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Corporate Presentation

August 4, 2021

Forward Looking Statements

This presentation contains forward-looking statements and information within the meaning of The Private Securities Litigation Reform Act of 1995. The words ‘‘anticipate,’’ ‘‘believe,’’ ‘‘continue,’’ ‘‘could,’’ ‘‘estimate,’’ ‘‘expect,’’ ‘‘intend,’’ ‘‘may,’’ ‘‘plan,’’ ‘‘potential,’’ ‘‘predict,’’ ‘‘project,’’ ‘‘target,’’ ‘‘should,’’ ‘‘would,’’ and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Forward-looking statements in this presentation include statements regarding the initiation, timing, progress and results of the Company’s preclinical and clinical studies and its research and development programs, including beginning patient dosing in the RUBY trial by the end of 2021, the timing for the Company’s receipt and presentation of data from its clinical trials and preclinical studies, including presenting initial clinical data from the BRILLIANCE trial in September 2021, and the timing or likelihood of regulatory filings and approvals, including filing an IND for EDIT-301 for the treatment of beta-thalassemia by the end of 2021. The Company may not actually achieve the plans, intentions, or expectations disclosed in these forward-looking statements, and you should not place undue reliance on these forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in these forward-looking statements as a result of various factors, including: uncertainties inherent in the initiation and completion of pre-clinical studies and clinical trials and clinical development of the Company’s product candidates; availability and timing of results from pre-clinical studies and clinical trials; whether interim results from a clinical trial will be predictive of the final results of the trial or the results of future trials; expectations for regulatory approvals to conduct trials or to market products and availability of funding sufficient for the Company’s foreseeable and unforeseeable operating expenses and capital expenditure requirements. These and other risks are described in greater detail under the caption “Risk Factors” included in the Company’s most recent Annual Report on Form 10-K, which is on file with the Securities and Exchange Commission, as updated by the Company’s subsequent filings with the Securities and Exchange Commission, and in other filings that the Company may make with the Securities and Exchange Commission in the future. Any forward-looking statements contained in this presentation represent Company’s views only as of the date hereof and should not be relied upon as representing its views as of any subsequent date. Except as required by law, the Company explicitly disclaims any obligation to update any forward-looking statements.

Editas is Transforming Medicine

Harnessing the power and potential of gene editing to develop medicines for people living with serious diseases around the world

Striving to discover, develop, manufacture, and commercialize transformative, durable genomic medicines

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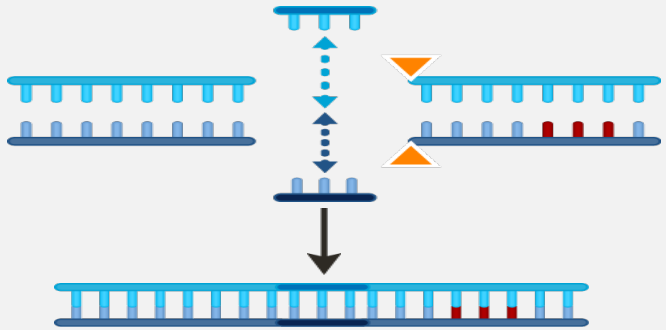
Editas Medicine's Powerful Engine

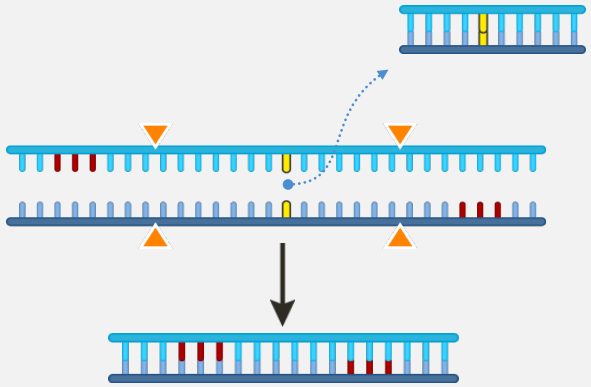
Editing Strategies for Precise Gene Editing Medicines

Disrupt



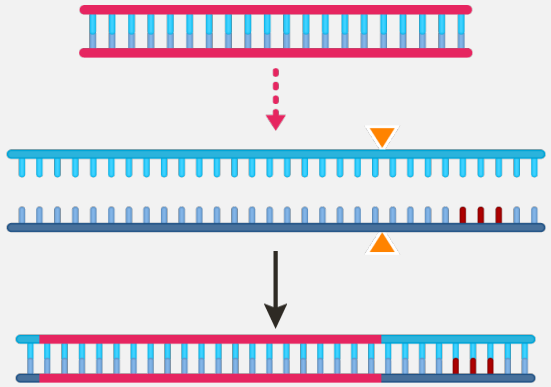
- T cell and iPSC programs
- EDIT-301 (*HBG* promoter)

Remove



- EDIT-101 (*CEP290*)
- EDIT-102 (*USH2A*)

Insert or Replace



- T cell and iPSC programs
- RP4 (*RHO*)

Differentiated Platform
The *only* company with multiple proprietary CRISPR editing systems

Unparalleled IP
Broadest and deepest CRISPR IP portfolio

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Three Platforms of Medicine Development

