Corporate Deck

Alector Brain Shuttle Platform & Pipeline

March 2026

Forward-Looking Statement

This presentation contains forward-looking statements that involve substantial risks and uncertainties. All statements other than statements of historical facts contained in this presentation are forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as "anticipate," "believe," "continue," "could," "estimate," "expect," "intend," "may," "plan," "potentially," "predict," "should," "will" or the negative of these terms or other similar expressions. Forward-looking statements contained in this presentation also include, but are not limited to, statements regarding: our future financial condition, including the sufficiency of cash to fund operations through 2027; results of operations; business strategy and plans; the beneficial characteristics, safety, efficacy, and therapeutic effects of our product candidates and our Alector Brain Carrier (ABC) blood-brain barrier technology platform; our plans, timelines and expectations related to our product candidates in our clinical and pre-clinical programs and our ABC platform, including with respect to the availability of data, the initiation of future clinical trials and plans and expectations regarding planned regulatory filings with respect to such programs; expectations regarding the timing and financial benefit of our collaborations; and objectives of management for future operations, as well as statements regarding industry trends.

We, Alector, Inc. ("Alector"), have based these forward-looking statements largely on our current expectations and projections about future events and trends that we believe may affect our financial condition, results of operations, business strategy and financial needs. These forward-looking statements are subject to a number of risks, uncertainties and assumptions, including, among other things: Alector's plans relating to its research programs, preclinical and clinical development programs and the development and manufacturing of its product candidates and its Alector Brain Carrier (ABC) blood-brain barrier technology platform, including its programs and product candidates incorporating the ABC platform; the ability of Alector's clinical trials to demonstrate safety and efficacy of its product candidates, and other positive results; the timing and focus of Alector's clinical trials, and the reporting of data from those trials, including the anticipated timing and detail regarding PROGRESS-AD; the expected potential benefits of strategic collaborations with third parties and Alector's ability to attract collaborators with development, regulatory and commercialization expertise; Alector's estimates of the number of patients in the United States, the European Union and world-wide who suffer from the diseases it is targeting and the number of patients that will enroll in its clinical trials; the anticipated timing of enrollment in its clinical trials; the size of the market opportunity for Alector's product candidates in each of the diseases it is targeting; Alector's ability to expand its product candidates into additional indications and patient populations; the success of competing therapies that are or may become available; the beneficial characteristics, safety, efficacy, and therapeutic effects of Alector's product candidates; the timing or likelihood of regulatory filings and approvals, including Alector's plans for IND submissions and any expectation to seek special designations, such as orphan drug or breakthrough designation, for its product candidates for various diseases; Alector's ability to obtain and maintain regulatory approval of its product candidates; Alector's plans relating to the further development and manufacturing of its product candidates, including additional indications that it may pursue; existing and future regulations and regulatory developments in the United States and other jurisdictions; Alector's reliance on third parties to conduct clinical trials of its product candidates, and for the manufacture of its product candidates for preclinical studies and clinical trials; the impact of worldwide economic conditions, including macroeconomic conditions, inflation, supply chain disruptions, trade tariffs, and economic impacts of pandemics or other public health outbreaks and geopolitical events on our business; and the other risks, uncertainties and assumptions discussed in the public filings we have made and will make with the Securities and Exchange Commission ("SEC"). These risks are not exhaustive. New risk factors emerge from time to time, and it is not possible for our management to predict all risk factors, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in, or implied by, any forward-looking statements. You should not rely upon forward-looking statements as predictions of future events. Although we believe that the expectations reflected in the forward-looking statements are reasonable, we cannot guarantee future results, levels of activity, performance or achievements.

This presentation may contain results from our clinical trials or statements regarding ongoing clinical trials, and this presentation does not speak to, and you should make no assumptions about, any further information or data relating to those trials. In addition, the information we have chosen to publicly disclose regarding our product candidates has been selected from a more extensive amount of available information. You or others may not agree with what we determine is the material or otherwise appropriate information to include in our disclosure, and any information we determine not to disclose may ultimately be deemed significant with respect to future decisions, conclusions, views, activities or otherwise. If the initial data that we report differ from updated, late, final or actual results, or if others, including regulatory authorities, disagree with the conclusions reached, our ability to obtain approval for, and commercialize our product candidates may be harmed, which could harm our business, financial condition, results of operations and prospects.

This presentation discusses certain investigational therapeutic agents which have not yet been approved for marketing by the U.S. Food and Drug Administration. No representation is made as to the safety or effectiveness of our product candidate for the therapeutic use for which it is being studied.

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Except as required by law, we undertake no obligation to update any statements in this presentation for any reason after the date of this presentation. We have filed Current Reports on Form 8-K, Quarterly Reports on Form 10-Q, Annual Reports on Form 10-K, and other documents with the SEC. You should read these documents for more complete information about us. You may obtain these documents for free by visiting EDGAR on the SEC website at www.sec.gov.

Unlocking the Brain: A New Era in Multi-Billion Dollar CNS Markets

Platform + Pipeline Multiplier: Alector Brain Carrier (ABC) and Pipeline De-Risk Each Other

ABC PLATFORM

Versatile TfR-based delivery, enabling differentiated brain exposure across antibodies, enzymes, and siRNA with reduced anemia risk

PIPELINE

Alector is deploying this platform to advance multiple wholly-owned brain-enabled therapeutics in large CNS markets

Fully Owned Platform and Pipeline;
Opportunity for Pharma Partnerships and Standalone Enterprise

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4 ABC-Enabled Programs Across Large CNS Markets; Targeting Multiple INDs 2026–2028;
$256M Cash & Investments; Runway at least through 2027

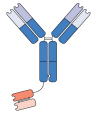
Our Assets

Nivisnebart (AD)
PGRN elevating antibody

2026: Ph 2 futility analysis + trial completion
2027/8: Potential Ph 3 pivotal trial, early AD

- FIC PGRN elevation for AD
- Standalone and combination therapy
- Favorable safety profile

AL137/AL037 (AD)*
ABC Anti-Aβ Antibody



2026: NHP IND-enabling studies ongoing
2027/8: Ph 1b – amyloid PET reduction, safety

- High brain exposure, planning SC delivery
- Full effector function to optimize efficacy
- Targeting no ARIA, anemia, or IRR

AL064/AL164 (AD)*
ABC Tau siRNA

2026: NHP knockdown validation
2027/8: Ph 1b – Tau reduction & safety

- Durable target knockdown
- Targeting SC delivery every 3 months
- Favorable safety

ADP062 (PD)*
ABC α–Syn siRNA

2026: NHP safety and efficacy planned
Targeting 2028: First-in-human – Synuclein reduction

- Potentially superior efficacy vs. blocking Abs
- Durable target knockdown
- Targeting SC delivery every 3 months

ADP065 (AD)*
ABC NLRP3 siRNA

2026: NHP safety & efficacy planned
Targeting 2028: First-in-human – functional readouts of NLRP3 reduction in CSF (IL-1β, IL-18)

- Potentially superior brain penetration and knockdown vs. small molecules
- Targeting SC delivery every 3 months

AL050 (PD, LBD)*
ABC GCase ERT

2026: NHP – 18x brain exposure, durable rescue
2027/8: Ph 1b – Engagement, GlcSph reduction

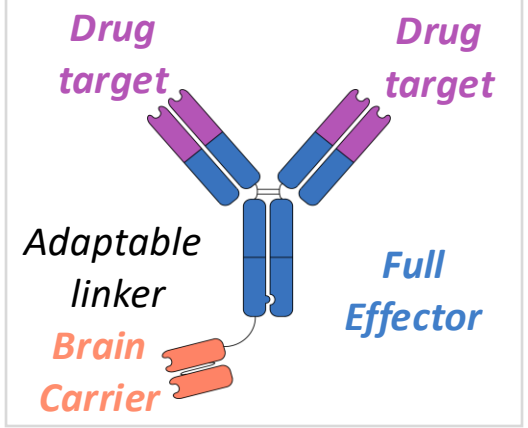
- Targeting First ERT for PD-GBA
- Targeting all GCase LOF mutations
- Enables flexible dosing regimen

*Fully owned programs
** Dates and differentiation represent Forward-Looking Statement associated with substantial risks and uncertainties listed in slide 2. Ph- Phase; PGRN- Progranulin; FIC- First In Class; AD- Alzheimer's disease; PD-Parkinson's disease; LBD - Lewy Body Dementia ; α-Syn-Alpha Synuclein ; SC- subcutaneous; NHP- Non-Human Primates; ABC- Alector Brain Carrier; ARIA- Amyloid-Related Imaging Abnormalities; IRR- Infusion-related reaction; ERT- Enzyme Replacement Therapy

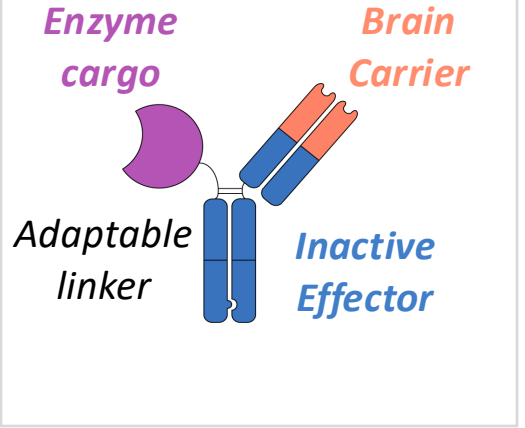
Alector Brain Carrier (ABC) Platform Built on Seven Years of Experience

- Evaluated Across 13 Cargos and 3 Modalities in Cell Culture, Rodents, and NHPs
- Tunable TfR Binding Affinity & Kinetics for Maximal Brain Penetration
- Fully-Owned & Unencumbered
- High Manufacturability & Stability
- Minimized Hematologic Risk Even With Full Effector Function
- Applicable Across Antibodies, Enzymes & siRNA

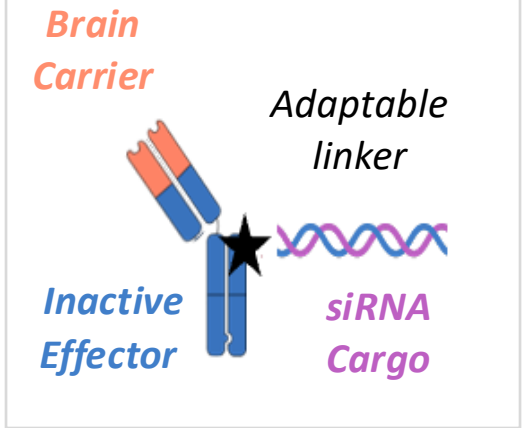
Antibody Cargo



Enzyme Cargo



Nucleic Acid Cargo



Alector Brain Carrier Facilitates High Brain Concentration of Cargo at Low Dose

Company	alector	DENALI	Roche	BIOARCTIC
Calculated Brain Concentration with 10mg/kg ***Extrapolated Brain Concentration 24-72h, following 10 mg/kg IV injection in NHP. Linear dose response assumed.***	**12-28 nM (Anti-Aβ antibody)**	**3 nM (Anti-BACE antibody)**	**2.1 nM (Anti-Aβ antibody)**	**5.7 nM (Anti-Aβ antibody)**

