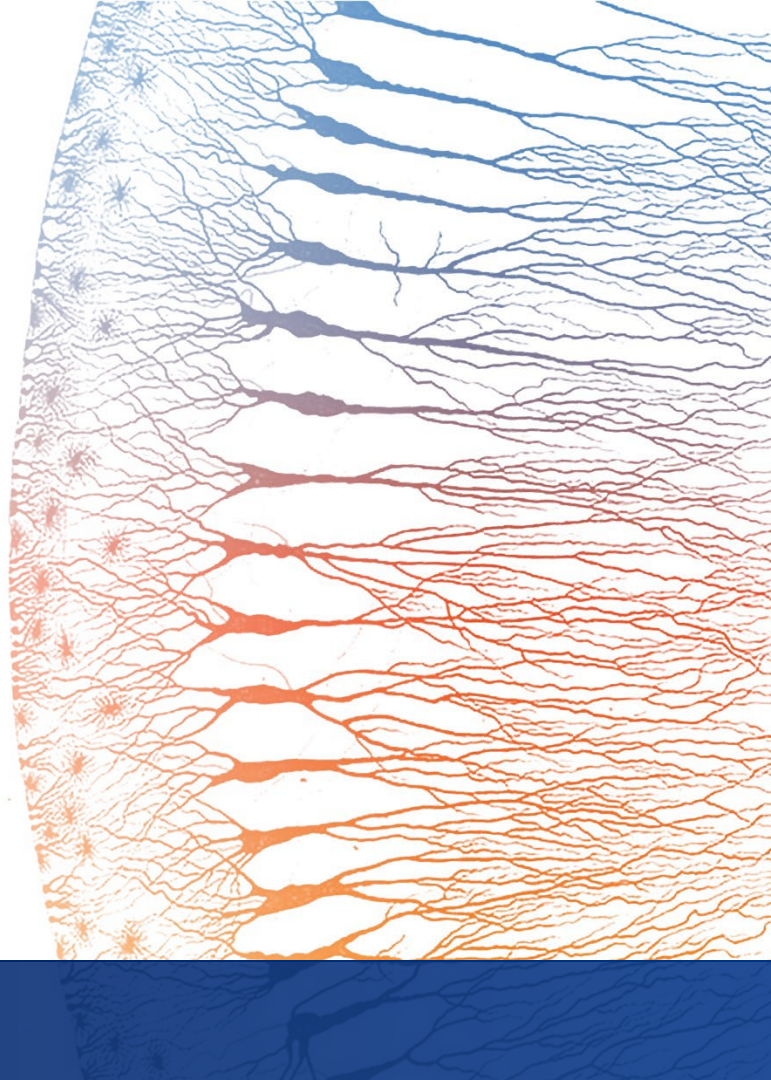
voyager therapeutics

Leveraging Genetics to Treat Neurological Diseases

Corporate Deck / January 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 “expect,” “anticipate,” “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 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 enter into new partnerships or research and development collaborations involving its platform technology and product development programs; Voyager’s ability to expand from gene therapy and antibodies into other modalities of neurogenetic medicine; Voyager’s ability to leverage receptors to its TRACER-derived capsid families for the delivery of non-viral neurogenetic medicines to the central nervous system; the potential for an antibody targeting tau to slow the accumulation of tau in the brain of Alzheimer’s patients and for this slowing to offer a clinically significant benefit in some patients; the ability of Voyager’s tau silencing gene therapy program to provide a single-dose treatment for Alzheimer’s disease; 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; 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. 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 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 antibody screening technology; Voyager’s scientific approach and program development progress, and the restricted supply of critical research components; the development by third parties of capsid identification platforms 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 platform, the capsids identified by the platform, 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) Investment Rationale

PIPELINE	Multiple clinical data readouts expected 2025/26; cash into 2027[1]	Two approaches to tau (mAb and GTx) – and all-star team
PLATFORMS	Intravenous CNS GTx platform; robust preclinical data + partnerships	Emerging ALPL shuttle may enable multi-modality CNS delivery
PARTNERSHIPS	Blue-chip partners include Novartis, Neurocrine, Alexion	$8.2B in potential milestone payments, incl. $2.9B development

[1] Based on our current operating plans, cash and cash equivalents and marketable securities as of September 30, 2024, along with amounts expected to be received as reimbursement for development costs under the Neurocrine and Novartis collaborations and interest income.

