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Breaking Through Barriers in Neurology and Gene Therapy

Corporate Deck | March 2023

Forward-looking statements

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Voyager Therapeutics (Nasdaq: VYGR) Investment Thesis

Breaking Through Barriers in Neurology and Gene Therapy

CAPSID PLATFORM

The TRACER™ AAV Difference

- Potential to address gene therapy's narrow therapeutic window: superior BBB penetration at low doses
- Receptor identification supports potential for human translation
- Alliances with gene therapy leaders: Pfizer, Novartis, Neurocrine

CNS PIPELINE

Capsids Plus Diverse Payloads

- Validated targets and biomarkers allow potential for rapid proof-of-biology
- Indications with high unmet need: ALS, Alzheimer's, Parkinson's, Friedreich's Ataxia
- Nominating lead candidates; IND filings expected 2024/2025

PARTNERSHIPS

Generating Non-Dilutive Revenue

- Capsid license structure exclusive to target, NOT capsid – enables multiple licenses
- Pfizer and Novartis: license agreements for capsids against CNS targets
- Neurocrine: Collaboration on GBA1, FA, and five undisclosed targets
- Exploring additional partnerships

CNS pipeline focuses on validated targets with high potential value

Program (Mechanism)	Ownership	Early Research	Late Research	IND-Enabling
ALZHEIMER'S DISEASE Passive Tau Antibody	Wholly-Owned			
FRIEDREICH'S ATAXIA FXN Gene Therapy (Gene Replacement)	Neurocrine Collaboration (VYGR has 40% cost/profit split option)*			
ALS SOD1 Gene Therapy (Gene Silencing)	Wholly-Owned			
PARKINSON'S / OTHERS GBA1 Gene Therapy (Gene Replacement)	Neurocrine Collaboration (VYGR has 50% cost/profit split option)**			
EARLY RESEARCH PROGRAMS Allele-specific mHTT+MSH3 gene silencing for HD; Tau gene silencing for Alzheimer's; vHER2 antibody for brain mets	Wholly-Owned			

UNDISCLOSED DISEASES / Five Gene Therapy Programs	Neurocrine Collaboration
RARE NEUROLOGICAL DISEASE / Gene Therapy	Pfizer License
CNS DISEASES / Two Gene Therapy Programs	Novartis License

*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) grant Neurocrine Biosciences full U.S. commercial rights in exchange for milestone payments and royalties based on U.S. sales.
** 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) grant Neurocrine Biosciences full U.S. commercial rights in exchange for milestone payments and royalties based on U.S. sales.

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TRACER™ AAV Capsid Platform

The TRACER™ AAV Difference

Delivery will enable the future of neuro genetic medicine

DELIVERY *currently limits* **NEUROLOGY**

Difficult to deliver across blood-brain barrier (BBB)

DELIVERY *currently limits* **GENE THERAPY**

Narrow therapeutic windows with local or systemic delivery

NEURO DELIVERY *of* **GENETIC MEDICINES**
COMBINES THESE DELIVERY CHALLENGES

IV dosing: Low BBB penetration. Weak CNS transduction.
Local dosing (IT, IP*): Steep gradients. Restricted penetration within brain.

VOYAGER IS ENABLING DELIVERY *of* **NEURO-GENETIC MEDICINES**

*IT: intrathecal, IP: intraparenchymal.

Voyager's novel TRACER™-derived capsids power next-gen gene therapy

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

Minimally invasive **I.V. Delivery**

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

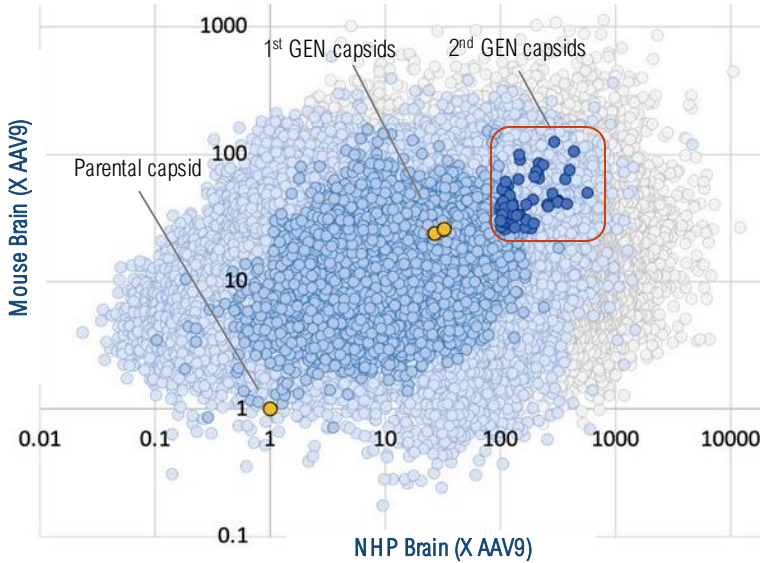
Receptor identification enables rational design

