



KORRO BIO

August 2026 Corporate Presentation

Forward-Looking Statement and Disclaimers

Forward-Looking Statements

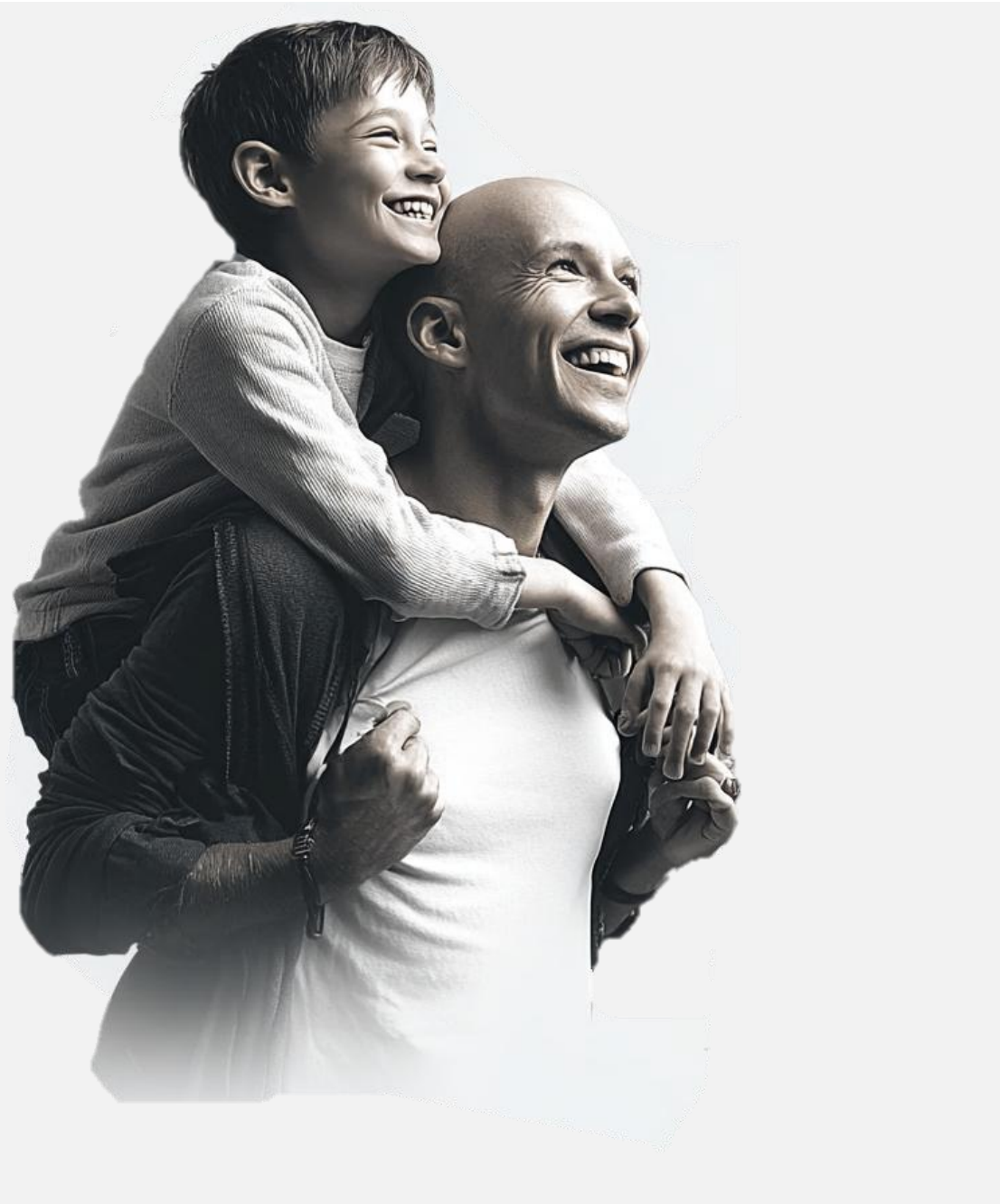
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Our Vision

**Developing Transformative
Genetic Medicines for
Rare and Highly Prevalent
Diseases**



Activating Biological Pathways



Editing RNA

Without permanently modifying DNA



Modular Delivery

Potential to deliver to multiple cell types



Learning from Genetics

To support predictable biological impact



Positioned for Value Creation in 2026 and Beyond



KRRO-111 Development Candidate (DC) nominated for GalNAc-conjugated alpha-1 antitrypsin deficiency (AATD) program



Regulatory approval to commence FIH clinical trial for KRRO-121 anticipated in H2 2026



DC expected for a 3rd GalNAc-conjugated liver asset in H2 2026



Cash runway into H2 '28 enabling multiple milestones ¹

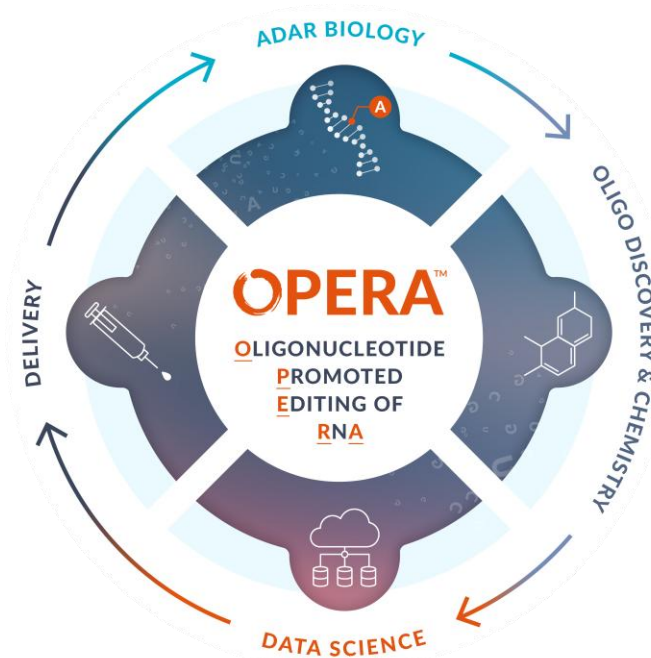
Potential for partnership across our pipeline

