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

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

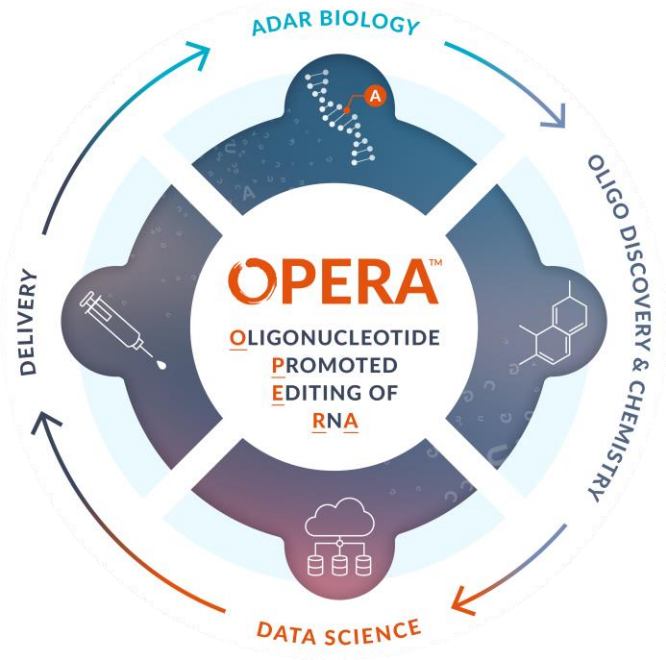
Potential for partnership across our pipeline

1. Cash, cash equivalents and marketable securities of $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

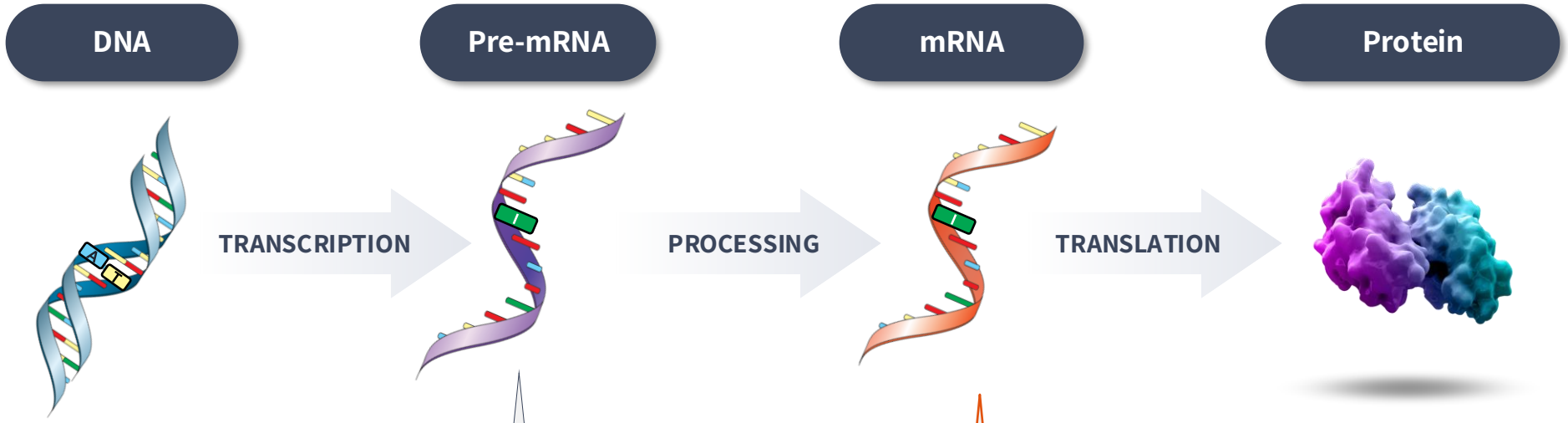
Leveraging known mechanisms to derisk Delivery



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



Modifying gene expression

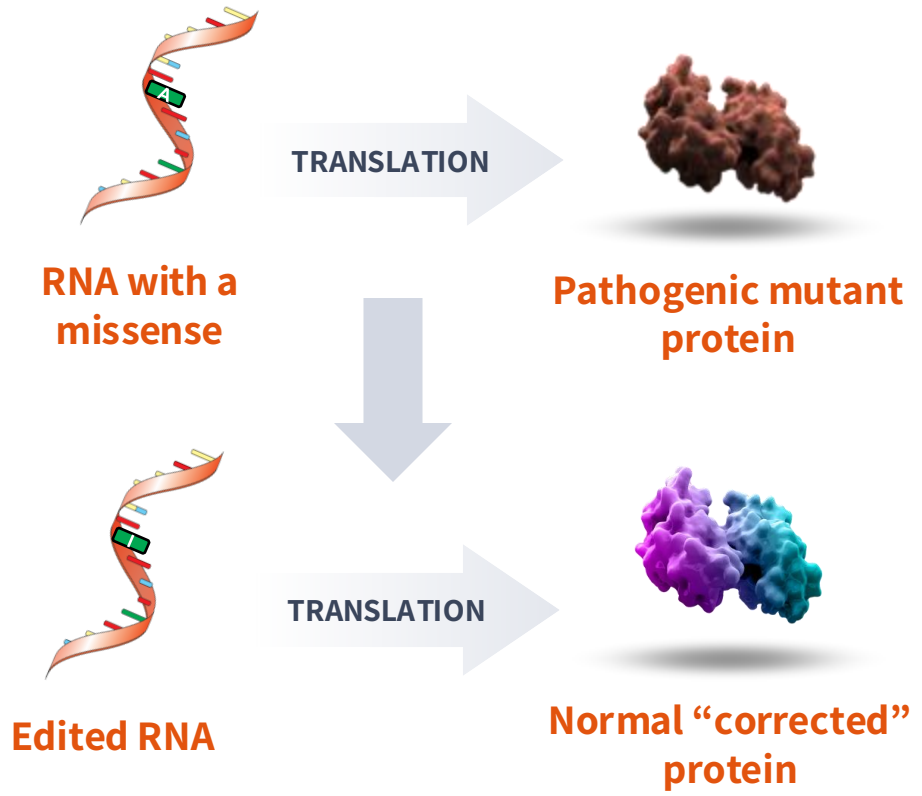
- **Repair** a G->A mutation to correct the protein
- **Generate a *de novo* protein** with preferred properties (can alter 12 amino acids)