Multi-Species Validation

Improved, broad **CNS transduction**

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

>100-fold improved CNS delivery across species

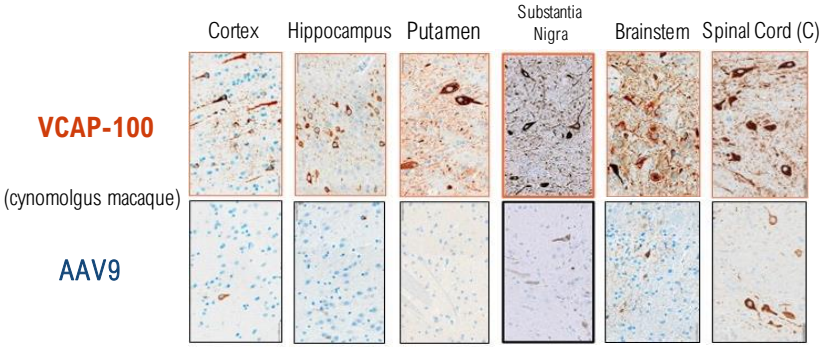


Data Presented at ASGCT 2022, Moyer et. al.

*Compared to conventional AAV9 dosed intravenously in non-human primates (NHPs)

Novel IV delivered capsids with potential to transform CNS treatment

Widespread CNS distribution



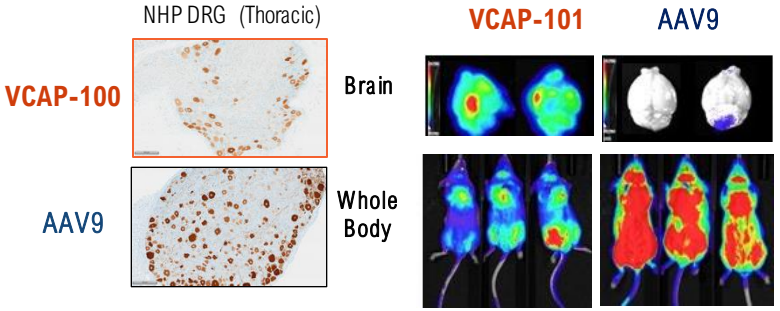
CNS neurons and glia transduced

9P801 capsid in NHP (cynomolgus macaque)

VCAP-102 capsid in mouse

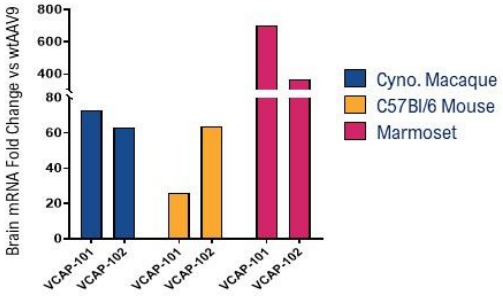
VCAP-100 capsid in NHP (macaque)

