INHIBRX Investor Presentation

Innovation Driven
Outcomes Focused





November 2026

INHIBRX

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

Key financial highlights

(as of 9/30/2026)

$199.5M
Cash and cash equivalents

14.8M
Common stock outstanding

~110
employees with an experienced leadership team

19.7M*
Fully diluted 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

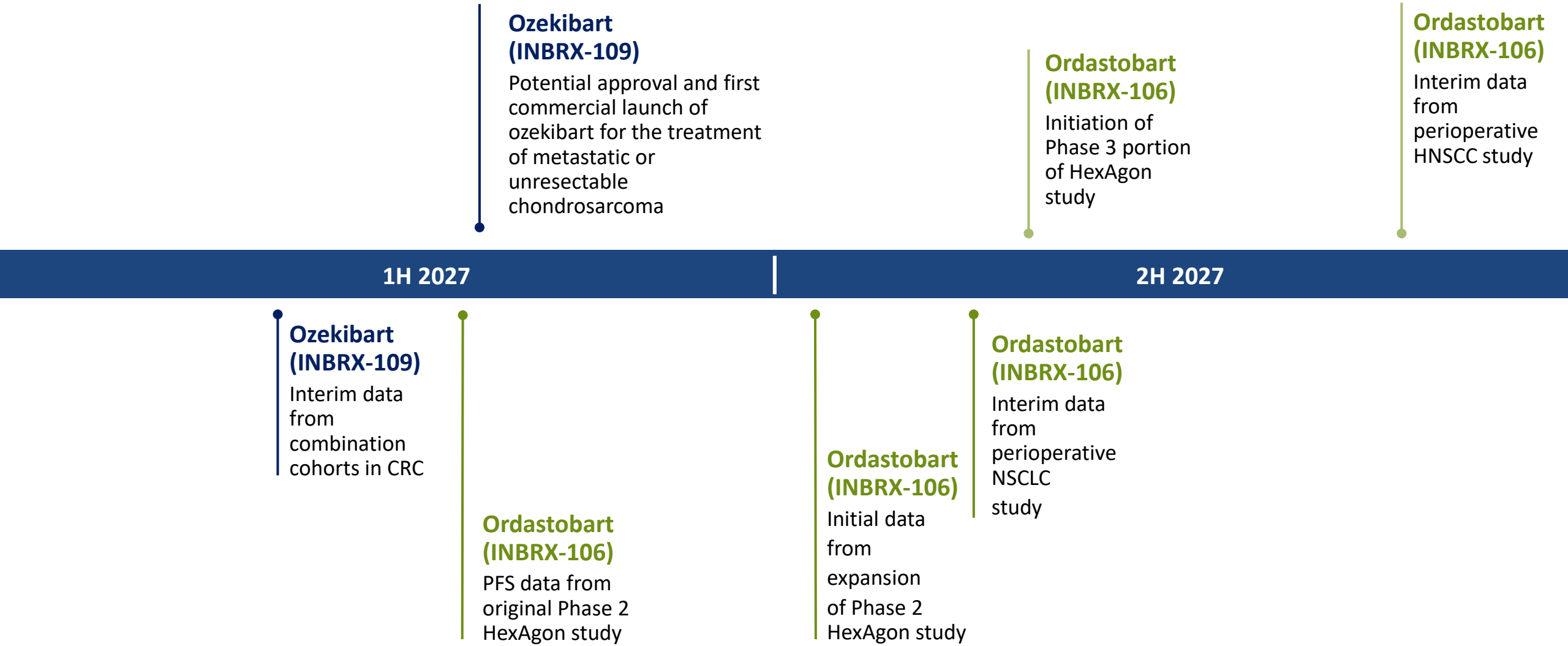
>175
Ordastobart (INBRX-106) Patients treated to date

In-house expertise

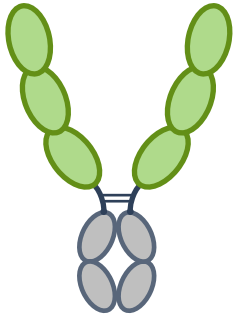
- Discovery
- Protein engineering
- Cell biology
- Translational research
- Chemistry
- Manufacturing and controls
- Clinical development and operations
- Commercial

** Includes 4.0M employee and BOD option reserve and approximately 1M pre-funded warrants and 0.2M warrants outstanding*

Near term expected milestones







Ordastobart (INBRX-106)

Boosting PD-1 efficacy through next-generation hexavalent OX40 agonism

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Ordastobart (INBRX-106) executive summary

Ordastobart is the first clinically active OX40 agonist and T-cell costimulatory therapy

Combination with pembro demonstrates superiority to pembro mono in 1LR/M HNSCC

- ORR: 48.3% vs 26.5%
- Depth of Response
- CR Rate: 13.8% vs 0%
- PFS: 9.6 months vs 4.9 months
- PFS6 Improvement: (72.4% vs. 42.8%)
- These data are supported by superior t-cell proliferation (up to 15-fold increase) and activation (up to 4-fold increase) vs pembrolizumab alone

Potentially practice-changing efficacy profile in HPV+ patients

- ORR: 80% vs 33%
- CR Rate: 30% vs 0%
- PFS Data: NR vs 4.6 months
- PFS6 Rate: 90% vs 33%
- Expansion of Phase 2 to include 50 additional HPV+ patients may serve as potential path to accelerated approval by end of ‘28/early ‘29. HPV+ HNSCC is an open $4B+ market

Large expansion opportunity in highly immunogenic tumors

- Bladder, NSCLC, Melanoma, MSI High/TMB High, TNBC present $50B+ commercial opportunity
- Ongoing neoadjuvant lung cancer study serves as proof of concept for broad expandability with pembrolizumab across highly immunogenic tumors

OX40 agonism enhances anti-tumor T cell activity in combo with PD-1 blockade

INBRX-106

T-cell activation critical steps:

The ignition
Signal 1: TCR tumor antigen recognition

Patients must have an immune response to their tumor(s) for signal 2 and check point inhibition to be impactful

The accelerator
Signal 2: OX40 T-cell co-stimulation

OX40 agonism enhances proliferation, survival and memory formation for tumor specific T-cells

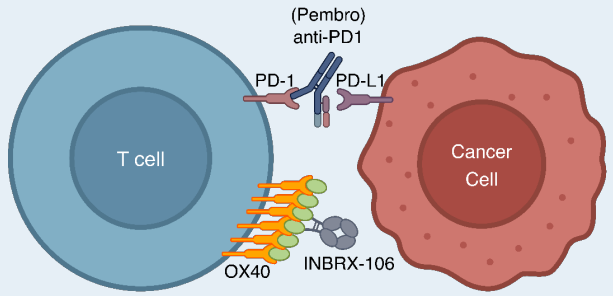
The brake
Check point inhibition

PD-1 signaling (in T-cells) activated via PD-L1 expressed in APCs and tumor cells dampens signal 1 and 2. PD-1 inhibition is necessary to remove this brake

Ordastobart is designed to:

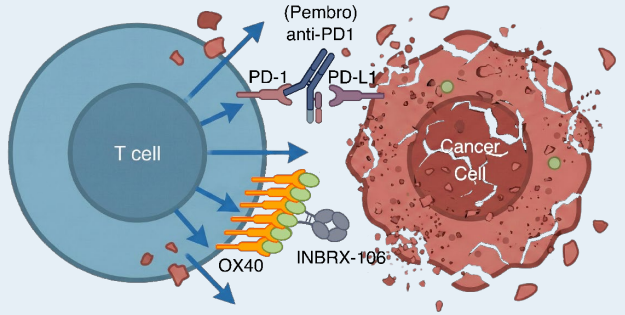
- Supercharge the immune system against the tumors [1-4]
- Reverse immune suppression [1-4]
- **Complement checkpoint inhibitors** (anti-PD-1/PD-L1), enhancing activity

Optimized T cell activation by OX40 agonism and PD-1 blockade



T cell activation restored/enhanced

Synergistic Tumor destruction via Ordastobart and Pembro



Dual Immunotherapy:
Releasing the Brakes and Stepping on the Gas

[1] Croft. Ann Rev Immunol 2010. [2] Salek-Ardakani, Curr Imunol Rev 2006. [3] Weinberg, Immunol Rev 2011. [4] Piconese, J Exp Med 2008

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Underwhelming activity among the 1st generation of bivalent OX40 therapeutics leads to Ordastobart hexavalent OX40

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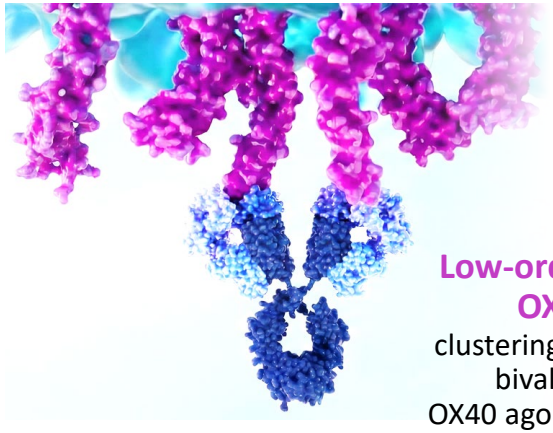
Bivalent OX40 Agonists



Low-order OX40 clustering by bivalent OX40 agonist

Next Generation

Hexavalent OX40 Agonist



High-order OX40 clustering by **Ordastobart**

Limitations of bivalent attempts

Suboptimal target engagement — Bivalent IgG formats fail to drive high-order OX40 clustering required for co-stimulatory signaling

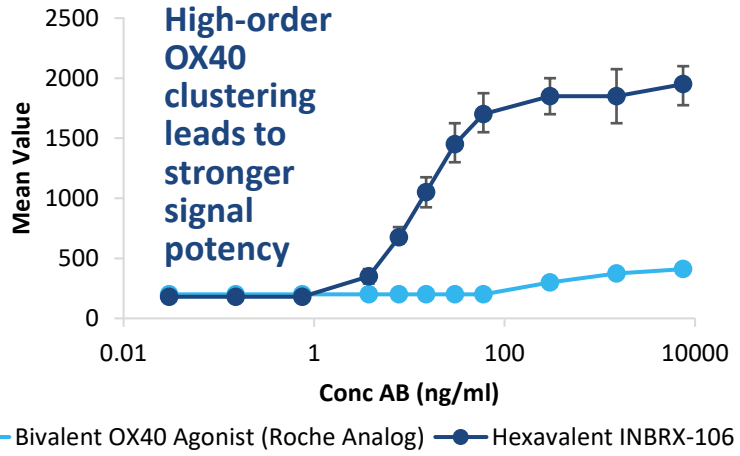
Insufficient T-cell activation — Weak clustering impairs signaling and stalls proliferation

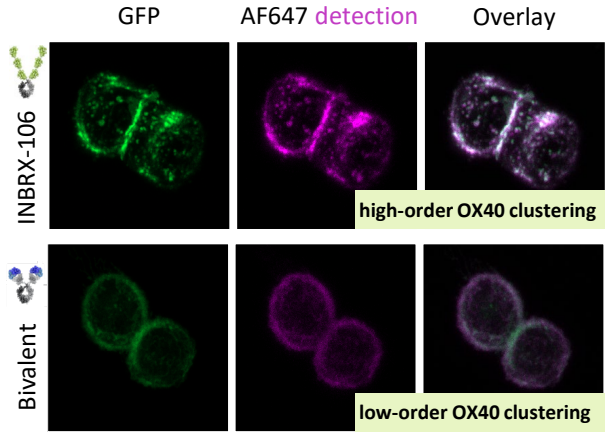
JITC Providence Publication

Advantages of Ordastobart format

Hexavalent — Simultaneously ***engages*** multiple OX40s to drive more potent clustering/signaling

Hyper-clustering — Receptor hyperclustering enables more ***efficient co-stimulation*** of OX40 *(key for OX40 low expressing cells such as CD8 + T-cells)*





**Holay et al. Journal for Immunotherapy of Cancer 2025*

Initial Study Design of HexAgon: Seamless Phase 2/3 study in 1L R/M HNSCC with PD-L1 CPS ≥20

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Head-to-head study serves as proof of concept, validating ordastobart

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



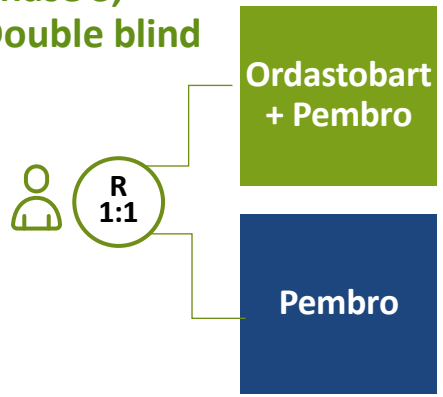
Primary Criteria:

ORR

Secondary Criteria:

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

Phase 3, Double blind



Survival Follow-up

Co-primary endpoint:

PFS and OS

Secondary endpoints:

ORR, DOR, CBR, TTCx, safety, PROs

Original Goals of HexAgon-HN Design

- ❑ - Contribution of components - Randomized add-on (Ordastobart + pembro vs pembro) cleanly isolates the benefit added by 106, aligns with regulatory expectations
- ❑ - Seamless Phase 2/3- fastest path to market

Randomization stratified by: *Disease status (locoregional advanced vs metastatic), HPV status (positive vs negative), ECOG PS (0 vs 1). In KEYNOTE-048, pembrolizumab achieved an ORR of 23.3% in the PD-L1 CPS ≥ 20 HNSCC population*

Clinicaltrials.gov (NCT06295731). Protocol version 1.0; January 31, 2024. Ordastobart 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.

