This presentation contains forward-looking statements. In some cases, you can identify forward-looking statements by the words “will,” “expect,” “intend,” “plan,” “objective,” “believe,” “estimate,” “potential,” “continue” and “ongoing,” or the negative of these terms, or other comparable terminology intended to identify statements about the future. These statements are based on management’s current beliefs and expectations. These statements include but are not limited to statements regarding Inhibrx, Inc.’s (the “Company”) business strategy, the Company’s plans to develop and commercialize its product candidates, the safety and efficacy of the Company’s product candidates, the Company’s plans and expected timing with respect to clinical trials and regulatory filings and approvals, manufacturing matters, strength of intellectual property protection, and the size and growth potential of the markets for the Company’s product candidates, and any implication that pre-clinical data or preliminary or topline results will be representative of the results of later trials. This presentation also contains certain projections and estimates regarding the Company’s future financial performance, namely potential future revenue for certain of the Company’s product candidates. This information also constitutes forward-looking information and is for illustrative purposes only and should not be relied upon as necessarily being indicative of any future results. The assumptions and estimates underlying this estimated financial information are inherently uncertain and subject to a wide variety of significant business, economic competitive and other risks and uncertainties that could cause actual results to differ materially from those contained in the prospective financial information. These potential financial information and other forward-looking statements involve substantial known and unknown risks, uncertainties and other factors that may cause the Company’s actual results, levels of activity, performance or achievements to be materially different from the information expressed or implied by these forward-looking statements.

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Our company

Our mission

- To evolve into a commercial-stage biopharmaceutical company with a differentiated and sustainable product portfolio by focusing on the following:
  - Rapidly advance and optimize clinical development
  - Create differentiated, next-generation therapeutics in focused disease areas
  - Maintain our culture of innovation, execution and efficiency
  - Maximize the potential of our therapeutic pipeline

- To discover and develop effective biologic treatments for people with life-threatening conditions

Key Financial Highlights:
(as of 9/30/2023)

<table>
<thead>
<tr>
<th></th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cash and cash equivalents</td>
<td>$337.3M</td>
</tr>
<tr>
<td>Common stock outstanding</td>
<td>47.3M</td>
</tr>
<tr>
<td>Fully diluted outstanding</td>
<td>61.2M</td>
</tr>
</tbody>
</table>

- 165+ employees with an experienced leadership team and deep expertise in protein engineering
- In-house experience: discovery, protein engineering, cell biology, translational research, chemistry, manufacturing and controls, clinical development and operations and commercial

- Founded in 2010
- First IND in 2018
- IPO in 2020
Inhibrx’s innovative approach to therapeutic discovery

Our core belief:
Our technology and protein engineering expertise enables us to efficiently identify optimal therapeutic formats bespoke to the target biology.

Modular protein engineering platforms
- Single domain antibodies
- Recombinant proteins
- Engineered cytokines
- Engineered Fc Domains

Rapid and iterative optimization process
- Biological function
- Biophysical properties
- Pharmacokinetic profile
- Therapeutic Format
  - Affinity
  - Developability / Manufacturability

Therapeutics tailored to disease biology
- Multi-valent molecules
  - Molecule delivers higher clustering correlating with greater activity and more robust signal

- Multi-specific molecules
  - Molecule binds to multiple targets allowing cross linking – a novel way of delivering the drug closer to where it’s needed

- Fc-Fusion Proteins
  - Endow proteins with antibody-like PK properties
### Pipeline

**Oncology:**

<table>
<thead>
<tr>
<th>Product</th>
<th>Stage</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>INBRX-109</td>
<td>Preclinical</td>
<td>Precisely engineered valency to mediate optimal balance of efficacy and safety</td>
</tr>
<tr>
<td>INBRX-106</td>
<td>Phase 2</td>
<td>Registration-enabling study in chondrosarcoma underway</td>
</tr>
<tr>
<td>INBRX-105</td>
<td>Phase 3</td>
<td>Expansion of Ewing cohort following preliminary efficacy data as shown at CTOS</td>
</tr>
</tbody>
</table>

- **INBRX-109** tetravalent DR5 agonist
- **INBRX-106** hexavalent OX40 agonist
- **INBRX-105** tetravalent PD-L1 targeted 4-1BB agonist

**Anti-Inflammatory / Rare disease:**

<table>
<thead>
<tr>
<th>Product</th>
<th>Stage</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>INBRX-101</td>
<td>Preclinical</td>
<td>Optimize to achieve and maintain normal functional Alpha-1 antitrypsin levels with less frequent dosing</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Registration-enabling studies in AATD underway</td>
</tr>
<tr>
<td></td>
<td></td>
<td>De-risked opportunity in GvHD with studies beginning in 1H '24</td>
</tr>
</tbody>
</table>

- **INBRX-101** recombinant Alpha-1 antitrypsin Fc-fusion protein (AAT-Fc)

