CHINOOK THERAPEUTICS

Chinook Therapeutics

Developing Precision Medicines for Kidney Diseases

February 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.

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)
- Healthy volunteer data demonstrates dose-dependent and durable reductions in free APRIL and IgA levels
- Phase 1b proteinuria data in IgAN expected in 2021

CHK-336

- Oral small molecule LDHA inhibitor with liver-targeted tissue distribution for primary hyperoxaluria
- Potential to treat all disorders of excess oxalate
- Phase 1 initiation planned for H2 2021

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

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

Opportunity is Now

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.

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 initiation expected in early 2021					ALIGN
	Basket of glomerular diseases	Phase 2 initiation expected in H1 2021					AFFINITY
BION-1301	IgA Nephropathy	Phase 1b ongoing					
CHK-336	Primary Hyperoxaluria	Ph1 initiation expected in H2 2021					
Research Programs	Rare, severe chronic kidney diseases including ADPKD						
Discovery Programs	Rare, severe chronic kidney diseases						

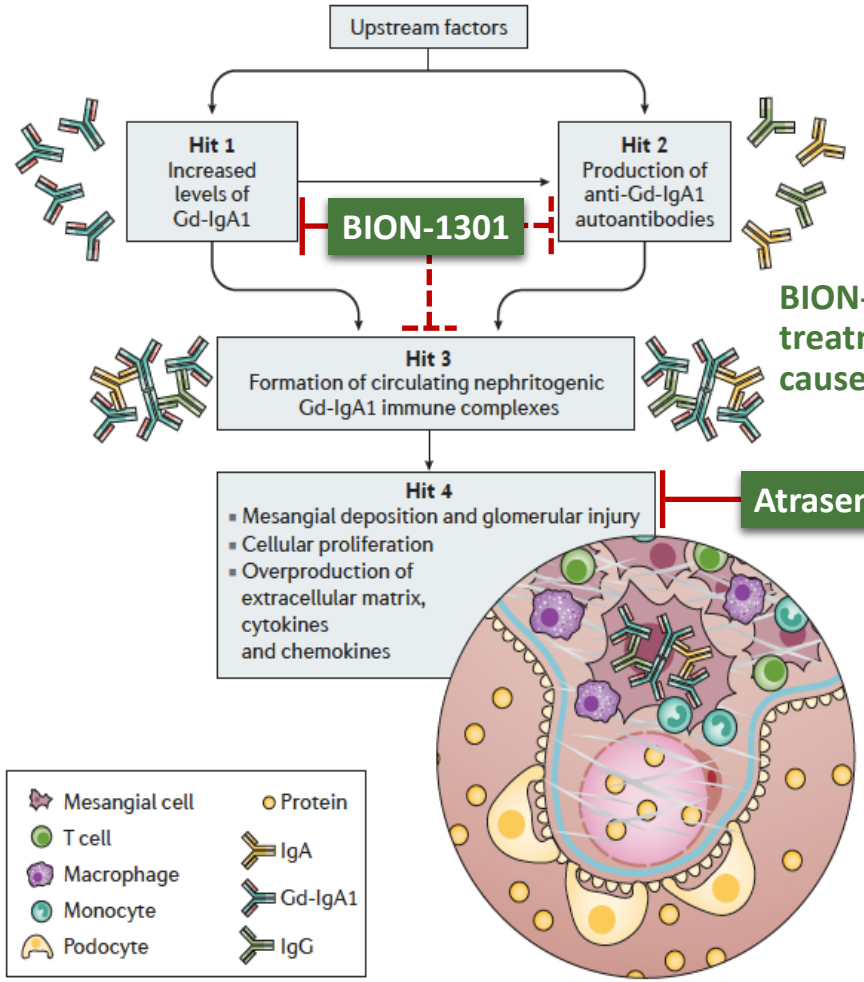
Global commercial rights to all pipeline programs

Continue to evaluate opportunities to add kidney disease programs to pipeline

Why Target IgA Nephropathy?

- Most common primary glomerular disease globally with ~140K 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).

