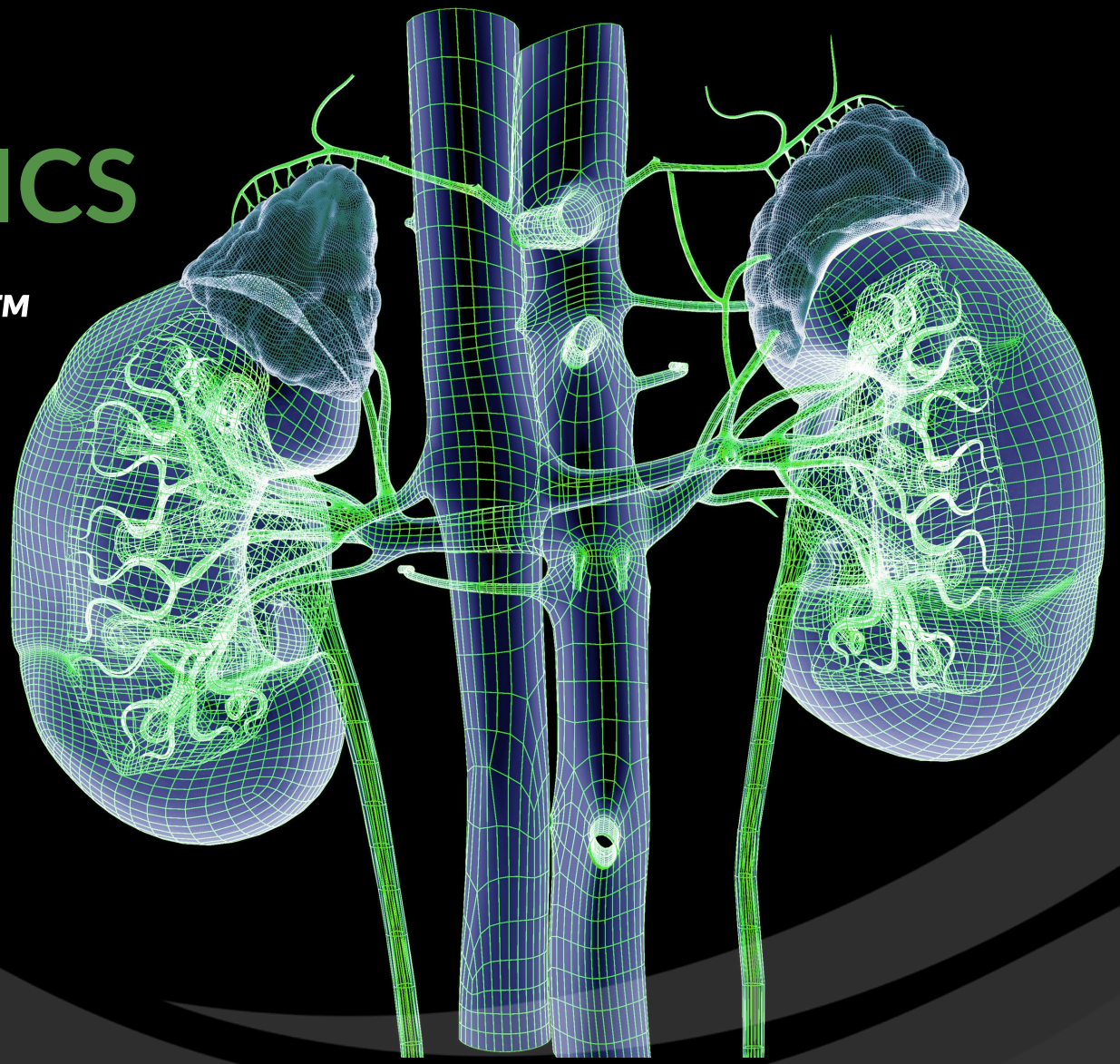
CHINOOK™ THERAPEUTICS

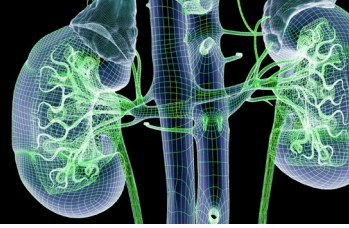
CHINOOK THERAPEUTICS

Changing the Course of Kidney Care™

February 2023



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. 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.

CHRONIC KIDNEY DISEASES ARE A

SEVERE & GROWING

WORLDWIDE PROBLEM

KIDNEY DISEASE AFFECTS

10%

OF GLOBAL POPULATION

800M people worldwide, including 37M in U.S.[1, 2]

STAGGERING COSTS TO HEALTHCARE SYSTEM

>$130B

Annual U.S. healthcare costs driven by kidney diseases[3]

KIDNEY DIALYSIS

156
Average hospital and dialysis center visits a year/patient[4]

$200K
Per patient annually[5]

KIDNEY TRANSPLANT

~23K
Transplants annually (U.S)[6]

$400K
Per transplant[7]

4 YRS
Waitlist (U.S.)[6]

1. GBD Chronic Kidney Disease Collaboration, The Lancet, 2020; 395(10225):709-733; 2. Centers for Disease Control and Prevention, Chronic Kidney Disease in the United States, 2021; 3. 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; 4. National Kidney Foundation, Dialysis, https://www.kidney.org/atoz/content/dialysisinfo; 5. Childers CP, Dworsky JQ, Kominski G, Maggard-Gibbons M. A Comparison of Payments to a For-profit Dialysis Firm From Government and Commercial Insurers. JAMA Intern Med. 2019;179(8):1136–1138. doi:10.1001/jamainternmed.2019.0431. 6. Kidney Disease Statistics for the United States, National Institute of Diabetes and Digestive and Kidney Disease: https://www.niddk.nih.gov/health-information/health-statistics/kidney-disease; 7. Average amount charged for select organ transplantations in the U.S. as of 2020, https://www.statista.com/statistics/808471/organ- transplantation-costs-us/).

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THE TIME IS NOW FOR

KIDNEY DISEASE DRUG DEVELOPMENT

CLEAR DEVELOPMENT PATHS

- Increased understanding of underlying disease biology
- New and more validated drug targets
- FDA now recognizes surrogate markers, such as proteinuria and eGFR, as registration endpoints[1]

SHIFT AT THE FDA ALLOWS FOR MORE EFFICIENT STUDIES

5+ YRS

Traditional hard kidney outcomes trials require large numbers of patients and take many years to complete

2-3 YRS

Surrogate endpoint studies can be much smaller, with potential for accelerated approval in 6-9 months and full approval in 2-3 years

VAST SHORTAGE OF NEW KIDNEY DRUGS

IN THE PAST 2 YEARS THERE HAVE BEEN

5 approved drugs for kidney diseases[2]

CURRENTLY, THERE ARE

40+ late-stage drugs in development[3]

1. Thompson et al., CJASN March 2019, 14 (3) 469-481. 2. Estimates based on U.S. FDA, Novel Drug Approvals for 2020 – 2022 and American College of Cardiology data; 3 Evaluate Pharma Competitor Analyzer

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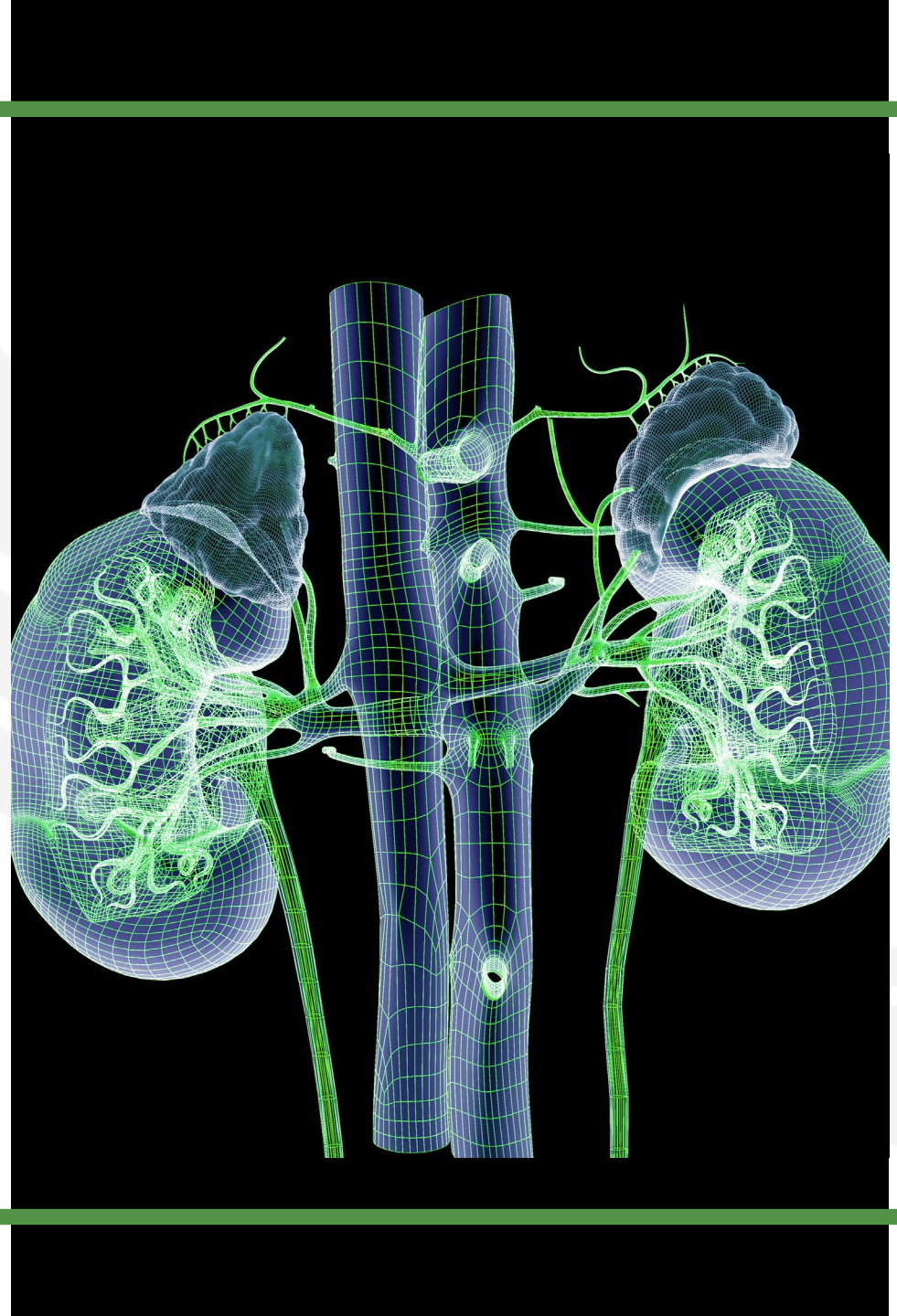

