Exhibit 99.1

KORRO BIO

Analyst Day 2026

KRRO-121: A Potential First-in-Class Treatment for Ammonia Control

January 27th, 2026

Forward-Looking Statements and Disclaimers

Forward-Looking Statements
Certain statements in this presentation may constitute "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. Forward-looking statements include, but are not limited to, express or implied statements regarding expectations, hopes, beliefs, intentions or strategies of Korro Bio, Inc. (Korro) regarding the future including, without limitation, express or implied statements regarding: the timing of the regulatory filing for KRRO-121; KRRO-121's pan-urea cycle disorder (UCD) potential; KRRO-121's first in class potential as a treatment for ammonia control; KRRO-121's ability to drive strong patient engagement and recruitment in clinical trials; KRRO-121's pipeline-in-a-product, blockbuster potential; KRRO-121's differentiation and potential impact for patients; among others. In addition, any statements that refer to projections, forecasts, or other characterizations of future events or circumstances, including any underlying assumptions, are forward-looking statements. The words "anticipate," "believe," "continue," "could," "estimate," "expect," "intend," "may," "might," "plan," "possible," "potential," "predict," "project," "should," "strive," "would," "aim," "target," "commit," and similar expressions may identify forward-looking statements, but the absence of these words does not mean that statement is not forward looking. Forward-looking statements are based on current expectations and assumptions that, while considered reasonable are inherently uncertain. New risks and uncertainties may emerge from time to time, and it is not possible to predict all risks and uncertainties. Factors that may cause actual results to differ materially from current expectations include, but are not limited to, various factors beyond management's control including risks inherent in biopharmaceutical development; risks associated with pre-clinical studies and clinical studies; risks associated with validating in clinical trials observations from pre-clinical studies; along with other risks inherent in biopharmaceutical development; and other risks associated with obtaining regulatory approvals and protecting intellectual property; as well as risks associated with general economic conditions (including recent geopolitical uncertainty and potential supply chain disruptions due to changes in economic policy); and other risks and uncertainties indicated from time to time in Korro's filings with the SEC, including "Risk Factors" in Korro's most recent Quarterly Report on Form 10-K or Form 10-Q filed with the SEC, as such may be amended or supplemented by its other filings with the SEC. Nothing in this presentation should be regarded as a representation by any person that the forward-looking statements set forth herein will be achieved or that any of the contemplated results of such forward-looking statements will be achieved. You should not place undue reliance on forward-looking statements in this presentation, which speak only as of the date they are made and are qualified in their entirety by reference to the cautionary statements herein. Except as required by law, Korro does not undertake or accept any duty to release publicly any updates or revisions to any forward-looking statements to reflect any change in their expectations or in the events, conditions or circumstances on which any such statement is based. This presentation does not purport to summarize all of the conditions, risks and other attributes of an investment in Korro.

Industry and Market Data
Certain information contained in this presentation relates to or is based on studies, publications, surveys and Korro's own internal estimates and research. In this presentation, Korro relies on, and refers to, publicly available information and statistics regarding market participants in the sector in which Korro competes and other industry data. Any comparison of Korro to any other entity assumes the reliability of the information available to Korro. Korro obtained this information and statistics from third-party sources, including reports by market research firms and company filings. In addition, all of the market data included in this presentation involve a number of assumptions and limitations, and there can be no guarantee as to the accuracy or reliability of such assumptions. Finally, while Korro believes its internal research is reliable, such research has not been verified by any independent source and Korro has not independently verified the information.

Trademarks
This presentation may contain trademarks, service marks, trade names and copyrights of Korro or other third parties, which are the property of their respective owners. Solely for convenience, some of the trademarks, service marks, trade names and copyrights referred to in this presentation may be listed without the TM, SM © or ® symbols, but Korro will assert, to the fullest extent under applicable law, the rights of the applicable owners, if any, to its trademarks, service marks, trade names and copyrights.

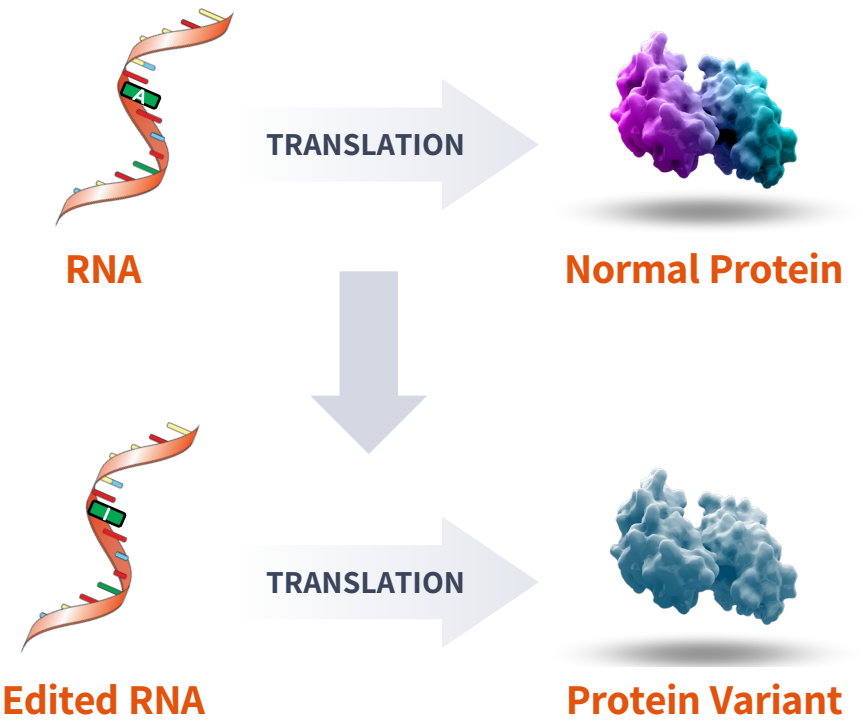
Expanding to New Biological Frontiers with RNA Editing

Ram Aiyar, PhD, MBA

Chief Executive Officer

Developing Transformative Genetic Medicines for Rare and Highly Prevalent Diseases

Modulate Protein Function (Activate pathway)



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

Editing RNA
Without permanently modifying DNA

Modular Delivery
Potential to deliver to multiple cell types

Learning from Genetics
To support predictable biological impact

KRRO-121 Scientific Overview and Preclinical Data

Loïc Vincent, PhD

Chief Scientific Officer

Mechanism: Stabilizing Glutamine Synthetase to Clear Ammonia

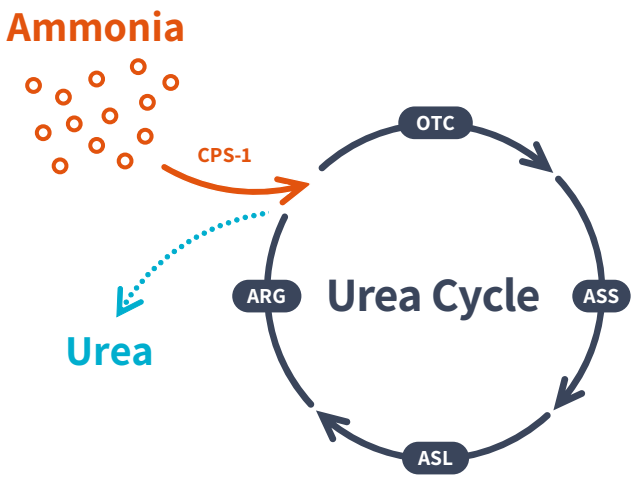
Glutamine Synthetase (GS) is a critical ammonia clearing mechanism

- Genetic evidence uncovers a key amino acid modification that can **augment GS protein stability**
- **Ammonia-lowering benefits** of stabilized GS activity may address substantial unmet need in patients with poor ammonia control, including UCD and hepatic encephalopathy
- KRRO-121 is a GalNAc-conjugated ASO that edits GS mRNA to generate a stable, *de novo* GS variant **specifically in the liver**
- KRRO-121 demonstrates potential to enable **robust ammonia clearance**, supporting a pan-UCD approach that may enable dietary liberalization as well as clinical activity in other **ammonia-driven diseases**, such as HE

KRRO-121 regulatory submission to enable commencement of FIH trial is anticipated in the 2nd half of 2026

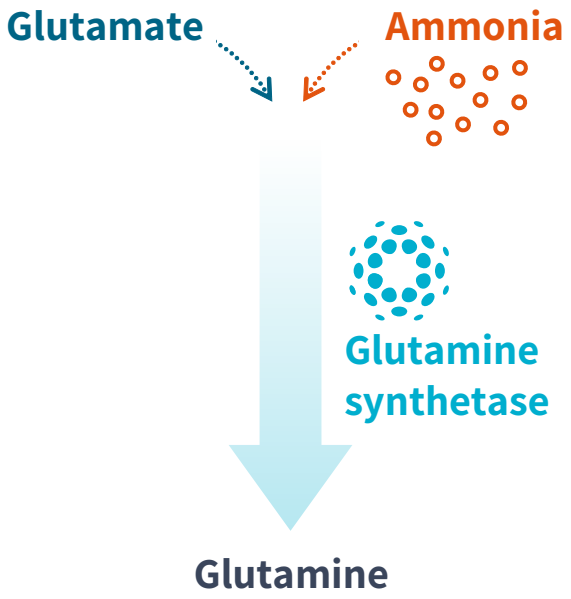
Two Complementary Pathways for Ammonia Clearance: Urea Cycle and Glutamine Synthetase (GS)

