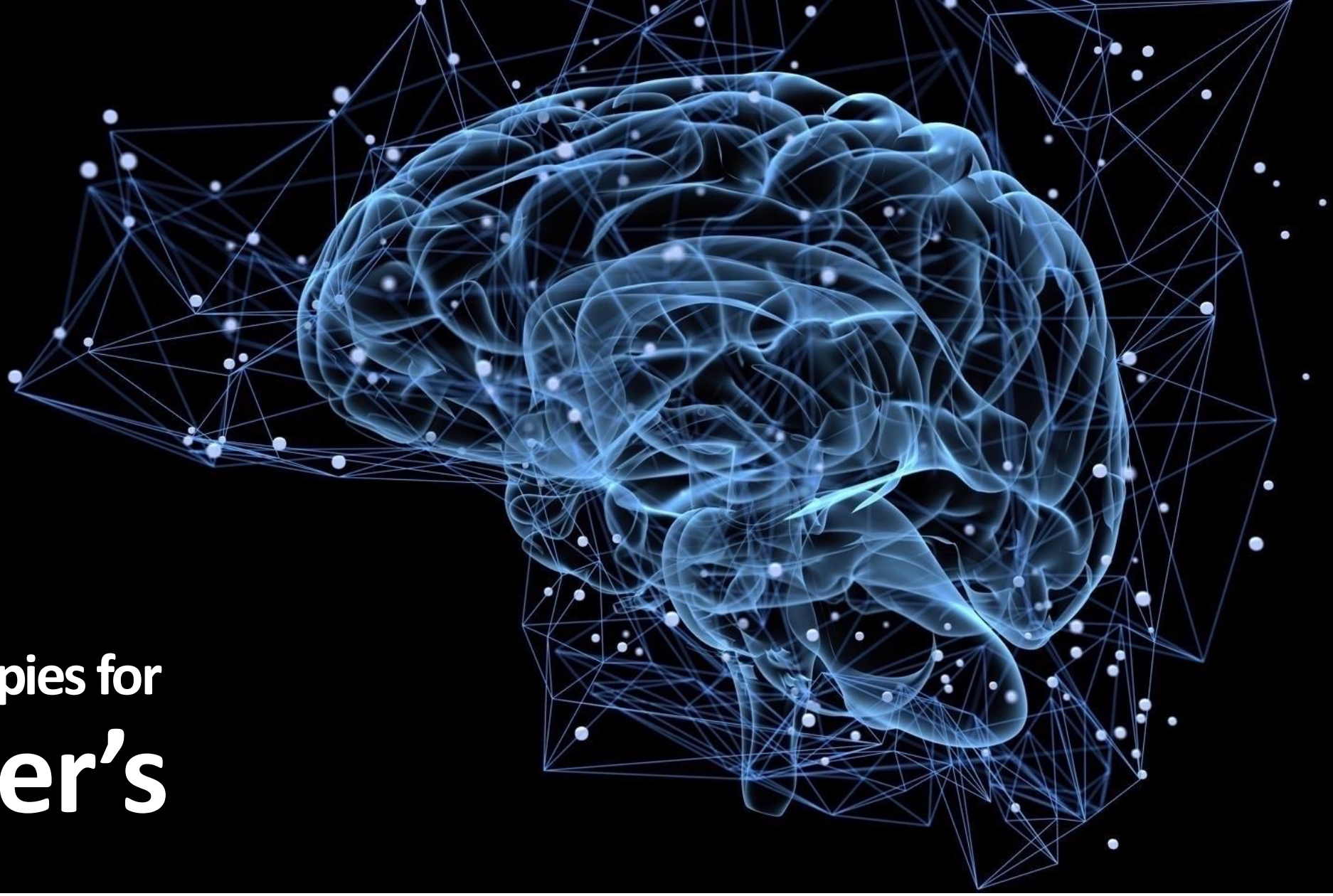
April 9, 2026



Developing therapies for Alzheimer's

European Innovation Council

AlzeCure

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AlzeCure

AlzeCure Pharma in brief

AlzeCure

AlzeCure Pharma – In brief

- Working in **Alzheimer's Disease** (AD) & **Pain** – high unmet medical need & multi-billion sales potential
- Spin-out from **AstraZeneca** – as a result of their exit from the CNS area
- Founded in 2016, out of a research foundation sponsored by **Alzheimerfonden**
- Based at Novum Science Park, **Karolinska Institute**, Stockholm, Sweden
- Three project platforms with multiple **small molecule** candidates with **first-in-class properties**
  - **NeuroRestore®** – A novel symptomatic treatment for cognitive disorders, e.g. AD with disease modifying potential
  - **Alzstatin®** – An innovative preventive & disease-modifying treatment against AD
  - **PainLess** – Innovative projects for osteoarthritic & neuropathic pain
- Listed on **Nasdaq First North Premier** Growth Market, Sweden, (Ticker: ALZCUR)
- Market cap: **183 MSEK** (260407)
- **Cash position: 50.3 MSEK** (Q4 2025)
- **European Innovation Council** (EIC) **grant** of **27.5 MSEK** (€ 2.5M) for phase IIa Alzheimer's trial

Professor Jan Lundberg – Board member

Former global head of R&D at Eli Lilly & global head of research at AstraZeneca joining & investing

Professor Jan Lundberg

Jan Lundberg, Professor in neurotransmission

Professor Jan Lundberg has more than 25 years of experience from leading positions in global pharmaceutical companies, including as global head of research at Astra and then AstraZeneca (1996 – 2009) and **global head of research & development** at Eli Lilly (2010 – 2018). He has led the development of more than 200 drug candidates, including Alzheimer's & pain, with 30 approved products in several therapeutic areas.

Before joining Astra, Dr. Lundberg was professor of pharmacology at the Karolinska Institute, where he also received his PhD. His research has resulted in over 500 publications in peer-reviewed scientific journals.

Lilly AstraZeneca

European Innovation Council

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Our Business Model

Are a **Research & Development** company

Research & **develop through early clinical phase** and then to **out-license** or **partner** on our projects

