Nordic Asia Life-science Summit
May 28, 2025

Developing therapies for Alzheimer's & Pain

Martin Jönsson, CEO

European Innovation Council

AlzeCure

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AlzeCure Pharma – in brief

- Working in **Alzheimer's Disease** (AD) and **Pain** – Hugh 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**
  - **Alzstatin®** – An innovative preventive & disease-modifying treatment against Alzheimer's (AD)
  - **NeuroRestore®** – A novel symptomatic treatment for cognitive disorders, e.g. AD with disease modifying potential
  - **Painless** – Innovative projects for osteoarthritic & neuropathic pain

- Listed on **Nasdaq First North Premier** Growth Market, **Sweden**, since Nov. 2018 (Ticker: ALZCUR)
- Market cap: **SEK 246m** (May 27, 2025)
- Cash position: **SEK 20.9m** (Q1 2025 interim report)

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Our Business Model

- We 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

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A pipeline of small-molecule programs

– Multiple candidates increase chance of success

Platform	Candidate	Target	Indication	Research phase	Preclinical phase	Phase I	Phase II	Phase III
NeuroRestore®	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	ACD440	TRPV1 antagonist	Neuropathic Pain					
	ACD137	Negative allosteric modulator (NAM) of TrkA-receptors	Osteoarthritic Pain & other severe pain conditions					

Positive read-out Phase I trial
Safety, Tolerability & Target engagement

European Innovation Council
Grant for phase 2

Selected ACD680 as lead going forwards towards clinic

Positive read-out Phase IIa
Safety, Tolerability & Pain

Selected new CD
ACD137

Phase completed

Phase ongoing

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Close cooperation with leading experts & institutions

Professor Bengt Winblad
Karolinska Institute

Professor Maria Eriksdotter
Karolinska Institute

Professor Henrik Zetterberg
Sahlgrenska and UCL

Karolinska Institutet

GÖTEBORGS UNIVERSITET
SAHLGRENSKA AKADEMIN

UCL

AlzeCure

Professor Jan Lundberg – New Board Member

Former global head of R&D at Eli Lilly & global head of research at AstraZeneca joins AlzeCure's board of directors

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.

Professor Jan Lundberg

https://www.alzecurepharma.se/en/eli-lillys-and-astrazenecas-former-head-of-research-dr-jan-lundberg-invests-in-alzecure-and-is-proposed-for-election-to-its-board/

Board of Directors

- Complementing expertise for the continuous development of AlzeCure

BOARD OF DIRECTORS		EXPERIENCES
	**Thomas Pollare** Chairman of the Board Ph.D., M.D.	› Strong background in both Life science industry and the Private equity market › Education: M.D. Karolinska Institutet, Ph.D. Uppsala University
	**Jan Lundberg** Director of the Board Ph.D.	› Extensive experience from leading positions in global pharmaceutical companies, including as global head of research at AstraZeneca & global head of research and development at Eli Lilly › Education: Ph.D. of pharmacology at the Karolinska Institute
	**Eva Lilienberg** Director of the Board M.Sc.	› Extensive experience from Big Pharma and consulting within Regulatory & Market Access › Education: MSc in Pharmaceutical sciences at Uppsala University
	**Ragnar Linder** Director of the Board M.Sc.	› Strong industrial marketing and strategic experience, also in the CNS/Dementia area › Education: Master of Chem Ing., Royal Institute of Technology, Stockholm
	**Dr Janet Hoogstraate** Director of the Board Ph.D.	› Extensive experience from in life science, in preclinical research, the pain area and biotech › Education: PhD in Biopharmaceutical Sciences from the University of Leiden in the Netherlands and an eMBA from Hult International Business School.

The AlzeCure Management team

- Extensive experience from the industrial drug development value chain

MANAGEMENT TEAM		EXPERIENCES
 **Martin Jönsson** CEO	› Extensive experience in various senior management positions with >20 years of international experience in the industry › Education: MBA from Lund University in Sweden, Ottawa, Canada and Freiburg, Germany	Lund University, Roche, Ferring Pharmaceuticals
 **Johan Sandin** CSO, Ph.D.	› Expert in in vivo Pharmacology with over 20 years of experience from the drug discovery industry within Neurology › Education: Ph.D. in Neuropharmacology, Karolinska Institute, Sweden	AstraZeneca, Karolinska Institutet
 **Märta Segerdahl Storck** CMO M.D., Ph.D.	› Broad and extensive experience in global development and clinical operations from Pharma industry within CNS and Pain › Education: M.D., Karolinska Institute, Stockholm, Sweden	Lundbeck, Karolinska Institutet, Grünenthal, AstraZeneca
 **Pontus Forsell** Head of Discovery, Ph.D.	› Expert in drug screening with +20 years of experience from industrial research and drug development within CNS and Pain › Education: Ph.D. in Medical Biochemistry & Biophysics from Karolinska Institute, Stockholm, Sweden	AstraZeneca, Merck Frosst, Orexo, Karolinska Institutet
 **Birgitta Lundvik** CFO	› More than 25 years of experience from industry with a broad experience of venture capital companies › Education: MSc in business from Uppsala University and an eMBA in finance from Stockholm Business school, Sweden	Favro, Lobsor Pharmaceuticals

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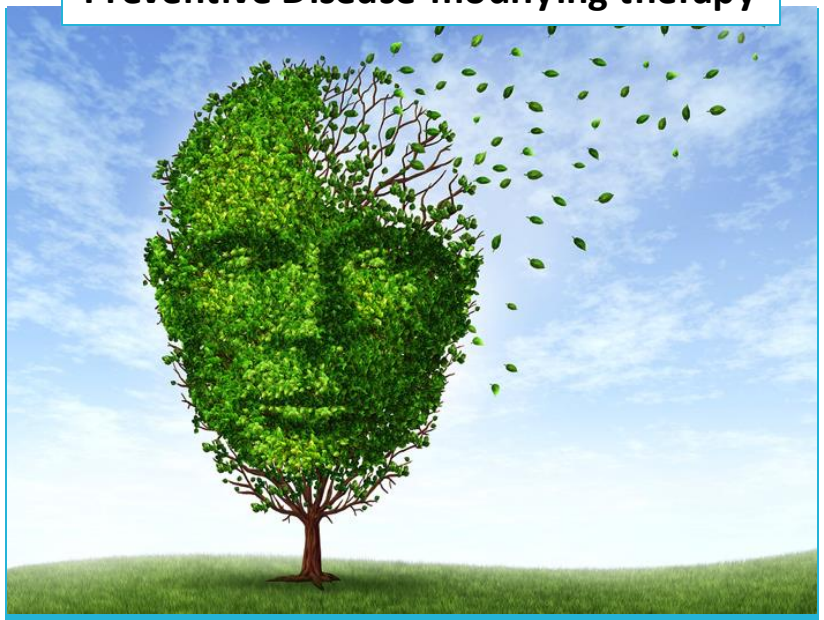
Two Alzheimer's platforms - 1st-in-class properties & potential game-changers

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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Preventing or delaying Alzheimer's

