Investor Presentation

October 9, 2018

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 Company's goals of submitting of an IND for the LCA10 program in October 2018, the Company's 2022 goals, achieving preclinical proof-of-concept for additional programs and establishing alliances. 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.

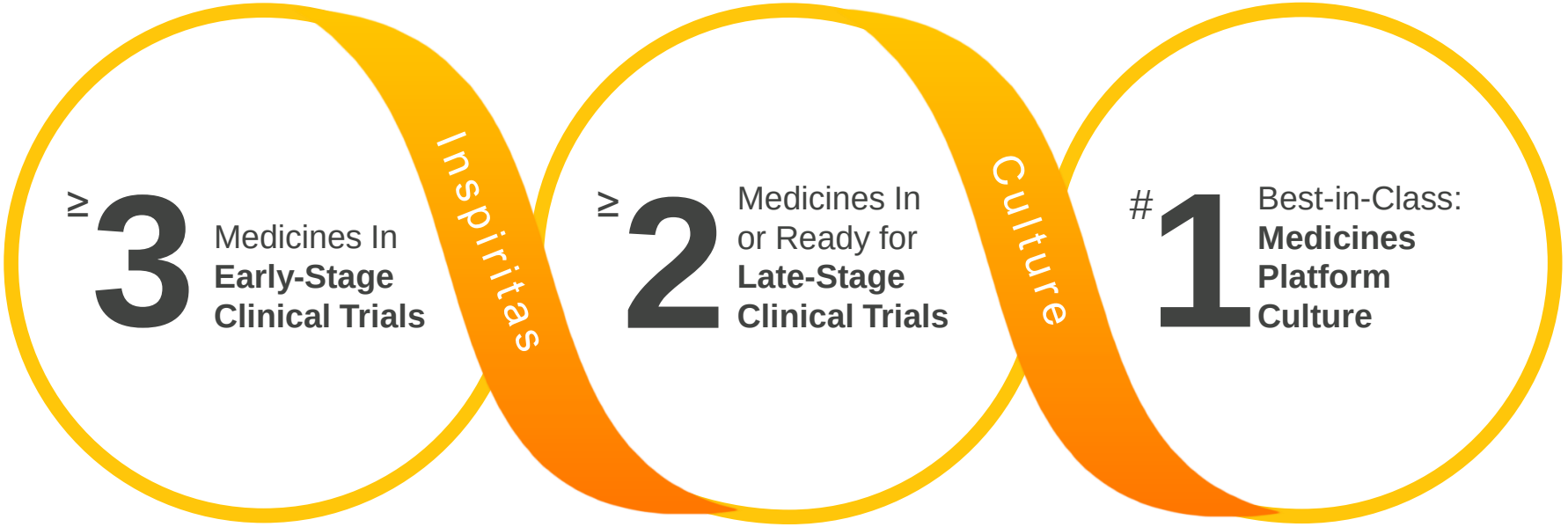
Gene Editing Unleashing Wave of Genomic Medicines

Convergence of technologies for advanced medicines

Gene editing expands and accelerates the universe of genomic medicines

Building the Preeminent Genomic Medicine Company

Editas Medicine 2022 Goals – EM22



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

Unparalleled Platform for Genomic Medicines

Broadest Access to Genomic Targets

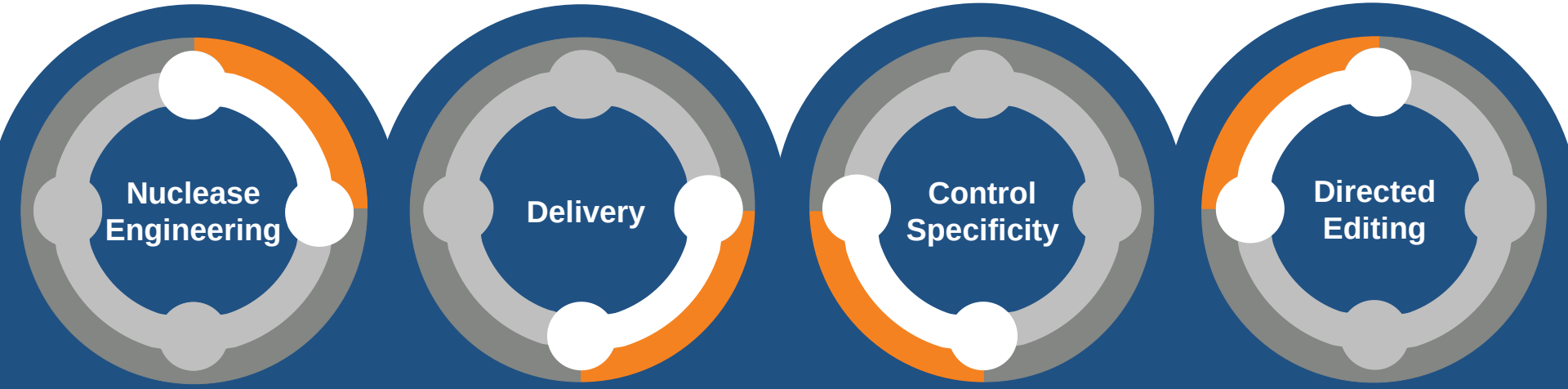
Proprietary portfolio of Cas9 and Cpf1 enzymes may directly edit ~95% of the human genome

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



AAV: Adeno-associated Virus; RNP: Ribonucleoprotein; LNP: Lipid Nanoparticle

Developing Best-in-Class CRISPR Medicines

Ocular Medicines

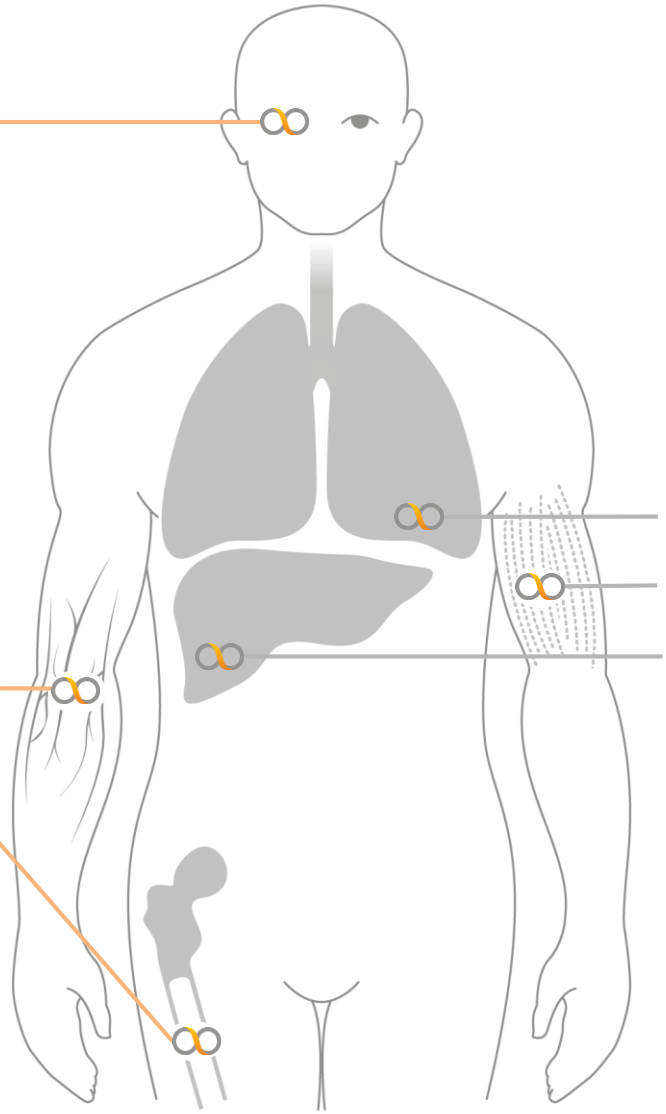
Inherited Retinal Diseases
- LCA10 (EDIT-101)*
- USH2A
- Additional unnamed targets