WE ARE
CHANGING THE COURSE OF KIDNEY CARE™

ATRASENTAN
ET_A Antagonist

BION-1301
Anti-APRIL Monoclonal Antibody

CHK-336
LDHA Inhibitor

R&D PROGRAMS
Precision Medicine Pipeline

Disclaimer: Atrasentan, BION-1301 and CHK-336 are investigational compounds that have not been approved by the FDA for any use.

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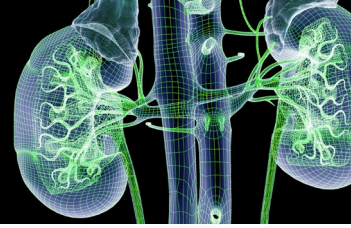
Advancing a Diversified Pipeline of Best-in-Class Programs



Program	Indication	Target Validation	Lead Optimization	IND-Enabling	Phase 1	Phase 2	Phase 3
Atrasentan (ALIGN™)	IgA Nephropathy			*Phase 3 ongoing with topline data expected in Q3 2023*			
Atrasentan (AFFINITY™)	Basket of glomerular diseases					*Phase 2 ongoing*	
BION-1301	IgA Nephropathy					*Phase 1/2 ongoing with phase 3 planned to start in mid-2023*	
CHK-336	Primary & Idiopathic Hyperoxalurias				*Phase 1 HV study ongoing*		
Research & Discovery Programs	Rare, severe chronic kidney diseases		*Multiple programs*				

Continuing to evaluate opportunities to add kidney disease programs to pipeline

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IgA NEPHROPATHY HAS A

LARGE UNMET MEDICAL NEED

IgAN is the most common primary glomerular disease globally and requires chronic treatment

~150,000 Biopsy-confirmed IgAN patients in the U.S.[1]

~45% with >1 g/day[1]

~25% with 0.5 – 1 g/day[1]

Patients with uncontrolled proteinuria despite optimized standard of care RAS inhibition (ACEi or ARB)

~100,000 Patients remain at high risk for progression

High-risk patients living with IgAN will cycle through **multiple therapies** over their disease course, resulting in **several million patient years of treatment**, which will increase as time to ESKD is further delayed

~30-45% of IgAN patients progress to ESKD over 20-25 years[3-6]

ACHIEVING

30% PROTEINURIA REDUCTION

EQUATES TO

>10 YEAR DELAY IN TIME TO ESKD[2]

Greater proteinuria reductions are associated with greater clinical benefit

1. Based on Chinook market research; 2. Carroll KJ, et al. ERA-EDTA Congress 2021, MO246; 3. Reich et al, 2007, JASN; 4. Moriyama et al, 2014, PLOS ONE; 5. Rauen et al, 2020, Kidney Int; 6. Hastings et al, 2018, Kidney Int Rep

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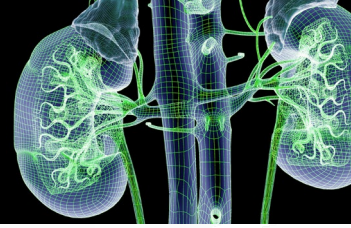
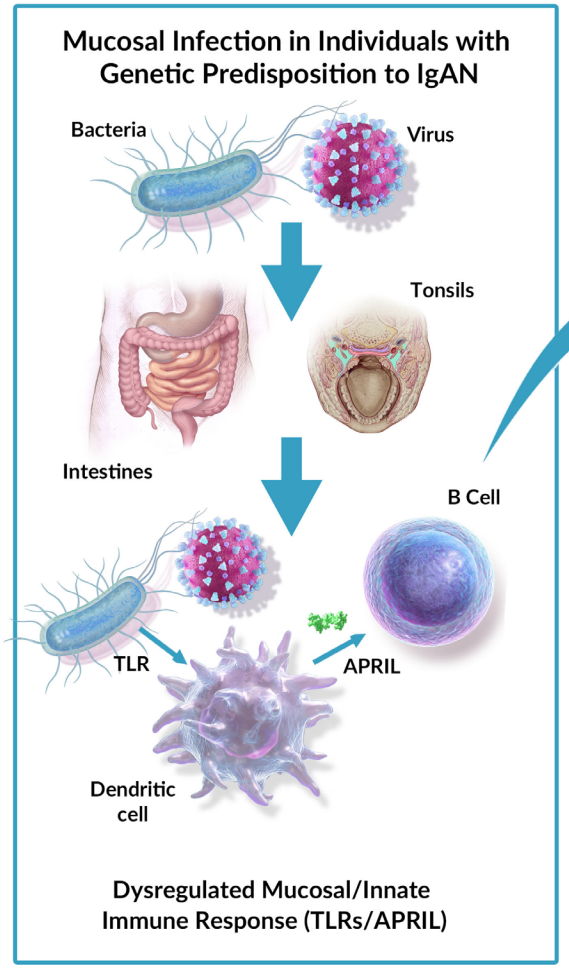
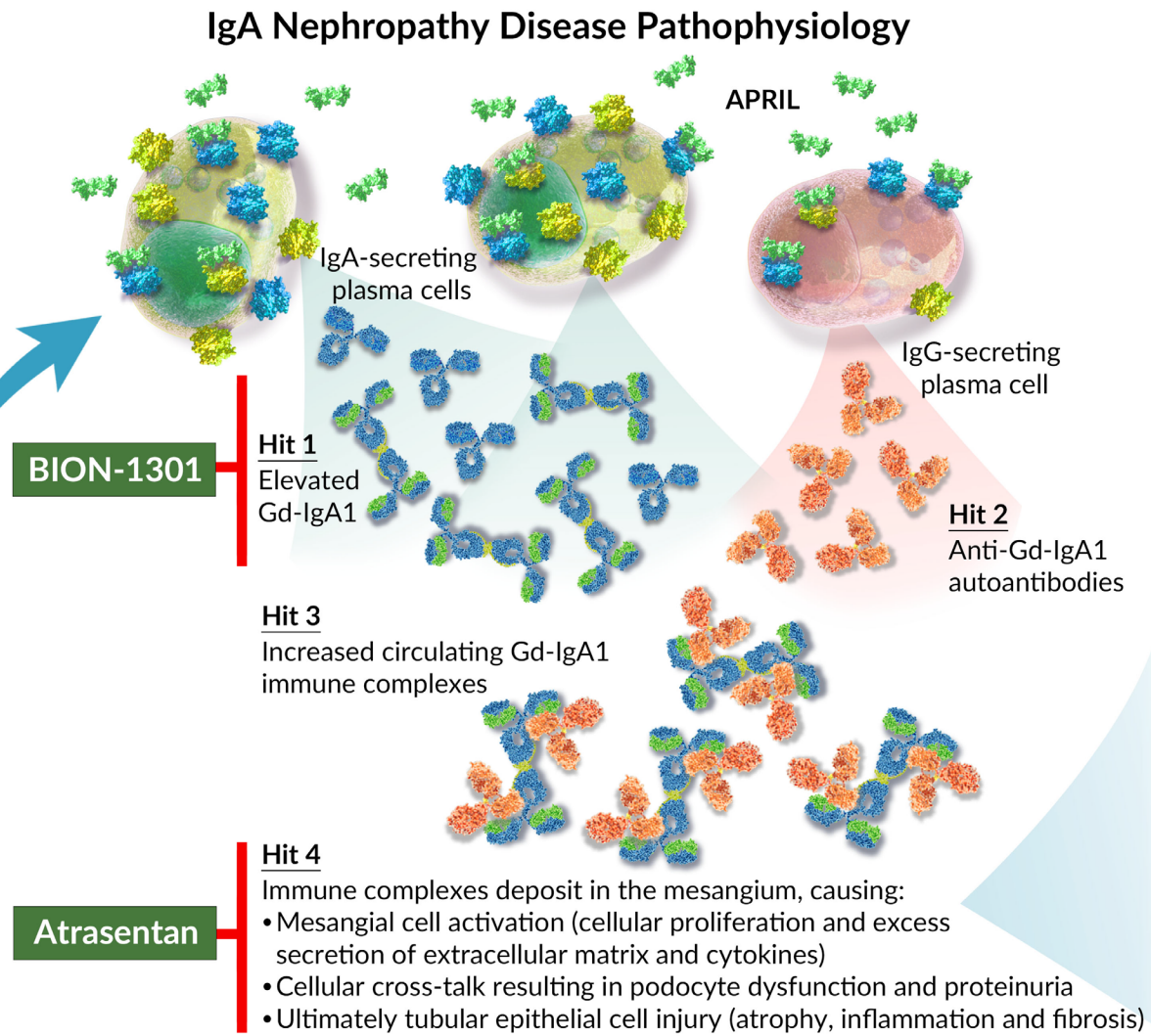
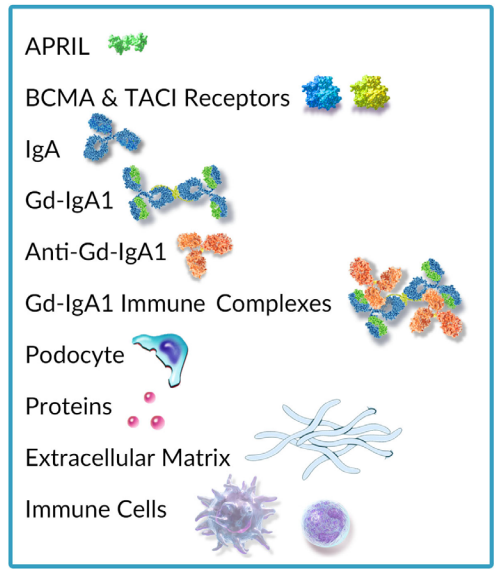
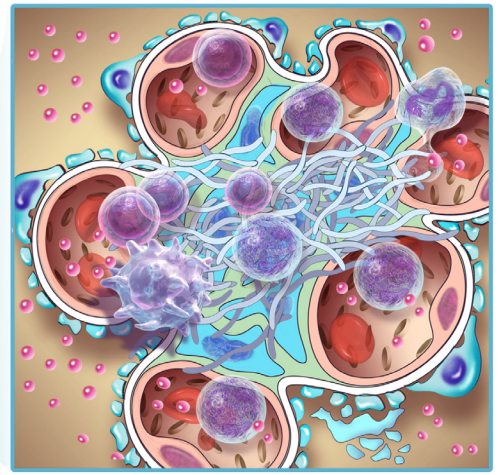
Atrasentan & BION-1301: Two Complementary Programs for IgA Nephropathy











