

June 5, 2026



Developing therapies for Alzheimer's & pain

European
Innovation
Council



 AlzeCure

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Agenda

- Briefly about AlzeCure
- Projects update & achievements
- Rights issue & purpose





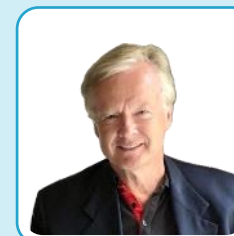
AlzeCure Pharma in brief

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)
- **Cash position: 33.0 MSEK** (Q1 2026) + Fully secured rights issue in June 2026 of **30.1 MSEK** (Gross)
- **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

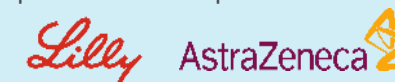


Professor Jan Lundberg

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.



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

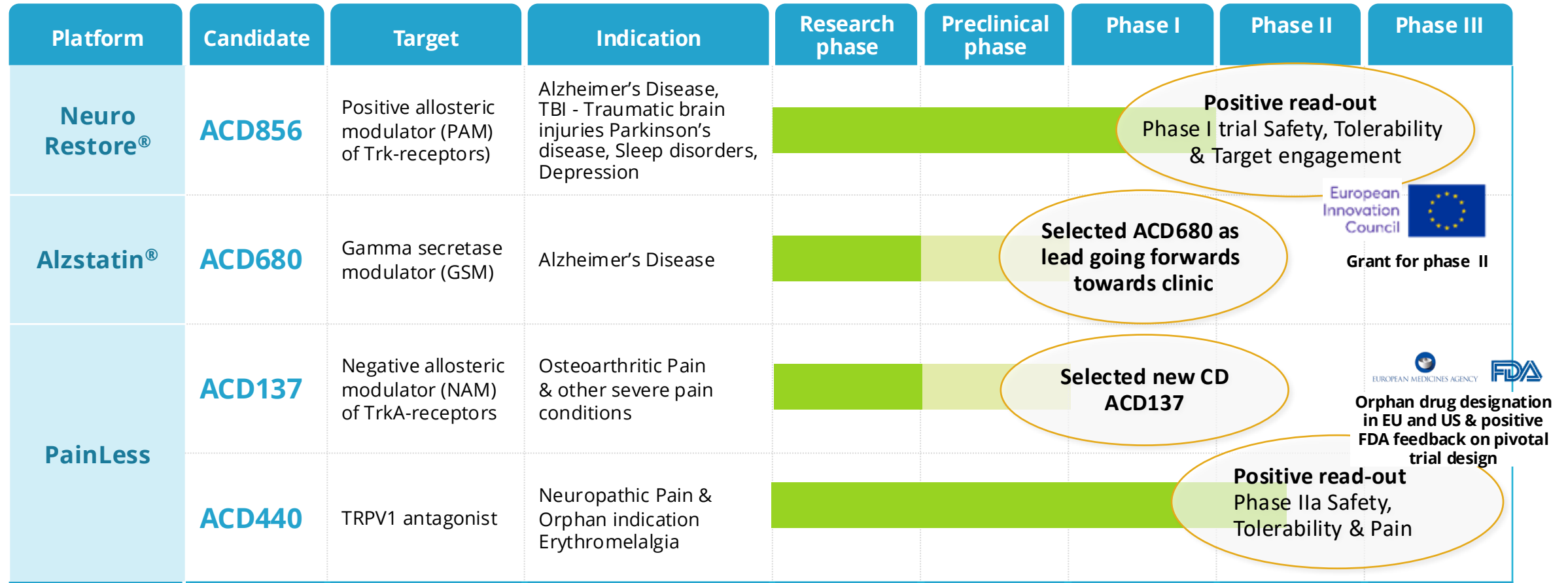


Royalties on
sold products



