ProMIS™ Neurosciences

Targeting underlying causes of neurodegenerative diseases



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Data for interim analysis presented as of July 22, 2026

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WHO WE ARE

A clinical-stage biotech targeting the misfolded proteins that drive neurodegenerative disease

Our pipeline of antibodies and vaccines are designed to selectively target the specific, disease-causing misfolded forms of proteins.

OUR FOCUS

Protein misfolding

Targeting only the toxic, misfolded forms of disease-causing proteins

OUR PLATFORM

EpiSelect™

Computational tool that predicts the misfold and identifies the exposed epitope

OUR LEAD ASSET

Phase 1b

PRECISE-AD trial in early Alzheimer's
Topline data expected in Q1 2027

BALANCE SHEET

Cash runway through 2027, well beyond Q1 2027 PRECISE-AD topline data

NASDAQ: PMN · Cambridge, MA

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THE PROBLEM

7.4 million Americans are living with Alzheimer’s today, and that number could nearly double to 13.8M by 2060



1 in 9

Americans age 65 and older has Alzheimer’s

Each one is a parent, a spouse or a friend losing memory, independence and the ability to recognize the people they love. Much of their care falls on family.

THE SCALE OF THE PROBLEM

13.8M	**Americans 65+ projected by 2060** Absent treatments that slow or prevent progression
1 in 3	**Older Americans die with dementia** More than breast and prostate cancer combined
$409B	**U.S. care costs in 2026** Excluding the unpaid care given by families
~47M	**Americans with preclinical Alzheimer’s[1]** No symptoms yet; not addressed by current labels

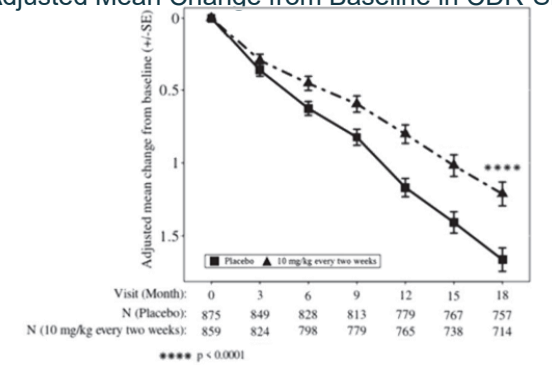
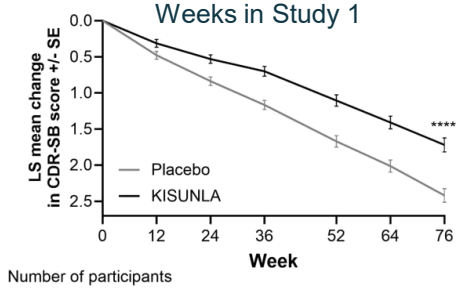
Our pursuit: design precision therapies against the toxic proteins that drive Alzheimer's Disease (AD)

PMN310 is designed to target **only** toxic Aβ oligomers, an early driver of synaptic loss, neuroinflammation, and memory loss in AD.

Source: Alzheimer’s Association, 2026 Alzheimer’s Disease Facts and Figures (Americans 65+). 1. Brookmeyer R et al., Alzheimer’s Dement 2018;14:121–129 (2017 estimate; amyloid and/or neurodegeneration; many will not progress to dementia).

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Anti-amyloid plaque therapies already sell ~$1.4B a year despite significant safety risks and a boxed warning

	Clinical Benefit (CDR-SB)	Boxed Warning: ARIA	Annual Sales Ests.(1)
Leqembi *(lecanemab)* *Approved 2023*	Adjusted Mean Change from Baseline in CDR-SB in Study 2 	FDA BOXED WARNING **~13%** ARIA-E (brain swelling)(2) **~17%** ARIA-H (microbleeds)(2)	21% CAGR(1) ~$2.8B ~$0.7B 2026 2033E
Kisunla *(donanemab)* *Approved 2024*	CDR-SB Change From Baseline in Combined Population Through 76 Weeks in Study 1  Number of participants Placebo 838 825 784 752 713 678 672 KISUNLA 794 774 731 682 650 603 598	FDA BOXED WARNING **~16%** ARIA-E (brain swelling)(3) **~25%** ARIA-H (microbleeds)(3)	23% CAGR(1) ~$2.9B ~$0.7B 2026 2033E

Demand is proven
Uptake keeps growing despite the safety risk.

A safer option could widen use

1. 2026: annualized Q2 2026 in-market sales (Leqembi $184M, Biogen; Kisunla $167M, Lilly). 2033E: GlobalData, October 2026. CAGR 2026–2033E calculated by ProMIS. 2. Leqembi Prescribing Information. 3. Kisunla Prescribing Information, July 2025; Eli Lilly press release, July 9, 2025; PharmExec.

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An estimated ~95% of eligible U.S. patients remain untreated, facing safety, access and cost barriers

Market opportunity

~1.26M are early-stage and label-eligible today[2]



~95%
untreated (~1.2M patients)

~5%
treated (~63K patients)[2]

~$32B/yr
U.S. drug value of untreated eligible patients at list price[3]

~$60–70B
analyst sizing of the next-gen U.S. MCI market[4]

Top obstacles to current anti-amyloid therapies

1. **ARIA (boxed warning)**
   ARIA-E in ~13% (lecanemab) and ~16% (donanemab); 40–50% of APOE4 homozygotes experience ARIA[5]
2. **Other side effects**
   Infusion reactions in 26% (lecanemab) and 9% (donanemab)[6]
3. **MRI monitoring and IV infusion**
   Access to MRI and infusion-center infrastructure required[7]
4. **Contraindications**
   Patients on blood thinners face higher risk of brain hemorrhage; APOE4 homozygotes face the highest ARIA risk and are excluded from EU approvals[5]
5. **Out-of-pocket costs**
   Estimated ~$5–7K/yr for a Medicare patient without supplemental coverage, plus infusion and MRI fees[8]

Demand is proven
~$1.4B in annualized sales despite these barriers.[1]

PMN310 designed to target the biggest barriers

1. Annualized Q2 2026 global in-market sales (Leqembi $184M per Biogen; Kisunla $167M per Lilly). 2. Monfared AAT et al., Neurol Ther 2025 (Eisai-sponsored U.S. model): 1,259,263 eligible, 62,963 treated. 3. Untreated patients × Leqembi U.S. first-year list price (~$26,500/yr); illustrative, ProMIS calculation. 4. TD Cowen via Axios, Sept 28, 2026: $60B if 75% of ~3.8M U.S. MCI patients are treated at $20K/yr, $70B by 2035. 5. Leqembi and Kisunla Prescribing Information (Kisunla rev. July 2025, modified titration); Wang H et al., J Prev Alzheimers Dis 2025 (TRAILBLAZER-ALZ 6, 18-month ARIA-E 15.6%); Smith EE et al., J Prev Alzheimers Dis 2025 (APOE4 homozygote ARIA, EU exclusion). 6. van Dyck CH et al., NEJM 2023; Sims JR et al., JAMA 2023. 7. Cummings J et al., Lecanemab appropriate use recommendations, J Prev Alzheimers Dis 2023. 8. Medicare Part B 20% coinsurance × CMS Part B ASP payment limits, Q4 2026 (Leqembi ~$26.5K/yr; Kisunla ~$33K/yr, modified titration, year 1); ProMIS calculation.

