KORRO BIO

Corporate Deck

Edit the Message, Rewrite the Future

November 2024

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Expanding the Frontiers of Genetic Medicines via Activation of Biological Pathways

Affecting a single base RNA edit (A-to-I edit) using proprietary oligonucleotide platform (OPERA™)

- Specific, transient, titratable and reversible using only an oligonucleotide
- Key internal discoveries driving the potential to develop multiple drug candidates
- Initial focus in rare liver, CNS and cardiometabolic diseases

Advancing KRRO-110 program for alpha-1 antitrypsin deficiency (AATD) with first participant dosing anticipated in Q1'25

Ongoing collaboration with Novo Nordisk to develop up to two therapeutic candidates in cardiometabolic diseases

Enabling multiple milestones for KRRO-110 and other pipeline programs with cash runway into 2H'26[1]

[1] Cash, cash equivalents and marketable securities of $169.1 million as of September 30, 2024

Experienced Management Team with Proven Track Record

Ram Aiyar, Ph.D.
Chief Executive Officer and President

Kemi Olugemo, M.D.
Chief Medical Officer

Vineet Agarwal, MBA
Chief Financial Officer

Todd Chappell, MBA
Chief Operating Officer

Jeffrey Cerio, Pharm.D., J.D.
SVP, General Counsel

Stephanie Engels
SVP, HR People and Culture

Venkat Krishnamurthy, Ph.D.
SVP, Head of Platform

CORVIDIA

ultragenyx pharmaceutical

J.P.Morgan

CombinatoRx

Atomwise Better medicines faster.

moderna

Lilly

Sofinnova partners

laronde

HealthCare VENTURES

moderna

Shire

AstraZeneca

IONIS

Johnson & Johnson

parexel

Alkermes

ZIMMER BIOMET

Dicerna pharmaceuticals

Board of Directors with Strong Development and Management Expertise

Nessan Bermingham, Ph.D.
Founder and Executive Chairman; Operating Partner, Khosla Ventures

Rachel Meyers, Ph.D.
Experienced operator in RNA medicines

Timothy Pearson
CEO, Carrick Therapeutics

Jean-Francois Formela, M.D.
Founder
Partner, Atlas Venture

Ali Behbahani, M.D.
General Partner, NEA

Katharine Knobil, M.D.
Seasoned pharmaceutical and biotech executive

Ram Aiyar, Ph.D.
President and CEO

kv

Intellia THERAPEUTICS

Alnylam PHARMACEUTICALS

FAZE MEDICINES

MILLENNIUM PHARMACEUTICALS, INC

Carrick therapeutics

TESARO

MedImmune

ATLAS VENTURE

IFM THERAPEUTICS

NEA

The Medicines Company

GSK

Agilent

kaleido

CORVIDIA

Sofinnova partners

Johnson & Johnson

Causal Missense Variants Have Been Identified in Both Rare and Common Diseases

nature genetics

Genetic variation in *PNPLA3* confers susceptibility to nonalcoholic fatty liver disease

Stefano Romeo[1,8], Julia Kozlitina[2,3,8], Chao Xing[1,2], Alexander Pe
Eric Boerwinkle[6], Jonathan C Cohen[1] & Helen H Hobbs[1,7]

> Hum Mol Genet. 2021 Apr 30;30(6):454-466. doi: 10.1093/hmg/ddab058.

Identification of LRRK2 missense variants in the accelerating medicines partnership Parkinson's disease cohort

[1], Cornelis Blauwendraat [2], Zhiyong Liu [1];

> J Med Genet. 2022 Sep 30;jmg-2022-108798. doi: 10.1136/jmg-2022-108798.
Online ahead of print.

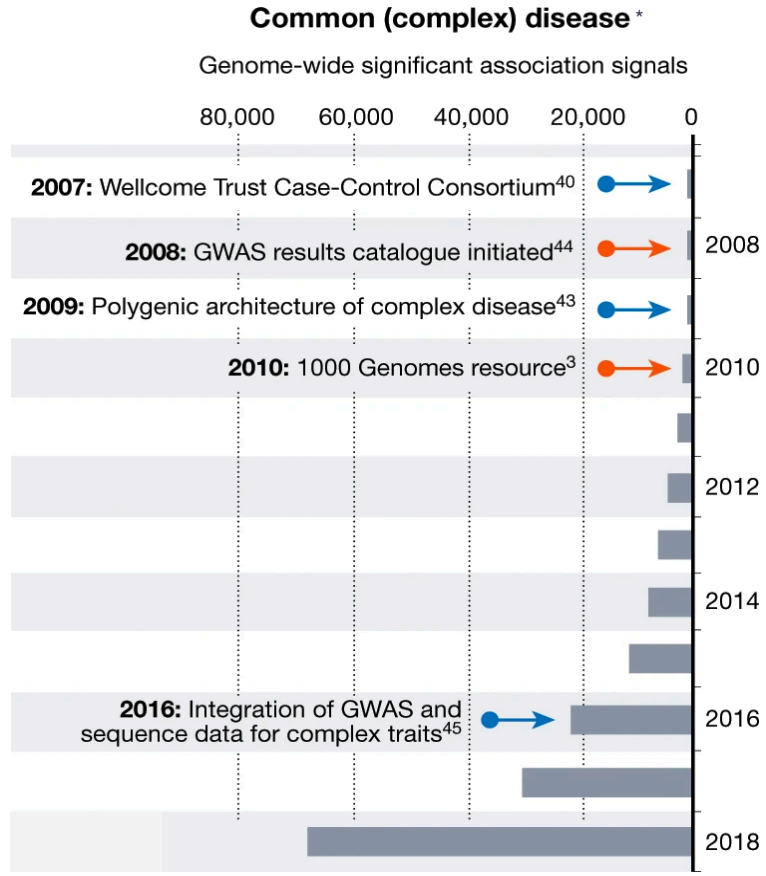
Identifying the molecular drivers of ALS-implicated missense mutations

Stephanie Portelli [1 2 3], Amanda Albanaz [4], Douglas Edua
David Benjamin Ascher [1 2 3]

> Pain Med. 2018 May 1;19(5):1010-1014. doi: 10.1093/pm/pnx261.

Common Missense Variant of SCN9A Gene Is Associated with Pain Intensity in Patients with Chronic Pain from Disc Herniation

Mateusz Kurzawski [1], Marcin Rut [2], Violetta Dziedziejko [3], Krzysztof Safranow [3], Anna Machoy-Mokrzynska [1], Marek Drozdzik [1], Monika Bialecka [4]

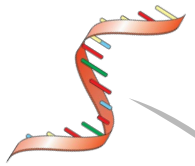


Need for an approach to transiently edit variants to modify biology and alleviate pathology

* Adapted from *Nature* Volume 577, pages 179–189 (2020)

RNA Editing: Transiently Effecting an A-to-I Edit on RNA Using an Oligonucleotide

Harnessing an endogenous editing enzyme, ADAR, that is ubiquitously expressed in all human cells



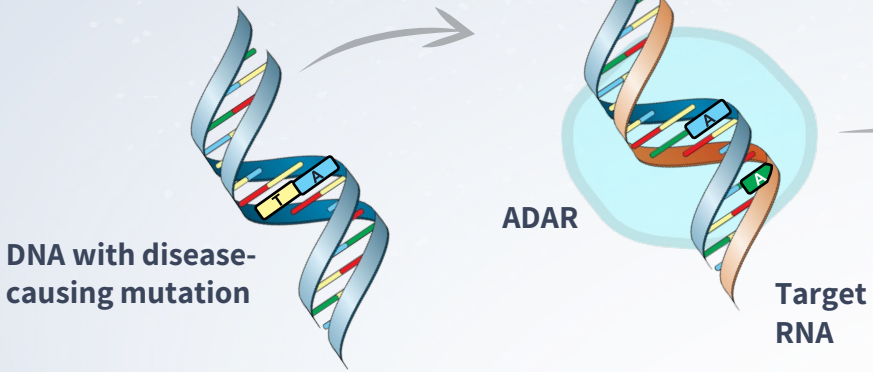
1 Non-viral intracellular delivery of Korro oligo designed to edit a specific adenosine on the target RNA