**Other Programs on the Horizon**

- FcRN Antagonist
- Radiopharmaceuticals
- T-cell Engagers - ContraMAB® Platform
- γδ T-cell Targeted Cisleukin™ Molecule
Near term expected clinical milestones

INBRX-101
(GvHD) Trial initiations for aGvHD and cGvHD

INBRX-106
(OX40) Keytruda combination update

INBRX-105
(PD-L1x41BB) Single agent data in r/r HNSCC

INBRX-109
(DR5) Registration-enabling Phase 2 Chondrosarcoma data

INBRX-101
(AATD) Initial readout from ElevAAte study

INBRX-106
(OX40) Randomized, CPI r/r NSCLC data
INBRX-101
AATD
recombinant Alpha-1 antitrypsin Fc-fusion protein (AAT-Fc)

Goal:
To develop a donor-independent source of AAT protein able to keep patients in the range of normal for extended periods of time
INBRX-101 for the treatment of alpha-1 antitrypsin (AAT) deficiency (AATD)

Disease background
AATD is an inherited rare disease of the lungs and liver (~15% of cases) characterized by low levels of AAT protein, a neutrophil elastase inhibitor, causing progressive deterioration of the tissue.

Liver
Hepatocytes synthesize and secrete AAT into the blood stream

Lung
AAT protects from lung tissue damage

Healthy:
- AAT protein inactivates neutrophil elastase thus preventing elastin degradation.

AATD:
- Without AAT protein, elastase activity is unchecked and causes damage to the lung tissue and restricts airflow.

Current standard of care
Weekly augmentation of plasma donor derived AAT (pAAT) brings patients to roughly half the normal level.
INBRX-101 for the treatment of AATD

INBRX-101 is a precisely engineered recombinant human AAT-Fc fusion protein

**INBRX-101 characteristics:**

- **Sustainable supply**: Not dependent on plasma donation
- **Purified product**: Independent of risks associated with blood derived products
- **Less frequent dosing**: Better quality of life
- **Normalization of AAT levels**: Dosed to keep patients in the healthy normal range

**AAT**
- Two recombinant human AAT proteins
- Oxidation-prone residues eliminated
- Prevents loss of activity in the lungs

**IgG4 Fc**
- Extended half-life
- Provides patients with normal level of protein for up to 3-4 weeks

INBRX-101 is a precisely engineered recombinant human AAT-Fc fusion protein designed to address the needs of patients with AATD by offering sustainable supply, purified product, less frequent dosing, and normalization of AAT levels.
## INBRX-101: phase 1 study design

### INBRX-101 phase 1 study: open-label, multicenter, dose-escalating study

### Eligibility

**Parts 1 and 2:**
Adult patients with AATD, regardless of prior treatment with augmentation therapy

AAT serum concentration <11 µMa

Nonsmoker

### Part 2

80 and 120-mg/kg cohorts:
Postbronchodilator FEV1 of ≥40% of predicted normal value

### Primary endpoint:
Safety & tolerability of INBRX-101, determined by the frequency & severity of adverse events

### Secondary endpoints:
Pharmacokinetics, pharmacodynamics, and immunogenicity of INBRX-101

### Part 1

**Single ascending dose (SAD)**

<table>
<thead>
<tr>
<th>Dose</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 mg/kg</td>
<td>6</td>
</tr>
<tr>
<td>40 mg/kg*</td>
<td>6</td>
</tr>
<tr>
<td>80 mg/kg</td>
<td>6</td>
</tr>
<tr>
<td>120 mg/kg</td>
<td>6</td>
</tr>
</tbody>
</table>

**Complete**

### Part 2

**Multiple ascending dose escalation (MAD)**

<table>
<thead>
<tr>
<th>Dose</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>40 mg/kg</td>
<td>6</td>
</tr>
<tr>
<td>80 mg/kg*</td>
<td>6</td>
</tr>
<tr>
<td>120 mg/kg*</td>
<td>6</td>
</tr>
</tbody>
</table>

Each patient receives 3 doses, Q3W

* bronchoalveolar lavage
Topline results from phase 1, part 2

**Parts 2:**

Favorable safety and tolerability profile with only mild and a few moderate AEs that were transient and fully reversible with minimal or no symptomatic care.

Dose related increases in maximal and total exposure occurred across entirety of SAD and MAD ranges of 10-120 mg/kg.

Overall, antidrug antibodies (ADAs) had no significant impact on INBRX-101 PK.

MAD cohorts demonstrate observed Cavg of functional AAT of 37.6 µM and 45.4 µM over the 21-day dosing interval following the third 80 mg/kg and 120 mg/kg doses, respectively.

In contrast, fAAT levels from PiMM genotype healthy volunteers (n=65) ranged from 21 to 54 micromolar (µM), with a mean of 36 µM.

fAAT levels at Day 70 (28 days following the 3rd dose), on average, were within the normal range for the 120 mg/kg dose level.