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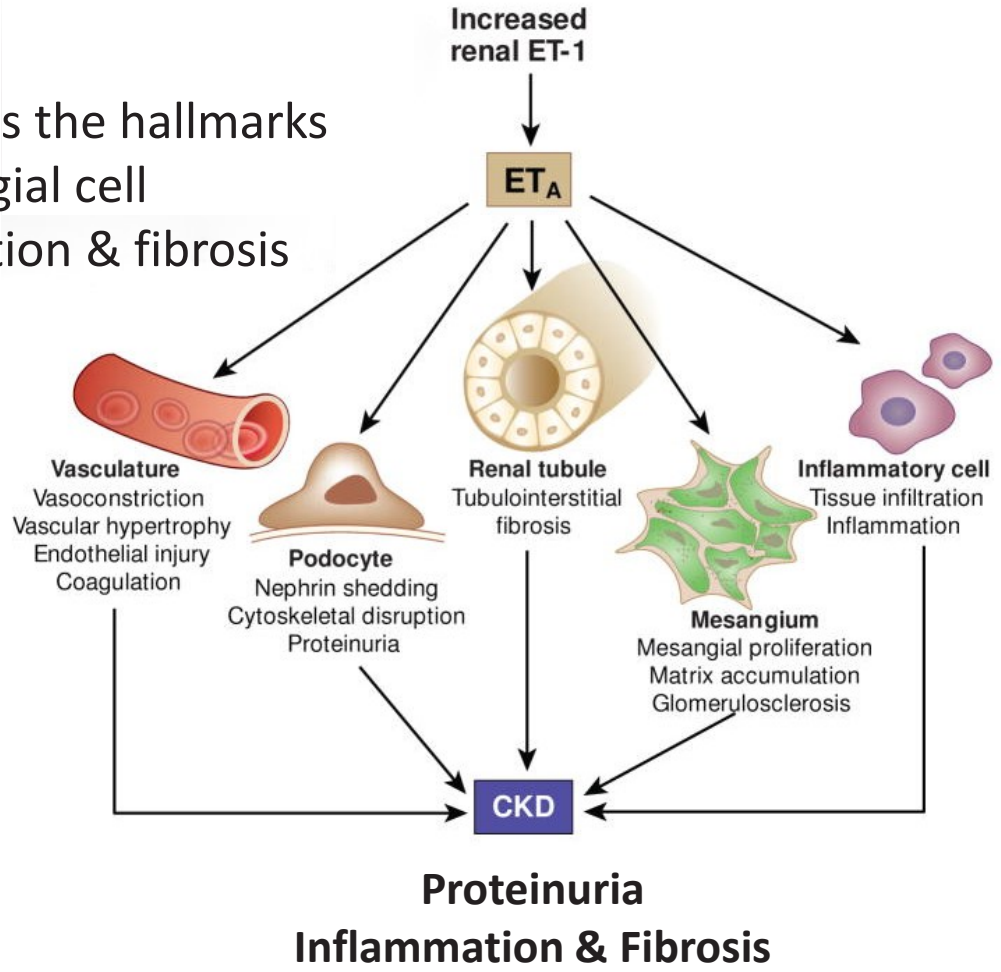
Atrasentan

Potent and Selective Endothelin A Receptor Antagonist

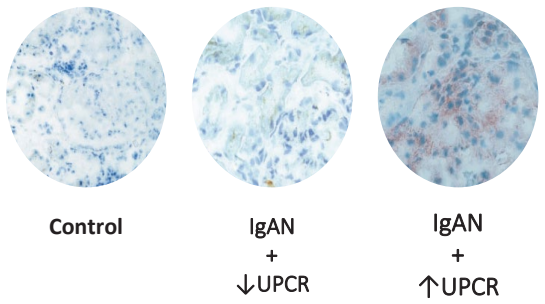
Atrasentan: a Potent and Selective ET_A Antagonist

