CERVOMED

Medicines for the Brain

Corporate Presentation

June 2026

NASDAQ: CRVO CERVOMED

Forward-Looking Statements

This presentation includes express and implied forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, as amended, regarding the intentions, plans, beliefs, expectations or forecasts for the future of CervoMed Inc. (Company or CervoMed), including, but not limited to: the Company's anticipated cash runway; the Company's need to acquire sufficient funding, including funding for any Phase 3 trial in patients with dementia with Lewy bodies (DLB); the Company's plan to focus on strategic partnering to advance neflamapimod into Phase 3 for DLB and the timing of entering into any such partnership, if at all; the therapeutic potential of neflamapimod in DLB, non-fluent variant progressive primary aphasia (nfvPPA), amyotrophic lateral sclerosis (ALS), or any other indication, including the degree of sustainability of any therapeutic effects; the anticipated timing and achievement of clinical and development milestones, including the Company's initiation of any Phase 3 trial in patients with DLB; the anticipated data readouts from the Company's Phase 2a trial in nfvPPA and the anticipated dosing of the first patient with neflamapimod in the EXPERTS-ALS trial; any other expected or implied benefits or results, including the extent (if any) to which neflamapimod may demonstrate efficacy or other clinical or biomarker improvements in patients; and expectations with respect to neflamapimod, including the timing of any regulatory submissions and potential approvals thereof, if any, in DLB or any other indication. Terms such as "believes," "estimates," "anticipates," "expects," "plans," "aims," "seeks," "intends," "may," "might," "could," "might," "will," "should," "approximately," "potential," "target," "project," "contemplate," "predict," "forecast," "continue," or other words that convey uncertainty of future events or outcomes (including the negative of these terms) may identify these forward-looking statements. Although there is believed to be reasonable basis for each forward-looking statement contained herein, forward-looking statements by their nature involve risks and uncertainties, known and unknown, many of which are beyond the Company's control and, as a result, actual results could differ materially from those expressed or implied in any forward-looking statement. Particular risks and uncertainties include, among other things, those related to: the Company's available cash resources, the availability of additional funds on acceptable terms, and the Company's ability to continue as a going concern; the Company's ability to successfully enter into a partnership to advance neflamapimod into Phase 3 for DLB in a timely manner, on acceptable terms, or at all; the results of the Company's clinical trials; the likelihood and timing of any regulatory approval of neflamapimod or the nature of any feedback the Company may receive from the FDA or other regulators; the ability to implement business plans, forecasts, and other expectations in the future; general economic, political, business, industry, and market conditions, inflationary pressures, and geopolitical conflicts; and the other factors discussed under the heading "Risk Factors" in the Company's Annual Report on Form 10-K for the year ended December 31, 2025 filed with the U.S. Securities and Exchange Commission (SEC) on March 13, 2026, and other filings that the Company may file from time to time with the SEC. Any forward-looking statements in this presentation speak only as of June 15, 2026 (or such earlier date as may be identified). The Company does not undertake any obligation to update such forward-looking statements to reflect events or circumstances after the date of this presentation, except to the extent required by law. Certain analyses reported herein are exploratory in nature; p-values and indications of statistical significance, along with 95% confidence intervals, are being reported to provide a measure of the probability that any differences identified between the samples are due to chance.

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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 disease[1]

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.

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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

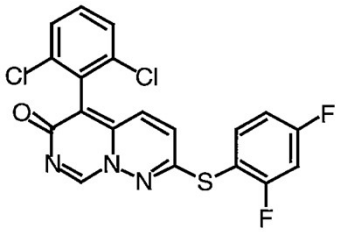
Robust pipeline in brain disorders

- Advancing clinical programs in nfvPPA and RAS
- 24-week biomarker data in Phase 2a trial in nfvPPA anticipated in 4Q26; 24-week clinical endpoint data in 1Q27
- 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 p38α, 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 p38α
- 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**

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

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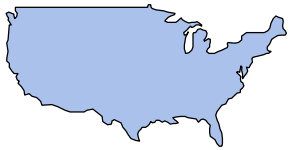
Pure DLB (those without AD co-pathology, ~50% of DLB overall) represents a substantial, untapped specialty market with high commercial potential

About Dementia with Lewy Bodies

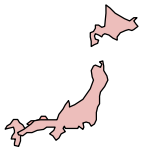
- Progressive α-synucleinopathy characterized by widespread cortical and subcortical Lewy bodies
- Alzheimer's disease (AD) co-pathology is common – present in up to ~50% of all DLB patients[1]
- Patients are generally managed by neurologists and the clinical diagnostic criteria are highly specific (>90%)[2]
- 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 care[3]
  - 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

DLB presents peak global revenue opportunity of $5B+

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.

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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

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

Schumacher et al, Brain (2022) 145:1773–1784; Alam et al, Neurology, (2023) 101 (17) e1708-e1717; Schumacher et al. Alz Res & Ther (2025) 17:28

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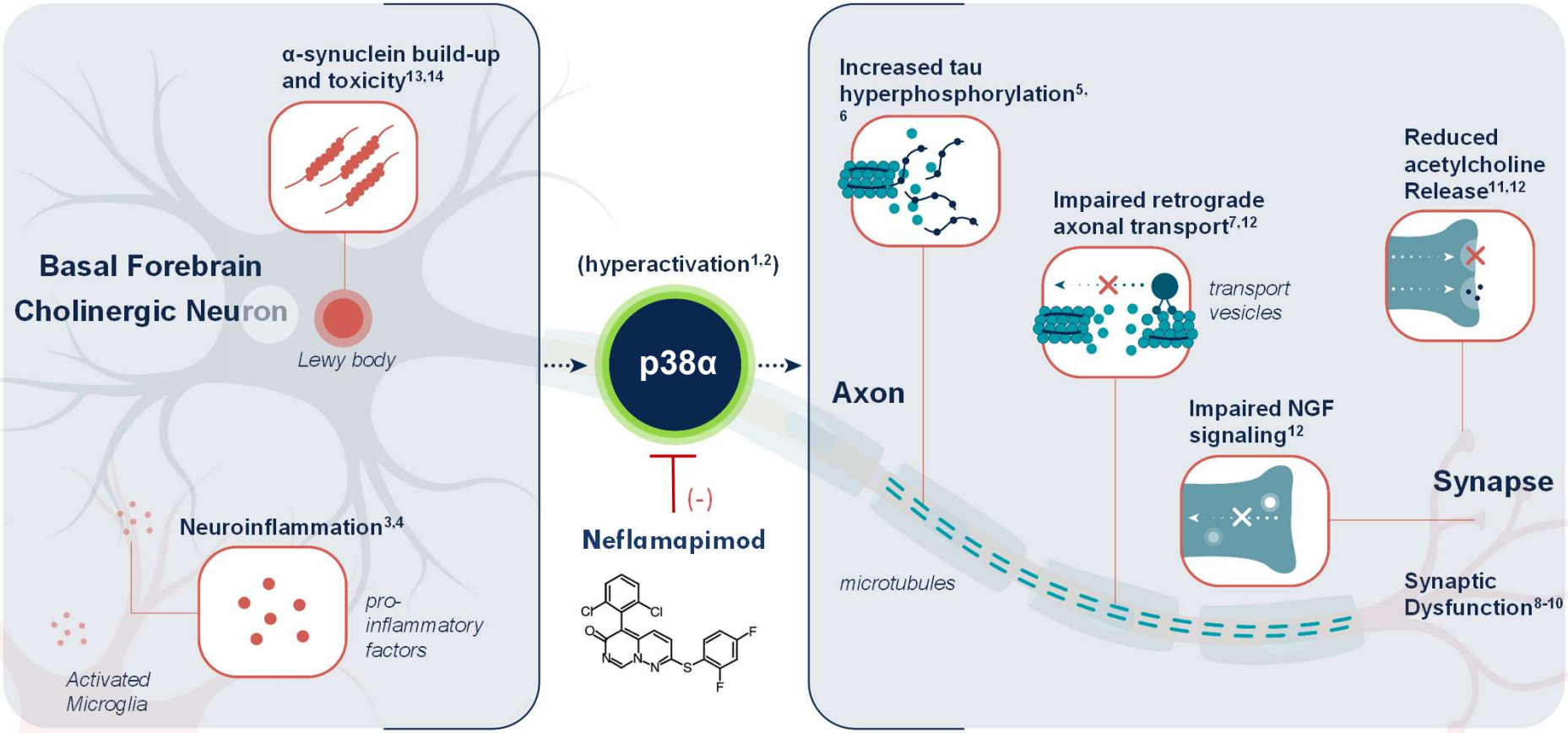
By blocking p38α, neflamapimod aims to reverse synaptic dysfunction and improve neuron health



