ASX ANNOUNCEMENT

ACW Alzheimer's Disease biomarker study clinical results presentation slides

Sydney, 10 October 2022. Actinogen Medical ASX: ACW ("ACW" or "the Company") is pleased to release the attached presentation slides relating to the positive Phase 2a clinical data from its Alzheimer's Disease (AD) biomarker study announced this morning on the ASX platform.

Webcast today at 11am AEDT

Actinogen CEO Dr Steven Gourlay and CMO Professor Paul Rolan will present the slides at a **webcast at 11am AEDT today** to review the positive clinical results from the biomarker study.

Register for the webcast by clicking on the link below or copying it into a web browser:

https://us02web.zoom.us/webinar/register/WN__5Yufq4xQ4-upem9WPnHwQ

A full recording of the webcast will be made available on the Actinogen website www.actinogen.com.au as soon as practicable following the conclusion of the webcast.

ENDS

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Announcement authorised by the Board of Directors 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.

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 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. 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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Alzheimer's Disease biomarker study validates Xanamem® program

Positive Phase 2a clinical data

Dr. Steven Gourlay MBBS PhD MBA, CEO & MD

Professor Paul Rolan MD FRACP, CMO

10 October 2022

Authorised by the Board of Directors of Actinogen Medical Limited

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Actinogen

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CEO & MD

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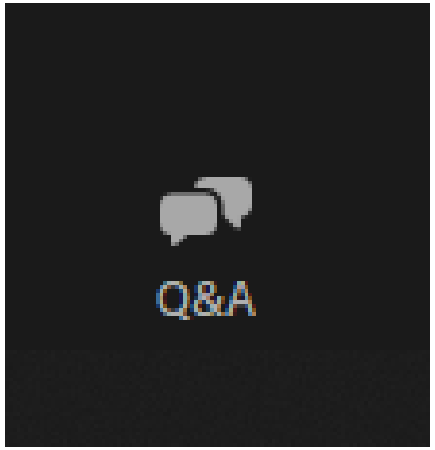
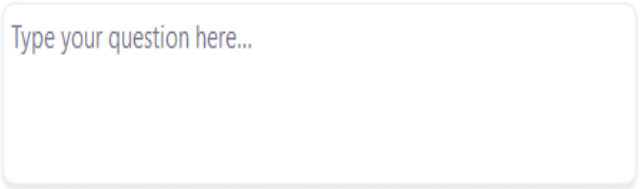
Online Q&A

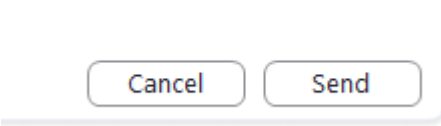
1. Click on the Q&A icon



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



3. Hit enter on your keyboard to submit your message



To contact support:
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Alzheimer's Disease biomarker study validates Xanamem® program

Positive Phase 2a clinical data

Dr. Steven Gourlay MBBS PhD MBA, CEO & MD

Professor Paul Rolan MD FRACP, CMO

10 October 2022

Authorised by the Board of Directors of Actinogen Medical Limited

Alzheimer's Disease (AD) key clinical findings

Actinogen

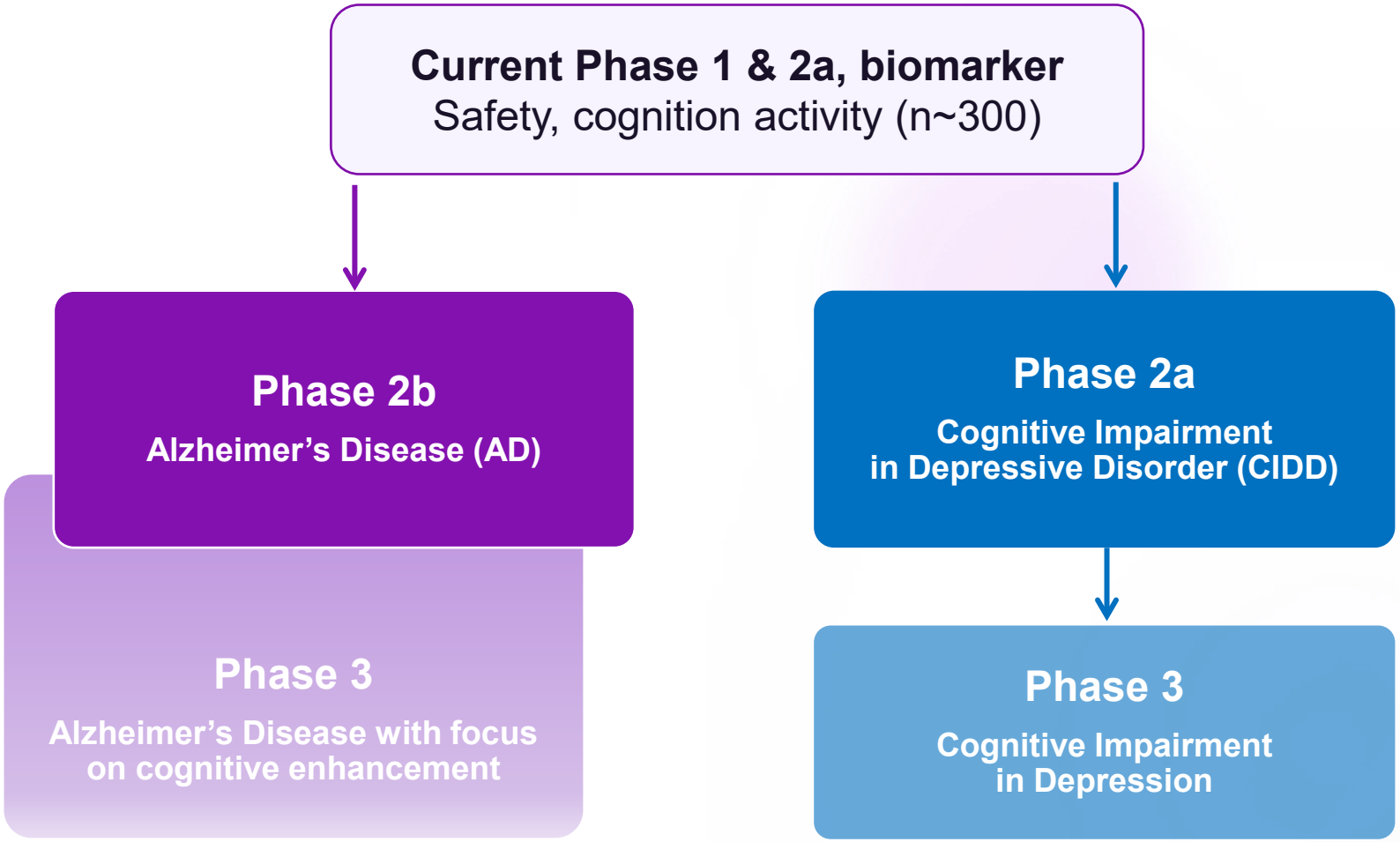
Major points

- Phase 2a placebo-controlled trial of Xanamem 10 mg vs. placebo over 12 weeks was re-run in 72 biomarker-positive patients with mild AD
- Used a pre-specified protocol & analysis plan to avoid bias
- Patients with elevated blood pTau showed a clinically significant Xanamem effect on the CDR-SB endpoint representing a 60% relative reduction in progression of disease
- CDR-SB effect of 0.6 – 0.8 points is larger than the 0.45 points reported recently for a successful Phase 3 trial of lecanemab[1] (amyloid antibody)
- Positive trends in a Neurologic Test Battery (NTB) of executive function & Mini Mental State Exam (MMSE)
- Regulatory path to approval clear and uncontroversial with FDA-approved CDR-SB
- Findings significantly de-risk and improve AD program efficiency

