



Medicines for the Brain

Corporate Presentation

June 2026

NASDAQ: CRVO



Forward-Looking Statements

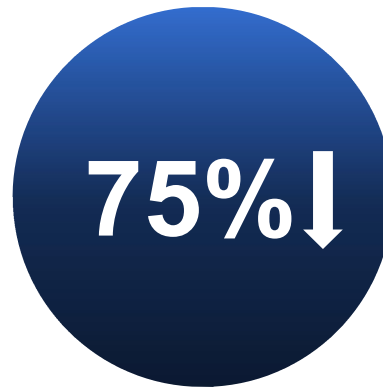
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Neflamapimod DP Batch B demonstrated proof-of-concept efficacy in Dementia with Lewy Body (DLB) patients

Drug product that achieved target exposure in Phase 2b study demonstrated clinically meaningful treatment effects compared to placebo



Reduction in **mean change in Clinical Dementia Rating Sum of Boxes test (CDR-SB)** (primary outcome measure)



Reduction in **risk of clinical progression** (≥ 1.5 -point increase in CDR-SB)



Reduction in disease-specific elevation of key **blood biomarker of neurodegeneration (plasma glial fibrillary acidic protein (GFAP))**

Magnitude of treatment effects 3X the reductions observed with approved anti-amyloid therapies in Alzheimer's disease¹

³ Planned Phase 3 patient population = DLB patients without AD co-pathology, defined by exclusion of patients with pTau181 ≥ 21 pg/mL at screening; data from within-subject comparison in patients who received placebo during randomized phase and DP Batch B during the extension. 1. 27% and 36% reduction in CDR-SB for Leqembi (Biogen / Eisai) and Kisunla (Lilly), respectively. No head-to-head comparisons have been completed.

CervoMed is well-positioned for a transformative year in 2026



First- and best-in-class potential in DLB

- ✓ Positive further analysis of Phase 2b data demonstrated proof-of-concept in DLB
- ✓ Engagement with FDA on Phase 3 study design



Robust pipeline in brain disorders

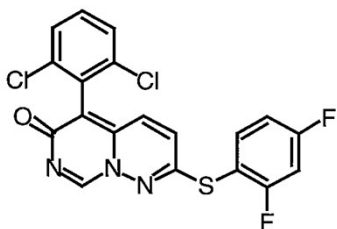
- Advancing clinical programs in nfvPPA and RAS
- Phase 2a data readouts expected for nfvPPA in mid-2026 and 1Q27 and for RAS in 2H26
- Dosing of the first patient with neflamapimod in the EXPERTS-ALS trial anticipated in 4Q26
- EIP200 provides flexibility to advance clinical development for these and other non-DLB indications



Primed for long-term success

- High unmet need in attractive commercial markets and an advanced development stage may create the potential for regional or global partnership opportunities.
- Experienced management team and board of directors

Neflamapimod is an oral, small molecule drug that selectively inhibits p38 α , a key driver of neuroinflammation and synaptic dysfunction in the basal forebrain



Preclinical proof-of-concept achieved

1

- Potent (<10nM IC₅₀), **highly selective inhibitor** of p38 α
- Blood-brain-barrier penetrant with brain to plasma ratio of ~2
- Reversed neurodegenerative process in basal forebrain in relevant animal disease models
- Improves both histological and behavioral outcomes in preclinical pharmacology studies

Target engagement demonstrated in clinical studies

2

- **Highly selective**
- Reduction in CSF levels of IL-8 (marker of IL-1b signaling)
- Reduction in cerebrospinal fluid (CSF) levels of phosphorylated tau and total tau
- Increase in volume of basal forebrain and its functional connectivity by MRI

Safety profile well defined

3

- Clinical safety data in >700 volunteers and patients, with up to 48 weeks treatment duration
- Chronic, repeat dose toxicology studies completed
- Human 40mg TID dose has **10-fold safety margin to NOAEL** in long-term toxicology studies

Clinical proof-of-concept achieved in DLB*

4

- Positive Phase 2a and 2b results
- **Phase 3-ready**



5 | *Neflamapimod is an investigational product currently in clinical development for the treatment of DLB, RAS, and nfvPPA.

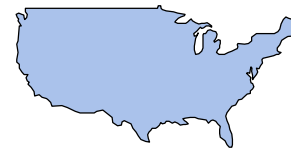
Pure DLB (those without AD co-pathology, ~50% of DLB overall) represents a substantial, untapped specialty market with high commercial potential

About Dementia with Lewy Bodies

- Progressive α -synucleinopathy characterized by widespread cortical and subcortical Lewy bodies
- Alzheimer's disease (AD) co-pathology is common – present in up to ~50% of all DLB patients¹
- Patients are generally managed by neurologists and the clinical diagnostic criteria are highly specific (>90%)²
- High unmet clinical need
 - Significant impact on quality of life and caregiver burden
 - Progresses more rapidly than AD, with average of 2 years from diagnosis to requiring nursing home care³
 - No approved therapies that target the underlying disease process in the US or EU



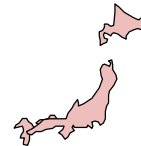
DLB becomes more common with age, accounting for an average of 12%¹ of dementia cases



- **~360,000 DLB patients w/o AD co-pathology**
- Dementia affects **~6 million Americans**
- Estimated DLB prevalence of **~720,000 cases**



- **~405,000 DLB patients w/o AD co-pathology**
- Dementia affects **~9 million Europeans**
- Estimated DLB prevalence of **~1,080,000 cases**



- **300,000 DLB patients w/o AD co-pathology**
- Dementia affects **~5 million Japanese**
- Estimated DLB prevalence of **~600,000 cases**

Despite highly specific clinical criteria, there is often a delay in diagnosis; highlighting a need for increased physician education

6 | 1. Diaz-Galvan P, et al. *Alzheimers Dement.* 2024;20(4):2485-2496. 2. McKeith IG et al. *Neurology.* 2017 Jul 4;89(1):88-100 3. Rongve A, et al. *Int J Geriatr Psychiatry.* 2014;29(4):392-398. 4. Average prevalence calculated from neuropathology studies. Sabbagh et al. 2023; Hogan et al. 2016; GBDF Collaborators 2022, Fang et al. 2025; Alzheimer's Disease International; MHLW estimates; Awata et al. 2020; Ikejima et al. 2009; NIH Report: Risk and future burden of dementia in the United States 2025.

AD co-pathology determines severity of disease and treatment response when targeting the basal forebrain cholinergic system

DLB Without AD Co-Pathology = Low Levels of Plasma pTau181



+



=



Basal Forebrain Cholinergic System

Diseased

Medial Temporal Lobe

No Atrophy

Reversible Function Deficient

Clinical Effect of Basal Forebrain-Directed Treatment

With **cholinergic dysfunction as the primary driver** of disease progression and the medial temporal lobe structurally intact, targeting the basal forebrain can slow clinical decline

VS

DLB With AD Co-Pathology = Elevated Levels of Plasma pTau181



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Basal Forebrain Cholinergic System