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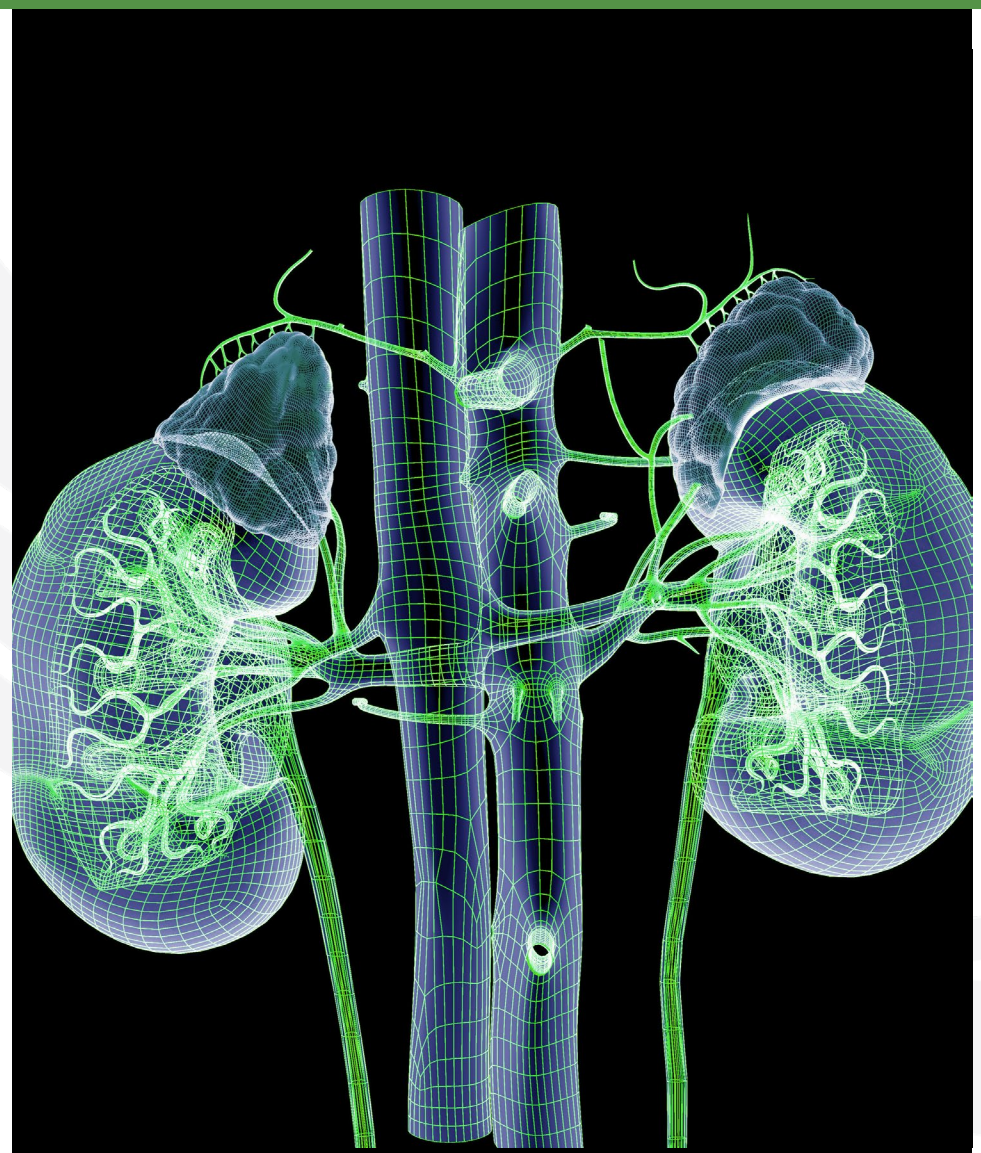

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2023

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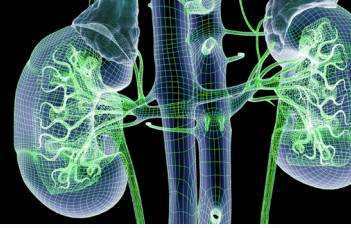
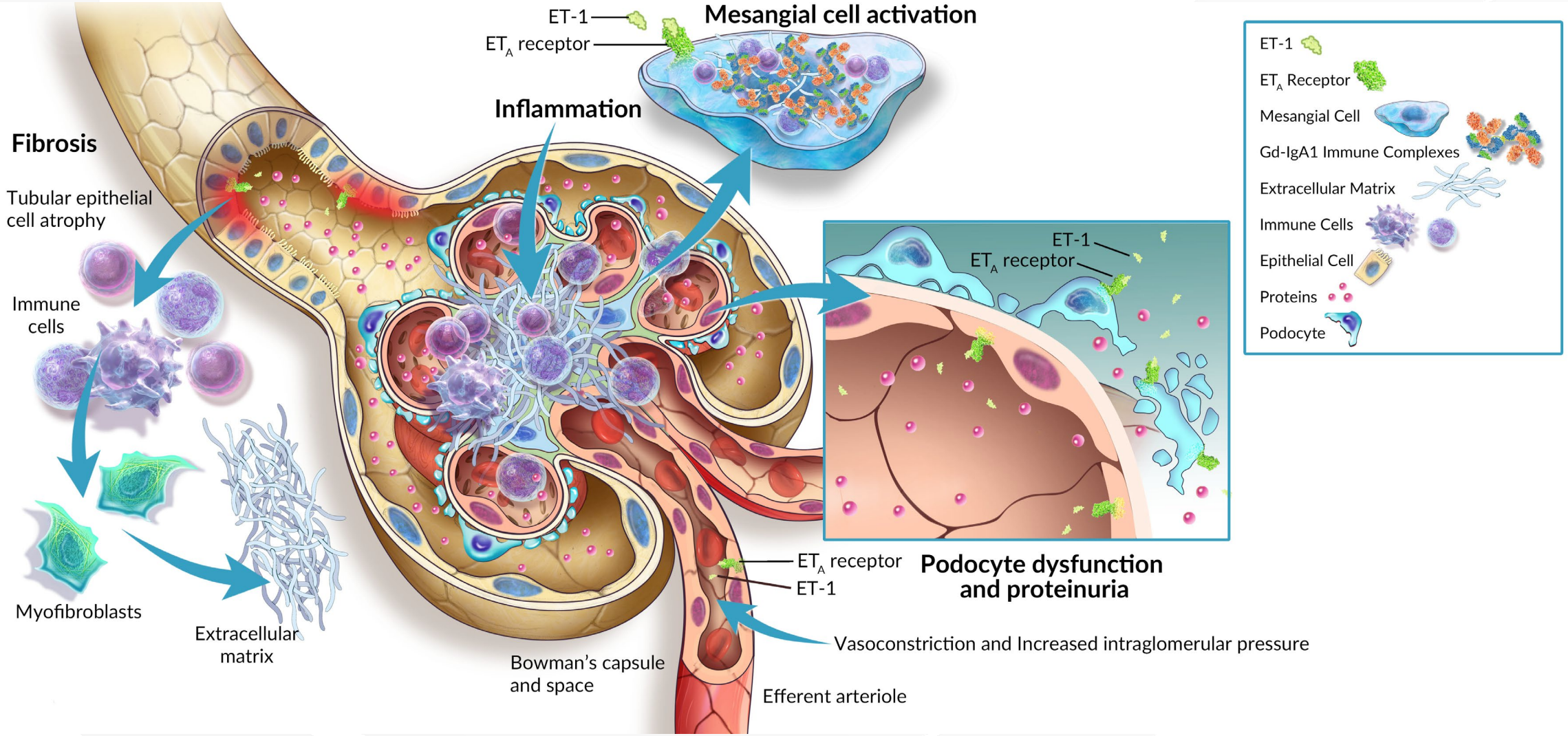
Atrasentan: A Potent and Selective ET_A Antagonist

