Corporate Presentation

August 7, 2019

PIONEERING THE POSSIBLE

Forward Looking Statements

This presentation contains forward-looking statements within the meaning of the "safe harbor" provisions of The Private Securities Litigation Reform Act of 1995. All statements, other than statements of historical facts, contained in this presentation, including statements regarding the Company's strategy, future operations, future financial position, future revenue, projected costs, prospects, plans, and objectives of management, are forward-looking statements. 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 clinical trial timeline for EDIT-101 (AGN-151587) and the Company's 2022 goals. 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 preclinical studies and clinical trials and clinical development of the Company's product candidates; 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; availability of funding sufficient for the Company's foreseeable and unforeseeable operating expenses and capital expenditure requirements; and other factors discussed in the "Risk Factors" section of the Company's most recent Quarterly Report on Form 10-Q, which is on file with the Securities and Exchange Commission, and in other filings that the Company may make with the Securities and Exchange Commission in the future. In addition, the forward-looking statements included in this presentation represent the Company's views as of the date of this presentation. The Company anticipates that subsequent events and developments will cause its views to change. However, while the Company may elect to update these forward-looking statements at some point in the future, it specifically disclaims any obligation to do so. These forward-looking statements should not be relied upon as representing the Company's views as of any date subsequent to the date of this presentation.

Transforming Patient Lives

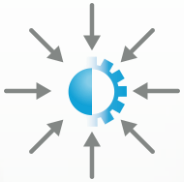
OUR MISSION

is to translate the promise of genome editing into a broad class of transformative genomic medicines to benefit the greatest number of patients

Pioneering the Possible

Clinical-stage company working to transform patient lives

- First *in vivo* CRISPR clinical trial in history (EDIT-101 for LCA10)
- EDIT-101 patient screening initiated and dosing on track for 2H19
- Rapidly translating exciting science into revolutionary medicines



Leader across CRISPR *in vivo* and engineered cell medicines

- Proven AAV delivery capability for *in vivo* CRISPR medicines
- Advancing engineered cell medicines to treat blood diseases and cancer
- Enabled by unparalleled platform of Cas9 and Cpf1 (Cas12a) enzymes

Strong progress towards EM22 goals

- EDIT-101 IND accepted by FDA upon initial review
- Usher syndrome 2A (USH2A) *in vivo* proof of concept established
- EDIT-301 for sickle cell and beta-thalassemia IND-enabling activities initiated

2019 Achievements and Goals

ACHIEVEMENTS

- Formed collaboration with BlueRock Therapeutics to advance universal allogeneic cell medicines for cancer
- Initiated IND-enabling activities for EDIT-301 for sickle cell and beta-thalassemia
- Established in vivo proof-of-concept for Usher syndrome 2A

GOALS

- EDIT-101 first patient dosing in second half
- EDIT-301 pre-clinical data in second half
- Usher syndrome 2A ready for IND-enabling studies by year-end

EM22: Our 2022 Vision for Editas Medicine

Build on Our Current Success

At least one program from our Celgene collaboration

More than one program in ocular diseases

Establish New Areas & Leverage Our Platform

At least one engineered cell medicine program beyond engineered T cells in cancer

At least one program in an additional cell or tissue type or using an advanced editing modality

Developing Best-in-Class CRISPR Medicines

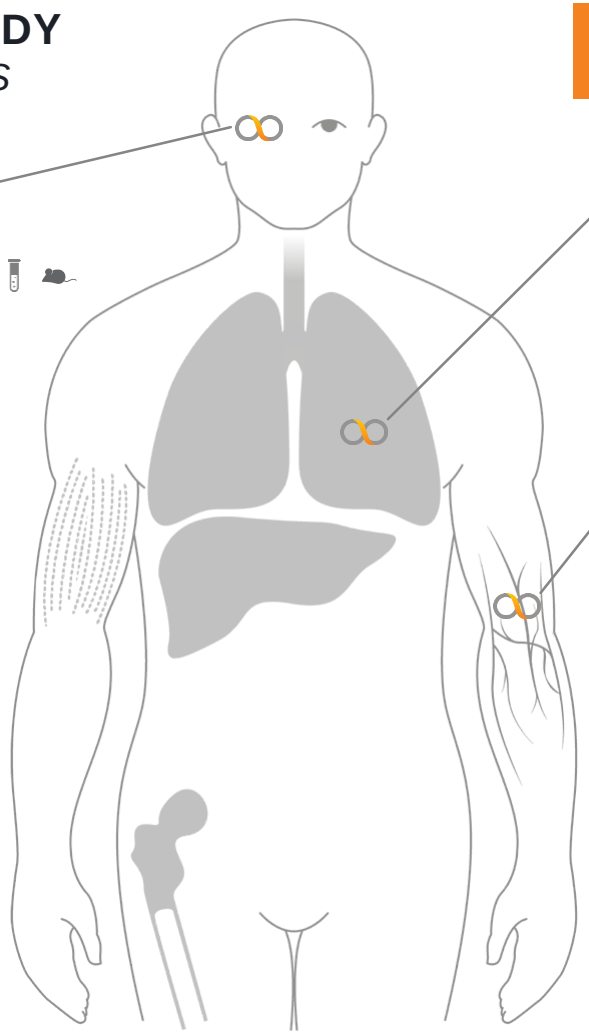
EDITING INSIDE THE BODY
IN VIVO *CRISPR MEDICINES*

OCULAR DISEASES
Leber congenital amaurosis 10*

Usher syndrome 2A

Retinitis pigmentosa

Ocular HSV



EDITING OUTSIDE THE BODY
ENGINEERED CELL MEDICINES

CANCER
Autologous T cell medicines**

Allogeneic cell medicines

BLOOD DISEASES
Sickle cell disease

Beta-thalassemia

EARLY DISCOVERY
Liver – AATD

Muscle – DMD

Lung – CF

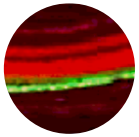
in vitro proof of biology

in vivo proof of concept

AAV delivery

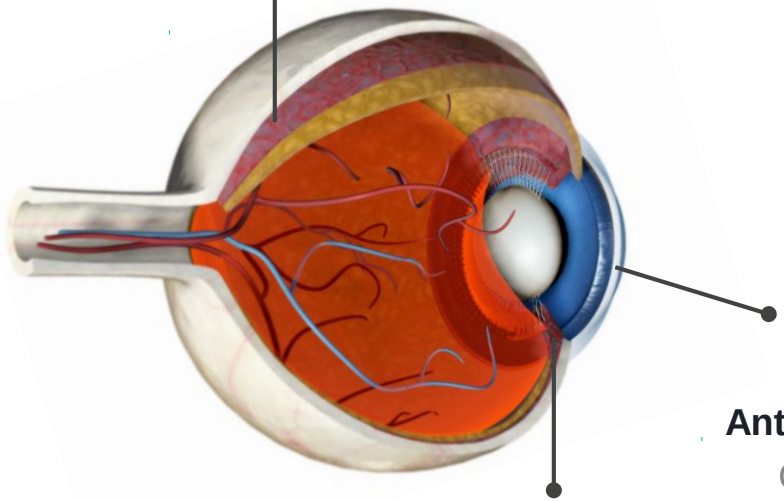
*EDIT-101 (AGN-151587) partnered with Allergan; **Partnered with Celgene; LCA10: Leber congenital amaurosis 10; HSV: herpes simplex virus; CF: cystic fibrosis; DMD: Duchenne muscular dystrophy; AATD: alpha-1 antitrypsin deficiency; AAV: adeno-associated virus