Urea Cycle



Expressed primarily in liver

Glutamine Synthetase

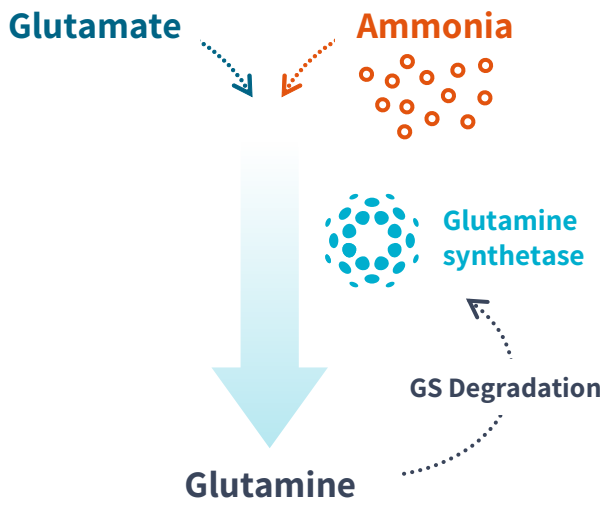


Expressed in many tissues, including liver, brain, and muscle

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

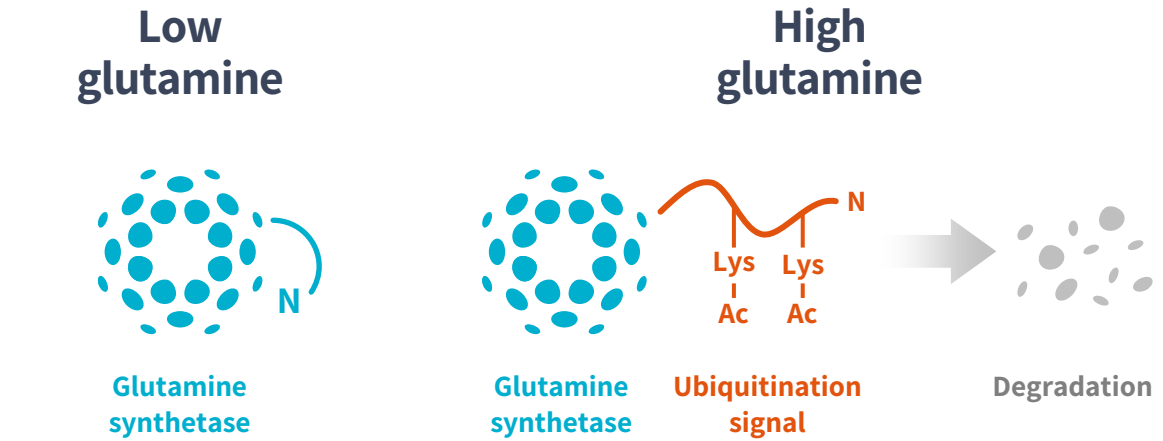
Degradation of GS Controlled by Levels of Glutamine

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

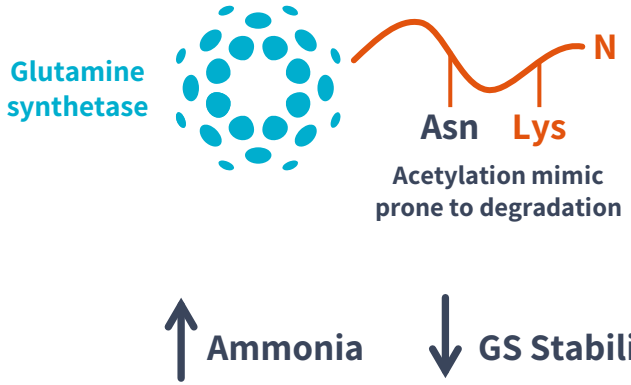
Source: Van Nguyen et al., Mol Cell (2017)

Human Genetic Evidence Supports Stabilization of GS by Preventing Degradation

Loss of Function

Case Report

Two Siblings With Valproate-Related Hyperammonemia and Novel Mutations in Glutamine Synthetase *(GLUL)* Treated With Carglumic Acid

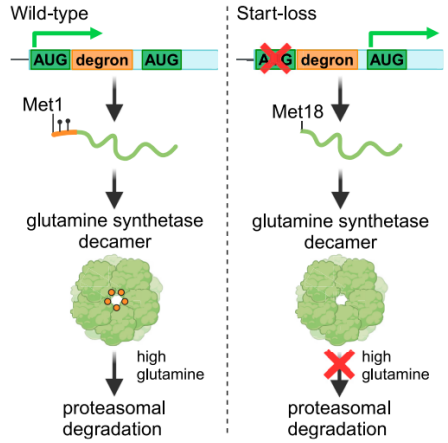


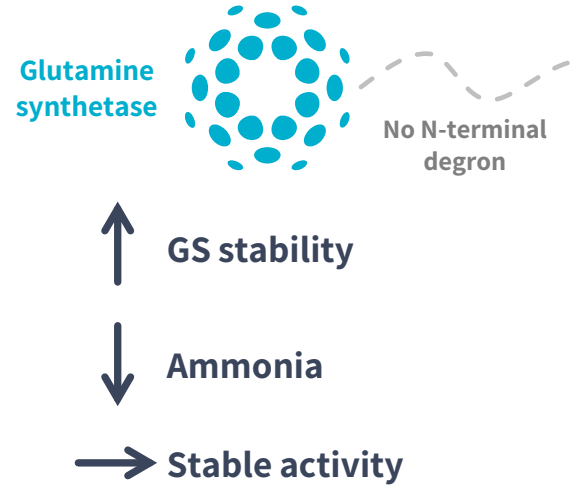
Patient with Lys14Asn mutation (mimicking acetyl-lysine) resulted in GS deficiency, hyperammonemia

Gain of Function

ARTICLE

Clustered *de novo* start-loss variants in *GLUL* result in a developmental and epileptic encephalopathy via stabilization of glutamine synthetase



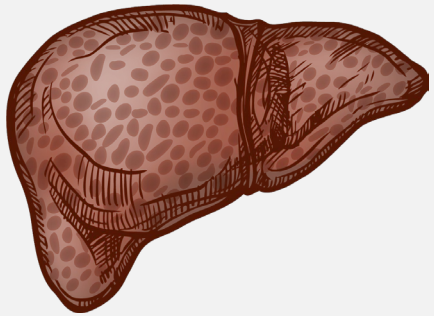


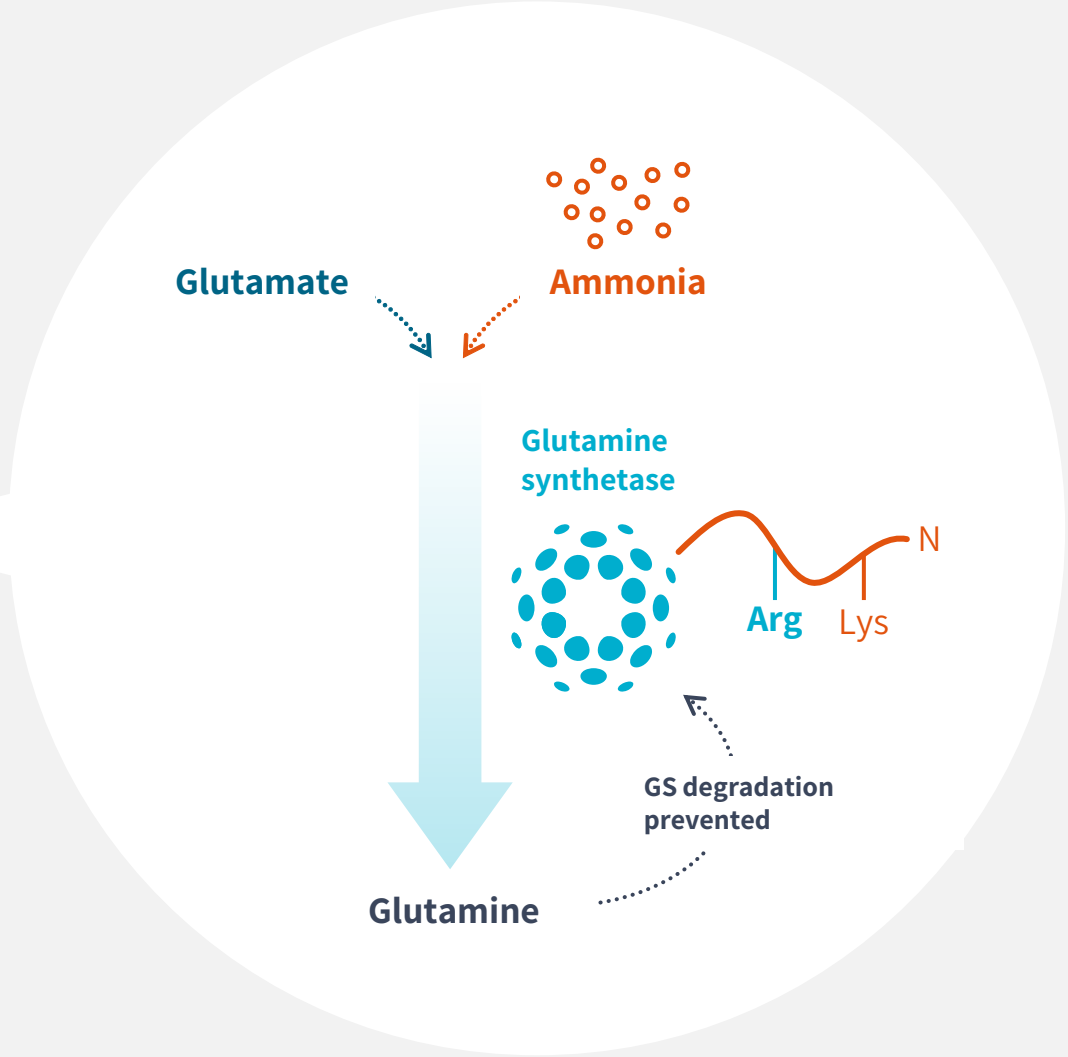
9 patients with start-loss variants, stabilizing GS due to loss of N-terminal Lys residues

Source: Bennet et al., Child Neurology Open (2020); Jones et al., The American Journal of Human Genetics (2024); Van Nguyen et al., Mol Cell (2017)