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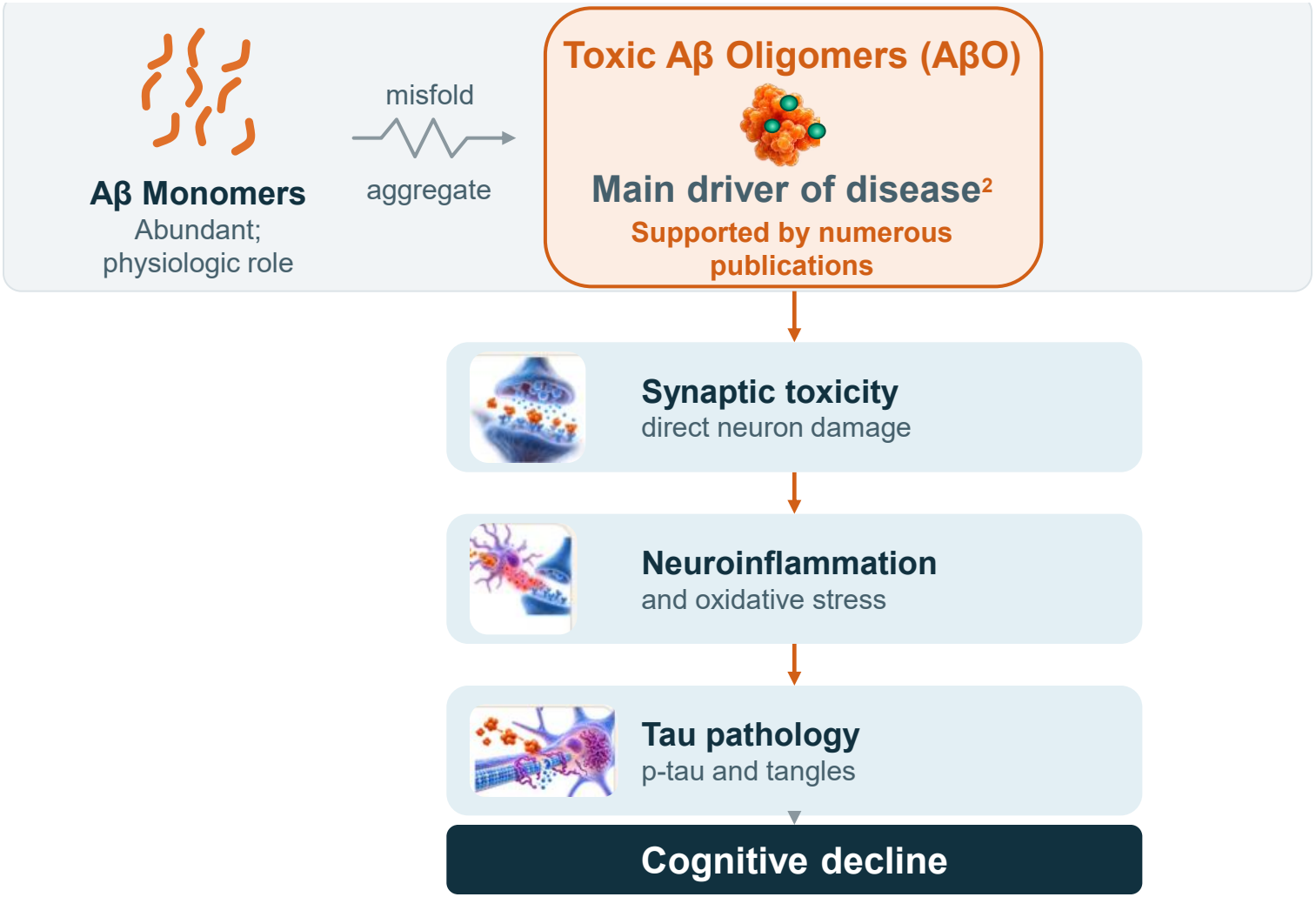
Lead Clinical Candidate PMN310 in Alzheimer's Disease

Selectivity for
Toxic Aβ Oligomers



ProMIS™ Neurosciences

Neutralizing toxic AβOs upstream can reduce downstream damage



PMN310 acts upstream of the pathological cascade

Binds toxic AβOs selectively, avoiding monomers and plaque

IgG1 effector function engages microglia to clear the bound oligomers

CSF oligomer reduction demonstrated in Phase 1a (sFIDA assay)

Potential to inhibit the downstream processes that lead to cognitive decline

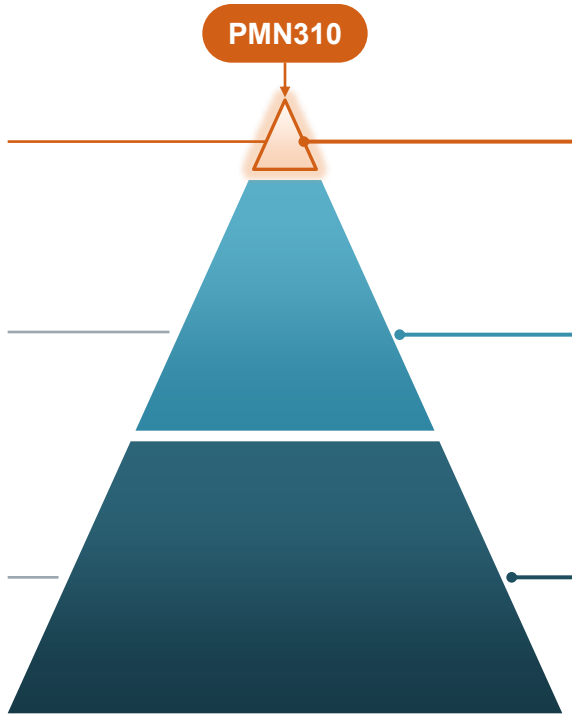
1.The Amyloid Cascade Hypothesis. Castellani, Rudy J. et al. The American Journal of Pathology, Volume 195, Issue 11, Nov 2025

2.Cline EN, et al. The Amyloid-β Oligomer Hypothesis: Beginning of the Third Decade. J Alzheimers Dis. 2018;64(s1):S567–S610

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Toxic Aβ oligomers: low abundance, high toxicity, the target of PMN310 in Alzheimer' disease

ILLUSTRATIVE RELATIVE ABUNDANCE OF Aβ SPECIES[1]



Toxic Aβ Oligomers
Small, soluble clusters of Aβ
Main driver of disease

Aβ Plaque
Large, insoluble aggregates of Aβ

Aβ Monomers
Abundant, produced over lifetime

Illustrative; not to scale

Target: Toxic Aβ Oligomers, the primary driver of disease
Present in **picomolar quantities**; low abundance means less antibody is needed for target saturation

Avoid: sink effect and ARIA risk
Antibody binding to plaque soaks up dose and can result in ARIA

Avoid: sink effect
Present in **nanomolar quantities**; monomer binding adds no efficacy and can lead to a sub-therapeutic dose

PMN310 achieved CSF concentrations 100–600x greater than the target in Ph1a

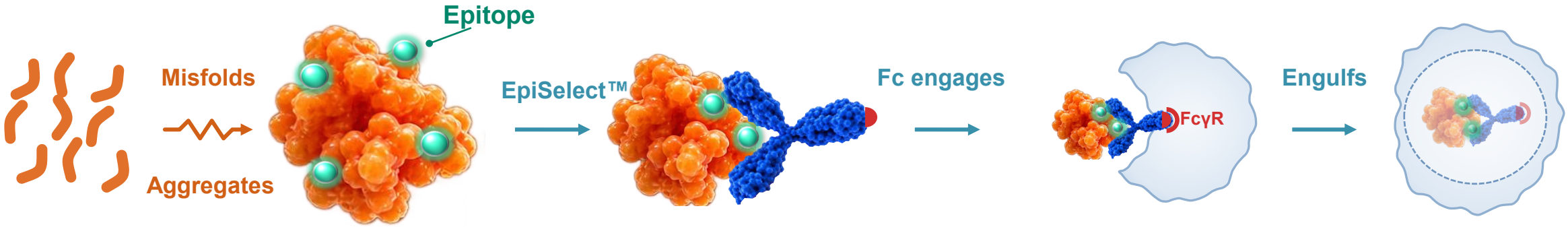
1. 1. Goure WF, Krafft GA, Jerecic J, Hefti F. Targeting the proper amyloid-beta neuronal toxins: a path forward for Alzheimer's disease immunotherapeutics. Alzheimers Res Ther. 2014 Jul 9

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EpiSelect™ computational platform allows for the generation of antibodies selective for toxic misfolded proteins

EpiSelect™ computational platform
Predicts the misfold and identifies the exposed epitope

IgG1 effector function
Fc recruits microglia for clearance



Normal protein
(monomers)

Toxic oligomer
Conformational epitope exposed

PMN310 binds
only the toxic-form epitope

Microglia binding
FcγR captures the Fc portion

Complex removed
Microglia engulf and clear oligomers

The most toxic AβOs are low molecular weight and unstable:
their shape keeps changing

Illustrations are schematic. 1. Gibbs E et al., Sci Rep 2019;9:9870. Selectivity: Kaplan JM et al., Alzheimer's Dement TRCI 2025;11:e70184.