References: 1. Asih, 2020 2. Son I, 2023 3. Prieto, 2015 4. Yang , 2023 5. Stefanoska , 2022 6. Yeganeh, 2025 7. Gibbs, 2018 8. Scali 2003 9. Ehrlich, 2012 10. Gamage, 2023 11. Jiang, 2022 12. Alam & Nixon, 2023. 13. Borland, 2022 14. Yang, 2025.

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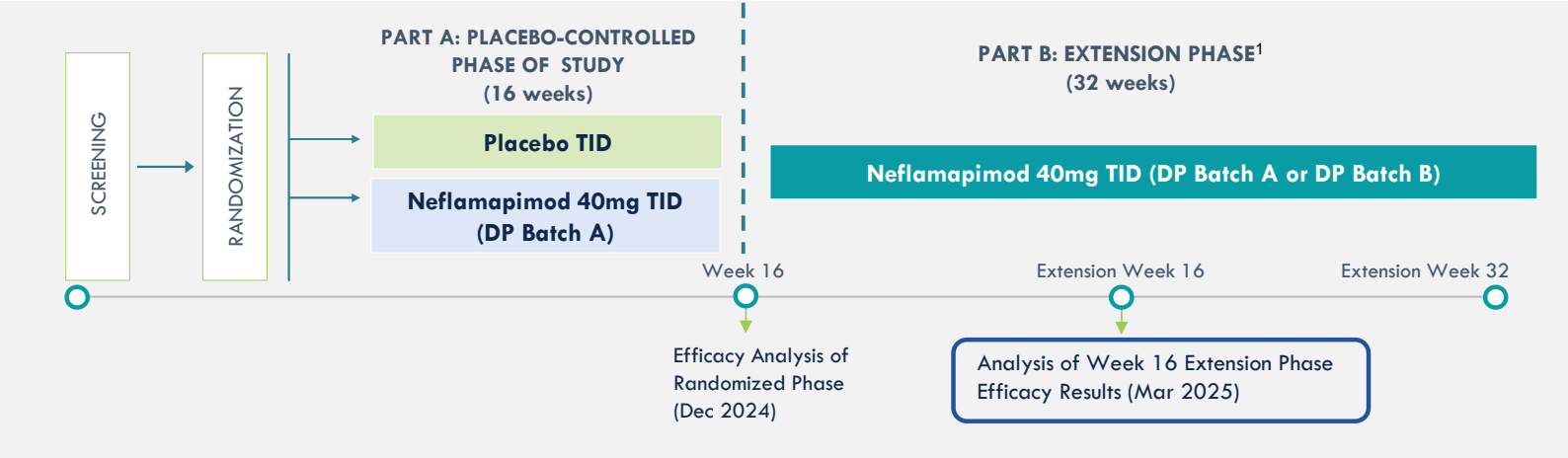
RewinD-LB open label Phase 2b study in DLB: Design and summary of results

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

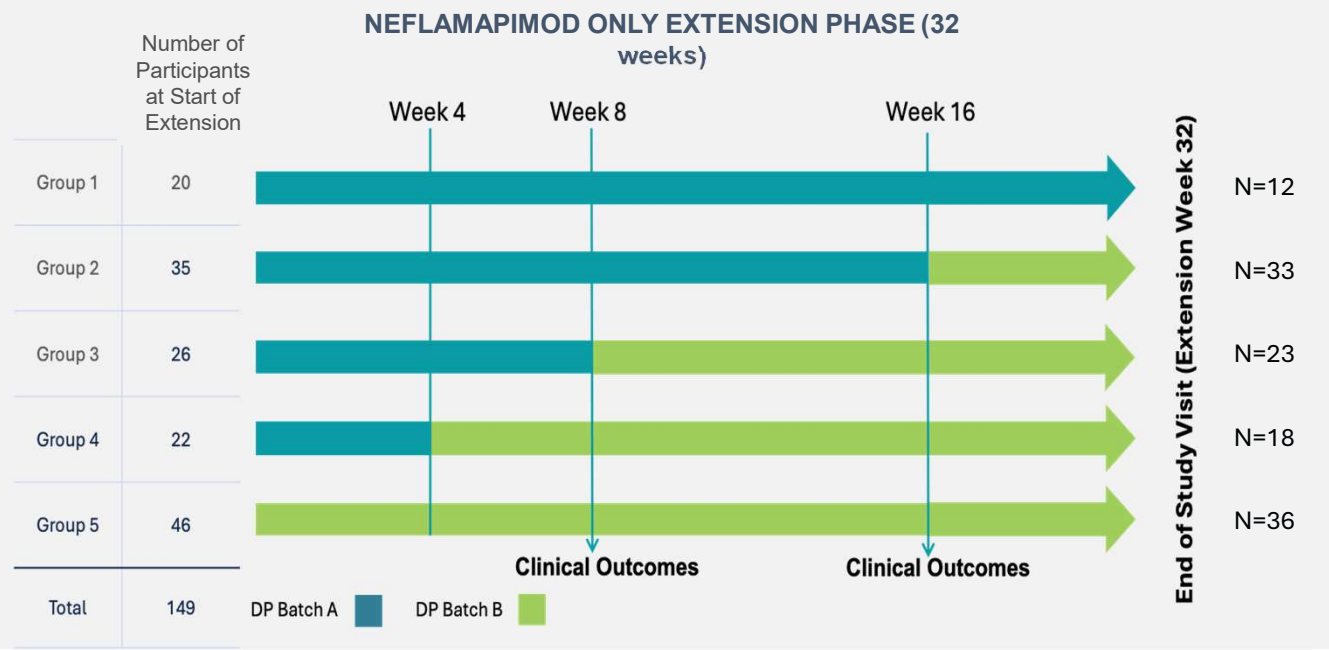
DP Batch B Results:

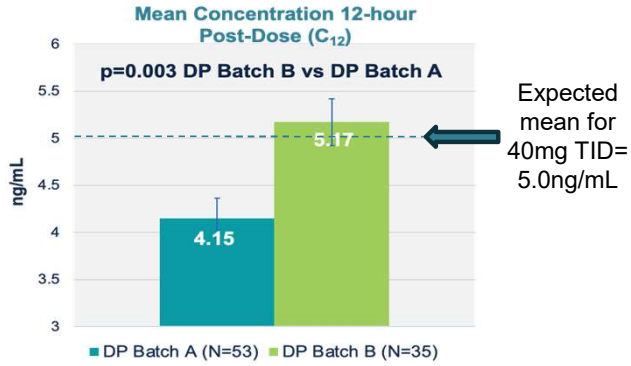
- Achieved plasma drug exposure expected with 40mg TID dosing regimen
- Significant improvement compared to Batch A on mean change in CDR-SB (primary outcome measure) and on ADCS-CGIC during Part B
- Significant reduction in plasma GFAP, compared to baseline and compared to DP Batch A

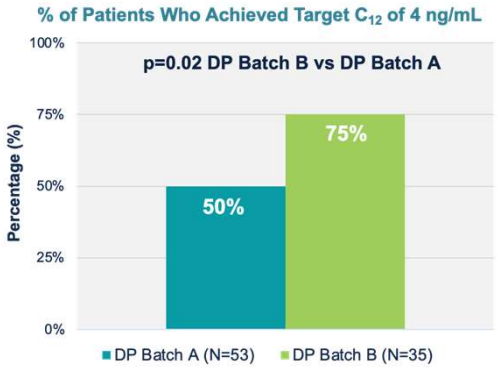
1. Participants were all aware that they were receiving neflamapimod in the Extension phase, but neither they nor study site personnel were aware if they were receiving DP Batch A or DP Batch B capsules

DP Batch B achieved expected and targeted plasma drug concentrations

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







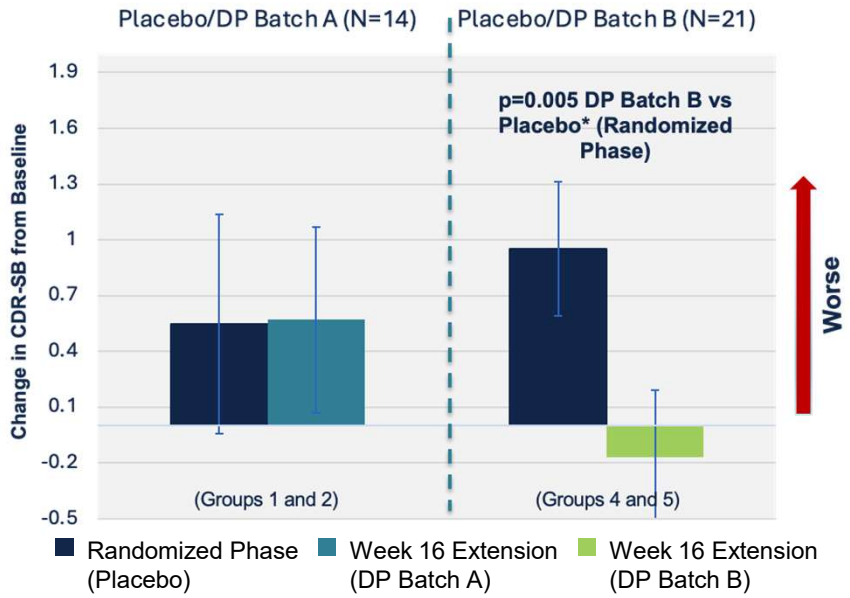
- Mean plasma drug trough concentration (12-hour post-dose, C_{12}) of 4.15ng/mL was less than expected mean 5.0ng/mL for 40mg TID, effectively underdosing participants, while mean C_{12} of DP Batch B was 5.17ng/mL
- Only 50% of DP Batch A (vs. 75% of DP Batch B) recipients achieved the individual patient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

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

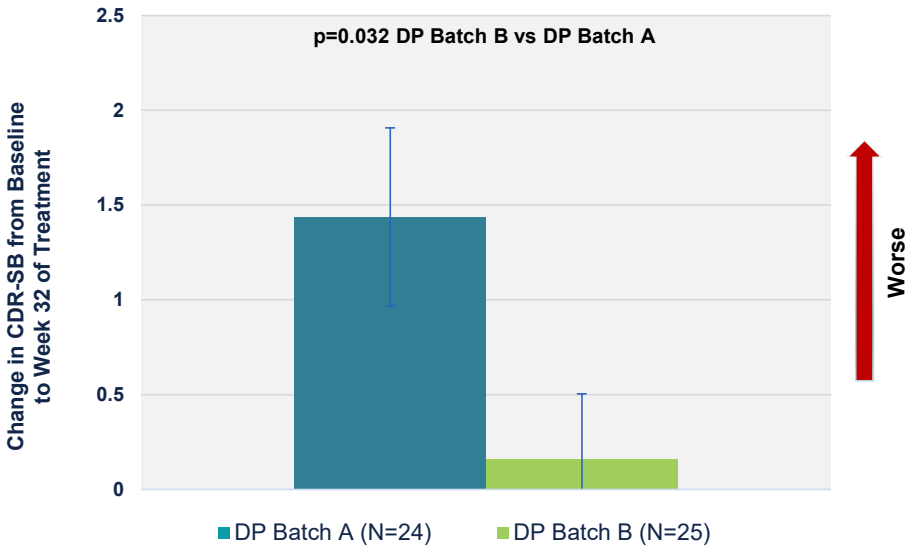
Drug product that achieved targeted plasma drug concentrations demonstrated >1-point improvement in mean change in CDR-SB in patient population to be enrolled in planned Phase 3 trial

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



Difference to Placebo: -1.12 with DB Batch B

32 Week Data Highlights Durability



Difference: -1.20, representing an 89% reduction in CDR-SB change with DP Batch B

- 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

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.

