



Oral Xanamem[®]

Controlling brain cortisol to treat depression and slow progression in Alzheimer's disease - a novel therapeutic mechanism

Corporate Presentation

October 2024

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Overview





Novel 11 β -HSD1 cortisol control mechanism, oral, attractive safety profile

- Brain cortisol has long been proposed as a pathogenic mechanism in Major Depressive Disorder (MDD) and Alzheimer's (AD)
- Unique brain-penetrant **tissue cortisol** synthesis inhibitor that leaves adrenal cortisol synthesis unaffected
- Approximately 400 people treated to date with excellent safety profile and low drug interaction risk



Positive phase 2 clinical data de-risk clinical program

- Phase 2a MDD trial data showing clinically significant activity with benefits across multiple end points
- Disease-modifying activity on CDR-SB in AD phase 2a trial in biomarker-positive AD patients 0.6 points over 12 weeks
- Positive data from both trials read through to other indications in psychiatry and the dementias



Patent protection and manufacturing

- Composition of matter protection to 2031 – to 2036 with extensions in major markets
- Additional recent patents protect further against future competition e.g. use patent for MDD
- Manufacturing process scaled up and patented, contractors Asymchem (China) & Catalent (US)



Large clinical and commercial opportunities

- No other brain-penetrant cortisol control molecules are in development
- Anti-depressant market is currently ~\$20 billion, with major opportunities for novel mechanisms & better-tolerated drugs
- Alzheimer's market likely to be \$20 billion by 2030, with major opportunity for a safe & effective oral agent

Experienced board and management team



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PhD



Michael Roberts
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B.Ec (Hons), CPA, FFIN



Company



ASX-listed company founded in 2014

- Market Cap A\$80-100 million
- Cash runway to late 2026
- Conducted three phase 1 (Australia) and four phase 2 trials (Australia, US and UK)



Key shareholders

- Biotech Value Fund (BVF) ~8%
- CEO Steve Gourlay ~5% (in part via \$2 million invested personally)
- Top 20 ex BVF & Gourlay ~23%



Phase 2b/3-stage clinical programs

- Alzheimer's disease (AD) phase 2b/3 on-going – interim mid 2025, final results mid 2026
- Major depressive disorder (MDD) phase 2a just completed



Fundraising history

- Initially Wellcome Trust support for University of Edinburgh
- 2014 merger of U Edinburgh spinout Corticrine with Actinogen ASX-listed shell
- Equity raises on ASX and Australian R&D tax incentive cash rebates (e.g. \$9 million this year)

State of play for Xanamem programs



Evidence of durable benefit on depression from control of brain cortisol validates the Xanamem program in terms of:

- ✓ “Cortisol control” mechanism of action
- ✓ 10 mg daily proof-of-concept dose being used in Alzheimer’s phase 2b/3 trial
- ✓ 10 mg daily dose is also suitable for next depression trial

Phase 2a MDD data support further clinical development in MDD

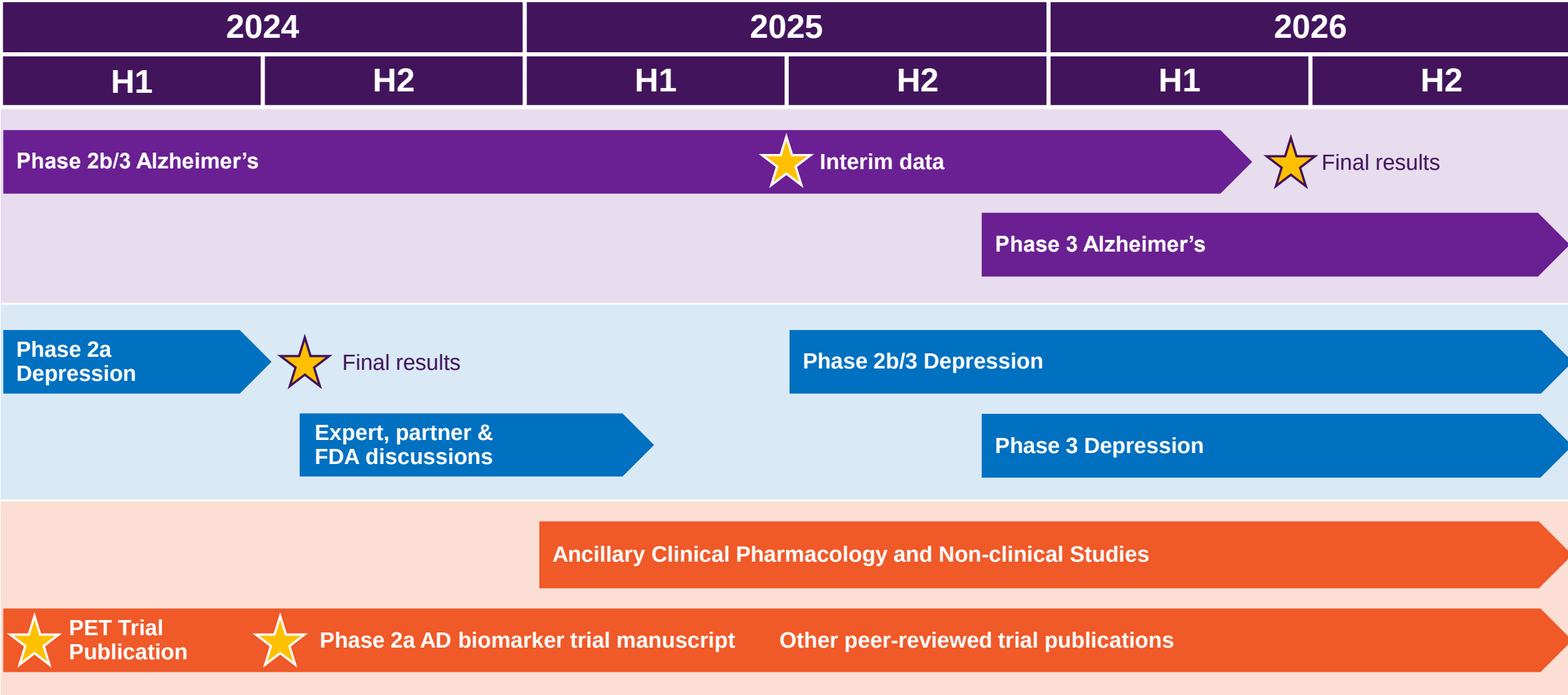
Clinical activity on depression supports the likelihood of seeing a disease-modifying effect in Alzheimer’s disease over 36 weeks

