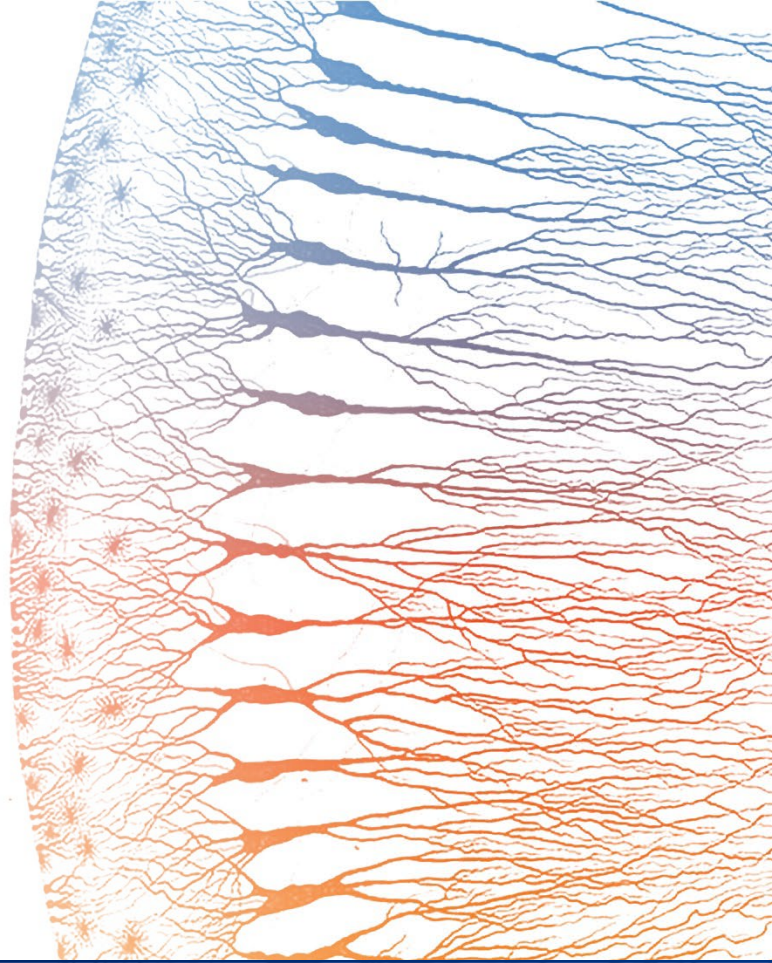
voyager therapeutics

Defining Neurogenetic Medicines

Corporate Deck / December 2024



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 Voyager’s strategy and ability to become a leader in neurogenetic medicine 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; 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: Leveraging Genetics to Treat Neurological Diseases

NASDAQ: VYGR Investment Highlights

PIPELINE

Pipeline of wholly-owned and partnered neurogenetic medicines; VY7523 anti-tau antibody in the clinic; three gene therapy candidates with IND filings expected in 2025[1], potential for **clinical data** in 2025/2026.

PLATFORM

Leading platform for CNS gene therapy delivery; cross-species preclinical data show widespread payload expression across CNS following IV delivery; enabling multiple development candidates in CNS gene therapy programs[2].

PARTNERSHIPS

Blue-chip partnerships support strong cash position: **runway into 2027**[3], not including $8.2B in potential milestone payments or potential new partnerships.

POTENTIAL

Potential to expand into additional **neurogenetic medicine** modalities. Evaluating potential for identified receptor to enable non-viral delivery of payloads across BBB.

