CHINOOK THERAPEUTICS

Chinook Therapeutics

Developing Precision Medicines for Kidney Diseases

J.P. Morgan Healthcare Conference
January 12, 2022

Note Regarding Forward-Looking Statements

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The Time is Now for Kidney Disease Drug Development

Large Unmet Needs

Up to 10% 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

Clear Development Paths

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, selective ET_A antagonist
- Phase 2 AFFINITY data from IgAN cohort in H1 2022 and from other glomerular disease cohort(s) in H2 2022
- Phase 3 ALIGN proteinuria data expected in 2023

BION-1301

- Anti-APRIL monoclonal antibody (mAb)
- Demonstrated reductions in mechanistic biomarkers and clinically meaningful proteinuria improvements in patients with IgAN
- Additional IV and SC phase 1/2 data in IgAN patients and updates on pivotal trial planning expected in 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 initiation in HVs planned for H1 2022

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 2024

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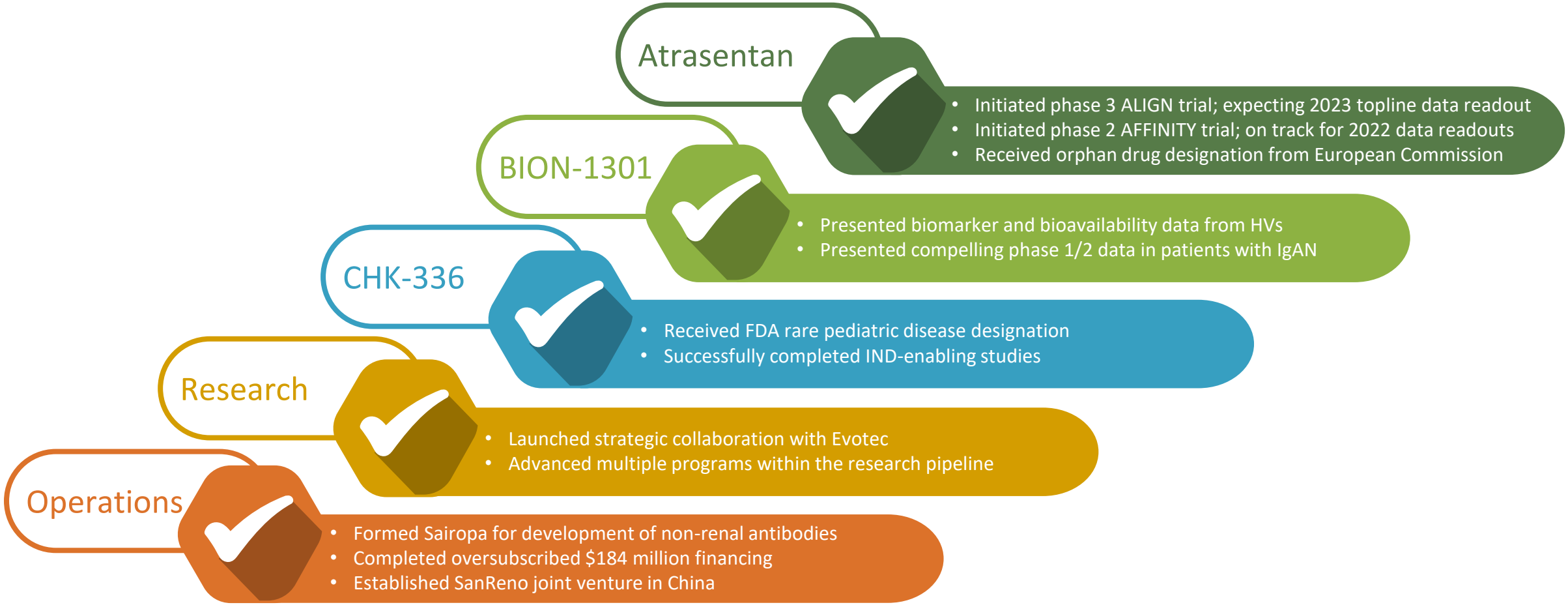
Advancing a Diversified Pipeline of Best-in-class Programs

