

INHIBRX Investor Presentation

Innovation Driven
Outcomes Focused

June 2024



INHIBRX

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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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Our company



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

We aim 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 5/30/2024)

○ **\$255.4M**

Cash and cash
equivalents

○ **14.5M**

Common stock
outstanding

○ **19.1M**

Fully diluted
outstanding

Our company



160+

employees with
an experienced
leadership team



>300

ozekibart (INBRX-109)
Patients treated
to date

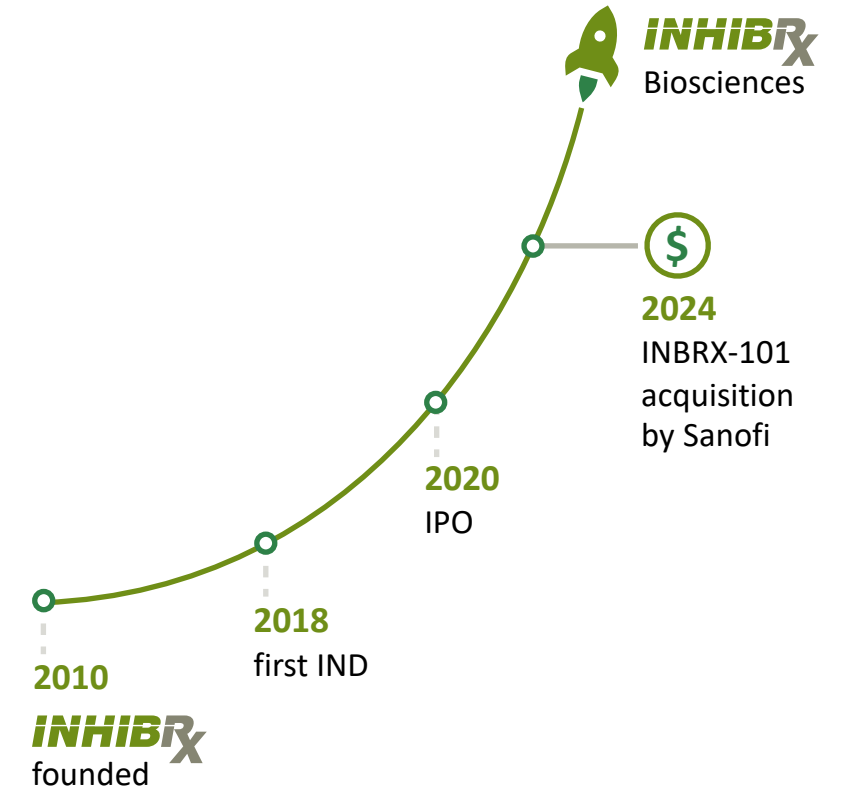
>175

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

Company snapshot:

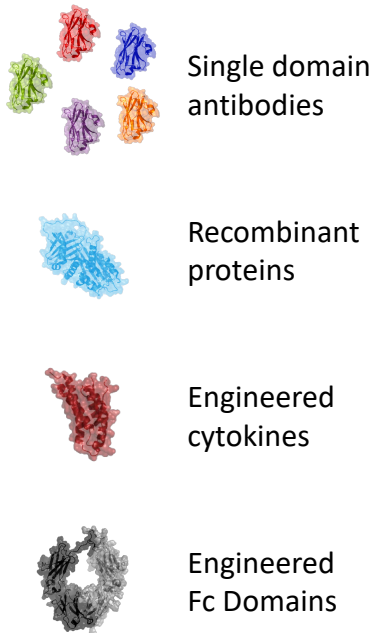


Inhibrx's innovative approach to therapeutic discovery

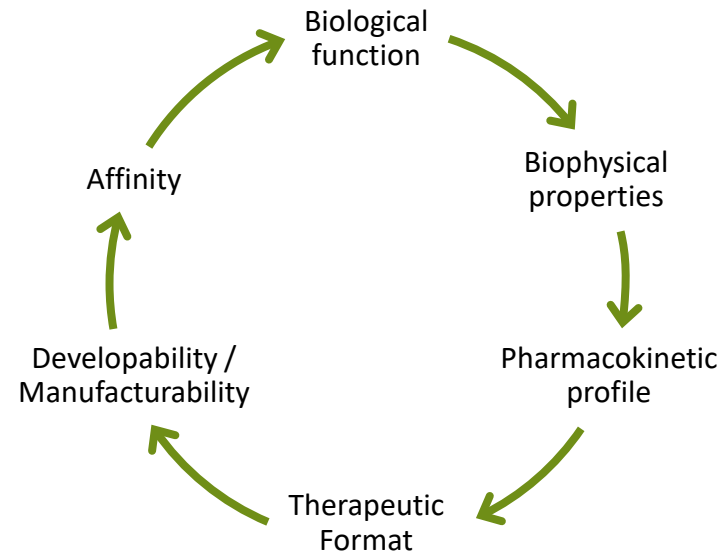
Our core belief:

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

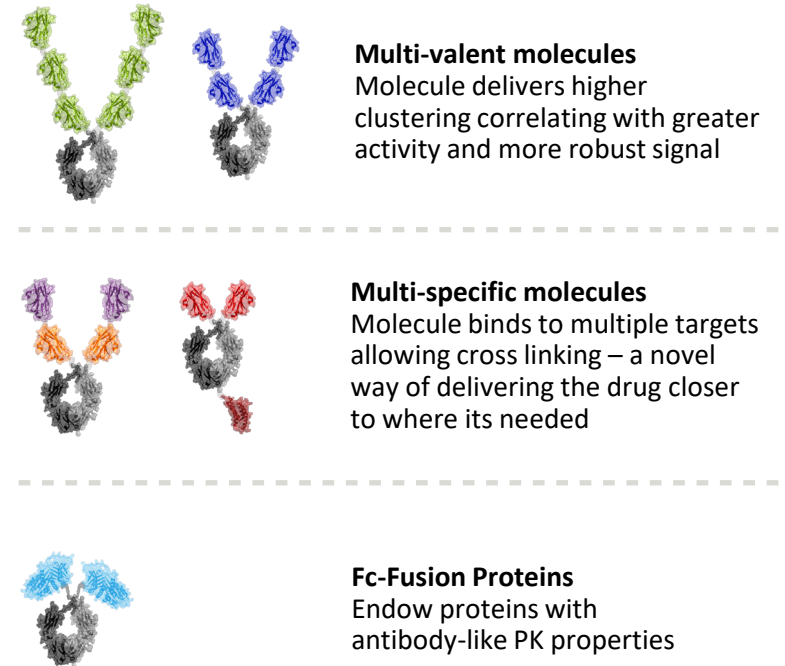
Modular protein engineering platforms



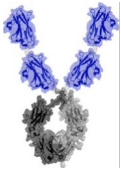
Rapid and iterative optimization process