1. Cash, cash equivalents and marketable securities of \$137.9 million as of June 30, 2026

OPERA: Our Approach for RNA Editing to Generate Product Candidates

Expertise in ADAR biology
driving potency and translation

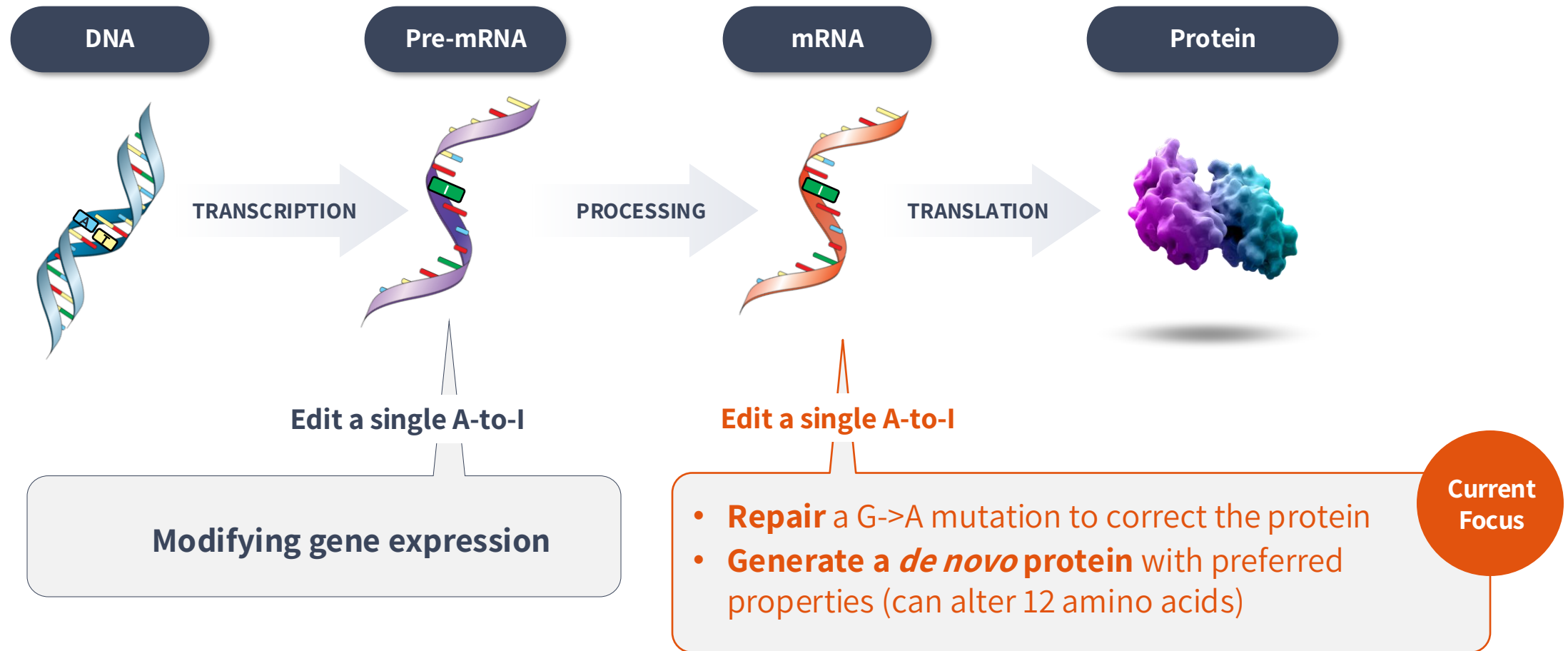
Leveraging known mechanisms to
derisk Delivery



Expertise in Chemistry
driving potency and drug designs

Expertise in Machine Learning
driving efficiency and Target ID

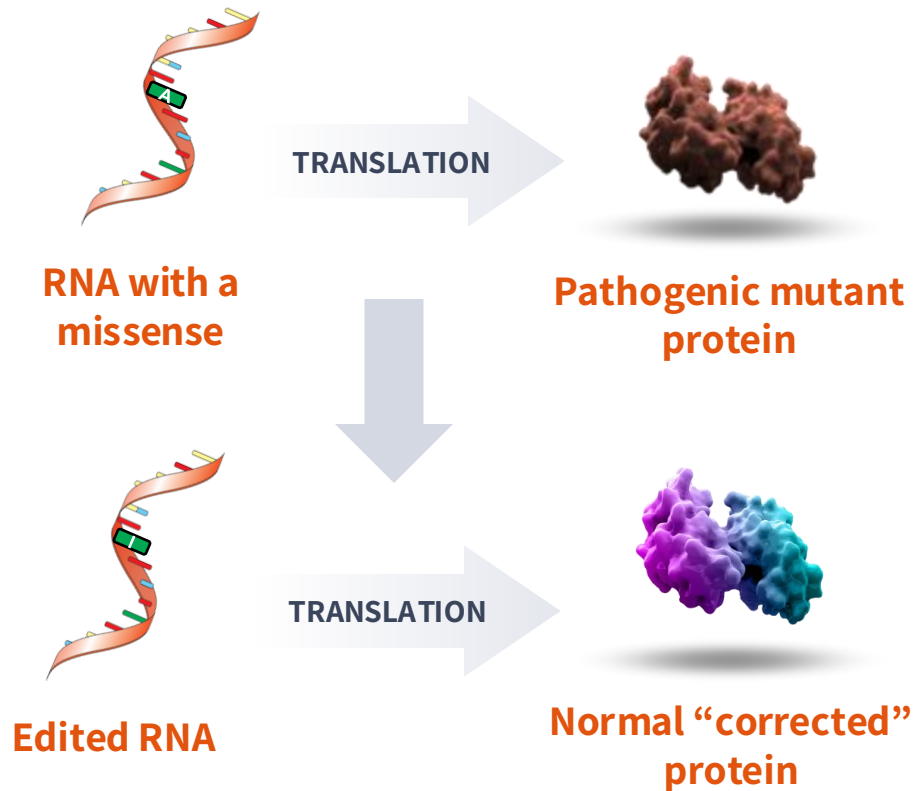
RNA Editing Enables Potential for High Impact in Range of Disease Areas



Human genetics guiding the possibilities

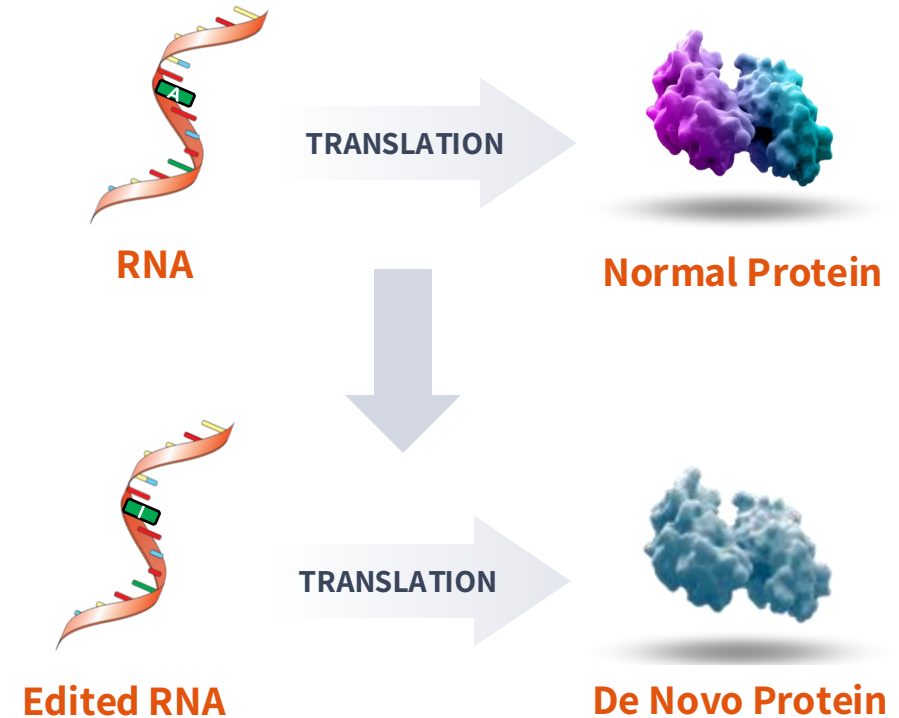
Pipeline Programs Primarily Focused on Generation of De Novo Proteins

Repair the Protein



Examples of Repair = E342K AATD, G2019S Parkinson Disease, Dravet's Syndrome...

Modulate Protein Function (Activate pathway)



Examples of Modulate = Hyperammonemia, ALS, MASH, Fibrosis...

Pipeline with Potential High-Value Programs and Anticipated Milestones

CONCEPT	PROGRAM / INDICATION	DELIVERY	DISCOVERY	PRECLINICAL DEVELOPMENT	PHASE 1	PHASE 2	PHASE 3
Stabilize Protein	KRRO-121 Hyperammonemia	GalNAc (SC)	GS Reg approval in 2H 2026				
Repair Pathogenic Variant	KRRO-111 AATD	GalNAc (SC)	AAT				
Allosteric Activator	Longevity (Liver)	GalNAc (SC)	AMPK γ 1				
Overcome LoF and GoF ¹	Amyotrophic lateral sclerosis (ALS)	Intrathecal (IT)	TDP43				

