Developing Xanamem[™]



Presented by Dr. Bill Ketelbey CEO & Managing Director



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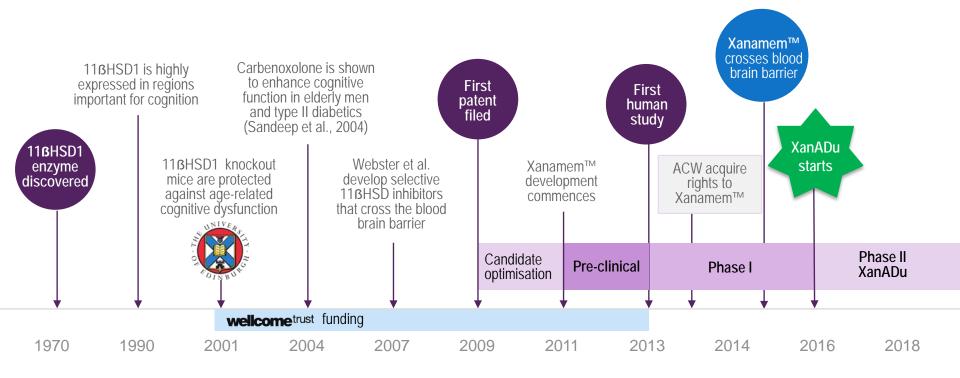


Xanamem[™]: A prime investment opportunity

- Alzheimer's a significant unmet need in a huge and growing global market
- Xanamem™'s innovative, differentiated mechanism of action targeting the stress hormone cortisol
- Evidence Xanamem[™] is both symptomatic and disease modifying
- Phase II study fully funded through to completion
- Patent protected to 2031 composition of matter
- Value enhancing additional indications in substantive markets of interest to big pharmaceutical companies

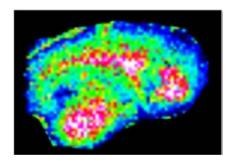


Actinogen's journey of discovery





Xanamem[™] unique value proposition





- High potency at low oral doses
- Highly selective and specific enzyme binding
- Safe and well tolerated in humans
- Delivered to the brain to target site of action
- Established proof of principle in models of Alzheimer's Disease



Leadership Team



Dr. Bill Ketelbey CEO & MD





Dr. Jason LoveridgeNon-Executive Director





Martin Rogers
Executive Chairman

Oncosil
MEDICAL Ltd

PRIMA BIOMED LTD
Australian Cancer Treatment Company





Dr. Anton UvarovNon-Executive Director





Clinical Advisory Board:

world renowned neuroscience leaders



Professor Craig Ritchie

- Chair, Xanamem[™] Clinical Advisory Board
- Professor of Psychiatry of Aging, University of Edinburgh, UK.
- Senior Investigator in over 30 Alzheimer's clinical trials.
- Published extensively on dementia.



Professor Colin Masters

- Xanamem™ Clinical Advisory Board
- Professor, University of Melbourne, Australia.
- Executive Director of Mental Health Research Institute.
- Senior Deputy Director: Florey Inst. of Neuroscience & Mental Health.



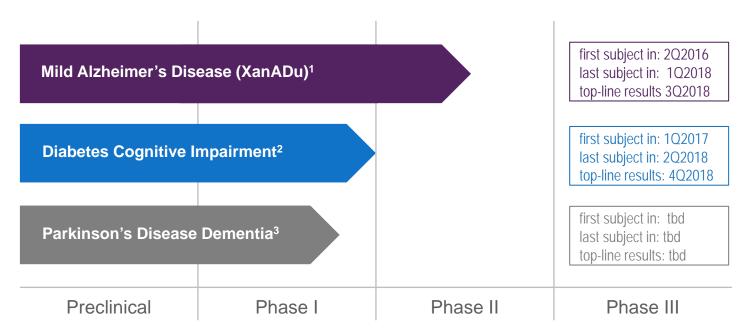
Professor Jeffrey Cummings

- Xanamem™ Clinical Advisory Board
- Professor of Medicine (Neurology), Cleveland Clinic, USA.
- Chair of the Neurological Institute of Cleveland Clinic.
- Edited 39 books and published over 650 papers.