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 planned for H1 2022		
Research & Discovery Programs	Rare, severe chronic kidney diseases		Potential 2022 DC				
			Multiple programs				

Continuing to evaluate opportunities to add kidney disease programs to pipeline

Achieved Many Milestones in 2021



Well-positioned to continue strong execution in 2022

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China JV: SanReno Therapeutics

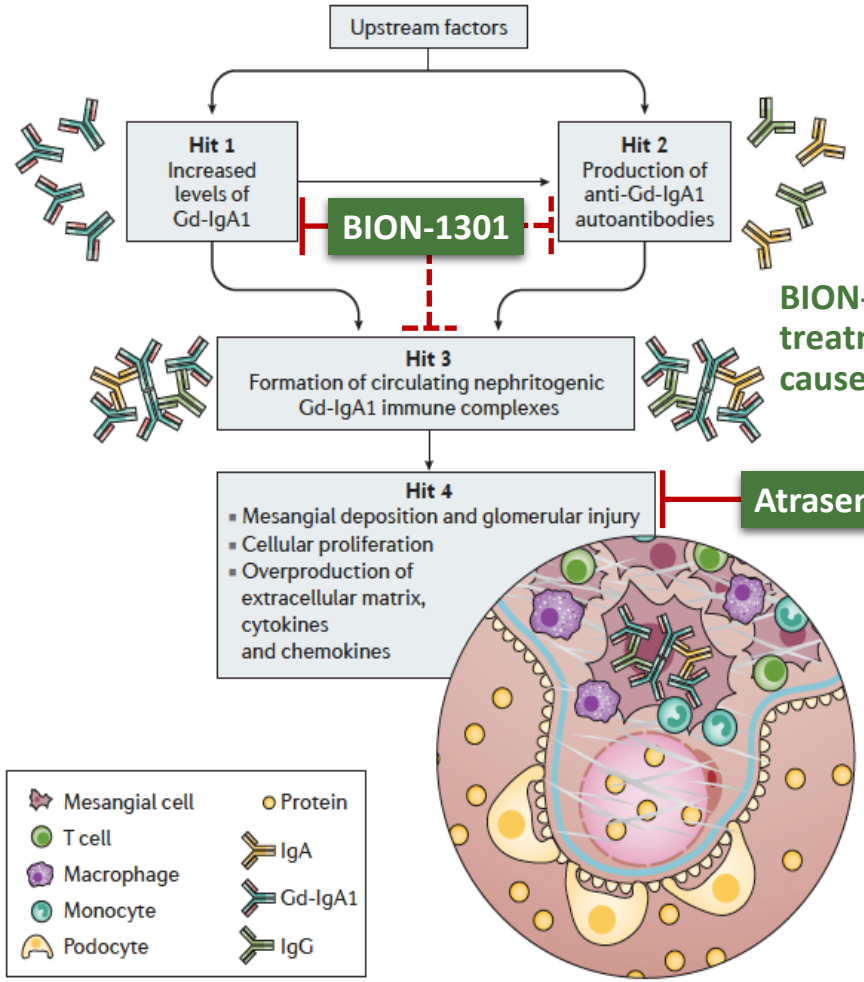
Expands global reach and establishes strong presence in region with high unmet need

- Chronic kidney disease is a major public health concern in China, affecting >10% of the population
  - High prevalence of IgAN among east-Asian populations likely due to genetic variants
  - Current treatment landscape limited to RASi, traditional Chinese medicine & immunosuppressants
- 50/50 joint venture formed to build leading kidney disease company in the People's Republic of China, Hong Kong, Macau, Taiwan and Singapore
  - Expands Chinook's access to Chinese patient populations, KOL networks and local operations to accelerate enrollment and regulatory approval of Chinook's programs
  - SanReno to develop, manufacture and commercialize atrasentan and BION-1301 in the region
  - Chinook eligible for milestone and royalty payments
  - Reciprocal rights of first negotiation for future developed or in-licensed kidney disease products
- Investor syndicate led by Frazier Healthcare Partners & Pivotal bioVenture Partners China
  - Initial investment of $40M
  - Extensive experience with company formation, drug development and commercialization in the region

Why Target IgA Nephropathy?

