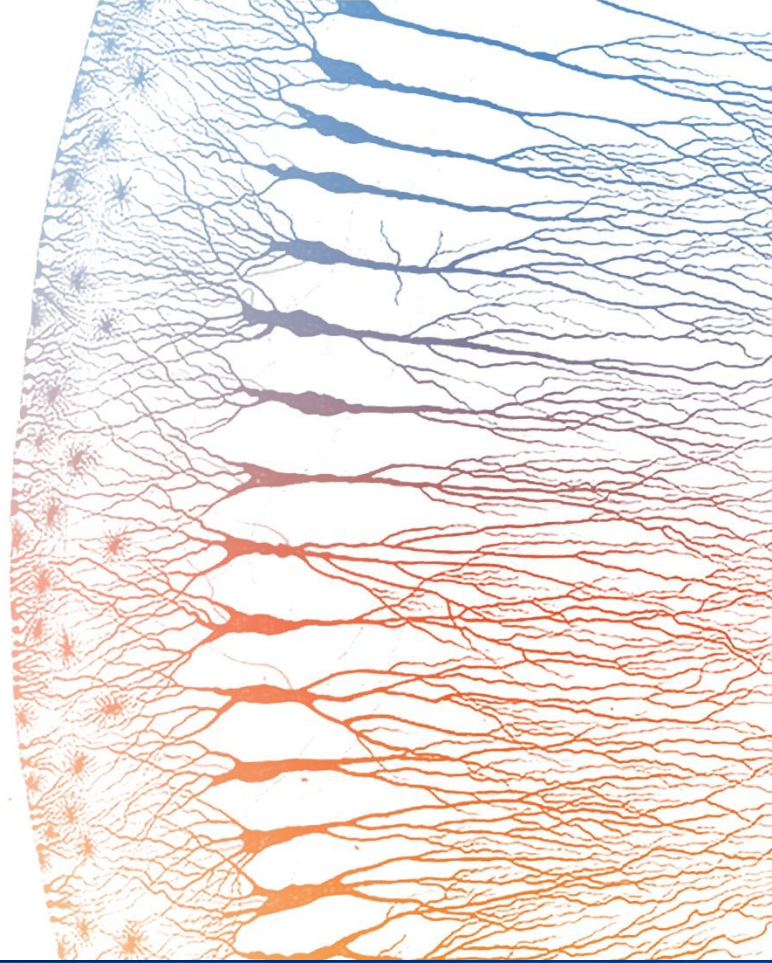
voyager therapeutics

Leveraging Genetics to Treat Neurological Diseases

Corporate Deck / March 2026



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This presentation contains forward-looking statements for the purposes of the safe harbor provisions under The Private Securities Litigation Reform Act of 1995 and other federal securities laws. The use of words such as "anticipate," "expect," "believe," "plan," "estimate," "may," or "potential," and other similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. These forward-looking statements include, among other things, statements about expectations for Voyager's achievement of preclinical and clinical development milestones for its potential development candidates such as the identification of lead development candidates, IND and CTA filings, the initiation of clinical trials and clinical trial enrollment, including achievement of first-in-human dosing for our wholly-owned tau silencing gene therapy in Alzheimer's disease, and the generation of clinical data and proof-of-concept data; the potential for third-party clinical data to inform Voyager's product development programs; Voyager's ability to expand beyond gene therapy and antibodies into other modalities of neurogenetic medicine, including Voyager NeuroShuttles and small molecule therapies; Voyager's ability to generate near-term and long-term funding through reimbursement, upfront, milestone and royalty-based fees (as applicable) under its existing licensing and collaboration agreements, and to obtain data regarding the performance of its TRACER-derived capsid families licensed to its collaborators and partners under such agreements; Voyager's ability to maintain and advance product development programs under its current partnerships and collaborations, including the anticipated submission of an IND and clinical trial initiation by Neurocrine in the FA-partnered program and advancement of development of the four other gene therapy programs partnered with Neurocrine, including the GBA1 program; our estimates regarding the market opportunity of our product candidates, 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.

All forward-looking statements are based on estimates and assumptions by Voyager's management that, although Voyager believes such forward-looking statements to be reasonable, are inherently uncertain and subject to risks and uncertainties that may cause actual results to differ materially from those that Voyager expected. Such risks and uncertainties include, among others, the expectations and decisions of regulatory authorities; the timing, initiation, conduct, and outcomes of Voyager's preclinical studies and clinical trials; our ability to replicate positive results from earlier preclinical studies or clinical trials conducted by us or third parties in current or future clinical trials; the availability of data from internal or third-party clinical trials; the success of Voyager's product candidates; the availability or commercial potential of product candidates under collaborations; the willingness and ability of Voyager's collaboration partners to meet obligations under collaboration agreements with Voyager; the need to align with our collaborators, which may hamper or delay our development efforts and timelines; the continued development of Voyager's technology platforms, including Voyager's TRACER capsid discovery platform and its non-viral discovery platform; 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 and capsids and non-viral ligands that may be competitive to Voyager's discovery platforms; Voyager's ability to create and protect intellectual property rights associated with the TRACER capsid discovery and non-viral platforms, the capsids and ligands identified by the platforms, and development candidates for Voyager's pipeline programs; the possibility and the timing of Voyager's receipt of program reimbursement, development or commercialization milestones, option exercise, and other payments under Voyager's existing licensing or collaboration agreements; the ability of Voyager to negotiate and complete licensing or collaboration agreements with other parties on terms acceptable to Voyager and the third parties; the success of programs controlled by third-party collaborators in which Voyager retains a financial interest; the availability of additional funding on acceptable terms when we need it, or at all; the ability to attract and retain talented directors, employees, and contractors; the sufficiency of Voyager's cash resources and cash runway; any of the foregoing events could impair the drivers and value creation opportunities for our business; and we may encounter technical and other unexpected hurdles in the development and manufacture of product candidates, which may delay our timing or change our plans, increase our costs, or otherwise negatively impact our business. These statements are also subject to a number of material risks and uncertainties that 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. Any forward-looking statement speaks only as of the date on which this presentation was presented. Voyager undertakes no obligation to publicly update or revise any forward-looking statement, whether as a result of new information, future events or otherwise, except as required by law.

