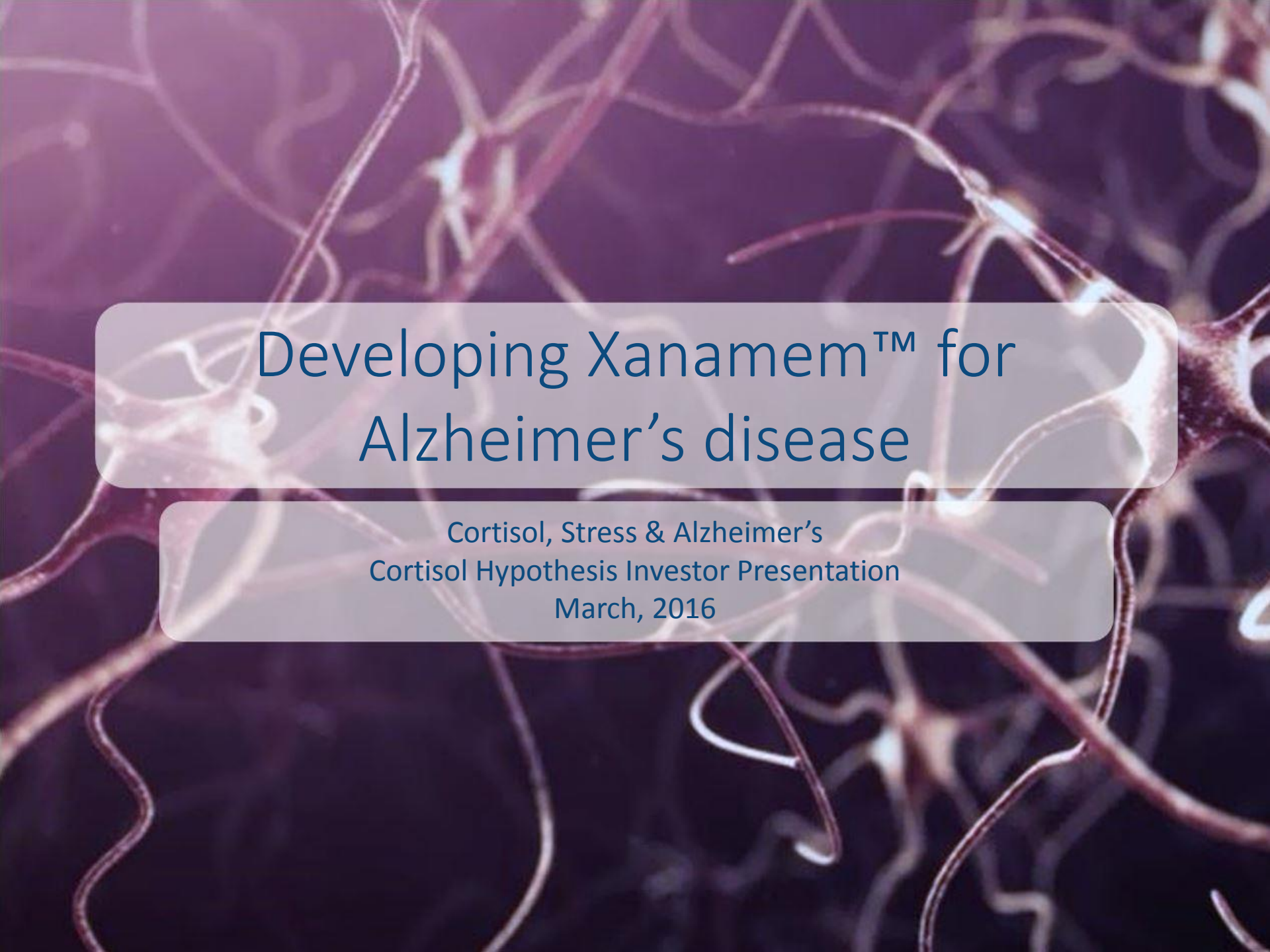




Developing Xanamem™ for Alzheimer's disease

Cortisol, Stress & Alzheimer's
Cortisol Hypothesis Investor Presentation
March, 2016

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Actinogen Medical Management Board

A highly experienced team with a wealth of drug development, commercialisation, and clinical research expertise.



Martin Rogers
Executive
Chairman

- Biotechnology entrepreneur and executive, with financial market capital raising experience, having raised over \$100 M cash equity.
- Non-Executive Director of Oncosil Medical (ASX:OSL).



Dr. Bill Ketelbey
CEO & MD

- MD with 30 years' experience in pharmaceuticals.
- Senior roles at Pfizer, including development of Aricept™, the current leading AD treatment.



Dr. Jason Loveridge
Non-Executive
Director

- Over 20 years experience in developing clinical stage biotechnology companies.
- Director JAFCO Nomura London.
- Participated in over 29 biotech investments in the EU, US and Israel.
- Non-Executive Director of Resonance Health (ASX:RHT)



Dr. Anton Uvarov
Non-Executive
Director

- Healthcare and biotech equities analyst, formerly Citibank NY.
- Non-Executive Director Imugene (ASX: IMU).

Xanamem™ Clinical Advisory Board

Powerhouse advisory board of world experts to drive Xanamem™'s clinical development for early Alzheimer's disease.



Prof. Craig Ritchie

- Professor of Psychiatry of Aging, University of Edinburgh, UK.
- Senior Investigator in over 30 Alzheimer's clinical trials.
- Published extensively on dementia.



Prof. Colin Masters

- Professor, University of Melbourne, Australia.
- Executive Director of Mental Health Research Institute.
- Senior Deputy Director of the Florey Institute of Neuroscience and Mental Health.



Prof. Jeffrey Cummings

- Professor of Medicine (Neurology), Cleveland Clinic, Ohio and Nevada, USA.
- Chair of the Neurological Institute of Cleveland Clinic.
- edited 39 books and published over 650 papers.

Xanamem™ Overview

Cortisol, Stress and Alzheimer's: A promising treatment for Alzheimer's disease and cognitive impairment.

- A novel mechanism of action blocking the production of cortisol (the stress hormone) in the brain through selective inhibition of 11bHSD1.
- Excess cortisol is associated with memory loss, amyloid plaques and neurodegeneration— the hallmarks of Alzheimer's disease (AD).
- Link between excess cortisol and cognitive decline identified in patients with Cushing's disease, Alzheimer's, depression, and in normal aging.
- Discovery and early development of Xanamem™ funded by the Wellcome Trust – ~\$25m invested.
- Second Phase I multiple ascending dose and fed-fasted, CNS PK studies completed in Sept. 2015; defined Ph II dose.
- Phase II trial in patients with mild AD expected to start in Q2 2016 – **study fully funded**.
- Xanamem™ likely to be used in combination with other AD therapies.
- Granted patents; protection to 2031.



THOMSON REUTERS

Xanamem™ named in top five drugs in Phase 1 development in the global pharmaceutical or biotech industries

Global Impact

The increasing burden of dementia is a critical driver for innovation.

The global impact of dementia

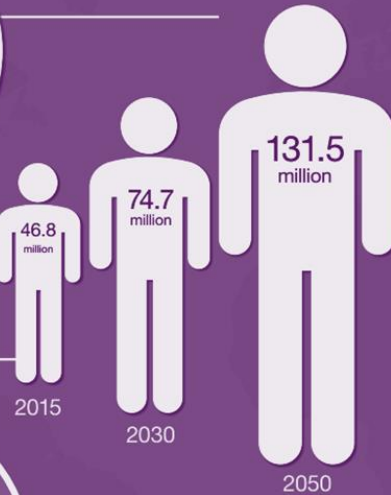


Around the world, there will be 9.9 million new cases of dementia in 2015,

one every 3 seconds

46.8 million people worldwide are living with dementia in 2015.

