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Nurix Drugs Engage Ligases for the Treatment of Cancer

Targeted Protein Modulation: \( \text{TPM} = \text{TPD} + \text{TPE} \)

**Targeted Protein Degradation (TPD)**

Ubiquitin is ligated to target proteins to tag them for degradation by the proteasome.

**Harness ligases to decrease specific protein levels**

**Targeted Protein Elevation (TPE)**

Inhibit ligases to increase specific protein levels.

**A Powerful Cellular System**
Nurix is advancing a pipeline of propriety and partnered programs in oncology and autoimmune/inflammatory diseases.

<table>
<thead>
<tr>
<th>MOA</th>
<th>Drug program</th>
<th>Target</th>
<th>Therapeutic area</th>
<th>Discovery</th>
<th>IND enabling</th>
<th>Phase 1a</th>
<th>Phase 1b</th>
</tr>
</thead>
<tbody>
<tr>
<td>TPD</td>
<td>NX-2127</td>
<td>BTK-IKZF</td>
<td>B-cell malignancies</td>
<td></td>
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<tr>
<td>TPD</td>
<td>NX-5948</td>
<td>BTK</td>
<td>B-cell malignancies</td>
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<td>TPE</td>
<td>NX-1607</td>
<td>CBL-B</td>
<td>Immuno-Oncology</td>
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<tr>
<td>TPD</td>
<td>NX-0479 / GS-6791</td>
<td>IRAK4</td>
<td>Rheumatoid arthritis and other inflammatory diseases</td>
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<tr>
<td>TPM</td>
<td>5 programs</td>
<td>Undisclosed</td>
<td>Oncology / autoimmune disease</td>
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<td>TPD</td>
<td>4 programs</td>
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<tr>
<td>DAC</td>
<td>Multiple programs</td>
<td>Undisclosed</td>
<td>Oncology</td>
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</tbody>
</table>
Advancing a New Therapeutic Class

Degrader-Antibody Conjugates (DACs)

- DACs combine the catalytic activity of a Targeted Protein Degrader (TPD) with the specificity of an antibody
- DACs represent the next generation of antibody drug conjugates (ADCs)

Deal Terms

- $60 million upfront cash payment
- $3.4 billion in potential research, development, regulatory and commercial milestone payments
- Mid-single to low double-digit tiered royalties on future product sales
- Option for U.S. profit sharing and co-promotion on up to two products arising from the collaboration
Leveraging Partnerships to Advance a Broad Pipeline of Targeted Protein Degraders

<table>
<thead>
<tr>
<th>Ligand Discovery</th>
<th>Degrader Discovery</th>
<th>Cellular Profiling</th>
<th>In vivo Profiling</th>
<th>Oral exposure</th>
<th>DC nomination</th>
<th>Partner License</th>
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<tbody>
<tr>
<td>IRAK4 (Gilead)</td>
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<td>Target 10</td>
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<td>Target 2</td>
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<td>Target 3</td>
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<td>Target 5</td>
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<td>Target 6</td>
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<td>Target 7</td>
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<td>Target 10</td>
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</table>

- Novel ligands identified for multiple unprecedented targets
- $44M in drug discovery milestones achieved to date
- $20M licensing payment from Gilead for IRAK4 degrader
- Option to co-develop and co-promote 4 drug candidates
Targeted Protein Degradation
Harnessing the ubiquitin proteosome system to eliminate disease proteins

NX-5948 MOA

BTK Destroyed by the Proteasome

BTK

Poly Ubiquitinated Proteins

Ubiquitination of Protein

Ubiquitin

Cereblon E3 Ligase Complex

E2

Degrader recycling

Nurix degrader drugs
Nurix Degraders:

1) Are effective against resistance mutations through binding cooperativity between BTK and the ligase complex
2) Eliminate the scaffolding function of BTK oncogenic signals

Removal of BTK disrupts the signaling complex, effectively destroying the scaffolding function of the protein.
A First-In-Class Franchise of BTK Degraders: NX-5948 & NX-2127 – The Big Picture

