ProMIS™ Neurosciences

Targeting underlying causes of neurodegenerative diseases

Forward Looking Statements Disclaimer

This slide deck may contain certain forward-looking information. Such information involves known and unknown risks, uncertainties and other factors that may cause actual results, performance or achievements to be materially different from those implied by statements herein, and therefore these statements should not be read as guarantees of future performance or results. Such forward-looking statements include, among others, statements pertaining to anticipated milestone timing, as unforeseen delays in conducting and monitoring the Company's pre-clinical and clinical trials may occur from time to time, which may result from delays in trial enrollment or our ability to raise any additional required capital with respect thereto, among other factors. All forward-looking statements are based on ProMIS Neurosciences Inc.’s (the “Company”) current beliefs as well as assumptions made by and information currently available to it, as well as other factors. Readers are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date of this slide deck. Due to risks and uncertainties, including the risks and uncertainties identified by the Company in its public securities filings available online at sec.gov, actual events may differ materially from current expectations. The Company disclaims any intention or obligation to update or revise any forward-looking statements, whether as a result of new information, future events, or otherwise.

ProMIS™ Neurosciences

Public (PMN:Nasdaq), based in Cambridge, MA

Clinical-stage biopharma with pipeline selectively targeting specific, disease-causing misfolded proteins

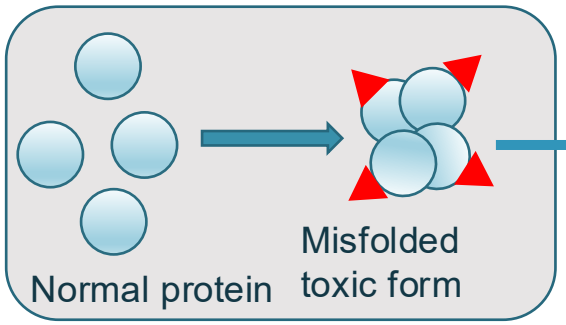
Unique potential in areas of great unmet need

- **Unique selectivity** may create potential to address the unmet need for safer, more efficacious therapies
- **Data expected in 2026:** Interim 6-month blinded analysis expected early Q3'26; top-line data expected early Q1'27 in robust Ph1b Alzheimer's trial
- **Significant market potential** for PMN310 in early Alzheimer's patients expected peak sales >$10Bn
- **Seasoned leadership team** with global development and deep domain experience
- **Financials:** Recent funding of up to $175M provides cash through 2027, A-list syndicate: Janus, Ally Bridge, Wellington, Great Point Partners, Deep Track

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

EpiSelect™ Proprietary Computational Modeling Approach

Computational modeling

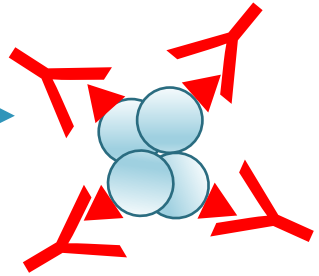


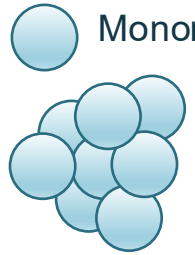
Identification of regions - **conformational epitopes** - likely to be exposed in *only* misfolded toxic forms

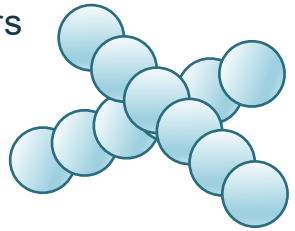
Immunization with conformational epitope restricted to toxic form

Generation and screening of monoclonal antibodies

Antibodies highly selective for toxic form





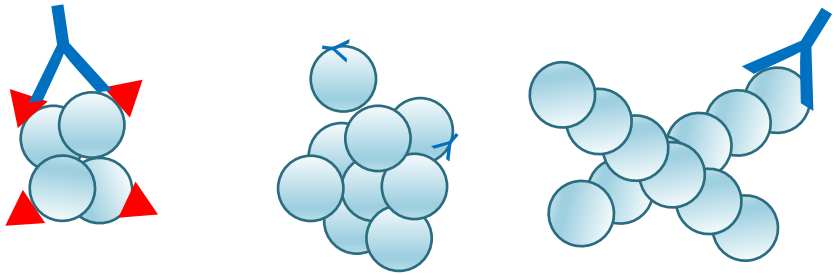


Conventional Approach Used by Others

Linear epitope

Immunization with synthetic aggregates or linear peptides not unique to misfolded toxic form

