UNITED STATES
SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): January 12, 2026

PASSAGE BIO, INC.

(Exact name of registrant as specified in its charter)

Delaware	**001-39231**	**82-2729751**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

One Commerce Square **2005 Market Street, 39th Floor** **Philadelphia, PA**	**19103**
(Address of principal executive offices)	**(Zip Code)**

(267) 866-0311
(Registrant's telephone number, including area code)

N/A
(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading symbol(s)	Name of each exchange on which registered
Common Stock, $0.0001 Par Value Per Share	PASG	The Nasdaq Stock Market LLC (Nasdaq Global Select Market)

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 2.02 Results of Operations and Financial Condition.

On January 12, 2026, Passage Bio, Inc. (the "***Company***") updated its corporate presentation, which reports the preliminary, unaudited amount of the Company's cash, cash equivalents and marketable securities position as of December 31, 2025, as approximately $46 million, which the Company expects will enable it to fund its operating expenses and capital expenditure requirements into the first quarter of 2027. This amount is preliminary, unaudited and may change, was prepared by management and is based on the most current information available to management, and is subject to completion by management of the financial statements as of and for the year ended December 31, 2025, including completion of the review procedures, final adjustments and other developments that may arise between now and the time the financial results for this period are finalized, and completion of the audit of such financial statements.

The information in this Item 2.02, including Exhibit 99.1 to this report, shall not be deemed to be "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "***Exchange Act***"), or otherwise subject to the liabilities of that section or Sections 11 and 12(a)(2) of the Securities Act of 1933, as amended (the "***Securities Act***"). The information contained in this Item 2.02 and in the accompanying Exhibit 99.1 shall not be incorporated by reference into any other filing under the Exchange Act or under the Securities Act, except as shall be expressly set forth by specific reference in such filing.

Item 7.01 Regulation FD Disclosure.

On January 12, 2026, the Company updated its corporate presentation. A copy of the corporate presentation is attached as Exhibit 99.1 to this Current Report on Form 8-K.

The information in this Item 7.01, including Exhibit 99.1 to this Current Report on Form 8-K, shall not be deemed to be "filed" for purposes of Section 18 of the Exchange Act, or otherwise subject to the liabilities of that section or Sections 11 and 12(a)(2) of the Securities Act. The information contained in this Item 7.01 and in the accompanying Exhibit 99.1 shall not be incorporated by reference into any other filing under the Exchange Act or under the Securities Act, except as shall be expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits.

(d)Exhibits

Exhibit No.	Description
99.1	Corporate Presentation.
104	Cover Page Interactive Data File (formatted as Inline XBRL).

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

PASSAGE BIO, INC.

Date: January 12, 2026

By: /s/ Kathleen Borthwick
Kathleen Borthwick
Chief Financial Officer

Exhibit 99.1

PassageBio

Redefining the Course of Neurodegenerative Conditions

Corporate Presentation

January 2026

PASG
LISTED
NASDAQ

Forward-Looking Statement

This presentation includes "forward-looking statements" within the meaning of, and made pursuant to the safe harbor provisions of, the Private Securities Litigation Reform Act of 1995, including, but not limited to: our expectations about timing and execution of anticipated milestones, including the progress of clinical studies and the availability of clinical data from such trials; timing of feedback from regulatory authorities; the potential of our product candidates versus other treatment options and clinical candidates; our expectations about cash runway; the ability of PBFT02 to treat FTD-*GRN* or FTD-*C9orf72*, and the potential development of other product candidates. These forward-looking statements may be accompanied by such words as "aim," "anticipate," "believe," "could," "estimate," "expect," "forecast," "goal," "intend," "may," "might," "plan," "potential," "possible," "will," "would," and other words and terms of similar meaning. These statements involve risks and uncertainties that could cause actual results to differ materially from those reflected in such statements, including: our ability to develop and obtain regulatory approval for our product candidates; the timing and results of preclinical studies and clinical trials; risks associated with clinical trials, including our ability to adequately manage clinical activities, unexpected concerns that may arise from additional data or analysis obtained during clinical trials, the timing of and our ability to obtain and maintain regulatory approvals; our expectations about the willingness of healthcare professionals to use our product candidates, the timing, or amount, the occurrence of adverse safety events; the risk that positive results in a preclinical study or clinical trial may not be replicated in subsequent trials or success in early stage clinical trials may not be predictive of results in later stage clinical trials; failure to protect and enforce our intellectual property, and other proprietary rights; our dependence on collaborators and other third parties for the development and manufacture of product candidates and other aspects of our business, which are outside of our full control; the timing, or amount, of receipt of any potential future milestone and royalty payments; risks associated with current and potential delays, work stoppages, or supply chain disruptions; and the other risks and uncertainties that are described in the Risk Factors section in documents the company files from time to time with the Securities and Exchange Commission (SEC), and other reports as filed with the SEC. Passage Bio undertakes no obligation to publicly update any forward-looking statement, whether written or oral, that may be made from time to time, whether as a result of new information, future developments or otherwise.

