



ASX ANNOUNCEMENT

Actinogen CEO & CMO present at Sachs Neuroscience Innovation Forum & JPM Week meetings

World Health Organization grants new and unique nonproprietary name 'emestedastat' for Xanamem[®]

Sydney, 13 January 2025. Actinogen Medical ASX: ACW (“ACW” or “the Company”) is pleased to announce that its Chief Medical Officer, Dr Dana Hilt, and CEO, Dr Steven Gourlay, will present today at the Sachs Associates 8th Annual Neuroscience Innovation Forum in San Francisco (12 January USA PST). Drs Hilt and Gourlay are joined in San Francisco by ACW Chief Financial Officer, Will Souter, and Chief Commercial Officer, Andrew Udell.

The Sachs Forum brings together large and small pharmaceutical companies, analysts and investors who are focused on neuroscience as a key component of their pipelines and portfolios. The audience includes - buy and sell side analysts from investment banks and funds, along with partnering executives from pharma, biotech, medtech, neurotech, and diagnostics companies.

While in San Francisco, the ACW team will participate in a significant number of partnering, analyst and investor meetings associated with the 43rd Annual J.P. Morgan Healthcare Conference from January 13 to 16, 2025 (JPM Week).

The information used for all Sachs Innovation Forum and JPM Week presentations and meetings is attached to this announcement or has been previously announced.

The attached presentation recaps the success of emestedastat (Xanamem), ACW's novel once daily oral treatment for Alzheimer's disease (AD) and major depressive disorder (MDD) and examines the near-term major phase 2b/3 readouts in the XanaMIA AD trial in 2025 and 2026.

New and unique nonproprietary name of 'emestedastat' granted for Xanamem

The Company is also pleased to announce that the World Health Organization (WHO) has granted the nonproprietary name 'emestedastat' to Actinogen for its Xanamem¹ once-a-day small molecule in accordance with the WHO's Procedure for the Selection of Recommended International Nonproprietary Names (INN) for Pharmaceutical Substances.

An INN is a unique, globally recognized name for a pharmaceutical drug or active ingredient. The WHO established an INN expert committee to select and assign a single, unique name of worldwide acceptability for each active substance that is to be marketed as a pharmaceutical, ensuring clear identification, safe

[®] Xanamem is a registered trademark of Actinogen Medical Limited

¹ Also known as UE2343 which is the original identifier used by the University of Edinburgh

prescription and dispensing of medicines to patients. Nonproprietary names are intended for wide use ranging from labelling and product information to drug regulation and scientific literature.

By granting this INN, the WHO has recognized emestedastat (Xanamem) as the first drug to be named for the class of enzyme inhibitors of 11 β -HSD1 by assigning it the unique suffix of '-stedastat' pertaining to its mechanism of action on 11 β -HSD1.

Emestedastat (Xanamem) is a unique orally administered molecule in its own class as a 'brain tissue cortisol synthesis inhibitor.' It is currently being developed as a promising new therapy for the treatment of Alzheimer's disease and depression. Its novel mechanism of action is to control the level of the 'stress hormone' cortisol in the brain through the inhibition of the cortisol synthesis enzyme, 11 β -HSD1. The Company is currently conducting a phase 2b/3 trial of emestedastat in Alzheimer's disease and is planning for future depression trials.

Dr Steven Gourlay, Actinogen's CEO and Managing Director, said:

"It is pleasing to have emestedastat (Xanamem®) recognized as first-in-class with the award of an INN name with a new and unique suffix. The suffix used in the name highlights Actinogen's leading position in the field of 11 β -HSD1 enzyme inhibition which is designed to control brain cortisol and result in clinically meaningful benefits for patients with Alzheimer's disease (AD) and major depressive disorder (MDD).

"Meanwhile, in San Francisco this week, we are focused on briefing potential pharmaceutical partners on the significant progress of our XanaMIA phase 2b/3 AD trial and the seminal proof-of-concept data obtained from the XanaCIDD phase 2a trial in patients with MDD.

"The clinical benefits seen in phase 2 trials with the 10mg daily dose of emestedastat in both AD and MDD give us confidence that the outcomes of current and future trials in these and other diseases will be positive."

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.

Current Clinical Trials

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

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 its ability to slow progression of Alzheimer's disease is assessed with a variety of endpoints. The primary endpoint of the trial is the internationally-recognized CDR-SB (Clinical Dementia Rating scale – Sum of Boxes). The trial is being conducted in Australia and the US. Initial results from an interim analysis of the first 100 participants are anticipated in Q3 2025 and final results H2 2026.

About Xanamem

Xanamem's novel mechanism of action is to control the level of cortisol in the brain through the inhibition of the cortisol synthesis enzyme, 11 β -HSD1, without affecting production of cortisol by the adrenal glands. Xanamem is designed to get into the brain after it is absorbed in the intestines upon swallowing.

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

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

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

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



Actinogen

Oral emestedastat¹ (Xanamem[®]/UE2343)

***Controlling brain cortisol to slow progression in Alzheimer's disease
and treat major depressive disorder***

**Sachs Neuroscience Innovation Forum & JP Morgan Healthcare week
January 2025**

1. Emestedastat is the new nonproprietary or generic name for Xanamem allocated by the World Health Organization recognizing the “-stedastat” suffix as a unique, new identifier for 11 β -HSD1 inhibitors

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Overview



Emestedastat is now significantly de-risked



Novel 11 β -HSD1 cortisol control mechanism, oral, attractive safety profile

- Brain cortisol has long been proposed as a pathogenic mechanism in major depressive disorder (MDD) and Alzheimer's (AD)
- Unique brain-penetrant tissue cortisol synthesis inhibitor that leaves adrenal cortisol synthesis unaffected
- Approximately **400 people** treated to date with excellent safety profile and low drug interaction risk



Positive phase 2 clinical data in both indications

- **Phase 2a MDD trial showing clinically & statistically significant activity - benefits across multiple endpoints**
- Disease-modifying activity on CDR-SB in AD phase 2a trial in biomarker-positive AD patients 0.6 points over 12 weeks
- Positive data from both trials read through to other indications in psychiatry and the dementias



Patent & data exclusivity protection and manufacturing

- Composition of matter protection to 2031 – to 2036 with extensions in major markets, new chemical entity data protections
- **Additional recent patents protect further** against future competition e.g. use patent for MDD
- **Manufacturing process scaled up and patented**, contractors Asymchem (China) & Catalent (US)



Large clinical and commercial opportunities

- **No other brain-penetrant cortisol control molecules are in development**
- Anti-depressant market is currently ~\$20 billion, with major opportunities for novel mechanisms & better-tolerated drugs
- Alzheimer's market likely to be \$20 billion by 2030, with major opportunity for a safe & effective oral agent

Experienced board and management team

Board of Directors



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Chairman
MBBS; MBA



Dr. Steven Gourlay
CEO & MD
MBBS; FRACP; PhD; MBA



Mr. Malcolm McComas
Non-Executive Director
BEC, LLB; FAICD; SF Fin



Dr. George Morstyn
Non-Executive Director
MBBS; PhD; FRACP CD



Dr. Nicki Vasquez
Non-Executive Director
PhD



Management Team



Dr. Steven Gourlay
CEO & MD



Dr. Dana Hilt
Chief Medical Officer
MD



Will Souter
Chief Financial Officer
BComm, LLB



Andrew Udell
Chief Commercial Officer
MBA



Cheryl Townsend
VP Clinical Operations
RN, M Health Law



Fujun Li
Head of Manufacturing
PhD



Michael Roberts
Head of IR & Comms
B.Ec (Hons), CPA, FFIN



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Phase 2 activity in AD and MDD validates cortisol MoA

