Innovation Driven

Outcomes Focused

April 2023

INHIBRX

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Why Inhibrx Should Be Front and Center on Your Radar

Four Biologic Programs in the Clinic

All are demonstrating clinical activity with multi-billion-dollar potential peak sales. Two registration-enabling programs are underway in 1H 2023.

Active Discovery Process

Six programs are expected to enter the clinic over the next three years.

Opportunities to Both Partner Assets and Commercialize In-house

We believe our assets appeal to major pharmaceutical companies as licensing targets. We are building the infrastructure to internally execute on small commercial footprint opportunities.

Our Interests are Aligned With Our Investors

Credible base of major holders and ~30% internally owned. Potential to become financially self-sustaining within 18 months with no debt and possible return of capital to investors through share buy-backs and/or special dividends.

Innovative Approach to Biologic Therapeutic Discovery & Development

Settlers, Not Pioneers

- Best targets are known and already explored, but what is not known is how to drug them properly
- We learn through prior clinical failures and therapeutic liabilities
- We only move programs into the clinic that are first or best-in-class with a clear path to point of concept

+

Broad Internal Expertise

- The modular nature of our platforms and technologies combined with our protein engineering expertise allow for rapid exploration of therapeutic approaches
- Teams with deep biologics drug development expertise across translational sciences, clinical, technical operations, manufacturing and commercial

=

Success Where Others Have Failed

- First company to optimize and develop recombinant AAT
- First company to overcome DR5 agonism toxicity challenges and advance into registration-enabling study
- First company to find a therapeutic window for 4-1BB

Our Clinical Therapeutic Candidates

	Preclinical	Phase 1	Registration-enabling
INBRX-101- *AATD* *Our Recombinant Alpha-1-Anti-trypsin Fc-Fusion Protein (AAT-Fc)*	●	●	
INBRX-109 *Our Tetravalent DR5 Agonist*	●	●	
INBRX-105 *Our Tetravalent PD-L1 targeted 4-1BB Agonist*	●		○
INBRX-106 *Our Hexavalent OX40 Agonist*	●		○

- **INBRX-101- *AATD***
  - + No innovation for 30+ years and multiple recombinant AAT failures
  - + Favorable safety and tolerability profile w/potential to achieve normal AAT levels with monthly dosing
  - + Initiation of registration-enabling trial in April 2023; Potential path to launch in 2026; $3B+ peak sales potential
- **INBRX-109**
  - + Prior DR5 agonist efforts failed due to limited activity or hepatotoxicity
  - + At-risk population for severe liver toxicity now identified and screening criteria protocol updated
  - + Data from registration-enabling trial expected in 2H 2024 with possible path to approval in chondrosarcoma in 2025; $1B peak sales potential and exploring expansion into other indications
- **INBRX-105**
  - + We believe we are the first to find a therapeutic window for 4-1BB agonism
  - + Based on incoming clinical data, we are growing more confident this could be a blockbuster drug, potentially as a single agent
  - + Data update in 2H 2023 with potential for registration studies to start early next year
- **INBRX-106**
  - + OX40 is a validated target but no one has been able to build a viable multivalent antibody
  - + Clinical data looks promising with durable single agent activity
  - + Substantial data update in 1H 2024 with registration studies as early as 2H 2024

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Our Upcoming Therapeutic Candidates

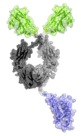
All potential best-in-class with differentiated profiles

INBRX-101- *GvHD*

Our Recombinant Alpha-1-Anti-trypsin Fc-Fusion Protein (AAT-Fc)

- De-risked opportunity with promising clinical data with pdAAT
- Potential to reach >$3B peak sales in Acute GvHD and Chronic GvHD
- Clinical trial(s) to initiate 2H 2023

INBRX-121



Our NK cell targeted IL-2

- NKp46 targeting with engineered IL-2 drives selective NK cell expansion and enhancement of cytotoxicity capacity
- Phase 1 trial could initiate in Q4 2023

Other Programs on the Horizon

- FcRN Antagonist
- Radiopharmaceuticals
- T-cell Engagers - ContraMAB® Platform
- γδ T-cell Targeted Cisleukin™ Molecule

Partnerships with Industry Leaders

PARTNER	FOCUS
Bristol Myers Squibb	CD47 checkpoint inhibitor
Chiesi — People and ideas for innovation in healthcare	Option to Ex-North America rights to INBRX-101 AATD
2seventybio	Use of INBX sdAb platform for certain cell therapy products for up to 13 programs
MERCK	Merck-supplied Keytruda for INBRX-106 Phase 1 combo trial
ARROWMARK PARTNERS	Joint venture with ArrowMark affiliate, Phylaxis Bioscience, LLC: license of IP and know-how to develop certain compounds
Elpiscience	Greater China rights to INBRX-105 and INBRX-106

*Does not include academic partnerships, product development arrangements and various other collaborations

Key financial highlights

$274M	Cash and cash equivalents
> 30%	Internal ownership
43.6M	Common stock outstanding
49.1M	Fully diluted outstanding
~130	Employees

*As of 12/31/2022

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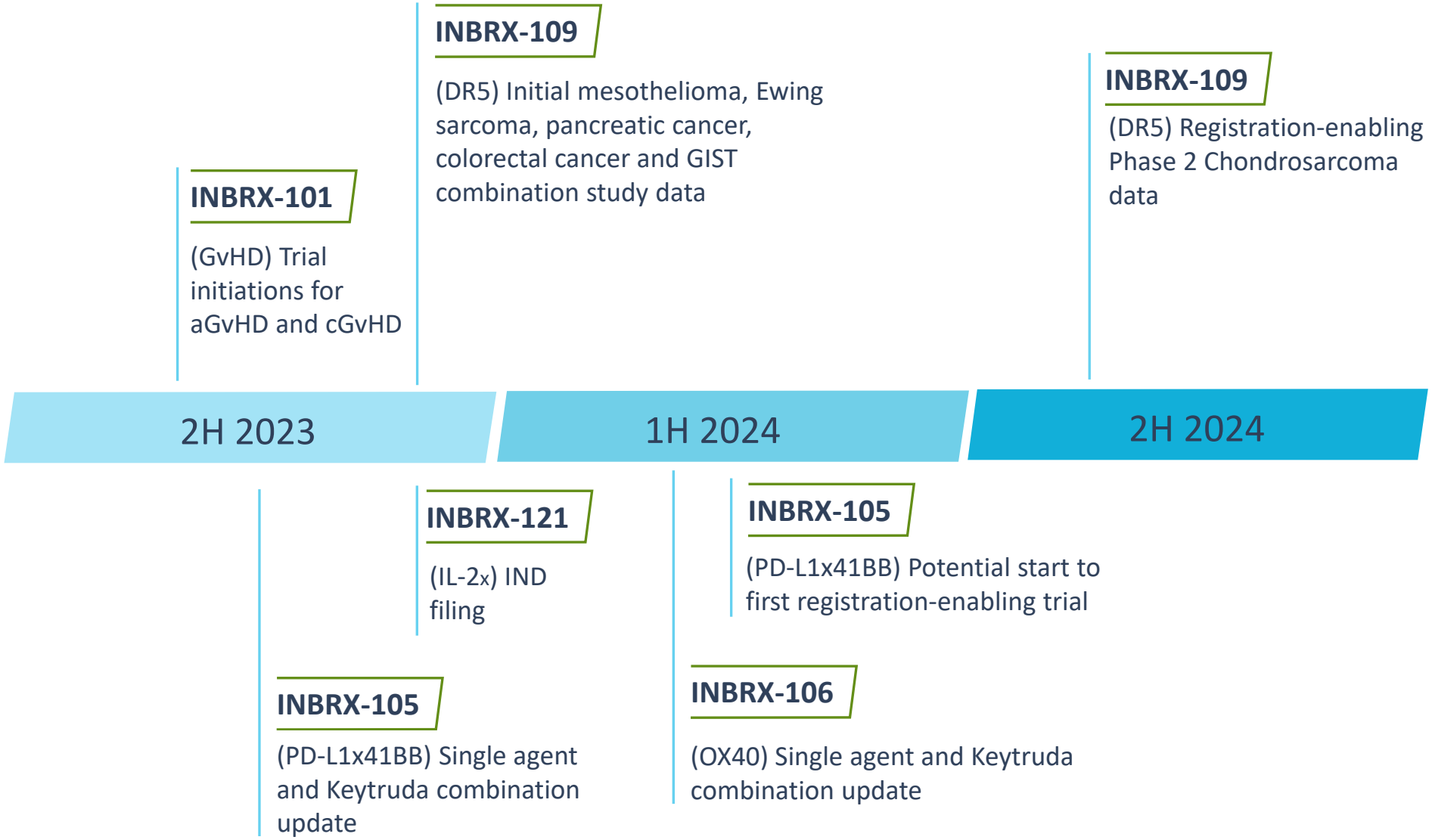
Near Term Expected Clinical Milestones



INBRX-101

Recombinant Alpha-1
Antitrypsin Fc-fusion Protein

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Alpha-1 Antitrypsin Deficiency (AATD)

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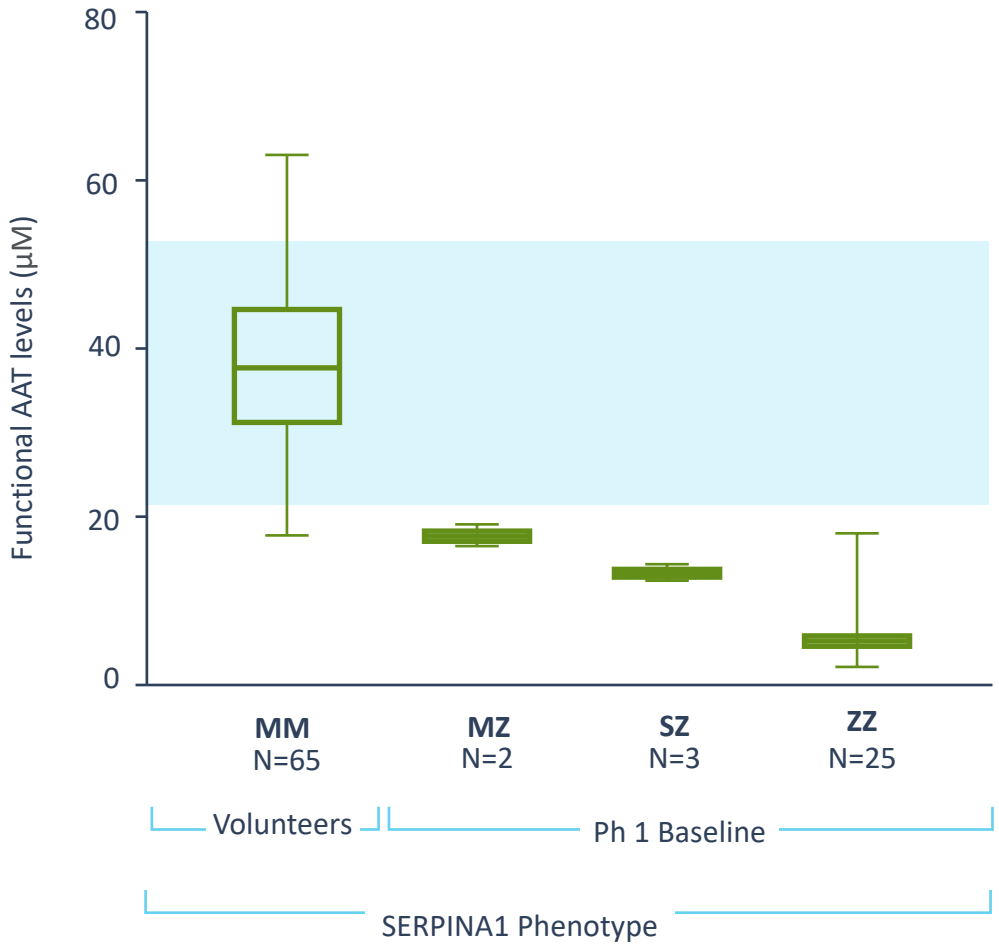
Functional AAT Levels in Healthy Individuals vs. AATD Patients



