KORRO BIO

J.P. Morgan Healthcare Conference

Edit the Message, Rewrite the Future

January 2024

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Certain statements in this Presentation may constitute "forward-looking statements". Forward-looking statements include, but are not limited to, express or implied statements regarding expectations, hopes, beliefs, intentions or strategies of Korro Bio, Inc. (Korro) regarding the future including, without limitation, express or implied statements regarding: Korro's RNA editing technology and the benefits of OPERA; the market opportunity for KRRO-110 and potential benefits over other alpha-1 anti-trypsin deficiency (AATD) modalities; the potential of KRRO-110 to be a best-in-class drug candidate for AATD; the potential safety and efficacy of KRRO-110; Korro's expected cash runway and plans for discovery and preclinical studies, as well as clinical trials, including timing of regulatory filings and data readouts and other developments or results in connection therewith. In addition, any statements that refer to projections, forecasts, or other characterizations of future events or circumstances, including any underlying assumptions, are forward-looking statements. The words "anticipate," "believe," continue," " could," "estimate," " expect," " intend," "may," "might," "plan," "possible," "potential," "predict," "project," " should," " strive," "would," "aim," "target," "commit," and similar expressions may identify forward-looking statements, but the absence of these words does not mean that statement is not forward looking. Forward-looking statements are based on current expectations and assumptions that, while considered reasonable are inherently uncertain. New risks and uncertainties may emerge from time to time, and it is not possible to predict all risks and uncertainties. Factors that may cause actual results to differ materially from current expectations include, but are not limited to, various factors beyond management's control including risks inherent in biopharmaceutical development; risks associated with pre-clinical studies and clinical trials; and other risks associated with obtaining regulatory approvals and protecting intellectual property; as well as risks associated with general economic conditions; the inability to recognize the anticipated benefits of the recently completed merger, which may be affected by, among other things, competition, Korro's ability to grow and manage growth profitably, maintain relationships with customers and suppliers and retain key employees; costs related to merger; the possibility that Korro may be adversely affected by other economic, business, and/or competitive factors; other risks and uncertainties indicated from time to time in Korro's filings with the SEC, including in Exhibit 99.2 to its Current Report on Form 8-K filed with the SEC on November 6, 2023, as such may be amended or supplemented by its other filings with the SEC. Nothing in this Presentation should be regarded as a representation by any person that the forward-looking statements set forth herein will be achieved or that any of the contemplated results of such forward-looking statements will be achieved. You should not place undue reliance on forward-looking statements in this Presentation, which speak only as of the date they are made and are qualified in their entirety by reference to the cautionary statements herein. Korro does not undertake or accept any duty to release publicly any updates or revisions to any forward-looking statements to reflect any change in their expectations or in the events, conditions or circumstances on which any such statement is based. This Presentation does not purport to summarize all of the conditions, risks and other attributes of an investment in Korro.

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Uniquely Positioned to Expand the Frontiers of Genetic Medicines through RNA Editing

- **Built an experienced team with a proven track record in genetic medicines**
- **Built an oligonucleotide-based approach (OPERA™) to affect a single base edit on RNA (efficient, specific and transient)**
- **Nominated a candidate (KRRO-110) for alpha-1 antitrypsin deficiency (AATD) with potential for best-in-class profile**
- **Continuing to build a unique, wholly-owned pipeline with broad opportunities in rare and common diseases**
- **Strong balance sheet with cash runway into ’26 enabling a potential interim clinical readout for AATD in 2H ‘25[1,2]**

[1] Subject to submission of an Investigational New Drug (IND) application to the U.S. Food and Drug Administration (FDA) or similar application with regulatory agencies in other geographies and subsequent authorization to proceed
[2] Korro Bio closed reverse merger and financing transaction on 11/3/23 with cash, cash equivalents and short-term investments of approximately $170.0 million

Create Transformative Genetic Medicines for Diseases with High Prevalence

- A transient and reversible way to edit RNA (A-to-I edit) using an endogenous "editor"
- Expanding the genetic medicines tool-kit by providing an “activation” approach
- Key internal discoveries driving the potential to develop multiple drug candidates
- Initial focus on unique opportunities in rare liver and CNS indications

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.

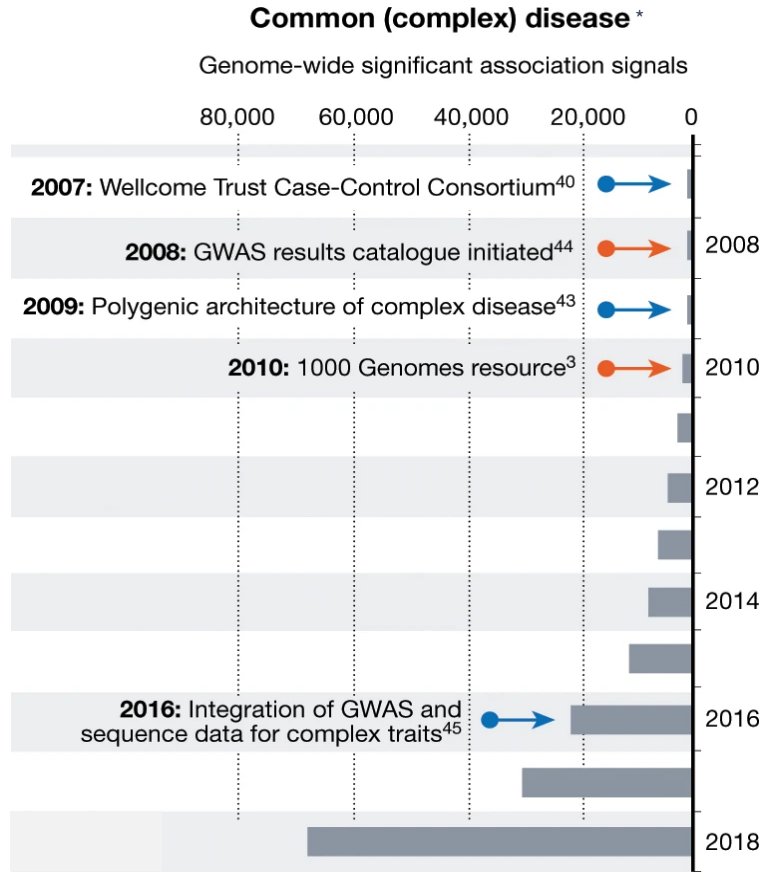
Identifying the molecular drivers of ALS-implicated missense mutations

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]

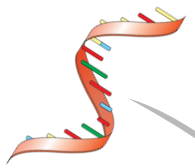


Need for an approach to transiently edit variants to modify biology and alleviate pathology

* 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

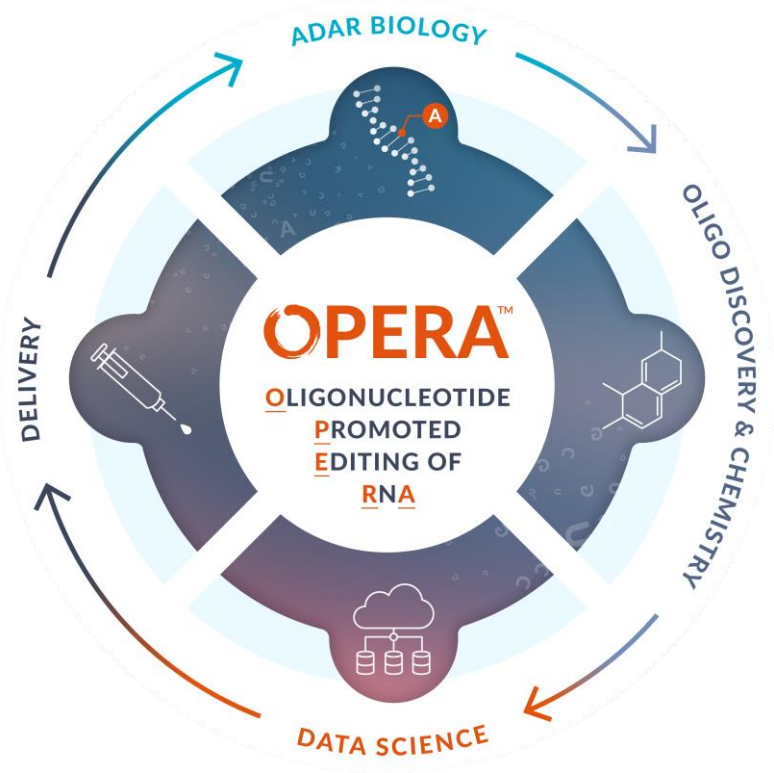
4 mRNA translated to protein with 'I' read as 'G'

5 Resultant therapeutic protein

OPERA: Our Differentiated Approach for RNA Editing

Deep understanding of ADAR biology

Expertise in oligonucleotide chemistry



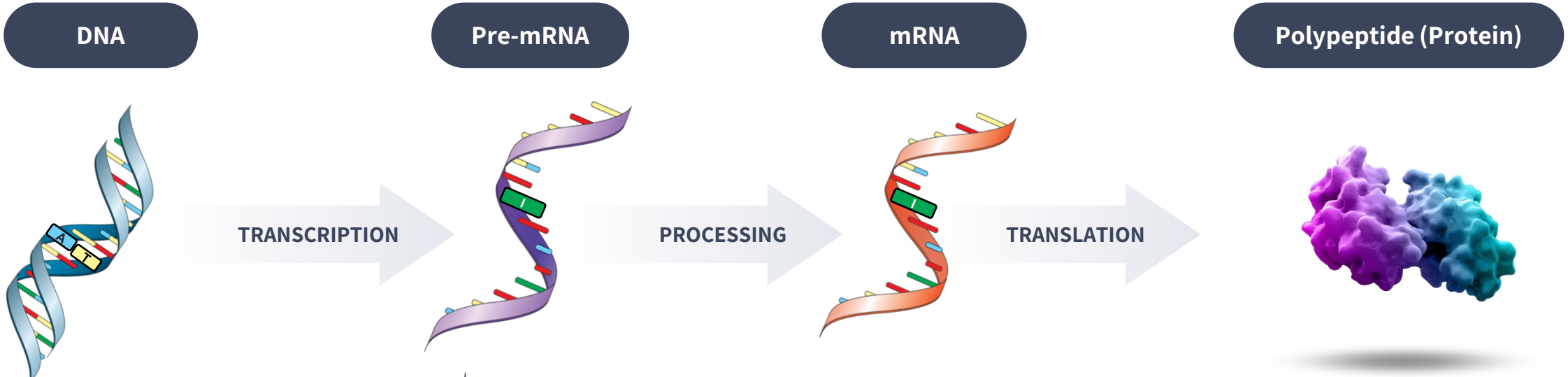
Fit-for-purpose delivery

Machine learning optimization of oligonucleotides

Comprehensive IP portfolio with 32 patent families[1] covering Korro platform technology and editing strategies

