ASX ANNOUNCEMENT

Actinogen Clinical Trials Science Forum 2024 today – Pipeline in a Pill

Actinogen CMO, Dr Dana Hilt and guests explore the unique properties of Xanamem® for the potential treatment of cognitive impairment in multiple diseases at 3pm AEST today

Sydney, 23 May 2024. Actinogen Medical ASX:ACW ("ACW" or "the Company") is pleased to announce that Actinogen's Chief Medical Officer, Dr Dana Hilt and guests Professors John Harrison and Paul Rolan will join in a highly informative 'plain English' panel discussion exploring the unique properties of Xanamem for the potential treatment of cognitive impairment in multiple diseases.

Pre-register now, or register and join at 3pm AEST today:
https://actinogenmedical.zoom.us/webinar/register/WN_3YoG2A5JS7Ky5qe67dfkIA

A copy of the webinar presentation is attached to this announcement. At the conclusion of the presentation, there will be an opportunity for questions from webinar attendees. A recording of the forum will be made available as soon as possible after the conclusion of the event on the Company's YouTube channel and links to the recording will be provided on the Company's website https://actinogen.com.au/ and social media platforms.

ENDS

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Announcement authorised by the Board of Directors of Actinogen Medical

® Xanamem is a registered trademark of Actinogen Medical Limited

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.

Current Clinical Trials

The **XanaCIDD Phase 2a cognition & depression trial** is a double-blind, six-week proof-of-concept, placebo-controlled, parallel group design trial in 167 patients. Participants are evenly randomized to receive Xanamem 10 mg once daily or placebo, in some cases in addition to their existing antidepressant therapy, and effects on cognition and depression are assessed.

The **XanaMIA Phase 2b 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 effects on cognition, function and progression of Alzheimer's disease are assessed. Thus, Xanamem is being assessed in this trial for its potential effects as a both a cognitive enhancer and a disease course modifier.

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

Clinical Trials Science Forum 2024

Xanamem® – A pipeline in a pill for the potential treatment of cognitive impairment in multiple diseases

23 May 2024

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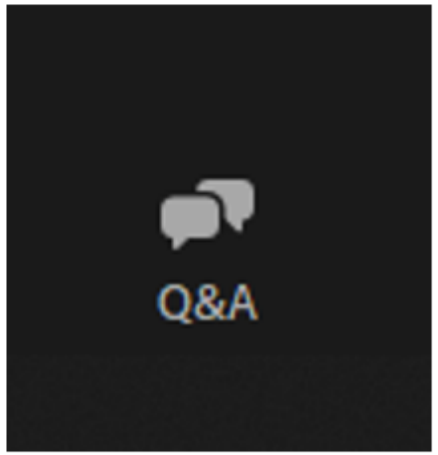
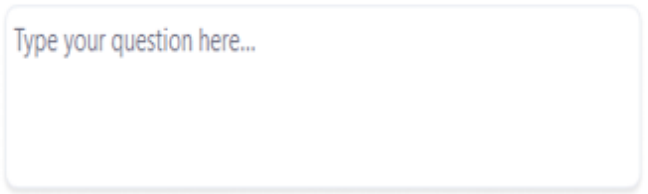
Online Q&A

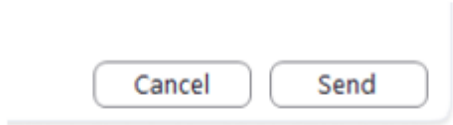
1. Click on the Q&A icon



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



3. Hit enter on your keyboard to submit your message



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Current state of Xanamem development: Enhancement of cognition in three studies with excellent safety and tolerability

Dr Dana C Hilt MD FAAN

ACW Chief Medical Officer

Xanamem Summary

- Xanamem is an oral 11β-HSD1 inhibitor: safe, well tolerated, pro-cognitive effects
- Designed to lower brain cortisol with rapid onset of clinical activity
- Cortisol target validation in animal models of Alzheimer's disease (AD) and by cognitive benefit shown in multiple controlled trials of Xanamem (healthy human volunteers and AD patients)
- Excellent safety profile, low drug interaction potential, can be administered with current medications
- Initial phase 2 AD trial demonstrated pro-cognitive effects and indications of clinical benefit (CDR-SB slowing)
- Phase 2 trials are on-going in Cognitive Impairment in Major Depressive Disorder (CIDD) and AD

Targeting large clinical opportunities with unmet need

Current clinical focus of Xanamem development: AD and CIDD

- Alzheimer's Disease global prevalence: 33 million patients
- Major Depressive Disorder: 280 million patients, ~70% associated with cognitive impairment, average cog deficit 0.5 SD below normal

Potential future indications:

- Other neurodegenerative diseases such as Parkinson's disease, Frontotemporal dementia & Lewy-Body dementia: 17 million patients
- Schizophrenia-associated cognitive impairment: 24 million patients
- Cognitive impairment in bipolar disease: 46 million patients

Alzheimer's Disease International, other dementias 33% of all (link)
World Health Organisation – Depression facts (link)

Research & Markets CIAS Report (link)
https://www.singlecare.com/blog/news/bipolar-disorder-statistics/

Actinogen Xanamem Phase 2 trials underway

Supported by extensive existing clinical data from four previous trials of Xanamem 10mg

Phase 2a proof-of-concept trial in patients with Cognitive Impairment & Depression (n=167) → **Results early Q3 2024**

Phase 2b confirmatory trial in patients with mild-moderate Alzheimer's disease (n=220) → **Interim results Mid 2025 N=100**