Alzstatin®

MoA: Gamma-Secretes-Modulator

Targeting Toxic Amyloid Production

- *Novel Oral Small Molecule*

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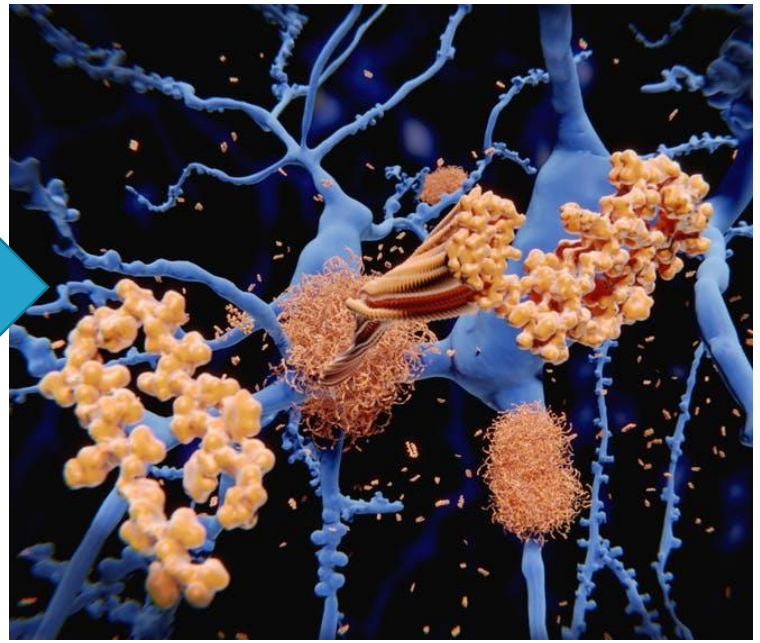
The Alzheimer's brain and its destruction

Toxic protein formations – **Aβ42** amyloid pathology & plaque – are harming and destroying the brain. The formation process is called the **Amyloid Cascade**



Toxic protein formations, built up of Aβ-42, are harming and destroying the brain structures.

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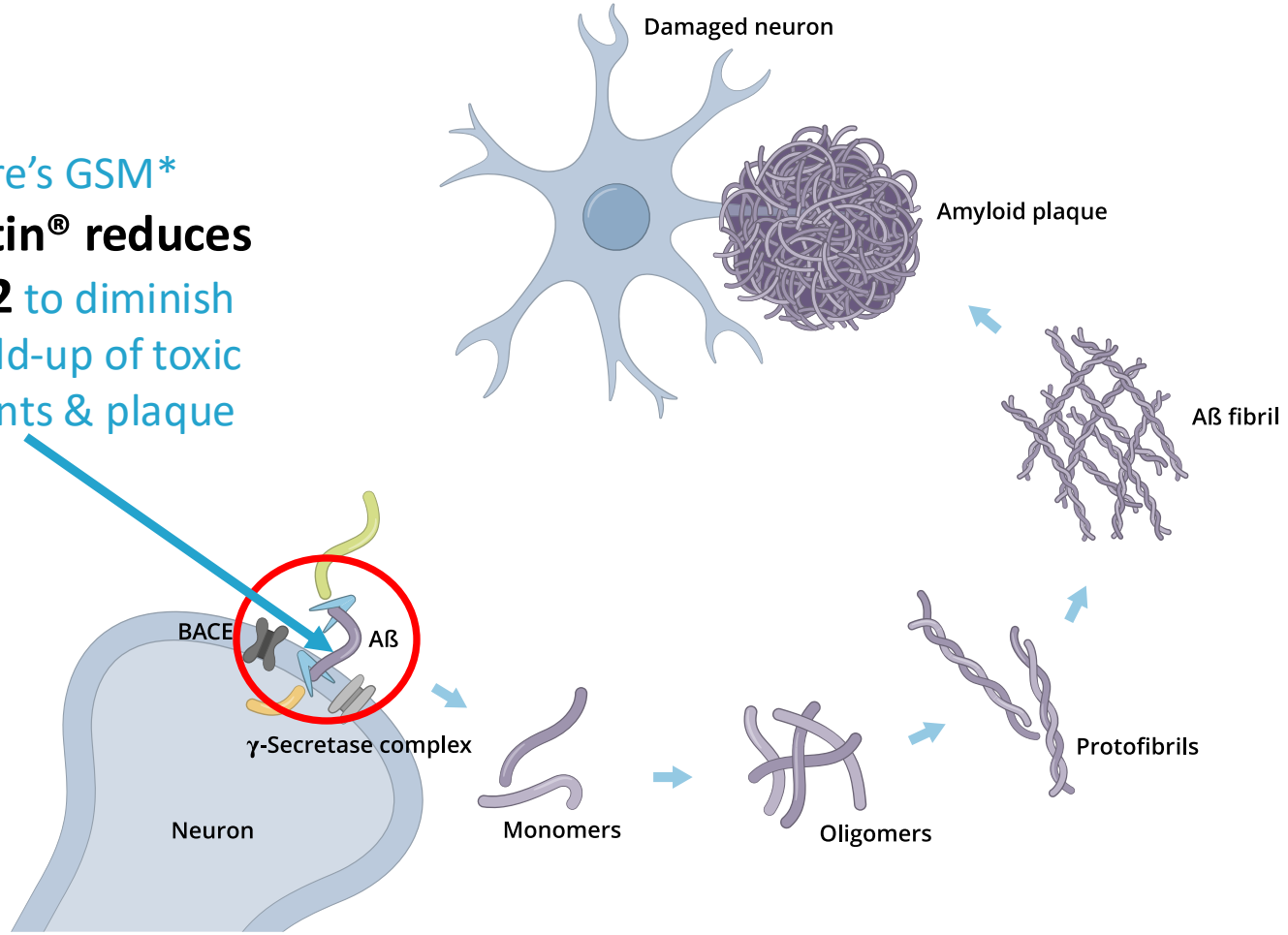
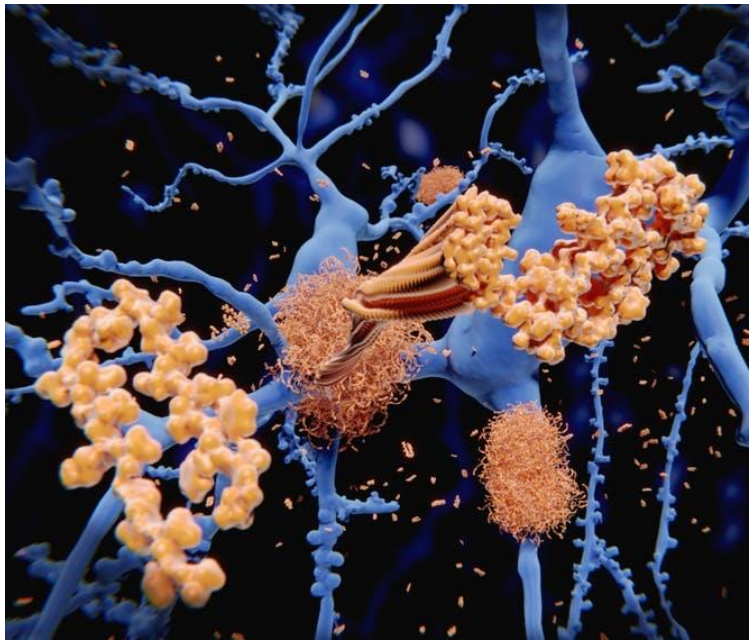
The Amyloid Cascade -