Phase 2b/3 underway in AD, preparing phase 2b in MDD

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Evidence of durable benefit on depression from control of brain cortisol validates the emestedastat program in terms of:

- a. Clinical proof-of-concept for the “CNS cortisol control” MoA
- b. Utility of the 10 mg once-daily dose used in Alzheimer’s phase 2b/3 trial
- c. Utility of the 10 mg once-daily dose for the next MDD trial

Phase 2a MDD efficacy and safety data support further clinical development in MDD

Clinical activity on depression supports the likelihood of seeing a disease-modifying effect in Alzheimer’s disease over 36 weeks in current XanaMIA trial

Emestedastat – Development Pipeline



Indication	Preclinical	Phase 1	Phase 2	Phase 3	Next Milestone
Alzheimer's disease	On-going phase 2b/3			Open INDs	Results 25-26
Major depressive disorder	Phase 2a complete, Phase 2b/3 in planning				FDA Type C in Q1 25
Fragile X syndrome	Phase 2a on hold				On hold
Bipolar disorder	[Dotted arrow]			Potential next indications	
PTSD	[Dotted arrow]				
Lewy-body dementia	[Dotted arrow]				
Frontotemporal dementia	[Dotted arrow]				

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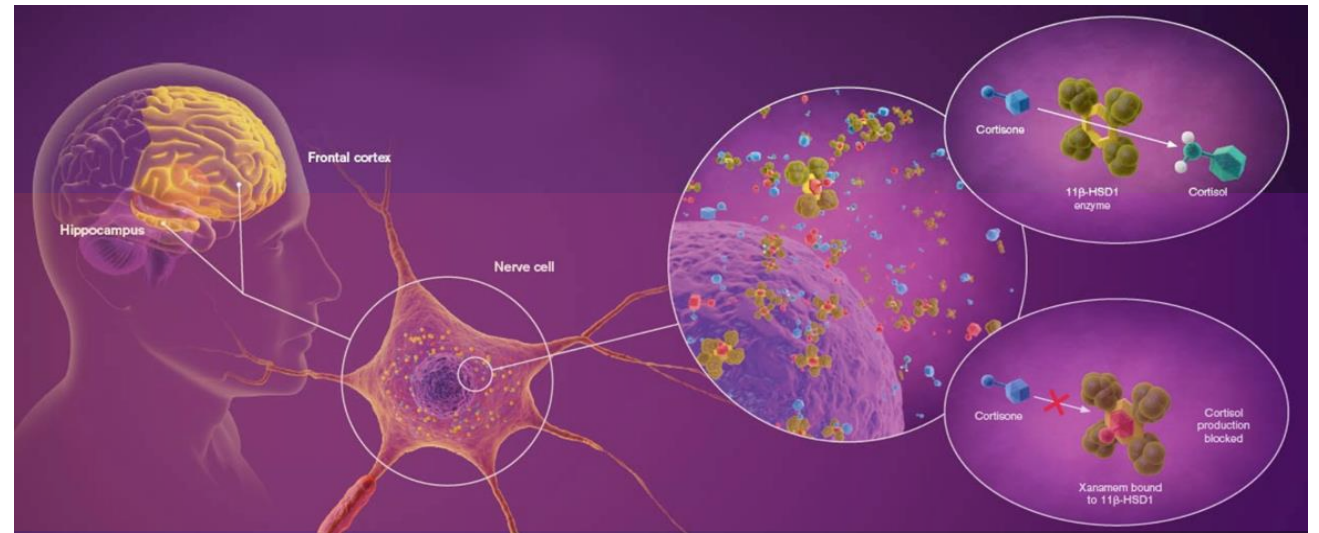
Emestedastat



Once-daily oral treatment with a unique mechanism

Small molecule tissue cortisol synthesis inhibitor (11 β -HSD1 enzyme)

- ✓ Good safety profile in ~400 treated
- ✓ Brain-penetrant at low doses
- ✓ Potentially disease-modifying in AD
- ✓ Anti-depressant activity in phase 2
- ✓ Low drug interaction potential ideal for combination therapy



Mouse experimental studies, brain cortisol levels & human clinical trials validate cortisol as a target for the treatment of AD

Emestedastat and CNS 11β -HSD1 inhibition¹

Controlling brain cortisol² has potential durable benefits

Reduction of the ‘stress’ response in brain

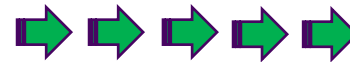
RAPID changes in kinases, cell function, neurotransmitters over hours to days lead to short-term “low stress” settings



“Lower stress” shorter term e.g.

- Reducing inflammation
- Improving neurotransmitter balance
- Decreasing cell death

SLOW changes in gene expression and protein synthesis over days to weeks lead to durable “low stress” settings

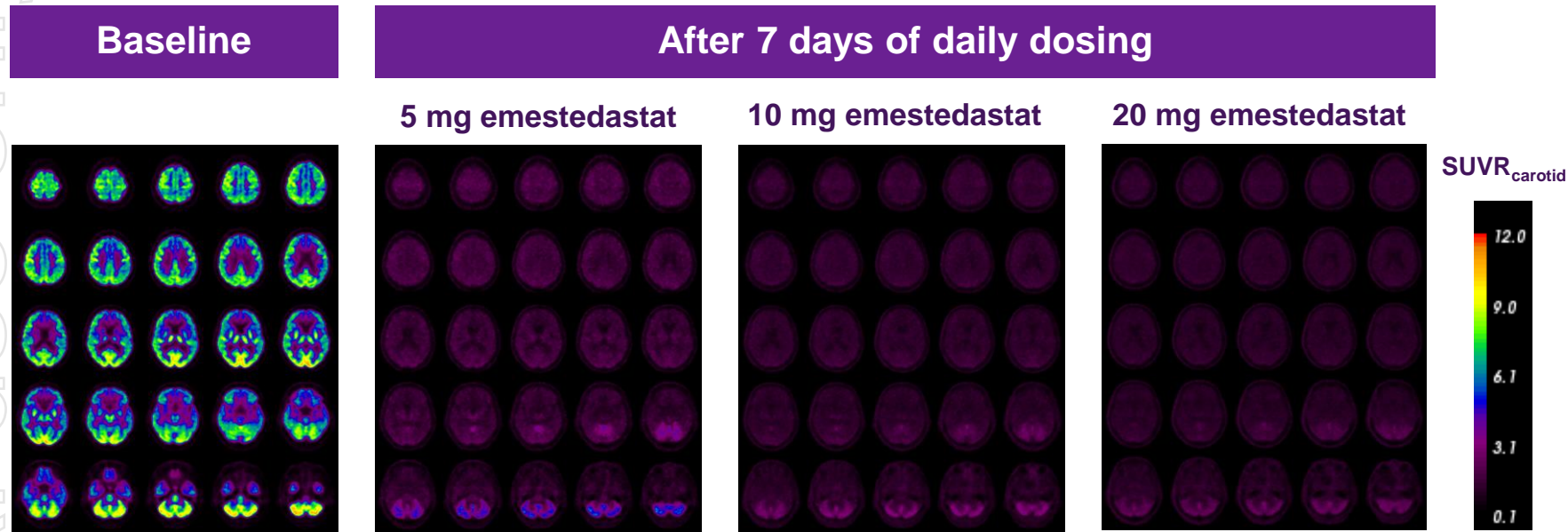


“Lower stress” longer term e.g.