Infectious Diseases
- Ocular HSV

Engineered Cell Medicines

Immune Cells
- T Cells – Cancer**
- T Cells – Autoimmune diseases

Stem Cells
- HSCs – Sickle Cell Disease
- HSCs – Beta-thalassemia



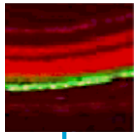
Early Discovery
- Lung – CF
- Muscle – DMD
- Liver – AATD

in vitro proof-of-concept

in vivo proof-of-concept

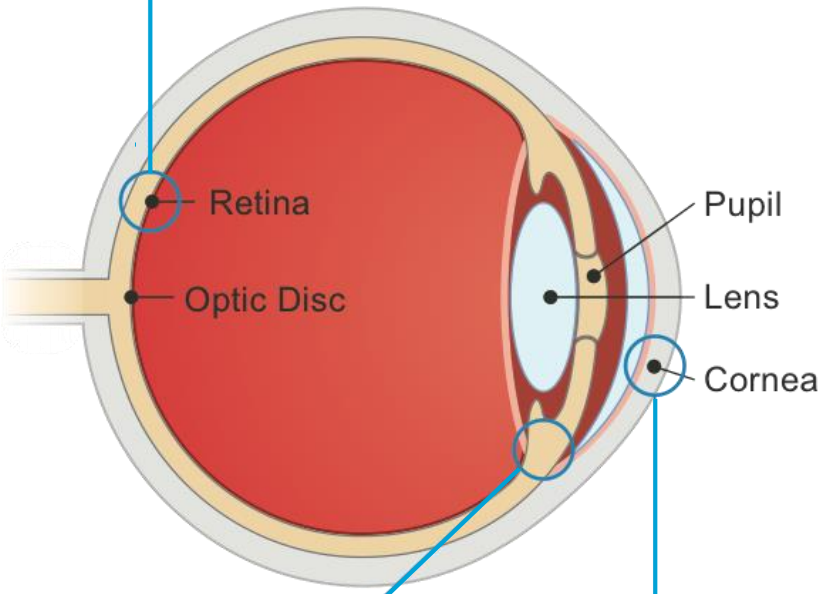
*Partnered with Allergan – US 50/50 plus milestones and ex-US royalties; **Partnered with Celgene – global milestones and royalties; LCA10: Leber Congenital Amaurosis Type 10; USH2A: Usher Syndrome Type 2A; HSV: Herpes Simplex Virus; CF: Cystic Fibrosis; DMD: Duchenne Muscular Dystrophy; AATD: Alpha-1 Antitrypsin Deficiency; HSC: Hematopoietic Stem Cell

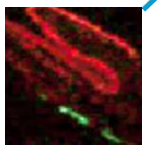
Durable Medicines for Serious Eye Diseases



Retina

Leber Congenital Amaurosis
Usher Syndrome
Cone-rod Dystrophy
Age-related Macular Degeneration
Stargardt Disease
Retinitis Pigmentosa





Trabecular Meshwork
Glaucoma

Anterior Chamber
Ocular HSV
Fuchs Dystrophy

Hundreds of thousands of patients may benefit from durable CRISPR medicines addressing ocular diseases

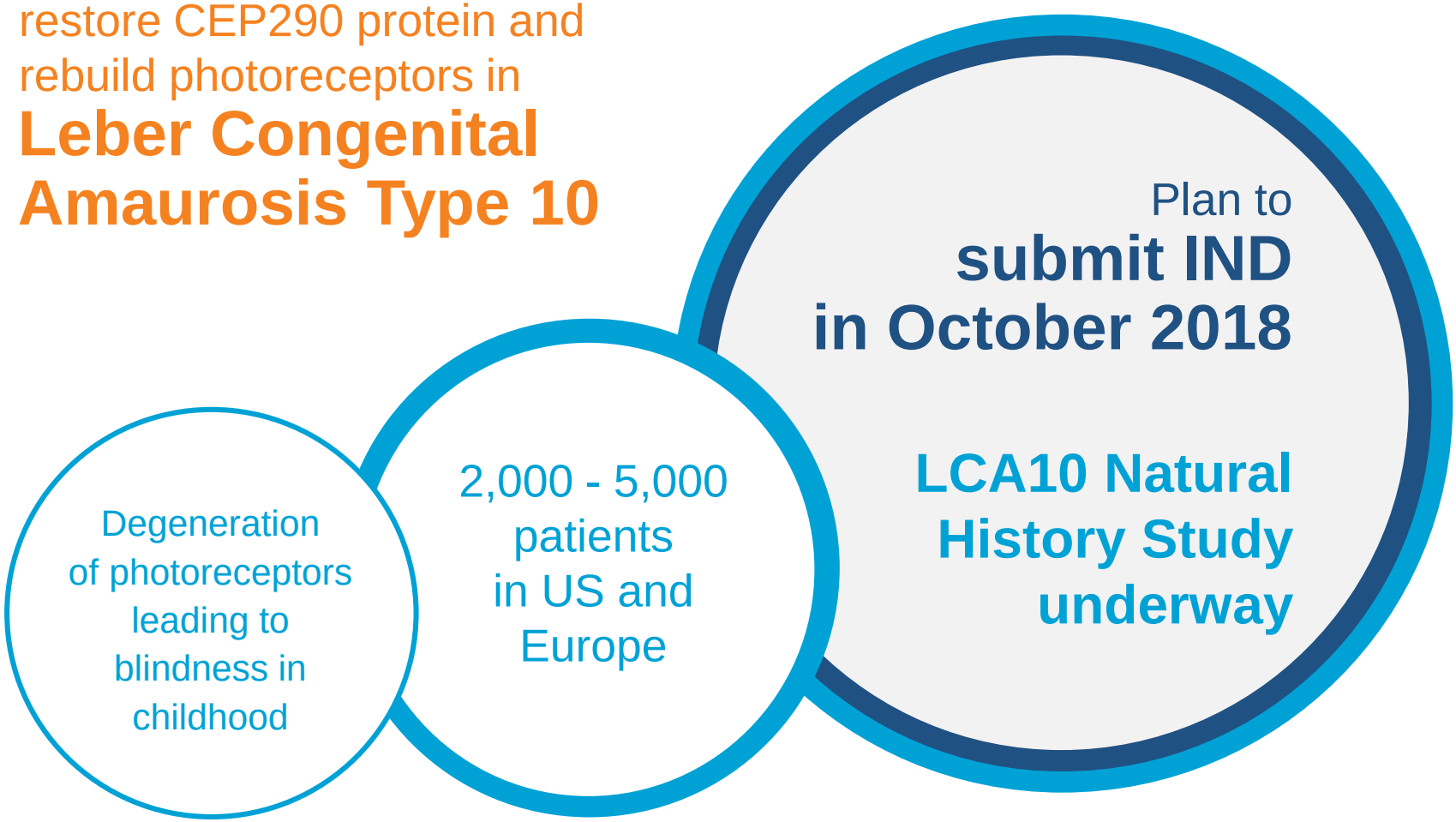
Targeted local injection using proven viral vectors enables precise delivery to multiple compartments of the eye

Promising clinical and regulatory path with readily measurable endpoints and serious unmet need