PassageBio

Redefining the Course of Neurodegenerative Conditions

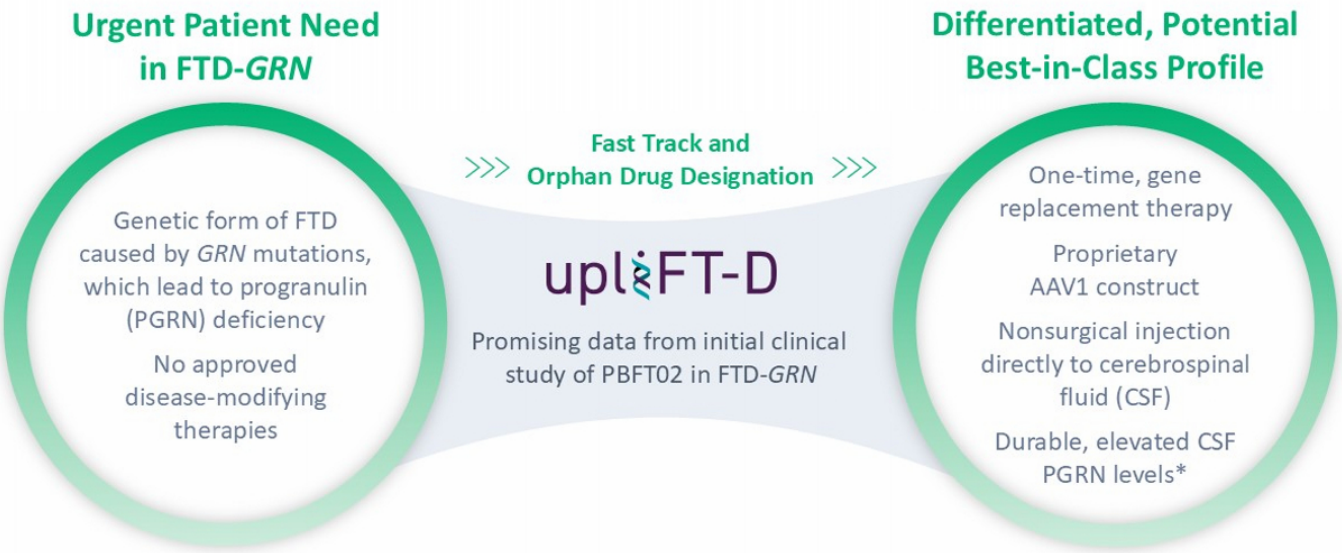
Advancing clinical stage, potential best-in-class, one-time progranulin raising gene therapy for FTD

Pursuing preclinical development of differentiated gene therapy approach in Huntington's disease

Cash runway expected into 1Q 2027*

* Based on cash, cash equivalents, and marketable securities as of December 31, 2025.

Validating the Therapeutic Potential of PBFT02



* Based on interim data.

Significant Market Opportunity Addressing Neurodegenerative Diseases

1. Greaves CV, et al. *J Neurol* 2019; 266:2075-2086. 2. Galvin JE, et al. *Neurology* 2017; 89:2049-2056. 3. Onyike CU, et al. *Int Rev Psychiatry* 2013; 25:130-137.
4. Moore KM, et al. *Lancet Neurol* 2020; 19: 145–156. 5. Crowell et al. Neuroepi. 2021; 55:361-368.

PassageBio

PBFT02
Frontotemporal Dementia

PassageBio

Frontotemporal Dementia (FTD): A Devastating Adult Disease

OVERVIEW

- Fatal adult-onset neurodegenerative disease affecting the frontal and temporal lobes of the brain, characterized by a decline in behavior, language, and executive function
- One of the most common causes of early-onset dementia worldwide, disproportionately affecting individuals aged 40–65 years

CLINICAL SYMPTOMS

Disease progression is rapid and degenerative, including loss of speech, loss of expression, behavioral changes, and immobility

Loss of inhibition

Apathy

Social withdrawal

Hyperorality (mouthing of objects)

Ritualistic compulsive behaviors

On average, people with FTD live 8 years after the onset of symptoms

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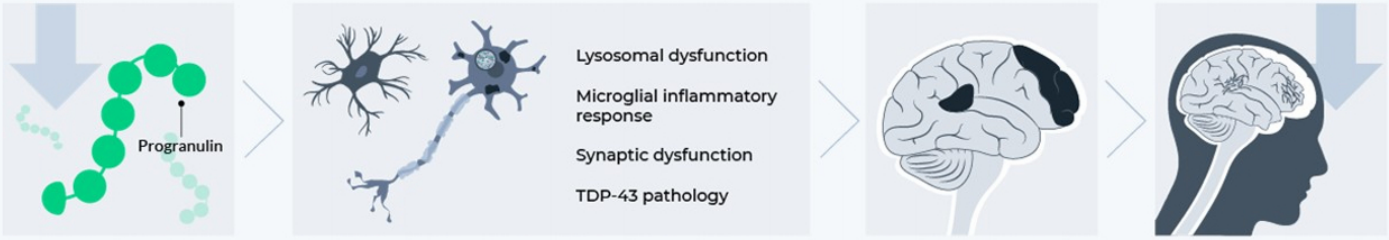
Progranulin Deficiency is the Defining Characteristic of FTD-*GRN* and Leads to Neurodegeneration

Progranulin is critical to maintaining CNS cell homeostasis



Decrease in PGRN levels

Neuronal dysfunction, pathological changes, and inflammation

Vulnerability of neurons in affected regions

Neurodegeneration

Rhinn H et al. *Trends Pharmacol Sci.* 2022, 43:641-652.

