Actinogen

Oral Xanamem® (emestedastat)

Controlling brain cortisol to slow progression in Alzheimer's disease and treat depression

Corporate Presentation

May 2025

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Overview

XanaMIA phase 2b/3 trial in AD accelerating enrolment

Full enrolment Q4 2025, final results late 2026

- ***100th patient enrolled expected Q2 2025, triggering interim analysis late 2025***
- ***220 participants full enrolment Q4 2025 with final results late 2026***
- 35 active clinical sites in Australia and the US
- More than 550 screened with pTau blood test, screen fail rate as expected
- Validation of Xanamem 10 mg dose seen from benefits on depressive symptoms supports the likelihood of seeing a disease-modifying effect in Alzheimer's
- Xanamem target patient population is different than recently approved antibody drugs (later stage disease, no invasive monitoring required) providing clinical trial and commercial advantages
- Key FDA meeting on optimal path to marketing approval for Alzheimer's in H2 2025

Xanamem is in advanced stages of development

Novel 11β-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
- Over **400 people** treated with excellent safety and low drug interaction risk

Positive phase 2 clinical data de-risk Xanamem program

- **Disease-modifying activity on CDR-SB** in phase 2a trial in biomarker-positive Alzheimer's patients
- **Phase 2a MDD trial showing clinically & statistically significant activity - benefits across multiple endpoints**
- Positive data from both trials read through to other indications in psychiatry and the dementias

Patent/data protection and advanced manufacturing

- **Composition of matter protection** to 2031, and 2036 with extensions in major markets, newer patents in process
- **Data exclusivity protects Xanamem data** from use by others for 5 to 10 years from approval e.g. 10 years in EU
- **Manufacturing process scaled up and patented**, contractors Asymchem (China) & Catalent (US)

Large clinical and commercial opportunities

- **No other brain-penetrant cortisol control drugs in development, first to be awarded INN and USAN names**[1]
- 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

1. Xanamem's International Nonproprietary Name (INN) , emestedastat, was awarded by a naming committee of the World Health Organization: "-stedastat" chosen for the first time for all 11β-HSD1 inhibitors; USAN (United States Adopted Name)

Experienced board and management team

Board of Directors

Dr. Geoff Brooke
Chairman
MBBS; MBA

Dr. Steven Gourlay
CEO & MD
MBBS; FRACP; PhD; MBA

Mr. Malcolm McComas
Non-Executive Director
BEc, LLB; FAICD; SF Fin

Dr. George Morstyn
Non-Executive Director
MBBS; PhD; FRACP CD

Dr. Nicki Vasquez
Non-Executive Director
PhD

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Dr. Steven Gourlay
CEO & MD

Dr. Dana Hilt
Chief Medical Officer
MD

Will Souter
Chief Financial Officer
BComm, LLB

Andrew Udell
Chief Commercial Officer
MBA

Cheryl Townsend
VP Clinical Operations
RN, M Health Law

Fujun Li
Head of Manufacturing
PhD

Michael Roberts
Head of IR & Comms
B.Ec (Hons), CPA, FFIN

Corporate snapshot

ASX-listed company founded in 2014

- Market Cap ~$100 million
- **Cash balance of $18.7 million as of Mar 31, 2025, provides runway to at least mid 2026**
- Conducted three phase 1 (Australia) and four phase 2 trials (Australia, US and UK)

Key shareholders

- Biotech Value Fund (BVF) ~6%
- **CEO Steve Gourlay ~5% (including via ~$2 million invested personally)**
- Top 20 ex BVF & Gourlay ~23%