2 Oligo-RNA duplex recruits adenosine deaminase acting on RNA (ADAR)

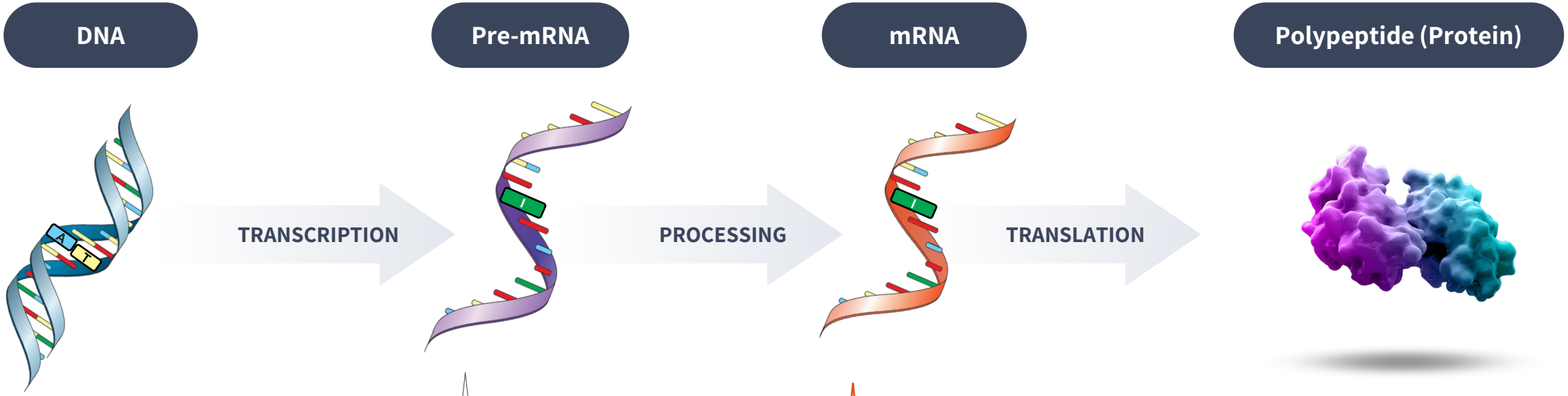
3 ADAR catalyzes deamination: 'A' to 'I' edit

4 mRNA translated to protein with 'I' read as 'G'

5 Resultant therapeutic protein



Broad and Versatile Opportunity to Impact Biology and Potentially Bring Multiple Therapeutic Options to Patients



Edit a single A-to-I

Modifying gene expression

Edit a single A-to-I

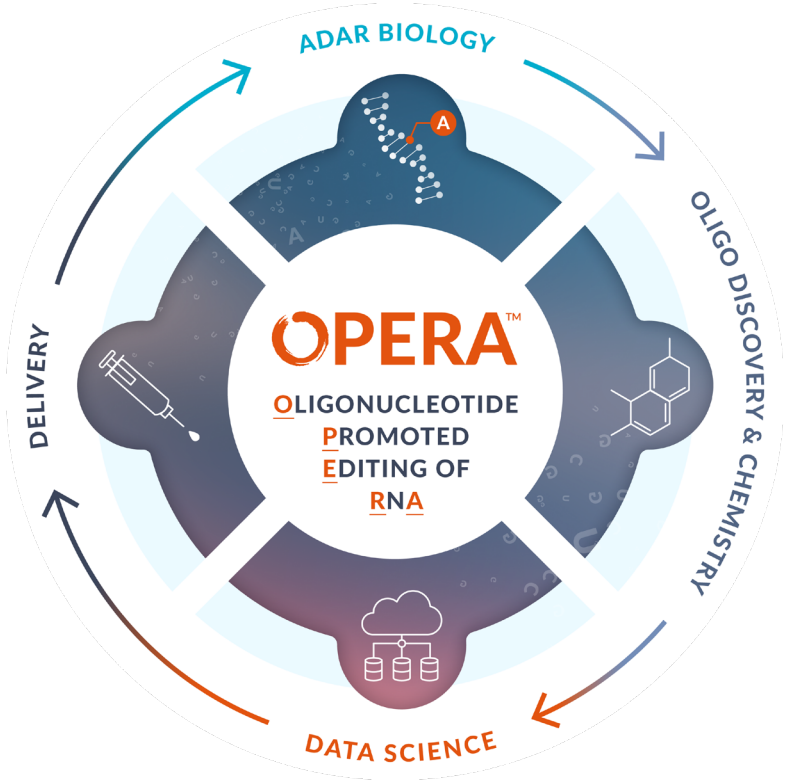
- **Repair** a G->A mutation to correct the protein
- **Generate a *de novo* protein** with preferred properties (can alter 12 amino acid sequences)

Current Focus

OPERA: Our Differentiated Approach for RNA Editing

Deep understanding of ADAR biology

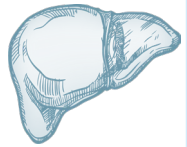
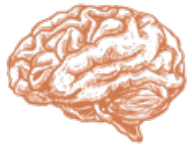
Expertise in oligonucleotide chemistry



Fit-for-purpose delivery

Machine learning optimization of oligonucleotides

Robust Pipeline with Multiple High-Value Targets

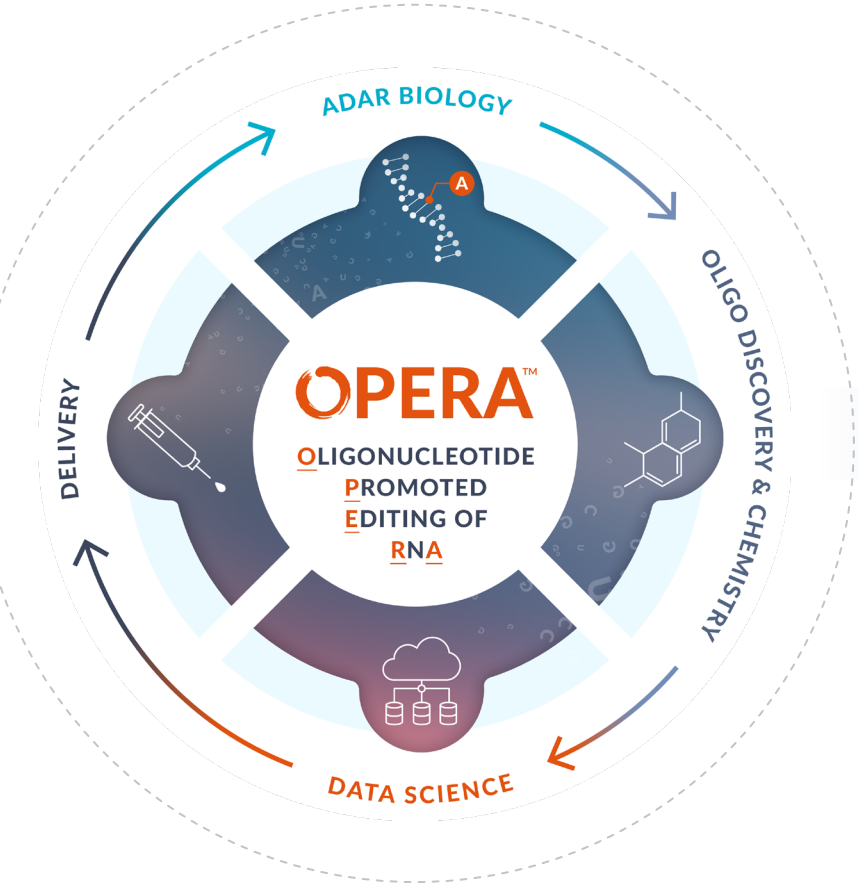
	CONCEPT	PROGRAM / INDICATION	DELIVERY	DISCOVERY	PRECLINICAL DEVELOPMENT	PHASE 1	PHASE 2	PHASE 3
	**Repairing a pathogenic variant**	**KRRO-110** AATD	LNP (IV)	**AAT**		**(Phase 1/2a Study)**		
	***De novo* protein to inhibit degradation**	Rare metabolic disorder	GalNAc (SQ)	**Undisclosed**				
	**Repairing a pathogenic variant**	Parkinson's disease	Undisclosed	**LRRK2**				
	***De novo* protein to overcome LoF and GoF** [1]	Amyotrophic lateral sclerosis	Undisclosed	**TDP43**				
	***De novo* protein to modulate currents**	Subsets of pain	Undisclosed	**$Na_v1.7$**				
novo nordisk®	**Undisclosed**	Cardiometabolic	Undisclosed	**Up to 2 Targets**				

Advancing KRRO-110 program with first participant dosing anticipated in Q1’25 and interim data in 2H’25

[1]De Novo protein variant to prevent toxic gain of function (GoF) with TDP43 aggregation, and still continue STMN2 signaling by overcoming toxic Loss-of-function (LOF)

OPERA: Our Approach

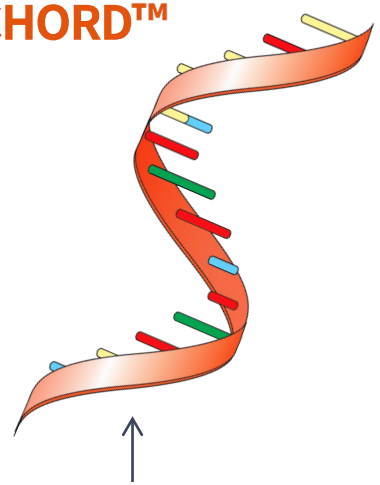
Customized High-fidelity Oligonucleotides for RNA Deamination (CHORD™)