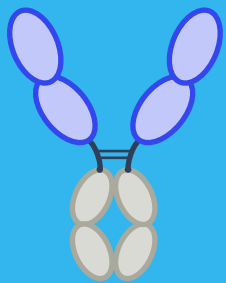


Therapeutics tailored to disease biology



Pipeline

	Phase 1	Phase 2	Phase 3	Milestones	
 ozekibart (INBRX-109) tetraivalent DR5 agonist				1H 2025 Registration- enabling Phase 2 Chondragon chondrosarcoma data	+ 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				+ 1H 2025 Phase 1 randomized, CPI r/r NSCLC data + 2H 2025 Phase 2 HNSSC initial data	+ Robust OX40 agonist superior to bivalent antibodies. + Promising clinical activity in ongoing phase 1/2. + Cohort in NSCLC- SA and in combo with IO and chemotherapy.



ozekibart (INBRX-109)

tetravalent
DR5 agonist

INHIBRX

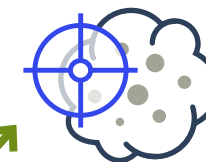
Goal:

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

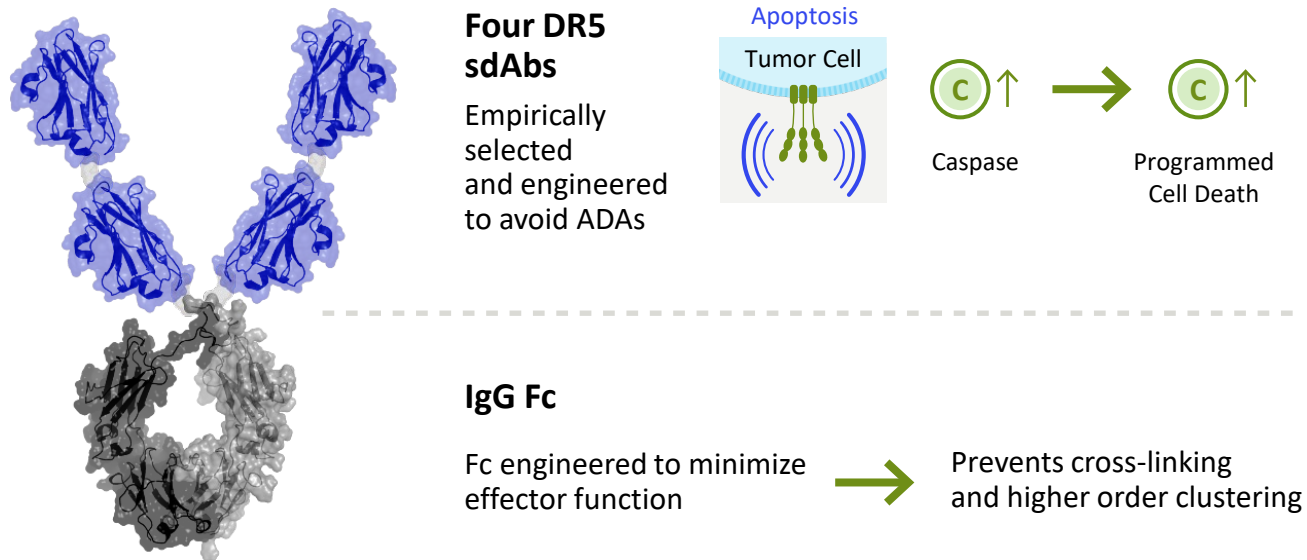
Inhibrx solution

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

INBRX-109



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 oncology¹⁻⁴



ozekibart (INBRX-109) characteristics:

Tetraivalent

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

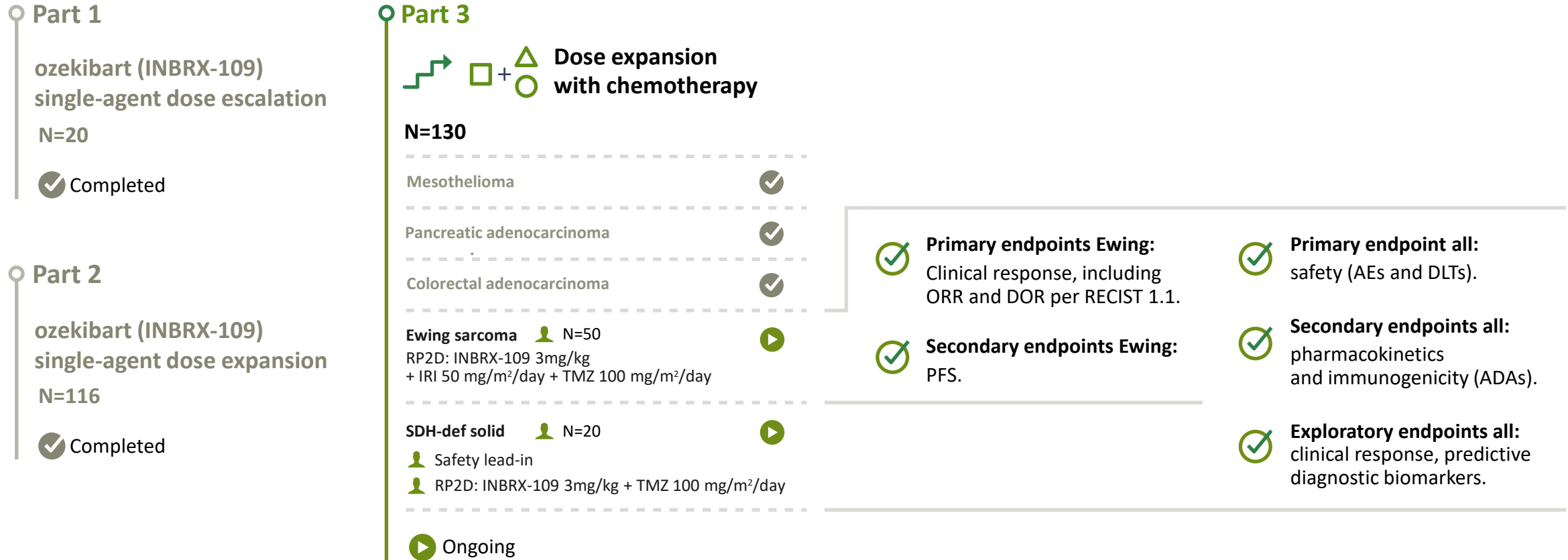
sdAb backbone limits molecule size (106 kDa) which may allow for better tumor penetration

ozekibart (INBRX-109): Phase 1 trial design

INBRX-109



Study of ozekibart (INBRX-109) in patients with locally advanced or metastatic solid tumors, including sarcomas (NCT03715933)



