ASX ANNOUNCEMENT

Actinogen XanaMIA Phase 2b Alzheimer's disease (AD) trial amendment and Clinical Trials Science Forum 2023 presentation slides

Sydney, 24 May 2023. Actinogen Medical ASX: ACW ("ACW" or "the Company") announces specific amendments to the upcoming XanaMIA Phase 2b clinical trial in patients with AD and release of the presentation slides for this morning's ***Clinical Trials Science Forum*** webinar commencing at 11am AEST.

XanaMIA Phase 2b AD trial amendment

Having completed a number of key steps to develop a global syndicate of investigators and trial sites for the upcoming trial, Actinogen is making several improvements to the trial design while submitting updated regulatory documentation to the FDA and other regulators. FDA approval of the tablet formulation and protocol changes outlined below is expected early in H2 CY23 and the first patient is expected to be treated in the subsequent months. Results are expected in H2 CY25.

Principal components of the XanaMIA Phase 2b trial amendment are:

- The newly manufactured tablet formulation will be used in the Phase 2b trial
- Patients will be enrolled for a 36-week double-blind treatment period (24 weeks previously) to improve initial assessment of disease-modification effects
- An interim analysis will be included around the middle of trial enrolment
- Patients with "moderate AD" are now included to match the Phase 2a population more closely with elevated pTau levels in the blood analysed and previously reported to have a large Xanamem® effect
- Following extensive data analysis of prior trial data, the primary endpoint will be a cognitive composite of several tests, reflecting the strong cognitive effects of Xanamem in prior trials. The CDR-SB functional score remains a key secondary endpoint along with the assessment of activities of daily living.

Clinical Trials Science Forum webinar

Event registration: https://us02web.zoom.us/webinar/register/WN_5gdyj5DpRJ-XORhbhaflPQ

'Following the Science' is fundamental to all Actinogen's activities and a primary foundation for today's presentation titled *Alzheimer's disease: amyloid therapies are only part of the answer*. The presentation is designed to explain the science behind targeting amyloid in Alzheimer's disease and the opportunity for non-amyloid treatments such as Xanamem.

The quest to find drug therapies to cure AD has only just begun. The approval of two recent anti-amyloid antibody drugs in the USA and another with positive late-stage clinical results is a great beginning for AD

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therapy but this approach does not halt disease progression, requires intravenous infusion, and is associated with serious but rare side effects.

Actinogen is a leader in clinical AD research having the only oral therapy to have demonstrated benefits on cognition in multiple placebo-controlled trials.[1] We consider the 'weight of evidence' from academic work and our own trials has validated Xanamem's cortisol mechanism and are excited to now conduct our two larger Phase 2 controlled trials in AD and cognitive impairment in depression.

Today's plain English webinar has been designed for a broad audience, including those from non-technical backgrounds. The presentations will explore the following topics:

- An overview of the current 'state of play' on clinical AD research (CEO, Dr Steven Gourlay)
- An introduction to the Xanamem drug product and in particular its tablet formulation by Dr Fujun Li, Head of Manufacturing, that has been developed for the upcoming XanaMIA Phase 2b AD trial and all subsequent trials
- An insightful high-level analysis from ACW CMO and neurologist, Dr Dana Hilt on the role of treating AD with anti-amyloid antibody therapy and the significant opportunity remaining for alternative and complementary approaches

A copy of the full presentation is attached to this announcement. As soon as practicable after the conclusion of the webinar a full recording will be made available on the company's website: www.actinogen.com.au.

Dr Steven Gourlay, Actinogen CEO and MD, commented:

"Actinogen is at a remarkably important juncture in the world of drug development with its promising oral therapy Xanamem. While recent data on new amyloid antibody infusions give Alzheimer's patients hope, they do not halt disease progression, highlighting the continued and urgent need to find effective and safe non-amyloid therapies.

"Today's Clinical Trials Science Forum will cover three important topics: Xanamem manufacturing, understanding how to put amyloid and non-amyloid therapies into context, and the XanaMIA Phase 2b trial design update.

"We remain committed to our high-quality and timely Phase 2 trials in both Alzheimer's and cognitive impairment in depression and believe they will demonstrate and confirm the potent clinical benefit of Xanamem therapy."

ENDS

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Announcement authorised by the Board of Directors of Actinogen Medical

[1] Companies claiming efficacy based on uncontrolled data, biomarkers or imaging not included in this comparison

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.

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.

About Xanamem

Xanamem's novel mechanism of action is to block the production of cortisol inside cells through the inhibition of the 11β-HSD1 enzyme in the brain. Xanamem is designed to get into the brain after it is absorbed in the intestines upon swallowing.

Chronically elevated cortisol is associated with cognitive decline in Alzheimer's Disease and excess cortisol is known to be toxic to brain cells. Cognitive impairment is also a feature in Depression and many other diseases. Cortisol itself is also associated with depressive symptoms and when targeted via other mechanisms has shown some promise in prior clinical trials.

The Company has studied 11β-HSD1 inhibition by Xanamem in more than 300 volunteers and patients, so far finding a statistically significant improvement in working memory and attention, compared with placebo, in healthy, older volunteers in two consecutive trials and clinically significant improvements in functional and cognitive ability in patients with biomarker-positive mild AD. Previously, 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. A series of Phase 2 studies in multiple diseases is being conducted to further confirm and characterize Xanamem's therapeutic potential.

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

FOLLOWING THE SCIENCE

Clinical Trials Science Forum

Alzheimer's disease: amyloid therapies are only part of the answer

Xanamem® targets brain cortisol and is one of a few programs that has demonstrated clinical benefit

Wednesday 24 May 2023 | 11.00am AEST

Disclaimer

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Introduction and welcome

Michael Roberts BEc Hons, CPA, Ffin

Investor Relations

- 25+ years' business experience
- Senior IR & comms professional in top-50 listed companies
- Strategic corporate comms advisor

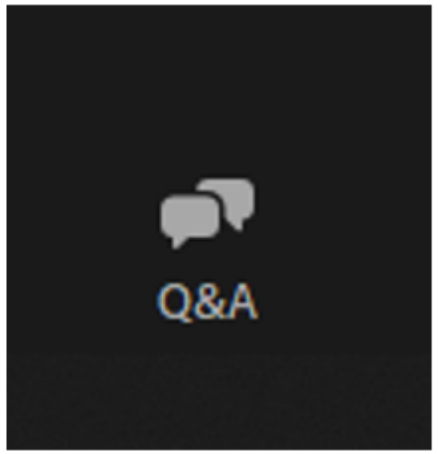
Dr Steven Gourlay MBBS FRACP PhD MBA

Chief Executive Officer

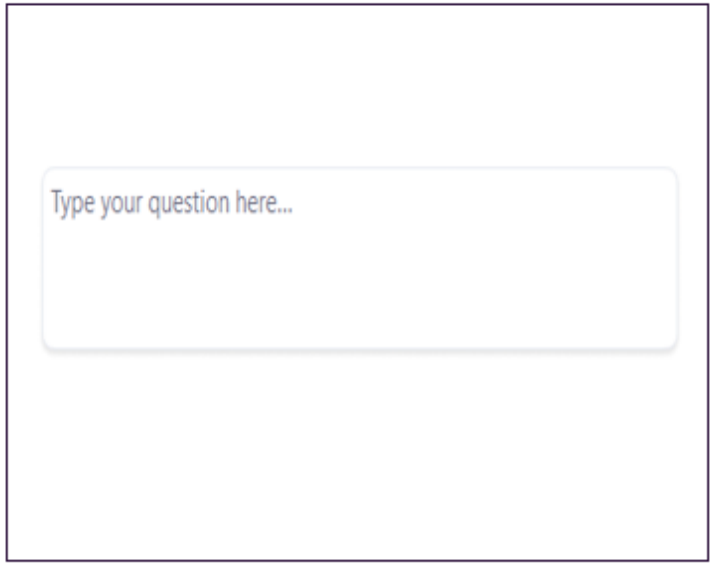
- Industry physician with more than 25 years experience
- Venture capital 8 years
- Recent successful startup exit with sale of Principia Biopharma to Sanofi for US$3.7 billion