---

**INBRX-101 topline results – 3rd dose of 40, 80 or 120 mg/kg (Q3W):**

<table>
<thead>
<tr>
<th>Time (days)</th>
<th>Baseline: 0</th>
<th>7</th>
<th>14</th>
<th>21</th>
<th>28</th>
</tr>
</thead>
<tbody>
<tr>
<td>40 mg/kg</td>
<td></td>
<td></td>
<td>100</td>
<td>60</td>
<td>20</td>
</tr>
<tr>
<td>80 mg/kg</td>
<td></td>
<td></td>
<td>70</td>
<td>40</td>
<td>20</td>
</tr>
<tr>
<td>120 mg/kg</td>
<td></td>
<td></td>
<td>50</td>
<td>30</td>
<td>0</td>
</tr>
</tbody>
</table>

Indicates timing after third dose
* Baseline values shown at Day 0

Healthy (PiMM) Normal Range
INBRX-101 is present in the lung in every patient sampled following IV dosing suggesting penetration to target organ

Bronchoalveolar lavage fluid (BALF) sample collection and analysis

BALF samples were collected from 3 lobes of the lung for each patient in the 80 (N = 5) and 120 (N = 6) mg/kg MAD cohorts prior to dosing and two weeks after completion of multiple dosing. INBRX-101 concentrations were measured using a proprietary validated mass spectrometry assay specific to INBRX-101.

BALF assessment results

Rolled over from the Part 1 SAD2 had measurable INBRX-101 while drug was undetectable in INBRX-101 naïve patients (data not shown).

Post-dose, INBRX-101 was present in each lung lobe of every patient for which a bronchoscopy was performed.

The Phase 1 study data provide emerging evidence of a dose-dependent increase in INBRX-101 lung exposure.

Each point represents the average INBRX-101 concentration measured across three lobes in an individual subject.

Horizontal lines are the median values for each dose level.
Modeled phase 2 dose of 120 mg/kg Q4W achieves AAT normal range

- INBRX-101 PK/PD modeling and simulation projections: 120 mg/kg Q4W is predicted to achieve and maintain steady-state functional AAT levels within the normal range.

- pdAAT is only projected to achieve normal functional AAT levels for 2 – 2.5 days per weekly dose in AATD patients (9/28 days per month).

- Troughs fully greater than 21.1 μM
- Average concentration of approximately 36 μM
- Overall exposure at steady-state (AUCs) twice that of pdAAT

Projected mean steady-state functional AAT levels

- INBRX-101
- pdAAT
- 36 μM mean of functional AAT normal range
- 11 μM historical putative threshold

Functional AAT levels (μM)

Baseline: 0

Time (days)
INBRX-101 AATD registration-enabling trial

**Main Eligibility Criteria**
- Adult patients aged 18-80 with AATD and evidence of emphysema
- AAT antigenic serum concentration <11 µM
- Nonsmoker or former smoker
- 5-week washout for those on augmentation therapy
- Randomization stratified by baseline antigenic AAT & FEV1 (% predicted)

**Study INBRX101-01-201: ElevAAATe**

- Randomized, active controlled, double-blind
- Head-to-head superiority study:
  - INBRX-101 vs. pdAAT
  - 32-week treatment period
  - ~40 US, AUS, NZ sites

**Primary Endpoint:** Mean change in avg fAAT concentration as measured by anti-neutrophil elastase capacity (ANEC) from baseline to average serum trough fAAT concentration at steady state ($C_{\text{trough,ss}}$)

**Key Secondary Endpoints:** INBRX-101 vs pdAAT: mean change in fAAT concentration from baseline to fAAT avg concentration at steady state ($C_{\text{avg,ss}}$), and % of days with fAAT above the lower limit of the normal range during steady-state dosing; Bronchoscopy sub-study of ~30 patients to run at designated sites

**Study INBRX101-01-202: ElevAAATe-OLE (Open Label Extension)**

- Open label, long-term safety and tolerability study
- Combination of naïve and rollover patients from ElevAAATe
- Minimum treatment duration of 3 years
- ~40 US, AUS, NZ sites

- n=36 INBRX-101 at 120 mg/kg Q4W & placebo on non-dosing weeks
- n=36 INBRX-101 at 120 mg/kg Q3W & placebo on non-dosing weeks
- n=18 pdAAT at approved dose of 60 mg/kg QW
- n=130 INBRX-101 120 mg/kg Q3W
INBRX-101: AATD market opportunity

Severe* AATD cases
*defined as <11 μM serum AAT levels

US PI*ZZ & PI*SZ AATD market is growing at ~15% annually and projected to grow to $4B due to increased diagnosis.