Voyager (NASDAQ: VYGR) 2026 Investment Rationale

Transformative year for tau, with two shots on goal

Knockdown: VY1706 first-in-human dosing expected H2 ‘26 following 3rd-party data (BIIB080, expected mid-’26)

Antibody: VY7523 data in AD patients expected H2 ‘26

Validating brain-targeted capsids in humans

VY1706 for AD first-in-human dosing expected H2 ‘26

NBIB-’223 for Friedreich’s ataxia (NBIX-partnered) expected to enter clinic in 2026; dual-targeting of brain and heart

Demonstrating the value of Voyager NeuroShuttle™

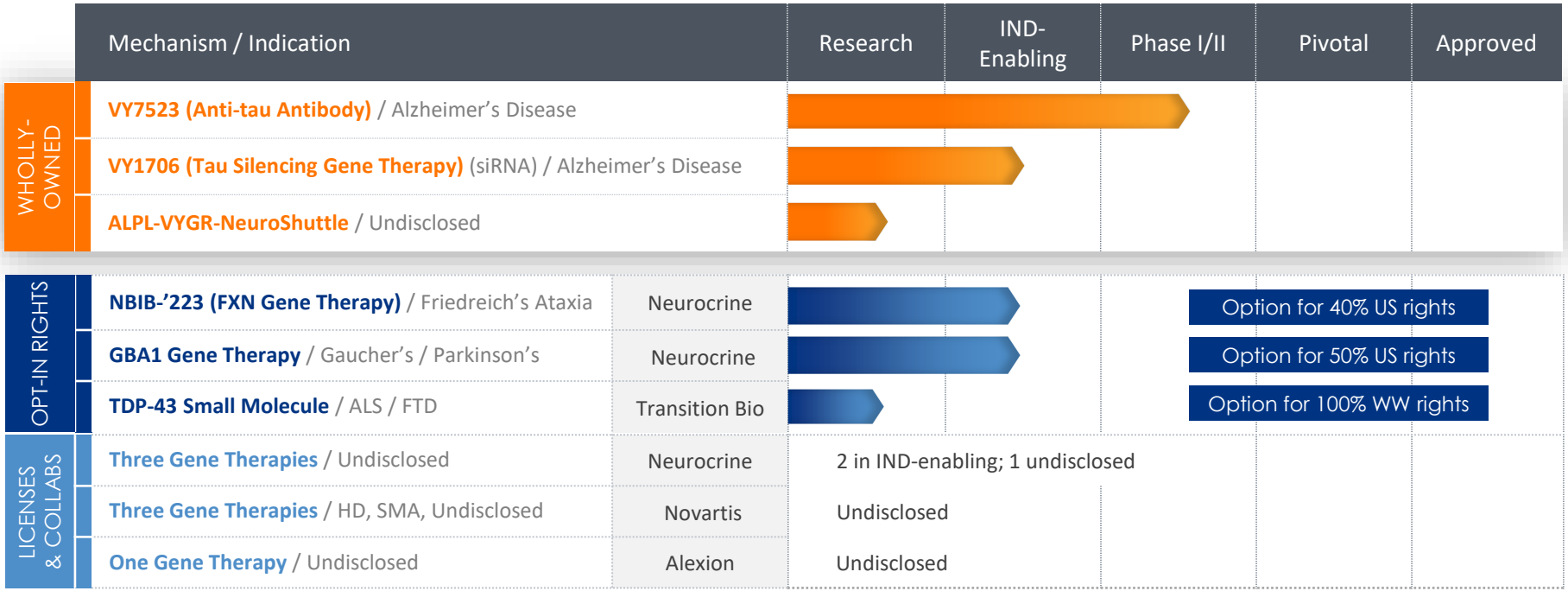
Sustained brain delivery vs TfR shuttles; shuttled antibody has superior target engagement vs unshuttled

2026 data: NHP translatability, safety, program advancement

Cash runway into 2028, not including $6.8B in potential milestone payments from existing partnerships[1]

[1] Based on our current operating plans, cash and cash equivalents and marketable securities as of December 31, 2025, 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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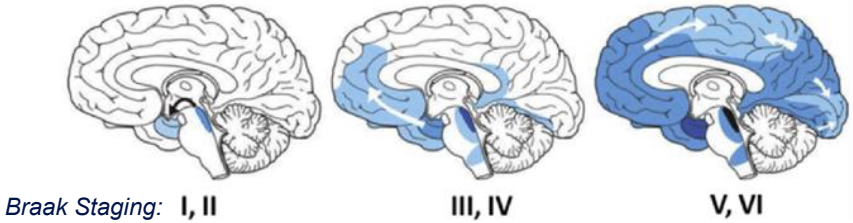
Tau Critical in Alzheimer's Disease; Two Ways to Target

