KORRO BIO



November 2026

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

NASDAQ: **KRRO**

Forward-Looking Statements 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-121 trial ongoing; UCD patient dosing expected 1H27; Interim readout 2H27

KRRO-111 with robust 8-week preclinical data; Regulatory submission expected 1H27

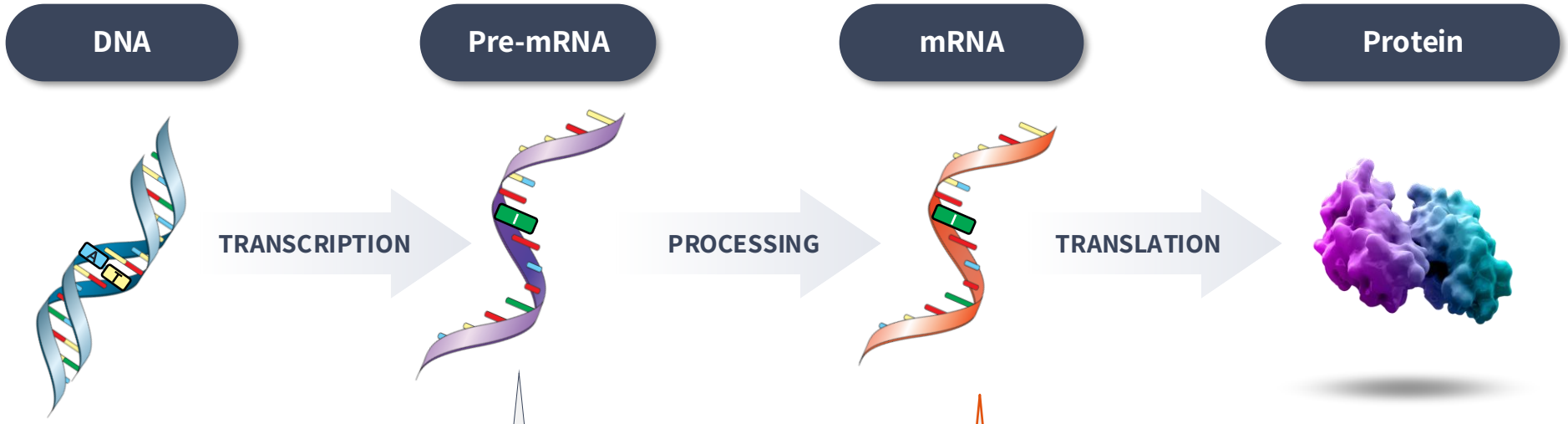
Cash runway into 2H28 enabling multiple milestones [1]

Potential for partnership across our pipeline

UCD = Urea cycle disorders; DC = Development candidate

1. Cash, cash equivalents and marketable securities of $124.8 million as of September 30, 2026

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



Edit a single A-to-I

Modifying gene expression

Edit a single A-to-I

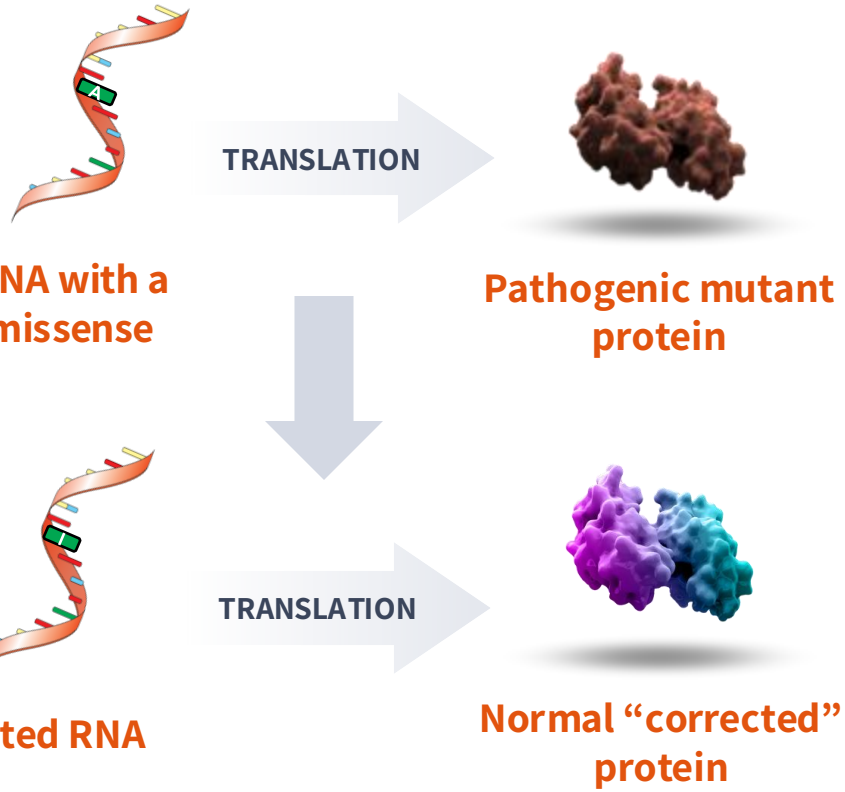
- **Repair** a G->A mutation to correct the protein
- **Generate a *de novo* protein** with preferred properties (can alter 12 amino acids)

Current Focus

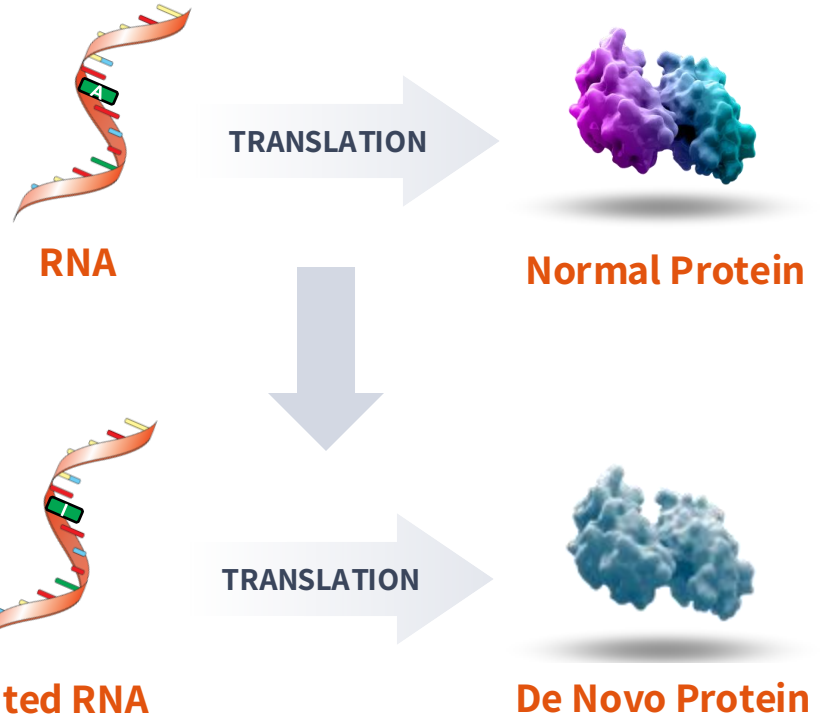
Human genetics guiding the possibilities

Pipeline Programs Primarily Focused on Generation of De Novo Proteins

Repair the Protein



Modulate Protein Function (Activate pathway)