Commercial viability of therapy requires abundant supply only available via INBRX-101

Sources: KOL interviews, Sandhaus chronic obstr pulm dis 2016; Barjaktarevic and Miravitlles BMC pulm med 2021
Clinical data and established guidelines exist for AAT therapy in acute GvHD

### Existing clinical data for Jakafi: Current standard of care
#### 2L (steroid resistant) acute GVHD (aGVHD)

<table>
<thead>
<tr>
<th>Efficacy</th>
<th>Ruxolitinib, Incyte¹ (n=49)</th>
<th>Fred Hutch/Baxalta² (n=12)</th>
<th>U of Michigan/CSL³ AAT +/- Prednisone Ph2 (n=40)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ORR (%) at day 28 (per CIBMTR)</td>
<td>28/49 (57%)</td>
<td>8/12 (67%)</td>
<td>26/40 (65%)</td>
</tr>
<tr>
<td>CR (%) at day 28</td>
<td>15/49 (31%)</td>
<td>4/12 (33%)</td>
<td>14/40 (35%)</td>
</tr>
<tr>
<td>OS</td>
<td>51% at 6 months</td>
<td>6/12 alive</td>
<td>45% at 6 months</td>
</tr>
</tbody>
</table>

#### Safety (n=71)
- Grade 3+ AEs: 97.2%
- Most Frequent AEs: + Anemia: 64%
  + Thrombocytopenia 62%
  + Neutropenia 48%
- Incidence of Infection: 80%
- Dosing: 5-10 mg twice daily

### Existing clinical data for plasma-derived AAT therapies
#### 2L (steroid resistant) aGVHD

<table>
<thead>
<tr>
<th>Efficacy</th>
<th>Ruxolitinib, Incyte¹ (n=49)</th>
<th>Fred Hutch/Baxalta² (n=12)</th>
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</tr>
<tr>
<td>OS</td>
<td>51% at 6 months</td>
<td>6/12 alive</td>
<td>45% at 6 months</td>
</tr>
</tbody>
</table>

#### Safety (n=71)
- Grade 3+ AEs: 0%
- Most Frequent AEs: “No clinical apparent toxicity in any patient” 2 d/c due to lack of efficacy
  “well tolerated with no infusion reactions or drug-related grade 3 to 4 toxicity”
- Incidence of Infection: 0 |
- Dosing: 90 mg/kg loading dose followed by either 30 or 60 mg/kg every other day
  60mg/kg per day every four days

### Current Guidelines for aGVHD⁵

<table>
<thead>
<tr>
<th>National Comprehensive Cancer Network (NCCN)</th>
<th>Ruxolitinib (category 1)</th>
<th>Alectuzumab</th>
<th>Alpha-1 antitrypsin</th>
<th>Anti-thymocyte globulin</th>
<th>Basiliximab</th>
<th>Calcineurin inhibitors</th>
<th>Etanercept</th>
</tr>
</thead>
<tbody>
<tr>
<td>European Society for Blood and Marrow Transplantation (EBMT)</td>
<td>Alectuzumab</td>
<td>Alpha-1 antitrypsin</td>
<td>Basiliximab</td>
<td>Cellular therapies</td>
<td>Daclizumab</td>
<td>Extracorporeal photopheresis</td>
<td>Faecal microbiota transplantation</td>
</tr>
</tbody>
</table>


### Active Phase 2/3 studies sponsored by CSL Behring
- The safety and efficacy of alpha-1 antitrypsin (AAT) for the prevention of graft-versus-host disease (GVHD) in patients receiving hematopoietic cell transplant (MODULAAE) (NCT03805789)³
- Treatment of GVHD in hematopoietic stem cell transplant (HSCT) recipients using AAT plus corticosteroids (CS) compared with corticosteroids alone (NCT04167514)⁴

Unlike other existing options, INBRX-101 is expected to be combinable with other therapies due to its clean safety profile.
Goal:
To develop a more precise DR5 agonist able to selectively induce apoptosis in tumor cells

Previous generation

Empirically selected tetravalent DR5 agonist that restricts unwanted secondary clustering

DR5 agonists with limited on target effect or unwanted off tumor toxicity

Inhibrx solution
INBRX-109: a next generation DR5 agonist with an optimized balance of efficacy and safety

Death Receptor 5 (DR5 / TRAIL-R2) is highly expressed on tumorigenic, transformed or damaged cells but not normal cells, making DR5 a promising therapeutic target in oncology\(^1\)-\(^4\)

INBRX-109 characteristics:

- **Tetravalent**: Empirically designed to simultaneously engage four DR5 molecules
- **Immunogenic Epitopes removed**: Prevents unwanted higher order clustering via anti-drug antibodies
- **Effector Disabled Fc**: Prevents higher order clustering and allows for antibody-like PK
- **Smaller Size**: sdAb backbone limits molecule size (106 kDa) which may allow for better tumor penetration

---

INBRX-109 is a potent inducer of extrinsic cell death via the DR5 pathway

**DR5 (TRAIL-R2)** is a pro-apoptotic receptor for TRAIL that is widely expressed on the surface of damaged, transformed or tumor cells, but rarely and at low levels on normal cells.1-4 TRAIL selectively induces programmed cell death via activation of the FADD and downstream caspase pathway, therefore playing an important role in tumor and viral immune surveillance.5

While the DR5 trimer is the minimal functional unit for TRAIL activity, clustering of multiple receptors at the cell-cell interface results can generate more potent apoptotic activity.6-8

INBRX-109, a tetravalent DR5 agonist, is designed to simultaneously engage four DR5 molecules to drive enhanced clustering/signaling in tumor cells while minimizing off-target effects.

