CHINOOK THERAPEUTICS

Chinook Therapeutics

Developing Precision Medicines for Kidney Diseases

August 2021

Note Regarding Forward-Looking Statements

Certain of the statements made in this presentation are forward looking, including those relating to Chinook's business, future operations, advancement of its product candidates and product pipeline, clinical development of its product candidates, including expectations regarding timing of initiation and results of clinical trials and sufficiency of its cash resources. In some cases, you can identify these statements by forward-looking words such as "may," "will," "continue," "anticipate," "intend," "could," "project," "expect" or the negative or plural of these words or similar expressions. Forward-looking statements are not guarantees of future performance and are subject to risks and uncertainties that could cause actual results and events to differ materially from those anticipated, including, but not limited to, our ability to develop and commercialize our product candidates, whether results of early clinical trials or preclinical studies will be indicative of the results of future trials, our ability to obtain and maintain regulatory approval of our product candidates, our ability to operate in a competitive industry and compete successfully against competitors that may be more advanced or have greater resources than we do, our ability to obtain and adequately protect intellectual property rights for our product candidates and the effects of COVID-19 on our clinical programs and business operations. Many of these risks are described in greater detail in our filings with the SEC. Any forward-looking statements in this presentation speak only as of the date hereof. Chinook assumes no obligation to update forward-looking statements whether as a result of new information, future events or otherwise, after the date of this presentation.

The Time is Now for Kidney Disease Drug Development

Large Unmet Need

~9.1% of global population suffers from kidney disease[1]

Kidney diseases drive >$120B of annual U.S. healthcare costs[2]

Few drugs approved to prevent kidney disease progression

Historical Barriers

Heterogeneous patient populations with many distinct disease drivers

Drugs with non-specific mechanisms

Large and lengthy outcomes-based clinical trials previously required

Clear Development Path

Increased understanding of underlying disease biology

New and validated drug targets

FDA recognizing surrogate markers, such as proteinuria and eGFR, as registration endpoints[3]

1. GBD Chronic Kidney Disease Collaboration, The Lancet, 2020; 395(10225):709-733; 2. USRDS Annual Data Report 2019 (https://www.usrds.org/media/2371/2019-executive-summary.pdf); 3. Thompson et al., CJASN March 2019, 14 (3) 469-481.

Building a Leading Kidney Disease Company

Atrasentan

- Highly potent and selective ET_A antagonist evaluated in more than 5,300 CKD patients
- Phase 2 data in glomerular diseases expected in 2022
- Phase 3 proteinuria data in IgAN expected in 2023

BION-1301

- Anti-APRIL monoclonal antibody (mAb)
- Data demonstrates durable reductions in free APRIL, IgA & Gd-IgA1 levels in HVs and patients with IgAN
- Additional phase 1/2 data in patients in H2 2021

CHK-336

- Oral small molecule LDHA inhibitor with liver-targeted tissue distribution for primary hyperoxaluria
- Potential to treat all disorders of excess oxalate
- Preparing for IND submission & phase 1 HV initiation in late 2021/early 2022

Precision Medicine R&D Pipeline

- Focus on rare, severe chronic kidney diseases
- Design novel, targeted and differentiated molecules
- Execute clinical trials in defined patient populations with surrogate endpoints

Strong cash position with operating capital through H1 2023

Advancing a Diversified Pipeline of Best-in-class Programs

Program	Indication	Target Validation	Lead Optimization	IND-Enabling	Phase 1	Phase 2	Phase 3
Atrasentan	IgA Nephropathy	Phase 3 ongoing ALIGN					
	Basket of glomerular diseases	Phase 2 ongoing AFFINITY					
BION-1301	IgA Nephropathy	Phase 1b ongoing					
CHK-336	Primary Hyperoxaluria	IND-enabling studies ongoing					
Research & Discovery Programs	Rare, severe chronic kidney diseases						

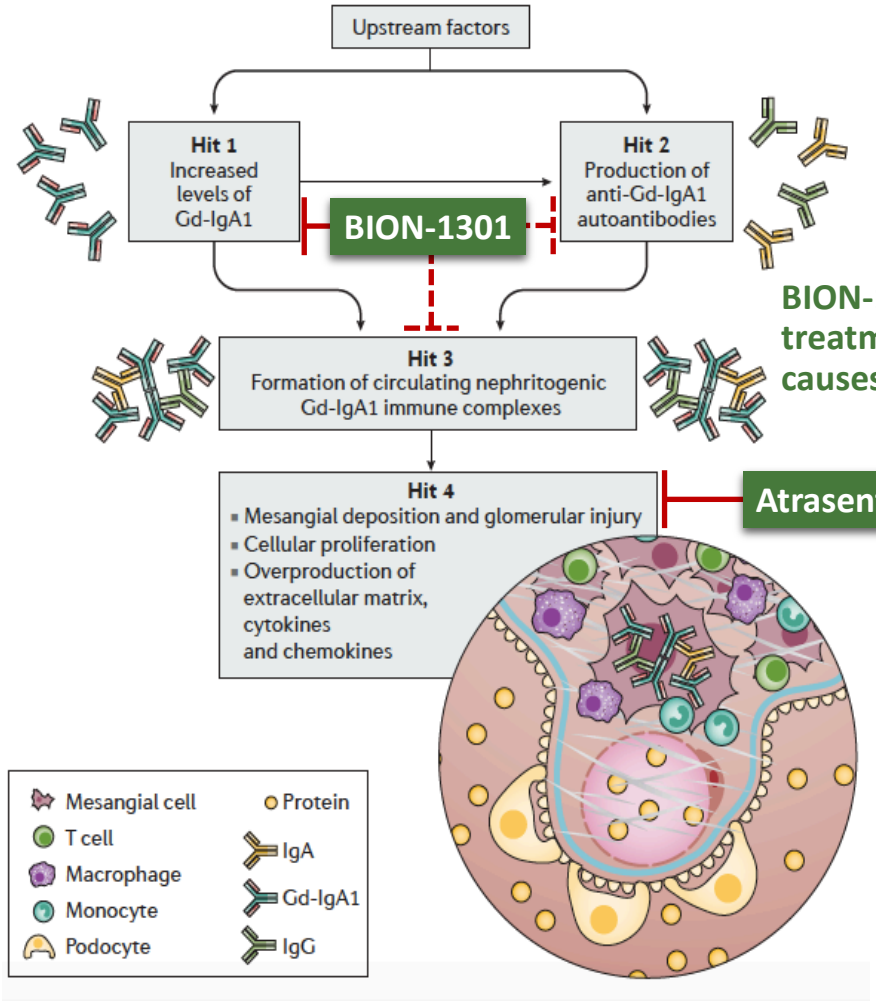
Global commercial rights to all pipeline programs

Continue to evaluate opportunities to add kidney disease programs to pipeline

Why Target IgA Nephropathy?

