CHINOOK THERAPEUTICS

Chinook Therapeutics

Developing Precision Medicines for Kidney Diseases

ASN Kidney Week 2020 Reimagined
Investor Webcast & Conference Call
October 22, 2020

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Today's Presenters

Eric Dobmeier

President & CEO

Richard Lafayette, MD, FACP

Associate Professor of Medicine (Nephrology) & Director of the Stanford Glomerular Disease Center at Stanford University Medical Center

Alan Glicklich, MD

Chief Medical Officer

Andrew King, PhD

Head of Renal Discovery & Translational Medicine

Agenda

Introduction		Eric Dobmeier
Overview of IgA Nephropathy & Treatment Options		Richard Lafayette, MD, FACP
INFO29	Phase 3, Randomized, Double-Blind, Placebo-Controlled Study of **Atrasentan** in Patients with IgA Nephropathy (The ALIGN Study)	Alan Glicklich, MD
PO1843	Results of a Phase 1 Trial to Investigate the Safety, Tolerability, Pharmacokinetics, and Pharmacodynamics of **BION-1301** in Healthy Volunteers	Andrew King, DVM, PhD
PO1620	Discovery of **CHK-336**: A First-in-Class, Liver-Targeted, Small Molecule Inhibitor of Lactate Dehydrogenase for the Treatment of Primary Hyperoxaluria	
SA-OR21	Single-Cell Transcriptomic Analysis to Define Cellular Heterogeneity in Human ADPKD	
Closing		Eric Dobmeier
Q&A		

CHINOOK THERAPEUTICS

Introduction to Chinook

Advancing a pipeline of precision medicines for kidney diseases

Building the Leading Company Developing Precision Medicines for Kidney Disease

2019
- Founded Chinook
- Announced $65M Series A financing
- In-licensed atrasentan from AbbVie

2020
- Preparing to initiate phase 3 and phase 2 basket trials for atrasentan
- Conducting IND-enabling studies of CHK-336 for primary hyperoxaluria
- Completed merger with Aduro Biotech and $115M financing
- Began trading on Nasdaq as “KDNY” with >$275M in operating capital

2021
- Initiate phase 2 and 3 trials for atrasentan
- Report interim phase 1b patient data from BION-1301
- Initiate phase 1 trial for CHK-336
- Progress multiple preclinical programs
- Build partnerships (regional, strategic)

Long-Term Vision

Kidney Disease Industry Leader

- Commercial and clinical validation
- Discovery engine generating new product opportunities
- Strategic partner of choice across kidney indications

Guiding R&D Principles:	Focus on key pathways in kidney disease	Design novel, target and differentiated molecules	Utilize new and efficient translational approaches to speed R&D	Execute clinical trials in defined patient populations with surrogate endpoints

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Robust, Diversified and Focused Pipeline

Advancing pipeline of precision medicines for kidney diseases

Program	Indication	Target Validation	Lead Optimization	IND-Enabling	Phase 1	Phase 2	Phase 3
Atrasentan	IgA Nephropathy	Phase 3 initiation planned in early 2021					
	Basket of glomerular diseases	Phase 2 initiation planned in H1 2021					
BION-1301	IgA Nephropathy	Phase 1b ongoing					
CHK-336	Primary Hyperoxaluria	Phase 1 initiation planned in H2 2021					
Research Programs	Other rare, severe chronic kidney diseases including ADPKD						
Discovery Programs	Other rare, severe kidney diseases						

We will continue to evaluate opportunities to add additional kidney disease programs to pipeline

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IgA Nephropathy (IgAN)

Chronic autoimmune kidney disease with no approved therapies

Immunoglobulin A (IgA) Nephropathy (IgAN)

A Progressive, Chronic Glomerular Disease with Limited Treatment Options

IgAN is the most common primary glomerular disease globally, but still classified as an orphan indication with ~140K patients in the US

- Variable clinical presentation, ranging from asymptomatic microscopic hematuria to severe, rapidly progressive glomerulonephritis

Diagnosis requires kidney biopsy and immunofluorescence microscopy

- Dominant mesangial IgA deposits and other hallmark histological hallmarks

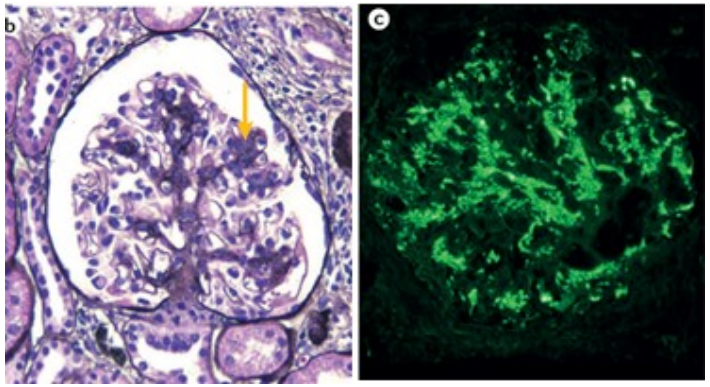
Approximately 30-45% of IgAN patients will develop end-stage kidney disease (ESKD) over a period of 20-25 years

- ESKD requires chronic dialysis or transplantation for management

No approved treatments and limited options for high-risk patients

- Renin Angiotensin System inhibition (RASi) (ACEi/ARB) is frontline (KDIGO 1B)
- Immunosuppressive agents provide inconsistent therapeutic benefit and are accompanied by significant side effects (KDIGO 2C)

Histological Diagnosis



Mesangial hypercellularity

Glomerular IgA deposits

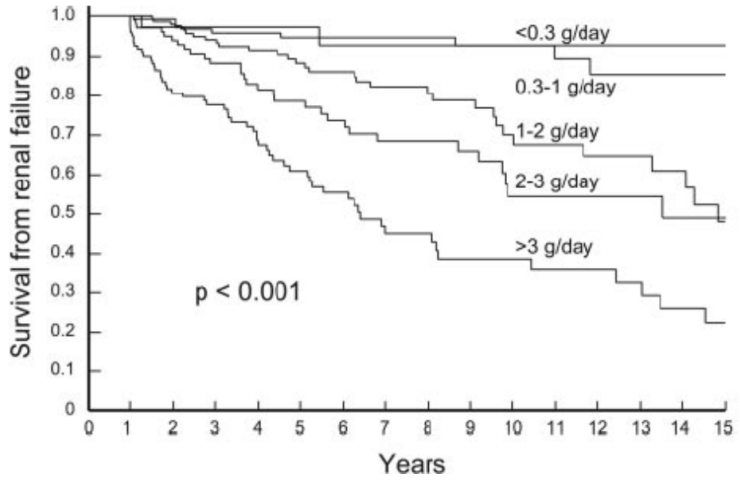
Urgent need for new treatments, in this typically young and otherwise healthy IgAN patient population at high risk for progressive kidney function loss

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

Proteinuria Identifies IgAN Patients at Risk of Progression

Proteinuria Recognized as a Surrogate Endpoint for Accelerated Approval

High proteinuria levels are the most important predictor of kidney progression in IgAN



CJASN

KHI KIDNEY HEALTH INITIATIVE FDA ASN American Society of Nephrology

Proteinuria Reduction as a Surrogate End Point in Trials of IgA Nephropathy

Aliza Thompson,[1] Kevin Carroll,[2] Lesley A. Inker,[3] Jürgen Floege,[4] Vlado Perkovic,[5] Sonia Boyer-Suavet,[6] Rupert W. Major,[7] Judith I. Schimpf,[4] Jonathan Barratt,[8] Daniel C. Cattran,[9] Barbara S. Gillespie,[10] Annamaria Kausz,[11] Alex W. Mercer,[12] Heather N. Reich,[9] Brad H. Rovin,[13] Melissa West,[14] and Patrick H. Nachman[15]

Kidney progression with proteinuria **>1 g/day**

Sustained lowering of proteinuria to **<1 g/day** is associated with favorable **long-term prognosis**

Proteinuria reduction is anticipated to be the **surrogate marker to obtain accelerated approval in IgAN**

Clin J Am Soc Nephrol;14(3):469-481, 2019.