Detargeting of peripheral tissues

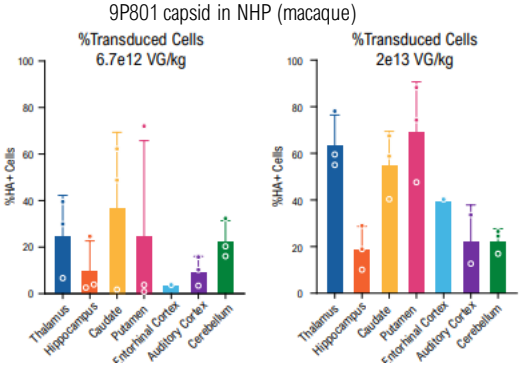


VCAP-101 capsid in mouse

Cross species transduction



Transduction at low doses



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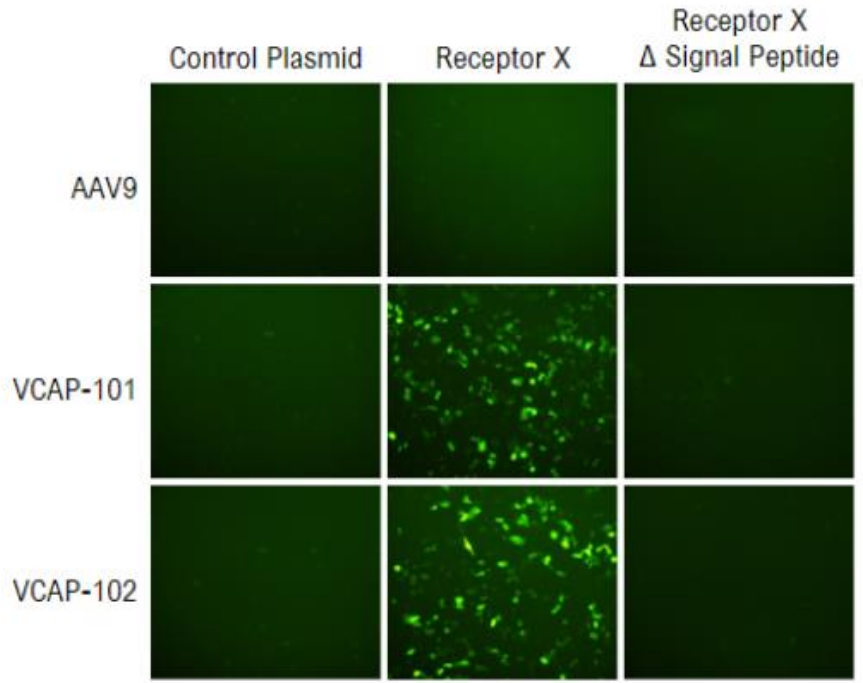
Receptor identified for TRACER™ capsid family

- ✓ Receptor identified for one of our most promising TRACER™ AAV capsids (ESGCT 2022)
- ✓ Expression confirmed in human endothelial cells and multiple CNS cell types

Characterizing the applicable receptor and confirming activity with the human orthologue increases probability that the related capsid will cross the BBB in humans

- Should facilitate rational design of AAV capsids for targeted IV delivery
- May enable IV delivery to CNS for diverse therapeutic modalities (preclinical experiments underway)



ESGCT 2022: Identification of a Cell Surface Receptor Utilized by an Engineered BBB-Penetrant Capsid Family with Enhanced Brain Tropism in Non-Human Primates and Mice

Transformative CNS Pipeline

Combining capsids with diverse payloads

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Collaboration demonstrates how Voyager is enabling neuro genetic medicine

NOVEL CAPSIDS

- IV-delivered
- 100-1000X CNS transduction
- Multiple CNS cell tropisms (neurons, glial cells); liver/DRG de-targeting
- Capsid receptor identification

✓ **Pfizer license option exercised**

✓ **Novartis license option exercised**

✓ **Neurocrine collaborating on multiple targets**

+

DIVERSE PAYLOADS

- CNS diseases
- CNS targets
- Preclinical models
- Vectorizing payloads (siRNA, antibodies, gene replacement)

✓ **Neurocrine collaborating on GBA1 and FA programs plus five discovery programs**

✓ **Voyager advancing wholly-owned programs in gene replacement, silencing, antibodies**

=

ENABLING NEURO-GENETIC MEDICINES

OPTIMAL CAPSID

+

OPTIMAL PAYLOAD

=

PROGRAM VALUE CREATION

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Anti-tau antibody offers a new twist on an Alzheimer's target

HIGH UNMET NEED + COMMERCIAL POTENTIAL

~6 million people in U.S.*

Multiple approaches needed: Like oncology, combination treatment may improve outcomes (i.e. anti-amyloid + anti-tau)