Non-selective antibodies



Robust Pipeline Targeting Toxic Misfolded Proteins Across a Range of Neurodegenerative Diseases

	Product Candidate	Target Protein	Disease Indication(s)
ANTIBODY	PMN310*	Amyloid-Beta	Alzheimer's Disease
	PMN267	TDP-43	ALS, FTD
	PMN442	Alpha-Synuclein	PD, MSA, DLB
VACCINE	PMN311	Amyloid-Beta Vaccine	Alzheimer's Prevention
	PMN260	TDP-43 Vaccine	TDP-43 Proteinopathies
	PMN440	Alpha-Synuclein Vaccine	Alpha Synucleinopathies
DISCOVERY		TDP-43 (Intrabody)	ALS, FTD
		RACK1	ALS, FTD, HD
		DISC1	Mental disorders, e.g., Schizophrenia, Bipolar disorder

* U.S. FDA Fast Track designation granted | ALS: Amyotrophic lateral sclerosis, FTD: Frontotemporal dementia, PD: Parkinson's disease, MSA: Multiple system atrophy, DLB: Dementia with Lewy Bodies; HD: Huntington's Disease

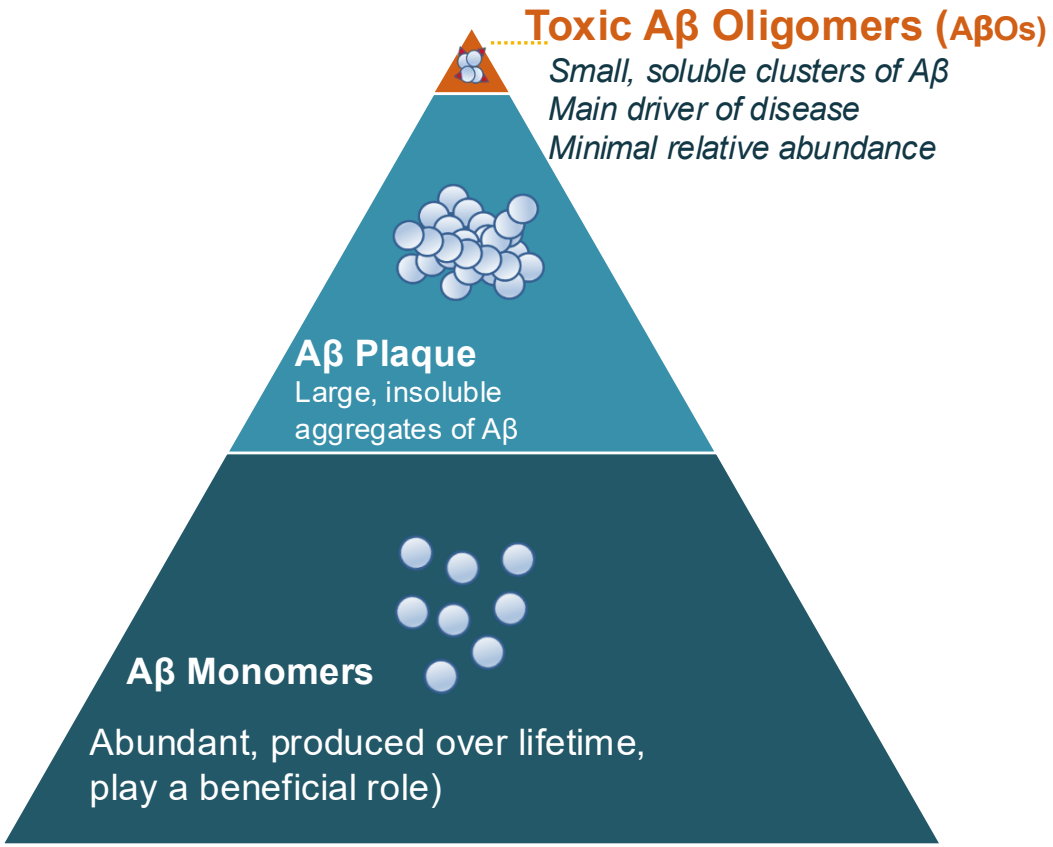
Lead Clinical Candidate PMN310 in Alzheimer's Disease

