KORRO BIO

March 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) expected for GalNAc-conjugated AATD construct in Q2 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

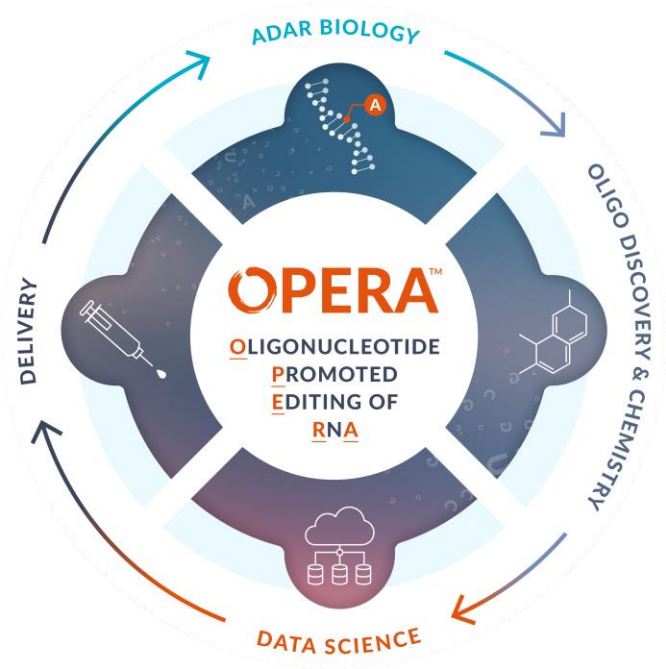
1. Cash, cash equivalents and marketable securities of $85.2 million as of December 31st, 2025

* Proforma unaudited cash, cash equivalents and marketable securities of $157 million post PIPE financing in March 2026

OPERA: Our Approach for RNA Editing to Generate Product Candidates

Expertise in ADAR biology driving potency and translation

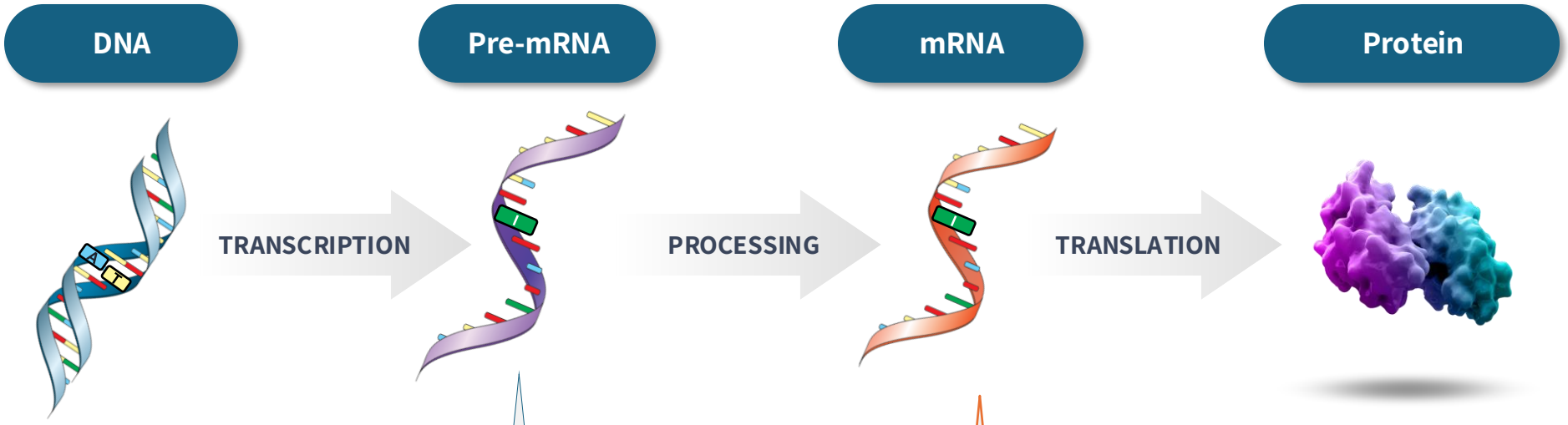
Leveraging known mechanisms to derisk Delivery



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



Edit a single A-to-I

Modifying gene expression

Edit a single A-to-I

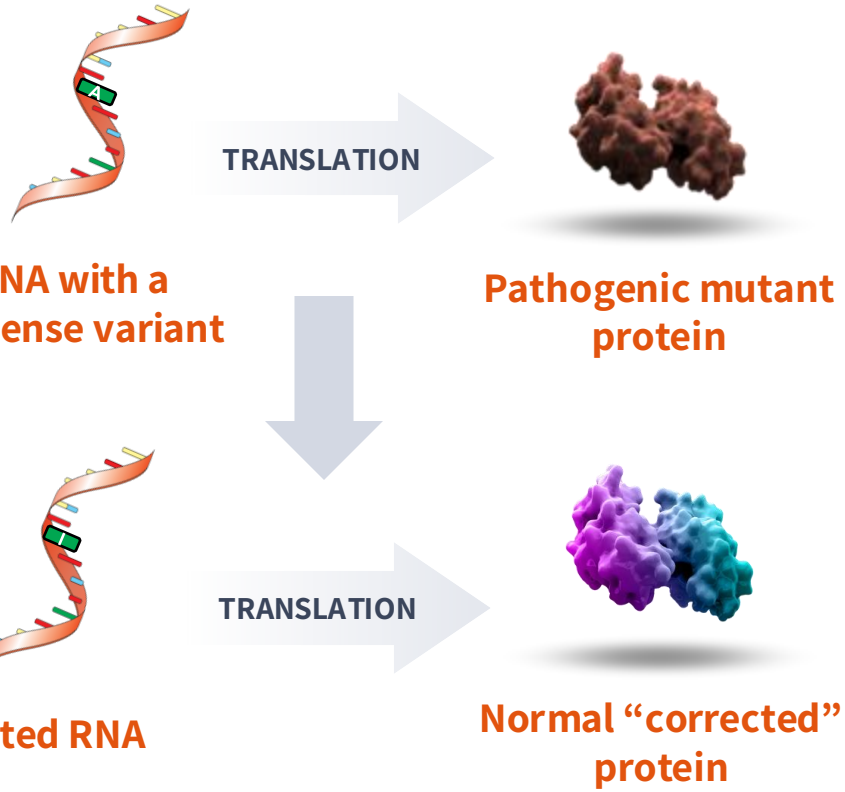
- **Repair** a G->A mutation to correct the protein
- **Generate a *de novo* protein** with preferred properties (can alter 12 amino acids)

