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Korsana Biosciences Overview

April 2026

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Certain information set forth in this presentation contains "forward-looking statements" within the meaning of applicable United States securities legislation. Except for statements of historical fact, certain information contained herein constitutes forward-looking statements which include but are not limited to statements regarding: our business strategy, including our ability to develop potentially best-in-class therapies initially focused on neurodegenerative disorders like Alzheimer's disease, including a potentially best-in class shuttled anti-Aβ therapy offering meaningful improvements over trontinemab; the efficacy, safety profile, dosing regime, convenience, half-life, and tolerability of KRSA-028, including expectations regarding subcutaneous formulation, dosing volume, and projected human pharmacokinetics; Korsana's ongoing and future clinical development activities, including the expected timing of CTN and IND filings, healthy volunteer PK and CSF data, and interim clinical proof of concept data for KRSA-028; the expected timing of unveiling additional THETA™ enabled programs; estimated market sizes, potential growth opportunities, potential value creation and comparable company valuations and deal economics; the anticipated growth of the Alzheimer's therapeutics market and the expansion of the eligible patient population, including through presymptomatic indications and blood-based biomarker adoption; the length of time that the Company believes its existing cash resources will fund its operations, including expectations of cash runway extending into 2029; the proposed reverse merger transaction and related pre-closing financing, including the expected timing and completion thereof, estimated post-closing capitalization, and the expected ownership percentages of the combined company; and management's assessment of future plans and operations which are based on current internal expectations, estimates, projections, assumptions and beliefs, which may prove to be incorrect. 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This presentation is not a substitute for the registration statement or for any other document that Cyclerion may file with the SEC in connection with the proposed transactions. In connection with the proposed transactions between Cyclerion and Korsana, Cyclerion intends to file relevant materials with the SEC, including a registration statement on Form S-4 that will contain a proxy statement/prospectus of Cyclerion. CYCLERION URGES INVESTORS AND SHAREHOLDERS TO READ THE REGISTRATION STATEMENT, PROXY STATEMENT/PROSPECTUS AND ANY OTHER RELEVANT DOCUMENTS THAT MAY BE FILED WITH THE SEC, AS WELL AS ANY AMENDMENTS OR SUPPLEMENTS TO THESE DOCUMENTS, CAREFULLY AND IN THEIR ENTIRETY IF AND WHEN THEY BECOME AVAILABLE BECAUSE THEY WILL CONTAIN IMPORTANT INFORMATION ABOUT CYCLERION, KORSANA, THE PROPOSED TRANSACTIONS AND RELATED MATTERS. Investors and shareholders will be able to obtain free copies of the proxy statement/prospectus and other documents filed by Cyclerion with the SEC (when they become available) through the website maintained by the SEC at www.sec.gov. Shareholders are urged to read the proxy statement/prospectus and the other relevant materials when they become available before making any voting or investment decision with respect to the proposed transactions. In addition, investors and shareholders should note that Cyclerion communicates with investors and the public using its website (www.cyclerion.com).

Participants in the Solicitation

Cyclerion, Korsana and their respective directors and executive officers may be deemed to be participants in the solicitation of proxies from shareholders in connection with the proposed transactions. Information about Cyclerion's directors and executive officers, including a description of their interests in Cyclerion, is included in Cyclerion's most recent Annual Report on Form 10-K, subsequent Quarterly Reports on Form 10-Q filed with the SEC, including any information incorporated therein by reference, as filed with the SEC, and other documents that may be filed from time to time with the SEC. Additional information regarding these persons and their interests in the transaction will be included in the proxy statement/prospectus relating to the proposed transactions when it is filed with the SEC. These documents can be obtained free of charge from the sources indicated above.

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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
- Built on **Therapeutic Targeting (THETA™),** our next-generation BBB-penetrant shuttle platform
- Committed to **move fast** and develop a pipeline with **long-term defensibility**
- Proposed **reverse merger with Cyclerion Therapeutics and concurrent $380M private placement** expected to close in 3Q26, after which Korsana expected to trade on NASDAQ with ticker **KRSA**

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**

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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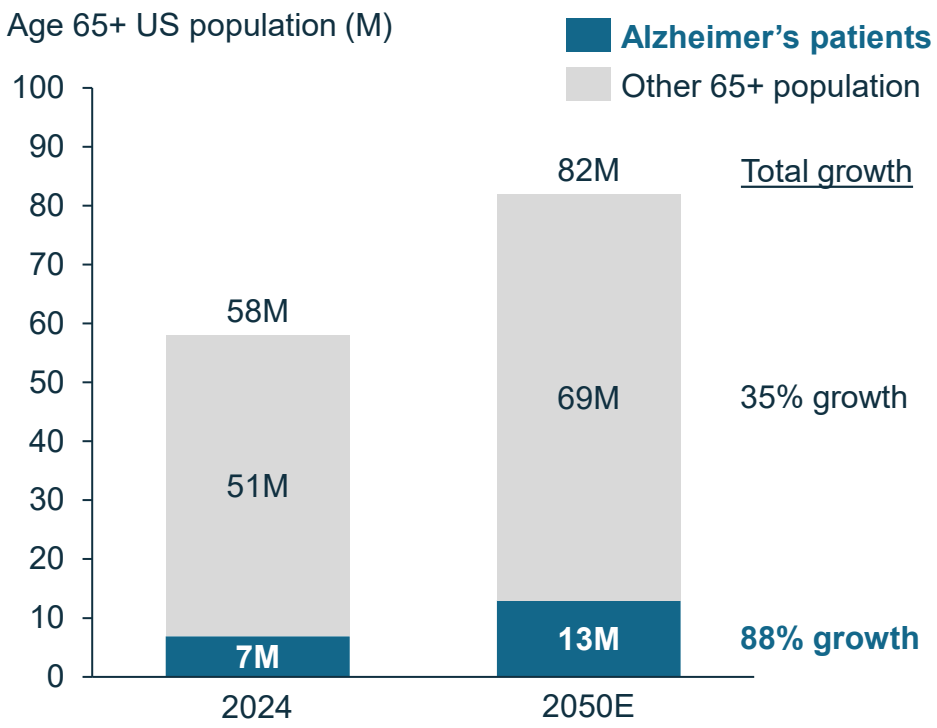
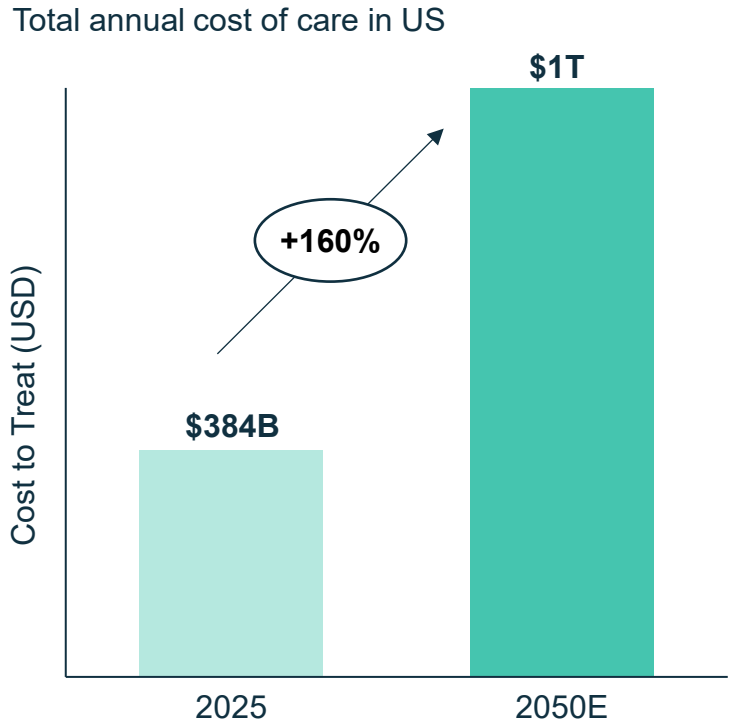
Alzheimer’s is a vast and de-risked opportunity