Durable Medicines for Serious Eye Diseases



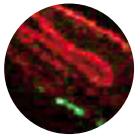
Retina

LEBER CONGENITAL AMAUROSIS
USHER SYNDROME
RETINITIS PIGMENTOSA
Cone-rod dystrophy
Age-related macular degeneration
Stargardt disease



Anterior Chamber
Ocular HSV
Fuchs dystrophy

Trabecular Meshwork
Genetic glaucoma



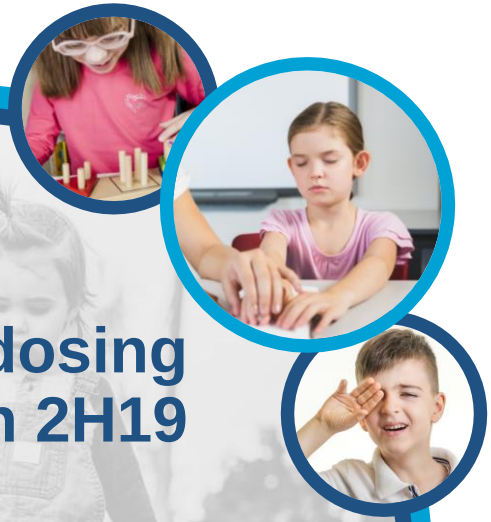
Hundreds of thousands of patients

Targeted local injection with proven AAV vectors

Promising clinical and regulatory path

Targeting Leading Genetic Form of Blindness

Remove genetic mutation to restore CEP290 protein and rebuild photoreceptors in **Leber congenital amaurosis 10**



Degeneration of photoreceptors leading to blindness in childhood

2,000 – 5,000 patients in US and Europe

First patient dosing expected in 2H19

Allergan

and

editas MEDICINE

50/50 in US

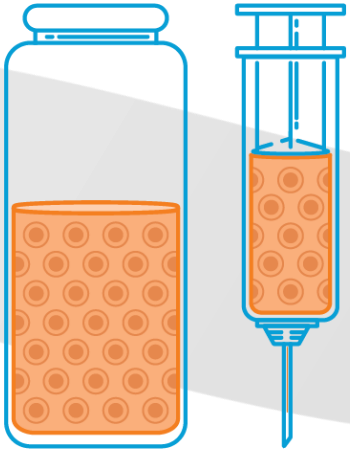
CEP290: centrosomal protein 290

EDIT-101 Aims to Rescue Vision in LCA10

LCA10 Photoreceptor

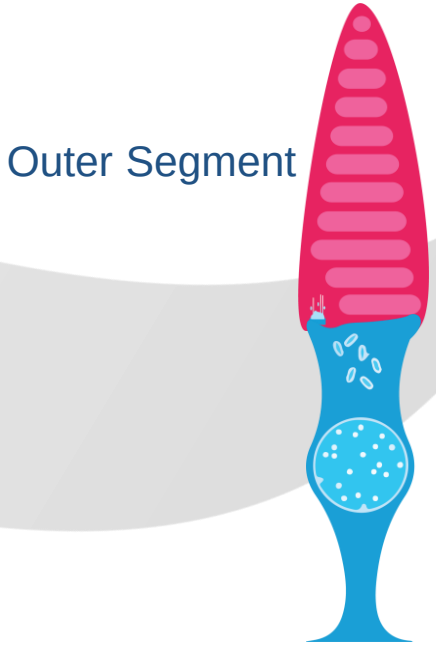
Outer segment degenerates due to CEP290 deficiency

EDIT-101



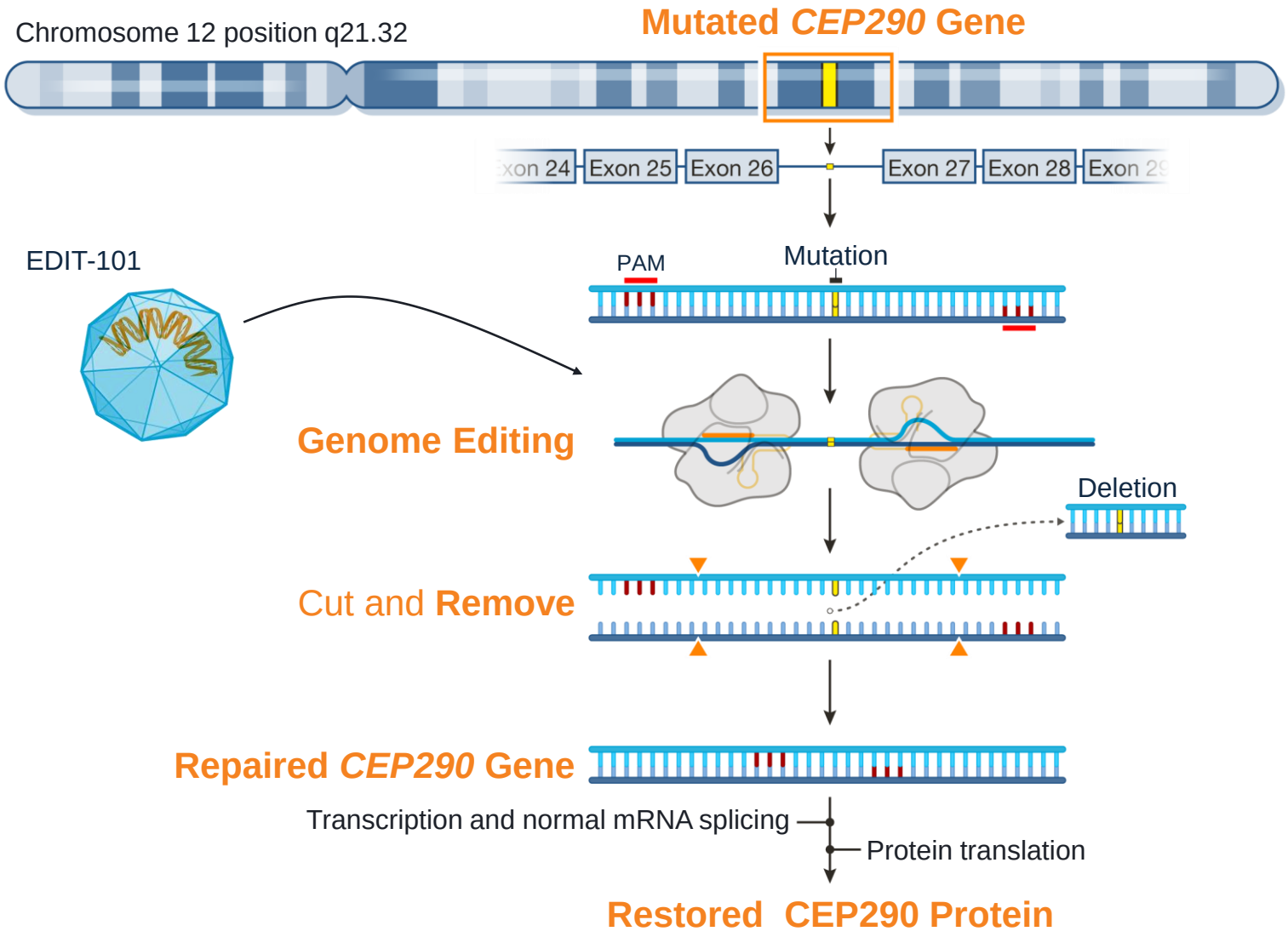
Editing removes disease-causing mutation