Current Focus

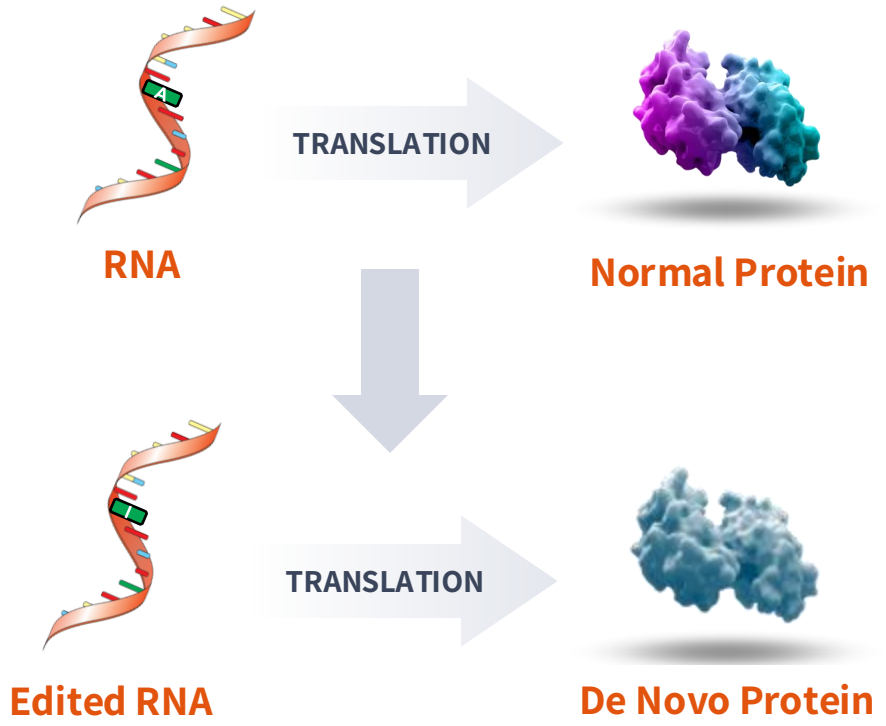
Human genetics guiding the possibilities

Pipeline Programs Primarily Focused on Generation of De Novo Proteins

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	KRRO-111 AATD	GalNAc (SC)	AAT				
Allosteric Activator	Longevity (Liver)	GalNAc (SC)	AMPKγ1				
Overcome LoF and GoF [1]	Amyotrophic lateral sclerosis (ALS)	Intrathecal (IT)	TDP43				

De Novo protein creation

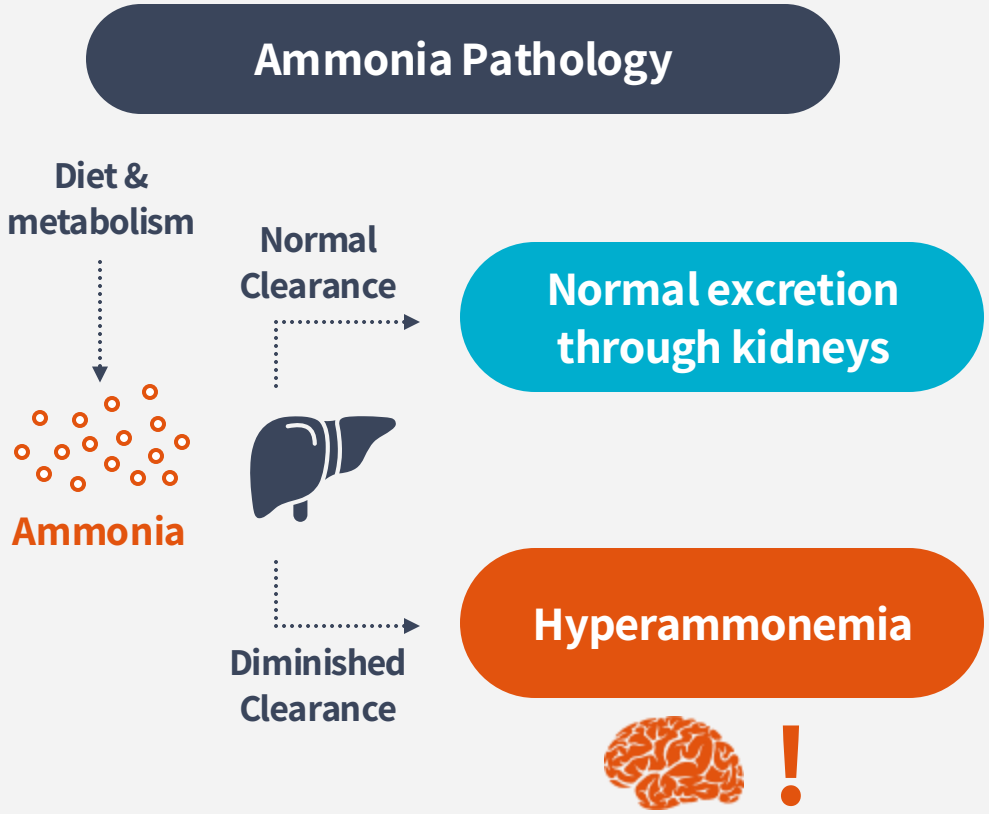
Protein repair

[1] *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 γ1 = 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

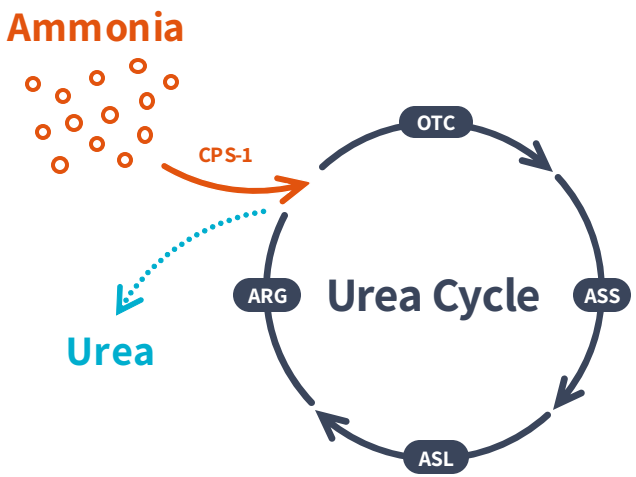


- **High ammonia** leads to:
  - Neurological impairment, potentially permanent
  - Frequent hospitalization
  - Highly restricted diet
  - Elevated infection risk
  - Additional non-neurological complications
- Can be caused by **cirrhosis or urea cycle dysfunction**
- Clinical studies have shown benefit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

