



CERVOMED

Medicines for the Brain

Corporate Presentation

October 2026

NASDAQ: CRVO

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, its potential impact on the rate of disease progression and/or clinical worsening, the optimal dosing regimen to achieve therapeutic effects, or any other treatment effects observed in any clinical trial on any clinical, biomarker, or other outcome measure, including initial biomarker from the Company's Phase 2a trial in nfvPPA; 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 or the impact thereon of the Innovation Passport Designation or any other regulatory designation. 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 Company's ability to maintain the intellectual property protection afforded by the Company's patent portfolio; 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 October 1, 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.

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

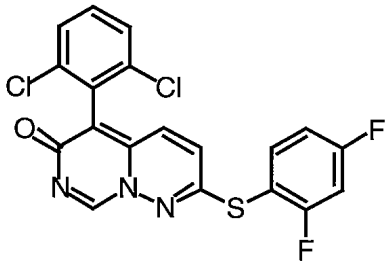


Leader in developing the first specific therapy for dementia with Lewy bodies (DLB), while expanding clinical program into nonfluent variant primary progressive aphasia (nfvPPA) and ALS



Experienced executive leadership team, with strong board of directors and advisors

Neflamapimod is an oral, brain-penetrant, selective p38 α inhibitor and the most advanced disease modifying drug candidate for DLB



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-1 β 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 >550 volunteers and patients**, with up to 48 weeks treatment duration
- Chronic, repeat dose toxicology studies completed
- Favorable PK profile of new **50mg oral tablet**, allows for **increased plasma drug exposure**, while **limiting additional safety risk**.
- FDA **lifted the PK exposure limit**, following confirmation of a higher NOAEL in a 39-week dog toxicology study

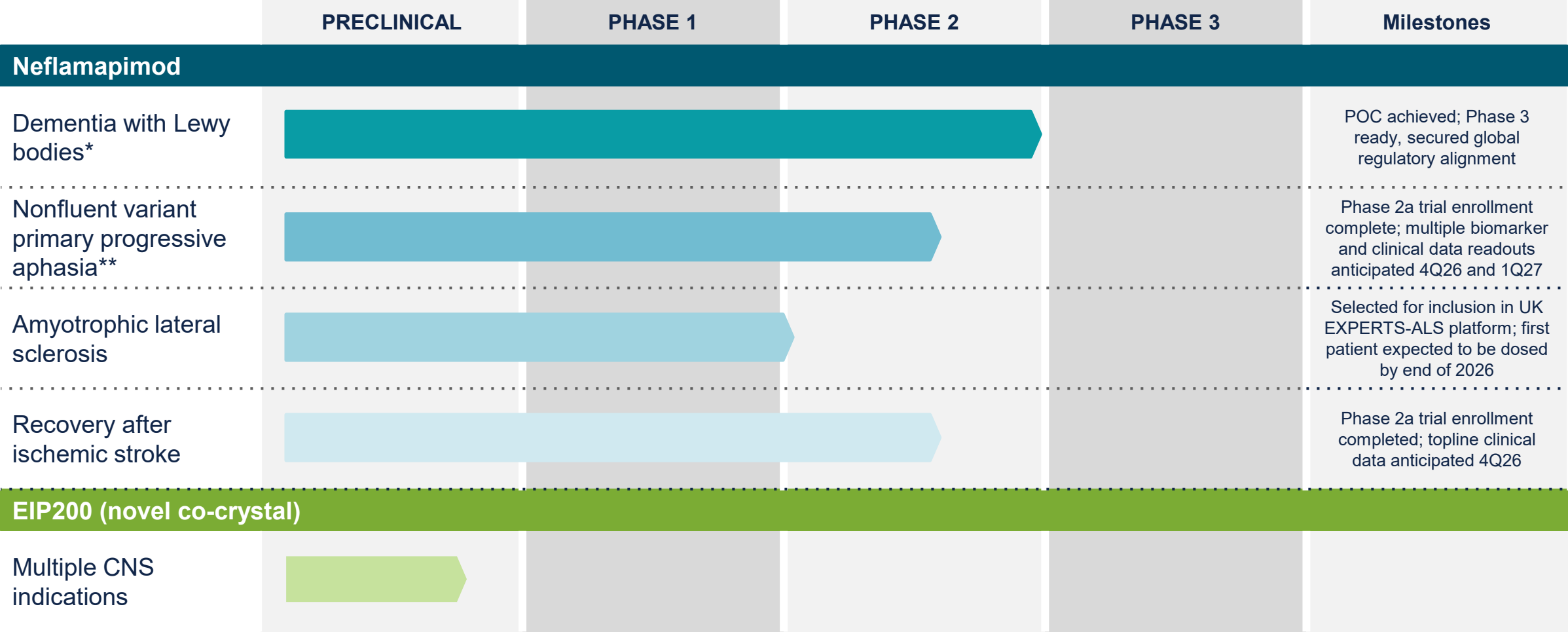
Clinical activity demonstrated in DLB

4

- Across two Phase 2 studies, shown:
 - ✓ **Improvement** in key **clinical endpoints**, including CDR-SB,
 - ✓ **Reduction** in a plasma neurodegeneration **biomarkers** (e.g., GFAP)
 - ✓ **Increase in brain volume and functional connectivity** by MRI



Neflamapimod is a late-stage clinical asset being evaluated in multiple high unmet need neurological diseases



*Received FDA Fast Track designation; **Received FDA Orphan Drug designation

The Opportunity for Neflamapimod in Dementia with Lewy Bodies



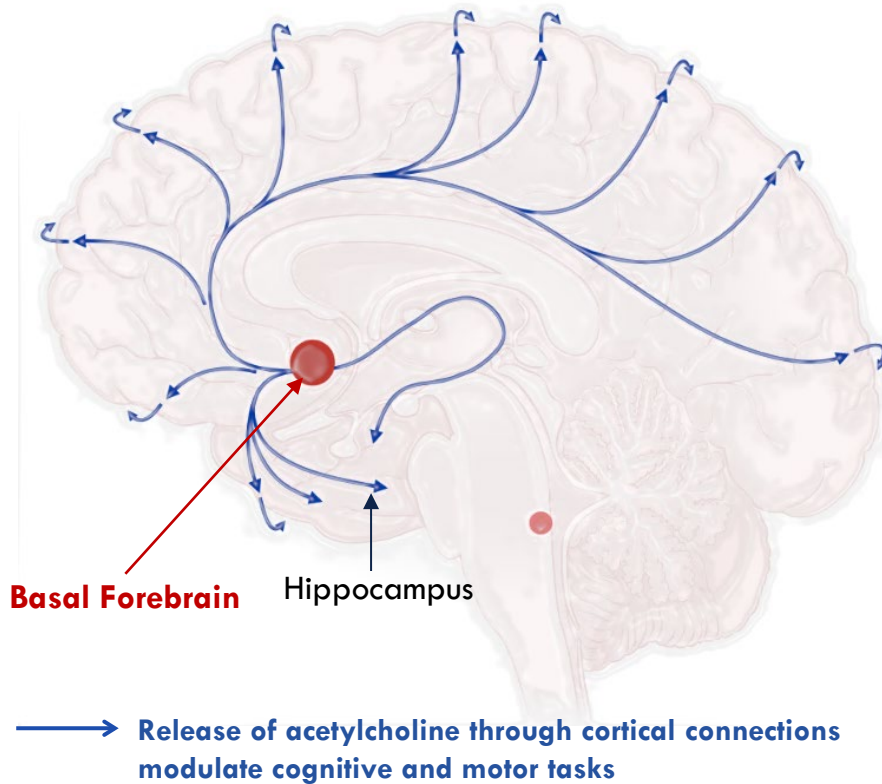
About dementia with Lewy bodies (DLB)



- High unmet clinical need
 - Second most common neurodegenerative dementia
 - Significant impact on quality of life and caregiver burden
 - Associated with greater functional disability and higher economic costs than Alzheimer's disease (AD)
 - Progresses more rapidly than AD, with average time from diagnosis to requiring nursing home care being ~2 years
- Progressive α -synucleinopathy characterised by widespread cortical and subcortical Lewy bodies
 - AD co-pathology is common; present in up to ~50% of all DLB patients
- Clinical diagnostic criteria are highly specific (>90%)
- In early stages, a major driver of disease expression and progression is dysfunction and degeneration of basal forebrain cholinergic neurons

Dysfunction and degeneration of basal forebrain cholinergic neurons is a key pathologic driver of disease expression in DLB

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 consistently documented in DLB
- **Mechanistically validated therapeutic target**, where restoring cholinergic function offers potential for broad clinical impact

Disease processes in basal forebrain are reversible

In DLB, AD co-pathology affects severity of disease and treatment response to therapy that targets basal forebrain cholinergic degeneration

Absence of AD Co-Pathology = Low Levels of Plasma pTau181

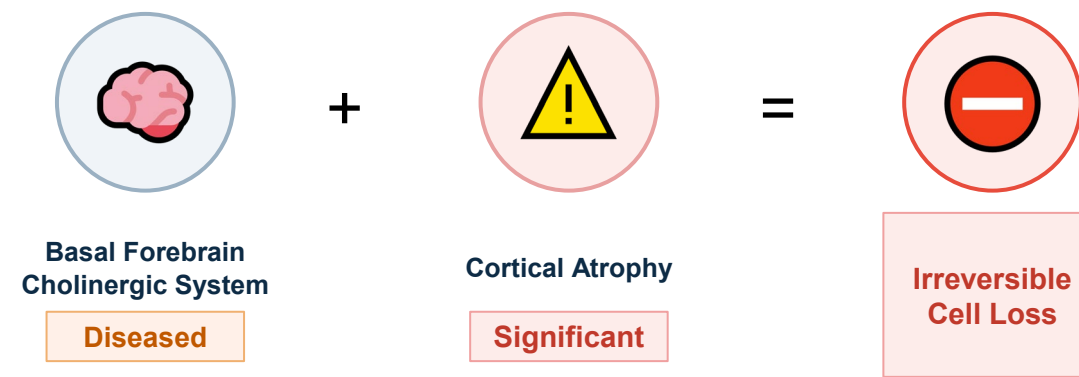


Clinical Effect of Basal Forebrain-Directed Treatment

With cholinergic dysfunction as the primary driver of disease progression targeting the basal forebrain can slow clinical decline

VS

Presence of AD Co-Pathology = Elevated Levels of Plasma pTau181

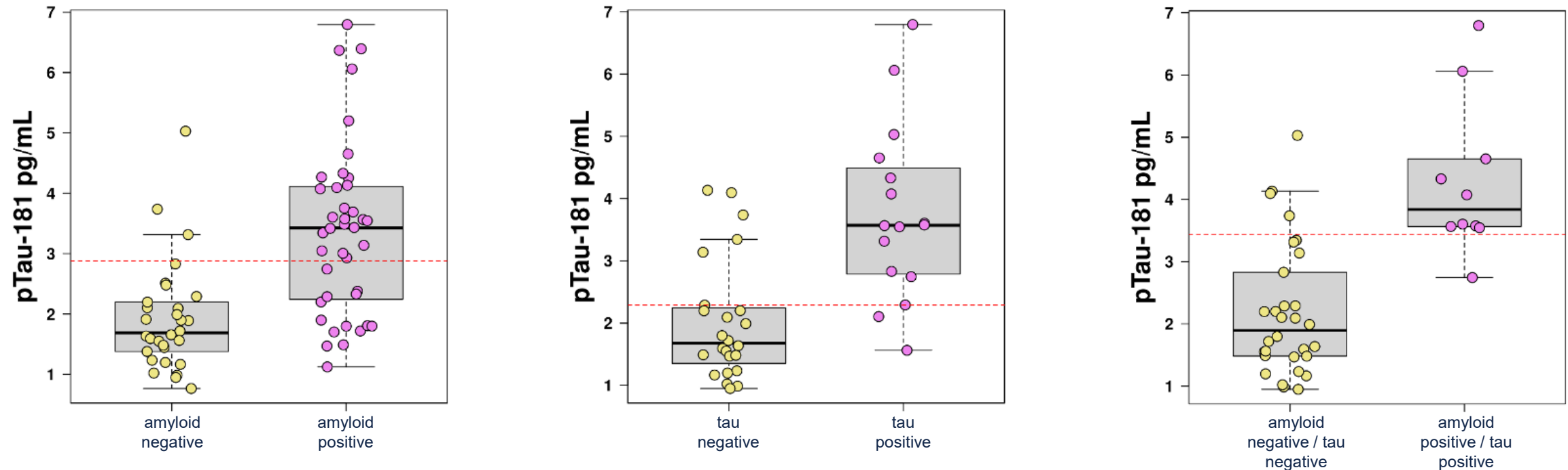


Clinical Effect of Basal Forebrain-Directed Treatment

In the **presence of AD co-pathology**, significant cortical atrophy from AD becomes the primary driver of disease progression, **resulting in irreversible deficits** and limiting the effectiveness of targeting the basal forebrain

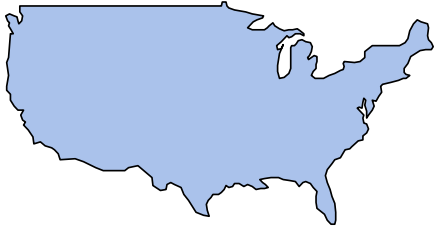
Plasma levels of phosphorylated Tau (pTau) to identify and exclude patients with DLB who also have AD co-pathology in clinical trials

PET Scan Results



- DLB patients with AD co-pathology have elevated levels of pTau181, a specific form of plasma pTau
- pTau blood tests obviate the need for PET scans or spinal taps to identify amyloid and/or tau pathology
- Sensitivity and specificity of test to identify AD pathology dependent on the cutoff utilized

High-value, untapped commercial potential in DLB without AD co-pathology



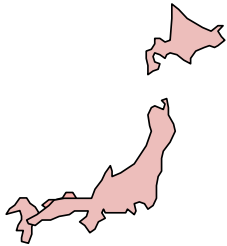
150,000–180,000 diagnosed DLB patients w/o AD co-pathology in US

- ~6M individuals with dementia¹
- 5-6% of all cases are due to DLB²
- ~300,000 –360,000 diagnosed total DLB pts.



~280,000-420,000 diagnosed DLB patients w/o AD co-pathology in EU

- ~14M individuals with dementia³
- 4-6% of all cases are due to DLB
- ~ 560,000 – 840,000 diagnosed total DLB pts.



~100,000-150,000 diagnosed DLB patients w/o AD co-pathology in Japan

- ~5M individuals with dementia⁴
- 4-6% of all cases are due to DLB⁵
- ~200,000 – 300,000 diagnosed total DLB pts.

Current Treatment Landscape

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



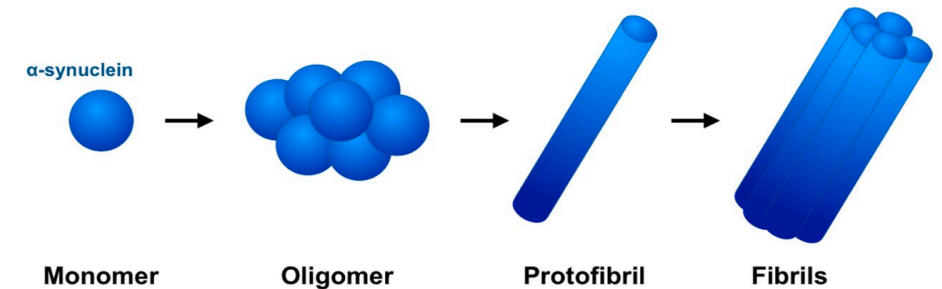
Interplay of α -synuclein and neuroinflammation is a critical pathogenic driver of DLB / basal forebrain cholinergic dysfunction and degeneration

Key Evidence: α -synuclein

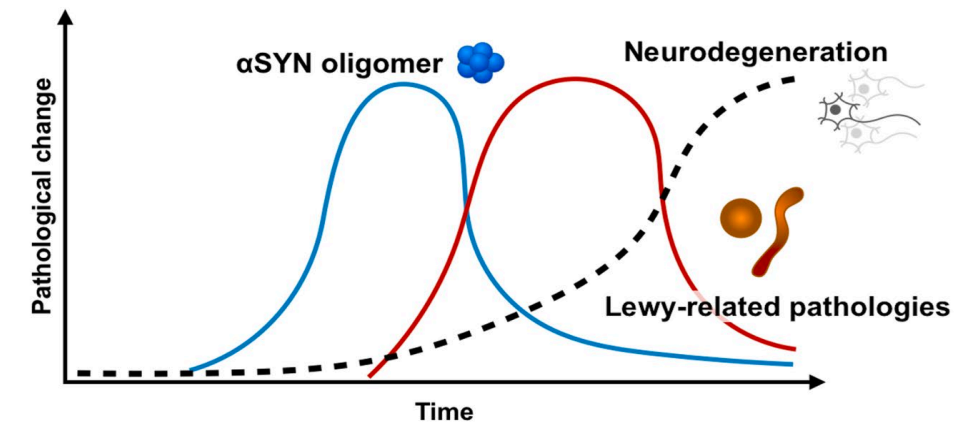
- ✓ APP and α -synuclein additively and selectively lead to basal forebrain cholinergic neuronal loss/pathology^{1,2,3,4}
- ✓ Lewy body pathology is the primary driver of basal forebrain cholinergic atrophy in human studies, with basal forebrain atrophy as the MRI signature of CSF α -syn-positive dementia^{5,6}
- ✓ Axonal transport defects associated with α -synuclein are mediated by p38 α , and inhibitors correct axonal transport defects⁷
- ✓ Overexpression of α -synuclein disrupts APP and endo-lysosomal axonal trafficking in a mouse model of synucleinopathy⁸
- ✓ Rab5 abnormalities involved in neurotoxicity on α -synuclein^{9,10}
- ✓ Toxicity and propagation of α -synuclein exacerbated by IL-1 β ^{11,12}
- ✓ p38 α inhibition decreases synaptic degeneration in an α -synuclein induced mouse model of DLB/PD¹³