Rescued Photoreceptor



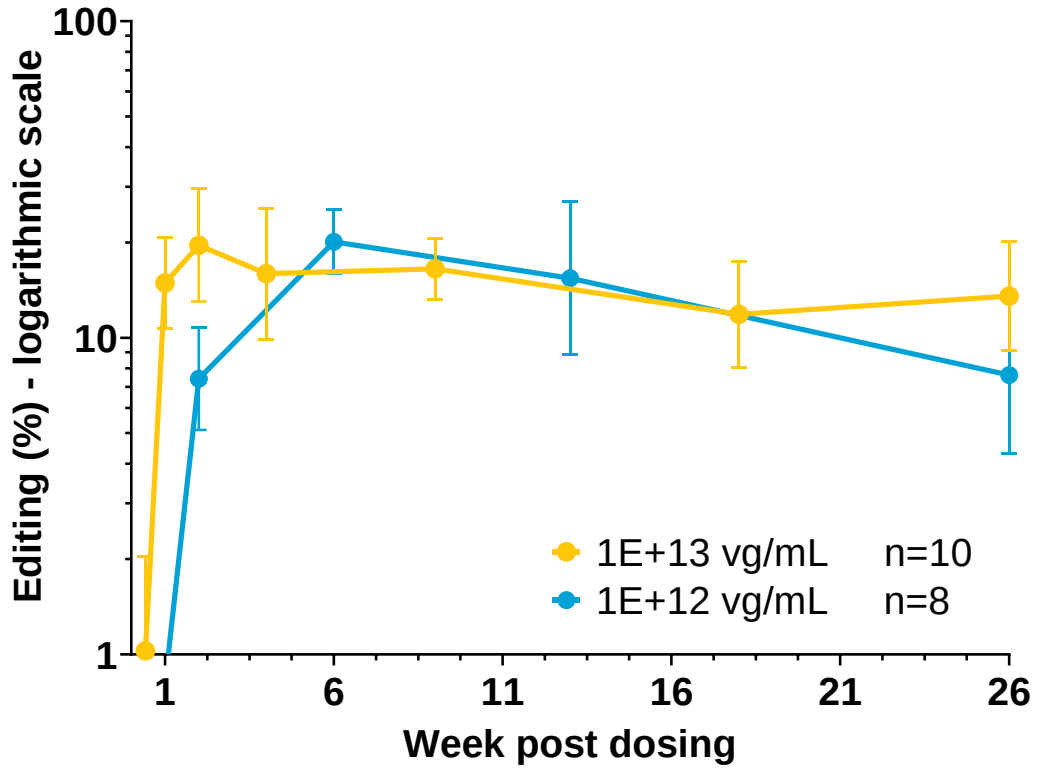
Outer segment regenerates with CEP290 protein

EDIT-101 Aims to Rescue Vision in LCA10



EDIT-101 Demonstrates Rapid Editing

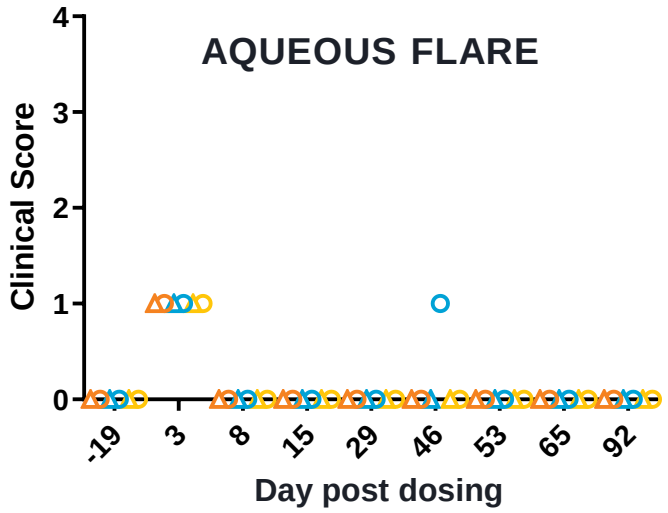
CEP290 GENE EDITING
MOUSE FULL-THICKNESS RETINA

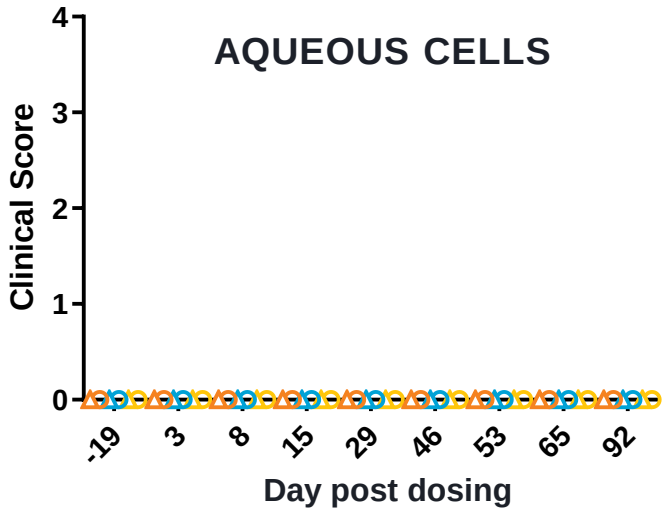


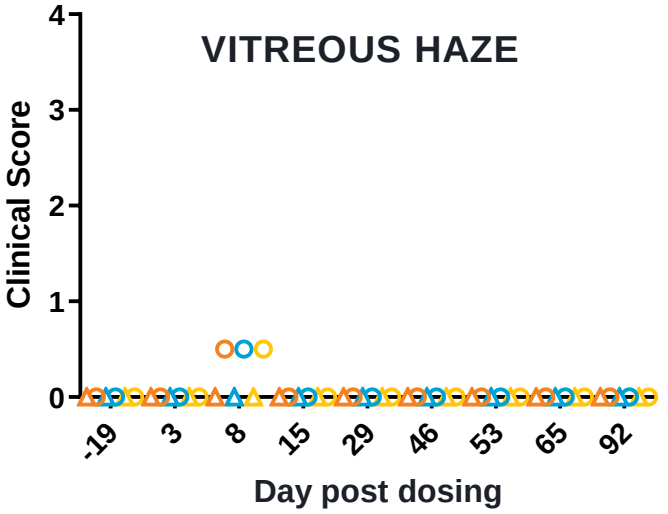
Rapid onset of therapeutically relevant editing at AAV dose that has been safely administered to humans