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

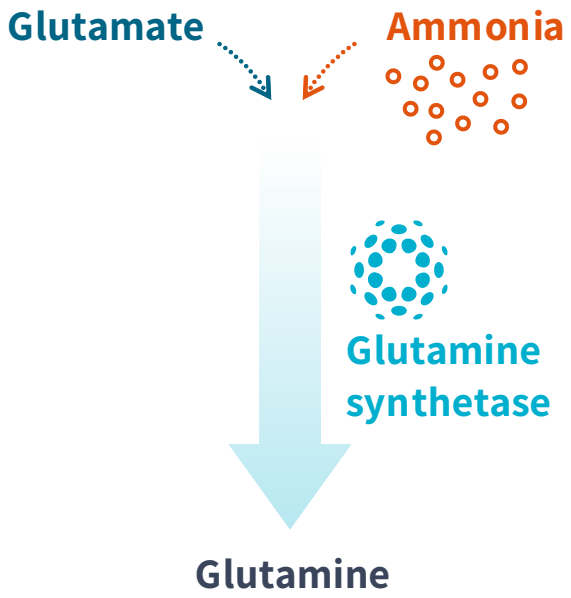
Two Complementary Pathways for Ammonia Clearance: Urea Cycle and Glutamine Synthetase (GS)

Urea Cycle



Expressed primarily in liver

Glutamine Synthetase

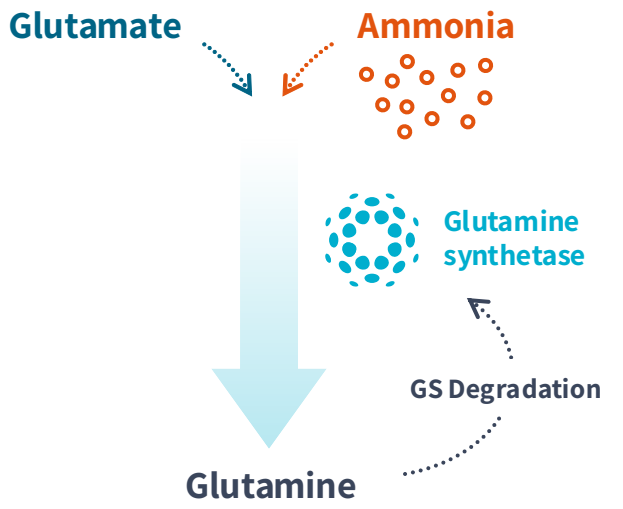


Expressed in many tissues, including liver, brain, and muscle

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

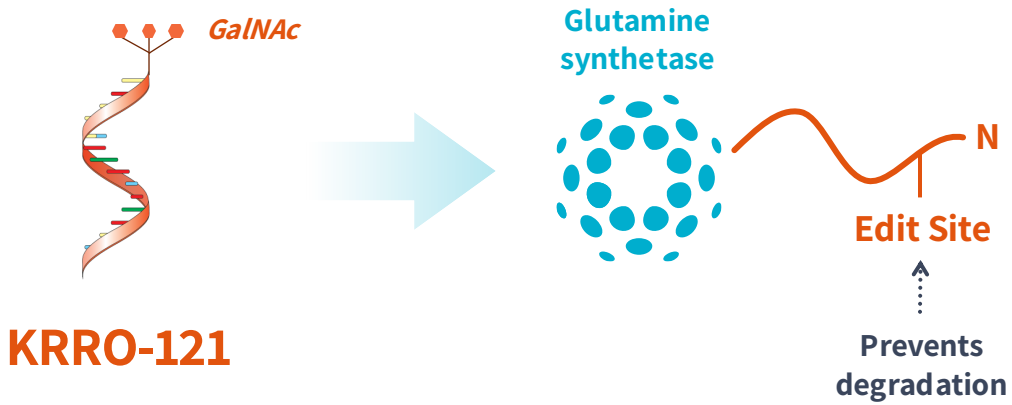
KRRO-121 is a GalNAc-conjugated Oligonucleotide Designed for Liver-specific RNA Editing of GS to Enhance Ammonia Clearance Capacity

