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Korsana Biosciences Overview

September 2026

Nasdaq: KRSA

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Korsana is developing potentially best-in-class therapies, with an initial focus on neurodegenerative disorders

- Initially focused on **potentially best-in-class therapies** for neurodegenerative disorders like Alzheimer's disease
- KRSA-028 incorporates **Therapeutic Targeting (THETA™)**, a next-generation BBB-penetrant shuttle platform
- Committed to **move fast** and develop a pipeline with **long-term defensibility**

Program	Indication	Stage	Mechanism of Action
KRSA-028	*Alzheimer's disease*	*IND-enabling*	**Anti-3pE amyloid beta mAb** (THETA-enabled)
Undisclosed	*Undisclosed*	*Discovery*	**Undisclosed**
Undisclosed	*Undisclosed*	*Discovery*	**Undisclosed**

Strong financial position with ~$475M[1] cash expected to fund operations into 2029

[1]Pro-forma cash a/o 6/30 including PIPE, net of projected deal costs. Notes: 3pE-Aβ: Amyloid beta with post-translational pyroglutamate modification (pE) at the 3rd amino acid position, which is enriched in AD amyloid plaques. BBB: Blood-brain barrier

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Korsana is founded on four key beliefs

Alzheimer's is a vast and de-risked opportunity

*For the first time, there is a **validated, disease-modifying target for Alzheimer's** – but first-generation amyloid beta therapies leave **substantial room for improvement**.*

Shuttling is the best way to improve existing agents

*Transferrin receptor (TfR)-based shuttling is a de-risked modality to **increase brain penetration**; Roche's **trontinemab has provided proof-of-principle** in Alzheimer's disease.*

Korsana has the potential best-in-class approach

*Lead program KRSA-028 is potentially **superior to trontinemab,** and we are **advancing multiple next-generation programs**.*

Korsana has a rapid path to value creation

*KRSA-028 development can be **highly de-risked in Phase 1,** as amyloid clearance is proven to translate to clinical benefit – creating **significant early value inflection**.*

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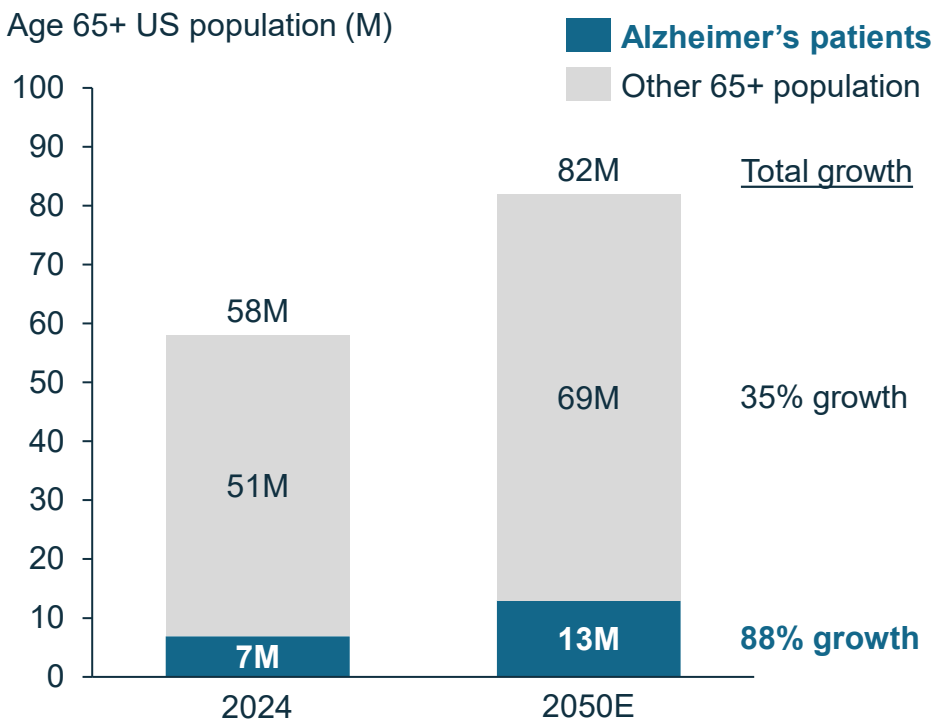
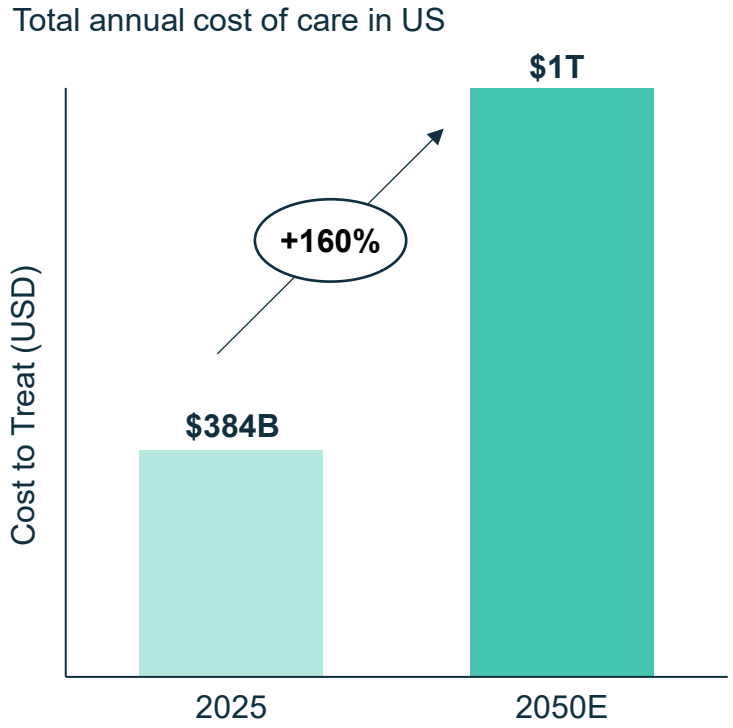
Alzheimer’s is a vast and de-risked opportunity

Alzheimer’s is a devastating neurological disorder with significant unmet need and a rapidly growing patient population

The number of Americans with Alzheimer’s is **projected to nearly double** by 2050





Alzheimer’s disease is a **massive and growing unmet need**,
with associated long-term healthcare costs projected to be **>$1T by 2050**.

Sources: 2024 Alzheimer’s Association (Alzheimer’s Dement); 2025 Alzheimer’s Association (Alzheimer’s Dement).
Cost of care includes CMS payments, out of pocket costs, and other costs for health care and long-term care for people with Alzheimer’s or other dementias.

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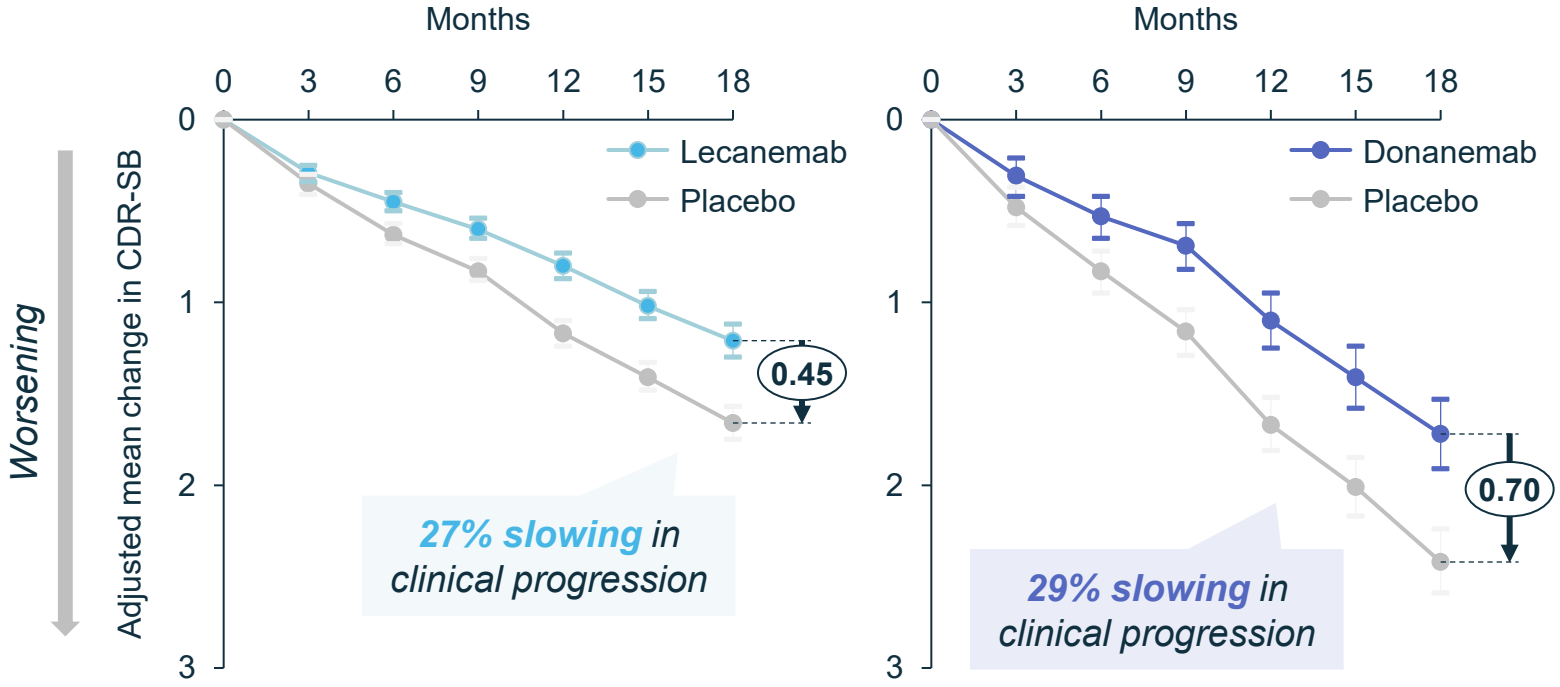
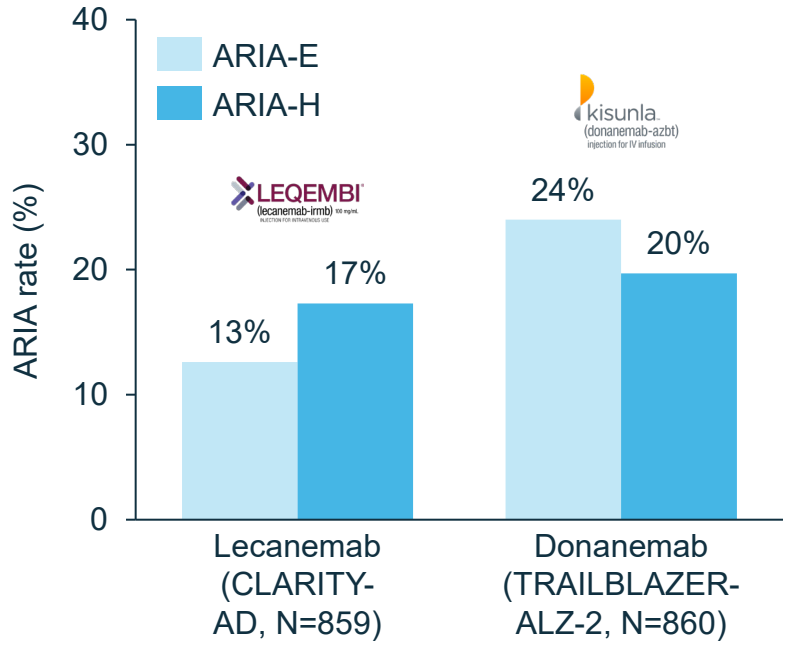
Despite clinical progress and approvals, today's anti-Aβ therapies leave significant room for improvement