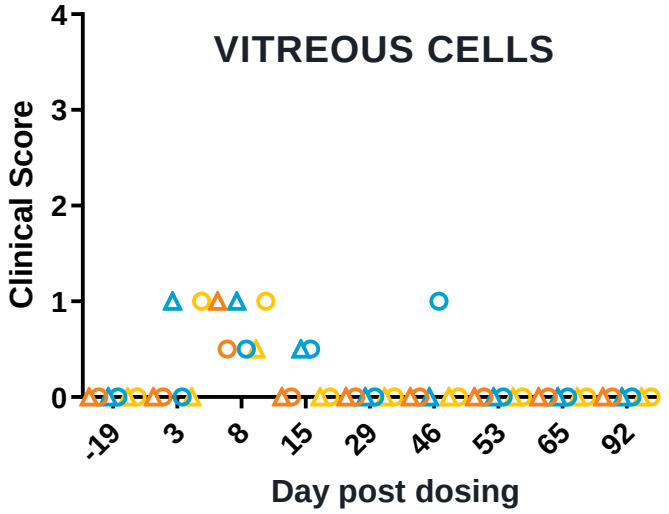
Stefanidakis et al., *Efficient in vivo editing of CEP290 IVS26 by EDIT-101 as a novel therapeutic for the treatment of Leber Congenital Amaurosis 10*, Association for Research in Vision and Ophthalmology 2018 Annual Meeting; AAV: adeno-associated virus

EDIT-101 Well Tolerated in Non-Human Primates









LEGEND

- EDIT-101
- Placebo
- Animal 1
- Animal 2
- Animal 3

CLINICAL SCORE

4.0	Severe
3.0	Marked
2.0	Moderate
1.0	Mild
0.5	Minimal
0	None

4 weeks of steroid prophylaxis; 1E+12 vg/mL x 100 µL/eye

Jiang et al., *Evaluation of Tolerability and Immunogenicity of EDIT-101 Following Subretinal Injection in Non-human Primates*, American Society of Gene & Cell Therapy 21st Annual Meeting

Natural History Study Facilitating Interventional Trial

LCA10 NATURAL HISTORY STUDY

enLIGHTen
Focusing on the future

PATIENTS

~40 patients, aged 3 and above

OBJECTIVES

Characterize patients, assessments, rate of change and validate endpoints

SITES

7 sites across US and Europe

FOLLOW-UP

6 visits over 1 year; baseline data presented in April 2019

Initiated LCA10 Phase 1/2 Clinical Trial

LCA10 PHASE 1/2 TRIAL

brilliance

DESIGN

Open-label, dose escalation study
to evaluate safety, tolerability, and efficacy

PATIENTS

~18 patients with IVS26 mutation*

COMPARATOR

Patient's own baseline value
for each efficacy measure

FOLLOW-UP

Core measurements
every 3 months for 1st year

*Intervening sequence 26 in CEP290 gene containing the c.2991+1655A>G mutation

Pursuing Usher Syndrome 2A Medicine

Rescue vision by restoring USH2A protein leveraging **same proprietary enzyme, vector, and promoter as EDIT-101**

***in vivo* proof of concept demonstrated**

in collaboration with Massachusetts Eye and Ear

Massachusetts Eye and Ear®

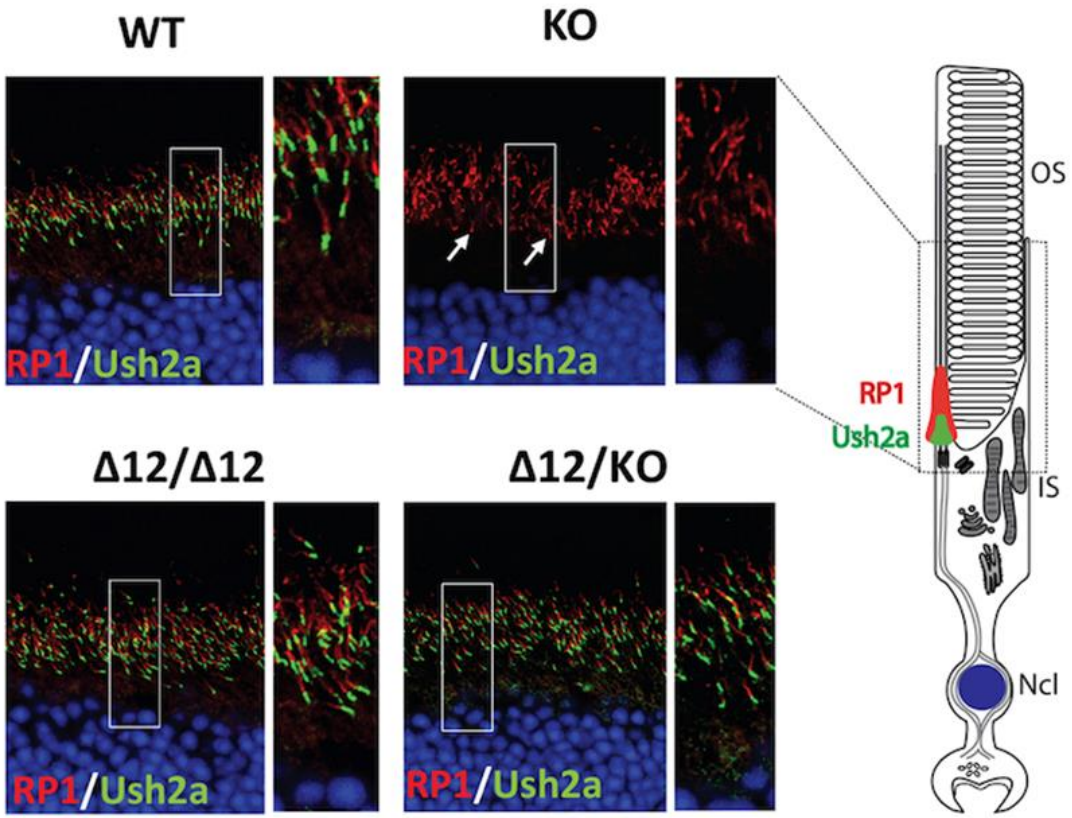
HARVARD MEDICAL SCHOOL AFFILIATE

4,000 patients with target mutation

Additional 10,000 potentially addressable

Progressive vision loss leading to blindness due to degeneration of photoreceptors

Editing *USH2A* Increases Cilia Length *in vivo*

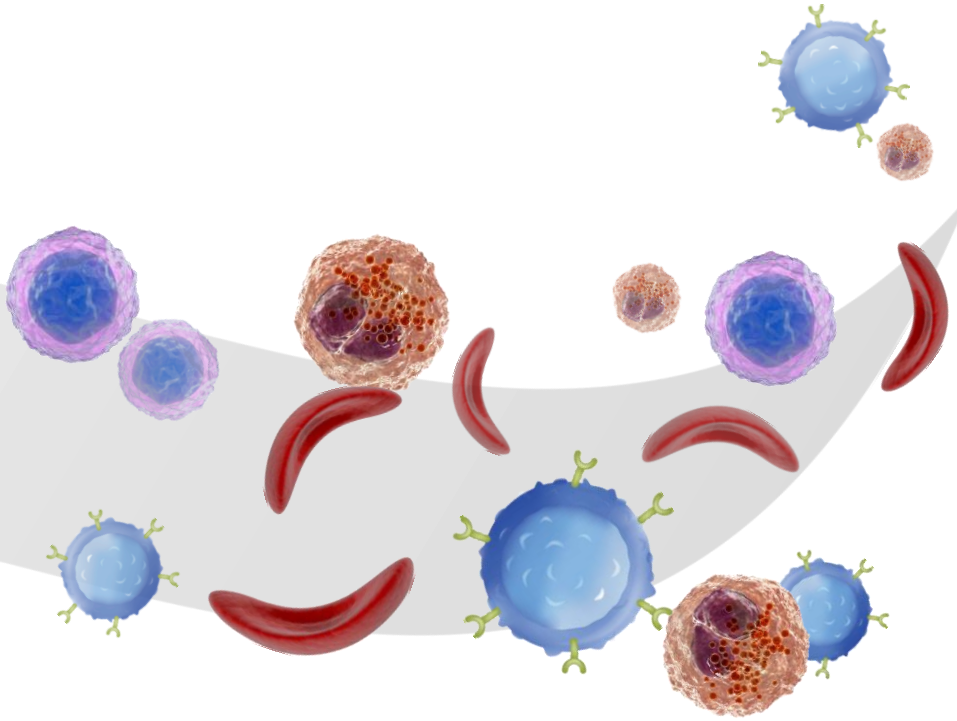


Editing strategy results in desired phenotype

Pendse et al., *CRISPR/Cas Based Evaluation of the Therapeutic Potential for USH2A Associated Diseases*, American Society of Gene & Cell Therapy 22nd Annual Meeting