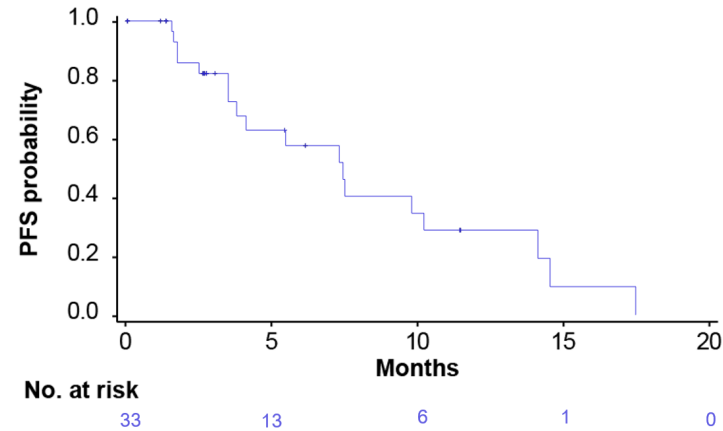
Encouraging mPFS and clinical responses observed in Chondrosarcoma patients treated with ozekibart (INBRX-109)

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 historically¹⁻³

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 options⁴ have demonstrated modest activity as measured by RECIST criteria

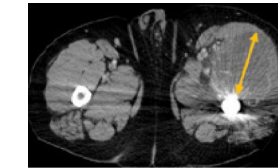
- + ivosidenib resulted in no CR or PR, an SD rate of 52% in patients with advanced chondrosarcoma (n=21)⁵
- + pazopanib in patients with unresectable or metastatic conventional chondrosarcoma (N=47) reported a DCR of 43% at week 16⁶

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).

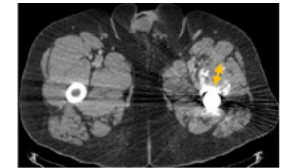
Selected case reports

Partial response:

March 4, 2020 (baseline)



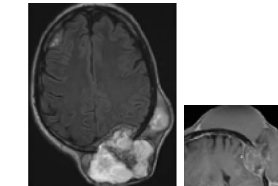
September 3, 2020 (-61%)



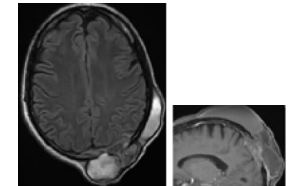
- + 29-year-old white male, histologic Grade 3
- + 61% decrease in target lesions (RECISTv1.1)
- + Patient was on study for 45 weeks

Stable disease:

April 29, 2020



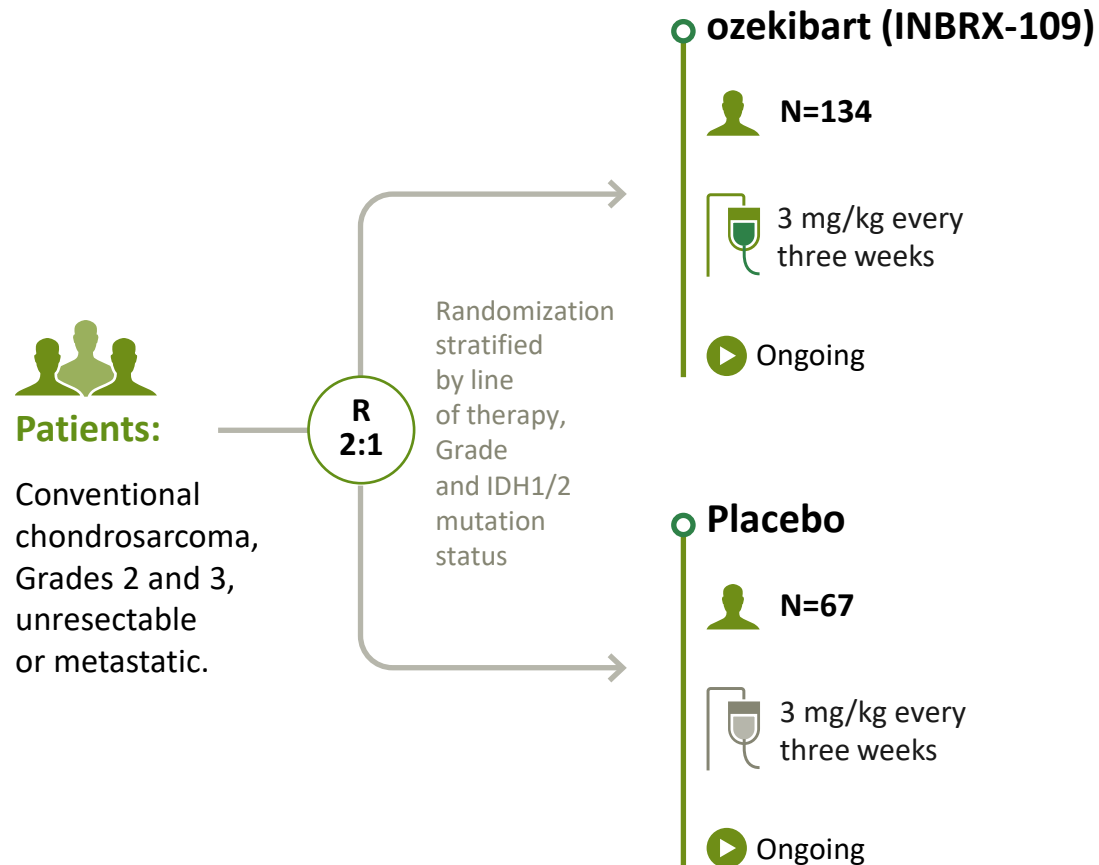
September 4, 2020 (-20%)



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

ozekibart (INBRX-109) Phase 2 registration enabling study in chondrosarcoma

INBRX-109



- ✓ **Primary endpoint:**
Progression free survival.
- ✓ **Secondary endpoints:**
Overall survival, quality of life, overall response rate, duration of response, disease control rate, safety, etc.
- ✓ **Completion projected 2H 2024**
- ✓ **DSMB reviewed interim analyses in April 2024 and made the recommendation for trial continuation**