Xanamem - Pipeline



Indication	Preclinical	Phase 1	Phase 2	Phase 3	Next Milestone
Alzheimer's disease (AD)	On-going phase 2b/3			Open INDs	Results 25-26
MDD	Phase 2a complete, Phase 2b/3 in planning				FDA Type C in Q1 25
Fragile X syndrome	Phase 2a on hold				On hold
Bipolar disorder	Potential next indications				
PTSD					
Lewy-body dementia					
Frontotemporal dementia					

Xanamem – AD and MDD Program Timelines



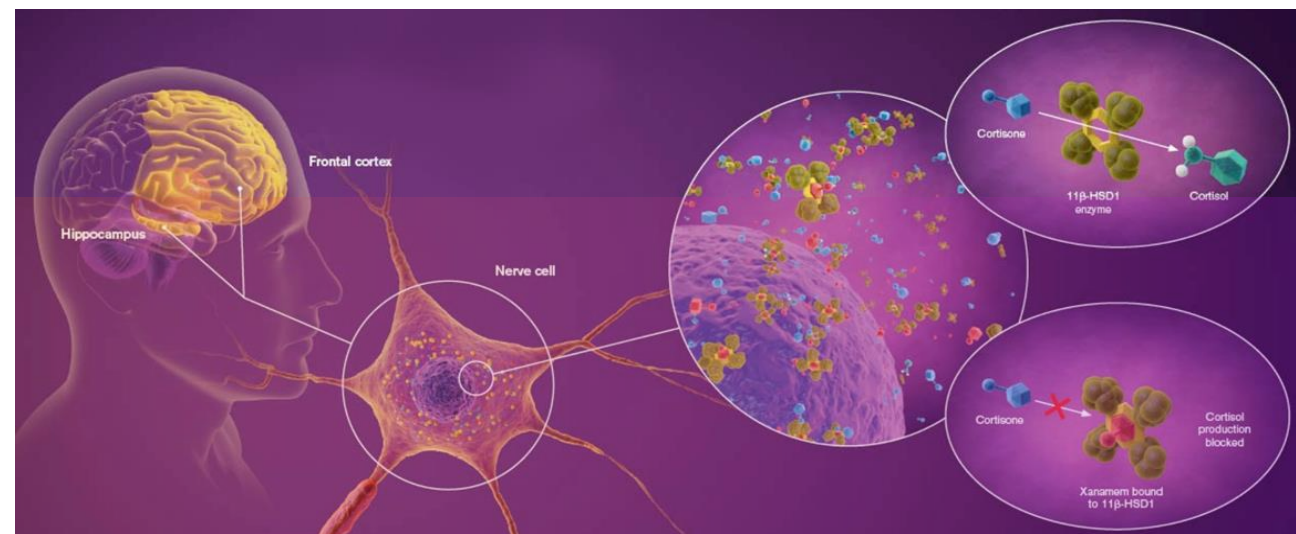
Xanamem



Once-daily oral treatment with a unique mechanism

Xanamem is a small molecule tissue cortisol synthesis inhibitor (11 β -HSD1 enzyme)

- ✓ Good safety profile in >380 treated
- ✓ Brain-penetrant at low doses
- ✓ Potentially disease-modifying in AD
- ✓ Anti-depressant activity in phase 2
- ✓ Low drug interaction potential ideal for combination therapy



Mouse experimental studies, brain cortisol levels & human clinical trials validate cortisol as a target for the treatment of AD

Xanamem controls cortisol by inhibition of 11 β -HSD1¹

Controlling brain cortisol² has potential durable benefits

“STRESS” in the brain becomes “CHILL”

RAPID changes in kinases, cell function, neurotransmitters over hours to days lead to short-term “low stress” settings



“Lower stress” shorter term e.g.

- Reducing inflammation
- Improving neurotransmitter balance
- Decreasing cell death

SLOW changes in gene expression and protein synthesis over days to weeks lead to durable “low stress” settings

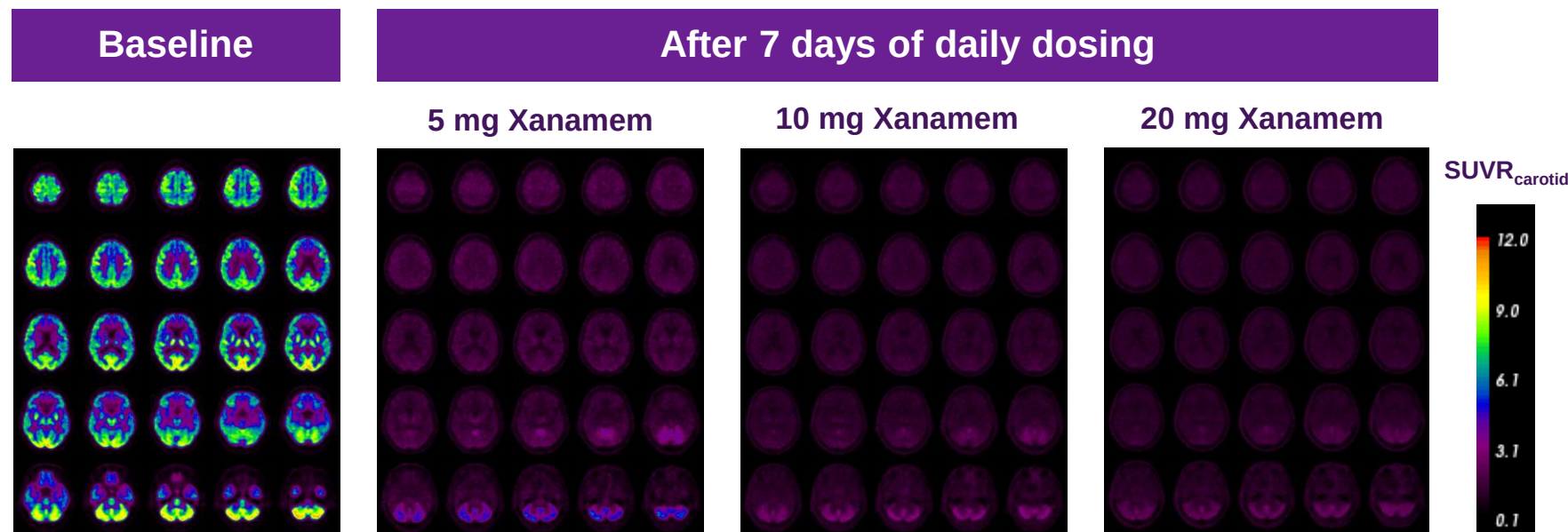


“Lower stress” longer term e.g.

