



ASX ANNOUNCEMENT

Actinogen CEO discusses company's late-stage Alzheimer's trial and commercialization program at the 2025 Bioshares Biotech Summit

Sydney, 7 August 2025. Actinogen Medical ASX: ACW ("ACW" or "the Company") is pleased to announce that its CEO, Dr Steven Gourlay, will present today at the 2025 Bioshares Biotech Summit in Hobart in the *Drug Development Journey* session of the conference.

The Summit is a key event for leading emerging ASX-listed lifescience and biotech organisations, with 35 companies presenting to investors over the two-day conference. Dr Gourlay will be joined at Bioshares by ACW's Chief Financial Officer, Mr Will Souter.

Dr Gourlay's Q&A style presentation is titled *Oral Xanamem® (emestedastat) Controlling brain cortisol to slow progression in Alzheimer's disease and treat depression*. His presentation addresses important investor questions in ACW's drug development story to date:

1. What previous results give the company confidence in a positive phase 2b/3 trial outcome in Alzheimer's?
2. What does XanaMIA trial success look like and what level of interest is there from potential partners?
3. What have you learned about commercialization so far?

Dr Gourlay's conference presentation is attached to this announcement.

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Investors

Dr. Steven Gourlay
CEO & Managing Director
P: +61 2 8964 7401
E: steven.gourlay@actinogen.com.au

Michael Roberts
Investor Relations
M: +61 423 866 231
E: michael.roberts@actinogen.com.au

Media

George Hazim
Media & Public Affairs Australia
M: +61 417 516 262
E: georgehazim@mediaaffairs.com.au

Announcement authorised by the Managing Director of Actinogen Medical

About Actinogen Medical

Actinogen Medical (ACW) is an ASX-listed, biotechnology company developing a novel therapy for neurological and neuropsychiatric diseases associated with dysregulated brain cortisol. There is a strong association between cortisol and detrimental changes in the brain, affecting cognitive function, harm to brain cells and long-term cognitive health.

® Xanamem is a registered trademark of Actinogen Medical Limited

Cognitive function means how a person understands, remembers and thinks clearly. Cognitive functions include memory, attention, reasoning, awareness and decision-making.

Actinogen is currently developing its lead compound, Xanamem, as a promising new therapy for Alzheimer's Disease and Depression and hopes to study Fragile X Syndrome and other neurological and psychiatric diseases in the future. Reducing cortisol inside brain cells could have a positive impact in these and many other diseases. The cognitive dysfunction, behavioural abnormalities, and neuropsychological burden associated with these conditions is debilitating for patients, and there is a substantial unmet medical need for new and improved treatments.

Clinical Trials

The XanaMIA Phase 2b/3 Alzheimer's disease trial is a double-blind, 36-week treatment, placebo-controlled, parallel group design trial in 220 patients with mild to moderate AD and progressive disease, determined by clinical criteria and confirmed by an elevated level of the pTau181 protein biomarker in blood. Patients receive Xanamem 10 mg or placebo, once daily, and its ability to slow progression of Alzheimer's disease is assessed with a variety of endpoints. The primary endpoint of the trial is the internationally-recognized CDR-SB (Clinical Dementia Rating scale – Sum of Boxes). The trial is being conducted in Australia and the US. Initial results from an interim analysis triggered by the 100th participant reaching 24 weeks of treatment are anticipated in January 2026 and final results Q4 2026.

The XanaMIA-DUR Alzheimer's disease open-label extension trial is an open-label trial of up to 24 months where all participants will receive active Xanamem 10 mg once daily. The trial will evaluate safety and a limited number of efficacy endpoints such as the CDR-SB. The trial will commence in Q1 2026 and be open to all former and current participants in the XanaMIA Phase 2b/3 trial.

The XanaCIDD Phase 2a depression trial was a double-blind, six-week proof-of-concept, placebo-controlled, parallel group design trial in 167 patients with moderate, treatment-resistant depression and a degree of baseline cognitive impairment. Participants were evenly randomized to receive Xanamem 10 mg once daily or placebo, in most cases in addition to their existing antidepressant therapy, and effects on cognition and depression were assessed. Trial results were reported in August 2024 and showed clinically and statistically significant benefits on depression symptoms with positive effects on the MADRS scale (a validated scale of depression symptom measurement) and the PGI-S (a valid patient reported assessment of depression severity). Cognition improved markedly and to a similar extent in both Xanamem and placebo groups.