Phase 2b/3-stage clinical programs are the “sweet spot” for partnering

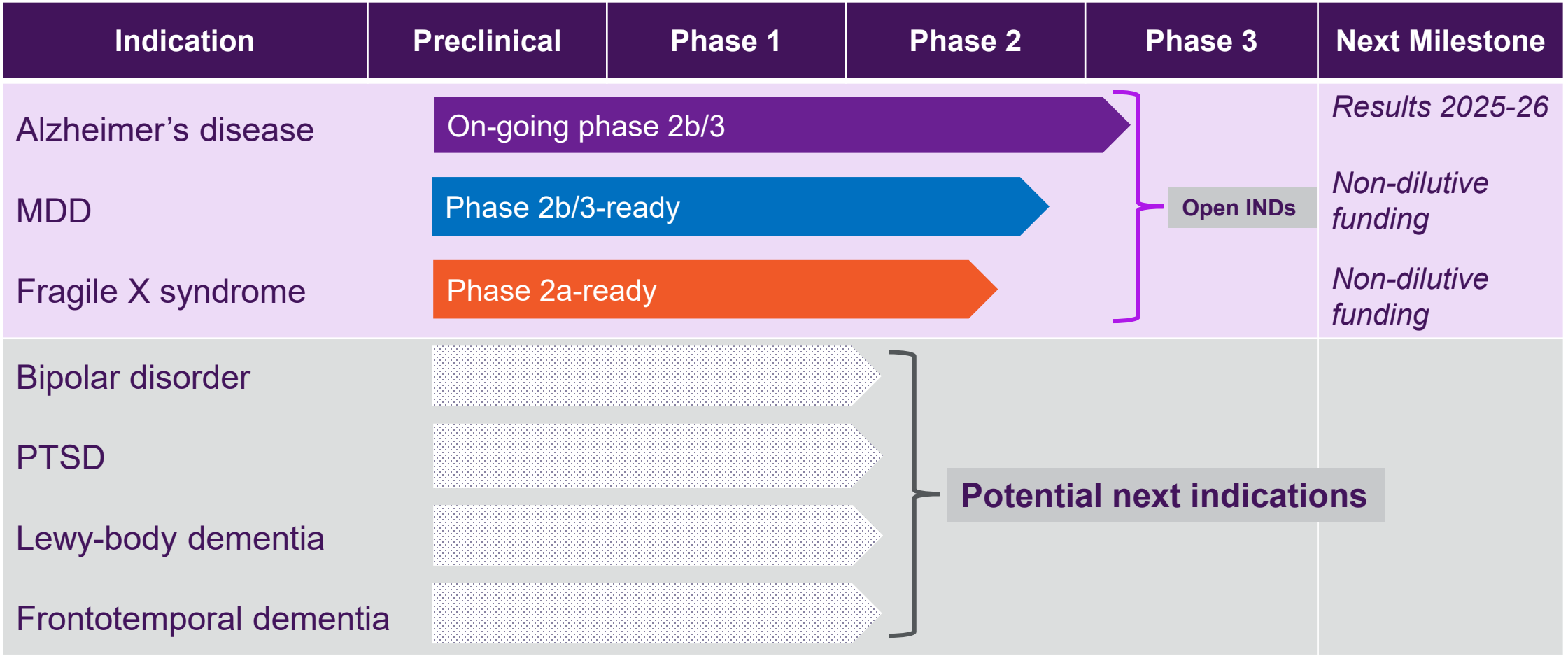
- **Alzheimer’s disease phase 2b/3 ongoing – interim Q4 2025, final results Q4 2026**
- **Major depressive disorder phase 2a completed – seeking partner(s) and grants**
- Type C meeting with FDA to discuss approval requirements for AD H2 2025

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 received in 2024)

Xanamem – Pipeline focused on Alzheimer's as lead

Depression phase 2a results support further development



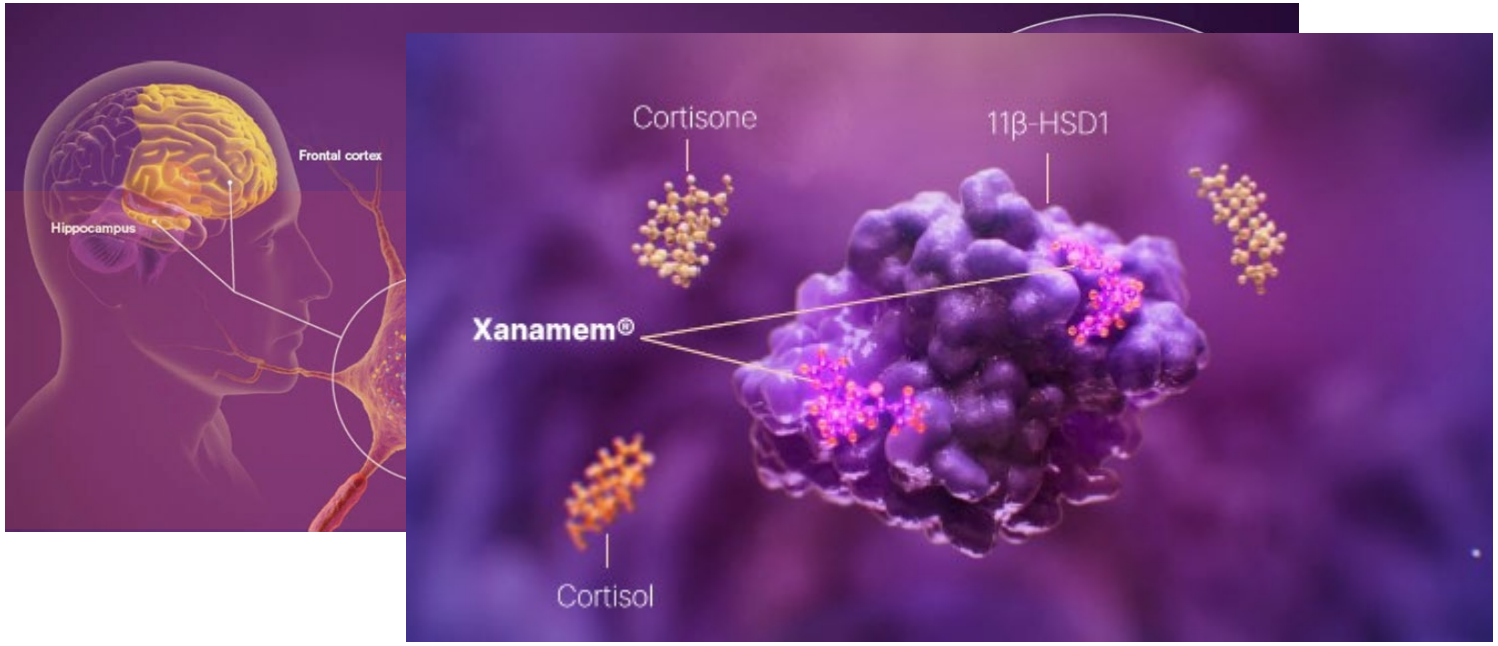
MDD: Major depressive disorder

Xanamem

Once-daily oral treatment with a unique mechanism

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

- Good safety profile in ~400 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 11β-HSD1[1]