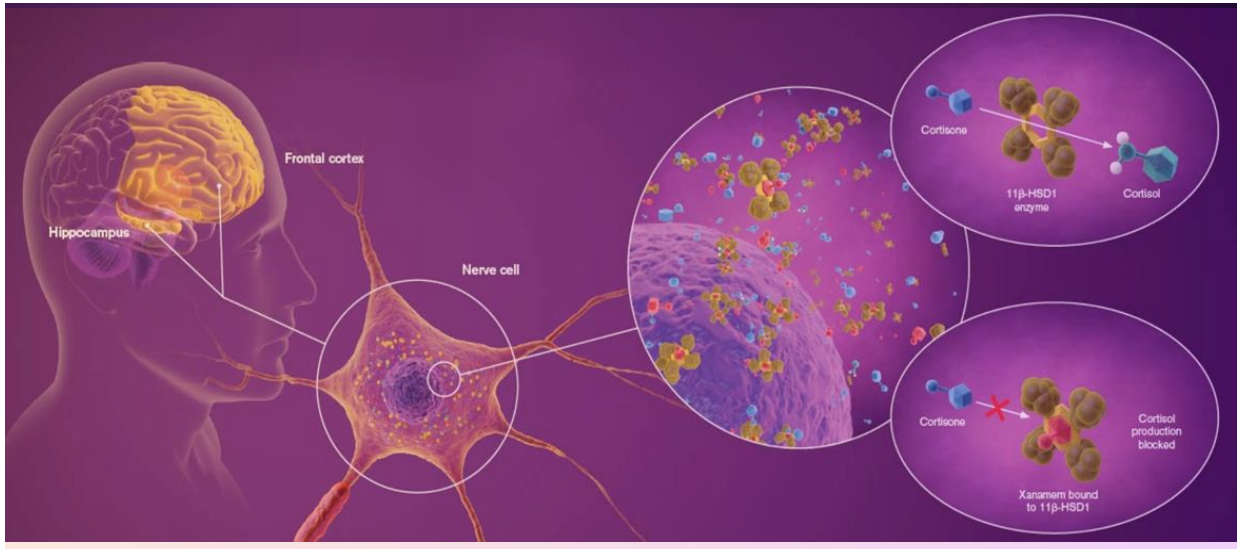
Xanamem: oral, low-dose, once-a-day treatment with a unique, non-amyloid/tau mechanism

Rodent experimental studies & clinical trials validate cortisol target for treatment of AD[1-4]

Brain penetrant 11β-HSD1 small molecule enzyme inhibitor **reduces cortisol inside brain** cells[3,4] - modulating signalling pathways and underlying disease processes

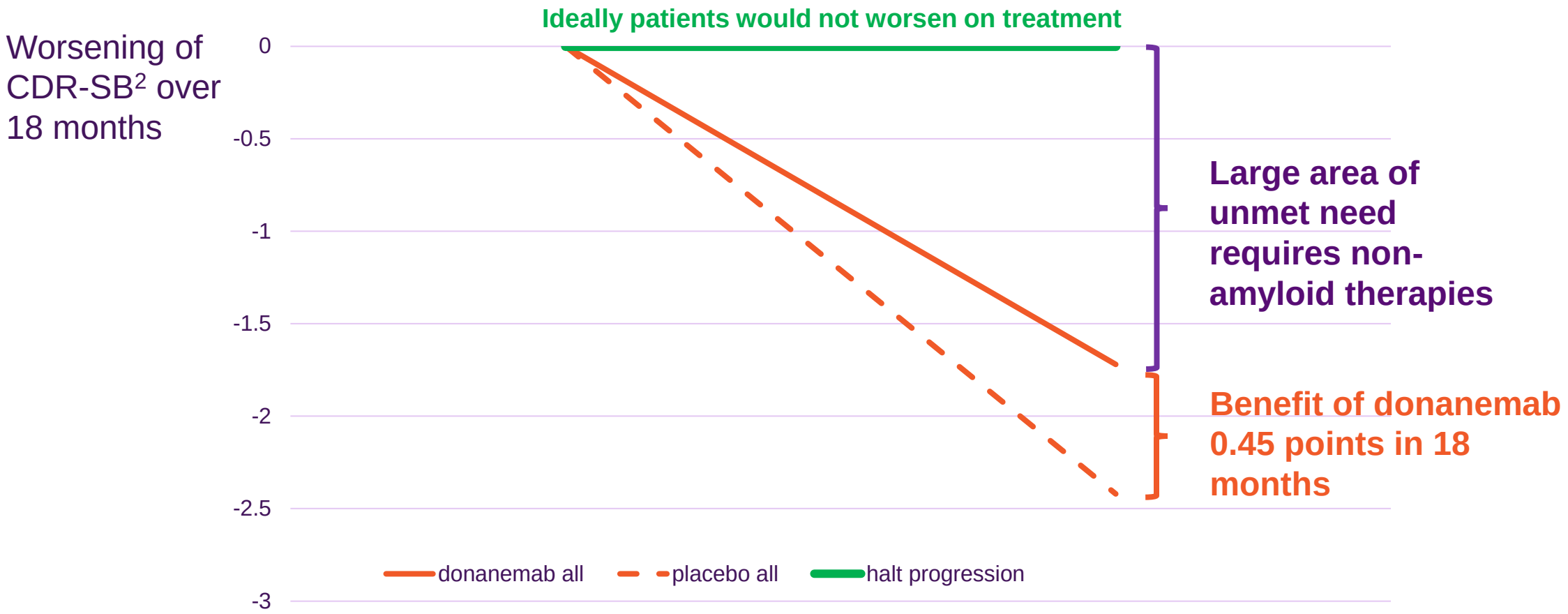
Potential to be:

- Rapidly **cognitive enhancing**
- **Disease-modifying** (slow or halt progression) in AD[1,3]
- **Anti-depressant**
- **Enhances insulin sensitivity**
- **May have central anti-inflammatory effects**



1. 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 – at 13 month cognitive protection was independent of continued amyloid deposition; 2. Popoli et al. 2011 microglial cell modulation in rats, effects on glutamate, cannabinoid and other signalling pathways; 3. Hilt, D. Oral symposium AD/PD International Conference 2023; Actinogen website: Actinogen – News; 4. based on human PET scan evidence (data on file), Webster et al. 2017 Selection and early clinical evaluation of the brain-penetrant 11β-hydroxysteroid dehydrogenase type 1 (11β-HSD1) inhibitor UE2343 (Xanamem™)

Newer anti-amyloid “immunotherapy” antibodies shown to slow but not halt progression of AD[1]