About Xanamem (emestedastat)

Xanamem's novel mechanism of action is to control the level of cortisol in the brain through the inhibition of the cortisol synthesis enzyme, 11 β -HSD1, without affecting production of cortisol by the adrenal glands. Xanamem is a first-in-class, once-a-day pill designed to deliver high levels of cortisol control in the brain. To view Xanamem's two-minute Mechanism of Action video, [click here](#).

Chronically elevated cortisol is associated with progression in Alzheimer's Disease and excess cortisol is known to be toxic to brain cells. Cortisol itself is also associated with depressive symptoms and when targeted via other mechanisms has shown some promise in prior clinical trials. The recent XanaCIDD trial demonstrated clinically and sometimes statistically significant benefits on depressive symptoms.

The Company has studied 11 β -HSD1 inhibition by Xanamem in approximately 400 volunteers and patients in eight clinical trials. Xanamem has a promising safety profile and has demonstrated clinical activity in patients with depression, patients with biomarker-positive Alzheimer's disease and cognitively normal volunteers. High levels of target engagement in the brain with doses as low as 5 mg daily have been demonstrated in a human PET imaging study.

Xanamem is an investigational product and is not approved for use outside of a clinical trial by the FDA or by any global regulatory authority. Xanamem® is a trademark of Actinogen Medical.

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ACTINOGEN MEDICAL ENCOURAGES ALL CURRENT INVESTORS TO GO PAPERLESS BY REGISTERING THEIR DETAILS WITH THE DESIGNATED REGISTRY SERVICE PROVIDER, AUTOMIC GROUP.



Oral Xanamem[®] (emestedastat)

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

Bioshares Annual Conference Q&A presentation
Hobart August 7, 2025

Disclaimer



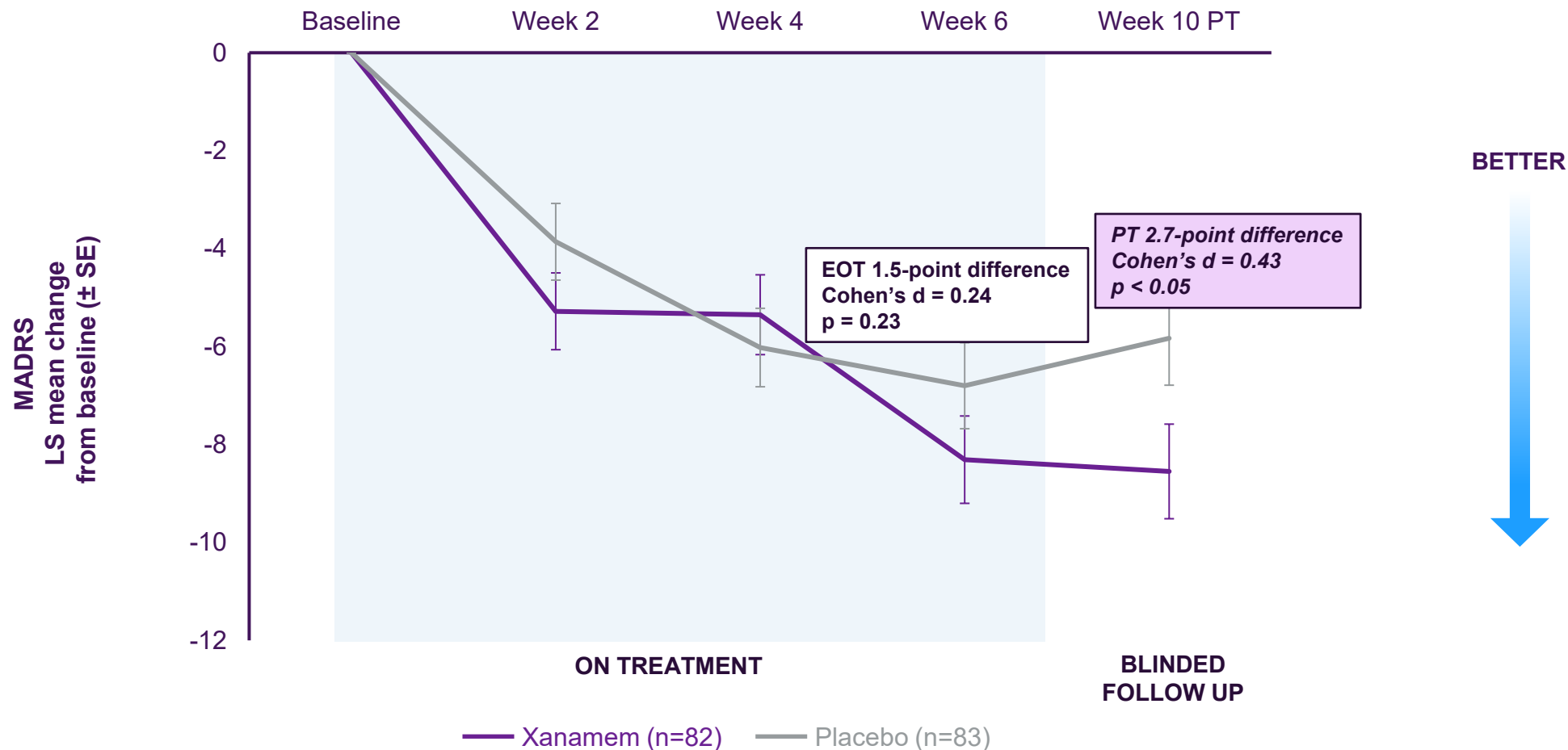
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What previous results give the company confidence in a positive phase 2b/3 trial outcome in Alzheimer's?