This number will almost double every 20 years.



68% 2050

Much of the increase will take place in low and middle income countries (LMICs): in 2015, 58% of all people with dementia live in LMICs, rising to 63% in 2030 and 68% in 2050.



The total estimated worldwide cost of dementia in 2015 is US\$ 818 billion.

By 2018, dementia will become a trillion dollar disease, rising to

US\$ 2 trillion by 2030

If global dementia care were a country, it would be the

18th largest economy

in the world exceeding the market values of companies such as Apple and Google



Apple
\$742 billion



Dementia
\$818 billion

Google
\$368 billion

(source: Forbes 2015 ranking).



This map shows the estimated number of people living with dementia in each world region in 2015.

We must now involve more countries and regions in the global action on dementia.



Alzheimer's Disease International

The global voice on dementia

The World Alzheimer Report 2015 was independently researched by King's College London and supported by Bupa.



Actinogen
Medical

Limitations of Current Treatments

Creating a significant unmet need in the Alzheimer's field.



Alzheimer's is the only cause of death
among the top 10 in America that

**CANNOT BE PREVENTED,
CURED OR EVEN SLOWED.**

❖ Alzheimer's disease is emerging as one of the most significant health challenges of our time.

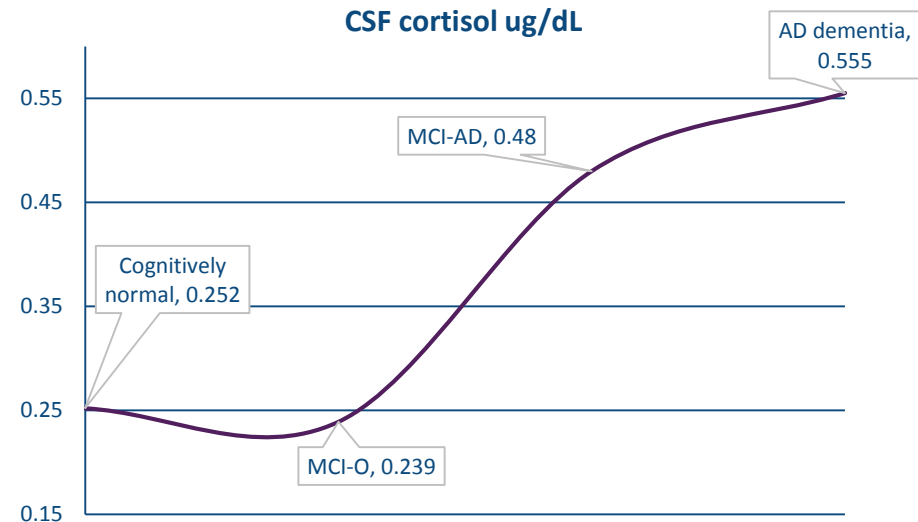
- Affects 11% of people aged 65 years and older, and 32% of people aged 75 years and older.
- Contributes to 1 in 3 deaths.¹
- None of the treatments available today prevent, slow or stop the malfunction and death of neurons in the brain that cause Alzheimer's symptoms.¹
- Current treatment with neurotransmitters (e.g. cholinesterase inhibitors) provides short-term, modest symptomatic relief with no disease modification.
- New treatments with potential to slow progression of mild AD, and halt the structural changes are desperately needed to reduce the burden of disease.

¹Alzheimer's Association- Facts and Figures 2014

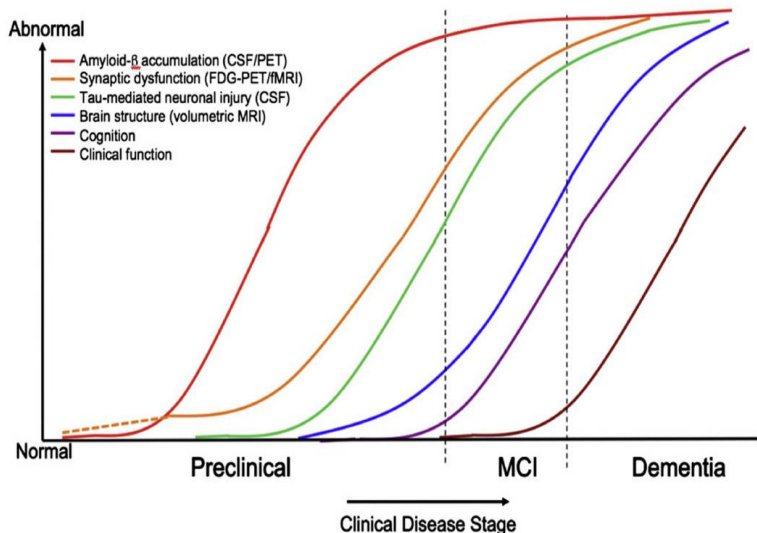
Advances in Understanding Alzheimer's

Presenting opportunities for novel approaches

- Brain pathological and structural changes precede the onset of clinical symptoms.
- Early detection and treatment before onset of structural changes may lead to slowing of disease progression.
- Plasma biomarkers and *in vitro* diagnostic tools would offer population-based screening.
- Goals for reducing burden of disease includes identifying individuals in the earliest pre-symptomatic stages of the disease - this population is most likely to respond.
- Targeting elevated cortisol offers a clinically meaningful solution.

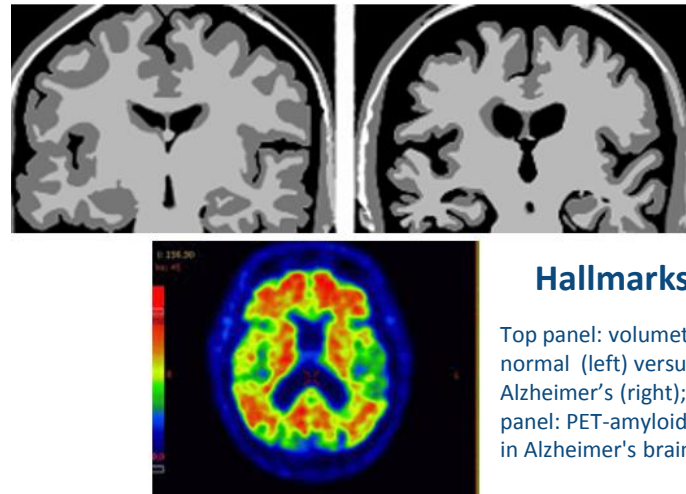


Source: Data points taken from Popp et al 2015. MCI-O = mild cognitive impairment other. MCI-AD = mild cognitive impairment Alzheimer's disease