Pipeline

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CNS Pipeline Focuses on Validated Targets with High Potential Value

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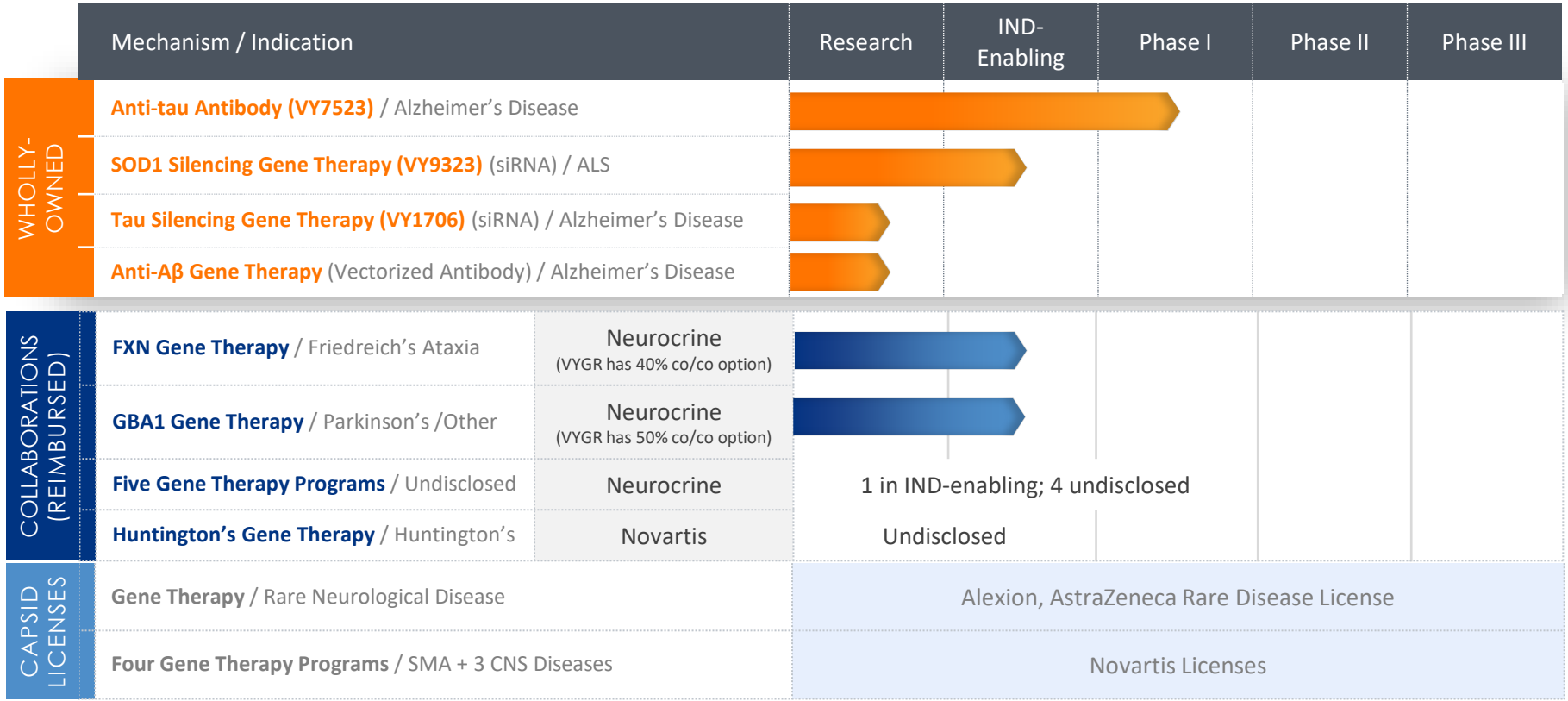