1. Clinical benefit on depression symptoms (n = 165)
2. Large clinical benefit in pilot Alzheimer's data (n = 34)
3. Human PET study showing high brain target engagement (n = 40)
4. Strong cortisol scientific rationale in Alzheimer's
5. Optimal trial design based on pilot data in pTau positive patients, CDR-SB endpoint

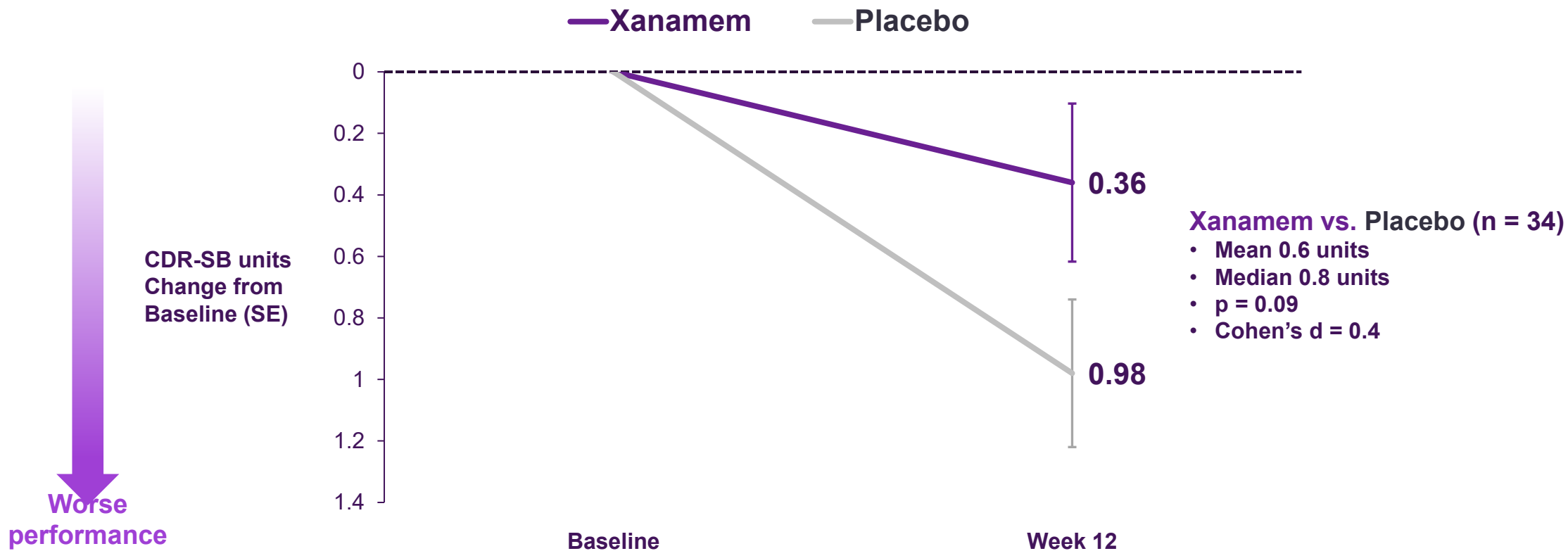
1. Xanamem clinical activity validation in depression