ET_A Receptor Activation Drives the Hallmarks of IgAN Progression Through Multiple Mechanisms





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Phase 2 AFFINITY™ Basket Study

Exploring Potential Across Proteinuric Glomerular Diseases

TRIAL DESIGN:

- Open-label
- 12-week proteinuria primary endpoint
- 52-week treatment period
- 20 patients per cohort

COHORTS INCLUDE:

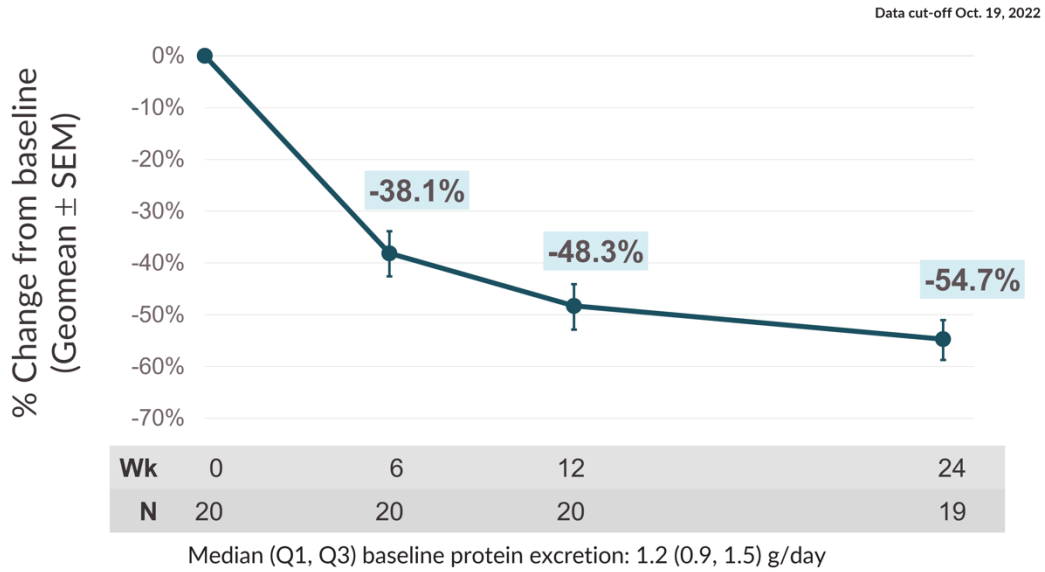
- IgAN with proteinuria 0.5 – <1 g/g
- Two FSGS cohorts (with dose escalation to 1.5 mg)
- Alport syndrome
- DKD combined with SGLT2 inhibitors

Phase 2 AFFINITY™ IgAN Cohort Interim Results:

- >50% mean proteinuria reduction at 24 weeks
- Well-tolerated with no treatment-related severe AEs to date
- No weight gain or other evidence of significant fluid retention

Phase 2 AFFINITY™ IgAN Cohort Interim Results

% Reduction in UPCR



Median (Q1, Q3) baseline protein excretion: 1.2 (0.9, 1.5) g/day

2020	2021	2022	2023	2024	2025	2026	2027
AFFINITY™		IgAN cohort data at ERA & ASN	Data from additional cohorts as available				

Rastogi, A, et al., ASN Kidney Week 2022, TH-PO497

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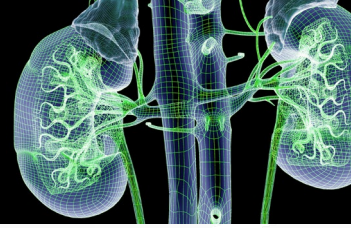
Phase 3 ALIGN™ Study in IgA Nephropathy



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/1.73m^2
- ~320 pts, 1:1 placebo randomization
- Global study at ~170 sites
- Primary endpoint: 24-week proteinuria (accelerated approval)
- Secondary endpoint: 2.5-year eGFR (full approval)

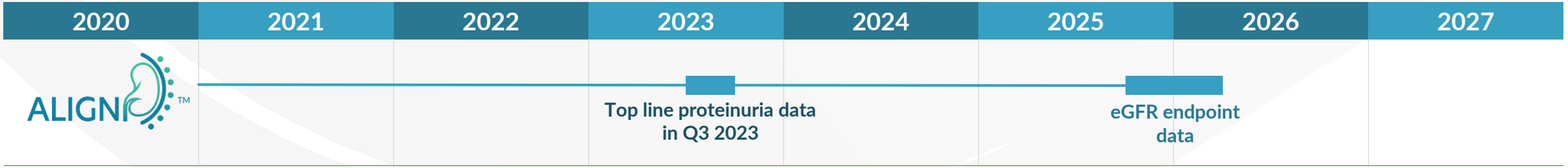
Two-Pronged Approach to Evaluate Atrasentan in Combination with SGLT2i

1 ALIGN™ SGLT2i + Atrasentan Combination Stratum Underway

- Stratum of up to 64 patients in ALIGN™ on stable dose of SGLT2i
- Safety and efficacy (proteinuria + eGFR) exploratory analysis with no change to enrollment timelines or primary analysis population

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

- Randomize patients 1:1 to atrasentan or placebo
- Primary endpoint: change in UPCR from baseline to week 12
- Data to support future use of atrasentan in combination with SGLT2is



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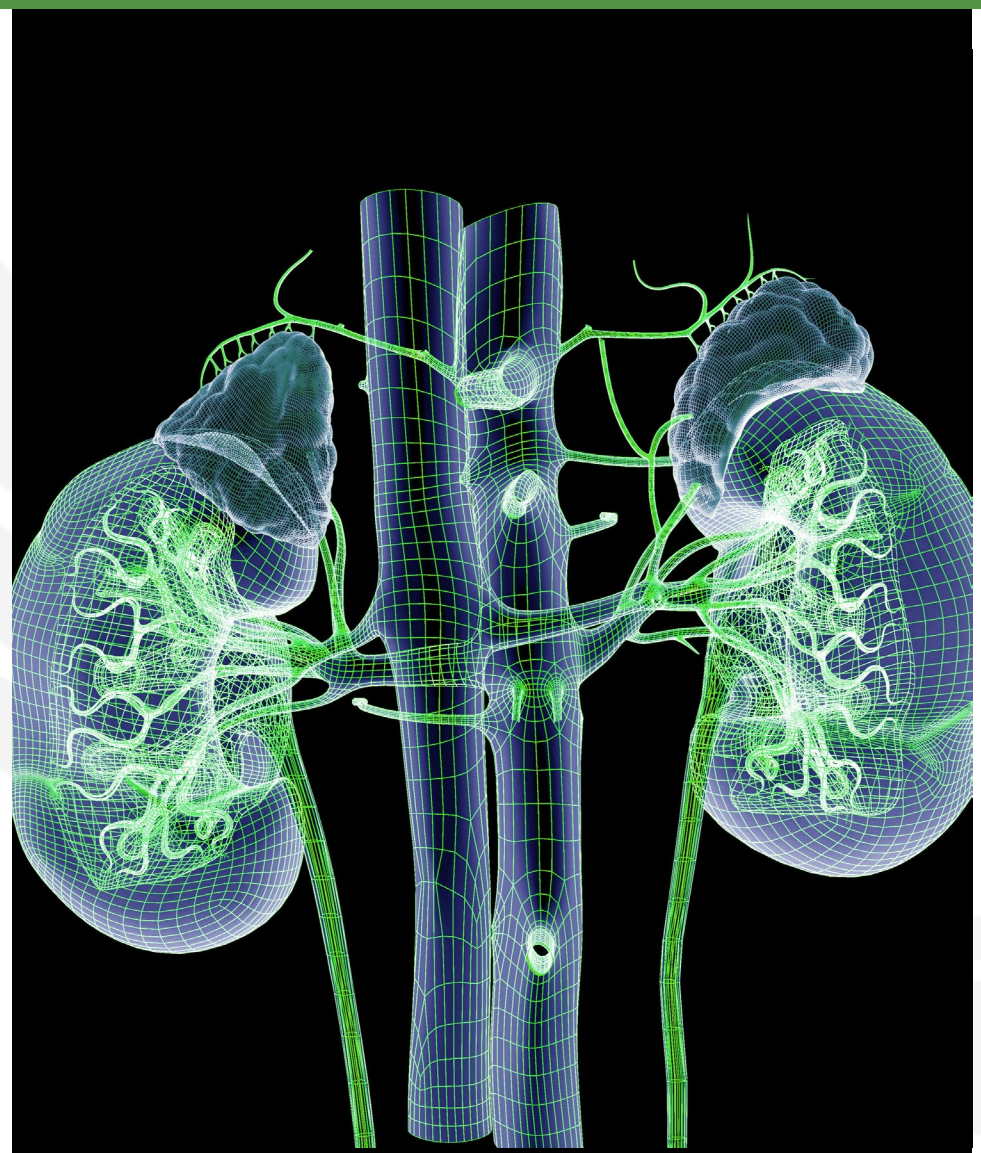