Online Q&A

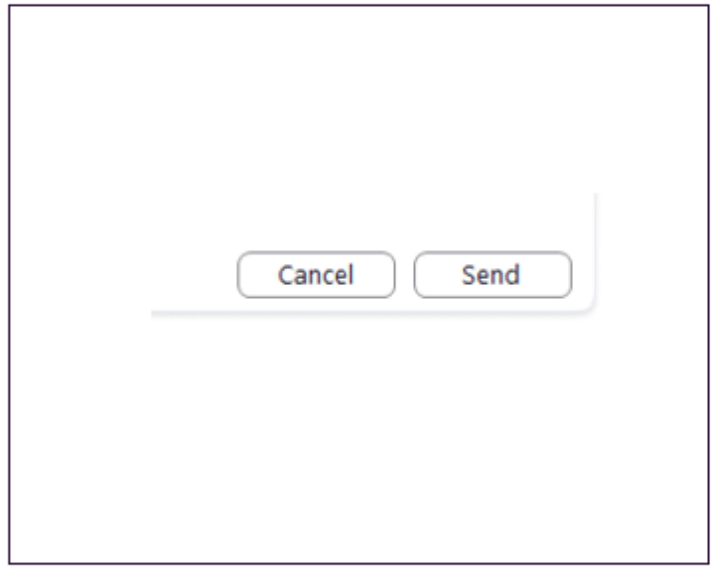
1. Click on the Q&A icon



2. Type your question in the new Q&A window



3. Hit enter on your keyboard to submit your message



To contact support:
Please call 1300 816 159 (within Australia) or +61 2 8072 1479 (outside of Australia)

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Agenda

Welcome		**Michael Roberts** Investor Relations
Introduction		**Dr Steven Gourlay** Chief Executive Officer
Introduction of Xanamem drug product		**Dr Fujun Li** Head of Manufacturing
The treatment of Alzheimer's disease: Amyloid is not the entire problem … so, it is only part of the solution		**Dr Dana Hilt** Chief Medical Officer
Summary		Moderator **Dr Steven Gourlay**
Questions and wrap-up		Moderator **Dr Steven Gourlay**

Introduction

Dr Steven Gourlay

Chief Executive Officer

Actinogen is a leader in clinical Alzheimer's research

Only amyloid antibody infusions and oral Xanamem have multiple, positive cognitive trial data[1]

Actinogen Oral Xanamem	• Safely targets **brain tissue cortisol** • 2 trials: improved attention & working memory • 1 trial: trends to reduce AD progression, improve cognition
Eisai-Biogen i.v. infusion of lecanemab	• Approved on ability to reduce **brain amyloid** • Causes brain swelling and bleeding • 2 trials reduced progression modestly • Will need to be combined with other therapies
Lilly i.v. infusion of donanemab	• Full approval expected ~8 months, reduces **brain amyloid** • Causes brain swelling and bleeding • 2 trials reduced progression modestly • Will need to be combined with other therapies

[1] Companies claiming efficacy based on uncontrolled data, biomarkers or imaging not included in this comparison

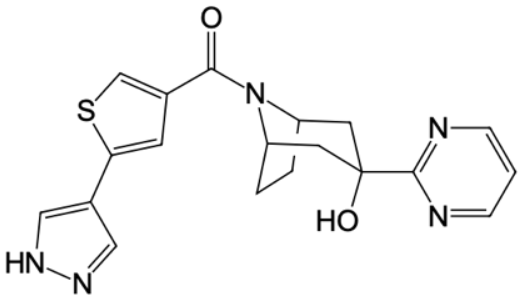
Introduction of Xanamem drug product

Dr Fujun Li, Ph.D.

Head of Manufacturing

Xanamem is promising small molecule pharmaceutic

- Small molecule, once-a-day pill or capsule for oral administration
- High solubility and high membrane permeability - well absorbed through gastrointestinal tract (intestines)
- Crystalline material and stable at room temperature



$C_{19}H_{19}N_5O_2S$
Molecular Wt: 381.45

Drug in tablet → Dissolution in stomach → **Drug in solution** → Absorption in intestines → **Drug in blood and brain**

Xanamem has favourable characteristics for an oral drug

Xanamem capsules and now (commercial) tablets

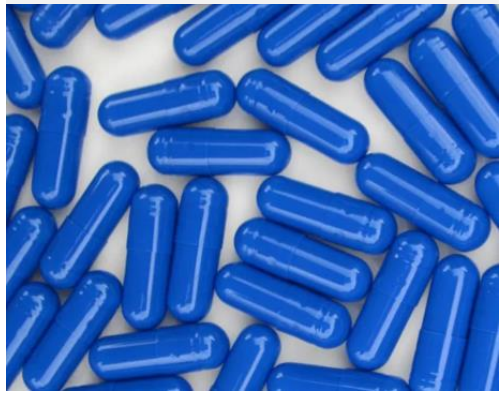
Xanamem was initially formulated in gelatin capsules that were used in early Phase 1 and Phase 2 clinical studies

- Capsules contained simple blend of drug substance and inactive ingredients
- Capsule formulation offered flexibility to accommodate changes of dose in early clinical trials

As Actinogen prepares for and implements late phase clinical studies, a tablet formulation has been developed that provides advantages over capsules

- Typically, has longer shelf-life
- Variety of shapes, sizes, colors and identifiers
- Manufacturing is easy to scale-up and more cost effective

Capsule formulation



Tablet formulation

Advantages of Xanamem tablets over injectables like anti-amyloid antibodies

- Xanamem tablets are manufactured using a conventional tablet manufacturing process according to Good Manufacturing Practice (GMP) standards
- Small pill and easy to swallow for elderly patients
- Once a day oral dosing at home unlike intravenous injections or infusions needing clinic visits
- No infusion-related adverse effects
- No requirement for frequent brain scans to monitor for brain swelling
- Storage at room temperature - no need for refrigeration

Xanamem oral tablet is convenient to take, cost effective to manufacture and designed to minimize patient burden

The treatment of Alzheimer's disease (AD)

