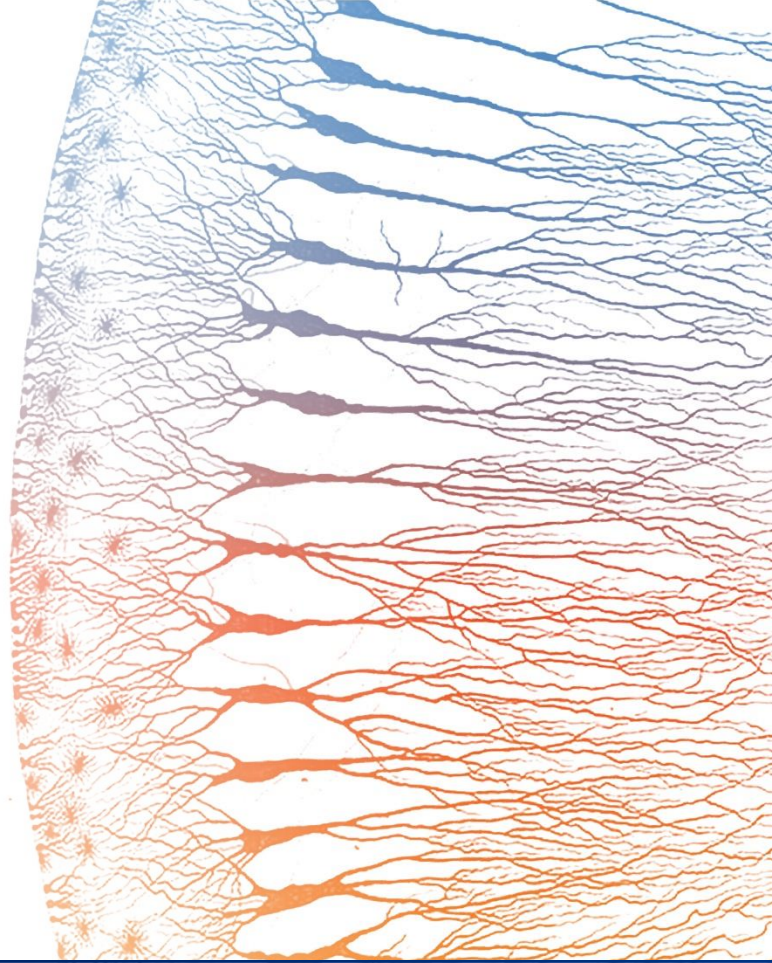
voyager therapeutics

Leveraging Genetics to Treat Neurological Diseases

Corporate Deck / August 6, 2025



Forward-Looking Statements

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 the generation of clinical data and proof-of-concept; the potential for third-party clinical data to inform Voyager’s product development programs; Voyager’s ability to expand from gene therapy and antibodies into other modalities of neurogenetic medicine; 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 timing of regulatory submissions by collaborators; 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; 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 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 identification platforms and capsids that may be competitive to Voyager’s TRACER capsid discovery platform; 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 ability to attract and retain talented directors, employees, and contractors; and the sufficiency of Voyager’s cash resources. 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) Strategy

voyager therapeutics

Cash runway into 2028, not including $7.4B in potential milestone payments from existing partnerships[1]

ADVANCE NEURO PIPELINE	Expect 4 programs in clinic in 2026; VY7523 data in AD patients expected H2 2026	4 wholly-owned Alzheimer's programs covering amyloid, tau, APOE
ADDRESS TECHNICAL + COMMERCIAL NEURO RISK	Focus on validated targets, biomarker-based path to POB/POC, transformative effects for high unmet needs	I.V. AAV and shuttles designed to optimize delivery of therapeutics to the CNS
ATTRACT TOP PEOPLE + PARTNERS	Validation and non-dilutive funding ($500M+) from partners including Novartis, Neurocrine, Alexion	Neuro team with track record of successful CNS drugs