Designed to have…

- High target efficiency
- High target specificity
- Computational efficiency
- Leveraging chemistry
- Leveraging delivery

CHORD™

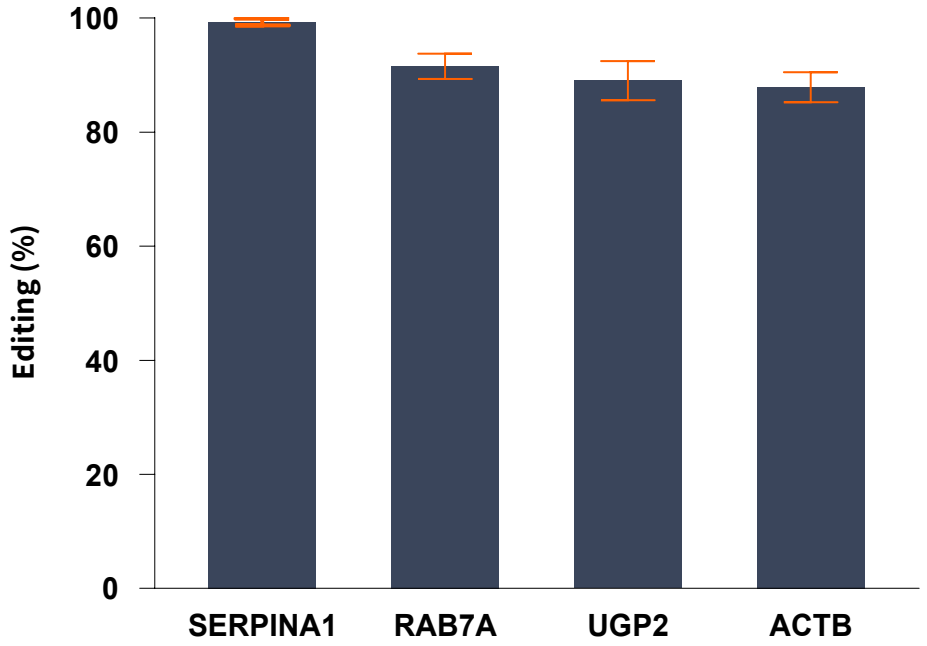


Gen 1.0:
A single-stranded, anti-sense oligonucleotide RNA editor

High Efficiency: Ability to Potentially Target Any "A" of Interest on Any Transcript

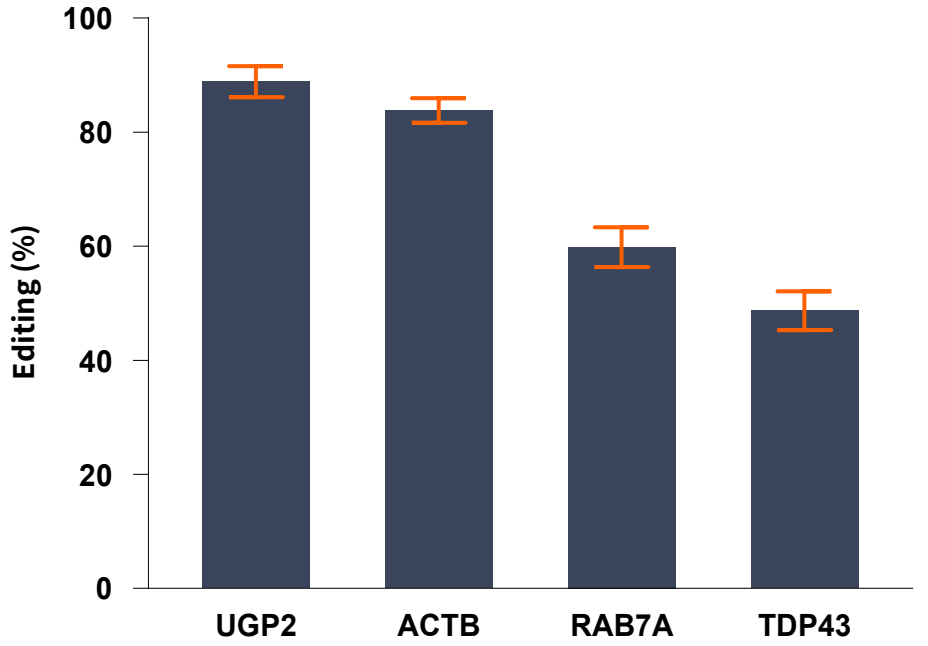
Primary Mouse Hepatocytes[1]

>80% editing achieved



Patient-derived Neuroblastoma Cells

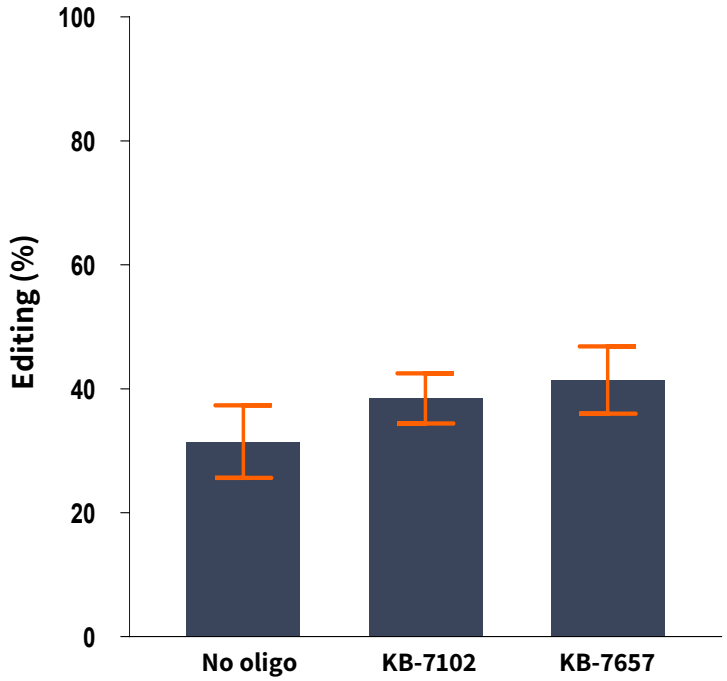
>45% editing achieved



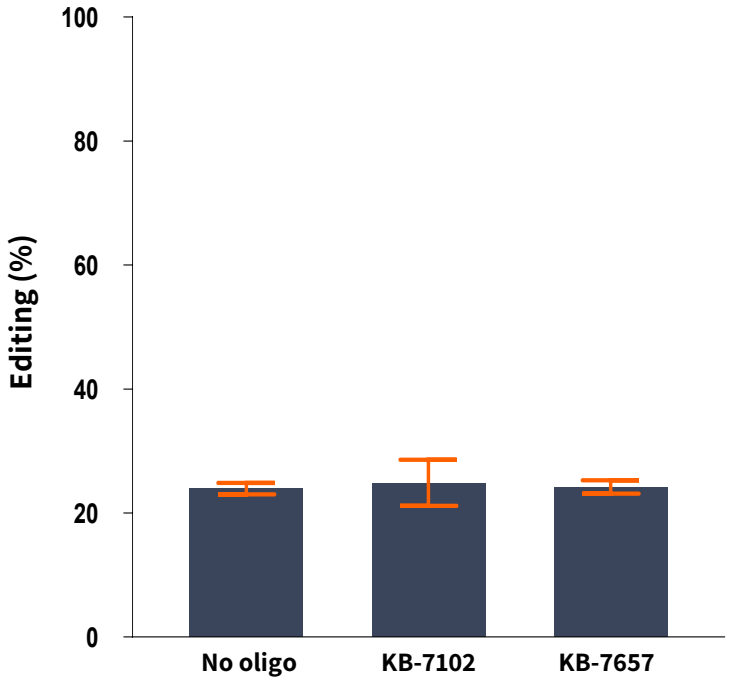
[1] SERPINA1 data is from PiZ primary mouse hepatocytes and ACTB, RAB7, and UGP2 data is from wild-type primary mouse hepatocytes (PMH)

High Specificity: CHORDs Do Not Interfere with Endogenous ADAR Activity in Preclinical Mouse Models

