INHIBRX Investor Presentation

Innovation Driven
Outcomes Focused

November 2024

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Additional information regarding the Company's risks and uncertainties are described from time to time in the "Risk Factors" section of our Securities and Exchange Commission filings, including those described in our Annual Report on Form 10-K as well as our Quarterly Reports on Form 10-Q, and supplemented from time to time by our Current Reports on Form 8-K. The Company may not actually achieve the plans, intentions or expectations disclosed in its forward-looking statements, and you should not place undue reliance on the Company's forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward-looking statements the Company makes. The forward-looking statements in this presentation represent the Company's views as of the date of this presentation. The Company anticipates that subsequent events and developments will cause its views to change. However, while the Company may elect to update these forward-looking statements at some point in the future, the Company has no current intention of doing so except to the extent required by applicable law. You should, therefore, not rely on these forward-looking statements as representing the Company's views as of any date subsequent to the date of this presentation.

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This presentation shall not constitute an offer to sell or the solicitation of an offer to buy securities.

Our mission: To discover & develop effective biologic treatments for people with life-threatening conditions

Key financial highlights:

(as of 9/30/2024)

$196.3M
Cash and cash equivalents

14.5M
Common stock outstanding

150+
employees with an experienced leadership team

19.5M*
Fully diluted outstanding

** Includes 4.0M employee and BOD option reserve and approximately 1M pre-funded warrants outstanding*

2010
INHIBRX founded

2018
first IND

2020
IPO

2024
INBRX-101 acquisition by Sanofi

INHIBRX Biosciences

>300
ozekibart (INBRX-109) Patients treated to date

>175
INBRX-106 Patients treated to date

In-house expertise:

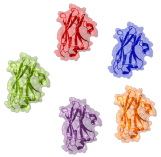
- Discovery
- Protein engineering
- Cell biology
- Translational research
- Chemistry
- Manufacturing and controls
- Clinical development and operations
- Commercial

Inhibrx's innovative approach to therapeutic discovery

Our core belief:

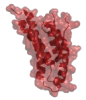
Our technology and protein engineering expertise enables us to efficiently identify optimal therapeutic formats bespoke to the target biology.

Modular protein engineering platforms



Single domain antibodies

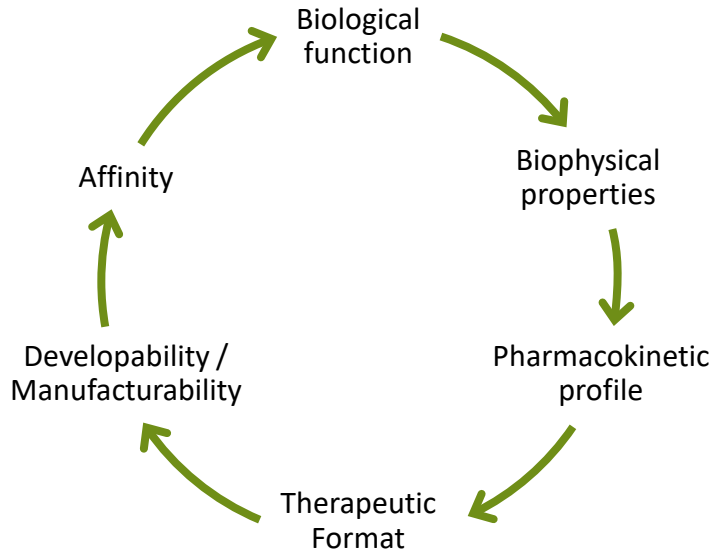
Recombinant proteins



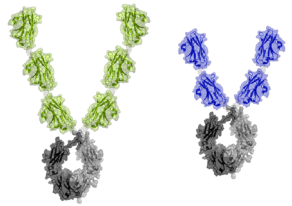
Engineered cytokines

Engineered Fc Domains

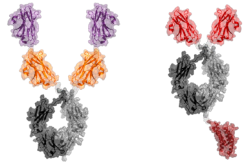
Rapid and iterative optimization process



Therapeutics tailored to disease biology



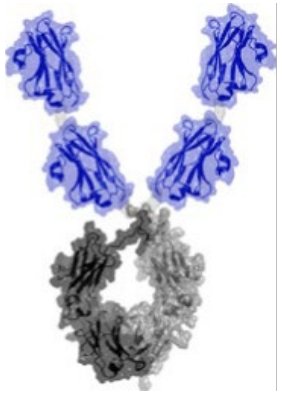
Multi-valent molecules
Molecule delivers higher clustering correlating with greater activity and more robust signal

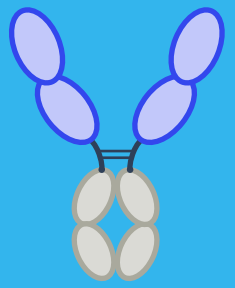


Multi-specific molecules
Molecule binds to multiple targets allowing cross linking – a novel way of delivering the drug closer to where its needed

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

Current clinical pipeline

Programs		Upcoming milestones	
	**ozekibart (INBRX-109)** tetravalent DR5 agonist	+Registration-enabling chondrosarcoma data +2-3L Ewing sarcoma and 3-4L colorectal data	**Mid 2025**
	**INBRX-106** hexavalent OX40 agonist	+Phase 2 randomized HNSSC initial data vs. Keytruda +Phase 1/2 CPI r/r NSCLC data	**2H 2025**



ozekibart (INBRX-109)

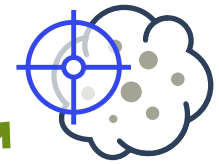
tetravalent
DR5 agonist

Goal:

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

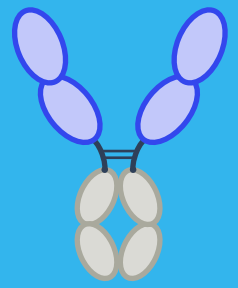
DR5 agonists with limited on target effect or unwanted off tumor toxicity

Previous generation



Empirically selected tetravalent DR5 agonist that restricts unwanted secondary clustering

Inhibrx solution



ozekibart (INBRX-109)

tetravalent
DR5 agonist

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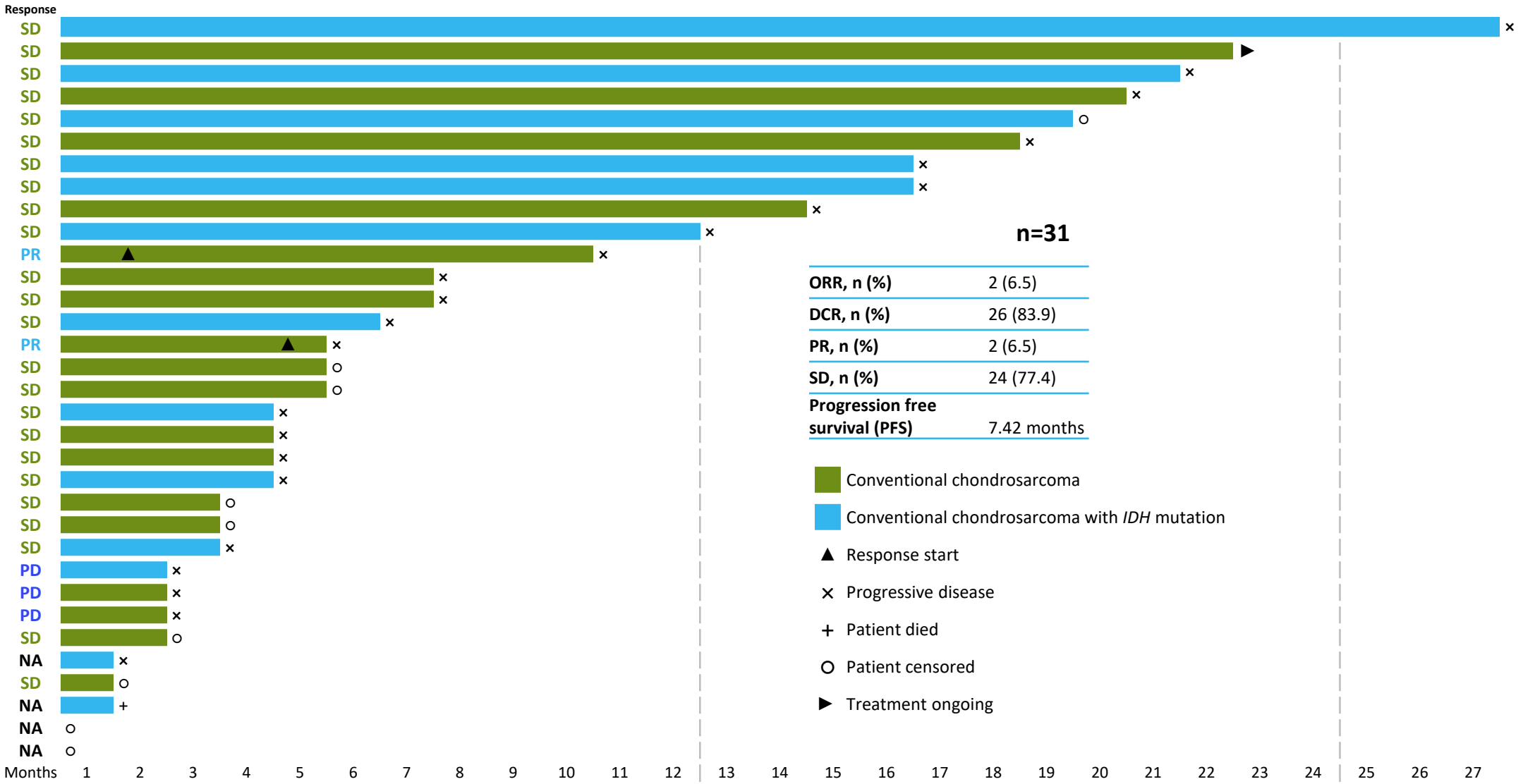
Clinical Data

