AlzeCure Pharma
February 19, 2024

AlzeCure Pharma
Company Overview

Martin Jönsson, CEO

AlzeCure

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 neuroprotective & disease modifying potential
  - **Painless** – Innovative projects for osteoarthritic & neuropathic pain
- Listed on **Nasdaq First North Premier** Growth Market, **Sweden**, since Nov. 2018 (Ticker: ALZCUR)

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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					
	ACD857	Positive allosteric modulator (PAM) of Trk-receptors	Alzheimer's Disease					
Alzstatin®	ACD679	Gamma secretase modulator (GSM)	Alzheimer's Disease					
	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

Selected new additional CD
ACD680

Positive read-out Phase IIa
Safety, Tolerability & Pain

Selected new CD
ACD137

Phase completed

Phase ongoing

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Progress & Increased Activity in the Alzheimer's field

Scientific validation & de-risking

- Validation of treatment approach: - Amyloid protein targeting
- Positive out-comes in clinical trials: - incl in phase III + approvals
- Vastly improved biomarkers & diagnostics identifying patients
  => *Increased probability of success in future studies*

Increased investments

- Additional Big Pharma companies entering & re-enteringthe field
- More funds and private equity investment in Alzheimer's companies & projects

However …

Still No Curative or Cognitive Enhancing Treatments against Alzheimer's have so far not been developed

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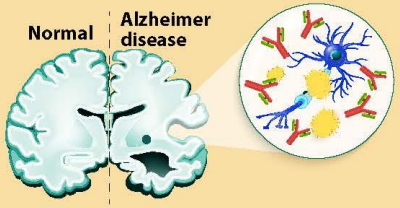
Only 5-8% of Alzheimer's patients* are estimated to be eligible for anti-bodies treatment

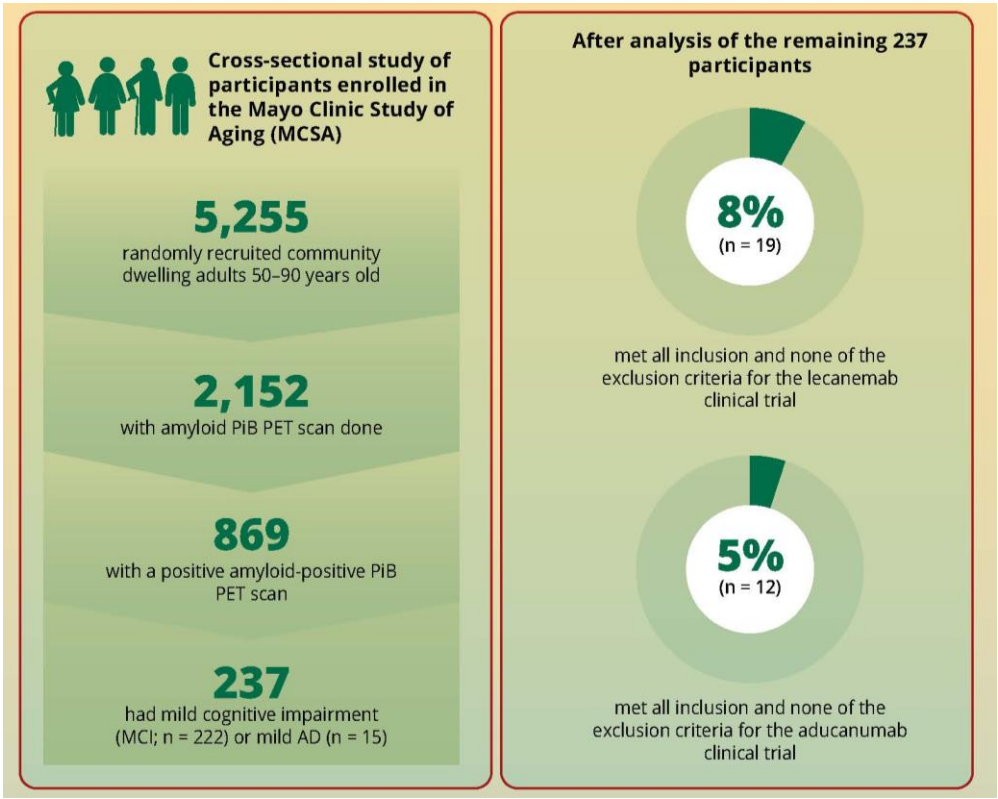
Determining Eligibility for Anti-Beta Amyloid Antibody Treatment in Adults with Mild Cognitive Impairment or Dementia