Multi-Hit IgAN Pathogenesis

An Immune-mediated Primary Glomerular Disease

Initiating event thought to be an aberrant mucosal immune response

- IgAN commonly presents following a respiratory or gastrointestinal infection

Hit 1

- Increased production of Gd-IgA1 by IgA secreting plasma cells

Hit 2

- Gd-IgA1 is recognized as an autoantigen, by circulating IgG antiglycan antibodies

Hit 3

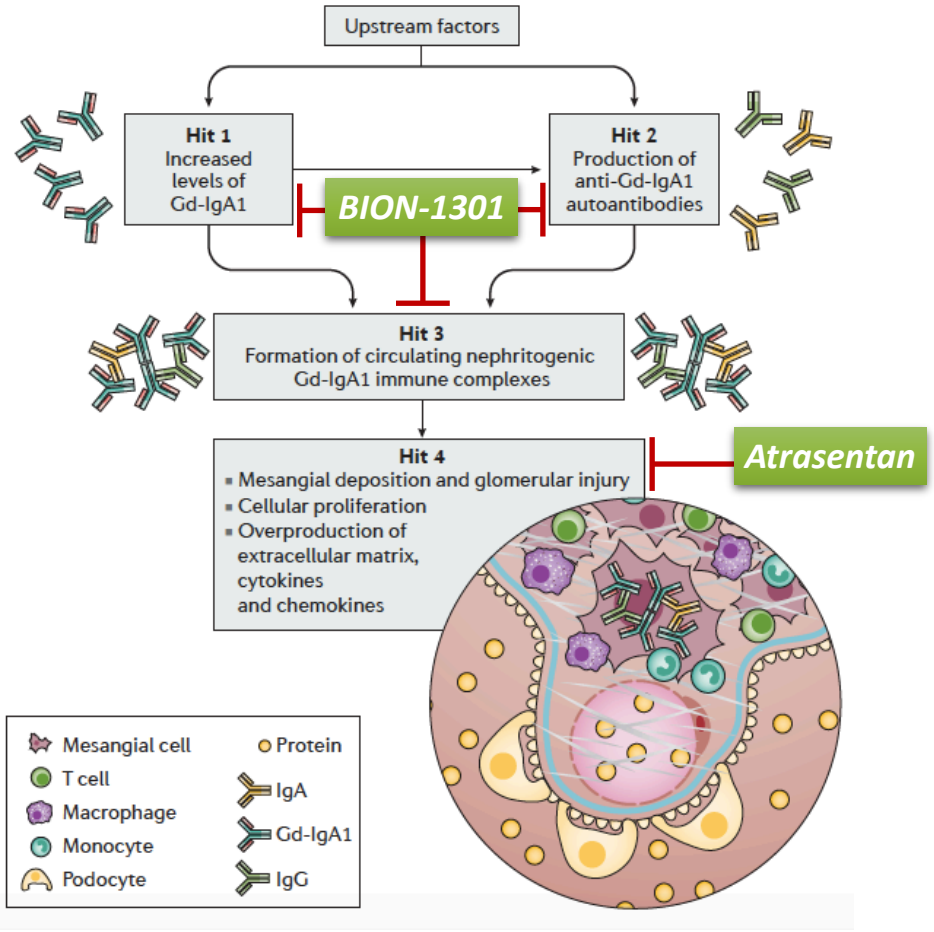
- Immune recognition results in the formation of immune complexes (ICs)

Hit 4

- Immune complexes deposit in the kidney, activating glomerular mesangial cell proliferation and production of extracellular matrix and inflammatory mediators
- Cellular crosstalk results in proteinuria, tubulointerstitial inflammation and fibrosis, leading to progressive kidney function loss

New treatments are needed to target the formation of IgA ICs (Hits 1-3) and the kidney's pathogenic response to deposition of ICs (Hit 4)

Atrasentan & BION-1301 target distinct and complementary mechanisms of IgAN



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

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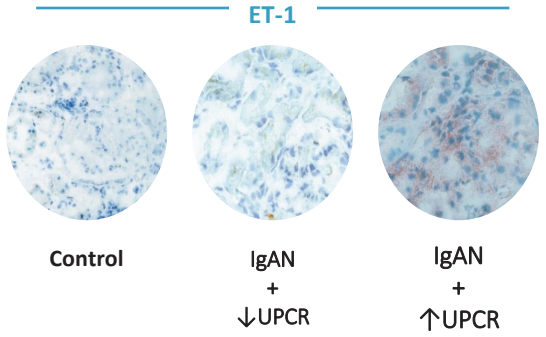
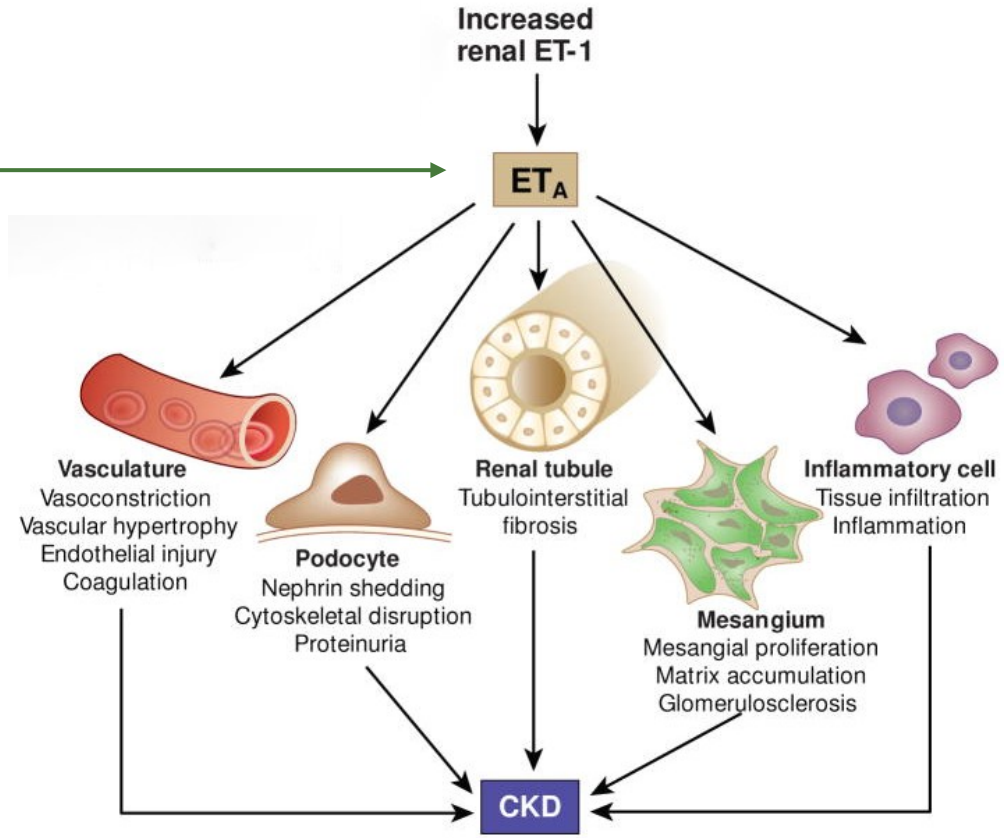
Atrasentan: A Potent and Selective ET_A antagonist