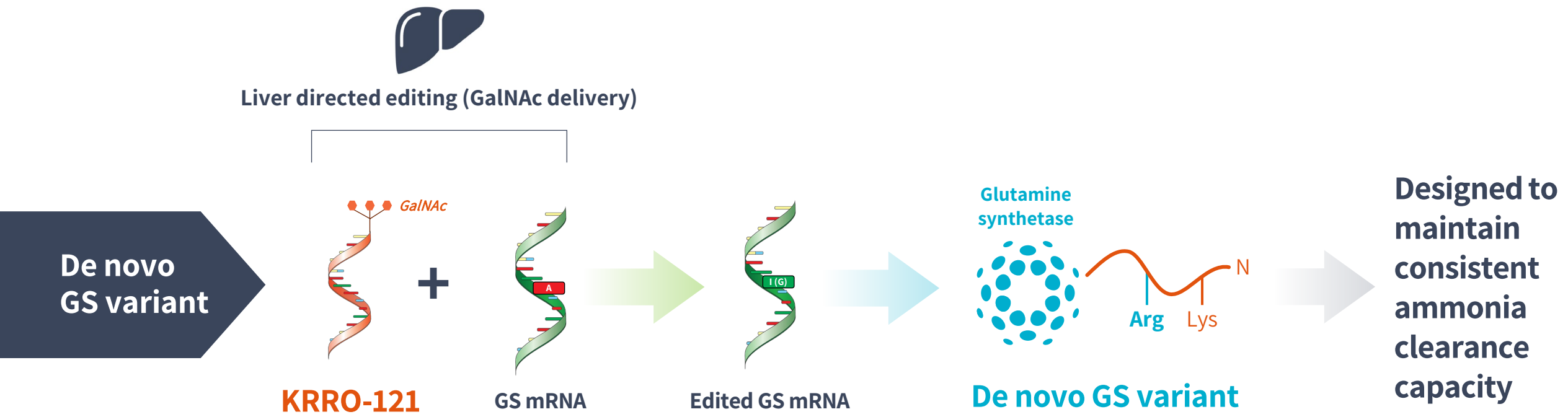
Hypothesis: Preventing GS Degradation Will Stabilize the Protein and Enable Increased Ammonia Clearance

Liver-specific GS modification may prevent degradation, increase ammonia clearance





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



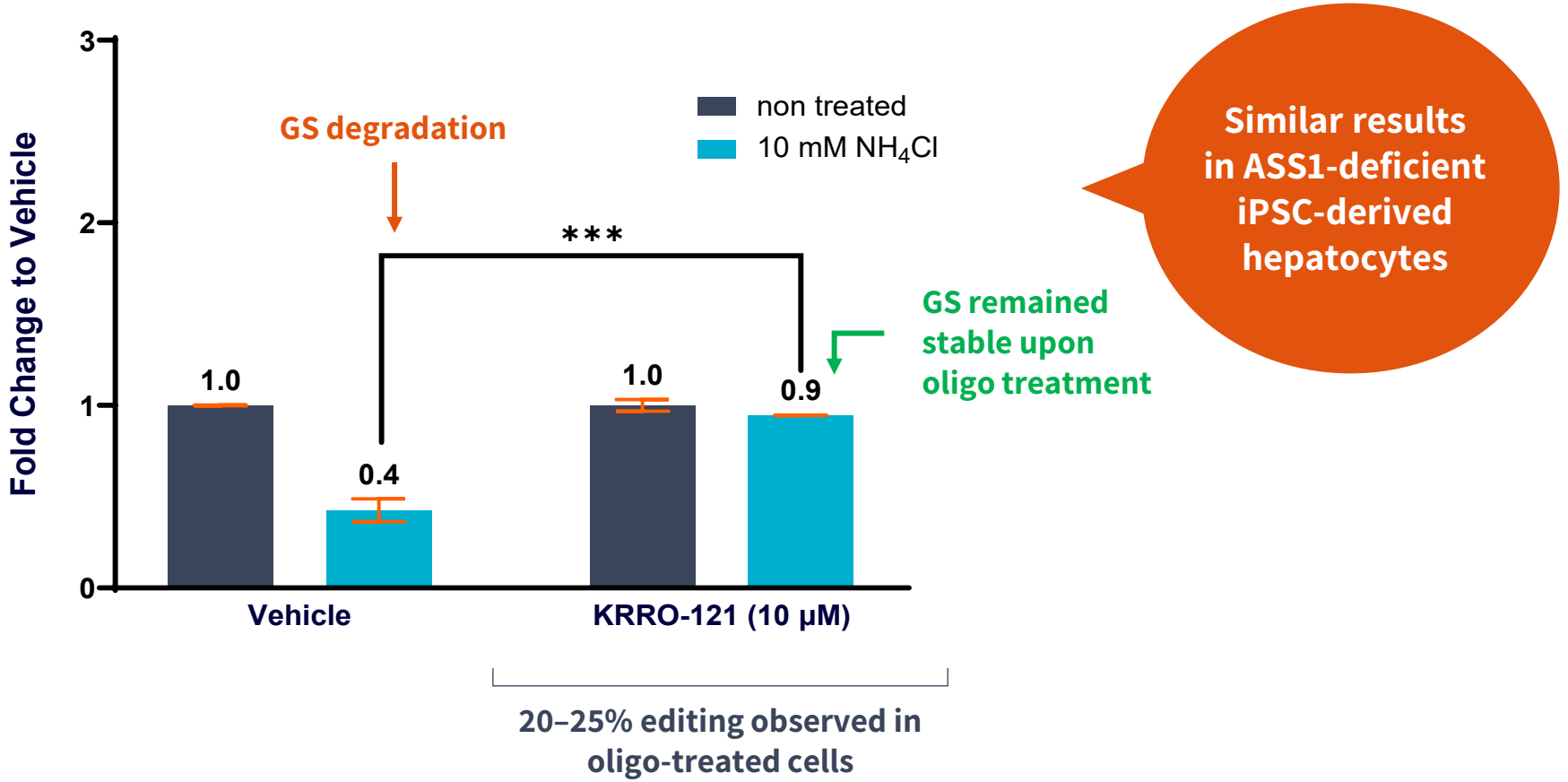
KRRO-121: GalNAc-conjugated oligonucleotide designed for liver-specific RNA editing of GS to enhance ammonia clearance capacity

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

OTC iPSC

KRRO-121

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

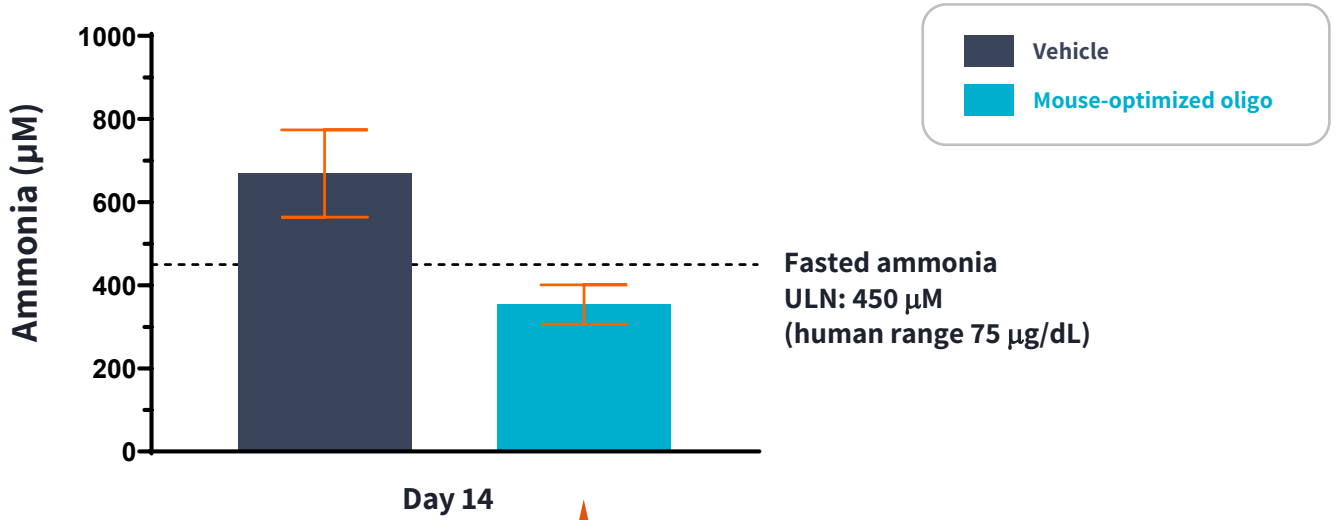


Note: OTC D175V human iPSC-derived hepatocytes differentiated for 14 days, then treated with oligo for 48 hours where indicated (10 mM NH_4Cl added after 24 hours where indicated).
GS concentration measured at conclusion of 48-hour incubation.

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

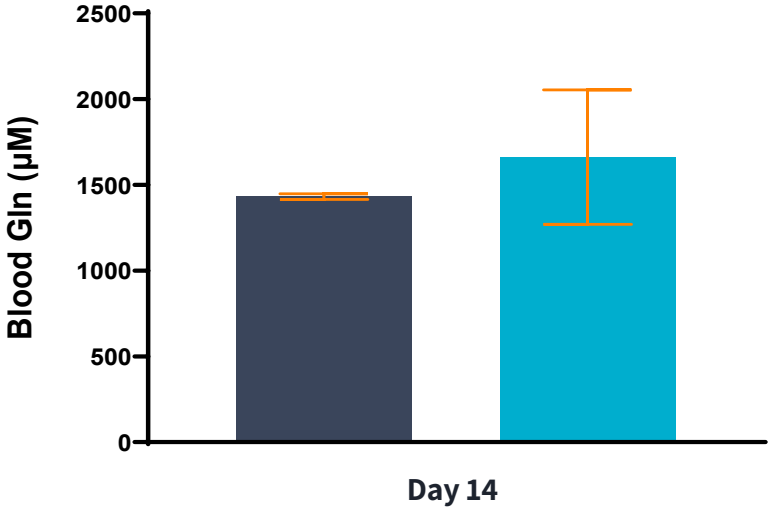
OTC spf/ash

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

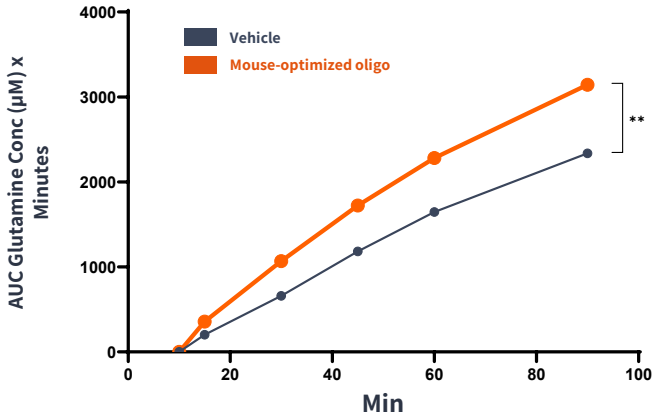


Note: Vehicle or Mouse-optimized oligo dosed at 10 mg/kg-SC daily on Days 0-4. Ammonia and glutamine measured following ammonia challenge (150 mg/kg) on Day 14