1. 28 September 2022 https://www.eisai.com/news/2022/news202271.html

Xanamem Phase 2 & 3 program

Building on four independent Phase 1 and 2 studies showing activity



AD biomarker study fundamentals

Assessment of efficacy in the Xanamem AD program is based on the proven use of clinical endpoints of cognition and function for regulatory approvals without reliance on biomarkers

However, as a supplement to clinical data, biomarkers have one major and two lesser potential uses:

Choosing patients with a disease likely to get treatment benefit

Confirming patients who have "real" AD and are likely to progress and thus have measurable treatment benefits

Confirming biologic activity & dose

Biologic activity at a given dose to indicate potential use as a surrogate endpoint with suitable validation

In regulatory submissions as "surrogate" endpoints prior to more definitive Phase 3 trials

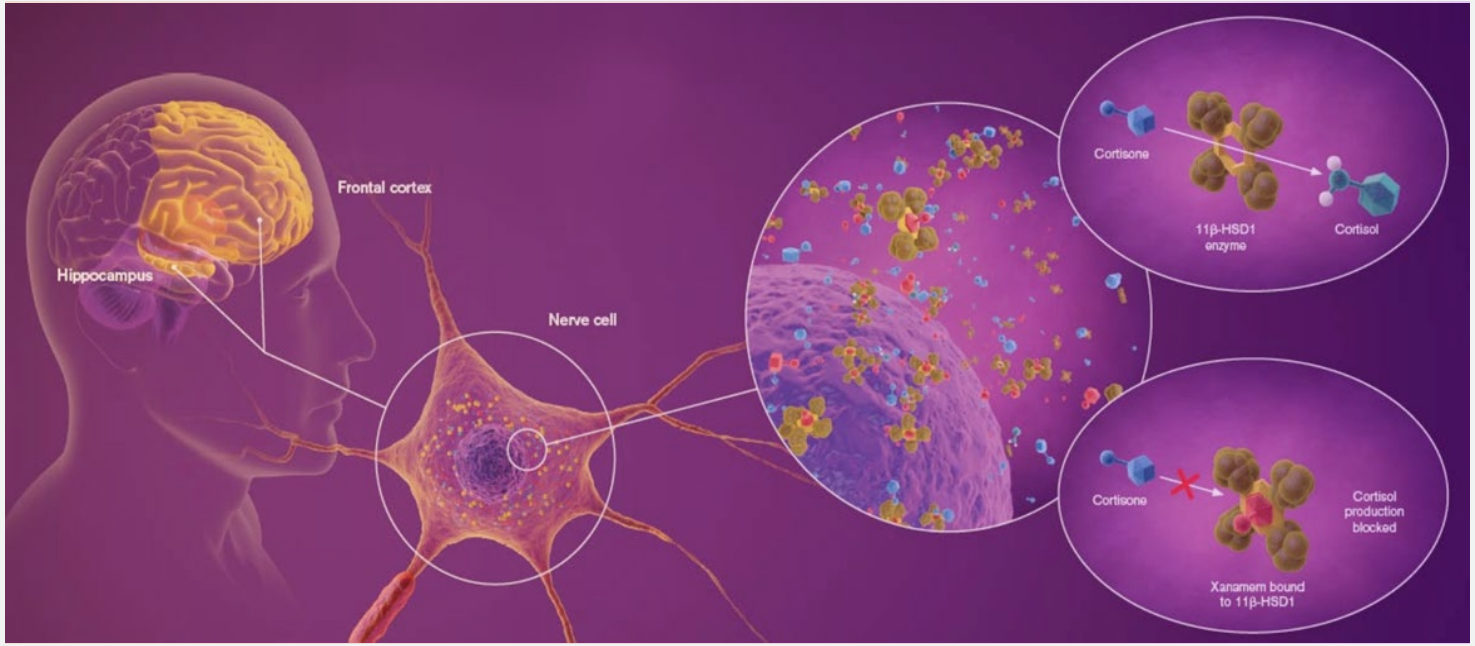
e.g. accelerated approvals to shave time off marketing approvals for serious diseases like cancer

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

Brain penetrant 11β-HSD1 small molecule enzyme inhibitor reduces cortisol inside brain cells - modulating signaling pathways and underlying disease processes[1,2]

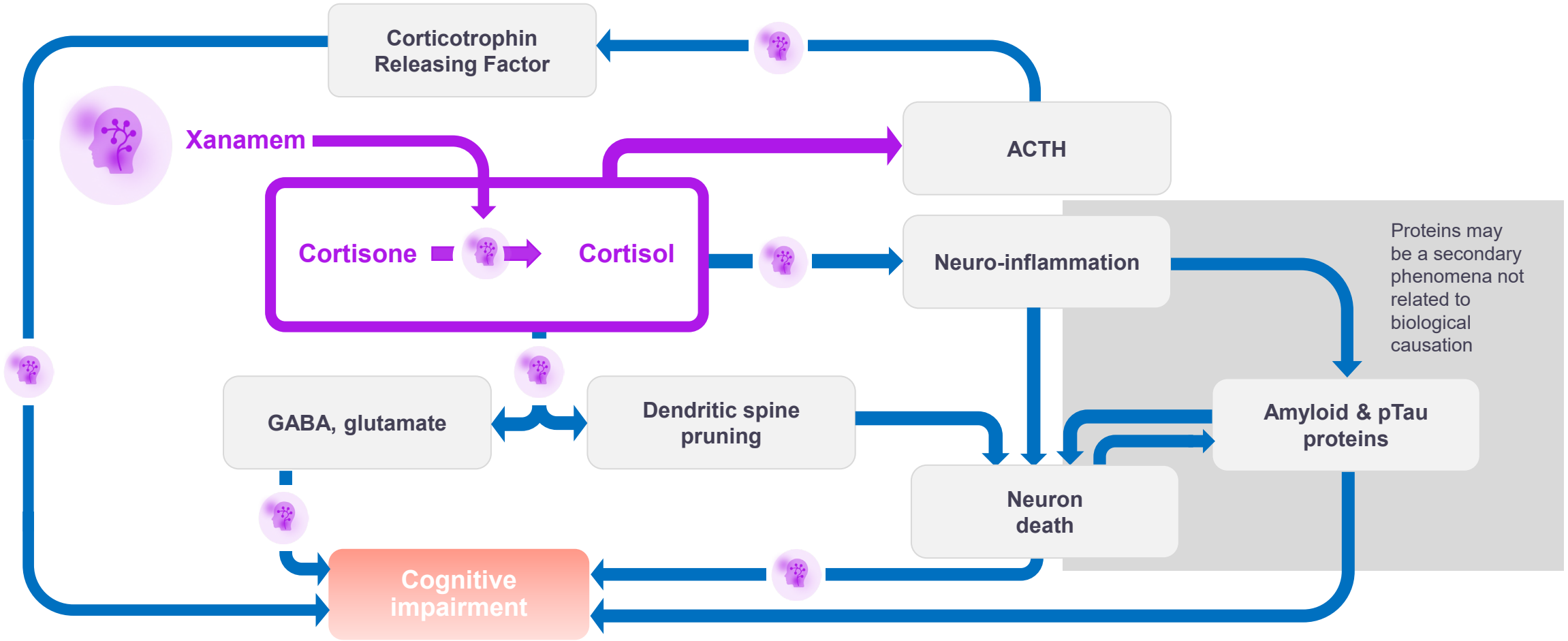
Potential to be:

- Rapidly cognitive enhancing
- Disease-modifying (slow or halt progression) in AD



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

Xanamem's mechanism is different to anti-amyloid antibodies



Previously: evidence of Xanamem activity in cognition from multiple sources

- ✓ **Protection against cognitive decline in animal model of AD using a Xanamem analogue independent of amyloid plaque**
- ✓ **Human PET scan study showing high levels of target binding at doses of 5 mg and above**
- ✓ **Consistent target engagement measured by ACTH response**
- ✓ **XanaHES trial in cognitively normal older volunteers – Cogstate attention & working memory**
- ✓ **XanaMIA trial in cognitively normal older volunteers – Cogstate attention & working memory**

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

A brief history of Alzheimer's Disease drug development

See also Actinogen Clinical Trials Science Forum 03 August 2022

https://youtu.be/Bm9ATZx1zEk

A small number of drugs to tackle cognition have worked and been approved in AD

Approved on clinical scales[1] in mild-moderate AD, 6-month trials

Mechanism	First Approval	Benefit	Comments
Oral anticholinesterase inhibitors e.g. donepezil, rivastigmine, galantamine	1996	Cognitive & functional scales	Gastrointestinal side effects Multiple trials
Oral NMDA inhibitors e.g. memantine, some in development	2003	Cognitive & functional scales	Gastrointestinal side effects Multiple trials
Oral oligomannate (GV-971, brown algae)	2019 China only	Cognitive & functional scales	Single positive trial

The primary Xanamem strategy is to follow this proven clinical pathway

1. Clinical scales measure how a patient feels, performs and functions

Clinical Dementia Rating – Sum of Boxes (CDR-SB) endpoint to assess dementia in early-stage AD

Test domain	Impairment				
	None	Questionable	Mild	Moderate	Severe
	0	0.5	1	2	3
Memory					
Orientation					
Judgment & Problem Solving					
Community Affairs					
Home & Hobbies					
Personal Care					

Score is sum of each line i.e. score between 0 and 18 (0 = normal)

Amyloid antibodies are “disease modifying” based on Positron Emission Tomography (PET) brain scan and CDR-SB

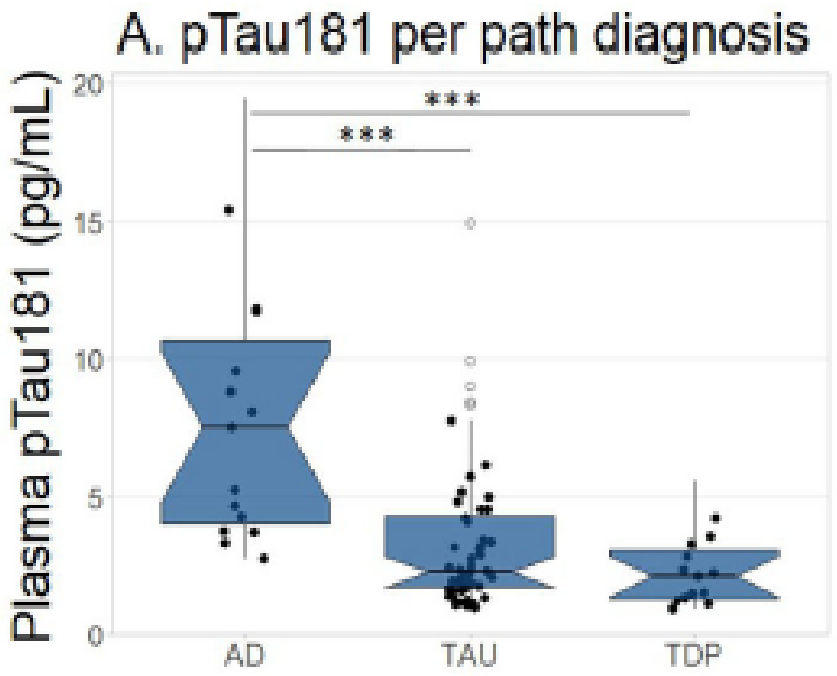
Use of blood and cerebrospinal fluid (CSF) levels is in development

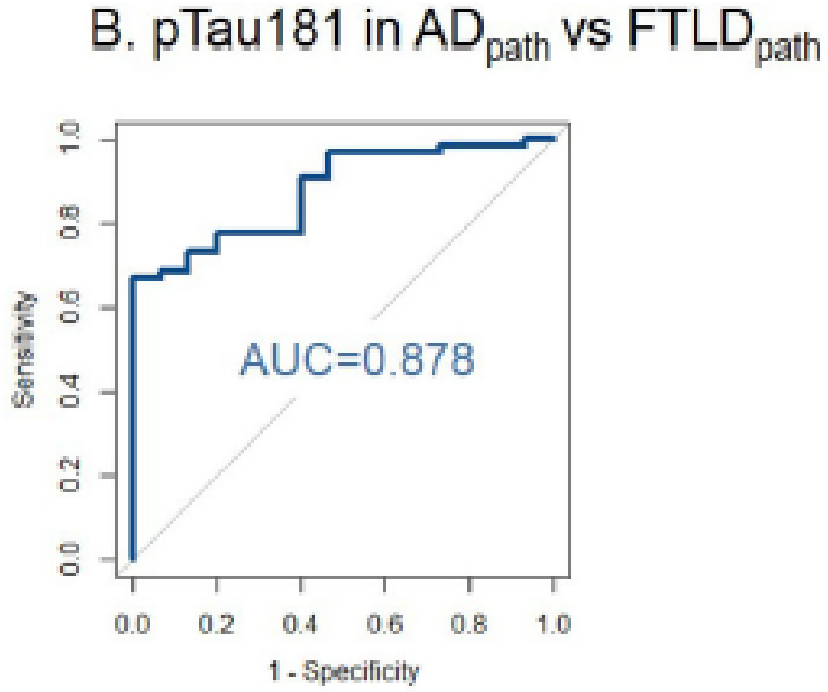
Predictive biomarker	How measured	Observations	FDA	Impact of drug treatment for target
Brain amyloid	PET scan	Aducanumab - major amyloid reduction[1]	✓	Clinically significant benefit **0.4 points** on CDR-SB
	PET scan	Lecanemab – under consideration by FDA	✓	Statistically and clinically significant benefit **0.45 points** on CDR-SB
CSF amyloid	Spinal tap	Part of ATN[2] diagnostic criteria for AD, prediction evolving		
Blood amyloid	Blood test	Promising as alternative to spinal tap for CSF or imaging		

CDR-SB is an accepted endpoint for pivotal trials in early-stage AD

1. Aduhelm US Product Information
2. ATN: Amyloid, Tau, Neurodegeneration

Elevated pTau181 diagnostic for AD type dementia[1]

