Developing **Xanamem**™ for Alzheimer's Dementia

Dr. Bill Ketelbey CEO, Actinogen Medical

Investor Presentation April 2015







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Actinogen Medical - Overview



Developing a novel treatment for Alzheimer's disease (AD) and its prodromal stage mild cognitive impairment (MCI)

 Lead compound Xanamem[™] blocks production of cortisol – the stress hormone – in the brain. A novel mechanism of action

Successful early development – Phase II planning underway

- Positive results in pre-clinical and first Phase I study early development funded by the Wellcome Trust
- Second Phase I study and final pre-clinical study underway results due mid-2015.
 Research fully funded.
- Phase II study planned for 2016 in AD and prodromal AD/MCI
- US FDA's designation of Mild Cognitive Impairment as an indication shifts the landscape for AD drug development to much earlier treatment

Alzheimer's - a significant and growing unmet medical need

- AD population expected to triple over the next generation, increasing prevalence underpinned by shifting age demographic
- American Alzheimer's Association estimates the direct healthcare cost in 2013 of US\$250bn



Board and Management



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



Martin Rogers Chairman

- Biotechnology entrepreneur and executive
- Non-Executive Director of OncoSil (ASX:OSL), Chair of Rhinomed (ASX:RNO) and Non-Executive Director of Cellmid (ASX:CDY)



Bill Ketelbey CEO

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



Vince Ruffles VP Clinical Research

- Extensive drug development experience over 20 years
- Responsible clinical development and regulatory strategy



Jason Loveridge Non-Executive Director

 Former head of Nomura Life Sciences Fund in the UK with 28 out of 34 investment wins in investing in Biotech



Anton Uvarov Non-Executive Director

- Healthcare and biotech equities analyst, formerly Citibank NY
- Executive Director of Sun Biomedical



A significant unmet need



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

- A person develops AD almost every minute in the US¹
- AD cost the US healthcare system \$US250 billion in 2013
- Estimated to increase to **US\$1 trillion** by 2050, outstripping the cost of treating all other diseases
- Current treatments are of limited benefit. New and alternative treatments are desperately needed
- Xanamem[™] has the potential to be a multi-billion dollar product

¹Alzheimer's Association- Facts and Figures 2014) http://www.alz.org/downloads/Facts_Figures_2014.pdf?utm_content=bufferb49b5&utm_m edium=social&utm_source=twitter.com&utm_campaign=buffer







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

CANNOT BE PREVENTED, CURED OR EVEN SLOWED.



1 in 3 Seniors

DIES WITH ALZHEIMER'S

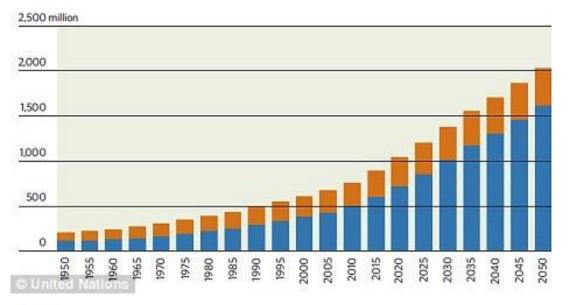
or another dementia.



AD consequence of a rapidly ageing population



Number of people aged 60 or over: World, developed and developing countries, 1950-2050



Developed countries

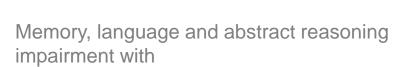
Developing countries

Source: UNDESA, World Population Ageing 2011 (2012; forthcoming), based on UNDESA Population Division medium projection scenario, World Population Prospects: The 2010 Revision. Note: The group of 'developed countries' corresponds to the "more developed regions" of the World Population Prospects: The 2010 Revision, and the group "developing countries" corresponds to the "less developed regions" of the same publication.

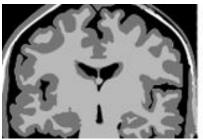
- Affects nearly 36 million patients worldwide
- In Australia, there are currently 320,000 AD sufferers – by 2050, this is expected to rise to close to 1 million.
- Commonly diagnosed in patients in their 60's, with 25% of 85 year olds and up to 50% of 95 year olds developing the disease
- AD is the second leading cause of death in Australia.