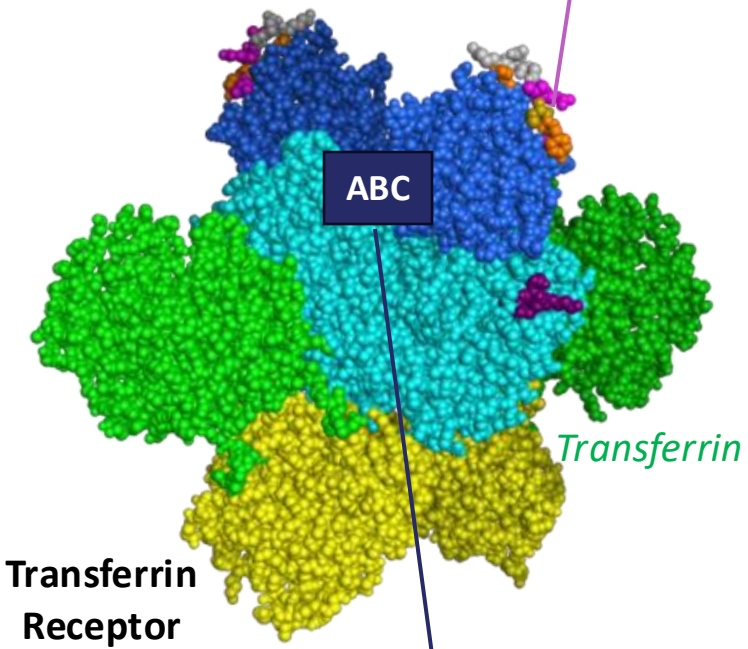
alector

	Frontal cortex levels	Fold increase*	Dosing and administration
AL037 Anti-Aβ	0.4 ug/mL, 3.8nM	18x	3 mg/kg, 24hrs. post IV administration
AL137 Anti-Aβ	0.8 ug/mL, 8.4nM	32x	

*Kariolis et al, STM 2020; Edavettal et al, MED 2022; Grimm et al., MABS, 2023; Freskgård, PEGS Europe 2024; *Relative to naked (no shuttle) molecule; Roche had reported 400 ng/g in NHP cortex, which in our calculations is equivalent to 2.1 nM with 10 mg/kg IV injection; Bioarctic had reported 0.1 nmol/L/nmol/kg in NHP parietal cortex, equivalent to 5.7 nM with 10 mg/kg IV injection predicted; Denali had reported reaching 9 nM in NHP frontal cortex with 30 mg/kg IV injection, which with the imprecise assumption of a linear dose response would represent. ~3 nM with 10mg/kg.*

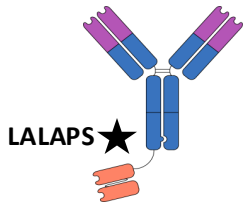
TfR Binding Epitope Masks FcγR Co-Binding, Driving an Improved Hematologic Profile

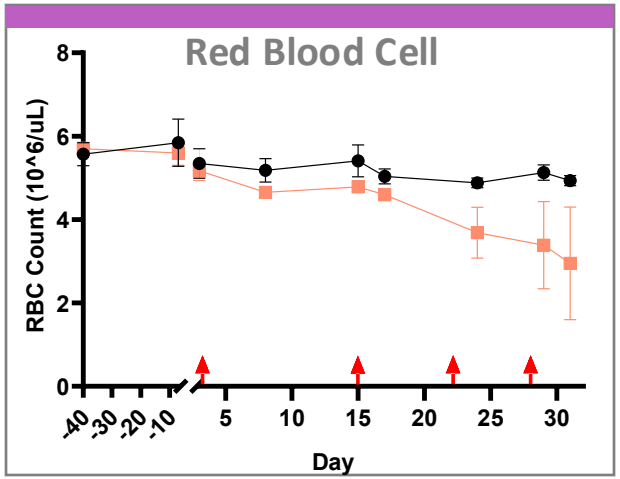
Exposed Epitope
(Does not prevent co-binding to FcγR)
(same epitope as Korsana, Denali, Roche)

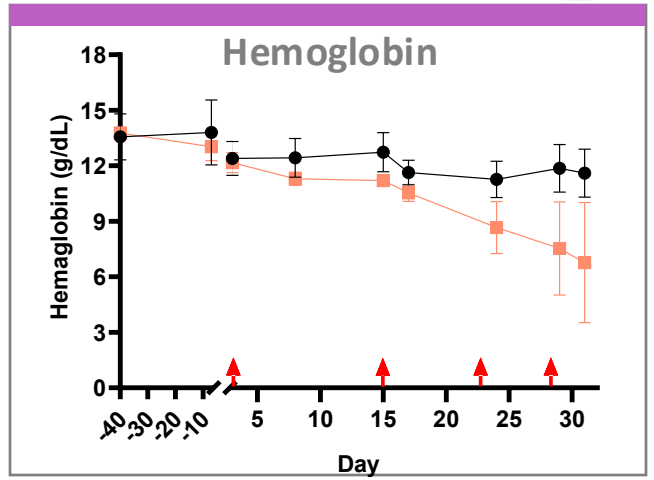


Alector ABC Epitope
(Prevents co-binding to FcγR)

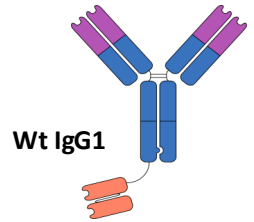
Exposed Epitope + Cis LALAPS

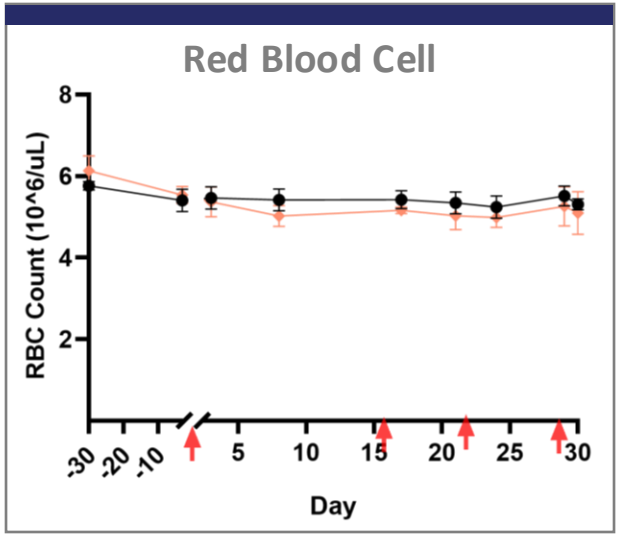


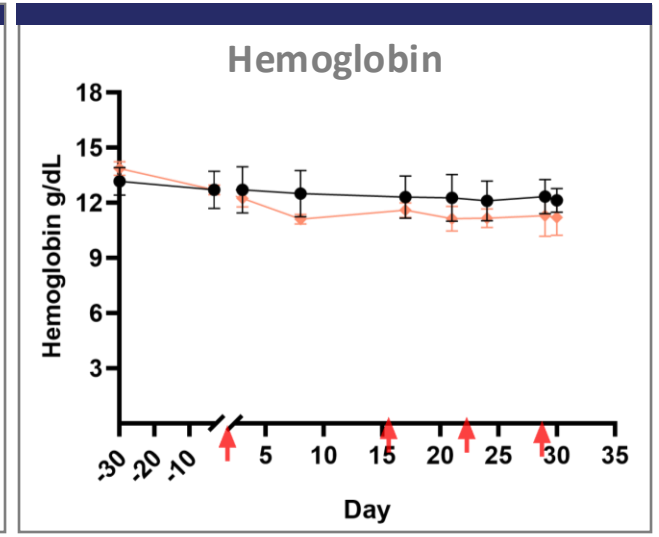




Alector Masked ABC Epitope + Full Effector Function

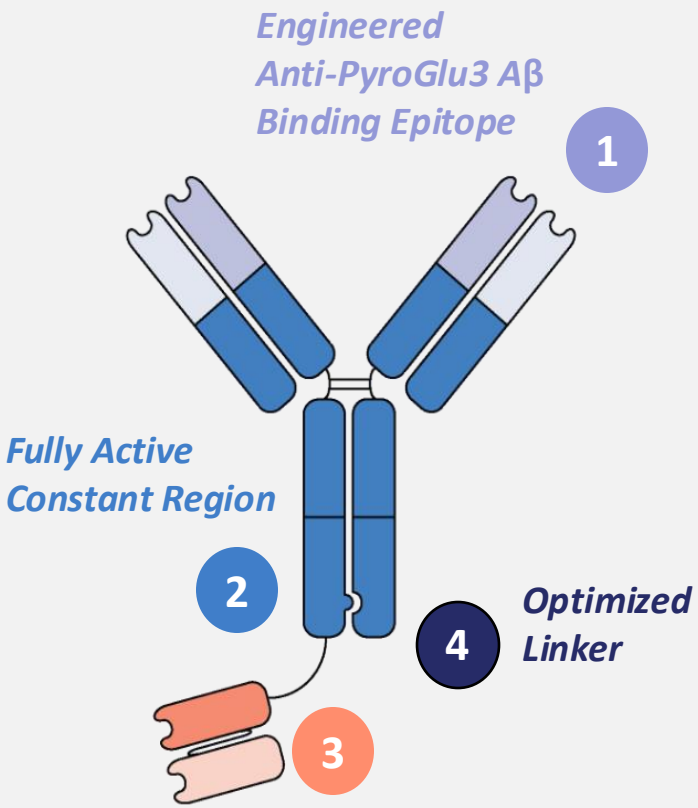






Structure of human transferrin receptor-transferrin complex from 1SUV..ABC and exposed epitopes bind TfR with similar affinity. The exposed epitope targets the same TfR region as Roche (*Alector internal data)*, Denali (*Kariolis et al., 2000)*, and Korsana (February 2026 corporate deck) NHP IV dosing (50 mg/kg, D1–D29) showed stable hematology (reticulocytes, hemoglobin, RBCs).

AL137 / AL037*: Designed for Full Potency Without Sacrificing Safety



Optimized TfR-binding Epitope, Affinity and Binding Kinetics.

THE CHALLENGE

Aβ drives AD progression, yet anti-Aβ antibodies face ARIA or anemia; anemia mitigation via dampened effector may compromise plaque clearance.