Examples of Repair = E342K AATD, G2019S Parkinson's 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 Phase 1/2 - Interim data in 2H27			ANCHOR	
Repair Pathogenic Variant	KRRO-111 AATD	GalNAc (SC)	AAT Reg. Submission 1H27				
Allosteric Activator	Longevity (Liver)	GalNAc (SC)	AMPKγ1				
Overcome LoF and GoF [1]	Amyotrophic lateral sclerosis (ALS)	Intrathecal (IT)	TDP-43				

De Novo protein creation

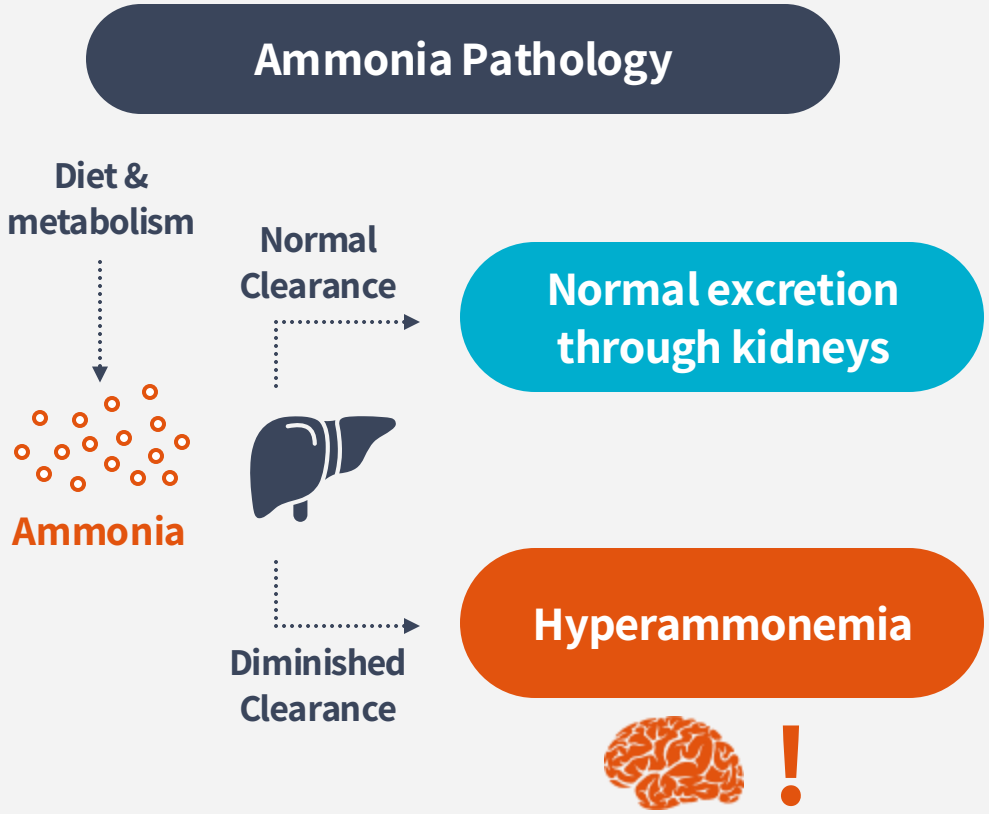
Protein repair

GS = Glutamine Synthetase; ANCHOR (**A**ssessme**N**t of **C**ontrol of **H**yperammonemia through **O**ligonucleotide-based **R**NA editing) NCT07773246
AAT = Alpha-1 antitrypsin; AATD = AAT deficiency; AMPK γ1 = Regulatory subunit of AMP-activated protein kinase; TDP43 = TAR DNA-binding protein 43; SC = subcutaneous
[1] *De Novo* protein prevents toxic gain-of-function (GoF) with TDP43 aggregation, and continue downstream signaling by overcoming toxic loss-of-function (LoF)

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

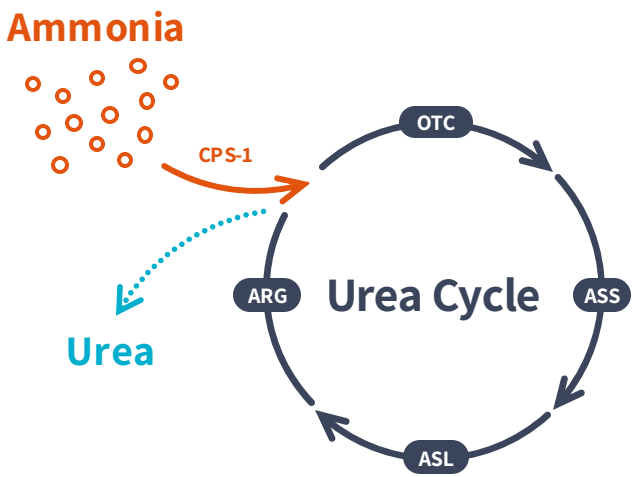
KRRO-121 Has Blockbuster Potential in Multiple Indications

	Urea Cycle Disorders (UCD)	Hepatic Encephalopathy (HE)
Addressable Patients	4,200 US[1] 5,100 EU + UK[1]	80,000 US[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.

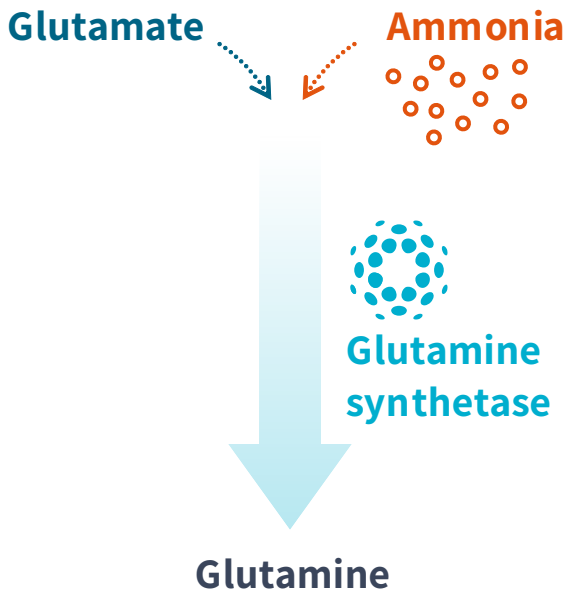
Two Complementary Pathways for Ammonia Clearance: Urea Cycle and Glutamine Synthetase (GS)

