Note Regarding Forward-Looking Statements

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Building the Leading Company Developing Precision Medicines for Kidney Disease

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

- 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)

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

2019

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

2020

2021

Long-Term Vision

Kidney Disease Industry Leader

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

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## Robust, Diversified and Focused Pipeline

Advancing pipeline of precision medicines for kidney diseases

<table>
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<tr>
<th>Program</th>
<th>Indication</th>
<th>Target Validation</th>
<th>Lead Optimization</th>
<th>IND-Enabling</th>
<th>Phase 1</th>
<th>Phase 2</th>
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<td>Phase 3 initiation planned in early 2021</td>
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<td>Research Programs</td>
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<td>Discovery Programs</td>
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We will continue to evaluate opportunities to add additional kidney disease programs to pipeline
The Time is Now for Kidney Disease Drug Development

Underserved market with >$120B of annual healthcare costs in U.S.
Few new drugs approved in past 20 years

Emerging patient stratification approaches and translational platforms
Causal mutations, biomarkers, single-cell sequencing, translational models (e.g. organoids)

Recent acceptance of surrogate endpoints based on understanding of pathophysiology
Proteinuria, eGFR (vs. hard renal outcomes in historical trials)
Developing Two Therapies to Address Large Unmet Medical Need in IgA Nephropathy

Strategy to target key molecular drivers of progressive IgA Nephropathy in high-risk patients with two distinct and potentially complementary MOAs

IgAN is the most common primary glomerular disease; orphan disease – approximately 140K patients in US

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

Roughly 30-45% of IgAN patients will develop end-stage renal disease (ESRD) over a period of 20-25 years

- Up to 50% of high-risk patients develop ESKD in 10 years

No approved treatments and limited options for high-risk patients

- Renin Angiotensin System inhibition (RASi) with ACEi/ARB is frontline treatment
- Immunosuppressive agents typically provide inconsistent therapeutic benefit and are accompanied by significant side effects

Potential to seek accelerated approval on surrogate endpoint (proteinuria reduction) and full approval on kidney function (change in eGFR)

Proteinuria Identifies IgAN Patients at High 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

Rapid 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
Atrasentan

Potent and Selective Endothelin A Receptor Antagonist for the Potential Treatment of IgA Nephropathy (IgAN) and Other Primary Glomerular Diseases
Atrasentan is an Investigational Potent, Highly-Selective ETₐ Inhibitor with Clinically Established Kidney Protection

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 ETₐ (1860-fold vs. ET₉)

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 ETₐ inhibition in IgAN
- Exclusivity period based on IP and potential for orphan designation
- Unmet medical need creates strong commercial opportunity

Entered into license agreement with AbbVie in December 2019

- Paid upfront licensing fee and issued ~6.8M shares of private Chinook common stock
- $135M in future potential developmental, regulatory & commercial milestone payments
- High-single-digit to high teens royalties

UACR (percent change in geometric mean from baseline) in the AbbVie phase 2 RADAR study
AbbVie Global SONAR Phase 3 Outcome Trial in DKD

 SONAR Topline Results

- 3,600 high-risk DKD patients randomized
- 2-year median treatment duration
- 35% decreased risk of ESRD or doubling of serum creatinine in responders* (28% in all randomized)
- 27% risk reduction of End Stage Renal Disease
- 39% risk reduction of 57% decline in eGFR
- Safety profile consistent with class
- Clinically manageable fluid retention

“These data support a potential role for selective endothelin receptor antagonists in protecting renal function in patients with type 2 diabetes at high risk of developing end-stage kidney disease.”

- Heerspink et al.