Phase 1 data in unresectable or metastatic conventional chondrosarcoma

INBRX-109



Data cutoff: August 9, 2024; presented at the CTOS Annual Meeting, November 13-16, 2024; San Diego, CA

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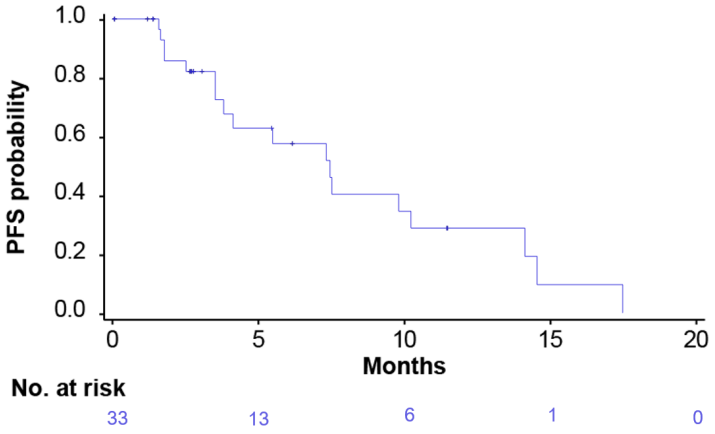
Encouraging mPFS and clinical responses observed

INBRX-109

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

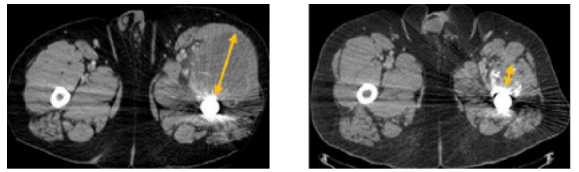
+ ivosidenib resulted in no CR or PR, an SD rate of 52% in patients with advanced chondrosarcoma (n=21)[5]
+ pazopanib in patients with unresectable or metastatic conventional chondrosarcoma (N=47) reported a DCR of 43% at week 16[6]

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).

Selected case reports

Partial response:

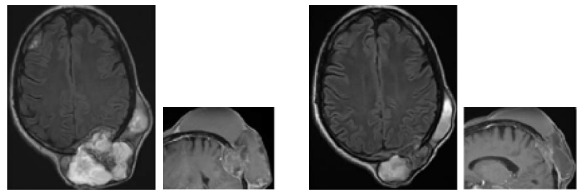
March 4, 2020 (baseline) | September 3, 2020 (-61%)



+ 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

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.

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Ongoing registration-enabling trial in unresectable or metastatic conventional chondrosarcoma

INBRX-109

Data readout expected mid-2025

+ FDA fast track designation and orphan-drug designation

+ EMA orphan-drug designation

Conventional chondrosarcoma, Grades 2 and 3, unresectable or metastatic.

R 2:1

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

ozekibart (INBRX-109)

N=134

3 mg/kg every three weeks

Ongoing

Placebo

N=67

3 mg/kg every three weeks

Ongoing

ChonDRAgon

Chondrosarcoma Study with Death Receptor Agonist

Primary endpoint:
Progression free survival.

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

DSMB reviewed interim analyses in April 2024 and made the recommendation for trial continuation

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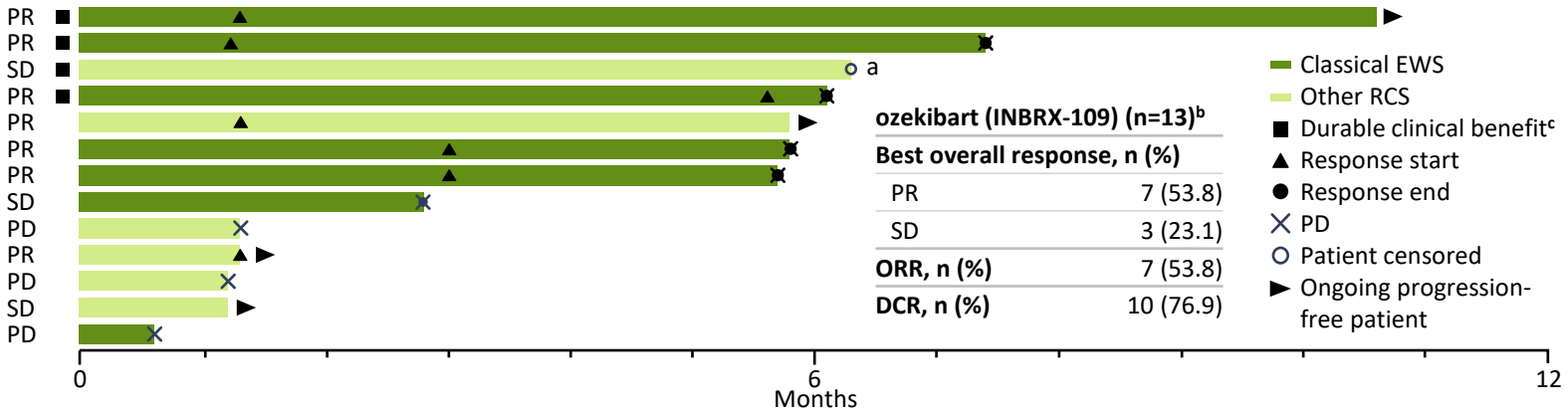
Early results in phase 1 metastatic, unresectable Ewing sarcoma

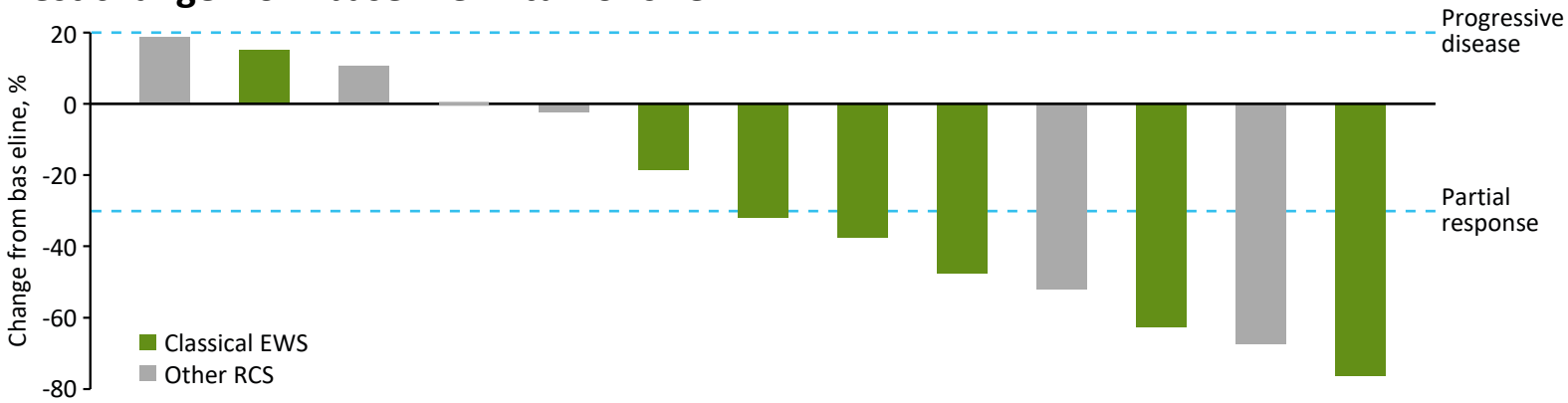
INBRX-109

Best tumor response



ozekibart (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

76.9%

Disease control rate was 76.9%, or 10 out of 13 patients as measured by RECISTv1.1.

53.8%

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.

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Ongoing phase 1/2 trial in Ewing sarcoma and colorectal adenocarcinoma

INBRX-109

Data readouts expected mid-2025

Key inclusion criteria

- LA/M, unresectable, R/R EWS
- Aged ≥12 to <85 years
- *EWSR1-FLI1*, *-ERG* or *-FEV* rearrangement
- 1-2 prior lines of chemotherapy in metastatic setting
- Prior IRI + TMZ allowed
- No chronic or acute liver disease

EWS 2-3L with IRI/TMZ

N=50

Ozekibart 3 mg/kg + IRI 50 mg/m^2/day +
TMZ 100 mg/m^2/day

Key inclusion criteria

- LA/M, unresectable, R/R colorectal adenocarcinoma
- Aged 18 to <85 years
- 2-3 prior lines of systemic therapy
- Prior IRI allowed, if not immediate prior line of therapy
- No chronic or acute liver disease

CRC 3-4L with FOLFIRI

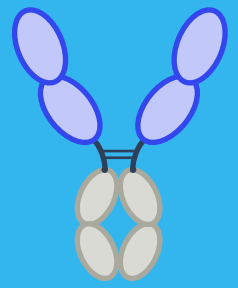
N=50

Ozekibart 3 mg/kg + FOLFIRI
(FU, 2400 mg/m^2;
leucovorin, 400 mg/m^2;
IRI, 180 mg/m^2)

Primary endpoints:
Clinical response, including ORR and DOR per RECIST 1.1., safety (AEs and DLTs)