Multiple Potential Catalysts from Later-Stage Pipeline + Third Parties

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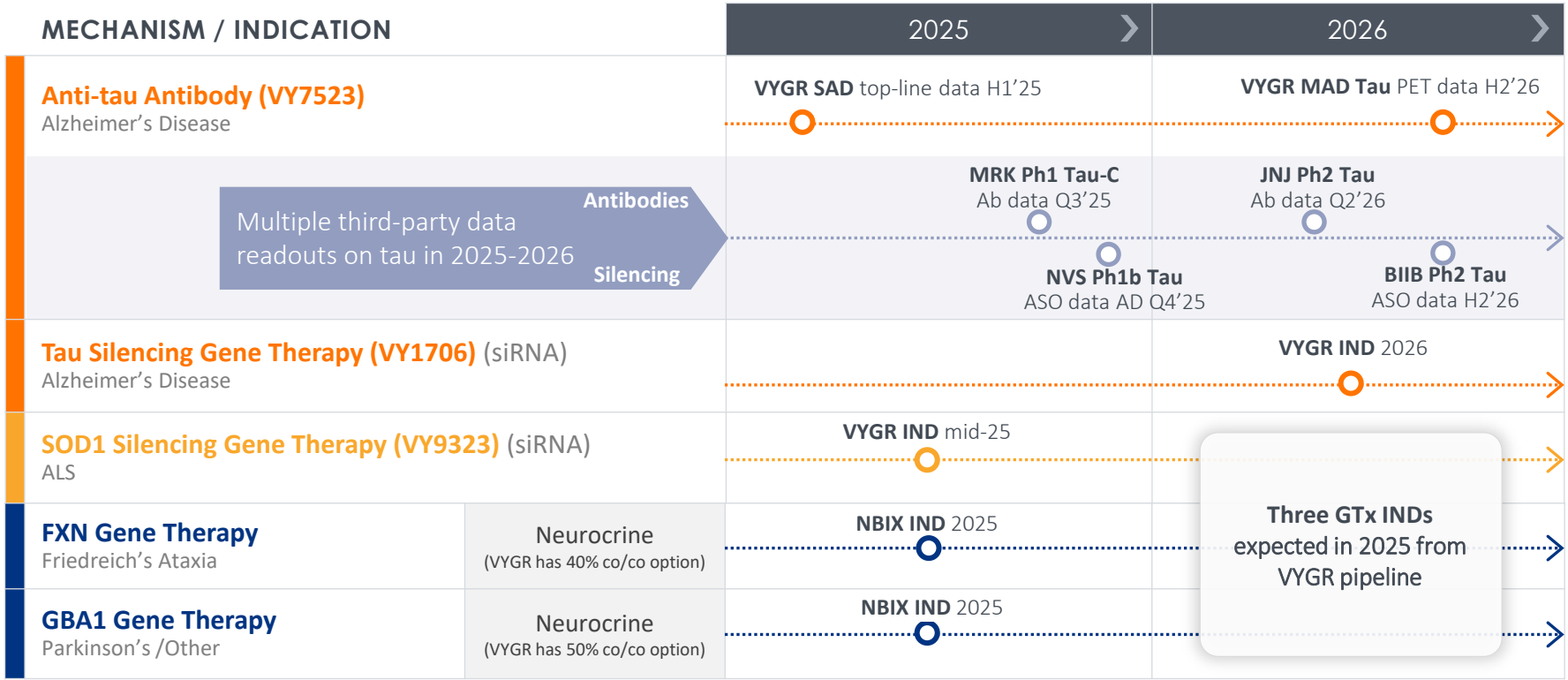
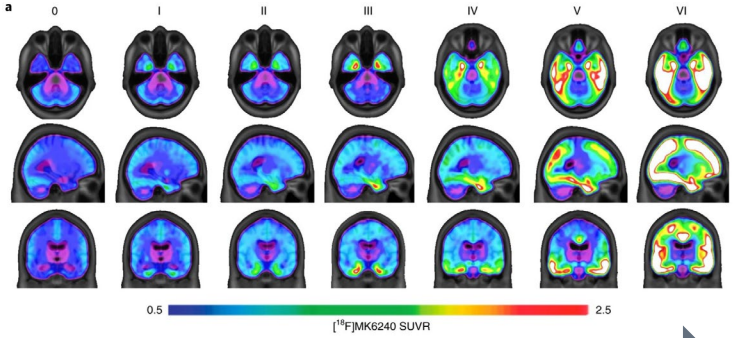


Tau Burden Correlates with Alzheimer's Disease Progression

WHY TAU IN AD?

Tau PET imaging-based staging aligns with neuropathological staging[1]



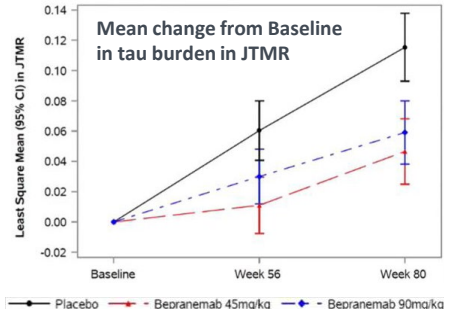
AD progression (Braak Staging) | Worsening of disease

- Genetics: Tau mutations cause neurodegenerative diseases.[2]
- Data: emerging tau PET and clinical data with antibody and silencing approaches.[3,4]

WHY ANTIBODY?

