

# KORRO BIO



May 2026 Corporate Presentation

# Forward-Looking Statement and Disclaimers

## Forward-Looking Statements

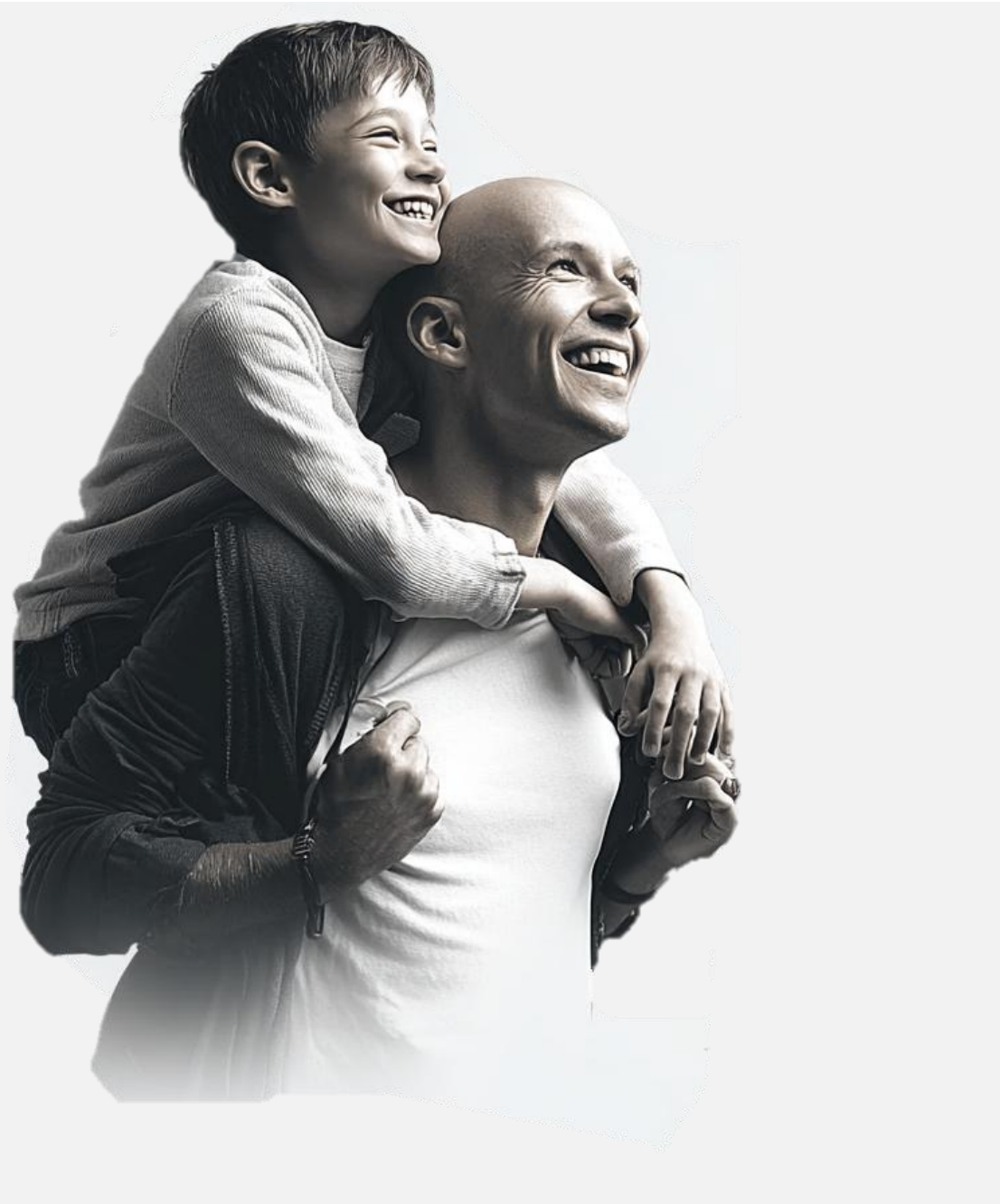
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## **Our Vision**

**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



Regulatory filing for KRRO-121 anticipated in H2 2026



Development Candidate (DC) announcement for GalNAc-conjugated alpha-1 antitrypsin deficiency (AATD) construct expected in Q2 2026



DC expected for a 3<sup>rd</sup> GalNAc-conjugated liver asset in H2 2026



Cash runway into H2 '28 enabling multiple milestones <sup>1</sup>

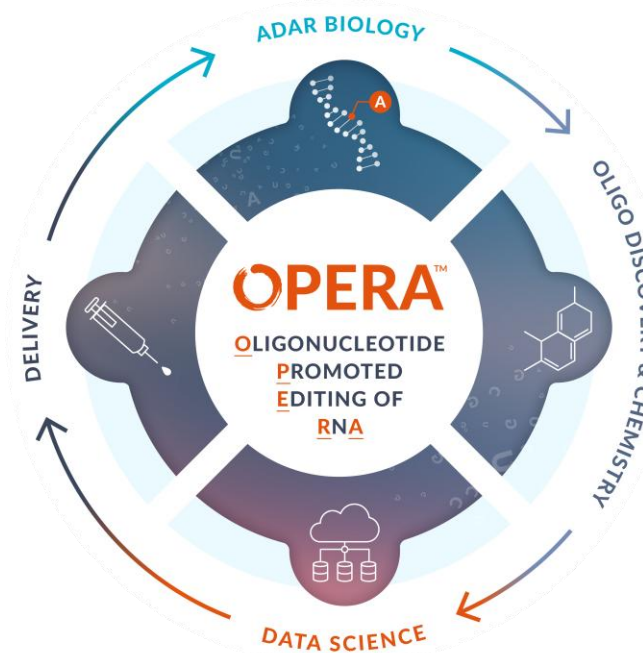
Potential for partnership across our pipeline