- Improving neural circuitry
- Generating new brain cells
- Ideal receptor configurations

Human PET study shows full target engagement

Other 11 β -HSD1 enzyme inhibitors have not achieved adequate brain levels



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

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

Journal of Alzheimer's Disease 97 (2024) 1463–1475
 Brain 11-Hydroxysteroid Dehydrogenase Type 1 Occupancy by Xanamem™
 Assessed by PET in Alzheimer's Disease and Cognitively Normal Individuals
 Victor L. Villemagne, Vincent Dor, Lee Chong, Michael Kassiou, Rachel Mulligan,
 Azadeh Feizpour, Jack Taylor, Miriam Roesner, Tamara Miller and Christopher C. Rowe

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Alzheimer's disease program

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Alzheimer's disease

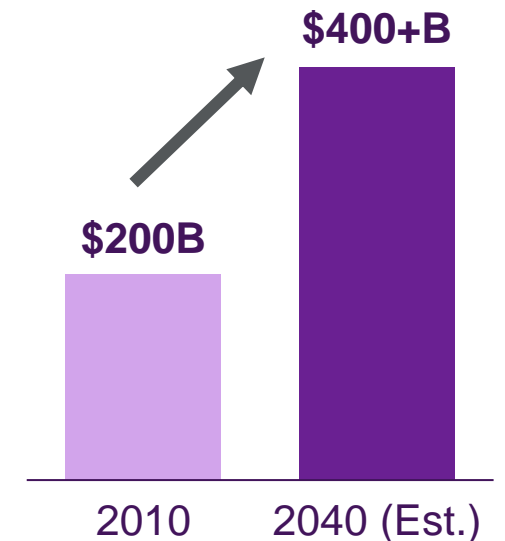
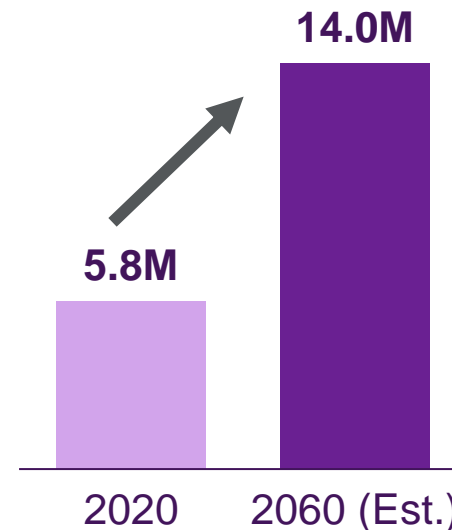
Strong cortisol control scientific rationale to address huge unmet medical need

Rationale

- Cortisol levels are elevated in brain fluid in early AD
- Chronic corticosteroid treatment leads to hippocampal atrophy and cognitive impairment
- Elevated cortisol levels are associated with clinical progression
- Alzheimer's disease mouse model: 30–60% inhibition of 11 β -HSD1 provides full neuroprotection
- AD Phase 2a trial shows slowed disease progression in biomarker-positive patients
- **Safe & effective oral therapy is "holy grail"**

Growing Alzheimer's Disease market – U.S.

Large, unsatisfied and growing market



Anti-amyloid therapy modestly slows AD progression

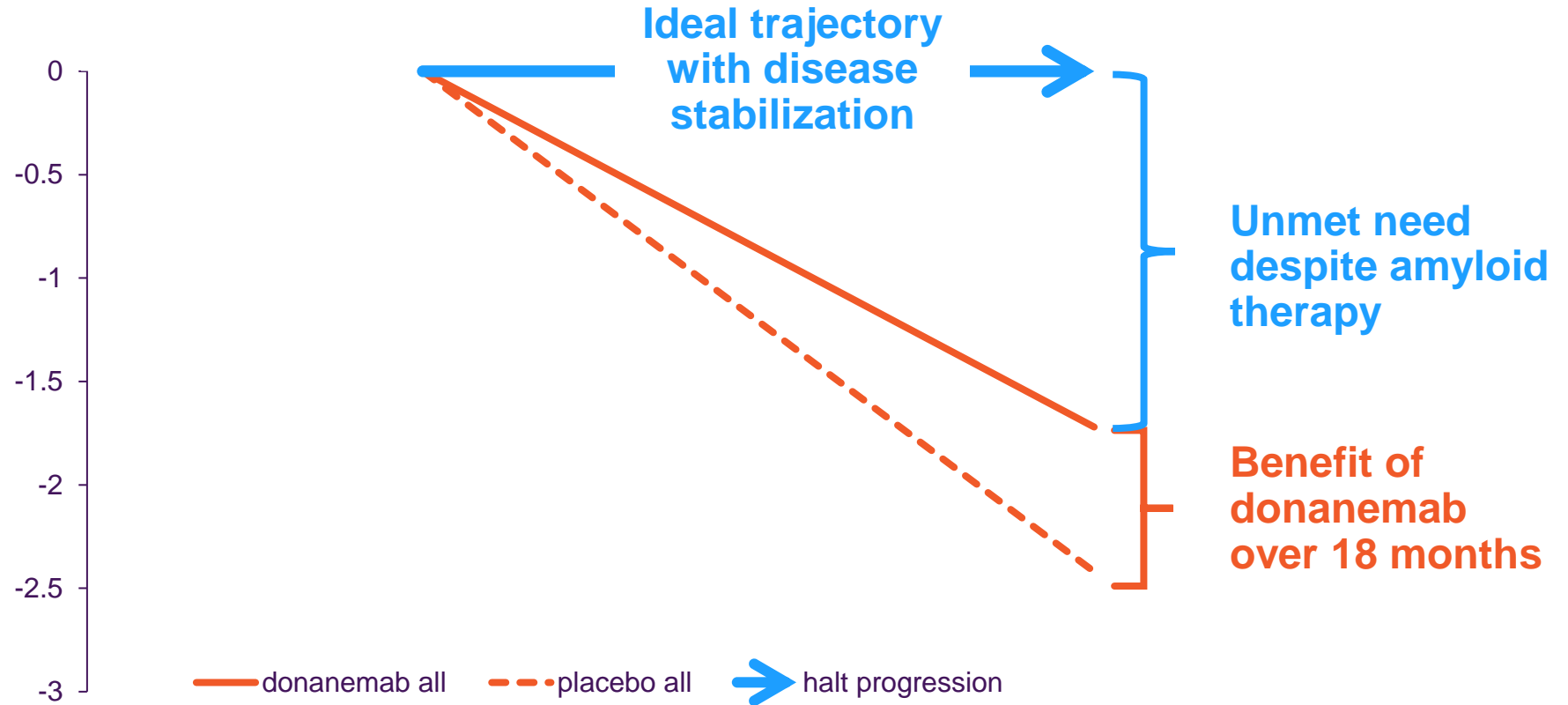
Ideally patients with AD would not worsen on treatment at all