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



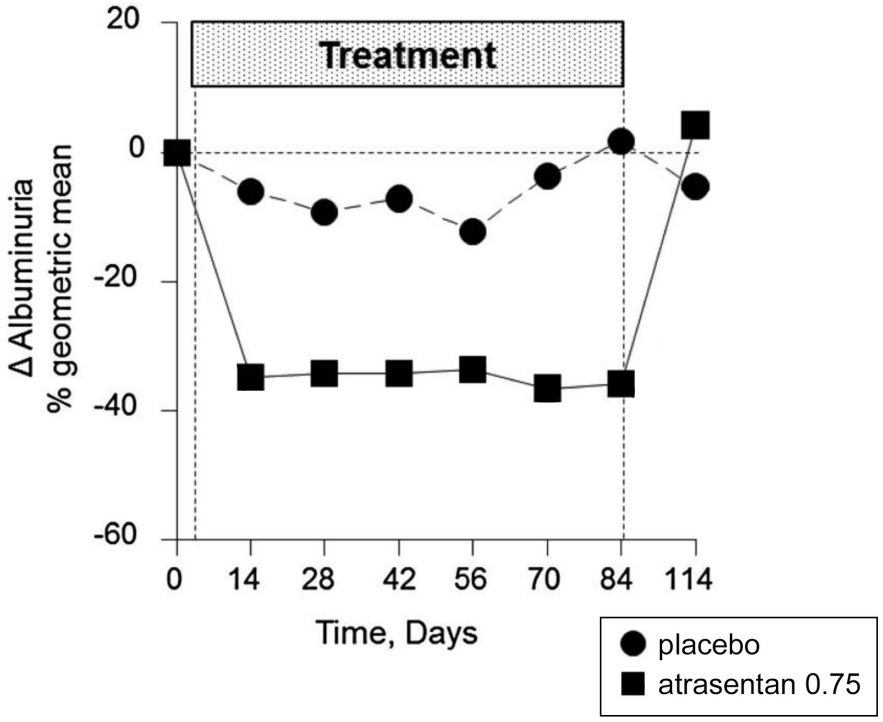
- ET system activation appears to be a key molecular determinant of progressive IgAN
- Elevated kidney ET-1 expression strongly predicts progression of IgAN
- **ET_A receptor blockade through atrasentan is a promising approach to treat IgAN patients**

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.

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

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)

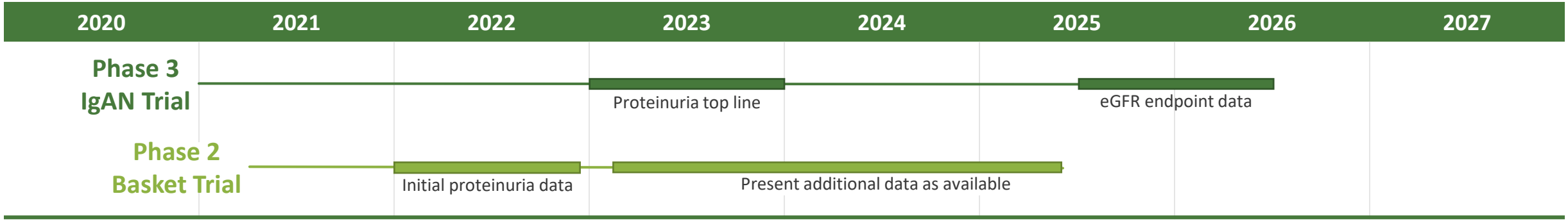
Phase 2 Basket Trial to Expand Potential Across Multiple Indications

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



ALIGN ClinicalTrials.gov Identifier: NCT04573478;
AFFINITY ClinicalTrials.gov Identifier: NCT04573920

