ASX ANNOUNCEMENT

Actinogen presents at ADPD™ Alzheimer's and Parkinson's diseases conference

Sydney, 30 March 2023. Actinogen Medical ASX: ACW ("ACW" or "the Company") is pleased to announce that its Chief Medical Officer, Dr Dana Hilt MD is making an oral presentation to the ***International Conference on Alzheimer's and Parkinson's Diseases and related neurological disorders*** (ADPD™ 2023) tomorrow in Gothenburg, Sweden. Dr Hilt's presentation slides are attached to this announcement.

Dr Hilt was invited to present Actinogen's novel Phase 2a data as an oral presentation in a symposium session entitled *AD Drug Development Clinical Trials.* The presentation details the unique longitudinal dataset demonstrating 1) the utility of using the plasma biomarker pTau181 for selection of patients with a more progressive form of mild Alzheimer's disease (AD) in clinical trials and 2) an encouraging beneficial effect of Xanamem on clinical endpoints of function (CDR-SB[1]) and cognition (NTB[1]) in these patients.

Dr Steven Gourlay, Actinogen's CEO and MD, said:

"Actinogen is excited to present its novel Phase 2a dataset that is one of the first to show that the blood pTau biomarker is a highly effective method for selection of patients with a progressive form of mild Alzheimer's disease. As a simulation of the upcoming 330-patient Phase 2b XanaMIA trial the data give us confidence in our study design, endpoints and patient selection criteria."

ENDS

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

Announcement authorised by the Board of Directors of Actinogen Medical

1. CDR-SB (Clinical Dementia Rating Scale – Sum of Boxes); NTB (Neurologic Test Battery of cognition)

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.

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 its capsule.

Chronically elevated cortisol is associated with cognitive decline in Alzheimer's Disease, and Xanamem has shown the ability to enhance cognition in healthy, older volunteers. 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 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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This announcement and attachments may contain certain "forward-looking statements" that are not historical facts; are based on subjective estimates, assumptions and qualifications; and relate to circumstances and events that have not taken place and may not take place. Such forward looking statements should be considered "at-risk statements" - not to be relied upon as they are subject to known and unknown risks, uncertainties and other factors (such as significant business, economic and competitive uncertainties / contingencies and regulatory and clinical development risks, future outcomes and uncertainties) that may lead to actual results being materially different from any forward looking statement or the performance expressed or implied by such forward looking statements. You are cautioned not to place undue reliance on these forward-looking statements that speak only as of the date hereof. Actinogen Medical does not undertake any obligation to revise such statements to reflect events or any change in circumstances arising after the date hereof, or to reflect the occurrence of or non-occurrence of any future events. Past performance is not a reliable indicator of future performance. Actinogen Medical does not make any guarantee, representation or warranty as to the likelihood of achievement or reasonableness of any forward-looking statements and there can be no assurance or guarantee that any forward-looking statements will be realised.

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Actinogen

Xanamem® CNS Activity in Alzheimer's disease

Utility of plasma p-Tau181 as a predictor of progression and Xanamem clinical effect in mild Alzheimer's disease: XanADu Phase 2a biomarker trial

Dana C Hilt MD, CMO Actinogen Medical

AD/PD™ 2023: International Conference on Alzheimer's & Parkinson's Diseases

March 30, 2023

® Xanamem is a registered trademark of Actinogen Medical Limited

Dana C Hilt MD Disclosures

D C Hilt MD is employed by Actinogen as Chief Medical Officer.

D C Hilt MD is presently a consultant to the following entities: Frequency Therapeutics, Alacrita, Recognify Therapeutics, Lysosomal Therapeutics, Bial BioTech, and Cognition Therapeutics.

Served as a referee/panel member on numerous NIH and EU grant review panels.

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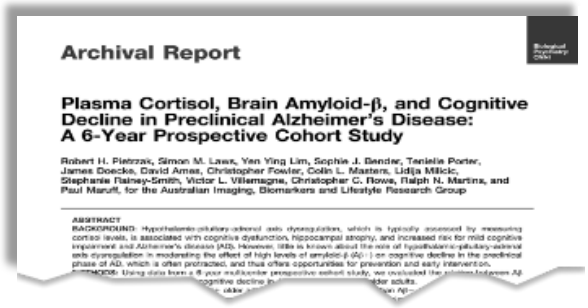
Elevated cortisol may contribute to CNS dysfunction

- Multiple studies support the association between elevated cortisol and Alzheimer's disease (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 and low folate predict probable AD after age 75[12]

