Results from the Phase 2b AuTonomy Trial of Anti-p-tau Monoclonal Antibody Posdinemab for Early Alzheimer's Disease

Sharon Cohen[1*], Stephen Salloway[2], David Watson[3], Reisa Sperling[4], Ziad S. Saad[5], Maggie Fedgchin[6], Jennifer Bogert[7], Pieter-Jan Berghmans[8], David Henley[9], Lingjue Li[10], Gallen Triana-Baltzer[5], Serge Van der Geyten[8], Haiyan Xu[11], Arthur Simen[12], Fiona Elwood[12], Janice Wong[12]

[1]Toronto Memory Program, Toronto, ON, Canada; [2]Department of Neurology, Butler Hospital, Providence, RI, USA; [3]Alzheimer's Research And Treatment, Wellington, FL, USA; [4]Center for Alzheimer Research and Treatment, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA; [5]Neuroscience Biomarkers, Johnson & Johnson, San Diego, CA, USA; [6]Neuroscience, Johnson & Johnson, Titusville, NJ, USA; [7]Statistics and Decision Sciences, Johnson & Johnson, Raritan, NJ, USA; [8]Neuroscience, Johnson & Johnson, Beerse, Belgium; [9]Hagerstown, IN, USA; employee of Johnson & Johnson at time of the study; [10]Clinical Pharmacology & Pharmacometrics, Johnson & Johnson, Titusville, NJ, USA; [11]Statistics and Decision Sciences, Johnson & Johnson, Titusville, NJ, USA; [12]Neuroscience, Johnson & Johnson, Cambridge, MA, USA.

**Presenting Author*

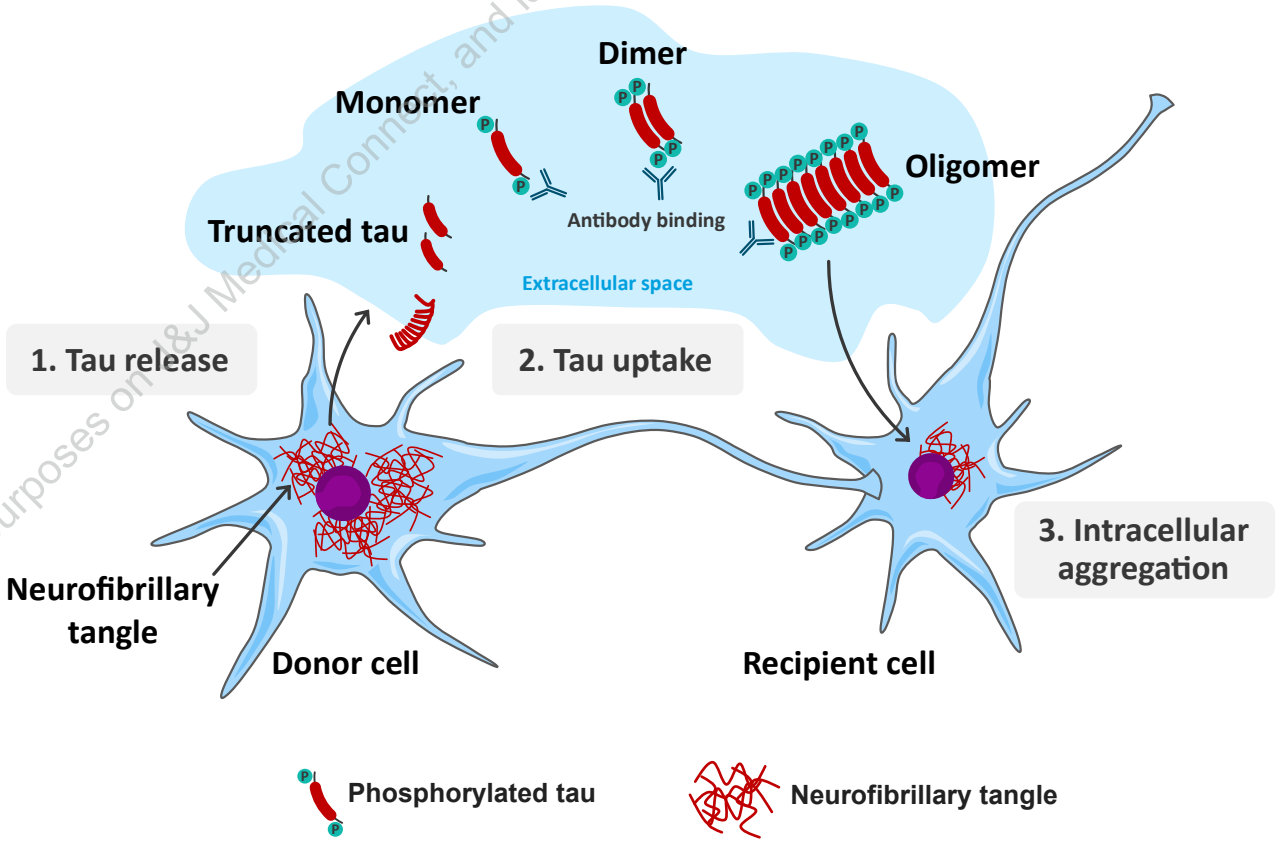
Presented by Dr. Sharon Cohen at the 20th International Conference on Alzheimer's and Parkinson's Diseases (AD/PD™ 2026); March 17–21, 2026; Copenhagen, Denmark

This study was sponsored by Johnson & Johnson

Name	Consulting / Advisory Board	Funded Research
Dr Sharon Cohen	AbbVie, Alzheon, Biogen, BMS, Cognivue, Cogstate, Eisai, Eli Lilly, GSK, INmune Bio, Janssen, Novartis, Novo Nordisk, RetiSpec, Roche	Acumen, Alnylam, Alzheon, Biogen, BMS, Eisai, Eli Lilly, GSK, INmune Bio, Janssen, Merck, Novartis, Novo Nordisk, RetiSpec, Roche, UCB Biopharma, Voyager

INTRODUCTION – Tau is hypothesized to spread via extracellular tau seeds in Alzheimer's disease

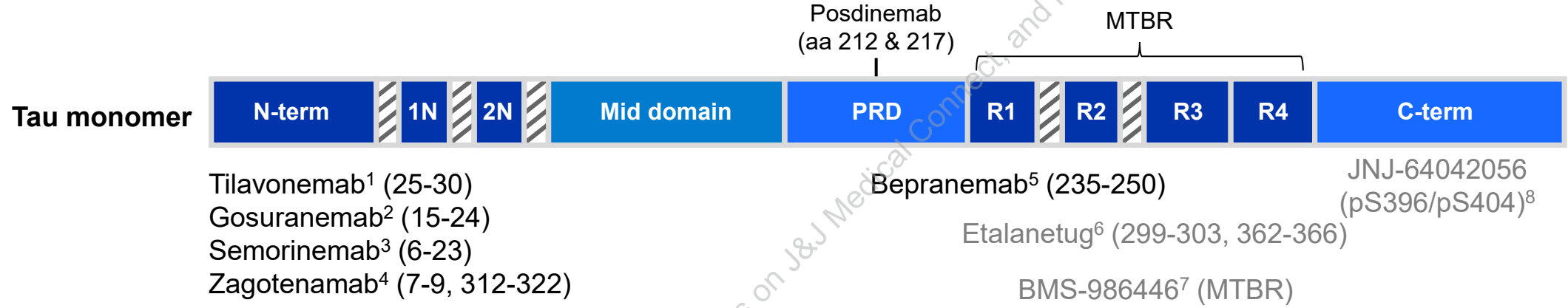
- Intracellular neurofibrillary tangles (NFT) – abnormal aggregates of tau – are pathological hallmarks of Alzheimer's disease (AD) that correlate with cognitive impairment and predict future cognitive decline[1,2]
- It is hypothesized that tauopathy spreads via extracellular release of tau aggregates (tau seeds) that are taken up by previously unaffected neurons and induce NFT formation
- Based on this hypothesis, targeting tau seeds may interfere with tau spread and slow AD progression[3,4]