De Novo protein creation

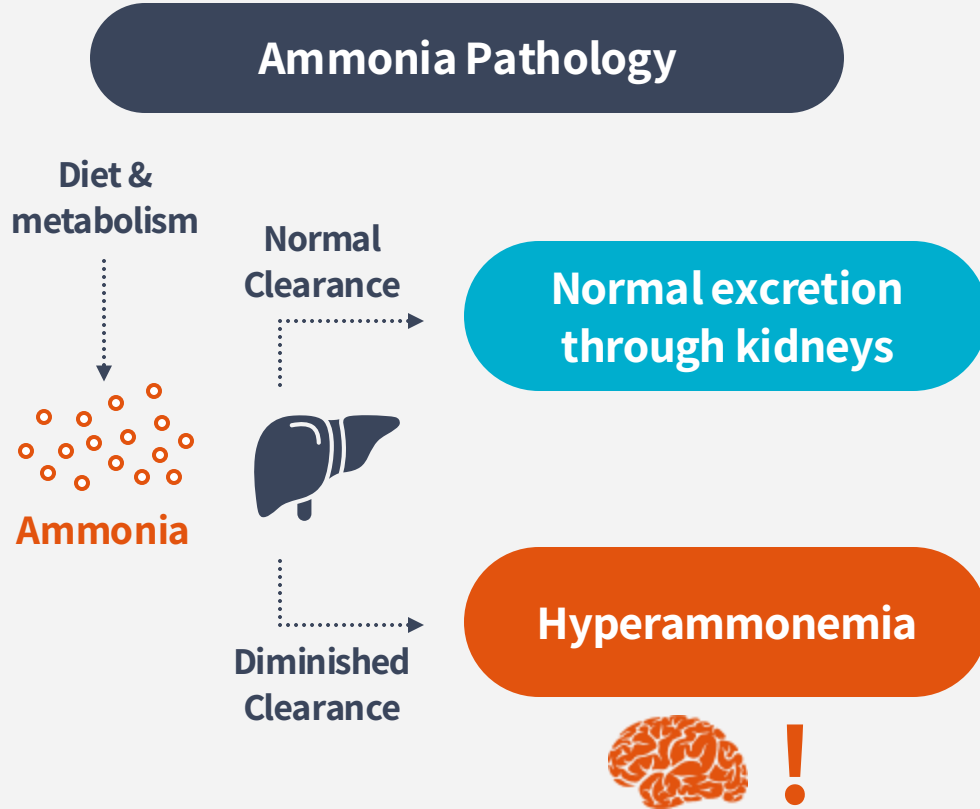
Protein repair

¹De Novo protein prevents toxic gain-of-function (GoF) with TDP43 aggregation, and continue downstream signaling by overcoming toxic loss-of-function (LOF)
 GS = Glutamine Synthetase; AAT = Alpha-1 antitrypsin; AATD = AAT deficiency; AMPK γ 1 = Regulatory subunit of AMP-activated protein kinase; TDP43 = TAR DNA-binding protein 43; SC = subcutaneous

KRRO-121: Hyperammonemia

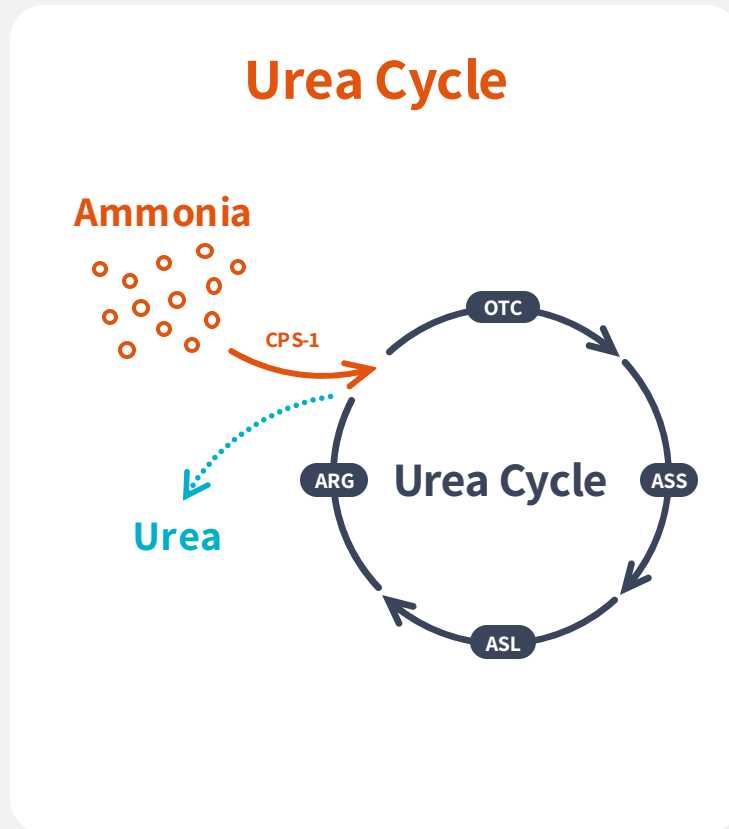
Synthetic Rescue

Plasma Ammonia Significantly Impacts Pathology Across Multiple Diseases

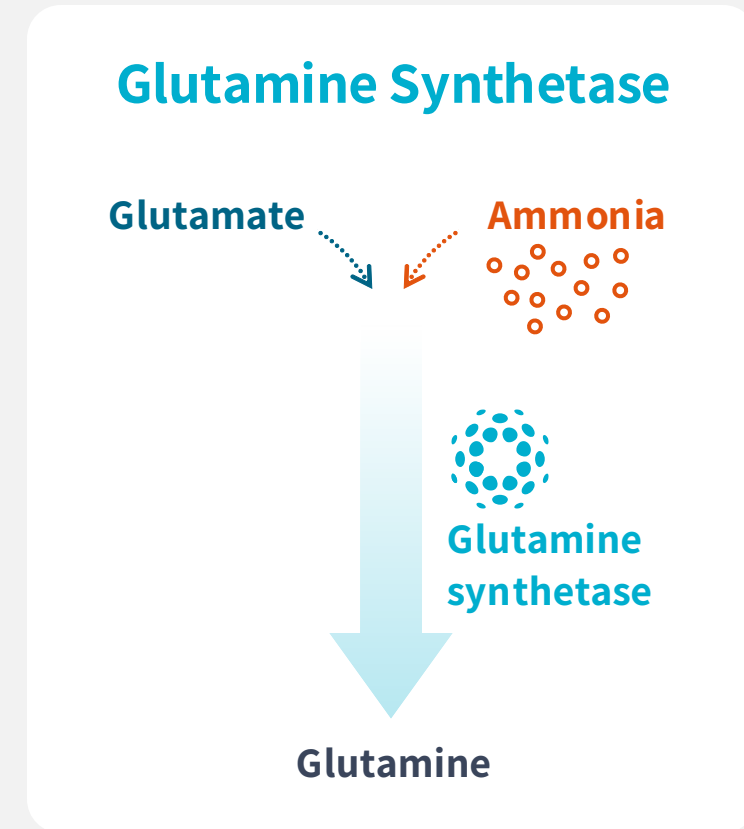


- **High ammonia** leads to:
 - Neurological impairment, potentially permanent
 - Frequent hospitalization
 - Highly restricted diet
 - Elevated infection risk
 - Additional non-neurological complications
- Can be caused by **cirrhosis or urea cycle dysfunction**
- Clinical studies have shown benefit of **lowering ammonia** in multiple indications

Two Complementary Pathways for Ammonia Clearance: Urea Cycle and Glutamine Synthetase (GS)



