CERVOMED

Medicines for the Brain

Corporate Presentation

March 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 the CervoMed Inc. (Company), including, but not limited to: the Company's need to acquire sufficient funding, including funding for its planned Phase 3 trial; our anticipated cash runway; the therapeutic potential of neflamapimod in DLB or any other indication, including the degree of sustainability of any therapeutic effects and the plasma drug concentrations that may be achieved with neflamapimod treatment in any of the Company's future clinical trials; the anticipated timing and achievement of clinical and development milestones, including the Company's initiation of the Company's planned Phase 3 trial in DLB patients and the announcement of any data therefrom; the anticipated data readouts from the Phase 2a trials in RAS and 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 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 March 17, 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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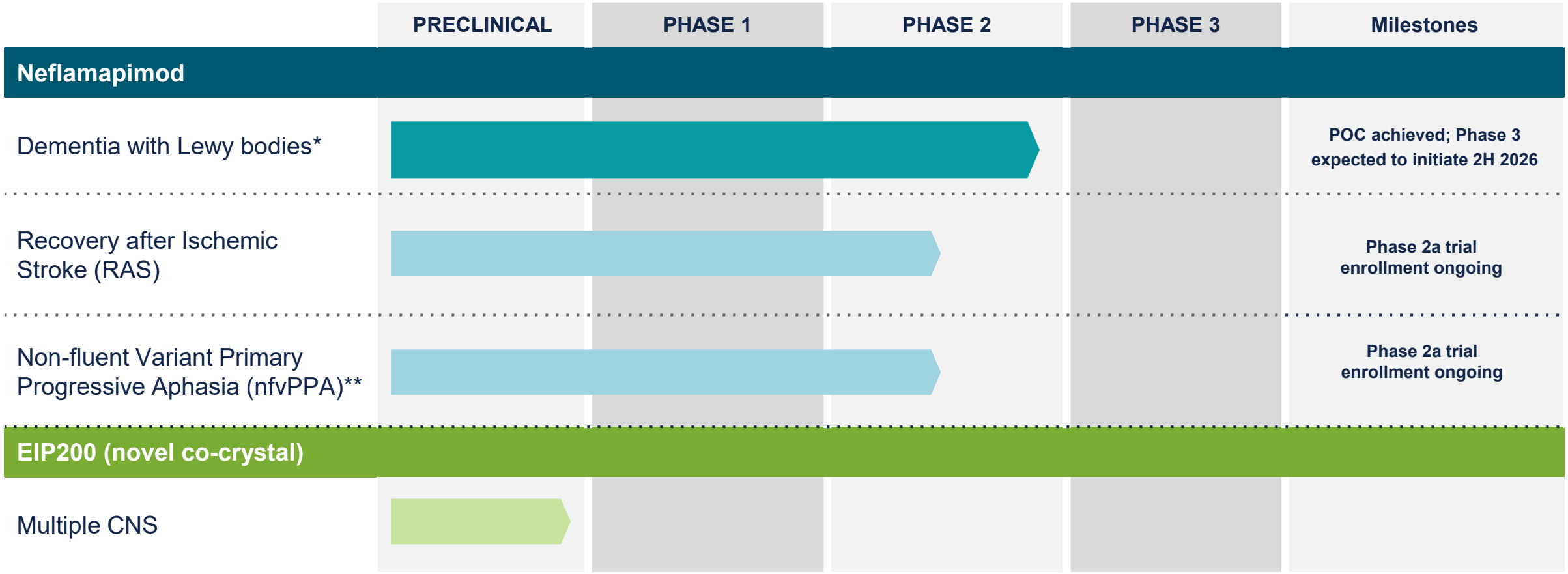
CervoMed Summary

- CervoMed is a clinical-stage company developing treatments for age-related brain disorders
- Our lead drug candidate, neflamapimod, is an oral, small molecule targeting critical disease processes underlying degenerative disorders of the brain by inhibiting a key enzyme involved in neuroinflammation and neurodegeneration
- Multiple catalysts anticipated over next 12 to 18 months[1], including planned initiation of Phase 3 in dementia with Lewy bodies (DLB) and Phase 2a data readouts in non-fluent variant primary progressive aphasia (nfvPPA) and recovery after ischemic stroke (RAS)
- Experienced executive leadership team, with strong board of directors and advisors

1. *Subject to the Company's receipt of sufficient funding.*

Robust pipeline targeting critical unmet needs in neurology



Worldwide commercial rights across programs
*Received FDA Fast Track designation
**Sub-type of Frontotemporal Disorders, for which neflamapimod has received FDA Orphan Drug designation

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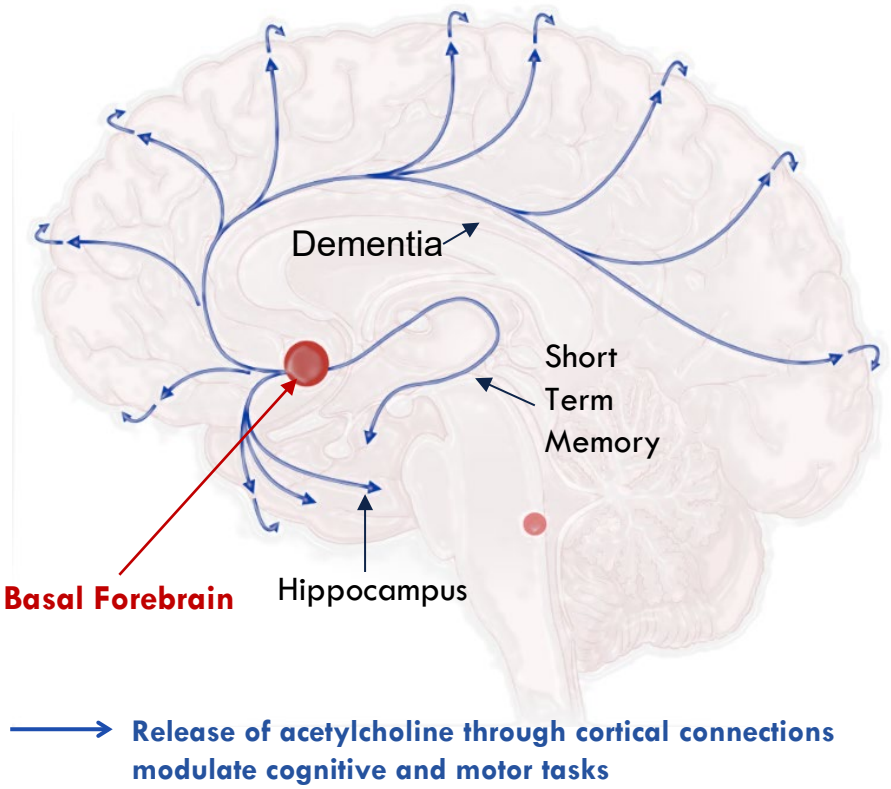
Degeneration of basal forebrain cholinergic neurons drives pathology across multiple neurological disorders

Basal Forebrain Cholinergic Complex



- **Central hub for cognition and behavior**, providing cholinergic input to cortex and hippocampus that drives memory, executive function, mobility, and attention
- **Highly vulnerable to degeneration**, with progressive basal forebrain cholinergic loss documented across multiple neurological disorders
- **Mechanistically validated therapeutic target**, where restoring cholinergic function offers broad clinical impact in high-unmet-need indications

Disease processes in basal forebrain are reversible

Adapted from Alam & Nixon, *Molecular Neurodegeneration*, 2023. 1. APP: Amyloid Precursor Protein

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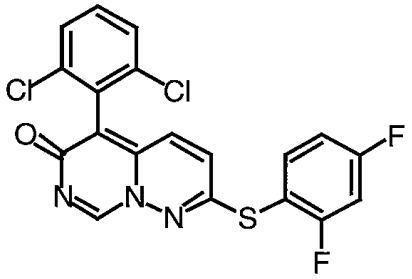
Neflamapimod is an oral, small molecule drug that selectively inhibits p38α, a key driver of neuroinflammation and synaptic dysfunction in the basal forebrain



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 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, recovery after ischemic stroke, and frontotemporal dementia.

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Age-related brain disorders affect millions and have widespread cognitive and functional impact

Dementia with Lewy Bodies

- Patients accumulate protein deposits, called Lewy bodies, in the brain's nerve cells
- Negatively affects cognitive ability, including attention, judgement, and reasoning, along with motor function

Frontotemporal Disorders

- Primarily affects the frontal and temporal lobes of the brain
- Symptoms characterized by progressive deterioration in behavior, personality, and language abilities
- Two major categories are frontotemporal dementia (FTD) and primary progressive aplasia (PPA)

Recovery after Ischemic Stroke

- Stroke, caused by blockage of arteries supplying blood to the brain, is the number one cause of long-term disability in adults
- Most stroke survivors have some level of recovery after a stroke
- In moderately severe and severe stroke, recovery is often incomplete

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CervoMed is well-positioned for a transformative year in 2026 and beyond

First- and best-in-class potential in DLB

- ✓ Positive Phase 2b data demonstrated proof-of-concept in DLB
- ✓ Alignment with FDA on Phase 3 study design and path to registration
- Phase 3 trial (32-week treatment duration) expected to initiate in 2H 2026[1]

Robust pipeline in brain disorders

- Advancing clinical programs in non-fluent variant primary progressive aphasia and recovery after stroke
- Phase 2a data readouts expected for nfvPPA in mid-2026 and for RAS in 2H 2026
- 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

[1] *Subject to the Company's receipt of sufficient funding.*

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The Opportunity for Neflamapimod in Dementia with Lewy Bodies

About dementia with Lewy bodies

- High unmet clinical need
  - Significant impact on quality of life and caregiver burden
  - Progresses more rapidly than AD, with average time from diagnosis to requiring nursing home care being 2 years
- 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
- Clinical diagnostic criteria are highly specific (>90%)
  - Biomarker confirmation typically not required
- In early stages, a major driver of disease expression and progression is dysfunction and degeneration of basal forebrain cholinergic neurons