Amyloid is not the entire problem …
so, it is only part of the solution

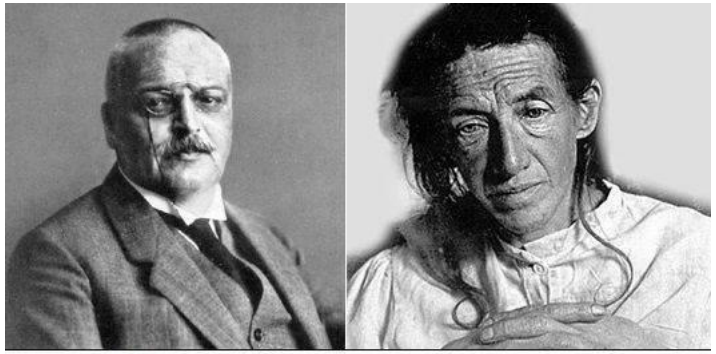
Dr Dana C Hilt MD

Chief Medical Officer

Alois Alzheimer and amyloid

The historical background of amyloid in AD

In November 1901, Dr. Alois Alzheimer, a German neurologist, made his first examination of a 51-year-old woman named Auguste Deter who was experiencing problems with memory and language as well as various psychological problems such as disorientation and hallucinations. These symptoms matched the definition of what was then called ‘dementia’, but she was very young to be displaying them, so he diagnosed her as having “presenile dementia”. This came to be known as Alzheimer’s disease.

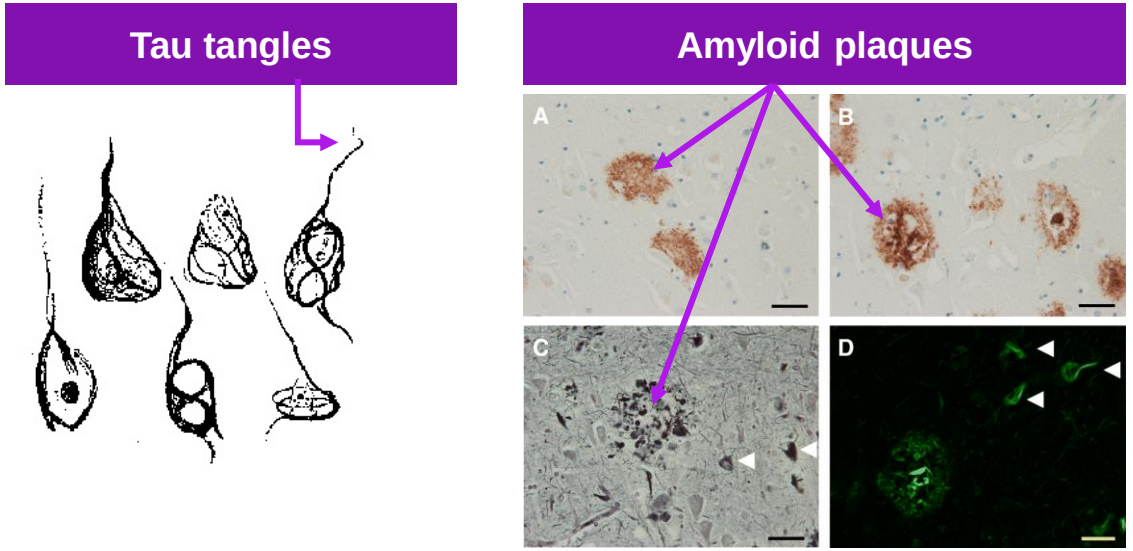


Alois Alzheimer Auguste Deter

The pathological hallmarks of AD include:

- amyloid plaques - deposits of extracellular amyloid; and
- neurofibrillary tangles - intracellular deposits of tau protein

The plaques and tangles of AD



Amyloid as the cause of AD: A closer look

Reducing amyloid to treat AD

- All genetic cases of AD are associated with some defect in amyloid protein processing: mutation in the amyloid gene or mutation in the enzymes processing amyloid
- All cases of AD have the signature of amyloid and tau protein deposition in the brain
- Overexpression of amyloid in animal models of AD can lead to nervous system pathology and behavioral abnormalities

Amyloid can not be the entire solution

- Mutations in amyloid and enzymes processing amyloid account for a very small fraction <2% of all AD patients[1]
- Amyloid (or Tau) mutations are **not** the cause of the vast majority of AD cases
- AD greatest genetic risk - ApoE4 gene - encodes a protein linked to lipid/fat metabolism **not** amyloid metabolism
- The association with amyloid and tau deposition in the brain and AD clinical stage is low[2]
- ***~33% of older cognitively normal people*** have amyloid in their brains at the same level as in AD[3]

1. Haas etal Plos Biol 2022
2. Tideman etal Neurology 2022
3. Jansen etal JAMA Neurol 2022

Amyloid as the cause of AD: A closer look

Reducing amyloid to treat AD

- The amyloid gene is contained on chromosome 21
- Trisomy (3 copies) of chromosome 21 causes Down Syndrome which is characterized by early cognitive impairment (early AD) and AD pathology
- A mutation in the amyloid gene decreasing production of amyloid has been detected and individuals with decreased amyloid production are partially protected against AD

Amyloid can not be the entire solution

- >15 anti-amyloid therapies have been taken into large clinical trials
  - Some of these drugs have demonstrated significant clearing of amyloid from the brain - ***but no benefit***
  - Stated at the recent AD Congress: 'To date we have ***'Only 1½ drugs which have an effect'*** [1]: lecanemab and aducanumab'
- **Other drugs that have cleared amyloid to approximately the same degree… have had no effect… some have made the condition worse**
- **The treatment effects of the most successful anti-amyloid antibodies (lecanemab and donanemab) are modest**

For every complex question, there is a simple answer… and it is wrong[2]

1. AD/PD Congress, April 2023
2. Variously attributed the either HL Mencken or Mark Twain

The effects of clearing amyloid in AD

Correlations between amyloid clearance and clinical benefit in anti-amyloid antibody studies are modest