- Improving neural circuitry
- Generating new brain cells
- Ideal receptor configurations

Human PET study shows full target engagement

Other 11 β -HSD1 enzyme inhibitors have not achieved adequate brain levels



Xanamem extensively binds to the 11 β -HSD1 enzyme throughout the brain, with high post-treatment effects (absence of colour) after 7 days at all doses, slightly less at a 5 mg dose.

This is consistent with full hormonal pharmacodynamic activity seen in clinical trials with doses as low as 5 mg.

Journal of Alzheimer's Disease 97 (2024) 1463–1475
 Brain 11-Hydroxysteroid Dehydrogenase Type 1 Occupancy by Xanamem™
 Assessed by PET in Alzheimer's Disease and Cognitively Normal Individuals
 Victor L. Villemagne, Vincent Dor, Lee Chong, Michael Kassiou, Rachel Mulligan,
 Azadeh Feizpour, Jack Taylor, Miriam Roesner, Tamara Miller and Christopher C. Rowe

Alzheimer's disease program

Alzheimer's disease

Strong cortisol control scientific rationale to address huge unmet medical need

Rationale

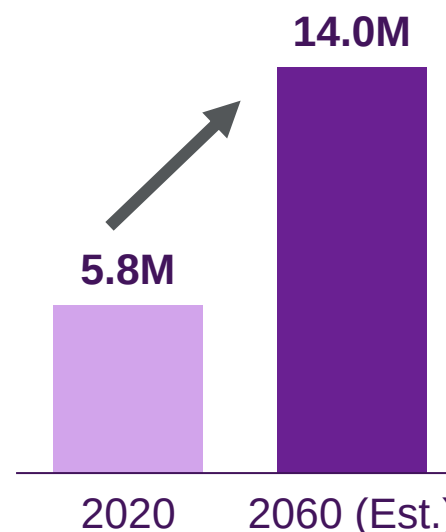
- Cortisol levels are elevated in brain fluid in early AD
- Chronic corticosteroid treatment leads to hippocampal atrophy and cognitive impairment
- Elevated cortisol levels are associated with clinical progression
- Alzheimer's disease mouse model: 30–60% inhibition of 11 β -HSD1 provides full neuroprotection
- AD Phase 2a trial shows slowed disease progression in biomarker-positive patients
- ***Safe & effective oral therapy is “holy grail”***

Growing Alzheimer's Disease market – U.S.