A pipeline of Small-Molecule Programs

Multiple candidates increase chance of success



 Phase completed

 Phase ongoing

Close cooperation with leading experts & institutions



Professor Bengt Winblad
Karolinska Institute



Professor Maria Eriksdotter
Karolinska Institute



Professor Henrik Zetterberg
Sahlgrenska and UCL



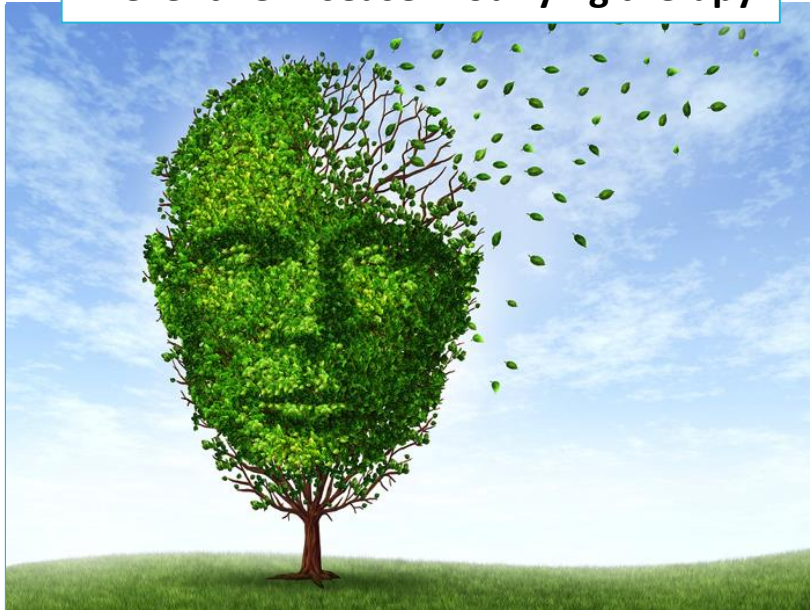
GÖTEBORGS UNIVERSITET
SAHLGRENKA AKADEMIN



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

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

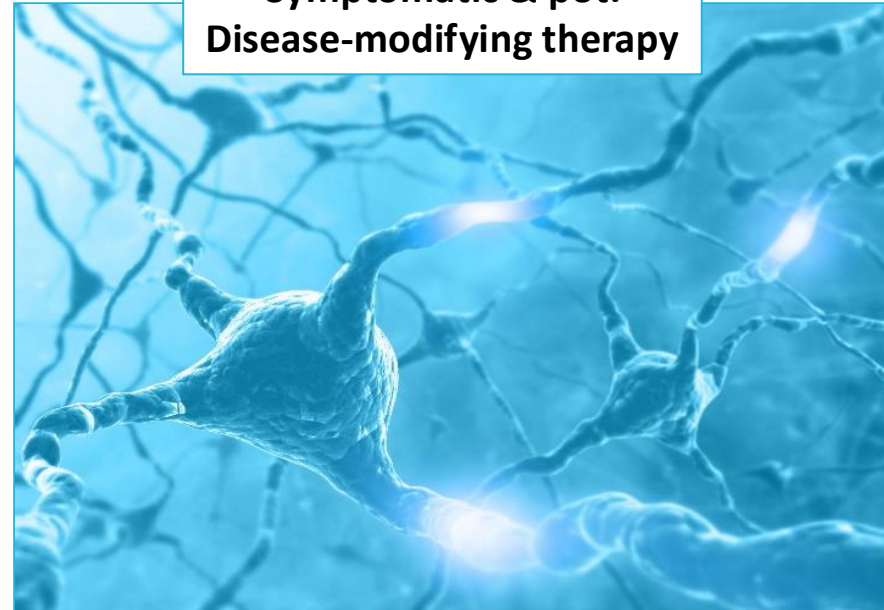
Preventive Disease-modifying therapy



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*

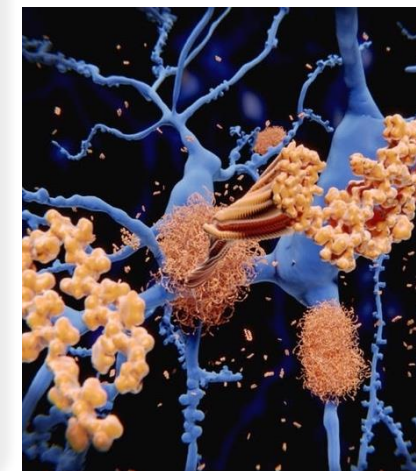


NeuroRestore[®] – *Trk-PAM improving neuronal function & cognition in Alzheimer's disease*