<table>
<thead>
<tr>
<th>NX-5948</th>
<th>BTK degraders have the potential to displace inhibitors in the markets where BTK inhibitors currently dominate (e.g., CLL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SELECTIVE BTK DEGRADATION</td>
<td>Nurix has demonstrated that BTK degraders can overcome treatment emergent resistance mutations to both covalent and non-covalent inhibitors</td>
</tr>
<tr>
<td>NX-2127</td>
<td>BTK degraders may expand the market for BTK targeted agents into other B-cell malignancies such as DLBCL and potentially into autoimmune diseases</td>
</tr>
<tr>
<td>BTK DEGRADATION &amp; IMMUNOMODULATION</td>
<td></td>
</tr>
<tr>
<td>NX-5948</td>
<td>NX-5948 and NX-2127 are two distinct drugs with differentiated profiles, each with the potential to be multi-billion dollar B-cell malignancy therapeutic franchises</td>
</tr>
</tbody>
</table>
Nurix BTK Degrader Franchise: Two BTK Degraders to Cover the Landscape of B-Cell Malignancies

**B-Cell Malignancies Annual Incidence (US & EU)**

- **CLL**
  - 39,700 Chronic Lymphocytic Leukemia
- **WM**
  - 6,300 Waldenstrom’s macroglobulinemia
- **MCL**
  - 6,200 Mantle cell lymphoma
- **MZL**
  - 10,700 Marginal Zone Lymphoma
- **DLBCL**
  - 55,100 Diffused Large B-Cell Lymphoma
- **FL**
  - 26,200 Follicular Lymphoma

**NX-5948**
- for BTK inhibitor resistance mutations in CLL with potential for early lines of therapy
- NX-5948 potential in combination

**NX-2127**
- for aggressive NHL and advanced CLL including BTK inhibitor resistance mutations
- NX-2127 in 3L+
- Goal: Displace BTKi in indications where single-agent BTKi is standard of care and address BTKi resistance mutations
- Goal: Expand role for BTK target therapy

*BTK, Bruton tyrosine kinase; DLBCL, Diffuse large B-cell lymphoma; CLL, Chronic lymphocytic leukemia; SLL, small lymphocytic lymphoma; MCL, Mantle cell lymphoma; WM, Waldenstrom’s macroglobulinemia; MZL, Marginal zone lymphoma; FL, Follicular lymphoma; NHL, non-Hodgkin lymphoma

Estimates based on 2020 incidence from DRG, GlobalData and secondary research; EU comprised of France, Germany, Italy, Spain and UK
Blockbuster Opportunity in BTK Market
$8.4 billion in annual sales

- Next generation BTK inhibitors are currently taking market share from Imbruvica
- Nurix BTK degraders have the potential to be game changing and take shares from the inhibitor market in CLL
- Opportunity for Nurix BTK degraders to expand the market in other B cell malignancies and autoimmune diseases
Evolution of BTK Targeted Therapies

1st Generation Covalent inhibitor

Ibrutinib

Next Generation Covalent inhibitors

Acalabrutinib
Zanubrutinib

Non-Covalent Inhibitors

Pirtobrutinib

Degraders

NX-2127
NX-5948

Resistance is Futile
Emerging Unmet Medical Need with Resistance Mutations to Existing BTK Inhibitors

NX-5948 and NX-2127 can degrade all treatment emergent inhibitor mutations identified to date.
NX-5948 was Designed for Potent and Rapid Degradation of Wildtype and C481S-Mutated BTK

TMD8 cells harboring WT BTK or a knock-in BTK mutation (C481S) were incubated with NX-5948 for 24 hours, and BTK degradation was assessed by flow cytometry.
Structural and Enzymatic Studies of New BTKi-Resistant Mutations Confirms BTK Scaffolding Function

Some mutations that confer resistance to BTK inhibitors lack kinase activity yet still potentiate BCR signaling

Montoya et al., ASH 2022 and unpublished data
NX-5948 Is More Potent and Broadly Active Than All BTK Inhibitors Tested

- **BTK degradation and activation marker suppression in TMD8 tumor cells**
- **TMD8 tumor cell killing**
  - EC50 (nM)
  - NX-5948
  - Ibrutinb
  - Pirtobrutinib
  - Acalabrutinib
  - Zanubrutinib
  - Nemtabrutinib

- **Average of n = 3 independent experiments ± SEM**

- **Most potent cell killing**

- **• All inhibitors have resistance mutation liabilities**
- **• NX-5948 displays potent cell killing and maintains suppression of CD86 in the context of key resistance mutations**
NX-2127 Induces Positive Binding Cooperativity Between BTK and Cereblon

