INBX Investor Presentation

Innovation Driven

Outcomes Focused

November 2023

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Our company

Our mission

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

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

Key Financial Highlights:

(as of 9/30/2023)

$337.3M	47.3M	61.2M
Cash and cash equivalents	Common stock outstanding	Fully diluted outstanding

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

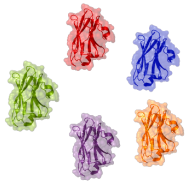
IPO in 2020

Inhibrx's innovative approach to therapeutic discovery

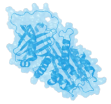
Our core belief:

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

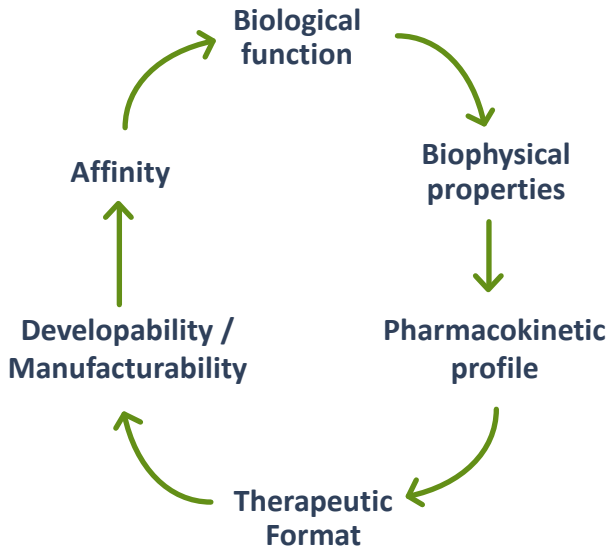


Recombinant proteins

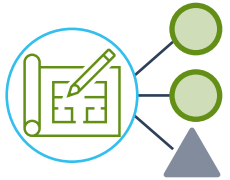
Engineered cytokines

Engineered Fc Domains

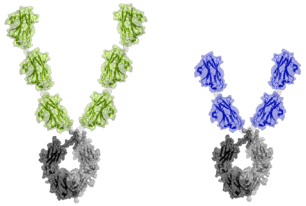
Rapid and iterative optimization process

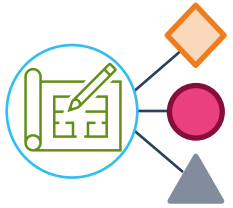


Therapeutics tailored to disease biology

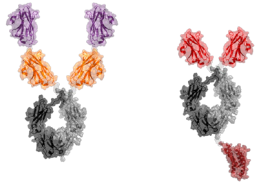


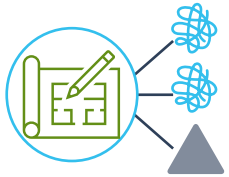
Multi-valent molecules
Molecule delivers higher clustering correlating with greater activity and more robust signal



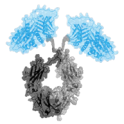


Multi-specific molecules
Molecule binds to multiple targets allowing cross linking – a novel way of delivering the drug closer to where its needed

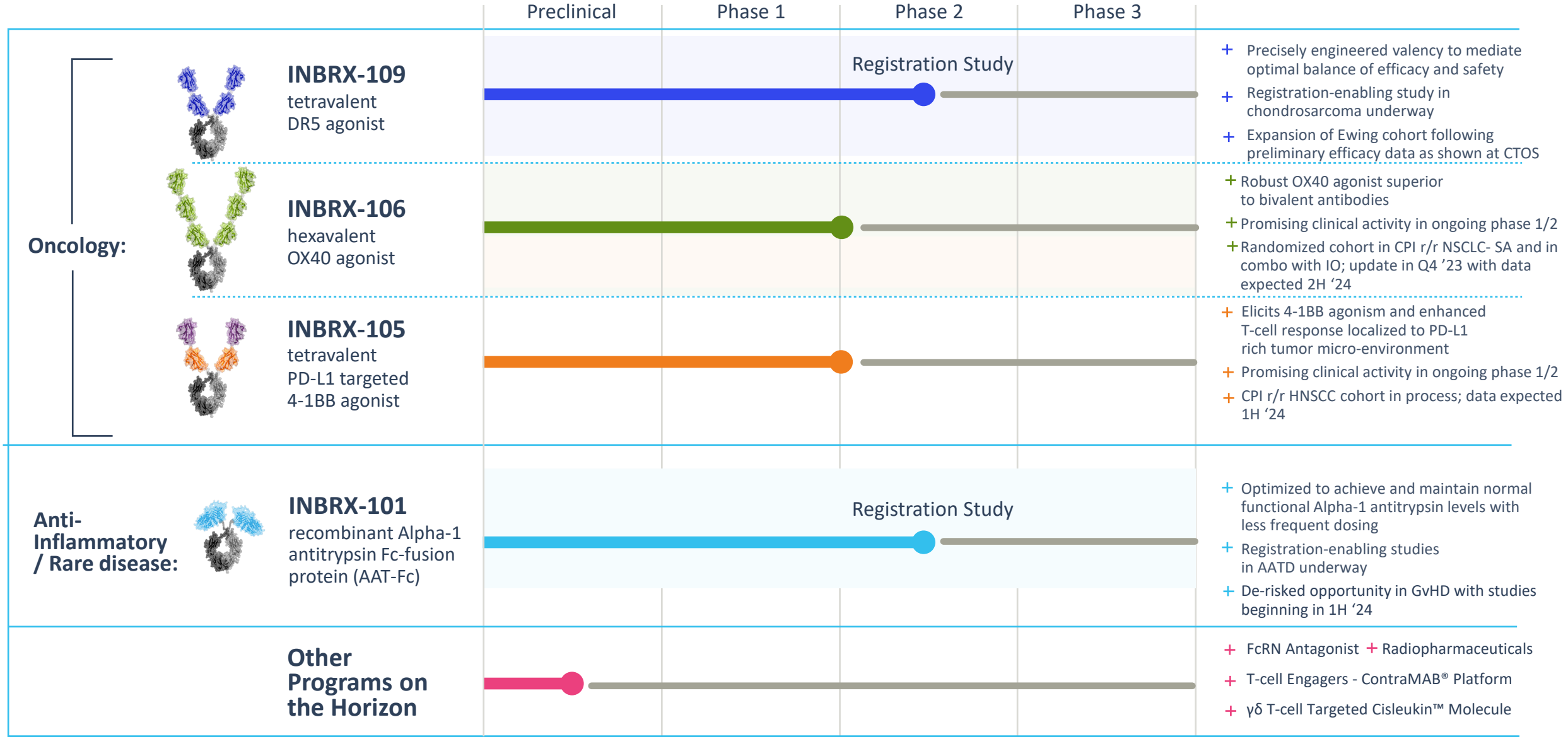




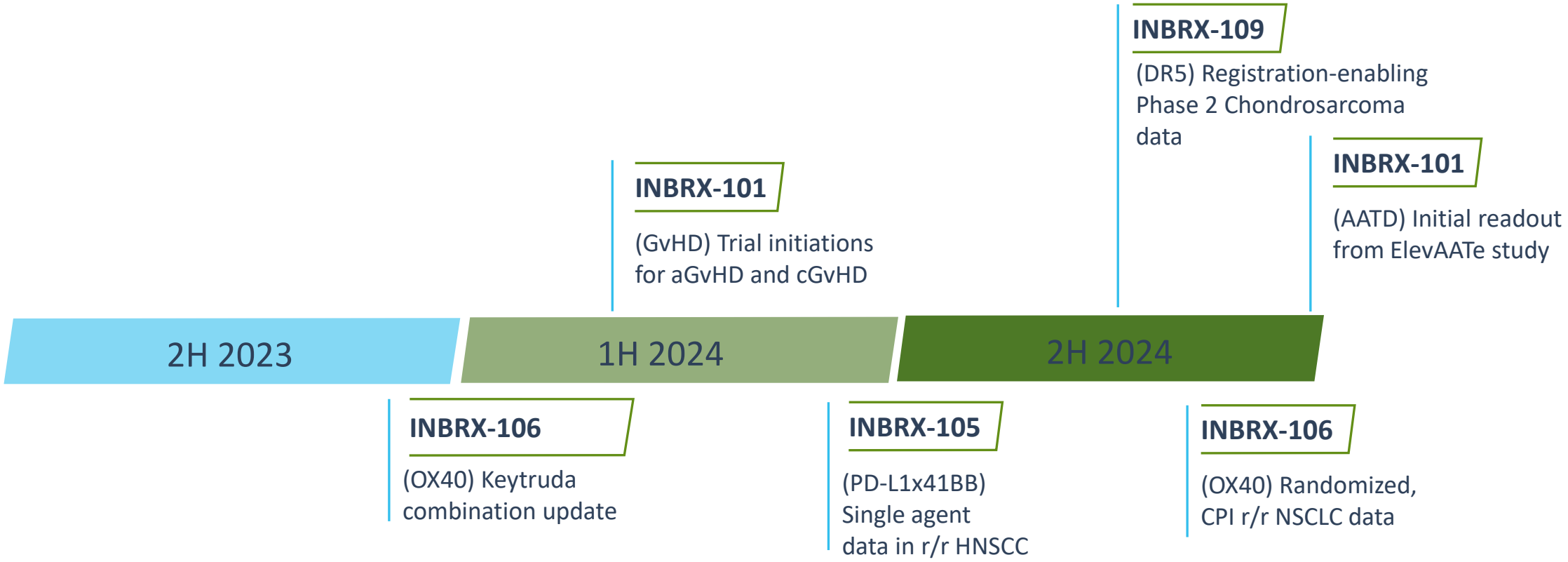
Fc-Fusion Proteins
Endow proteins with antibody-like PK properties

