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, 2021

BEAM THERAPEUTICS INC.
(Exact name of registrant as specified in its charter)

Delaware	001-39208	81-5238376
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)
26 Landsdowne St. Cambridge, MA (Address of principal executive offices)		02139 (Zip Code)

(Registrant's telephone number, including area code): (857) 327-8775

Not Applicable
(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 (see General Instruction A.2. below):

☐ 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, par value $0.01 per share	BEAM	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 7.01 Regulation FD Disclosure.

Beam Therapeutics Inc. (the "Company") will be conducting meetings with participants attending the 39th Annual J.P. Morgan Healthcare Conference (the "Conference"). The slides to be presented by the Company at the Conference are furnished with this report as Exhibit 99.1, which is incorporated herein by reference.

The information in this Form 8-K (including Exhibit 99.1 attached hereto) is being furnished and shall not be deemed "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, nor shall it be deemed incorporated by reference into any filing by the Company, under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such filling.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Beam Therapeutics Inc. Presentation at the 39th Annual J.P. Morgan Healthcare Conference

SIGNATURES

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.

BEAM THERAPEUTICS INC.

Date: January 12, 2021

By: /s/ John Evans

John Evans
Chief Executive Officer

Exhibit 99.1

Beam Therapeutics

PRECISION GENETIC MEDICINES THROUGH BASE EDITING

NASDAQ: BEAM

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 reflect, among other things, our current expectations and anticipated results of operations, all of which are subject to known and unknown 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," "believe," "intend," "project," "forecast," "estimates," "targets," "projections," "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 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; the potential impact of the COVID-19 pandemic; that preclinical testing of our product candidates and preliminary or interim data from preclinical and clinical trials may not be predictive of the results or success of ongoing or later clinical trials; that enrollment of our clinical trials may take longer than expected; 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 heading "Risk Factors" and elsewhere in our annual report on Form 10-K for the year ended December 31, 2019, our quarterly reports on Form 10-Q for the quarters ended March 31, 2020, June 30, 2020 and September 30, 2020, 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 (except as otherwise noted) 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.

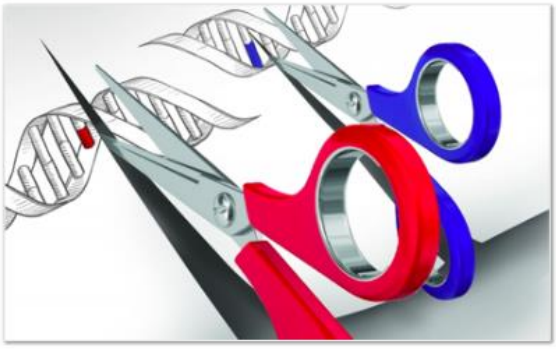
Our vision is to provide life-long cures for patients suffering from serious diseases

- Coming era of **one-time, curative therapies**
- Gene editing for **rare and common diseases**
- Platform for **rapidly-programmable precision medicines**

Base editing is a new approach to gene editing

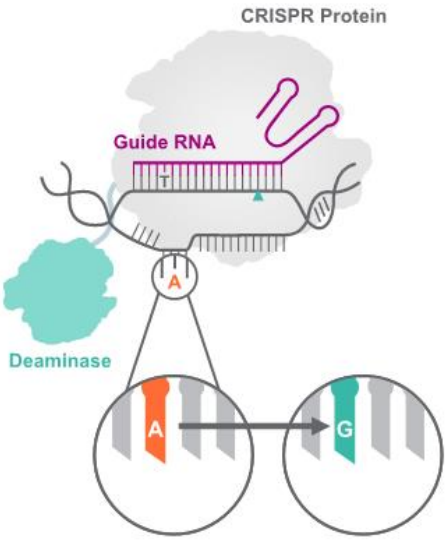
Nuclease editing

CRISPR, Zinc Fingers, TALEs



Base editing

Base editing is a highly-differentiated, potentially best-in-class gene editing technology



B Biology
- Direct editing of single base pairs in disease-causing genes

A Application
- Gene correction, activation, silencing, multiplexing

S Specificity
- Low to undetectable off-target profile
- "Gentler" editing without double-stranded DNA breaks

E Efficiency
- High levels of precise editing in any cell type, including non-dividing cells

Diversified portfolio of wholly-owned base editing programs

DELIVERY	THERAPEUTIC AREA	PROGRAM / DISEASE		APPROACH	RESEARCH	LEAD OPTIMIZATION	IND ENABLING	PHASE I/II	PIVOTAL
ELECTRO-PORATION 	Hematology 	BEAM-101	Sickle Cell Disease Beta Thalassemia	Fetal hemoglobin activation					
		BEAM-102	Sickle Cell Disease	Direct correction of sickle-causing mutation					
	Oncology 	BEAM-201	T-cell Acute Lymphoblastic Leukemia	Multiplex silenced CD7 CAR-T					
		Acute Myeloid Leukemia		Multiplex silenced CAR-T					
NON-VIRAL (LNP) 	Liver Diseases 	Alpha-1 Antitrypsin Deficiency		Precise correction of E342K					
		Glycogen Storage Disorder 1a		Precise correction of Q347X					
				Precise correction of R83C					
		Undisclosed		Multiplex editing					
VIRAL (AAV) 	Ocular and CNS 	Stargardt Disease		Precise correction of G1961E					
		Undisclosed		Precise correction					
		Undisclosed		Gene silencing					

LNP = Lipid Nanoparticle; AAV = Adeno Associated Virus; CNS = Central Nervous System

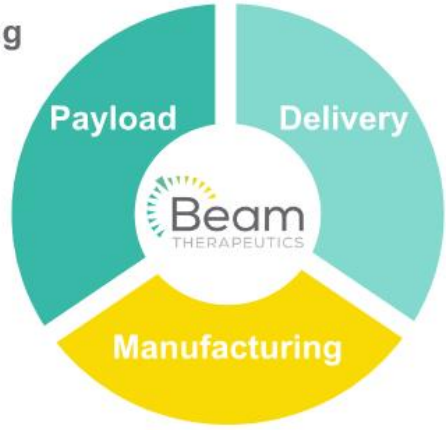
Key progress and milestones

	2020 achievements	Next milestones in 2021
ELECTRO-PORATION 	☑ *In vivo* proof of concept and development candidate nomination for **BEAM-101**	☐ File IND for **BEAM-101** during the second half
	☑ Initiate IND-enabling studies for **BEAM-101**	☐ Initiate IND-enabling studies for **BEAM-102**
	☑ >80% direct correction of sickle mutation and development candidate nomination for **BEAM-102**	☐ Initiate IND-enabling studies for **BEAM-201**
	☑ *In vivo* proof of concept and development candidate nomination for **BEAM-201**	
NON-VIRAL (LNP) 	☑ *In vivo* proof of concept for direct correction of alpha-1 antitrypsin deficiency	☐ Evaluate Beam LNP formulation in non-human primates and release initial data in early 2021
	☑ *In vivo* editing of both R83C and Q347X mutations in GSDIa mouse models	☐ Nominate first liver development candidate
VIRAL (AAV) 	☑ Research collaboration with Institute Ophthalmology Basel for preclinical development of Stargardt program	☐ Initiate non-human primate studies for Stargardt