- Most common primary glomerular disease globally with ~140K – 150K US prevalence
- No approved treatments; current options ineffective for many
- Most important predictor of kidney progression in IgAN is proteinuria
- Proteinuria reduction recognized by FDA as surrogate endpoint for accelerated approval with full approval based on kidney function (eGFR)

IgA Nephropathy Disease Pathophysiology



BION-1301 is a potential disease-modifying treatment that targets the underlying causes of IgAN (Hit 1)

Atrasentan has potential to reduce proteinuria and preserve kidney function with direct vascular, anti-inflammatory and anti-fibrotic effects (Hit 4)

Wyatt, R. J., & Julian, B. A. (2013). IgA nephropathy. New England Journal of Medicine, 368(25), 2402-2414; Thompson et al., CJASN March 2019, 14 (3) 469-481.
Lai, K., Tang, S., Schena, F. et al. IgA nephropathy. Nat Rev Dis Primers 2, 16001 (2016).

CHINOOK THERAPEUTICS

CHINOOK THERAPEUTICS

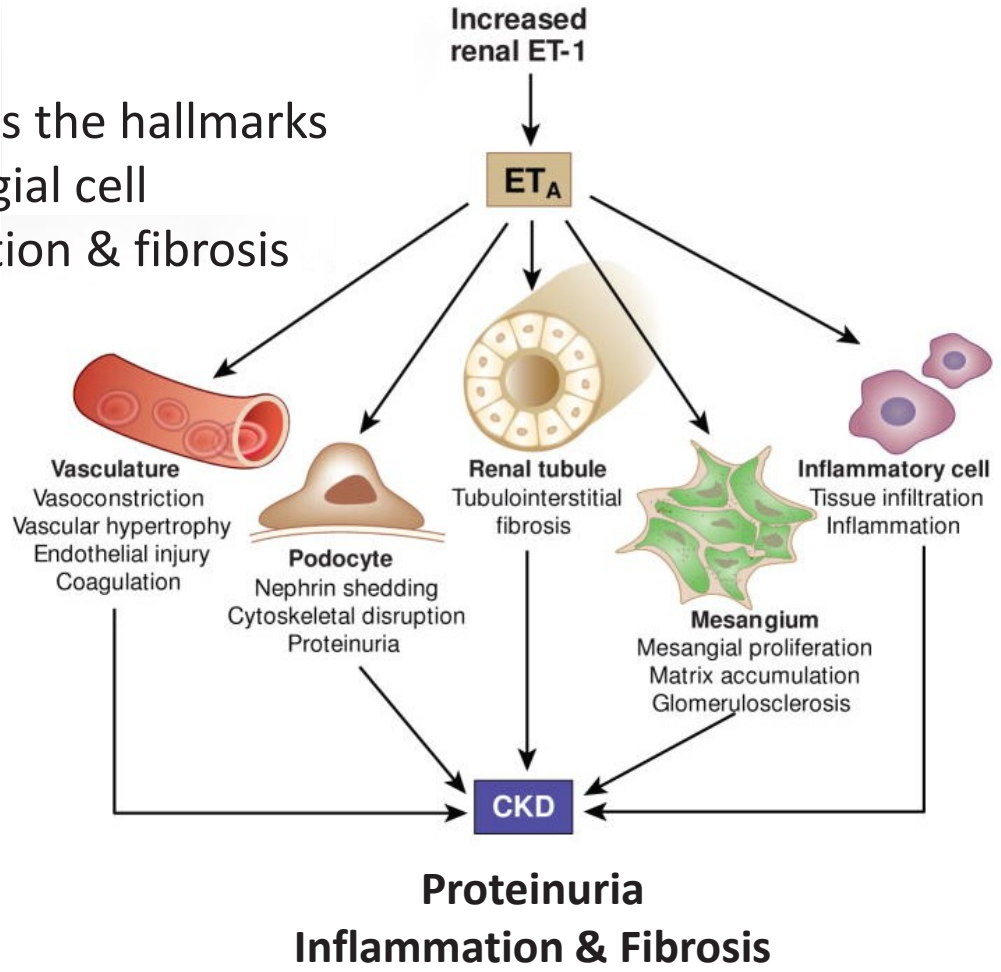
Atrasentan

Potent and Selective Endothelin A Receptor Antagonist

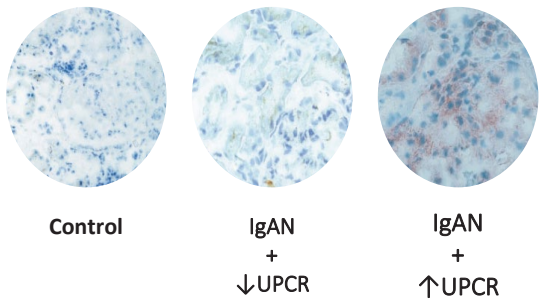
Atrasentan: a Potent and Selective ET_A Antagonist

ET_A receptor activation drives IgAN progression through multiple potential mechanisms

- ET_A receptor activation drives the hallmarks of IgAN: proteinuria, mesangial cell activation, kidney inflammation & fibrosis



Intense kidney ET-1 & ET_A receptor immunostaining in IgAN patients with significant proteinuria



