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Developing Transformative Genetic Medicines for Rare and Highly Prevalent Diseases



Editing RNA

Without modifying DNA



Modular Platform

Delivering drug to multiple cell types

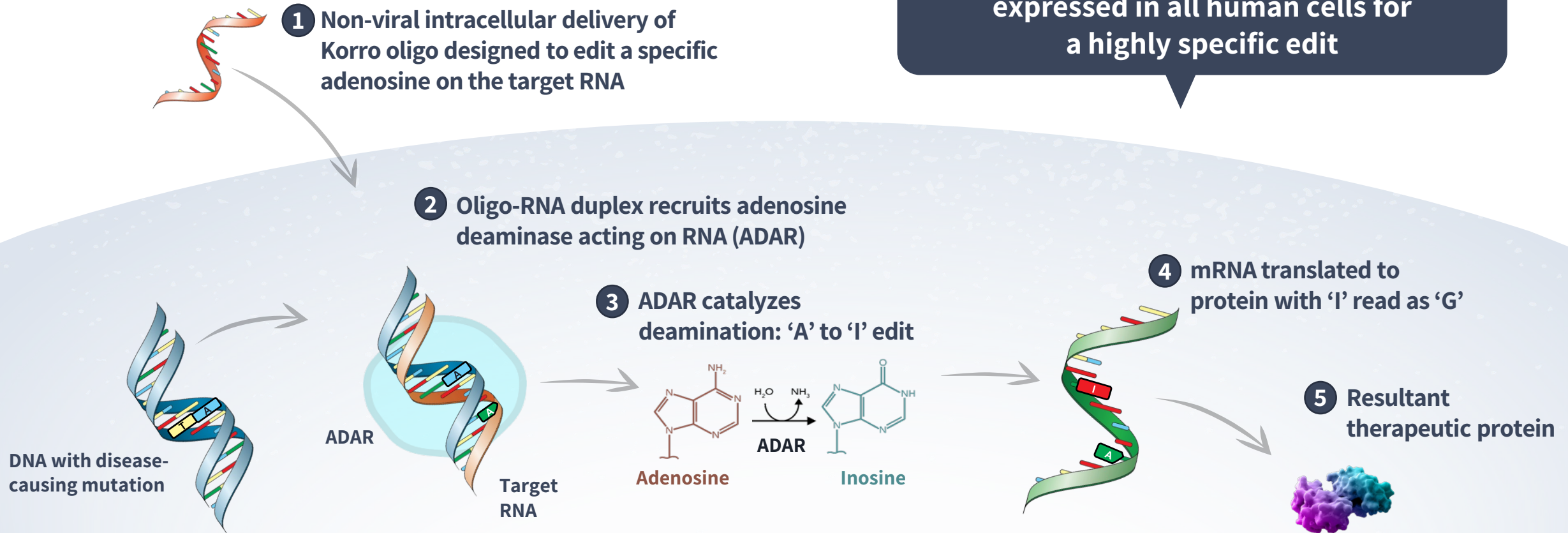


Activating Biological Pathways

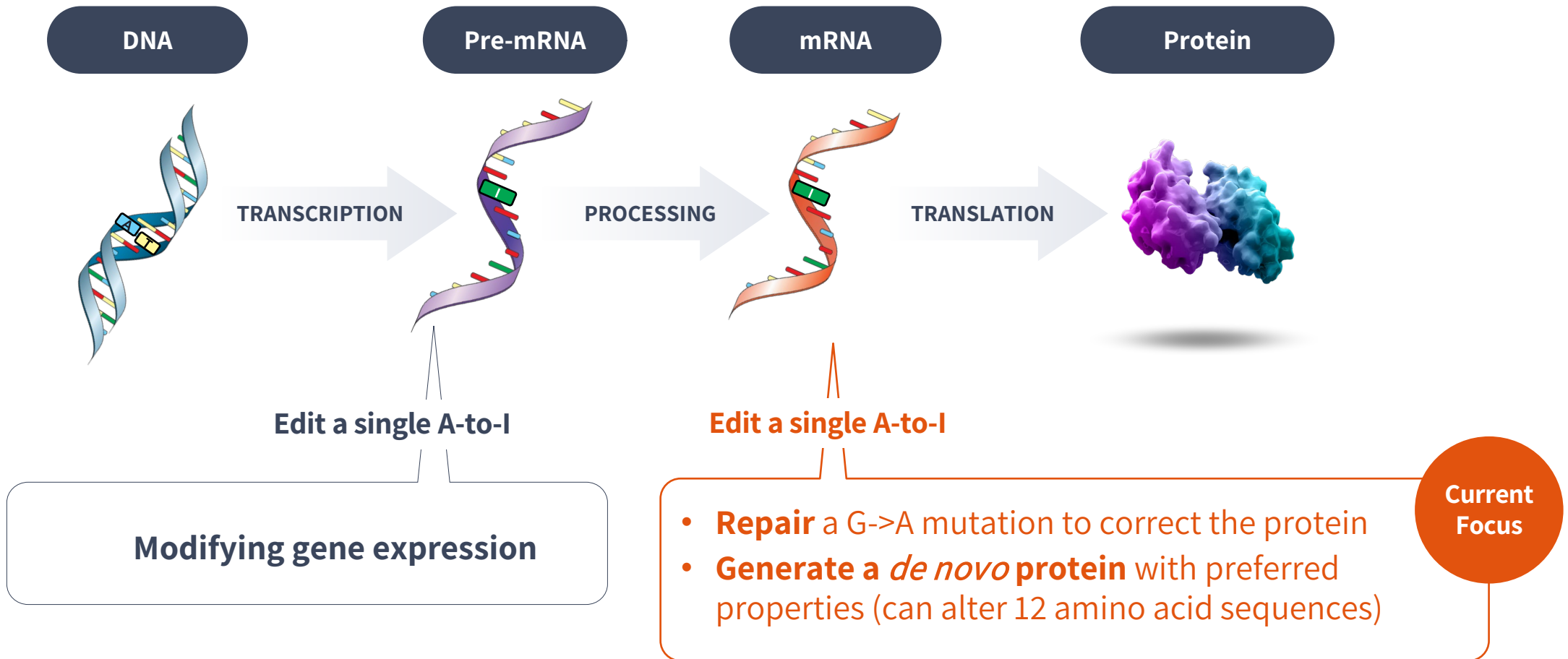
Learning from genetics

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

Harnessing an endogenous enzyme, ADAR, expressed in all human cells for a highly specific edit



