BEAM-302 Clinical Data at ERS Congress 2026

September 8, 2026

NASDAQ: BEAM

Beam THERAPEUTICS

Today's agenda

TOPIC	PARTICIPANT
Introduction	**Holly Manning** Vice President, Investor Relations & External Communications
Beam Overview	**John Evans** Chief Executive Officer
AATD Overview and BEAM-302 Clinical Program	**Amy Simon, M.D.** Chief Medical Officer
Updated BEAM-302 Data at ERS	**Gerry McElvaney, M.D.** RCSI Education & Research Centre, Beaumont Hospital
Closing Remarks	**Giuseppe Ciaramella, Ph.D.** President
Q&A	**Mr. Evans, Dr. Simon, Dr. McElvaney & Dr. Ciaramella**

Cautionary note regarding forward-looking statements

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Such forward-looking statements include statements regarding: the therapeutic applications and potential of our technology, including with respect to AATD; our plans, and anticipated timing, to advance our programs, including risto-cel, BEAM-103, BEAM-301, BEAM-304 and BEAM-302; our plans and anticipated timing to present data from ongoing clinical trials; our anticipated regulatory interactions and filings; our current expectations and anticipated results of operations, including our expected use of capital; the sufficiency of our capital resources to fund operating expenses and capital expenditure requirements and the period in which such resources are expected to be available; and the therapeutic applications and potential of our technology, including our potential to develop lifelong, curative, precision genetic medicines for patients through base editing, including potential safety advantages, all of which are subject to known and unknown important risks, uncertainties and other factors that may cause our actual results, performance or achievements, market trends, or industry results to differ materially from those expressed or implied by such forward-looking statements. Therefore, any statements contained herein that are not statements of historical fact may be forward-looking statements and should be evaluated as such. Without limiting the foregoing, the words "anticipate," "expect," "suggest," "plan," "vision," “strategy,” “possibility,” “promise,” "believe," "intend," "project," "forecast," "estimates," "targets," "projections," "potential," "should," "could," "would," "may," "might," "will," and the negative thereof and similar words and expressions are intended to identify forward-looking statements. Each forward-looking statement is subject to important risks and uncertainties that could cause actual results to differ materially from those expressed or implied in such statement, including, without limitation, risks and uncertainties related to: our ability to develop, obtain regulatory approval for, and commercialize our product candidates, which may take longer or cost more than planned; our ability to raise additional funding, which may not be available; our ability to obtain, maintain and enforce patent and other intellectual property protection for our product candidates; that preclinical testing of our product candidates and preliminary or interim data from preclinical studies and clinical trials may not be predictive of the results or success of ongoing or later clinical trials; that initiation and enrollment of our clinical trials may take longer than expected; that our product candidates or the delivery modalities we rely on to administer them may cause serious adverse events; the uncertainty that our product candidates will receive regulatory approval necessary to initiate or continue human clinical trials, that our product candidates may experience manufacturing or supply interruptions or failures; risks related to competitive products; and the other risks and uncertainties identified under the headings "Risk Factors Summary" and "Risk Factors" and elsewhere in our annual report on Form 10-K for the year ended December 31, 2025, our quarterly reports on Form 10-Q, and in any subsequent filings with the Securities and Exchange Commission (the "SEC") which are available on the SEC's website at www.sec.gov. Additional information will be made available by our annual and quarterly reports and other filings that we make from time to time with the SEC. These forward-looking statements speak only as of the date of this presentation. Factors or events that could cause our actual results to differ may emerge from time to time, and it is not possible for us to predict all of them. We undertake no obligation to update any forward-looking statement, whether as a result of new information, future developments or otherwise, except as may be required by applicable law.

Our vision is to provide lifelong cures
for patients suffering from serious diseases

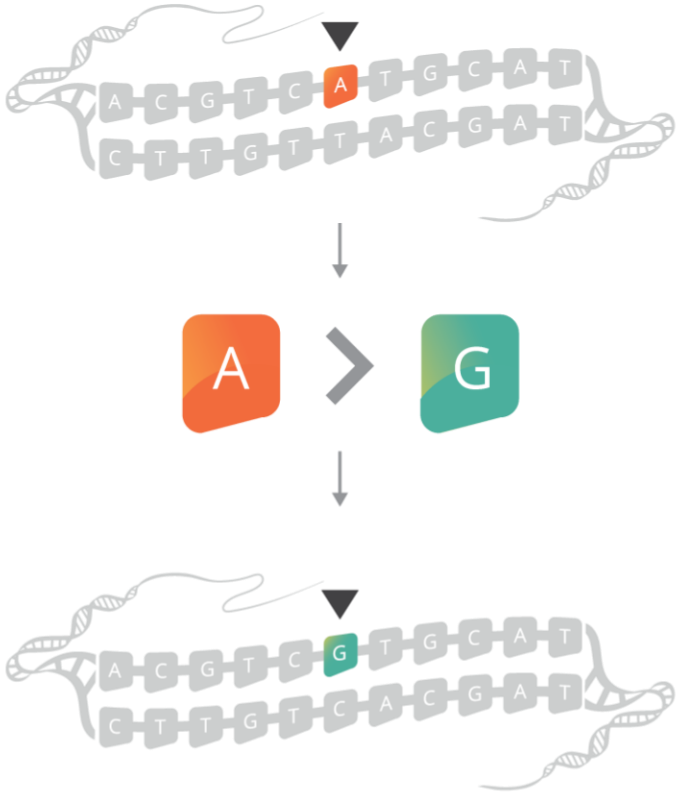
GENE EDITING FOR
rare and common diseases

POTENTIAL FOR
one-time, curative therapies

PLATFORM FOR
rapidly programmable precision medicines

Beam was founded on a simple concept with profound implications

BASE EDITING
TECHNOLOGY



CONSISTENT gene sequence outcomes

DURABLE correction for one-time cures

LESS GENOTOXICITY than traditional gene editing

Predictable, Reproducible Outcomes for Patients

The power of predictability:
Beam is building a reliable model for advancing genetic medicine

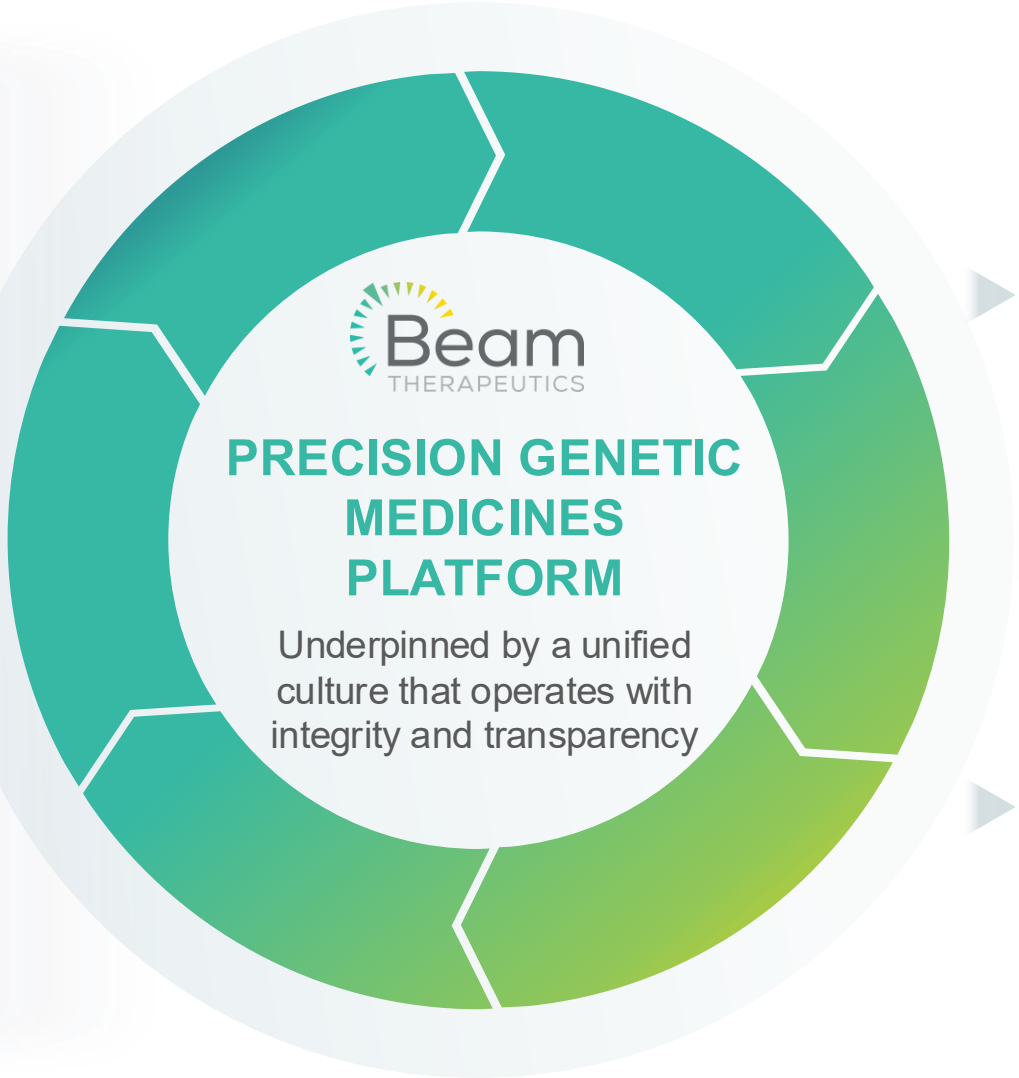
Easily **ADAPTABLE** to new targets

REUSABLE delivery technology

CONSISTENT preclinical and clinical outcomes

HIGHLY SCALABLE internal manufacturing

FLEXIBLE emerging regulatory framework



Multiple growing, high-value franchises

Active pipeline **expansion**

Creative platform **partnerships**

Three high-value programs with blockbuster potential

Differentiated development programs across hematology and liver-targeted genetic diseases addressing large, underserved U.S. patient populations