Selectivity for
Toxic Aβ Oligomers

ProMIS™ Neurosciences

Toxic Aβ oligomers are minor in relative in-vivo abundance and constitute prime targets for Alzheimer's Disease

ILLUSTRATIVE RELATIVE ABUNDANCE OF Aβ SPECIES[1]



- As **AβOs** are the primary drivers of disease, target concentration is critical to slowing of disease progression
- Given the relative minimal concentration of toxic **AβOs**, lower quantities of antibodies can achieve target saturation
- Antibody binding to **Aβ Plaque** also leads to off-target sink effect and importantly can result in ARIA (swelling and bleeding of the brain)
- Binding to **Aβ Monomers** does not confer efficacy and can lead to a sink effect, resulting in a sub-therapeutic dose

Because of low target antibody quantity requirements and PMN310's ability to avoid a sink effect, **PMN310 achieved 100-600x target saturation in Ph1a**

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

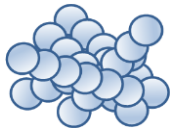
Importance of selectivity for toxic amyloid-β oligomers

Monomers
Abundant
Play physiologic role

No efficacy or ARIA impact from monomer binding
Acts as a sink for mAbs

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

Primary efficacy target
Minimal ARIA liability



Plaque
Large, insoluble aggregates of Aβ

Limited efficacy
ARIA associated with plaque binding

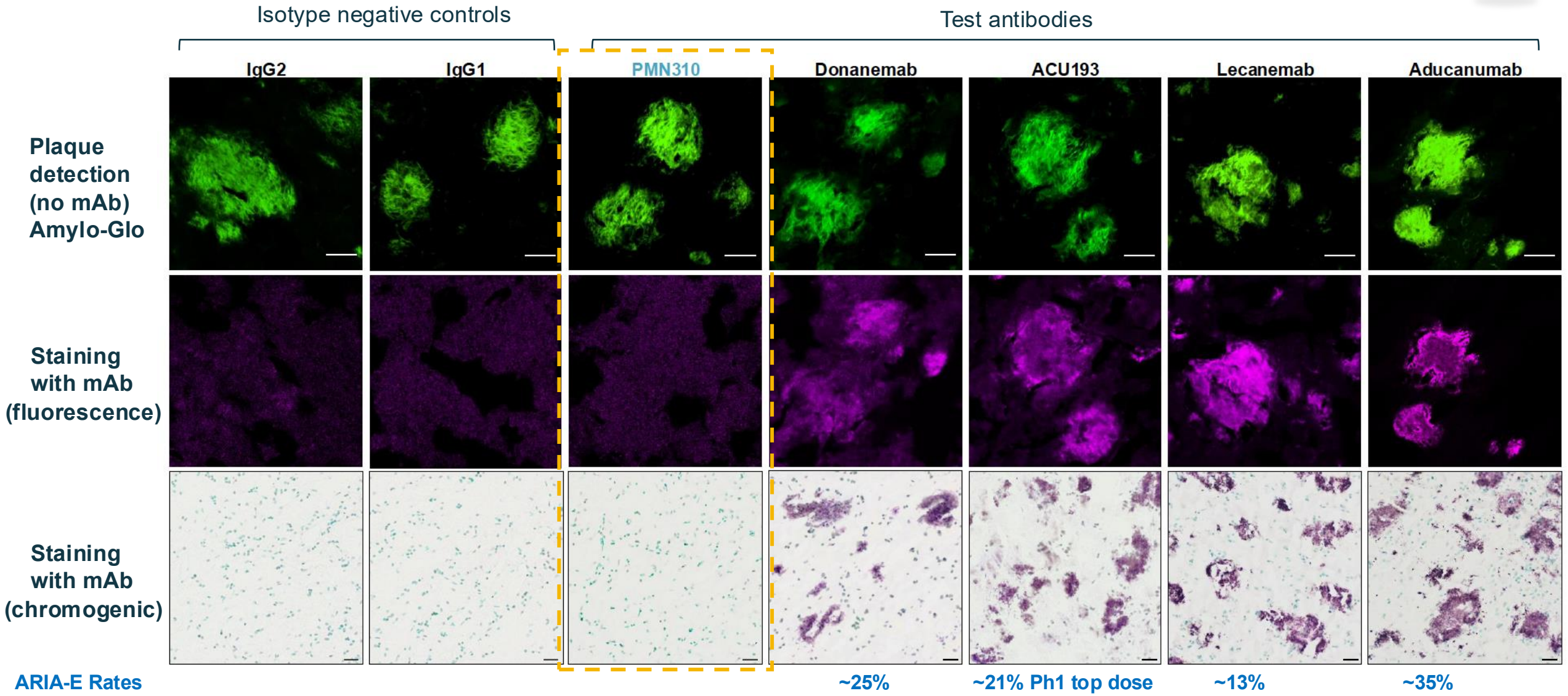
	Efficacy *% Slowing of Cognitive Decline[1]*	**Safety** *ARIA-E Rates[2]*
Solanezumab	None	0%
Gantenerumab	None	25%
Aducanumab	22%	35%
Donanemab	22%	25%
Lecanemab	27%	13%
PMN310	TBD	TBD