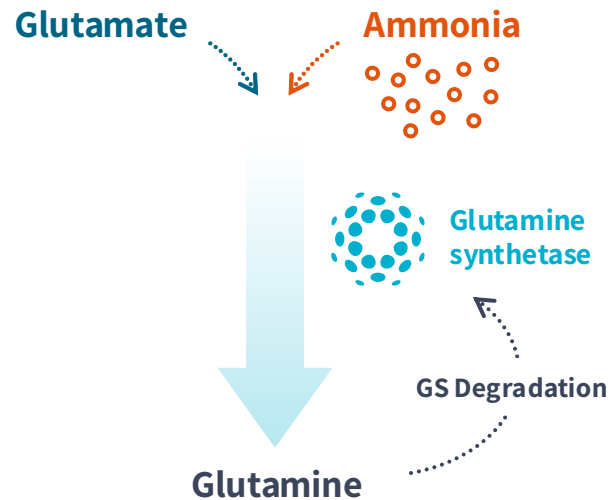
**Expressed
primarily in liver**



**Expressed in many tissues,
including liver, brain, and muscle**

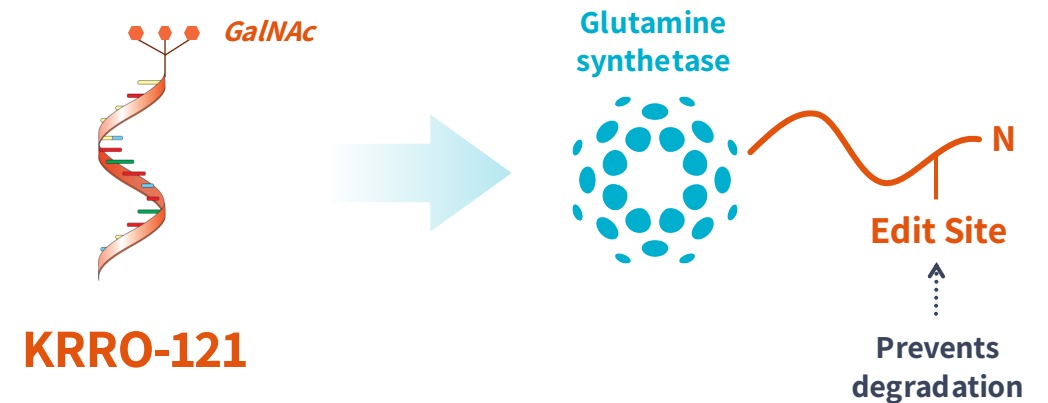
KRRO-121 is a GalNAc-conjugated Oligonucleotide Designed for Liver-specific RNA Editing of GS to Enhance Ammonia Clearance Capacity

Glutamine Drives Degradation of GS



GS degraded when glutamine rises, reducing ammonia clearance capacity

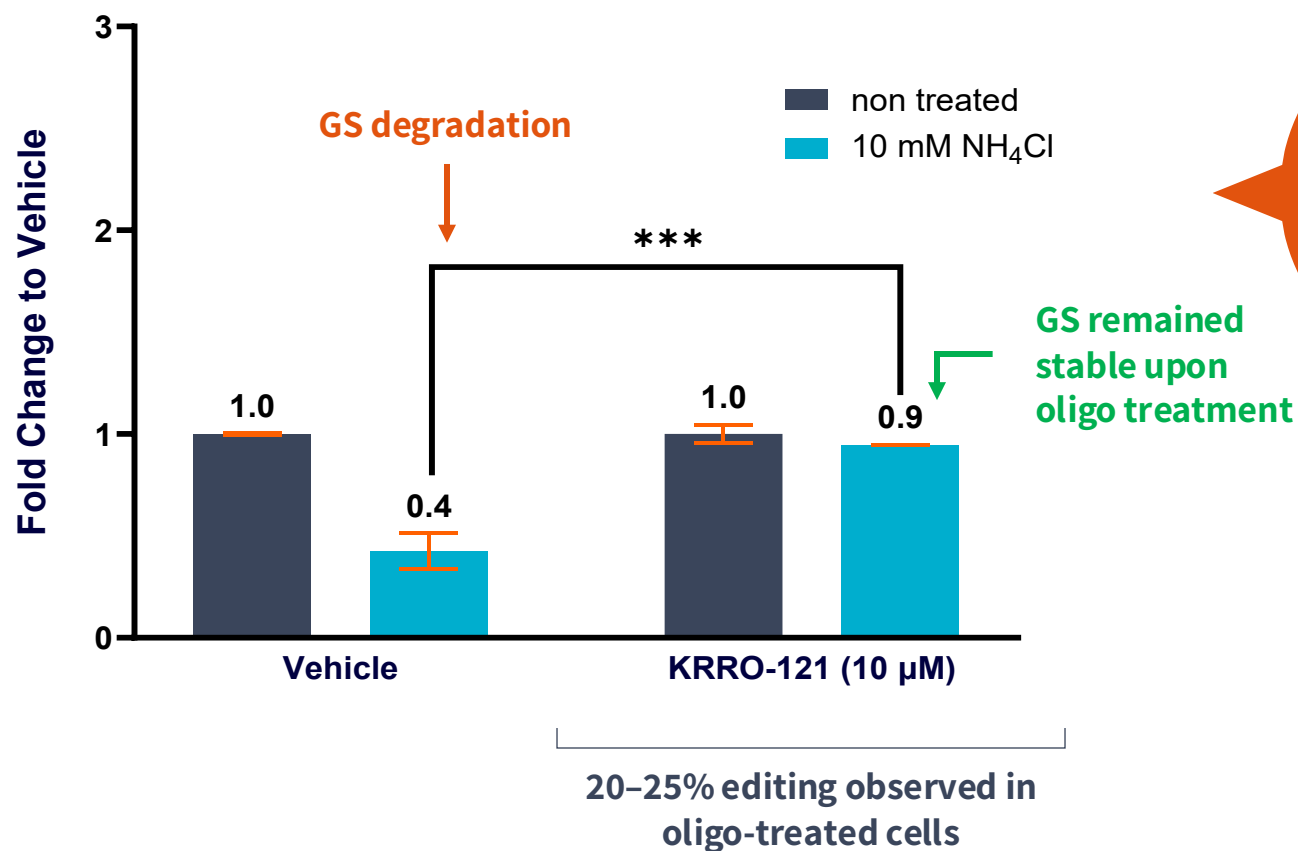
Our Approach



Liver-specific GalNAc-conjugated Oligo
Generates Stabilized, De Novo GS

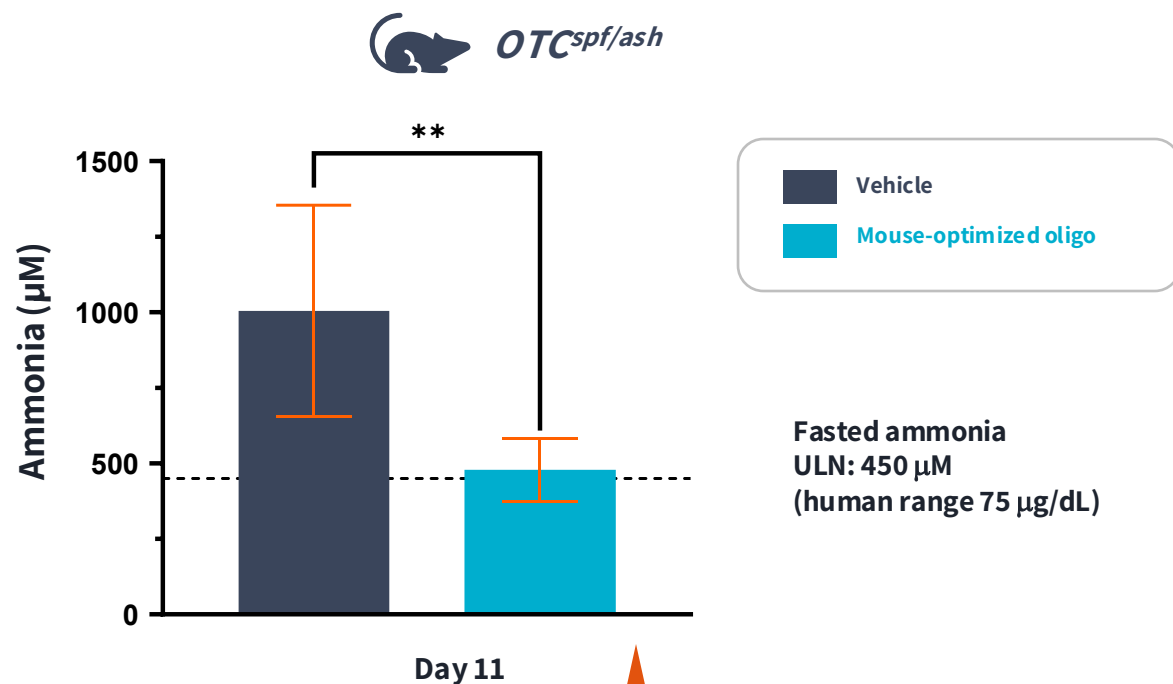
KRRO-121 Stabilized GS in UCD-derived Human Cell Models