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Baseline characteristics

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Baseline characteristics (randomized population)*	Ordastobart + Pembro N=33	Pembro N=35	Total N=68
Median age, y (min, max)	62.0 (35, 80)	66.0 (44, 89)	64.5 (35, 89)
Age ≥65y, n (%)	13 (39.4)	21 (60.0)	34 (50.0)
Male, n (%)	30 (90.9)	28 (80.0)	58 (85.3)
ECOG PS 1, n (%)	17 (51.5)	18 (51.4)	35 (51.5)
Distant metastatic disease, n (%)	18 (54.5)	21 (60.0)	39 (57.4)
HPV positive, n (%)	10 (30.3)	9 (25.7)	19 (27.9)
PD-L1 CPS ≥50, n (%)	24 (72.7)	23 (65.7)	47 (69.1)
Prior systemic therapy, n (%)**	21 (63.6)	19 (54.3)	40 (58.8)

Arms overall well-balanced

**Median follow-up: 8.02 months (Ordastobart + Pembrolizumab) vs. 6.31 months (Pembrolizumab).*
*** Systemic therapy received in the curative setting*

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Safety & tolerability

INBRX-106



Overall safety — HexAgon-HN vs published KEYNOTE-048 pembrolizumab-monotherapy arm**

Patients with ≥1, n (%) (safety population)*	Ordastobart + Pembro N=31	Pembro (HexAgon) N=34	KN-048 Pembro mono N=300
TEAE, any grade	31 (100)	29 (85.3)	290 (96.7)
SAE, any grade	12 (38.7)	12 (35.3)	123 (41.0)
Grade ≥3 AE, any/related	18 (58.1) / 15 (48.4)	16 (47.1) / 4 (11.8)	164 (55)
Grade 5 AE	0	2 (5.9)	25 (8)
Tx discontinuation	11 (35.5)	5 (14.7)	15 (5.0)

Most common TRAEs in HexAgon-HN ≥20%

Most common TRAEs	106+Pembro All	106+Pembro ≥G3	Pembro All	Pembro ≥G3
Rash maculo-papular	10 (32.3)	3 (9.7)	4 (11.8)	0
Rash	10 (32.3)	1 (3.2)	0	0
Fatigue	9 (29.0)	1 (3.2)	4 (11.8)	1 (2.9)
Diarrhea	9 (29.0)	3 (9.7)	2 (5.9)	0
ALT increased	8 (25.8)	2 (6.5)	3 (8.8)	1 (2.9)
AST increased	7 (22.6)	1 (3.2)	3 (8.8)	1 (2.9)
Infusion related reactions	7 (22.6)	1 (3.2)	0	0

The most frequent related adverse events are rash, fatigue, and diarrhea which were mostly grade 1 and 2

**Safety population = patients who at least received one dose of study treatment*

***Data comparison of HexAgon to KN-048 is not from head-to-head study; cross-trial comparisons are limited by differences in study designs, populations, regimens and follow-up*

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Discontinuation due to AE did not compromise antitumor activity in the combination arm

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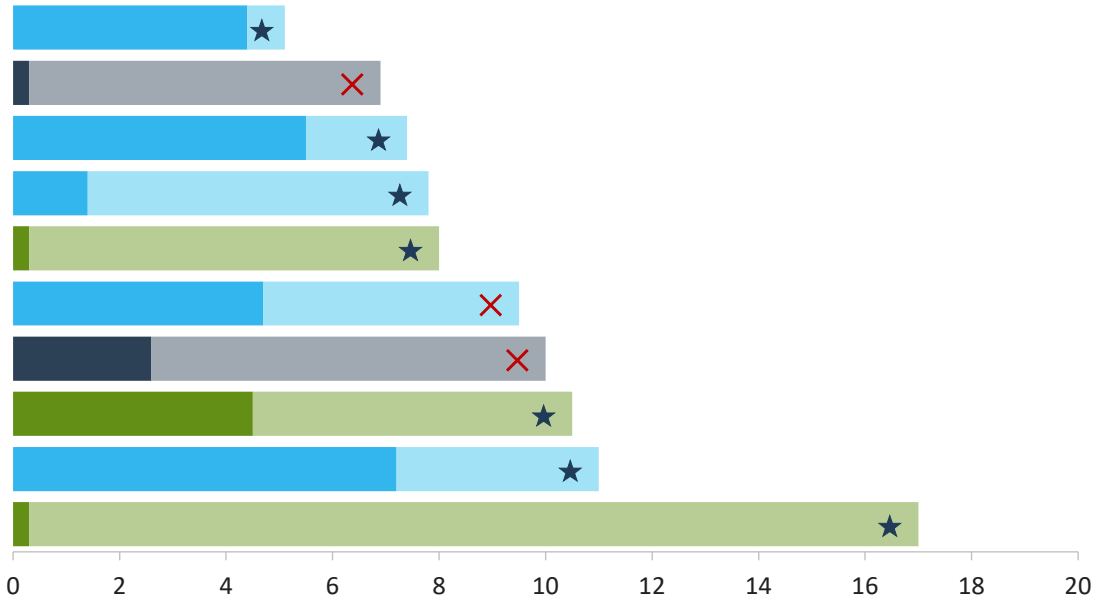
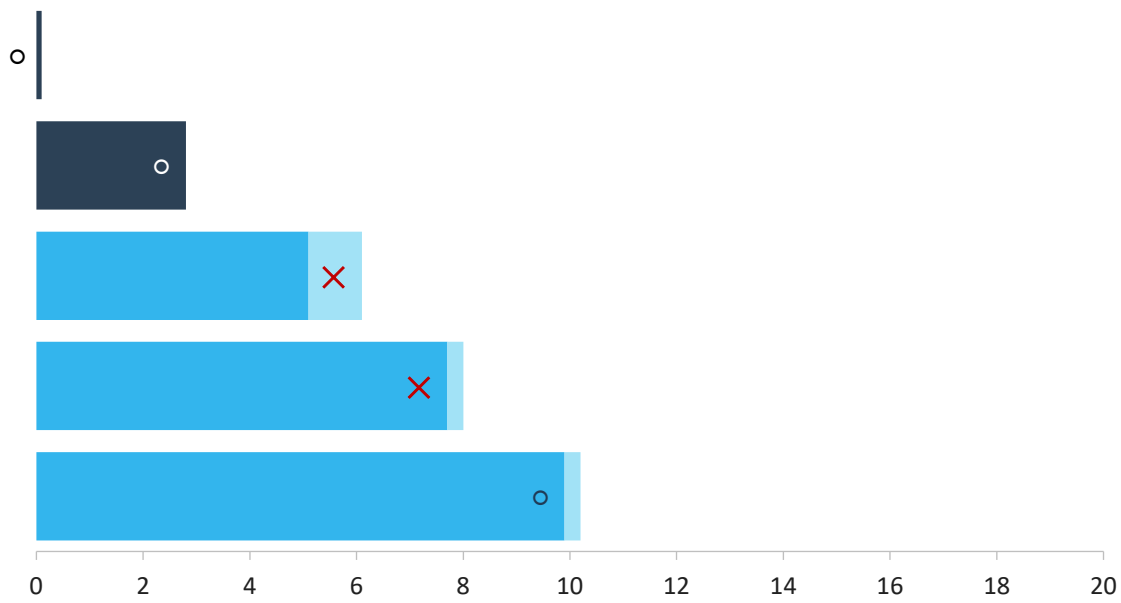
At least 9/10 (90%)
Remained progression-free after discontinuing for an AE
(Ordastobart + pembrolizumab)

0/5 (0%)
Remained progression-free after discontinuing for an AE
(Pembrolizumab alone)

Ordastobart + Pembrolizumab (n=10* discontinued for AE)



Pembrolizumab alone (n=5 discontinued for AE)



Best response: ■ CR ■ PR ■ SD ★ Ongoing, progression-free ✕ Progressive disease/died ○ Censored
Darker segment = time on treatment before AE-related discontinuation. Lighter segment = subsequent follow-up off treatment.

One additional patient discontinued due to AE in the combo arm but withdrew consent after discontinuation therefore not reflected in the graph.

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Ordastobart + pembro drives up to 15-fold change in proliferation and up to 4-fold change in activation of T-cells vs. pembro mono

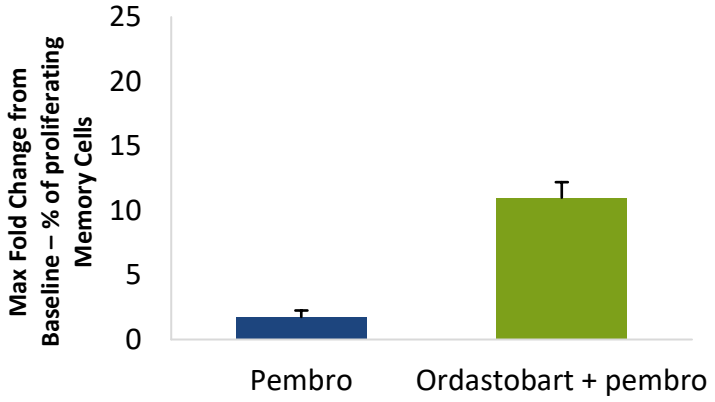
INBRX-106

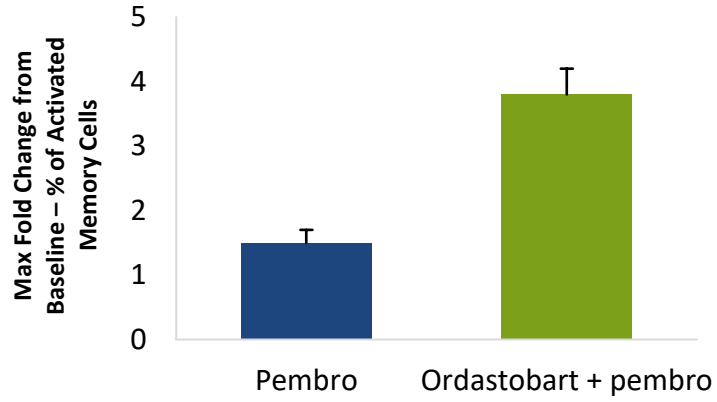
HexAgon Phase 2 – 1L HNSCC

Proliferation

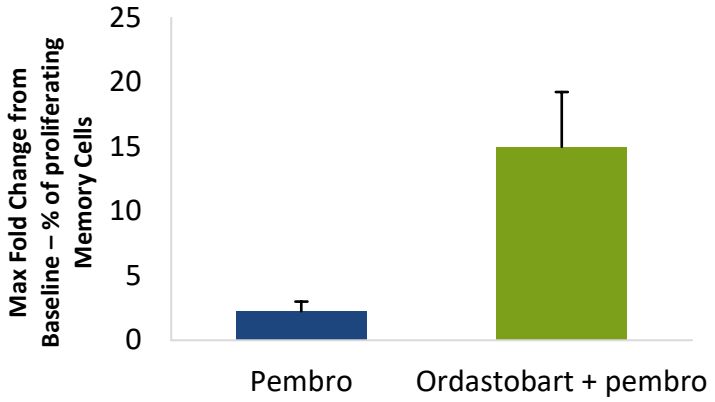
Activation

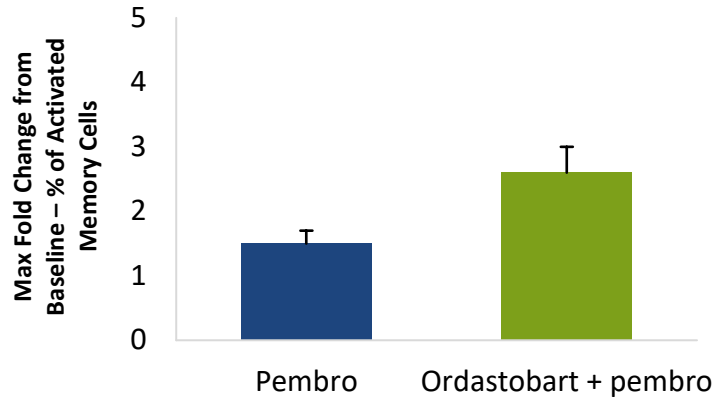
CD4+ T cells





CD8+ T cells





- Ordastobart is the key driver of T-cell activity in the combination group
- Ordastobart drives up to 15-fold change increase in proliferation and up to 4-fold change increase in activation of T-cells
- Clear validation of Ordastobart as a potent T-cell co-activator

Data cutoff date: April 2, 2026. RP2D (recommended phase 2 dose) for combination is 0.1 mpk of INBRX-106. Data is representative of Hexagon U.S. cohorts only (9 Pembro and 13 Combo patients). Data represented as Mean ±SEM.

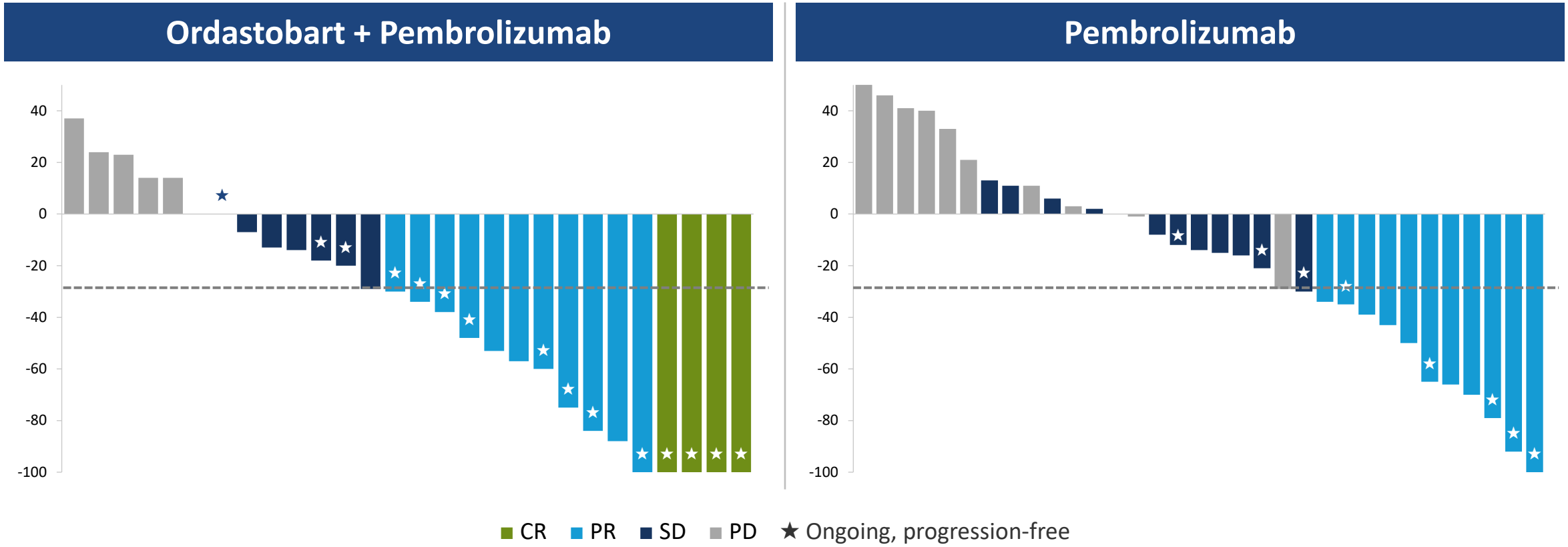
Primary endpoint: addition of Ordastobart to pembro improves cORR

