



Neflamapimod: A Targeted Therapy For Age-Related Disorders of the Brain

October 2026

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CervoMed at a glance

A clinical-stage company developing treatments for age-related brain disorders



LEAD ASSET

Neflamapimod

Oral small molecule

Targets inflammatory signaling pathways in the brain that leads to synaptic dysfunction

LEAD INDICATION

Dementia with Lewy Bodies (DLB)

- High unmet medical need with no approved therapies
- Mechanism aligns with underlying disease process
- Phase 2 clinical trial data demonstrate positive clinical, blood biomarker, and MRI effects
- Alignment with global regulators on a registration path

OTHER INDICATIONS

PHASE 2a DATA (4Q26-1Q27)

nfvPPA

Non-fluent variant primary progressive aphasia



PHASE 2a DATA (4Q26)

RAS

Recovery after ischemic stroke



FIRST PATIENT DOSED (YE26)

EXPERTS-ALS

Phase 2a amyotrophic lateral sclerosis trial; fully-funded by UK government and charities

Consistent clinical, biomarker, imaging, and safety data support advancing neflamapimod as a potential therapy for DLB

CLINICAL ACTIVITY AND BIOLOGY



Exposure-dependent, clinically meaningful slowing of dementia progression¹

Greater benefit at target drug levels, sustained through 32 weeks of treatment



Biomarkers and imaging further support biological activity

Basal forebrain MRI effects and plasma GFAP changes support on-target drug effects



Optimal patient population identified

Clinical activity consistently greatest in patients without evidence of AD co-pathology

SAFETY AND TOLERABILITY

>550
participants

Well-defined, favorable safety profile

Participants dosed across clinical trials to date, with treatment up to 48 weeks

<2%
of patients

Low rate of treatment discontinuation

Reversible liver enzyme elevation, the main risk with p38 MAPK inhibitors, led to discontinuation in <2% of patients in trials to date

~2x
brain : plasma

Selectivity and brain penetration underpin safety profile

Highly p38 α -selective; estimated brain levels ~2x plasma favor CNS target engagement over peripheral inhibition

DLB changes how people think, move, and live



Dementia with Lewy bodies (DLB)

A rapidly progressing brain disease associated with abnormal deposits of the protein alpha-synuclein

More than memory loss

Fluctuating attention, visual hallucinations, movement difficulties, and disrupted sleep

High unmet need

3rd most common chronic neurodegenerative disease; no FDA-approved medications specifically for DLB

Neflamapimod inhibits p38 α , a key driver of inflammation-induced cholinergic neuron injury in DLB

Disease process in DLB

Inflammation impairs and injures cholinergic neurons that support cognition and movement

Mechanism of action

Oral, brain-penetrant drug that blocks p38 α activation and potently inhibits IL-1 β signaling

Supporting evidence

Animal models and early human studies demonstrated effects on inflammatory signaling and basal forebrain biology

Therapeutic goals: reverse synaptic dysfunction, restore function in vulnerable brain circuits, improve neuron health, and slow disease progression

CDR-SB: a well-established scale for evaluating the severity and progression of dementia

- In DLB, CDR-SB is superior to cognitive testing (e.g., MMSE) to assess the disease^{1,2,3}:
 - ✓ Better captures clinical progression
 - ✓ Less subject to cognitive fluctuations
- Mean group difference ≥ 0.5 -point to placebo considered clinically meaningful^{4,5,6}

Aligned with FDA and global regulatory authorities on change in CDR-SB as the primary endpoint in potential registration trial

Clinical Dementia Rating Sum of Boxes (CDR-SB, Total Score = 0-18)

Cognitive Domains:

- Memory (0-3)
- Orientation (0-3)
- Judgement and Reasoning (0-3)

Functional Domains:

- Community Affairs (0-3)
- Home & Hobbies (0-3)
- Personal Care (0-3)

Phase 2 studies in DLB support and define the development strategy

Phase 2a: clinical signal

Significant benefit ($p=0.023$; mITT analysis) on CDR-SB, major secondary endpoint