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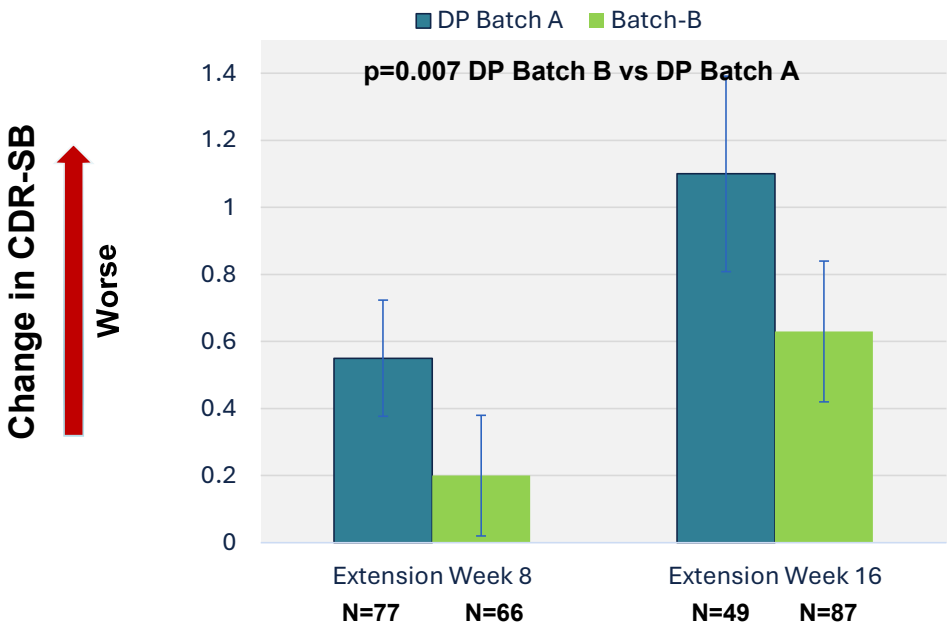
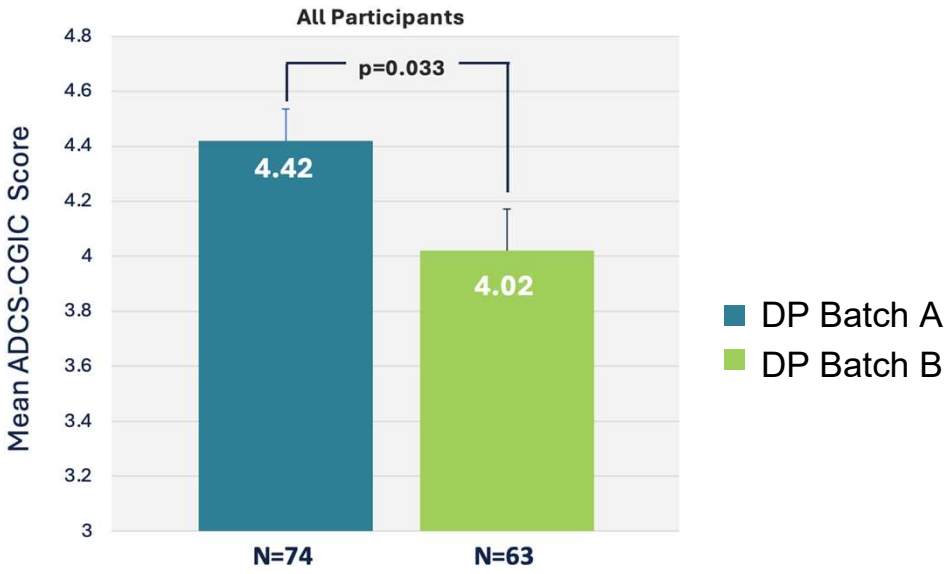
DP Batch B demonstrated statistically significant improvement in mean change in CDR-SB and ADCS-CGIC vs. Batch A

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



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



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.

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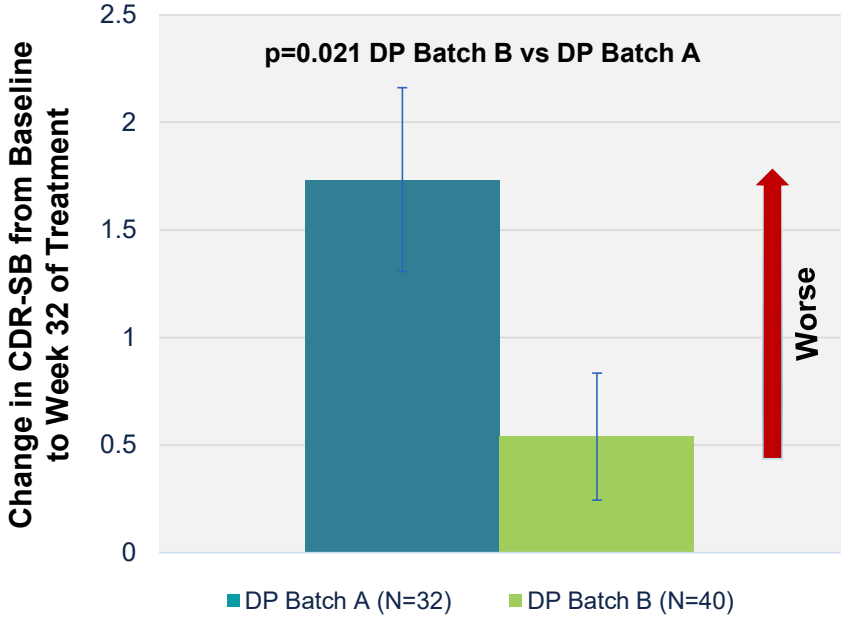
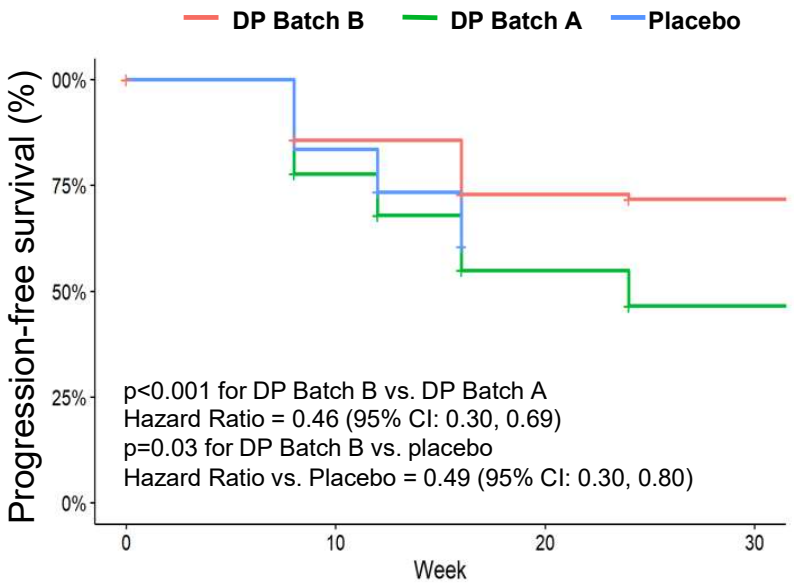
Clinical effect on CDR-SB with neflamapimod DP Batch B was durable out to 32 weeks of treatment

Mean Change in CDR-SB



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



Number at Risk			
	Week 8	Week 16	Week 24
DP Batch B	126	107	62
DP Batch A	117	68	26
Placebo	79	57	

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.

