CHINOOK THERAPEUTICS

Chinook Therapeutics

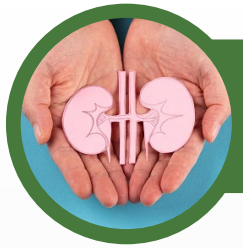
Developing Precision Medicines for Kidney Diseases

August 2022

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 Needs

Up to 10%	Percentage of global population suffering from kidney disease[1]
>$130B	Annual U.S. healthcare costs driven by kidney diseases[2]
Few drugs	Limited treatment options to prevent kidney disease progression

Clear Development Paths

- Increased understanding of underlying disease biology
- New and more 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. United States Renal Data System. 2021 USRDS Annual Data Report: Epidemiology of kidney disease in the United States. National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, Bethesda, MD, 2021.; 3. Thompson et al., CJASN March 2019, 14 (3) 469-481.

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Dedicated to Kidney Disease Drug Development

Atrasentan

- Highly potent, selective ET_A inhibitor
- Phase 2 AFFINITY IgAN cohort demonstrated >50% reductions in proteinuria
- Phase 3 ALIGN proteinuria data expected in 2023

BION-1301

- Anti-APRIL monoclonal antibody (mAb)
- Strong, durable impact on mechanistic biomarkers and 50-70% proteinuria reductions in patients with IgAN
- Additional phase 1/2 data and pivotal trial update in H2 2022

CHK-336

- Oral small molecule LDHA inhibitor with liver-targeted tissue distribution for primary hyperoxaluria
- Potential to treat all excess endogenous oxalate disorders
- Phase 1 in healthy volunteers ongoing

Precision Medicine R&D Pipeline

- Focused on rare, severe chronic kidney diseases
- Designing novel, targeted and differentiated molecules
- Plan to execute clinical trials in defined patient populations with surrogate endpoints

Strong cash position with operating capital into 2025

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Advancing a Diversified Pipeline of Best-in-Class Programs

Program	Indication	Target Validation	Lead Optimization	IND-Enabling	Phase 1	Phase 2	Phase 3
Atrasentan (ALIGN)	IgA Nephropathy						Phase 3 ongoing
Atrasentan (AFFINITY)	Basket of glomerular diseases					Phase 2 ongoing	
BION-1301	IgA Nephropathy					Phase 1/2 ongoing	
CHK-336	Primary Hyperoxaluria				Phase 1 HV study ongoing		
Research & Discovery Programs	Rare, severe chronic kidney diseases	Multiple programs at different stages					

Continuing to evaluate opportunities to add kidney disease programs to pipeline

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IgA Nephropathy Has Large Unmet Medical Need

IgAN is the most common primary glomerular disease globally, with the following diagnosed prevalence:

- US: ~150,000
- EU: ~200,000
- Japan: ~180,000
- China: ~800,000 due to low diagnosis rate; potentially 3x higher

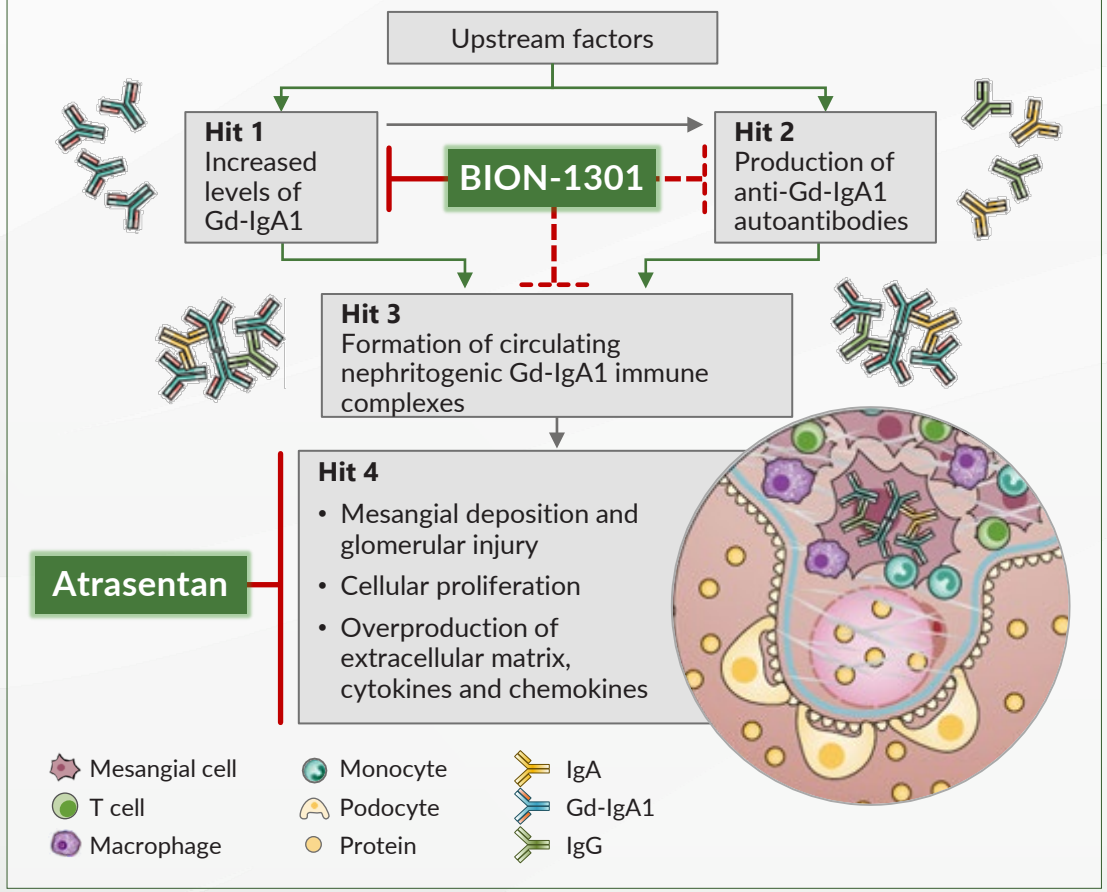
Current IgAN treatment paradigm:

- RAS inhibition (ACEi or ARB) is frontline SOC
- Steroids may potentially be considered in high-risk patients though toxicity risk must be carefully evaluated
- SGLT2i use increasing recently

- **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 eGFR**

>40% of biopsy-confirmed IgAN patients have uncontrolled proteinuria and remain at risk for progression despite being on RASi

IgA Nephropathy Disease Pathophysiology



Thompson et al., CJASN March 2019, 14 (3) 469-481; KDIGO 2021 Clinical Practice Guideline for the Management of Glomerular Diseases, Kidney International (2021) 100, S1–S276; Spherix Global Insights, RealWorld Dynamix, IgA Nephropathy 2021, 2022; Lai, K., Tang, S., Schena, F. et al. IgA nephropathy. Nat Rev Dis Primers 2, 16001 (2016).

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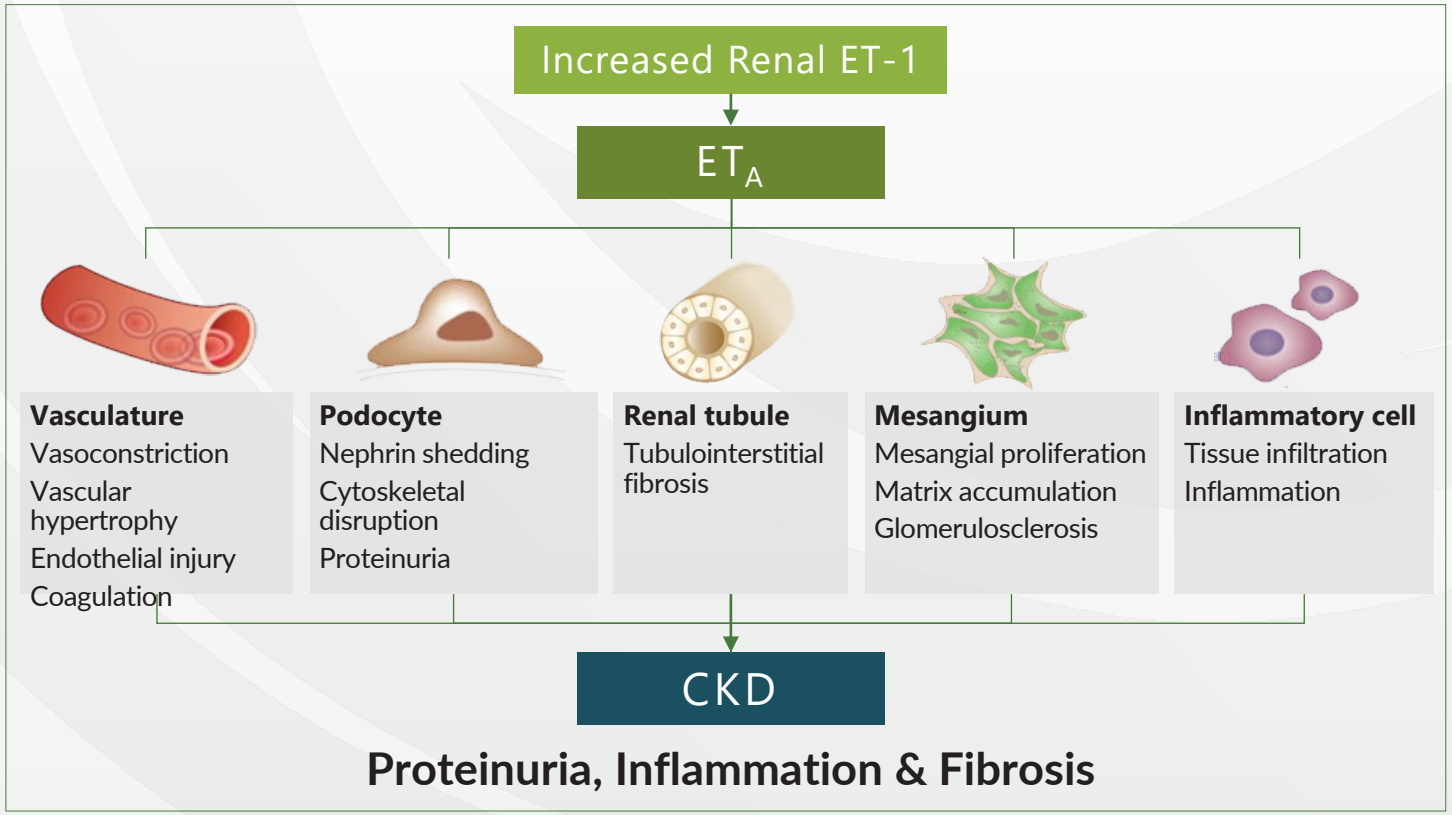
Atrasentan

Potent and Selective Endothelin A Receptor (ET_A) Antagonist

Atrasentan: a Potent and Selective ET_A Antagonist

ET_A receptor activation drives IgAN progression through multiple potential mechanisms

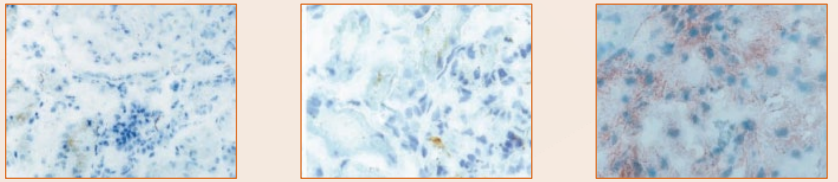
ET_A receptor activation drives the hallmarks of IgAN: proteinuria, mesangial cell activation, kidney inflammation & fibrosis



- 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 by atrasentan is a promising approach to treat IgAN patients***

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

ET-1



Control | IgAN + ↓UPCR | IgAN + ↑UPCR

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.