Approved therapies only demonstrate **~30% slowing of disease progression** at 18 months…



…and carry **black box warnings for ARIA risk,** affecting **~15-25%** of treated patients



Although these therapies are disease-modifying, patients **still experience progression**, with potential for **new therapies to deliver superior efficacy and safety**.

Notes: CDR-SB: Clinical Dementia Rating – Sum of Boxes, the functional Phase 3 endpoint for Alzheimer's trials. Data from some time points was digitized from original publications. Donanemab data based on mixed models for repeated measures (MMRM). ARIA: amyloid-related imaging abnormality; ARIA-E: edema and effusion; ARIA-H: microhemorrhage.
Sources: 2023 Sims (JAMA); 2023 van Dyck (NEJM)

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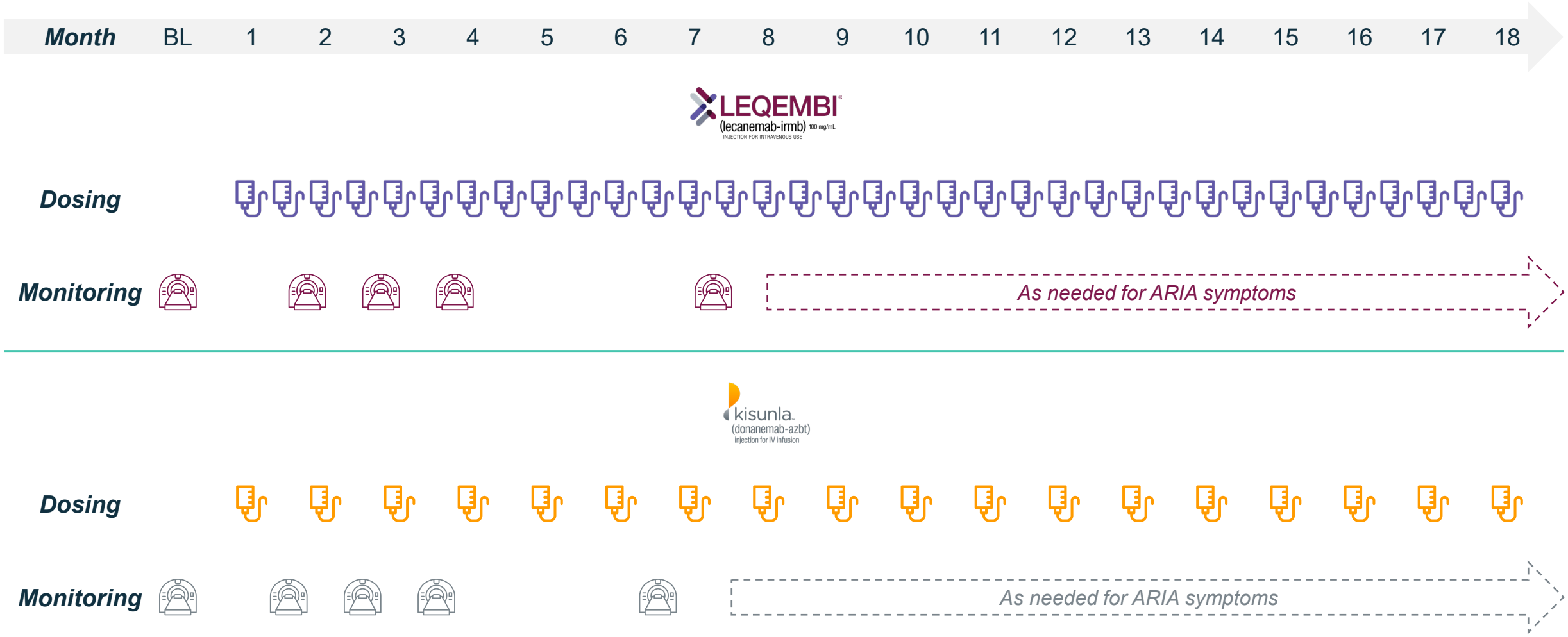
Approved therapies are also highly inconvenient, with onerous IV dosing and MRI monitoring on label



Notes: BL: baseline. Patients can be taken off Kisunla if they are “amyloid negative” at physician’s discretion.
Sources: Leqembi FDA Label; Kisunla FDA Label

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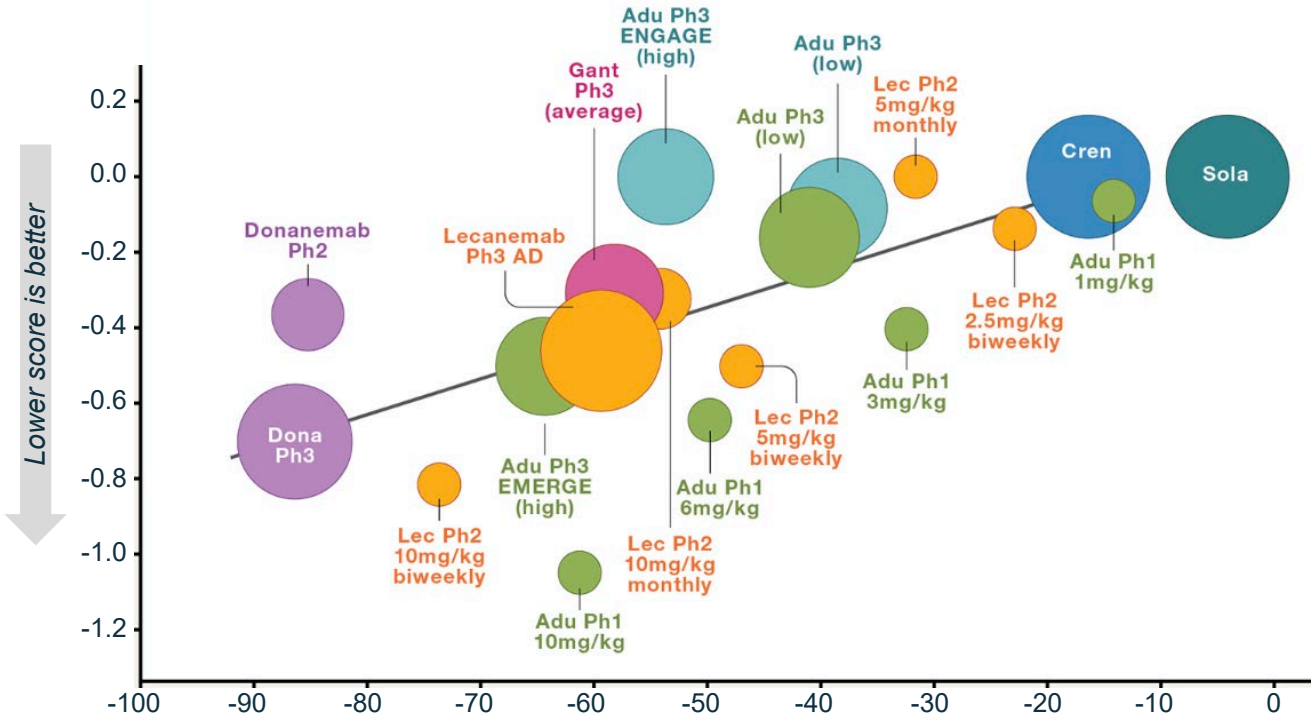
First-gen therapies have laid out the roadmap for success: amyloid reduction predicts slowing of cognitive decline

Cognitive Decline
CDR-SB adjusted mean difference from placebo



Plaque Clearance
Amyloid PET adjusted mean difference from placebo (centiloids)
Greater reduction is better

*"The Agency has found… that **reduction of brain Aβ plaque on PET is reasonably likely to predict clinical benefit in Alzheimer's disease.**"*
– Donanemab FDA Review

*"It is reasonable to conclude that **treatment that is targeted at reducing amyloid plaque**, and that successfully accomplishes that reduction, **has the potential to convey clinical benefit.**"*
– Lecanemab FDA Review

The field now has a **well-trodden path to regulatory success** for anti-amyloid beta therapies, with risk **discharged early in clinical development** with a validated biomarker.

Notes: Bubble size indicates sample size at baseline. CDR-SB: Clinical Dementia Rating Scale-Sum of Boxes, the functional Phase 3 endpoint for Alzheimer's trials. CDR-SB is a dementia severity scale ranging from 0 to 18 with higher scores indicating greater impairment.
Sources: 2023 Boxer (Cell).

