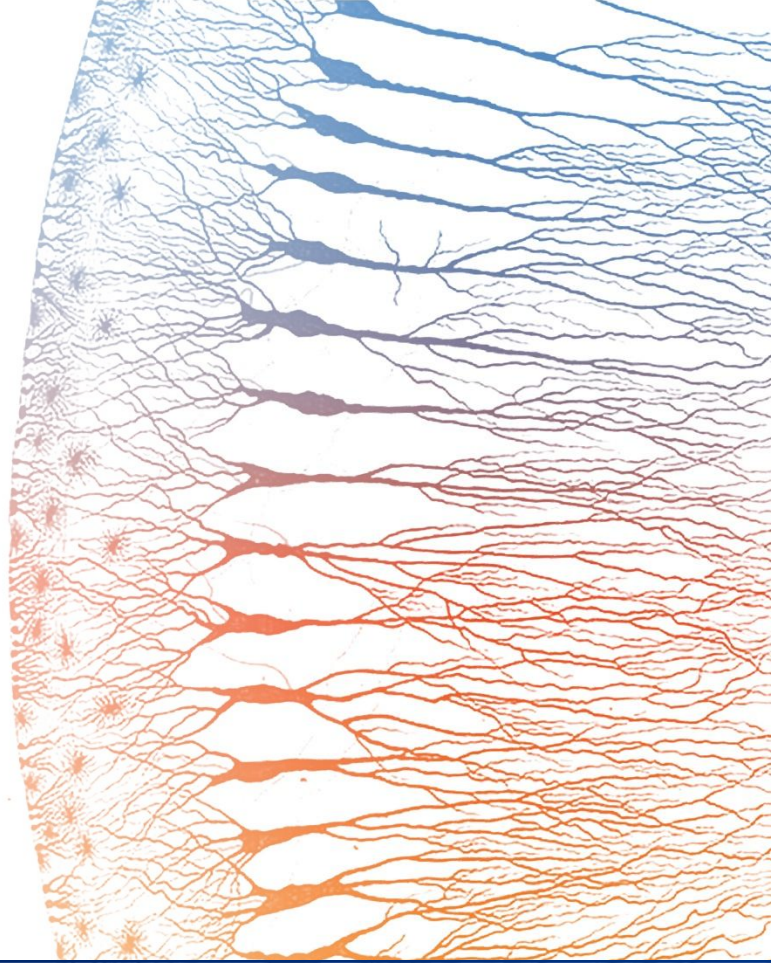
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Leveraging Genetics to Treat Neurological Diseases

Corporate Deck / Nov 2026



Disclaimer and Forward-Looking Statements

This presentation contains forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995 and other federal securities laws, including, without limitation, implied and express statements about Voyager's belief, expectations, plans and prospects regarding the development and regulatory status of its product candidates and advancement of preclinical and clinical development programs, including Voyager's advancement of its tau silencing gene therapy program in AD, VY1706, and the expected timing of enrollment, dosing, initial acute safety data, and initial biomarker-based data; the progress and timing of Voyager's anti-tau antibody program in AD, VY7523, including timing of expected clinical tau PET imaging efficacy data and other clinical data; Voyager's ability to advance programs and product candidates under its current partnerships and collaborations including the anticipated progression into the clinic by Neurocrine of NBIB-'223 in FA, and advancement of gene product candidates under the Novartis licenses and collaboration; the role of tau in the treatment of AD, including as a critical disease-modifying target; the potential for third-party clinical data to inform, derisk or validate Voyager's development programs; the therapeutic potential, safety, and pharmacological effect of Voyager's current and future product candidates; Voyager's ability to develop and validate its brain-targeted capsids in humans, and its viral and non-viral platforms; Voyager's ability to generate near-term and long-term funding under its existing and future licensing and collaboration agreements; Voyager's estimates regarding the market opportunity of its product candidates; the mission, goals and value drivers for its business; and Voyager's cash runway and the sufficiency of Voyager's cash resources. These forward-looking statements are only predictions, and Voyager may not actually achieve the plans, intentions, or expectations disclosed in the forward-looking statements. The use of words such as "anticipate," "believe," "potential," "contemplate," "continue," "could," "estimate," "expect," "goals," "intend," "may," "might," "plan," "potential," "predict," "project," "seek," "should," "target," "will," or "would" and other similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words.

All forward-looking statements are based on management's current estimates and assumptions and are subject to a number of risks, uncertainties and important factors that may cause actual results to differ materially from any forward-looking statements in this presentation. Factors include, among others, the risks and uncertainties inherent in the development of product candidates, including the timing, initiation, enrollment, cost, and conduct of preclinical studies and clinical trials, including potential delays in timing as a result of slower than expected site initiation, slower than expected enrollment, the need or decision to expand the trials or other changes, which may impact its ability to meet its expected timelines and may increase its costs; the expectations and decisions of regulatory authorities; the availability of data from and outcomes of Voyager's preclinical studies and clinical trials and those conducted by its partners and collaborators, including that success in earlier preclinical studies may not be repeated or observed in ongoing or future preclinical studies or clinical trials, ongoing and future clinical trials may not meet their primary or key secondary endpoints, which may substantially impair development, and Voyager may encounter adverse events that could negatively impact further development; Voyager's ability to demonstrate that current or future product candidates are safe and effective for their proposed indications; the availability, commercial potential and success of Voyager's wholly owned candidates; the availability of data from and the outcomes of third-party preclinical studies and clinical trials and the potential impact on Voyager's development plans; the continued development of Voyager's technology platforms, including Voyager's TRACER and nonviral discovery platforms; Voyager's scientific approach and program development progress and the restricted supply and increased costs of critical research components; the development by third parties of capsid and non-viral identification platforms that may be competitive to Voyager's TRACER capsid and nonviral discovery platform and programs; Voyager's ability to create and protect intellectual property rights associated with the TRACER capsid and nonviral discovery platforms, the capsids and ligands identified by the platforms, and the development of clinical candidates and related data from Voyager's pipeline programs; the willingness and ability of Voyager's collaboration partners to meet obligations under collaboration agreements with Voyager and their projections with respect to such programs; the need to align with its collaborators, which may hamper or delay its development efforts and timelines; the possibility or timing of Voyager's receipt of program reimbursement, development or commercialization milestone payments, option exercise, and other payments under Voyager's existing licensing or collaboration agreements; the success of programs controlled by third-party collaboration partners in which Voyager retains a financial interest, including that the anticipated benefits of these ongoing collaborations, including the receipt of payments or the successful development or commercialization of products and generation of revenue, may never be achieved at the levels or timing Voyager expects or at all; the adverse impact on its business if any of its key collaborators fails to perform its obligations or terminates its collaboration; the ability of Voyager to negotiate and complete licensing or collaboration agreements with other parties on terms acceptable to Voyager and the third parties; additional funding may not be available on acceptable terms when needed, or at all, which could hamper its development efforts; Voyager's expected succession plan; the ability to attract and retain talented directors, employees, and contractors and the resulting impact to its business and ability to meet its goals and timelines; the sufficiency of Voyager's cash resources to fund its operations and pursue its corporate objectives; any of the foregoing events could impair the drivers and value creation opportunities for its business; and technical and other unexpected hurdles in the development, manufacture and supply of product candidates, may delay its timing, change its plans, increase its costs, or otherwise negatively impact its business. These risks and uncertainties are described in Voyager's most recent Annual Report on Form 10-K filed with the Securities and Exchange Commission, as updated by its subsequent filings with the Securities and Exchange Commission. All information in this presentation is as of the date of this presentation, and any forward-looking statement speaks only as of the date on which it was made. Voyager undertakes no obligation to publicly update or revise this information or any forward-looking statement, whether as a result of new information, future events or otherwise, except as required by law. © Voyager Therapeutics, Inc.