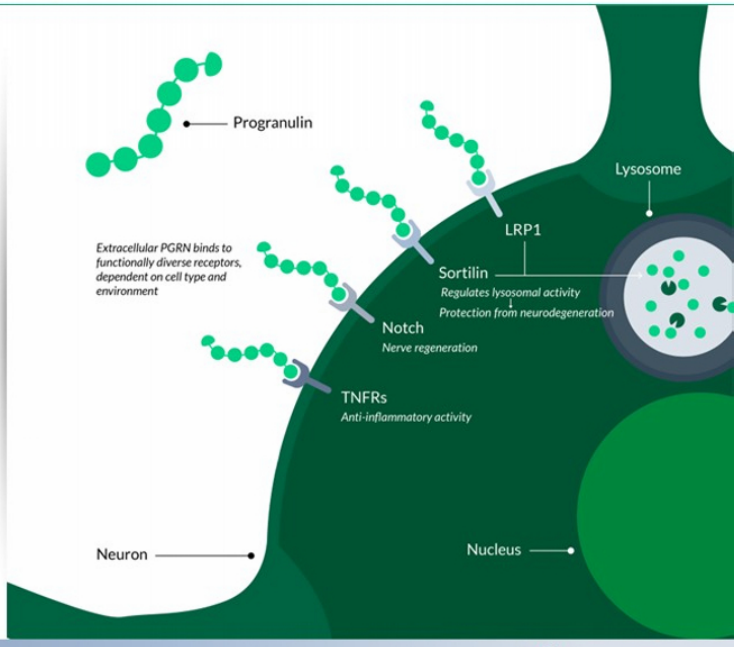
Elevated PGRN Increases Potential for Improved Cellular Function

Progranulin is a secreted protein that binds to cell membrane receptors to affect multiple intracellular pathways

- Major role is regulating intracellular lysosomal activity
- Extracellular PGRN is endocytosed via multiple receptors

Driving elevated PGRN levels in the extracellular space increases the amount of PGRN available to enter target CNS cells

Able to leverage cross-correction mechanism: secreted PGRN can be taken up by non-transduced cells



Paushter et al. *Acta Neuropathol*. 2018;136:1-17. Rhinn et al. *Trends Pharmacol Sci*. 2022; 43:641-652.

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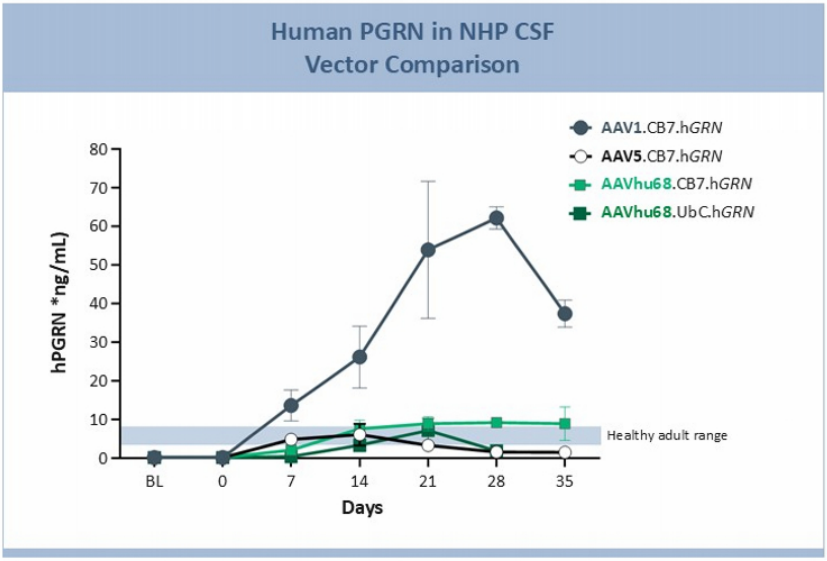
Preclinical NHP: AAV1 Achieved the Highest Levels of CSF PGRN

After ICM administration to NHPs:

- AAV1 capsid resulted in CSF hPGRN levels 5x higher than AAVhu68 (an AAV9 variant)
- Superior hPGRN response led to selection of AAV1 capsid for PBFT02



Rhesus macaques (n=2/gp) ICM-delivered AAV.hPGRN (3.3 x 10^{11} GC/g brain), day 0

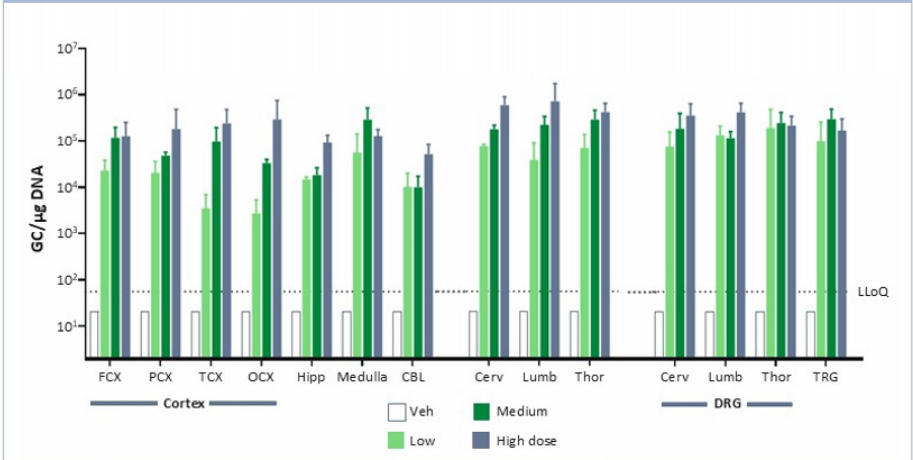
*Size and duration of elevation muted by immune response to human PGRN. *Shading:* Healthy adult sample range for CSF PGRN, n = 61 (Passage Bio data)

CSF, cerebrospinal fluid; **GC**, genome copies; **ICM**, intra-cisterna magna; **NHP**, non-human primate. *Reference:* Hinderer et al., *Ann Clin Trans Neurol.* 2020; 7:1843-1853.