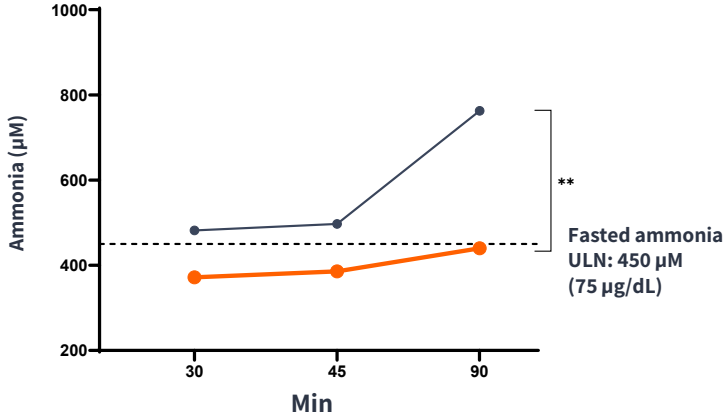
De Novo GS Variant Enabled Ammonia Control in OTC Mice Under Protein Load, with Stable Isotope Tracer Validating MOA

OTC spf/ash

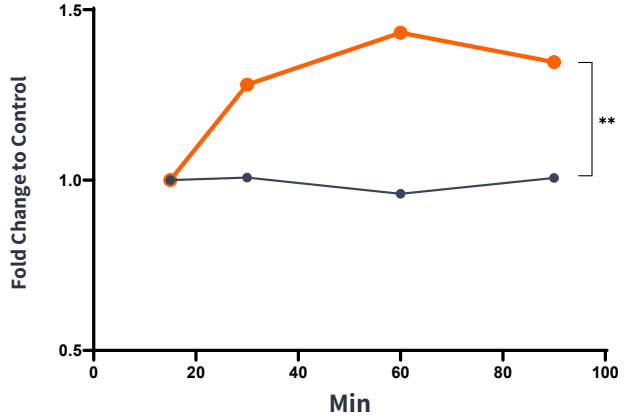
Increased Plasma N-15 Glutamine



Decreased Plasma Ammonia



Increased Total Liver GS Concentration



N-15 glutamate used as target engagement tracer

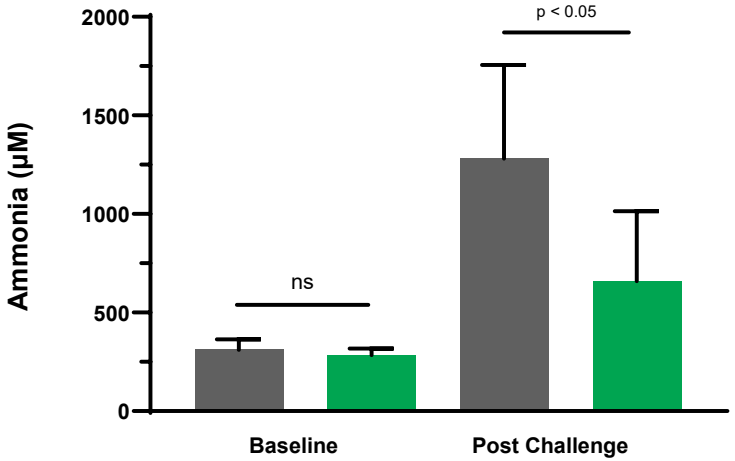
Demonstrated GS target engagement in OTC-deficient mice; similar results observed in wild-type mice (not shown)

Note: Vehicle or Mouse-optimized oligo dosed at 10 mg/kg-SC daily on Days 0-4. Glutamine, ammonia, and GS concentration measured following challenge with 100 mg/kg glutamine + N-15 glutamate on Day 11

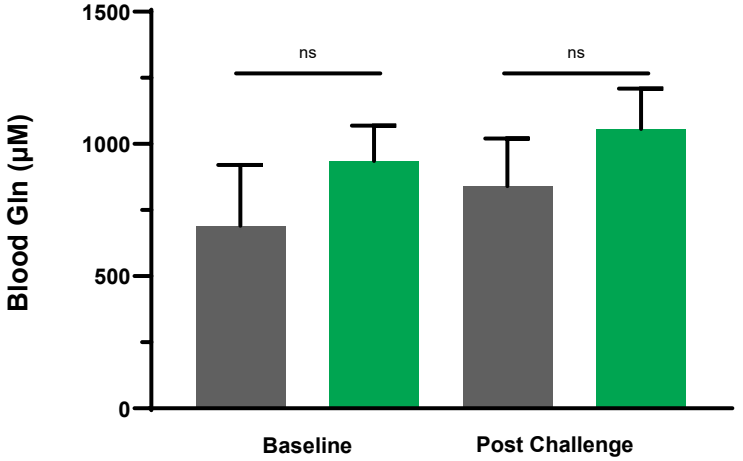
Ammonia Reduction in CPS-1 Deficient Mice Further Validates Potential Pan-UCD Applicability and Diet Liberalization

CPS-1

Reduction in Ammonia Following Ammonia Challenge



Nonsignificant Increase in Plasma Glutamine Levels



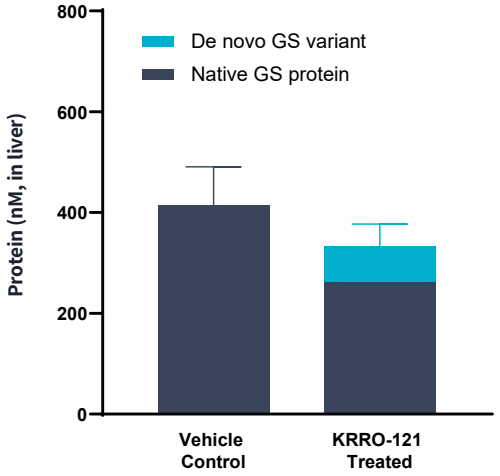
Note: Vehicle or Mouse-optimized oligo dosed at 10 mg/kg-SC daily on Days 0-4. Ammonia and glutamine measured following ammonia challenge (150 mg/kg) on Day 8

KRRO-121 Significantly Reduced Ammonia Levels in Basal State and Following Ammonia Challenge in Humanized Liver Mouse Model

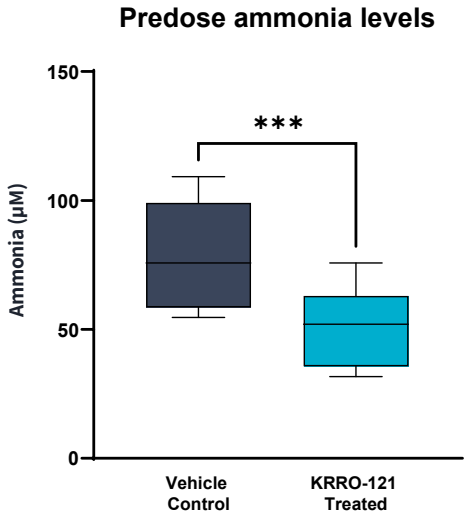
PXB

KRRO-121

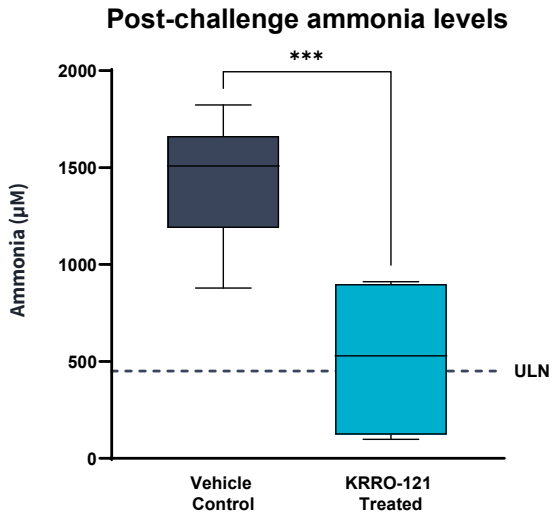
Stabilized GS Variant and Normal GS Protein Levels