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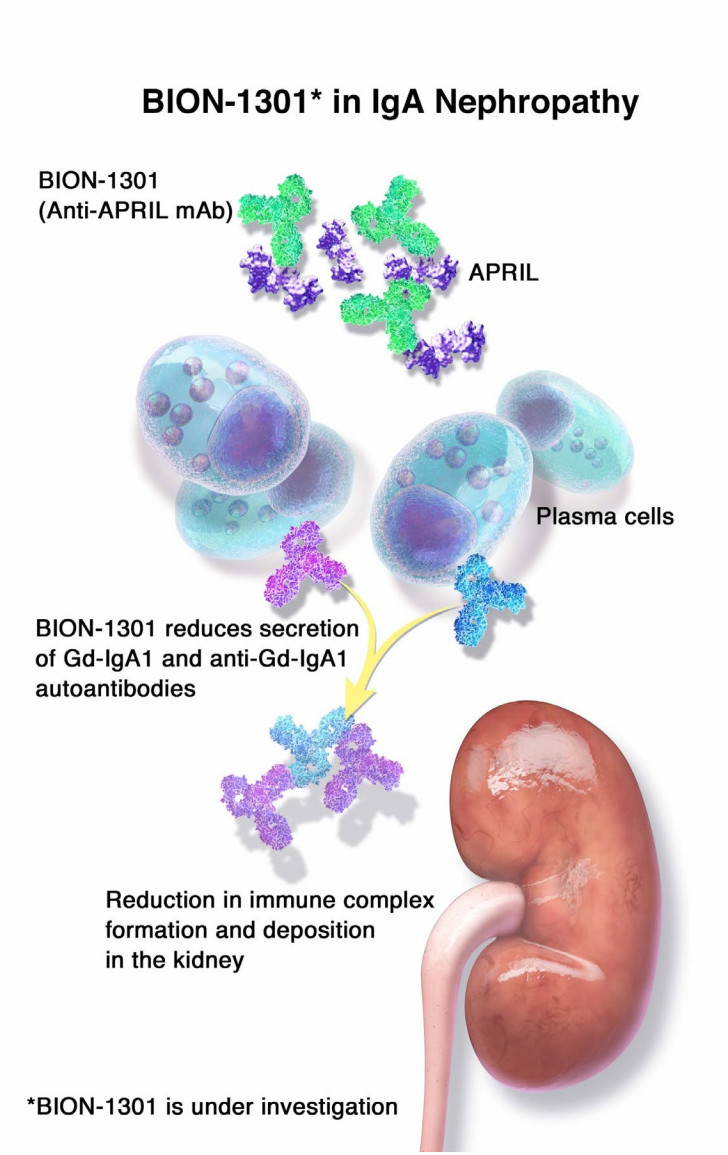
BION-1301

Anti-APRIL 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]

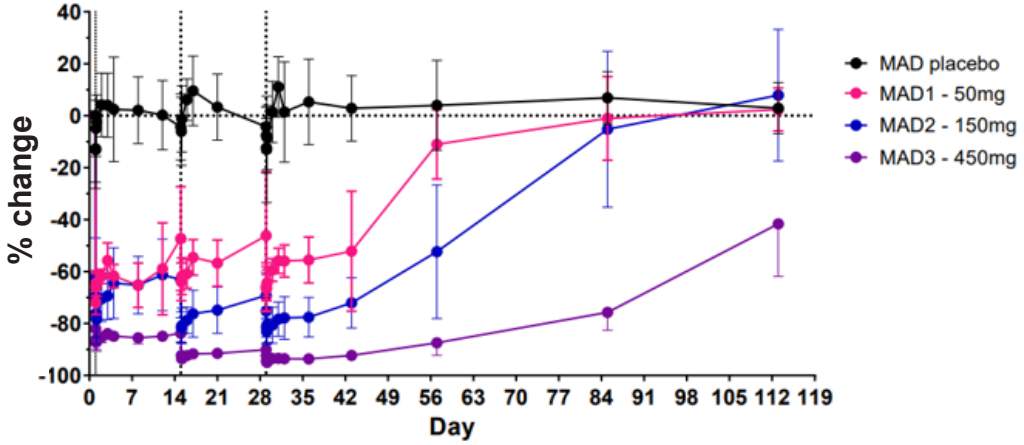
BION-1301: humanized IgG4 monoclonal antibody that blocks APRIL binding to its receptors

- 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.