THE AL137/AL037 SOLUTION

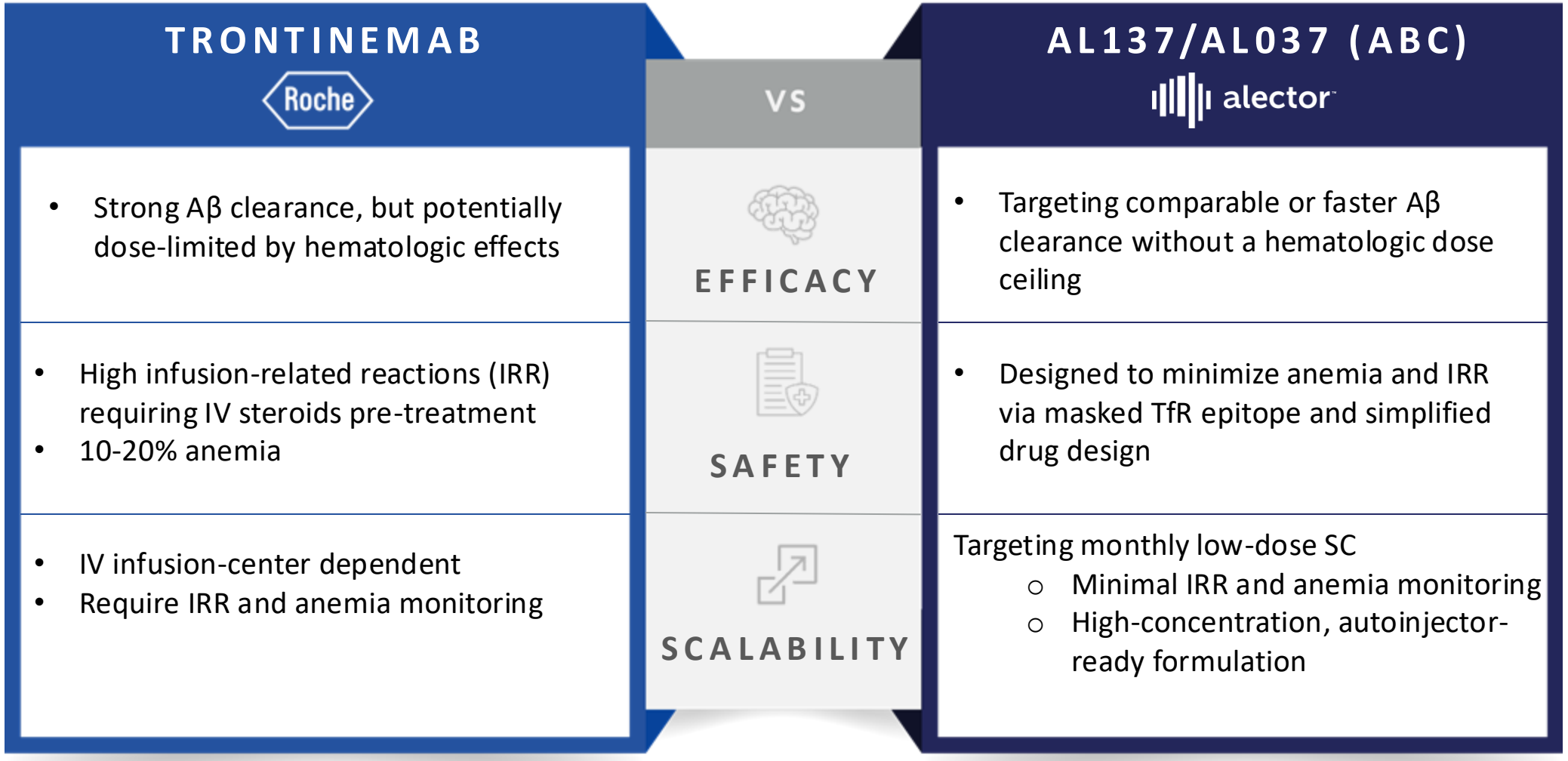
Engineered for high brain penetration, efficacy, and safety. The TfR-binding epitope prevents FcγR co-binding, while preserving FcγR engagement at Aβ plaques, improving hematologic safety without compromising efficacy.

THE DELIVERABLES

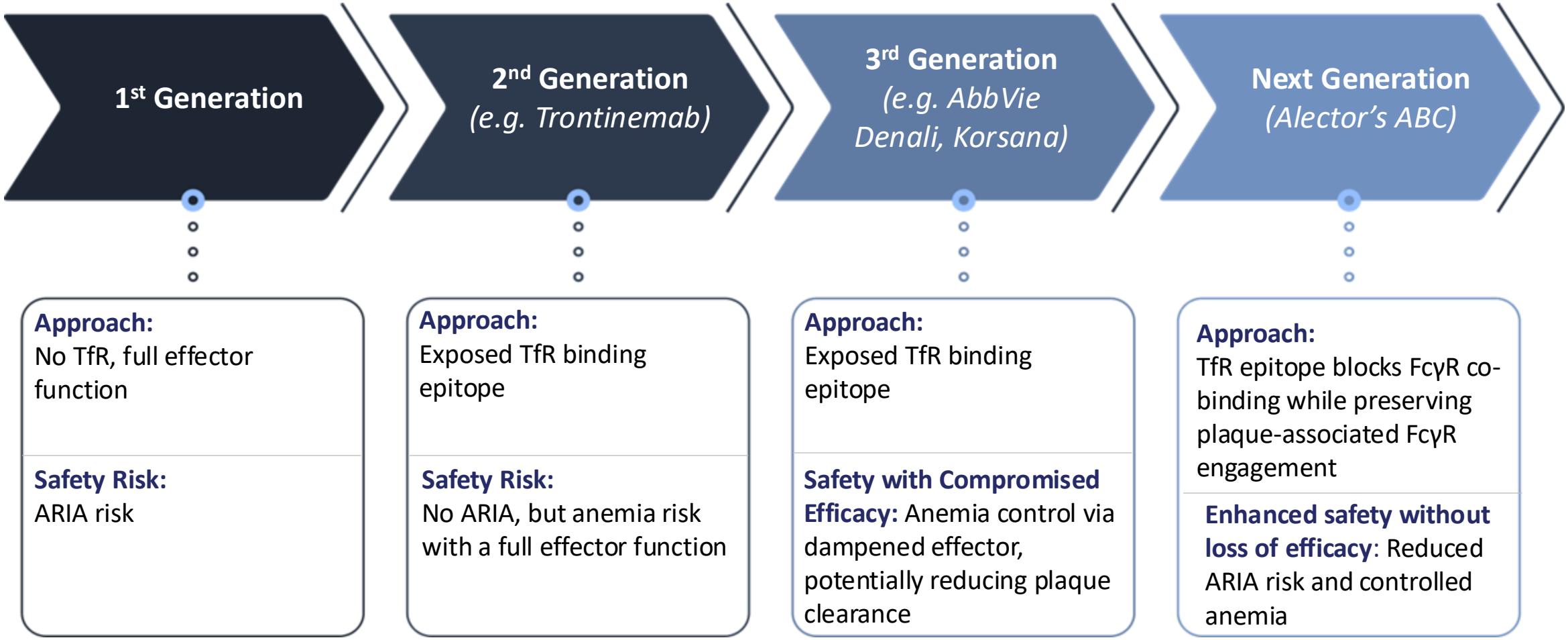
2025/6 Potent brain penetration, NHP supported, monthly SC optionality
2H 2026: IND-enabling studies to select AL137 or AL037 as the lead
1H 2027: First-in-human to confirm CNS exposure and SC dosing

**AL137 and AL037 have distinct TfR binding domains but are otherwise identical.*

AL137 / AL037: Next-Generation Brain Enabled Anti-Aβ Antibody Designed to Overcome Trontinemab's Tolerability and Scalability Constraints



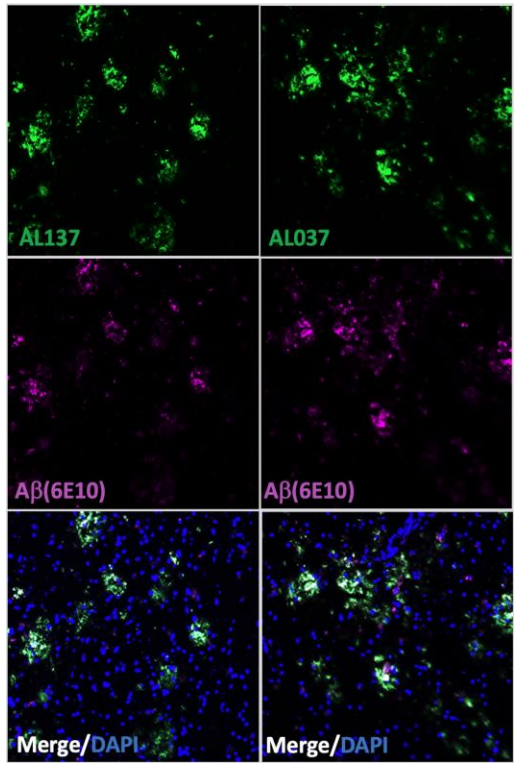
AL137 / AL037 Designed to Overcome Efficacy Constraints of 3rd Generation Anti-Aβ Antibodies



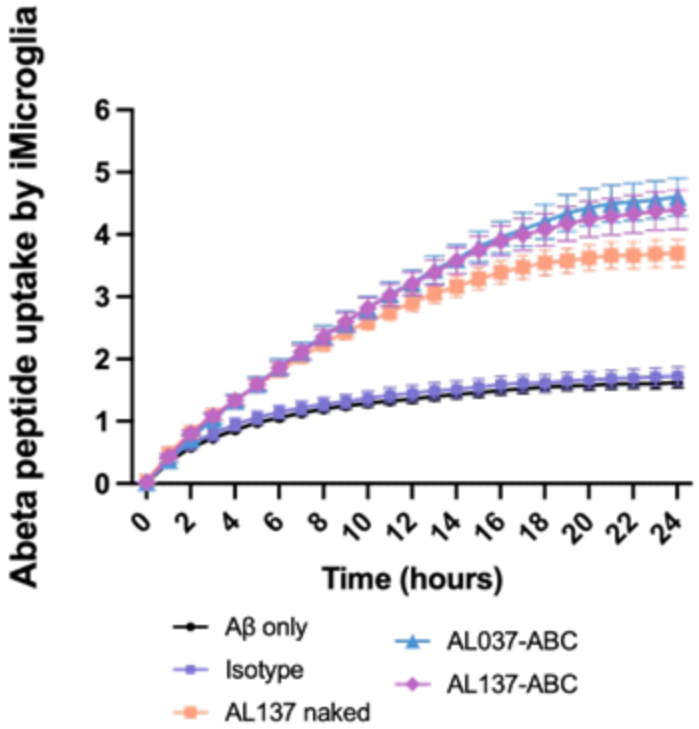
The Alector Advantage: Our 'Masked Epitope' technology targets high brain penetration and full efficacy without the safety trade-offs of previous generations. We don't choose between potency and safety—we aim to capture both in full.

AL137 / AL037* Bind Aβ Plaques in AD Brain and Facilitate Phagocytosis

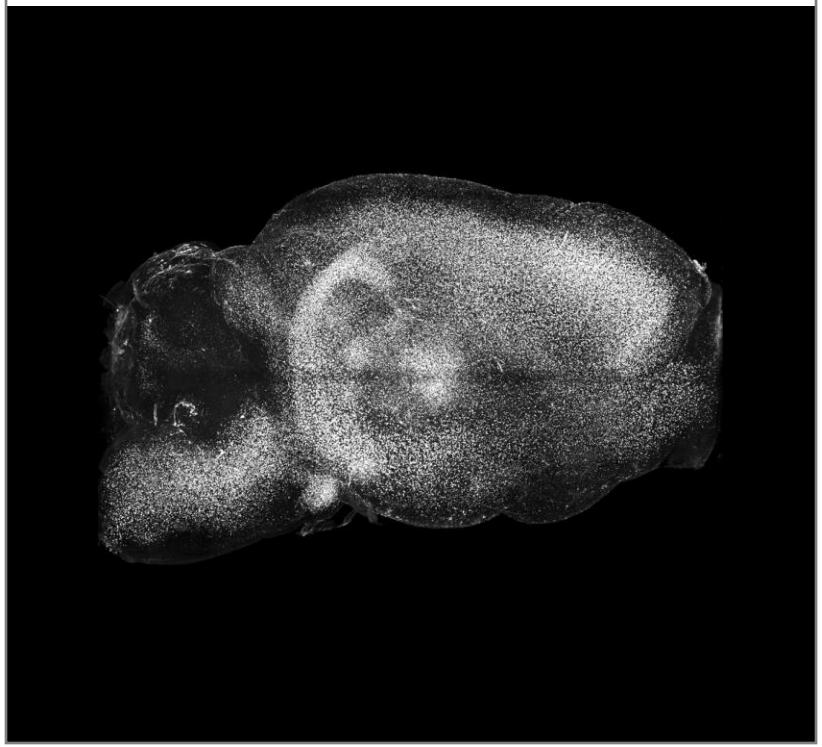
AL137/AL037 Colocalize with Aβ Plaques on Human AD Brain Sections