Voyager (NASDAQ: VYGR) 2026 Investment Rationale

Transformative year for tau, with two shots on goal

Knockdown: VY1706 first-in-human dosing expected in next few months; one-time IV tau silencing gene therapy for AD
Antibody: VY7523 data in AD patients expected Q4 '26

Validating brain-targeted capsids in humans

VY1706 for AD first-in-human dosing expected next few months

NBIB-'223 for Friedreich's ataxia (NBIX-partnered) expected to enter Phase 1 clinical study in coming months with initial patient-level data anticipated in 2027; dual-targeting of brain and heart

Demonstrating the value of Voyager NeuroShuttle™

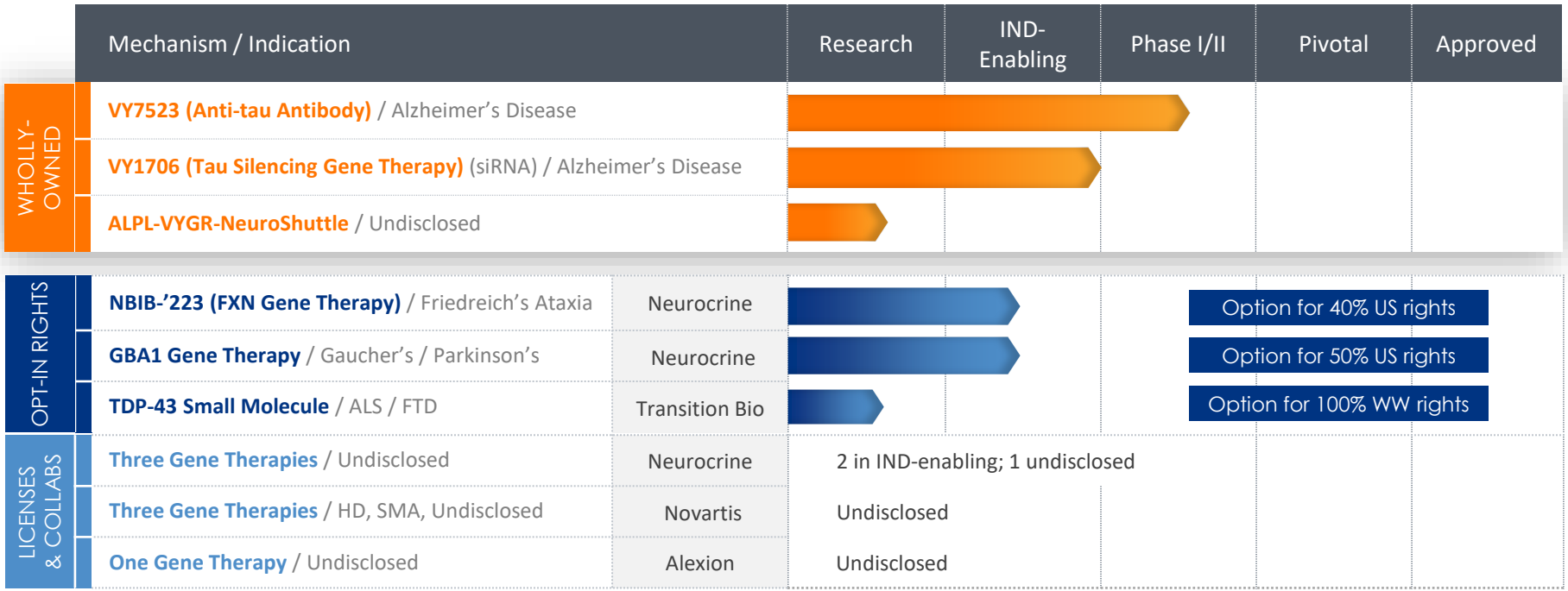
Sustained brain delivery vs TfR shuttles; shuttled antibody showed superior target engagement vs unshuttled[1,2]

2026 data: NHP translatability, safety, program advancement

VY1706 initial acute safety data expected Q1 '27[3], and initial CSF tau biomarker-based data ~1yr after dosing[3]; cash runway into Q2 2028[4]

1. Data on file. 2 Data on file. 3. Pending enrollment progress and timing. 4. Anticipated runway based on our current operating plans, cash and cash equivalents and marketable securities as of November 5, 2026, along with amounts expected to be received as reimbursement for development costs under the Neurocrine and Novartis collaborations, and interest income.

Multi-Modality CNS Pipeline with High Potential Value



Transformative Year for Tau – Voyager Has Two Approaches



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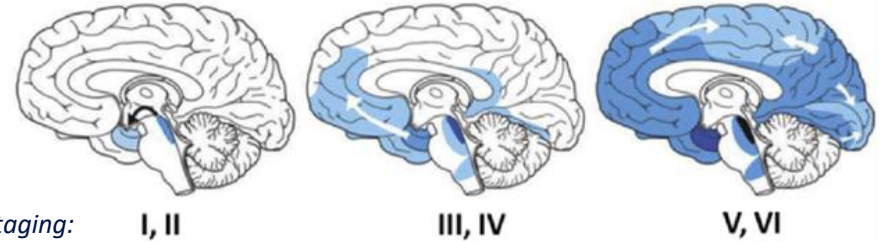
Tau Critical in AD; Biological Rationale + Growing Clinical Dataset

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Biological Rationale

Tau Spread = Disease Worsening

Spread of pathological tau closely correlates with Alzheimer's Disease progression



Adapted from Jouanne M, Rault S, Voisin-Chiret AS. Eur J Med Chem. **2017** Oct 20;139:153-167. Darker shading indicates increased tau pathology.

Third-Party Clinical Data

BIIB080
(Knockdown, intracellular tau)

Phase 2 (CELIA): **Reduced tau pathology and slowed cognitive decline;** missed primary endpoint CDR-SB[1]

Bepranemab
(Antibody, extracellular tau)

Phase 2a (TOGETHER): **Slowed rate of tau accumulation and cognitive decline;** missed primary endpoint CDR-SB[2]

1. Mummery C. et al. Topline Results from CELIA: A Phase 2 Study to Evaluate the Tau-Targeting ASO Diranersen (BIIB080) in Patients with Early Alzheimer's Disease. AAIC 2026. 2. Barton M, et al. Results from TOGETHER, a double-blind, placebo-controlled Phase II study evaluating efficacy, safety and tolerability of bepranemab in prodromal–mild AD. CTAD 2024.

Voyager Advancing Two Approaches to Targeting Tau for AD

VY1706



Gene therapy targeting intracellular tau

- **Differentiation:** One-time, I.V. administration designed to durably reduce tau in the brain
- **Data:** Dosing expected in next few months, pending enrollment
- **Commercial feasibility:** Early payer research indicated willingness to cover[1]; COGS reduced via tech ops[2,3,4]

VY7523



Anti-tau antibody targeting extracellular tau

- **Differentiation:** Targets unique epitope in C-terminal region; highly specific for pathological tau[2]
- **Data:** Clinical efficacy data expected Q4 ’26 from tau PET imaging in AD patients
- **Commercial path** laid by anti-amyloid antibodies; no ARIA observed to date with anti-tau antibodies[4]

1. Data on file. 2. Ibe-Enwo A, et al. Exploiting an AAV Capsid Specific Receptor to Develop Stable Cell Lines for Transduction Based Assays for Gene Therapies. ASGCT 2026. 3. Schrock, A. et al. Optimized Transfection Platform with Improved Productivity and Transgene Packaging for Scalable rAAV Production. ASGCT 2026. 4. Guzman, J. et al. Evaluating Affinity Chromatography Media for Capture of Novel Blood-Brain-Barrier Penetrant AAV Capsids. ASGCT 2026. 4. Cai W, et al. Comparative efficacy and safety of Gosuranemab, Semorinemab, Tilavonemab, and Zagotenemab in patients with AD. Front Aging Neurosci. 2025;16:1465871. doi:10.3389/fnagi.2024.1465871.