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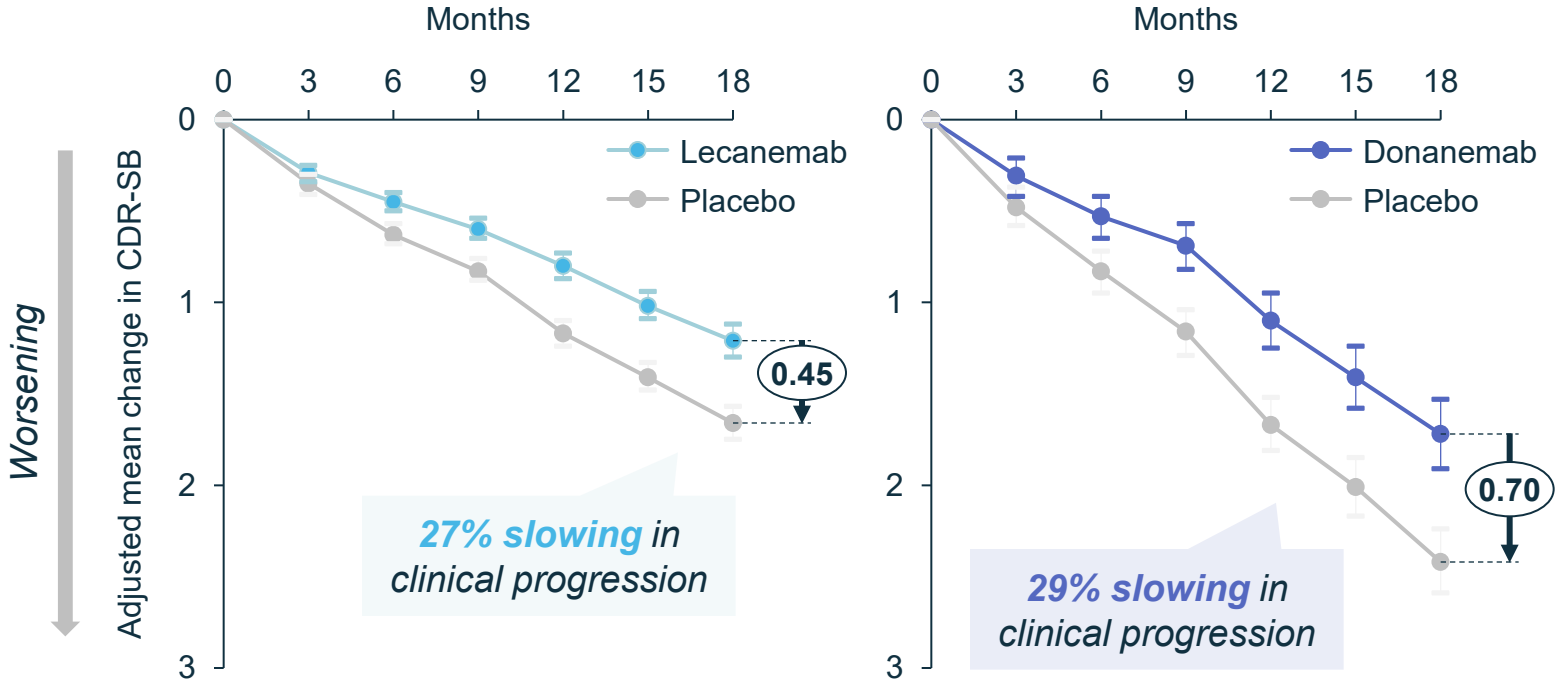
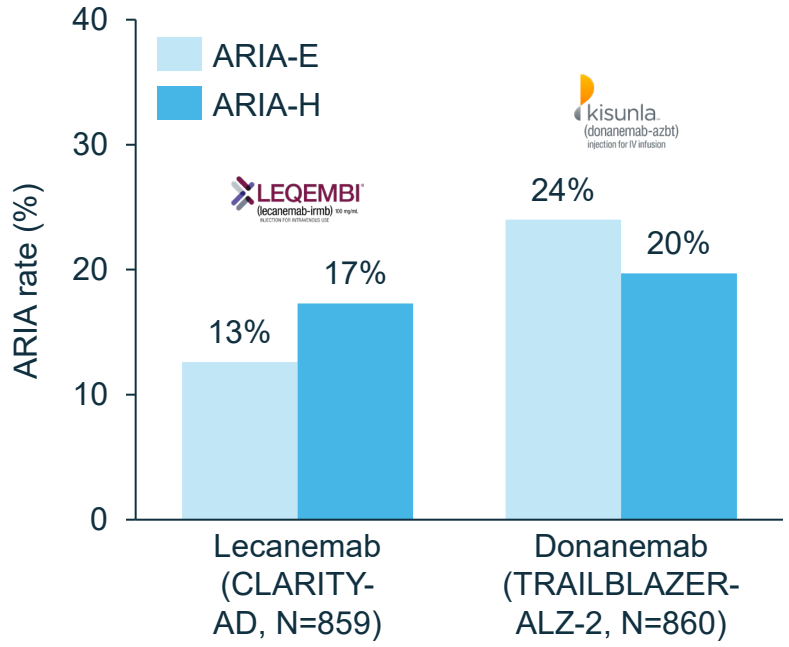
Despite clinical progress and approvals, today's anti-Aβ therapies leave significant room for improvement

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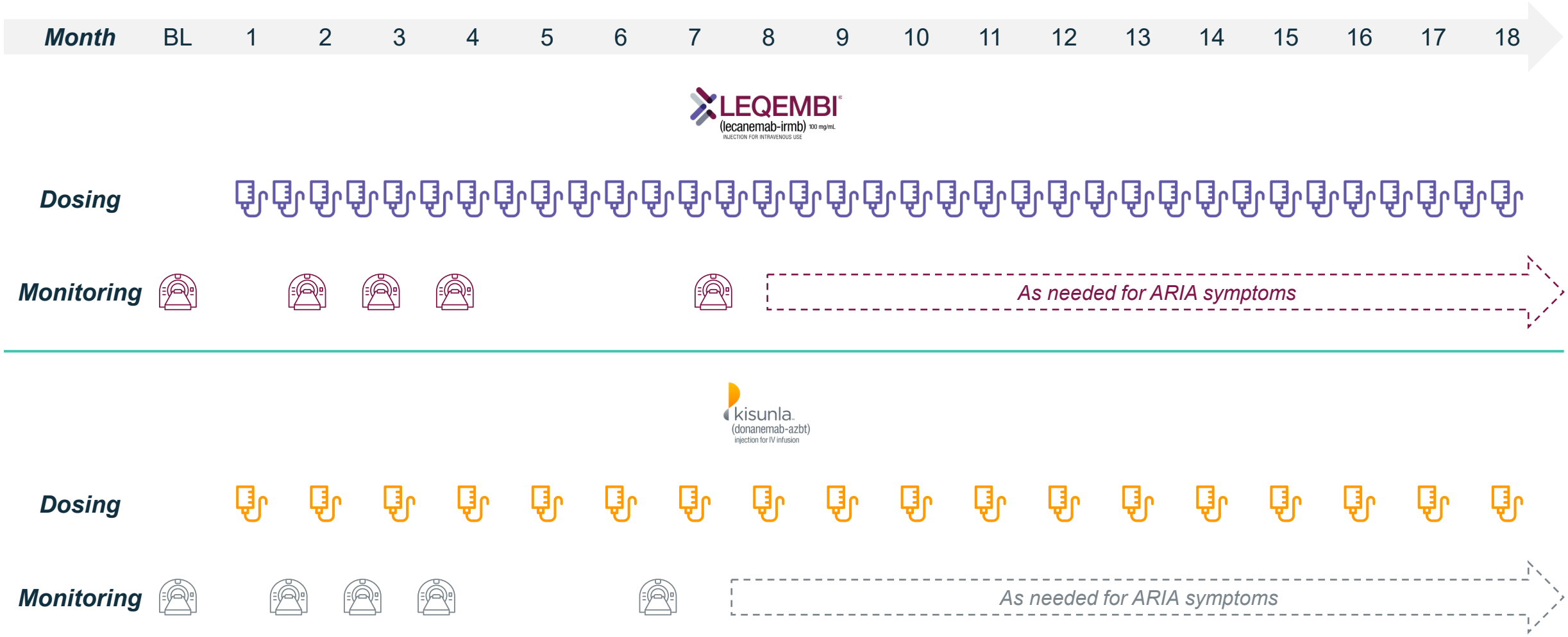
Approved therapies are also highly inconvenient, with onerous IV dosing and MRI monitoring on label