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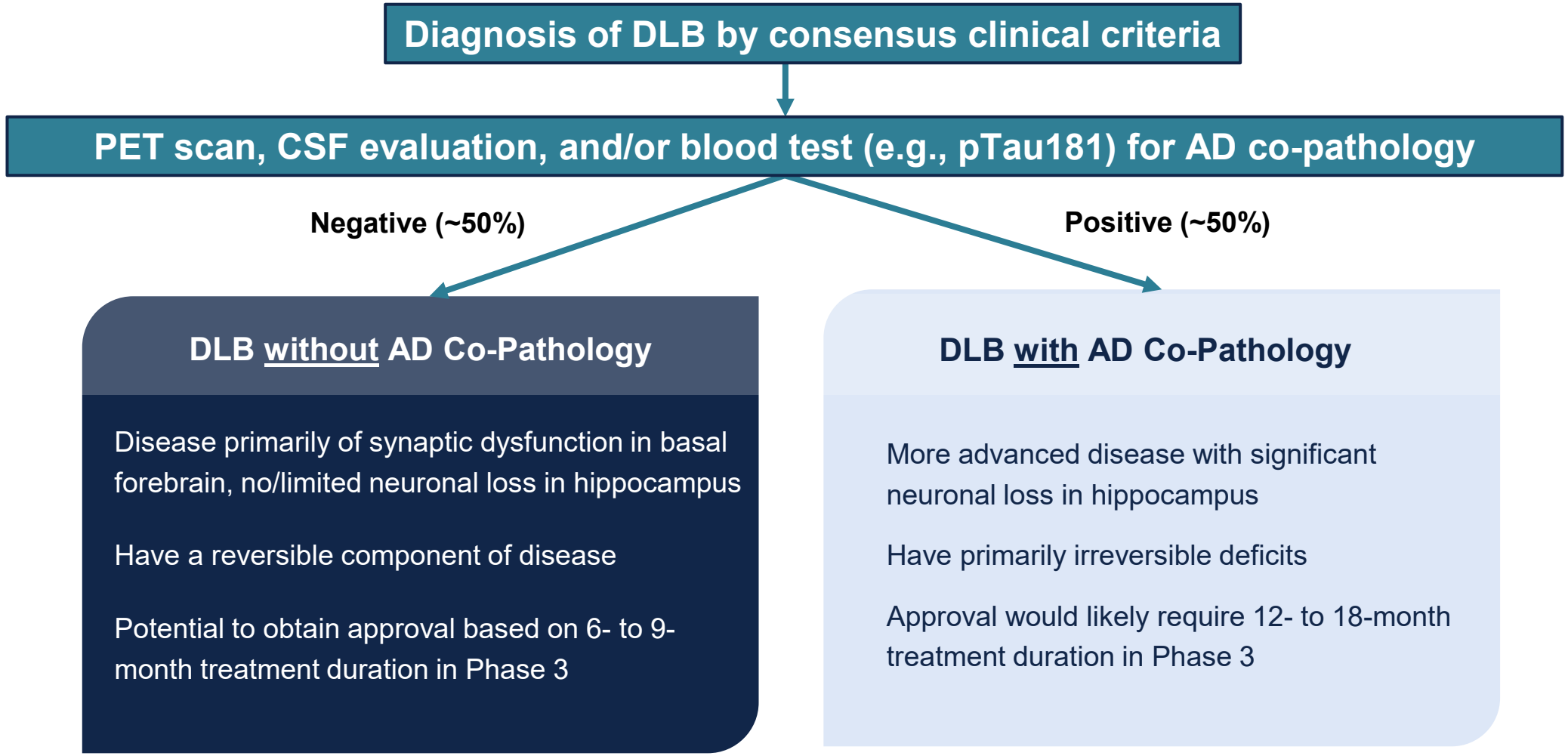
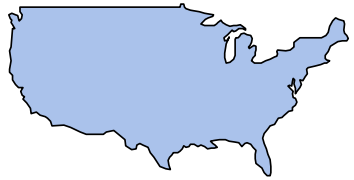
DLB without AD co-pathology offers clear target and focused drug development opportunity with potentially faster timeline to approval



Effectively treating synaptic dysfunction can rapidly lead to significant clinical effects in DLB without AD co-pathology

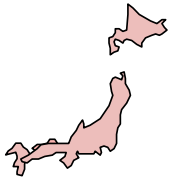
DLB without AD co-pathology (~50% of DLB patients) represents a substantial, untapped specialty market with high commercial potential

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**

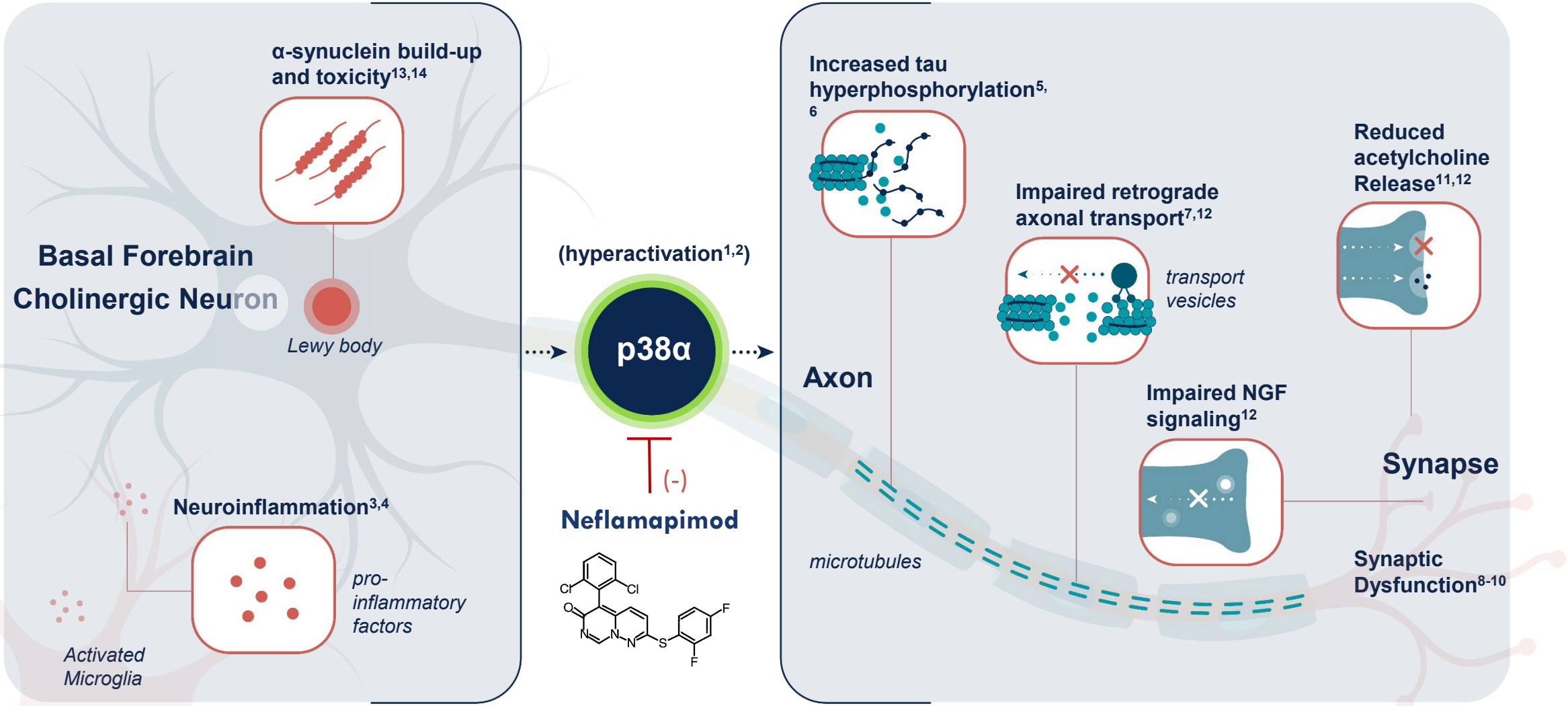
<u>Despite highly specific clinical criteria there is often a delay in diagnosis; highlighting a need for increased physician education</u>

Current Treatment Landscape

- Acetylcholinesterase inhibitors are the mainstay of treatment
  - Provide transient improvement in cognition, but no improvement in motor function
- There are no approved therapies that target the underlying disease process in the US or EU
- Patients are generally managed by neurologists

1. 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.

By blocking p38α, neflamapimod aims to reverse synaptic dysfunction, improve neuron health, and slow disease progression in the basal forebrain



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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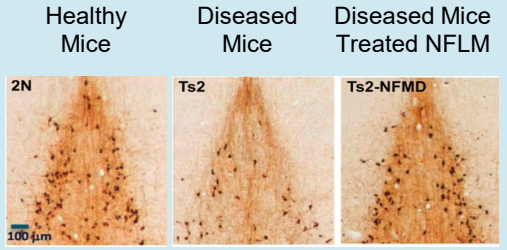
Preclinical and clinical results prior to Phase 2b clinical trial

Preclinical — *Disease processes in basal forebrain reversed*[1]

When administered in mice that develop basal forebrain cholinergic degeneration, neflamapimod:

- ✓ Reduced Rab5 activity and tau phosphorylation
- ✓ Reversed loss of cholinergic (ChaT+) neurons in the basal forebrain; and
- ✓ Normalized performance in behavioral tests of cholinergic function[2]

Cholinergic Neurons in Basal Forebrain

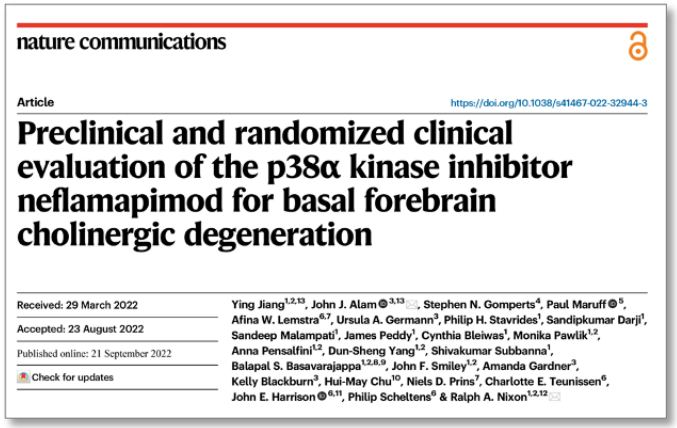


Neflamapimod preserved cholinergic neurons in the basal forebrains of mice

Phase 2a Clinical — *Improvement on multiple clinical endpoints*

In AscenD-LB, a 91-patient, 16-week, placebo-controlled Phase 2a trial in patients with DLB, neflamapimod:

- ✓ Significantly improved dementia severity (assessed by Clinical Dementia Rating Sum-of Boxes, (CDR-SB), p=0.023 vs. placebo)
- ✓ Significantly improved gait (assessed by Timed Up and Go (TUG), *p*=0.044 vs. placebo)
- ✓ Reduced levels of plasma biomarker of neurodegeneration (glial fibrillary acidic protein (GFAP))
- ✓ Results most prominent in patients without AD co-pathology (i.e., patients without elevation in plasma ptau181)


nature communications

Article

https://doi.org/10.1038/s41467-022-32944-3

Preclinical and randomized clinical evaluation of the p38α kinase inhibitor neflamapimod for basal forebrain cholinergic degeneration