Pathological Mechanism¹⁴

(A) Aggregation process of α -synuclein (α SYN)



(B) Temporal transition of α SYN aggregates (hypothesis)

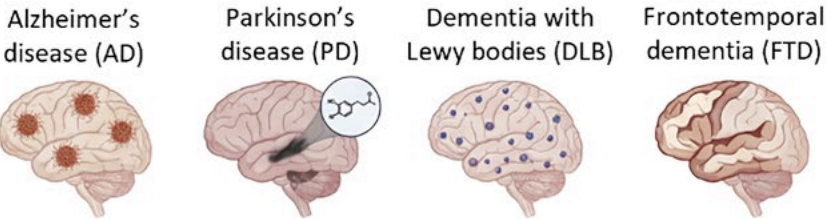


Recent, robust scientific evidence implicates cytokine (IL-1 β , IL-6) signaling as a key therapeutic target for DLB

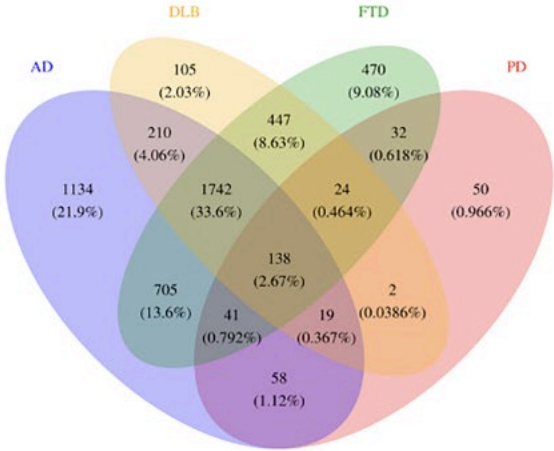
1. Biofluid samples



2. Neurodegenerative diseases



3. Proteomic Analysis Results



Authors

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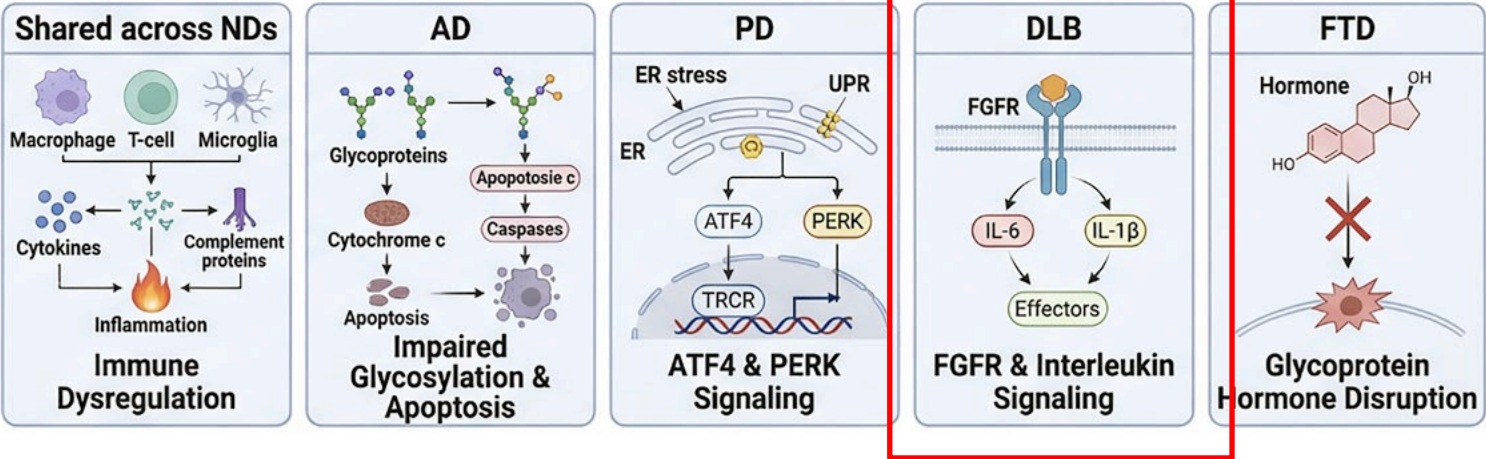
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In brief

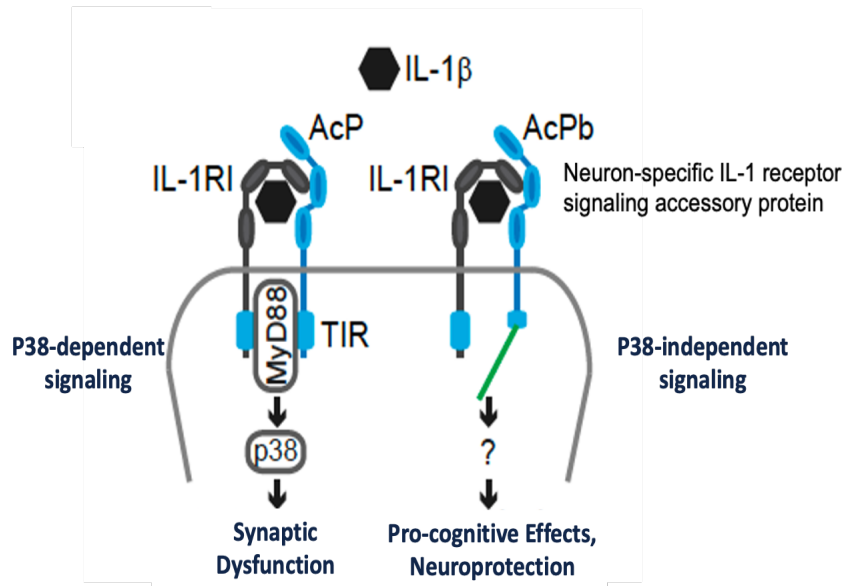
Ali et al. analyze over 5,700 cerebrospinal fluid and plasma samples across Alzheimer's disease, Parkinson's disease, dementia with Lewy bodies, and frontotemporal dementia. They identify shared immune dysregulation and distinct pathway alterations, including glycosylation and interleukin signaling, and develop predictive models that accurately classify each disorder, highlighting biomarkers and pathways for therapeutic targeting.

4. Pathways & Mechanisms

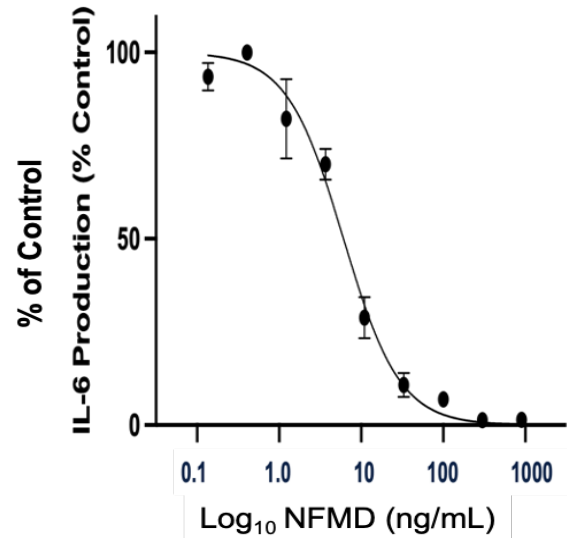


In both *in vitro* and in patient studies, neflamapimod potently blocked IL-1 β signaling and IL-6 production

In neurons, p38 α inhibition blocks deleterious IL-1 β signaling pathway, while sparing neuroprotective pathway

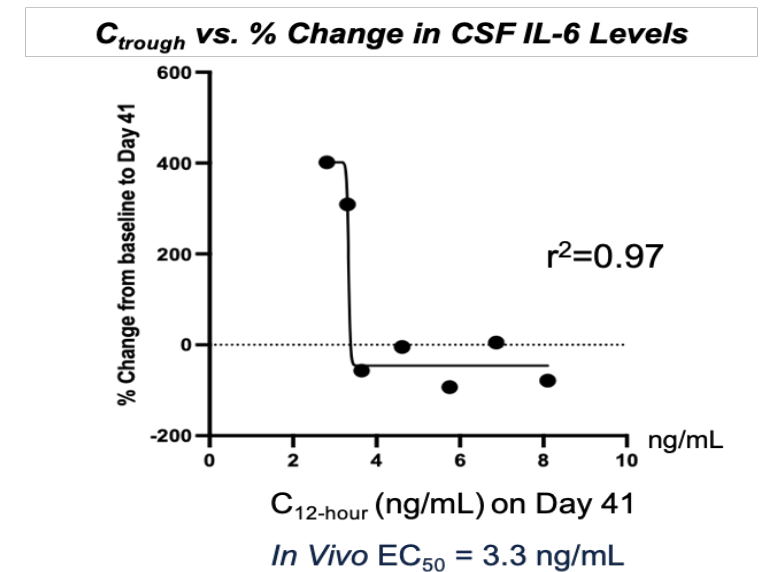


In Vitro: IL-1 β stimulated IL-6 Production from PBMCs



In vitro EC₅₀ (n=5) = 2.7 $\pm$ 0.6 ng/mL

In Patients with Early Alzheimer's Disease: Change in CSF IL-6 After 6 Weeks of Neflamapimod Treatment



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

Across preclinical and Phase 2a data, neflamapimod shows reversal of basal forebrain cholinergic dysfunction and benefits on clinically significant endpoints

Preclinical

Disease processes in basal forebrain reversed¹

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

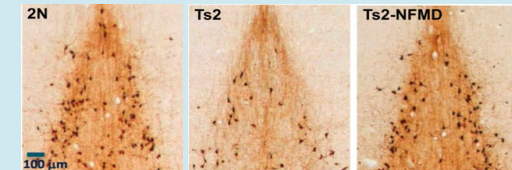
- ✓ Reduced Rab5 activity and Tau phosphorylation
- ✓ Reversed loss of cholinergic (ChAT+) neurons in the basal forebrain; and
- ✓ Normalized performance in behavioral tests of cholinergic function²

Cholinergic Neurons in Basal Forebrain

Healthy Mice

Diseased Mice

Diseased Mice Treated NFLM



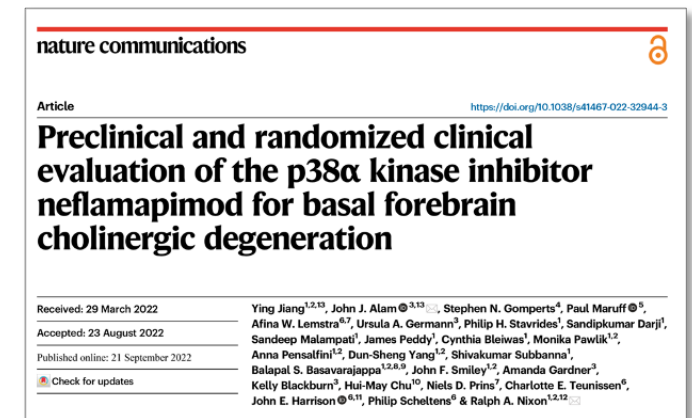
Neflamapimod preserved cholinergic neurons in the basal forebrains of mice

Phase 2a Clinical

Improvement on multiple clinical and 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)³



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

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Neurology® 2023;101:e1708-e1717. doi:10.1212/WNL.000000000000207755

Clinical Dementia Rating Sum of Boxes (CDR-SB) is a clinical meaningful endpoint for DLB clinical trials

- “Gold standard” for evaluating severity and progression of dementia
- Clinical endpoint that most effectively captures clinical progression in DLB^{1,2,3}
- Traditional cognitive testing (e.g. MMSE) less effectively captures clinical progression
 - Cognitive testing also significantly impacted by cognitive fluctuations
- Mean group difference ≥ 0.5 -point to placebo considered clinically meaningful^{4,5,6}

Aligned with FDA and global regulatory authorities on a potential registration path in DLB, including CDR-SB as the primary endpoint to support a new drug application

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)

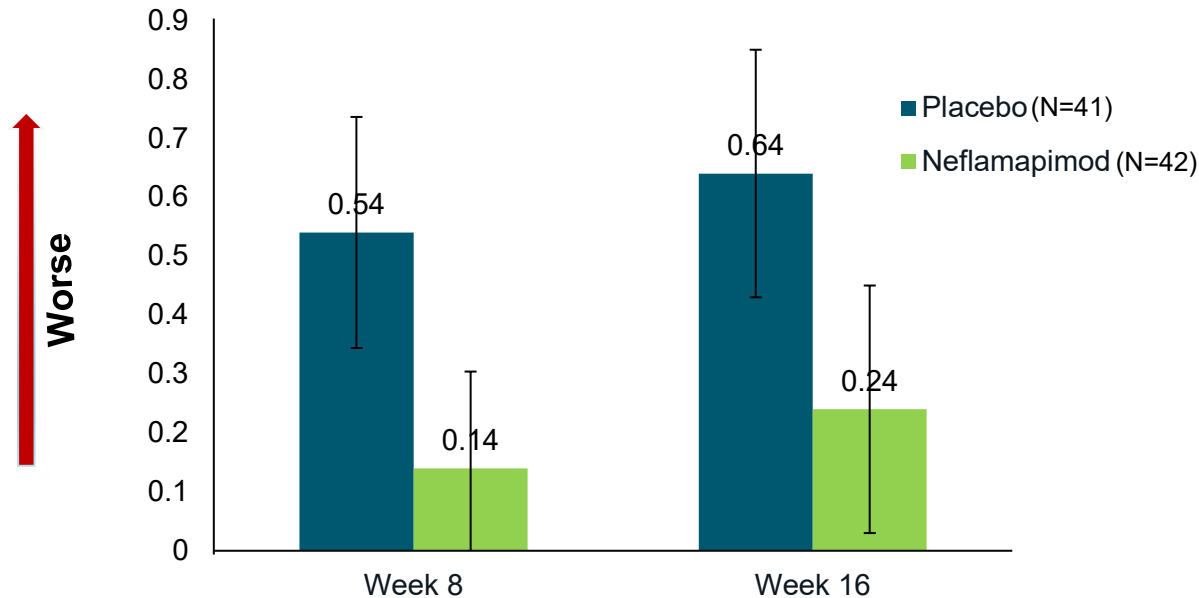
Functional Domains:

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

Key Phase 2a result: Significant improvement in change in CDR-SB, most prominently in patients without AD co-pathology (pTau181 < 21 pg/mL*)

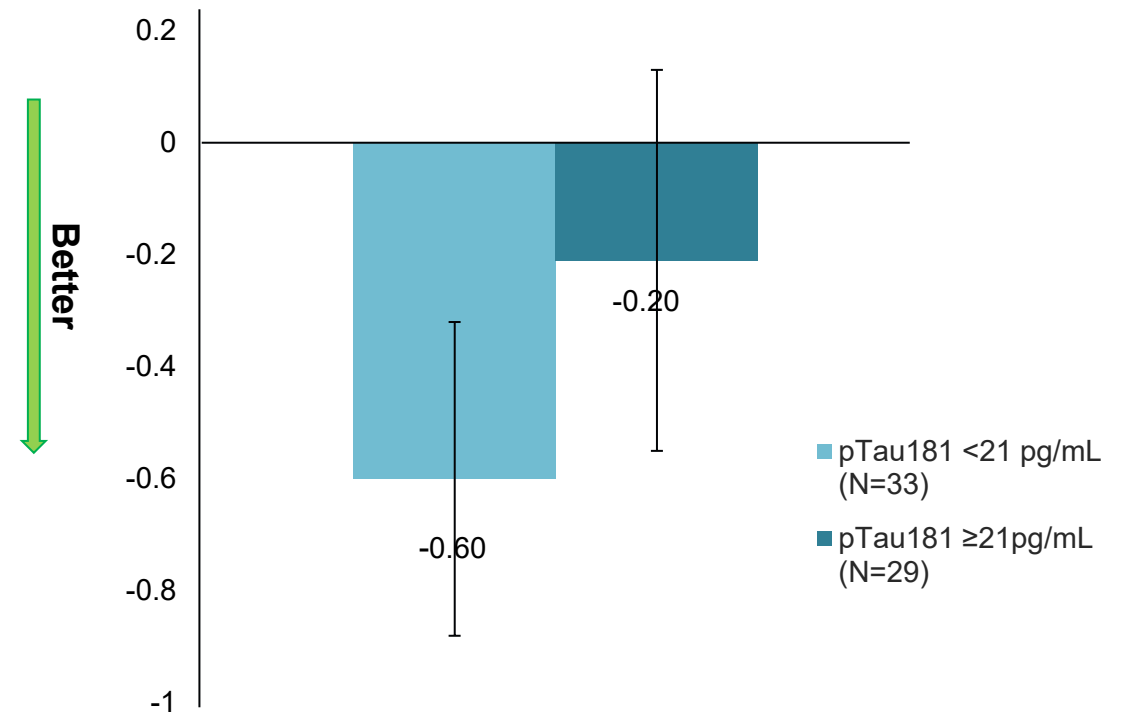
16-Week placebo-controlled study

**Mean Change from Baseline in CDR-SB
(mITT Analysis)**



p=0.023 (neflamapimod-placebo difference = - 0.45,
95% CI: -0.83, -0.06; MMRM Analysis)

