-Remitting MS; SPMS: Secondary Progressive MS; PPMS: Primary Progressive MS; DMT: Disease-Modifying Treatments

1. Filippi M, et al. Nat Rev Dis Primers. 2018; 4:43;
2. McGinley MP, et al. JAMA. 2021; 325:765–779;
3. Kalincik T, et al. Multiple Sclerosis Journal. 2014;20(11):1511-1522; Bennett JL. Continuum (Minneap Minn). 2019; 25(5):1236-1264
4. Bennett JL. Continuum (Minneap Minn). 2019; 25(5):1236-1264

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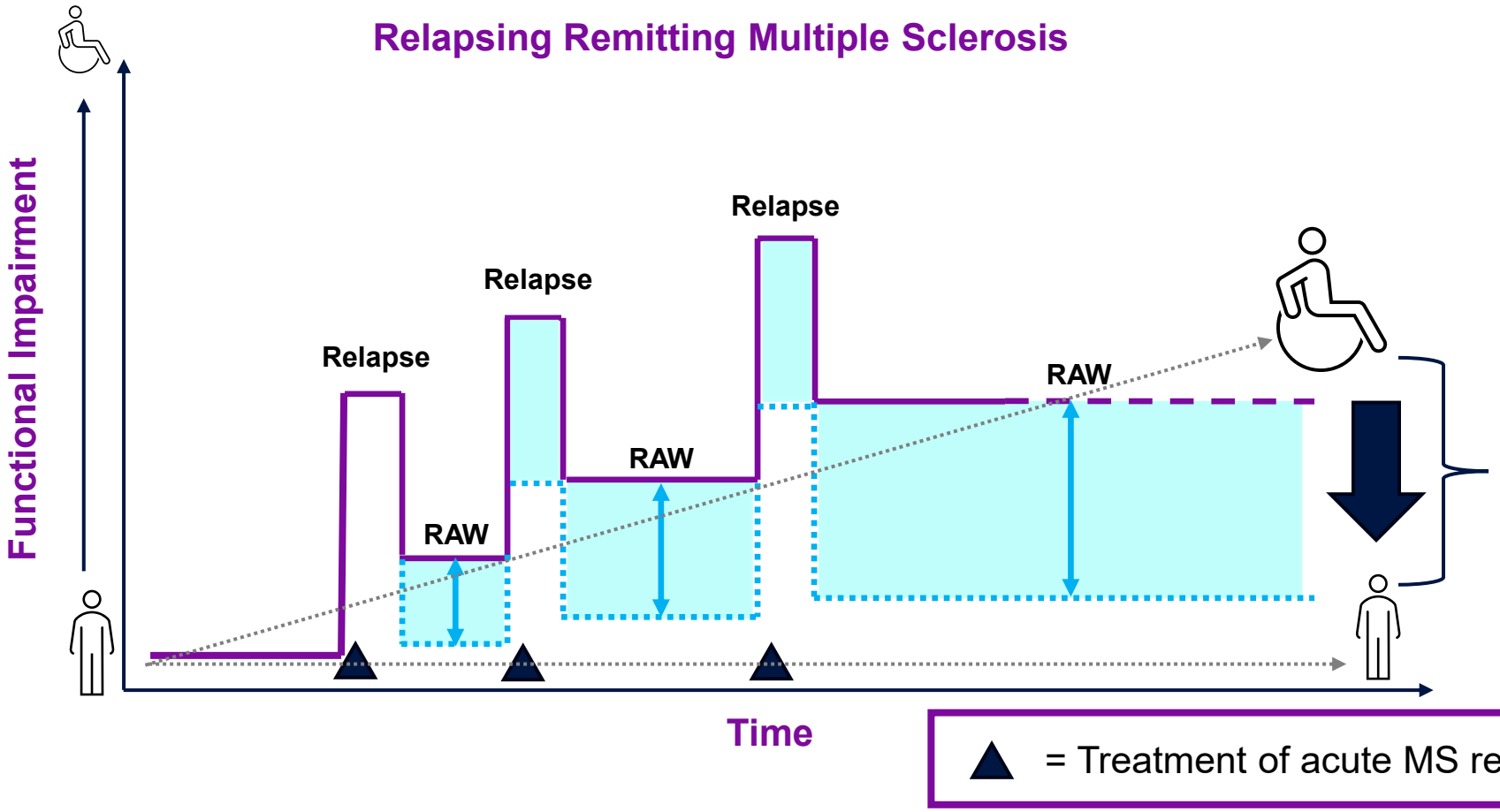
By Treating Relapses, We May Be Able to Reduce RAW