INBRX-106

48.3%
cORR — Ordastobart + Pembro (n=29*)
95% CI 29.4–67.5

26.5%
cORR — Pembro (n=34*)
95% CI 12.9–44.4

Δ +21.8 points
Difference in cORR
favoring Ordastobart + Pembro



Best % change in target lesion sum of diameters from baseline, RECIST v1.1. cORR = confirmed CR/PR on 2 consecutive occasions ≥4 weeks apart. Data from ongoing DB - CCOD 19 Aug 2026
** Evaluable population = Patients who have at least one post-baseline scan or have died or progressed*

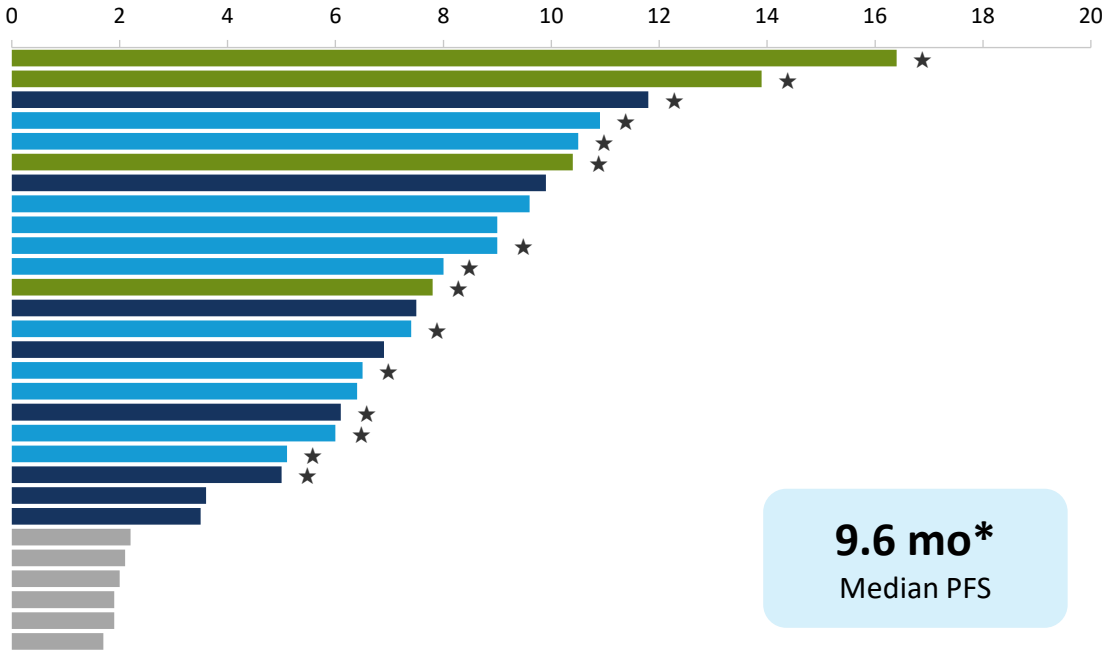
Ordastobart improves overall durability & PFS across all patients

INBRX-106

72.4%
PFS at 6 months — Ordastobart + Pembro

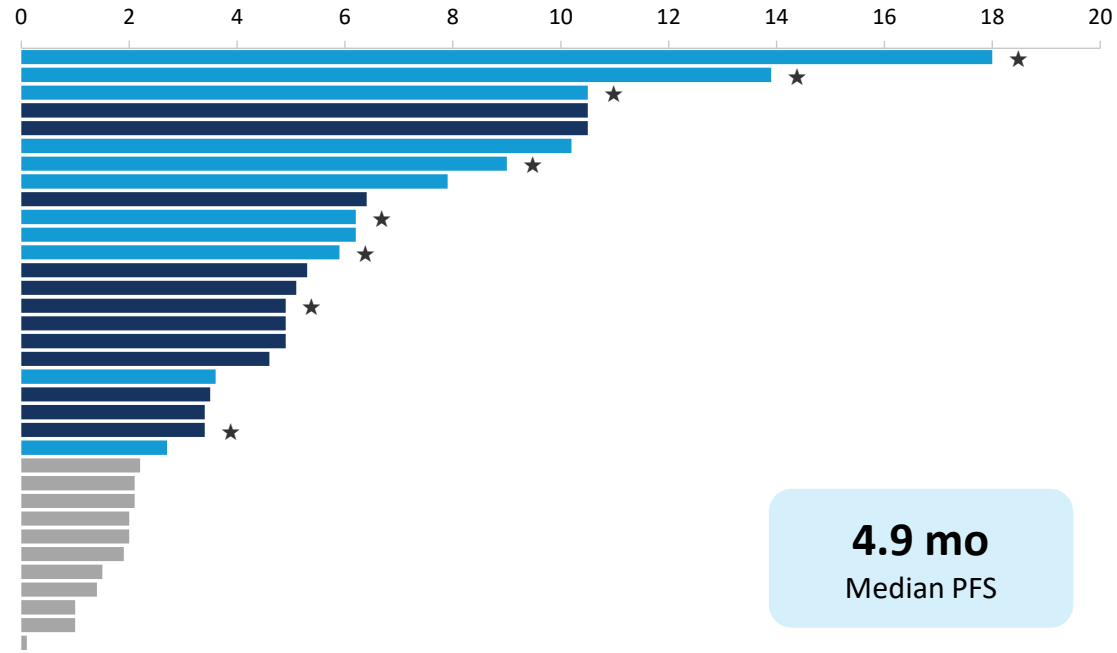
42.8%
PFS at 6 months — Pembrolizumab

Ordastobart + Pembrolizumab



9.6 mo*
Median PFS

Pembrolizumab



4.9 mo
Median PFS

CR PR SD PD ★ Ongoing, progression-free

**Median PFS still maturing; CCOD 19 Aug 2026*
Bars show months of PFS follow-up, ranked longest to shortest, colored by best overall response. Data from ongoing DB - CCOD 19 Aug 2026.
Note: Survival estimates are calculated using the Kaplan-Meier method.

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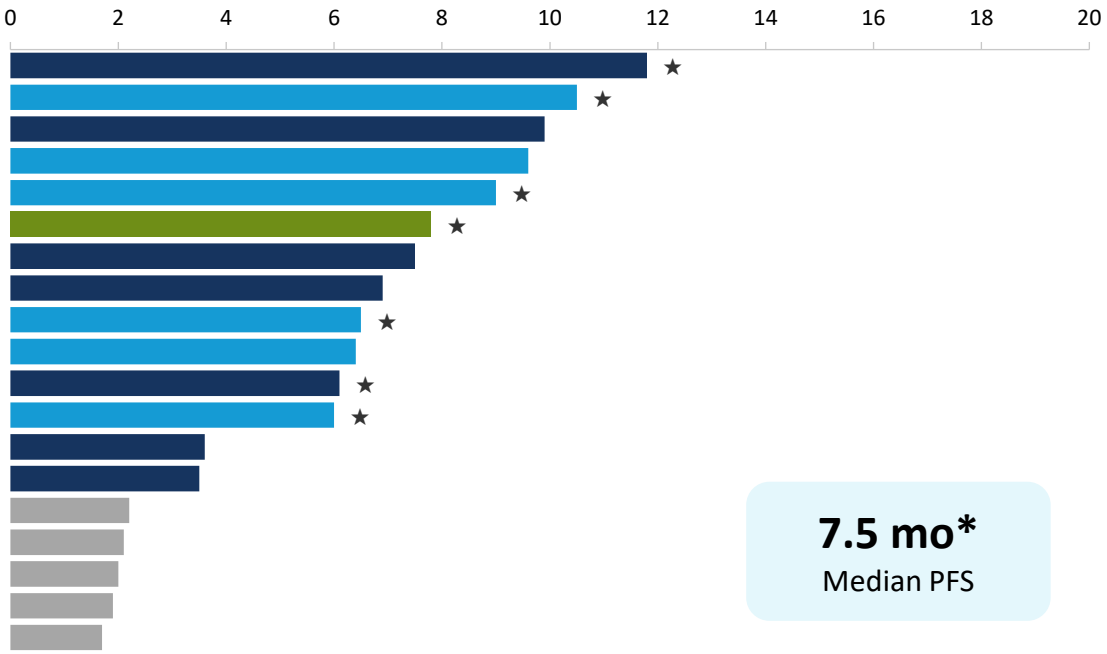
Improved response & duration observed in HPV-negative patients

INBRX-106

63.2% PFS at 6 months, HPV-negative
31.6% cORR, HPV-negative (n=19)

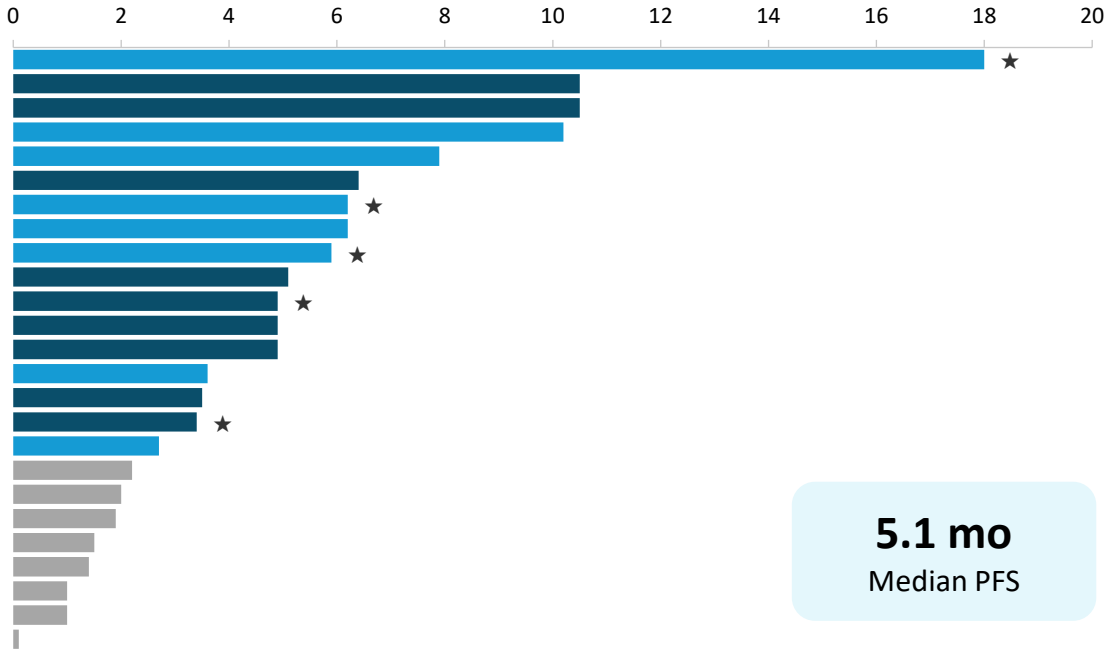
46.8% PFS at 6 months, HPV-negative
24.0% cORR, HPV-negative (n=25)

Ordastobart + Pembrolizumab, HPV-negative



7.5 mo*
Median PFS

Pembrolizumab, HPV-negative



5.1 mo
Median PFS

CR PR SD PD ★ Ongoing, progression-free

**Median PFS still maturing; CCOD 19 Aug 2026.*
Note: Survival estimates are calculated using the Kaplan-Meier method.

Increased benefit was observed in HPV+ patients

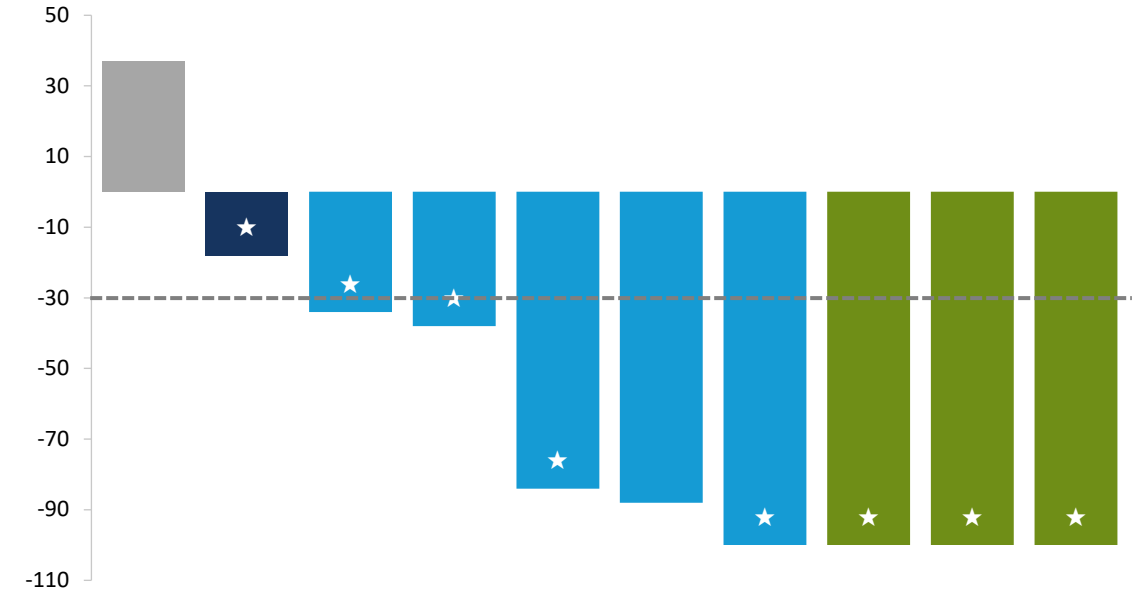
INBRX-106

80.0%
cORR, HPV+
Ordastobart + pembro (n=10)
95% CI 44.4–97.5

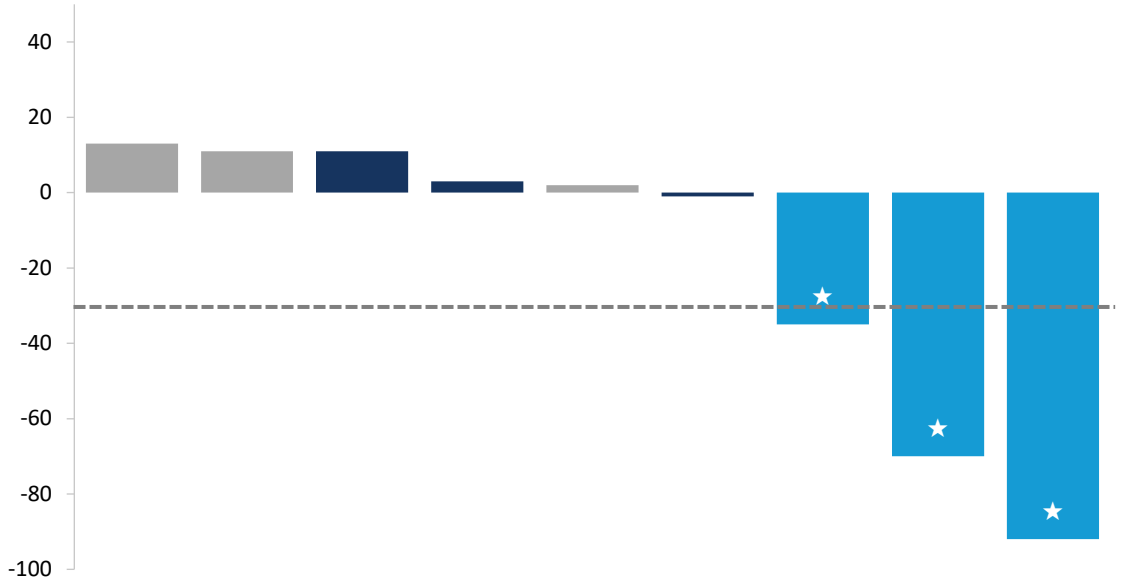
33.3%
cORR, HPV+
Pembrolizumab (n=9)
95% CI 7.5–70.1