**Neflamapimod 40mg TID vs. Placebo Difference
in Mean Change in CDR-SB by pTau181 strata**



Mean (SE) for Difference from MMRM Analysis

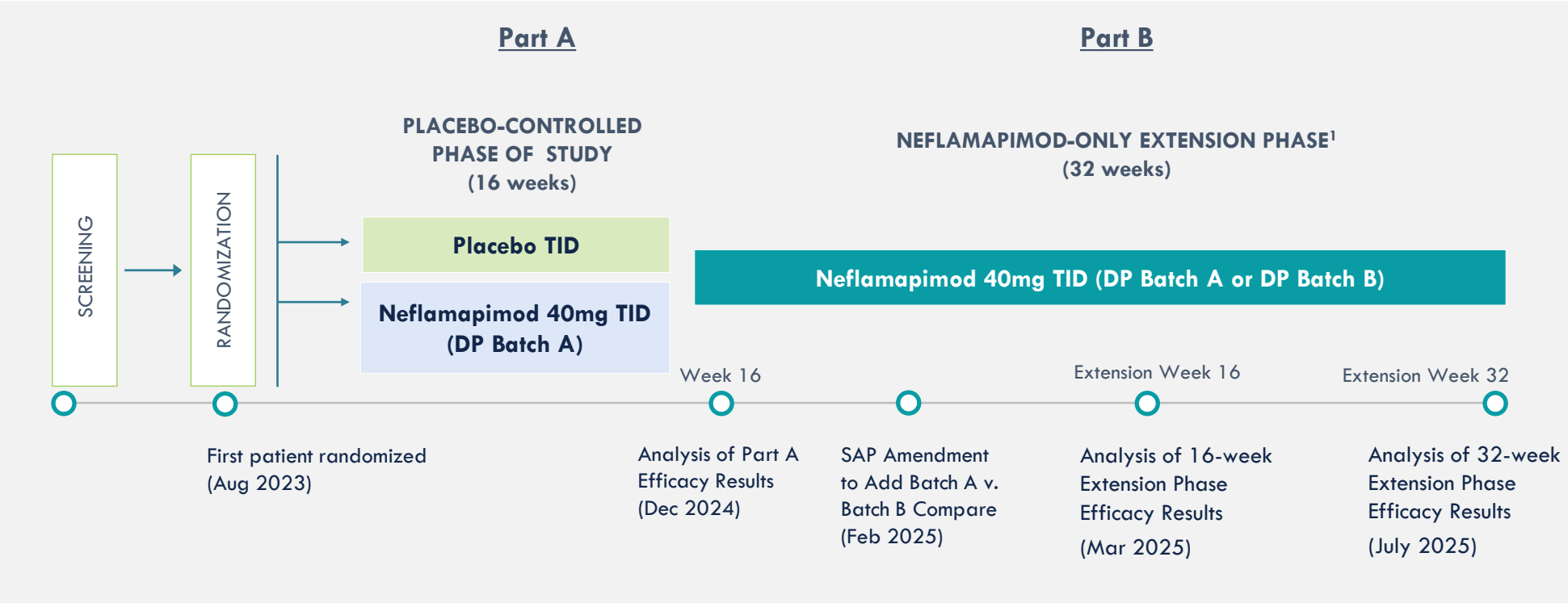
RewinD-LB Phase 2b study in DLB: A two-part study design

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)

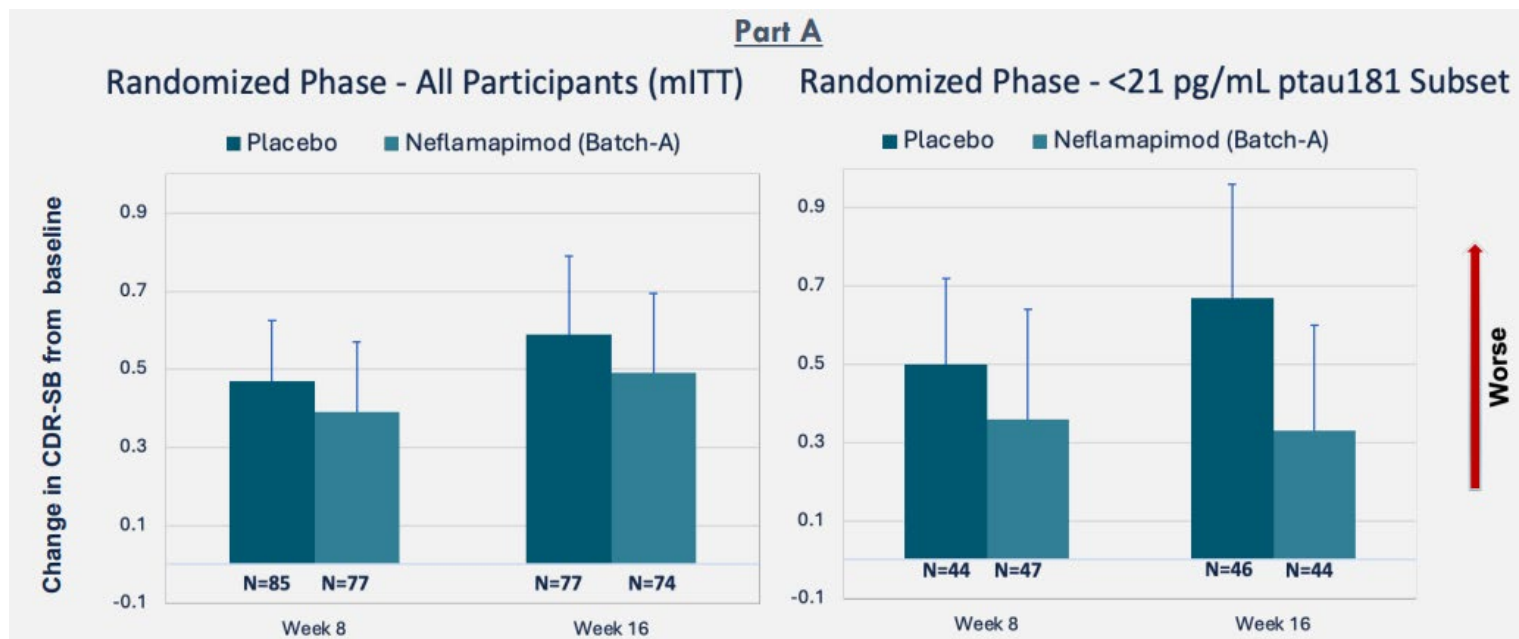
SELECTED KEY CLINICAL OUTCOME MEASURES

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

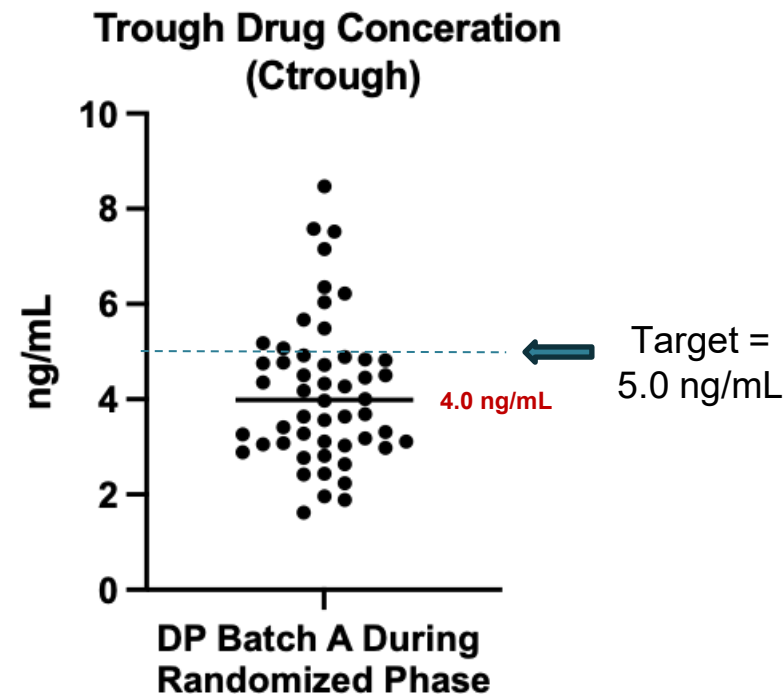


	DP Batch A	DP Batch B
Manufacturing Date	Nov 2020	March 2023
Use in RewinD-LB Study	Part A and Part B	Part B only
Reached target plasma drug concentration?	No	Yes

No significant differences between placebo and neflamapimod in primary outcome measure (CDR-SB) were observed during randomized phase

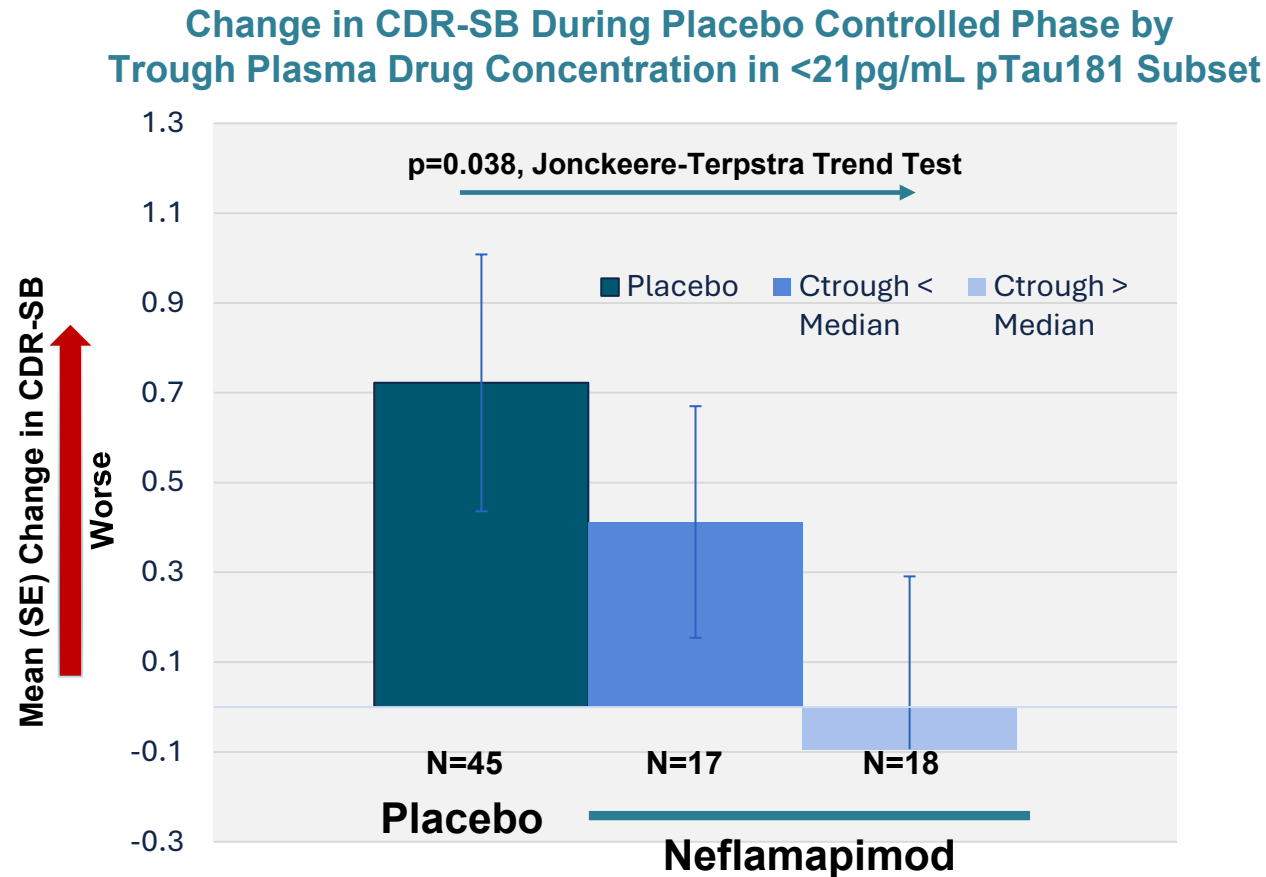


Difference = -0.70, favoring neflamapimod, $p=0.054$



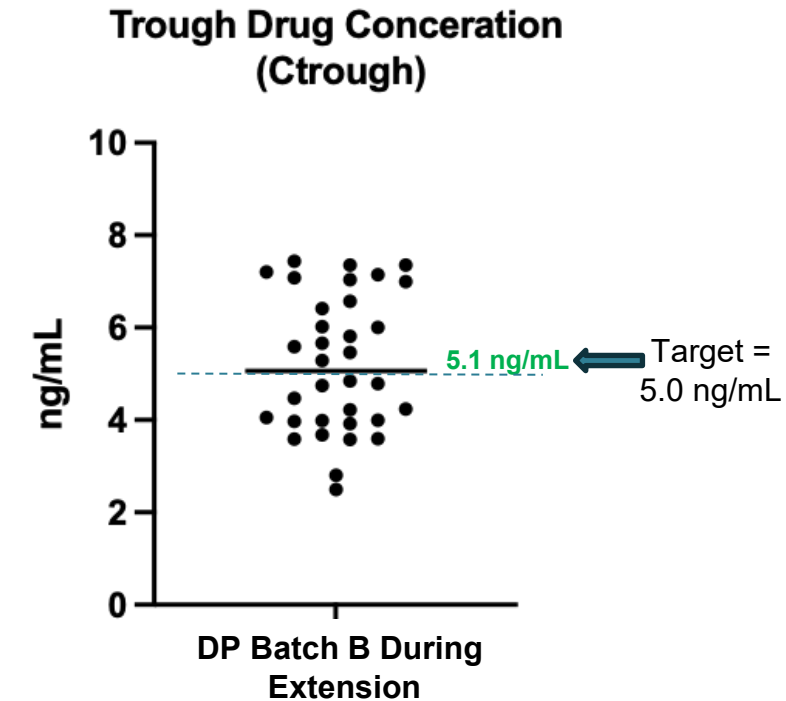
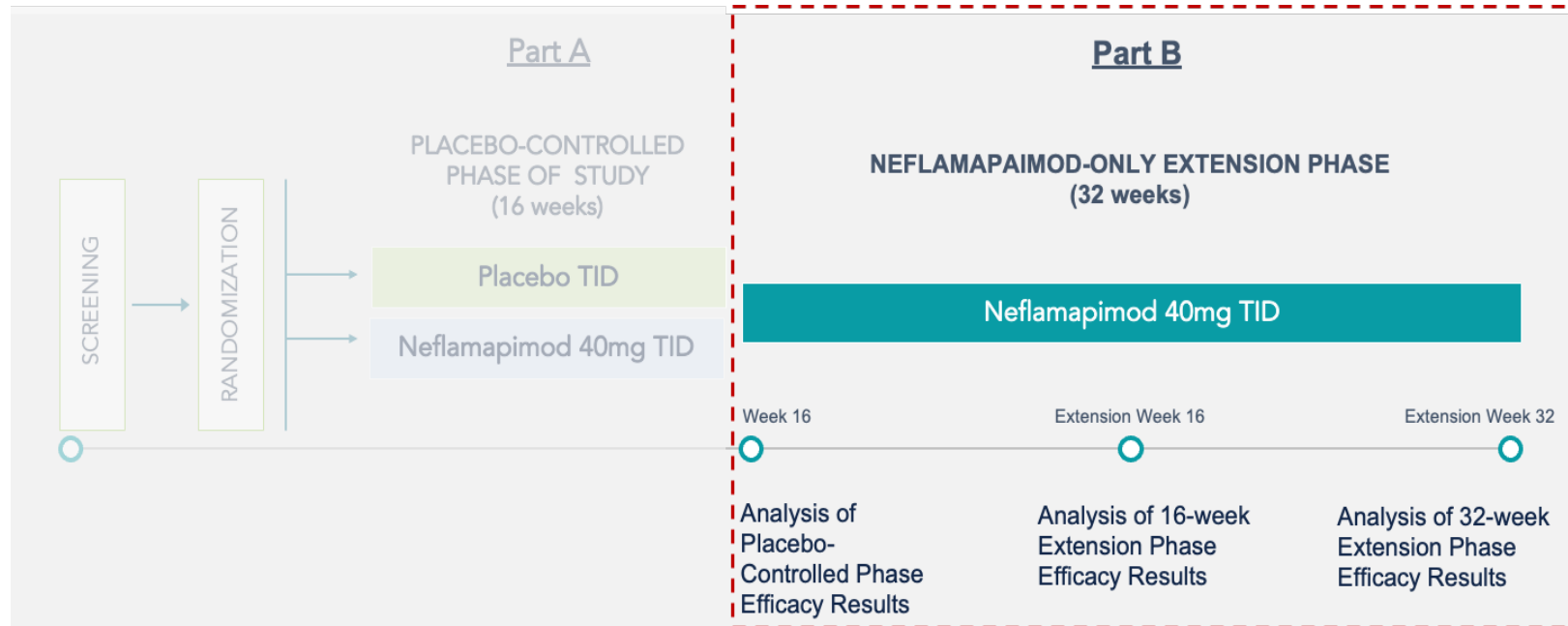
Median plasma drug trough concentration (C_{trough}) during randomized phase was 4.0 ng/mL, below expected 5.0 ng/mL¹ and similar to that seen with a lower and ineffective dose tested in Phase 2a trial; effectively underdosing participants

PK-PD analysis of randomized phase demonstrates signal of clinical activity in patients with trough plasma drug concentration (C_{trough}) above the median



