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 Need
~9.1% 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 Path
Increased understanding of underlying disease biology

New and validated drug targets

FDA recognizing surrogate markers, such as proteinuria and eGFR, as registration endpoints\(^3\)

Building a Leading Kidney Disease Company

**Atrasentan**
- Highly potent and selective ET$_A$ antagonist evaluated in more than 5,300 CKD patients
- Phase 2 data from IgAN cohort expected in H1 2022 and from other glomerular disease cohort(s) in H2 2022
- Phase 3 proteinuria data in IgAN expected in 2023

**BION-1301**
- Anti-APRIL monoclonal antibody (mAb)
- Demonstrated durable reductions in mechanistic biomarkers and clinically meaningful proteinuria improvements in patients with IgAN
- Additional subcutaneous phase 1/2 data in IgAN patients expected in H1 2022

**CHK-336**
- Oral small molecule LDHA inhibitor with liver-targeted tissue distribution for primary hyperoxaluria
- Potential to treat all disorders of excess endogenous oxalate
- 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 through H1 2023*
Advancing a Diversified Pipeline of Best-in-class Programs

<table>
<thead>
<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>
<th>Phase 3</th>
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<tr>
<td><strong>Atrasentan</strong></td>
<td>IgA Nephropathy</td>
<td></td>
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<td>Phase 3 ongoing</td>
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<td><strong>AFFINITY</strong></td>
<td>Basket of glomerular diseases</td>
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<td>Phase 2 ongoing</td>
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<tr>
<td><strong>BION-1301</strong></td>
<td>IgA Nephropathy</td>
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<tr>
<td><strong>CHK-336</strong></td>
<td>Primary Hyperoxaluria</td>
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<td>Phase 1 HV study planned for H1 2022</td>
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<tr>
<td><strong>Research &amp; Discovery Programs</strong></td>
<td>Rare, severe chronic kidney diseases</td>
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<td>Potential 2022 DC</td>
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</tbody>
</table>

continue to evaluate opportunities to add kidney disease programs to pipeline
Why Target IgA Nephropathy?

- Most common primary glomerular disease globally with ~140K – 150K US prevalence
- No approved treatments and >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)

Atrasentan has potential to attenuate hallmark characteristics of IgAN, including mesangial cell activation, proteinuria and kidney inflammation and fibrosis (Hit 4)

BION-1301 is a potential disease-modifying treatment that targets the underlying cause of IgAN (Hit 1)


Atrasentan
Potent and Selective Endothelin A Receptor (ET$_A$) Antagonist
Atrasentan: a Potent and Selective ETₐ Antagonist

ETₐ receptor activation drives IgAN progression through multiple potential mechanisms

- ETₐ 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

- ETₐ receptor blockade by atrasentan is a promising approach to treat IgAN patients

**Visualization:**

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

- Diagram illustrating the role of ET-1 and ETₐ in IgAN progression with proteinuria, inflammation, and fibrosis markers.
AbbVie Evaluated Atrasentan in >5,300 DKD Patients

Potential to benefit IgAN patients with a rapid registration pathway

Optimized ET$_A$ antagonist studied extensively in DKD

- Picomolar potency and highly selective for ET$_A$
- Optimal dose of 0.75 mg daily established
- Rapid and sustained ~30-35% proteinuria reductions consistently observed in phase 2 and 3

Strong rationale for development in IgAN

- Clinical validation of proteinuria lowering with ET$_A$ blockade in IgAN
- Proteinuria reductions predict stronger clinical benefit in IgAN
- Younger IgAN patient population optimal for tolerability
Global SONAR Phase 3 Outcome Trial in DKD

SONAR Topline Results

3,600 high-risk DKD patients randomized and treated for up to 5 years (median 2.2 years)

- Decreased risk of ESRD or doubling of serum creatinine in responders* (28% in all randomized patients)

- Proteinuria (UACR) reduction

- 0.0005 p-value for eGFR preservation in responders*

Well characterized safety profile | 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% UACR reduction following 6-week enrichment period
### 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)

#### Phase 2 Basket Trial to Expand Potential Across Proteinuric Glomerular Diseases
- 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

### Timeline

<table>
<thead>
<tr>
<th>2020</th>
<th>2021</th>
<th>2022</th>
<th>2023</th>
<th>2024</th>
<th>2025</th>
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<tr>
<td><strong>Phase 3</strong> IgAN Trial</td>
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<td>Proteinuria top line</td>
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<td></td>
<td>eGFR endpoint data</td>
<td></td>
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<tr>
<td><strong>Phase 2</strong> Basket Trial</td>
<td>IgAN cohort data</td>
<td>Data from additional cohorts as available</td>
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</table>
BION-1301
Anti-APRIL Monoclonal Antibody
BION-1301: Potentially Disease-Modifying Anti-APRIL mAb