PassageBio

Preclinical NHP: ICM Administration of PBFT02 Led to Broad Distribution of Vector Throughout Brain/Spinal Cord

Vector Biodistribution in NHPs 90 Days Post-ICM PBFT02

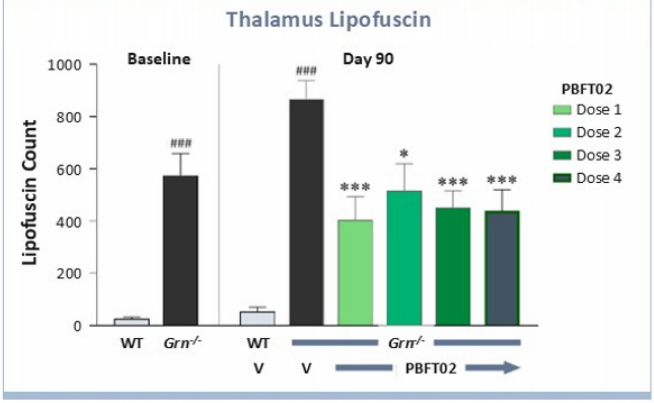


- Robust, dose-dependent vector delivery to cortical and sub-cortical brain regions affected in FTD
- NHP low dose, equivalent to clinical Dose 1 of PBFT02 in upliFT-D study, resulted in ~10^4 GC/µg DNA in all sampled areas throughout the brain

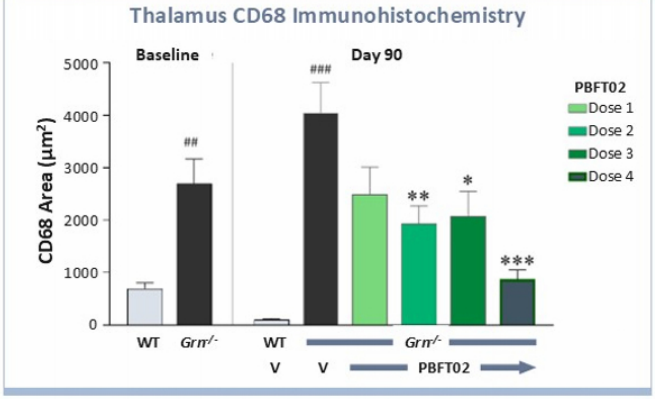
n=3/gp. Data are mean +/- SEM.
CBL, cerebellum; Cerv, cervical; DRG, dorsal root ganglion; FCX, frontal cortex; GC, genome copies; Hipp, hippocampus; ICM, intra-cisterna magna; LLoQ, lower limit of quantitation; Lumb, lumbar; OCX, occipital cortex; PCX, parietal cortex; TCX, temporal cortex; Thor, thoracic; TRG, trigeminal root ganglion; Veh, vehicle

Preclinical Grn–/– Mice: Expression of hPGRN Improved Lysosomal Dysfunction and Neuroinflammation in the Brain

PBFT02 Reduced Lipofuscin Deposition at All Doses, Suggesting Improved Lysosomal Dysfunction



Dose-Dependent Elevations in CSF PGRN after PBFT02 Led to Progressive Reductions in Microglial Activation



Greatest pathological benefit was associated with the highest PGRN levels in the CSF

Lipofuscin deposition and microglial activation are hallmark pathologies seen in FTD; Improvements in both measures were seen in cerebral cortex, thalamus, and hippocampus after PBFT02 administration

Grn$^{-/-}$ and WT mice (n=14-15/gp) ICV-administered PBFT02 or vehicle (V). Baseline controls were untreated mice on Day 1. Bars: mean +/- SEM.

$^{\#\#}p < 0.01$, $^{\#\#\#}p < 0.005$ vs WT control; $^{*}p < 0.05$, $^{***}p < 0.005$ vs *Grn*$^{-/-}$+ V , one-way ANOVA followed by Tukey's multiple comparisons test.

Grn, granulin gene; **ICV**, intra-cerebroventricular; **PGRN**, progranulin; **WT**, wildtype

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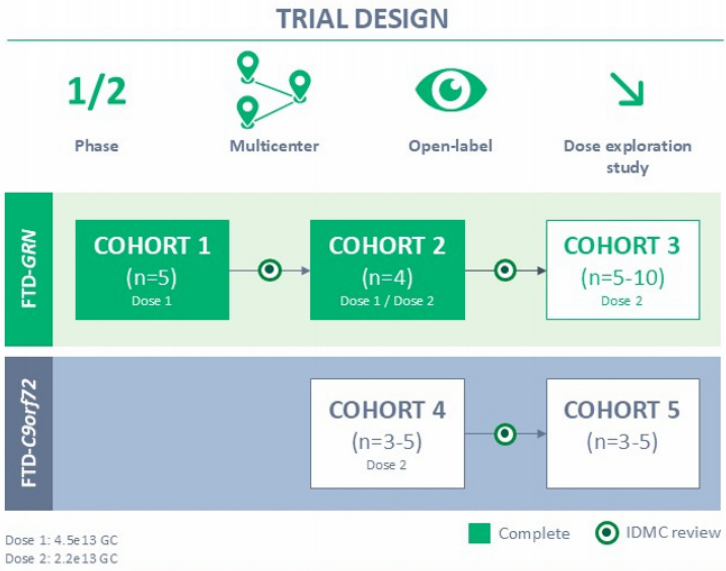
upliFT-D: Global Phase 1/2 Trial with PBFT02