VY7523 ANTI-TAU ANTIBODY

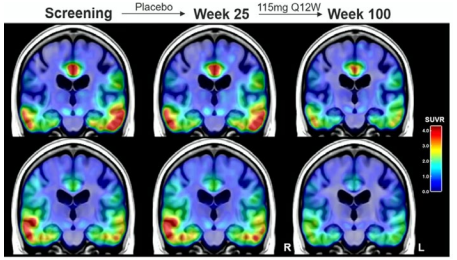
Bepranemab tau PET data demonstrates, for first time, decreased tau accumulation after anti-tau antibody treatment.[3]



WHY SILENCING?

VY1706 TAU SILENCING GENE THERAPY

ASO targeting tau showed 2.04 - 2.44 slowing of decline in CDR-SB in exploratory analysis, greater than observed change with anti-amyloid antibodies.[4,5,6]



[1] Therriault, Nature Aging 2022 [2] Wolfe, Journal of Biological Chemistry 2008. [3] UCB CTAD 2024. [4] 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. [5] Budd Haeberlein et al. J Prev Alz Dis 2022, [6] Van Dyck et al. NEJM 2023.

VY7523 Selected from 700+ Antibodies for Tau Spread Inhibition

Starting Pool

728

anti-tau antibodies/ hybridomas (328 murine, 400 human – mouse chimeric)

113

hits selective for pathological tau

27

anti-tau Abs with potent inhibition of seeding in *vitro*

11

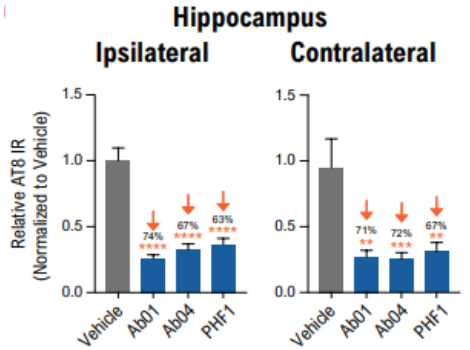
Targeted epitopes span mid-domain + C-terminal

anti-tau (5 murine, 6 human) for *in vivo* efficacy study

5

anti-tau Abs with robust inhibition of seeding *in vivo*

VY7523 (Ab01) selected for greater reduction in pathological tau spread in mouse seeding model.[1, 2, 3]



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. Vehicle = negative control. PHF1 = positive control. Ab01 and Ab04 = Voyager murine antibodies; Ab01 is murine surrogate of VY7523. *, **, *** and **** indicate $p < 0.05$, 0.005, 0.0005 and 0.0001, respectively, compared to the vehicle control group.

VY7523 Potential Points of Differentiation and Next Steps

Differentiation based on mechanism

VY7523 is pathologic tau specific:
- Important in anti-amyloid antibodies
- UCB's bepranemab is pan-tau

VY7523 binds C terminal:
- UCB, JNJ, Eisai + Prothena/BMS programs target epitopes in mid/MTBR regions
- Merck's MK-2214 targets epitope in C-term

Differentiation based on trial design

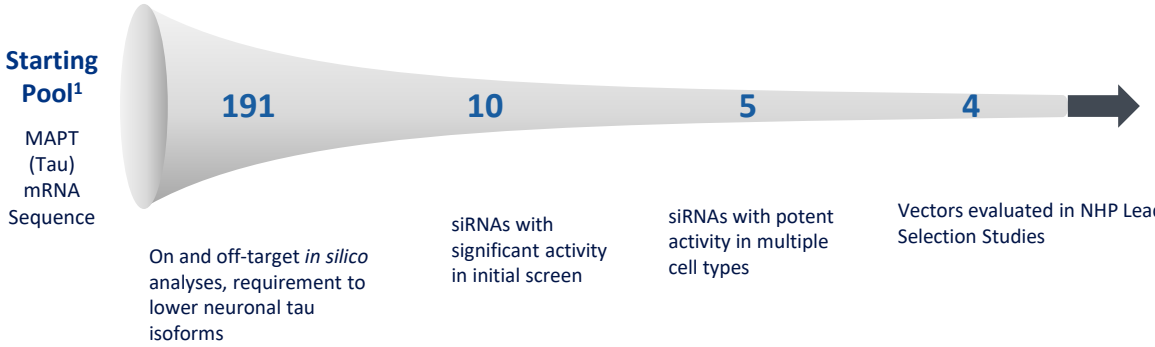
- **Safety/dose:** bepranemab was well-tolerated[1]; VYGR plans to dose higher
- **Tau PET Tracer/biomarkers:** bepranemab used GTP1 tracer[1]; VYGR plans to use MK6240 tracer and fluid biomarkers
- **Different population:** bepranemab enrolled prodromal/mild AD[1]; VYGR plans to enroll early AD/MCI, consider enriching for tau burden/APOE4 at highest dose