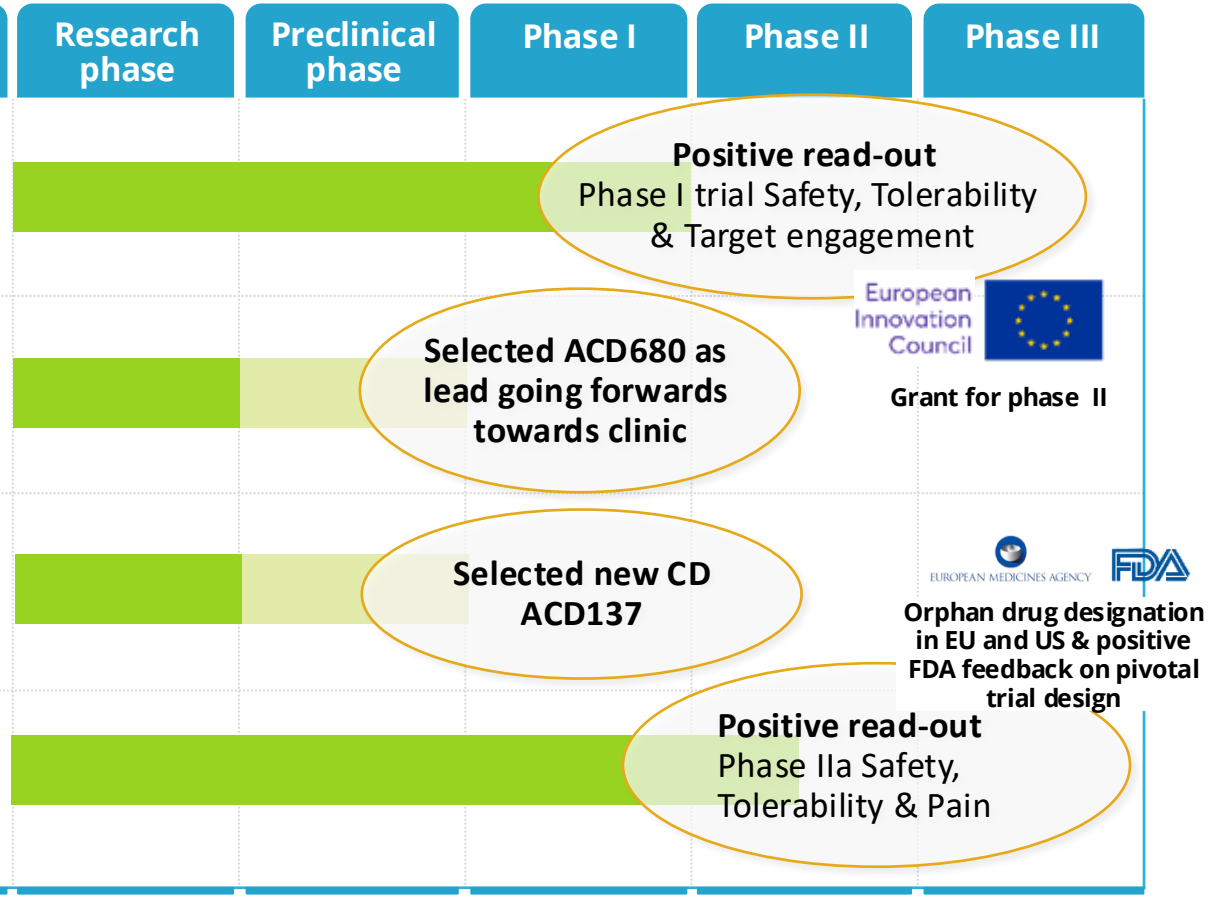
Gain incomes through:

- Upfront payments
- Milestone payments
- Royalties on sold products

A pipeline of Small-Molecule Programs

Multiple candidates increase chance of success

Platform	Candidate	Target	Indication
Neuro Restore®	ACD856	Positive allosteric modulator (PAM) of Trk-receptors)	Alzheimer's Disease, TBI - Traumatic brain injuries Parkinson's disease, Sleep disorders, Depression
Alzstatin®	ACD680	Gamma secretase modulator (GSM)	Alzheimer's Disease
PainLess	ACD137	Negative allosteric modulator (NAM) of TrkA-receptors	Osteoarthritic Pain & other severe pain conditions
	ACD440	TRPV1 antagonist	Neuropathic Pain & Orphan indication Erythromelalgia



Phase completed **Phase ongoing**

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Focus Area

Alzheimer's Disease

- Cost the society more than oncology, diabetes and cardiovascular diseases TOGETHER!!!
- The cost will triple in the next 30 years

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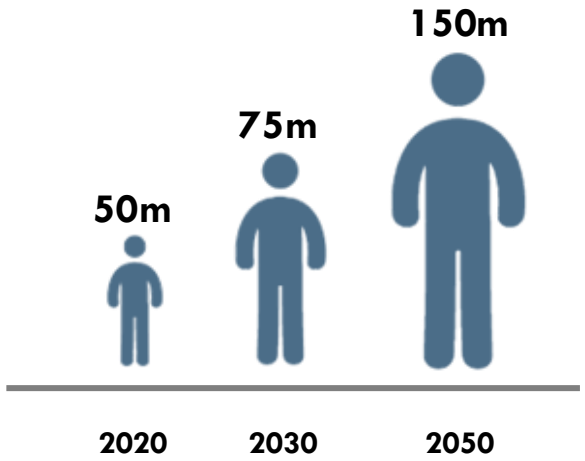
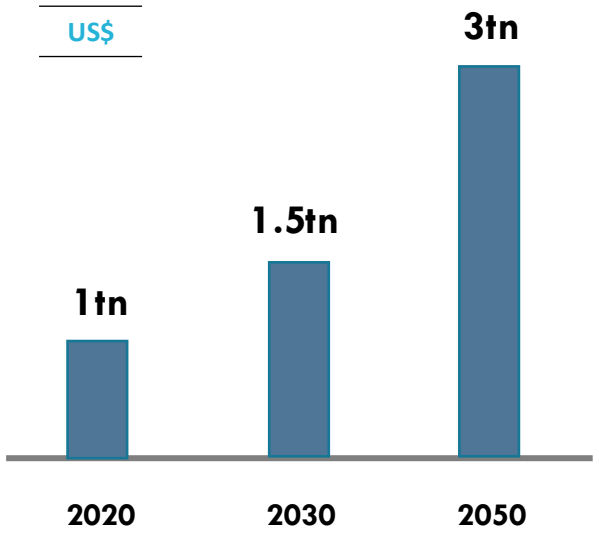
Dementia & Alzheimer is a Major Global Concern - A Tripling Patient Population & Cost





- **50 million** people worldwide live with Dementia …
- … and **doubling every 20 years**
- Alzheimer's stands for 60-70% of all cases of dementia

If **the total cost** for Dementia would be a country based on total cost in 2026, it would represent the 15th largest economy in the world