Δ +46.7 points
Difference in cORR, HPV+
Ordastobart +pembro vs. pembro mono
95% CI –2.7, 80.3

Ordastobart + Pembrolizumab, HPV+



Pembrolizumab, HPV+



CR PR SD PD ★ Ongoing, progression-free

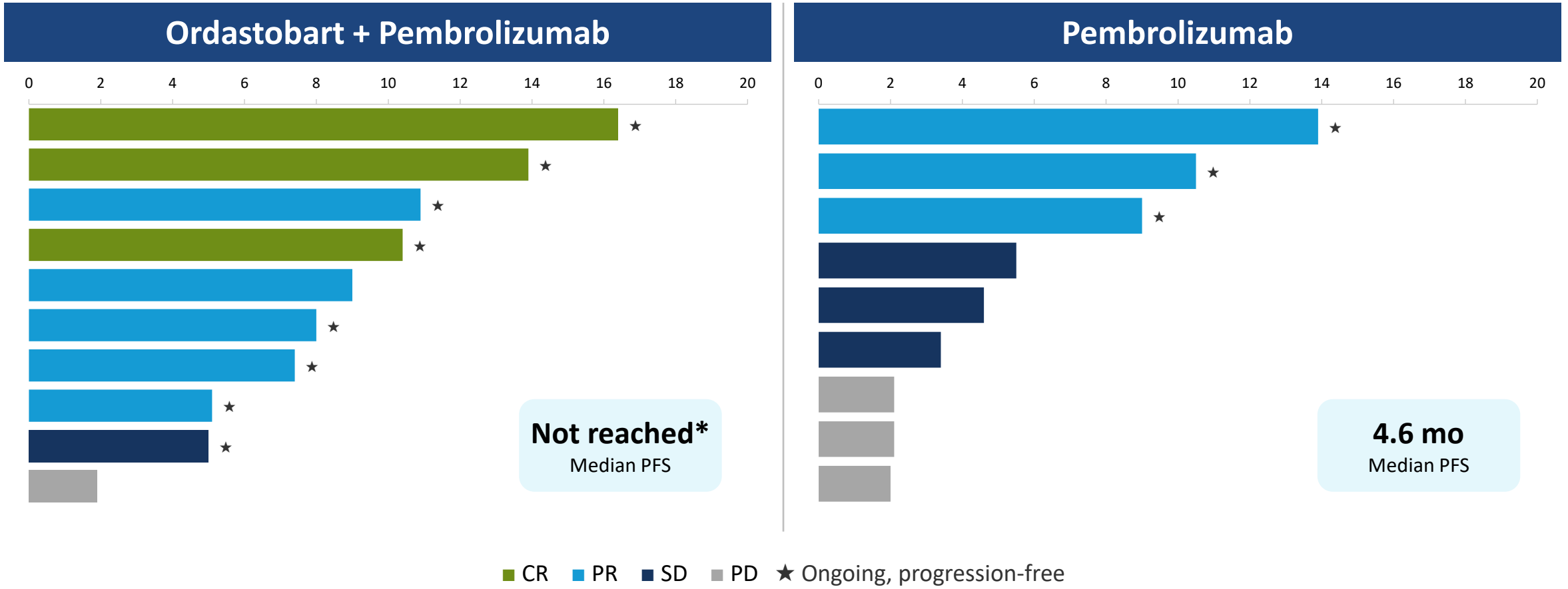
Best % change in target lesion sum of diameters from baseline

Enhanced durability & PFS observed in HPV+ patients

INBRX-106

90%
PFS at 6 months, HPV+
INBRX-106 + Pembro

33%
PFS at 6 months, HPV+
Pembrolizumab



**Median PFS still maturing; CCOD 19 Aug 2026.*
Bars show months of PFS follow-up, ranked longest to shortest, colored by best overall response. Data from ongoing DB - CCOD 19 Aug 2026.
Note: Survival estimates are calculated using the Kaplan-Meier method.

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The HPV+ signal is what the Ordastobart mechanism predicts

INBRX-106



Compelling Clinical Activity

80% vs 33%

cORR, HPV+
(Ordastobart +Pembro
vs Pembro)

90% vs 33%

PFS6, HPV+
(Ordastobart +Pembro
vs Pembro)

Up to a 15-fold increase in peripheral $CD8^+/CD4^+$ T-cell proliferation and up to a 4-fold increase in activation (HexAgon-HN, interim Ph2) over ~2-fold pembrolizumab alone.

- HPV-driven tumors express viral E6/E7 neoantigens; these are foreign, high-quality targets that OX40 co-stimulation (Ordastobart's MoA) amplifies.
- The enhanced results observed in HPV+ HNSCC are consistent with the expected underlying biology.



Sound, mechanistic rationale

Foreign viral antigen (E6/E7) driving a strong Signal 1, activated by OX40 co-stimulation on antigen-experienced T-cells



High unmet need

Attractive commercial opportunity: Potentially first dedicated HPV+ approval, differentiated from EGFR bispecifics (active mainly in HPV negative)



Potential for fast revenue realization

Profound response-rate difference supports a potentially accelerated path to market

Opportunity to own HPV+ market

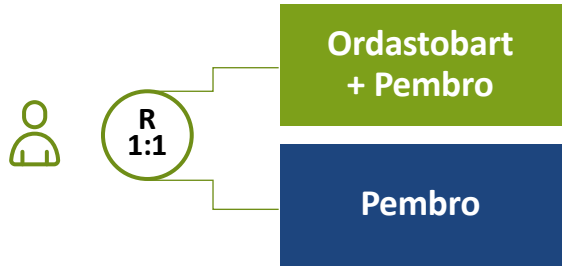
Expansion of Phase 2 HexAgon-HN with potential for an accelerated path to market in HPV+

INBRX-106

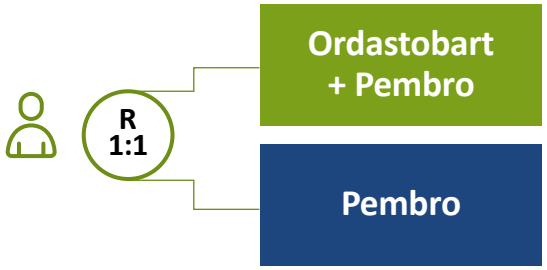
The new study amendment adds an additional 50 HPV+ patients to the existing HexAgon design

Phase 2 (additional 50 HPV+)
Open label

Key inclusion criteria:
R/M HNSCC
HPV+ OPSCC
PD-L1 CPS≥1



Phase 3 (n=TBD)
Double blind, seamless



Opens the door to possibilities for accelerated development

Potential for breakthrough therapy designation

Potential, based on preliminary signal magnitude in a serious, unmet-need indication

Potential for accelerated approval

Potential accelerated path by end of 2028/ early 2029 based on ORR as a surrogate likely to predict long-term clinical benefit (PFS & OS), and established precedent with petosemtamab and ficerafusp alfa

Phase 3: confirmatory

Already part of the seamless design: serves as the confirmatory study for full approval

Rationale for expanding HPV+ enrollment to PD-L1 CPS≥1

All Phase 2a patients both HPV+ and HPV-negative were enrolled at PD-L1 CPS≥20

- PD-L1 expression was therefore held constant across the two subgroups and despite this the increase in response rates with the combination was much greater in the HPV+
- If CPS>20 was the driver you would see similar increase in ORR independently of HPV status
- If Ordastobart's HPV+ benefit is driven by antigen availability rather than PD-L1 level, lowering to CPS>1 should not impact the ability of OX-40 to amplify the tumor response

	Pembrolizumab alone	+ Ordastobart
HPV+ cORR	33.3% (3/9)	80.0% (8/10)
HPV-negative cORR	24.0% (6/25)	31.6% (6/19)

Working hypothesis

This data reflects the distinct immunogenic biology of HPV-driven tumors, (viral E6/E7 oncoproteins provide a foreign), recognizable antigen source for OX40 to amplify rather than a PD-1-pathway-sensitivity effect.

Validation

The Phase 2b expansion: HPV+ OPSCC patients across CPS≥1, stratified by CPS (1–19 vs ≥20) is designed to test this directly and prospectively.

Mandal R, et al. JCI Insight. 2016;1(17):e89829. Burtness B, et al. Lancet. 2019;394(10212):1915-1928.

Set to pursue a breakthrough pathway in 1L R/M HPV+ CPS ≥1 HNSCC

~$4B+ U.S. market opportunity including stage I-III setting

INBRX-106

Rapidly Growing Market



US Annual Growth in HPV+

~30,000
2029 US Incidence (De Novo Stage I-IV + Recurrent) HPV+ HNSCC Patients in the US

US Epidemiology Estimates

~8,000 Stage I-III Surgical	~16,000 Stage I-III Non-Surgical	~6,000 1L R/M

Valuation Drivers

High morbidities in current therapies	Clinicians actively seeking de-escalation therapy in HPV+. Significant radiation- and surgery-related morbidities — e.g., impaired speech, feeding tube
High share; higher systemic usage	Significant unmet need for a therapy specifically studied in the HPV+ population; where current EGFR bi-specifics do not perform well
Premium pricing	Ground-breaking results and limited patient population would provide opportunity for premium pricing. Premium priced therapies currently on guidelines
Long duration	High response rate, and deep and durable responses on Ordastobart point to longer duration on therapy

US Sales Potential

~$1B	~$2B	~$1B

Source: SEER Explorer, CDC, Clarivate Disease Landscape Report

Ordastobart: multiple attractive expansion opportunities

INBRX-106



Increased opportunity for OX40 agonism due to increased ongoing antigen-driven T-Cell stimulation

	Highly Immunogenic Tumors	Virally Antigen-Driven
	• NSCLC • Melanoma • Renal Cell Carcinoma • TNBC • Bladder • MSI-H / dMMR • TMB-High	• HPV+ H&N • HPV+ beyond H&N (e.g., Anus/ Rectum, Cervix, Vagina, Vulva, Penis) • Non-HPV, commonly reported virally-linked cancers: EBV, HBV, HCB, HTL1-1, HHV-8
WW Market Opportunity:	**$50 B+[1]**	**$15 B+[2]**
Ongoing studies & development plan:	Ongoing clinical trial in peri-operative NSCLC and IST in peri-operative TNBC serves as fast proof of concept for broad expandability to other immunogenic tumors	Expand Phase 2 portion of HexAgon-HN trial to increase HPV+ patients for potential accelerated approval

Highest impact expected in early stage disease

Source: 1. Evaluate estimates for PD-1s projected to 2031 based on 2025-2027 growth rates (market size independent of biosimilar entry). 2. Virally antigen estimated based at 3x HPV based on topline epidemiology from CDC or IARC (de Martel) projected WW. 3. Company estimate, full impact of V940 data release 8/19/26 not yet propagated through reports.

INHIBRX

Highly immunogenic tumors, peri-operative NSCLC expansion: pCR is a fast, validated readout of long-term benefit

INBRX-106

- Pathologic complete response (pCR) at surgery:
  - Available in months, not years
  - Highly correlated with improved survival and can generate a second, independent proof-of-activity signal well ahead of any survival endpoint
- pCR in peri-operative NSCLC opens a multibillion-dollar market opportunity and will serve as proof of concept for expandability to other highly immunogenic tumors, broadening the platform thesis beyond HNSCC

KEYNOTE-671

0.58

Event-free survival HR
(95% CI 0.46–0.72)[1]

18.1%

Pathologic complete response
Pembro+chemo

Neoadjuvant study – Ongoing Ph1/2 study expected to read-out by mid-2027

Participants (N~40)

Stage II–IIIB NSCLC,
ECOG 0–1,
surgical candidates

Neoadjuvant (4 cycles)

Platin doublet
+ pembro
+ Ordastobart

Surgery

Adjuvant (13 cycles)

Pembro
+ Ordastobart

Primary endpoints

+ pCR
+ Safety

1. *Wakelee et al., KEYNOTE-671, N Engl J Med 2023*

Ordastobart conclusion

Ordastobart is the first clinically active OX40 agonist and T-cell costimulatory therapy

Combination with pembro demonstrates superiority to pembro mono in 1LR/M HNSCC

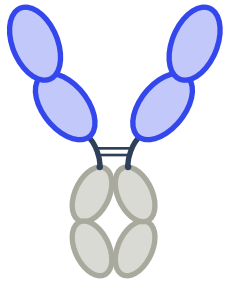
- ORR: 48.3% vs 26.5%
- Depth of Response
- CR Rate: 13.8% vs 0%
- PFS: 9.6 months vs 4.9 months
- PFS6 Improvement: (72.4% vs. 42.8%)
- These data are supported by superior t-cell proliferation (up to 15-fold increase) and activation (up to 4-fold increase) vs pembrolizumab alone

Potentially practice-changing efficacy profile in HPV+ patients

- ORR: 80% vs 33%
- CR Rate: 30% vs 0%
- PFS Data: NR vs 4.6 months
- PFS6 Rate: 90% vs 33%
- Expansion of Phase 2 to include 50 additional HPV+ patients may serve as potential path to accelerated approval by end of ‘28/early ‘29. HPV+ HNSCC is an open $4B+ market

Large expansion opportunity in highly immunogenic tumors

- Bladder, NSCLC, Melanoma, MSI High/TMB High, TNBC present $50B+ commercial opportunity
- Ongoing neoadjuvant lung cancer study serves as proof of concept for broad expandability with pembrolizumab across highly immunogenic tumors



Ozekibart (INBRX-109)

Selectively inducing apoptosis in tumor cells through tetravalent DR5 agonism

INHIBRX

Ozekibart Executive Summary

Ozekibart (INBRX-109)