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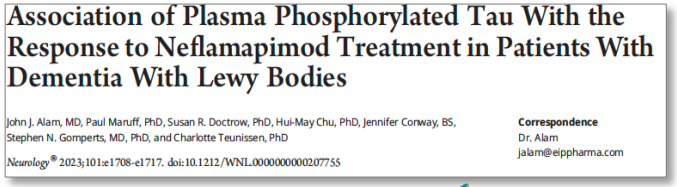
Check for updates

Ying Jiang[1,2,13], John J. Alam[3,13], Stephen N. Gomperts[4], Paul Maruff[5], Afina W. Lemstra[6,7], Ursula A. Germann[3], Philip H. Stavrides[1], Sandipkumar Darji[1], Sandeep Malampati[1], James Peddy[1], Cynthia Bleiwas[1], Monika Pawlik[1,2], Anna Pensalfini[1,2], Dun-Sheng Yang[1,2], Shivakumar Subbanna[1], Balapal S. Basavarajappa[1,2,8,9], John F. Smiley[1,2], Amanda Gardner[3], Kelly Blackburn[3], Hui-May Chu[10], Niels D. Prins[7], Charlotte E. Teunissen[6], John E. Harrison[6,11], Philip Scheltens[6] & Ralph A. Nixon[1,2,12]


Association of Plasma Phosphorylated Tau With the Response to Neflamapimod Treatment in Patients With Dementia With Lewy Bodies

John J. Alam, MD, Paul Maruff, PhD, Susan R. Doctrow, PhD, Hui-May Chu, PhD, Jennifer Conway, BS, Stephen N. Gomperts, MD, PhD, and Charlotte Teunissen, PhD

Neurology® 2023;101:e1708-e1717. doi:10.1212/WNL.0000000000207755

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1. Alam & Nixon, *Molecular Neurodegeneration, 2023.* 2. Nature Communications, 13: 5308 (2022). https://www.nature.com/articles/s41467-022-32944-3
2. Neurology® 2023;101:e1708-e1717. doi:10.1212/WNL.0000000000207755

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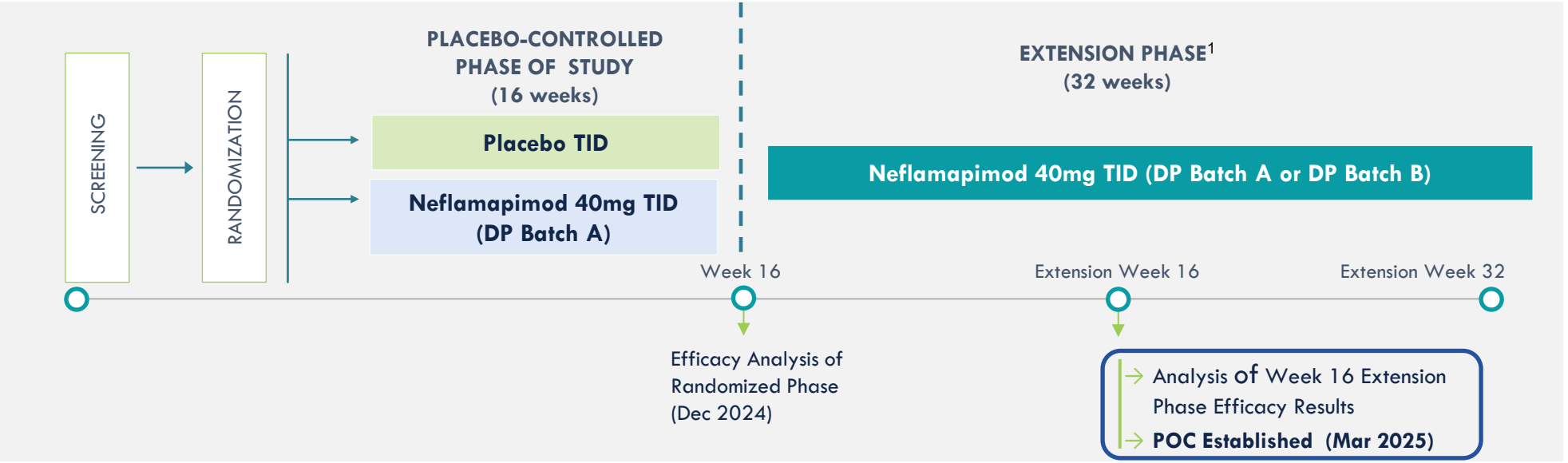
RewinD-LB Phase 2b Study in DLB: Design and Conduct

PATIENTS

- 159 patients with dementia with Lewy bodies 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)

SELECTED KEY CLINICAL OUTCOME MEASURES

- **Primary**: Clinical Dementia Rating Sum of Boxes (CDR-SB)
- **Secondary**: Clinical Global Impression of Change (CGIC) , Timed Up and Go, NTB,

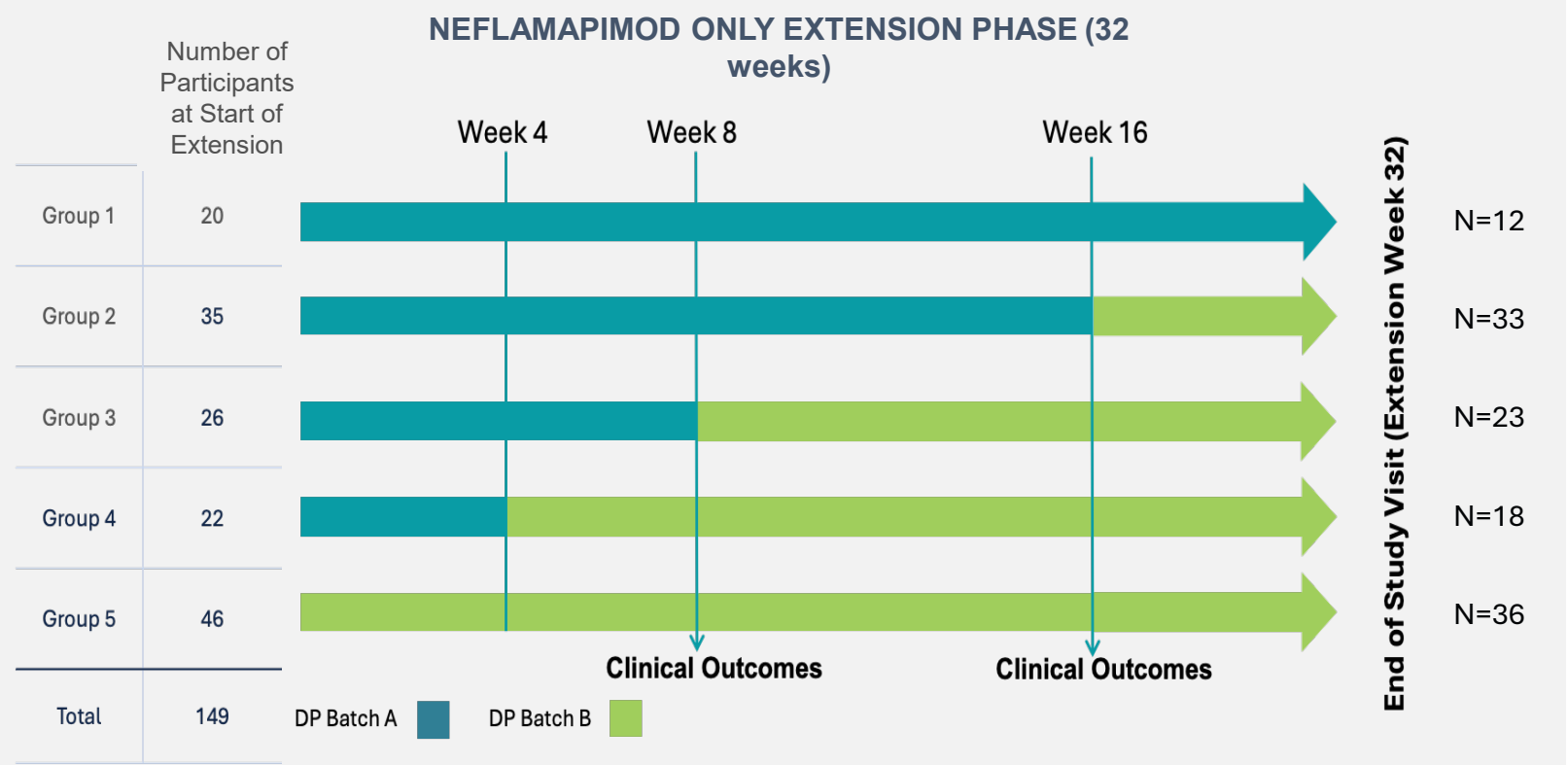


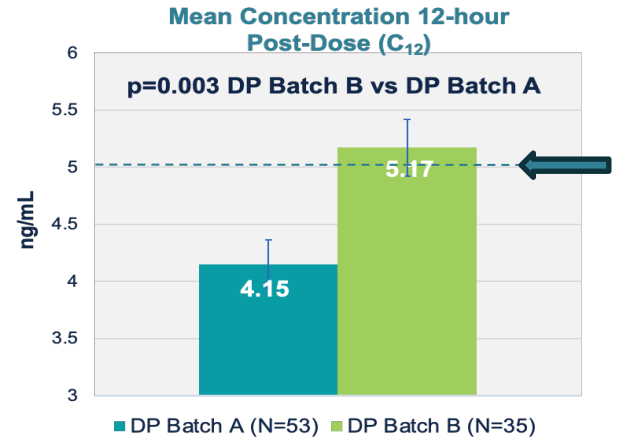
Neflamapimod Dosing Groups and Comparisons

- **Drug Product (DP) Batch A:** Due to batch- to-batch variability in polymorphic crystal forms, did not achieve expected plasma drug concentrations for 40mg TID.
- **DP Batch B:** Achieved the expected plasma drug concentrations for 40mg TD.
- **Comparisons:** (1) Placebo vs DP Batch A during placebo-controlled period (specified in Statistical Analysis Plan, SAP); (2) DP Batch B vs. DP Batch A during the extension (specified in Feb 2025 SAP Addendum)

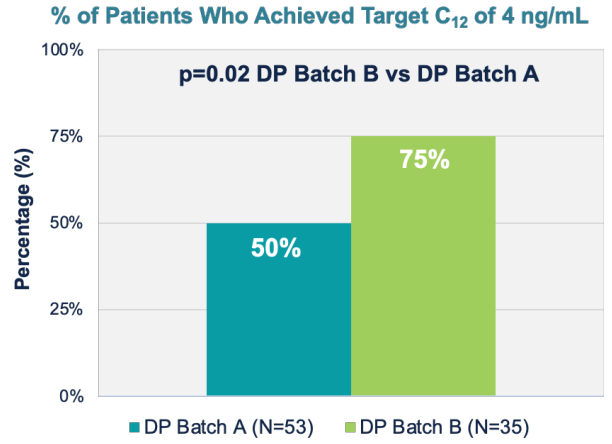
Participants were all aware that they were receiving neflamapimod in the Extension phase (*i.e.* treatment was "open label"), but neither they nor study site personnel were aware if they were receiving DP Batch A or DP Batch B capsules