Generates toxic & damaging fragments, including plaques, destroying neurons & brain structures

AlzeCure's GSM*
Alzstatin® reduces Aβ -42 to diminish the build-up of toxic fragments & plaque

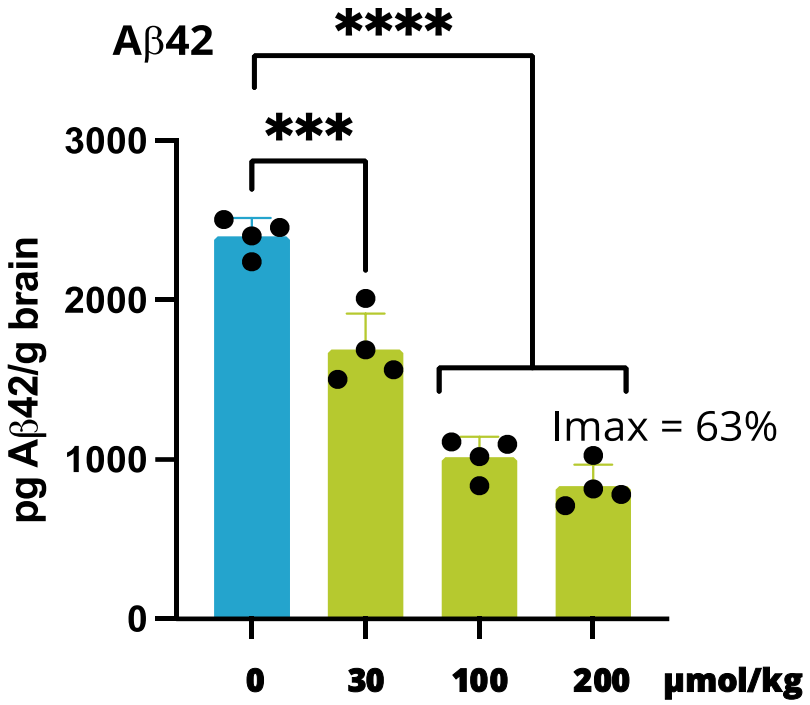




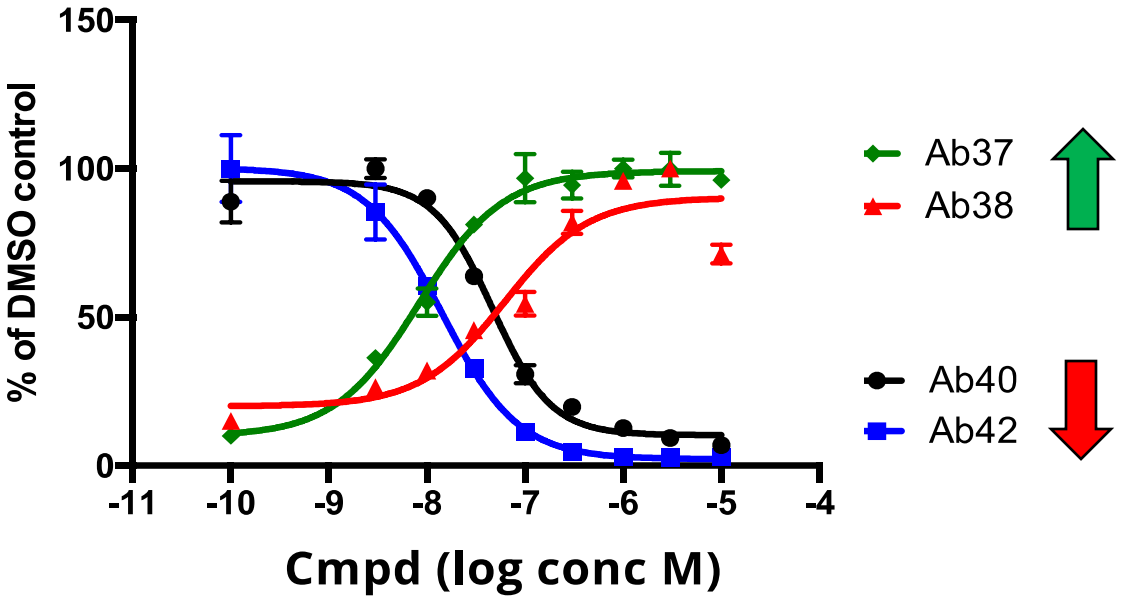
Toxic protein formations, built up of Aβ-42, are harming and destroying the brain structures.