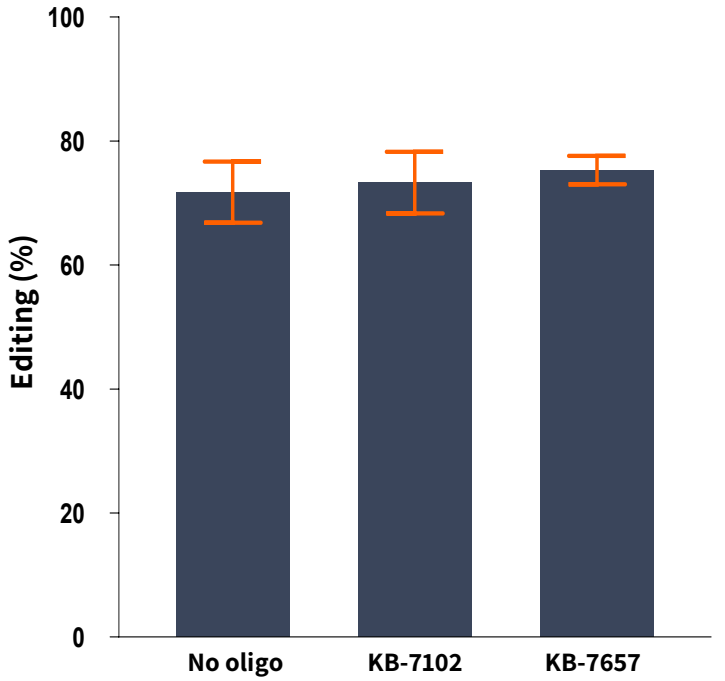
Endogenous site: COG



Endogenous site: COPA



Endogenous site: AJUBA



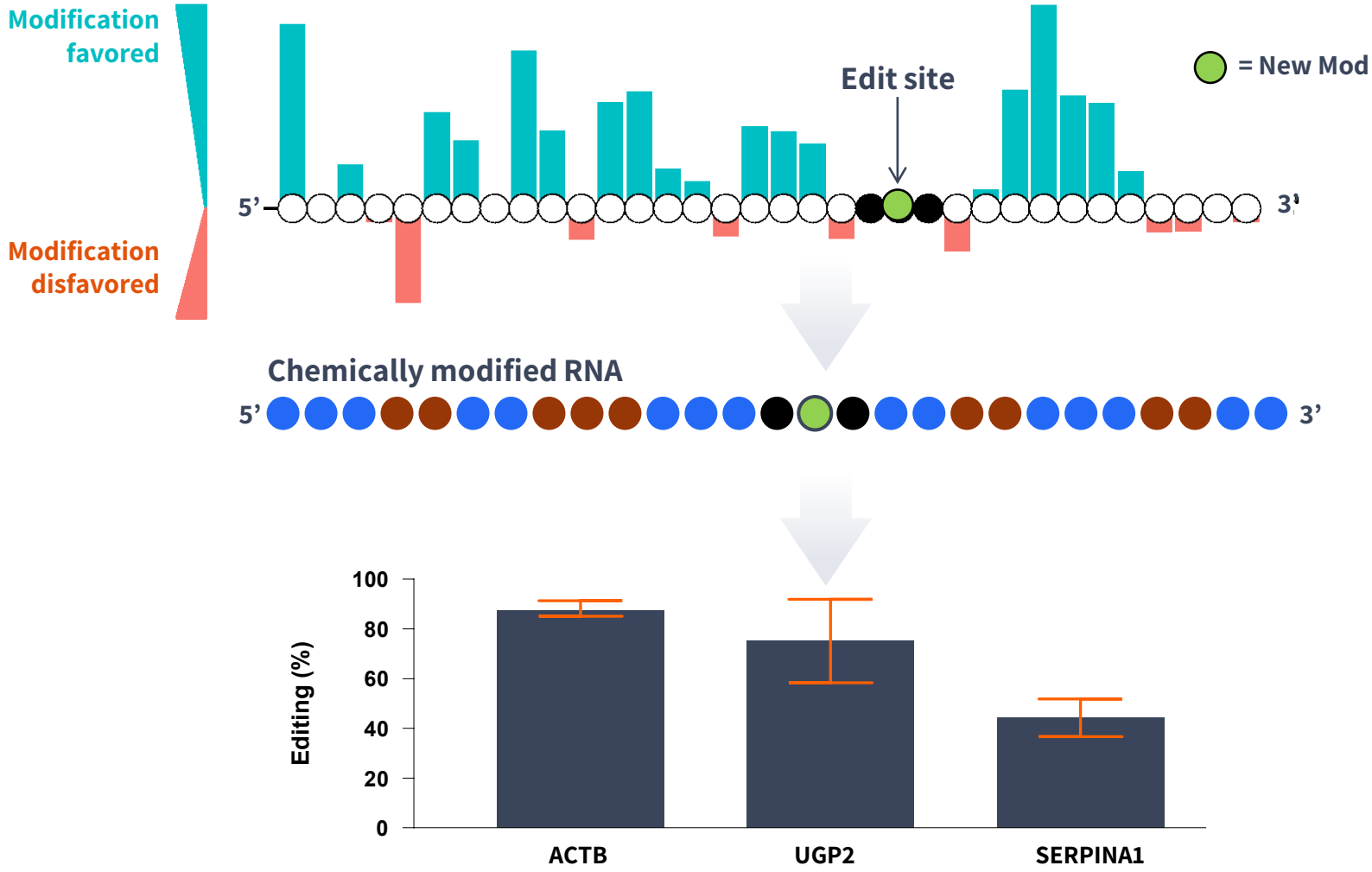
Note: KB-7102 -Target: Rab7; KB-7657 – Target SERPINA1
Ref: Edits on serotonin receptor lead to 24 different isoforms: Brenda Bass et. al. Nature Comm. 2011; 2: 319.; COG & COPA are edited by ADAR2 primarily. Tenen, D. J. et. al. Blood 2023; 141; 3078, AJUBA is edited by ADAR1 only, Jin Billy Li et. al. Nature Comm. 2021;12: 2165

Computational Efficiency: Machine Learning-Driven Identification of CHORDs Across Targets

Oligo models built through deep learning models

Template oligo design

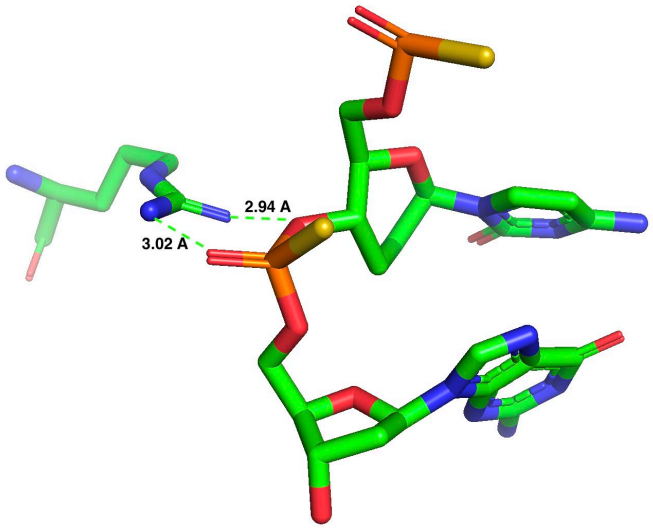
Replicated for multiple targets and sequences at baseline pre-optimization



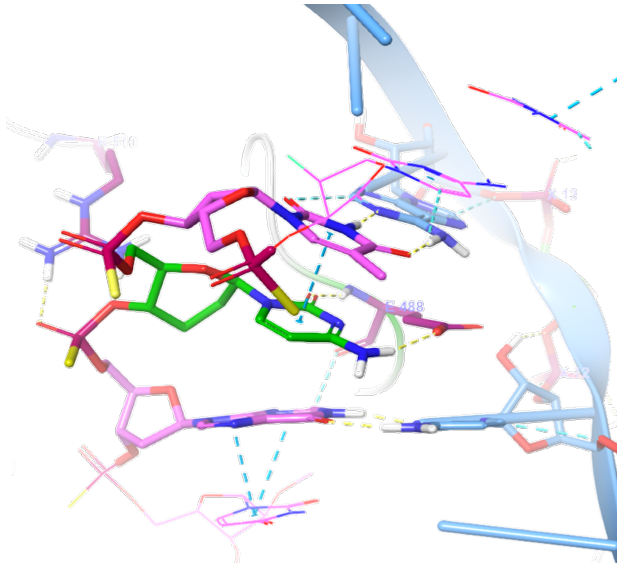
Note: ACTB and UGP2 data from primary mouse hepatocytes (PMH); SERPINA1 data from hepatocyte like cells (zzHLCs)

Leveraging Chemistry: Structural Biology Insights Enable Potency Boosts *In Vivo*