Targeting Leading Genetic Form of Blindness

Remove genetic mutation to restore CEP290 protein and rebuild photoreceptors in

Leber Congenital Amaurosis Type 10



EDIT-101 Aims to Rescue Vision in LCA10

LCA10 Photoreceptor

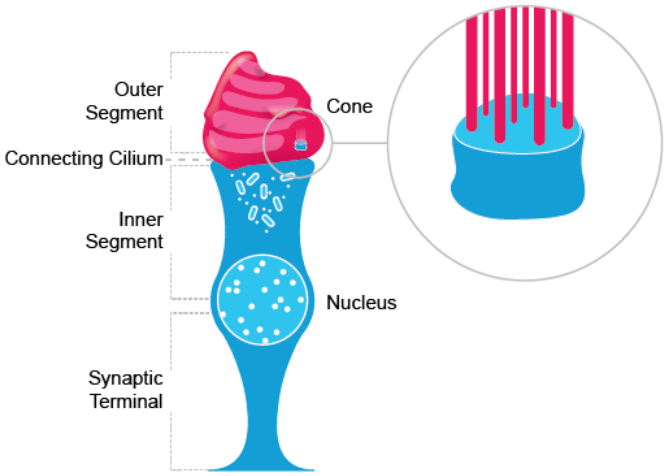
Degenerates because CEP290 lacking

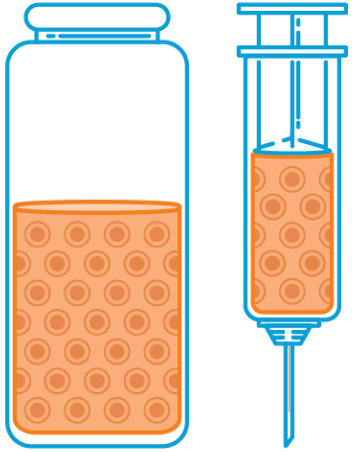
EDIT-101

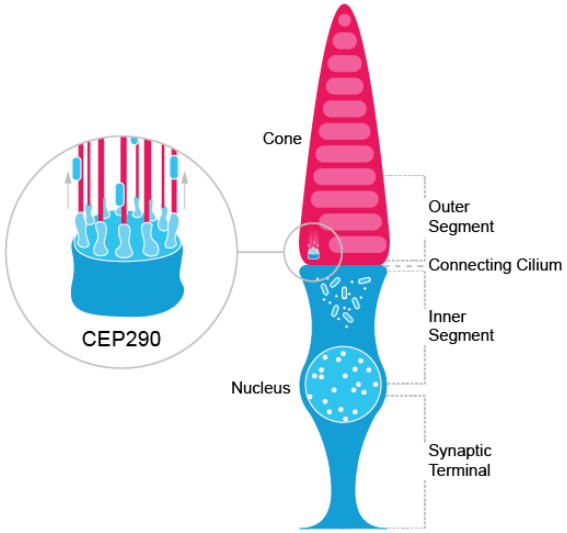
Removes disease-causing mutation

Rescued Photoreceptor

By correcting CEP290 protein







Degeneration of outer segment but cell body remains intact

EDIT-101 subretinal injection to remove disease-causing mutation

Restoration of full-length protein and rebuilding of outer segment

CEP290: Centrosomal Protein 290

From Gene to Genomic Medicine for LCA10

Critical Achievements Advancing EDIT-101 to Human Clinical Trials

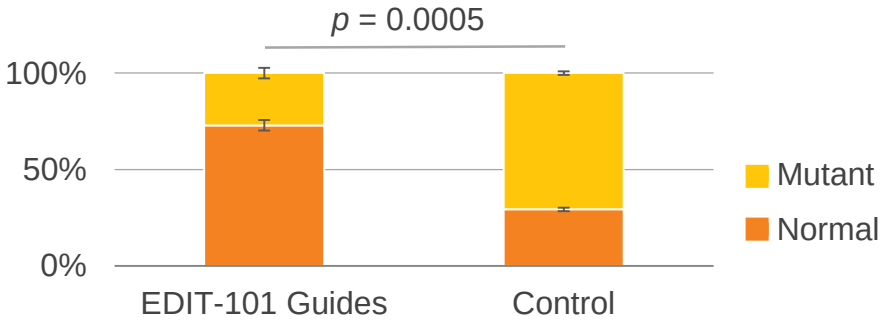
1. DOES EDITING RESTORE PROTEIN EXPRESSION IN PATIENT CELLS?
2. CAN WE EDIT TARGET CELLS IN BEST PRECLINICAL MODEL ANIMAL?
3. DOES PRODUCT CANDIDATE ACHIEVE THERAPEUTIC EDITING IN HUMAN TISSUE?
4. DOES PRODUCT CANDIDATE HAVE SPECIFICITY FOR HUMAN TESTING?
5. WHAT ARE BEST CLINICAL TRIALS TO PROVE VALUE FOR PATIENTS?

EDIT-101 Advancing Towards Clinical Trials

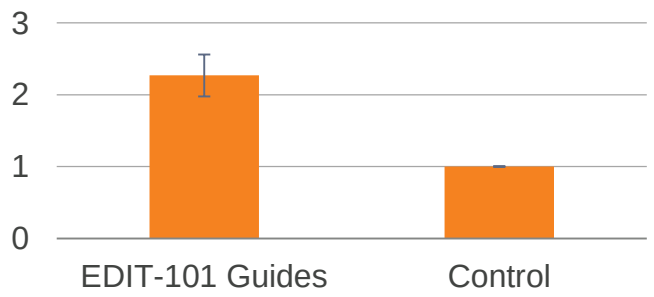
1 **EDITING APPROACH RESTORES FULL LENGTH CEP290 mRNA AND PROTEIN**

Demonstrated in cells from LCA10 patients

Relative Level of CEP290 mRNA



CEP290 Protein Normalized to Control



Deleting the disease-causing mutation **corrects full-length mRNA for CEP290**

Deleting the disease-causing mutation **restores full-length CEP290 protein**

mRNA: Messenger RNA

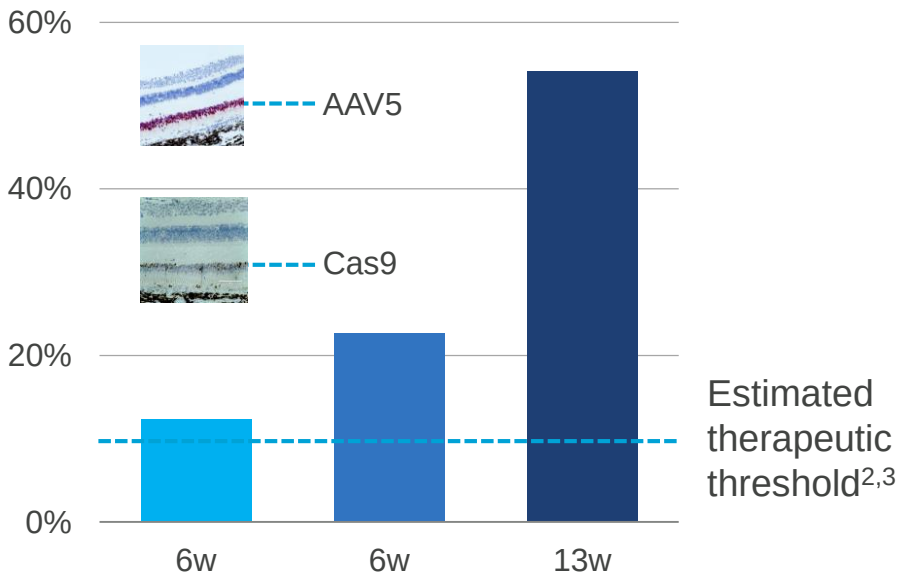
EDIT-101 Advancing Towards Clinical Trials

2 **PREDICTED THERAPEUTIC EDITING ACHIEVED IN NON-HUMAN PRIMATES**

Estimated productive editing in primate photoreceptors *in vivo*[1]

Delivery vehicle specifically targets photoreceptors

Estimated Productive Editing
Non-human Primate Photoreceptors



AAV5 vector and GRK1 promoter limit expression to photoreceptors, providing a **highly targeted therapy**

Productive editing with subretinal delivery in anatomically relevant animal model **well above therapeutic threshold**

1. Editing measured across entire retina multiplied by 3.5 based on photoreceptors estimated to represent 25-30% of retina; 2. Geller, Sieving, and Green, *J. Opt. Soc. Am.*, 1992; 3. Geller and Sieving, *Vision Res.*, 1993; Guide RNAs in NHP experiments specific to NHP genome; NHP: Non-human Primate; GRK1: G Protein-Coupled Receptor Kinase 1