The importance of BDNF & NGF signaling in Alzheimer's

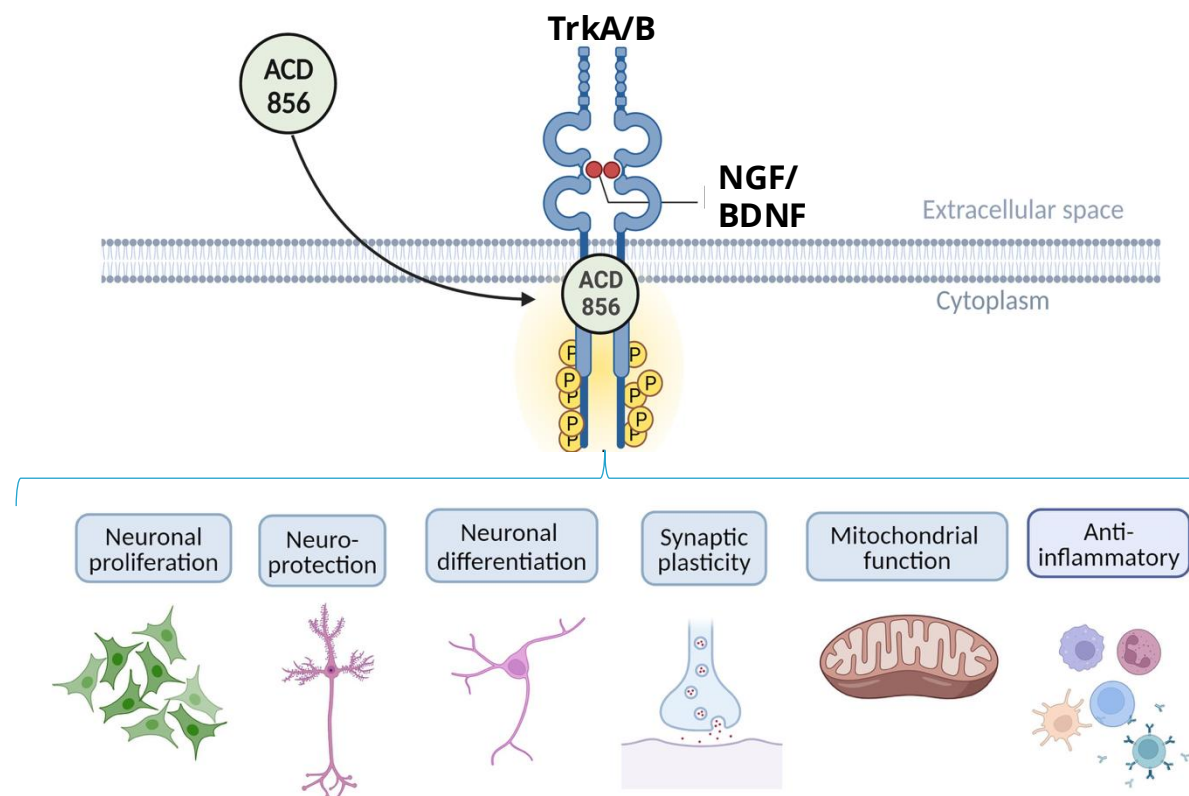
- The neurotrophins Brain-Derived Neurotrophic Factor (BDNF) and Nerve Growth Factor (NGF) bind to their respective receptors, TrkB and TrkA and are **key for brain health and cognition**
- BDNF& NGF signaling are implicated in several key neuronal functions, including **cholinergic function**, hippocampal 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

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**

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

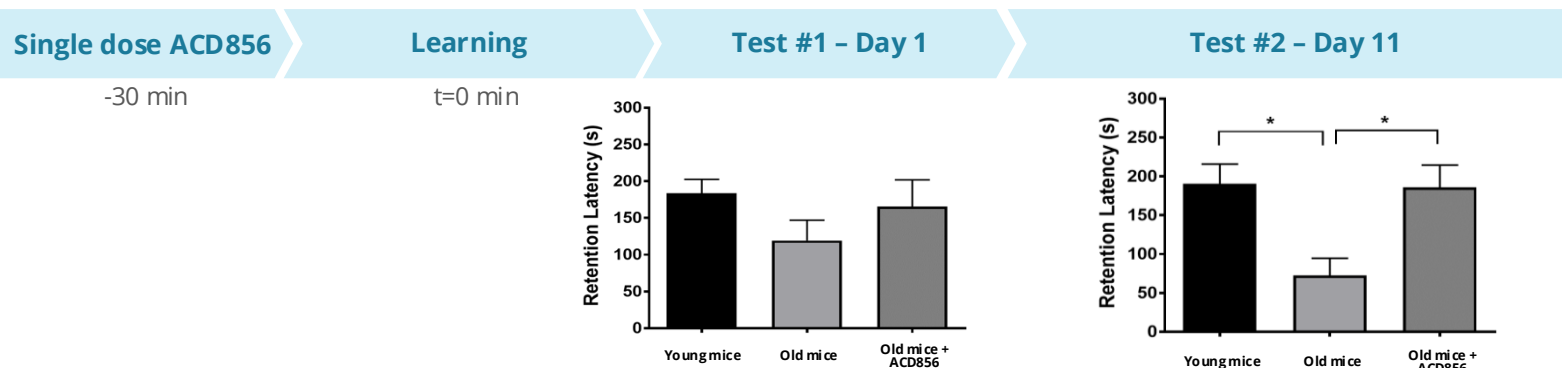
Re-accessing the information from the past which has been encoded & stored