ET_A Activation Drives IgAN Progression Through Multiple Potential Mechanisms (Hit 4)

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

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





Proteinuria
Inflammation & Fibrosis

ET system activation appears to be a key molecular determinant of the clinical course in IgAN

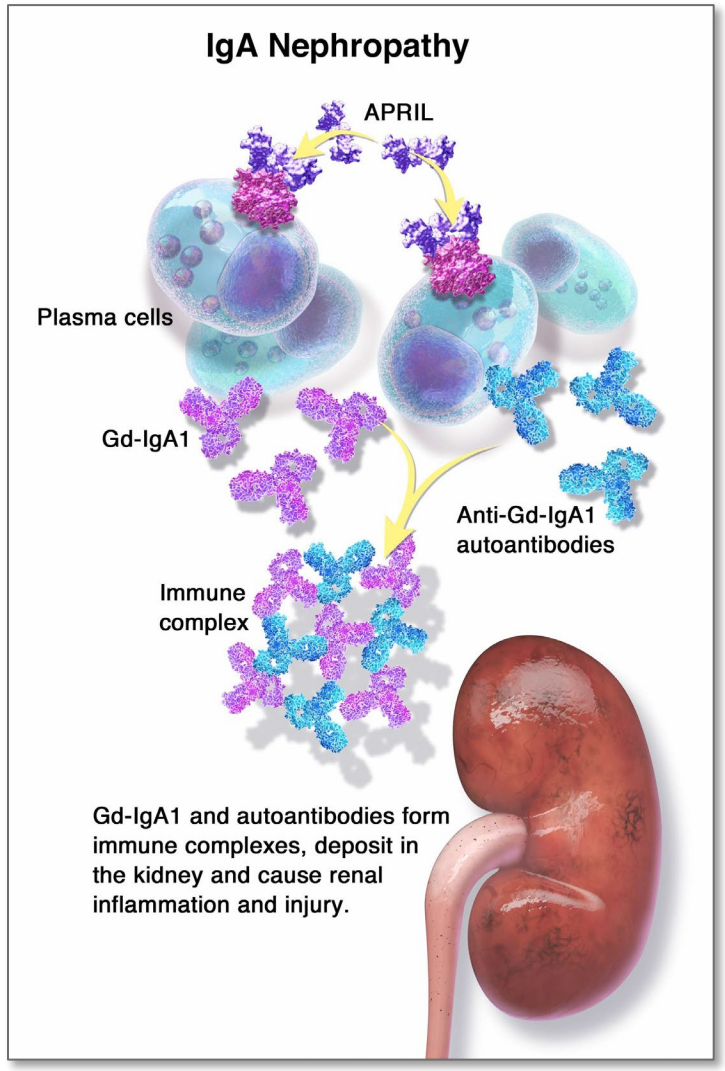
Elevated kidney ET-1 expression strongly & prospectively predicted progression of IgAN, 12 months following kidney biopsy

Blockade of the ET_A receptor through atrasentan represents a promising approach to treat IgAN patients at high risk of progression (Hit 4)

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

BION-1301: A Humanized Anti-APRIL Monoclonal Antibody

A Potentially Disease Modifying Approach to Reduce IgA Immune Complex Formation (Hits 1-3)



A proliferation-**i**nducing **l**igand (**APRIL**), is a cytokine that promotes IgA class-switching and survival of IgA-producing plasma cells, by activating its receptors, TACI & BCMA, and has been shown to increase Gd-IgA1 secretion [1]

In a study of IgAN patients, high plasma APRIL levels associated with higher Gd-IgA1 and proteinuria and lower estimated glomerular filtration rates [2]

BION-1301 is a humanized IgG4 monoclonal antibody, that binds APRIL and functionally blocks APRIL binding to both of its receptors, BCMA and TACI

Blocking APRIL is a distinct approach to address the underlying cause of IgAN by reducing circulating levels of IgA, Gd-IgA1, anti-Gd-IgA1 autoantibodies and immune complex formation (Hits 1-3)

1. He B, et al. Nat Immunol. 2010; 2. Zhai YL, et al. Medicine (Baltimore). 2016.

CHINOOK THERAPEUTICS

INFO29

Phase 3, Randomized, Double-Blind, Placebo-Controlled Study of Atrasentan in Patients with IgA Nephropathy (The ALIGN Study)

Hiddo J. L Heerspink[1], Donald E. Kohan[2], Richard A. Lafayette[3], Adeera Levin[4], Vlado Perkovic[5], Hong Zhang[6], Andrew J. King[7], Alan Glicklich[7], Jonathan Barratt[8]

1. University Medical Center Groningen, Groningen, Netherlands. 2. The University of British Columbia Faculty of Medicine, Vancouver, BC, Canada. 3. University of Utah Health, Salt Lake City, UT, United States. 4. Chinook Therapeutics, Seattle, WA, United States. 5. Peking University First Hospital, Beijing, Beijing, China. 6. Stanford Medicine, Stanford, CA, United States. 7. University of New South Wales Faculty of Medicine, Sydney, NSW, Australia. 8. University of Leicester Medical School, Leicester, Leicester, United Kingdom.

Atrasentan is an Investigational Potent, Highly-Selective ET_A Inhibitor with Extensive Clinical Data in CKD

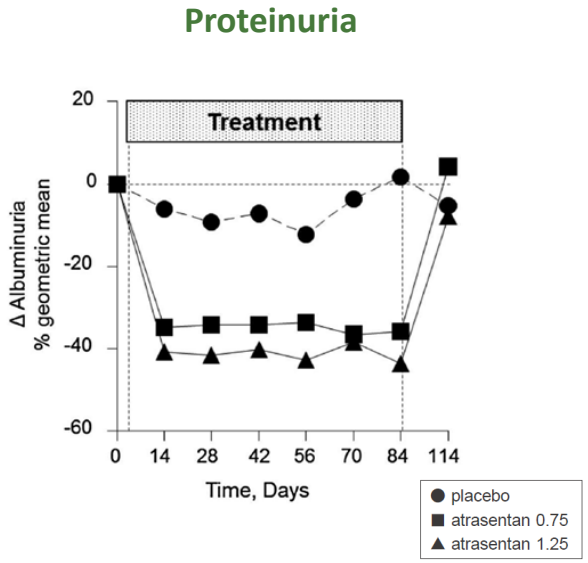
Potential to benefit IgA Nephropathy patients with a rapid registration pathway

Previously developed by AbbVie

- Extensive clinical data in **>5,300 diabetic kidney disease (DKD) patients,** including improved clinical outcomes in the global SONAR phase 3 study
- **Rapid/sustained proteinuria reduction** and **preserved kidney function** observed in clinical trials
- **Well-characterized safety profile** (dosed up to 5 years in trials)
- **Optimal dose of 0.75 mg daily** established via detailed PK / PD modelling
- **Picomolar potency** and **highly selective for ET_A** (1860-fold vs. ET_B)

Strong rationale for development in IgAN

- Potential to seek **accelerated approval** on proteinuria reduction as surrogate endpoint
- Patient population that is otherwise **younger** and **healthier** than DKD
- **Endorsement from KOLs** supporting the potential of ET_A inhibition in IgAN
- **Exclusivity period** based on IP and potential for orphan designation
- Unmet medical need creates **strong commercial opportunity**