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Atrasentan Clinical and Regulatory Plan

Phase 3 Targeting IgAN patients at High Risk for Disease Progression

- 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 ~160 – 170 sites
- 6-month proteinuria primary endpoint (accelerated approval)
- 2.5 year eGFR secondary endpoint (full approval)

ALIGN

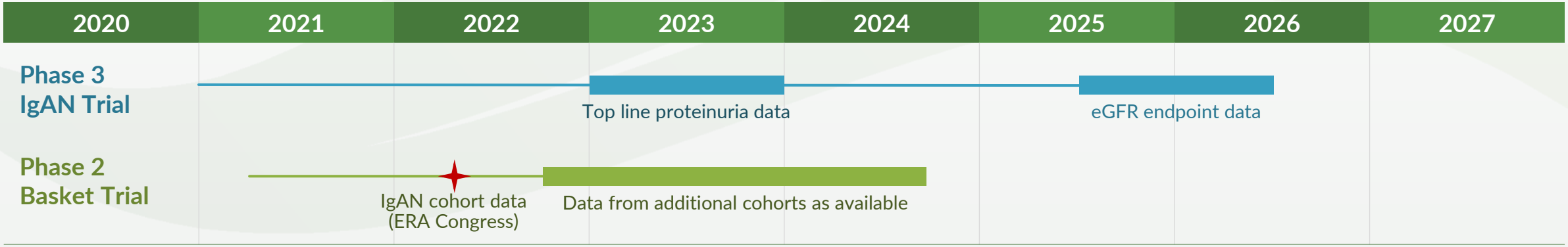
Phase 2 Basket Trial to Expand Potential Across Proteinuric Glomerular Diseases

- Open-label design
- 12-week proteinuria primary endpoint
- 52-week treatment period
- ~20 patients / cohort

Cohorts include:

- IgAN with proteinuria 0.5 – <1 g/g
- FSGS (dose escalation to 1.5 mg)
- Alport syndrome
- DKD combined with SGLT2 inhibitors

AFFINITY



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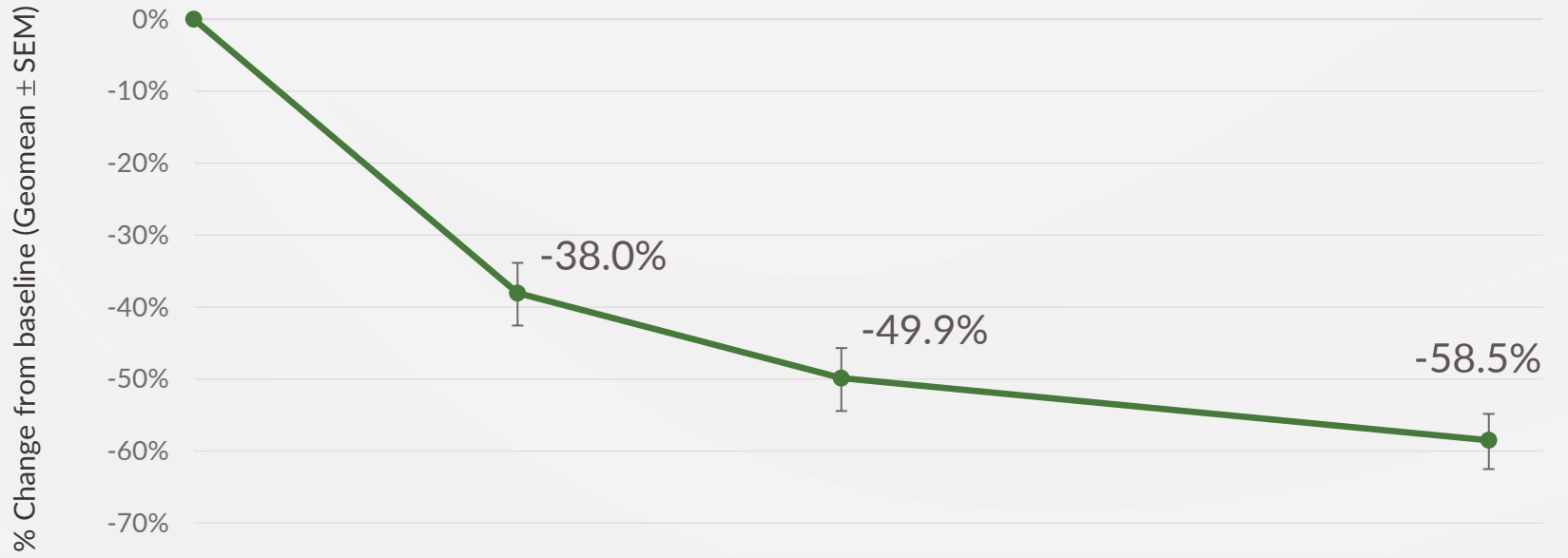
Atrasentan Provides >50% Proteinuria Reductions in Patients with IgAN Receiving Optimized SOC in AFFINITY Trial

% Reduction in UPCR*



Wk	0	6	12	24
n	20	20	18	11

- **Atrasentan was well-tolerated with no treatment-related SAEs**

Data cut-off: April 22, 2022

Median baseline 24-h urine protein excretion: 1.17 g/day (Q1,Q3: 0.85, 1.46 g/day)

* Results plotted are based on the least squares mean +/- SE of change from baseline on natural log scale from the MMRM back-transformed to a percent reduction from baseline scale