The anti-beta amyloid monoclonal antibodies (mAbs) lecanemab and aducanumab were recently approved by the FDA for use in the treatment of early symptomatic Alzheimer disease (AD)

What proportion of patients with mild cognitive impairment(MCI) or mild AD seen in a memory clinic would meet the inclusion criteria used in the clinical trials of aducanumab and lecanemab?

Only a small proportion of participants in the MCSA would be eligible for anti-amyloid mABS if the criteria used in the clinical trials of lecanemab and aducanumab were applied in routine practice



- **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

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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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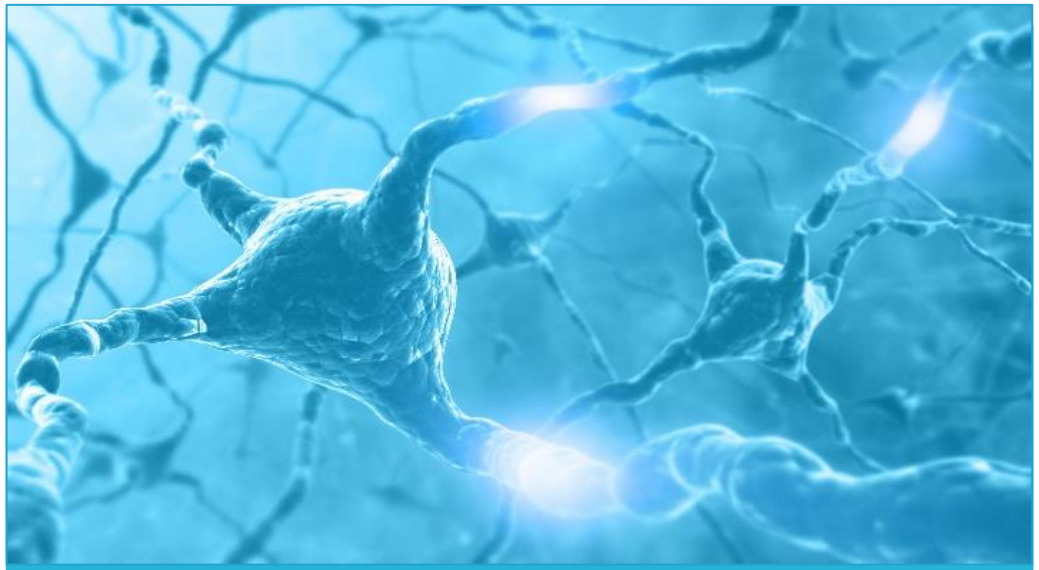
Improving Learning & Memory Capabilities



NeuroRestore®

Trk-PAM

Improving Neuronal Function & Cognition

- *Novel Oral Small Molecule*

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

=> *Prepation for phase 2*

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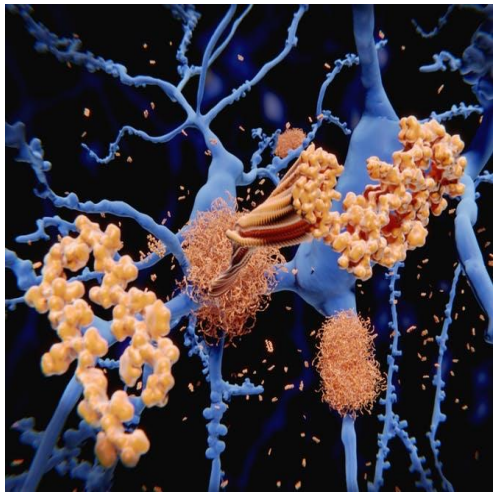
The Relevance of BDNF & NGF Signaling in Alzheimer's