We are establishing the leading platform for precision genetic medicine

Full suite of gene editing technologies

- Base editing
- Nuclease editing
- RNA editing
- Prime editing



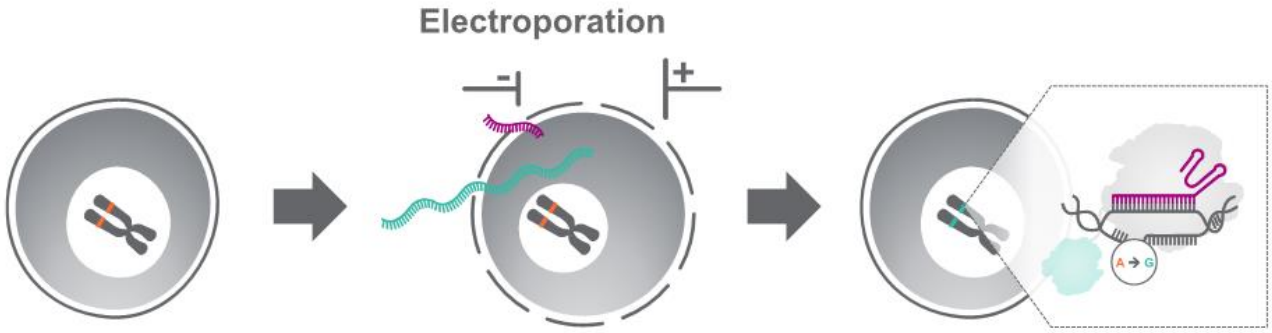
Full suite of delivery technologies

- Auto / allo cell products
- mRNA
- LNP vectors
- Viral vectors

Wholly-owned manufacturing capability

- 100,000 square foot GMP clinical/commercial facility in NC, phased build, online by 2023

Ex vivo delivery of base editors using clinically validated electroporation technology

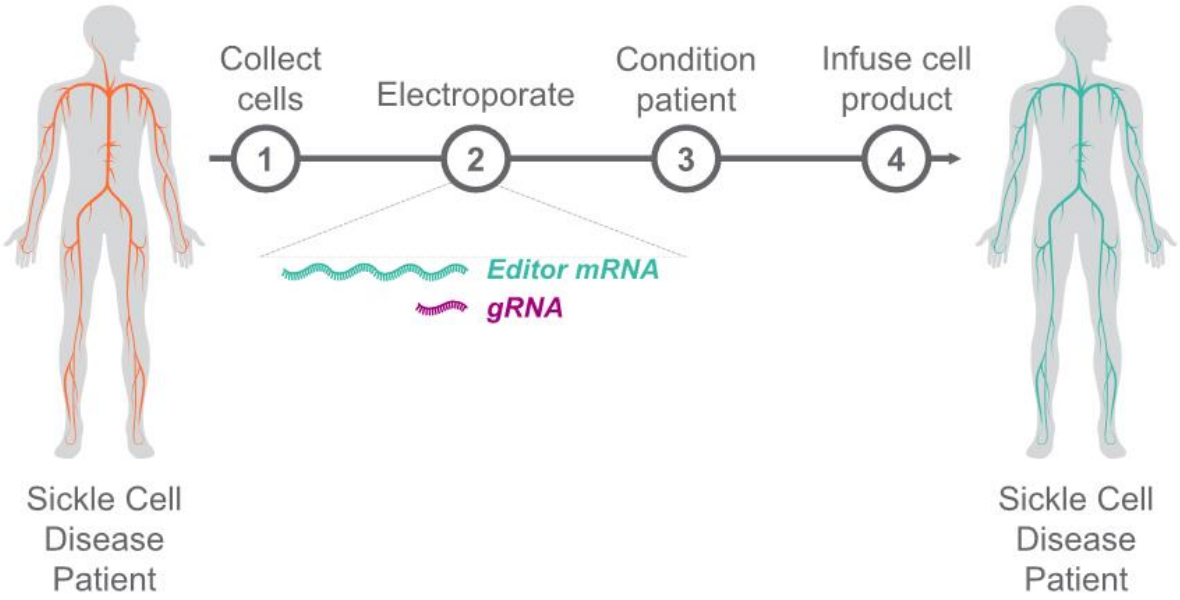


Target cell

Electroporation of ***program-specific gRNA*** and ***base editor mRNA***

Base editor performs gRNA -dependent editing

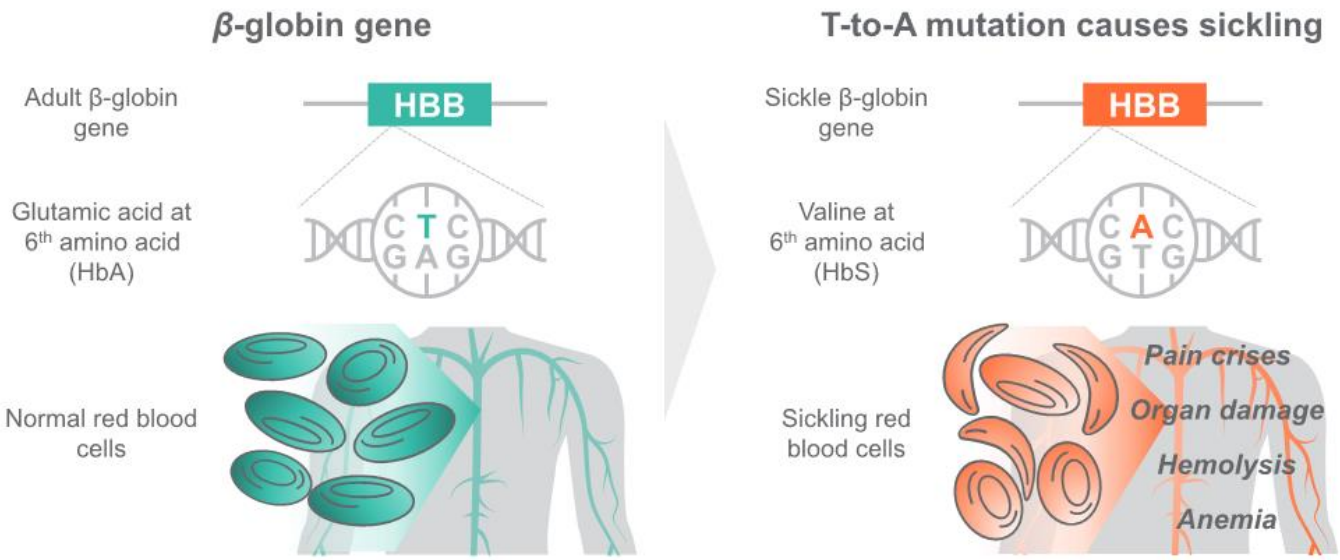
Autologous *ex vivo* cell process for editing hematopoietic stem cells