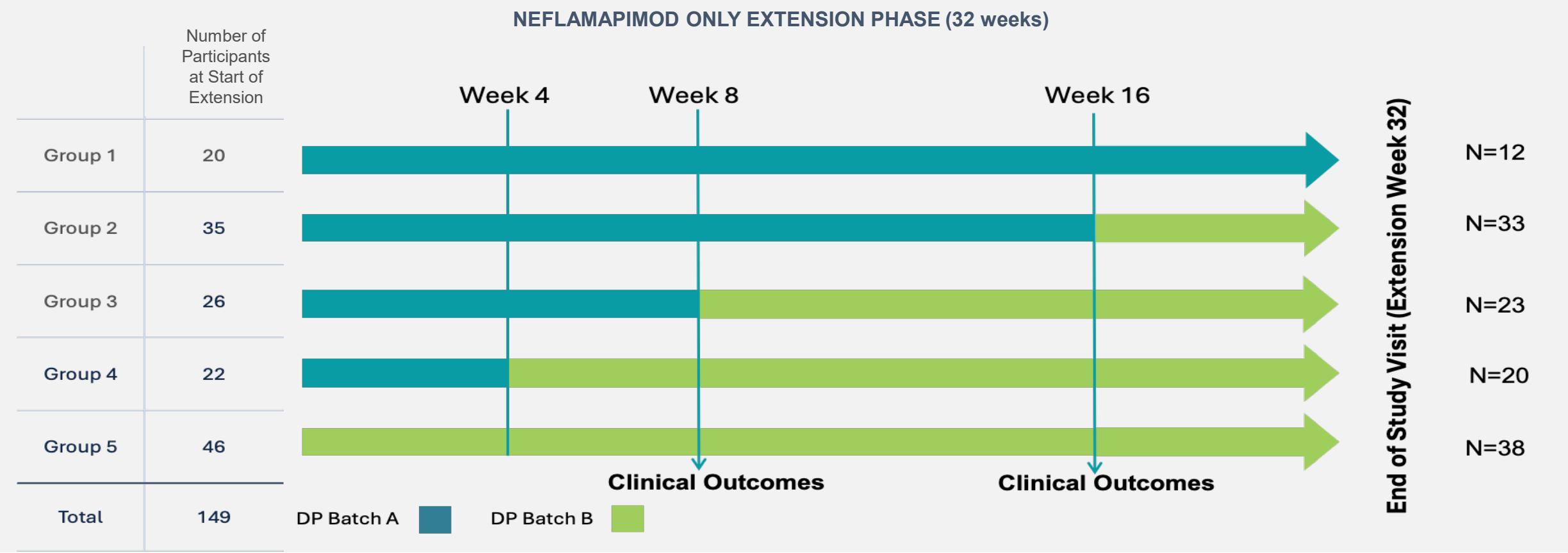
Median Observed C_{trough} = 3.64 in low pTau181 subset (< 21 pg/mL)

RewinD-LB Phase 2b study in DLB: A two-part study design



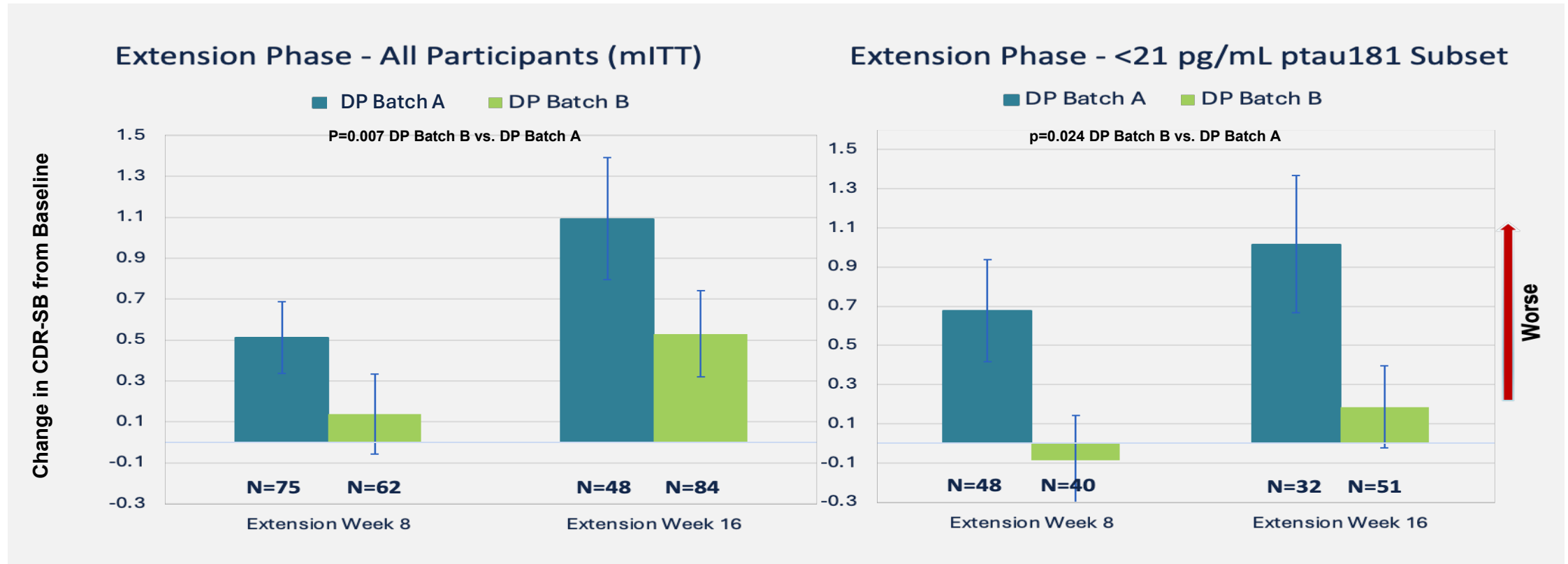
Pre-planned introduction of a new batch of drug product during the extension (DP Batch B) achieved median $C_{\text{trough}} = 5.1 \text{ ng/mL}$, achieving expected and targeted plasma drug concentrations

Introduction of new batch of neflamapimod (DP Batch B) achieved target drug concentrations and enabled robust Extension Phase analyses



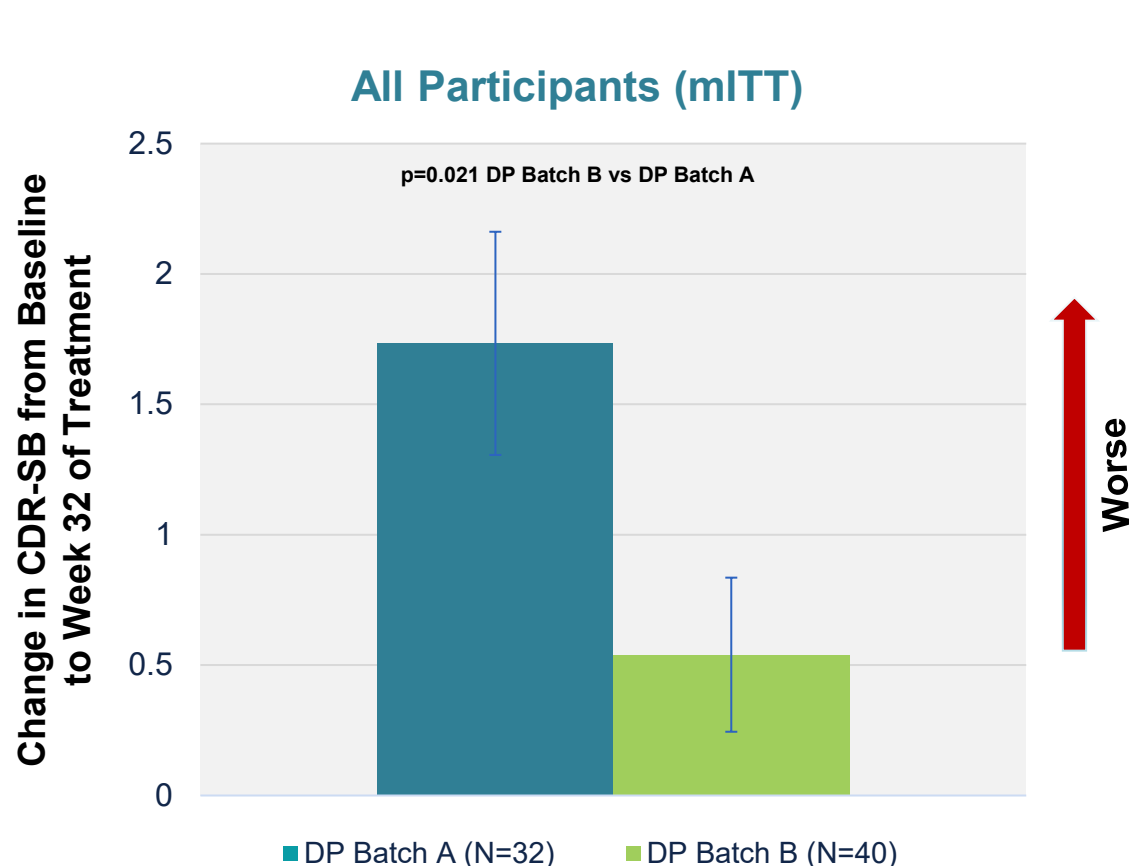
Participants were aware that they were receiving neflamapimod in the Extension Phase (i.e., treatment was open label), but neither they nor study site personnel were aware if they were receiving DP Batch A or DP Batch B

Significant effect on primary outcome measure (CDR-SB) over 16 weeks when target plasma concentrations achieved with DP Batch B

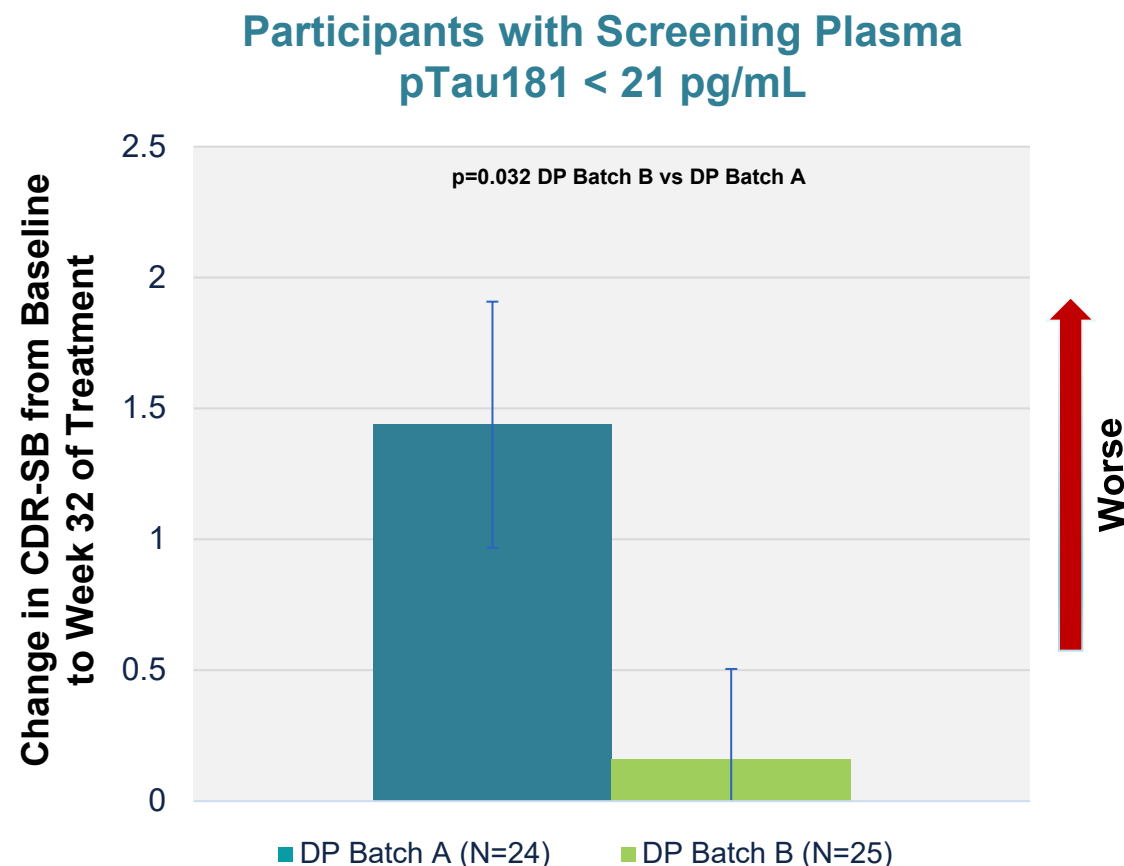


- Significant effect on clinical progression when target plasma concentrations were achieved, most prominently in participants w/o AD co-pathology
- At week 16 of Extension Phase, mean change was 52% lower with DP Batch B compared to DP Batch A in all participants and 82% lower in <21 pg/mL subset

Clinical effect observed by CDR-SB durable out to 32 weeks of treatment



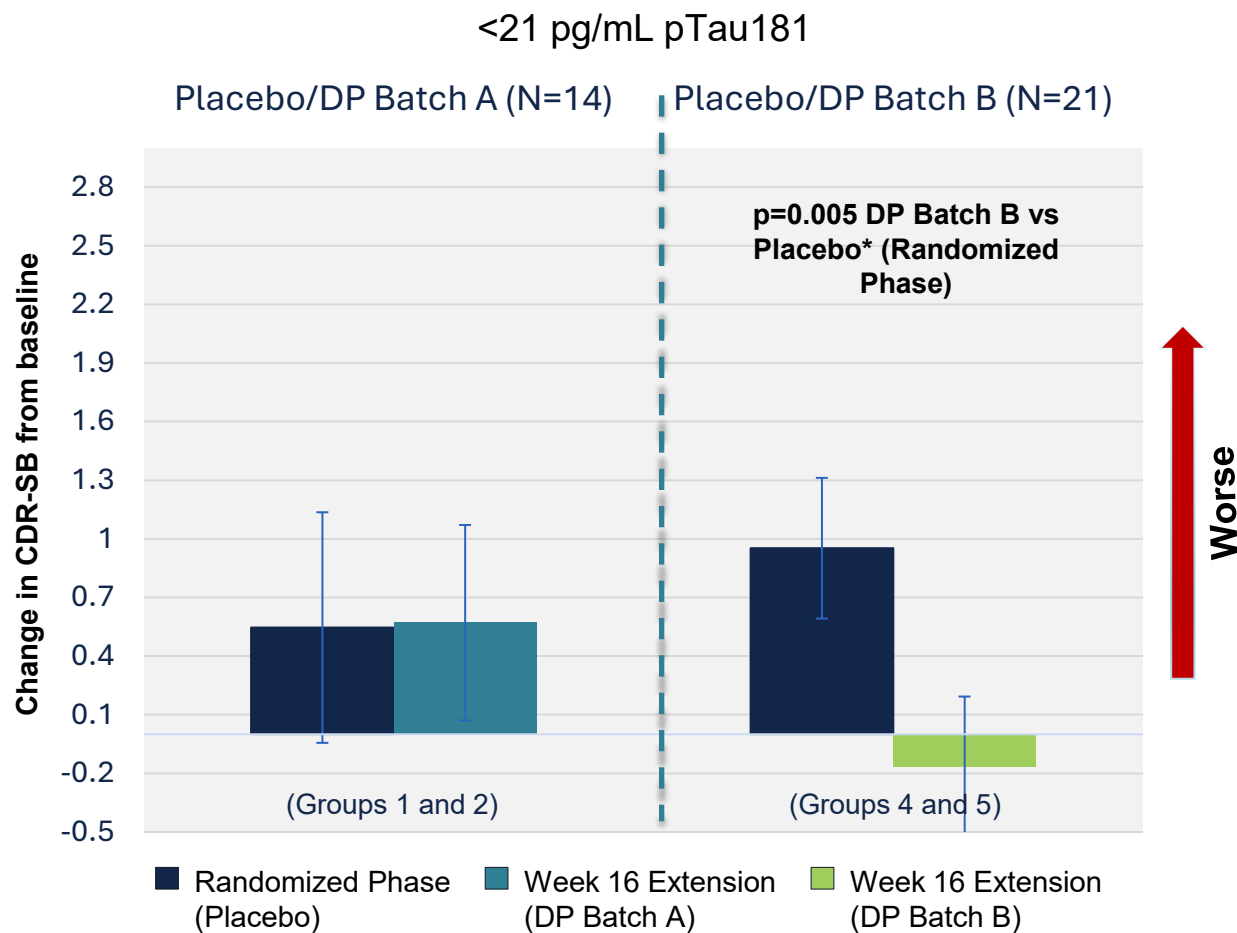
Difference: -1.12, representing a 65% reduction in CDR-SB change when targeted plasma drug concentration achieved



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

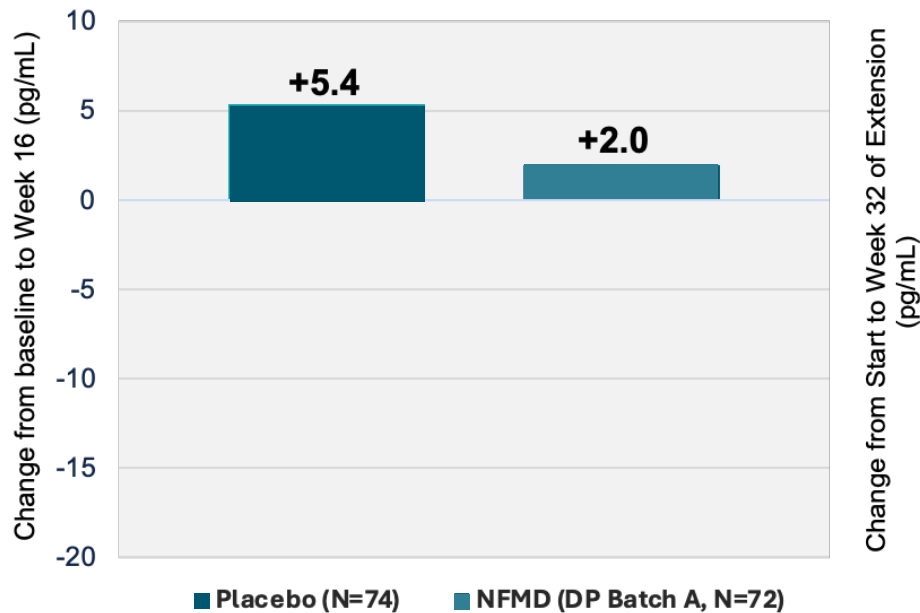
Within-subject analysis of change in CDR-SB shows neflamapimod's potential versus placebo when target plasma concentration is achieved