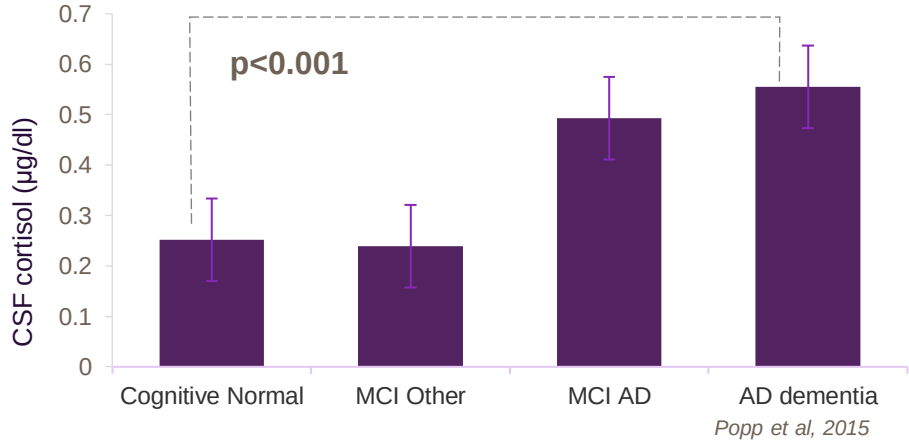

Archival Report

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

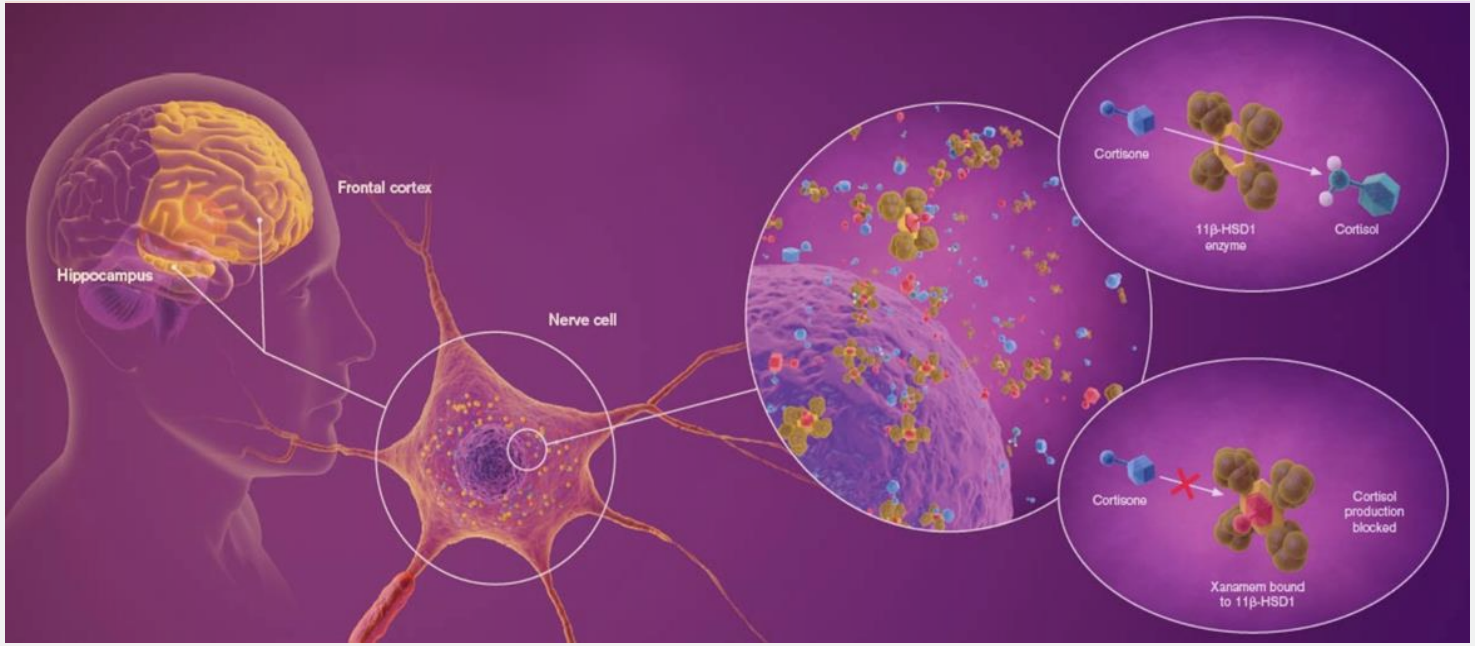
Only known brain penetrant 11β-HSD1 small molecule enzyme inhibitor

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 may have potential to be:

- Enhance cognition
- Slow progression or produce durable delay in symptoms progression in AD
- Anti-depressant/procognitive in depression with cognitive impairment

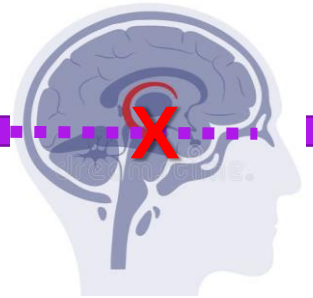


1. Xanamem® is a CNS (Central Nervous System) penetrant small molecule based on human PET scan evidence and cerebrospinal fluid (CSF) measurements
2. Sooy et al. 2015 showing effects on amyloid plaque reduction in an aged mouse model after 28 days associated with increases in insulin degrading enzyme;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

11β-HSD1 inhibition and attenuated cognitive decline

Potential mechanisms of action to reduce or halt cognitive decline

cortisone (inert)



Lowers brain cortisol

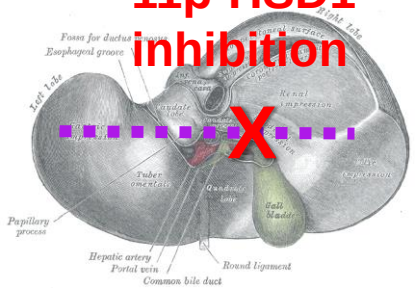
[reduced GR/MR activation]

Less negative feedback

- Increases pituitary ACTH within normal range (biomarker)
- No reduction of plasma cortisol at doses ≤10 mg

11β-HSD1 inhibition

cortisone (inert)



Lowers liver & fat cortisol

[improved Insulin sensitivity]

- Increased synaptic plasticity
- Greater dendritic complexity
- Larger hippocampal volume

+

- Reduced amyloid synthesis
- Increased amyloid catabolism
- Reduced tau phosphorylation
- Less excitotoxicity - glutamate
- Altered neuroinflammation