- ET system activation appears to be a key molecular determinant of progressive IgAN
- Elevated kidney ET-1 expression strongly predicts progression of IgAN
- **ET_A receptor blockade through atrasentan is a promising approach to treat IgAN patients**

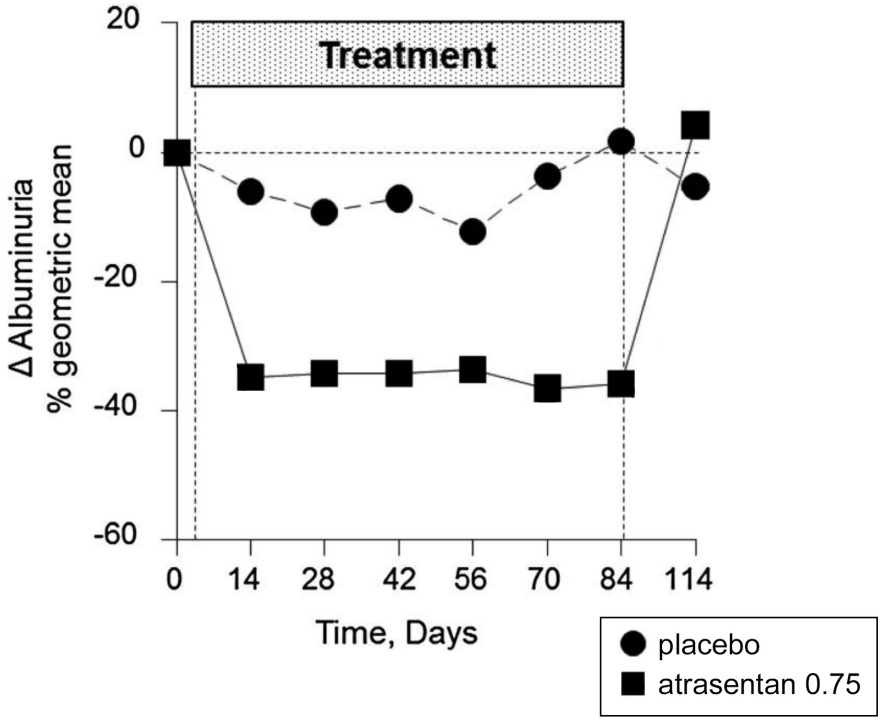
Tycova et al. Physiol. Res. 67: 93-105, 2018; Lehrke et al. J Am Soc Nephrol 2001 12: 2321–2329; Zanatta et al, Renal Failure, 2012, 34: 308–315; Kohan DE et al., Kidney Int. 2014.

CHINOOK THERAPEUTICS

AbbVie Evaluated Atrasentan in >5,300 DKD Patients

Potential to benefit IgAN patients with a rapid registration pathway

Proteinuria



UACR (percent change in geometic mean from baseline) in AbbVie phase 2 RADAR study

Strong rationale for development in IgAN

- Picomolar potency and highly selective for ET_A
- Optimal dose of 0.75 mg daily established
- Rapid and sustained ~30-35% proteinuria reductions consistently observed in phase 2 and 3
- Improved hard kidney outcomes in phase 3 SONAR study
- Well-characterized and acceptable safety profile
- Optimized tolerability anticipated in younger IgAN patients

J Am Soc Nephrol. 2014 May; 25(5): 1083–1093.

Global SONAR Phase 3 Outcome Trial in DKD

SONAR Topline Results

3,600 high-risk DKD patients randomized and treated for up to 5 years (median 2.2 years)

35% — Decreased risk of ESRD or doubling of serum creatinine in responders* (28% in all randomized patients)

30-35% — Proteinuria (UACR) reduction

0.0005 — p-value for eGFR preservation in responders*

Safety profile consistent with class | **Clinically manageable fluid retention**

"These data support a potential role for selective endothelin receptor antagonists in protecting renal function in patients with type 2 diabetes at high risk of developing end-stage kidney disease." - Heerspink et al.


THE LANCET

Articles

Atrasentan and renal events in patients with type 2 diabetes and chronic kidney disease (SONAR): a double-blind, randomised, placebo-controlled trial

Hiddo J L Heerspink, Hans-Henrik Parving, Dennis L Andress, George Bakris, Ricardo Correa-Rotter, Fan-Fan Hou, Dalane W Kitzman, Donald Kohan, Hirofumi Makino, John J V McMurray, Joel Z Melnick, Michael G Miller, Patricio E Pergola, Vlado Perkovic, Sheldon Tobe, Tingting Yi, Melissa Wigderson, Dick de Zeeuw, on behalf of the SONAR Committees and Investigators*

*Responders classified as patients who achieved >30% UACR reduction following 6-week enrichment period

Heerspink et al., Diabetes Obes Metab. 2018;20:1369–1376
Heerspink et al., SONAR Trial, Lancet 2019.

Atrasentan Clinical and Regulatory Plan

Phase 3 Targeting IgAN patients at High Risk for Disease Progression (ongoing)

ALIGN

- Biopsy-proven IgAN
- Patients on maximally-tolerated, optimized and stable dose of RASi or RASi intolerant
- Proteinuria >1 g/day and eGFR >30 ml/min
- ~320 pts, 1:1 placebo randomization
- Global study with ~140 sites
- 6-month proteinuria primary endpoint (accelerated approval)
- 2.5 year eGFR secondary endpoint (full approval)