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Worsening of CDR-SB over 18 months



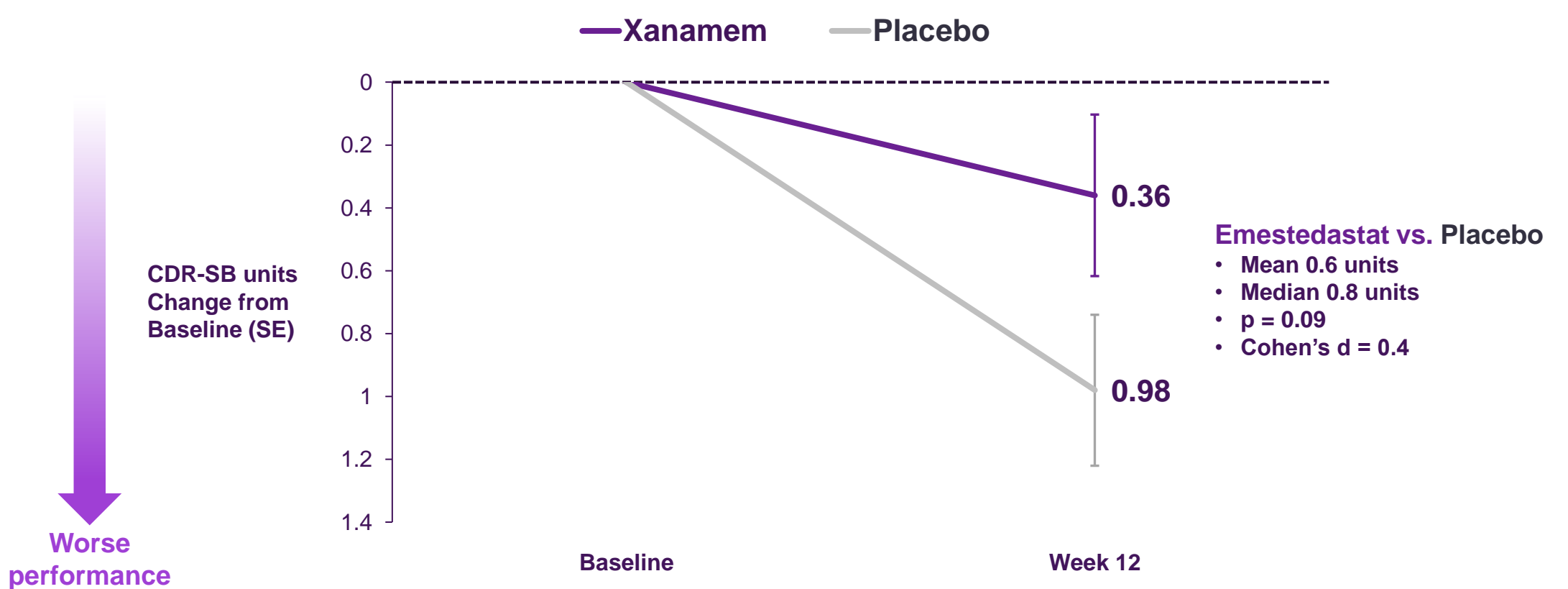
Worse performance



Drugs targeting other mechanisms like emestedastat are needed

Emestedastat benefit in pTau181-positive AD patients

Phase 2a biomarker study: major slowing of CDR-SB decline (n=34)

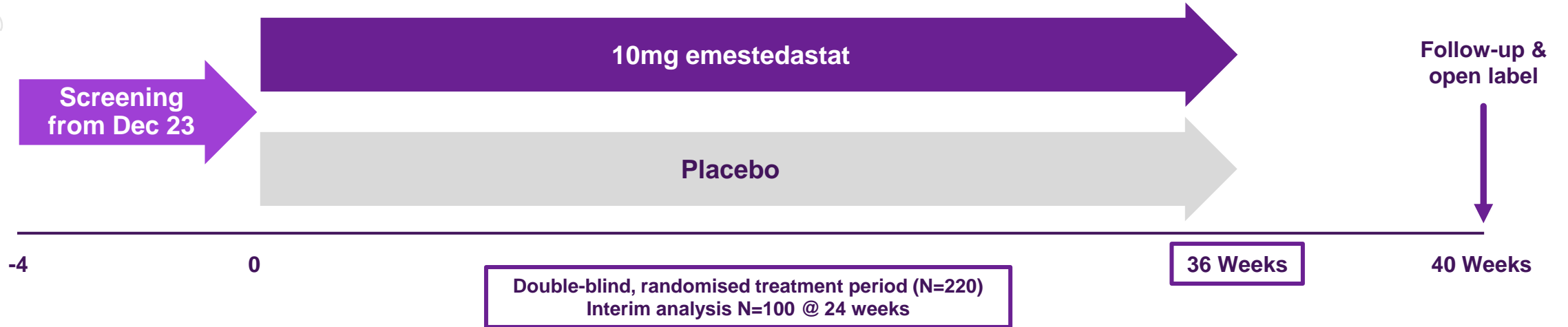


Journal of Alzheimer's Disease 100 (2024) 139–150
Plasma pTau181 Predicts Clinical Progression in a Phase 2 Randomized Controlled Trial of the 11-HSD1 Inhibitor Xanamem® for Mild Alzheimer's Disease
Jack Taylor, Mark Jaros, Christopher Chen, John Harrison and Dana Hilt

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XanaMIA phase 2b/3 trial in Alzheimer's disease

Initial, interim results in Q3 2025, final results H2 2026



Key Inclusion Criteria	Primary Endpoint	Key Secondary Endpoints	Implementation
<ul style="list-style-type: none"> Blood pTau biomarker positive Mild-moderate Alzheimer's by NIA-AA criteria 	<ul style="list-style-type: none"> CDR-SB (functional and cognitive measure) 	<ul style="list-style-type: none"> Cognitive Test Battery (7 cognitive measures well-validated in the Alzheimer's field) Amsterdam Activity of Daily Living (functional measure) 	<ul style="list-style-type: none"> Enrolment at 15 Australian & 10 US sites Interim analysis planned when ~100 people complete 24 weeks

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Phase 2b/3 AD trial in progress

XanaMIA Alzheimer's disease trial implemented as a pivotal, phase 2b/3 design

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XanaMIA AD trial is established as one of two potential “pivotal” trials:

- a. Independent Data Monitoring Committee
- b. “Phase 3-standard” statistical methods
 - Full statistical power for primary endpoint at 36 weeks ($p < 0.05$)
 - Sequential examination of secondary endpoints after primary ($p < 0.05$)
- c. High standard of quality oversight
- d. Commercial tablet formulation

More than 270 screened, approximately 40 enrolled

Interim data expected Q3 2025, final results H2 2026

Depression program

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There remains significant unmet need in depression

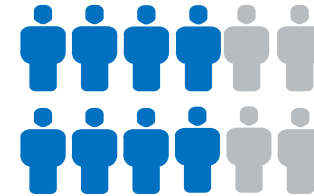
Emestedastat's unique mechanism and safety differentiate it from older drugs