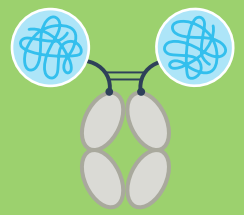


Pipeline


Preclinical
Phase 1
Phase 2
Phase 3
Oncology:
INBRX-109
tetravalent
DR5 agonist
Registration Study
+ Precisely engineered valency to mediate optimal balance of efficacy and safety
+ Registration-enabling study in chondrosarcoma underway
+ Expansion of Ewing cohort following preliminary efficacy data as shown at CTOS
INBRX-106
hexavalent
OX40 agonist
+ Robust OX40 agonist superior to bivalent antibodies
+ Promising clinical activity in ongoing phase 1/2
+ Randomized cohort in CPI r/r NSCLC- SA and in combo with IO; update in Q4 '23 with data expected 2H '24
INBRX-105
tetravalent
PD-L1 targeted
4-1BB agonist
+ Elicits 4-1BB agonism and enhanced T-cell response localized to PD-L1 rich tumor micro-environment
+ Promising clinical activity in ongoing phase 1/2
+ CPI r/r HNSCC cohort in process; data expected 1H '24
Anti-Inflammatory / Rare disease:
INBRX-101
recombinant Alpha-1 antitrypsin Fc-fusion protein (AAT-Fc)
Registration Study
+ Optimized to achieve and maintain normal functional Alpha-1 antitrypsin levels with less frequent dosing
+ Registration-enabling studies in AATD underway
+ De-risked opportunity in GvHD with studies beginning in 1H '24
Other Programs on the Horizon
+ FcRN Antagonist + Radiopharmaceuticals
+ T-cell Engagers - ContraMAB® Platform
+ γδ T-cell Targeted Cisleukin™ Molecule

Near term expected clinical milestones





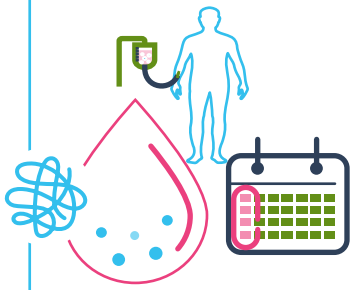
INBRX-101 AATD

recombinant Alpha-1 antitrypsin Fc-fusion protein (AAT-Fc)

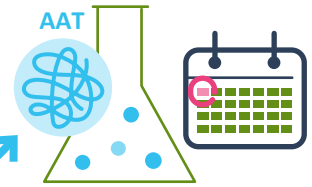
INHIBRx

Goal:

To develop a donor-independent source of AAT protein able to keep patients in the range of normal for extended periods of time



Plasma Donor derived AAT protein



Recombinant AAT protein

Current SOC

Inhibrx solution

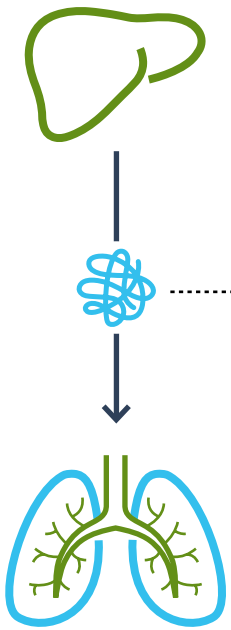
INBRX-101 for the treatment of alpha-1 antitrypsin (AAT) deficiency (AATD)

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.

ZZ
SZ

Pi*Z and PI*S alleles cause the majority of severe AATD cases



Liver

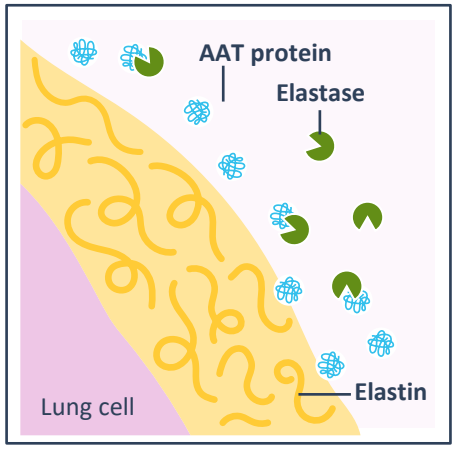
Hepatocytes synthesize and secrete AAT into the blood stream

AAT protein

Lung

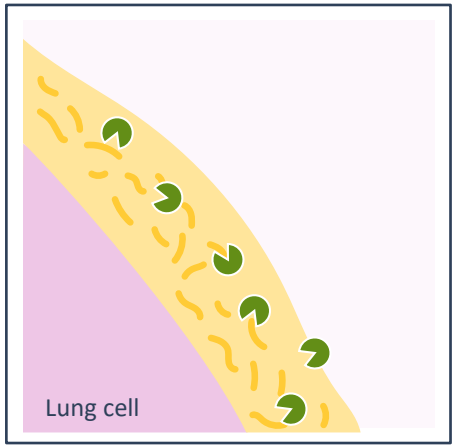
AAT protects from lung tissue damage

Healthy:



AAT protein inactivates neutrophil elastase thus preventing elastin degradation.

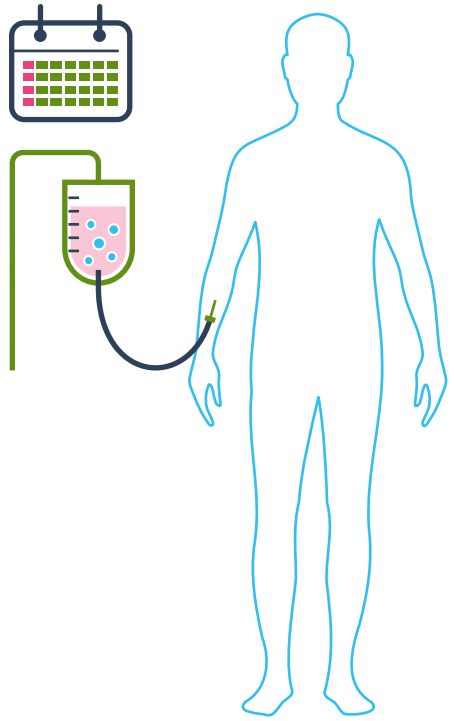
AATD:



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

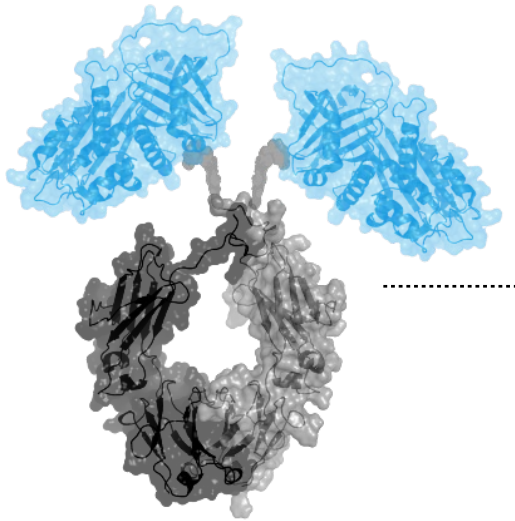
Current standard of care

Weekly augmentation of plasma donor derived AAT (pdAAT) brings patients to roughly half the normal level



INBRX-101 for the treatment of AATD

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



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 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

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

Part 1

Single ascending dose (SAD) — Complete

n=24	One dose
n=6	10 mg/kg
n=6	40 mg/kg
n=6	80 mg/kg
n=6	120 mg/kg

Part 2

Multiple ascending dose escalation (MAD) — Complete

n=18	Each patient receives 3 doses, Q3W
n=6	40 mg/kg
n=6	80 mg/kg*
n=6	120 mg/kg*

Primary endpoint:

Safety & tolerability of INBRX-101, determined by the frequency & severity of adverse events

Secondary endpoints:

Pharmacokinetics, pharmacodynamics, and immunogenicity of INBRX-101

* bronchoalveolar lavage

Topline results from phase 1, part 2

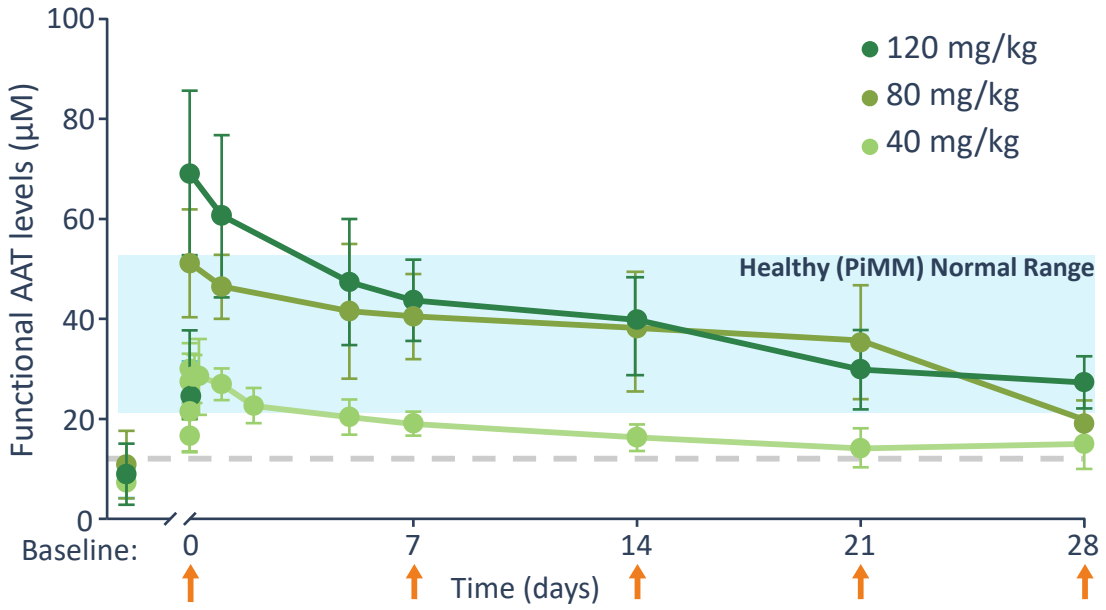
Parts 2:

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

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



- 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 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.