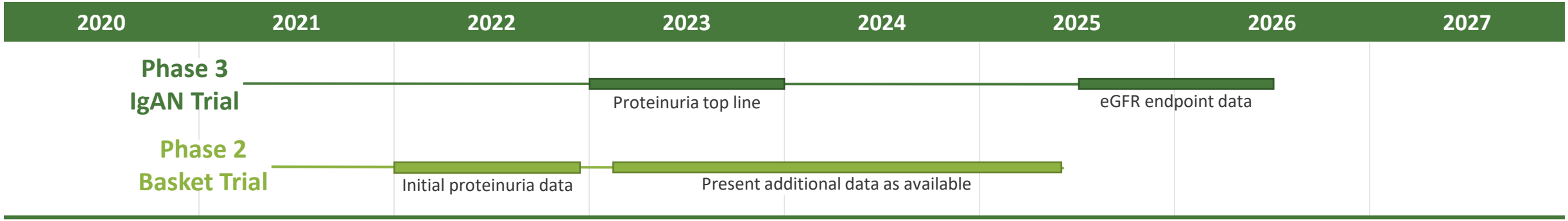
Phase 2 Basket Trial to Expand Potential Across Proteinuric Glomerular Diseases

AFFINITY

- Open-label design, 12-week proteinuria primary endpoint
- ~20 patients / cohort
- Overlap with phase 3 sites to support enrollment

Cohorts include:

- IgAN with proteinuria 0.5 – <1 g/day
- FSGS
- Alport Syndrome
- DKD as add on SGLT2 inhibitors



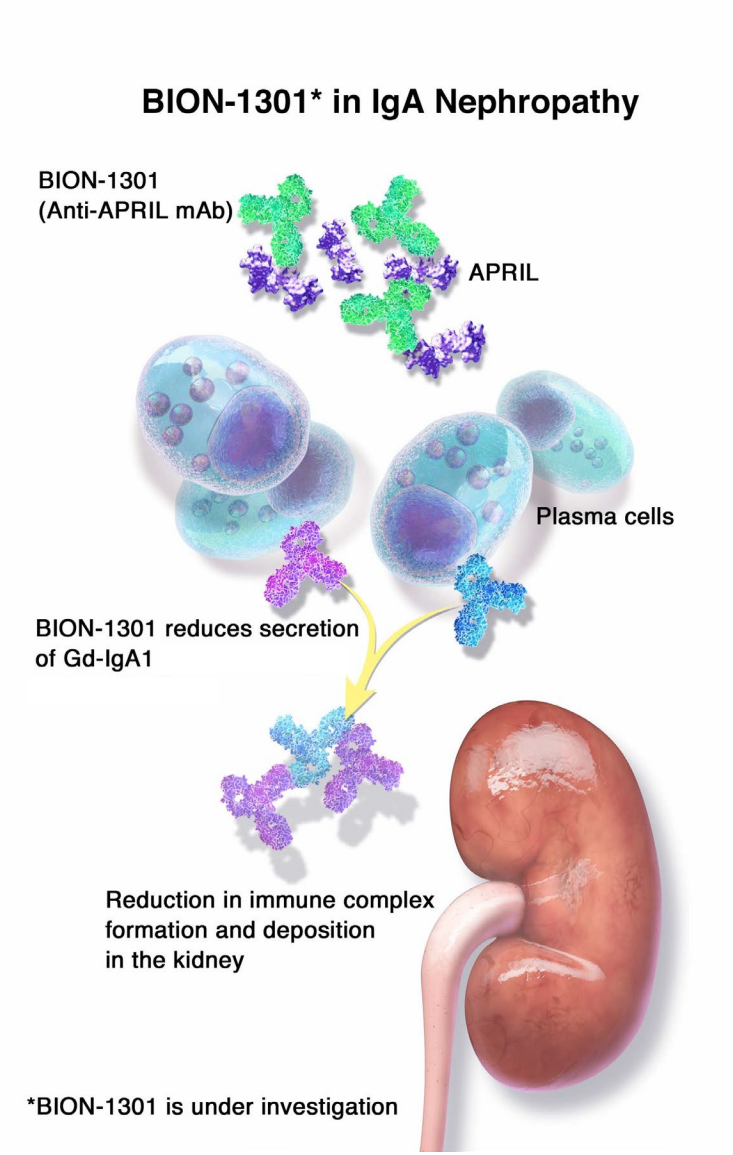
CHINOOK THERAPEUTICS

CHINOOK THERAPEUTICS

BION-1301

Anti-APRIL Monoclonal Antibody

BION-1301: Disease-Modifying Anti-APRIL mAb



APRIL: TNF-family cytokine involved in B-cell signaling[1]

- Drives IgA production and survival of IgA-secreting plasma cells[2]
- Shown to increase Gd-IgA1 secretion[3]
- Higher APRIL levels in IgAN patients correlated with higher Gd-IgA1 and proteinuria and lower eGFR[3]
- APRIL gene variants confer increased risk of IgAN[4]

BION-1301: humanized IgG4 monoclonal antibody that blocks APRIL binding to its receptors

- Potentially disease-modifying mechanism to deplete Gd-IgA1 (**Hit 1**) and prevent pathogenic immune complex formation (**Hit 3**)
- No adverse effects reported in NHP tox studies of IV BION-1301 for up to 6 months and SC for up to 1 month[5]
- Well-tolerated up to 2700mg in phase 1 multiple myeloma study[6]

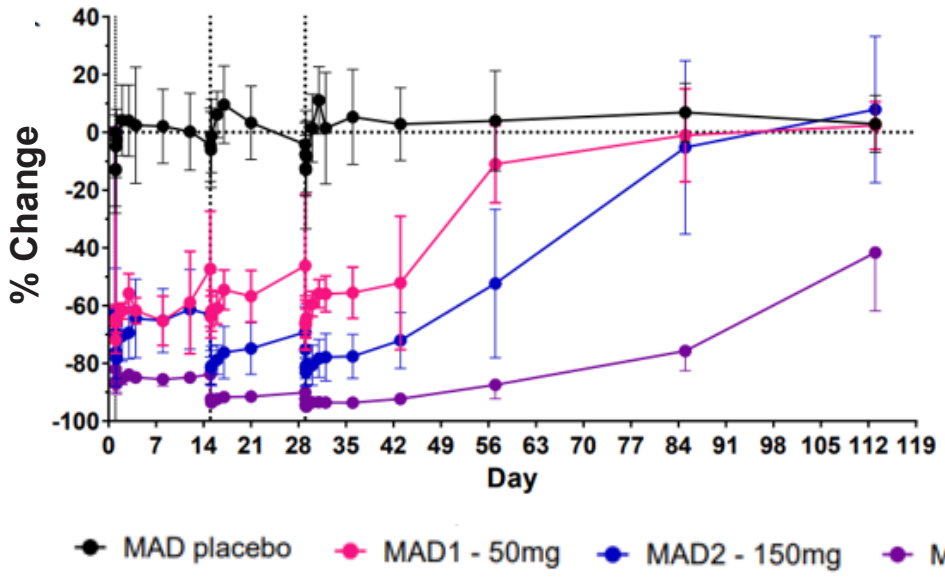
1. Guadagnoli, M, et al. Blood. 2016. 2. He B, et al. Nat Immunol. 2010; 3. Zhai YL, et al. Medicine (Baltimore). 2016. 4. Yu XQ, et al. Nature Genetics. 2012 Feb;44(2):178-182.
5. Kreijtz J, et al. ERA-EDTA 2020 poster presentation: poster #P0379. 6. Bensinger W, et al. ASCO 2019 poster presentation: poster #338.