[1] Two of these IND filings are pursuant to Neurocrine-partnered programs;. [2] Three of these development candidates are pursuant to Neurocrine-partnered programs. [3] 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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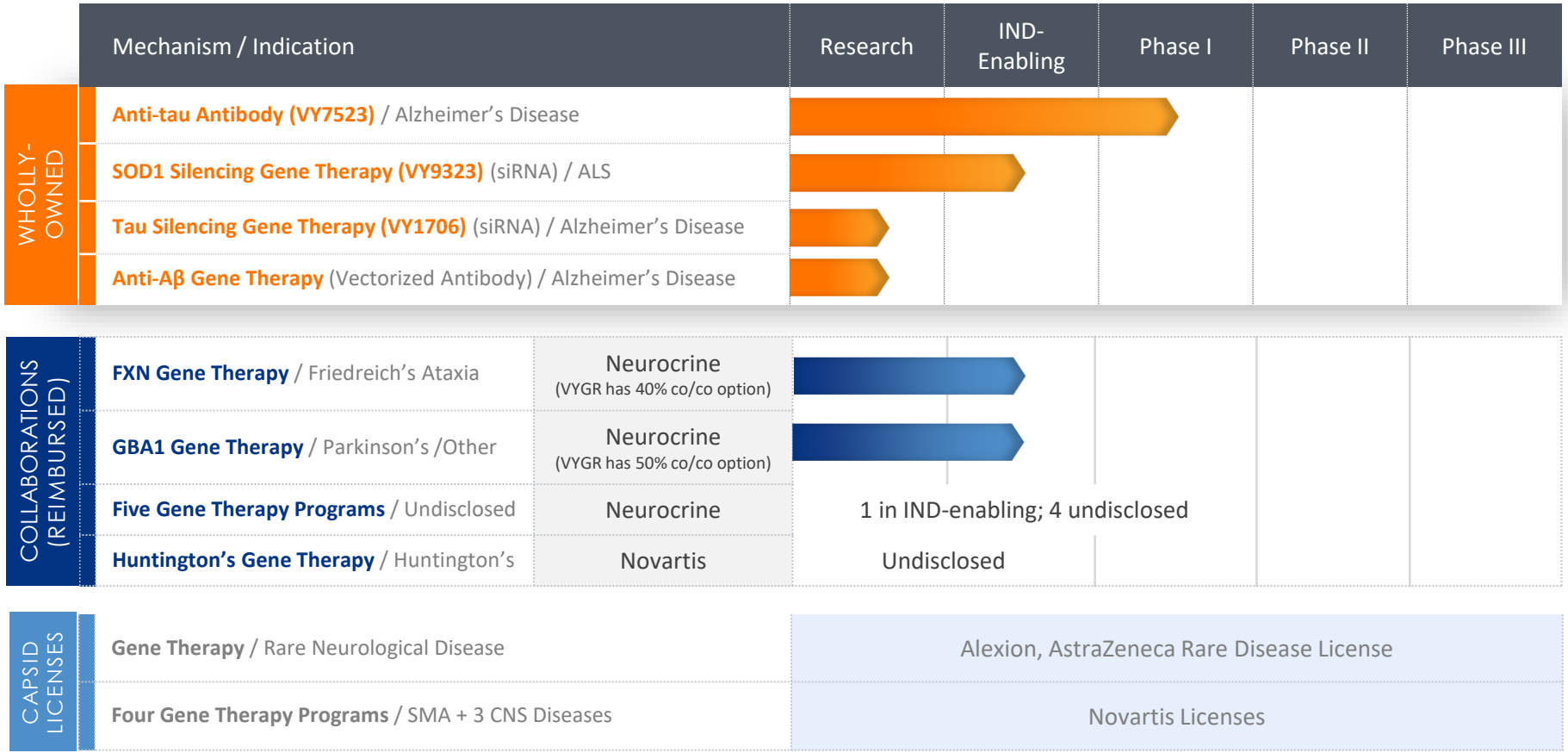
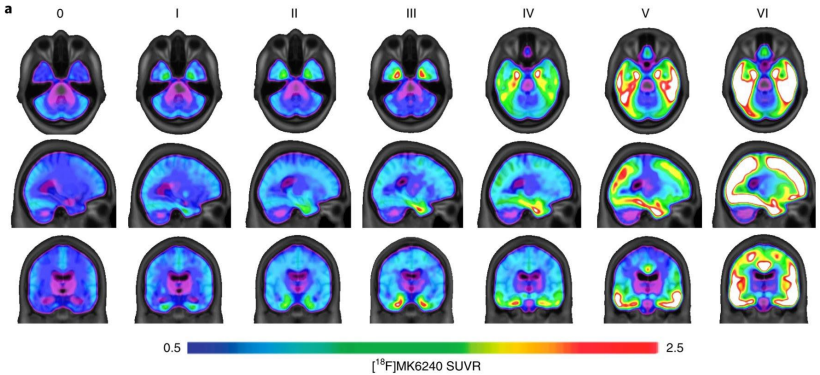