Biomarker expression over disease life course

Source: adapted from <http://www.re-cognitionhealth.com>

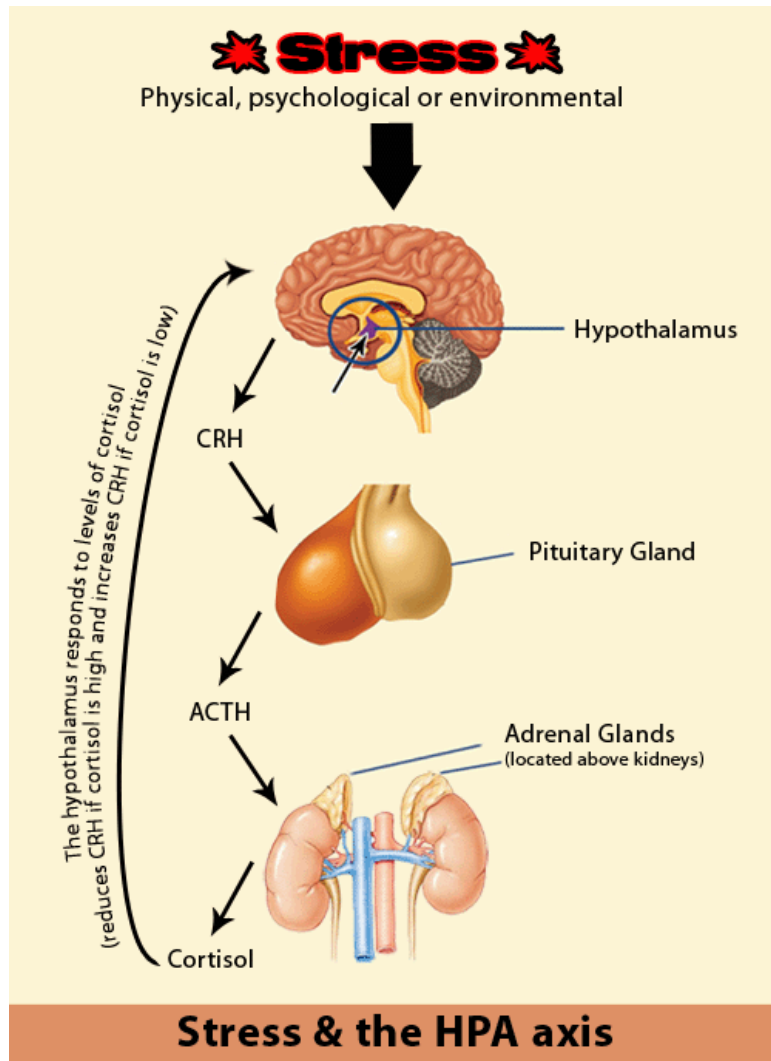


Hallmarks of Alzheimer's

Top panel: volumetric MRI; normal (left) versus Alzheimer's (right); lower panel: PET-amyloid deposition in Alzheimer's brain

Cortisol Hypothesis

Hypothalamic Pituitary Axis dysregulation is linked to neurodegeneration.



- The hypothalamic pituitary axis (HPA) is a stress-responsive neuroendocrine system that ties the CNS to the endocrine system.
- Studies in humans show that HPA axis dysregulation, as evidenced by elevated cortisol levels, is associated with:
 - Reduced hippocampal volume, grey matter and cognitive function in cognitively normal (CN), community-dwelling older adults.^{1,2}
 - Increased cognitive decline in patients with MCI and dementia of Alzheimer's type³, and
 - Increased risk of developing dementia in patients with MCI⁴

1. Geerlings et al. (2015) *Neurology*; 85(11):976-983.
2. Lupien et al. (1998) *Nat Neurosci.*;1(1):69-73.
3. Popp et al. (2015) *Neurobiol. Aging*;36(2):601-607.
4. Hinterberger et al. (2013) *J Am Geriatr Soc.*;61(4):648-651.

Peer Reviewed Medical & Scientific Literature

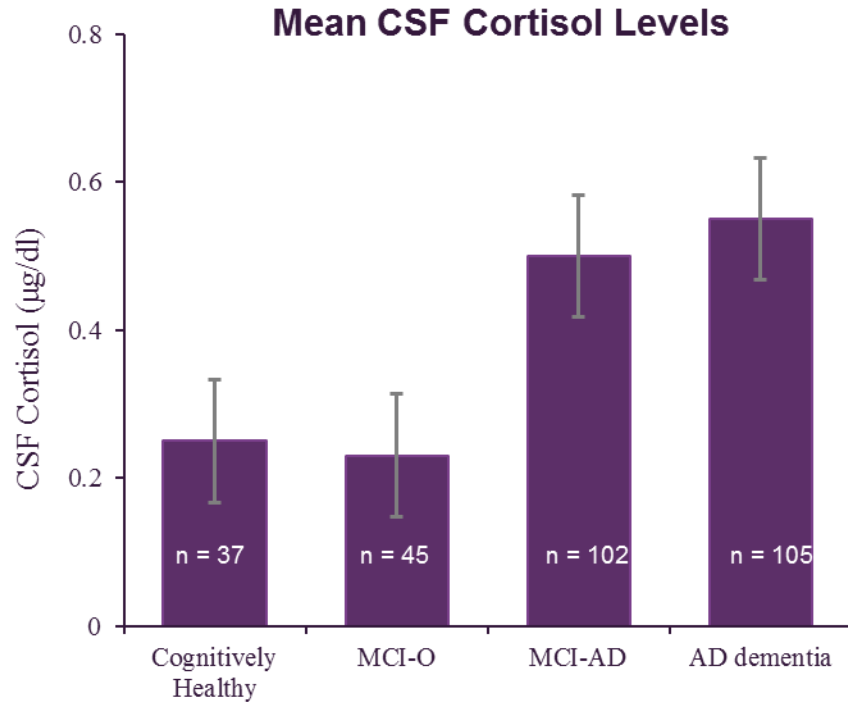
Clear link between 11 β -HSD1, elevated cortisol and cognition.

✿ Extensive human and animal evidence impacting elevated cortisol in cognitive impairment:

- 11 β -HSD1 generates cortisol in brain regions important for cognition (Seckl *et al.* 2004).
- 11 β -HSD1 inhibition (with non-selective inhibitor, carbenoxolone) improves cognition in humans (Sandeep *et al.* 2004).
- Specific human haplotypes of 11 β -HSD1 are associated with sporadic AD risk (de Querain *et al.* 2004).
- 11 β -HSD1 knockout mice are protected against age-related cognitive impairment (Yau *et al.* 2001).
- Small molecule inhibition of 11 β -HSD1 improves cognition in rodent ageing and AD models (Sooy *et al.* 2010, Sooy *et al.* 2015).
- Small molecule inhibition of 11 β -HSD1 reduced A β plaque burden (Sooy *et al.* 2015) and plasma A β (Abbott, ICAD 2010) in rodent AD models.