Clinically & statistically significant benefits seen in the XanaCIDD phase 2a trial



2. Large Xanamem benefit in high pTau181 patients

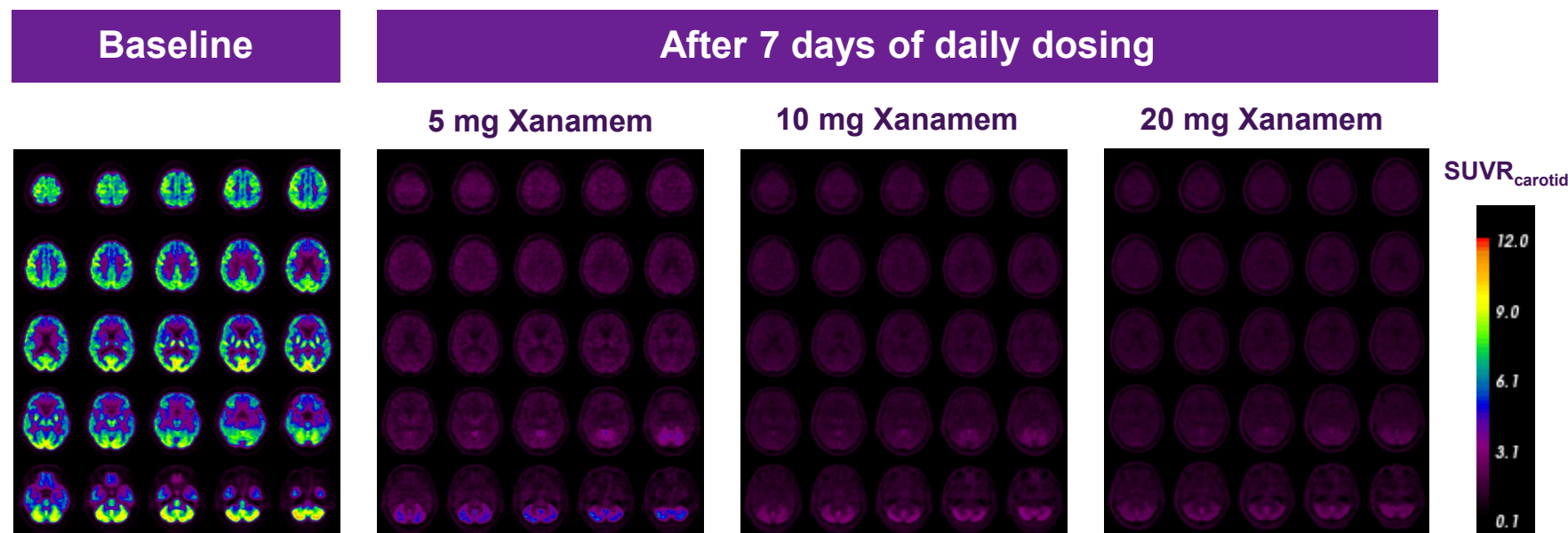
Phase 2a data suggests far greater benefit on CDR-SB than anti-amyloid antibodies



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

3. 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 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

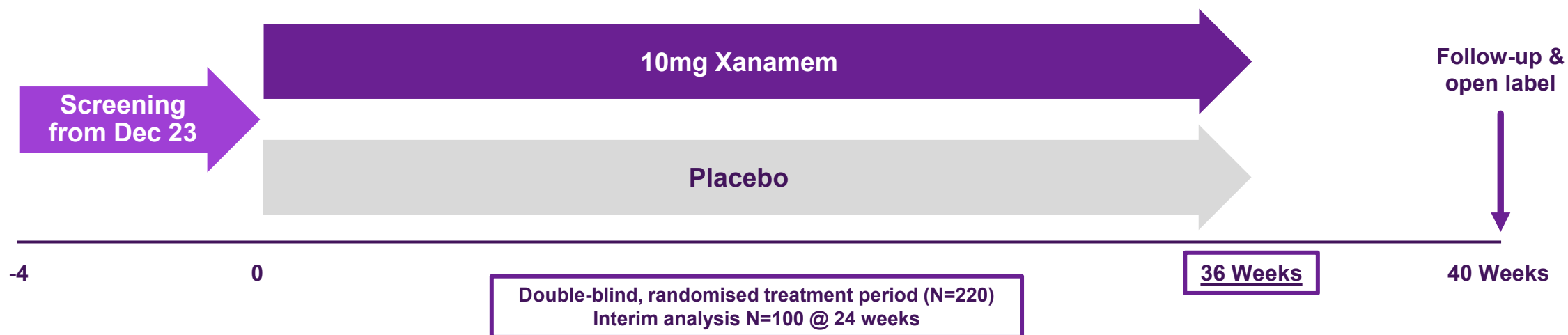
4. Unique brain-penetrating, regional cortisol action, that doesn't interfere with adrenal cortisol regulation