RNA Editing Enables Potential for High Impact in Broad Range of Disease Areas

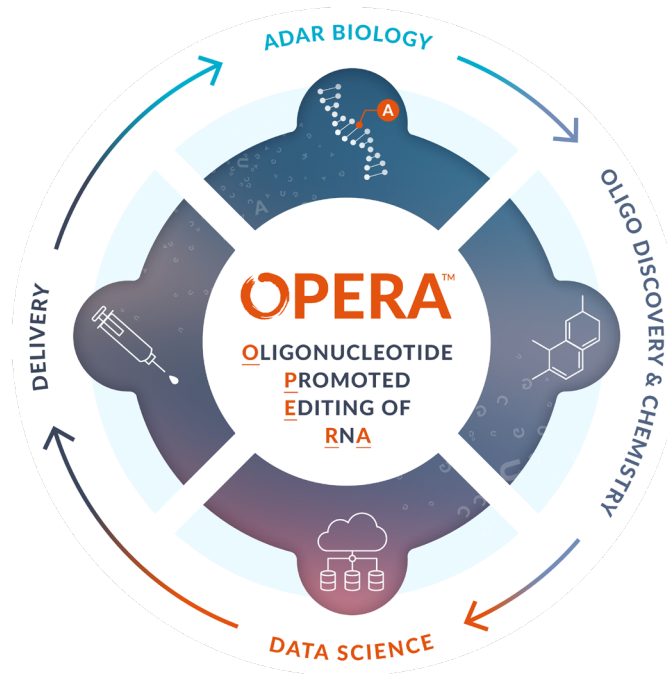


Human genetics guiding the possibilities

OPERA™: Our Approach for RNA Editing to Generate Product Candidates

Expertise in ADAR biology
driving potency and translation

Leveraging known Delivery
driving derisked access to
indications



Expertise in Chemistry
driving potency and drug designs

Expertise in Machine Learning
driving efficiency and Target ID

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 - Interim data in 2H '25		REWRITE Clinical Trial	
<i>De novo</i> protein to inhibit degradation	Rare metabolic disorder	GalNAc (SC)	Undisclosed	DC in '25			
<i>De novo</i> protein to overcome LoF and GoF ¹	Amyotrophic lateral sclerosis	Undisclosed	TDP43				
<i>De novo</i> protein to modulate currents	Subsets of pain	Undisclosed	Na _v 1.7				
Repairing a pathogenic variant	Parkinson's disease	Undisclosed	LRRK2				
Undisclosed	Cardiometabolic	Undisclosed	Up to 2 Targets				



KRRO-110 program with first participant dosed in January '25

¹De 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)

Korro is Poised for Value Creation Through Multiple Milestones in 2025

2024 Accomplishments

- ✓ Initiated KRRO-110 study (REWRITE)
- ✓ Advanced multiple discovery targets
- ✓ Announced partnership with Novo Nordisk
- ✓ Closed a \$70M PIPE
- ✓ Appointed key Board and team members

2025 Anticipated Milestones

- 🎯 Share Interim clinical data for KRRO-110 from REWRITE study in 2H'25
- 🎯 Nominate a candidate with SC delivery (GalNAc) in Liver in '25 that can create a de novo protein variant
- 🎯 Progress and expand a wholly owned pipeline
- 🎯 Progress partnership with Novo Nordisk in cardiometabolic diseases with high prevalence

Cash runway into 2H'26¹ enables multiple milestones for KRRO-110 and other pipeline programs

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

Our 3-2-1 Strategy Through 2027: Potential for Developing Differentiated Therapies for Patients