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

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



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

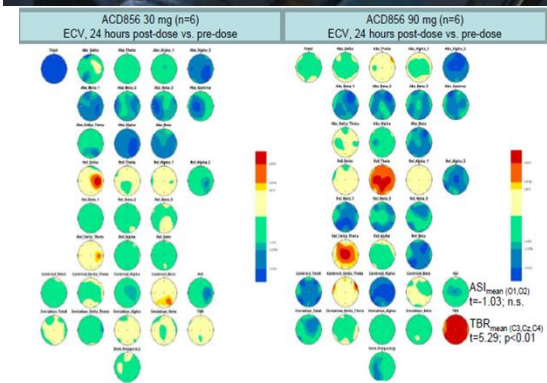
Supporting data

- ✓ 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

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

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

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



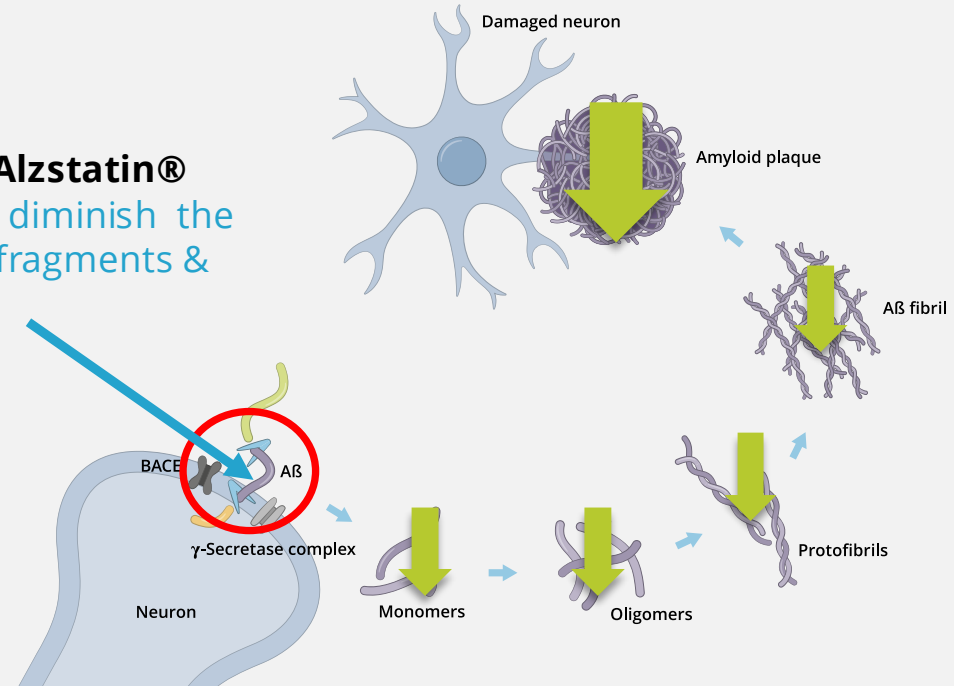
The Alzheimer's Brain and its Destruction by Toxic Protein

The Amyloid Cascade

Toxic protein formations, built up of A β 42, are harming and destroying brain structures

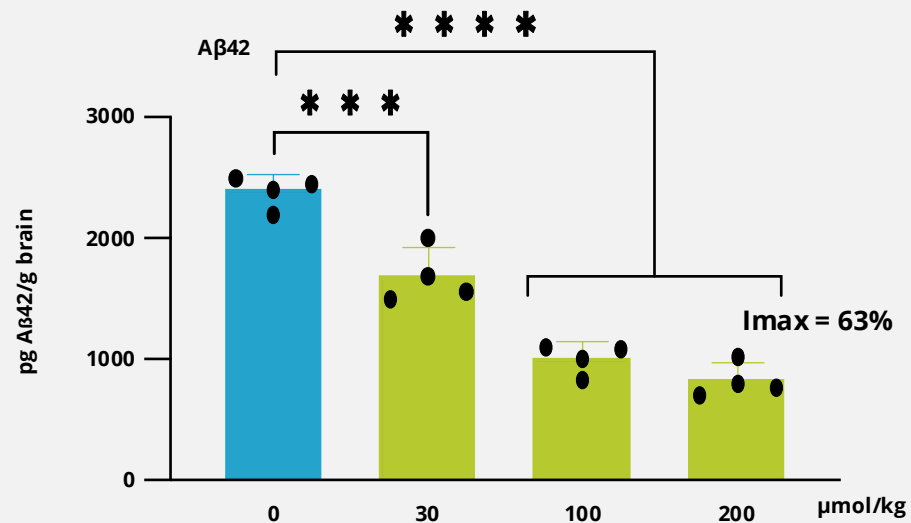


AlzeCure's GSM¹ **Alzstatin®** reduces A β 42 to diminish the build-up of toxic fragments & plaque