APRIL: TNF-family cytokine involved in B-cell signaling
- Drives IgA production and survival of IgA-secreting plasma cells
- Shown to increase Gd-IgA1 secretion
- Higher APRIL levels in IgAN patients correlated with higher Gd-IgA1 and proteinuria and lower eGFR
- APRIL gene variants confer increased risk of IgAN

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
- Well-tolerated up to 2700mg in phase 1 multiple myeloma study
- Phase 1 bioavailability study in HVs supports SC dosing

BION-1301 Ongoing Phase 1/2 IgAN Trial

Currently dosing patients with subcutaneous BION-1301 in Cohort 2

BION-1301 in HVs and patients with IgAN to date:
- Well-tolerated with no SAEs or BION-1301-related treatment discontinuations
- PK: T½ ~33 days / PD: rapid and sustained free APRIL reductions
- Durable reductions in Gd-IgA1, IgA and IgM, and to a lesser extent, IgG
  - Provides a PD window to deplete IgA and Gd-IgA1, while minimizing impact on IgG
- Proof of concept established: clinically meaningful proteinuria reductions in patients with IgAN

<table>
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<tr>
<th>Part 1 SAD in Healthy Volunteers</th>
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<tr>
<td>10 – 1350 mg IV</td>
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<table>
<thead>
<tr>
<th>Part 2 MAD in Healthy Volunteers</th>
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<tbody>
<tr>
<td>50 – 450 mg IV q2w</td>
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</table>

<table>
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<tr>
<th>Part 3 in IgAN Patients</th>
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</thead>
<tbody>
<tr>
<td>✓</td>
</tr>
<tr>
<td>Cohort 1: 450 mg IV q2w for 52 weeks</td>
</tr>
<tr>
<td>Cohort 2: 600 mg SC q2w for 52 weeks</td>
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</tbody>
</table>

Potential Cohort 3: SC Dose/Schedule TBD for 52 weeks

Enrollment ongoing
## Phase 1/2: Demographics & Baseline Characteristics

**IgAN Cohort 1: 450 mg BION-1301 IV q2w**

### Demographics (n=10)

<table>
<thead>
<tr>
<th>Demographics</th>
<th>Value</th>
</tr>
</thead>
</table>
| **Age, years**<br>
Median (min, max)             | 39 (27, 59)         |
| **Sex, male**<br>
   n (%)                      | 9 (90)              |
| **Race, white**<br>
   n (%)                      | 10 (100)            |
| **Ethnicity, Hispanic**<br>
   n (%)                      | 2 (20)              |
| **Country, US**<br>
   n (%)                      | 10 (100)            |

### Baseline Characteristics

<table>
<thead>
<tr>
<th>Baseline Characteristics</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Renin-angiotensin system inhibitor use</strong>&lt;br&gt;%</td>
<td>100</td>
</tr>
</tbody>
</table>
| **Time from biopsy, years**<br>
Median (min, max)        | 2.0 (0.2, 3.4)      |
| **Blood pressure (mmHg)**<br>
   Systolic - Median (min, max)   | 127 (113, 133)     |
|   Diastolic - Median (min, max)  | 83 (69, 88)         |
| **eGFR (mL/min/1.73 m²)**<br>
Median (min, max)        | 69 (30, 122)        |
| **24-hour urine protein excretion (g/day)**<br>
Median (min, max)        | 1.22 (0.74, 6.47)   |
| **24-hour UPCR (g/g)**<br>
Median (min, max)        | 0.64 (0.41, 4.55)   |

* eGFR by CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration
Phase 1/2: Safety and Tolerability

IgAN Cohort 1: 450 mg BION-1301 IV q2w

• To date, BION-1301 has been well-tolerated in IgAN patients (n=10)

<table>
<thead>
<tr>
<th>AE Category</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects with any TEAE</td>
<td>5 (50)</td>
</tr>
<tr>
<td>Any TEAE occurring in N&gt;1 subjects</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Treatment-related AE</td>
<td>0 (0)</td>
</tr>
<tr>
<td>AE leading to discontinuation</td>
<td>0 (0)</td>
</tr>
<tr>
<td>SAE</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Infusion-related reactions</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>

• Data cutoff: October 6, 2021
  – IgG concentrations remained above study-defined threshold in all patients
  – No notable changes in frequency of circulating naïve and memory B-cell subsets
  – 8 patients remain on treatment, with time on treatment ranging from <1 month to >14 months

AE = adverse event; SAE = serious adverse event; TEAE = treatment-emergent adverse event
Phase 1/2: Changes in Free APRIL Concentrations

IgAN Cohort 1: 450 mg BION-1301 IV q2w

Rapid and durable reductions in free APRIL demonstrate target neutralization sustained through 1 year

BION-1301 pharmacokinetics in patients with IgAN is consistent with previous experience in healthy volunteers

No anti-drug antibodies observed in patients with IgAN to date
BION-1301 durably reduces IgA, IgM, and to a lesser extent, IgG in patients with IgAN.