Sickle Cell Disease Patient

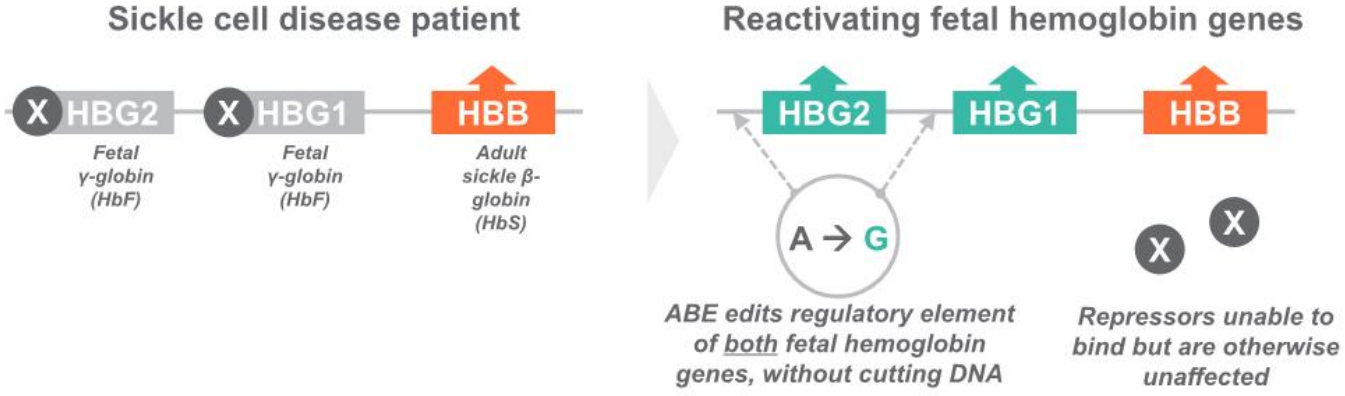
Sickle Cell Disease Patient

Sickle cell disease (SCD)



Approximately 100,000 sickle cell disease patients in the US

BEAM-101: Recreating hereditary persistence of fetal hemoglobin (HPFH) with base editing

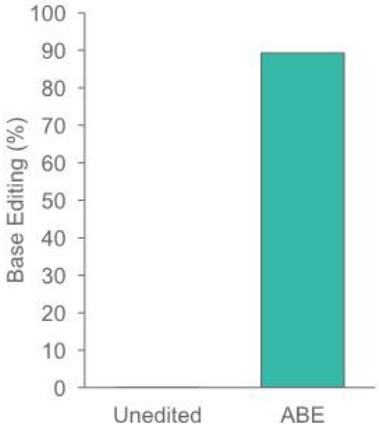


- Naturally-occurring base changes cause Hereditary Persistence of Fetal Hemoglobin (HPFH), which protects patients from SCD/B-Thal
- Base editors can reproduce these changes, leading to elevated levels of fetal hemoglobin
- Higher fetal hemoglobin likely to correlate with further reductions in disease symptoms

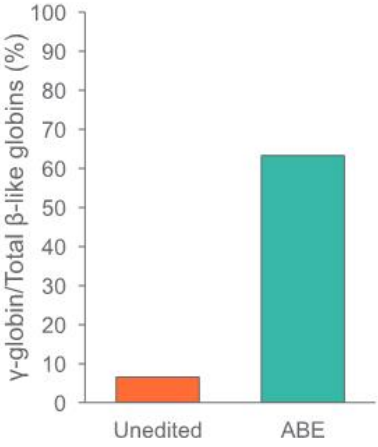
Mussallam, et al. 2012. *Blood.*

BEAM-101: Robust base editing at HBG1/2 gene promoters in sickle cell disease patient cells

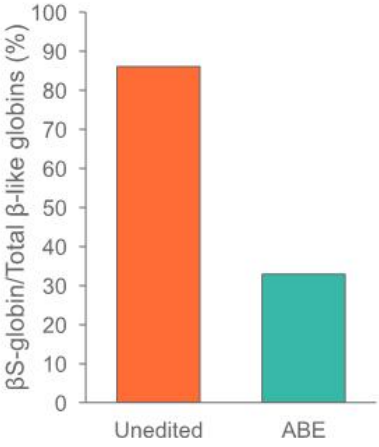
>80% base editing at HBG1/2 promoters



>60% levels of HbF gamma globin protein



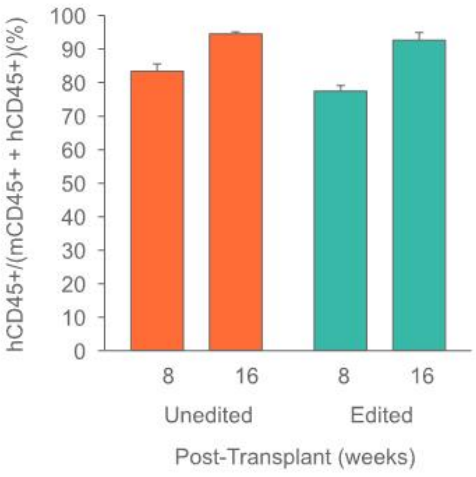
<40% levels of HbS sickle protein



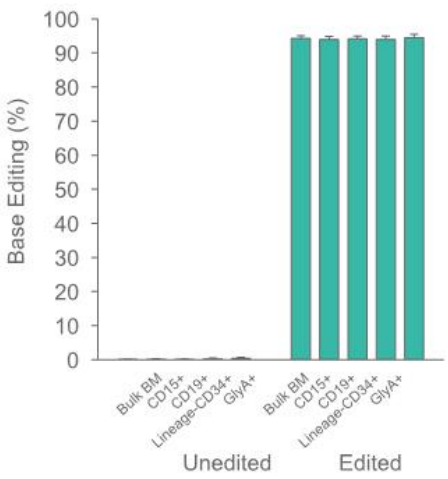
Experiments shown are *in vitro*-derived erythroid cells

BEAM-101: High levels of editing and robust HbF induction after long-term *in vivo* engraftment

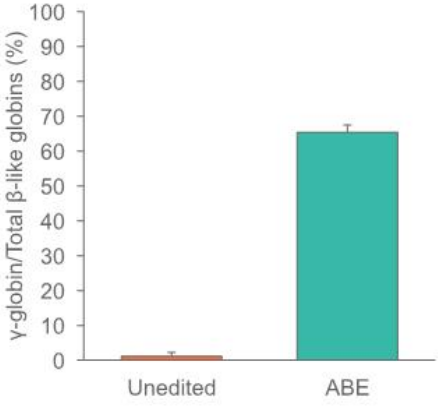
>90% human chimerism in bone marrow 16 weeks post-transplant[a]



>90% base editing at *HBG1/2* promoters in multilineage cells[b]