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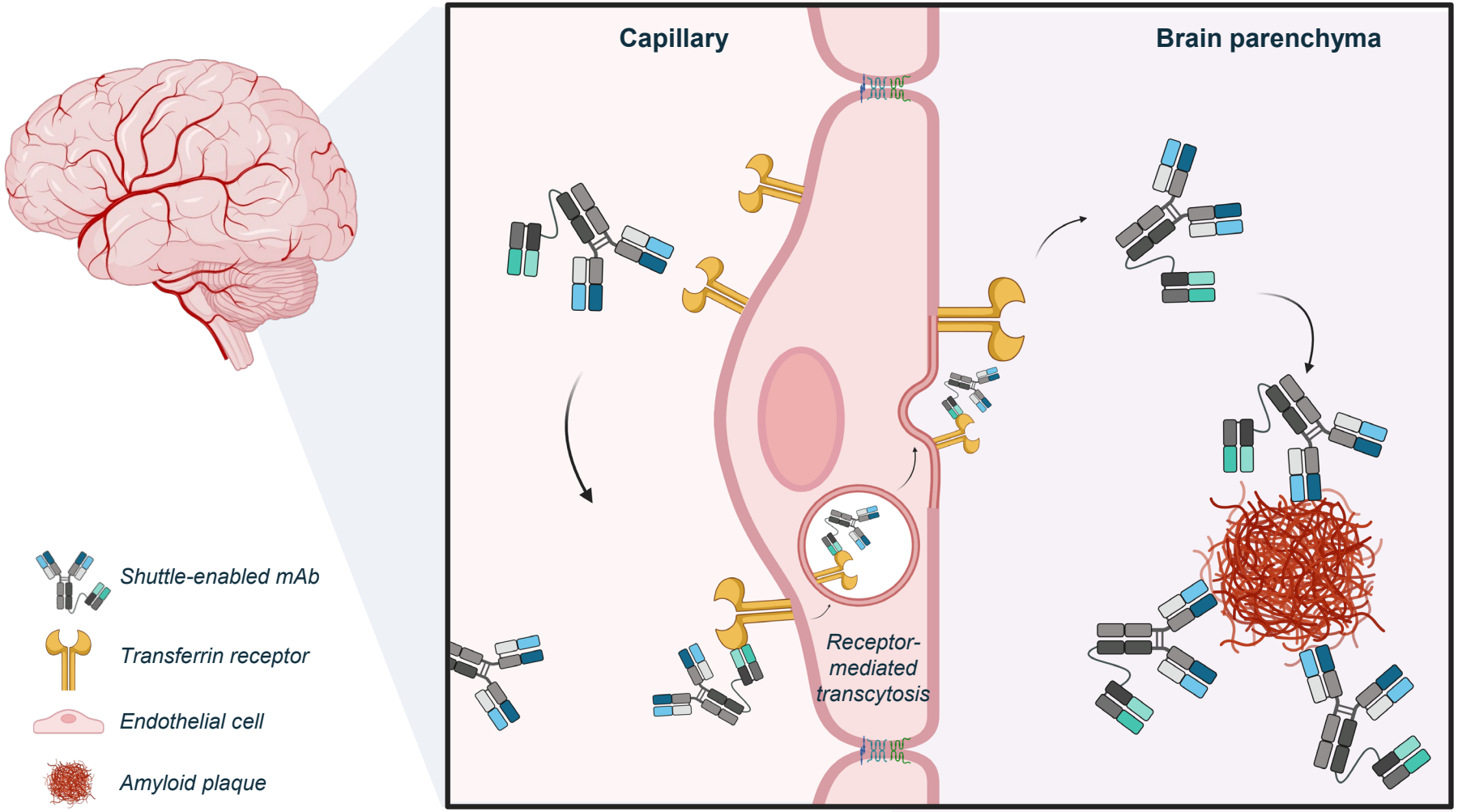
Shuttling is the best way to improve existing agents

Shuttling approaches actively ferry large molecules across the blood-brain barrier (BBB), fundamentally changing CNS penetration



Notes: Figure made in BioRender. Adapted from 2024 Kulic (CTAD Presentation); 2022 Edavettal (Med).

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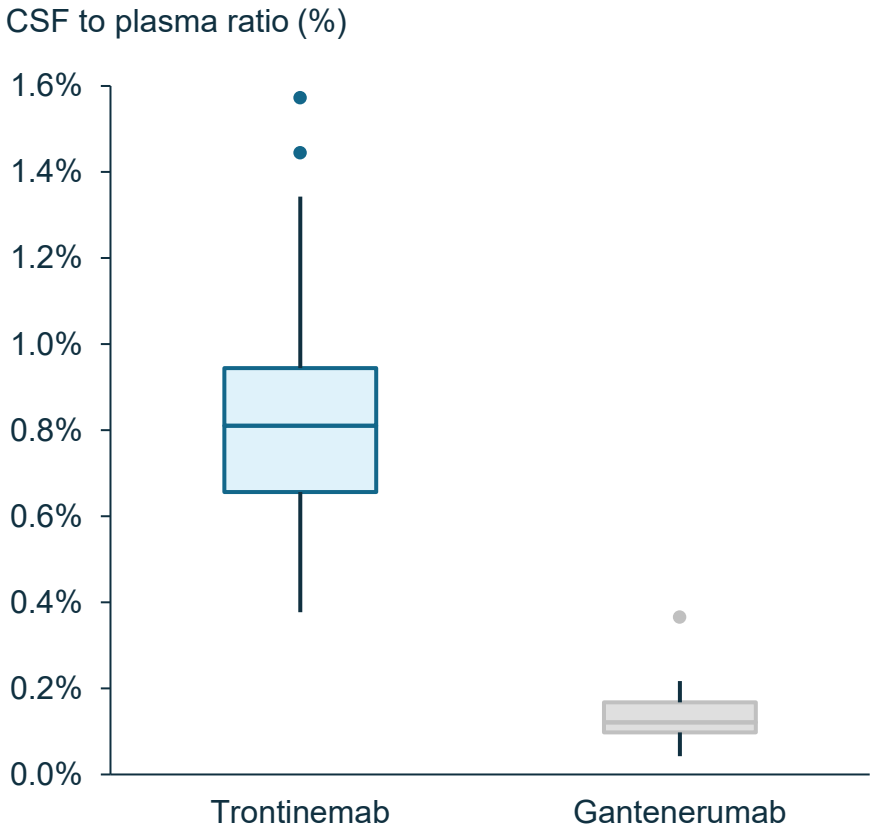
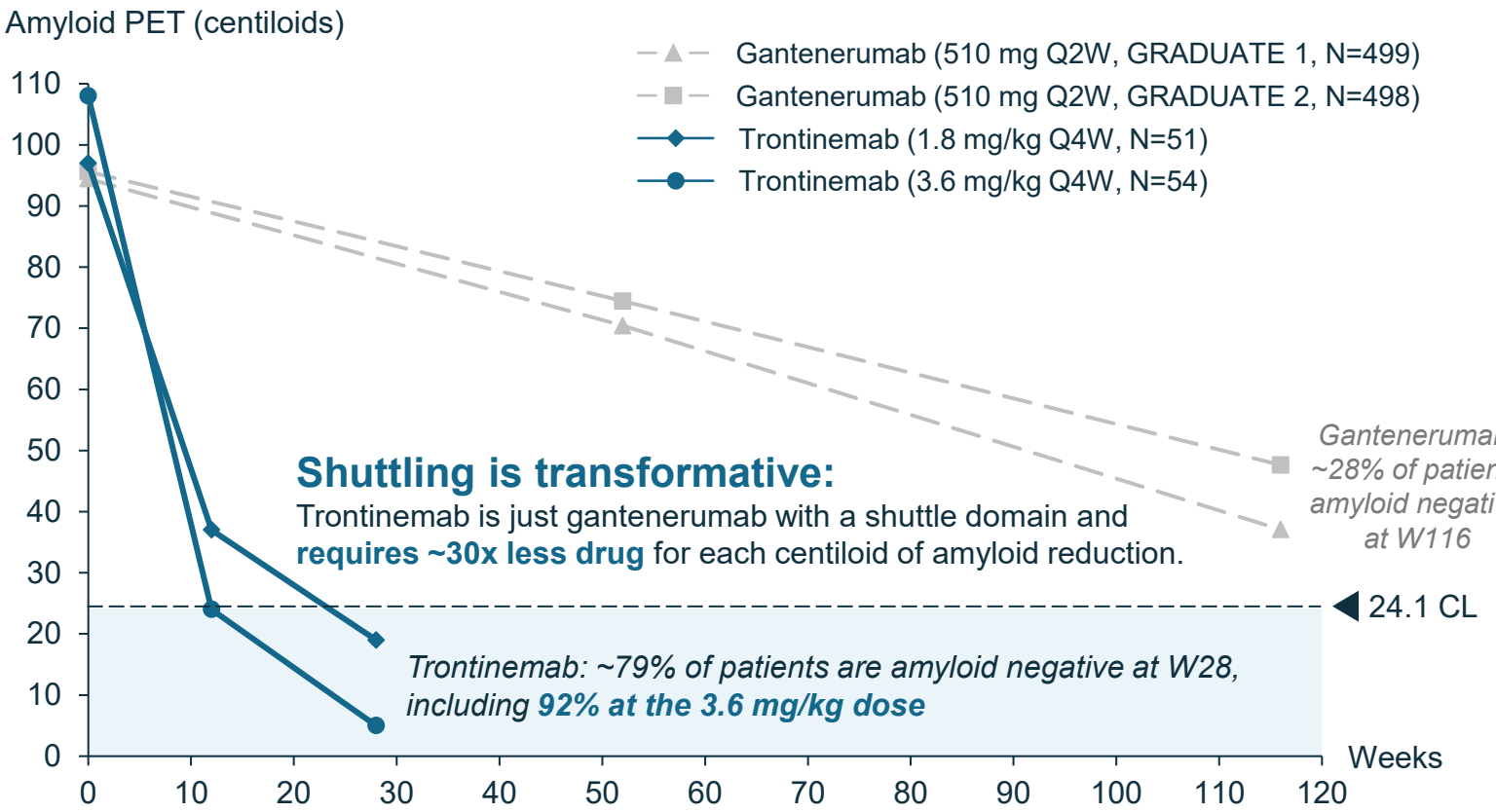
Roche's trontinemab, the first shuttled anti-Aβ antibody, has shown a significant efficacy improvement over first-gen gantenerumab…

Trontinemab shows **~8x increased CSF exposure** over gantenerumab in humans…



… and most trontinemab-treated Alzheimer's patients **reached "amyloid-negative" status** by W28



Notes: CSF: cerebrospinal fluid. Roche CSF data digitized from AD/PD presentation and represents Roche's own cross-trial comparison, with trontinemab CSF to plasma ratio from single-dose IV study compared to historical data from a prior gantenerumab SAD trial. Amyloid reduction is a cross-trial comparison. Gantenerumab dosing titrates up to 510 mg Q2W maintenance dosing. 0 CL anchored on "high certainty" young, healthy controls & 100 CL anchored on typical AD patients. A threshold of 24.1 CL discriminates sparse from moderate plaque presence and is generally viewed as the cutoff for classifying patients as "amyloid negative." Sources: 2021 Kulic (AD/PD Presentation); 2023 Bateman (NEJM); 2025 Kulic (CTAD Presentation), 2025 Klein (AAIC Presentation).