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

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

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AbbVie Global SONAR Phase 3 Outcome Trial in DKD

SONAR Topline Results

3600 3,600 high-risk DKD patients randomized

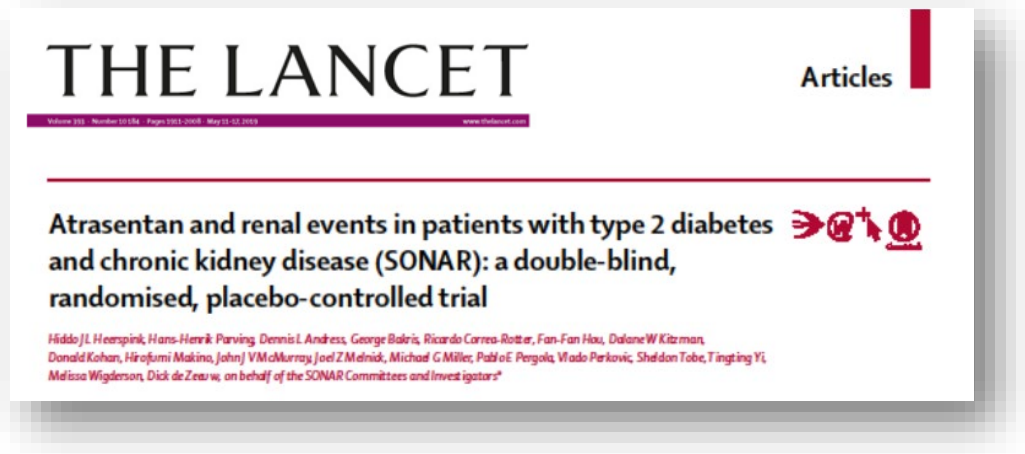
2 2-year median treatment duration

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

27% 27% risk reduction of End Stage Renal Disease

39% 39% risk reduction of doubling of serum creatinine

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


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, Pablo 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% reduction in proteinuria

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)

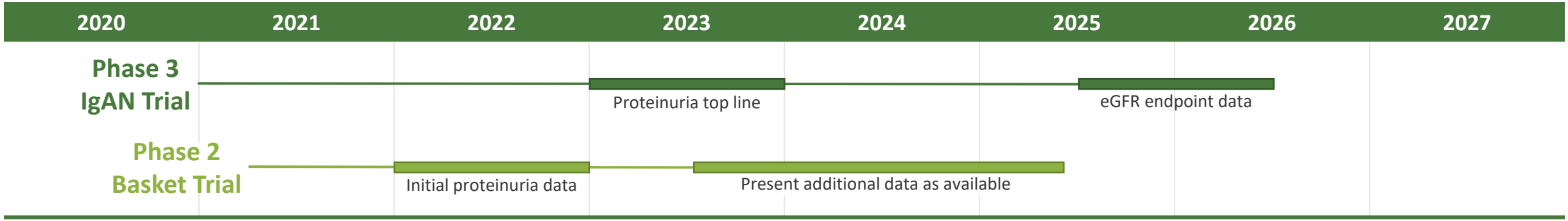
Phase 2 Basket Trial to Expand Potential Across Multiple Indications

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 to SGLT2 inhibitors



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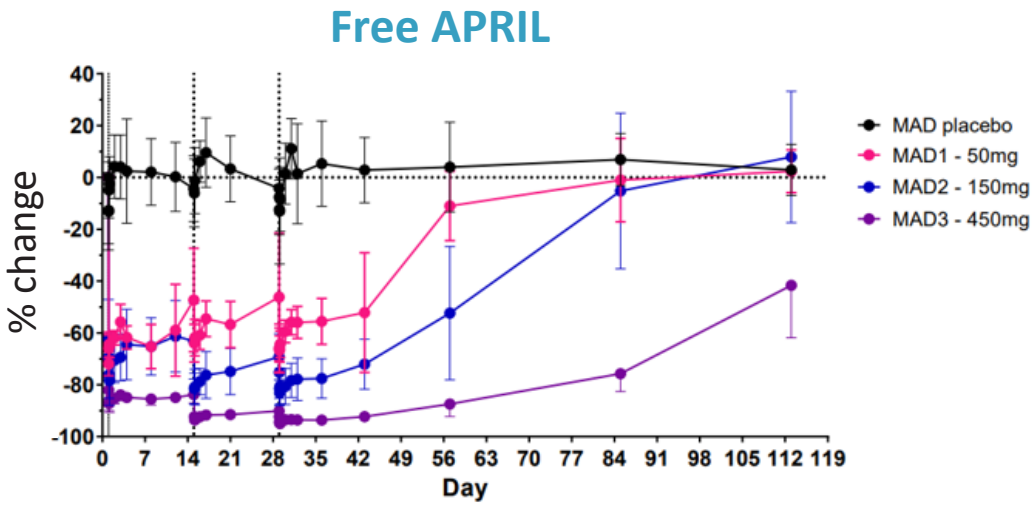
PO1843

Results of a Phase 1 Trial to Investigate the Safety, Tolerability, Pharmacokinetics, and Pharmacodynamics of BION-1301 in Healthy Volunteers (encore)

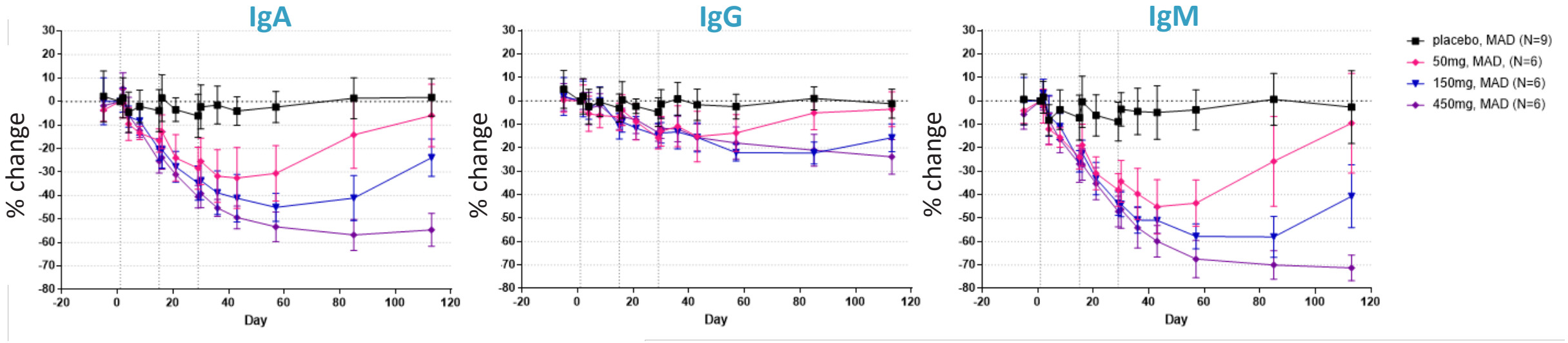
Jeannette Lo,[1] Sharon Yavrom,[1] Jessy Fan,[1] Aaron N. Endsley,[2] Tamara Schroeder,[1] Jonathan Barratt,[4] David M. Essayan.[3]

1. Chinook Therapeutics, Inc., Berkeley, CA;
2. Certara LP, Princeton, NJ;
3. ONCORD, Inc., Westlake Village, CA;
4. University of Leicester, Leicester, United Kingdom.