Urea Cycle



Expressed primarily in liver

Glutamine Synthetase

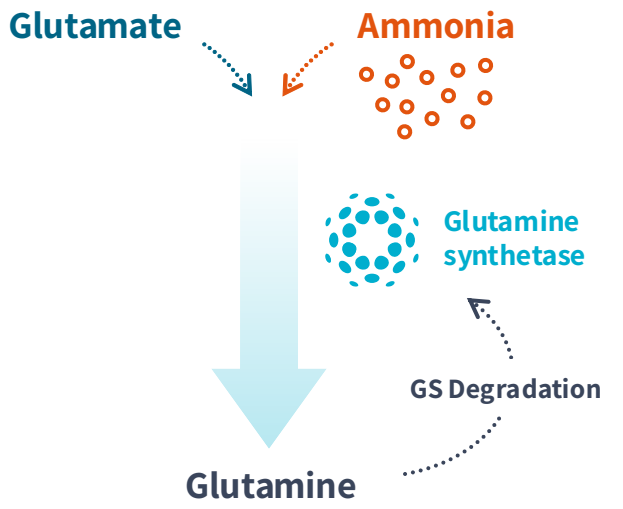


Expressed in many tissues, including liver, brain, and muscle

Source: Inoue et 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

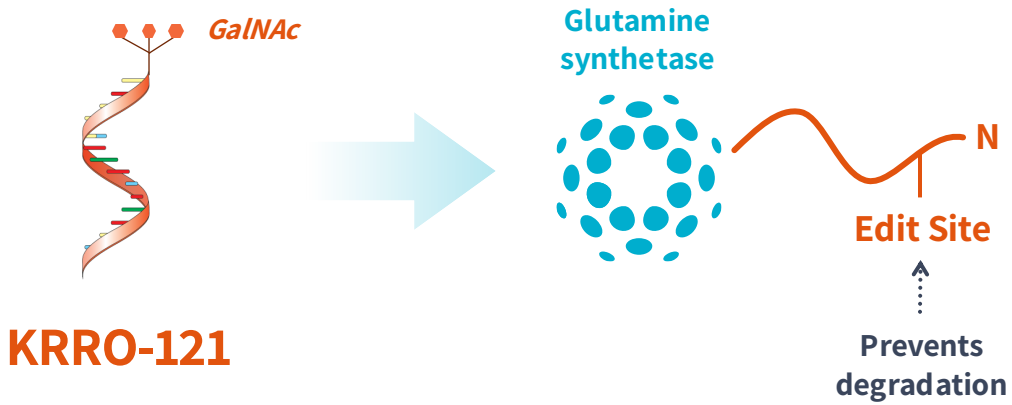
KRRO-121 is a GalNAc-conjugated Oligonucleotide Designed for Liver-Specific RNA Editing of GS to Enhance Ammonia Clearance Capacity

Glutamine Drives Degradation of GS



GS degraded when glutamine rises, reducing ammonia clearance capacity

Our Approach

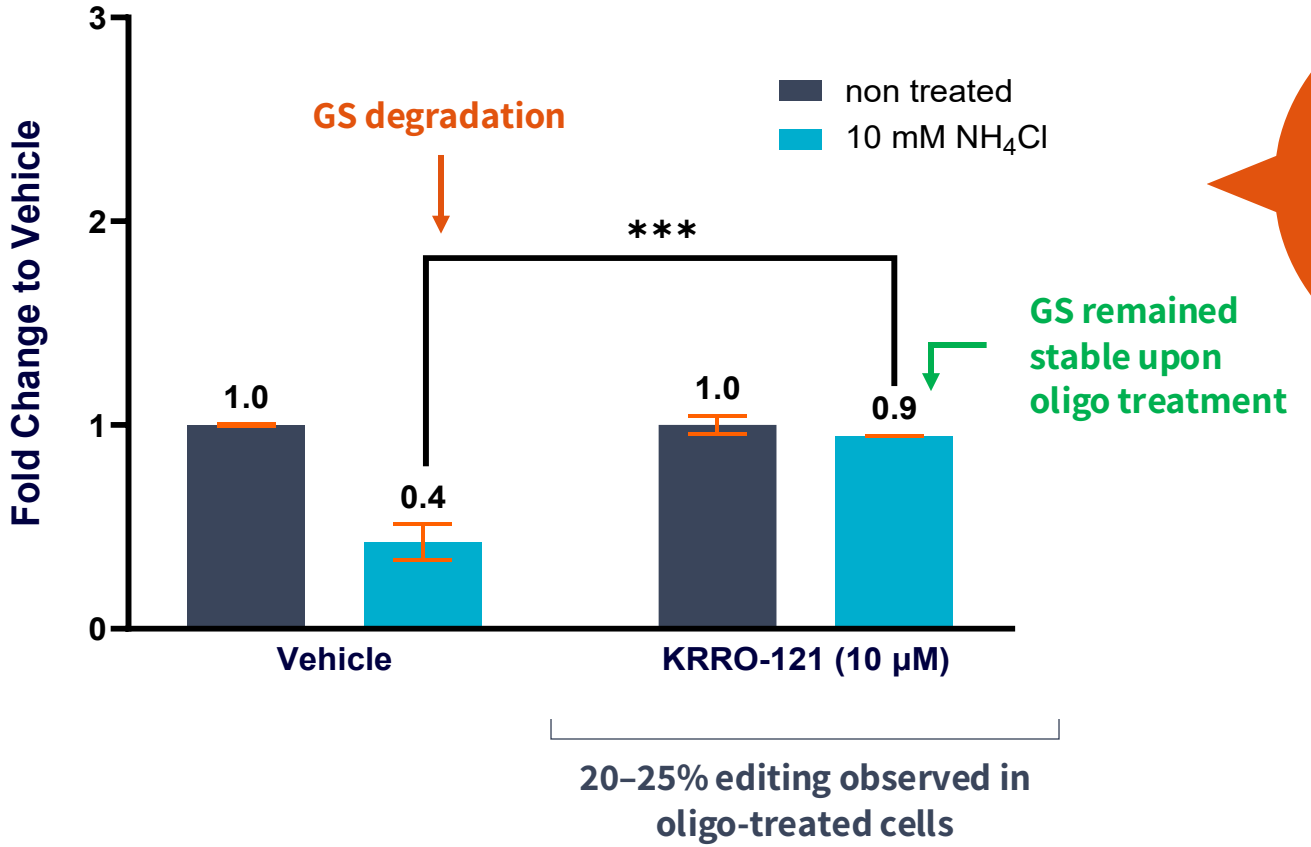


Liver-specific GalNAc-conjugated Oligo Generates Stabilized, De Novo GS

Source: Van Nguyen et al., Mol Cell (2016)