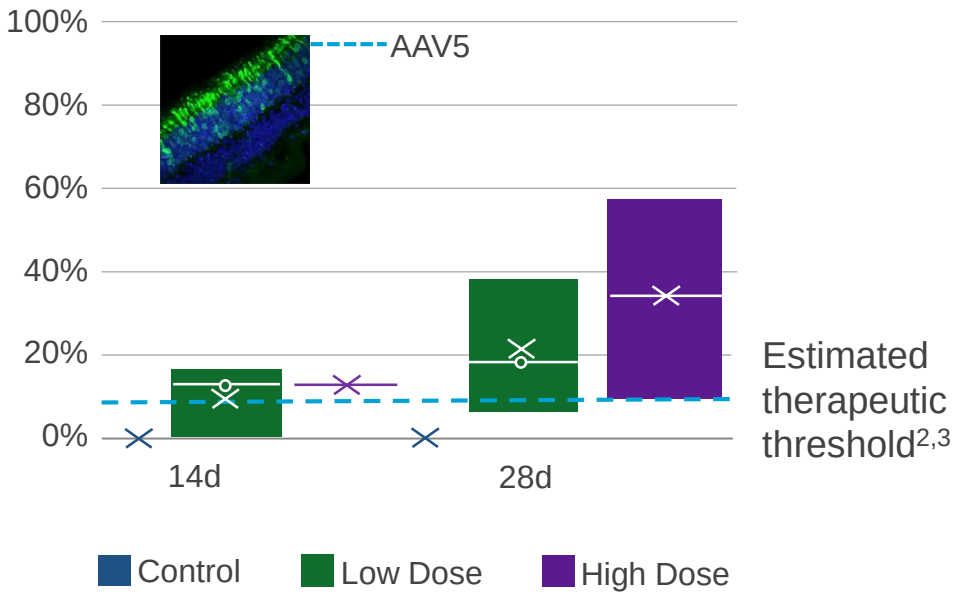
EDIT-101 Advancing Towards Clinical Trials

3 **PREDICTED THERAPEUTIC EDITING ACHIEVED IN HUMAN RETINA**

Productive editing in human retinal explant photoreceptors[1]

Targeted transduction of photoreceptors

Estimated Productive Editing
Human Retinal Explant Photoreceptors



AAV5 vector
selectively targets human photoreceptor cells

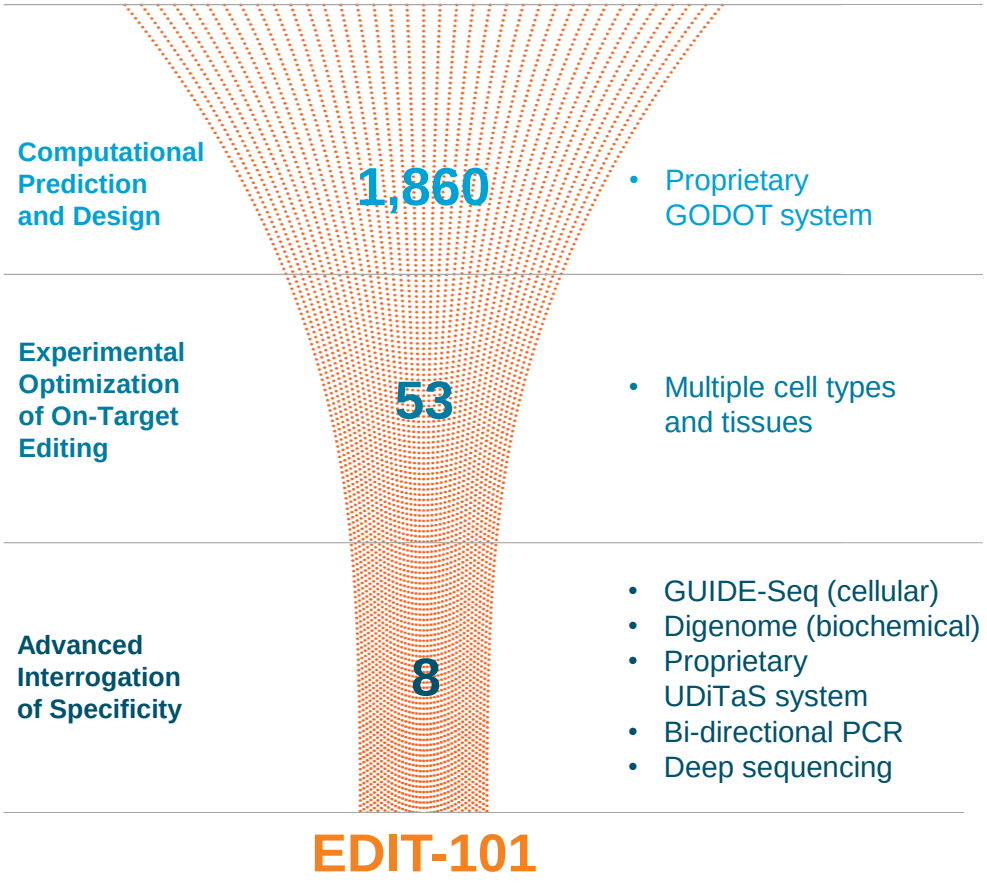
Product candidate
EDIT-101 achieves predicted therapeutic levels of editing in **human photoreceptors**

1. Editing measured across entire retina multiplied by 3.5 based on photoreceptors estimated to represent 25-30% of retina; 2. Geller, Sieving, and Green, *J. Opt. Soc. Am.*, 1992; 3. Geller and Sieving, *Vision Res.*, 1993.

EDIT-101 Advancing Towards Clinical Trials

4 **COMPREHENSIVE METHODS TO IDENTIFY EFFICIENT AND SPECIFIC GUIDE RNAs**

Proprietary computational, biochemical, and cellular approaches



Systematic approach to guide RNA characterization
using a suite of comprehensive, empirical, and unbiased methods

Identified and selected product candidates with
no detected off-targets
verified in cells and tissues

GODOT: gRNA Design and Off-Target finder; UDiTaS: Uni-directional Targeted Sequencing; PCR: Polymerase Chain Reaction

EDIT-101 Advancing Towards Clinical Trials

5 SETTING THE STAGE FOR INTERVENTIONAL TRIALS

Ongoing Natural History Study

Patients	~40 patients, aged 3 and above
Objectives	Characterize patients, assessments, and rate of change and validate endpoints
Sites	6 to 8 sites in US and Europe
Follow-up	6 visits over 1 year

EDIT-101 Advancing Towards Clinical Trials

PHASE 1/2 TRIAL DESIGN IN DEVELOPMENT

Design	Open-label, dose escalation
Patients	~10 to 20 patients with IVS26 mutation
Comparator	Non-randomized comparison to natural history, contralateral eye, and patient baseline
Duration	1 year evaluation of efficacy and safety

IVS26 mutation: Intervening Sequence 26 in CEP290 gene containing the c.2991+1655A>G mutation

Pursuing Usher Syndrome Type 2A Medicine

Rescue vision by
restoring USH2A protein using
similar product construct and delivery to EDIT-101

Progressive vision loss leading to blindness due to degeneration of photoreceptors

4,000 patients with target mutation

Additional 10,000 potentially addressable

Collaboration
with Drs. Eric Pierce and Qin Liu
to validate gene editing approach
in transgenic mouse model

Massachusetts Eye and Ear®

HARVARD MEDICAL SCHOOL AFFILIATE

Preventing Blindness from Ocular HSV

Knock out critical viral genes
to disable the latent virus

Recurrent stromal ocular herpes simplex virus leading to corneal scarring and blindness