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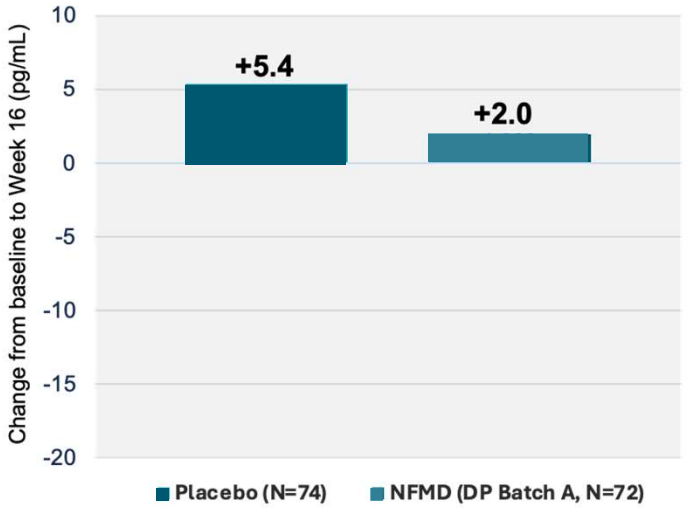
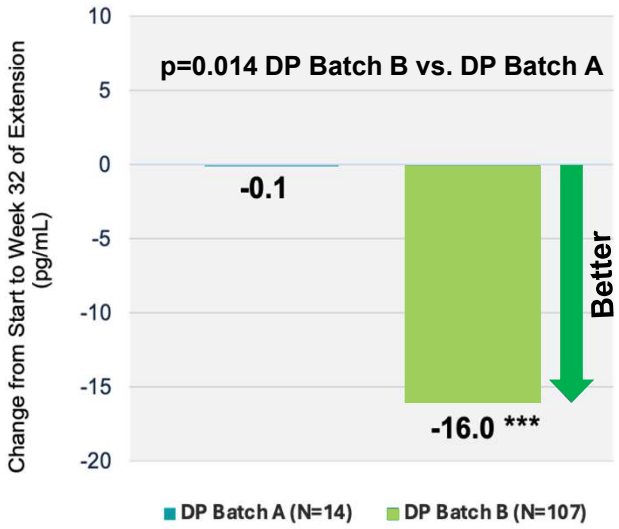
Neflamapimod DP Batch B achieved significant reduction in GFAP, a key biomarker of neurodegenerative disease activity

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

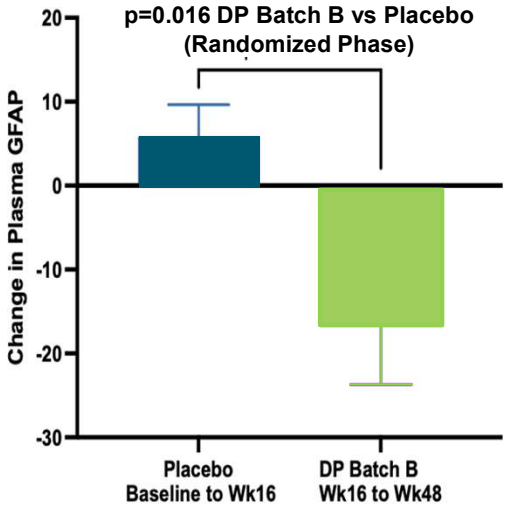


Median Change from Start to Week 32 of Extension



***p<0.001 for reduction from start of Extension for participants that received DP Batch B

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)

In center panel, DP Batch A data includes participants in Group 1 and DP Batch B includes all other participants. In right panel, DP Batch B data includes participants in Groups 2, 3, 4, and 5 who received placebo during placebo-controlled phase of study.

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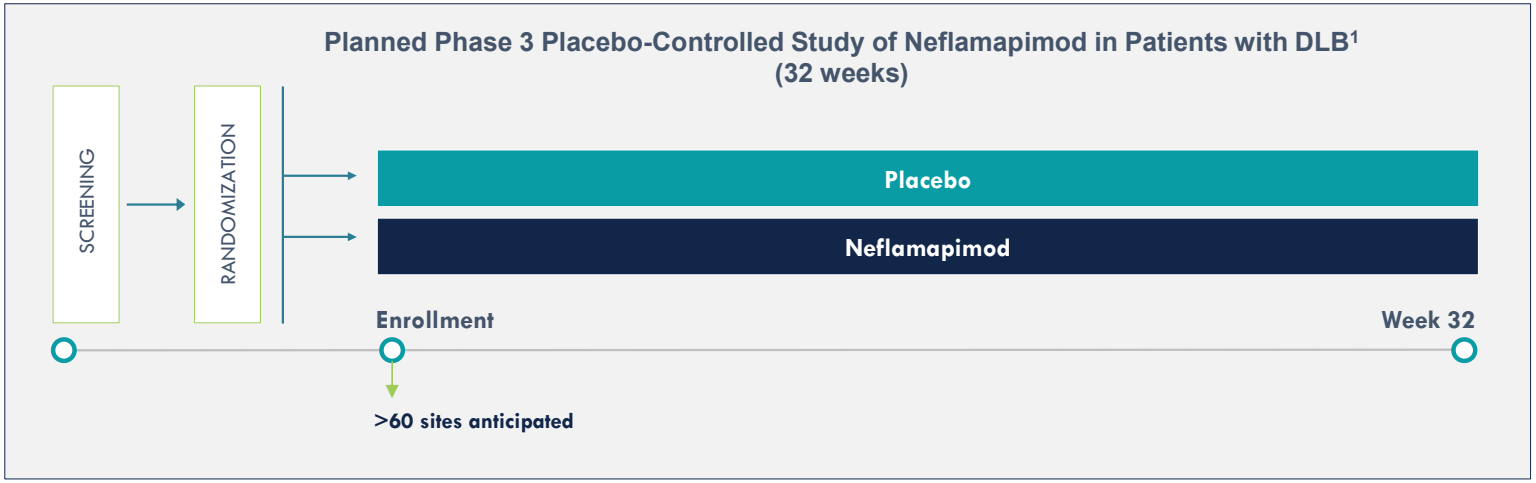
Engaged with the FDA on registrational trial design for DLB in November 2025