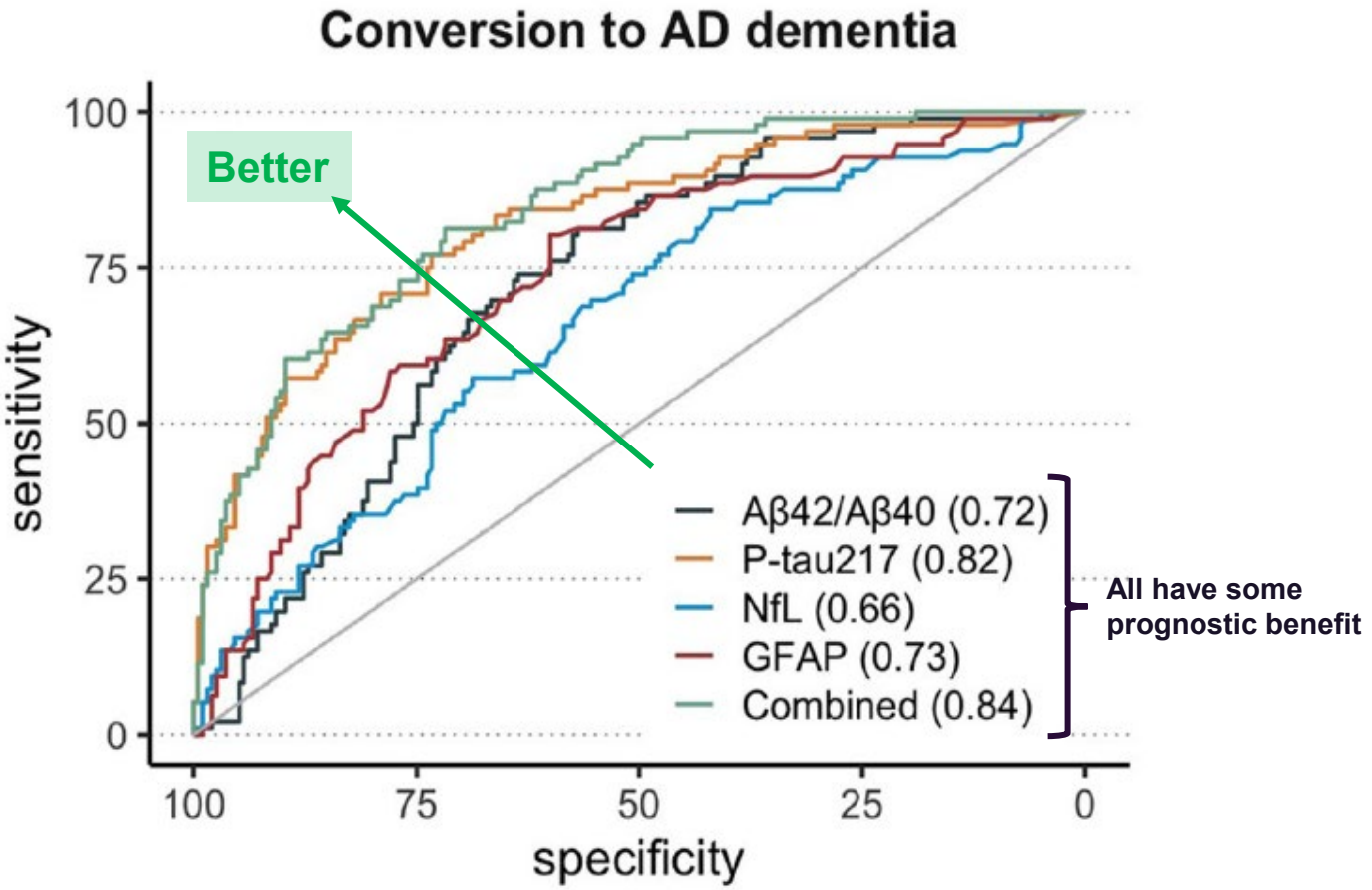




Blood pTau proposed to select patients with confirmed AD in Phase 2b

1. Data shown from Thijssen et al 2020; ADpath Alzheimer's Disease pathology diagnosis; FTLD Frontotemporal Dementia pathology diagnosis; TAU FTLD type dementia; TDP a different type of FTLD dementia

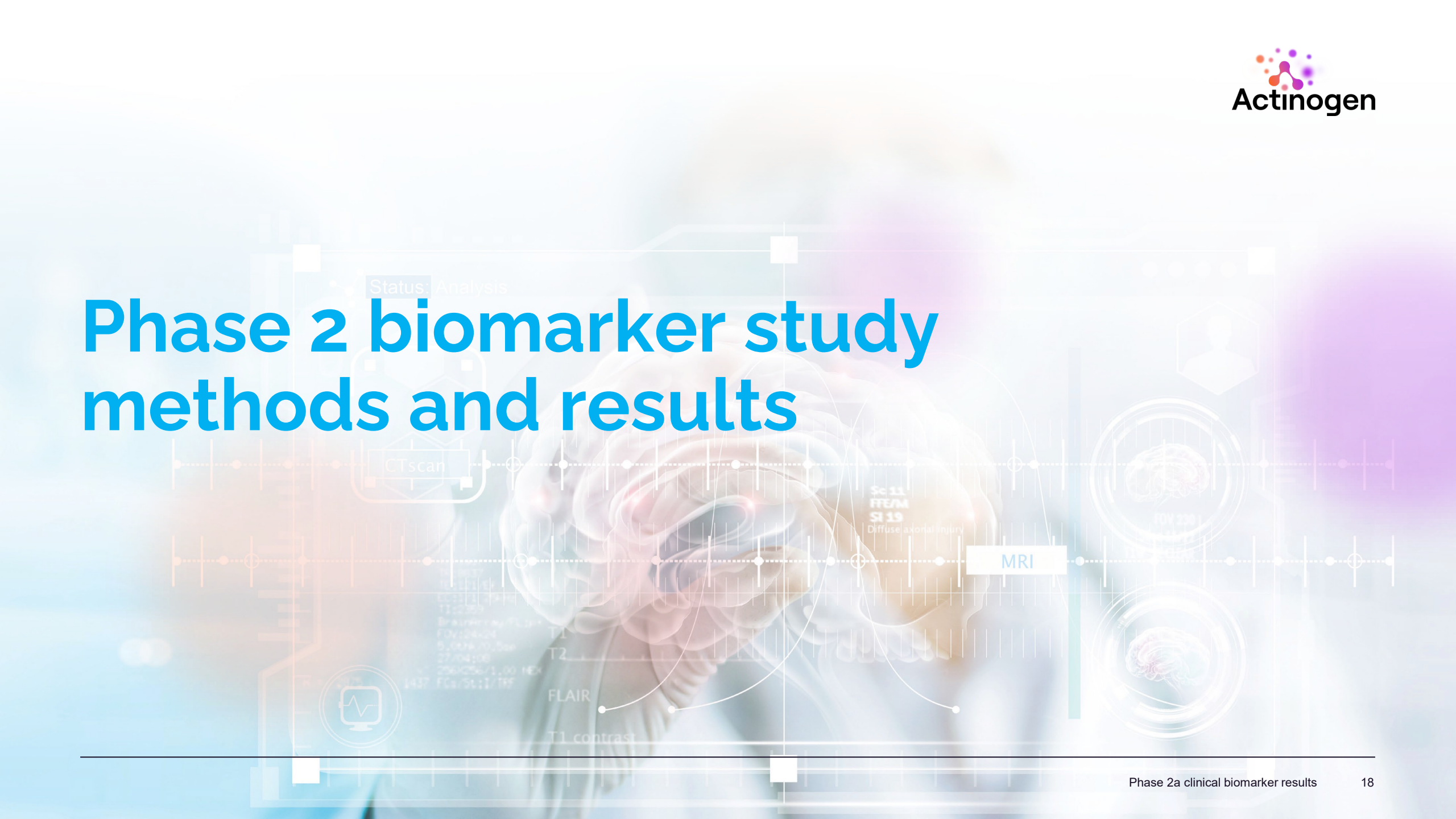
Elevated blood pTau is the single best predictor of progression in AD[1]