Colors represent successive generations of Abeta products.

Note: Illustrative graphic. Data are derived from different clinical trials with different trial designs and patient populations. No cross-trial comparisons can be made. No head-to-head clinical studies have been conducted.

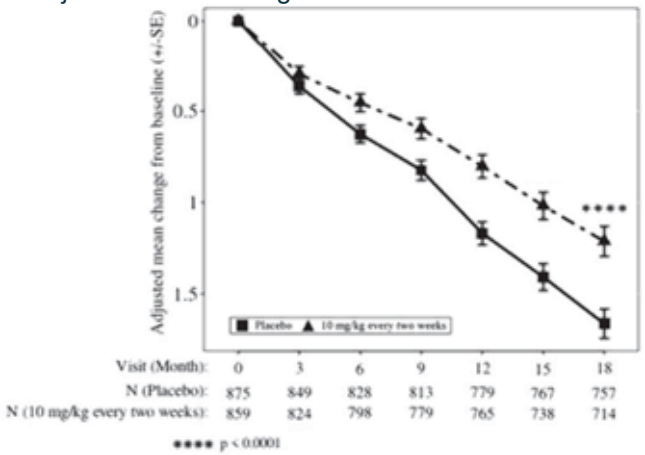
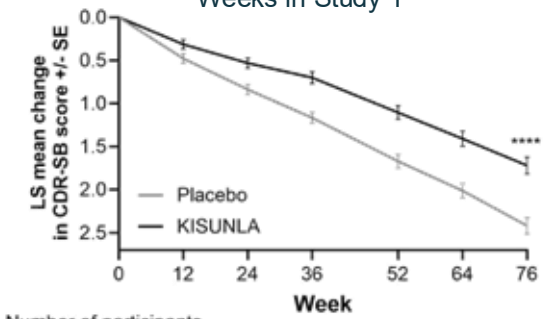
1, 2. Rates observed in Ph3 trials; where multiple trials were conducted, rate represents the average. Sol: Sperling, et al. Trial of Solanezumab in Preclinical Alzheimer's Disease. NEJM, Jul 2023. Gant: https://www.roche.com/media/releases/med-cor-2022-11-14; Adu: Budd Haeberlein S et al, 2022, J Prev Alz Dis; Don: Mintun MA et al, 2021, NEJM; Lec: Swanson CJ et al, 2021, Alzheimer's Research and Therapy 2. Average ARIA-E rates observed in respective Ph3 trials