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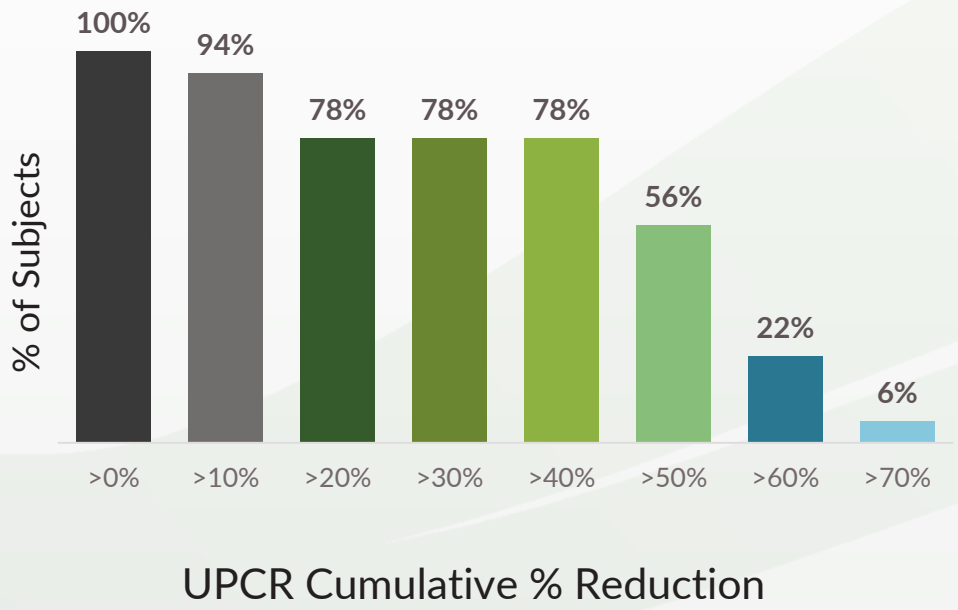
Atrasentan's Proteinuria Lowering Effects Are Highly Consistent Across Patients with IgAN Treated in AFFINITY Trial

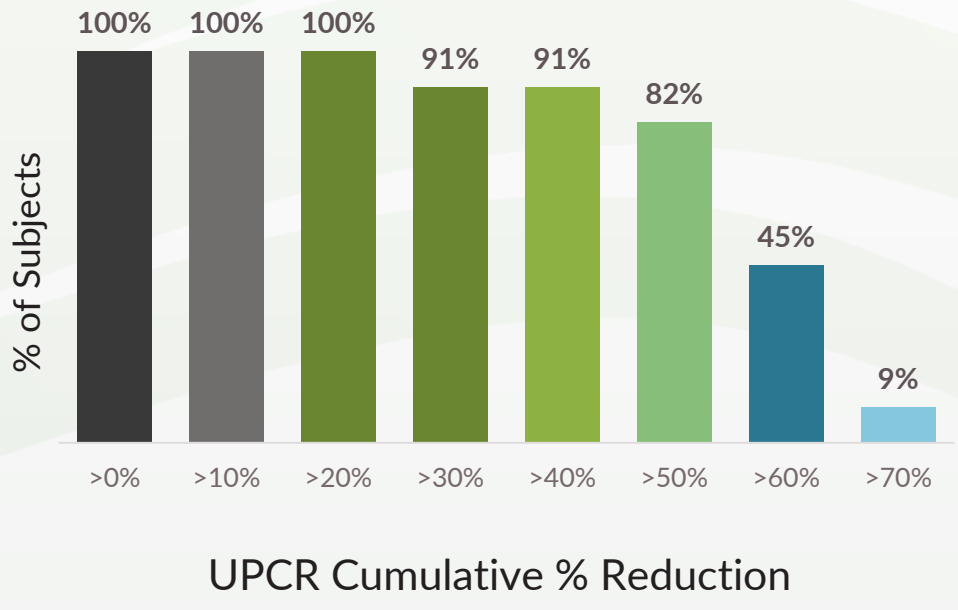
91% of patients achieved >40% reduction in proteinuria at Week 24

Week 12 (n=18)



UPCR Cumulative % Reduction

Week 24 (n=11)



UPCR Cumulative % Reduction

Atrasentan + SGLT2i Combination Strategy in IgAN

Establish atrasentan as combination of choice with SGLT2i by evaluating safety and efficacy

Two-pronged approach to evaluate atrasentan in combination with SGLT2i

1 ALIGN SGLT2i + Atrasentan Combination Stratum Underway

- Executed protocol amendment to enable enrollment of stratum of patients on stable dose of SGLT2i
- Allows for safety and efficacy exploratory analysis (UPCR at 24 weeks and eGFR at 136 weeks) with no change to enrollment timelines or primary analysis population

2 Planned Phase 2 Study of SGLT2i + Atrasentan Combination in IgAN

- Data will corroborate exploratory analysis from ALIGN SGLT2i + atrasentan combination strata
- Enroll IgAN patients at high risk for disease progression despite stable optimized RASi and stable SGLT2i
- Randomize patients 1:1 to placebo/atrasentan
- Primary endpoint: change in UPCR from baseline to week 12
- Goal is for data to support future use and is not required for approval