BION-1301 Reduced Free APRIL, IgA & Gd-IgA1 in HVs

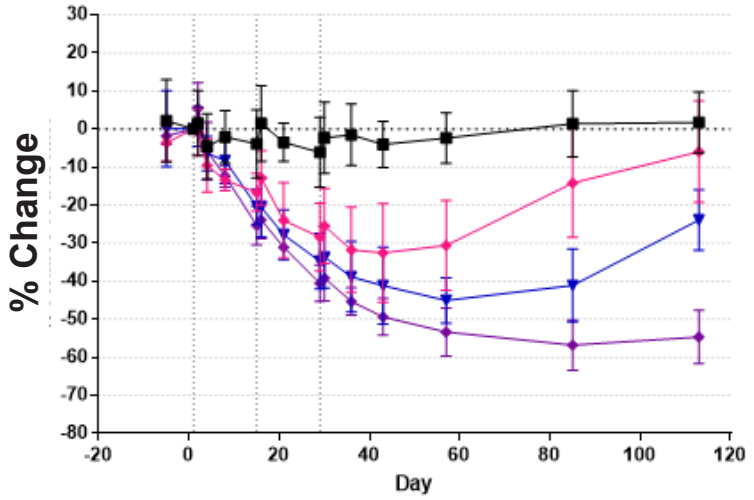
- SAD (up to 1350mg) / MAD (up to 450mg)
- MAD: IV q2w x3 doses

- Well-tolerated with no SAEs or treatment discontinuations
- PK T ½ ~33 days

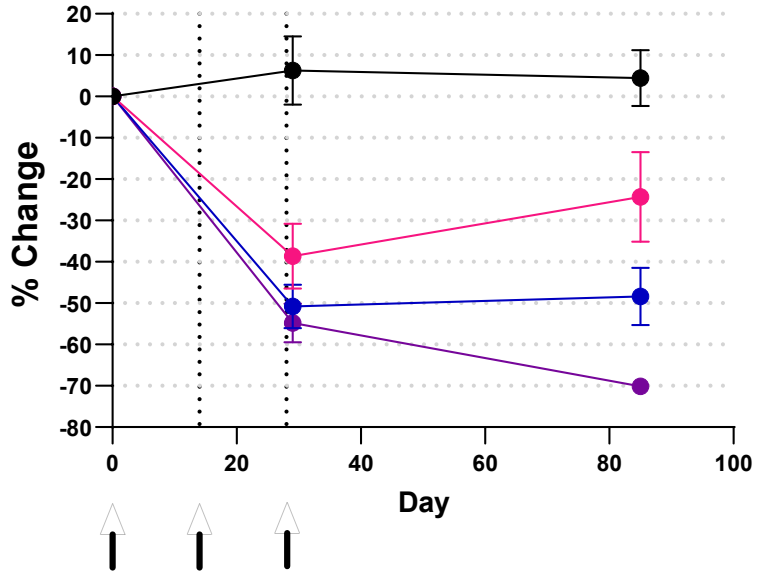
Free APRIL



IgA



Gd-IgA1



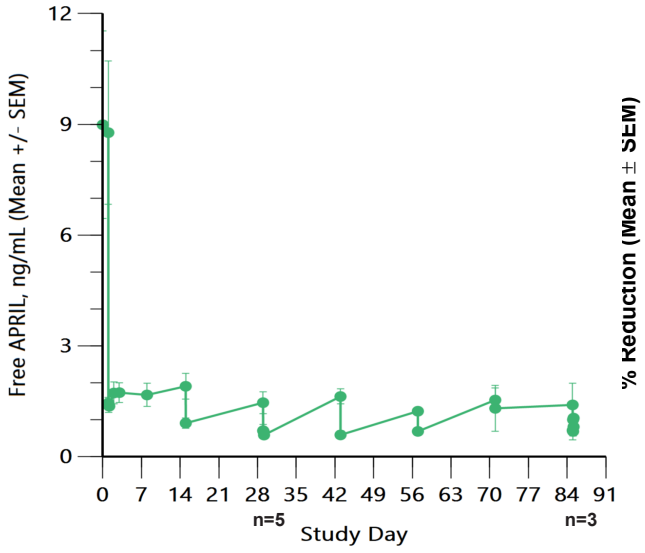
- Reductions in IgA ~ IgM > IgG (20-25% in MAD 450mg)
- Provides PD window to deplete pathogenic Gd-IgA1, with lesser effects on IgG
- Phase 1 SC study in HVs complete – observed APRIL and IgA reductions support potential for SC dosing

Lo J, et al. ERA-EDTA 2020 poster presentation: poster #P0500.