Xanamem[™] research pipeline

milestone timelines are estimates



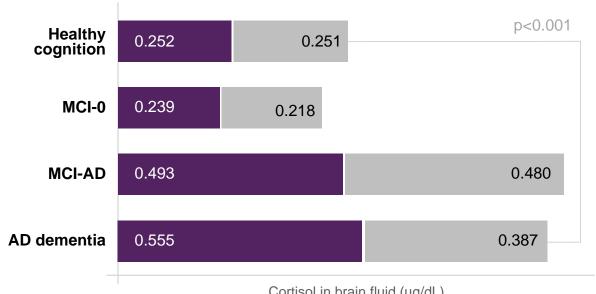
¹ Trial commenced March 2016.



² Phase II trial design complete. Final operational planning underway. Trial expected to start late 2016.

³ Planning ongoing for Phase II trial design

Cortisol and Alzheimer's disease



Neuroendocrine dysfunction leading to elevated cortisol precedes disease state in AD dementia

Elevated cortisol was associated with progressive cognitive decline

Cortisol in brain fluid (µg/dL)

The transitional stage between 'normal' functional ability and a full-blown clinical picture of dementia is described as mild cognitive impairment (MCI). The term MCI refers to decrease in cognitive function, from a formerly normal level towards a mildly impaired level. (Kornhuber et al., 2009).

Source: Popp et al., 2015 MCI-AD = MCI of Alzheimer's type MCI-O = MCI of other type







© 2015 American Academy of Neurology

Plasma Cortisol and Progression of Dementia in Subjects With Alzheimer-Type Dementia John G. Csernansky, M.D. Hongxin Dong, Ph.D. Objective: Studies of subjects with de-Anne M. Fagan, Ph.D.

Article

mentia of the Alzheimer type have reported correlations between increases in activity of the hypothalamic pituitary adrenal (HPA) axis and hippocampal degeneration. In this study, the authors sought ension, ni inio aviet, nic avienore avigin to determine whether increases in plasma cortisol, a marker of HPA activity, Plasma cortion, a marker or ren autrosy, were associated with clinical and cognitive measures of the rate of disease progression in subjects with Azbeimer-type

tive measures were derived from grov Results: In the subjects with dementic

but not in those without dementia, highe plasma cortisol levels were associated with more rapidly increasing symptoms of dementia and more rapidly decreasing performance on neuropsychological tests associated with temporal lobe function. No associations were observed between plasma cortisol levels and clinical and cognitive assessments obtained at the single assessment closest in time to the plasma

Conclusions: Higher HPA activity, as reflected by increased plasma contisol levels, is associated with more rapid disease progression in subjects with Alzheimer-

[A.m. J. Psychiatry 2006; 163:2164-2169]

Combined Plasma and Cerebrospinal Fluid Signature for the Prediction of Midterm Progression From Mild Cognitive Impairment to Alzheimer Disease

Benoit Lehallier, PHO, Laurent Essioux, PhO; Javier Gayen, PhO; Roxana Alexandridis, PhO; Tania Nikolchava, MD, PhD, Tony Wyss-Coray, PhD, Markus Britschgi, PhD; for the Altheimer's Disease Neuroimaging Initiative

IMPORTANCE A reliable method of detecting Alzhi needed for patient stratification in clinical trials or upcoming therapies. Current cerebrospinal fluid (C for AD offer reliable identification of patients with u the rate of AD progression.