- Most common primary glomerular disease globally with ~140K – 150K US prevalence
- Only one steroidal treatment approved; >50% of patients remain at risk for progression
- 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 cause of IgAN (Hit 1)

Atrasentan has potential to attenuate hallmark characteristics of IgAN, including mesangial cell activation, proteinuria and kidney inflammation and fibrosis (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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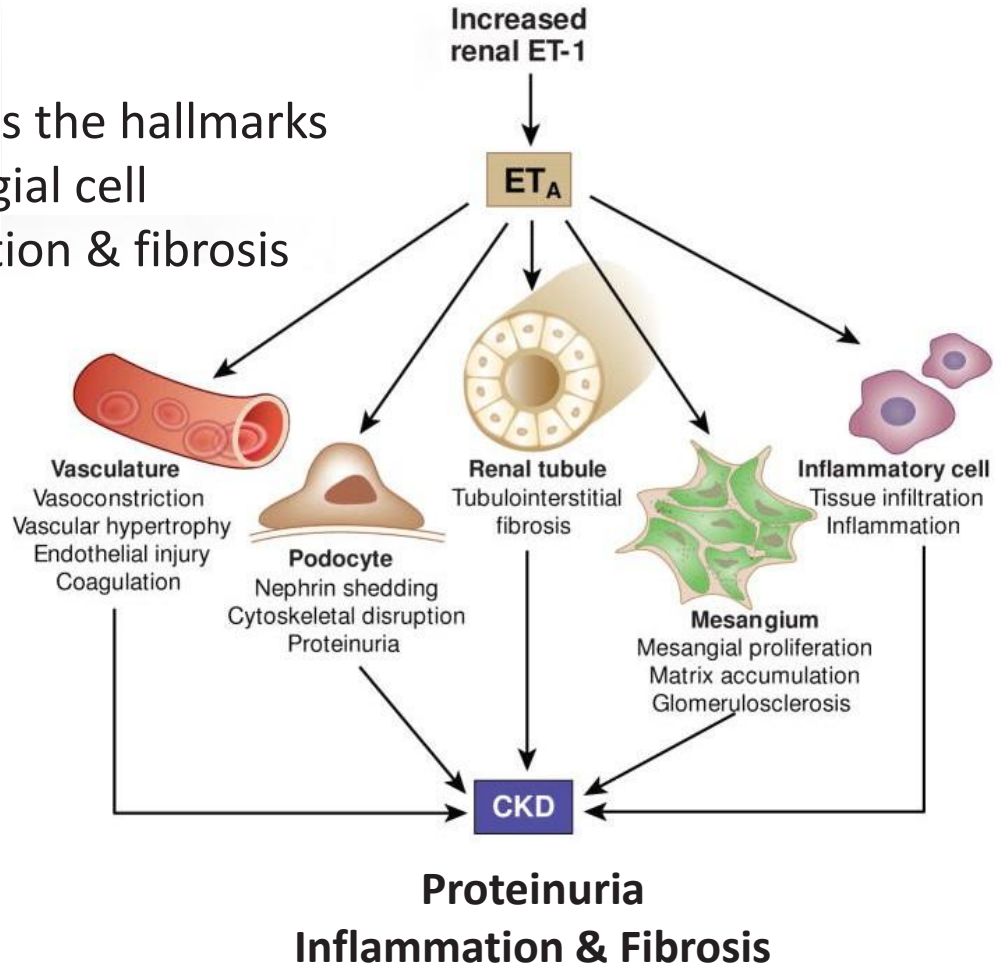
Atrasentan

Potent and Selective Endothelin A Receptor (ET_A) Antagonist

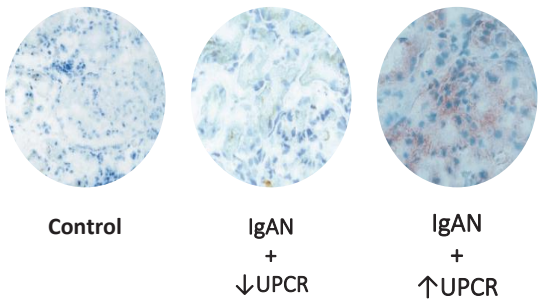
Atrasentan: a Potent and Selective ET_A Antagonist

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



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 by 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.