[1] IP estate count as of September 18, 2023 for Korro technology (excludes legacy Frequency Therapeutics IP)

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

Wholly-Owned Pipeline with Multiple High-Value Targets

CONCEPT	PROGRAM / INDICATION	DISCOVERY	PRECLINICAL DEVELOPMENT	PHASE 1	PHASE 2	PHASE 3	WHOLLY OWNED?
Repairing a pathogenic variant	**KRRO-110** Alpha-1 antitrypsin deficiency	**AAT**	**FIH-enabling regulatory filing expected 2H'24**[1]				✓
Repairing a pathogenic variant	Parkinson's disease	**LRRK2**					✓
***De novo* protein to disrupt aggregation**	Amyotrophic lateral sclerosis	**TDP43**					✓
***De novo* protein to modulate currents**	Subsets of pain	**$Na_v1.7$**					✓

Cash runway into '26 enabling a potential interim clinical readout for AATD in 2H '25[1,2]

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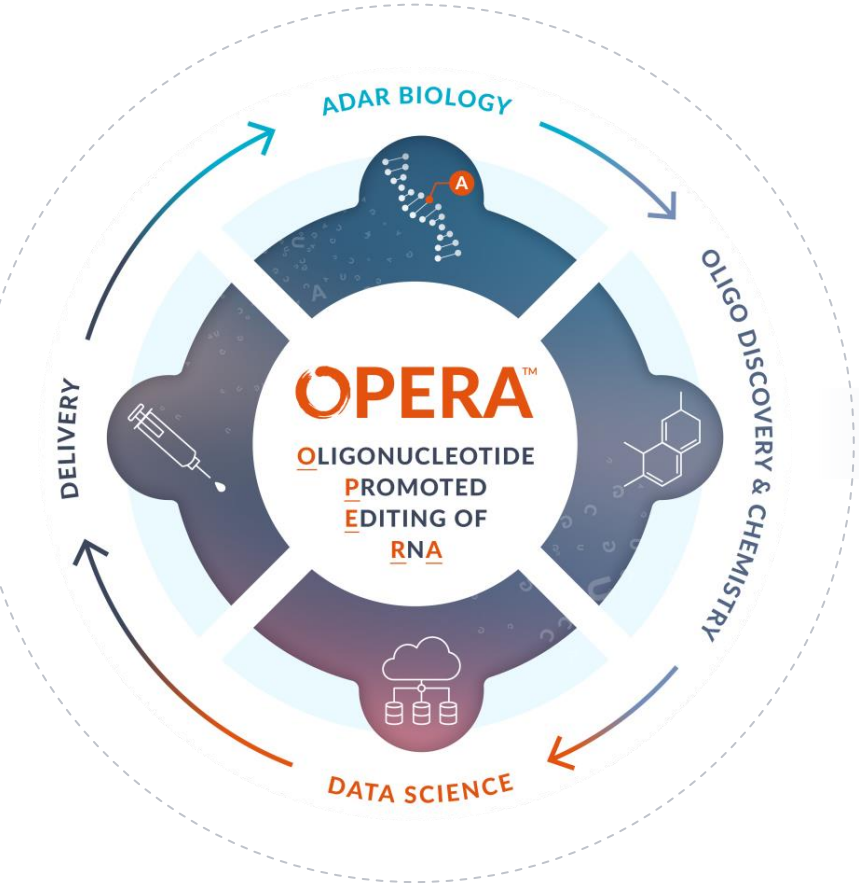
OPERA: Our Approach

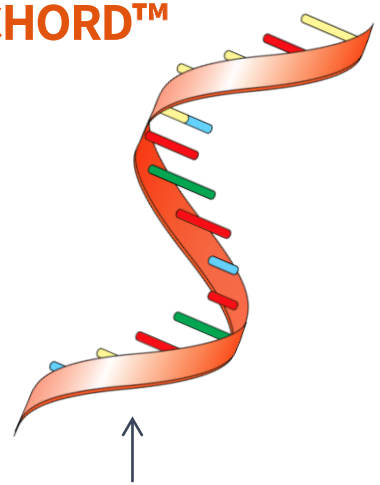
Customized High-fidelity Oligonucleotides for RNA Deamination (CHORD™)



Designed to have…

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

CHORD™

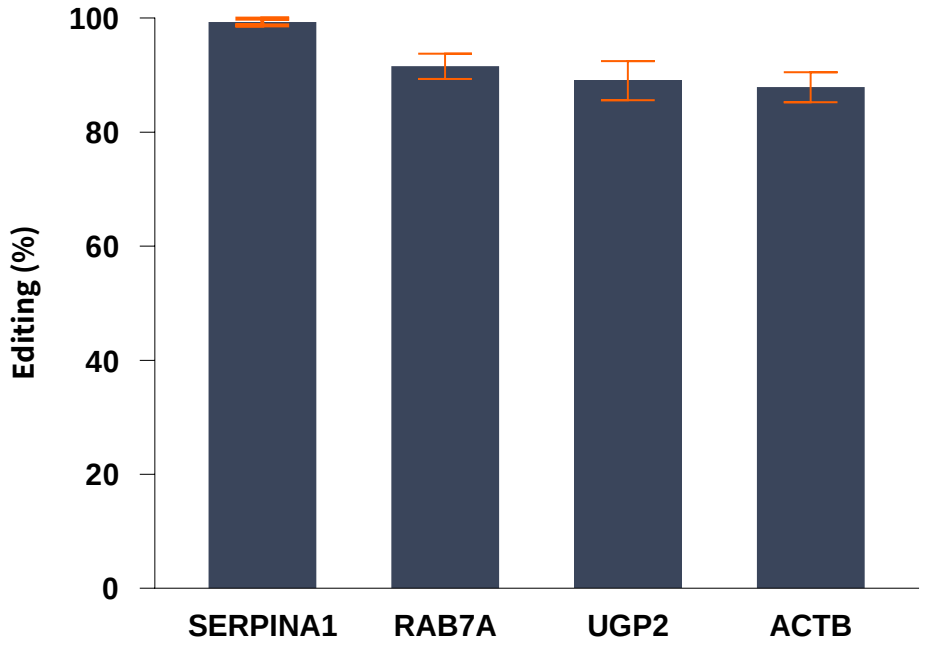


Gen 1.0:
A single-stranded, anti-sense oligonucleotide RNA editor

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

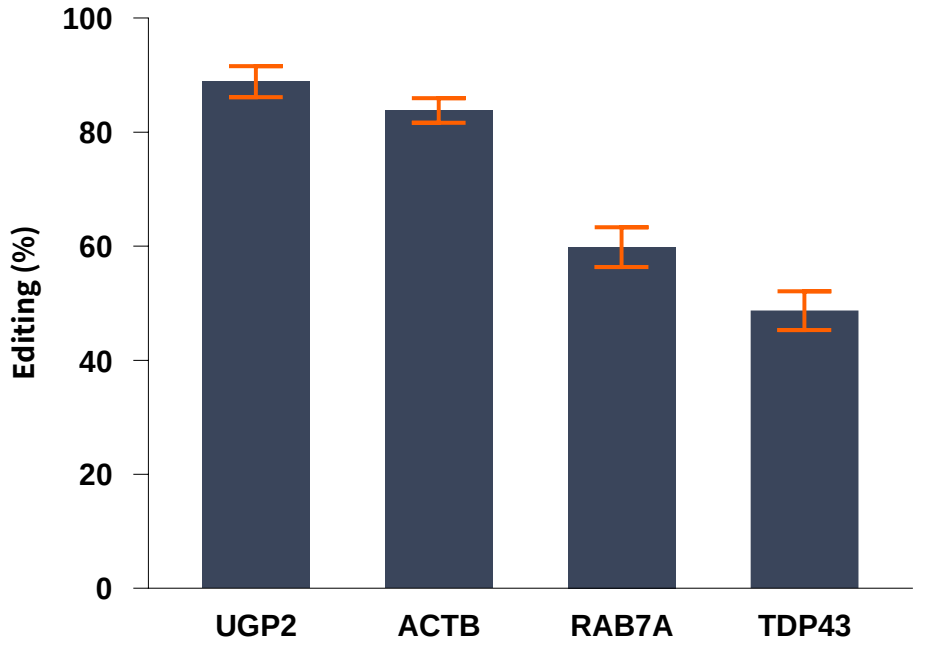
Primary Mouse Hepatocytes[1]