Built on a Powerful Gene Editing Engine and a Talented Team

1 *In Vivo*

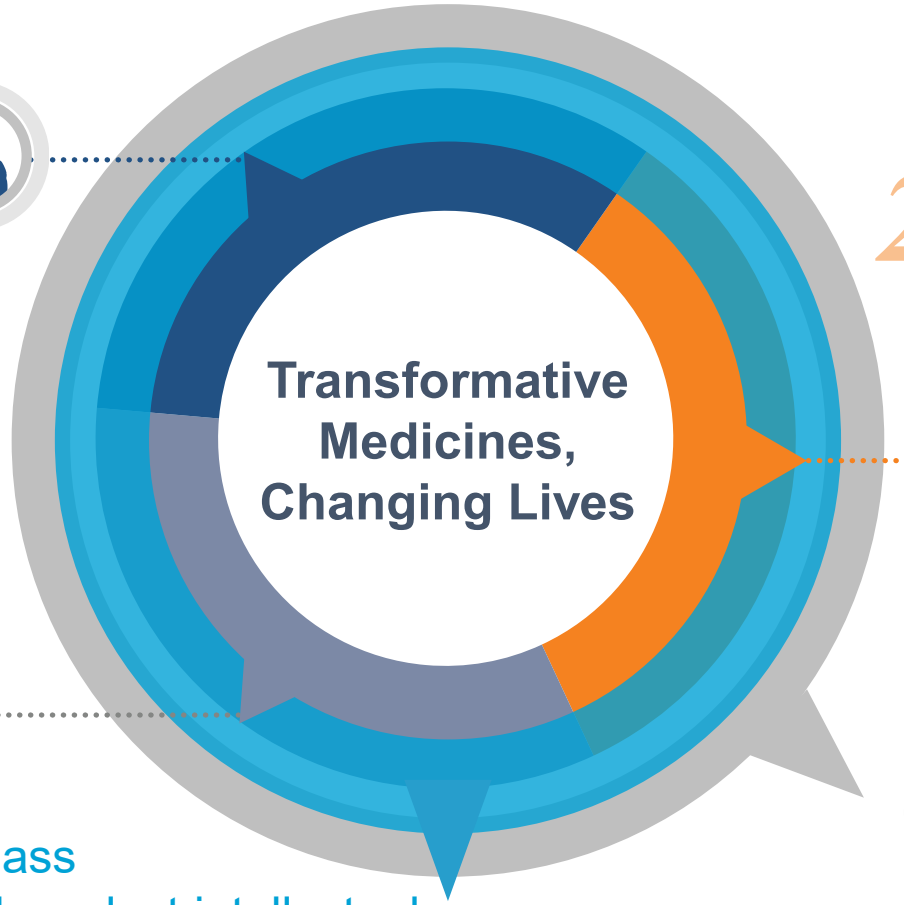
Global leader in *in vivo* gene editing medicines, starting with LCA10, moving into other inherited retinal diseases, and eventually into different tissues

2 *Ex Vivo*

Differentiated approach for treating sickle cell and beta thalassemia

3 *Cell Therapy*

Gene edited iPSC NK cells to revolutionize cancer treatments for multiple tumor types



The Engine: best-in-class gene editing platform, broadest intellectual property, flexible and robust manufacturing capabilities

The People: seasoned executive team supported by world-class scientists

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Right Edits, Right Cells, Right Delivery

In Vivo

Successful editing of the CEP290 gene for LCA10

Efficient editing in LCA10 patient derived cells correcting mutations *in vitro* and NHPs *in vivo*

First ever *in vivo* gene editing medicine administered to humans

Leverage findings from LCA10 to move into other inherited retinal diseases and eventually into different tissues

Ex Vivo

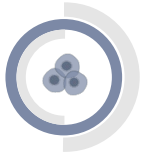
Efficient and reproducible genome editing in human HSCs *ex vivo*

Highly efficient and safer editing of beta-globin locus leading to reduced sickling and higher fetal hemoglobin

Entering clinic with potential best-in-class product for sickle cell disease

Expand *ex vivo* platform to address other autologous cell therapies

Cell Therapy



Demonstrated >90% of editing in initial targets

Successful multiplex editing on various cell lines

Enhanced tumor killing with double knockout iNK cells

Develop off-the-shelf iPSC-derived cells to revolutionize cancer treatments for multiple tumor types and other cell mediated diseases

PROOF OF CONCEPT	VALIDATION	PROGRESS	FUTURE

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Highly Differentiated Editas-Engineered Nuclease

Proprietary Eng. AsCas12 Overcomes the Activity/Specificity Tradeoff of Prior Nucleases

	Why it matters	SpCas9	SaCas9	Eng. AsCas12a
Specificity	Reduces safety risks	Low	High	High
Single AAV delivery	Efficient AAV delivery for in vivo medicines	No	Yes	Yes
Guide RNA total size (bp) (shorter is better)	Ease of guide RNA manufacturing	100	100	40-66
Guide RNA targeting	On-target efficacy and specificity for synthetic RNAs	Low	Low	High