Disease history

- Alpha-1 antitrypsin deficiency (AATD) is an inherited orphan respiratory disease characterized by deficient levels of alpha-1 antitrypsin (AAT)
- This causes loss of lung function and decreased life expectancy
- A small percentage of patients also develop liver disease

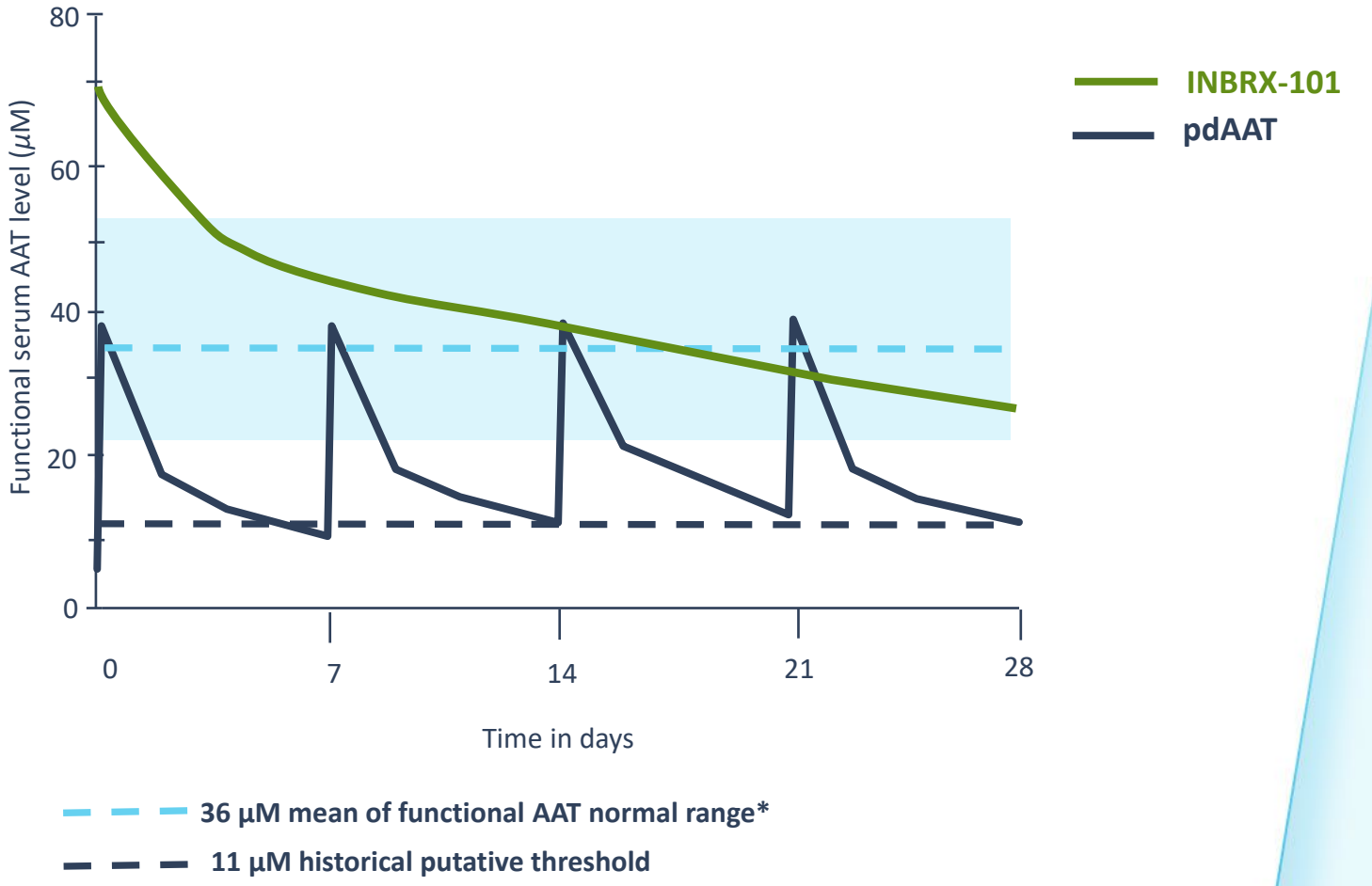
Study results

- Functional AAT levels from 65 MM genotype healthy volunteers ranged from 21 to 54 micromolar (µM), with a mean of 36 µM.
- Baseline levels of functional AAT for 30 Phase 1 patients prior to dosing of INBRX-101 ranged from 2 to 18 µM, with a median of 4.7 for ZZ genotype patients.

- *Box plots show the minimum, lower quartile, median, upper quartile and maximum*
- *The shaded region represents the 5th-95th percentiles of the normal range of functional AAT in healthy MM genotype adults*
- *AAT variant determination was conducted by the Mayo Clinic Laboratories using an LC-MS/MS method (A1ALC)*
- *The Phsae 1 baseline data represents the functional AAT levels measured in patients at the beginning of the study prior to dosing INBRX-101*

Current Standard of Care Does Not Maintain Normal AAT Levels

Functional AAT serum levels at steady-state (INBRX-101 vs. pdAAT)



INBRX-101

+ INBRX-101, dosed ***every four weeks*** at 120 mg/kg, is predicted to maintain patients above the lower threshold of the normal range and achieve an average level (C_{avg}) of functional AAT that approximates that of healthy MM genotype adults.*

+ The current standard of care, plasma-derived AAT (pdAAT)**, dosed ***once weekly*** at 60 mg/kg, achieves C_{avg} of functional AAT of 17.8 µM over the weekly dosing interval as calculated from steady-state area under the curve (AUC) values***. Due to its short half-life, patients require weekly infusions to achieve target levels, but levels typically fall below the normal range within 1-2 days of infusion.

**Source~ Normal range calculated based on Inhibrx ANEC assay results from 65 healthy MM genotype adults*
***Current pdAAT therapies include: Aralast, Glassia, Prolastin-C & Zemaira*
****Source~ reported in Stocks et al. BMC Clinical Pharmacology 2010, 10:13*

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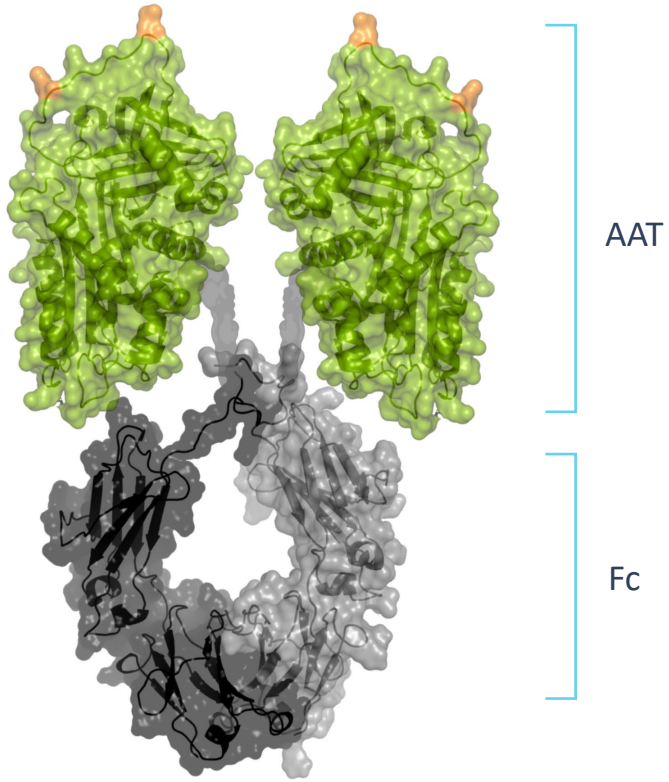
Potential Advantages of Recombinant AAT Fc-fusion Protein

Potential to extend
the dosing interval
from weekly to monthly

Has demonstrated potential to
maintain patients in normal
functional AAT range

Recombinant manufacturing
provides abundant supply
with no pathogen risk



INBRX-101 - Topline Results from Phase 1, Part 2

PART 2

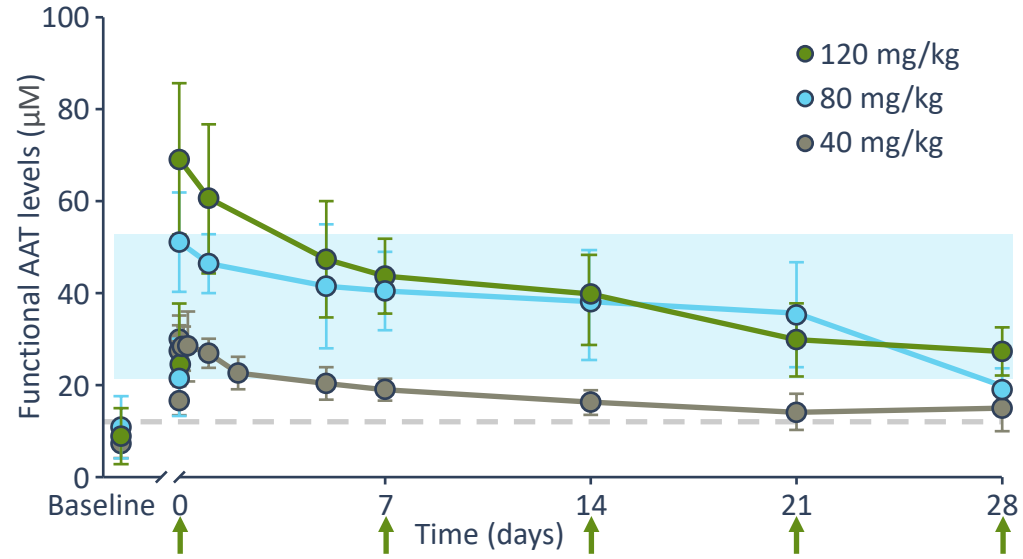
Multiple ascending dose escalation (MAD)

Complete

N=18		
N=6	40 mg/kg	
N=6	80 mg/kg*	
N=6	120 mg/kg*	

* bronchoalveolar lavage

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



↑ Indicates timing after third dose

* Baseline values shown at Day 0

- 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
- Revealed potential to achieve and maintain normal functional AAT levels with monthly dosing

- Significant accumulation observed following each MAD dose in-line with the prolonged terminal elimination half-life of INBRX-101
- 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
- Functional AAT levels at Day 70 (28 days following the 3rd dose), on average, were within the normal range for the 120 mg/kg dose level

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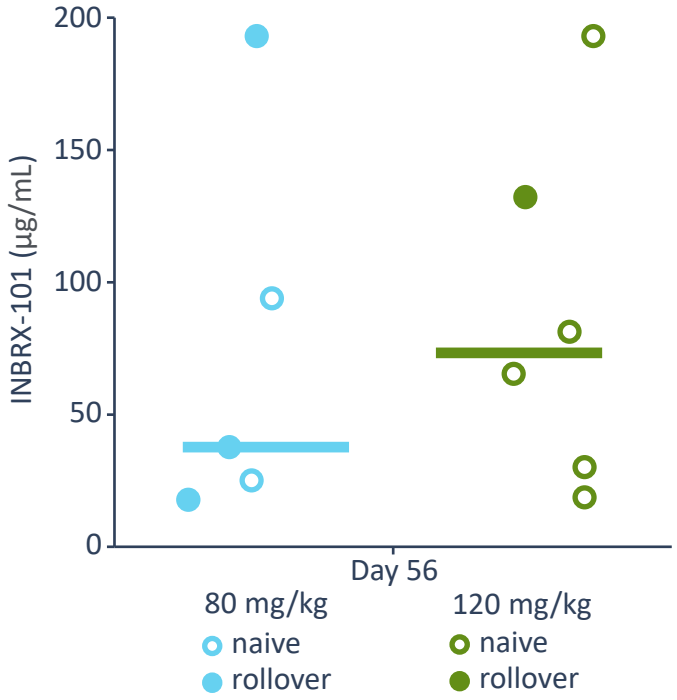
INBRX-101 is Present in the Lung in Every Patient Sampled Following IV Dosing

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)[1] 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

- At baseline, BALF samples from subjects that rolled over from the Part 1 SAD[2] 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*
- *Data is preliminary and has not been fully verified*

[1] One 80 mg/kg patient did not have a post-dose sample collected
[2] In rollover patients, baseline collection was at least 84 days after the SAD

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INBRX-101 AATD Registration-enabling Trial