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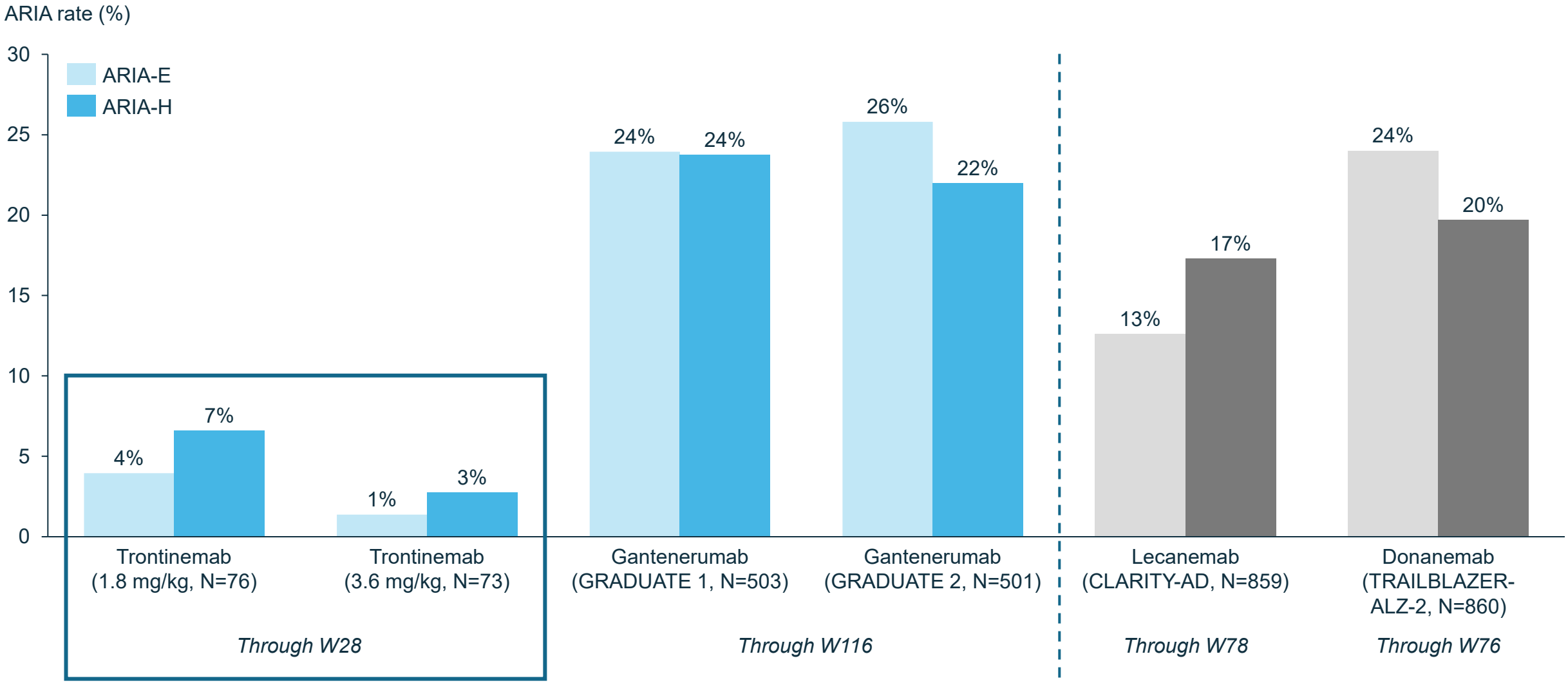
… and greatly reduces ARIA, the critical safety signal for this class of therapies



Notes: Comparisons are across trials with different patient populations and trial designs, including study duration. No head-head-to-head comparison studies have been conducted. Trontinemab values include placebo patients (patients were randomized 4:1); N otherwise refers to patients on drug. ARIA: amyloid-related imaging abnormality; ARIA-E: edema and effusion; ARIA-H: microhemorrhage.
Sources: 2023 Bateman (NEJM); 2022 van Dyck (NEJM); 2023 Sims (JAMA) 2025 Kulic (AAIC Presentation)

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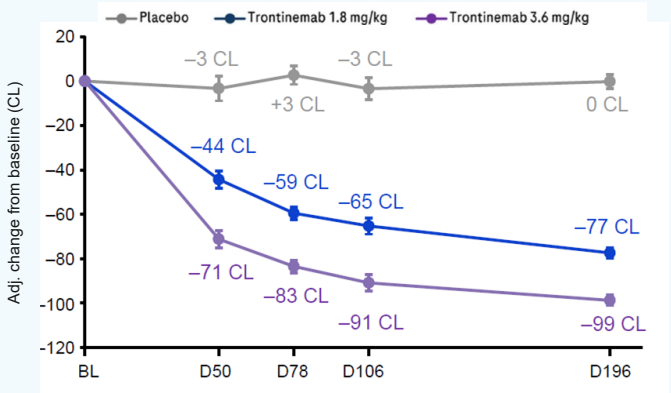
However, as a first-gen shuttled Aβ, trontinemab's profile leaves substantial headroom for competitive differentiation

Efficacy

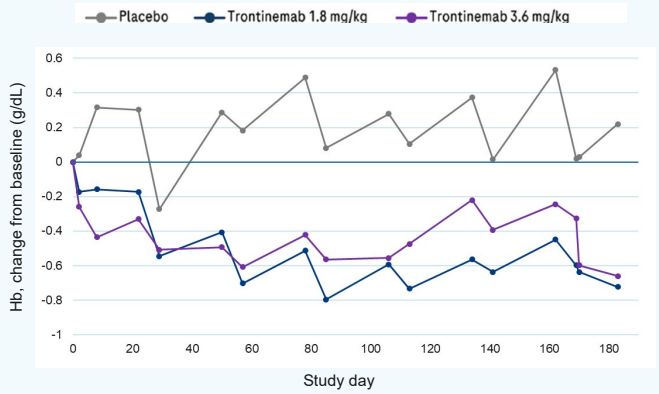
Mean amyloid plaque reductions from baseline



No evidence that maximum efficacy was reached in Phase 2 dose-response; **further room to improve on trontinemab efficacy** [1]

Safety

Mean hemoglobin changes from baseline



Trontinemab's full effector function leads to reticulocyte destruction, with **decreased hemoglobin** and **10-20% rates of clinical anemia** observed in Phase 2 [1]

Dosing & Tolerability

Incidence of infusion related reactions

Treatment emergent AEs: infusion-related reactions (IRRs)

IRRs are common and generally mild-to-moderate in severity

1.8 mg/kg or placebo	**50%** N=38 of 76
3.6 mg/kg or placebo	**44%** N=33 of 75

Trontinemab requires **IV dosing** and is associated with a **high rate of infusion-related reactions,** even with steroid pre-medication [2]

Sources: [1] Adapted from 2025 Kulic (CTAD, AAIC presentations). [2] Adapted from 2025 Kulic (CTAD Presentation). Parts 1 and 2 of study, pooled with placebo (4:1 randomization). Steroid pre-medication for all doses was implemented starting in Part 2 of the study. Per Kulic 2025 AAIC slides, steroid pre-medication was used in a majority of infusions. Roche has presented data showing most infusions did not lead to IRR, but table above shows the percent of patients in Part 1 + 2 who experienced an IRR at some point during the study.