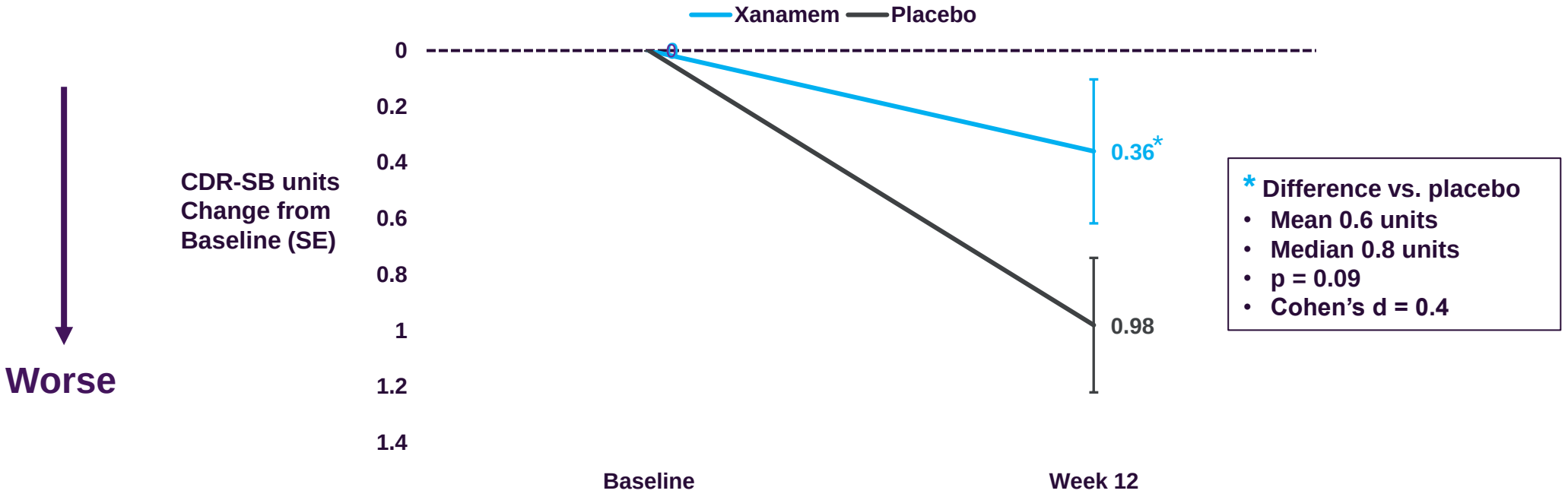


Drugs targeting other mechanisms like Xanamem are needed

1. Donamemab is an anti-amyloid antibody given as an intravenous infusion every 4 weeks until amyloid clearance (Sims JR at al. *JAMA*. Published online July 17, 2023. doi:10.1001/jama.2023.13239 Data shown are for whole population studied with absolute difference to placebo of 0.7 points, intermediate tau population difference also 0.7 points
2. CDR-SB is an 18-point scale measuring functional and cognitive status, patients in the donanemab trial had an average baseline score of 4 ± 2 points

Xanamem slows the rate of CDR-SB functional decline in mild AD[1]

Patients with elevated plasma pTau181 indicating progressive, amyloid-positive disease (n=34): Xanamem slows CDR-SB worsening