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

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

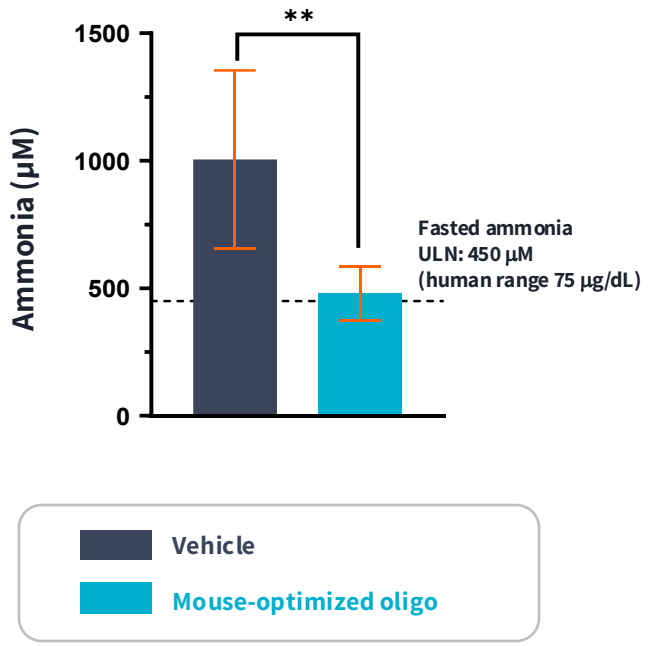


Similar results in ASS1-deficient iPSC-derived hepatocytes

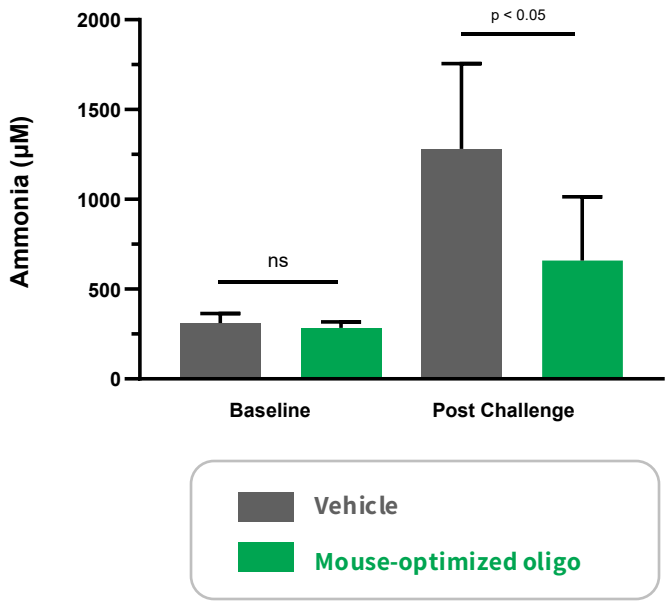
Note: OTC D175V human iPSC-derived hepatocytes differentiated for 14 days, then treated with oligo for 48 hours where indicated (10 mM NH_4Cl added after 24 hours where indicated). GS concentration measured at conclusion of 48-hour incubation.

Ammonia Lowering and Survival Benefit Observed Across Multiple UCD Mouse Models Support Pan-UCD Applicability

Blunted Ammonia Excursion Post-Challenge in OTC



Blunted Ammonia Excursion Post-Challenge in CPS-1

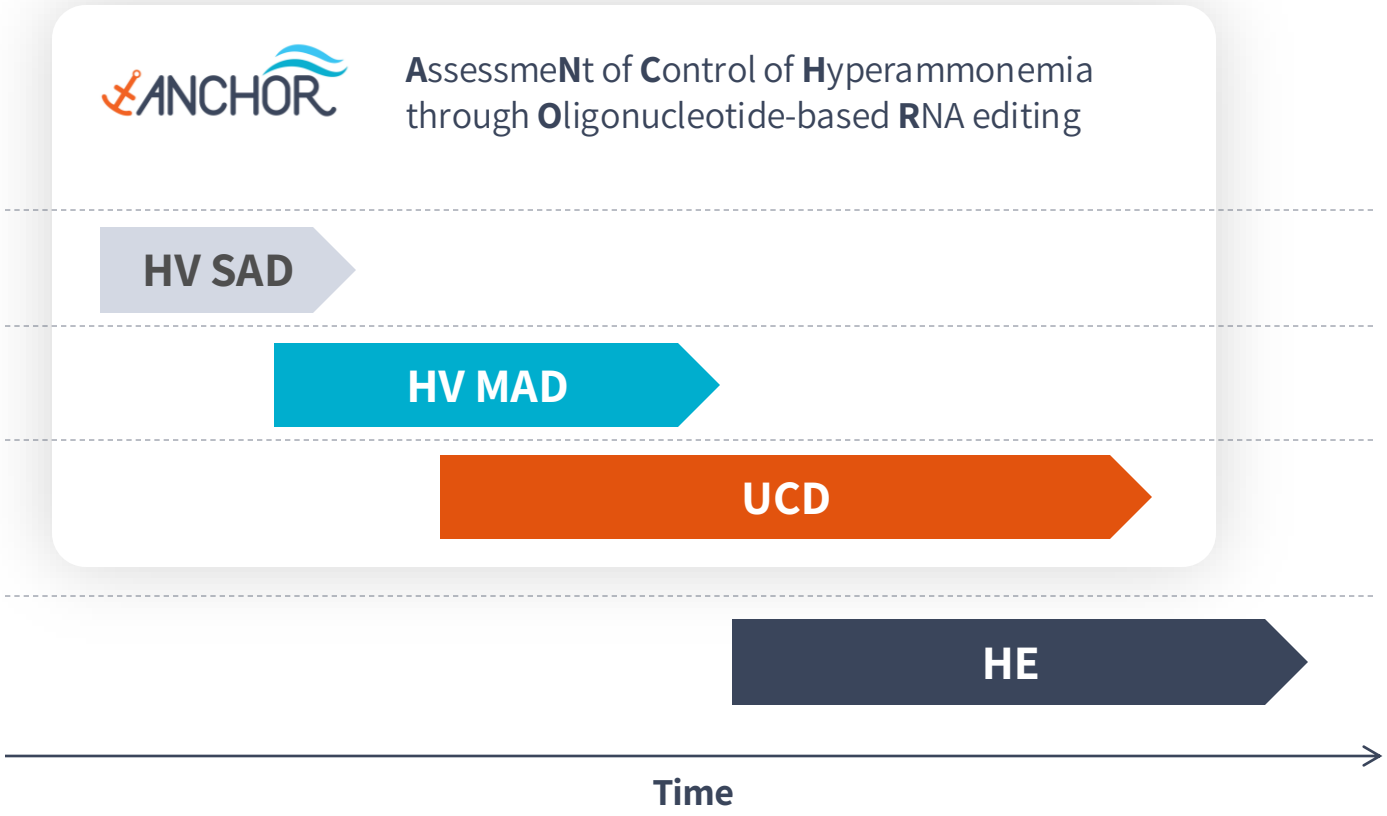


Improved Survival in ASL



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). 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. ASL – Treatment of the ASL$^{Neo/Neo}$ mice with a mouse-optimized oligo improved median survival rate of 28%, from 18 days to 23 days (p=0.0363) compared to vehicle. CPS-1 and ASL data generated in collaboration with Dr. Nicola Brunetti-Pierri and Leandro R. Soria.

Early Studies to Establish KRRO-121 Lowers Ammonia, Enabling Readiness for UCD and HE Pivotal Studies



Healthy Volunteers

Provide safety data for HE, start UCD at higher dose without delaying UCD timeline