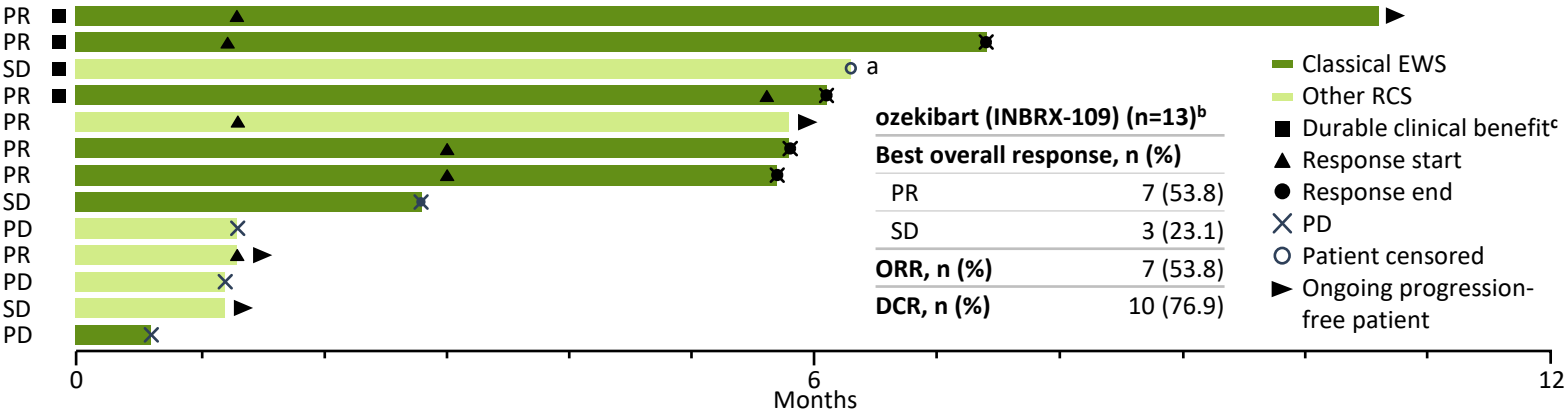
+ FDA fast track designation and orphan-drug designation

+ EMA orphan-drug designation

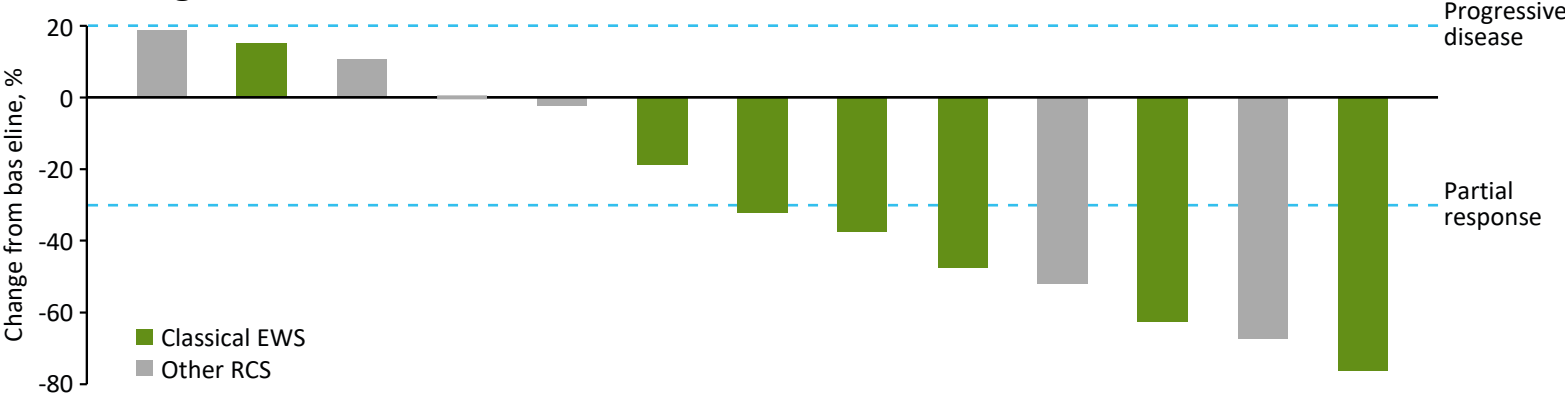
ozekibart (INBRX-109) in combo with IRI/TMZ in metastatic, unresectable Ewing sarcoma



Best tumor response



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.

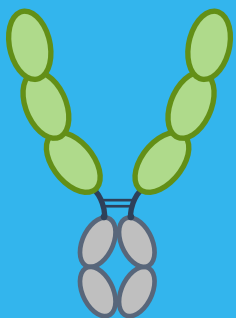
53.8% 7 patients who achieved partial responses (53.8%), 5 of which were observed in classical EWS patients (71.4%) and 2 of which were observed in RCS patients (33.3%)

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

12 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**

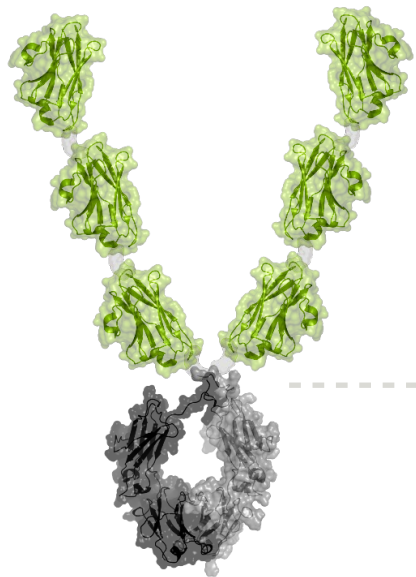
Inhibrx solution

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

INBRX-106

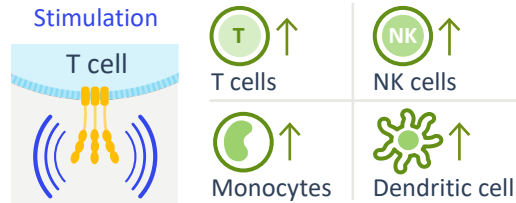


INBRX-106 is designed to boost anti-tumor T-cell activity by potently activating the OX40 co-stimulatory pathway