BION-1301 Demonstrated IgA Reductions in a Phase 1 Healthy Volunteer Study



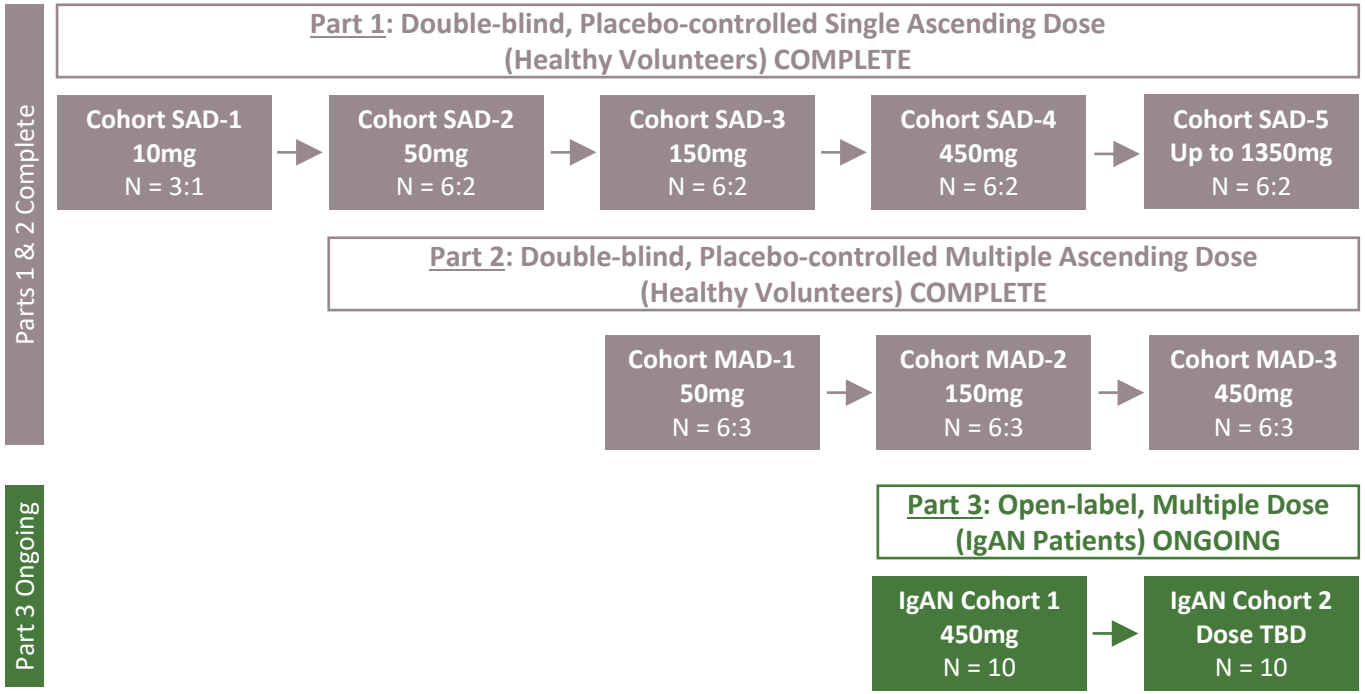
- BION-1301 was well-tolerated with low incidence of non-neutralizing ADAs reported
- Dose-dependent and durable reductions in free APRIL, IgA, IgM, and to a lesser extent, IgG
- Target of **~50-60% IgA reduction** achieved with 150-450 mg IV q2w
- PK profile was well-behaved, generally dose-proportional and demonstrated a half-life that supports the potential to be administered by monthly dosing
- Some accumulation observed, leading to greater & sustained PD responses with repeat dosing



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

BION-1301 Phase 1 in IgAN Patients Ongoing

- Part 3 currently enrolling IgAN patients
- BION-1301 administered by IV infusion every 2-4 weeks for 12 weeks
- Patients completing Part 3 may be eligible for a long-term extension trial for an additional 2 years
- Phase 1 IV/SC bioavailability study in healthy volunteers ongoing with potential transition to SC administration in the long-term extension and phase 2 studies



Part 3 Key Eligibility Criteria

- ≥ 18 years and older
- IgAN verified by biopsy within the last 10 years
- If kidney biopsy performed within 2 years is without fibrosis, eGFR >45 mL/min per 1.73m^2 OR 30-45 mL/min per 1.73m^2
- Urine protein ≥0.5 g/24h; OR UPCR ≥0.5 g/g (or ≥ 50 mg/mmol)
- On a stable dose of RASi for >3 months or RASi intolerant

ClinicalTrials.gov Identifier: NCT03945318

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PO1620

Discovery of CHK-336: A First-in-Class, Liver-Targeted, Small Molecule Inhibitor of Lactate Dehydrogenase for the Treatment of Primary Hyperoxaluria

Jennifer H. Cox[1], Marc-Olivier Boily[1], Alex Caron[1], Amandine Chefson[1], Oliver Chong[1], Jim Ding[1], Valerie Dumais[1], Samuel Gaudreault[1], Robert Gomez[1], James Guthrie[1], Ross P. Holmes[2], Andrew J. King[1], John Knight[2], Jeff Lester[1], W. Todd Lowther[3], Renata Oballa[1], Michael D. Percival[1], Tao Sheng[1], Jayakumar Surendradoss[1], Joyce Wu[1], David A. Powell[1]

1. Chinook Therapeutics Canada, Inc., Vancouver, BC, Canada
2. University of Alabama at Birmingham, Birmingham, AL, United States
3. Wake Forest School of Medicine, Winston-Salem, NC, United States

Primary Hyperoxaluria (PH)

Rare and severe disorder leading to excess oxalate and end stage renal disease (ESRD)

Genetic liver enzyme deficiency resulting in excess oxalate

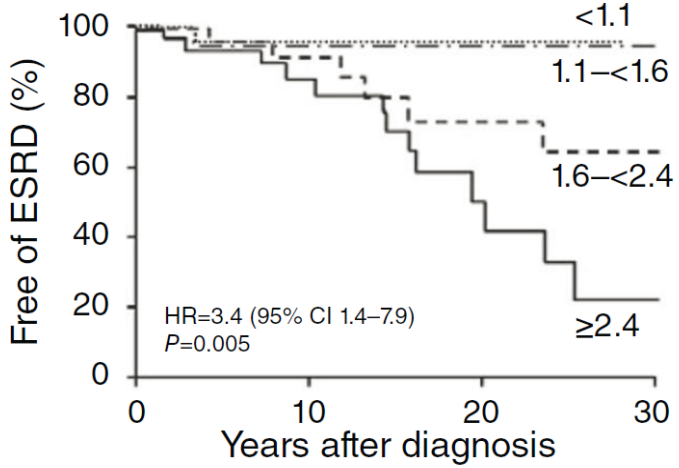
- Patients form many calcium oxalate kidney stones
- Median age of kidney failure in PH1 is 23 years
- Only curative treatment is dual kidney-liver transplant; no approved drug therapies

Lower urinary oxalate (UOx) levels associated with reduced risk of ESRD

Clinical proof of concept achieved by injectable siRNA agents (GO-siRNA for PH1 and LDHA-siRNA for PH1/PH2)

No oral, small molecule therapies with potential to treat patients with all types of PH have been reported

ESRD Risk by Urinary Oxalate Levels[1]



Disease Progression of PH

- Abnormal liver metabolism of glyoxylate produces **excess oxalate**
- Calcium **oxalate crystals form in the kidneys**
- Decline in kidney function results in systemic **oxalosis**
- Onset of **kidney failure**
- Dialysis awaiting **dual liver / kidney transplant**