Current Focus

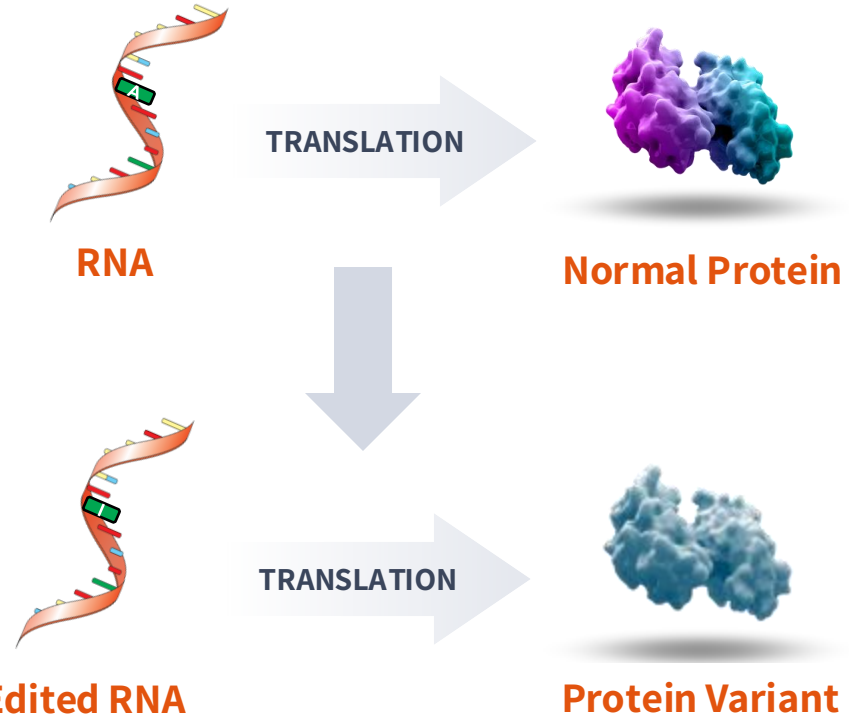
Human genetics guiding the possibilities

Pipeline Programs Primarily Focused on Generation of Protein Variants

Repair the Protein



Modulate Protein Function (Activate pathway)



Examples of Repair = E342K AATD, 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)	AMPKγ1				
Overcome LoF and GoF [1]	Amyotrophic lateral sclerosis (ALS)	Intrathecal (IT)	TDP43				

Protein variant creation

Protein repair

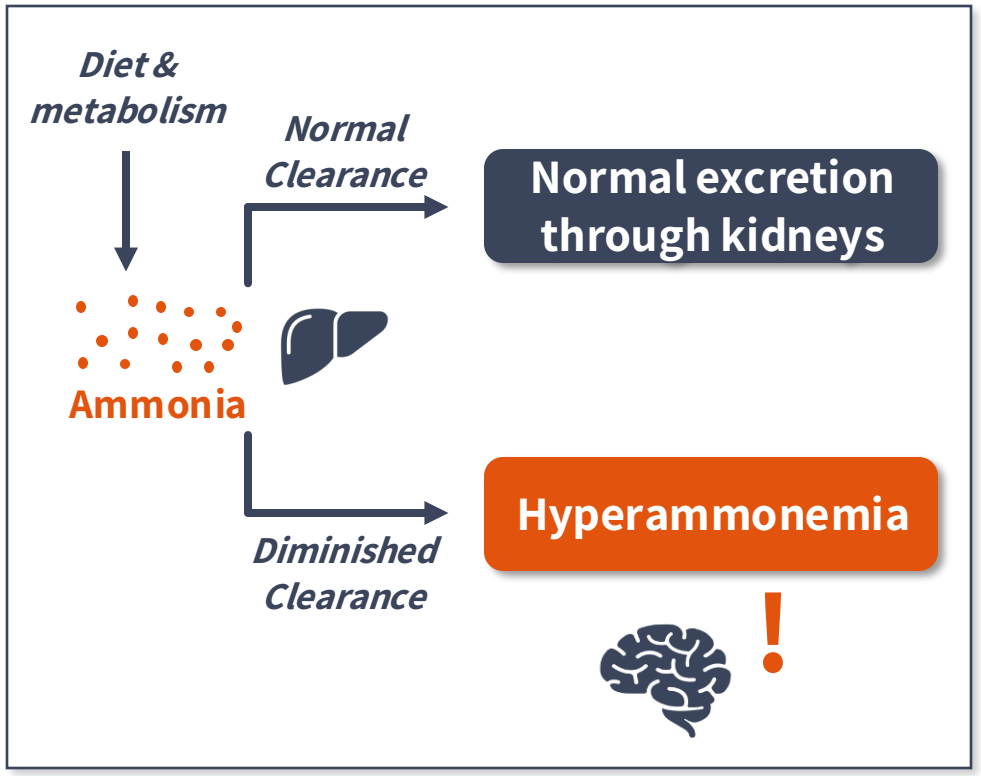
[1] *De Novo* protein variant to prevent toxic gain-of-function (GoF) with TDP43 aggregation, and continue downstream signaling by overcoming toxic loss-of-function (LOF)

GS = Glutamine Synthetase; AAT = Alpha-1 antitrypsin; AATD = AAT deficiency; AMPK γ1 = 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



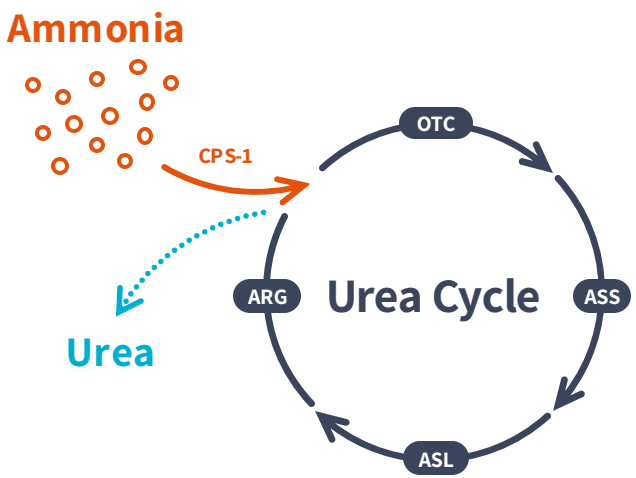
- **High ammonia leads to:**
  - Neurological impairment
  - Frequent hospitalization
  - Highly restricted diet
  - Elevated infection risk
- Can be caused by cirrhosis or urea cycle dysfunction
- Clinical studies have shown benefit of lowering ammonia in multiple indications

Novel approach to reduction of ammonia could have a **profound impact** to patient lives

Source: 3rd party primary market research study and analysis (April 2025); KOL interviews

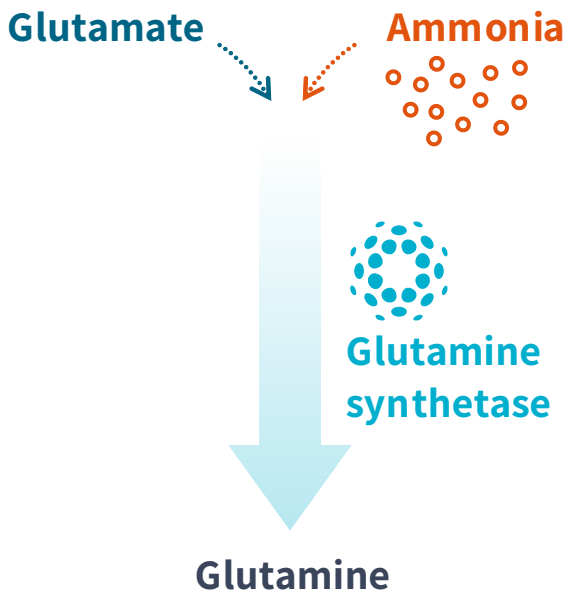
Two Complementary Pathways for Ammonia Clearance: Urea Cycle and Glutamine Synthetase (GS)