>80% editing achieved



Patient-derived Neuroblastoma Cells

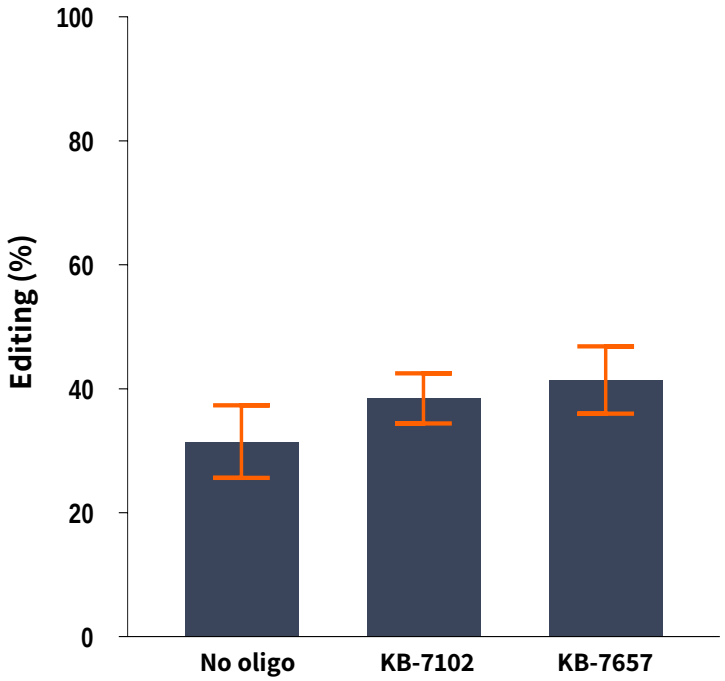
>45% editing achieved



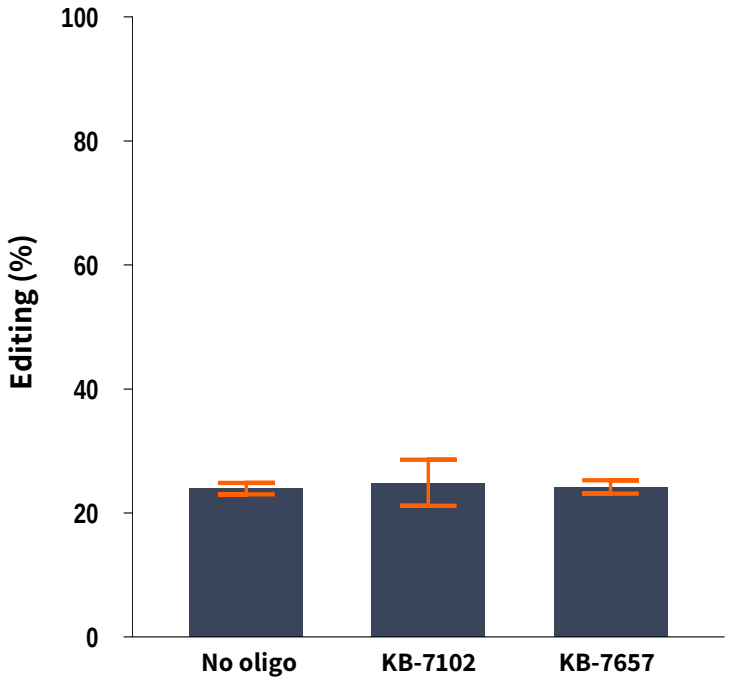
[1] SERPINA1 data is from PiZ primary mouse hepatocytes and ACTB, RAB7, and UGP2 data is from wild-type primary mouse hepatocytes (PMH)

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

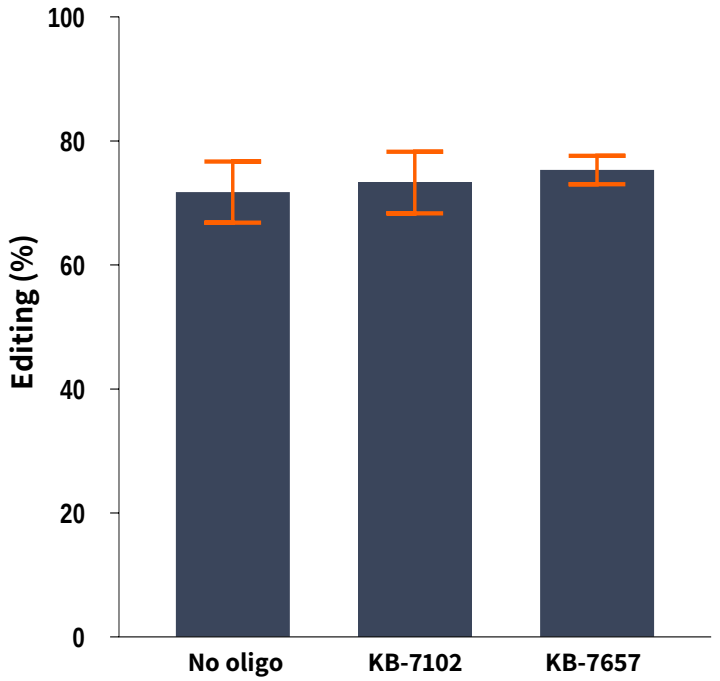
Endogenous site: COG



Endogenous site: COPA



Endogenous site: AJUBA



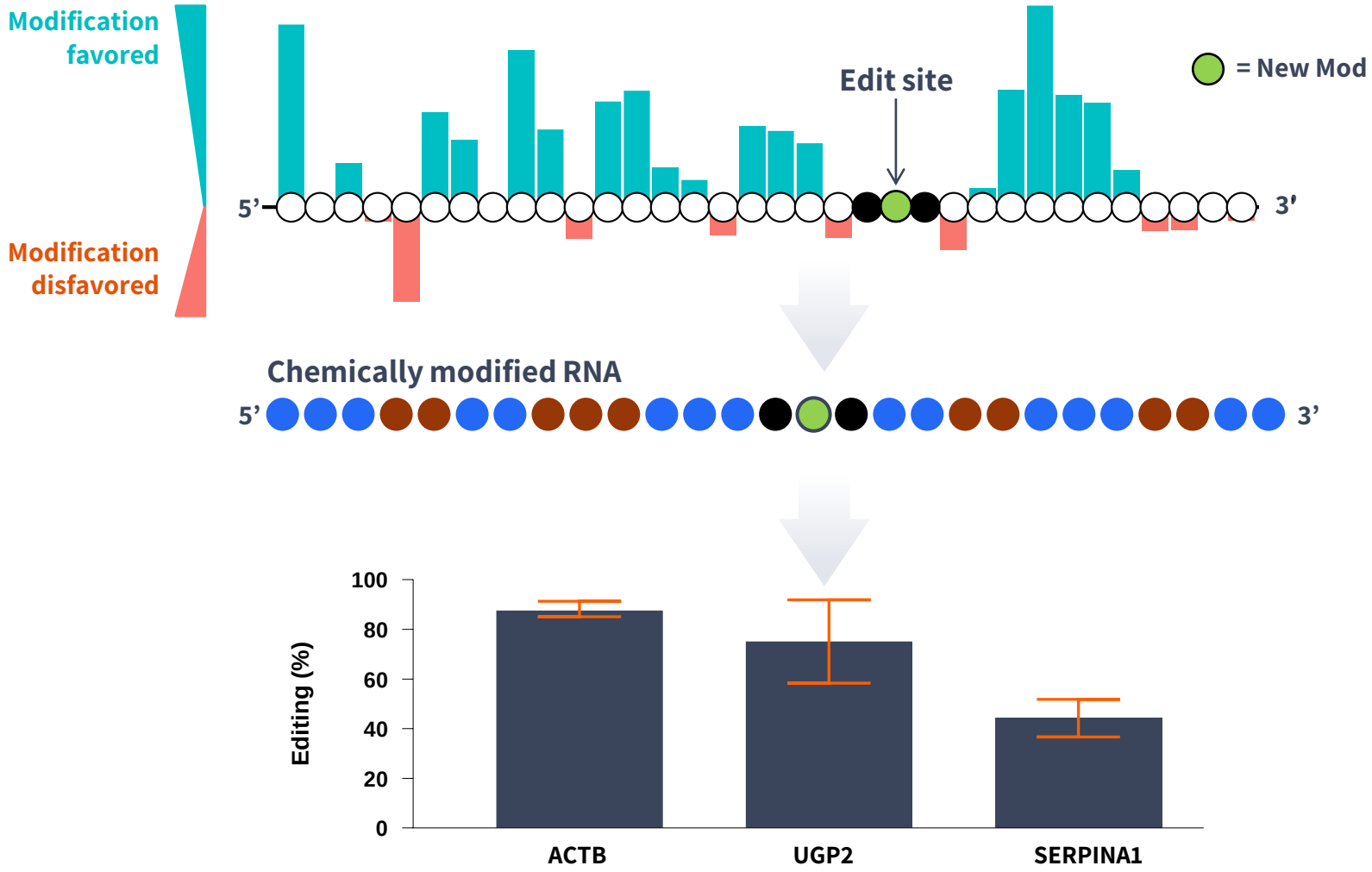
Note: KB-7102 -Target: Rab7; KB-7657 – Target SERPINA1
Ref: Edits on serotonin receptor lead to 24 different isoforms: Brenda Bass et. al. Nature Comm. 2011; 2: 319.; COG & COPA are edited by ADAR2 primarily. Tenen, D. J. et. al. Blood 2023; 141; 3078, AJUBA is edited by ADAR1 only, Jin Billy Li et.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

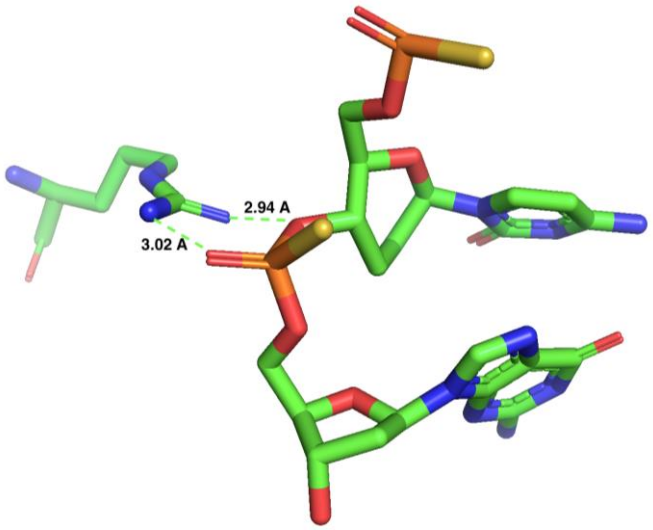
Replicated for multiple targets and sequences at baseline pre-optimization



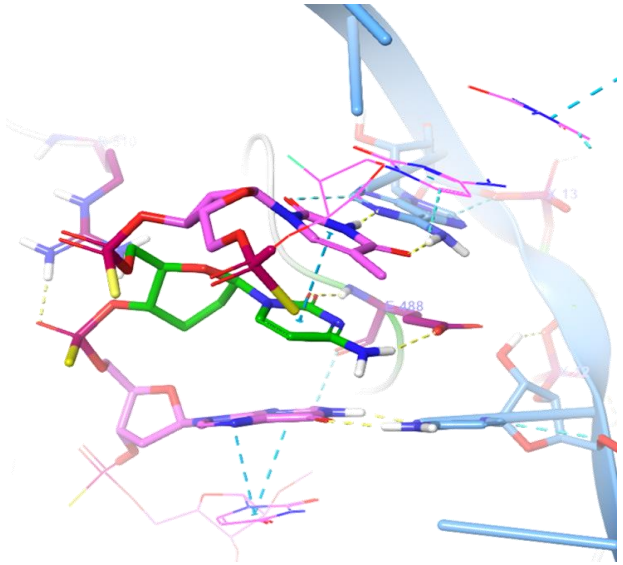
Note: ACTB and UGP2 data from primary mouse hepatocytes (PMH); SERPINA1 data from hepatocyte like cells (zzHLCs)