---

INBRX-109 is precision-engineered for optimal potency and safety

Valency drives both DR5-induced tumor cell death and hepatocyte destruction

**Impact of valency on DR5-mediated cell death**

- Impact of valency on cell death:
  - Graph showing the effect of valency on cell death with different test articles.

- Impact of valency on cell death:
  - Bar graph showing EC50 values for different valencies with INBRX-109 tetravalent and Hexavalent DR5 hexavalent.

**Impact of valency on hepatotoxicity**

- Graph showing hepatocyte viability (% Cell death) with different test articles and valencies.

- Bar graph showing the impact of different valencies on hepatotoxicity.

- Graph showing the effect of Ab (nM) on hepatocyte viability.

- Graph showing the impact of test articles and valencies on hepatocyte viability.

- Notes on valency and DR5-mediated cell death and hepatocyte destruction.
INBRX-109: Phase 1 trial design

Study of INBRX-109 in patients with locally advanced or metastatic solid tumors, including sarcomas

### Part 1

**INBRX-109 single-agent dose escalation**

- **n=20**
- All comers 3+3 design evaluating doses of 0.3 to 30 mg/kg.
- INBRX-109 was well tolerated; MTD was not reached
- 3 mg/kg selected as RP2D

### Part 2

**INBRX 109 single-agent dose expansion**

- **n=116**

<table>
<thead>
<tr>
<th>Tumor Type</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colorectal adenocarcinoma</td>
<td>20</td>
</tr>
<tr>
<td>Gastric adenocarcinoma</td>
<td>10</td>
</tr>
<tr>
<td>Malignant pleural mesothelioma</td>
<td>20</td>
</tr>
<tr>
<td>Chondrosarcoma</td>
<td>20</td>
</tr>
<tr>
<td>Synovial sarcoma</td>
<td>10</td>
</tr>
<tr>
<td>IDH1/2-mutant conventional chondrosarcoma</td>
<td>12</td>
</tr>
<tr>
<td>Nonconventional chondrosarcoma</td>
<td>12</td>
</tr>
<tr>
<td>Solid tumors, BMI &gt;30</td>
<td>20</td>
</tr>
</tbody>
</table>

### Part 3

**Dose expansion with chemotherapy**

- **n=100**

<table>
<thead>
<tr>
<th>Tumor Type</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mesothelioma with carboplatin or cisplatin</td>
<td>10</td>
</tr>
<tr>
<td>Mesothelioma with carboplatin and pemetrexed or cisplatin and pemetrexed</td>
<td>20</td>
</tr>
<tr>
<td>Colorectal adenocarcinoma with FOLFIRI</td>
<td>20</td>
</tr>
<tr>
<td>Pancreatic adenocarcinoma 2L with fluorouracil and irinotecan (mFOLFIRI)</td>
<td>20</td>
</tr>
<tr>
<td>SDH-def solid tumors or GIST with temozolomide</td>
<td>20</td>
</tr>
<tr>
<td>Ewing sarcoma 2-4L with irinotecan and temozolomide</td>
<td>20-50</td>
</tr>
<tr>
<td>Colorectal adenocarcinoma with FOLFIRI</td>
<td>20</td>
</tr>
</tbody>
</table>

Ongoing
Encouraging mPFS and clinical responses observed in Chondrosarcoma patients treated with INBRX-109

Impact of valency on DR5-mediated cell death

Overall median PFS: 7.6 months (range, 0.03-17.8 mo) vs. <4 months historically\(^1-3\)

PFS by Kaplan-Meier analysis

Among evaluable chondrosarcoma patients (n=31), the disease control rate (DCR) was 87.1% (27/31)
- 2 patients achieved PR (objective response rate, 6.5%), and 25 patients SD (80.6%)
- Of those who experienced SD, 13 (52.0%) had decreases from baseline in tumor size
Although no approved agents, current guideline recommended treatment options\(^4\) have demonstrated modest activity as measured by RECIST criteria
-ivosidenib resulted in no CR or PR, an SD rate of 52% in patients with advanced chondrosarcoma (n=21)\(^5\)
-pazopanib in patients with unresectable or metastatic conventional chondrosarcoma (N=47) reported a DCR of 43% at week 16\(^6\)

Selected case reports

Partial response:
- 29-year-old white male, histologic Grade 3
- 61% decrease in target lesions (RECISTv1.1)
- Patient was on study for 45 weeks

Stable disease:
- 55-year-old white male, histologic Grade 3
- 24% decrease in target lesions (RECISTv1.1)
- Patient was on study for 77 weeks

---

INBRX-109 Phase 2 registration enabling study

**Primary endpoint:**
Progression free survival

**Secondary endpoints:**
Overall survival, quality of life, overall response rate, duration of response, disease control rate, safety, etc.