Controlling brain cortisol[2] has potential durable benefits

Reduction of "stress response" in brain

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

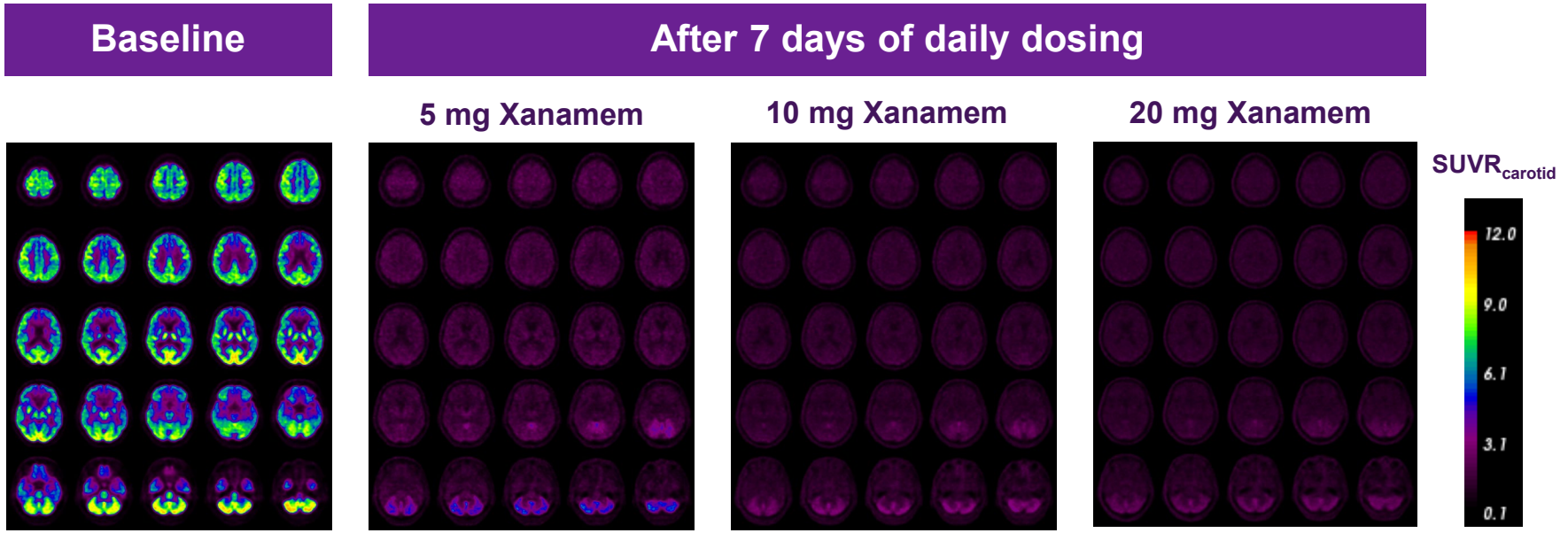
"Lower stress" longer term e.g.

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

1. For a comprehensive review of 11β-HSD1 see Seckl J. 2024 2. Ramamoorthy & Cidlowski 2016

Human PET study shows full target engagement

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



Xanamem extensively binds to the 11β-HSD1 enzyme throughout the brain, with high post-treatment effects (absence of color) 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 market is large and growing

Strong cortisol control scientific rationale to address huge unmet medical need

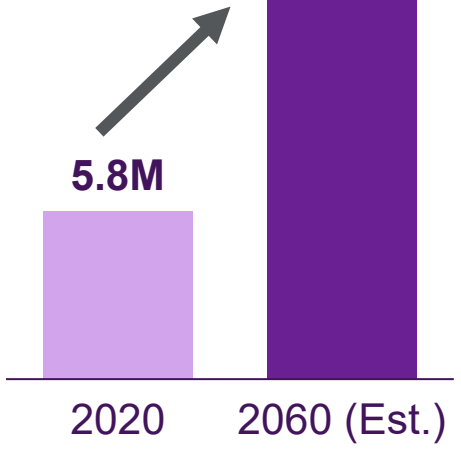
Rationale

- Cortisol levels 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 11β-HSD1 provides full neuroprotection
- AD phase 2a trial shows slowed disease progression in biomarker-positive patients
- ***Safe & effective oral therapy is "holy grail"***

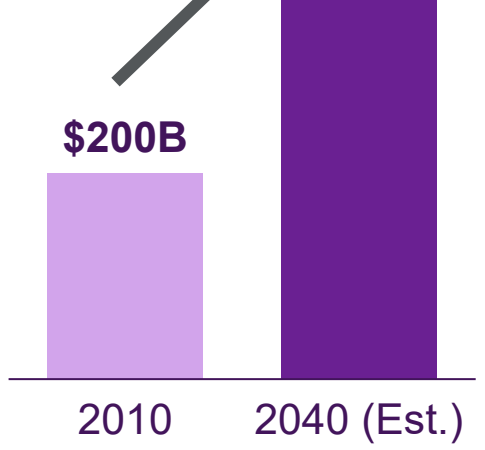
Growing Alzheimer's Disease market – U.S.