VY1706: Expect Dosing Next Few Months

VY1706 = tau-targeted miRNA + IV-delivered, BBB-targeted AAV capsid

VY1706 IND Cleared: First known FDA IND clearance for a gene therapy designed to treat AD by reducing tau protein production in the brain[1]; Canadian CTA also cleared

Multi-site, open-label, dose-escalation study of VY1706 administered as one-time IV dose in up to 18 adults with early AD

Dosing	Three cohorts; highest dose not to exceed 5E13 vg/kg (highest GLP tox study dose)
Primary endpoint	Safety and tolerability
Secondary endpoints	Effect on tau biology, including changes in CSF biomarkers of tau, and changes in tau pathology measured by tau PET imaging

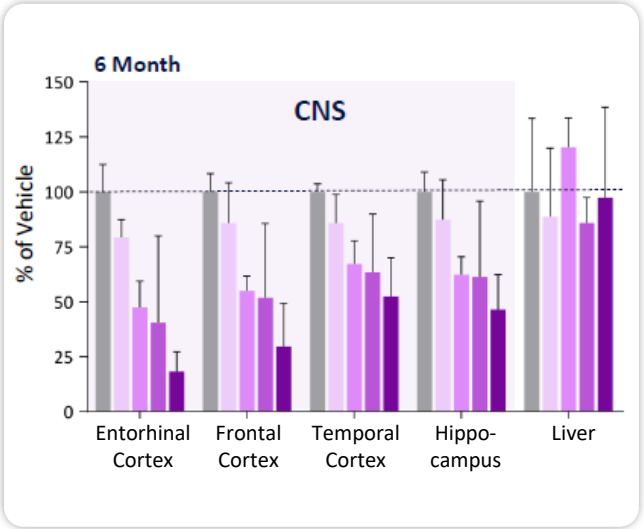


- **Next few months:** First-in-human dosing expected[2]
- **Q1 2027:** Initial acute safety data[2]
- **~1 year post-dosing:** Initial CSF tau biomarker-based data to support capsid platform validation[2]

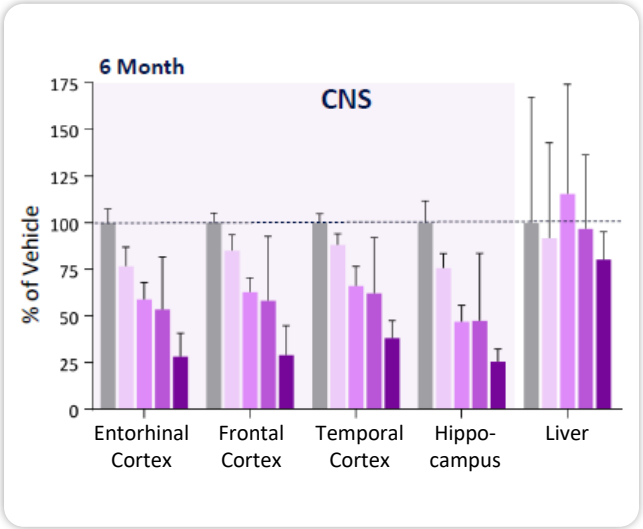
1. Data on file. 2. Pending enrollment progress and timing.

VY1706: Significant and Durable Tau Reductions in 6-Month GLP Tox Study

Up to 75% Reduction of Tau mRNA



48-64% Reduction of Tau Protein



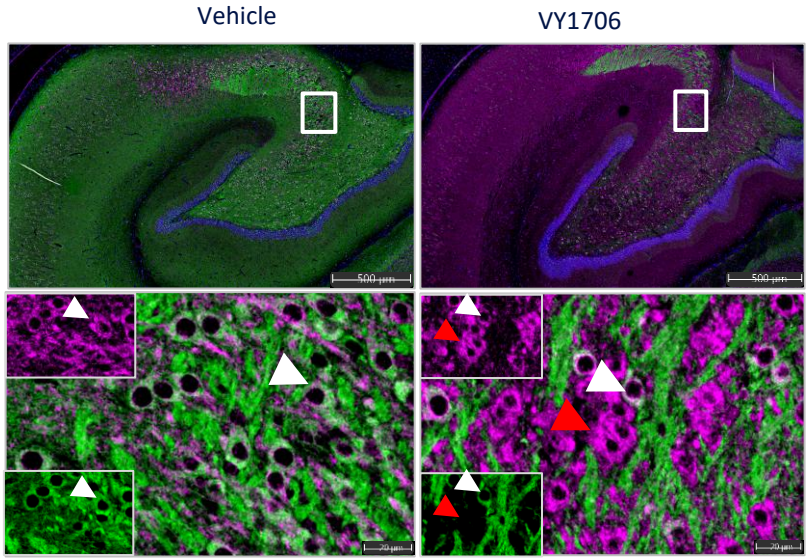
IND-enabling GLP study of VY1706 featured in late-breaking presentations at ASGCT 2026 and AAIC 2026

- No adverse pathology or histopathological findings observed up to the highest dose tested (5E13 vg/kg).

Vehicle | Low Dose | Mid Dose 1 | Mid Dose 2 | 5E13 vg/kg

Arora, V., et al. IND-Enabling GLP NHP Study of VY1706, a BBB-Crossing AAV Gene Therapy Targeting Tau in Alzheimer's Disease. AAIC 2026.

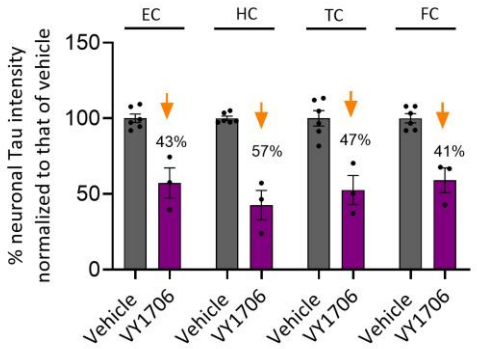
VY1706: Robust Neuronal Tau Knockdown Across AD-Relevant Brain Regions in Additional NHP Study



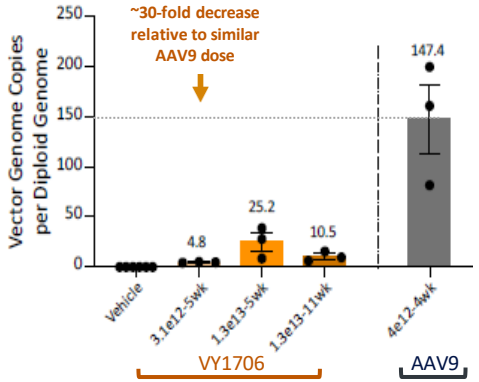
Tau Protein Expression in SMI311^{+ve} Neurons at 11 weeks: **SMI311 (neurons); TAU protein; DAPI (nuclear)**[1]

Single IV dose of 1.3e13 VG/KG; 11-week time point in NHP; non-GLP DRF study;
EC: Entorhinal Cortex; HC: Hippocampus; TC: Temporal Cortex; FC: Frontal Cortex

Neuronal Tau protein lowering[1]



VY1706 de-targets the liver[2]



1. Sivasankaran, R. et al. Intravenous Delivery of VY1706, a CNS Penetrant AAV Gene Therapy for Tauopathies, Provides Broad Tau Lowering in Mouse Models of Tauopathy and Non-Human Primates. ADPD 2026.