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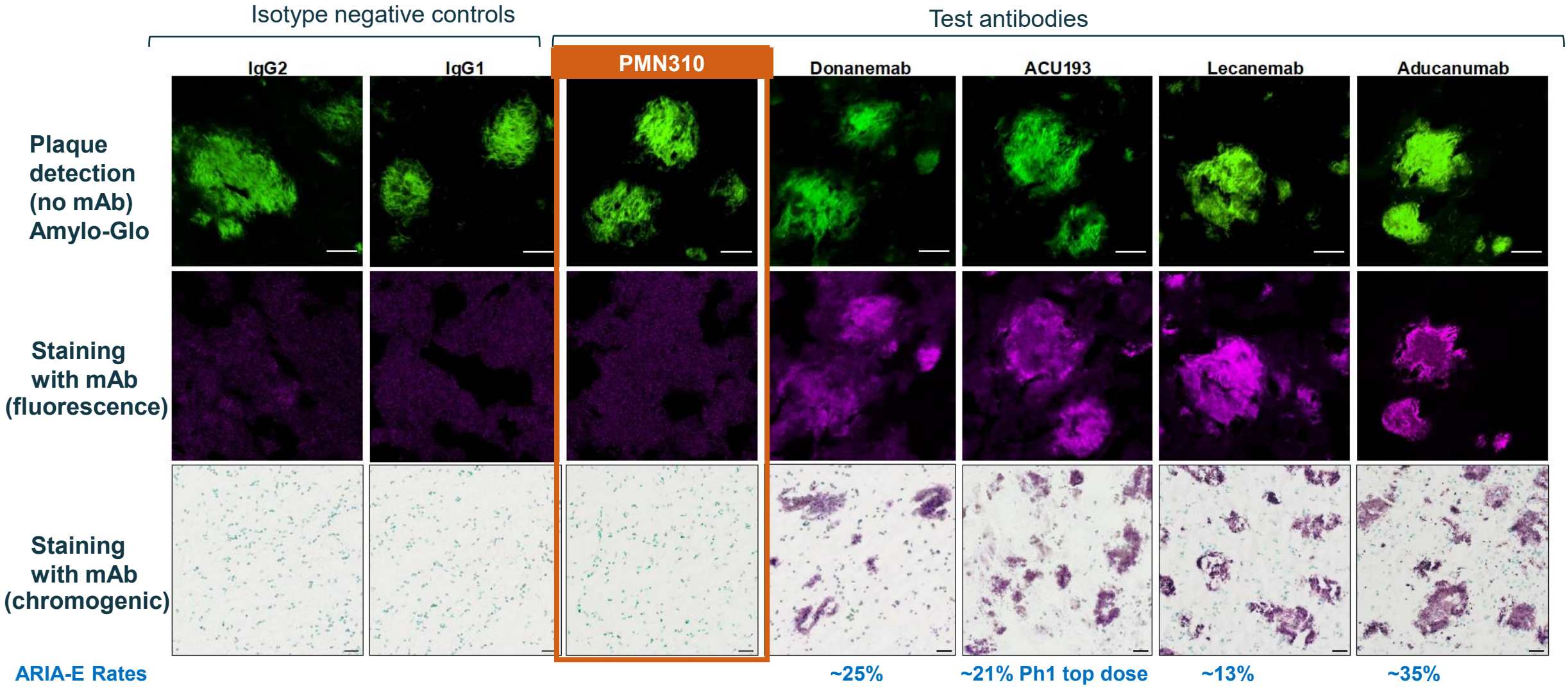
PMN310 is the only IgG1 mAb designed to solely target toxic amyloid-beta (Aβ) oligomers

	PMN310 (ProMIS) *Phase 1b ongoing*	Lecanemab (Eisai) *Approved*	Donanemab (Eli Lilly) *Approved*	Sabirnetug (Acumen) *Phase 2*
Target binding *Plaque binding is linked to ARIA-E. Cross-reactivity reduces oligomer targeting.*	**Toxic Aβ oligomers only**	Aβ Oligomers and Plaques	Aβ Oligomers and Plaques	Aβ Oligomers and Plaques
Isotype *Determines effector function;* *IgG1 – neutralizes, actively clears* *IgG2 – neutralizes only*	**IgG1 · active clearance**	IgG1 · active clearance	IgG1 · active clearance	IgG2 · passive clearance *Weak effector function*
ARIA-E rate *Main safety risk of the class*	**0% ARIA-E**[1] 4.4% ARIA-H, all mild	12.6% ARIA-E[2] 17.3% ARIA-H	15.6% ARIA-E[3] 20.3% ARIA-H	~10% ARIA-E[4] Details not disclosed

Sources: 1. ProMIS PRECISE-AD interim, Jul 28, 2026; 2. van Dyck et al., NEJM 2023 (Clarity AD); 3. Wang et al., Alzheimer's & Dementia 2025 and JPAD 2025 (TRAILBLAZER-ALZ 6); 4. Acumen ALTITUDE-AD topline, Oct 30, 2026; company disclosures (isotype). Note: These data are derived from different clinical trials at different points in time, with differences in trial design, including endpoints, and patient populations. As a result, cross-trial or product comparisons cannot be made, it is only provided for illustrative purposes, and no head-to-head clinical trials have been conducted.

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PMN310 was the only mAb tested with undetectable plaque binding through fluorescent and chromogenic staining

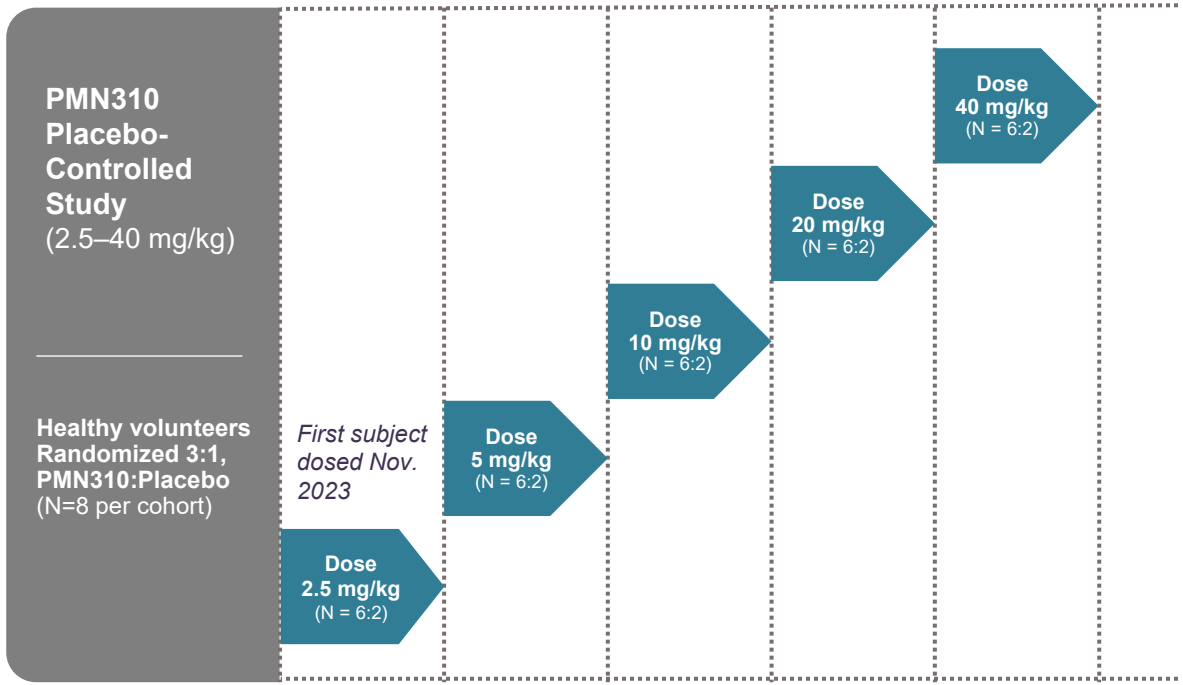


Staining conducted using 1.0 µg/mL concentration of each antibody; similar results achieved at 0.1 and 0.3 µg/mL of each antibody and quantification of plaque stain fluorescent intensity across concentrations can be provided upon request

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Phase 1a first-in-human single ascending dose (SAD) study met all objectives and informed Phase 1b design and dosing

Phase 1a SAD Trial Design (40 healthy volunteers, 5 dose levels)



Per protocol, doses fixed per cohort with a 70 kg body weight assumption

Key Findings

- PMN310 was generally well tolerated in healthy volunteers across all five dose levels tested
- **Linear dose-proportionality of PMN310 CSF levels exhibited**
- **CSF half-life was ~27 days**, supporting once-monthly dosing, and potential subcutaneous formulation
- PMN310 crossed the BBB, reaching **CSF concentrations of 100–600 times the estimated CSF AβO molar concentration**
- **Achieved CSF levels believed to be required for target saturation** without a brain shuttle