Sickle Cell Disease

RISTO-CEL

~10K-100K

U.S. patients eligible for *ex vivo* treatment and *in vivo* treatment, respectively

KEY DIFFERENTIATORS

- Best-in-class clinical profile for risto-cel
- Internal manufacturing
- Poised to enter an established market with high patient demand
- Next-wave *in vivo* approach making rapid progress

Alpha-1 Antitrypsin Deficiency (AATD)

BEAM-302

~100K

U.S. patients with severe (PiZZ) AATD

KEY DIFFERENTIATORS

- Potential first-in-class one-time *in vivo* therapy addressing liver and lung manifestations
- Only genetic medicine in pivotal development for AATD
- Converts patients' disease risk to carrier-like status
- Long-term leadership and commitment to AATD patients

Phenylketonuria (PKU)

BEAM-304

~20K

U.S. patients with PKU

KEY DIFFERENTIATORS

- Potential first-in-class one-time *in vivo* treatment
- Multi-mutation approach using novel regulatory pathways
- Limited competition
- Evidence of Beam's growing metabolic expertise

U.S. addressable population estimates per Beam internal data and market research.

Rapidly advancing and growing a portfolio of liver-targeted *in vivo* programs for genetic diseases

Potential **best-in-class** and **first-in-class** AATD program

Strategic **pipeline expansion into PKU**

Industry-leading LNP capabilities

Platform synergies potentially enable use of novel regulatory pathways and established clinical footprint

PROGRAM	DISEASE	DELIVERY	EDITING APPROACH	RESEARCH	LEAD OPTIMIZATION	IND ENABLING	PHASE I/II	PIVOTAL
BEAM-302	Alpha-1 antitrypsin deficiency (AATD)	*In vivo* LNP	Correction of E342K mutation					
BEAM-301	Glycogen storage disease type Ia (GSDIa)	*In vivo* LNP	Correction of R83C mutation					
BEAM-304	Phenylketonuria (PKU)	*In vivo* LNP	Correction of multiple mutations					

LNP = lipid nanoparticle

Updated data from BEAM-302 Phase 1/2 trial support potential as best-in-class and first-in-class one-time treatment for AATD

CORRECTION OF ROOT CAUSE OF DISEASE

BEAM-302 continues to demonstrate the potential to become the first one-time treatment to address both lung and liver manifestations of AATD through direct correction of the disease-causing mutation.

TOTAL AAT ABOVE THERAPEUTIC THRESHOLD

A single dose of 60 mg BEAM-302 demonstrated durable correction of AAT biology, with all patients achieving total AAT levels above the protective threshold.

RESTORATION OF AAT FUNCTION

BEAM-302 restored normal AAT physiology, including production of corrected M-AAT for the first time that is functional and under normal physiologic control in response to inflammation.

REDUCTION OF MUTANT Z-AAT

BEAM-302 led to substantial reductions in disease-driving mutant Z-AAT and circulating Z-polymers, supporting its potential to address key drivers of both liver and lung disease.

CONSISTENT SAFETY PROFILE

Safety profile remains consistent with prior findings and those seen with LNP-based therapies.

PIVOTAL LEADERSHIP

As the most advanced genetic medicine in AATD and the only genetic medicine in pivotal development, BEAM-302 is now dosing patients globally in its pivotal cohort.

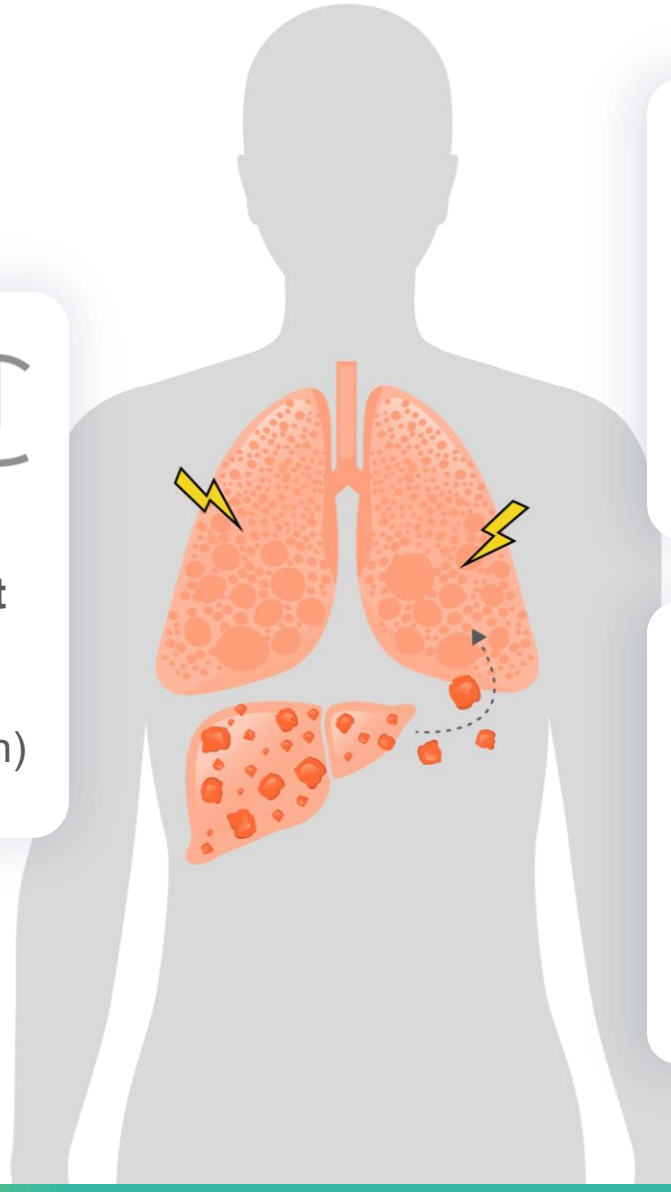
Data cutoff June 24, 2026

Alpha-1 Antitrypsin Deficiency and BEAM-302 Overview

Amy Simon, M.D., Chief Medical Officer

Severe AATD (PiZZ genotype) impacts >100,000 individuals in the U.S. with limited treatment options

Single G to A point mutation in the SERPINA1 gene
(Pi*Z or “Z” mutation)



Progressive lung disease due to:

- Low and poorly functioning systemic Z-AAT levels
- Circulating Z-AAT aggregates, causes inflammation

× Routine **COPD care**

× IV augmentation therapy given **weekly is only approved option**

Progressive liver disease with fibrosis and cirrhosis due to:

- Aggregation and accumulation of mutant Z-AAT

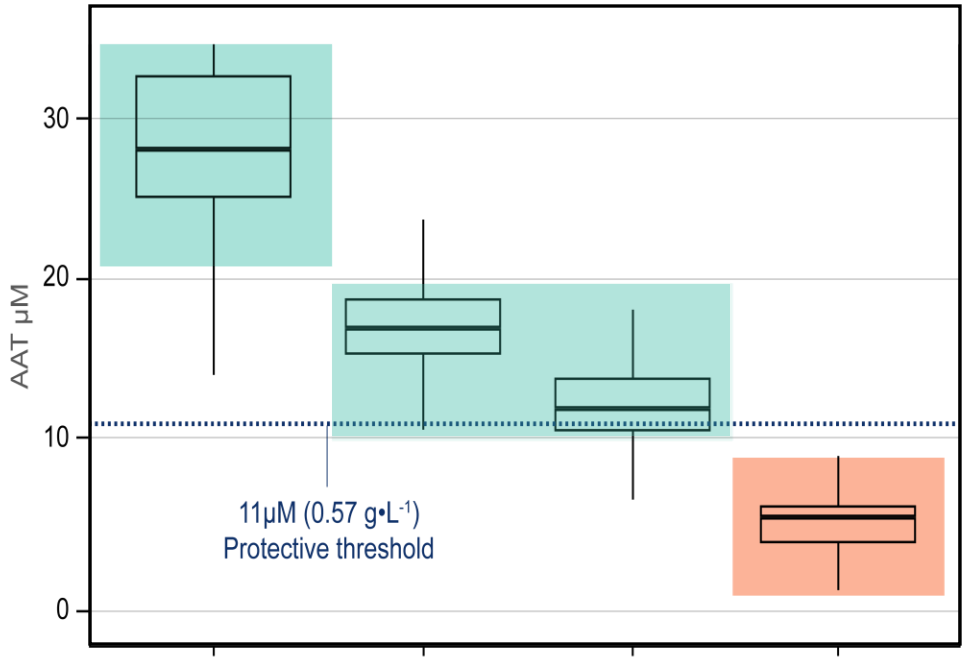
× **Supportive care and liver transplant** for advanced disease