Developing Transformative Engineered Cell Medicines

Hematopoietic stem cells
could yield **medicines for multiple blood diseases**



T cells and NK cells
are therapeutic platform for **cancer, autoimmune, and infectious diseases**

Editas Medicine editing
enables both **autologous and allogeneic cell medicines**

NK: natural killer

Towards Durable Sickle Cell Disease Medicine

Editing hematopoietic stem cells to increase fetal hemoglobin
and alleviate disease morbidity and mortality

Sickle cell disease and beta-thalassemia
causing anemia, pain crises, organ failure, and mortality

Over
100,000 hospitalizations annually
in US alone

Differentiated editing strategy
designed to deliver
best-in-class medicines

Harnessing Genetics to Treat Sickle Cell Disease

Three Critical Product Candidate Criteria

1

SUCCESSFUL EDITING
of long-term hematopoietic stem cells

2

MAINTENANCE
of normal HSC function

3

DURABLE
predicted therapeutic induction of fetal hemoglobin

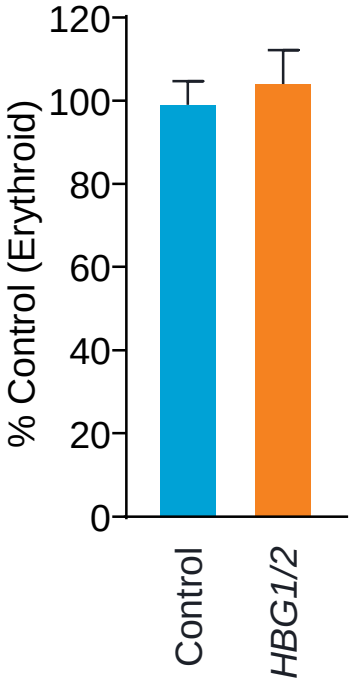
Differentiated editing strategy directly targets the genetic cause of the disease