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

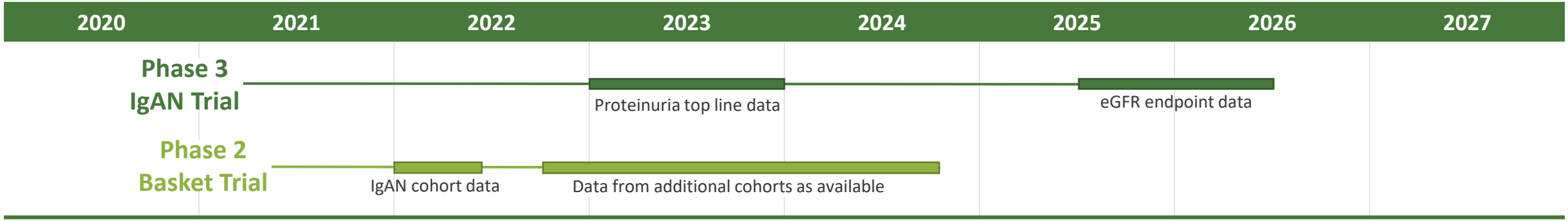
Phase 2 Basket Trial to Expand Potential Across Proteinuric Glomerular Diseases

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/g
- FSGS
- Alport syndrome
- DKD combined with SGLT2 inhibitors



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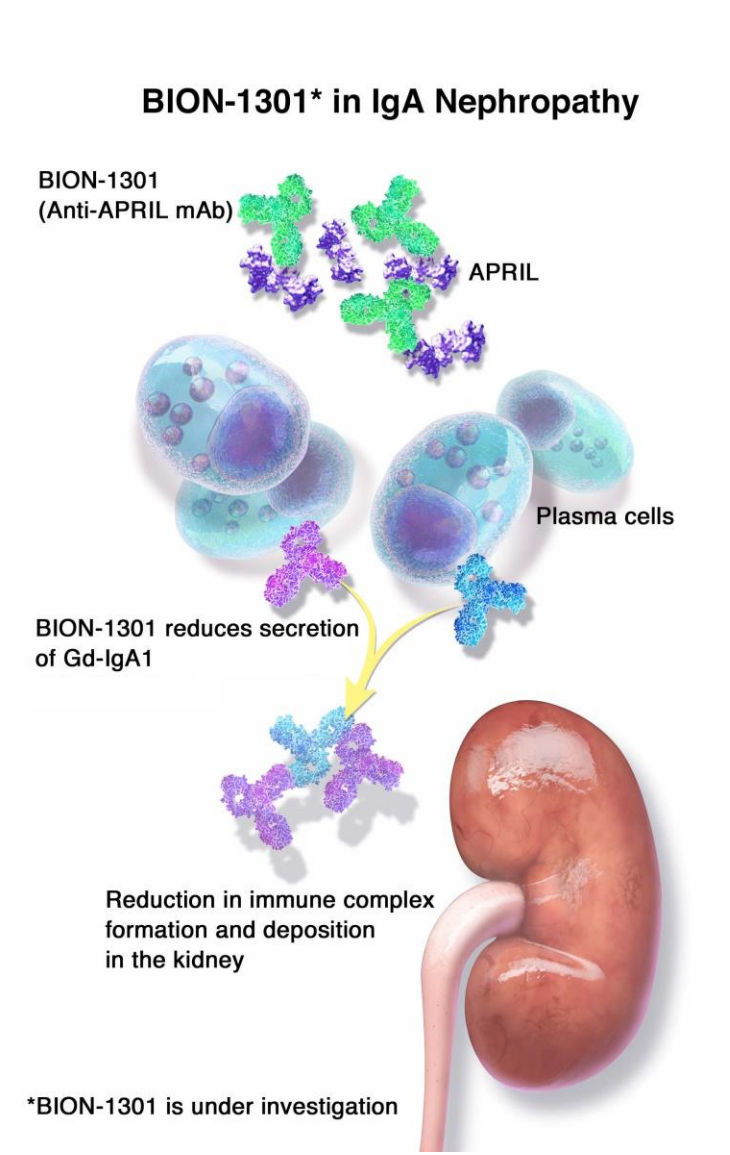
BION-1301