Study INBRX101-01-201: ElevAATe

ELEVAATE

Main Eligibility Criteria

- Adult patients aged 18-75 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)

Initiated

- Randomized, controlled, double-blind
- Head-to-head superiority study: INBRX-101 vs. pdAAT
- 32-week treatment period
- ~30 US and AUS sites

N=36	INBRX-101 at 120 mg/kg Q3W & placebo on non-dosing weeks
N=36	INBRX-101 at 120 mg/kg Q4W & placebo on non-dosing weeks
N=18	pdAAT at approved dose of 60 mg/kg QW

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 ($C_{trough,ss}$)

Key Secondary Endpoints: INBRX-101 vs pdAAT: mean change in fAAT concentration from baseline to fAAT avg concentration at steady state ($C_{avg,ss}$), 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: ElevAATe-OLE (Open Label Extension)

ELEVAATE OLE

- Open label, long-term safety and tolerability study
- Combination of naïve and rollover patients from ElevAATe
- Minimum treatment duration of 3 years
- ~35 US, AUS, NZ sites

N=130 — INBRX-101 120 mg/kg Q3W

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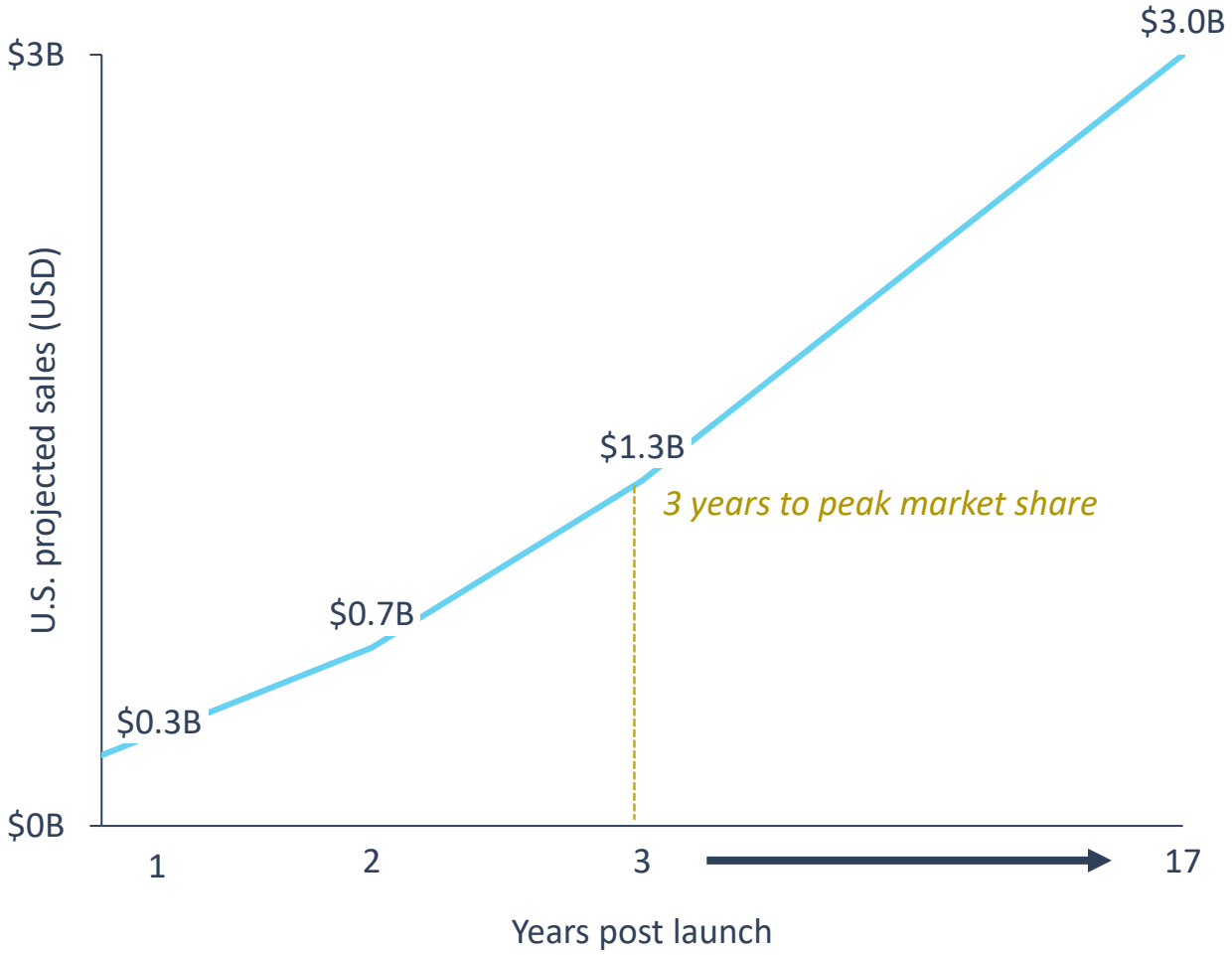
INBRX-101 has the Potential to Achieve ~$3B in Annual U.S. Revenue with Expected Rapid Uptake in Patients with Severe AATD

INBRX-101 top line projected U.S. sales & key assumptions



Key assumptions

- "Severe (ZZ/SZ)" AATD patients (same as pdAATs today)
- 7% CAGR throughout forecast period (conservative estimate given CAGR of ~17% from 2016 to 2020)
- ~75% peak market share
- 3-year time to peak share
- Price parity with current pdAATs* & 2% annual price growth
- Little to no generic erosion due to high barriers to entry

Notes: *Pricing assumption is for modelling purposes only
Source: KOL Qualitative Interviews (n=~25); EvaluatePharma, Datamonitor, IQVIA, Fortune Business Insights, Analog analysis of other recombinant products

INBRX-101 has the Potential to Shift the Treatment Paradigm, Expanding Augmentation Therapy to a Broad Group of AATD Patients in the U.S.

	TODAY	FUTURE
	PI*ZZ or PI*SZ	PI*ZZ or PI*SZ
U.S. prevalence	~100K	~100K (same as today)
Treatment rate	~8-10%	~40% (driven by increased diagnosis rates)
Total treated U.S. patients	~8K	~40K
Market revenue potential	~$1 Billion	~$4 Billion

Key Takeaways for AATD Market:

+ pdAATs only utilized for severe AATD patients and market is still worth ~$1B today despite only ~8-10% treatment rate
+ PI*ZZ & PI*SZ AATD market is growing at ~17% annually and projected to grow to $4B due to increased diagnosis
+ Upside market potential from earlier intervention of augmentation therapy, which can help to prevent lung decline
+ Commercial viability and expansion of augmentation therapy use requires abundant supply only available via INBRX-101

"The results of the RAPID trial stress the importance of early intervention. Patients who started augmentation late were unable to regain lung tissue lost during placebo treatment and did not 'catch up' to patients who started augmentation early." – U.S. KOL

Sources: KOL Interviews, 1. Sandhaus Chronic Obstr Pulm Dis 2016; 2. Barjaktarevic and Miravitlles BMC Pulm Med 2021

INBRX-101

Graft versus Host Disease (GvHD)

Strong 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)

		Ruxolitinib, Incyte[4] (n=49)
Efficacy	ORR (%) at day 28 (per CIBMTR)	28/49 (57%)
	CR (%) at day 28	15/49 (31%)
	OS	51% at 6 months
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

		Fred Hutch/Baxalta [1] Ph1/2 (n=12)	U of Michigan/CSL [2] AAT +/- Prednisone Ph2 (n=40)
Efficacy	ORR (%) at day 28 (per CIBMTR)	8/12 (67%)	26/40 (65%)
	CR (%) at day 28	4/12 (33%)	14/40 (35%)
	OS	6/12 alive	45% at 6 months
Safety	Grade 3+ AEs	**0%**	**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	13/40 (32.5%) Through 30 days
	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 aGVHD[5]

National comprehensive cancer network (nccn)	Ruxolitinib (category 1)	Alemtuzumab	**Alpha-1 antitrypsin**	Anti-thymocyte globulin	Basiliximab	Calcineurin inhibitors	Etanercept
European society for blood and marrow transplantation (ebmt)	Alemtuzumab	**Alpha-1 antitrypsin**	Basiliximab	Cellular therapies	Daclizumab	Extracorporeal photopheresis	Faecal microbiota transplantation

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Two 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 (MODULAATE) (NCT03805789)[3]
- Treatment of GVHD in hematopoietic stem cell transplant (HSCT) recipients using AAT plus corticosteroids (CS) compared with corticosteroids alone (NCT04167514)[3]

Sources:

[1] Response of Steroid-Refractory Acute GvHD to a1-Antitrypsin, Marcondes et at, 2016. http://dx.doi.org/10.1016/j.bbmt.2016.05.011

[2] a1-Antitrypsin infusion for treatment of steroid-resistant acute graft-versus-host disease, Magenau et al, 2018. http://ashpublications.org/blood/article-pdf/131/12/1372/1405639/blood815746.pdf

[3] https://clinicaltrials.gov/

[4] https://www.jakafi.com/pdf/prescribing-information.pdf, https://ashpublications.org/blood/article/135/20/1739/452638/Ruxolitinib-for-the-treatment-of-steroid

[5] Listed in alphabetical order and not comprehensive of all consensus recommendations for steroid-refractory GVHD.

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INBRX-101 has the Potential for Fast Entry into Acute GVHD, Expanding to Prophylaxis, Achieving >$1bn in the US Market

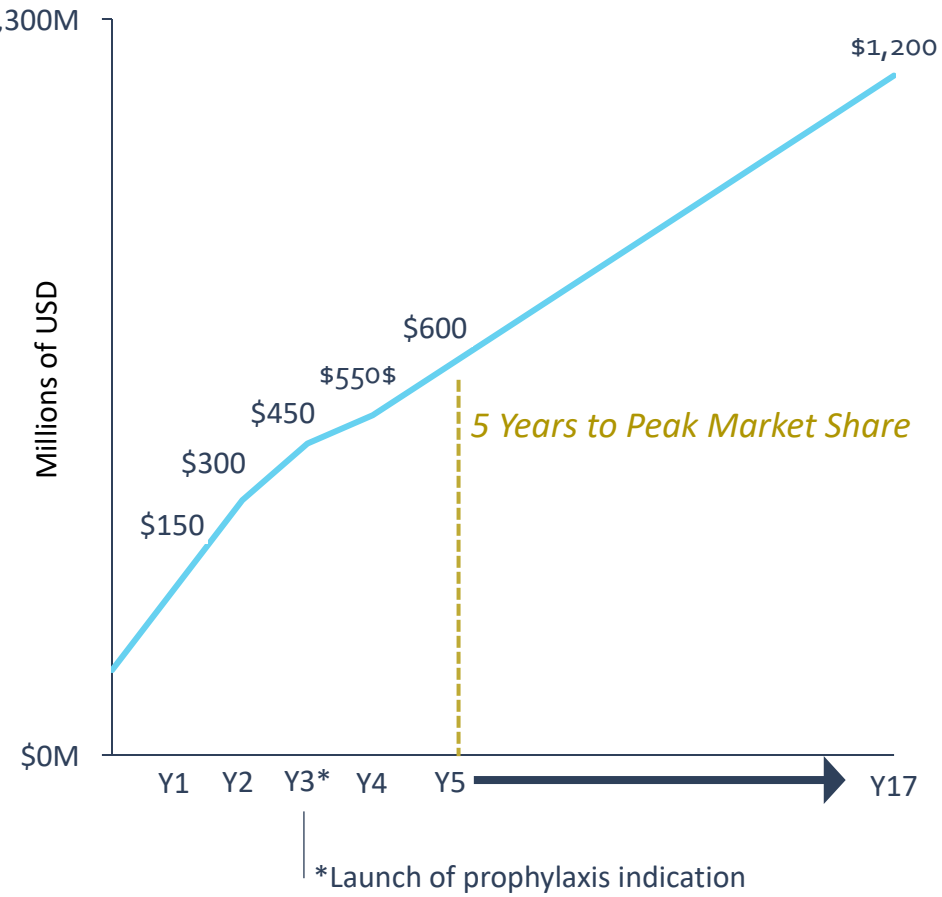
INBRX-101 acute & prophylaxis GvHD top line projected US sales & key assumptions