+

- Improved glucose handling
- Reduced vascular pathology

Better working & episodic memory

Reduced neurodegeneration

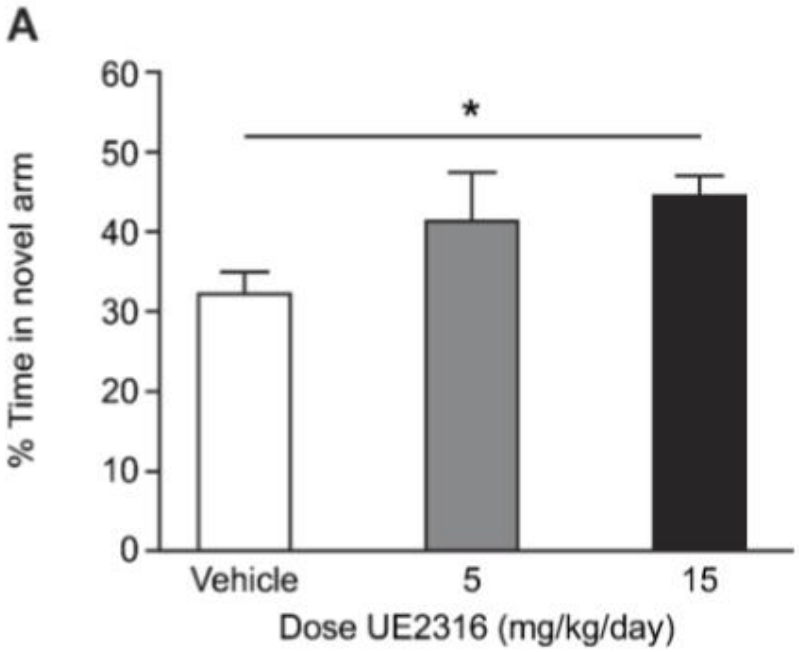
Potential metabolic benefits

Xanamem Preclinical Pharmacology

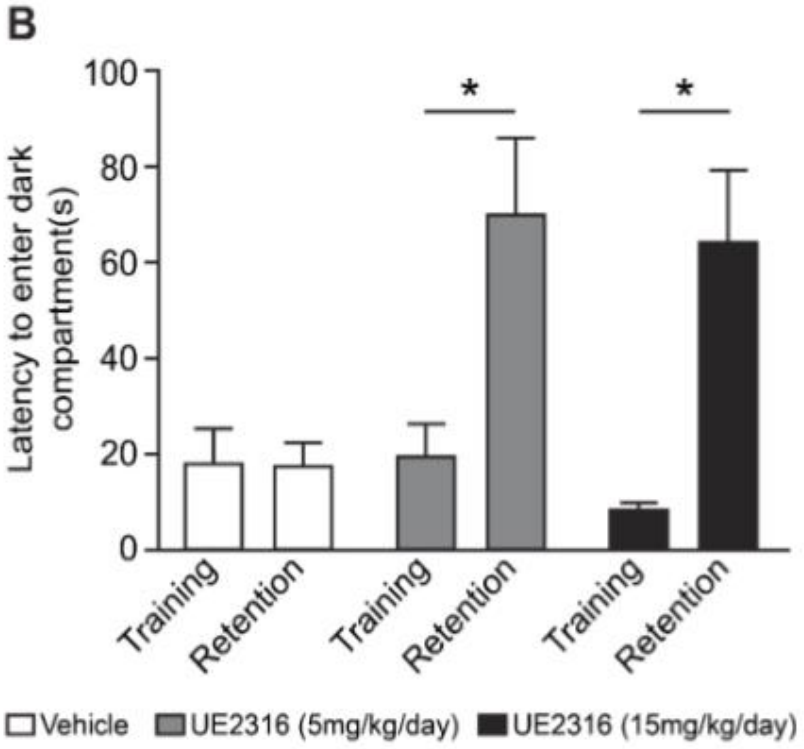
Inhibition of 11β-HSD1 enhances cognition in aged wild-type mice

Significant improvement in cognition after only 28 days treatment, continuing out to 41 weeks

Passive Avoidance: 10mg/kg s.c.



Passive Avoidance: oral dosing 30mg/kg



In human amyloid precursor protein-transgenic (Tg2576) mice, an established AD model, UE2316, a closely related Xanamem analogue, delivered for 29 days subcutaneously at 10mg/kg/day
(A) or for 41 weeks orally in food at ~30mg/kg/day
(B) to 14-month old Tg2576 mice. Increased latency to enter the dark compartment 6 hours after shock observed to a greater extent in Tg2576 mice than in WT mice. * * $\rho < 0.01$.
Sooy et al., 2015, Endocrinology 156(12):4592-4603
Preclinical rodent pharmacology studies conducted with closely related analogues as UE2343 does not bind to rodent 11β-HSD1 enzyme

Clinical data

Actinogen

Two separate normal volunteer studies have shown procognitive effects of Xanamem: Attention, working memory, and executive function

Re-analysis of the Phase 2 XanADu AD study shows procognitive and potentially clinical benefit in high pTau subgroup

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

Two large Phase 2 studies will be conducted

- **Depression with cognitive impairment (XanaCIDD)**
- **Mild/moderate AD with elevated pTau (XanaMIA)**

Evidence of Xanamem activity on cognition from multiple sources

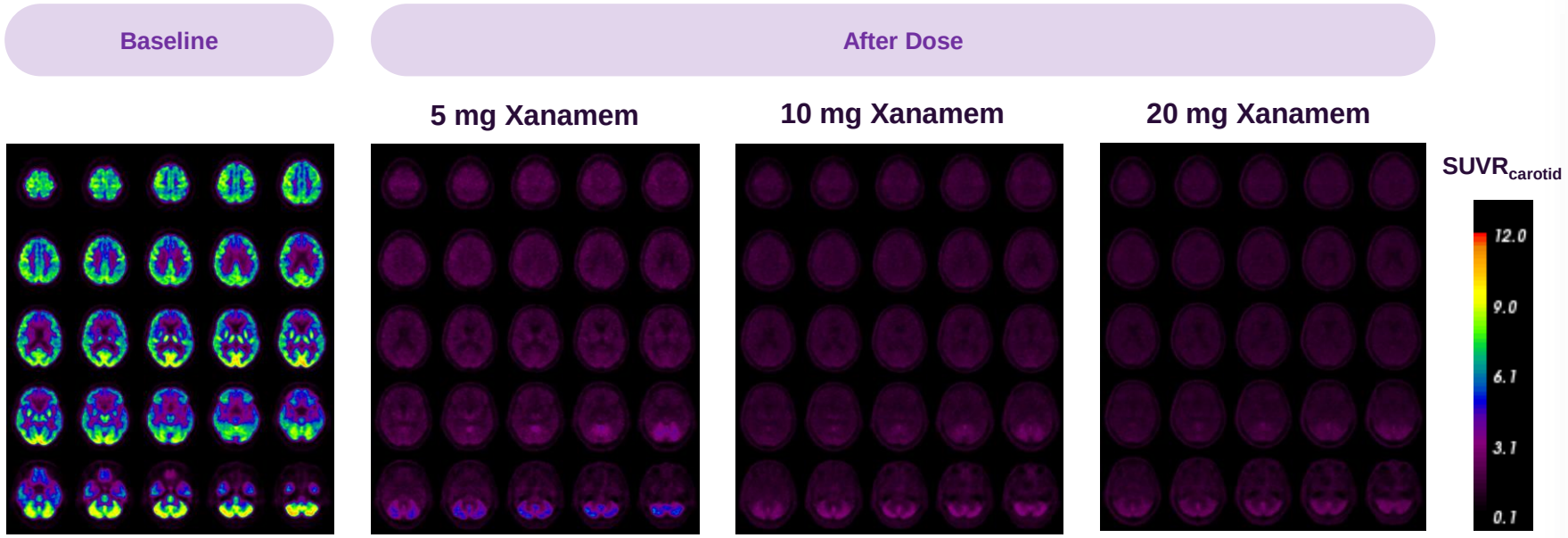
- ✓ **In animals**
  - ✓ **Protection against cognitive decline in animal model of AD using a Xanamem analogue independent of amyloid plaque[1]**
- ✓ **In humans**
  - ✓ **Consistent target engagement measured by PET[2] (significant/optimal target occupancy at 5/10 mg) & minimal ACTH response**
  - ✓ **XanaHES trial in cognitively normal older volunteers – Positive effects on CogState attention & working memory[3]**
  - ✓ **XanaMIA trial in cognitively normal older volunteers – Positive effects on CogState attention & working memory[4]**
  - ✓ **Re-analysis of Phase 2 AD XanADu data shows activity in patients with mild AD with elevated pTau181[2]**

Human and animal data support Xanamem activity at doses of 5 to 10 mg daily

1. Sooy et al. 2015 2. Publication pending 3. Actinogen data on file 4. Presented at CTAD 2022: https://wcsecure.weblink.com.au/pdf/ACW/02608009.pdf

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

Previous molecules 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.