Large, unsatisfied and growing market

# of patients

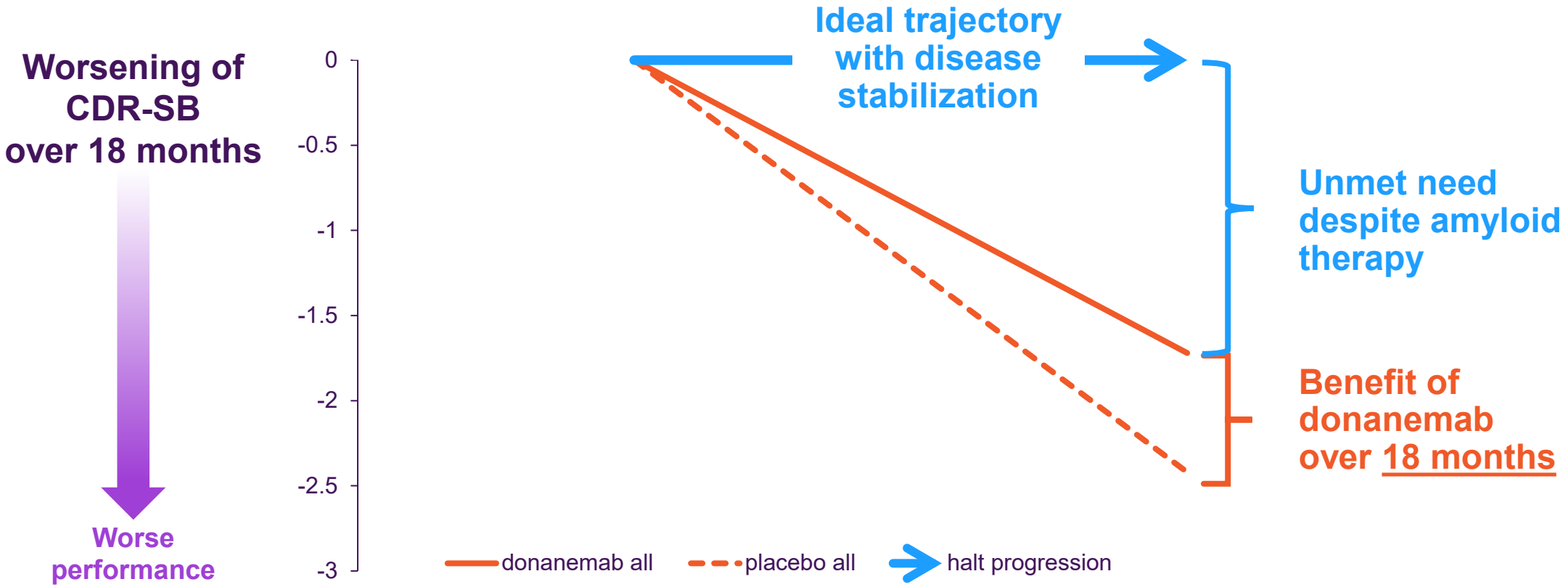


Cost to treat



Anti-amyloid therapy modestly slows AD progression

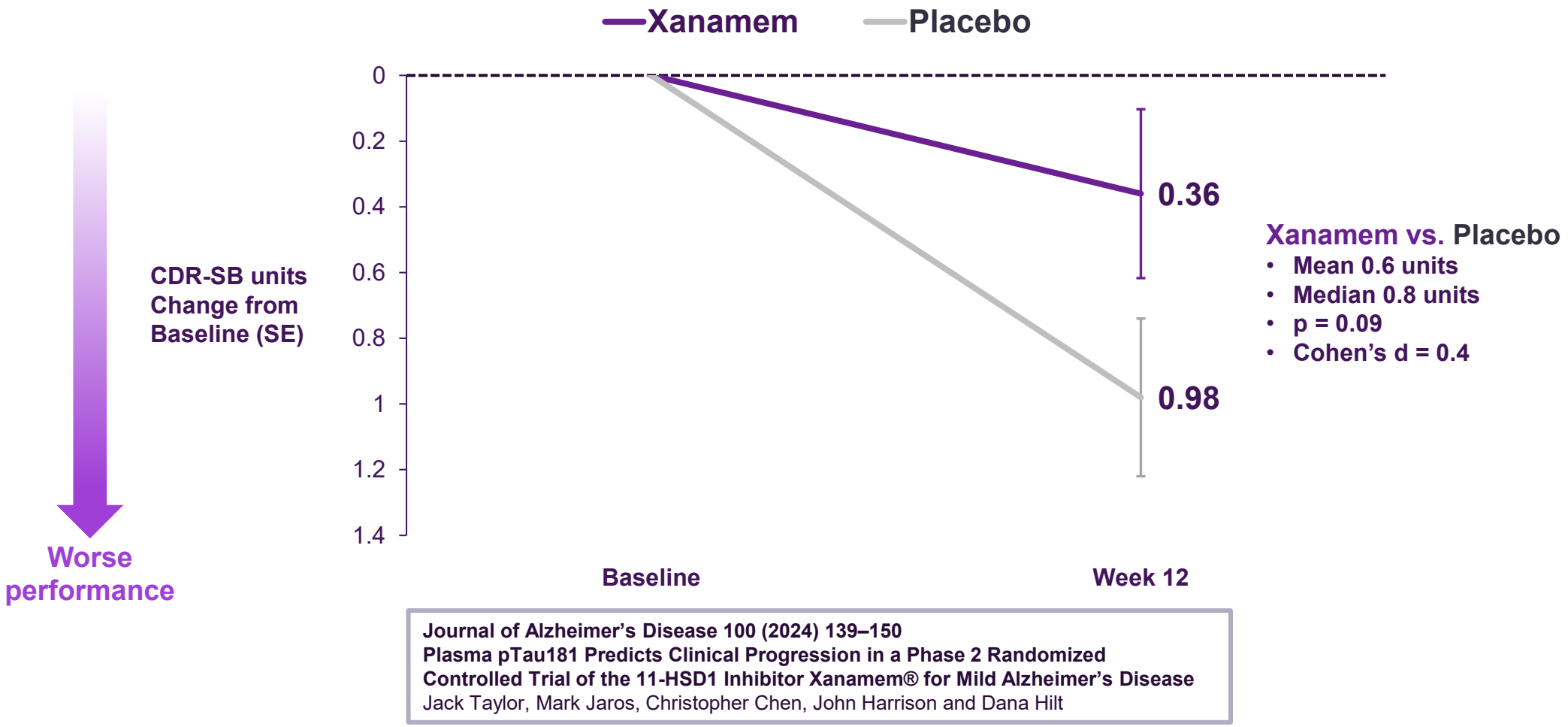
Ideally patients with AD would not worsen on treatment at all



Drugs targeting other mechanisms like Xanamem are needed

Large Xanamem benefit in high pTau181 patients

Phase 2a biomarker study: major slowing of CDR-SB decline over 12 weeks (n=34)



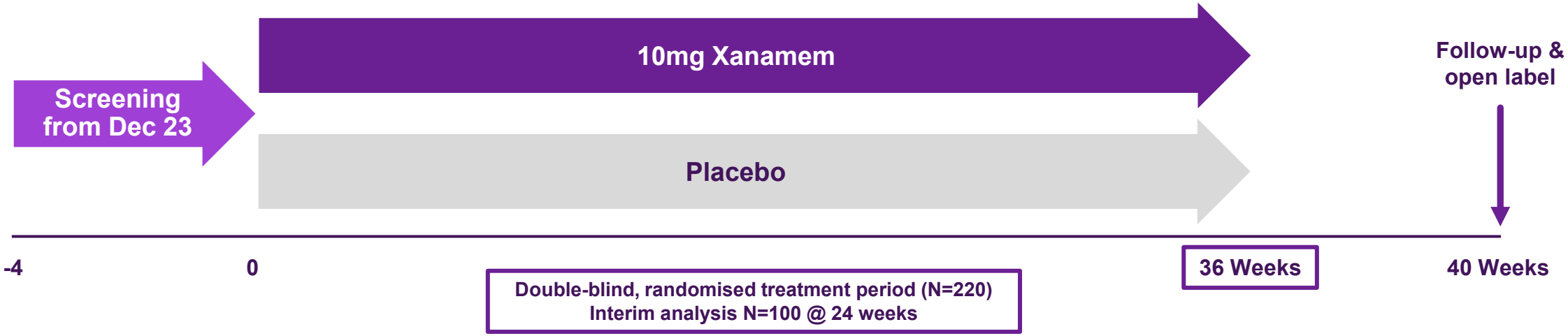
Xanamem vs. Placebo

- Mean 0.6 units
- Median 0.8 units
- p = 0.09
- Cohen's d = 0.4

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 Q4 2025, final results Q4 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) @36 weeks**	• Cognitive Test Battery (7 cognitive measures well-validated in the Alzheimer's field) • Amsterdam Activity of Daily Living (functional measure)	• Enrolment at 15 Australian & 20 US sites • Interim analysis planned when ~100 people complete 24 weeks

NIA-AA=National Institute of Aging - Alzheimer's Association; **CDR-SB** Clinical Dementia Rating Scale – Sum of Boxes

Depression program

There remains significant unmet need in depression

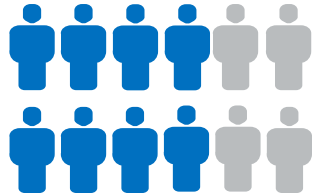
Xanamem's unique mechanism and safety differentiate it from older drugs

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 mifepristone[3]
- ***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 AND Alzheimer's

3. Ding et al 2021

Phase 2a depression symptom benefits (2024) - major scientific and drug development achievement