Potential advantages over pdAAT

+ Potential for sustainable dosing schedule
+ Potential for transformational efficacy at higher dose
+ Ability to price in-line with current standard of care while pdAAT would be more expensive due to COGS and the high dose required
+ Near elimination of pathogenic risk for immunocompromised patients

Potential advantages over standard of care

+ Expected superior safety benefits with potential for greater efficacy



Key assumptions:

+ TPP: Superior safety, efficacious in prophylaxis, same efficacy in acute
+ Patients: Allogeneic stem cell transplants (2.3% annual growth), Acute 2L+, high risk prophylaxis (30%)
+ Share: Acute 2L: 50%, 3L: 70%, Prophylaxis: 21%
+ Time to peak: 2 years for acute and prophylaxis (5 years peak total)
+ Price: In-line with approved GvHD branded agents, 2.5% annual growth

Upside potential:

+ Potential for transformational efficacy at high dose
+ Safety profile could enable broad combinability across current and future therapies, including 1L

INBRX-101

Sources:
Qualitative research, third-party analysis, HRSA Blood Stem Cell (https://bloodstemcell.hrsa.gov/data)

INBRX-101 GvHD Expansion Opportunities

U.S. chronic GvHD – progression beyond steroids

US Prevalence (2022)	~16,000 [6]
Extensive disease requiring therapy (50%) [4]	8,000
Progression beyond Steroids (71%) [4]	5,700

~$2bn

2030 US Market Opportunity

- + Less competitive future market landscape
- + Longer therapy duration requires reduced toxicity and steroid-sparing agents
- + Favorable safety profile enables opportunity for combining with standards of care
- + Long half-life enables sustainable long-term utilization of 101 relative to pdAAT therapies

Prophylaxis, acute, & chronic GvHD – EU / Japan

Europe & Japan Allogeneic Transplants	21,000 [2, 3]

>$3bn

2030 EU & JP Market Opportunity [5]

- + Favorable pricing and reimbursement as compared to typical European/Japanese standards due to high mortality and significant unmet medical need
- + There were 9,400[1] allogeneic transplants in the U.S. market; ex-US transplant market represents a large global opportunity

Sources:

[1] 2019 figures from HRSA Blood Stem Cell (https://bloodstemcell.hrsa.gov/data)

[2] 2017 figures for 40 European countries and 10 related countries: https://www.nature.com/articles/s41409-019-0465-9

[3] 2020 figures from JDCHCT https://drive.google.com/file/d/16Vv8k1aHTMc0KbmOHGiUmEk4rFGwgBEy/view?usp=drive_web

[4] Qualitative research, third-party analysis and current therapy pricing research, HRSA Blood Stem Cell (https://bloodstemcell.hrsa.gov/data).

[5] Assumes pricing corridor 50% smaller than the US

[6] Epidemiology and Treatment of Chronic Graft-versus-Host Disease Post-Allogeneic Hematopoietic Cell Transplantation: A US Claims Analysis, Bachier, et. al., 2021

INBRX-109

Tetravalent DR5 Agonist

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A Next Generation DR5 Agonist with an Optimized Balance of Efficacy and Safety

- **Death Receptor 5 (DR5/TRAIL-R2)** has been a target of interest in oncology due to the differential sensitivity of cancerous cells over healthy cells to TRAIL-mediated killing.[1-5] DR5 is a key receptor for TRAIL-induced apoptosis of unwanted, damaged, virally infected and transformed cells[6]
- Previous generation DR5 agonists have been ineffective due to poor clustering or led to unintended apoptosis in normal hepatocytes likely due to unwanted hyperclustering[7]

Our Engineering Goal:

Design a DR5 agonist that can selectively induce enhanced apoptosis in tumor cells

Our Solution:

Tetravalent DR5 agonist empirically designed to simultaneously engage four DR5 molecules to drive enhanced clustering/signaling in tumor cells while minimizing off-target effects

Four DR5 sdAbs
with key immunogenic epitopes removed

Smaller than conventional mAb
may allow for better tumor penetration

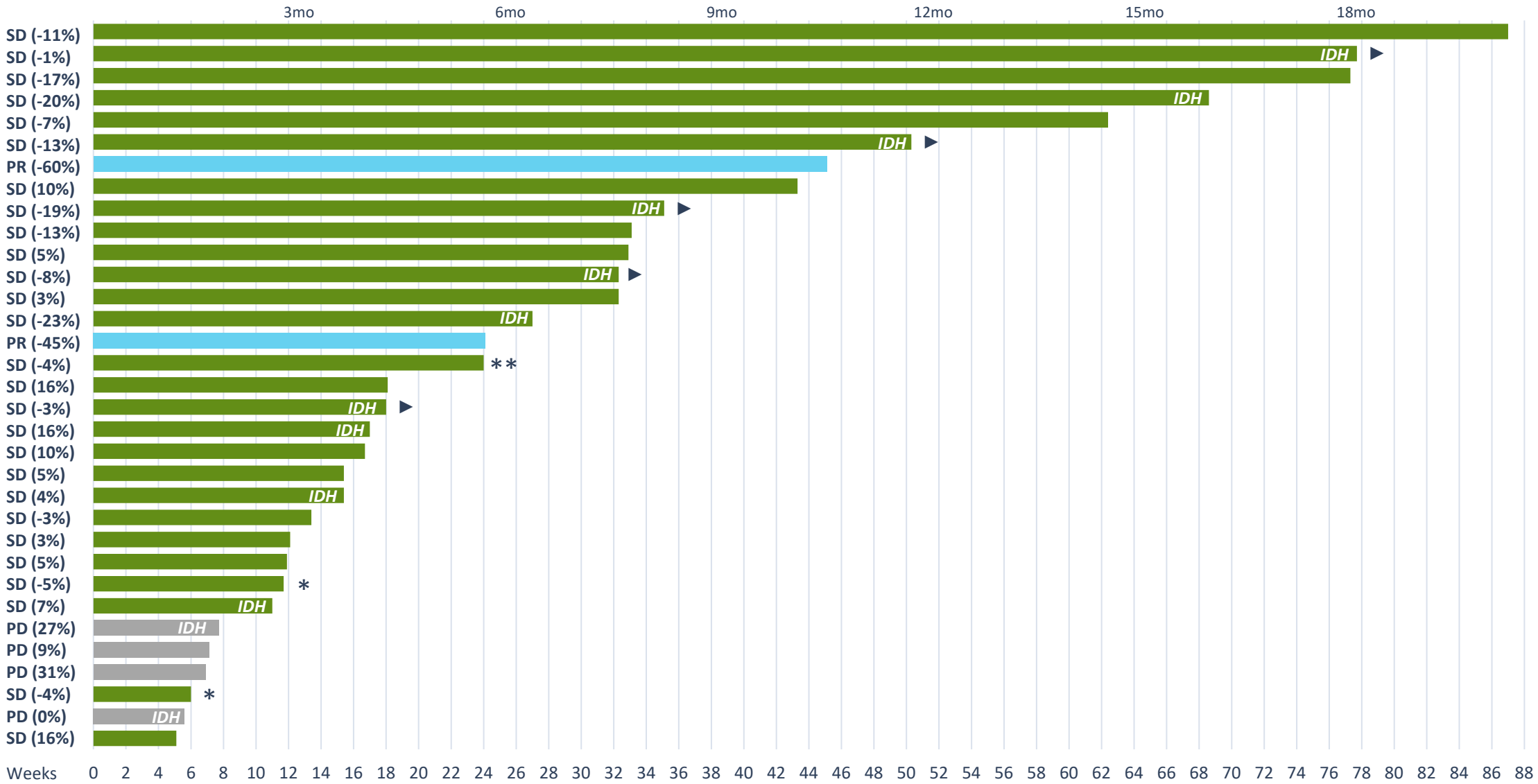
Engineered Fc
Minimize effector function

106 kDa

1. Ashkenazi A, et al. *J Clin Invest*. 1999;104(2):155-162. 2. Walczak H, et al. *Nat Med.* 1999;5(2):157-163. 3. Valley CC et al. *J Biol Chem*. 2012;287(25):21265-21278.
4. Pan L, et al. *Cell*. 2019;176(6):1477-1489.e1414. 5. Nair PM, et al. *Proc Natl Acad Sci.* 2015;112(18):5679-5684. 6.Razeghian E, et al. *Front Immunol*. 2021;12:699746.
7. Papadopoulos KP, et al. *Cancer Chemother Pharmacol*. 2015;75(5):887-895.

Preliminary Phase 1 Data in Unresectable or Metastatic Conventional Chondrosarcoma

INBRX-109



- Data cut point **8-Nov-2022**, study ongoing
- Response per RECISTv1.1 per Investigator assessment, data subject to change (e.g., some data raw and not verified)

- PR=Partial Response, SD=Stable Disease, PD=Progressive Disease
- ▶ Patient still on treatment ***IDH-*** isocitrate dehydrogenase (IDH1/IDH2) mutant
- *Off-study per subject request (e.g., resection) or **Investigator discretion

INHIBRX

INBRX-109 Phase 2 Registration-enabling Study Design in Chondrosarcoma

ChonDRAgon

Randomization
Conventional chondrosarcoma, Grades 1, 2 and 3, unresectable or metastatic

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

Ongoing

INBRX-109	Placebo
N=134*	N=67 *
3 mg/kg every three weeks	Until PD or toxicity with cross-over to INBRX-109

ENDPOINTS

Primary: Progression free survival

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

2H 2024

- \+ No approved systemic therapeutic for the treatment of chondrosarcoma
- \+ FDA Fast Track designation and orphan-drug designation in unresectable and metastatic conventional chondrosarcoma

PFS from other placebo-controlled chondrosarcoma studies

Therapeutic	IPI-926 (HH)
Control arm	Placebo
Subject number	100 (2:1)
Placebo arm	**Median PFS 2.9 months**

Source~ CTOS 2013 Wagner et al.

Therapeutic	Regorafenib
Control arm	Placebo
Subject number	46 (2:1)
Placebo arm	**Median PFS~ 2 months**

Source~ European Journal of Cancer 2021 Florence Duffaud et al.

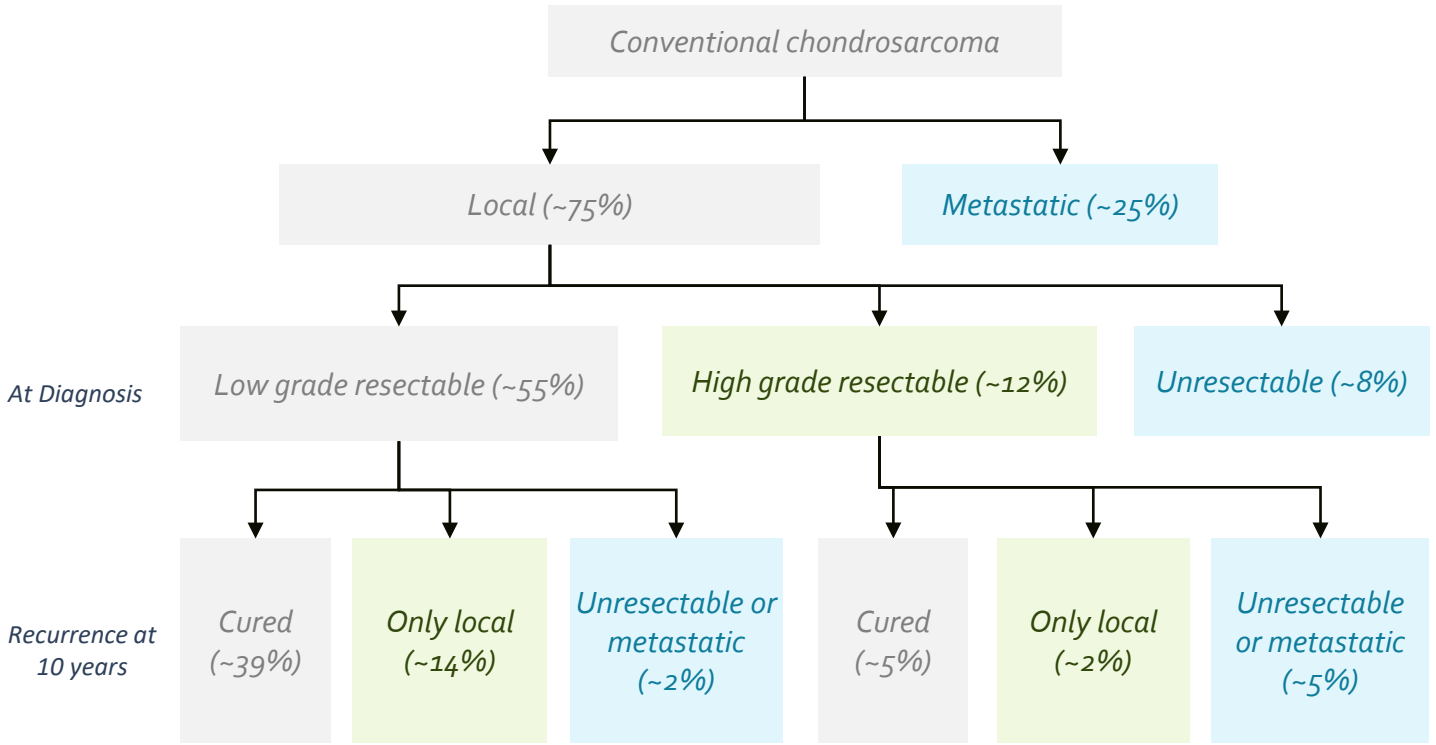
*Including interim analysis

INHIBRX

Many Patients with Local Disease Eventually Progress to Unresectable or Metastatic Chondrosarcoma, Providing an Annual Prevalent Patient Pool of ~2.5K in the U.S.