KRRO-121 Stabilized GS in OTC-Deficient iPSC-Derived Hepatocytes

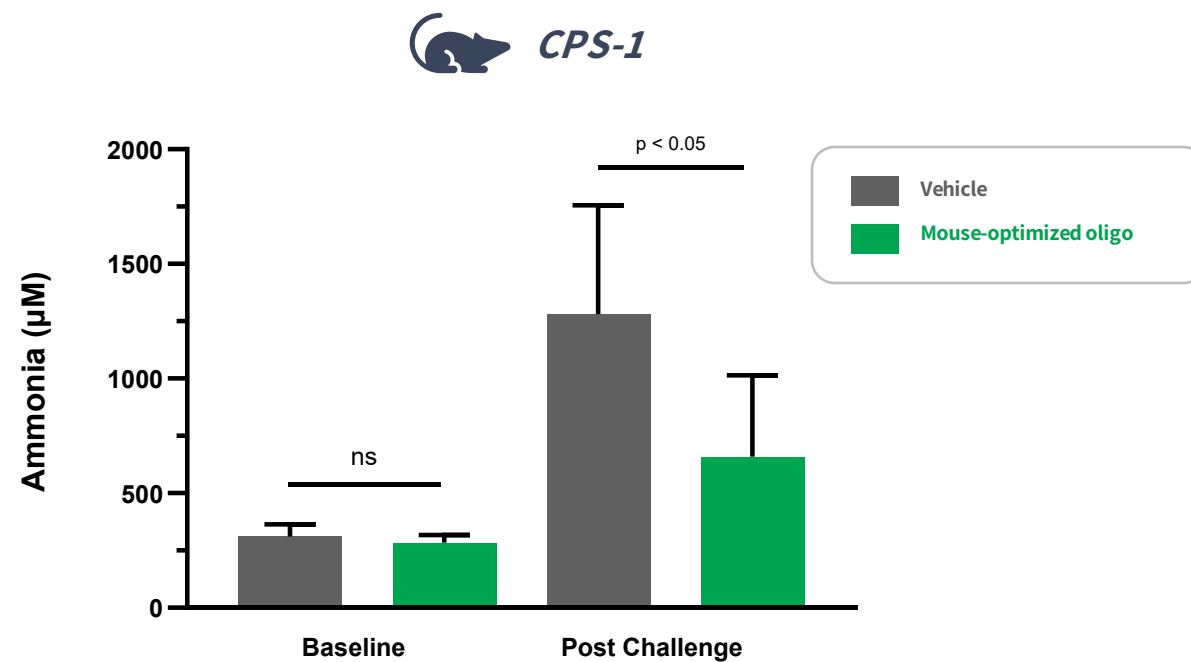


Similar results
in ASS1-deficient
iPSC-derived
hepatocytes

Ammonia Reduction in OTC- and CPS-1-Deficient Mice Challenged with Ammonia Supports Pan-UCD Potential and Diet Liberalization

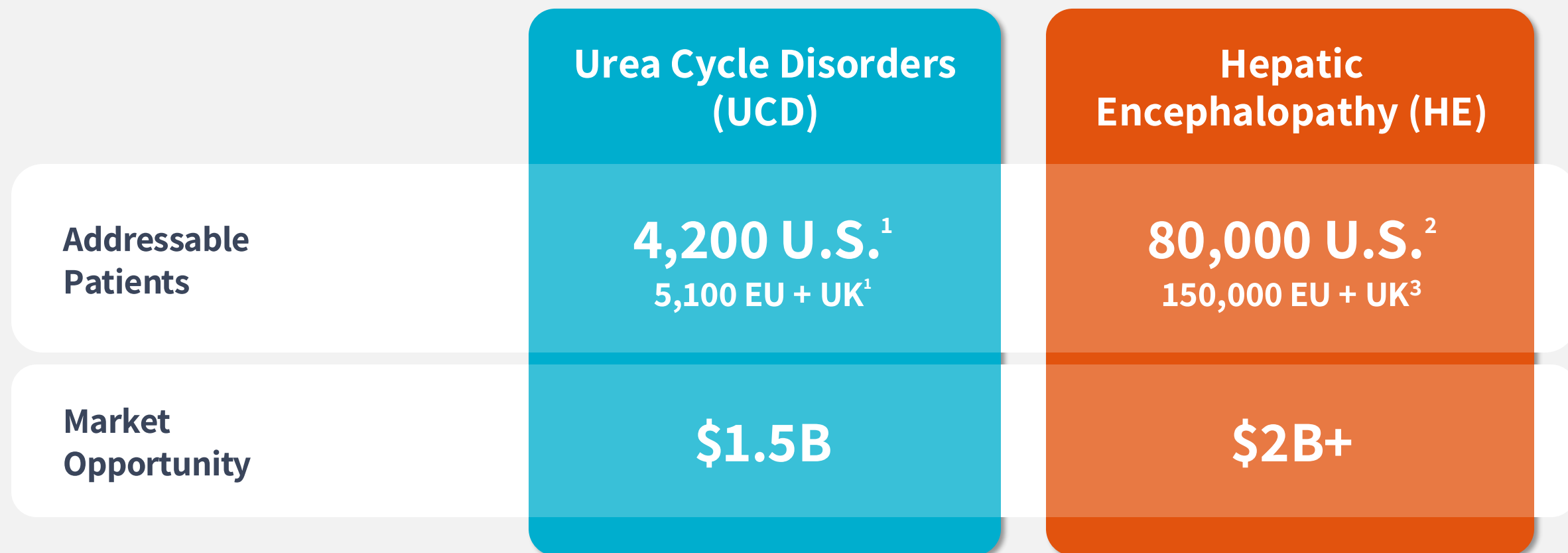


Ammonia challenge designed to
model patient protein consumption



Nonsignificant increase in plasma glutamine across both models

KRRO-121 Has Blockbuster Potential in Multiple Indications



Note: 1. Severe late-onset UCD patients; 2. Patients prescribed rifaximin +/- lactulose with $\geq 1.5\times$ normal ammonia and satisfactory liver function as assessed by laboratory values; 3. EU + UK estimate applies U.S. epidemiology assumptions to estimated EU + UK cirrhosis population

Source: 3rd party primary market research study (April 2025); KOL interviews; GlobalData; Electronic medical records analysis (data from 2022). All figures approximate.

KRRO-121: A Potential First-in-class Treatment For Ammonia Control

Preclinical Activity

- **Pan-UCD potential** impacting multiple UCD subtypes
- **Robust ammonia control** in OTC and CPS-1 mice challenged with ammonia¹
- **Diet liberalization potential** demonstrated by ammonia reduction during protein challenge

Preclinical Safety

- **NHP: No adverse safety signals** in repeat QWx3 dose range finding tox studies
- **NHP: No impact on coagulation, complement, platelets, cytokines**
- No evidence of editing observed in **mouse brain tissue**
- No increase in **mouse astrocyte staining** in KRRO-121 treated mice relative to vehicle treatment

Demonstrated Translation

- Production of **stable, *de novo* GS protein** which increased ammonia clearance and maintained normal glutamine levels
- Scaled from **mouse to monkey** and showed **targeted liver delivery**

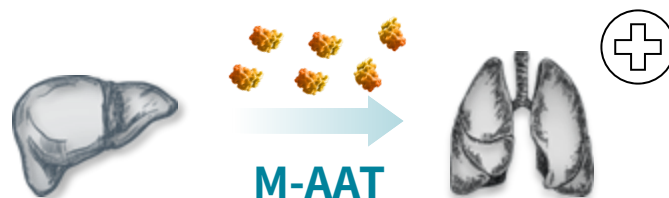
Strong preclinical data support KRRO-121's anticipated regulatory approval