1. Medeiros R, et al. *CNS Neurosci Ther*. 2010;17(5):514–524; 2. Nelson PT, et al. *J Neuropathol Exp Neurol*. 2012;71(5):362-81. 3. Furman JL, et al. *Acta Neuropathol* 2017;133(1):91–100. 4. Schoonhoven DN, et al. *Brain* 2023;146(10):4040–4054.
AD, Alzheimer's disease; NFT, neurofibrillary tangles.

Presented by Dr. Sharon Cohen at the AD/PD™ Conference; Mar 17-21, 2026; Copenhagen, Denmark

INTRODUCTION- Posdinemab is an anti-tau monoclonal antibody targeting the mid-domain epitope p-tau217

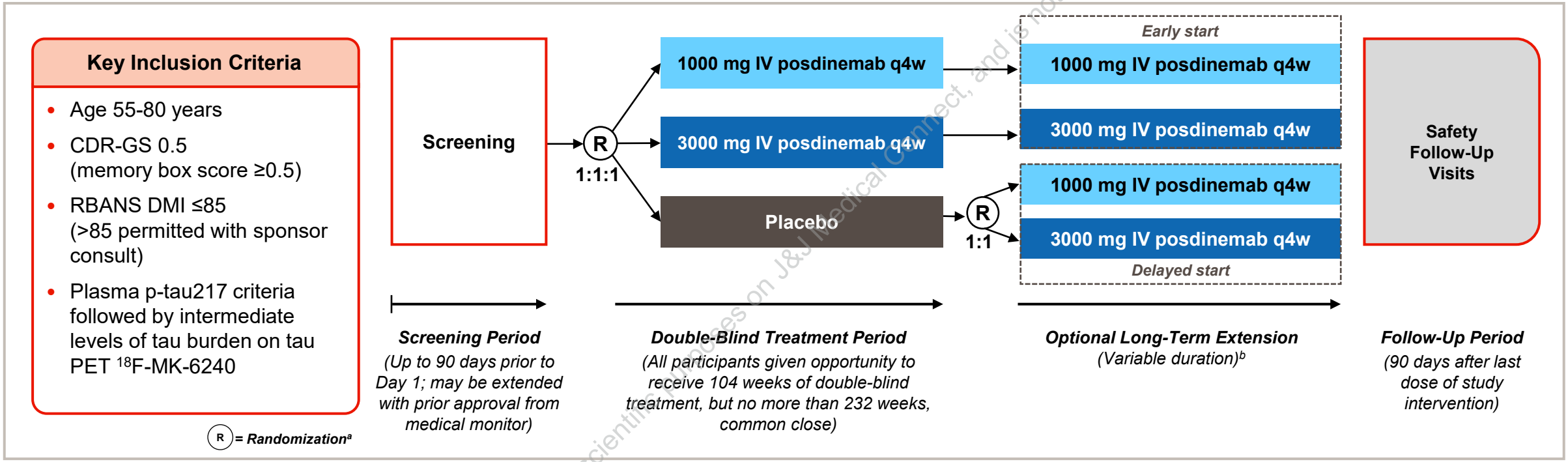


- Posdinemab (JNJ-63733657), an anti-tau IgG1 monoclonal antibody, targets extracellular tau in the mid-domain proline-rich domain (PRD), binding with high affinity to phosphorylated tau and weakly to non-phosphorylated tau[9]
  - The core epitope of tau recognized by posdinemab contains phosphorylation at amino acid 217 (p-tau217), with potentially other phosphorylation sites nearby.
  - Reduced tau seeding in AD brain-derived neuronal assays along with dose-dependent reductions in free CSF p-tau217 observed in completed phase 1 studies of posdinemab suggest engagement of extracellular tau[9-11]

1. Yanamandra K, et al. *Neuron.* 2013;80(2):402-414. 2. Bright J, et al. *Neurobiol Aging.* 2015;36(2):693-709. 3. Teng E, et al. *JAMA Neurol.* 2022; 79(8):758-767. 4. Fleisher AS, et al. *Neurology.* 2024;102(5):e208061. 5. Albert M, et al. *Brain.* 2019;142(6):1736-1750. 6. Roberts M, et al. *Acta Neuropathol Commun.* 2020;8(1):13. 7. van Dyck CH, et al. *Alzheimers Dement.* 2025;20(Suppl 8):e094677. 8. ClinicalTrials.gov. Identifier: NCT06544616. Available at: https://clinicaltrials.gov 9. Galpern WR et al. *J Prev Alzheimers Dis.* 2024;11(6):1592-1603. 10. Van Kolen K, et al. *J Alzheimers Dis.* 2020;77(4):1397-1416. 11. Albert M, et al. *Brain.* 2019;142(6):1736-1750. aa, Amino acid; AD, Alzheimer's disease; CSF, cerebrospinal fluid; C-term, C terminal; iADRS-MCI, integrated Alzheimer's Disease Rating Scale for Mild Cognitive Impairment; IgG: immunoglobulin G; MTBR, microtubule-binding region; N, N-terminal inserts; N-term, N terminal; p-tau217, tau phosphorylated at amino acid residue 217; PRD, proline-rich domain; R, repeat regions.

Presented by Dr. Sharon Cohen at the AD/PD™ Conference; Mar 17-21, 2026; Copenhagen, Denmark

METHODS – Posdinemab was evaluated in early Alzheimer's disease in AuTonomy, a global phase 2b proof-of-concept trial