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

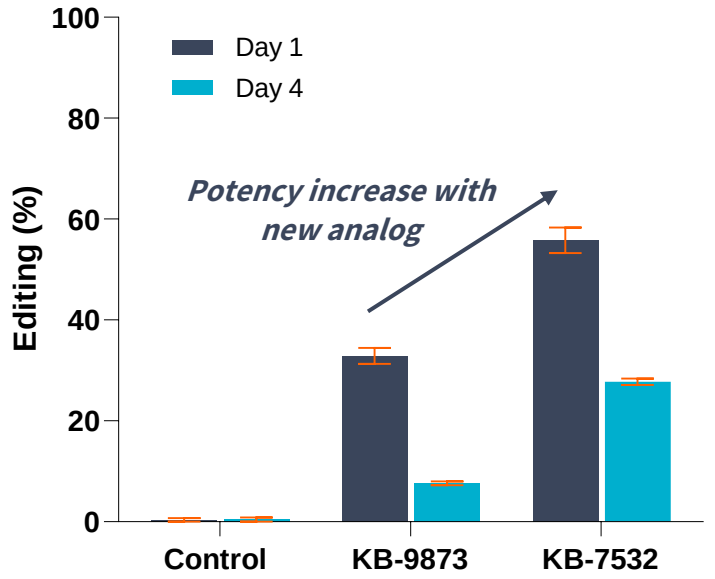
CHORD and target mRNA complexed with ADAR



New chemistry introduces improved potency



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

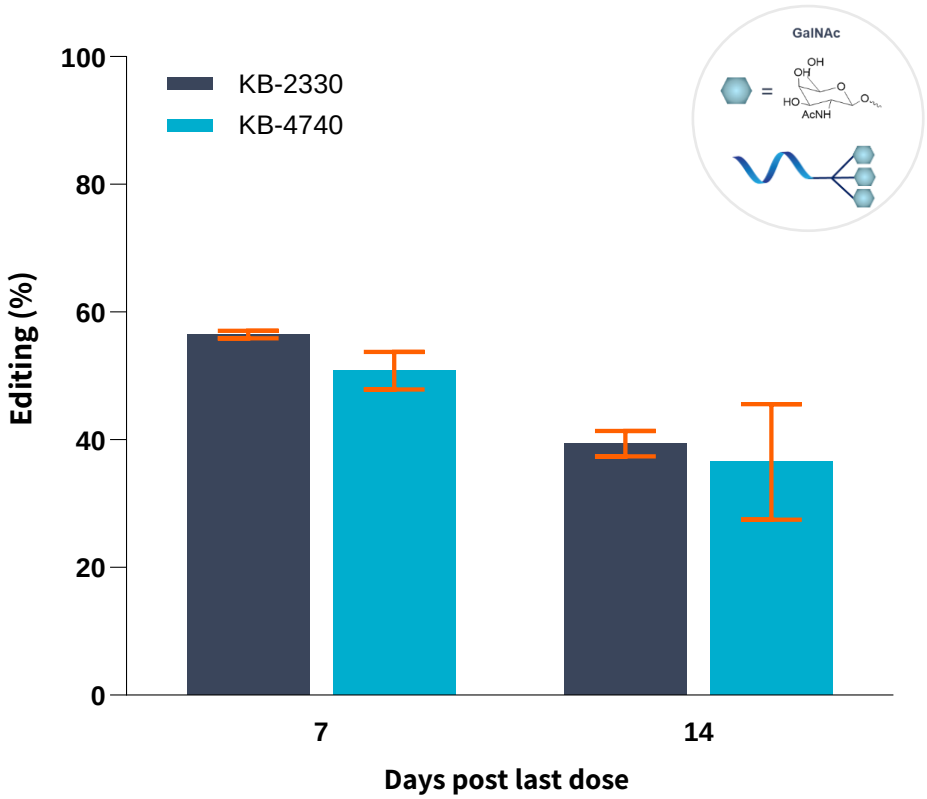


*3mg/kg oligo formulated in MC3 LNP injected IV

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

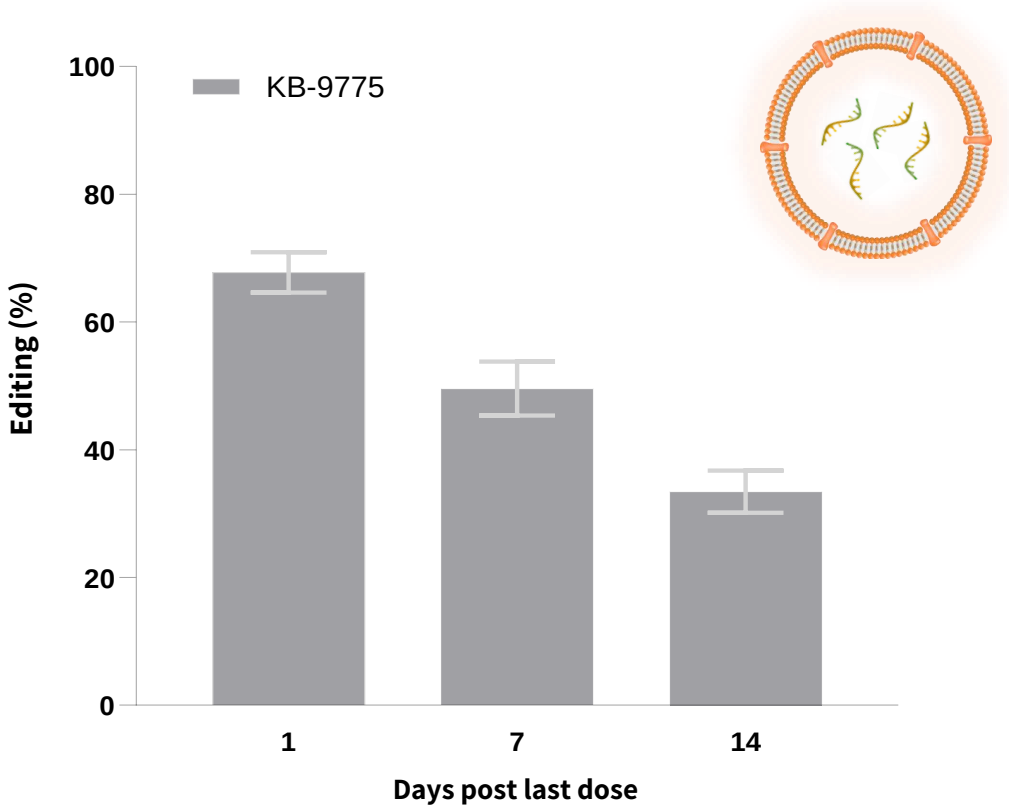
GalNAc (ACTB)

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)

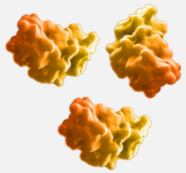
Delivering a Potential Best-in-Class Candidate

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

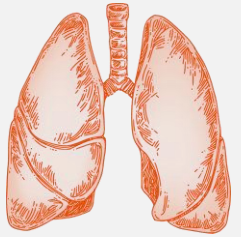
MM Genotype
(normal liver and lung)