Tau Burden Correlates with Alzheimer's Disease Progression

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

Alzheimer's disease progression (Braak Staging) → Worsening of disease



- ~ 7M Alzheimer's disease patients in U.S.[2]
- Multiple clinical trials now show slowing tau accumulation may reduce cognitive decline.[3,4]
- An ASO targeting tau showed 2.04 - 2.44 slowing of decline in CDR-SB in an exploratory analysis.[3]

Voyager advancing two tau-targeting programs:

VY7523 ANTI-TAU ANTIBODY

VY1706 TAU SILENCING GENE THERAPY

[1] Therriault, Nature Aging. 2022. [2] Alzheimer's Facts and Figures Report | Alzheimer's Association [3] 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.
[4] CTAD 2024. Results from TOGETHER, a double-blind, placebo-controlled Phase 2 study evaluating efficacy, safety and tolerability of bepranemab in prodromal–mild Alzheimer's disease. M Citron et al.

VY7523 Selected from 700+ Antibodies for Tau Spread Inhibition

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Starting Pool

728
anti-tau antibodies/ hybridomas (328 murine, 400 human – mouse chimeric)

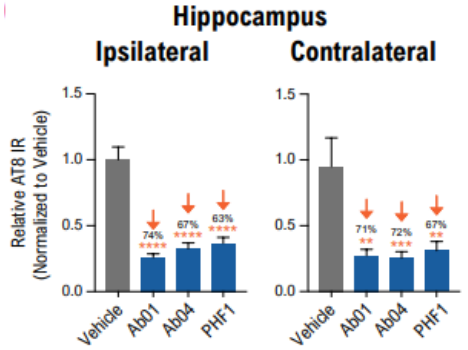
113
hits selective for pathological tau

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 Phase I Clinical Development Plan

- **First-in-human, dose-escalation trial to assess safety**
- **Dosing completed in Single Ascending Dose (SAD) trial in healthy volunteers**
  - *Design:* Randomized, placebo-controlled, single dose trial in multiple cohorts with 48 participants
  - *Timing:* top-line safety and pharmacokinetic data expected H1 2025
- **Multiple Ascending Dose (MAD) trial expected to be conducted in patients with early Alzheimer's disease**
  - *Timing:* expected to initiate in 2025; potential to generate initial tau PET imaging data in H2 2026 to determine if treatment can slow the spread of pathological tau

Key Milestones:

✓ **Q2 2023:** Received pre-IND feedback from the FDA

✓ **Q1 2024:** Completed GLP toxicology studies

✓ **H1 2024:** Filed IND with FDA

✓ **H1 2024:** Initiated Phase 1a single ascending dose (SAD) trial in healthy volunteers

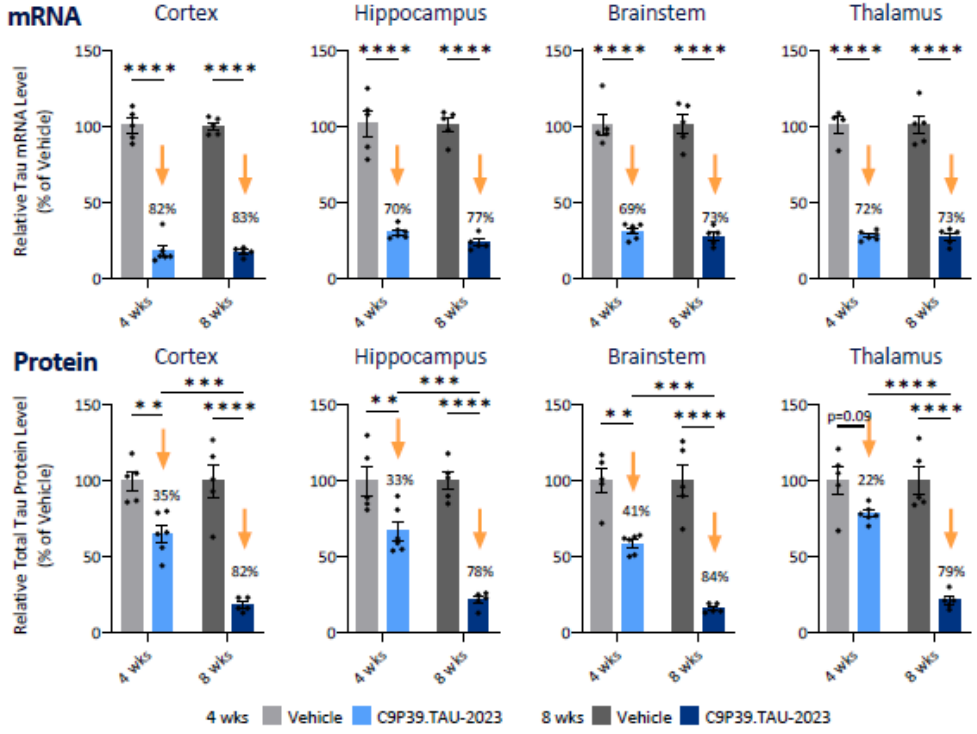
H1 2025: Topline safety/PK data expected from SAD trial