*) GSM = Gamma Secretes Modulator

Alzstatin – potent reduction of toxic Aβ42 and increasing protective Aβ37 & 38



Alzstatin potently reduces the amount of toxic brain Aβ42 in mice



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

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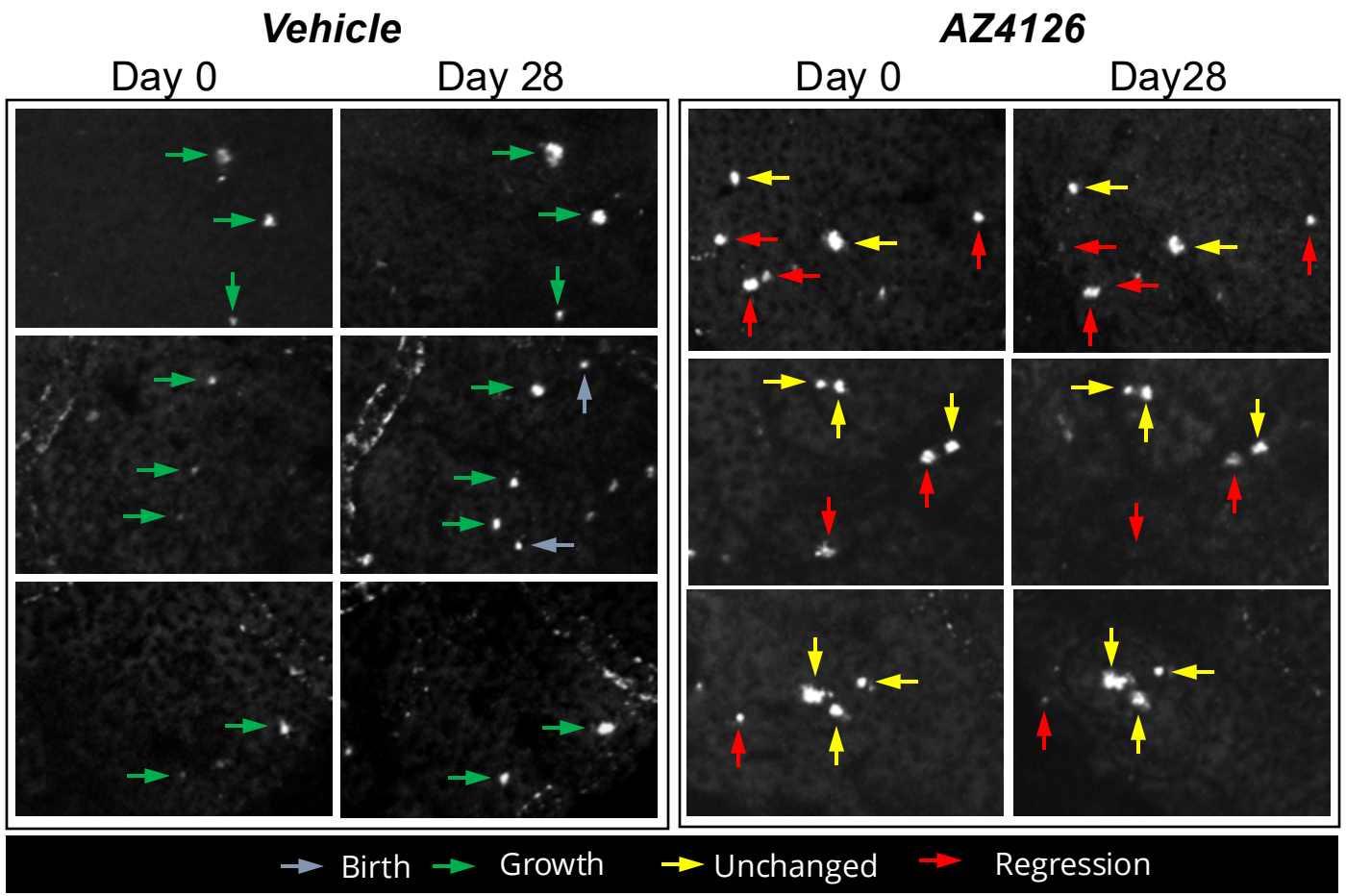
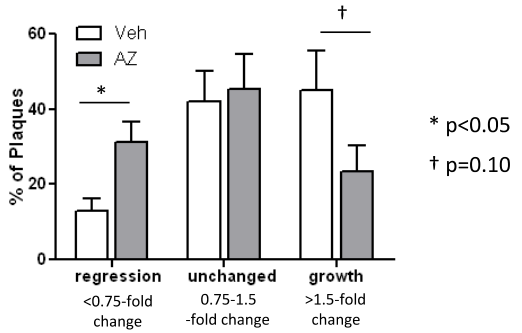
GSM induces plaque regression – New data

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



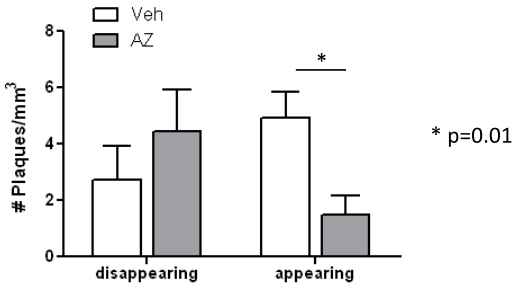
Distribution of Plaque Growth and Regression



* p<0.05

† p=0.10

Plaque appearance or disappearance



* p=0.01

28-day GSM treatment:

- **Reduces** amyloid **plaque growth**
- **Decreases new plaque** appearance
- **Induces plaque regression***

**Plaque regression not reported for Gamma- or Beta-secretase inhibitors suggest that increase in Aβ37 could be responsible*

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/S0022-3565(25)39613-8/fulltext

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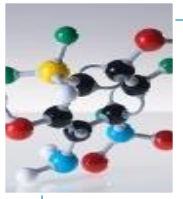
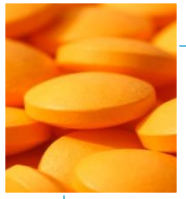
ACD680 differentiation from the Aβ antibodies*

- Key advantages of GSMs



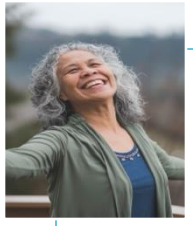
Small molecule therapy

- High CNS exposure
- More cost-effective



Oral formulation

- Convenient for patients
- Lower cost



Fewer side-effects

- Not expected to give rise to brain oedema and brain microbleeds (ARIA)
- No regular brain scans needed

Suitable for early treatment

- GSMs optimal for early treatment

Suitable for large population

- Aβ antibodies only amenable for 5-8% of population**
- Potential as a standalone or combination treatment

*) E.g. Leqembi® (lecanumab), Kinsunla® (donanemab)

**) 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

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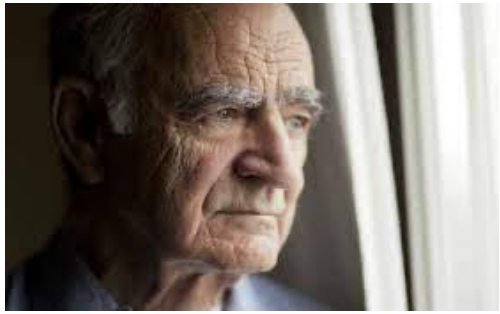
Multiple Target Populations - maintenance & preventive therapy

Maintenance therapy - in patients with established Alzheimer's

- 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* 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

*) E.g. ApoE4

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Attractive Profile
- Early Clinical Proof-of-Mechanism in Phase I/II

- Phase I will demonstrate PoM **and** central target engagement
  - **SAD/MAD studies conducted in healthy volunteers + AD patients**
  - Evaluation of safety and tolerability after single and repeated administration
  - Possibility to explore biomarker effects **showing central target engagement** already in Phase I
    - Aβ42/40 – show **reduction of toxic Aβ-species**
    - Aβ37/38 – show **increase of shorter protective Aβ-species**, establishing gamma-secretase involvement and MoA
    - Measurements done both in CSF and plasma – **biomarker kits available**

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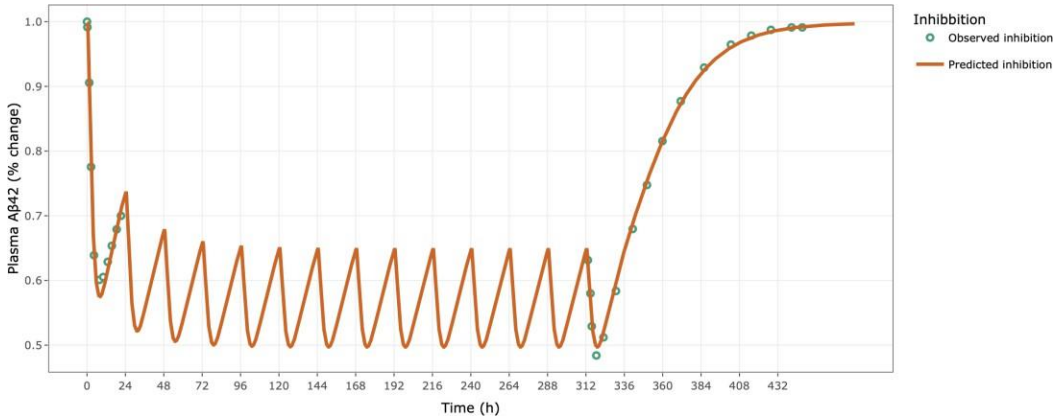
ACD680 more potent in vivo than Roche's clinical compound