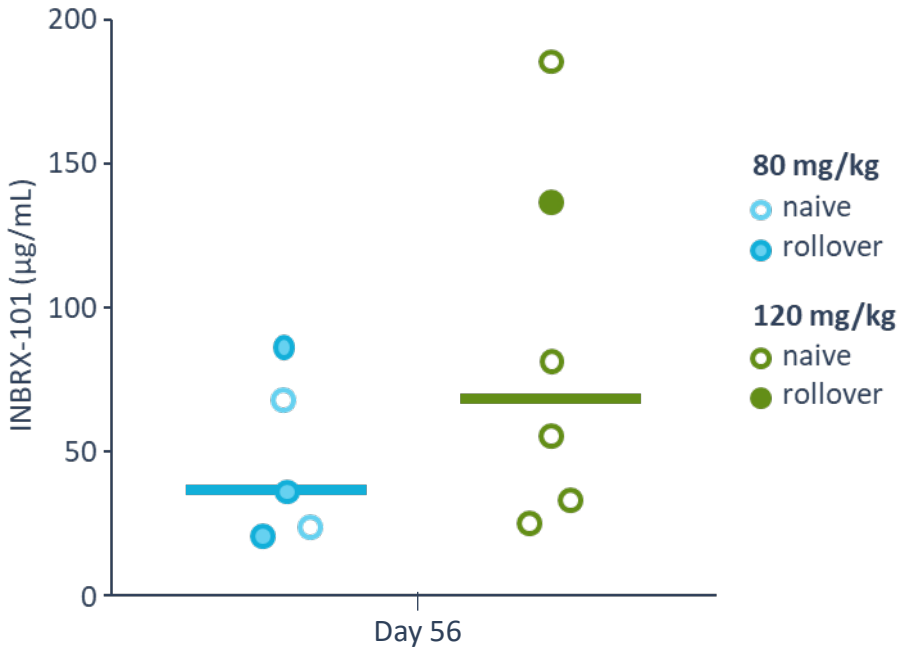
INBRX-101 concentrations were measured using a proprietary validated mass spectrometry assay specific to INBRX-101.

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.

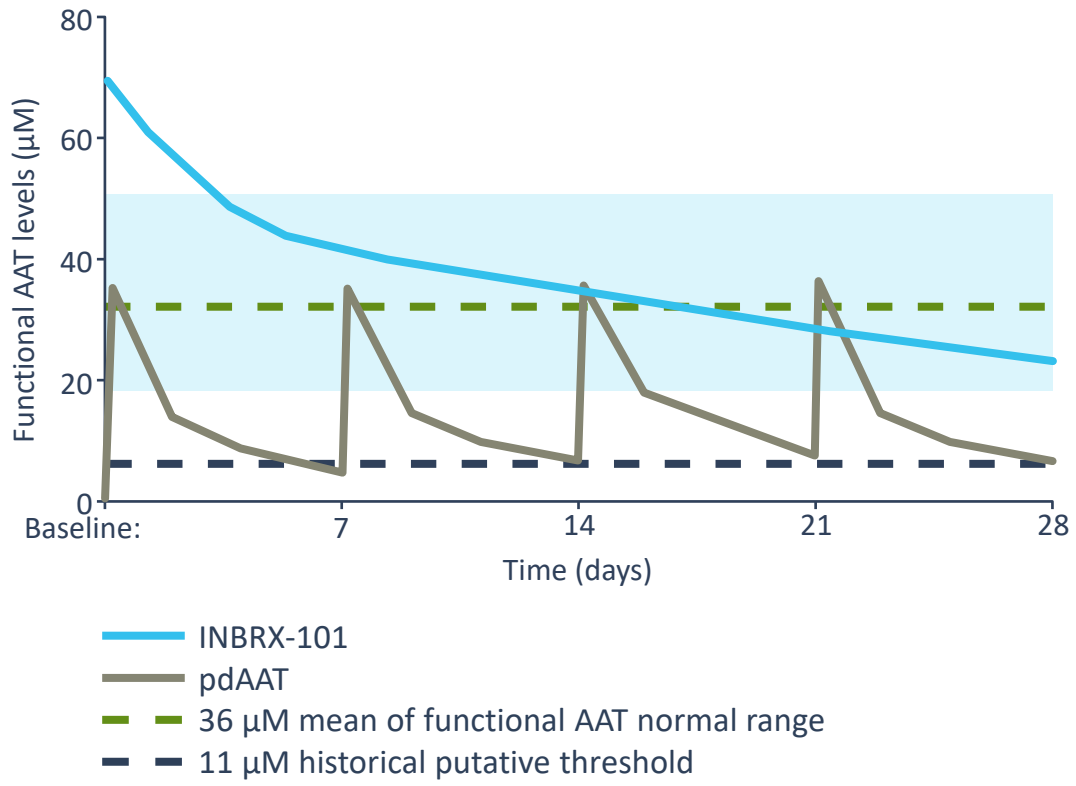
The Phase 1 study data provide emerging evidence of a dose-dependent increase in INBRX-101 lung exposure.



- 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

Projected mean steady-state functional AAT levels



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

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: ElevAATe

ELEVAATE

Initiated

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

n=130 — INBRX-101 120 mg/kg Q3W

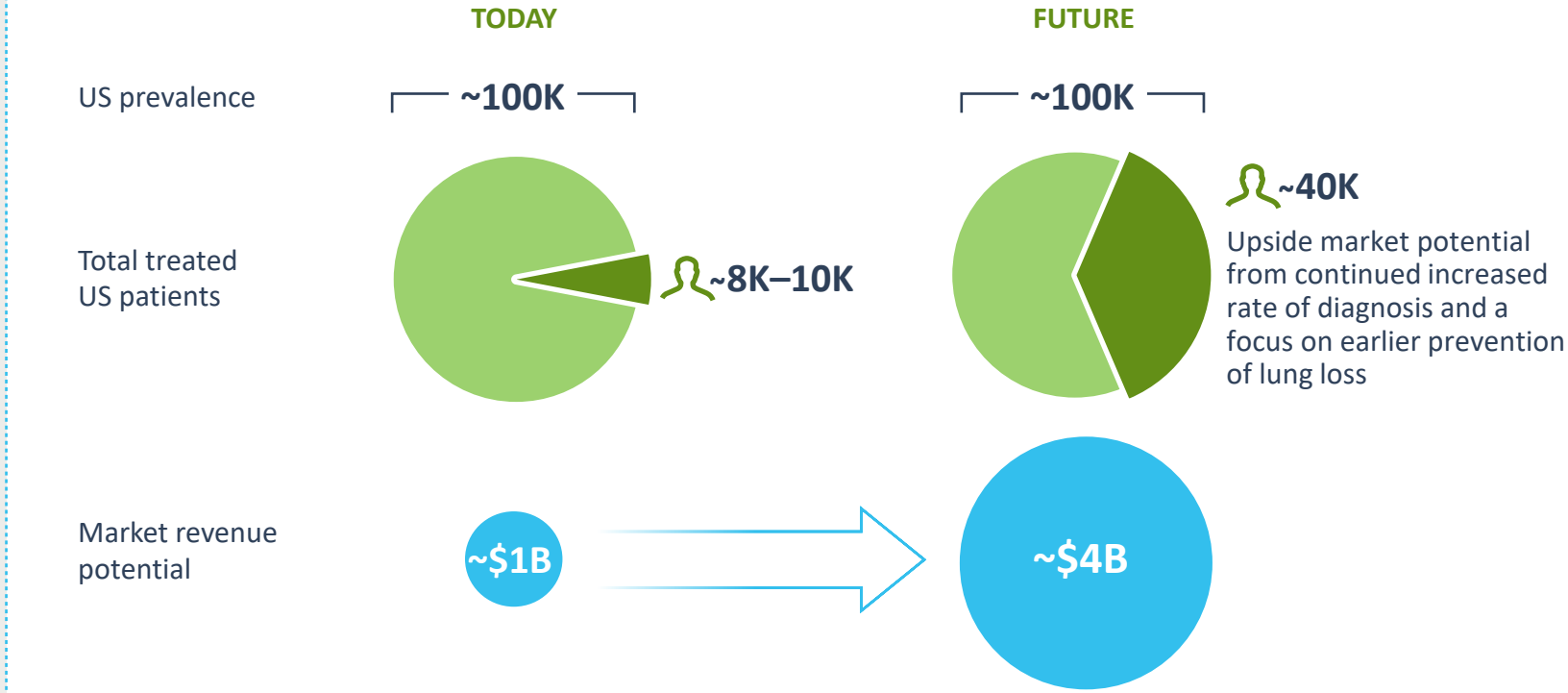
INBRX-101: AATD market opportunity

Severe* AATD cases

*defined as <11 µM serum AAT levels

ZZ SZ

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)

		Ruxolitinib, Incyte[1] (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[2] Ph1/2 (n=12)	U of Michigan/CSL[3] 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

Active Phase 2/3 studies sponsored by CSL Behring

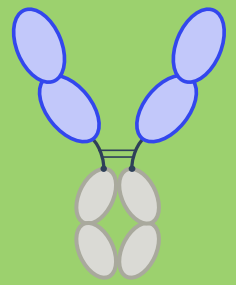
- The safety and efficacy of alpha-1 antitrypsin (AAT) for the prevention of graft-versus-host disease (GVHD) in patients receiving hematopoietic cell transplant (MODULAATE) (NCT03805789)[3]
- Treatment of GVHD in hematopoietic stem cell transplant (HSCT) recipients using AAT plus corticosteroids (CS) compared with corticosteroids alone (NCT04167514)[4]

Unlike other existing options, INBRX-101 is expected to be combinable with other therapies due to its clean safety profile.

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

Sources: 1. https://www.Jakafi.Com/pdf/prescribing-information.Pdf, https://ashpublications.Org/blood/article/135/20/1739/452638/ruxolitinib-for-the-treatment-of-steroid 2. Response of steroid-refractory acute GVHD to a1-antitrypsin, marcondes et at, 2016. http://dx.Doi.Org/10.1016/j.Bbmt.2016.05.011 3. 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 4. https://clinicaltrials.Gov/ 5. listed in alphabetical order and not comprehensive of all consensus recommendations for steroid-refractory GVHD.