1. Zhao et al CJASN 2016; 11(1): 119-126

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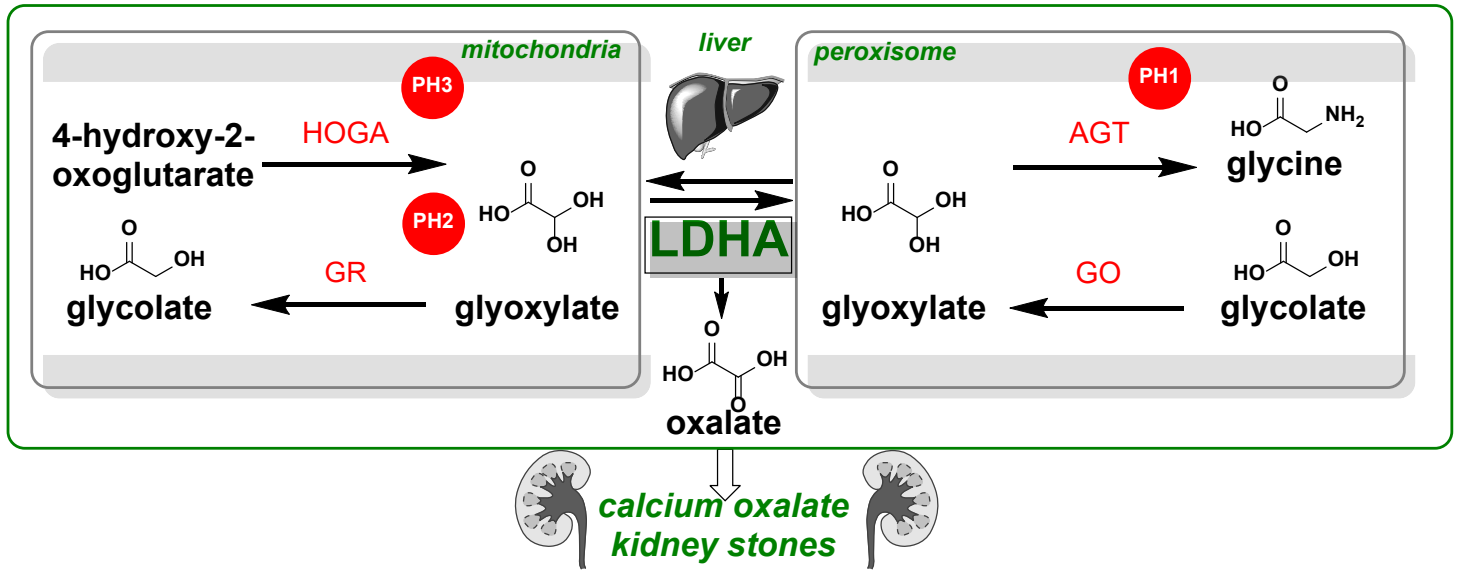
Targeting LDHA Addresses All PH-Causing Mutations and Pathways



Three types of PH caused by different mutations:

- PH1: AGXT (AGT protein)
- PH2: GRHPR (GR protein)
- PH3: HOGA1 (HOGA protein)

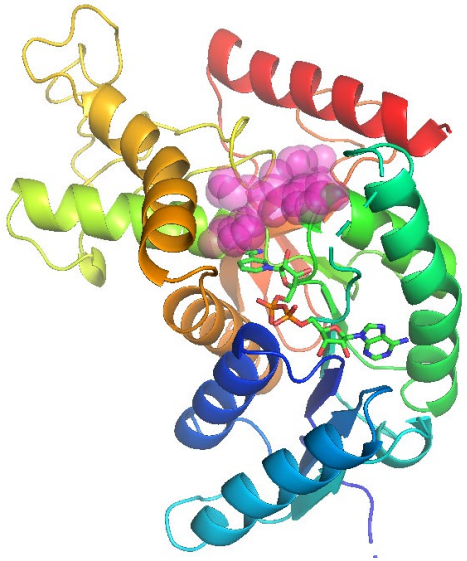
Lactate dehydrogenase (LDHA) is the final and committed step in production of oxalate from glyoxylate in the liver

- Represents a potential therapeutic target for all forms of PH, as well as other disorders arising from excess oxalate
- Complete loss-of-function of LDHA in humans results in exercise-induced muscle symptoms[1], therefore liver-targeting with low systemic levels is needed
- Liver-specific LDHA inhibition is expected to be safe and well tolerated

Chinook designed, synthesized and characterized hundreds of LDHA inhibitors with the goal of identifying a potent and selective compound with a liver-targeted tissue distribution profile for the treatment of all types of PH

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

CHK-336 is a Potent LDHA Inhibitor in Enzyme and Hepatocyte Assays Across Multiple Species



Compound design and sub-nM potency guided by structural biology and X-ray crystallography of LDHA-inhibitor complexes

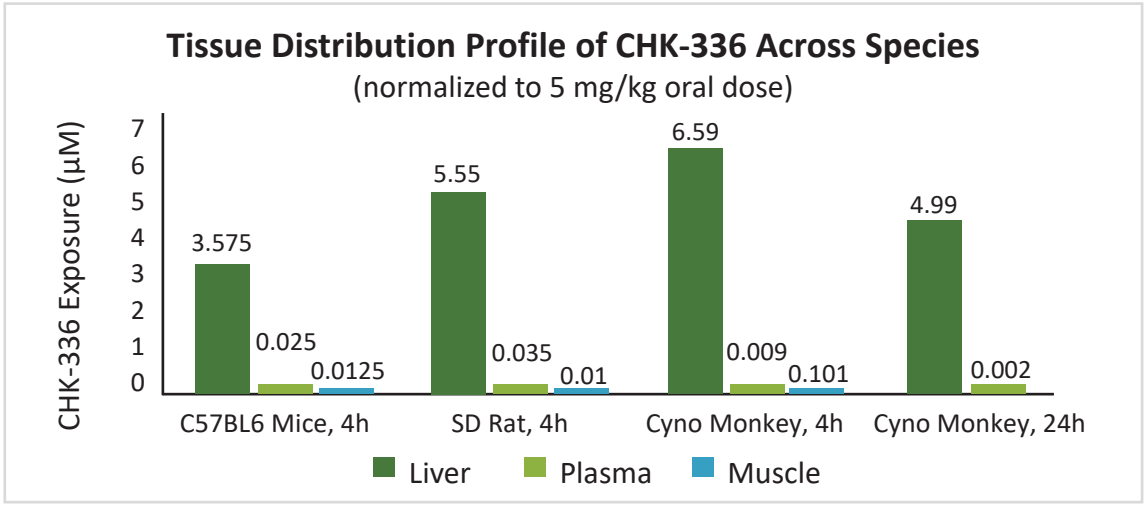
	ASSAY	CHK-336 IC_{50}
Enzyme	Human LDHA	**0.4 nM**
	Mouse LDHA	**0.1 nM**
Hepatocyte	Mouse Fresh Hepatocytes	**52 nM**
	Mouse Cryopreserved Hepatocytes	**80 nM**
	Rat Cryopreserved Hepatocytes	**130 nM**
	Monkey Cryopreserved Hepatocytes	**130 nM**
	Human Cryopreserved Hepatocytes	**131 nM**
PH1 Cell	Mouse *Agxt* Knockdown Hepatocytes (Oxalate Production)	**293 nM**