Normal levels of M-AAT secreted

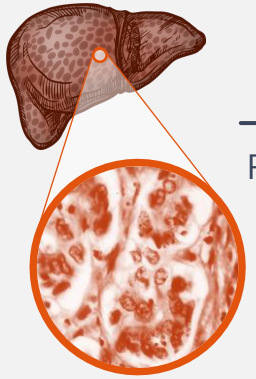
M-AAT



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

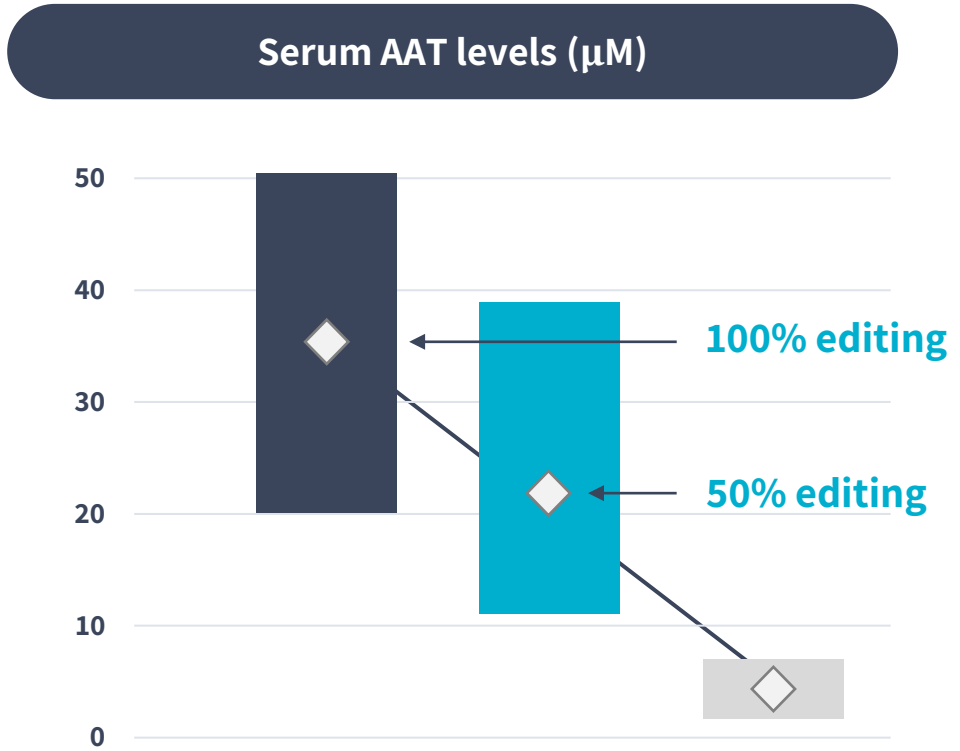
Z-AAT*

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

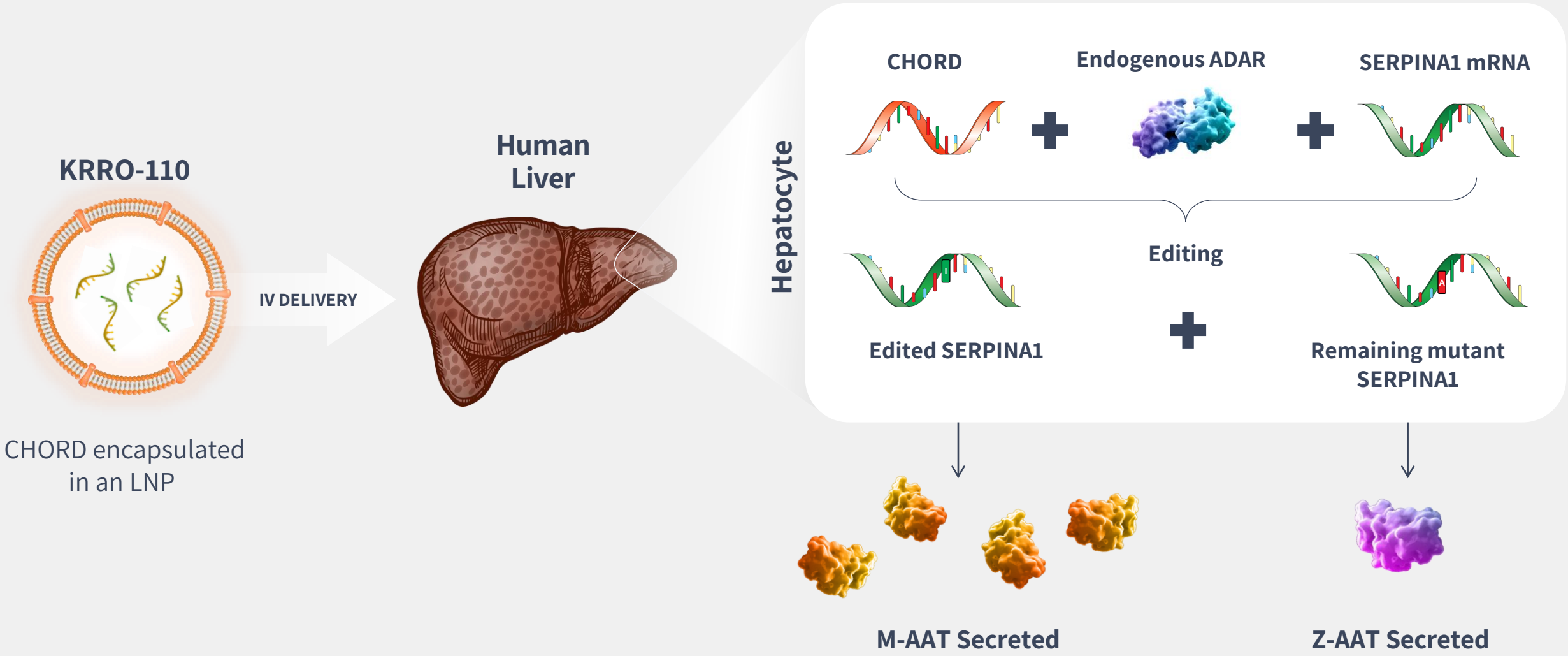
Korro's goal for median editing has potential to reduce lung and liver risk

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



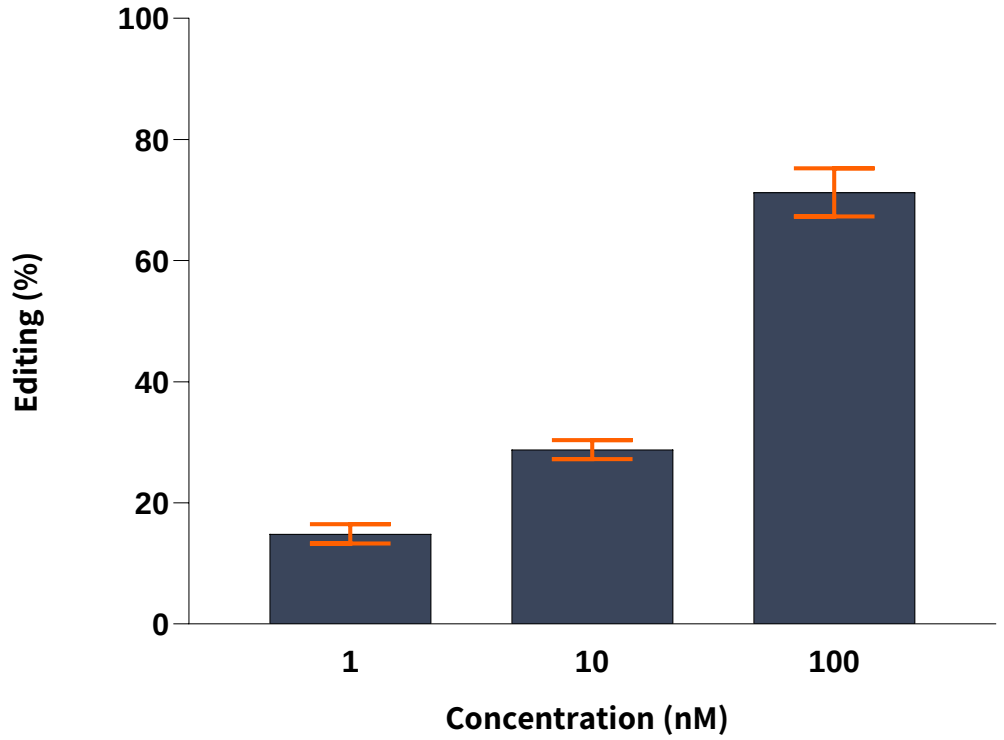
CHORD encapsulated in an LNP

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

KRRO-110 Demonstrated >50% Editing in *In Vitro* Systems with the Z Genotype

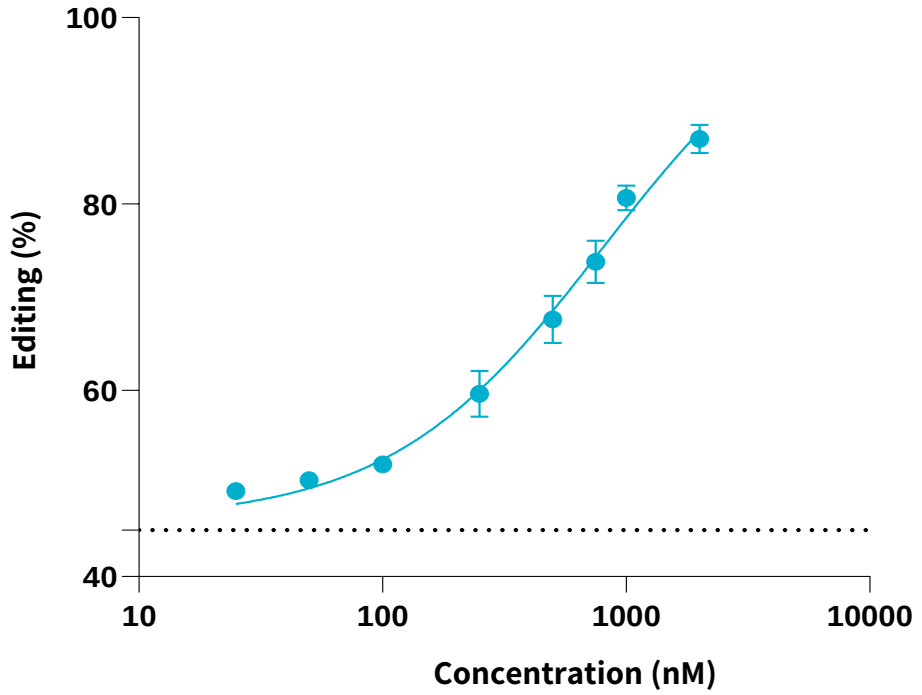
Editing in hepatocyte like cells (HLCs)[1]

KRRO-110 Transfection +IFN



Editing in human MZ hepatocytes[2]

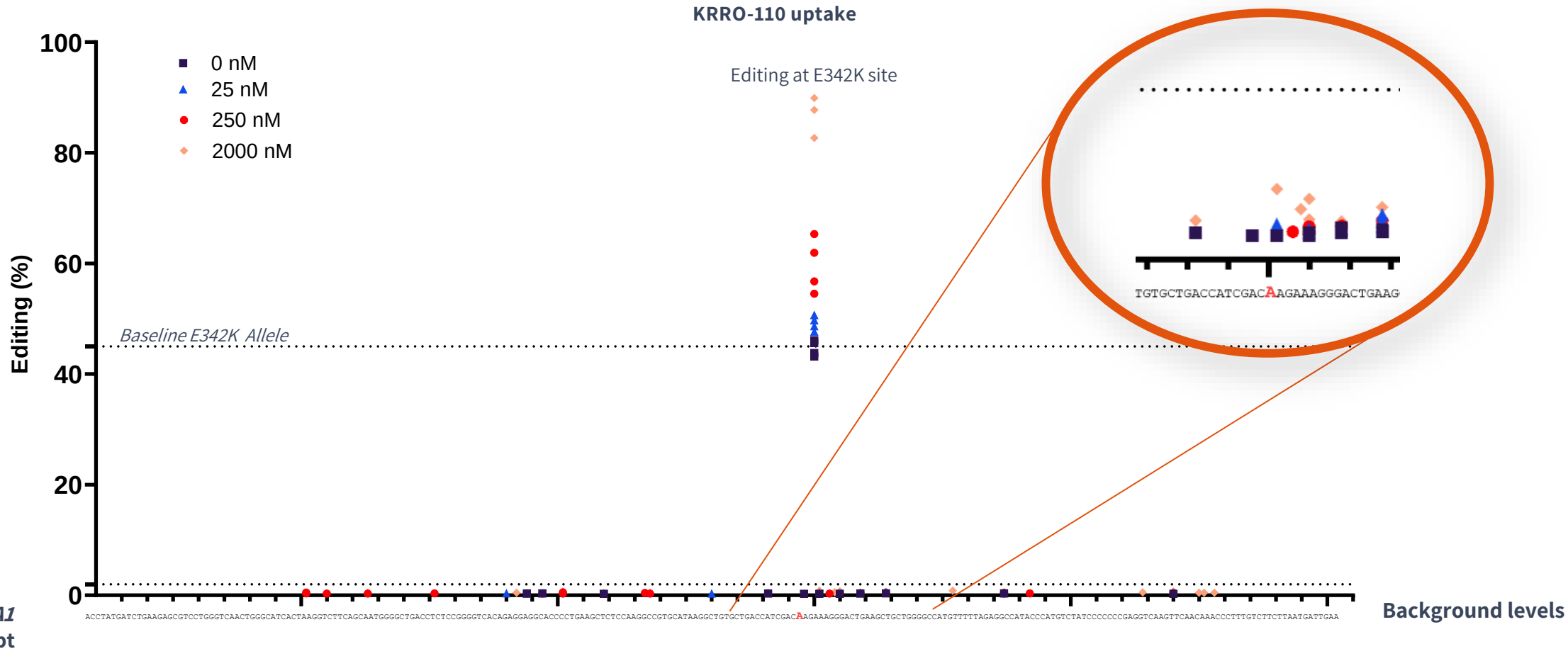
KRRO-110 uptake



Note: Data represented as average values +/- SEM
[1] HLCs derived from ZZ patient, transfected with RNAiMAX with 1U/uL of IFN, editing measured 48-hours post transfection via amplicon-seq
[2] Primary hepatocytes from MZ donor, free-uptake of LNP, editing measured 48-hours post uptake via amplicon-seq