- 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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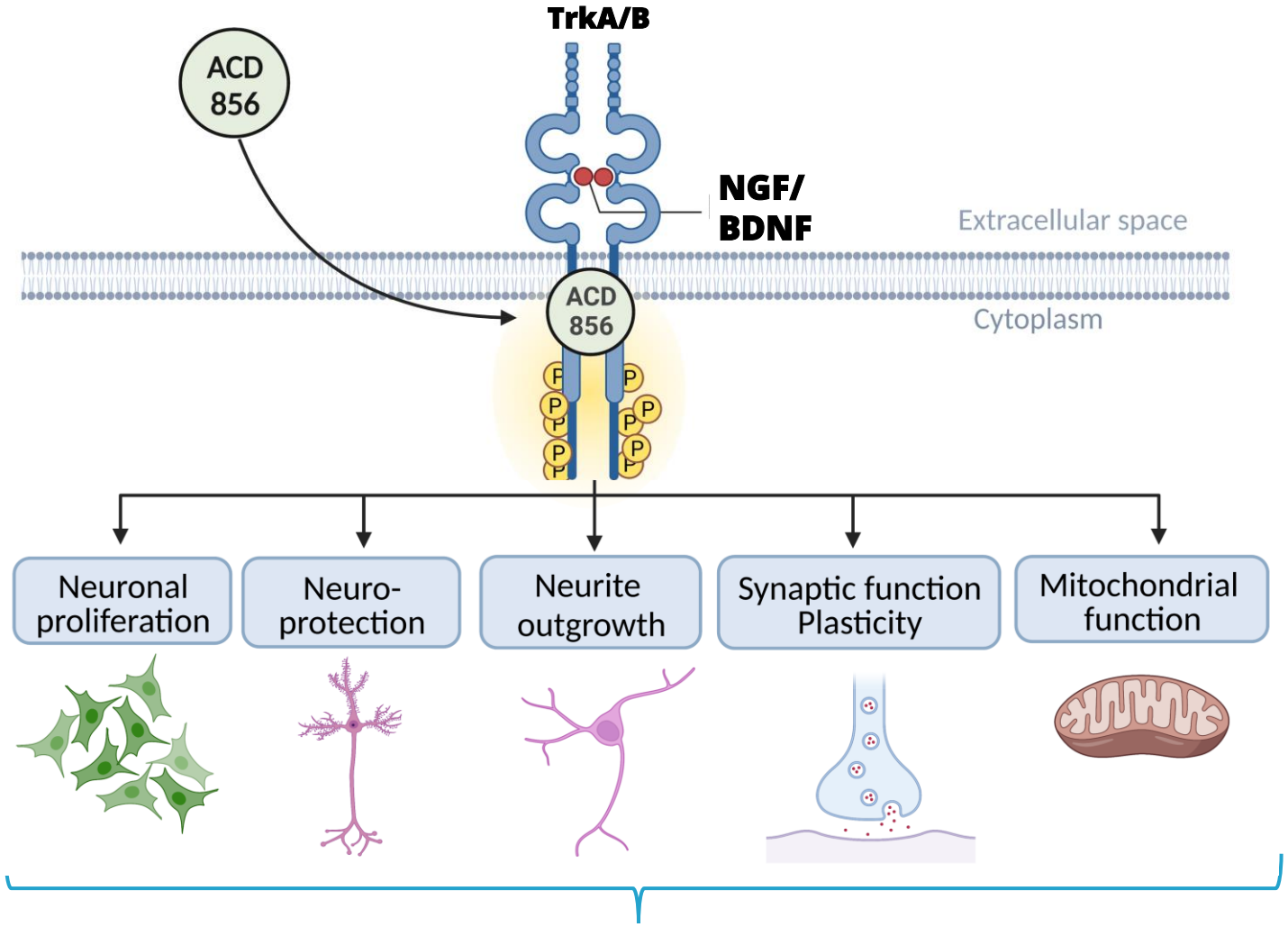
ACD856 - MoA & data supports Disease Modifying Potential

- 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**



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

Potential for Disease Modifying Effect +

Improved Learning, Memory & Depression

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ACD856 – Preclinical Data Suggests Disease Modification Effects

ACD856:

- Induces **neurite outgrowth** in PC12 cells at concentrations similar to what is found in CSF in the MAD study
- **Increases levels of synaptic markers** in PC12 cells
- Leads to **increased phosphorylation** of Trk-receptors
- **Improves mitochondrial function** and is **neuroprotective** in an energy-deprived neurotoxicity assay
- **Enhances synaptic plasticity** in the hippocampus – an area critically involved in cognitive function
- Demonstrates **long-term plasticity effects** after repeated dosing

Data Suggest a Disease Modifying Effect, Mediated by Increased Plasticity that Could Explain the Pro-Cognitive & Anti-Depressant Effects

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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TrkA-PAM by Eisai: Disease-Modifying Therapy for Neurodegenerative Diseases

FIRST-IN-HUMAN (FIH), SINGLE- AND MULTIPLE-ASCENDING-DOSE (SAD/MAD) STUDIES IN HEALTHY SUBJECTS OF E2511, A NOVEL TROPOMYOSIN RECEPTOR KINASE A (TrkA) POSITIVE ALLOSTERIC MODULATOR (PAM)

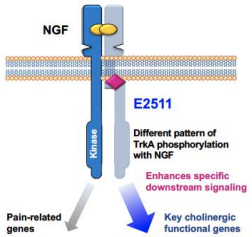
Pau Aceves[1], Nancy Hall[2], Cuiyuan Cai[3], Jagadeesh Aluri[2], Satish Dayal[1], Takuya Yagi[2], Julia Chang[2], Masaki Mikamoto[3], Garth E. Ringheim[2], Tomioka Takesuya[3], Naoki Hiramatsu[3], Robert Gordon[1], Luigi Giorgi[1]

1. Eisai Ltd., Hatfield, United Kingdom. 2. Eisai Inc., Nutley, NJ, USA. 3. Eisai Co, Ltd., Tsukuba, Japan.

Introduction

- Cholinergic innervation is affected early in multiple neurodegenerative diseases and correlates with cognitive dysfunction[1-3]
  - The progressive senescence and eventual loss of cholinergic neurons in Alzheimer's disease (AD) is correlated with cognitive impairment
- Nerve growth factor (NGF) plays a major role in the maintenance and function of cholinergic neurons[4] (see Figure 1)

Figure 1. Activation of specific trophic signaling of TrkA



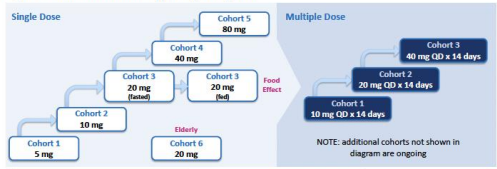
- Activation of NGF-induced trophic signaling via TrkA has potential as a disease modifying therapy to restore cholinergic innervation in neurodegenerative diseases, including AD[5-8]
- E2511 is a novel orally bioavailable small molecule TrkA biased PAM that has shown reinnervative effects on cholinergic neurons in nonclinical studies without NGF-associated hyperalgesia

Methods

- The main objectives of the E2511 FIH Phase 1 studies (SAD [completed] and MAD [ongoing]) are to evaluate the safety, tolerability and pharmacokinetics (PK) in healthy subjects
- Both SAD and MAD are randomized, double-blind and placebo-controlled studies
- The SAD evaluated 5 cohorts in adults and one cohort in elderly subjects (Figure 2); food effect was also evaluated in Cohort 3
- Repeat dosing with E2511 (once daily for 14 days) is currently under evaluation in the MAD study (3 cohorts completed; Figure 2)
- Safety was evaluated through the review of adverse events, physical examinations, clinical laboratory tests, vital signs, electrocardiogram and electroencephalograms
- Serial blood samples were collected for determination of plasma E2511 concentrations
- In MAD Cohorts 1 to 4, CSF was collected via lumbar puncture (LP) for PK/PD assessments in two occasions (1 predose and 1 postdose)

Methods *(continued)*

Figure 2. SAD and MAD Study Design Diagram



Results

Subjects

- A total of 40 and 24 healthy adult subjects have been dosed in the SAD and MAD studies, respectively (Table 1); and 5 Elderly subjects (3 E2511: 2 placebo) in the SAD