Change in CDR-SB During Either Randomized Phase or 16 Weeks of Extension Phase

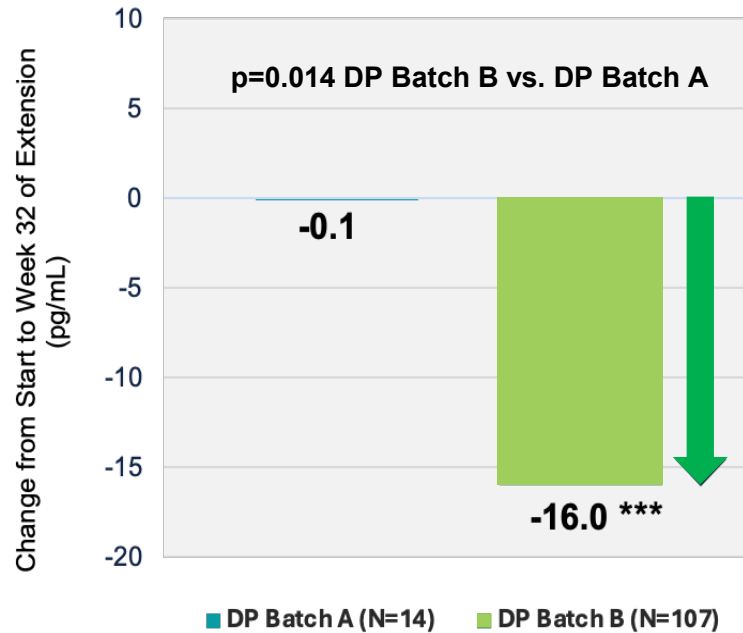


Neflamapimod achieved significant reduction in plasma glial fibrillary acidic protein (GFAP), a key biomarker of neurodegenerative disease activity

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

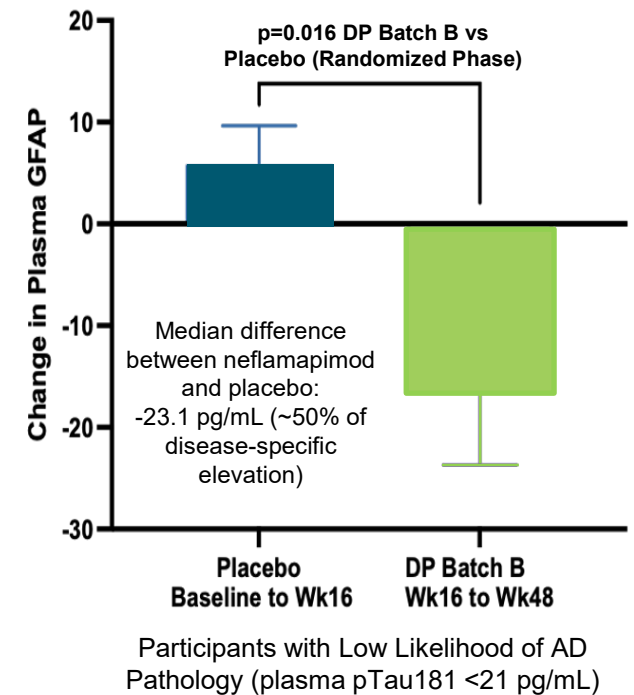


Median Change from Start to Week 32 of Extension



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

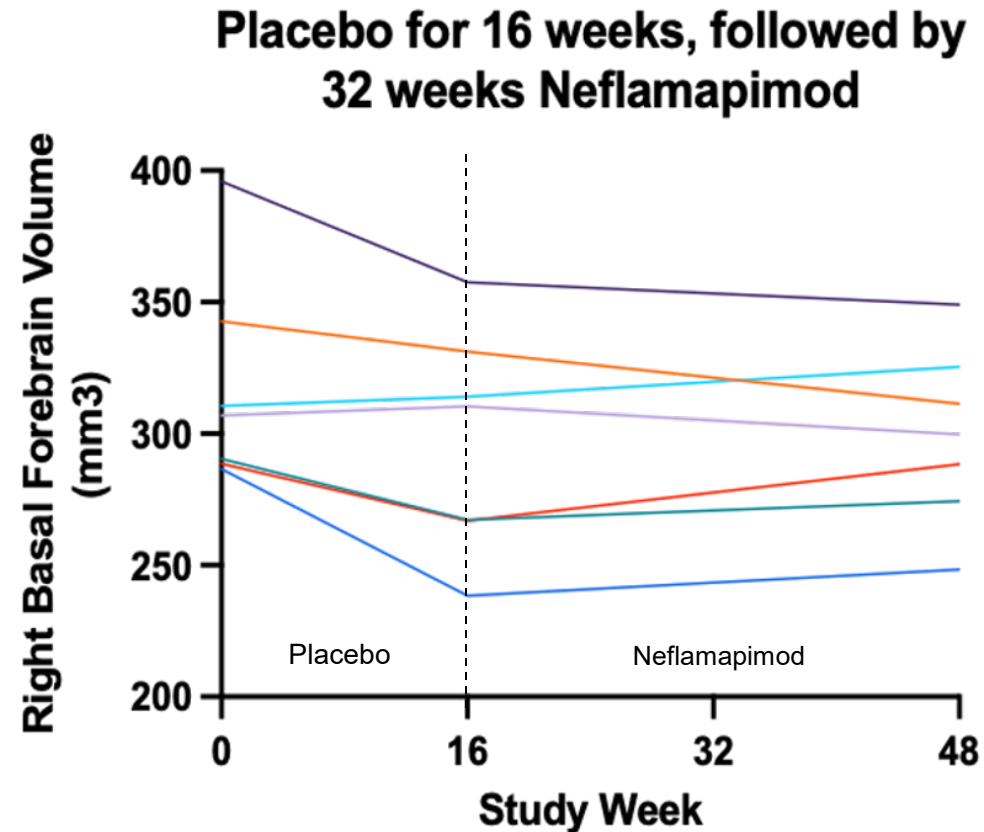
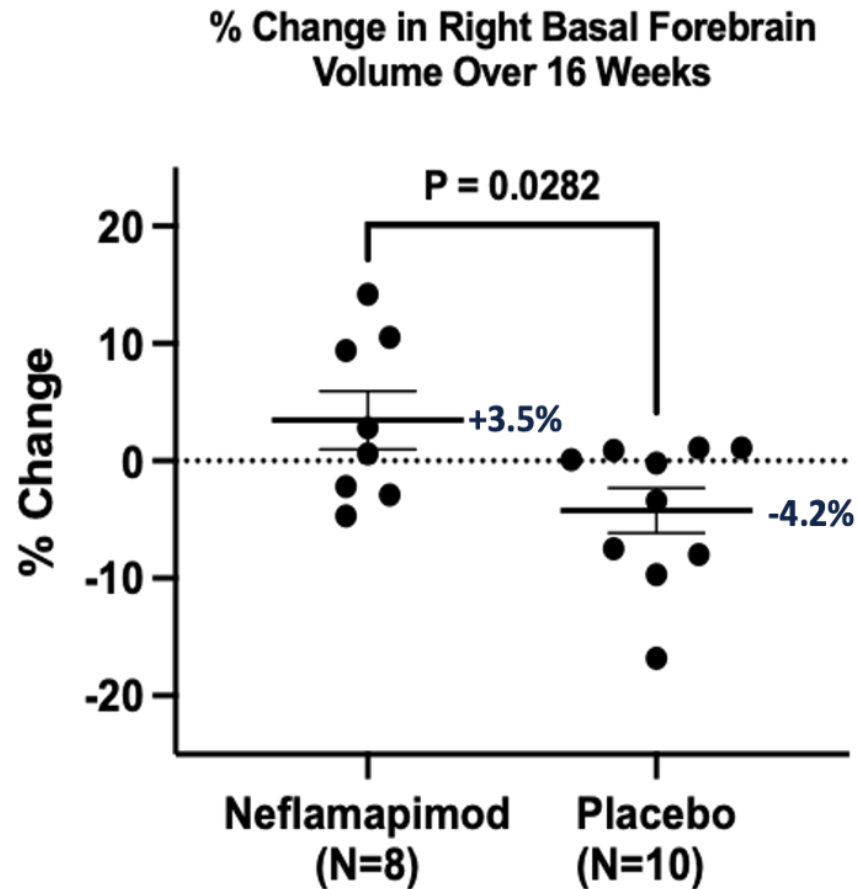
Within-Participant Comparison (N=48) of Effect on Plasma GFAP: DP Batch B vs. Placebo



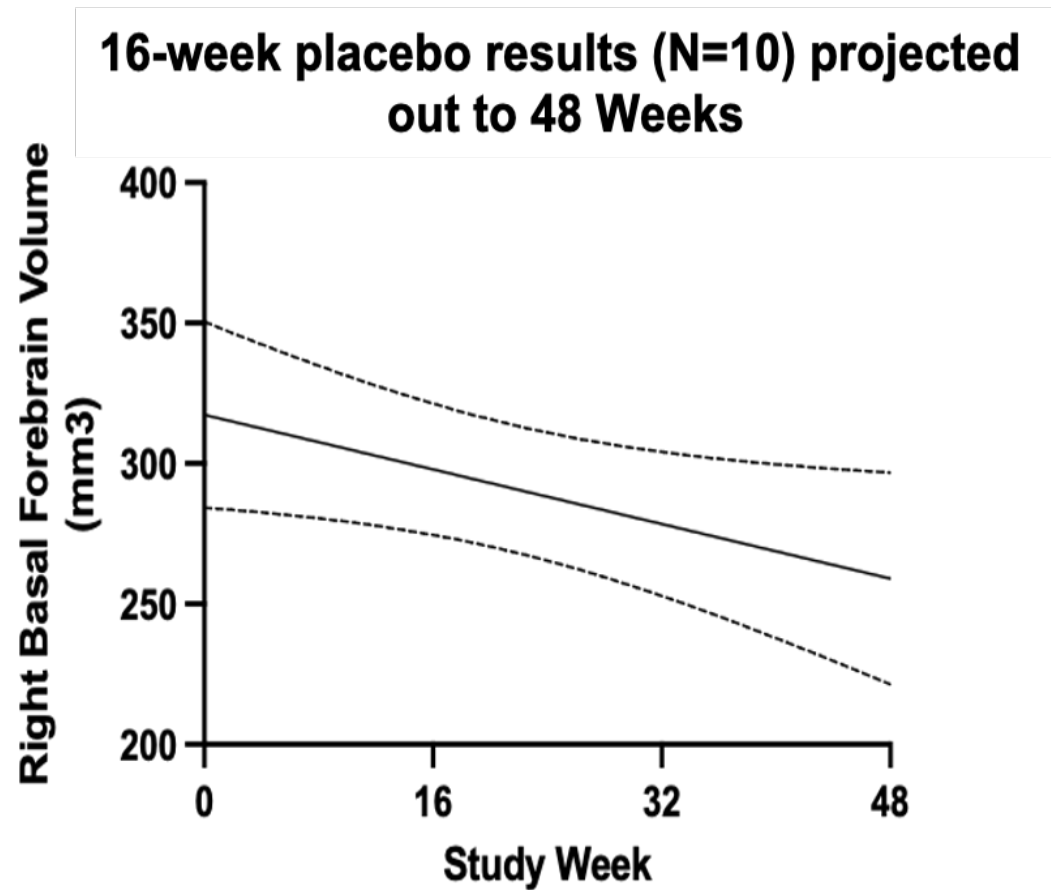
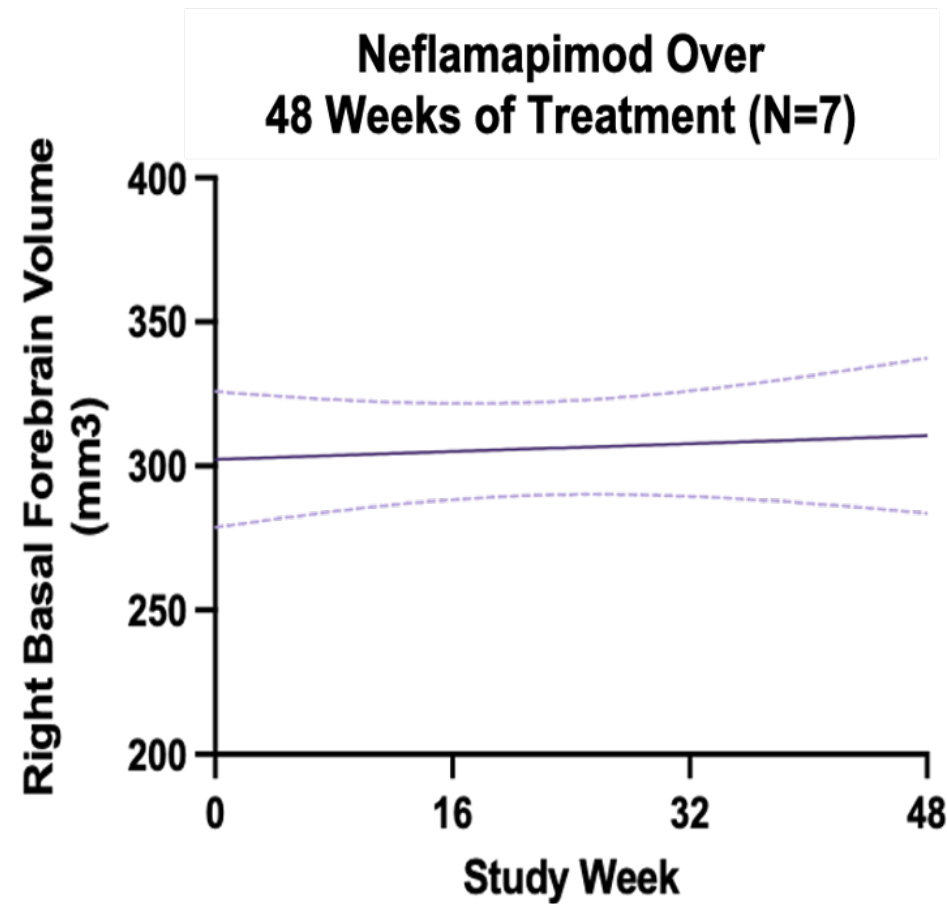
Participants with Low Likelihood of AD Pathology (plasma pTau181 <21 pg/mL)

During the Extension Phase, change in plasma GFAP correlated with treatment response as assessed by change in CDR-SB in participants with low likelihood of AD co-pathology

BF volume, by MRI, increased on neflamapimod vs. placebo (16 weeks) and stabilized in prior placebo recipients with neflamapimod during Extension



In contrast to placebo patients, which mirror DLB natural history, patients treated with neflamapimod over 48 weeks demonstrated stabilization of BF volume

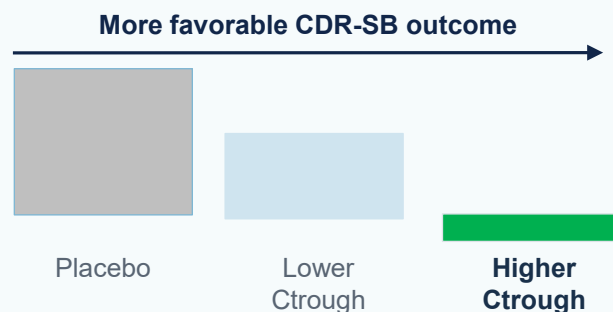


RewinD-LB demonstrated “dose dependent” clinical activity that largely replicate and extend on Phase 2a result

RANDOMIZED PHASE

Clinical activity was limited to participants with higher Ctrough levels

- Drug exposure in Randomized Phase was below that expected and observed in Phase 2a study
- In the <21 pg/mL p-Tau181 subset, outcomes followed an ordered exposure pattern:



- Overall primary endpoint not met; however, a “dose-dependent” signal was observed

EXTENSION PHASE

DP Batch B achieved the expected 40 mg TID exposure and demonstrated clinical activity

Mean steady-state Ctrough

5.1 ng/mL

p=0.003 vs randomized-phase concentrations

Extension (first 16 weeks): DP Batch B vs DP Batch A

CDR-SB **-0.57** (p=0.007)

ADCS-CGIC **-0.40** (p=0.03)

Effects on CDR-SB were durable through 32 weeks of treatment

pTau181 ENRICHMENT

Clinical effects strengthened as level of AD co-pathology was minimized

The low plasma pTau181 (<21 pg/mL) identified the subgroup with the most consistent clinical response across both phases of the trial

Within-subject comparison in participants with pTau181 <21 pg/mL who received placebo then Batch B:

>1-point improvement in CDR-SB vs. placebo

Concordant improvement in ADCS-CGIC observed (Difference vs. placebo = -0.82; 95% CI: -1.33,-0.33; p=0.004)