INBRX-109

tetravalent
DR5 agonist

Goal:

To develop a more precise DR5 agonist able to selectively induce apoptosis in tumor cells

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

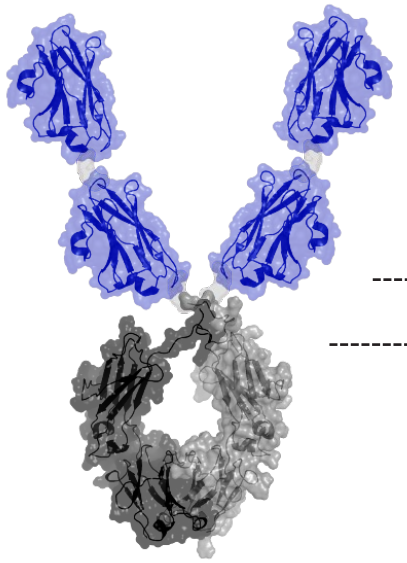
Previous generation

Empirically selected tetravalent DR5 agonist that restricts unwanted secondary clustering

Inhibrx solution

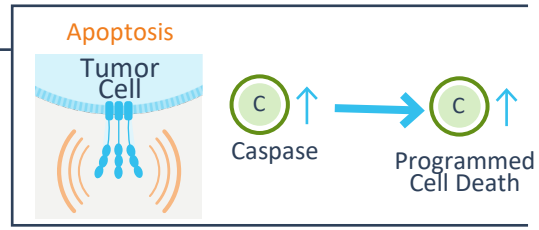
INBRX-109: a next generation DR5 agonist with an optimized balance of efficacy and safety

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]



Four DR5 sdAbs

Empirically selected and engineered to avoid ADAs



IgG Fc

Fc engineered to minimize effector function	Prevents cross-linking and higher order clustering

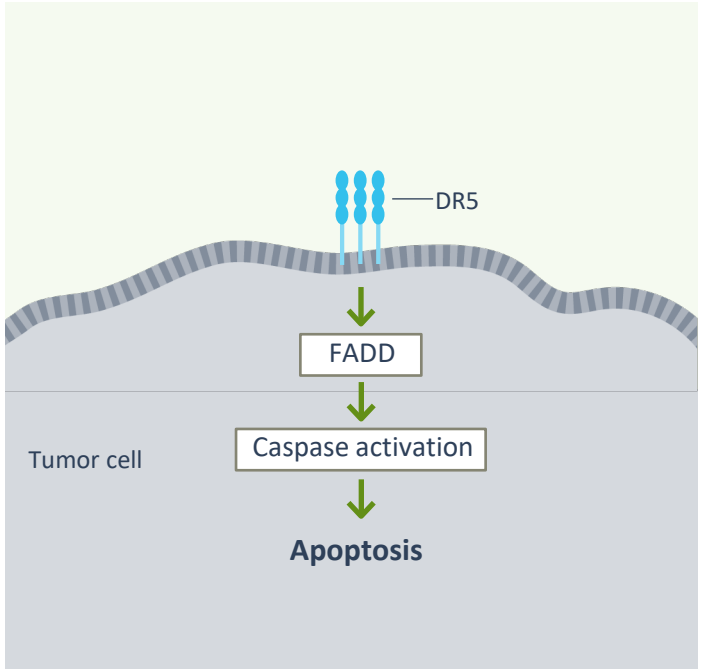
INBRX-109 characteristics:

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

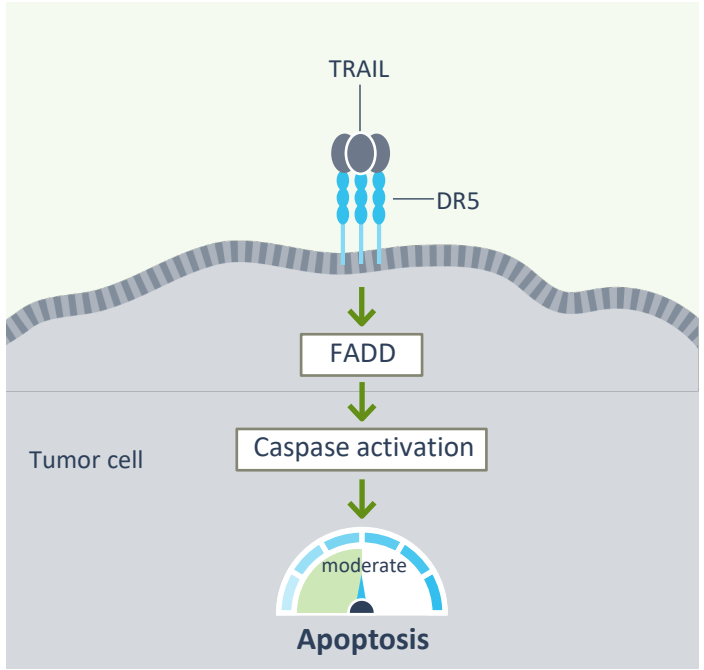
1. Mol Cancer Ther. 2012;11(11):2541-2546. 2. Cancer Cell. 2014;26(2):177-189. 3. Haematologica. 2005;90(5):612-624. 4. Cell Res. 2005;15(6):430-438

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

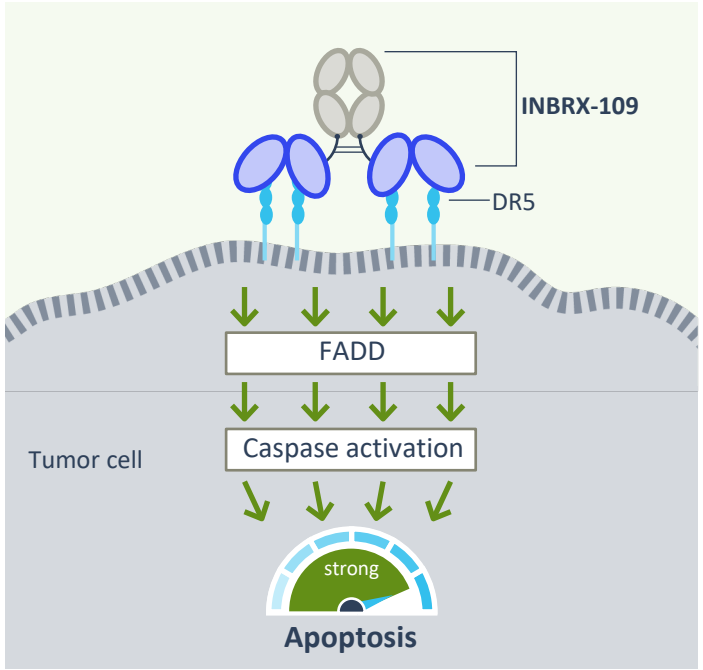
INBRX-109



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

1. Mol Cancer Ther. 2012;11(11):2541-2546. 2. Cancer Cell. 2014;26(2):177-189. 3. *Haematologica.* 2005;90(5):612-624. 4. *Cell Res.* 2005;15(6):430-438. 5. Antibodies (Basel). 2017;6(4) 6. J Biol Chem. 2012;287(25):21265-21278. 7. Cell. 2019;176(6):1477-1489.e1414. 8. Proc Natl Acad Sci U S A. 2015;112(18):5679-5684.

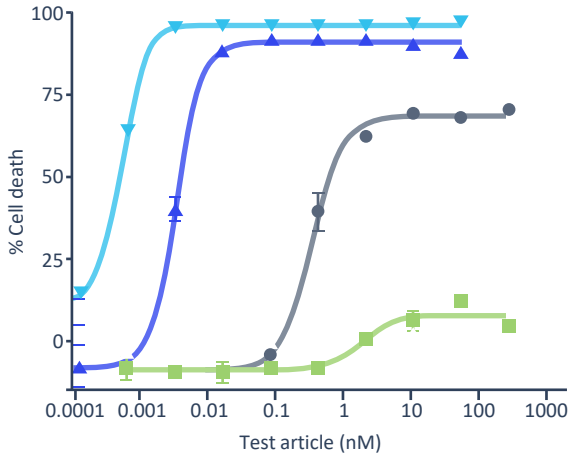
INBRX-109 is precision-engineered for optimal potency and safety

INBRX-109

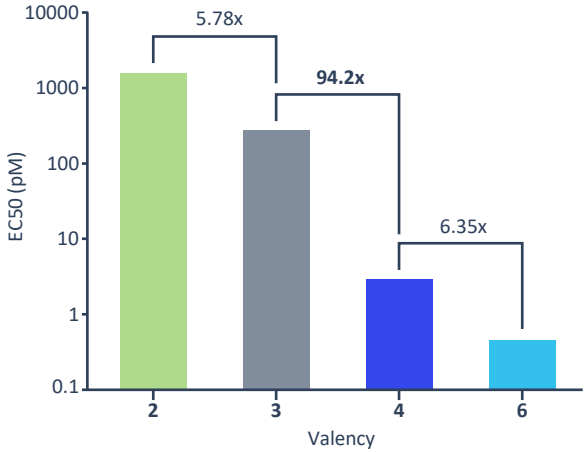
Valency drives both DR5-induced tumor cell death and hepatocyte destruction

Impact of valency on DR5-mediated cell death

Impact of valency on cell death:



Impact of valency on cell death:



anti DR5 mAb
bivalent

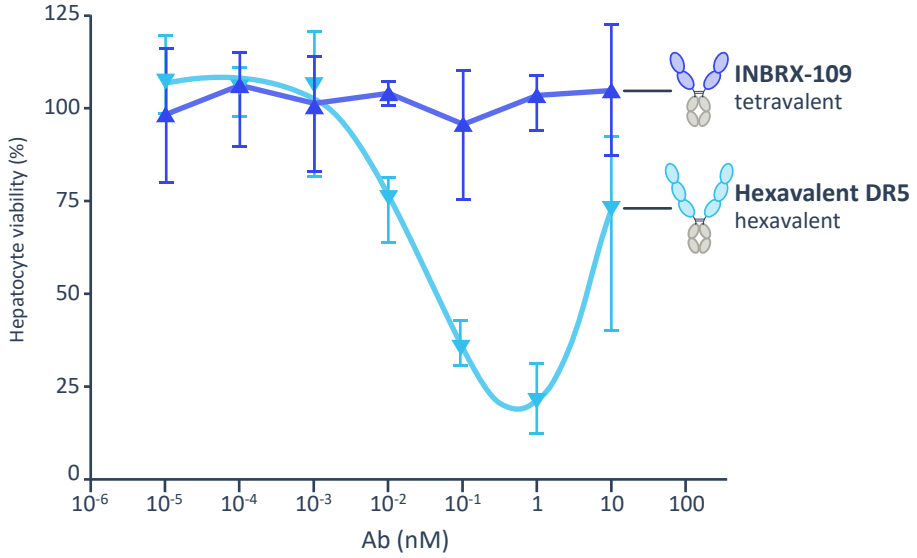
TRAIL
trivalent

INBRX-109
tetravalent

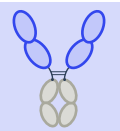
Hexavalent DR5
hexavalent

Impact of valency on hepatotoxicity

InSphero 3D inSight™ human liver microtissue model:



INBRX-109: Phase 1 trial design

INBRX-109 

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

Part 1

INBRX-109 single-agent dose escalation

Complete

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

Complete

n=116

n=20	Colorectal adenocarcinoma	n=10	Synovial sarcoma
n=10	Gastric adenocarcinoma	n=12	IDH1/2-mutant conventional chondrosarcoma
n=20	Malignant pleural mesothelioma	n=12	Nonconventional chondrosarcoma
n=20	Chondrosarcoma	n=12	Solid tumors, BMI >30

Part 3

Dose expansion with chemotherapy

Ongoing

n=100

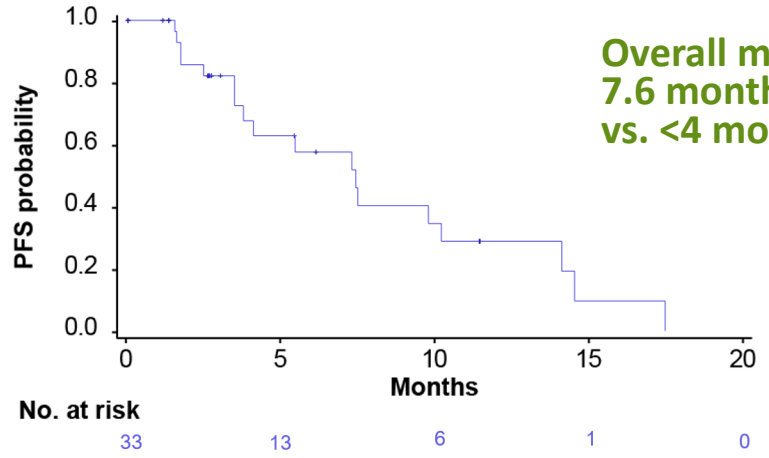
n=10	Mesothelioma with carboplatin or cisplatin	n=20-50	Ewing sarcoma 2-4L with irinotecan and temozolomide
n=10	Mesothelioma with carboplatin and pemetrexed or cisplatin and pemetrexed	n=20	Colorectal adenocarcinoma with FOLFIRI
n=20	Pancreatic adenocarcinoma 2L with fluorouracil and irinotecan (mFOLFIRI)	n=20	SDH-def solid tumors or GIST with temozolomide

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

INBRX-109

Impact of valency on DR5-mediated cell death

PFS by Kaplan-Meier analysis



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

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

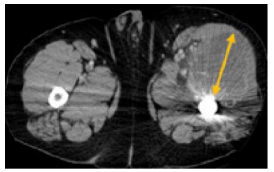
Although no approved agents, current guideline recommended treatment options[4]
have demonstrated modest activity as measured by RECIST criteria

+ 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]

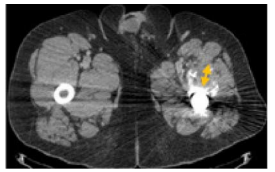
Selected case reports

Partial response:

March 4, 2020 (baseline)



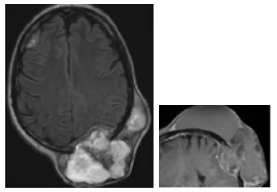
September 3, 2020 (-61%)



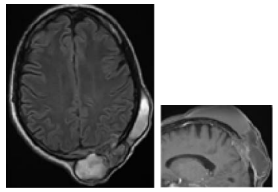
+ 29-year-old white male, histologic Grade 3
+ 61% decrease in target lesions (RECISTv1.1)
+ Patient was on study for 45 weeks

Stable disease:

April 29, 2020



September 4, 2020 (-20%)



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

IDHmt, isocitrate dehydrogenase 1/2 mutant; PFS, progression-free survival. a Includes 1 patient from dose-escalation cohort A4 (INBRX-109 10 mg/kg) and 22 patients from dose-expansion cohort B4 (INBRX-109 3 mg/kg); b Two patients were excluded due to taking prohibited medication (n=1) or having dedifferentiated chondrosarcoma (n=1).

1. van Maldegem A, et al. *Oncologist* 2019;24(1):110–6. 2. Livingston JA, et al. *Oncotarget* 2016;7(39):64421–30. 3. Duffaud F, et al. *Eur J Cancer* 2021;150:108–18. 4. NCCN. Bone cancer version 2.2022. 5. Tap WD, et al. *J Clin Oncol*. 2020;38(15):1693-1701. 6. Chow W, et al. *Cancer*. 2020;126(1):105-111.

INBRX-109 Phase 2 registration enabling study

ChonDRAgon
Chondrosarcoma Study with Death Receptor Agonist

Patients

Conventional chondrosarcoma, Grades 2 and 3, unresectable or metastatic

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

R 2:1

INBRX-109	Placebo
Ongoing	Ongoing
n=134*	n=67*
3 mg/kg every three weeks	3 mg/kg every three weeks

Primary endpoint:
Progression free survival

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

INBRX-109 for the treatment of unresectable and metastatic conventional chondrosarcoma

+ FDA fast track designation and orphan-drug designation
+ EMA orphan-drug designation

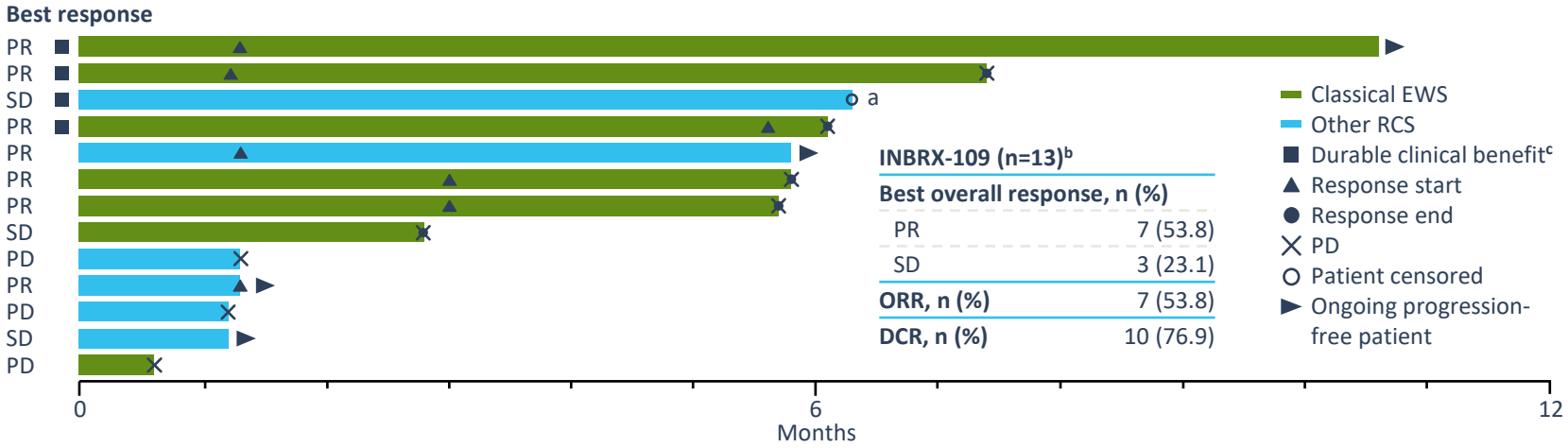
Completion projected 2H 2024

*Including interim analysis

INBRX-109 in combo with IRI/TMZ in metastatic, unresectable Ewing sarcoma

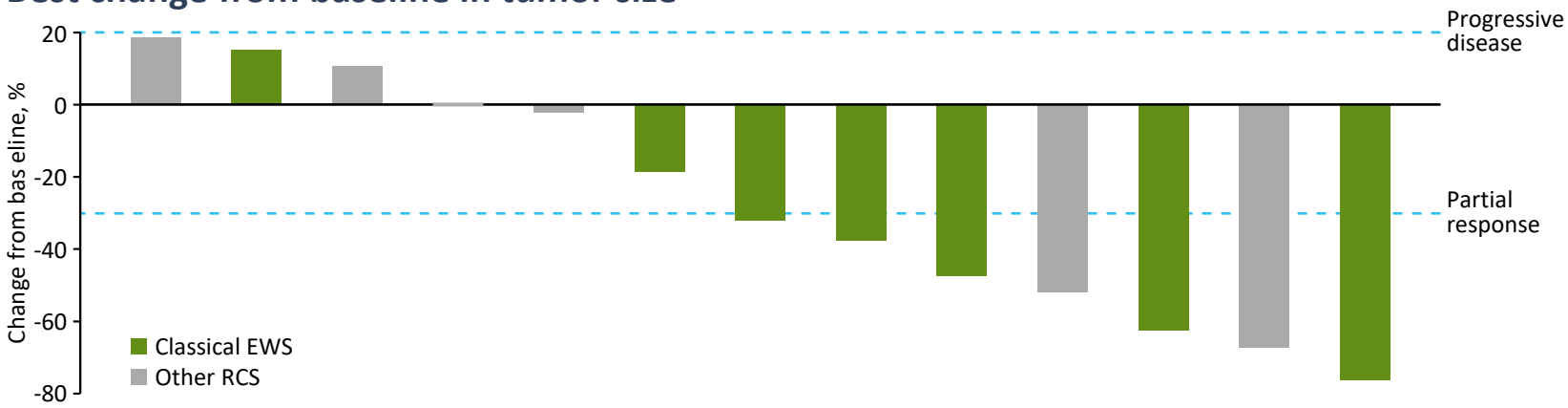
INBRX-109

Best tumor response



INBRX-109 (n=13)[b]	
Best overall response, n (%)	
PR	7 (53.8)
SD	3 (23.1)
ORR, n (%)	7 (53.8)
DCR, n (%)	10 (76.9)