AL137/AL037 -Dependent Phagocytosis of PyroGlu3 Aβ by Human iPSC Microglia



AL137/AL037 with a Surrogate TfR Binding to Aβ Plaques in 5XFAD



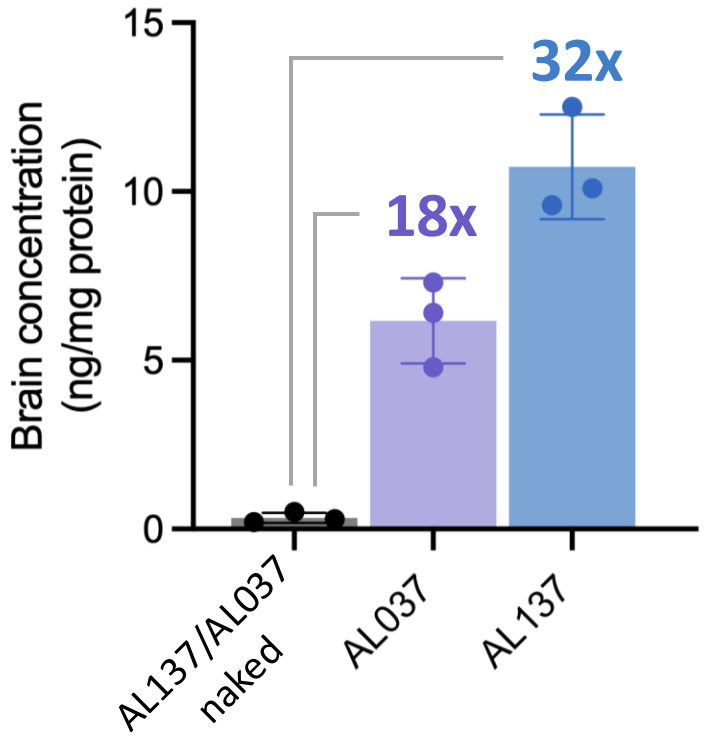
*AL137 and AL037 have distinct TfR binding domains but are otherwise identical.

Left: *Unfixed human AD sections (medial temporal gyrus) stained by IFL with AL137/AL037 (green) and anti-Aβ (6E10, pink); nuclei in DAPI (blue).* ***Middle:*** *Isotype, AL137-Naked, AL137, or AL037 incubated with pHrodo-pyroGlu3 Aβ in iPSC-derived CNS triple cultures; microglial uptake imaged over 24h.* ***Right:*** *Light sheet microscopy in 9-month 5xFAD mice dosed 3×/week at 5 mg/kg.*

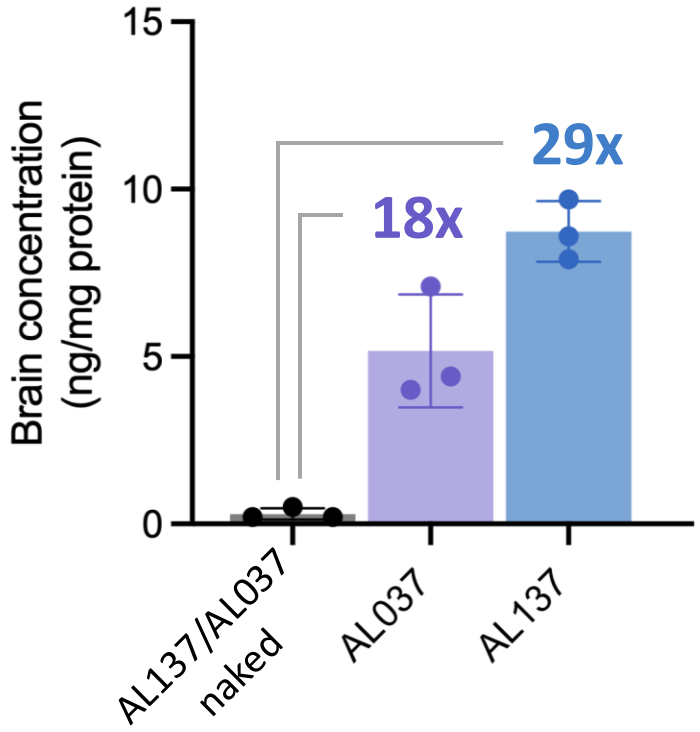
Enhanced Brain Uptake with AL037 and AL137

AL137 and AL037 at 3mg/kg reach 8.4nM and 3.8nM in frontal cortex, respectively representing ~6 and ~15-fold higher brain levels than trontinemab*

Frontal Cortex



Hippocampus

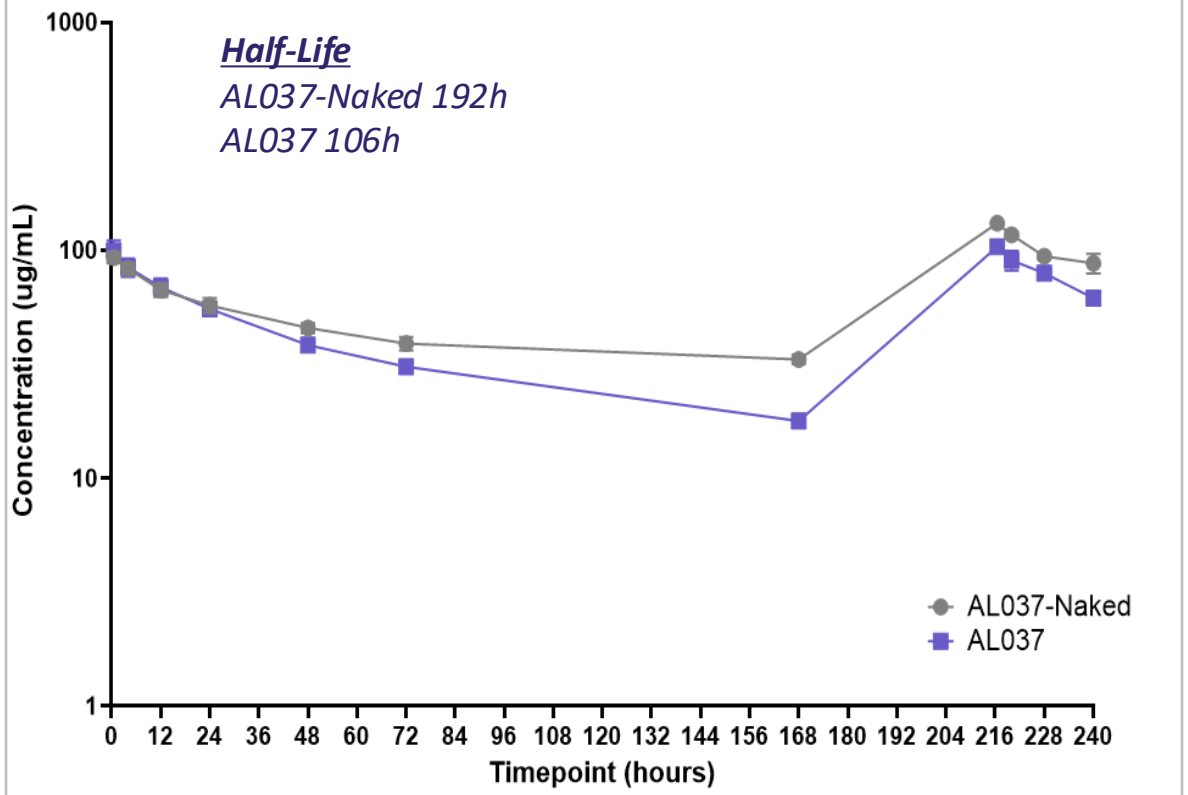


*Three NHP per group were dosed on days 1 and 9. Brain tissues were collected 24 hours after the second injection and drug levels were measured in the vessel-depleted fraction. *E.g. to our calculations, trontinemab is reaches 2.1nM in the NHP vessel-containing cortex 24h following a single injection of 10mg/kg (Grimm et al., MABS, 2023).*
AL137/AL037 Naked = AL137/AL037 without ABC platform.

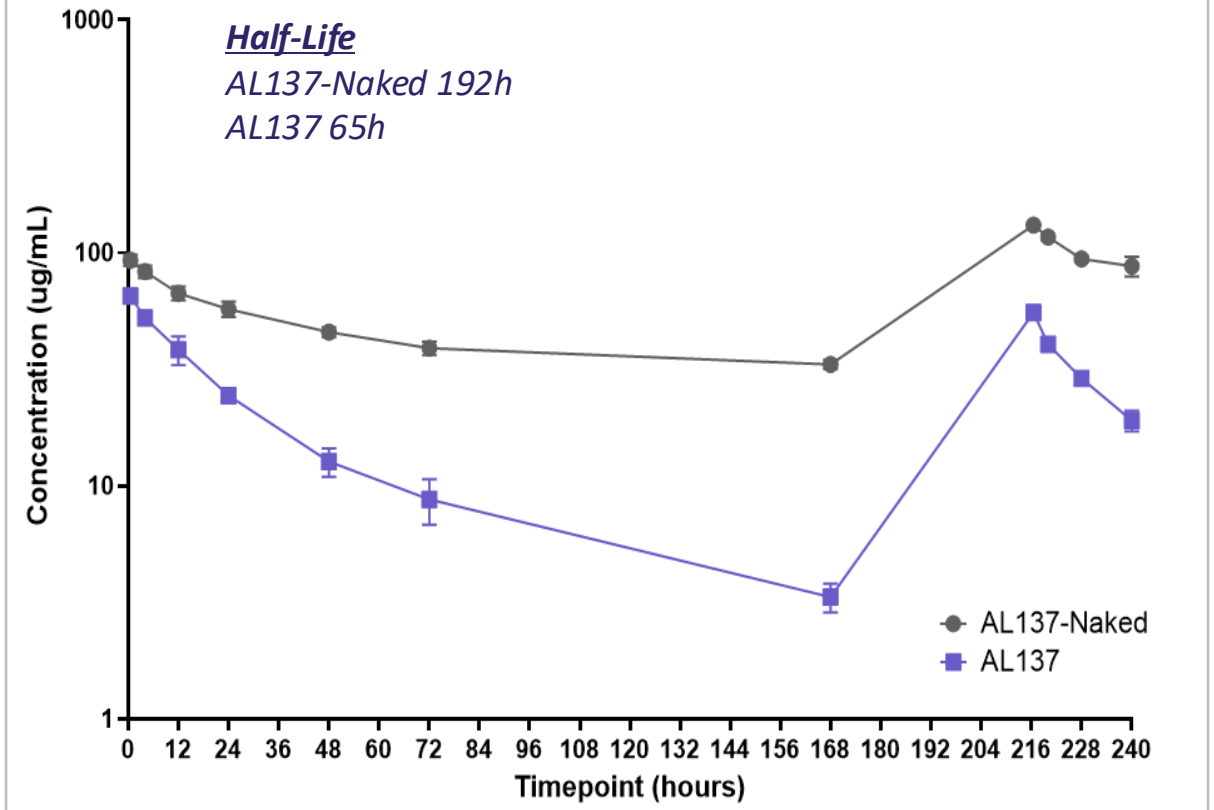
AL037 and AL137 Display Durable Serum Half-life

A shorter systemic half-life of AL037 and AL137 than a naked antibody may improve safety by limiting peripheral reticulocyte engagement (anemia) and vascular amyloid exposure (ARIA) with minimal effect on efficacy