Spread of Pathological Tau Corresponds to Alzheimer's Disease Progression

Tau Spread = Disease Worsening



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

Two Approaches to Targeting Tau

1. Intracellular

Block production of tau within cells, where it aggregates into toxic tangles

2. Extracellular

Block cell-to-cell spread of tau as it moves between neurons

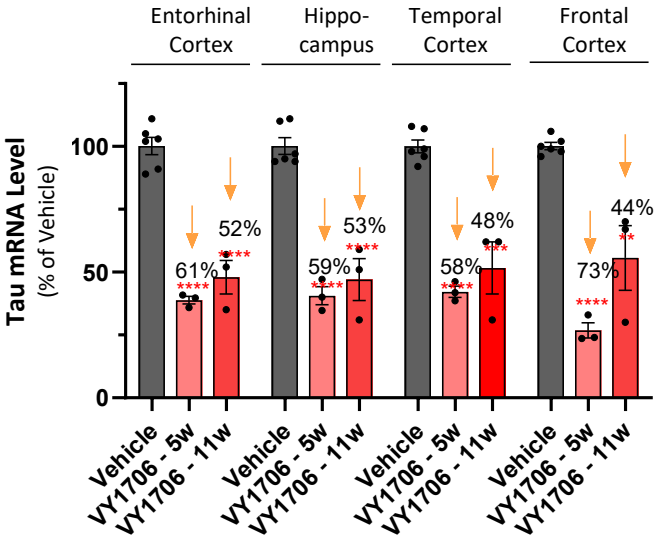
Knockdown (ASO, siRNA, etc)
(i.e. VY1706, BIIB080)
Intracellular and Extracellular

Antibodies
(i.e. VY7523)
Extracellular

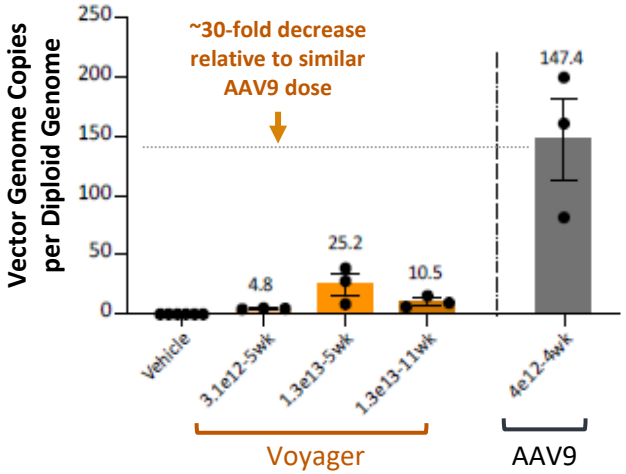
VY1706: Expect to Dose First Patient this Year

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

44-73% Reduction of Tau mRNA



~30-Fold Liver De-Targeting



Single IV dose of 1.3E13 vg/kg in NHP resulted in:

- Tau mRNA reduced 44-73%
- Tau protein reduced 27-55%
- Tau protein reduced 41-57% in neurons
- Broad distribution across brain
- ~30-fold liver de-targeting

GLP toxicology study ongoing

EXPECTED NEXT STEPS:

- Complete GLP toxicology study Q1 ’26
- File IND Q2 ’26

BIIB080 data expected mid-’26

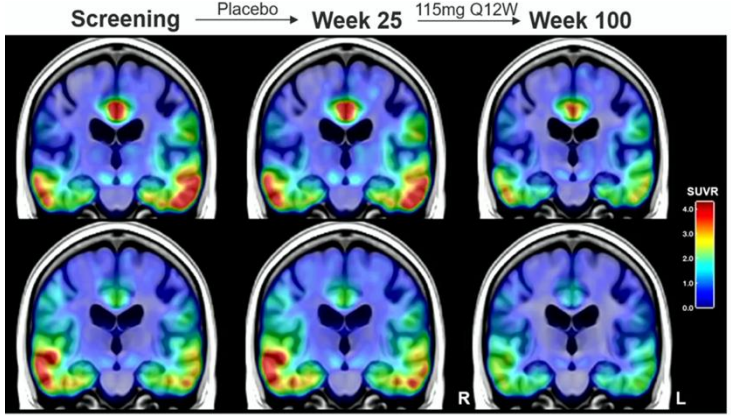
- First-in-human H2 ‘26

ASGCT 2025, Sivasankaran, et al and ASGCT 2025, Bao, et al.

BIIB080 Sets Expectations for Tau Knockdown Approach

BIIB080 (ASO knockdown, Biogen):

- Reduced mRNA 50-75%[1] in NHP
- Reduced tau per tau PET imaging in clinic
- Impacted CDR-SB in exploratory analysis



Initial data from Ph1b trial. Exploratory analyses also showed 2.04 - 2.44 slowing of decline in CDR-SB, greater than observed change with anti-amyloid antibodies (~0.5 point).[2,3,4, *]

Data from BIIB080 Phase 2 trial in 400+ AD patients expected mid-2026 could **validate tau knockdown approach** if it demonstrates:

1. CDR-SB treatment effect **better than anti-amyloid antibodies** (~0.5 point)**
2. Correlation between **tau PET imaging reduction** and improvement on clinical symptoms

VY1706 differentiation from BIIB080

BIIB080 = repeated intrathecal injection
VY1706 = one-time intravenous; broad biodistribution

[1] DeVos, et.al. Science Translational Medicine. 2017. [2] Exploratory clinical outcomes from BIIB080 (MAPT ASO) phase 1b multiple ascending dose and long-term extension study in mild Alzheimer's disease. N. Ziogas, et al. [3] Budd Haeberlein et al. J Prev Alz Dis 2022, [4] Van Dyck et al. NEJM 2023. *CDR-SB effect size estimated by comparison to a matched external natural history cohort and matched placebo cohort from distinct studies and reflect possible but not confirmed clinical effect. **Caution should be taken when making cross-trial comparisons.