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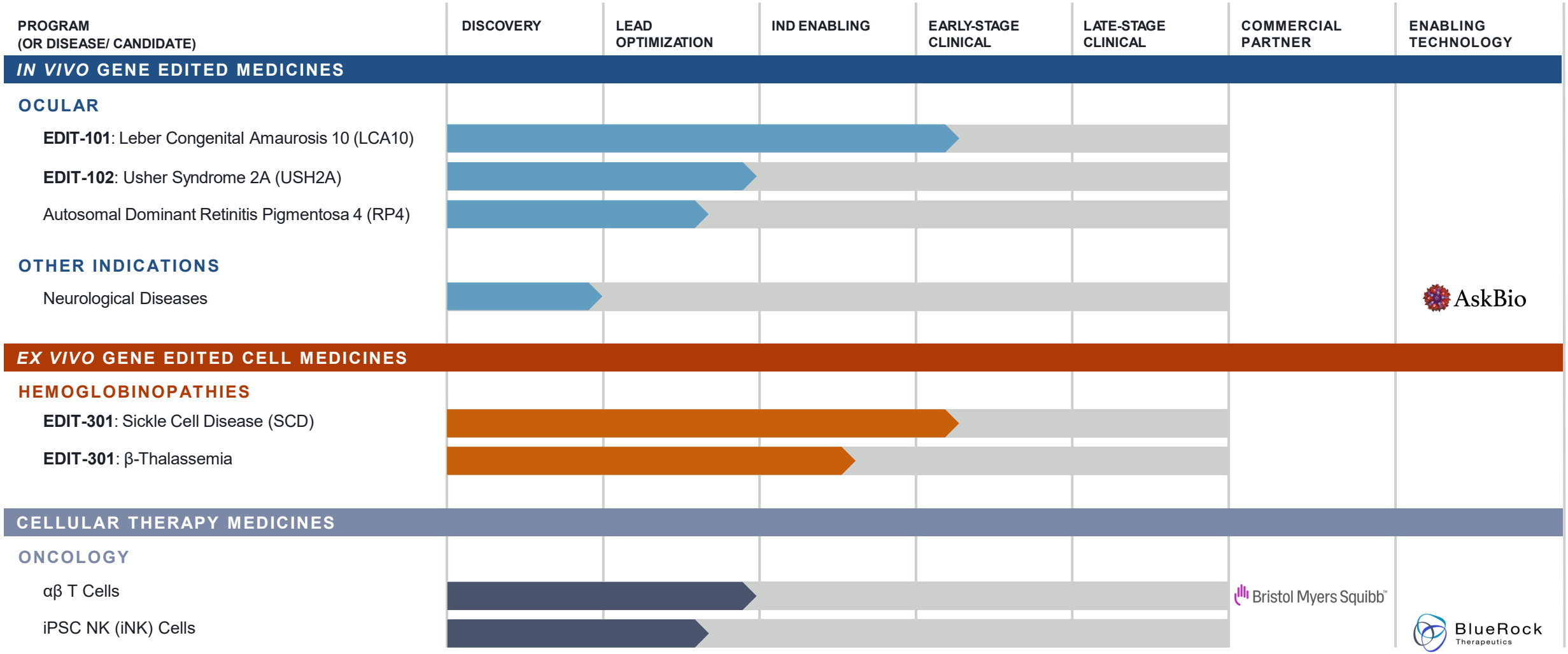
Pipeline



iPSC: induced pluripotent stem cell

2021 Anticipated Milestones

In Vivo Gene Edited Medicines

Ocular

- ☑ Initiate dosing of second cohort for Brilliance trial for EDIT-101 in Q1 2021
- ○ Present clinical data for EDIT-101 by year-end
- ○ Declare development candidate for RP4 by year-end

Other Indications

- ○ Advance *in vivo* gene edited medicines with AskBio

Ex Vivo Gene Edited Cell Medicines

Hematology

- ○ Initiate dosing of EDIT-301 for Ruby trial for sickle cell disease
- ○ File IND for EDIT-301 for beta-thalassemia by year-end

Cellular Therapy Medicines

Oncology

- ○ Advance *ex vivo* preclinical studies for a gene edited iNK cell medicine to treat solid tumors
- ○ Advance αβ T cell medicines in collaboration with Bristol Myers Squibb

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In Vivo Gene Edited Medicines

Potential to Address Significant Unmet Need

Initial Focus: Ocular

5.5 million patients with IRDs worldwide[1]

EDIT-101: Leber congenital amaurosis 10 (LCA10)

EDIT-102: Usher syndrome 2A (USH2A)

RP4 (Autosomal dominant retinitis pigmentosa 4)

Other inherited retinal diseases

Next Indication: Neurology

- Undisclosed neurological indication

Future Indications

Over 6,000 human genetic disorders[2]

- Neuromuscular
- Liver
- Hematology
- Central nervous system
- Cardiology
- Other therapeutic areas

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1 PNAS February 4, 2020 117 (5) 2710-2716
2 OMIM Gene Map Statistics

Ocular Program Overview

	EDIT-101: LCA-10	EDIT-102: USH2A	RP4	Undisclosed Target
Inheritance	Autosomal Recessive	Autosomal Recessive	Autosomal Dominant	
Gene	CEP-290	Usherin	Rhodopsin	
Mutation	c.2991+1655A>G mutation in intron 26 (IVS26)	Exon 13 mutations	RHO mutations	
Target Cells	Photoreceptors	Photoreceptors	Photoreceptors	
Presentation	Blindness/ severe visual impairment at or near birth	Loss of peripheral and night vision, eventual legal blindness	Reduced rod function, leading to night blindness, loss of peripheral vision	

EDIT-101 progress is de-risking subsequent ocular indications

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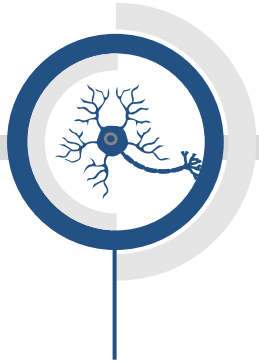
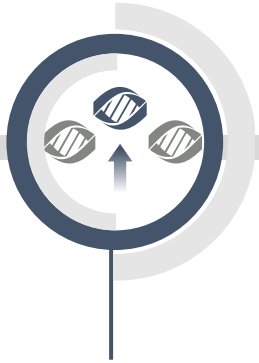
EDIT-101 to Treat Leber Congenital Amaurosis 10 (LCA10)

First Ever *In Vivo* Gene Edited Medicine Administered to Humans



Disease

Lack of outer segment of photoreceptors leading to blindness in childhood



Approach

Potentially cure genetic blindness by ***removing*** CEP290 genetic mutation in photoreceptors

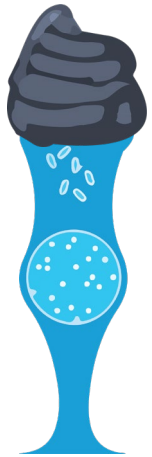
Status

Concurrently enrolling pediatric mid-dose and adult high-dose cohorts of Brilliance trial