Ozekibart is a best-in-class DR5 agonist

Unprecedented single agent activity in chondrosarcoma registrational trial

- 52% reduction in progression or death: HR 0.46; P < 0.0001
- Median PFS more than doubled placebo: 5.55 months vs 2.69 months
- Improved disease control rate: 54% vs 27.5%
- Benefit was consistent across all pre-specified subgroups

Potential for FDA approval in April 2027 with commercial sales beginning in 1H 2027

- BLA submission accepted with anticipated PDUFA date in April 2027
- Potential to be the first treatment approved in chondrosarcoma
- Commercialization as early as 1H 2027

Encouraging Ewing sarcoma data

- Updated results from Ewing cohort of 51 evaluable patients showed an ORR of 62.7%
- DCR rate of 86.3% and a median PFS of 6 months
- Intend to rapidly advance our plans in order to allow patients access as soon as possible

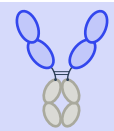
Potential for expansion in colorectal cancer and other solid tumor types

- Initial CRC data (predominantly 4L): 20% ORR
- 2L (FOLFIRI + Avastin) and 3L/4L (Lonsurf + Avastin) combo cohort data expected Q1 2027
- Potential to expand into 1L Ph3 (ozekibart + FOLFIRI + bev)
- MoA supports potential expansion into other tumor types and combinations, including ADCs

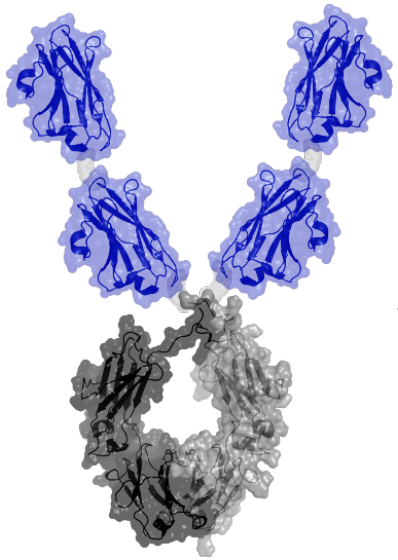
INHIBRX

Ozekibart: A next generation DR5 agonist with an optimized balance of efficacy and safety

Ozekibart (INBRX-109)

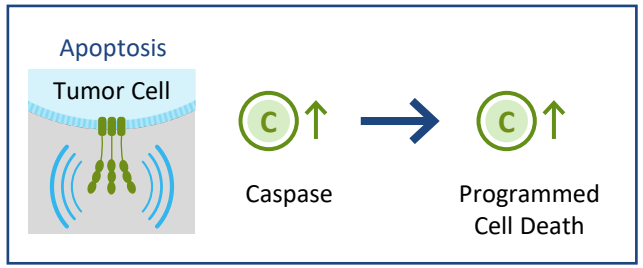


Death Receptor 5 (DR5 / TRAIL-R2) is highly expressed on tumorigenic, transformed or damaged cells but not normal cells, making DR5 a promising therapeutic target in oncology[1-4]



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

Ozekibart characteristics:

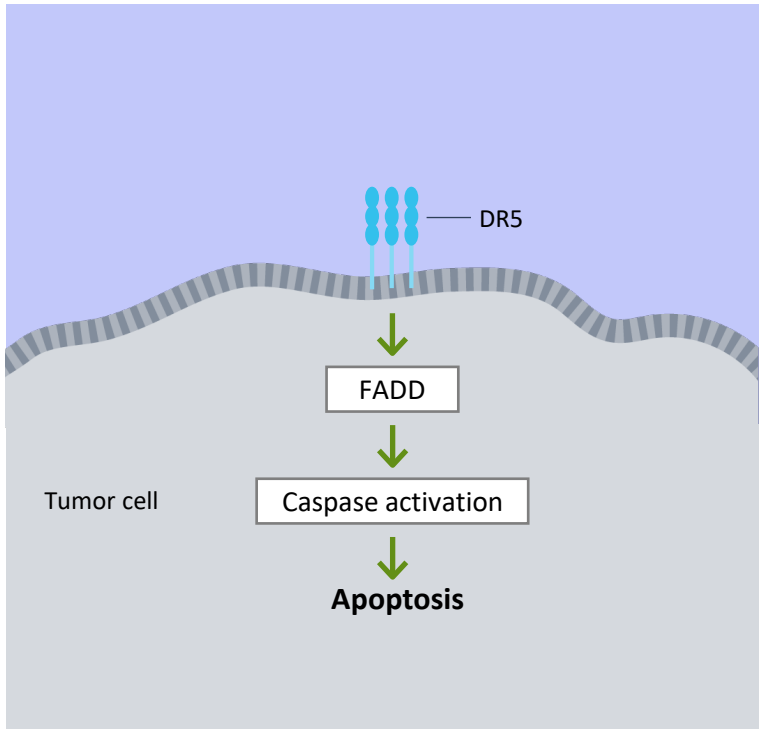
Tetravalent	Empirically designed to simultaneously engage four DR5 molecules
Immunogenic Epitopes removed	Prevents unwanted higher order clustering via anti-drug antibodies
Effector Disabled Fc	Prevents higher order clustering and allows for antibody-like PK
Smaller Size	sdAb backbone limits molecule 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

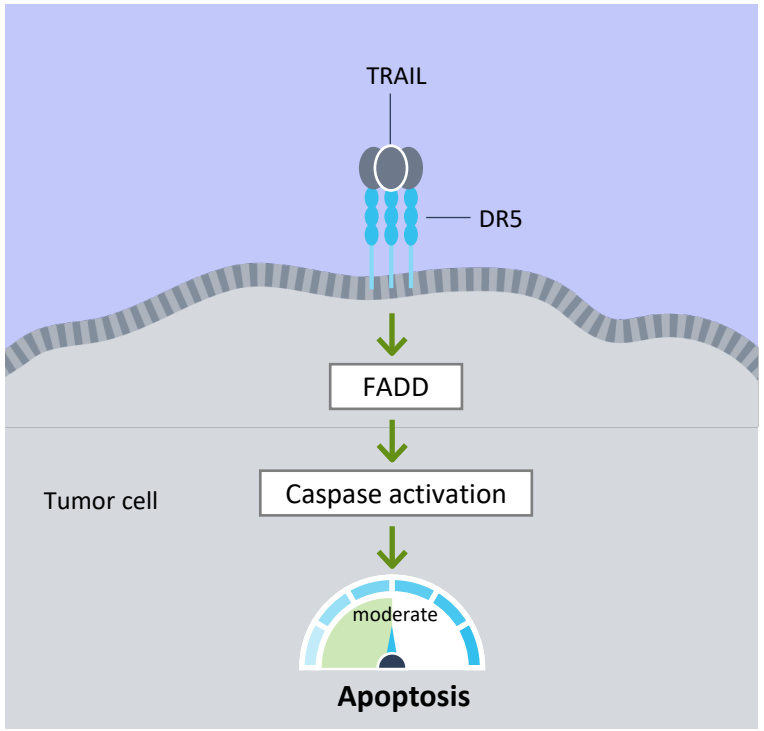
INHIBRX

Ozekibart is a potent inducer of extrinsic cell death via the DR5 pathway

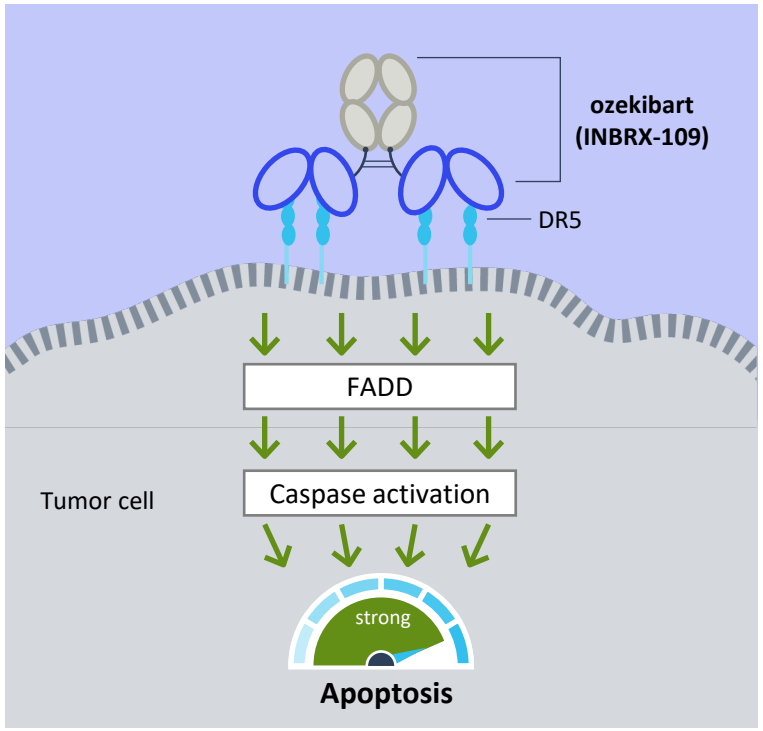
Ozekibart (INBRX-109)



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, 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.

INHIBRX

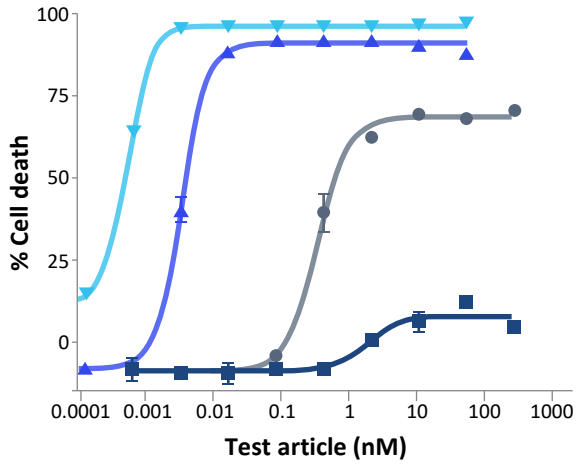
Ozekibart is precision-engineered for optimal potency and safety

Ozekibart (INBRX-109)

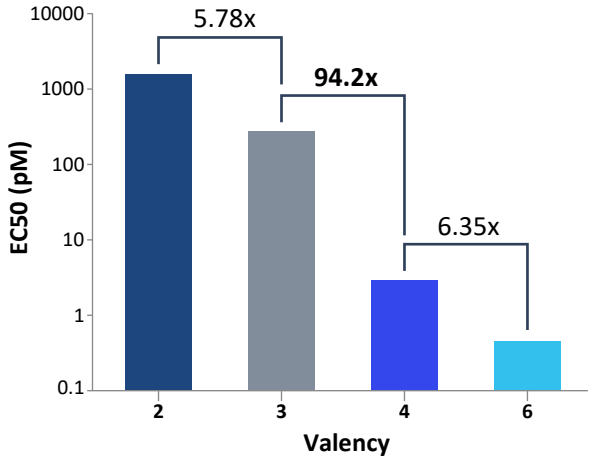
Valency drives both DR5-induced tumor cell death and hepatocyte destruction

Impact of valency on DR5-mediated cell death

Impact of valency on cell death:



Impact of valency on cell death:



anti DR5 mAb
bivalent

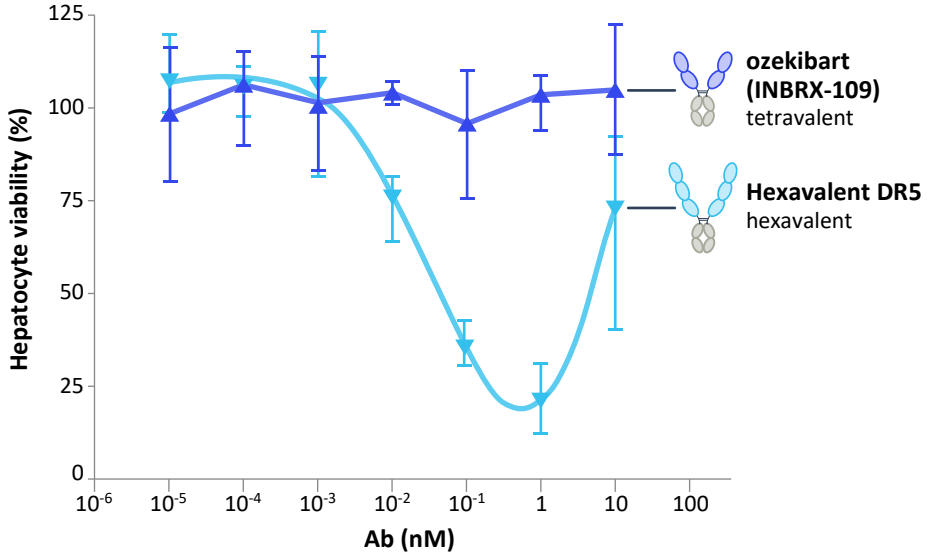
TRAIL
trivalent

ozekibart
tetravalent

Hexavalent DR5
hexavalent

Impact of valency on hepatotoxicity

InSphero 3D inSight™ human liver microtissue model:



Ozekibart chondrosarcoma registrational trial results

Ozekibart (INBRX-109)

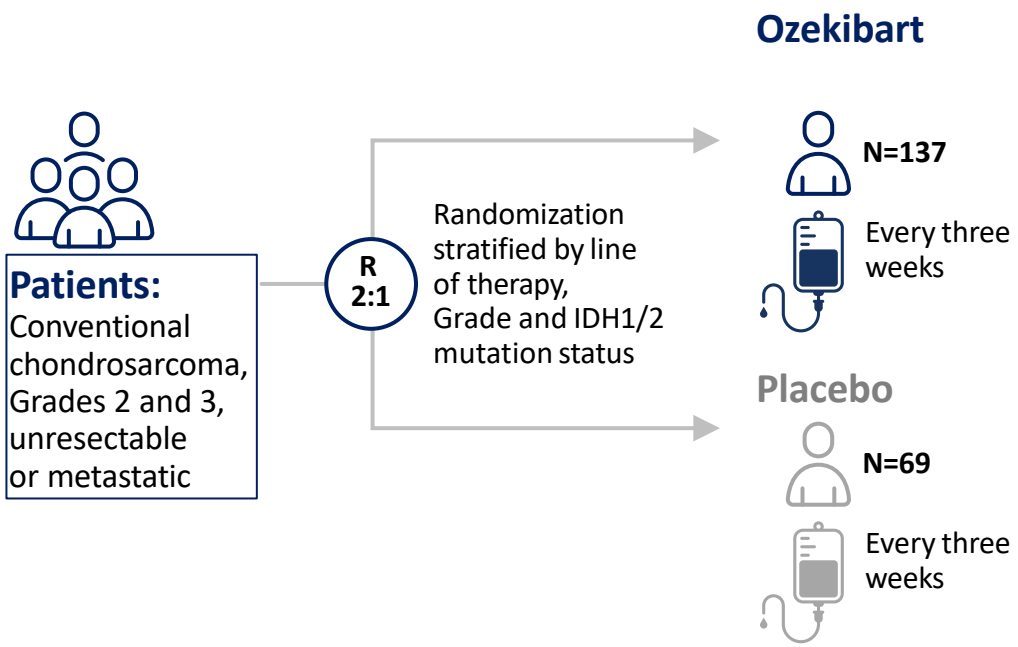
Unprecedented data in chondrosarcoma

52% reduction in progression or death
HR 0.479;
P < 0.0001

Median PFS more than doubled
5.52 months vs
2.66 months

Improved disease control rate
54% vs 27.5%

Regulatory momentum
Orphan designations (FDA & EMA) and Fast track (FDA)