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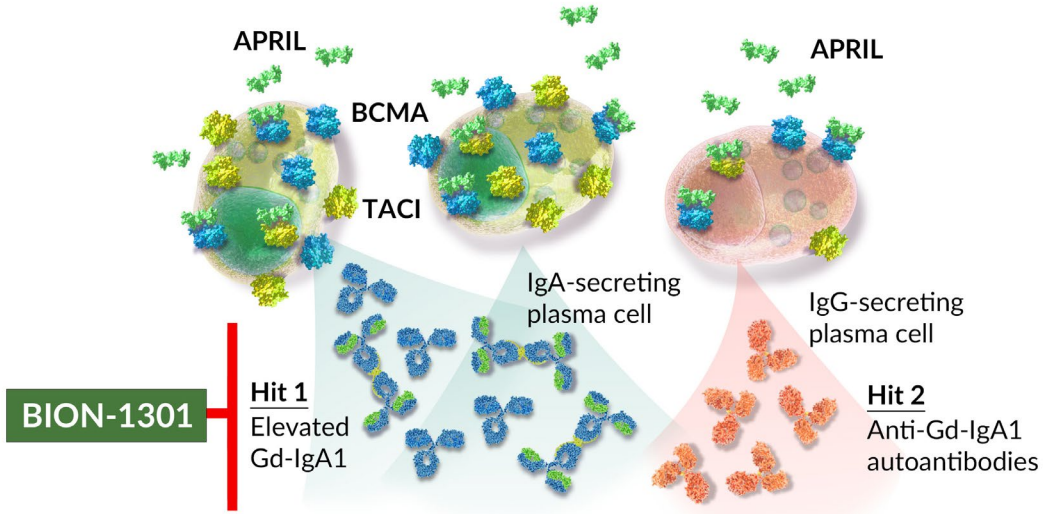
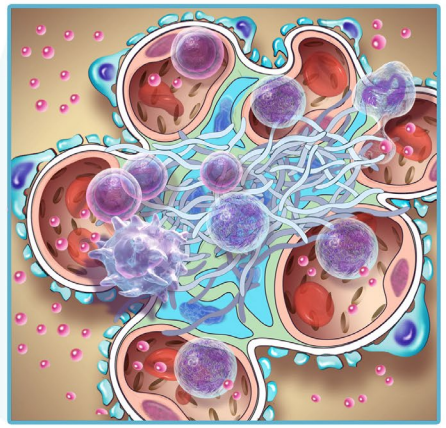
BION-1301: Potentially Disease-Modifying Anti-APRIL mAb in IgAN



Hit 3
Increased circulating Gd-IgA1 immune complexes

Hit 4
Immune complexes deposit in the mesangium, causing:
- Mesangial cell activation (cellular proliferation and excess secretion of extracellular matrix and cytokines)
- Cellular cross-talk resulting in podocyte dysfunction and proteinuria
- Ultimately tubular epithelial cell injury (atrophy, inflammation and fibrosis)



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

- Well-tolerated to date in:
  - ✓ NHPs[5]
  - ✓ Healthy volunteers[7]
  - ✓ Patients with multiple myeloma at doses up to 2700mg[6]
  - ✓ Patients with IgAN[8]
- **Demonstrated disease-modifying potential in patients with IgAN[8]**

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. 8. Barratt, J, et al., ASN Kidney Week 2022, FR-PO659.

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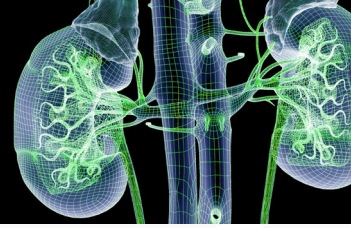
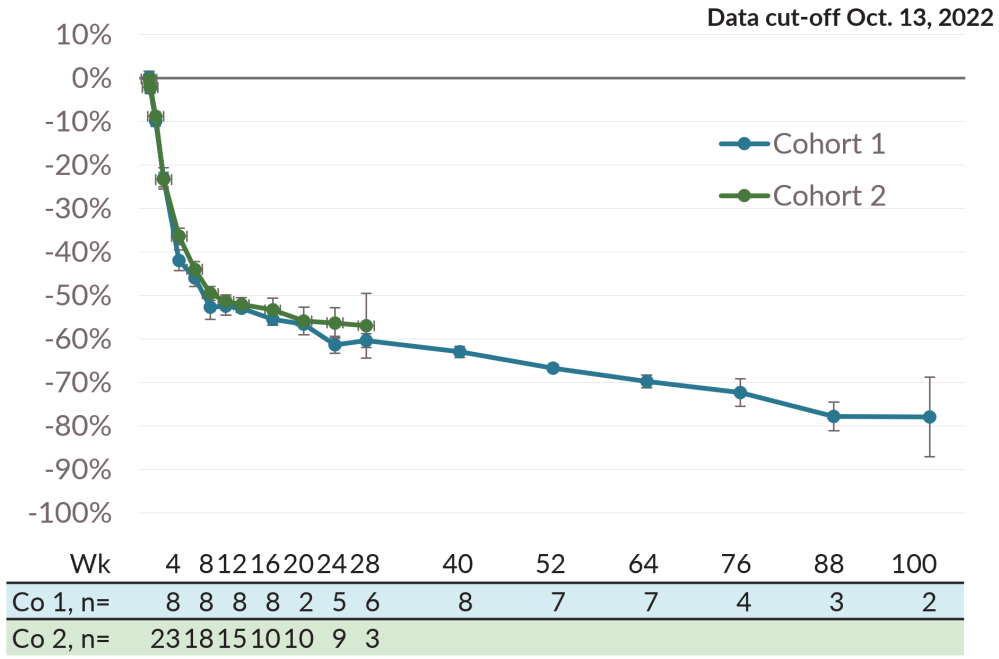
BION-1301 Results in Rapid and Durable Reductions in IgA and Gd-IgA1 in Patients with IgAN

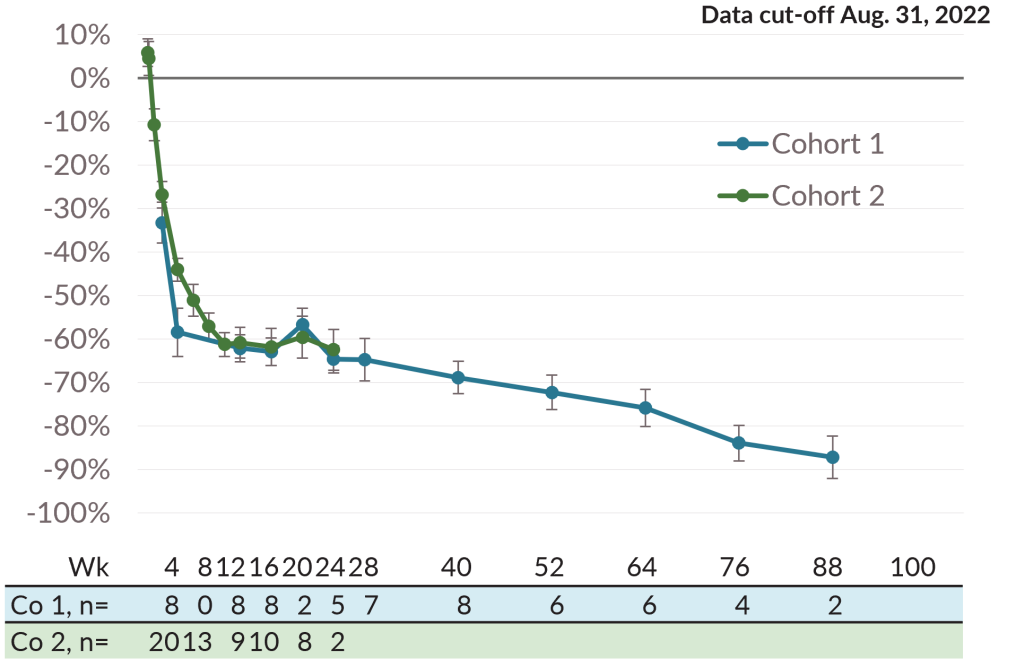


IgA % Change from Baseline (Mean ± SE)



Wk	4	8	12	16	20	24	28	40	52	64	76	88	100
Co 1, n=	8	8	8	8	2	5	6	8	7	7	4	3	2
Co 2, n=	23	18	15	10	10	9	3						

Gd-IgA1 % Change from Baseline (Mean ± SE)



Wk	4	8	12	16	20	24	28	40	52	64	76	88	100
Co 1, n=	8	0	8	8	2	5	7	8	6	6	4	2	
Co 2, n=	20	13	9	10	8	2							

Mean Gd-IgA1 are not available at week 100

- Reductions in IgM, and to a lesser extent IgG, also observed
- BION-1301 generally well-tolerated in IgAN patients, with no reported deaths, SAEs, or AEs leading to discontinuation of study drug to date
- No ADAs observed to date