This is consistent with full hormonal pharmacodynamic activity seen with 10 mg in clinical trials. 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 in normal volunteers: Attention domains improved in XanaHES* by Week 4

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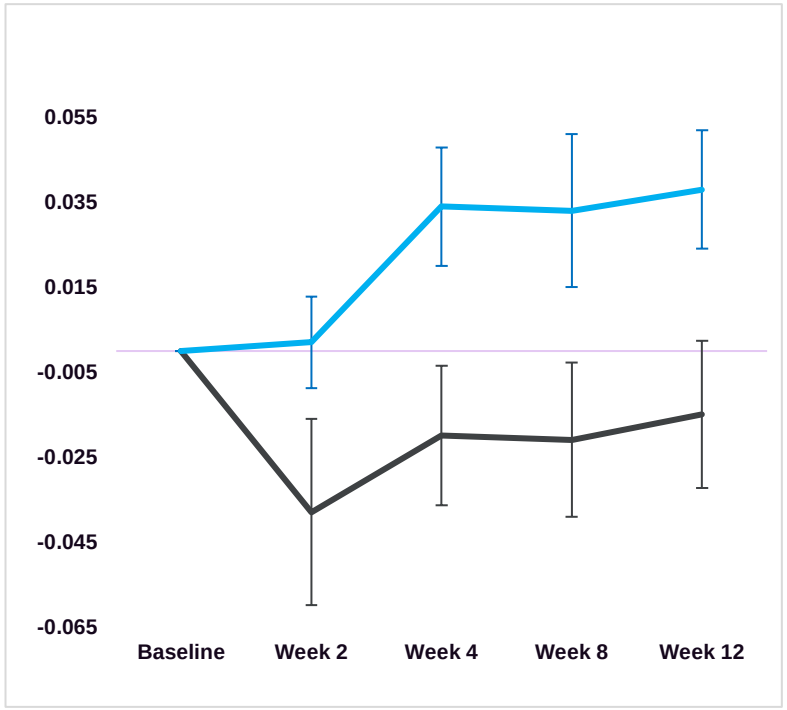
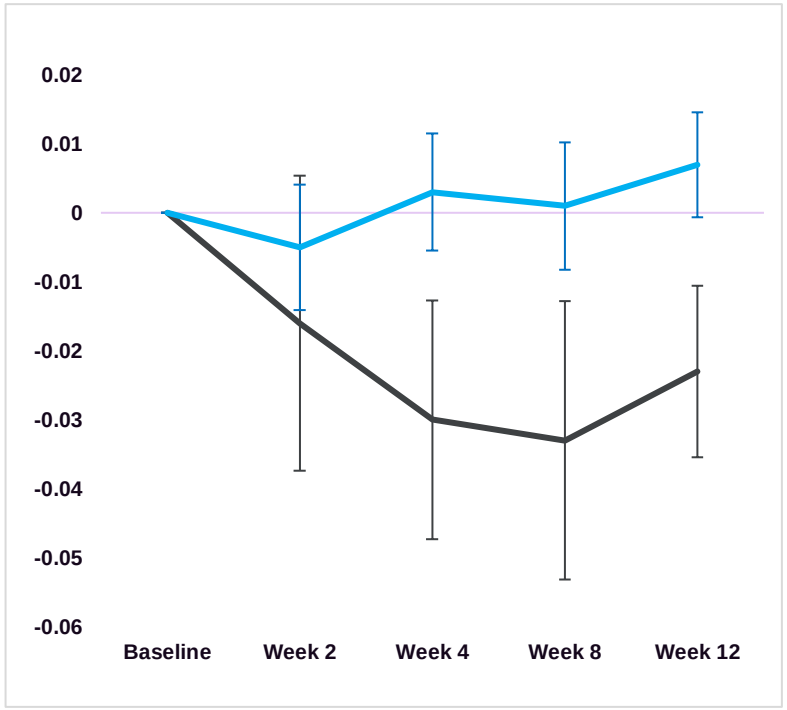
One Back Test (working memory)



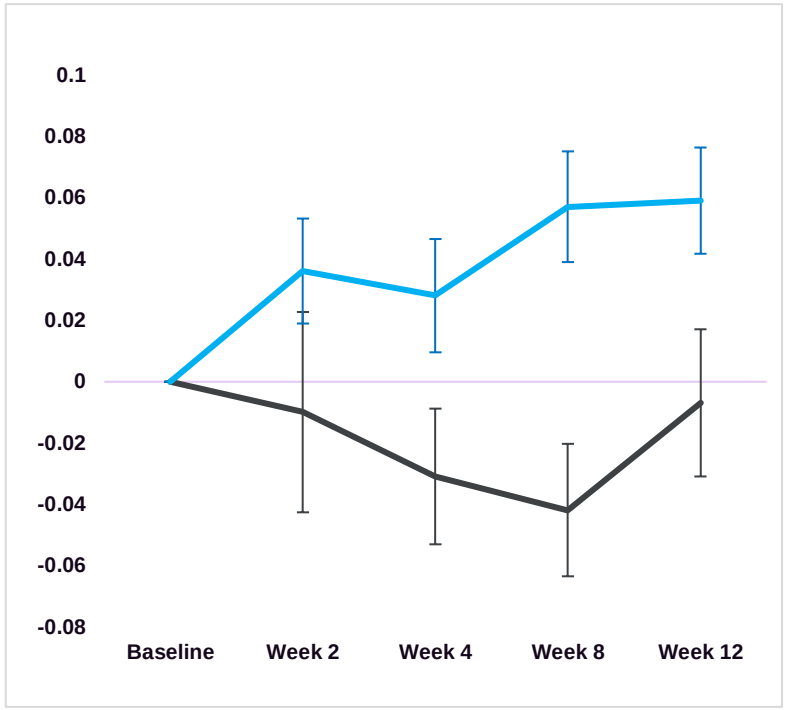
P<0.01

Identification Test (visual attention)



P=0.05

Detection Test (psychomotor function)