Primary endpoint met:

- HR= 0.479; (95% CI: 0.33, 0.68); P<0.0001 (52% reduction in the risk of disease progression or death)
- More than doubling median PFS to 5.52 months compared to 2.66 months for placebo
- Ozekibart's benefit was consistent across all pre-specified subgroups

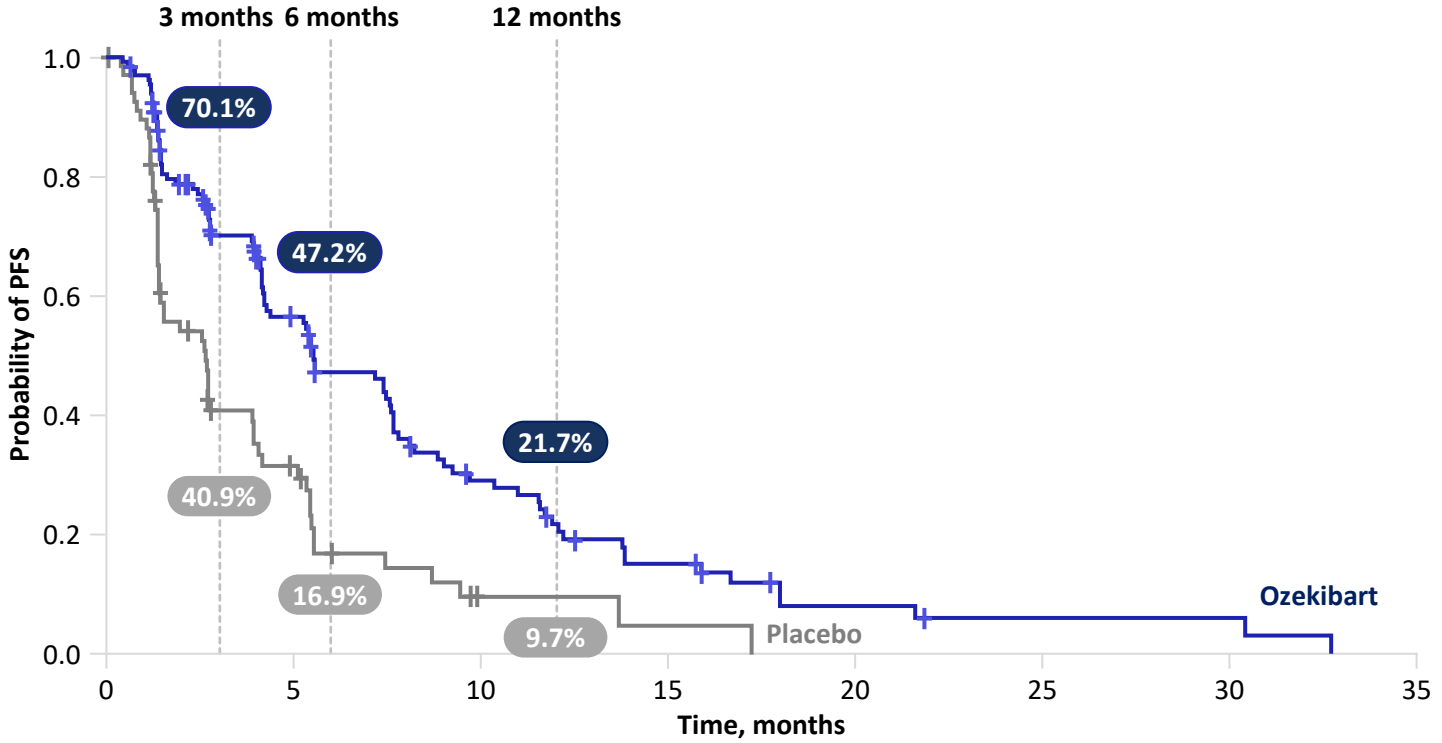
Secondary endpoint:

- Disease control rate (54% vs 27.5%)

ChonDRAgon Chondrosarcoma Study with Death Receptor Agonist | INHIBRX

Primary endpoint: progression-free survival by CIRR

Ozekibart (INBRX-109)



No. at risk:								
Ozekibart	137	56	24	11	4	2	2	0
Placebo	69	16	2	1	0	0	0	0

	Ozekibart N=137	Placebo N=69
Primary Efficacy Endpoint		
Events / Censored, n (%)	94 (68.6) / 43 (31.4)	55 (79.7) / 14 (20.3)
mPFS, months	**5.52**	2.66
Stratified HR (95.02% CI)	**0.479** (0.335–0.684)	
Log-rank P value	**<0.0001**	
Disease Control Rate by CIRR		
DCR, n (%)[d]	**74 (54.0)**	19 (27.5)
95% CI[e]	45.3–62.6	17.5–39.6
Percentage point difference (95% CI)	25.9 (11.7–38.3) $P < .001$[f]	

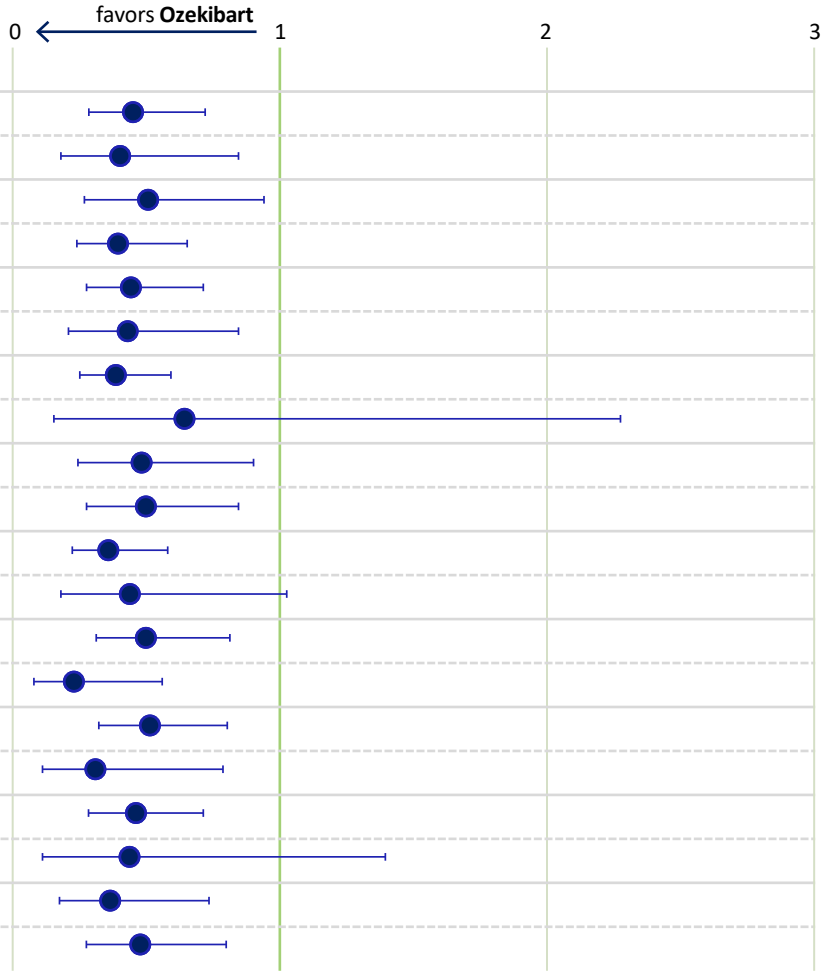
Ozekibart significantly prolonged mPFS vs placebo and led to a 52% reduction in the risk of disease progression or death

[a] Per RECIST, v1.1. [b] CI was calculated using Clopper-Pearson exact method, which does not take stratification factors into consideration. [c] A Cochran-Mantel-Haenszel test stratified by randomization stratification factors to adjust for possible confounding effects of the stratification factors was used to calculate the P value. [d] The BOR was NE in 7 patients who received ozekibart and 2 who received placebo due to no available postbaseline scans. [e] The study inclusion criteria of measurable disease according to RECIST (and progression within 6 months) was evaluated using the investigator assessment of tumor images only; confirmation of the investigator's assessment by the central reader was not required. Due to differences in lesion selection and/or lesion measurements upon central review, 4 patients did not have measurable target lesions per central reader. These patients were followed for disease progression based on non-target disease; thus, the target lesion response was considered NE by the central reader, and only the non-CR/non-PD designation could be selected for SD in these 4 patients.
Data cutoff date: September 30, 2025. Crosses indicate censored patients. Data from the double-blind period of the study are presented.
CIRR, central independent radiology review; HR, hazard ratio; mPFS, median PFS; PFS, progression-free survival.

ChonDRAgon Chondrosarcoma Study with Death Receptor Agonist | INHIBRX

Progression-free survival by CIRR: subgroup analyses

INBRX-109



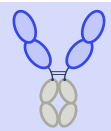
		Ozekibart		Placebo		
		Events, n/N[a]	mPFS, mo	Events, n/N[a]	mPFS, mo	HR (95% CI)
***IDH* mutation status**	Wild type	65/93	5.5	38/47	2.7	0.493 (0.324-0.750)
	Mutant	29/44	5.5	17/22	2.3	0.442 (0.224-0.871)
Prior treatment	Yes	38/58	5.3	21/28	2.7	0.546 (0.307-0.971)
	No	56/79	7.4	34/41	2.7	0.441 (0.280-0.693)
Histologic grade at entry	2	65/96	7.2	36/48	2.7	0.485 (0.316-0.747)
	3	29/41	5.3	19/21	1.4	0.464 (0.246-0.875)
Disease status at entry	Metastatic	87/122	5.5	47/56	1.5	0.424 (0.288-0.624)
	Nonmetastatic unresectable	7/15	5.5	8/13	5.4	0.675 (0.199-2.286)
Region	US	42/65	7.6	24/32	2.0	0.517 (0.288-0.928)
	Outside of US	52/72	4.3	31/37	2.7	0.524 (0.316-0.870)
Age	<65 years	68/104	7.4	40/52	2.6	0.398 (0.257-0.615)
	≥65 years	26/33	5.3	15/17	2.7	0.476 (0.217-1.047)
Sex	Male	69/96	5.4	37/42	2.7	0.545 (0.353-0.840)
	Female	25/41	7.6	18/27	2.6	0.270 (0.123-0.592)
Race	White	75/114	5.5	38/47	2.7	0.546 (0.358-0.835)
	Other	19/23	7.4	17/22	2.7	0.348 (0.148-0.818)
BMI	<30 kg/m^2	76/112	5.6	43/53	2.6	0.499 (0.333-0.747)
	≥30 kg/m^2	18/25	4.3	12/16	2.7	0.472 (0.156-1.423)
ECOG PS	0	33/51	7.6	23/29	2.7	0.404 (0.215-0.758)
	1	60/85	5.5	32/39	2.7	0.515 (0.319-0.830)

PFS benefit was consistent across key prespecified subgroups, including *IDH* mutation status & prior therapy

Data cutoff date: September 30, 2025. [a] N values represent the number of patients with available data for each parameter.
BMI, body mass index; CIRR, central independent radiology review; ECOG PS, Eastern Cooperative Oncology Group performance status; HR, hazard ratio; CI, confidence interval; IDH, isocitrate dehydrogenase; mPFS, median progression-free survival.

ChonDRAgon Chondrosarcoma Study with Death Receptor Agonist | INHIBRX

Ozekibart (INBRX-109)



Overall safety data

	Double-blind period	
	Ozekibart N=136	**Placebo** N=67
Patients with ≥1 AE (including unrelated AEs), n (%)	**129 (94.9)**	**62 (92.5)**
Grade ≥3	53 (39.0)	17 (25.4)
SAE	38 (27.9)	12 (17.9)
HAE	21 (15.4)	9 (13.4)
Resulting in death	5 (3.7)	1 (1.5)
Patients with ≥1 treatment-related AE, n (%)	**77 (56.6)**	**32 (47.8)**
Grade ≥3	14 (10.3)	1 (1.5)
SAE	8 (5.9)	0
HAE	16 (11.8)	3 (4.5)
Leading to interruption of study drug	8 (5.9)	2 (3.0)
Leading to discontinuation of study drug	4 (2.9)	0
Resulting in death	1 (0.7)	0

Data cutoff date: September 30, 2025.

AE, adverse event; HAE, hepatic adverse event; SAE, serious adverse event. HAEs included alanine aminotransferase increased, aspartate aminotransferase increased, bile duct stone, bilirubin conjugated increased, blood alkaline phosphatase increased, blood bilirubin increased, feces discolored, gamma-glutamyl transferase increased, hepatic failure, hyperbilirubinemia, hypertransaminasemia, and liver disorder.