Urea Cycle



Expressed primarily in liver

Glutamine Synthetase

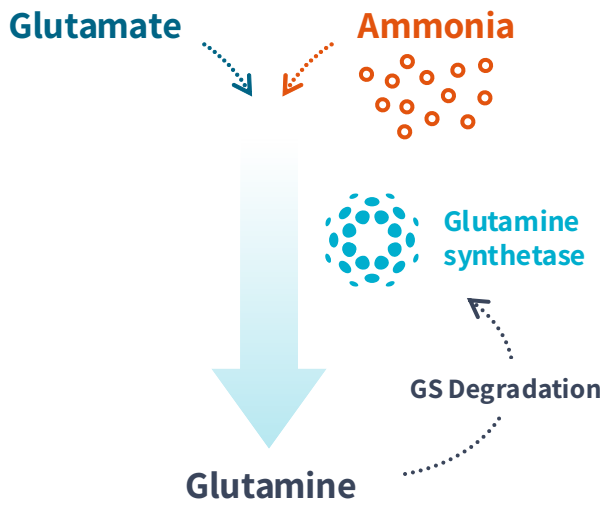


Expressed in many tissues, including liver, brain, and muscle

Source: Inoue at al., Seizure (2015); Bennet et al., Child Neurology Open (2020); Jones et al., The American Journal of Human Genetics (2024); Soria et al., J Inherit Metab Dis (2019); KOL interviews

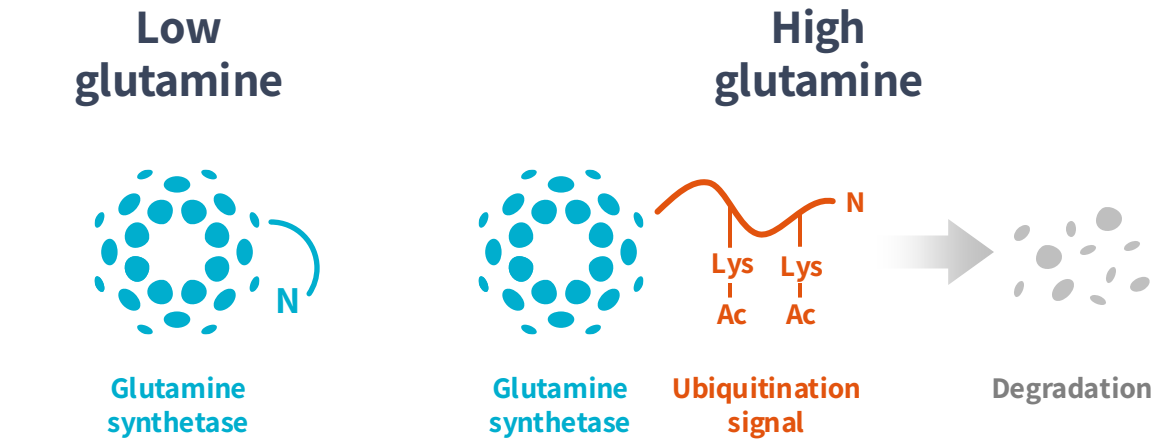
Degradation of GS Controlled by Levels of Glutamine

Glutamine Drives Degradation of GS



GS degraded when glutamine rises, reducing ammonia clearance capacity

Degradation Mechanism: Acetylation of Key N-terminal Residues

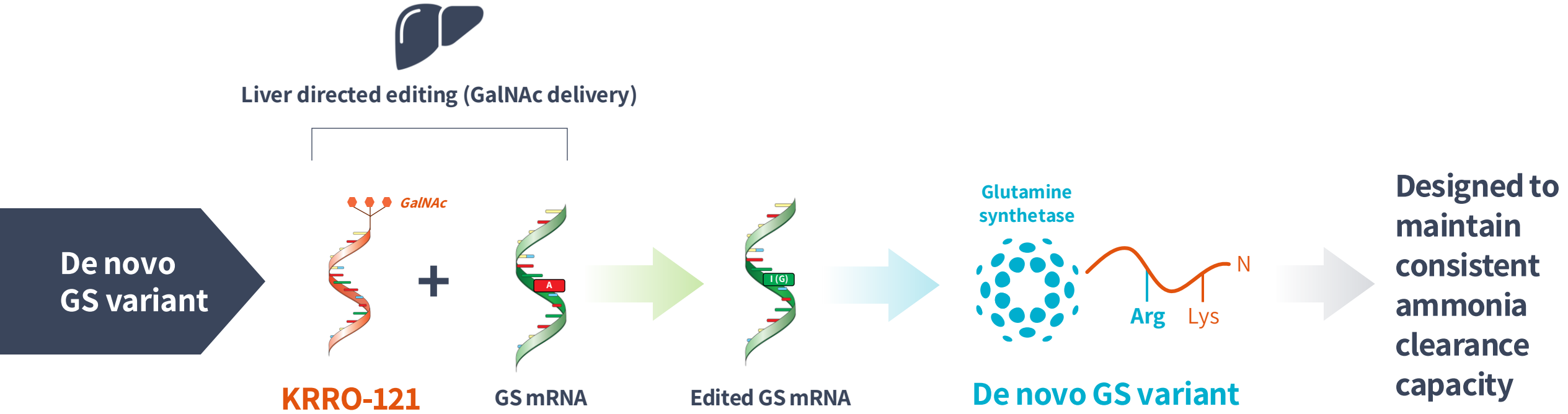


No lysine acetylation, GS is stable

Acetylation of lysine residues, leading to ubiquitination and protein degradation

Source: Van Nguyen et al., Mol Cell (2017)