Key Milestones:

- ✓ **Q1 2023:** Development candidate selected
- ✓ **H1 2024:** Filed IND with FDA
- ✓ **H1 2024:** Initiated Phase 1a single ascending dose (SAD) trial in 48 healthy volunteers
- ✓ **H2 2024:** Completed enrollment and dosing in Phase 1a SAD trial
- ○ **H1 2025:** Topline safety/PK data expected from SAD trial
- ○ **2025:** Initiate Phase 1b multiple ascending dose study in early AD patients
- ○ **H2 2026:** Initial clinical data expected (Tau PET imaging)

[1]UCB CTAD 2024.

VY1706 Tau Silencing Gene Therapy: IV Knock-Down Approach



VY1706 advancing to IND-enabling studies

Murine Data: ASGCT 2024

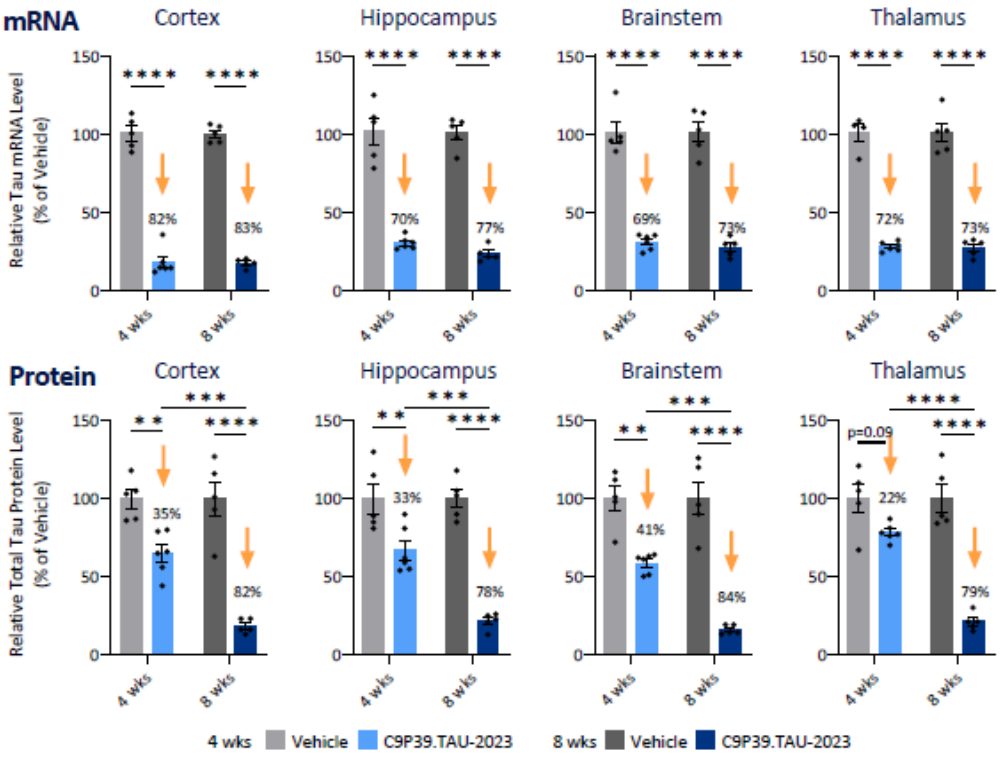
Single IV dose of VY1706 resulted in reductions in tau mRNA levels (up to 90%) and reductions in human tau protein levels (up to 84%) across the brain.

NHP Data: To be presented at upcoming scientific meeting

Single IV dose of VY1706 resulted in reductions in tau mRNA levels (50% to 73%) across cerebral cortex.

[1] VYGR ASGCT 2024.

VY1706 Significantly Reduced (Up to 84%) Human Tau Protein Levels Across the Brain (Murine Data, ASGCT 2024)



Robust reductions in human Tau mRNA and protein across multiple brain regions of hTau mice following a single intravenous administration; presented at ASGCT 2024

Anticipate IND filing in 2026

Vectorized primary artificial microRNA targeting human tau mRNA delivered by BBB-penetrant capsid administered by intravenous injection to hTau transgenic mice at 12-13 weeks of age; tissue harvested for analysis 4 weeks following administration; mRNA measured by RT-qRCR; protein measured in soluble fraction of brain regions by AlphaLISA. Asterisks correspond to statistical significance, with * indicating p<0.05 and **** indicating p<0.0001