Scientific rationale

- More than 50 years of research associates cortisol with depression
- Elevated CSF and plasma cortisol levels associated with diagnosis, treatment outcomes and relapse
- Positive effects of cortisol receptor antagonism reported with mifepristone¹
- ***Now positive phase 2a data on depressive symptoms (MADRS, PGI-S)***

U.S. Depression market large unmet need

- 21M patients have had ≥ 1 MDD episode

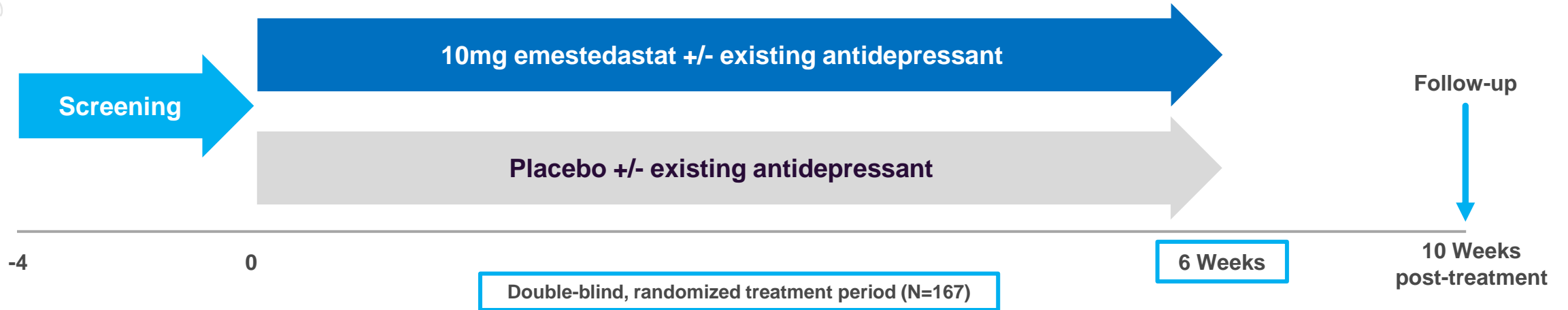


- Two-thirds with an episode **with severe impairment** in the past year
- 61% of all adults with MDD episodes receive treatment
- ≥ 365 M prescriptions per year

A safe, durably effective and combinable small molecule is a very attractive product profile for depression

Completed Phase 2a XanaCIDD trial

Double-blind, proof-of-concept controlled trial to assess safety and efficacy



Primary Endpoint

- **Cogstate Cognitive Test Battery Attention Composite** (attention and working memory)

Key Secondary Endpoints

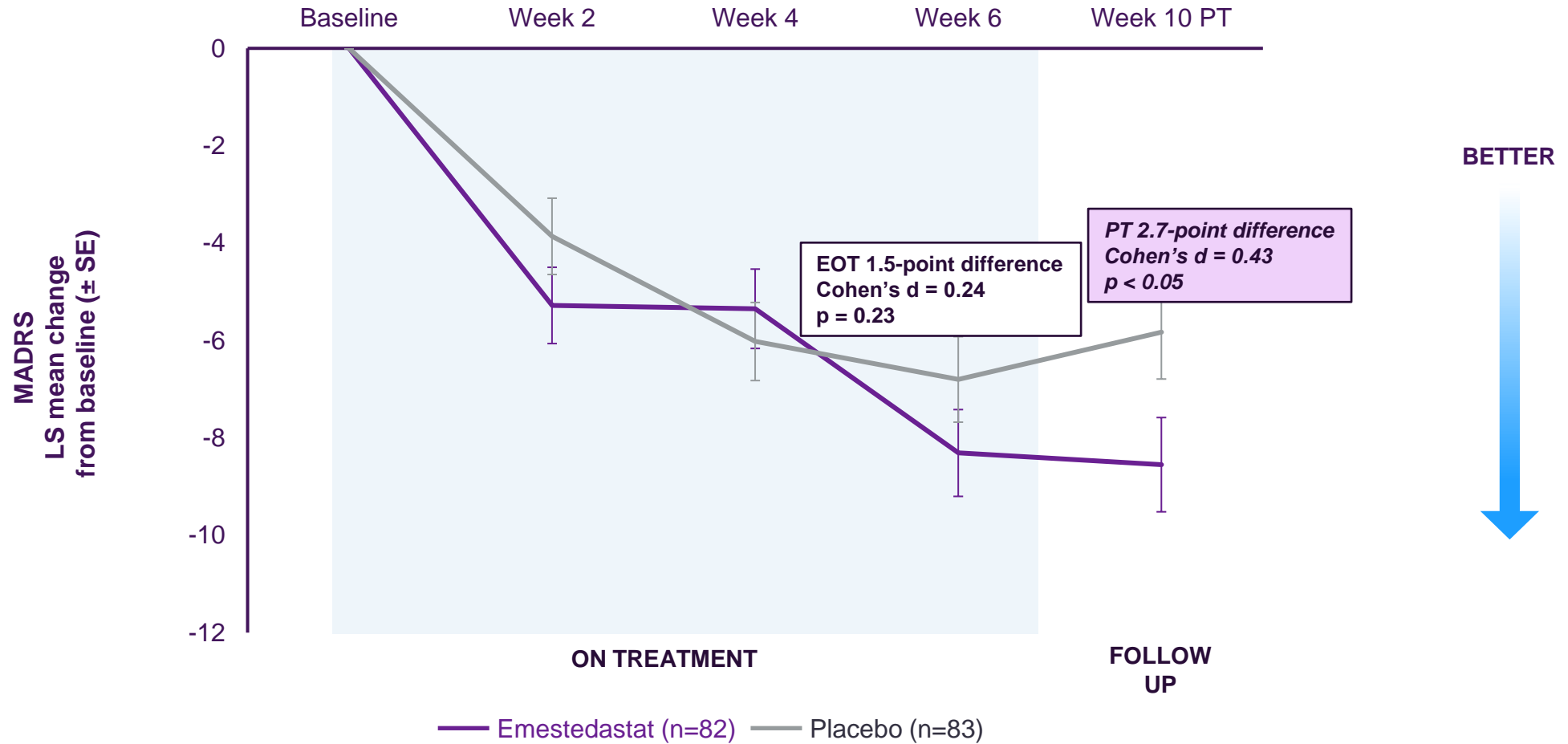
- Montgomery-Åsberg Depression Rating Scale (**MADRS**)
- Patient Global Impression-Severity (**PGI-S**)
- Other MDD and cognitive measures

Baseline characteristics were well-balanced

	Emestedastat (n = 82)	Placebo (n=83)
Mean (SD) age	49 (13)	49 (14)
% female	63	61
Mean (SD) HAM-D	21 (3)	21 (3)
Mean (SD) MADRS	24 (6)	26 (7)
Median number of prior MDD episodes	4	4
% on anti-depressant therapy	77%	86%
Mean (SD) Cognition – Boxfiller coding test	22 (5)	21 (6)

MADRS separation from Week 6 (n=165)