VY7523: Tau PET Imaging Data in AD Patients Expected Q4 2026

VY7523 single ascending dose (SAD) trial:

- 48 healthy volunteers
- No serious/severe adverse events or infusion reactions reported
- Half-life supports monthly dosing
- Serum concentrations increased in dose-proportionate manner; CSF-to-serum ratio 0.3%

MAD STUDY OBJECTIVES AND ENDPOINTS (NCT06874621)

Multiple Ascending Dose (MAD) Study of VY7523 in Early Alzheimer's Disease (AD)

	OBJECTIVES	ENDPOINTS
1	**Primary**	
	To characterize VY7523 safety and tolerability	• Incidence of treatment-emergent adverse events (TEAEs) • Clinically significant changes from baseline vital signs, electrocardiograms (ECGs) and clinical and laboratory parameters
2	**Secondary**	
	To characterize VY7523 pharmacokinetics (PK) in serum and determine cerebrospinal fluid (CSF) concentrations	• Serum concentrations at specified timepoints • PK parameters • CSF concentrations
	To evaluate VY7523 ability to prevent the spread of pathologic tau	• Changes from baseline in the standardized uptake value ration (SUVR) using tau-positron emission tomography (PET)
	To evaluate VY7523 immunogenicity	• Incidence of treatment emergent anti-drug antibodies (ADAs)

Last Patient Visit Complete in MAD Trial

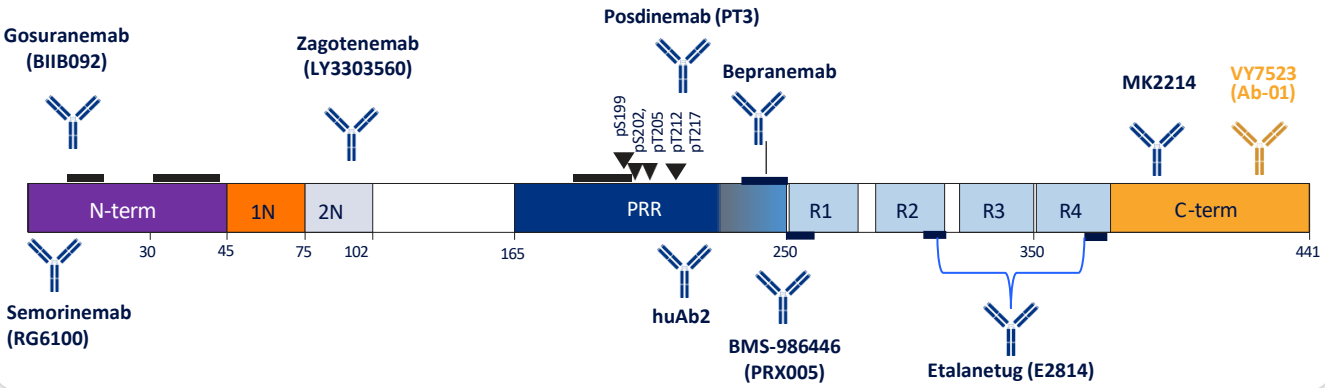
Tau PET Imaging Efficacy Data Expected Q4 '26

1. Ratti E. et al. Results of a Phase 1 study to evaluate the safety, tolerability, and pharmacokinetics of ascending single IV doses of VY7523, a tau-targeting therapeutic, in healthy adult participants. CTAD 2025, 2. Butler, et. al. Study Design of a Multiple Ascending Intravenous Dose Study Evaluating the Safety, Tolerability, and Pharmacokinetics of VY7523 in Participants with Early AD. CTAD 2025.

VY7523 Differentiated from Other Anti-Tau Antibodies

VY7523 = antibody targeting epitope in C-terminal, highly specific for pathological tau

Epitope Matters[1,2,3]:



Screened over 700 antibodies across N-terminal, PRR (including pT217), and C terminal

Chose VY7523 based on performance in tau seeding in vivo model

VY7523 targets C-terminal epitope distinct from other antibodies

Pathological Specificity May Matter:

Antibody	ePHF Binding Potency EC50 (pM)	WT-tau Binding Potency EC50 (pM)
Ab-01 (murine VY7523)	55	undetectable
Ab-D (murine bepranemab)	47	11
posdinemab	50	8760

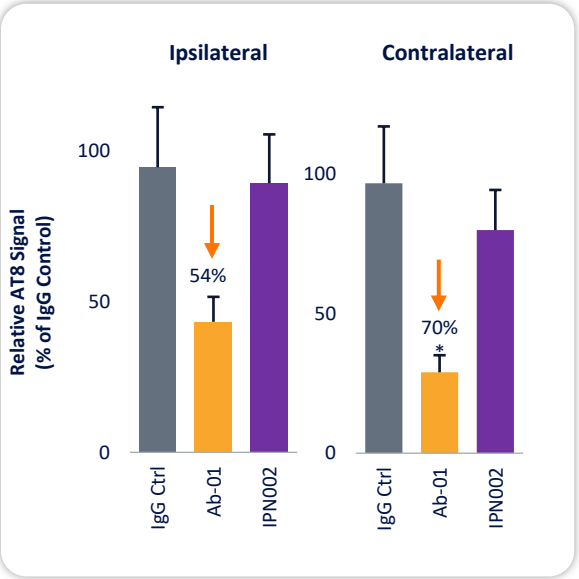
With anti-amyloid antibodies, lack of specificity for pathologic form resulted in lack of potency / peripheral sink[4]

VY7523 is highly specific for pathological tau

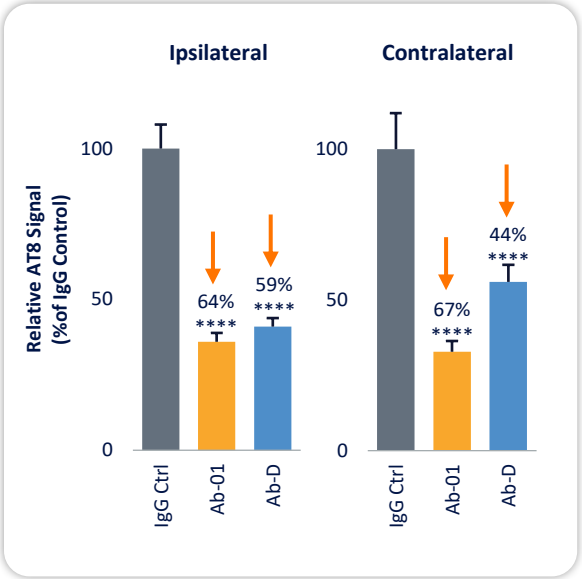
1. Davidson, R.; Krider, R.I.; Borsellino, P.; Noorda, K.; Alhwayek, G.; Vida, T.A. Untangling Tau: Molecular Insights into Neuroinflammation, Pathophysiology, and Emerging Immunotherapies. Curr. Issues Mol. Biol. 2023, 45, 8816-8839. https://doi.org/10.3390/cimb45110553. 2. ADPD 2025, Liu, et al.. 3. huAb2 is a murinized research antibody that targets the p212/p214/p217 region, similar to posdinemab. 4. Arndt, J.W., Qian, F., Smith, B.A. et al. Structural and kinetic basis for the selectivity of aducanumab for aggregated forms of amyloid-β. Sci Rep 8, 6412 (2018). https://doi.org/10.1038/s41598-018-24501-0