- Positive Cooperativity ($\alpha > 1$)
- Stable ternary complex
- Induced protein-protein interactions
- Greater tolerance for reduced binary affinity

**CRBN**, cereblon; **DDB1**, DNA damage binding protein 1 (component of the ubiquitin ligase complex)
NX-5948-301: Trial Design
Phase 1 trial in adults with relapsed/refractory B-cell malignancies

Objectives:
- Assess safety and tolerability
- Identify maximum tolerated dose (MTD) & biologically active dose
- Evaluate PK/PD

Dose escalation

- Dose level 1: 50 mg
- Dose level 2: 100 mg
- Dose level 3: 200 mg
- Dose level 4: 300 mg

Oral daily dosing

Dose expansion options

- CLL/SLL failed BTKi and BCL2i
- DLBCL, MCL
- FL, MZL, WM
- PCNSL

- Phase 1a dose escalation is ongoing at clinical sites in the U.S. and U.K.
- Anticipate initiating expansion cohort(s) in H2 2023

BTK, Bruton tyrosine kinase; CLL, chronic lymphocytic leukemia; DLBCL, diffuse large B-cell lymphoma; FL, follicular lymphoma; MCL, mantle cell lymphoma; MZL, marginal zone lymphoma; PCNSL, primary CNS lymphoma; PD, pharmacodynamics; PK, pharmacokinetics; WM, Waldenstrom's macroglobulinemia
First Report of BTK Degradation with NX-5948 in Patients with B Cell Malignancies

Initial proof of mechanism

- Rapid and sustained degradation of BTK
- Robust BTK degradation observed in all patients tested to date
- Dose escalation ongoing in patients with relapsed/refractory B cell malignancies
NX-2127-001: Trial Design
Phase 1 trial in adults with relapsed/refractory B-cell malignancies

***Objectives:***
- Assess safety and tolerability
- Identify maximum tolerated dose (MTD) & biologically active dose
- Evaluate PK/PD

**Dose escalation**

- Dose level 1: 100 mg
- Dose level 2: 200 mg
- Dose level 3: 300 mg

**Oral daily dosing**

**Dose expansions**

- **CLL**
  - Failed 2 or more prior treatments including a BTK inhibitor and regardless of baseline BTK mutation status (up to 40 pts)

- **DLBCL**
  - Failed 2 or more prior treatments (up to 20 pts)

- **MCL**
  - Failed 2 or more prior treatments including a BTK inhibitor (up to 20 pts)

**Patients Eligible for Dose Expansions:**
- CLL Phase 1b expansion cohort ongoing at 100 mg dose
- DLBCL Phase 1b expansion cohort ongoing at 300 mg dose
- MCL Phase 1b expansion cohort ongoing at 300 mg dose
- Phase 1a dose escalation is ongoing at 200 mg and 300 mg doses for patients with NHL

**Key Terms:**
- BTK, Bruton tyrosine kinase; CLL, chronic lymphocytic leukemia; DLBCL, diffuse large B-cell lymphoma; FL, follicular lymphoma; MCL, mantle cell lymphoma; MZL, marginal zone lymphoma; NHL, non-Hodgkin lymphoma; PCNSL, primary CNS lymphoma; PD, pharmacodynamics; PK, pharmacokinetics; WM, Waldenstrom's macroglobulinemia
## Baseline Characteristics

Elderly population with multiple prior lines of targeted therapies and acquired mutations