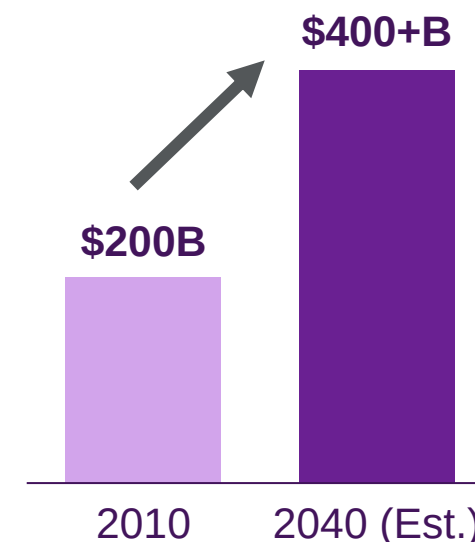
Large unsatisfied and growing market



of patients



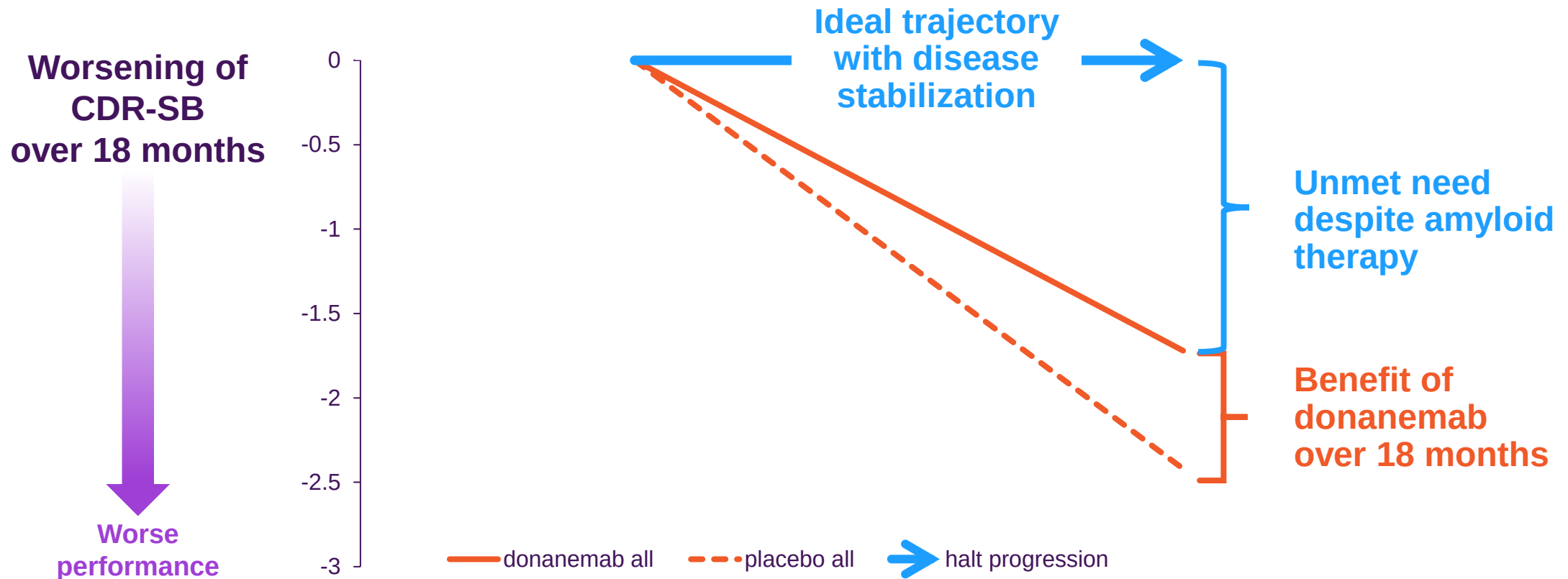
Cost to treat



Anti-amyloid therapy modestly slows AD progression



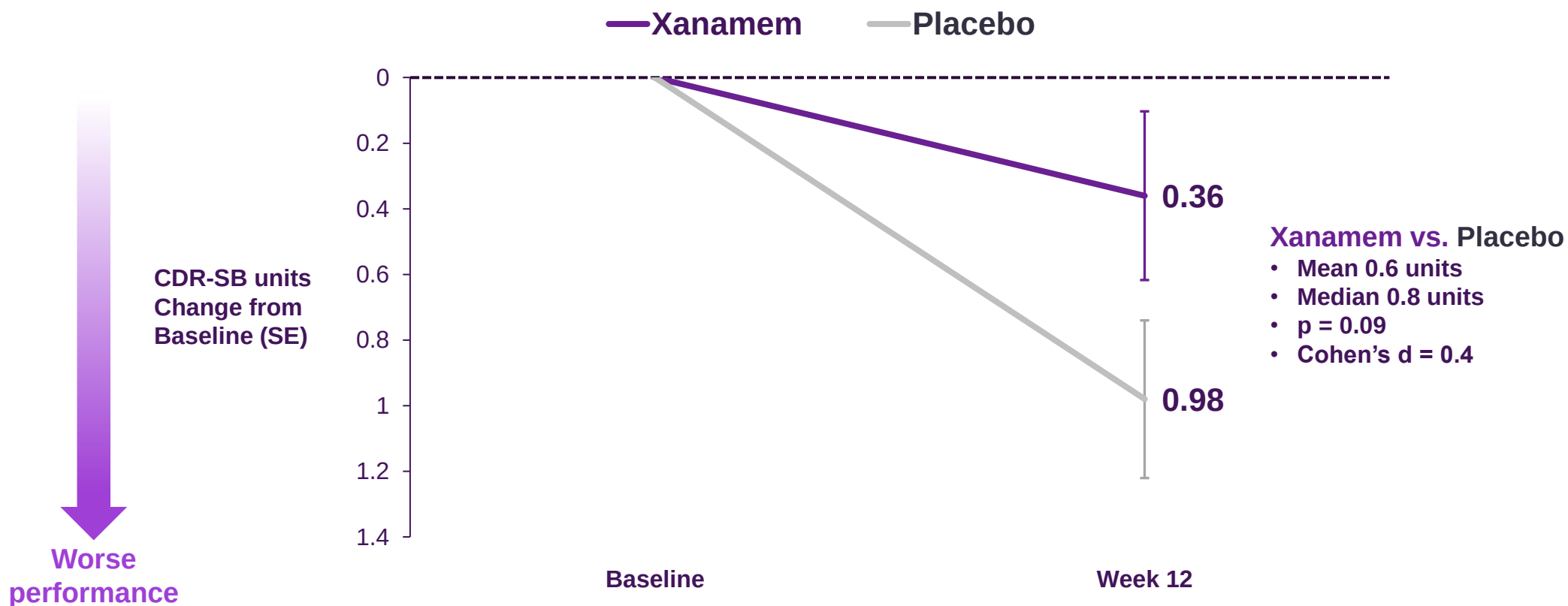
Ideally patients with AD would not worsen on treatment at all



Drugs targeting other mechanisms like Xanomem are needed

Xanamem benefit in pTau181-positive AD patients

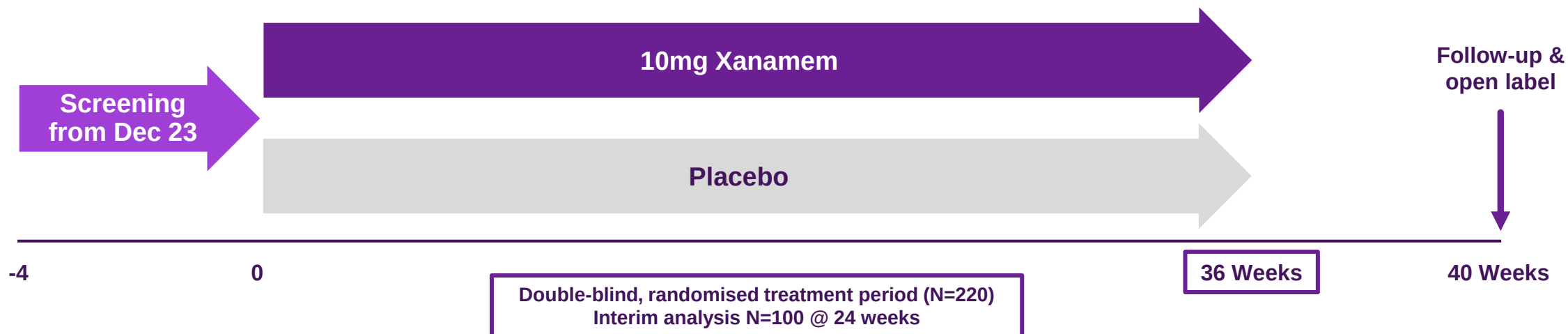
Phase 2a biomarker study: major slowing of CDR-SB decline (n=34)¹



Journal of Alzheimer's Disease 100 (2024) 139–150
 Plasma pTau181 Predicts Clinical Progression in a Phase 2 Randomized
 Controlled Trial of the 11-HSD1 Inhibitor Xanamem® for Mild Alzheimer's Disease
 Jack Taylor, Mark Jaros, Christopher Chen, John Harrison and Dana Hilt

XanaMIA phase 2b/3 trial in Alzheimer's disease

Initial, interim results in mid 2025, final results mid 2026



Key Inclusion Criteria	Primary Endpoint	Key Secondary Endpoints	Implementation
- Blood pTau biomarker positive - Mild-moderate Alzheimer's by NIA-AA criteria	CDR-SB (functional and cognitive measure)	- Cognitive Test Battery (7 cognitive measures well-validated in the Alzheimer's field) - Amsterdam Activity of Daily Living (functional measure)	- Enrolment at 15 Australian & 4 US sites - Currently expanding sites in US - Interim analysis planned when ~100 people complete 24 weeks

Depression program



There remains significant unmet need in depression

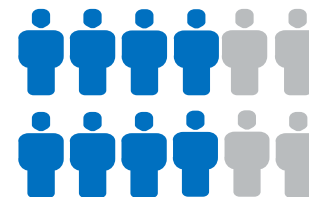
Xanamem's unique mechanism and safety differentiate it from older drugs

Scientific rationale

- More than 50 years of research associates cortisol with depression
- Elevated CSF and plasma cortisol levels associated with diagnosis, treatment outcomes and relapse
- Positive effects of cortisol receptor antagonism reported with mifepristone³
- ***Now positive phase 2a data on depressive symptoms for Xanamem (MADRS, PGI-S)***