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

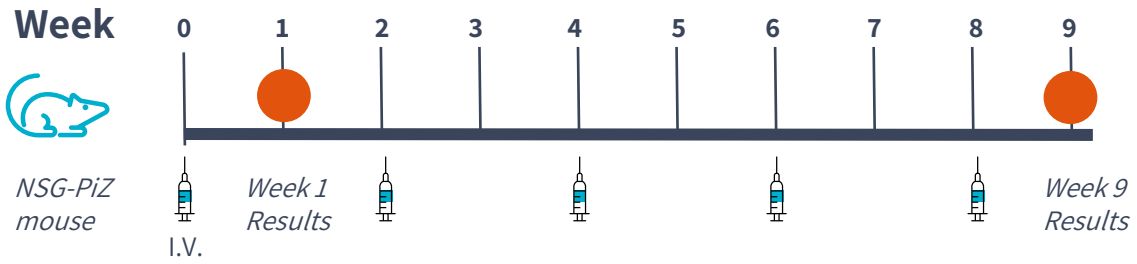
MZ Primary Human Hepatocytes*



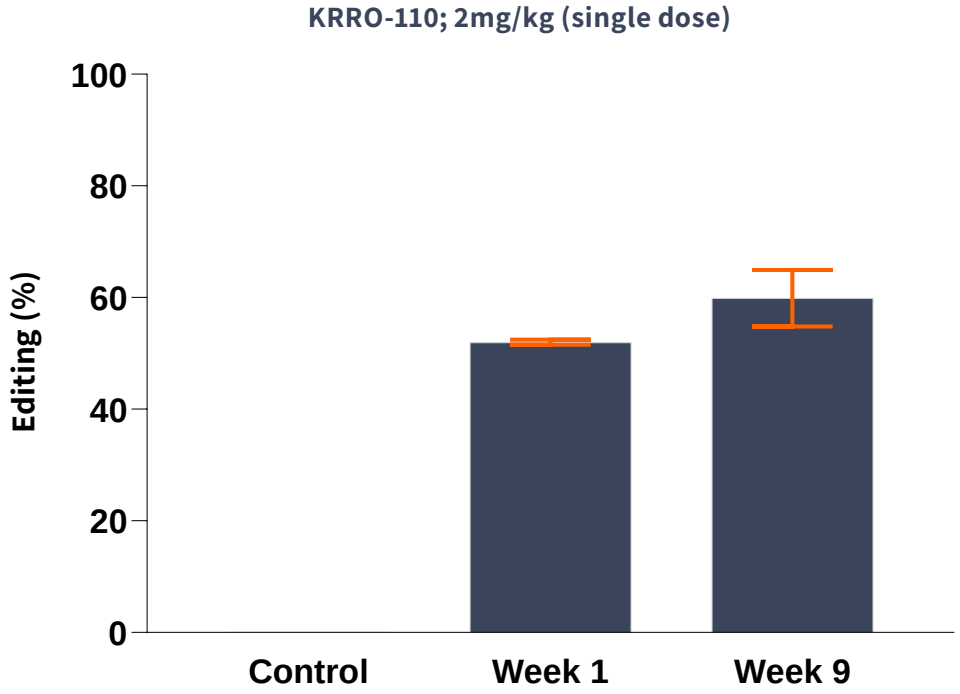
*Note: Each data point represents one experimental replicate with statistically significant editing above sequencing error

Achieved >50% Editing in Human Transgenic Mouse Model of Z Genotype with a Single Dose

Study design



Editing in NSG-PiZ mouse



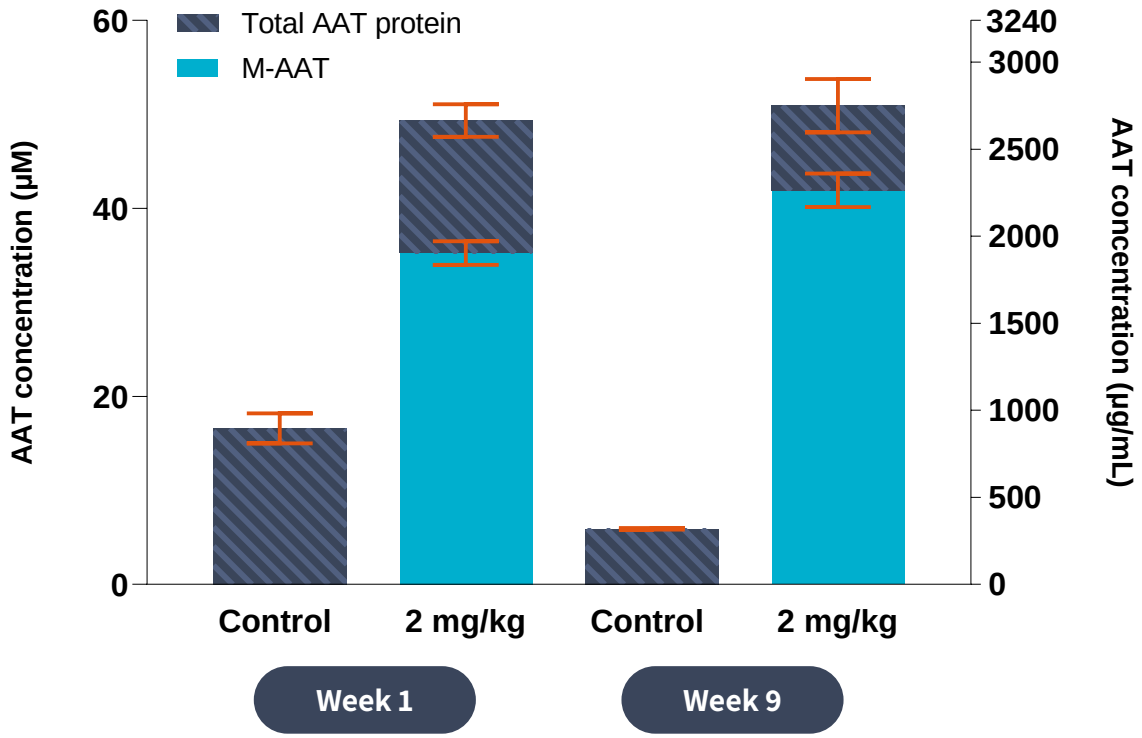
Well-tolerated in mice toxicity studies at 5 mg/kg

Note: Data represented as average values (n=3) +/- SEM
Similar results obtained in C57BL/6-PiZ mice licensed from Dr. Jeff Teckman

Secretion of Functional AAT (~50uM) as Early as 7 Days Post-Single Dose

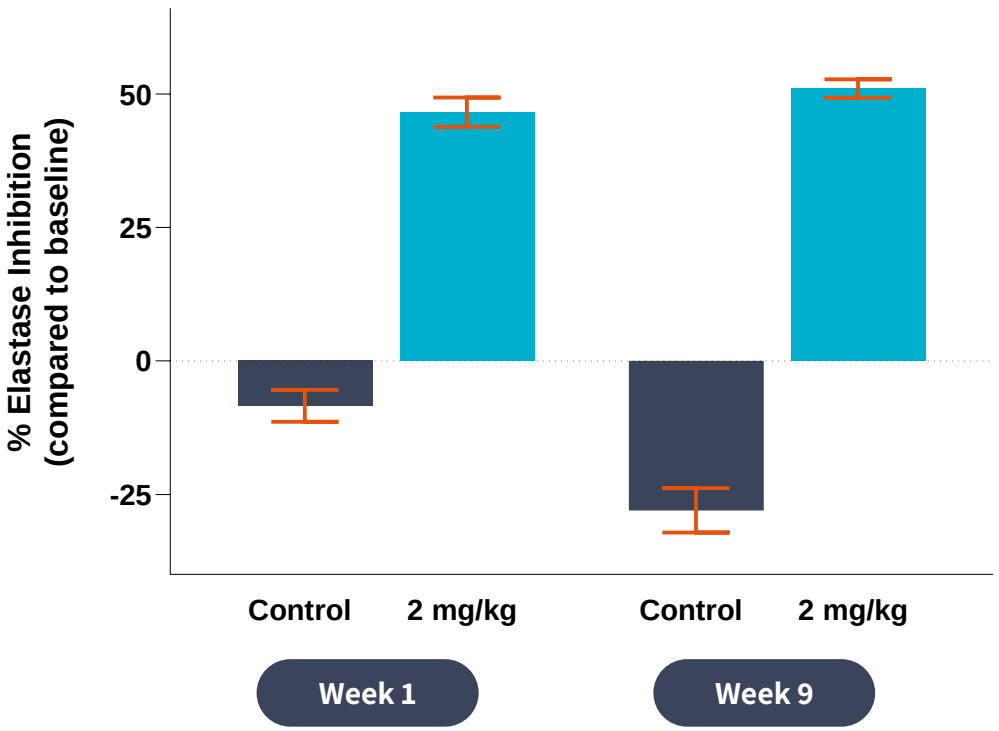
Serum human-AAT concentration

KRRO-110; 2mg/kg (single dose)



NSG-PiZ mice elastase inhibition

KRRO-110; 2mg/kg (single dose)



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

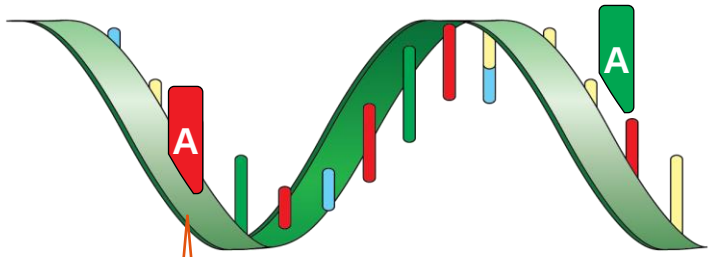
Editing *De Novo* Adenosine on Cyno SERPINA1 to Elucidate Editing in Higher Species