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>CLL (n=23)</th>
<th>Overall population (N=36)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median age, years (range)</td>
<td>75 (61–90)</td>
<td>75 (50–92)</td>
</tr>
<tr>
<td>Female, n (%)</td>
<td>9 (39.1)</td>
<td>13 (36.1)</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>14 (60.9)</td>
<td>23 (63.9)</td>
</tr>
<tr>
<td>Lines of prior therapy, median (range)</td>
<td>5 (2–11)</td>
<td>4 (2–11)</td>
</tr>
<tr>
<td>BTKi, n (%)</td>
<td>23 (100)</td>
<td>31 (86.1)</td>
</tr>
<tr>
<td>Pirtobrutinib, n (%)</td>
<td>8 (34.8)</td>
<td>11 (30.6)</td>
</tr>
<tr>
<td>BTKi and BCL2i, n (%)</td>
<td>18 (78.3)</td>
<td>19 (52.8)</td>
</tr>
<tr>
<td>cBTKi, ncBTKi, and BCL2i, n (%)</td>
<td>7 (30.4)</td>
<td>7 (19.4)</td>
</tr>
<tr>
<td>BTK mutation present(^a), n (%)</td>
<td>10 (48)</td>
<td>11 (35)</td>
</tr>
<tr>
<td>C481</td>
<td>5 (24)</td>
<td>5 (16)</td>
</tr>
<tr>
<td>L528W</td>
<td>4 (19)</td>
<td>4 (13)</td>
</tr>
<tr>
<td>T474</td>
<td>3 (14)</td>
<td>4 (13)</td>
</tr>
<tr>
<td>V416L</td>
<td>1 (5)</td>
<td>1 (3)</td>
</tr>
<tr>
<td>BCL2 mutation present(^a), n (%)</td>
<td>4 (19)</td>
<td>4 (13)</td>
</tr>
<tr>
<td>PLCG2 mutation present(^a), n (%)</td>
<td>0 (0)</td>
<td>1 (3.2)</td>
</tr>
</tbody>
</table>

\(^a\)Specific mutations are not additive as some patients have multiple BTK mutations. Mutations were tested by NGS centrally in those patients with available samples (n=31 in total population; n=21 in CLL population)

Data cutoff: September 21, 2022
NX-2127 Leads to Robust BTK Degradation and Decrease in B-cell Activation

- Daily treatment with NX-2127 resulted in a rapid and sustained suppression of BTK (CD19+) as measured in patient whole blood using a flow cytometry assay. BTK suppression target of 80% reached consistently (data not shown here).
- Robust decrease of plasma CCL4 by Cycle 1 Day 8 and suppression was maintained through Cycle 2 Day 1, consistent with clinically observed lymphocytosis occurring in majority of patients with nodal disease by Cycle 1 Day 8.
- NX-2127 treatment also resulted in degradation of cereblon neo-substrate Ikaros.

**BTK**, Bruton's tyrosine kinase; **CCL4**, C–C motif ligand 4; **LLOQ**, lower limit of quantification.

**Data cutoff: September 21, 2022**
**NX-2127 Preliminary Efficacy**

**Positive Initial Findings in CLL**

<table>
<thead>
<tr>
<th>Disease-evaluable patients</th>
<th>n=15</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Objective response rate</strong>, a % (95% CI)</td>
<td>33 (12–62)</td>
</tr>
<tr>
<td><strong>Best response, n (%)</strong></td>
<td></td>
</tr>
<tr>
<td>CR</td>
<td>0 (0)</td>
</tr>
<tr>
<td>PR</td>
<td>5 (33.3)</td>
</tr>
<tr>
<td>SD</td>
<td>5 (33.3)</td>
</tr>
<tr>
<td>PD</td>
<td>2 (13.3)</td>
</tr>
<tr>
<td>NE b</td>
<td>3 (20)</td>
</tr>
</tbody>
</table>

*a* Objective response rate includes CR + CRi + nPR + PR-L + PR

*b* Patients who discontinued after a single assessment of SD are considered as NE

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**BCL2i**, B-cell lymphoma-2 inhibitor; **BTK**, Bruton’s tyrosine kinase; **BTKi**, BTK inhibitor; **CR**, complete response; **CRi**, complete response with incomplete count recovery; **NE**, not evaluable; **PD**, progressive disease; **PR**, partial response; **SD**, stable disease

---

*One patient, not shown above, with prior BTKi and BCL2i treatment and with a BTK mutation detected at baseline, had no nodal disease at baseline. Their treatment is ongoing with a PR.*
First Demonstration of Clinical Activity of a Degrader Against a Range of BTK Mutations

NX-2127 Preliminary Efficacy in Patients with CLL

- BTK degradation of 80% achieved in CLL patients including those harboring BTK C481, T474, L528, and V416 resistance mutations

Patients with kinase dead mutations are classified as kinase dead regardless of co-occurrence of kinase proficient mutations.

Disease- evaluable patients

- BTK WT
- BTK Kinase Dead (C481R, L528W and V416L); n=5
- BTK Kinase Proficient (C481S and T474F/I); n=4

Data cutoff: September 21, 2022
Rapid and Sustained Complete Response on Single-Agent NX-2127

FDG-PET CT Scan Disease Assessment

Baseline

Confirmatory Week 16 Scan

- 84-year-old woman with multiply relapsed ABC-DLBCL following 4 lines of aggressive therapy (including combination of rituximab, ibrutinib, and lenalidomide).