Glutamine Drives Degradation of GS



GS degraded when glutamine rises, reducing ammonia clearance capacity

Our Approach

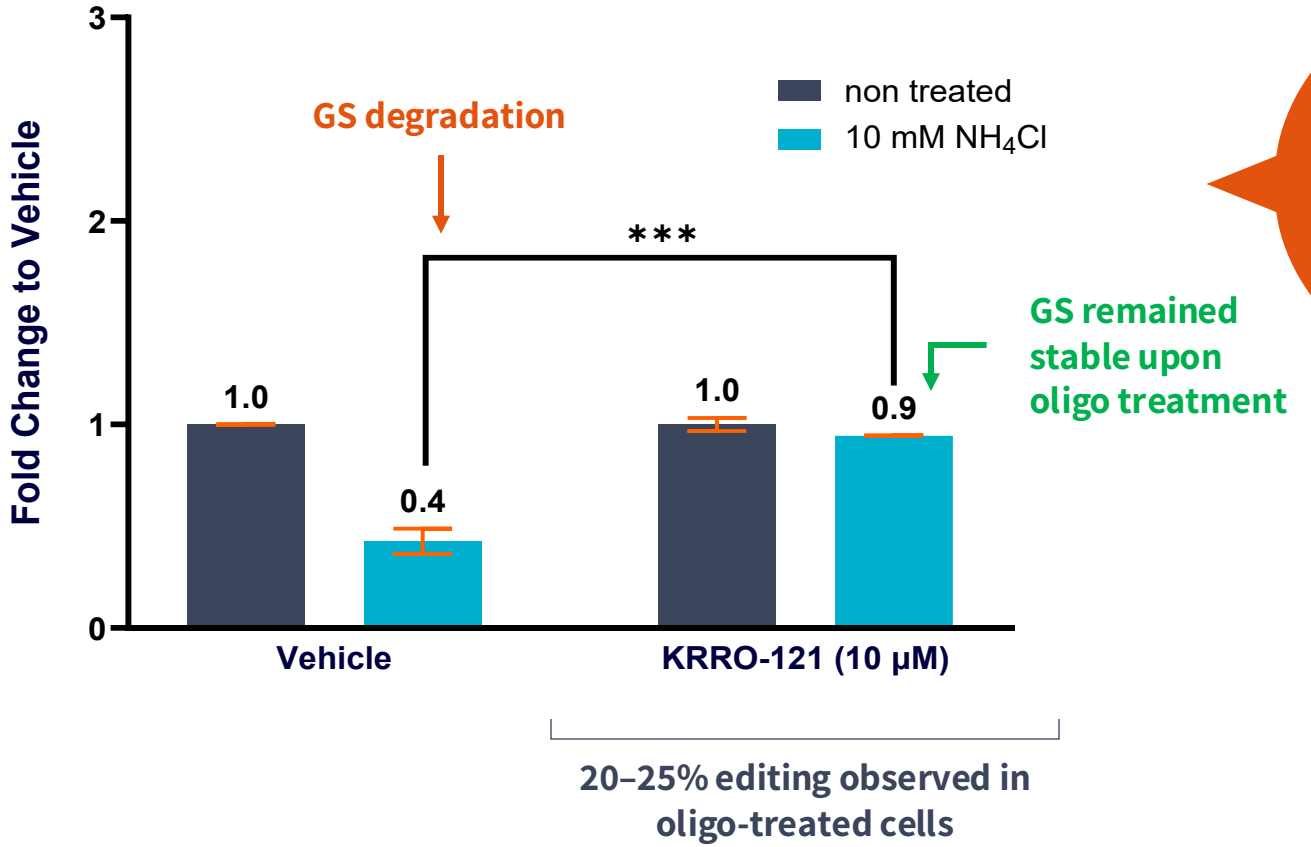


Liver-specific GalNAc-conjugated Oligo Generates Stabilized, De Novo GS

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

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

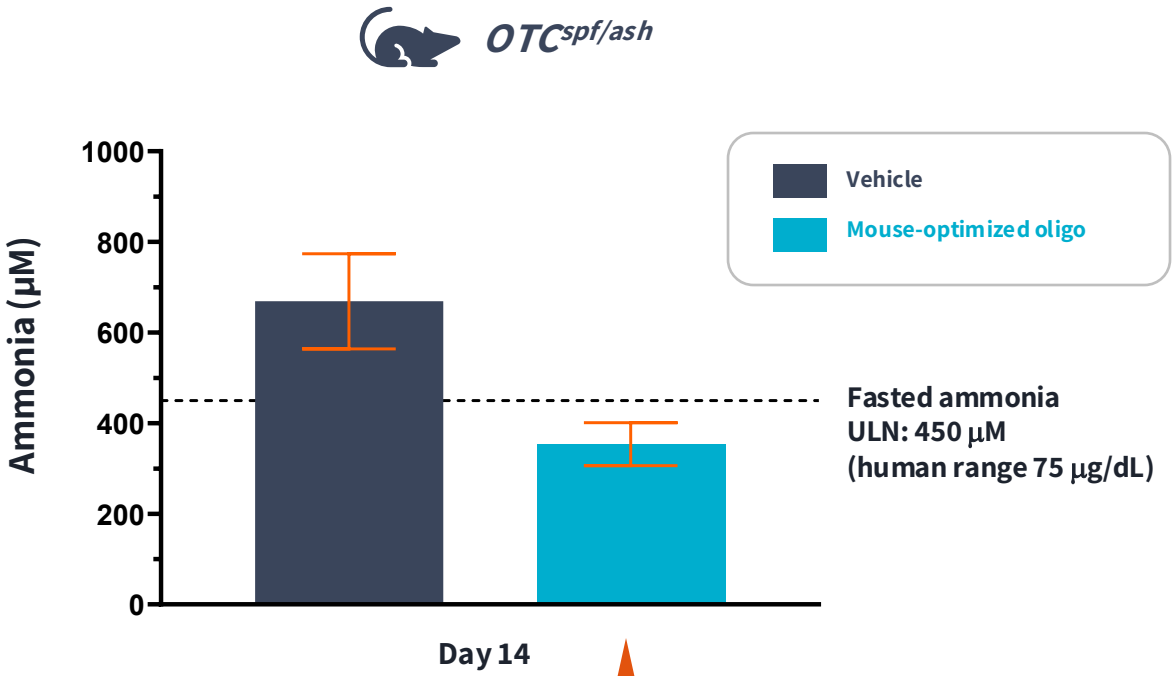
KRRO-121 Stabilized GS in OTC-Deficient iPSC-Derived Hepatocytes