2025: Initiate Phase 1b multiple ascending dose study in early AD patients

H2 2026: Initial clinical data expected (Tau PET imaging)

VY1706 Tau Silencing Gene Therapy: IV Knock-Down Approach

Tau Silencing Gene Therapy VY1706 offers knock-down approach – intracellular



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

Three Gene Therapies On Track for INDs in 2025

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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

SOD-1 ALS

~20,000 ALS patients in U.S.[1];
~600 are caused by SOD1 mutations[1,2,3].
One approved monthly intrathecally administered, disease-modifying treatment; unmet need remains.

FXN Gene Therapy

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

Friedreich's Ataxia

~4,000 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].

GBA1 Gene Therapy

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

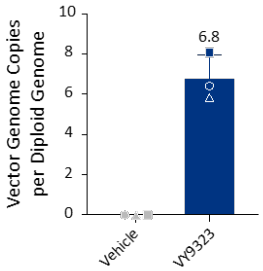
GBA-1 Parkinson's Disease

~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].

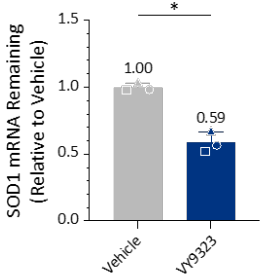
1. Mehta P., et al. Prevalence of amyotrophic lateral sclerosis in the United States, 2018. Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration. 2023 Aug 21:1-7. doi: 10.1080/21678421.2023.2245858. Epub ahead of print. PMID: 37602649. 2. Brown C., et al. Estimated Prevalence and Incidence of 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

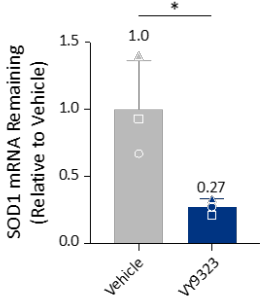
A Cervical Ventral Horn VG Biodistribution



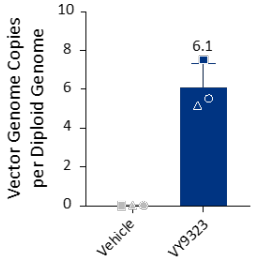
C Cervical Ventral Horn SOD1 mRNA



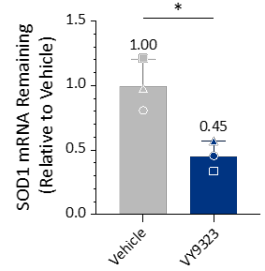
E Cervical Laser Captured Motor Neurons



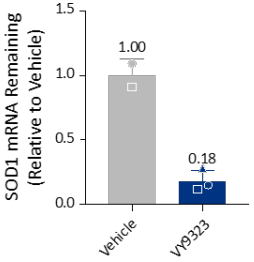
B Lumbar Ventral Horn VG Biodistribution