Currently enrolling patients in Cohort 3 and Cohort 4

TRIAL DESIGN



Dose 1: 4.5e13 GC
Dose 2: 2.2e13 GC

DURATION
2 years; with additional 3 years of follow-up for safety and durability of effect

PRIMARY ENDPOINTS
Safety and tolerability

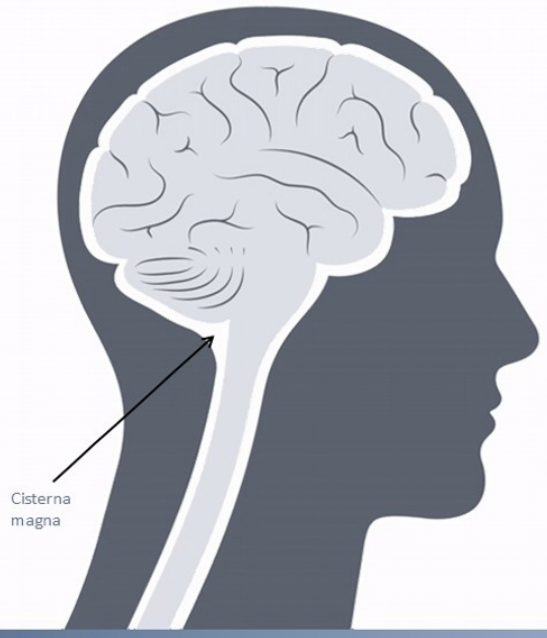
SECONDARY ENDPOINTS

Biomarkers
- Progranulin (CSF, plasma)
- vMRI
- Retinal nerve fiber layer and retinal lipofuscin deposits via OCT
- NfL (CSF, plasma)

Clinical
- CDR + NACC FTLD sum of boxes

EXPLORATORY BIOMARKERS
- Cathepsin D (CSF)
- GFAP (CSF, plasma)
- LAMP 1 (CSF)
- Lys-GL1 (CSF)

Intra-Cisterna Magna (ICM) Administration



Directly deliver vector into the CSF via a single injection

- Allows for broad CNS biodistribution[1]
- Lower doses compared to IV systemic delivery
- Reduced impact of neutralizing antibodies

Brief (<60 min), non-surgical, CT-guided procedure for precise delivery to the cisterna magna

Procedure avoids penetration of brain tissue

1. Hinderer et. al, *Hum Gene Ther.* 2018; 29:15-24.

Key Baseline Demographics for FTD-*GRN* Participants*

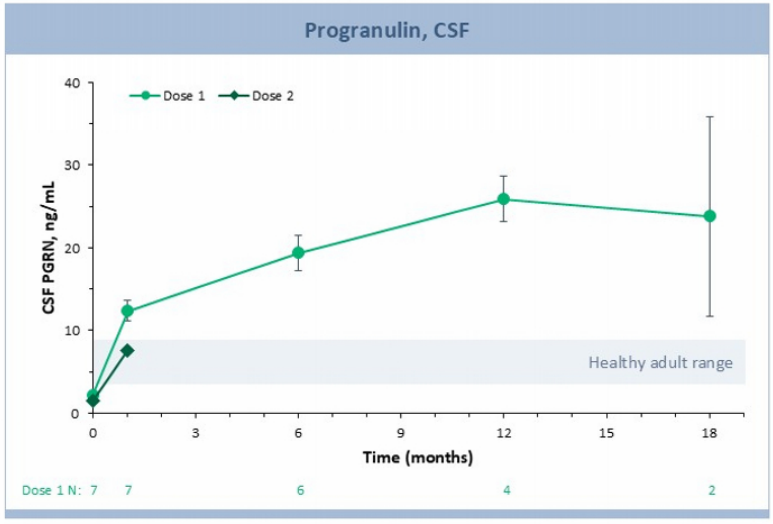
Dose 1 (n=7); Dose 2 (n=1)

(n=8)	Mean / % / n	Range
Age (yrs)	64.4	51–72
Sex	M: 50% F: 50%	
FTD-*GRN* phenotype (n)	bvFTD: 5 PPA: 3	
Disease duration at baseline (yrs)	2.9	1 –5
PGRN, CSF (ng/mL)	2.1	1.5 –2.9
PGRN, plasma (ng/mL)	36.6	22.4 –89.0
NfL, plasma (pg/mL)	51.9	12.4 –111
Clinical Dementia Rating Scale[1], Global (%)	1: 50% 2: 50%	
Clinical Dementia Rating Scale[1], Sum of Boxes	10.3	5–17

*Data as of June 15, 2025
1. CDR® +NACC FTLD.
bvFTD, behavioral variant; **PPA**, primary progressive aphasia.