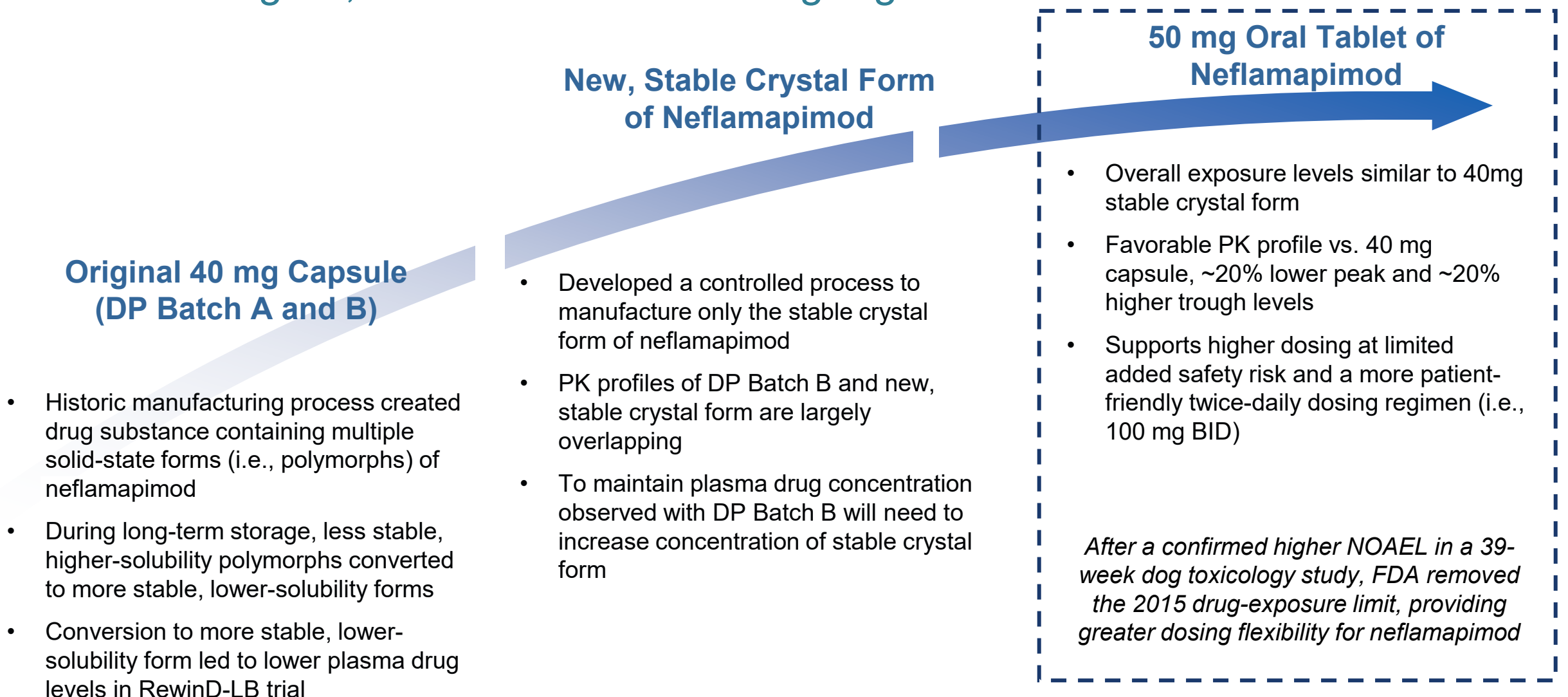
Convergent support:

Plasma GFAP significantly reduced at Week 32 of the Extension Phase

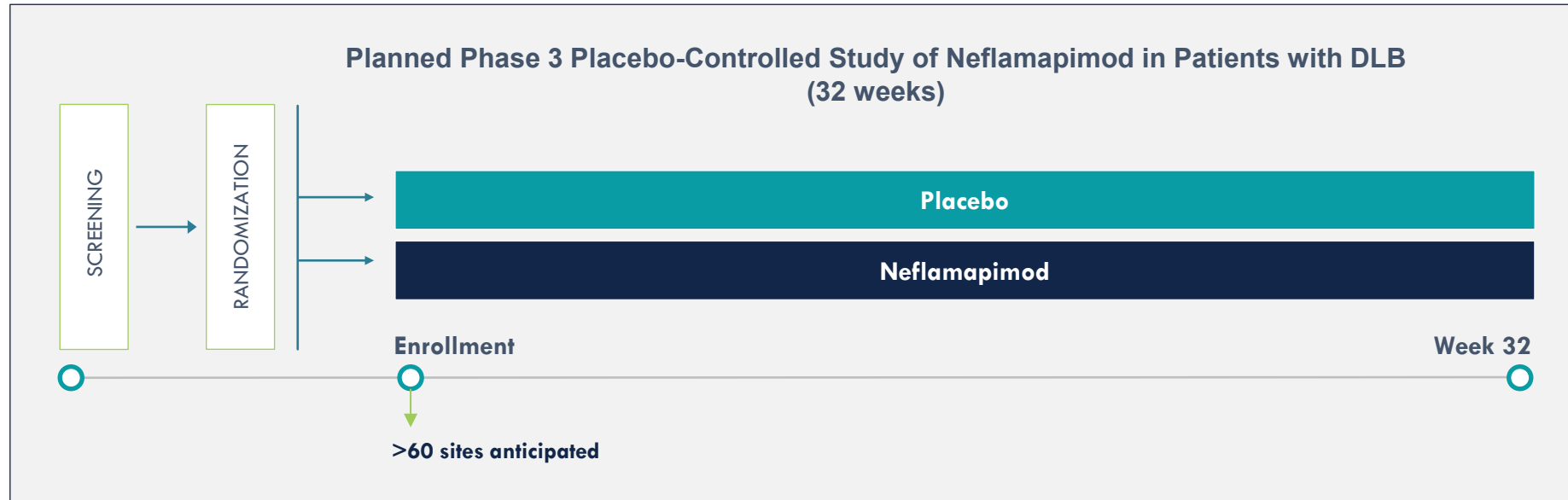
Basal forebrain MRI at end of Randomized phase: +3.5% vs -4.2% in placebo; p=0.028

Same directional pattern across clinical, PK, biomarker, and imaging measures

Favorable pharmacokinetic profile and FDA removal of drug exposure limit, enables a higher, more convenient dosing regimen



Secured alignment from global regulatory authorities on proposed Phase 3 trial in DLB and completed activities to support improvements in drug product formulation



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
- 50mg BID dosing regimen with new, stable crystal form; initial clinical batches manufactured and released
- >90% statistical power to achieve similar clinical effect as observed in Phase 2b study
- Potential to conduct futility analysis after 150 patients reach 16-week timepoint: discontinue study if p-value ≥ 0.07 for improvement with neflamapimod compared to placebo at week 16

A year of progress for neflamapimod

- ✓ Further **defined mechanism of action** and relevance to neurodegenerative disease processes
 - 7nM inhibitor (EC50) of IL-1 β signaling, which aligns with current understanding of pathogenesis of DLB (interplay of neuroinflammatory signaling pathways and alpha synuclein is primary driver of disease expression and progression)
- ✓ Repeated **39-week repeat dose toxicology study** in dogs – raised NOAEL > 3-fold
- ✓ Completed and **reported out RewinD-LB** clinical study in DLB
 - Positive effect on CDR-SB and ADCS-CGIC, particularly in patients with < 21 pg/mL pTau181 at screening (corresponds to cutoff in phase 2a that showed optimal efficacy)
 - Reduction in a neurodegenerative disease marker, plasma GFAP at 32 weeks of treatment
 - Reduced basal forebrain atrophy, primary pathology in DLB, vs. placebo
 - Defined optimal dose regimen – 50mg BID
- ✓ Alignment with **FDA, EMA, MHRA, PMDA on Phase 3 design** in DLB and path to approval
- ✓ Developed **Phase 3 and commercial ready tablet** BID formulation
- ✓ Received a notice of allowance from the U.S. Patent and Trademark Office for a **new patent** covering the **use of neflamapimod in the treatment of DLB in patients pure DLB**
- ✓ Awarded Innovation Passport Designation under UK **Innovative Licensing and Access Pathway (ILAP)** for DLB treatment
- ✓ Based on independent scientific publications, **expanded into rare neurologic disorders**
 - Phase 2a nonfluent variant primary progressive aphasia (subtype of FTD) fully enrolled; biomarker results at ISFTD and CTAD2026
 - Phase 2 study in ALS to start in Q4'26 (under UK-government funded rolling platform trial)

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)
- ❑ **Full 12-week and initial 24-week Phase 2a biomarker data in nfvPPA (4Q26)**
- ❑ **First patient expected to be dosed with neflamapimod in EXPERTS-ALS trial in ALS (YE26)**
- ❑ **Topline Phase 2a clinical data in RAS (2H26)**
- ❑ **Continue to advance strategic objective to establish partnership to advance DLB into Phase 3 (2H26)**

2026

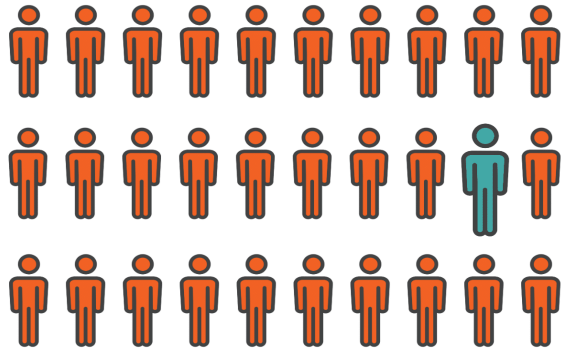
2027

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

The Opportunity for Neflamapimod in Nonfluent Variant Primary Progressive Aphasia (nfvPPA)

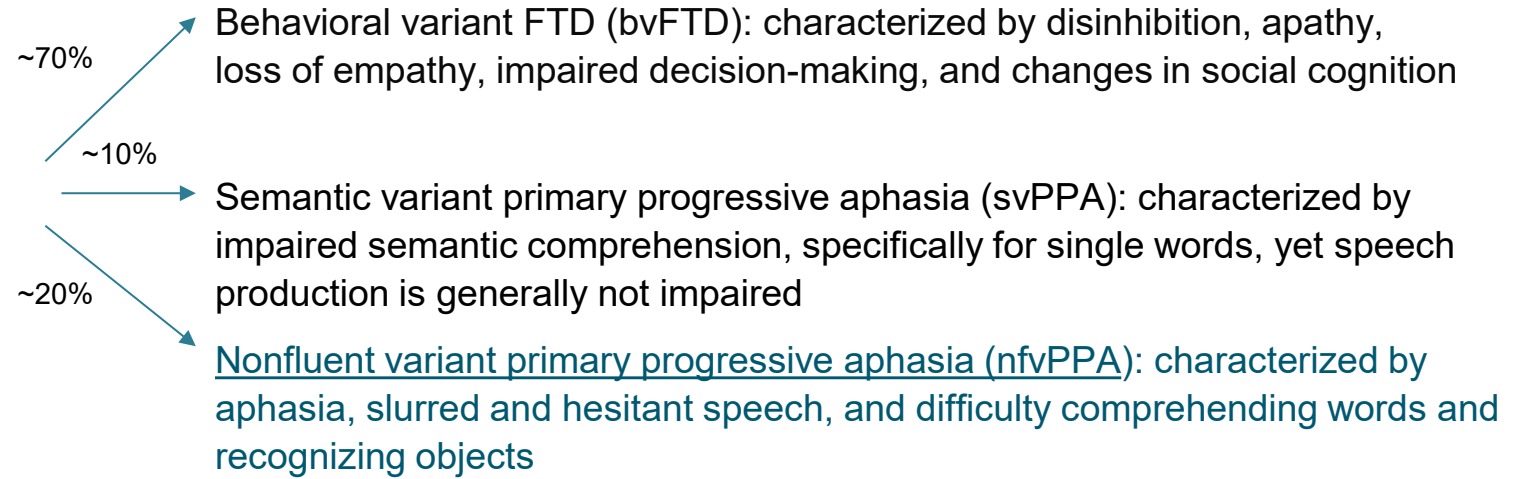


FTD is characterized by selective degeneration of the frontal and/or temporal lobe, areas responsible for personality, behavior, and language



1 in 30 people with dementia have FTD (~50-60k cases in US)¹

3 main clinical FTD subtypes



Patients with nfvPPA experience significant morbidity and mortality



Median age of onset is 62 years (range 49 to 73 years) with median survival from symptom onset of ~7 years²

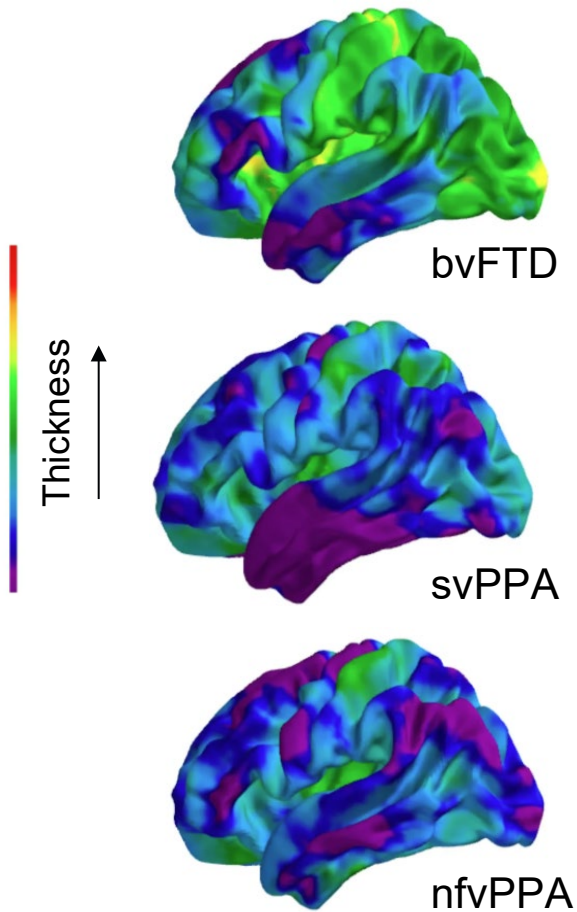


Cognitive impairment, behavioral disturbances, and/or motor deficits can occur as disease progresses^{3,4,5}



No approved treatments for nfvPPA in US; language impairments are managed by speech pathologists^{6,7}

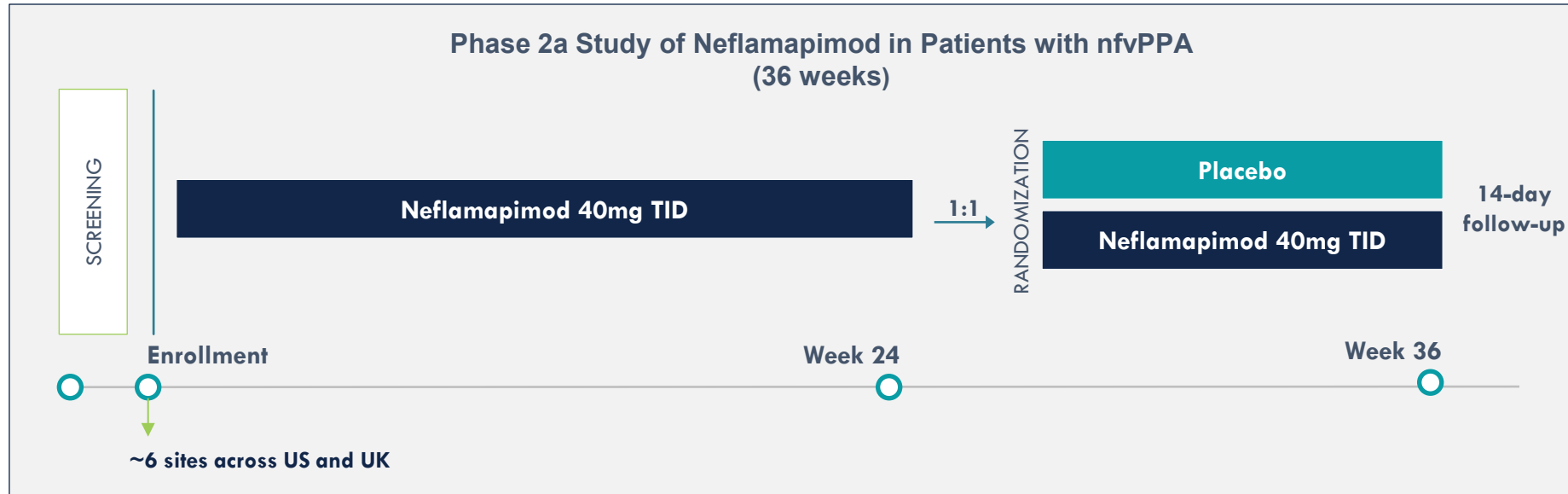
Neflamapimod is the first therapy evaluated in FTD designed to directly address the effect of tau pathology, the key driver of disease in nfvPPA



- Pathologically, PPA is characterized by significant neuronal loss in the frontal and temporal cortex
- nfvPPA is strongly linked to tau protein aggregation (88% of nfvPPA cases at exhibited FTLD-tau pathology at autopsy)¹
 - svPPA is more closely associated with TDP-43 pathology ^{2,3,4}
- Patients with FTD-tau pathology have also been shown to have significant atrophy in the basal forebrain, the major site of cholinergic neurons within the brain^{5,6}
 - In patients with FTD, the smallest MRI volumes of the basal forebrain were seen in those with tau pathology at autopsy

Progressive changes in cortical thickness over a 12-month period, demonstrating differential regional changes for each variant, with the greatest atrophy in the purple and blue colors. Modified from <https://imaging.cro.biospective.com/resources/neuroimaging-ftd-clinical-trials>. 1. Spinelli, E.G., et al., Typical and atypical pathology in primary progressive aphasia variants. Ann Neurol, 2017. 81(3): p. 430-443; 2. Josephs, K.A., et al., Clinicopathological and imaging correlates of progressive aphasia and apraxia of speech. Brain, 2006. 129(Pt 6): p. 1385-98; 3. Mesulam, M., et al., Alzheimer and frontotemporal pathology in subsets of primary progressive aphasia. Ann Neurol, 2008. 63(5): p. 709-19; 4. Kish, J.A., et al., Clinical and pathological characterization of progressive aphasia. Ann Neurol, 2006. 59(1): p. 156-65; 5. Convery, R.S., et al., Basal forebrain atrophy in frontotemporal dementia. Neuroimage Clin, 2020. 26: p. 102210; 6. Lagarde, J., et al., Combined in vivo MRI assessment of locus coeruleus and nucleus basalis of Meynert integrity in amnesic Alzheimer's disease, suspected-LATE and frontotemporal dementia. Alzheimers Res Ther, 2024. 16(1):p. 97.