UCD

Evaluate ammonia control, KRRO-121 mechanism in multi-dose study

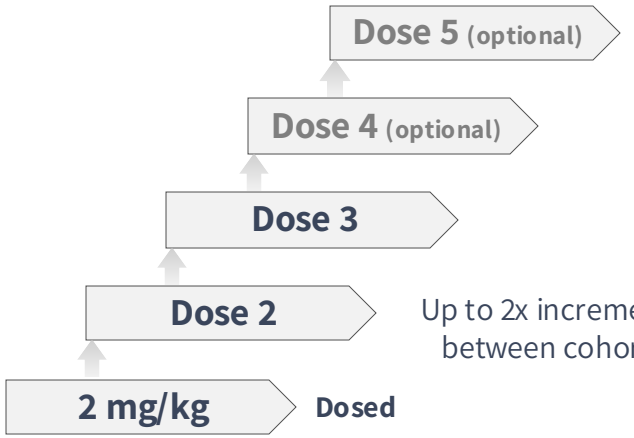
HE

Evaluate ammonia control in multi-dose study

HV Study Designed to Assess KRRO-121 Tolerability to Inform Patient Dosing

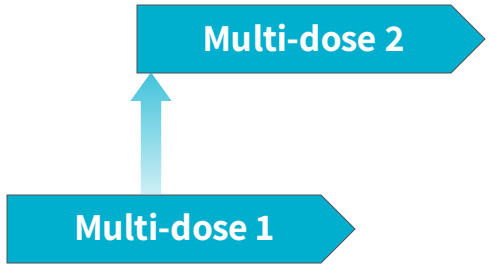
HV SAD

Up to 30 HVs, randomized 4:2



HV MAD

Up to 20 HVs, randomized 8:2

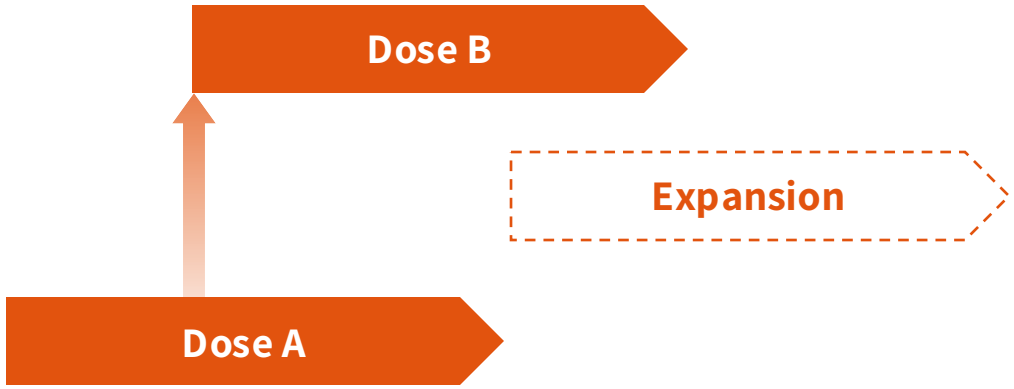


Key Planned Endpoints: Safety, tolerability, and PK profile

Note: PK – pharmacokinetics; SAD – single ascending dose; MAD – multiple ascending dose

Pan-UCD Study Designed to Assess Clinical Activity of KRRO-121

Open-label; up to five doses per patient in up to 15 patients, on stable background SOC



Potential Endpoints

- Safety and tolerability
- PK profile
- Ammonia AUC
- Plasma glutamine
- Protein intake

KRRO-121 has the Potential to Provide UCD Patients with Peace of Mind, Irrespective of Their Mutational Background

Note: Planned design and endpoints subject to regulatory approval; endpoints non-exhaustive. AUC = Area under the curve.

KRRO-121 Has Potential to Provide Peace of Mind for UCD and HE Communities

Synthetic Rescue to Blunt Ammonia Excursions with Infrequent SC Dosing

- **Pan-UCD approach**
- **Potential reduction in HACs / HE events**
- **Potential to delay transplant**
- **Potential diet liberalization (UCD)**
- **Improved quality of life**

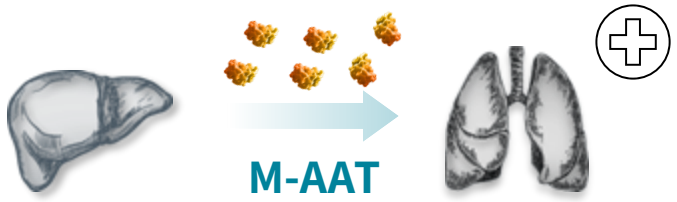
Note: GS – glutamine synthetase; HACs – hyperammonemic crises; HE – hepatic encephalopathy; SC – subcutaneous

KRRO-111: Alpha-1 Antitrypsin Deficiency

Repair Pathogenic Variant

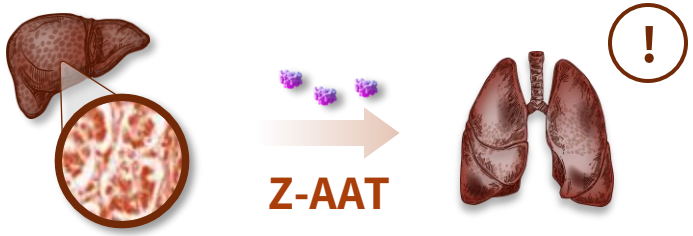
AATD: A Genetic Disorder Leading to Liver and Lung Pathology

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:

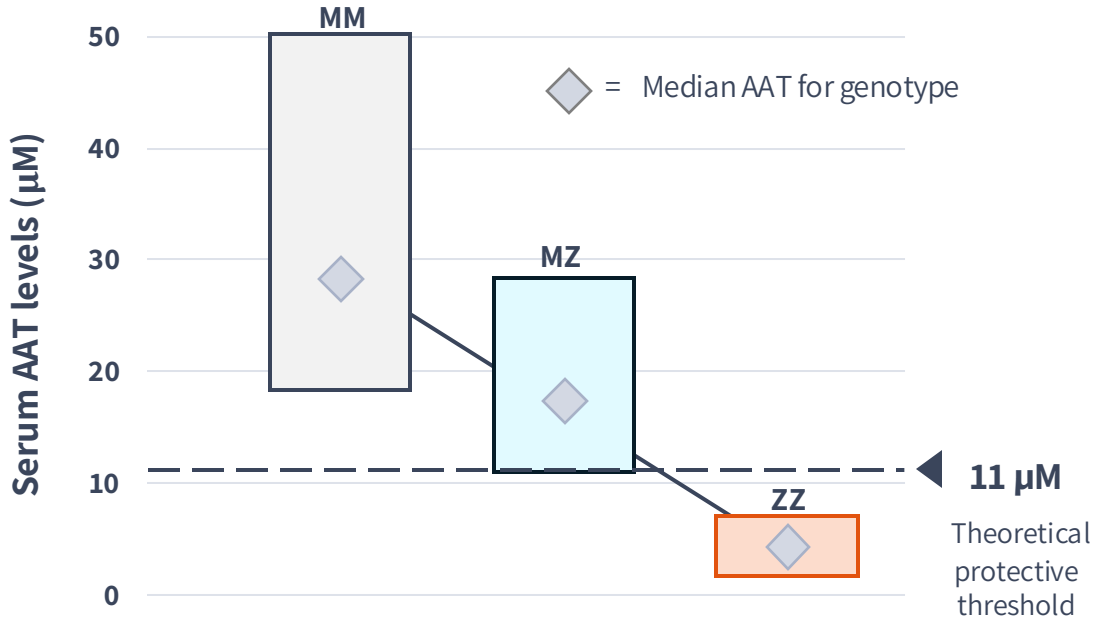
- **Liver damage from Z-AAT aggregates**
- **Low circulating AAT, causing lung damage**

>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

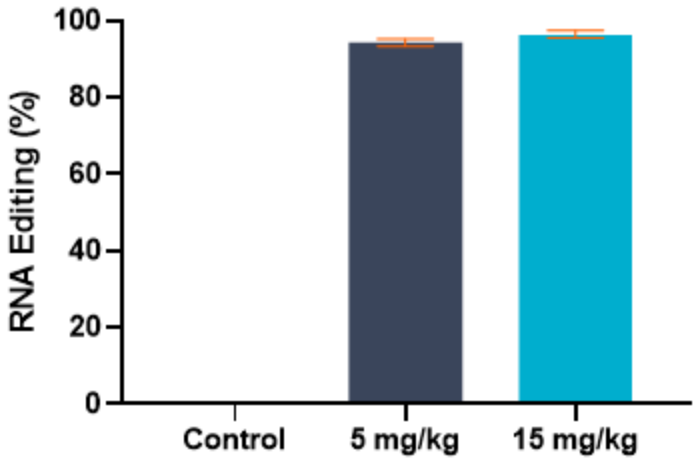
1. 11 µM T-AAT represents the lower bound of the 95% CI of T-AAT in MZ patients

