Actinogen Ltd

ACQUISITION OF

Corticrine Ltd

A UK DEMENTIA COMPANY

Today, someone in North America develops Alzheimer's Disease every 68 seconds. By 2050, there is expected to be one new case of AD every 33 seconds

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Post Acquisition and Settlement

Corporate Overview

ASX Code:	ACW
Share Price:	$0.02
Market cap:	$8.6m
Cash:	$3.0m
Shares on issue:	427.6m
Options:	54m

Acquisition Breakdown

	Actinogen Ltd	Corticrine Ltd
Shares	202.6m	125m
Options	54m @ 2c	
Cash	approx. $3.0m	
$2.0m placement @ $0.02 to issue 100m shares		

Board of Directors

Mr. Martin Rogers
Non-Executive Chairman

Currently Non-Executive Chairman of Oncosil Ltd, Non-Executive Chairman of Rhinomed Ltd, Non-Executive Director of Cellmid Ltd

Dr. Jason Loveridge
Non-Executive Director

Formerly at JAFCO Nomura, currently Non-Executive Director of Resonance Health Ltd

Dr. Brendan de Kauwe
Non-Executive Director

Currently Non-Executive Director of Virax Ltd

Dr. Anton Uvarov
Non-Executive Director

Formerly Healthcare Equities Analyst at Citigroup (US), currently Executive Director of Sun Biomedical Ltd

Major Shareholders

Corticrine Ltd Founders	~ 35%
University of Edinburgh	~ 11%
Top Twenty	~ 70%

Assets

100% of Corticirine Ltd	UE2343 for Dementia associated with Alzheimer's Disease
Actinogen Library	Anti-CSCs (cancer stem cells)
Actinogen Library	Antibiotics for MRSA infections

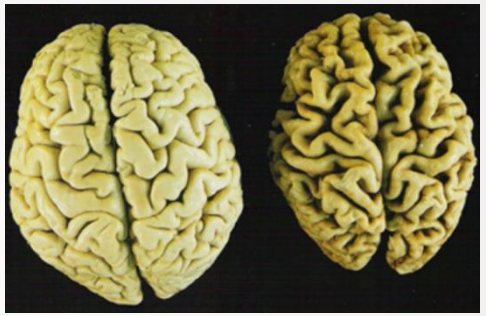
Executive Summary

- Corticrine is a mid-stage pharmaceutical R&D company focused on development of novel treatments for Alzheimer's disease
- Corticrine is a spin-out company from Edinburgh BioQuarter, the commercialisation arm of the College of Medicine and Veterinary Medicine of the University of Edinburgh in the United Kingdom. The University received significant support from the Wellcome Trust's Seeding Drug Discovery program to advance UE2343 into clinical development
- UE2343 is a patented (year 2028 and above) inhibitor of 11β-HSD1 – a novel target for AD
- UE2343 has been developed to target CNS
  - Disease modifying effects observed in pre-clinical models with UE inhibitor
  - Pre-clinical and clinical proof of concept for cognition obtained for UE inhibitor
  - Disease modifying effects observed in pre-clinical models with UE inhibitor
  - UE2343 successfully completed single dose Phase I study in humans

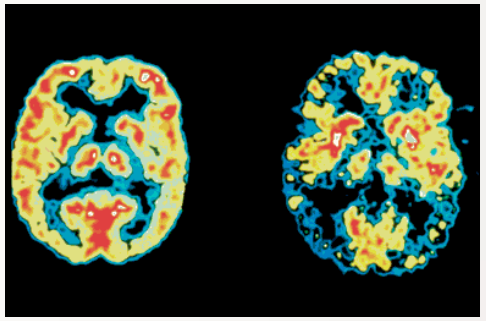
Alzheimer's Disease & Dementia

- Alzheimer's dementia is a degenerative brain disease around loss of memory and also the loss of use and understanding of language
- There is no known cure or treatment to slow progression of the disease
- It takes a disastrous toll on not only the patient but everyone around them
- Patients are robbed of their independence, their relationships and their very identity