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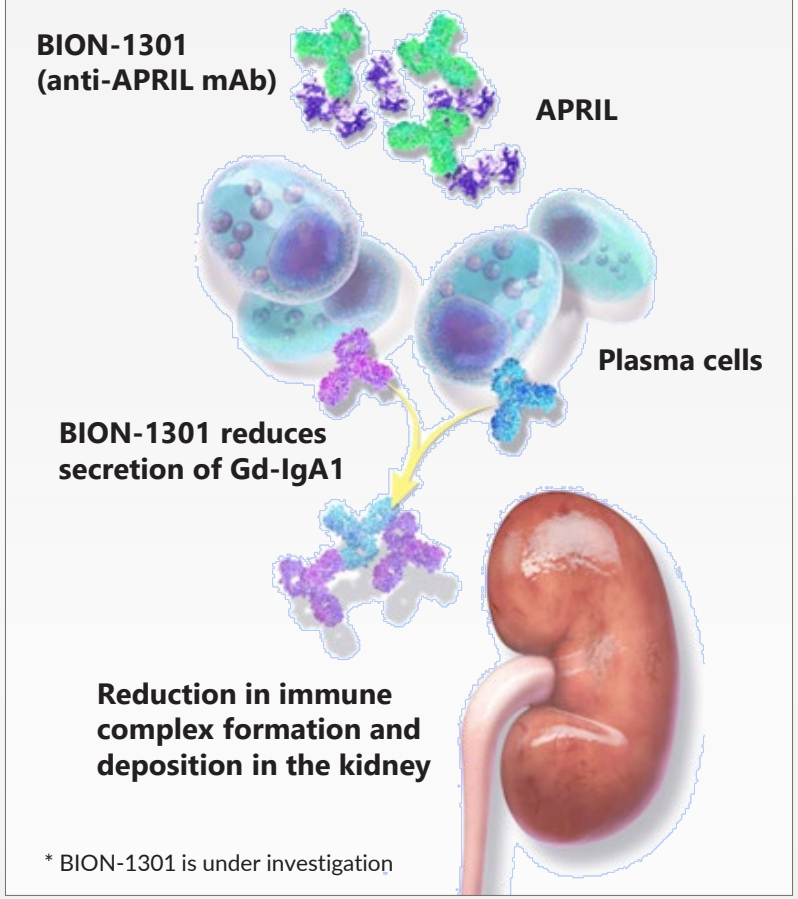
BION-1301

Anti-APRIL Monoclonal Antibody

BION-1301: Potentially Disease-Modifying Anti-APRIL mAb

BION-1301* in IgA Nephropathy



* BION-1301 is under investigation

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 toxicity observed 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]
- Phase 1 bioavailability study in healthy volunteers supports SC dosing[7]

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. 7. Lo J, et al. ISN WCN 21 poster presentation: poster #P0500.

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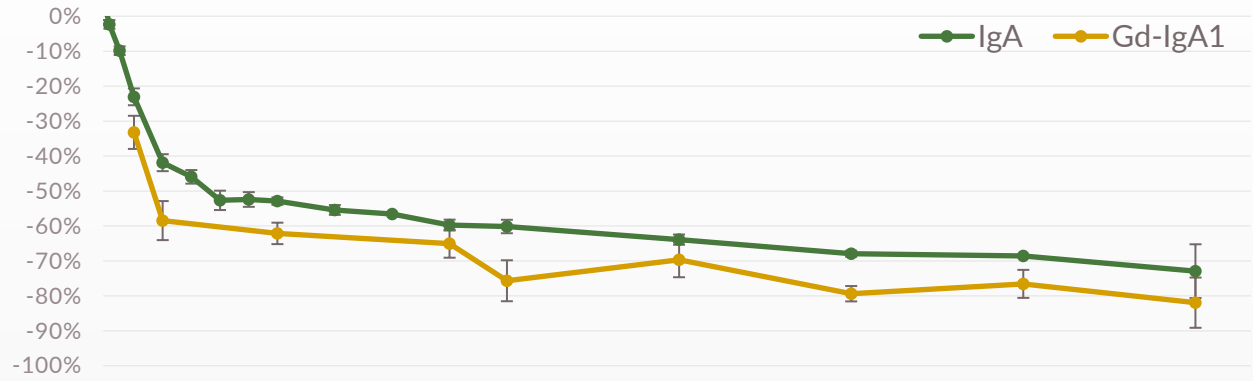
BION-1301 Durably Reduces IgA and Gd-IgA1

IgA and Gd-IgA1

% change from Baseline (Mean ± SEM)

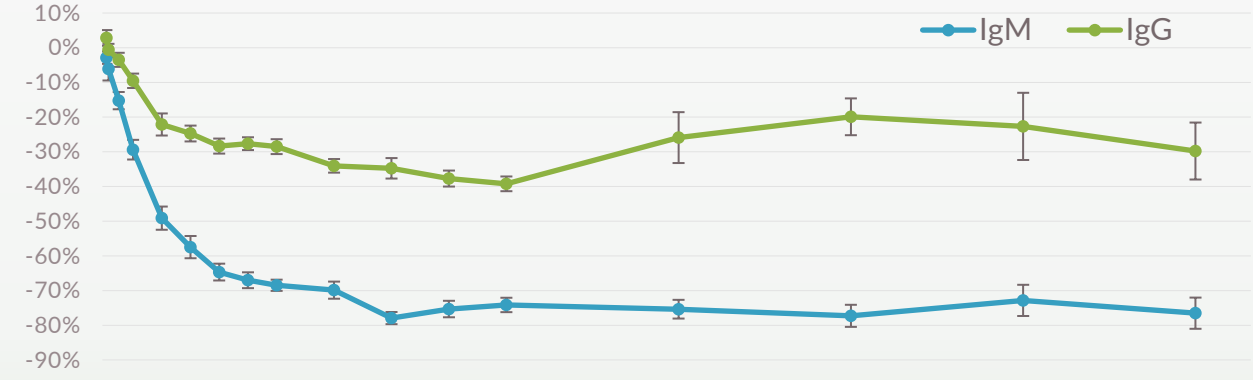


Wk	1	4	8	12	16	20	24	28	40	52	64	76
n, IgA	8	8	8	8	8	2	4	5	6	3	2	2
n, Gd-IgA		8		8			4	3	6	4	2	2

- **BION-1301 durably reduces IgA, IgM, and to a lesser extent, IgG in patients with IgAN**

IgM and IgG

% change from Baseline (Mean ± SEM)



Wk	1	4	8	12	16	20	24	28	40	52	64	76
n	8	8	8	8	8	2	4	5	6	3	2	2

- **BION-1301 also produces sustained reductions in Gd-IgA1, the pathogenic IgA variant (Hit 1), demonstrating the potential disease-modifying mechanism of BION-1301**