Data support further MDD development and are a positive for Alzheimer's too

- ***Clinically and statistically significant treatment benefits on depressive symptoms for MADRS and patient-reported outcome of severity***
- Predominantly co-treated population with moderate MDD
- Consistent depression efficacy across subgroups
- Xanamem was safe and well tolerated (n=165 treated) with no observed suicide risk or withdrawal syndrome
- The trial was well-conducted with no major differences between Australia and the UK or at high enrolling clinical sites
- Benefits on depression are a desirable feature for an Alzheimer's drug
- Funding for the next depression trial being investigated with potential partners and/or granting bodies

Further XanaCIDD phase 2a depression trial results shown in the Appendix

Conclusion

Building momentum toward Alzheimer's results

Numerous value-add milestones in 2025 and 2026 before final XanaMIA results late 2026

- ***Positive phase 2a MDD data validates multiple Xanamem programs***
  - ✓ Clinical activity of unique "cortisol control" mechanism of action
  - ✓ Clinical activity of the 10 mg daily dose being used in all trials
  - ✓ Reinforces the likelihood of seeing a disease-modifying effect in Alzheimer's disease over 36 weeks in current XanaMIA trial
- ***Company funded to at least mid 2026***
- ***Commercial planning underway*** in preparation for early marketing approvals and partnership(s)
- ***Trial, regulatory, publication and presentation milestones*** in coming 18 months

Multiple upcoming 2025 milestones

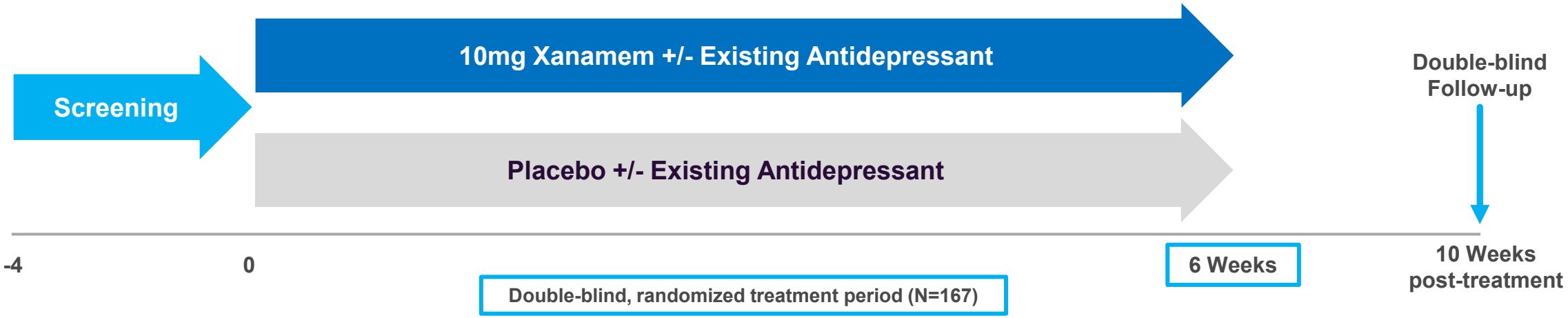
Milestone	Likely Timing
First patient randomized and treated in US, XanaMIA trial	Q4 24
Meetings at JP Morgan Healthcare conference week, San Francisco	Q1 25
Clinical pharmacology manuscript peer-reviewed publication	Q1 25
FDA Type C meeting for MDD	Q1 25
ADPD conference AD presentation in Vienna	Q2 25
All 20 US sites actively enrolling	Q2 25
Clinical Trials Science Forum – focus on commercial planning	Q2 25
100th patient enrolled, XanaMIA trial	Q2 25
XanaCIDD MDD peer-reviewed journal publication	Q3-4 25
American Psychiatric Association MDD presentation, Los Angeles	Q2 25
FDA Type C meeting for AD	H2 25
Interim analysis, XanaMIA trial	Q4 25
Full enrolment, 220 patients with AD, XanaMIA trial	Q4 25
AAIC conference AD presentation in Toronto	Q3 25
CTAD conference AD presentation in San Diego	Q4 25