Initial clinical data in September

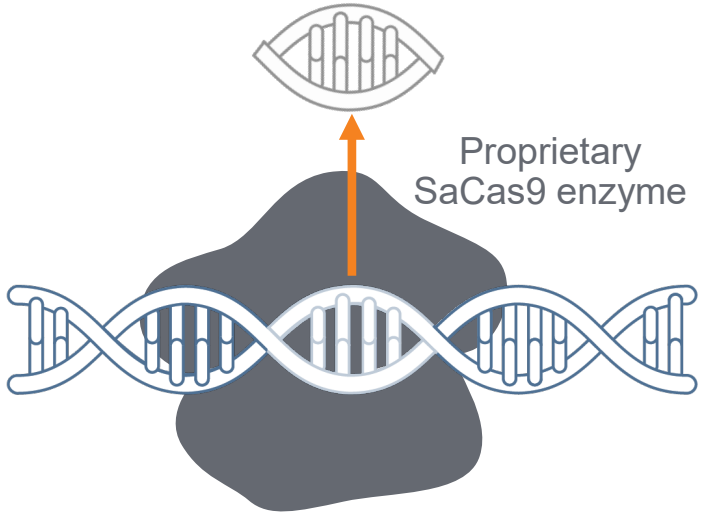
Removing Mutation with Editing to Correct Vision

LCA10 Photoreceptor



Outer segment absent due to CEP290 deficiency

EDIT-101



Single dose editing removes disease-causing mutation

Rescued Photoreceptor

Outer segment regenerates with CEP290 protein

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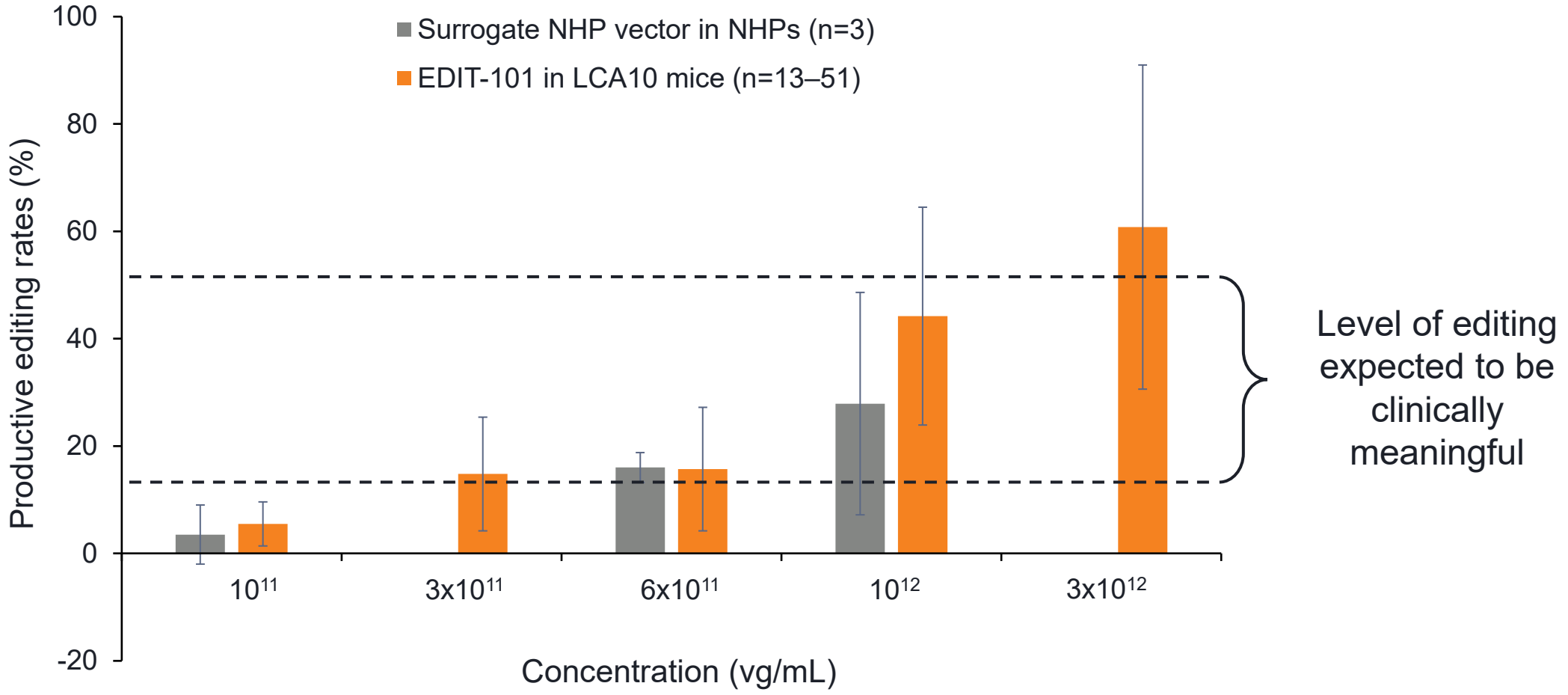
EDIT-101 Demonstrates Robust Productive Editing



At maximally tolerated doses, >50% editing is observed
EDIT-101 was well tolerated in NHPs

EDIT-101 Trial Design, Status & Update

brilliance
LCA10 Phase I/II Clinical Trial

STATUS

Enrolling patients in the pediatric mid-dose and adult high-dose cohorts.