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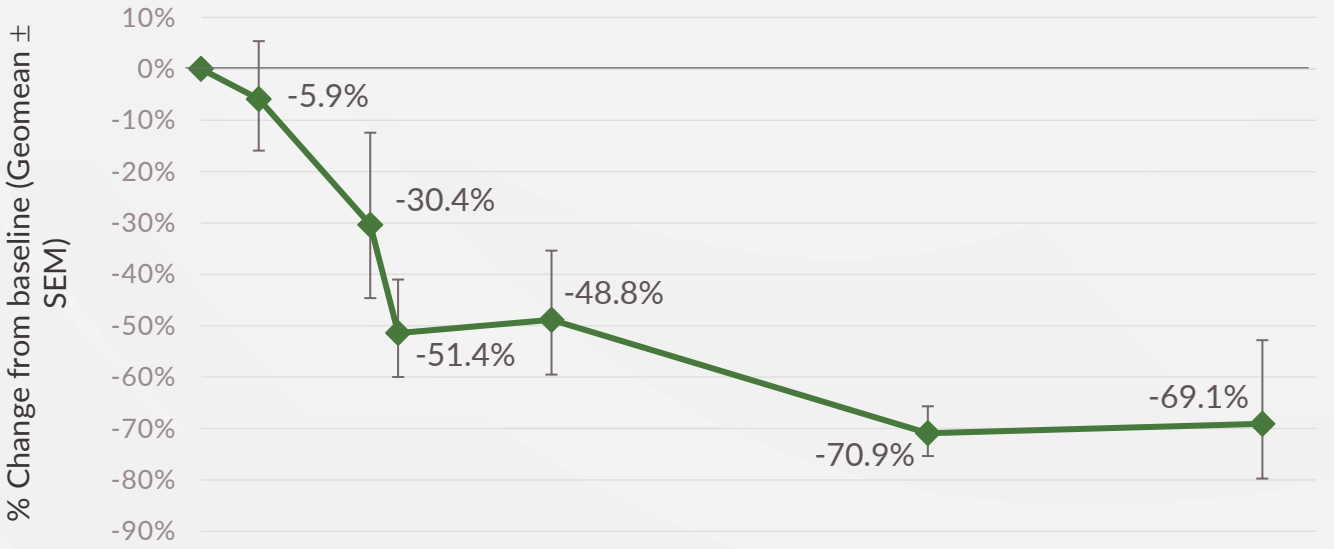
BION-1301 Treatment Results in Sustained 50-70% Proteinuria Reductions

% Reduction in UPCR*



Wk	0	4	12	14	24	52	76
n IV	8	8	7	6	8	2	
n SC						4	2

- **BION-1301 treatment results in proteinuria reductions within 3 months, which are sustained and continue to decline through one year in patients across a range of disease severity**

Median baseline 24-h urine protein excretion: 1.22 g/day (range: 0.74 - 6.47 g/day)

* Results plotted are based on the least squares mean +/- SE of change from baseline on natural log scale from the MMRM back-transformed to a percent reduction from baseline scale

BION-1301 Moving Forward

Plan to initiate pivotal trial in 2023, given strong clinical data and disease-modifying potential

Status

Cohort 1 in IgAN	450 mg IV q2w	Enrollment of 10 patients completed
Cohort 2 in IgAN	600 mg SC q2w	Enrolling up to 30 patients
Optional Cohort 3	Not deemed necessary	

BION-1301

Demonstrates ~50% proteinuria reductions in patients with IgAN after three to six months of treatment, with ~70% reductions observed in six patients at one year and in two patients at 1.5 years of treatment

Next Steps

- Advance BION-1301 into phase 3 with the current Cohort 2 dose of 600 mg SC q2w
- Finalize phase 3 trial design, conduct site/country feasibility and align with regulatory authorities
- Initiate global phase 3 trial in 2023

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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

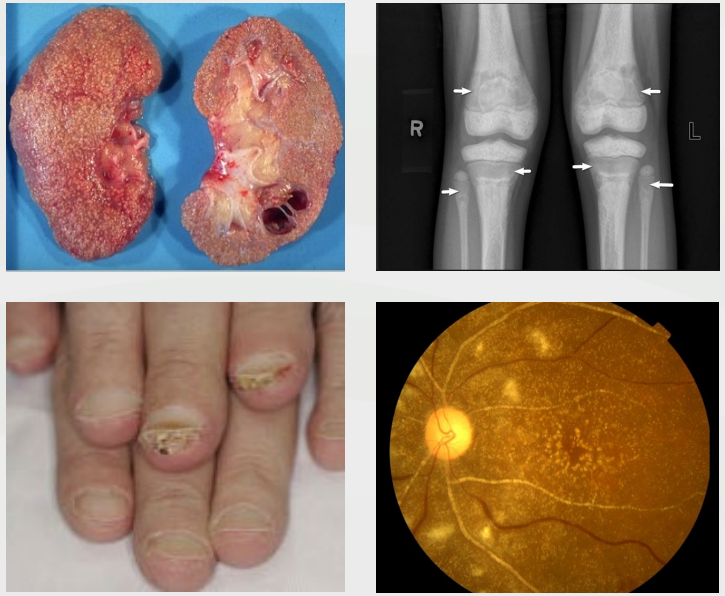
Primary hyperoxalurias (PH) 1-3 are ultra-rare diseases

- 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

Idiopathic hyperoxaluria is 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

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



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.