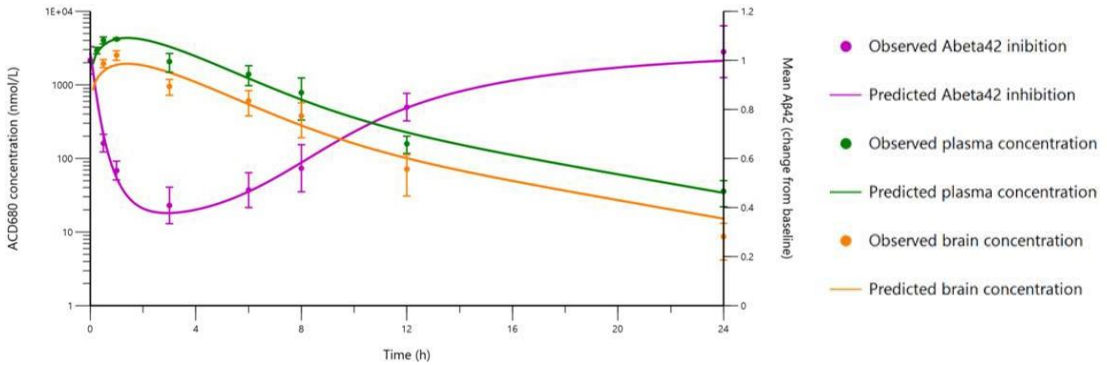
Human PK and Efficacy Phase I Data for RG6289 **Stripped From Published Posters**



Free plasma *In vivo* IC50 = **7.5-15 nM**
In vitro IC50 10 nM*

Higher IC50 values indicate that RG6289 ***requires higher concentrations to achieve reduction***

PK and Efficacy Studies in Mouse Performed for **ACD680 In-House**



Free plasma *In vivo* IC50 = **4.3 nM**
In vitro IC50 = 8.1 nM (In vitro free IC50 = 3.2 nM)

- **Improved Structure** (reduced aromatic content)
- **Increased Potency** (may require lower doses)
- **Improved IP** (substantial patent time)

Roche RG6289 Clinical Compound

AlzeCure ACD680 Lead Compound

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Roche Interim Ph 2 Data Expected in 2026

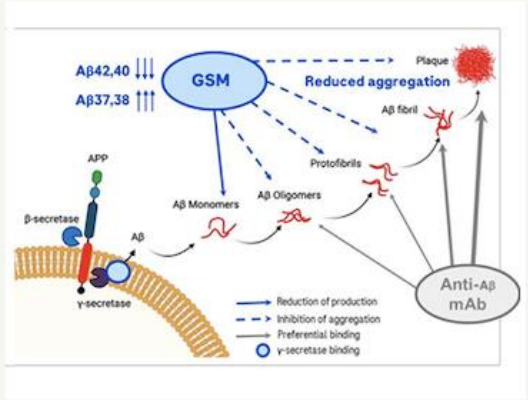
Neurology

Roche

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

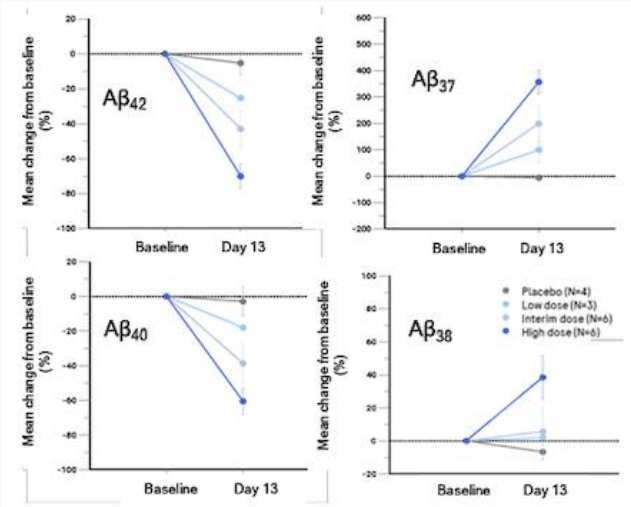
Targeting amyloid precursor protein processing to prevent Aβ-aggregation

GSMs reduce Aβ aggregation[1]



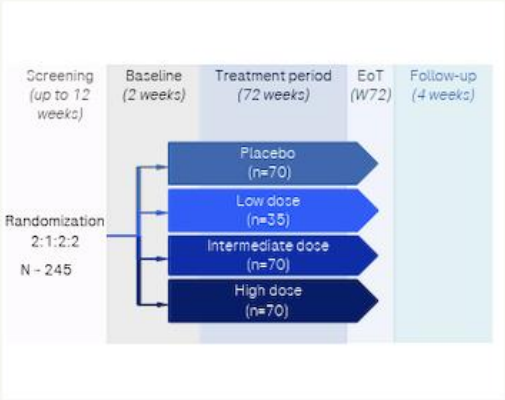
- RG6289 a potentially first-in-class oral GSM with high potency and selectivity
- GSMs alter APP processing: Reduction of Aβ42/40 and elevation of Aβ38/37
- Selective for APP with no effect on Notch
- GSMs prevent amyloid accumulation and halt plaque formation in animal model

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

[1]Figure adapted from Vogt et al., Int. J. Mol. Sci. 2023; [2]Sturm et al; presented at CTAD 2023; [3]Tortelli et al. presented at ADPD 2024; FIC=first-in-class; GSM=gamma-secretase modulator; AD=Alzheimer's disease APP=amyloid precursor protein; Aβ=amyloid β; mAb=monoclonal antibody; HV=healthy volunteers

127

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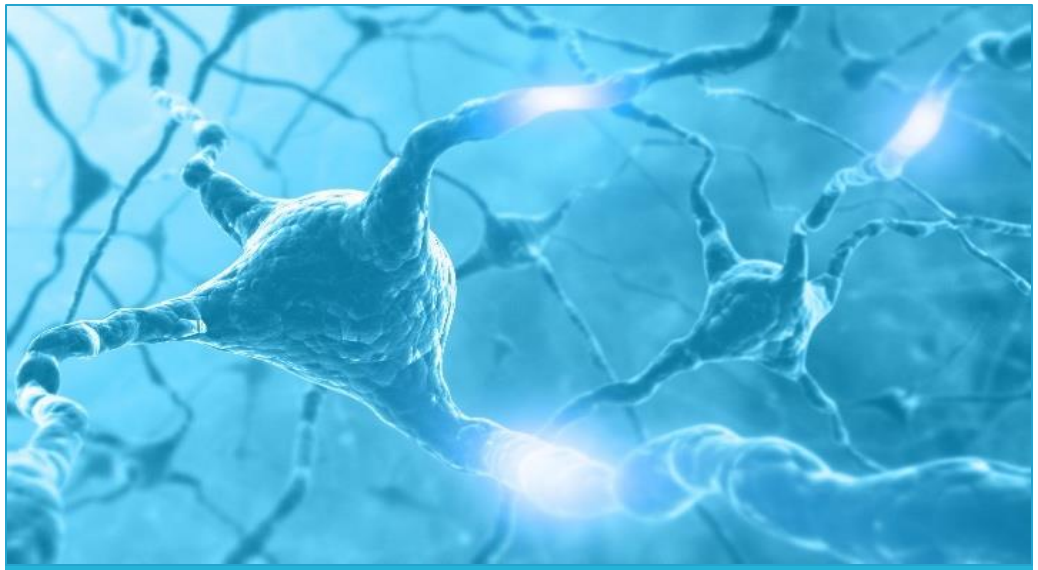
IP position – ACD680