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

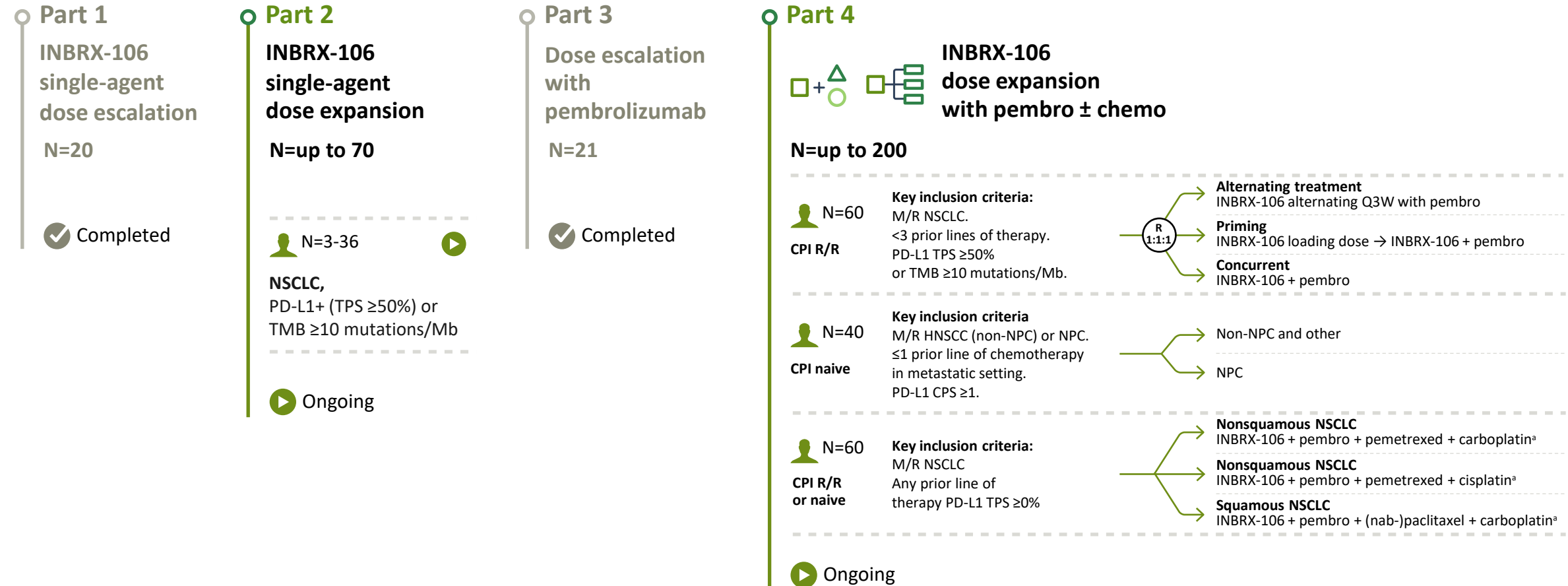
sdAb backbone limits molecule size (129 kDa) which may allow for better tumor penetration

INBRX-106 study design

INBRX-106



Phase 1/2 study of INBRX-106 ± pembrolizumab in adults with locally advanced or metastatic solid tumors—cohorts now enrolling



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.

INHIBRX

Seamless Phase 2/3 study of the hexavalent OX40 agonist INBRX-106 with pembrolizumab in 1L R/M HNSCC with PD-L1 CPS ≥ 20

INBRX-106

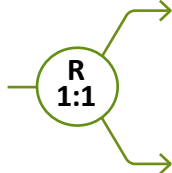


Phase 2, Open label



Key inclusion criteria:

R/M HNSCC
PD-L1 CPS ≥ 20
HPV status confirmed
No prior systemic Tx
for R/M HNSCC



INBRX-106
+ Pembro

Pembro

Gating Phase 2/3

Criteria:

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

Phase 3, Double blind

Survival Follow-up



R
1:1

INBRX-106
+ Pembro

Pembro



Co-primary endpoint:
PFS and OS.



Secondary endpoints:
cORR, DOR, CBR,
TTCx, safety, PROs.



Ongoing



Ongoing

Randomization will be stratified by:

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

ClinicalTrials.gov identifier, 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; PFS_{6mo}, progression-free survival rate at 6 months; PRO, patient-reported outcome; R, randomization; R/M, recurrent/metastatic; TTCx, time to chemotherapy; Tx, treatment.

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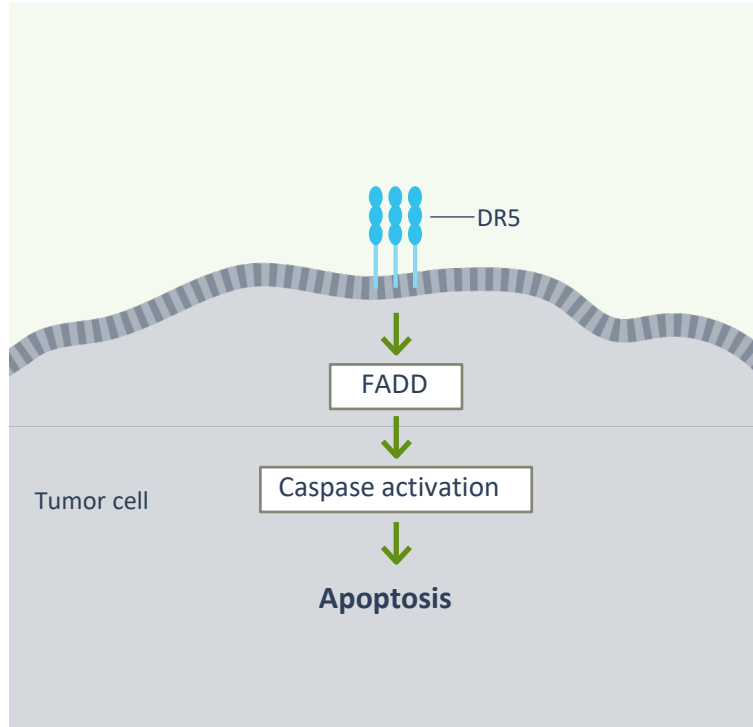