AL037 (3 mg/kg) Serum Half-Life in NHP

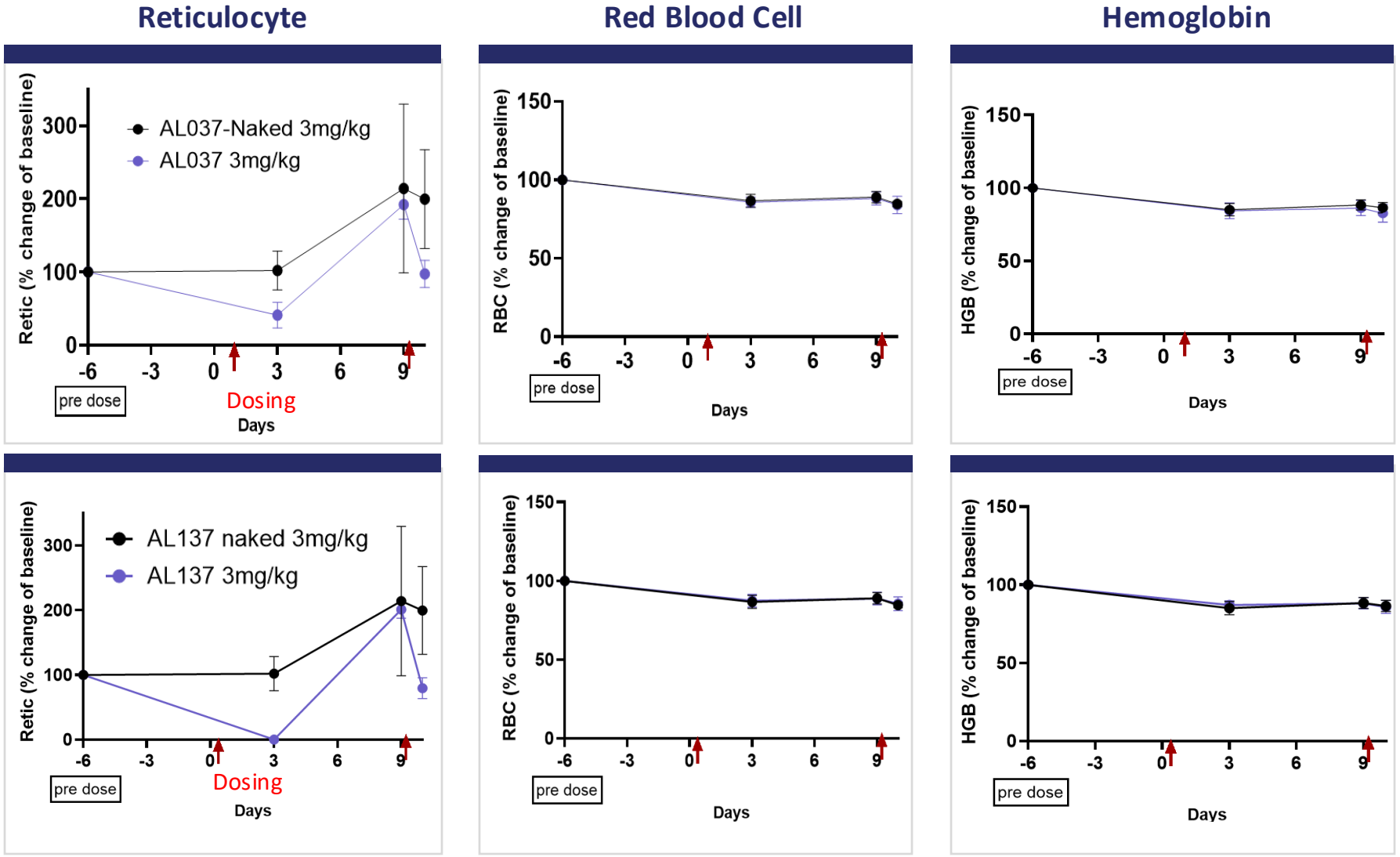


AL137 (3 mg/kg) Serum Half-Life in NHP



Three NHPs per group were injected on days 1 and 9. Serum samples were analyzed for the levels of AL037-Naked (without the ABC platform) and AL037.

AL037 and AL137 Impact on Reticulocytes RBC and Hemoglobin



Three NHP per group were injected on days 1 and 9 (red arrows). Blood samples were analyzed for reticulocytes, red blood cell counts and hemoglobin levels at the indicated times. AL037 and AL137 caused a transient decrease in reticulocytes but did not negatively impact red blood cell count. AL037-Naked = AL037 without ABC platform.

Safety Features: AL037 was Well-Tolerated in NHPs at Doses Up to 30 mg/kg

Toxicological Parameters Evaluated:

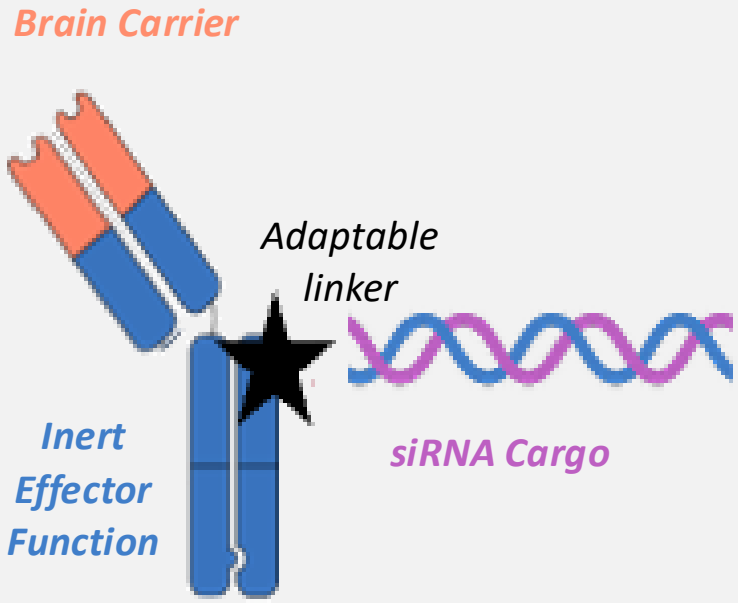
- Mortality/Morbidity
- Clinical observations
- Body weight
- Food consumption
- Hematology
- Gross necropsy observations
- Tissues collected for potential histopathology evaluation

Summary:

- AL037 was well tolerated at doses up to 30mg/kg (subcutaneous compatible)
- No reduction in RBC or hemoglobin were identified throughout the conduct of the study
- No test-article related findings were identified throughout the conduct of the study

Three NHP per group were dosed on days 1 and 9. peripheral and Brain tissues were collected 24 hours after the second injection and toxicological parameters were evaluated

AL064/AL164*: Aiming to Achieve Brain-Wide Tau Reduction Without Invasive Spinal Injections



THE CHALLENGE

Tau drives AD progression, yet antibodies fail against its intracellular pathology, and Tau ASOs require invasive intrathecal delivery with uneven distribution

THE AL064/AL164 SOLUTION

AL064/AL164 is designed for intracellular Tau reduction, directly targeting a central driver of neurodegeneration. IV/SC systemic delivery and broad homogeneous brain distribution are intended to **improve safety, scalability, and access**.

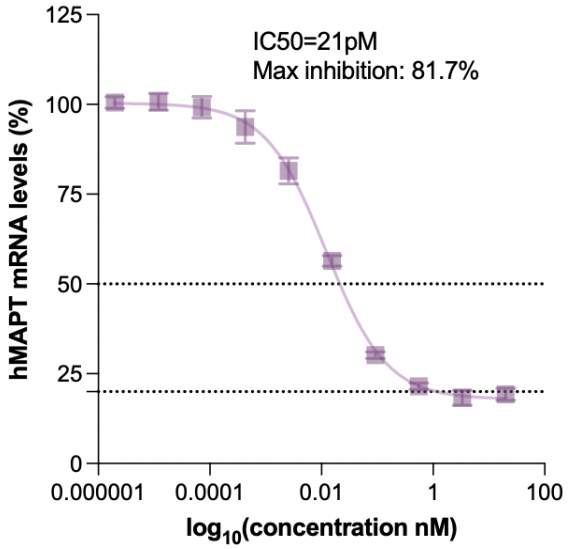
THE DELIVERABLES

2027/8: Aiming for first-in-human studies to confirm safety, exposure, and Tau reduction with SC dosing; followed by Phase 1b (AD) to assess Tau reduction (PET and soluble biomarkers) and safety.

**AL064 incorporates a well-validated chemical modification that enhances siRNA stability and is advancing into IND-enabling studies as AL164.*

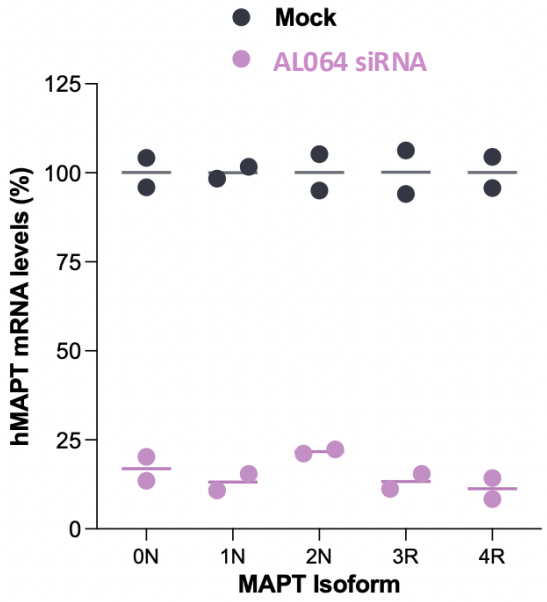
AL064 siRNA Demonstrates High Potency in Vitro

Dose-Dependent Knockdown in MAPT Expressing Cell Line



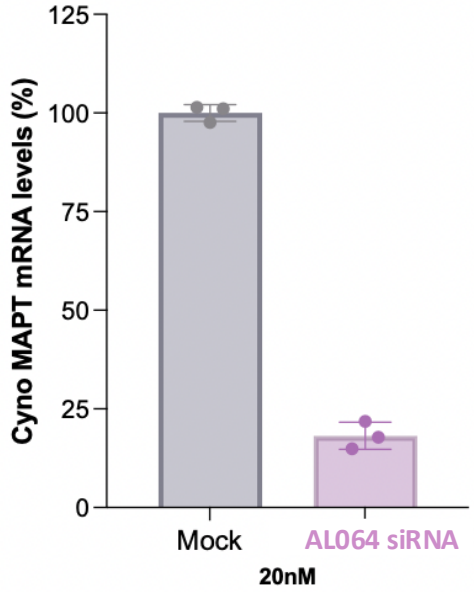
80% inhibition and pM range IC50. AL064 siRNA was transfected into MCF7 cells for 24hr to determine the dose response.

Knockdown all MAPT Isoforms in Human Neuroblastoma Cell Line



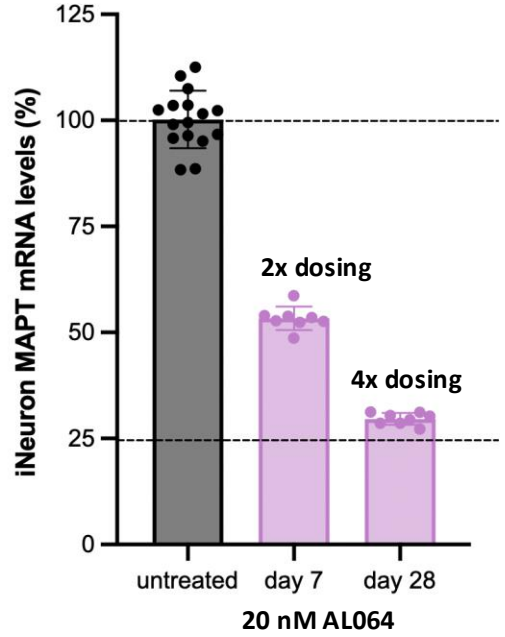
AL064 siRNA was transfected into SH-SY5Y cells for 24hr to determine the knockdown of MAPT isoforms.