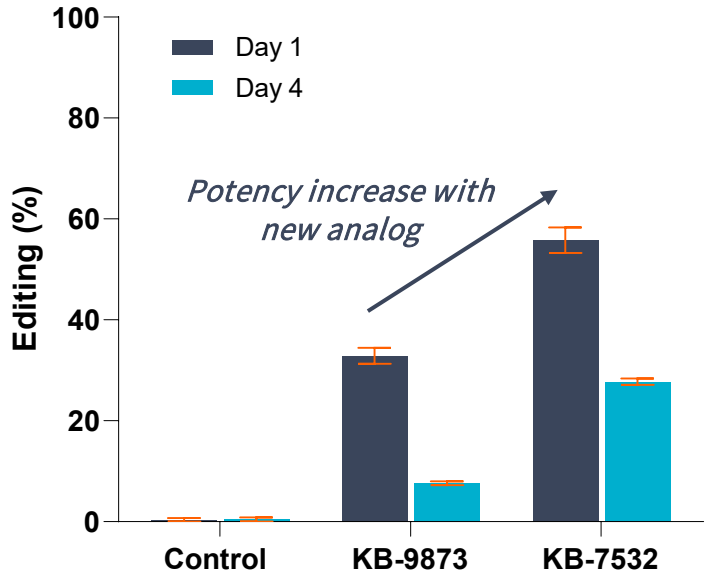
CHORD and target mRNA complexed with ADAR



New chemistry introduces improved potency



Significant improvement in editing *in vivo* in C57BL/6 mouse*

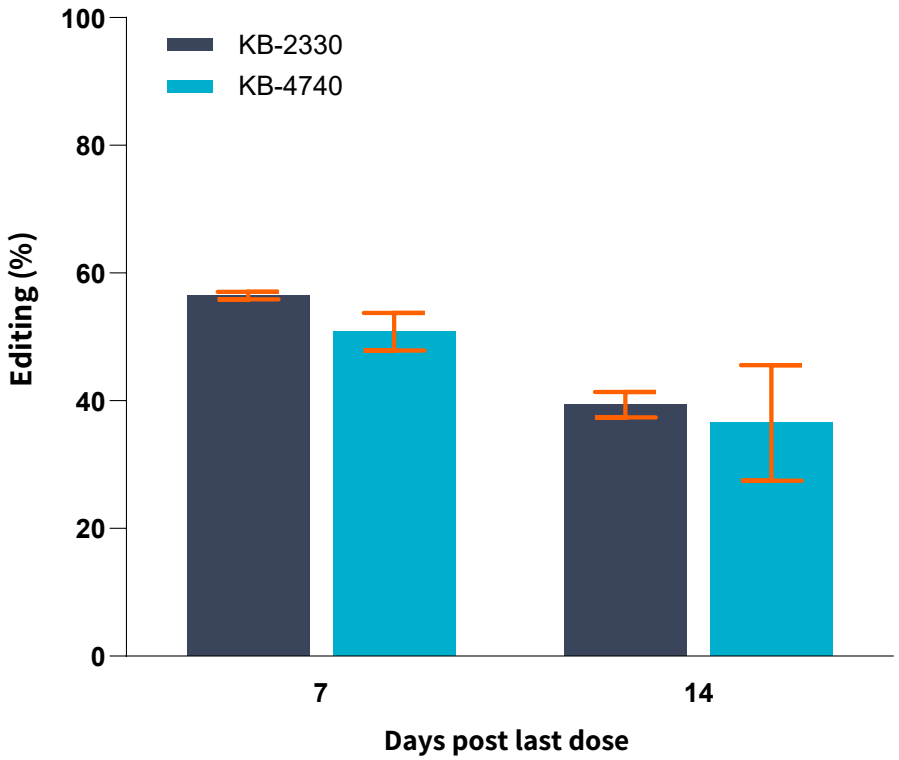


*3mg/kg oligo formulated in MC3 LNP injected IV

Leveraging Delivery: Fit-for-Purpose Based on Target Product Profile

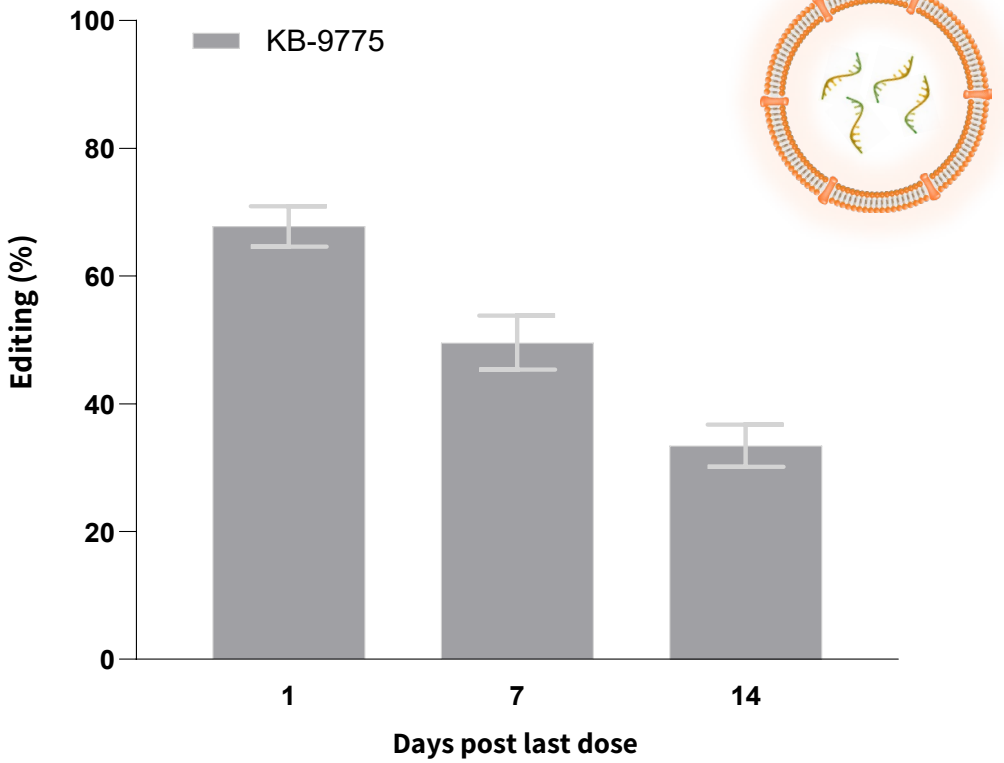
GalNAc (ACTB)

10mg/kg (QDx5); SC administration



MC3-LNP (RAB7A)

2mg/kg (single dose); IV administration



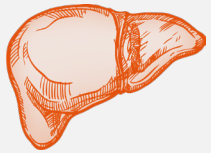
Note: GalNAc and LNP data from C57BL/6 mice, N=3/group

Alpha 1 Anti-trypsin Deficiency (AATD)

Delivering a Potential Best-in-Class Candidate

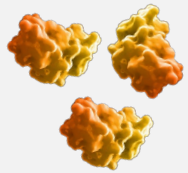
AATD Most Commonly Caused by a Single Missense (G-to-A) Mutation in SERPINA1 Gene in the Liver

MM Genotype
(normal liver and lung)

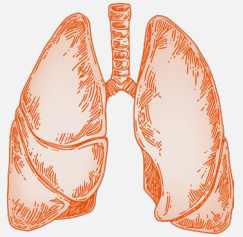


Normal levels of M-AAT secreted

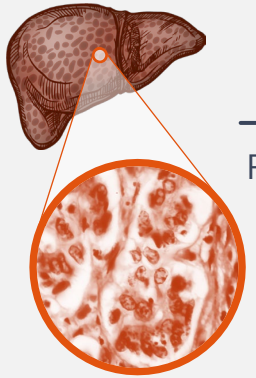
M-AAT



Inhibits neutrophil elastase in the lung



ZZ Genotype
(fibrotic liver and decreased lung function)