Our Approach: Liver-specific, GalNAc-ASO to Generate a Stable GS Variant



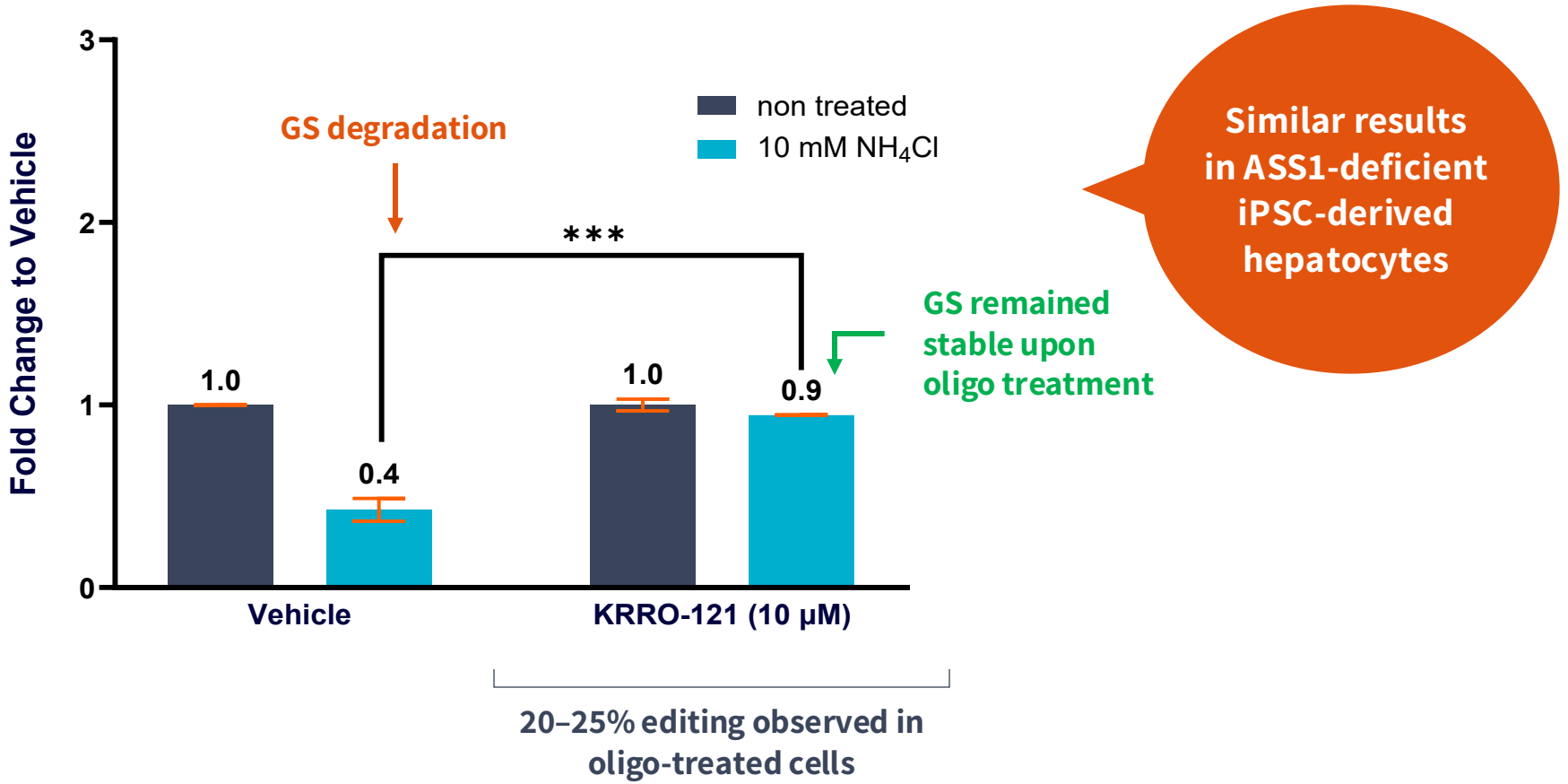
KRRO-121: GalNAc-conjugated oligonucleotide designed for liver-specific RNA editing of GS to enhance ammonia clearance capacity

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

OTC iPSC

KRRO-121

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

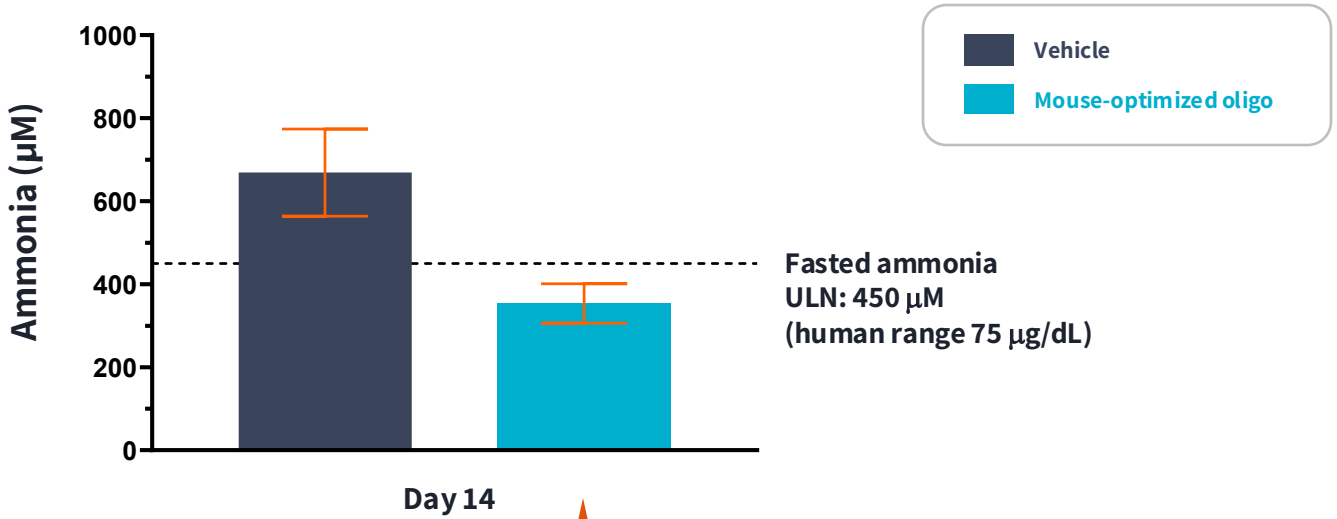


Note: OTC D175V human iPSC-derived hepatocytes differentiated for 14 days, then treated with oligo for 48 hours where indicated (10 mM NH_4Cl added after 24 hours where indicated). GS concentration measured at conclusion of 48-hour incubation.

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

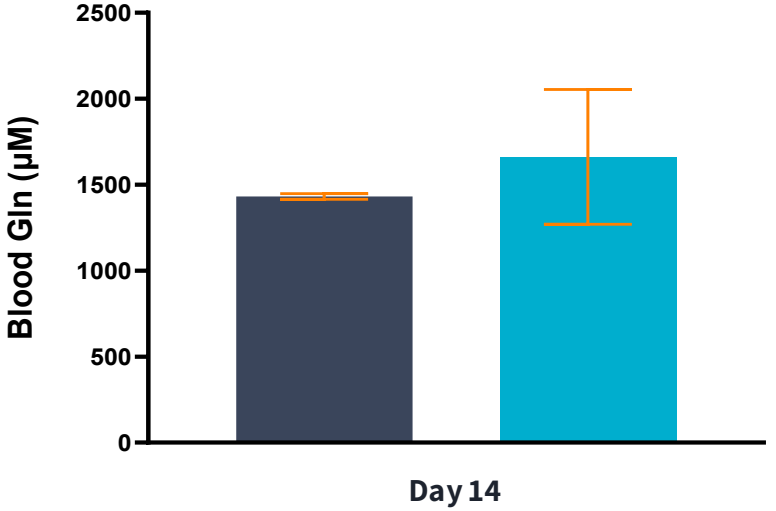
OTC spf/ash

Improved Clearance in Ammonia Challenge Supports Potential to Increase Protein Intake