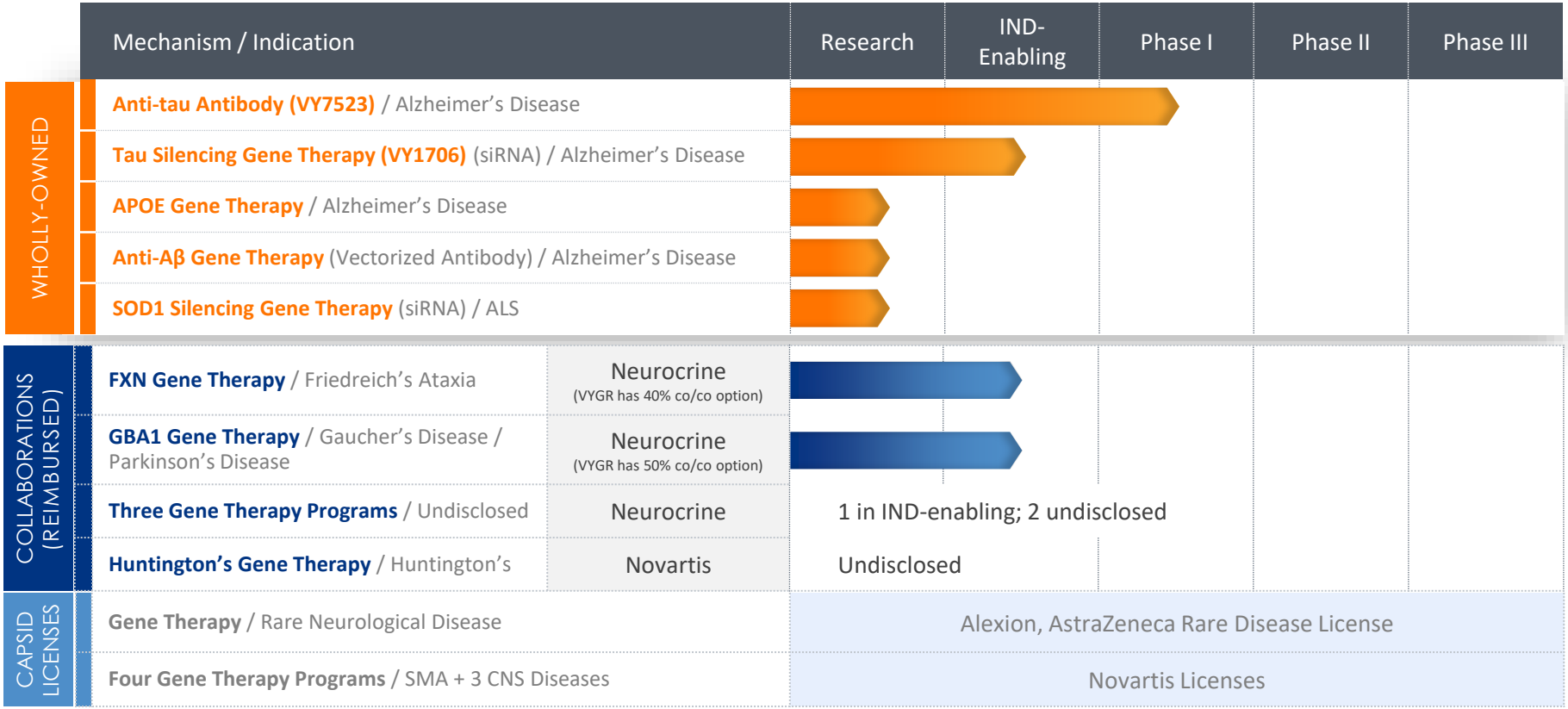
[1] Based on our current operating plans, cash and cash equivalents and marketable securities as of June 30, 2025, along with amounts expected to be received as reimbursement for development costs under the Neurocrine and Novartis collaborations and interest income.