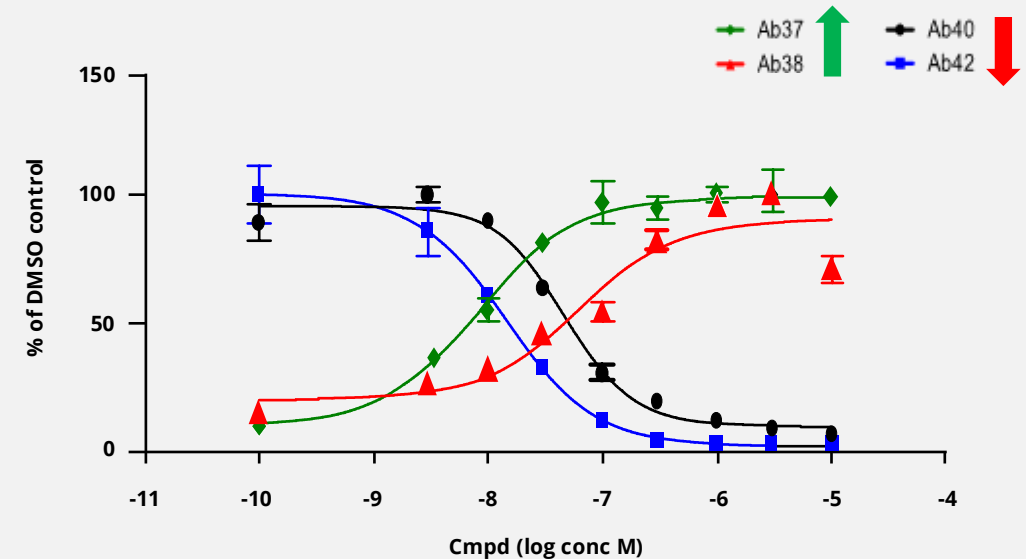


Alzstatin® – Potent Reduction of Toxic A β 42 & Increasing Protective A β 37 & A β 38