Table 1. SAD and MAD Baseline Subject Characteristics

	Single Dose								Multiple Dose					
		E2511								E2511				
	Placebo N=10	5 mg N=6	10 mg N=6	20 mg N=6	40 mg N=6	80 mg N=6	E2511 Total N=30	Total N=40	Placebo N=6	10 mg N=6	20 mg N=6	40 mg N=6	E2511 Total N=18	Total N=24
Age, mean years (SD)	34 (9)	38 (13)	40 (13)	34 (8)	35 (9)	36 (9)	36 (10)	36 (10)	45 (5.8)	27 (4)	36 (9)	32 (5)	32 (7)	35 (8)
Female, %	40	50	33	17	33	33	33	35	0	33	50	33	39	29
Race, %														
White	70	67	67	33	67	100	67	68	33	50	67	17	44	42
Black or African American	20	33	33	50	17	0	27	25	67	17	0	67	28	38
Asian	10	0	0	0	17	0	3	5	0	33	17	17	22	17
American Indian or Alaska Native	0	0	0	17	0	0	3	3	0	0	17	0	6	4
Weight, mean kg (SD)	71 (12)	73 (14)	75 (14)	71 (13)	76 (14)	72 (9)	73 (12)	72 (12)	84 (13)	76 (16)	74 (12)	73 (10)	74 (13)	77 (13)
BMI, mean kg/m², (SD)	25 (3)	25 (2)	25 (3)	24 (4)	26 (1)	25 (4)	25 (3)	25 (3)	26 (3)	25 (4)	25 (4)	25 (3)	25 (14)	25 (14)

Safety

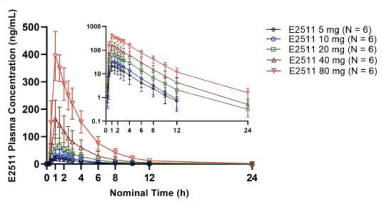
- Single and multiple oral (PO) doses of E2511 were safe and well-tolerated across the investigated doses in adult and elderly healthy subjects
- Most common (≥2) treatment-emergent adverse event (TEAE) in the SAD was contact dermatitis (same incidence in active and placebo-treated subjects [10%])
- Most common (≥2) TEAEs in the MAD were post-LP syndrome, headache, back pain and nausea; generally thought to be related to the LP procedures
- No subjects were withdrawn due to drug-related safety events
- There were no dose dependent, serious or severe TEAEs
- There were no clinically significant changes in the evaluated safety parameters (safety laboratory, vital signs, ECG, EEG and physical exam) on E2511 compared to placebo
- SAD exposure-response analyses confirmed no effects on the Holter ECG parameters, including HR, cardiac repolarization (QTcF), and cardiac conduction (PR, QRS)

Results *(continued)*

SAD: E2511 Clinical Pharmacokinetic Results

- Rapidly absorbed (t_{max}: 1 hour); with a plasma half-life of 3.19 hours (Figure 3)
- Plasma exposures (C_{max} and AUC) increased dose proportionally over the dose range of 5 to 80 mg (power model exponent [90% CI]: 1.09 [0.92, 1.25] for C_{max} and 1.08 [0.91, 1.25] for $AUC_{(0\text{-}inf)}$)

Figure 3. SAD: E2511 Plasma Concentration-Time Profiles and PK Parameters



Parameter (SD)	5 mg/kg (n=6)	10 mg/kg (n=6)	20 mg/kg (n=6)	40 mg/kg (n=6)	80 mg (n=6)
C_{max}, ng/mL	21.5 (40.8)	29.1 (98.0)	63.7 (51.8)	156 (50.8)	402 (18.2)
t_{max}, hours[1]	1.0 (1.0, 1.5)	1.0 (1.0, 2.0)	1.3 (1.0, 3.1)	1.0 (0.6, 1.5)	1.1 (1.0, 1.5)
$AUC_{(0\text{-}24h)}$, h·ng/mL	75.6 (43.9)	97.5 (91.8)	222 (55.7)	497 (55.1)	1400 (23.5)
$AUC_{(0\text{-}inf)}$, h·ng/mL	75.9 (44.1)	97.8 (92.2)	223 (55.9)	500 (55.2)	1410 (23.6)
CL/F, L/h	65.9 (44.0)	102 (92.2)	89.7 (55.9)	80.2 (55.2)	56.8 (23.7)
V_z/F, L	248 (42.9)	401 (57.2)	420 (37.1)	407 (52.4)	314 (26.5)
$t_{1/2}$, hours	2.61 (39.6)	2.72 (40.5)	3.25 (29.4)	3.52 (8.38)	3.83 (8.79)