Salivary cortisol, brain volumes, and cognition in community-dwelling elderly without dementia

Melissa E. Garcia, MPH

Tamara B. Harris, MD

Vilmundur Gudnason,

Lenore J. Launer, PhD

MD. PhD

Signature Signatures. MSc Objective: We investigated the associations of morning and evening salivary cortisol levels with Uspective You investigated the associations of maximity and evening associate investor regional brain volumes and cognitive functioning in community-dwelling older persons without Mirjam I. Geerlings, PhD ABSTRACT Gudny Eiriksdottir, MSc

Method: From the Age, Gene/Environment Susceptibility (AGES)-Reykjavík Study, we included 4,244 persons without dementia (age 76 ± 5 years, 58% women) who had 1.5T brain MRI, ч.сч» ригоско милиси ситиентие wage го ± э years, 10 № моляте who нас 4.51 оган мет, аssessment of cognitive functioning, and saliva collected at home 45 minutes after awakening озможениям от содетните изполнять, это запус совесию от потве ча minutes after амаменты, and at night. Linear regression analysis was used to estimate the cross-sectional relationship. among cortisol levels, brain volumes, and cognitive functioning, adjusting

Cortisol levels during human aging predict hippocampal atrophy and memory deficits

Ø € 1998 Nature America Inc. - http://neurosci.nature.com

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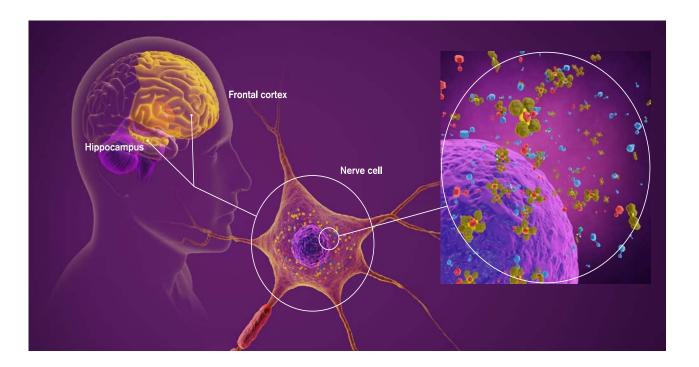
John C. Morris, M.D.

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wated glucocorticoid levels produce hippocampal dysfunction and correlate with individual ficits in spatial learning in aged rats. Previously we related persistent cortisol increases to memor pairments in elderly humans studied over five years. Here we demonstrate that aged humans with significant prolonged cortisol elevations showed reduced hippocampal volume and deficits in hippocampus-dependent memory tasks compared to normal-cortisol controls. Moreover, the degree of hippocampal atrophy correlated strongly with both the degree of cortisol elevation over time and current basal cortisol levels. Therefore, basal cortisol elevation may cause hippocampal damage and impair hippocampus-dependent learning and memory in humans.

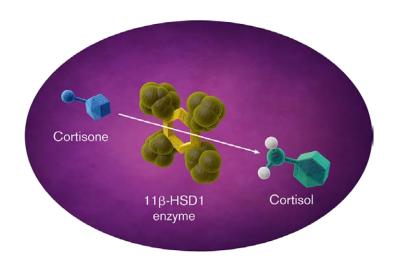
Actinogen Medical

Targeting elevated cortisol at the site of action

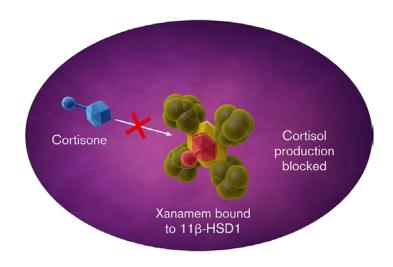




Xanamem[™]- inhibiting action of 11βHSD1



11βHSD1 enzyme activates cortisone producing cortisol



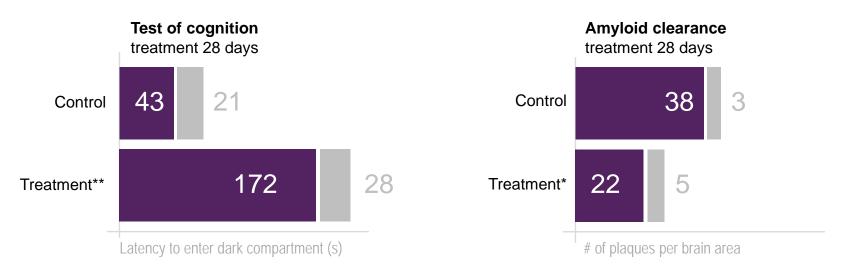
Xanamem[™] binds to 11βHSD1, blocking cortisol production

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



Xanamem™

Symptomatic and disease modifying effects in mouse models



Significant improvement in cognition in only 28 days treatment which continues out to 41 weeks.