Secondary endpoints:
PFS, pharmacokinetics and immunogenicity (ADAs)

Exploratory endpoints:
clinical response, predictive diagnostic biomarkers



Ozekibart (INBRX-109)

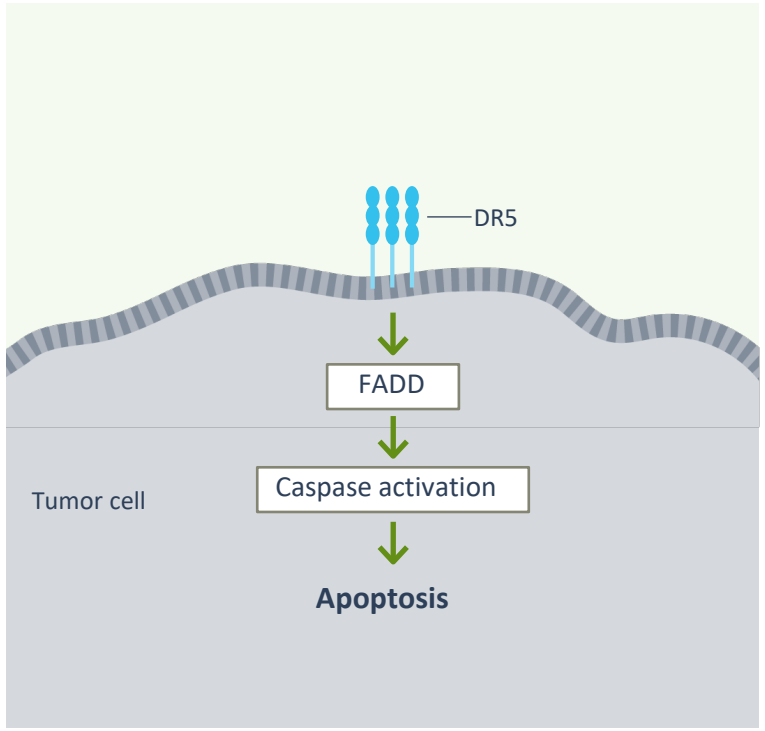
tetravalent
DR5 agonist

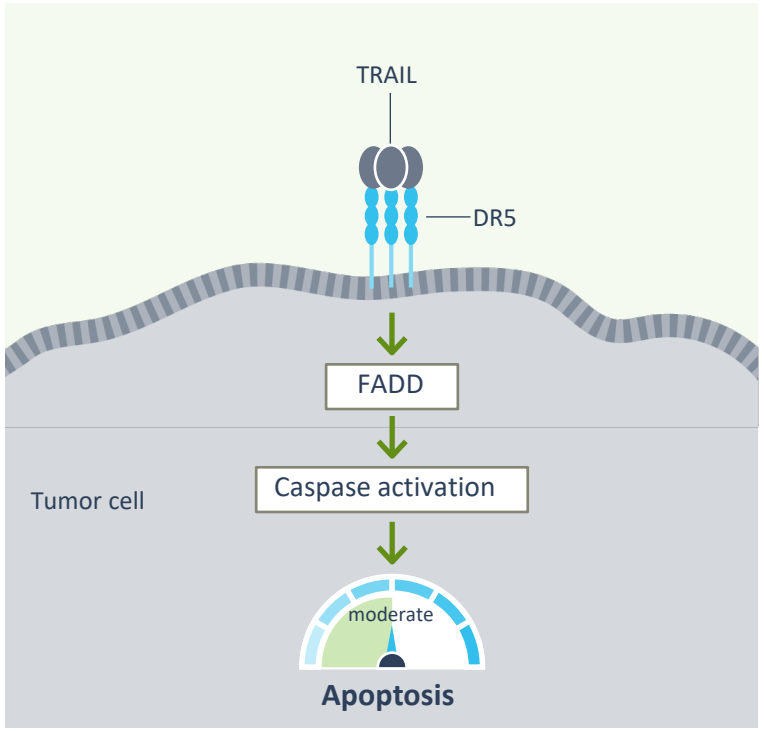
INHIBRX

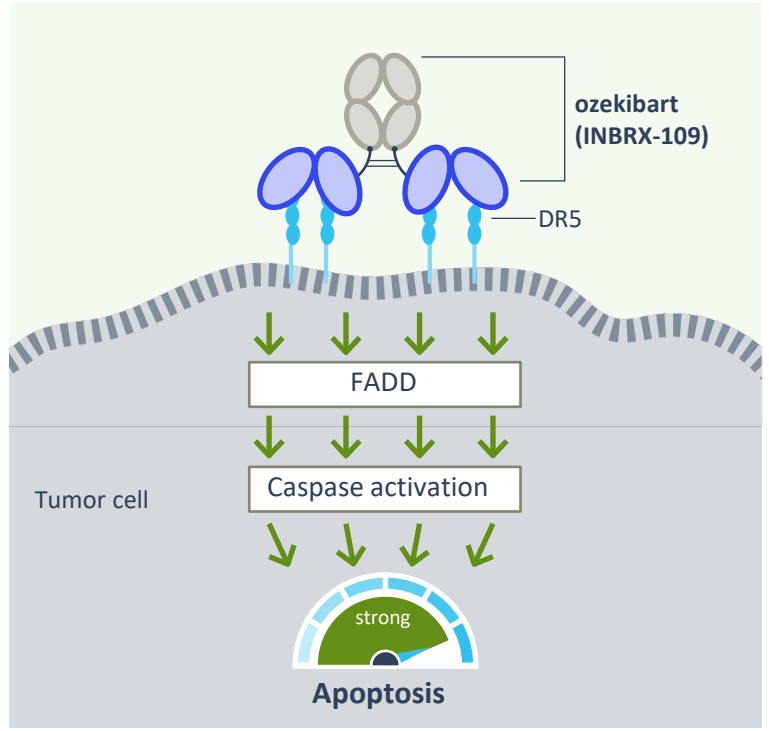
Preclinical data and MOA

ozekibart (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]

Ozekibart (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.

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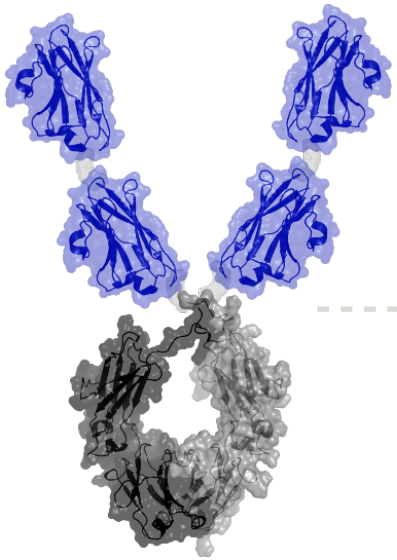
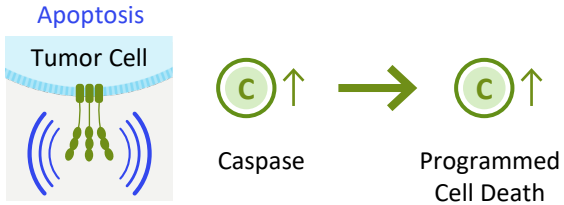
ozekibart (INBRX-109): a next generation DR5 agonist with an optimized balance of efficacy and safety

INBRX-109



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

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]

ozekibart (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

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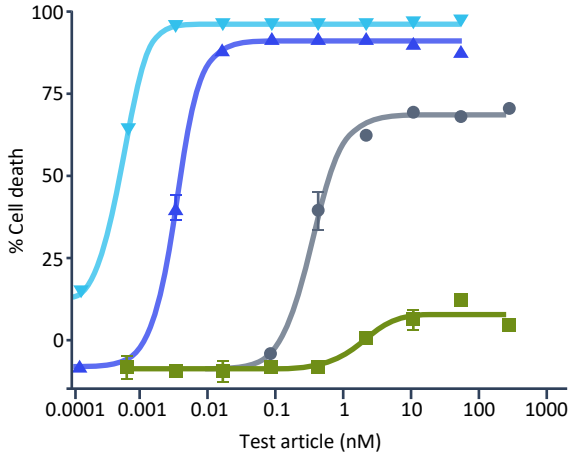
ozekibart (INBRX-109) is precision-engineered for optimal potency and safety

INBRX-109

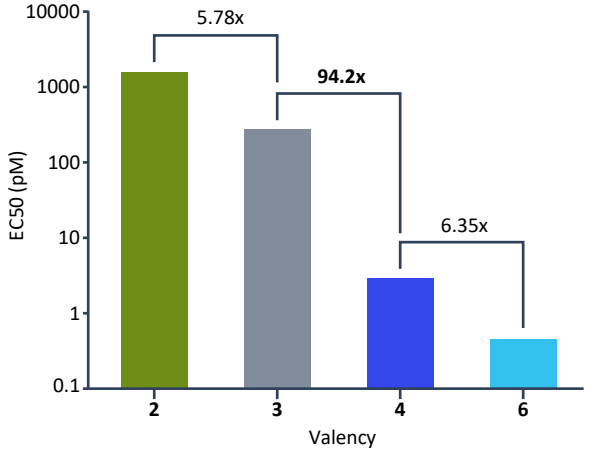
Valency drives both DR5-induced tumor cell death and hepatocyte destruction

Impact of valency on DR5-mediated cell death

Impact of valency on cell death:



Impact of valency on cell death:



anti DR5 mAb
bivalent

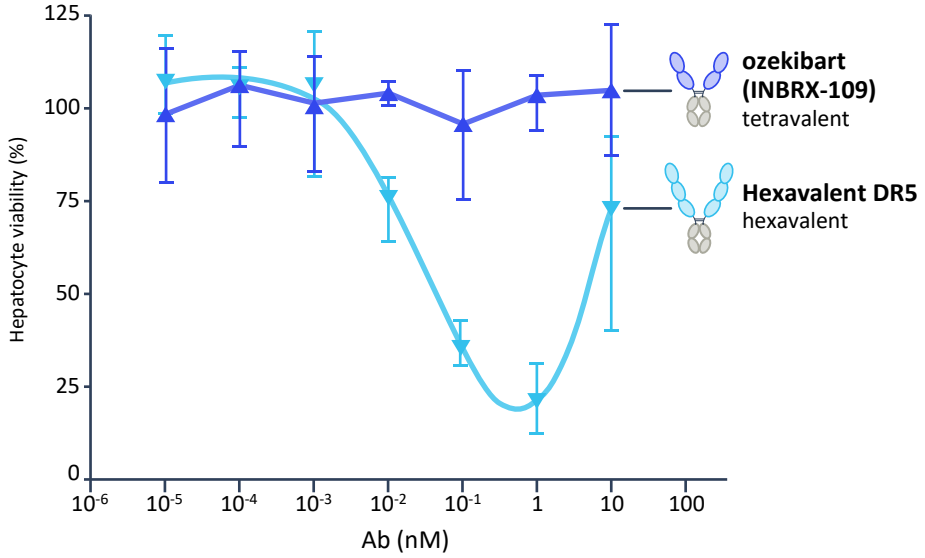
TRAIL
trivalent

ozekibart (INBRX-109)
tetravalent

Hexavalent DR5
hexavalent

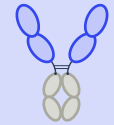
Impact of valency on hepatotoxicity

InSphero 3D inSight™ human liver microtissue model:



ozekibart (INBRX-109) is a best-in-class DR5 agonist with no competition

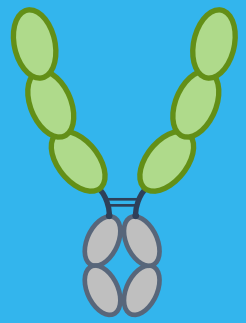
INBRX-109



CANDIDATE	VALENCY	SIZE (KDA)	STATUS
INBRX-109	**Tetravalent**	**105**	Ongoing
TAS-266*	Tetravalent	60	Terminated
Eftozanermin alpha (TRAIL-Fc fusion)	Hexavalent	167	Terminated
GEN1029	Dodecavalent	150 ka (2x mAbs)	Terminated
IGM-8444	Decavalent	~950	Terminated
Dulanermin (recombinant TRAIL)	Trivalent	150	Terminated
Tigatuzumab	Bivalent	150	Terminated
LBY-135			Terminated
Conatumumab			Terminated
Drozitumab			Terminated
Lexatumumab			Terminated

**TAS-266 was ultimately terminated due to hyper-clustering of TAS266 caused by pre-existing anti-sdAb antibodies (PE-ADAs). This hyper-clustering increased the effective valency of TAS266, causing apoptosis of healthy liver cells. The sdAb modifications made to INBRX-109 reduce recognition by PE-ADAs in humans, which lessens the potential for hyper-clustering and increased valency.*

INHIBRX



INBRX-106

hexavalent
OX40 agonist

Goal:

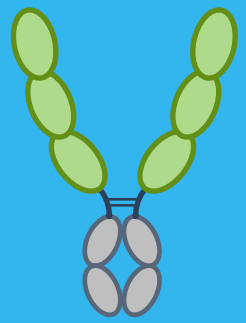
To develop a potent OX40 agonist able to induce robust signal activation

Bivalent OX40 agonists elicit weak downstream signals with limited clinical activity

Previous generation

Hexavalent OX40 agonist with enhanced clustering/signaling

Inhibrx solution



INBRX-106

hexavalent
OX40 agonist

INHIBRX

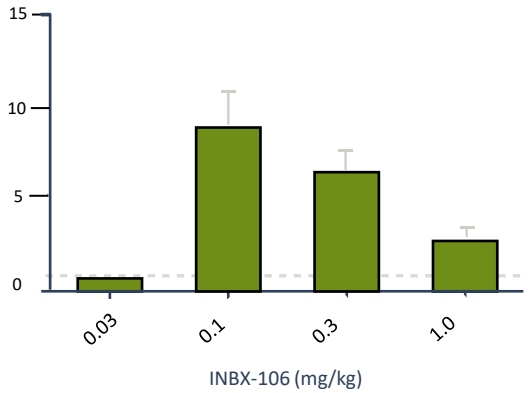
Clinical Data

INBRX-106 is a biologically active drug in patients

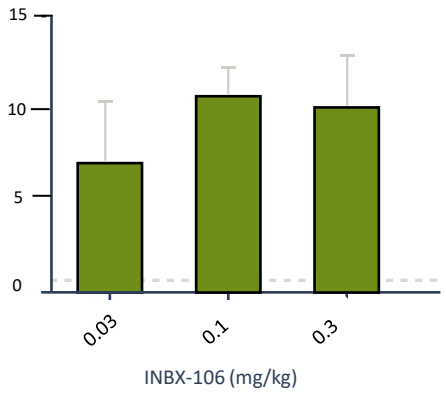
INBRX-106

Max fold change from baseline in percentage of Ki-67+ memory cells

CD4+ T-cells

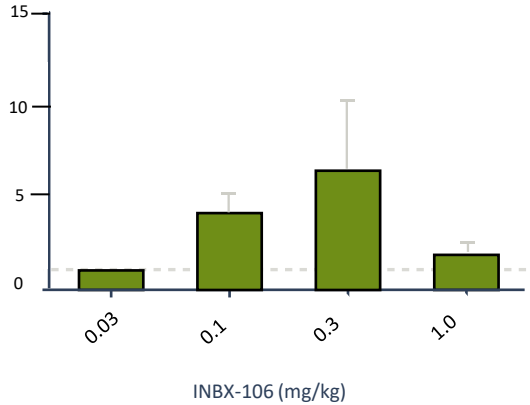


Single-agent

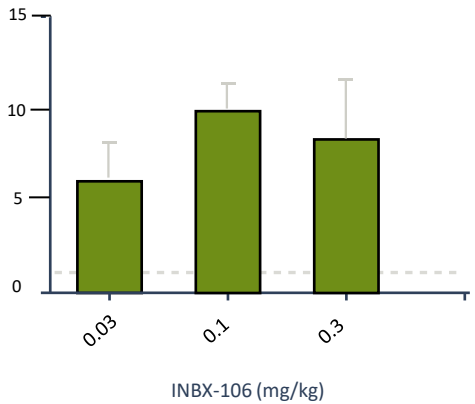


Pembrolizumab combination

CD8+ T-cells



Single-agent



Pembrolizumab combination

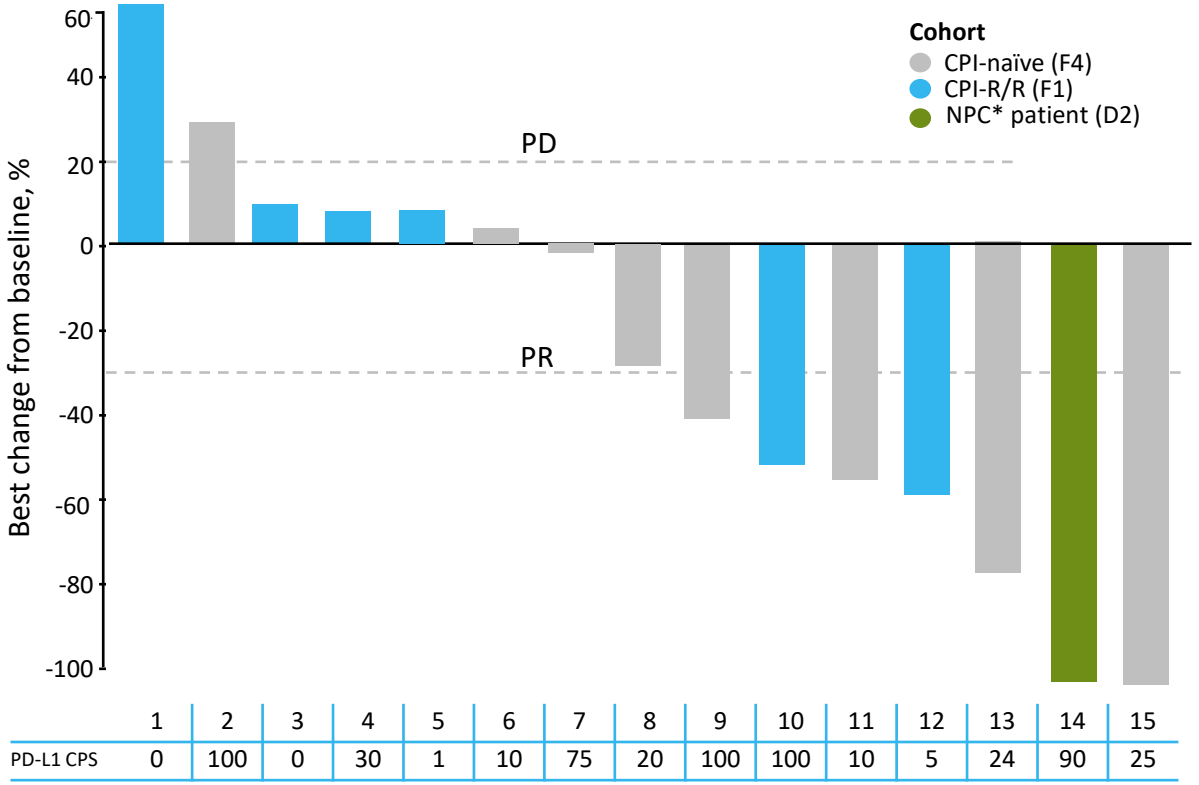
- Observed PD consistent with T-cell co-stimulation by INBRX-106
- INBRX-106 induced dose-dependent proliferation of CD4+ and CD8+ memory T-cells
- Expected bell-shaped response curve was observed
- Based on published data for bivalent OX40 agents, INBRX-106 shows superior single-agent biological activity, as measured by frequency of peripheral activated CD8+ T-cells

Data cut: July 16, 2024. RP2D, recommended phase 2 dose.
[a] Current RP2D selected dose for combination (single agent escalation still in progress).
[b] Combo data at this dose level is representative of cohorts E1 (closed) and F4 (active).