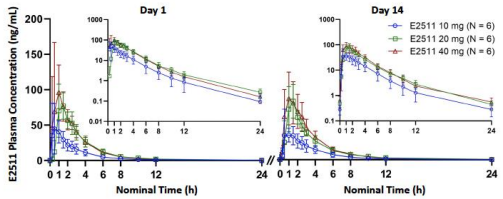
Geometric mean (%CV) values shown unless otherwise noted.
1: Median (range)

- Food (standardized high-fat, high calorie meal) led to a decrease of 19% in C_{max} and 15% in AUC compared to fasted conditions (at a dose of 20 mg)
- Plasma exposures in elderly and younger adult subjects were deemed comparable (at a dose of 20 mg), as Elderly plasma PK exposures (N=3) were fully contained within the observed range of younger adults (N=6, at same dose)

MAD: E2511 Clinical Pharmacokinetic Results

- There was little or no accumulation observed following 14 days of dosing (Figure 4)
- There was no evidence of time dependent-kinetics

Figure 4. MAD: E2511 Plasma Concentration-Time Profiles and PK Parameters



Parameter (SD)	10 mg QD		20 mg QD		40 mg QD	
	Day 1 (n=6)	Day 14 (n=6)	Day 1 (n=6)	Day 14 (n=5)	Day 1 (n=6)	Day 14 (n=6)
C_{max}, ng/mL	47.4 (88)	81.7 (50.2)	86.1 (32.3)	91.8 (22.3)	110 (59.9)	95.0 (35.9)
t_{max}, hours[1]	1.0 (0.3, 1.5)	1.0 (0.5, 2.5)	1.3 (1.0, 1.5)	1.0 (0.5, 1.5)	1.0 (0.5, 2.0)	1.0 (0.5, 2.0)
$AUC_{(0\text{-}24h)}$, ng·h/mL	116 (57.1)	140 (24.6)	247 (22.7)	271 (16.2)	276 (34.6)	309 (30.8)
CL/F, L/h	85.7 (57.4)	71.7 (24.6)	80.4 (22.8)	73.7 (16.2)	145 (34.7)	129 (30.8)
V_z/F, L	355 (43.3)	478 (26.7)	431 (20.7)	448 (20.9)	682 (25.4)	792 (39.0)
$t_{1/2}$, hours	2.87 (46.9)	4.63 (19.2)	3.72 (20.8)	4.21 (12.7)	3.27 (31.0)	4.25 (17.8)
R_{ac}, C_{max}[2]	N/A	1.06 (132)	N/A	0.997 (48.3)	N/A	0.863 (34.9)
R_{ac}, AUC[3]	N/A	1.2 (37.8)	N/A	1.03 (25.2)	N/A	1.12 (19.1)
Rss[4]	N/A	1.2 (38.1)	N/A	1.02 (25.1)	N/A	1.12 (19.2)

Geometric mean (%CV) values shown unless otherwise noted. N/A: not applicable.
1: Median (range);
2: R_{ac}, C_{max}: Accumulation ratio determined based on C_{max} [Day 14]/ C_{max} [Day 1];
3: R_{ac} AUC: Accumulation ratio determined based on $AUC_{(0\text{-}24h)}$ [Day 14]/ $AUC_{(0\text{-}24h)}$ [Day 1];
4: R_{ss}: Time dependency ratio was determined based on $AUC_{(0\text{-}24h)}$ [Day 14]/ / $AUC_{(0\text{-}inf)}$ [Day 1]