Completed

Appendix

XanaCIDD trial design and methods – completed CY2024

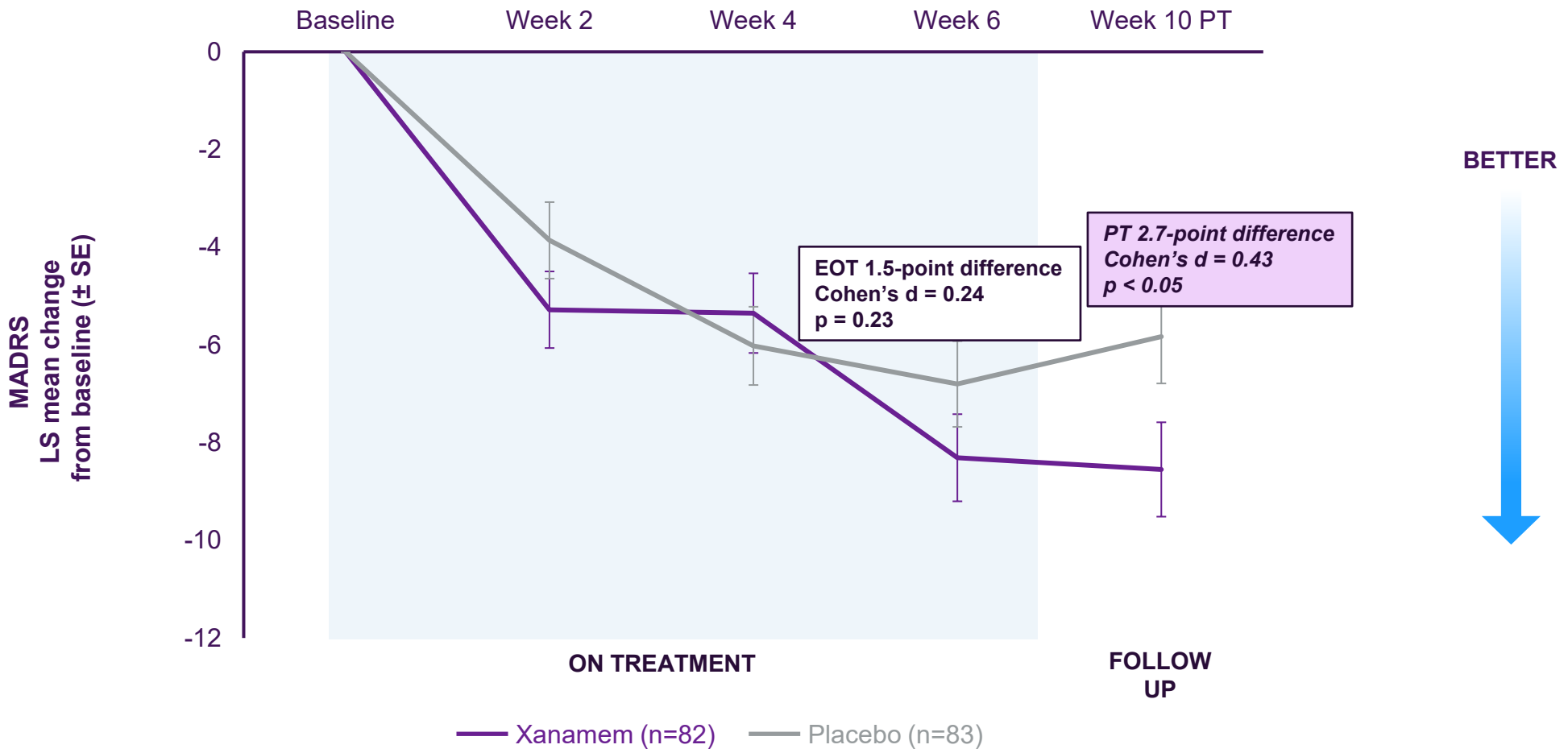
Phase 2, double-blind, proof-of-concept controlled trial to assess safety and efficacy



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)** • Other MDD and cognitive measures

Xanamem MADRS improvement from Week 6 (n=165)