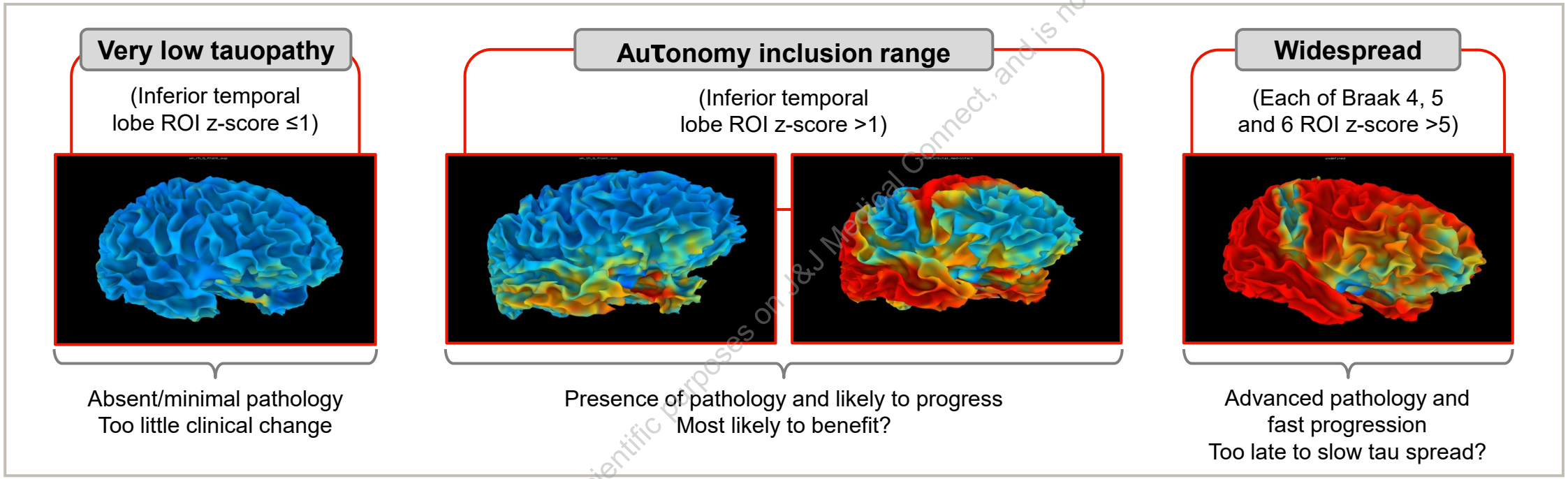


The proof-of-concept, phase 2b AuTonomy study (NCT04619420) evaluated the primary hypothesis that posdinemab would decrease tau spread and thereby slow clinical decline as measured by iADRS-MCI, a composite of cognition and function, as compared with placebo.

[a]Randomization was stratified by geographic region (North America, Europe, Australia, and Japan) and baseline tau burden on tau PET (Braak 4 ROI z-score ≤ or >4.62). [b]Until 2 years after marketing authorization for the indication in a participant's respective country or until posdinemab becomes commercially available or can be provided via another access mechanism or the participant no longer benefits from further treatment or withdraws consent, or the sponsor terminates clinical development. CDR-GS, Clinical Dementia Rating Global Score; DB, double-blind; DMI, Delayed Memory Index; iADRS-MCI, integrated Alzheimer's Disease Rating Scale for Mild Cognitive Impairment; IV, intravenous; p-tau217, tau phosphorylated at amino acid residue 217; q4w, every 4 weeks; R, randomization; RBANS, Repeatable Battery for the Assessment of Neuropsychological Status; ROI, region of interest; tau PET, tau positron emission tomography.

Presented by Dr. Sharon Cohen at the AD/PD™ Conference; Mar 17-21, 2026; Copenhagen, Denmark

METHODS – Tau PET was used for precision selection of participants



After screening cognitive criteria were met, participants underwent precision selection based on plasma p-tau217 followed by tau PET

- Those with very low tau in inferior temporal cortex (with <1 SD from the average in cognitively unimpaired amyloid negative controls) or widespread neocortical tau were excluded

Z-score reference population was cognitively normal amyloid negative older individuals.
PET, positron emission tomography; p-tau217, tau phosphorylated at amino acid residue 217; ROI, region of interest; SD, standard deviation.

Presented by Dr. Sharon Cohen at the AD/PD™ Conference; Mar 17-21, 2026; Copenhagen, Denmark

METHODS – Autonomy study endpoints for the DB treatment period

The following endpoints from the primary DB analysis* will be presented:

Primary endpoint

Change from baseline at Week 104 in
iADRS-MCI total score

Key secondary endpoints

Change from baseline at Week 104 in
- **ADAS-Cog13 total score**
- **ADCS-ADL-MCI total score**

Other secondary endpoints

Change from baseline at Week 104 in
- **CDR-SB**
- **Brain tau burden, as measured by tau PET**
- **CSF concentrations of free p-tau217 fragments**

PK: CSF and serum concentrations of posdinemab

Safety and tolerability

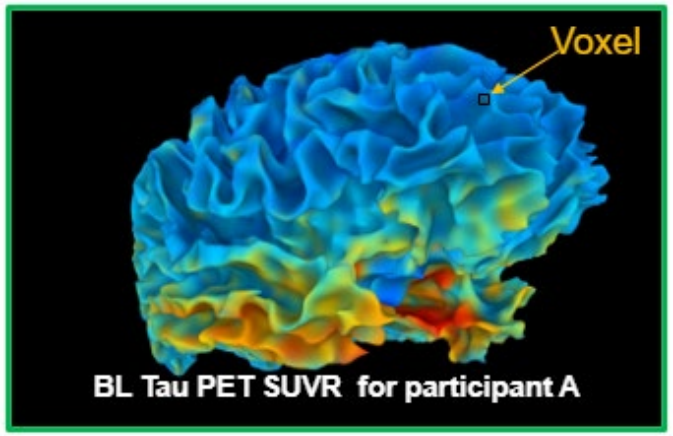
**Primary DB analysis was performed when the last participant completed the Week 104 visit of the DB treatment period*

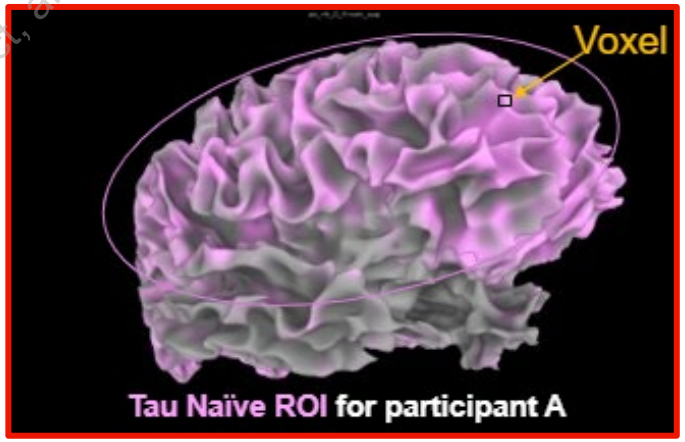
ADCS-ADL-MCI, Alzheimer's Disease Cooperative Study Activities of Daily Living for Mild Cognitive Impairment; ADAS-Cog13, Alzheimer's Disease Assessment Scale - Cognitive, 13-item version; CDR-SB, Clinical Dementia Rating - Sum of Boxes; CSF, cerebrospinal fluid; DB, double-blind; iADRS-MCI, integrated Alzheimer's Disease Rating Scale for Mild Cognitive Impairment; PK, pharmacokinetics; p-tau217, tau phosphorylated at amino acid residue 217; tau PET, tau positron emission tomography.