Diseased

Medial Temporal Lobe

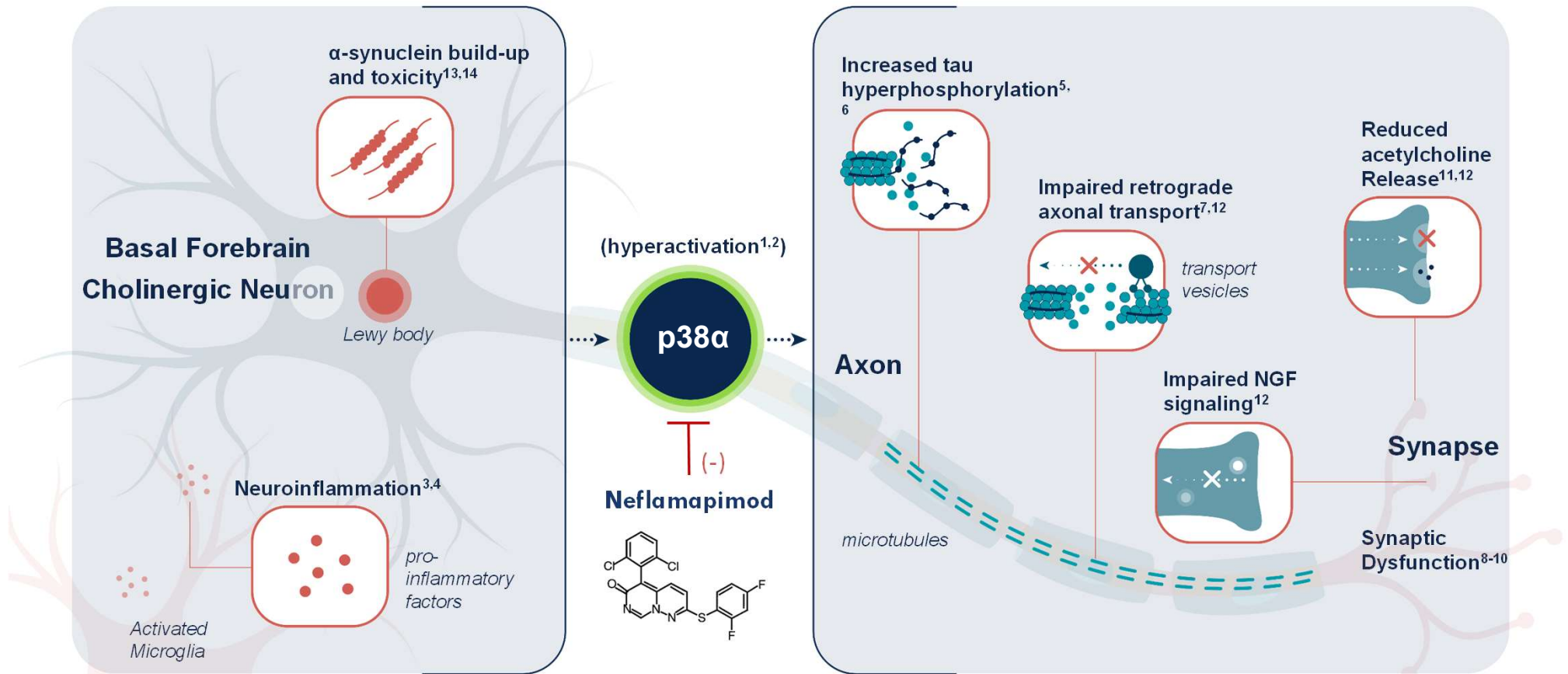
Atrophy

Irreversible Cell Loss

Clinical Effect of Basal Forebrain-Directed Treatment

More advanced disease with significant **hippocampal atrophy from AD co-pathology** which becomes the **primary driver of disease progression** and results in irreversible deficits

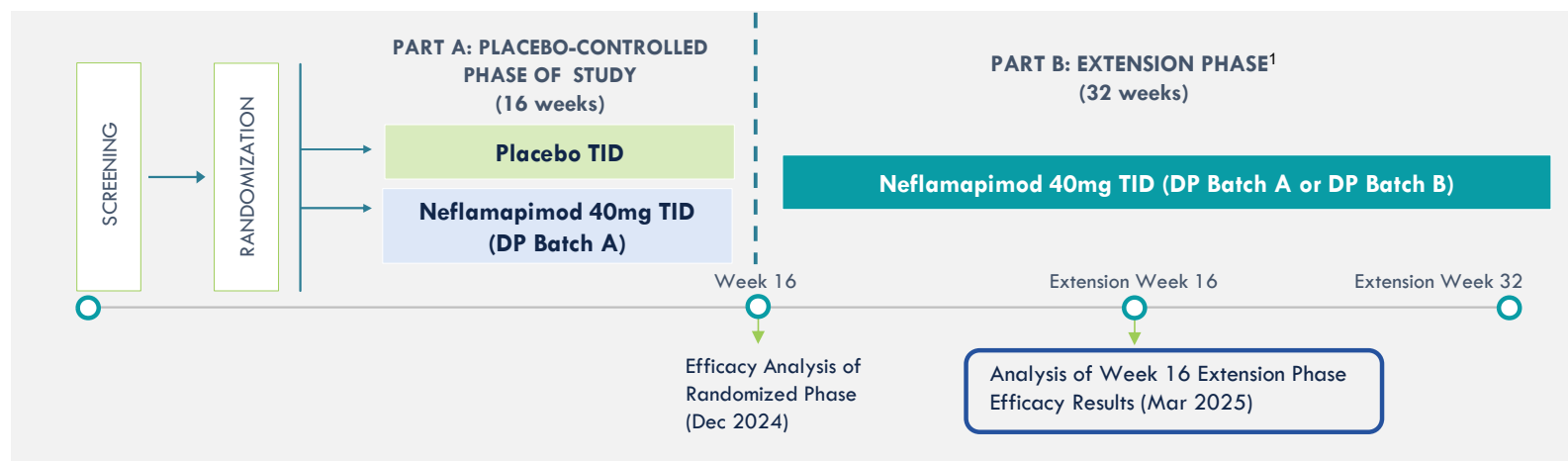
By blocking p38 α , neflamapimod aims to reverse synaptic dysfunction and improve neuron health



RewinD-LB open label Phase 2b study in DLB: Design and summary of results

PATIENTS:

- 159 patients with DLB by consensus clinical criteria
- CDR global score of 0.5 or 1.0 at baseline
- Baseline plasma pTau181 < 27.2 pg/mL (Simoa v2.1)



Drug Product (DP) Batch A Results:

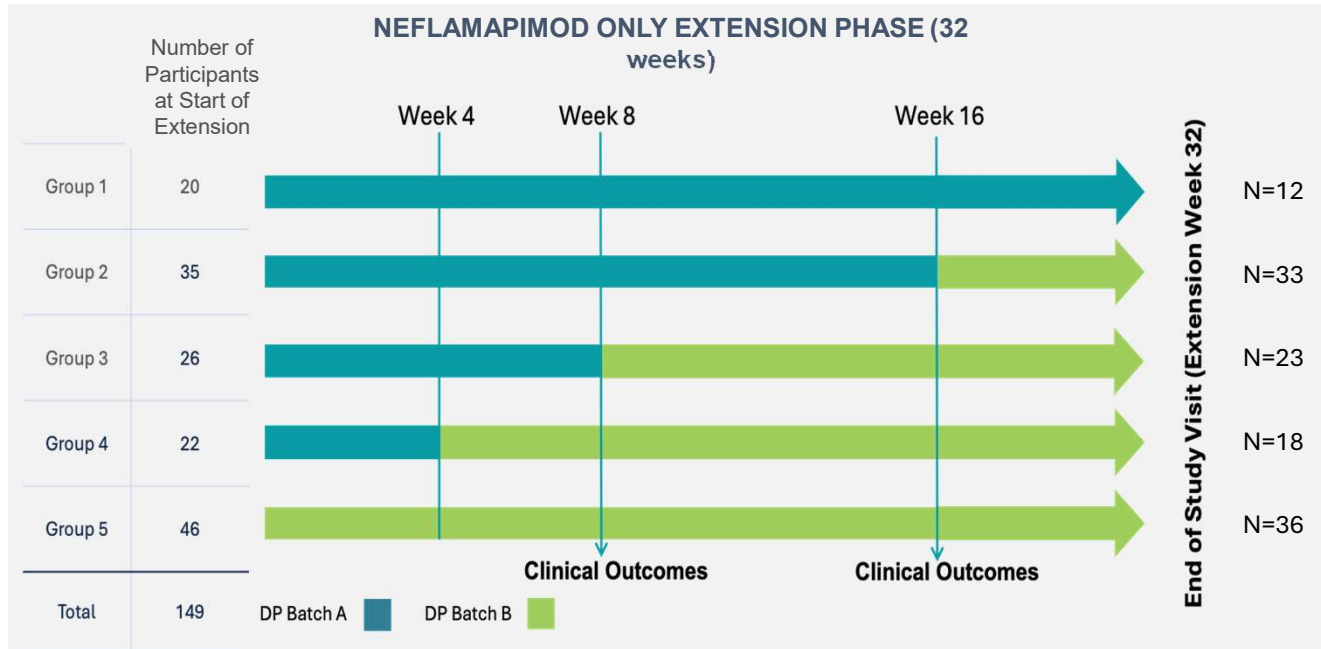
- Plasma drug exposure approaching that seen with 40mg BID, rather than that expected with 40mg TID
- No significant difference to placebo during Part A of study; acted as the control arm in Part B
- No significant effect on plasma glial fibrillary acidic protein (GFAP) in Part A, nor in Part B

DP Batch B Results:

- Achieved plasma drug exposure expected with 40mg TID dosing regimen
- Significant improvement compared to Batch A on mean change in CDR-SB (primary outcome measure) and on ADCS-CGIC during Part B
- Significant reduction in plasma GFAP, compared to baseline and compared to DP Batch A

DP Batch B achieved expected and targeted plasma drug concentrations

Pre-planned introduction of second batch enabled robust extension phase analyses



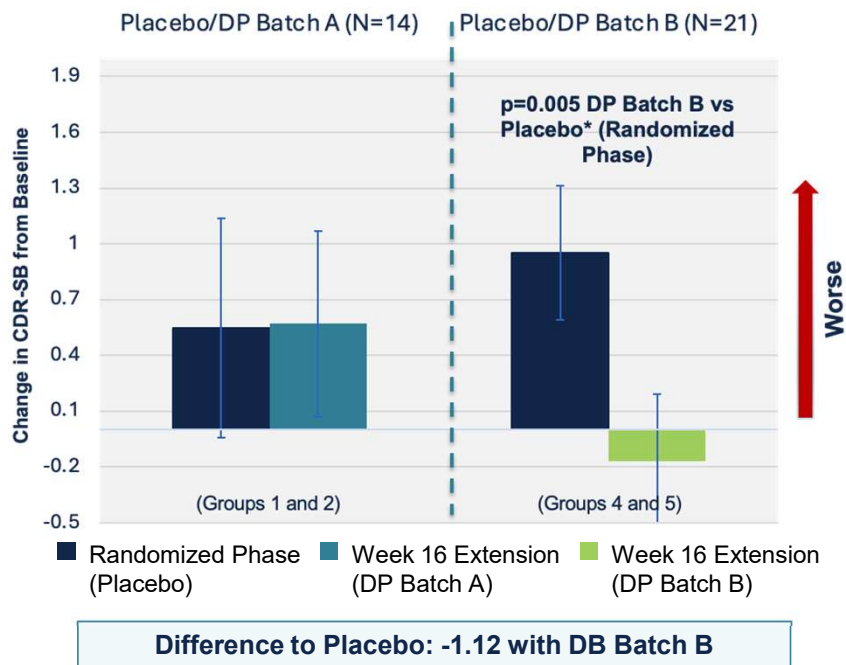
- Mean plasma drug trough concentration (12-hour post-dose, C_{12}) of 4.15ng/mL was less than expected mean 5.0ng/mL for 40mg TID, effectively underdosing participants, while mean C_{12} of DP Batch B was 5.17ng/mL
- Only 50% of DP Batch A (vs. 75% of DP Batch B) recipients achieved the individual patient target C_{12} of 4ng/mL

10 | While participants were aware that they were receiving neflamapimod in the Extension Phase (i.e., treatment was open label), neither participants nor site personnel were aware if they were receiving DP Batch A or DP Batch B.

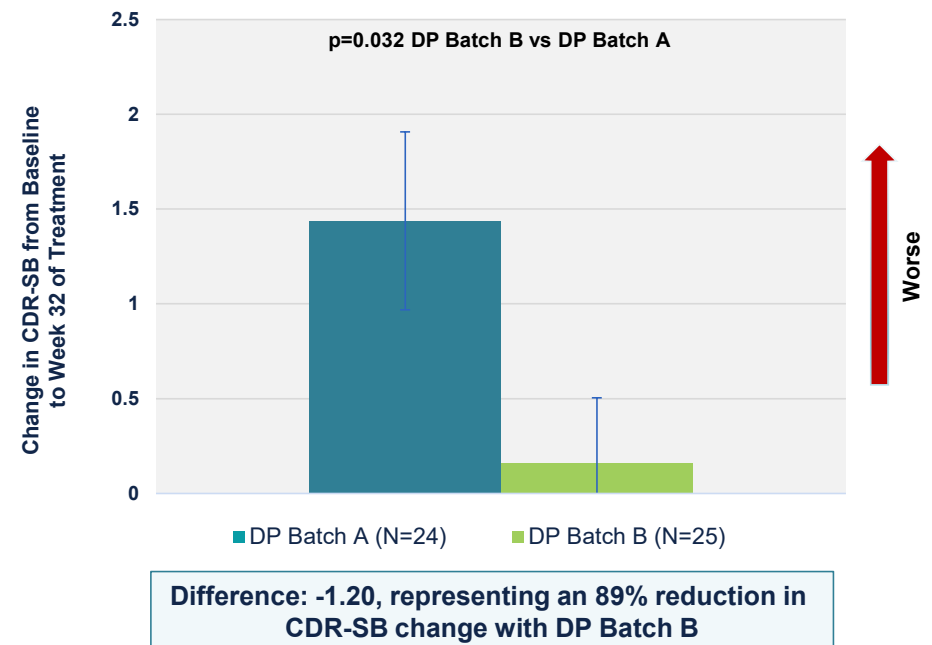
In Phase 2b, neflamapimod Batch B demonstrated statistically significant improvement in CDR-SB

Drug product that achieved targeted plasma drug concentrations demonstrated >1-point improvement in mean change in CDR-SB in patient population to be enrolled in planned Phase 3 trial