× **No approved treatments** for liver disease

AAT, alpha-1 antitrypsin; AATD, alpha-1 antitrypsin disease. National Center for Biotechnology Information. NM_001127701.1(SERPINA1):c.1096G>A (p.Glu366Lys) AND alpha-1-antitrypsin deficiency. Updated February 2, 2025. Available from: https://www.ncbi.nlm.nih.gov/clinvar/RCV000148877.67/ (Accessed March 27, 2026); Dasí F. Med Clin (Barc) 2024; 162:336–342

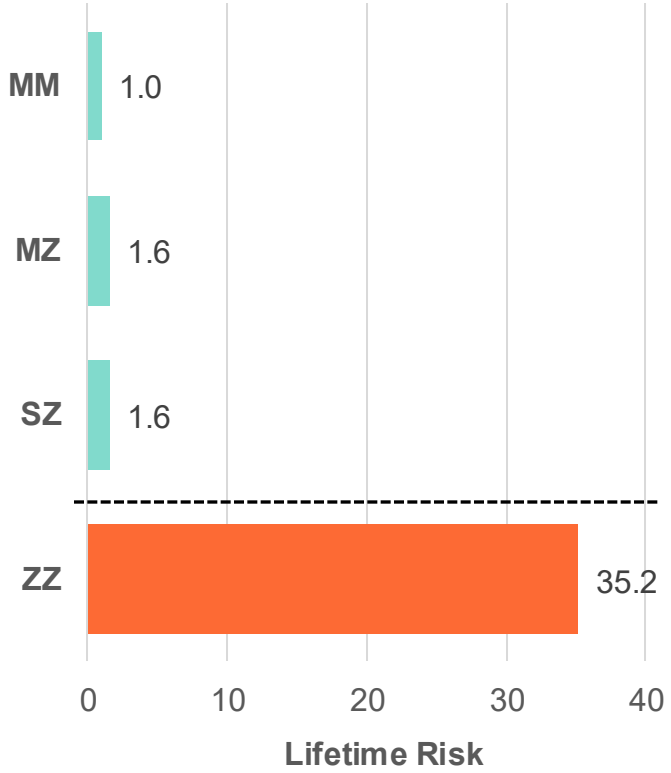
AAT protective threshold >11 µM informed by clinical genetics, translates into marked risk reduction for AATD lung and/or liver disease

AAT Levels Across Genotypes

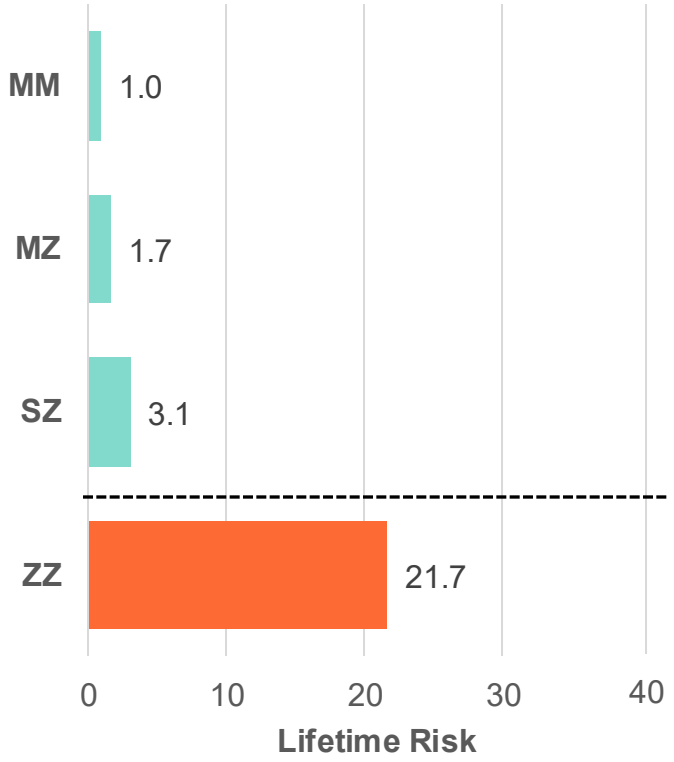


Genotype	MM	MZ	SZ	ZZ
< 11 µM	0%	0%	40%	100%

Emphysema Risk



Liver Fibrosis / Cirrhosis Risk

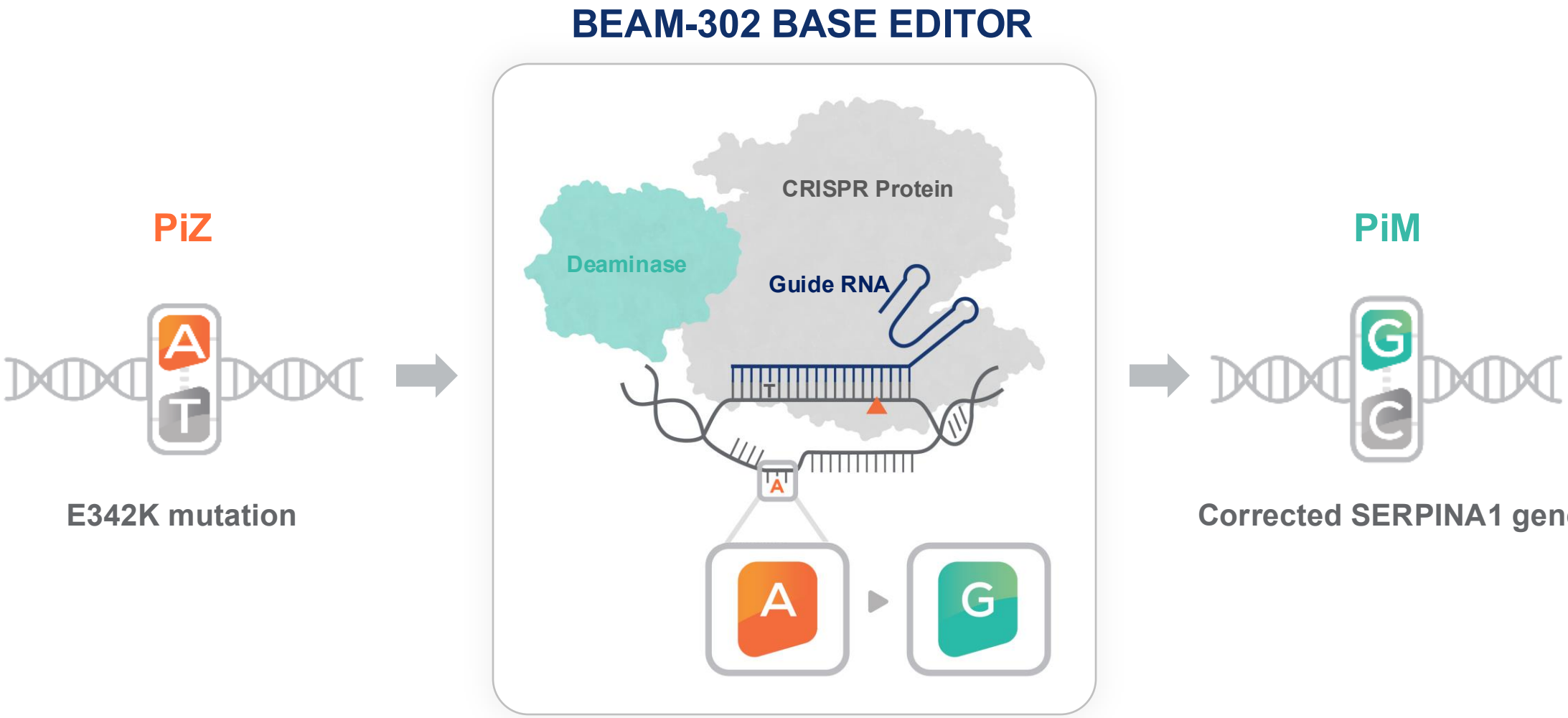


Disease in MZ and SZ individuals requires presence of additional risk factor, e.g., smoking or obesity

Micromolar (µM)

Adapted from Franciosi et al, ERJ 2022 and Vidal et al, Arch Bronconeumol 2006; Brode et al, CMAJ 2012; Fromme et al. 2022

BEAM-302 is a potential one-time therapy that uses base editing to directly correct the E342K mutation causing AATD