Programs in the clinic



Tissues targeted

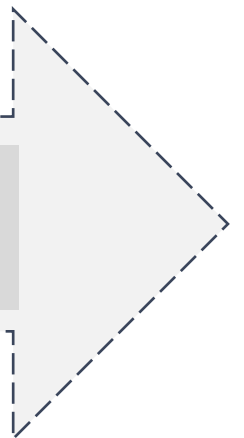


Editing Platform

Validate platform
in AATD representing a \$3B+
market opportunity

Demonstrate outside liver
the validity of our RNA
editing platform

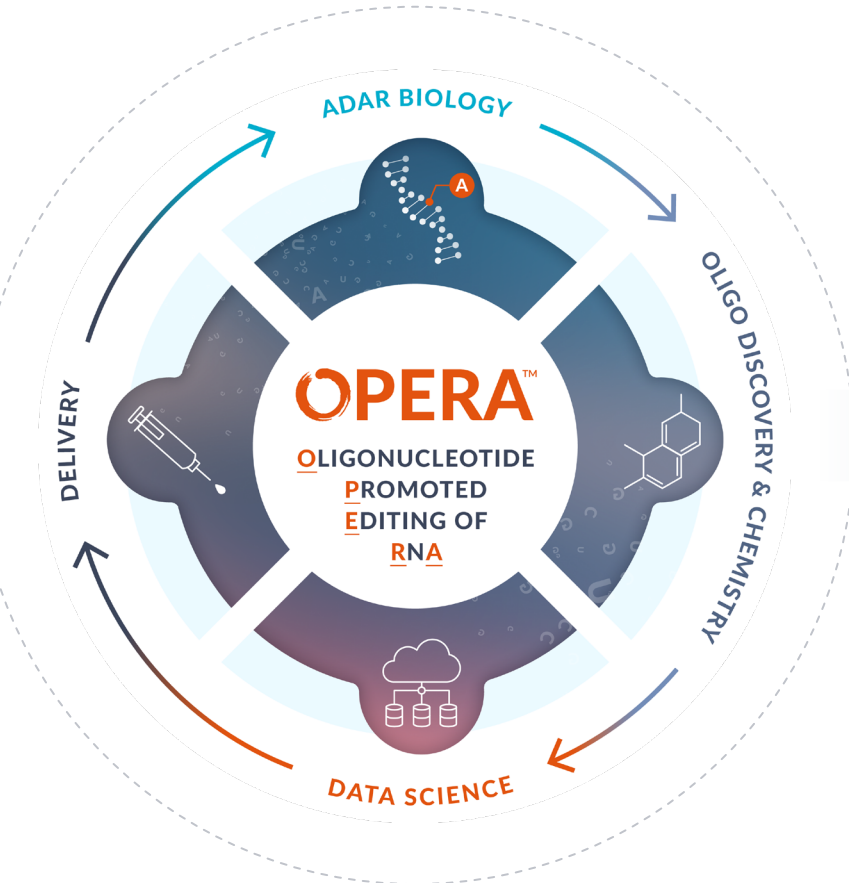
Demonstrate pathway modulation
outside of single nucleotide variant
repair



OPERA™

Our Approach: Oligonucleotide Promoted Eediting of RNA

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



Designed to have...

High target efficiency

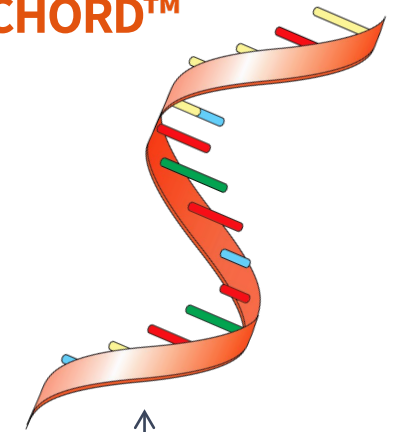
High target specificity

Computational efficiency

Leveraging chemistry

Leveraging delivery

CHORD™

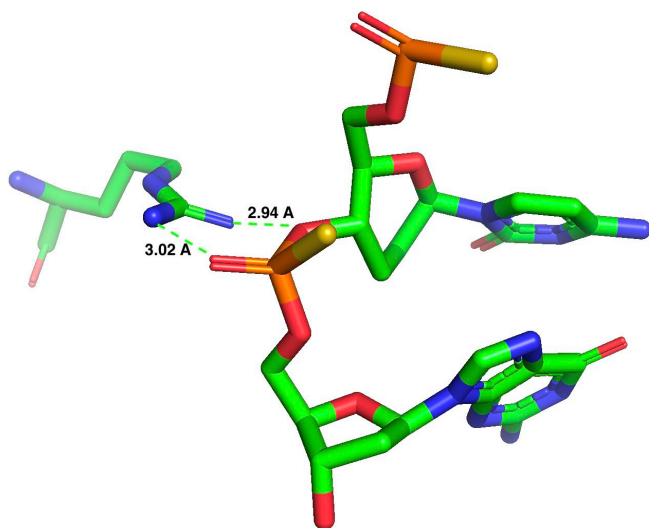


Gen 1.0:

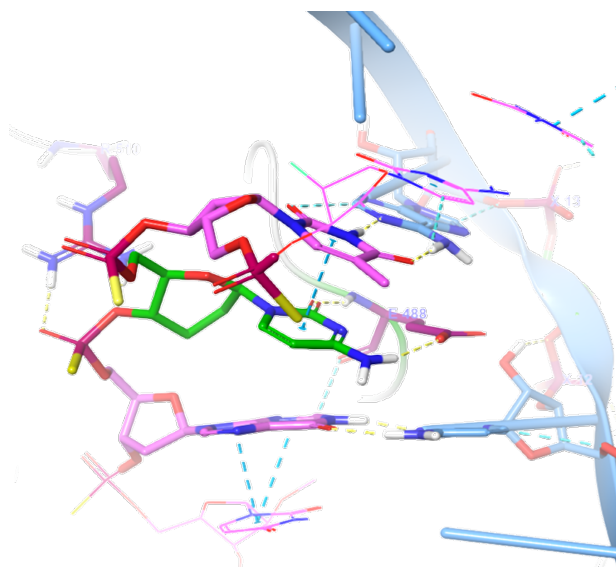
A single-stranded, anti-sense oligonucleotide RNA editor

Structural Biology Insights of ADAR binding Enable Potency Boosts *In Vivo*

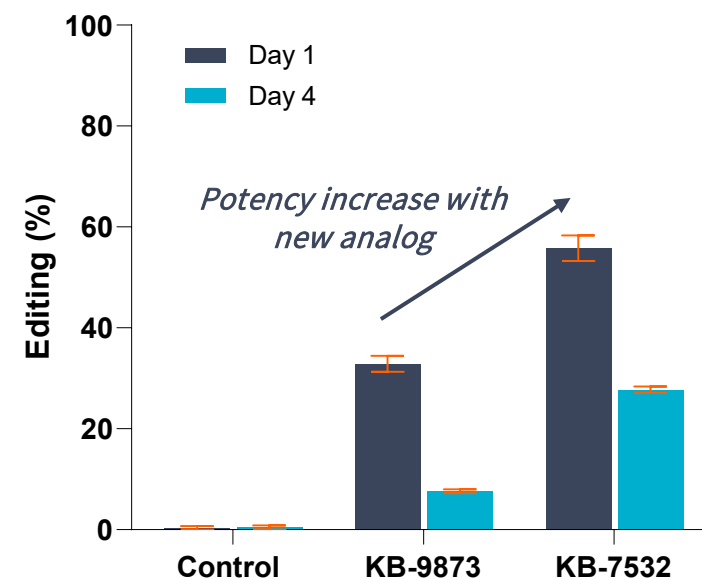
CHORD and target mRNA complexed with ADAR