VALIDATED TARGET

Tau

Pathology closely correlates with disease progression and cognitive decline

Targets C-terminal domain

Failed approaches targeted N-terminal; more consistent than mid-domain

EFFICIENT PATH TO PROOF-OF-BIOLOGY

New Tau PET tracers enable imaging for tau pathology and use as clinical trial biomarkers

ROBUST PRECLINICAL PHARMACOLOGY

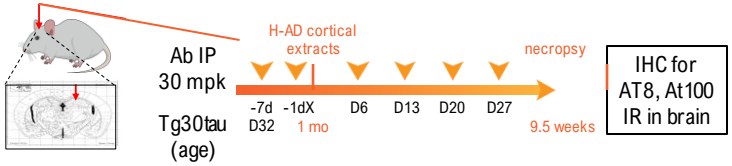
Voyager IV-delivered antibody inhibited spread of pathological tau by >70% in mouse seeding model (AAIC 2022)

STATUS: Lead development candidate selected

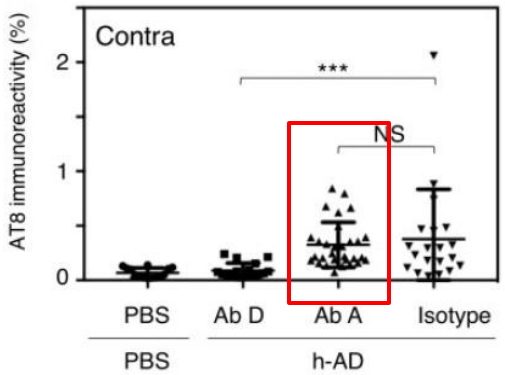
MILESTONE: Expect to initiate GLP tox in 2023 to enable IND H1 2024

*Alzheimer's Facts and Figures Report | Alzheimer's Association

Voyager's anti-tau antibody is differentiated from other anti-tau antibodies

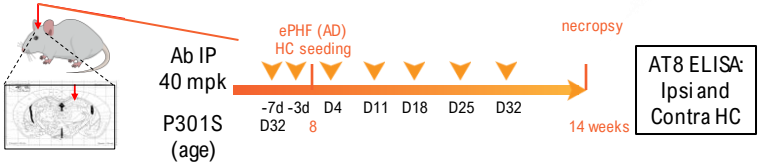


N-terminal Ab IPN002 is **not effective** in both mouse seeding model and clinic

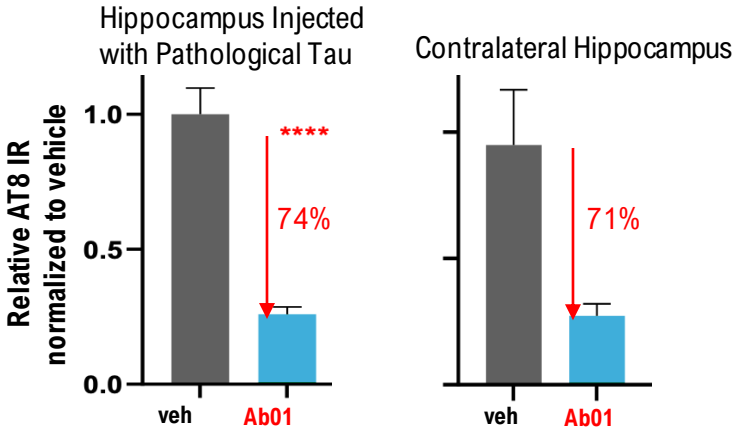


Note: Ab A targets N-terminus (aa15-24, IPN002)

Albert, *Brain*, 2019



Ab01 inhibits spread of pathological tau in mouse seeding model



Liu, *AAIC* 2022

Gene therapy approach to a validated target in ALS*

HIGH UNMET NEED + COMMERCIAL POTENTIAL

~20,000 people in U.S.**

~800 ALS patients have a SOD1 mutation

Incidence: 1 in 50,000**

Existing treatments are minimally effective; disease is typically fatal within 3 years of diagnosis

VALIDATED TARGET

SOD1

SOD1 mutations cause toxic gain of function in forms of familial ALS

Tofersen, under FDA review, is an ASO targeting SOD1 that has demonstrated clinical effect. Gene therapy approach could follow with more durable solution.