PATIENTS

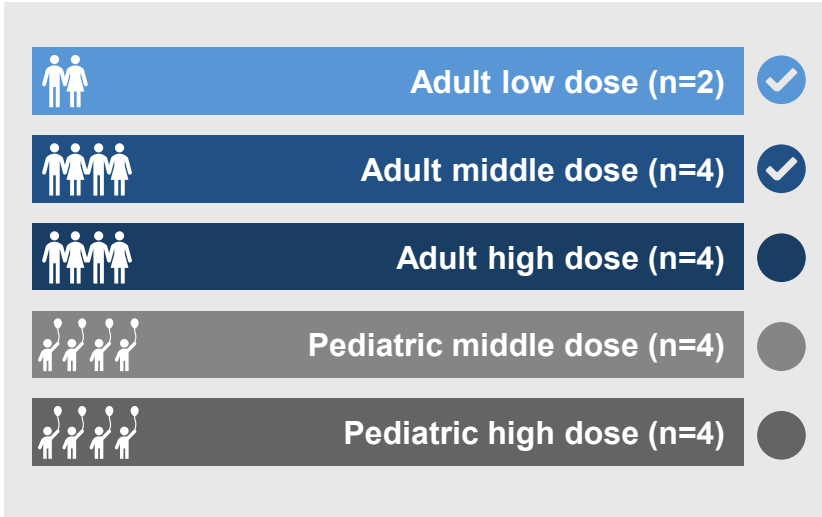
18 patients, aged 3 years and above

INTERVENTION

Single dose of EDIT-101 administered via subretinal injection to eye with worse vision

ENDPOINTS

- **Primary: Safety** including frequency and number of adverse events related to drug, procedure, and dose limiting toxicities
- **Secondary: Efficacy** including visual acuity, mobility course, macula thickness, pupillometry, and electroretinogram using patient's own baseline value for each efficacy measure



PROTOCOL

Based on safety in the first cohort, protocol was amended to broaden inclusion criteria of sentinel patients

SAFETY REVIEW

IDMC endorsed proceeding with first pediatric cohort based on a review of clinical safety data from adult low-dose and adult mid-dose cohorts

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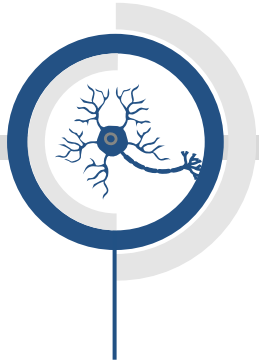
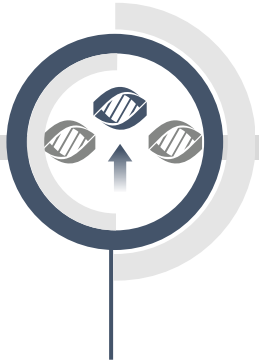
EDIT-102 to Treat Usher Syndrome Type 2A



Disease

Degeneration of photoreceptors causing progressive vision loss and blindness



Approach

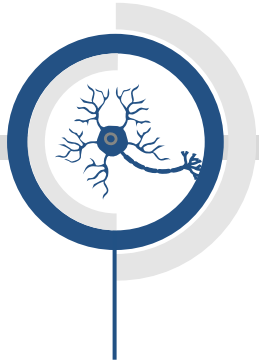
Remove USH2A mutation in photoreceptors using same AAV and promoter as EDIT-101

Status

Development candidate optimization

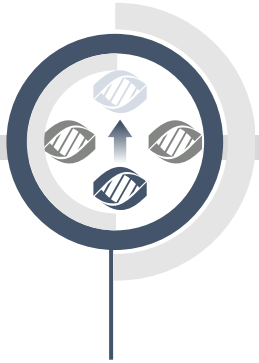
Supporting preclinical data presented at ARVO

Autosomal Dominant Retinitis Pigmentosa 4 (RP4)



Disease

Progressive decline in night vision, followed by peripheral vision, and eventual blindness



Approach

Replace rhodopsin gene in photoreceptors to correct all RP4 mutations with AAV

Status

Declare development candidate by year end 2021

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Ex Vivo Gene Edited Cell Medicines for Hemoglobinopathies

Potential Best-in-Class Medicine for Sickle Cell Disease and Beta-Thalassemia

Use proprietary Cas12a enzyme to edit beta-globin locus to safely, robustly, and durably increase fetal hemoglobin with single administration

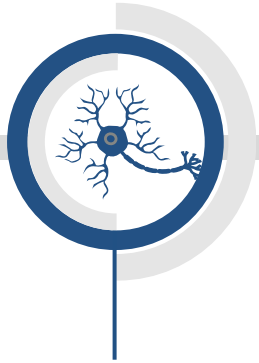
- Naturally occurring mutations support clinical relevance and safety of editing at the HBG1/2 promoter region
- Cas12a makes naturally more precise genomic alterations, reduces sickle globin, in contrast to lentiviral gene therapy
- Editing beta-globin locus provides level of inherent safety and more robustly repopulates red blood cell lineage, in contrast to editing at the BCL11A site
- Demonstration of no measurable off-targets

Epidemiology

165,000+ sickle cell patients and **15,000+** beta-thalassemia patients in the U.S. and Europe[1,2,3]

1 National Center on Birth Defects and Developmental Disabilities, Centers for Disease Control and Prevention
2 European Medicines Agency: EU/3/19/2242
3 DRG: Rare Diseases and Orphan Drugs, Oct 15, 2020

EDIT-301 to Treat Sickle Cell Disease and Beta-Thalassemia



Disease

Deformed and diminished blood cells causing anemia, organ failure, mortality, and pain crises in sickle cell disease

Approach

Leverage proprietary Cas12a enzyme to safely and effectively edit the HBG 1/2 promoter to increase fetal hemoglobin

Status

Screening sickle cell patients for dosing by year-end

On track to submit IND for β-thalassemia by year-end

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Goal is Superior Safety and Efficacy

Differentiated Approach Utilizing Proprietary Cas12a at the HBG1/2 Promoter

	EDIT-301: β-globin Locus	BCL11A Editing	Lentiviral Gene Therapy
EFFICACY			
Upregulates fetal hemoglobin?	✓	✓	✗
Reduces sickle globin expression?	✓	✓	✗
SAFETY			
Precise editing at specified location in genome?	✓	✓	✗
Targets natural locations of fetal hemoglobin mutations?	✓	✗	✗

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Cellular Therapy Medicines for Oncology

Leveraging iPSC Platform to Create Off-the-Shelf Treatments for Cancer

Gene edited iPSC cells are revolutionizing cancer therapy through numerous cellular advantages