SAD, Single Ascending Dose; MAD, Multiple Ascending Dose; AD, Alzheimer's Disease

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PMN310 reduced Aβ oligomers in CSF in Phase 1a, measured directly by the sFIDA assay

What it measures

Aβ oligomer particles in CSF, the target of PMN310

How it works

- Captures oligomers and detects them with a fluorescence-tagged antibody
- Signal scales with particle size

Why it matters

- Femtomolar sensitivity
- A direct pharmacodynamic readout of target engagement

Phase 1a: PMN310 reaches its target in CSF[1]

- Oligomer reduction detected by sFIDA at Days 3 and 29, in healthy volunteers with low baseline levels

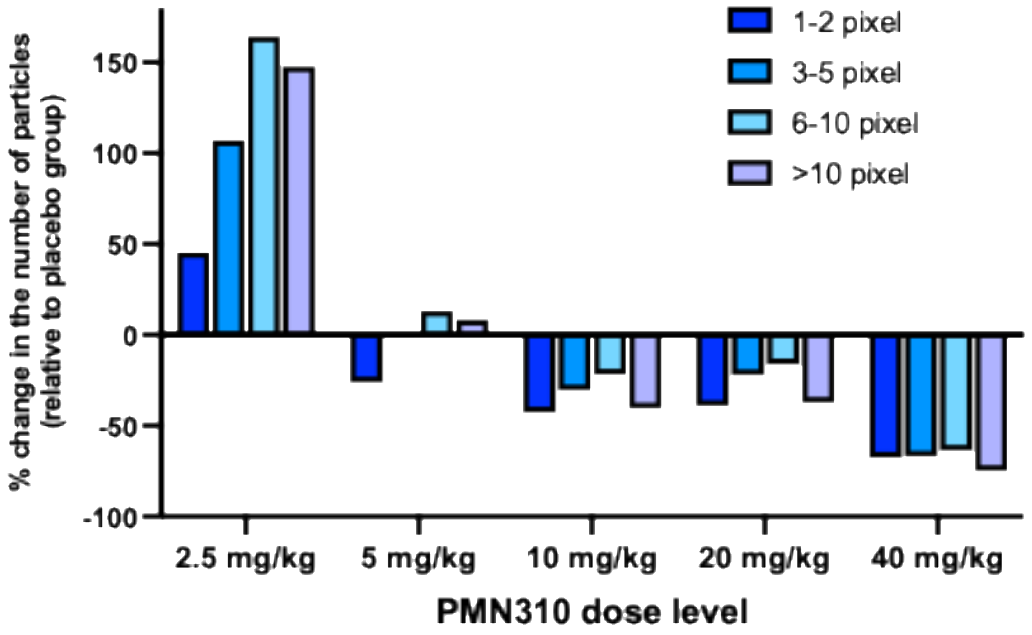
First evidence consistent with PMN310's mechanism of action

sFIDA = surface-based fluorescence intensity distribution analysis (attyloid GmbH, Düsseldorf); exploratory assay. 1. Kaplan et al. Poster, Alzheimer's Association International Conference (AAIC), July 2026. Phase 1a SAD: NCT06105528, 40 healthy volunteers.

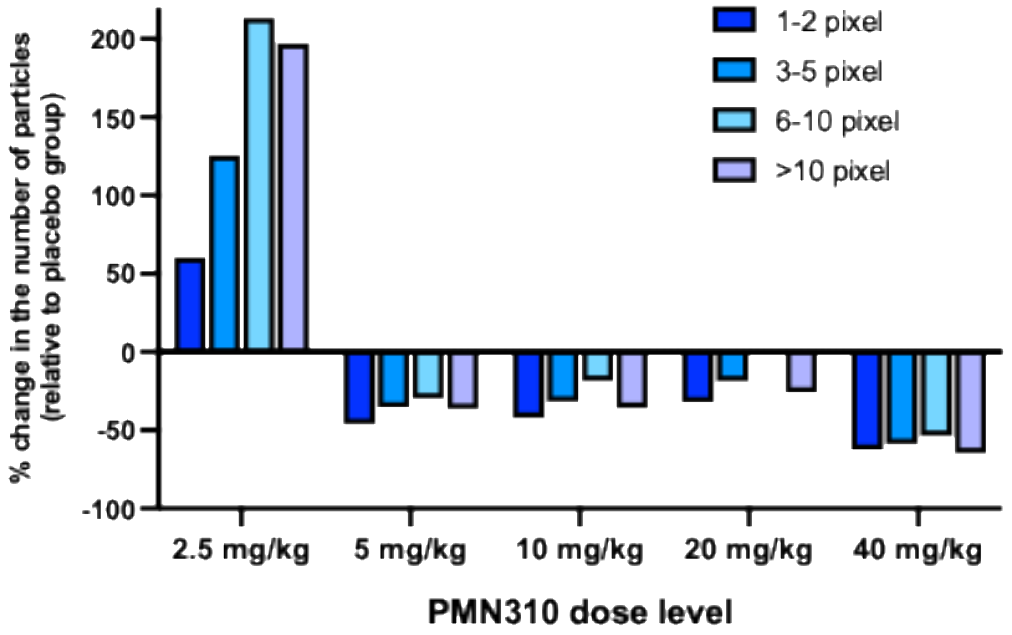
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PMN310 produced a dose-dependent reduction in CSF Aβ oligomer particles

Day 3 post-dose



Day 29 post-dose



First human evidence of Aβ oligomer target engagement by PMN310

- Relative to placebo, the number of detectable oligomer particles declined at doses of 5–40 mg/kg across all particle sizes, at both Day 3 and Day 29 post-dose.
- Exploratory assay, but the first direct pharmacodynamic evidence that PMN310 engages and removes the oligomer species it was designed to target.

sFIDA analysis of CSF (attyloid GmbH); number of oligomer particles by size vs. placebo. Phase 1a SAD, NCT06105528, N = 40 healthy volunteers. Exploratory endpoint; presented at AAIC 2026 (Kaplan et al.).

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PRECISE-AD Interim Analysis Results

Presented July 28, 2026

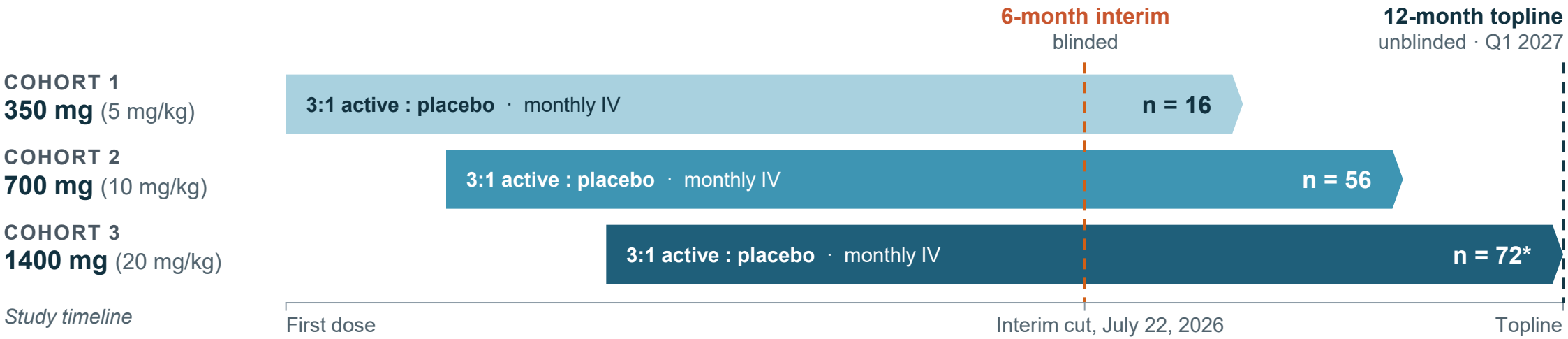
1. Demographics and Study Design
2. Safety Results
3. Biomarker Trends

TRIAL DESIGN

PRECISE-AD: Phase 1b with FDA Fast Track

Randomized, double-blind, placebo-controlled multiple ascending dose study of PMN310 in early AD

STUDY DESIGN · STAGGERED COHORTS · 12 MONTHS EACH



KEY ASSESSMENTS

Powered for 6-month biomarker change; 95% confidence to detect ≥1 ARIA case

Fluid biomarkers
CSF & plasma · baseline, 3, 6, 9, 12
tau pathology, synaptic damage, neuroinflammation

MRI for ARIA
Baseline, 2, 4, 6, 9, 12 mo

Amyloid PET
Baseline, 12 mo

Clinical outcomes
Baseline, 6, 12 mo
iADRS, CDR-SB, ADAS-Cog and other cognition, function and composite scales

*56 planned. 128 planned overall to achieve 100 completers; 144 enrolled across 22 sites. Interim data blinded.