Presented by Dr. Sharon Cohen at the AD/PD™ Conference; Mar 17-21, 2026; Copenhagen, Denmark

METHODS – Tau naïve region of interest is a novel biomarker for tau spread

Defining participant-specific tau naïve ROI





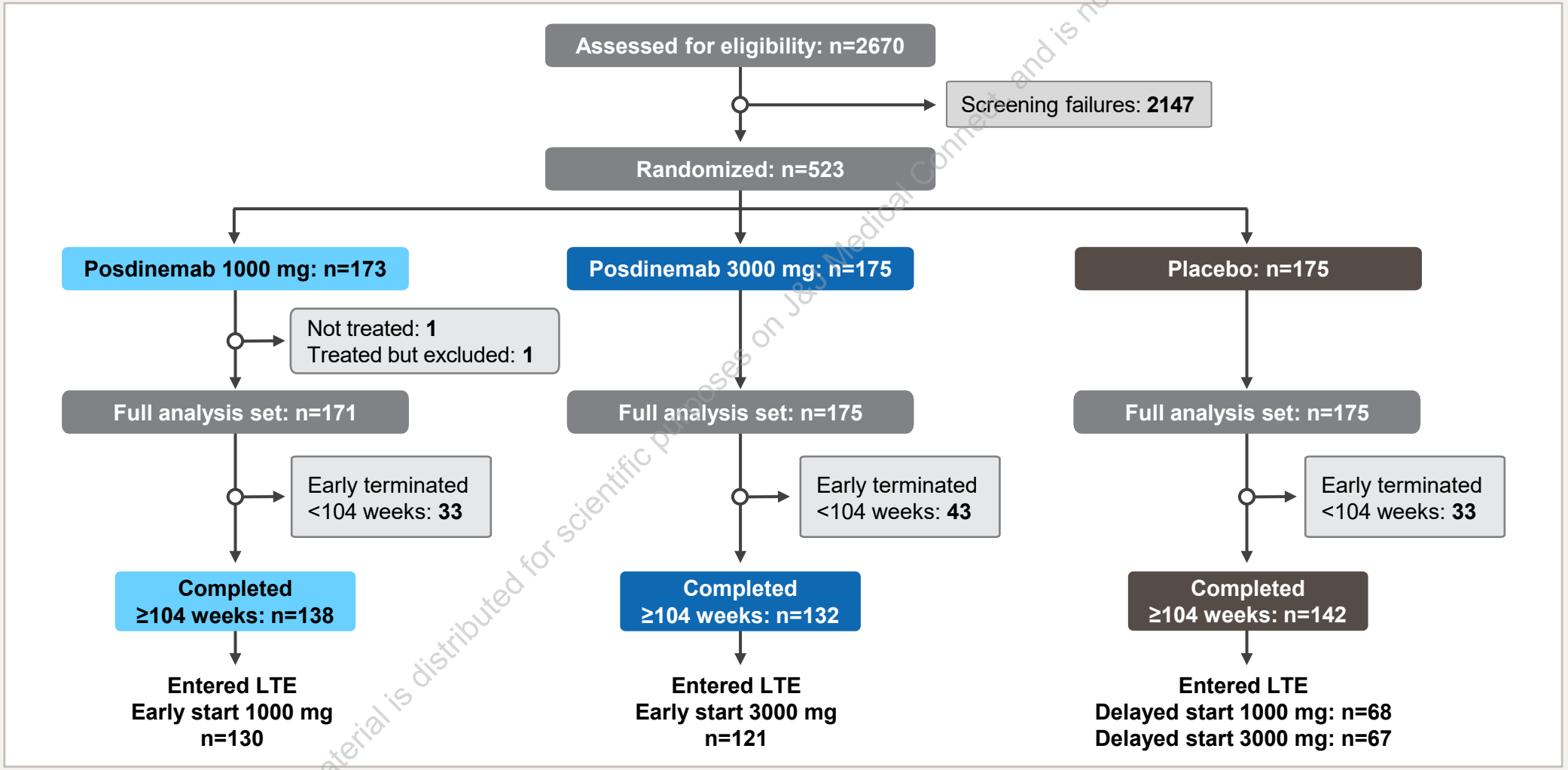
1. Start with a participant's baseline tau PET scan. The color represents the distribution of tau PET. Red and yellow are regions with increased tangles compared to blue regions.
2. Mark a voxel as being tau naïve (regions in pink) if its baseline tau PET is within ±1SD of the average tau PET SUVR at that location in a cognitively unimpaired amyloid negative control cohort.

Tau naïve ROI was designed to measure the spread of tau

- Detects new NFT accumulation by tau PET in brain regions where SUVR was within 1 SD of cognitively normal amyloid-negative controls at baseline

BL, baseline; NFT, neurofibrillary tangles; PET, positron emission tomography; ROI, region of interest; SD, standard deviation; SUVR, standardized uptake value ratio.

RESULTS – Study completion at Week 104 was similar across groups



AD, Alzheimer's Disease; DB, double-blind; LTE, long-term extension.

RESULTS – Baseline characteristics were generally balanced across groups

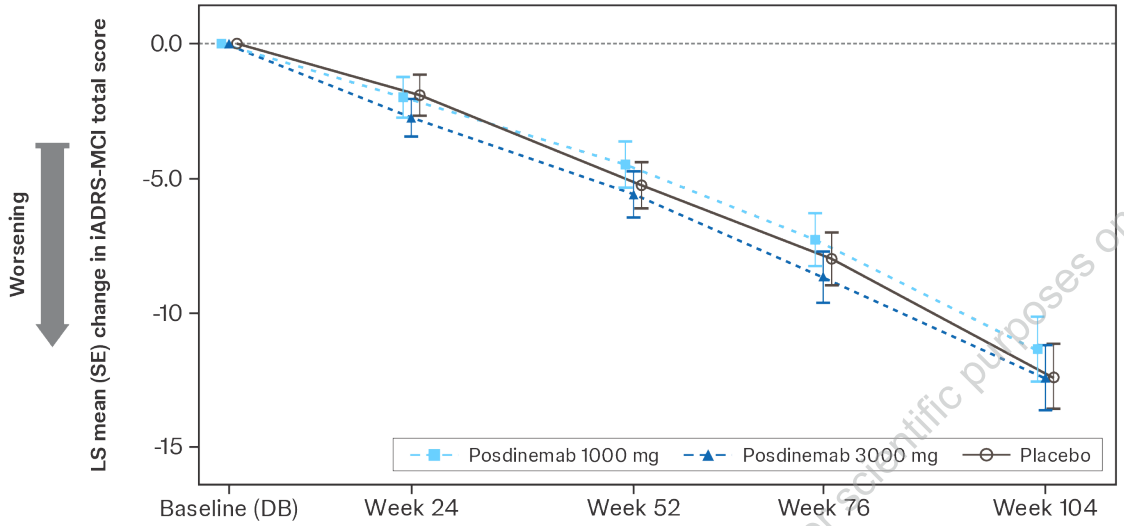
	1000 mg (n=171)	3000 mg (n=175)	Placebo (n=175)	Total (N=521)
Sex (female, %)	89 (52.0)	91 (52.0)	92 (52.6)	272 (52.2)
Age, mean (SD)	71.2 (5.78)	71.5 (5.92)	71.5 (5.90)	71.4 (5.86)
Education level (at least some high school or greater, %)	145 (84.8)	145 (82.9)	157 (89.7)	447 (85.8)
Clinical diagnosis, n (%) MCI due to AD Mild AD dementia	 85 (49.7) 86 (50.3)	 107 (61.1) 68 (38.9)	 103 (58.9) 72 (41.1)	 295 (56.6) 226 (43.4)
Baseline tau burden on tau PET, n (%) Low High	 76 (44.4) 95 (55.6)	 76 (43.3) 99 (56.6)	 77 (44.0) 98 (56.0)	 229 (44.0) 292 (56.0)
Baseline iADRS-MCI total score, mean (SD)	101.41 (11.409)	101.64 (12.004)	102.29 (12.132)	101.78 (11.838)
Baseline ADAS-Cog13 score, mean (SD)	25.50 (7.712)	25.32 (7.946)	24.71 (8.311)	25.18 (7.987)
Baseline ADCS-ADL-MCI score, mean (SD)	41.82 (6.262)	41.96 (6.422)	41.95 (6.170)	41.91 (6.274)
Screening RBANS DMI score, mean (SD)	57.08 (16.265)	56.14 (15.393)	58.54 (17.502)	57.25 (16.409)
Baseline CDR-SB, mean (SD)	2.91 (1.145)	2.92 (1.094)	2.87 (1.202)	2.90 (1.146)
Baseline CDR-GS, n (%) 0 0.5 1	 0 155 (90.6) 16 (9.4)	 1 (0.6) 155 (88.6) 19 (10.9)	 0 163 (93.1) 12 (6.9)	 1 (0.2) 473 (90.8) 47 (9.0)
Use of AchEi and/or memantine at baseline, yes (%)	98 (57.3)	96 (54.9)	110 (62.9)	304 (58.3)
APOE ε4 genotype, n (%) Homozygous carrier Heterozygous carrier Non-carrier	 35 (20.5) 89 (52.0) 47 (27.5)	 34 (19.4) 85 (48.6) 56 (32.0)	 31 (17.7) 94 (53.7) 50 (28.6)	 100 (19.2) 268 (51.4) 153 (29.4)