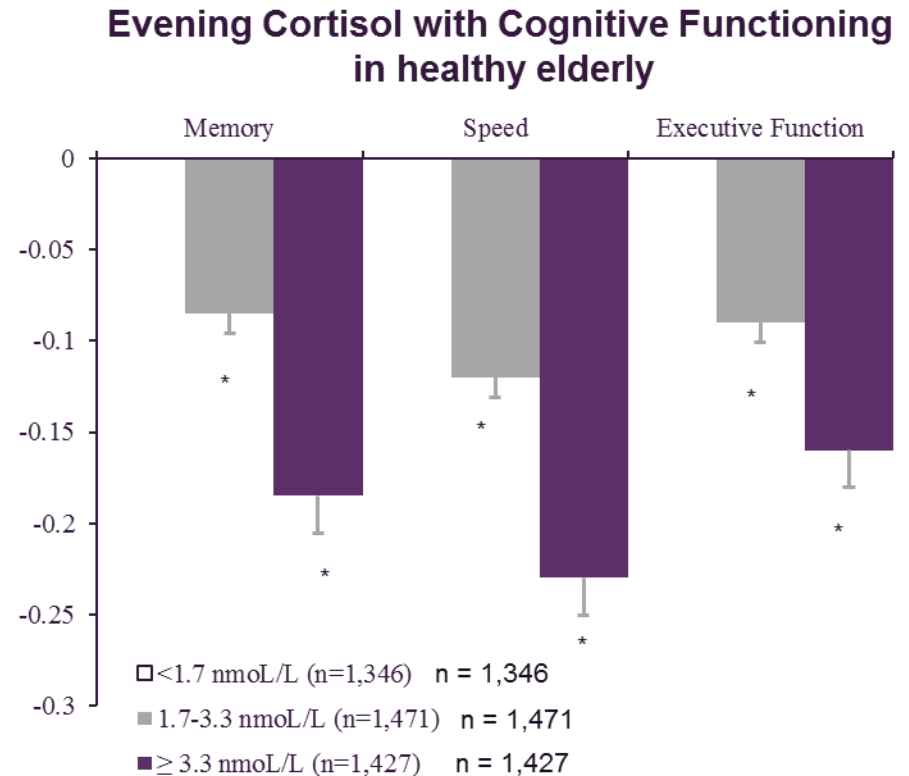
Elevated Cortisol Associated with:

Cognitive decline, amyloid plaques and neural death.



- An increase of CSF cortisol levels in subjects with MCI of AD type is of similar magnitude as in participants with AD dementia, suggesting that HPA-axis dysfunction in AD precedes the dementia disease stages.
- Increased baseline CSF cortisol levels in subjects with AD at the MCI stage are associated with faster cognitive decline and progression of dementia severity over time.

(Popp et al., 2015)



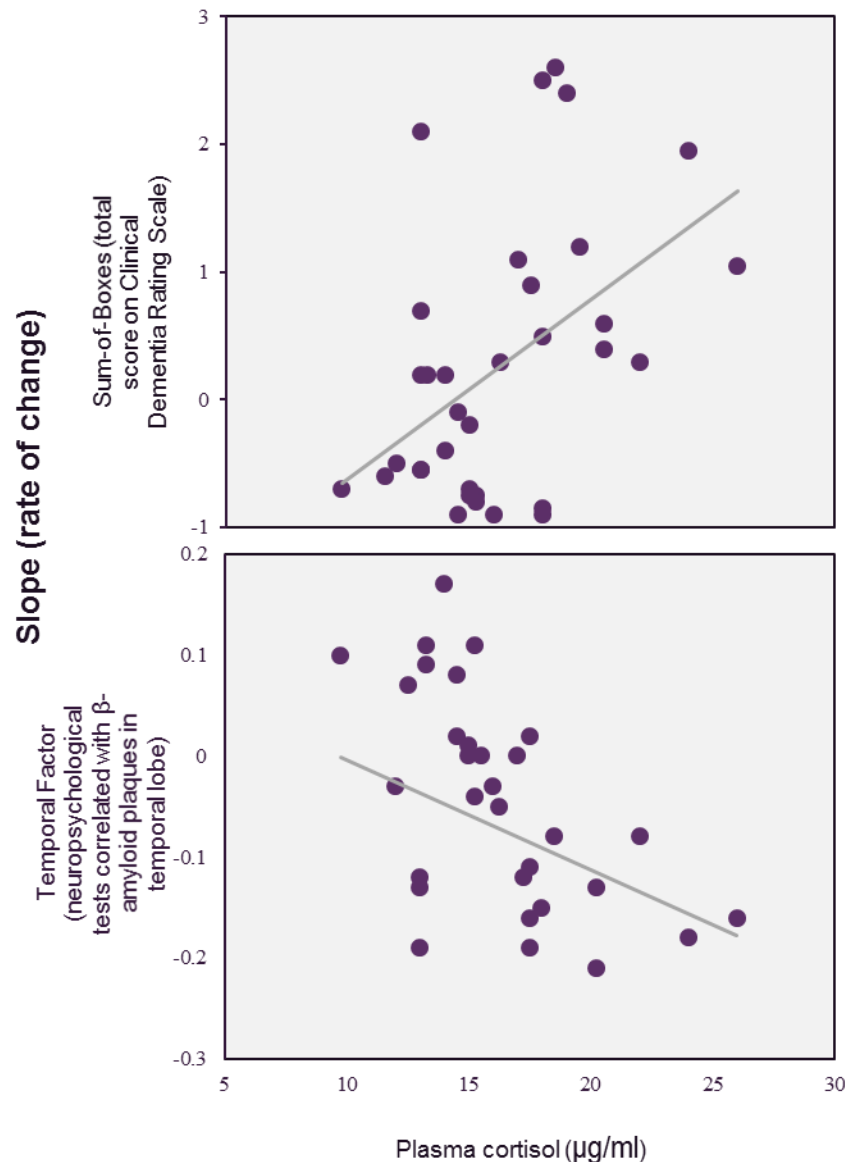
- Higher evening cortisol levels were associated with poorer cognitive performance, while higher morning cortisol levels were associated with better cognitive performance.

* $p < 0.05$ over lowest tertile

(Geerlings et al., 2015)

Cortisol:

Amyloid plaques and loss of neuropsychological function.