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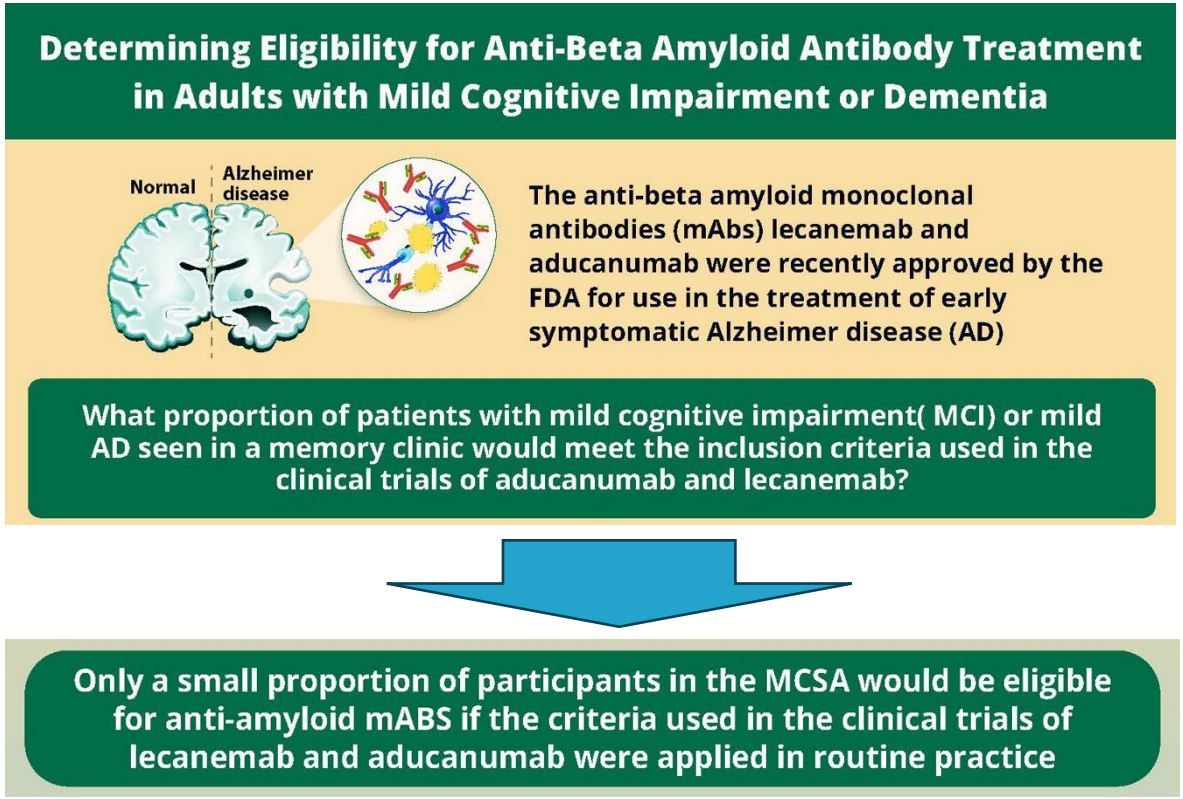
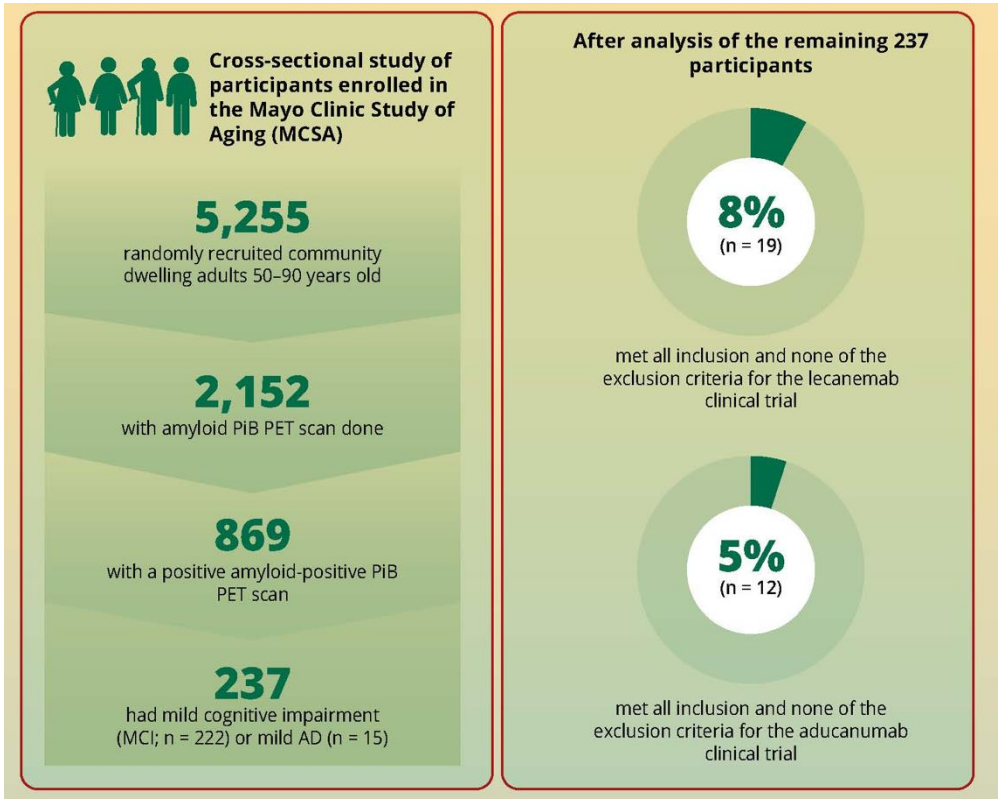
Only 5-8% of Alzheimer's patients* are estimated to be **eligible for anti-bodies treatment**





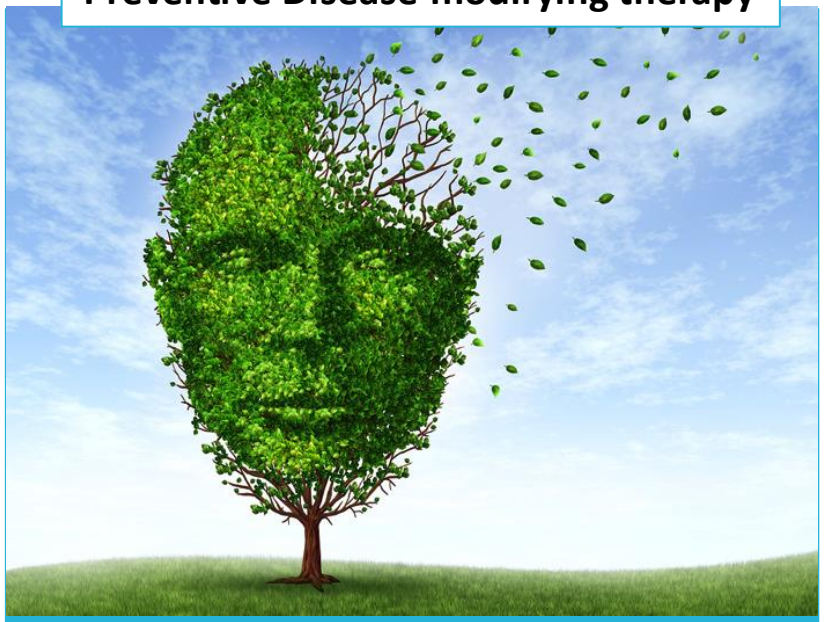
- Huge unmet medical need remains for alternative Alzheimer's treatments

*) Eligibility for Anti-Amyloid Treatment in a Population-Based Study of Cognitive Aging; Rioghna R. Pittock et al; **Neurology, 2023**;101:e1837-e1849. https://doi.org/10.1212/WNL.0000000000207770