Analysis set- Full efficacy analysis set: all randomized participants who receive ≥1 dose of study intervention in the double-blind treatment period. AD, Alzheimer's disease; ADCS-ADL-MCI, Alzheimer's Disease Cooperative Study Activities of Daily Living for Mild Cognitive Impairment; ADAS-Cog13, Alzheimer's Disease Assessment Scale - Cognitive, 13-item version; APOE, apolipoprotein E; CDR-GS, Clinical Dementia Rating - Global Score; CDR-SB, Clinical Dementia Rating - Sum of Boxes; DB, double-blind; iADRS-MCI, integrated Alzheimer's Disease Rating Scale for Mild Cognitive Impairment; MCI, mild cognitive impairment;
SD, standard deviation; tau PET, tau positron emission tomography.

Presented by Dr. Sharon Cohen at the AD/PD™ Conference; Mar 17-21, 2026; Copenhagen, Denmark

RESULTS – No significant difference between posdinemab and placebo in the primary endpoint iADRS-MCI total score and secondary endpoint CDR-SB

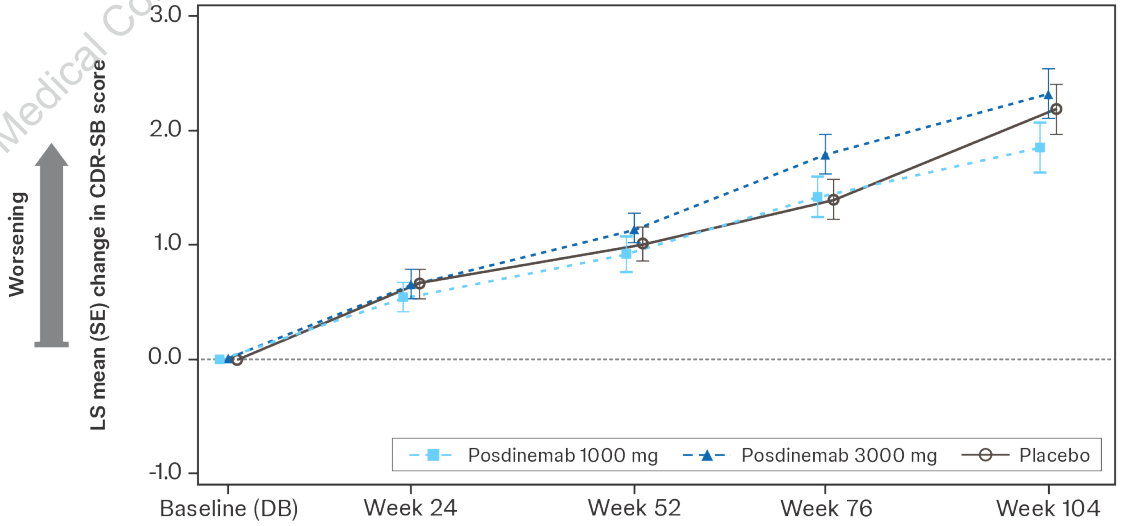
Least squares mean change from baseline in iADRS-MCI total score (full efficacy analysis set)



Number of participants

	Baseline (DB)	Week 24	Week 52	Week 76	Week 104
Posdinemab 1000 mg	170	165	152	144	142
Posdinemab 3000 mg	174	166	151	143	132
Placebo	172	166	153	147	137

Least squares mean change from baseline in CDR-SB score (full efficacy analysis set)



Number of participants

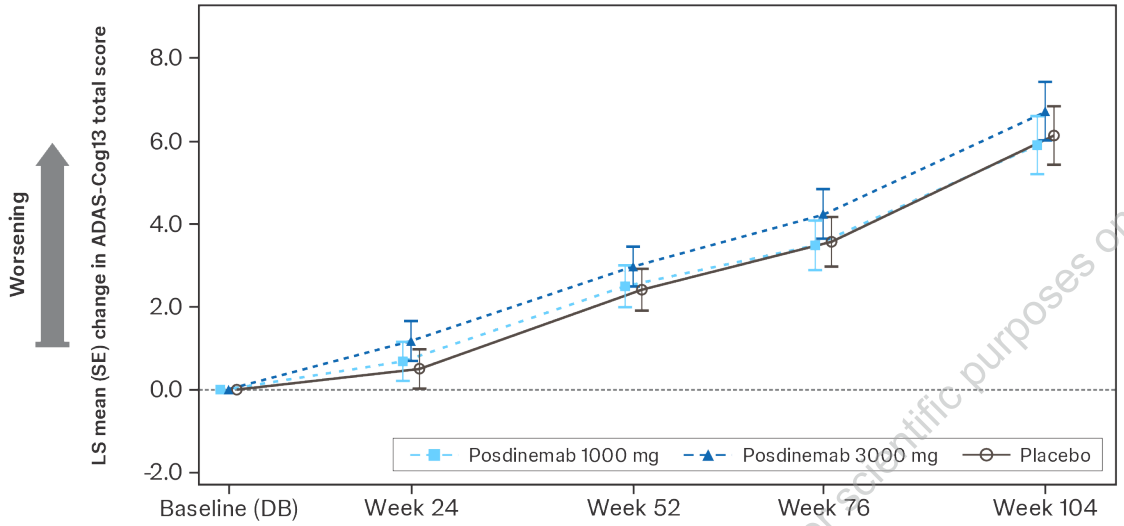
	Baseline (DB)	Week 24	Week 52	Week 76	Week 104
Posdinemab 1000 mg	171	167	153	147	143
Posdinemab 3000 mg	175	165	155	148	137
Placebo	175	170	155	153	144