KRRO-111: Alpha-1 Antitrypsin Deficiency

Repair Pathogenic Variant

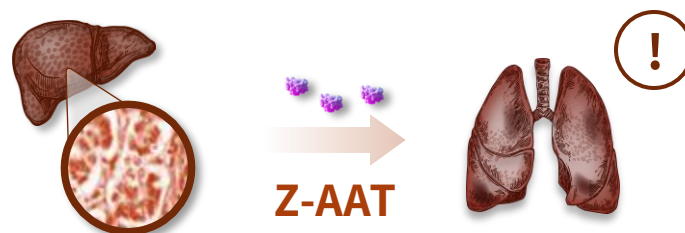
AATD: An Autosomal Recessive Disorder Leading to Liver and Lung Pathology

**Healthy
Genotype (MM)**



M-AAT is secreted from the liver and transported to the lungs, protecting against inflammatory damage

**AATD
Genotype (ZZ)**



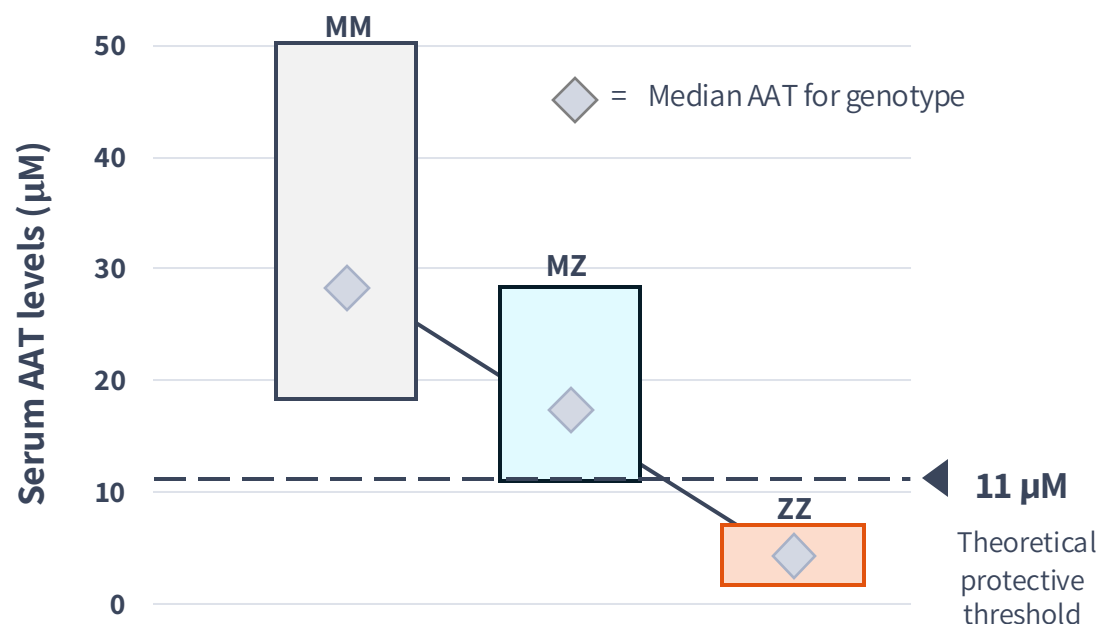
Z-AAT aggregates in the liver, leading to:

- **Liver damage from Z-AAT aggregates**
- **Low circulating AAT, causing lung damage**

>200k Pi*ZZ individuals in the US + EU

Current Standard of Care Approved Based on Lower Limit of MZ Phenotype

Serum Ranges Across AAT Genotypes



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

- The current standard of care is **weekly intravenous AAT augmentation therapy** (60 mg/kg/week)
- Augmentation therapy targets serum AAT levels $> 11 \mu\text{M}$ ¹** to slow progression of lung disease and does not address liver damage

Improved outcomes may be attainable by targeting higher total AAT levels

1. 11 μM T-AAT represents the lower bound of the 95% CI of T-AAT in MZ patients

Note: COPD = Chronic obstructive pulmonary disease

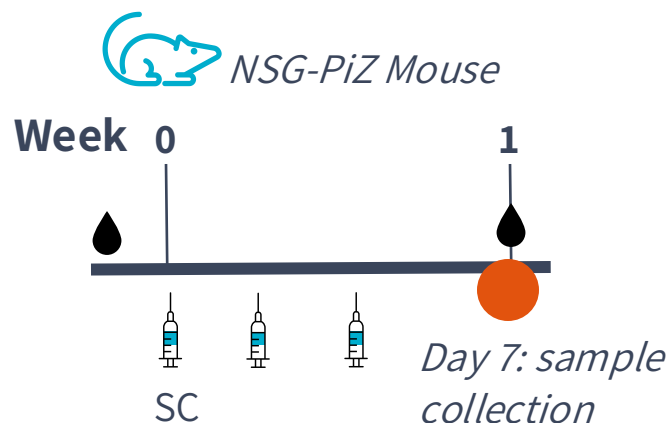
Source: Nakanishi T. et al. Eur Respir J. (2020); The undiagnosed disease burden associated with alpha-1 antitrypsin deficiency genotypes

KRRO-111, a GalNAc-conjugated RNA Editing Oligo, Demonstrated >90% Editing and ~90% M-AAT Protein *In Vivo*