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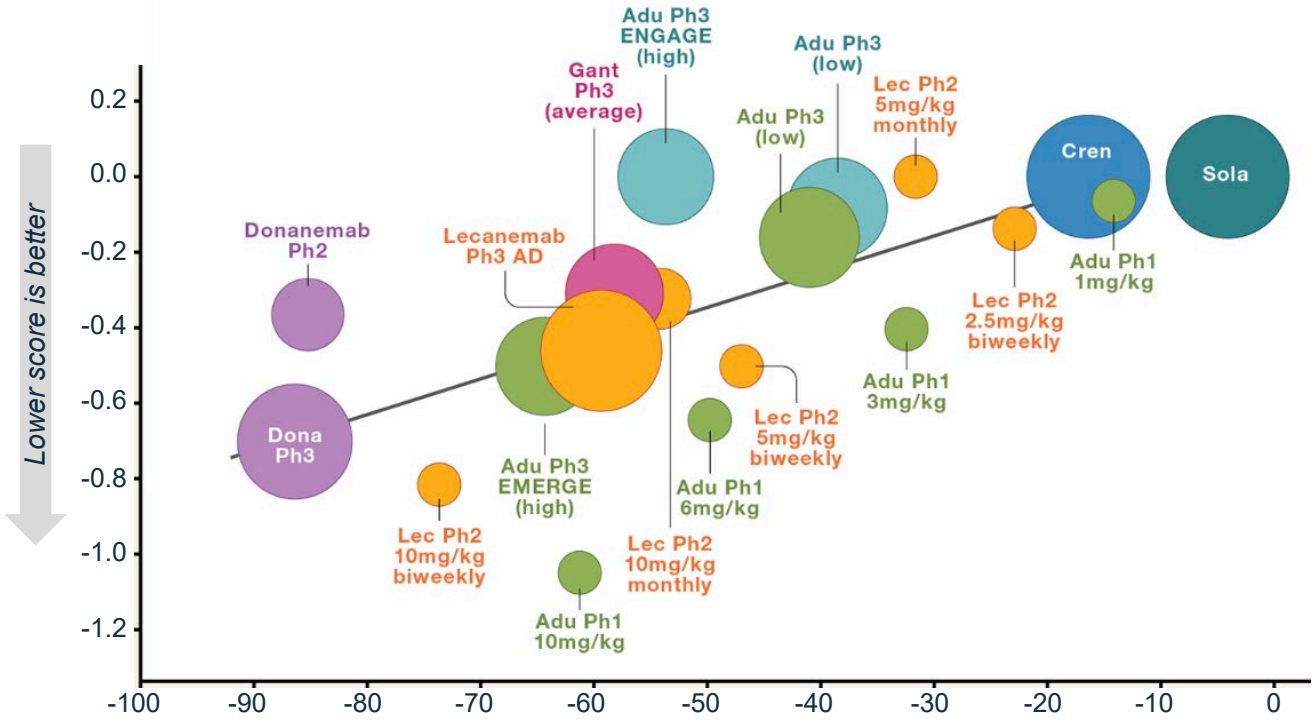
First-gen therapies have laid out the roadmap for success: amyloid reduction predicts slowing of cognitive decline

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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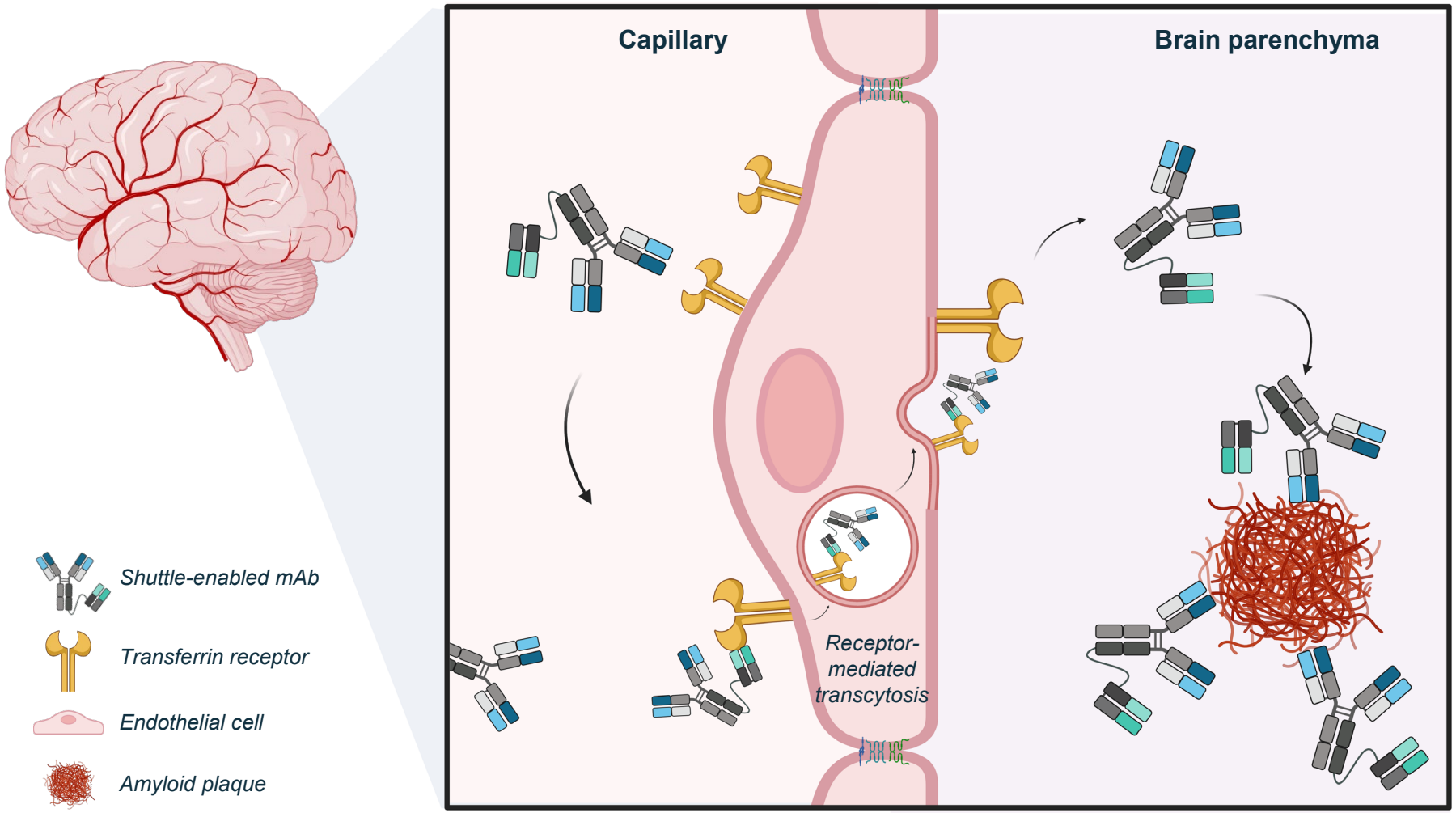
Shuttling is the best way to improve existing agents