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

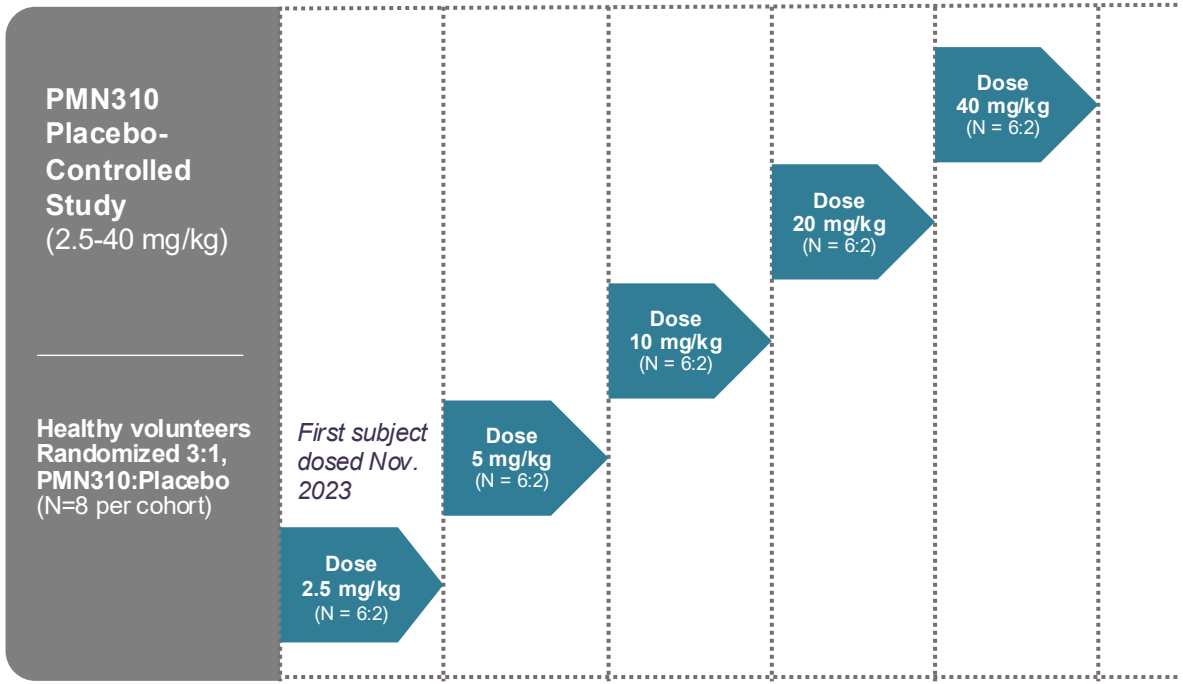
Multi-Billion Dollar Sales Expectations for SoC Therapies Despite Limited Clinical Efficacy and Challenging Safety (ARIA)

	Modest Clinical Benefits	Challenging Label	Annual Sales Ests.(1)
Leqembi *(lecanemab)*	Adjusted Mean Change from Baseline in CDR-SB in Study 2 	**Black Box Warning:** Amyloid related imaging abnormalities (ARIA) ARIA-E Incidence(2): 13% ARIA-H Incidence(2): 17%	**~$0.5B → ~$3.2B** 2025E 2032E *29.1% CAGR*
Kisunla *(donanemab)*	CDR-SB Change From Baseline in Combined Population Through 76 Weeks in Study 1 	**Black Box Warning:** Amyloid related imaging abnormalities (ARIA) ARIA-E Incidence(3): 25% ARIA-H Incidence(3): 28%	**~$0.2B → ~$2.6B** 2025E 2032E *43.6% CAGR*

1. Evaluate Pharma 2. Warnings and precautions in Leqembi Prescribing Information 3. TRAILBLAZER-ALZ 6 (Lilly) ARIA results