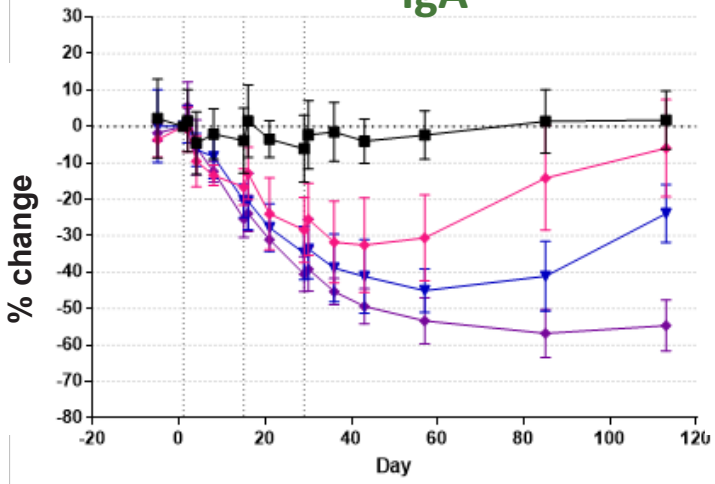
BION-1301 Demonstrated ~50-60% IgA Reductions in HVs

Free APRIL

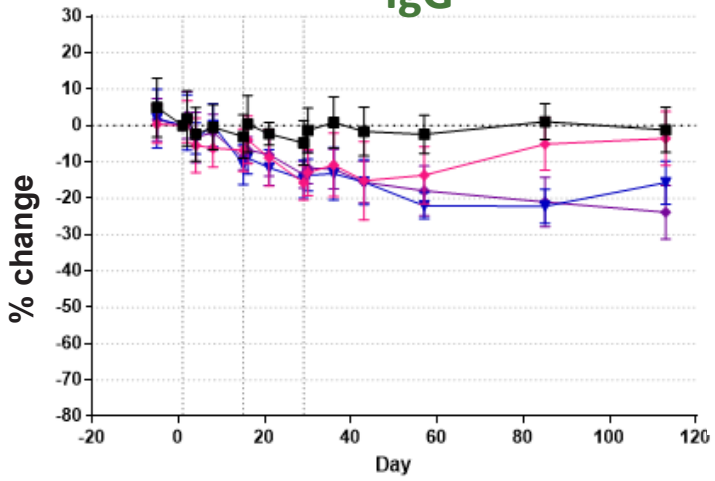


- BION-1301 well-tolerated with no SAEs
- Half-life of ~33 days supports potential for monthly dosing
- Dose-dependent and durable reductions in free APRIL, IgA, IgM and to a lesser extent, IgG
- Target of **~50-60% IgA reduction** achieved with 150-450 mg IV q2w

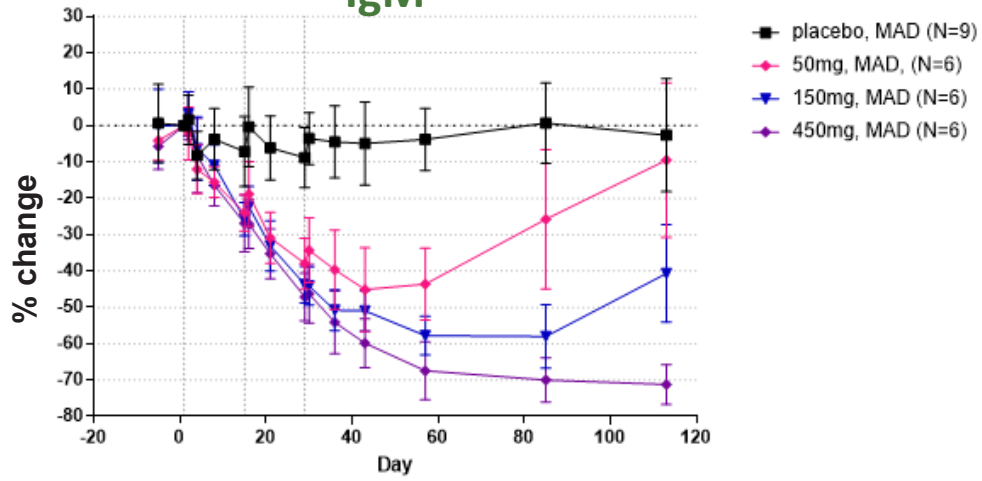
IgA



IgG



IgM



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

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BION-1301 Phase 1b Currently Enrolling IgAN Patients