Primary cognitive endpoint not met overall but met in higher dose cohort

Identified **plasma pTau181** as a strategy to select optimal patient population

AscenD-LB Phase 2a: 91 participants, 16 weeks

Phase 2b: exposure and mechanistic insights

Inadequate plasma drug exposure (formulation issue) led to randomized primary endpoint not being met

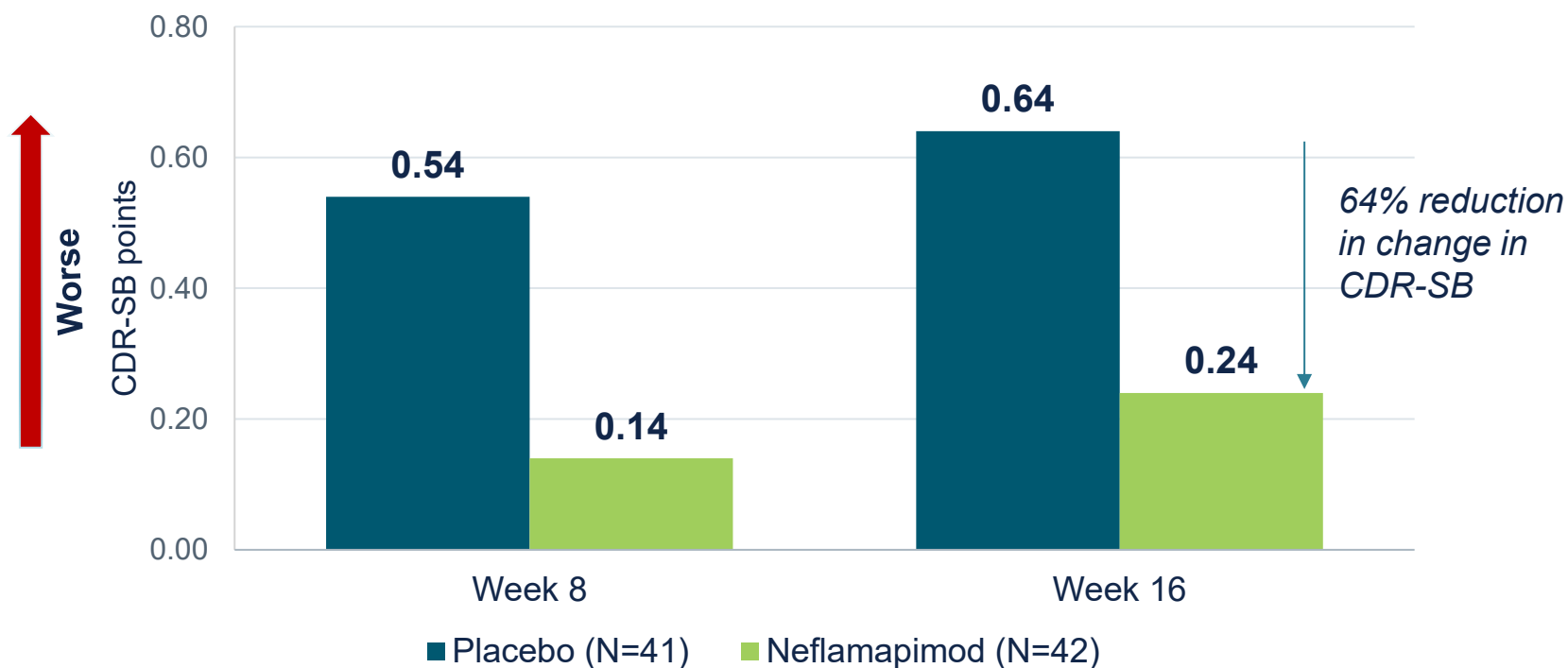
Exploratory analyses linked **higher exposure and lower pTau181** with **positive outcomes on CDR-SB**