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

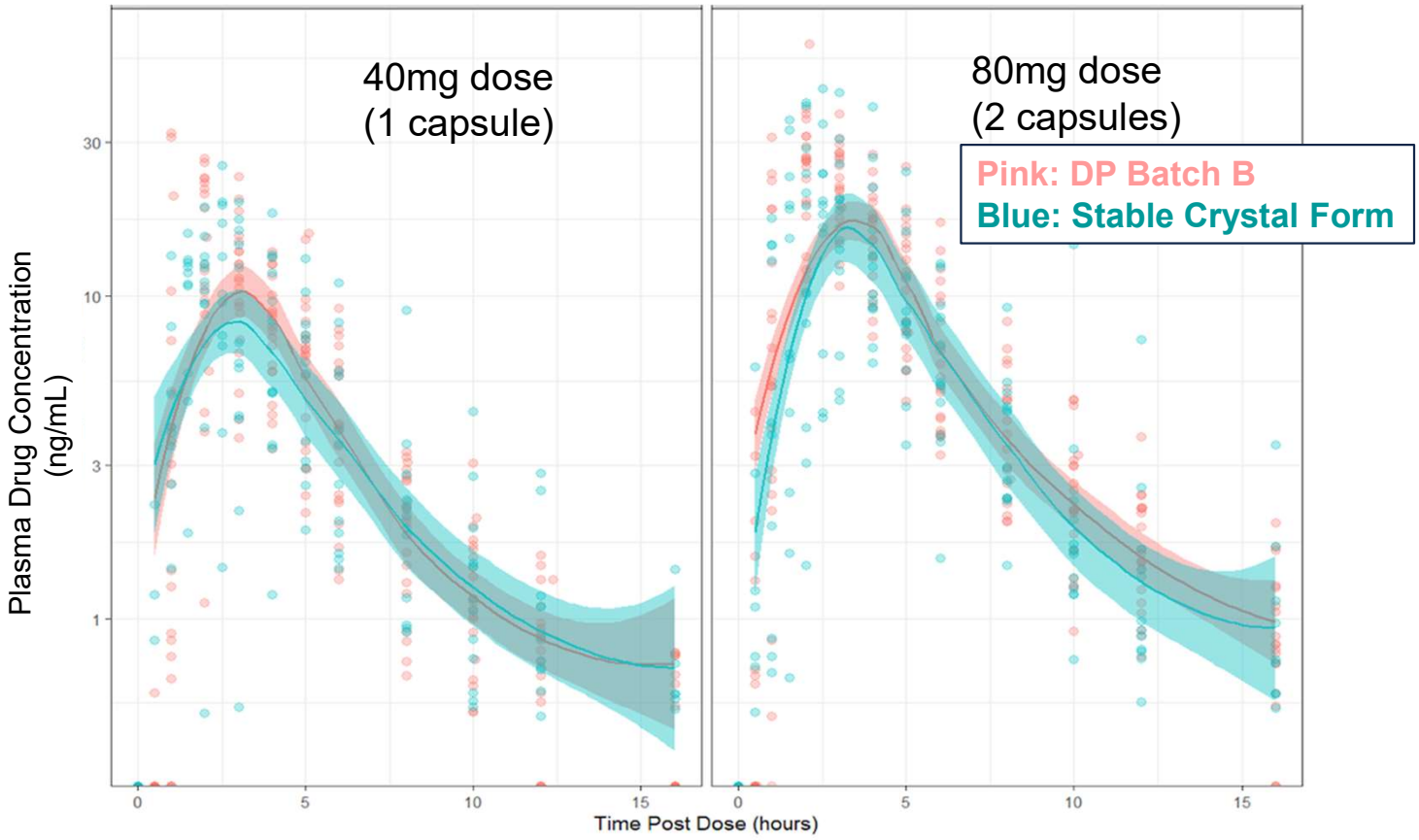
- Single Phase 3 clinical trial of 32 weeks duration, with change in CDR-SB as primary endpoint
- Obtaining feedback from global regulatory authorities and completing activities to support improvements in drug product formulation
- 50mg TID dosing regimen with new, stable crystal form of neflamapimod

1. Any trial initiation subject to the Company's establishment of a strategic partnership and/or receipt of sufficient funding.

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

- 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

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



PK = Pharmacokinetic.

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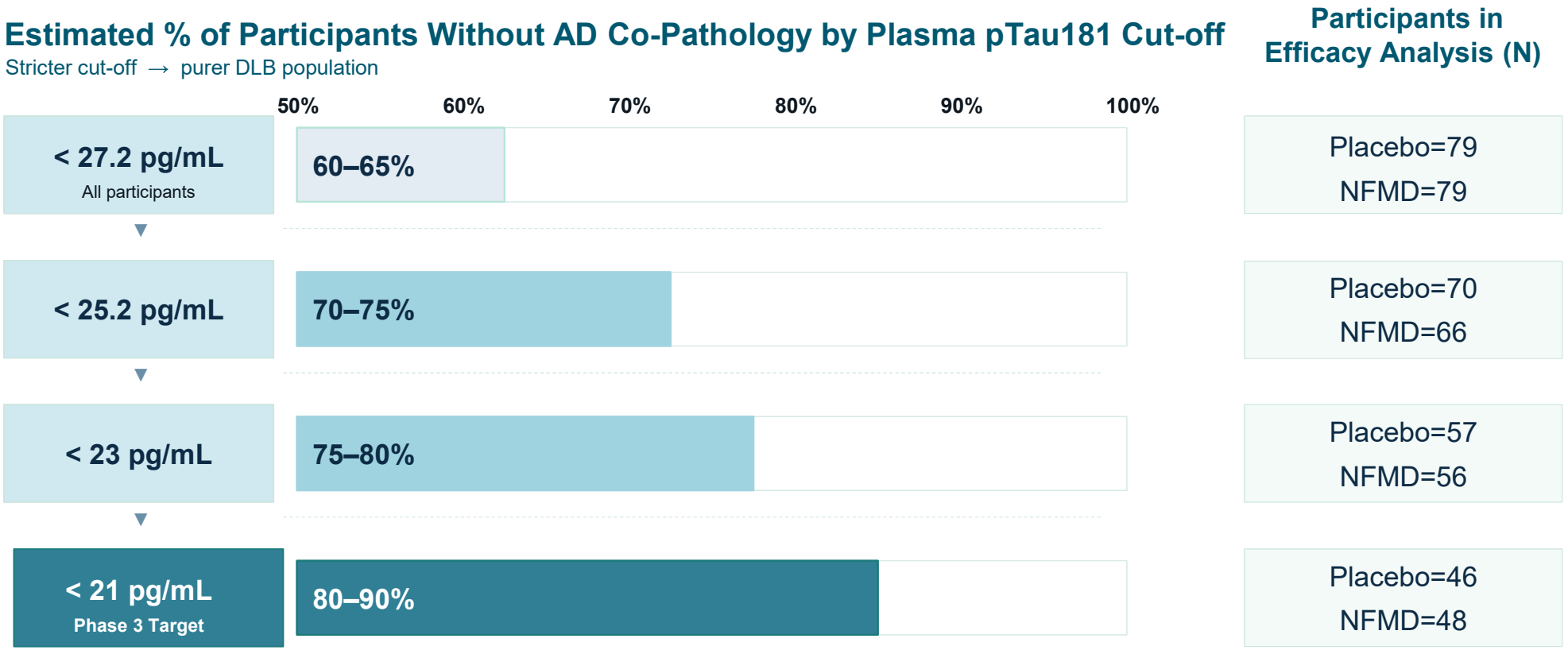
Lowering plasma pTau181 cut-off progressively enriches for patients without AD co-pathology



Optimal pTau181 cut-off of <21pg/ml validated externally in large (N=1,298) third party validation study published in February 2025[1]

NFMD = neflamapimod. 1. Doecke et al. 2025, Alzheimer's & Dementia, 2025;21:e14573.