1. Cash, cash equivalents and marketable securities of \$157.1 million as of March 31, 2026

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

Expertise in ADAR biology  
driving potency and translation

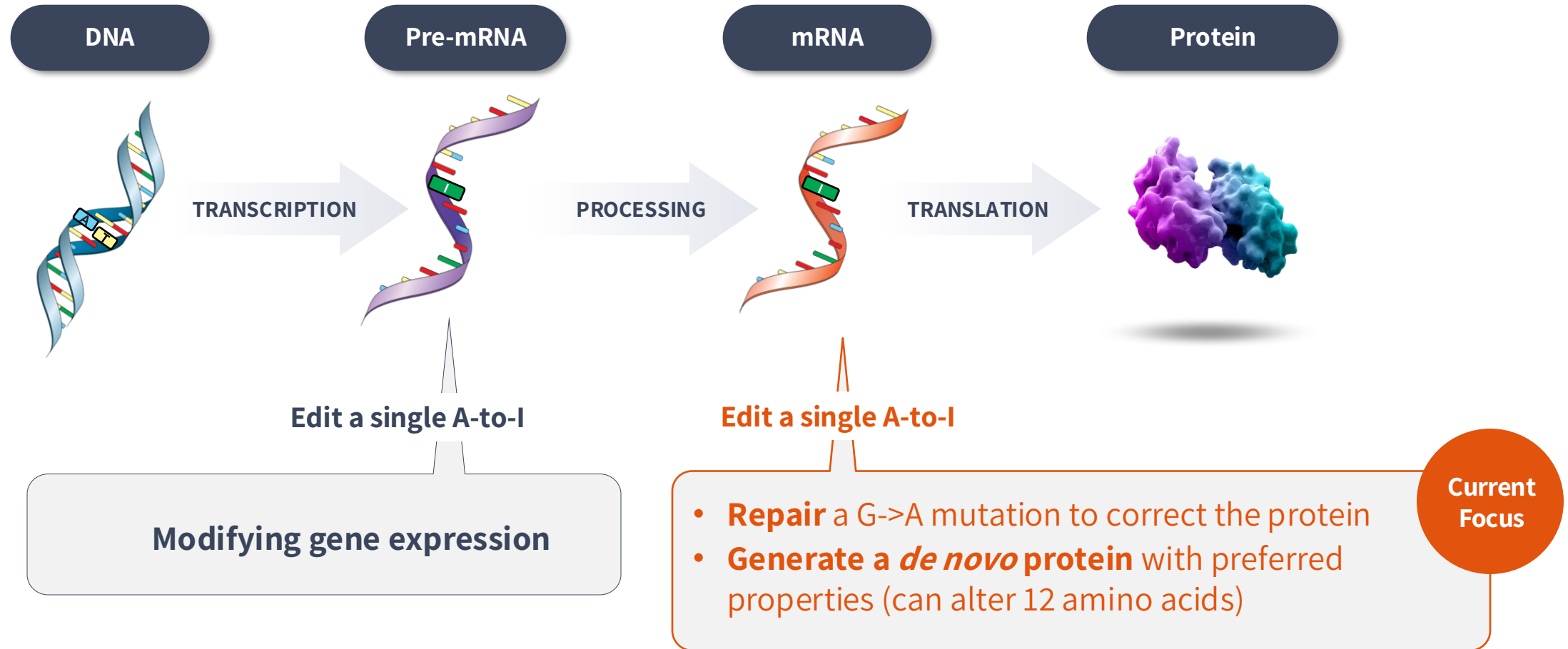
Leveraging known mechanisms to  
derisk Delivery



Expertise in Chemistry  
driving potency and drug designs

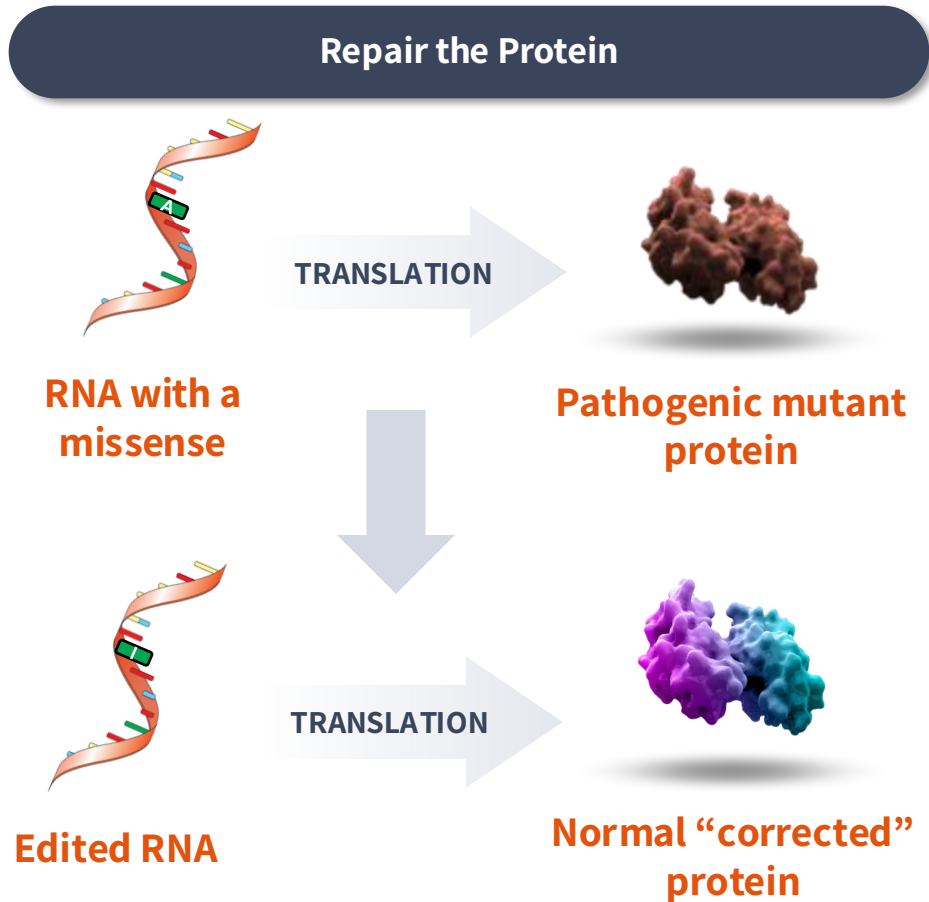
Expertise in Machine Learning  
driving efficiency and Target ID

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

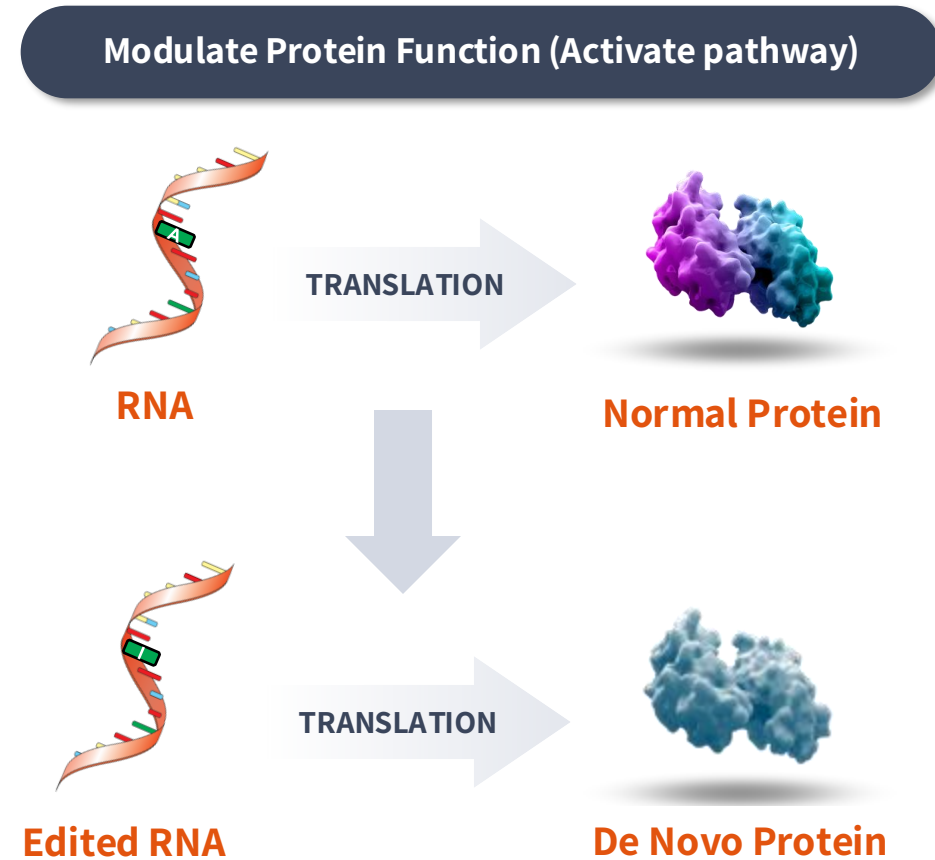


Human genetics guiding the possibilities

# Pipeline Programs Primarily Focused on Generation of De Novo Proteins



Examples of Repair = E342KAATD, G2019S Parkinson Disease, Dravet's Syndrome...