LS mean change from baseline and SE are derived using MMRM with factors for treatment, visit, treatment-by-visit interaction, geographic region, sex, APOE ε4 carrier status, education level, AChEI and/or memantine use at baseline, and covariates for baseline score, baseline tau burden on tau PET, and age at baseline. Full efficacy analysis set: all randomized participants who receive ≥1 dose of study intervention in the double-blind treatment period. AChEI, acetylcholinesterase inhibitors; APOE ε4, apolipoprotein E ε4; CDR-SB, Clinical Dementia Rating - Sum of Boxes; DB, double-blind; iADRS-MCI, integrated Alzheimer's Disease Rating Scale for Mild Cognitive Impairment; LS, least squares; MMRM, mixed model for repeated measures; SE, standard error; tau PET, tau positron emission tomography

Presented by Dr. Sharon Cohen at the AD/PD™ Conference; Mar 17-21, 2026; Copenhagen, Denmark

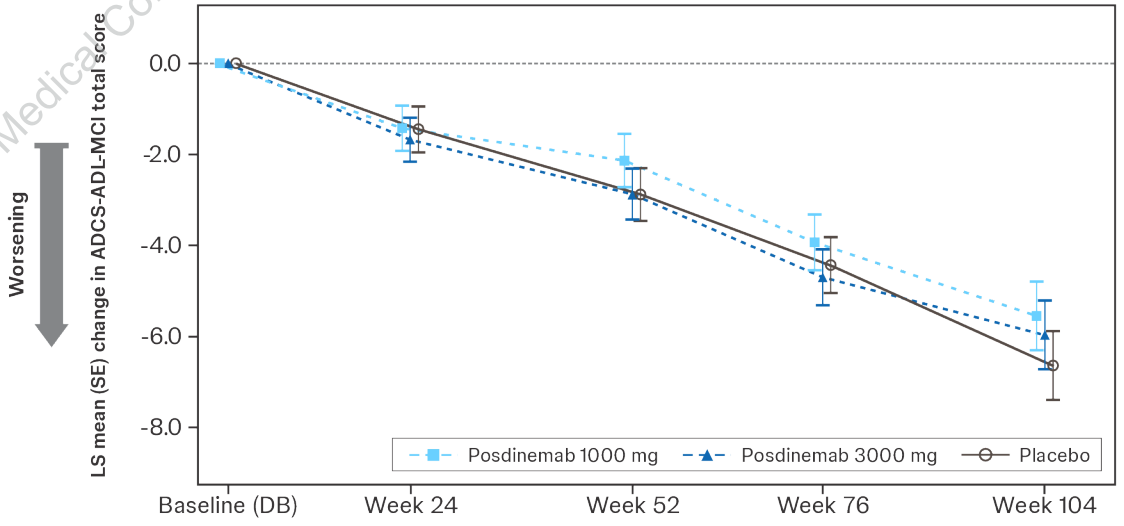
RESULTS – No significant difference between posdinemab and placebo in the key secondary endpoints ADAS-Cog13 and ADCS-ADL-MCI total scores

Least squares mean change from baseline in ADAS-Cog13 total score (full efficacy analysis set)



Number of participants	Baseline (DB)	Week 24	Week 52	Week 76	Week 104
Posdinemab 1000 mg	170	166	153	146	142
Posdinemab 3000 mg	174	166	153	143	133
Placebo	173	167	157	150	140

Least squares mean change from baseline in ADCS-ADL-MCI total score (full efficacy analysis set)



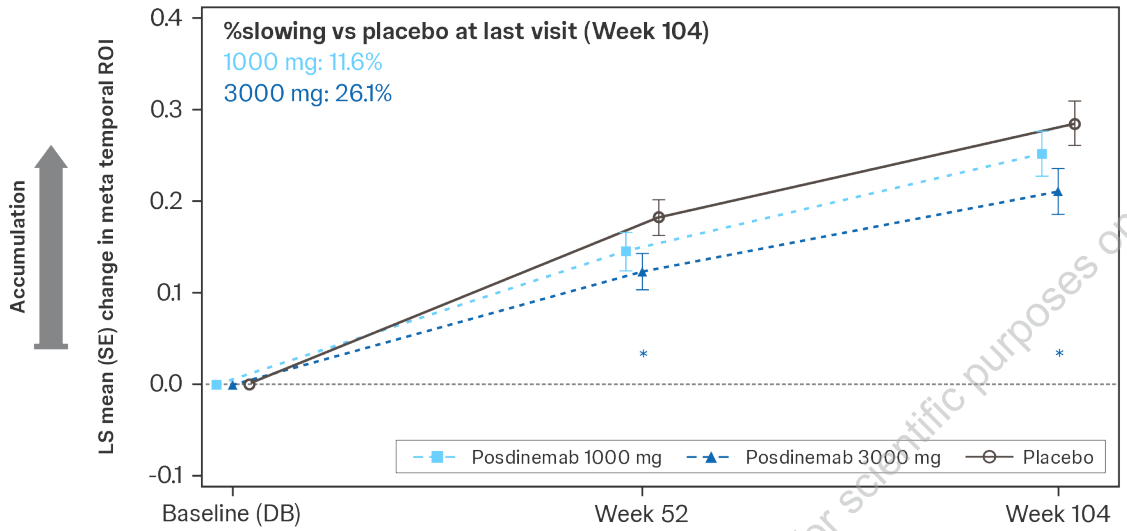
Number of participants	Baseline (DB)	Week 24	Week 52	Week 76	Week 104
Posdinemab 1000 mg	171	166	153	149	144
Posdinemab 3000 mg	174	167	153	146	136
Placebo	173	168	155	150	141

LS mean change from baseline and SE are derived using MMRM with factors for treatment, visit, treatment-by-visit interaction, geographic region, sex, APOE ε4 carrier status, education level, AChEI and/or memantine use at baseline, and covariates for baseline score, baseline tau burden on tau PET, and age at baseline. Full efficacy analysis set: all randomized participants who receive ≥1 dose of study intervention in the double-blind treatment period. AChEI, acetylcholinesterase inhibitors; APOE ε4, apolipoprotein E ε4; ADCS-ADL-MCI, Alzheimer's Disease Cooperative Study Activities of Daily Living for Mild Cognitive Impairment; ADAS-Cog13, Alzheimer's Disease Assessment Scale - Cognitive, 13-item version; DB, double-blind; LS, least squares; MMRM, mixed model for repeated measures; SE, standard error; tau PET, tau positron emission tomography.