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

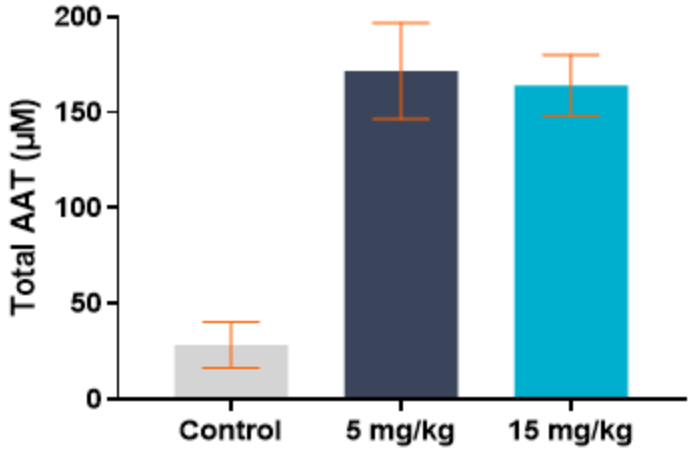
KRRO-111 Induced Robust Editing in NSG-PiZ Mice Over 8 Weeks

SERPINA1 RNA Editing (Day 57)



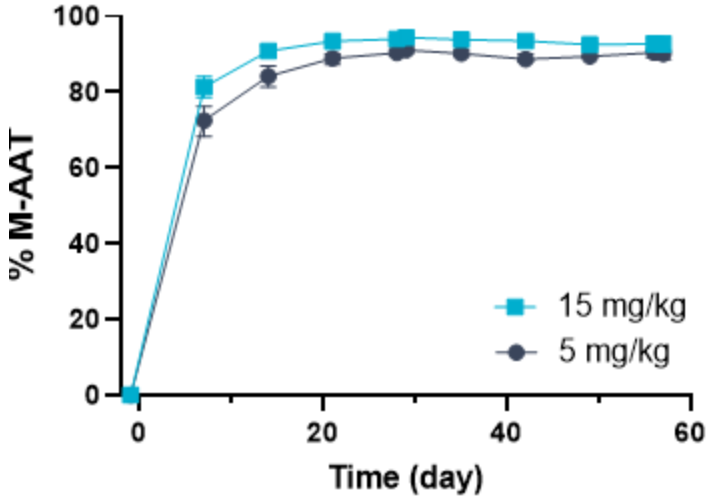
96% editing observed at highest dose

Total AAT Protein (Day 57)



~6x increase in T-AAT vs vehicle control

% M-AAT Protein

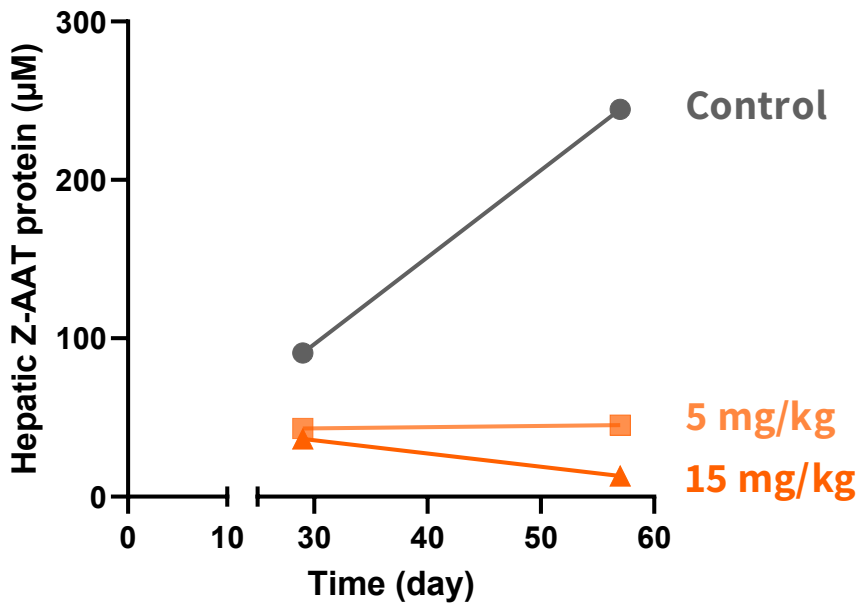


≥90% M-AAT observed at both doses

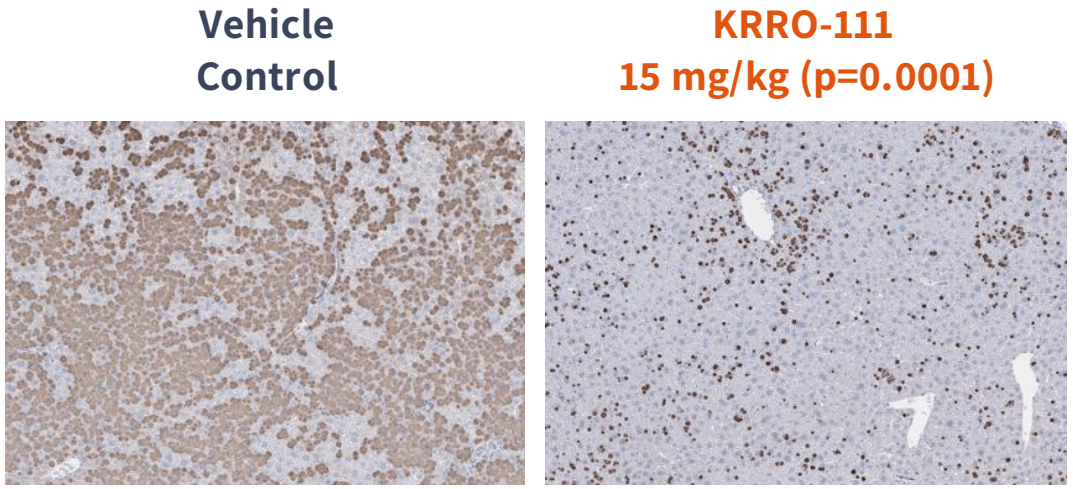
Note: NSG-PiZ mice were injected SC with weekly (QWx9) doses of KRRO-111. Editing was measured on day 57. Total AAT was measured by LC/MS. % M-AAT was calculated by dividing the concentration of M-AAT by the sum of concentrations of M-AAT and Z-AAT observed by LC/MS. Dosing began on day 0.

KRRO-111 Reversed Hepatic Z-AAT Accumulation in C57BL/6-PiZ Mice

Near-Complete Elimination of Whole Liver Z-AAT Protein



Liver histology: Near-Complete Clearance of AAT Aggregates at Day 57



Up to 95% decrease in whole liver Z-AAT on Day 57

Note: C57BL/6-PiZ mice were dosed weekly (QWx9) SC with KRRO-111. Left side: Whole liver Z-AAT protein concentration measured by LC-MS/MS . Right side: Immunohistochemistry (IHC) at 140X, Day 57 representative images shown of sections stained with antibody specific for polymeric AAT.