Ammonia challenge designed to model patient protein consumption

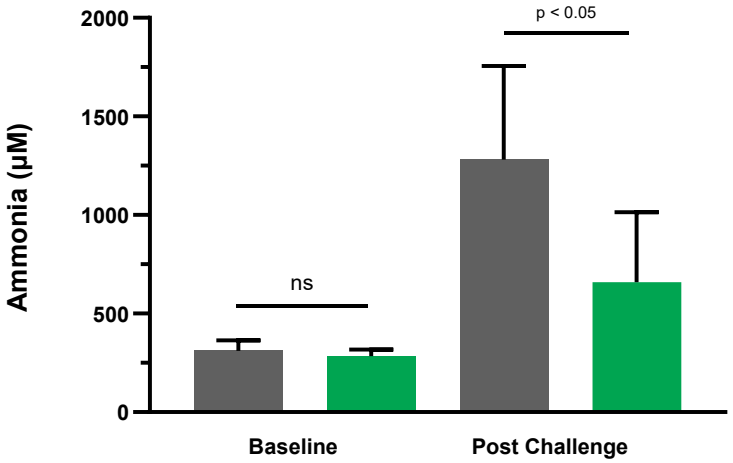
Nonsignificant Increase in Plasma Glutamine Levels

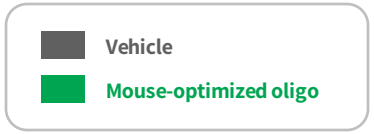


Note: Vehicle or Mouse-optimized oligo dosed at 10 mg/kg-SC daily on Days 0-4. Ammonia and glutamine measured following ammonia challenge (150 mg/kg) on Day 14

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

Reduction in Ammonia Following Ammonia Challenge





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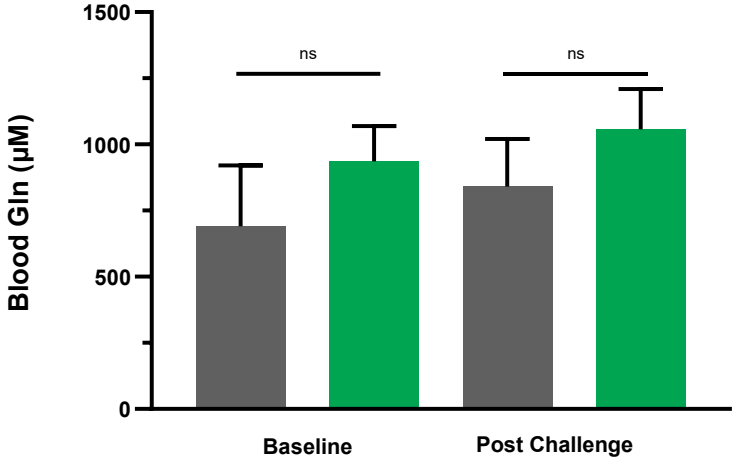


"… Korro's RNA editing approach targeting **glutamine synthetase** in hepatocytes has been proven to effectively **redirect excess toxic ammonia** towards the synthesis of glutamine in UCD animal models …"

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

Note: Vehicle or Mouse-optimized oligo dosed at 10 mg/kg-SC daily on Days 0-4. Ammonia and glutamine measured following ammonia challenge (150 mg/kg) on Day 8

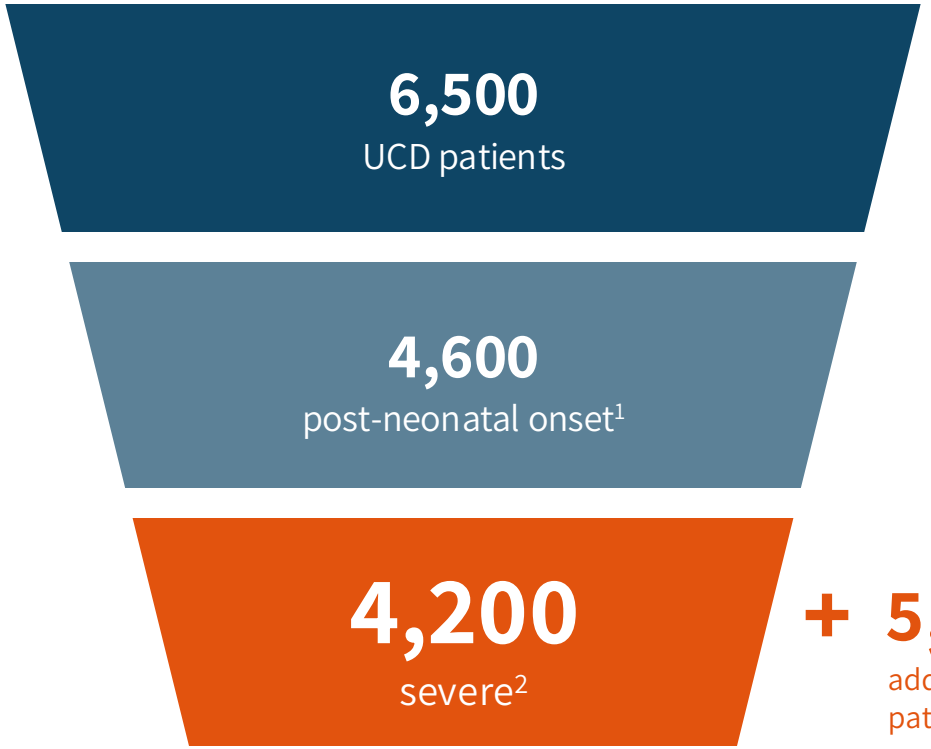
KRRO-121 Has Blockbuster Potential in Multiple Indications

	Urea Cycle Disorders (UCD)	Hepatic Encephalopathy (HE)
Addressable Patients	4,200 U.S.[1] 5,100 EU + UK[1]	80,000 U.S.[2] 150,000 EU + UK[3]
Market Opportunity	$1.5B	$2B+

Note: 1. Severe late-onset UCD patients; 2. Patients prescribed rifaximin +/- lactulose with ≥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: 3rd 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