- Recently submitted composition of matter patent application for ACD680
- Patent life expected until **2045**
- Additional 5-year market exclusivity in the USA → **2050**

Improving Learning & Memory Capabilities



NeuroRestore®

Trk-PAM

Improving Neuronal Function & Cognition

- *Novel Oral Small Molecule*

Shown safety, tolerability & target engagement in clinical trial phase 1

=> In preparation for ph 2

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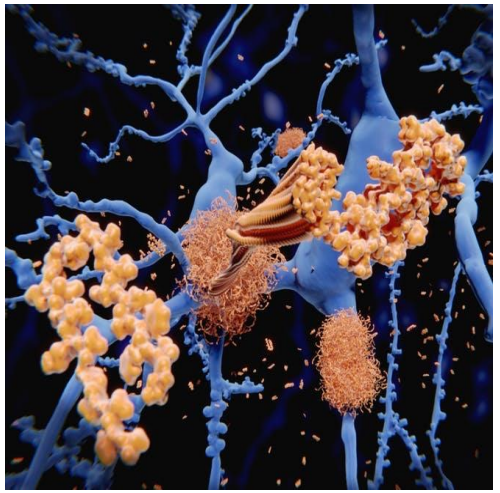
The Relevance of BDNF & NGF Signaling in Alzheimer's

- The neurotrophins Brain-Derived Neurotrophic Factor (BDNF) and Nerve Growth Factor (NGF) are **key for brain health and cognition**
- BDNF& NGF bind to their respective receptors, TrkB and TrkA
- 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, like the 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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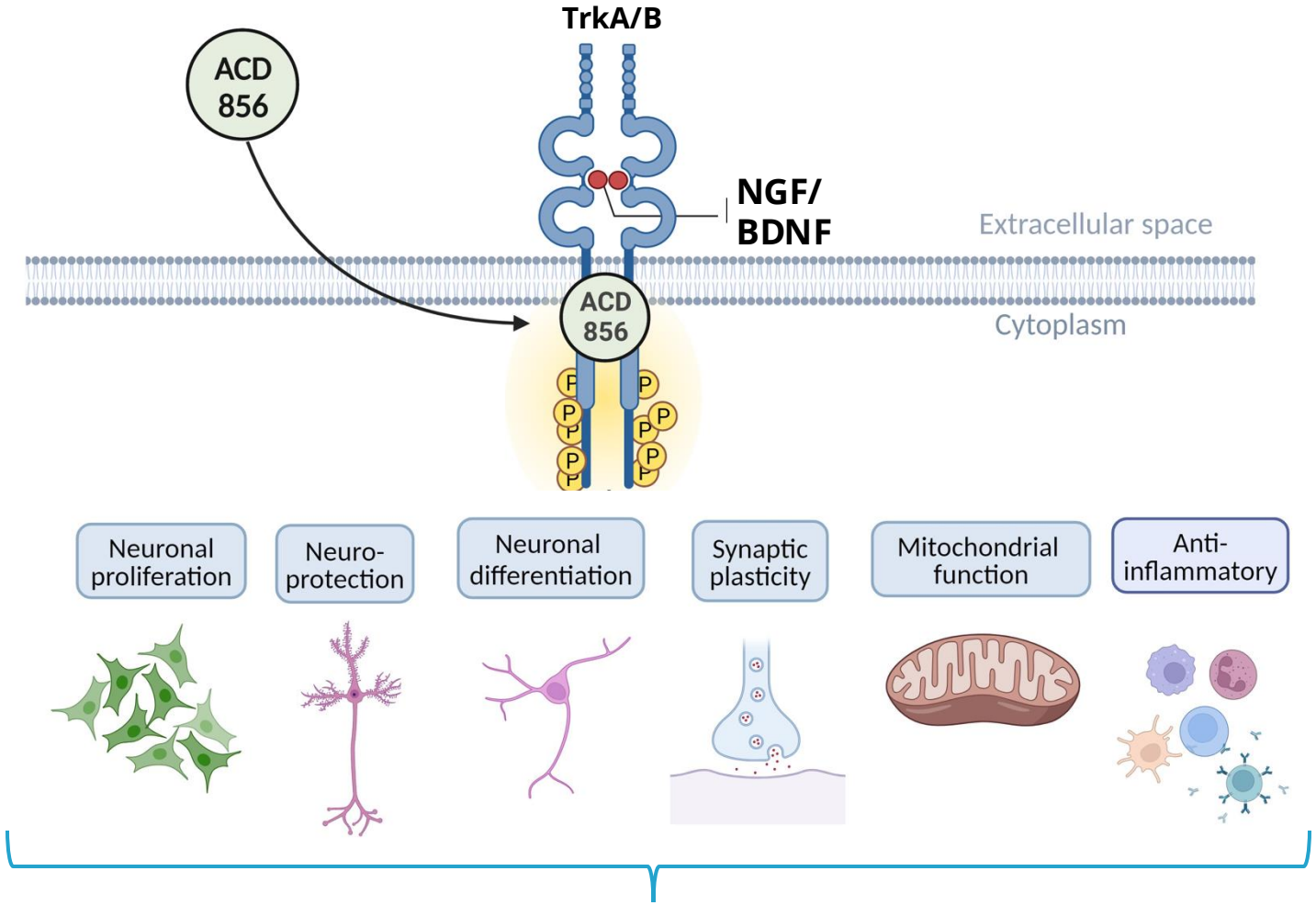
ACD856 - MoA & data supports Disease Modifying Potential

- ACD856 is a novel 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, Memory & Depression

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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NeuroRestore - Cognitive Enhancer Improving Learning and 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 and stored

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

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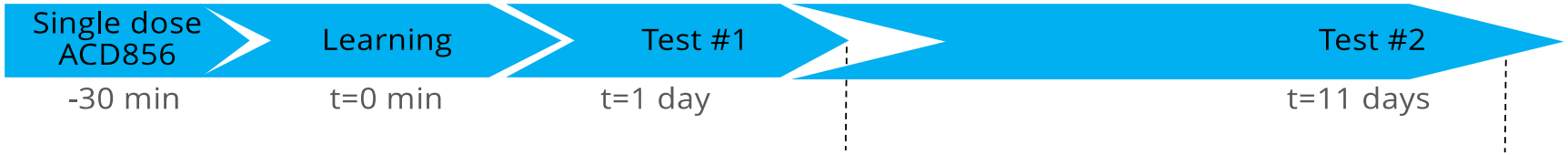
ACD856 – Evidence of Improvements to Learning & Memory