Presented by Dr. Sharon Cohen at the AD/PD™ Conference; Mar 17-21, 2026; Copenhagen, Denmark

RESULTS – Tau PET demonstrated slowing of NFT increase in high tau burden areas but not in tau naïve ROI

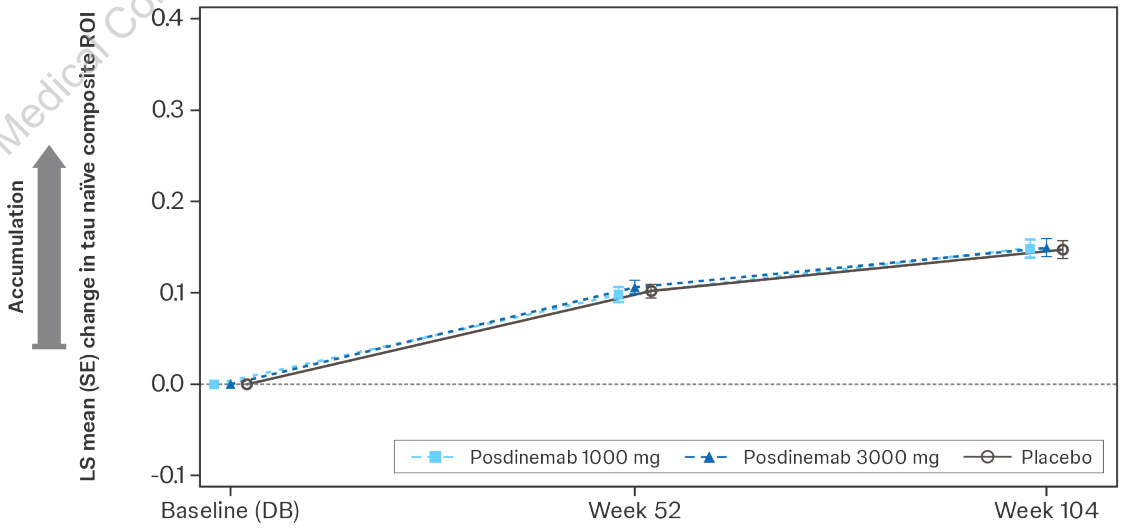
LS mean change from baseline in meta-temporal ROI SUVR



Number of participants

	Baseline (DB)	Week 52	Week 104
Posdinemab 1000 mg	170	147	138
Posdinemab 3000 mg	175	154	130
Placebo	175	155	137

LS mean change from baseline in tau naïve ROI SUVR



Number of participants

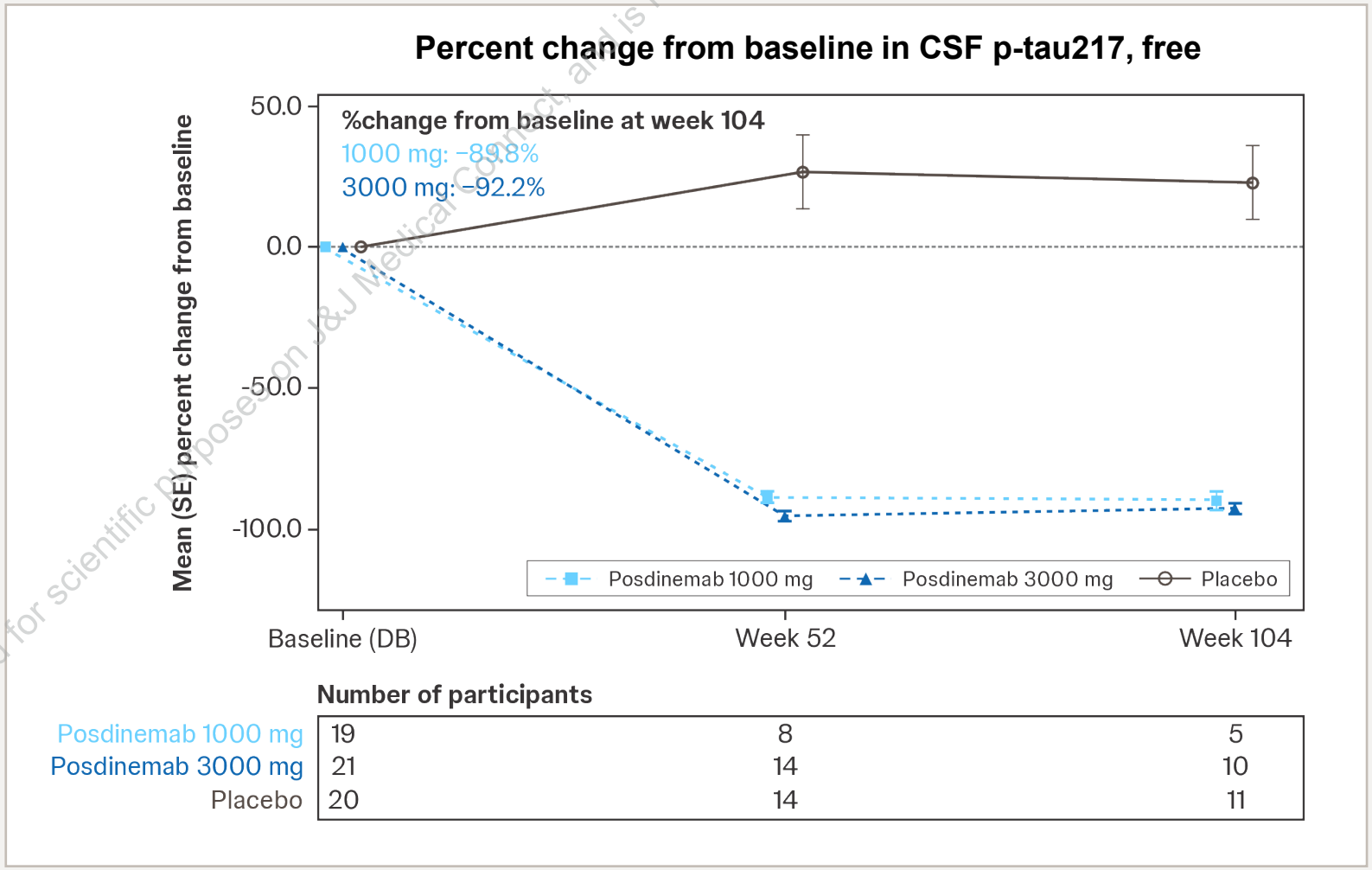
	Baseline (DB)	Week 52	Week 104
Posdinemab 1000 mg	170	147	138
Posdinemab 3000 mg	175	154	130
Placebo	175	155	137

*indicates nominal p-value for posdinemab 3000 mg versus placebo <0.05 (non-gated). LS mean change from baseline, SE, and p-value are derived using MMRM with factors for treatment, visit, treatment-by-visit interaction, geographic region, sex, APOE ε4 carrier status, and covariates for baseline SUVR, baseline tau burden on tau PET, and age at baseline. APOE ε4, apolipoprotein E ε4. DB, double-blind; LS, least squares; MMRM, mixed model for repeated measures; NFT, neurofibrillary tangles; ROI, region of interest; SE, standard error; SUVR, standardized uptake value ratio; tau PET, tau positron emission tomography.