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)

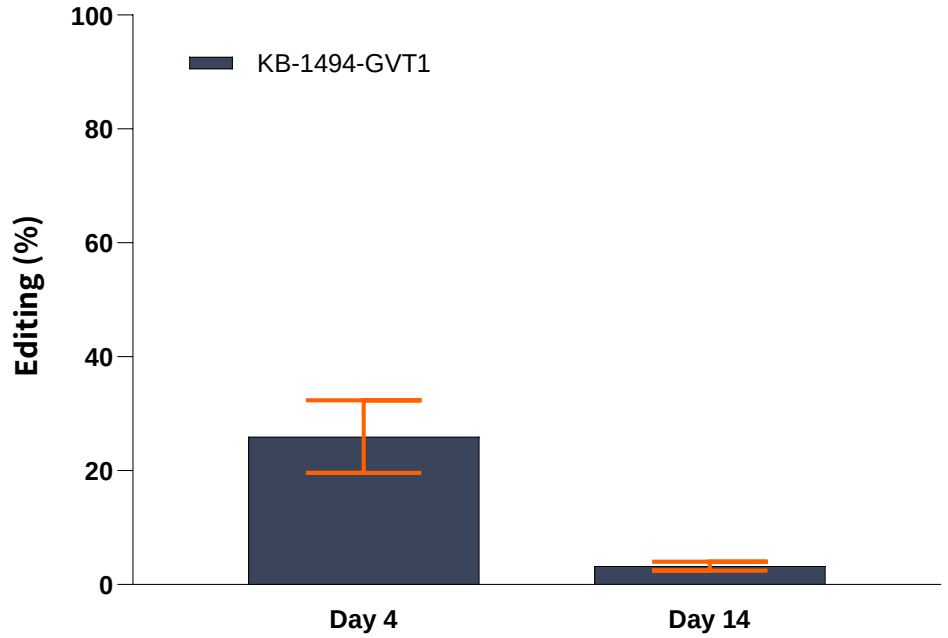
Utility in PiZ mouse and in NHPs

Edited protein detected

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

Editing in C57BL/6-PiZ mice (%)

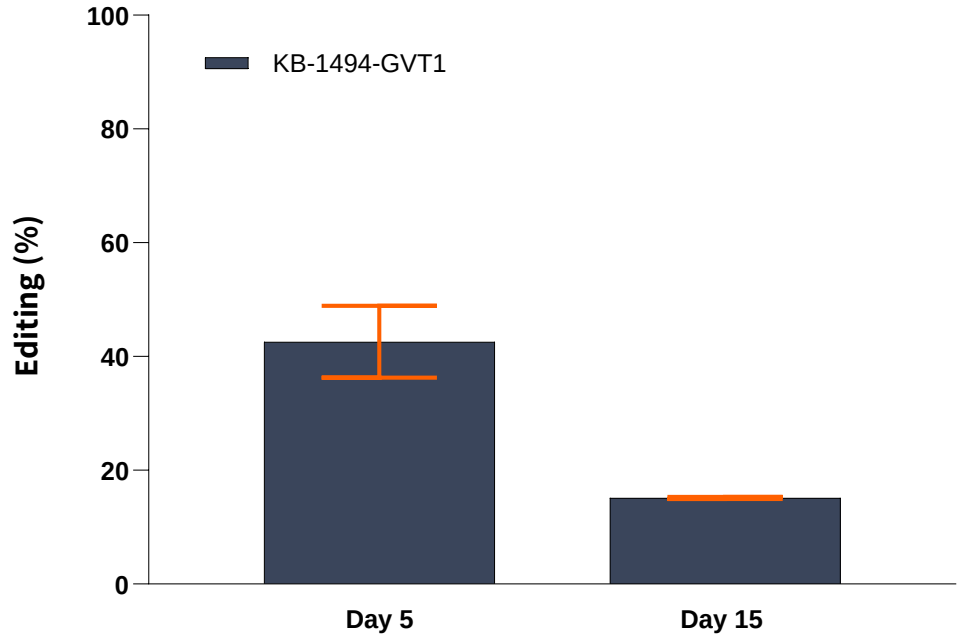
2mg/kg (single dose)



Surrogate Target Site: SERPINA1

Editing in NHPs (%)

2mg/kg (single dose)



Surrogate Target Site: SERPINA1

Note: Data represented as average values +/- SEM

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

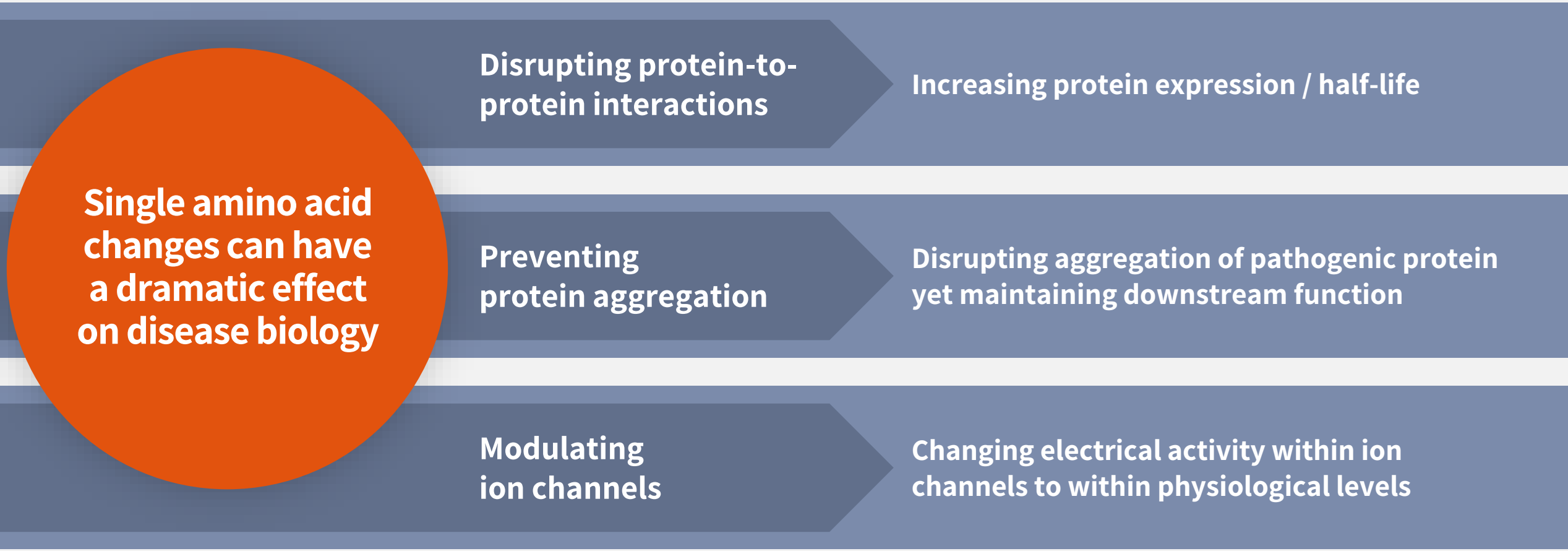
Preclinical data package supports goal to submit regulatory filing in 2H 2024 and enable FIH study[1]

[1] Subject to submission of an Investigational New Drug (IND) application to the U.S. Food and Drug Administration (FDA) or similar application with regulatory agencies in other geographies and subsequent authorization to proceed

Creating *De Novo* Proteins

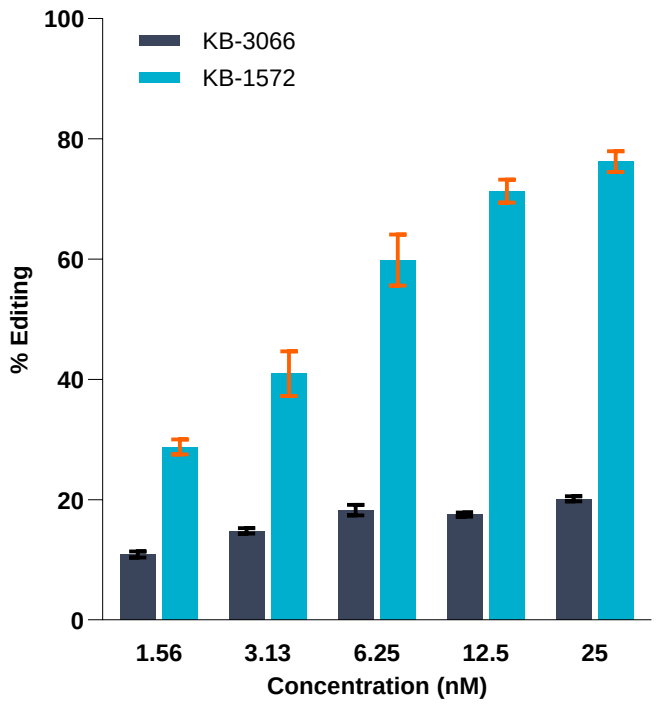
Going Beyond “Repairing" a Single Pathogenic Point Mutation