Mean Change in CDR-SB vs. Placebo After 16 Weeks



32 Week Data Highlights Durability

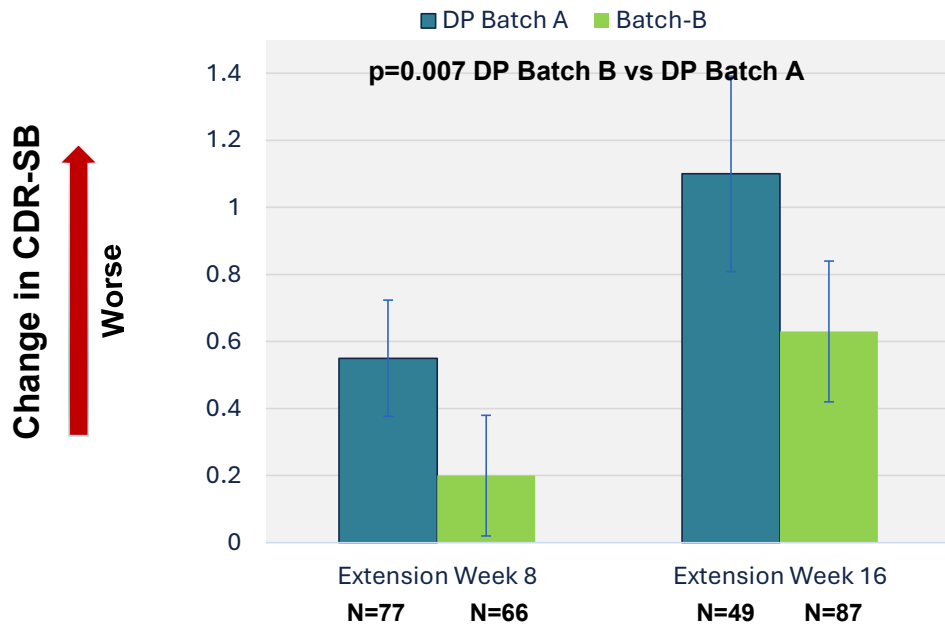


- DP Batch A – did not reach expected plasma drug concentration for 40mg TID dosing regimen
- DP Batch B – achieved expected plasma drug concentrations for 40mg TID dosing regimen

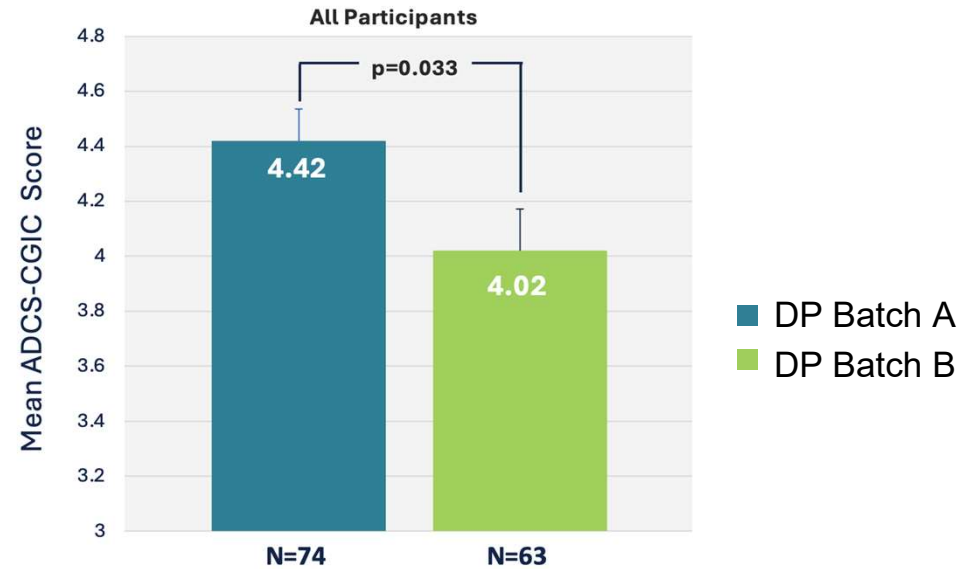
11 | DP Batch A data includes (i) all participants in Group 1 and (ii) participants in Group 2 who received DP Batch A during the placebo-controlled phase of the study, in each case, who completed 32-weeks of total treatment on DP Batch A. DP Batch B data includes participants in Group 5. 21 pg/mL pTau181 subset.

DP Batch B demonstrated statistically significant improvement in mean change in CDR-SB and ADCS-CGIC vs. Batch A

First 16 Weeks of the Extension Phase, Mean Change in CDR-SB

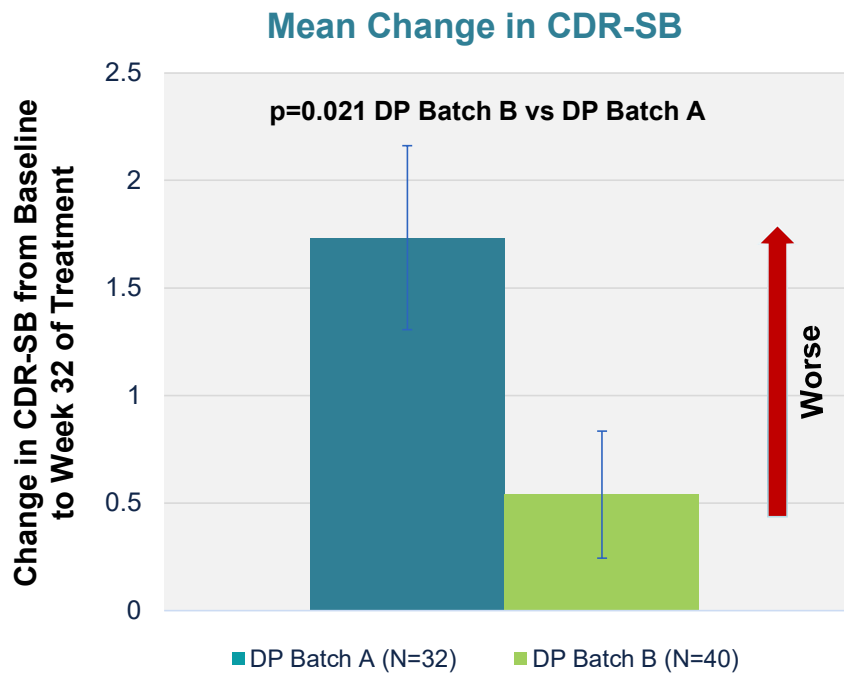


Extension Phase (Week 8), Mean ADCS-CGIC Score



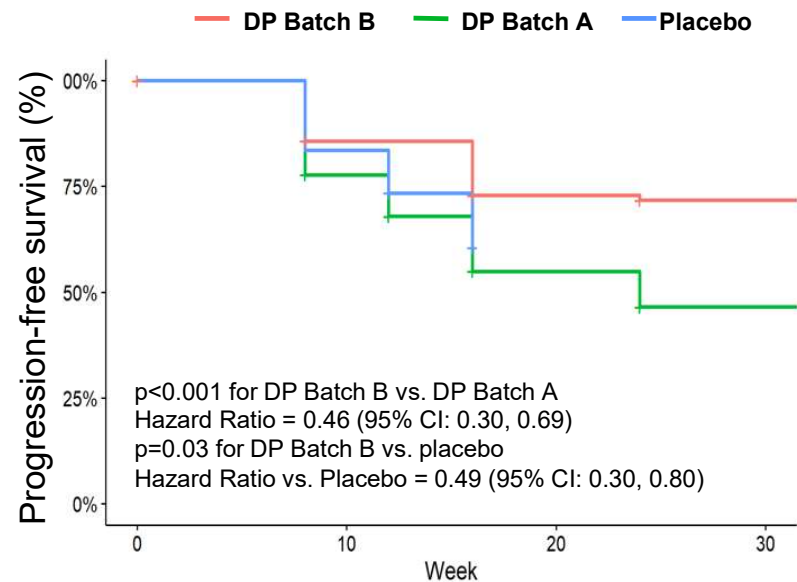
12 | MMRM with baseline CDR-SB, Sex, Age and MMSE as covariates. DP Batch A data includes participants in Groups 1, 2, and 3 at Week 8 and in Groups 1 and 2 at Week 16. DP Batch B data includes participants in Groups 4 and 5 at Week 8 and Groups 3, 4, and 5 at Week 16. ADCS = Alzheimer's Disease Cooperative Study.