EFFICIENT PATH TO PROOF-OF-BIOLOGY

SOD1 measurable in CSF; plasma neurofilament light chain biomarkers measurable in plasma

ROBUST PRECLINICAL PHARMACOLOGY

Preclinical data showed robust SOD1 knockdown and significant improvements in motor performance and survival

STATUS: Lead optimization underway

MILESTONE: ID lead candidate projected in H1 2023

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*ALS: Amyotrophic Lateral Sclerosis **FYI: Epidemiology of ALS and Suspected Clusters | The ALS Association

SOD1 knockdown approach shows preclinical survival benefit in mouse models

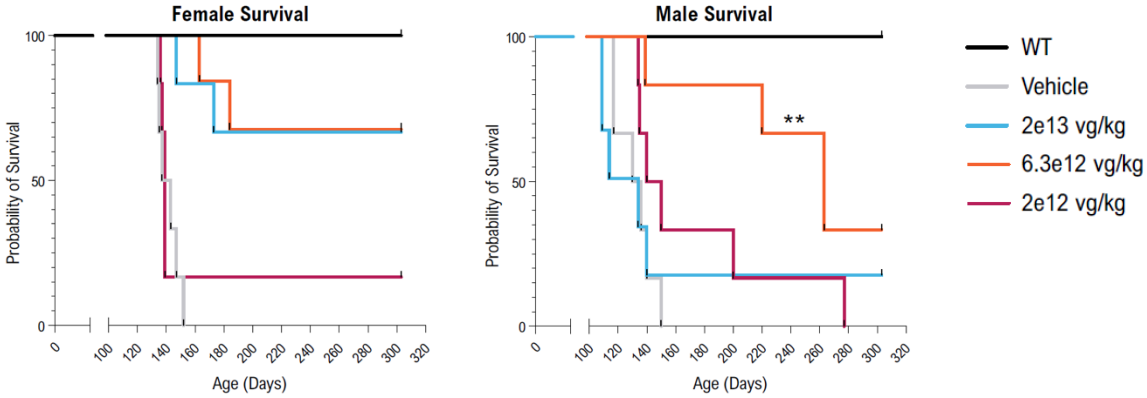
Strategy to combine highly potent siRNA construct with CNS-tropic, BBB penetrant TRACER capsid

- May enable broad CNS knockdown of SOD1, potentially addressing disease manifestations beyond the spinal cord

Promising preclinical results in mouse model

- Robust SOD1 knockdown in all levels of the spinal cord with IV dosing using a mouse BBB penetrant capsid
- Significant improvements in motor performance, body weight and survival

Increase in Survival



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GBA1 gene replacement; partnered with Neurocrine Jan 2023

HIGH UNMET NEED + COMMERCIAL POTENTIAL

~1 million people in U.S.*

>10% of PD patients have a GBA1 mutation

Potential to treat idiopathic PD

VALIDATED TARGET

GBA1

GBA1 mutations increase the risk of PD ~20-fold*

STATUS:

Lead optimization underway

EFFICIENT PATH TO PROOF-OF-BIOLOGY

GBA1 mutations decrease expression of GCase protein, leading to substrate elevation.

GCase and substrate measurable in CSF

ROBUST PRECLINICAL PHARMACOLOGY

Preclinical data demonstrate CNS target engagement and delivery of therapeutically relevant levels of GCase in GBA loss of function mouse model.

MILESTONE: Advancing in collaboration with Neurocrine

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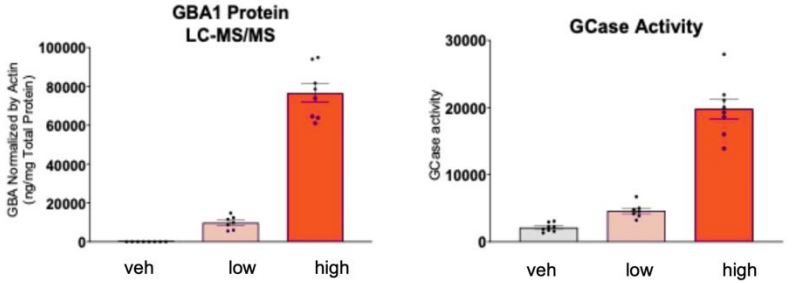
*Schapira et al., Mol Cell Neurosci, 2015; Sidransky et al., NEJM, 2009