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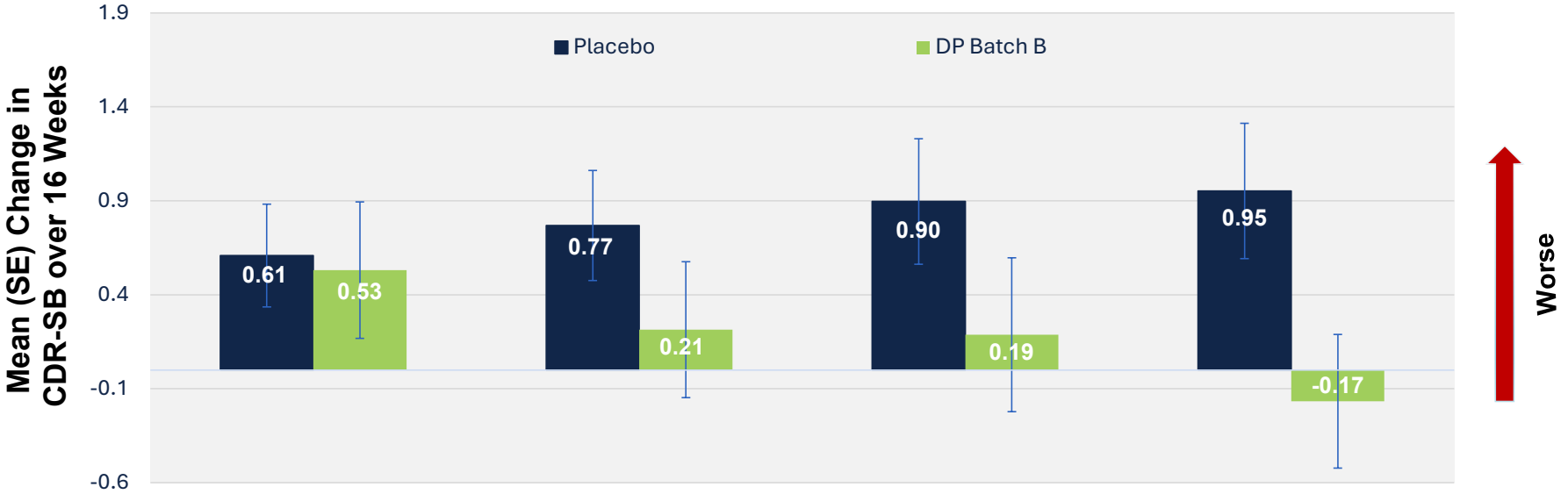
Stratified[1] further analysis of Phase 2b results support anticipated benefit of refined enrichment strategy to be utilized in planned Phase 3 trial



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

NFMD = neflamapimod. 1. Group 4 and Group 5 Patients, i.e. >12 weeks DP Batch B Treatment During 1st 16 weeks of Extension

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Anticipated milestones have potential to drive value in coming months

- ✓ Select dose and dosing regimen for planned Phase 3 trial (1Q26)
- ✓ Present additional Phase 2b clinical data in DLB at AD/PD Conference (1Q26)
- ❑ **Topline Phase 2a clinical data in RAS (2H26)**
- ❑ **24-week Phase 2a biomarker data in nfvPPA (4Q26)**
- ❑ **First patient dosed with neflamapimod in EXPERTS-ALS trial in ALS (4Q26)**
- ❑ **Target for establishing partnership to advance DLB into Phase 3 (YE26)**

2026

2027

- ❑ **24-week Phase 2a clinical data in nfvPPA (1Q27)**
- ❑ **Biomarker data from EXPERTS-ALS trial in ALS (2H27)**[1]

1. Subject to first patient being dosed by YE26 and sufficient funding. nfvPPA = non-fluent variant primary progressive aphasia. RAS = recovery after ischemic stroke.

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

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Professor, Dept. of Neurology & Neurosurgery, McGill University

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

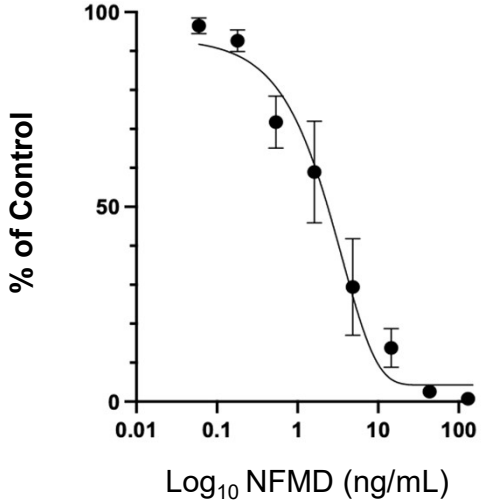
Potential registration path

CERVOMED

PK / PD Annex

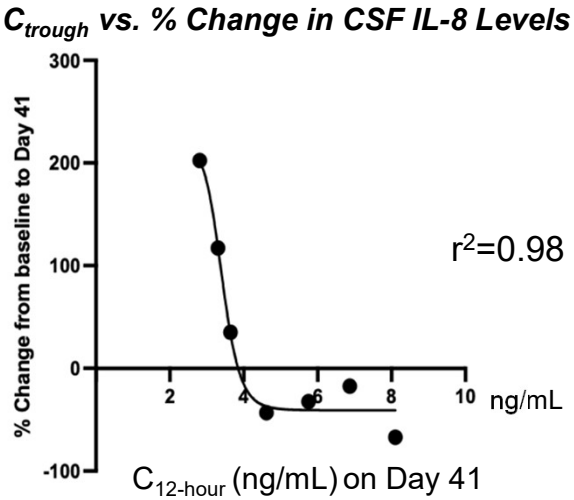
Concentration-pharmacodynamic (PD) relationship effect in vitro and in patients with early Parkinson's Disease