New chemistry introduces improved potency



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

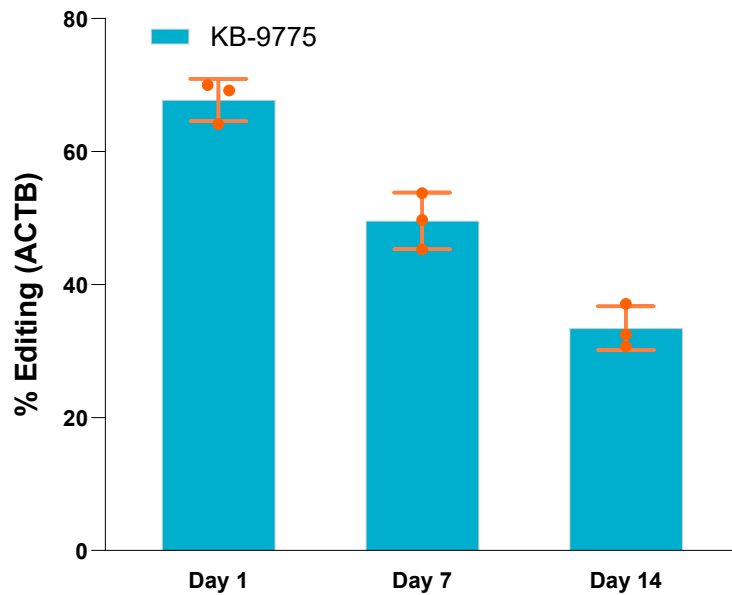


*3mg/kg oligo formulated in MC3 LNP injected IV

Leveraging Validated Delivery Across Tissue Types

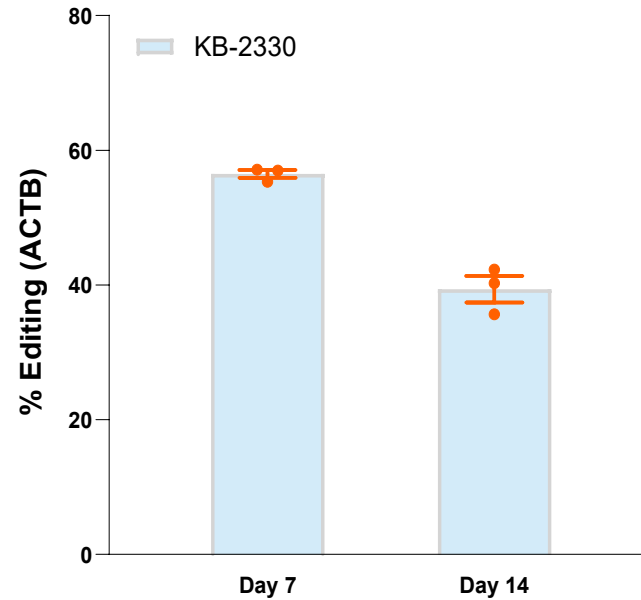
Liver - IV

IV; MC3 LNP; 2 mpk



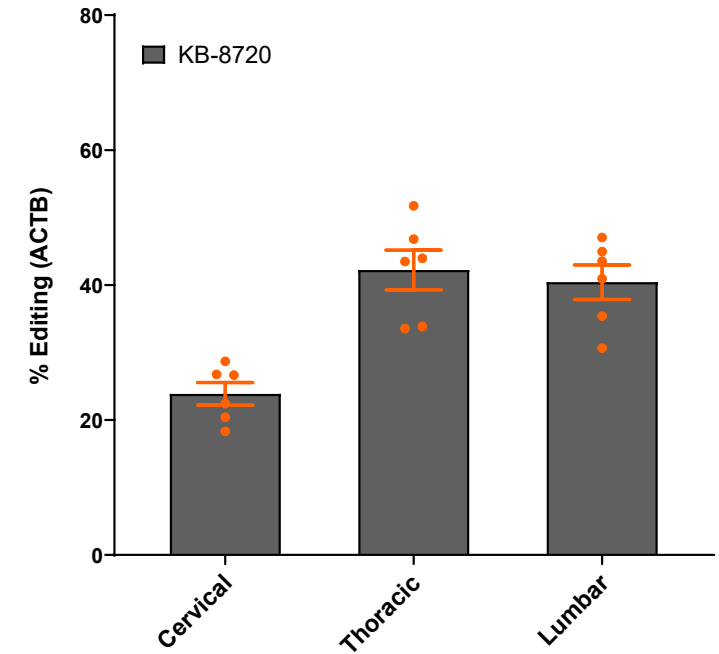
Liver - SC

SC; 10 mpkx5; GalNAc Delivery



CNS - IT

IT Dose (50 ug; Day 4)