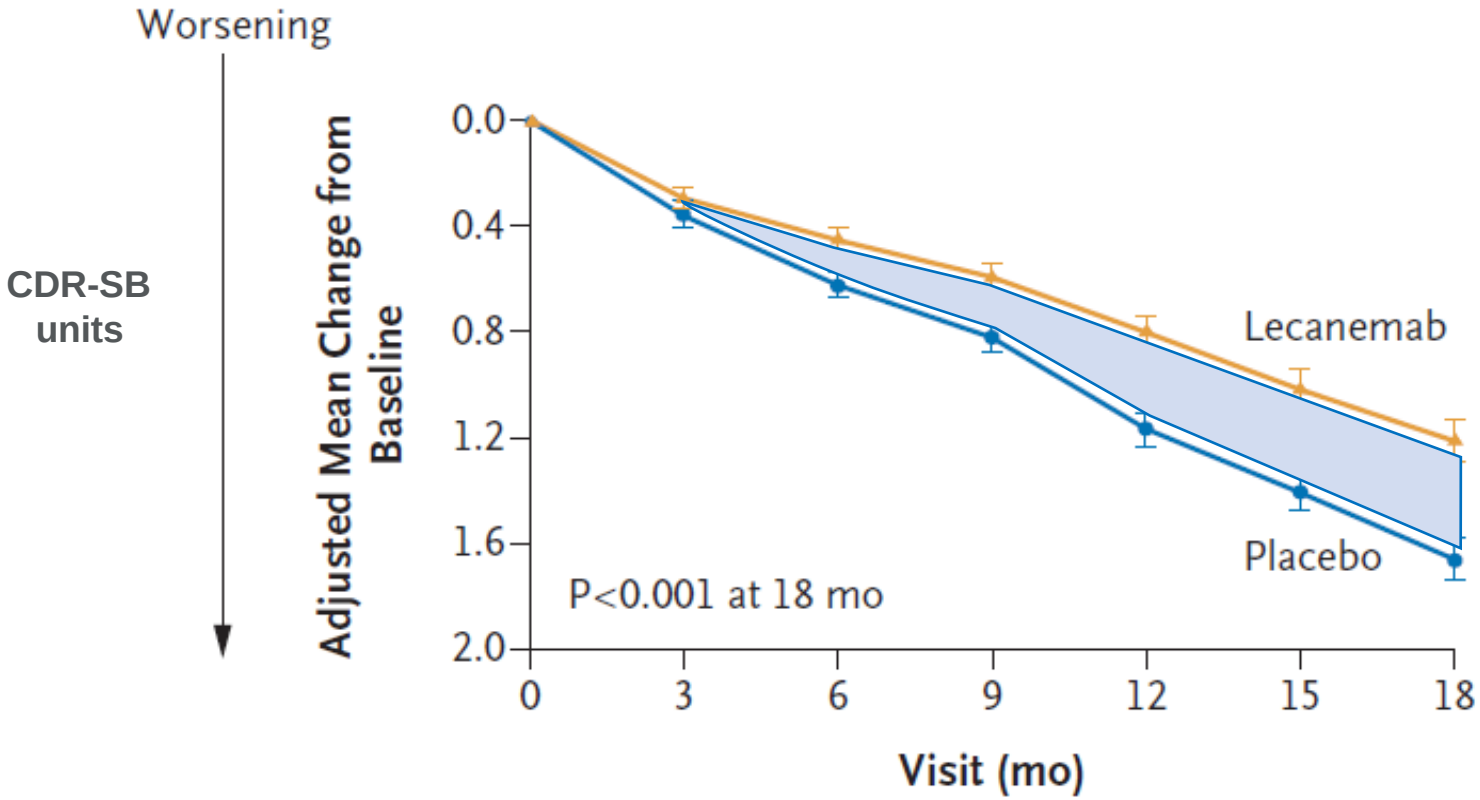
- A number antibodies have shown clearance of amyloid but few have shown benefit[1]
- In some antibody studies (e.g. aducanumab and donanemab) brain volume actually shrinks on treatment
- Weak correlation between amyloid removal and clinical benefit (CDR-SB)[2]. Recently, more robust clearance does seem to correlate with clinical benefit for lecanemab and donanemab
- Lecanemab showed an ~0.45 point CDR-SB change in the Phase 3 study over 18 months – well below the Minimal Clinically Important Difference (MCID) for the CDR-SB in AD of ~1.5 points[3]

The clinical benefits of anti-amyloid antibody infusions are modest

1. Haas etal Plos Biology 2022
2. Clinical Dementia Rating Scale- Sum of Boxes
3. Andrews etal Alzheimer's Dementia 2022

Anti-amyloid approach leaves significant unmet medical need

Lecanemab[1] slows progression modestly during the first 12 months

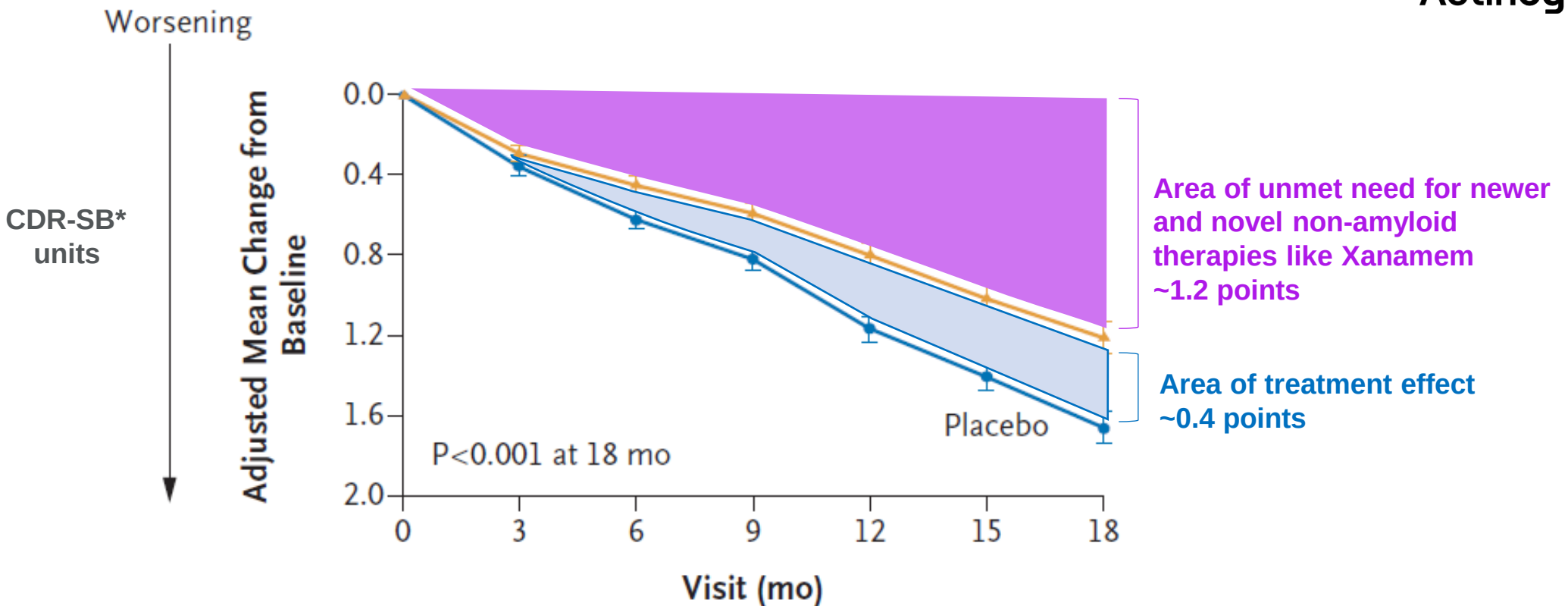


Blue area = treatment effect

No. of Participants							
Lecanemab	859	824	798	779	765	738	714
Placebo	875	849	828	813	779	767	757

1. Lecanemab is an anti-amyloid antibody given as an intravenous infusion every 2 weeks and largely clears brain of amyloid largely by 12 months, accelerated approval given by the US FDA based the ability of the drug to clear amyloid, full approval pending. Used a functional AD scale called the Clinical Dementia Rating Scale Sum of Boxes (CDR-SB) with an effect size reported of 0.4-0.45 points at 18 months; Leqembi USPI & van Dyck et al. 2022; DOI: 10.1056/NEJMoa2212948