Anti-APRIL Monoclonal Antibody

BION-1301: Potentially 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]

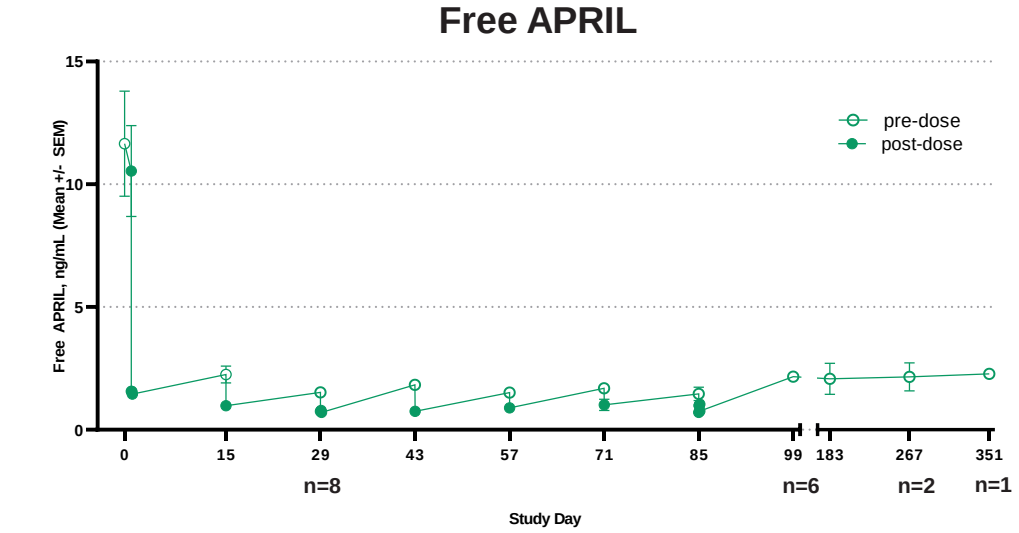
BION-1301: humanized IgG4 monoclonal antibody that blocks APRIL binding to its receptors

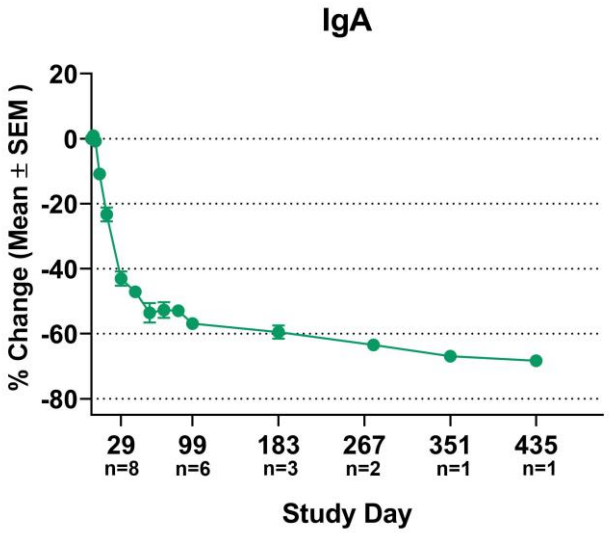
- Potentially disease-modifying mechanism to deplete Gd-IgA1 (**Hit 1**) and prevent pathogenic immune complex formation (**Hit 3**)
- No toxicity observed in NHP tox studies of IV BION-1301 for up to 6 months and SC for up to 1 month[5]
- Well-tolerated up to 2700mg in phase 1 multiple myeloma study[6]
- Phase 1 bioavailability study in HVs supports SC dosing[7]