HSC: hematopoietic stem cell

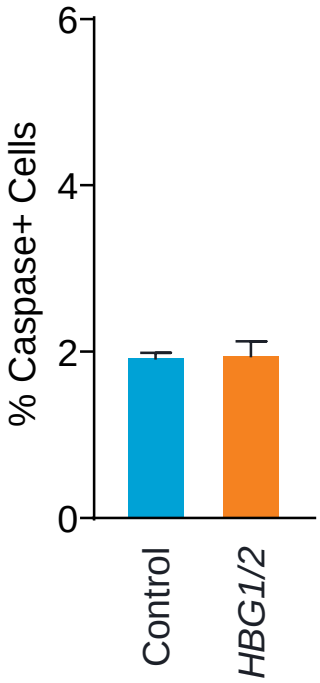
Editing Strategy Maintains HSC Function

GLOBIN LOCUS EDITING
EDITAS MEDICINE APPROACH

SIMILAR LEVELS OF ERYTHROID OUTPUT

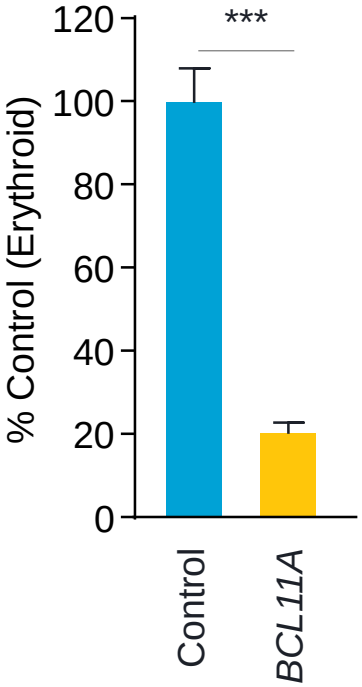


SIMILAR LEVELS OF CELL DEATH

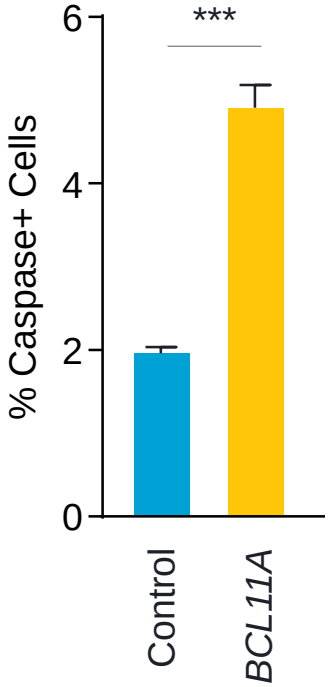


BCL11Ae EDITING
COMPETING APPROACH

REDUCED ERYTHROID OUTPUT



INCREASED CELL DEATH

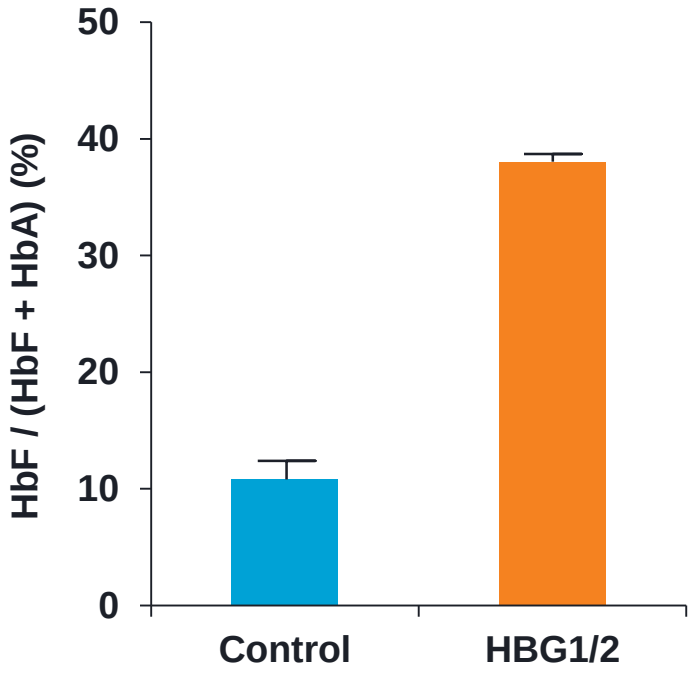


n = 5 healthy human donors; ***p<0.001

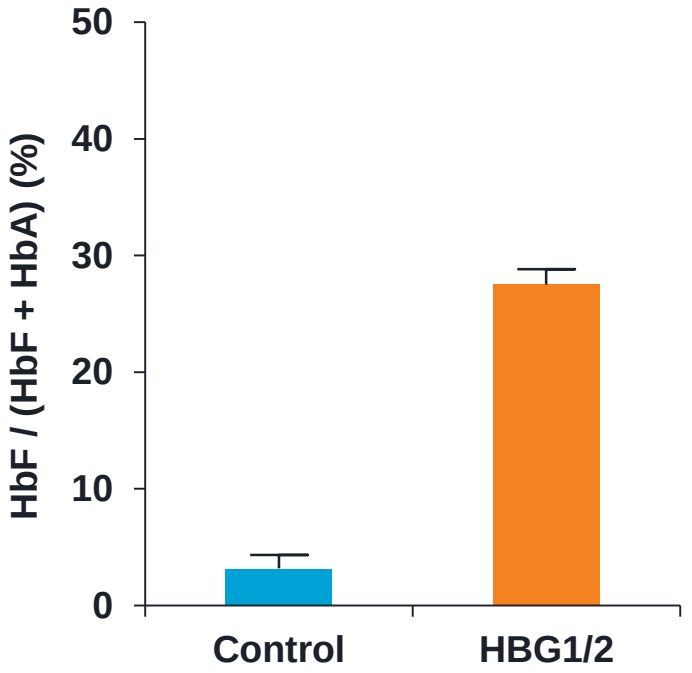
Chang et al., *Genome Editing of HBG1/2 Promoter Leads to Robust HbF Induction In Vivo While Editing of BCL11A Erythroid Enhancer Shows Erythroid Defect*, 60th ASH Annual Meeting & Exposition

Editing Strategy Induces Robust Fetal Hemoglobin

HbF INDUCTION *ex vivo*
CD34+ CELLS



HbF INDUCTION *in vivo*
RED BLOOD CELL PRECURSORS



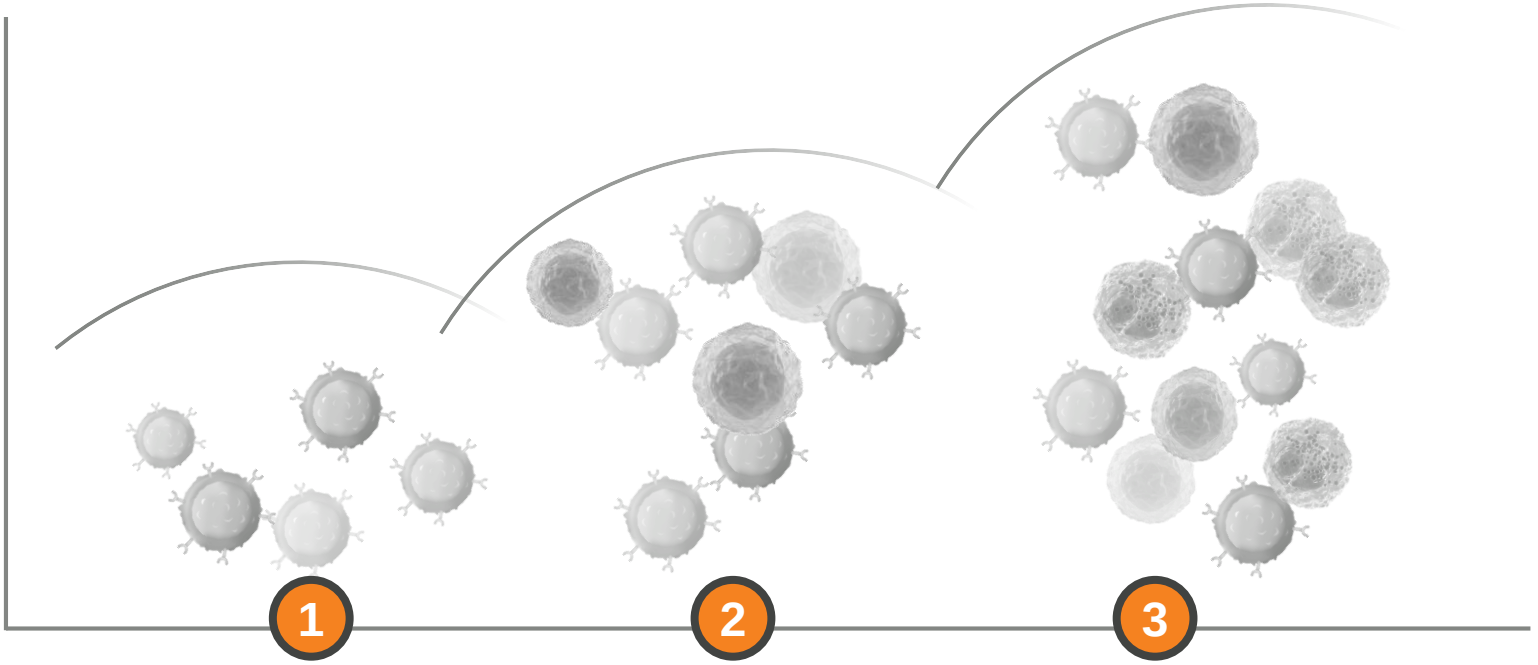
Potential to safely and effectively induce fetal hemoglobin *in vivo*

HbF: fetal hemoglobin; HbA: adult hemoglobin

Editing Strategy Achieves Product Candidate Criteria

	1 SUCCESSFUL EDITING of long-term hematopoietic stem cells	2 MAINTENANCE of normal HSC function	3 DURABLE predicted therapeutic induction of fetal hemoglobin
BCL11Ae	✓	✗	
GLOBIN LOCUS	✓	✓	✓

Rapid Innovation in Cell Medicines for Cancer



CELL SOURCE	Patient Donor	Healthy Donor	Universal (iPSC)
CELL TYPE	αβ T cells	NK cells, αβ T cells, γδ T cells	NK cells, αβ T cells, γδ T cells
MULTIPLEXING	Modest	Modest	High

iPSC: induced pluripotent stem cell

Investing to Develop Best in Class Medicines

	PATIENT DONOR	HEALTHY DONOR	UNIVERSAL (iPSC)
T CELLS (αβ and γδ)	Celgene	Celgene	Celgene
Milestones & royalties to	editas MEDICINE	editas MEDICINE	editas MEDICINE
NK CELLS	editas MEDICINE	editas MEDICINE	editas MEDICINE Milestones & royalties to BlueRock Therapeutics

Driving programs across both T cells and NK cells for best in class medicines to treat liquid and solid tumors