Conventional chondrosarcoma patient flow

Legend:
- Unresectable or metastatic population ***(opportunity at launch)***
- Peri-operative opportunity in high-risk patients ***(growth opportunity)***



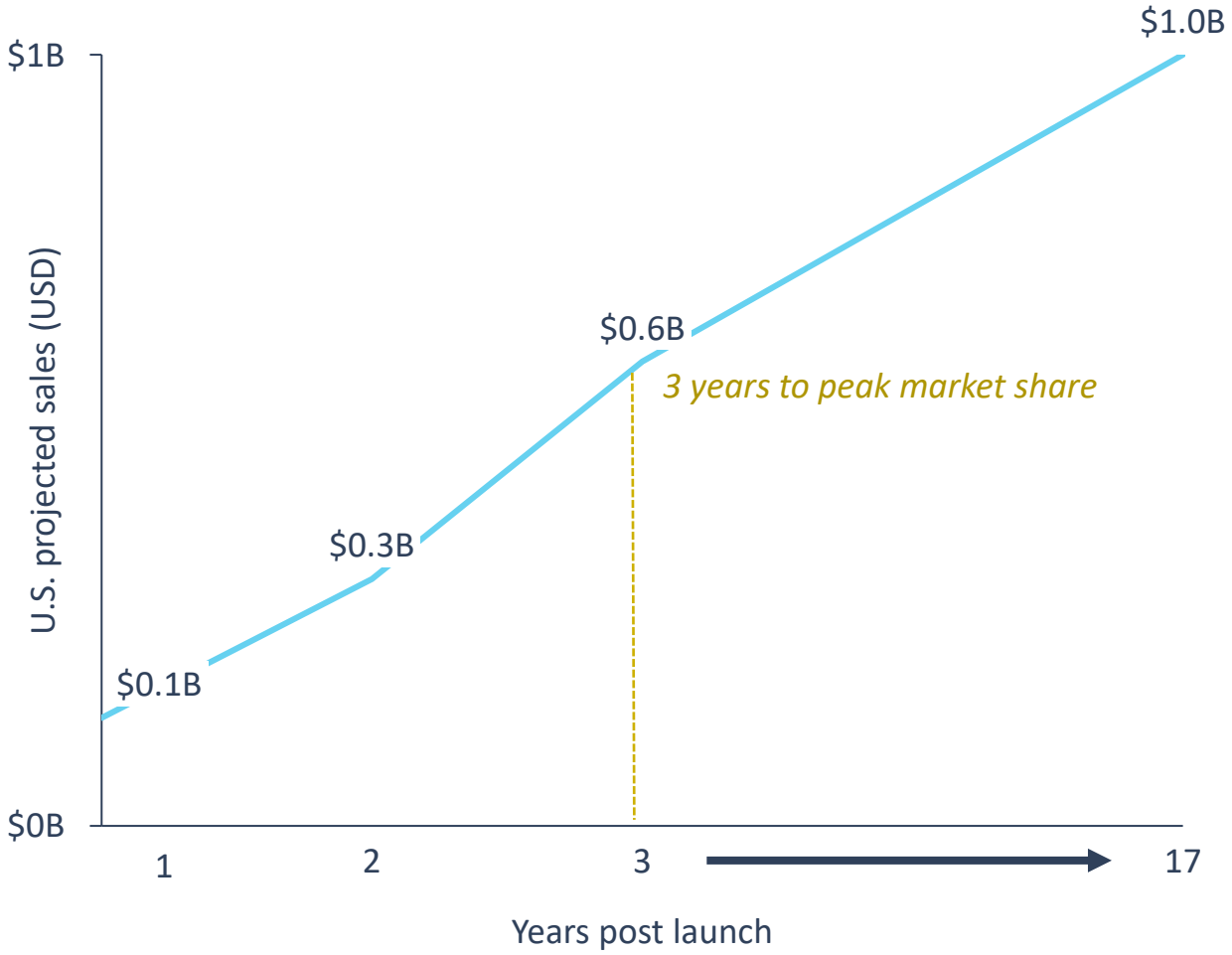
KEY TAKEAWAYS

+ Unresectable or metastatic opportunity estimated at **~2.5K** patients in the U.S. today vs. reported incidence of ~1.5K given tendency for local disease to progress to unresectable or metastatic
+ Longer term growth opportunity in the peri-operative setting for high-risk patients with **~1.6K** prevalent patients in the U.S. today

Sources: KOL Interviews (n=20); Kythera claims database (~60% of all U.S. insurance claims); Inhibrx secondary research; Hua et al., Treatment Method and Prognostics…, 2020

Based on Current Trends and Lack of Approved Options, INBRX-109 has the Potential to Achieve ~$1B in Annual Revenue with Rapid Uptake Post Launch

INBRX-109 top line projected U.S. sales in the unresectable/metastatic setting & key assumptions



Key assumptions

- \+ ~375K* annual price per patient
- \+ ~85% peak share
- \+ ~30% 10-year recurrence rate for local, low-grade patients
- \+ ~65% 10-year recurrence rates for local, high-grade patients

Incremental growth opportunity

- \+ Peri-operative setting provides an incremental **~$500M** annual opportunity in the U.S. alone

Notes: *Pricing assumption is for modelling purposes only based on analogous market research for oncology products in rare indications; Sources: KOL qualitative interviews (n=~20); Inhibrx secondary research; Hua et al., Treatment Method and Prognostics…, 2020

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INBRX-109 on the Horizon

PART 3

Combination studies

Ongoing

N=20	Pancreatic adenocarcinoma 2nd line with mFOLFIRI
N=20	Ewing sarcoma with Irinotecan + Temozolomide
N=20	Colorectal Cancer with FOLFIRI
N=20	SDH-deficient GIST with Temozolomide

Data releases: 2H 2023

POTENTIAL FUTURE OPPORTUNITIES

- **Solid Tumors**
  - + IAP antagonists
  - + Targeted therapies
  - + Checkpoint inhibitors
  - + Selective kinase inhibitors
  - + Additional combo agents
- **Hematologic tumors**
  - + Bcl-2 inhibitors
  - + Proteosome inhibitors
  - + Additional combo agents
- **Additional sarcoma indications**

INBRX-105

PD-L1 x 4-1BB Multispecific

Localizing and Potentiating the Anti-cancer Effects of the 4-1BB Pathway

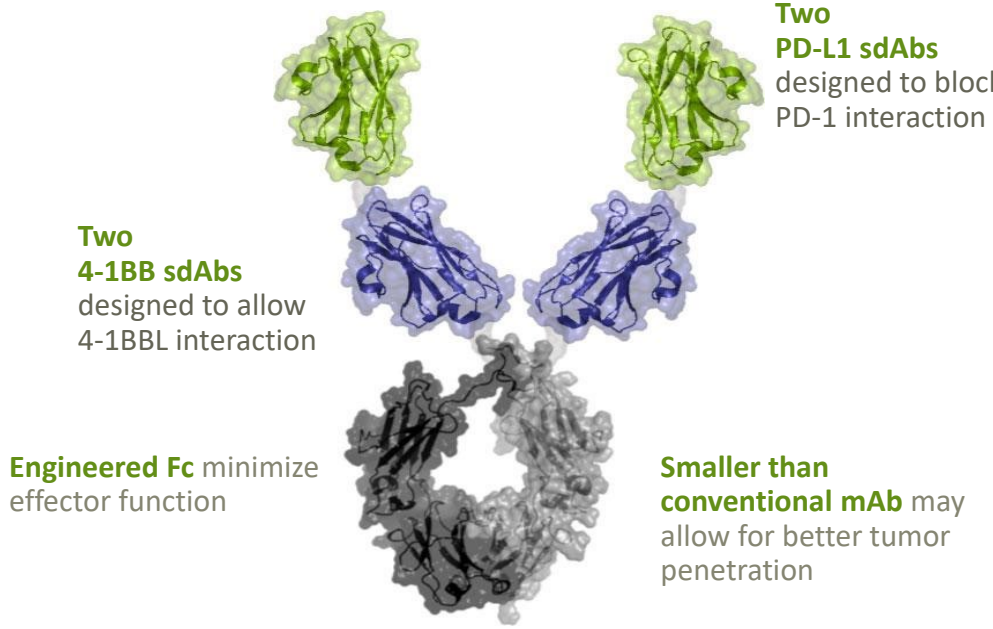
- 4-1BB (CD137/TNFRS9) is a member of the tumor necrosis factor (TNF) receptor superfamily and is an attractive target for immunotherapy due to its increased expression on tumor reactive TILs[1]
- Agonistic 4-1BB mAbs have shown promising anti-tumor activity in early clinical studies[2,3]
- However, systemic activation of 4-1BB has led to a narrow therapeutic window limited by toxicity[2,4]

Our Engineering Goal:

Design a 4-1BB agonist with an optimized therapeutic index

Our Solution:

Tetravalent PD-L1 and 4-1BB bispecific antibody that localizes the 4-1BB costimulatory effect to a PD-L1 rich tumor microenvironment



1. Kim AM, et al. Front Oncol 2022;10.3389. 2. Chester C, et al. Blood 2018;131:49–57. 3. Etxeberria I, et al. ESMO Open 2020;4:e000733. 4. Bartkowiak T, et al. Front Oncol 2015;5:117.