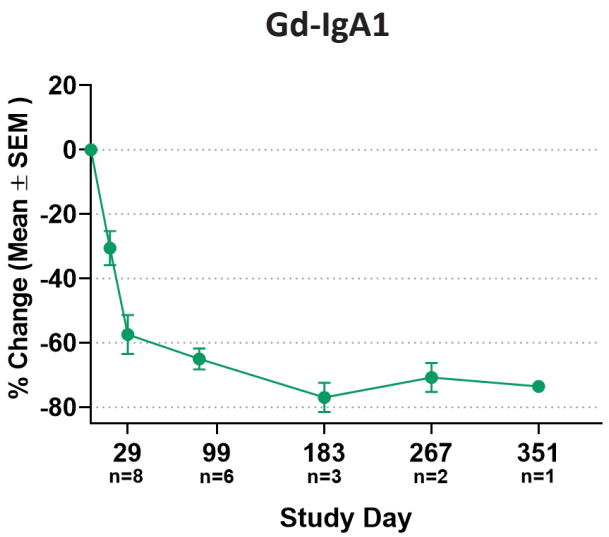
1. Guadagnoli, M, et al. Blood. 2016. 2. He B, et al. Nat Immunol. 2010; 3. Zhai YL, et al. Medicine (Baltimore). 2016. 4. Yu XQ, et al. Nature Genetics. 2012 Feb;44(2):178-182. 5. Kreijtz J, et al. ERA-EDTA 2020 poster presentation: poster #P0379. 6. Bensinger W, et al. ASCO 2019 poster presentation: poster #338. 7. Lo J, et al. ISN WCN 21 poster presentation: poster #P0500.

Initial BION-1301 Biomarker & Proteinuria Responses

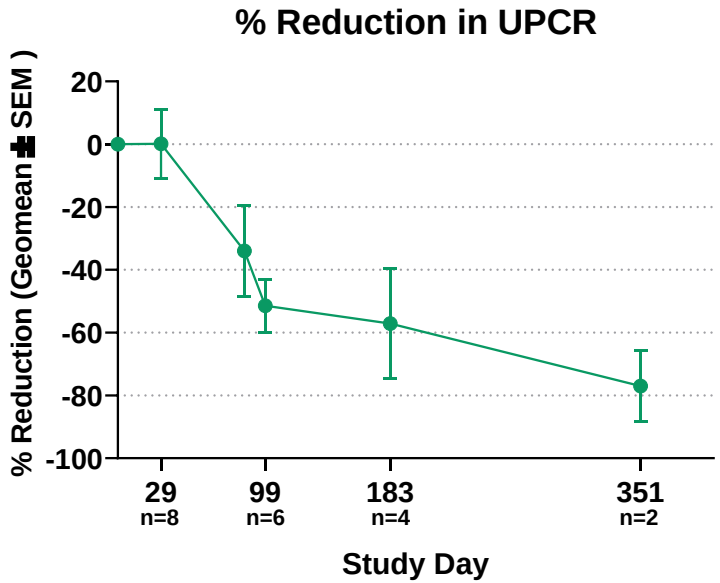
Cohort 1: 450 mg BION-1301 IV q2w







- In patients with IgAN, BION-1301 is well-tolerated w/no drug related adverse events, and demonstrates:
  - Rapid and sustained free APRIL reductions
  - Durable reductions in IgA and Gd-IgA1 biomarkers



- Median baseline 24-h urine protein excretion*: 1.22 g/day (range: 0.74 - 6.47 g/day)
- BION-1301 treatment results in clinically meaningful proteinuria reductions within 3 months in patients across a range of disease severities

Barratt, J, et al. ASN Kidney Week 2021 poster presentation: poster #PO1632.