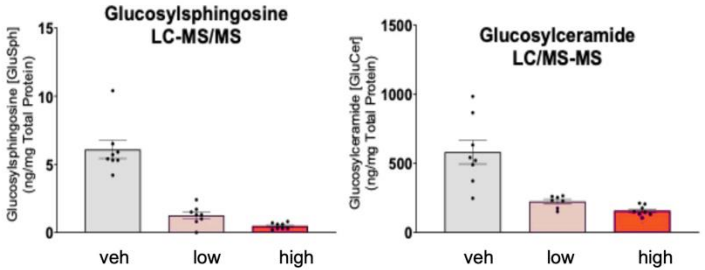
Restoration of GCase with IV-administered, BBB-penetrant AAV capsids

- BBB-penetrant AAV capsid provides *in vivo* proof-of-concept (POC) for increase in GCase with concomitant reduction of substrates
- Delivery of therapeutically relevant levels of GCase may disrupt disease process, and potentially slow neurodegeneration
- IV delivery with CNS-tropic capsid may enable widespread distribution to multiple affected brain regions and avoid need for more invasive approaches

GCase increased in GBA1 LOF mouse model



Substrate decreased in GBA1 LOF mouse model



Data shown reflects Day 0 IV dosing in GBA1 loss-of-function(LOF) mouse of 2E12, 2E13 vg/kg dosing of Voyager-optimized PHP.eB..GBA1, Day 28 necropsy with measures of GCase protein, activity, and substrate levels

HD gene therapy initiative leverages latest in disease biology

HIGH UNMET NEED + COMMERCIAL POTENTIAL

~41,000 people in U.S.*

Incidence: 0.7 in 100,000**

Currently no cure or treatment that can halt, slow or reverse HD*

VALIDATED TARGETS

Allele-specific mHTT

Target the mutant protein while preserving the healthy version, which may improve safety profile

MSH3

DNA repair enzyme potentially involved in harmful DNA expansions in the HTT gene

EFFICIENT PATH TO PROOF-OF-BIOLOGY

Leveraging emerging fluid-based biomarkers and imaging

ROBUST PRECLINICAL PHARMACOLOGY

First-in-class approach based on evolving research on the role of somatic expansion in HD

STATUS: Voyager is developing a vectorized siRNA approach to silence HTT allele-specifically and MSH3

MILESTONE: Early research initiative to determine if advancement warranted

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* Overview of Huntington's Disease - Huntington's Disease Society of America (hdsa.org)

** Modeling Manifest Huntington's Disease Prevalence Using Diagnosed Incidence and Survival Time - FullText - Neuroepidemiology 2021, Vol. 55, No. 5 - Karger Publishers

siRNA tau gene silencing approach for Alzheimer's disease

HIGH UNMET NEED + COMMERCIAL POTENTIAL

~6 million people in U.S.*

Multiple approaches needed: Like oncology, combination treatment may improve outcomes (i.e. anti-amyloid + anti-tau, or multiple anti-tau approaches)

VALIDATED TARGET

Tau

Pathology closely correlates with disease progression and cognitive decline

siRNA gene silencing approach to lower tau within neurons (intracellular)

EFFICIENT PATH TO PROOF-OF-BIOLOGY

New tau PET tracers enable imaging for tau pathology and use as clinical biomarkers

ROBUST PRECLINICAL PHARMACOLOGY

New initiative leverages Voyager's tau expertise to target intracellular tau; may complement extracellular tau antibody

STATUS:	Voyager is optimizing siRNA tau gene silencing payloads
MILESTONE:	Early research initiative to determine if advancement warranted

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*Alzheimer's Facts and Figures Report | Alzheimer's Association

Partnerships

Track Record of Non-Dilutive Revenue

Multiple partnership structures driving value

CAPSID LICENSES

PROVIDE:

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

Pfizer NOVARTIS

PROGRAM PARTNERSHIPS

PROVIDE:

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

NEUROCRINE BIOSCIENCES

CREATIVE COLLABORATIVE STRUCTURES

PROVIDE:

- Opportunities to combine TRACER capsids and receptor technology with cutting-edge payloads and biologics, placing Voyager at forefront of neurogenetic medicine
- Various deal structures with range of participation being explored