*Responders classified as patients who achieved >30% reduction in proteinuria
SONAR Outcomes Encouraging for Potential Benefit in IgA Nephropathy

Proteinuria reduction at 6 months expected to be potential endpoint to seek accelerated approval in IgAN

*In Responders (patients who achieved >30% reduction in proteinuria)

Kidney Function (eGFR Slope)*

- eGFR slope is potential confirmatory efficacy endpoint to seek full approval in IgAN
- Treatment effects on proteinuria predict stronger effects on eGFR slope in IgAN

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 ~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

- Open-label design, 12-week proteinuria primary endpoint
- ~20 patients / cohort
- Overlap with phase 3 sites to support enrollment

Cohorts include:
- IgAN with proteinuria 0.5 – <1 g/day
- FSGS
- Alport Syndrome
- DKD as add on SGLT2 inhibitors

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<th>2020</th>
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<td>Phase 2 Basket Trial</td>
<td>Initial proteinuria data</td>
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<td>Present additional data as available</td>
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</table>
BION-1301

Investigational Anti-APRIL Antibody for the Potential Treatment of IgA Nephropathy (IgAN)
Role of APRIL in IgA Nephropathy

A critical step in IgAN pathogenesis is the production of galactose-deficient IgA1 (Gd-IgA1) leading to the generation of anti-Gd-IgA autoantibodies and immune complex formation that result in kidney damage.

A proliferation-inducing ligand (APRIL) promotes IgA class-switching and survival of IgA-producing plasma cells.

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

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.
BION-1301 is an Investigational Anti-APRIL Antibody

• Humanized IgG4 monoclonal antibody
• Blocks APRIL binding to both of its receptors, BCMA and TACI
• Potential disease-modifying treatment that targets the underlying causes of IgAN
• In nonhuman primates, BION-1301 reduces serum IgA, IgG and IgM levels without drug-related toxicity
• Well-tolerated in clinical studies
  • Enrollment of IgAN patients in Phase 1b ongoing
  • Single and multiple IV doses in healthy volunteers completed; data presented at ERA-EDTA Virtual Congress in June 2020
  • Evaluated up to 2700mg IV q2w in a Phase 1 safety and PK/PD study of late stage multiple myeloma patients
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 supporting the potential to be administered by monthly dosing
- Some accumulation observed, leading to greater & sustained PD responses with repeat dosing
BION-1301 Phase 1 in IgAN Patients Ongoing

- Part 3 currently enrolling IgAN patients in the US; UK enrollment to begin in early 2021
- BION-1301 administered by IV infusion every 2-4 weeks for 12 weeks
- Patients completing Part 3 may be eligible for the long-term extension trial to receive BION-1301 for an additional 2 years
- Phase 1 IV/SC bridging study in healthy volunteers ongoing with potential for SC administration in the long-term extension and phase 2 studies

ClinicalTrials.gov Identifier: NCT03945318

**Part 1: Double-blind, Placebo-controlled Single Ascending Dose (Healthy Volunteers) COMPLETE**

- Cohort SAD-1: 10mg N = 3:1
- Cohort SAD-2: 50mg N = 6:2
- Cohort SAD-3: 150mg N = 6:2
- Cohort SAD-4: 450mg N = 6:2
- Cohort SAD-5: Up to 1350mg N = 6:2

**Part 2: Double-blind, Placebo-controlled Multiple Ascending Dose (Healthy Volunteers) COMPLETE**

- Cohort MAD-1: 50mg N = 6:3
- Cohort MAD-2: 150mg N = 6:3
- Cohort MAD-3: 450mg N = 6:3
- Cohort MAD-4: 1350mg N = 6:2

**Part 3: Open-label, Multiple Dose (IgAN Patients) ONGOING**

- IgAN Cohort 1: 450mg N = 10
- IgAN Cohort 2: Dose TBD N = 10

**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 1.73m² or 30-45 mL/min per 1.73m²
- 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
CHK-336

Potent and Selective Small Molecule Lactate Dehydrogenase A (LDHA) Inhibitor for the Potential Treatment of Hyperoxalurias
Diseases caused by excess oxalate, a potentially toxic metabolite typically filtered by the kidneys and excreted as a waste product in urine

Primary hyperoxalurias (PH) 1-3 are ultra-rare diseases caused by genetic mutations that result in hepatic overproduction of oxalate

- Symptoms of PH include recurrent kidney stones, severe pain, blood in the urine and urinary tract infections, which when left untreated, can result in kidney failure
- Median age of kidney failure in PH1 is 23 years
- ~5,000 – 7,000 PH1 patients in the US and Europe