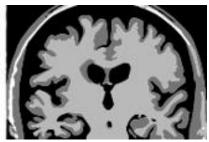


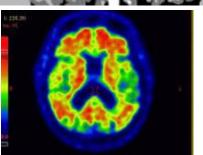
The hallmarks of AD

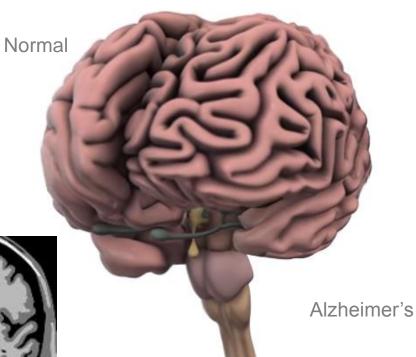


- brain shrinkage particularly hippocampus and cortex
- neuronal loss
- amyloid plaques
- neurofibrillary tangles











Signs of AD



Dementia is typically documented by decreasing performance on neuropsychological tests assessing memory, general knowledge, language, abstract reasoning and the ability to perform particular tasks requiring minimal skill:

'Please draw a clock. Put the hours on it and set the time at 2:45'



Score 10: Normal



Score 8:
Mild Cognitive
Impairment
(numbers
error and
placement of
hands)



Score 4: Moderate cognitive impairment



Score 2: Severe cognitive impairment



Overview of Xanamem™



Xanamem[™] - under development as a treatment for Alzheimer's disease and prodromal Alzheimer's/mild cognitive impairment



THOMSON REUTERS

recently named Xanamem[™] as one of the top five drugs in Phase 1 development in the global pharmaceutical or biotech industries

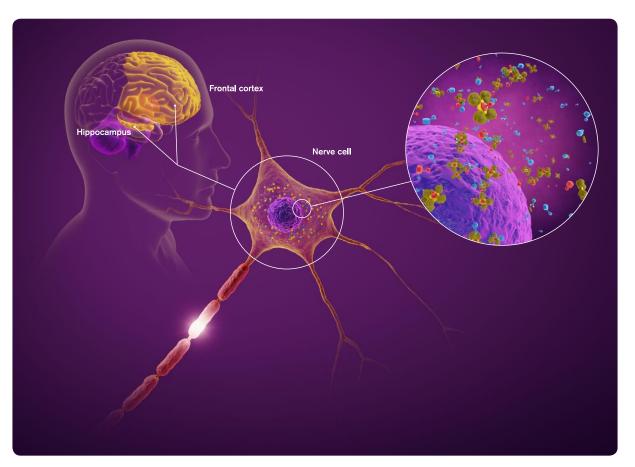
- A novel mechanism of action blocking the production of cortisol in the brain
- Excess cortisol (the stress hormone) has been shown to lead to reversible memory loss, amyloid plaques and neural death – hallmarks of AD
- Link between excess cortisol and cognitive performance identified in patients with Cushing's disease, Alzheimer's, depression and normal aging
- Early development of Xanamem[™] funded by the Wellcome Trust: \$25m over seven years
- Second Phase I study underway data expected mid-2015
- Phase II to target early and prodromal AD.
- Expected to be synergistic with other AD therapies marketed and in research
- Patent protection to 2031



Mechanism of action: a key differentiator



Xanamem™'s novel mechanism of action sets it apart from other AD treatments



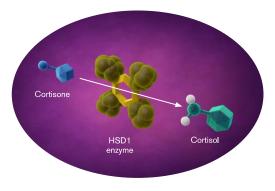


Mechanism of action: a key differentiator

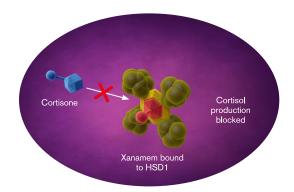


Xanamem™'s novel mechanism of action sets it apart from other AD treatments

- The HSD1 enzyme activates cortisone, producing cortisol – the stress hormone
- Xanamem[™] blocks the HSD1 enzyme and prevents production of cortisol
- Excess cortisol contributes to the memory loss, amyloid plaques and neural death associated with AD
- The HSD1 enzyme is most concentrated in the hippocampus and frontal cortex of the brain – the areas most impacted by AD
- Pre-clinical and clinical data suggests Xanamem[™] has the potential to treat AD and its early prodromal stage, mild cognitive impairment and significantly alter the course of the disease



HSD1 enzyme actives cortisone producing cortisol



Xanamem[™] binds to HSD1, blocking cortisol production



Pre-clinical data

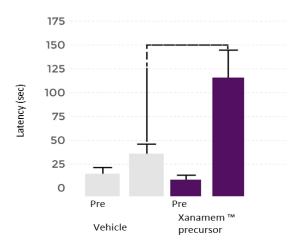


Xanamem[™]- a highly selective HSD1 inhibitor in pre-clinical animal models.