Pipeline

voyager therapeutics

CNS Pipeline Focuses on Validated Targets with High Potential Value

voyager therapeutics



Multiple Potential Catalysts from Later-Stage Pipeline + Third Parties

voyager therapeutics

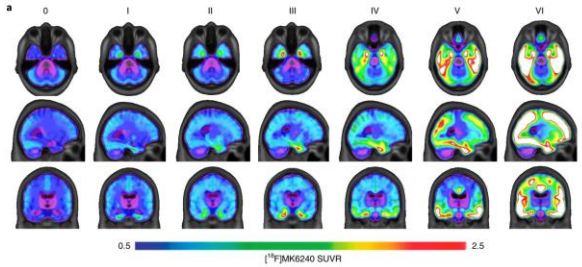
MECHANISM / INDICATION		2025	2026
Anti-tau Antibody (VY7523) Alzheimer's Disease		**VYGR SAD** top-line data H1'25	**VYGR MAD Tau** PET data H2'26
Multiple third-party data readouts on tau in 2025-2026 (Antibodies / Silencing)		**MRK Ph1 Tau-C** Ab data Q3'25 **NVS Ph1b Tau** ASO data AD Q4'25	**JNJ Ph2 Tau** Ab data Q2'26 **BIIB Ph2 Tau** ASO data H2'26
Tau Silencing Gene Therapy (VY1706) (siRNA) Alzheimer's Disease			**VYGR IND** 2026
FXN Gene Therapy Friedreich's Ataxia	Neurocrine (VYGR has 40% co/co option)	**NBIX IND** 2025	
GBA1 Gene Therapy Gaucher Disease / Parkinson's Disease	Neurocrine (VYGR has 50% co/co option)	**NBIX IND** 2025	

Both programs expected to enter clinic in 2026, per NBIX guidance

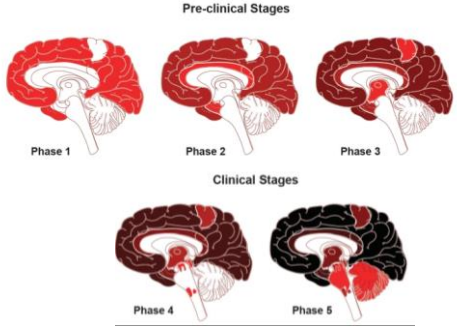
Voyager's Industry-Leading Alzheimer's Franchise

Alzheimer's disease impacts approximately **7 million Americans** and is expected to impact nearly 13 million by 2050[1]

Tau	Amyloid-beta	APOE
Likely to be necessary once tau spread begins[2]	More effective earlier in disease, before tau spreads[2]	APOE4: 11% homozygotes; 50-60% heterozygotes[1]



Tau spread correlates to worsening AD[3]



amyloid-β therapies approved for AD

APOE4 increases AD risk[1]

APOE2 may decrease AD risk[4]

- **VY7523 Anti-tau Antibody**
- **VY1706 Tau Silencing**
- **Vectorized Anti-Aβ Antibody**
- **Bifunctional: APOE Silencing with APOE2 replacement**

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. Therriault, Nature Aging volume 2, p526–535 (2022).4. Fortea, J., Pegueroles, J., Alcolea, D. et al. APOE4 homozygosity represents a distinct genetic form of Alzheimer's disease. Nat Med 30, 1284–1291 (2024). https://doi.org/10.1038/s41591-024-02931-w 4

VY7523 (Anti-Tau Antibody): 1st AD Patient Data Expected H2 2026

Multiple ascending dose (MAD) trial ongoing (52 early AD patients); initial tau PET imaging data expected H2 2026

Single ascending dose (SAD) trial complete (48 healthy volunteers):

- No serious/severe adverse events or infusion reactions reported
- Serum concentrations increased in dose-proportionate manner; CSF-to-serum ratio 0.3%