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 filing in 2H 2026				
Repair Pathogenic Variant	AATD	GalNAc (SC)	AAT DC in 2Q 2026				
Allosteric Activator	Longevity (Liver)	GalNAc (SC)	AMPK $\gamma$ 1				
Overcome LoF and GoF <sup>1</sup>	Amyotrophic lateral sclerosis (ALS)	Intrathecal (IT)	TDP43				

De Novo protein creation

Protein repair

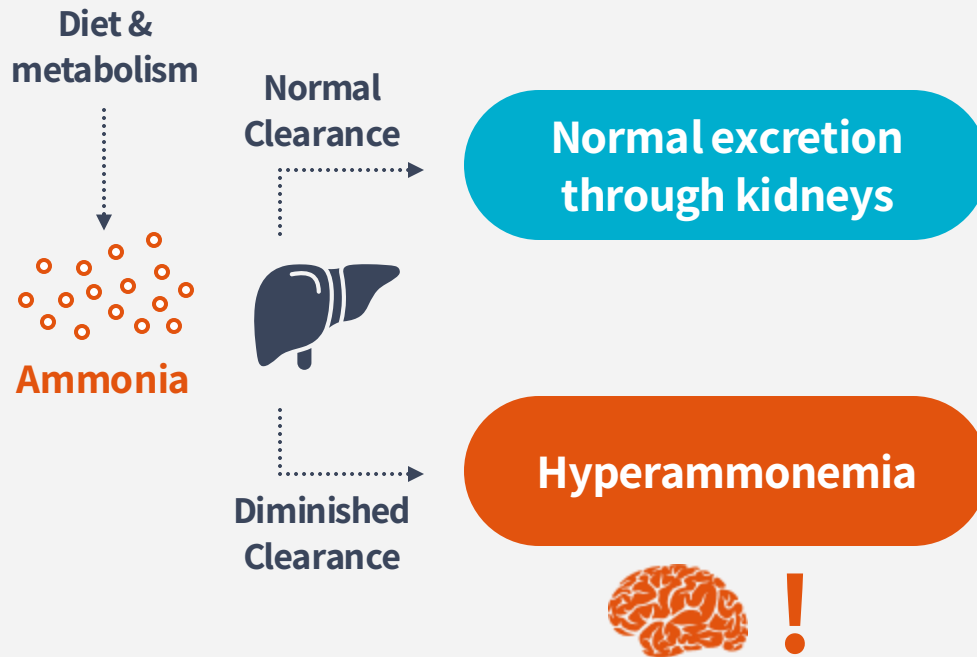
<sup>1</sup>De Novo protein prevents toxic gain-of-function (GoF) with TDP43 aggregation, and continue downstream signaling by overcoming toxic loss-of-function (LOF)  
 GS = Glutamine Synthetase; AAT = Alpha-1 antitrypsin; AATD = AAT deficiency; AMPK  $\gamma$ 1 = Regulatory subunit of AMP-activated protein kinase; TDP43 = TAR DNA-binding protein 43; DC = Development Candidate

# KRRO-121: Targeting Hyperammonemia

**Synthetic Rescue**

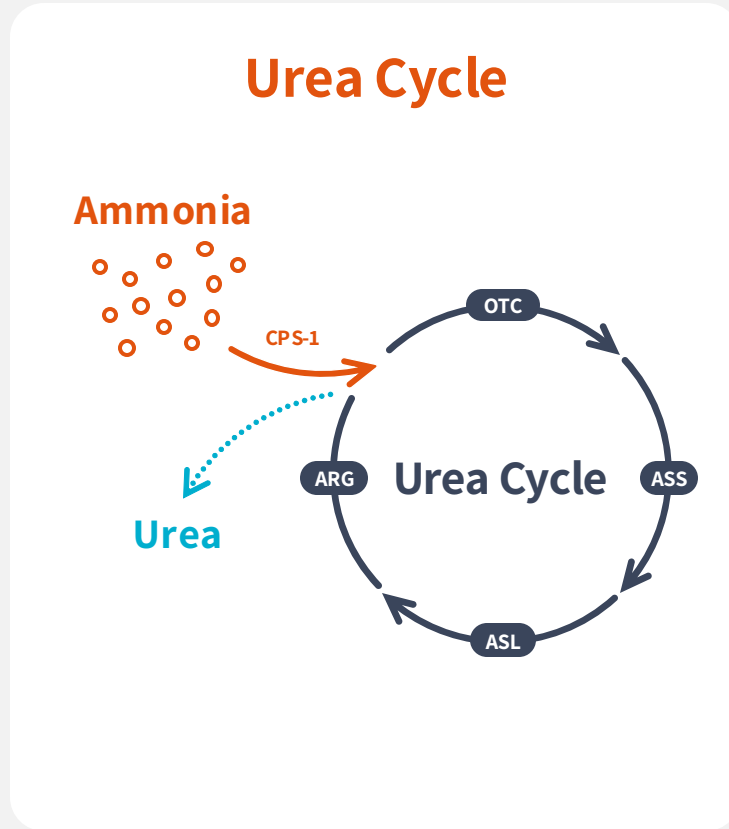
# Plasma Ammonia Significantly Impacts Pathology Across Multiple Diseases

## Ammonia Pathology

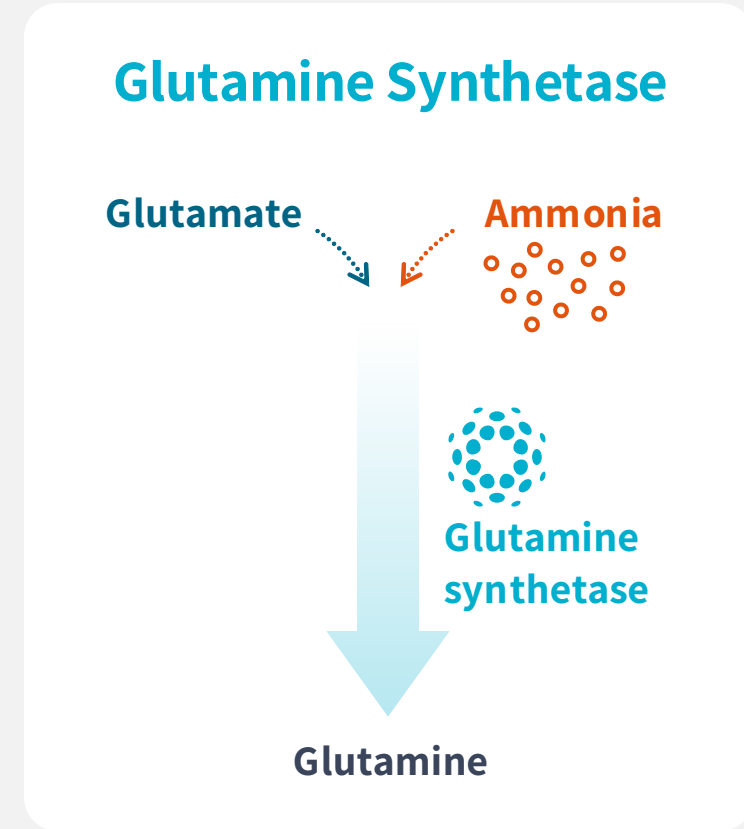


- **High ammonia** leads to:
  - Neurological impairment, potentially permanent
  - Frequent hospitalization
  - Highly restricted diet
  - Elevated infection risk
  - Additional non-neurological complications
- Can be caused by **cirrhosis or urea cycle dysfunction**
- Clinical studies have shown benefit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