Healthy Alzheimer's



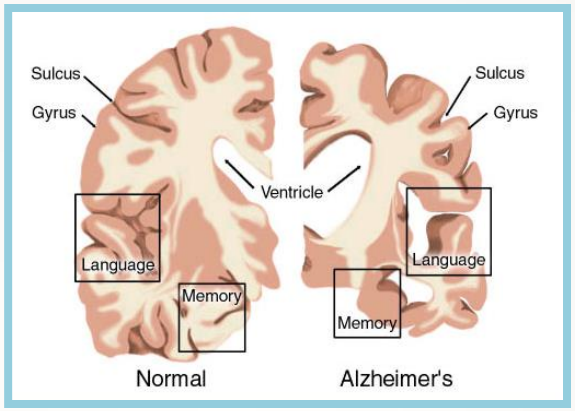
Structural changes



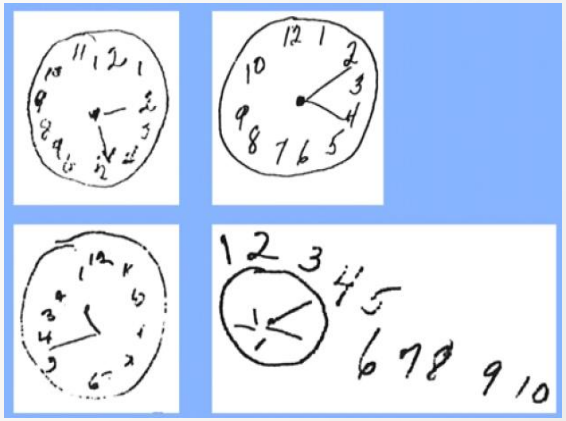
Brain activity (PET scan)

Alzheimer's Disease & Dementia

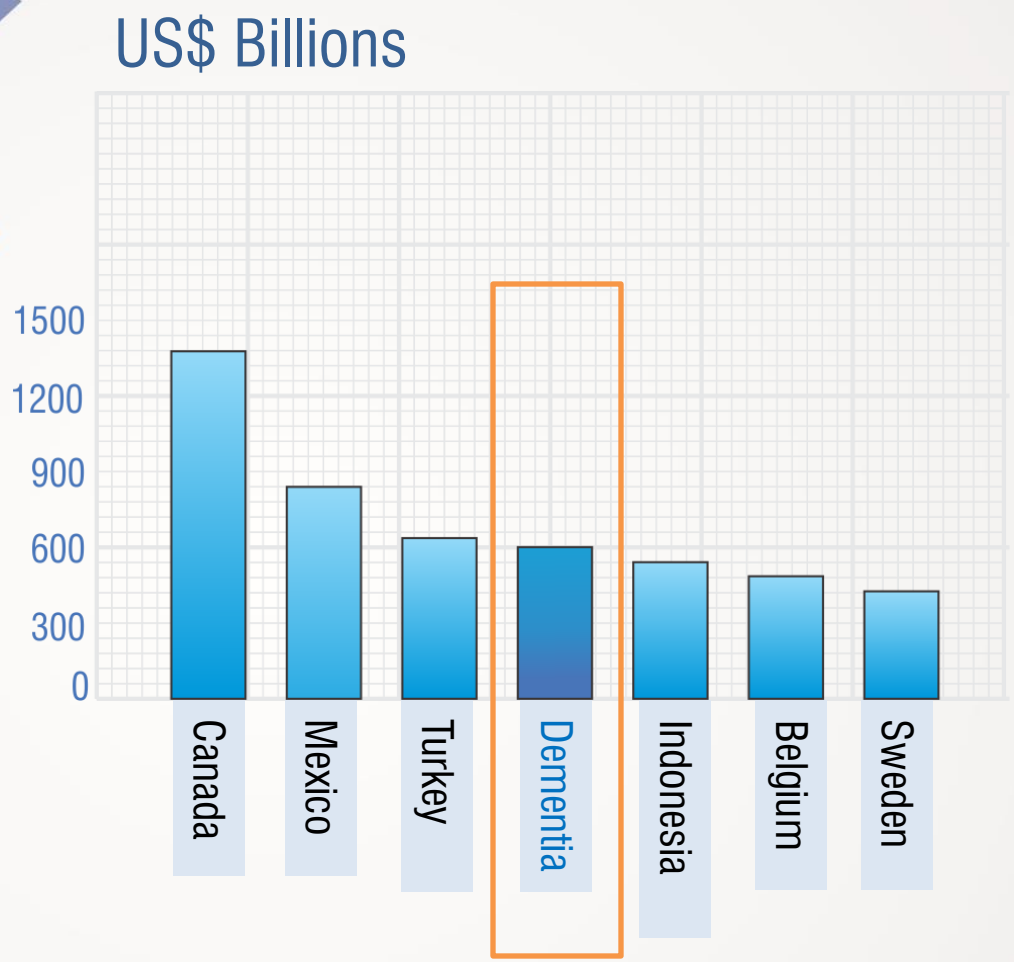
Alzheimer's disease (AD) commonly diagnosed in patients around 60 years of age resulting in progressive cognitive impairments (see areas affected). Alzheimer's dementia is associated with formation of both amyloid plaques and neurofibrillary tangles in the brain.