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TRIAL DEMOGRAPHICS

PRECISE-AD Enrolled a Representative AD Population, Including the Highest ARIA-Risk Patients

KEY DEMOGRAPHICS · Early AD (MCI or mild dementia due to AD) · 144 enrolled across 22 sites

144	**136**	**73.3**	**58%**	**69%**	White	**24%**	Hispanic
enrolled	safety-evaluable	mean age (yrs)	female	**6%**	Black	**1%**	Asian

APOE4 STATUS

61% APOE4 carriers



39% non-carrier | 50% heterozygous | 11% ε4/ε4

ε4/ε4 homozygotes are the highest ARIA-risk group and are largely excluded from current anti-amyloid therapies

WHY APOE4 MATTERS

- ✓ **Representative population, including highest ARIA-risk patients**
- ✓ **Treatment options may open for ~15% of AD patients** who are APOE4 homozygotes, the group current therapies largely exclude.

Source: AD population genotype percentage and allele frequencies; Yamazaki et al. Nat Rev Neurol 2019

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INTERIM SAFETY · 6-MONTH BLINDED

Favorable Safety Data, Observed Across Key Measures

6-month blinded interim · N = 144 dosed, 136 safety-evaluable · a representative population

0.0% ARIA-E

The most severe treatment-driven form of ARIA

4.4% ARIA-H

In line with placebo-range ARIA-H, all mild, asymptomatic, non-serious

No treatment-related serious AEs

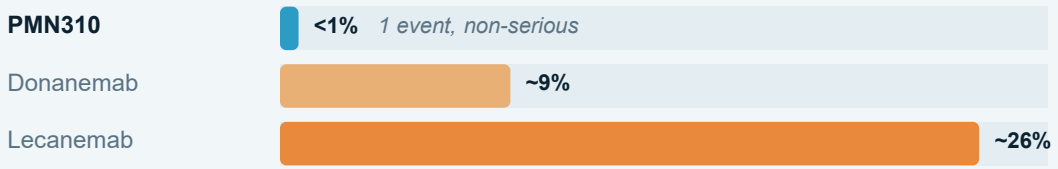
Across all genotypes

No treatment-related discontinuations

Low overall dropout rate

Minimal infusion reactions

A recognized liability of approved anti-amyloids and brain-shuttle candidates.
PRECISE-AD: one non-serious event, non-systemic, dosing continued.



APOE4 carriers included

61% carry ≥1 APOE4 allele

11% ε4/ε4 homozygotes — the highest ARIA-risk group

✓ **No ARIA-E in any genotype**

ARIA detection using **Susceptibility-Weighted Imaging (SWI)** for increased sensitivity.

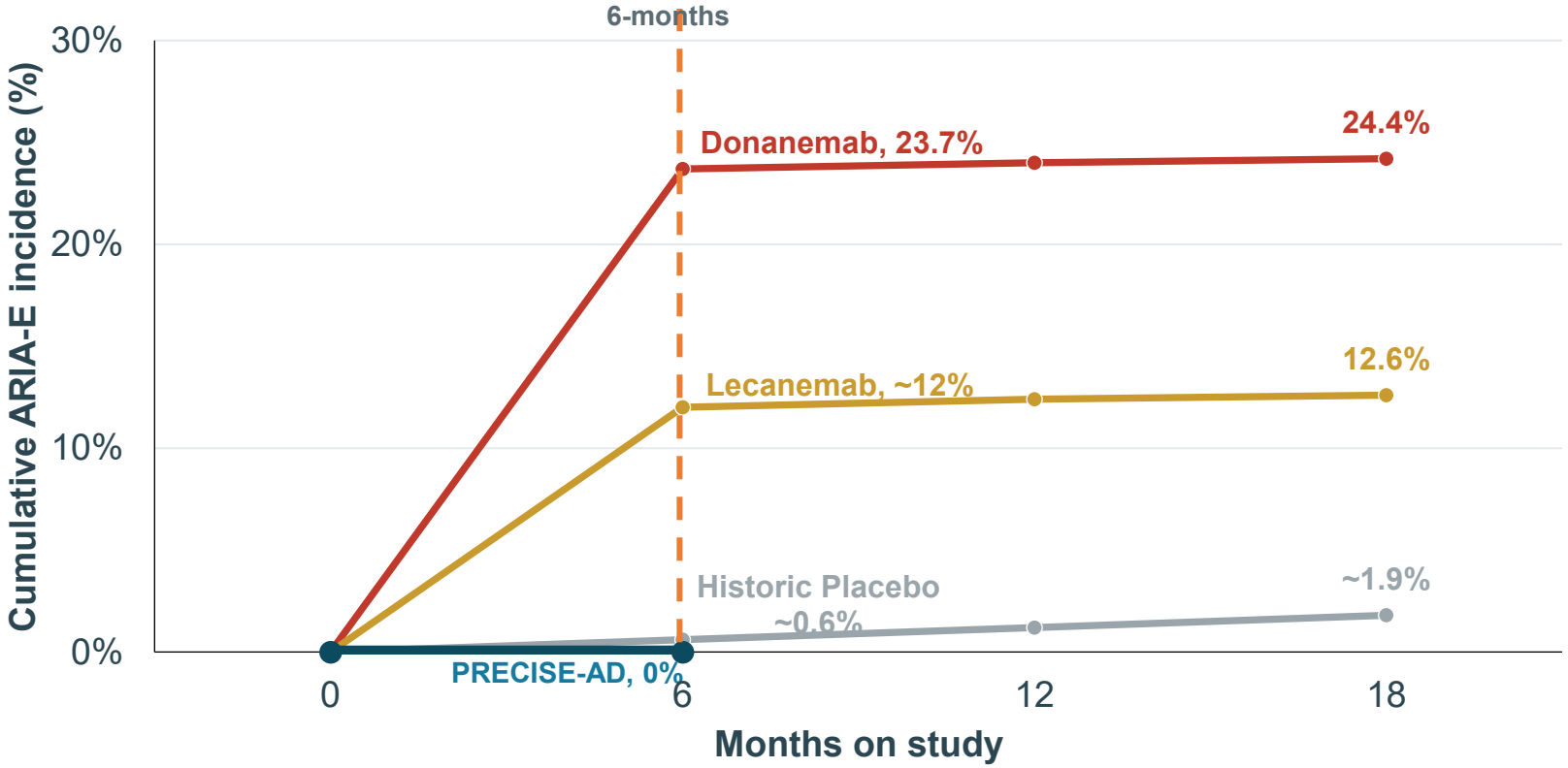
6-month blinded. Infusion-reaction comparators: lecanemab 26.4% (CLARITY AD, van Dyck NEJM 2023); donanemab 8.7% (TRAILBLAZER-ALZ 2, Sims JAMA 2023). Descriptive comparison only. No head-to-head studies have been conducted to compare PMN310 with approved products or other product candidates in development.

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Cumulative ARIA-E Over Time: Zero Edema Observed in First 6 Months

ARIA-E (the most serious ARIA type) has been observed to occur almost entirely in the first six months of dosing.

Comparator ARIA-E plateaus after the first few months; rates are time-matched to published data where available, with ~ indicating values estimated between published time points.



AT 6 MONTHS

Time-matched to estimated or published 6-month rates

Donanemab	23.7%
Lecanemab	~12%
Historic, third-party placebo data (avg across both trials)	~0.6%*
PRECISE-AD	**0%**

Illustrative cross-trial comparison — not head-to-head; ~ denotes values estimated between published time points.