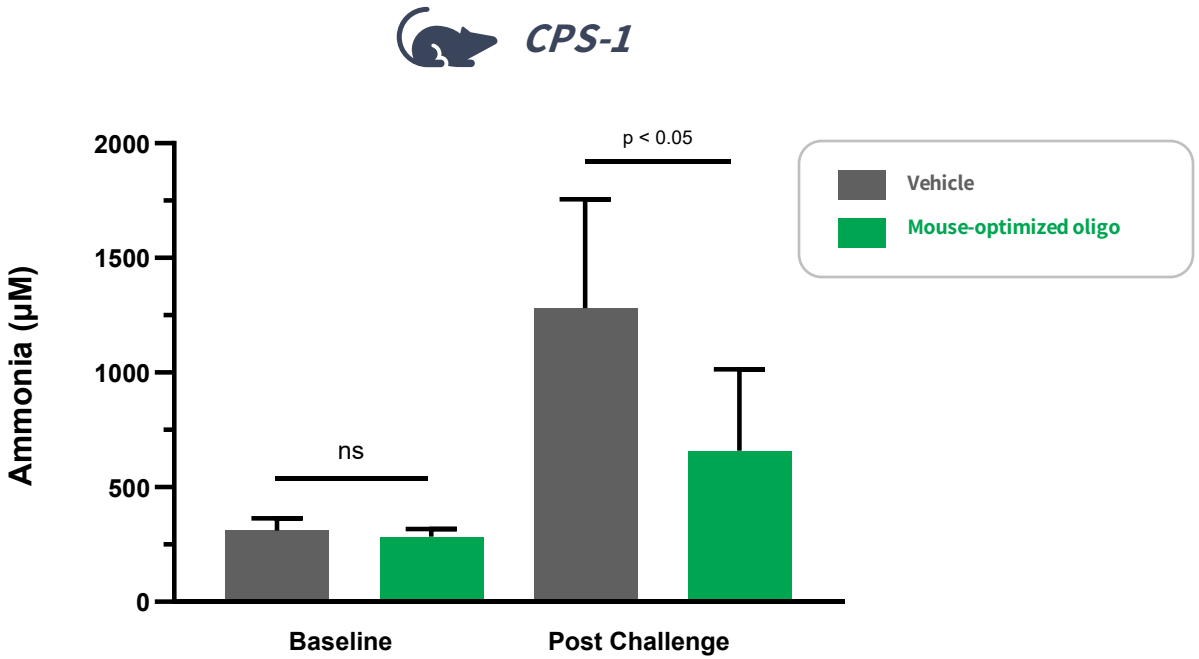


Similar results in ASS1-deficient iPSC-derived hepatocytes

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- 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

Note: OTC – 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. CPS-1 – 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.
CPS-1 data generated in collaboration with Dr. Nicola Brunetti-Pierri and Leandro R. Soria.

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: 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 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

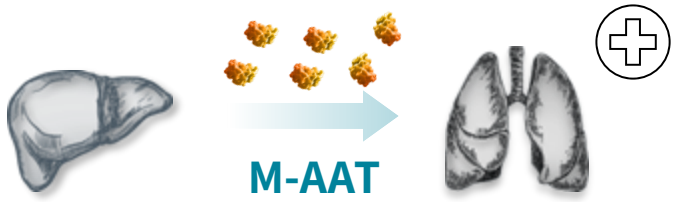
Note: 1. As demonstrated using a surrogate mouse-optimized oligo

KRRO-111: Alpha-1 Antitrypsin Deficiency

Repair Pathogenic Variant

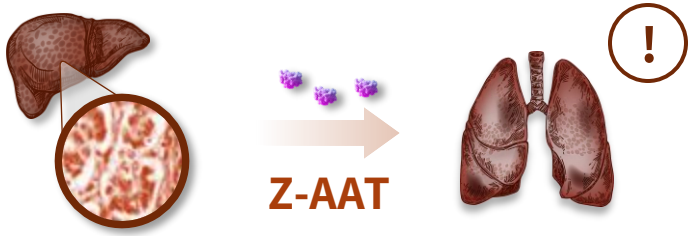
AATD: An Autosomal Recessive Disorder Leading to Liver and Lung Pathology

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:

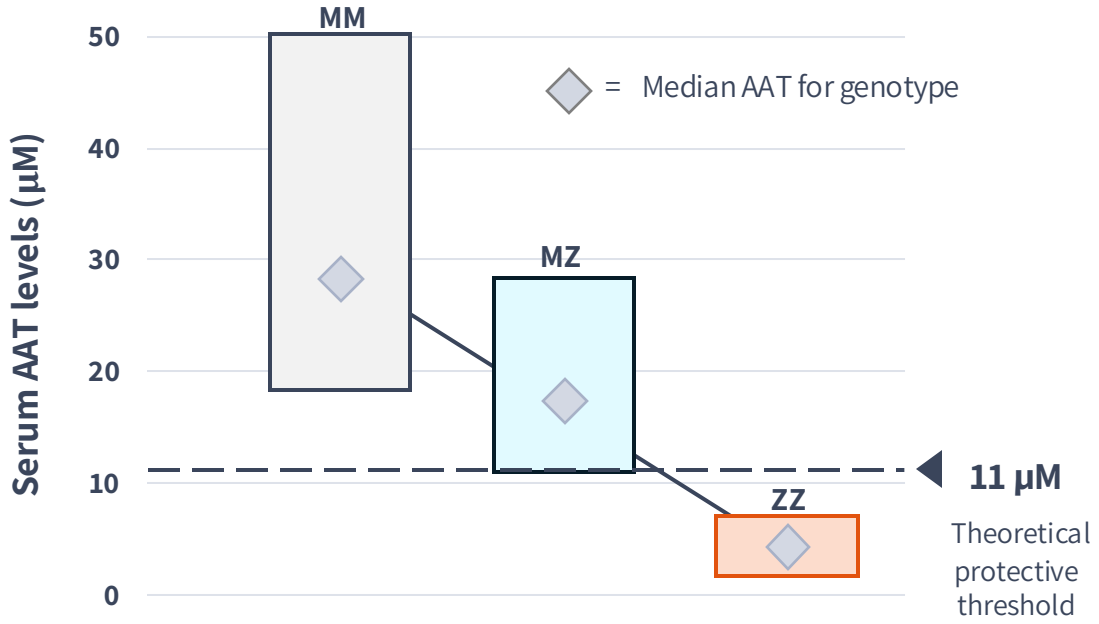
- **Liver damage from Z-AAT aggregates**
- **Low circulating AAT, causing lung damage**

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

Source: Strnad et al., NEJM (2020); de Serres et al., Chest (2002)

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 µM** [1] to slow progression of lung disease and does not address liver damage

Improved outcomes may be attainable by targeting higher total AAT levels

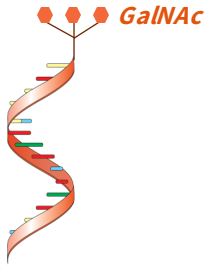
1. 11 µM T-AAT represents the lower bound of the 95% CI of T-AAT in MZ patients