INBRX-105 is a Potential Best-in-class 4-1BB Agonist

PD-L1 x 4-1BB Bispecifics

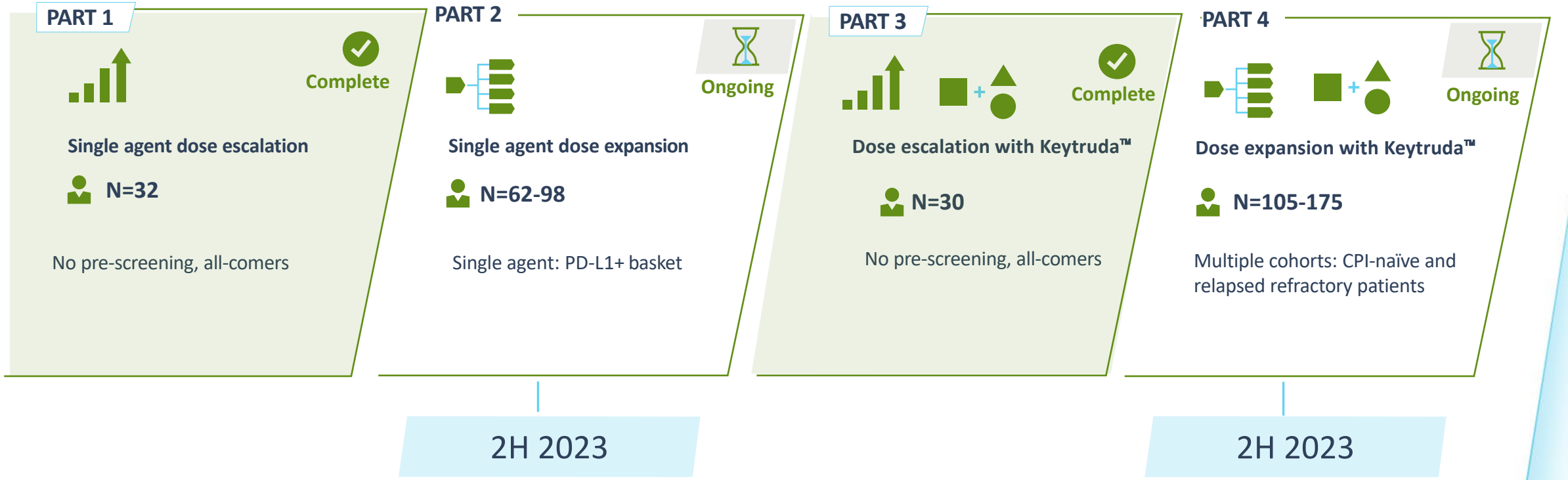
CANDIDATE	FORMAT	4-1BBL BLOCKING
INBRX-105	**Bivalent/Bivalent**	**No**
Gen-1046	Monovalent/Monovalent	n/a
MCLA-145	Monovalent/Monovalent	Yes
FS222	Bivalent/Bivalent	n/a
PRS-343	Bivalent/Bivalent	No
ND021	Monovalent/Monovalent	n/a

Monoclonal 4-1BB Antibodies

CANDIDATE	IGG SUBCLASS	4-1BBL BLOCKING
Urelumab	IgG4	Yes
Utomilumab	IgG2	No
CTX-471	IgG4	No
ADG106	IgG4	Yes
ATOR-1017	IgG4	Yes
AGEN2373	IgG1	No
LVGN6051	unknown	n/a

INBRX-105 has the Potential to be the First 4-1BB Agonist with a Robust Therapeutic Window

Phase 1 Trial Design



+ Therapeutic window observed in the CPI-refractory population with responses both in single agent and in combination with Keytruda
+ Single agent complete response observed

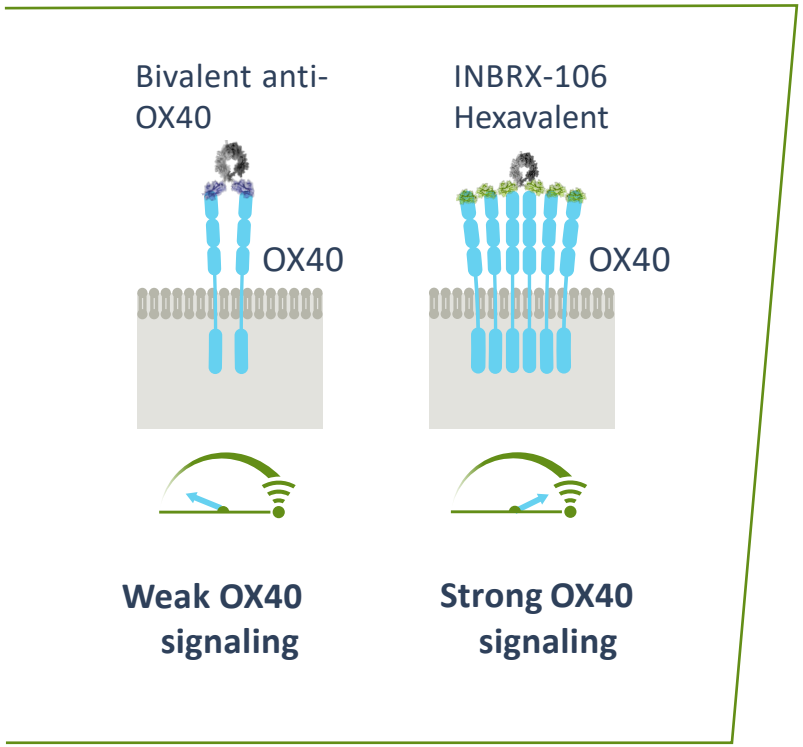
INBRX-106

Hexavalent OX40 Agonist

INHIBRX

INBRX-106 is a Potential Best-in-class OX40 Agonist

CANDIDATES	VALENCY	ISOTYPE	LIGAND BLOCKING
INBRX-106	**Hexa-**	**IgG1**	**N**
MOXR-0916	Bi-	IgG1	Y
GSK-3174998			
BMS-986178			
INCAGN-1949			
ABBV-368			
IBI-101			
MEDI-0562			
PF-04518600	Bi-	IgG2	Y
BGB-A445	Bi-	IgG1	N
BAT6026	Bi-	IgG1 afucosylated mAb	n/a



Phase 1 INBRX-106 Trial as a Single Agent and in Combination with Keytruda®

INBRX-106

Single agent

PART 1

INBRX-106 single-agent dose escalation

Complete

N=20

- 3+3 design
- Locally advanced or metastatic solid tumors
- All-comers

PART 2

INBRX 106 single-agent dose expansion

Complete

N=82-94

- 0.03 mg/kg (RP2D) at 2 dosing schedules (Q3W or Q9W) in tumor types responsive to CPIs

NSCLC
Melanoma
HNSCC
G/GEA
RCC

1H 2024

In combination

PART 3

Dose escalation with Keytruda

Complete

N=30

- Locally advanced or metastatic solid tumors
- mTPI design
- >3 subjects per dose level:
  - 0.01, 0.03, 0.1 and 0.3 mg/kg (Q3W)
- No prescreening; all-comers

PART 4

Dose expansion with Keytruda

Ongoing

N=170

- Relapsed or refractory to CPI
- PDL1 TPS ≥ 1% (NSCLC)
- PDL1 CPS ≥ 1% (PD-L1 basket)

PDL1⁺ NSCLC r/r
PDL1⁺ basket
PDL1⁺ cutaneous melanoma
PDL1⁺ uveal melanoma
PDL1⁺ HNSCC
PDL1+ nasopharyngeal carcinoma

1H 2024

CPI *r/r*

CPI naïve

Single agent

- 4/10 response evaluable NSCLC & melanoma patients with duration of stable disease* greater than 6 months (three CPI-exposed patients and one CPI-naïve uveal melanoma patient)
- Longest duration of stable disease was 2+ years (NSCLC patient refractory to Keytruda)**
- Well-tolerated with mild or moderate immune-related toxicities

In combination

- Durable responses with anti-PD-1 in CPI refractory patients across multiple tumor types
- Well-tolerated with mild or moderate immune-related toxicities

*per investigator assessment

**patient came off study May 12, 2022 after 112 weeks on treatment with INBRX-106

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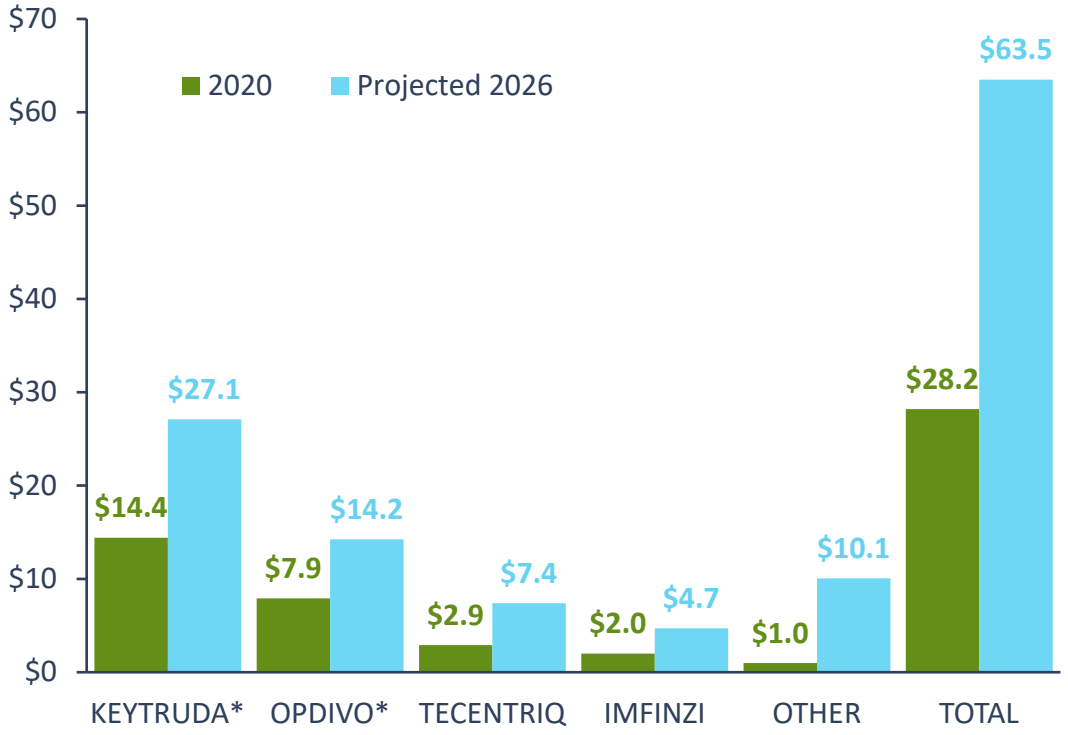
Potential Market Opportunity for INBRX-106 and INBRX-105

PD-1/PD-L1 WW Revenue

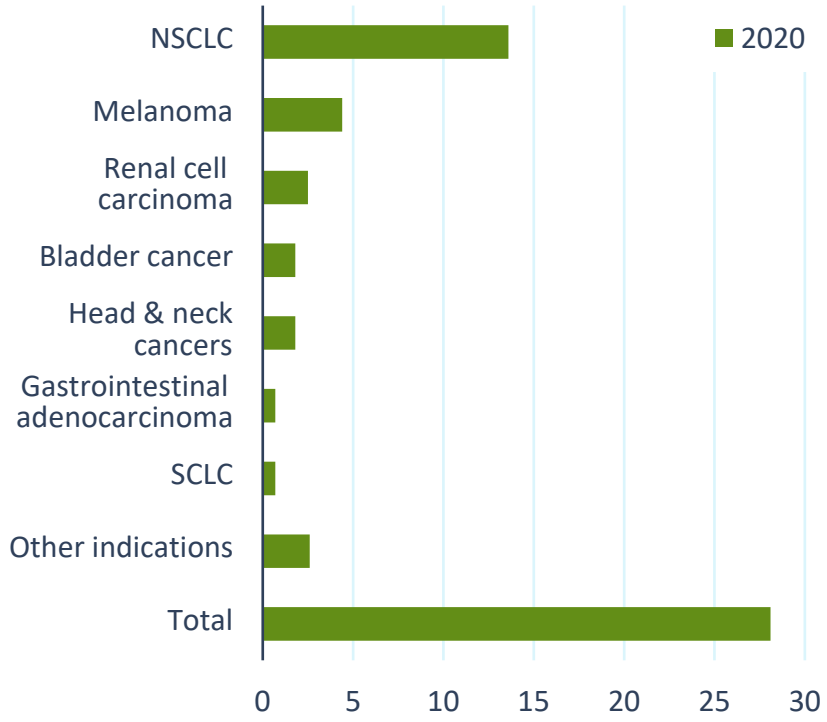
(in $ billions)



Keytruda and Opdivo® go off patent in 2028

PD-1/PD-L1 WW Revenue by Indication

(in $ billions)



The figures on this slide represent market research estimates from Evaluate Pharma

Cisleukin™ Platform

A Targeted Cis-IL2 Platform

INHIBRX

Targeting the Robust Anti-tumor Effects of IL-2 to Overcome Off-target Toxicity

- **IL-2 is a potent stimulator of cytotoxic cell types with natural anti-tumor activity** that has shown great promise as a single agent in multiple cancers[1]
- However, efforts to mitigate the toxicities of IL-2 therapy have been at the expense of anti-tumor efficacy restricting its therapeutic window

Our Engineering Goal:

Design a potent, targeted IL-2 able to widen the therapeutic window and minimize off-target toxicity