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Korsana has the potential best-in-class approach

Korsana's goal is to achieve a best-in-class shuttled Aβ therapy, offering meaningful improvements over trontinemab

Key Value Drivers for KRSA-028

FAST, ROBUST AMYLOID REDUCTIONS	**Efficacy on par or greater than trontinemab**
DIFFERENTIATED SAFETY PROFILE	**Minimal ARIA risk, avoid hematologic AEs**
CONVENIENT, PATIENT-FRIENDLY DOSING	**Low-volume subcutaneous autoinjector for infrequent dosing (Q4W or less)**

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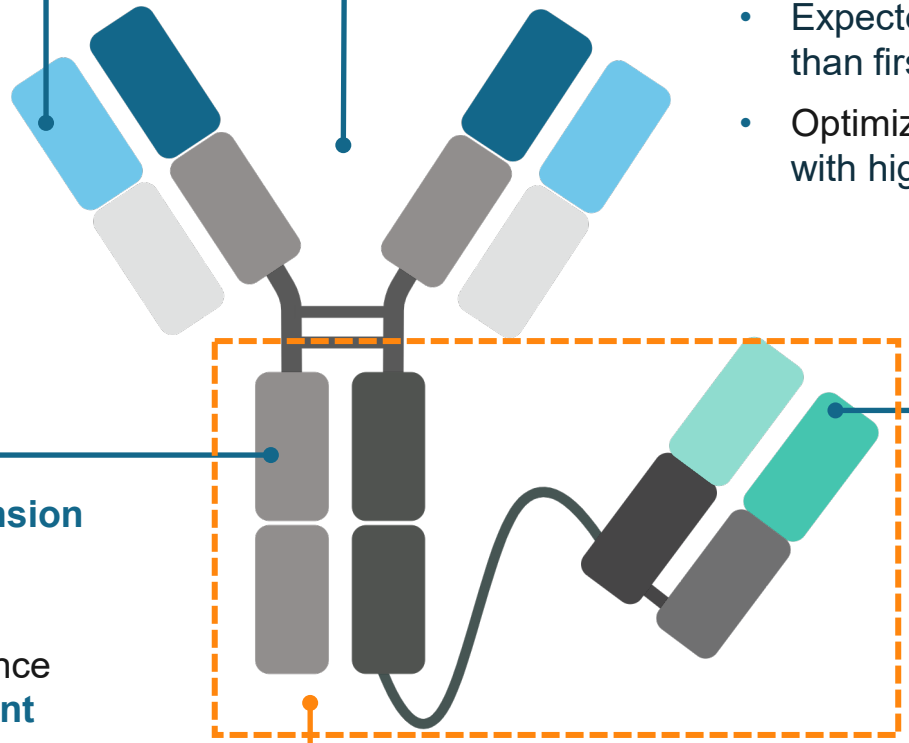
KRSA-028 is a next-gen, potentially best-in-class shuttled anti-Aβ

Pyroglutamate-Aβ targeted backbone

- Targets the Aβ epitope associated with the **greatest amyloid plaque clearance** in clinical studies
- **Preferential, high-affinity binding to amyloid plaques**

Proprietary Fc engineering

- Leverages clinically validated **half-life extension** to reduce dosing frequency
- Selective **effector function modulation** designed to maintain amyloid plaque clearance by phagocytosis while **reducing complement activation and risk of anemia**



Subcutaneous formulation

- Expected to be **efficacious at lower dose** than first-generation shuttled therapies
- Optimized for **low volume monthly SC dosing,** with high concentration and low viscosity

Validated shuttle targeting

- Novel sequence leverages proven TfR1 target and epitope, designed to **improve brain penetration** and **reduce ARIA risk**

Enabled by **Therapeutic Targeting (THETA™)**

- Precision-engineered for **optimized half-life, distribution, and effector function**
- Proprietary combination of clinically validated technologies offers **de-risked differentiation**

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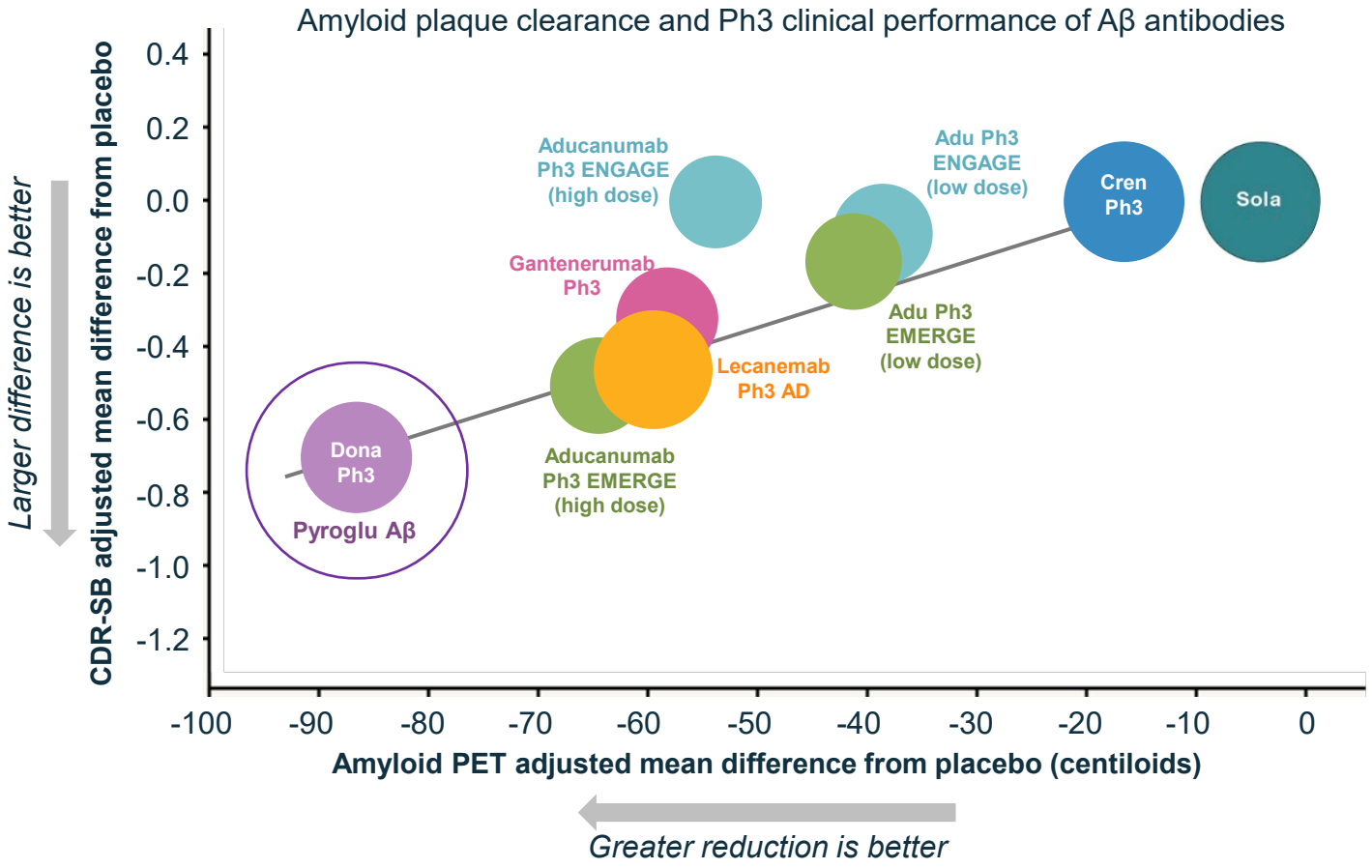
Targeting the plaque-specific pyroglu-Aβ epitope has been shown to deliver the most promising clinical efficacy in Phase 3 trials



Antibody	*Aβ epitope / species targeted*
Donanemab	**3pE (pyroglutamate) / plaques**
Aducanumab	Oligomers, protofibrils, fibrils, plaques
Lecanemab	Oligomers, protofibrils, fibrils
Gantenerumab	Oligomers, protofibrils, fibrils, plaques
Crenezumab	Monomers, oligomers, fibrils, plaques *Fc-null – no phagocytosis*
Solanezumab	Primarily monomers

Note: Remternetug (not shown) is Lilly's follow-on program to donanemab and also targets pyroglu-Aβ epitope; Phase 3 trial is ongoing

Notes: Bubble size indicates sample size at baseline. Image adapted from 2023 Boxer (Cell).
Other sources: KISUNLA FDA review; 2012 DeMattos (Neuron); ADUHELM FDA review; LEQEMBI FDA Review; 2023 Soderberg (Neurotherapeutics); 2016 Ultsch (Sci Rep); 2023 Sperling (New Engl J Med); 2024 Soderberg (Sci Reports); company press releases.

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KRSA-028 targets plaque-selective pyroglu-Aβ to maximize plaque clearance through phagocytosis

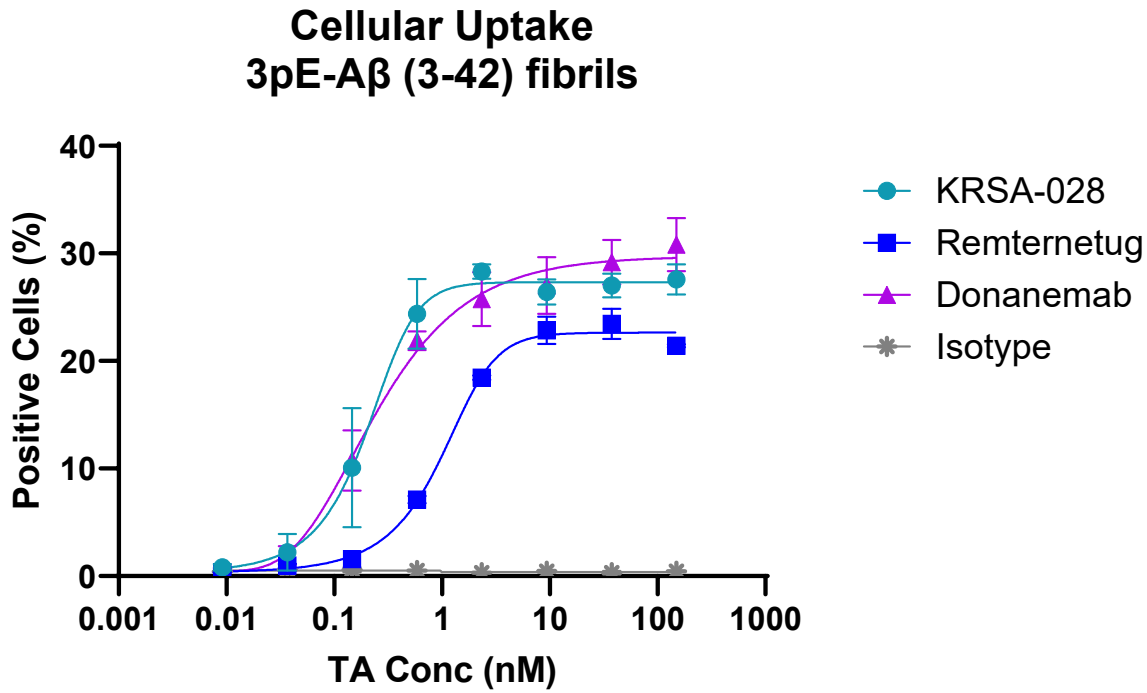
KRSA-028 Aβ backbone:

- Same **plaque-selective epitope** as donanemab and remternetug
- **High Aβ affinity,** K_D = 17 nM
- **Potent phagocytosis (ADCP),** EC_{50} = 0.21 nM

Donanemab *(Kisunla) is the only approved Alzheimer's treatment that targets 3pE-Aβ, but it features high immunogenicity and ARIA.*

Remternetug *is Lilly's follow-on program to donanemab with reduced immunogenicity, currently in Ph3 as a subcutaneous treatment.*

KRSA-028 exhibits potent pyroglu-Aβ phagocytosis



Representative plot shows mean ± SD, n=3 replicates. 20 µg/mL pHrodo-red labeled 3pE-Aβ fibrils incubated 24 hrs with activated monocytes (U937 cells)

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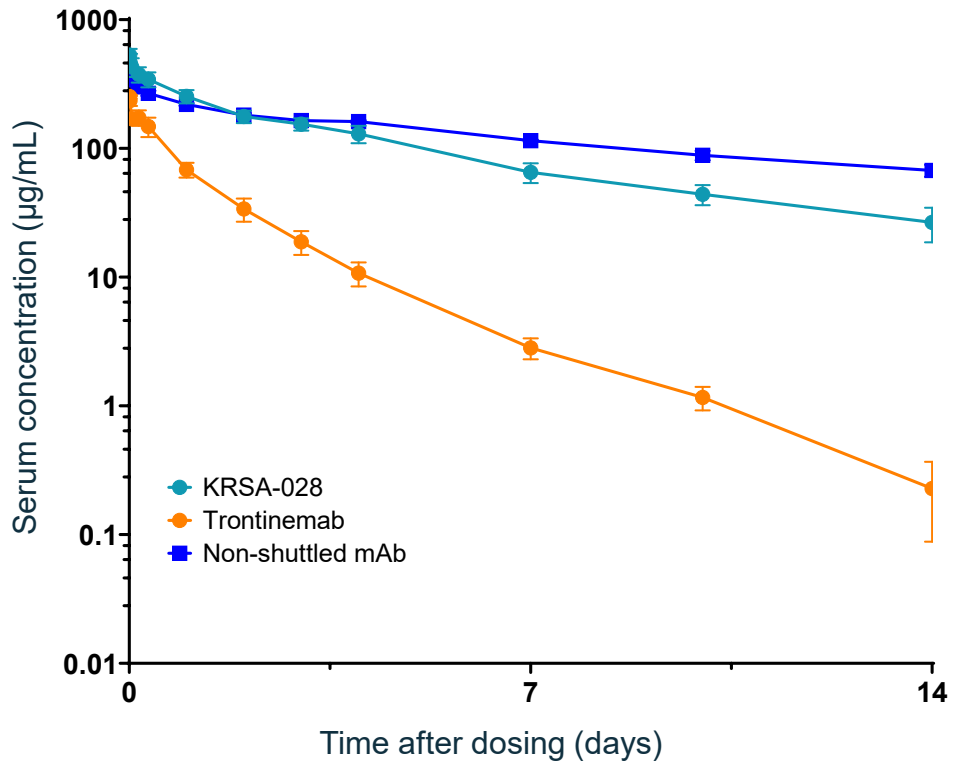
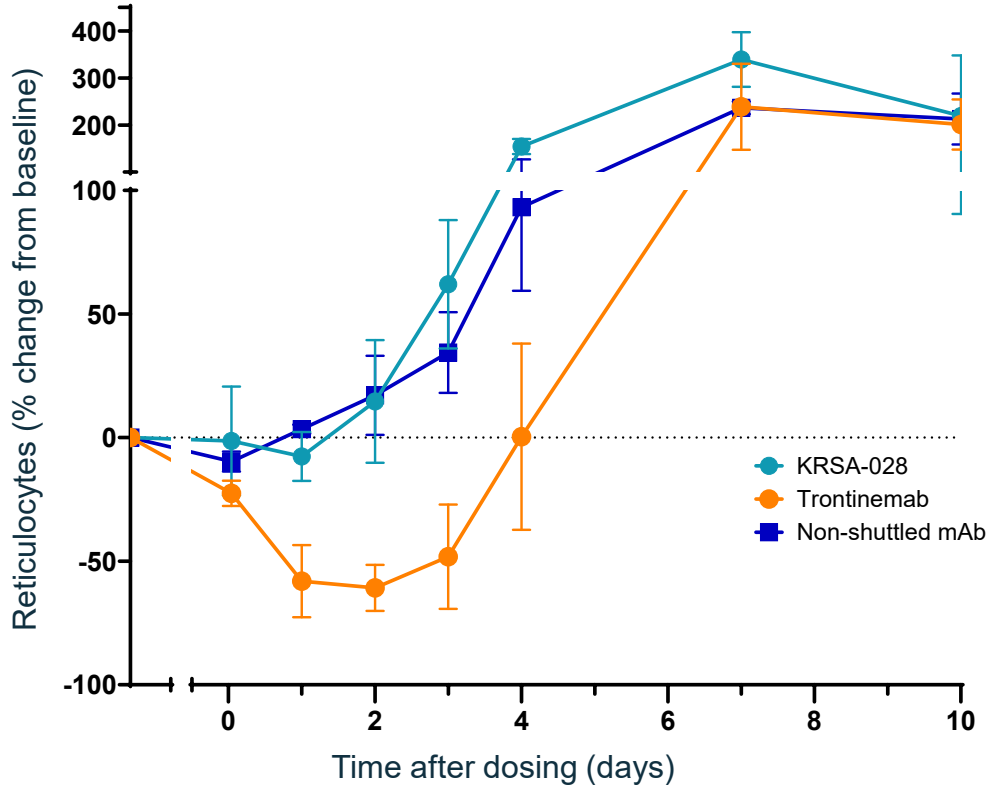
KRSA-028 has a longer half-life than trontinemab and avoids reticulocyte depletion in NHPs

KRSA-028 has >2.5x half-life in NHPs compared to trontinemab



KRSA-028 avoids reticulocyte depletion in NHPs



All compounds were dosed single dose in non-human primates (NHP, n=3/group), equimolar to 20 mg/kg trontinemab
Reticulocyte data normalized to reticulocyte count at day -7 and presented as mean ± SEM
Non-shuttled mAb (remternetug), with less immunogenic potential than donanemab, chosen as negative control.

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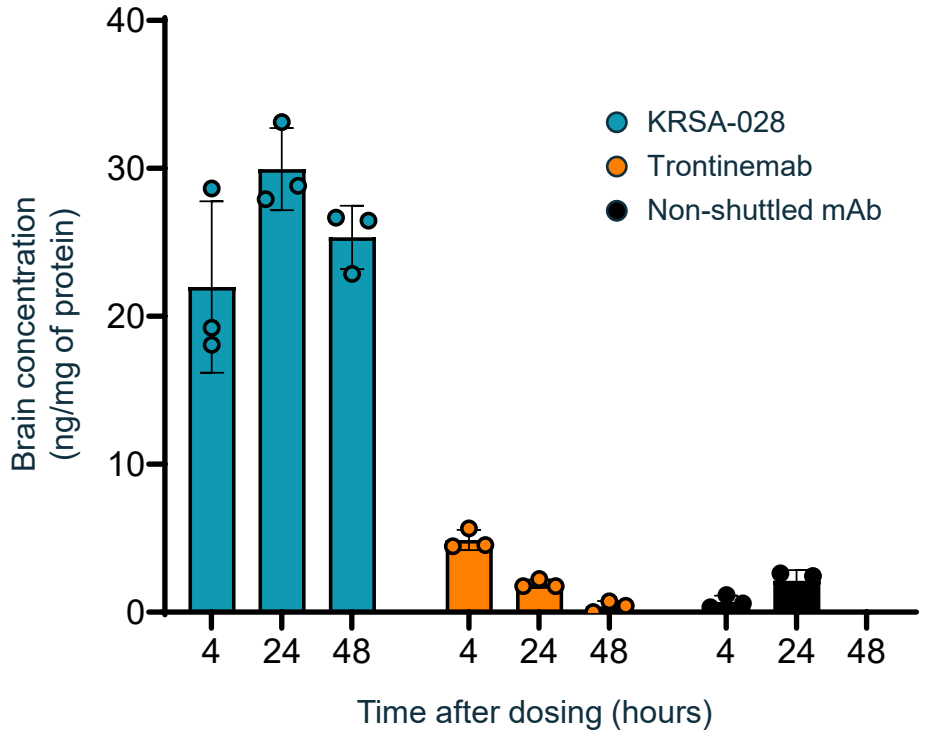
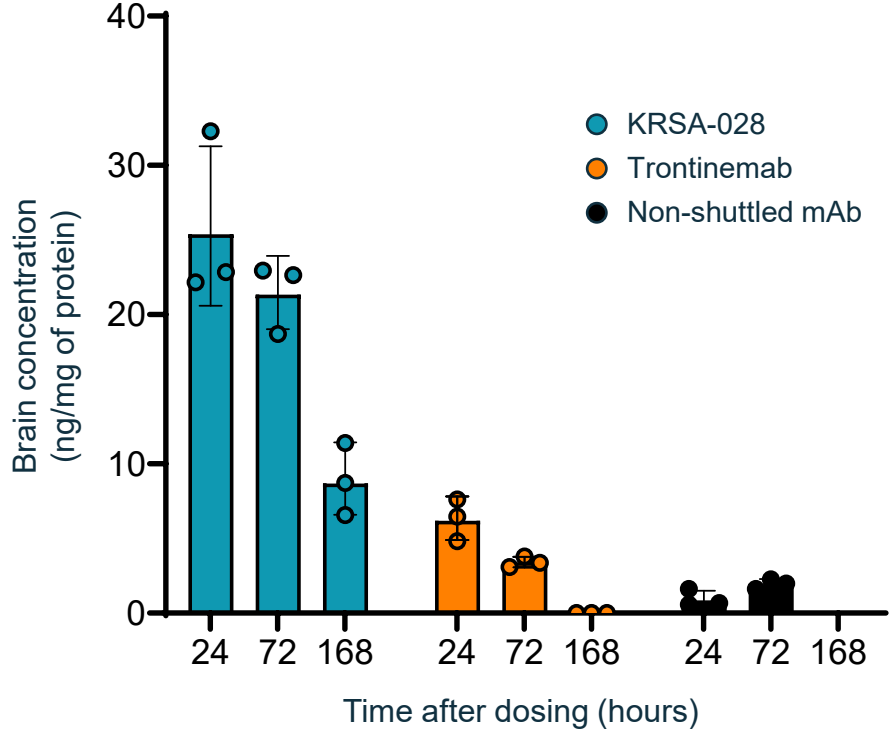
KRSA-028 shows improved brain penetration in mouse and NHP