D Lumbar Ventral Horn SOD1 mRNA

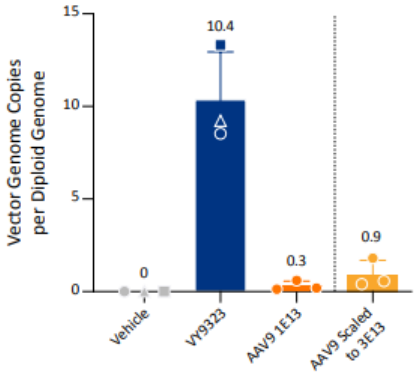


F Lumbar Laser Captured Motor Neurons

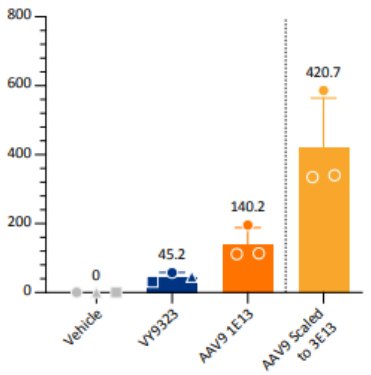


Biodistribution across spinal cord and in motor cortex; detargeting of liver; presented at ASGCT 2024

A Motor Cortex VG Biodistribution



B Liver VG Biodistribution



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)*

EUROPEAN SOCIETY OF GENE & CELL THERAPY

Identification of a Cell Surface Receptor Utilized by an Engineered BBB-Penetrant Capsid Family with Enhanced Brain Tropism in Non-Human Primates and Mice *(Hoffman, 2022)*

ASGCT 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

"…the partnership with Voyager positions us to really take advantage of these industry-leading BBB penetrant capsids, these capsids have incredible CNS tropism and transduction efficiency and allows us a noninvasive IV delivery…"

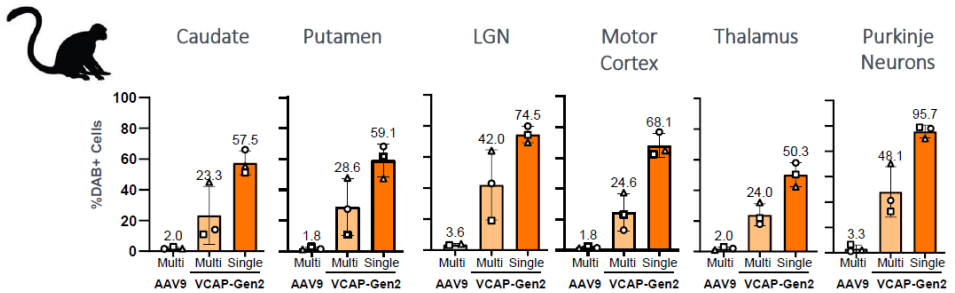
– **JUDE ONYIA**, Ph.D., CSO Neurocrine Biosciences and Director of Voyager Therapeutics, speaking at 2023 Neurocrine Analyst Day event

NEUROCRINE BIOSCIENCES

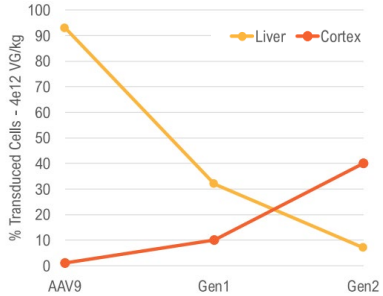
Voyager's TRACER-Derived Capsids Have Potential to Power Next-Gen Gene Therapy

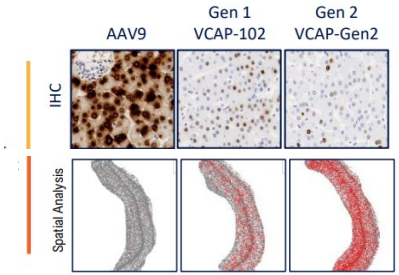
TRACER capsid discovery platform derived from evaluation of 200M+ variants of AAV5 and AAV9

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**

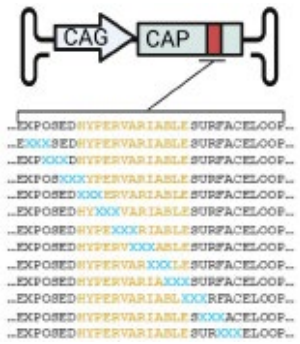
Fully integrated: capsid engineering, NHP in vivo validation, scalable production (HEK, Sf9)