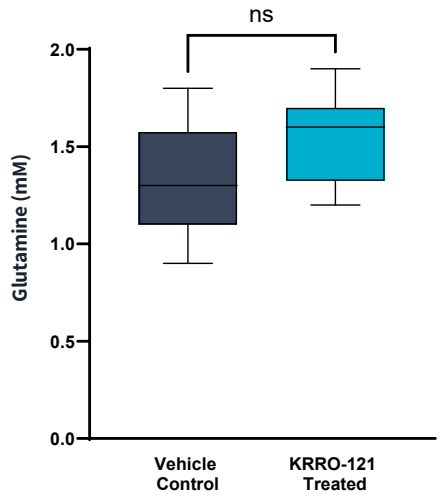
Reduction in Basal Ammonia



Enhanced Ammonia Clearance in Challenge



Steady Glutamine Post-Challenge



Potent ammonia lowering through a minimal amount of de novo GS

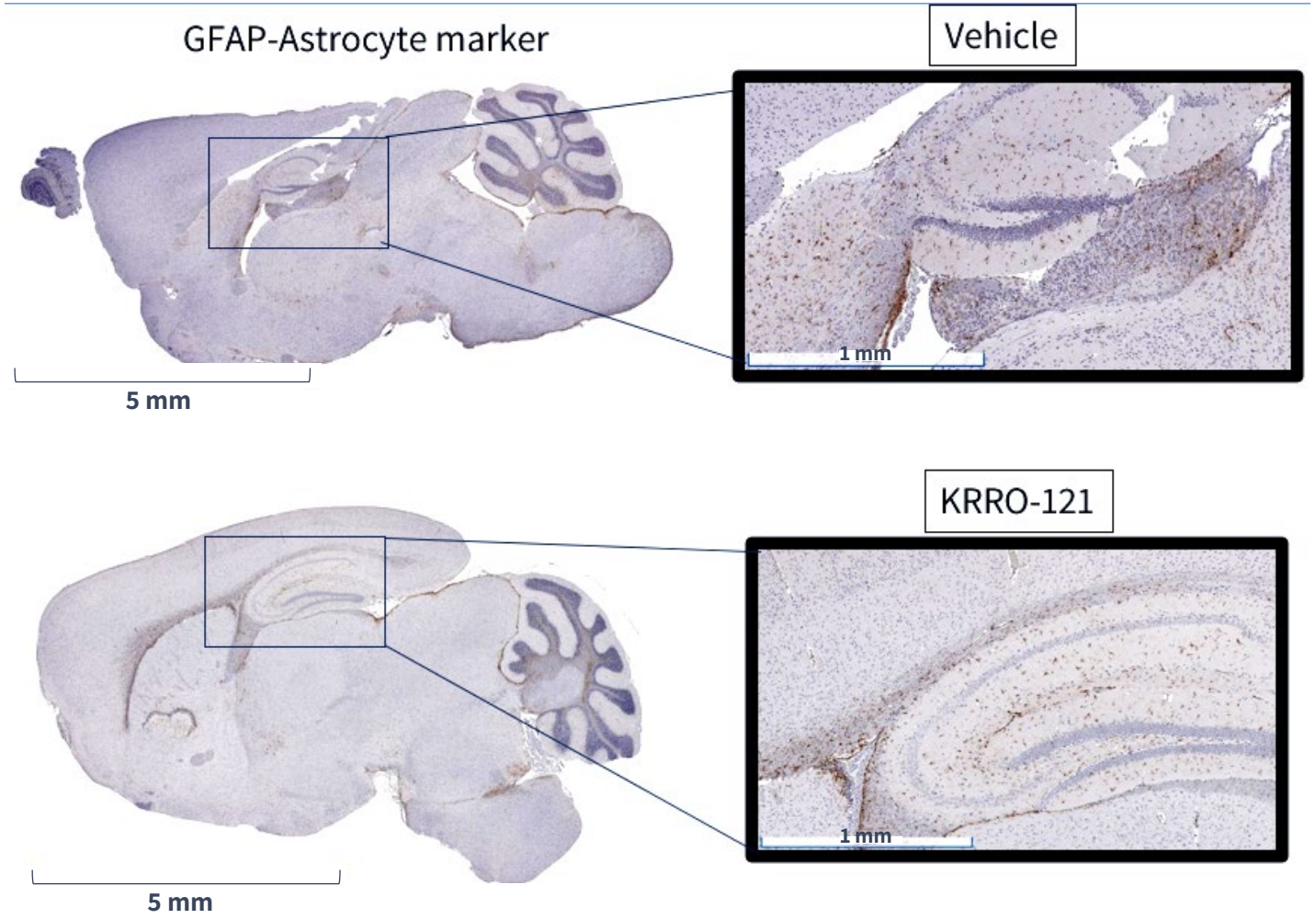
KRRO-121 stabilized GS levels, providing robust ammonia control in a humanized mouse model

Note: Vehicle or KRRO-121 dosed at 50 mg/kg-SC on Days 0, 14 and 28. Mice challenged with 350 mg/kg ammonia on Day 21. GS levels measured on Day 31. PXB mice retain zonal GS expression.
Source: J Toxicol Pathol 2025; 38: 183–189

KRRO-121 Showed No Increase in Astrocyte Activation in Brain

OTC spf/ash

KRRO-121



GFAP+ cells represented as brown dots

Note: Vehicle or KRRO-121 dosed at 20 mg/kg-SC daily on Days 0-4. Editing and GFAP measured following ammonia challenge (150 mg/kg) on Day 14

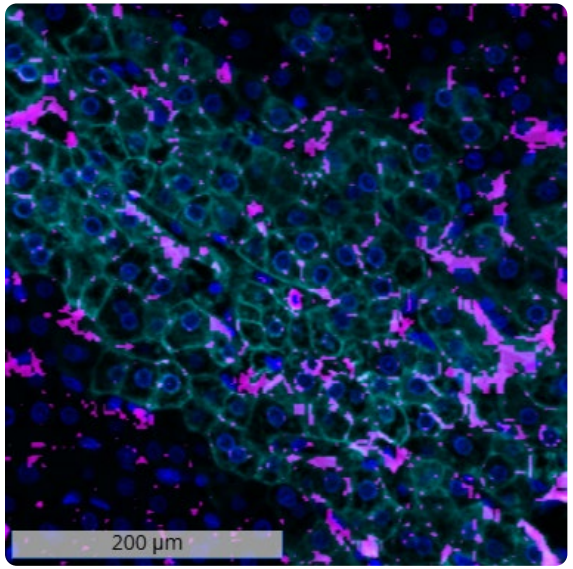
KRRO-121 Displayed Strong Liver Uptake and No Adverse Findings in Non-Human Primates

KRRO-121

>90% Delivery of KRRO-121 to Liver

<0.05% delivery to bone marrow, brain, heart, lymph nodes and muscle

Confirmed Liver Localization of KRRO-121 with Pericentral GS

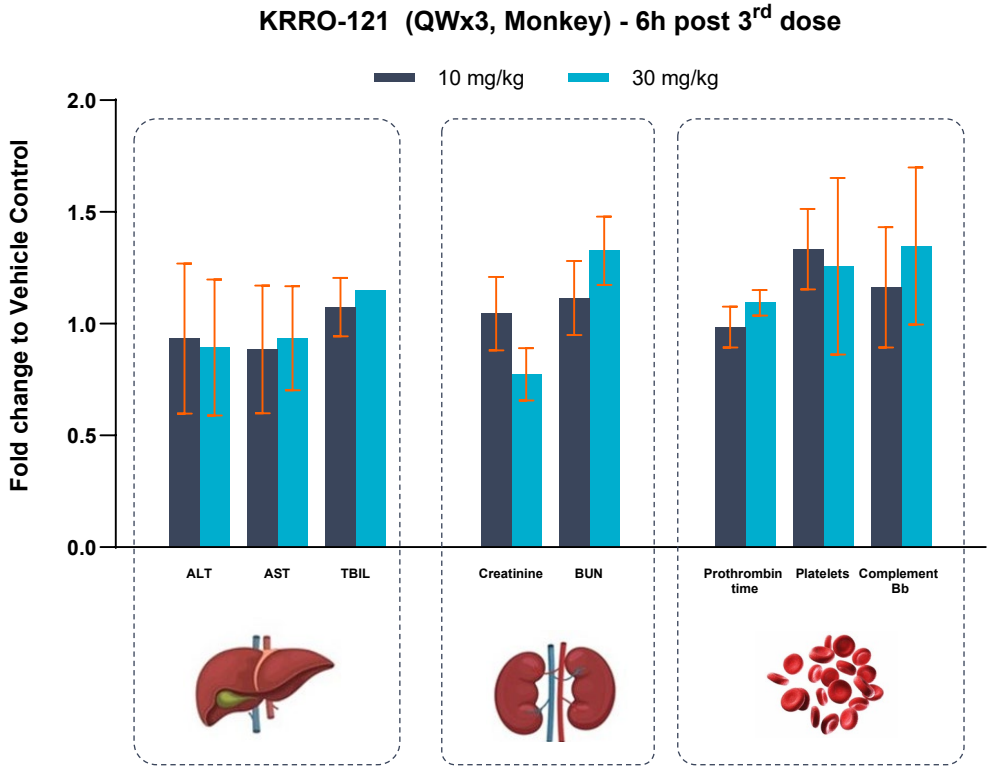


Cy5 (purple) = KRRO-121

Cy7 (teal) = GS

DAPI (blue) = nuclei

No Changes in Liver or Kidney Function



Note: Vehicle or KRRO-121 dosed to cynomolgus monkeys (n=3 male/group) QWx3 (10 or 30 mg/kg), samples collected for biodistribution and histopathology 48h post third dose and clinical chemistries and hematology 6 and 48h post first and last dose

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

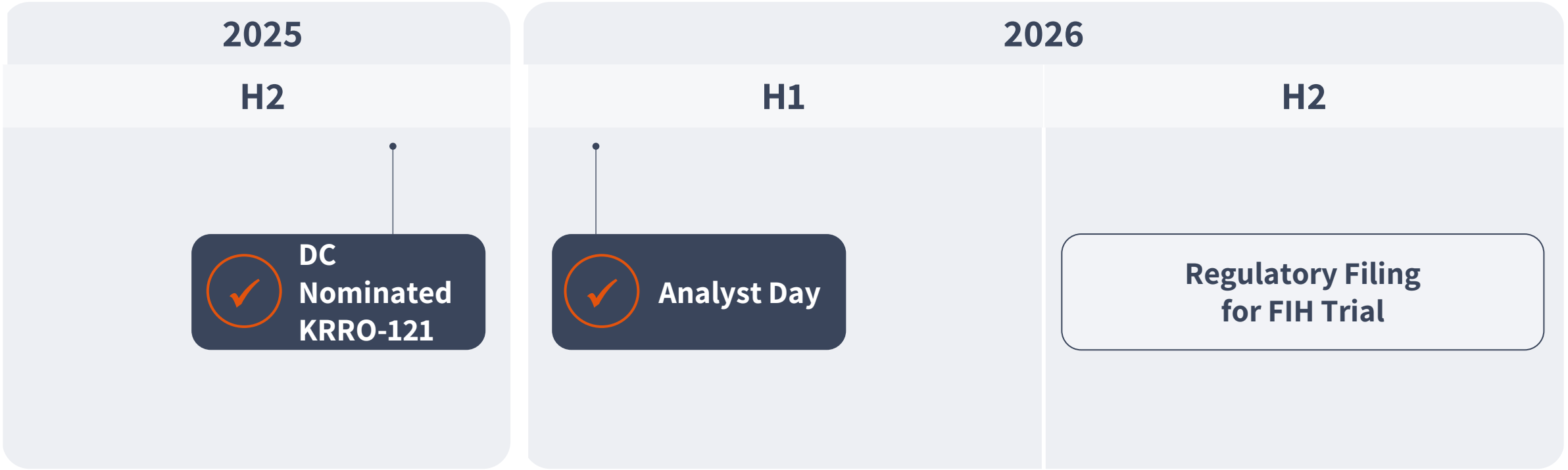
Demonstrated Translation

- 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

KRRO-121: Anticipated Regulatory Filing in Second Half of 2026



Compelling product profile for controlling ammonia expected to drive strong patient engagement and recruitment