Blood pTau proposed to select AD patients likely to progress in Phase 2b

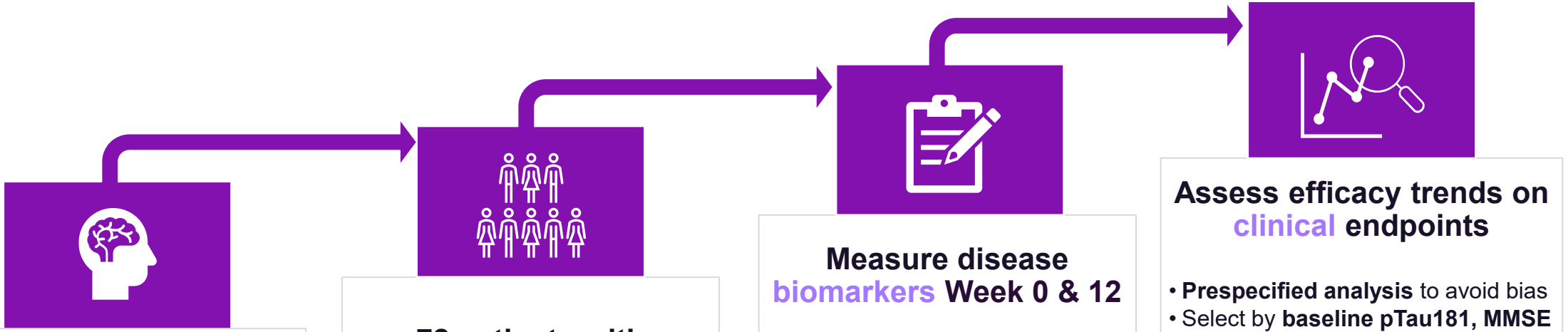
1. Data shown from Cullen et al. 2022; Thijssen et al 2020 showed similar findings for pTau181; similar findings Cullen et al. 2021, Aβ amyloid beta; P-tau phosphorylated tau protein; NfL neurofilament light (a nerve protein); GFAP Glial Fibrilliary Acidic Protein (only in brain)

Phase 2 biomarker study methods and results



Phase 2 blood biomarker study design & methods

Uses a pre-specified protocol and analysis plan



Phase 2a trial (XanADu)

- **185 people with mild AD** (MMSE 20-26)
- No amyloid imaging or biomarkers
- Placebo-controlled
- 12 weeks
- 10 mg Xanamem vs. placebo

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**
- **tTau (insufficient volume)**
- **NfL (insufficient volume)**

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**)

Creates valuable data relating to patient selection and efficacy in a mild AD population

Baseline characteristics of patients confirms similar populations

Slightly higher ADASCog14, CDR-SB & ADCOMS, lower MMSE in biomarker study

	Biomarker Study N=72	XanADu Phase 2a N=185
Age (mean, SD)	71 (8)	71 (8)
% female	54%	57%
ADASCog14 (mean, SD)	33 (8)	29 (9)
ADCOMS (mean, SD)	0.52 (0.19)	0.50 (0.20)
MMSE (mean, SD)	22 (3)	23 (3)
CDR-SB (mean, SD)	3.9 (1.6)	3.8 (1.7)
pTau pg/mL (mean, SD)	7.7 (6.8)	-
% pTau > 10.2 pg/mL	13%	
Aβ42/40 ratio (mean, SD)	21.9 (15.5)	-
GFAP pg/mL (mean, SD)	118 (73)	-

Study population is similar to overall "mixed" non-AD and AD Phase 2a population

Higher pTau (> 6.74 pg/mL[1]) subgroup shows clinically significant effect on CDR-SB (n=34)

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Group most likely to have pathological AD

Assessment	Desired change	Xanamem (n=16)	Placebo (n=18)	Cohen's d	p value
ADASCog14 total (mean)	Down	1.5	0.8	0.00	0.74
ADCOMS (mean)	Down	0.07	0.09	0.13	0.57
ADASCog14 167 units (mean)	Down	1.0	0.9	0.06	0.93
MMSE units (mean)	Up	-0.9	-1.2	0.16	0.80
CDR-SB units (mean)[1]	**Down**	**0.4**	**1.0**	**0.41**	**0.09**
CDR-SB units (median)	**Down**	**0.0**	**0.8**	**-**	**-**
NTB units (mean)	Up	0.5	-2.3	0.26	0.48
RAVLT units (mean)	Up	0.7	0.5	0.02	0.91
NPI units (mean)	Down	1.3	0.5	-0.18	0.42

Clinically significant effect size of CDR-SB 0.6 – 0.8 units

1. Median value in this study

Benefit on CDR-SB in both high pTau subgroups

Groups most likely to have pathological AD

	N	CDR-SB				
Group		Desired change	Xanamem	Placebo	Cohen's d	p value
pTau >6.74 pg/mL[1] (mean)	34	Down	0.4	1.0	0.41	0.09
pTau >6.74 pg/mL (median)	34	Down	0.0	0.8	-	-
pTau >10.2 pg/mL[1] (mean)	9	Down	0.1	0.8	0.62	0.33
pTau >10.2 pg/mL (median)[1]	9	Down	-0.3	0.5	-	-

Clinically significant effect size of CDR-SB 0.7 - 0.8 units in very high pTau group

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

Xanamem doubled rate of disease stabilization

Response analysis in pTau-positive patients

Twice as many patients in the Xanamem group had stable or improved disease compared with placebo[1]

56% of patients treated with Xanamem were stable or improved

0.6-point effect size represents a 60% relative reduction of disease progression vs. placebo