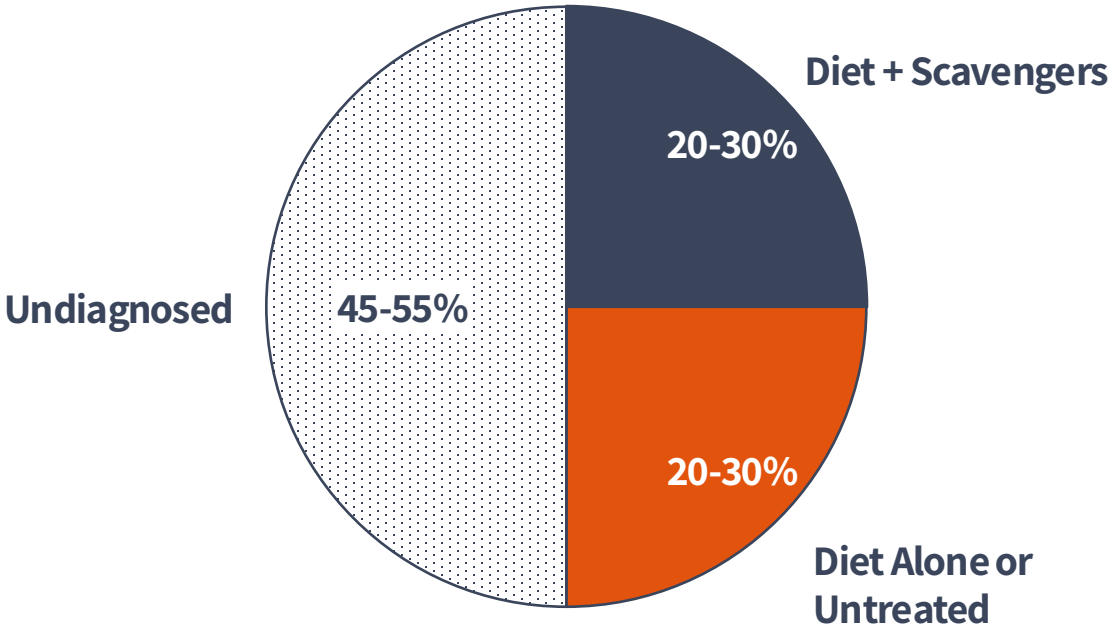
U.S. UCD Epidemiology



+ **5,100**
additional addressable patients in EU + UK

UCD Market Share (2026)[1]

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



Note: 1. Onset of symptoms at age >1 month; 2. Severe defined as symptomatic patients expected to benefit from pharmacological therapy. Carbaglu (NAGS) and Loargys (ARG) not shown (collectively addresses <5% of market). Supplements not shown.
Source: 3rd party primary market research study (April 2025); KOL interviews; GlobalData. All figures approximate. Martin-Hernandez et al., Nutrients (2025); Burrage et al., Mol Genet Metab (2014); Company estimates; Analyst reports; KOL interviews

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[1]
- **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 variant** 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