Stages of memory formation

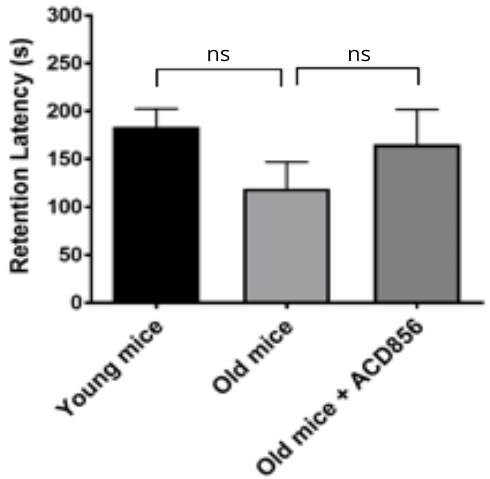
LEARN → STORE → REMEMBER

ACD856 has shown in preclinical models the capacity to improve the ability to learn & remember information

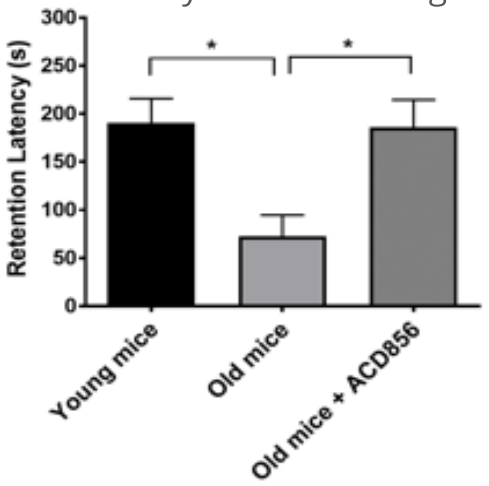


ACD856 **improves age-induced memory impairment** in 18-month-old animals to the level of young animals

Memory test #1,
1 day after learning



Memory test #2,
11 days after learning



Dahlström M et al. Cells. 2021 Jul 23;10(8):1871.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8391421/pdf/cells-10-01871.pdf

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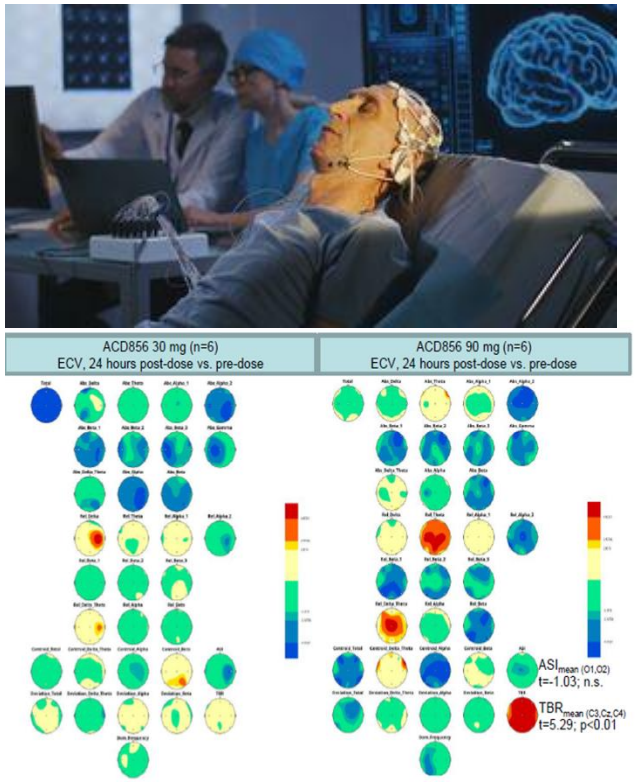
Phase I Study Summary

- 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

=> Now preparing for ph 2



Vigilance control brain maps for 30 and 90 mg cohorts

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

Awarded European Innovation Council Grant

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/

AlzeCure

NeuroRestore ACD856 – Candidate in Clinical Phase

- Patent in the US, China, Japan and Europe to **2039**

AlzeCure receives US patent for NeuroRestore ACD856

Long Patent Time

AlzeCure Pharma AB (publ) (FN STO: ALZCUR), a pharmaceutical company that develops a broad portfolio of small molecule candidate drugs for diseases affecting the central nervous system, with projects in both Alzheimer's disease and pain, today announced that the United States Patent Office (USPTO) has issued a patent covering ACD856, which is being developed against Alzheimer's disease and other disorders with cognitive impairment.

USPTO has announced that they have now approved the company's patent application in the US, which refers to ACD856, the leading drug candidate in the NeuroRestore platform, which is being developed against Alzheimer's disease. The patent number is US 11,352,332 and the patent is valid until 2039.

ACD856 and other substances in the NeuroRestore platform stimulate several important signaling systems and signaling molecules in the brain such as BDNF (Brain Derived Neurotrophic Factor) and NGF (Nerve Growth Factor), which can lead to improved cognition. Previous preclinical studies have shown that AlzeCure's drug candidates strengthen communication between nerve cells and improve cognitive ability, including learning and memory functions. New preclinical results also show potential neuroprotective and disease-modifying effects with these substances. The biological mechanism behind NeuroRestore enables several indications, such as Alzheimer's and Parkinson's disease, but also depression.

=> In addition, being a NCE, there is 5 years of expected market exclusivity in the US => 2039 + 5 years = **2044**

https://www.alzecurepharma.se/en/alzecure-receives-us-patent-for-neurorestore-acd856/
https://www.alzecurepharma.se/en/alzecure-is-granted-european-patent-for-neurorestore-acd856/

AlzeCure

Our platform PAINLESS – Targeting Unmet Medical Needs within Pain

Osteoarthritis & severe pain conditions

> 300 million patients

Project: **TrkA-NAM**

Negative allosteric modulator of TrkA-receptors in research phase

Neuropathic pain

> 600 million patients

Project: **ACD440**

Topical TRPV1 antagonist in clinical Phase II

AlzeCure

TrkA-NAM – Non-Opioid Treatment of 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*

- **TrkA 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

*) NAM = Negative Allosteric Modulator

AlzeCure

Competition: Limited Competition with small molecule approach

Compound	Company	TrkA (nM)	TrkB (nM)	TrkA vs TrkB	Status	Indication
ACD137	**AlzeCure**	**0.087**	**26 400**	**30,300**	**Preclin**	**OA* & other pain conditions**
Cmpd #10 AK1830	Array Bio. Ashai Kasei	0.038	210	5,500	Phase 2b	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 ph2b** with expected **read-out summer of 2025**

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

TrkA-NAM Results – Efficacy on par with NGF antibodies

- We have used the rat **MIA model** to assess the cartilage degeneration, localized inflammation, and pain behaviors after administration of TrkA-NAM compounds, including **ACD137**, as well as **Tanezumab**.