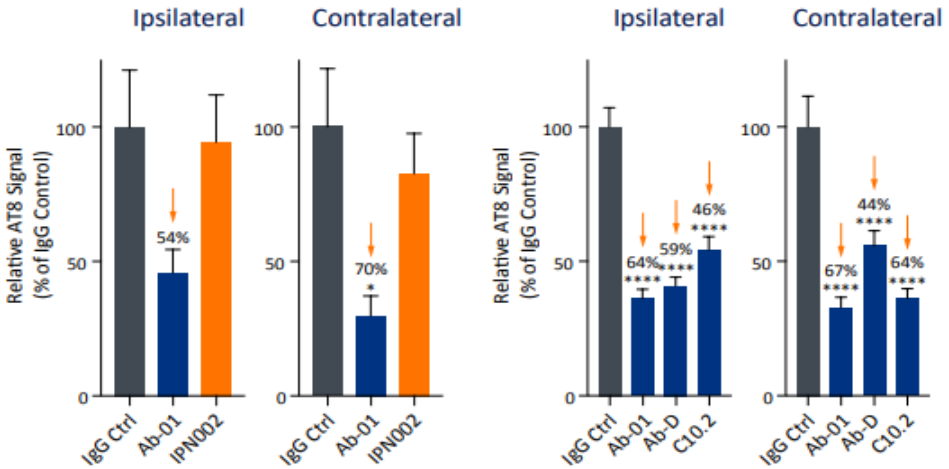
VY7523 represents a DIFFERENTIATED APPROACH to anti-tau antibodies

Selected from 700+ antibody candidates for pathologic tau specificity and efficacy in murine seeding model: [1, 2, 3]

- N-term antibody that failed in model also failed in clinic [4]
- VY7523, UCB's bepranemab, and Lundbeck's LuAF87908 work in model

Binds C terminal

- UCB, JNJ, Eisai + Prothena/BMS target mid/MTBR regions
- Merck's MK-2214 targets C-term

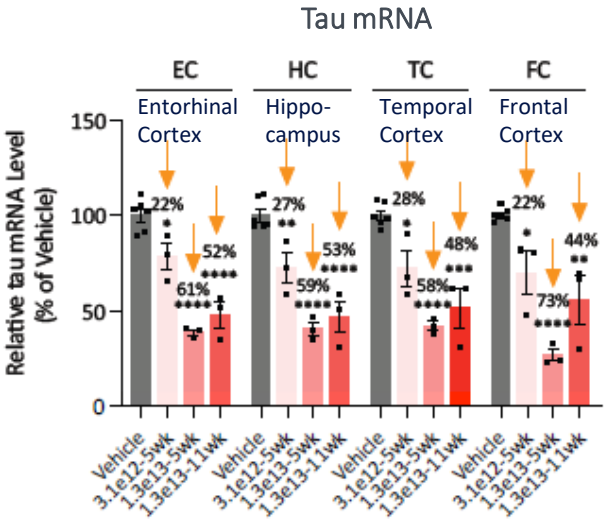


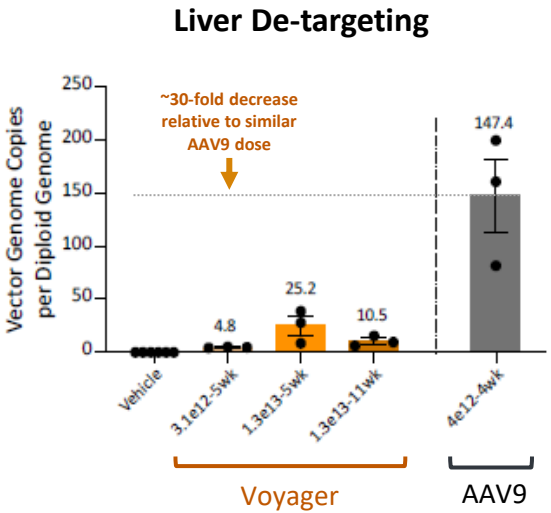
All antibodies represent murine versions:
- Ab01 / VY7523
- IPN002 / BIIB092
- Ab-D /bepranemab
- C10.2 / LuAF87908