DISCUSSIONS ONGOING

January 2023: Voyager strategic collaboration with Neurocrine for GBA1+

VOYAGER RECEIVES:

Strengthens balance sheet	**Up-front consideration of $175M** ($136M cash, $39M equity purchase at 50% premium)
Secures program funding	**Program costs fully reimbursed***
Transformational downstream value	**Up to $4.2B in potential milestones** ($1.5B development, $2.7B commercial) **+ royalties [%]** • GBA: U.S. low double-digit to twenty; ex-U.S. high single-digit to mid-teen • Rare CNS targets: U.S. high single-digit to mid-teen; ex-U.S. mid single-digit to low double-digit **Option to elect 50/50 cost/profit sharing in U.S.** for GBA1 program following Phase 1

NEUROCRINE RECEIVES:

Worldwide rights to Voyager's GBA1 gene therapy program for Parkinson's disease and other GBA1-mediated diseases* and three gene therapy programs directed to rare CNS targets, each enabled by Voyager's next-generation TRACER™ capsids, as well as a Board seat.

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*Subject to Voyager election for a 50/50 cost/profit share for U.S. rights to GBA1 program

Existing partnership highlights

	Disease/Target (Cells, Tissues, Transgenes)	Upfront Payment	Potential Option + Option Exercise Fees	Potential Milestone Payments	Tiered Royalties
NEUROCRINE BIOSCIENCES	GBA1 Program + 3 undisclosed targets	$175 million ($136 million cash; $39 million equity	N/A	$4.2 billion	GBA, U.S. low double-digit to twenty; ex-U.S. high single-digit to mid-teen. Rare CNS targets, U.S. high single-digit to mid-teen; ex-U.S. mid single-digit to low double-digit
NOVARTIS	2 undisclosed CNS targets (expandable to 2 additional rare CNS targets)	$54 million	$25 million – exercised, $61 million potential expansion	$600 million for exercised targets, $600 million potential expansion	Mid- to high-single-digit
Pfizer	1 undisclosed rare neurologic disease target	$30 million	$10 million – exercised	$290 million	Mid- to high-single-digit
NEUROCRINE BIOSCIENCES	Friedreich's Ataxia + 2 undisclosed targets	$165 million ($115 million cash; $50 million equity)	N/A	$1.3 billion	High-single-digit to high-teens in the U.S. and mid-single-digit to mid-teens ex-U.S.

$200 MILLION in 2023 payments extended cash runway into 2025

Summary

Management team brings neurology and gene therapy expertise

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

Robin Swartz
Chief Operating Officer
SANOFI GENZYME

Todd Carter, Ph.D.
Chief Scientific Officer
BROAD INSTITUTE

Peter Pfreundschuh
Chief Financial Officer
FREQUENCY THERAPEUTICS

Allen Nunnally
Chief Business Officer
FOUNDATION MEDICINE

Michelle Quinn Smith
Chief Human Resources Officer
Flagship Pioneering

Robert Hesslein
General Counsel
FOUNDATION MEDICINE

Trista Morrison
SVP Corporate Affairs
saniona

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Recent highlights and upcoming milestones

Q3 2022 ✓	**Pfizer option exercised** on capsid for rare neurologic target; $10M payment
Q1 2023 ✓	**Neurocrine collaboration** for GBA1 + 3 discovery programs; $175M payment, potential $4.4B deal
Q1 2023 ✓	**Launched updated HD early research initiative:** allele-specific mHTT + MSH3 gene silencing
Q1 2023 ✓	**Selected lead candidate** in anti-tau antibody program for Alzheimer's disease
Q1 2023 ✓	**Novartis option exercised** on capsids for two CNS targets; $25M payment
Q1 2023 ✓	**Launched Alzheimer's early research initiative:** tau gene silencing
Q1 2023 ○	AD/PD 2023 Conference: presenting data on GBA1 Parkinson's and anti-tau antibody programs
H1 2023 ○	Expect to ID lead candidate for SOD1 ALS gene therapy
ONGOING ○	Advancing GBA1 Parkinson's and FXN Friedreich's Ataxia gene therapy programs with Neurocrine
ONGOING ○	Potential for additional value-creating partnerships; discussions ongoing

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