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Moving BION-1301 Forward

Plans to accelerate development given strong clinical data and disease-modifying potential

BION-1301 has demonstrated >50% proteinuria reduction in patients with IgAN after three to six months of treatment, with further reductions observed in two patients through approximately one year of treatment

Next Steps:

- Determine optimal SC dose and schedule based on data from phase 1/2 cohorts
- Finalize late-stage clinical development strategy and pivotal trial design
- Determine combination strategy with other mechanisms, including atrasentan
- Provide updates on development plan in 2022

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CHK-336

Potent and Selective Small Molecule LDHA Inhibitor

Hyperoxalurias are Diseases Caused by Excess Oxalate

LDHA is a potential therapeutic target to treat all forms of primary hyperoxaluria (PH)

PH1-3 are a group of ultra-rare autosomal recessive disorders resulting in hepatic overproduction of oxalate

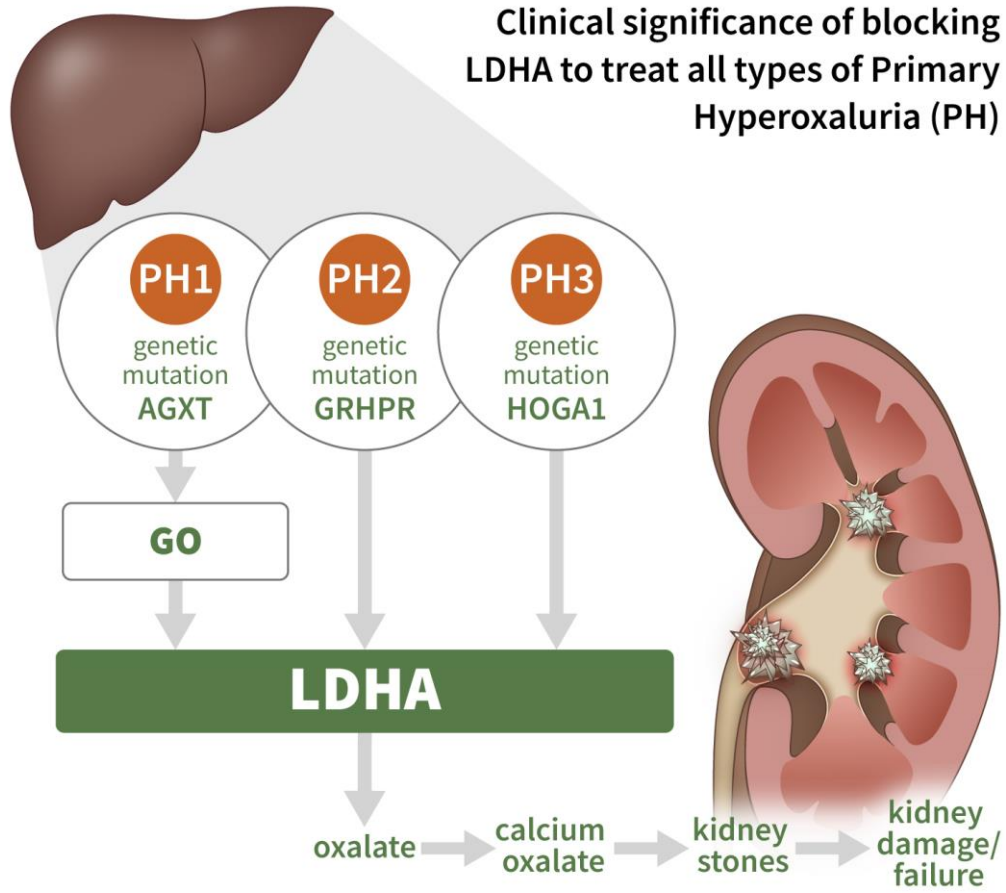
- PH leads to recurrent kidney stones and can lead to kidney failure, if left untreated
- Median age of kidney failure for PH1 is 23 years
- ~5,000 – 7,000 PH1 patients in the US and Europe

Secondary hyperoxalurias are more common

- Acquired condition resulting from increase in dietary oxalate intake, intestinal oxalate absorption or endogenous oxalate overproduction