VY7523: Murine P301S Data Differentiated from Posdinemab, Bepranemab

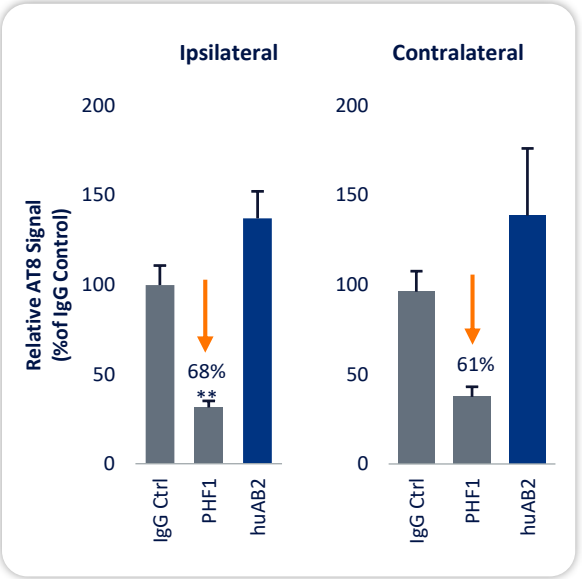
VYGR antibody reduced tau spread; N-terminal antibody did not



VYGR antibody reduced tau spread; bepranemab did also



huAb2 antibody binding similar epitope as posdinemab did not reduce tau spread



P301S murine model; all antibodies represent murine versions. Ab-01 is murine VY7523; IPN002 is murine BIIB092; Ab-D is murine bepranemab; huAb2 is a murinized research antibody that targets the p212/p214/p217 region, similar to posdinemab. PHF1 is a positive control antibody.

Liu, et al. Discrimination of Anti-tau Antibodies Targeting Different tau Epitopes by a P301S Mouse Hippocampal Seeding Model of Tauopathy. ADPD 2025. *, **, *** and **** indicate $p < 0.05$, 0.005, 0.0005 and 0.0001, respectively, compared to the vehicle control group.

Validating Brain-Targeted Capsids in Humans



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Establishing Novel Capsid Platform Validation in Humans

STEP 1: Acute Safety

Determine if dosing in first few patients warrants continuation

STEP 2. Capsid Platform Validation

Biomarker-based assessment (i.e. CSF/tau) to confirm capsid was delivered to brain

STEP 3. Asset Proof-of-Concept

Tau PET imaging to confirm impact on tau and suggest possible efficacy (for VY1706)

Two Opportunities for First-in-Human Dosing:

VY1706

- First-in-human anticipated next few months[1]
- Wholly-owned
- Tau knockdown for Alzheimer's disease

NBIB-'223

- Received FDA orphan drug designation
- Expected to enter Phase 1 clinical study in coming months with initial patient-level data anticipated in 2027
- NBIX partnered (VYGR has opt-in for 40% US)
- FXN replacement for Friedreich's ataxia

1. Pending enrollment progress and timing.

Leading Indications for 2026 Gene Therapy Trials

Alzheimer's Disease

Impacts approximately **7M Americans** and is expected to impact nearly **13M** by 2050[1]

VY1706

Potential one-time, I.V. gene therapy designed to knock down tau for AD

U.S. IND and Canadian CTA cleared

Expert KOL Ad Board: enthusiasm for VY1706; support for proposed safety and monitoring plan[3]

Early Payer Market Research: Enthusiasm for potential greater disease slowing and durability following single treatment[4]

Friedreich's Ataxia

Impacts approximately **5,000 Americans**[2]

NBIB-'223

Potential one-time, I.V. gene therapy designed to replace FXN in brain and heart for FA

Granted FDA orphan drug designation

All VYGR-NBIX partnered gene therapy programs continue to progress, including the GBA1 program for Gaucher and Parkinson's disease.

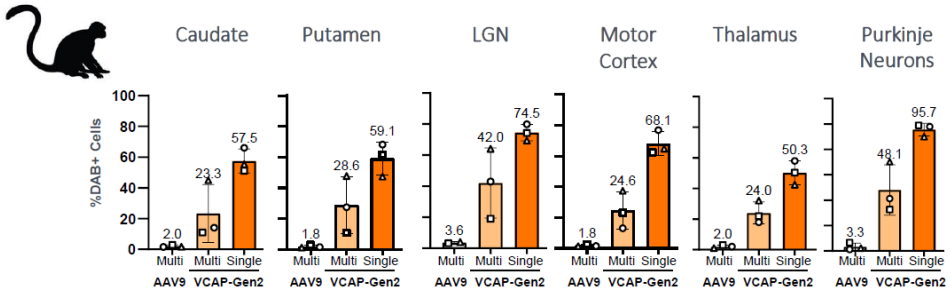
1. Alzheimer's Association; https://www.alz.org/alzheimers-dementia/facts-figures (accessed July 2025). 2. Friedreich's Ataxia Research Alliance (FARA). What is FA? Available at: https://www.curefa.org/what-is-friedreichs-ataxia. Accessed: May 2024. 3. Data on file. 4. Data on file.

TRACER™-Derived Capsids to Power Next-Gen Gene Therapy

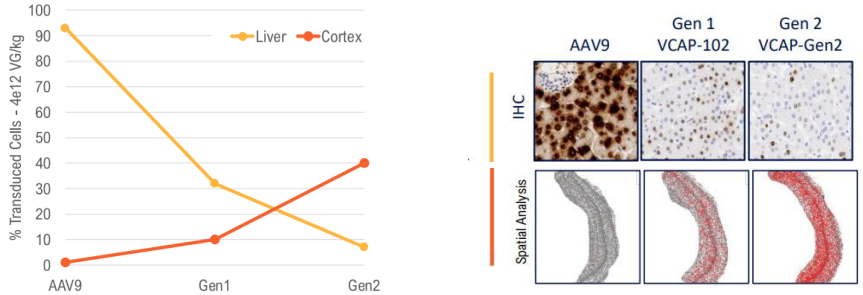
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TRACER capsid discovery platform derived from evaluation of 40M variants of AAV9[1]

VCAP-Gen2: 50-75% Cells Transduced Across Diverse Brain Regions at 3E13 vg/kg[2]



Gen2 Capsids: Increased Brain Tropism and Liver Detargeting[2]



Minimally invasive **I.V. Delivery**

Customizable cell tropisms (neurons, glial cells) and levels of liver de-targeting

Receptor identification enables rational design

Multi-Species Validation

Improved, broad **CNS transduction**

Fully integrated: capsid engineering, NHP in vivo validation, scalable production (HEK, Sf9)

Moyer T. et al. Highly conserved brain vascular receptor ALPL mediates transport of engineered AAV vectors across the blood-brain barrier. Molecular Therapy, 2025; 33, 3902-3916. 2. Moyer T et al. Continued directed evolution of VCAP-101 and VCAP-102 identifies second generation capsids with increased brain tropism in nonhuman primates and mice. ASGCT 2024.

Voyager NeuroShuttle™ Nonviral Platform



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VYGR-NeuroShuttle: Next-Gen Approach to Neurotherapeutics

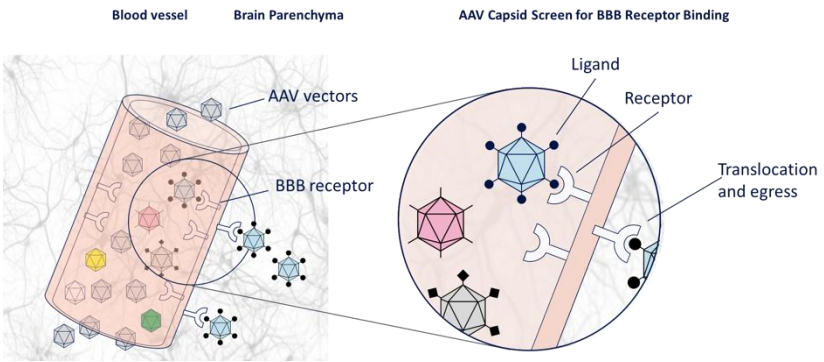
voyager NeuroShuttle™

Voyager NeuroShuttle™ (VYGR-NeuroShuttle) is a non-viral platform leveraging novel receptor-binding molecules to transport multiple modalities of neurotherapeutics across the blood-brain barrier (BBB).