Alzstatin® potently reduces the amount of toxic brain A β 42 in animals

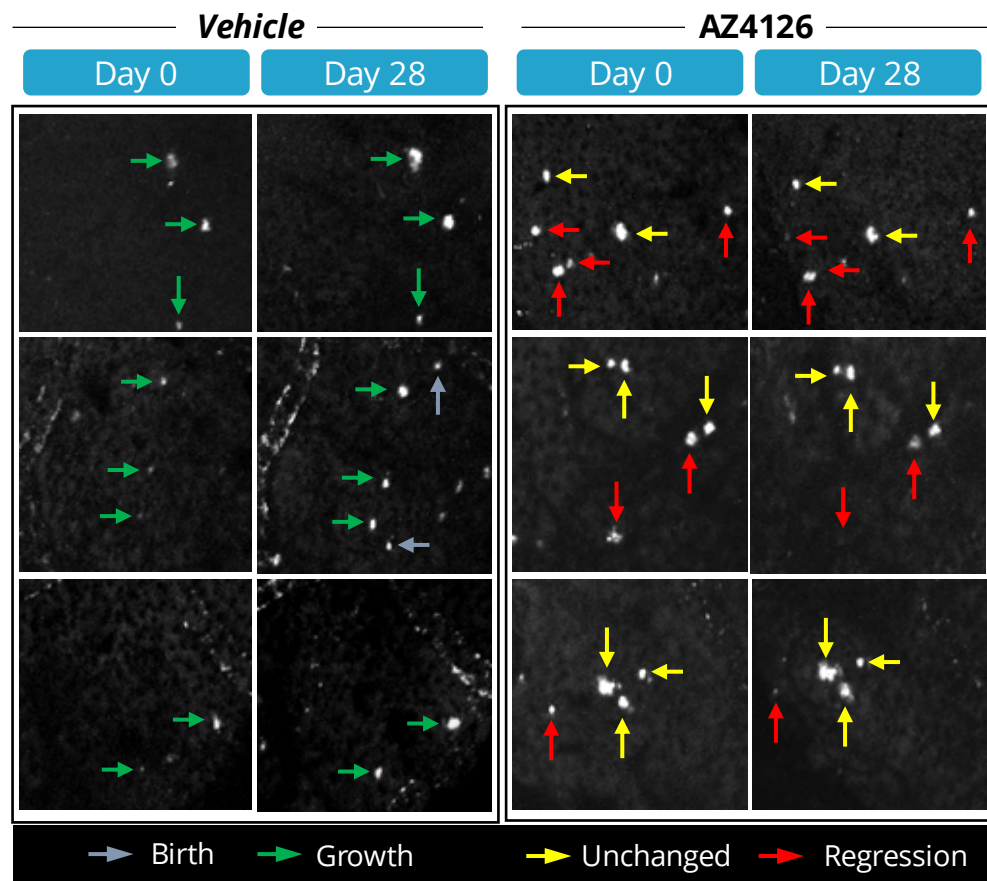


Alzstatin® reduces toxic A β 42 and A β 40, while increasing protective A β 38 and A β 37 peptides

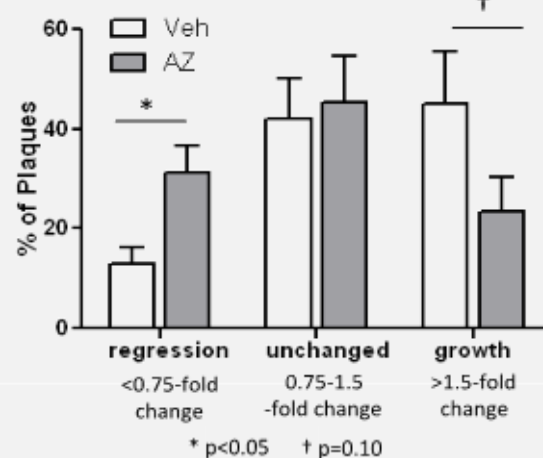


New preclinical data shows Induced Plaque Regression

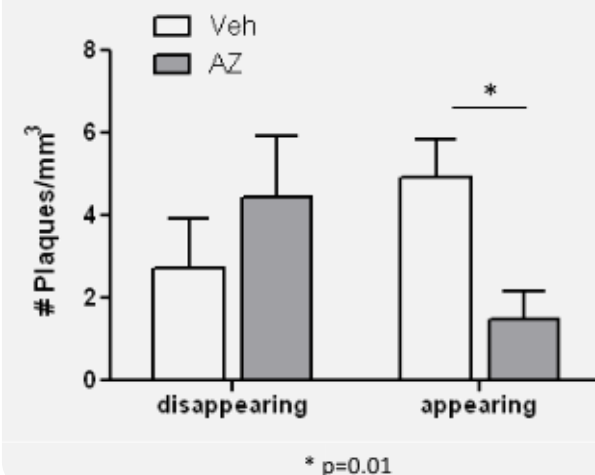
Two-photon-study preclinical study in APP/PS1 mice



Distribution of plaque growth & regression



Plaque appearance or disappearance



28-day GSM treatment

Reduces amyloid plaque growth

Decreases new plaque appearance

Induces plaque regression¹

Multiple Potential Treatment Populations for Alzstatin

– Maintenance, Combination & Preventive Therapy

Maintenance therapy - in patients with established Alzheimer's disease

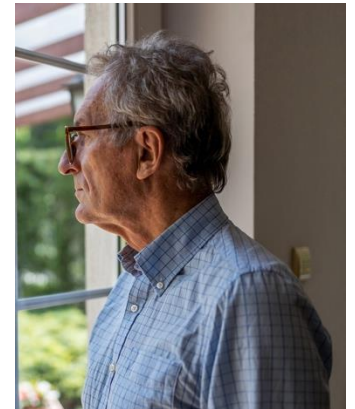
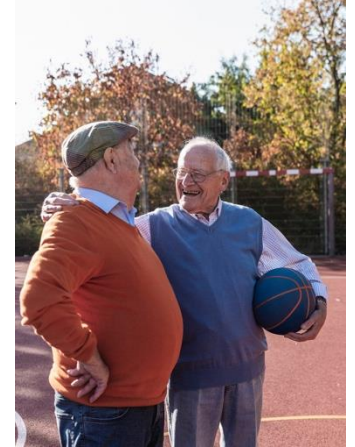
- Treatment after initial plaque clearance by monoclonal antibody treatment (as initially proposed by Lilly) e.g., with: Lecanemab (Eisai/Biogen/Bioarctic) & Donanemab (Lilly)