Dementia is typically documented by poorer performance on neuropsychological tests which assess memory, general knowledge, language, abstract reasoning and the ability to perform certain tasks of minimal skill (i.e. *'Please draw a clock. Put the hours on it and set the time at 2:45'*).

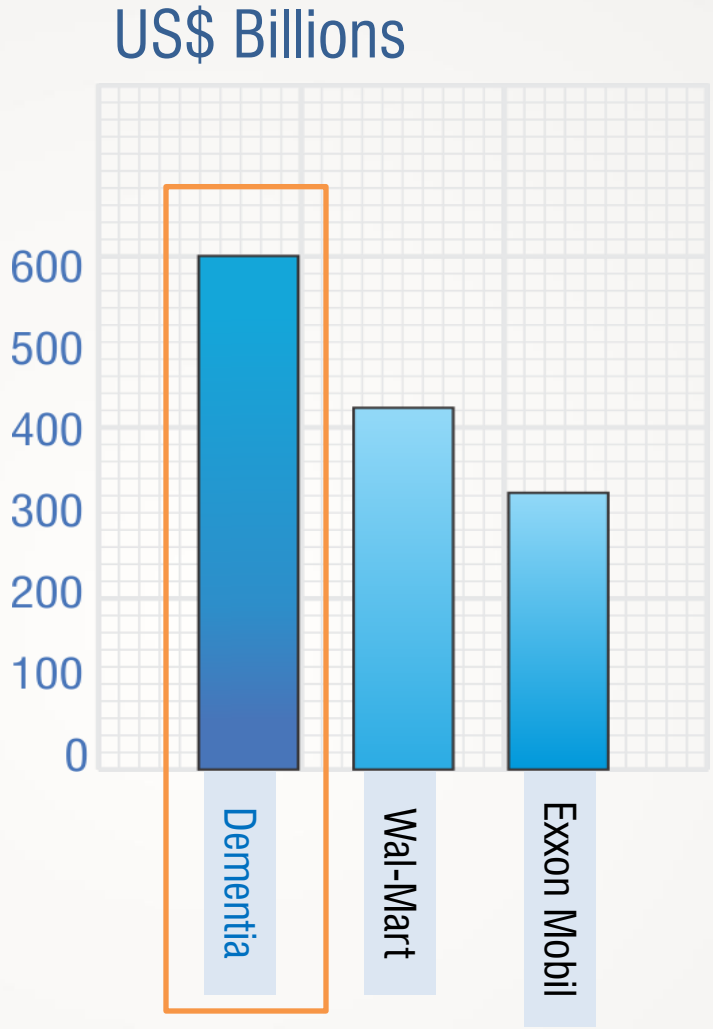


Market Opportunity



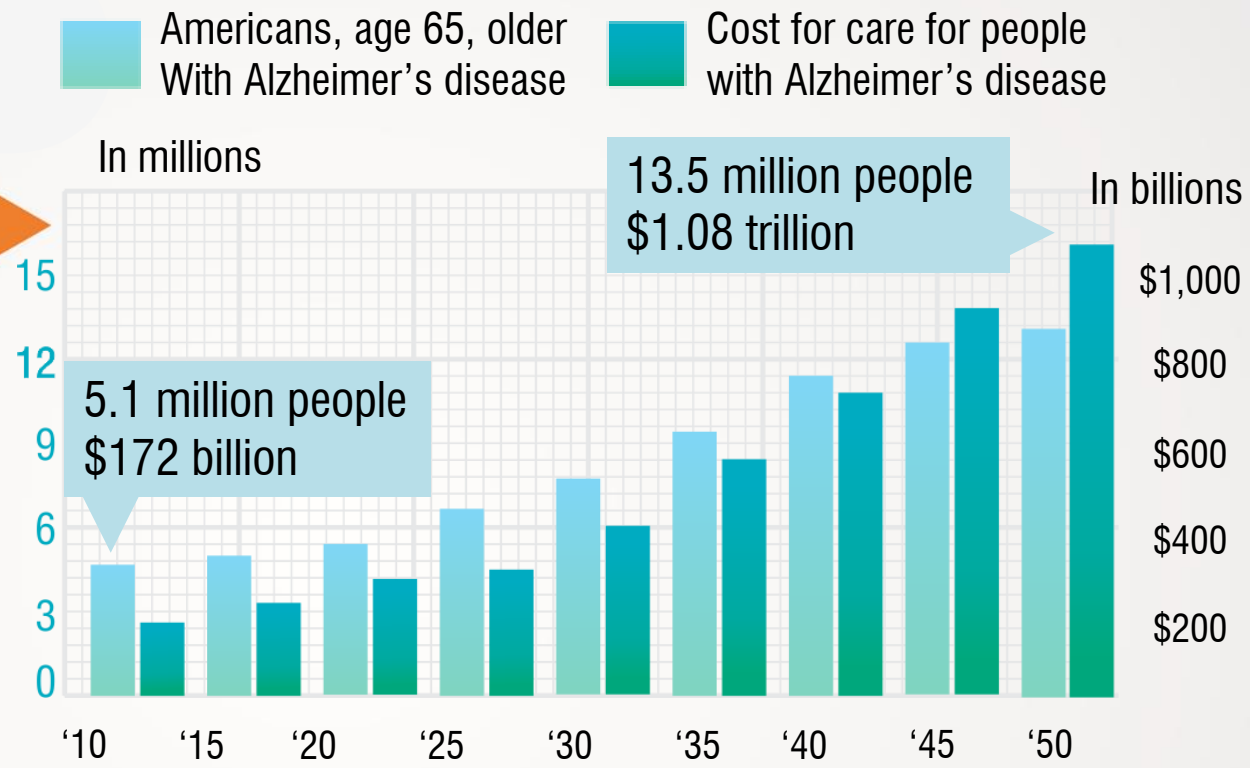
If dementia care were a country, it would be the world's 18th largest economy

Market Opportunity



If dementia care were a company, it would be the world's largest by annual revenue exceeding Wal-Mart (US$476 billion) and Exxon Mobil (US$421 billion)

Market Opportunity



US is the largest market

18m patients worldwide (according to WHO)