Anti-amyloid approach leaves significant unmet medical need



Drugs targeting other mechanisms like Xanamem will be required to fill this gap

* Lecanemab is an anti-amyloid antibody given as an intravenous infusion every 2 weeks and largely clears brain of amyloid by 12 months, accelerated approval given by the US FDA based the ability of the drug to clear amyloid, full approval pending. Used (CDR-SB) with an effect size reported of 0.4-0.45 points at 18 months; Leqembi USPI & van Dyck et al. 2022; DOI: 10.1056/NEJMoa2212948 n=1795)

The road forward to better treatment

- **Reducing amyloid to treat AD can provide modest clinical benefit (lecanemab and donanemab antibodies)**
- **The benefit seen for lecanemab and donanemab is likely to be the maximal effect of anti-amyloid therapy as they clear amyloid rapidly and to a great extent**
- **The effects may wane after ~12 months**
- **Intravenous infusions required, side effects like brain swelling and bleeding may be fatal, require monitoring including multiple MRI brain scans**

The challenge	The goal
Identification of additional therapies to add to the presently approved treatments	Enhance function and clinical benefit for patients

Virtually all chronic illnesses require multiple therapies to make a significant impact on the disease

AD is no exception: Better or combination therapies will be the key to effective treatment

Xanamem: Oral, low dose, once-a-day treatment with a unique non-amyloid/tau mechanism

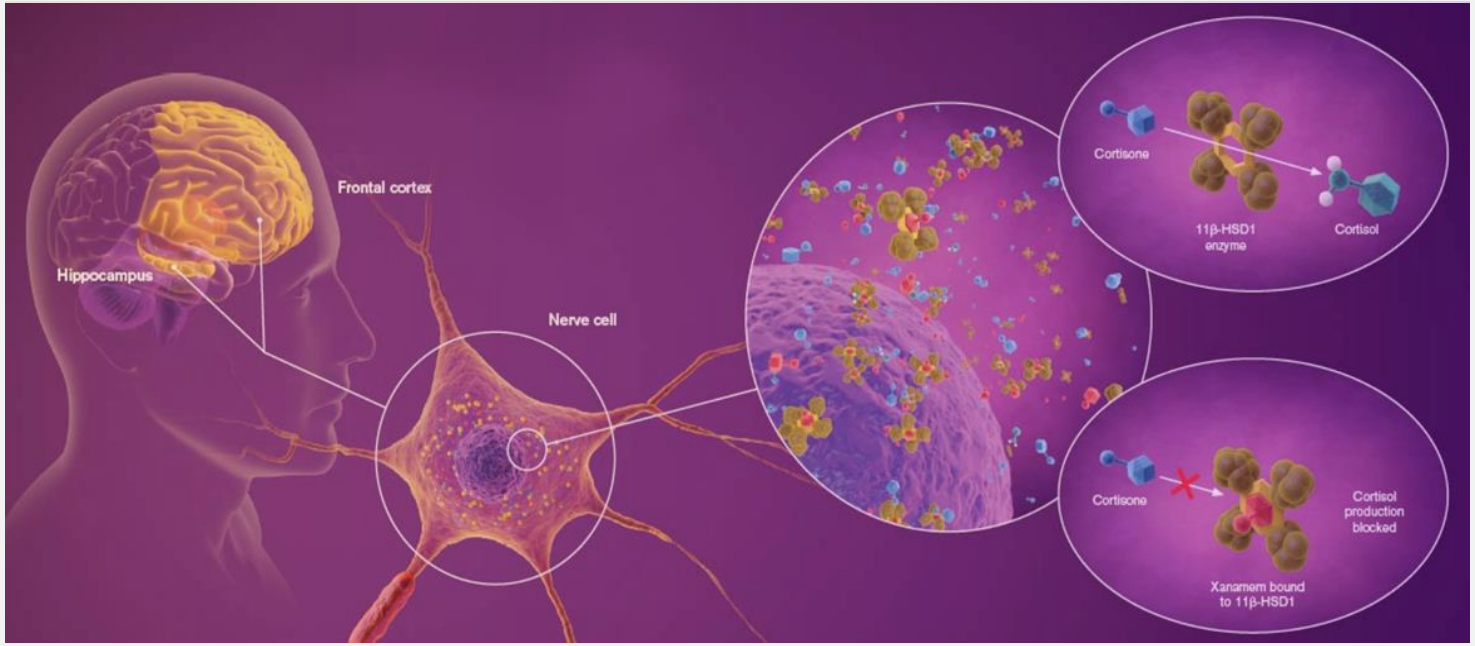
Only known **brain penetrant** 11β-HSD1 small molecule enzyme inhibitor

Reduces cortisol in brain - modulating signalling pathways and potentially underlying disease processes[1,2]

11β-HSD1 is preferentially expressed in brain and liver but minimally expressed in endocrine tissues

Xanamem has potential to:

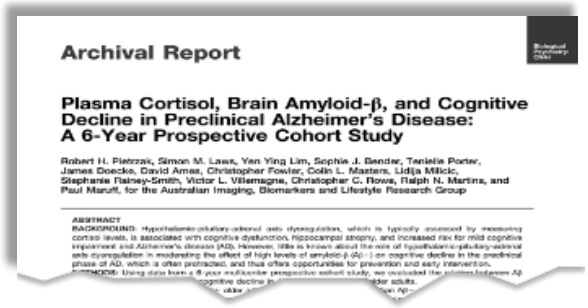
- **Enhance cognition**
- **Slow progression or produce durable delay in symptoms in AD**
- **Be anti-depressant/procognitive in depression**



1. Xanamem® is a CNS (Central Nervous System) penetrant small molecule based on human PET scan evidence and cerebrospinal fluid (CSF) measurements
2. Sooy et al. 2015 showing effects on amyloid plaque reduction in an aged mouse model after 28 days associated with increases in insulin degrading enzyme; Popoli et al. 2011 microglial cell modulation in rats, effects on glutamate, cannabinoid and other signalling pathways

Elevated cortisol leads to human brain dysfunction

- Multiple studies support the association between elevated cortisol and AD development and progression[1-5]
- Cognitive impairment in patients with neuroendocrine dysfunction[6-9]
- Compelling evidence provided by the Australian Imaging, Biomarker & Lifestyle Study of Ageing (AIBL) study (2017)[5]
  - Higher plasma cortisol leads to a much greater risk of developing AD
  - Accelerated effect of Aβ+ on decline in global cognition, episodic memory, and attention
- Individuals with the APOE-ε4 allele have higher CSF cortisol[8]
- Higher CSF cortisol levels in AD patients are associated with more rapid clinical worsening and cognitive impairment[10,11]
- High cortisol is one of the predictors of probable AD after age 75[12]