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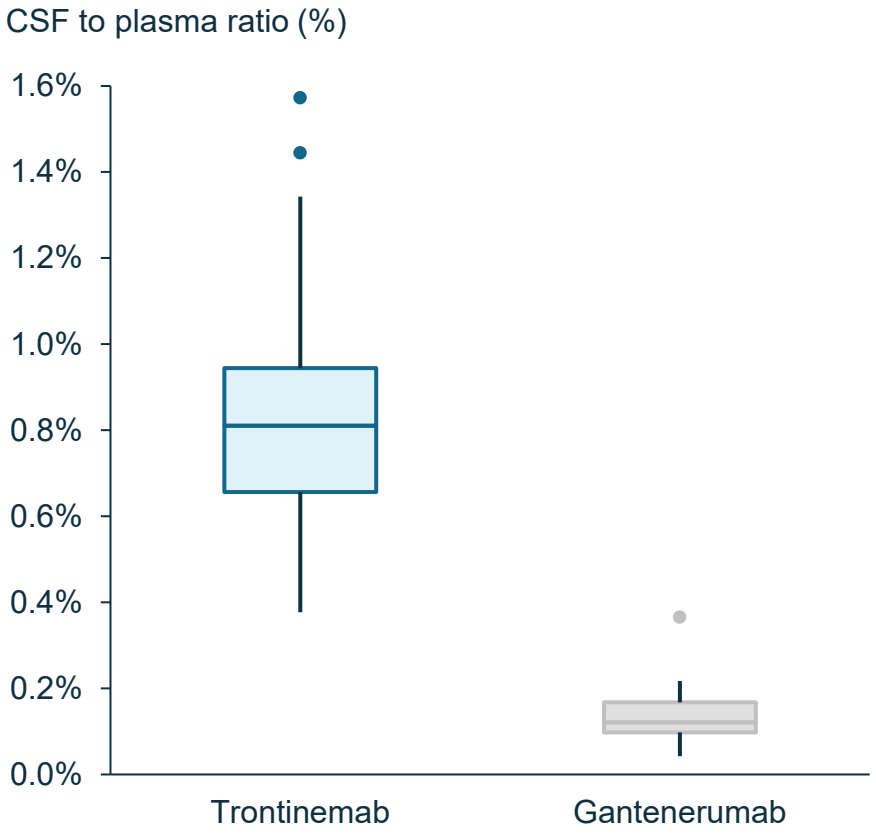
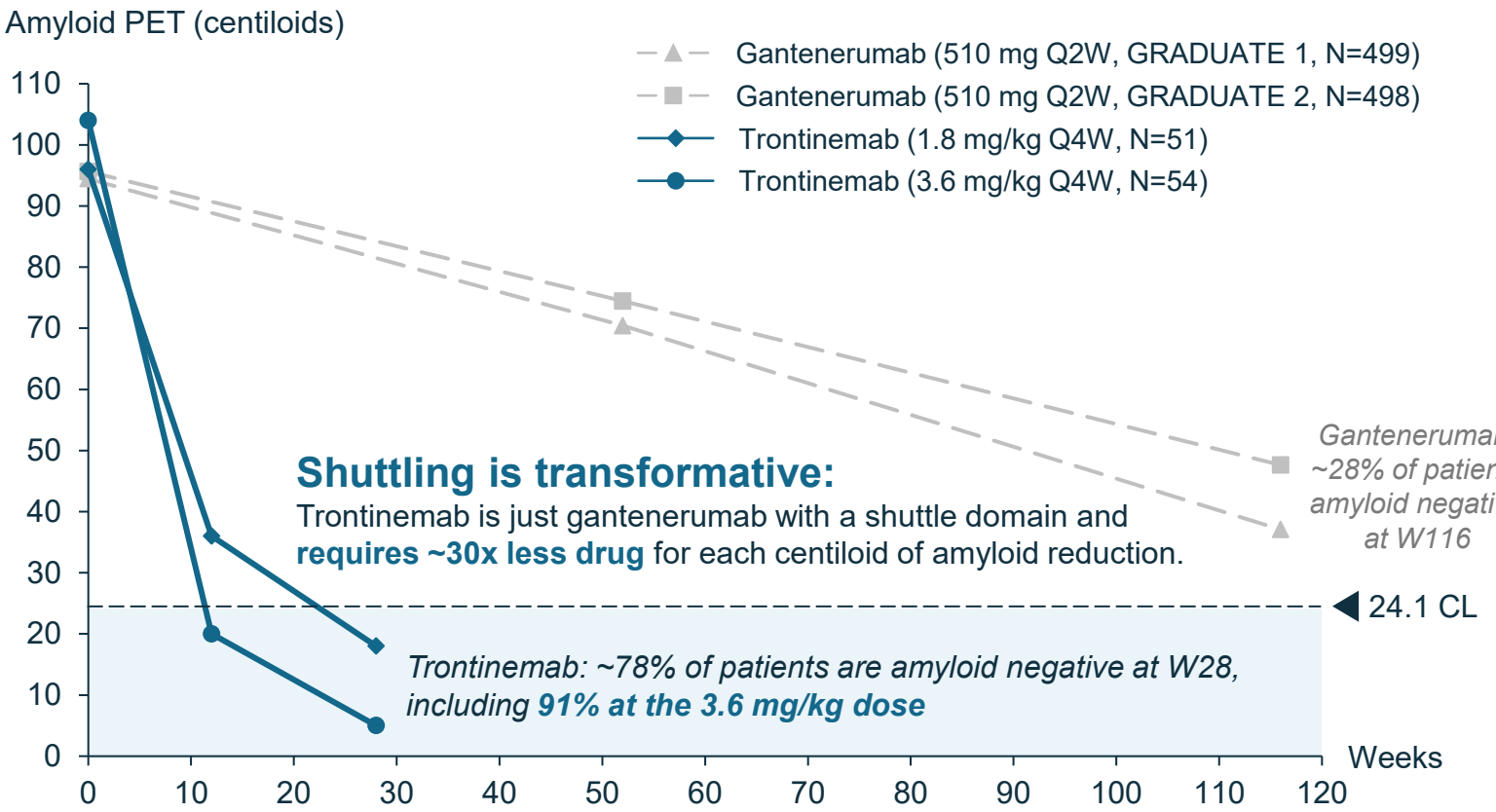
Roche's trontinemab, the first shuttled anti-Aβ antibody, has shown a significant efficacy improvement over first-gen gantenerumab…

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 (AAIC Presentation), 2025 Klein (AAIC Presentation).

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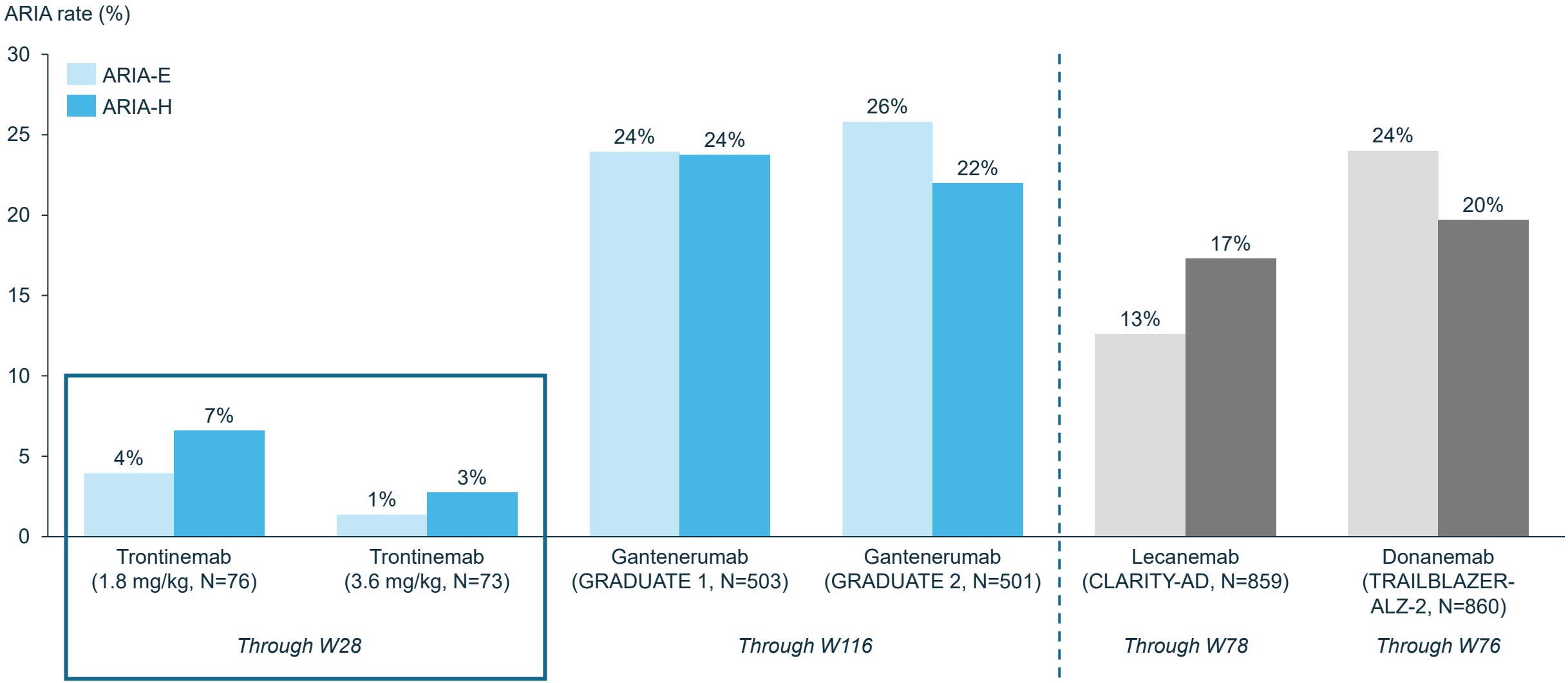
… and greatly reduces ARIA, the critical safety signal for this class of therapies