Two Alzheimer's platforms - 1st-in-class properties & potential game-changers

MEETING NEEDS OF AD PATIENTS – ADRESSING SHORT TERM NEEDS WITH LONG-TERM BENEFITS

Preventive Disease-modifying therapy



Alzstatin®

Gamma-secretase modulator

Targeting Amyloid Production

- *Novel Oral Small Molecule*

Symptomatic & pot. Disease-modifying therapy

NeuroRestore®

Trk-PAM

Improving Neuronal & Cognitive Function

- *Novel Oral Small Molecule*

Two fast followers to **Roche**, **Eisai** respectively → Provides Concept **Validation**

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Alzstatin® – *gamma-secretase modulator targeting amyloid production for prevention and treatment of Alzheimer's disease*

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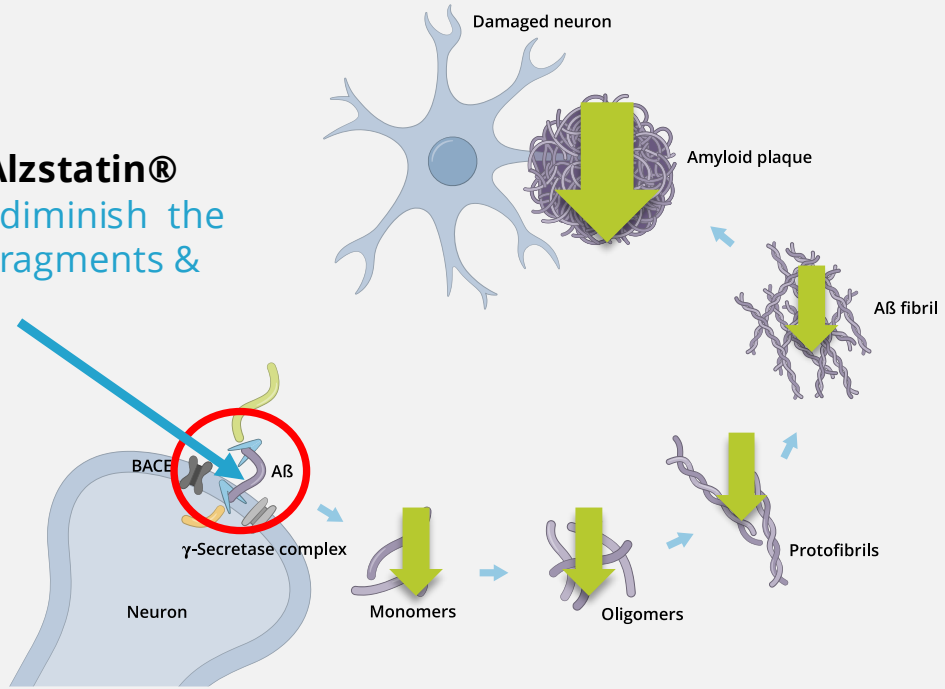
The Alzheimer's Brain and its Destruction by Toxic Protein

The Amyloid Cascade

Toxic protein formations, built up of Aβ42, are harming and destroying brain structures

AlzeCure's GSM[1] **Alzstatin® reduces Aβ42** to diminish the build-up of toxic fragments & plaque



1) GSM = Gamma Secretes Modulator – Reducing build-up of Aβ-42 and plaque

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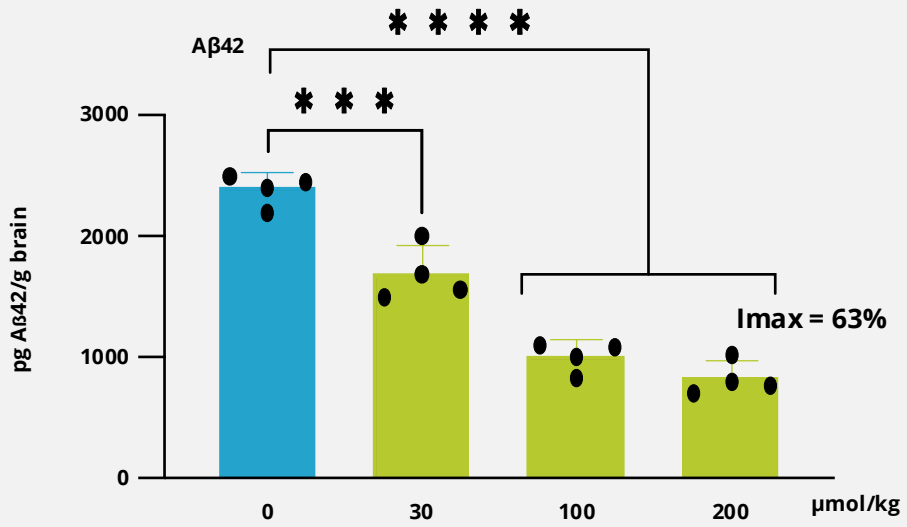
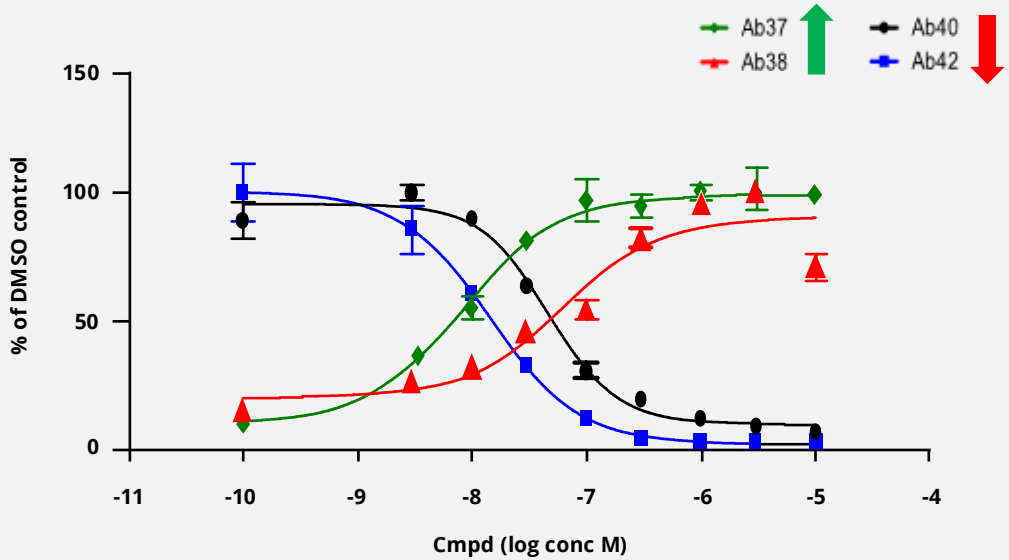
Alzstatin® – Potent Reduction of Toxic Aβ42 & Increasing Protective Aβ37 & Aβ38

Alzstatin® potently reduces the amount of toxic brain Aβ42 in animals



Alzstatin® reduces toxic Aβ42 and Aβ40, while increasing protective Aβ38 and Aβ37 peptides