Part 1 in Healthy Volunteers Completed Double-blind, Placebo-controlled, **Single Ascending Dose**				
10mg	50mg	150mg	450mg	1350mg

Part 2 in Healthy Volunteers Completed Double-blind, Placebo-controlled, **Multiple Ascending Dose** (IV q2w)		
50mg	150mg	450mg

Part 3 in IgAN Patients Ongoing Open Label, Multiple Dose	
450mg	Dose TBD

Part 3 Assessing Safety, PK/PD & Activity

- Two cohorts of ten patients each with biopsy-proven IgAN
- All patients on maximally-tolerated, optimized and stable dose of RASi or RASi intolerant
- Proteinuria ≥0.5 g/day
- eGFR >45 mL/min or eGFR 30 - 45 mL/min if kidney biopsy performed within prior 2 years with no evidence of fibrosis
- IV infusion every 2 - 4 weeks for 12 weeks
- Patients completing Part 3 may be eligible for long-term extension trial for additional 2 years
- Phase 1 IV to SC bioavailability study ongoing with transition to SC administration planned
- Multiple data presentations in 2021

ClinicalTrials.gov Identifier: NCT03945318

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CHK-336

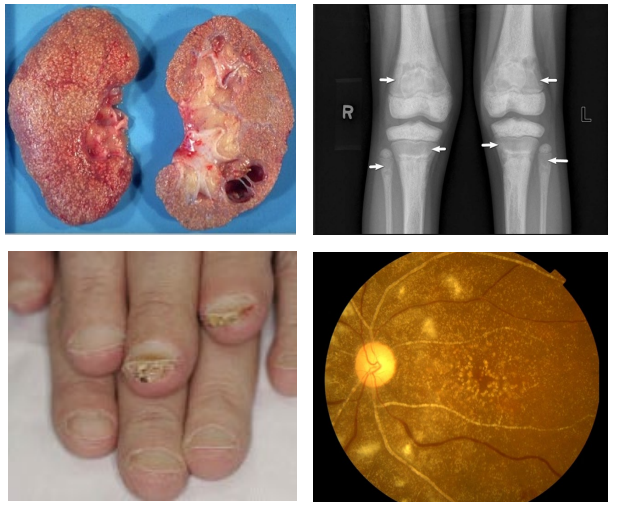
Potent and Selective Small Molecule LDHA Inhibitor