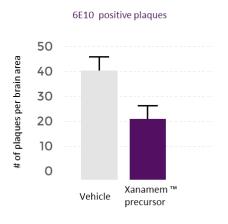
- Inhibition of HSD1 improves cognition in ageing and AD models
- Inhibition of HSD1 reduces Aβ plaque burden and plasma Aβ in AD models

Cognitive Enhancement with Xanamem™ in AD (Performance in Passive Avoidance Test, treatment for 28 days)

Xanamem[™] reduces number of Aβ plaques in AD brain (28 day treatment)



AD - progressive cognitive decline



AD - associated with amyloid plagues in the brain



Clinical trials overview



Second Phase 1 study in healthy volunteers underway

- Trial conducted at Linear Clinical Research, Sir Charles Gairdner Hospital Perth, Western Australia.
- 40 healthy volunteers enrolled across 3 studies
- First study to confirm how the body absorbs and metabolises Xanamem[™] and the optimal dose
- Two follow-on studies:
 - Fast-fed study in a cohort of 12 patients
 - Additional study of 4 patients to confirm the central nervous system pharmacokinetics of Xanamem™
- Trial on-time and on-budget. Full results expected by mid 2015

Phase II efficacy and safety study in patients with early and prodromal AD/ mild cognitive impairment. Planned for 2016 in US, Australia and UK







Xanamem[™] Clinical Advisory Board



Powerhouse Advisory Board to drive Xanamem™'s clinical development. World experts to help design the optimum Phase II efficacy trial for Xanamem™ in early and prodromal Alzheimer's patients



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



Pipeline

Xanamem™'s novel mechanism of action – blocking excess cortisol production – offers many additional possible applications relevant to diseases of the central nervous and endocrine/metabolic systems

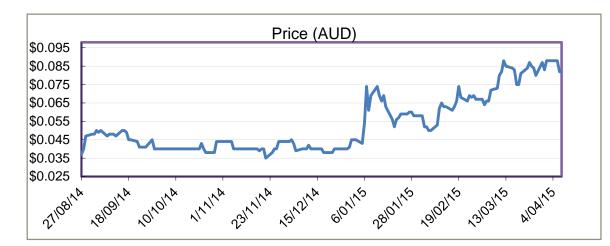
- Potential development opportunities:
 - Cognitive dysfunction in schizophrenia, depression
 - PTSD (post traumatic stress disorder)
 - Diabetes cognitive dysfunction and treatment
 - Cardiovascular disease
 - Post myocardial infarction
 - Obesity
 - Neuroprotection in metabolic disease

cortisolession diabetes diabet



Financial profile





Key Corporate Data:	
Market Cap:	\$41.34m
Share Price	\$0.084
Cash as of 31 Dec 2014	\$2.05m
Shares on issue	492m

Top Ten Shareholders	Percentage
Edinburgh Technology Fund Limited	9.78%
JK Nominees Pty Ltd	7.05%
Tisia Nominees Pty Ltd	6.83%
Mr Martin Rogers	5.08%
Warmbi SARL	4.41%
Denlin Nominees Pty Ltd	4.06%
Mr Jason Peterson & Mrs Lisa Peterson	3.56%
Webinvest Pty Ltd	3.35%
Oaktone Nominees Pty Ltd	2.99%
Dr John William Ketelbey	2.48%



Milestones



Second Phase I trial

✓ initiated

Results for Phase I trial and final preclinical study

Phase II efficacy study in patients with AD and Mild Cognitive Impairment

Final	
re-clinical	

EARLY-2015

✓ initiated

study for IND

MID-2015

Establishment of Xanamem™ Clinical Advisory Board

√ completed

2016

Phase II study and protocol design



Investment highlights

- Xanamem[™] a potential treatment for early and prodromal AD/mild cognitive impairment
- Significant unmet need in a huge and growing global market
- Novel mechanism of action, targeting the stress hormone cortisol – a key differentiator
- Hypothesis backed by good pre-clinical and clinical evidence. Early development funded by Wellcome Trust
- Final Phase I and preclinical results due mid-2015; funded through to completion of these studies
- IND filing and Phase II study planned for 2016
- Expected to be synergistic with other AD therapies marketed and in research
- Patent protection to 2031
- Tight capital structure with top 20 shareholders owning more than 70%





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Thank you and questions