Pre-planned introduction of 2nd batch of neflamapimod (DP Batch B) achieved expected and targeted plasma drug concentrations and enabled robust Extension Phase analyses





Expected mean for 40mg TID= 5.0 ng/mL



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

Primary outcome measure of the Phase 2b was mean change in CDR-SB; ADCS-CGIC was a key secondary endpoint and used to corroborate CDR-SB effect

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)

Alzheimer's Disease Cooperative Study-Clinical Global Impression of Change (ADCS-CGIC)

Blinded clinician's assessment of change in clinical status of patient from start of treatment	
Marked Improvement	+1
Moderate Improvement	+2
Mild Improvement	+3
Neutral	+4
Mild Worsening	+5
Moderate Worsening	+6
Marked Worsening	+7

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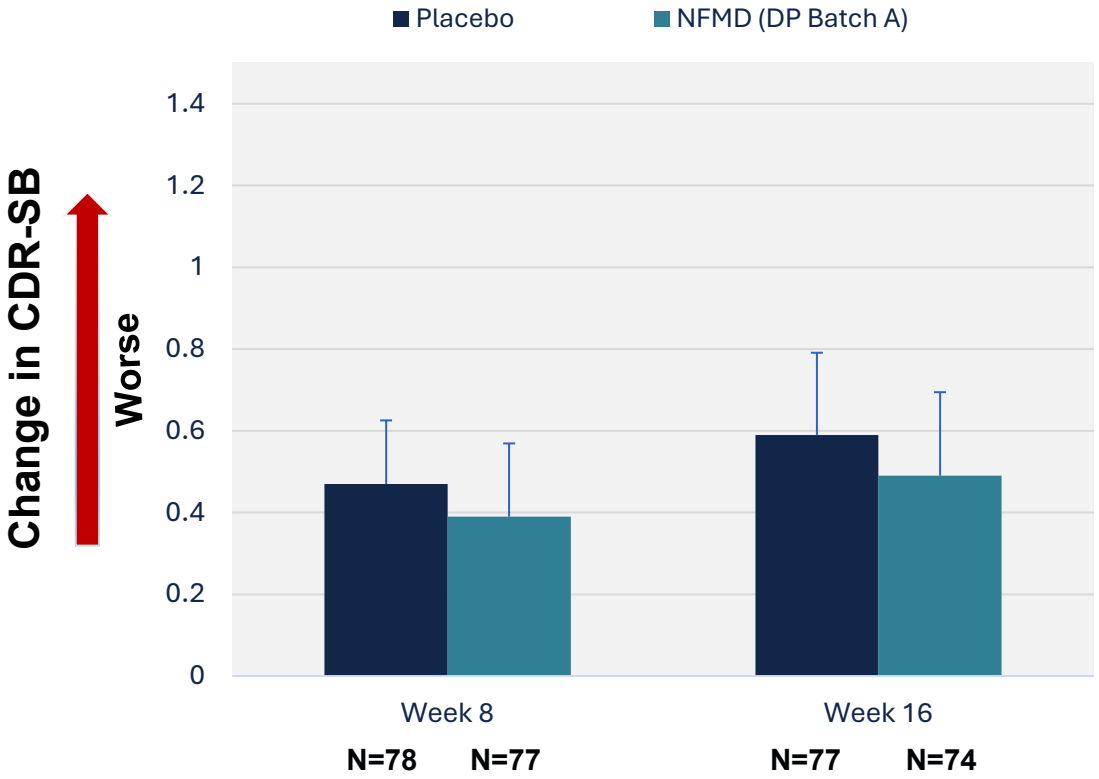
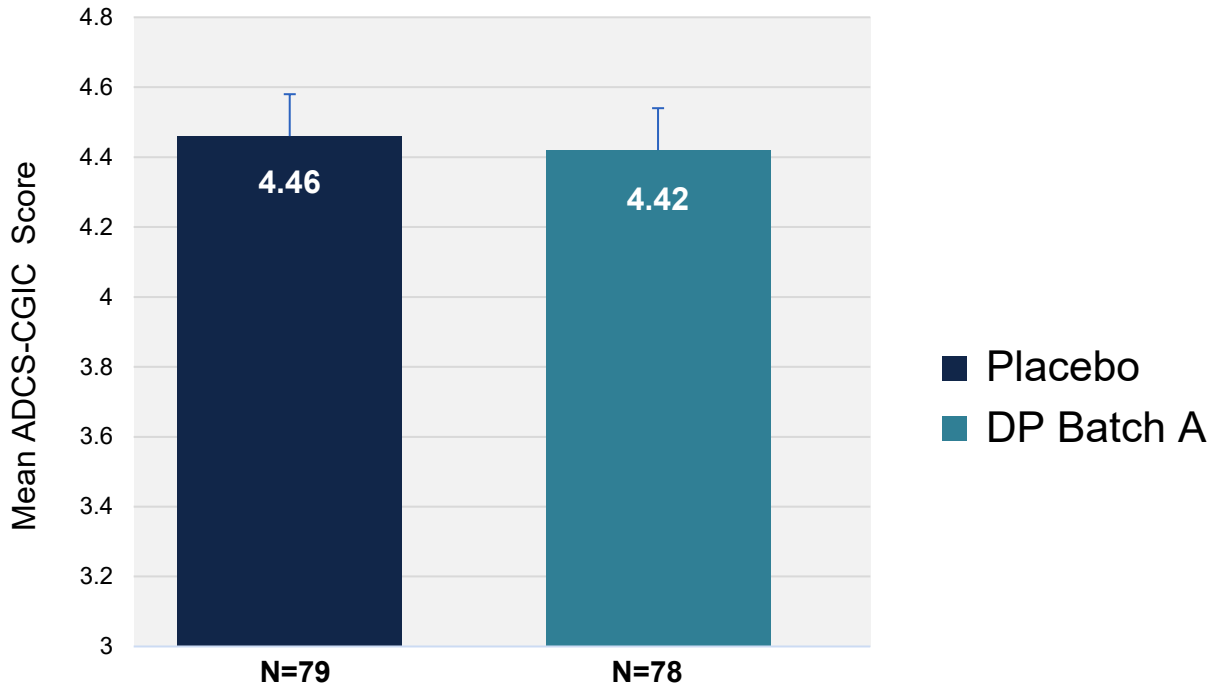
No significant differences between placebo and neflamapimod (DP Batch A) in mean change in CDR-SB or ADCS-CGIC during randomized phase

Randomized Phase (16 Weeks) Mean Change in CDR-SB



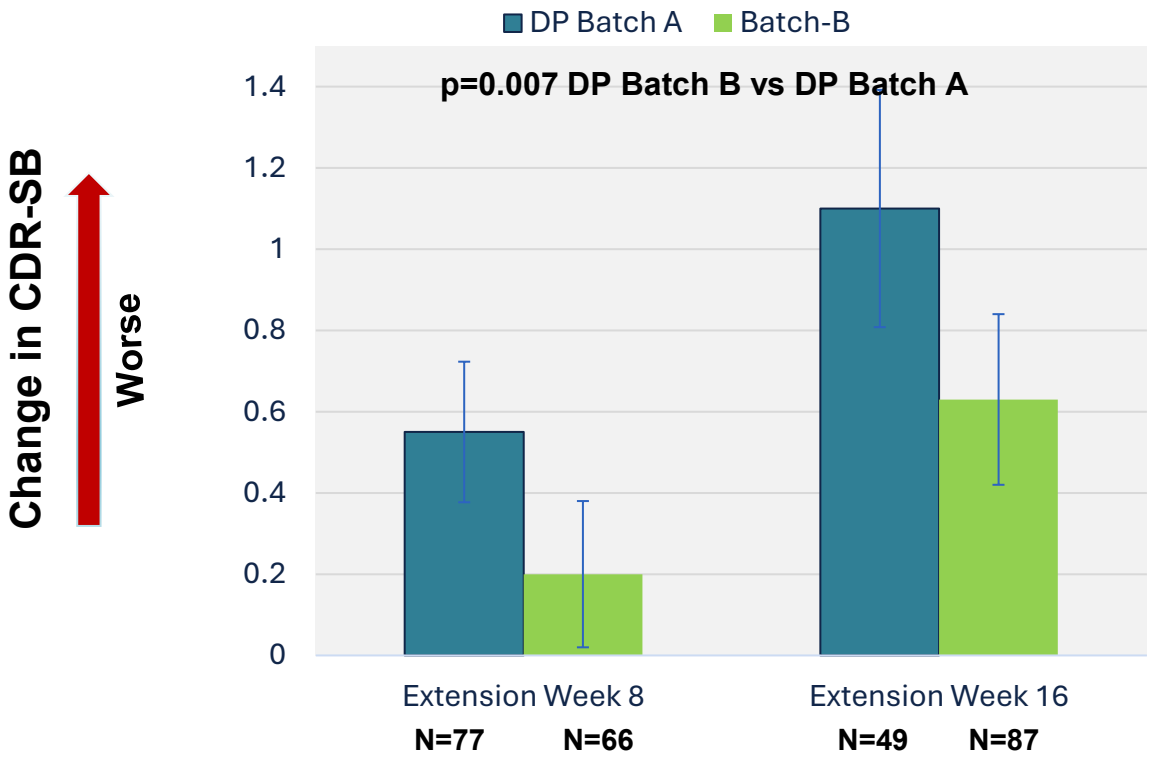
Randomized Phase Week 8, Mean ADCS-CGIC Score