BION-1301 produces sustained reductions in serum Gd-IgA1. The depletion of this pathogenic IgA isoform (Hit 1) in patients with IgAN demonstrates the potential disease-modifying mechanism of BION-1301.

IgG concentrations remained above the study-defined threshold in all patients, providing a pharmacodynamic window to deplete IgA while minimizing impact on IgG.

Phase 1/2: Changes in Serum Ig Concentrations

IgAN Cohort 1: 450 mg BION-1301 IV q2w

- BION-1301 durably reduces IgA, IgM, and to a lesser extent, IgG in patients with IgAN.
- BION-1301 produces sustained reductions in serum Gd-IgA1.
  - The depletion of this pathogenic IgA isoform (Hit 1) in patients with IgAN demonstrates the potential disease-modifying mechanism of BION-1301.
- IgG concentrations remained above the study-defined threshold in all patients, providing a pharmacodynamic window to deplete IgA while minimizing impact on IgG.
Phase 1/2: Clinically Meaningful Proteinuria Reductions

IgAN Cohort 1: 450 mg BION-1301 IV q2w

Study Day

% Reduction (Geomean ± SEM)

-100 -80 -60 -40 -20 0 20 40 60 80 100

% Reduction in UPCR

- 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

*1/10 patients missed 24-hour collection; morning void used
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 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 H1 2022
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

Secondary hyperoxalurias are 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

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
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
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

Translational Models

- Modeling Human Disease
  - Disease Mechanisms
  - Target Validation
  - Deep Biological Insights

Detailed insights into molecular pathogenesis of stratified CKDs

Target Execution

Development Candidates

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

Novel & differentiated molecules

Partnerships

Drug Discovery +
Chinook’s Precision Medicine Platform Fueled by One of the Most Comprehensive PANOMICS Kidney Programs

UK Academic / Industry consortium for CKD biobank

Kidney single-cell RNAseq

Washington University Nephrology

Translational Models

Isolated glomeruli
Podocytes
Human iPSC kidney organoids

Multi-OMICS Integration Platform

Target Validation
Target ID Pipeline
Patient Stratification
Financial Strength

NASDAQ: KDNY

Strong Balance Sheet
• $204.8 M in cash, cash equivalents and marketable securities as of September 30, 2021

Cash Guidance
• Operating capital through H1 2023 based on current business plan

Common Stock Outstanding
• ~45.1 million shares as of November 4, 2021
• ~46.0 million fully diluted shares as of November 4, 2021*

Asia Partnering Strategy for Atrasentan & BION-1301
• Higher incidence and prevalence of IgAN creates large unmet medical need
• Evaluating partnering opportunities in China and related territories
• Local presence may accelerate clinical development and maximize commercial potential

* 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

<table>
<thead>
<tr>
<th>Program</th>
<th>Indication</th>
<th>Catalyst</th>
<th>H1 2021</th>
<th>H2 2021</th>
<th>H1 2022</th>
<th>H2 2022</th>
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<tbody>
<tr>
<td><strong>Atrasentan</strong></td>
<td>IgA Nephropathy</td>
<td>Initiate phase 3 ALIGN study</td>
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<td>✔️</td>
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<tr>
<td></td>
<td>Basket of Glomerular Diseases</td>
<td>Initiate phase 2 AFFINITY study</td>
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<tr>
<td></td>
<td></td>
<td>Present data from IgAN patient cohort of AFFINITY</td>
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<td>✔️</td>
<td>✔️</td>
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<tr>
<td></td>
<td></td>
<td>Present data from additional AFFINITY patient cohort(s)</td>
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<td>✔️</td>
<td></td>
</tr>
<tr>
<td><strong>BION-1301</strong></td>
<td>IgA Nephropathy</td>
<td>Present additional biomarker data and IV-to-SC bioavailability data in healthy volunteers</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
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<tr>
<td></td>
<td></td>
<td>Present phase 1/2 data in IgAN patients</td>
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<td>✔️</td>
<td>✔️</td>
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<td>Analyze phase 1/2 data and announce update on later-stage clinical development strategy</td>
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<tr>
<td><strong>CHK-336</strong></td>
<td>Primary Hyperoxaluria</td>
<td>Complete IND-enabling studies</td>
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<td></td>
<td>✔️</td>
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<tr>
<td></td>
<td></td>
<td>Initiate phase 1 study in healthy volunteers</td>
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