KRRO-111: Potential Best-in-Class RNA Editor Capable of Providing Differentiated Clinical Benefit

Designed to Repair Pathogenic Z-AAT Variant in AATD



Enable production of native M-AAT, protecting the lungs



Reduce harmful Z-AAT, protecting the liver



Restore acute-phase AAT production



Achieve favorable safety / tolerability profile



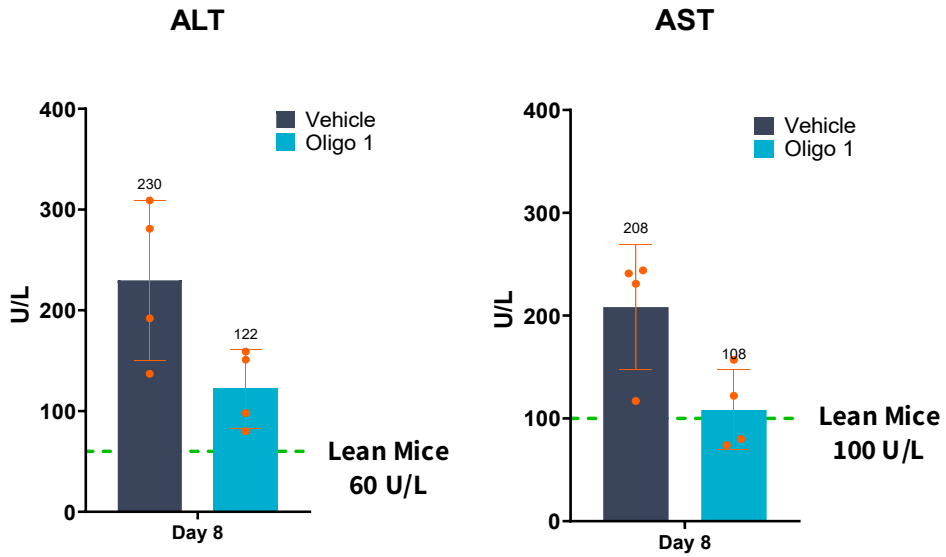
Avoid permanent genetic modifications or bystander edits

Early Pipeline

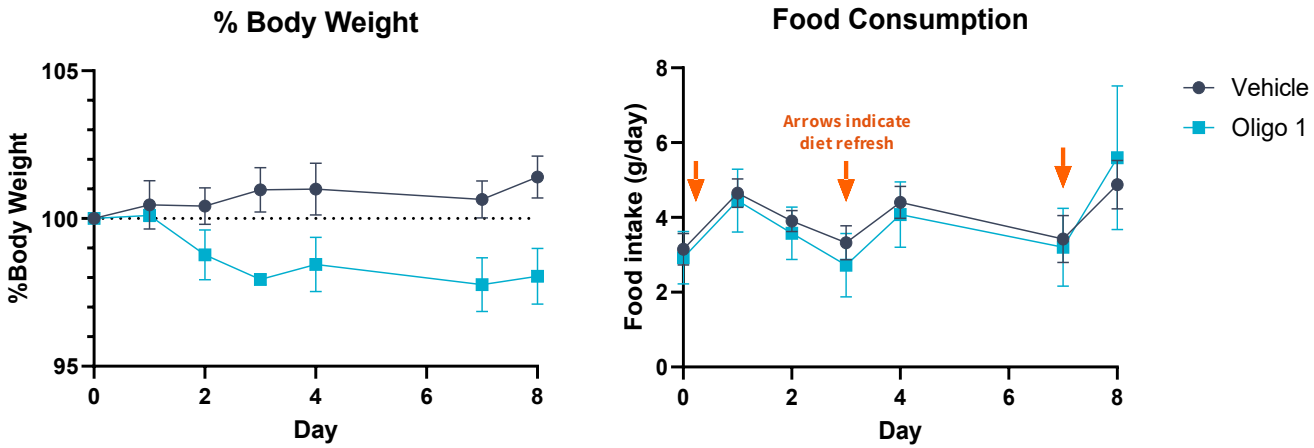
Hepatic AMPKγ1 Activation Improved Liver Function in Obese Mice

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.

Creation of a De Novo TDP-43 Protein Provides the Potential to Treat ALS

URGENT, VALIDATED NEED

- Fatal — 2–5 yr survival for ~59,000 patients (US + EU4/UK)
- 97% ALS have TDP-43 pathology

DIFFERENTIATED MECHANISM

- De novo TDP-43 protein via RNA editing — no DNA change
- Renews splicing and localization, cuts aggregates

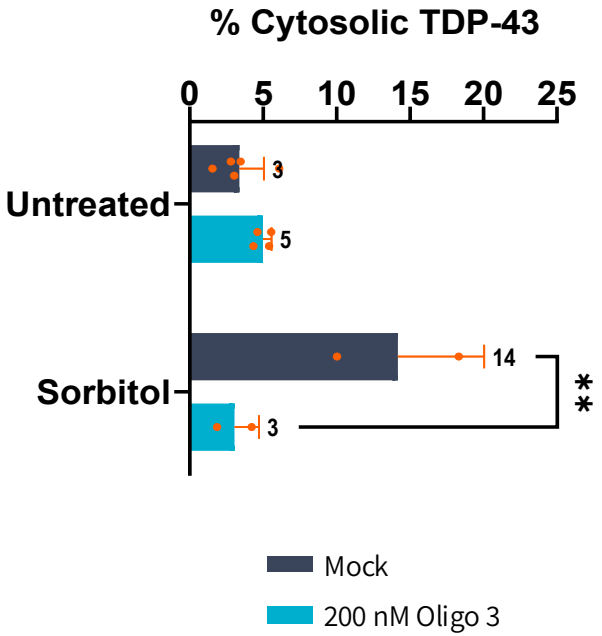
DE-RISKED BY DATA

- Dose-dependent editing in iPSC motor neurons leading to less aggregation and more nuclear retention (2 models)
- Encouraging editing in NHPs with non-optimized oligos