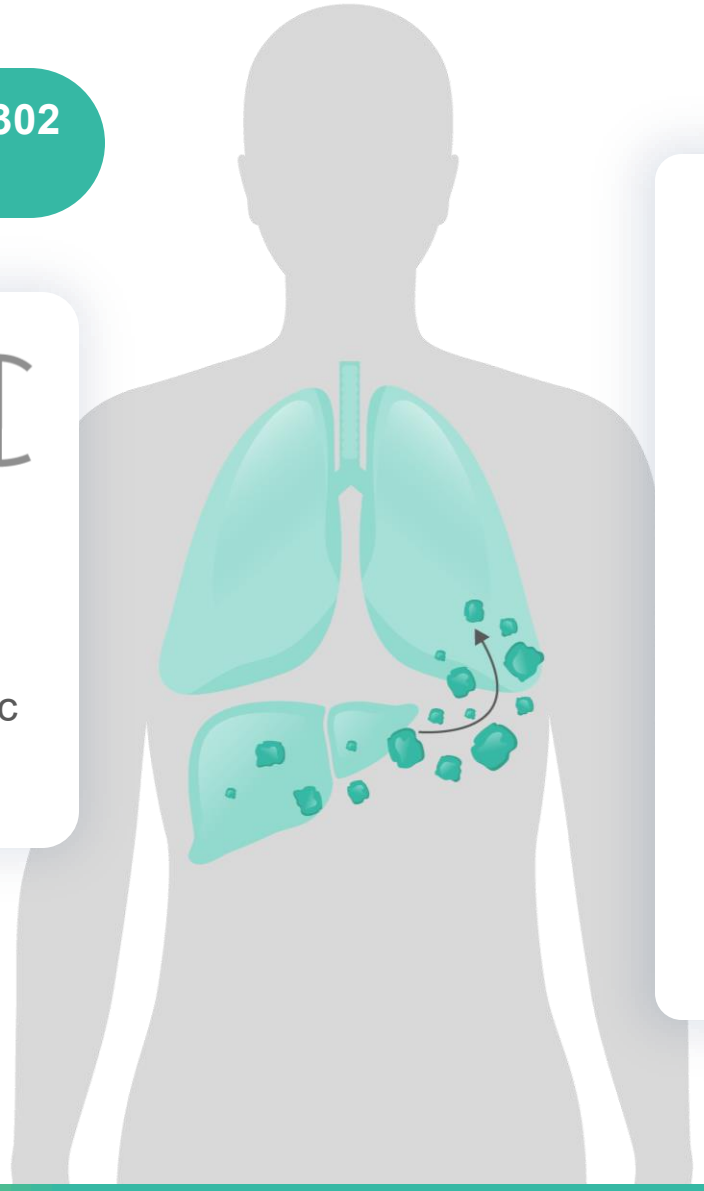


BEAM-302 has the potential to be first, one-time treatment to address full spectrum of disease manifestations of AATD

GOALS OF BEAM-302 TREATMENT

Correction at root cause of disease

Restore physiologic control of AAT



- **Liver produces M-AAT** for the first time
- Significantly **reduces Z-AAT**
- Total AAT **above 11 µM protective threshold**
- Increased total **AAT is functional**
- AAT increases when needed with **acute inflammation**

✔ **Durable, single-course** treatment

✔ **Address both lung and liver** manifestations

Beam intends to pursue accelerated approval pathway for BEAM-302 based on FDA feedback to date

Q1 2025

- ✓ Clinical proof of concept
- ✓ U.S. IND clearance

Q2 2025

- ✓ RMAT Designation
- ✓ Orphan Drug Designation

Q4 2025

FDA alignment

- Alignment reached with FDA on potential accelerated approval pathway for BEAM-302 in AATD
- Primary endpoint based on AAT biomarkers evaluated over 12 months
- Plan to enroll 50 additional patients in expansion of global, open-label Phase 1/2 trial
- Ongoing FDA dialogue on confirmatory trial design
- Accepted to FDA CMC Development and Readiness Pilot (CDRP) program

IND = investigational new drug; RMAT Designation = Regenerative Medicine Advanced Therapy Designation

Robust and flexible Phase 1/2 trial design supported rapid advancement to pivotal development

PART A:
AATD-associated Lung Disease

DOSE ESCALATION & EXPANSION

- Data reported at ERS from 21 patients treated with single-dose BEAM-302 in dose escalation
- 3 patients treated in multi-dose cohort
- 6 patients treated in 60 mg expansion

PART B:
AATD-associated Liver Disease with or without Lung Disease

DOSE ESCALATION

- Data reported at ERS from 8 patients treated with single-dose BEAM-302 in dose escalation

PART C: PIVOTAL COHORT
AATD-associated Lung Disease with or without Liver Disease

- Dosing initiated in global pivotal cohort
- Significant site and patient interest
- Utilizing existing extensive global site network: 14+ sites in 6 countries
- Multiple U.S. sites activated with more planned

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First-in-human in vivo gene editing for severe alpha-1 antitrypsin deficiency (AATD): initial data from the Phase 1/2 study of BEAM-302

John R Hurst, Emily FA van' t Wout, Thomas Ashdown, Matthew Conron, N. Gerry McElvaney, Jeffrey Garrett, Ed Gane, Andrew A Wilson, Joe Kaserman, Charlie Strange, J. Michael Wells, Catherina L Chang, Mark Holmes, Aishath Fazleen, Mark O'Carroll, Ulrike Lorch, Jan Stolk, Amir Majid, Reem Alluhibi, Nicolas Currier, Katy Hayes, Beatrice Chiang, Gary Liu, James Maier, Arthur Lo, Karen Cao, Bahru Habtemariam, Priya S Chockalingam, John J Ko, Sunita Goyal, Amy Simon, Alice Turner

Aims

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We present results from the BEAM-302 clinical study (NCT06389877) a Phase 1/2, multicenter, open-label, dose-exploration and dose-expansion study evaluating safety and efficacy of BEAM-302 in adults with PiZZ-AATD–associated lung disease and/or liver disease

AATD, alpha-1 antitrypsin deficiency

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BEAM-302 clinical study enrolled patients with both AATD-associated lung and liver disease

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PART A:

AATD-associated lung disease

- *15 mg N=3*
- *30 mg N=3*
- *60 mg N=6*
- *75 mg N=9*

PART B:

AATD-associated liver disease with or without lung disease

- *30 mg N=3*
- *60 mg N=5*

NCT06389877 on ClinicalTrials.gov for full study design

- **Select key endpoints:**
  - Rates of **TEAEs**
  - Blood levels of **total AAT**, **Z-AAT** and **M-AAT**, and **functional AAT**
  - **HNE activity** in blood
  - **Z-polymer** levels in blood
- **Baseline characteristics:**
  - **Age range 35–69 years**; homozygous for **PiZZ** mutation; mean post-bronchodilator **ppFEV$_1$: 62.0%** (range 38.8–101.5%)
  - **Mean blood AAT level: 4.8 µM*** (range 2.4–8.4 µM), with clinical diagnosis of **emphysema**; no evidence of liver disease (**Part A only**)
  - Clinical diagnosis of AATD-related liver disease (**Part B only**); METAVIR fibrosis stage F1 (n=4) and F2 (n=4)

Data cutoff June 24, 2026. *Blood AAT was measured by turbidimetry. AAT, alpha-1 antitrypsin; AATD, alpha-1 antitrypsin deficiency; ppFEV$_1$, percent predicted forced expiratory volume in 1 second; HNE, human neutrophil elastase; METAVIR, meta-analysis of histological data in viral hepatitis; TEAE, treatment-emergent adverse event

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Initial safety data suggest that BEAM-302 safety profile is consistent with that of LNP-based therapies

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	Part A (N=21)				Part B (N=8)	
n (%)	**15 mg (N=3)**	**30 mg (N=3)**	**60 mg (N=6)**	**75 mg (N=9)**	**30 mg (N=3)**	**60mg (N=5)**
Any TEAEs	3 (100)	3 (100)	6 (100)	8 (88.9)	3 (100)	5 (100)
Related to BEAM-302	2 (66.7)	0	3 (50.0)	5 (55.6)	1 (33.3)	3 (60.0)
Any TEAEs Grade ≥3	0	0	0	0	0	1 (20.0)*
Related to BEAM-302	0	0	0	0	0	1 (20.0)*
Serious TEAEs	0	0	0	0	0	1 (20.0)[†]
Related to BEAM-302	0	0	0	0	0	0
IRRs	2 (66.7)	0	3 (50.0)	5 (55.6)	1 (33.3)	1 (20.0)
Grade 1	2 (66.7)	0	1 (16.7)	1 (11.1)	0	1 (20.0)
Grade 2[‡]	0	0	2 (33.3)	4 (44.4)	1 (33.3)	0
ALT/AST elevations[§]	3 (100)	1 (33.3)	6 (100)	7 (77.8)	2 (66.7)	3 (60.0)
Grade 1	3 (100)	1 (33.3)	6 (100)	7 (77.8)	2 (66.7)	2 (40.0)
Grade 2	0	0	0	0	0	0
Grade 3	0	0	0	0	0	1 (20.0)*

Data cutoff June 24, 2026. *ALT/AST elevation above baseline began on Day 14 and reached Grade 3 on Day 25; bilirubin remained normal and resolved without intervention; [†]shortness of breath, assessed as unrelated to BEAM-302 and related to underlying disease; [‡]Grade 2 IRRs required treatment intervention and/or infusion interruption; [§]ALT/AST elevations were identified from laboratory data during the dose-limiting toxicity monitoring period and graded per CTCAE v5.0. ALT, alanine aminotransferase; AST, aspartate aminotransferase; IRR, infusion-related reaction; LNP, lipid nanoparticle; TEAE, treatment-emergent adverse event

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Part A single-dose cohorts: BEAM-302 (60 mg) led to sustained increases in total AAT levels to above the 11 µM protective threshold