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

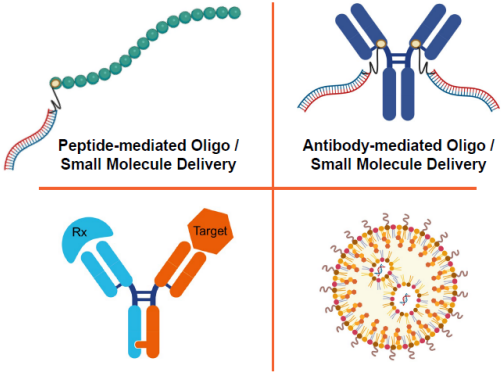
- In vitro data confirm functional interaction of VCAP-102 with human, macaque, mouse and porcine ALPL (ASGCT 2024)
- In vivo data support ALPL's role in BBB transport of VCAP-102; In silico modelling predicts binding of VCAP-102 with ALPL (ASGCT 2024)
- Ligands identified

Actively using Receptor X to speed evolution of novel capsid families

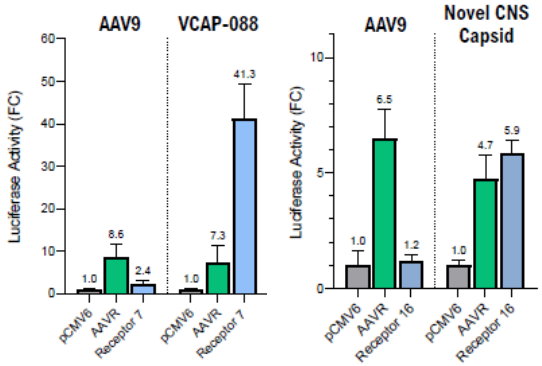


in vitro screen

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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2024 Novartis Deals Build Voyager's Blue-Chip Partnering Portfolio

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	Disease/Target (Cells, Tissues, Transgenes)	Upfront Payment[4]	Option Exercise and License Fees	Potential Milestone Payments[1]	Tiered Royalties
NEUROCRINE BIOSCIENCES	**NBIX1:** FA + 2 targets	NBIX1: $165M	N/A	NBIX1: $1.6B[2]	**NBIX1**: U.S. high-single-digit to high-teens; ex-U.S. mid-single-digit to mid-teens[2]
NEUROCRINE BIOSCIENCES	**NBIX2:** GBA1 + 3 targets	NBIX2: $175M		NBIX2: $4.2B[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	NVS1: $54M	NVS1: $40M	NVS1: $905M for licensed targets	**NVS1:** Mid- to high-single-digit
NOVARTIS	**NVS2:** HD + SMA	NVS2: $100M[4]	NVS2: N/A	NVS2: $1.2B	**NVS2:** High-single-digit to low-double-digit tiered royalties on global net sales.
ALEXION AstraZeneca Rare Disease	1 rare neurologic disease target	$30M	$10M	$290M	Mid- to high-single-digit

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.

Multiple Partnership Structures Driving Potential Long-Term Value

CAPSID LICENSES

DESIGNED TO PROVIDE:

- Validation (third-party buy-in, potential program use)
- Near-term funding (upfront and option exercise payments)
- Long-term funding (potential milestone payments and royalties)
- Multiple 'shots on goal' to demonstrate human translation

ALEXION AstraZeneca Rare Disease NOVARTIS

PROGRAM PARTNERSHIPS

DESIGNED TO PROVIDE:

- Potential for significant long-term value (profit share or milestone payments and royalties)
- Cost savings (program funding)
- Validation (investment into program)
- Near-term funding (potential upfront, early development milestone payments)

NEUROCRINE BIOSCIENCES NOVARTIS

ADDITIONAL STRUCTURES

DESIGNED TO PROVIDE:

- Opportunities to combine TRACER capsids and receptor technology with novel payloads and biologics, placing Voyager at forefront of neurogenetic medicine
- Opportunistic evaluation of alternative deal structures

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
Jounce Therapeutics · 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 · 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)
Q1 2024 ✓	Executed **$100M public offering**; extending runway into 2027
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 filings anticipated with NBIX-partnered GBA-1 and Friedreich's ataxia gene therapies
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

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