[Click here for animation video](#)

5. Optimal trial design & patient selection

Initial, interim results in Jan 2026, 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 (efficacy futility & safety)

What does XanaMIA trial success look like and what level of interest is there from potential partners?

What have you learned about commercialization so far?

1. Anti-amyloid infusions have a borderline risk-benefit profile
2. Xanamem is being developed with a better risk-benefit profile aimed at stabilizing the disease safely and more effectively
3. Desired Xanamem benefits include multiple aspects of cognition and life functioning – ideally to halt Alzheimer's decline completely
4. XanaMIA trial is already exciting clinical and commercial interest

1. Anti-amyloid drugs only modestly slow disease

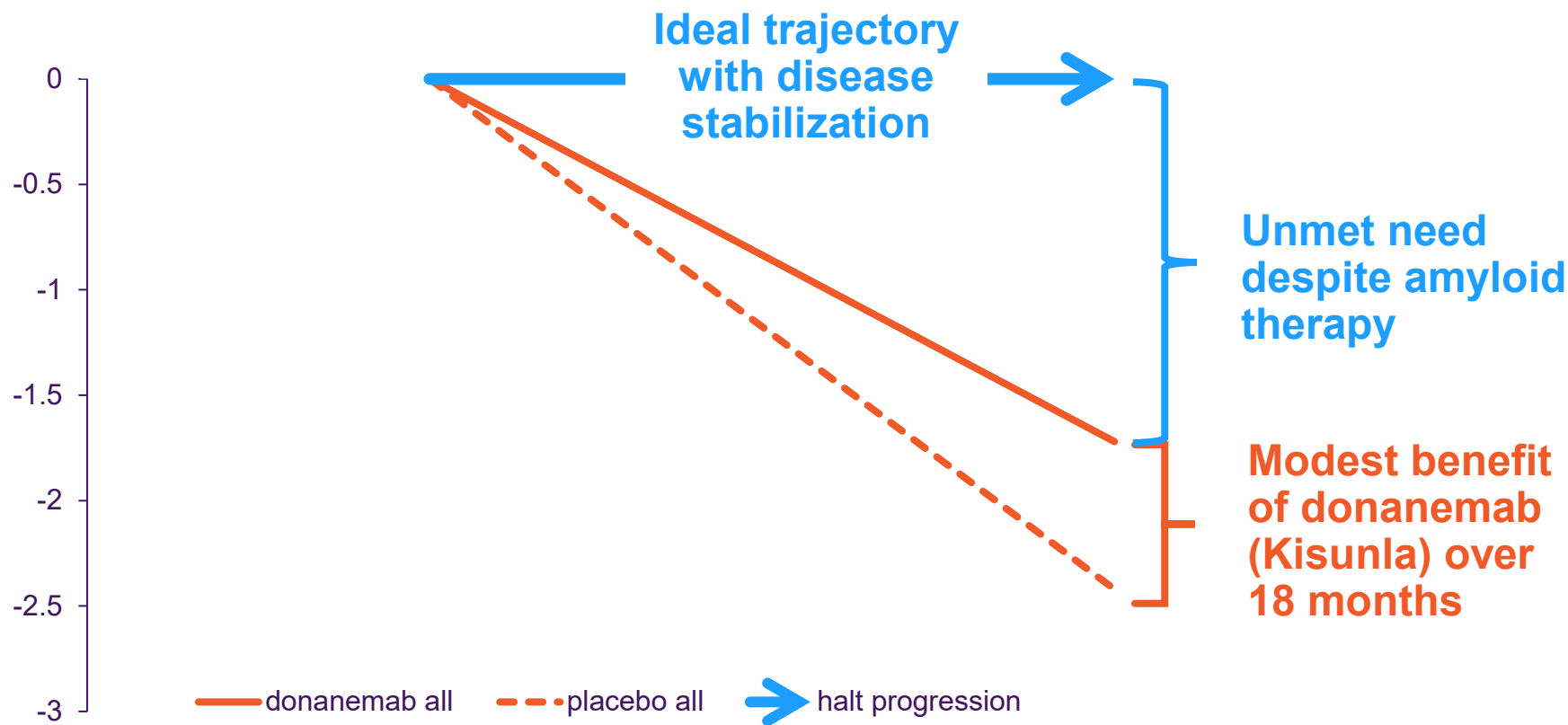
Ideally patients with AD would not worsen on treatment at all