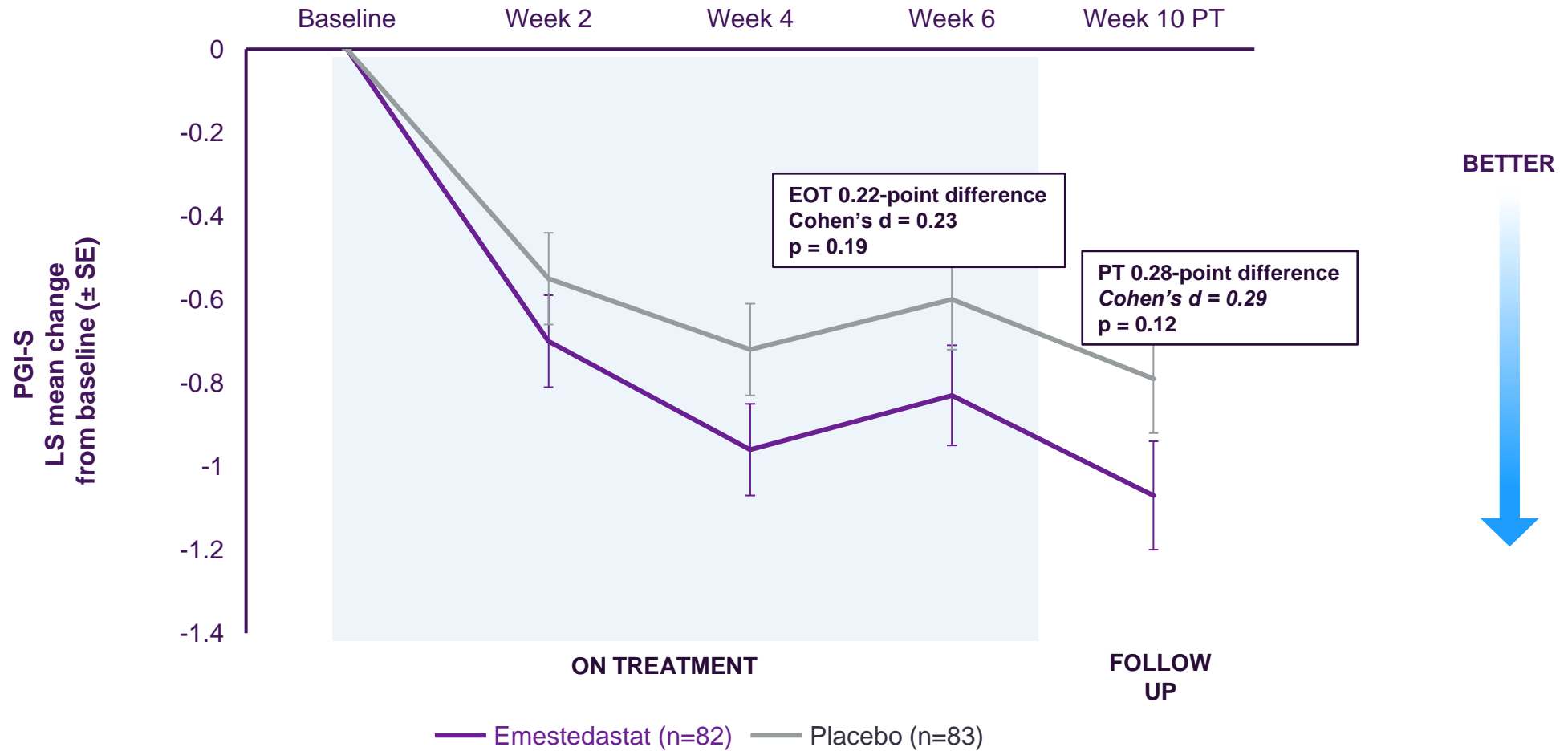
All randomized participants



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PGI-S separation from Week 2 (n=165)

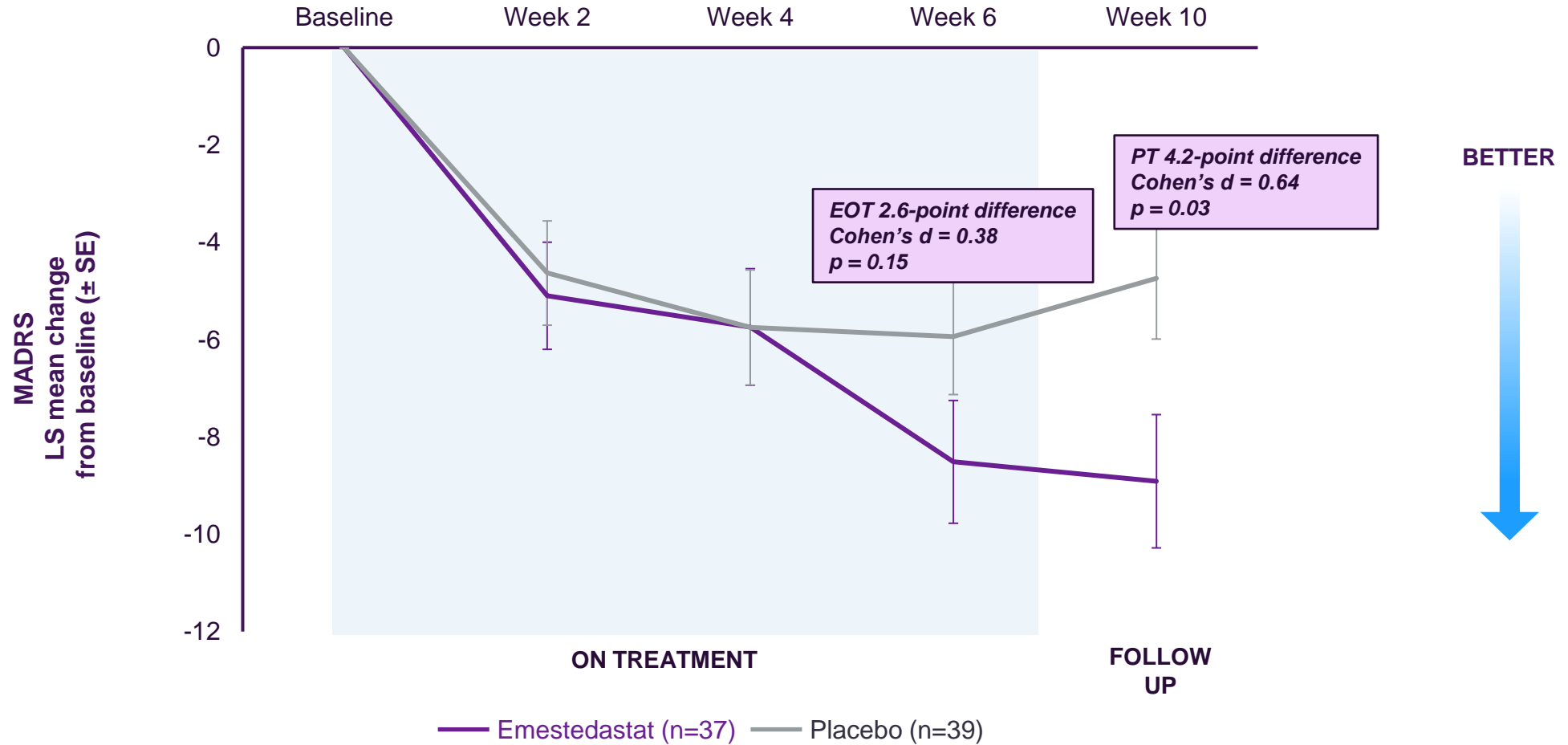
All randomized participants



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MADRS in patients taking concurrent SSRI (n=76)