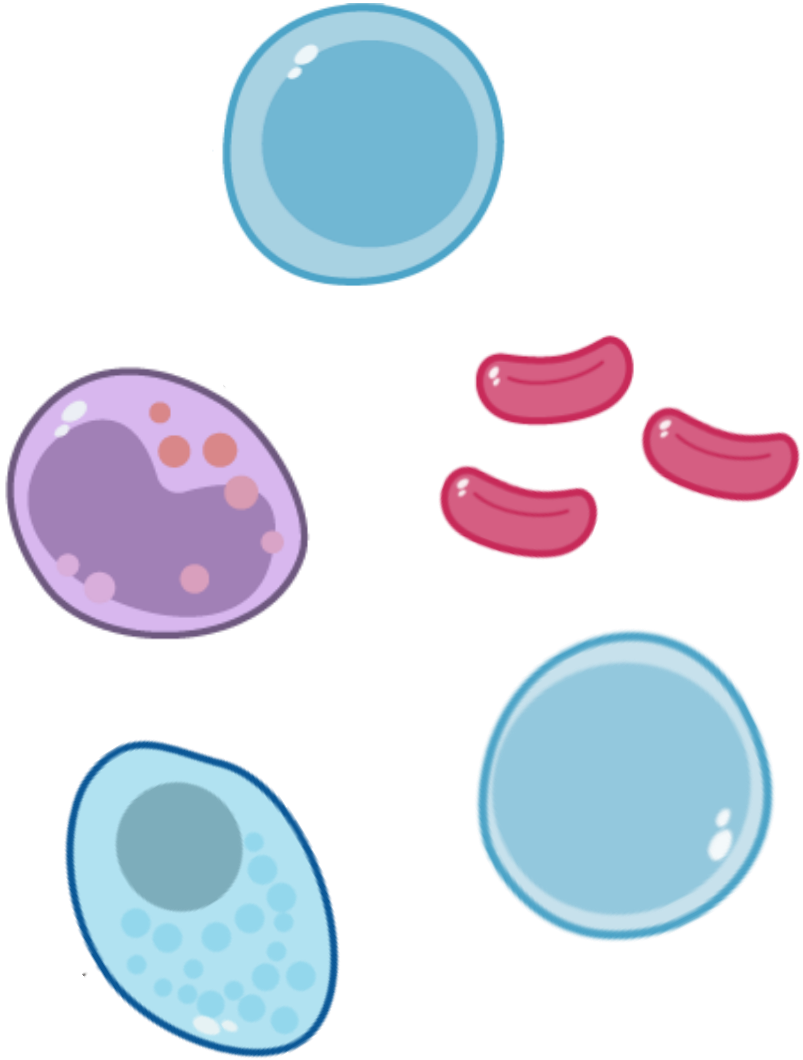
25,000 per year in developed economies

135,000 globally

***in vivo* proof-of-concept in rabbit model**
presented at ARVO 2018 Annual Meeting

ARVO: Association for Research in Vision and Ophthalmology

Developing Transformative Engineered Cell Medicines



Hematopoietic stem cells could yield **multiple medicines for blood diseases** including sickle cell disease and beta-thalassemia

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

Editas editing enables medicines across **many additional cell types**

Next-Gen Engineered T Cells for Cancer

Expand range of cancers that can be treated
with Editas engineered CAR T and TCR cell medicines

Achieved highly efficient editing of multiple gene targets, both individually and in combination

Celgene developing **at-scale gene editing manufacturing** process

Multiple product candidates in alliance advancing including **an engineered TCR candidate for HPV-associated solid tumors**

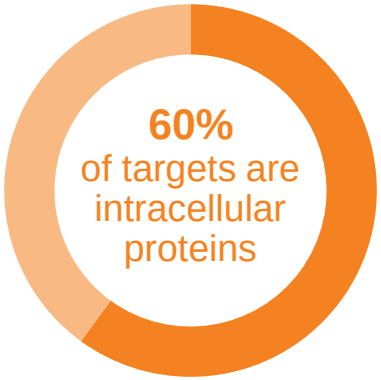
JUNO THERAPEUTICS
A Celgene COMPANY

CAR: Chimeric Antigen Receptor; TCR: T Cell Receptor

Next-Gen Engineered T Cells for Cancer

JUNO THERAPEUTICS A Celgene COMPANY

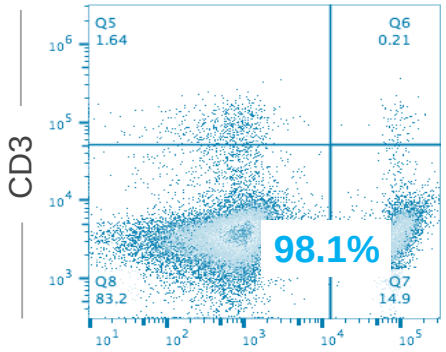
"Top 50" Cancer Antigen Targets[1]



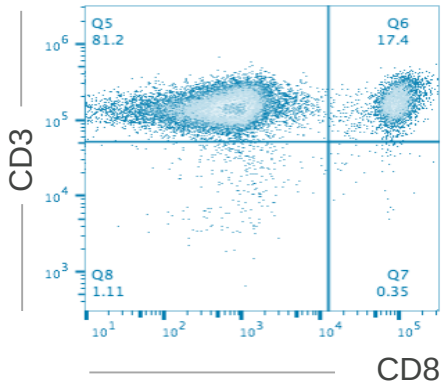
Rank	Antigen	Mechanism
1	WT1	Oncogenic
3	LMP2	Viral
4	HPV	Viral/Oncogenic
8	MAGE A3	Mixed
9	P53 WT	Oncogenic
10	NY-ESO-1	Prognosis
14	MelanA/ MART1	Differentiation
15	Ras Mutant	Oncogenic
16	gp100	Differentiation
17	p53 Mutant	Oncogenic

Nearly Complete TCR Knockout

TCR KO

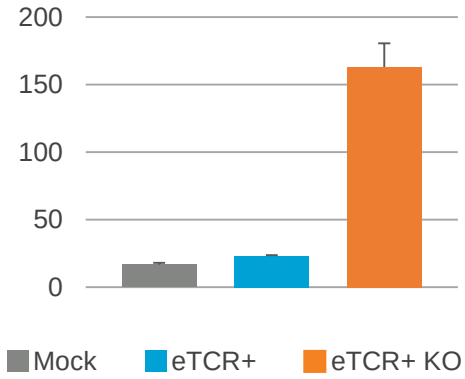


Control

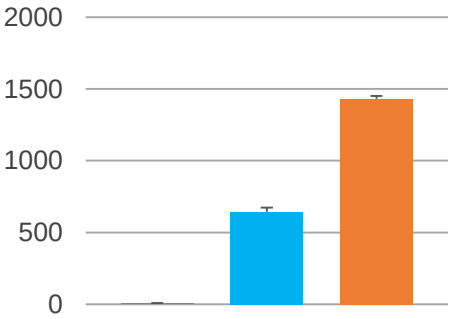


Increase in Functional Activity

CD4 IFNγ (pg/mL)



CD8 IFNγ (pg/mL)



[1]*Clin Cancer Res*. 2009 Sep 1, 15(17); KO: Knockout; eTCR: Engineered T Cell Receptor; IFNγ: Interferon Gamma

Engineered HSCs for Hemoglobinopathies

Gene disruption to increase
fetal hemoglobin levels

Gene insertion to restore
adult hemoglobin expression

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

Over
100,000 hospitalizations annually
in US alone

Candidates from two
distinct editing strategies
designed to deliver
best-in-class medicines