Note: COPD = Chronic obstructive pulmonary disease

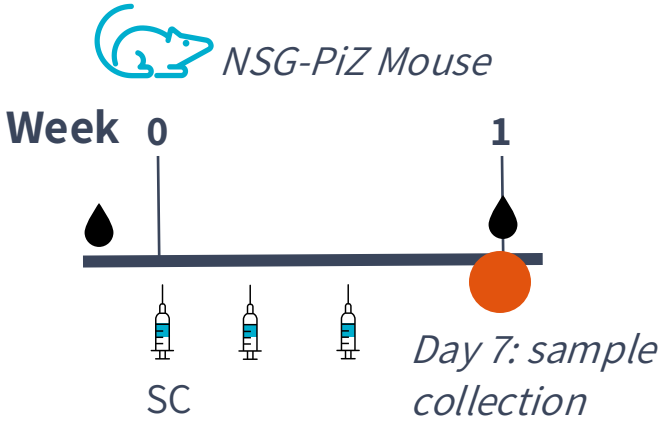
Source: Nakanishi T. et al. Eur Respir J. (2020); The undiagnosed disease burden associated with alpha-1 antitrypsin deficiency genotypes

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

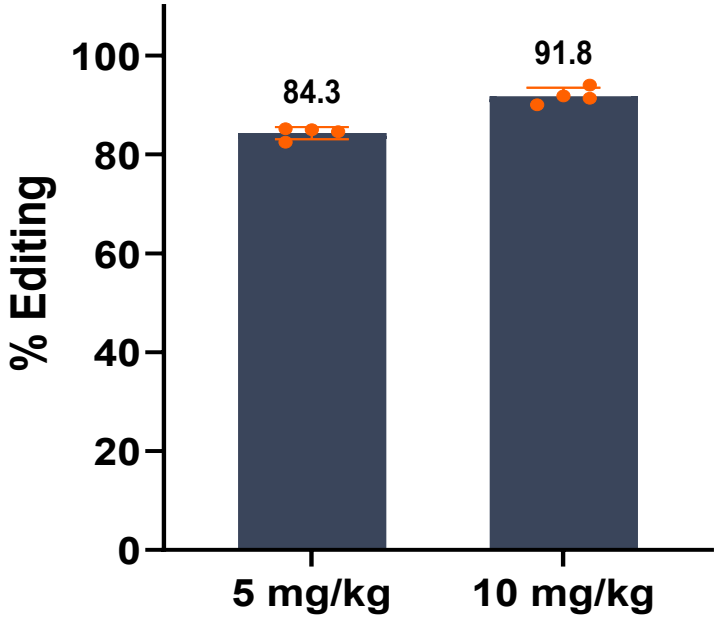
KRRO-111



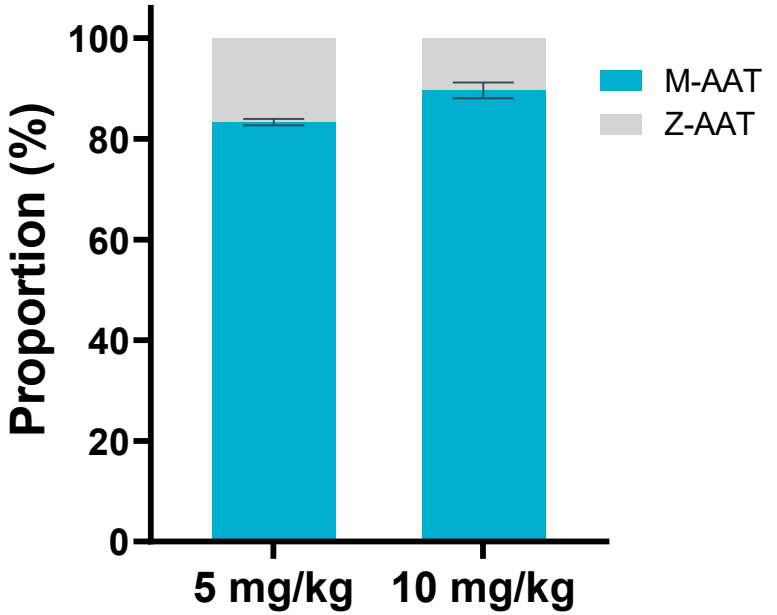
Dosing: Q2Dx3 at 5 or 10 mg/kg



SERPINA1 RNA Editing



% M-AAT Protein



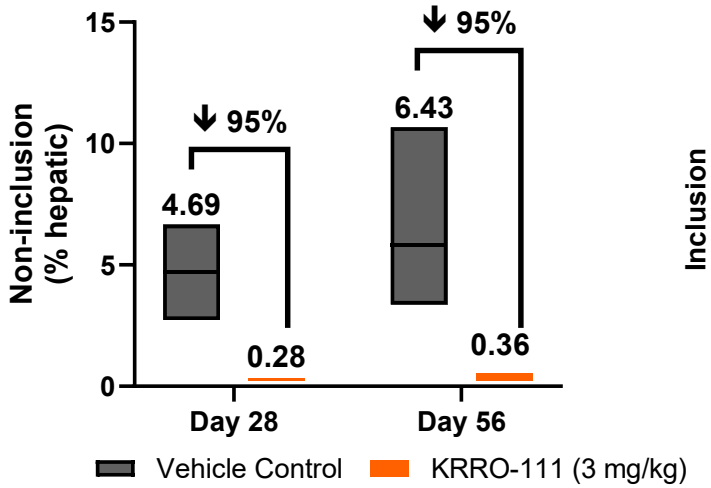
Note: Subcutaneous dosing Q2Dx3 at 5 or 10 mg/kg for each dose; editing efficiency and protein percentage are measured on day 7 by NGS and LC/MS, respectively. Proportion of M-AAT was calculated from the sum of M-AAT and Z-AAT detected in serum.