Powerful Engine for Genomic Medicines

Broadest Access to Genomic Targets

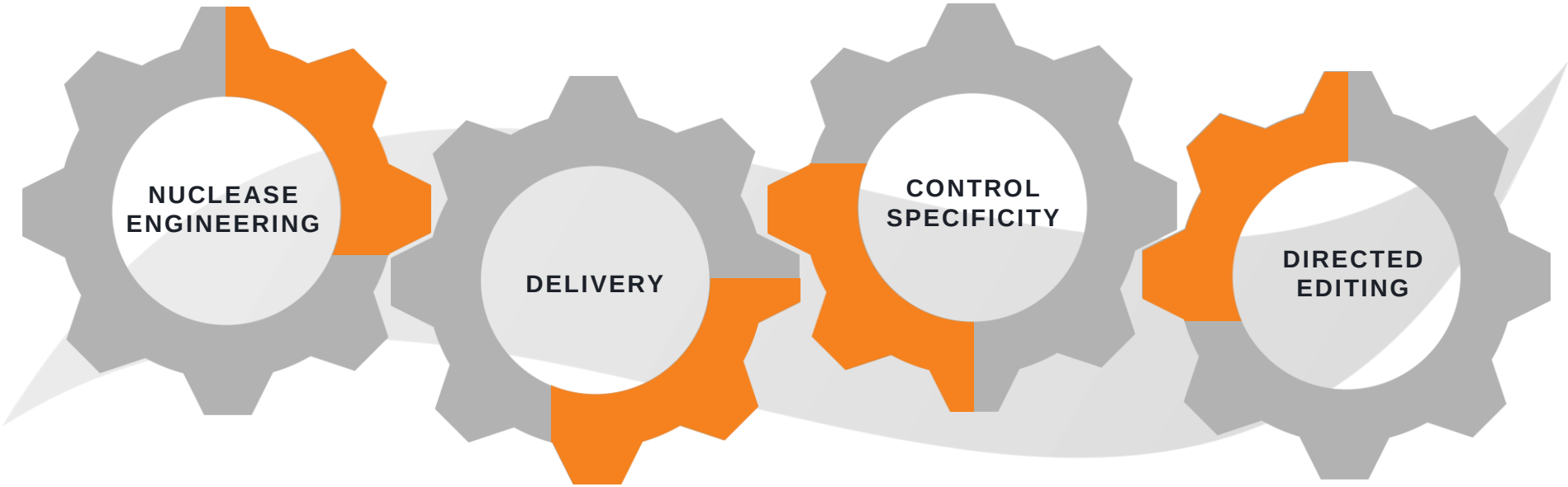
Proprietary portfolio of Cas9 and Cpf1 enzymes may directly edit ~95% of the human genome

Widest Range of Tissues and Cells

Ability to use AAVs, RNPs, and LNPs to address diseases throughout the body

Diverse Spectrum of Therapeutic Edits

Disrupt, remove, replace, or insert DNA to precisely and durably treat illness



Strong Foundation for Long-Term Leadership

PARTNERS

OCULAR MEDICINES

Allergan

Option to license up to 5 ocular programs

Partnership with innovator in ophthalmology

$90 million upfront plus > $1 billion contingent milestones and tiered royalties; option for 50/50 profit split in US on 2 programs, including EDIT-101

ENGINEERED T CELL MEDICINES FOR CANCER

Celgene

CAR T and TCR cell medicines to treat cancer

Partnership with leader in oncology

$30 million upfront and up to $22 million in R&D funding; ~$930 million in contingent milestones plus tiered royalties; $10 million in milestones achieved to date

PATENTS

BROADEST & DEEPEST PORTFOLIO OF CRISPR IP

Exclusive access to foundational Cas9 and Cpf1 patent estates

Exclusive access to multiple species and engineered forms of Cas9 and Cpf1

Over 70 issued patents and over 600 patent applications pending

Issued patents covering EDIT-101

Allergan terms per March 2017 agreement; Juno terms per May 2015 agreement as restated and amended in May 2018