- Unlimited self-renewal potential
- Single cell clonability for generating homogeneous cell population
- Proprietary site-specific knock-in technology ensures high-level expression of CARs
- Enhancing innate properties of NK cells
- Multi-edited, knock-ins & knock-outs, iNK cells increase activity and persistence
- Developing allogeneic therapeutic products for repeat dosing

Epidemiology

Over **1.3 million** new cases of solid tumor cancers, linked to over **400,000** deaths, per year in the US[1]

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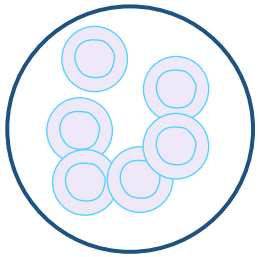
[1] American Cancer Society: Cancer Facts & Figures 2020

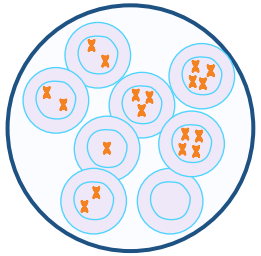
iNK Cell Medicine Process

START



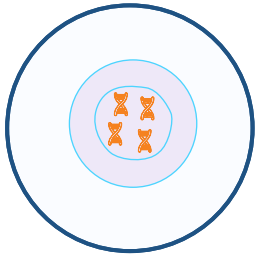
Access BlueRock Therapeutics' GMP IPS cells from renewable master cell bank

EDIT



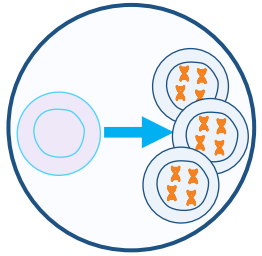
Make multiple edits in iPSCs with high efficiency using proprietary Cas12a

CLONE SELECTION



Screen and select single clone with perfectly characterized genome to create cell bank that is infinitely renewable

DIFFERENTIATE

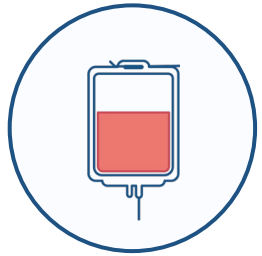


Differentiate edited iPSCs into edited iNK cells with proprietary, feeder-free differentiation process

FREEZE

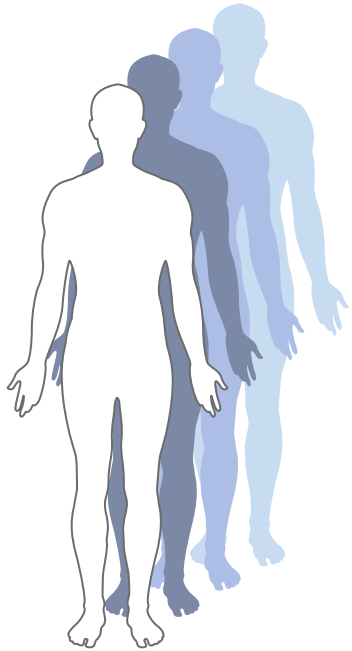
Edited iNK cells cryopreserved and stored for off-the-shelf availability to treat many patients

TREAT

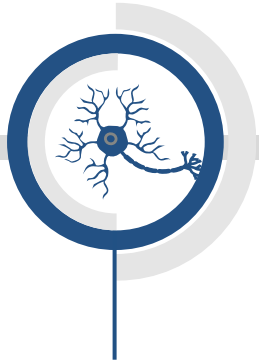


Edited iNK cells thawed and infused into patient

Patients



Gene Edited iPSC-Derived NK Cells For Solid Tumors



Disease

Malignant solid tumors that develop in lung, colon, breast, and other organs

Approach

Multiplexed gene editing of NK cells for enhanced tumor-killing, off-the-shelf cell therapies

Status

Advancing preclinical studies for development candidate

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Gene Edited iNK Cells Will Be Transformational for Cancer Patients

Advantages of NK Cells

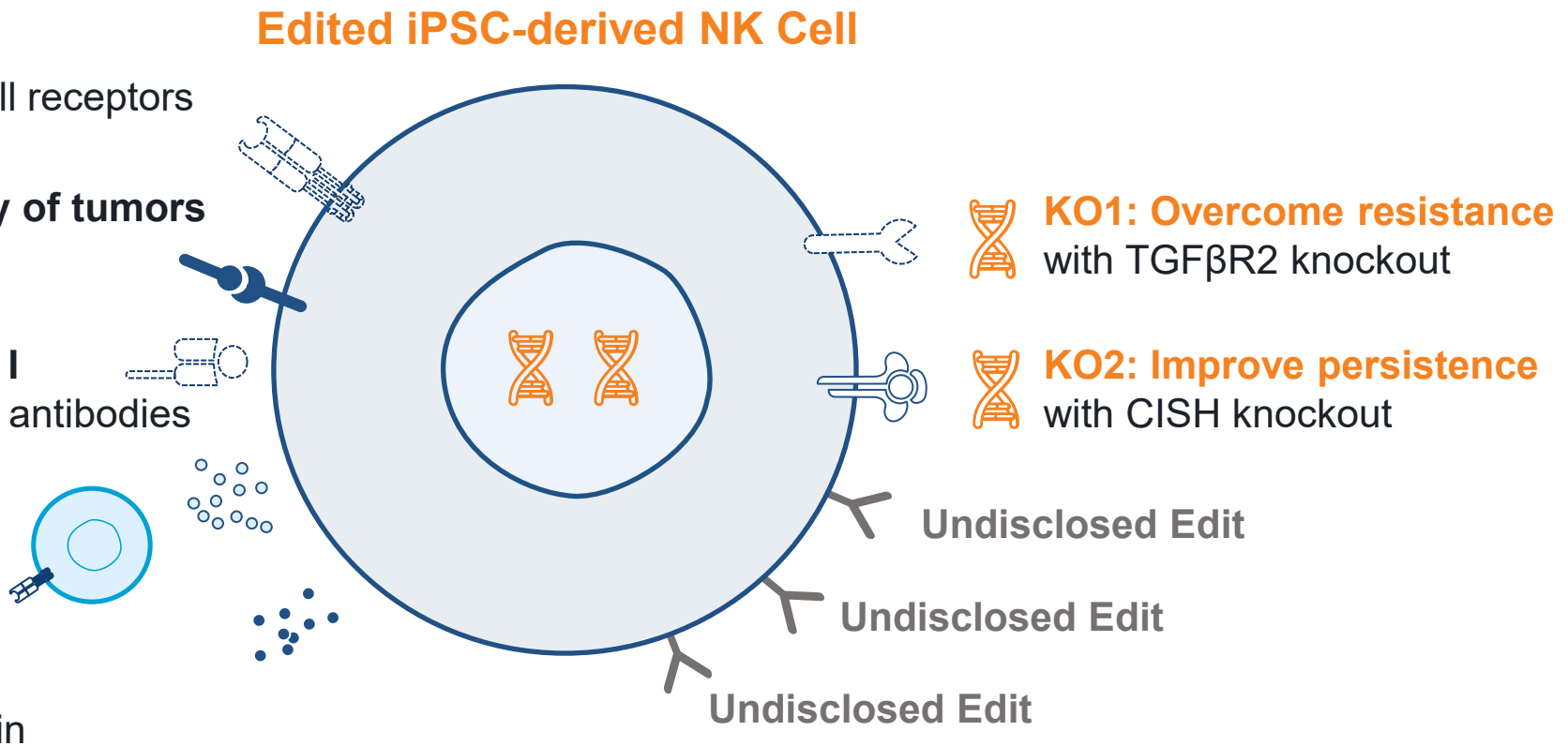
Naturally allogeneic
because they do not express T cell receptors