Presented by Dr. Sharon Cohen at the AD/PD™ Conference; Mar 17-21, 2026; Copenhagen, Denmark

RESULTS – No indication that the lack of efficacy was due to insufficient dose

- **Pharmacodynamics:** Robust reductions were observed in CSF free p-tau217 for both doses.
- **Pharmacokinetics:** Serum and CSF exposure was in line with predictions from previously completed phase 1 studies.[1,2]



1. Galpern WR et al. *J Prev Alzheimers Dis*. 2024;11(6):1592-1603. 2. Wang F, et al. *J Clin Pharmacol*. 2026;66(2):e70116.
CSF, cerebrospinal fluid; DB, double-blind; p-tau217, tau phosphorylated at amino acid residue 217; SE, standard error.

Presented by Dr. Sharon Cohen at the AD/PD™ Conference; Mar 17-21, 2026; Copenhagen, Denmark

RESULTS – Overall TEAE rates were similar across groups

Event, n (%)	1000 mg (n=171)	3000 mg (n=175)	Placebo (n=175)
TEAE leading to death	1 (0.6)[a]	2 (1.1)[b]	2 (1.1)[c]
Serious TEAE	34 (19.9)	42 (24.0)	43 (24.6)
Study drug-related serious TEAE	3 (1.8)	4 (2.3)	1 (0.6)
TEAE leading to permanent discontinuation of study treatment	13 (7.6)	8 (4.6)	9 (5.1)
Study drug-related TEAE	42 (24.6)	47 (26.9)	42 (24.0)
Any TEAE	158 (92.4)	163 (93.1)	165 (94.3)
Mild	70 (40.9)	63 (36.0)	76 (43.4)
Moderate	68 (39.8)	75 (42.9)	72 (41.1)
Severe	20 (11.7)	25 (14.3)	17 (9.7)

Most common TEAEs (1000 mg vs 3000 mg vs placebo) were generally balanced across groups

- COVID-19: 25.7% vs 21.7% vs 20.0%
- Fall: 14.0% vs 14.9% vs 18.9%
- Headache: 12.3% vs 12.6% vs 10.3%

Note: Participants are counted only once for any given event, regardless of the number of times they actually experienced the event. Analysis set- safety analysis set: all randomized participants who received ≥1 dose of DB study intervention. Adverse events are coded using MedDRA version 28.0. [a]Dementia Alzheimer's type (n=1); [b]Amyotrophic lateral sclerosis (n=1), mixed dementia (n=1); [c]Colon cancer recurrence (n=1), aortic dissection rupture, (n=1); MedDRA, Medical Dictionary for Regulatory Activities; n, number; TEAE, treatment-emergent adverse event.

Presented by Dr. Sharon Cohen at the AD/PD™ Conference; Mar 17-21, 2026; Copenhagen, Denmark

RESULTS – Posdinemab was not associated with cerebral amyloid angiopathy or infusion reactions, but increased risk for hepatic enzyme elevations

Incidence of MRI safety findings (safety analysis set)

Finding, n (%)	1000 mg		3000 mg		Placebo	
	Baseline (n=171)	DB phase (n= 164)	Baseline (n=175)	DB phase (n=168)	Baseline (n=175)	DB phase (n=172)
Vasogenic edema	0	0	0	5 (3.0)	0	6 (3.5)
Microhemorrhage, any	29 (17.0)	49 (29.9)	31 (17.7)	51 (30.4)	34 (19.4)	51 (29.7)
Microhemorrhage, >4	2 (1.1)	4 (2.4)	2 (1.1)	5 (3.0)	4 (2.3)	10 (5.8)
Superficial siderosis	7 (4.1)	14 (8.5)	4 (2.3)	7 (4.2)	7 (4.0)	10 (5.8)
Macrohemorrhage (>10mm)[a]	0	0	0	0	0	1 (0.6)

Other TEAEs (safety analysis set)

Participants with ≥1 event, n (%)	1000 mg (n=171)	3000 mg (n=175)	Placebo (n=175)
Infusion related reaction[b]	3 (1.8)	7 (4.0)	9 (5.1)
ALT or AST ≥3xULN	8 (4.7)*	8 (4.6)*	1 (0.6)

*16 of 17 participants with ALT or AST ≥3xULN were on posdinemab treatment; all cases had ALT and AST values return to normal range by time of study completion. Of these 16 participants, 3 had SUSARs of DILI (n=1) or suspected DILI (n=2), which resolved after treatment was stopped.

Note: Participants are counted only once for any given event, regardless of the number of times they actually experienced the event. Adverse events are coded using MedDRA version 28.0. Safety analysis set: all randomized participants who received ≥1 dose of DB study intervention. [a]In total there were 4 serious adverse events of intracerebral or intraventricular hemorrhage during the double-blind treatment period: n=2,1000 mg posdinemab; n=1, 3000 mg posdinemab; n=1,placebo. The MRI safety findings table includes only one macrohemorrhage because it is limited to centrally read MRIs (performed every six months per protocol) and does not include local MRI results.[b]Infusion reaction: AEs occurring on the same day as a study intervention dose with either a preferred term of 'Infusion related reaction' or AEs indicated on the AE eCRF as AESI of 'Infusion Reaction'. AE, adverse event; AESI, adverse event of special interest; ALT, alanine transaminase; AST, aspartate aminotransferase; DB, double-blind; DILI, drug-induced liver injury; eCRF: electronic case report form; MedDRA, Medical Dictionary for Regulatory Activities; SUSAR, suspected unexpected serious adverse reaction; TEAEs, treatment-emergent adverse events; ULN, upper limit of normal.

CONCLUSIONS

- AuTonomy study did not meet primary or key secondary endpoints, demonstrating that posdinemab did not slow clinical decline in early AD.
- A precision approach was utilized to enroll participants with tau pathology and to thoroughly assess tau accumulation and tau spread via tau PET.
- Tau PET demonstrated slowing of accumulation of NFT in high tau burden areas, however, this did not impact clinical efficacy. Of note, there was no slowing in the tau naïve ROI.
- Despite reductions in CSF p-tau217 suggesting robust target engagement in the CSF, no clinical efficacy was observed.
- Treatment was generally safe and well-tolerated; however, an increased risk of hepatic enzyme elevation was observed.

AD, Alzheimer's disease; CSF, cerebrospinal fluid; NFT, neurofibrillary tangles; PET, positron emission tomography; ptau217, tau phosphorylated at amino acid residue 217; ROI, region of interest.

Presented by Dr. Sharon Cohen at the AD/PD™ Conference; Mar 17-21, 2026; Copenhagen, Denmark

IMPLICATIONS

- Based on findings from the phase 2b AuTonomy study, clinical development of posdinemab in AD has been discontinued.
- These results do not invalidate tau as a therapeutic target in AD. Instead, they highlight the complexity of tau biology and the need to re-examine premises about how anti-tau monoclonal antibodies interact with NFTs.
- These data deepen our understanding of AD and will help guide future therapeutic research and development.
- Analyses from the AuTonomy study are being conducted to provide further insights into tau biology and targeting.

AD, Alzheimer's disease; NFT, neurofibrillary tangles.

Presented by Dr. Sharon Cohen at the AD/PD™ Conference; Mar 17-21, 2026; Copenhagen, Denmark

ACKNOWLEDGEMENTS

We sincerely thank the study participants, their caregivers and families, investigators, study-site teams, and partners whose dedication and collaboration made the AuTonomy study possible. Their extraordinary commitment continues to move the field forward.