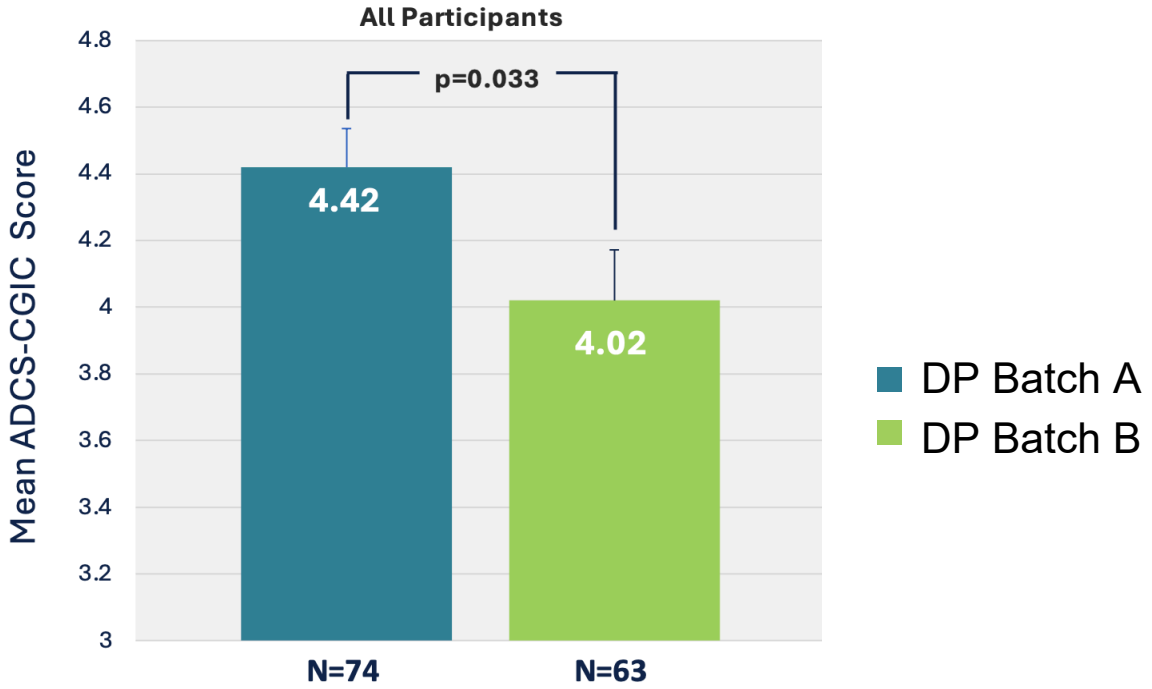
Linear Mixed-Effects Model for Repeated Measures (MMRM) with baseline CDR-SB, Sex, Age and Mini-Mental State Examination (MMSE) as covariates. DP Batch A data includes participants in Groups 1, 2, and 3 at Week 8 and in Groups 1 and 2 at Week 16. DP Batch B data includes participants in Groups 4 and 5 at Week 8 and Groups 3, 4, and 5 at Week 16. See Slide 16 for additional details.

Significant improvement demonstrated on mean change in CDR-SB and ADCS-CGIC, during first 16 weeks of the Extension Phase with DP Batch B

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



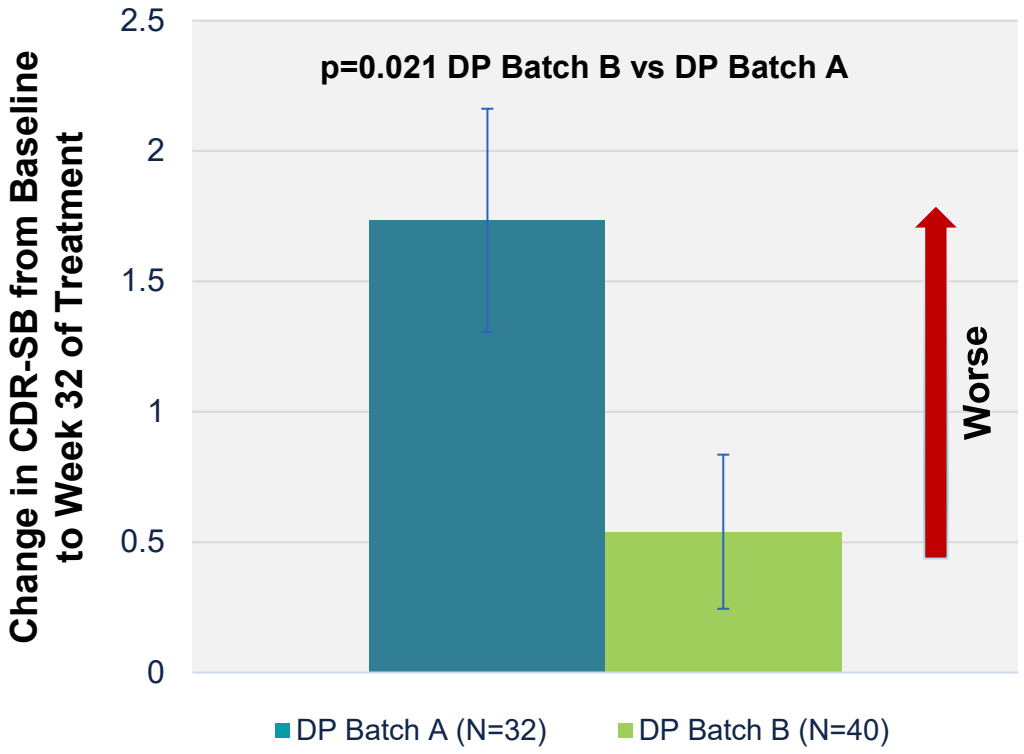
Extension Phase (Week 8), Mean ADCS-CGIC Score



Linear Mixed-Effects Model for Repeated Measures (MMRM) with baseline CDR-SB, Sex, Age and Mini-Mental State Examination (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. See Slide 16 for additional details.

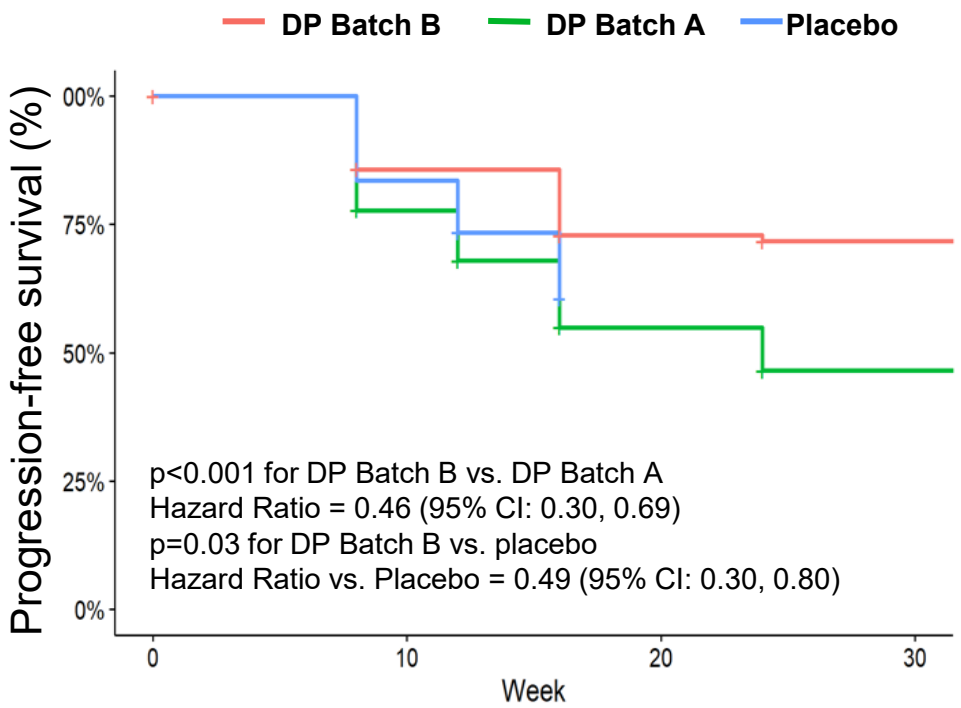
Clinical effect on CDR-SB with neflamapimod DP Batch B was durable out to 32 weeks of treatment

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. See Slide 16.

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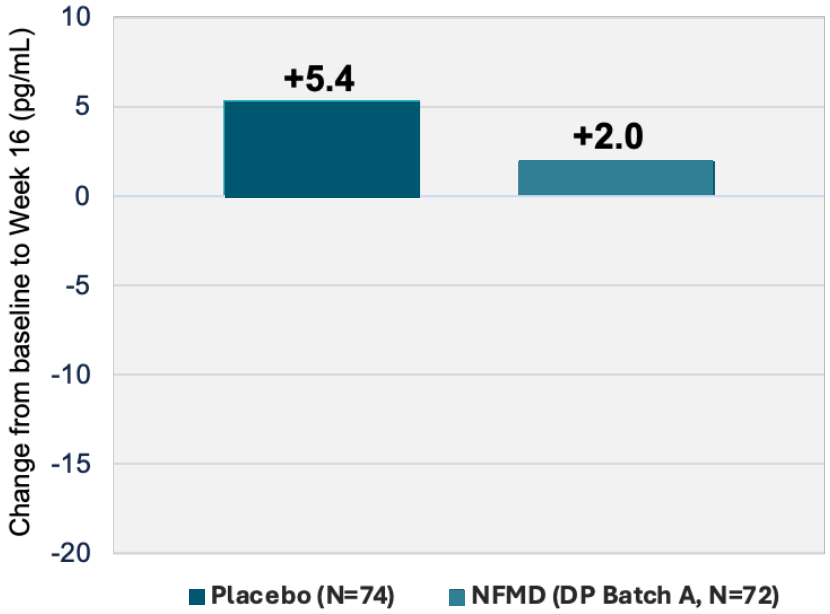
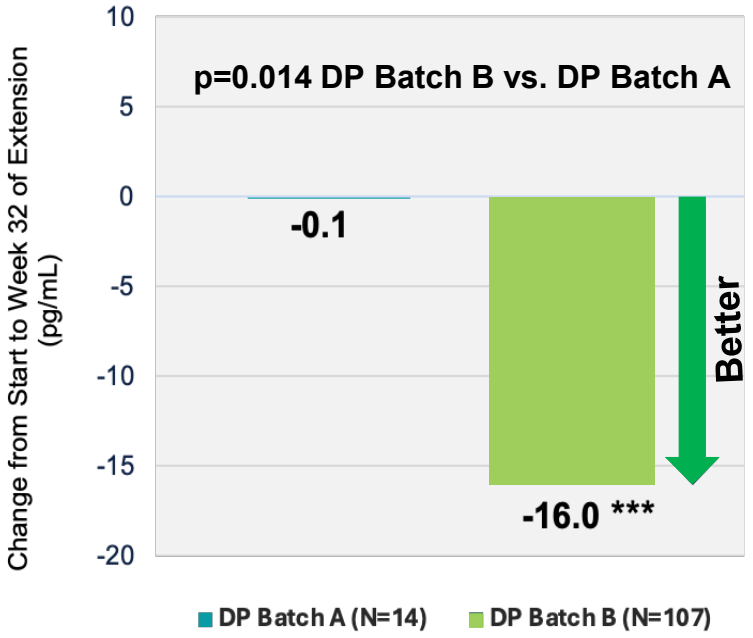
Neflamapimod DP Batch B achieved significant reduction in glial fibrillary acidic protein (GFAP), a key biomarker of neurodegenerative disease activity