1. AAIC 2022, Liu, et al. and data on file. 2. ADPD 2023, Liu, et al. and data on file. 3. ADPD 2024, Liu, et al. 4. ADPD 2025, Liu, et al.. *, **, *** and **** indicate $p < 0.05$, 0.005, 0.0005 and 0.0001, respectively, compared to the vehicle control group.

VY1706 (Tau Silencing): Shows Robust Tau Knockdown IV in NHPs

VY1706, A BBB-Penetrant, IV-delivered AAV Gene Therapy Provides Broad and Robust CNS tau Lowering in Tauopathy Mouse Models and Non-Human Primate





- Single IV dose in 11-week NHP study
- At 1.3E13 vg/kg:
  - *Tau mRNA reduced 44-73%*
  - *Tau protein reduced 27-55%*
- Broad distribution across the brain, including areas impacted by AD
- ~30-fold liver de-targeting vs wild type AAV9

Anticipate IND filing and into clinic in 2026

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

Potential for Impressive 2025 with TWO Gene Therapy INDs

FXN Gene Therapy

- Gene replacement of FXN for Friedreich's Ataxia
- Partnered with Neurocrine
- Voyager has 40/60 opt-in rights in U.S.

GBA1 Gene Therapy

- Gene replacement of GBA1 for Gaucher disease and Parkinson's disease
- Partnered with Neurocrine
- Voyager has 50/50 opt-in rights in U.S.

Friedreich's Ataxia

~5,000 patients living with FA in US[1].

All cases caused by mutations of the FXN gene[1].

One treatment available but does not replace FXN; unmet need remains[1].

Gaucher Disease

~6,000 patients with Gaucher disease in the U.S.[2]
All cases caused by mutations of the GBA gene. [2]
No treatment available for neurological involvement in Type 2 and Type 3 Gaucher disease; Type 1 treated with enzyme replacement therapy[2].

and/or

GBA-1 Parkinson's Disease

~1 million patients with Parkinson's disease in the U.S.[3]; up to 10% with GBA1 mutations, which increase the risk of Parkinson's by ~20-fold[4].
No disease-modifying treatments available[5].

1. Friedreich's Ataxia Research Alliance (FARA). What is FA? Available at: https://www.curefa.org/what-is-friedreichs-ataxia. Accessed: May 2024. 2. Cleveland Clinic: Gaucher Disease. Available at: https://my.clevelandclinic.org/health/diseases/16234-gaucher-disease. Accessed April 2025. 3. Parkinson's Foundation. Statistics. Available at: https://www.parkinson.org/understanding-parkinsons/statistics. Accessed: May 2024. 4. 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. 5. Parkinson's Foundation. What is Parkinson's? Available at: https://www.parkinson.org/understanding-parkinsons/statistics. Accessed: May 2024.

Success in Gene Therapy IS Possible – with the Right Elements

Zolgensma® proves success in gene therapy IS possible – for both patients and investors

>3,700 patients treated | Approved in >51 countries | $1.2B in 2024 sales | Over $6B total sales

Maximizing Probability of Technical Success

- Genetically-validated target, central to disease pathology
- Neurology drug development expertise

Maximizing Probability of Commercial Success

- Transformative benefit in disease of significant unmet need
- Intravenous delivery

Voyager's Strategy is to Replicate These Success Factors

TRACER™
AAV Capsid Platform

voyager therapeutics

Voyager's TRACER Has the Potential to Revolutionize Delivery Across the BBB

Molecular Therapy
Methods & Clinical Development

Rapid evolution of blood-brain-barrier-penetrating AAV capsids by RNA-driven biopanning *(Nonnenmacher, 2020)*

American Society of Gene + Cell Therapy

Identification and characterization of a highly conserved cell surface receptor utilized by engineered BBB-penetrant AAV capsids with enhanced brain tropism in non-human primates and mice *(Hoffman, 2024)*

American Society of Gene + Cell Therapy

Continued directed evolution of VCAP-101 and VCAP-102 identifies second generation capsids with increased brain tropism in non-human primates and mice *(Moyer, 2024)*

"Frankly, when I saw the first non-human primate data, I literally fell out of my chair."