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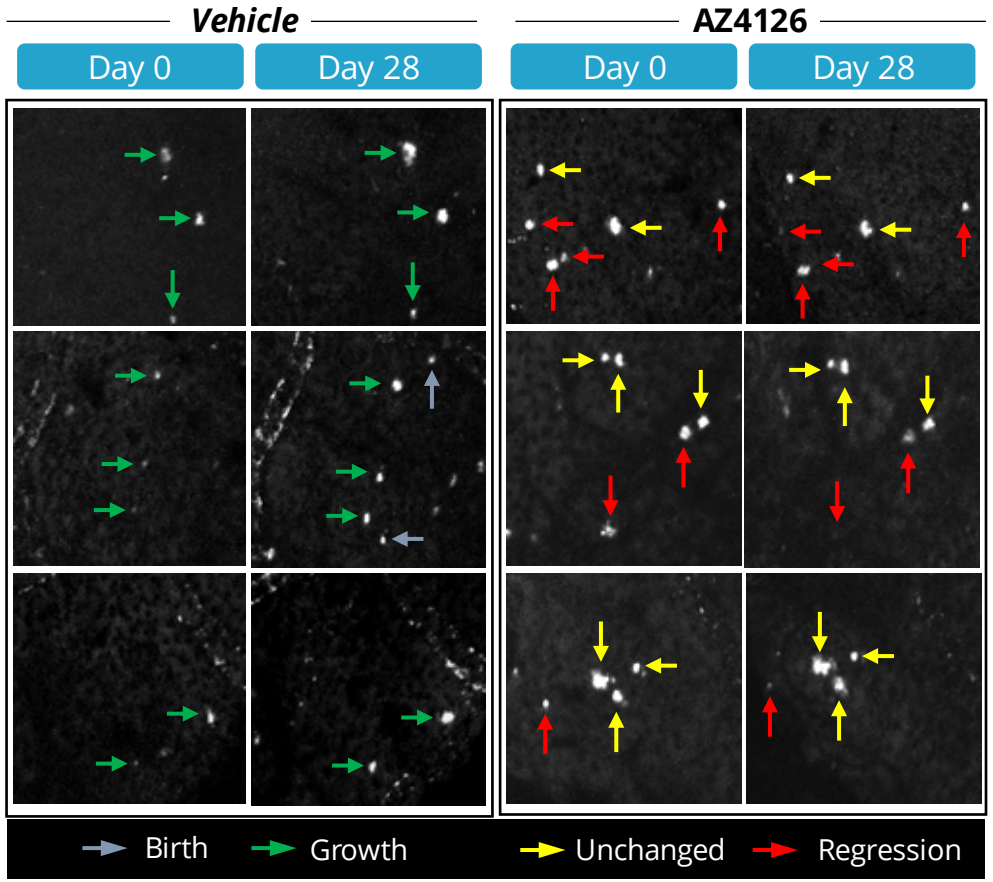
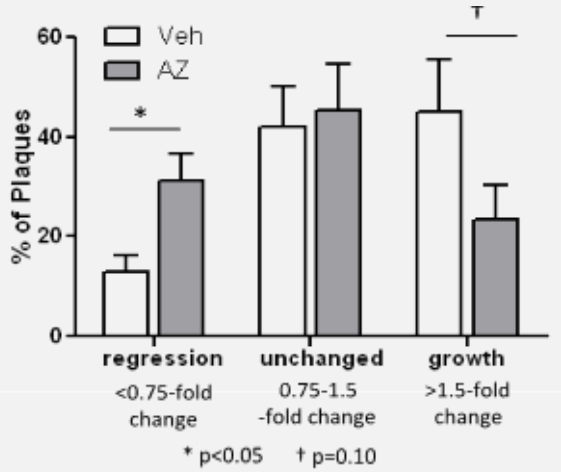
New preclinical data shows Induced Plaque Regression

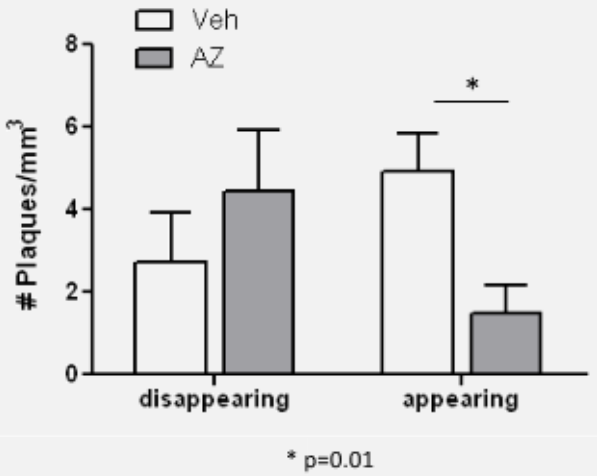
Two-photon-study preclinical study in APP/PS1 mice



Distribution of plaque growth & regression



Plaque appearance or disappearance



28-day GSM treatment

- **Reduces** amyloid **plaque growth**
- **Decreases new plaque** appearance
- **Induces plaque regression**[1]

Nordvall et al, Feb. 12, 2025: γ-Secretase Modulation inhibits Amyloid Plaque Formation and Growth and stimulates Plaque Regression in APP/PS1 Mice, JPET, Feb 12, 2025: https://jpet.aspetjournals.org/article/S0022-

1) Plaque regression not reported for Gamma- or Beta-secretase inhibitors suggest that increase in Ab37 could be responsible

Multiple Potential Treatment Populations for Alzstatin – Maintenance, Combination & Preventive Therapy

Maintenance therapy - in patients with established Alzheimer's disease

- Treatment after initial plaque clearance by monoclonal antibody treatment (as initially proposed by Lilly) e.g., with: Lecanemab (Eisai/Biogen/Bioarctic) & Donanemab (Lilly)

Combination therapy - together with monoclonal antibody treatment - early AD

- Combine plaque clearance by monoclonal antibody treatment and reduction of Aβ42 production by GSM

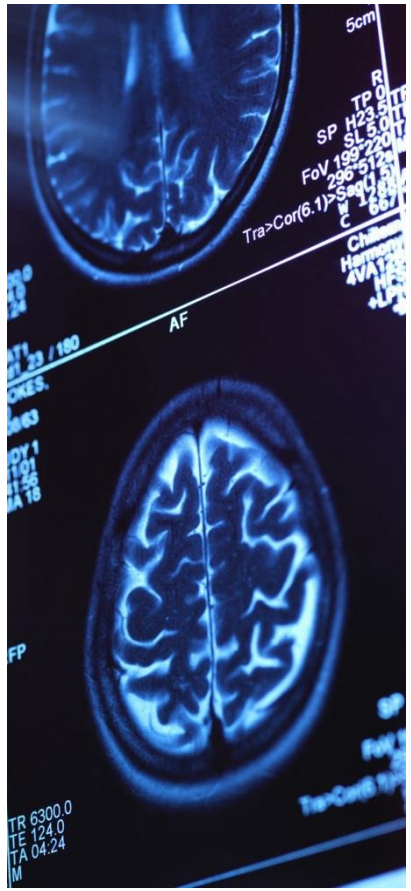
Preventive therapy - based on genetic risk factors[1] and biomarkers