PBFT02 Generated Robust, Durable Increases in CSF PGRN in FTD-*GRN* Patients

upliFT-D



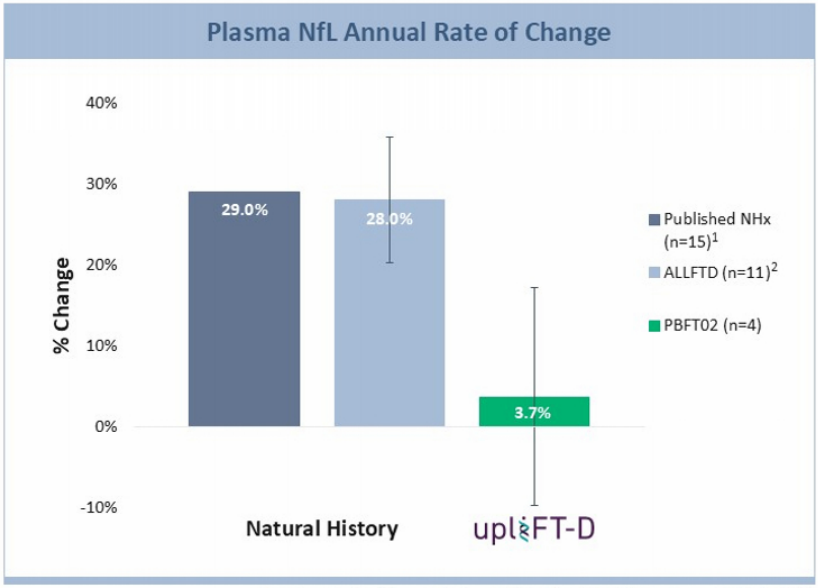
First **Dose 2** patient (Patient 8) increased from 1.5 ng/mL at baseline to 7.6 ng/mL at M1, approaching the upper healthy adult range

Data as of June 15, 2025. Mean +/- SEM
Shading: Healthy adult sample range for CSF PGRN (range: 3.28 – 8.15 ng/mL, mean: 4.76 ng/mL, n = 61) (Passage Bio data)
Dose 1: 4.5e13 GC; Dose 2: 2.2e13 GC
CSF, cerebrospinal fluid; **M**, month.

Plasma NfL Showed Early Evidence of Improvement in a Disease Progression Biomarker vs. Natural History

upliFT-D

- Plasma NfL is the only FTD-GRN disease progression biomarker with longitudinal natural history data available[1,3]
- PBFT02-treated patients with 12 months follow-up (n=4) had a reduced annual rate of change in plasma NfL compared to published natural history data



Note: Annual rate of increase in plasma NfL in a healthy adult sample reported to be ~4%[1].

Data as of June 15, 2025. Mean +/- SEM
PBFT02 patients (n=4, Dose 1): Baseline plasma NfL (neurofilament light chain) range: 39.6 to 45.6 pg/mL. Average time since diagnosis 2.3 years.
1. Published natural history. Chart (left): 15 symptomatic, untreated FTD-*GRN* patients; mean years since diagnosis: 2.9; Note (right): 65 healthy adults. (Saracino et al, *J Neurol Neurosurg Psych* 2021; 92:1278-1288). **2.** Passage Bio analysis of ALLFTD natural history sample comprised of individuals with a pathogenic *GRN* mutation and a CDR+NACC FTLD global score between 0.5 and 2, inclusive. **3.** van der Ende et al, *Lancet Neurol* 2019; 18:1103-11.

PBFT02 Interim Safety Profile

Data as of June 15, 2025

PBFT02 was generally well tolerated

- SAEs related to PBFT02 all asymptomatic; all occurred at Dose 1
  - Venous sinus thrombosis (2)
  - LFT increase (1)
- No SAEs related to PBFT02 at Dose 2
- No evidence of thrombotic microangiopathy
- No evidence of dorsal root ganglion (DRG) toxicity in any patient
- No complications from ICM procedure

	N	Events
TEAE considered related to PBFT02	7	26
Serious TEAE unrelated to PBFT02	1	1
Serious TEAE related to PBFT02	2	3

SAE, serious adverse event; LFT, liver function test; ICM, intracisterna magna; TEAE, treatment emergent adverse event.

PBFT02 Offers Best-in-Class Therapeutic Potential

	PBFT02		
Product Candidate	**AAV1 gene therapy delivering *GRN***	**AAV9 gene therapy delivering *GRN***	**AAV9 gene therapy delivering *GRN***
Stage of Development	Phase 1/2	Phase 1/2	Phase 1/2
Route of Administration	ICM (non-surgical, 1 hour procedure)	ICM	Intrathalamic (neurosurgery, lengthy procedure)
CSF PGRN Level at 12m[1]	**26 ng/mL (mean; n=4)**	~4-8 ng/mL (n=7 higher dose)[2]	–
Durability of CSF PGRN Elevation[1]	**Durable at 18 m (n=2)**	Declining from 2 to 12 m (n=7 higher dose)[2]	–

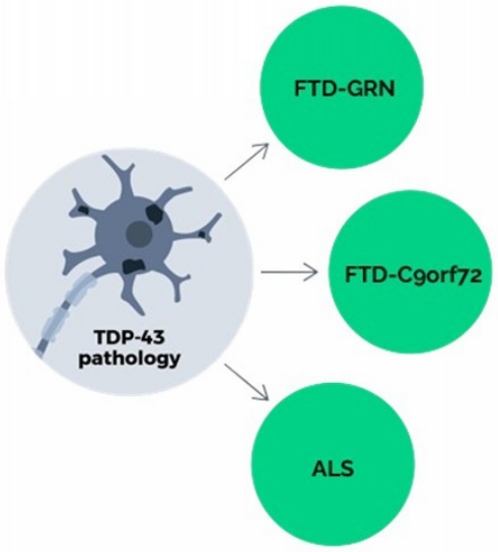
PBFT02 uniquely positioned to offer a one-time therapy capable of achieving highest progranulin levels

1. Clinical evidence based on public disclosure. Results are derived from different clinical trials at different points in time. No head-to-head trials have been conducted among the results shown. Comparing the results from different trials may be unreliable due to different protocol designs, trial design, patient selection and populations, number of patients, trial endpoints, trial objectives and other parameters that may not be the same between trials. **2.** Lilly/Prevail AD/PD Mar 2024 presentation and abstract.