Conclusions

- E2511 presents an adequate single and multiple dose safety profile in healthy adults up to the currently highest evaluated doses (80 mg PO in SAD; 40 mg QD PO in MAD)
- E2511 has shown predictable single and multiple dose PK with a short plasma half-life that is consistent with human PK projections based on non-clinical data
  - Dose-proportional plasma PK over the dose range of 5 to 80 mg (in the SAD) and time-invariant PK (up to at least 40 mg QD in the MAD)
  - Mild effect of food on rate and extent of plasma E2511 exposure supports dosing without regards to food
  - Plasma E2511 exposures (C_{max}, AUC) in a small group of elderly subjects suggest there is no need for dose adjustment in the target population
- These results support further development of E2511 as a disease-modifying therapy for neurodegenerative diseases

References

1. Ferreira-Vieira TH, et al. Curr Neuropharmacol. 2016;14(1):101-15. 2. Hampel H, et al. Brain. 2018;141(7):1917-33. 3. Mufson EJ, et al. Expert Rev Neurother. 2008;8(11):1703-18. 4. Ginsberg SD, et al. J Neurochem. 2006;97(2):475-87. 5. Hefti F. J Neurosci. 1986;6(8):2155-62. 6. Hagg T, et al. Exp. Neurol. 1988;101:303-12. 7. Hilgert M, et al. Neurochem.Res. 2003;28:467-72. 8. Wu K, et al. Brain Res.2005;1061:107-13.

Acknowledgments

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If you have any questions about this poster, please email or call Eisai Medical Information at ESI_MedInfo@eisai.com or 888-274-2378

The data is **validating & increasing interest in NeuroRestore**

” These results support further development of E2511 as a disease-modifying therapy for neurodegenerative diseases”

AlzeCure's ACD856 in Comparison to Eisai's E2511

	Eisai E2511	AlzeCure NeuroRestore ACD856
Mechanism of Action	Positive allosteric modulator	Positive allosteric modulator
Target	TrkA	TrkA, TrkB and TrkC
Type	Novel small molecule (<400 Da)	Novel small molecule (<400 Da)
IP	Patent granted	Granted patent in US, Japan, EU, and several other territories are processing applications
Stage of dev.	Phase I: SAD/MAD, half-life = 3.2 h	Phase I: SAD/MAD, half-life = 19 h
Effect on neurite outgrowth	No, not reported	Yes, in two different in vitro models
Neuroprotective	Yes, in two in vivo models	Yes, in two in vitro models
Effect on neuro-transmitters	Yes ACh	Yes Serotonin, noradrenaline and dopamine
Effect on cognition	Not reported	**Yes, cognitive enhancement** in several models
Effect on depression	Not reported	**Yes, anti-depressant & long-term effects + additive to SSRI**

Alzecure: Cells. 2021; Drug Discov Today 2022; Psychopharmacology, 2023, International J. Mol. Sciences 2023
Eisai: AAIC, P51985, 2021; ADPD, P186, 2022; AAIC, P62590 and P66208, 2022

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ACD856 – Evidence of Improvements to Learning & Memory