VY7523: Tau PET Imaging Data in AD Patients Expected H2 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)

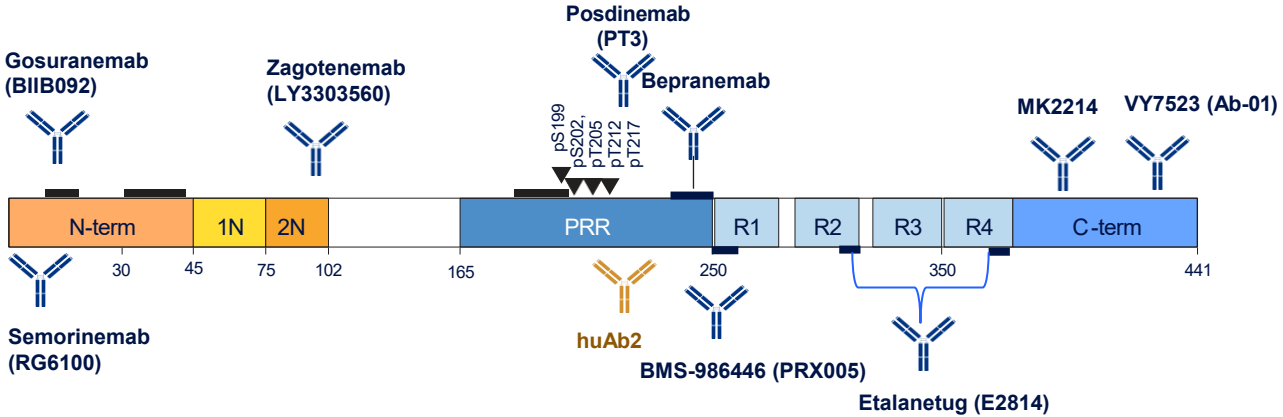
VY7523 Enrollment Complete in MAD Trial

Tau PET Imaging Data Expected H2 '26

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 unique epitope in C-terminal region

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

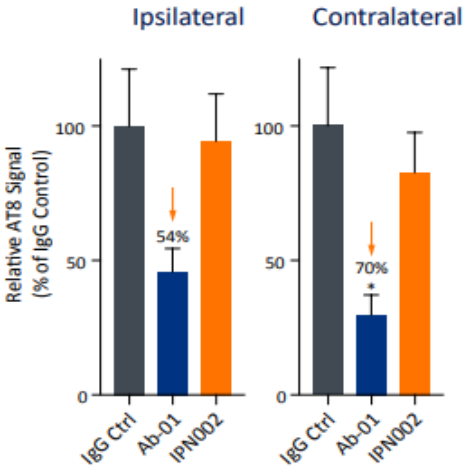
With anti-amyloid antibodies, lack of specificity for pathologic form resulted in lack of potency / peripheral sink

VY7523 is exquisitely specific for pathological tau

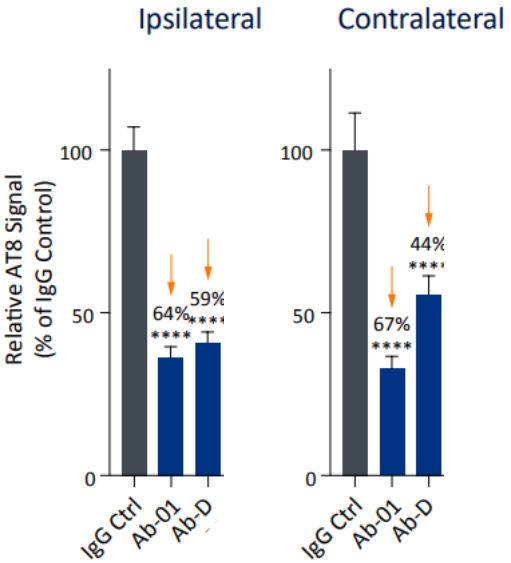
1. Davidson, R.; Krider, R.I.; Borsellino, P.; Noorda, K.; Alhwayek, G.; Vida, T.A. Untangling Tau: Molecular Insights into Neuroinflammation, Pathophysiology, and Emerging Immunotherapies. Curr. Issues Mol. Biol. 2023, 45, 8816-8839. https://doi.org/10.3390/cimb45110553. 2. ADPD 2025, Liu, et al.. 3. huAb2 is a murinized research antibody that targets the p212/p214/p217 region, similar to posdinemab

VY7523: P301S Data Differentiate from Posdinemab, Bepranemab

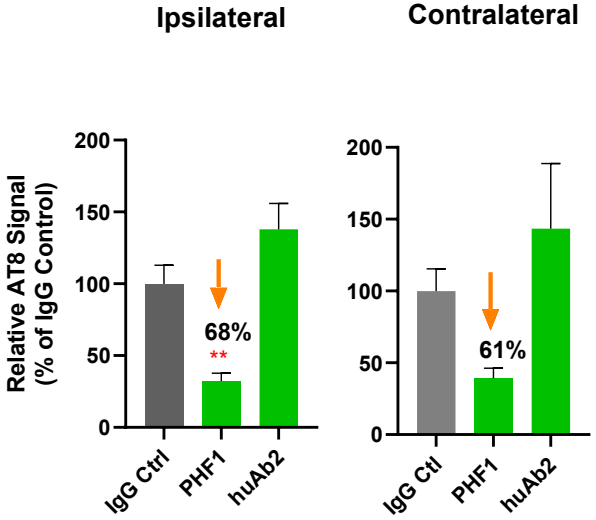
VYGR reduces tau spread; N-terminal antibody does not



VYGR reduces tau spread; bepranemab does also



huAb2 antibody binding similar epitope as posdinemab does 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.