- **RESULTS SHOW**:
  - Potent **pain relief** in both movement-induced and evoked pain
  - Significant **anti-inflammatory effect** was observed following administration of TrkA-NAM compounds

- The **analgesic effect** of ACD137 is **similar to the effect of the anti-NGF antibody Tanezumab**, which have in several clinical trials demonstrated significant and robust pain relief.
- ACD137 was also found to display a **protective effect towards articular cartilage damage** and significantly improved a number of cartilage and knee joint structural parameters.
- ACD137 was **well tolerated** with **no clinical symptoms or histopathological findings** of rapidly progressing OA, a problematic side effect observed for anti-NGF antibodies, nor were any effects seen on neuronal ganglia.

The results emphasizes the **broad applicability** of our **TrkA-NAM ACD137 in various severe pain states**, including OA

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

AlzeCure

IP position – ACD137

- Recently submitted composition of matter patent application for ACD137
- Patent life expected until **2045**
- Additional 5-year market exclusivity in the USA → **2050**

AlzeCure

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

Positive clinical trial results in 1b & 2a

- Developed **topical formulation to avoid AEs** associated with systemic TRPV1 blockade
- **Clinical trial** with topical formulation was initiated and **successfully finalized**
- Phase 1b study addressed **safety, tolerability & efficacy – POSITIVE OUTCOMES**

Now presented detailed **positive phase 2a data**

AlzeCure

A place in the market for a non-opioid for Peripheral Neuropathic Pain

Current treatment options are insufficient

- Oral treatments used for neuropathic pain have limited efficacy – **Up to 80% aren't experiencing sufficient pain relief**
- Topical treatment, including lidocaine patches and capsaicin patches are used but with limited level of efficacy
- **More than 50% of patients received opioids** despite that not recommended and demonstrated not effective in chronic use
- Recently, a novel non-opioid drug candidate for neuropathic pain received fast track designation by the FDA

=> *Huge potential* *for ACD440 Gel*

AlzeCure

Positive Phase 2a 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 2 trial

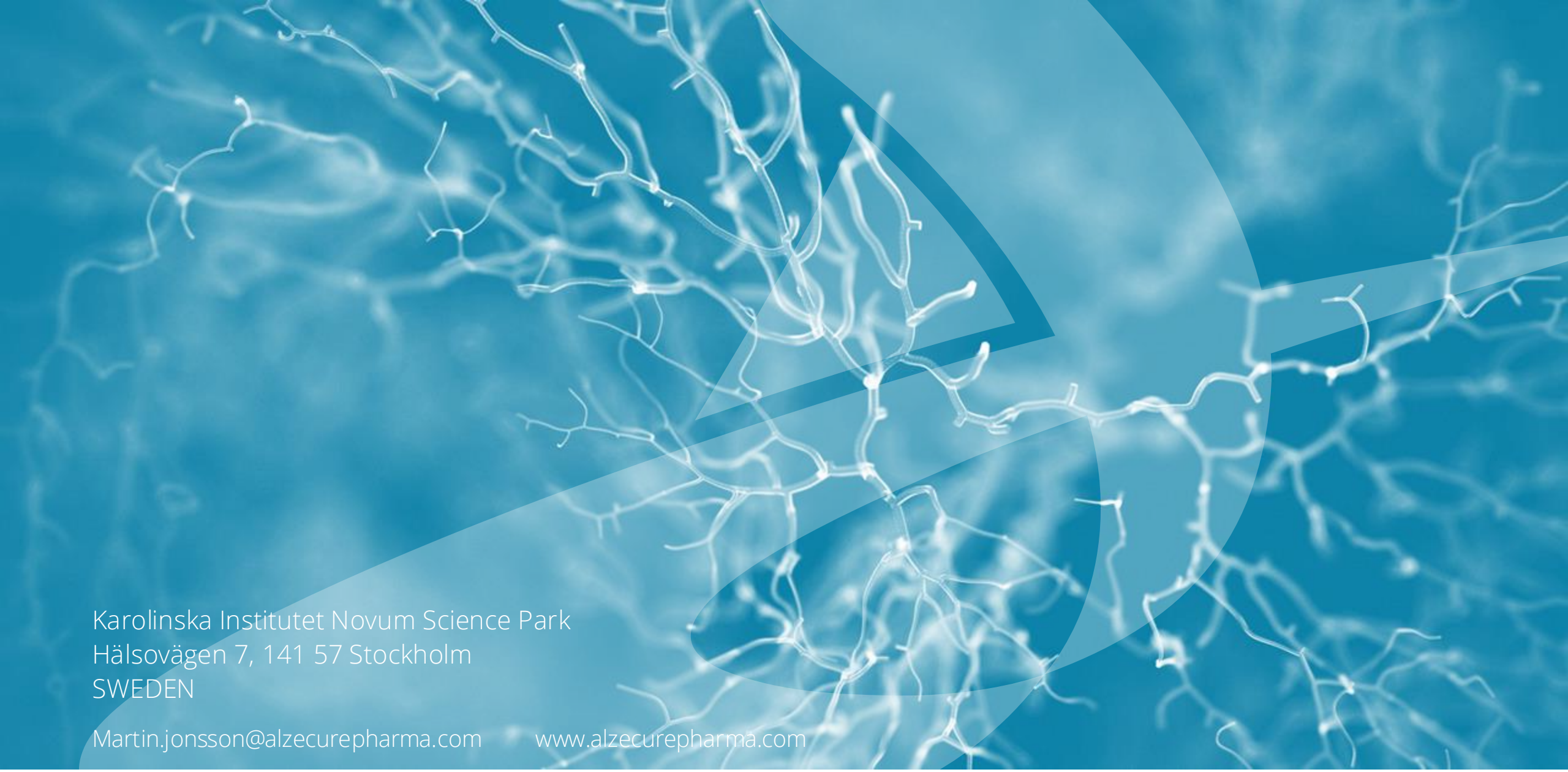
AlzeCure

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***

European Innovation Council

AlzeCure



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AlzeCure

Ownership structure

Name	Num. of shares	Capital	Votes	Verified
BWG Invest Sàrl	13,120,942	14.86%	14.86%	2025-02-28
Sjuenda Holding AB	6,999,900	7.93%	7.93%	2025-04-28
FV Group AB	6,600,000	7.47%	7.47%	2025-04-28
SEB-Stiftelsen	3,429,999	3.88%	3.88%	2025-04-28
Avanza Pension	2,630,116	2.98%	2.98%	2025-04-28
Nordnet Pension Insurance	2,563,861	2.90%	2.90%	2025-04-28
Thomas Pollare	2,272,126	2.57%	2.57%	2025-04-28
Futur Pension	2,060,496	2.33%	2.33%	2025-04-28
AlzeCure Discovery AB	1,710,000	1.94%	1.94%	2025-04-28
Acturum Life AB	1,478,872	1.67%	1.67%	2025-04-28
Total 10	**42,866,312**	**48.55%**	**48.55%**	
Others	**45,428,888**	**51.45%**	**51.45%**	
Total number of owners	3,315			2025-05-19
Total number of shares	88,295,200			2025-05-19

William Gunnarsson, Founder of Europe Orphan

Peter Thelin, Brummer & Partners

Rolf Karlsson

One of Sweden's leading bank's

AlzeCure's Chairman of the board

AlzeCure