PBFT02 has Potential to Correct Underlying Pathology in FTD-GRN, FTD-C9orf72 and ALS

TDP-43 pathology is a hallmark of multiple neurodegenerative diseases[1]

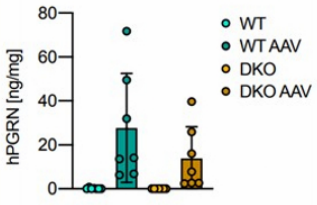
- TDP-43 mislocalizes from nucleus to cytoplasm
- Forms inclusion bodies associated with neurodegeneration



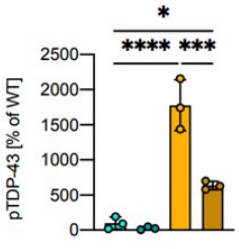
Elevated PGRN Ameliorates TDP-43 Pathology in Preclinical Models

TDP-43 pathology due to lysosomal dysfunction (*GRN*/ *TMEM106* double knockout, DKO) reduced by AAV.hPGRN[1]

AAV delivered hPGRN to mouse brain

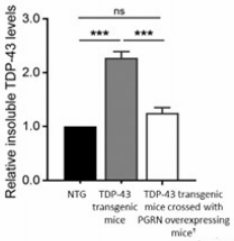


TDP-43 pathology in DKO mice reduced by AAV.hPGRN

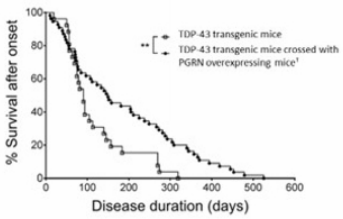


Elevated PGRN ameliorated TDP-43 pathology and disease course in a preclinical model[2]

Elevated PGRN reduced insoluble TDP-43 in mouse spinal cord



Elevated PGRN extended survival of TDP-43 mutant mice



† *PGRN increased to >2x endogenous levels*

- Elevated PGRN also prevented degeneration of large axon fibers in TDP-43 mice
- PGRN neuroprotection from pleiotropic effect, not single pathway

1. Reich et al. *Sci Transl Med.* 2024; 16(750); 2. Beel et al. *Mol Neurodegen.* 2018: 13:55.; Laird et al. *PLoS One* 2010; 5:e13368.
DKO, double gene knockout; *GRN*, granulin gene; PGRN, progranulin; TDP-43, transactive response DNA binding protein 43 kDa

PassageBio

PBFT02: Summary of Approach

- AAV delivery of functional GRN gene to **express new PGRN, increasing levels both intra- and extra-cellularly**
  - Preserves all natural pathways to properly traffic PGRN intracellularly where it is needed
- ICM route of administration enables **low doses of AAV and broad CNS biodistribution**
  - Non-surgical, brief procedure (< 60 minutes)
- Promise of a **one-time therapy** for patients
  - Durable elevation of CSF PGRN[1]

1. Interim data from upliFT-D as of June 15, 2025.

Huntington's Disease Preclinical Program

PassageBio

Huntington's Disease: A Fatal Neurodegenerative Disease with No Disease-Modifying Therapy

OVERVIEW

- Fatal, monogenic, autosomal dominant neurodegenerative disease
- Caused by trinucleotide (CAG) expansion in the huntingtin (*HTT*) gene resulting in mutant huntingtin (mHTT) protein expression
- More than 200,000 people estimated to be at risk in the US[1]

CLINICAL SYMPTOMS

- Symptom onset typically occurs between 30–50 years old
- Characterized by progressive motor, cognitive, and behavioral deterioration, due to neuronal dysfunction then degeneration

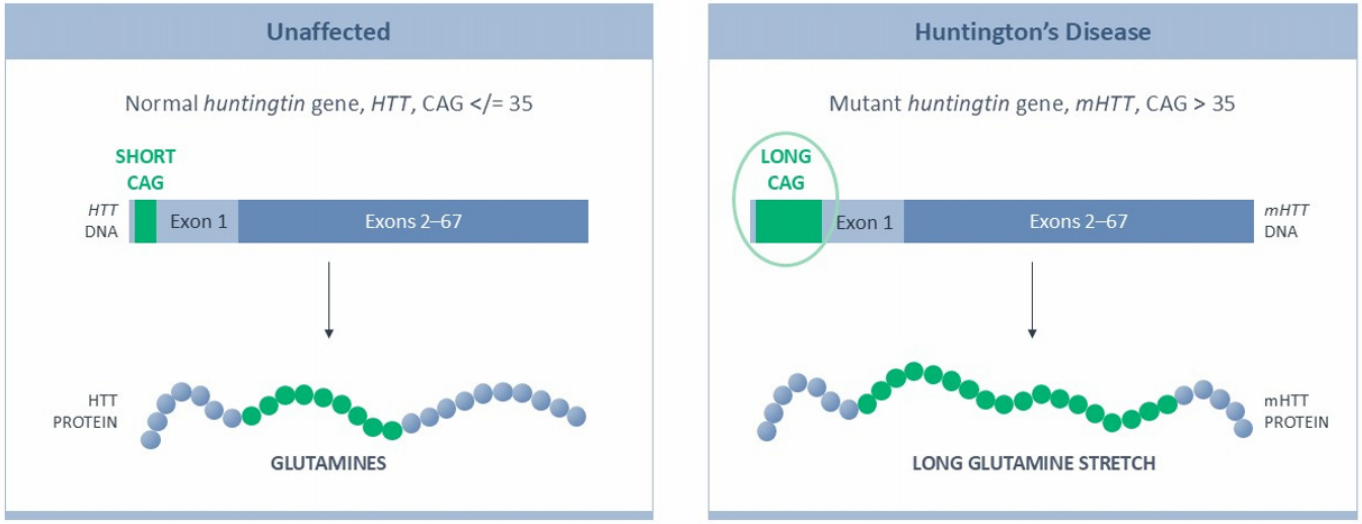
Average life expectancy after symptom onset is 15–20 years