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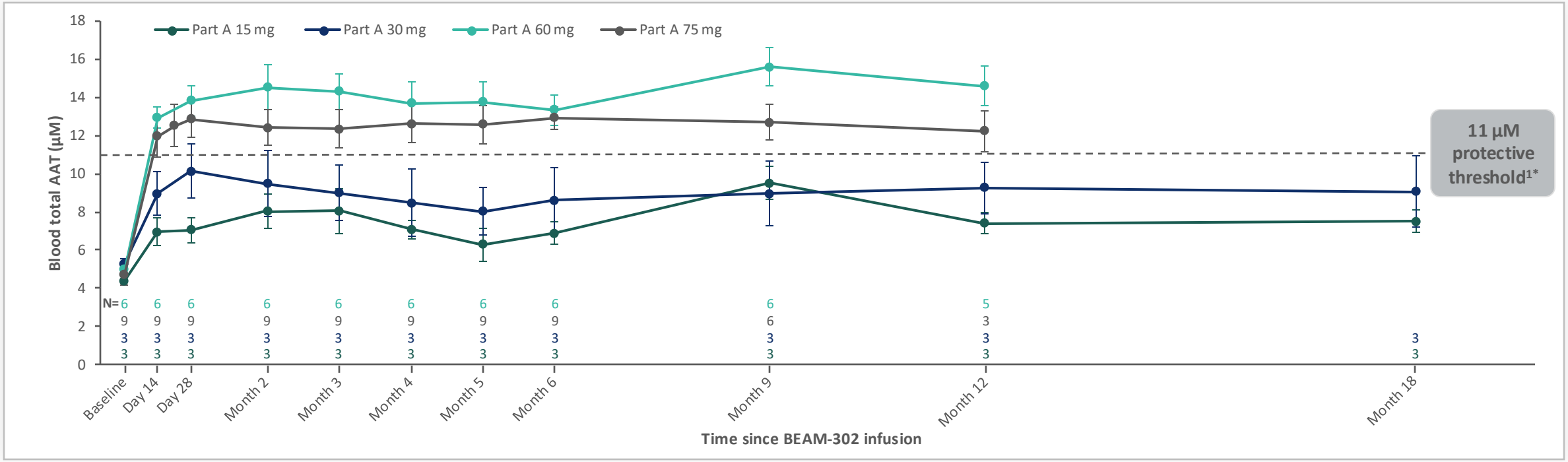

Total AAT (µM)	15 mg (N=3)	30 mg (N=3)	60 mg (N=6)	75 mg (N=9)
Baseline,† mean (SEM)	4.4 (0.2)	5.3 (0.3)	5.0 (0.3)	4.7 (0.6)
Steady state,‡ mean (SEM) Median (min, max)	7.5 (0.2) 7.6 (7.2, 7.8)	9.0 (1.5) 8.5 (6.7, 11.8)	14.4 (0.9) 15.2 (11.0, 16.4)	12.8 (0.8) 12.6 (9.0, 16.3)

Data cutoff June 24, 2026. Total blood AAT was measured by turbidimetry. *Protective serum AAT concentration above which protease-mediated lung damage is reduced, and which serves as a therapeutic target; †baseline for each patient is defined as the average of all assessments conducted within the 84-day screening period prior to BEAM-302 infusion; ‡steady state is defined as the period beginning on a patient's Day 28 visit through their last available visit. AAT, alpha-1 antitrypsin; SEM, standard error of the mean. 1. Franciosi AN, et al. Eur Resp J 2022;59:2101410

Beam THERAPEUTICS

Part B cohorts: Post-treatment total AAT levels consistent with Part A

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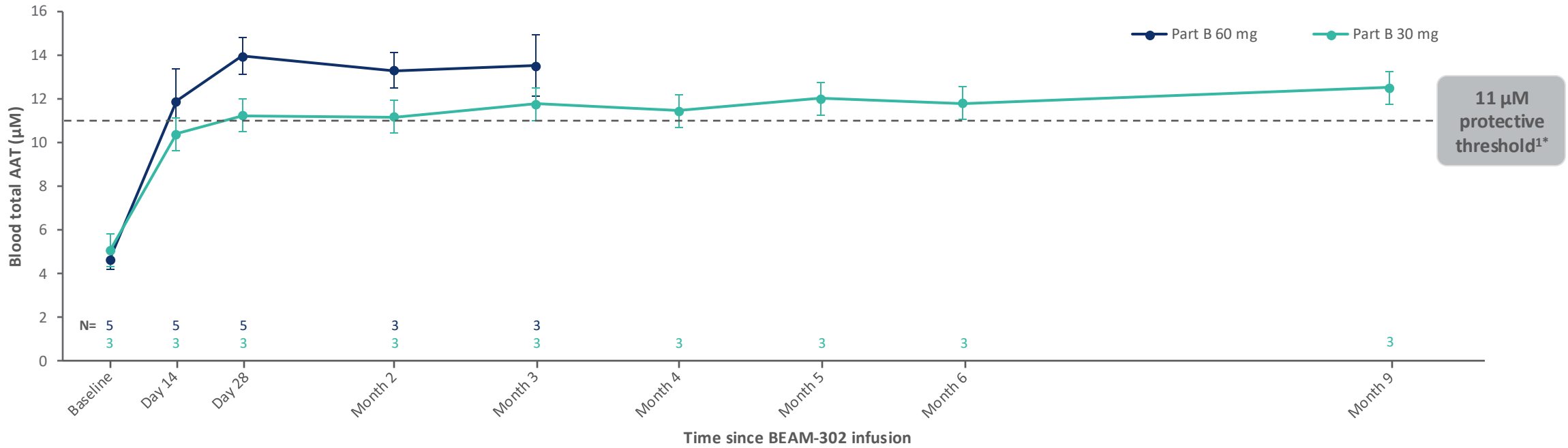

Total AAT (µM)	30 mg (N=3)	60 mg (N=5)
Baseline,† mean (SEM)	5.1 (0.5)	4.7 (0.5)
Steady state,‡ mean (SEM) Median (min, max)	11.7 (1.0) 11.4 (10.2, 13.5)	13.5 (0.9) 13.8 (11.1, 15.4)

Data cutoff June 24, 2026. Total blood AAT was measured by turbidimetry. *Protective serum AAT concentration above which protease-mediated lung damage is reduced, and which serves as a therapeutic target; †baseline for each patient is defined as the average of all assessments conducted within the 84-day screening period prior to BEAM-302 infusion; ‡steady state is defined as the period beginning on a patient's Day 28 visit through their last available visit. AAT, alpha-1 antitrypsin; SEM, standard error of the mean. 1. Franciosi AN, et al. Eur Resp J 2022;59:2101410

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Functional AAT increased and human neutrophil elastase activity levels decreased with BEAM-302 at 60 mg dose

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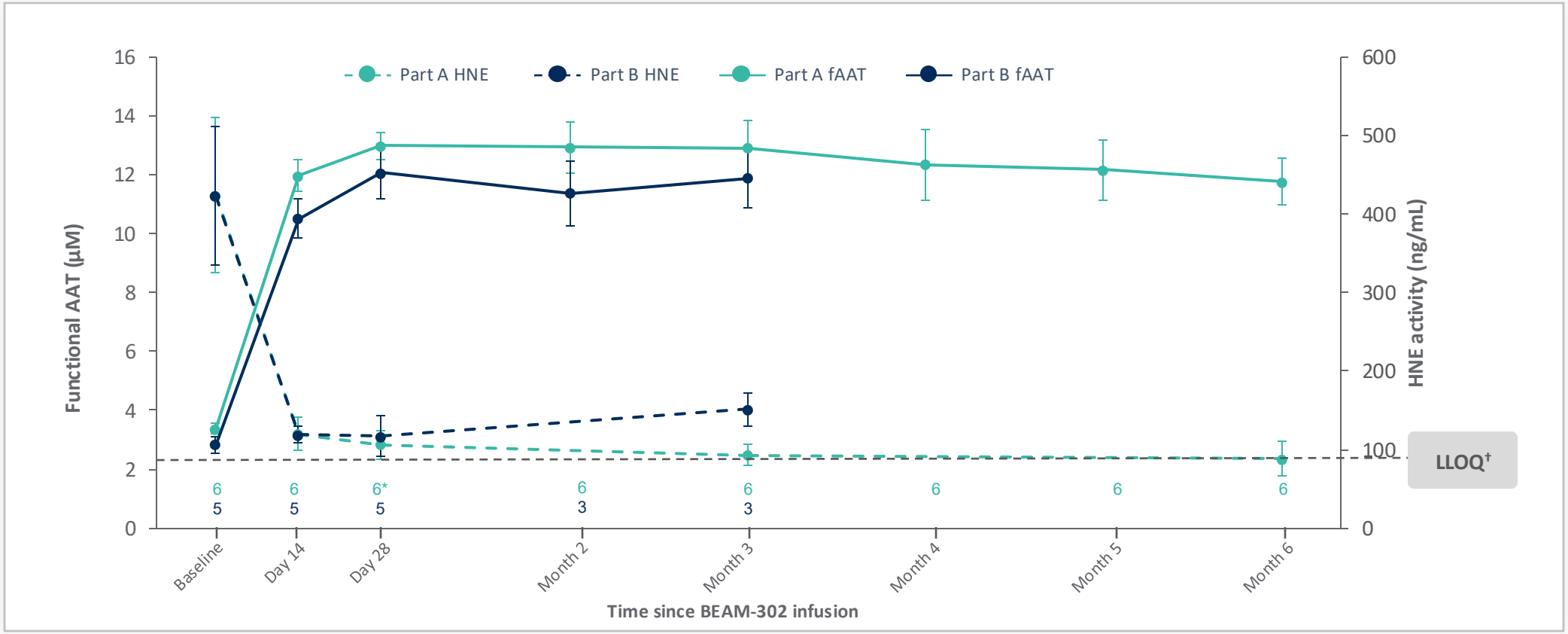

- Following a 60 mg dose, functional AAT levels increased and >80% of participants in **Part A** had at least one human neutrophil elastase (HNE) activity measurement below lower limit of quantification of 87.8 ng/mL; in **Part B**, functional AAT also increased and HNE activity decreased