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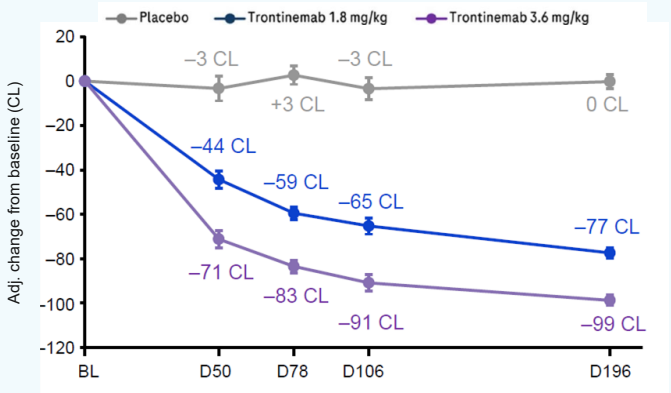
However, as a first-gen shuttled Aβ, trontinemab's profile leaves substantial headroom for competitive differentiation

Efficacy

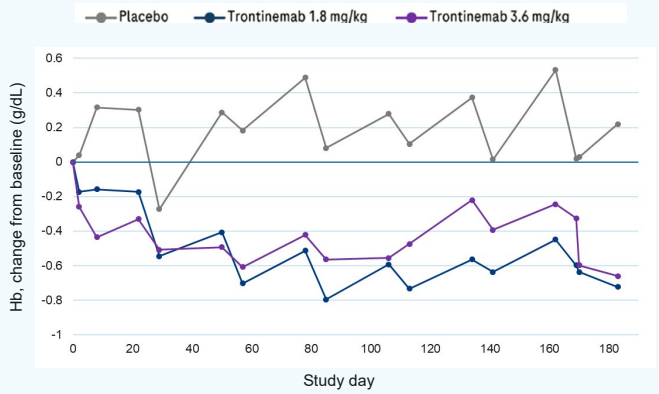
Mean amyloid plaque reductions from baseline



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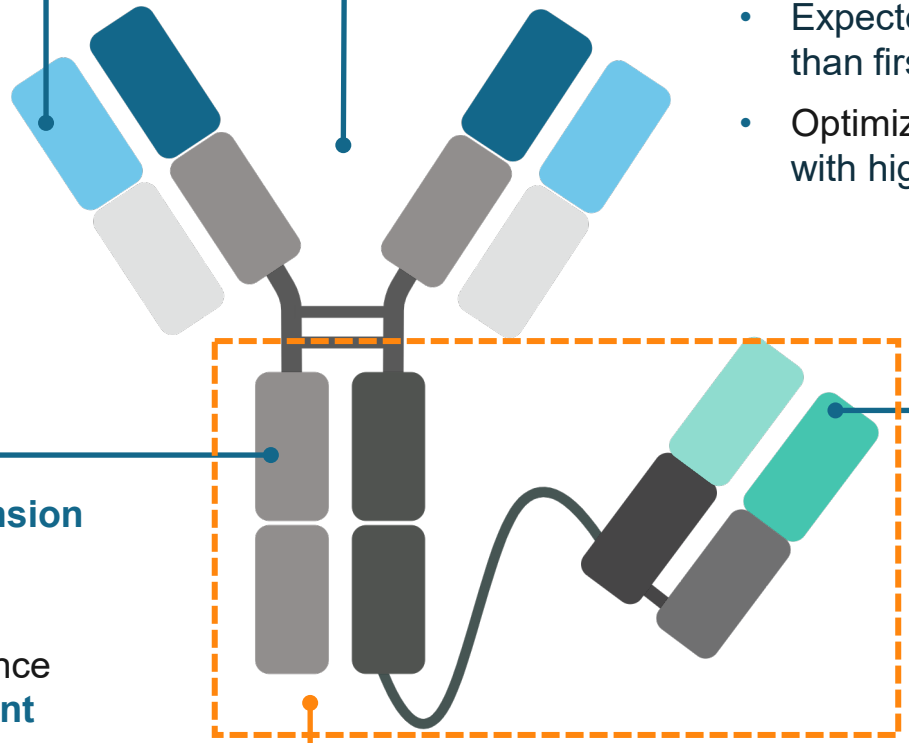
KRSA-028 is a next-gen, potentially best-in-class shuttled anti-Aβ

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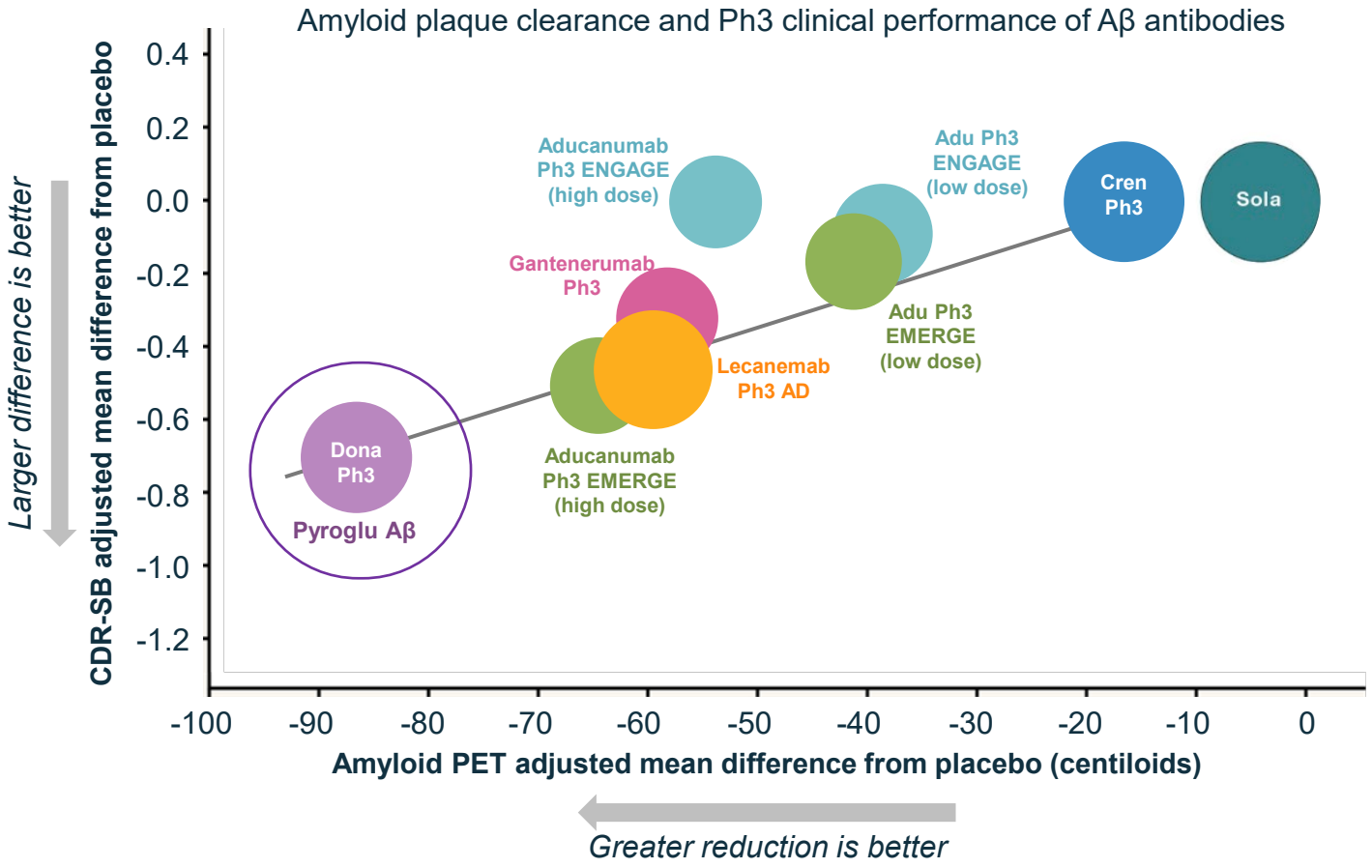
Targeting the plaque-specific pyroglu-Aβ epitope has been shown to deliver the most promising clinical efficacy in Phase 3 trials