ChonDRAgon Chondrosarcoma Study with Death Receptor Agonist | INHIBRX

Ongoing phase 1/2 trial in Ewing sarcoma in combination with IRI/TMZ

Ozekibart (INBRX-109)

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

EWS 2-3L with IRI/TMZ

N=60

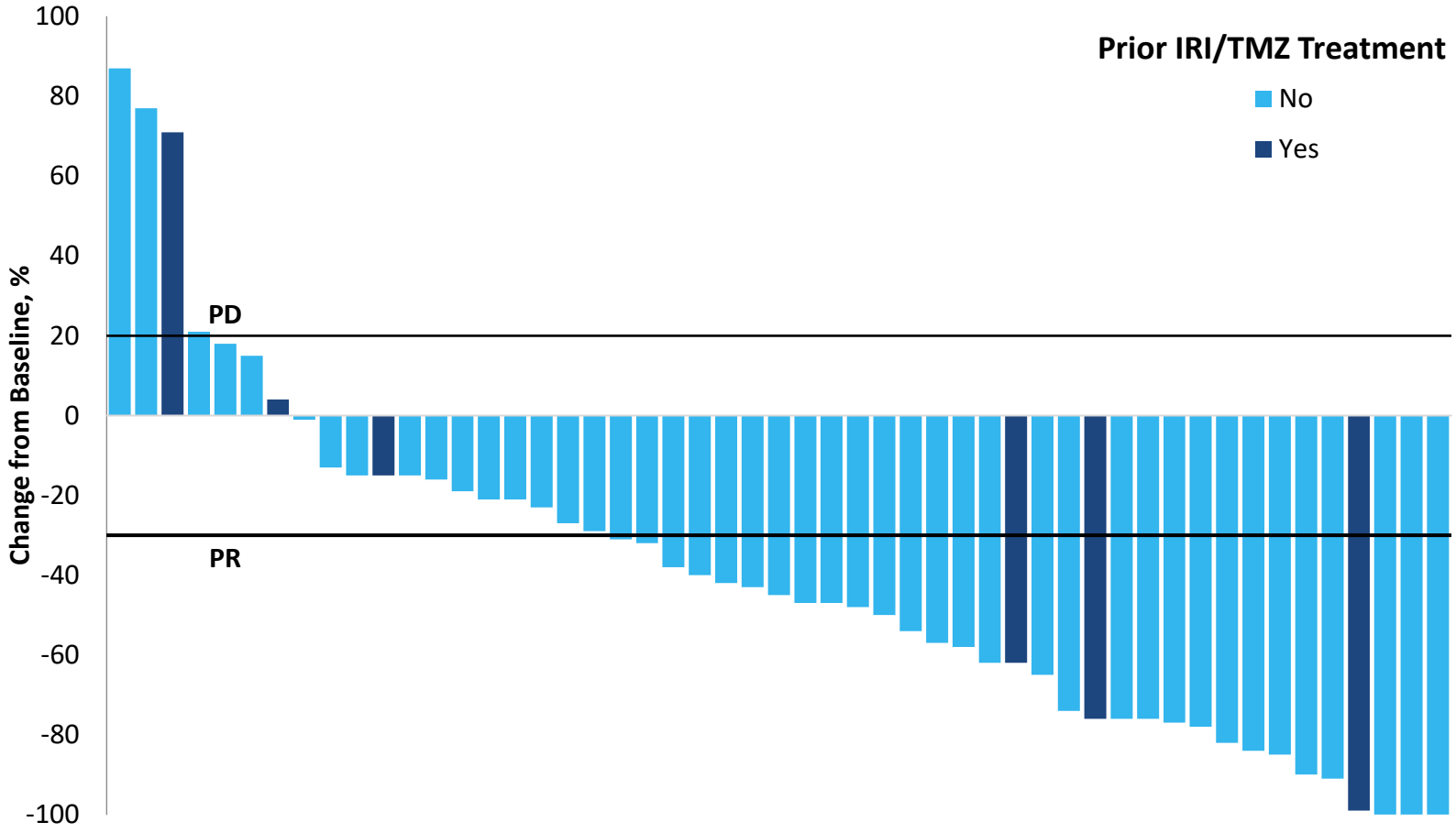
Ozekibart 3 mg/kg + IRI
50 mg/m^2/day +
TMZ 100 mg/m^2/day

Expected outcomes with current SoC

Treatment	Study Type	ORR (%)	PFS (months)
Irinotecan + Temozolomide	**Phase 2 / single-arm**	**~15–30**	**3–4**
HD Ifosfamide	**Phase 2 / rEECur**	**~20–25**	**5.7**

Updated data from expansion cohort: Ewing sarcoma + IRI/TMZ

Ozekibart (INBRX-109)



Safety: well-tolerated with the most common adverse events were diarrhea, nausea, anemia, and fatigue, all consistent with the known safety profile of IRI/TMZ.

Updated Efficacy results:

60 patients dosed with 51 patients currently evaluable for response:

(N=51)

86.3%

Disease control rate was 86.3%, or 44 out of 51 patients, as measured by RECISTv1.1

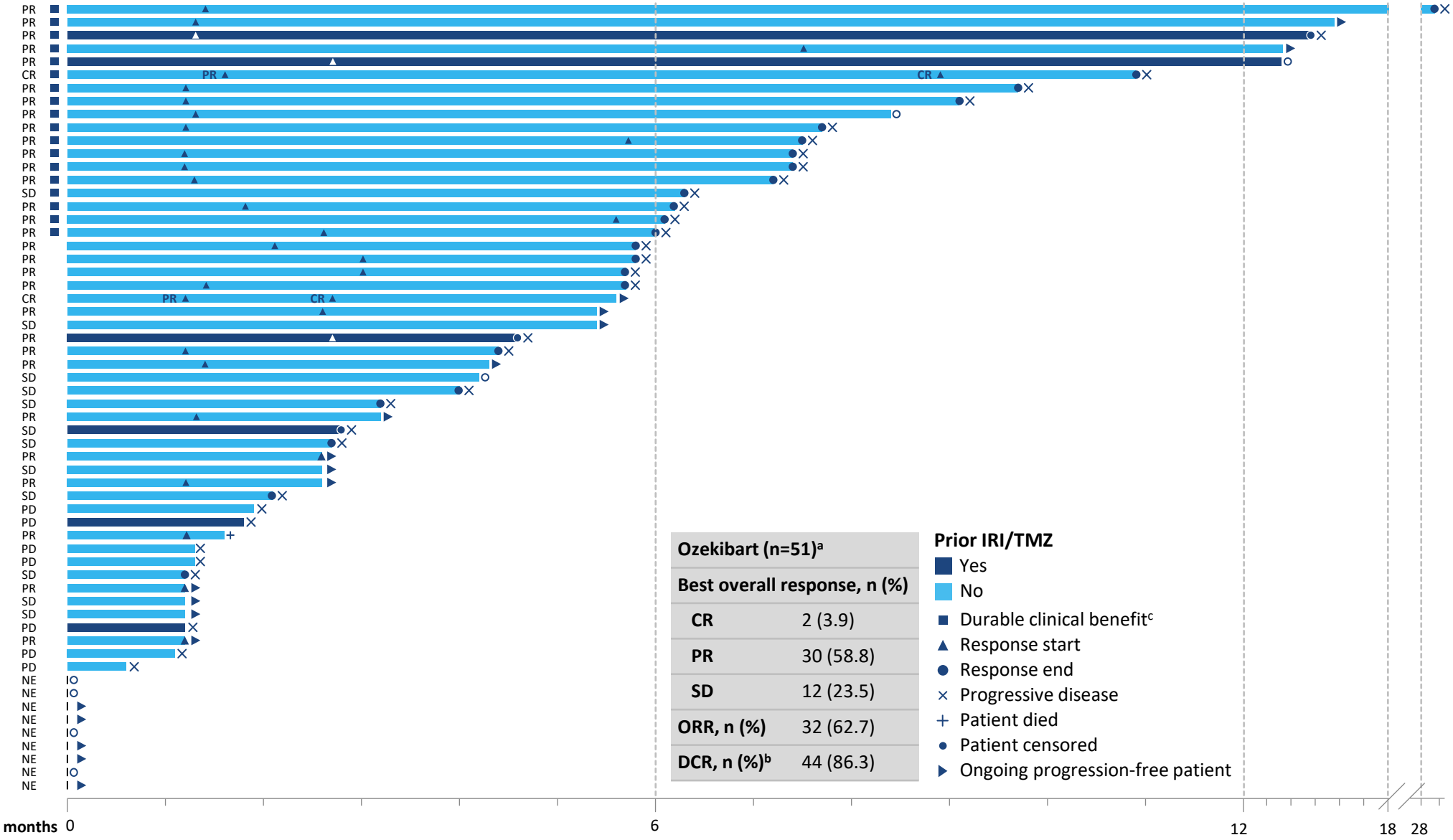
62.7%

Objective response rate was 62.7%, or 32 of 51 patients, consisting of 2 CRs and 30 PRs, as measured by RECISTv1.1

Data cutoff: September 18, 2026, as presented at CTOS 11/18-21 in Seoul, South Korea.
PD, progressive disease; PR, partial response; ORR, objective response rate; DCR, disease control rate.

Updated data from expansion cohort: Ewing sarcoma + IRI/TMZ

Ozekibart (INBRX-109)



Ozekibart (n=51)[a]	
Best overall response, n (%)	
CR	2 (3.9)
PR	30 (58.8)
SD	12 (23.5)
ORR, n (%)	32 (62.7)
DCR, n (%)[b]	44 (86.3)

Prior IRI/TMZ
- Yes
- No

- Durable clinical benefit[c]
- Response start
- Response end
- Progressive disease
- Patient died
- Patient censored
- Ongoing progression-free patient

Data cutoff: September 18, 2026. CR, complete response; DCR, disease control rate; NA, not evaluable; ORR, objective response rate; PD, progressive disease; PR, partial response; SD, stable disease.
[a] Response evaluable patients were those were treated and have ≥1 post-treatment scan or who died prior to having a post-treatment scan. [b] DCR defined as percentage of patients with a best response of CR, PR, or SD (any duration). [c] Durable clinical benefit was defined as having SD, PR, or CR for >6 months.

Initial data from Phase 1/2 colorectal adenocarcinoma in combination with FOLFIRI

Ozekibart (INBRX-109)

Key inclusion criteria

- LA/M, unresectable, R/R colorectal adenocarcinoma
- Aged 18 to <85 years
- 2-3 prior lines of systemic therapy

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)

Safety: well-tolerated with the most common treatment-related adverse events to include diarrhea, fatigue, and nausea with the majority being low-grade and consistent with the known safety profile of FOLFIRI.

68% of patients presented with liver metastases prior to treatment and no significant liver toxicity issues were observed in these patients.

45 patients evaluable for response

~70% are **4th line** & ~30% are **3rd line**

~80% have received prior IRI-containing regiment

87% Disease control rate was 87% as measured by RECISTv1.1

20% Objective response rate was 20% as measured by RECISTv1.1

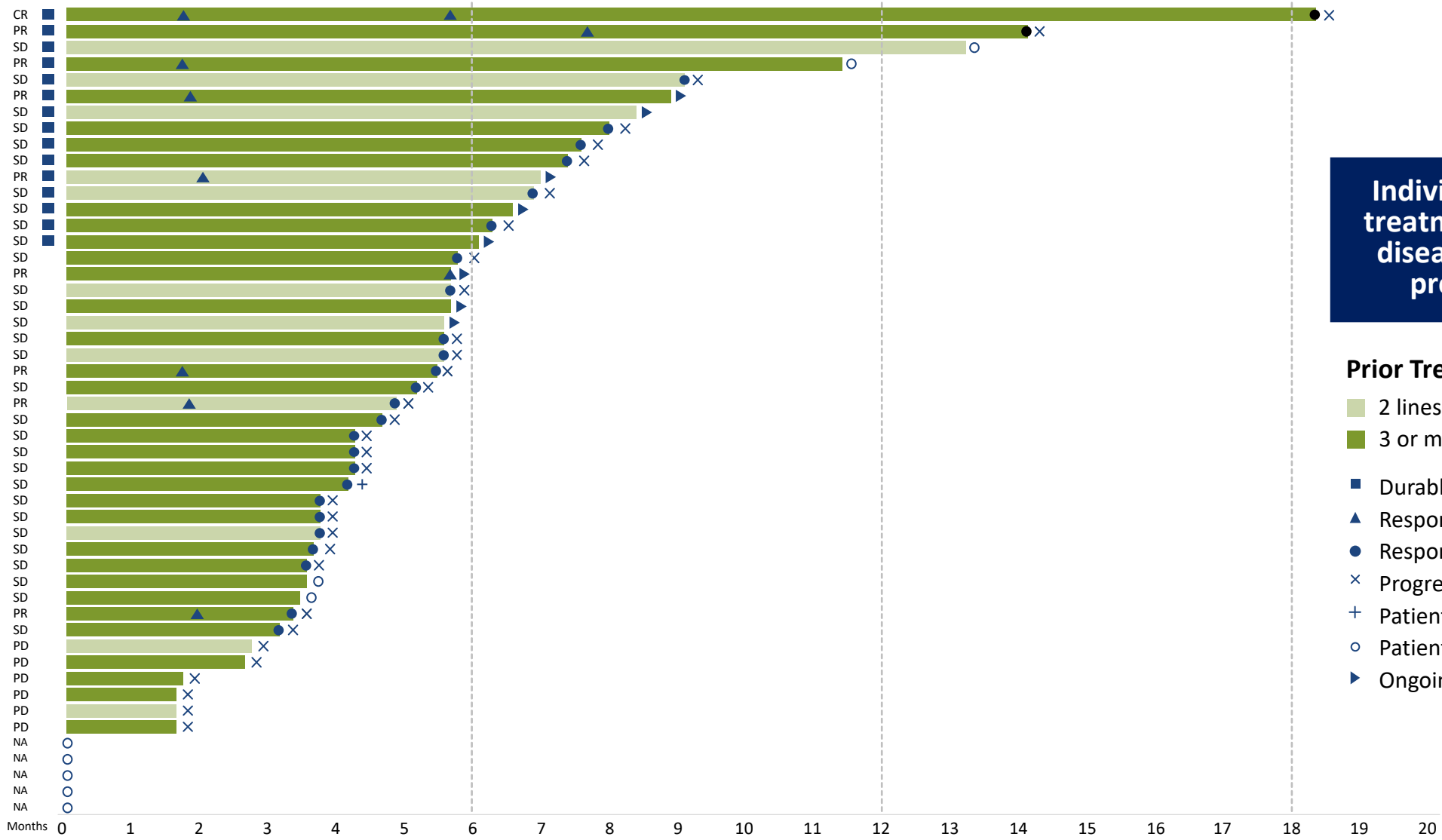
5.5 Median progression free survival was 5.5 months

42% 42% of patients remained progression-free at the 6-month mark

Data cutoff: April 10, 2026.