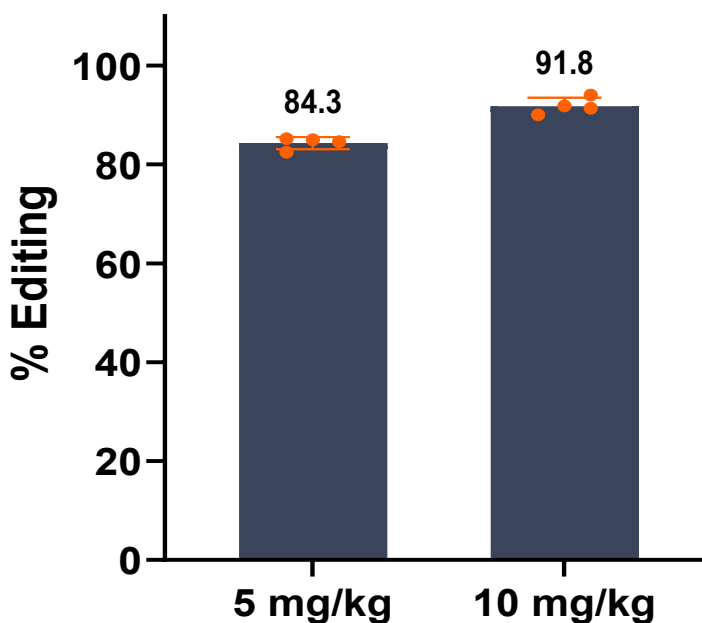
KRRO-111



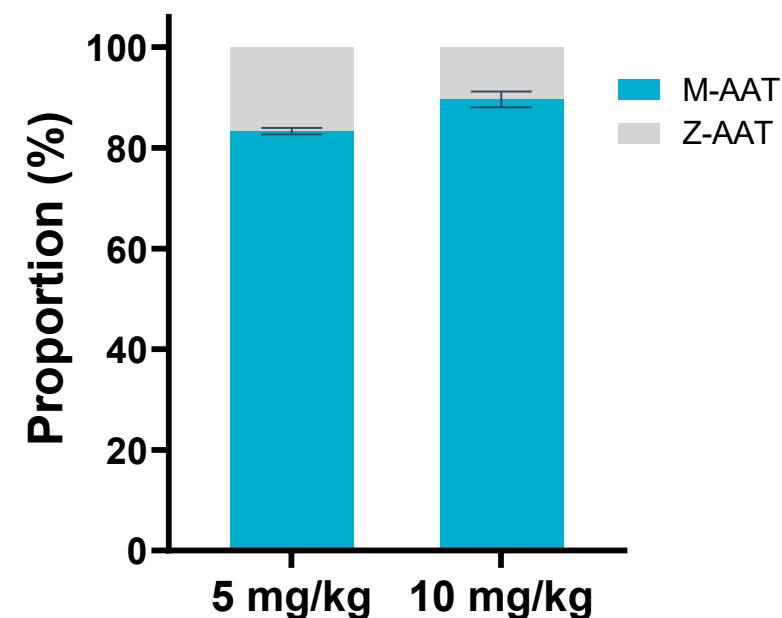
Dosing: Q2Dx3 at 5 or 10 mg/kg



SERPINA1 RNA Editing



% M-AAT Protein



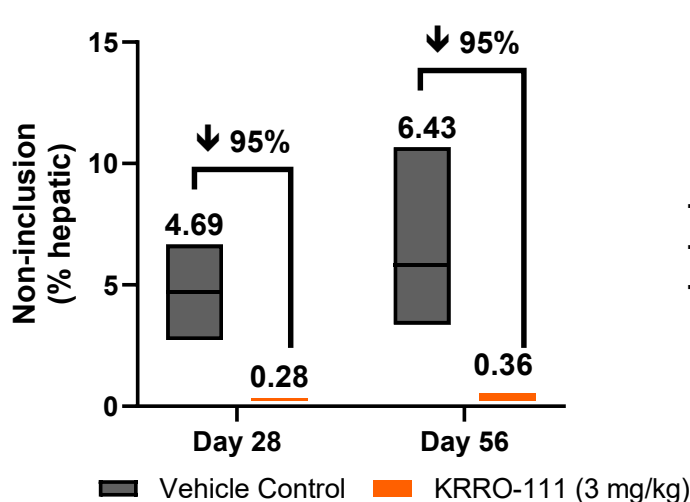
Note: Subcutaneous dosing Q2Dx3 at 5 or 10 mg/kg for each dose; editing efficiency and protein percentage are measured on day 7 by NGS and LC/MS, respectively. Proportion of M-AAT was calculated from the sum of M-AAT and Z-AAT detected in serum.

KRRO-111 Nearly Eliminates Active Z Protein Production and Drives Progressive Clearance of Pre-existing Aggregates *In Vivo*

Near-Complete Cessation of Z-AAT Protein Synthesis

NON-INCLUSION Z-AAT

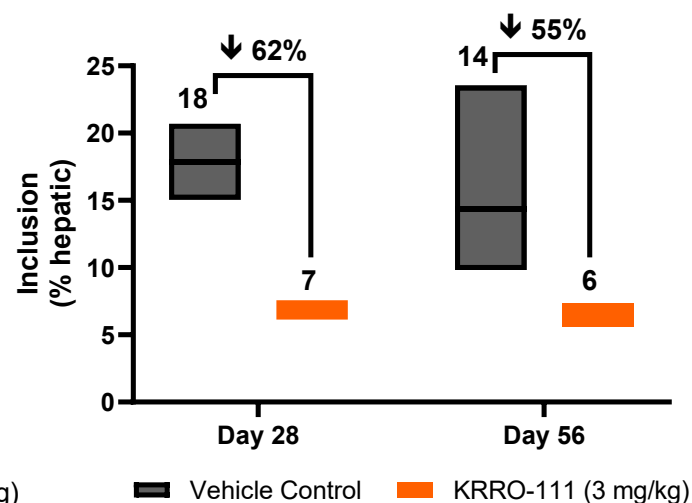
Active Z protein · editing efficacy



Robust Reduction of Pre-Existing Z-AAT Aggregates

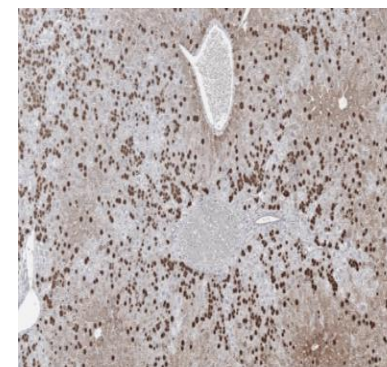
INCLUSION-ASSOCIATED Z-AAT

Pre-existing aggregates · autophagic clearance

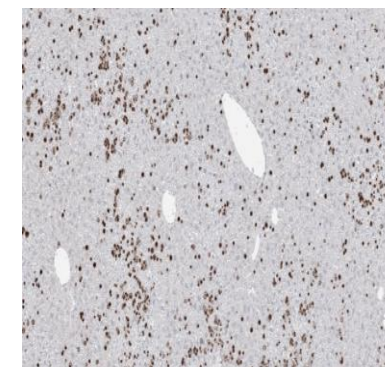


Elimination of Active Z-AAT Protein Production and Progressive Clearance of Pre-existing Aggregates

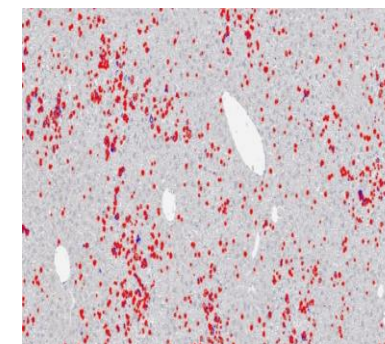
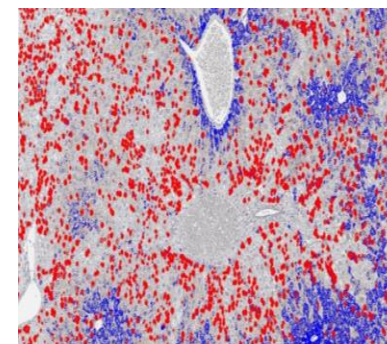
Vehicle Control



KRRO-111



Z-AAT protein (brown)



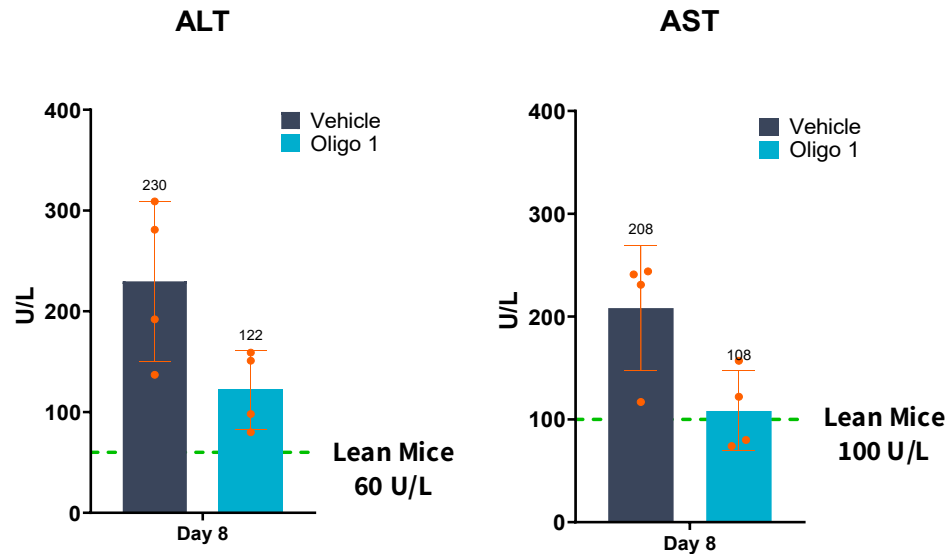
Non-inclusion Z-AAT (blue)

Inclusion-associated Z-AAT (red)