ADPD 2025, Liu, et al.. *, **, *** 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 Proof-of-Concept in Humans

STEP 1: Safety

Determine if dosing in first few patients warrants continuation

DERISKING:

- ✓ Low doses / liver de-targeting
- ○ GLP tox in NHP

STEP 2. Delivery to Brain

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

DERISKING:

- ✓ Preclinical delivery confirmed cross-species (mouse, multiple NHPs)
- ✓ Receptor identified for VY1706 capsid; capsid binds human homolog

STEP 3. Tau Impact

Tau PET assessment to confirm impact on tau and suggest possible efficacy

DERISKING:

- ✓ VY1706 derisked by preclinical efficacy in mouse/NHP
- ○ Potential further derisking with BIIB080 data

Two Opportunities for First-in-Human Dosing in 2026:

VY1706

- First-in-human anticipated 2026
- Wholly-owned
- Tau knockdown for Alzheimer's disease

NBIB-'223

- Clinical trial initiation anticipated 2026, pending IND clearance
- NBIX partnered (VYGR has opt-in for 40% US)
- FXN replacement for Friedreich's ataxia

Leading Indications for 2026 Gene Therapy Trials

Alzheimer's Disease

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

- Initial anti-amyloid antibodies approved; tau targeting likely to be necessary once tau spread begins[2]
- Currently no tau-targeted treatments approved for AD

VY1706

Potential one-time, I.V. gene therapy to knock down tau for Alzheimer's disease

Recent AD Expert KOL Advisory Board:
high enthusiasm for VY1706; support for proposed safety approaches and monitoring plan

Friedreich's Ataxia

Impacts approximately **5,000 Americans**[3]

- All cases caused by mutations of FXN gene. One treatment available but does not replace FXN; unmet need remains.
- No treatments in development thus far have shown brain and heart activity

NBIB-'223

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

All VYGR-NBIX partnered gene therapy programs
continue to make 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. Sims JR, Zimmer JA, Evans CD, et al. Donanemab in Early Symptomatic Alzheimer Disease: The TRAILBLAZER-ALZ 2 Randomized Clinical Trial. JAMA. 2023;330(6):512–527. doi:10.1001/jama.2023.13239 3. Friedreich's Ataxia Research Alliance (FARA). What is FA? Available at: https://www.curefa.org/what-is-friedreichs-ataxia. Accessed: May 2024

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

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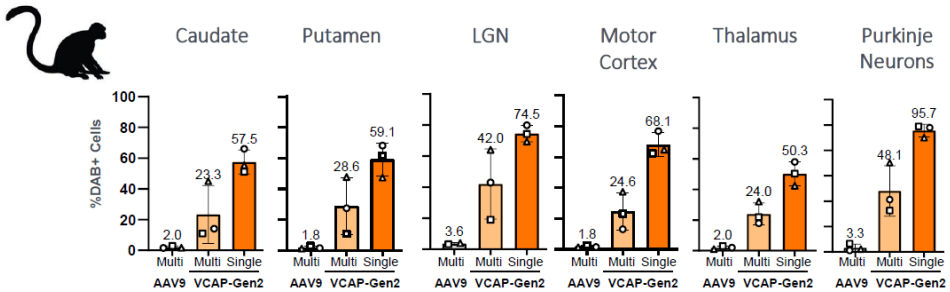
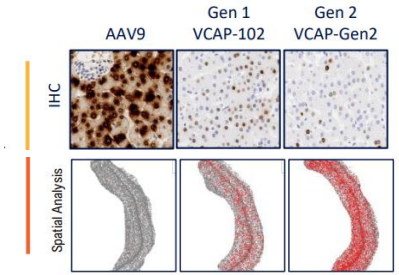
TRACER capsid discovery platform derived from evaluation of 200M+ variants of AAV5 and AAV9

VCAP-Gen2: 50-75% of Cells Transduced Across Diverse Brain Regions at 3E13 vg/kg (ASGCT 2024)



Gen2 Capsids: Increased Brain Tropism and Liver Detargeting (ASGCT 2024)



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)

Voyager NeuroShuttle™ Nonviral Platform

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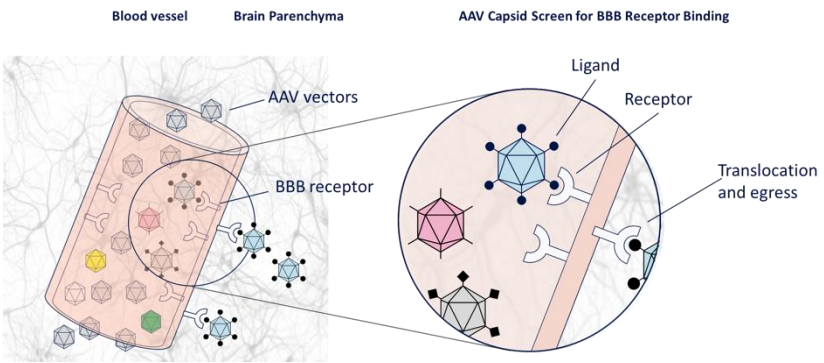
VYGR-NeuroShuttle: Next-Gen Approach to Neurotherapeutics

voyager NeuroShuttle™

Voyager NeuroShuttle™ (VYGR-NeuroShuttle) is a non-viral delivery 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

TRACER engine for receptor discovery



ALPL-VYGR-NeuroShuttle

Potential to deliver a diversity of therapeutic modalities:

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

Handful of additional receptors identified

ALPL-VYGR-NeuroShuttle Demonstrates Sustained Brain Exposures

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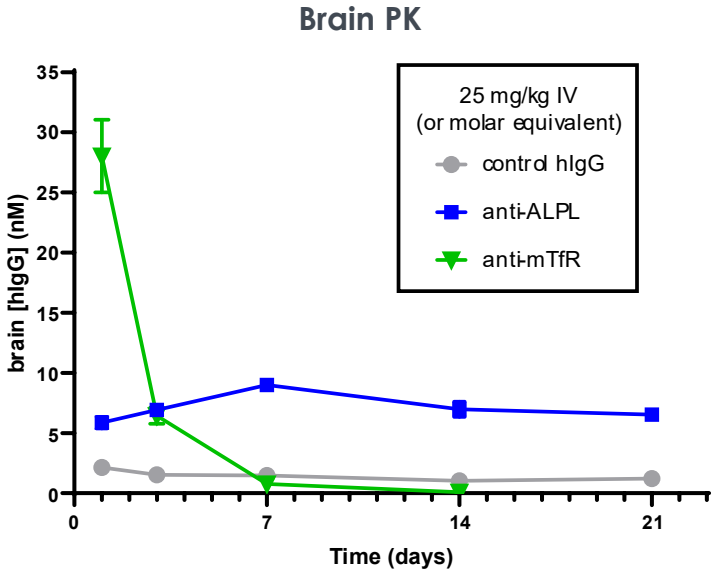
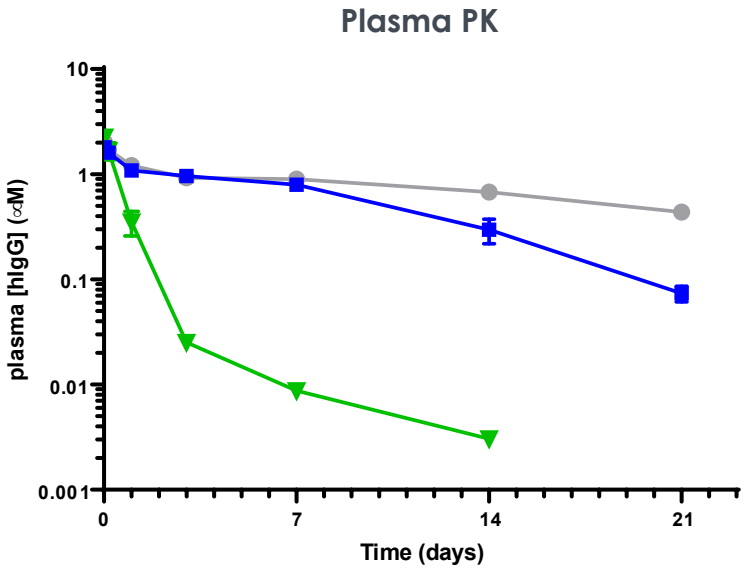
Proof of concept murine data[1] show sustained CNS exposure for ALPL versus TfR shuttles

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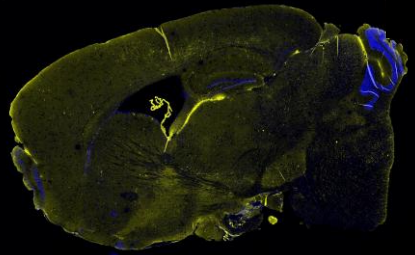
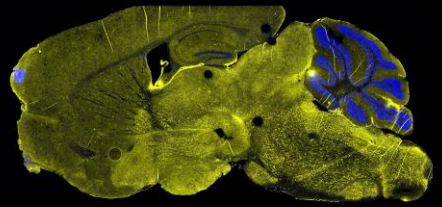
Greater parenchymal distribution observed with shuttle vs. isotype control in mouse