For personal use only

Worsening of
CDR-SB
over 18 months



Worse
performance



Drugs targeting other mechanisms like Xanomem are needed

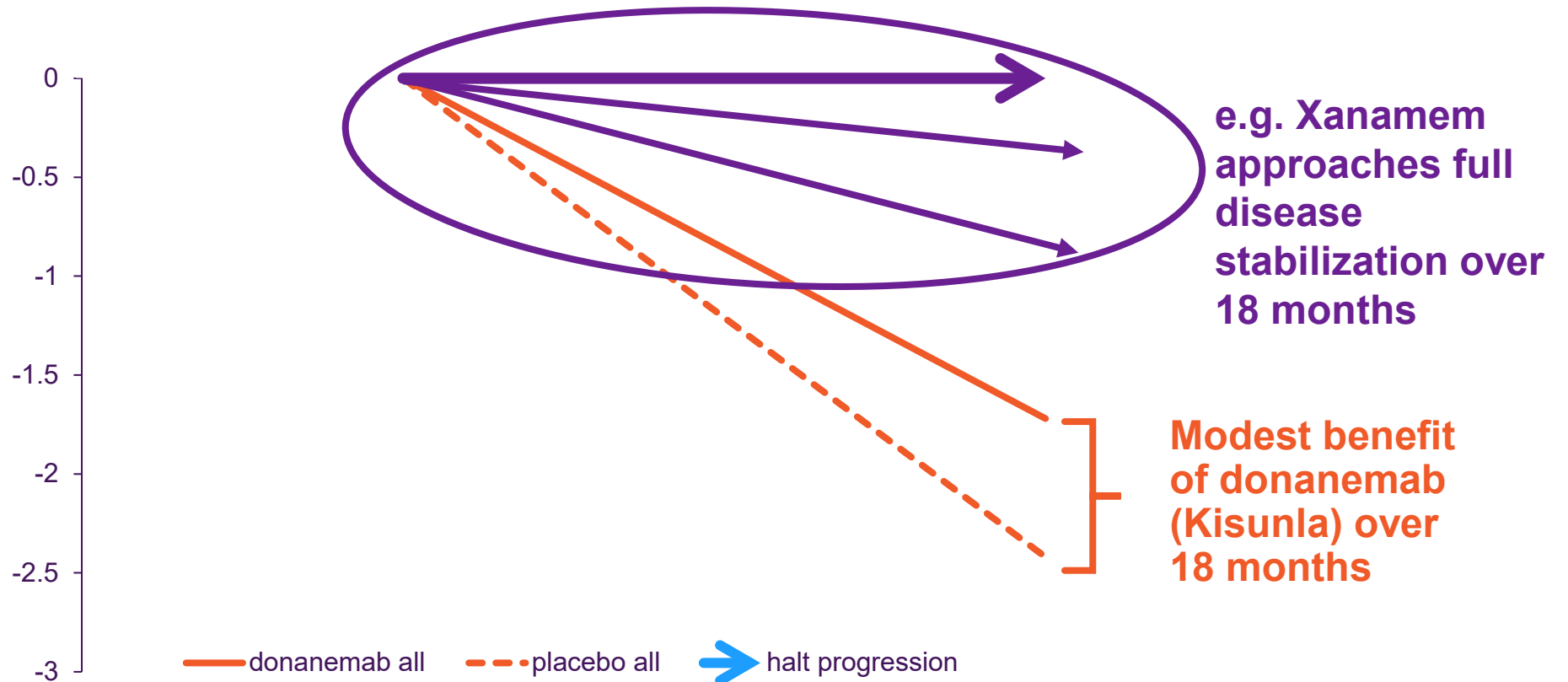
2. The hope is Xanamem is better than anti-amyloids on the primary endpoint of CDR-SB and other endpoints