Combination therapy - together with monoclonal antibody treatment - early AD

- Combine plaque clearance by monoclonal antibody treatment and reduction of A β 42 production by GSM

Preventive therapy - based on genetic risk factors¹ 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. Down's syndrome) suitable for initial proof of concept clinical studies



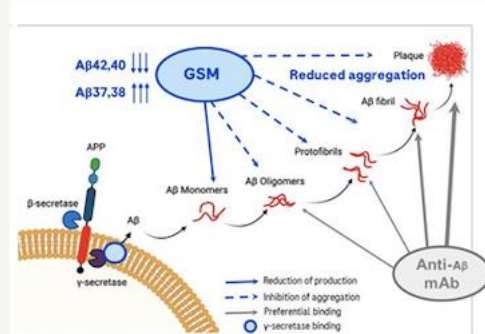
Interim phase II data on Roche GSM expected in 2026

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

Targeting amyloid precursor protein processing to prevent $A\beta$ -aggregation

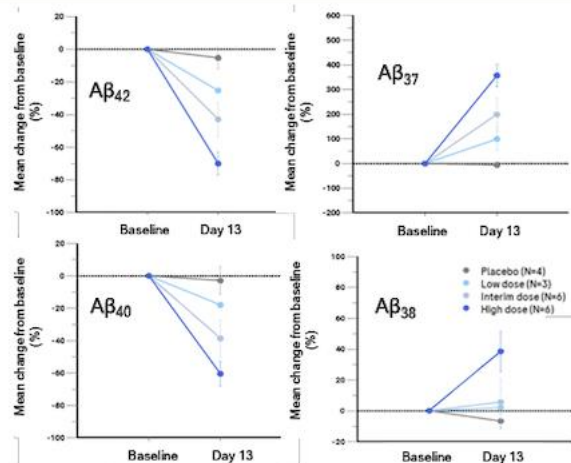


GSMs reduce $A\beta$ aggregation¹



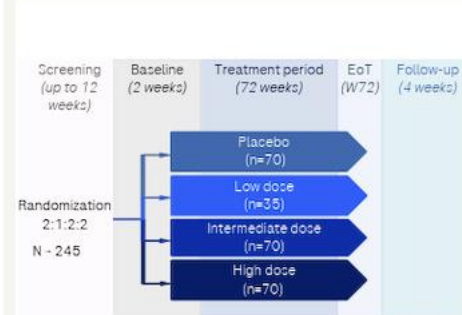
- RG6289 a potentially first-in-class oral GSM with high potency and selectivity
- GSMs alter APP processing: Reduction of $A\beta_{42/40}$ and elevation of $A\beta_{38/37}$
- Selective for APP with no effect on Notch
- GSMs prevent amyloid accumulation and halt plaque formation in animal model

Ph I dose escalation results for RG6289²



- Daily administrations of RG6289 decreased $A\beta_{42/40}$ and increased $A\beta_{37/38}$ concentrations in CSF of healthy volunteers in a dose dependent manner

Ph II (GABriella) study design³



- 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®**



PainLess – targeting unmet medical needs within pain



Introduction to our platforms PAINLESS



Osteoarthritis & severe pain conditions

> 300 million patients

Project: **ACD137**

Negative allosteric modulator of TrkA-receptors in research phase



Neuropathic pain

> 600 million patients

Project: **ACD440**

Topical TRPV1 antagonist in clinical Phase II

ACD137 – Non-Opioid Treatment of Osteoarthritis & Severe Pain

Attractive target population

E.g., **osteoarthritis** in patients who have experienced insufficient pain relief from 1st line standard of care or unacceptable side effects

Clinical validation

Mechanism with **strong validation** – step-changing clinical efficacy with novel anti-NGF mAbs - sets the standard for future therapies

Blockbuster opportunities

Blockbuster opportunities for NGF therapies that would avoid adverse events and allow non parenteral routes of administration



Differentiation factors for TrkA-NAM ACD137¹





- **TrkA ACD137 selective MoA** vs anti-NGF antibodies also effecting p75 signaling
 - Maintain **potent clinical efficacy**
 - **Better safety profile**
- Convenient **oral administration** - small molecule compound
- **No addiction** compared to opioids

1) OA = Osteoarthritis ^) LBP = Lower Back Pain

<https://www.alzecurepharma.se/sv/wp-content/uploads/sites/3/2025/09/alzecure-trka-nam-neupsig-2025-poster.pdf>