Clinical effect on CDR-SB with neflamapimod DP Batch B was durable out to 32 weeks of treatment



Difference: -1.12, representing a 65% reduction in CDR-SB change when targeted plasma drug concentration achieved

Likelihood of Remaining Free of Progression (≥1.5 point increase in CDR-SB) over 32 Weeks

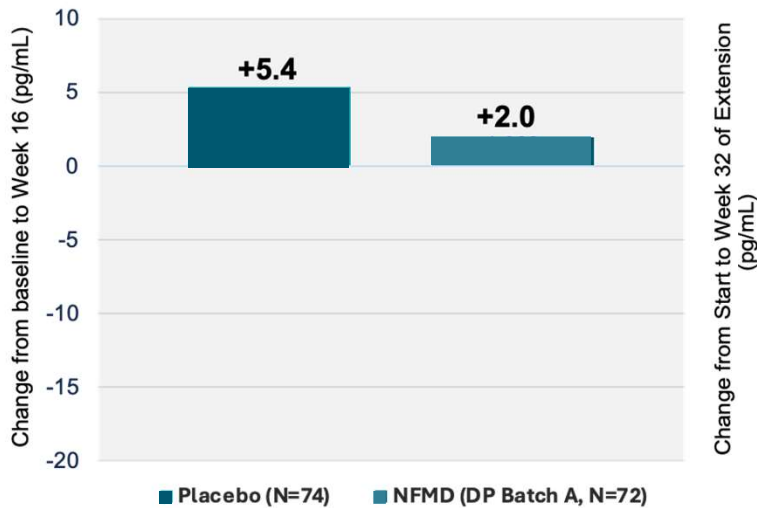


13

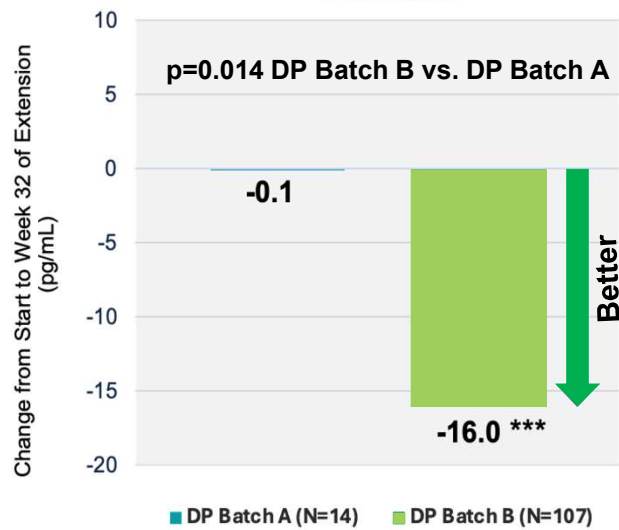
DP Batch A data includes (i) all participants in Group 1 and (ii) participants in Group 2 who received DP Batch A during the placebo-controlled phase of the study, in each case, who completed 32-weeks of total treatment on DP Batch A. DP Batch B data includes participants in Group 5. The mean change in the 12 Group 1 patients who completed the 32-week Extension was +1.5 points; the other 8 Group 1 patients discontinued early.

Neflamapimod DP Batch B achieved significant reduction in GFAP, a key biomarker of neurodegenerative disease activity

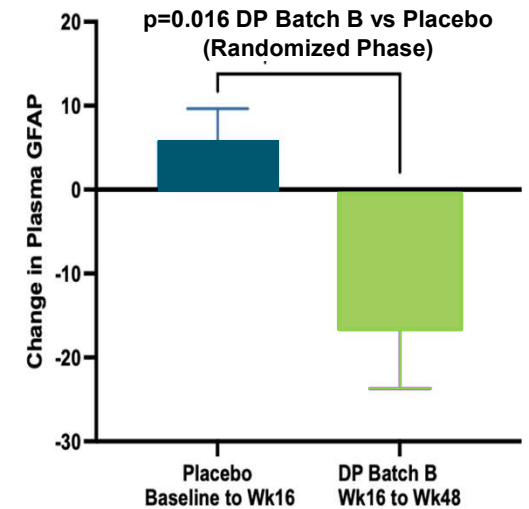
Median Change from Baseline to Week 16 During Placebo-Controlled Phase



Median Change from Start to Week 32 of Extension

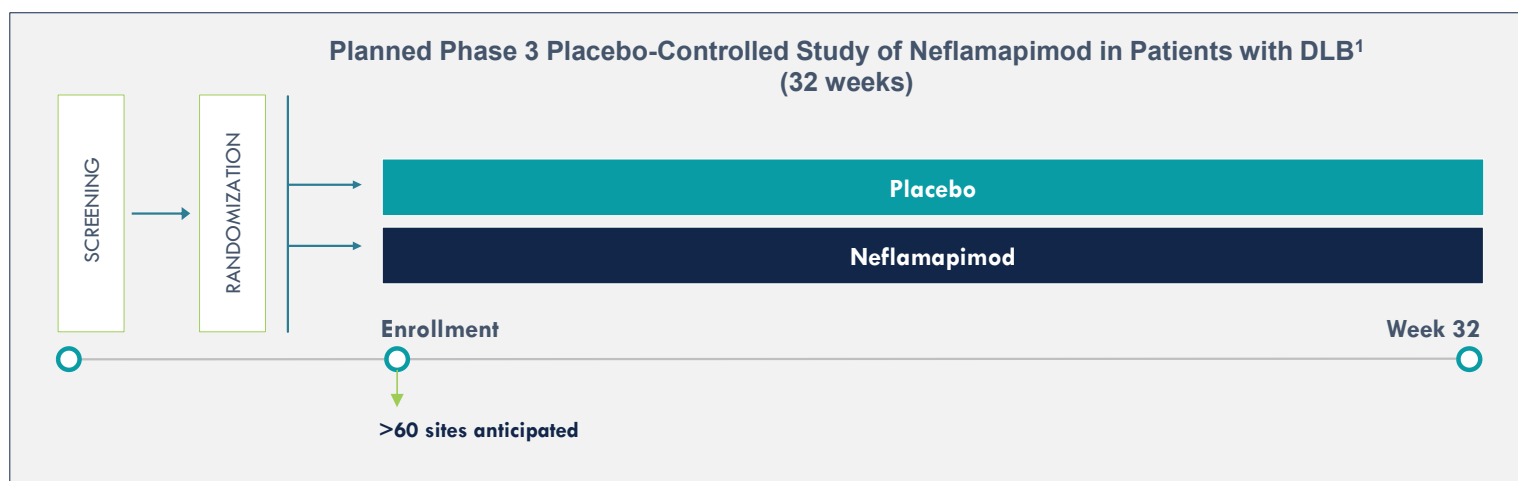


Within-Participant Comparison (N=48) of Effect on Plasma GFAP: DP Batch B vs. Placebo



Median difference -23.1 pg/mL (~50% of disease-specific elevation)