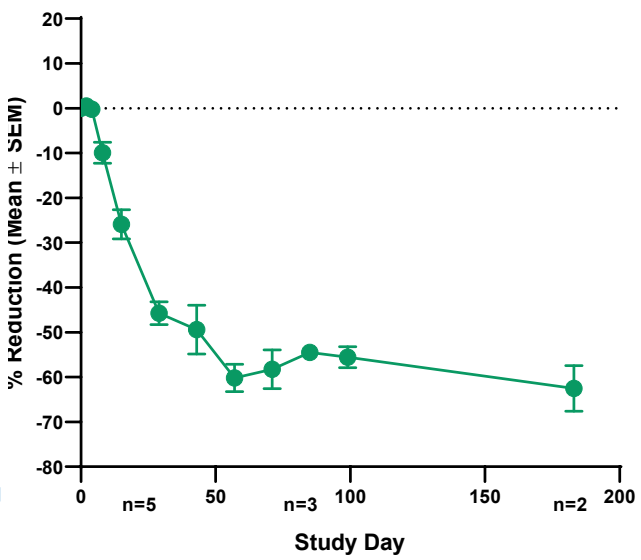
Initial BION-1301 Responses in IgAN Patients

- Cohort 1 in IgAN patients: 450mg IV q2w

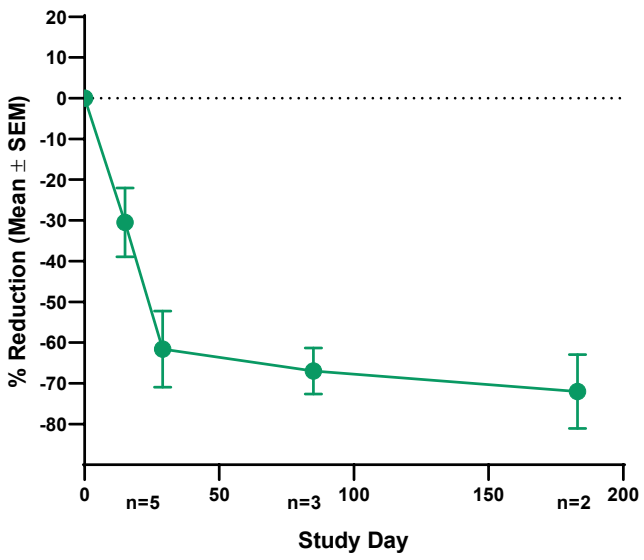
Free APRIL



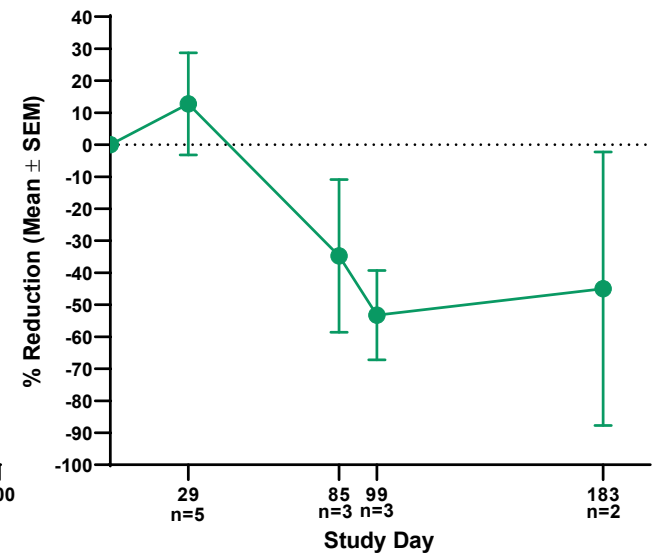
IgA



Gd-IgA1



Proteinuria



- In patients with IgAN treated to date, BION-1301 has been well-tolerated and resulted in:
  - Rapid APRIL neutralization
  - IgA and Gd-IgA1 depletion
  - Proteinuria (24 hr UPCR) reduction

Mean only shown for clarity

BION-1301 Phase 1/2 Trial Design & Enrollment Update

- ✓ **Established safety, PK, immunogenicity & biomarker effects in HVs and IgAN patients**
- ✓ **Established proof of mechanism: free APRIL, IgA & Gd-IgA1**
- ✓ **Established preliminary proof of concept: clinically meaningful proteinuria reduction**
- ✓ **Exploring IV & SC dose/schedule in patients necessary to achieve reductions in IgA, Gd-IgA1 and proteinuria**
- ✓ **SC dosing in Cohort 2 will begin in Q3**

Part 1 SAD in Healthy Volunteers	
10 – 1350 mg IV	✓
Part 2 MAD in Healthy Volunteers	
50 – 450 mg IV q2w	✓
Part 3 in IgAN Patients	
Cohort 1: 450 mg IV q2w for 52 weeks	**Expect to complete enrollment in Q3 2021**
Cohort 2: 600 mg SC q2w for 52 weeks	**Expect to initiate enrollment in Q3 2021**
Potential Cohort 3: SC Dose/Schedule TBD for 52 weeks	**Dose/schedule to be driven by Cohort 2 data**

- Improving enrollment dynamics due to:
  - ✓ Streamlined protocol: simplified operational complexity by combining Phase 1 with Phase 2 OLE for total treatment duration of 1 year
  - ✓ Opened new clinical trial sites with IgAN patient treatment expertise
  - ✓ Increasing awareness of BION-1301 proof of mechanism and proof of concept data with PIs
  - ✓ Improved COVID-19 situation in many geographies

BION-1301 Clinical Development Strategy

Data-driven decision based on magnitude and consistency of treatment effect on biomarkers and UPCR