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



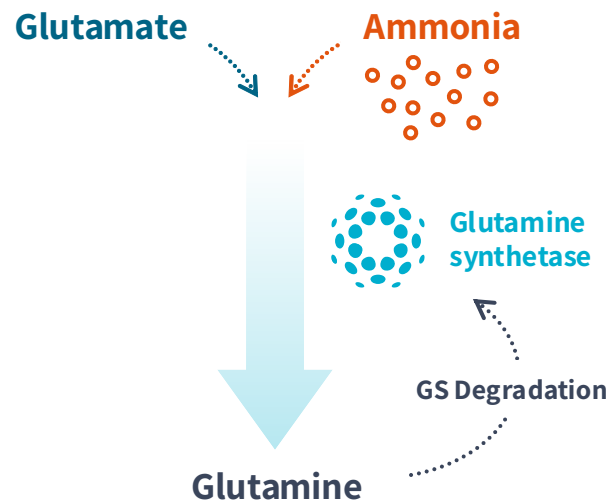
**Expressed  
primarily in liver**



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

# Degradation of GS Controlled by Levels of Glutamine

## Glutamine Drives Degradation of GS



GS degraded when glutamine rises, reducing ammonia clearance capacity

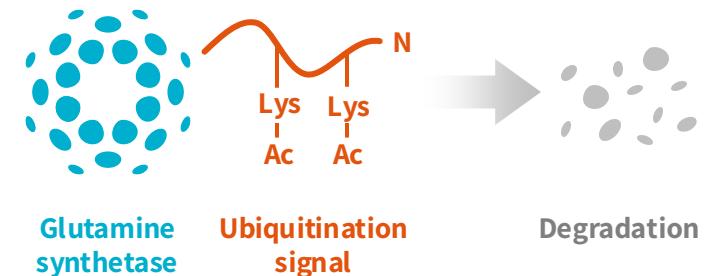
## Degradation Mechanism: Acetylation of Key N-terminal Residues

Low glutamine



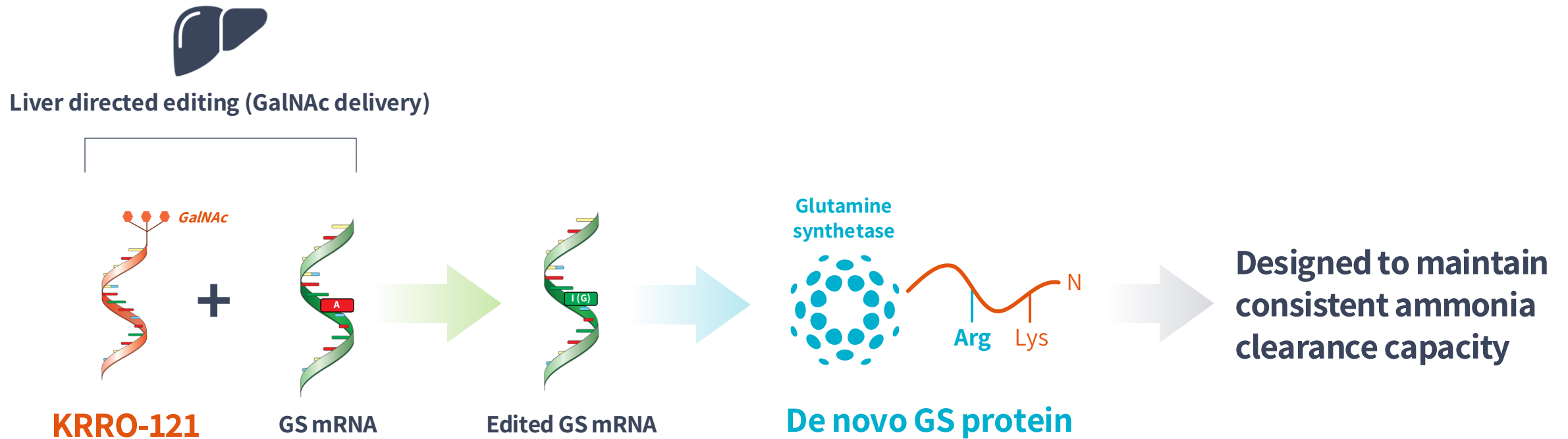
No lysine acetylation, GS is stable

High glutamine



Acetylation of lysine residues, leading to ubiquitination and protein degradation

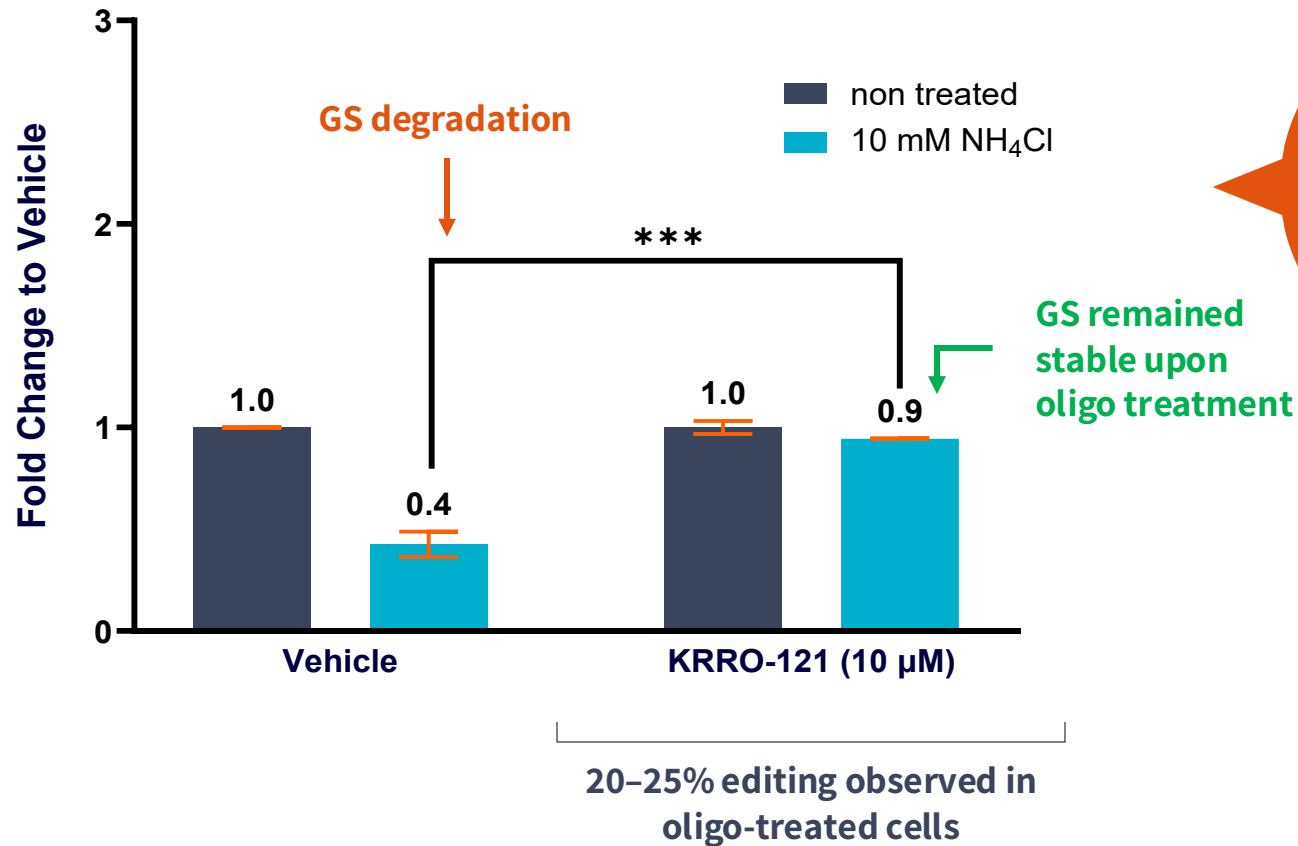
# Our Approach: Liver-specific, GalNAc-Oligo to Generate a Stabilized, De Novo GS Protein