Creating *De Novo* Protein Variants to Modulate Protein Function

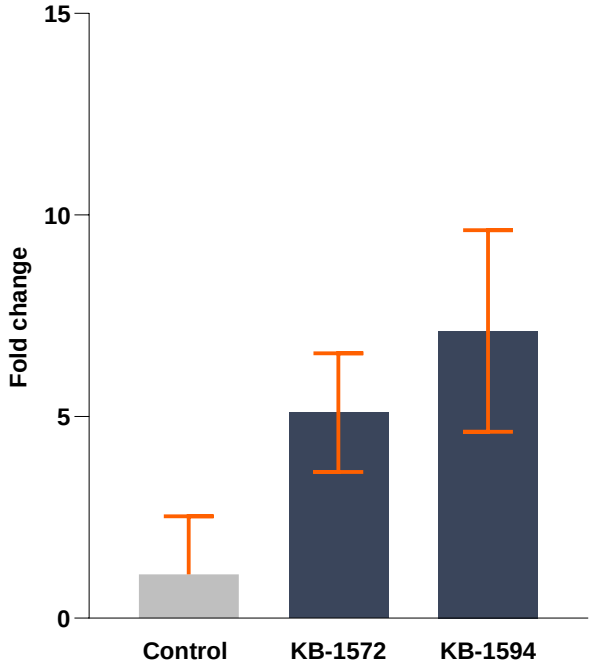


Activation of Transcription Factor (TFX) by Creation of *De Novo* Protein…

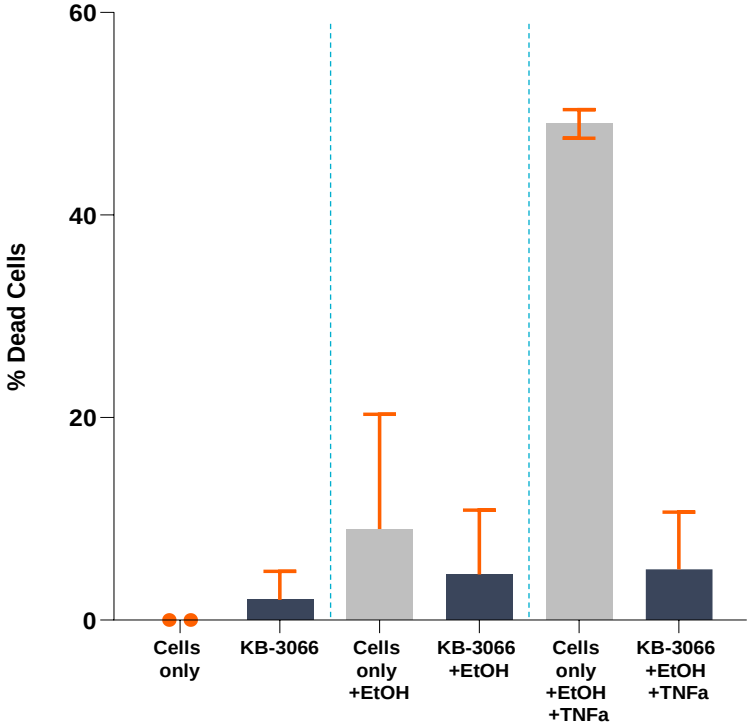
In vitro editing of normal TFX in Hep3B cells[1]



Downstream target gene expression _in vivo_ in mouse liver[2]



TFX variant rescues Hep3B-CYP2E1 cells from cytotoxicity[3]

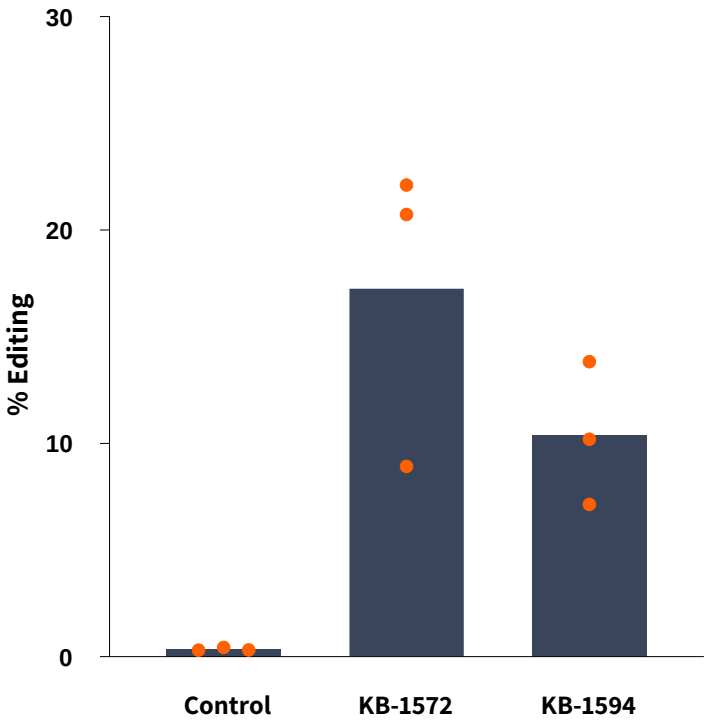


[1] Hep3B cells transfected with RNAiMAX at the indicated concentrations with two targeting oligos, editing measured 48-hours post transfection via amplicon-seq
[2] Wild type mice dosed with LNP-targeting oligos at a concentration of 3 mg/kg, gene expression measured via quantitative PCR from liver harvested 1 day post dose
[3] Hep3B-CYP2E1 cells transfected with RNAiMAX at the indicated concentrations with two targeting oligos, cell viability measured 48-hours post transfection via CellTiter-Fluor Cell Viability Assay from Promega

…and Sustained Downstream Activity in NHPs Lasting Longer than 21 Days

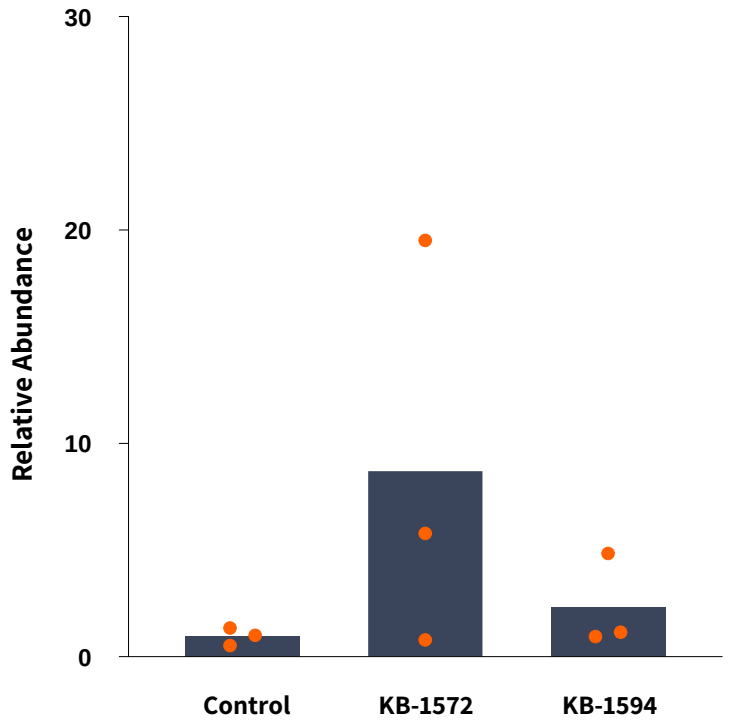
Editing in liver cores (Day 3)

2mg/kg; QWx3



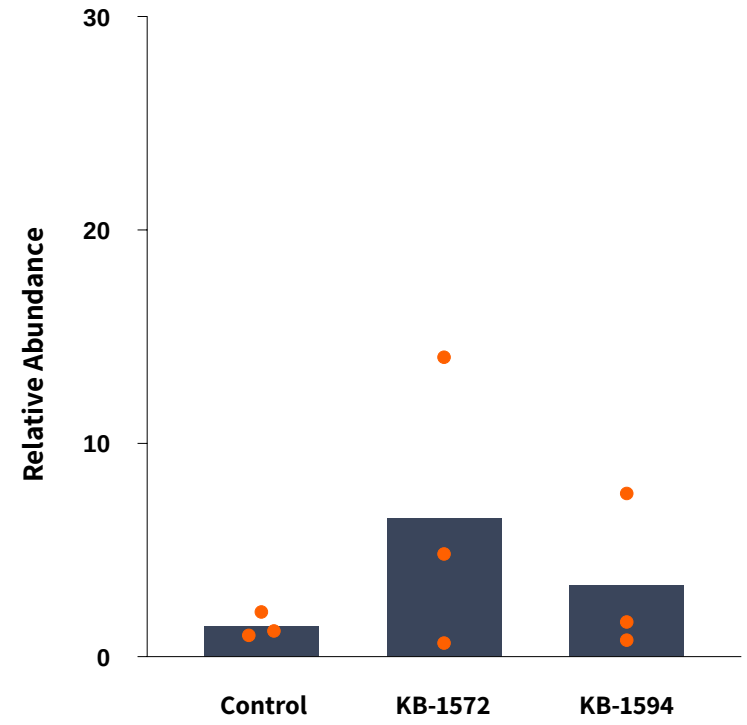
Presence of TFX variant (Day 3)

2mg/kg; QWx3



Durable presence of TFX variant (Day 21)

2mg/kg; QWx3



Durable presence of protein variant correlates with sustained downstream expression of biomarker*

*More expansive dataset not shown

The Team

Experienced Management Team with Proven Track Record

Ram Aiyar, PhD
Chief Executive Officer

Steve Colletti, PhD
Chief Scientific Officer

Vineet Agarwal
Chief Financial Officer

Todd Chappell
Chief Operating Officer

Shelby Walker
SVP, General Counsel

Stephanie Engels
SVP, HR People and Culture

Venkat Krishnamurthy, PhD
SVP, Head of Platform

CORVIDIA
MERCK
J.P.Morgan
CombinatoRx
CRISPR THERAPEUTICS
moderna
Lilly
Sofinnova partners
zymergen
HealthCare VENTURES
GINKGO BIOWORKS
Shire
AstraZeneca
Johnson & Johnson
LODO THERAPEUTICS
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

David Lucchino
Co-founder, and ex-CEO, Frequency Therapeutics

Ram Aiyar, Ph.D.
President and CEO

kv

Alnylam PHARMACEUTICALS

Carrick therapeutics

ATLAS VENTURE

NEA

FREQUENCY THERAPEUTICS

CORVIDIA

Intellia THERAPEUTICS

FAZE MEDICINES

TESARO

IFM THERAPEUTICS

The Medicines Company

entrega

Sofinnova partners

MILLENNIUM PHARMACEUTICALS, INC

MedImmune

Johnson & Johnson

Uniquely Positioned to Expand the Frontiers of Genetic Medicines through RNA Editing

Built an experienced team with a proven track record in genetic medicines

Built an oligonucleotide-based approach (OPERA™) to affect a single base edit on RNA (efficient, specific and transient)

Nominated a candidate (KRRO-110) for alpha-1 antitrypsin deficiency (AATD) with potential for best-in-class profile

Continuing to build a unique, wholly-owned pipeline with broad opportunities in rare and common diseases

Strong balance sheet with cash runway into ’26 enabling a potential interim clinical readout for AATD in 2H ‘25[1,2]

[1] Subject to submission of an Investigational New Drug (IND) application to the U.S. Food and Drug Administration (FDA) or similar application with regulatory agencies in other geographies and subsequent authorization to proceed
[2] Korro Bio closed reverse merger and financing transaction on 11/3/23 with cash, cash equivalents and short-term investments of approximately $170.0 million

Create transformative genetic medicines for diseases with high prevalence