* Placebo: 6-mo ARIA-E (~0.6%) is ProMIS-calculated by linear accrual — (6/18) × ~1.9%, the average of the two trials' 18-mo placebo rates (1.7% and 2.1%). Lecanemab: 12.6% treated / 1.7% placebo ARIA-E at 18 mo; 6-mo (~12%) approximated from this front-loaded cumulative (van Dyck et al., NEJM 2023;388:9–21). Donanemab, TRAILBLAZER-ALZ 2: 24.0% treated / 2.1% placebo over 76 wk (Sims et al., JAMA 2023;330:512–527). Donanemab standard regimen, TRAILBLAZER-ALZ 6: 23.7% ARIA-E at wk 24 (6 mo), 24.2% at 76 wk (Wang et al., Alzheimer's & Dementia 2025;21:e70062); pooled standard-dosing 18-mo ~24.4%. PRECISE-AD: blinded pooled cohort (active + placebo, 3:1); 0/136 ARIA-E at 6-mo interim — observed, not projected.

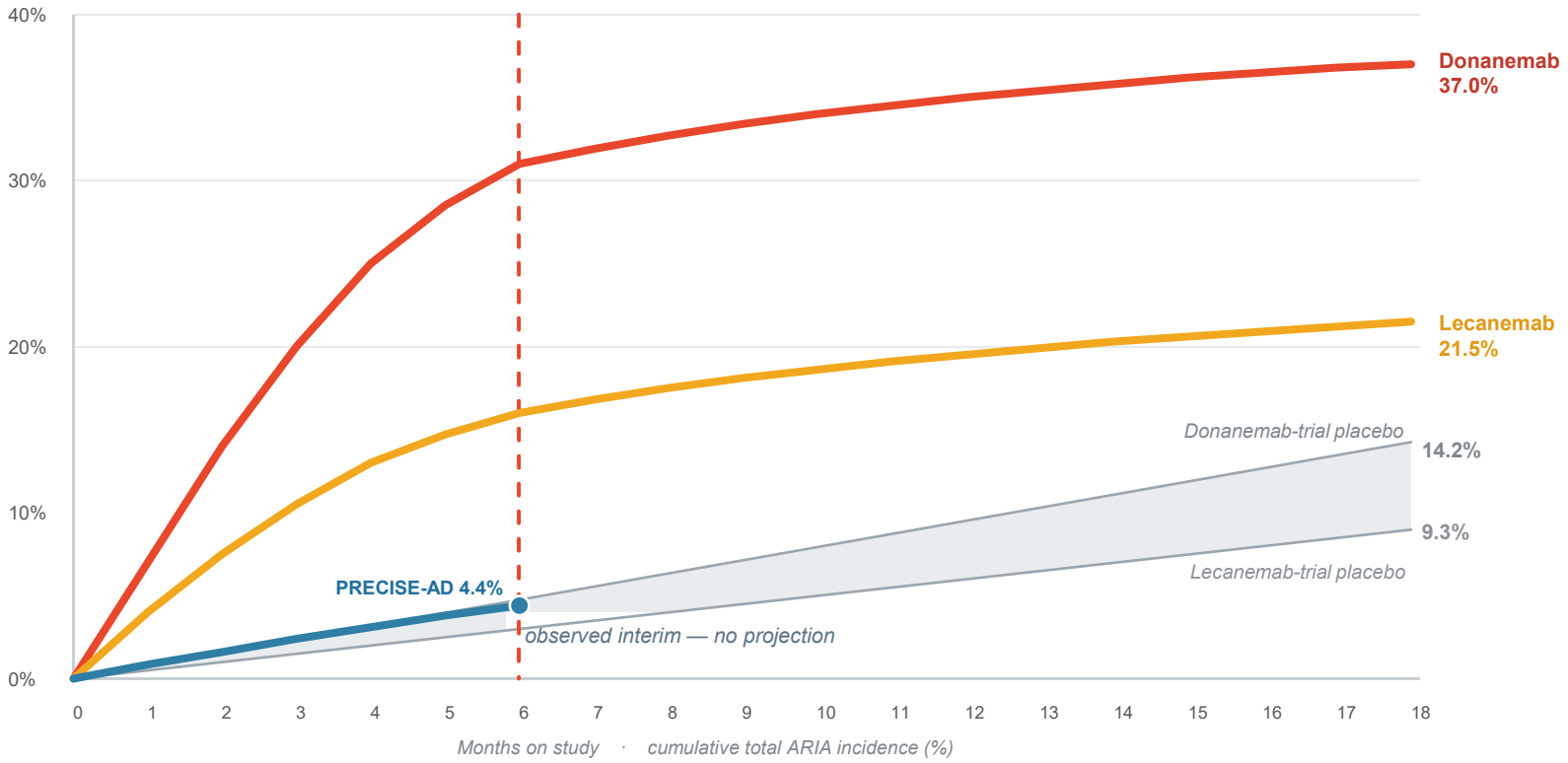
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TIME-MATCHED SAFETY

Cumulative Total ARIA Over Time: Blinded PRECISE-AD Data Tracks the Placebo Range Reported in Other Trials

Approved anti-amyloids accrue ARIA steeply in the first months of dosing, with 90%+ of ARIA-E occurring in the first 6 months.

Comparator rates are time-matched to published data where available, with ~ indicating values estimated between published time points.



AT 6 MONTHS

**Calculated based on published data*

Donanemab	*~31%
Lecanemab	*~16%
Historic, third-party placebo data (both trials)	*~3–5%
PRECISE-AD	**4.4%**

MODELED SHAPE, ACTUAL ENDPOINTS — comparator and placebo curves are reconstructed from published trial time-course; trial-end values are as reported. Illustrative, not a head-to-head comparison.
Any ARIA (ARIA-E and/or ARIA-H): Lecanemab 21.5% / 9.3% placebo at 18 mo (van Dyck, NEJM 2023); Donanemab 37.0% / 14.2% placebo, TRAILBLAZER-ALZ 2 ~76 wk (Sims, JAMA 2023; Zimmer, JAMA Neurol 2025). 6-mo points calculated from the published time-course (>90% of ARIA-E within 6 mo): Donanemab ~31%, Lecanemab ~16%, placebo ~3–5%; placebo accrues linearly to reported endpoints. PRECISE-AD: blinded pooled cohort (active + placebo, 3:1); 4.4% total ARIA (6/136) at 6-mo interim — observed, not projected.

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PRECISE-AD Interim Biomarker Results

Two tau biomarkers reported, both expected to rise without treatment

Plasma pTau217 BLOOD-BASED

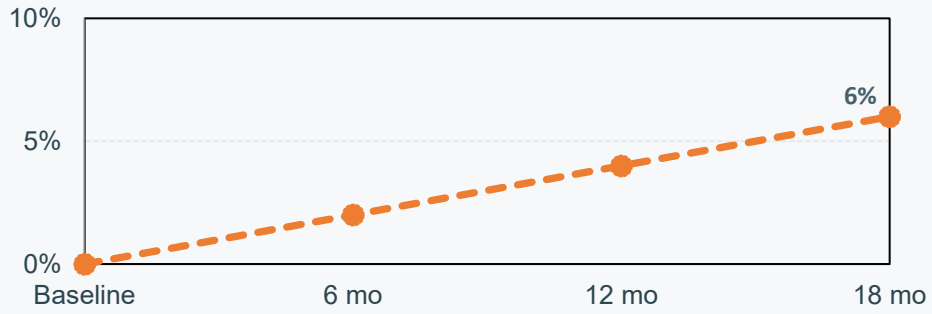
WHAT IT MEASURES

Disease progression: a downstream marker of amyloid-positive AD pathology and tau pathway activation.

IN UNTREATED PATIENTS

In untreated (placebo) patients, pTau217 rises ~6% over 18 months[1].

NATURAL HISTORY PROGRESSION Donanemab Ph2 placebo arm[1]



CSF MTBR-tau243 CEREBROSPINAL FLUID

WHAT IT MEASURES

A CSF marker of tauopathy, the pathology most closely tied to symptoms.

Correlates with tau-tangle burden and cognition comparable to tau-PET, and more strongly than other CSF markers.

IN UNTREATED PATIENTS

MTBR-tau243 **rises as tangles accumulate** and moves in step with disease progression over time.