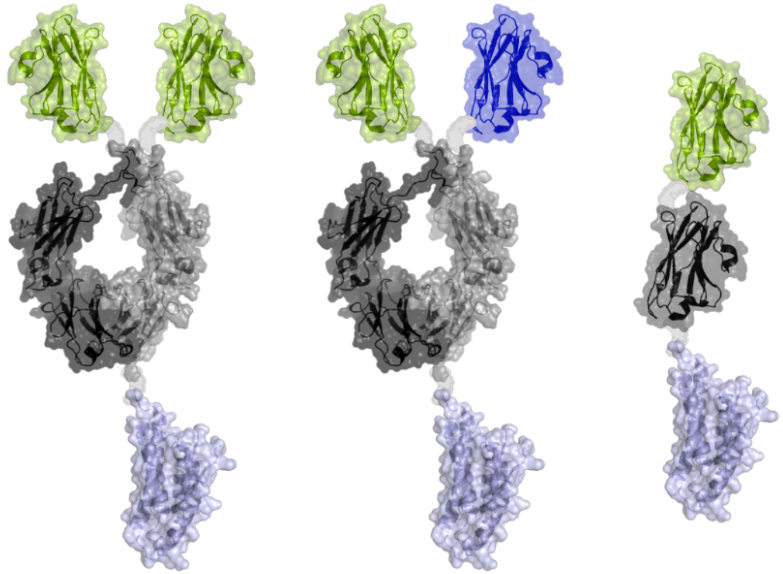
Our Solution:

Proprietary Cisleukin™ platform able to restrict IL-2 effects to a specific target cell/antigen utilizing high affinity sdAb and an engineered cis-binding IL-2 variant (IL2-X)

High affinity sdAb targeting

Low affinity IL2-X

Targeting of IL2-X via high-affinity sdAbs allows pinpointed signaling on defined target cell populations



Low affinity IL2-X binding of CD25 and CD122 when not bound to a target cell/antigen that is recovered upon binding of the sdAb

Cisleukin Platform

Dutcher J , et al. Med Oncol. 2001;18:209–2109

INHIBRx

INBRX-121

NK Cell Targeted Cisleukin™ Molecule

INHIBRX

An NK Cell Targeted Cisleukin™ Molecule

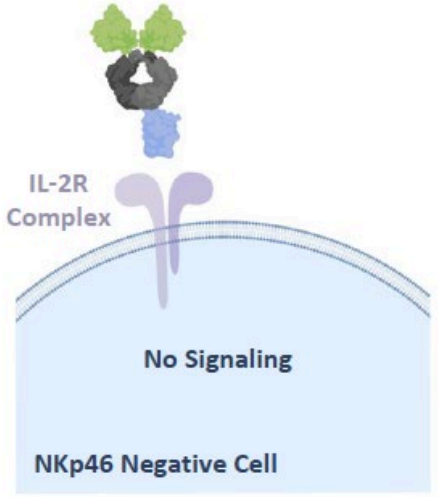
Two NKp46 sdAbs designed to allow natural ligand binding

Engineered Fc minimize effector function

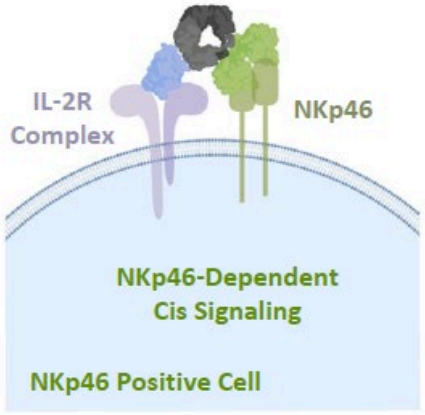
Cisleukin™ Platform IL2-X with low CD25/122 affinity until bound to target

Description/MOA

- Natural Killer (NK) cells have potent cytolytic activity and are not limited by MHC-I presentation of tumor-associated antigens like T cells
- NKp46 is an NK cell-specific marker that maintains expression on tumor-infiltrating NK cells
- Targeting of affinity-reduced IL2-X via high-affinity sdAbs for NKp46 ensures specific modulation of NK cells without impacting unwanted cell populations like regulatory T cells



Weak binding to IL-2 receptors on NKp46 negative cells (Tregs, vascular cells)



NKp46-dependent binding to IL-2 receptors on NKp46 expressing cells (NK cells)

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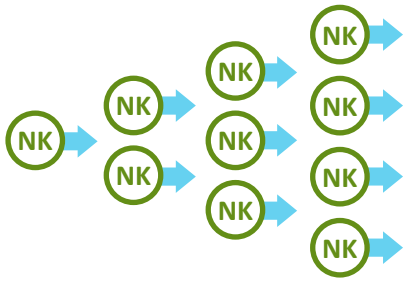
INBRX-121 is poised to bring NK cells to the forefront of immunotherapy

Improved NK Activity

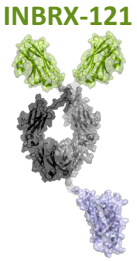


+ Expands NK cell numbers
+ Overcomes suppression
+ Enhances cytotoxic capacity

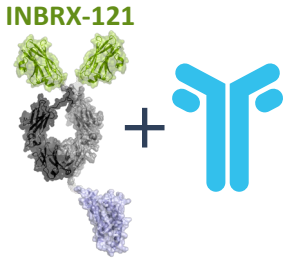
Safety with durability

+ Cytokine release syndrome not caused by NK cells
+ Extended exposure drives durability

Multiple potential paths forward



Single agent
Activated NK cells exhibit immediate cytotoxicity

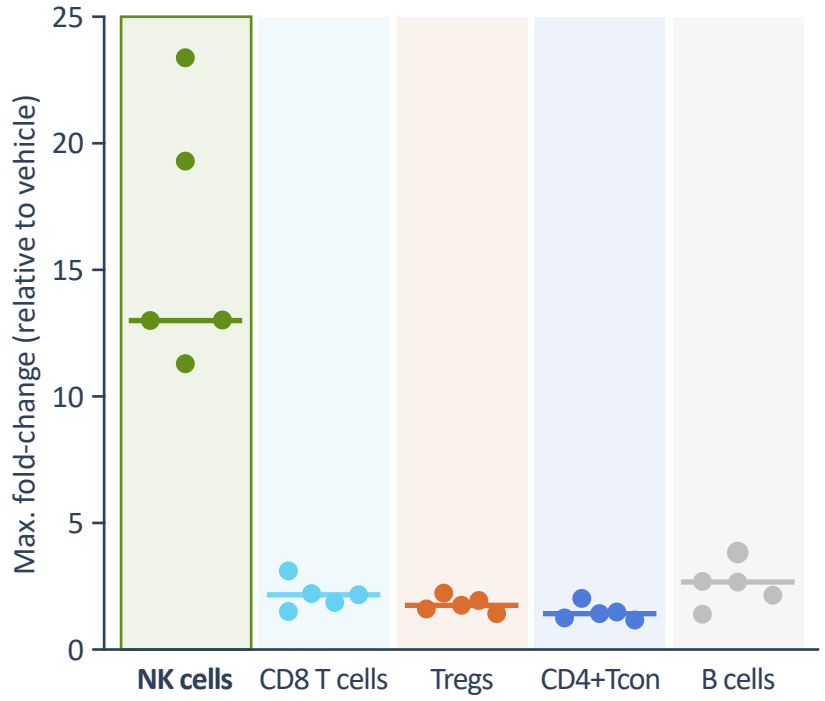


Combination therapy
Enhances the activity of therapeutic antibodies

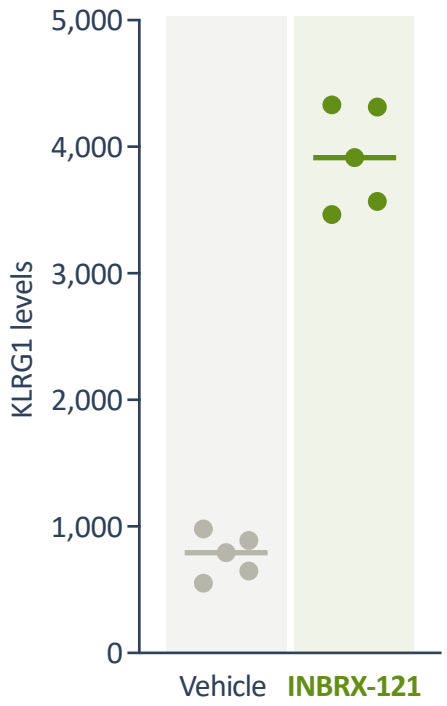
Expanded mouse NK cells and enhanced their cytotoxic potential

INBRX-121

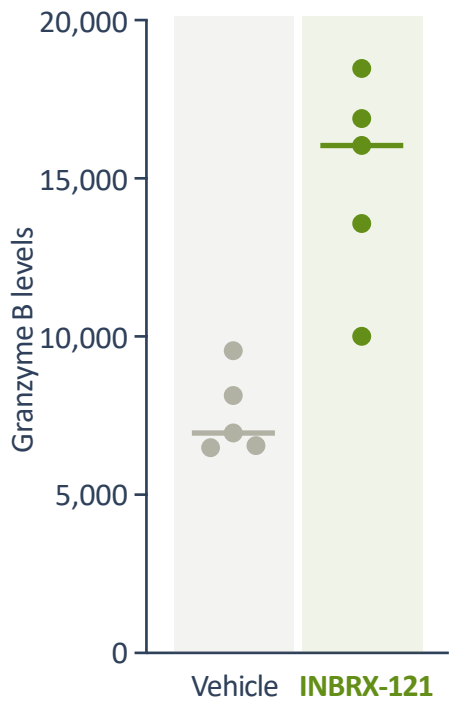
NK cell expansion



NK cell activation



NK cell cytolytic activity

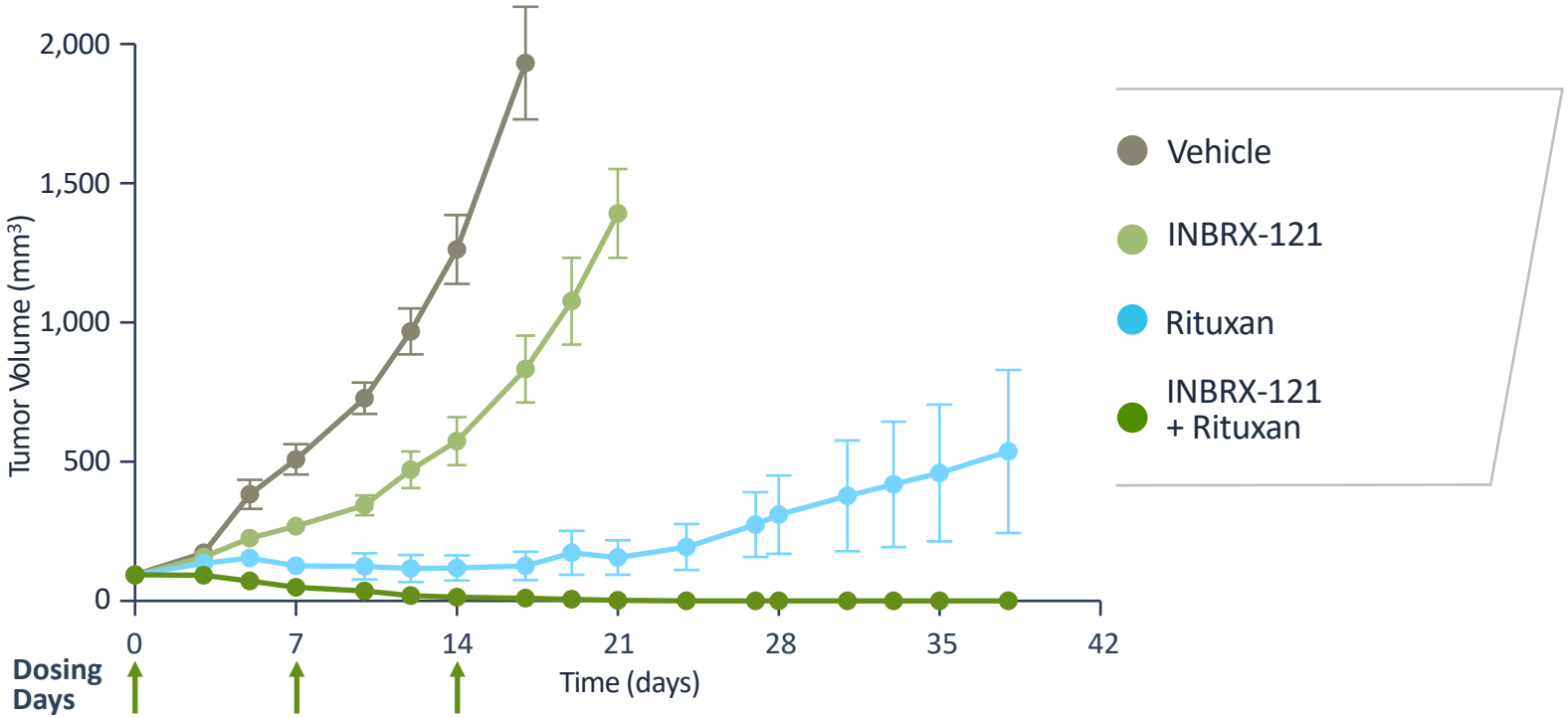


INHIBRX

Synergized with approved therapeutic antibodies

INBRX-121

Raji tumor growth



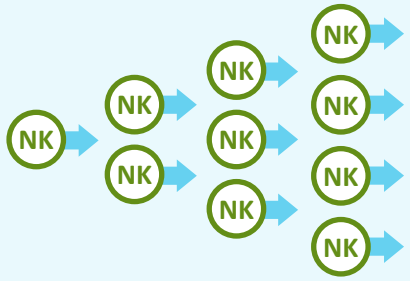
TREATMENT	COMPLETE RESPONSES
Rituxan®	0/10
INBRX-121 + Rituxan®	**9/10**