If Dose Range is Well-defined from Phase 1/2

Phase 2/3

Dose 1
Dose 2
Placebo

Dose X
Placebo

Formal Interim Analysis

< OR >

If Additional Dose-finding is Required

Phase 2

Dose 1
Dose 2
Dose 3
Placebo

Phase 3

Dose X
Placebo

Formal Analysis

Phase 2: Assess multiple doses for proteinuria effect

Advance optimal dose to Phase 3

Phase 3: assessment of proteinuria/eGFR

CHINOOK THERAPEUTICS

CHK-336

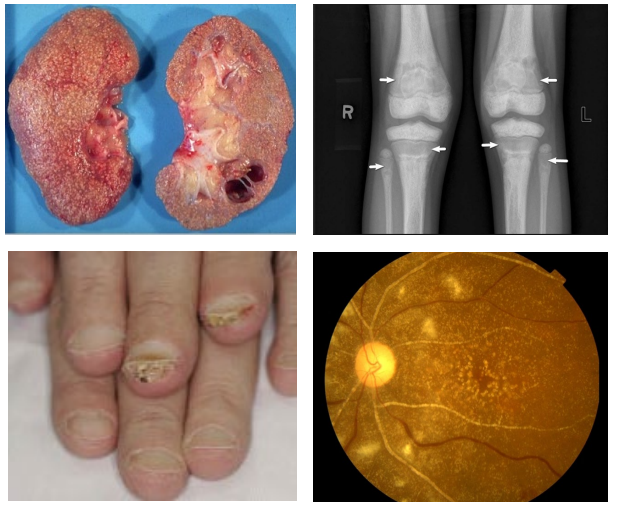
Potent and Selective Small Molecule LDHA Inhibitor

Hyperoxalurias are Diseases Caused by Excess Oxalate

Hyperoxaluria is an important risk factor for kidney stones

Primary hyperoxalurias (PH) 1-3 are ultra-rare diseases

- Caused by genetic mutations resulting in hepatic overproduction of oxalate
- PH leads to recurrent kidney stones and can lead to kidney failure, if left untreated
- Median age of kidney failure for PH1 is 23 years
- ~5,000 – 7,000 PH1 patients in the US and Europe



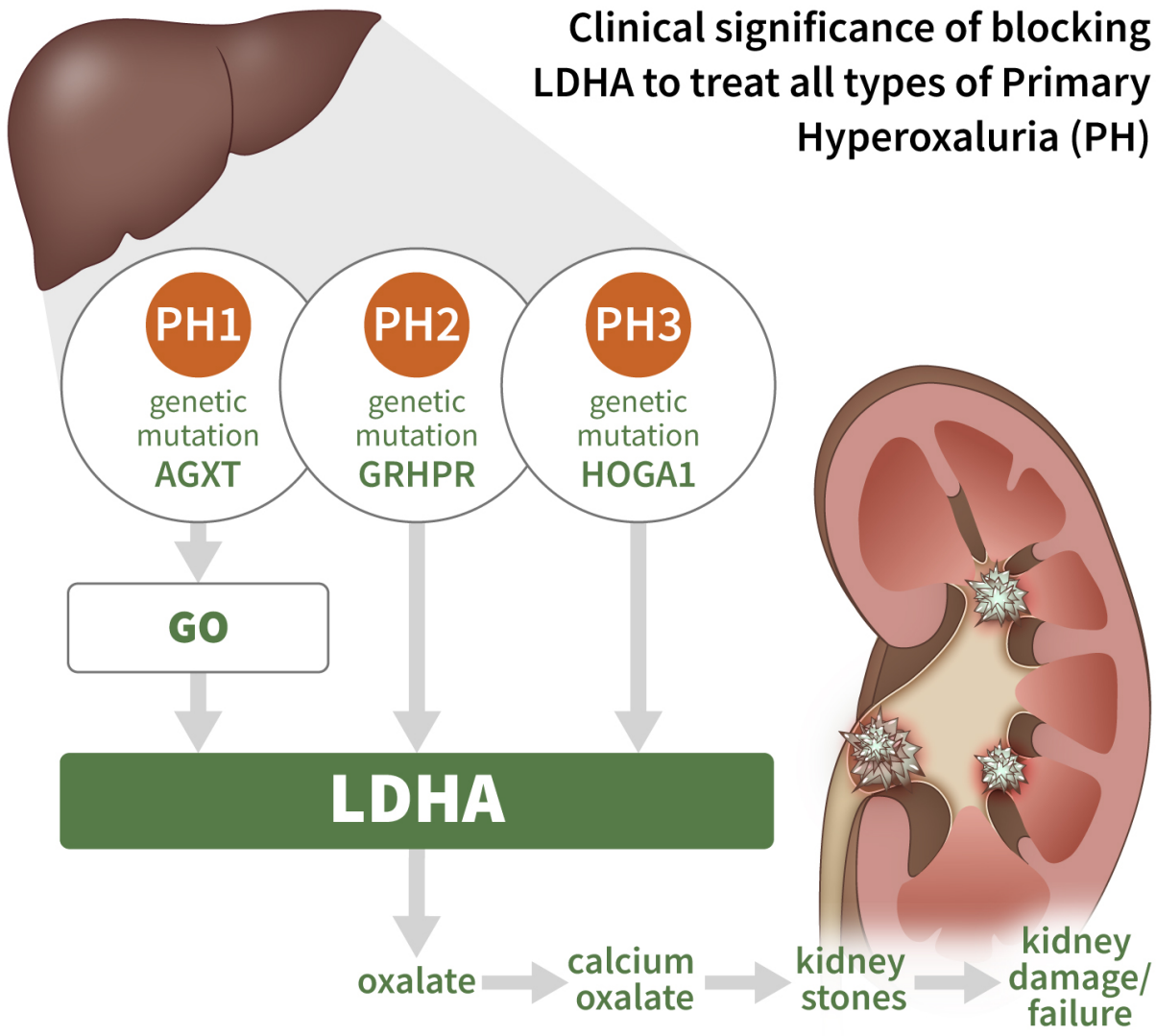
Decline in kidney function results in systemic oxalosis, affecting multiple organs

Secondary hyperoxalurias are more common

- Acquired condition resulting from increase in: dietary oxalate intake, intestinal oxalate absorption or endogenous oxalate overproduction
- Hyperoxaluria, usually defined as urinary excretion of >40 mg/d, is present in ~20 – 40% of stone formers

Zhao et al., CJASN 2016, 11, 119–126; Hoppe et al., Kidney Int. 2009, 75(12), 1264-1271; Goldfarb et al., CJASN July 2007, 2(4) 745-749.
Chu, HK J Paediatr (New Series) 2011, 16:41-46; Gregoriou et al., Acta Derm Venereol. 2011, 91(2):195-196; ASRS Retina Image Bank.