Best change from baseline in tumor size



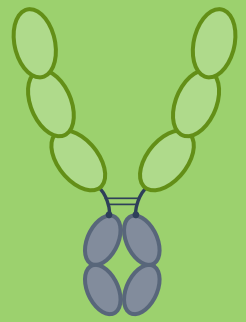
Efficacy

- 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.
[a] Patient discontinued treatment to undergo tumor resection surgery. [b] One patient had not reached the first set of restaging scans and was considered nonevaluable. [c] 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

INHIBRx

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

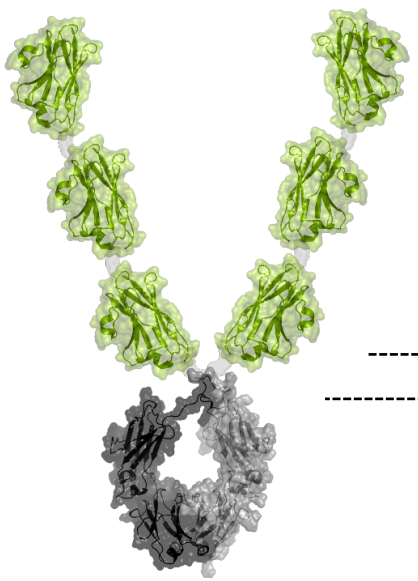
Hexavalent OX40 agonist with enhanced clustering/signaling

Inhibrx solution

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

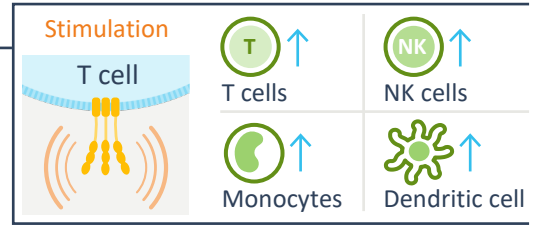
INBRX-106

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



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

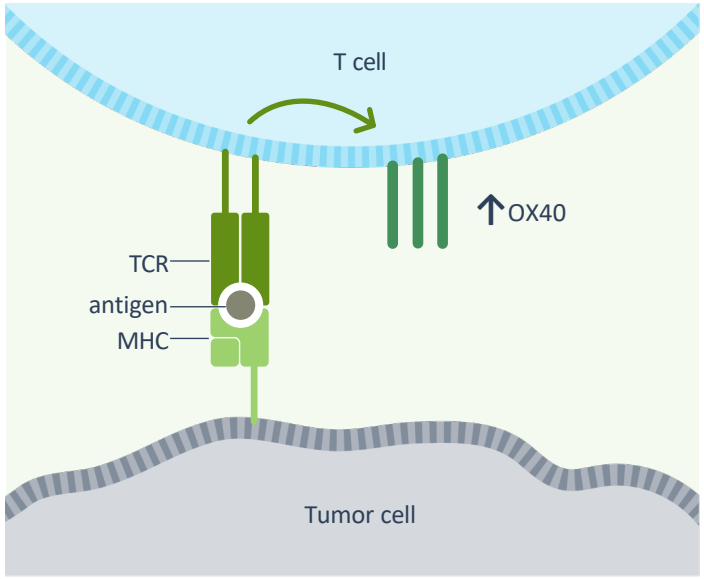
INBRX-106 characteristics:

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

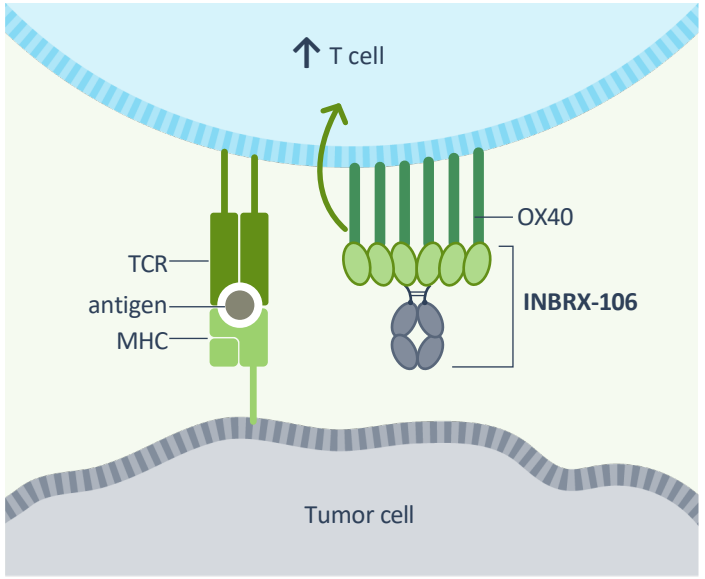
INBRX-106: mechanism of action

INBRX-106

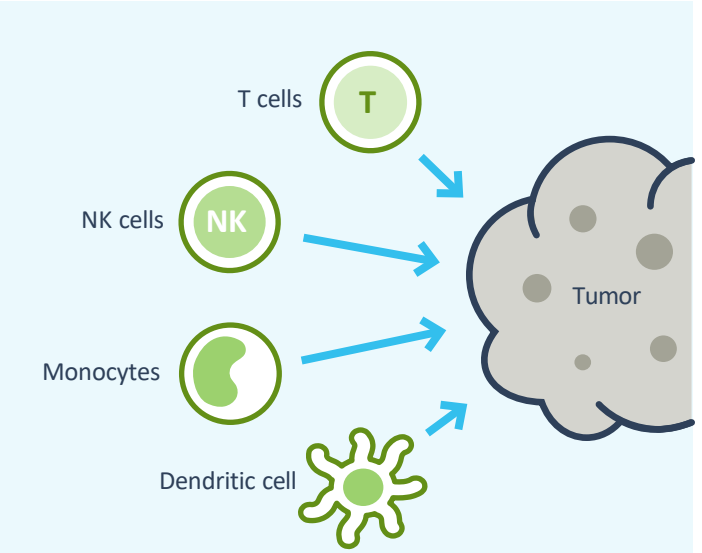
Effective OX40 agonism potentiates the body's immune response towards a tumor



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.

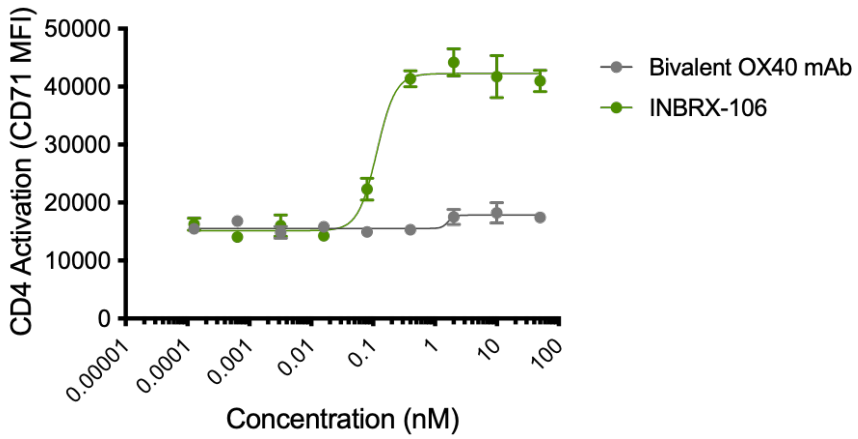


Costimulatory signaling via OX40 and the TCR-MHC receptors induce survival and proliferation of activated antigen-specific CD4 T cells, increases memory T cell generation, CD8+ effector T cells, and suppress the inhibitory capacity of regulatory T cells.

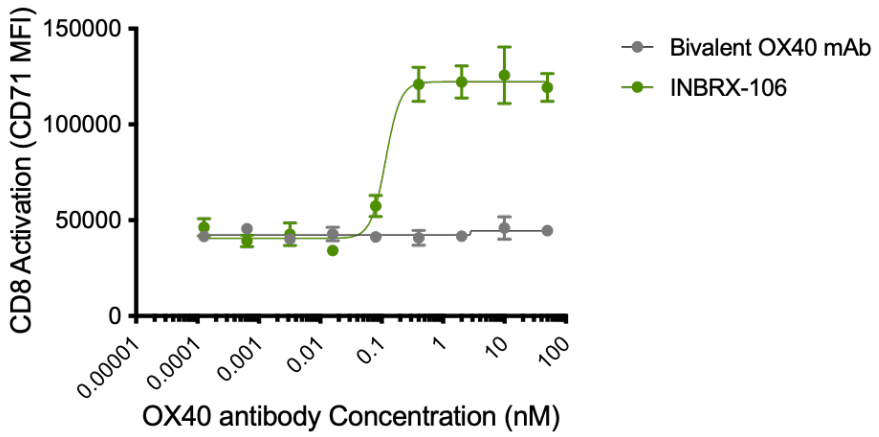
Higher OX40 valency drives superior T cell activation and reduces T_{reg} suppression