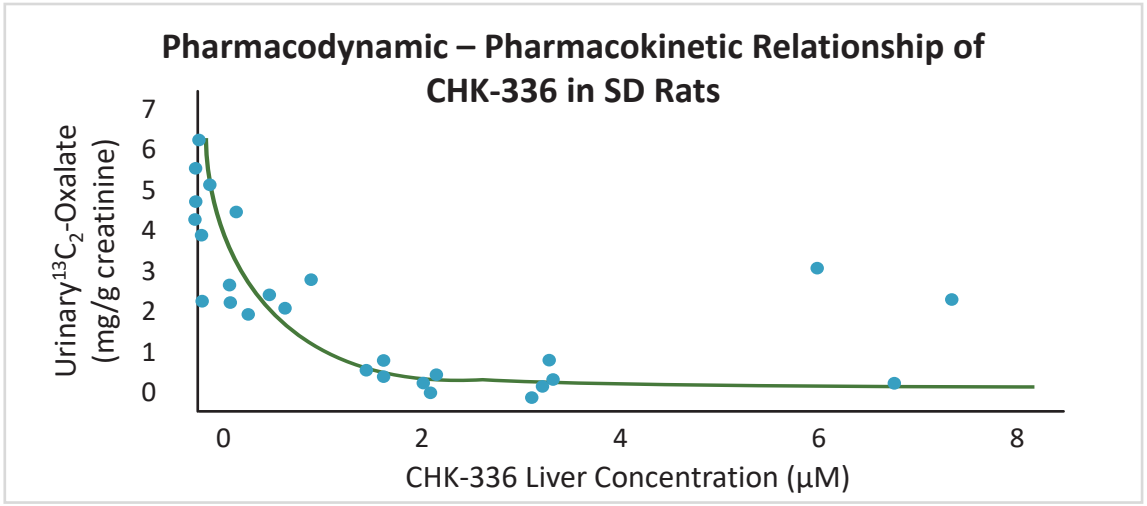
CHK-336 demonstrates potent inhibition of LDHA in enzyme assays (IC_{50} = 0.1-0.4 nM) and primary hepatocyte assays across multiple species (IC_{50} = 52-293 nM)

CHK-336 also demonstrated tight LDHA binding with a very slow off-rate (hours-days)

Pharmacokinetic and Pharmacodynamic Properties of CHK-336

- CHK-336 exhibits a liver-targeted tissue distribution profile in mice, rats and monkey with high liver concentrations and low extra-hepatic tissue exposures
- Long liver half-life observed across species; driven by good metabolic stability and tight, slow-off rate binding of CHK-336 to LDHA in the liver
- Well-profiled pharmacodynamic effect in mice and rats driven by liver concentrations: liver EC_{50} of ≈3 µM
- Human PK predictions suggest CHK-336 has the potential to be a low, once-daily oral dose in humans





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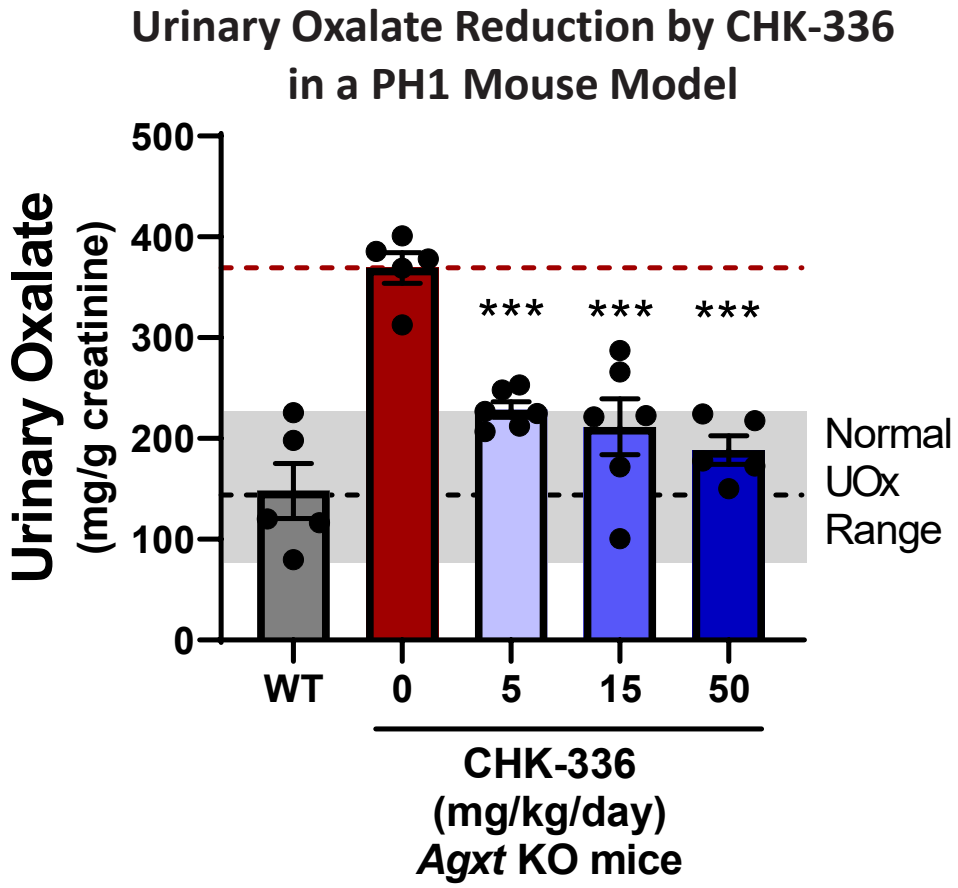
CHK-336 Produced Significant and Dose-dependent Reductions in Urinary Oxalate in a PH1 Mouse Model



- A mouse model of PH1 was generated by CRISPR-Cas9 deletion of exons 3-8 of *Agxt*; these mice exhibited elevated urinary oxalate as expected
- CHK-336 was dosed orally, once-daily for 7 days in male *Agxt* KO mice and urinary oxalate concentrations were compared to a vehicle control group
- **Low, oral, once-daily doses of CHK-336 significantly reduced urinary oxalate; majority of treated mice reached the normal range observed in wild-type mice**
- Analysis of liver concentrations of CHK-336 resulted in a PK/PD relationship with a liver EC_{50} of 1 – 5 μM CHK-336, consistent with rat liver PD values
- Similar data generated in a mouse *Agxt* knockdown model of PH1

Non-Clinical Safety Assessment Supports Continued Advancement of CHK-336 into IND-enabling Studies

Excellent in vitro Safety Profile

- Low risk of hERG mediated QT prolongation (IC_{50} > 30 µM)
- Non-mutagenic (negative in 5-strain AMES up to 5000 µg/well)
- Excellent off-target selectivity profile (<50% inhibition at 10 µM for 86 target panel, >450-fold selectivity for LDHA)

Low Drug-Drug Interaction (DDI) Potential

- Low risk of CYP-mediated DDI
  - No CYP3A4 inhibition or time-dependent inhibition (IC_{50} > 30 µM)
  - No CYP3A4 induction in hepatocytes (IC_{50} > 10 µM)

Promising Non-GLP in vivo Safety Profile

- Non-GLP in vivo safety studies suggest wide therapeutic margins over anticipated efficacious exposures
- Doses up to 1000 mg/kg/day explored in 14-day rat study