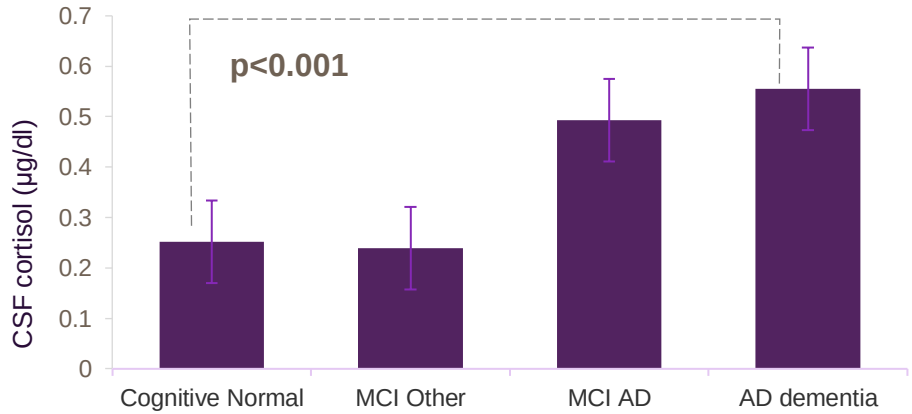

Archival Report

Plasma Cortisol, Brain Amyloid-β, and Cognitive Decline in Preclinical Alzheimer's Disease: A 6-Year Prospective Cohort Study

aibl

The Australian Imaging, Biomarkers and Lifestyle Flagship Study of Ageing

MEAN CSF CORTISOL LEVELS



Popp et al, 2015

[1] Geerlings et al., 2015, Neurology 85: 1-8; [2] Lehallier et al., 2016, JAMA Neurology 73(2), 203-212; [3] Popp et al., 2015, Neurobiol. Aging 36:601–607; [4] Ennis et al., 2017, Neurology 88(4):371-378; [5] Pietrzak et al., 2017, Biol Psychiatry: Cognitive Neuroscience and Neuroimagery, 2:45-52; [6] Lupien et al., 2009, Nat Rev Neurosci 10:434–445; [7] Starkman et al., 1999, Biol Psychiatry 46: 1595–1602; [8] Lupien et al., 1998, Nat Neurosci 1:69–73; [9] MacLullich et al., 2005, Psychoneuroendocrinology 30:505–515; ; [10] Cernansky et al., 2006, Am J Psychiatry 163:2164-2169; [11] Kornhuber & Jensen, 2015, Neurobiol Aging 36:601-607; [12] Hinterberger et al., J Am Ger Soc 2013 61(4):648-651;

Xanamem clinical trial data

Two separate normal volunteer studies have shown positive procognitive effects of Xanamem: attention, working memory, and executive function[1]

Oral Xanamem 5 and 10 mg/d have excellent CNS penetration and target occupancy by PET scanning while being safe and well tolerated

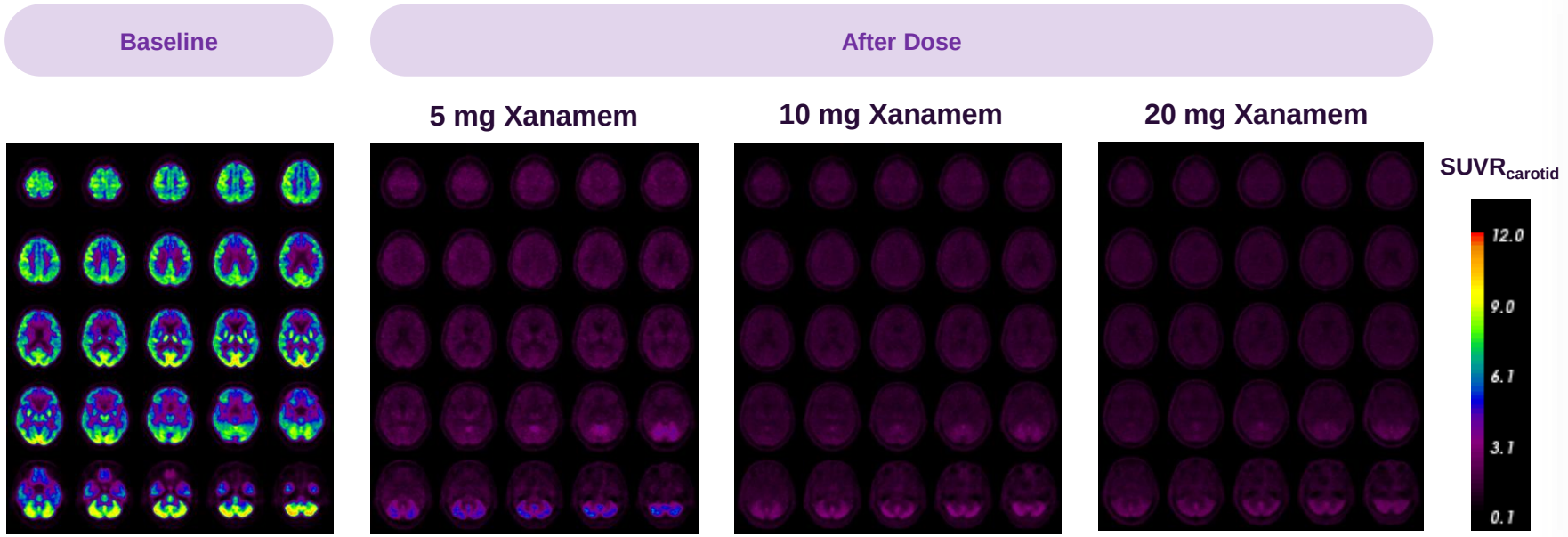
New analyses of the Phase 2 XanADu AD study show procognitive and clinical benefits in biomarker-positive patients[2]

These data taken together support further studies of oral Xanamem as a procognitive and potential disease-course altering drug in AD and depression

1. Actinogen data on file
2. ref AD/PD Congress

PET data shows full target engagement in the brain in the dose range of 5-30 mg (5-20 mg shown)

Previous drug candidates to this target have not achieved adequate brain concentrations as they were poorly CNS penetrant



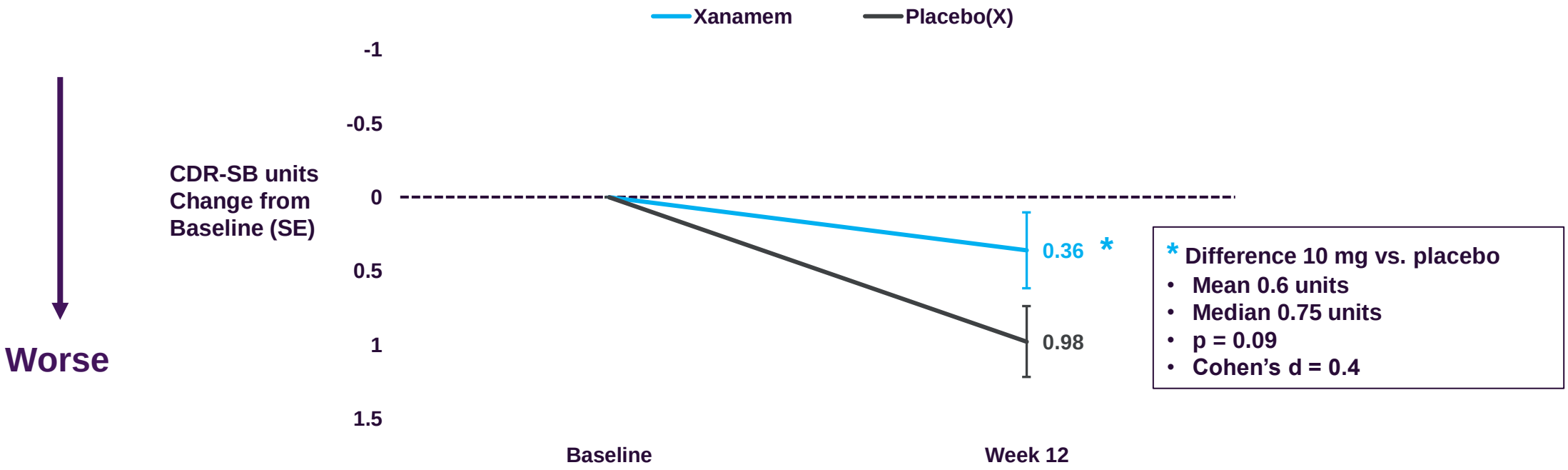
PET data demonstrates that Xanamem extensively binds to the 11β-HSD1 enzyme throughout the brain, with high post-treatment effects (absence of colour) after 7 days at all doses, slightly less at a 5 mg dose.