**KRRO-121: GalNAc-conjugated oligonucleotide designed for liver-specific RNA editing of GS to enhance ammonia clearance capacity**

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

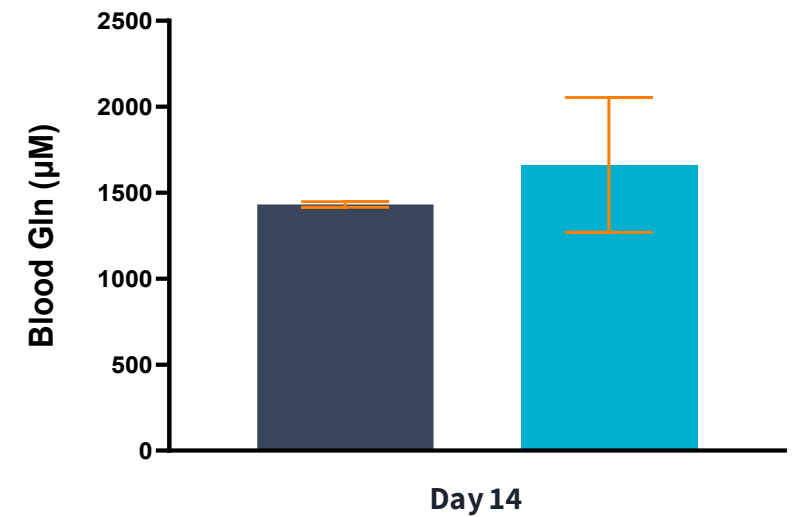
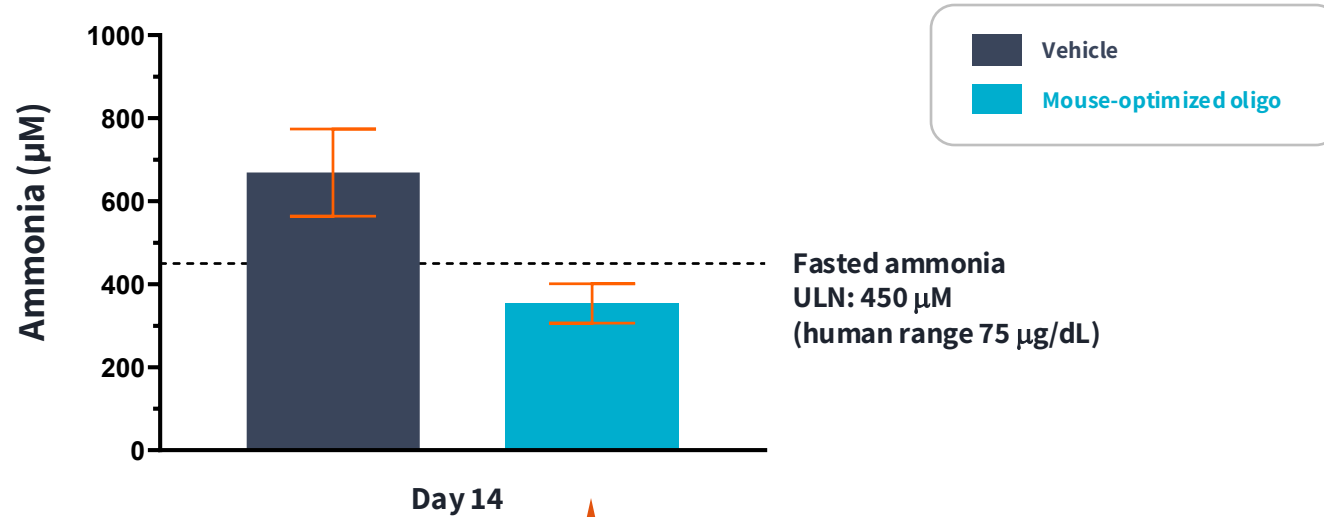
## KRRO-121 Stabilized GS in OTC-Deficient iPSC-Derived Hepatocytes



# Ammonia Reduction in OTC-Deficient Mice Challenged with Ammonia Supports Clinical Activity, Diet Liberalization

Improved Clearance in Ammonia Challenge Supports Potential to Increase Protein Intake

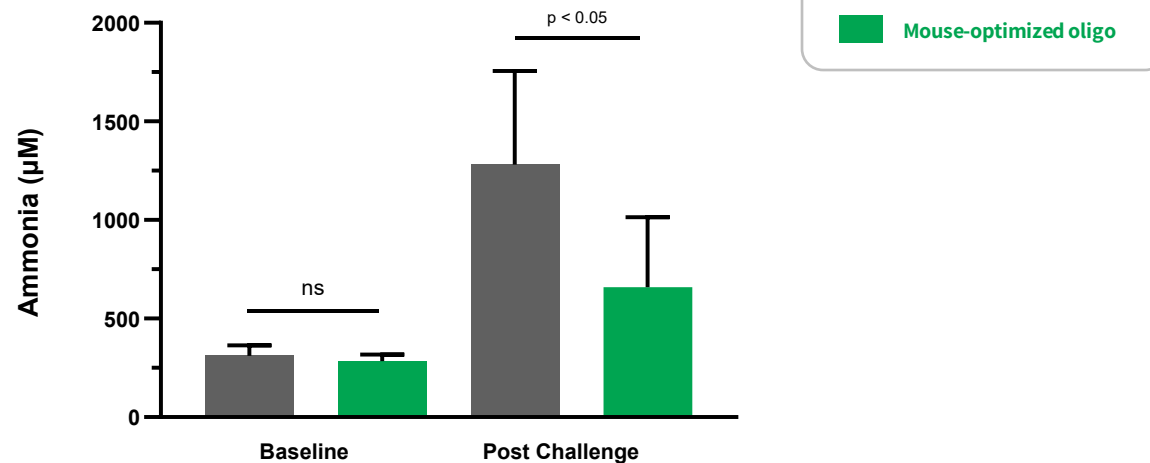
Nonsignificant Increase in Plasma Glutamine Levels



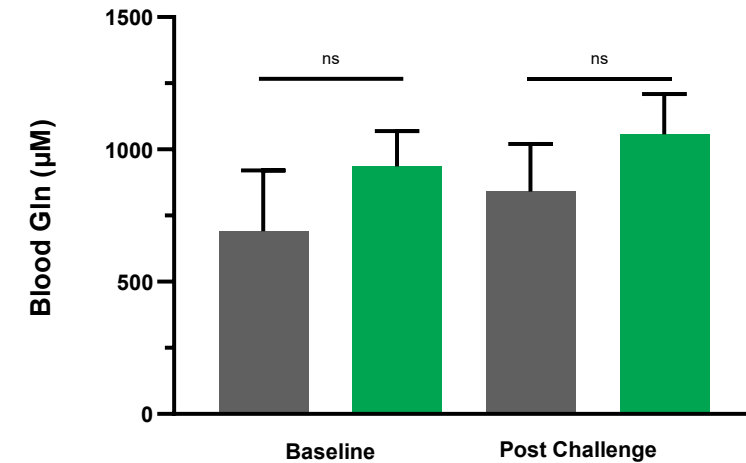
Ammonia challenge designed to model patient protein consumption

# Ammonia Reduction in CPS-1 Deficient Mice Further Validates Potential Pan-UCD Applicability and Diet Liberalization

## Reduction in Ammonia Following Ammonia Challenge



## Nonsignificant Increase in Plasma Glutamine Levels



“... Korro’s RNA editing approach targeting **glutamine synthetase** in hepatocytes has been proven to effectively **redirect excess toxic ammonia** towards the synthesis of glutamine in UCD animal models ...”