### Patients
Conventional chondrosarcoma, Grades 2 and 3, unresectable or metastatic

Randomization stratified by line of therapy, Grade and IDH1/2 mutation status

<table>
<thead>
<tr>
<th>INBRX-109</th>
<th>Placebo</th>
</tr>
</thead>
<tbody>
<tr>
<td>n=134*</td>
<td>n=67*</td>
</tr>
</tbody>
</table>

3 mg/kg every three weeks

**INBRX-109 for the treatment of unresectable and metastatic conventional chondrosarcoma**
+ FDA fast track designation and orphan-drug designation
+ EMA orphan-drug designation

Completion projected 2H 2024

*Including interim analysis*
INBRX-109 in combo with IRI/TMZ in metastatic, unresectable Ewing sarcoma

**Efficacy**

- Disease control rate was 76.9%, or 10 out of 13 patients as measured by RECISTv1.1.
- 7 patients who achieved partial responses (53.8%), 5 of which were observed in classical EWS patients (71.4%) and 2 of which were observed in RCS patients (33.3%).

**Safety**

- Most common adverse events (diarrhea, nausea, and fatigue) were consistent with the known safety profile of IRI/TMZ.
- One patient had increased alanine aminotransferase (grade 1); no other liver-related AEs were reported.

**Data cutoff:** September 8, 2023.

CR, complete response; DCR, disease control rate; EWS, Ewing sarcoma; ORR, objective response rate; PD, progressive disease; PR, partial response; RCS, round cell sarcoma; SD, stable disease; AE, adverse event.

* Patient discontinued treatment to undergo tumor resection surgery. * One patient had not reached the first set of restaging scans and was considered nonevaluable. * Durable clinical benefit was defined as having SD, PR, or CR for >6 months. ** Best response prior to progression is displayed; however, if a patient’s first scan result was progressive disease, then that result is displayed. EWS, Ewing sarcoma; RCS, round cell sarcoma.
INBRX-106
hexavalent OX40 agonist

Goal:
To develop a potent OX40 agonist able to induce robust signal activation

Bivalent OX40 agonists elicit weak downstream signals with limited clinical activity

Previous generation

Hexavalent OX40 agonist with enhanced clustering/signaling

InhibitRx solution
INBRX-106: generating robust OX40 signaling to drive anti-tumor activity

INBRX-106 is designed to boost anti-tumor T-cell activity by potently activating the OX40 co-stimulatory pathway

**INBRX-106 characteristics:**

- **Hexavalent**: Simultaneously engage multiple OX40 to drive enhanced clustering/signaling
- **Non-Competitive Binding**: Complements natural ligand (OX40L) activity
- **Effector Enabled**: Facilitates higher order clustering
- **Smaller Size**: sdAb backbone limits molecule size (129 kDa) which may allow for better tumor penetration

**Six OX40 sdAbs**
- Designed to agonize OX40 while allowing endogenous OX40L binding

**IgG Fc**
- Effector enabled Fc facilitates higher order clustering and greater downstream OX40 pathway signaling
INBRX-106: mechanism of action

Effective OX40 agonism potentiates the body’s immune response towards a tumor

The T-Cell Receptor (TCR) recognizes a tumor-associated antigen presented via MHC. In response, OX40 is upregulated on tumor reactive TILs facilitating an immune response directed towards the tumor.

Due to its six OX40 sdAb domains, binding of hexavalent INBRX-106 facilitates higher order clustering versus bi-valent and tri-valent endogenous OX40L amplifying the downstream costimulatory signal.