INHIBRX

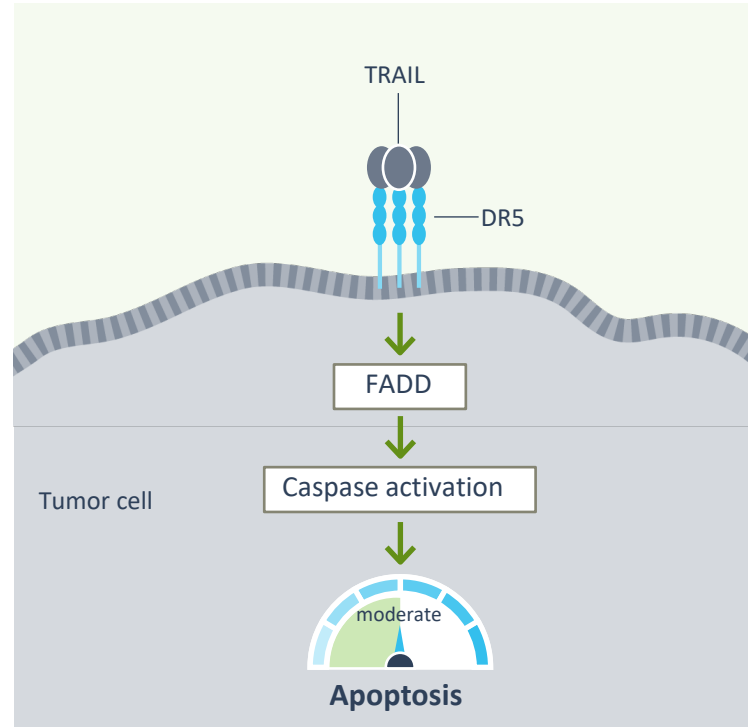
Appendix

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

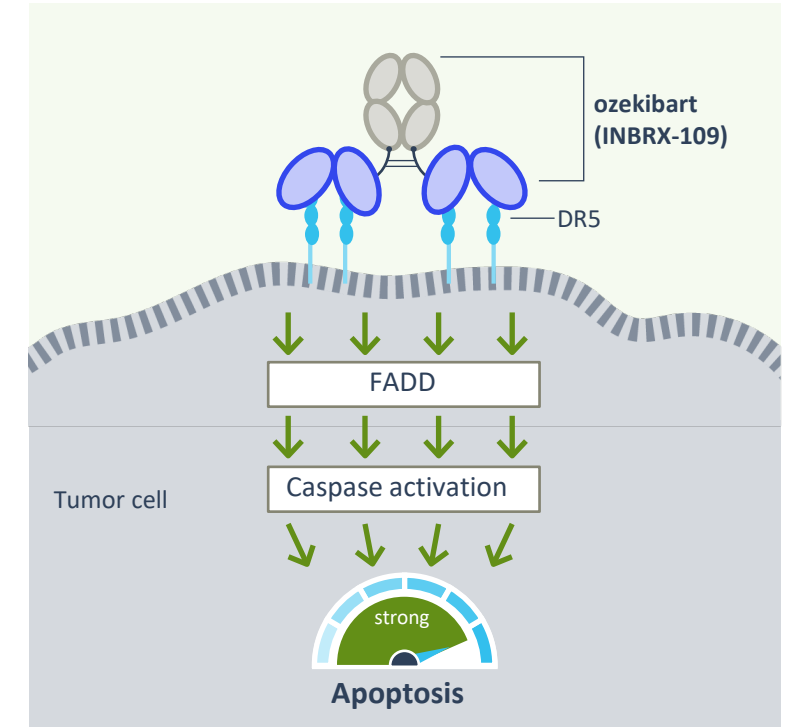
INBRX-109



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 surveillance⁵



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 activity⁶⁻⁸



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

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

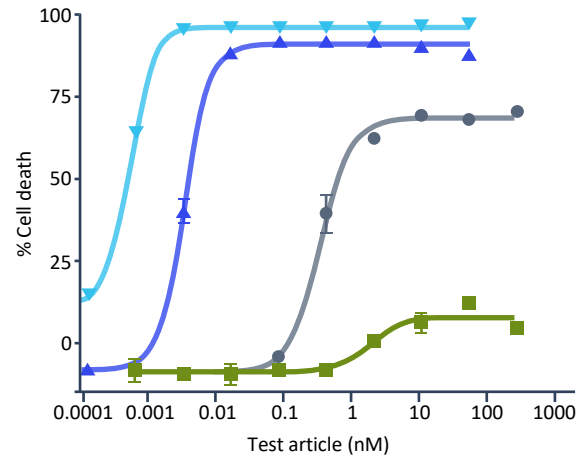
INBRX-109



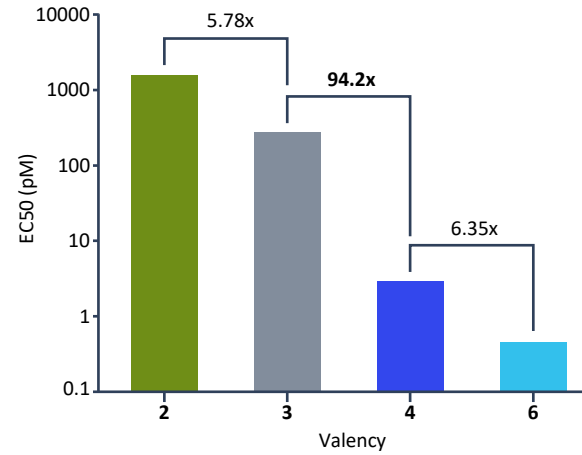
Valency drives both DR5-induced tumor cell death and hepatocyte destruction

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



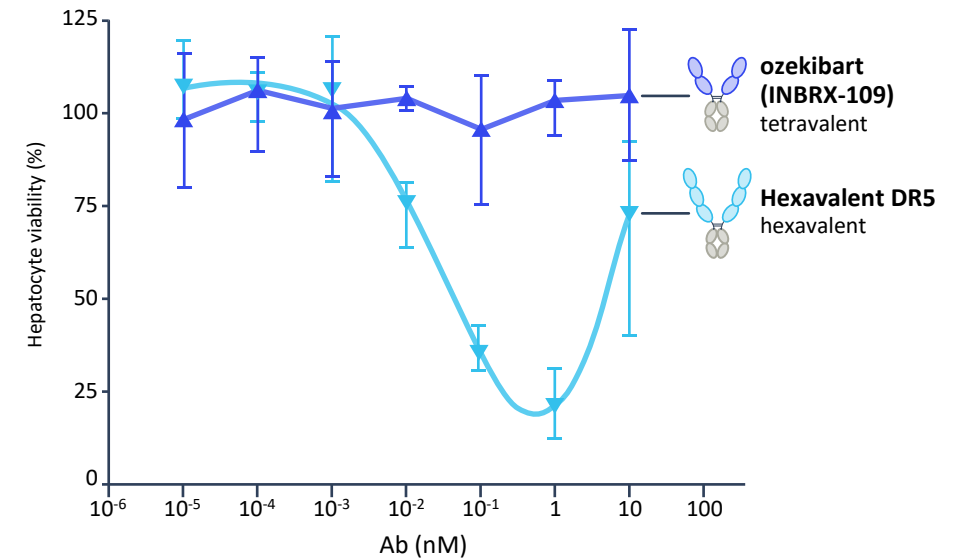
ozekibart (INBRX-109)
tetravalent



Hexavalent DR5
hexavalent

Impact of valency on hepatotoxicity

InSphero 3D inSight™ human liver microtissue model:



ozekibart
(INBRX-109)
tetravalent

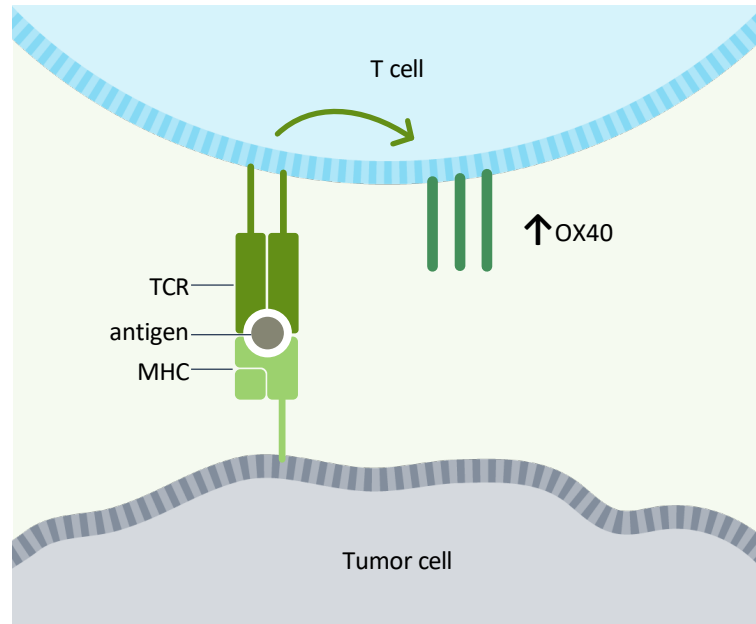
Hexavalent DR5
hexavalent

INBRX-106: mechanism of action

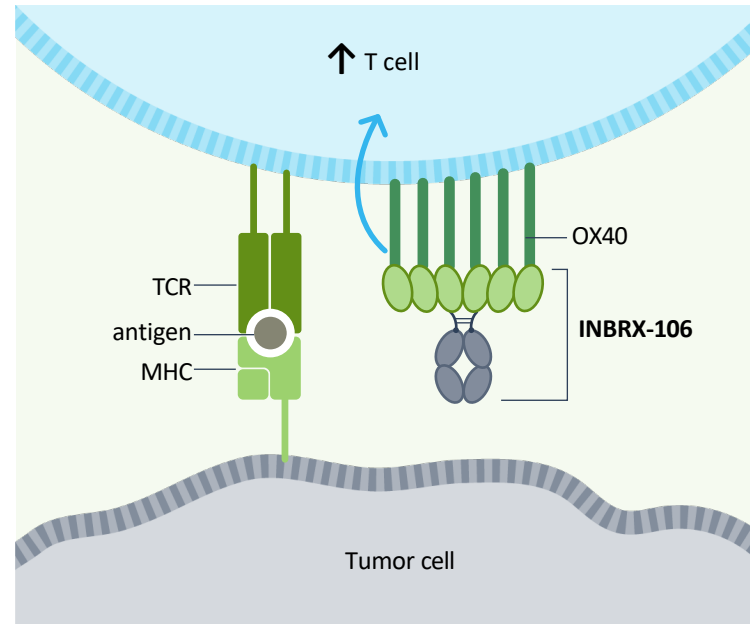
INBRX-106



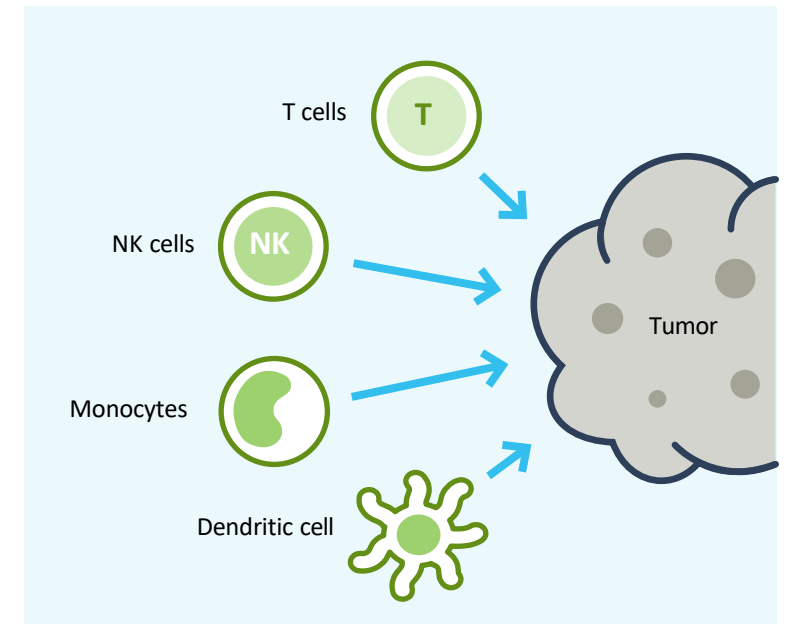
Effective OX40 agonism potentiates the body's immune response towards a tumor



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.



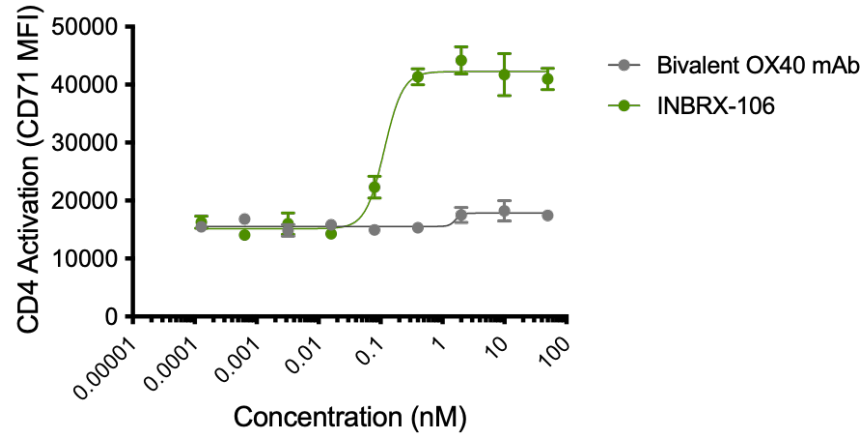
Costimulatory signaling via OX40 and the TCR-MHC receptors induce survival and proliferation of activated antigen-specific CD4 T cells, increases memory T cell generation, CD8+ effector T cells, and suppress the inhibitory capacity of regulatory T cells.