Knockdown of MAPT in Cyno Primary Astrocytes



AL064 siRNA was transfected into cyno primary astrocytes cells for 48hr to determine the knockdown of MAPT mRNA.

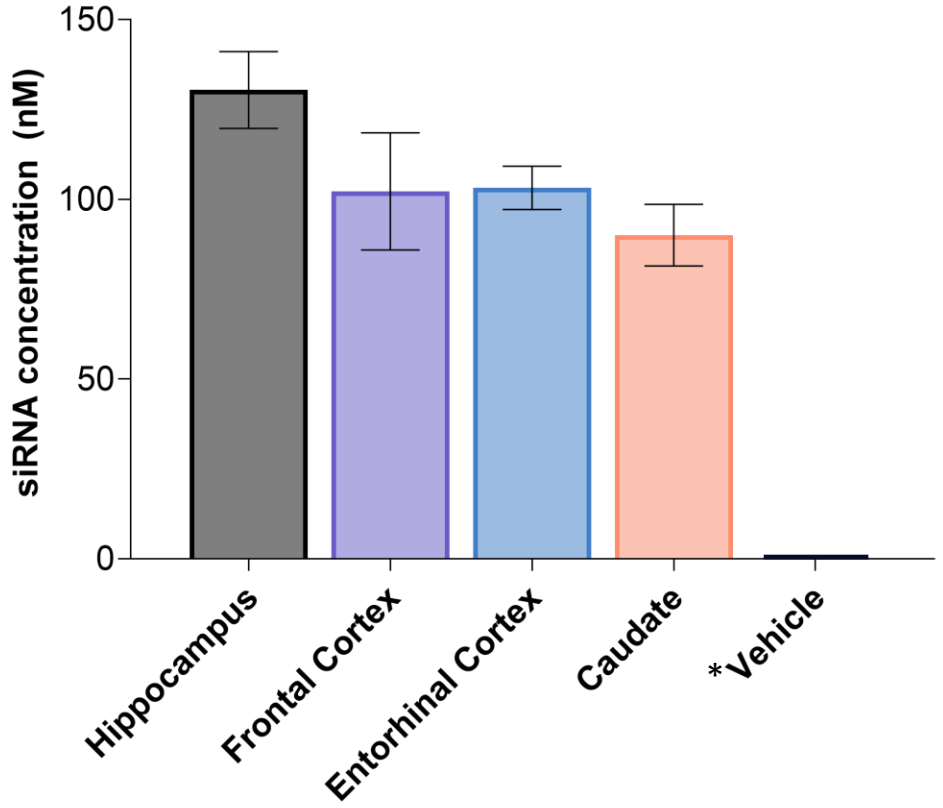
Knockdown of MAPT in iPSC Neurons by AL064



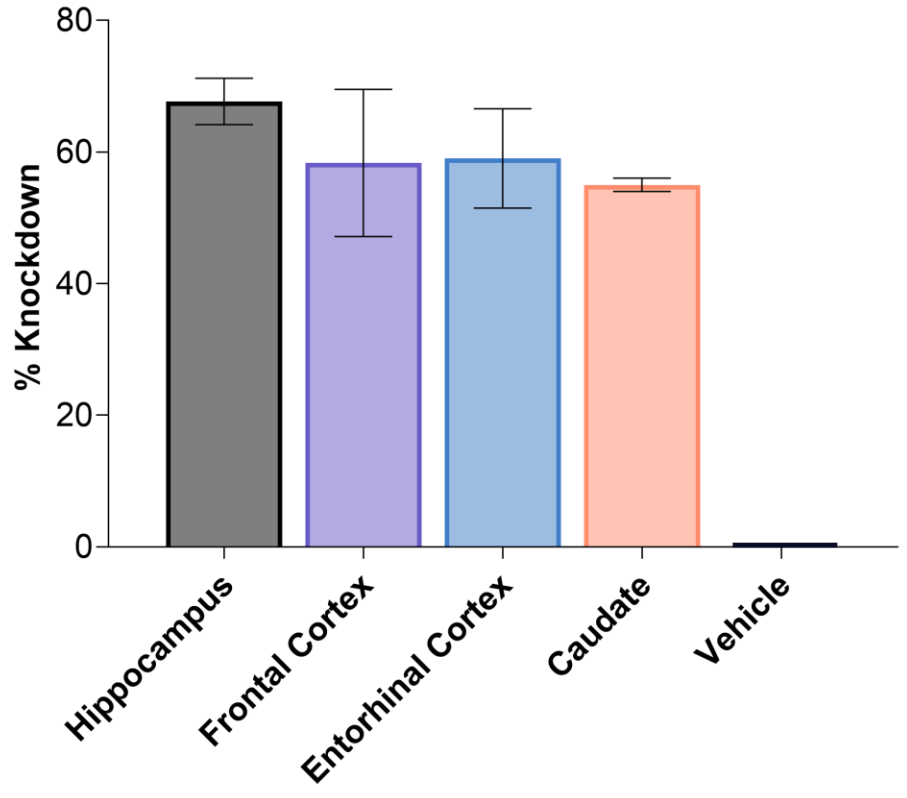
iPSC-derived neurons were treated with AL064 2 or 4 times and Tau mRNA knockdown was determined after 7 or 28 days.

Peripheral AL064: Up to ~130 nM in Brain and ~70% mRNA Knockdown in NHPs

Concentrations of AL064 in Brain Tissues (28 Days)



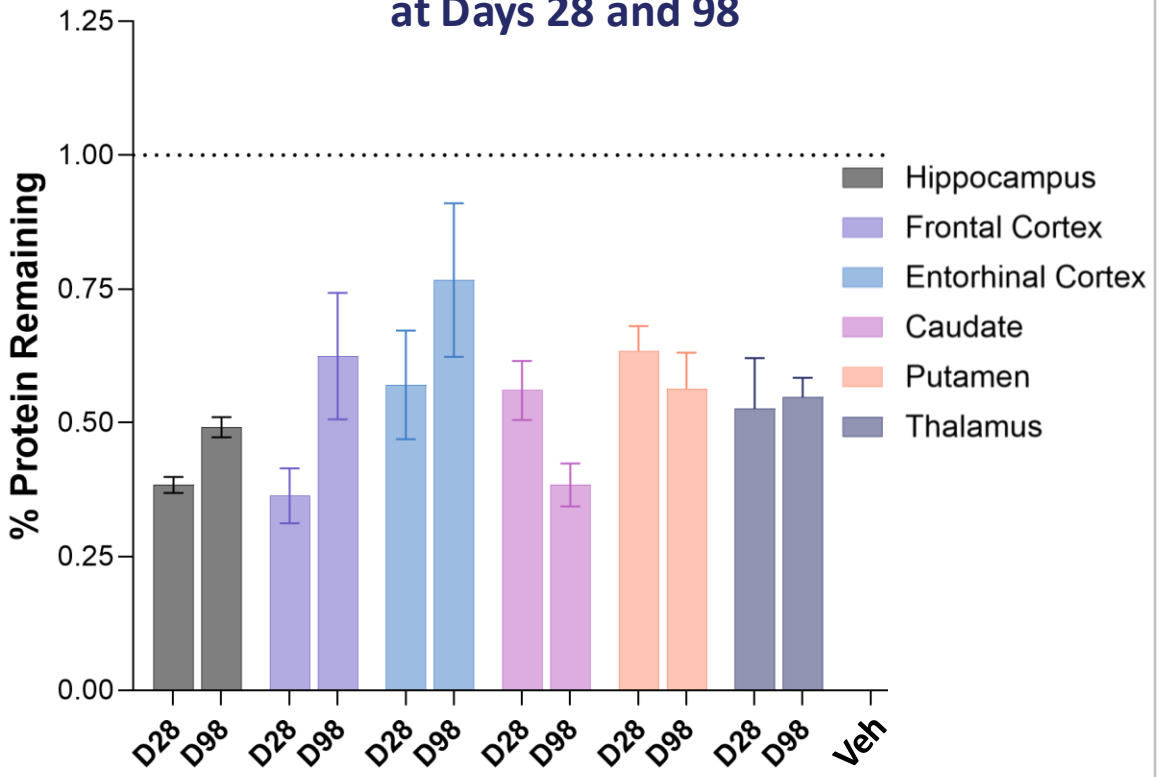
% Knockdown of Tau mRNA in Brain Tissues (28 Days)



*NHPs were dosed by IV infusion on days 1, 8, and 15 with AL064 at a siRNA dose-equivalent of 3 mg/kg. Levels of the AL064 antisense strand were measured across multiple brain regions on day 28 using an MSD assay. *Vehicle control was below assay sensitivity of 0.0156nM. MAPT mRNA levels in the same brain regions were also measured on day 28 using qRT-PCR.*

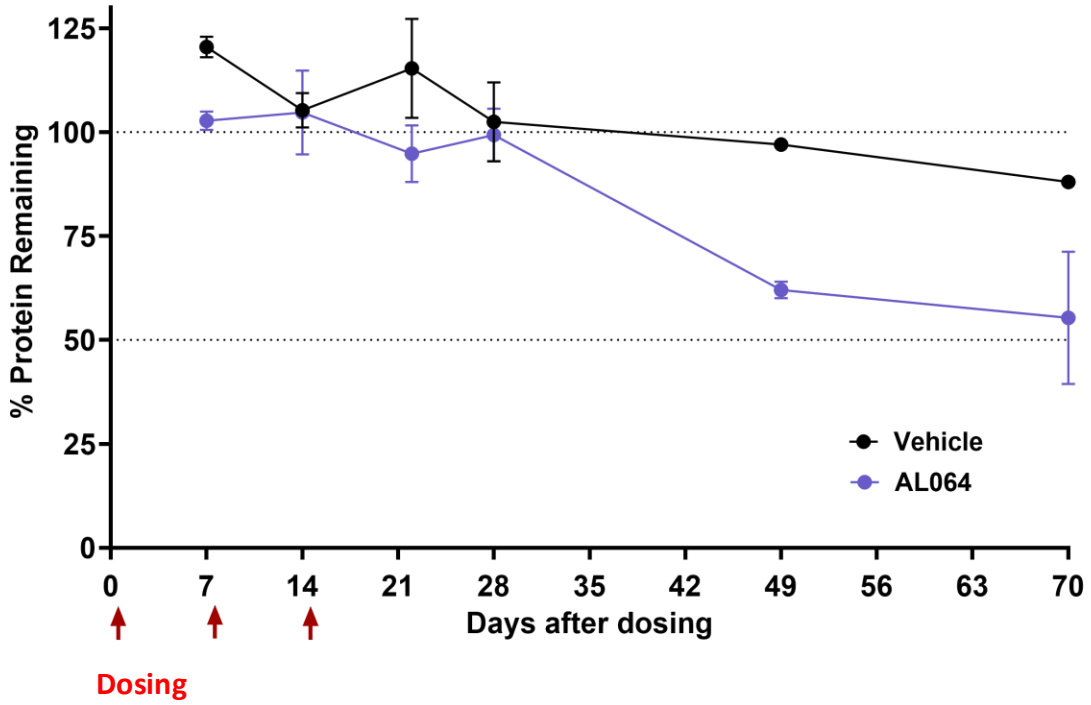
Peripheral AL064: Durable Reduction of Phospho-Tau 217 in NHP

Up to 60+% Reduction of pTau 217 in NHP Brain at Days 28 and 98



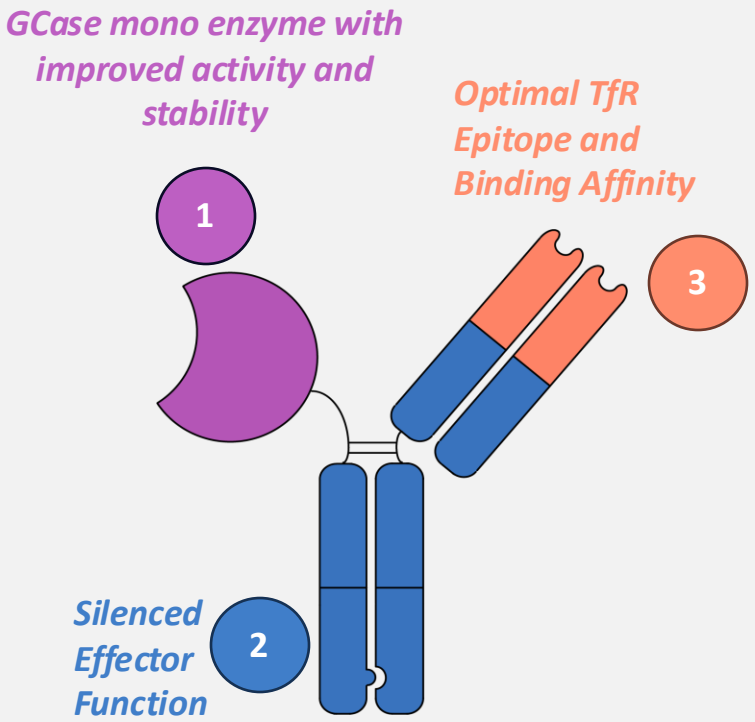
D28 and D98 normalized to experiment-specific vehicle treated cohort.