- Early stand-alone treatment before onset of symptoms and any major neurodegeneration occurs (prior to rapid increase in Tau pathology)
- Prevents build-up of amyloid – an early pathological feature of AD
- Familiar forms of the disease (incl. Downs syndrome) suitable for initial proof of concept clinical studies

1) E.g. ApoE4

Early clinical Proof-of-Mechanism – Already in Phase I

- Phase I will demonstrate **proof of mechanism** (PoM) and **central target engagement**
  - SAD/MAD studies conducted in **healthy volunteers + Alzheimer's patients**
  - Evaluation of safety and tolerability after single and repeated administration – SAD/MAD study
  - Possibility to **explore biomarker effects** showing central target engagement already in **Phase I**
    - Show **reduction of toxic Aβ-species** – Aβ42/40
    - Show **increase of shorter protective Aβ-forms** – Aβ37/38
    - Measurements done both in CSF and plasma – using established **biomarker kits**



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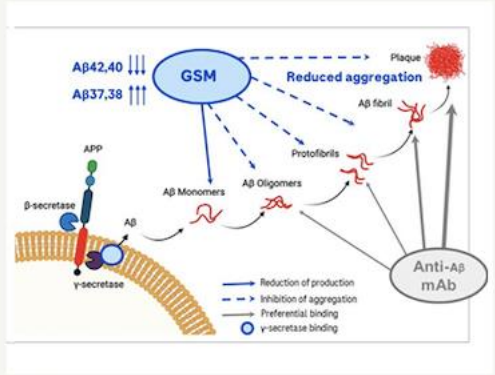
Interim phase II data on a GSM by Roche expected in 2026

RG6289, a FIC oral γ-secretase modulator in Alzheimer's disease

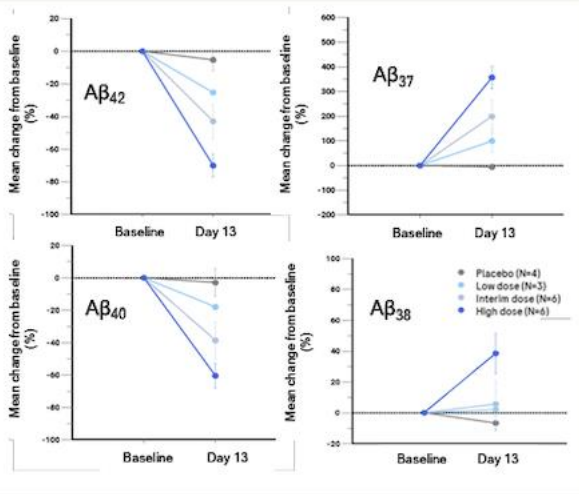
Targeting amyloid precursor protein processing to prevent Aβ-aggregation

GSMs reduce Aβ aggregation[1]



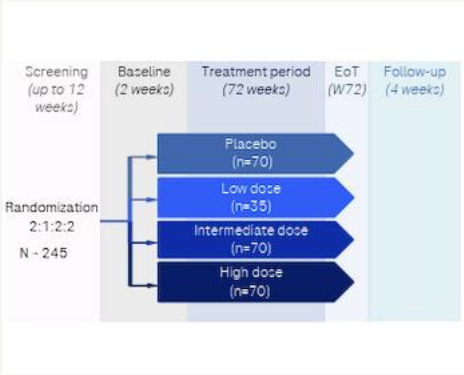
- RG6289 a potentially first-in-class oral GSM with high potency and selectivity
- GSMs alter APP processing: Reduction of Aβ42/40 and elevation of Aβ38/37
- Selective for APP with no effect on Notch
- GSMs prevent amyloid accumulation and halt plaque formation in animal model

Ph I dose escalation results for RG6289[2]



- Daily administrations of RG6289 decreased Aβ42/40 and increased Aβ37/38 concentrations in CSF of healthy volunteers in a dose dependent manner

Ph II (GABriella) study design[3]



- Ph IIa (GABriella) investigates RG6289 in individuals at risk for or at prodromal stage of AD exploring safety, tolerability and effects on AD-related biomarkers
- Interim data expected in 2026

The data is **validating & increasing interest in Alzstatin®**

NeuroRestore® – *Trk-PAM improving neuronal function & cognition in Alzheimer's disease*

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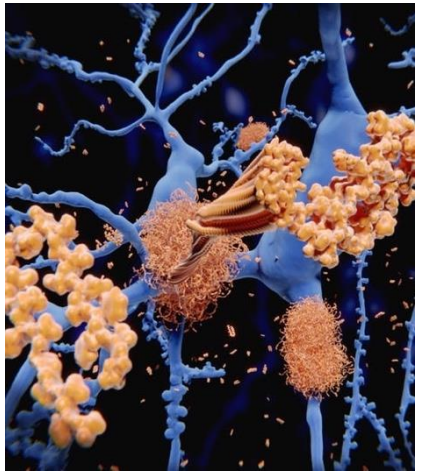
The importance of BDNF & NGF signaling in Alzheimer's

- The neurotrophins Brain-Derived Neurotrophic Factor (BDNF) and Nerve Growth Factor (NGF) bind to their respective receptors, TrkB and TrkA and are **key for brain health and cognition**
- BDNF& NGF signaling are implicated in several key neuronal functions, including **cholinergic function**, hippocampal neurogenesis and **synaptic plasticity**
- **Loss of NGF-dependent cholinergic neurons** in the basal forebrain and hippocampal atrophy are early hallmarks of Alzheimer's disease and **correlates with cognitive decline**
- **Certain genetics** in man, e.g. BDNF-Val66Met polymorphism, leads to **lower levels of BDNF,** and is associated with **more rapid cognitive impairment** and **increased disease progression** in Alzheimer's



Reduced BDNF and/or NGF-levels could limit the brain's ability to withstand pathological conditions