Costimulatory signaling via OX40 and the TCR-MHC receptors induce survival and proliferation of activated antigen-specific CD4 T cells, increases memory T cell generation, CD8+ effector T cells, and suppress the inhibitory capacity of regulatory T cells.
Higher OX40 valency drives superior T cell activation and reduces $T_{reg}$ suppression

**CD4 T Cell Co-Stimulation**

- Hexavalent INBRX-106 engagement drives superior co-stimulation of both CD4 and CD8 T-cells versus bi-valent OX40 mAbs
- INBRX-106, but a not bivalent OX40 mAb, reduces regulatory T-cell ($T_{reg}$) mediated suppression of effector T-cells ($T_{eff}$)

**Reversal of $T_{reg}$ Suppression**

- ~2-fold increase vs bi-valent and untreated

---

1. T-cell activation monitored by assessing CD71 expression following suboptimal anti-CD3-mediated stimulation
2. Reversal of Treg suppression assessed by CD25 upregulation following anti-CD3 stimulation
Valency drives OX40 agonism in CPI-resistant tumor models

**Syngeneic B16F10 Mouse Tumor Model**

- OX40 agonism upregulates PD-L1 expression on CD4 and CD8 T-cells supporting rationale of combination with anti-PD1 agents
- Hexavalent INBRX-106-a* demonstrated single-agent, single-dose activity in checkpoint-inhibitor responsive and resistant syngeneic tumor models
- INBRX-106-a* induced more robust anti-tumor activity as single agent and in combination with anti-PD1

**Table: CRs**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Vehicle</th>
<th>Bivalent OX40 mAb</th>
<th>Hexavalent INBRX-106-a*</th>
</tr>
</thead>
<tbody>
<tr>
<td>CRs</td>
<td>0/10</td>
<td>0/10</td>
<td>3/10</td>
</tr>
<tr>
<td>Anti-PD1 Alone</td>
<td>0/10</td>
<td>1/10</td>
<td>6/10</td>
</tr>
<tr>
<td>Anti-PD1 + Bivalent OX40 mAb</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anti-PD1 + Hexavalent INBRX-106-a*</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*INBRX-106 mouse surrogate*
INBRX-106 study design

Phase 1/2 study of single agent INBRX-106 and INBRX-106 in combination with pembrolizumab in patients with locally advanced or metastatic solid tumors

### Part 1

**INBRX-106 single-agent dose escalation**

- **n=20**

### Part 2

**INBRX-106 single-agent dose expansion**

- **n=52-64**
  - Basket Dose Regimen #1
  - Basket Dose Regimen #2
  - **NSCLC PD-L1+ (TPS ≥ 50%) or TMB high**
  - Basket Dose Regimen #3

### Part 3

**Dose escalation with pembrolizumab**

- **n=21**
  - Complete

### Part 4

**Dose expansion with pembrolizumab**

- **n=136**
  - Basket PDL1+
  - NSCLC PDL1+
  - Ongoing

### NSCLC TMB or PDL1 high

- **Dose Regimen #1**
- **Dose Regimen #2**
- Pembrolizumab Monotherapy
- **Basket HNSCC, PDL1 + (CPS ≥1)**
- **Basket, MMR-def/ MSI-high**
- **Uveal melanoma**

---

Durable responses with anti-PD-1 in CPI-refractory patients across multiple tumor types

Well-tolerated with mild or moderate immune-related toxicities

* Cohorts highlighted in green are actively recruiting

| a | Alternating treatment: INBRX-106 0.3 mg/kg followed by pembrolizumab 400 mg/kg 3 weeks later; Alternates every three weeks
| b | Priming: INBRX-106 0.3 mg/kg 0.1 mg/kg Q3W + pembrolizumab
| c | Melanoma (cutaneous or uveal) and HNSCC (NPC or non-NPC); Currently only HNSCC sub cohorts are being prioritized and are enrolling
INBRX-105

tetravalent
PD-L1 targeted
4-1BB agonist

**Goal:**
Restrict potent 4-1BB agonism to areas of high PD-L1 expression

Indiscriminate 4-1BB activation leads to a narrow therapeutic window limited by hepatotoxicity

Previous generation therapy

Localized 4-1BB agonist specific to PD-L1+ tissues

Inhibrx solution
INBRX-105: localizing and potentiating the anti-cancer effects of the 4-1BB pathway

INBRX-105 designed to boost anti-tumor T-cell activity in PD-L1 expressing tissues

Two PD-L1 sdAbs to target and locally antagonize PD-1 interaction
Two 4-1BB sdAbs to conditionally agonize 4-1BB while allowing 4-1BBL binding

FC
Fc engineered to minimize effector function

INBRX-105 characteristics:

- Bispecific/Conditional Agonist: Designed to co-express PD-L1 and 4-1BB in order to confer PD-L1 dependent 4-1BB agonism
- Localization: Targeted to the tumor microenvironment in order to minimize hepatotoxicity
- Non-Competitive Binding: Complements natural ligand (4-1BBL) activity
- Smaller Size: sdAb backbone limits molecule size (105 kDa) which may allow for better tumor penetration
INBRX-105 mechanism of action

**PD-L1 targeted 4-1BB agonism amplifies anti-tumor response and localizes T-cell activity**

The T-Cell Receptor (TCR) recognizes a tumor-associated antigen presented via MHC. This drives upregulation of 4-1BB on tumor reactive TILs to facilitate an immune response directed towards the tumor.