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

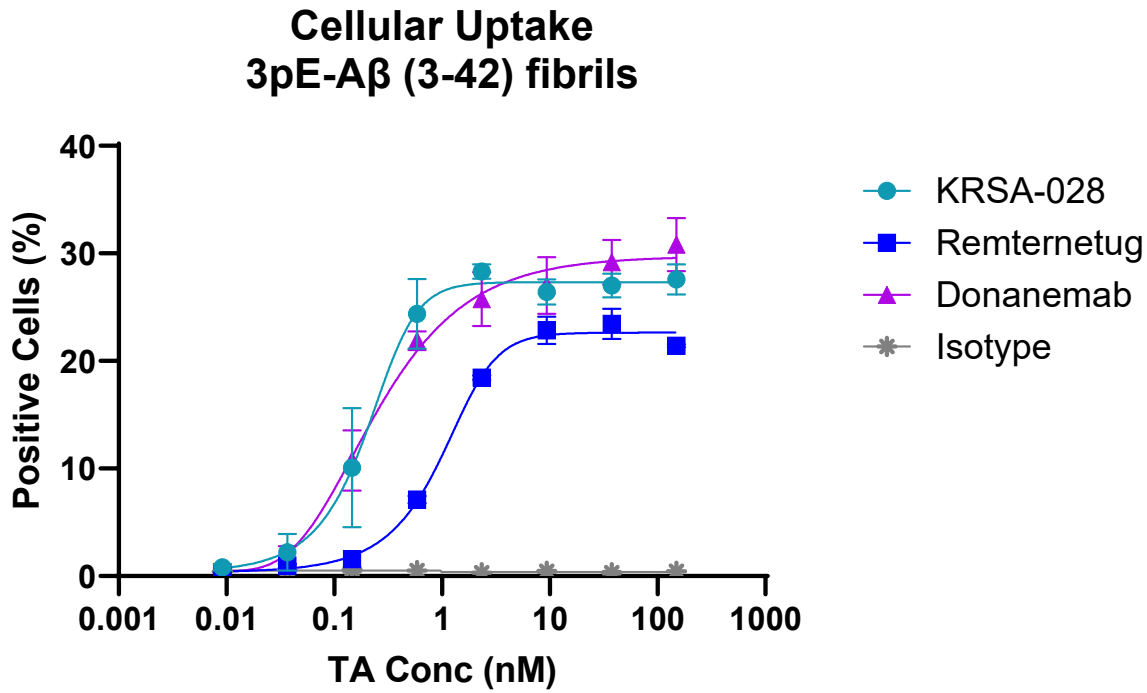
KRSA-028 Aβ backbone:

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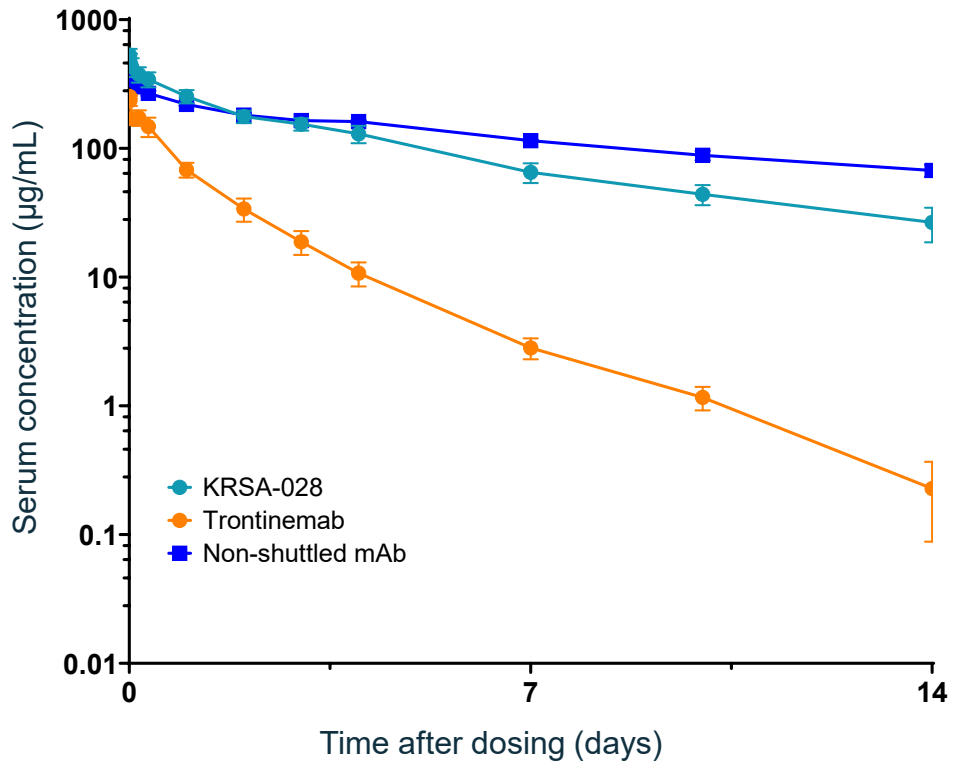
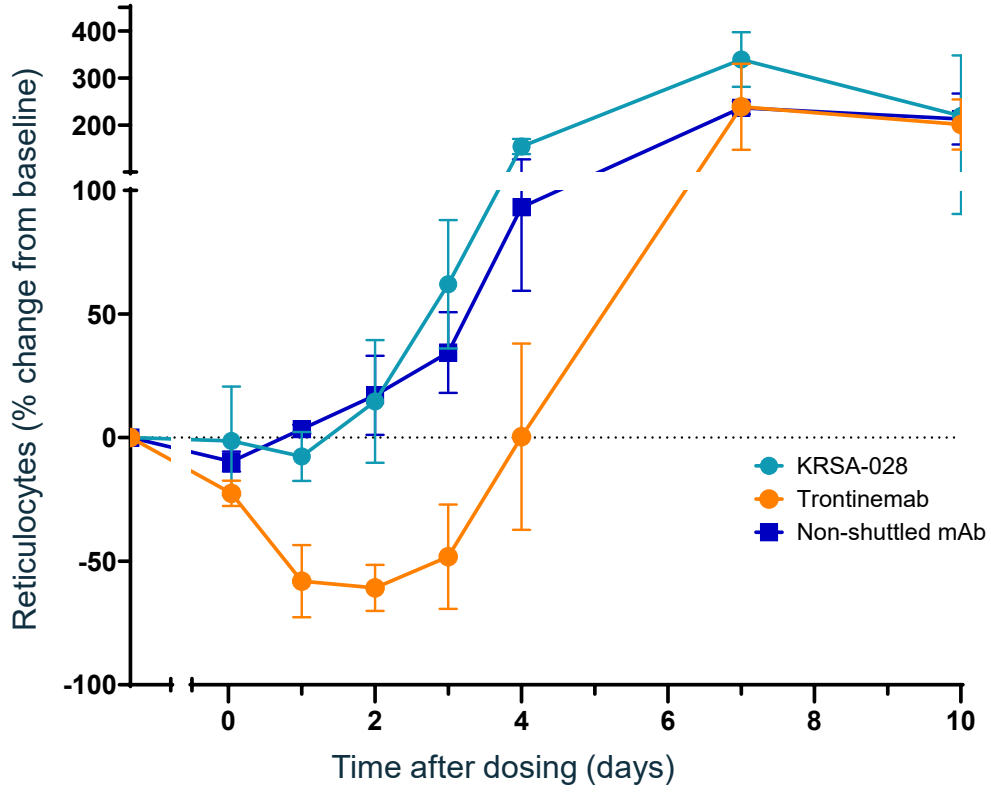
KRSA-028 has a longer half-life than trontinemab and avoids reticulocyte depletion in NHPs