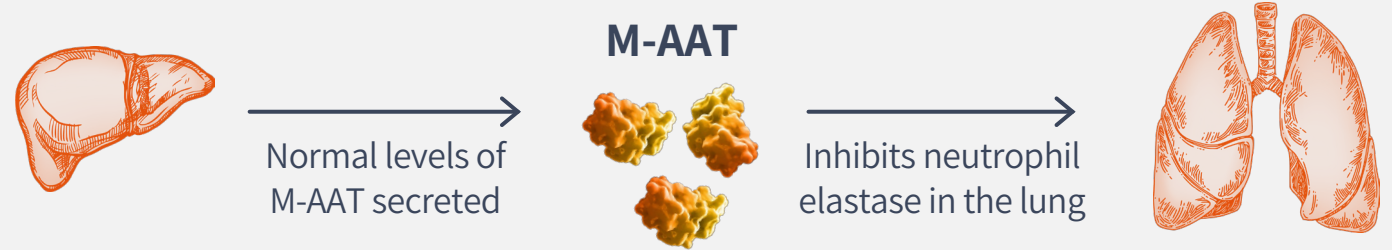


Alpha-1 Antitrypsin Deficiency (AATD)

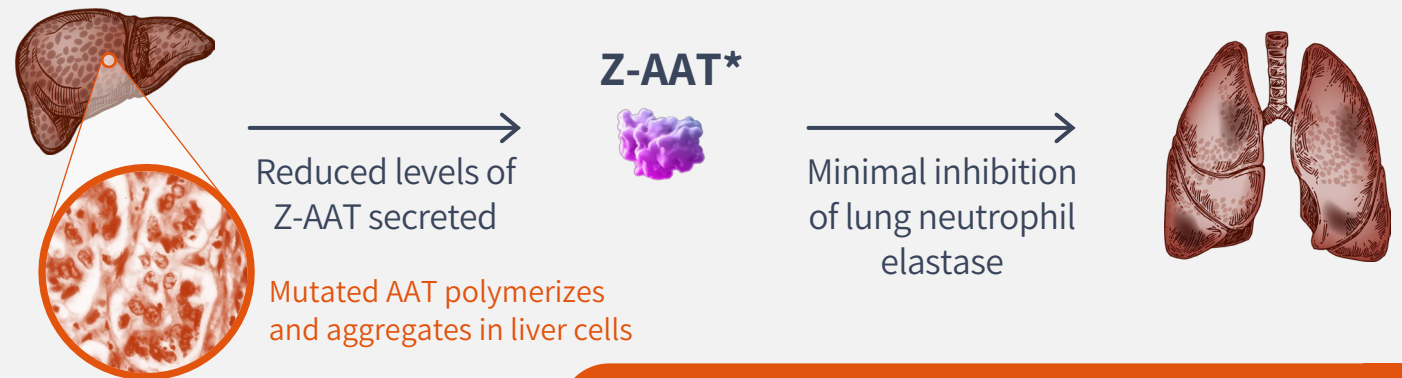
Delivering a Potential Best-in-Class Candidate with KRRO-110

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

MM Genotype (normal liver and lung)



ZZ Genotype (fibrotic liver and decreased lung function)