U.S. Depression market large unmet need

- 21M patients have had ≥ 1 MDD episode

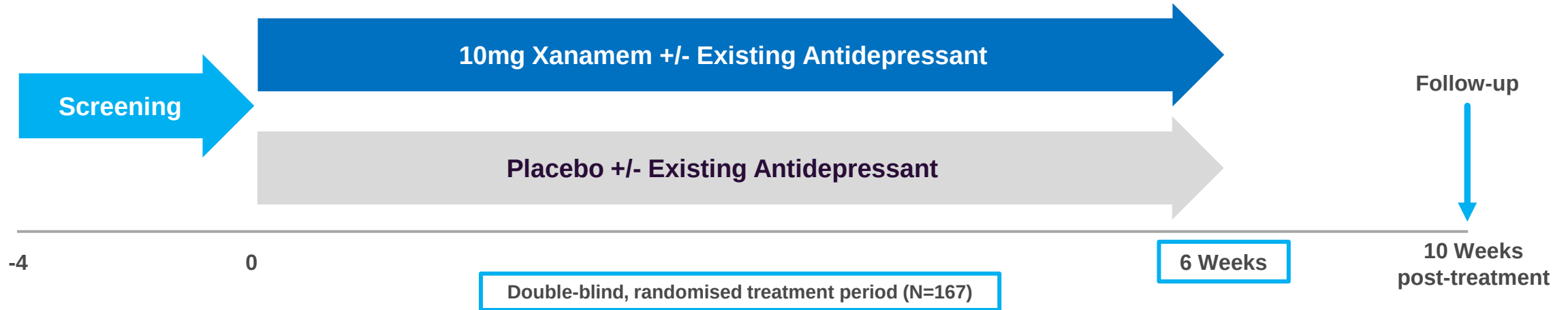


- Two-thirds with an episode **with severe impairment** in the past year
- 61% of all adults with MDD episodes receive treatment
- ≥ 365 M prescriptions per year

A safe, durably effective and combinable small molecule is a very attractive product profile for depression

XanaCIDD trial design and methods

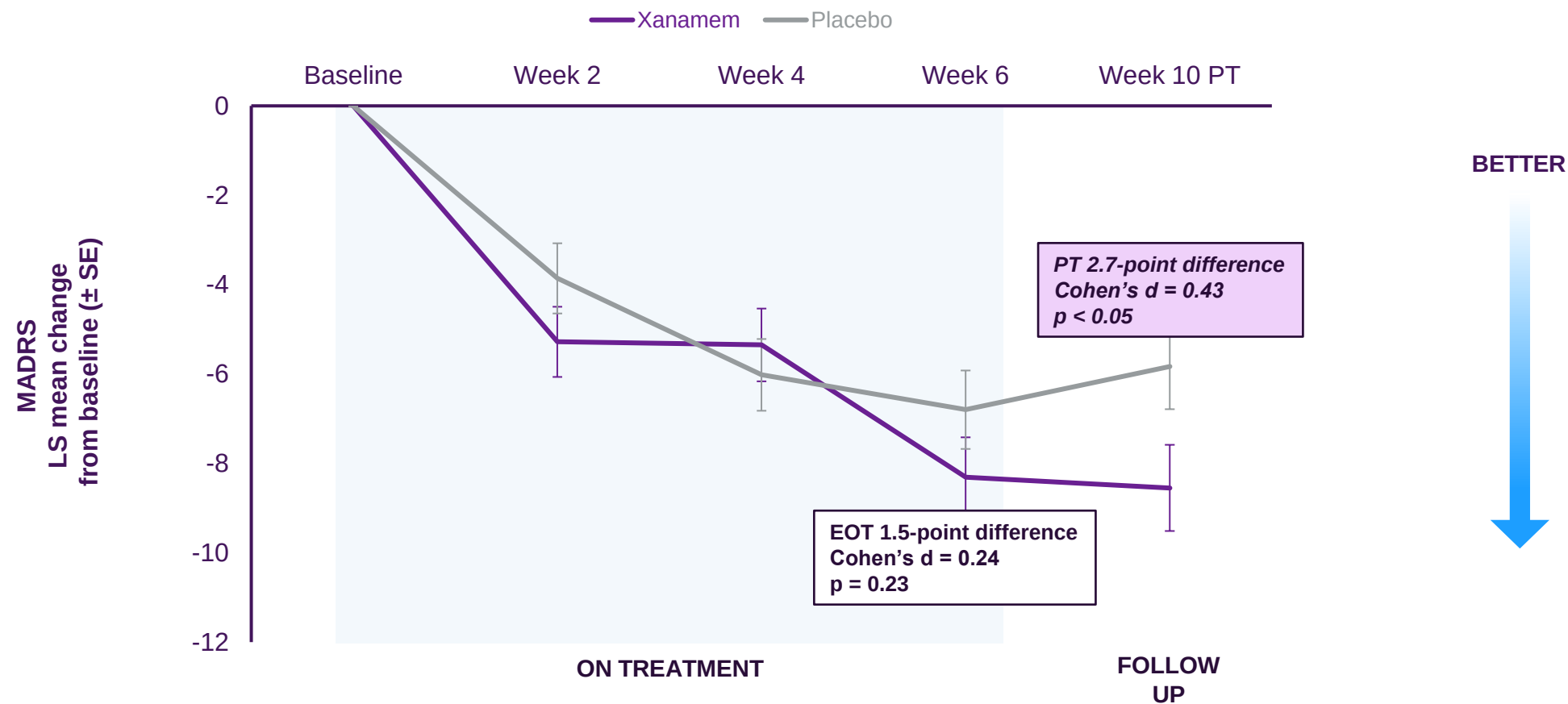
Phase 2, double-blind, proof-of-concept controlled trial to assess safety and efficacy