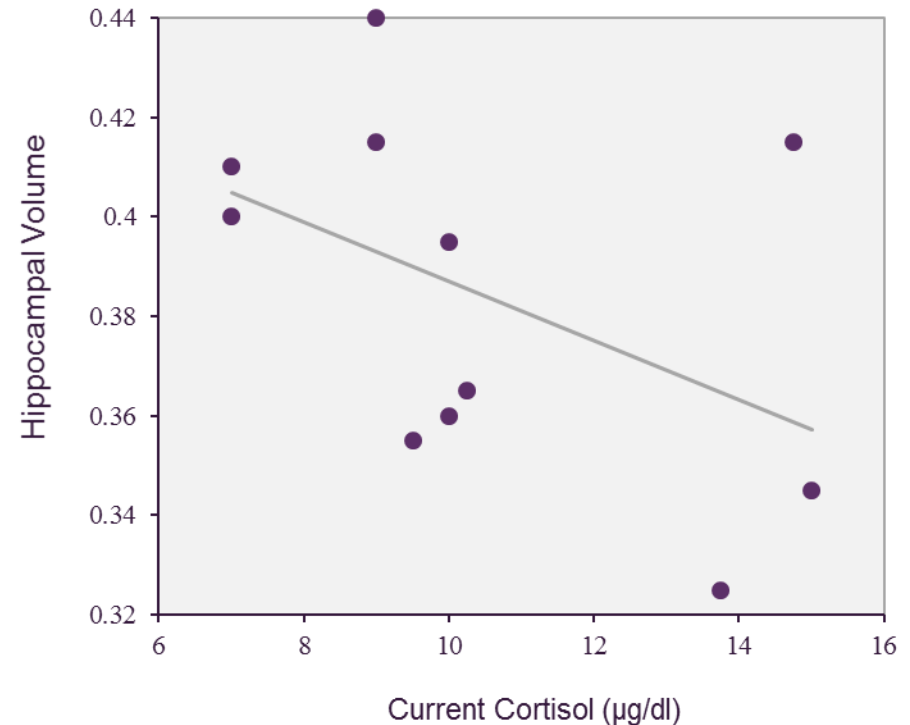
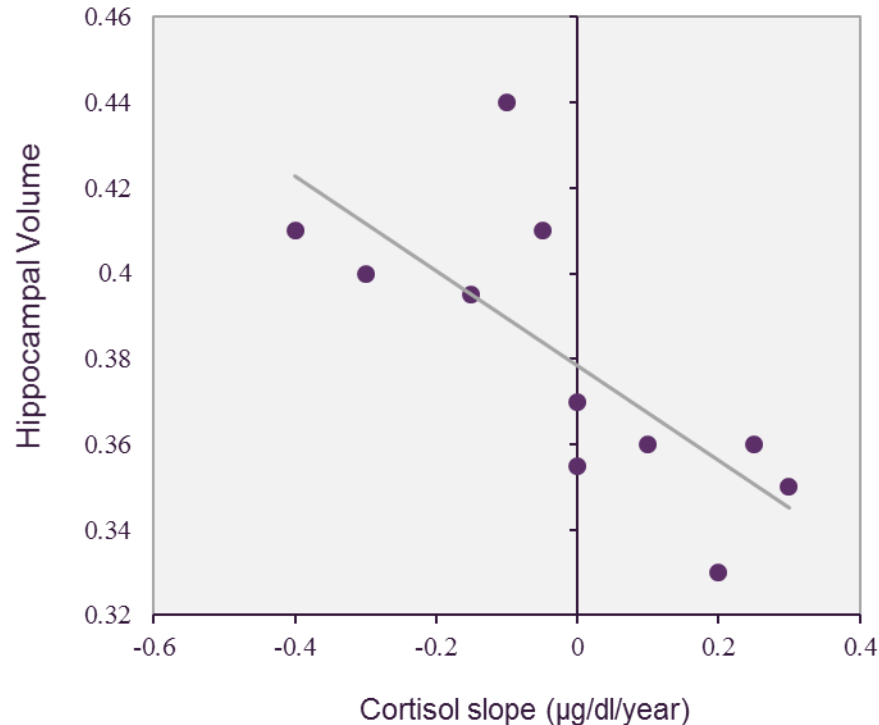
- Higher HPA activity, as reflected by increased plasma cortisol levels, is associated with more rapid disease progression in subjects with Alzheimer type dementia.
- 54 subjects (33 = very mild and mild Alzheimer's type dementia; 21 without dementia) were assessed annually for up to 4 years

(Csernansky et al., 2006)

Elevated Cortisol:

Associated with hippocampal atrophy in Alzheimer's disease.

Hippocampal volume as assessed by MRI is an objective measure of Alzheimer's

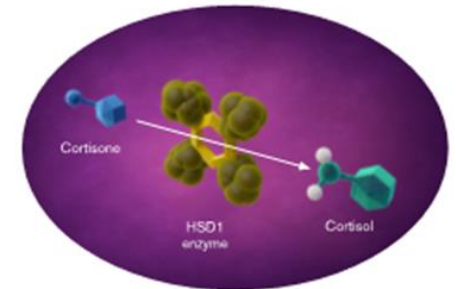
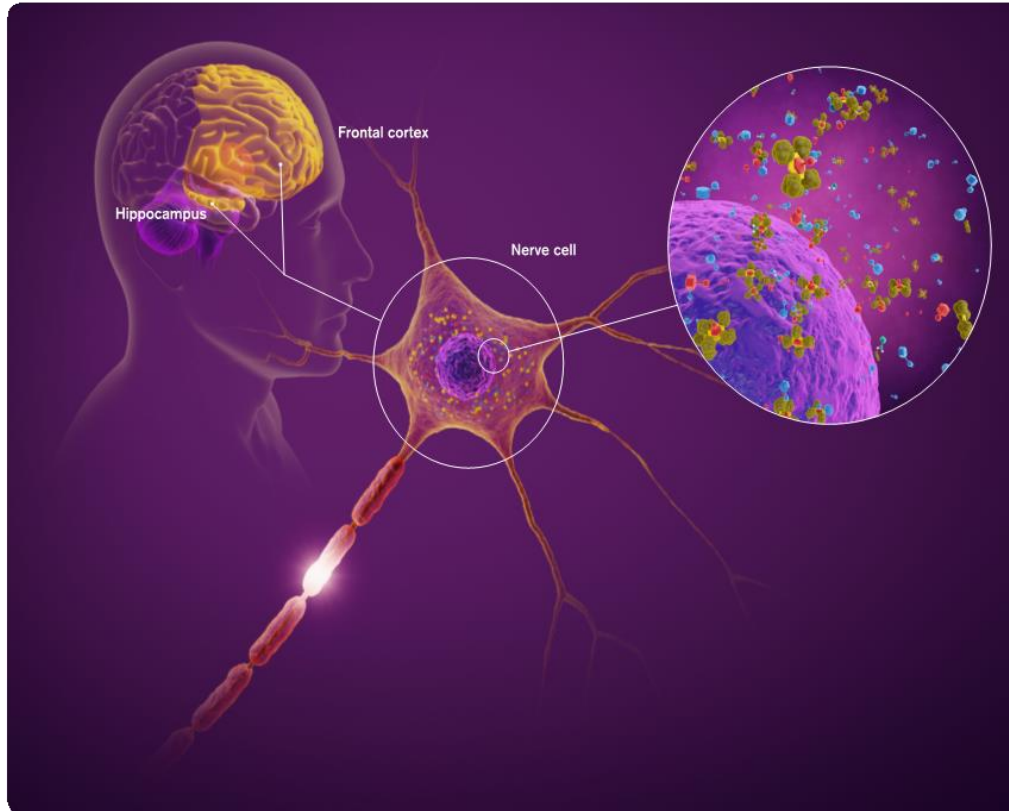


- Aged humans with significant prolonged cortisol elevations showed reduced hippocampal volume and deficits in hippocampus-dependent memory tasks compared to normal-cortisol controls.
- The degree of hippocampal atrophy correlated strongly with both the degree of cortisol elevation over time and current basal cortisol levels.

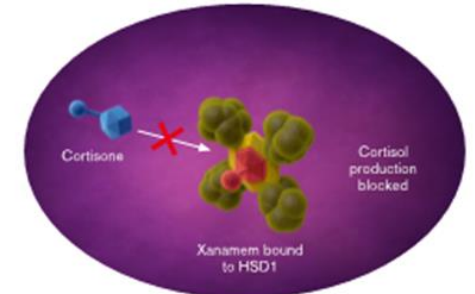
Xanamem™:

*Supressing cortisol production through the inhibition of 11 β -HSD1**

Xanamem™'s mechanism of action is a key differentiator



HSD1 enzyme activates cortisone producing cortisol.



Xanamem™ binds to HSD1, blocking cortisol production.

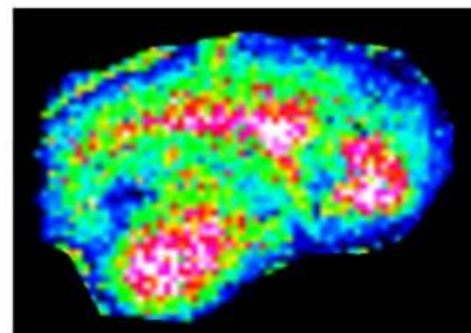
*11 β -HSD1 =11 β -hydroxysteroid dehydrogenase type 1

Xanamem™ Candidate Series Profile

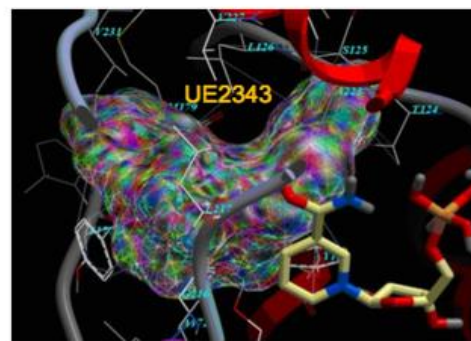
UE2343 selected as a development candidate for Alzheimer's disease.