- Complete response at first assessment (week 8), confirmed at week 16, and ongoing through week 24.

- As of June 14, 2023, this patient remains on treatment with over 12 months of follow up.
Phase 2 Smart Start: Ibrutinib, Lenalidomide, and Rituxan + Chemo in Newly Diagnosed Non-GCB DLBCL

“The combination of RLI alone and with chemotherapy resulted in high response rates and promising survival outcomes in patients with newly diagnosed DLBCL.”

“Smart Start resulted in PFS and OS rates at 2 years, of 91.3% and 96.6%, respectively. R-CHOP with and without ibrutinib resulted in a 3-year PFS rates of 70.8% and 68.1%, respectively. R-CHOP with and without lenalidomide resulted in a 2-year PFS rates of 67% and 64%, respectively.”

Source: Westin et al; Journal of Clinical Oncology, published online August 11, 2022
Targeting CBL-B Enhances Antitumor Response
A Master Orchestrator of the Immune System

CBL-B mediated mechanisms strongly restrains a productive anti-tumor response

CBL-B inhibition increases:
• DC and NK infiltration and function
• T cell priming
• Cytotoxic T cells function
• Ability of T cells to resist tumor immunosuppressive mechanisms: Treg, MDSC, and TGF-β
NX-1607 Mechanism of Action: Intramolecular Glue

1. Kinase
   Phosphorylation locks CBL-B in the ACTIVE Conformation

2. E2/substrate

NX-1607 acts as an intramolecular glue forcing CBL-B in its folded INACTIVE state

Immune Response

Substrate protein
NX-1607 Increases IL-2 and IFN-γ Secretion in TCR Stimulated Primary Human T cells

NX-1607 increases TCR stimulation-dependent production of IL-2 and IFN-γ in primary human T cells

NX-1607 has no impact in the absence of T cell stimulation as measured by proliferation, activation, or cytokine release.

Cytokine Response  
Baseline Response
Single-Agent NX-1607 Induces Antitumor Response in Multiple Models

**NX-1607 Reduced Tumor Volume**
- Colorectal

**NX-1607 Prolonged Survival**
- Triple-Negative Breast

**NX-1607 Reduced Tumor Volume**
- B Cell Lymphoma

**Graphs and Data**
- Tumor Volume (mm$^3$)
- % Survival
- Days post implant
- NX-1607 30 mg/kg day 7 to 46
- NX-1607 30 mg/kg day 16 to 28
- Statistical significance indicated:
  - p<0.0001
  - NS

Shaded area indicates dosing period
NX-1607 and Anti-PD-1 Synergize to Enhance Anti-tumor Effects and Survival of Mice in Multiple Tumor Models

Colorectal (CT26)

Long-Term Survival

Colorectal (MC38)

Long-Term Survival

Triple-Negative Breast (4T1)

Day 28 4T1 Lung Metastases

Shaded area indicates dosing period: NX-1607 (30 mg/kg, PO daily) and anti-PD-1 twice a week at 10 mg/kg dosing period.
NX-1607-101: Phase 1 first-in-human clinical trial design

Two-Part Phase 1 Monotherapy Trial of NX-1607 in Relapsed or Refractory Tumors

Objectives:
- Assess safety and tolerability
- Identify maximum tolerated dose
- Evaluate PK/PD including proprietary biomarkers

Oral daily dosing

Dose Escalation

Potential Cohort Expansion

- Melanoma
- Squamous Cell Carcinoma of the Head and Neck (HNSCC)
- Non-small Cell Lung Cancer (NSCLC)
- Platinum Resistant Epithelial Ovarian Cancer (EOC)
- Gastric Cancer
- Metastatic Castration Resistant Prostate Cancer (mCRPC)
- Mixed Solid Tumor Cohort
- Diffuse Large B-cell Lymphoma (DLBCL)

- Checkpoint resistant tumors
- Immunosuppressive microenvironment
- Poorly immunogenic tumors
UbiScan Identified Direct CBL Substrates Within the T Cell Receptor (TCR) Signaling Cascade