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

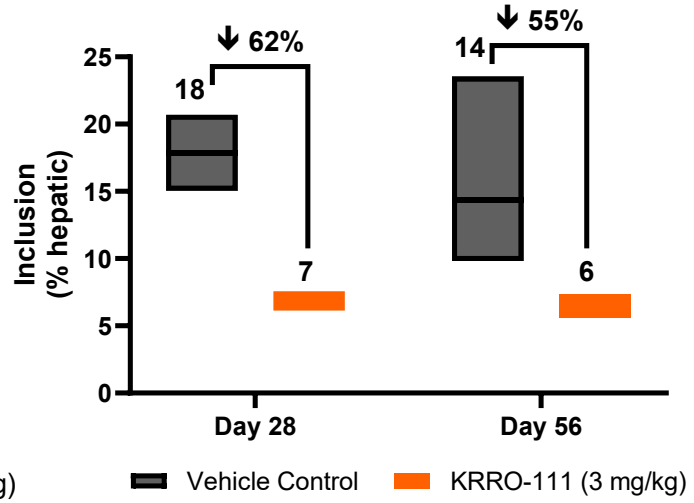
Active Z protein · editing efficacy



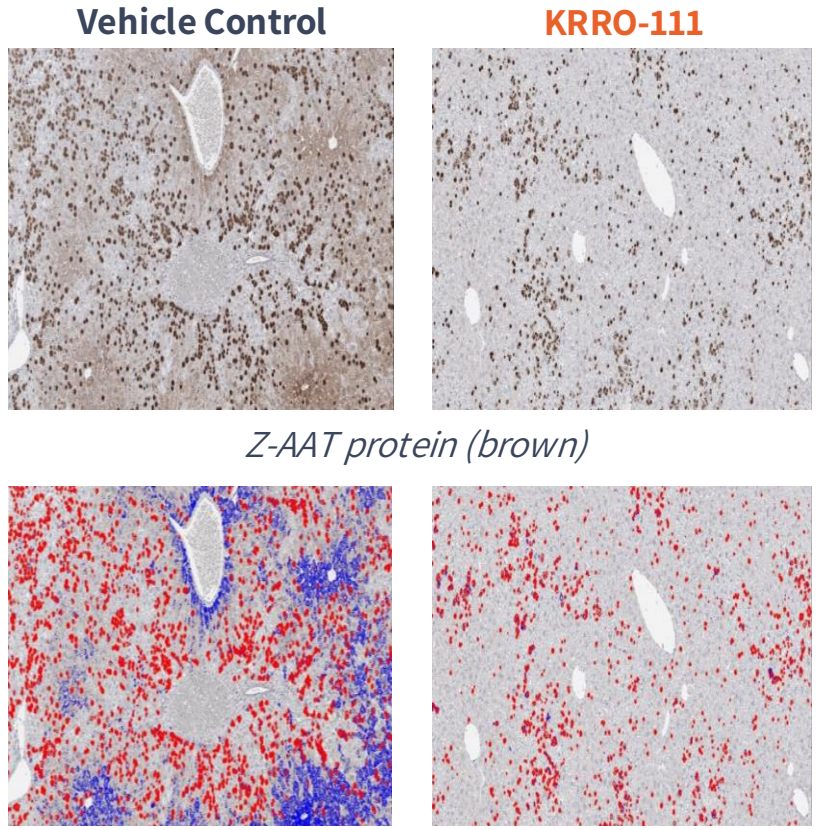
Robust Reduction of Pre-Existing Z-AAT Aggregates

INCLUSION-ASSOCIATED Z-AAT

Pre-existing aggregates · autophagic clearance



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



Z-AAT protein (brown)

Non-inclusion Z-AAT (blue)
Inclusion-associated Z-AAT (red)

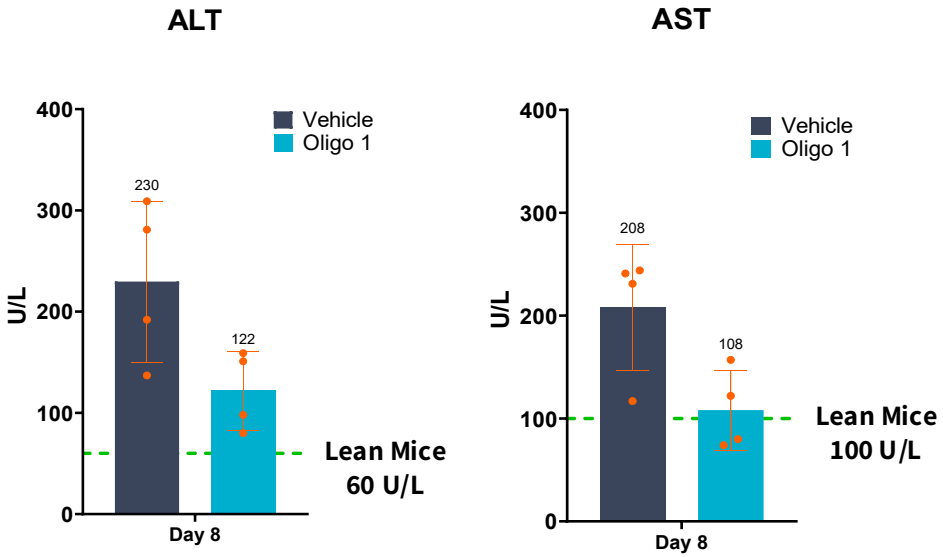
IHC: immunohistochemistry, 140X, Day 56 representative images, Z-AAT: homozygous Z-variant AAT
Study design: Q2Dx3, Q2Wx4: every two weeks in C57BL/6-PiZZ mice, dose = 3mg/kg

Early Pipeline

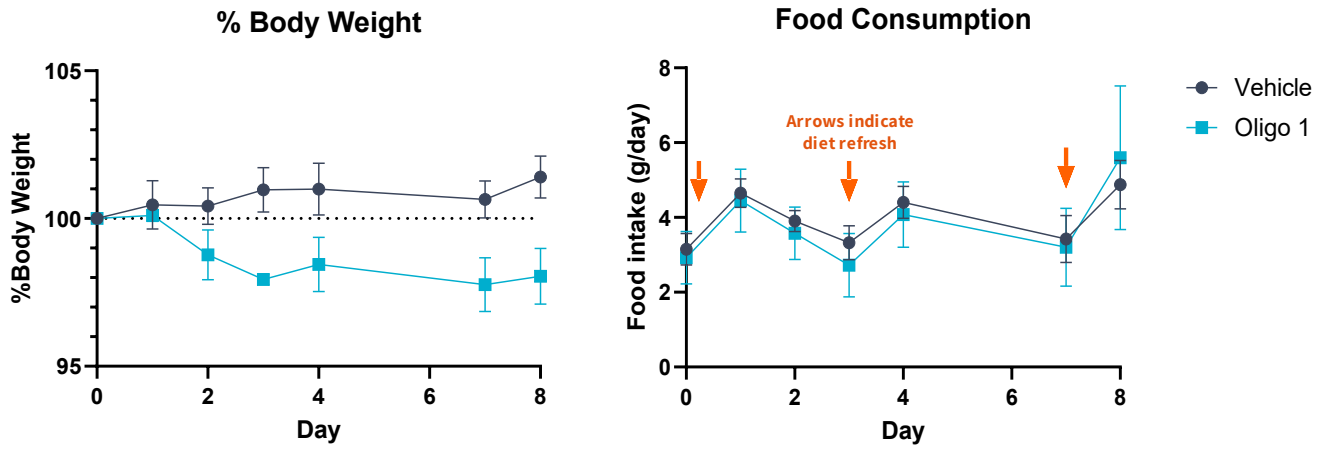
Hepatic AMPKγ1 Activation Improved Liver Function in Obese Mice