P=0.09

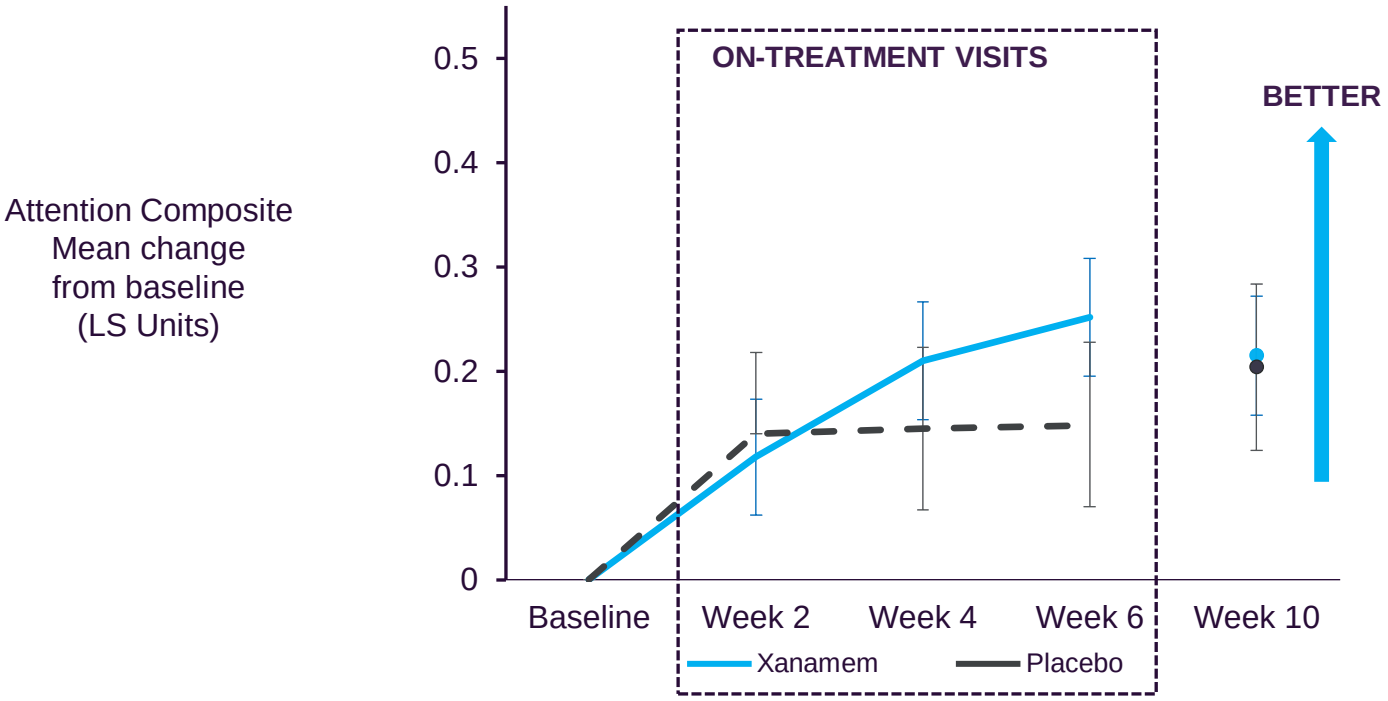
Xanamem Placebo

* n = 30 Xanamem 20mg vs n = 12 Placebo (Actinogen data on file)

Xanamem confirms improved attention/working memory by 4-6 weeks at lower doses in second trial

Computerized Cogstate test battery positive results in cognitively normal older people

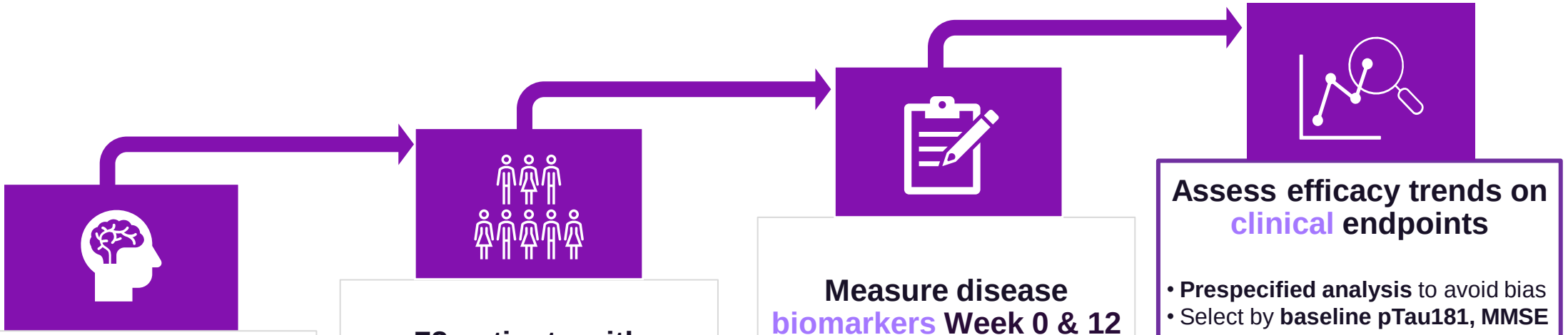
XanaMIA Phase 1b trial (n=107, Xanamem 10 mg & 5 mg)



Procognitive effects of Xanamem confirmed in second randomized trial

2022: Phase 2a AD blood biomarker study design and methods

Uses a pre-specified protocol and analysis plan to avoid bias



Phase 2a trial (XanADu)

- **185 people with mild AD** (MMSE 20-26)
- Clinical diagnosis: no amyloid imaging or biomarkers
- Placebo-controlled
- 12 weeks
- 10 mg Xanamem vs. placebo
- Primary analysis (ADAS-Cog) neg

72 patients with stored blood samples

- Available plasma stored at -80°C
- Evaluated by expert biomarker laboratory blinded to treatment assignment