45% Reduction of pTau 217 in the CSF at Day 70



Three NHP per group were injected via IV infusion on days 1, 8, 15 with 3 mg/kg siRNA-dose-equivalent and the levels of p-Tau217 in different brain regions was measured at day 28 and 98 with MSD assay. pTau217 levels were measured in the CSF at the following timepoints: pre-dose, D7, 14, 22, 28, 49 and 70.

AL050: First-in-Class, Brain-Enabled, GCase Enzyme Replacement Therapy with Disease Modifying Potential



THE CHALLENGE

No disease-modifying therapies exist for PD/LBD; Progression is accelerated in GCase LOF and low-activity patients

THE AL050 SOLUTION

AL050: Brain-penetrant GCase enzyme replacement designed to restore activity and reduce toxic substrates, targeting GCase LOF PD/LBD and broader low-activity patients.

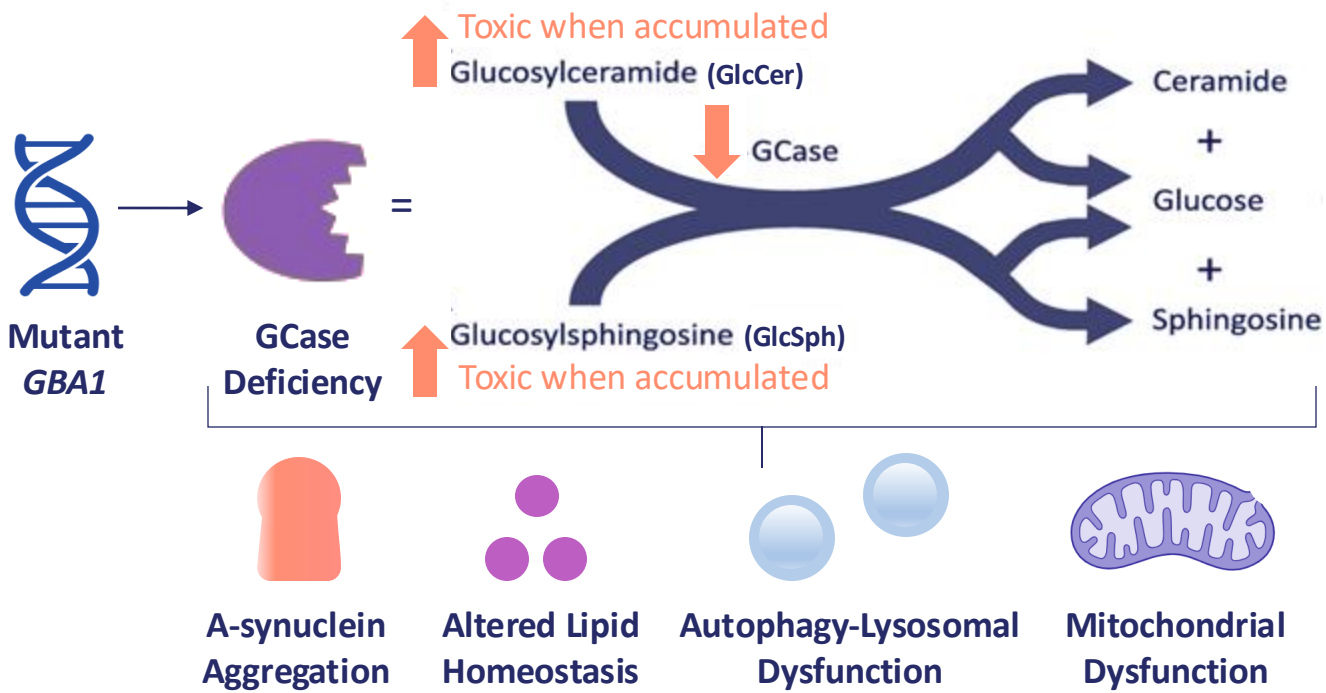
THE DELIVERABLES:

2027: First-in-human SAD for safety and CNS exposure
2028 Phase 1b (PD-GBA): Target engagement and GlcSph reduction

Mutations in *GBA1* (the Gene Encoding the GCase Enzyme) are a Major Risk Factor for Parkinson's Disease, Lewy Body Dementia and Gaucher's Disease

GBA1 mutations lead to reduced **GCase enzyme activity** and toxic substrate accumulation (GlcCer and GlcSph) in the brain.

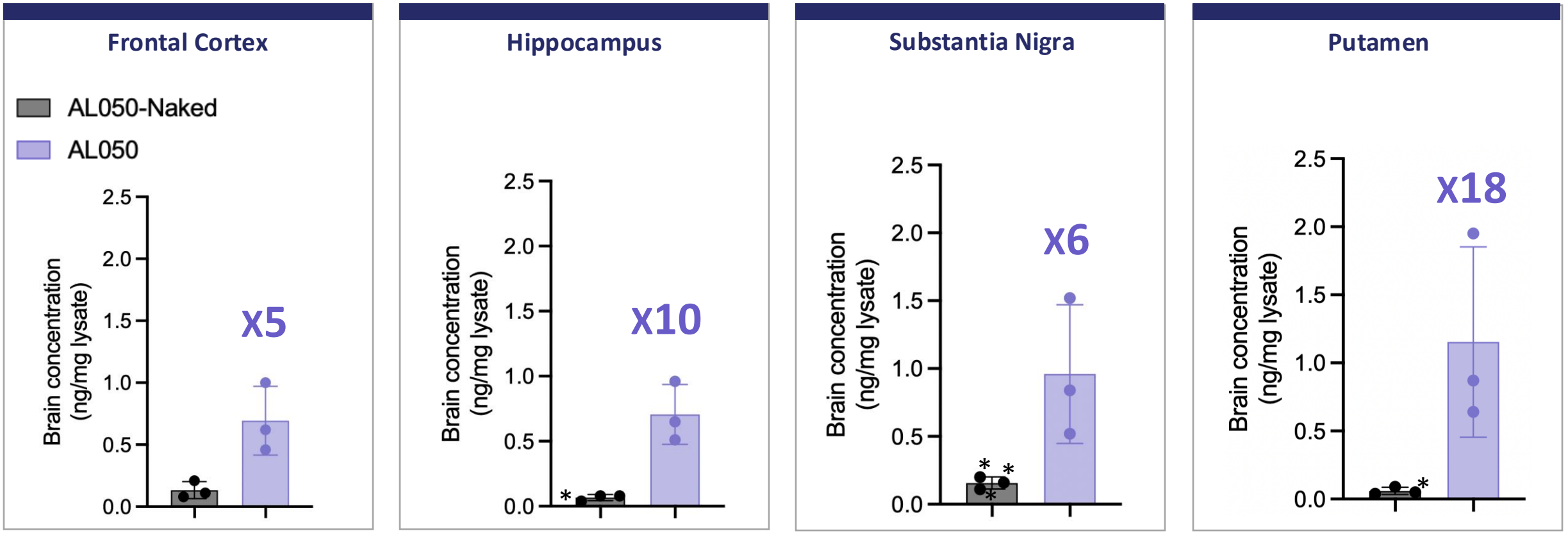
No therapy has effectively restored GCase activity in the brain: Current GCase enzyme replacement therapy does not enter the brain



- **Parkinson's Disease (PD)**
  - ~10 million patients worldwide[1]
  - 0.5-1.5 millions are GBA1 mutation carriers[2]
  - Activity is reduced in non-carriers[2]
- **Gaucher's Disease (GD)**
  - ~125,000 patients with GBA1 mutation worldwide[5]
  - GD type 1 have increased risk of PD[6]
  - GD type 2 and 3 are neuronopathic[7]
- **Lewy Body Dementia (LBD)**
  - ~5-8 million patients worldwide[3]
  - 0.15--2.4 millions are GBA1 mutation carriers[4]
  - Activity is reduced in non-carriers[4]

1. Parkinson's Foundation Statistics
2. Smith L, Schapira AHV. GBA Variants and Parkinson Disease: Mechanisms and Treatments. *Cells*. 2022 Apr 8;11(8):1261.
3. Alzheimer's Disease International, Dementia with Lewy Bodies
4. Nalls MA, et al. A multicenter study of glucocerebrosidase mutations in dementia with Lewy bodies. *JAMA Neurol*. 2013 Jun;70(6):727 - 35.
5. Meikle PJ, et al. Prevalence of lysosomal storage disorders. JAMA. 1999 Jan 20;281(3):249- 54.
6. Bultron G, et al. The risk of Parkinson's disease in type 1 Gaucher disease *J Inherit Metab Dis*. 2010 Apr;33(2):167 - 73.
7. National Gaucher Foundation, Gaucher Disease Types 2 and 3

ABC Enhances Brain Delivery of AL050 by 5- to 18-Fold, Over Naked AL050

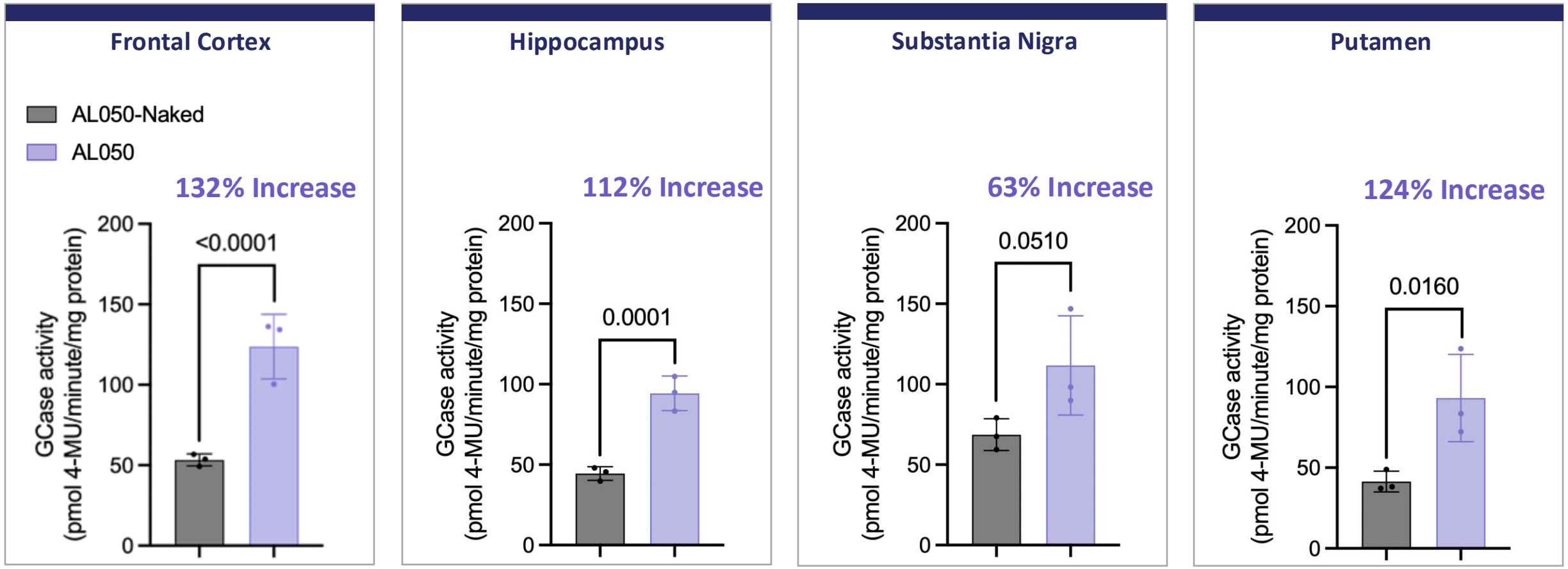


Three female cynomolgus monkeys per group were dosed intravenously on D1, D8, with 10mg/kg of AL050 and were sacrificed at D9. Brain tissues were collected 24 hours after the second injection and drug levels were measured in the vessel-depleted fraction. AL050-Naked = AL050 without ABC platform. Only AL050 but not endogenous GCase are being detected with this assay.