Community

Resilience

Ingenuity

Science

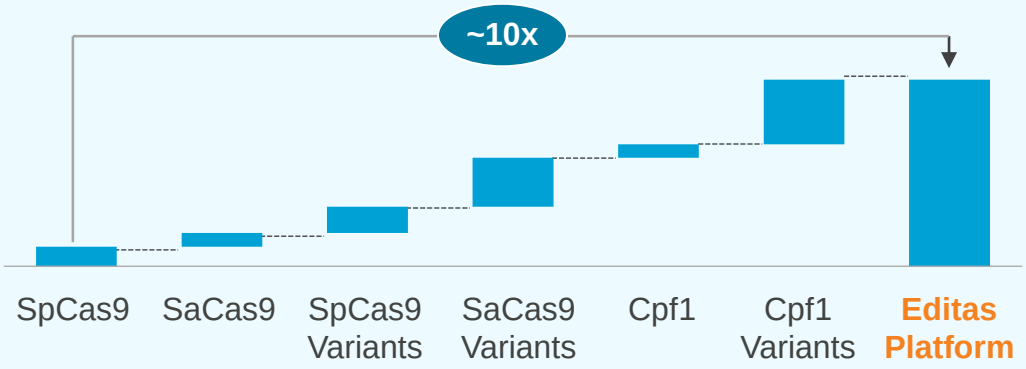
Passion

Revolution

Appendix

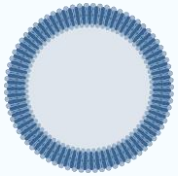
Platform Enables Broad Product Pipeline

Broad Range of Sites



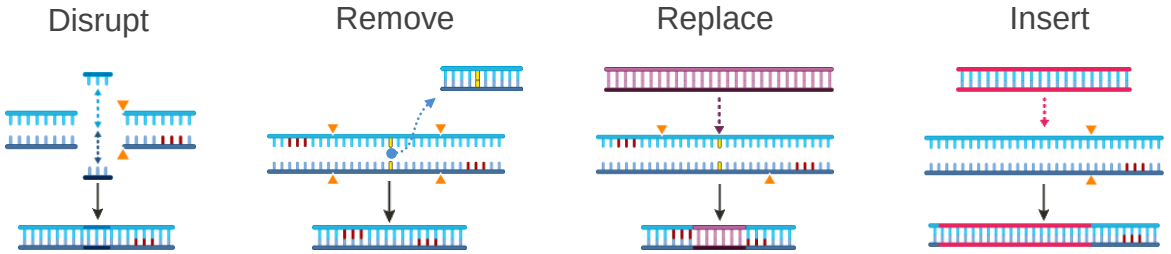
Wide Delivery Options


Viral Vector


Lipid Nanoparticle


Electroporation

Diverse Spectrum of Edits



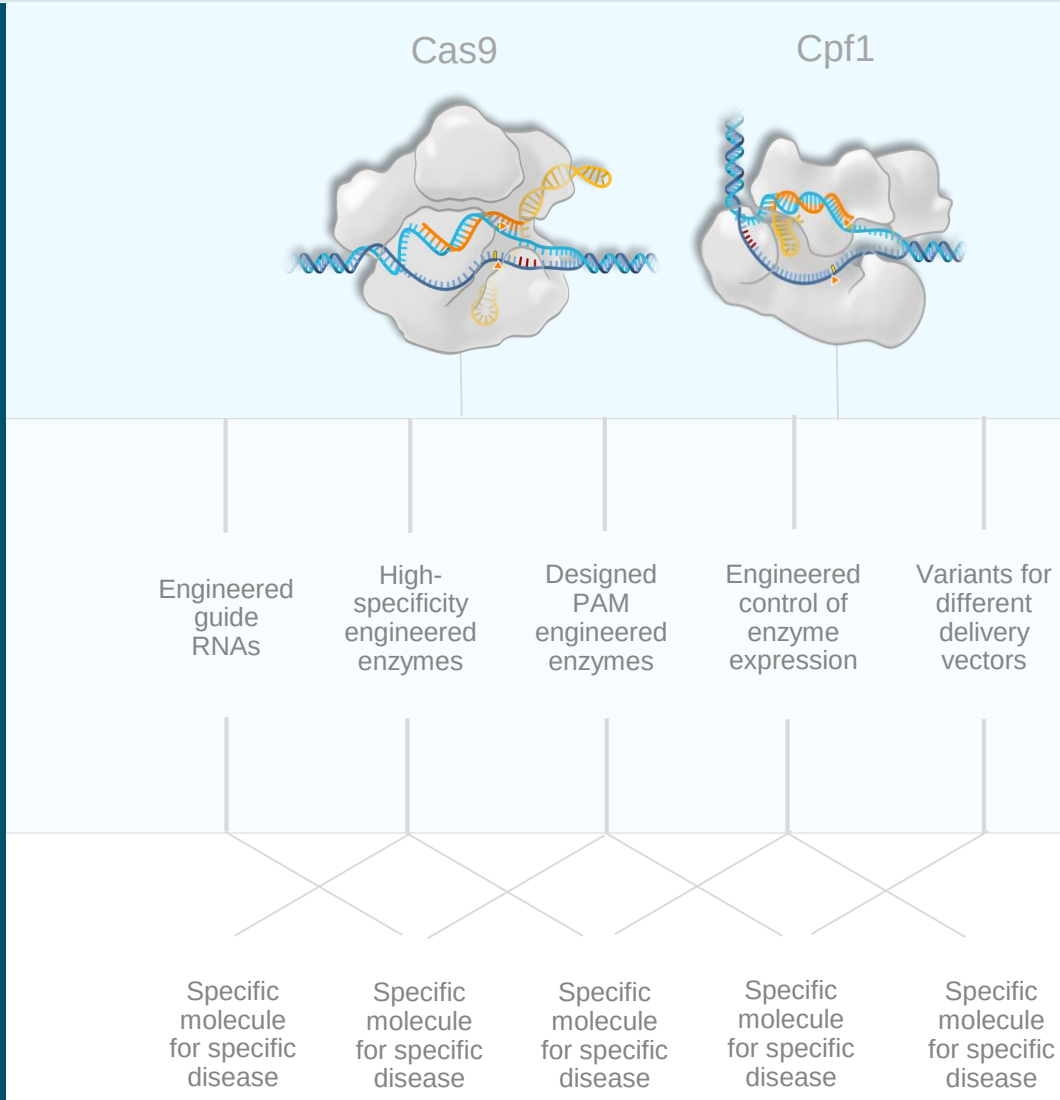
Unmatched Patent Position in CRISPR Gene Editing

Exclusive access to Cas9 and Cpf1
patent portfolios, which are independent of each other

Exclusive access to advanced forms
including high specificity, PAM variants, others

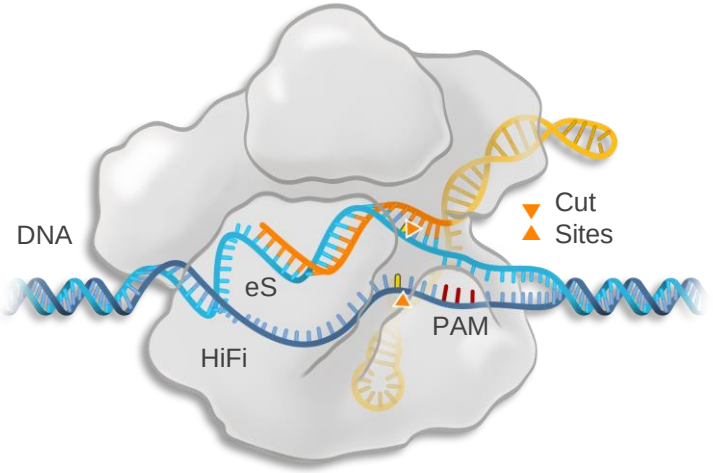
Over 70 issued patents
worldwide, including in United States, Europe, and Australia

Over 600 pending patent applications
from Editas Medicine and academic institutions

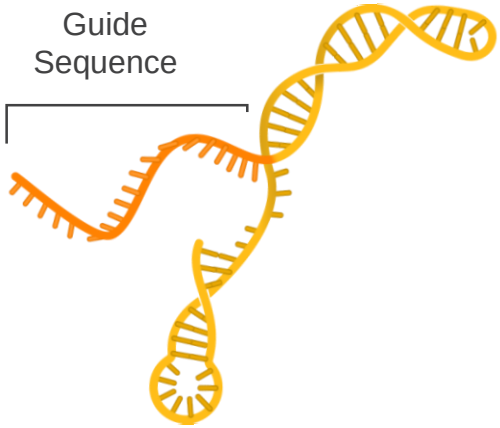


CRISPR Unlocks Genome Editing

Nuclease



Guide RNA



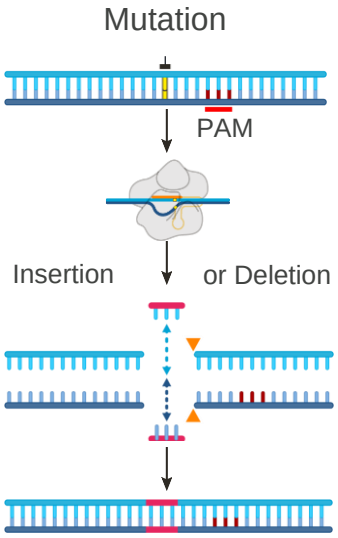
Complex of nuclease and guide RNA precisely locates and cuts genomic sites

Ability to target multiple sites simultaneously

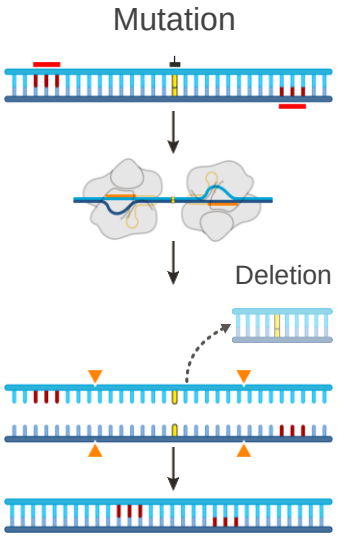
Nuclease can be engineered to reach more sites and to modulate cutting

CRISPR Addresses Diverse Mutations

Cut and
Disrupt



Cut and
Remove



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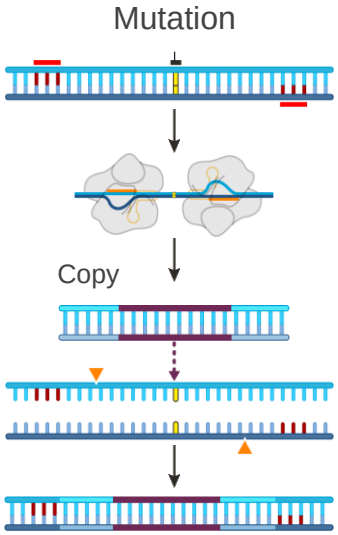
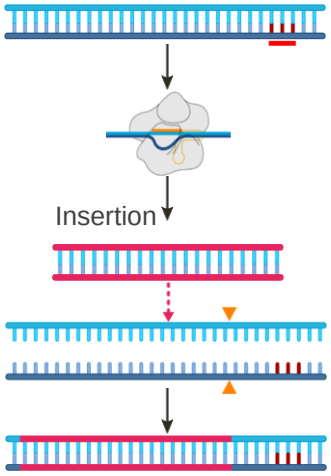
Replace



Cut and
Insert



Non-homologous end joining typically
disrupts a gene or eliminates a disease-causing mutation

Homology-directed repair and targeted insertion aim to
promote expression of correct DNA sequences