BOB SMITH, Senior VP, Global Gene Therapy Business, Pfizer, speaking about Voyager's TRACER capsids at The Meeting on the Mesa, 2023, as reported in *Cell&Gene*

cell & gene

"…these [VYGR] capsids have incredible CNS tropism and transduction efficiency and allows us a noninvasive IV delivery…"

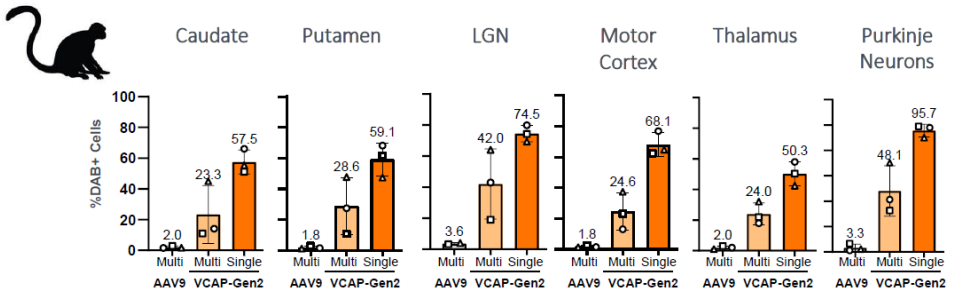
JUDE ONYIA, Ph.D., CSO Neurocrine Biosciences and Director of Voyager Therapeutics, speaking at 2023 Neurocrine Analyst Day event

NEUROCRINE BIOSCIENCES

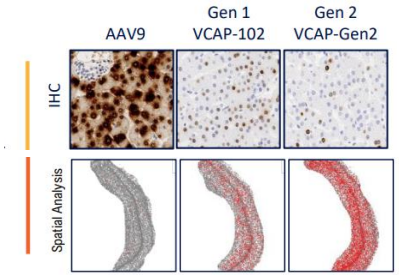
Voyager's TRACER-Derived Capsids Have Potential to Power Next-Gen Gene Therapy

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**

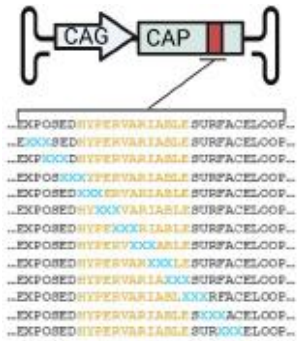
Fully integrated: capsid engineering, NHP in vivo validation, scalable production (HEK, Sf9)

Receptors Broaden Potential in Neurogenetic Medicines

Voyager identifies Alkaline Phosphatase (ALPL, formerly called Receptor X) as receptor that mediates enhanced brain tropism of VCAP-101/102 engineered capsid class

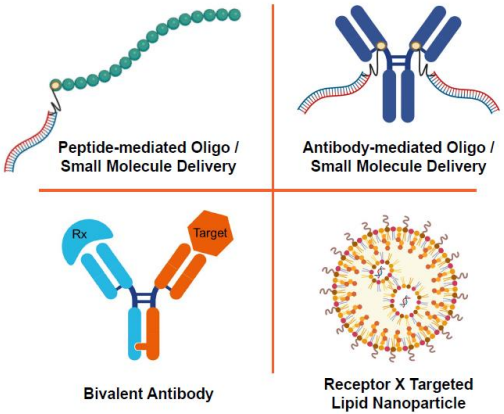
- In vitro data confirm functional interaction of VCAP-102 with human, macaque, mouse and porcine ALPL (ASGCT 2024)
- In vivo data support ALPL's role in BBB transport of VCAP-102; In silico modelling predicts binding of VCAP-102 with ALPL (ASGCT 2024)
- Ligands identified