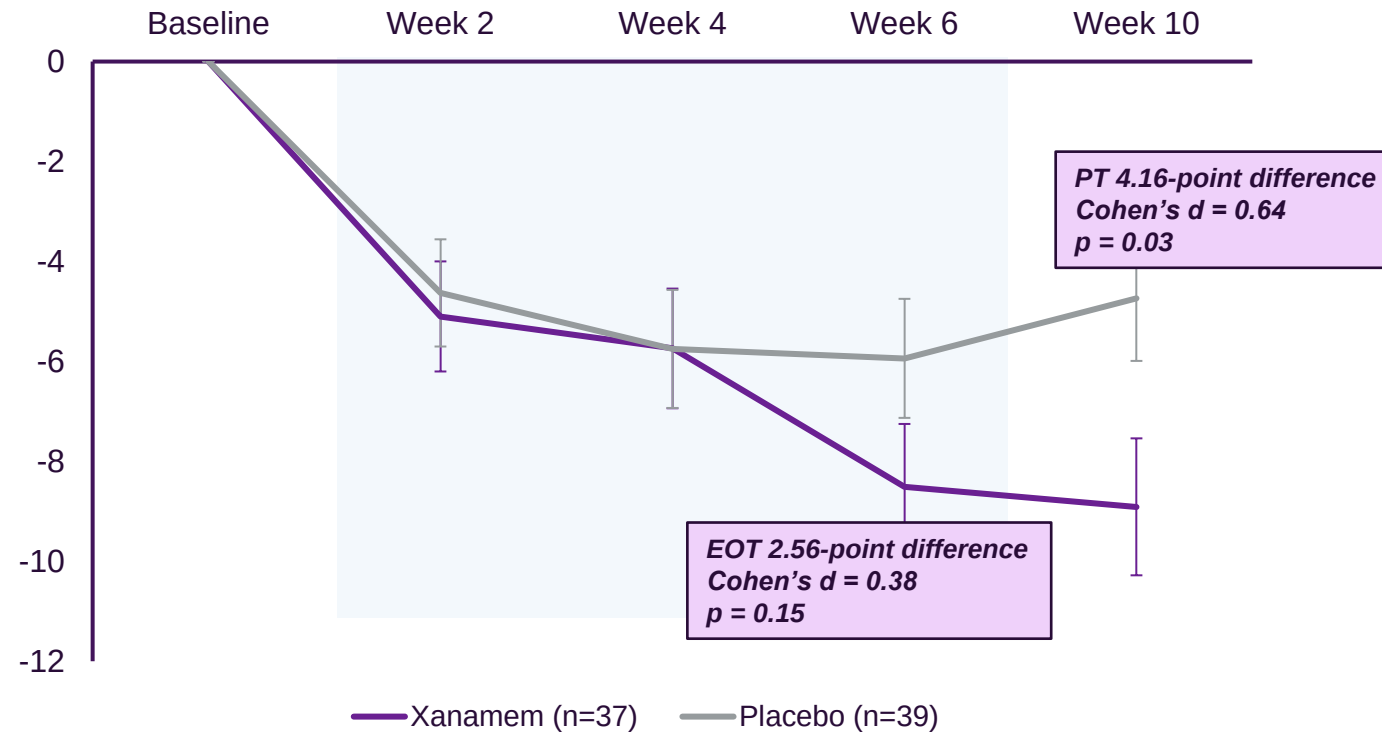
Primary Endpoint	Key Secondary Endpoints
• Cogstate Cognitive Test Battery Attention Composite (attention and working memory)	• Montgomery-Åsberg Depression Rating Scale (MADRS) • Patient Global Impression-Severity (PGI-S) • Executive Function Cognitive Composite (EFC) • Memory Function Cognitive Composite (MC)

Xanamem MADRS separation from Week 6 (n=165)

All randomized participants

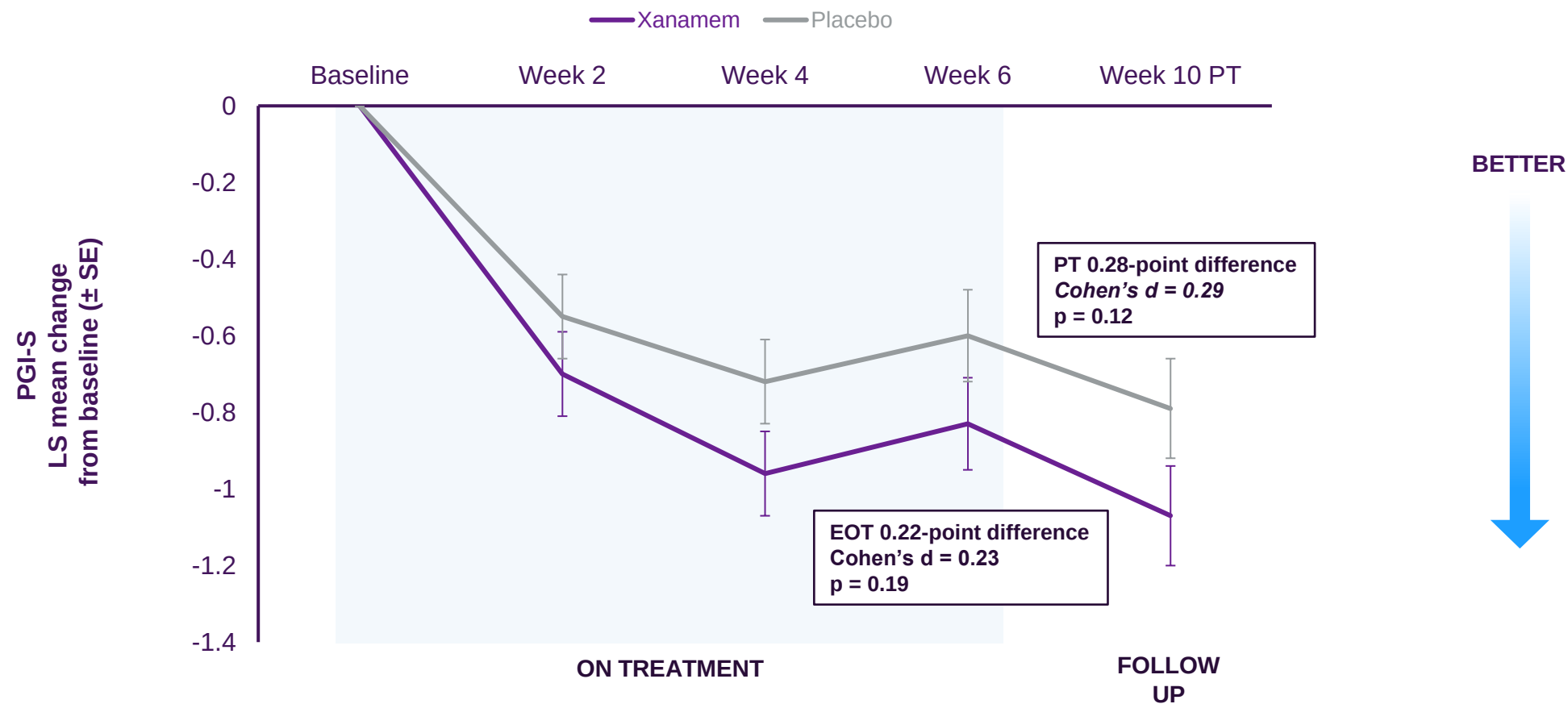


MADRS in subgroup taking concurrent SSRI* (n=76)