Phase 2a trial in patients with nonfluent variant of primary progressive aphasia (nfvPPA), a subtype of FTD, is ongoing; biomarker to be presented 4Q26



KEY PARAMETERS

- 40 to 75 years
- Clinical diagnosis of nfvPPA by consensus criteria¹
- Global CDR® plus NACC FTLD score of 0.5 or 1
- CDR® plus NACC FTLD language domain score of 0.5, 1 or 2

- Phase 2a clinical trial of 36 weeks duration (N=25)
- The objectives of this exploratory study are to evaluate the effect of neflamapimod in patients with nfvPPA on a range of clinical and biomarker outcomes:
 - Clinical measures of aphasia
 - Plasma biomarkers of neurodegeneration (Neurofilament light chain GFAP)
- Initial 12-week plasma biomarker data to be presented at ISFTD meeting in October, with 24-week data to be presented at CTAD meeting in November

Corporate Background



Financial Overview¹



Balance Sheet

- \$24.9M in cash, cash equivalents, and marketable securities as of June 30, 2026

Capitalization

- 15.1 million shares outstanding
 - 9.4 million shares underlying outstanding warrants
 - Weighted Average Exercise Price = \$12.99



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.



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



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



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



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



Marco Verwijs, PhD

EVP, Technical Operations

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

Experienced board of directors and scientific advisors, pairing biotech and business leadership with world-class scientific expertise

BOARD OF DIRECTORS

John Alam, MD

President, CEO & Co-Founder, Director
Former CMO and EVP Medicines Dvlpt., Vertex;
Former Global Head Alzheimer's R&D at Sanofi;
Led clinical development of Avonex for multiple sclerosis at Biogen

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, Revvity,
ViFor, Corvidia, Cubist

Jeff Poulton (Chair of Audit Committee)

CFO, Alnylam Pharmaceuticals (Nasdaq:ALNY)
Former CFO, Shire Pharmaceuticals; CFO,
Indigo Agr.

Jane H. Hollingsworth, JD

Managing Partner, Militia Hill Ventures
Former Chairman of the Board, Diffusion
Pharmaceuticals

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

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

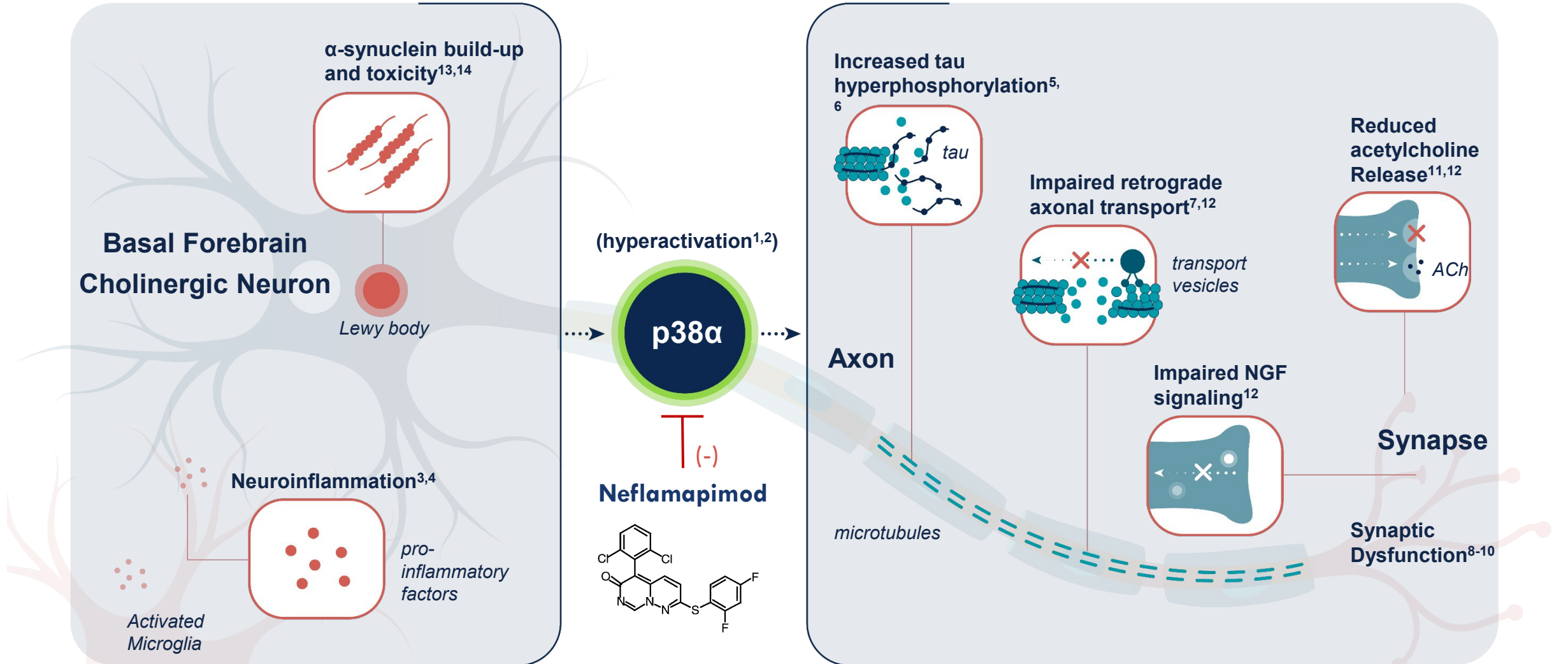
Appendix



Additional DLB Data



By blocking p38 α , neflamapimod aims to reverse synaptic dysfunction, improve neuron health, and slow disease progression in the basal forebrain



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

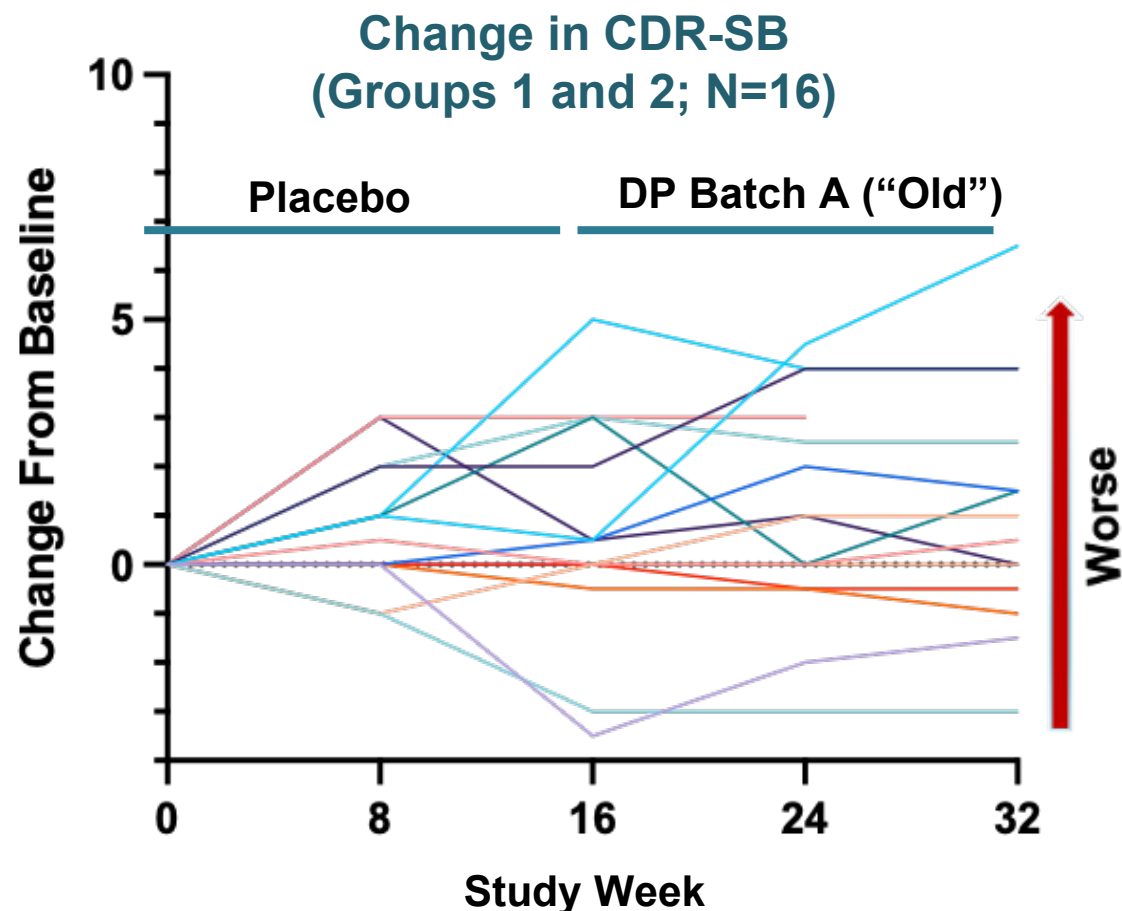
- Historic manufacturing process created drug substance containing multiple solid-state forms (i.e., polymorphs) of neflamapimod
- Polymorphs exhibited diverse properties:
 - One polymorph demonstrated high stability but lower-solubility, which leads to lower bioavailability
 - Other polymorphs demonstrated 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

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

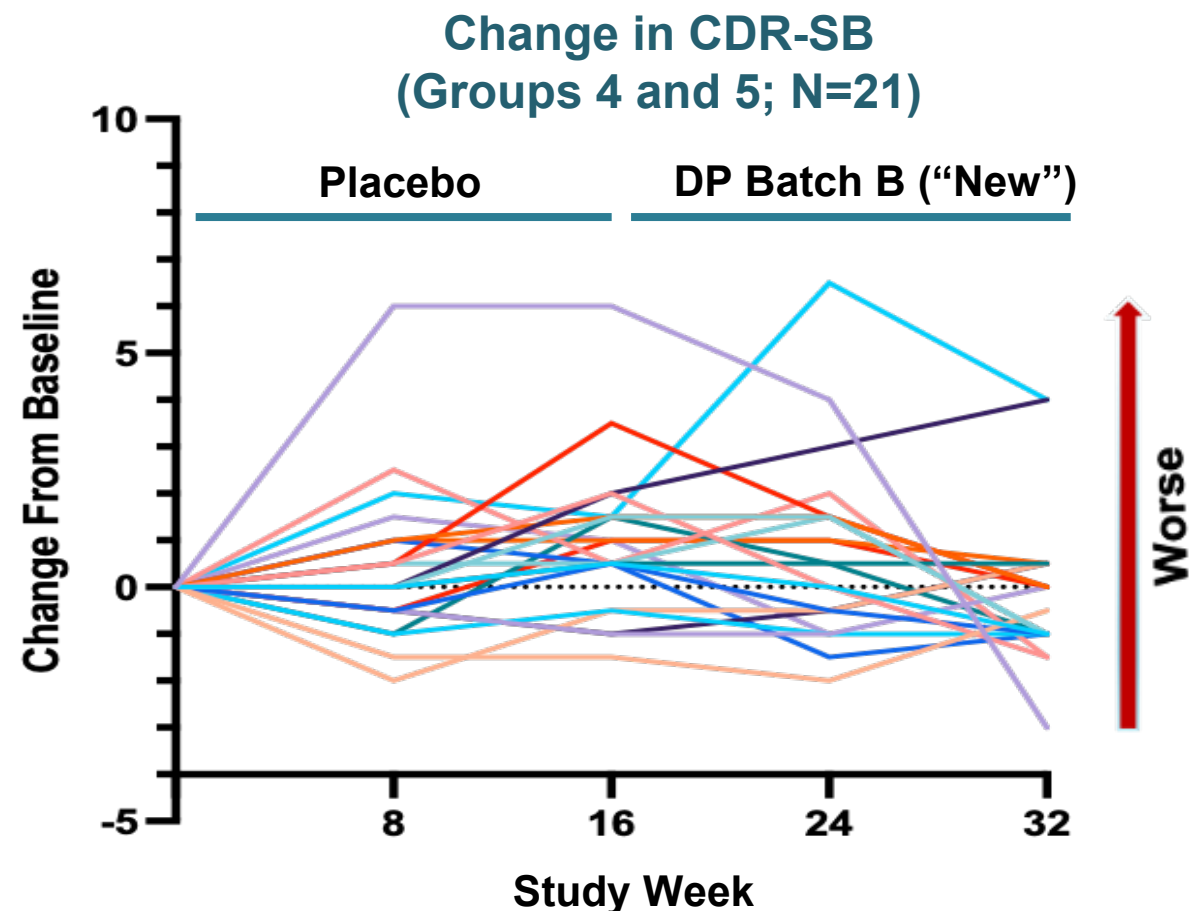
Phase 2b baseline characteristics were similar across study phases, patient subsets, and drug batches

	Randomized Phase		Extension Phase ¹ (All Participants)		Extension Phase ¹ (<21 pg/mL subset)	
Characteristics	Placebo	DP Batch A	DP Batch A	DP Batch B	DP Batch A	DP Batch B
Number of Participants	79	80	55	94	38	55
Age	70.7 (5.8)	72.1 (6.4)	70.9 (6.4)	71.8 (6.1)	69.9 (6.18)	70.6 (6.42)
Male	67 (83.8%)	69 (87.3%)	48 (87.3%)	80 (85.1%)	30 (83.3%)	52 (94.5%)
MMSE	23.3 (4.6)	23.6 (4.3)	23.8 (3.7)	23.4 (4.8)	23.6 (3.63)	24.1 (4.21)
CDR-SB	4.23 (1.71)	4.45 (2.17)	4.87 (2.41)	4.79 (2.45)	4.22 (2.04)	4.22 (1.73)
ISLT Immediate	14.8 (5.9)	13.0 (5.0)	13.5 (5.2)	13.8 (5.7)	13.1 (4.45)	14.6 (5.41)
Core Clinical Criteria:						
Cognitive fluctuations	59 (73.8%)	59 (74.7%)	32 (58.2%)	77 (81.9%)	20 (55.6%)	48 (87.3%)
Visual Hallucinations	45 (56.3%)	43 (54.4%)	26 (47.3%)	54 (57.4%)	17 (47.2%)	33 (60.0%)
REM sleep behavioral disorder	64 (80.0%)	60 (75.9%)	46 (83.6%)	69 (73.4%)	29 (80.6%)	42 (76.4%)
Parkinsonism	68 (85.0%)	70 (88.6%)	46 (83.6%)	85 (90.4%)	30 (83.3%)	49 (89.1%)
Background Therapy						
AChEI alone* ²	52 (65.0%)	50 (63.3%)	36 (65.4%)	46 (61.3%)	25 (69.4%)	34 (61.8%)
Memantine (with or without AChEI therapy)	11 (13.8%)	12 (15.2%)	9 (16.4%)	14 (18.7%)	7 (19.4%)	11 (20.0%)
No background therapy	17 (21.3%)	17 (21.5%)	10 (18.2%)	5 (20.0%)	4 (11.1%)	10 (18.2%)

Within-participant comparison to placebo in participants without AD co-pathology confirms effect on CDR-SB when target concentrations achieved



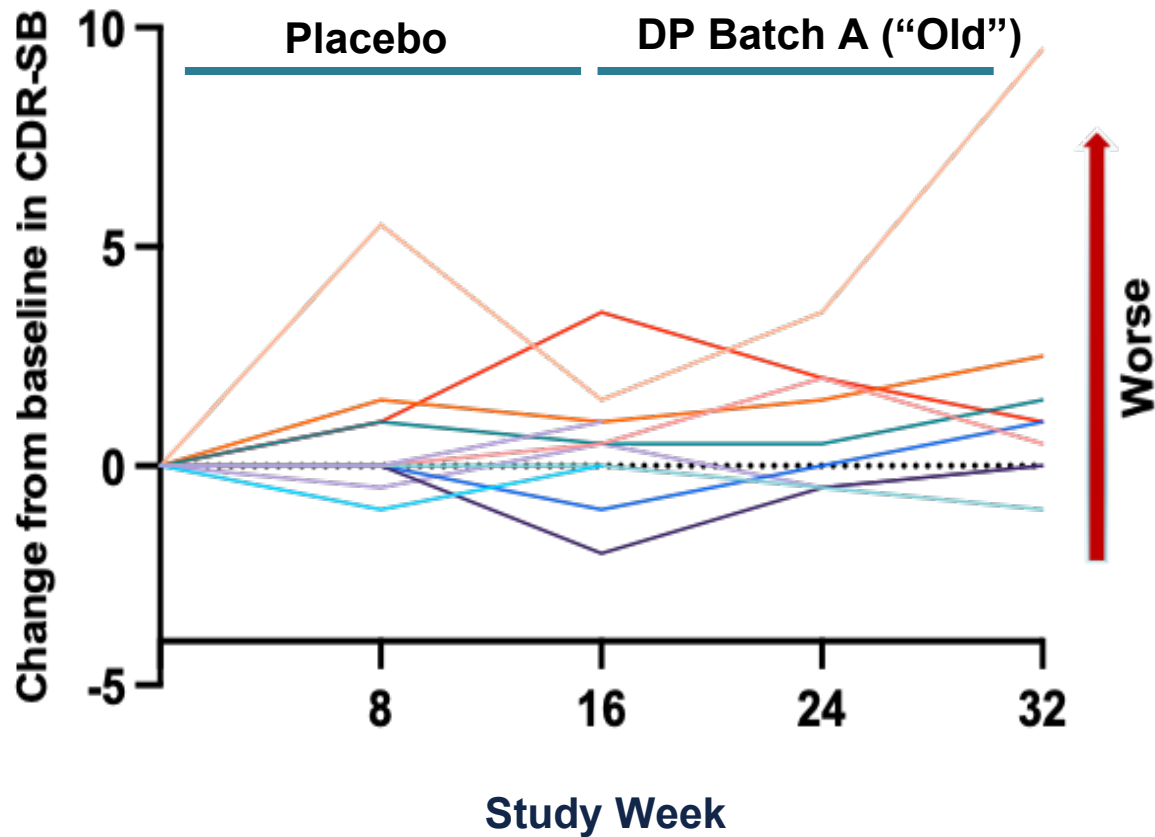
Within-participant comparison of DB Batch A vs. placebo: mean difference = +0.03, p=NS