Stages of memory formation

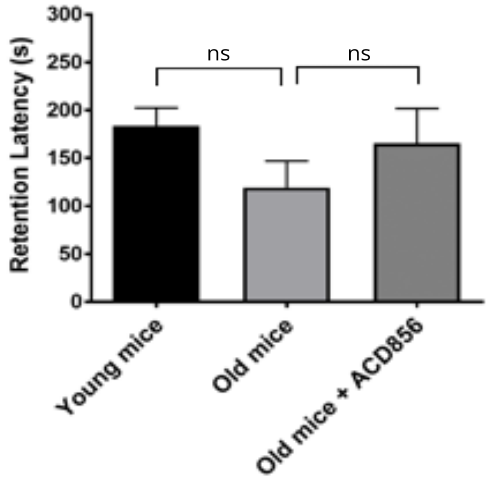
LEARN → STORE → REMEMBER

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

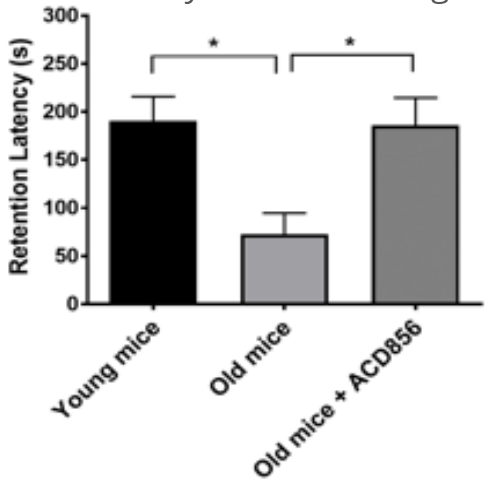
Single dose ACD856 (-30 min) → Learning (t=0 min) → Test #1 (t=1 day) → Test #2 (t=11 days)

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

Memory test #1,
1 day after learning



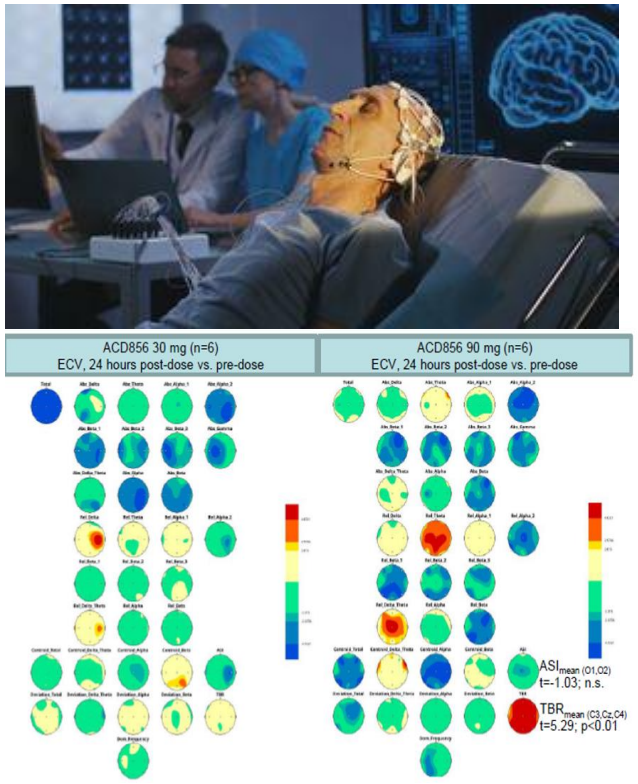
Memory test #2,
11 days after learning



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

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



Vigilance control brain maps for 30 and 90 mg cohorts

=> Being prepared for ph 2

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

AlzeCure

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

www.alzecurepharma.com - info@alzecurepharma.com

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Closing remarks & Take Home Messages

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Take Home Messages

On the Alzheimer's market & unmet medical needs

- **Early detection** of the risk to develop Alzheimer's, **up to 15 years before clinical symptoms**, opens up the **need for new treatments** aiming to minimize toxic amyloid load
- There is a **huge unmet medical need** in the Alzheimer's therapy market, **even if antibodies are available** (- only 5-8% of Alzheimer's pts are eligible)
- There are still **no curative or preventive Alzheimer's treatments approved**
- **Gamma-secretase modulators** is a new and **promising class of Alzheimer's therapies**, with **several different target patient populations**

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Take Home Messages

On AlzeCure Pharma

- AlzeCure is a clinical stage Alzheimer's and CNS company with **several novel first-in-class and/or best- in-class assets**
- AlzeCure's ambition is to develop Alzheimer's **therapies that both stop disease progression and improve cognition**
- **NeuroRestore ACD856**, currently in **preparation for clinical phase 2 studies**, is being developed as a cognitive enhancer with potential neuroprotective and neurodegenerative properties
- **Alzstatin,** AlzeCure's **gamma-secretase modulator** program, is currently in preclinical phase and is **planned to initiate clinical phase 1 studies in 2025**
- **Efficacy data & Proof of Mechanism** could be **established already in phase 1** for our gamma-secretase modulator, which is expected to be a strong value driver

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- The recorded presentation and slide sets will be made available on AlzeCure's web page
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Thank You
for attending

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SWEDEN

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