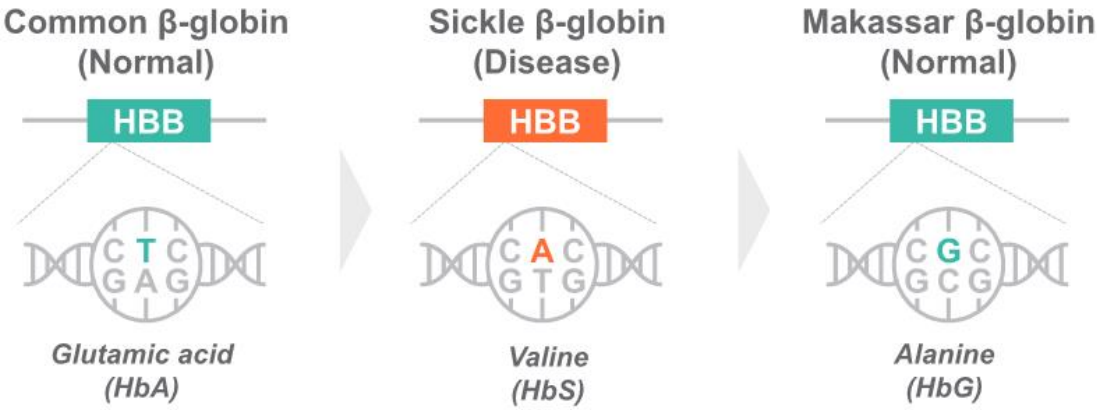


>65% gamma globin protein levels in sorted erythroid cells[c]



*Next Generation Variant; a. Mean±SEM; n=3 (8 weeks); n=6 (16 weeks); b. Mean±SEM; n=4-6 (16 weeks); Sorted human HSPCs (Lineage-CD34+), myeloid (CD15+), lymphoid (CD19+) and erythroid (GlyA+) cells (derived from BM samples) at 16 weeks post-transplantation; c. Mean±SEM; n=5 (16 weeks)

BEAM-102: Direct correction of the sickle causing mutation

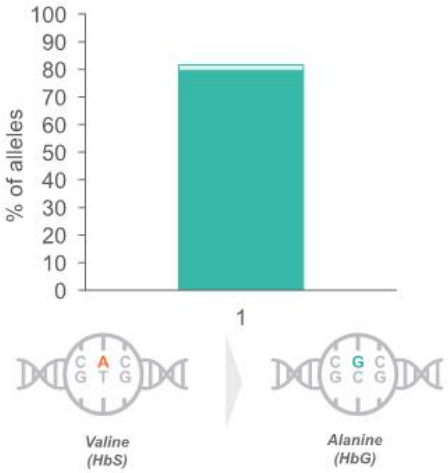


- Base editing recreates naturally-occurring human variant Hb-G Makassar which has alanine (E6A) instead of sickle-causing valine (E6V)[1]
- Hb-G Makassar is a normal β-globin variant and does not cause sickle disease, e.g., blood smear shows negative for sickle cells[2]

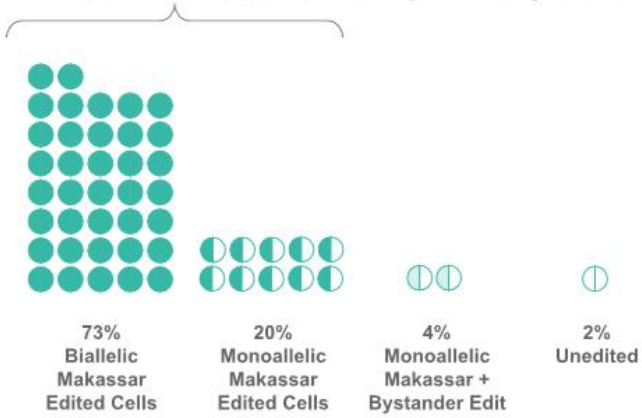
1. Cummings, MR. 2006. Human Heredity: Principles and Issues, Updated Edition.; 2. Mohamad, et al. 2018. Hematology Reports.

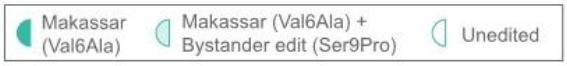
BEAM-102: Highly efficient, novel direct correction of sickle mutation in sickle patient cells

80% Sickle → Makassar correction in sickle patient CD34 cells with ABE (N=100)



~93% of cells have at least one sickle allele converted to Makassar and are potentially cured

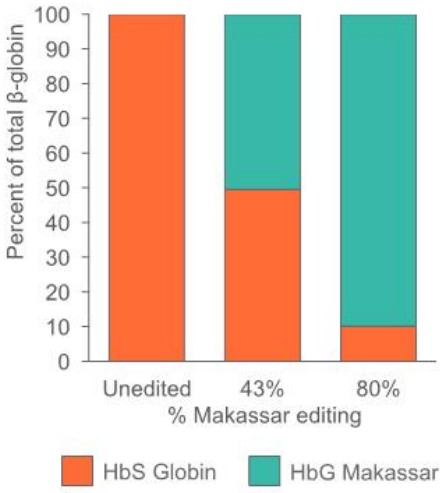




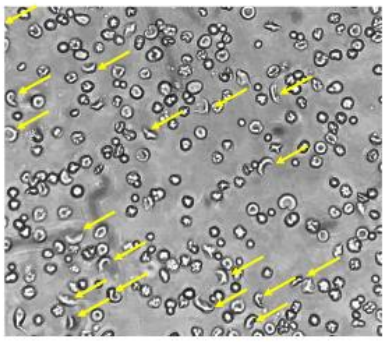
Allelic editing of 100 individual erythroid colonies post-electroporation was assessed for the Makassar edit by NGS using cells from HbSS donor.

BEAM-102: High editing of sickle mutation leads to significant elimination of HbS globin in patient donor cells

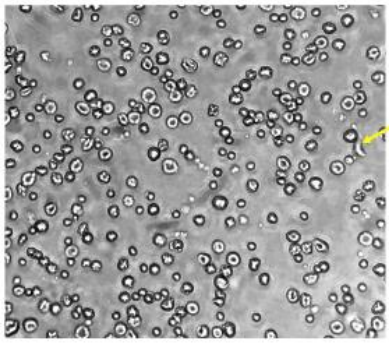
Elimination of HbS globin



Sickling in unedited HbSS cells



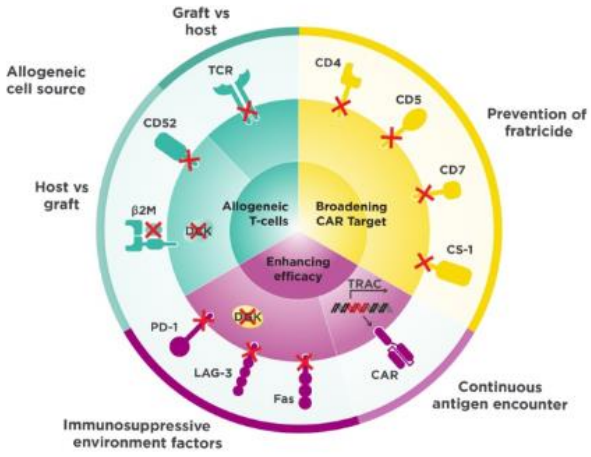
~89% Hb G-Makassar by UPLC



Sickle (HbS) and Makassar variant globin protein, at varying bulk levels of Makassar editing assessed by NGS, was measured by UPLC and expressed as a fraction of total beta globin in 18 day mature RBCs derived from edited HbSS CD34+s. UPLC was conducted on n = 2 for each bulk editing condition. CD34+ HbSS cells were edited and subsequently differentiated to generate mature erythroid red blood cells and exposed to low oxygen conditions (<2%) in a hypoxic chamber. Image is representative of n=2 different sickling assays from n=2 independent donors that were successfully edited at high levels (>80% by NGS) and confirmed to have near 90% Makassar globin by UPLC.