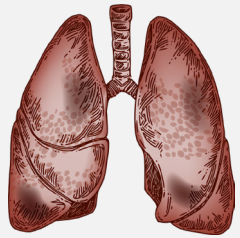


Reduced levels of Z-AAT secreted

Mutated AAT polymerizes and aggregates in liver cells

Z-AAT*

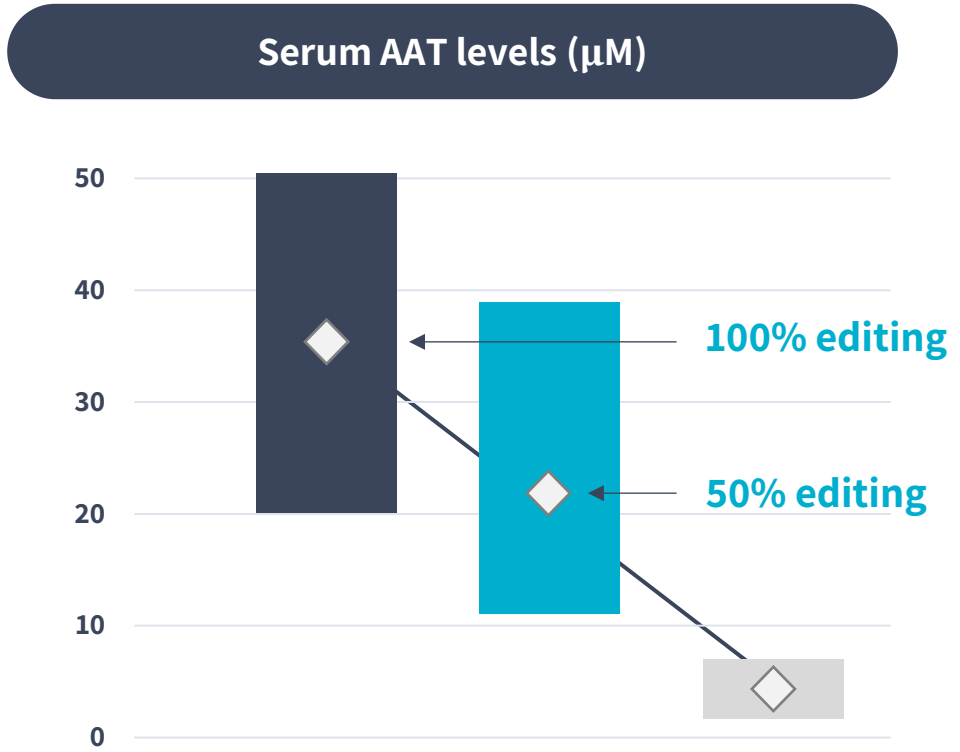
Minimal inhibition of lung neutrophil elastase



*~100K PiZZ adult patients in U.S.***

Note: AAT protein is encoded by the gene SERPINA1. The E342K mutation (G to A) in SERPINA1 (Z allele) is the most frequent mutation and causes severe lung and liver disease
*Z-AAT not as active as M-AAT
**Source: Alpha-1 Foundation. Numbers reflected here are carrier of ZZ genotypes

Focused on Increasing AAT levels in ZZ Patients to Between MM and MZ Levels



◇ = Median AAT for genotype

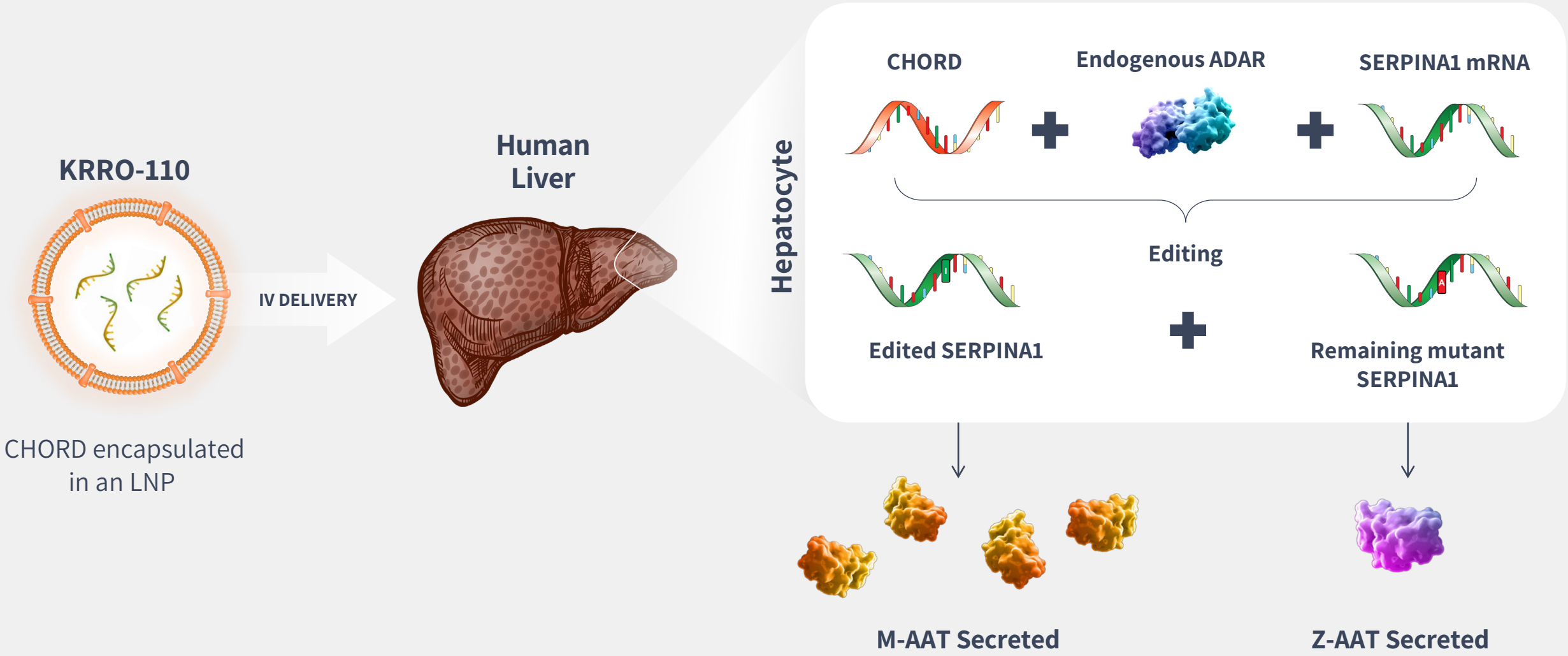
Korro's goal for median editing has potential to reduce lung and liver risk

Linear relationship with total AAT and genotype

Odds Ratio[1]	MM	MZ	ZZ
COPD[2]	1.0	1.0	8.8
Cirrhosis	1.0	1.5	7.8

[1]Nakanishi T. et al. Eur Respir J. 2020 Dec 10;56(6):2001441
[2] Chronic obstructive pulmonary disease

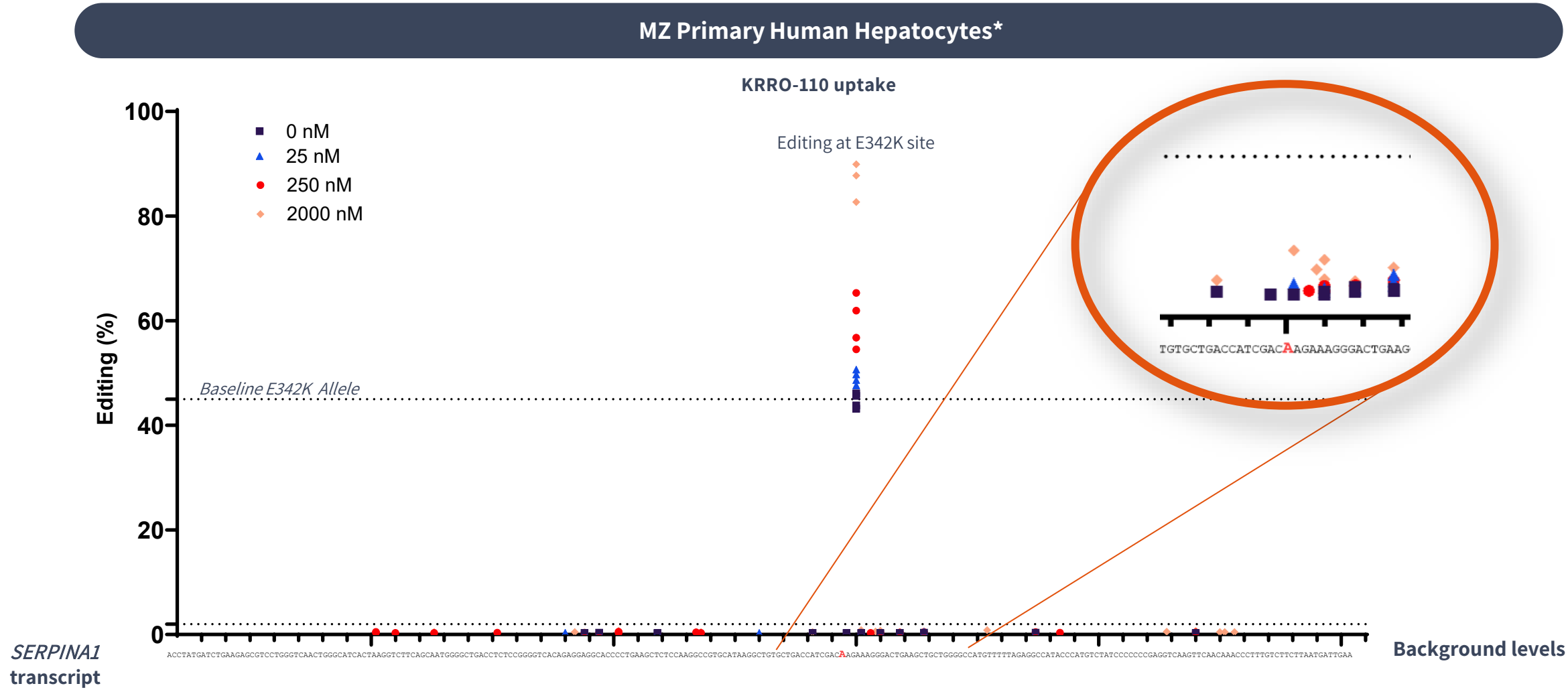
KRRO-110 Designed to Correct the Pathogenic Z-AAT Protein to M-AAT Protein in Preclinical Models