INBRX-106

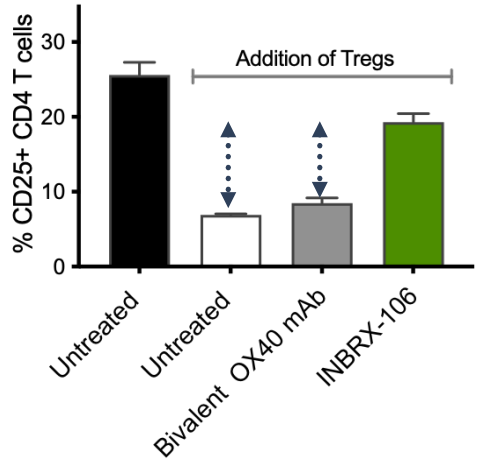
CD4 T Cell Co-Stimulation[1]



CD8 T Cell Co-Stimulation[1]



Reversal of T_{reg} Suppression[2]



~2-fold increase vs bi-valent and untreated

- 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})

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

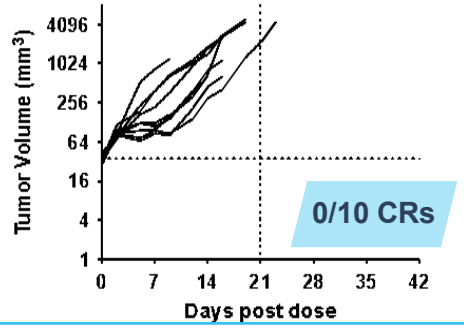
INHIBRx

Valency drives OX40 agonism in CPI-resistant tumor models

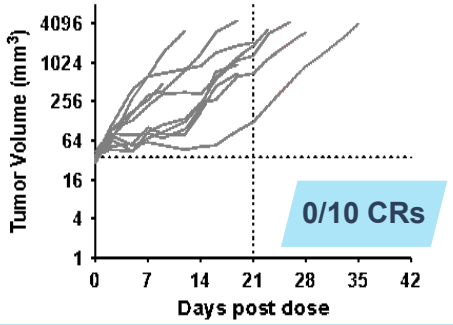
INBRX-106

Syngeneic B16F10 Mouse Tumor Model

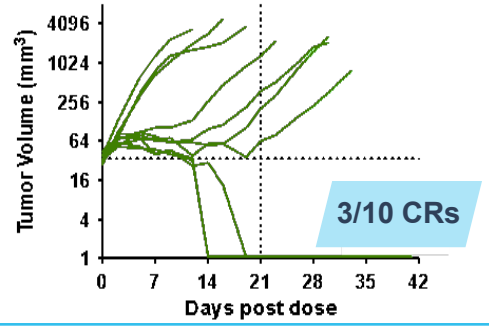
Vehicle



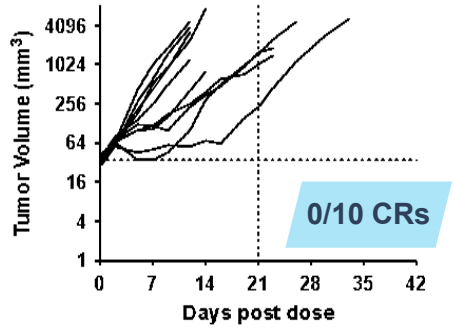
Bivalent OX40 mAb



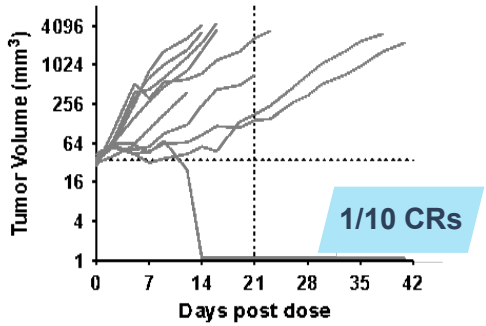
Hexavalent INBRX-106-a*



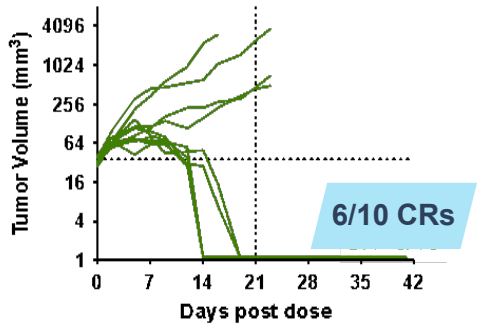
Anti-PD1 Alone



Anti-PD1 + Bivalent OX40 mAb



Anti-PD1 + Hexavalent INBRX-106-a*



- 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

*INBRX-106 mouse surrogate

INBRX-106 study design

INBRX-106

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 — Complete

n=20

Part 2

INBRX-106 single-agent dose expansion — Ongoing

n=52-64

- Basket Dose Regimen #1
- Basket Dose Regimen #2
- **NSCLC PD-L1+ (TPS ≥ 50%) or TMB high** — Ongoing
- Basket Dose Regimen #3

Part 3

Dose escalation with pembrolizumab — Complete

n=21

Part 4

Dose expansion with pembrolizumab — Ongoing

n=136

- Basket PDL1+
- NSCLC PDL1+
- **NSCLC TMB or PDL1 high** — Ongoing
  - Dose Regimen #1[a]
  - Dose Regimen #2[b]
  - Pembrolizumab Monotherapy
- Basket HNSCC, PDL1 + (CPS ≥1)[c] — Ongoing
- Basket, MMR-def/ MSI-high
- **Uveal melanoma**

All-comers CPI r/r CPI naïve

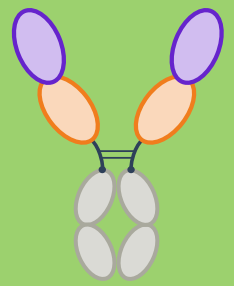
* Cohorts highlighted in green are actively recruiting

[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

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



INBRX-105

tetravalent
PD-L1 targeted
4-1BB agonist

INHIBRx

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

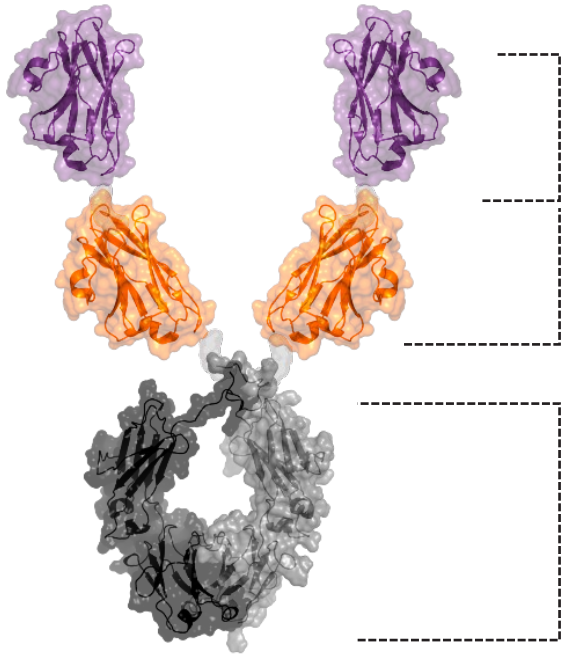
Localized 4-1BB agonist specific to PD-L1+ tissues

Inhibrx solution

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

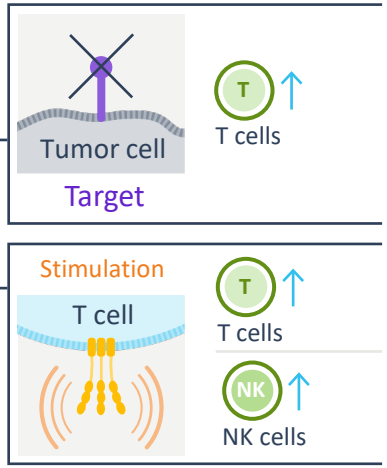
INBRX-105

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

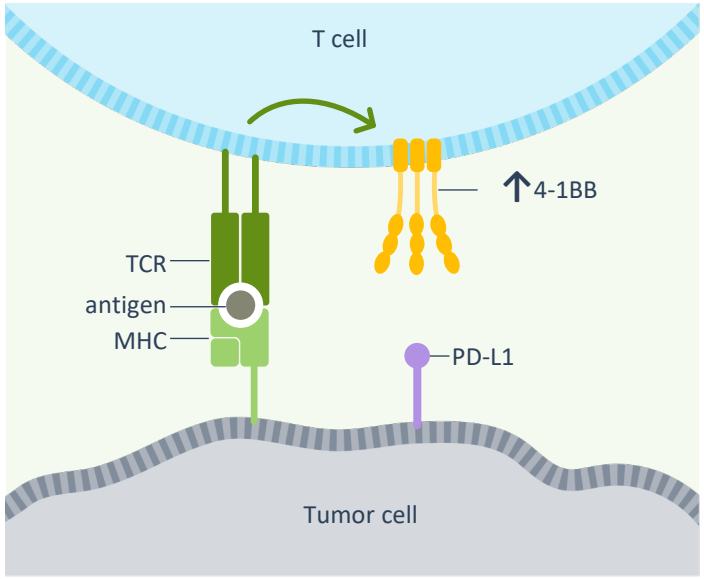
INBRX-105 characteristics:

Bispecific/ Conditional Agonist	Designed to co-engage 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