– Nicola Brunetti-Pierri MD and Leandro R. Soria PhD

# KRRO-121 Has Blockbuster Potential in Multiple Indications

## Urea Cycle Disorders (UCD)

## Hepatic Encephalopathy (HE)

**Addressable Patients**

**4,200 U.S.<sup>1</sup>**  
5,100 EU + UK<sup>1</sup>

**80,000 U.S.<sup>2</sup>**  
150,000 EU + UK<sup>3</sup>

**Market Opportunity**

**\$1.5B**

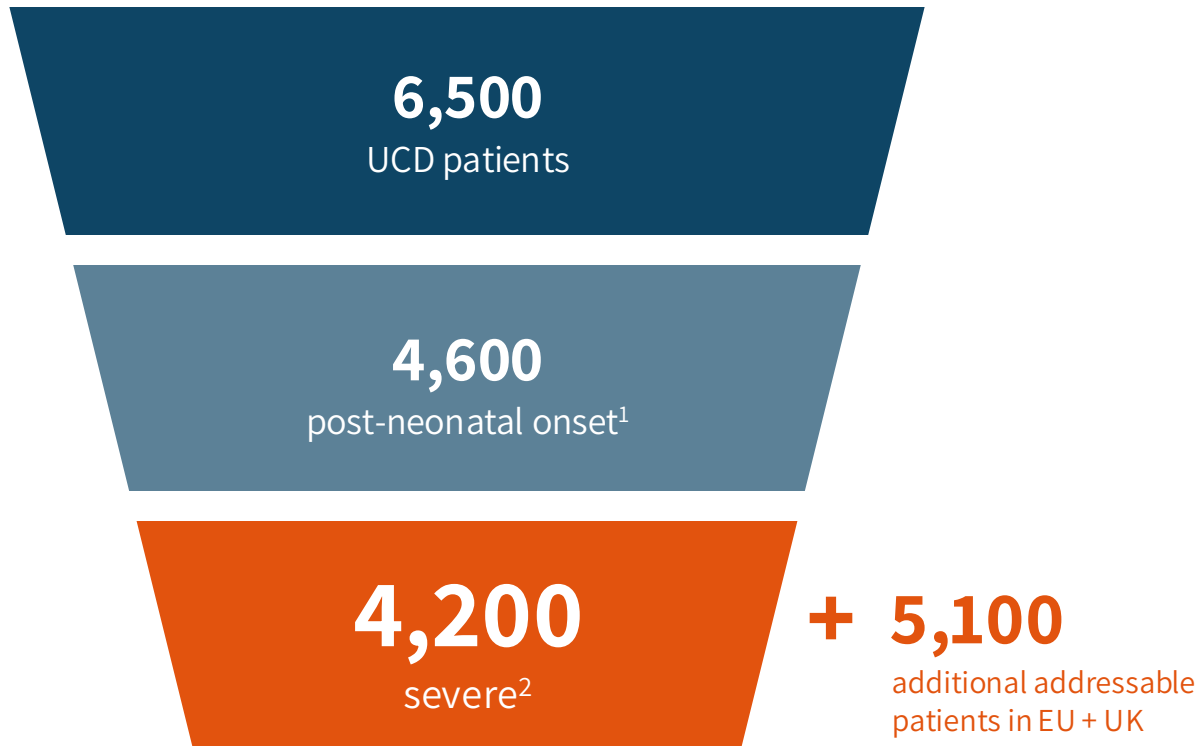
**\$2B+**

Note: 1. Severe late-onset UCD patients; 2. Patients prescribed rifaximin +/- lactulose with  $\geq 1.5x$  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: 3<sup>rd</sup> party primary market research study (April 2025); KOL interviews; GlobalData; Electronic medical records analysis (data from 2022). All figures approximate.

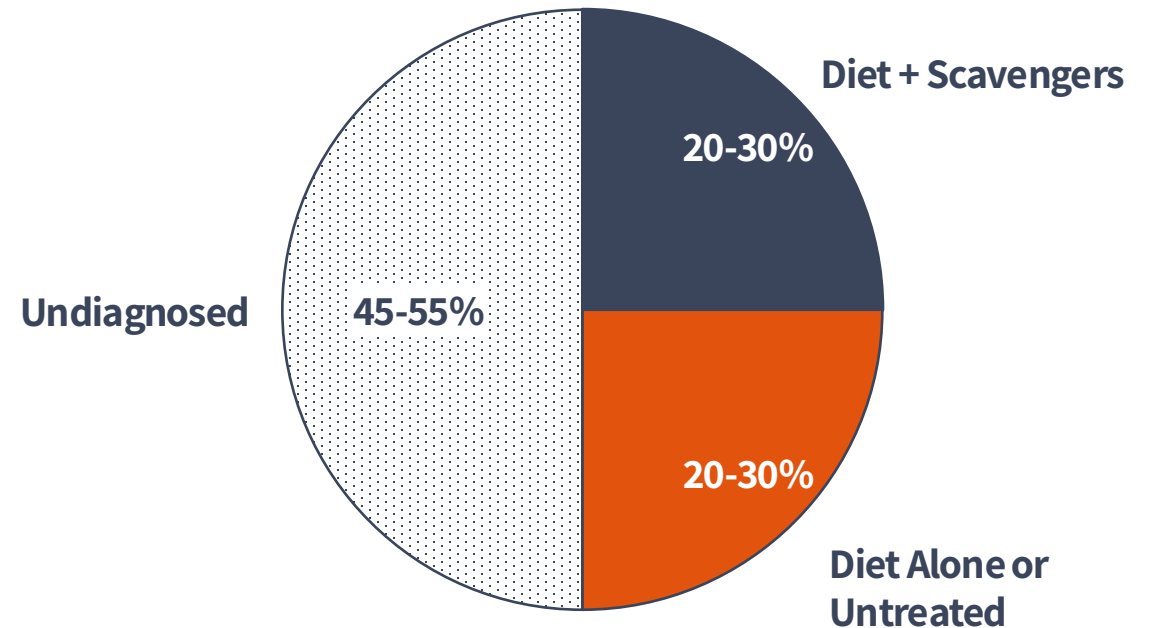
# KRRO-121 Can Potentially Address Patients Across All UCD subtypes and Expand Current Nitrogen Scavenger Market

## U.S. UCD Epidemiology



## UCD Market Share (2026)<sup>1</sup>

~10,000 late-onset patients (US + EU/UK)



# 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 ammonia<sup>1</sup>
- **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 submission**