TRACER™ AAV capsid discovery platform → Capsid family crosses BBB → Receptor ID: **ALPL** → ALPL ligands identified

ALPL-VYGR-NeuroShuttle

TRACER engine for receptor discovery



Handful of additional receptors identified

Potential to deliver a diversity of therapeutic modalities:

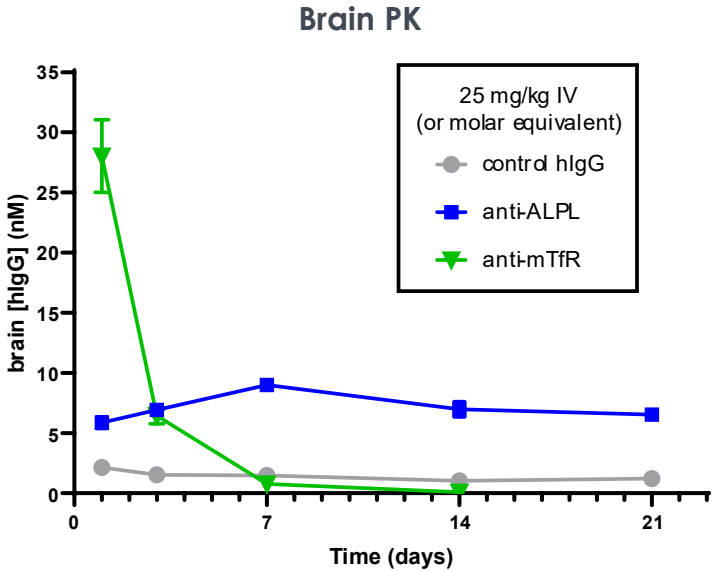
- Antibodies
- Enzymes
- Genome editors
- ASOs
- siRNAs
- Peptides

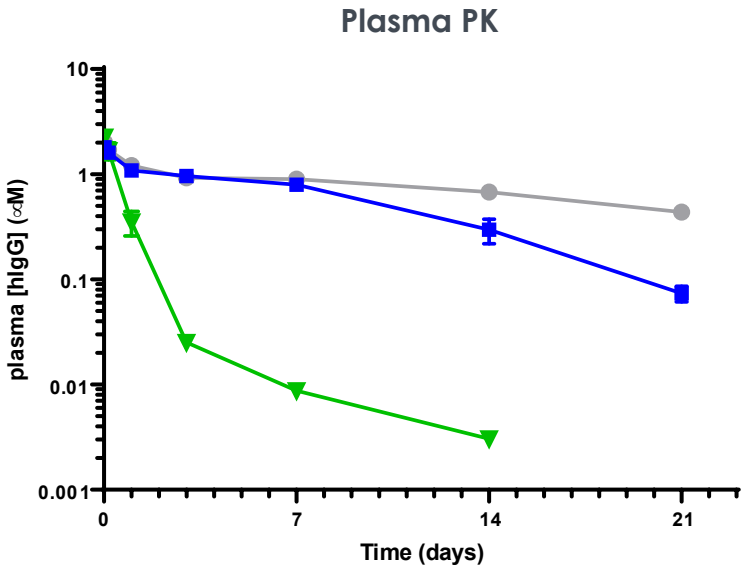
ALPL-VYGR-NeuroShuttle Demonstrated Sustained Brain Exposures

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Proof of concept murine data[1] show sustained CNS exposure for ALPL versus TfR shuttles

- Increased antibody concentration in brain; modest impact on peripheral clearance
- Brain uptake sustained for >3 weeks post-dose (vs <1 week for TfR shuttles)



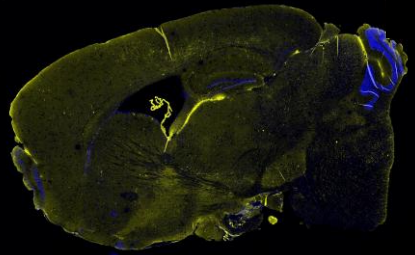


1. Voyager data on file.

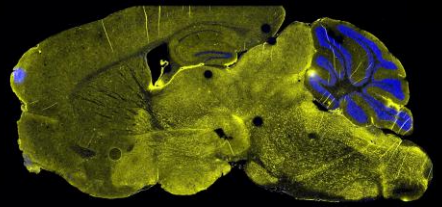
ALPL-VYGR-NeuroShuttle: Broad Biodistribution Including Neurons

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Greater parenchymal distribution observed with shuttle vs. isotype control in mouse



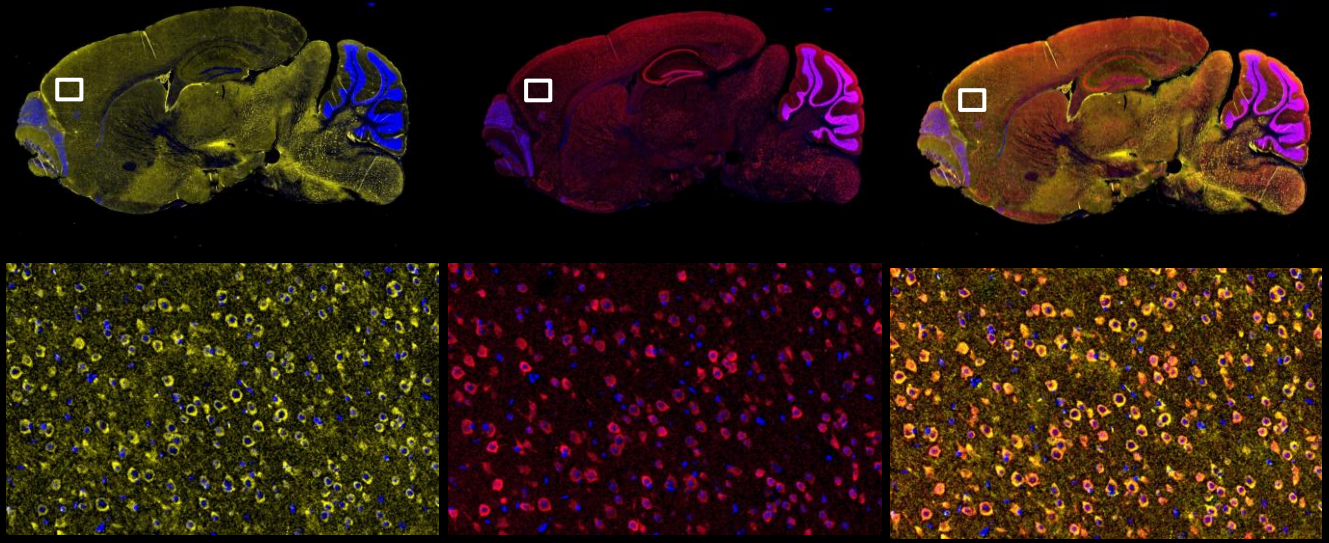
Isotype Control



Shuttle

Anti-human IgG
DAPI

Colocalization of shuttle and neurons seen across brain regions in mouse including cerebral cortex