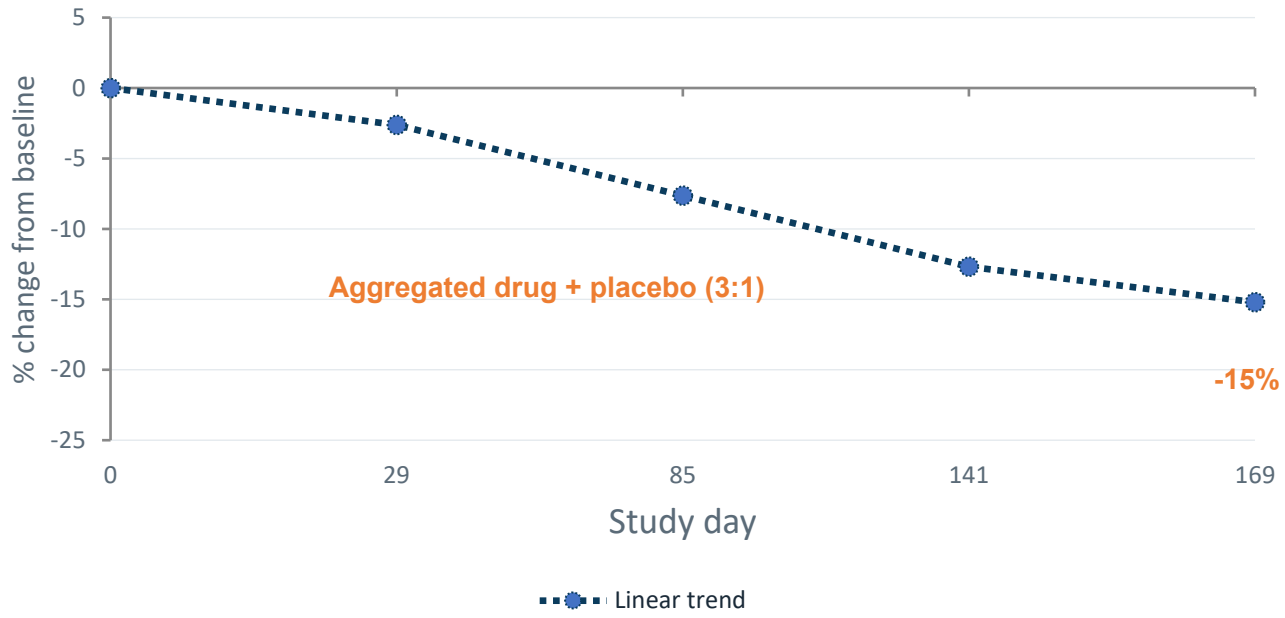
In untreated patients, MTBR-tau243 is therefore expected to trend upward.

PRECISE-AD interim results on the following slides are pooled and blinded. 1. Pontecorvo et al., JAMA Neurology 2022 (TRAILBLAZER-ALZ Ph2 donanemab trial, placebo-arm natural history). Horie et al., Nature Medicine 2023.

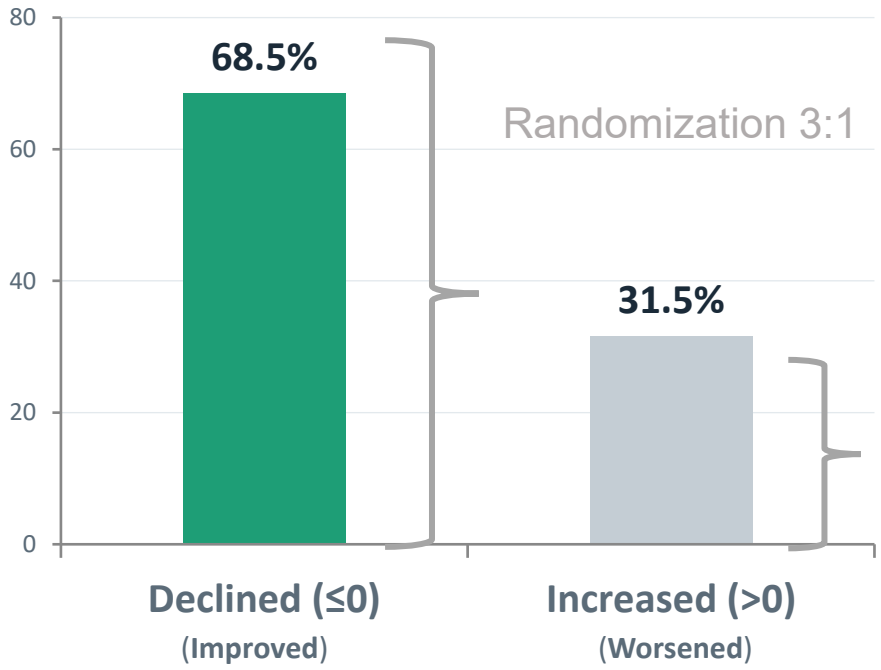
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PRECISE-AD INTERIM RESULTS: Plasma pTau217 declined steadily through Day 169

PRECISE-AD Mean % change from baseline over time



PRECISE-AD patients with a Day-169 decline



Why this is compelling at 6 months

Untreated pTau217 rises as the disease progresses. The 15% **decline shown in the PRECISE-AD** data (including both PMN and placebo treated patients) indicates a possible early treatment-associated reversal. Although responder data remains blinded, the ~68% responder fraction closely aligns with the 75% active allocation in the 3:1 design based on a prior ProMIS analysis. A 6-month pTau217 change could be an early leading indicator of benefit and a predictor of 12-month clinical outcome (Pentara/ProMIS).

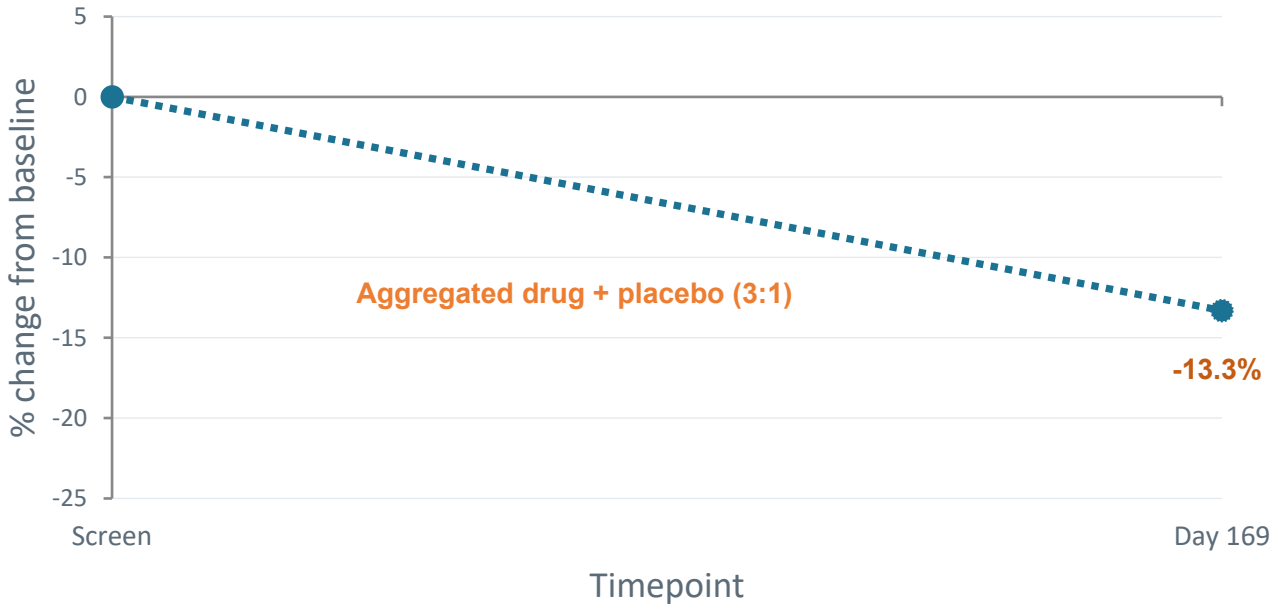
Ref: “Leveraging recent advances in plasma biomarkers to optimize early proof of concept trials in Alzheimer’s disease.” Alzheimer’s & Dementia: Translational Research & Clinical Interventions (TRCI), 2025. DOI 10.1002/trc2.70183.