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



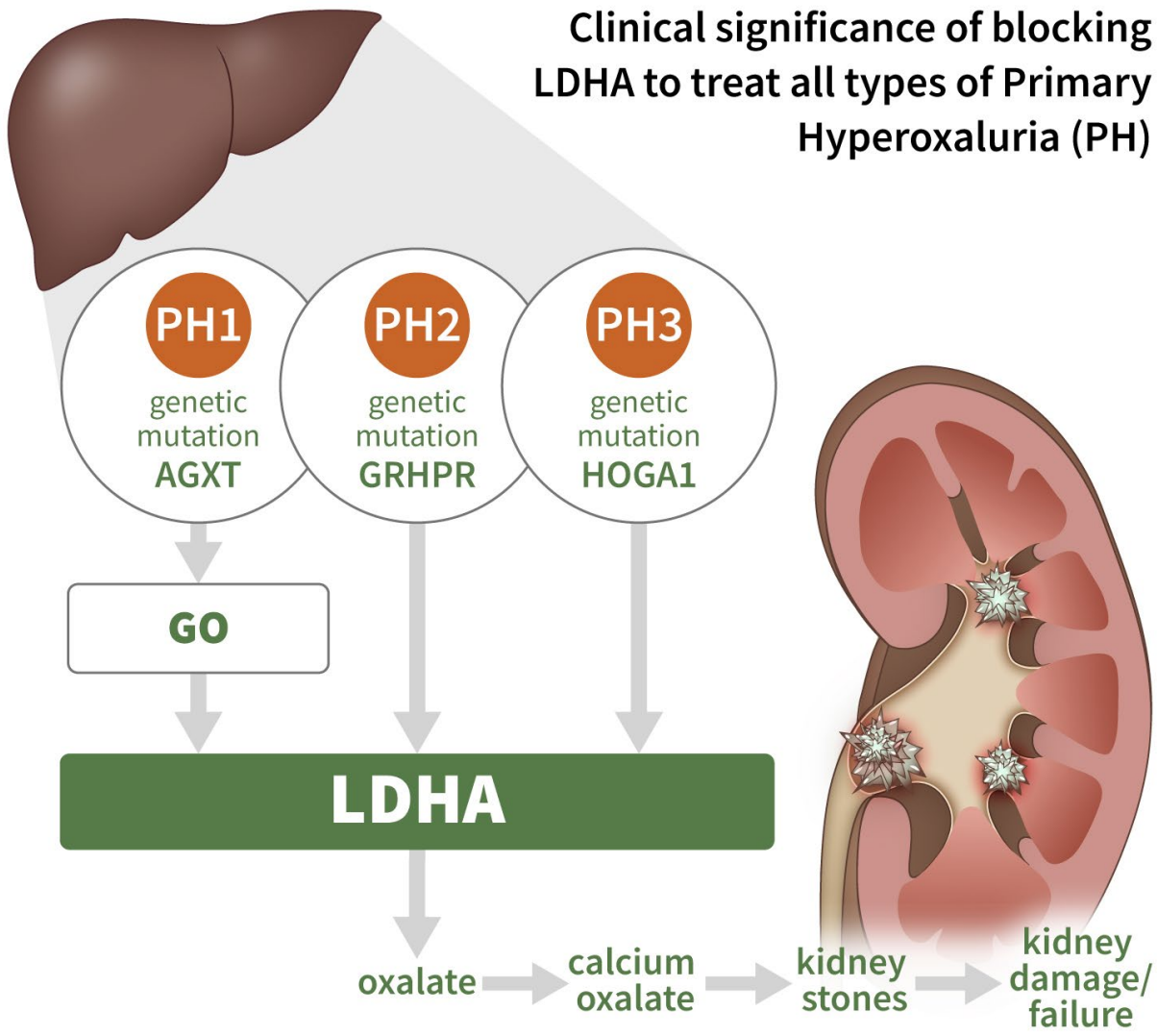
Decline in kidney function results in systemic oxalosis, affecting multiple organs

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



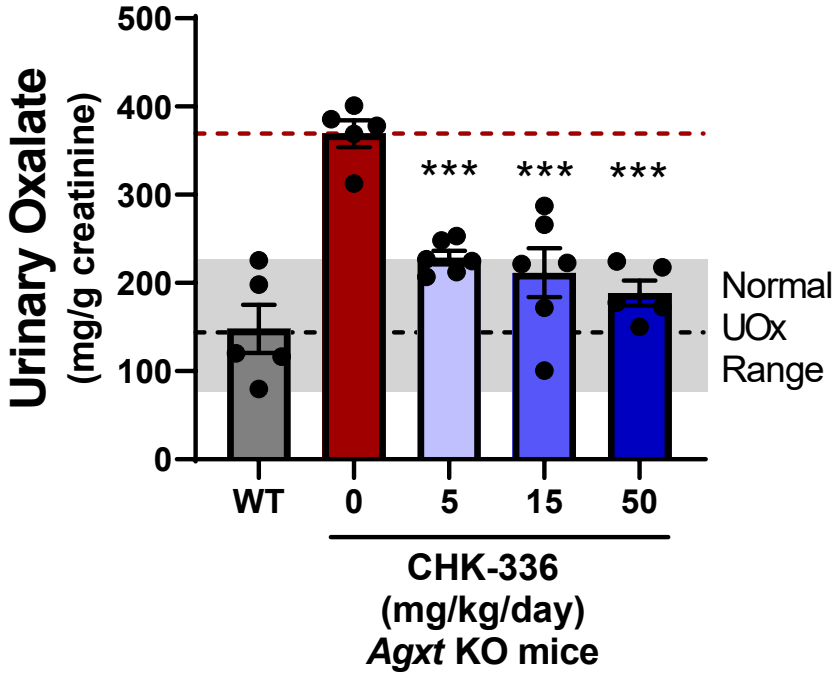
Lactate dehydrogenase (LDHA) is the final step in production of oxalate from glyoxylate (GO) in the liver