❖ Target Profile Achieved:

- Low nM potency vs. human 11 β -HSD1.
- High selectivity.
- High cellular potency (IC₅₀ = 24 nM).
- High microsomal stability.
- Low to medium plasma protein binding.
- Acceptable CYP450 inhibition profile.
- Excellent hERG inhibition safety margin.
- High CNS penetration (Pfizer CNS MPO Score = 5.2).
- High bioavailability and low clearance.
- Efficacy in rodent models of age-related cognitive impairment and AD.
- Acceptable safety, tolerability profile in repeat dose non-human studies.



MALDI FTICR MS image of UE2343 in rat brain

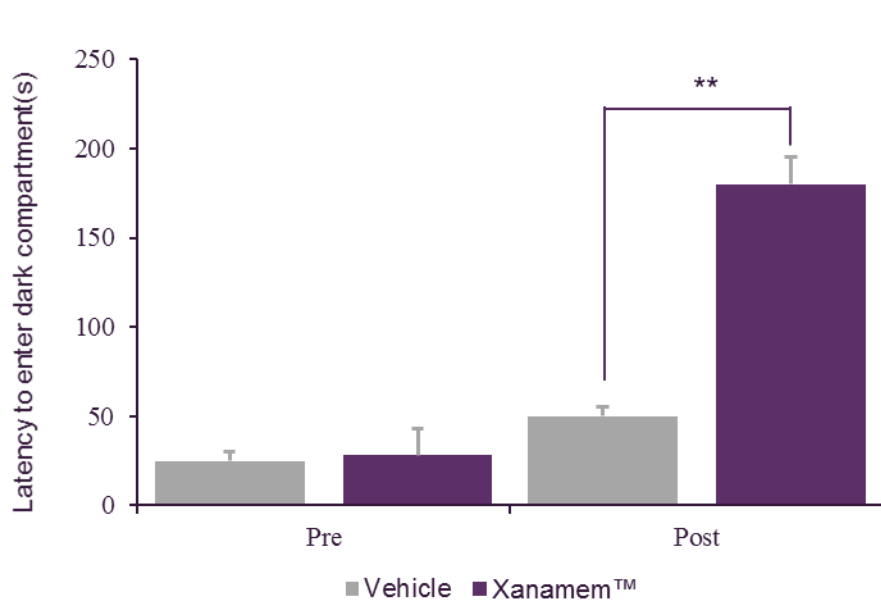


Model of UE2343 in hHSD1 active site

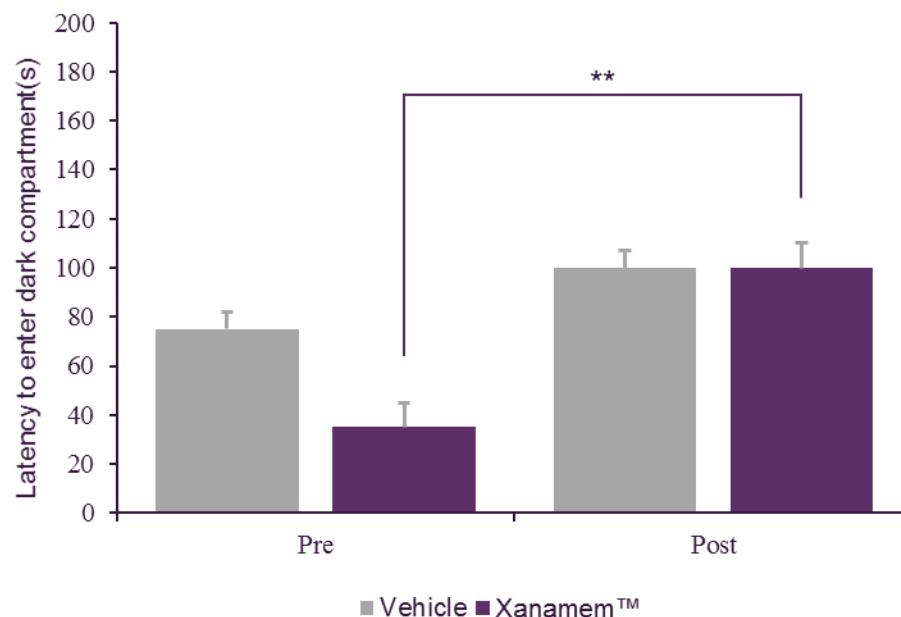
Cognitive efficacy of UE2316

Tg2576 mouse model – precursor to Xanmem™

Cognitive enhancement in a mouse model of Alzheimer's disease with UE2316



** $p = 0.004$ $n = 5$ per treatment group



** $p = 0.004$

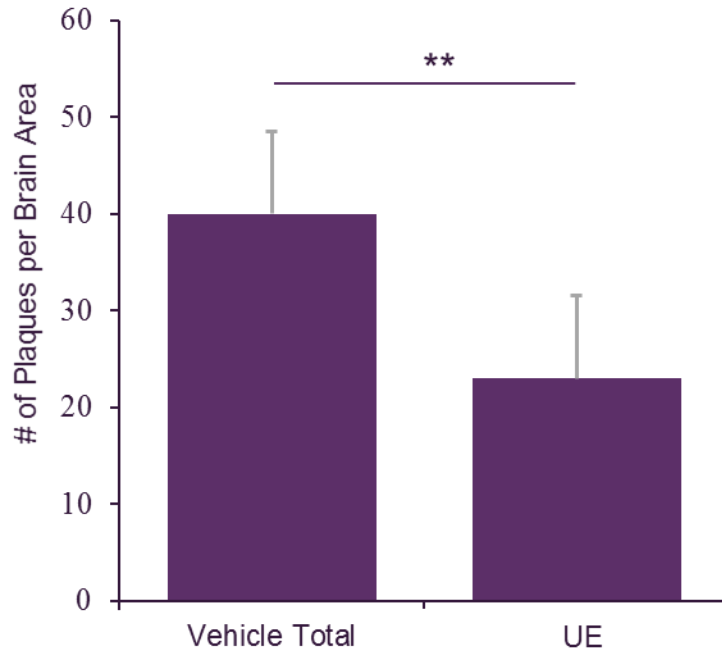
Tg2576 mouse model of Alzheimer's: passive avoidance test:

- Mice with normal learning and memory will avoid entering the chamber where they had previously been exposed to a shock.
- Mice with impaired memory, will typically enter sooner.
- Learning and memory is measured by recording the latency (seconds) until crossing through the gate between the compartments, 6 hours after initial shock.