27 <https://www.alzecurepharma.se/en/alzecure-presents-new-positive-data-for-the-trka-nam-pain-project-against-osteoarthritis/>

ACD137 positions AlzeCure in next-generation TrkA pain therapies

Compound	Company	TrkA (nM)	TrkB (nM)	TrkA vs TrkB	Status	Indication	
ACD137	AlzeCure	0.87	26 400	30,300	Preclin	OA¹ & other pain conditions	
Cmpd #10 AK1830	Array Bio. Ashai Kasei	0.038	210	5,500	Phase IIb	OA LBP [^]	
Cmpd #23	Pfizer	0.64	145	230	inactive	OA	
Cmpd #1	Merck	99	>81000	>820	inactive	OA	

AlzeCure has synthesized novel & highly potent & selective TrkA-NAM's ... and is a follower to Asahi Kasei's asset which has entered phase IIb with expected read-out in 2026

ACD440 – Novel TRPV1 antagonist in clinical phase for neuropathic pain

Project overview



Emanates from Big Pharma

- Approximately **20M USD** already **invested** in project development
- **Mode of action confirmed** in several Phase 1 clinical trials
- Synthesized compound and formulation developed

TRPV1 – Optimized for local delivery

- The vanilloid receptor subtype 1 (TRPV1) is expressed in nociceptive sensory neurons
- TRPV1 is upregulated in the skin of patients with neuropathic pain
- **Strong scientific support** for peripheral/local treatment with TRPV1 antagonists of **both neuropathic & nociceptive pain**

Positive clinical trial results in phase Ib & IIa

- Developed **topical gel formulation to avoid AEs** associated with systemic TRPV1 blockade
- **Clinical trial** with topical formulation was initiated and **successfully finalized**
- Phase Ib study addressed **safety, tolerability & efficacy – POSITIVE OUTCOMES**

Presented detailed positive phase IIa data

Positive phase IIa results for First-in-Class ACD440

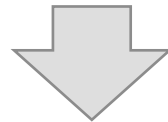
- Demonstrate **Positive Proof-of-Mechanism (PoM)** results **in patients** with chronic **peripheral neuropathic pain** in a placebo cross-over trial
- The patients have had **chronic pain** for many years & were on **concomitant medication**
 - A **significant analgesic effect** on pain induced by cold and heat.
 - The data are **in line with previously** reported phase Ib **results**
 - **Pain reduced** by appr. **50%** - a **clinically significant magnitude**
- The **bedside test identified eligible patients**
- **Well tolerated** as a topical gel on the skin, which shows **suitability for continued clinical development**

Now preparing for next phase II and phase III trial

Strong regulatory momentum in US and EU

Granted orphan drug designation (US & EU)

- **FDA granted Orphan Drug Designation (ODD)** for erythromelalgia
- European Medicines Agency (EMA) **granted orphan designation** in the **EU**
- **Positive FDA feedback** supporting **Phase II/III pivotal** trial pathway



Reduced regulatory risk across key markets

Erythromelalgia

- Erythromelalgia is a **rare chronic** painful disorder – “Burning Feet Syndrome” existing in **children & adults**
- Triad of redness, swelling and intense pain
- Pain comes in attacks, flares, **triggered by heat** or exercise
- One **pain flare can last** for an hour and **up to days**

Flare on Feet



BEFORE

DURING

Flare on Face



BEFORE

DURING

Flare on hands



BEFORE

DURING

- Divided into Primary erythromelalgia, is mostly hereditary
- **No efficacious medical treatment exists**

Ongoing Rights Issue June 2026

Rights Issue June 2026 – Summary:



- Taking in appr. **SEK 30,1M**
- **Fully secured**, to 100%, by our major shareholders, free of charge
- Subscription price of **SEK 1.31 per share**, whereby five (5) existing shares entitle the holder to subscribe for one (1) new share
- Purpose for the issue is to **strengthen the company's cash position** and provide additional resources for **business development**, supporting ongoing and new out licensing and partnership dialogues.

Last day of trading including the right to receive subscription right	10 June 2026
First day of trading excluding the right to receive subscription rights	11 June 2026
Record date for participation in the Rights Issue	12 June 2026
Trading in subscription rights	16 June – 25 June 2026
Subscription period	16 June – 30 June 2026
Announcement of the Rights Issues outcome	Around 2 July 2026

For complete information visit our home page: www.alzecurepharma.com



Summary



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***



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

Martin.jonsson@alzecurepharma.com

www.alzecurepharma.com