KRRO-121 Market Opportunity

Todd Chappell, MBA

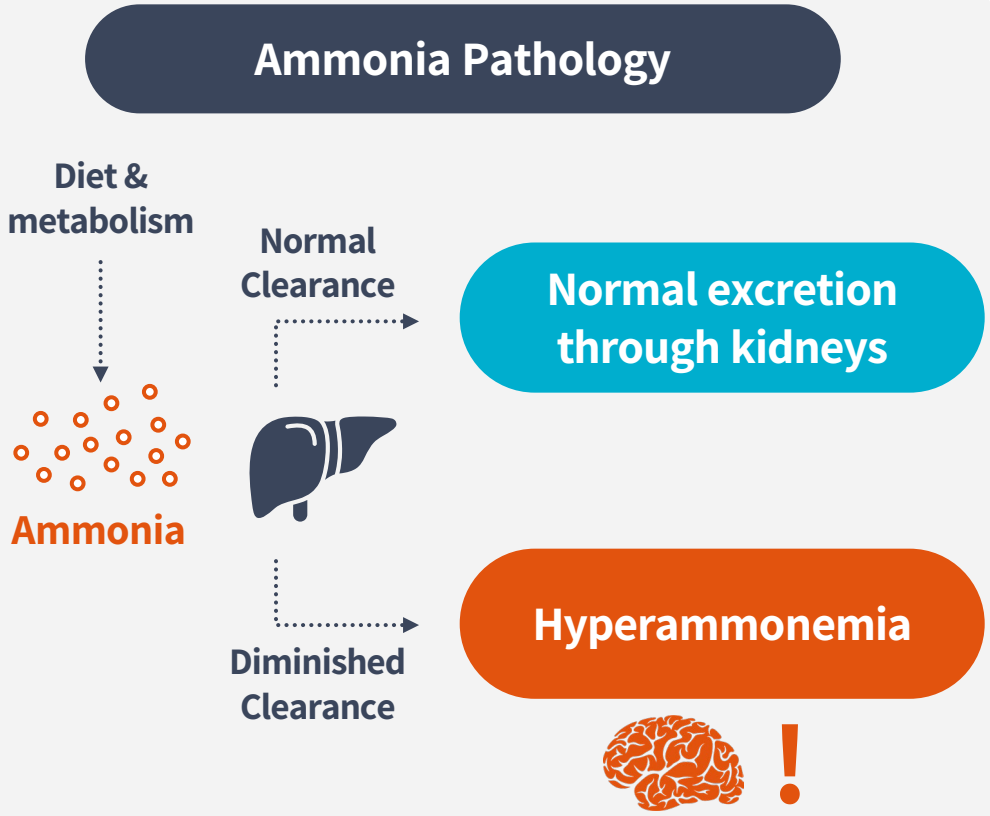
Chief Operating Officer

KRRO-121 Has Blockbuster Potential in Multiple Indications

	Urea Cycle Disorders	Hepatic Encephalopathy
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.

Plasma Ammonia Significantly Impacts Pathology Across Multiple Diseases

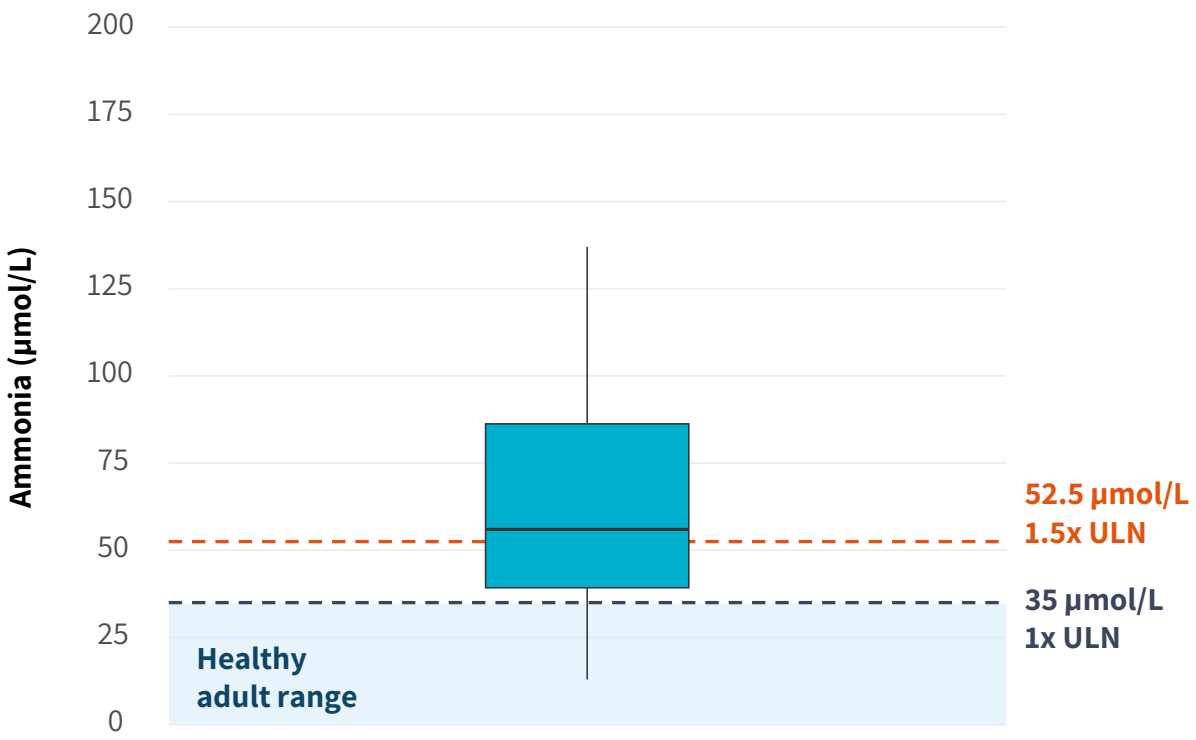


- **High ammonia** leads to:
  - Neurological impairment, potentially permanent
  - Frequent hospitalization
  - Highly restricted diet
  - Elevated infection risk
  - Additional non-neurological complications
- Can be caused by **cirrhosis or urea cycle dysfunction**
- Clinical studies have shown benefit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

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

Uncontrolled Ammonia is a Persistent Danger for UCD Patients

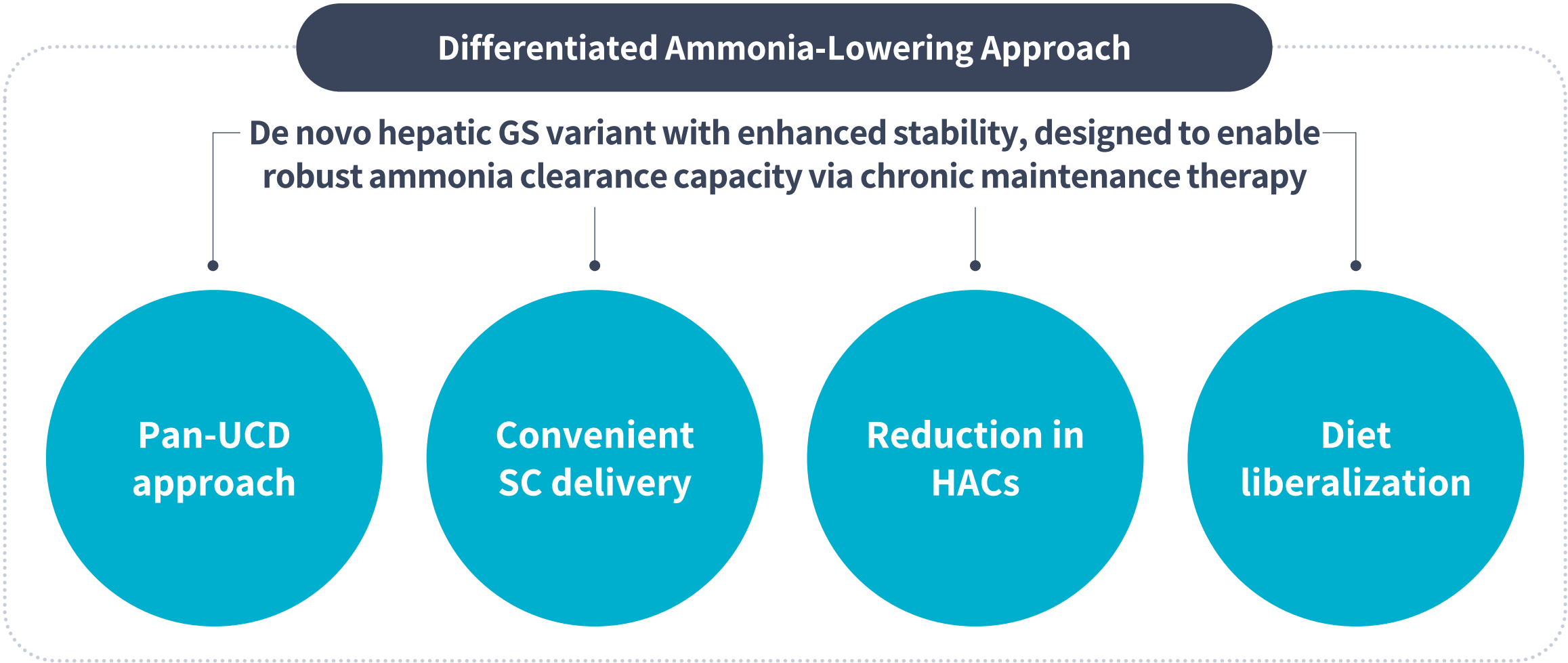
Ammonia Frequently >1.5x ULN in UCD, Leading to Increased Hyperammonemia Risk



Ammonia control is highly challenging in UCD patients today, often requiring nitrogen scavengers + strict diet that can lead to malnutrition