## Key Takeaways from KRRO-121

**Significant unmet medical need**  
for controlling ammonia

**Robust scientific / genetic evidence**  
supporting GS stabilization approach

**Transformative potential**  
to impact patients

**Vision for the future**  
as a leader in activating biological pathways



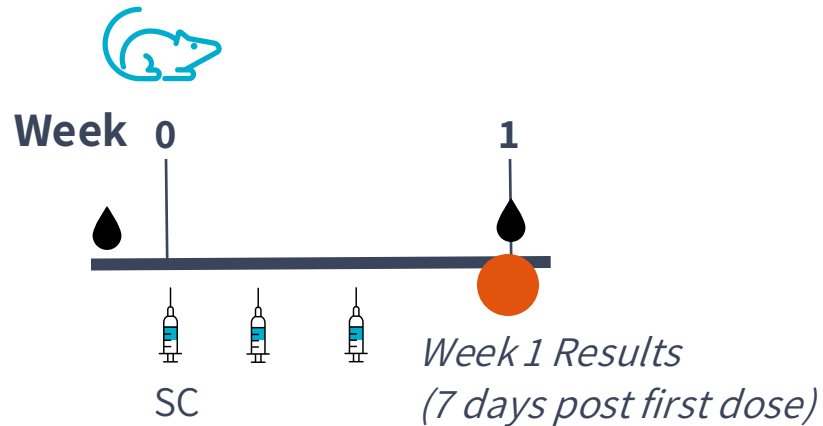
# Early Pipeline

# AATD: Achieved >90% Editing of SERPINA1 Transcript using GalNAc Delivery *in vivo*

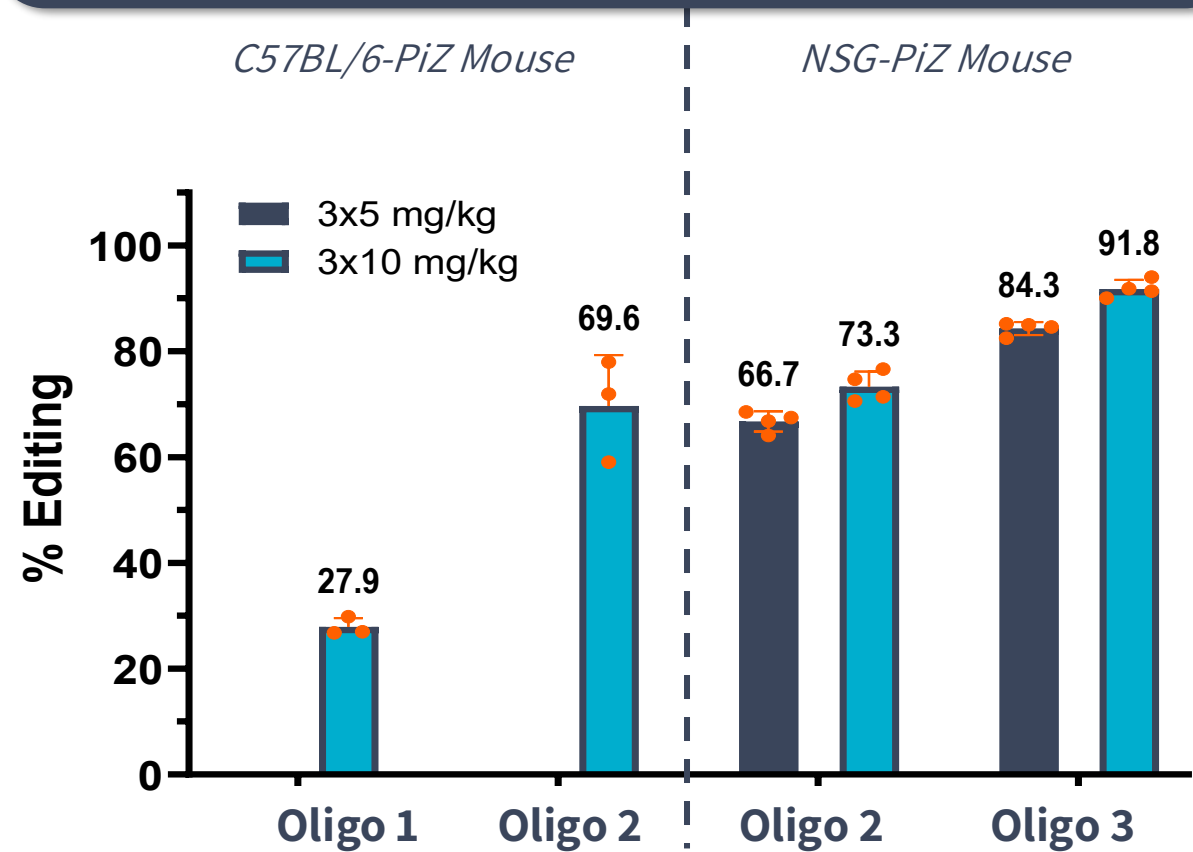
GalNAc-conjugated Oligo



Dosing: Q2Dx3 at 5 or 10 mg/kg

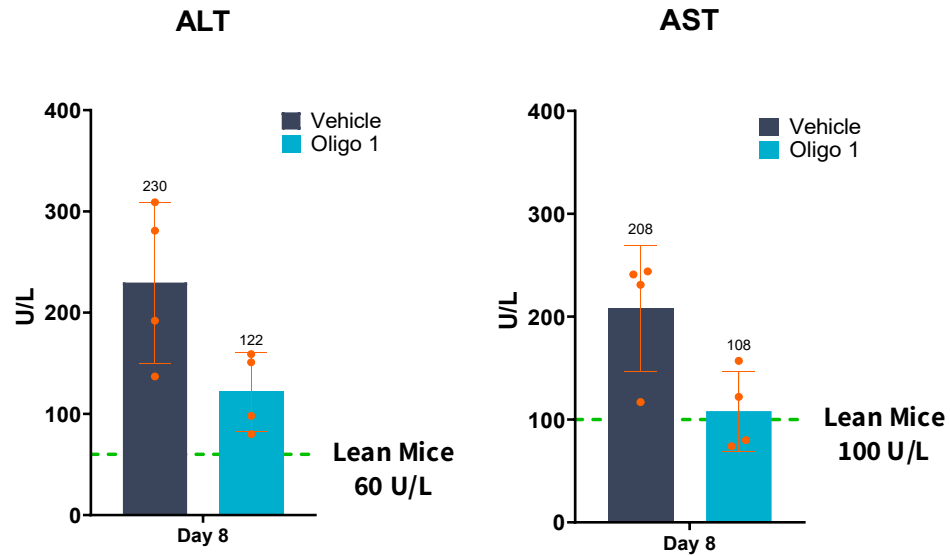


## RNA editing in PiZZ Mice (consistency in two models)

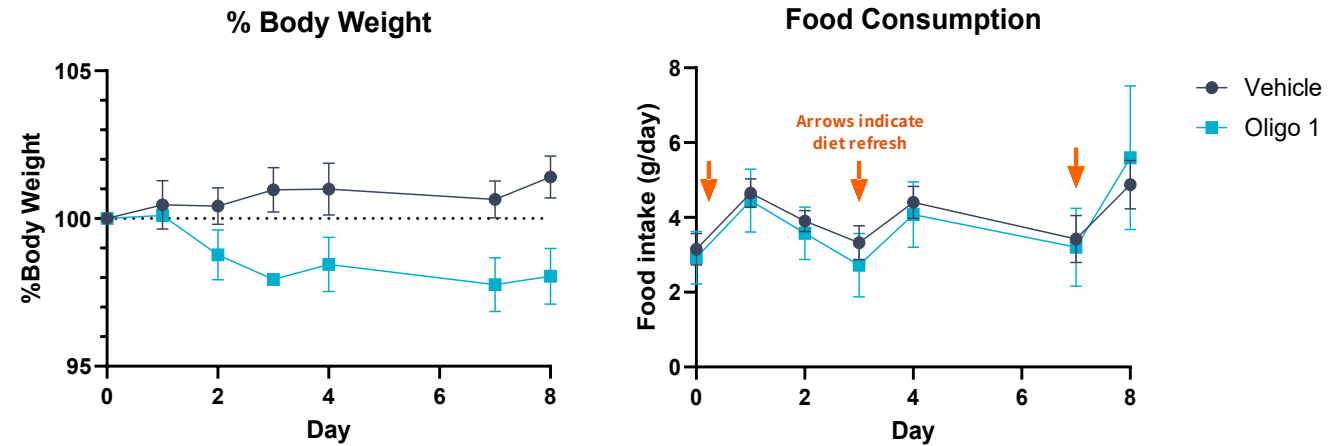


# Hepatic AMPK $\gamma$ 1 Activation Improved Liver Function in Obese Mice

## Normalizing Liver Function



## Reduction in Body Weight Despite Similar Food Intake



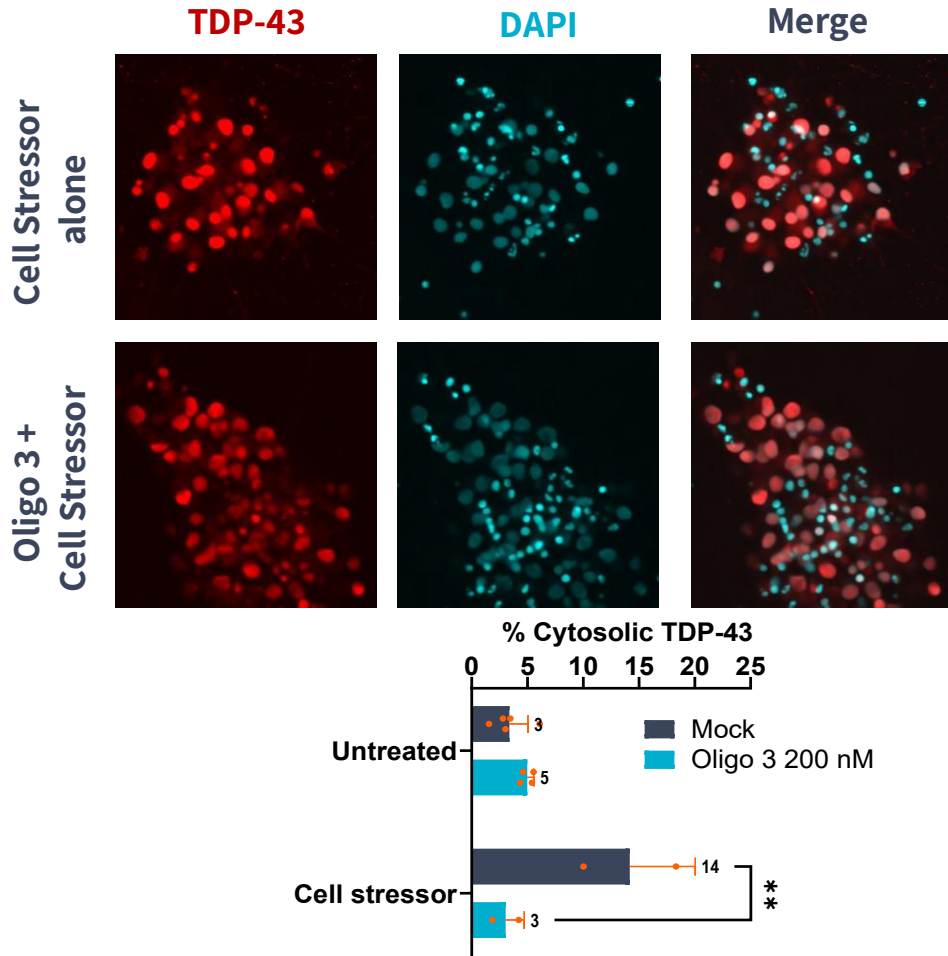