Measure disease biomarkers Week 0 & 12

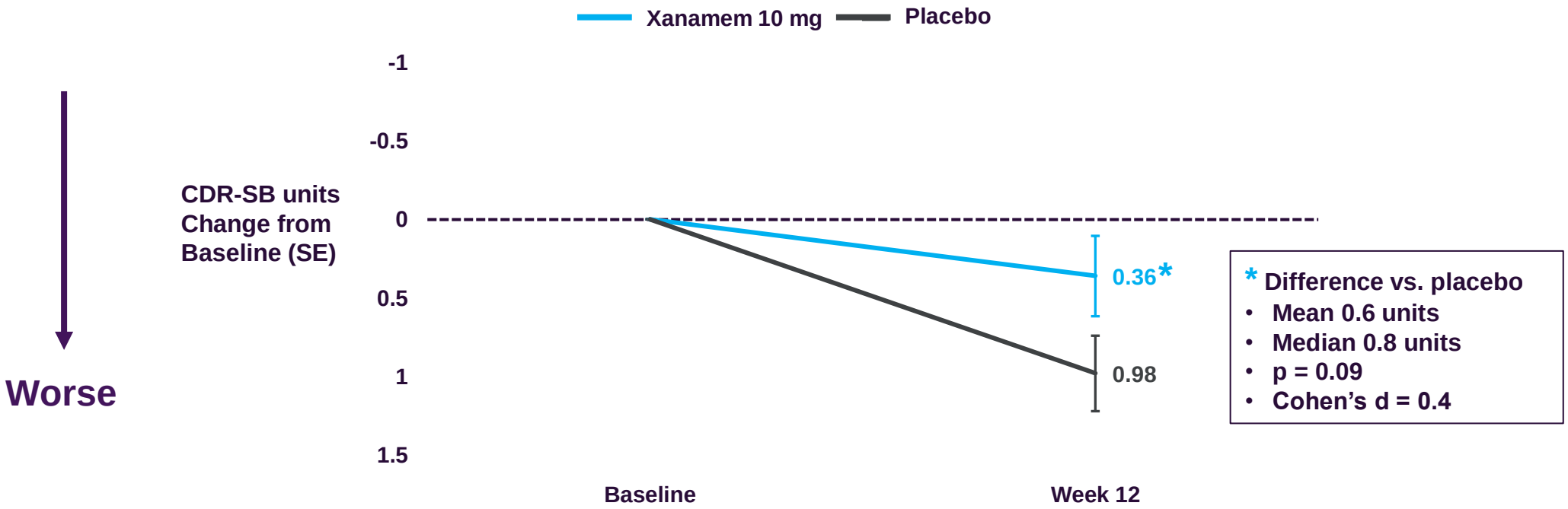
- **pTau181**
- **Amyloid 42/40 ratio**
- **GFAP**

Assess efficacy trends on clinical endpoints

- **Prespecified analysis** to avoid bias
- Select by **baseline pTau181, MMSE and amyloid ratio levels to reassess** efficacy
- Focus on **endpoints most sensitive to change** in a MCI/mild AD population (**CDR-SB)**
- Cohen's d to measure clinical effect size with no correction of p values for multiple comparisons

Xanamem prevents clinical decline in p-Tau181 elevated AD patients

In trial participants with p-Tau181 > 6.74 pg/mL, Xanamem demonstrates disease stabilization on CDR-SB



Xanamem largely prevented clinical progression over 12 weeks

Xanamem doubled rate of disease stabilization on CDR-SB in mild/moderate AD

Response analysis in pTau181-positive[1] XanADu patients (patients more likely to progress)

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

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

Xanamem treatment effect size vs. placebo of 0.6 – 0.8 SD units over 12 weeks

Xanamem 10 mg protected the majority of patients in the study from 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%)

Effect on CDR-SB in other three pre-specified groups

Groups defined by biomarker median value, MMSE by 20-23 vs. 24-26

	N	CDR-SB				
Group		**Desired change**	**Xanamem**	**Placebo**	**Cohen's d**	**p value**
pTau >10.2 pg/mL[1] (mean)	**9**	**Down**	**0.1**	**0.8**	**0.6**	**0.33**
Aβ42/40 ratio < 0.19 (mean)	29	Down	0.5	0.4	0.1	0.91
MMSE 20-23	46	Down	0.5	0.5	0.0	0.82

Clinically significant effect size of CDR-SB 0.7 units in very high pTau group, with no apparent utility of low amyloid ratio or lower MMSE

1. Published cutoff of 10.2 pg/mL[2] cutoff by Cullen et al. 2022 for progression to clinical AD. 6.74 pg/mL represents the median value of the dataset

High pTau group: NTB and other endpoints

NTB: a composite for executive function consisting of Controlled Oral Word Association (COWAT) and Category Fluency Test (CFT)

- **Improved NTB: Xanamem +0.5 vs. Placebo -2.3, Cohen's d = 0.3**
- **No effects on ADAS-Cog14, ADCOMS, MMSE, RAVLT, NPI (Cohen's d < 0.2)**

Clinically significant effect size on NTB further explored in analysis of composite characteristics

Exploring the high pTau group: baseline characteristics

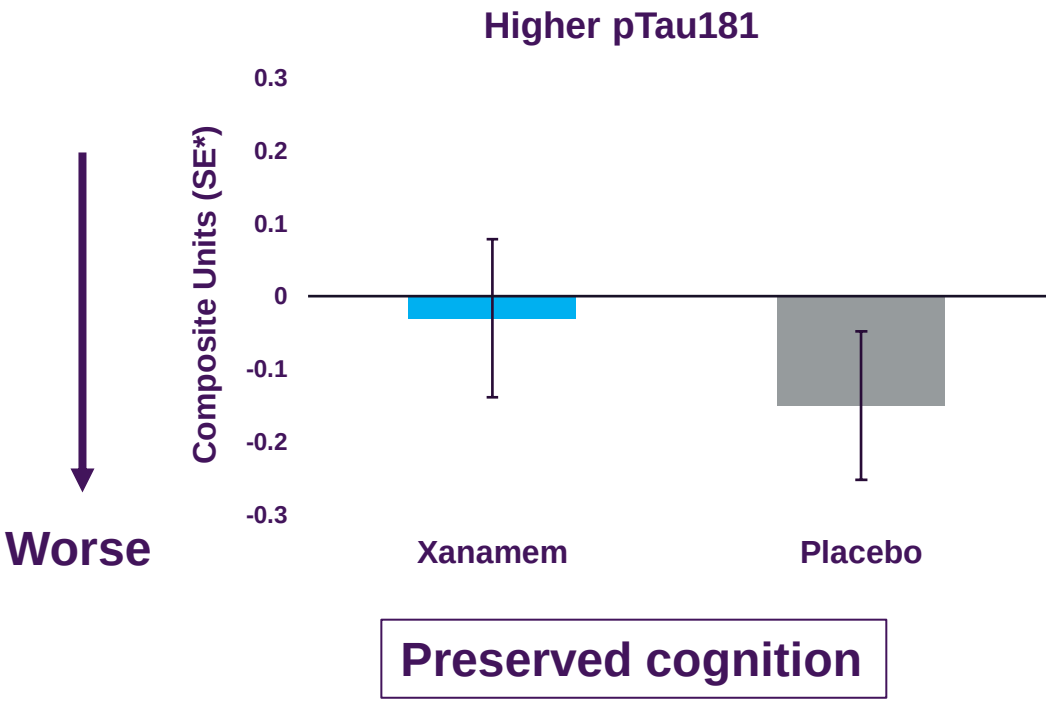
	Xanamem (n=16)	Placebo (n=18)
Age (mean, SD)	71 (8)	71 (8)
% female	50%	56%
% donepezil therapy[1]	44%	61%
ADASCog14 (mean, SD)	34 (5)	32 (8)
ADCOMS (mean, SD)	0.56 (0.13)	0.52 (0.19)
MMSE (mean, SD)	22 (3)	23 (2)
CDR-SB (mean, SD)	4.1 (1.2)	3.6 (1.6)
pTau pg/mL (mean, SD)	9.3 (2.6)	11.9 (11.6)
pTau pg/ml (median)	8.6	8.8
Aβ42/40 ratio (mean, SD)	0.19 (0.03)	0.19 (0.03)
GFAP pg/mL (mean, SD)	132 (77)	136 (95)