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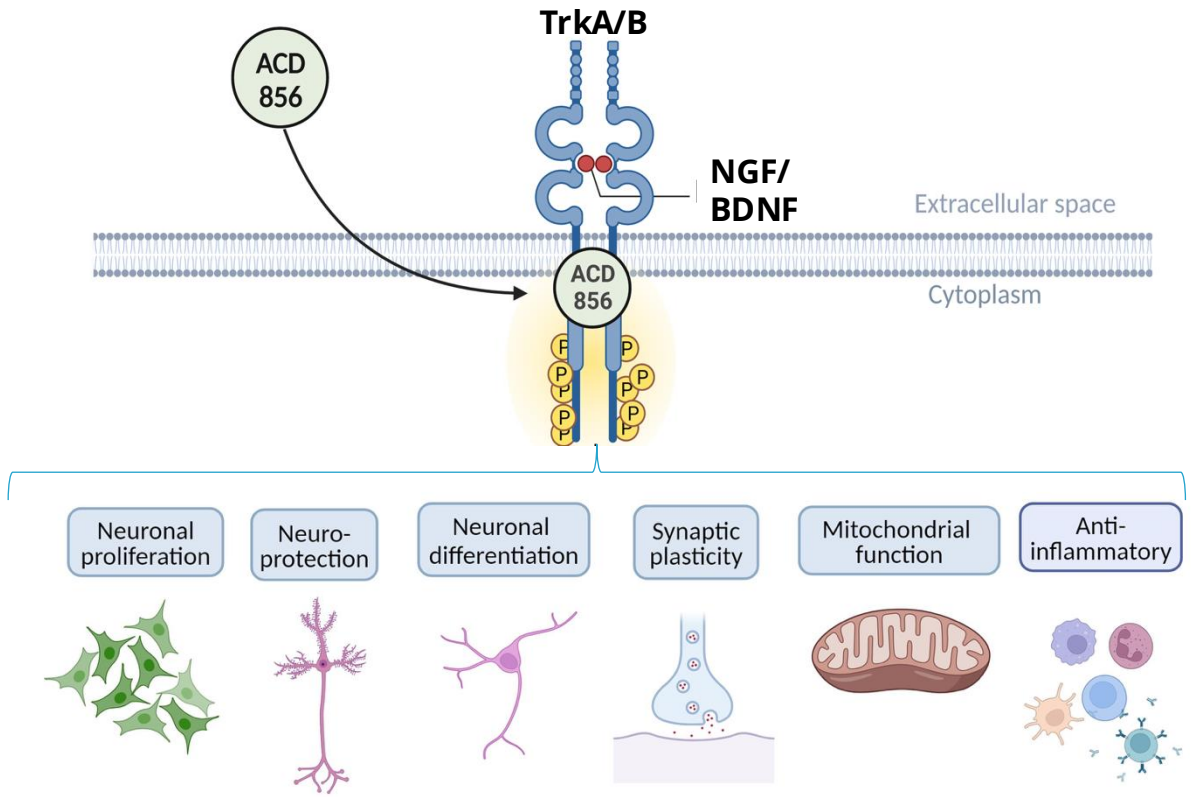
Mechanism of Action of NeuroRestore ACD856

- ACD856 is a novel oral small molecule positive modulator of Trk-receptors **enhancing the signaling** of neurotrophins such as **NGF & BDNF**
- The enhanced signaling leads to immediate **symptomatic effects** & with potential **long-term benefits**
- The long-term effects of ACD856 could include **improved neuronal function, increased mitochondrial function & enhanced cognition**, etc



Potential for **disease modifying effect & improved learning and memory**

Parrado Fernandez C et al. Int J Mol Sci. 2023 Jul 6;24(13):11159. https://doi.org/10.3390/ijms241311159
Dahlström M et al, Cells 2021, Volume 10, Issue 8, 1871. Identification of Novel Positive Allosteric Modulators of Neurotrophin Receptors for the Treatment of Cognitive Dysfunction

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NeuroRestore® – Cognitive Enhancer Improving Learning & Memory Ability

Stages of memory formation

LEARN

Transforming information into a form that can be stored in memory

STORE

Maintaining the encoded information in memory

REMEMBER

Re-accessing the information from the past which has been encoded & stored

NeuroRestore has in pre-clinical models shown that it can improve the ability to learn & remember information, so the information is accurately recollected when needed

Parrado Fernandez C et al. Int J Mol Sci. 2023 Jul 6;24(13):11159. https://doi.org/10.3390/ijms241311159

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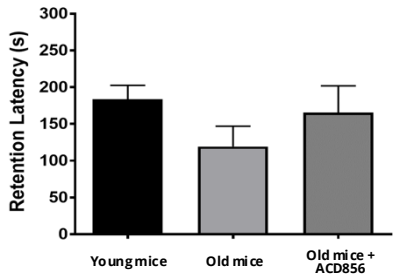
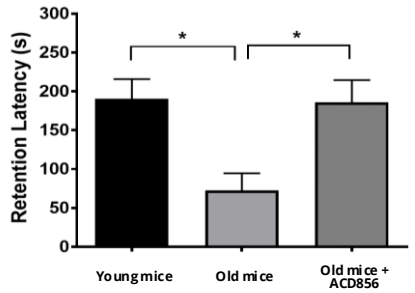
Preclinical data on Pro-cognitive & Disease Modifying Effects

ACD856 improves aged-induced memory impairment

- 18-months old animals were used to study effects on age-induced memory impairment and compared to young animals
- Two memory tests were performed 1 or 11 days after learning

Single dose ACD856	Learning	Test #1 – Day 1	Test #2 – Day 11
-30 min	t=0 min		





ACD856 could fully revert the memory impairment in old animals to the level in young animals

Supporting data

- ✓ **Improves memory impairment**, presumably via increased synaptic function/plasticity
- ✓ **Increases the levels of BDNF** in cortical neurons and in brains of aged mice
- ✓ **Enhances neurite outgrowth** and neuronal proliferation
- ✓ **Protects against amyloid-beta** induced neurotoxicity
- ✓ **Improves mitochondrial function**
- ✓ Displays a **sustained antidepressant-like effect**

Data also suggest a disease-modifying effect by increasing plasticity

Parrado Fernandez C et al. Int J Mol Sci. 2023 Jul 6;24(13):11159. https://doi.org/10.3390/ijms241311159
Märta Dahlström et al. Cells 2021, Volume 10, Issue 8, 1871. *Identification of Novel Positive Allosteric Modulators of Neurotrophin Receptors for the Treatment of Cognitive Dysfunction*

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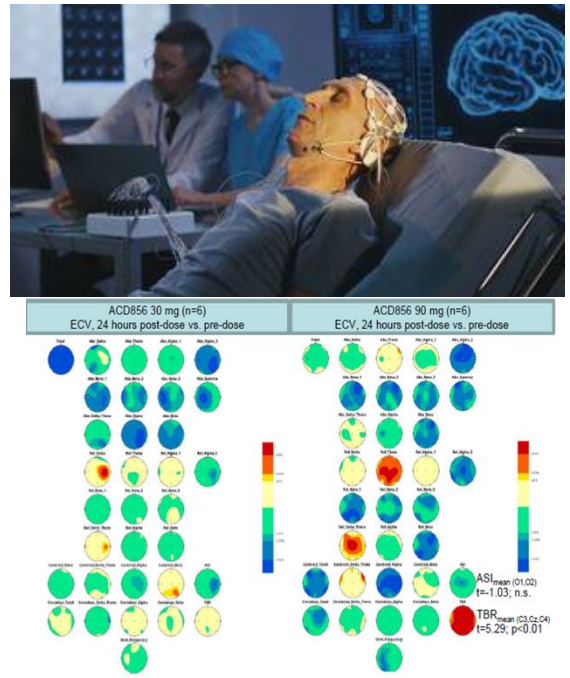
Phase I Demonstrated Safety, Tolerability & Target Engagement