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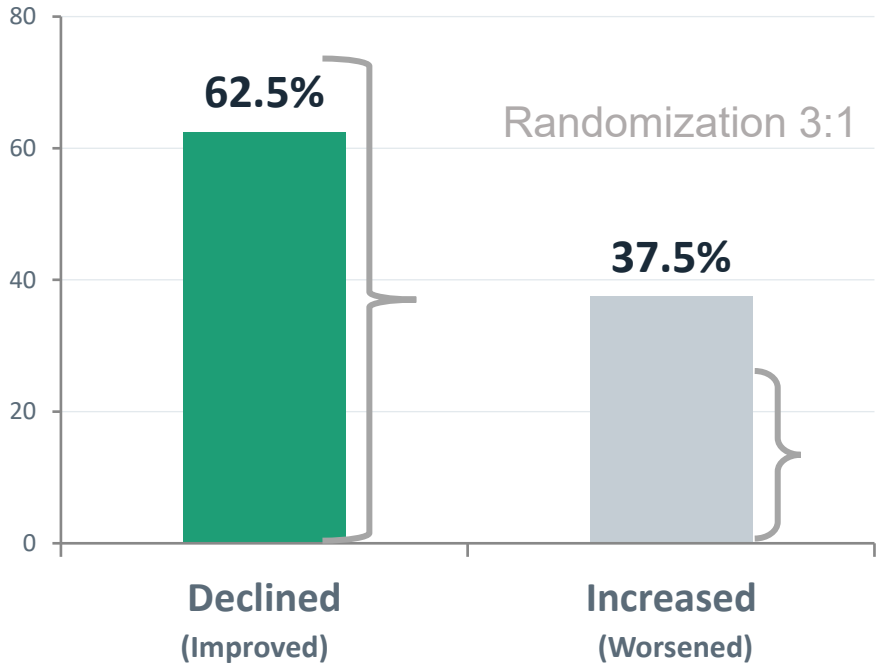
6-MONTH BLINDED ANALYSIS · CSF

PRECISE-AD INTERIM RESULTS: CSF MTBR-tau243 declined through Day 169

PRECISE-AD Mean % change, screen → Day 169



PRECISE-AD: Patients with a Day-169 decline



Why this matters at 6 months

MTBR-tau243 is **correlated with tau tangle burden** and normally **rises** as aggregates accumulate, so a downward move is a favorable, disease-relevant signal. Across the trial participants, it declined on average (13.3%) and in the majority of patients. The effect is early, not yet powered for significance, but its direction, **against a rising natural history**, is the meaningful result and is directionally consistent with the upstream plasma pTau217 decline and tracks the 3:1 randomization pattern.

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CONCLUSION

A Differentiated, Precision Approach to Alzheimer's — With a Clear Path Forward

PRECISE-AD's blinded interim analysis shows favorable safety data and early, mechanism-consistent biomarker signals, supporting a differentiated approach for patients underserved by approved anti-amyloid therapies.

01

DIFFERENTIATED MECHANISM

Oligomer-selective by design. PMN310 is engineered to bind toxic amyloid-β oligomers while avoiding plaque, decoupling efficacy from the ARIA liability that defines the approved class.

02

FAVORABLE SAFETY PROFILE

Zero ARIA-E; 4.4% total ARIA — all mild, asymptomatic ARIA-H. Zero treatment-related SAEs and zero drug-related discontinuations, minimal (n=1) drug-related infusion reactions as of data cutoff date.

03

TARGET ENGAGEMENT WITH RELEVANT BIOMARKERS

Coherent, biologically aligned signal. Upstream plasma pTau217 declined significantly (~69% of patients, tracking 3:1 randomization) and downstream CSF MTBR-tau243 moved favorably (~63% of patients declined), both opposite a rising natural history.

04

OPENING ACCESS TO APOE4 CARRIERS

No ARIA-E in any genotype. With 61% APOE4 carriers and 11% homozygotes enrolled, a favorable safety profile could remove the class's single biggest barrier, especially for the patients at highest risk.

05

POTENTIAL IN PRECLINICAL AD

A safety profile suited to earlier intervention. A placebo-level ARIA profile makes PMN310 a strong candidate to move upstream into preclinical AD, where prevention has the greatest impact, but tolerability is paramount.

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Topline in Q1 2027 and the Path Forward

Q1 2027 topline data aims to show a material shift in benefit-risk

TARGET SCENARIO

▼ LOWER RISK

Materially improved safety

- **ARIA similar to placebo (background) rate**
- **Minimal infusion reactions**

= COMPARABLE BENEFIT

Similar efficacy

- **~15–20% slowing of progression vs placebo**
  - *Similar efficacy to marketed drugs (derived from 18-month data) using iADRS or CDR-SB*
- **Coherent directional trends in biomarkers, supportive of oligomer-selective MOA**

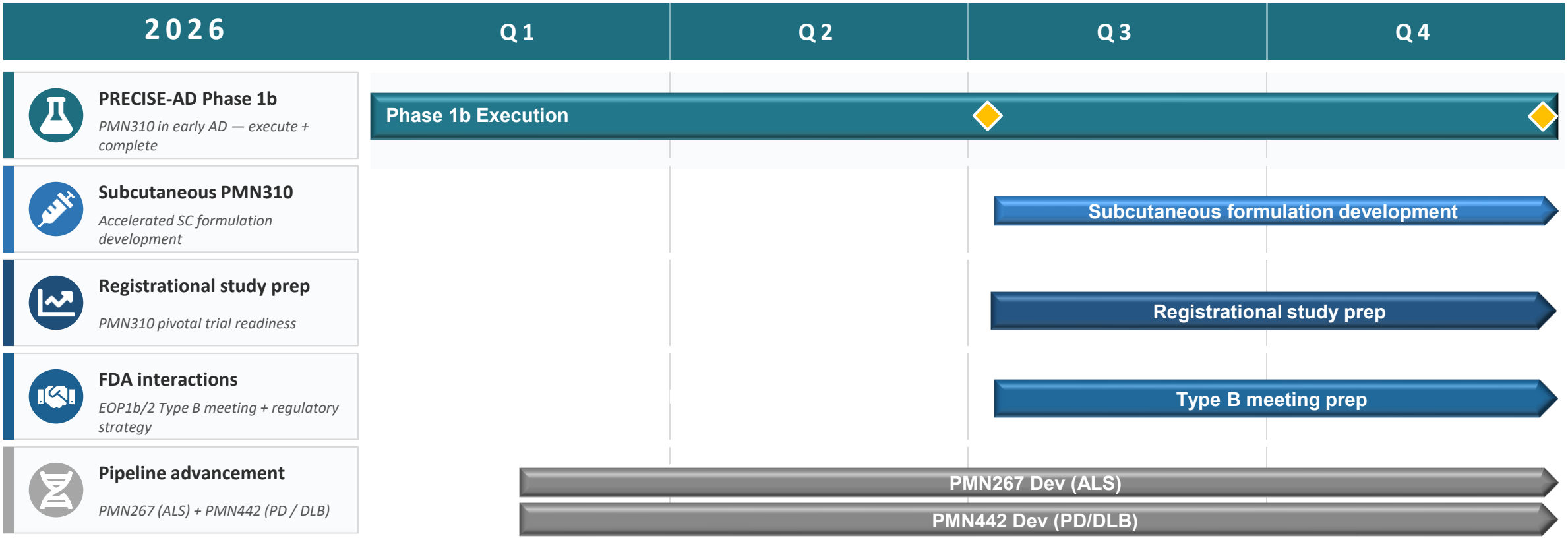
If successful, PMN310 could potentially offer a favorable and differentiated benefit-risk profile

Target scenario reflect management's view on possible safety and directional efficacy signals and is not based on any review of clinical data beyond the interim data in July 2026. PRECISE-AD is a 12-month Phase 1b study designed to assess safety and directional efficacy signals; it is not powered for statistical significance on clinical endpoints.

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2026 Strategic Priorities

Parallel workstreams converging on PMN310 Phase 3 readiness and broader pipeline advancement



Execution of 2H'26 activities is subject to interim data outcomes, regulatory feedback, and resource allocation

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Early PRECISE-AD signals support PMN310's differentiated profile

SUMMARY

01

Novel precision targeting

EpiSelect™ discovery platform, with a pipeline of antibodies and vaccines against toxic misfolded proteins

02

Clean safety profile at 6 months

No ARIA-E, 4.4% ARIA-H, no treatment-related SAEs

Minimal infusion reactions

03

Target engagement demonstrated

Toxic oligomers reduced in Phase 1a

pTau217 and MTBR-tau243 declining in Phase 1b

04

Topline results ahead

12-month PRECISE-AD data in Q1 2027

Cash runway through 2027

05

FDA Fast Track Designation

Granted for PMN310

Allows more frequent FDA interaction

A potentially favorable and differentiated benefit-risk profile

12-month PRECISE-AD topline in Q1 2027, with cash runway through 2027

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NASDAQ: PMN