Potential for Impressive 2025 with THREE Gene Therapy INDs

SOD1 Silencing Gene Therapy (VY9323)

- Vectorized siRNA targeting SOD1 for amyotrophic lateral sclerosis (ALS)
- Potential to provide single-dose, IV, disease-modifying treatment for SOD1-ALS patients.
- Potential to establish human proof-of-concept for BBB-penetration with Voyager's TRACER capsids.
- Wholly-owned

FXN Gene Therapy

- Gene replacement of FXN for Friedreich's Ataxia
- Partnered with Neurocrine

GBA1 Gene Therapy

- Gene replacement of GBA1 for Parkinson's and other GBA1-mediated diseases
- Partnered with Neurocrine

SOD-1 ALS

~600 ALS cases in U.S. caused by SOD1 mutations[1,2,3]. One approved monthly intrathecally administered, disease-modifying treatment; unmet need remains.

Friedreich's Ataxia

~5,000 patients living with FA in US[4]. All cases caused by mutations of the FXN gene[4]. One treatment available but does not replace FXN; unmet need remains[4].

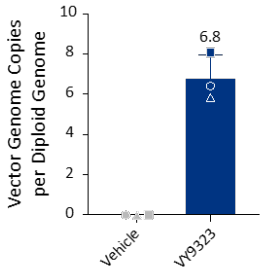
GBA-1 Parkinson's Disease

~1 million patients with Parkinson's disease in the U.S.[5]; up to 10% with GBA1 mutations, which increase the risk of Parkinson's by ~20-fold[6]. No disease-modifying treatments available[7].

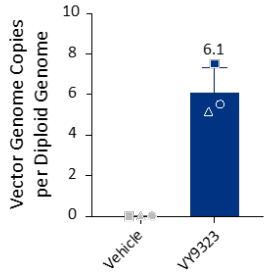
1. Mehta P., et al. Prevalence of amyotrophic lateral sclerosis in the United States, 2018. Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration. 2023 Aug 21:1-7. doi: 10.1080/21678421.2023.2245858. Epub ahead of print. PMID: 37602649. 2. Brown C., et al. Estimated Prevalence and Incidence of Amyotrophic Lateral Sclerosis and SOD1 and C9orf72 Genetic Variants. Neuroepidemiology. 2021;55(5):342-353. doi: 10.1159/000516752. Epub 2021 Jul 9. 3. Ricci C., et al. A Novel Variant in Superoxide Dismutase 1 Gene (p.V119M) in Als Patients with Pure Lower Motor Neuron Presentation. Genes (Basel). 2021 Sep 29;12(10):1544. doi: 10.3390/genes12101544. 4. Friedreich's Ataxia Research Alliance (FARA). What is FA? Available at: https://www.curefa.org/what-is-friedreichs-ataxia. Accessed: May 2024. 5. Parkinson's Foundation. Statistics. Available at: https://www.parkinson.org/understanding-parkinsons/statistics. Accessed: May 2024. 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. Parkinson's Foundation. What is Parkinson's? Available at: https://www.parkinson.org/understanding-parkinsons/statistics. Accessed: May 2024.

VY9323 Shows up to 82% Reduction of SOD1 mRNA in Spinal Cord Motor Neurons of Non-Human Primates

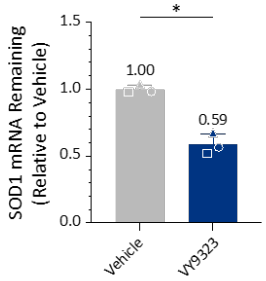
A Cervical Ventral Horn VG Biodistribution



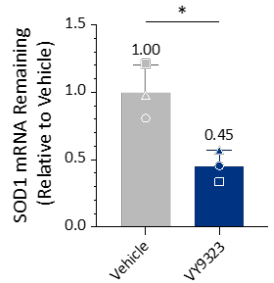
B Lumbar Ventral Horn VG Biodistribution