HSC: Hematopoietic Stem Cell

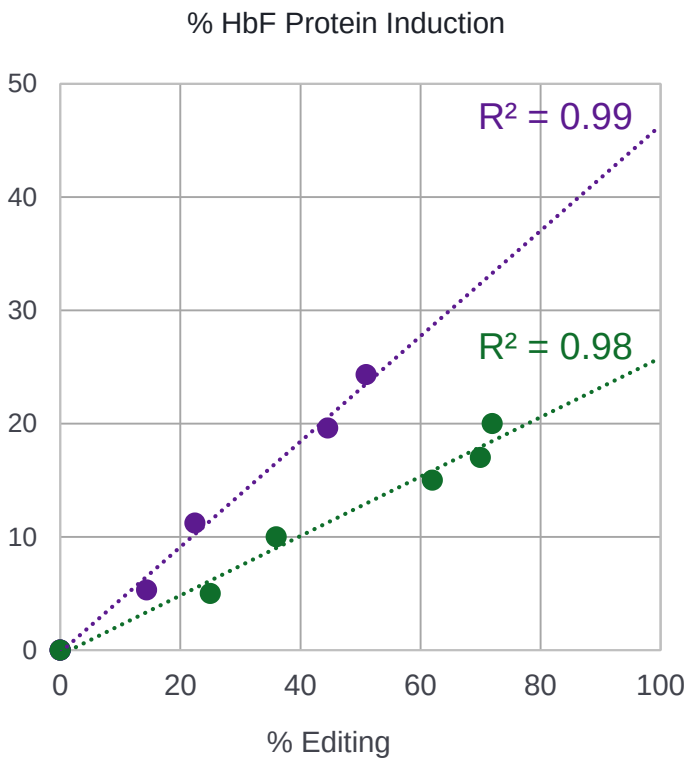
Aim for Best Hemoglobinopathy Medicines

Gene Disruption
to increase fetal hemoglobin with potentially more potent edit

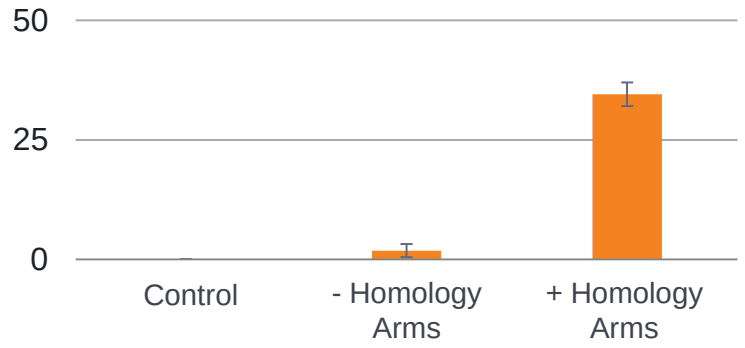
Gene Insertion
to restore hemoglobin expression and eliminate mutation

Editas Novel Approach
to Editing β-globin Locus[1]

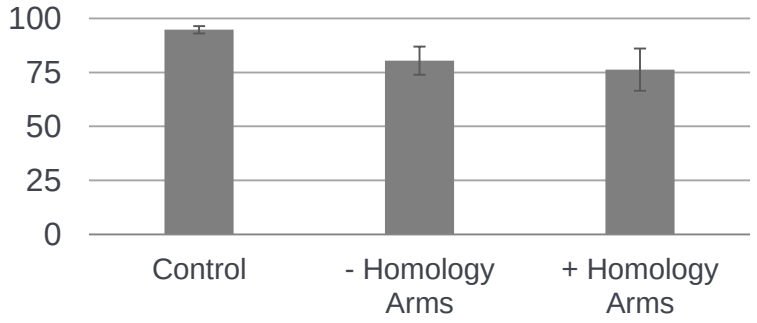
ZFN Published Approach
to Editing BCL11Ae[2]



% Homology Directed Repair at β-globin Locus



% Cells Viable at 48 hours



1. 0, 2.5, 3.75 µM RNP tested, 2 different HSC donors; 2. Data estimated based on Chang et al., *Molecular Therapy*, 2016
HbF: Fetal Hemoglobin; RNP: Ribonucleoprotein

Accelerating the Business through Alliances

MEDICINES

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

ENGINEERED T CELL MEDICINES FOR CANCER

JUNO THERAPEUTICS
A Celgene COMPANY

CAR T and TCR cell medicines to treat cancer

Partnership with leader in engineered T cells for cancer

$30 million upfront and up to $22 million R&D funding plus ~ $930 million milestones and tiered royalties

TECHNOLOGY

ADVERUM BIOTECHNOLOGIES

MONITOR BIOTECHNOLOGIES

CYSTIC FIBROSIS FOUNDATION THERAPEUTICS

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

2017 Sets Stage for Transformative 2018

2017 Accomplishments

- Established Allergan strategic alliance in ocular medicines
- Achieved preclinical proof-of-concept for multiple programs
- Initiated LCA10 clinical natural history study
- Expanded team to >110 Editors
- Further advanced our intellectual property leadership position

2018 Goals

- Submit IND for LCA10 program by mid-2018
- Report preclinical proof-of-concept for additional programs
- Advance manufacturing capabilities to enable additional IND(s) in 2019
- Establish additional important strategic alliances
- Continue to build a best-in-class organization and culture

Repairing broken genes is just the beginning

Community

Resilience

Ingenuity

Science

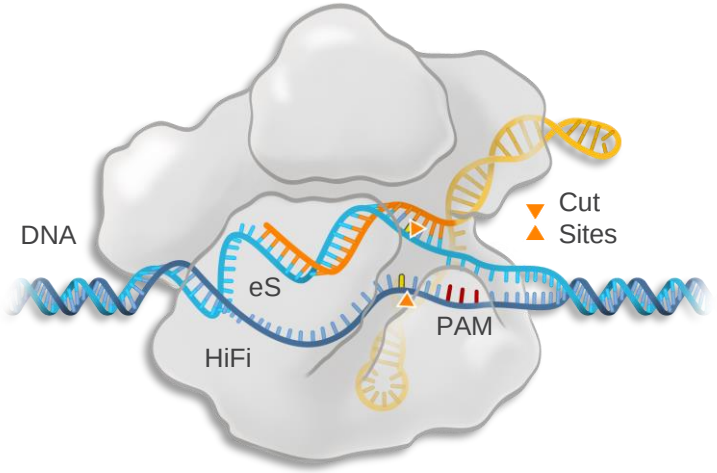
Passion

Revolution

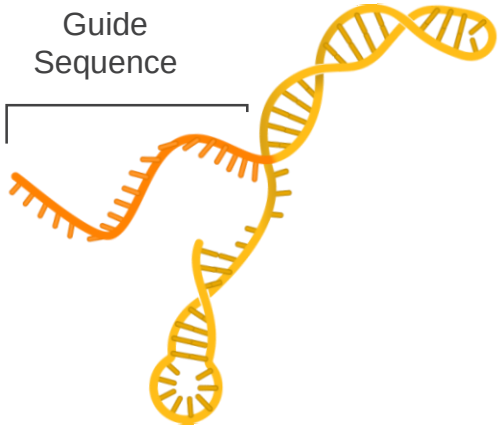
Appendix

CRISPR Unlocks Genome Editing

Nuclease



Guide RNA



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

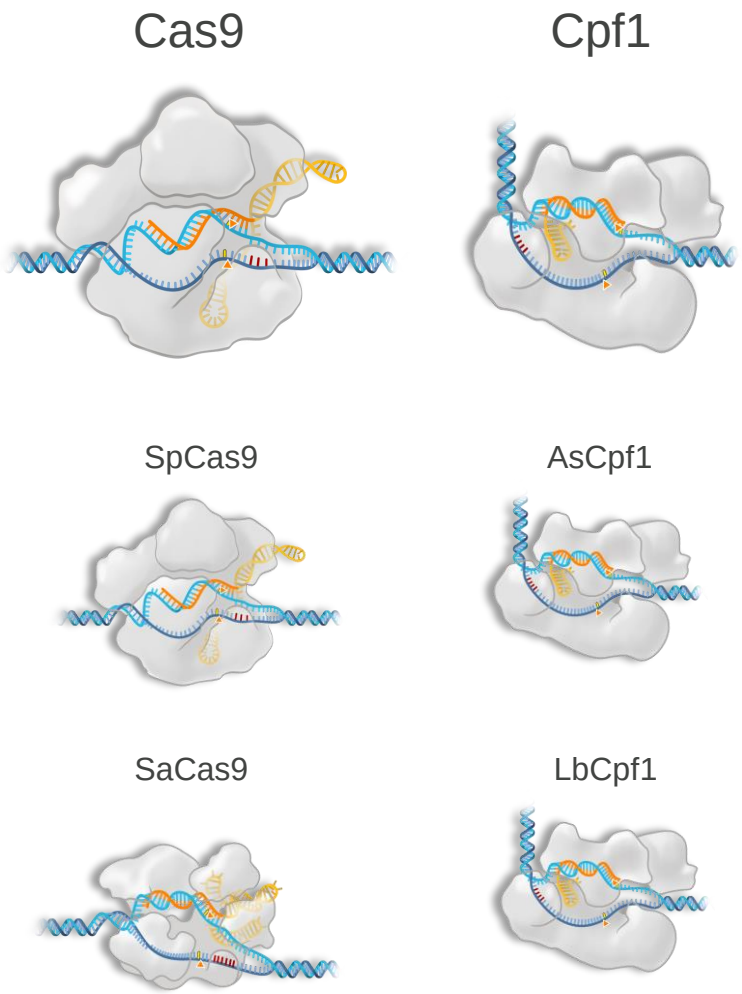
Ability to target multiple sites simultaneously