6th leading cause of death

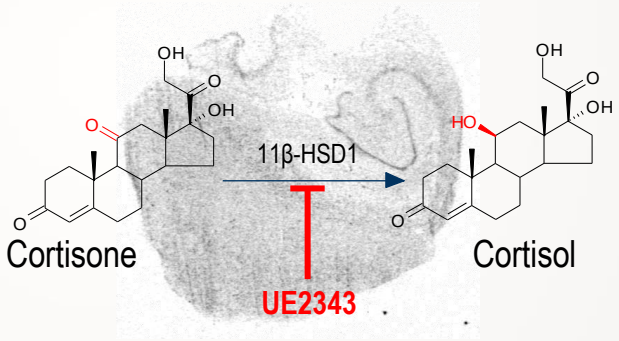
5yrs no major advancements in disease-modifying therapies

$200m will be funded towards Dementia research by Australian Government over 5 years

UE2343 -11β-HSD1 inhibition for AD

Pre-clinical proof of concept data

- 11β-HSD1 in brain is inversely associated with cognitive decline
- 11β-HSD1 knockout models are protected against age-related cognitive impairment
- Small molecule inhibition of 11β-HSD1 improves cognition in ageing and AD models
- Small molecule inhibition of 11β-HSD1 reduces Aβ plaque burden and plasma Aβ in AD models



Proof of concept in humans

- 11β-HSD1 generates cortisol in brain regions important for cognition
- Patients with cortisol excess (Cushing's syndrome) display reversible memory loss with hippocampal atrophy
- Elevated cortisol levels associate with cognitive decline in ageing and AD
- 11β-HSD inhibition with non-selective inhibitor improves cognition in humans