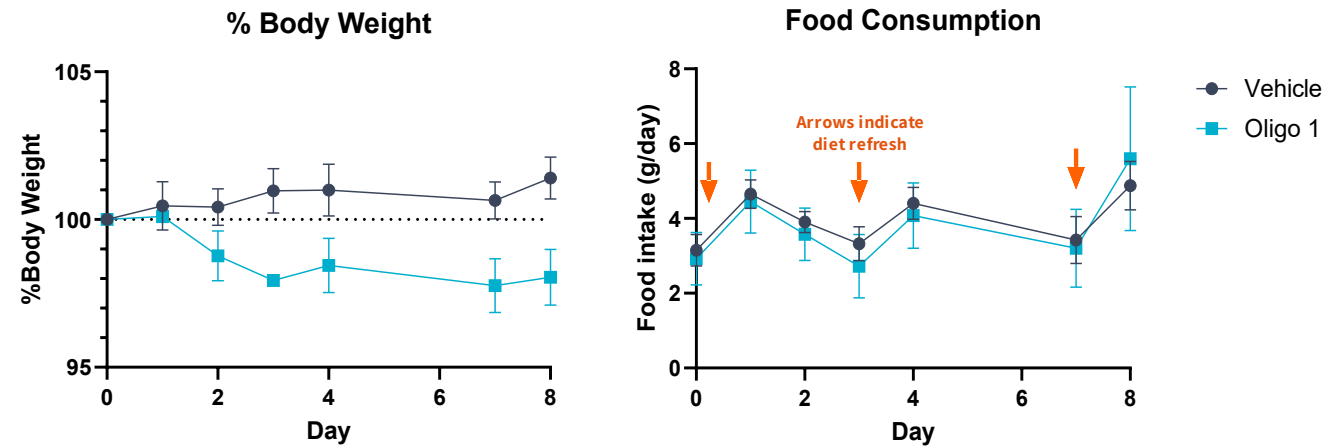
Early Pipeline

Hepatic AMPK γ 1 Activation Improved Liver Function in Obese Mice

Normalizing Liver Function



Reduction in Body Weight Despite Similar Food Intake



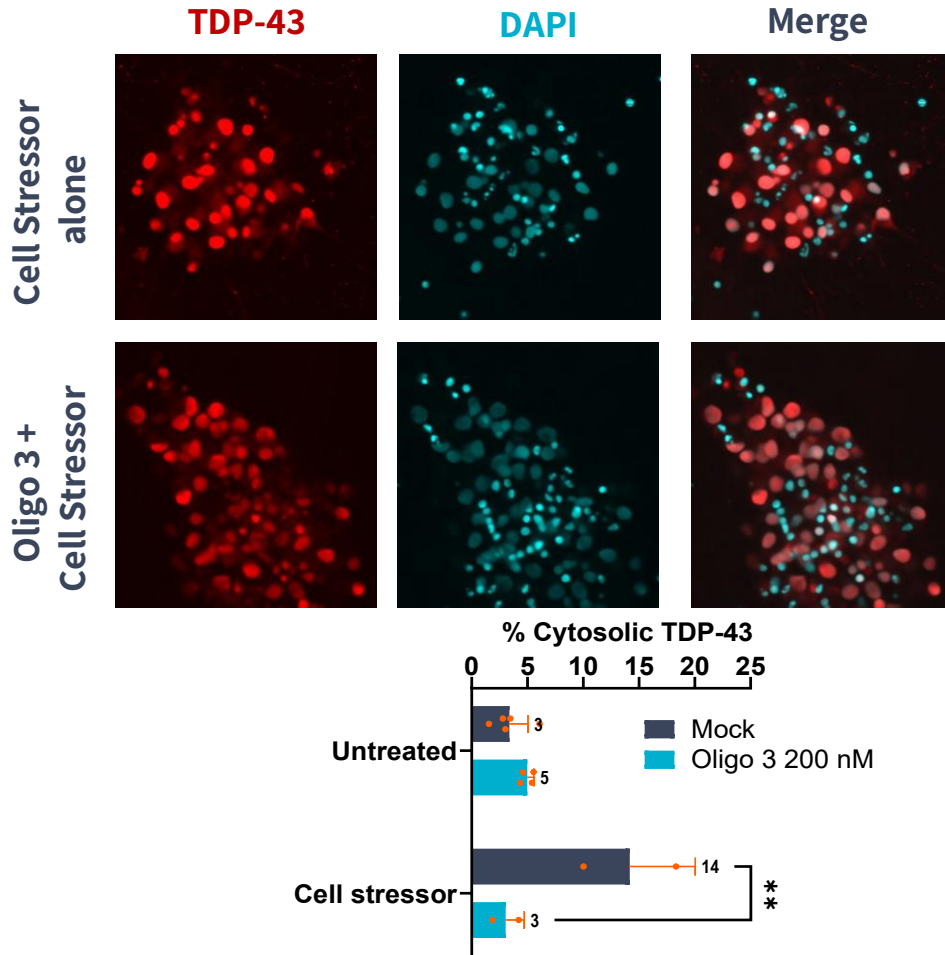
AMPK

- Activation of the Master Metabolic Regulator
- Designed to Improve Liver Function
- Potentially Restores Metabolic Signaling Without Affecting Food Intake

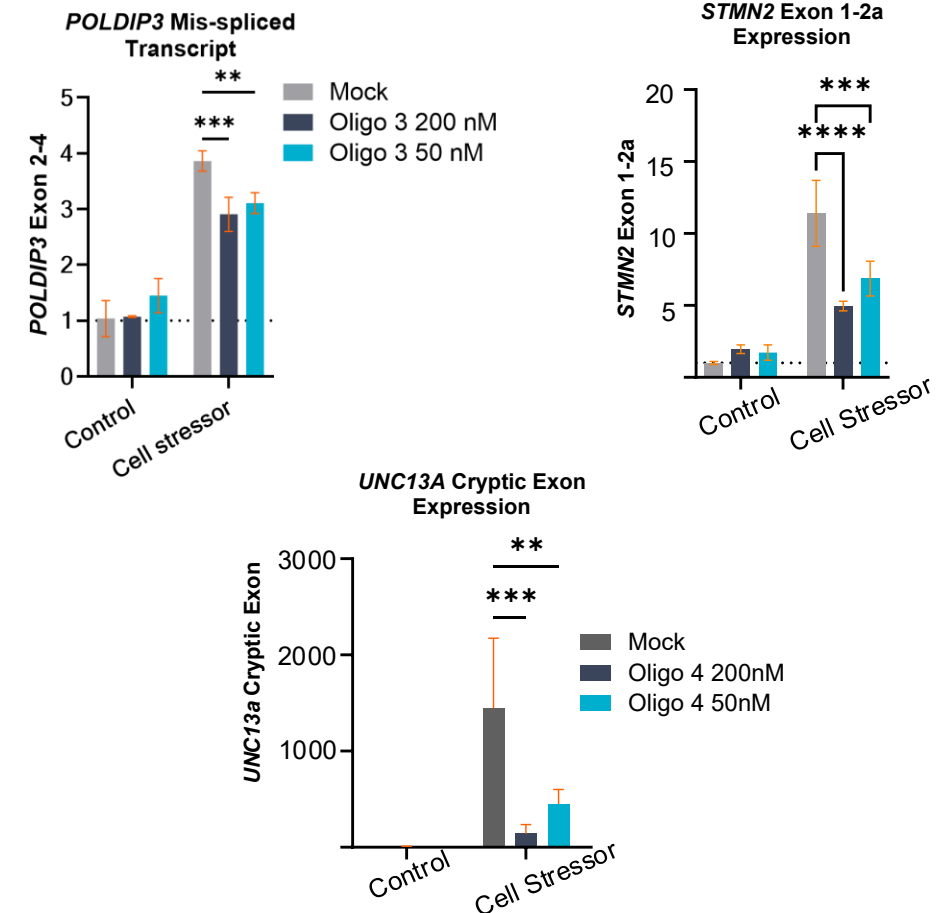
~20% editing was sufficient to normalize liver function and reduce body weight in mice

De Novo TDP-43 Demonstrated Reduced Mis-splicing and Decreased Cytosolic Mis-localization in iPSC Motor Neurons

Decreased Cytosolic Mis-localization of TDP-43 protein



Reduced Mis-splicing: Maintaining POLDIP3, STMN2, and UNC13A





Positioned for Value Creation in 2026 and Beyond



KRRO-111 DC nominated for GalNAc-conjugated AATD program



Regulatory approval to commence FIH clinical trial for KRRO-121 anticipated in H2 2026



DC expected for a 3rd GalNAc-conjugated liver asset in H2 2026



Cash runway into H2 '28 enabling multiple milestones ¹

Edit the Message Rewrite the Future

1. Cash, cash equivalents and marketable securities of \$137.9 million as of June 30, 2026