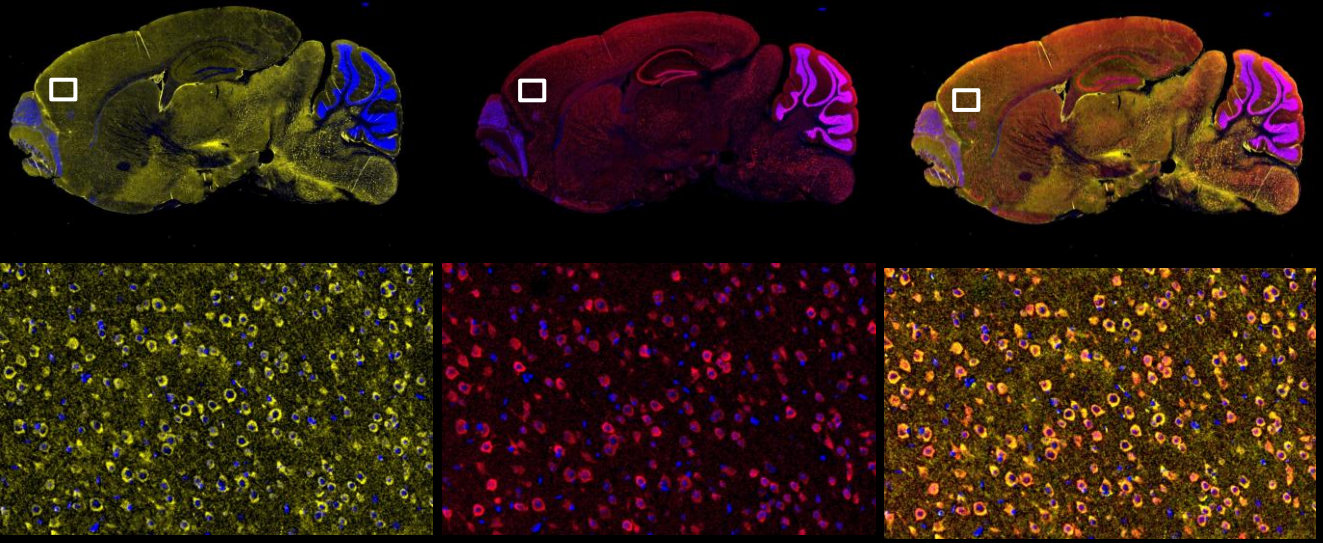
Isotype Control



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

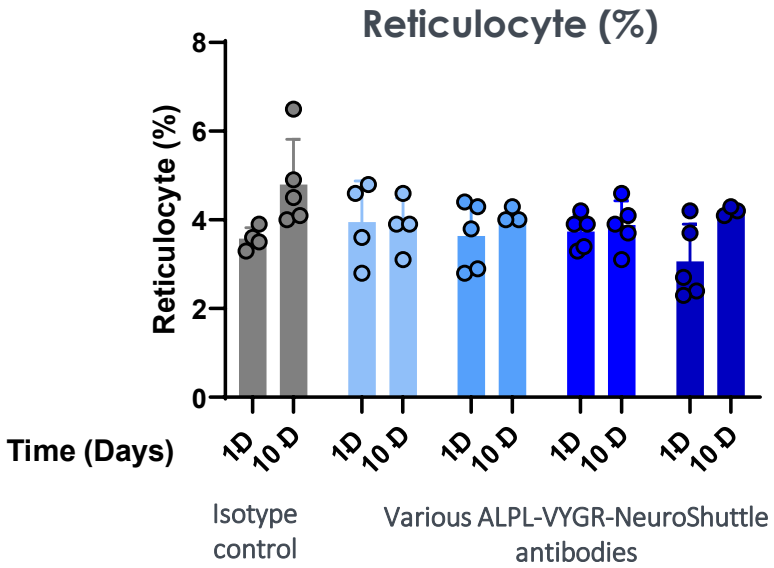
ALPL-VYGR-NeuroShuttle Does Not Impact Reticulocytes

Proof of concept murine data show negligible anemia risk for ALPL

ALPL-VYGR-NeuroShuttle technology does 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.

Anti-Amyloid Antibody as Proof-of-Concept: ALPL-Shuttled Shows Target Engagement Similar to TfR-Shuttled

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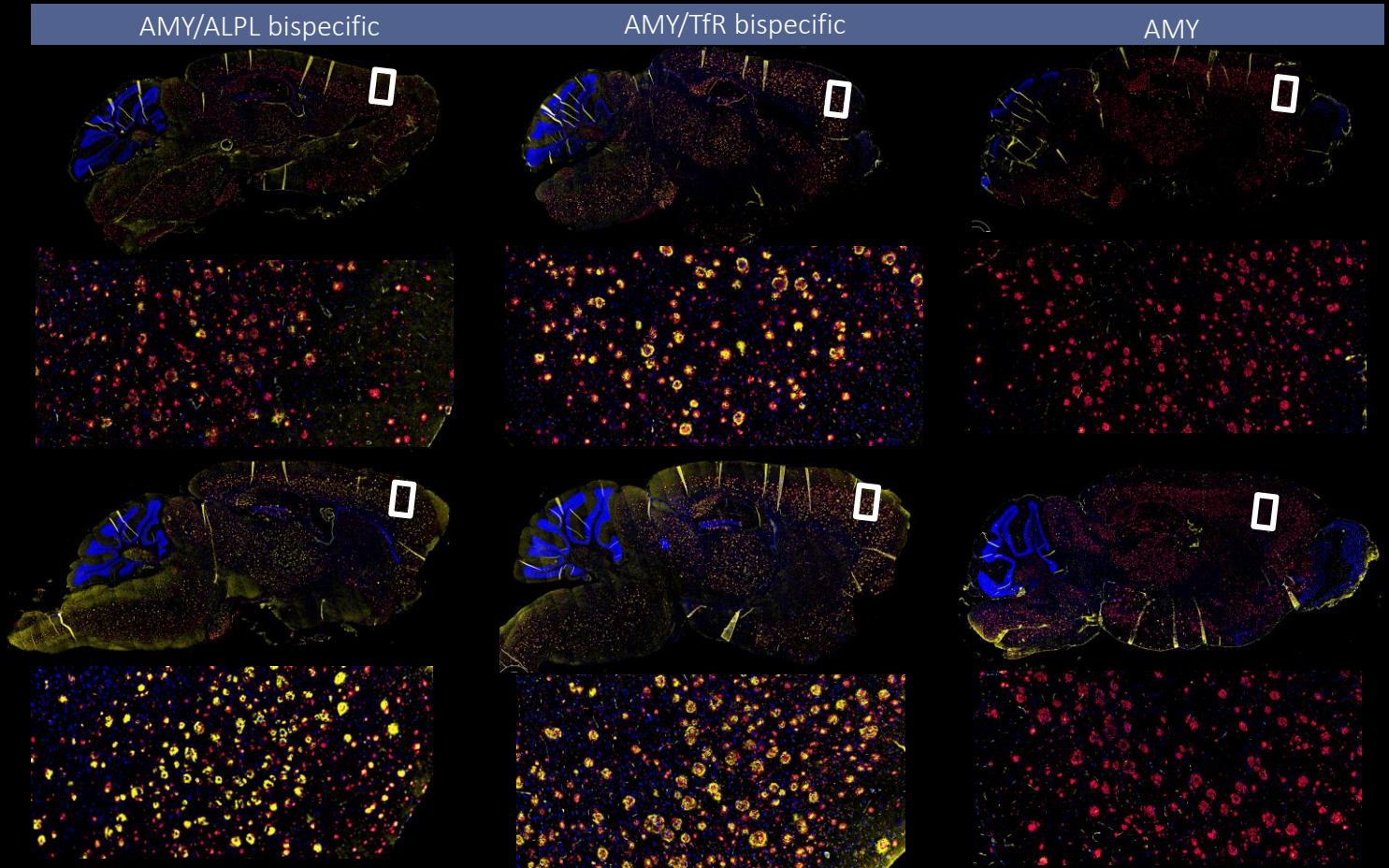
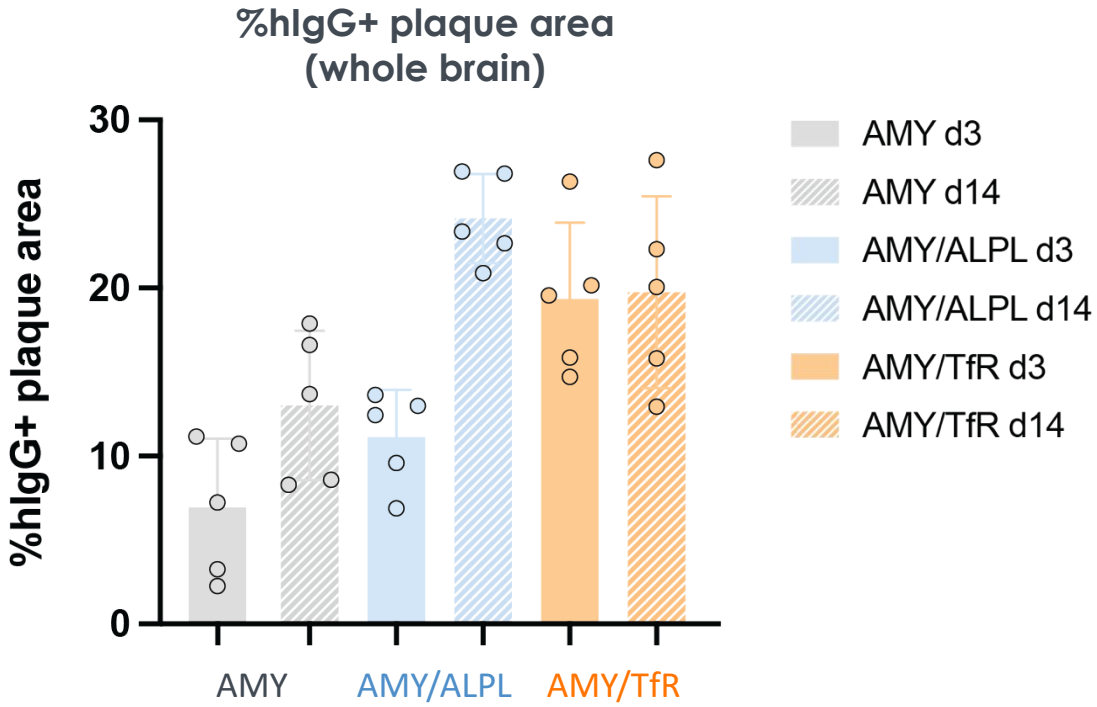
DAPI
hIgG
Aβ