Engaged with the FDA on registrational trial design for DLB in November 2025



KEY PARAMETERS

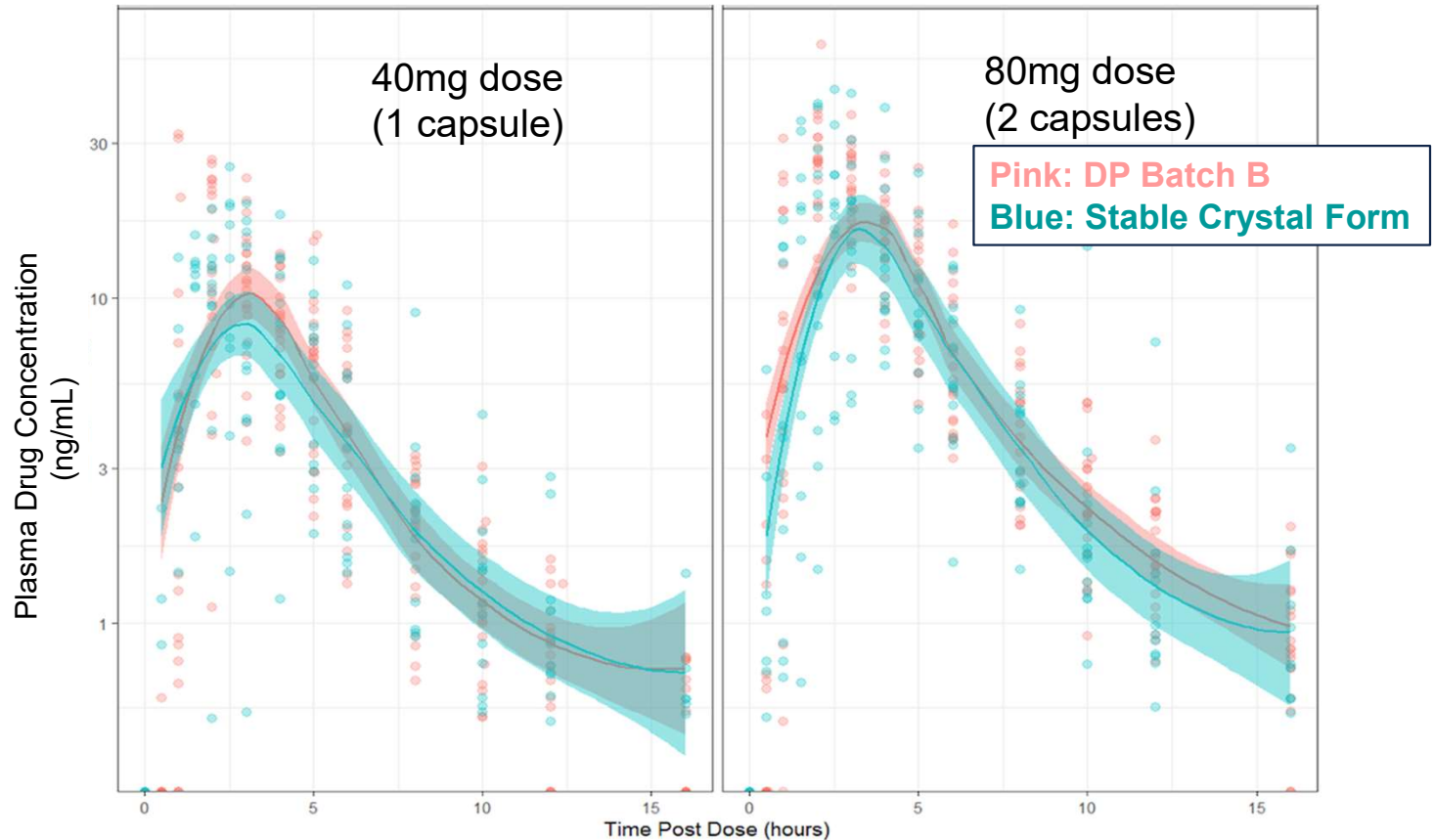
- DLB by consensus criteria, enriched for patients without AD co-pathology (**pTau181 < 21 pg/mL**)
- Primary endpoint: Change in CDR-SB
- Approximately 300 participants

- Single Phase 3 clinical trial of 32 weeks duration, with change in CDR-SB as primary endpoint
- Obtaining feedback from global regulatory authorities and completing activities to support improvements in drug product formulation
- 50mg TID dosing regimen with new, stable crystal form of neflamapimod

New, stable crystal form of neflamapimod supports target plasma exposure in planned Phase 3 trial

Mean (95% CI) and Individual Profiles After Single Doses of One or Two 40mg Capsules

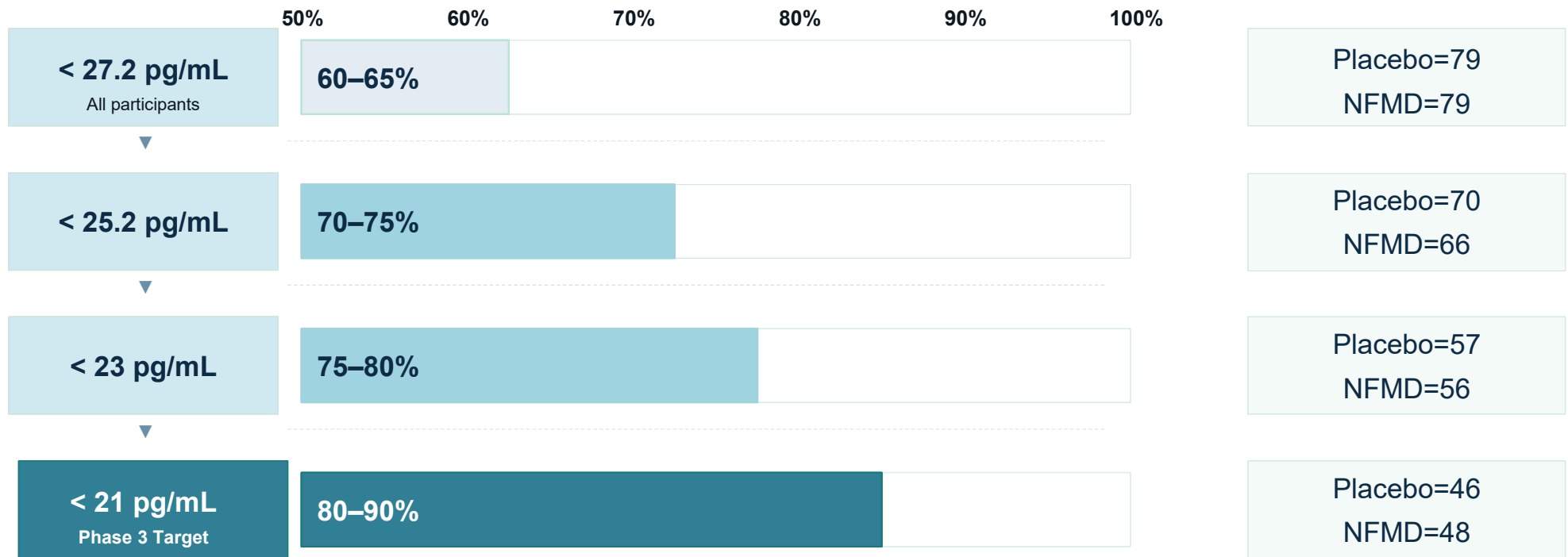
- PK profiles of DP Batch B and the new, stable crystal form of neflamapimod are largely overlapping
- Plan to increase dose to 50mg TID in future trials to maintain plasma drug concentration observed with DP Batch B
- With 50mg TID of the stable crystal form, expect ~80-90% of participants to achieve individual patient C_{trough} target of 4ng/mL



Lowering plasma pTau181 cut-off progressively enriches for patients without AD co-pathology