Secondary (SH) or idiopathic hyperoxalurias are more common and may be due to excess absorption of dietary oxalate or endogenous over-production of oxalate

Streamlined regulatory pathway with surrogate endpoint and small trial size

Systemic \textit{Oxalosis Affects Multiple Organs}

Disease Progression of PH

- Abnormal liver metabolism of glyoxylate produces \textit{excess oxalate}
- \textit{Calcium oxalate crystals} deposit in the kidney \textit{forming stones}
- Decline in kidney function results in \textit{systemic oxalosis}
- Onset of \textit{kidney failure}
- Dialysis awaiting \textit{dual liver / kidney transplant}
Lactate dehydrogenase (LDHA) is the final step in endogenous production of oxalate from glyoxylate in the liver

- LDHA inhibition potential to treat all forms of PH
- Human LDHA heterozygous deficient patients show no phenotype, while homozygous have exercise-induced muscle symptoms
- Liver-targeted LDH inhibition anticipated to be safe and well-tolerated

Three types of PH caused by different mutations:

- AGXT – PH1
- GRHPR – PH2
- HOGA1 – PH3
CHK-336 is a Potent and Selective Oral Small Molecule LDHA Inhibitor with Compelling Preclinical Efficacy

In preclinical studies, CHK-336 produced significant and dose-dependent urinary oxalate reductions in PH1 mouse models into the range observed in WT mice.

Exploratory toxicity studies in rodents demonstrated wide safety margins.

CHK-336 is currently progressing through IND-enabling studies with phase 1 initiation planned for H2 2021.

Differentiation of CHK-336

- Potential for superior efficacy through more complete target inhibition, resulting in increased urinary oxalate reduction and a more favorable impact on clinical manifestations, including kidney stones, as well as overall disease progression.

- Oral administration is more convenient and enables expansion into less severe forms of hyperoxaluria.

- Excellent safety margins in preclinical studies support continued development.

- Titration and customized dosing is possible for better individual efficacy.
Financials & Catalysts
Financial Strength

NASDAQ: KDNY

Strong Balance Sheet
• >$275M in operating capital

Cash Guidance
• Operating capital into 2023

Common Stock Outstanding:
• ~42M shares as of October 5, 2020

<table>
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<tr>
<th>Event</th>
<th>Date</th>
<th>$ Announced</th>
<th>Source</th>
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<tr>
<td>Series A</td>
<td>August 2019</td>
<td>$65M</td>
<td>Versant Ventures</td>
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<td>Apple Tree Partners</td>
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<td>Samsara Biocapital</td>
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<td>Private placement</td>
<td>October 2020</td>
<td>$115M</td>
<td>Top-tier investors</td>
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<td>Reverse merger</td>
<td>October 2020</td>
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## Planned Upcoming Catalysts

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<tr>
<th>Program</th>
<th>Indication</th>
<th>Catalyst</th>
<th>H2 2020</th>
<th>H1 2021</th>
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<tr>
<td>Atrasentan</td>
<td>IgA Nephropathy</td>
<td>Initiate Phase 3 ALIGN study</td>
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<td>Basket of Glomerular Diseases</td>
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<td>BION-1301</td>
<td>IgA Nephropathy</td>
<td>Report Phase 1 interim patient data</td>
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<td>CHK-336</td>
<td>Ultra-rare Kidney Disease</td>
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<td></td>
<td>Initiate Phase 1 study</td>
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Why Chinook?

- Kidney disease is a large underserved market poised to be transformed by recent advances in the understanding of underlying diseases and new approaches to drug development.
- Two clinical programs with potentially complementary mechanisms in IgA Nephropathy with atrasentan and BION-1301.
- Building a differentiated and proprietary pipeline of precision medicines targeting IgA nephropathy, glomerular diseases and other rare, severe chronic kidney diseases.
- Well-capitalized with >$275M to support multiple clinical and preclinical programs.
- Seasoned management and scientific team with track record of advancing transformative therapies.
- Pursuing opportunities for strategic partnerships and attractive profile for long-term value generation.