Data cutoff June 24, 2026; functional AAT irreversibly binds and inhibits HNE; therefore, reduced NE levels indicate functional AAT activity. *n=5 for Part A HNE concentration at Day 28; †values below the LLOQ of 87.78 ng/mL were set to LLOQ/2 for analysis. AAT, alpha-1 antitrypsin; HNE, human neutrophil elastase; LLOQ, lower limit of quantification

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Single dose BEAM-302 (60 mg) led to 84% reduction in circulating Z-AAT in Part A and Part B

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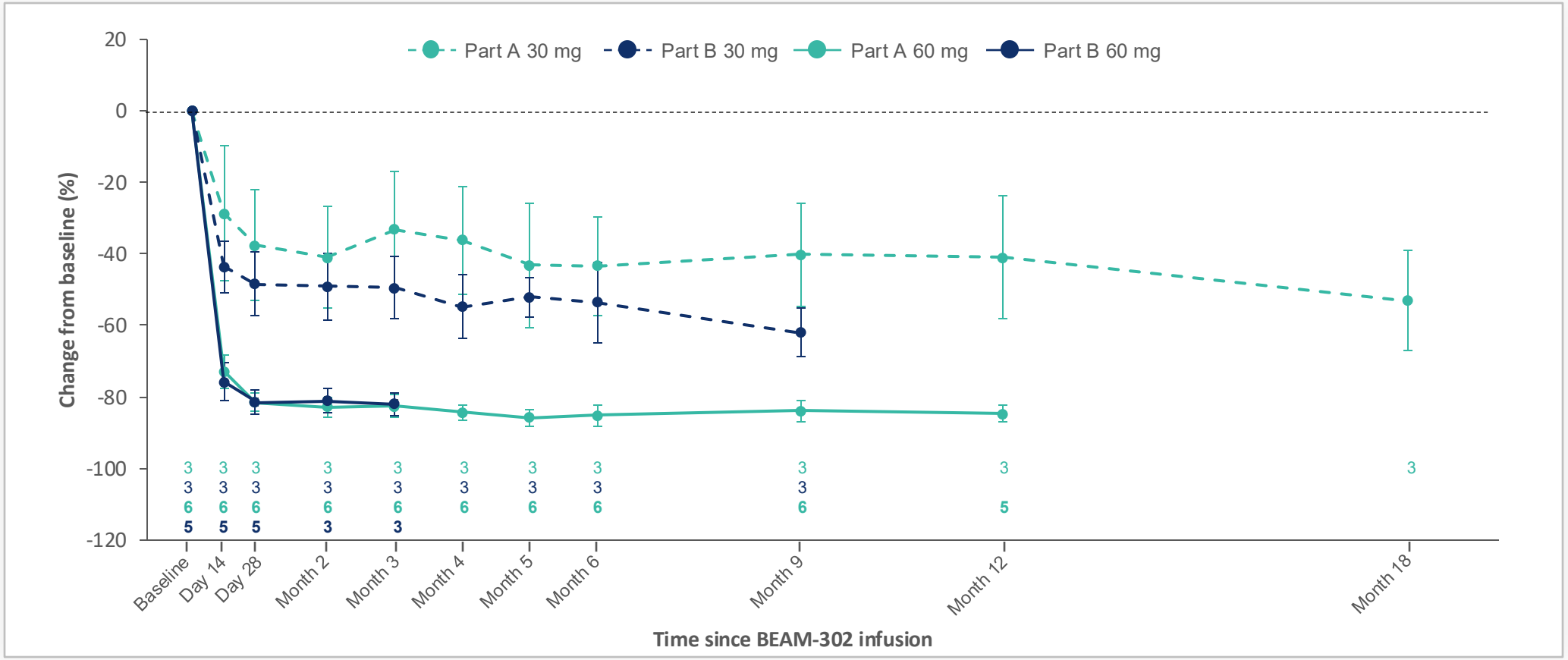

Blood Z-AAT (%)	Part A: 60 mg (N=6)	Part B: 60 mg (N=5)
Percentage change from baseline,* mean (SEM)	−83.9 (2.6)	−83.5 (2.7)

Data cutoff June 24, 2026; Z-AAT was measured by liquid chromatography-mass spectrometry; bold typeface indicates N numbers for 60 mg doses; *Measured at steady state, defined as the period beginning on a patient's Day 28 visit through their last available visit. AAT, alpha-1 antitrypsin

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BEAM-302 treatment led to a significant decrease in circulating Z-AAT protein aggregates, referred to as Z-polymers

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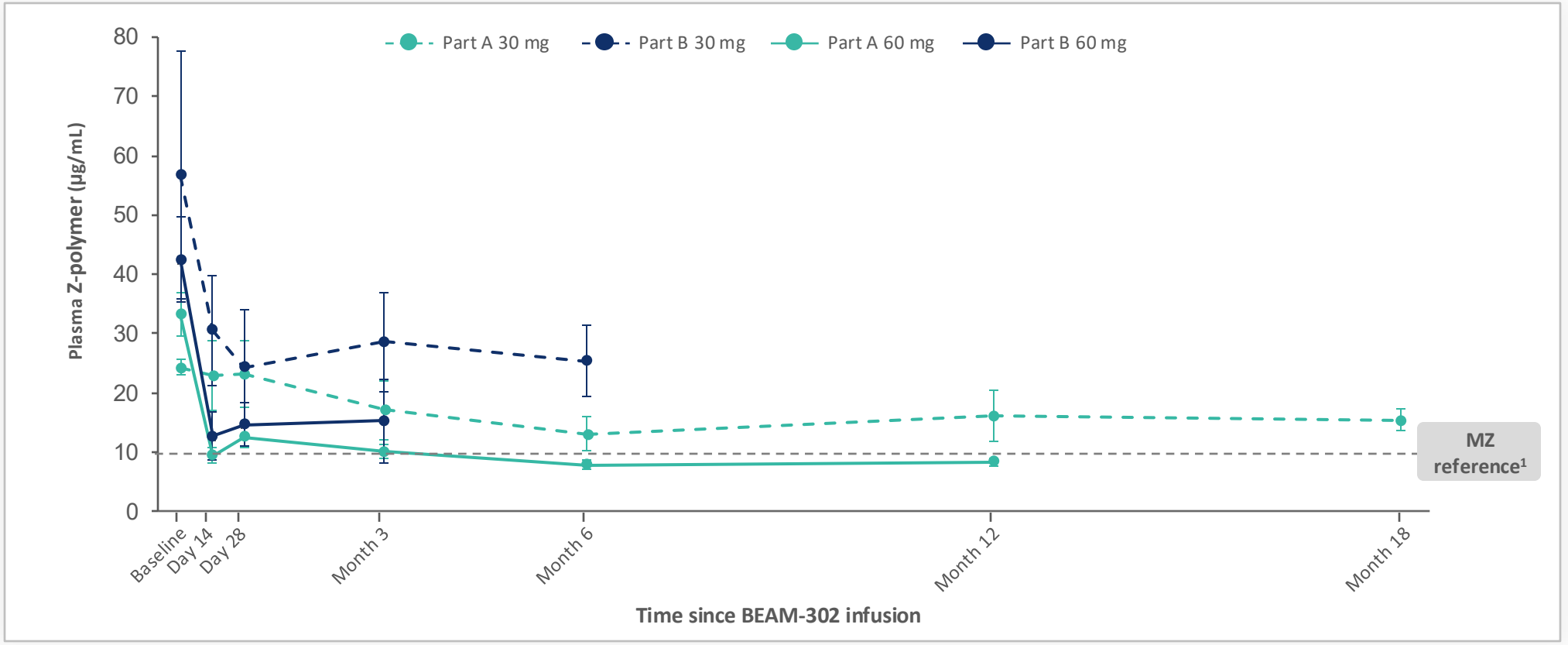

Data cutoff June 24, 2026; plasma Z-polymer was measured by enzyme-linked immunosorbent assay; patient numbers by visit: Part A 30 mg 1: 3, 3, 3, 3, 3, 3, 3 (Baseline–Month 18); Part A 60 mg: 6, 6, 5, 6, 6, 5 (Baseline–Month 12); Part B 30 mg: 3, 3, 3, 3, 3 (Baseline–Month 6); Part B 60 mg 2: 5, 5, 5, 3 (Baseline–Month 3)

1. Núñez et al. Respir Res; 2021;22:244

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BEAM-302 (60 mg) treatment achieved sustained circulating M-AAT levels of 93% in Part A and Part B