CHORD encapsulated in an LNP

Note: Editing is a function of number of transcripts in each cell

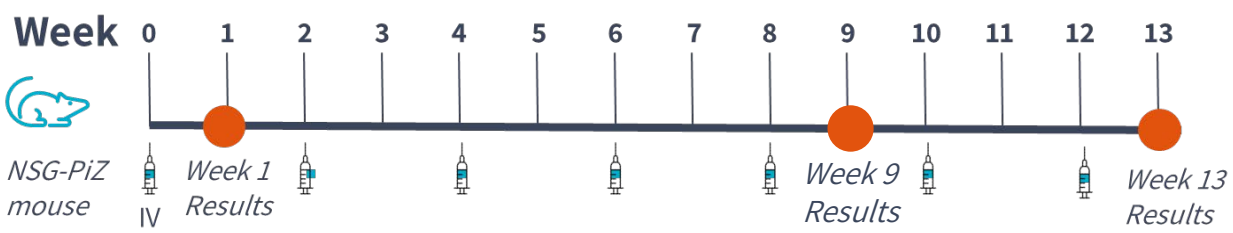
Negligible *In Vitro* Cis Off-Target Editing Observed for KRRO-110 in MZ Hepatocytes



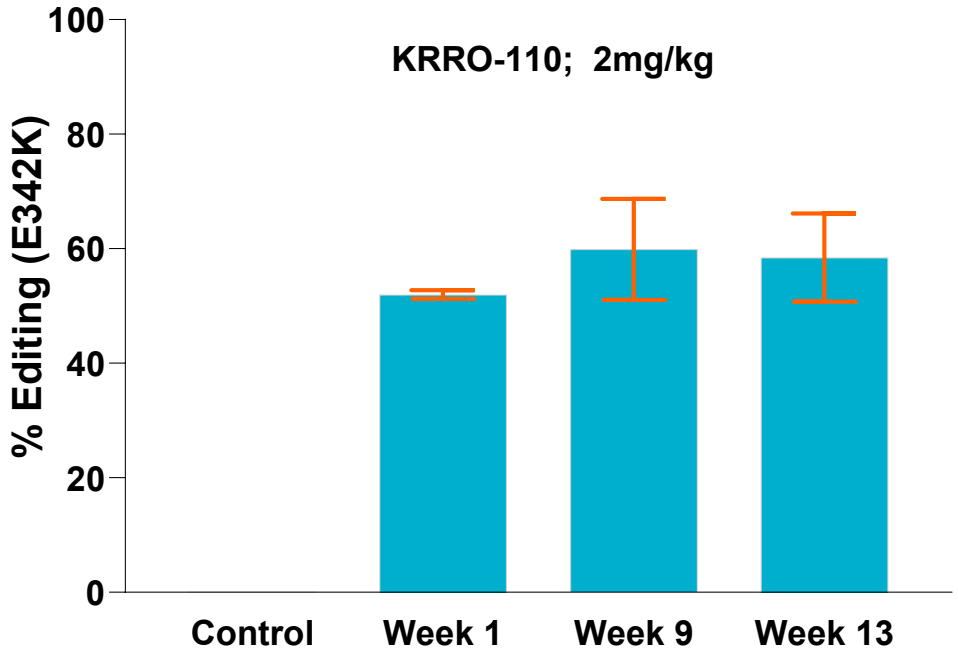
*Note: Each data point represents one experimental replicate with statistically significant editing above sequencing error

Achieved ~60% Editing in Human Transgenic Mouse Model After Multiple Doses

Study design



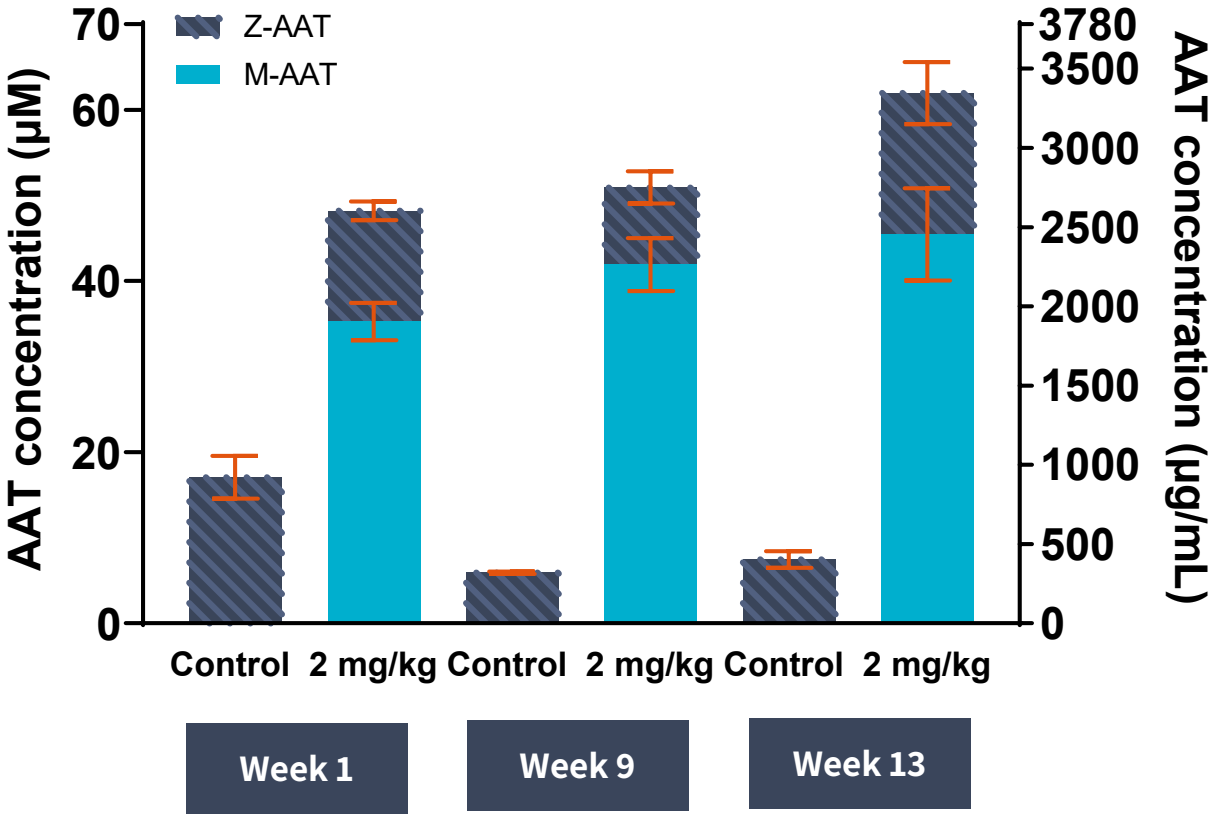
RNA Editing in NSG-PiZ mouse



Achieved Greater Than 60uM total AAT Protein and 45uM of M-AAT Levels at Week 13

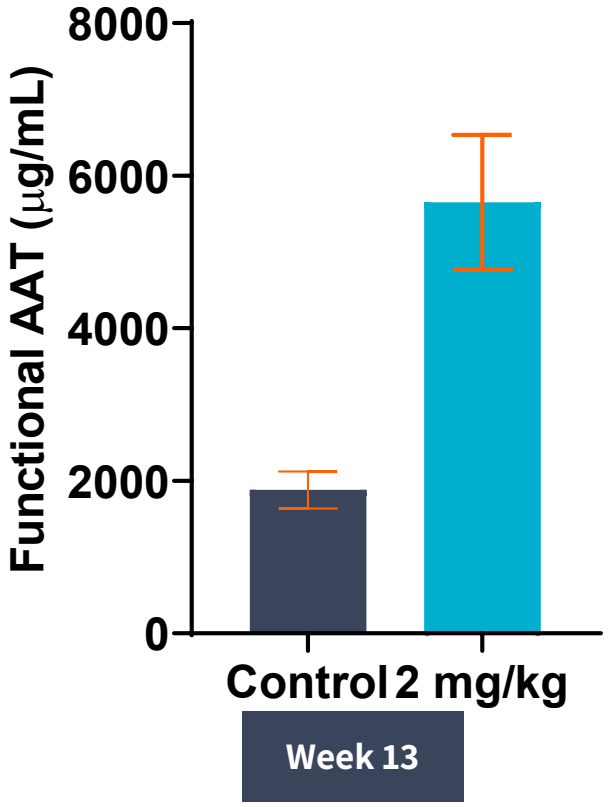
Serum human-AAT concentration

KRRO-110; 2mg/kg



NSG-PiZ mouse functional AAT concentration

KRRO-110; 2mg/kg



Note: Data represented as average values (n=3) +/- SD
* Positive control human serum inhibits the human neutrophil elastase