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

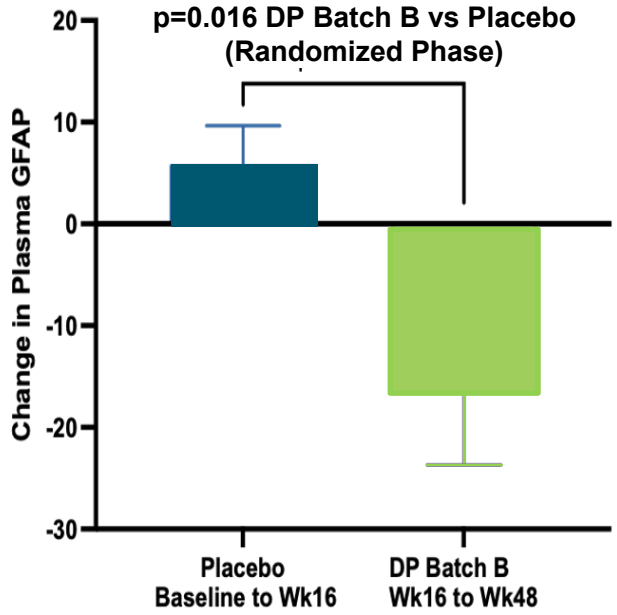


Median Change from Start to Week 32 of Extension



***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. See Group 16.

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Neflamapimod exhibited a favorable safety profile

Neflamapimod Was Well-tolerated with No New Safety Signals Identified

The incidence of discontinuation due to adverse events was low

- 4% with neflamapimod and 1% with placebo during the placebo-controlled phase
- 4% with DP Batch A and 2% with DP Batch B through to Week 16 of the Extension Phase

Low incidence of liver enzyme elevation (dose limiting toxicity of p38 inhibitors)

- 1 of 80 (1.3%) neflamapimod recipients discontinued because liver enzyme elevation during the placebo-controlled phase; event was reversible and not associated with bilirubin elevation
- 0 of 149 neflamapimod recipients discontinued for liver enzyme elevation during the Extension Phase

Only adverse event seen at greater than 10% incidence was falls

- 15% with neflamapimod and 19% with placebo during placebo-controlled phase
- 15% with DP Batch A and 7% DP Batch B through Week 16 of the Extension Phase

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Summary of the main results of the Phase 2b RewinD-LB study

- Once target plasma drug concentrations were achieved, neflamapimod demonstrated a meaningful effect on clinical progression over 16 weeks, as assessed by the CDR-SB and CGIC
- The effects on CDR-SB were durable out 32 weeks of treatment as assessed by both mean change in CDR-SB or risk of significant progression (≥ 1.5-point increase in CDR-SB)
- Neflamapimod also reduced plasma GFAP levels, a key marker of the neurogenerative disease activity, when target plasma drug concentrations were achieved
    - The effect size was substantial, with reductions relative to placebo corresponding to ~50% of the disease-specific elevation in plasma GFAP (i.e., the increase observed in DLB compared with historical healthy controls)
- Neflamapimod exhibited a favorable safety profile, including a very low rate (<1%) of discontinuation due to liver enzyme elevation

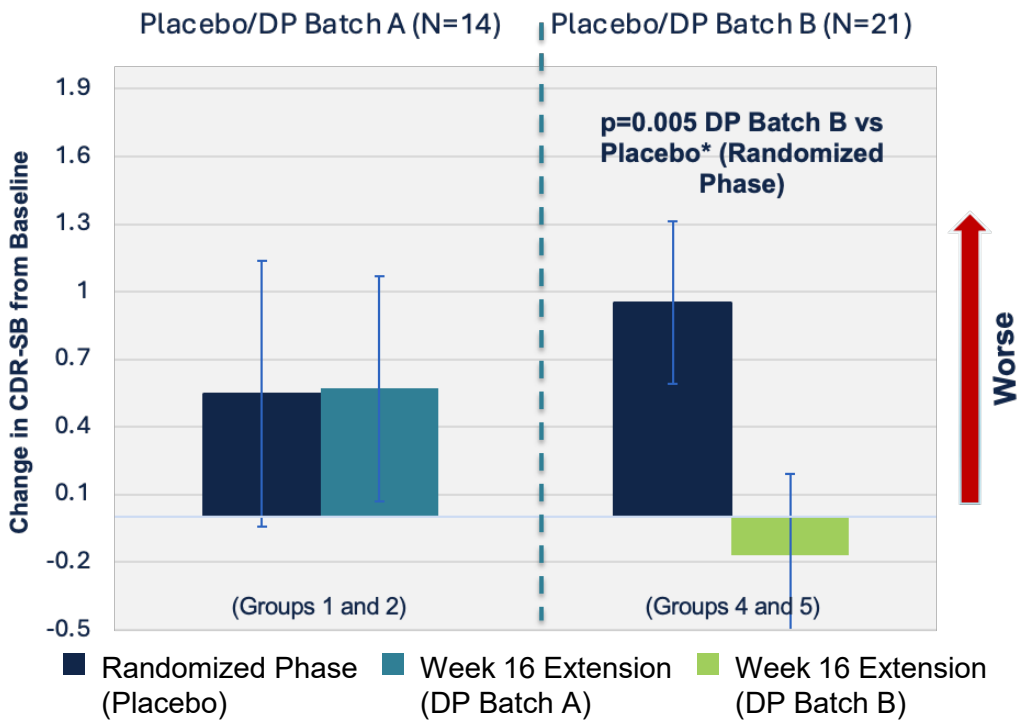
Phase 2b data and recent scientific literature suggests that lowering pTau181 cutoff excludes a greater percentage of DLB patients with AD co-pathology

Inclusion Criteria in RewinD-LB was <27.2 pg/mL[1]	At the time RewinD-LB was initiated (mid-2023), based on the limited data set available at that time, **27.2 pg/ml was estimated to be the optimal cutoff for AD co-pathology in patients with DLB**
Clinical Data Demonstrates Accentuated Effect with More Stringent Cutoffs	**Pre-specified analyses** of the Extension Phase data in RewinD-LB, conducted in March 2025 (and presented at AAIC in July 2025), **indicated that lower cutoff points (<25.2, <23, or <21) led to progressively greater treatment effect** size for CDR-SB and CGIC
New Third-Party Research Supports Lower Cut-Off	21 pg/mL as a cutoff was further confirmed in a large (N=1298), third-party validation study, published in June 2025[2], that indicated that a **pTau181 cutoff of 21 pg/mL was the high sensitivity cutoff for AD pathology** in AD and non-AD dementia by CSF criteria

1. Scale reported based on Quanterix v2.1 assay, which utilizes a different standard than prior version of assay (v2.0). Quantitative values for v2.1 are approximately ten-fold higher than corresponding values on v2.0 scale. 2. Doecke et al. 2025, Alzheimer's & Dement, 2025;21:e14573

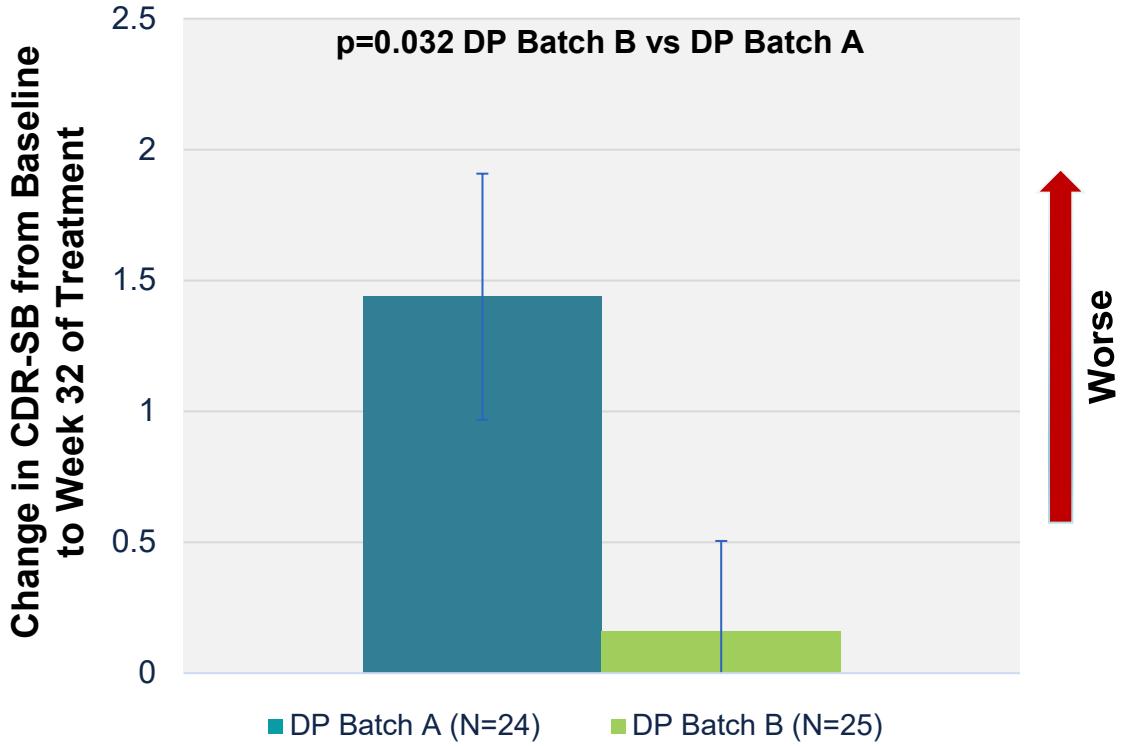
Neflamapimod demonstrated >1-point mean change in CDR-SB in Phase 2b in < 21 pg/mL pTau181 subset

Mean Change in CDR-SB vs. Placebo in Within-Subject Comparison



Difference to Placebo: -1.12, when targeted plasma drug concentration achieved

Mean Change in CDR-SB After 32 Weeks of Treatment



Difference: -1.20, representing an 89% reduction in CDR-SB change when targeted plasma drug concentration achieved

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. See Slide 16.