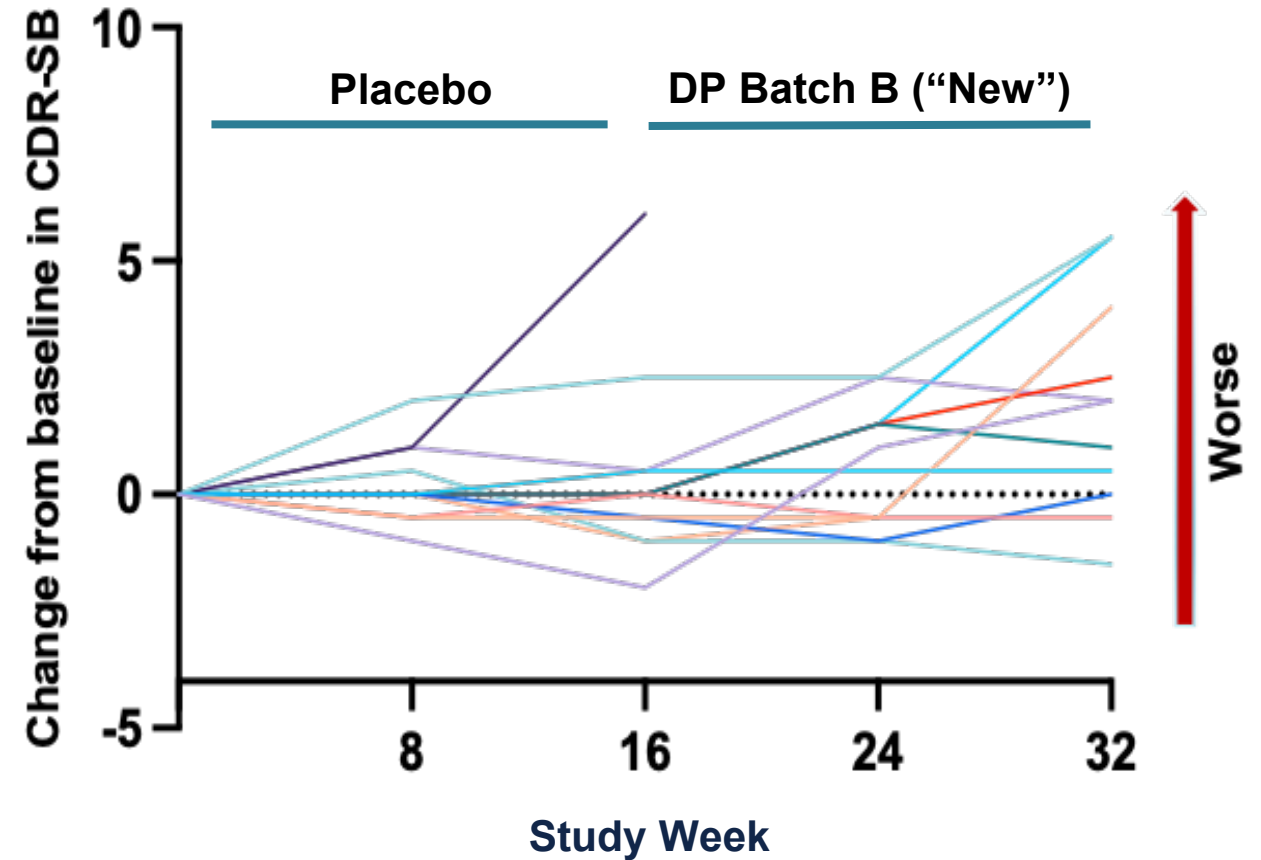
Within-participant comparison of DB Batch B vs. placebo: mean difference = -1.12, p=0.005

No effect of neflamapimod in within-participant comparison to placebo in participants with evidence of AD co-pathology

Change in CDR-SB
(Groups 1 and 2; N=11)

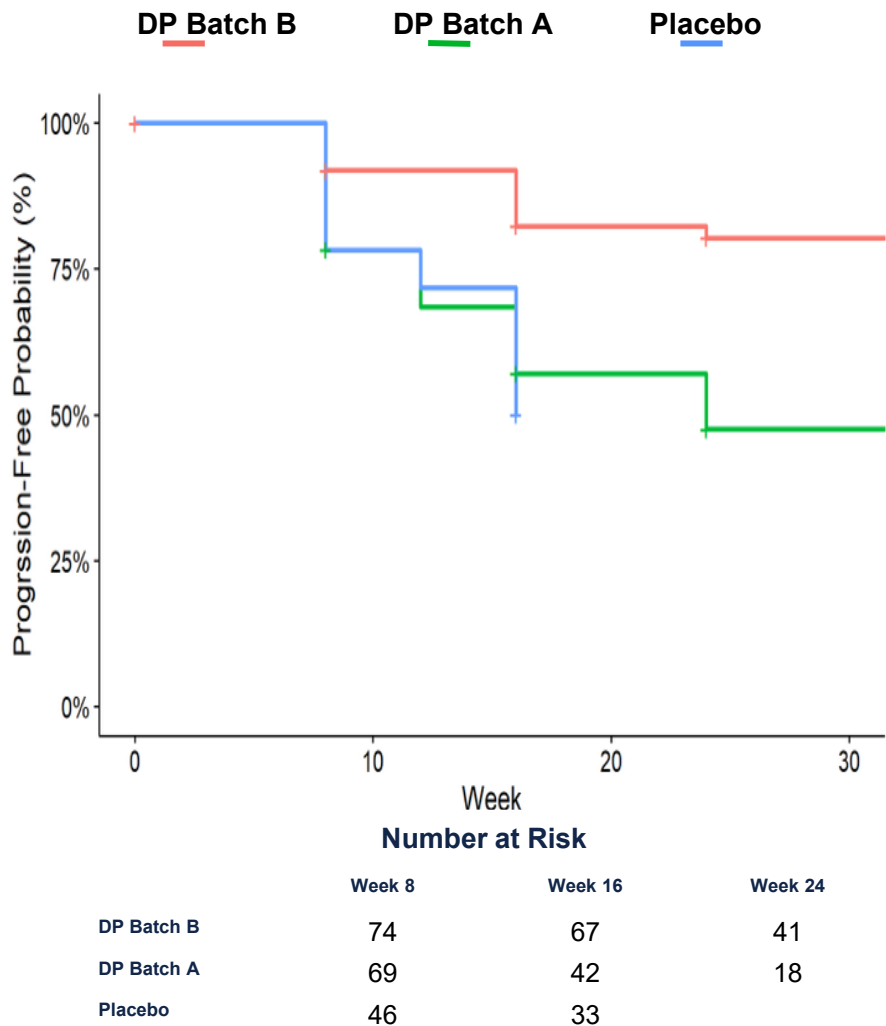


Change in CDR-SB
(Groups 4 and 5; N=14)



Risk of clinically meaningful progression (≥ 1.5 -point increase in CDR-SB) reduced and time to progression increased with neflamapimod treatment

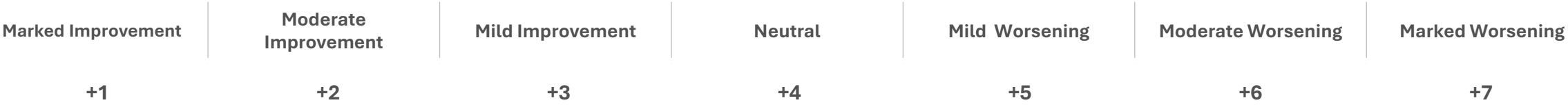
Participants with Screening Plasma pTau181 < 21 pg/mL



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)

Secondary global outcome measure: Alzheimer's Disease Cooperative Study-Clinical Global Impression of Change (ADCS-CGIC)

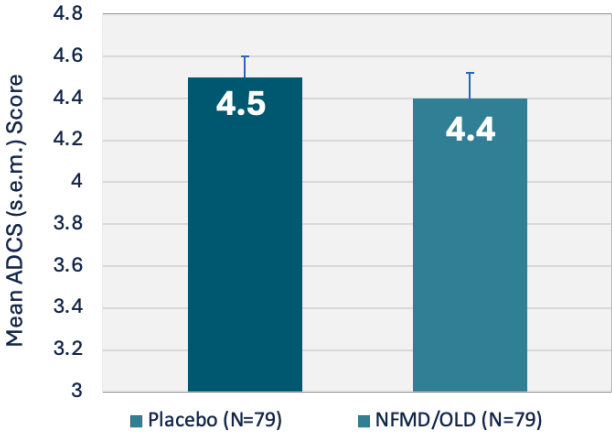


Blinded clinician's assessment of change in clinical status of patient from start of treatment

Calculate Group Means from Individual Scores

Interpretation of Group Mean Scores		
<4.0	4.0	>4.0
Improvement	No Change	Worsening

Results in Initial Phase of the Study

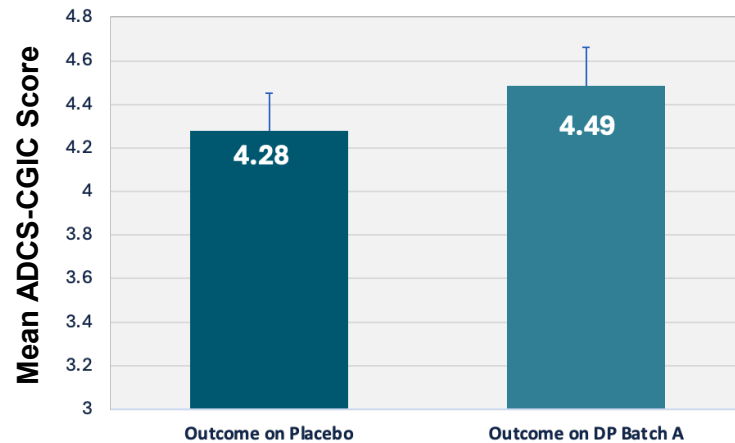


Positive effects on ADCS-CGIC achieved and support CDR-SB results

All
Participants

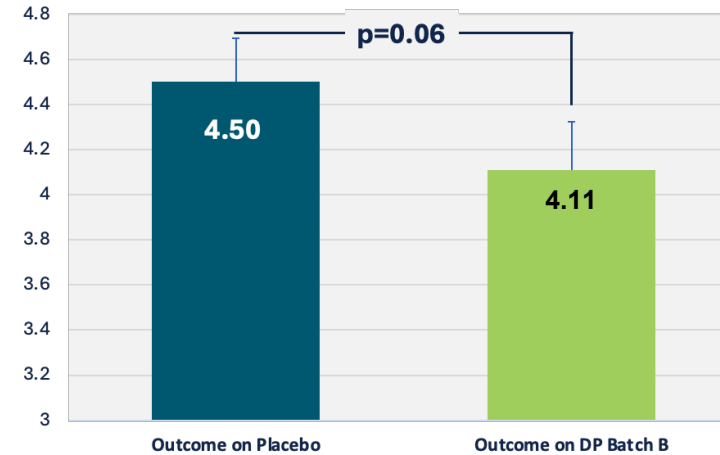
Target Concentration Not Achieved

Participants who received placebo initially and then DP Batch A in the Extension (N=38)



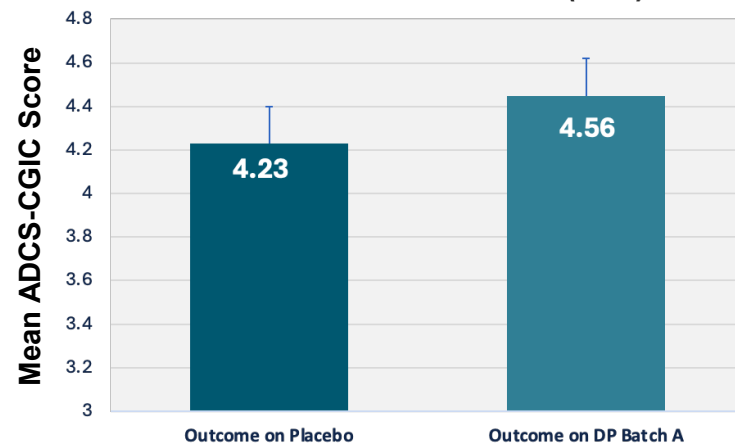
Target Concentration Achieved

Participants who received placebo initially and then DP Batch B in the Extension (N=36)

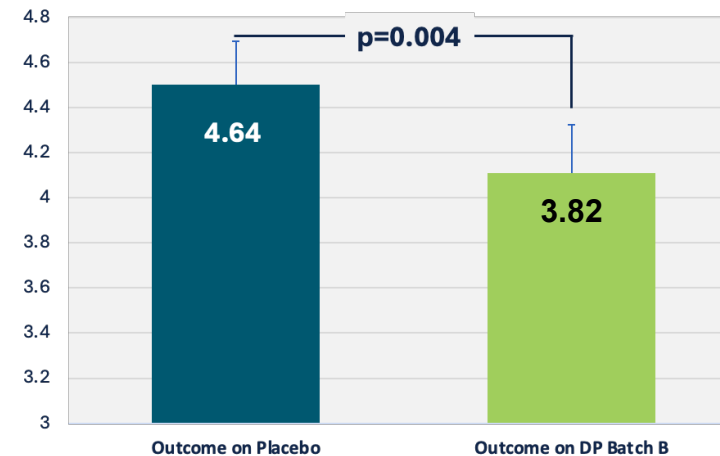


Participants with
Low
Likelihood of AD
Co-Pathology
(Screening plasma
pTau181 <21
pg/mL)

Participants who received placebo initially and then DP Batch A in the Extension (N=22)



Participants who received placebo initially and then DP Batch B in the Extension (N=22)



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

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

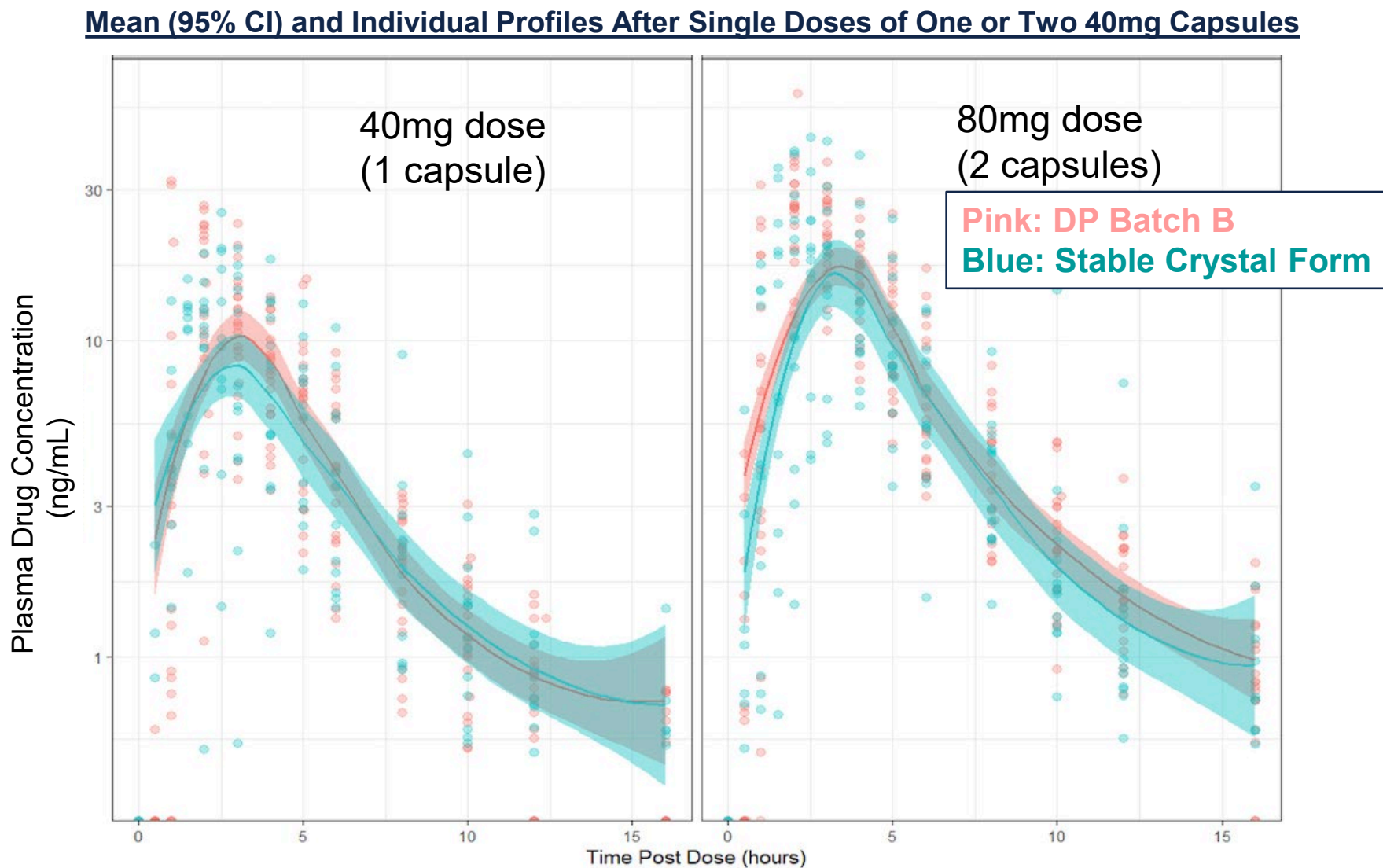


Only adverse event seen at greater than 10% incidence was falls

- 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

New, stable crystal form of neflamapimod supports target plasma exposure

- PK profiles of DP Batch B and the new, stable crystal form of neflamapimod are largely overlapping
- Plan to increase dose to 100mg BID in future trials



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

	Percent of patients who achieve $C_{\text{trough}} \geq 4 \text{ ng/ML}$	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