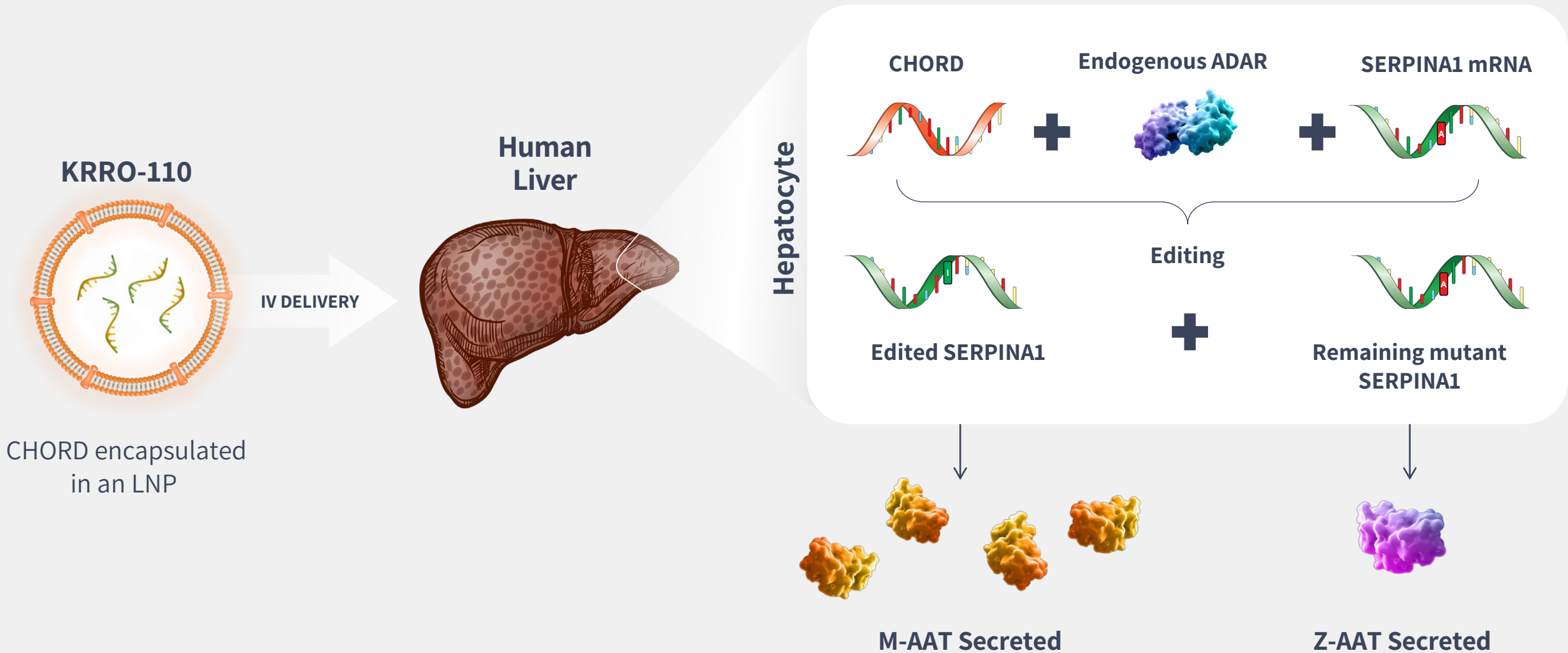
*~100K PiZZ adult patients in U.S. ***

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

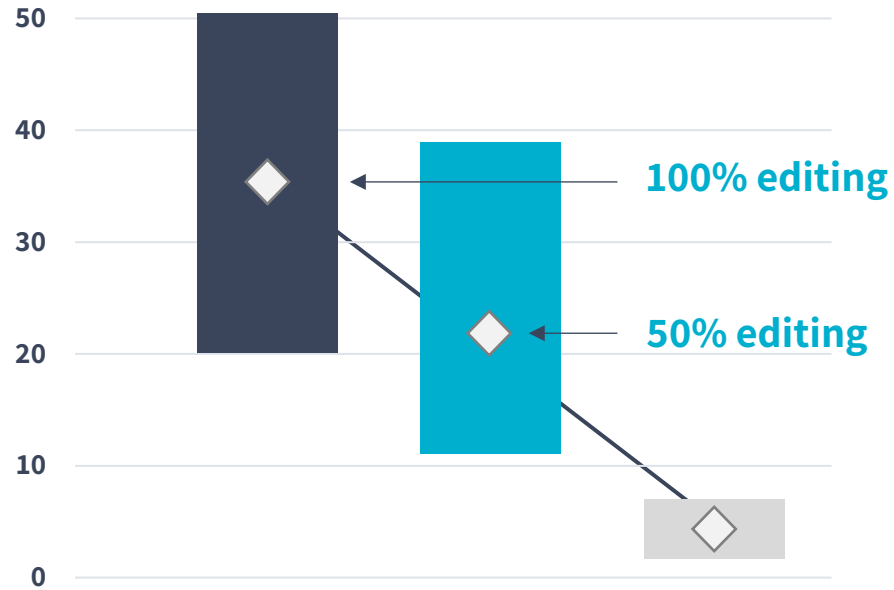
KRRO-110 Designed to Result in M-AAT Secretion and Reduced Pathogenic Z-AAT Protein



CHORD encapsulated in an LNP

KRRO-110 Aims to Restore Therapeutic AAT Levels in PiZZ Patients

Serum AAT levels (μM)



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

◇ = Median AAT for genotype

Korro's Objectives

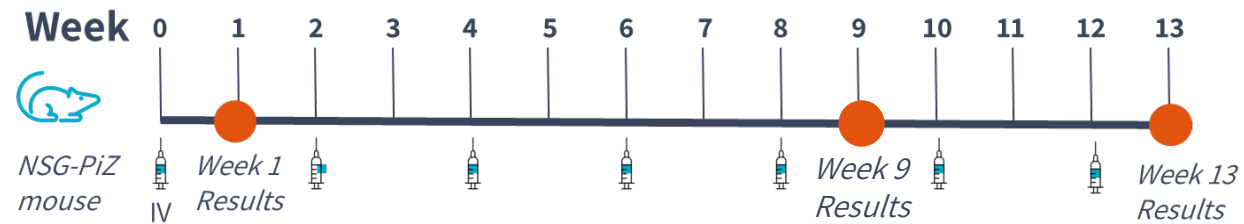
- >50% editing provides total AAT levels within the MZ range with
 - No lifetime risk for lung disease
 - Low lifetime risk for liver disease
- **Korro's goal** for ~50% median editing has the potential to provide benefit in both lung and liver disease in PiZZ individuals