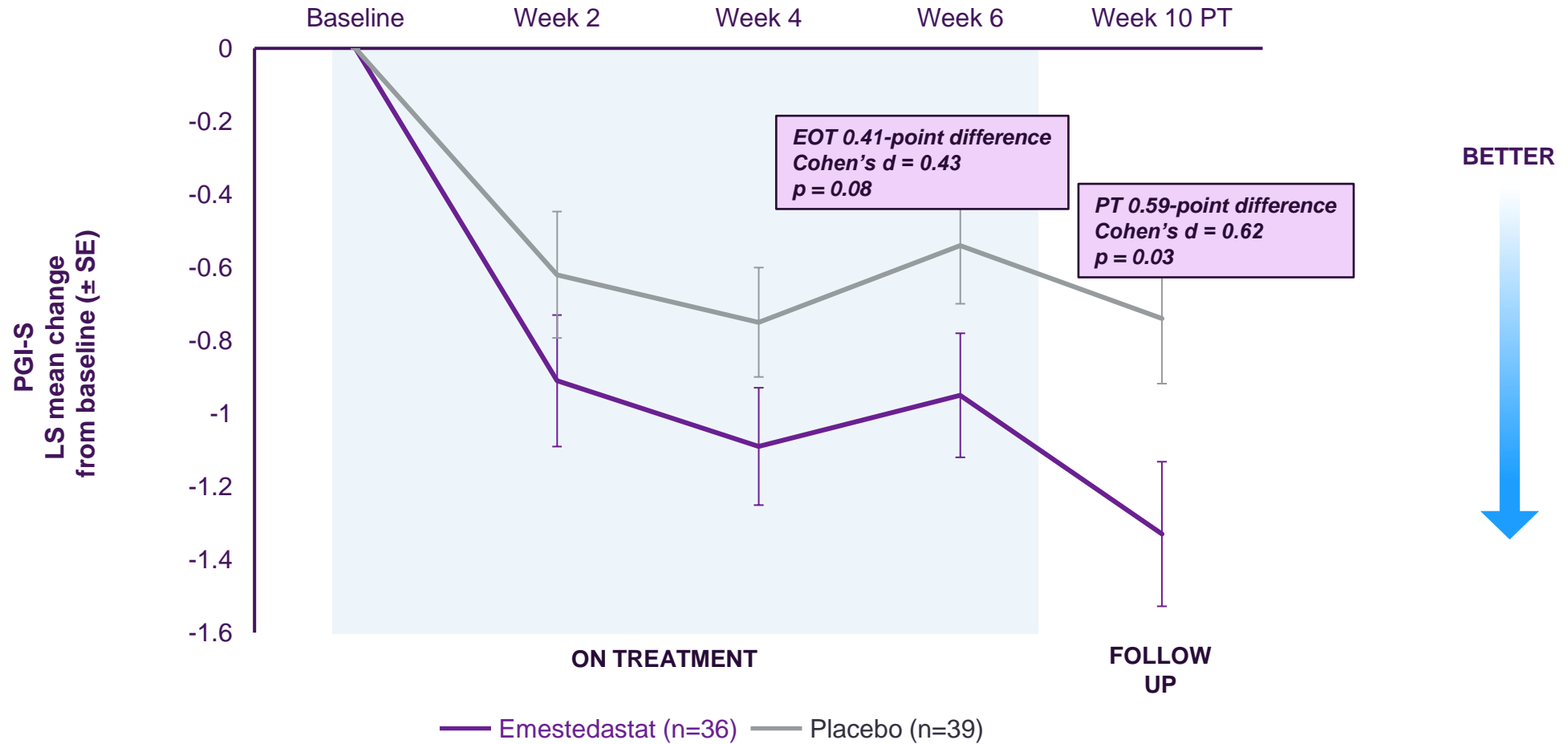
Largest co-treatment subgroup



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PGI-S benefit in patients taking SSRI (n=75)

Largest co-treatment subgroup



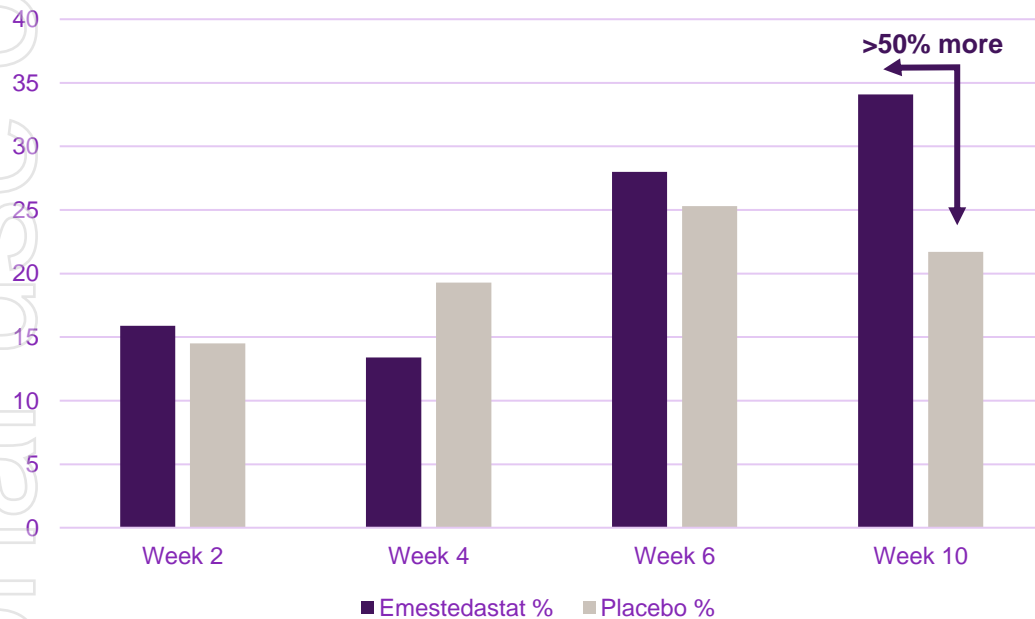
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Emestedastat improves MADRS response rates (n=165)