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



In vitro EC50 (n=5) = 3.1 ±1.4 ng/mL

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



In vivo EC_{50} = 3.4 ng/mL

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

Source: CervoMed Inc Data on File

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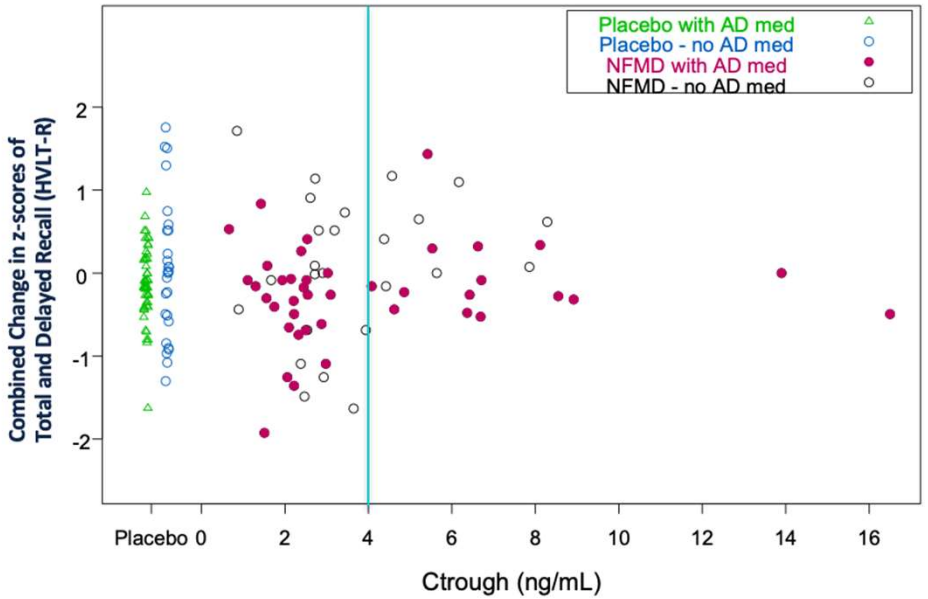
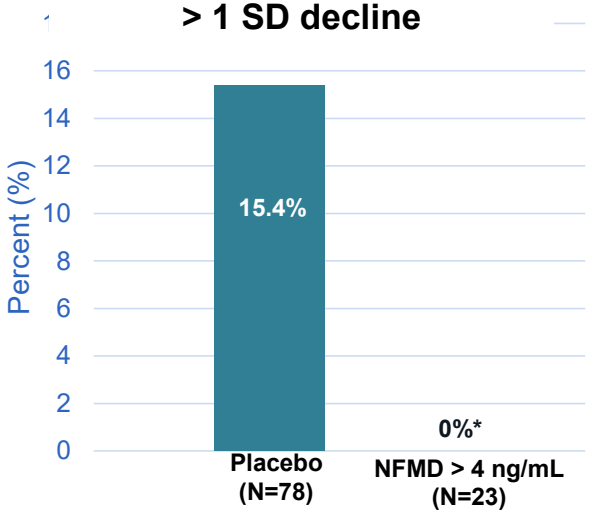
C_{trough} threshold of 4 ng/mL for clinical activity PK-PD analysis of 24-Week placebo-controlled study in early AD (Reverse-SD Study)

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

Primary endpoint: Combined z-score of total and delayed recall in HVLT-R (word recall test)

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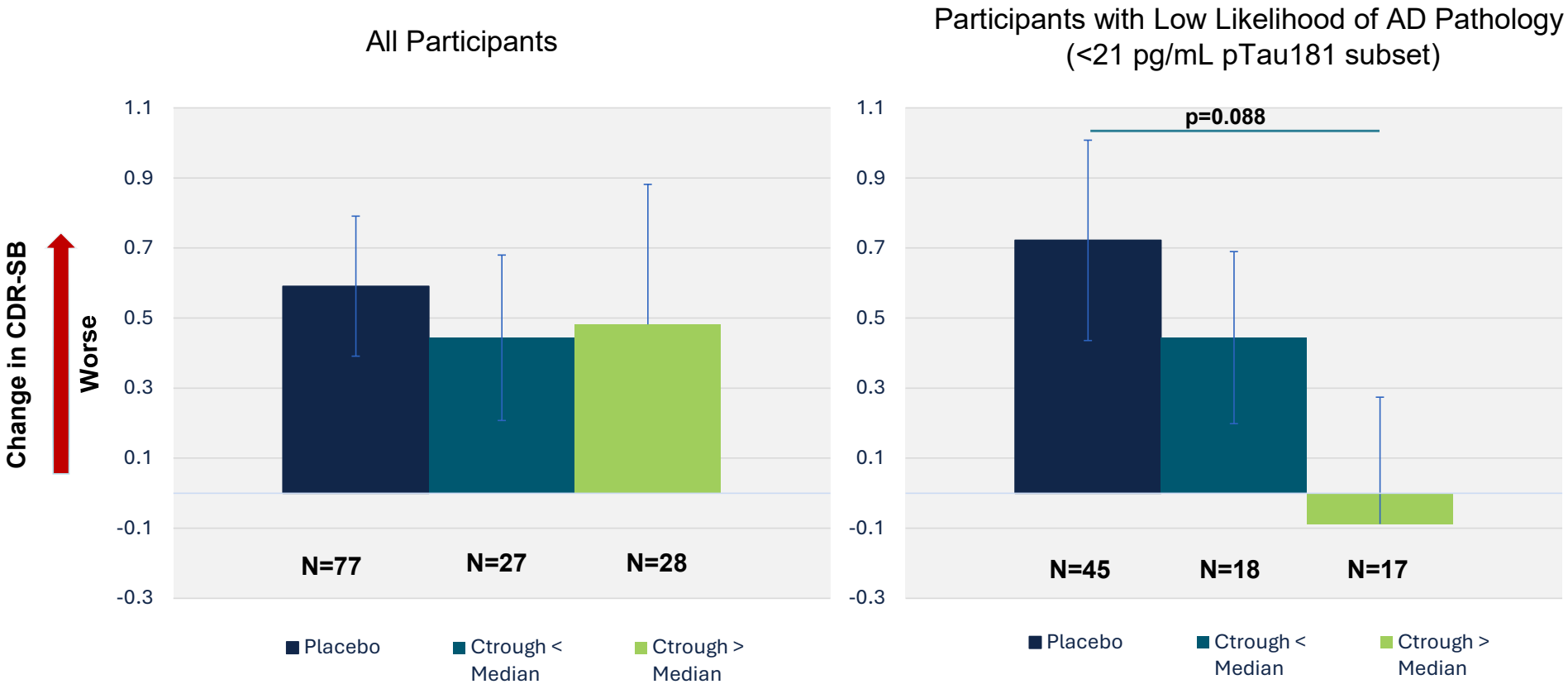
Change in CDR-SB By neflamapimod C_{trough} during Randomized Phase of RewinD-LB



Median Observed Ctrough = 3.6 ng/mL; calculated 12-hour post dose concentration, C12 = 4 ng/mL

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

	Percent of patients who achieve $C_{trough} \geq 4$ 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