Barratt, J, et al., ASN Kidney Week 2022, FR-PO659

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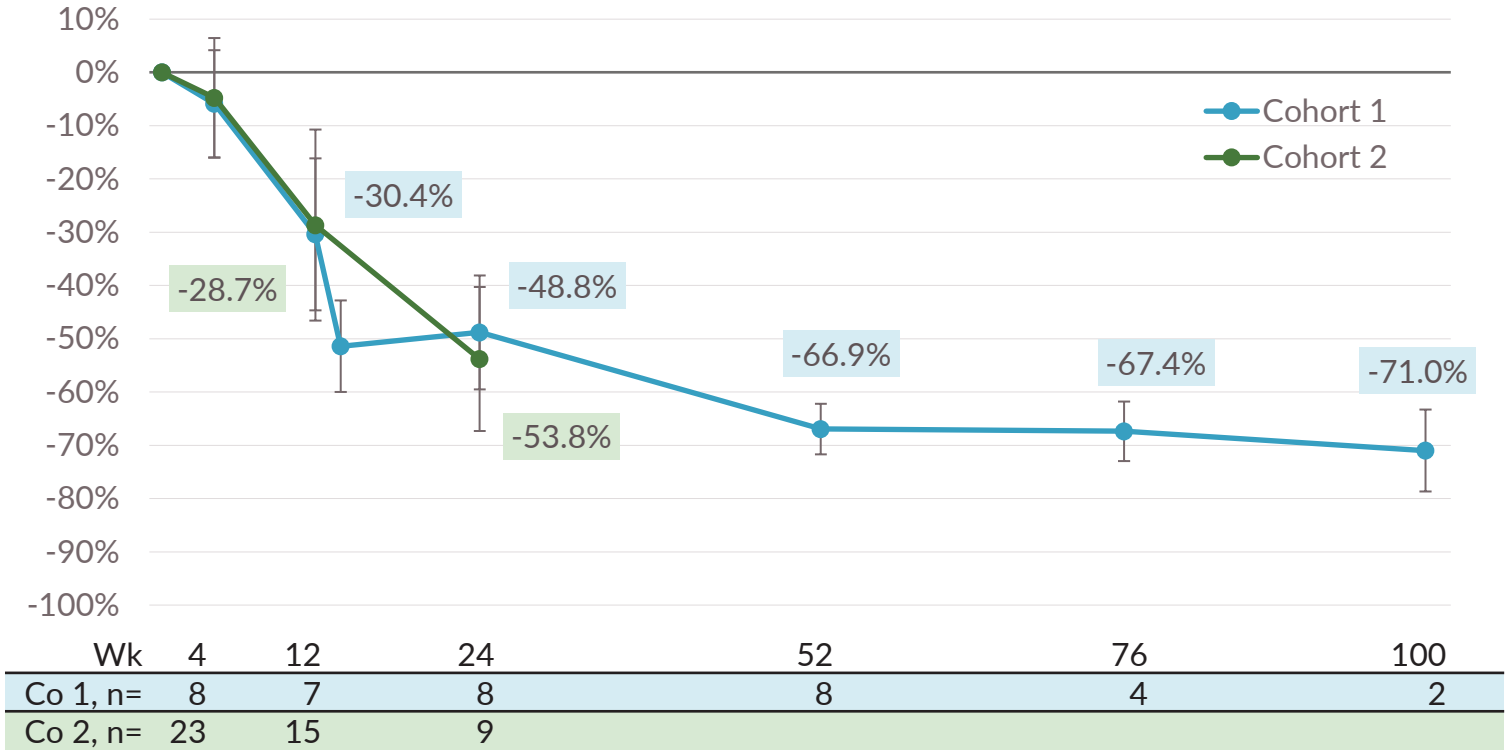
BION-1301 Treatment Results in Sustained, Clinically Meaningful Proteinuria Reductions in Patients with IgAN

UPCR % Reduction (Geometric Mean ± SE)

Data cut-off Oct. 13, 2022



Wk	4	12	24	52	76	100
Co 1, n=	8	7	8	8	4	2
Co 2, n=	23	15	9			

Median (min, max) baseline protein excretion: Cohort 1, 1.2 (0.7, 6.5) g/day ; Cohort 2, 1.0 (0.6, 2.7) g/day

COHORT 1 (IV → SC)

- Clinically meaningful reductions in UPCR were seen in patients with IgAN across a wide range of baseline proteinuria levels
- UPCR continued to decline through one year and was maintained through two years, providing evidence of sustained efficacy
- At Week 52, 7/8 evaluable patients demonstrated >50% reductions in UPCR

COHORT 2 (de novo SC)

- Mean reduction in UPCR of >50% at 24 weeks in Cohort 2 with de novo SC administration is consistent with Cohort 1

Barratt, J, et al., ASN Kidney Week 2022, FR-PO659

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BION-1301 MOVING FORWARD

Plan to advance cohort 2 dose/schedule in pivotal trial, given strong clinical data

STATUS

Cohort 1 in IgAN	**450 mg IV → 600 mg SC q2w** Enrollment of 10 Patients Completed
Cohort 2 in IgAN	**600 mg SC q2w** Enrollment of 30 Patients Completed

NEXT STEPS

- **Align with global health authorities *(ongoing)***
- **Conduct site and country feasibility *(ongoing)***
- **Initiate pivotal trial in mid-2023**

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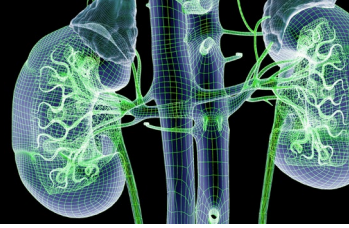
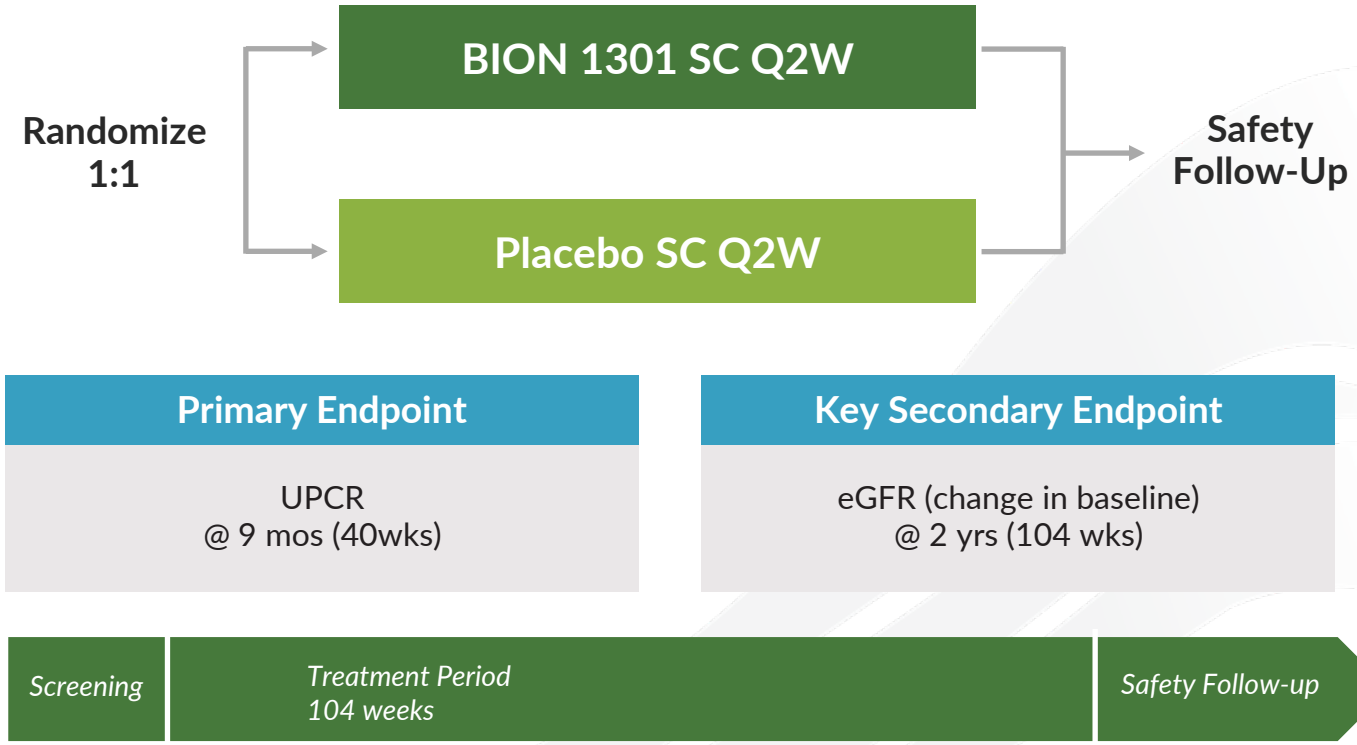
Proposed BION-1301 Phase 3 Trial Design

Initiate Pivotal Trial in Mid-2023, Given Strong Clinical Data and Disease-Modifying Potential



Planned Phase 3 Targeting IgAN Patients at Risk for Disease Progression

- Biopsy-proven IgAN
- Patients on maximally-tolerated, optimized and stable dose of RASi, or RASi intolerant
- Background optimized and stable dose of SGLT2i or ERA (if approved) allowed
- Proteinuria >1 g/day; eGFR >30 ml/min/1.73m2
- Global, double-blind, placebo-controlled
- 1:1 randomization
- Primary endpoint 9-month proteinuria (accelerated approval)
- Secondary endpoint 2-year eGFR (full approval)



Trial design to be finalized following interactions with global health authorities