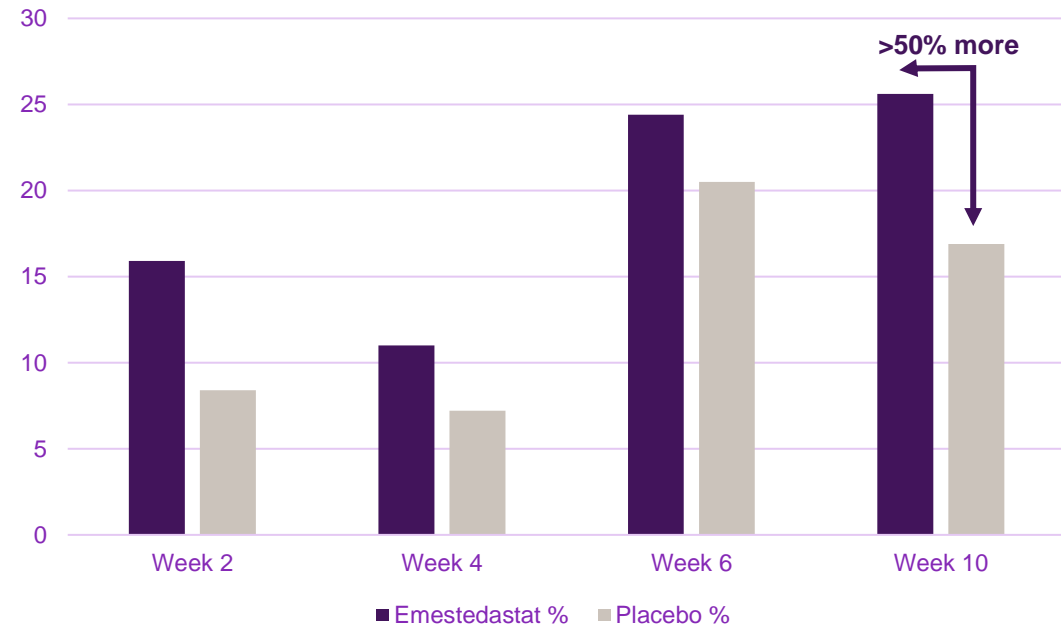


Increased rates of remission (MADRS < 10) and large (50%) improvements

% with $\geq 50\%$ reduction in MADRS



% with < 10 points on MADRS



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Broad & durable subgroup effects - MADRS & PGI-S



Measured at Weeks 2, 4, 6 & 10 (green favors emestedastat, red placebo)

Response/variable	All (n=165)	*No anti-D (n=31)	Yes anti-D (n=134)	*MADRS < 26 (n=81)	MADRS ≥ 26 (n=83)	*Cog. < 0.07 (n=82)	Cog. ≥ 0.07 (n=82)
MADRS (Cd ≥ 0.3) (week)	10	2,6	10	2,6,10	(4),10	2,6,10	-
MADRS (p < 0.05) (week)	10	-	10	6	-	2,10	-
PGI-S (Cd ≥ 0.3) (week)	-	6,10	4,6,10	4,6,10	6,10	4,6,10	-
PGI-S (p < 0.05) (week)	-	-	10	-	-	10	-

Excellent safety profile consistent with prior trials

Summary of Treatment-Emergent Adverse Effects (TEAE)

	Emestedastat n = 82	Placebo n = 83	Overall n = 165
Any TEAE	70 (85.4%)	67 (80.7%)	137 (83.0%)
TEAE related to trial drug	27 (32.9%)	24 (28.9%)	51 (30.9%)
Serious adverse event	0	1 (1.2%)	1 (0.6%)
Related TEAE discontinuation or interruption of drug	3 (3.7%)	1 (1.2%)	4 (2.4%)
TEAEs with incidence \geq 5% overall			
Headache	11 (13.4%)	16 (19.3%)	27 (16.4%)
Fatigue	6 (7.3%)	5 (6.0%)	11 (6.7%)
Nasopharyngitis	4 (4.9%)	6 (7.2%)	10 (6.1%)
Upper respiratory tract infection	5 (6.1%)	5 (6.0%)	10 (6.1%)

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Depression phase 2a conclusions and next steps

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- ***Clinically and statistically significant treatment benefits on depressive symptoms for MADRS and patient-reported outcome of severity***
- Heavily pre/co-treated population with moderate MDD
- Consistent depression efficacy across subgroups
- Strong placebo effect impaired ability to show expected pro-cognitive effect (primary endpoint, data not shown)
- Emestedastat was safe and well tolerated (n=165 treated) with no suggestion of suicide risk or withdrawal syndrome
- The trial was well-conducted, with excellent data quality, no major differences between Australia and the UK or at high enrolling clinical sites

Conclusion

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Clinical 'Proof of Concept' established in two separate clinical indications: AD and MDD



Proof-of-concept

- Strong scientific rationale for CNS cortisol control mechanism in MDD, AD, and other neurodegenerative and neuropsychiatric conditions
- Clinical activity established in AD with a strong CDR-SB effect in pTau-positive patients and in patients with moderate MDD who were heavily co/pre-treated

AD program

- AD Phase 2b/3 trial underway with interim analysis in 2025 and final read out in 2026 using CDR-SB as the primary endpoint

MDD program

- MDD preparations underway to confirm pivotal trials program design and registrational requirements with Regulatory Authorities

Emestedastat has broad therapeutic potential in a variety of neurological and psychiatric conditions

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Appendix

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Key references

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



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Currencies

- Currencies are in Australian dollars unless otherwise stated

Selected Glossary 1

- **11 β -HSD1** – 11 beta HydroxySteroid Dehydrogenase-1 enzyme. Selectively expressed in brain, liver, adipose.
- **A β** – Amyloid beta – a type of amyloid protein associated with Alzheimer’s Disease, 42 and 40 are different forms
- **ACTH** – Adrenocorticotrophic hormone that regulates blood levels of cortisol
- **AD** – Alzheimer’s disease
- **ADAS-Cog** – Alzheimer’s Disease Assessment Score - Cognition
- **ApoE4** – Apoprotein genotype associated with genetic risk of Alzheimer’s Disease
- **ATN** – Amyloid, Tau, Neurodegeneration
- **Clinical Scales** – Measure how a patient feels, performs and functions
- **CDR-SB** – Clinical Dementia Rating “Sum of Boxes” scale measuring cognition and function on an 18-point scale (high worse)
- **CNS** – Central nervous system
- **CSF** – Cerebrospinal fluid
- **CTAD** – Clinical Trials on Alzheimer’s Disease (conference)
- **CTB** – Cognitive Test Battery of computerized tests
- **Double-blind** – Investigators, participants and company do not know who has active vs placebo treatment during a trial
- **EMA** – European Medicines Agency
- **FDA** – US Food & Drug Administration
- **Filamen A** – A protein believed to relate to amyloid toxicity
- **GFAP** – Glial Fibrillary Acidic Protein – a marker of microglial cell activation in the brain
- **IDSST** – International Digit Symbol Substitution Test of cognition
- **IQCODE** – Informant Questionnaire on Cognitive Decline in the Elderly

Selected Glossary 2

- **MADRS** – Montgomery-Åsberg Depression Rating Scale
- **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
- **PGI-S** – Patient Global Impression of Severity – a patient-reported outcome
- **Placebo controlled** – Non-active treatment for double-blind design
- **p-Tau181 or 217 AD** – Biomarker of phosphorylated Tau protein
- **QPCT** – Glutaminyl-peptide cyclotransferase is an enzyme proposed to create toxic amyloid species
- **RAVLT** – Rey Auditory Visual Learning Test
- **RBANS** – Repeatable Battery for the Assessment of Neuropsychological Status (a test of mental abilities)
- **ROC AUC** – Receiver Operating Curve Area Under the Curve (1.0 ideal) – a type of statistical test to compared two methods of measurement
- **SSRI** – Selective serotonin reuptake inhibitor
- **Tau** – A brain protein
- **Ttau** – Total tau levels including both phosphorylated and non-phosphorylated tau

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