Conclusions

- Targeting LDHA, the terminal step in hepatic oxalate synthesis, represents a potential therapeutic strategy for all forms of PH, as well as other disorders arising from excess oxalate
- By potently blocking LDHA and engineering a liver-targeted tissue distribution profile, CHK-336 represents a potentially safe and effective oral small molecule for the treatment of primary hyperoxaluria
- CHK-336 shows robust efficacy in a PH1 mouse model at low, once-daily oral doses including the ability to reduce elevated urinary oxalate levels to the normal range
- The non-clinical safety assessment of CHK-336 conducted to date supports continued advancement into IND-enabling studies

CHK-336 is a first-in-class oral LDHA inhibitor with the potential to treat all subtypes of primary hyperoxaluria as well as other disorders arising from excess oxalate

CHINOOK THERAPEUTICS

SA-OR21

Single-Cell Transcriptomic Analysis to Define Cellular Heterogeneity in Human ADPKD

Yoshiharu Muto,[1] Eryn E. Dixon,[3] Chidambaram Ramachandran,[2] Andrew J. King,[2] Stephen L. Seliger,[3] Owen M.Woodward,[3] Paul A. Welling,[4] Terry J. Watnick,[3] Benjamin D. Humphreys.[1]

1. Washington University School of Medicine, St. Louis, MO, United States
2. Chinook Therapeutics Canada, Inc., Vancouver, BC, Canada
3. University of Maryland School of Medicine, Baltimore, MD, United States
4. Johns Hopkins School of Medicine, Baltimore, MD, United States

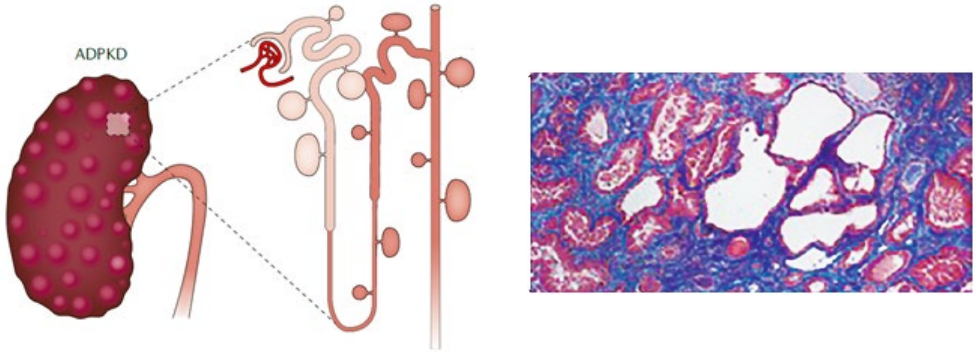
Single-Cell Transcriptomic Analysis to Define Cellular Heterogeneity in Human ADPKD

SA-OR21: Oral Presentation, Saturday, October 24, 2020 at 5:00 – 7:00 pm ET

Washington University in St. Louis

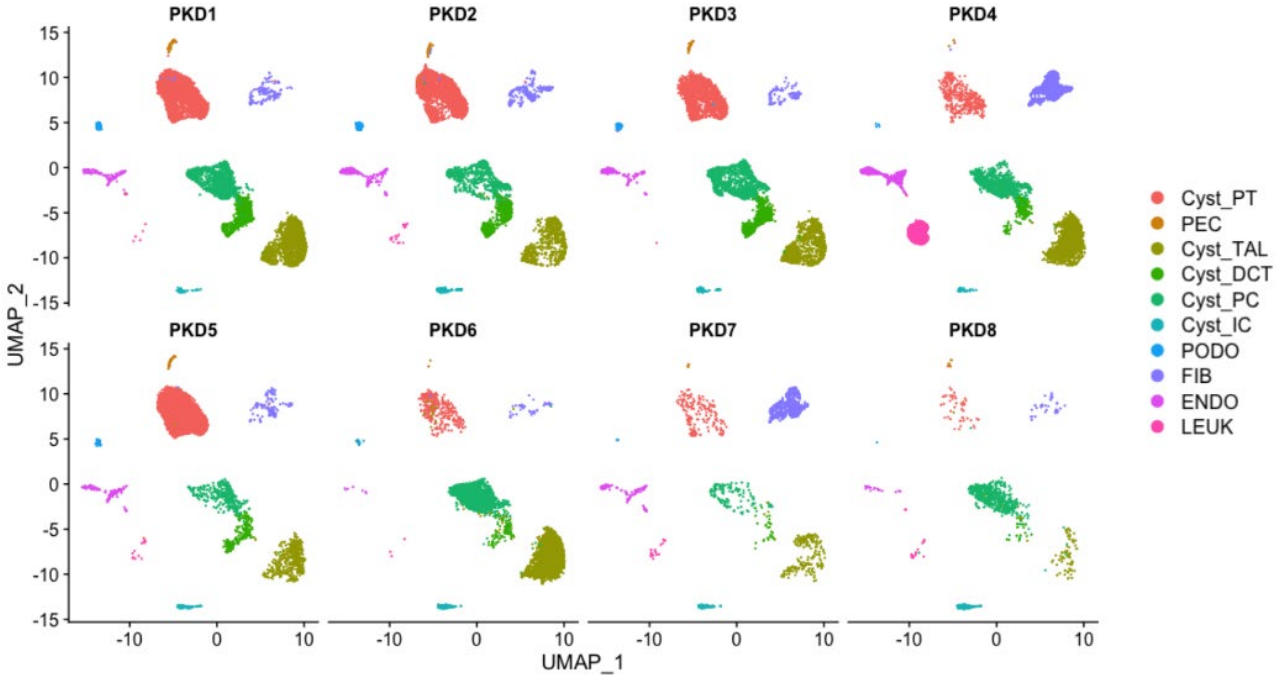
Ben Humphreys, MD, PhD

- ADPKD is the most common potentially lethal monogenic disorder globally and the most common inherited kidney disease with ~50% of patients progressing to ESKD by age 60



- The kidney has nearly 30 distinct cell types
- In ADPKD, cysts arise out of <1% of nephrons and only from discrete cell populations
- Single cell transcriptional resolution is required to define the central molecular drivers of cystogenesis

Multiple cell types detected in each ADPKD Patient Sample



- To our knowledge, this is the first single cell transcriptomic atlas described for human ADPKD
- Potential for unprecedented mechanistic insight into ADPKD

Funding: NIDDK Support, Commercial Support - Chinook Therapeutics

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Closing

Chinook Well-Positioned to Achieve Value-Generating Milestones

- **Building a differentiated and proprietary pipeline of precision medicines** targeting IgA nephropathy, glomerular diseases and other rare, severe chronic kidney diseases
- **Well-capitalized** to support multiple clinical and preclinical programs with >$275 million in capital upon closing of the Aduro merger and concurrent financing
- **Two clinical programs with potentially complementary mechanisms in IgA nephropathy** with atrasentan and BION-1301
  - IgA nephropathy is a large patient population with great unmet medical need
  - Atrasentan program de-risked through well-characterized safety profile and extensive data collected in >5,300 DKD patients
    - Phase 3 AFFINITY and phase 2 ALIGN trials initiating in H1 2021
  - BION-1301 has the potential to be a disease-modifying therapy with possibility to combine with other therapies, including atrasentan
    - Enrollment of IgAN patients ongoing in part 3 of phase 1 study, with interim data expected in H1 2021
- CHK-336, a **first-in-class, liver-targeted, oral LDHA inhibitor**, has the potential to treat all subtypes of PH and other disorders arising from excess oxalate
- Several research and discovery programs ongoing in other rare, severe chronic kidney diseases including ADPKD

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Q&A

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