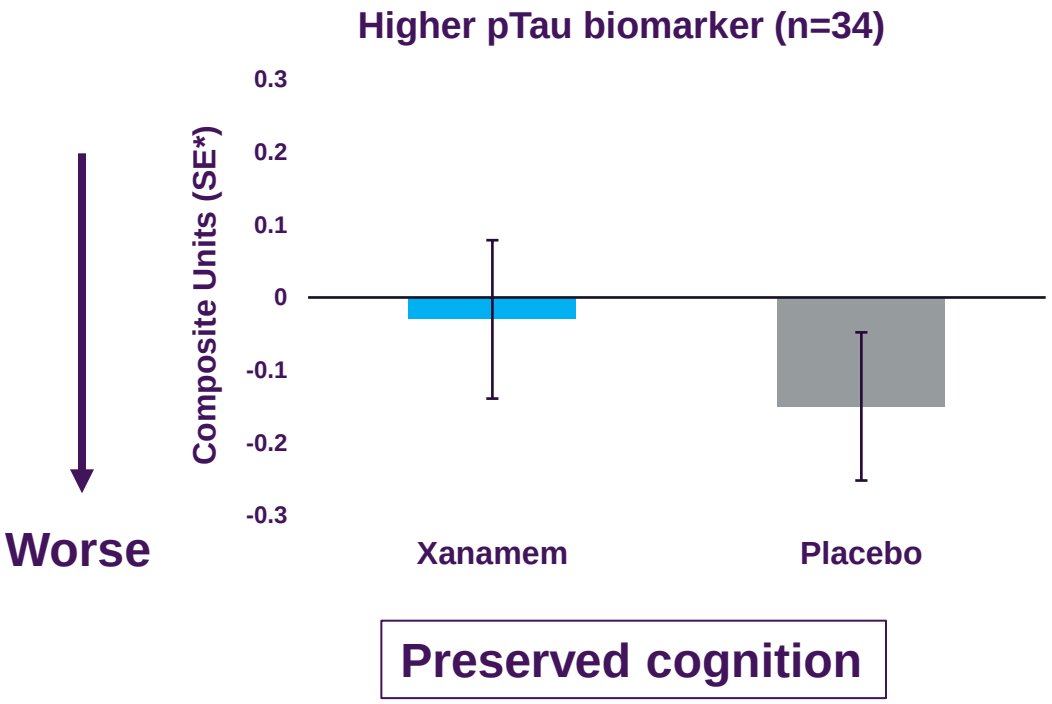


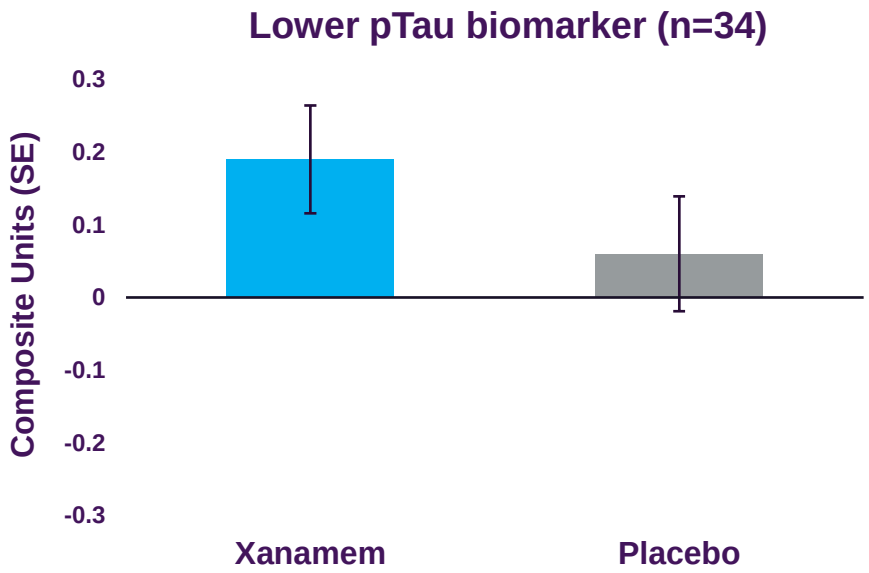
Extrapolated to 18 months ➡ effect size would be 3.7 points (8x donanemab's 0.45)

1 Patients with a pre-treatment plasma pTau181 level greater than the pre-specified median of 6.74 pg/mL to indicate AD pathology and likelihood of progressive disease; similar effect size for pTau >10.2 pg/mL cutoff; effect size 8-10 times greater than 0.4-0.45 reported for lecanemab (USPI Leqembi 2023 & van Dyck et al. 2022; DOI: 10.1056/NEJMoa2212948) if extrapolated to 18 months

Cognitive improvements suggest potential clinical benefits across dementia patient sub-types*

Positive trends in both high and low plasma pTau biomarker groups





Preserved cognition

Cognitive enhancement

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

* Post hoc analysis of composite of word recall & recognition, CFT & COWAT tests (p=NS), error bars show Standard Error of the Mean; low pTau patients less likely to have amyloid-positive disease, results consistent with volunteer data shown in Slide 7

Xanamem has excellent safety/tolerability profile

No emerging safety signals

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%)
Diarrhea	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%)

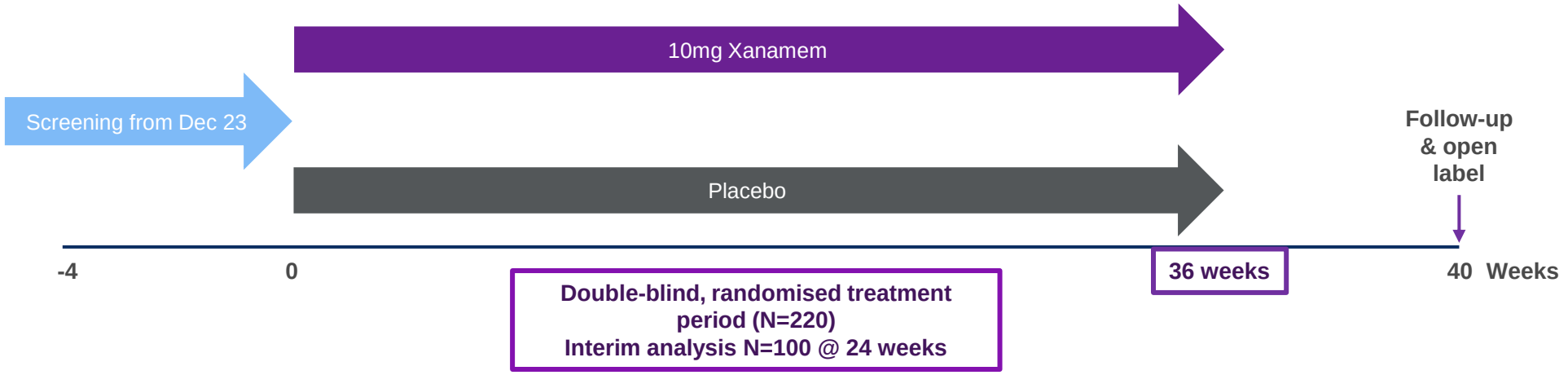
* TEAEs reported by more than one patient in any group in the largest clinical study to date

✓ **No treatment-related Serious Adverse Events in clinical program to date**

https://clinicaltrials.gov/ct2/show/results/NCT02727699?term=actinogen&draw=2&rank=3

XanaMIA Phase 2b trial in Alzheimer's Disease

Initial, interim results in mid 2025, final results H1 2026



Key inclusion criteria	Primary Endpoint	Key Secondary Endpoints	Implementation
• Blood pTau biomarker positive • Mild-moderate Alzheimer's by NIA-AA criteria	• Cognitive Test Battery (7 cognitive measures)	• CDR-SB (functional and cognitive measure) • Amsterdam Activity of Daily Living (functional measure)	• Commence enrolment at up to 15 Australian sites • Interim analysis when 100 people complete 24 weeks • Add US sites when feasible

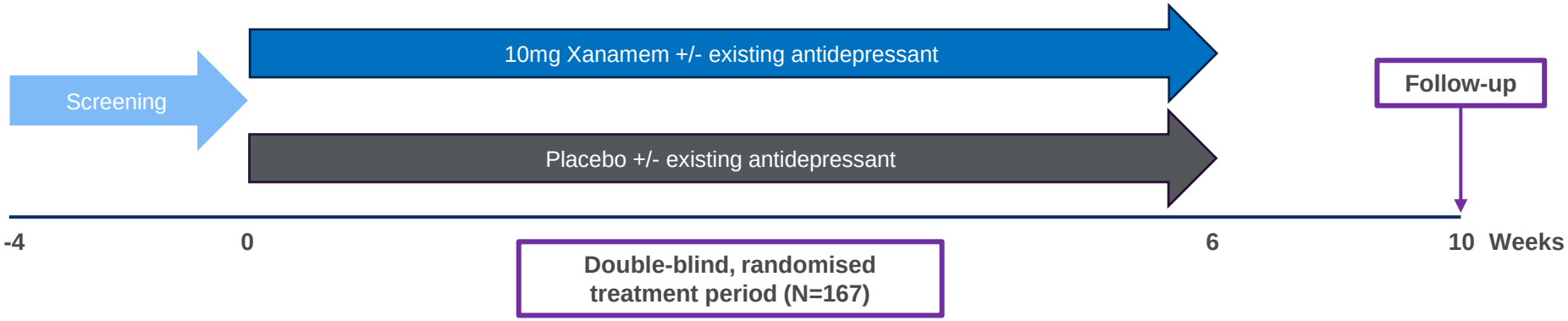
NIA-AA=National Institute of Aging - Alzheimer's Association; CDR-SB Clinical Dementia Rating Scale – Sum of Boxes