KRSA-028 demonstrates increased brain penetration in hTfR1/hFcRn mouse model



KRSA-028 shows >6x brain penetration in NHPs compared to trontinemab



Data are mean ± SD. Values below 5 ng/mg (mice) and 1.25 ng/mg (NHP) are estimates as concentrations are below lower limit of quantitation. All compounds were dosed as a single dose (n=3/group). Mice and NHPs were dosed KRSA-028 equimolar to 10 mg/kg trontinemab. Non-shuttled mAb (remternetug) chosen as negative control and dosed at 20 mg/kg. NHP brain PK is an average of concentrations (separately analyzed) from frontal cortex, caudate, putamen, temporal cortex, hippocampus, and cerebellum. Data only collected for first two timepoints for non-shuttled mAb.

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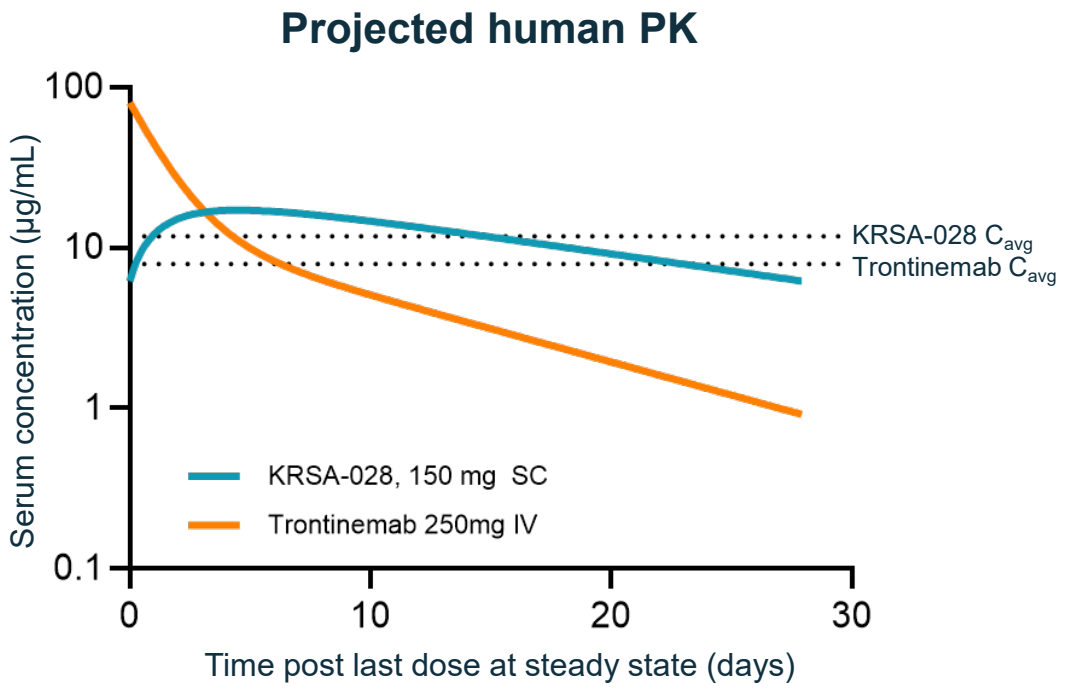
KRSA-028 is expected to match trontinemab Phase 3 IV exposure with low volume SC, compatible with autoinjector



Note: 250 mg trontinemab corresponds to the Ph3 dose of 3.6 mg/kg for average weight of 70 kg; model assumes 50% bioavailability

- PK model suggests that KRSA-028 will match trontinemab Phase 3 exposure with a **monthly SC volume of 1-2 mL**
- Initial KRSA-028 formulation achieved **150mg/mL with low viscosity**, compatible with autoinjector development
- **High stability** in human serum and in NHP
- Robust early formulation stress-test results **de-risk SC development pathway**
- Korsana plans to **initiate clinical development with SC dosing**

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We believe KRSA-028 preclinical data accelerate & de-risk development

Precision engineered for a differentiated therapeutic profile

- **Novel Aβ and TfR1 binding sequences** retain key features of clinically validated molecules
- **High affinity pyroglu-Aβ binding** and clearance via ADCP leverage best-proven mechanism of efficacy
- **TfR1 binding matches trontinemab epitope with similar affinity,** leveraging the best-proven mechanism of brain distribution
- **Clinically validated Fc modifications** add half-life extension and effector function modulation to enable a lower dose and reduce anemia risk
- **Early formulation work supports high concentration, low viscosity** to enable low-volume SC
- **Composition of matter** patent applications filed

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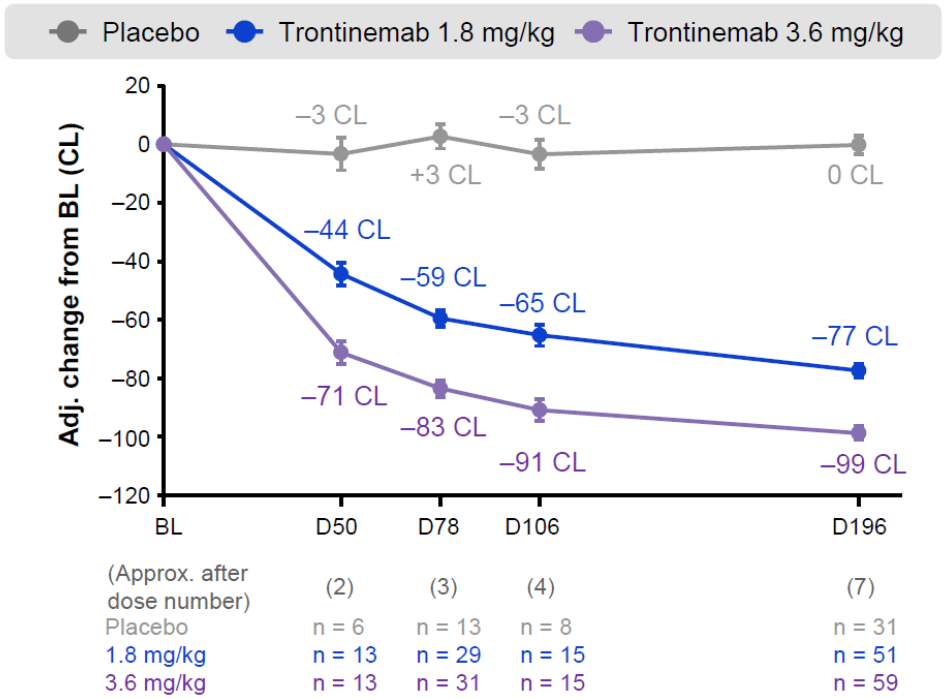
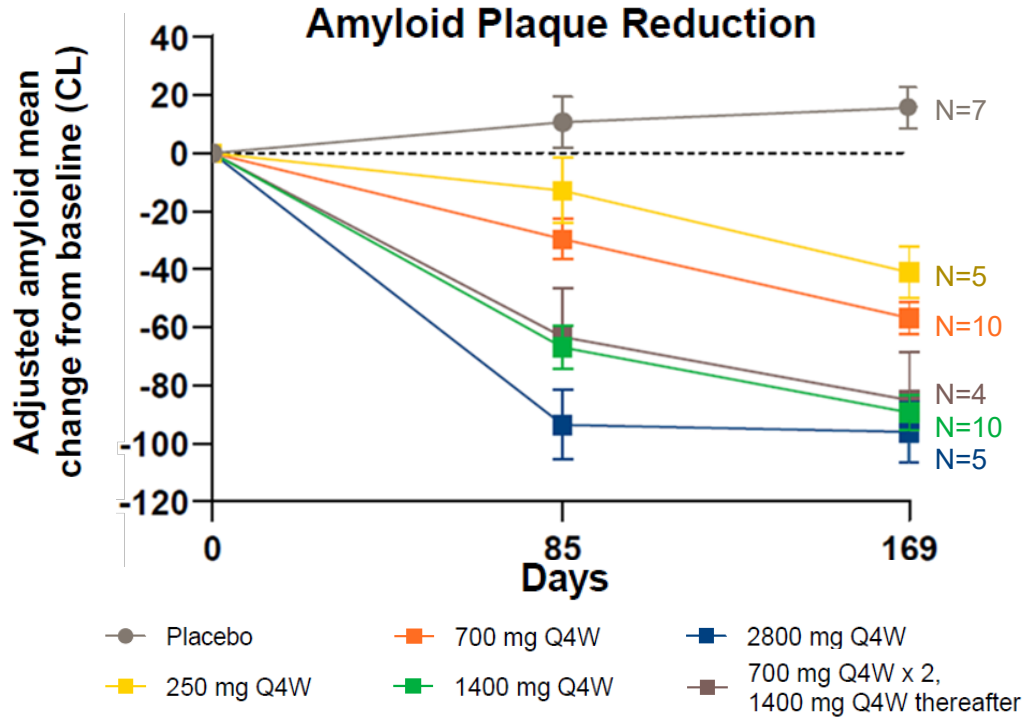
Korsana has a rapid path to value creation

PET imaging in early clinical development provides rapid proof of concept and dose-ranging for amyloid plaque clearing

Trontinemab Phase 1/2: Robust dose-response by Day 50



Remternetug Phase 1: Robust dose-response by Day 85



We believe **KRSA-028 amyloid PET** data – **achievable in a Phase 1/1b trial** – should provide high confidence in **predicting Phase 3 CDR-SB**, the endpoint for full approval.