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

DNA: Deoxyribonucleic Acid; HiFi: High Fidelity; eS: Enhanced Specificity; PAM: Protospacer Adjacent Motif; RNA: Ribonucleic Acid

Broad Toolkit of CRISPR Nucleases

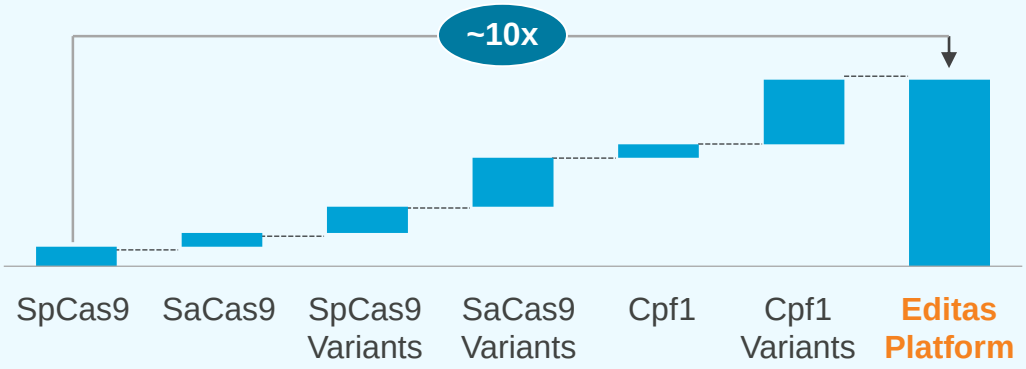
We are the ***only*** company with ***multiple editing systems***



Cas9: CRISPR Associated Protein 9; Cpf1: CRISPR from *Prevotella* and *Francisella*; SpCas9: *Streptococcus pyogenes* Cas9; SaCas9: *Staphylococcus aureus* Cas9; AsCpf1: *Acidaminococcus* species Cpf1; LbCpf1: *Lachnospiraceae* bacterium Cpf1