GalNAc

Normalizing Liver Function



Reduction in Body Weight Despite Similar Food Intake



AMPK

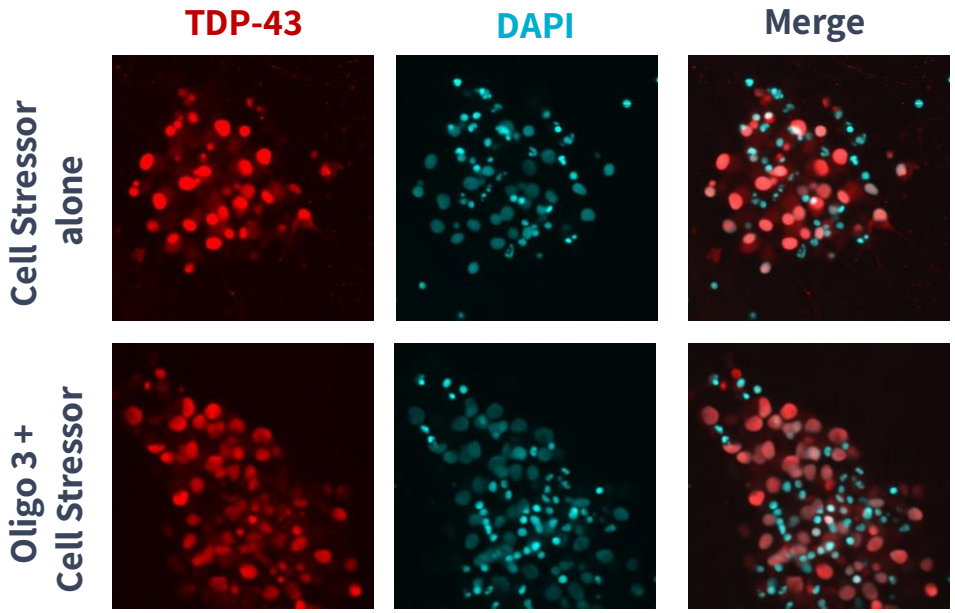
- Activation of the Master Metabolic Regulator
- Designed to Improve Liver Function
- Potentially Restores Metabolic Signaling Without Affecting Food Intake

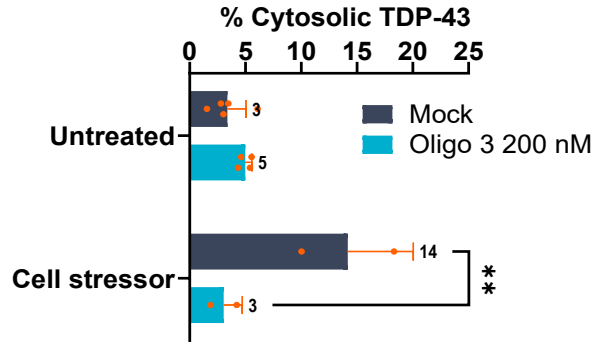
~20% editing was sufficient to normalize liver function and reduce body weight in mice

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

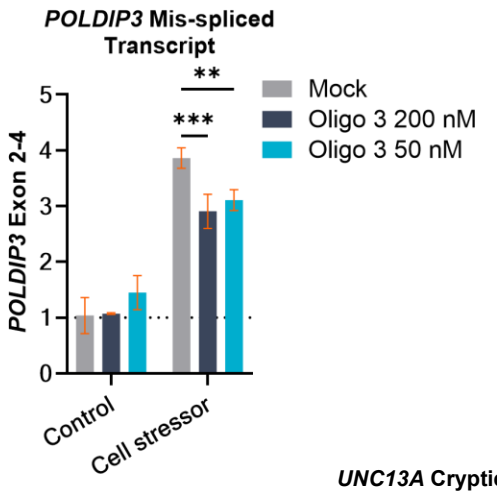
De Novo TDP-43 Demonstrated Reduced Mis-splicing and Decreased Cytosolic Mis-localization in iPSC Motor Neurons

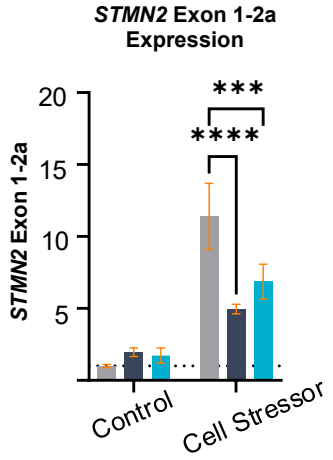
Decreased Cytosolic Mis-localization of TDP-43 protein

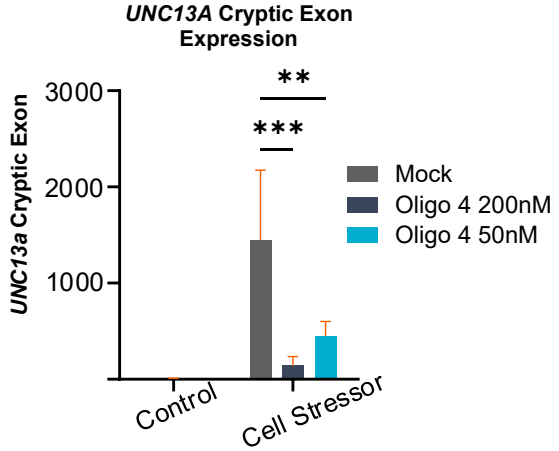




Reduced Mis-splicing: Maintaining POLDIP3, STMN2, and UNC13A







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

- KRRO-111 DC nominated for GalNAc-conjugated AATD program
- Regulatory filing 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 $157.1 million as of March 31, 2026