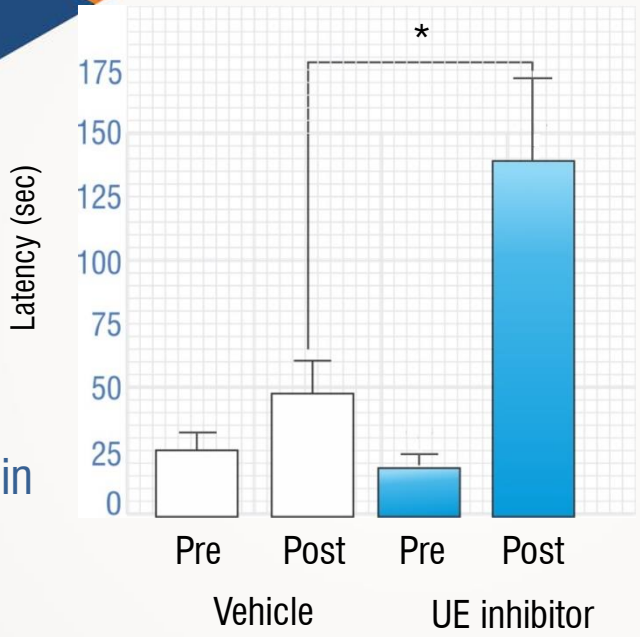
Pre-Clinical Data to Date

Disease modifying potential of UE2343

Cognitive Enhancement in AD with UE inhibitor

Performance in Passive Avoidance Test

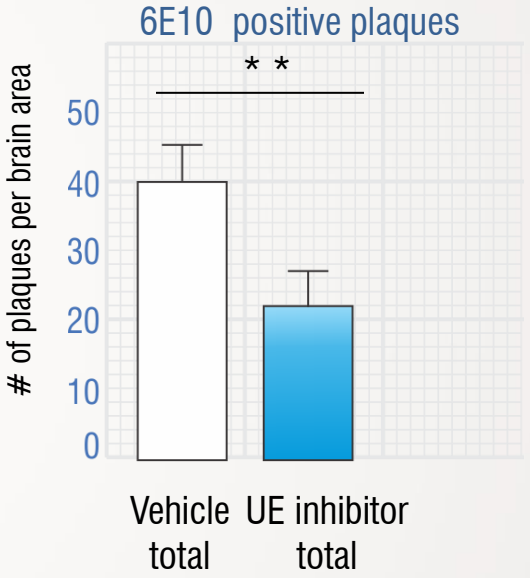
Treatment with UE inhibitor for 28 days



Symptom: AD results in progressive cognitive impairments

UE inhibitor reduces number of Aβ plaques in AD brain

treatment with UE inhibitor for 28 days

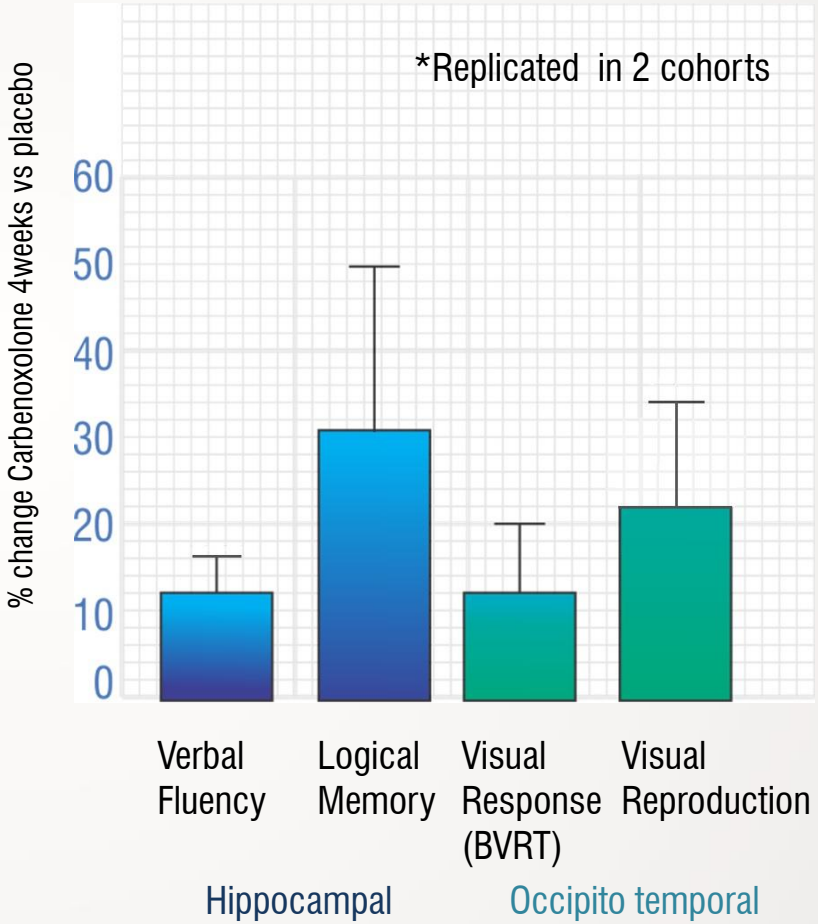