*Samples below lower limit of detection graphed at LLOD.

AL050 Increases Brain GCase Activity in NHP by >2 Fold Over Endogenous Levels

Would fully compensate for the less than 50% reduction in GCase activity in PD or LBD



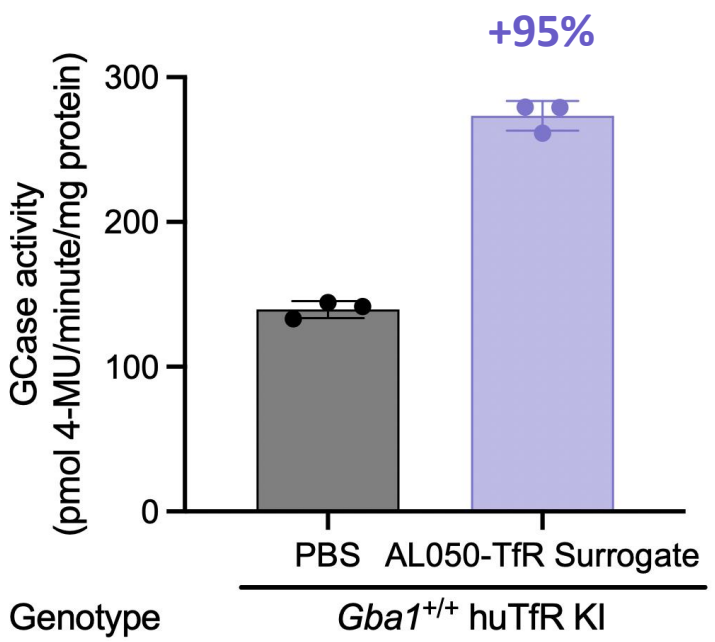
AL050 Was Well-Tolerated in NHPs at 10 mg/kg/week

Three NHPs per group were dosed intravenously on D1, 8, with 10mg/kg of AL050 and were sacrificed at D9. GCase enzymatic activity was determined using a 4-MUG kinetic assay (graphs represent the combination of endogenous NHP GCase and AL050. AL050-Naked = AL050 without ABC platform. The enzymatic activity of the Recombinant GCase appears stronger than that of the activity of the endogenous GCase as 27-39% increase in recombinant GCase leads to 63-175% increase in enzymatic activity.

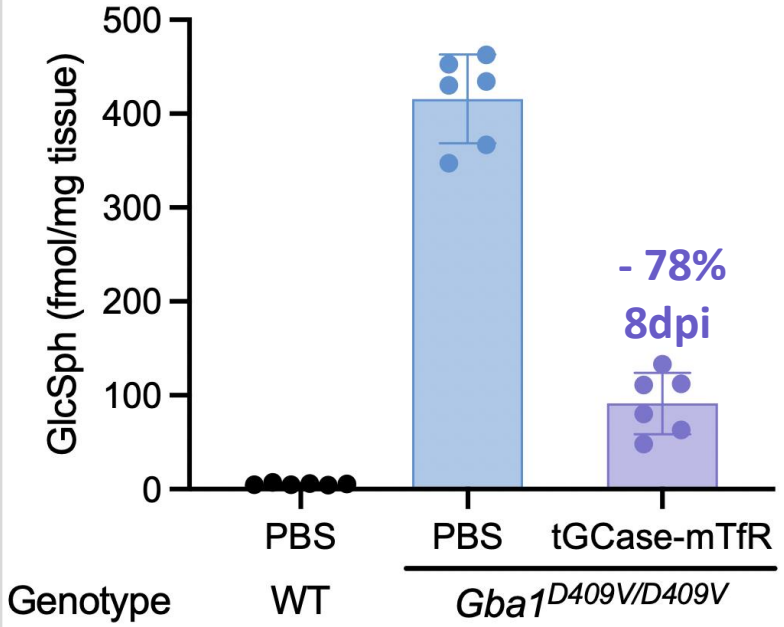
One way ANOVA with Dunnett's multiple comparison test

AL050 Surrogate Rescues Brain GCase Activity and Reduces Toxic Substrate Accumulation in the Brain of *Gba1*-Mutant Mice

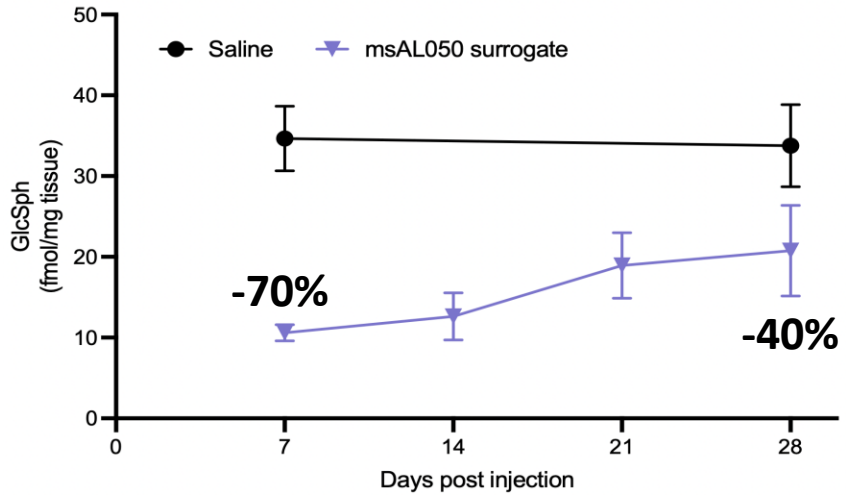
GCase-ABC Increases Brain GCase



GCase-ABC Reduces GlcSph



Durable (28D) Reduction in Brain GlcSph



Wild-type or Gba1-mutant mice were injected once (left, right) or twice (middle) with PBS, or 10mg/kg AL050-huTfR Surrogate (left), tool GCase-msTfR (middle), or AL050-msTfR Surrogate (right). GCase activity (left) was determined by 4-MUG kinetic assay in vessel-depleted brain lysates. Brain GlcSph concentration (middle, right) was determined by LC-MS/MS.

Value Creation Benchmarks

Pre-clinical

TfR Brain Shuttle Aβ mAb
$165M upfront, $1.5B milestones; royalties

TfR Brain Shuttle Aβ mAb
$100M upfront, $1.25B milestones; royalties

TfR Brain Shuttle siRNA & preclinical targets (4 clinical, 3 pre-clinical, 6 TBD)
$825M upfront, ~$8B milestones; royalties

TfR Brain Shuttle Tau, Trem2
$150M upfront, $1.02B milestones; Profit sharing, co-commercialization

Phase 1a

TfR Brain Shuttle Synuclein siRNA
$200M upfront, $2B milestones; Tiered royalties

TfR brain Shuttle Aβ mAb
$1.4B M&A

LRRK2 small molecule & 2 preclinical
$560M upfront, $465M equity, $1.125B milestones; 40/60 profit share

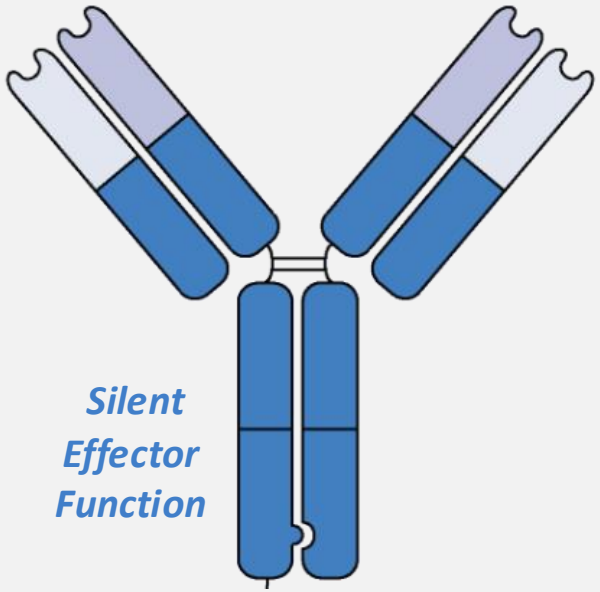
Phase 2

TfR-conjugated siRNA for peripheral delivery
$12B M&A

$700M upfront; $1.5B milestones; $160M U.S. launch; $90M ex-U.S.; 50/50 U.S. profit share; double-digit royalites ex-U.S.

Nivisnebart: Novel Disease-Modifying, PGRN-Elevating, Strategy for AD

Sortilin Blocking Domains
Prevent Sortilin mediated degradation of PGRN



THE CHALLENGE

Current therapies target Aβ or Tau, leaving progranulin-driven lysosomal and neuronal survival pathways unaddressed

THE NIVISNEBART SOLUTION

A Phase 2 AD therapy designed to **elevate progranulin and enhance lysosomal function and neuronal survival, with a favorable safety profile** and potential as a standalone or combination disease-modifying therapy

THE DELIVERABLES:

2026: Phase 2 AD Futility Analysis 1H26; Trial completion
2027/8: Potentially initiate Ph 3 pivotal study in early AD with partner, GSK (50/50 Profit share US)

Platform + Multi-Asset Optionality, Create Asymmetric Strategic Value*

Reproducing the ABC platform would require ~5–7 years, creating a strategic moat

Comparable BBB platforms acquired or encumbered — **future access at premium**

Delay **risks competitors defining next-gen** CNS standards

AL037/AL137 (ABC Anti-Aβ) Brain-enabled anti-Aβ; Large AD market; Scarce strategic asset. Targeting FIH 2027

AL050 (ABC GCase ERT): Brain-penetrant GCase; validated biology; high optionality. Targeting FIH 2027

ABC siRNA (Tau & α-Syn) Large unmet need; disruptive vs. non-BBB approaches. Targeting FIH 2027/8

Fully Owned Platform and Pipeline;
Rare Opportunity for Transformative Pharma Partnership or Independent Growth

alector

**Dates and statements represent forward-Looking statements associated with substantial risks and uncertainties listed in slide 2.*

Thank You