Anti-human IgG
DAPI

NeuN
DAPI

Anti-human IgG
NeuN
DAPI

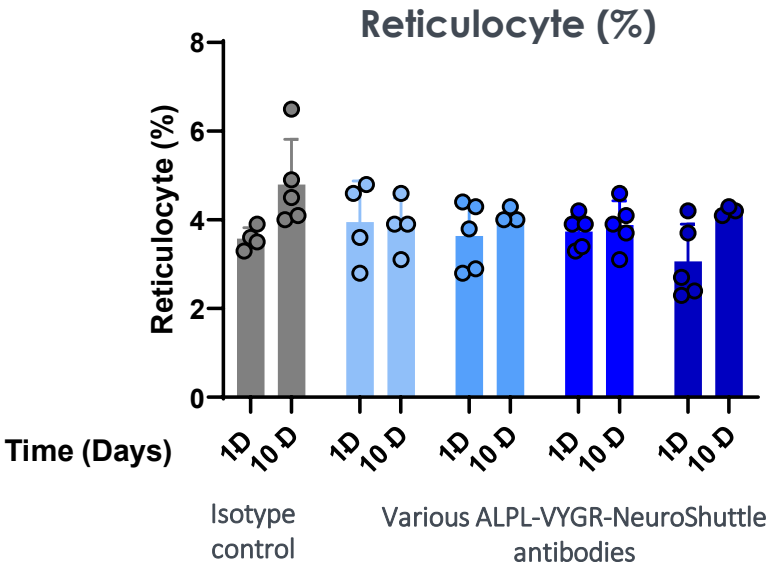
ALPL-VYGR-NeuroShuttle Not Observed to Impact Reticulocytes

Proof of concept murine data show negligible anemia risk for ALPL

ALPL-VYGR-NeuroShuttle technology did not impact reticulocytes in mice; potential reduced anemia risk[1]

ALPL plays a key role in skeletal and dental mineralization, liver function, and neurotransmitter synthesis.[2]

- Decreased levels of ALPL (~30% residual activity) can result in bone hypomineralization or cardiovascular complications.[3]
- Genetic data indicate partial loss of function is tolerable.[3]
- Preclinical studies ongoing to identify potential safety profile of ALPL-VYGR-NeuroShuttle.

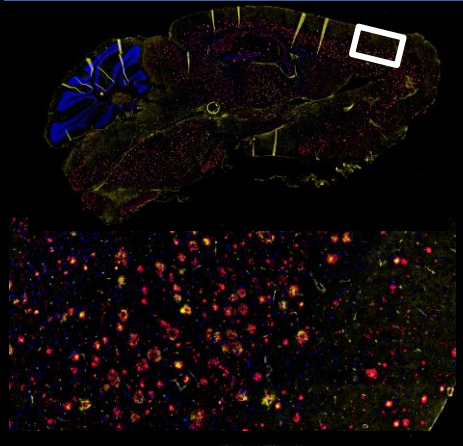
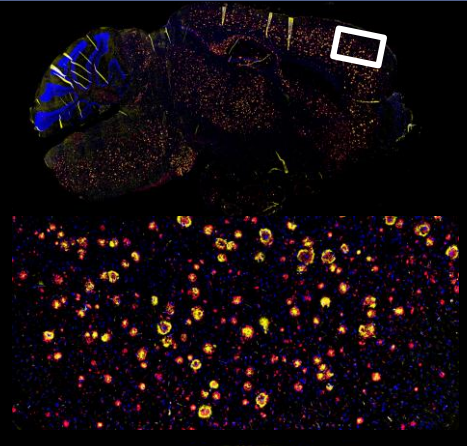
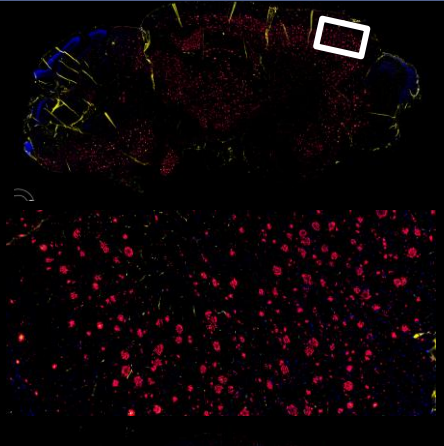
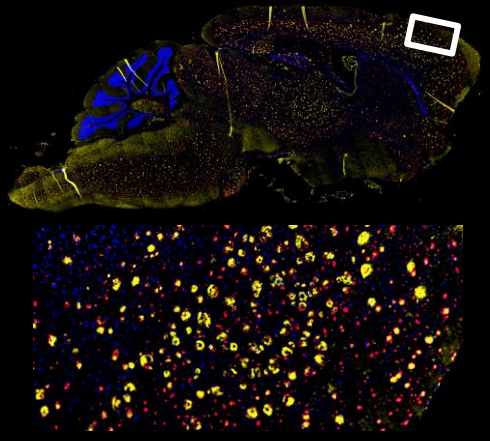
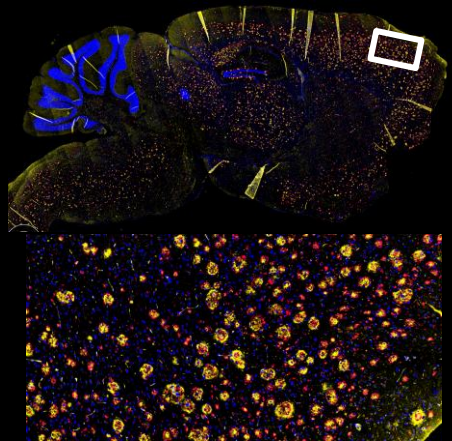
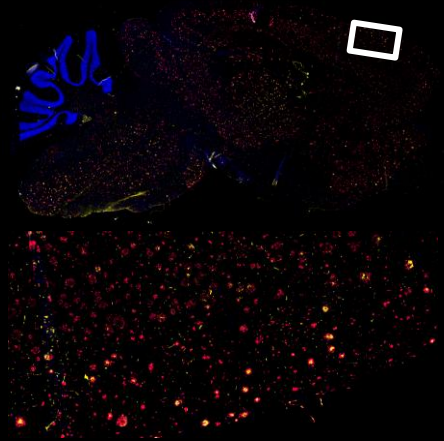


1. Voyager data on file. 2. Goettsch et al. Cardiovascular Research. 2022. 118:84-96. 3. Farman MR, et. al., The ALPL gene variant project: results of the first 100 reclassified variants. JBMR Plus. 2025 Mar 17;9(6):ziaf044. doi: 10.1093/jbmrpl/ziaf044. PMID: 40386289; PMCID: PMC12083982.