Targeting brain tissue cortisol with Xanamem is a promising strategy in cognitive impairment & depression

- ✓ Elevated cortisol associated with severe, melancholic depression[2]
- ✓ Cortisol levels associated with treatment outcomes, relapse, & cognition[3]
- ✓ 80-90% of MDD patients report cognitive symptoms[1]
- ✓ Cognitive symptoms often persist during remission[1]
- ✓ Positive effects with GR receptor antagonism with mifepristone[4]
- ✓ Meta-analysis of clinical cortisol approaches[5]
- ✓ **Xanamem has improved attention & working memory in two trials in cognitively normal, older volunteers[6]**

1. 3-year prospective study and review, Conradi et al. 2011
2. Quantitative summary of four decades of research, Stetler & Miller 2011
3. Depression literature review, Malhi & Mann 2018; HPA axis in major depression, Keller et al. 2016
4. GR, **glucocorticoid receptor;** Combined analysis of mifepristone for psychotic depression, Block et al. 2018; mifepristone effects on depression in biopolar disorder, Young et al. 2004; Evidence from clinical studies with CRH_1 receptor antagonists, Holsboer & Ising 2008
5. Meta-analysis of prior trials aimed at reducing cortisol, Ding et. al 2021
6. XanaHES, XanaMIA-DR trials, Cogstate “Attention Composite” also used in XanaCIDD trial (Actinogen data on file)

XanaCIDD proof-of-concept Phase 2a trial in Cognitive Impairment & Depression (MDD)



Key inclusion/exclusion criteria	Primary Endpoints	Key Secondary Endpoints	Key Implementation Features
• Primary diagnosis of **MDD** • **Persistent depressive symptoms/deficit (HAM-D) despite stable existing therapy or no therapy (but previously treated)** • **Cognitive impairment** relative to demographic norms (~0.5 SD)	• **Cogstate Cognitive Test Battery Attentional Composite** (attention and working memory)*	• Montgomery-Åsberg Depression Rating Scale (**MADRS**) • Executive Function Cognitive Composite • Memory Function Cognitive Composite	• **Australia & UK** trial sites • **Actinogen "hands-on" operational model** • **167 enrolled** • **Final results early Q3 2024**

* Same attention and working memory tests shown to demonstrate Xanamem effect in the XanaHES and XanaMIA Part A trials

Xanamem in CIDD and Alzheimer's Disease

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CIDD

- The XanaCIDD phase 2a trial will provide topline data in early Q3 2024
- Xanamem could have pro-cognitive effects and/or anti-depressant effects in MDD
- Cognitive benefits can be extrapolated to AD and other diseases

AD

- The previous phase 2a trial provides data showing that Xanamem has activity in patients with biomarker-positive, mild/moderate AD with pro-cognitive trends and indication of clinical benefit (slowed rate of progression in CDR-SB)
- The XanaMIA phase 2b AD trial is being conducted to confirm and extend the observed treatment effects of Xanamem. Interim analysis in mid 2025.

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The clinical pharmacology of Xanamem

Professor Paul Rolan MBBS MD FRACP FFPM(UK) FFPMANZCA

Clinical Pharmacologist

Key Xanamem properties – pharmacology and pharmacokinetics

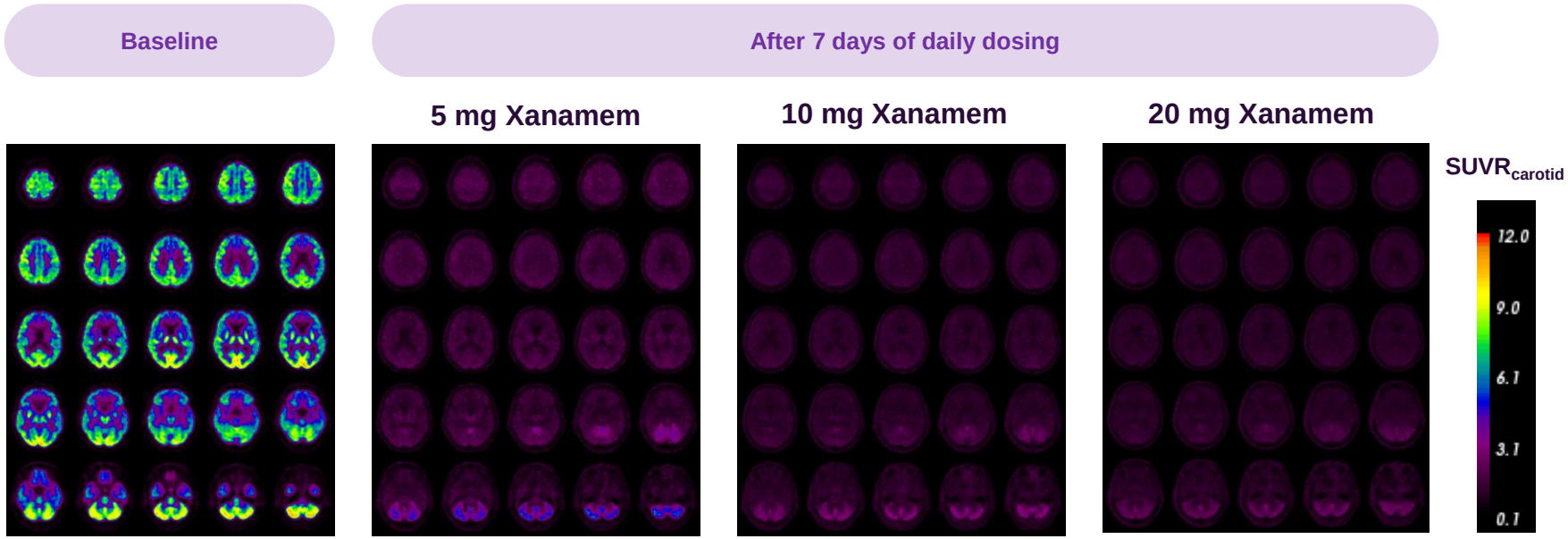
- Highly potent at target enzyme IC_{50} 28nM
- Highly selective compared to other enzymes / receptors
- The combination of **high potency** and **high selectivity** mean very low likelihood of off-target effects
  - enhances safety
  - also reduces drug-drug interactions – many elderly are on multiple medications
- Pharmacokinetics (distribution of drug in the body)
  - well absorbed, unaffected by food; half-life 14 hours
  - confirmed CSF levels approximately one third of plasma
  - consistent pharmacokinetics in young and elderly and AD patients
  - suitable for convenient once-daily dosing