Xanamem protected the majority of patients from progression

1. Where CDR-SB decreased or was unchanged - Xanamem 9 of 16 (56%) vs. Placebo 5 of 18 (28%)

MMSE shows Xanamem effect measurable in more impaired dementia

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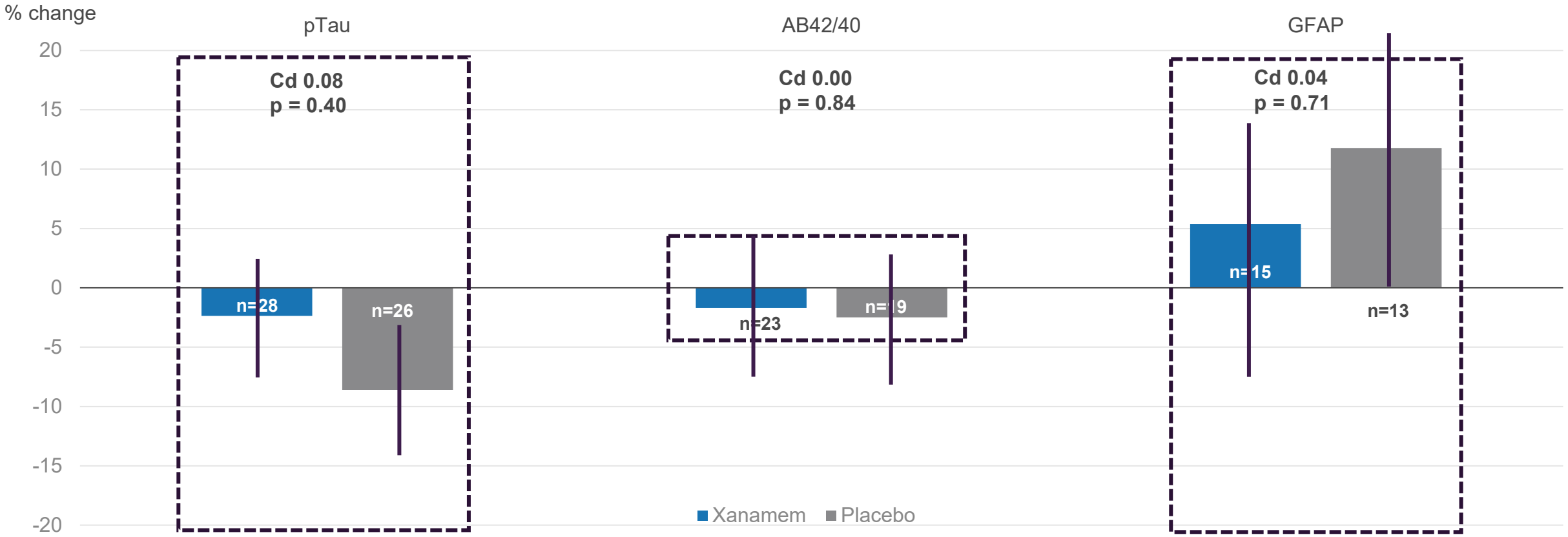
More clinically impaired subgroup

	N	MMSE score				
Group		Desired change	Xanamem	Placebo	Cohen's d	p value
MMSE 20-23 (mean)	46	Up	1.7	-0.3	0.93	0.02
MMSE 20-23 (median)	46	Up	2.0	-1.0	-	-

Clinically & statistically significant effect size of MMSE +2.0 to 3.0 units

No change observed over 12 weeks consistent with non-amyloid mechanism

10mg vs. placebo, mean (standard error bars), published test-retest range in boxes



Longer duration studies needed to evaluate potential disease modification effects

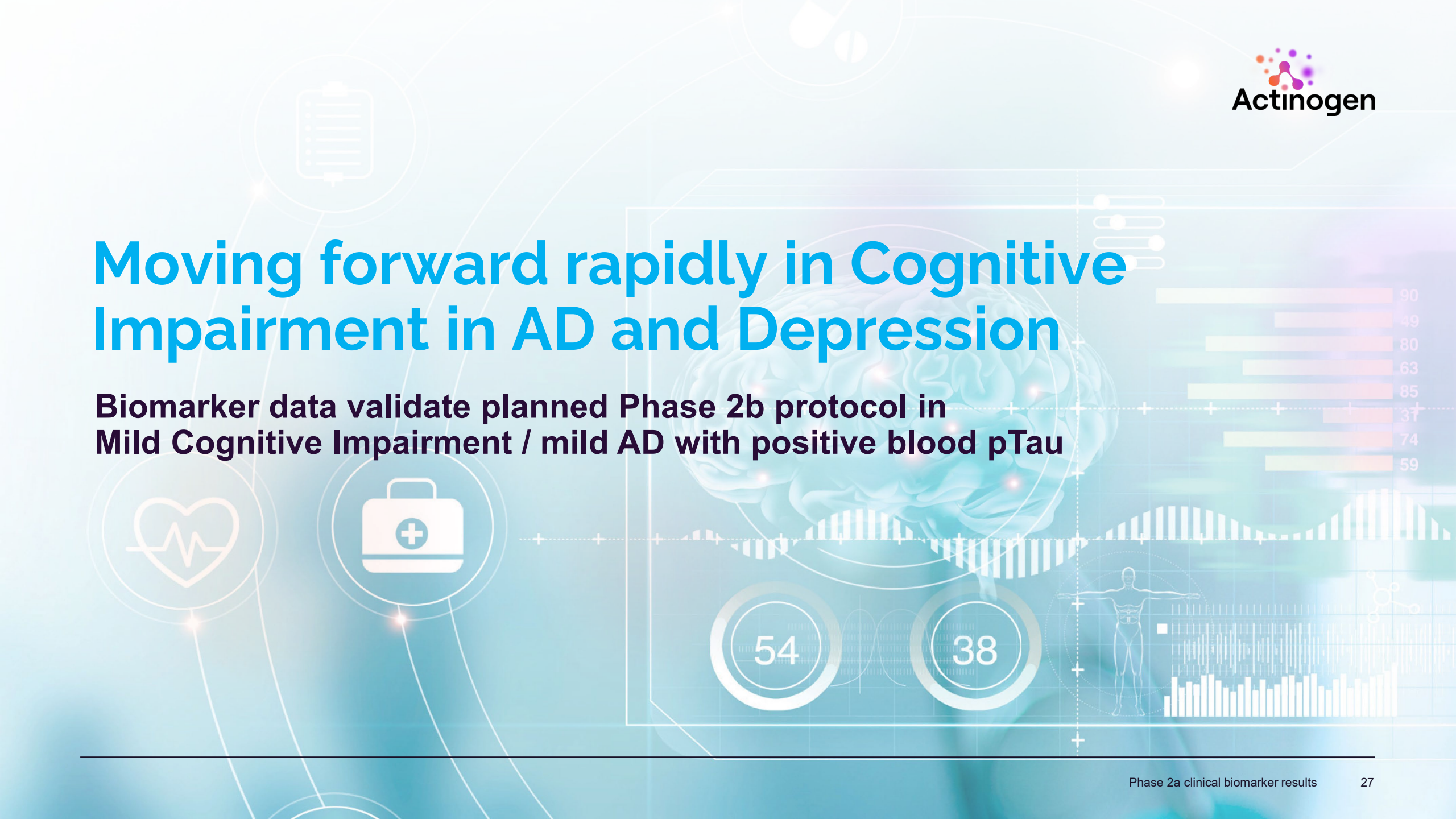
Efficacy conclusions

This analysis validates and de-risks the AD program by showing:

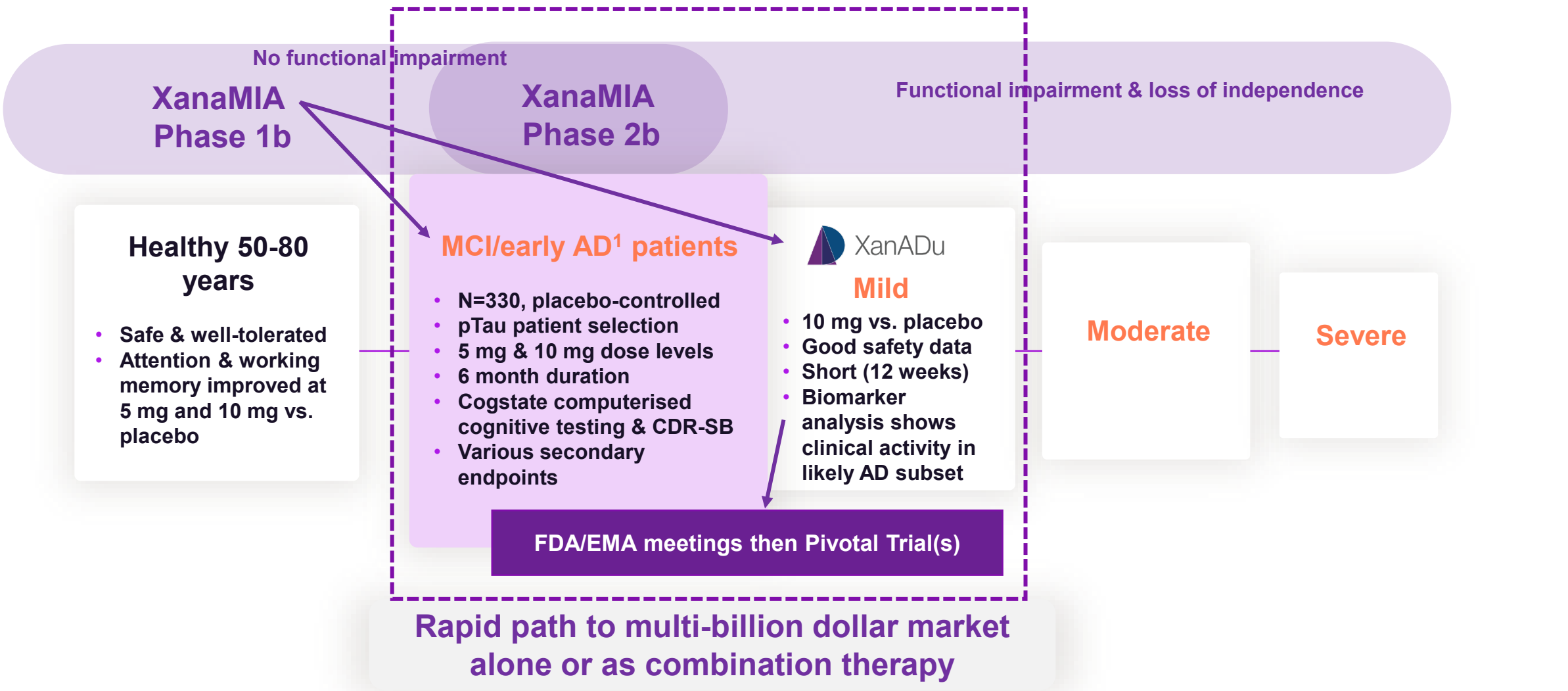
- ✓ **Clinical activity of Xanamem in AD patients**
- ✓ **Large clinical effect size**
- ✓ **Utility of blood pTau levels to select suitable patients for next Phase 2b trial**
- ✓ **Utility of CDR-SB to measure the benefit of Xanamem in future trials**
- ✓ **Complements positive prior trial findings on cognition**

Moving forward rapidly in Cognitive Impairment in AD and Depression

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

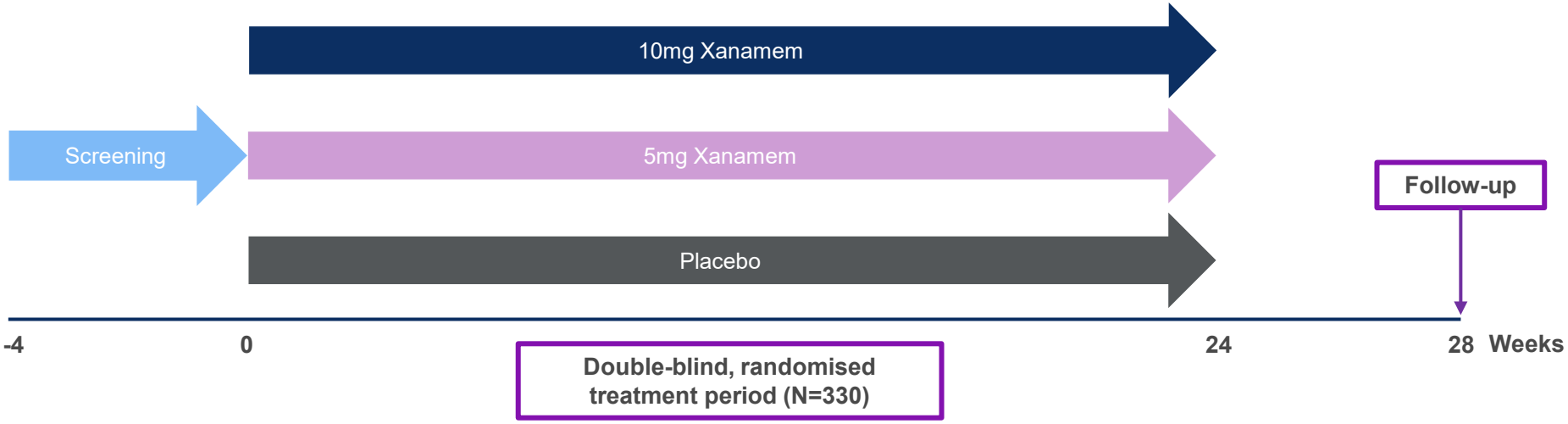


Focus on speed to market as a cognitive enhancing treatment



1. Mild Cognitive Impairment (MCI): memory, executive function deterioration with retained functional abilities; very mild AD with some functional impairment

XanaMIA Phase 2b trial design & implementation model: selecting AD patients by blood pTau level



Key inclusion/exclusion criteria	Primary Endpoints	Key Secondary Endpoints	Key Implementation Features
• Clinical diagnosis of **MCI or mild dementia** due to AD (NIA-AA) • Elevated blood **p-tau181** • Cognitive impairment relative to demographic norms • Excluded vascular cause of dementia	• **CDR-SB** • **Cogstate CTB attentional composite** (attention and working memory)	• **Amsterdam Activity of Daily Living** scale • Cogstate **Executive Function & Episodic Memory** Function Composites • Individual tests • Carer questionnaire / Patient Global Improvement	• **Australian trial sites plus selected international locations** • **Actinogen "hands-on" operational model** • Optimized for scalable addition of international sites as required

iDSST=international Digit Symbol Substitution Test - Symbols ; IQCODE=Informant Questionnaire on Cognitive Decline in the Elderly; MCI=mild cognitive impairment; NIA-AA=National Institute of Aging - Alzheimer's Association; RBANS=Repeatable Battery for the Assessment of Neuropsychological Status;