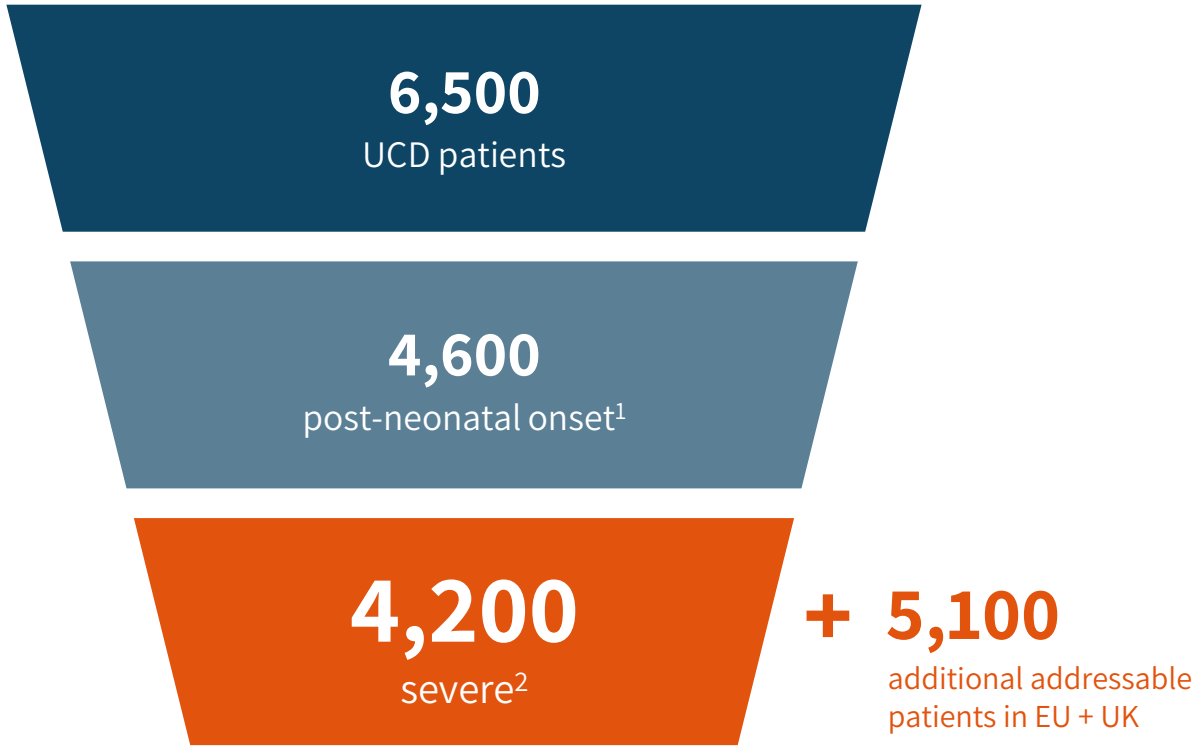
Note: 1. ~150 measurements in 16 patients with confirmed SNPs associated with urea cycle enzymes. ULN – Upper limit of normal
Source: Electronic medical records analysis (data from 2022)

KRRO-121 is Designed to Have a Compelling Product Profile to Potentially Address UCD Patients with Substantial Unmet Need



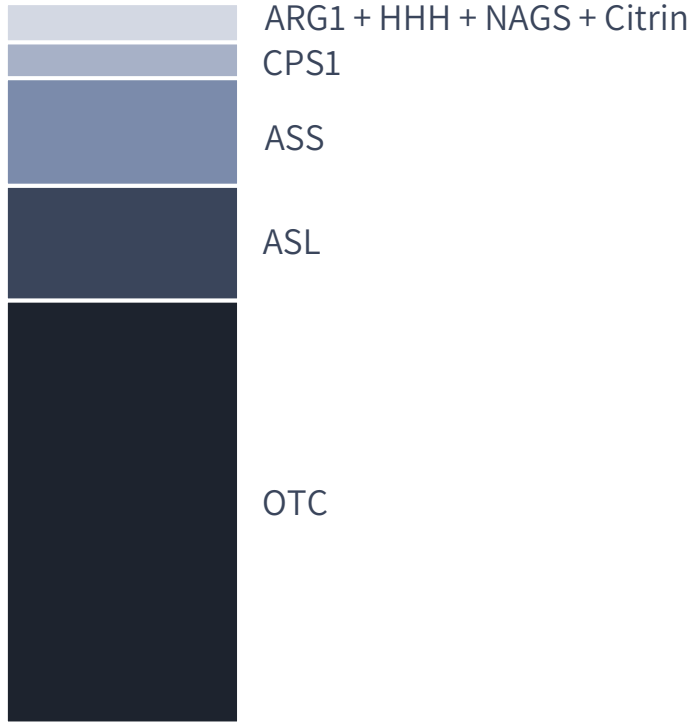
KRRO-121 Can Potentially Address Patients Across All UCD subtypes

U.S. UCD Epidemiology



+ 5,100
additional addressable patients in EU + UK

UCD Subtypes

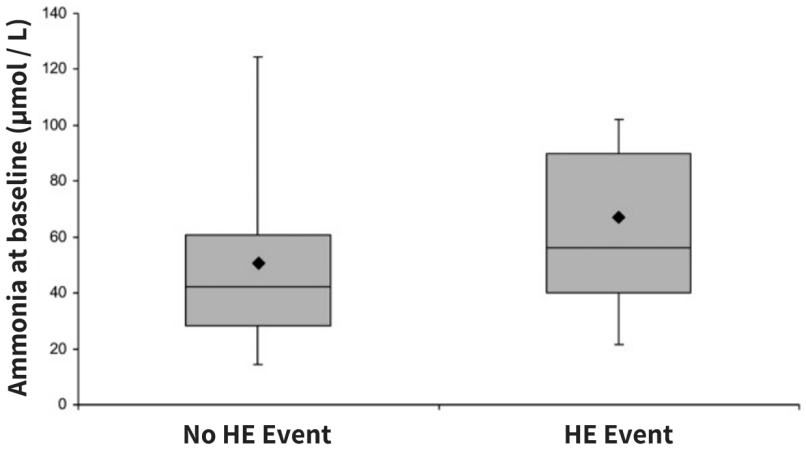


Note: 1. Onset of symptoms at age >1 month; 2. Severe defined as symptomatic patients expected to benefit from pharmacological therapy
Source: 3rd party primary market research study (April 2025); KOL interviews; GlobalData. All figures approximate.

Ammonia Measurements in Uncontrolled HE Patients Are Frequently Above Normal, Correlating with Higher HE Risk

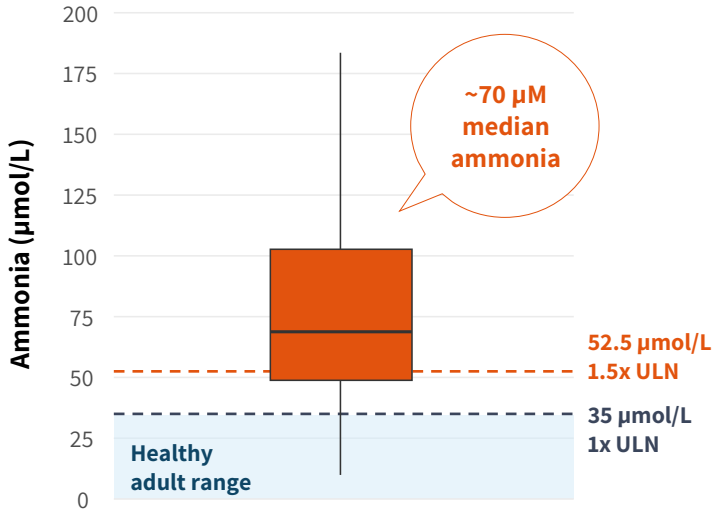
HE Events Correlate with Ammonia

Glycerol Phenylbutyrate Phase 2 HALT-HE Study

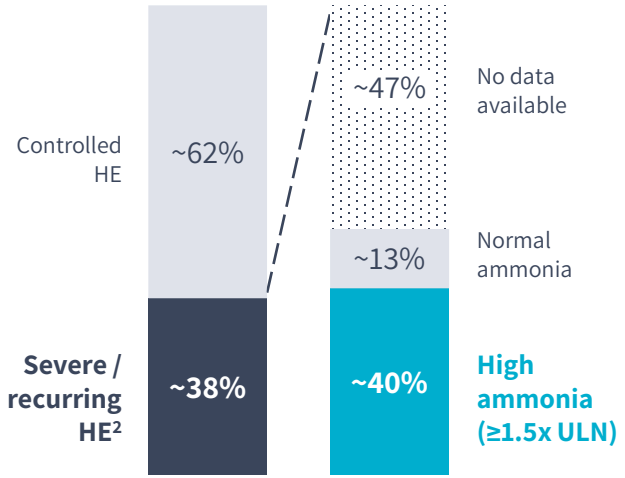


Ammonia Elevated in Many Severe / Recurring HE Patients[1]

Ammonia Measurements: Severe / Recurring HE



HE Patient Segmentation

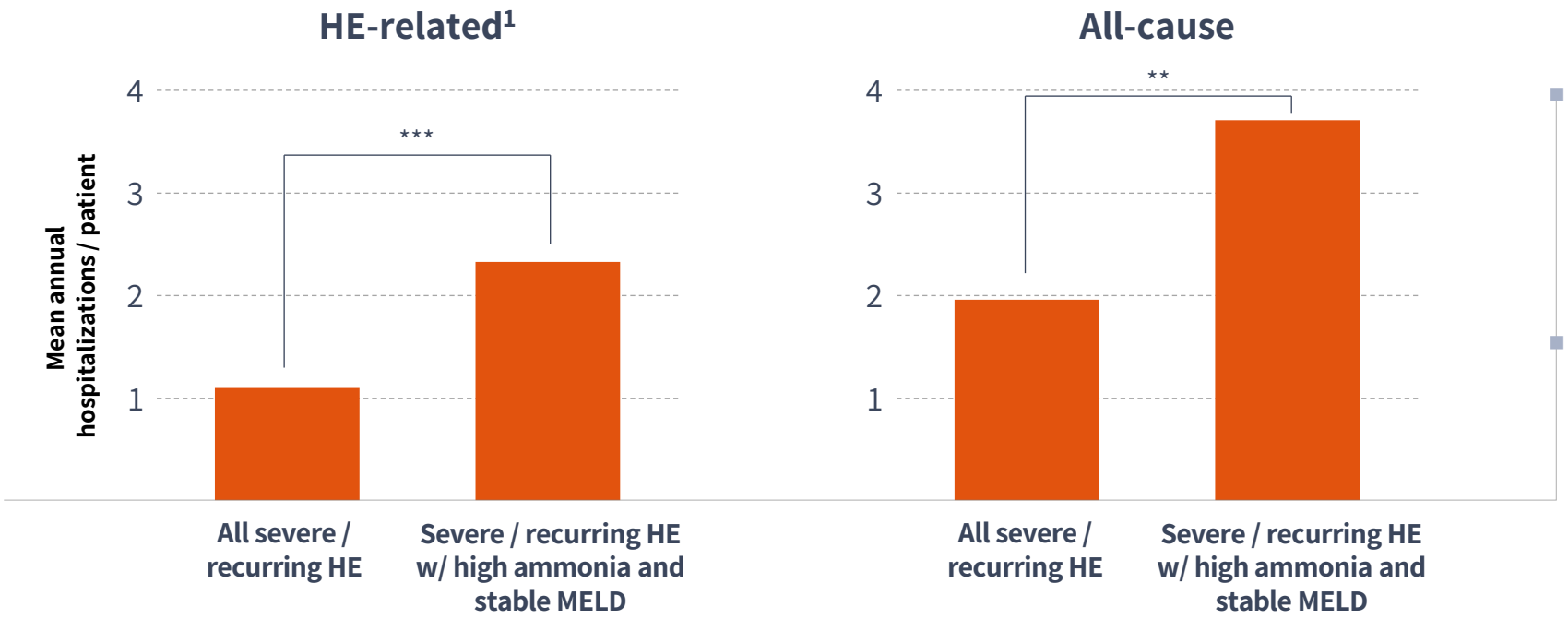


~76% of severe/ recurring HE patients with available ammonia data have an elevation ≥1.5x ULN[3]