Symptom: AD is the most common form of dementia and is associated with both amyloid plaques and neurofibrillary tangles in the brain

Human Proof of Concept Completed

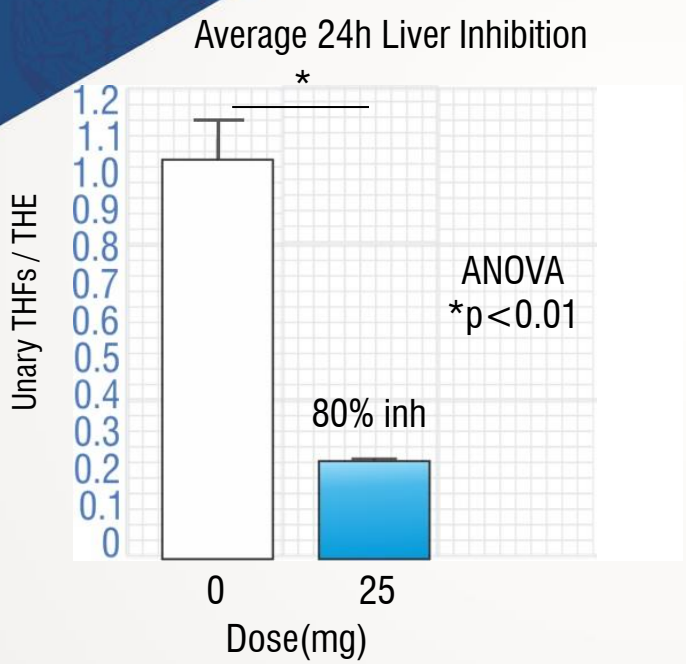
In two randomised, double-blind, placebo-controlled crossover studies, administration of the 11β-HSD1 inhibitor Carbenoxolone improved verbal fluency ($p < 0.01$) after 4 weeks in 10 healthy elderly men (aged 55-75 y, see Figure on the right) and improved verbal memory ($p < 0.01$) after 6 weeks in 12 patients with type 2 diabetes (52-70 y).