By crosslinking 4-1BB at sites of high PD-L1 expression, INBRX-105 increases 4-1BB agonism to enhance T-cell survival, activation, and target killing localized to the tumor microenvironment.

Costimulation of 4-1BB leads to downstream activation on effector cells, including increased proliferation, cytotoxicity, memory generation and possible reversal of exhaustion.
INBRX-105 shows improved T cell modulation over PD-L1 and 4-1BB agents alone or in combination

**T Cell Co-stimulation**

**Peptide-induced TCR signal**

<table>
<thead>
<tr>
<th>Antibody</th>
<th>IFN(\gamma)+/IFN(\alpha)+/IL-2+ cell counts</th>
</tr>
</thead>
<tbody>
<tr>
<td>INBRX-105</td>
<td><img src="image1" alt="Bar chart showing IFN(\gamma)+/IFN(\alpha)+/IL-2+ cell counts for INBRX-105, Urelumab, Atezolizumab, Urelumab + Atezolizumab, and No antibody." /></td>
</tr>
<tr>
<td>Urelumab</td>
<td><img src="image1" alt="Bar chart showing IFN(\gamma)+/IFN(\alpha)+/IL-2+ cell counts for INBRX-105, Urelumab, Atezolizumab, Urelumab + Atezolizumab, and No antibody." /></td>
</tr>
<tr>
<td>Atezolizumab</td>
<td><img src="image1" alt="Bar chart showing IFN(\gamma)+/IFN(\alpha)+/IL-2+ cell counts for INBRX-105, Urelumab, Atezolizumab, Urelumab + Atezolizumab, and No antibody." /></td>
</tr>
<tr>
<td>Urelumab + Atezolizumab</td>
<td><img src="image1" alt="Bar chart showing IFN(\gamma)+/IFN(\alpha)+/IL-2+ cell counts for INBRX-105, Urelumab, Atezolizumab, Urelumab + Atezolizumab, and No antibody." /></td>
</tr>
<tr>
<td>No antibody</td>
<td><img src="image1" alt="Bar chart showing IFN(\gamma)+/IFN(\alpha)+/IL-2+ cell counts for INBRX-105, Urelumab, Atezolizumab, Urelumab + Atezolizumab, and No antibody." /></td>
</tr>
</tbody>
</table>

**T Cell Co-stimulation**

**MHC mismatch-induced TCR signal**

![Graph showing IL-2 ng/mL against Antibody (nM) for INBRX-105, Urelumab, Atezolizumab, Urelumab + Atezolizumab, and No antibody.](image2)

- **Co-stimulation with INBRX-105 following TCR engagement yields superior T cell cytokine production.**
- **INBRX-105 is more potent and induces enhanced cytokine production than PD-L1 blockade and 4-1BB agonism alone or in combination.**

* Analog of Urelumab was synthesized based on publicly disclosed sequences.
1 - PBMC stimulated with CEF (Cytomegalovirus-, Epstein-Barr- and Influenza-Virus) peptide mix, cytokine production measured by FluoroSpot.
2 - Mixed-lymphocyte reaction between cells from two MHC/HLA-mismatched donors, cytokine production measured by ELISA.
Localizing 4-1BB agonism to the PD-L1-rich tumor microenvironment leads to potent anti-tumor activity in mouse models

Enhanced T Cell Infiltration to TME
Day 7

Dramatic increase in T cell frequency in the tumor microenvironment on Day 7 post dose of INBRX-105-a*

INBRX-105-a* drives complete responses in PD-L1+ mouse tumor models (5/8 CRs)

* Mouse reactive INBRX-105 surrogate.
1 - SD, IV C57BL/6, female. N=8/group.
INBRX-105 study design

An open-Label, multicenter, dose-escalation, Phase 1/2 study of INBRX-105 and INBRX-105 in combination with pembrolizumab in patients with locally advanced or metastatic solid tumors

**Part 1**
- Single agent dose escalation
- Complete
- n=32

**Part 2**
- Single agent dose expansion
- 2a n=32
- NSCLC TPS >50%
- Cutaneous Melanoma or solid tumor
- HNSCC
- In preliminary data, single agent CRs and PRs observed in CPI r/r patients

**Part 3**
- Dose escalation with pembrolizumab
- Complete
- n=30

**Part 4**
- Dose expansion with pembrolizumab
- Complete
- n= 50
- NSCLC TPS ≥ 50%
- Melanoma
- HNSCC CPS ≥ 1%, MSI/TMB-high solid tumors
- NSCLC TPS 1-49%
- HNSCC CPS ≥ 50%

* Cohort highlighted in green is actively recruiting
* CPS of ≥20 may be allowed if the cohort is expanded; CPI-naive patients with PC may be eligible if CPIs are not the current standard of care for the specific indication or treatment setting
* Includes HNSCC of the larynx, hypo-/pharynx, and sinus

**Signals in Part 1/2a led to added single-agent expansion cohorts in HNSCC**

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