UE 2316 The mean plus the SEM. ** = P< 0.004, * = P<0.01 Tq2576 rodent model of Alzheimer's disease. Source: Sooy et al., 2015. Endocrinology 156(12):4592-4603.



Xanamem™ development – Phase II

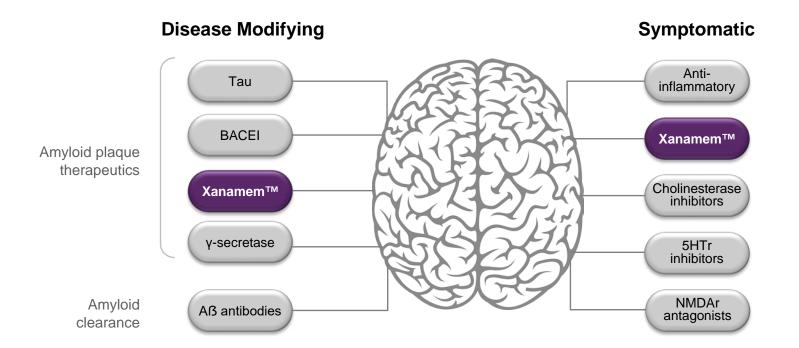
XanADu – Phase II double blind, randomised, placebo controlled study to assess the efficacy of Xanamem[™] in participants with mild AD

Co-primary end points Treatment course 200 ADCOMS + 12 weeks First patient enrolment expected Q2 ADAS-Cog Mild Alzheimer's patients Secondary Xanamem[™] twice Being trialled in daily dosage end-points AUS, USA Multiple: MMSE and UK CDR-SOB, RAVLT, NPI, NTP & CSF Aß and Tau

ADCOMS: AD Composite Score. Wang et al., 2016. J. Neurol. Neurosurg. Psychiatry 0:1-7. Clinicaltrials.gov: NCT02727699.



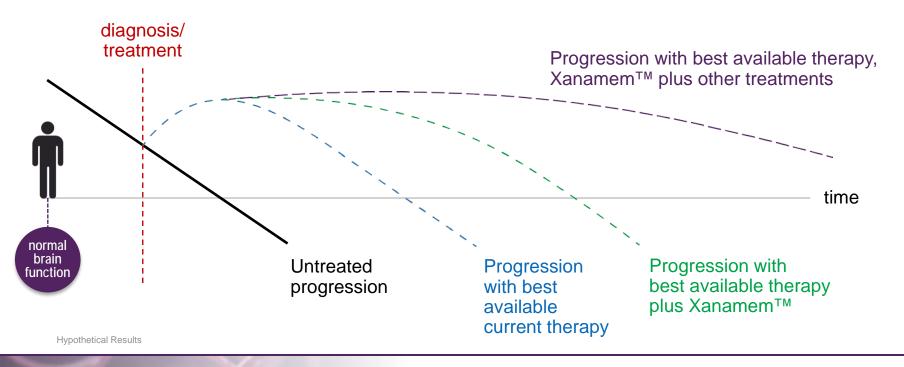
Xanamem[™] potential for dual mechanism of action





Clinical goal for Xanamem™

Filling an unmet need in the market





Xanamem's[™] estimated market share

Global peak sales in 2031



¹ Reference Baker Young Initiation Coverage, August 2015.



² Price comparator Aricept, assumes 10% market share at peak sales, optimistic scenario.

³ Price comparator Exelon patch, assumes 10% market share at peak sales, optimistic scenario.

Target clinical positioning

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

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





Xanamem[™]: A prime investment opportunity

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ACW financials

Key Corporate Data	
Market Cap*	~\$60 million
Entity	Public company listed on the Australia Stock Exchange (ACW)
Share Price*	0.10
Shares on issue^	~606 million
Cash position**	AU\$7.87 million
Ownership by top 20	55%

^{*}market cap and share price data as at 26 April 2016 > post placement and SPP



^{**}As at 31st December, 2015, Appendix 4D.