Blood biomarker and imaging results support **clinical effects**

RewinD-LB Phase 2b: 159 participants, 16 weeks + 32-week extension

Phase 2a provided an early clinical signal

Mean change in CDR-SB



Significant improvement
compared to placebo

p=0.023

Mean neflamapimod – placebo
difference = -0.45, 95% CI -0.83,
Linear Mixed Effects Model

**A signal to confirm
in a pivotal trial**

Validated blood test identified optimal patient population

Low Plasma pTau181 – “Pure” DLB

Less evidence of Alzheimer’s disease (AD) co-pathology and associated neuronal loss

Neflamapimod mechanism of action matches the underlying pathology in “pure” DLB patient population

Elevated Plasma pTau181 – Mixed DLB-AD

Greater likelihood of AD co-pathology and more extensive neurodegeneration, limiting potential to respond to treatment

2-fold Increase

In CDR-SB treatment effect size vs. placebo in low plasma pTau181 (< 21 pg/mL) cohort in Phase 2a study

Phase 2b fell short of the expected drug exposure

4.0 ng/mL

Reported mean trough exposure during the randomized phase

Later work linked lower exposure to multiple crystal forms in original capsules

5.0 ng/mL

Expected mean trough exposure benchmark¹

Exceeded by batch of capsules introduced in extension (DP Batch B)

Primary endpoint

No significant overall CDR-SB benefit versus placebo

A new consistent and controlled process to manufacture only the stable crystal form of neflamapimod has been implemented and a 50mg oral tablet formulation developed

Higher plasma drug exposures associated with slowing of dementia progression in randomized phase

16-week mean change in CDR-SB

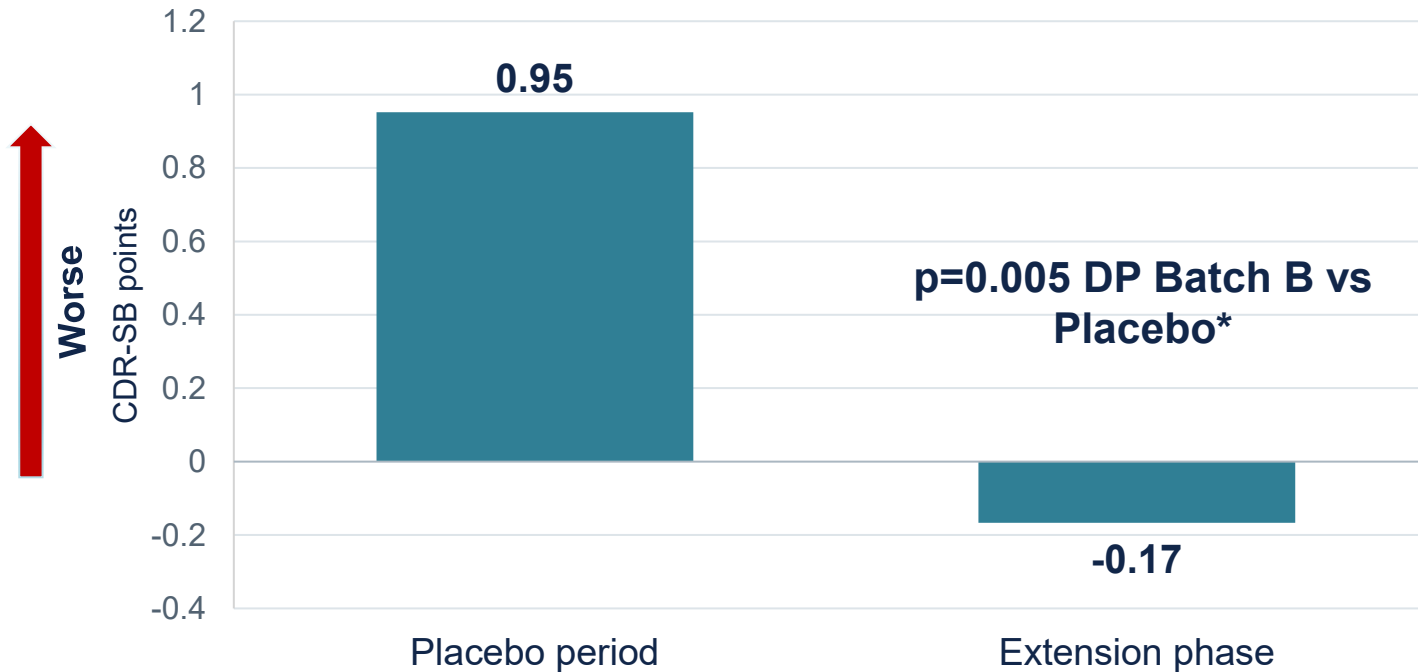
Group (pTau181 <21 pg/mL)	Patients (N)	Mean change (SE)
Placebo	45	+0.72 (0.29)
Drug exposure below median	17	+0.41 (0.21)
Drug exposure above median	18	-0.09 (0.39)