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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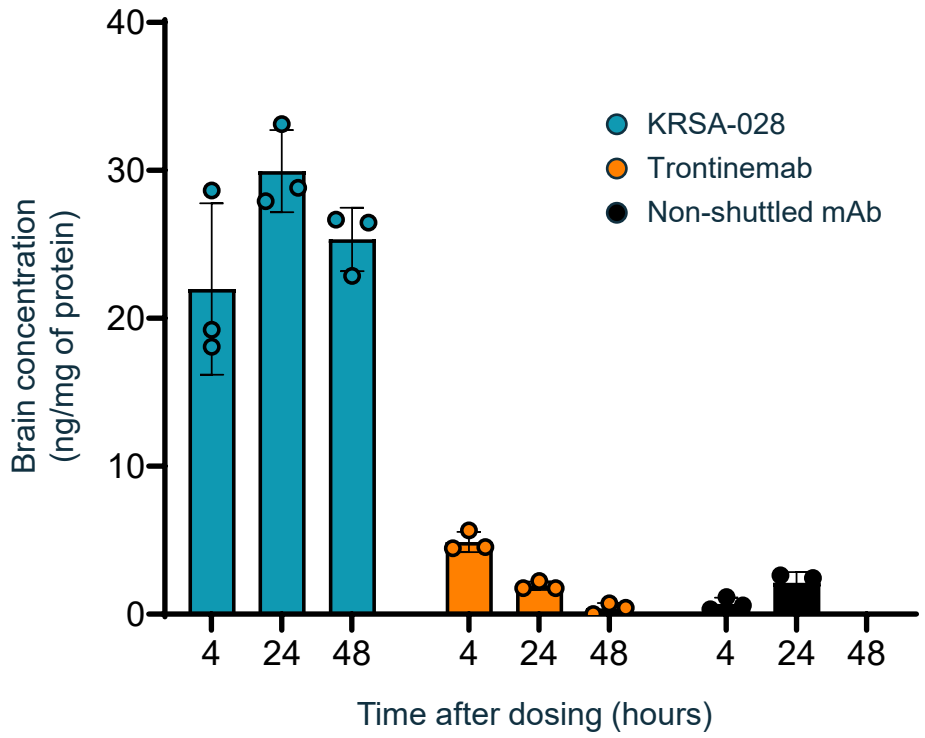
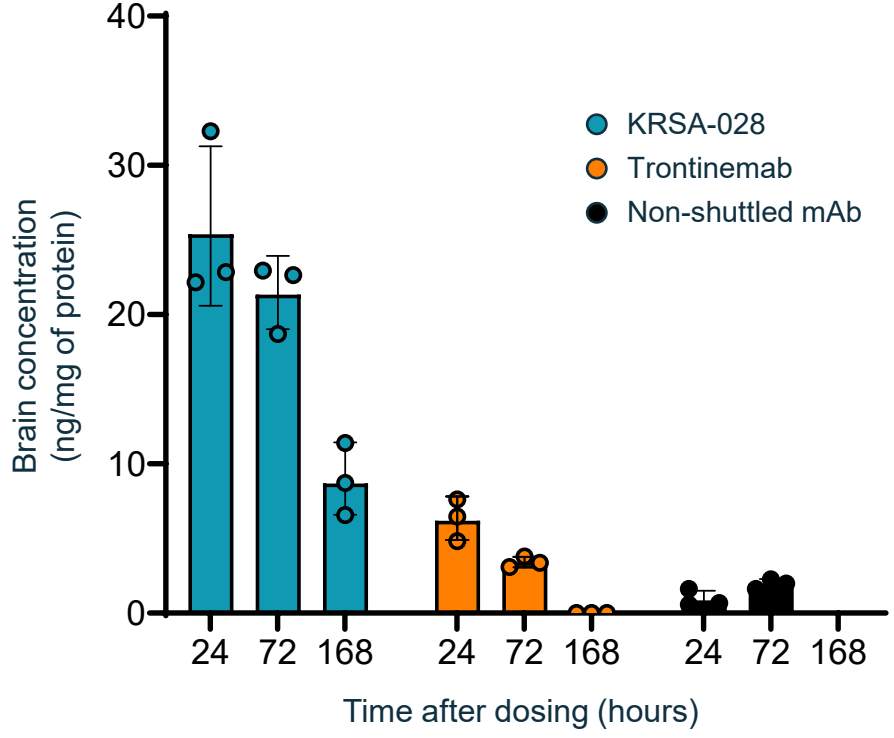
KRSA-028 shows improved brain penetration in mouse and NHP

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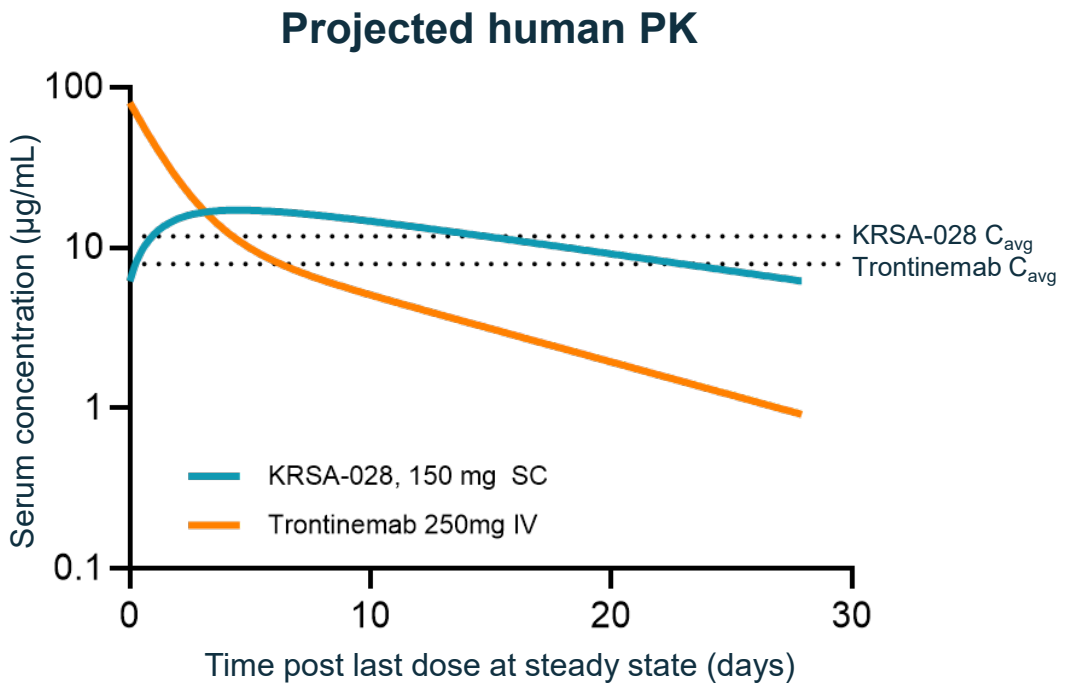
KRSA-028 is expected to match trontinemab Phase 3 IV exposure with low volume SC, compatible with autoinjector