Phase 1/2 colorectal adenocarcinoma in combination with FOLFIRI

Ozekibart (INBRX-109)



Individual patient time on treatment shows sustained disease control in heavily pre-treated patients

Prior Treatment Status:

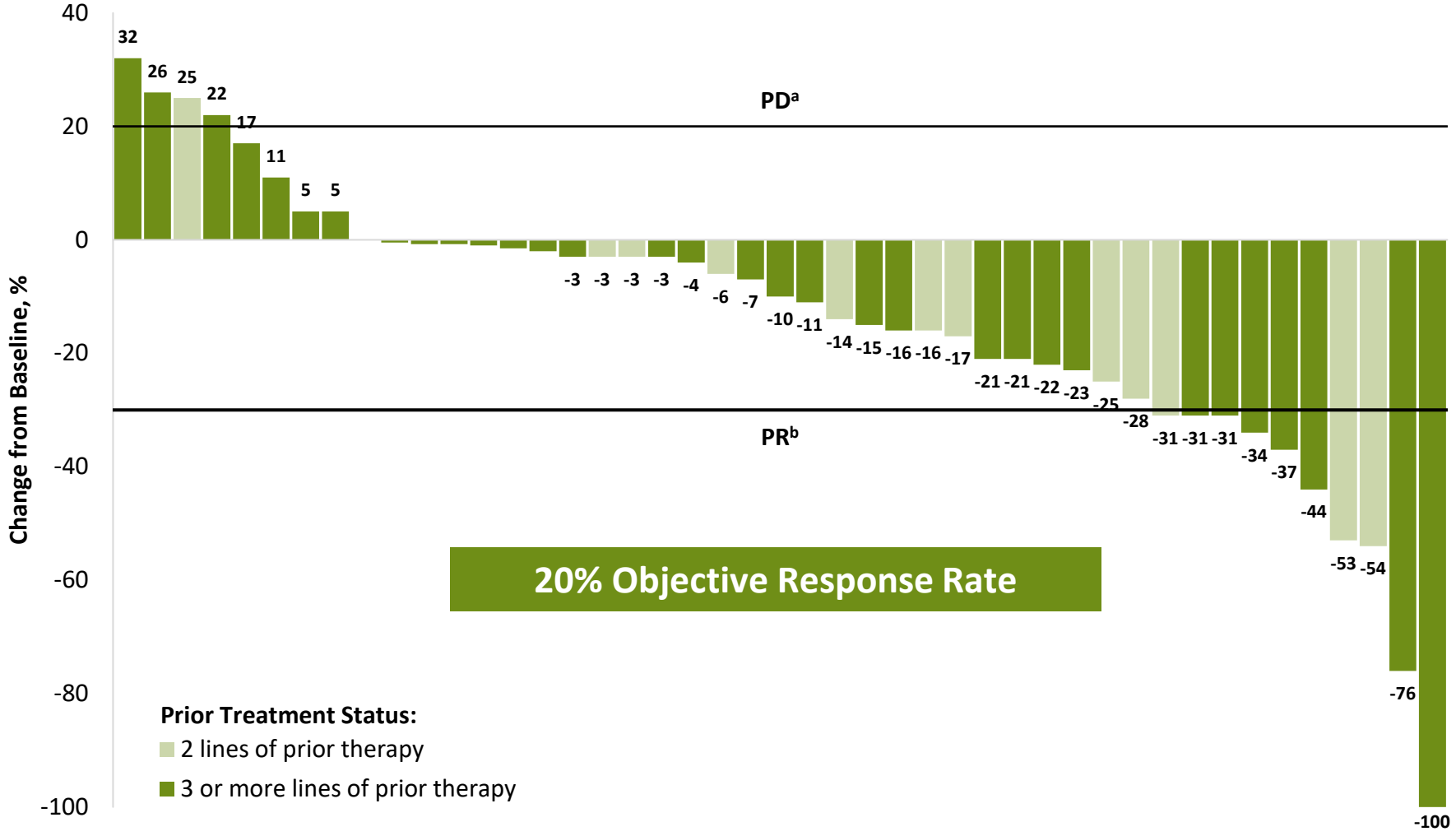
- 2 lines of prior therapy
- 3 or more lines of prior therapy

- Durable responder
- Response start
- Response end
- Progressive disease
- Patient died
- Patient censored
- Ongoing progression free patient

Data cutoff: April 10, 2026.
CR, complete response; PD, progressive disease; PR, partial response; SD, stable disease; NA, censored.

Phase 1/2 colorectal adenocarcinoma in combination with FOLFIRI

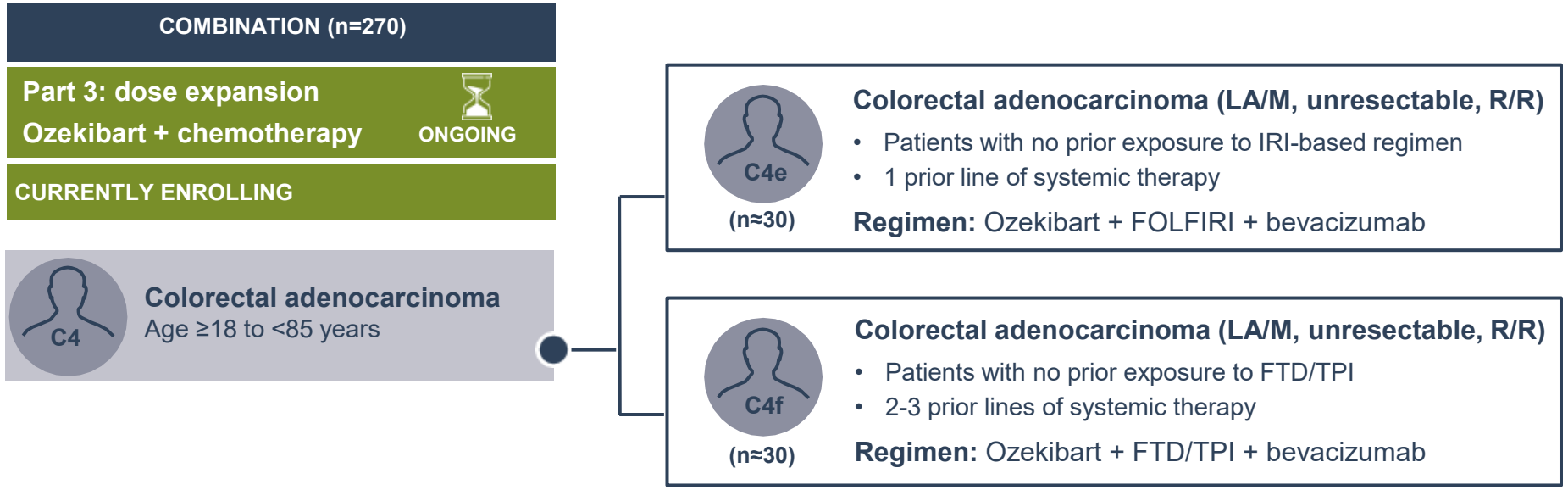
Ozekibart (INBRX-109)



Benchmarks	ORR (%)
Lonsurf + bev (SUNLIGHT, 2023)	**6.1**
regorafenib (CORRECT, 2013)	**1.0**
fruquintinib (FRESCO-2, 2023)	**1.5**

Data cutoff: April 10, 2026.
PD, progressive disease; PR, partial response; [a] Per RECIST v1.1 20% target lesion growth indicates progressive disease. [b] Per RECIST v1.1 30% shrinkage of target lesions indicates a partial response. contingent on the status of the non-target lesions and the presence of new lesions.

CRC expansion cohorts: open and enrolling

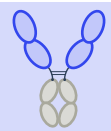


Cohort	N	Regimen	Population	Rationale
C4e	30	109+ FOLFIRI	2L+, No prior IRI-containing regimen	Provide safety data in combination with Bev and additional activity data on a IRI naïve populations
C4f	30	109 + Lonsurf + Bev	3L+ no prior exposure to Lonsurf	Efficacy signal w 3-4L with Lonsurf+Bev (opening opportunity for expanding to Ph3 in late setting)

2L, second line; 3L, third line; FOLFIRI, fluorouracil, leucovorin, and irinotecan; FTD/TPI, trifluridine-tipiracil; IRI, irinotecan; LA/M, locally advanced/metastatic; R/R, relapsed or refractory; TMZ, temozolomide.

Sarcomas offer a blockbuster $1B+ revenue opportunity; CRC offers a franchise-defining opportunity

Ozekibart (INBRX-109)



	Sarcomas		CRC	
	1L Conventional Chondrosarcoma	2L+ Ewing Sarcoma	1L	3L
US Epidemiology Estimates	Unresectable/metastatic			
	200 – 400 Newly diagnosed	**200 – 400** Newly diagnosed	**75,000**	**20,000**
	2,000 – 3,000 Prevalent at launch	**2,000 – 3,000** Prevalent at launch		
Valuation Drivers	• High share and no competition • Ultra rare oncology pricing • Long duration in cCS due to lack of available 2L options		• High share with limited competition in all-comers population • Premium oncology pricing • Very long 1L duration due to maintenance post chemo	
WW Product Sales Potential	**$500M +**	**$500M +**	**$10B+**	**$4.5B+**

Epidemiology based on SEER, SEER Explorer, and CancerMPact

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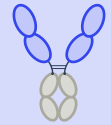
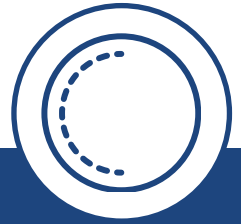
Ozekibart conclusion

Ozekibart (INBRX-109)



Ozekibart is a best-in-class DR5 agonist



Unprecedented single agent activity in chondrosarcoma registrational trial

- 52% reduction in progression or death: HR 0.46; P < 0.0001
- Median PFS more than doubled placebo: 5.55 months vs 2.69 months
- Improved disease control rate: 54% vs 27.5%
- Benefit was consistent across all pre-specified subgroups



Potential for FDA approval in April 2027 with commercial sales beginning in 1H 2027

- BLA submission accepted with anticipated PDUFA date in April 2027
- Potential to be the first treatment approved in chondrosarcoma
- Commercialization as early as 1H 2027



Encouraging Ewing sarcoma data

- Updated results from Ewing cohort of 51 evaluable patients showed an ORR of 62.7%
- DCR rate of 86.3% and a median PFS of 6 months
- Intend to rapidly advance our plans in order to allow patients access as soon as possible



Potential for expansion in colorectal cancer and other solid tumor types

- Initial CRC data (predominantly 4L): 20% ORR
- 2L (FOLFIRI + Avastin) and 3L/4L (Lonsurf + Avastin) combo cohort data expected Q1 2027
- Potential to expand into 1L Ph3 (ozekibart + FOLFIRI + bev)
- MoA supports potential expansion into other tumor types and combinations, including ADCs

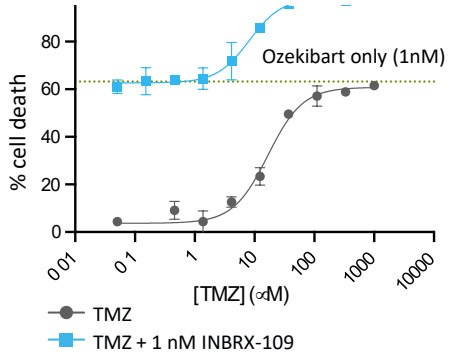
109 Appendix

Ozekibart exhibits anti-tumor activity in GBM models as monotherapy and in combination with TMZ

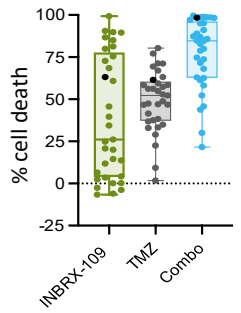
Ozekibart (INBRX-109)

Human GBM cell line models

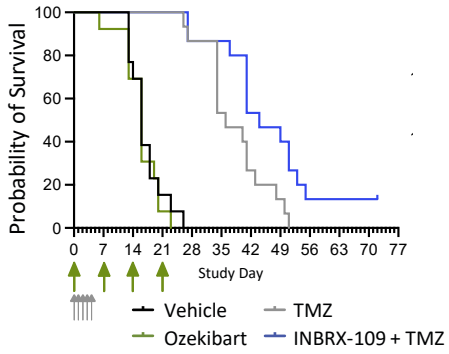
In vitro cytotoxicity
U-87 MG



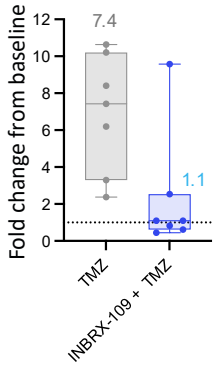
In vitro cytotoxicity
GBM cell lines



Intracranial U-87 MG
Survival analysis



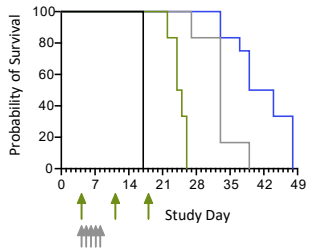
Intracranial U-87 MG
Survival analysis



- Combination treatment with ozekibart and temozolomide (TMZ) induced greater in vitro cell death than TMZ alone for a majority of GBM cell lines tested (n=31)
- In the intracranial U-87 MG tumor mouse model, combination treatment demonstrated superior tumor control and enhanced survival compared to TMZ alone

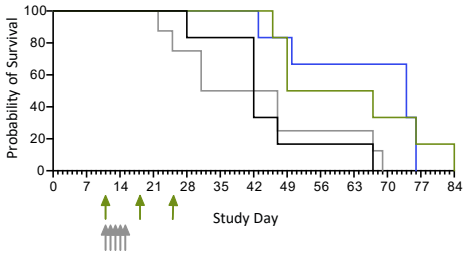
Human GBM patient-derived xenograft (PDX) models*

Intracranial GBM38
Survival analysis



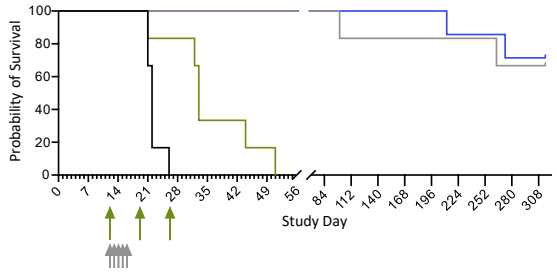
*p = 0.002, INBRX-109 vs vehicle
** p – 0.004, combo vs TMZ

Intracranial GBM44
Survival analysis



*p = 0.03, ozekibart vs vehicle
** p – 0.01, combo vs TMZ

Intracranial GBM39
Survival analysis



*p = 0.009, ozekibart vs vehicle

	Median Survival (d)		
Treatment	GBM38	GBM39	GBM44
Vehicle	17	22	42
Ozekibart	25	33	58
TMZ	33	n.d.	39
Combo	42	n.d.	74

- In all tested intracranial PDX tumor mouse models, ozekibart monotherapy significantly enhanced survival
- In 2 of 3 PDX models, combination treatment with ozekibart and TMZ led to a greater survival benefit than TMZ alone

In collaboration with Jann Sarkaria, M.D., Mayo Clinic

INHIBRX

Investor Relations:

KELLY DECK, CPA
CFO

11025 N. Torrey Pines Road
Suite 140
La Jolla, CA 92037

858.795.4260

ir@inhibrx.com