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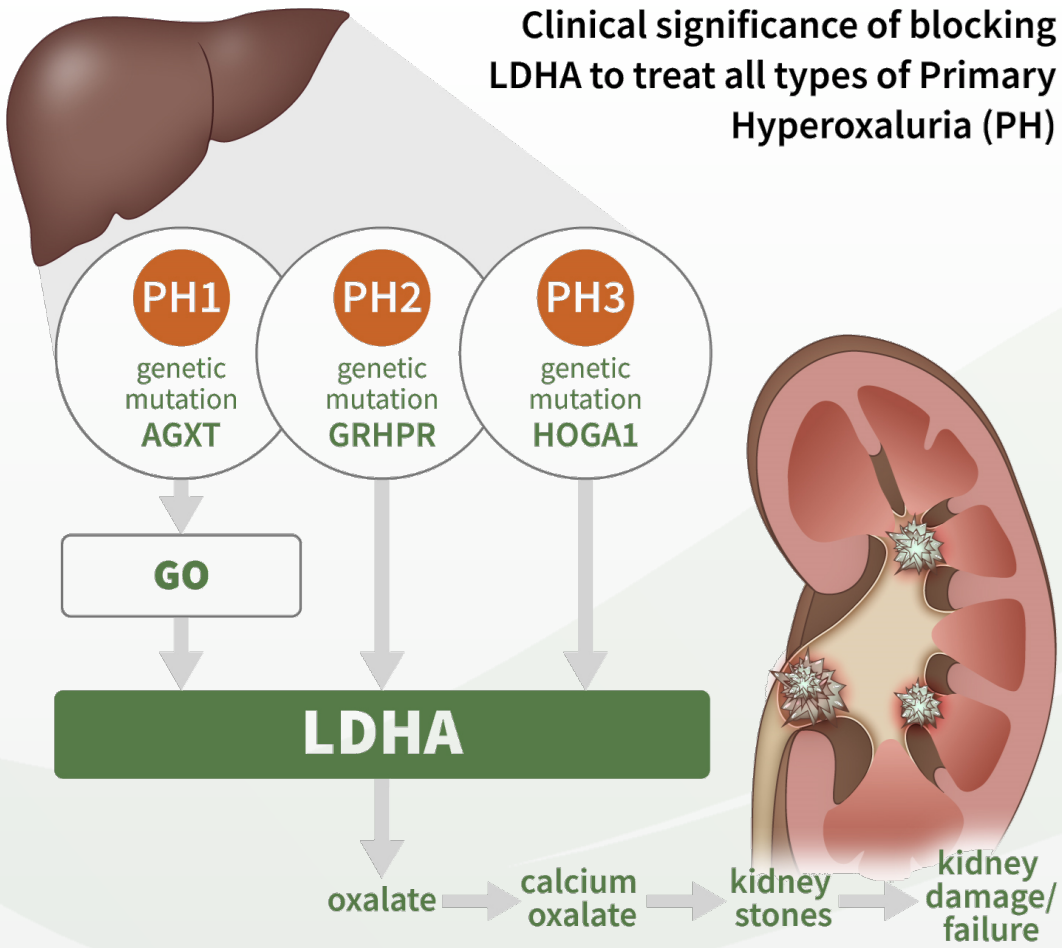
Targeting LDHA Addresses All Types of PH



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 endogenous oxalate
- Liver-targeting profile is desired to maximize target engagement and minimize systemic exposure
- CHK-336 is an oral small molecule LDHA inhibitor with liver-targeted tissue distribution

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

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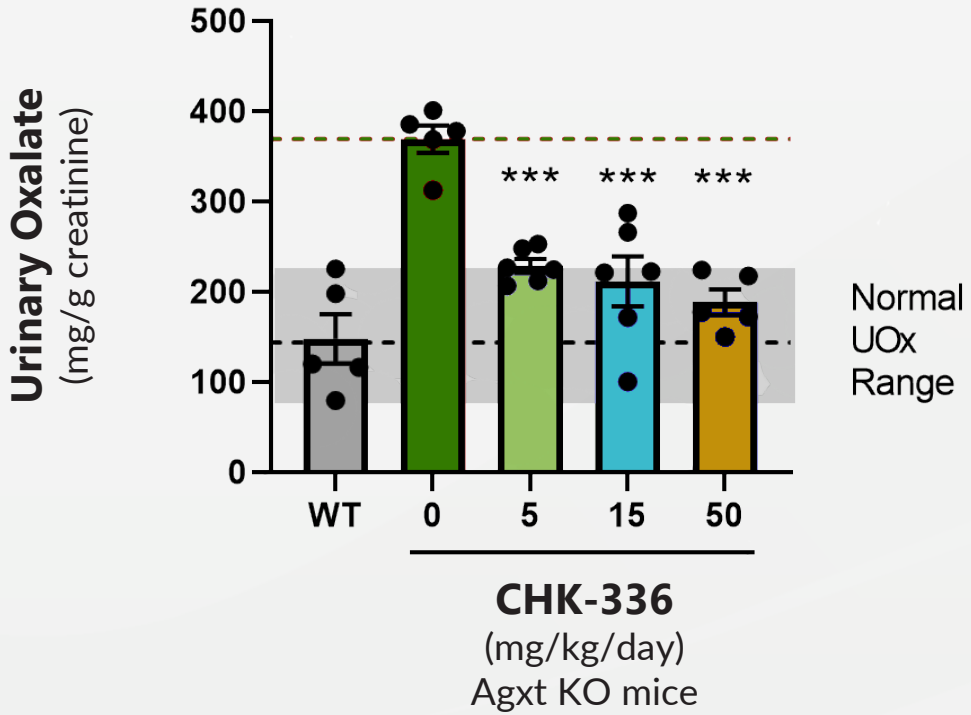
CHK-336: Oral Small Molecule LDHA Inhibitor for PH

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

Efficacy in Mouse PH1 Model



- 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
- Phase 1 healthy volunteer study ongoing

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 rare, severe CKDs with defined genetic and molecular drivers