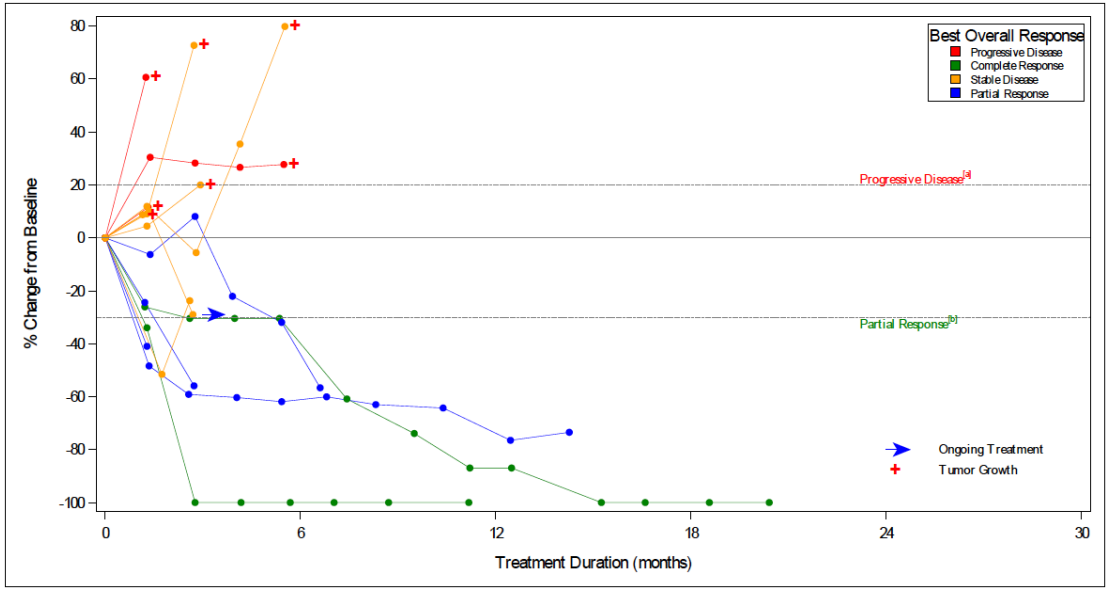
Phase 1 data: PD-L1+ CPI-R/R or CPI-naive HNSCC

INBRX-106 with pembrolizumab

INBRX-106



	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
PD-L1 CPS	0	100	0	30	1	10	75	20	100	100	10	5	24	90	25



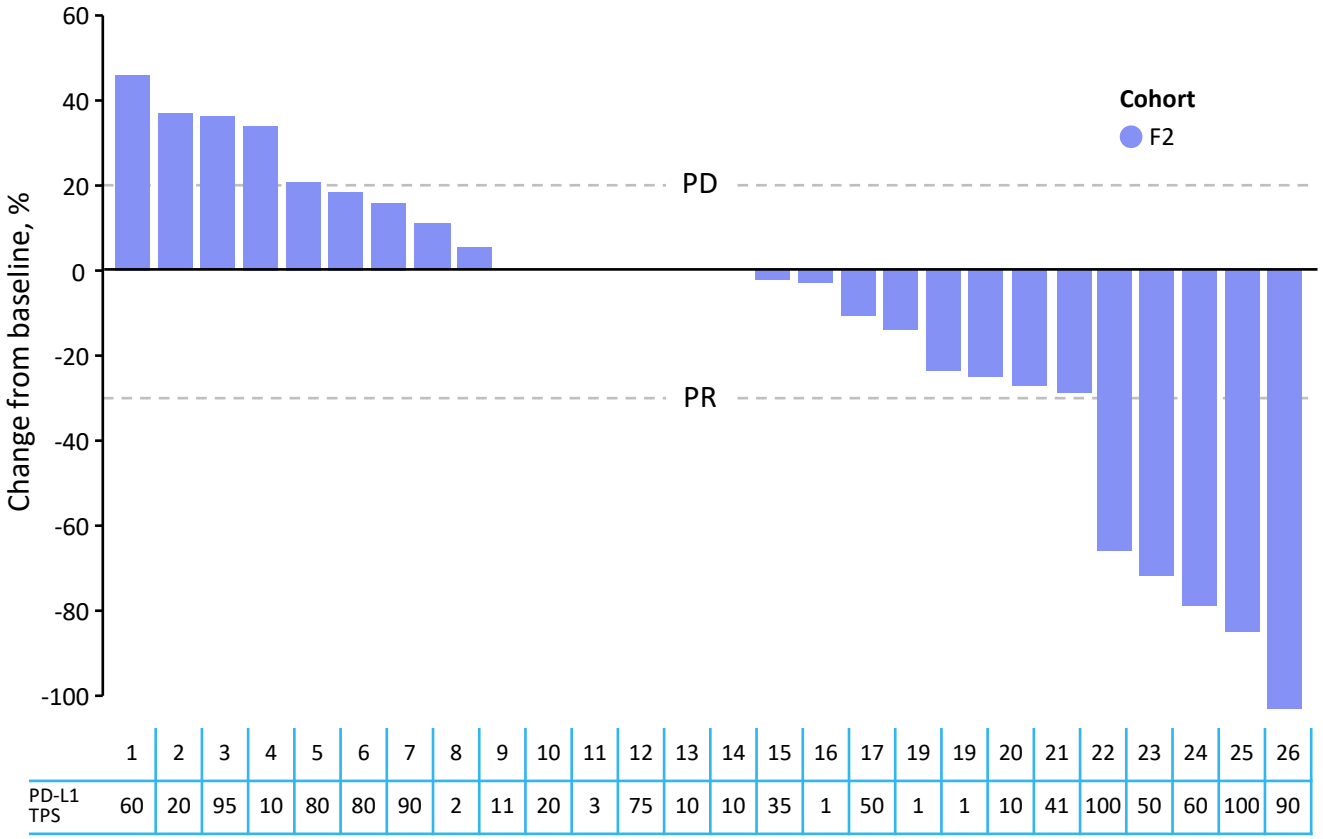
+ The HNSCC patient population included was heterogeneous (1L+) and included CPI-naive patients and those with CPI-R/R disease
+ More than half of patients experienced a reduction in target lesions, including two patients who achieved durable complete responses

Data cutoff: August 1, 2024. Preliminary data from an ongoing database (not fully validated and might slightly evolve with time). *NPC = nasopharyngeal carcinoma

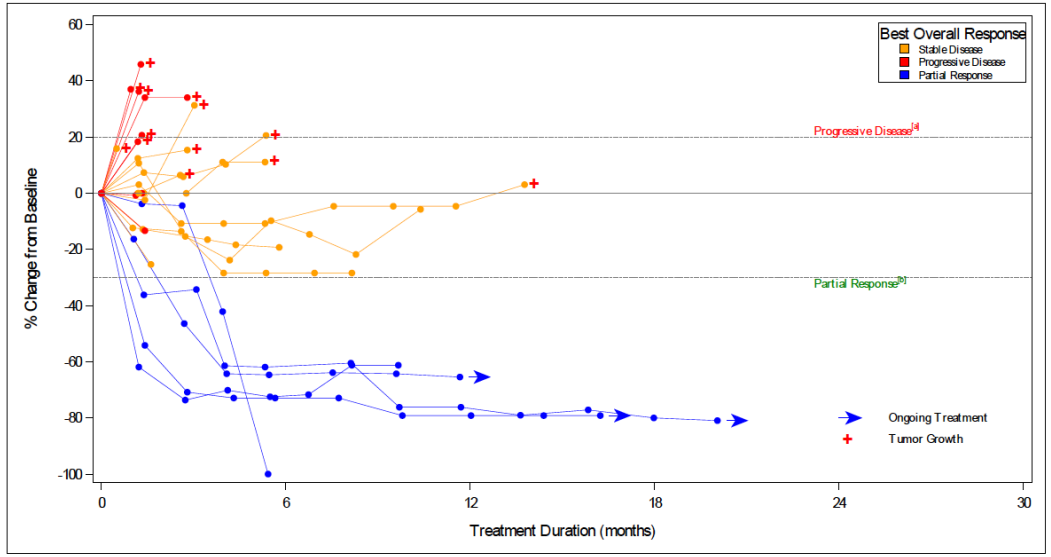
Phase 1 data: PD-L1+ CPI-R/R NSCLC

INBRX-106 with pembrolizumab

INBRX-106



	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	19	19	20	21	22	23	24	25	26
PD-L1 TPS	60	20	95	10	80	80	90	2	11	20	3	75	10	10	35	1	50	1	1	10	41	100	50	60	100	90



+ The NSCLC patients included were heavily pretreated (prior lines: median, 3.5; range, 1-11) and all had received prior CPI (some patients received several lines of CPI treatment)
+ Most patients experienced a reduction in or stabilization of target lesions. Three of the patients have had ongoing responses for more than a year.