- Decreased signal represents direct substrates ubiquitinated by CBL-B ligase activity.
- Inhibiting CBL-B decreases ubiquitination of important T Cell receptor signaling molecules.
Phospho-Protein Flow Cytometry Assay Identified Proximal Biomarkers

- Human PBMCs were stimulated with or without CBL-B inhibition
- Expression levels were determined for phospho-proteins downstream the TCR signaling
- Overlapping results from orthogonal assays (Ubiscan) provided confidence in proximal biomarker signals

HS1: Substrate of LYN receptor, and an essential adaptor protein at the immune synapse, via VAV1

PLCγ2: Expressed in both T cells and B cells; associates with LAT and SLP-76 & becomes phosphorylated upon TCR stimulation

ZAP70: Key organizer of downstream TCR signaling
Dose Dependent Increases of CBL-B Proximal Biomarker Correlates with Antitumor Effects of NX-1607

Pharmacodynamic relationship in mice following NX-1607 dosing

In vivo efficacy observed between 10-60 mpk which corresponds to ~20-60% pHS1+ CD8+ T Cells
Characterization of a Novel Biomarker and First Evidence of Target Engagement for a CBL-B Inhibitor in the Clinic

Human whole blood and dose projection modeling

Clinical data

Maximal % pHS1+ expressing CD8+ T cells observed in C1D1

<table>
<thead>
<tr>
<th>Proposed dose level*</th>
<th>NX-1607 dose (mg)</th>
<th>Estimated % HS1+/CD8+ T cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2.5</td>
<td>22.2</td>
</tr>
<tr>
<td>10</td>
<td>5</td>
<td>30.0</td>
</tr>
<tr>
<td>2</td>
<td>15</td>
<td>49.7</td>
</tr>
<tr>
<td>3</td>
<td>25</td>
<td>60.6</td>
</tr>
<tr>
<td>4</td>
<td>50</td>
<td>74.0</td>
</tr>
</tbody>
</table>

*Estimated % HS1+/CD8+ T cells calculated based on Dose level 4 (50mg) as a reference point.

*Minimum anticipated biological effect level (MABEL).
CBL-B Inhibition Has the Potential To Be the Small Molecule Centerpiece of Immuno-Oncology Therapy

- **Prostate, MSS Colorectal**: Low-Immunogenicity Tumors
- **Ovarian, Gastric**: Suppressive TME
- **Post-ICI (Melanoma, NSCLC, TNBC, Meso, Urothelial, etc.)**: Acquired Resistance
- **DLBCL (including Richter’s)**: T-Cell Dysfunction

**Immune Checkpoint Failure or Resistant**

**Single-Agent Monotherapy**

**Potential Combinations with FDA-Approved Agents**

- **Bi-specific T-cell Engager (BiTE)**: Blinatumomab
- **PARPi**: Olaparib, Rucaparib, Talazoparib
- **ADCC mAb’s**: Rituximab, Herceptin, Trastuzumab
- **Immune Checkpoint Inhibitors**: Pembrolizumab, Nivolumab, Cemiplimab Ipilimumab, Atezolizumab, Avelumab, Durvalumab
- **Immunogenic Cell Death Chemo**: Paclitaxel, Platinums
- **Antibody-Drug Conjugates (ADC)**: Trastuzumab Emtansine, Polatuzumab vedotin-piq
- **Cell Therapy (TIL, TCR, NKCAR)**: CART

**NX-1607**
Defining Success in 2023

**B-cell malignancies**

- Present updated Phase 1 clinical data in H2 2023
- Define regulatory strategy based on FDA feedback in H2 2023

**Immune oncology**

- Present initial clinical data from Phase 1a in H2 2023
- Define Phase 1b dose for cohort expansion in H2 2023

**Platform & pipeline**

- Present initial clinical data from Phase 1a in H2 2023
- Define Phase 1b dose for cohort expansion in H2 2023

- Select new targeted protein degrader development candidate
- Achieve substantial research collaboration milestones throughout 2023

Note: All anticipated timing is based on calendar-year periods
Strong Financial Position

*Proforma cash of $369M* provides cash runway into Q2 2025

Partnerships Generate Cashflow and Reduce Need for Dilutive Financing

- $400 million generated through discovery partnership payments
- Potential for profit splits on up to 6 programs across three collaboration partners
- $8.2 billion in potential future payments

*Based on $309M in cash and investments as of May 31, 2023*
Thank you