Rapidly recognize broader array of tumors
with stress ligand receptors

Recognize tumors lacking MHC I
that evade T cells and therapeutic antibodies

Recruit T cells to kill tumors
by releasing IFN-γ and TNF-α

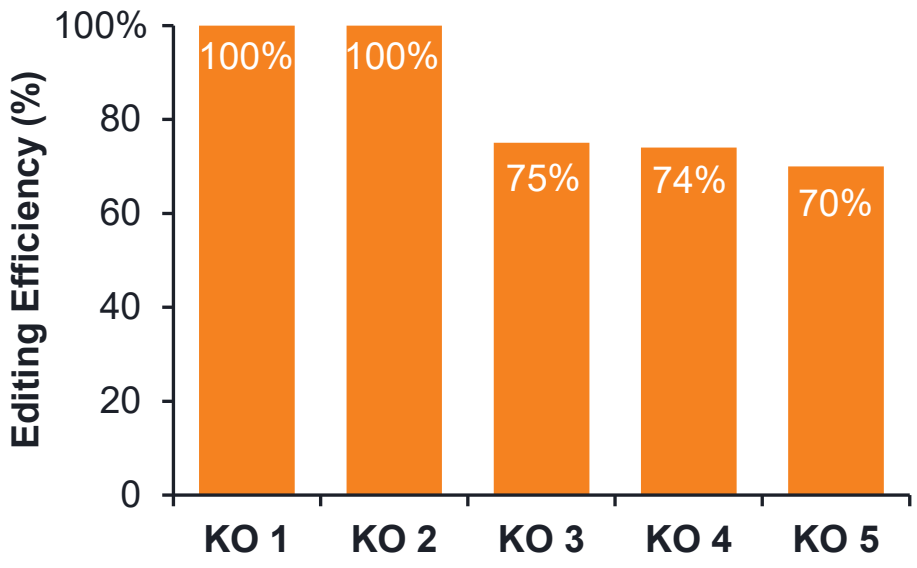
Directly kill tumors
by releasing granzyme and perforin



MHC I: major histocompatibility complex class 1; IFN-γ: interferon-gamma; TNF-α: tumor necrosis factor-alpha

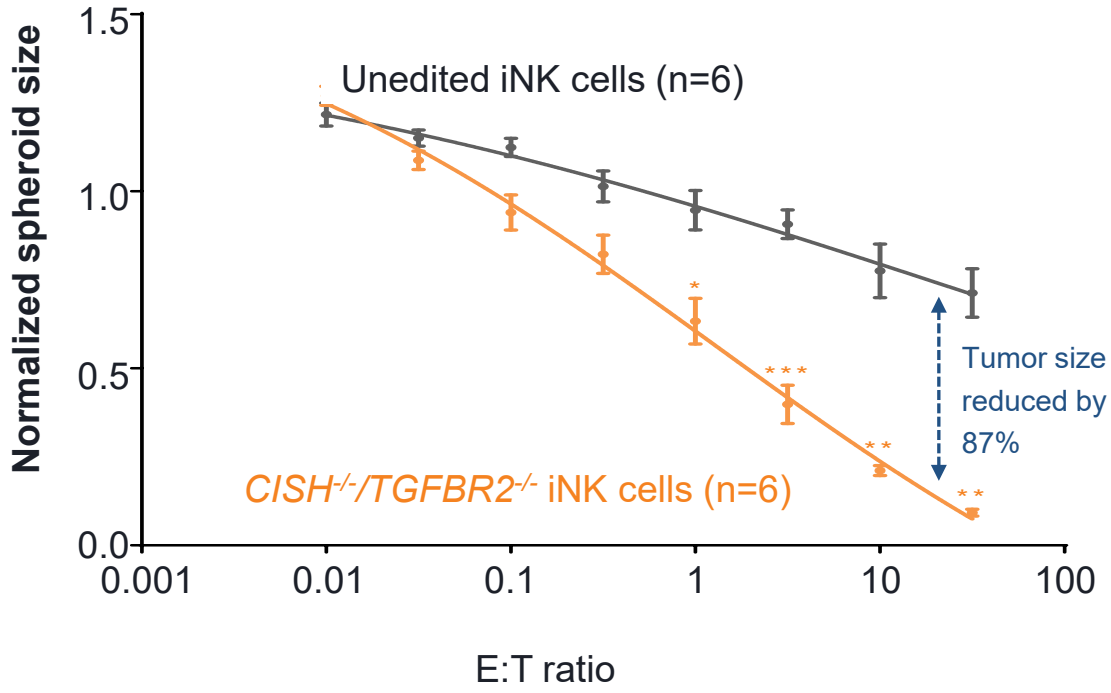
Efficient Editing and Sustained Anti-Tumor Activity