Disease Modifying Potential of UE2316

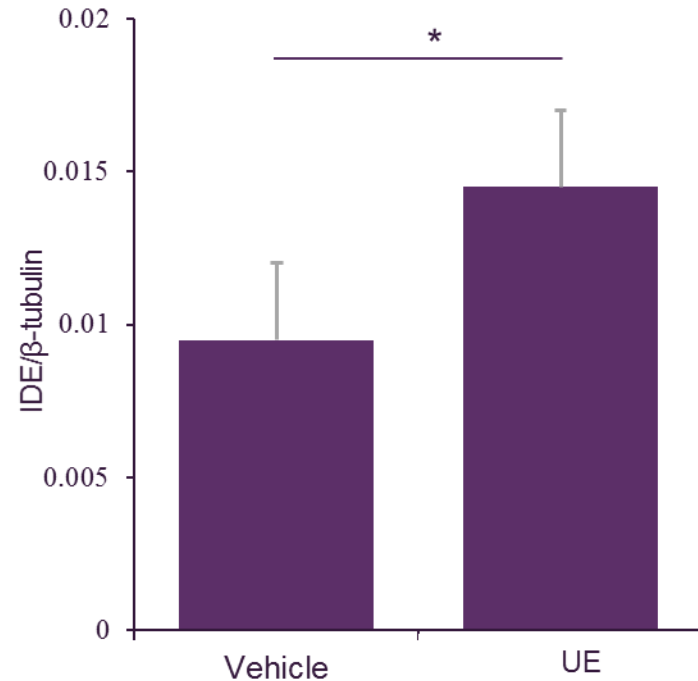
Precursor to Xanmem™

Number of A β Plaques in AD Brain
Treatment for 28 days



** $p = 0.01$

IDE Expression in AD Brain



* $p < 0.04$

- Tg2576 mouse model of Alzheimer's disease
- IDE is Insulin Degrading Enzyme; IDE is implicated in A β degradation.

Xanemem™ PK and PD profile in Humans

Completed Phase I Studies:

1. single ascending dose (SAD) study
2. multiple ascending dose (MAD) studies
3. fed/fasted 35 mg BD
4. CNS PK 35 mg BD

Conclusions of the MAD studies in healthy subjects:

- Safe and well tolerated when given in multiple doses of 10 mg, 20 mg and 35 mg BD for 10 days.
 - Dose dependant increase in treatment emergent AEs – all mild and unrelated.
 - No significant abnormalities for 12-lead ECG, physical examinations, vital signs, laboratory parameters or nerve conduction.
- Steady state achieved day 5.
- $T_{1/2}$: 10-14 hours.
- C_{max} day 10: 200-900 ng/ml.
- Median T_{max} : 4-6 hours.
- $AUC_{(0-12)}$: 4 fold accumulation at the highest dose.
- Recommended regimen: twice daily 35 mg
- Fed/fasted preferably taken with food.
- CNS PK – Xanemem present in CSF in concentrations that are predicted to effectively inhibit the 11 β -HSD1 enzyme in the brain.

Goals for Xanamem™ Phase II

“XanADu” Study

Title	A Phase II Double-Blind, Randomised, Placebo-Controlled Study to Assess the Safety, Tolerability and Efficacy of Xanamem™ in Subjects with Mild Dementia due to Alzheimer’s Disease (Mild AD)
Patient cohort and number	- 200 subjects aged 50 years or older with mild dementia due to probable AD, with MMSE 20-26 (inclusive), and CDR 0.5 or 1.0. - on stable dose of AChEI and/or memantine (at least 3 months) OR treatment naïve
Dose and administration	-35 mg twice daily for 12 weeks
Primary objective	- to evaluate the extent to which twice daily doses of Xanamem™ 35 mg improves performance from baseline to end of treatment compared to placebo, as measured by changes in ADCOMposite Scores (ADCOMs, composite data derived from Alzheimer’s Disease Assessment Scales -Cognitive subscale version 14 [ADAS-Cog v14], Clinical Dementia Rating Scale-Sum of Boxes [CDR-SOB], and Mini-Mental Status Examination [MMSE]) and ADAS-Cog v14 as primary end-points
Secondary objective	- to assess the extent to which twice daily doses of Xanamem™ 35 mg improves performance from baseline to end of treatment compared to placebo, as measured by changes to: Rey Auditory Verbal Learning Test (RAVLT), CDR-SOB, MMSE, Neuropsychiatric Inventory (NPI), Neuropsychological Test Batteries (NTB) – Executive Domain.
Exploratory assessments	- optional pharmacokinetic and pharmacodynamic sub-studies to gather further data in particular populations, and CSF sampling to explore disease modification through amyloid and tau clearance, as observed in animal studies, and assessing Xanamems impact on metabolic function. ApoE genotyping will also be undertaken.
Safety assessments	- incidence of adverse events, change in clinical safety laboratory values, ECG, CSSRS (Columbia Suicide Severity Rating Scale), nerve conduction velocity sub-study
Study sites	- approximately 20 sites in AU, UK, US; First patient expected on study – Q2 2016.

- Inputs in to the Alzheimer's Phase II trial design by the Scientific Advisory Board (Prof Brain Walker, Prof Alan Boyd, Dr Scott Webster, Prof Jonathan Seckl), the CEO Dr Bill Ketelbey and the Clinical Advisory Board (Prof. Craig Ritchie, Prof. Colin Masters and Prof. Jeff Cummings).

Xanamem™: Aspirational Positioning

Cognitive Impairment and mild Alzheimer's disease.

“An oral agent that provides durable symptomatic and disease modifying benefits in mild Alzheimer's disease by direct inhibition of cortisol production”

Xanamem™ is a novel agent likely to be used in combination with other AD therapies.



Xanamem™:

The potential for a pipeline in a product.

Development opportunities:

Xanamem™'s novel mechanism of action – blocking excess cortisol production – offers a number of additional potential indications with features of progressive cognitive impairment:

- Diabetes cognitive impairment (DCI)
 - Diabetes affects 347 million people globally.
 - 3.5% of diabetes patients will develop dementia (~12.2 million worldwide).¹
 - People with T2DM may have twice the risk of developing dementia compared with those without diabetes; and the risk is stronger in people who use insulin.^{1,2}
- Parkinson's disease dementia (PDD)
 - An estimated 7 to 10 million people worldwide are living with PD.³
 - Mild cognitive impairment can be identified in 15% of PD patients at time of diagnosis,
 - and may even precede motor symptoms.⁴
 - MCI progresses to dementia, known as PDD, in 24 -31% of PD patients;
 - PDD is present in all PD patients that survive more than 10 yrs.⁴
- Cognitive dysfunction in schizophrenia, depression.



1. Biessels et al. (2006) Risk of dementia in diabetes mellitus: a systematic review. *Lancet Neurology*; 5: 64–74.
2. Ott A, et al. (1999) Diabetes mellitus and the risk of dementia: The Rotterdam Study. *Neurology*; 53: 1937-42.
3. Parkinson's Disease Foundation http://www.pdf.org/en/parkinson_statistics:
4. Medtracker, 2015

Direct Molecule Competition:

11 β -HSD1 inhibitors in clinical development.