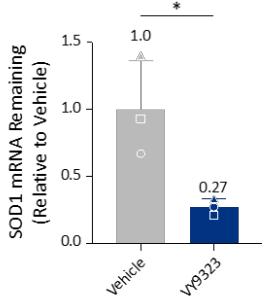
C Cervical Ventral Horn SOD1 mRNA



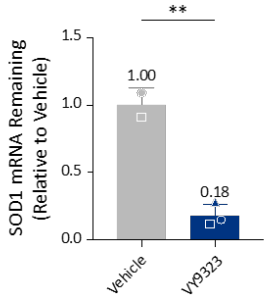
D Lumbar Ventral Horn SOD1 mRNA



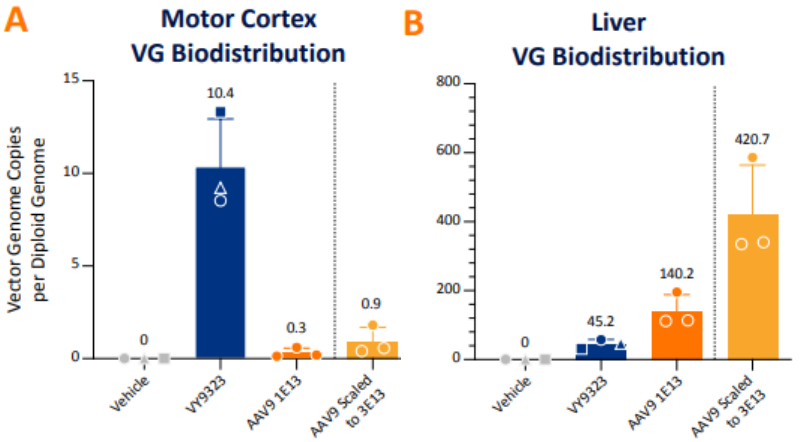
E Cervical Laser Captured Motor Neurons



F Lumbar Laser Captured Motor Neurons



Biodistribution across spinal cord and in motor cortex; detargeting of liver; presented at ASGCT 2024



 Samples collected from adult NHPs (*macaca fascicularis*) 56-days following IV administration of AAV gene therapy. LCM = Laser Capture Microdissection of presumed motor neurons. * p<0.05, ** indicating p<0.01.

TRACER™
AAV Capsid Platform

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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…"

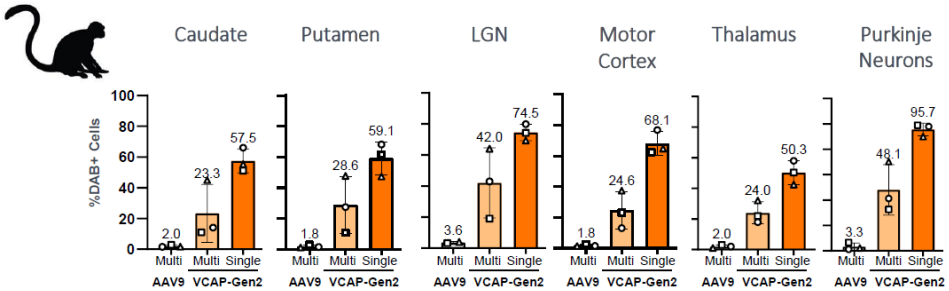
JUDE ONYIA, Ph.D., CSO Neurocrine Biosciences and Director of Voyager Therapeutics, speaking at 2023 Neurocrine Analyst Day event

NEUROCRINE BIOSCIENCES

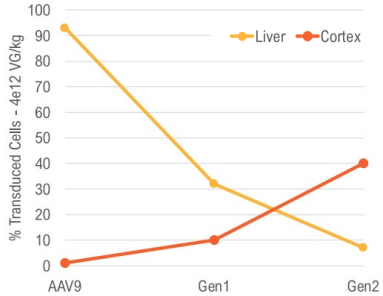
Voyager's TRACER-Derived Capsids Have Potential to Power Next-Gen Gene Therapy

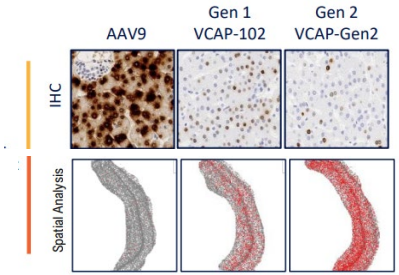
TRACER capsid discovery platform derived from evaluation of 200M+ variants of AAV5 and AAV9

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**

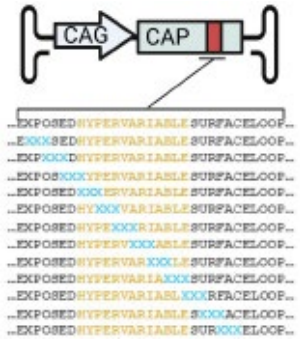
Fully integrated: capsid engineering, NHP in vivo validation, scalable production (HEK, Sf9)