Data cutoff: August 1, 2024. Preliminary data from an ongoing database (not fully validated and might slightly evolve with time).

INHIBRX

Seamless Phase 2/3 study in 1L R/M HNSCC with PD-L1 CPS ≥20

INBRX-106 with pembrolizumab

INBRX-106

Phase 2 data readout expected 2H 2025

Proof of concept study:

If successful in HNSCC, has the potential to work broadly against all approved checkpoint indications

Randomization will be stratified by:

+ Disease status (locoregional advanced vs metastatic)
+ HPV status (positive vs negative).
+ ECOG PS (0 vs 1)

Phase 2, Open label

Key inclusion criteria:

R/M HNSCC

PD-L1 CPS ≥20

HPV status confirmed

No prior systemic Tx for R/M HNSCC

R 1:1

- **INBRX-106 + Pembro**
- **Pembro**

Ongoing

Gating Phase 2/3

Primary Criteria:

ORR

Secondary Criteria:

+ DOR
+ CBR
+ PFS_{6m}
+ safety

Phase 3, Double blind

R 1:1

- **INBRX-106 + Pembro**
- **Pembro**

Survival Follow-up

Co-primary endpoint:

PFS and OS.

Secondary endpoints:

ORR, DOR, CBR, TTCx, safety, PROs.

Clinicaltrials.gov (NCT06295731). Protocol version 1.0; January 31, 2024. INBRX-106 to be administered every 3 weeks. Pembro 200 mg to be administered every 3 weeks. 1L, first line; CBR, clinical benefit rate; cORR, confirmed objective response rate; CPS, combined positive score; DOR, duration of response; ECOG PS, Eastern Cooperative Oncology Group performance status; HNSCC, head and neck squamous cell carcinoma; HPV, human papillomavirus; OS, overall survival; PD-L1, programmed cell death 1 ligand 1; pembro, pembrolizumab; PFS, progression-free survival; PFS6mo, progression-free survival rate at 6 months; PRO, patient-reported outcome; R, randomization; R/M, recurrent/metastatic; TTCx, time to chemotherapy; Tx, treatment.

INHIBRX

Ongoing Phase 1/2 trial in metastatic/recurrent NSCLC and HNSCC

INBRX-106

Readouts expected 2H 2025

Key inclusion criteria

M/R NSCLC
<3 prior lines of therapy. PD-L1 TPS ≥50%
or TMB ≥10 mutations/Mb

NSCLC: CPI relapsed/refractory

N=60

R 1:1:1

- **Alternating treatment**
  INBRX-106 alternating Q3W with pembro
- **Priming**
  INBRX-106 loading dose → INBRX-106 + pembro
- **Concurrent**
  INBRX-106 + pembro

Primary endpoints:

+ Objective response rate (ORR) per Response Evaluation Criteria in Solid Tumors (RECIST) version 1.1 and iRECIST, disease control rate (DCR), and duration of response (DOR)
+ Safety

Secondary endpoints:
Pharmacokinetics, immunogenicity, progression-free survival, and overall survival

HNSCC: CPI naïve

Key inclusion criteria

M/R HNSCC (non-NPC) or NPC
≤1 prior line of chemotherapy in metastatic setting
PD-L1 CPS ≥1

N=40

- Non-NPC and other
- NPC

Primary endpoints:

+ ORR per RECIST version 1.1 and iRECIST, DCR, and DOR
+ Safety

Secondary endpoints:
Pharmacokinetics, immunogenicity, progression-free survival, and overall survival

NSCLC: CPI R/R or naive

Key inclusion criteria

M/R NSCLC
Any prior line of therapy PD-L1 TPS ≥0%

N=60

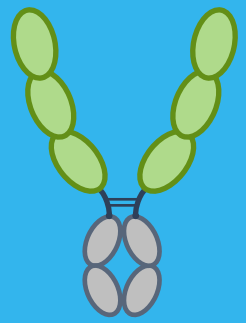
- **Nonsquamous NSCLC**
  INBRX-106 + pembro + pemetrexed + carboplatin[a]
- **Nonsquamous NSCLC**
  INBRX-106 + pembro + pemetrexed + cisplatin[a]
- **Squamous NSCLC**
  INBRX-106 + pembro + (nab-)paclitaxel + carboplatin[a]

Primary endpoint:
Safety

Secondary endpoints:

+ ORR per RECIST version 1.1 and iRECIST, DCR, and DOR
+ Pharmacokinetics, immunogenicity, progression-free survival, and overall survival

ClinicalTrials.gov identifier, NCT04198766. Protocol version 7.0; March 5, 2024. a Chemo will be administered during the first 4 cycles. Pemetrexed can be continued after 4 cycles until progression or up to 35 cycles. Chemo, chemotherapy; CPI, checkpoint inhibitor; CPS, combined positive score; HNSCC, head and neck squamous cell carcinoma; M/R, metastatic/recurrent; NPC, nasopharyngeal carcinoma; NSCLC, non-small cell lung cancer; PD-L1, programmed cell death 1 ligand 1; pembro, pembrolizumab; q3w, every 3 weeks; R, randomization; R/R, relapsed/refractory; TMB, tumor mutational burden; TPS, tumor proportion score.



INBRX-106

hexavalent
OX40 agonist

strong

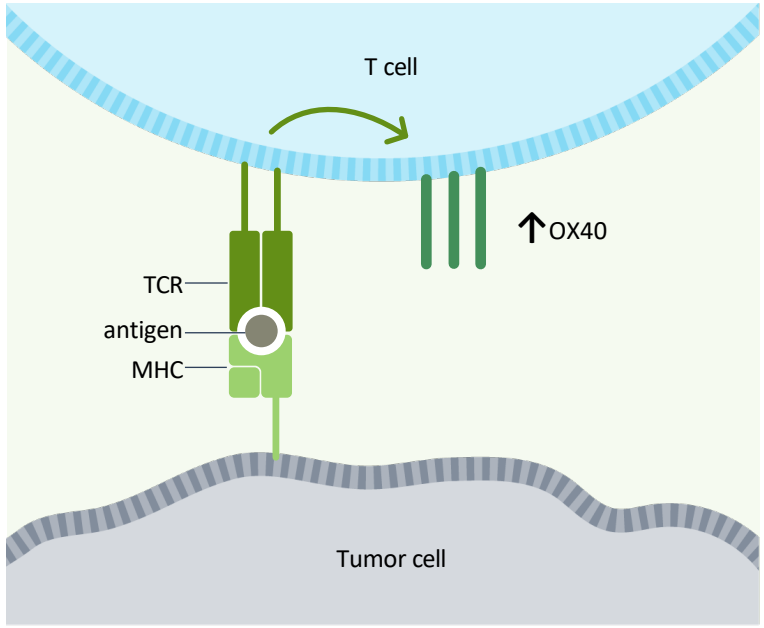
Preclinical Data and MOA

INHIBRx

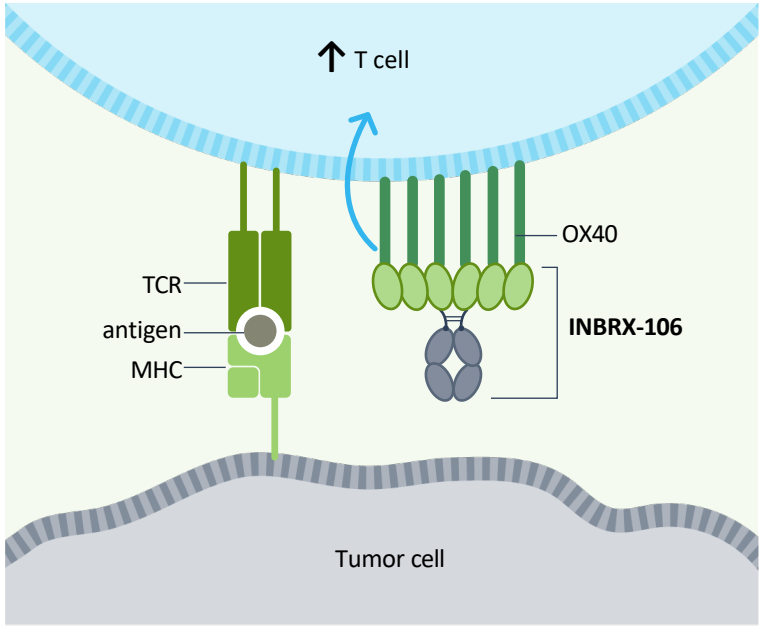
INBRX-106: mechanism of action

INBRX-106

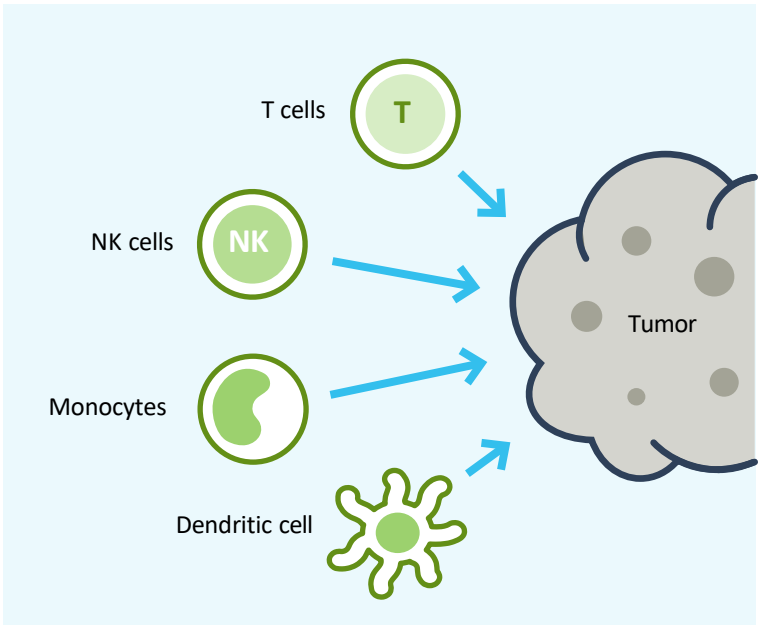
Effective OX40 agonism potentiates the body's immune response towards a tumor



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 bivalent and tri-valent endogenous OX40L amplifying the downstream costimulatory signal.