- **RAW:** Relapse-Associated Worsening
- **Effective neuroprotection** in relapses could translate to reduced accumulation of disability due to relapses over time [1-4]

Neuroprotection potential benefits

▲ = Treatment of acute MS relapses with Privosegtor

1. Susin-Calle S, Martinez-Rodriguez JE, Munteis E, Villoslada P. Ongoing phase 2 agents for multiple sclerosis: could we break the phase 3 trial deadlock? Expert Opinion Investig Drugs 2025;34:217-229.
2. Lublin FD, Haring DA, Ganjgahi H, et al. How patients with multiple sclerosis acquire disability. Brain 2022;145:3147-3161.
3. Montobbio N, Cordioli C, Signori A, Bovis F, Capra R, Sormani MP. Relapse-Associated and Relapse-Independent Contribution to Overall Expanded Disability Status Scale Progression in Multiple Sclerosis Patients Diagnosed in Different Eras. Ann Neurol 2024;97:95-103.
4. Zanghi A, Galgani S, Bellantonio P, et al. Relapse-associated worsening in a real-life multiple sclerosis cohort: the role of age and pyramidal phenotype. Eur J Neurol 2023;30:2736-2744

Conclusion

Oculis Pipeline Development Strategic Evolution

Advance

Privosegtor neuroprotection in optic neuritis

Licaminlimab precision medicine in DED

Expand

Privosegtor in neuro-ophthalmology and beyond

- **NAION**
- **MS relapses**

Explore

Potential for Privosegtor in multiple additional neuro-axonal diseases

~$8B

>$25B

~$50B

NEURO-OPHTHALMOLOGY & BEYOND

DED: dry eye disease, NAION: Non-arteritic Anterior Ischemic Optic Neuropathy MS: Multiple Sclerosis.
1. DED Disease and Landscape report 2020 - 2024 market value estimate for G7, 2. MS Disease and Landscape report October 2024 – 2024 market value estimate for G7, 3. Acute optic neuropathy market estimated to 7B in US based on rare disease price analogues and annual incidence of ON and NAION, 4. Optic nerve disorders, Transparency Market Research, 5. Global Market Insights, March 2024
https://www.gminsights.com/industry-analysis/neuroprotection-market

Upcoming Value-Driving Milestones Across Our Targeted Registrational Programs

	Candidate	Phase 1	Phase 2	Phase 3	Upcoming Anticipated Value Catalysts
OPHTHALMOLOGY	**Licaminlimab** **Genotype-based development program**	Dry Eye Disease			**PREDICT-1** TLR in **around year-end 2026**
NEURO-OPHTHALMOLOGY	**Privosegtor** **Neuroprotective candidate**	Optic Neuritis — **Breakthrough Therapy and PRIME designations, SPA**			**PIONEER-1** enrollment completion in **2027** **PIONEER-2** trial initiation in **Q4 2026**
		NAION			**PIONEER-3** trial initiation in **Q4 2026**
		Acute MS relapses			Cross-reference optic neuritis IND for **new IND submission** in MS relapses in **Q4 2026**

IND: investigational new drug, MS: multiple sclerosis, NAION: non-arteritic anterior ischemic optic neuropathy, PRIME: priority medicine, SPA: special protocol assessment, TLR: topline results.
Privosegtor and Licaminlimab are investigational drugs, their safety or efficacy has not been established, and they have not received regulatory approval for commercial use in any country.

Oculis

Thank you

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Visionary Innovation