Targeting LDHA Addresses All Types of PH



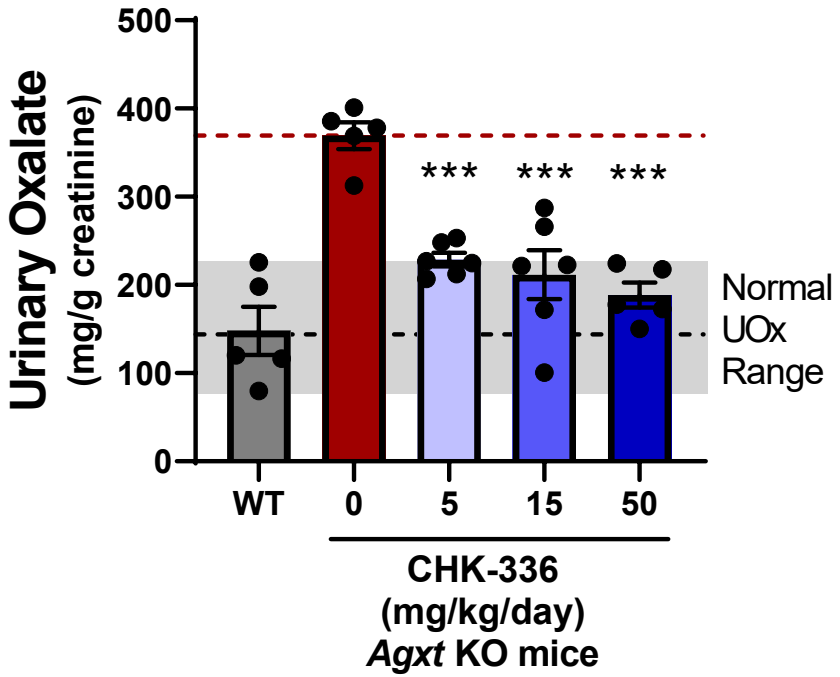
Lactate dehydrogenase (LDHA) is the final step in production of oxalate from glyoxylate (GO) in the liver

- Potential therapeutic target for all forms of PH and other disorders of excess oxalate
- Liver-targeting profile is desired to maximize target engagement and minimize systemic exposure
- CHK-336 is an oral small molecule LDHA inhibitor that has potential to be safe and well-tolerated

Kanno et al Clinica Chimica Acta 1980; 108: 267-276

CHK-336: Oral Small Molecule LDHA Inhibitor for PH

Liver-targeted tissue distribution profile enables potential to treat all PH types



- CHK-336 produces significant and dose-dependent urinary oxalate reductions in PH1 mouse models
- Titration and customized dosing is possible for better individual efficacy through more complete target inhibition
- Oral administration more convenient and desirable for patients; enables expansion into less severe, but much more common forms of hyperoxaluria
- CHK-336 currently progressing through IND-enabling studies, with phase 1 initiation in HVs planned for Q1 2022

Cox et al., ASN 2020, Abstract: PO1620.

CHINOOK THERAPEUTICS

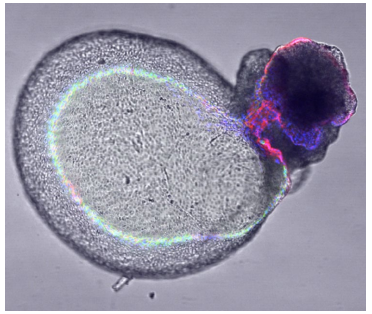
Research & Discovery

Precision Medicines for Kidney Diseases

Precision Medicine Approach to Research & Discovery

Focused on indications with defined causal molecular drivers & efficient development paths

- Leveraging deep insights in kidney disease biology



- Advanced translational models: pluripotent stem cell-derived kidney organoids & patient-derived 3-D cellular systems
- Established collaborations with academic experts using scRNAseq techniques to gain high-resolution molecular insights into kidney disease mechanisms

Washington University in St. Louis · Mayo Clinic · University of Washington · KU School of Medicine · Morehouse School of Medicine · Yale University

- Strategic Collaboration with Evotec

evotec

  - Define CKD in molecular terms, identify novel targets and selectively target specific CKD patient sub-populations
  - Accelerates precision medicine platform to identify, characterize and validate novel mechanisms and discover precision medicines for PKD, lupus nephritis, IgA nephropathy and other primary glomerular diseases
  - Leverages access to NURTuRE CKD Patient Biobank, which provides comprehensive PANOMICS characterization in thousands of CKD patients with prospective clinical follow-up and retained bio-samples of urine and blood for exploratory biomarker analysis, to enable patient stratification strategies

CHINOOK THERAPEUTICS

Financials & Catalysts

Financial Strength

NASDAQ: **KDNY**

Strong Balance Sheet

- $229.8 M in cash, cash equivalents and marketable securities as of June 30, 2021

Cash Guidance

- Operating capital through H1 2023 based on current business plan

Common Stock Outstanding

- 44.8 million shares as of August 10, 2021
- ~45.7 million fully diluted shares as of August 11, 2021*

* Treasury method. Includes 6.4 million options with average exercise price of $13.48 and 0.88 million RSUs outstanding.

Catalysts

Program	Indication	Catalyst	H1 2021	H2 2021	H1 2022
Atrasentan	IgA Nephropathy	Initiate phase 3 ALIGN study	✓		
	Basket of Glomerular Diseases	Initiate phase 2 AFFINITY study	✓		
		Present interim data from initial AFFINITY patient cohort(s)			●
BION-1301	IgA Nephropathy	Present additional biomarker data and IV-to-SC bioavailability data in healthy volunteers	✓		
		Present phase 1 data in IgAN patients	✓	●	●
		Analyze phase 1 data and announce update on later-stage clinical development strategy			●
CHK-336	Primary Hyperoxaluria	Complete IND-enabling studies and initiate phase 1 study in healthy volunteers		●	●

CHINOOK
THERAPEUTICS