For personal use only

Worsening of
CDR-SB
over 18 months



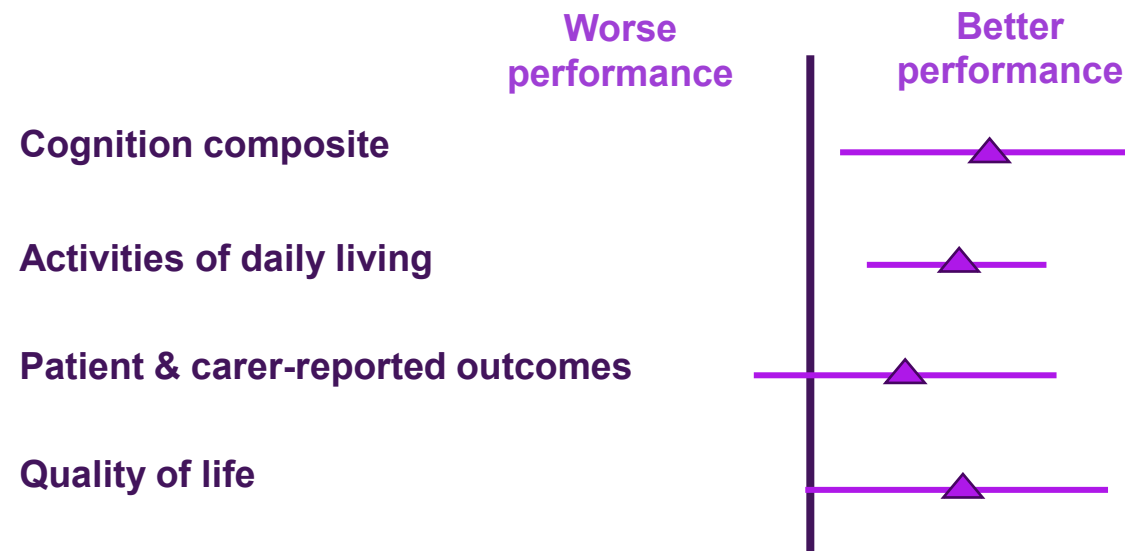
Worse
performance



If results are good, Xanamem could be many times more effective than other drugs

3. The hope is Xanamem shows consistent benefit in safety and key secondary efficacy endpoints

Safety¹: No serious adverse events related to Xanamem (n > 400)



EXAMPLE SECONDARY ENDPOINTS (MEAN, C.I.)

1. No serious adverse events related to Xanamem have been reported across all clinical trials to date; various other safety data are reported in peer reviewed publications (see <https://actinogen.com.au/xanamem/>)

4. And the trial excites commercial interest

We know:

- ✓ US Neurologists treating AD love the idea of a safe and effective, oral drug and indicate that uptake would be rapid in the first year
- ✓ Xanamem could easily move to first line therapy and displace many existing treatments
- ✓ Combinability with other small molecules and biologics a major plus

We hope:

- ✓ Multiple partners find the program compelling before interim and final results
- ✓ Final results are good enough to excite multiple, global partnership bids
- ✓ Final results are good enough for regulators to seriously consider accelerated approvals

Conclusion



Building momentum toward Alzheimer's results

Numerous value-add near-term milestones



- ***On-track with XanaMIA trial in patients with mild-moderate Alzheimer's disease***
 - ✓ Full enrolment of 220 participants in XanaMIA pivotal trial Q4 2025/Jan 2026
 - ✓ Interim analysis of XanaMIA safety and efficacy data January 2026
 - ✓ Final results Q4 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 beyond mid 2026***
- ***Commercial & partnership planning underway***
- ***Other trial, regulatory, publication and presentation milestones***

Appendix



Xanamem® is in advanced clinical development



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
- 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 names¹**
- 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 11 β -HSD1 inhibitors; USAN (United States Adopted Name)

Key references

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



11 β -HSD1 inhibition

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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
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Market & cost of treatment estimates

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- <https://www.cdc.gov/aging/aginginfo/alzheimers.htm#treated>
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- 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
- **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 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

Contacts

Michael Roberts

Investor Relations

P: +61 2 8964 7401

M: +61 423 866 231

E. michael.roberts@actinogen.com.au

Steven Gourlay

CEO & Managing Director

P: +61 2 8964 7401

E. steven.gourlay@actinogen.com.au