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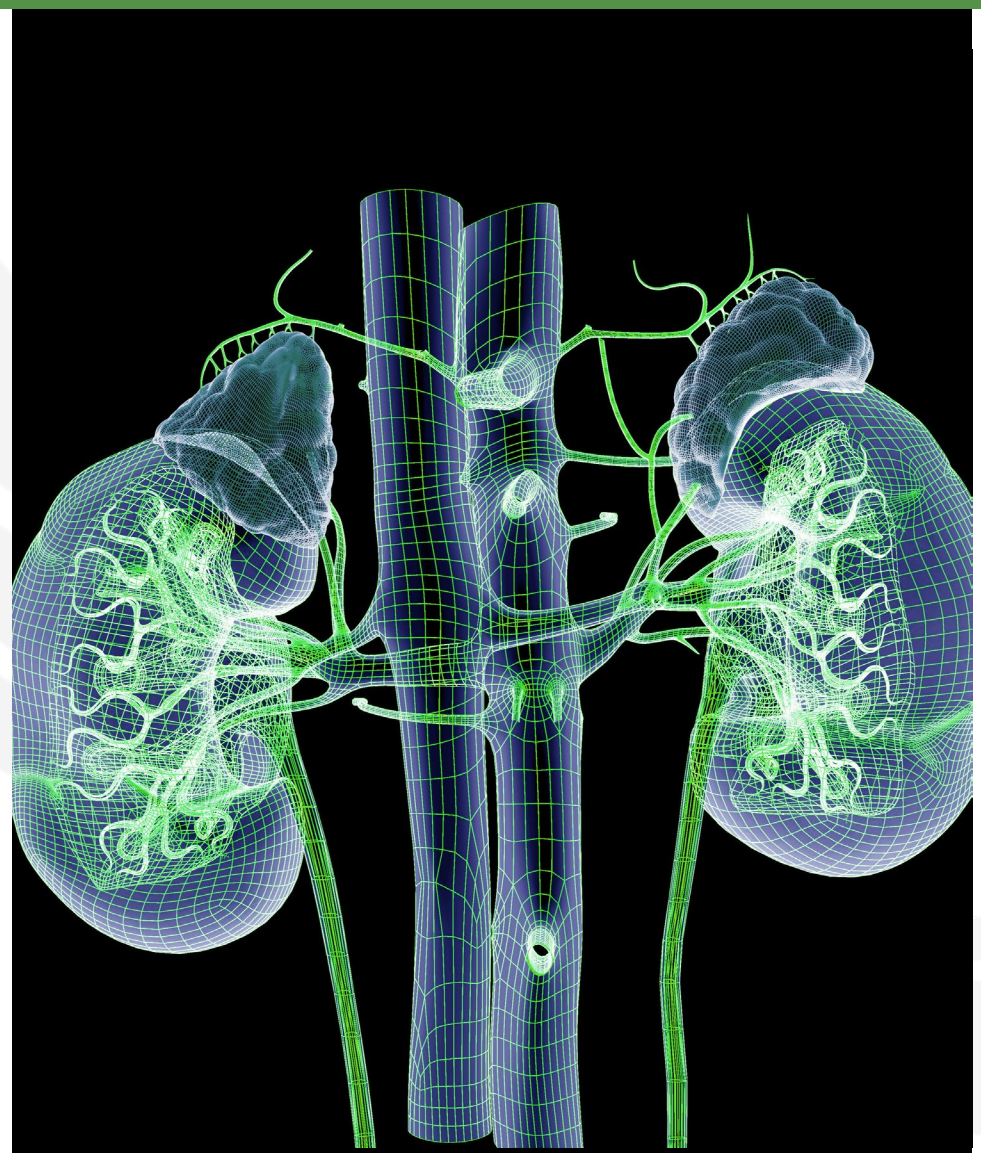

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Hyperoxalurias are Diseases Caused by Excess Oxalate

Hyperoxaluria is an Important Risk Factor for Kidney Stones

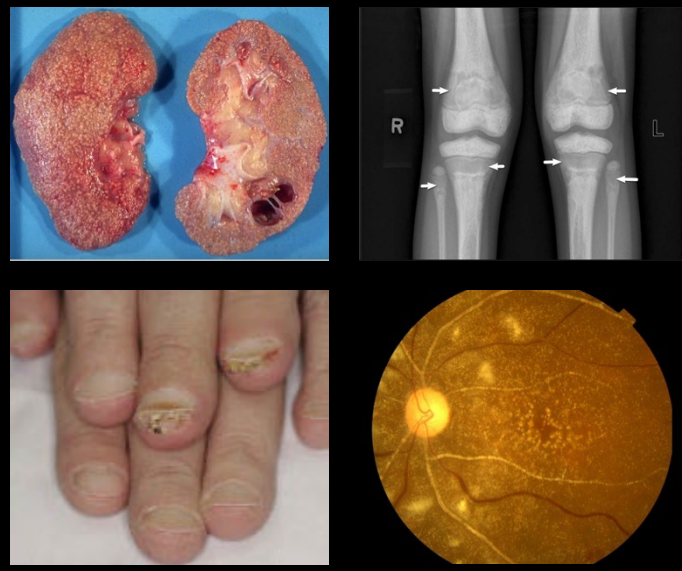
PRIMARY HYPEROXALURIAS (PH) 1-3 ARE ULTRA-RARE DISEASES

- Caused by genetic mutations resulting in overproduction of oxalate by the liver
- PH leads to recurrent kidney stones and can lead to kidney failure, if left untreated
- Median age of kidney failure for most severe form of disease (PH1) is 23 years[1]
- ~5,000 – 7,000 PH1 patients in the US and Europe[2]

IDIOPATHIC HYPEROXALURIA IS MORE COMMON

- May result from increased endogenous oxalate overproduction, particularly in association with metabolic diseases[3]
- Hyperoxaluria (generally defined as urinary excretion of >40 mg/d) is present in ~20–40% of frequent stone formers

Decline in kidney function results in systemic oxalosis, affecting multiple organs[4-7]



1. Bergstrahl et al, Am J Transpl 2010 10, 2493-2501 2. Hopp et al , J Am Soc Nephrol 2015 26(10): 2559-70 3. Gianmoena et al, Cell Rep 2021 36(8): 109526 4-7 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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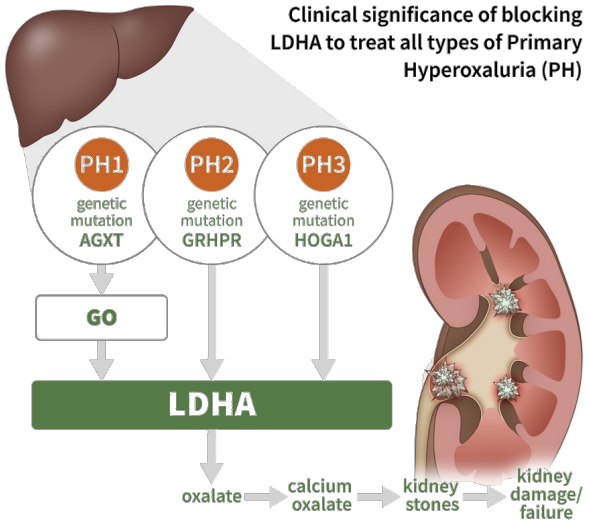
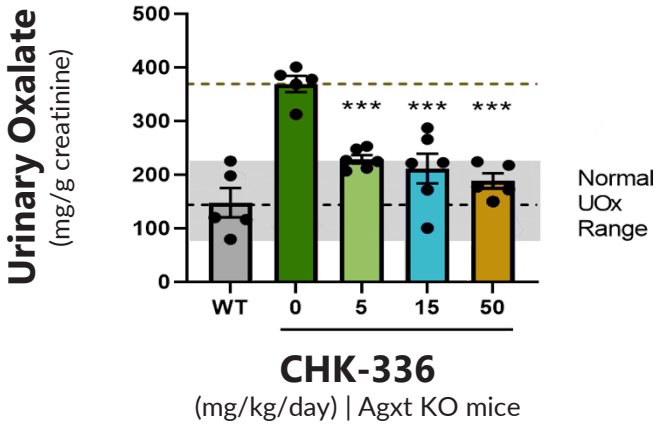
CHK-336: Oral Small Molecule LDHA Inhibitor for PH

Liver-Targeted Tissue Distribution Profile Enables Potential to Treat All PH Types



EFFICACY IN MOUSE PH1 MODEL



- Lactate dehydrogenase (LDHA) is the final step in production of oxalate from glyoxylate (GO) in the liver[1]
- Targeting LDHA may address all types of PH
- CHK-336 produces significant and dose-dependent urinary oxalate reductions in PH1 mouse models[2]
- Significantly reduces urinary oxalate excretion in a PH2 mouse model[3]

- Ongoing phase 1 SAD/MAD study evaluating safety, tolerability and PK of CHK-336 in healthy volunteers
- **Anticipate reporting phase 1 healthy volunteer data in H1 2023**
- Advancing towards initiation of phase 2 POC trials for patients with primary and idiopathic hyperoxalurias

1. Kanno et al Clinica Chimica Acta 1980; 108: 267-276
2. Cox et al., ASN Kidney Week 2020, PO1620; 3. Cox et al., ASN Kidney Week 2022, FR-PO334