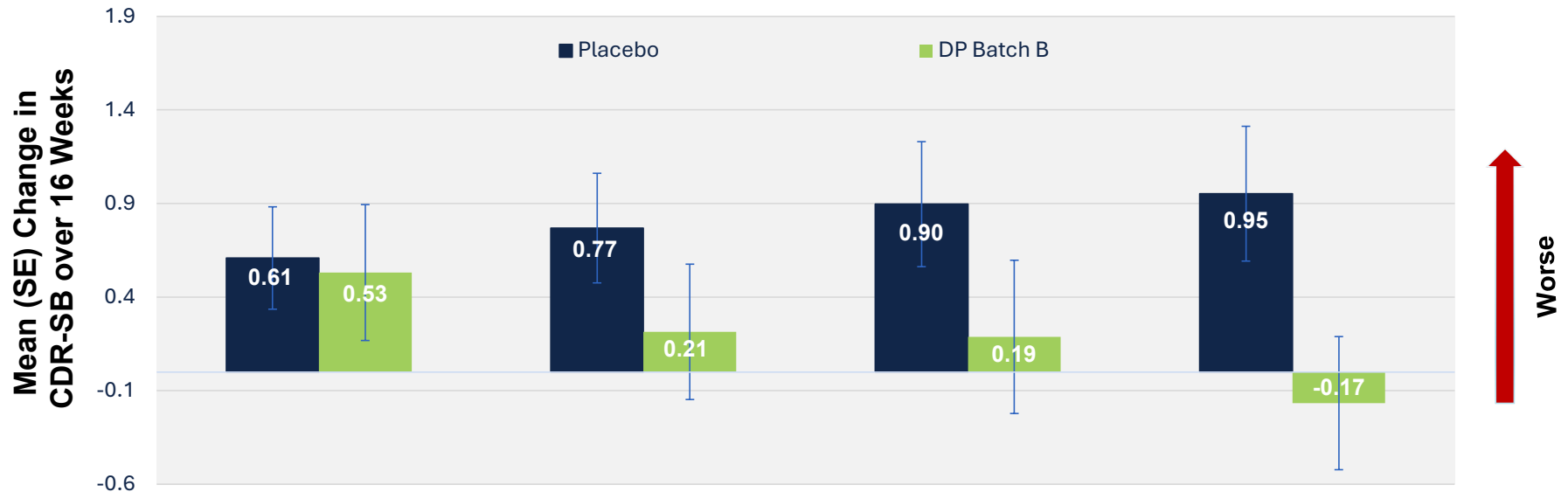
Estimated % of Participants Without AD Co-Pathology by Plasma pTau181 Cut-off

Stricter cut-off → purer DLB population



Optimal pTau181 cut-off of <21pg/ml validated externally in large (N=1,298) third party validation study published in February 2025¹

Stratified¹ further analysis of Phase 2b results support anticipated benefit of refined enrichment strategy to be utilized in planned Phase 3 trial



pTau181 Cut-off	<27.2 pg/mL	<25.2 pg/mL	<23 pg/mL	<21 pg/mL
Number of Participants	32	28	24	21
NFMD-Placebo Difference	-0.08	-0.55	-0.71	-1.11
P-value NFMD vs. Placebo	p=0.9	p=0.044	p=0.034	p=0.005

Anticipated milestones have potential to drive value through planned Phase 3 readout¹

- ✓ Select dose and dosing regimen for planned Phase 3 trial (1Q26)
- ✓ Present additional Phase 2b clinical data in DLB at AD/PD Conference (1Q26)
- ❑ **Initial Phase 2a biomarker data in nfvPPA (Mid-2026)**
- ❑ **Topline Phase 2a clinical data in RAS (2H26)**
- ❑ **Initial Phase 2a clinical data in nfvPPA (2H26)**
- ❑ **First patient dosed with neflamapimod in EXPERTS-ALS trial in ALS (4Q26)**

2026

2027

- ❑ **Initial topline Phase 2a clinical and biomarker data in nfvPPA (1Q27)**
- ❑ **Biomarker data from EXPERTS-ALS trial in ALS (2H27)²**

Experienced leadership team, committed to making a difference in age-related brain disorders

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CervoMed is advancing neflamapimod as a potential first-in-class therapy for the treatment of DLB



Well documented scientific rationale and clinically validated mechanism of action



Full Phase 2b data set demonstrates durable, clinically significant effect of neflamapimod in patients without AD co-pathology



DLB without AD co-pathology represents a large market opportunity with high unmet need



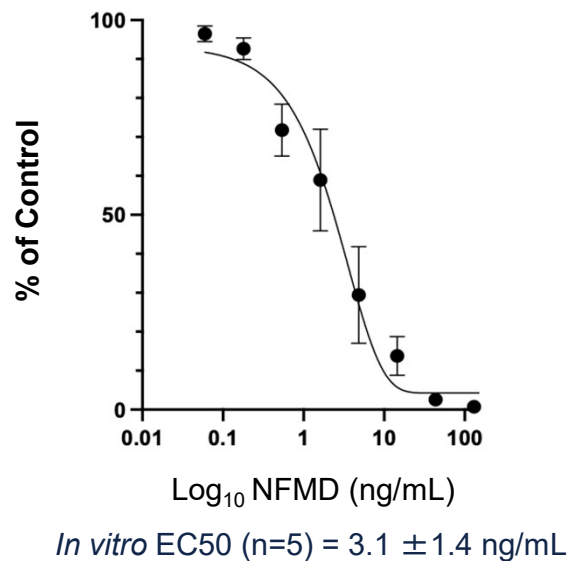
Potential registration path

PK / PD Annex

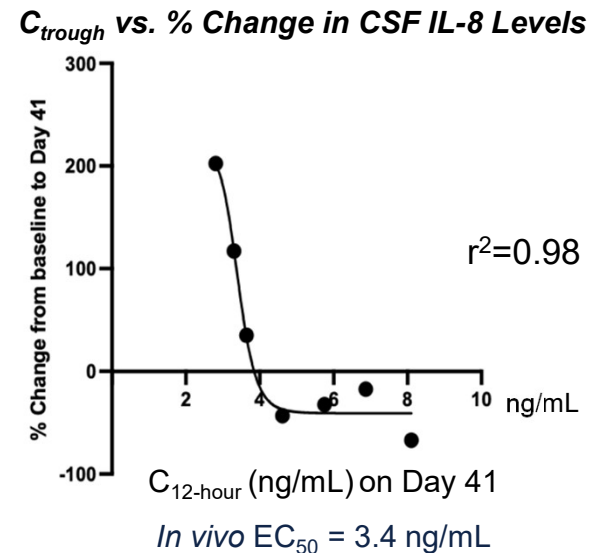


Concentration-pharmacodynamic (PD) relationship effect in vitro and in patients with early Parkinson's Disease

In Vitro: IL-1b stimulated IL-8 Production from PBMCs



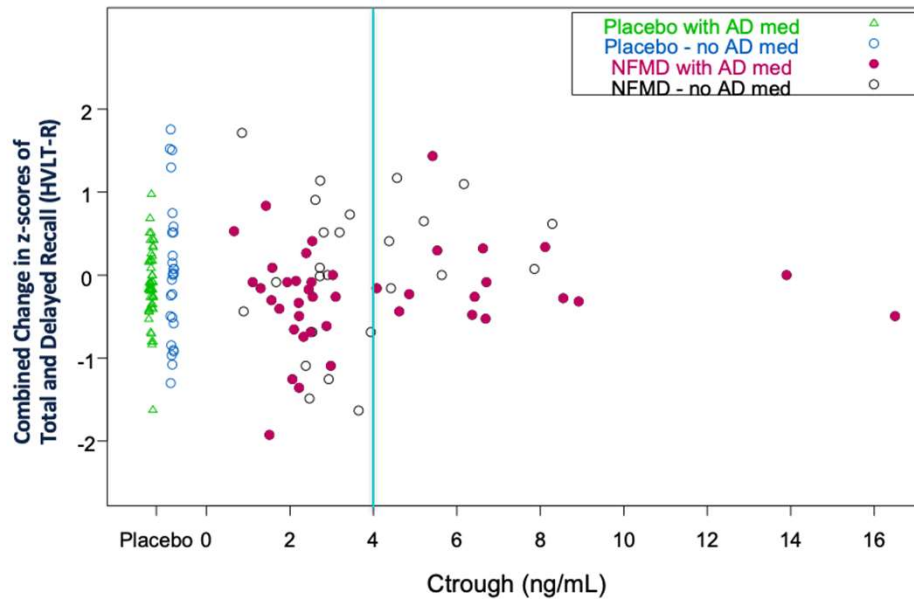
In Patients with Early AD: Change in CSF IL-8 After 6 Weeks of Neflamapimod Treatment