1. HDSA; Fisher and Hayden Mov Disord. 29:105-14, 2014.

PassageBio

HD Is Caused By A Mutation In *Huntingtin* (*HTT*) Gene: A Stretch Of CAG Nucleotides Is Expanded

CAG repeat expansion leads to production of mutant huntingtin protein (mHTT)



PassageBio

DNA Repair (DR) Proteins Play a Key Role in CAG Repeat Expansion

In Huntington's disease (HD), the CAG repeat in the *HTT* gene can elongate over time, termed *somatic instability*

- CAG expansion above a certain threshold leads to neurodegeneration
- Longer CAG repeats associated with worse disease pathology
- CAG expansion occurs at different rates in different neurons, and is fastest in the caudate and putamen brain regions which degenerate first

MSH3, a DR protein, is a key driver of somatic instability

- In the presence of certain CAG motifs, MSH3 can erroneously incorporate CAGs into DNA, leading to CAG expansion
- In HD: certain genetic variants altering MSH3 function are associated with delayed onset and slowed progression[1]
- In HD mice: MSH3 is essential for CAG expansion, and MSH3 knock-down reduced somatic instability and HTT pathology[2,3]

1. Genetic Modifiers of Huntington's Disease (GeM-HD) Consortium, *Nat Genet*. 2025; 57:1426-36.
2. Dragileva E et. al, *Neurobio Dis*. 2009; 33:37-47. **3.** Mouro Pinto et. al, *Nat Genet*. 2025; 57:314-322.

Our Approach: Decrease MSH3 to Reduce Somatic Instability in the *HTT* Gene

Program Status

Developing a differentiated approach to decrease MSH3 expression via AAV delivery of a miRNA

Plan to utilize an optimized intraparenchymal delivery approach

- One-time delivery
- Direct delivery to critical brain regions
- Reduced total procedure time
- Limited peripheral exposure to reduce safety risks

Proof-of-concept studies completed, with additional preclinical studies ongoing

Expect to declare a clinical candidate in 2H 2026

Looking Ahead

PassageBio

Critical Manufacturing Milestones Achieved to Enable Late-Stage Development of PBFT02

Functional Potency Assay

Developed assay and reached alignment with FDA on suitability of assay for PBFT02 release

Robust Manufacturing Process

Completed development of high-productivity, suspension-based manufacturing process

Single production lot estimated to yield >1,000 doses[1] with >70% full capsids

Process Comparability Plan

Aligned with the FDA on an analytical approach to establish comparability of suspension-based process

1. Estimated yield based on Dose 2.

Plan to Initiate Discussions with the FDA on a Registrational Study Design in 1H 2026

Rationale for a single-arm registrational study

1. FTD-*GRN* is a rapidly progressing disease with no approved disease-modifying therapies and a substantial unmet clinical need
2. Multiple recent gene therapy precedents demonstrate FDA receptivity to a single-arm registrational approach
3. Existing, well-structured FTD natural history studies with >300 FTD-*GRN* patients

Upcoming Milestones and Corporate Updates

TIMING	MILESTONE
1H 2026	Report updated interim safety and biomarker data from Dose 2 in FTD patients
1H 2026	Seek regulatory feedback on registrational trial design in FTD-*GRN*
2H 2026	Declare clinical candidate for Huntington's disease

BALANCE SHEET

- Cash balance of ~$46 million as of 12/31/25*
- Cash runway into 1Q 2027

* Based on cash, cash equivalents and marketable securities.

PassageBio

Redefining the Course of Neurodegenerative Conditions

Advancing clinical stage, potential best-in-class, one-time progranulin raising gene therapy for FTD

Pursuing preclinical development of differentiated gene therapy approach in Huntington's disease

Cash runway expected into 1Q 2027*

* Based on cash, cash equivalents and marketable securities as of December 31, 2025.

PassageBio

Thank you!

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Focused Pipeline Addressing Rare and Prevalent Neurodegenerative Indications

1. Greaves CV, et al. *J Neurol* 2019; 266:2075-2086. **2.** Galvin JE, et al. *Neurology* 2017; 89:2049-2056. **3.** Onyike CU, et al. *Int Rev Psychiatry* 2013; 25:130-137. **4.** Moore KM, et al. *Lancet Neurol* 2020; 19: 145–156. **5.** Brown et al. *Neuroepi* 2021; 55:342-353. **6.** CDC ALS Registry Dashboard. **7.** Sheng J, et al. *Gene* 2014; 141-145. **8.** Alz Assoc. 2023 Alzheimer's Disease Facts and Figures. Alzheimers Dement 2023;19. **9.** Crowell et al. *Neuroepi*. 2021; 55:361-368.

Demonstrated Leadership

Deep experience in rare disease, CNS disorders and genetic medicines

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