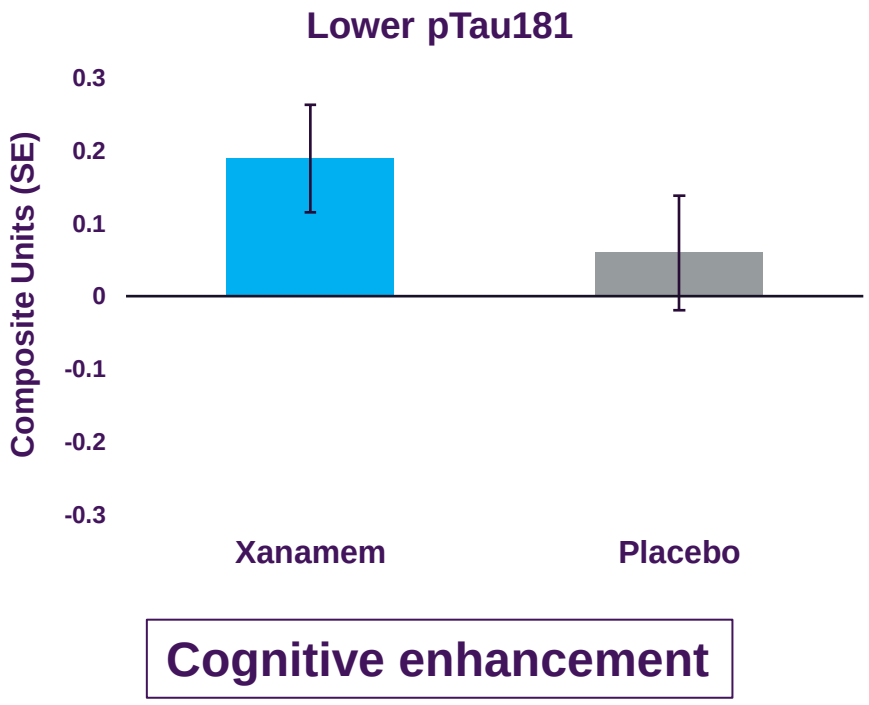
Groups were generally well balanced in key characteristics
Beneficial effects not likely due to group imbalance

1. Including other AChE inhibitiors and/or memantine

Exploratory analyses: Change from baseline in cognition composite

Trends in change of composite of word recall & recognition, CFT & COWAT (p=NS)





Preserved cognition

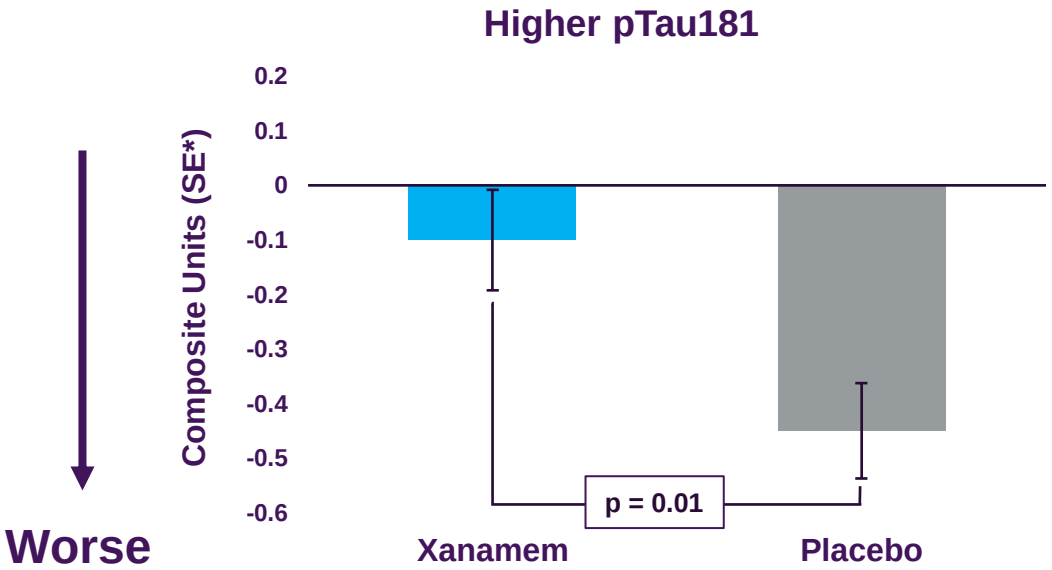
Cognitive enhancement

Consistent with Xanamem activity as a cognitive enhancer & disease-modifier

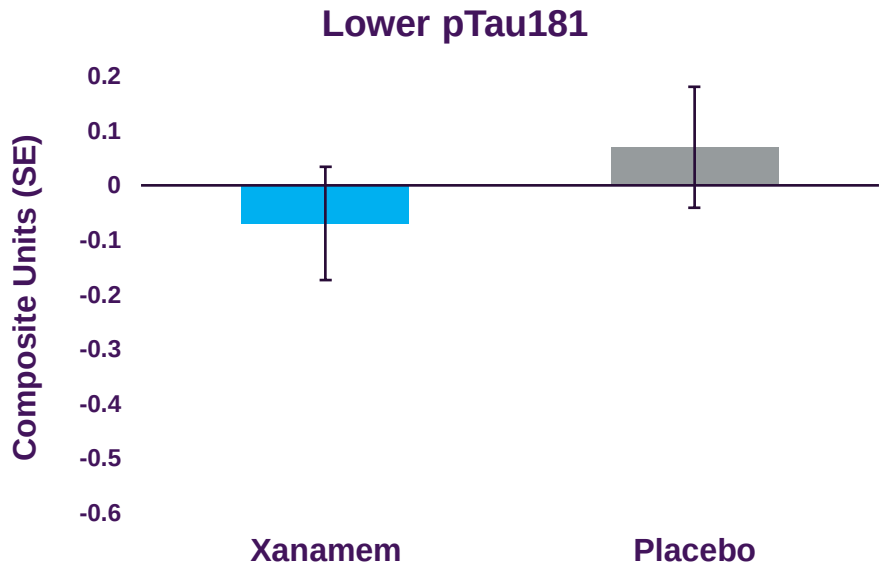
* Standard Error of the mean

Exploratory: Change from baseline in cognitive-functional composite (with CDR-SB)