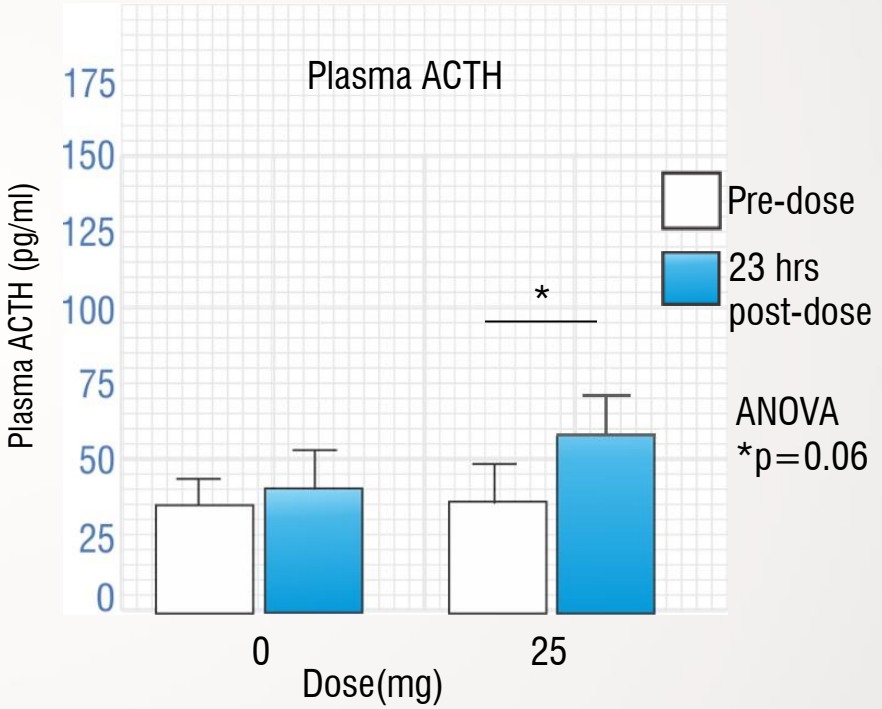
Pharmacological inhibition of HSD1with Carbenoxolone improves memory in humans



UE2343: Human Pharmacodynamic Data

UE2343 Pharmacodynamics Phase I study (Single Dose)

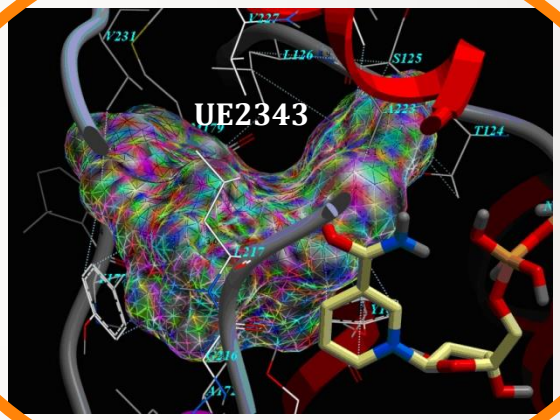




Maximal enzyme inhibition achieved over 24h with a single 25mg dose of UE2343 in Phase I study in humans