Notes: Remternetug data from Ph1 LAKB study (IV). Remternetug phase 3 regimen is administered subcutaneously; dose not disclosed.
Source: 2025 Kulic (CTAD Presentation); 2023 Jin (AD/PD Presentation).

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A confluence of scientific innovations and market dynamics are driving Alzheimer's treatment to a major inflection point

Despite a slower than expected launch, sales of Aβ therapies are accelerating

- **Improved dose regimens** (SC, dose titration)
- **Blood-based biomarkers are gaining traction**
- **Increasing footprint of global approvals**
- **Future entrants (e.g., trontinemab) will further grow market**

Sales expected to reach $1B in 2026, $5B+ by 2030

Upcoming catalysts in presymptomatic AD may lead to rapid market expansion

- **Donanemab TRAILBLAZER-ALZ 3** (~2027)
- **Lecanemab AHEAD 3-45** (~2028)
- **Remternetug TRAILRUNNER-ALZ 3** (~2029)
- **Trontinemab PrevenTRON** (study start 2026)

Data could greatly expand eligible patient pool

The future of Alzheimer's treatment will center on a **prevention-based paradigm**, where patients are **diagnosed before symptom onset** and given a **safe, convenient Aβ plaque-clearing therapy.**

Sources: Biogen SEC filings; Lilly SEC filings; VisibleAlpha

KRSA-028 Phase 1/2 trial: designed to rapidly deliver potentially differentiating data

Trial design: Single Ascending Dose in healthy volunteers, with integrated Multiple Ascending Dose in Alzheimer's disease patients, and expansion cohorts designed to enable Phase 3

Data Mid-27:
Safety, PK, and CNS distribution

Key Data YE27-1Q28:
Plaque clearance by PET imaging

Intended to rapidly enable
Phase 3 Program

Note: Trial design pending regulatory approval

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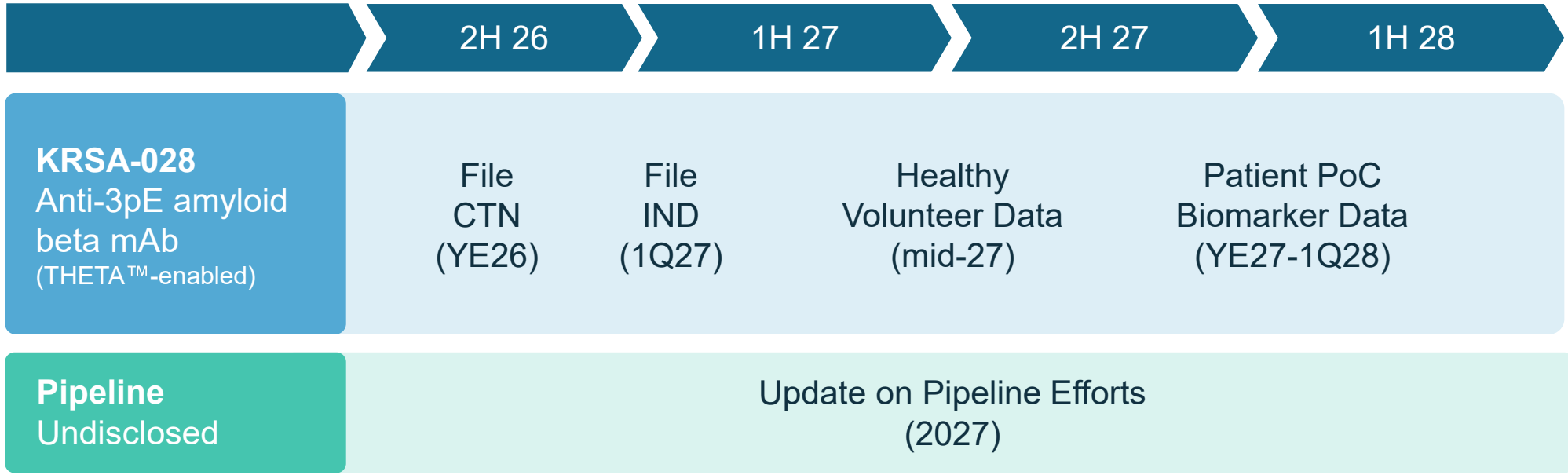
Korsana is well-funded through multiple clinical milestones



Move quickly to dose ranging & expansion cohorts, intended to support ***rapid initiation of pivotal trials***

Strong cash position of ~$475M[1] supports KRSA-028 Phase 3 readiness as well as pipeline expansion, with runway into 2029

1: Pro-forma cash a/o 6/30 including PIPE, net of projected deal costs

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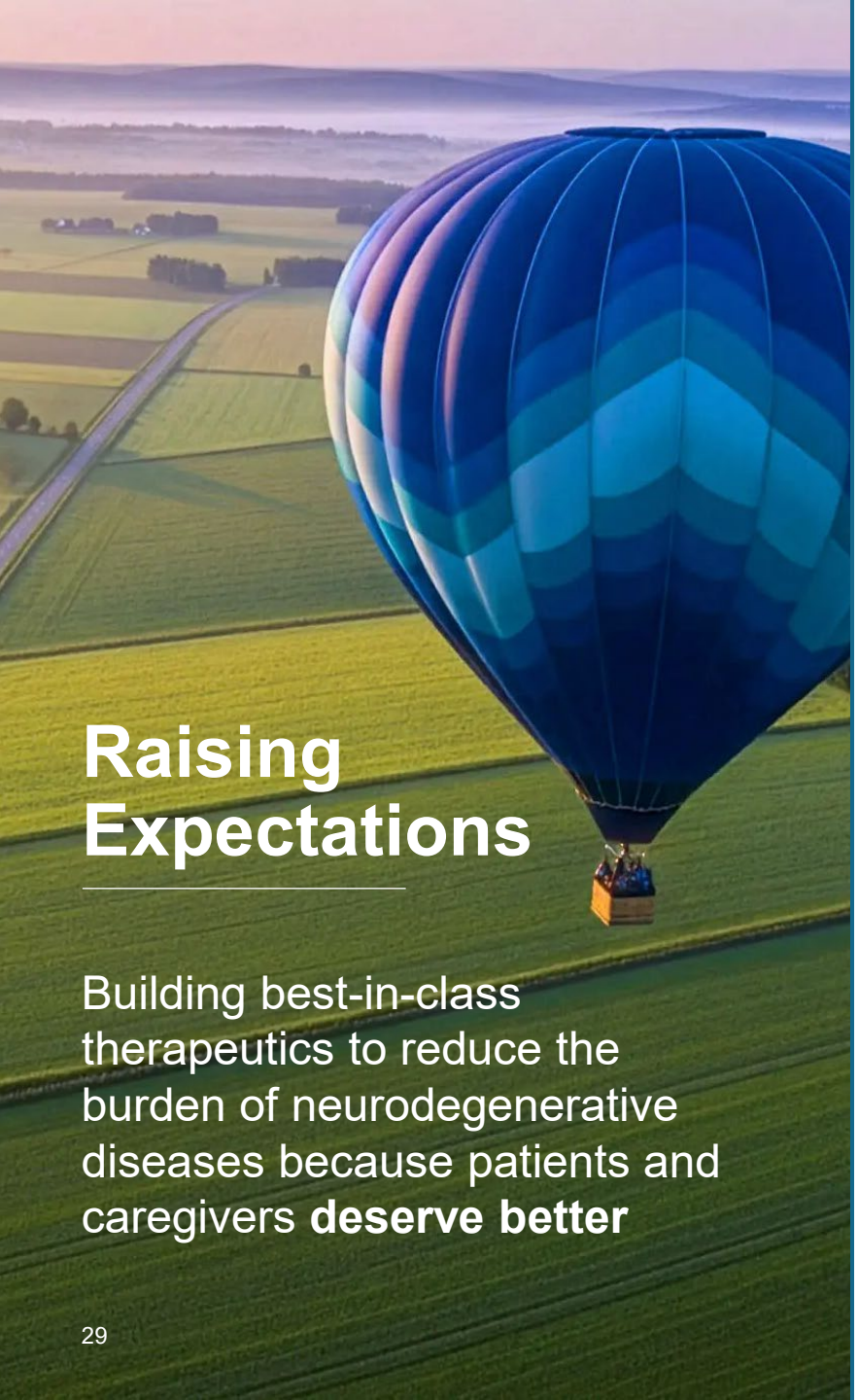

Raising Expectations

Building best-in-class therapeutics to reduce the burden of neurodegenerative diseases because patients and caregivers **deserve better**

1

Vast, de-risked opportunity

Alzheimer's is a massive, growing unmet need with amyloid clearance clinically and regulatorily validated as a disease-modifying approach.

2

KRSA-028: designed to be best-in-class

Next-generation shuttled anti-amyloid designed to build on clinically validated plaque-clearing biology with improved safety and convenience, including low-volume, monthly subcutaneous dosing.

3

Rapid and capital-efficient path to value

Healthy volunteer PK and CSF data expected in mid-2027, followed by Alzheimer's patient amyloid PET and biomarker PoC data by YE27–1Q28.

4

Well-capitalized, with a proven team

~$475M[1] cash funds operations into 2029, led by seasoned leadership and board, and backed by top-tier investors.

1: Pro-forma cash a/o 6/30 including PIPE, net of projected deal costs

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Shares outstanding post merger and financing

As of September 8, 2026		Number of Shares[1]
COMMON STOCK	Shares outstanding	45.5M
COMMON STOCK EQUIVALENTS	Series B preferred	4.1M
	Pre-funded warrants	5.4M
COMMON STOCK AND COMMON STOCK EQUIVALENTS	**Total shares outstanding[2]**	**55.1M**

1. Shown on an as-converted-to-common basis; 2. Excludes common stock equivalents issuable under the company's stock incentive plans and warrants issuable to Parasa. Details may not sum to totals due to rounding.

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Thank you