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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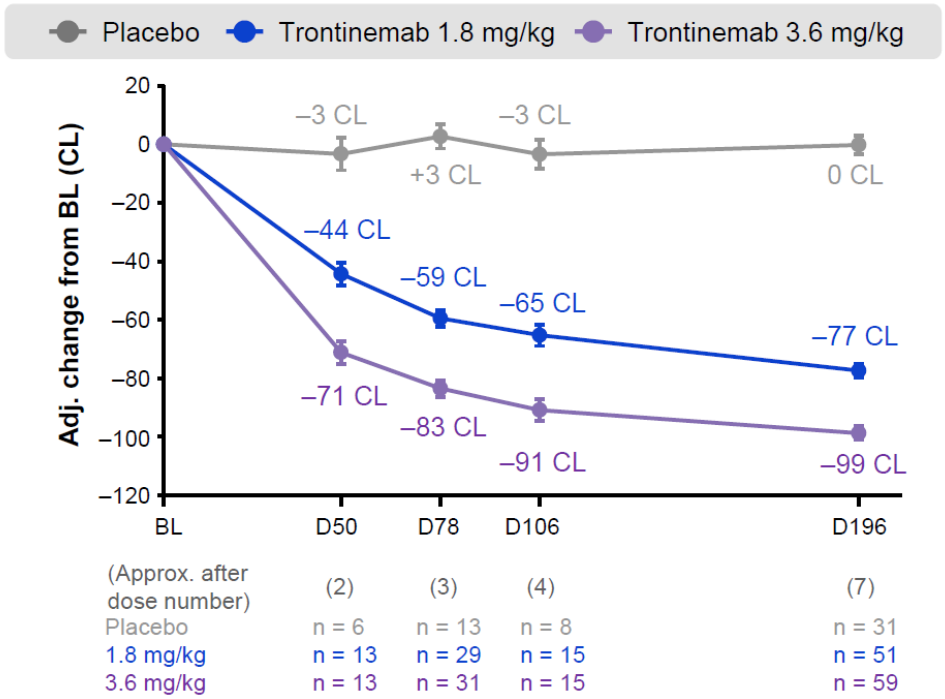
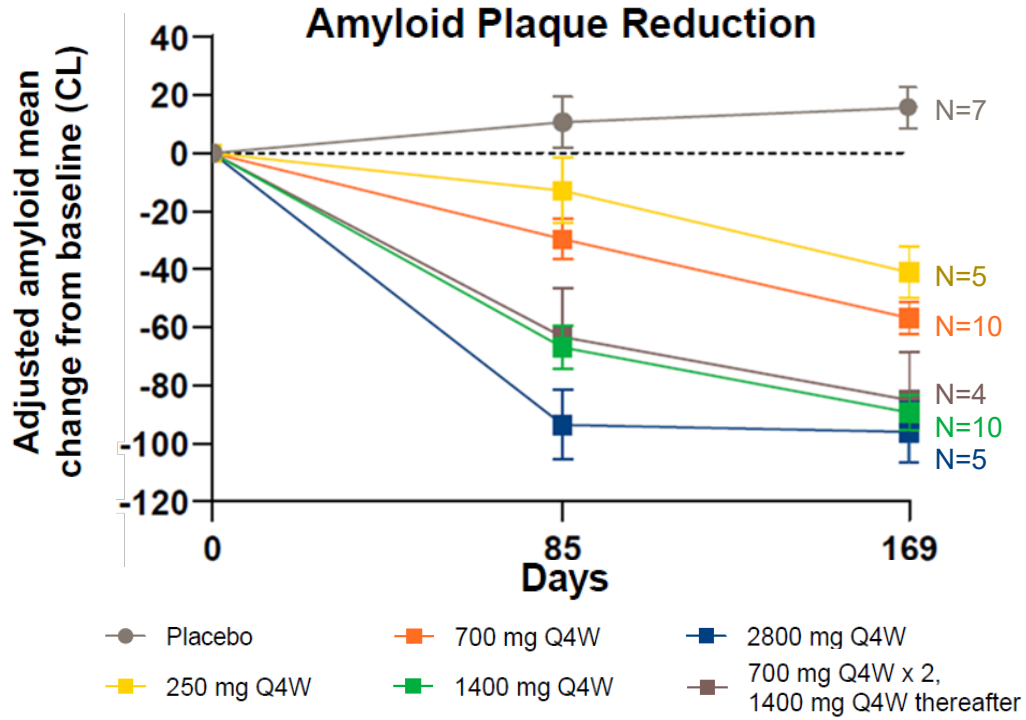
Korsana has a rapid path to value creation

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

Korsana has a clear path to early de-risking and value creation

Comparable public and acquired companies illustrate substantial potential near-term value for Korsana

KRSA-028 anticipated milestones:

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	✓ *Strong preclinical data package*	*De-risking healthy volunteer data (safety, PK, and CNS distribution)*	*Biomarker PoC data in patients demonstrating best-in-class potential*

Comparable public valuations / deal economics by asset stage:

SCINEURO PHARMACEUTICALS, NOVARTIS Shuttled Aβ mAb for AD **$165M upfront** (2026)	ALIADA THERAPEUTICS, abbvie Shuttled Aβ mAb for AD Ph1 SAD in HVs **$1.4B M&A** (2024)	AVIDITY BIOSCIENCES, NOVARTIS TfR-conjugated oligos Ph2 data in DM1, FSHD **$12B M&A** (2025)
BIOARCTIC, Bristol Myers Squibb™ Shuttled Aβ mAb for AD **$100M upfront** (2024)*	APOGEE THERAPEUTICS IL-13 for atopic dermatitis Ph1 SAD in HVs **~$3.5B market cap****	ALPINEImmuneSciences™, VERTEX BAFF/APRIL inhibitor Ph1/2 data in IgAN **$4.9B M&A** (2024)

Notes: SciNeuro/Novartis, Jan 2025. * BioArctic/BMS, Dec 2024. Deal also includes one non-shuttled Aβ mAb. Aliada/AbbVie, Oct 2024. Asset was in Ph1 SAD in HVs. **Apogee: Positive interim data from Ph1 SAD in HVs disclosed 5 March 2024. Market cap data from 15 March 2024 after a $483M public offering (FactSet). Avidity/Novartis, Oct 2025. Avidity pipeline also includes a pivotal-stage asset in exon 44 DMD. PD: Parkinson's disease. AD: Alzheimer's disease. HV: Healthy volunteers. DM1: Myotonic dystrophy type 1. FSHD: Facioscapulohumeral muscular dystrophy. IgAN: IgA nephropathy. Source: Company press releases and public filings.

Korsana is poised for rapid progress

Near-Term Catalysts

KRSA-028

- **CTN filing expected YE26**
- IND filing expected 1Q27
- Healthy volunteer **PK & CSF data expected Mid-Year 27**
- **Interim clinical PoC data in** Alzheimer's patients by expected **YE27**

Continued advancement of Alzheimer's field likely to solidify Korsana opportunity

Pipeline

- Unveiling **additional THETA™ enabled programs** in 2026-27

Focused on diseases with high unmet need where shuttling could drive best-in-class profile

Well-Financed

- **$25M** seed round Q4 2024
- **$150M** Series A Sept 2025
- **$380M** PIPE, expected to close in 3Q26 alongside merger with CYCN
- **Cash runway into 2029** after expected PIPE close

Strong Comps

- Rapid path to compelling PoC clinical data, comparable to multi-$B public and M&A valuations (e.g., Aliada Ph1, Alpine Ph1/2, Avidity Ph2)

Experienced Leadership

- Seasoned **CEO Jonathan Violin**
  - Prior CEO roles include: Viridian Therapeutics, Dianthus Therapeutics, Quellis Biosciences
- Discovery programs led by **Paragon Therapeutics**
- Board of Directors comprised of **leading biotech investors**
  - Tomas Kiselak (Chair), Fairmount
  - Andrew Gottesdiener, Venrock
  - Michelle Pernice, Fairmount
  - Nimish Shah, Venrock
  - Nilesh Kumar, Wellington

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Estimated capitalization following close of reverse merger & pre-closing financing

		Shares on an as-converted basis	Expected ownership of the combined company
CYCLERION THERAPEUTICS	• Shares of common stock outstanding (including underlying options, preferred & RSAs)	4,681,351	1.5%
KORSANA BIOSCIENCES	• Shares of common stock outstanding (including underlying options and warrants)	20,418,236	40.8%
	• Preferred stock	105,135,942	
PRE-CLOSING FINANCING	• Shares of common stock and PFWs	177,759,243	57.7%
	Estimated total shares of common stock of the combined company post-closing	307,994,772	

Estimated post-closing capitalization based on information as of the signing of the proposed reverse merger and pre-closing financing. Please see disclosures on slides 2–3.

PFW = Pre-funded Warrants

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Thank you