- 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 is expected 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
- Exploratory toxicity studies in rodents demonstrate wide safety margins
- 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 planned for H2 2021

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

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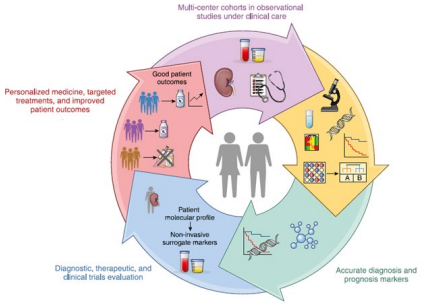
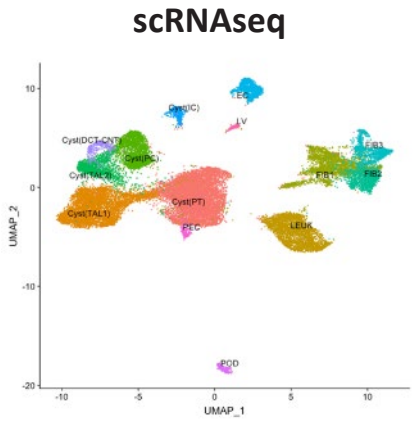
Research & Discovery

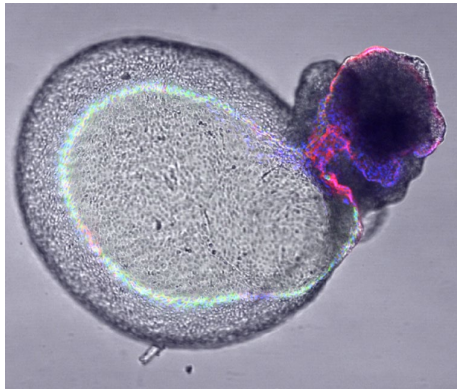
Precision Medicines for Kidney Diseases

Precision Medicine Approach to Research & Discovery

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







Leveraging deep insights in kidney disease biology & novel translational models

- Established collaborations with academic experts using scRNAseq techniques to gain high-resolution molecular insights into kidney disease mechanisms
- Advanced translational models from pluripotent stem cell-derived kidney organoids along with patient-derived 3-D cellular systems
- Emerging patient stratification approaches
- Targeting genetic or molecular drivers to prevent ESKD
- Active research programs in ADPKD and other severe chronic kidney diseases

Washington University in St. Louis · Yale University · MAYO CLINIC · KU School of Medicine · Morehouse School of Medicine · University of Washington

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Financials & Catalysts

Financial Strength

NASDAQ: **KDNY**

Strong Balance Sheet

- ~$290M in cash, cash equivalents and marketable securities*

Cash Guidance

- Operating capital through H1 2023

Common Stock Outstanding

- 42.2 million shares as of November 2, 2020
- 45.9 million fully diluted shares as of December 18, 2020**

* As of October 5, 2020, includes proceeds from private placement financing. Before payments of transactions costs related to merger with Aduro.

** Treasury method. Includes 4.6 million in-the-money options with average exercise price of $7.48 and 0.9 million RSUs outstanding.

Planned Upcoming Catalysts

Program	Indication	Catalyst	H1 2021	H2 2021
Atrasentan	IgA Nephropathy	Initiate phase 3 ALIGN study	●	
	Basket of Glomerular Diseases	Initiate phase 2 AFFINITY study	●	
BION-1301	IgA Nephropathy	Present IV to SC bioavailability data	●	
		Present data on Gd-IgA levels in healthy volunteers	●	
		Present interim phase 1 data in IgAN patients	●	●
CHK-336	Primary Hyperoxaluria	Initiate phase 1 study		●

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