### AMPK

- 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**

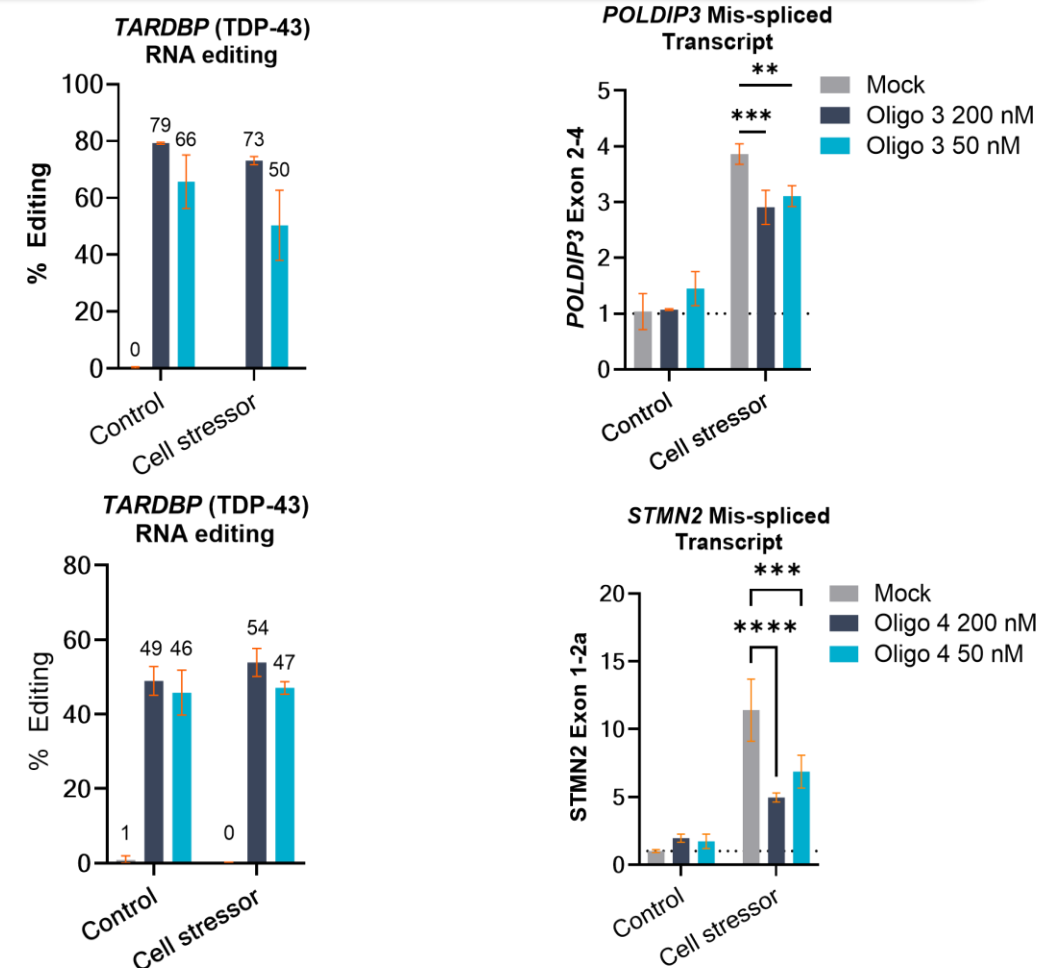
# De Novo TDP-43 Demonstrated Reduced Mis-splicing and Decreased Cytosolic Mis-localization in iPSC Motor Neurons

## Decreased Cytosolic Mis-localization of TDP-43 protein



TDP-43 signal quantified by confocal microscopy using thresholds defined from untreated controls, which were applied uniformly to all images to measure TDP-43 intensity while excluding background signal

## Reduced Mis-splicing: Maintaining STMN2 & POLDIP3





# Positioned for Value Creation in 2026 and Beyond



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DC expected for a 3<sup>rd</sup> GalNAc-conjugated liver asset in H2 2026



Cash runway into H2 '28 enabling multiple milestones <sup>1</sup>

**Edit the Message Rewrite the Future**

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