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

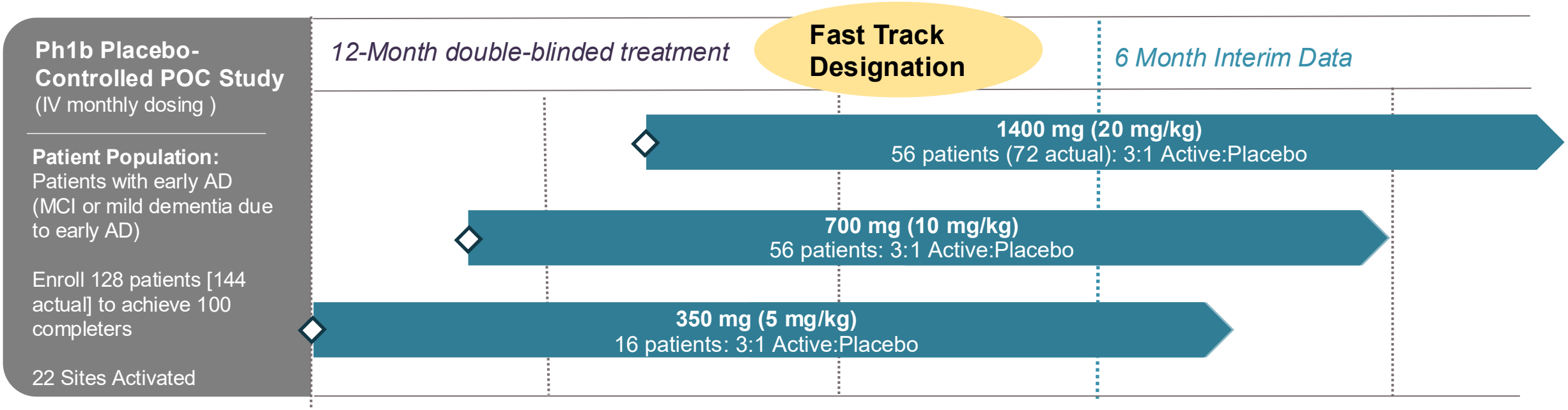
Key Findings

- 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

PRECISE-AD: PMN310 Phase 1b MAD Trial in Early AD Patients

12-month Double-blind Treatment, Interim 6-month Data, Target N=100 Completers



Efficacy: Assess pharmacodynamic markers of treatment effect at baseline, 3, 6, 9, 12 months; Sufficient power to detect biomarker changes at 6 months

Safety: Powered to provide 95% confidence to detect at least one ARIA case

CSF/ Plasma Biomarkers: baseline, 3, 6, 9, 12 months

- p-tau217, p-tau243
- GFAP
- Neurogranin
- SNAP-25
- Aβ42/40
- NfL

MRI for ARIA at baseline and months 2, 4, 6, 9, 12

Aβ PET

Clinical endpoints (cognition) measured at baseline, 6m, 12m and included in final analysis:

- CDR-SB (Clinical Dementia Rating, Sum of Boxes)
- ADAS-cog
- ADAS-ADL
- IADRS
- Clinical Global Impression of Change

Upcoming Planned Interim Analysis Readout

Blinded 6-month Qualitative Analysis | Safety and Biomarker signal | Early Q3 '26

Safety Profile to Date

- Snapshot of ARIA incidence rate
- Adverse event monitoring
- Overall tolerability, to date

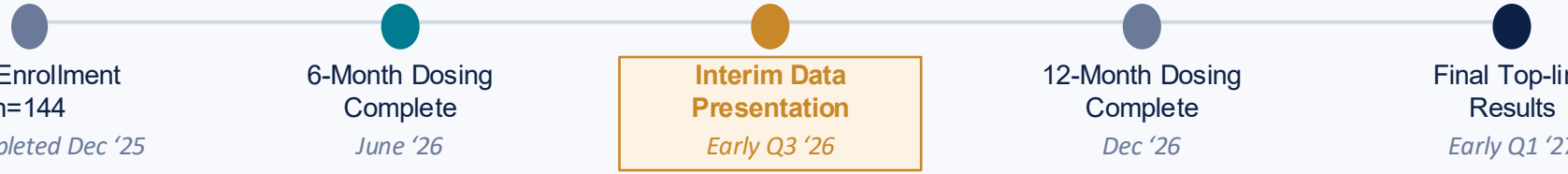
Blinded Biomarker Data

Selected blinded data across key disease-relevant biomarkers. This may include:

- **pTau 217** *Disease progression*
- **GFAP** *Neuroinflammation*
- **Neurogranin** *Post-synaptic function*
- **Aβ42/40 ratio** *Disease state*