ALPL-Shuttled Amyloid Ab Demonstrated Target Engagement

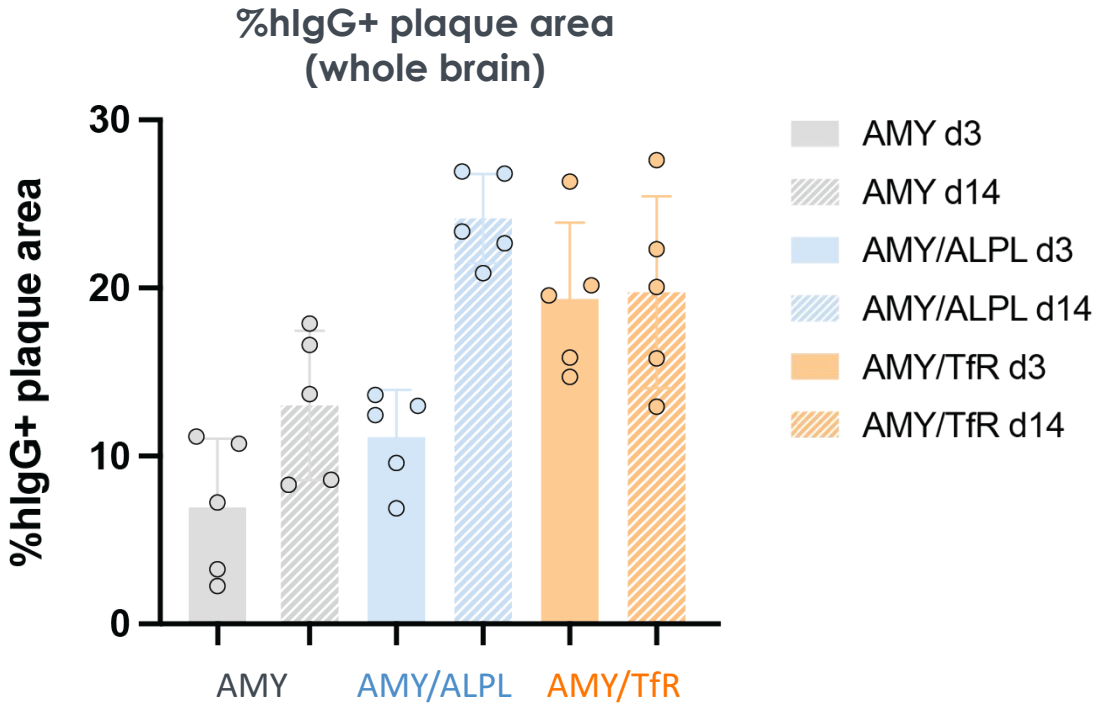
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DAPI
hIgG
Aβ

	AMY/ALPL bispecific 10mg/kg	AMY/TfR bispecific 10mg/kg	AMY 10mg/kg
Day 3			
Day 14			

- AMY/ALPL bispecific showed continued uptake in brain between d3 and d14 and is localized to amyloid plaques
- AMY/ALPL bispecific showed comparable target engagement to AMY/TfR at d14
- Both bispecifics showed enhanced distribution to brain vs. un-shuttled AMY

AMY-ALPL Plaque Binding Similar to AMY-TfR After Single IV Dose



- Both ALPL and TfR groups showed greater %hIgG+ plaque area vs. un-shuttled AMY at d14
- Clear increase in %hIgG+ plaque area observed from d3 to d14 in AMY/ALPL groups

Market + Acquirers Have Valued Shuttle Opportunities

All of these deals involve TfR-shuttled platforms and programs (75 orgs have filed TfR patents)[1]

Voyager's new receptors have the potential to reshape the shuttle technology space

Select M&A / Licensing Deals in the Shuttling Technology Space[2-9]

Partner	Target	Value ($mm)	# of Asset(s)	Indication	Phase
NOVARTIS	AVIDITY BIOSCIENCES	$12,000	3 + Platform	Neuromuscular Disease	Registration
GSK	ablbio	$2,720	Platform	Neurodegenerative Disease	Preclinical
Roche	Manifold Bio	$2,000	Platform	Neurology	Preclinical
NOVARTIS	SCINEURO	$1,700	Platform	Alzheimer's Disease	Preclinical
abbvie	ALIADA	$1,400	1 + Platform	Alzheimer's Disease	Phase I
Bristol Myers Squibb	BIOARCTIC	$1,350	2	Alzheimer's Disease	Preclinical
sanofi	ablbio	$1,060	1	Parkinson's Disease	Preclinical
NOVARTIS	BIOARCTIC	$800	1	Neurodegenerative Disease	Preclinical

M&A Deals

voyager NeuroShuttle™

Platform optimization ongoing in rodents and non-human primates.
Potential for additional value creating partnerships.

1. Data on file. 2. Novartis / Avidity: PR 3. GSK / ABL Bio: PR (GBP / USD Fx rate = 1.2638 as of deal announcement date). 4. Roche / Manifold Bio: PR. 5. Novartis / SciNeuro: PR. 6. AbbVie / Aliada: PR. 7. BMS / BioArctic: PR. 8. Sanofi / ALB Bio: PR 9. Novartis / BioArctic: PR

Business



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BD Opportunities to Transform Treatment of CNS Diseases



Alzheimer's Franchise

» Tau antibody (Ph 1/2) » Tau gene therapy (FIH expected next few months) » Discovery-stage assets



Rare Neurogenetic Diseases

» Out-licensing capsids for rare CNS targets » In-licensing early-stage assets for non-GTX targets



CNS Delivery

» IV TRACER gene therapies moving toward clinic[1] » Voyager NeuroShuttle platform emerging

[1] Includes wholly-owned and partnered programs.

Partnerships Have Brought $500M+; Potential for Another $6.8B

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	Disease/Target	Patients Potentially Impacted (U.S.)	Upfront / Option / License Payments	Potential Development Milestone Payments[1]	Potential Sales Milestone Payments[1]	Tiered Royalties
NEUROCRINE® BIOSCIENCES	**NBIX1:** FA	FA: ~5,000 pts[5]	$165M	$190M[2]	$550M[2]	**NBIX1**: U.S. high-single-digit to high-teens; ex-U.S. mid-single-digit to mid-teens[2]
	NBIX2: GBA1 + 3 targets	Gaucher: ~6,000 pts[9] GBA1 PD: ~100,000 pts[6]	$175M	$1.5B[3]	$2.7B[3]	**NBIX2**: GBA, U.S. low double-digit to twenty; ex-U.S. high single-digit to mid-teen. 3 targets, U.S. high single-digit to mid-teen; ex-U.S. mid single-digit to low double-digit[3]
NOVARTIS	**NVS1:** 1 CNS target	Not disclosed	$94M	$125M	$175M	**NVS1:** Mid- to high-single-digit
	NVS2: HD + SMA	HD: ~30,000 pts[7] SMA: ~10,000-25,000 pts[8]	$100M[4]	$425M	$775M	**NVS2:** High-single-digit to low-double-digit tiered royalties on global net sales.
ALEXION AstraZeneca Rare Disease	1 rare neurologic disease target	Not disclosed	$40M	$115M	$175M	Mid- to high-single-digit
				$2.4B	**+ $4.4B**	**= $6.8B + royalties**

1. Potential milestone payments represent maximum potential payments under applicable agreement(s).2. After the Phase 1 readout, Voyager has the option to either: (1) co-develop and co-commercialize with Neurocrine Biosciences in the U.S. under a 60/40 cost- and profit-sharing arrangement (Neurocrine/Voyager), or (2) permit Neurocrine Biosciences to retain full U.S. commercial rights in exchange for milestone payments and royalties based on U.S. sales. Assumes 2 FA collaboration products; totals may not add due to rounding. 3. After the Phase 1 readout, Voyager has the option to either: (1) co-develop and co-commercialize GBA1 with Neurocrine Biosciences in the U.S. under a 50/50 cost- and profit-sharing arrangement, or (2) permit Neurocrine Biosciences to retain full U.S. commercial rights in exchange for milestone payments and royalties based on U.S. sales. 4. NVS2 $100 million payment consists of $80 million in cash and $20 million equity investment. 5. Friedreich's Ataxia Research Alliance (FARA). What is FA? Available at: https://www.curefa.org/what-is-friedreichs-ataxia. 6. Migdalska-Richards A, Schapira AH. The relationship between glucocerebrosidase mutations and Parkinson disease. Journal of Neurochemistry. 2016 Oct; 139: 77-90. doi: 10.1111/jnc.13385. Epub 2016 Feb 10. 7. https://rarediseases.org/rare-diseases/huntingtons-disease/. 8. https://smafoundation.org/about-sma/ 9. Cleveland Clinic: Gaucher Disease. Available at: https://my.clevelandclinic.org/health/diseases/16234-gaucher-disease. Accessed April 2025.

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Thank You

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