Xanamem PGI-S separation from Week 2 (n=165)

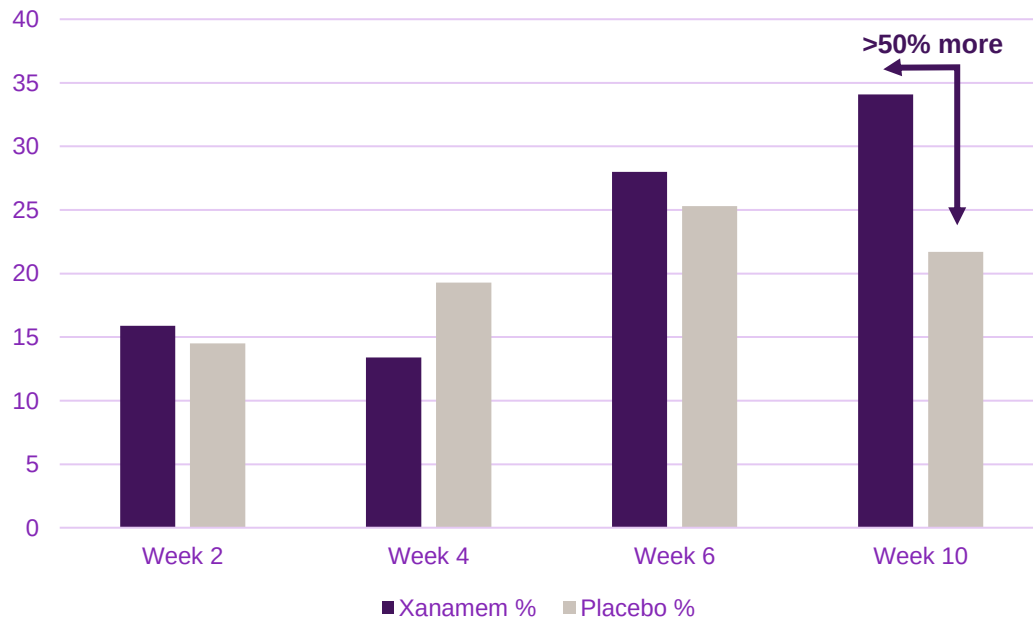
All randomized participants



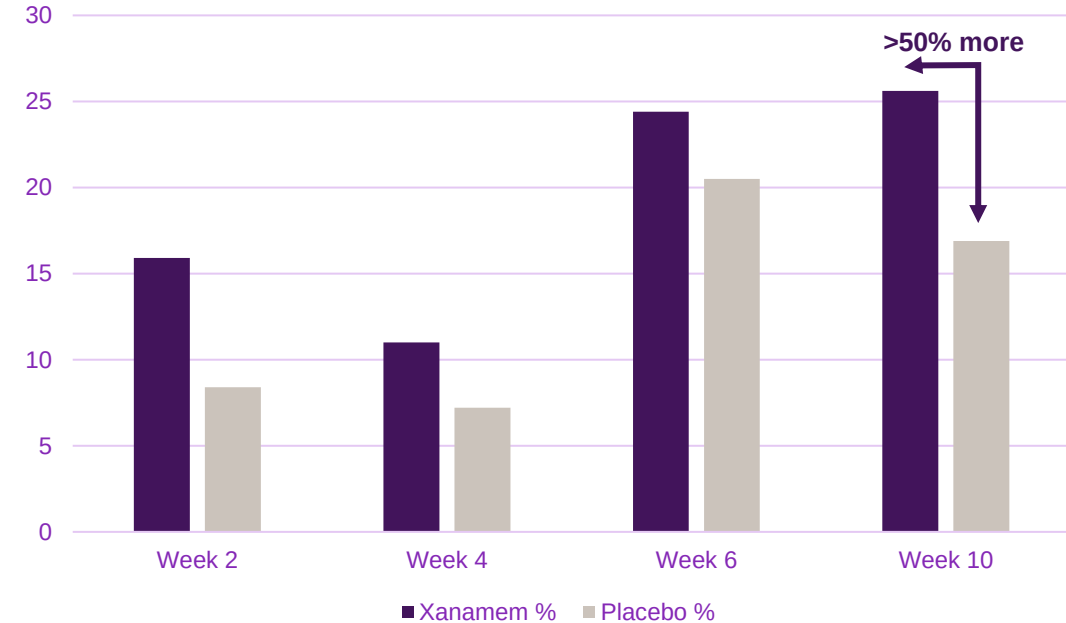
Xanamem improves MADRS response rates (n=165)

Increased rates of remission (MADRS < 10) and large (50%) improvements

% with $\geq 50\%$ reduction in MADRS



% with < 10 points on MADRS



Excellent safety profile consistent with prior trials

Summary of Treatment-Emergent Adverse Effects (TEAE)

	Xanamem N = 82	Placebo N = 83	Overall N = 165
Any TEAE	69 (84.1%)	67 (80.7%)	136 (82.4%)
TEAE related to trial drug	27 (32.9%)	24 (28.9%)	51 (30.9%)
Serious adverse event	0	1 (1.2%)	1 (0.6%)
Related TEAE discontinuation or interruption of drug	3 (3.7%)	1 (1.2%)	4 (2.4%)
TEAEs with incidence $\geq$ 5% overall			
Headache	11 (13.4%)	16 (19.3%)	27 (16.4%)
Fatigue	6 (7.3%)	5 (6.0%)	11 (6.7%)
Nasopharyngitis	4 (4.9%)	6 (7.2%)	10 (6.1%)
Upper respiratory tract infection	5 (6.1%)	5 (6.0%)	10 (6.1%)

Depression phase 2a conclusions and next steps



- ***Clinically and statistically significant treatment benefits on depressive symptoms for MADRS and patient-reported outcome of severity***
- Heavily pre/co-treated population with moderate MDD
- Consistent depression efficacy across subgroups
- Cognition improved markedly in both Xanamem and placebo groups without evidence of greater Xanamem benefit vs. placebo (data not shown)
- Xanamem was safe and well tolerated (n=165 treated) with no suggestion of suicide risk or withdrawal syndrome
- The trial was well-conducted, with excellent data quality, no major differences between Australia and the UK or at high enrolling clinical sites
- Funding for the next depression trial being investigated with potential partners and/or granting bodies