Key Xanamem properties – pharmacodynamics

- Confirmed high binding to brain target on PET scan at low doses 5-10mg
- 2x ACTH hormonal counterresponse (largely within laboratory normal limits) consistent across all doses studied, including 5 mg daily
- No durable effect on circulating cortisol level or other important related hormones – leaves stress response via the adrenal glands unaffected
- Cognitive improvement seen in three clinical placebo-controlled, double-blind trials

PET trial shows full target engagement in the brain at low doses

Target inhibition 30-60% per rodent efficacy models of cognition[1]
Previous enzyme inhibitors[2] have not achieved adequate brain concentrations



PET data[3] 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.

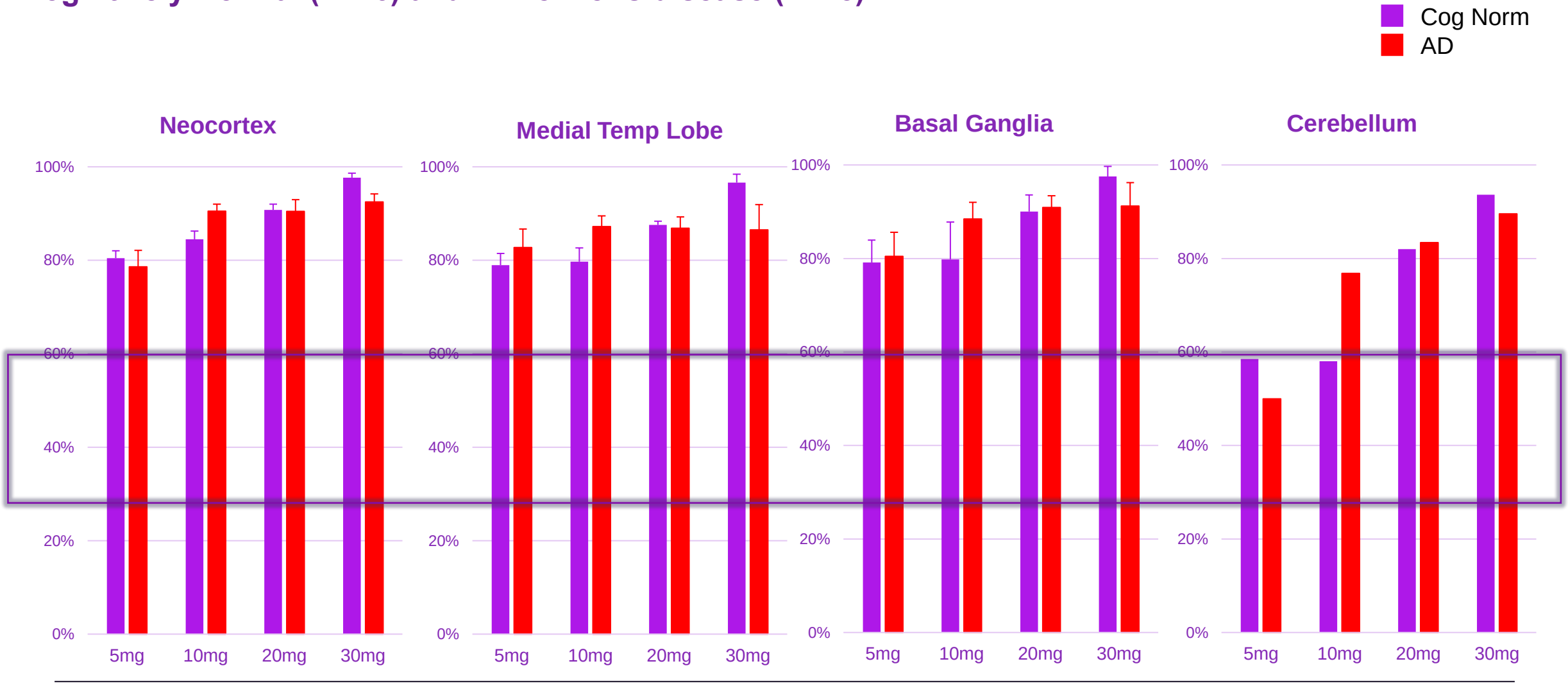
This is consistent with full hormonal pharmacodynamic activity seen in clinical trials with doses as low as 5 mg.