Efficient knock out of multiple genes in iNKs



DNA harvested from cells 4 days after electroporation

Greater reduction of SK-OV-3 spheroid size[1]



[1] Presented at ASH 2020
*p<0.05; **p<0.01; ***p<0.001 vs unedited iNK cells (two-way ANOVA, Sidak's multiple comparisons test)

Collaborations

GENE EDITED αβ T CELL MEDICINES FOR CANCER

DEVELOPMENT & COMMERCIALIZATION

Bristol Myers Squibb™

αβ T cell medicines to treat cancer and autoimmune diseases

$100 million in payments to date plus potential for milestones and tiered royalties

Currently working on **three programs** with **one declared development candidate**

IN VIVO GENE EDITED MEDICINES

ENABLING TECHNOLOGY

askbio

Collaboration bringing leading capsid development, clinical stage AAV vector delivery system, and manufacturing expertise

GENE EDITED iNK CELL MEDICINES FOR CANCER

BlueRock Therapeutics

Collaboration to create novel, allogeneic pluripotent stem cell (PSC) lines using BlueRock's induced pluripotent stem cell (iPSC) platform to develop CRISPR iNK cell medicines

Intellectual Property

Unmatched Intellectual Property Portfolio in CRISPR Gene Editing

Foundation

Exclusive foundational IP for CRISPR/ Cas9 and Cas12a (Cpf1) editing in human therapeutics

Breadth

Multiple species and CRISPR forms to address widest range of diseases

Depth

Over 220 issued patents, over 800 applications pending

Markets

Global coverage including US, Europe, Japan, Australia, Canada, China

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Executive Team

Seasoned Management Team Supported by World-Class Scientists

James C. Mullen
Chief Executive Officer

Biogen · Patheon

Michelle Robertson
Chief Financial Officer

Momenta · Baxalta / Shire · Sanofi Genzyme

Lisa A. Michaels, M.D.
Chief Medical Officer

Bayer · Rutgers Robert Wood Johnson Medical School · University of Virginia School of Medicine

Mark S. Shearman, Ph.D.
Chief Scientific Officer

agtc · EMD Serono · Merck

Bruce Eaton, Ph.D.
Chief Business Officer

somalogic · i2 Pharmaceuticals · NEXSTAR

Chi Li, Ph.D., MBA, RAC
Chief Regulatory Officer

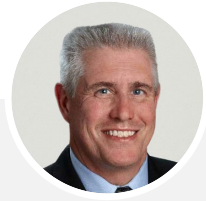
celularity · Allergan · Bayer

Charlene Stern, Ph.D., J.D.
Chief Legal Officer

AVEO Oncology · Goodwin · Foley Hoag LLP

Clare Carmichael
Chief Human Resources Officer

Alexion · Schering-Plough · Wave Life Sciences



Harry Gill
Senior Vice President, Operations

ThermoFisher Scientific · Pfizer · Baxter

Company Highlights

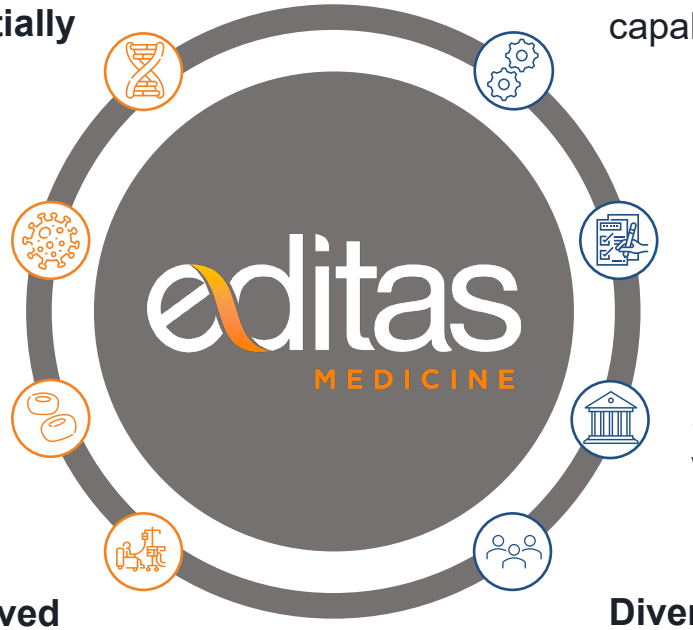
Best-In-Class *In Vivo* and *Ex Vivo* Gene Edited Medicines

First ever administration of an *in vivo* gene editing medicine in humans with **EDIT-101 for potentially curing genetic blindness**

Expanding *in vivo* gene editing medicines to address unmet monogenetic diseases worldwide

Developing **EDIT-301 as potential best-in-class *ex vivo* cell medicine** for sickle cell disease and beta-thalassemia

Developing potential **best-in-class iPSC-derived NK (iNK) cell medicines for solid tumors**



Financial, Operational and Organizational Excellence

Robust **internal and external manufacturing** capabilities, ready to scale for commercialization

Strong intellectual property position in the space with exclusive rights to foundational Cas9 and Cas12a patent estates

Sufficient capital to **sustain operations well into 2023**

Diverse and experienced leadership team bridging from research towards commercialization