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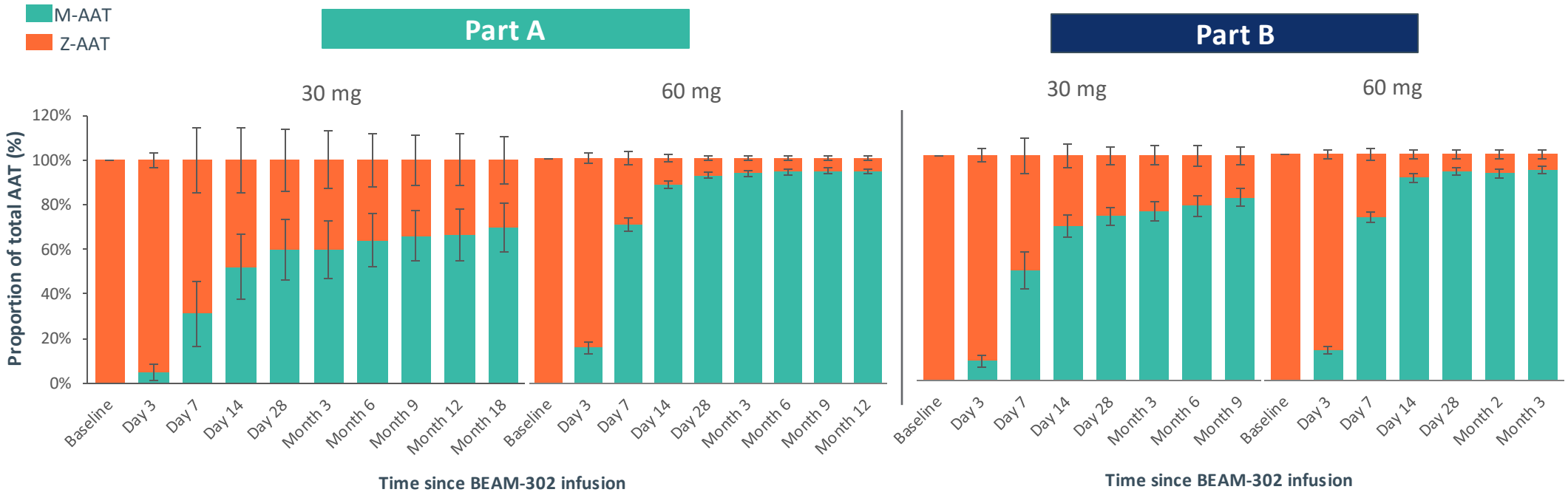
BEAM-302 restored predominant production of the M-AAT isoform



- Proportion of **M-AAT of >90% post-BEAM-302** at 60 mg dose is **greater than MZ genotype (80%)**[1]

M-AAT, as a proportion of total AAT	Part A: 60 mg (N=6)	Part B: 60 mg (N=5)
Baseline,* mean (SEM)	0 (0)	0 (0)
Steady state,† mean (SEM)	93.6 (1.1)	93.3 (1.5)

Data cutoff June 24, 2026. M-AAT/Z-AAT was measured by liquid chromatography mass spectrometry. %M-AAT=M-AAT/(M-AAT+Z-AAT)×100. Total AAT was measured by turbidimetry. *Steady state is defined as the period beginning on a patient's Day 28 visit through their last available visit. AAT, alpha-1 antitrypsin; SEM, standard error of the mean.

1. Donato LJ, et al. Respir Res 2015;16:96

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BEAM-302 restored normal physiologic control of AAT protein production

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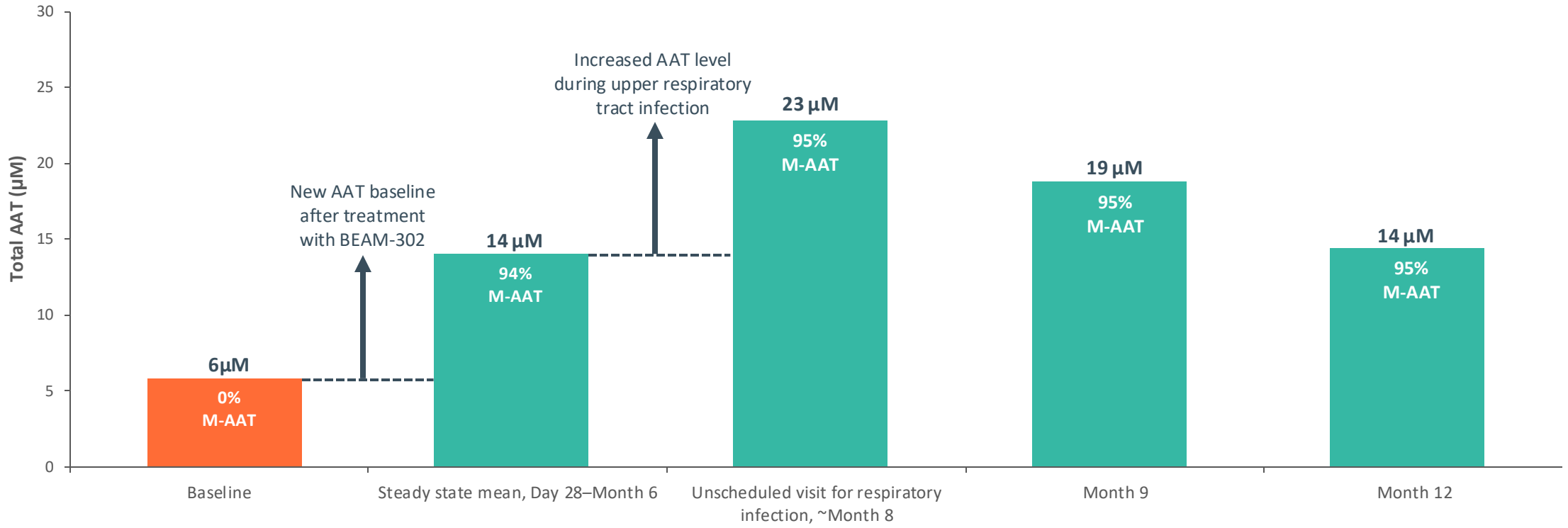
BEAM-302 resulted in normal AAT regulation during inflammation, while maintaining a beneficial M/Z ratio*



Data cutoff June 24, 2026; total AAT was measured by turbidimetry
*Data from a single patient treated with 60 mg BEAM-302
AAT, alpha-1 antitrypsin

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BEAM-302 offers the potential for a one-time therapy to correct the mutant Z allele in the *SERPINA1* gene and restore AAT function

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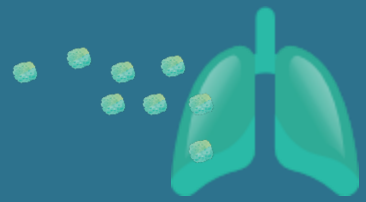
Correct DNA mutation – the root cause of disease

BEAM-302 is the **first therapy** in pivotal development to correct a disease-causing mutation

Safety profile is consistent with LNP-based therapies

Well tolerated at 60 mg, with mainly transient Grade 1 transaminase elevations and mild-to-moderate IRRs



Durable restoration of AAT function

60 mg dose led to: **increased total and functional AAT above the protective threshold,** the **majority of which was M-AAT**, **decreased Z-AAT, and increased circulating AAT** during inflammation

Please see accompanying ePoster (5575) for additional information

The Phase 2 **pivotal cohort** of patients with **AATD-associated lung disease with or without liver disease** (Part C of this study) is currently **dosing patients globally** using the **60 mg dose of BEAM-302**

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AAT, alpha-1 antitrypsin; AATD, alpha-1 antitrypsin deficiency; IRR, infusion-related reaction; LNP, lipid nanoparticle

What does the ideal AATD treatment look like?

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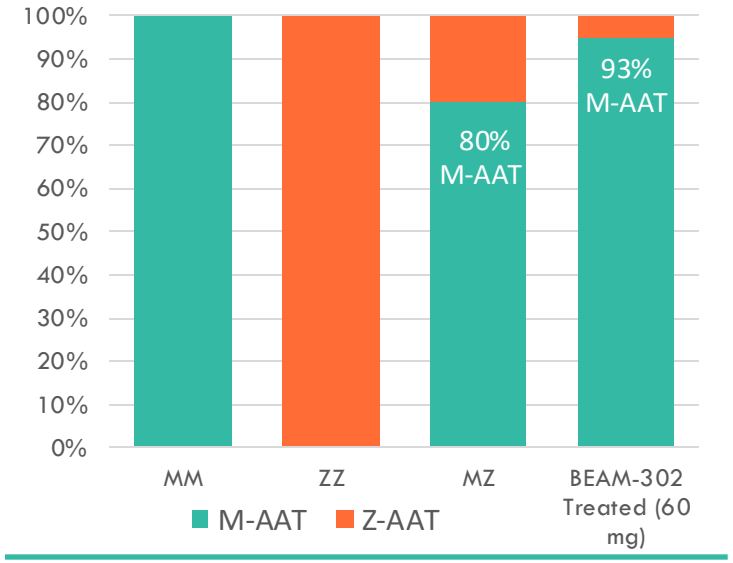
Total AAT above protective threshold of 11 µM

	No Disease	No Disease Unless Other Risk Factor Present		Disease
Genotype	MM	MZ	SZ	ZZ
AAT < 11 µM	0%	0%	~40%	100%
Emphysema risk	None	Low	Low	High
Liver disease risk	None	Low	Low	High

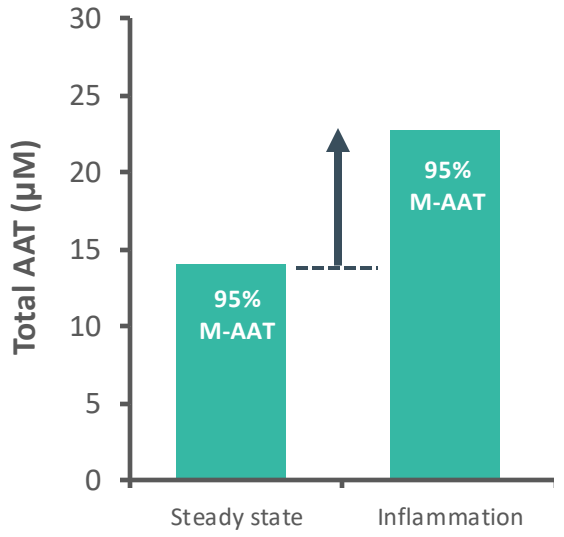
Total AAT above protective threshold eliminates risk unless second risk factor

AAT composition with higher M-AAT and lower Z-AAT



Lower Z-AAT and Z-polymer associated with decrease in liver and lung manifestations

M-AAT increases with acute phase reaction



Increase in total AAT maintains composition with high M-AAT to protect lungs

Adapted from Franciosi et al, ERJ 2022 and from Vidal et al, Arch Bronconeumol 2006; Brode et al, CMAJ 2012; Donato LJ, et al. Respir Res 2015;16:96; Mahadeva, Ravi et al. The American journal of pathology vol. 166,2 (2005): 377-86; Mulgrew, Alan T et al. Chest vol. 125,5 (2004): 1952-7.