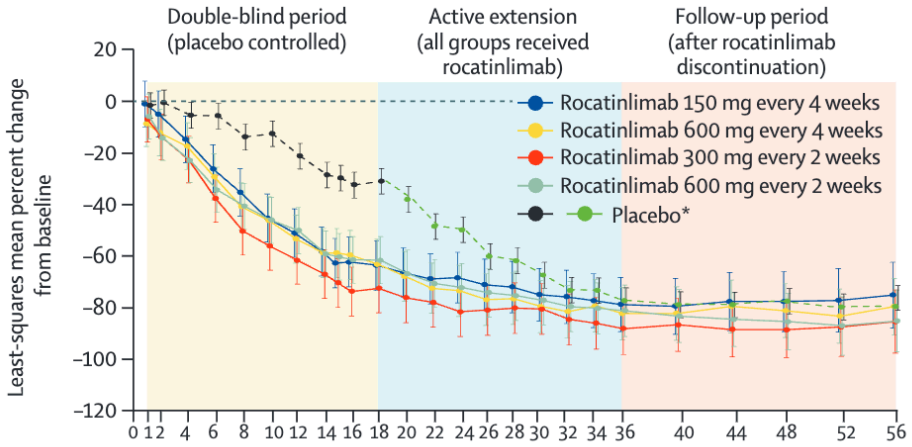


Costimulatory signaling via OX40 and the TCR-MHC receptors induce survival and proliferation of activated antigen-specific CD4 T cells, increases memory T cell generation, CD8+ effector T cells, and suppress the inhibitory capacity of regulatory T cells.

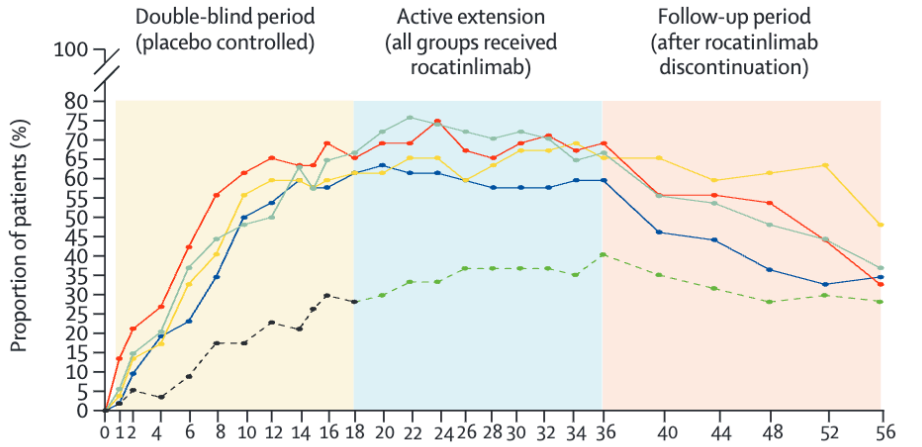
OX40 T-cell costimulation in human disease is validated by OX40 antagonists in autoimmune disease

INBRX-106

An anti-OX40 antibody to treat moderate-to-severe atopic dermatitis: a multicenter, double-blind, placebo-controlled phase 2b study



Percentage change				
Rocatinlimab 150 mg every 4 weeks	−62·2	−68·3	−78·7	−75·0
Rocatinlimab 600 mg every 4 weeks	−59·5	−73·4	−82·3	−79·5
Rocatinlimab 300 mg every 2 weeks	−73·6	−81·6	−88·1	−85·4
Rocatinlimab 600 mg every 2 weeks	−61·4	−72·2	−81·1	−85·1
Placebo	−32·3	−49·7	−77·2	−79·6



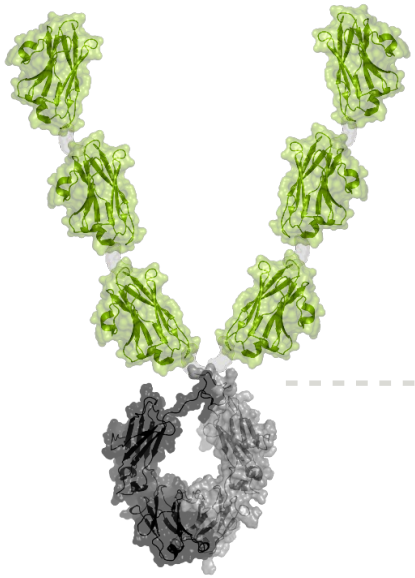
Number of patients (%)				
Rocatinlimab 150 mg every 4 weeks	30 (58%)	32 (62%)	31 (60%)	18 (35%)
Rocatinlimab 600 mg every 4 weeks	31 (60%)	34 (65%)	34 (65%)	25 (48%)
Rocatinlimab 300 mg every 2 weeks	36 (69%)	39 (75%)	36 (69%)	17 (33%)
Rocatinlimab 600 mg every 2 weeks	35 (65%)	40 (74%)	36 (67%)	20 (37%)
Placebo	17 (30%)	19 (33%)	23 (40%)	16 (28%)

Lancet 2023; 401: 204–14

INBRX-106: uniquely designed to maximize OX40 signaling activation strength, leading to enhanced t-cell-driven anti-tumor activity

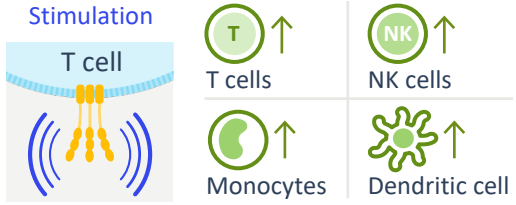
INBRX-106

INBRX-106 is designed to boost anti-tumor T-cell activity by maximizing the strength of 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

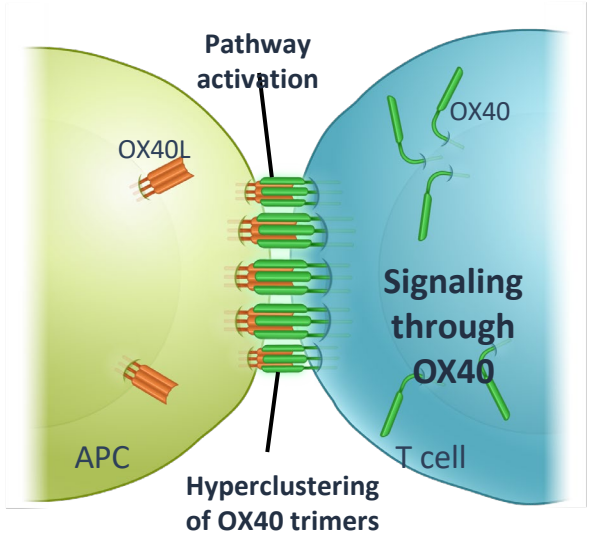
INBRX-106 features:

Hexavalent	Simultaneously engage multiple OX40 to drive enhanced clustering/signaling
Hyperclustering	Receptor hyperclustering enables more efficient coactivation of key OX40 low expressing cells such as $CD8^+$ T-cell activation
Non-Competitive Binding	Complements natural ligand (OX40L) activity
Effector Enabled	Facilitates further higher-order clustering
Smaller Size	sdAb backbone limits molecule size (129 kDa) which may allow for better tumor penetration

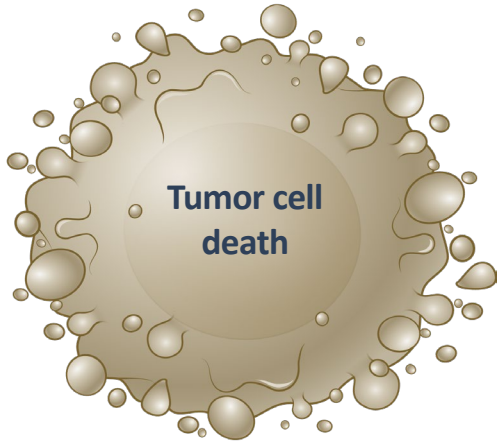
INHIBRX

Receptor hyperclustering induces stronger OX40 signaling and more effective t-cell-driven anti-tumor activity