Two base editing approaches to silence genes

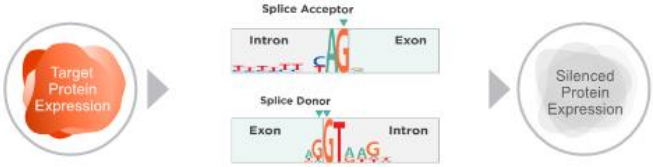
Expanding list of targets for CAR-T editing[1]



Two strategies for silencing with base editors

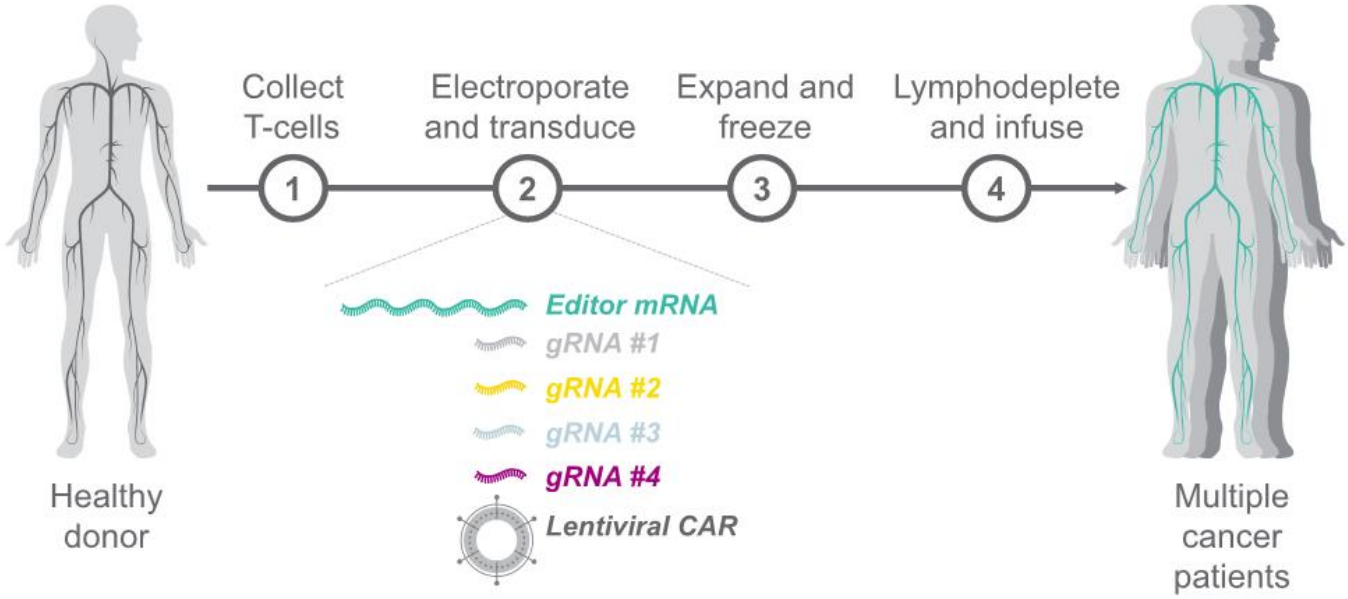
Create a stop codon with CBE

Splice disruption with CBE

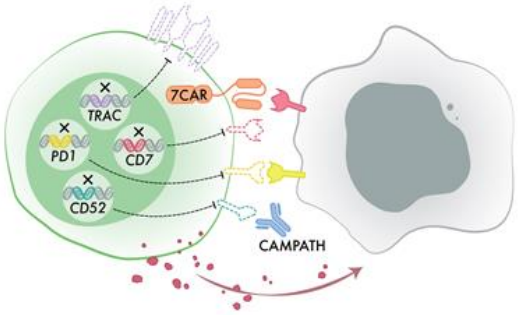


1. Adapted from Jung & Lee. 2018. *Molecules and Cells*.

Allogeneic multiplex edited CAR-T cell process



BEAM-201: Multiplex editing across four genomic loci with base editing

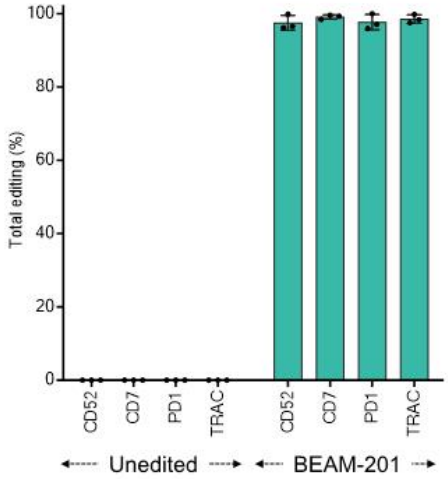


- Single electroporation of base editor mRNA and 4 gRNAs
- With few options to induce a second remission, post-relapse survival for T-cell acute lymphoblastic leukemia (T-ALL) is low

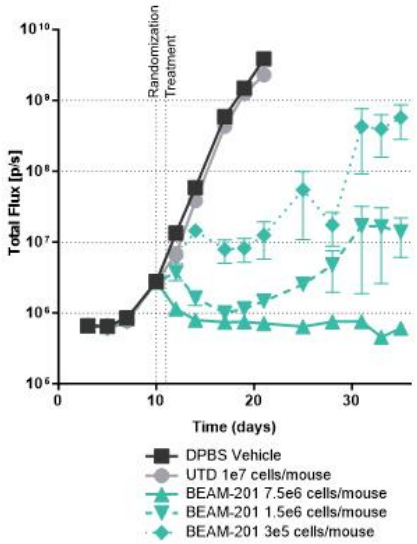
- TRAC: Prevent graft-vs-host disease
- CD52: Enable an allogeneic cell source to reduce host rejection of BEAM-201 cells
- PDCD1: Minimize immunosuppression of BEAM-201 cells and prolong efficacy for attacking the tumor
- CD7: Prevent fratricide (i.e., CAR-T cells attacking each other before they can attack the tumor)

Clinical scale BEAM-201 process produces 96-99% individual gene knock-out & potent in vivo activity

Clinical-scale process produces individually 96-99% editing, estimated 91% quad edited