Target Selection & Validation

Systems Biology

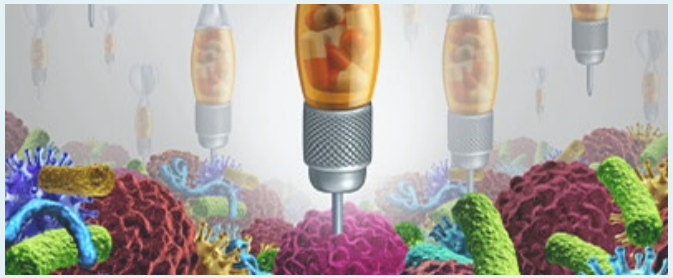
Molecular Classification of CKD

Target ID
Target Validation
Patient Stratification

evotec

NURTURE
National Unified Renal Translational Research Enterprise

Translational Models



Modeling Human Disease

Disease Mechanisms
Target Validation
Deep Biological Insights

Detailed insights into molecular pathogenesis of stratified CKDs

Target Execution

Development Candidates

Drug Discovery
+
Partnerships

Growing Pipeline

First-in-Class or Best-in-Class
Expert & Focused Chemistry, Biology, Pharmacology, DMPK, BD

Novel & differentiated molecules

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Chinook's Precision Medicine Platform Fueled by One of the Most Comprehensive PANOMICS Kidney Programs

UK Academic / Industry consortium for CKD biobank

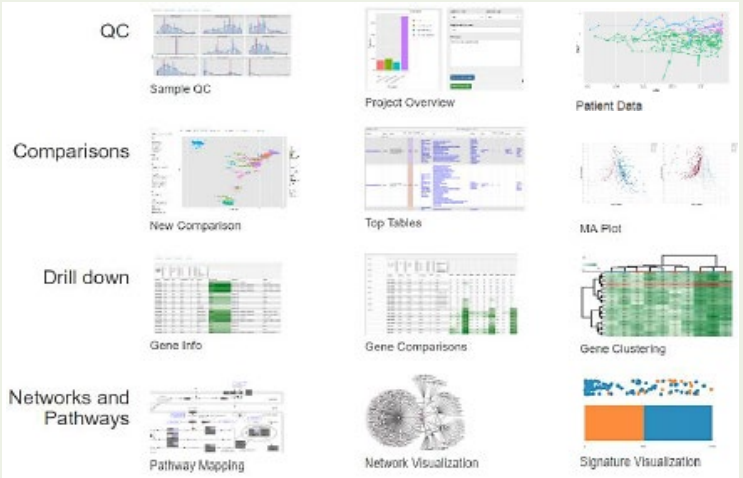
NURTURE
National Unified Renal Translational Research Enterprise

evotec

Liquid Samples
3000 CKD, 800 NS, 100 Controls

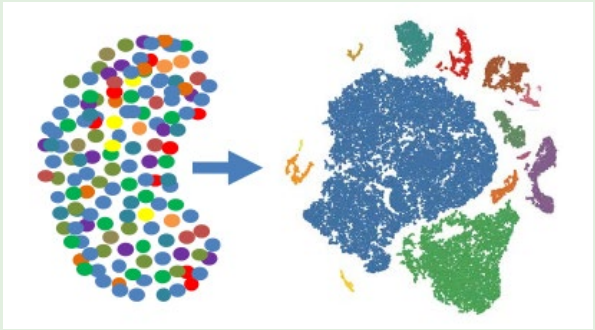
FFPE Kidney Biopsies
340 CKD, >400 NS

Multi-OMICS Integration Platform



- **Target Validation**
- **Target ID Pipeline**
- **Patient Stratification**

Kidney single-cell RNAseq



Washington University Nephrology
#nephStrong
Washington University in St. Louis School of Medicine

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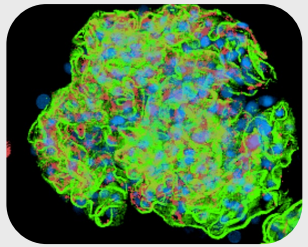
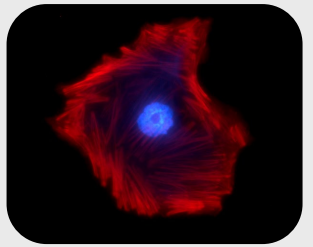
UNIVERSITY OF MICHIGAN

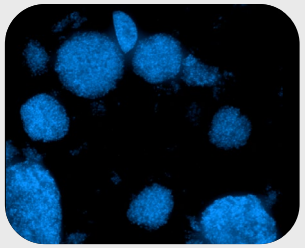
Translational Models



Isolated glomeruli



Podocytes



Human iPSC kidney organoids

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Financials & Catalysts

Financial Strength
NASDAQ: **KDNY**

Strong Balance Sheet	$405.2M in cash, cash equivalents and marketable securities as of June 30, 2022
Cash Guidance	Operating capital into 2025 based on current business plan
SanReno Joint Venture	• 50:50 joint venture in China for development and commercialization of atrasentan and BION-1301 • Provides benefits through better execution and greater access to large IgAN patient populations in Asia • Financial upside through equity ownership, milestones and royalties and reciprocal rights of first negotiation for future developed or in-licensed kidney disease products
Common Stock Outstanding	• ~67.5 million shares as of July 31, 2022* • ~71.3 million fully diluted shares as of July 31, 2022**

* Includes 4.6M pre-funded warrants
** Treasury method. Includes 7.0 million options with average exercise price of $13.21 and 1.3 million RSUs outstanding as of 6/30/22

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Catalysts

Program	Indication	Catalyst	H1 2022	H2 2022	2023
Atrasentan	*IgA Nephropathy*	**Present data from IgAN patient cohort of AFFINITY**	✓	●	
		Initiate phase 2 trial in combination with SGLT2i in IgAN			●
		Report topline proteinuria data from ALIGN			●
	Glomerular Diseases	**Present additional data from other AFFINITY cohorts**			●
BION-1301	*IgA Nephropathy*	**Present phase 1/2 data from Cohort 1 in IgAN**	✓	●	
		Present phase 1/2 data from Cohort 2 in IgAN		●	●
		Initiate phase 3 trial in IgAN			●
CHK-336	*Primary Hyperoxaluria*	**Initiate phase 1 study in healthy volunteers**	✓		
		Report phase 1 healthy volunteer data and initiate phase 2 POC trial in patients with primary hyperoxaluria			●

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