5 and 10 mg show excellent clinical tolerability and safety and minimal systemic endocrine effects.

Note: Study population consisted of ~50% healthy older subjects who were cognitively normal and ~50% with Alzheimer's disease. Subjects dosed for seven days. Baseline: Mean of baseline scans of patients in that dose group; After dose: Mean of post-dosing (7 days) scans in that dose group.

Xanamem prevents decline in CDR-SB (global functional score) in biomarker-positive AD patients[1]

Using pre-specified protocol, statistical analysis plan and blinded biomarker analysis



Oral Xanamem slowed AD progression over 12 weeks

1. Patients with elevated plasma pTau181 (> median of 6.74 pg/mL); CDR-SB Clinical Dementia Rating scale – Sum of Boxes (18-point score)

Xanamem provides clinical and procognitive benefit in mild/moderate AD patients

Response analysis in biomarker-positive[1] XanADu patients (patients more likely to progress)

Twice as many AD patients in the Xanamem group had stable or improved disease compared with placebo[2]

56% of AD patients treated with Xanamem were stable or improved vs. 28% in placebo

Xanamem also showed benefit on tests of cognitive ability as well as CDR-SB

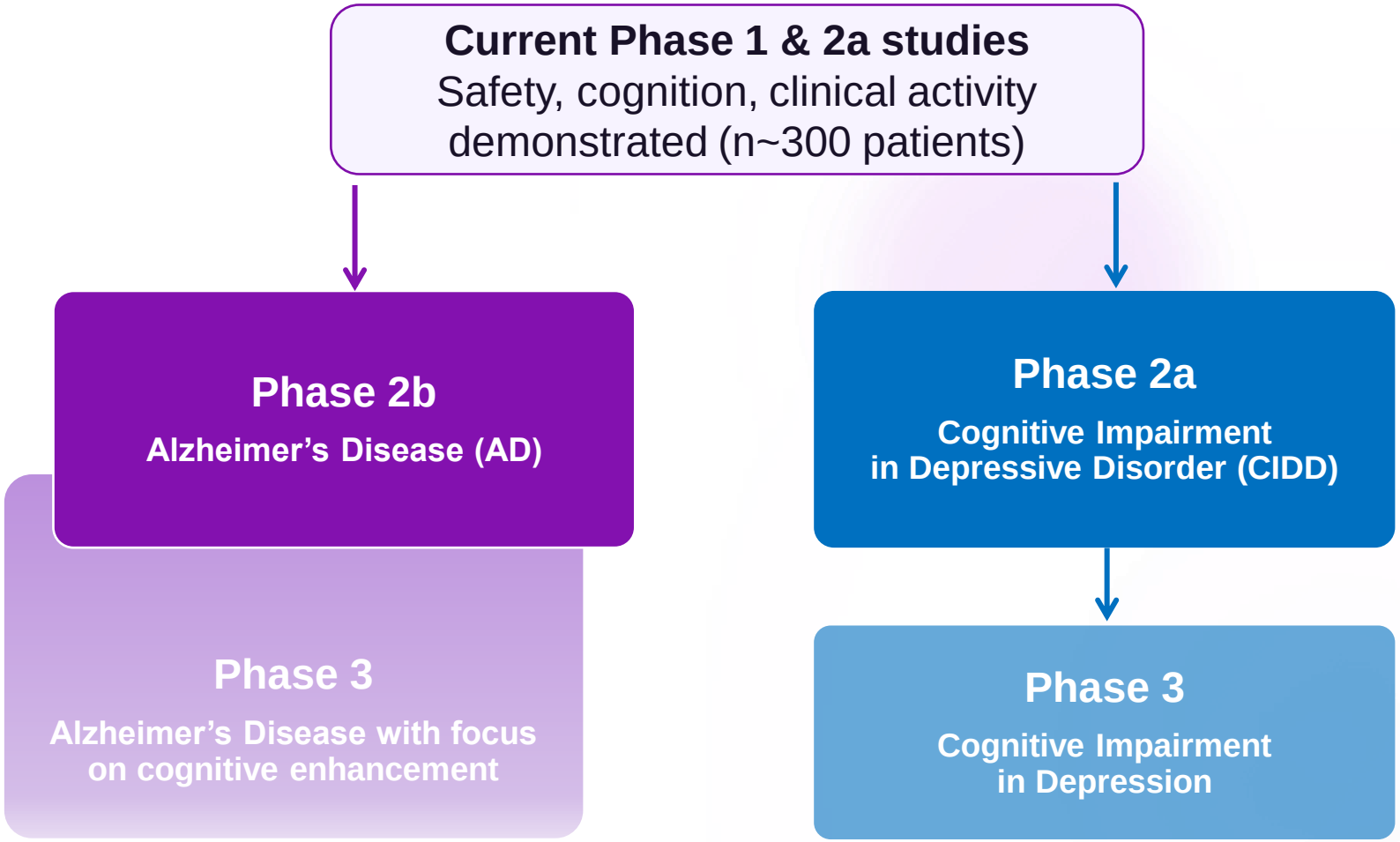
Xanamem preserved cognition and protected the majority of AD patients in the study from clinical progression

1. Pre-specified level of pTau181 above the median in plasma at baseline
2. Where CDR-SB decreased or was unchanged - Xanamem 9 of 16 (56%) vs. Placebo 5 of 18 (28%)