Negative change means less worsening/improvement
Median trough levels: 3.64 ng/mL

Trend test p=0.038

The extension phase strengthened the exposure hypothesis

Same patients (within subject analysis, N=21)
pTau181 <21 pg/mL subgroup



Neflamapimod-placebo
difference

-1.12

Consistent with the
hypothesis that adequate
exposure matters

Each period lasted 16 weeks

Negative change in CDR-SB indicates improvement

DP Batch B: batch of capsules introduced in extension that achieved target plasma drug concentration

Biomarker and imaging findings in Phase 2b trial support clinical data

Blood biomarker: GFAP

Participants with Low Likelihood of AD Pathology
(plasma pTau181 <21 pg/mL)

Median difference between neflamapimod
and placebo:

-23.1 pg/mL

~50% of disease-specific elevation

Median difference on Batch B versus the earlier placebo
period N=48, p=0.016

MRI: right basal forebrain volume

16-week change (%volume)	Mean
Neflamapimod (N=8)	+3.5%
Placebo (N=10)	-4.2%

Randomized comparison, p=0.0282

Clinical measures, biomarker results, and imaging data all point in same direction

New oral tablet delivers more consistent exposure

Stable crystal form

Controlled manufacturing of a single crystal form expected to **address drug-product variability** and **stability issues**

Twice-daily dosing

Single dose human pharmacokinetic data support moving **to 100 mg twice daily**, using 50 mg tablets

Higher trough levels

Projected to achieve **~33% higher trough drug level** at steady-state than the prior 40 mg three-times-daily regimen

FDA removal of prior exposure limit allows for greater dosing flexibility

Aligned with regulators on registration trial design

~300 participants

DLB with low likelihood of
Alzheimer's co-pathology
pTau181 <21 pg/mL

32 weeks

Randomized neflamapimod
versus placebo
Primary endpoint: CDR-SB

>60 sites

Global trial planning
Regulatory alignment with
FDA, MHRA, EMA, and
PMDA on key design
aspects

- Single Phase 3 clinical trial
- 100mg BID dosing regimen with new, stable crystal form; initial clinical batches manufactured and released
- >90% statistical power to achieve significant clinical effect

A targeted and unaddressed commercial market opportunity

>\$3B US Market Potential in “Pure” DLB

150,000–180,000 diagnosed DLB patients with
low likelihood of AD co-pathology in US

Similar numbers in Europe and Japan

- High unmet need
- Substantial strain on caregivers and the healthcare system
- Highly specific clinical diagnostic criteria (>90%)
- Personalized medicine approach
- Specialist indication
- No approved therapies

DLB leads a broader set of clinical opportunities

Program	Status / expected milestone
Dementia with Lewy bodies*	Registration trial planning; subject to funding
Recovery after ischemic stroke	Phase 2a clinical data: Q4 2026 (n=45)
Nonfluent primary progressive aphasia**	Biomarker / clinical readouts: Q4 2026–Q1 2027
ALS ¹	First patient dosing expected by year-end 2026 in EXPERTS-ALS