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

Target engagement after a single IV dose (low dose) in mouse

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



- Both ALPL and TfR groups show 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 Appreciate Shuttle Opportunity and Value

Multiple potential opportunities for Voyager's NeuroShuttle platform to create value:

- Increase efficacy by increasing on-target delivery
- Improve safety by reducing peripheral exposure
- Lower COGS by reducing dose needed
- Expand opportunity for shuttles:
  ALPL first of multiple receptors identified

Select M&A / Licensing Deals in the Shuttling Technology Space

Partner	Target	Value ($mm)	# of Asset(s)	Indication	Phase
GSK	ablbio	$2,720	Platform	Neurodegenerative 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
NOVARTIS	AVIDITY BIOSCIENCES	$12,000	3 + Platform	Neuromuscular Disease	Registration
Roche	Manifold Bio	$2,000	Platform	Neurology	Preclinical

M&A Deals

voyager NeuroShuttle™

VYGR-NeuroShuttle is a priority for Voyager.

Platform optimization and program development across therapeutic modalities ongoing, in rodents and non-human primates.

Business

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BD Opportunities to Transform Treatment of CNS Diseases

Alzheimer's Franchise

» Tau antibody (Ph 1/2) » Tau gene therapy (IND/Clinic 2026) » 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 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.

Management Team: Extensive Neurology Expertise

Al Sandrock, M.D., Ph.D.
Chief Executive Officer

Todd Carter, Ph.D.
Chief Scientific Officer

Nathan Jorgensen, Ph.D.
Chief Financial Officer

Trista Morrison
Chief Corporate Affairs Officer, Chief of Staff to CEO

Robin Swartz
Chief Business Officer, Chief Operating Officer

Recent Achievements and Upcoming Milestones Anticipated

Q3 2025 ✓	**Voyager NeuroShuttle™** unveiled; ALPL-VYGR-NeuroShuttle program advanced into pipeline
Q4 2025 ✓	Enrollment complete in VY7523 (anti-tau antibody) multiple ascending dose trial in AD patients
Q1 2026	Complete GLP toxicology study with VY1706 (tau silencing gene therapy) for AD
Q2 2026	IND filing with VY1706
Mid-2026	Data from **BIIB080 Phase 2 trial** in 400+ AD patients; potential to derisk tau knockdown approach
H2 2026	**First-in-human dosing of VY1706**; potential first TRACER-derived novel capsid in the clinic
H2 2026	**Initial tau PET imaging data for VY7523** in AD patients
2026	NBIX expects clinical trial initiation for NBIB-'223 for Friedreich's ataxia, pending IND clearance
2026	NeuroShuttle data on NHP translatability, safety, program advancement
Ongoing	Potential for additional value-creating partnerships; discussions ongoing

Runway into 2028; this does not include any potential milestone payments from existing partnerships[1]

[1] Based on our current operating plans, cash and cash equivalents and marketable securities as of Dec. 31, 2025, along with amounts expected to be received as reimbursement for development costs under the Neurocrine and Novartis collaborations and interest income.

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Thank You

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