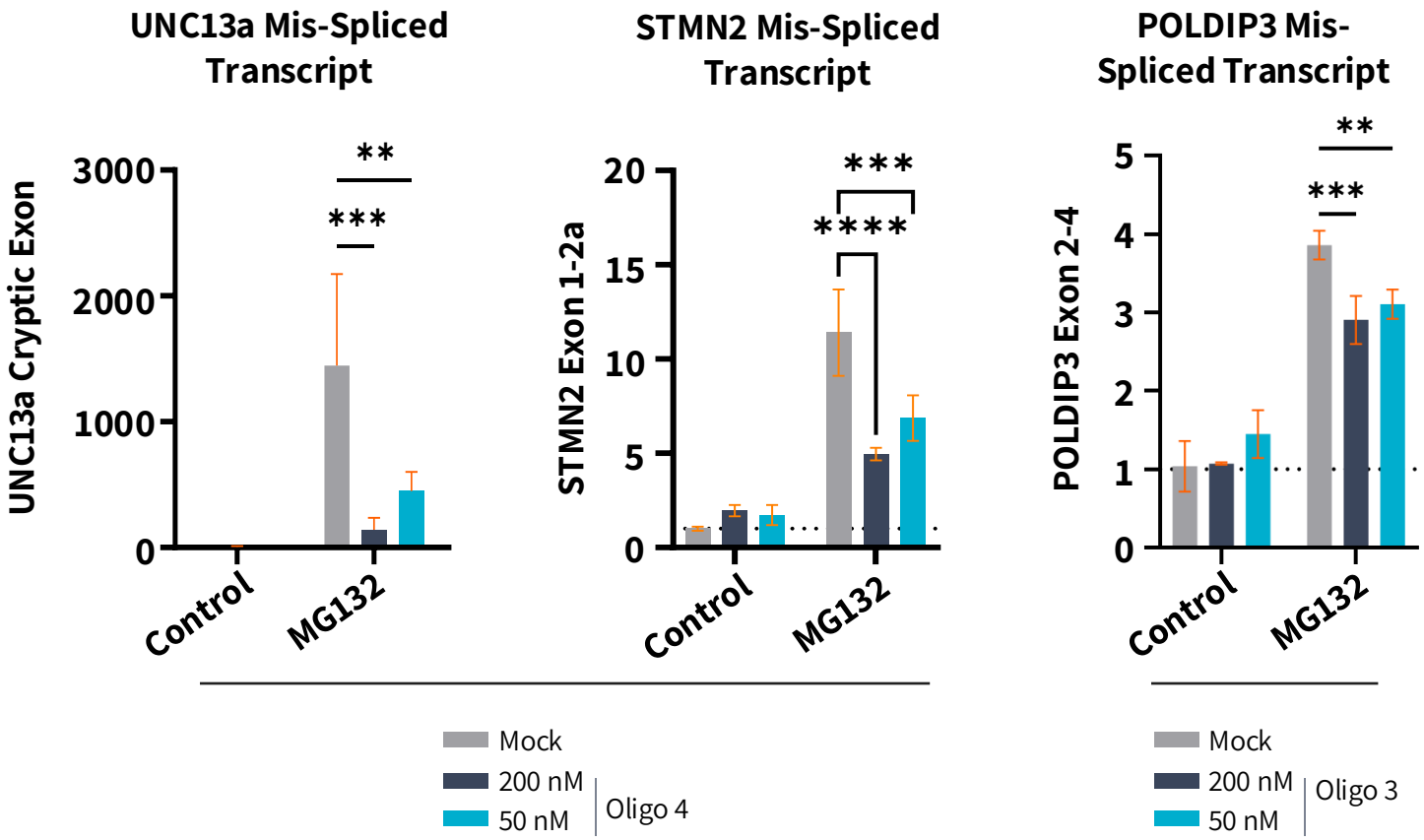
A potential first-in-class RNA-editing medicine with the potential to correct the root cause of TDP-43 pathology present in ~97% of ALS by addressing three key drivers of disease at once.

Edited TDP-43: Reduced Cytosolic Mislocalization & Mis-Splicing in iPSC Neurons

Decreased Cytosolic Mis-Localization of TDP-43 Protein



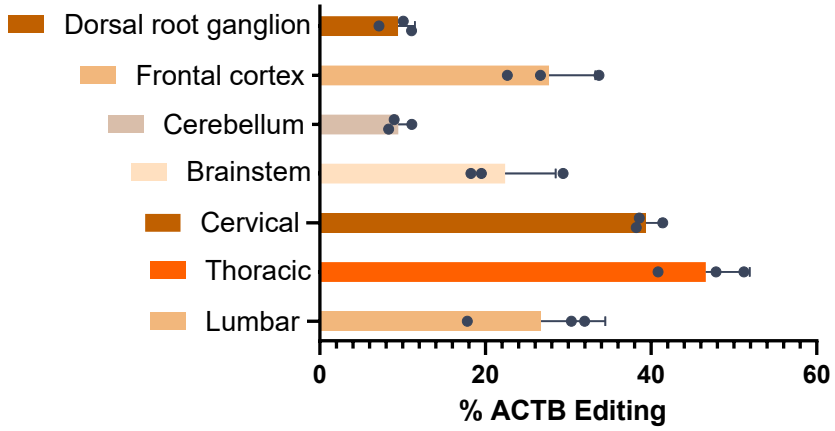
Reduced Mis-Splicing: Maintaining UNC13A, STMN2, and POLDIP3

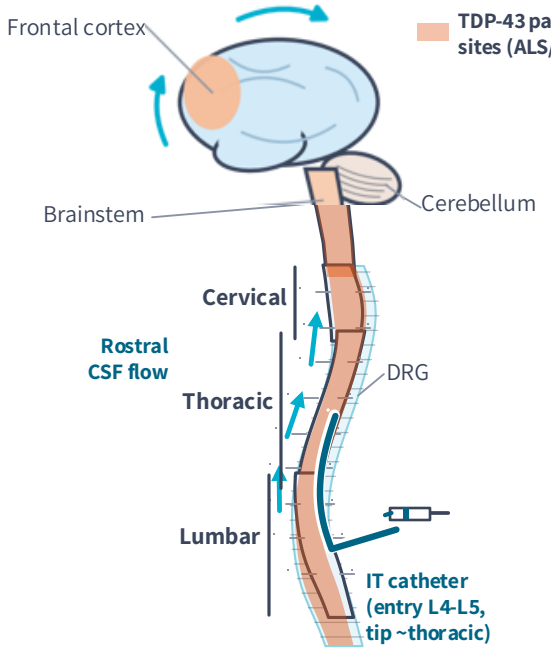


TDP-43 signal quantified by confocal microscopy using thresholds defined from untreated controls, which were applied uniformly to all images to measure TDP-43 intensity while excluding background signal

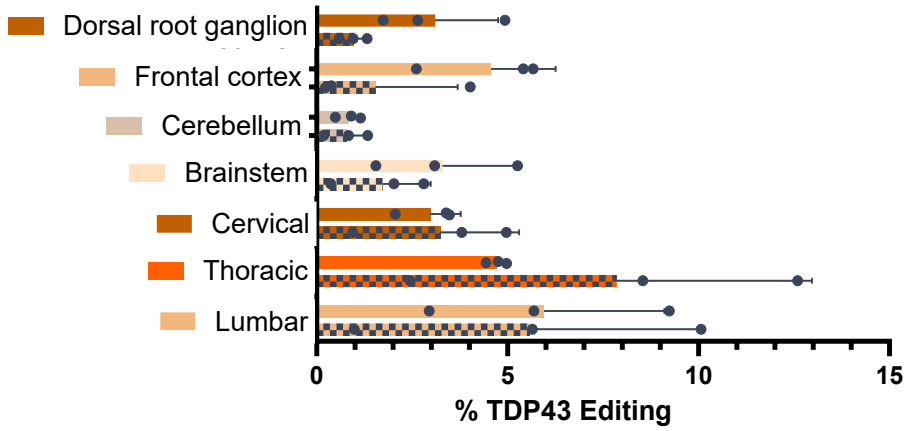
Editing Demonstrated in CNS with multiple oligos (ACTB and TARDP/TDP-43)

ACTB targeting REO





TARDP (TDP-43) targeting REO



- Generation 1 oligos in the CNS demonstrated safety and activity of the compounds via IT delivery
- Potential for ~20% editing in TDP43 sufficient to ameliorate altered splicing

NHP: Non-Human Primates, n=3/group; IT dosing via catheter (lumbar puncture, tip ~thoracic) on Days 0 and 14; necropsy Day 28. Delivered dose 12-14mg per dose. RNA editing by NGS; bars = mean, circles = individual animals



Positioned for Value Creation in 2026 and Beyond

KRRO-121 trial ongoing; UCD patient dosing expected 1H27; Interim readout 2H27

KRRO-111 with robust 8-week preclinical data; Regulatory submission expected 1H27

Cash runway into 2H28 enabling multiple milestones [1]

Potential for partnership across our pipeline

1. Cash, cash equivalents and marketable securities of $124.8 million as of September 30, 2026