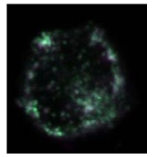
INBRX-106



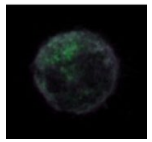
- T-cell proliferation
- T-cell survival
- T-cell differentiation
- Enhanced trafficking of T cells to the tumor
- Robust immune activation
- Decreased Treg-mediated immunosuppression



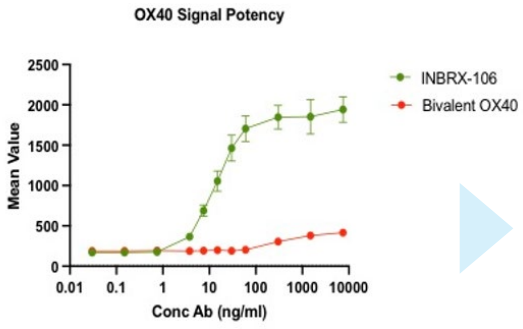
OX40 antibody detection

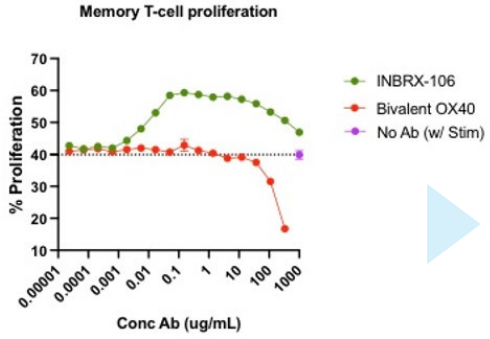


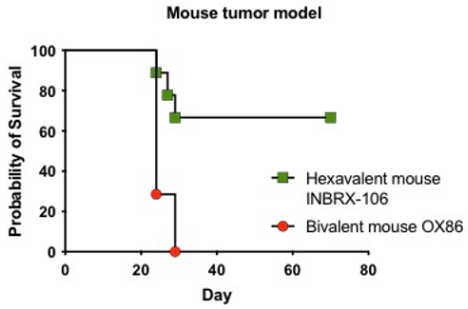
INBRX-106



Bivalent



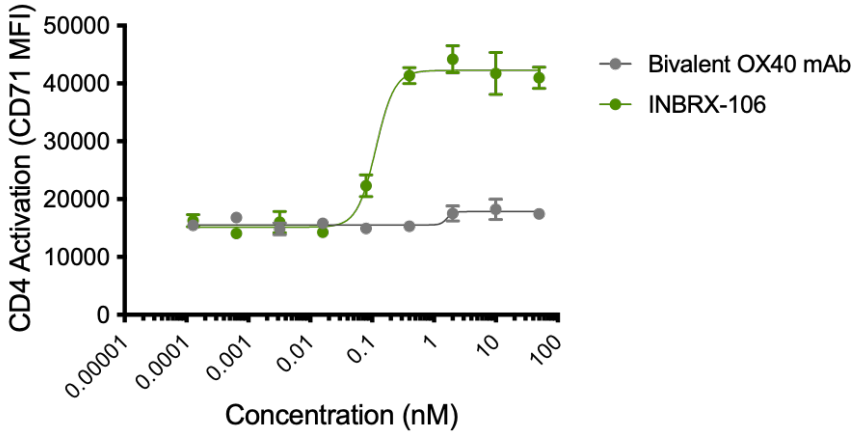




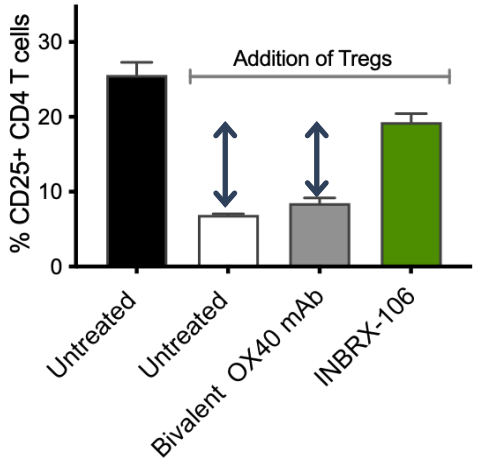
INBRX-106 drives superior CD4+ and CD8+ T-cell activation and reduces T_{reg} suppression

INBRX-106

CD4 T Cell Co-Stimulation[1]

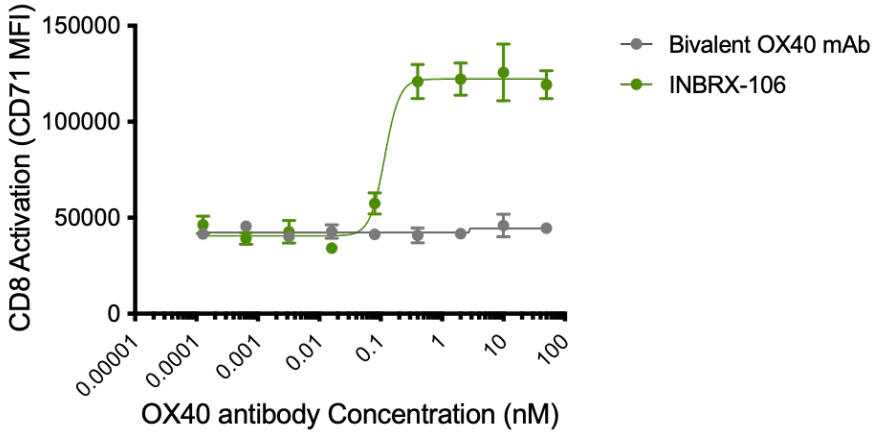


Reversal of T_{reg} Suppression[2]



~2-fold increase vs bi-valent and untreated

CD8 T Cell Co-Stimulation[1]



- 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

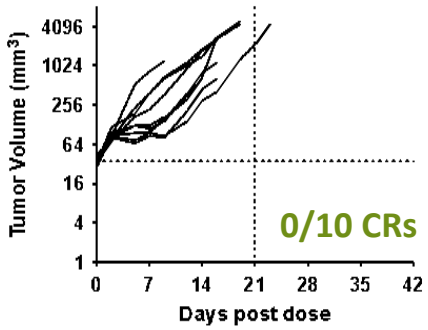
INHIBRX

Hexavalent OX40 and PD-1 antibody combination results in enhanced anti-tumor activity in CPI-resistant tumor models

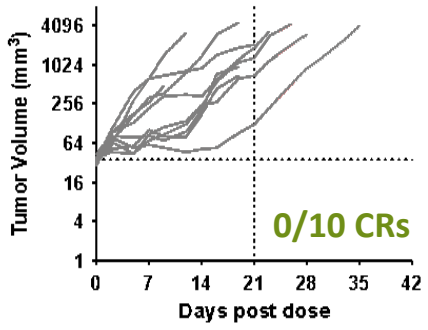
INBRX-106

Syngeneic B16F10 Mouse Tumor Model

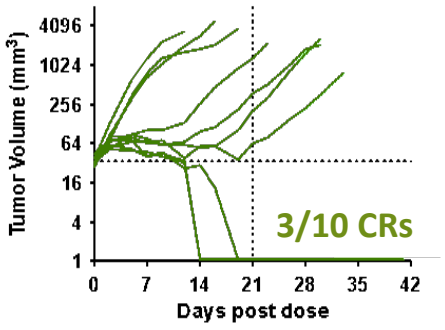
Vehicle



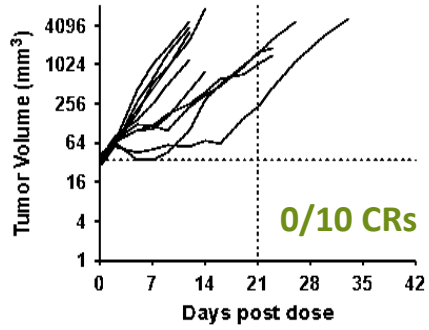
Bivalent OX40 mAb



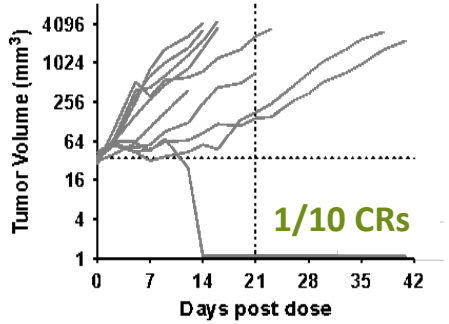
Hexavalent INBRX-106-a*



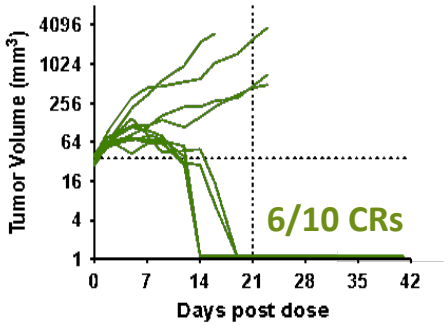
Anti-PD1 Alone



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 is a first and potentially best-in-class multi-valent OX40 agonist

INBRX-106 

CANDIDATES	VALENCY	STATUS
INBRX-106	**Hexa-**	Phase 2/3
GEN1055/BNT315	Dodeca-	Phase 1
MOXR-0916	Bi-	Terminated
GSK-3174998	Bi-	Terminated
BMS-986178	Bi-	Terminated
INCAGN-1949	Bi-	Terminated
ABBV-368	Bi-	Terminated
IBI-101	Bi-	Terminated
MEDI-0562	Bi-	Terminated
PF-04518600	Bi-	Terminated
BGB-A445	Bi-	Terminated
BAT6026	Bi-	Terminated

INHIBRX

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