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

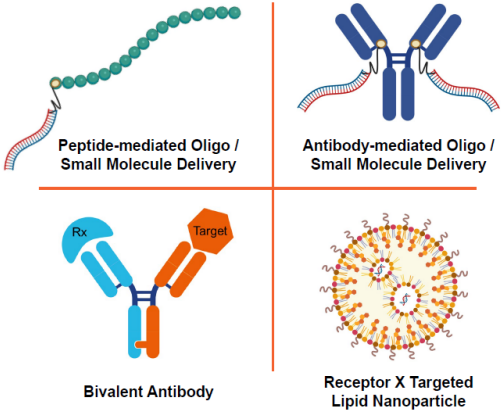
- In vitro data confirm functional interaction of VCAP-102 with human, macaque, mouse and porcine ALPL (ASGCT 2024)
- In vivo data support ALPL's role in BBB transport of VCAP-102; In silico modelling predicts binding of VCAP-102 with ALPL (ASGCT 2024)
- Ligands identified

Actively using Receptor X to speed evolution of novel capsid families

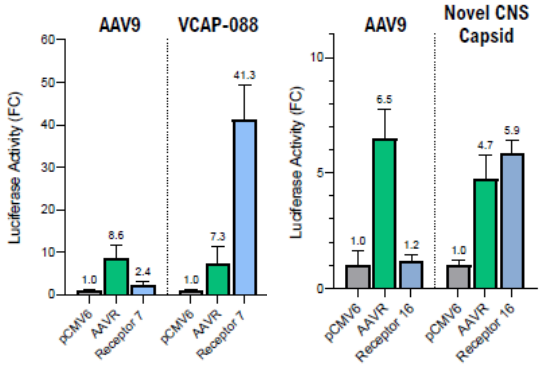


in vitro screen

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

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Multiple Opportunities for Partnership

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Alzheimer's Franchise

» Tau antibody (Ph 1) » Tau silencing gene therapy (IND 2026) » Anti-amyloid gene therapy

Rare Neurogenetic Diseases

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

CNS Delivery

» 5 TRACER gene therapies moving toward clinic (IV delivery)[1] » ALPL receptor platform emerging

[1] Five TRACER gene therapies includes wholly-owned and partnered programs..

$8.2B in Potential Milestones; including $2.9B for Development

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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	**NBIX1:** FA + 2 targets	FA: ~5,000 pts[5]	$165M	$450M[2]	$1.1B[2]	**NBIX1**: U.S. high-single-digit to high-teens; ex-U.S. mid-single-digit to mid-teens[2]
	NBIX2: GBA1 + 3 targets	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	1 rare neurologic disease target	Not disclosed	$40M	$115M	$175M	Mid- to high-single-digit
				$2.9B	**$5.3B**	

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. 3. 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 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/

Management Team: Extensive Neurogenetic Medicines Expertise

Al Sandrock, M.D., Ph.D.
Chief Executive Officer
Biogen · Harvard Medical School

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

Jacqui Fahey Sandell
Chief Legal Officer
Vericel · Takeda

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

Michelle Quinn Smith
Chief Human Resources Officer
Flagship Pioneering · Takeda

Nathan Jorgensen, Ph.D.
Chief Financial Officer
VOR Biopharma · QIA جهاز قطر للاستثمار 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

Runway into 2027 Expected to Enable Key Clinical Data on Several Programs[1]

Q1 2024 ✓	**Novartis collaboration** for HD program + SMA capsid; $100M payment (upfront and equity)
Q2 2024 ✓	**First participant dosed** in Phase 1a trial (Single Ascending Dose in healthy volunteers) with VY7523
2024 ✓	**Four development candidates** selected: wholly-owned tau silencing gene therapy VY1706 and three NBIX-partnered gene therapies (GBA-1, Friedreich's ataxia, undisclosed)
H1 2025 ○	Initial safety and pharmacokinetic data expected from VY7523 Phase 1a SAD trial
Mid-2025 ○	IND filing expected with SOD1 silencing gene therapy VY9323 in ALS patients, subsequent clinical trial has potential to generate **proof-of-concept** based on biomarkers
2025 ○	Phase 1b trial (Multiple Ascending Dose in AD patients) initiation expected with VY7523
2025 ○	IND filing anticipated with NBIX-partnered Friedreich's ataxia gene therapy
2025 ○	IND filing anticipated with NBIX-partnered GBA-1 gene therapy
2026 ○	IND filing anticipated with VY1706 tau silencing gene therapy for Alzheimer's disease
H2 2026 ○	**Initial tau PET imaging data** expected in Phase 1b trial of VY7523 in Alzheimer's disease
Ongoing ○	Potential for additional value-creating partnerships; discussions ongoing

[1] Based on our current operating plans, cash and cash equivalents and marketable securities as of September 30, 2024, 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

www.voyagertherapeutics.com