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Acknowledgments

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Thank you to the study participants, their families, and their caregivers for their participation, and the study investigators, study sites, Alpha-1 Foundation, and patient advocacy groups for their contributions

This clinical study is sponsored by Beam Therapeutics

Please see accompanying ePoster (5575) for additional information

US	UK
Boston University and Boston Medical Center, Boston, Massachusetts Medical University of South Carolina (MUSC), Charleston, South Carolina University of Alabama at Birmingham (UAB), Alabama University of Florida, Gainesville, Florida	University Hospital Southampton NHS Foundation Trust, Southampton Richmond Pharmacology Ltd, London Royal Free London NHS Foundation Trust, London Queen Elizabeth Hospital, University Hospitals Birmingham NHS Foundation Trust
AUS	**Ireland**
Royal Adelaide Hospital, Adelaide St Vincents Hospital Melbourne, Fitzroy, Melbourne	Beaumont Hospital, Dublin, Ireland
NZ	**The Netherlands**
Waikato Hospital, Hamilton New Zealand Clinical Research (NZCR), Auckland Aotearoa Clinical Trials (Middlemore Clinical Trials), Auckland	Leiden University Medical Center, Leiden

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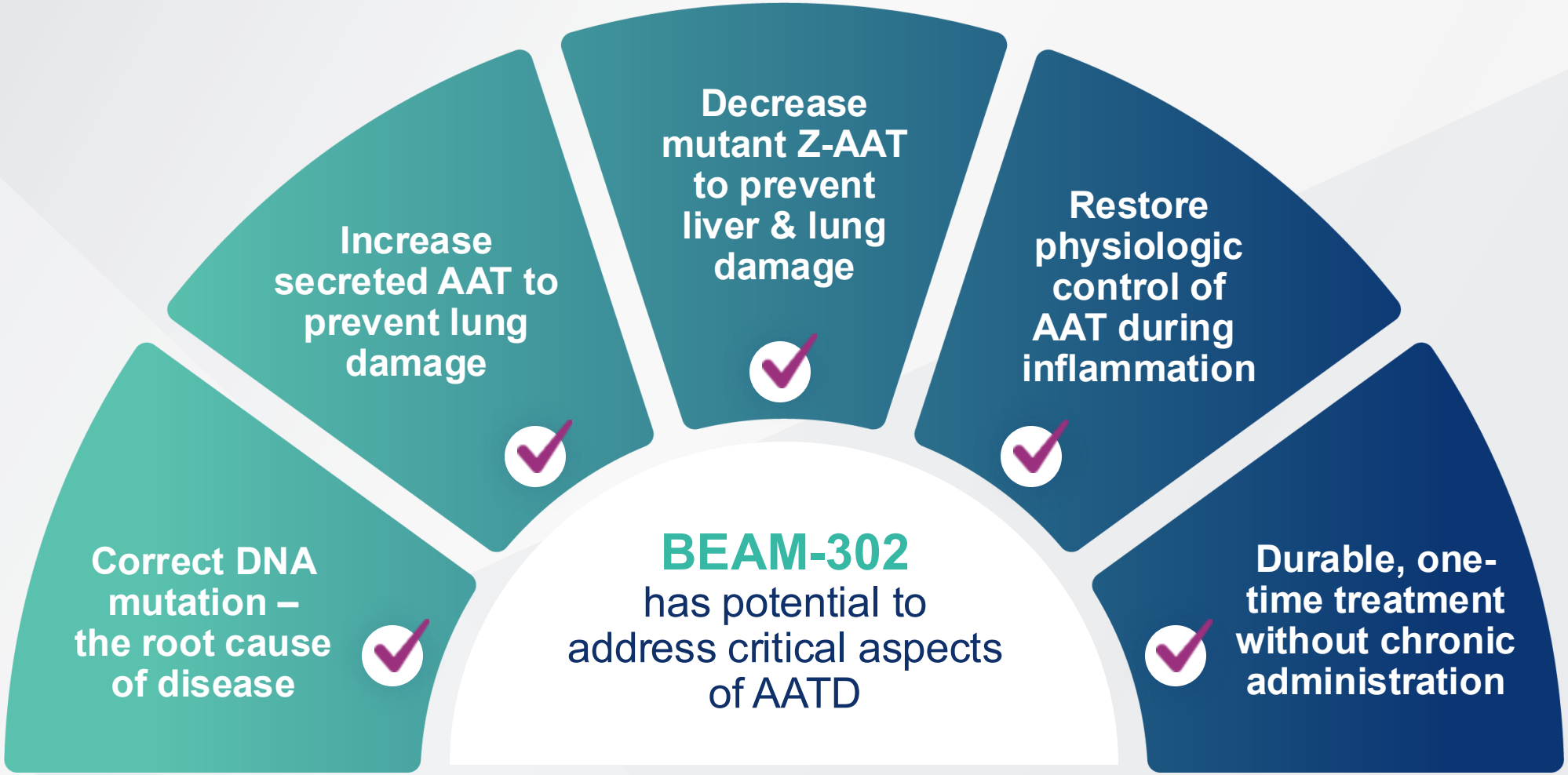
Beam THERAPEUTICS

Closing Remarks

Giuseppe Ciaramella, Ph.D., President

BEAM-302, the first genetic medicine in pivotal development, has the potential to be the first one-time treatment for both lung and liver manifestations of AATD



Beam's long-term commitment to leading innovation for the AATD community

- Internal Beam lifecycle management through ongoing R&D efforts
- Member of C-Path's Critical Path for AATD (CPA-1) consortium in collaboration with FDA to accelerate AATD research by identifying clinical efficacy endpoints
- Collaboration with Alpha-1 Foundation and Alpha-1 Europe Alliance to educate about gene editing and obtain critical input on clinical trial design and patient experience
- Inaugural industry sponsor of AlphaDetect, a nonprofit powered and funded by the Alpha-1 Foundation, to further strengthen efforts to accelerate route targeted detection of AATD

ALPHA-1 FOUNDATION

ALPHADETECT
Accelerating answers.
Informing decisions.

Alpha-1 Europe Alliance

Beam is well positioned to realize the power of predictability in 2026 through key anticipated milestones

Pursue Path to Approval for Lead Programs

- BEAM-302 pivotal cohort ongoing
- Plan to submit risto-cel BLA as early as YE 2026

Advance and Expand Pipeline

- Study start-up activities ongoing for BEAM-304 Phase 1/2 clinical trial
- Report initial BEAM-301 data by YE 2026
- Advance *in vivo* HSC editing program

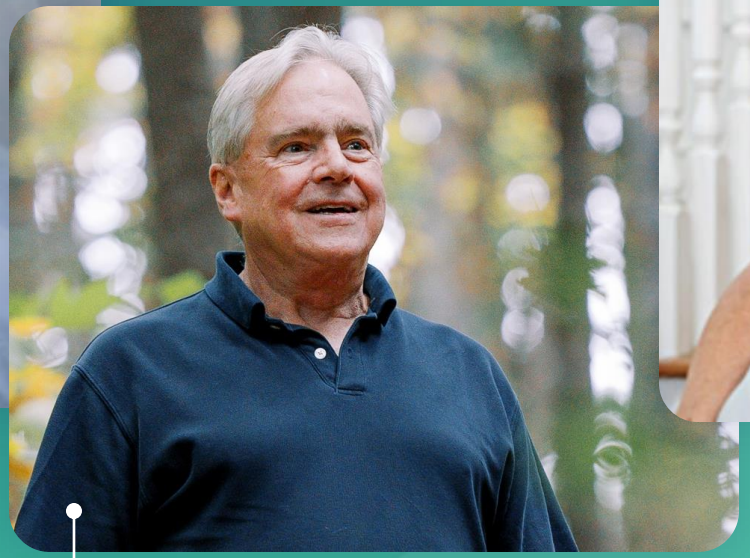
Maintain Financial Strength

- $1.2 billion in cash as of June 30, 2026*
- Expected runway into mid-2029 through anticipated risto-cel launch, execution of BEAM-302 pivotal development plan and clinical proof of concept for BEAM-304**

*Inclusive of cash, cash equivalents, and marketable securities.
**Inclusive of potential additional $200 million from Sixth Street facility.

THANK YOU

Kyle
LIVING WITH
SICKLE CELL DISEASE



Dan
LIVING WITH
ALPHA-1 ANTITRYPSIN DEFICIENCY

Alyssa and Gayle
LIVING WITH
GLYCOGEN STORAGE DISEASE TYPE IA