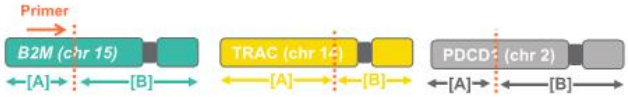


Potent in vivo tumor clearance or control across a 25-fold dose



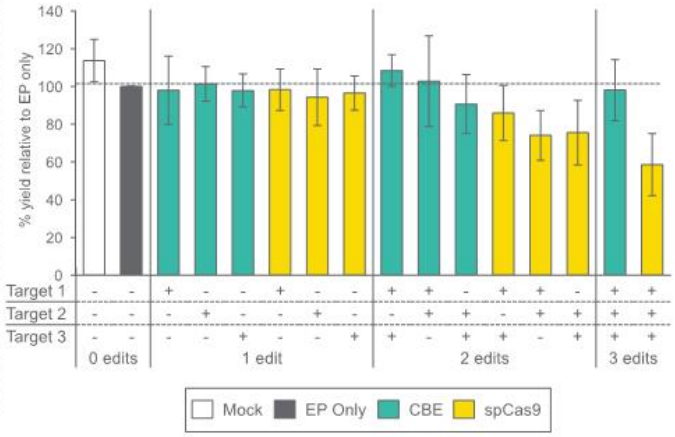
Multiplex base editing with no detectable translocations and maintained cell expansion

Unlike Cas9, no detectable CBE-induced rearrangements in triple-edited T cells (3 donors)[1]



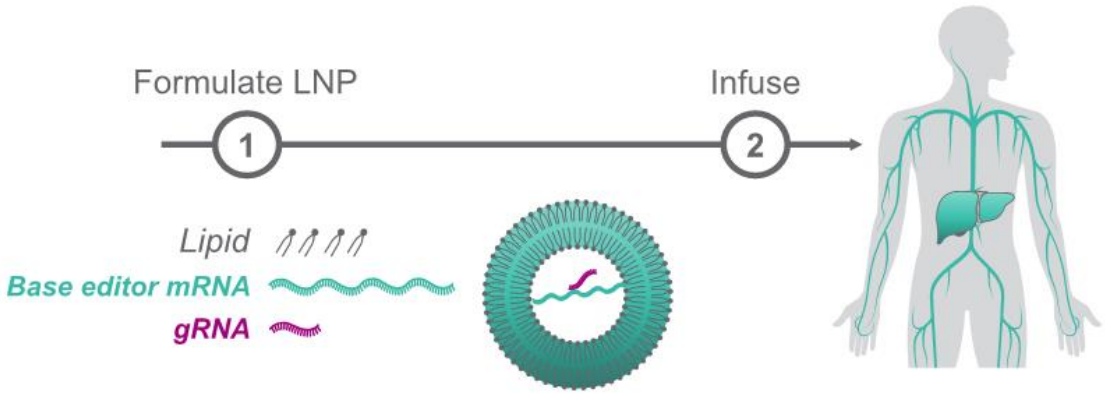
Type	Gene [Fragment]	Control	(% of reads) CBE-treated	(% of reads) Cas9-treated
Genomic rearrangement products (% rearranged)	B2M [A] / TRAC [A]	0	**0**	**0.9%**
	B2M [A] / TRAC [B]	0	**0**	**0.4%**
	B2M [A] / PDCD1 [A]	0	**0**	**1.6%**
	B2M [A] / PDCD1 [B]	0	**0**	**0.5%**
	B2M [B] / TRAC [A][2]	0	**0**	**0.5%**

Higher yield for CBE triple-edited cells relative to Cas9 triple-edited cells (N=6)[3]



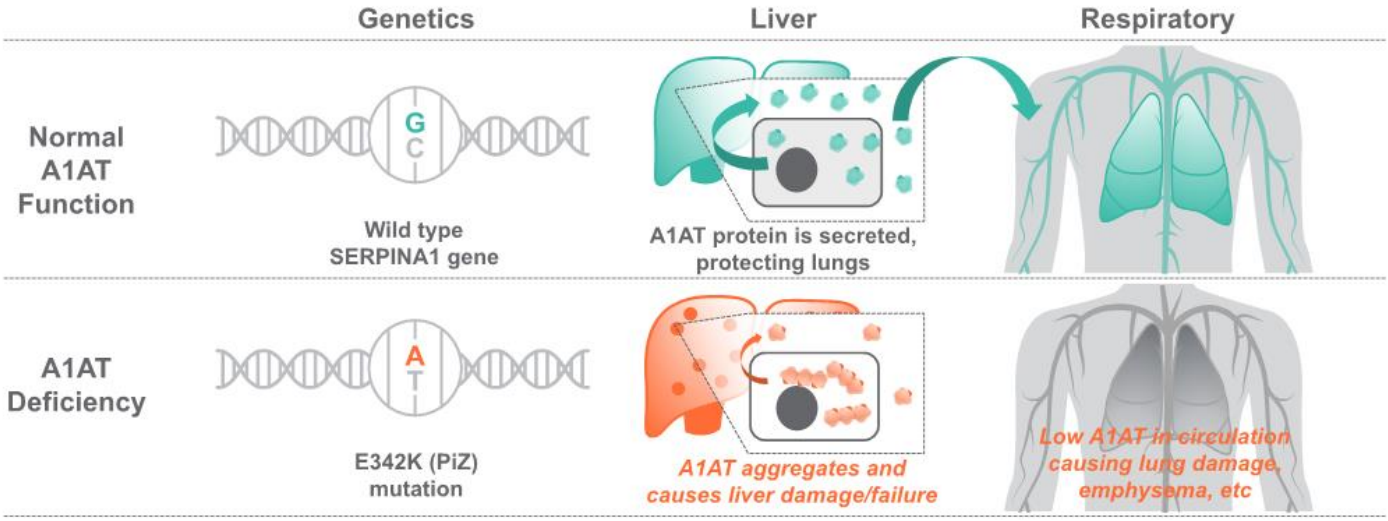
1. Lower limit of detection for rearrangements of 0.1%; 2. B2M-B only measurable if translocation includes local rearrangement in B2M; 3. Extensive guide screen across three targets, with both BE4 and spCas9 sgRNAs, selected for high editing efficiency and expansion in single-plex test, final cell yields compared between 1, 2 and 3 edits using BE4 and spCas9, normalized to electroporation only control; B2M = beta 2 microglobulin gene; TRAC = T-cell receptor alpha constant chain gene; PDCD1 = programmed cell death protein 1 gene;

Non-viral delivery for *in vivo* base editing



- Clinically validated technology for transient, in vivo delivery to the liver
- Scalable LNP manufacturing
- Beam has developed several proprietary LNP formulations

Alpha-1 Antitrypsin (A1AT) Deficiency

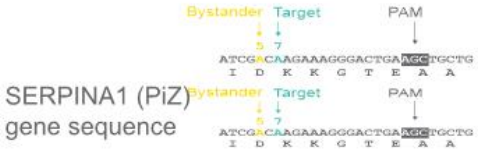


Direct correction of the PiZ mutation through base editing can:

1. Restore circulation of functional A1AT to protect the lungs
2. Reduce liver toxicity caused by mutant protein aggregates

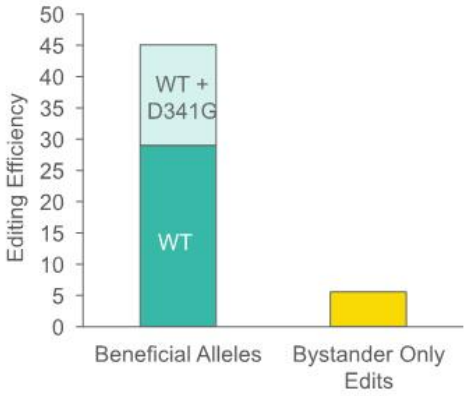
Direct correction of PiZ mutation by base editing in primary PiZZ fibroblasts

Alpha-1 Antitrypsin Deficiency base editing products

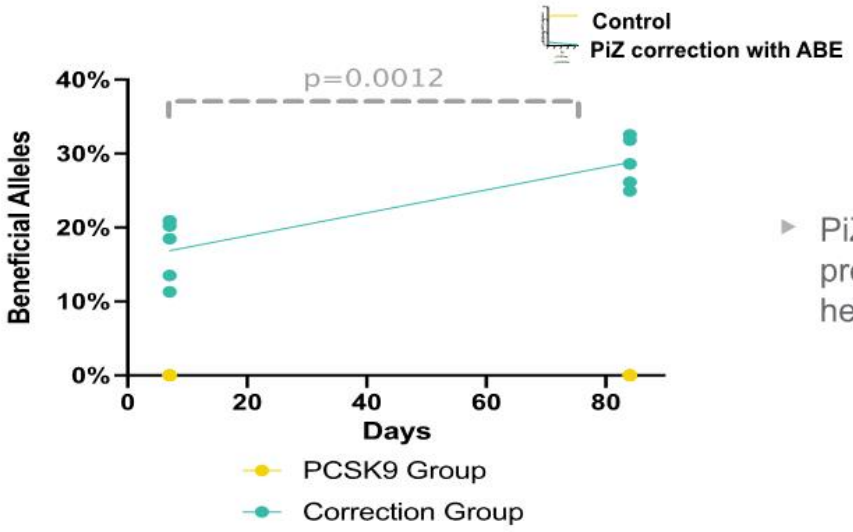


ABE edits		Resulting A1AT Variant	
Target	→	WT	Beneficial alleles are secreted and have elastase activity
Target + Bystander	→	WT + D341G	
Bystander only	→	E342K + D341G	

~45% correction of PiZ mutation in primary PiZZ fibroblasts



LNP-mediated *in vivo* correction of the PiZ mutation increases over time

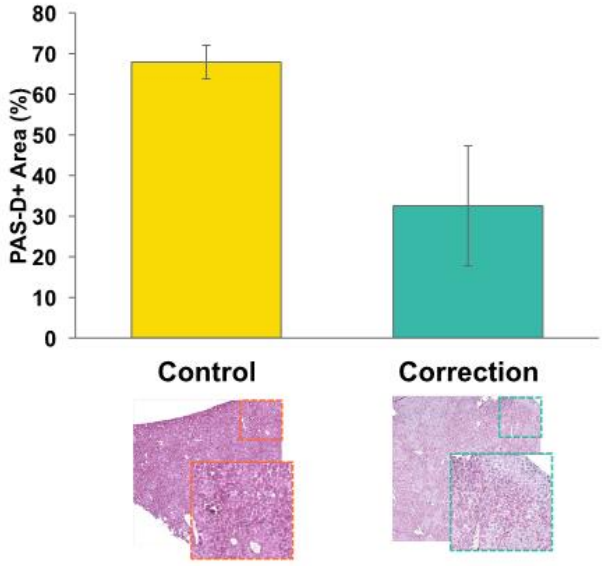


- PiZ correction may confer a proliferative advantage to edited hepatocytes

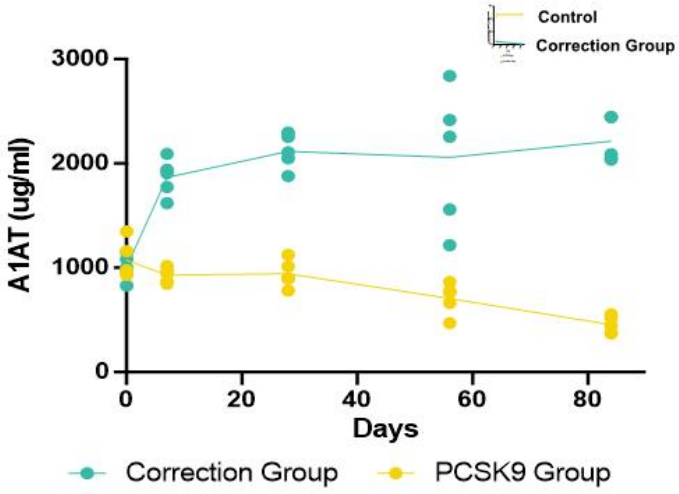
P value assessed by T-test

In vivo direct correction of A1AT mutation with base editing – addressing both liver and lung