Actively using Receptor X to speed evolution of novel capsid families

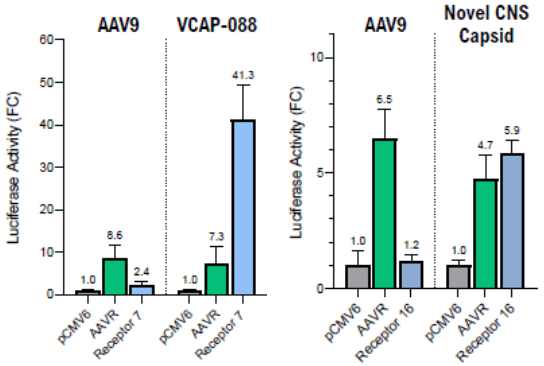


in vitro screen

Work underway to leverage receptors for potential non-viral CNS delivery



Searching for additional receptors for other capsid families; two preliminarily identified (ASGCT 2023)



Expanded opportunities in neurogenetic medicines: siRNAs, ASOs, etc.

Business

voyager therapeutics

Multiple Opportunities to Transform Treatment of CNS Diseases

voyager therapeutics

Alzheimer's Franchise

» Tau antibody (Ph 1) » Tau gene therapy (IND/Clinic 2026) » APOE + Anti-amyloid gene therapies

Rare Neurogenetic Diseases

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

CNS Delivery

» Multiple IV TRACER gene therapies moving toward clinic[1] » ALPL receptor platform emerging

[1] Includes wholly-owned and partnered programs.

Partnerships Have Brought $500M+; Potential for Another $7.4B

voyager therapeutics

	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:** 3 CNS targets	Not disclosed	$94M	$380M	$525M	**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
				$2.6B + $4.8B = $7.4B + 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
Biogen · Harvard Medical School

Todd Carter, Ph.D.
Chief Scientific Officer
Broad Institute · bci

Toby Ferguson, M.D.
Chief Medical Officer
Biogen · Temple University

Nathan Jorgensen, Ph.D.
Chief Financial Officer
VOR Biopharma · QIA Qatar Investment Authority

Trista Morrison
Chief Corporate Affairs Officer, Chief of Staff to CEO
sobi · saniona

Robin Swartz
Chief Business Officer, Chief Operating Officer
Sanofi Genzyme

Recent Achievements and Upcoming Milestones

Q1 2025 ✓	VY7523 (anti-tau antibody) well tolerated in single ascending dose clinical trial
Q1 2025 ✓	VY7523 multiple ascending dose (MAD) **clinical trial initiated** in AD patients
Q2 2025 ✓	Multiple data presentations at ADPD 2025 and ASGCT 2025
Q2 2025 ✓	APOE Gene Therapy program advanced into wholly-owned AD pipeline
Q3 25-26 ○	Potentially informative tau data read-outs expected from multiple third parties
2025 ○	IND filings anticipated with NBIX-partnered Friedreich's ataxia and GBA1 gene therapies
2026 ○	Clinical trial initiations expected for NBIX-partnered Friedreich's ataxia and GBA1 programs; potential to generate **proof-of-concept for capsids**
2026 ○	IND filing and clinical trial initiation anticipated with VY1706 (tau silencing gene therapy) for AD
H2 2026 ○	**Initial tau PET imaging data** expected in MAD trial of VY7523 in AD
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 June 30, 2025, along with amounts expected to be received as reimbursement for development costs under the Neurocrine and Novartis collaborations and interest income.

voyager therapeutics

Thank You

www.voyagertherapeutics.com