¹Nakanishi T. et al. Eur Respir J. 2020 Dec 10;56(6):2001441

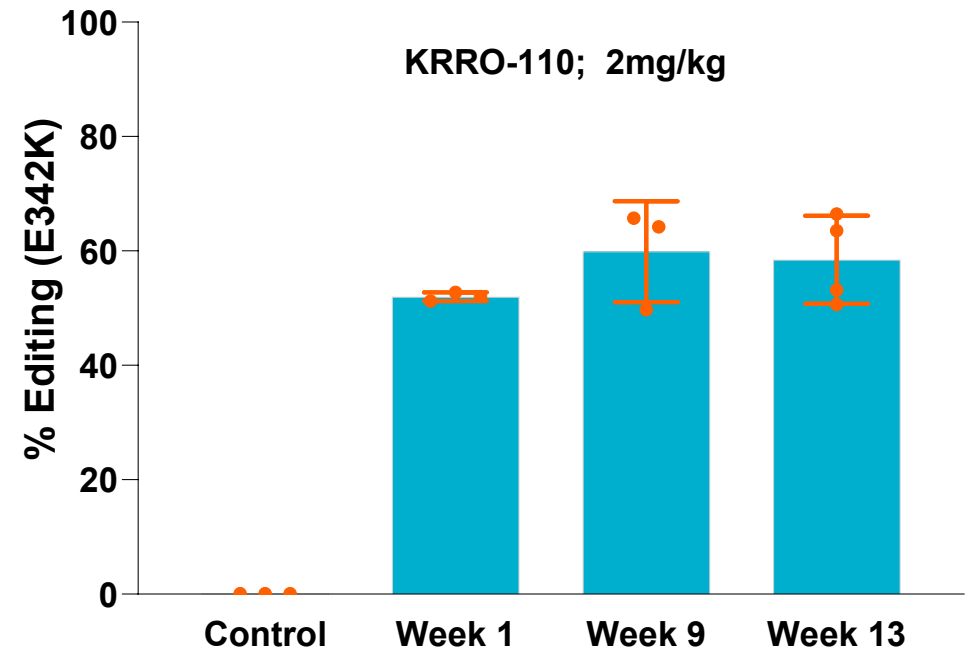
²Chronic obstructive pulmonary disease

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

Study design



RNA Editing in NSG-PiZ mouse

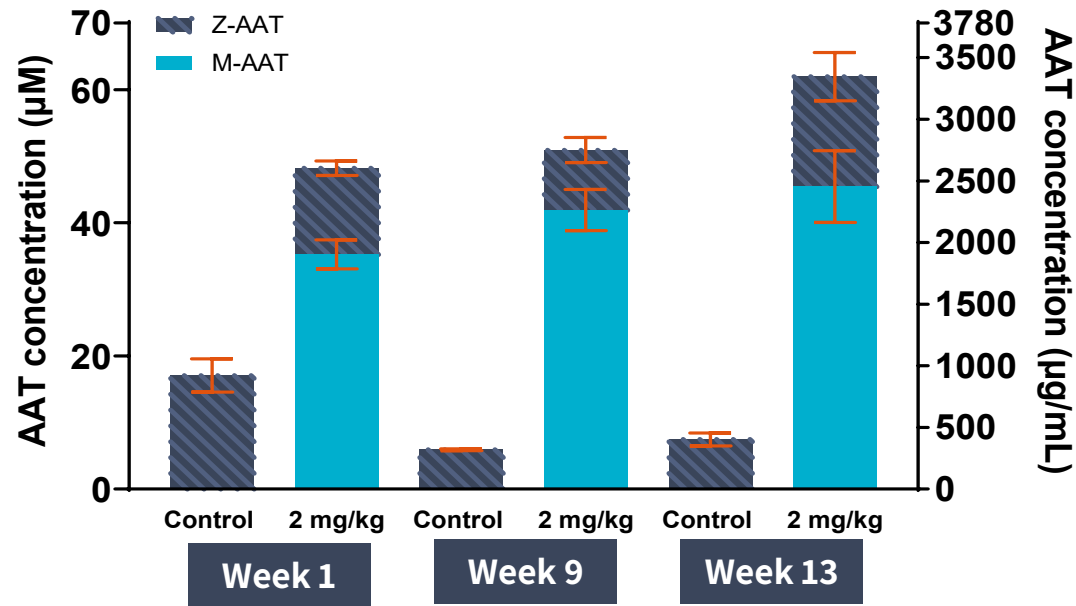


Protein Levels Commensurate with Editing Observed in the NSG-PiZ Model

Serum human-AAT concentration



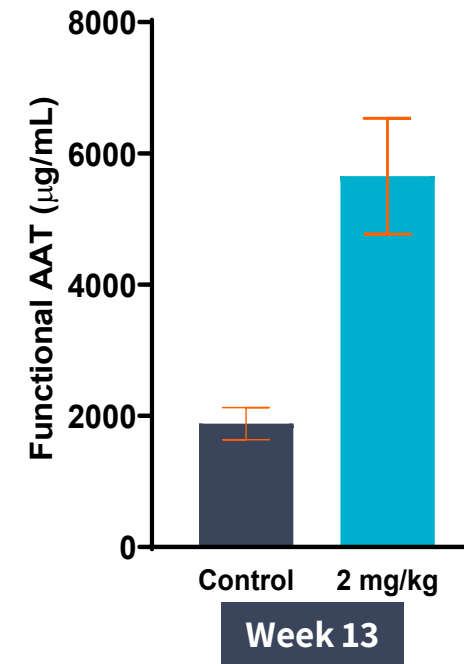
KRRO-110; 2mg/kg



Functional AAT concentration



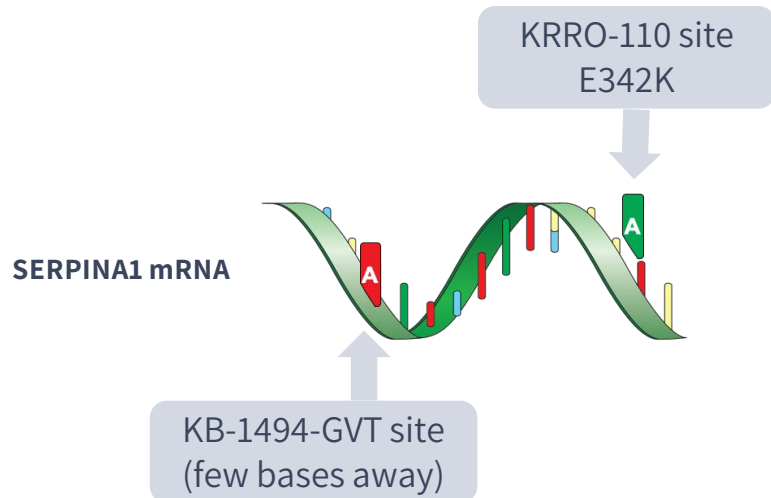
KRRO-110; 2mg/kg



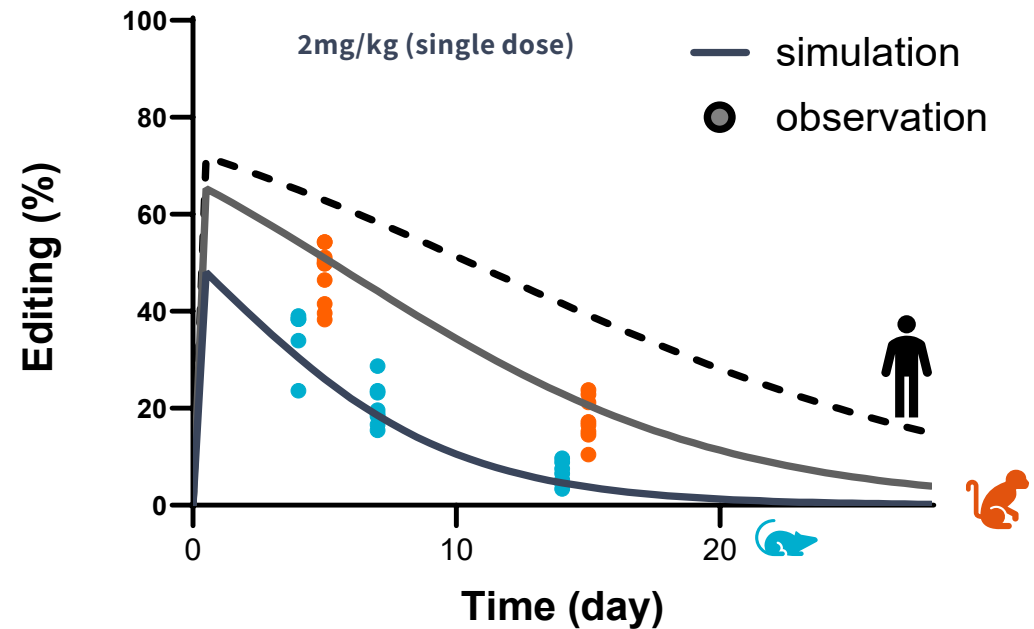
Achieved > 60 μM total AAT protein and 45 μM of M-AAT levels at week 13

SERPINA1 Surrogate Editors Demonstrate Good Translation to Higher Species

Surrogate SERPINA1 design: KB-1494-GVT



Observations for KB-1494-GVT in PiZ C57BL/6 and Cyno



KB-1494-GVT edits at ~2x in Cynos relative to PiZ mouse at same dose

KRRO-110: Progressing a Potential Best-in-Class Compound Into the Clinic

Preclinical Efficacy

- ✓ Achieved 60% editing
- ✓ Reduction in Z-AAT protein
- ✓ Secreted functional M-AAT

Preclinical Safety

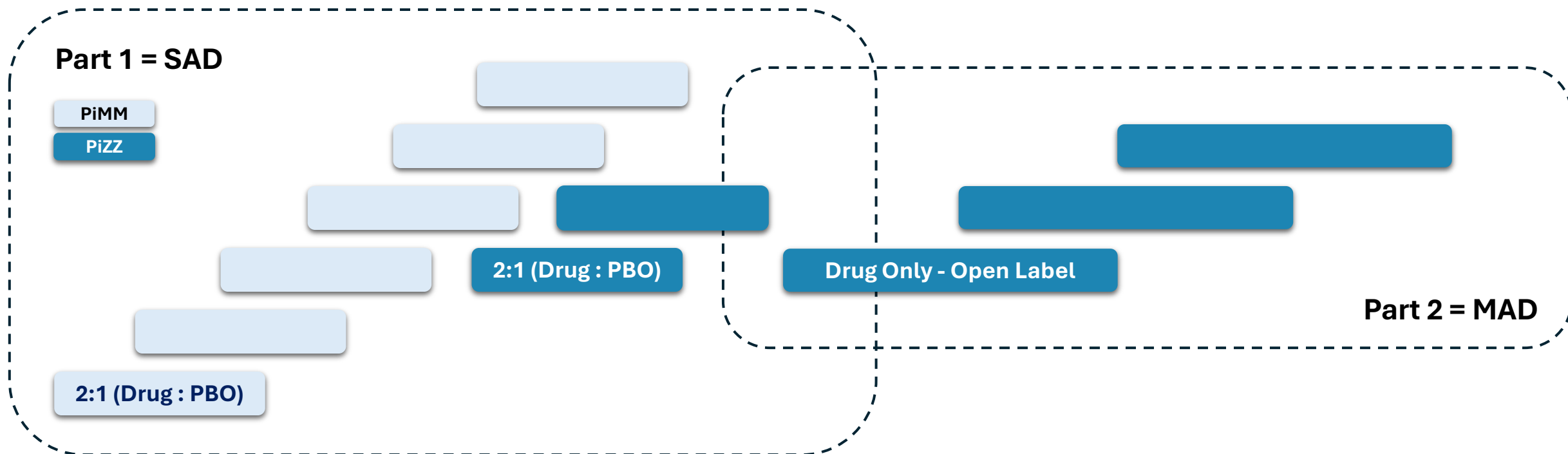
- ✓ No off-target effects
- ✓ Well tolerated in safety studies (mice, NHP)
- ✓ No signal of ASO-class effects

Translation to Higher Species