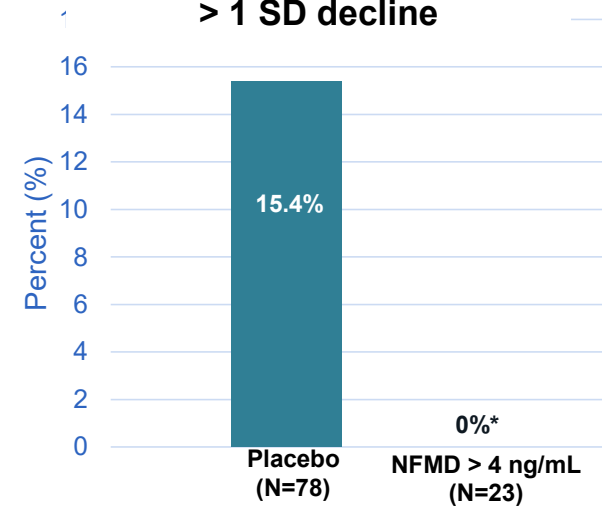
Maximal PD effect in patients achieved at ~4 ng/mL, i.e. when EC₅₀ is exceeded in plasma (2X EC₅₀ in brain)

C_{trough} threshold of 4 ng/mL for clinical activity PK-PD analysis of 24-Week placebo-controlled study in early AD (Reverse-SD Study)

Change from baseline to week 24 in primary endpoint



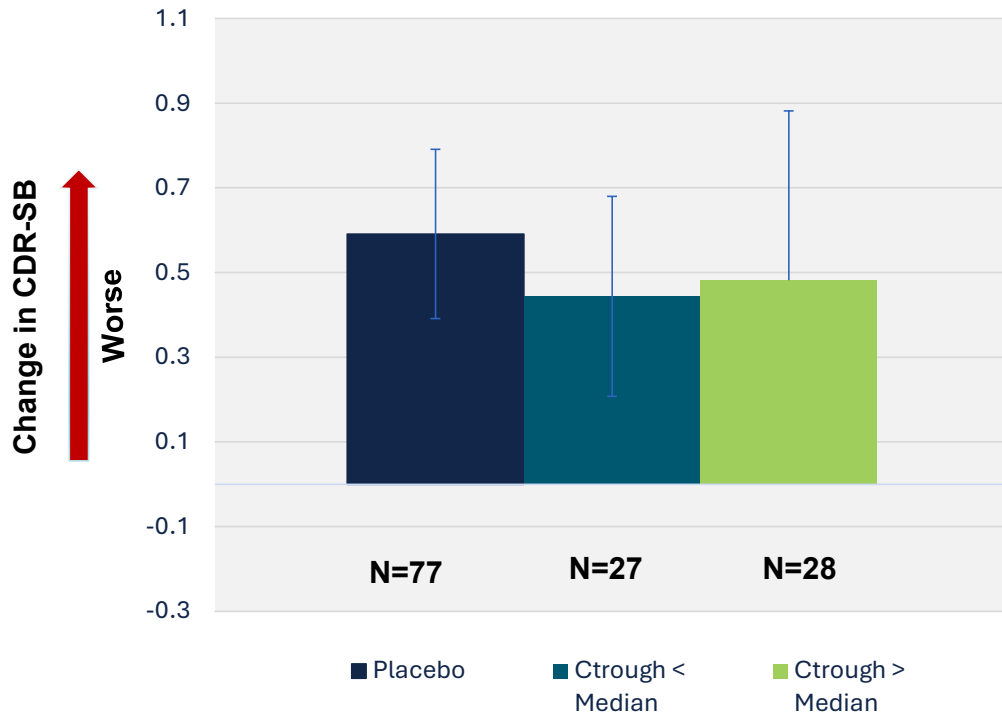
Percentage of patients with > 1 SD decline



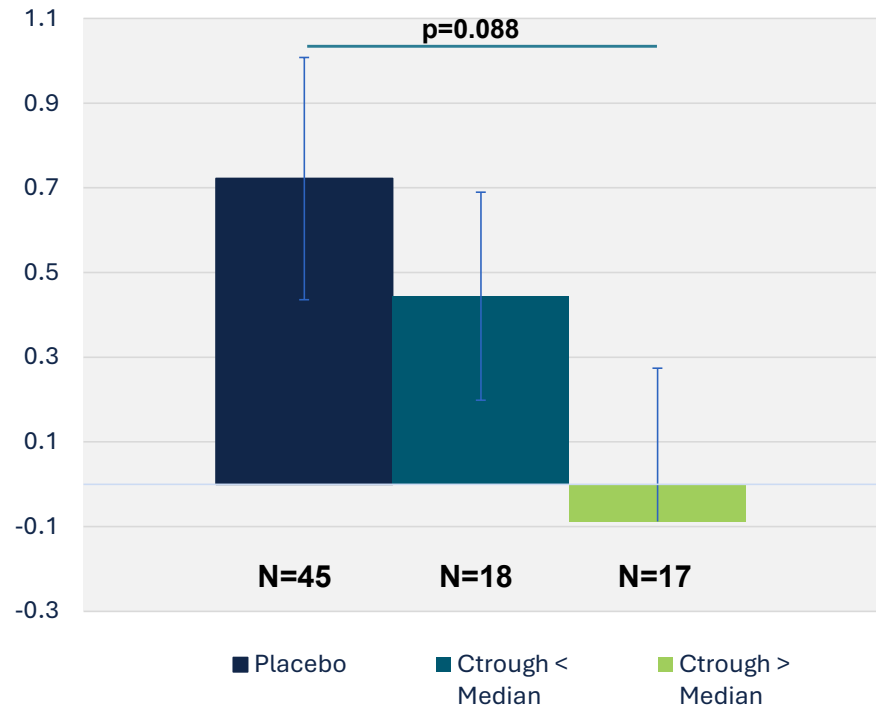
*p=0.06 vs. placebo, two-sided Fisher's exact test

Change in CDR-SB By neflamapimod C_{trough} during Randomized Phase of RewinD-LB

All Participants



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



PK-PD correlations for clinical activity are consistent with the potency and concentration relationship for primary pharmacology

	Percent of patients who achieve $C_{\text{trough}} \geq 4$ ng/ML	Clinical Activity
40mg BID (Phase 2a only)	25%	No discernible activity
40mg TID Batch A (Phase 2b)	50%	Marginal clinical activity, except potentially in those who achieve C_{trough} target
40mg TID Batch B (Phase 2b)	75%	Demonstrated improvement on CDR-SB, CGIC and plasma GFAP
Planned Phase 3 formulation and dose (50mg TID)	80-90% (Estimated)	TBD