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

- 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



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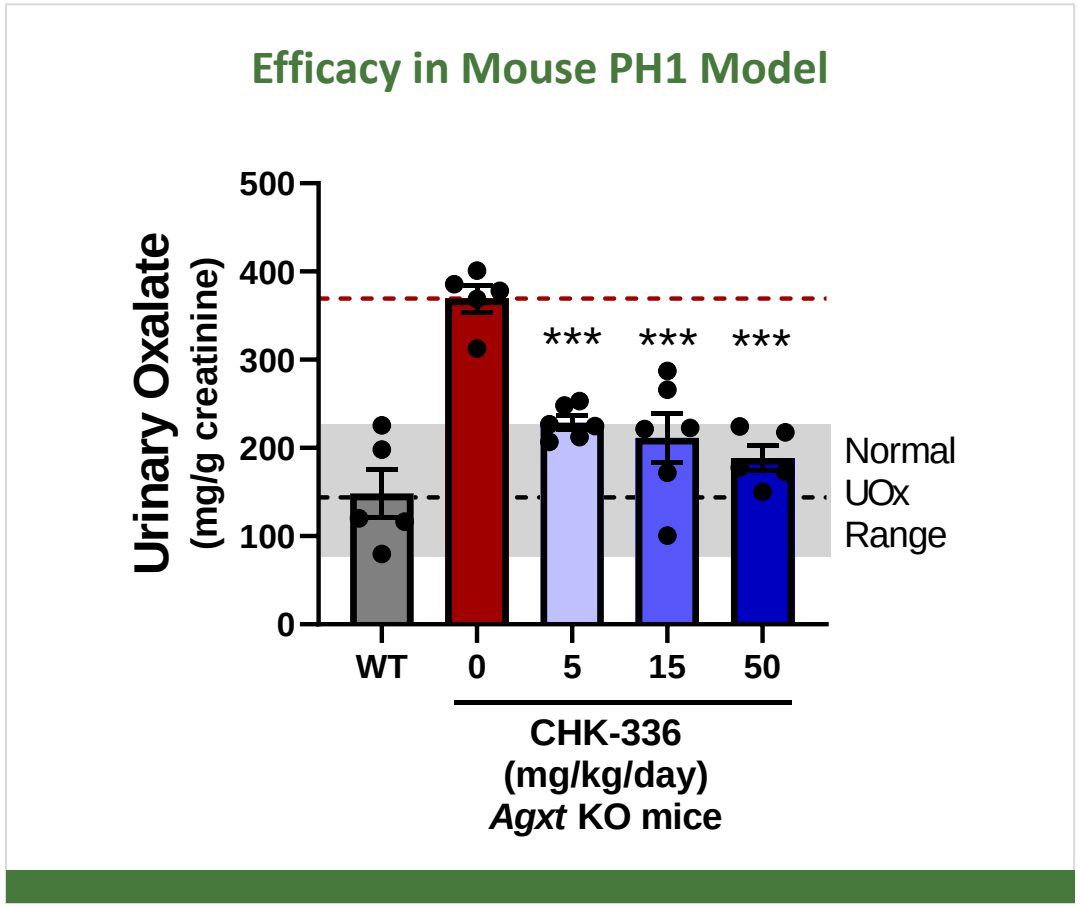
CHK-336: Oral Small Molecule LDHA Inhibitor for PH

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



- 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 planned for phase 1 HV initiation in H1 2022

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

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Financials & Catalysts

Financial Strength

NASDAQ: **KDNY**

Strong Balance Sheet

- $204.8M in cash, cash equivalents and marketable securities as of September 30, 2021
- ~$172M in net proceeds from upsized public offering in November 2021

Cash Guidance

- Operating capital well into 2024 based on current business plan

Common Stock Outstanding

- ~58.2 million shares as of November 15, 2021*
- ~60.4 million fully diluted shares as of November 15, 2021**

* Includes 3.6M pre-funded warrants

** Treasury method. Includes 6.2 million options with average exercise price of $13.04 and 0.9 million RSUs outstanding as of 9/30/21.

Catalysts

Program	Indication	Catalyst	H1 2022	H2 2022	2023
Atrasentan	IgA Nephropathy	Present data from IgAN patient cohort of AFFINITY	●		
		Report topline proteinuria data from ALIGN			●
	Glomerular Diseases	Present data from additional AFFINITY patient cohort(s) and determine strategy for life cycle management indications		●	●
BION-1301	IgA Nephropathy	Present additional phase 1/2 IV/SC data from Cohort 1 in IgAN	●		
		Present phase 1/2 SC data from Cohorts 2/3 in IgAN		●	●
		Initiate pivotal 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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