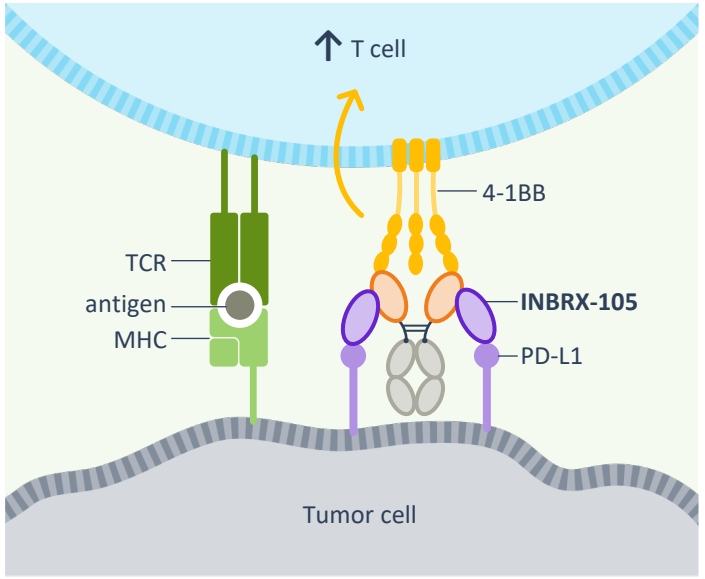
INBRX-105 mechanism of action

INBRX-105

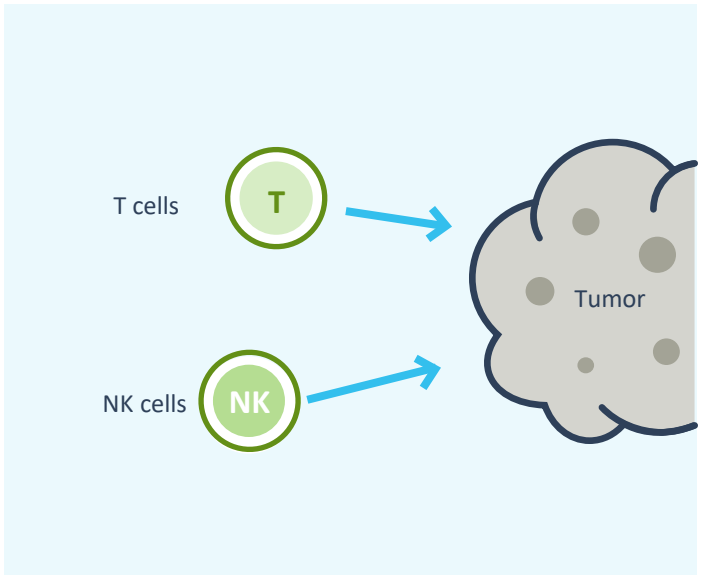
PD-L1 targeted 4-1BB agonism amplifies anti-tumor response and localizes T-cell activity



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.



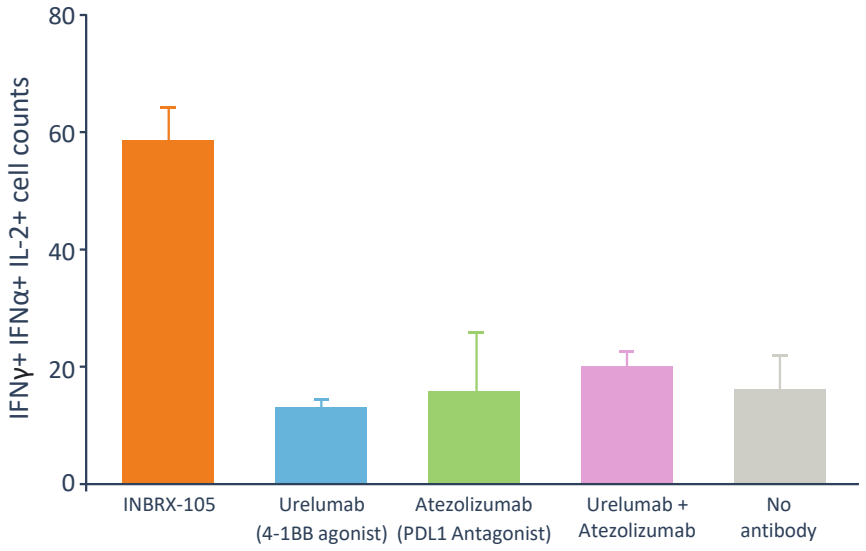
Costimulation of 4-1BB leads to downstream activation on effector cells, including increased proliferation, cytotoxicity, memory generation and possible reversal of exhaustion.

INBRX-105 shows improved T cell modulation over PD-L1 and 4-1BB agents alone or in combination

INBRX-105

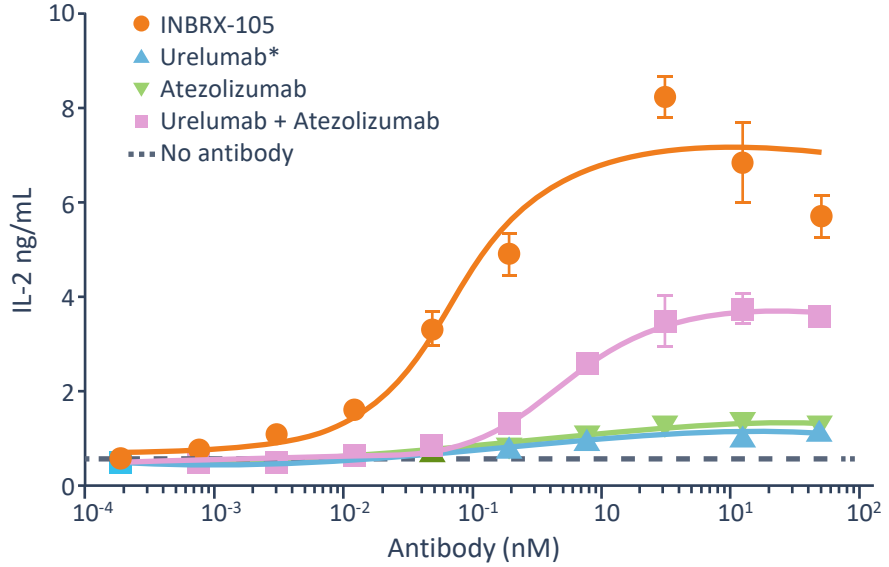
T Cell Co-stimulation[1]

Peptide-induced TCR signal



T Cell Co-stimulation[2]

MHC mismatch-induced TCR signal



- 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 (Cytomegalo-, Epstein-Barr- and Influenza-Virus) peptide mix, cytokine production measured by FluoroSpot.

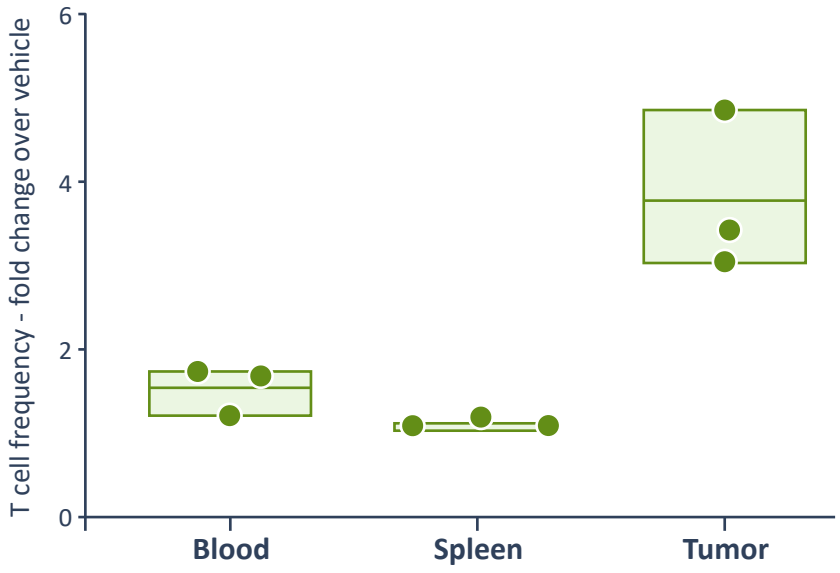
2 - Mixed-lymphocyte reaction between cells from two MHC/HLA-mismatched donors, cytokine production measured by ELISA.

Localizing 4-1BB agonism to the PD-L1-rich tumor microenvironment leads to potent anti-tumor activity in mouse models

INBRX-105

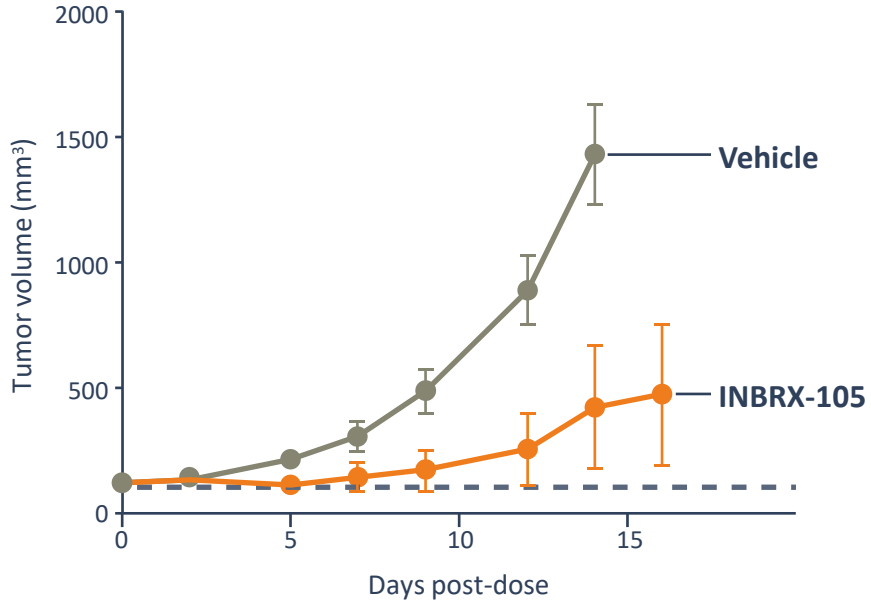
Enhanced T Cell Infiltration to TME

Day 7



MC38:

PD-L1+ Tumor Model[1]



- 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

INBRX-105

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

- Manageable early toxicity profile
- In preliminary data, single agent CRs and PRs observed in CPI r/r patients

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

Part 2

Single agent dose expansion — Ongoing

2a n=32

- NSCLC TPS >50%
- Cutaneous Melanoma or solid tumor
- HNSCC

2b n= 24-48

PD-L1 high HNSCC[a]

- NPC (CPS ≥50%)
- Non-NPC[b] (CPS ≥50%)

Ongoing

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

[a] CPS of ≥20 may be allowed if the cohort is expanded; CPI-naïve patients with PC may be eligible if CPIs are not the current standard of care for the specific indication or treatment setting

[b] Includes HNSCC of the larynx, hypo-/pharynx, and sinus

All-comers CPI r/r CPI naïve

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