Note: 1. As demonstrated using a surrogate mouse-optimized oligo

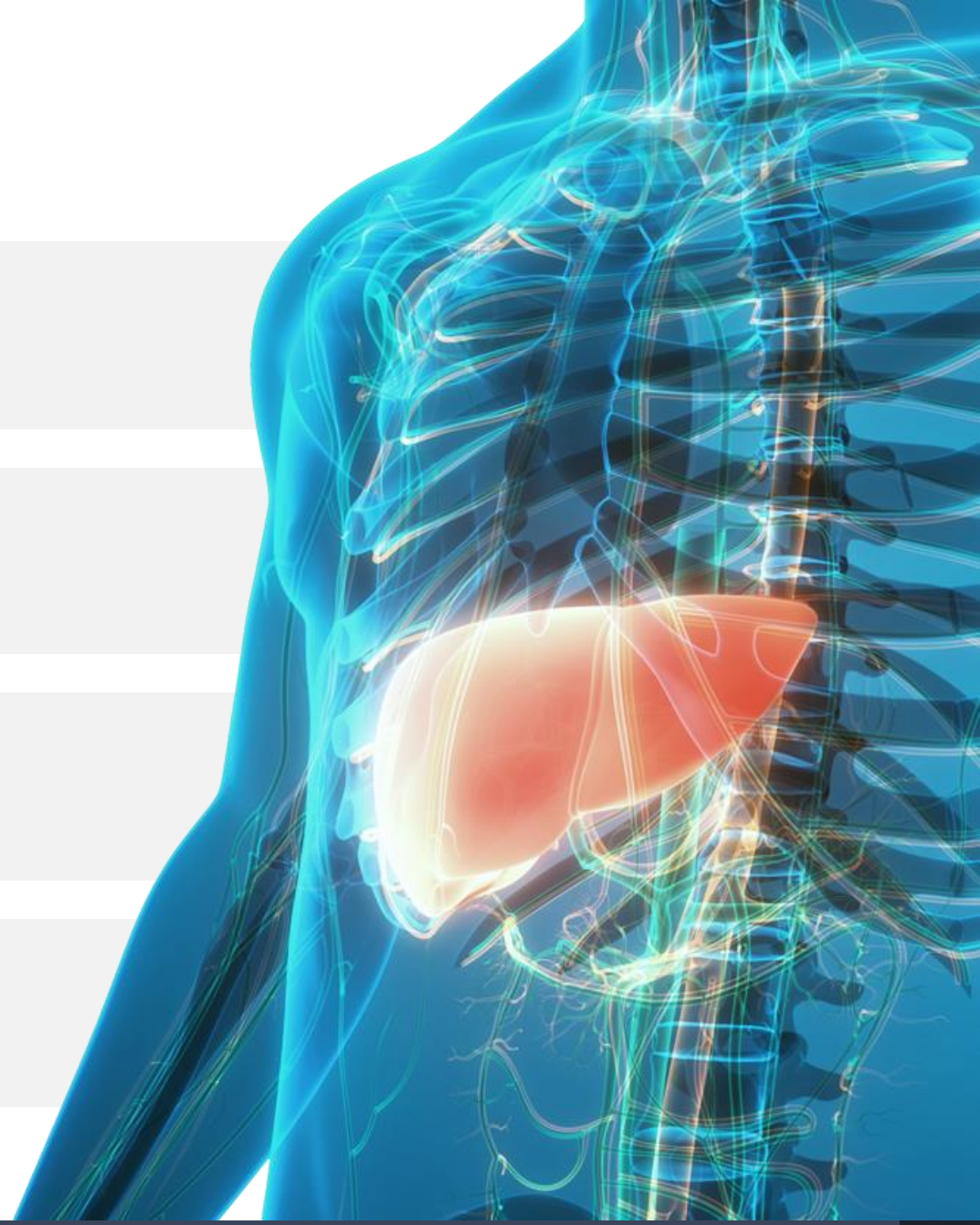
Key Takeaways from KRRO-121

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

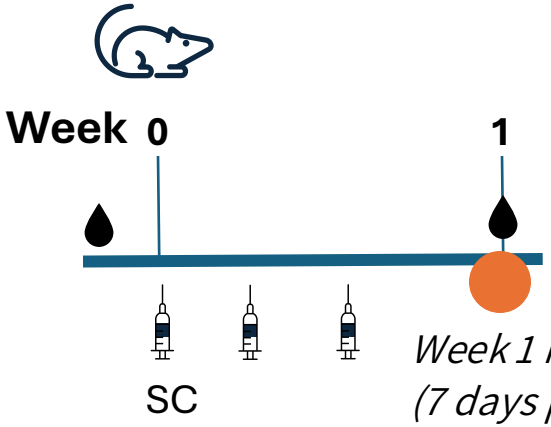


Early Pipeline

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

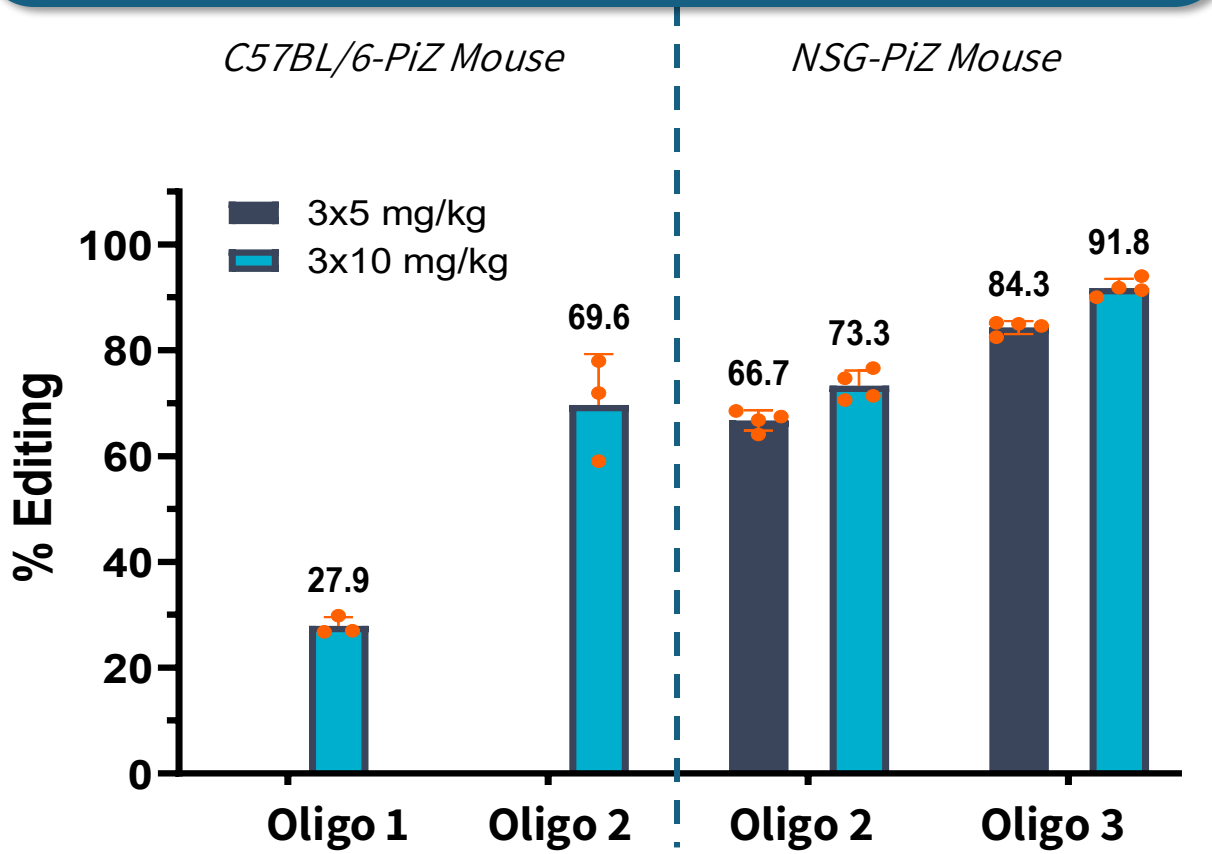
GalNAc-conjugated ASO

Dosing: Q2Dx3 at 5 or 10 mg/kg

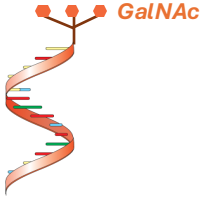


Week 1 Results
(7 days post first dose)

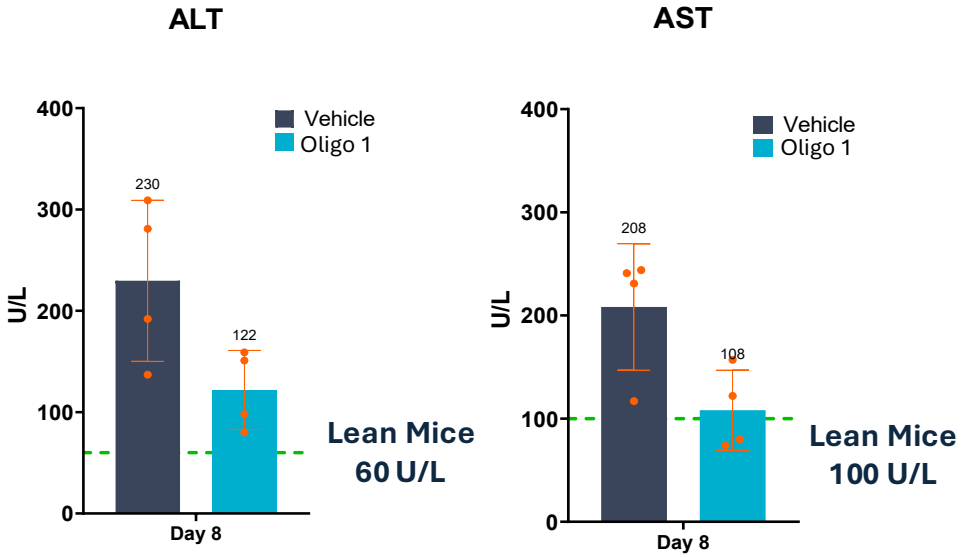
RNA editing in PiZZ Mice (consistency in two models)



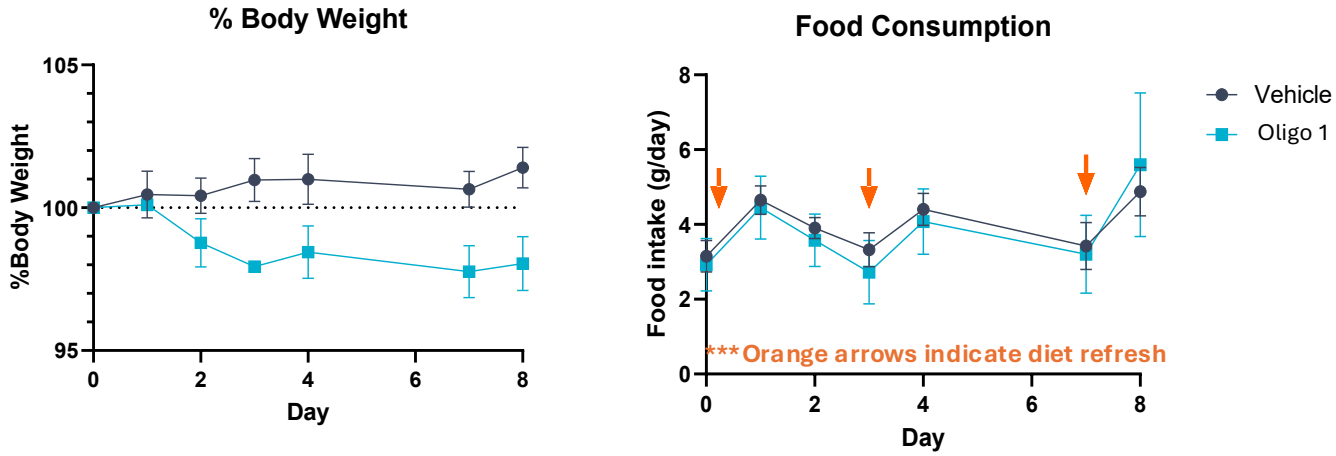
Hepatic AMPKγ1 Activation Improved Liver Function in Obese Mice