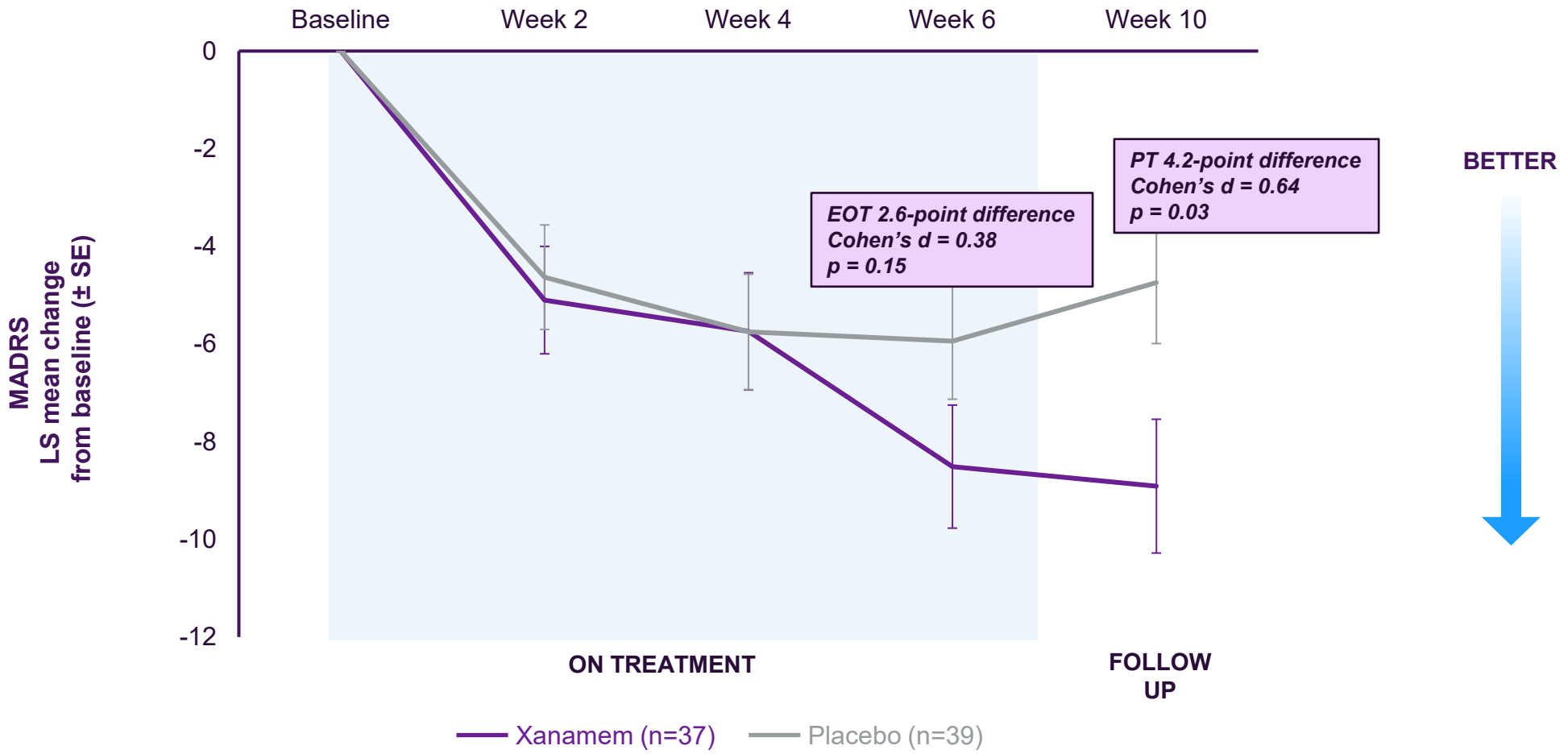
All randomized participants



Abbreviations: MADRS = Montgomery-Åsberg Depression Rating Scale – a structured interview to assess depression severity

MADRS benefit in patients also taking SSRI (n=76)

Largest co-treatment subgroup



Abbreviations: SSRI, selective serotonin reuptake inhibitor

Key references

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

11β-HSD1 inhibition

- Seckl J. 11β-Hydroxysteroid dehydrogenase and the brain: Not (yet) lost in translation. J Intern Med. 2024 Jan;295(1):20-37. doi: 10.1111/joim.13741. Epub 2023 Nov 8. PMID:37941106. https://onlinelibrary.wiley.com/doi/10.1111/joim.13741
- Cognitive and disease-modifying effects of 11β-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 11β-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 11β-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, Kassiouf 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 11β-hydroxysteroid dehydrogenase type 1 (11β-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.*
- Various podium and poster presentations on website

Technical references

- CDR-SB Clinical Dementia Rating Scale – Sum of Boxes is an 18-point, 6-domain measure of patient cognition and function and is a common endpoint used by regulators. Patients in the Xanamem biomarker phase 2a analysis had a baseline of approximately 4 points, similar to that in the donanemab phase 3.
- Cohen, J. (1992). A power primer. *Psychological Bulletin, 112*(1), 155–159. https://doi.org/10.1037/0033-2909.112.1.155
- Hengartner MP, Jakobsen JC, Sørensen A, Plöderl M (2020) Efficacy of new-generation antidepressants assessed with the Montgomery-Asberg Depression Rating Scale, the gold standard clinician rating scale: A meta-analysis of randomised placebo-controlled trials. PLOS ONE 15(2): e0229381. https://doi.org/10.1371/journal.pone.0229381

Alzheimer's disease and cortisol

- Plasma Cortisol, Brain Amyloid-β, and Cognitive Decline in Preclinical Alzheimer's Disease: A 6-Year Prospective Cohort Study Pietrzak RH, Laws SM, Lim YY et. al. for the Australian Imaging, Biomarkers and Lifestyle Research Group 2017. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging* 2017; 2(1):45-52
- Decrease in cortisol reverses human hippocampal atrophy following treatment of Cushing's disease Starkman, M. N., Giordani, B., Gebarski, S. S., Berent, S., Schork, M. A., & Schteingart, D. E. 1999. *Biol psych, 46(12), 1595-1602.*

Depression and cortisol

- Ding et. al. Front. Pharmacol 2021 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8461240/
- Effect of glucocorticoid and 11β-hydroxysteroid-dehydrogenase type 1 (11β-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

- Matthews, K. A., Xu, W., Gaglioti, A. H., Holt, J. B., Croft, J. B., Mack, D., & McGuire, L. C. (2018). Racial and ethnic estimates of Alzheimer's disease and related dementias in the United States (2015–2060) in adults aged≥ 65 years. Alzheimer's & Dementia. https://doi.org/10.1016/j.jalz.2018.06.3063
- Hurd MD, Martorell P, Delavande A, Mullen KJ, Langa KM. Monetary costs of dementia in the United States. *NEJM*. 2013;368(14):1326-34.
- 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

- **11β-HSD1 –** 11 beta HydroxySteroid Dehydrogenase-1 enzyme. Selectively expressed in brain, liver, adipose.
- **Aβ –** Amyloid beta – a type of amyloid protein associated with Alzheimer's Disease, 42 and 40 are different forms
- **ACTH –** Adrenocorticotropic hormone that regulates blood levels of cortisol
- **AD –** Alzheimer's disease
- **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 Fibrilliary 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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