Validates 10mg dose in efficacy trials

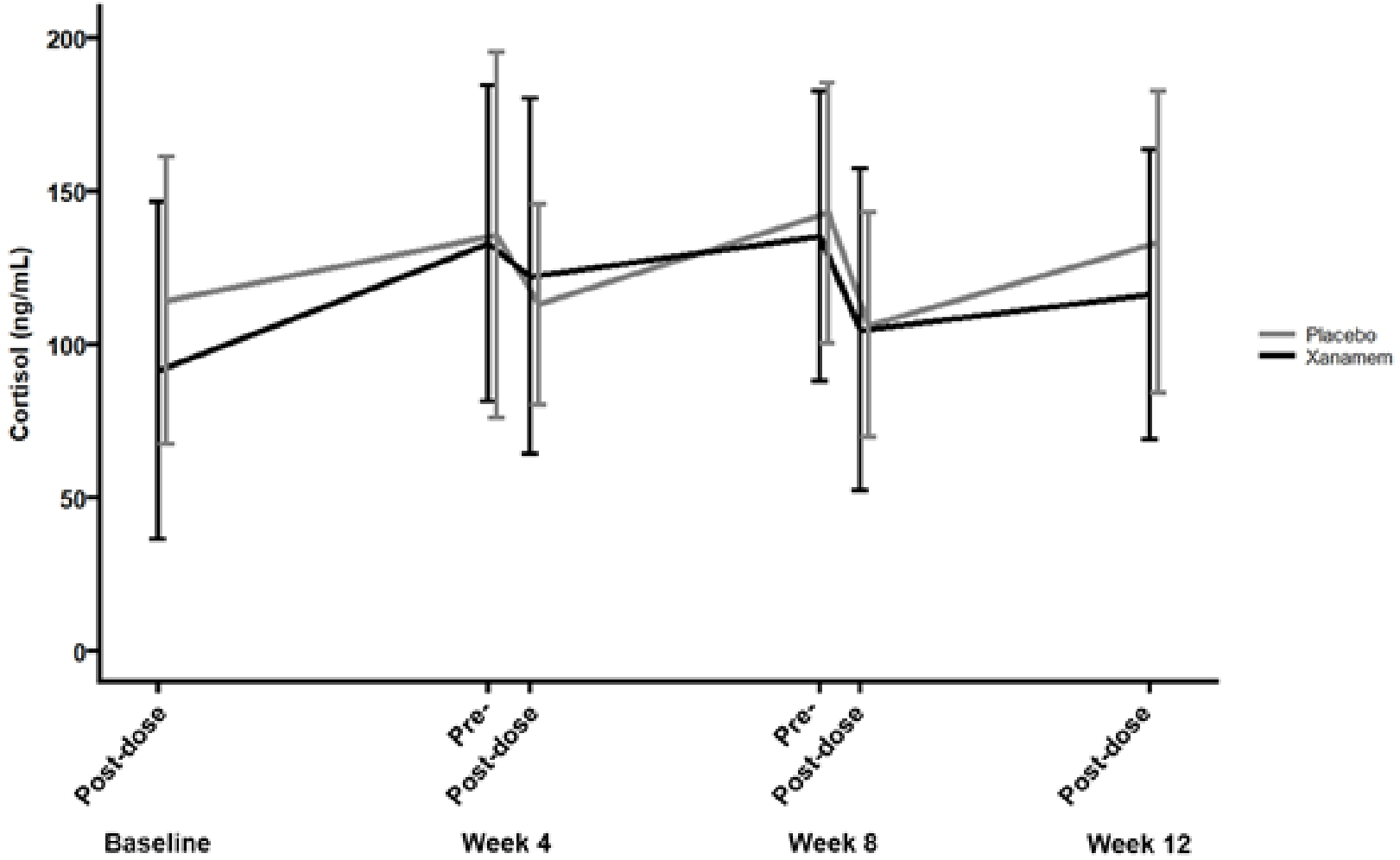
1. Sooy et al. 2010, 2015
2. ABT-384 was claimed to have brain penetrant ability based on likely hepatic effects on deuterated cortisol (Katz et al. 2013), negative 12-week AD trial (Marek et al. 2014)
3. Study population consisted of ~50% healthy older subjects who were cognitively normal and ~50% with 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.

Regional %Occupancy Exceeds 30-60% target

Cognitively Normal (n=16) and Alzheimer's disease (n=15)



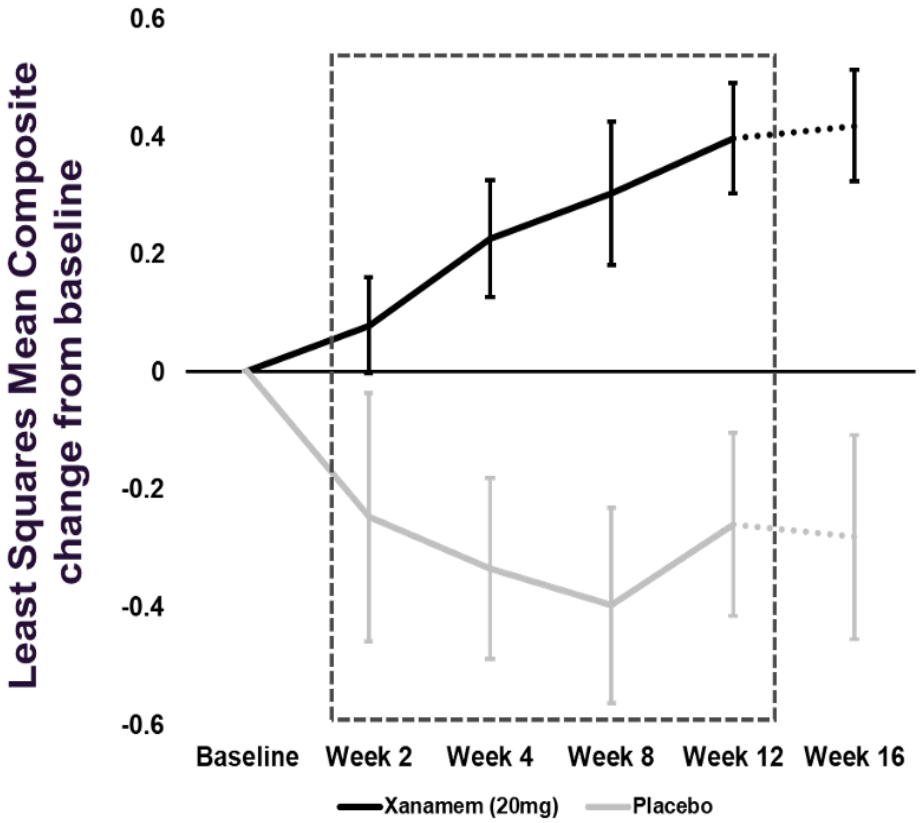
No effect on plasma cortisol levels in AD patients over 12 weeks – XanADu study