Xanamem Phase 2 & 3 programs

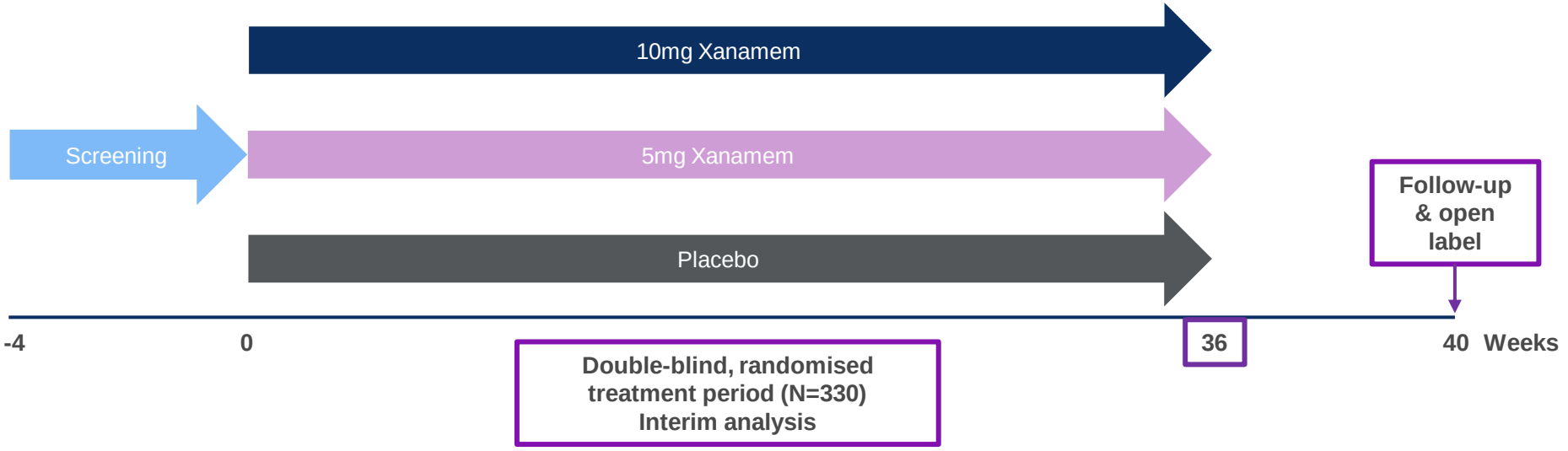
Actinogen

Building on three independent Phase 1 and 2 studies showing safety and procognitive activity



XanaMIA Phase 2b trial in Alzheimer's Disease

Matching patients and endpoints used in the positive Phase 2a analysis

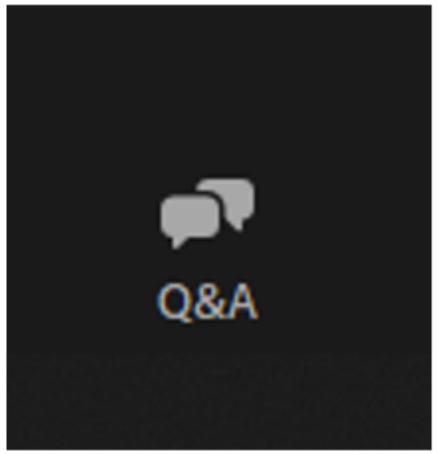


Key inclusion/exclusion criteria	Primary Endpoints	Key Secondary Endpoints	Key Implementation Features
• Clinical diagnosis of **mild to moderate dementia** due to AD (NIA-AA, MMSE 18-26) • Blood **p-tau181** to confirm progressive AD diagnosis • Cognitive impairment test	• **Cognitive Test Battery (cognitive measures)**	• **CDR-SB (functional measure)** • **Amsterdam Activity of Daily Living** scale • Executive Function & Episodic Memory Function Composites • Care Giver questionnaire / Patient Global Improvement	• **Global** trial sites including US, AU, Asia, EU and other • **Actinogen "hands-on" operational model** • **First patient enrollment with new tablets, updated protocol & regulatory dossiers (H2 CY23)**

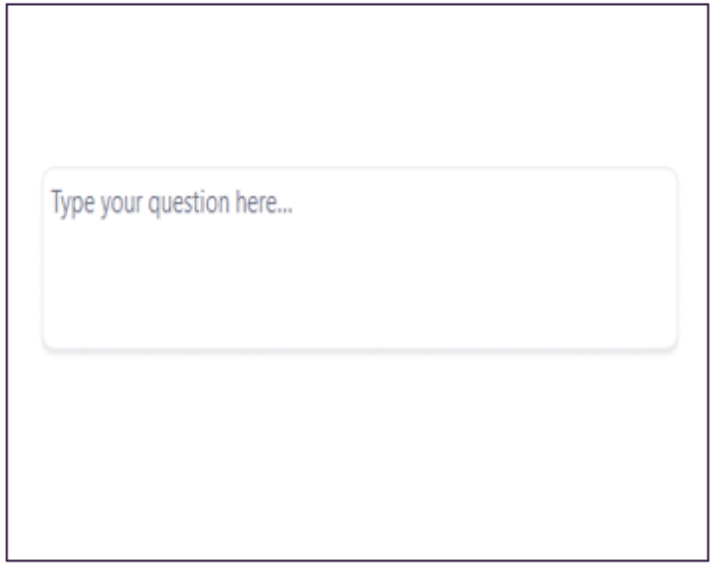
NIA-AA=National Institute of Aging - Alzheimer's Association; CDR-SB Clinical Dementia Rating Scale – Sum of Boxes

Online Q&A

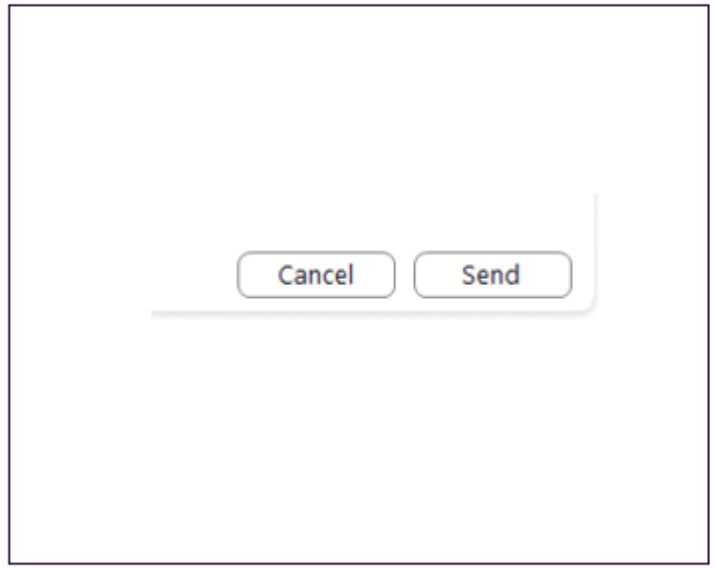
1. Click on the Q&A icon



2. Type your question in the new Q&A window



3. Hit enter on your keyboard to submit your message



To contact support:
Please call 1300 816 159 (within Australia) or +61 2 8072 1479 (outside of Australia)

AUTOMIC GROUP

Questions

Thank you

If you have any questions following the event please contact:

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Appendix

Selected glossary 1

11β-HSD1 11 beta HydroxySteroid Dehydrogenase-1 enzyme

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

ADAS-Cog Alzheimer's Disease Assessment Score - Cognition

APOE-ε4 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

Filament A Filament protein believed to relate to amyloid toxicity

GFAP Glial Fibrilliary Acidic Protein – a marker of microglial cell activation in the brain

GMP Good Manufacturing Practice

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

Tau – a brain protein

Ttau – total tau levels including both phosphorylated and non-phosphorylated tau