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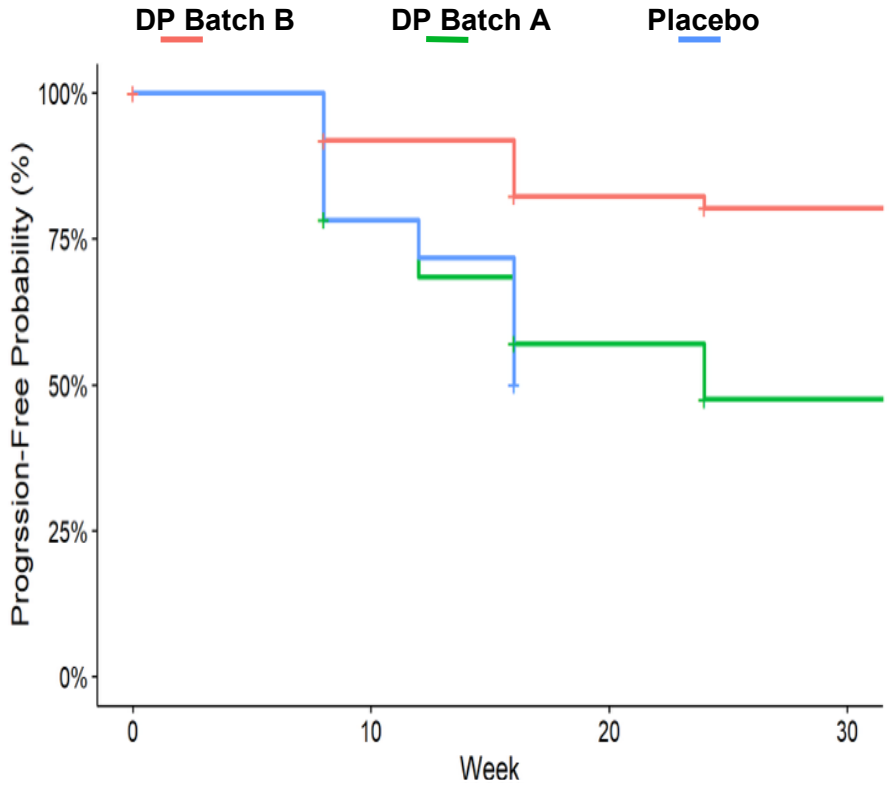
Risk of clinically meaningful progression (≥ 1.5-point increase in CDR-SB) and time to progression in < 21 pg/mL pTau181 subset

Participants with Screening Plasma pTau181 < 21 pg/mL



Number at Risk

	Week 8	Week 16	Week 24
DP Batch B	74	67	41
DP Batch A	69	42	18
Placebo	46	33	

Risk of Progression			
	Hazard Ratio (95% confidence interval)	**% Reduction in Risk**	**P-value**
DP Batch B vs. DP Batch A	0.33 (0.19, 0.58)	67%	<0.001
DP Batch B vs. Placebo	0.25 (0.13, 0.47)	75%	<0.001

Median Time to Progression	
Placebo	16 Weeks
DP Batch A	24 Weeks
DP Batch B	Not Reached (Projected: ~1.5 years)

Kaplan-Meier survival curve analysis utilizing data from both placebo-controlled and extension phase. DP Batch B, DP Batch A, and placebo data include all participants on such treatment at the time of measurement (i.e., participants may change groups over time).

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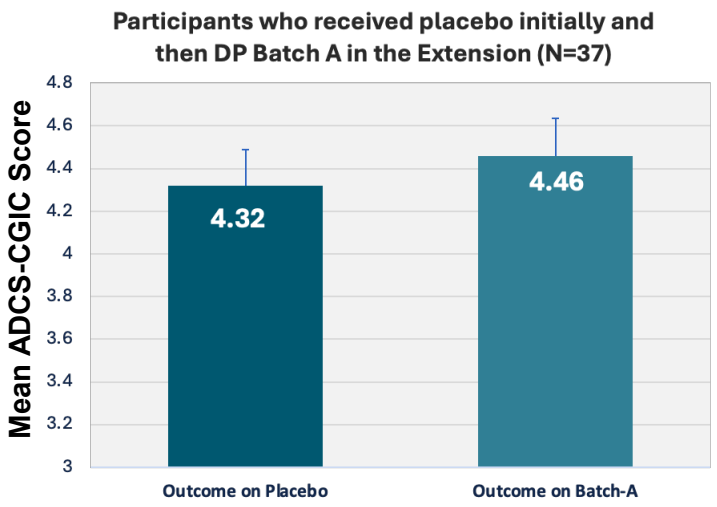
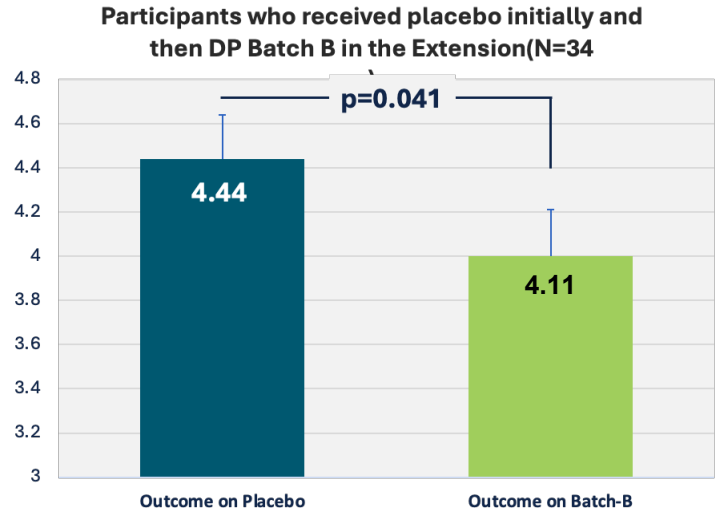
DP Batch B effects on ADCS-CGIC in <21 pg/mL pTau181 subset at 8 weeks

All Participants

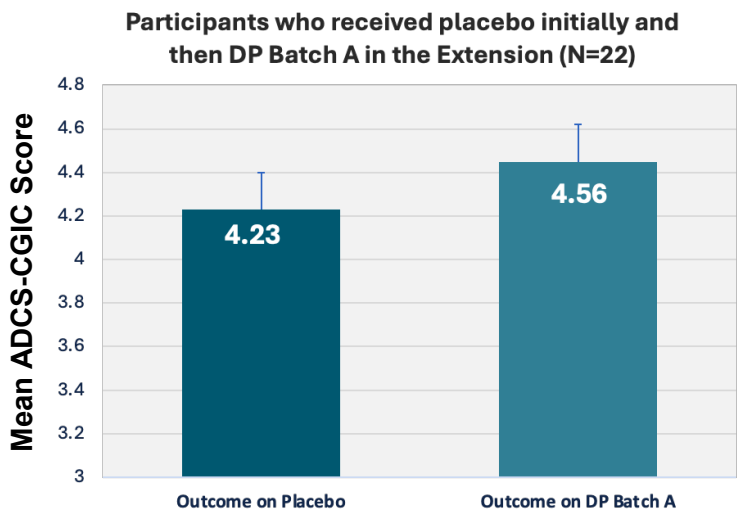
Target Concentration Not Achieved

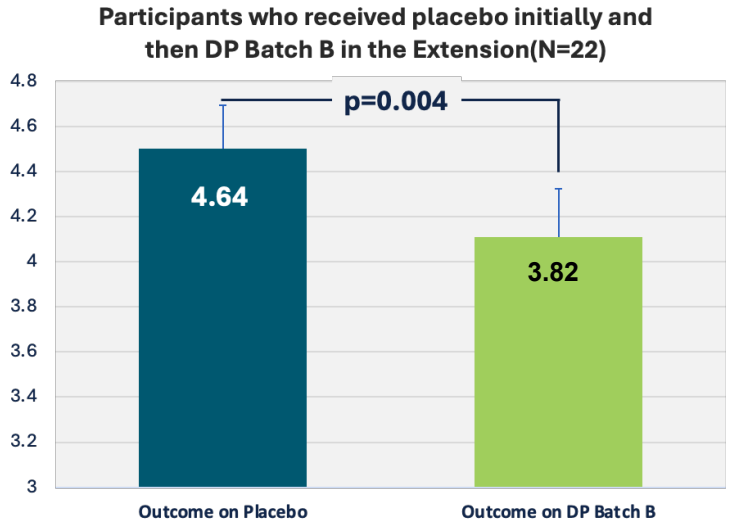


Target Concentration Achieved



Participants pTau181 <21 pg/mL at screening





Difference = -0.82
95% CI: -1.33,-0.33
p=0.004

P-values from Wilcoxon Rank Sum Test. DP Batch A data includes participants in Groups 1, 2, and 3 at Week 8 of Extension Phase. DP Batch B data includes participants in Groups 4 and 5 at Week 8 of Extension Phase. See Slide 16.

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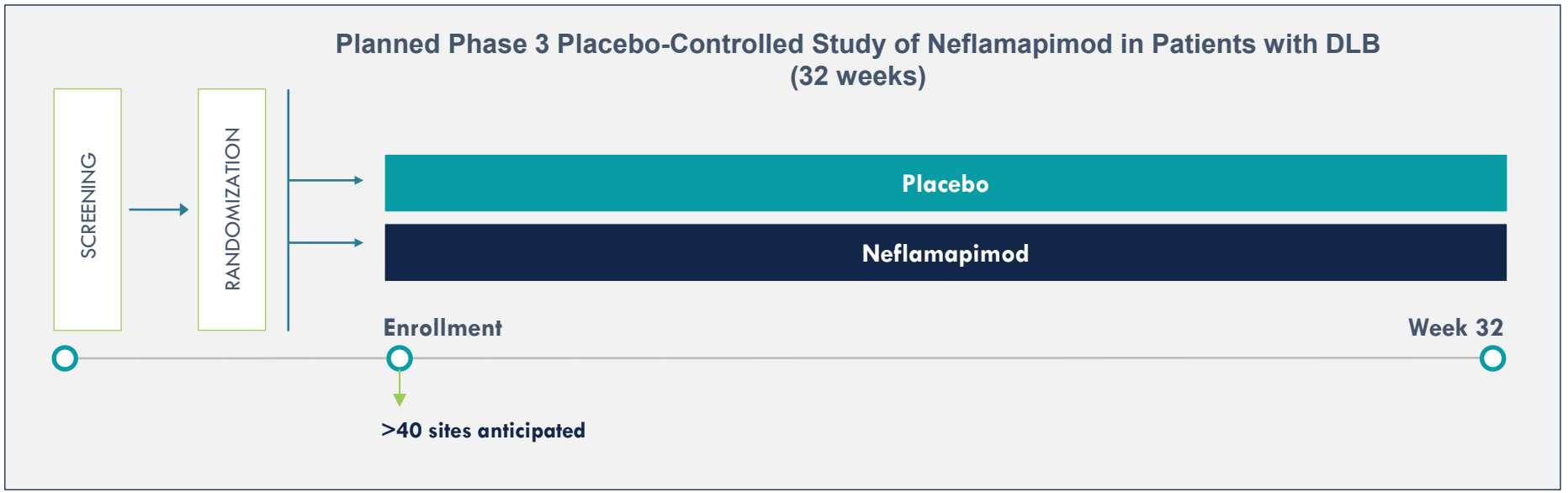
Reached alignment with the FDA on registration path for potential approval in dementia with Lewy bodies