UE 2343 Development

- Synthetic process re-engineered to allow for kilogram-scale manufacture
  - Convergent synthesis
  - Crystalline product
  - Attractive cost of goods



- Kilo-scale manufacture of GMP material
  - Simple excipient blend to allow dosing in capsules
  - Excellent long-term stability of IMP (investigational medicinal product)

- Clear safety and toxicology package for clinical development
- First time in human studies conducted
  - Well tolerated, no adverse toxicities noted at any dose

Clinical Development Steps

- CTA (Clinical Trial Application) approved for multiple ascending dose (MAD) study
- PD (pharmacodynamic) biomarker validation for CNS inhibition in healthy human subjects completed
- Phase Ib fast-fed study
- Phase IIa symptomatic efficacy study in mild to moderate AD patients
  - stratified patient group
  - cognitive testing
  - functional MRI and biomarkers

Additional Indications for Future Development

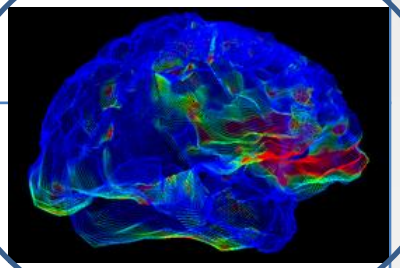
- Cognitive dysfunction in schizophrenia
- Indications beyond type 2 diabetes in cardiometabolic disease

Clinical Development Advisory Panel

Professor Brian Walker	⇝ Co-founder of Corticrine Ltd ⇝ Professor of Endocrinology and Head of the University of Edinburgh/British Heart Foundation Centre for Cardiovascular Science ⇝ Co-chair of the CORtisol NETwork (CORNET) international consortium on cortisol research ⇝ A member of the Wellcome Trust's Clinical Interview Committee
Dr Scott Webster	⇝ Co-founder of Corticrine Ltd ⇝ Currently a Director, Drug Discovery Core, College of Medicine and Veterinary Medicine, University of Edinburgh ⇝ A drug discovery advisor on a Wellcome Trust Seeding Drug Discovery program
Dr Jason Loveridge	⇝ Co-founder of Corticrine Ltd ⇝ Formerly Investment Director with JAFCO Nomura ⇝ Participated in the start up of over 24 companies in Europe, the US and Israel
Professor Alan Boyd	⇝ Co-founder of Corticrine Ltd ⇝ 30 years' pharmaceutical career with Glaxo Group Research Ltd. ⇝ Formerly Head of Medical Research for Zeneca Pharmaceuticals (now Astra Zeneca) ⇝ Vice-President of the Faculty of Pharmaceutical Medicine, Royal College of Physicians, UK

Proposed Phase I MAD study

Participants	20 Healthy Volunteers
Timetable	Study to commence early 2015
Intervention	Randomised double-blind crossover of placebo versus UE2343 at 3 doses UE2343 multiple doses predicted from MAD to obtain 24 CNS exposure above administered steady state
Study Outcome	Pharmacokinetics (PK), systemic Pharmacodynamics (PD) and toxicology
Secondary Outcome	Change in hippocampal signal during memory task Inhibition of systemic HSD1 measured by whole body D3-cortisol generation rate



Key Investment Highlights

- UE2343 was discovered in 2007 at the laboratory of Professor Brian Walker at the University of Edinburgh. Subsequently, the University received significant support from the Wellcome Trust's Seeding Drug Discovery program to advance UE2343 into early clinical development

http://clinicaltrials.gov/show/NCT01770886

- Extensive patent portfolio with patents protected until 2028 and beyond
- Successfully completed Phase Ia single ascending dose study in humans
- Experienced Management Team and Clinical Development Advisory Panel
- Planned commencement of Phase I PD/PK/safety study early in 2015
- Potential commencement of Phase IIa efficacy study in late 2015 / early 2016

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