The Xanamem opportunity in depression

Current anti-depressants

work slowly (3 weeks) and initial suicide risk

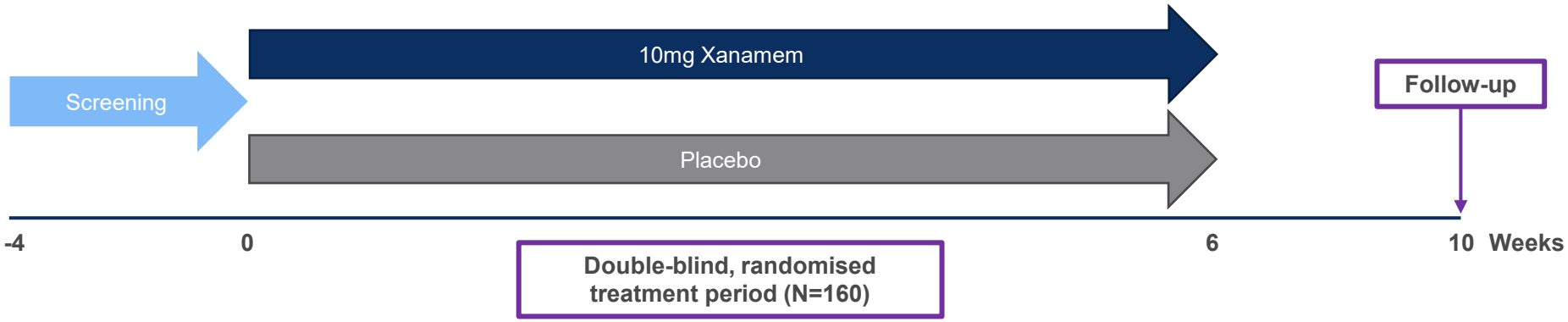
do not target cognition

multiple adverse effects
blood pressure, sexual function, appetite…

Xanamem improves cognition quickly

Xanamem may improve both depression and cognitive impairment

XanaCIDD trial design & implementation model



Key inclusion/exclusion criteria	Primary Endpoints	Key Secondary Endpoints	Key Implementation Features
• Primary diagnosis of **MDD** • **Persistent depressive symptoms despite existing therapy** • **Cognitive impairment** relative to demographic norms	• **Cogstate CTB attentional composite** (attention and working memory)	• Montgomery-Åsberg Depression Rating Scale (**MADRS**) • **Executive Function** Cognitive Composite • **Memory Function** Cognitive Composite	• **Australian trial sites** • **Actinogen "hands-on" operational model** • **First patient enrollment planned for 2022**

Xanamem's unique value proposition

Many other opportunities such as other types of dementia and neuropsychiatric conditions

Actinogen has the only non-amyloid mechanism drug in development with credible cognitive data

It is one of only three worldwide AD clinical trial programs for an oral medication with credible cognitive activity data

Mechanism	Stage	Cognitive endpoint	Company market cap. ($AU)[1]
Oral Xanamem to lower brain **cortisol** (**Actinogen**)	Phase 2	Cognitive test battery, **CDR-SB, NTB**	170 million
Oral inhibition filamin A to stop Aβ42 **amyloid** signal to TLR/α7nicR (**Cassava** Sciences)	Phase 3	Cognitive test battery	2.5 billion
Oral QPCT inhibitor to prevent toxic **amyloid** formation (**Vivoryon**)	Phase 2	Cognitive test battery	315 million

Xanamem is unique with 3 independent trials now showing cognitive and clinical benefits relevant to AD and other diseases

1. at 9 Oct 2022

Xanamem Strategy & Timeline

Actinogen strategy validated by new results

Accelerate clinical development

- ***Focus on cognitive enhancement***:
  - **Patients with early Alzheimer's Disease**
  - **Use pTau for patient selection**
  - **Phase 2b will use commercial tablets**
  - **Cognitive enhancement Depression** Phase 2
  - Trial **operations based in Australia** and selected other countries

Forward planning

- Scale up and optimise **manufacturing** to prepare for commercially viable, large scale production
- **Ancillary clinical and nonclinical** studies
- **Commercial** planning

Create value from partnerships

Pharma/biotech engagement

- **Actively engage large and mid-size potential partners with new results**
  - Seek value-add partnerships
  - Evaluate regional opportunities

Regulatory engagement

- Seek early **US FDA and EMA** interactions to agree endpoints for pivotal, approvable trials in AD

Xanamem timeline & catalysts

Actinogen

2022

- October **multiple partnering presentations of new results**
- November **CTAD XanaMIA presentation**
- Trial **enrollment starts for XanaCIDD** trial in Depression
- Key global **regulatory submissions** in AD with FDA, EMA, other
- Phase 2b XanaMIA **AD trial preparation**

2023

- **Partnering discussions**
- **XanaMIA Phase 2b** enrollment starts H1
- **XanaCIDD** enrollment ± results
- **Presentations & publications**

2024

- XanaMIA **Phase 2b results**
- **Expand Cognitive/Depression** program
- **Expand Alzheimer's Disease** program

Eminent world-leading authority on dementia, Associate Professor Michael Woodward, commented:

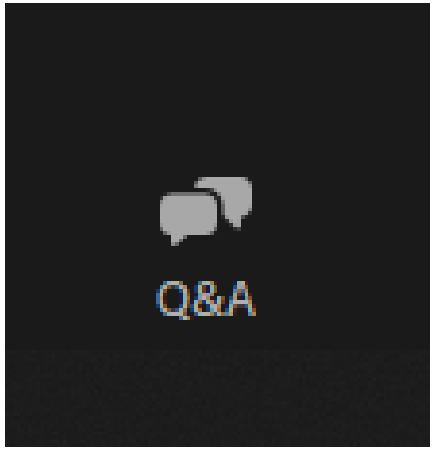
"The positive data for CDR-SB and other endpoints are encouraging and indicate a likely therapeutic effect of Xanamem in patients with the early stages of Alzheimer's Disease.

The use of pTau blood levels to confirm the diagnosis of Alzheimer's Disease in future trials represents a practical and efficient method to select patients at risk of disease progression and in whom a treatment effect is more likely to be observed."

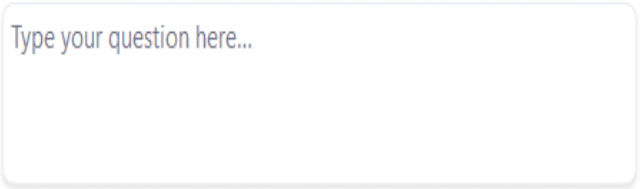
Questions

Online Q&A

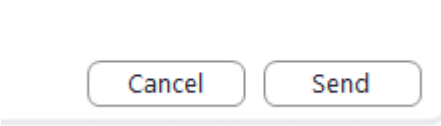
1. Click on the Q&A icon



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

Thank you

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Appendix

Xanamem Clinical Development Pipeline

	Phase 2 Pathway	Outlook
Cognitive impairment in early **Alzheimer's disease**	**Biomarker/efficacy analysis in** patients with mild AD - Phase 2 dataset of 10mg vs. placebo over 12 weeks, October 2022 **Cognitive benefit** in patients with early stages of AD - Phase 2 XanaMIA Part B commencing H1 2023	*"Big-to-market"* *Multiple Phase 2b/3 trials*
Cognitive Impairment in Depressive Disorder	**Depression and cognitive impairment placebo-controlled trial** - Phase 2 XanaCIDD trial to commence in Q4 2022	*Potential to treat both depression and related cognitive impairment*
Anxiety, sleep & behavioural problems in **Fragile X Syndrome**	**XanaFX (open IND with Phase 2 protocol)** - Phase 2 proof-of-concept in adolescent and young adult males	*Pending alternative funding or partnership*

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

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

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

Filamen A a protein believed to relate to amyloid toxicity

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