Conclusion



Positive depression data a boost to both AD and MDD



Evidence of durable benefit on depression from control of brain cortisol validates the Xanomem program in terms of:

- ✓ “Cortisol control” mechanism of action
- ✓ 10 mg daily proof-of-concept dose being used in Alzheimer’s phase 2b/3 trial
- ✓ 10 mg daily dose is also suitable for next depression trial

Phase 2a MDD data support further clinical development in MDD

Clinical activity on depression supports the likelihood of seeing a disease-modifying effect in Alzheimer’s disease over 36 weeks

Company funded to at least H2 2026

Appendix



Key references

Other references see also <https://actinogen.com.au/xanamem>



11 β -HSD1 inhibition

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- Cognitive and disease-modifying effects of 11 β -hydroxysteroid dehydrogenase type 1 inhibition in male Tg2576 mice, a model of Alzheimer's Disease: Sooy, K., Noble, J., McBride, A., Binnie, M., Yau, J. L. W., Seckl, J. R., Walker, B. R., & Webster, S. P. 2015. *Endocrinology*, 1-12.
- Partial deficiency or short-term inhibition of 11 β -hydroxysteroid dehydrogenase type 1 improves cognitive function in aging mice Sooy, K., Webster, S. P., Noble, J., Binnie, M., Walker, B. R., Seckl, J. R., & Yau, J. L. W. 2010. *Journal of Neuroscience*, 30(41), 13867-13872.

Xanamem clinical trials

- Plasma pTau181 Predicts Clinical Progression in a Phase 2 Randomized Controlled Trial of the 11 β -HSD1 Inhibitor Xanamem® for Mild Alzheimer's Disease Taylor J, Jaros M, Chen C, Harrison J, Hilt D *J Alz Dis* 2024; 100: 139-150
- Brain 11-Hydroxysteroid Dehydrogenase Type 1 Occupancy by Xanamem™ Assessed by PET in Alzheimer's Disease and Cognitively Normal Individuals Villemagne VL, Dore V, Chong L, Kassiof M, Mulligan, R, Feizpoura A, Taylor J, Roesner M, Miller T, Rowe CC *J Alz Dis* 2024; 97: 1463–1475
- Selection and early clinical evaluation of the brain-penetrant 11 β -hydroxysteroid dehydrogenase type 1 (11 β -HSD1) inhibitor UE2343 (Xanamem™) Webster, S. P., Ward, P., Binnie, M., Craigie, E., McConnell, K. M., Sooy, K., Vinter, A., Seckl, J.R. & Walker, B. R. 2007. *Bioorganic & medicinal chemistry letters*, 17(10), 2838-2843.
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Alzheimer's disease and cortisol

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Depression and cortisol

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- Effect of glucocorticoid and 11 β -hydroxysteroid-dehydrogenase type 1 (11 β -HSD1) in neurological and psychiatric disorders Dodd S, Skvarc D R, Dean OM, Anderson A, Kotowicz M, Berk M *Int J Neuropsychopharmacol* 2022; 25(5):387-398
- Depression and Hypothalamic-Pituitary-Adrenal Activation: A Quantitative Summary of Four Decades of Research Stetler C, Miller GE *Psychosom Med* 2011; 73(2):114-26

Market & cost of treatment estimates

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- <https://www.cdc.gov/aging/aginginfo/alzheimers.htm#treated>
- <https://www.nimh.nih.gov/health/statistics/major-depression>
- Symphony Health and ICON plc Company, Metys® database full year 2023

Currencies

- Currencies are in Australian dollars unless otherwise stated

Selected Glossary 1

- **11 β -HSD1** – 11 beta HydroxySteroid Dehydrogenase-1 enzyme. Selectively expressed in brain, liver, adipose.
- **A β** – Amyloid beta – a type of amyloid protein associated with Alzheimer’s Disease, 42 and 40 are different forms
- **ACTH** – Adrenocorticotrophic hormone that regulates blood levels of cortisol
- **ADAS-Cog** – Alzheimer’s Disease Assessment Score - Cognition
- **ApoE4** – Apoprotein genotype associated with genetic risk of Alzheimer’s Disease
- **ATN** – Amyloid, Tau, Neurodegeneration
- **Clinical Scales** – Measure how a patient feels, performs and functions
- **CDR-SB** – Clinical Dementia Rating “Sum of Boxes” scale measuring cognition and function on an 18-point scale (high worse)
- **CNS** – Central nervous system
- **CSF** – Cerebrospinal fluid
- **CTAD** – Clinical Trials on Alzheimer’s Disease (conference)
- **CTB** – Cognitive Test Battery of computerized tests
- **Double-blind** – Investigators, participants and company do not know who has active vs placebo treatment during a trial
- **EMA** – European Medicines Agency
- **FDA** – US Food & Drug Administration
- **Filamen A** – A protein believed to relate to amyloid toxicity
- **GFAP** – Glial Fibrillary Acidic Protein – a marker of microglial cell activation in the brain
- **IDSST** – International Digit Symbol Substitution Test of cognition

Selected Glossary 2

- **IQCODE** – Informant Questionnaire on Cognitive Decline in the Elderly
- **MCI** – Mild Cognitive Impairment – memory, executive function deterioration with retained functional abilities
- **MDD** – Major Depressive Disorder
- **MMSE** – Mini Mental State Examination – a 30-point scale of simple questions to assess mental abilities
- **NfL** – Neurofilament Light – a nerve protein in the brain and rest of the body too
- **NIA-AA** – National Institutes of Aging and Alzheimer’s Association
- **NMDA** – A type of receptor for glutamate in the brain
- **NPI** – Neuropsychiatric Inventory to assess psychiatric symptoms
- **NTB** – A Neurologic Test Battery, in this presentation one designed to measure executive function aspects of cognition
- **PET** – Positron Emission Tomography – a type of body scan
- **Placebo controlled** – Non-active treatment for double-blind design
- **p-Tau181 or 217 AD** – Biomarker of phosphorylated Tau protein
- **QPCT** – Glutaminyl-peptide cyclotransferase is an enzyme proposed to create toxic amyloid species
- **RAVLT** – Rey Auditory Visual Learning Test
- **RBANS** – Repeatable Battery for the Assessment of Neuropsychological Status (a test of mental abilities)
- **ROC AUC** – Receiver Operating Curve Area Under the Curve (1.0 ideal) – a type of statistical test to compared two methods of measurement
- **SSRI** – selective serotonin reuptake inhibitor
- **Tau** – A brain protein
- **Ttau** – Total tau levels including both phosphorylated and non-phosphorylated tau

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