Compound	Sponsor	Study	Indication	Primary endpoint	Source
ASP3662	Astellas	Ph II (recruiting) NCT02372578	Painful diabetic peripheral neuropathy	Analgesic efficacy	https://clinicaltrials.gov
ASP3662 and ¹¹ C-AS2471907	Astellas	Ph I (recruiting) NCT02194491	Alzheimer's disease	PET occupancy	https://clinicaltrials.gov
BI-135585/ VTP-34072	Boehringer Ingelheim Vitae Pharmaceuticals	Ph II (completed) NCT02150824	T2DM* metabolic syndrome	Did not meet primary efficacy end-point: fasting plasma glucose	https://clinicaltrials.gov
AZD-4017 (open innovation)	University of Birmingham	Ph II (recruiting) NCT02017444	Idiopathic intracranial hypertension	Change in intracranial pressure at 12 weeks	https://clinicaltrials.gov
AZD-4017 (open innovation)	University of Birmingham	Ph II (recruiting)	bone turnover in post-menopausal osteopenia	Change in biochemical markers of bone turn-over	http://www.hra.nhs.uk
ABT-384	Abbott	Ph II (completed) NCT01137526	Mild-moderate AD	Did not show non-inferiority to donepezil ADAS-Cog	https://clinicaltrials.gov

*Clinical development of 11 β HSD1 inhibitors for T2DM was unsuccessful: other candidates including MK-0736, RG4929, RG7234, AZD4017, LY2523199 were also discontinued for T2DM

ABT-382 Trial In mild-moderate Alzheimer's disease

Abbott's ABT-384 did not show non-inferiority against donepezil for the primary end-point of ADAS-Cog score during a 12-week trial¹.

Trial design raises doubt whether, at the doses tested in the phase II study, ABT-384 achieved optimal inhibition of 11bHSD1 enzyme in the brain:

- In the PK study, ABT-384 concentrations reached peak levels within 2.5–5 h in the CSF. ABT-384 CSF levels were ~10–50% of the free plasma levels and 0.3–1% of the total plasma levels. (Katz *et al.* 2013; Supplementary Figure 3)².
- The highest dose used in the efficacy study (50mg) achieved only 2.3ng/ml of drug at C_{max} in the CSF, which would deliver a maximum of 11% 11 β -HSD1 enzyme inhibition ³
- 18ng/ml of ABT-384 in the CSF is theoretically required to deliver a minimum of 50% inhibition of 11 β -HSD1 enzyme.
- The physicochemical properties of ABT-384 also predict low CNS penetration (CNS MPO = 3.4).
- Xanomem, in comparison to ABT-384, achieves higher plasma levels at a lower dose, delivers higher concentrations of drug to the CSF, is more potent and has higher free fraction, and thus would be expected to achieve much higher enzyme inhibition in the brain.

1. Marek et al. (2014) Efficacy and safety evaluation of HSD-1 inhibitor ABT-384 in Alzheimer's disease. *Alzheimer's & Dementia*, 10; S364-S373.

2. Katz et al. (2013) Peripheral and central nervous system inhibition of 11b-hydroxysteroid dehydrogenase type 1 in man by the novel inhibitor ABT-384. *Transl Psychiatry*, 3; e295; 1-7.

3. Enzyme inhibition at 2.3ng/ml of drug was calculated using the competitive inhibition equation: % inhibition = $1/(1+IC50/[ABT384]) \times 100$.

Xanamem™:

Predicted to have high brain penetration.

- CNS Multi-parameter Optimisation (MPO) Calculator shows Xanamem has characteristics that favour high CNS penetration.
- In the phase I multiple ascending dose study, Xanamem demonstrated high pharmacologically active concentrations in human CSF (manuscript in preparation).

ID	Clog P	Clog D	MW	TPSA	HBD	pKa	Clog P score	Clog D score	MW score	TPSA score	HBD score	pKa score	TOTAL (ex 6)
Xanamem	1.1	1.1	381.4	95	2	2.1	1.0	1.0	0.8	0.8	0.5	1.0	5.2
ABT384	3.24	3.2	493	91.5	3	6	0.9	0.4	0.1	1.0	0.2	1.0	3.4

■ desirable score ■ acceptable score ■ suboptimal score

CNS MPO is an algorithm for predicting the potential of a molecule to penetrate the brain based on six calculated physicochemical properties¹:

- 1) ClogP = lipophilicity, calculated partition co-efficient
- 2) ClogD = calculated distribution coefficient at pH 7.4
- 3) MW = molecular weight
- 4) TPSA = topological polar surface area
- 5) HBD = number of hydrogen bond donors
- 6) pKa = most basic centre

High CNS penetration is implied by MPO score ≥ 4 on a scale of 0 – 6.

¹Wager et al. (2010) Moving beyond the rules: the development of a central nervous system multiparameter optimisation (CNS MPO) approach to enable alignment of druglike properties. *ACS Chem Neurosci*. 1(6); 435-449.

Solid Patent Estate:

Broad range of compounds, compositions and uses.

Application/ priority	Title	Scope	Jurisdiction/ Status
WO 2009/112845 ("Webster-4") priority: 13 Mar 2008 US divisional application (Webster-4-PCT-US-CON1)	Amido-thiophene compounds and their use.	-broad coverage for a range of heterocyclic analogues, including UE2343; compounds, pharmaceutical compositions and the use of such compounds and compositions to treat disorders that are ameliorated by the inhibition of 11 β -HSD1 metabolic syndrome and CNS disorders including dementia, MCI, Alzheimer's disease	US granted US-con1 granted JP granted EP granted KR pending CA pending AU granted IL pending IN pending
WO2010/146338 ("Webster-6") priority: 15 Jun 2009	Amido-isothiazole compounds and their use as inhibitors of 11 β -HSD1 for the treatment of metabolic syndrome and related disorders	- certain amido-isothiazole compounds 11 β -HSD1; pharmaceutical compositions and the use of such compounds and compositions to treat disorders that are ameliorated by the inhibition of 11 β -HSD1 metabolic syndrome and CNS disorders including dementia, MCI, Alzheimer's disease	US granted JP granted EP granted KR pending CA pending AU pending IL granted IN pending
WO2011/135276 ("Webster-7") priority: 29 Apr 2010	3, 3 -disubstituted- (8 - aza - bicyclo [3.2.1] oct- 8 - YL) -[5- (1H - pyrazol - 4 yl) -thiophen- 3 -yl] methanones as inhibitors of 11 (BETA) -HSD1	a range of compounds, pharmaceutical compositions and the use of such compounds and compositions to treat disorders that are ameliorated by the inhibition of 11 β -HSD1 metabolic syndrome and CNS disorders including dementia, MCI, Alzheimer's disease	EP granted US pending JP pending KR pending CN pending CA pending AU pending IL pending IN pending MX pending BR pending RU pending



Cortisol, Stress and Alzheimer's

Continued information on Xanamem™.

- ✧ Alzheimer's - a significant unmet need in a huge and growing global market.
- ✧ Xanamem's™ novel mechanism of action targeting the stress hormone cortisol – a key differentiator.
- ✧ Cortisol inhibition hypothesis supported by good pre-clinical and clinical evidence.
- ✧ Evidence Xanamem™ is **both symptomatic and disease modifying**.
- ✧ Phase II trial in mild Alzheimer's patients expected to initiate in Q2 2016, and will run in the USA under an IND, Aus. and the UK .
- ✧ Phase II study **fully funded through to completion**.
- ✧ Patent protected to 2031 – composition of matter.
- ✧ Expect to use Xanamem™ in combination with other AD therapies – little or no market competition.
- ✧ A number of very significant additional indications being evaluated for development in parallel.
- ✧ Intent to grow and develop the business through partnering and licencing – in and out.

A microscopic image of neurons, showing their cell bodies and long, branching processes. The image is overlaid with a semi-transparent white rectangle containing text.

Thank You

Email info@actinogen.com.au for further information, or to sign up for our newsletter.