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

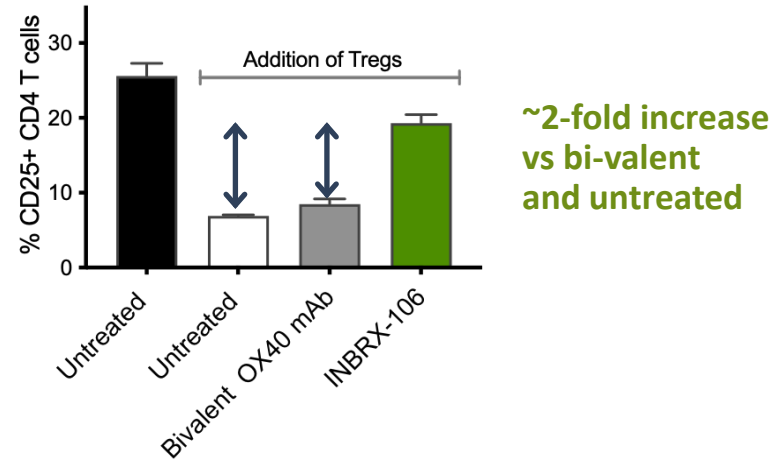
INBRX-106



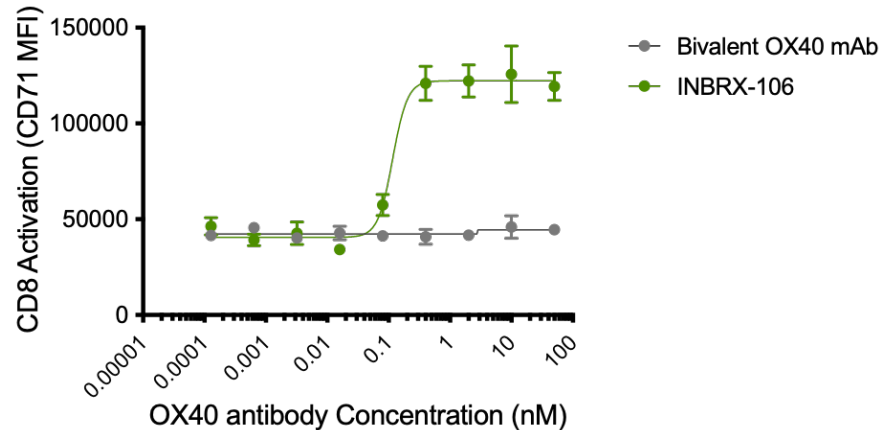
CD4 T Cell Co-Stimulation¹



Reversal of T_{reg} Suppression²



CD8 T Cell Co-Stimulation¹

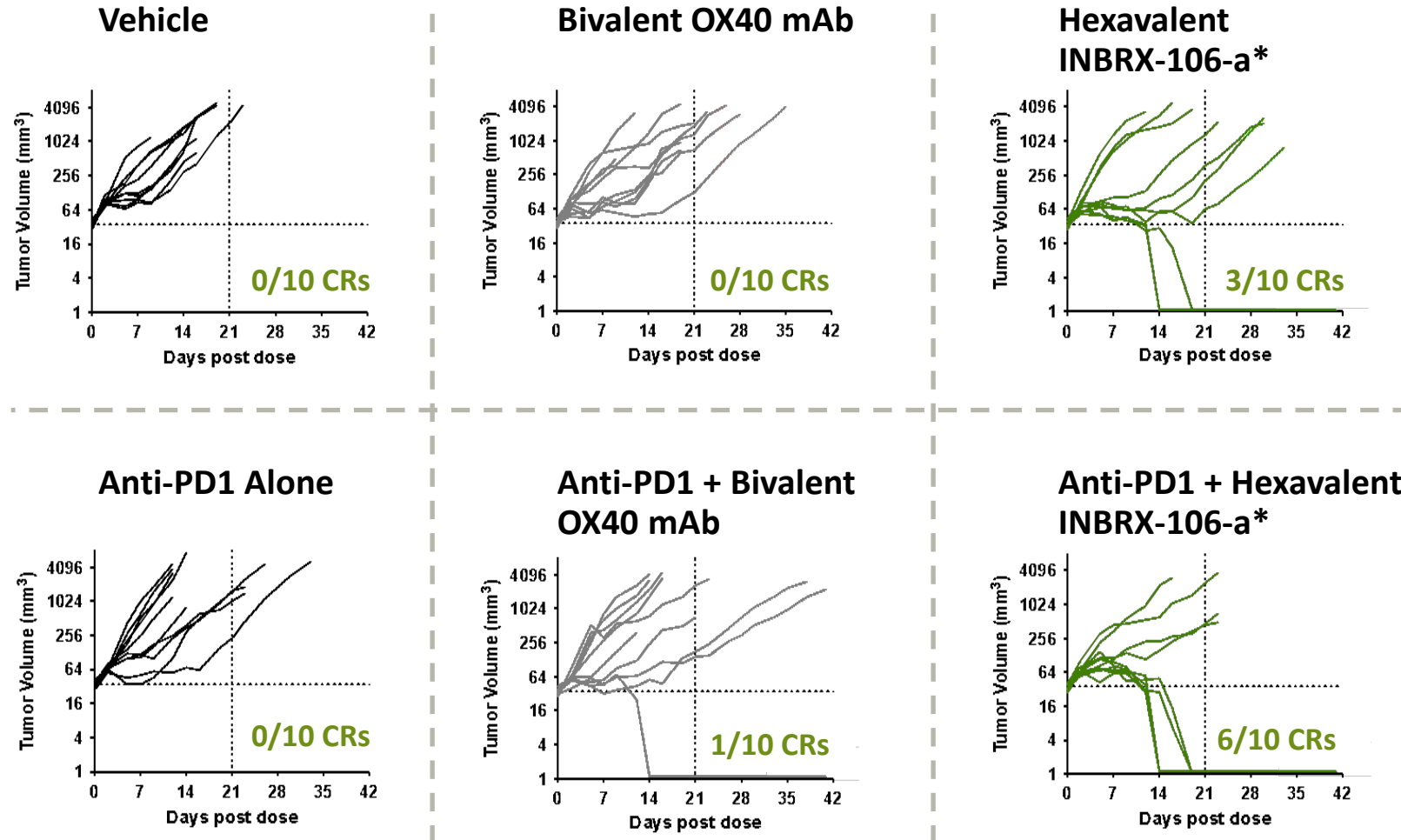


- ✓ Hexavalent INBRX-106 engagement drives superior co-stimulation of both CD4 and CD8 T-cells versus bi-valent OX40 mAbs
- ✓ INBRX-106, but not bivalent OX40 mAb, reduces regulatory T-cell (T_{reg}) mediated suppression of effector T-cells (T_{eff})

Valency drives OX40 agonism in CPI-resistant tumor models

Syngeneic B16F10 Mouse Tumor Model

INBRX-106



- ✓ 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