Normalizing Liver Function



Reduction in Body Weight Despite Similar Food Intake



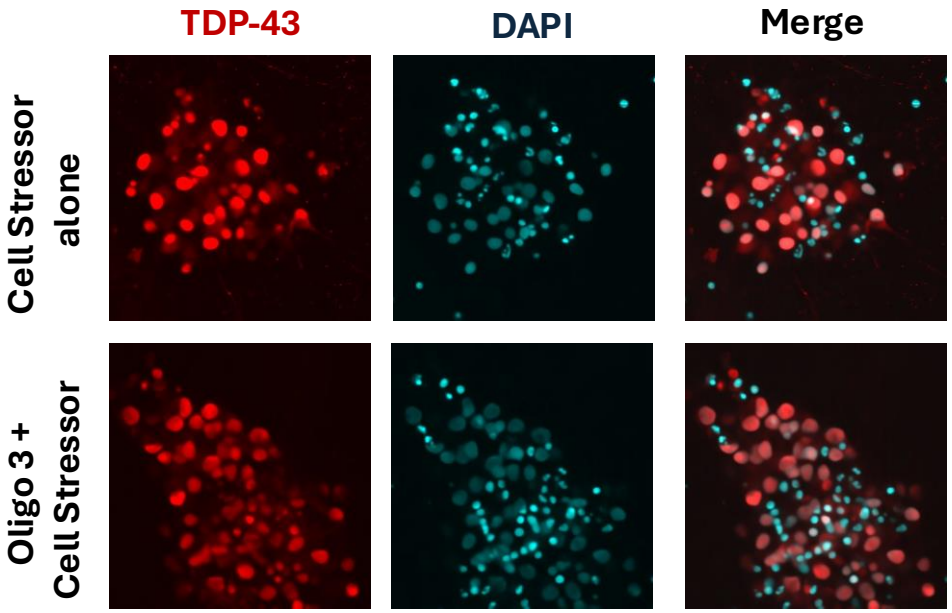
~20% editing was sufficient to normalize liver function and reduce body weight in mice

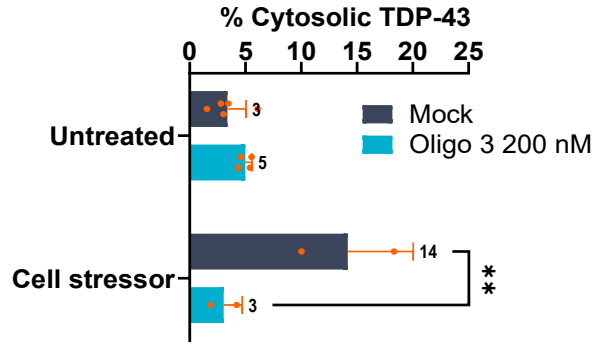
- Activation of the Master Metabolic Regulator
- Designed to Improve Liver Function
- Potentially Restores Metabolic Signaling Without Affecting Food Intake

Diet-Induced-Obese (DIO) mice were treated daily for 5 days with GalNAc-conjugated oligonucleotide at 10mg/kg and were sacrificed on day 8.

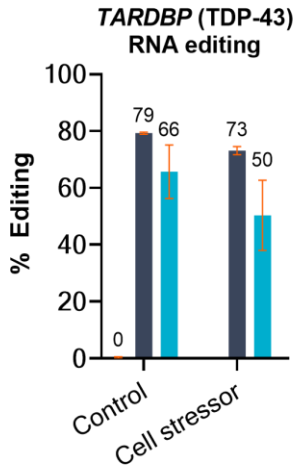
TDP-43 Variant Demonstrated Reduced Mis-splicing and Decreased Cytosolic Mis-localization in iPSC Motor Neurons

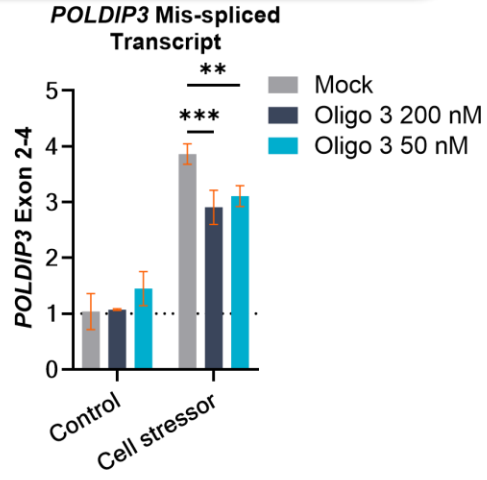
Decreased Cytosolic Mis-localization of TDP-43 protein

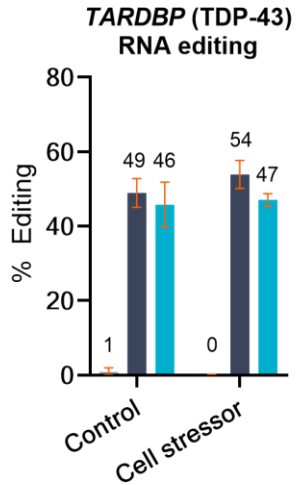


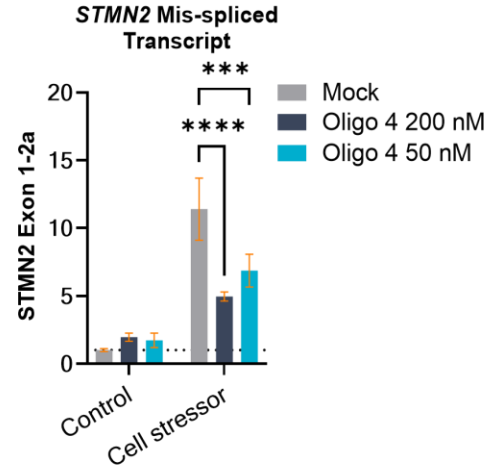


Reduced Mis-splicing: Maintaining STMN2 & POLDIP3









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

Positioned for Value Creation in 2026 and Beyond

- Regulatory filing for KRRO-121 anticipated in H2 **2026**
- DC expected for GalNAc-conjugated AATD construct in Q2 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 $85.2 million as of December 31st, 2025

* Proforma unaudited cash, cash equivalents and marketable securities of $157 million post PIPE financing in March 2026