Note: 1. 523 measurements from HE patients with rifaximin exposure in 2022 (27 outliers excluded from graph as defined by Q3 + 1.5 x IQR or Q1 – 1.5 x IQR); 2. Cirrhosis patients with exposure to rifaximin (+/- lactulose);
3. Excluding patients with no available ammonia data. ULN – Upper limit of normal
Source: Rockey et al., Hepatology (2014); Electronic medical records analysis (data from 2022)

Elevated Ammonia Levels Are Associated with a Greater Healthcare Burden in HE

High Ammonia Significantly Increases Hospitalization Risk

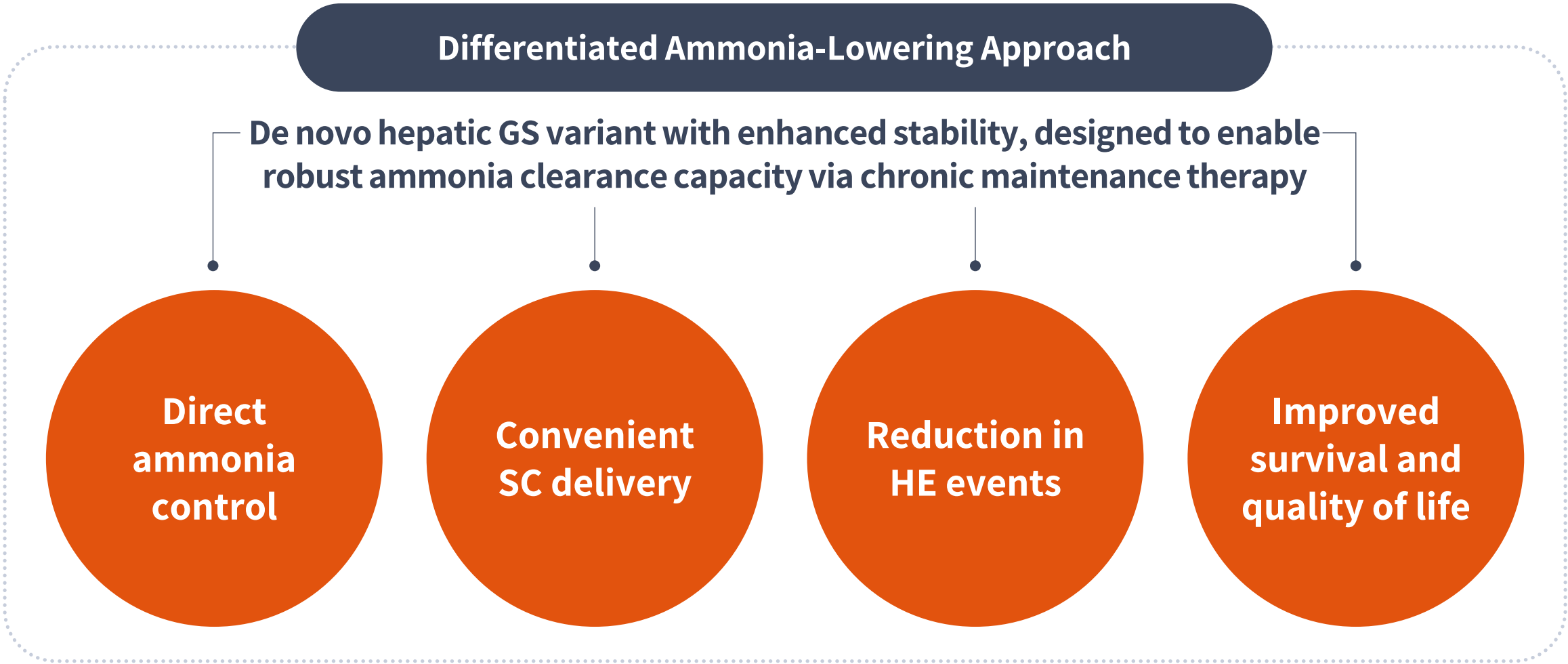


- **>2-fold increase** in HE-related hospitalization for addressable HE patients[2] vs all severe / recurring HE
- **>$10B** inpatient charges for HE in the U.S. each year; average cost per hospitalization **over $75K[3]**

Clear shift towards greater healthcare utilization in HE underscores strong pharmacoeconomic case for treatments that can reduce this burden

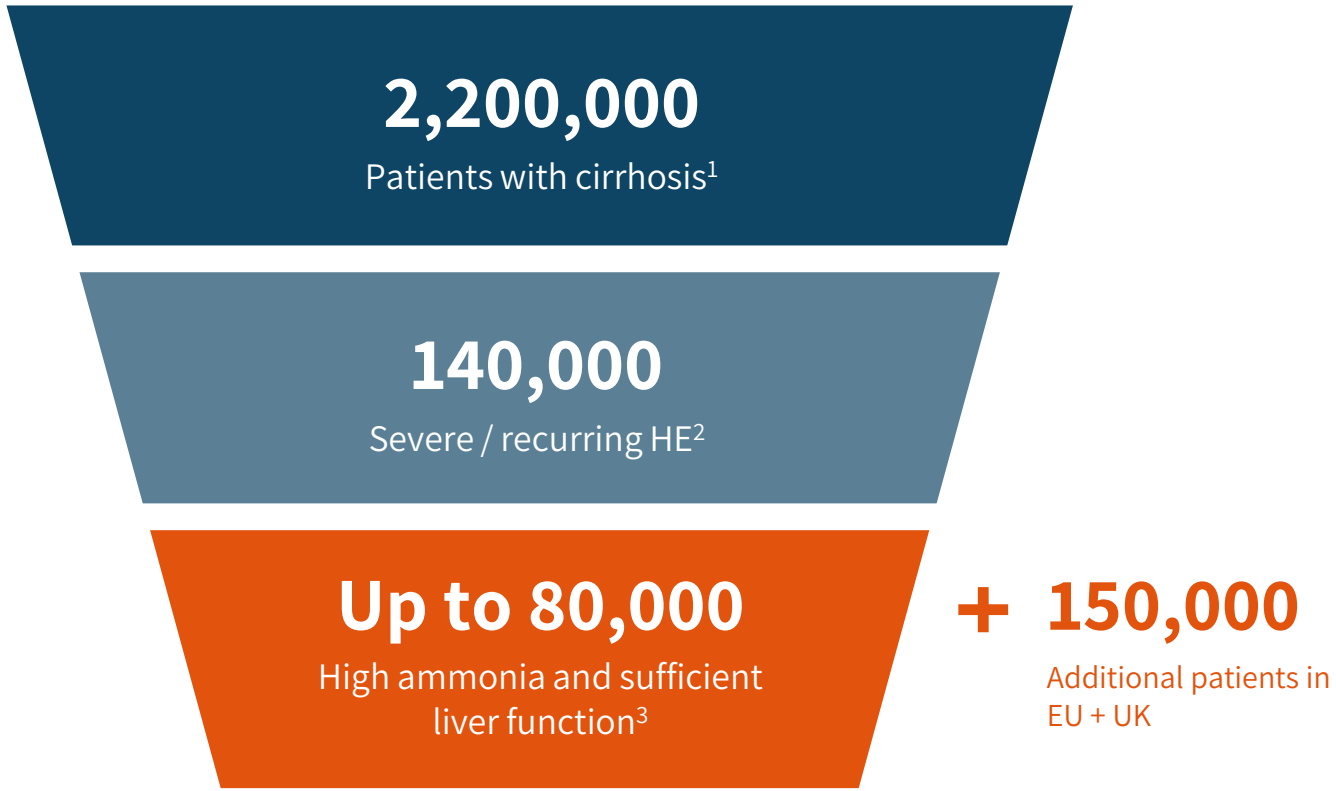
Note: 1. HE-related hospitalization defined as an inpatient visit with a concurrent lactulose prescription; 2. Addressable HE defined as HE patients prescribed rifaximin +/- lactulose with ≥1.5x normal ammonia and MELD ≤20; 3. Average payer costs for HE hospitalization in 2020 based on commercial claims database analysis; ** p <0.01; *** p <0.001
Source: Harris et al., Clinics in Liver Disease (2024); Wong et al., Clin Transl Gastroenterol (2025); Electronic medical records analysis (data from 2022)

KRRO-121 Also Has an Opportunity to Potentially Address Significant Unmet need in HE



Up to ~80K Addressable Patients in the U.S. with Severe / Recurring HE May Benefit from Ammonia-Lowering Treatment

U.S. HE Epidemiology



+ **150,000**
Additional patients in EU + UK

Additional opportunity can be unlocked in prevention of initial HE episode

Note: 1. U.S. cirrhosis prevalence as estimated by Tapper et al (2023); 2. Cirrhosis patients with exposure to rifaximin (+/- lactulose); 3. Ammonia measurement ≥52.5 µM (1.5x upper limit of normal) and average MELD score below 20 (excluding patients where no ammonia data was available)
Source: Tapper et al., JAMA (2023); Electronic medical records analysis (data from 2022). All figures approximate.

Closing remarks

Ram Aiyar, PhD, MBA

Chief Executive Officer

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 modulating disease biology