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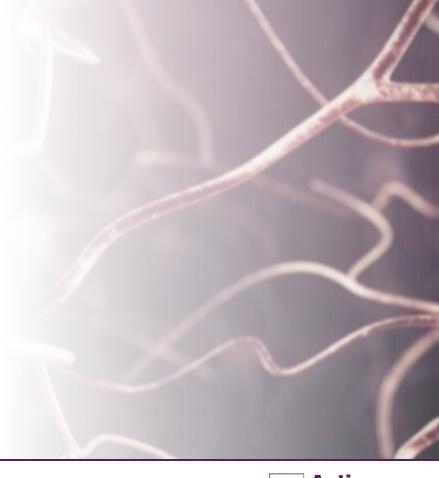
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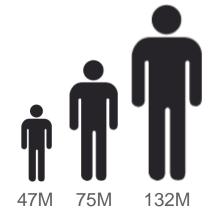


Alzheimer's disease is emerging as the most significant health challenge of our time



One person every 3 seconds

Globally there were ~10M new cases of dementia in 2015



Numbers will double every 20 years



Total cost rise to US\$2 trillion by 2030

Dementia will become a trillion dollar disease by 2018

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



Alzheimer's cannot currently be prevented, cured, or even slowed



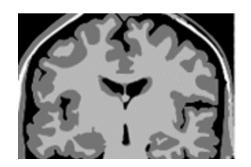
50% of 85 year olds have Alzheimer's Disease

1 in 3 seniors will die with Alzheimer's disease or other dementia

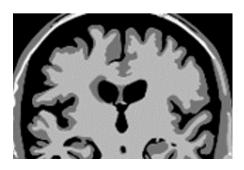
The Alzheimer's Association Facts and Figures, 2014.



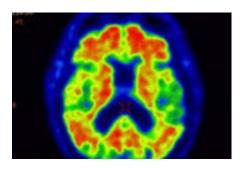
Hallmarks of Alzheimer's Disease



Normal brain (volumetric MRI)



Neural death and brain shrinkage

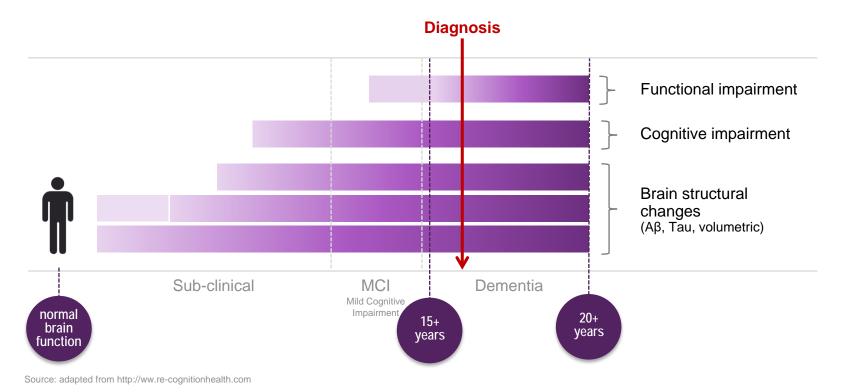


Abnormal β-amyloid plaque build up (red)

Alzheimer's research aspiration - earlier detection and treatment to slow disease progression



Disease progression and diagnosis





Focusing on markets with high risk of progressive cognitive decline





Unmet need in Diabetes Cognitive Impairment



422M¹
Suffer from Diabetes globally



of dementia 14.77M² suffer from dementia

Twice the risk



No-one is looking for a solution



¹ WHO Diabetes Fact Sheet March, 2016.

² Biessels et al., 2006.

Unmet need in Parkinson's Disease Dementia



7-10M¹
Living with Parkinson's Disease



31%
Progress from MCI to dementia
100% penetrance after 10 years



relief
Treatments are short term

symptomatic



¹ Parkinson's Disease Foundation http://www.pdf.org/en/parkinson_statistics. Medtrack Report, 2015.