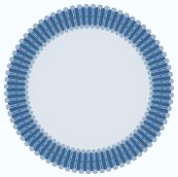
Platform Enables Broad Product Pipeline

Broad Range of Sites



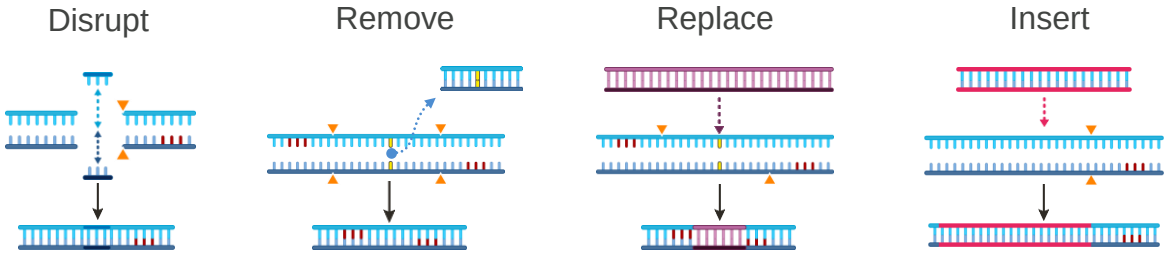
Wide Delivery Options


Viral Vector


Lipid Nanoparticle


Electroporation

Diverse Spectrum of Edits

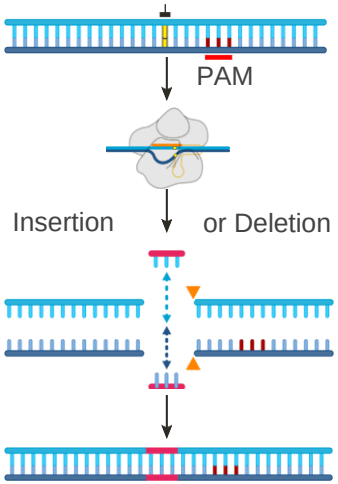


SpCas9: *Streptococcus pyogenes* Cas9; SaCas9: *Staphylococcus aureus* Cas9

CRISPR Addresses Diverse Mutations

Cut and
Disrupt

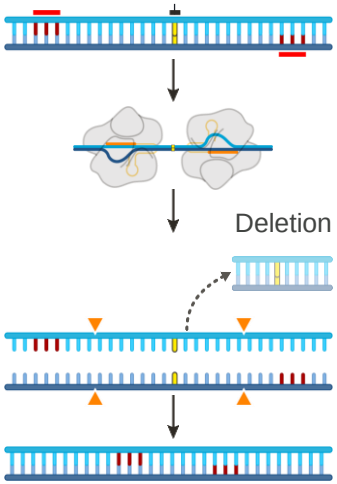
Mutation



e.g., Engineered T cells

Cut and
Remove

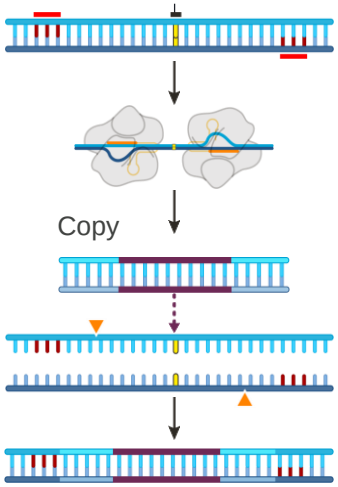
Mutation



e.g., LCA10

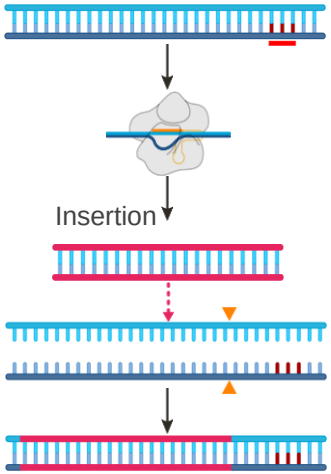
Cut and
Replace

Mutation



e.g., Hemoglobin Beta

Cut and
Insert



e.g., Safe harbor

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

Rigorous Approach to Specificity

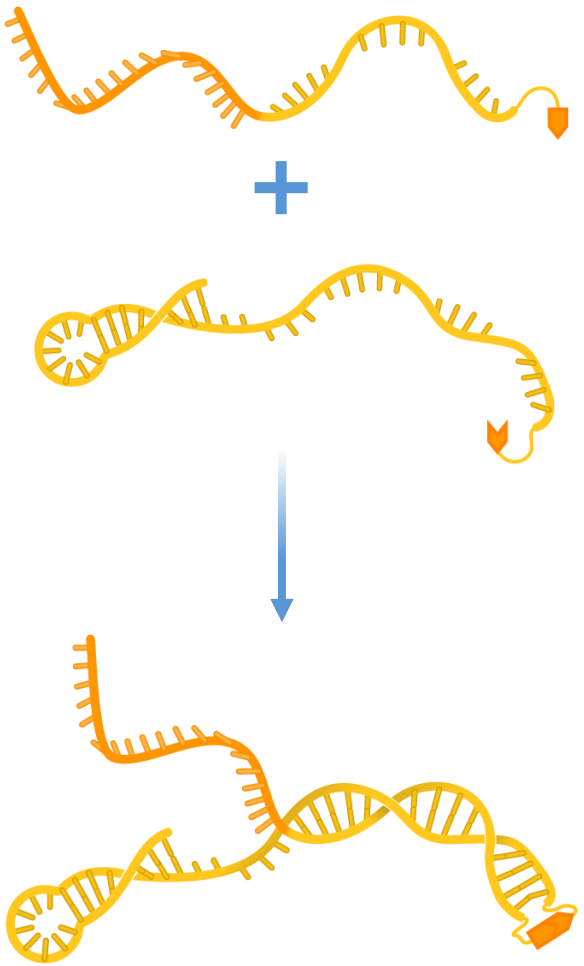
	COMPUTATIONAL SCREEN	CELLULAR & BIOCHEMICAL ASSAYS	TARGETED SEQUENCING PANELS
# GUIDE RNA	**1,000 – 2,000**	**50 – 100**	**5 – 10**
TARGETED		Biased Library of Targets (BLT)	Uni-directional Targeted Sequencing (UDiTaS) Bi-directional PCR
COMPREHENSIVE	GODOT	GUIDE-Seq CIRCLE-Seq Digenome	

i2 Asset Acquisition: Unmatched gRNA Expertise

World class RNA chemistry expertise

Enables best-in-class CRISPR medicines

Proprietary classes of guide RNAs with distinct intellectual property

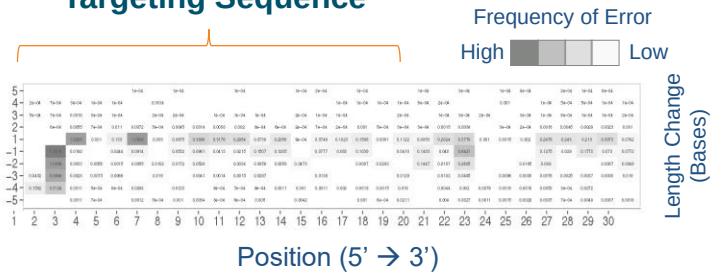


Proprietary Guide RNA Engineering

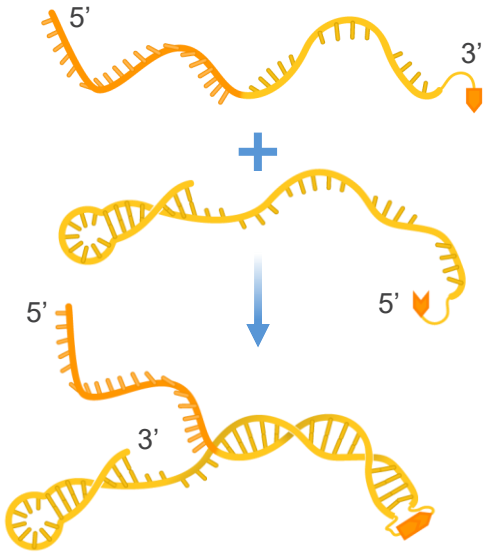
Single gRNA

Heterogeneous product
(full-length, truncated, errors)

Targeting Sequence

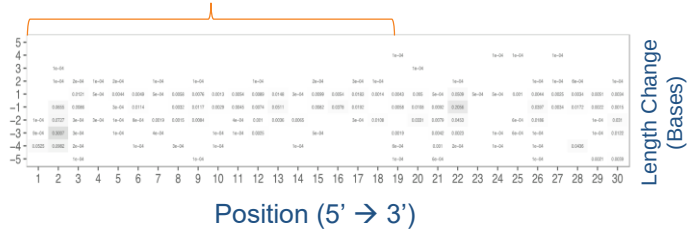


Covalently Coupled Dual gRNA



Well-defined product
(full-length only)

Targeting Sequence

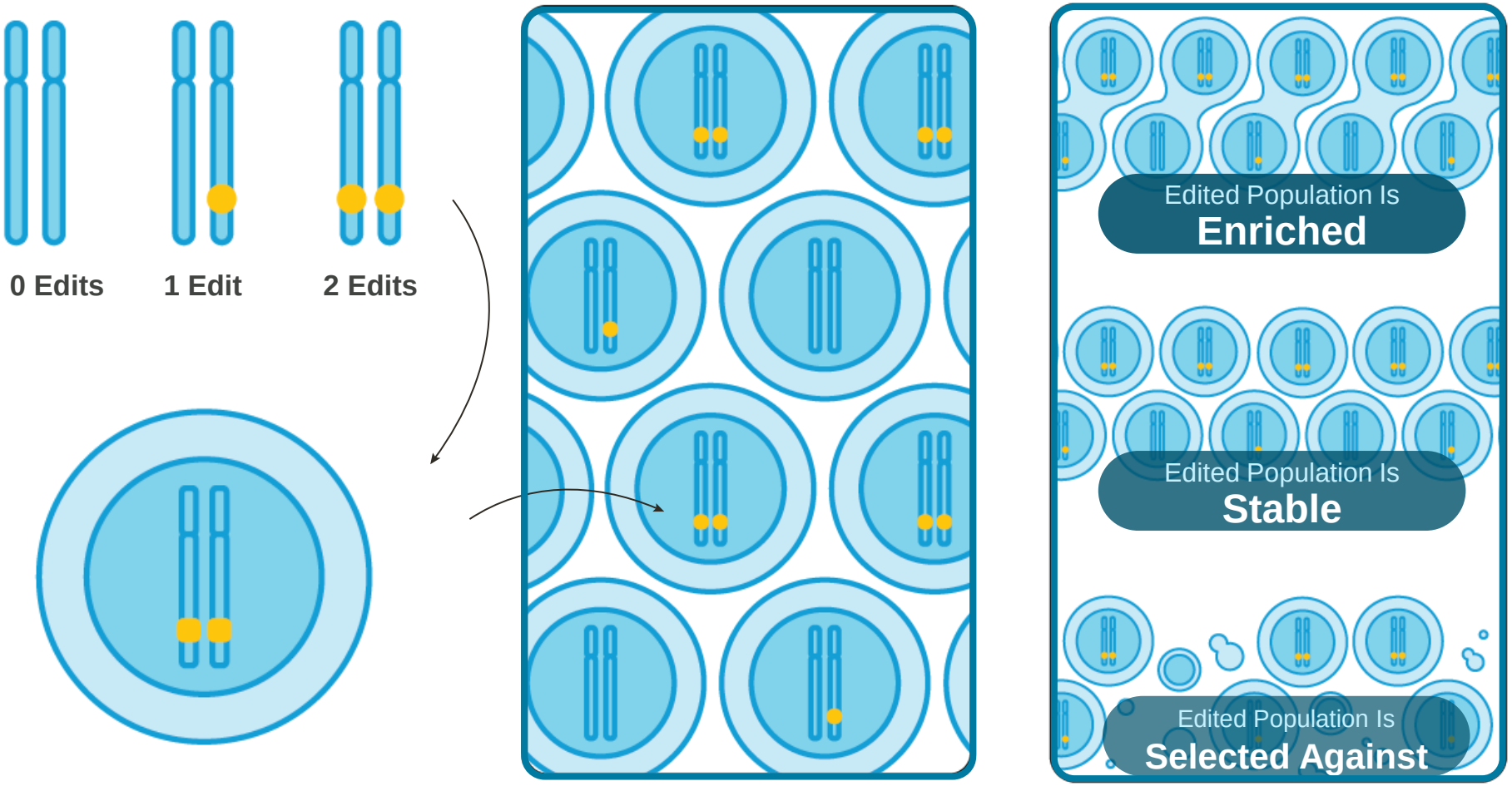


Fundamentals of Gene Editing Medicines

1 Editing Efficiency in Target Cell Type

2 Proportion of Target Cells Edited

3 Long-term Fate of Edited Cells



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 40 issued patents
worldwide, including in United States, Europe, and Australia

Over 500 pending patent applications
from Editas Medicine and academic institutions