- ACD856 is **safe & well tolerated**
  - Single and repeated dosing with ACD856 did not result in any serious adverse events in healthy volunteers at the tested dose levels
- ACD856 has an **excellent PK profile** suitable for **once daily** oral dosing
  - ACD856 has a rapid absorption, a long half-life of ~ 20 hours, high bioavailability and a linear dose-dependent exposure
- ACD856 **present in CSF** with a ratio of ACD856 in CSF to unbound average ACD856 levels in plasma at **> 37%**
  - **ACD856 pass the BBB** and measured concentrations in CSF confirms a **high exposure** at the level of the brain
- ACD856 MAD qEEG results shows clear **CNS target engagement**
  - EEG pattern supports pro-cognitive as well as antidepressant effects of ACD856



Vigilance control brain maps for 30 and 90 mg cohorts

Now preparing for phase IIa study in Alzheimer's patients

Önnestam, K et al. J Prev Alzheimers Dis (2023): https://link.springer.com/article/10.14283/jpad.2023.89

AlzeCure

Grant financed Phase IIa study to investigate the safety & efficacy of ACD856 in Alzheimer's disease

AlzeCure receives EU grant for Phase 2 clinical trial of NeuroRestore ACD856 for Alzheimer's disease

February 17, 2025

AlzeCure Pharma AB (publ) (FN STO: ALZCUR), a pharmaceutical company that develops candidate drugs for diseases affecting the nervous system, focusing on Alzheimer's disease and pain, today announced that its project NeuroRestore ACD856 has received €2.5m from the EU's European Innovation Council.

This funding will support AlzeCure's planned Phase IIa study of NeuroRestore ACD856 in Alzheimer's patients. In addition, higher doses of ACD856 will be evaluated in humans, as the good safety profile allows for further dosing. ACD856 has the potential to be the first drug in a new category of Alzheimer's drugs – positive allosteric modulators of Trk receptors (Trk-PAMs), which increase BDNF and NGF signaling.

"We are delighted to have been awarded a €2.5m grant from the European Innovation Council (EIC) for our innovative Alzheimer's treatment. The support from the EIC Accelerator program is a recognition of the project and will be crucial in advancing our clinical trials and bringing this much-needed Alzheimer's treatment to patients," said Johan Sandin, Chief Scientific Officer at AlzeCure Pharma

European Innovation Council

https://www.alzecurepharma.se/en/alzecure-receives-eu-grant-for-phase-2-clinical-trial-of-neurorestore-acd856-for-alzheimers-disease/
https://link.springer.com/article/10.1007/s00213-023-06410-x

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Summary

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Key Investment Highlights in AlzeCure

- Targeting areas of **significant unmet medical needs**
- **Strong team** with extensive experience and track record – from idea to clinic
- **Platforms** with **first-in-class properties** and potential **game-changers**
- **Parallel investments** in several candidates and potent **follow-up programs**
- **Multi-billion dollar** market **opportunities**
- Evolved from a discovery into a **phase II company** – **supported by EIC***

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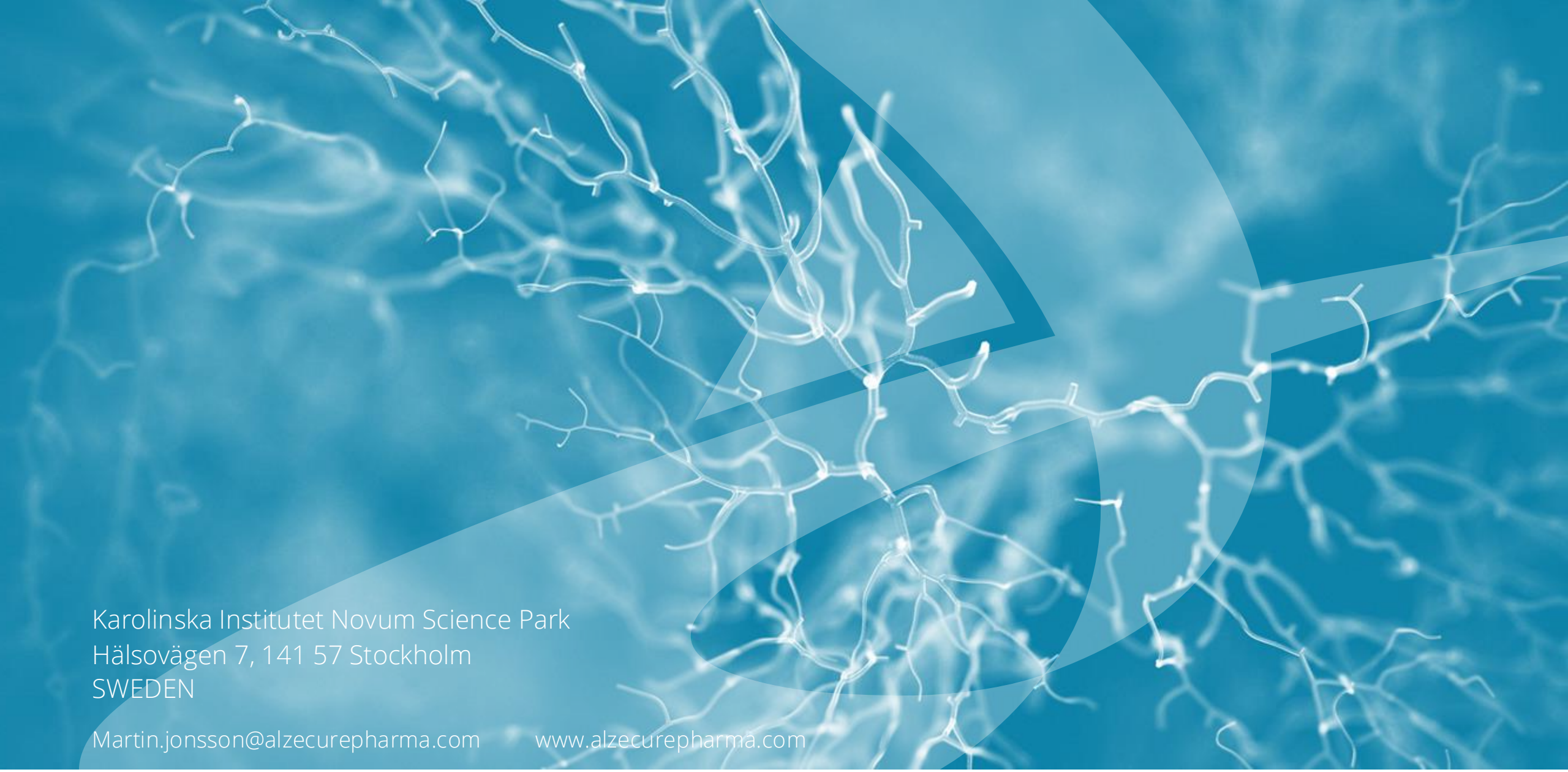

Karolinska Institutet Novum Science Park
Hälsovägen 7, 141 57 Stockholm
SWEDEN

Martin.jonsson@alzecurepharma.com www.alzecurepharma.com

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