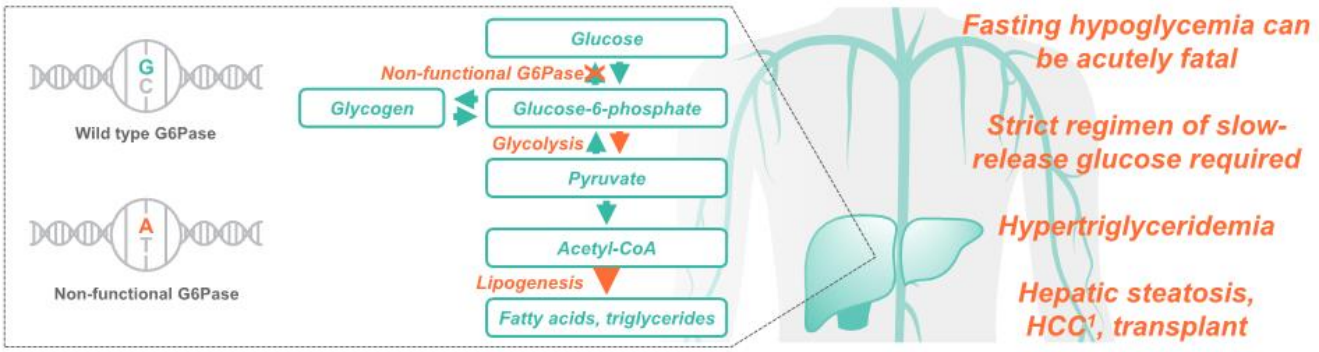
Reduction in toxic liver aggregates



4.9-fold increase in functional A1AT secretion



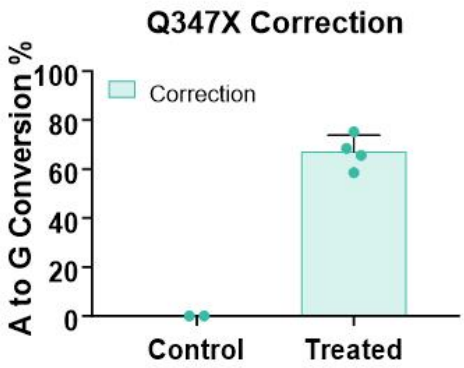
Glycogen Storage Disease Type 1a

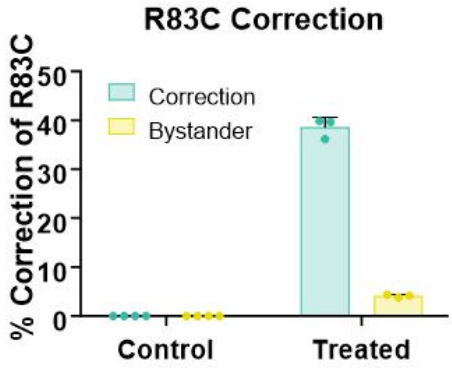


- Genetic liver disease caused by mutations in G6PC gene encoding G6Pase
- R83C and Q347X mutations affect 900 and 500 patients in US, respectively
- G6PC activity reaching >11% of wildtype activity may be enough to improve liver histology and survival[2]

1. Rake, et al. 2002. Eur. J. Pediatr.; 2. Chou & Mansfield. 2007. *Curr. Gen. Ther.*

ABE achieves ~70% Q347X correction and ~40% R83C correction in vivo

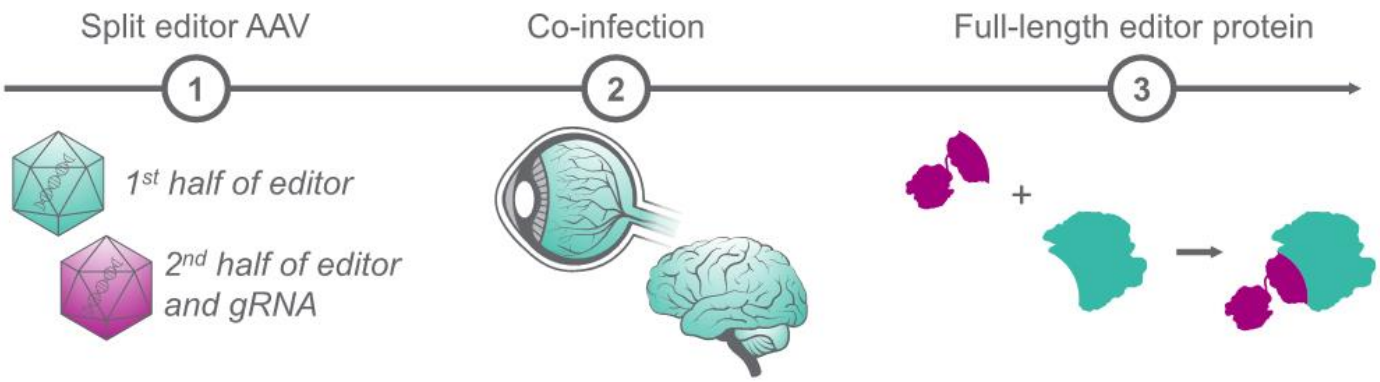




- Unlike AAV gene therapy that can wane, precise correction of G6PC may provide life-long genetic change, passed through cell division, and may enable treatment in children
- Precise correction at native locus would restore the endogenous regulation of tightly coordinated glucose metabolism

Data shown: In Vivo Correction of GSD1a Mutations in Livers of Transgenic Mouse Models, Heterozygous for either huG6PC-R83C or huG6PC-Q347X. Note control PCSK9 editing reached 50-60% editing and was consistent between models

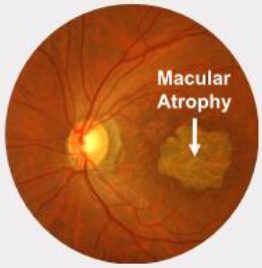
Viral delivery for *in vivo* base editing



- Clinically validated technology for in vivo delivery to a variety of tissue types
- Split intein technology designed to deliver base editor and guide RNA by co-infection with two viruses
  - DNA base editors are larger than the ~4.5kb limit of AAV vectors

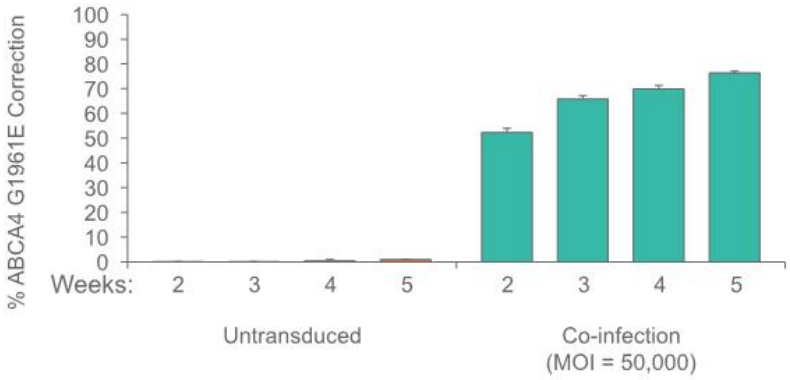
Stargardt Macular Dystrophy

- ABCA4 mutations cause central vision loss and death of photoreceptor cells
- Most prevalent G1961E mutation accounts for 5,500 patients in US
- No approved therapies today
- ABCA4 gene cannot be packaged into a single AAV vector due to size



Retina Image Bank

ABE achieves ~75% correction delivered with split AAV



- Editing levels in excess of 12-20% expected to be disease-modifying

Data shown: ABE editing of *surrogate base after split AAV delivery in retinal cells (N=3). *Surrogate base is the third base in the disease target GAA codon, which is also present in normal non-Stargardt cells; MOI = Multiplicity of Infection

Strategic collaborations and investment in future capabilities

- **Developing potential best-in-class disease-specific base editing therapies**

verve THERAPEUTICS	Base editing for the prevention of cardiovascular disease
magenta THERAPEUTICS	Non-ablative conditioning for improved hematology transplant in combination with BEAM-101 and BEAM-102

- **Translational and clinical research with world-class teams**

Boston Children's Hospital — Until every child is well	Clinical and research activities for sickle cell disease, leukemias, and other diseases
IOB Institute of Molecular and Clinical Ophthalmology Basel	Preclinical research collaboration for base editing in Stargardt Disease

- **Critical internal capabilities to accelerate future development**

ALEXANDRIA®	Custom build of 100,000 ft² cGMP facility supporting *ex vivo* and *in vivo* clinical and commercial manufacturing for base editing programs

Meet the Beam Team

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FOUNDATION MEDICINE
BIOMERIEUX
Pfizer
blueprint MEDICINES
Voyager THERAPEUTICS
Aegerion Pharmaceuticals
AMGEN
Johnson & Johnson
Biogen

Collectively contributed to development of 13 approved products and 70 IND filings

Thank you