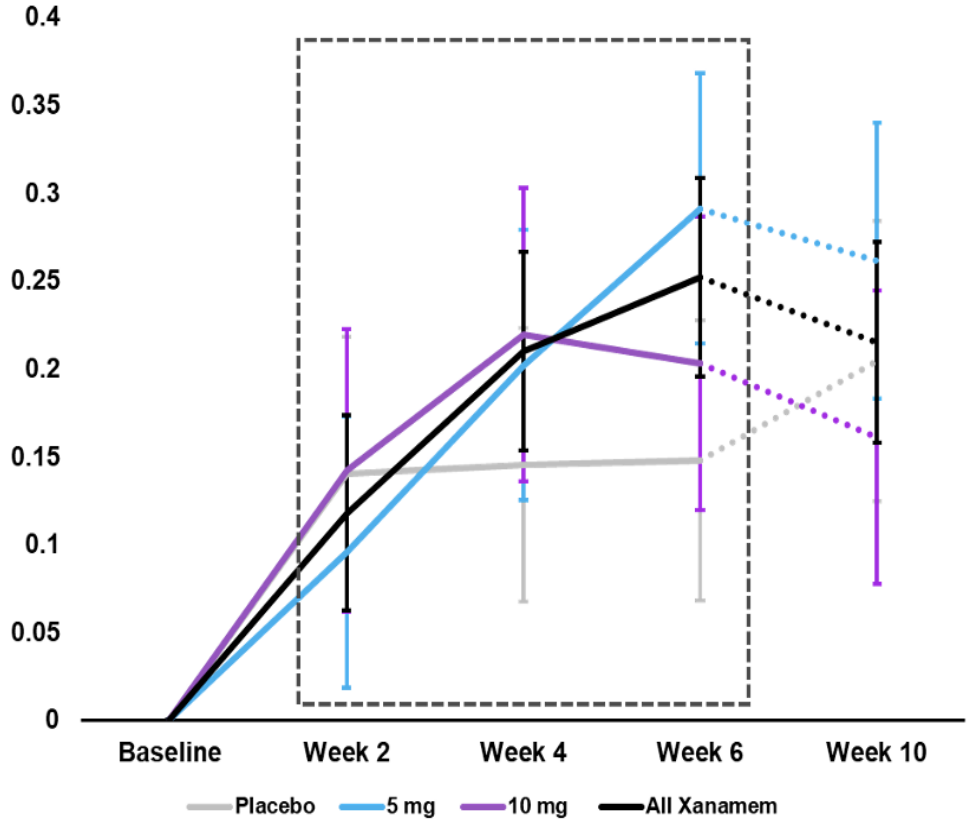
Plasma cortisol concentration pre- and post-dose over 12 weeks treatment with Xanamem or placebo.

Error bars represent +/- standard deviation.

Improvement in cognitive function (Attention Composite) in two clinical studies



XanaHES phase 1b trial (n=42)



XanaMIA-DR phase 1b trial n=107

Making a successful new medicine

- ✓ Hits the right target in the right part of the body
- ✓ Does not hit off-target systems
- ✓ Low potential for interaction with other drugs
- ✓ Simple dosing regimen
- ✓ Good safety profile
- ✓ Studied at the right dose

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Cognitive impairment in multiple neurologic & neuropsychiatric conditions - how Xanamem might aid treatment

Professor John Harrison PhD, PhD, CPsychol, CSci, AFBPsS

Chief Scientific Officer, Scottish Brain Sciences

Visiting Professor at the Institute of Psychiatry, Psychology & Neuroscience, King's College London, UK

Associate Professor at the Alzheimer Center, VUmc, Amsterdam, The Netherlands

Cognitive deficits in psychiatric disorders

Table 1 | **Main characteristics of cognitive impairment in psychiatric disorders, and a comparison with PD and AD***

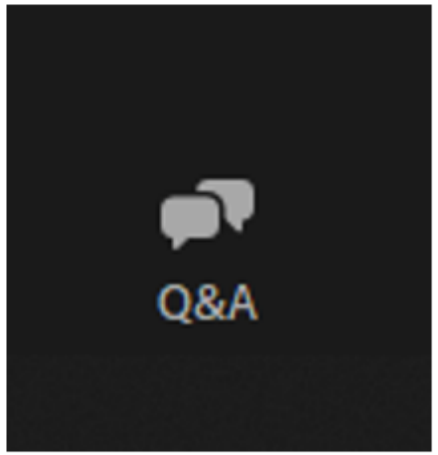
	Attention and/or vigilance	Working memory	Executive function	Episodic memory	Semantic memory	Visual memory	Verbal memory	Fear extinction	Processing speed	Procedural memory	Social cognition (theory of mind)	Language
Major depression	+(+)	++	++	++	+	+	+(+)	0/+?	++(+)	+	+(+)	+
Bipolar disorder	++(+)	++	++	++	+	+	++	+?	++	0	++	++
Schizophrenia	+++ M	+++M	+++M	+++	++	+(+)M	+++M	++	++M	+	+++M	+++
ASD	+++	+	+++	++	+	+	+(+)	+(+)	+ ++	0/+	+++	+++
ADHD	+++	++	+++	0/+	+	++	++	+	++	+	+	0/+
OCD	+++(↑)	+(+)	++	+	0/+	+	0/+	++	++	++	+	0/+
PTSD	+++(↑)	+(+)	+(+)	++	+	+	++(+)	+++	+	0	0/+	0
Panic disorder	+++(↑)	+	0/+	+	0/+	0/+	+	++	++	0	0	0
GAD	+	+	0	0	+	+	+	+	0	0	0/+	0
Parkinson's disease	++	++(+)	++	+	0/+	+	+	0?	+++	+++	+(+)	+(+)
Alzheimer's disease	+(+)	+(+)	+(+)	+++	+++	+++	++(+)	0?	+	+	+	++

* Cognitive deficits in the absence of treatment are depicted.

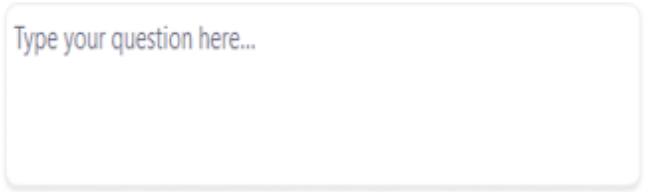
Millan MJ et al. (2012) Cognitive dysfunction in psychiatric disorders: characteristics, causes and the quest for improved therapy. *Nature*;11:141-168.

Online Q&A

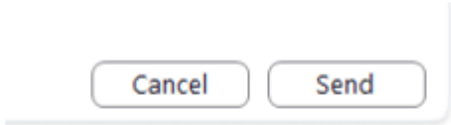
1. Click on the Q&A icon



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



3. Hit enter on your keyboard to submit your message



Appendix

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

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

Tau – a brain protein

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

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