Trends in change of composite of CDR-SB, word recall & recognition, CFT, COWAT



Preserved cognition & function



No effect

Consistent with Xanamem activity as a cognitive & functional preserver

* Standard Error of the mean

Safety data phase 2a AD patients

Actinogen

10mg daily over 12 weeks - clinically diagnosed, mild AD

✓ No treatment-related SAEs in program to date (n=301)

TEAE term ACW0002*	Xanamem (n=91)	Placebo (n=94)	Total (n=185)
Headache	5 (5.5%)	2 (2.1%)	7 (3.8%)
Dizziness	4 (4.4%)	3 (3.2%)	7 (3.8%)
Diarrhoea	1 (1.1%)	4 (4.3%)	5 (2.7%)
Fatigue	3 (3.3%)	1 (1.1%)	4 (2.2%)
Nerve conduction abnormal	1 (1.1%)	3 (3.2%)	4 (2.2%)
Somnolence	1 (1.1%)	3 (3.2%)	4 (2.2%)
Decreased appetite	2 (2.2%)	0 (0.0%)	2 (1.1%)

* TEAEs reported by more than one patient in any group in the largest clinical study to date

The new analyses indicate further study of Xanamem in AD is indicated

- ✓ **Analysis suggests clinical activity of Xanamem in mild AD patients with more rapidly progressing disease**
- ✓ **Large clinical effect size, (p=0.09 in modest sample size) will need to be confirmed in larger studies**
- ✓ **Indicates potential utility of elevated blood pTau 181 levels to select suitable patients that are likely to progress for next larger and longer Phase 2b trial**
- ✓ ***Phase 2b XanaMIA study will enroll mild/moderate AD patients with elevated pTau 181 and measure Cognition Composite, CDR-SB, Amsterdam IADL and other endpoints***

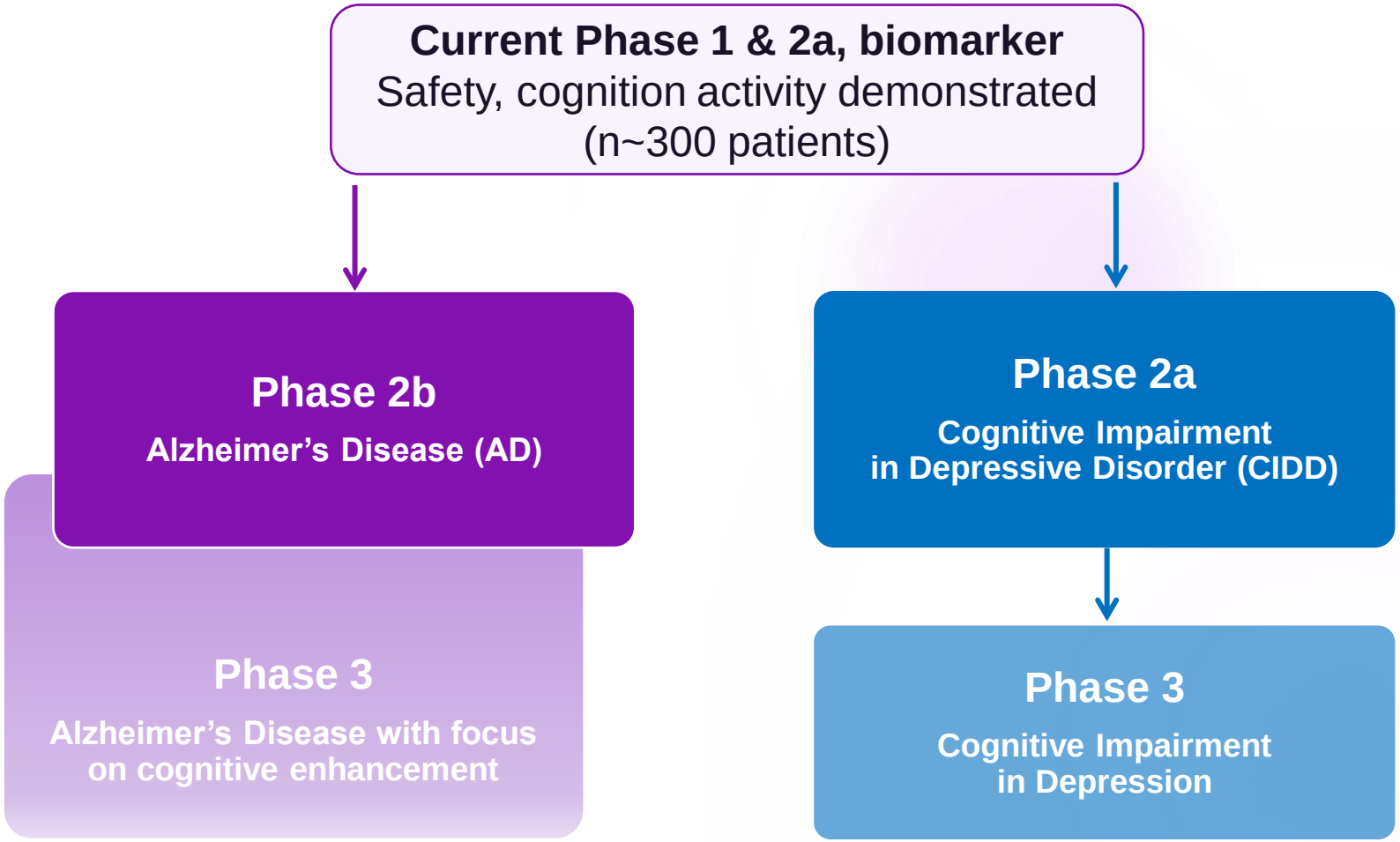
Confirms the procognitive & positive clinical effects of Xanamem at 10 mg daily

Xanamem moving to POC Phase 2 studies: AD and Cognitive Impairment in Depression

Biomarker data validate planned Phase 2b protocol in Mild Cognitive Impairment / mild AD with positive blood pTau

Xanamem Phase 2 & 3 program

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



The XanaMIA Phase 2b Trial

A double-blind, randomized, 6-month, 3-arm clinical trial to assess safety, tolerability, and efficacy of Xanamem 5 mg and 10 mg daily in patients with MCI and Mild AD