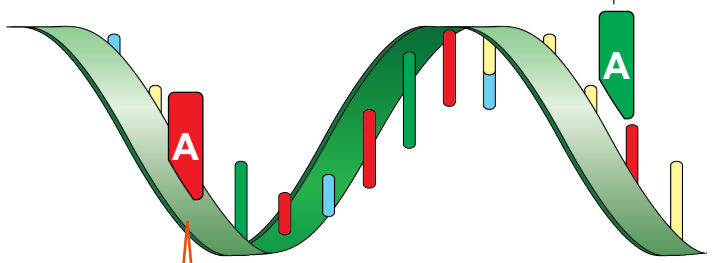
Editing *De Novo* Adenosine on Cyno SERPINA1 to Elucidate Editing in Higher Species

E342K target site for KRRO-110

Utility in PiZ mouse

Edited (M-AAT) protein detected

SERPINA1 transcript



>98% homology of human ADAR and cyno ADAR

Surrogate target site to induce amino acid change for KB-1494-GVT1
(different construct with similar chemistry)

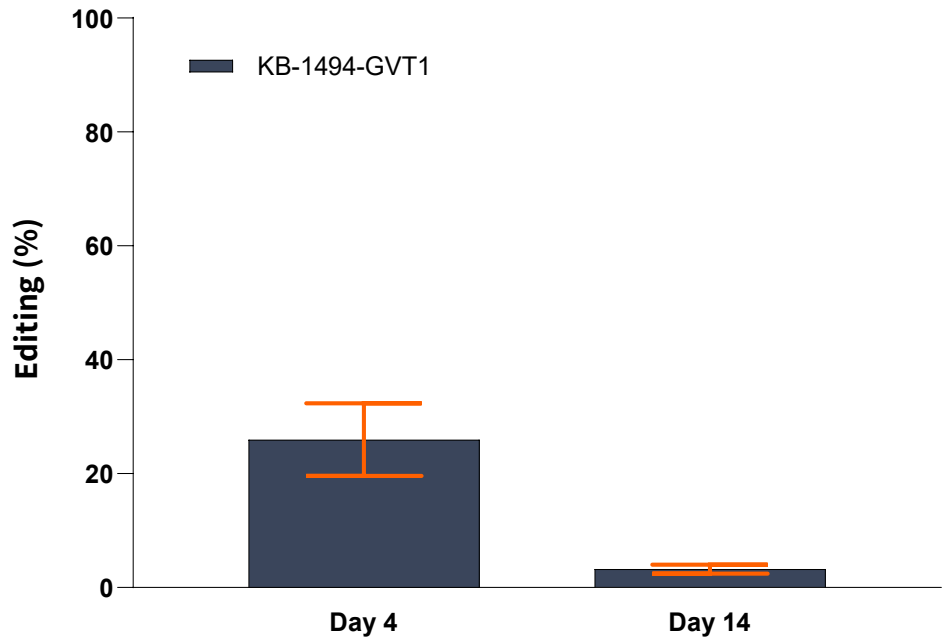
Utility in PiZ mouse and in NHPs

Edited protein detected

Editing at Surrogate Target Site in AATD Mouse Model Translated to Higher Species

Editing in C57BL/6-PiZ mice (%)

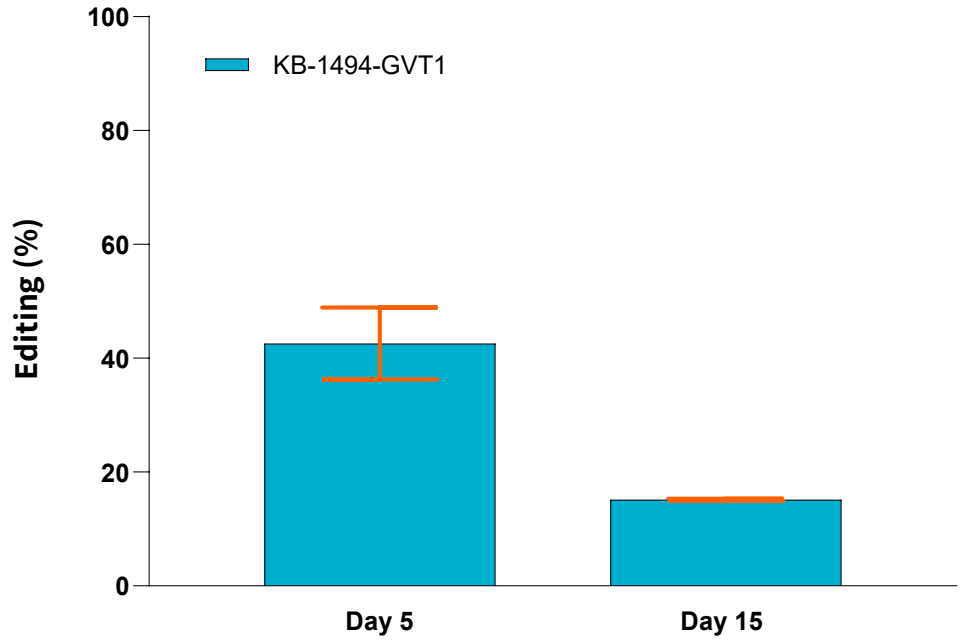
2mg/kg (single dose)



Surrogate Target Site: SERPINA1

Editing in NHPs (%)

2mg/kg (single dose)



Surrogate Target Site: SERPINA1

Note: Data represented as average values +/- SD

KRRO-110 Has Potential for Best-in-Class Profile for AATD Patients

Efficacy

- ✓ Achieved AAT levels between MM and MZ in rodents as early as Week 1
- ✓ Secreted functional AAT and inhibits neutrophil elastase
- ✓ Rapid reduction in Z-aggregates and Z-AAT protein

Safety

- ✓ No off-target effect observed to date
- ✓ No effect on endogenous ADAR activity observed to date
- ✓ Well tolerated in non-GLP safety studies (mice, NHP)

Translation to higher species

- ✓ Ability to edit in human cells
- ✓ Translation to NHP with surrogate oligo

First participant dosing expected in Q1'25; Interim data readout anticipated in 2H'25

Clinical Study of KRRO-110 for AATD

Phase 1/2a, Two Part, Single- and Multiple-Dose Escalation Study

REWRITE Clinical Trial

Study Population

- Up to 64 adult participants
- PiMM healthy volunteers or clinically stable PiZZ patients

Study Design

- **Part 1** (SAD): Active: PBO (2:1) Cohorts of PiMM and PiZZ
- **Part 2** (MAD): Open-label, Cohorts of PiZZ patients

Endpoints

- Primary: Safety and tolerability
- Secondary: Pharmacokinetic (PK) parameters; T-AAT, M-AAT, Z-AAT, functional antiprotease activity

Two clinical study sites identified in Australia with plans to expand into additional sites in Australia and other geographies

Clinicaltrials.gov | NCT06677307

Clinical Advisory Board with Leading Lung and Liver Experts in AATD

Pulmonary Experts

Daniel Chambers, MBBS, MRCP, FRACP, MD, FQA

Monica Goldklang, MD

Noel G. McElvaney, MBBCh, FRCPI, DSc

Alice M. Turner, MBChB (Hons), MRCP, PGCE (MedEd), PhD

Hepatic Experts

Pavel Strnad, MD

Jeffrey Teckman, MD

Expanding the Frontiers of Genetic Medicines via Activation of Biological Pathways

Affecting a single base RNA edit (A-to-I edit) using proprietary oligonucleotide platform (OPERA™)

- Specific, transient, titratable and reversible using only an oligonucleotide
- Key internal discoveries driving the potential to develop multiple drug candidates
- Initial focus in rare liver, CNS and cardiometabolic diseases

Advancing KRRO-110 program for alpha-1 antitrypsin deficiency (AATD) with first participant dosing anticipated in Q1'25

Ongoing collaboration with Novo Nordisk to develop up to two therapeutic candidates in cardiometabolic diseases

Enabling multiple milestones for KRRO-110 and other pipeline programs with cash runway into 2H'26[1]

[1] Cash, cash equivalents and marketable securities of $169.1 million as of September 30, 2024

Edit the message.
Rewrite the future.