- ✓ Editing in MZ hepatocytes
- ✓ Editing translated to NHPs on surrogate site in SERPINA1
- ✓ Modeling predicts >70% editing in humans at C_{max}

Preclinical data modeling shows potential to achieve monthly dosing

KRRO-110 for AATD Clinical Study Design: Phase 1/2a, Two Part, Single- and Multiple-Dose Escalation Study



- **Study initiated in Australia, expanding to US and other geographies**
- **Total enrollment** = 64 adult participants with PiMM or PiZZ genotype
- **Primary endpoints:** Safety and tolerability
- **Secondary endpoints:** Pharmacokinetic (PK) parameters; Total-AAT, M-AAT, functional antiprotease activity

First participant dosed January 2025; Interim data anticipated in 2H'25

Positioned for Growth and Value Creation in 2025 and Beyond

Anticipated Milestones:



Share Interim clinical data for KRRO-110 from REWRITE study in 2H'25



Nominate a candidate with SC delivery (GalNAc) in Liver in '25 that can create a de novo protein variant



Progress and expand a wholly owned pipeline



Progress partnership with Novo Nordisk in cardiometabolic diseases with high prevalence



Cash runway into 2H'26¹ enables multiple milestones for KRRO-110 and other pipeline programs

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**Edit the message.
Rewrite the future.**