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
- Plan to initiate the trial in 2H2026[1] after obtaining feedback from global regulatory authorities and completing activities to support improvements in drug product formulation

1. *Subject to the Company's receipt of sufficient funding.*

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Plasma drug concentration differences across DP batches in RewinD-LB attributed to presence of distinct crystal forms

	DP Batch A	DP Batch B
Use in RewinD-LB	Placebo-controlled phase (Part A) and extension phase (Part B)	Extension phase (Part B) only
Date of Production	October 2020 (3-4 yrs old during utilization in RewinD-LB)	March 2023 (~2 yrs old during utilization in RewinD-LB)
Median trough plasma drug concentrations	4.0 ng/mL; lower than expected, with only 50% reached individual target concentration of 4 ng/mL	5.0 ng/mL; attained targeted and expected plasma drug concentration for 40mg TID

DP Batch A and DP Batch B manufacturing processes were identical

- Historic manufacturing process created drug substance containing multiple solid-state forms (i.e., polymorphs) of neflamapimod
- Polymorphs exhibited diverse properties:
  - One polymorph demonstrated high stability but lower-solubility, which leads to lower bioavailability
  - Other polymorphs demonstrated lower stability but higher-solubility
- During long-term storage, less stable, higher-solubility polymorphs progressively converted to a stable, lower-solubility form
- Conversion to more stable, lower-solubility form of DP Batch A led to sufficient polymorph conversion and lower plasma drug levels in RewinD-LB trial

***A clear path forward**: we have developed a controlled process to manufacture only the stable crystal form and have increased the dosage strength to ensure achievement of the plasma drug concentration achieved with DP Batch B*

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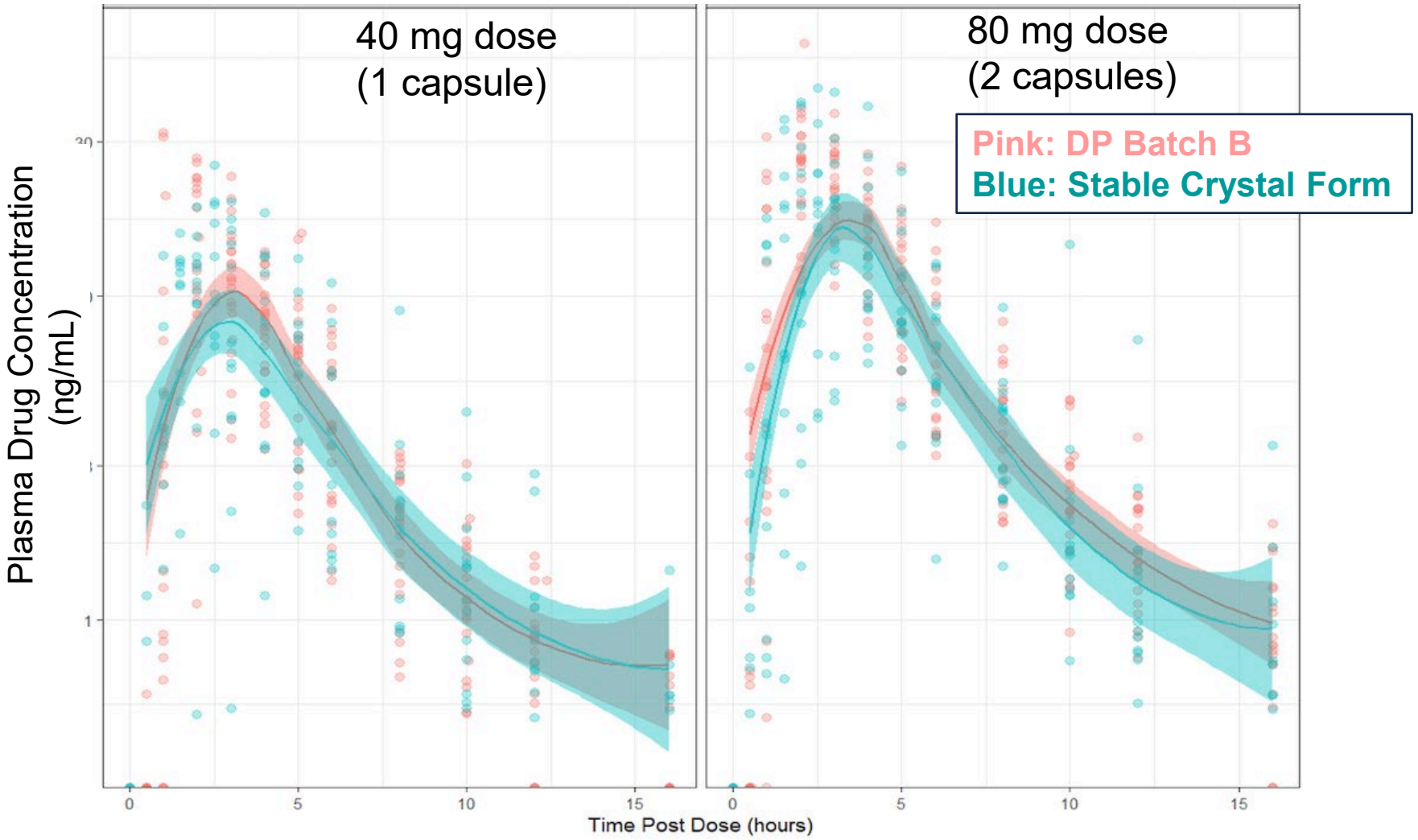
New, Stable Crystal Form v. DP Batch B: Comparison of 40mg Capsule PK Profiles

- Pharmacokinetic profiles of DP Batch B and the new, stable crystal form of neflamapimod are largely overlapping
- To ensure that the stable crystal form achieves the plasma drug concentrations seen with DP Batch B, the dose will be increased to 50 mg TID in the Company's planned Phase 3 trial
- With 50mg TID of the stable crystal form, expect ~80-90% of participants in Phase 3 will achieve individual patient C_{trough} target of 4 ng/mL

Mean (95% CI) and Individual Profiles After Single Doses of one or two 40mg capsules



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CervoMed is advancing neflamapimod as a potential first-in-class therapy for the treatment of dementia with Lewy bodies

Well documented scientific rationale and clinically validated mechanism of action

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

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

Alignment achieved with FDA on registration path in DLB

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Corporate Background

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

NON-EXECUTIVE DIRECTORS

Joshua Boger, PhD (Chair)
Executive Chair, Alkeus Therapeutics.
Founder, former CEO, Vertex Pharmaceuticals

Sylvie Gregoire, PharmD
Co-Founder; Board member, Abivax, F2G;
Former Executive VP, Biogen; Former President, HGT Division, Shire Pharmaceuticals;
Former Board member Novo Nordisk, Revitty, ViFor, Corvidia, Cubist

Jeff Poulton (Chair of Audit Committee)
CFO, Alnylam Pharmaceuticals (Nasdaq:ALNY)
Former CFO, Shire Pharmaceuticals; CFO, Indigo Agr.

David Quigley
Former Senior Partner McKinsey & Company;
Served as Global Head of Private Capital, North America Head of Life Sciences, and Global Lead of Life Sciences Commercial practices

Jane H. Hollingsworth, JD
Managing Partner, Militia Hill Ventures
Former Chairman of the Board, Diffusion Pharmaceuticals

Marwan Sabbagh, MD
Prof. of Neurology at the Alzheimer's and Memory Disorders division of the Barrow Neurological Institute at Dignity Health/St Joseph's Hospital in Phoenix, Arizona

Frank Zavrl
Former Board Member, Puma Biotechnology
Retired Partner, Adage Capital

SCIENTIFIC ADVISORS

Ole Isacson, MD (Chair)
Prof of Neurology (Neuroscience) Harvard Medical School

Lewis Cantley, PhD
Professor of Cell Biology, Harvard Medical School, Dana-Farber Cancer Institute; Laureate, Breakthrough Prize in Life Sciences

Heidi McBride, PhD
Professor, Dept. of Neurology & Neurosurgery, McGill University

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

John Alam, MD

President, CEO & Co-Founder, Director

Former Chief Medical Officer and EVP Medicines Development, Vertex
Former Global Head Alzheimer's R&D at Sanofi
Led clinical development of Avonex for multiple sclerosis at Biogen.
MD from Northwestern U. Medical School. S.B. Chemical Engineering MIT. Internal Medicine Residency at Brigham and Women's Hospital. Post-Doc at Dana-Farber Cancer Institute.

William Elder

Chief Financial Officer & General Counsel

Over a decade of experience working with biotechnology companies on capital markets and other corporate transactions; General Counsel of Diffusion Pharmaceuticals (2020-23); J.D. from University of Pennsylvania School of Law, M.S. Finance from Villanova University, B.A. Economics from Tufts University

Matthew Winton, PhD

Chief Commercial and Business Officer

Before joining CervoMed, Dr. Winton served as Chief Operating Officer at Inozyme Pharma. Previously, he held senior leadership roles at Biogen, including Senior Vice President and Head of the U.S. Multiple Sclerosis Franchise and Vice President and Head of the U.S. Spinal Muscular Atrophy Franchise. Ph.D. in Neuroscience from the Université de Montreal, M.B.A. from Boston University, and B.Sc. in Biology & Psychology from York University. Dr. Winton also completed a postdoctoral fellowship at the Center for Neurodegenerative Disease Research (CNDR) at the U. of Pennsylvania

Kelly Blackburn, MHA

EVP, Clinical Development

Former VP, Clinical Affairs at aTyr Pharma; VP, Clinical Development Operations at Vertex. Led global clinical operations for Kalydeco® for the treatment of cystic fibrosis, Incivek® for hepatitis C, and Velcade® for multiple myeloma

Mark De Rosch, PhD, FRAPS

EVP, Regulatory and Government Affairs, and Program Management

30+ years in industry. Former COO at Aura Biosciences; Chief Regulatory Officer at Epizyme; SVP, Regulatory, Quality and Medical Writing at Nightstar Therapeutics; SVP, Regulatory, Quality and CMC at Akebia Therapeutics. Before that, Dr. De Rosch served in roles of increasing responsibility at several life science and healthcare consulting firms

Marco Verwijs, PhD

EVP, Technical Operations

Nearly 20 years of product development and manufacturing experience, including as CTO at Adipo Therapeutics, CTO at Aerovate Therapeutics and senior management positions at Vertex, Flexion Therapeutics and Epizyme

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