INBRX-121 synergized with Rituxan® in a subcutaneous Raji tumor model resulting in complete tumor regression

INHIBRX

INBRX-121 safely expanded NK cells in non-human primates

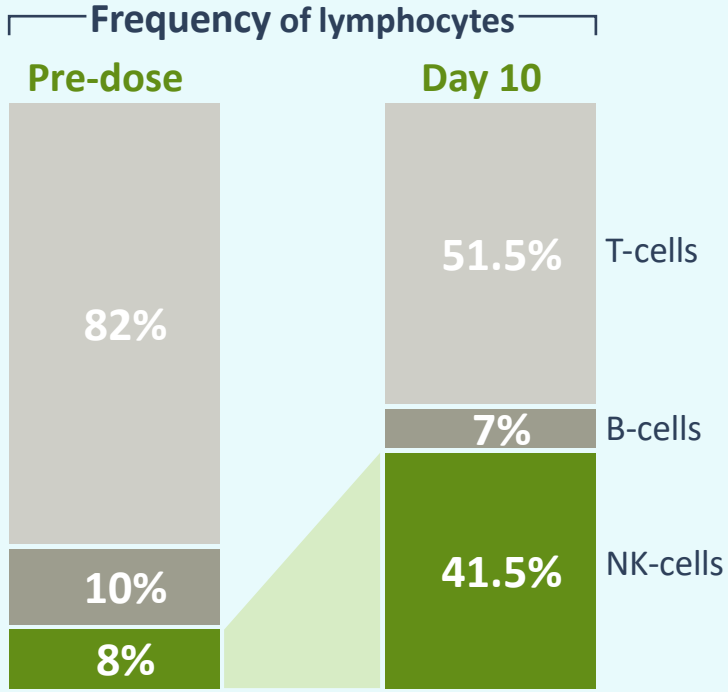
INBRX-121



Expanded NK cells in a dose-dependent manner (up to 12-fold) that persists for more than 21 days and can be dosed multiple times safely

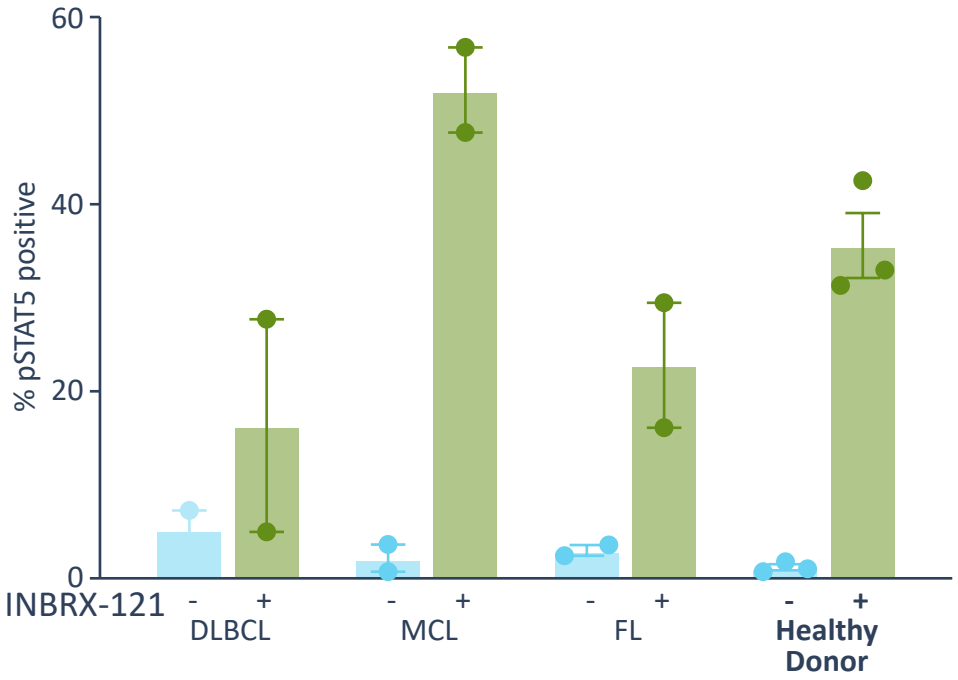
Tolerated in repeat dose range studies up to 10 mg/kg

NK cell expansion in blood after a single dose of INBRX-121 at 1 mg/kg:



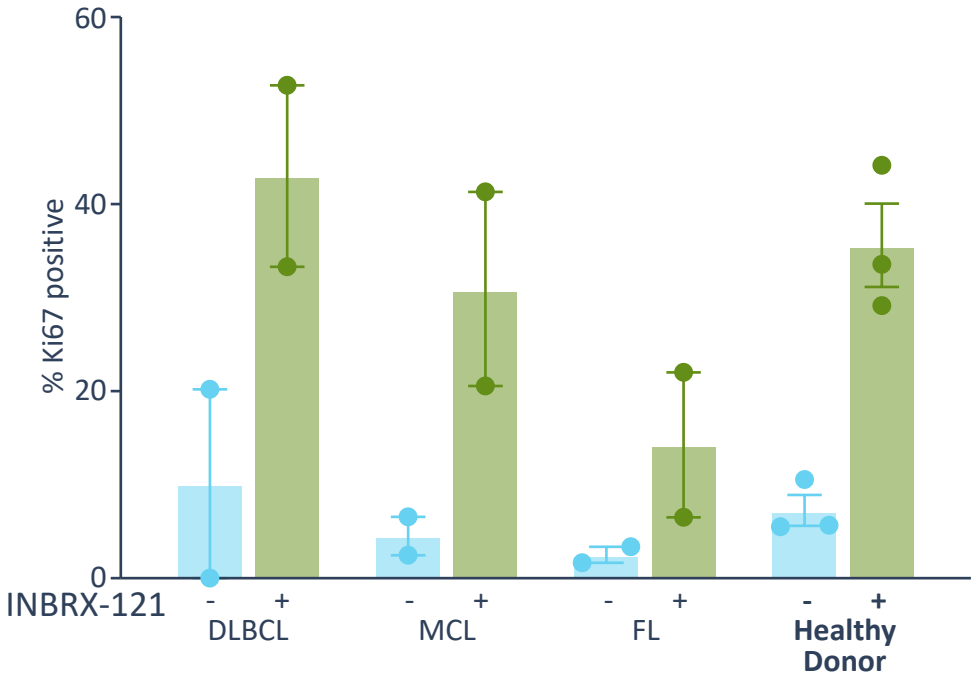
Expanded NK cells from Lymphoma patients

IL-2 signaling in patient NK cells



NK cells from Lymphoma patients expressed NKp46 at levels similar to or above that of healthy donors

Patient NK cell proliferation



Patient NK cells responded to stimulation with INBRX-121 by upregulating pSTAT5 and showed an increased proliferative potential

- +: *1 nM INBRX-121*

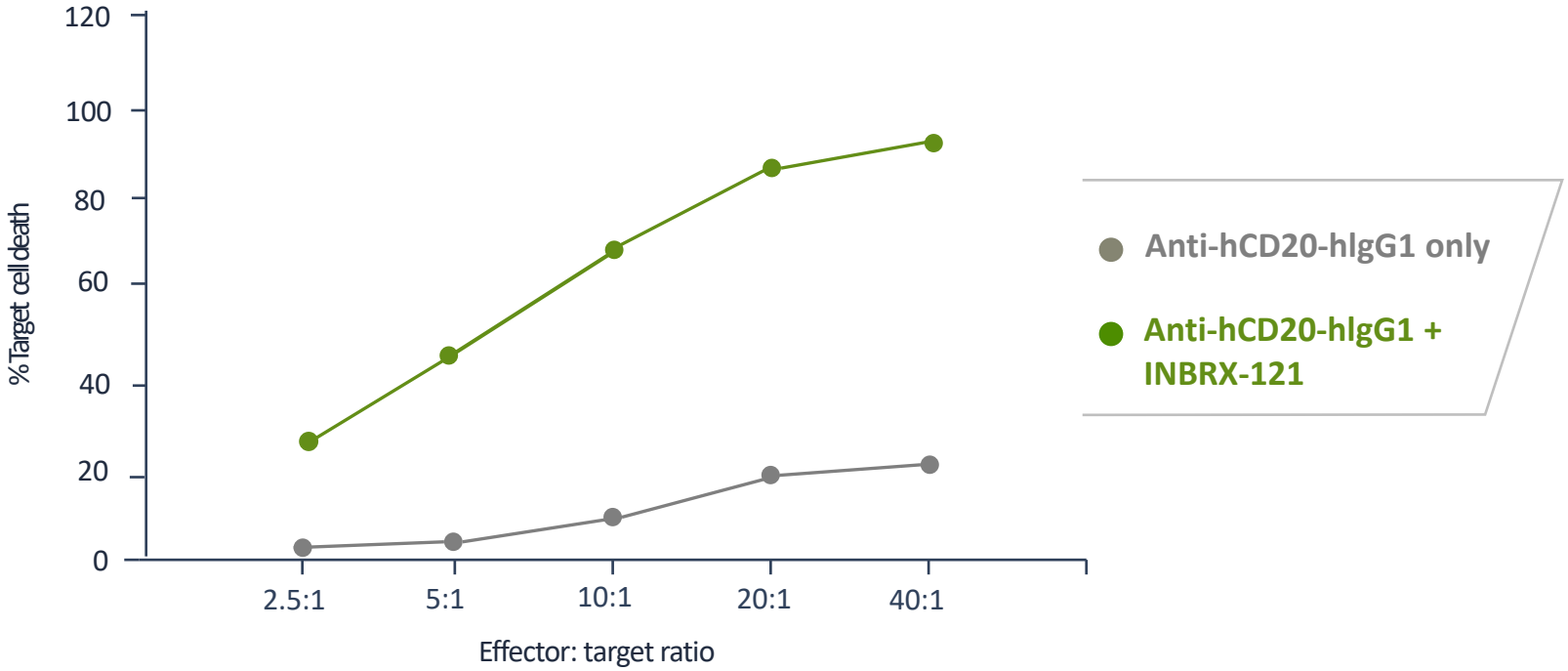
DLBCL: *Diffuse large B-cell Lymphoma*

MCL: *Mantle cell Lymphoma*

FL: *Follicular Lymphoma*

INBRX-121

Expanded the number of NK cells while enhancing their individual cytotoxic capacities



Raji cell killing after INBRX-121 pre-incubation

INBRX-121

INBRX-121 increased NK cell-mediated killing of Raji cells in the presence of a Rituximab sequence analog (Anti-hCD20-hIgG1).

INHIBRX

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