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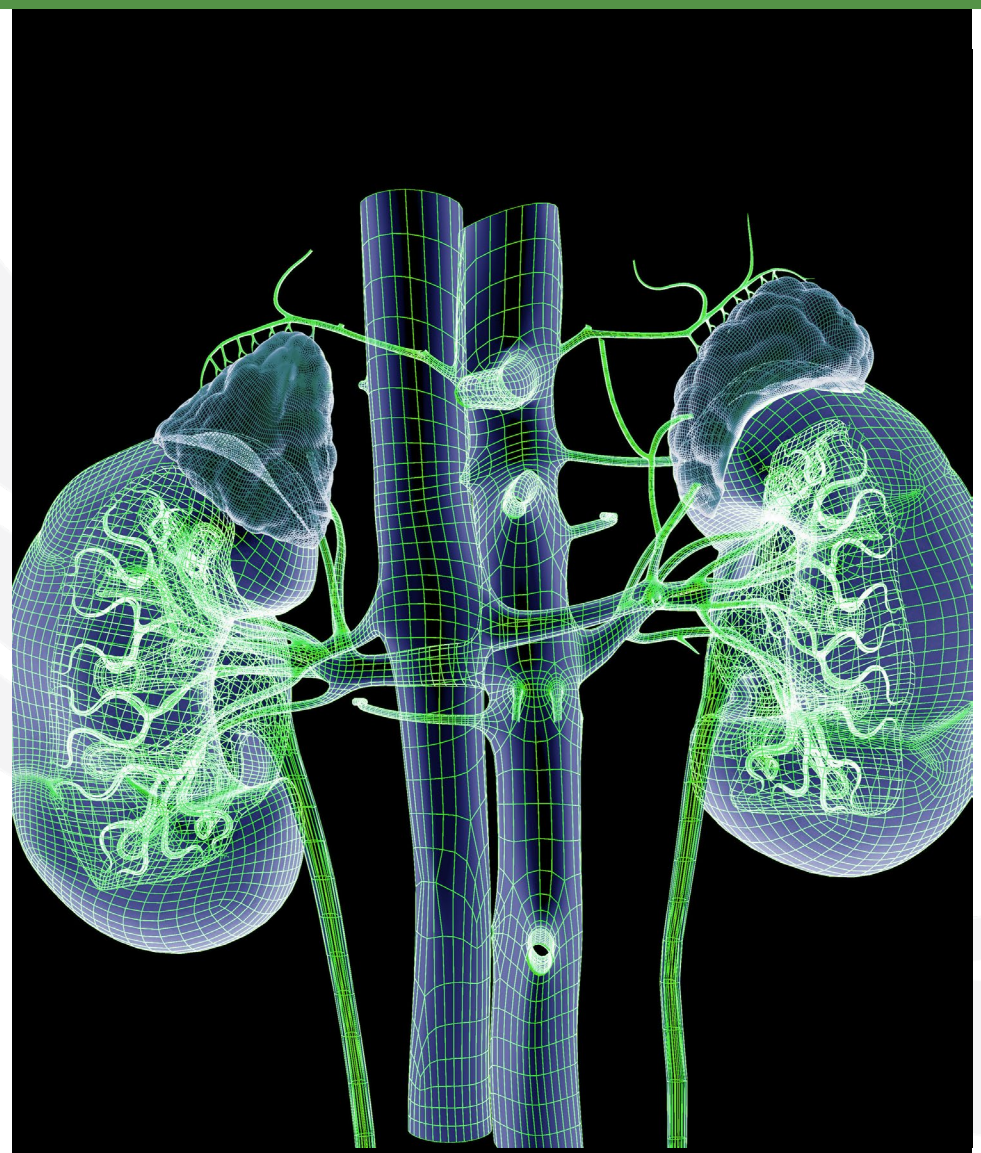


ATRASENTAN

ET_A Antagonist

BION-1301

Anti-APRIL Monoclonal Antibody

CHK-336

LDHA Inhibitor

R&D PROGRAMS

Precision Medicine Pipeline

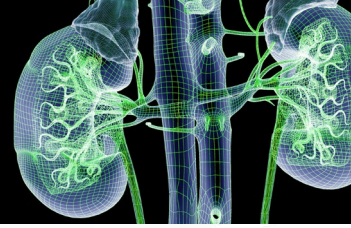
FINANCIALS & CATALYSTS

2023

CHINOOK™ THERAPEUTICS

Precision Medicine Approach to Research & Discovery

Focused on Rare, Severe CKDs with Defined Genetic and Molecular Drivers



TARGET SELECTION & VALIDATION

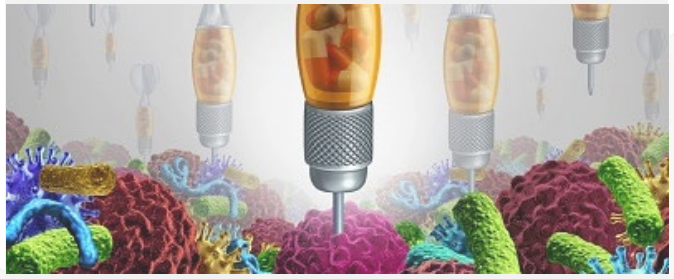
Systems Biology

Molecular Classification of CKD

- Target ID
- Target Validation
- Patient Stratification

evotec NURTURE

Translational Models



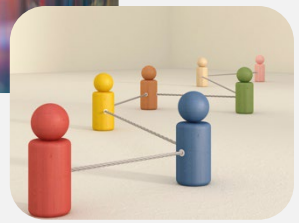
Modeling Human Disease

- Disease Mechanisms
- Target Validation
- Deep Biological Insights

TARGET EXECUTION

Development Candidates

Drug Discovery
+ Partnerships



Growing Pipeline

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

Detailed insights into molecular pathogenesis of stratified CKDs

Novel & differentiated molecules

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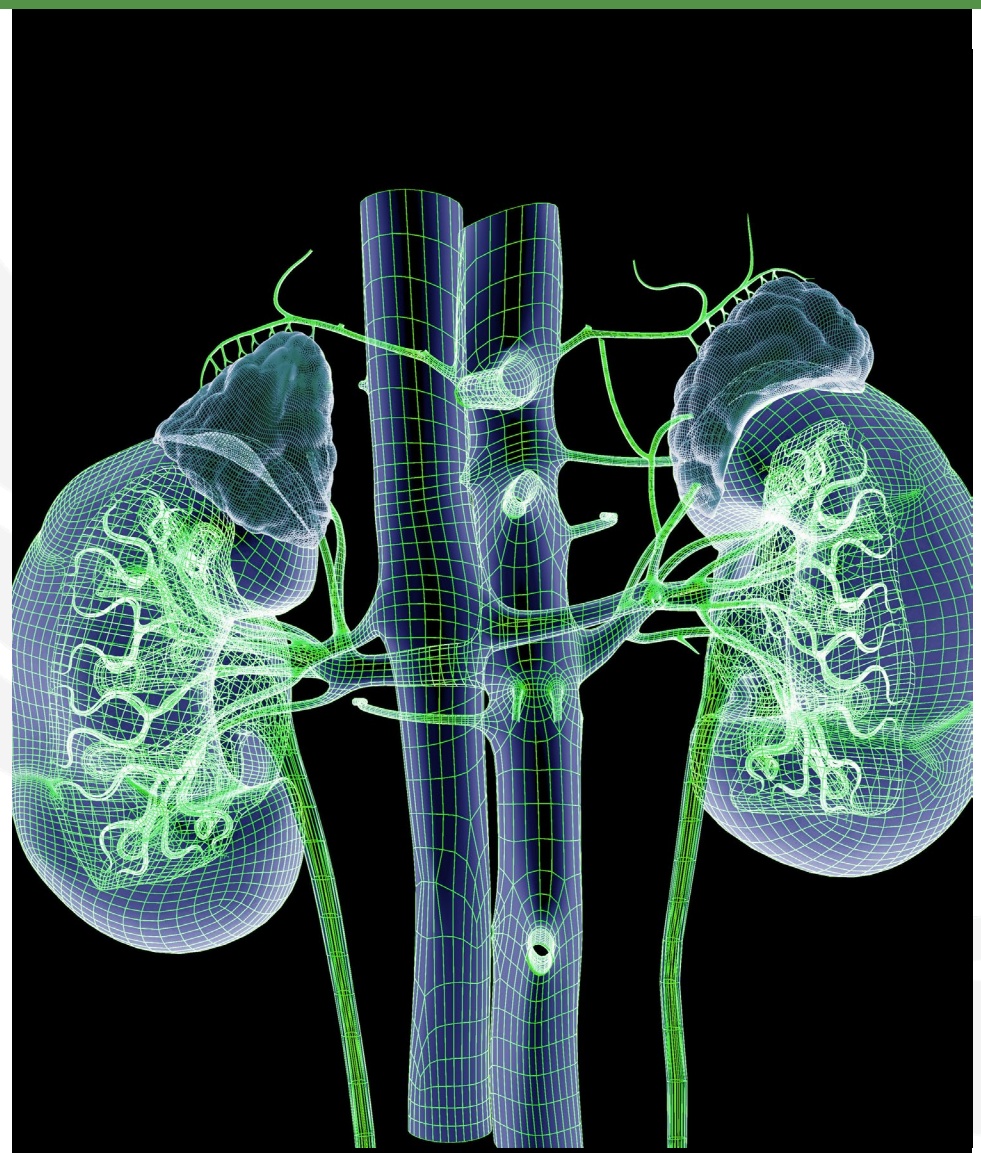

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2023

CHINOOK™ THERAPEUTICS

FINANCIAL STRENGTH

NASDAQ: **KDNY**

STRONG BALANCE SHEET

$397.7M

in cash, cash equivalents and marketable securities as of September 30, 2022

COMMON STOCK OUTSTANDING

~68.6 M

shares as of November 4, 2022*

~72.8 M

fully diluted shares as of November 10, 2022**

CASH GUIDANCE

Operating capital into 2025 based on current business plan

SANRENO JOINT VENTURE IN CHINA

- **50:50 JV** for atrasentan and BION-1301
- Provides local execution and enhanced access to large IgAN patient populations in Asia
- Financial upside through equity ownership, milestones and royalties
- Reciprocal rights of first negotiation for future developed or in-licensed products

*Includes 4.6M pre-funded warrants

**Treasury method. Includes 7.0 million options with average exercise price of $13.46 and 1.4 million RSUs outstanding as of 9/30/22

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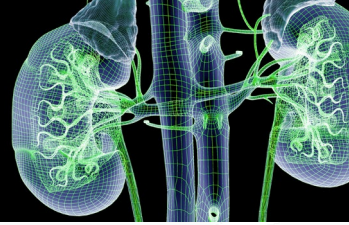
Catalysts



Program	Indication	Catalyst	H1 2023	H2 2023
Atrasentan	*IgA Nephropathy*	Initiate phase 2 trial in combination with SGLT2i in IgAN	●	
		Report topline proteinuria data from ALIGN in Q3 2023		●
	Glomerular Diseases	Present additional data from other AFFINITY cohorts		●
BION-1301	*IgA Nephropathy*	Present additional phase 1/2 data from Cohorts 1 and 2 in IgAN	●	●
		Initiate phase 3 trial in IgAN in mid-2023	●	
CHK-336	*Hyperoxalurias*	Report phase 1 healthy volunteer data and advance towards initiation of phase 2 POC trials in primary and idiopathic hyperoxalurias	●	●

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