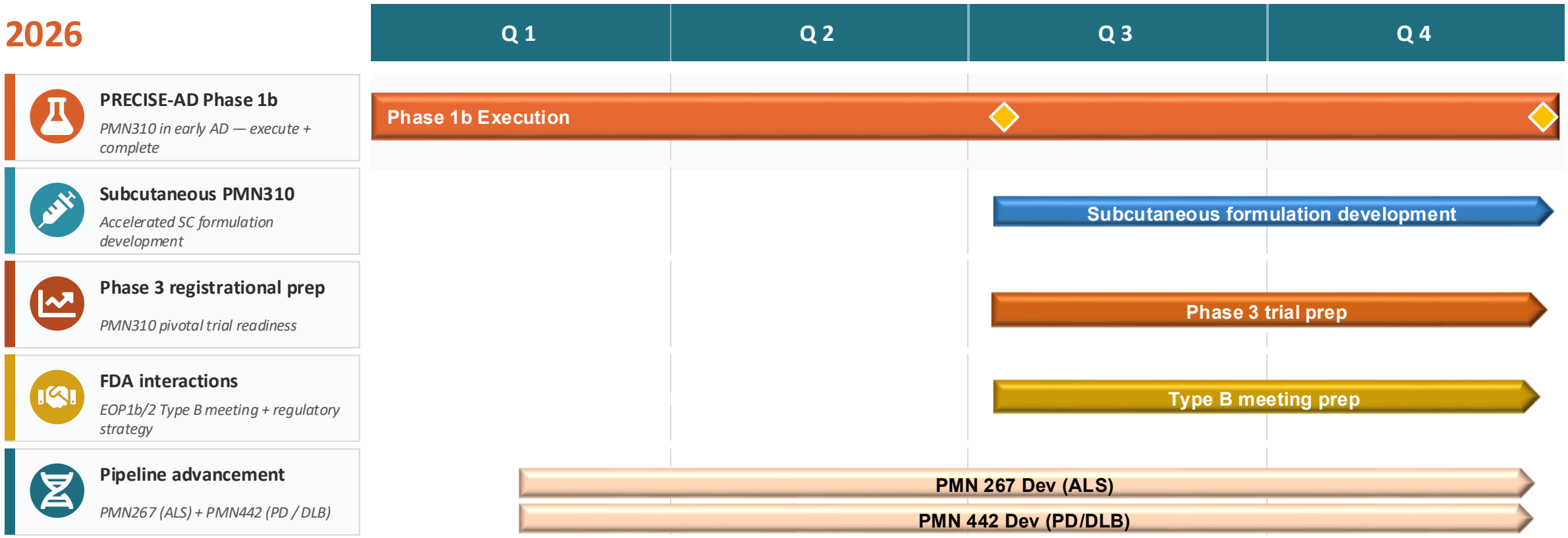
Important: This blinded interim analysis represents the 6-month midpoint of the 12-month trial.
It will **not include clinical endpoint data.** These remain blinded and are reserved exclusively for the final topline results readout

EXPECTED TRIAL TIMELINE



A catalyst-rich year: 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

ProMIS Neurosciences: Investment Summary

A New Standard for Disease-Modifying Alzheimer's Therapy

Unique Differentiation

- First & only therapy that appears to selectively target toxic oligomers – the drivers of neurodegeneration
- Potential to significantly reduce ARIA and improve efficacy
- Potential for Sub-cutaneous development

Near Term Catalysts

- Blinded analysis expected early Q3 '26
- Top-line results expected early Q1 '27
- Provides early signal on safety and potential efficacy through target engagement

Strong Clinical Profile

- Robust ongoing Phase 1b clinical trial
- Fully enrolled (n=144)
- FDA Fast Track granted
- Encouraging safety profile to date
- Success may enable direct Phase 3 registration

Financial Strength

- Recently closed $175M PIPE
- Backed by world-class A-list investors
- Well capitalized through key milestones

ProMIS™ Neurosciences

For further information, contact:
info@promisneurosciences.com

NASDAQ: PMN