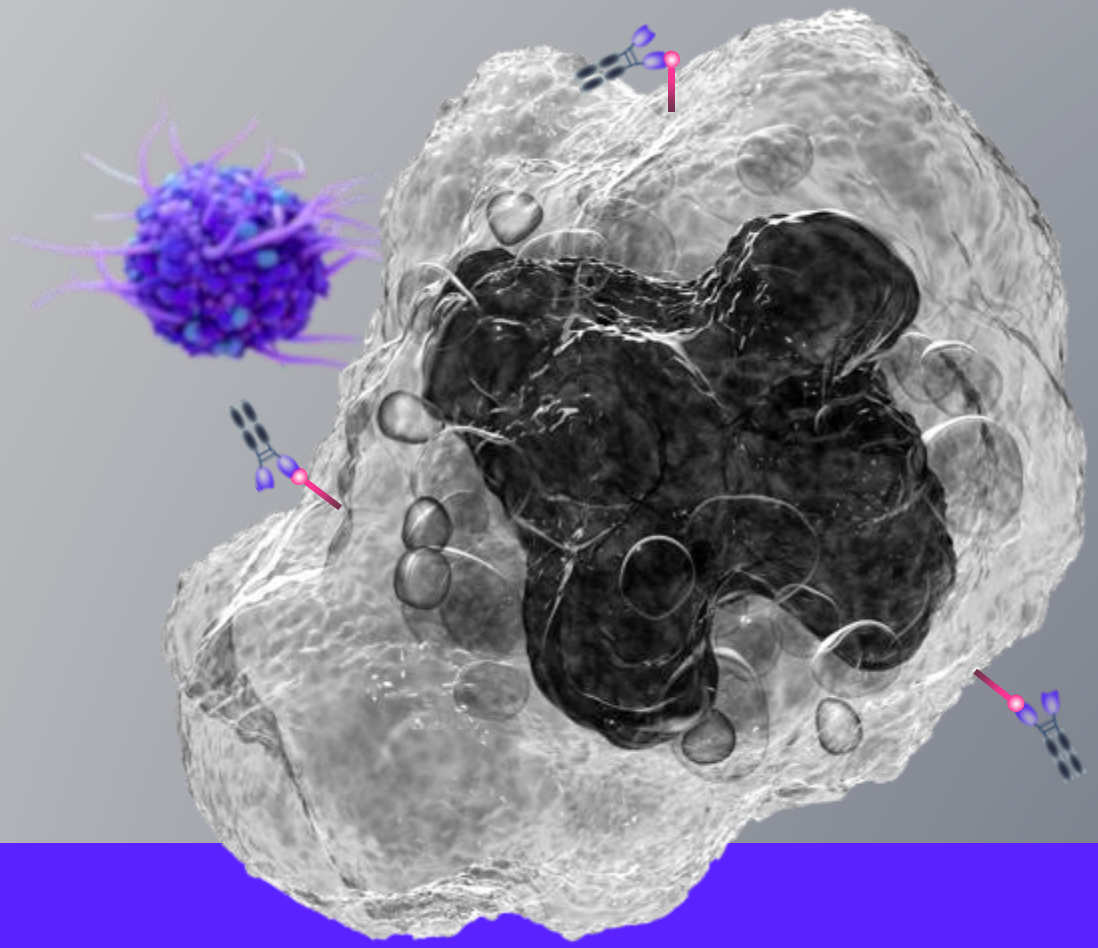

NASDAQ – ALXO

ALX™ ONCOLOGY

Corporate Overview

November 2026

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Forward-Looking Statements

Certain information set forth in this presentation contains “forward-looking information”, under applicable laws collectively referred to herein as forward-looking statements. Except for statements of historical fact, information contained herein constitutes forward-looking statements and includes, but is not limited to the (i) results and cost and timing of our product development activities and clinical trials; (ii) completion of the Company’s clinical trials that are currently underway, in development or otherwise under consideration; (iii) our expectations about the timing of achieving regulatory approval and the cost of our development programs; (iv) projected financial performance of the Company; (v) the expected development of the Company’s business, projects, collaborations and joint ventures; (vi) execution of the Company’s vision and growth strategy, including with respect to global growth; (vii) sources and availability of third-party financing for the Company’s research and development; (viii) future liquidity, working capital, and capital requirements; and (ix) industry trends. These and other risks are described more fully in ALX Oncology’s filings with the Securities and Exchange Commission (“SEC”), including ALX Oncology’s Annual Report on Form 10-K and other documents ALX Oncology files with the SEC from time to time.

Although forward-looking statements contained in this presentation are based upon what management of the Company believes are reasonable assumptions, there can be no assurance that forward-looking statements will prove to be accurate. Actual results and future events could differ materially from those anticipated in such statements. The Company undertakes no obligation to update forward-looking statements if circumstances or management’s estimates or opinions should change except as required by applicable securities laws.

This presentation concerns product candidates that are under clinical investigation, and which have not yet been approved for marketing by the U.S. Food and Drug Administration. These product candidates are currently limited by federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated. Interim, initial and preliminary data from our clinical trials that we announce or publish from time to time may change as more patient data become available and are subject to audit and verification procedures that could result in material changes in the final data.

This presentation also contains estimates and other statistical data made by independent parties and by ALX Oncology relating to market size and growth and other industry data. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions, and estimates of ALX Oncology’s future performance and the future performance of the markets in which it operates are necessarily subject to a high degree of uncertainty and risk.

A registration statement on Form S-3 has been filed with and declared effective by the SEC. The offering of these securities will be made only by means of a prospectus supplement and base prospectus forming part of the effective registration statement relating to the securities. Copies of the prospectus supplement for this offering may be obtained, when available, by contacting Piper Sandler & Co., 350 North 5th Street, Suite 1000, Minneapolis, MN 55401, Attention: Prospectus Department, by telephone at (800) 747-3924, or by email at prospectus@psc.com; or Wells Fargo Securities, Attention: Equity Syndicate Department, 500 West 33rd Street, New York, New York, 10001, by telephone at (833) 690-2713 or by email at cmclientsupport@wellsfargo.com.

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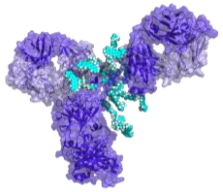
ALX is Rapidly Advancing Novel Cancer Treatments

Evorpacept

- Leading CD47 program in development with potential to be next targeted immuno-oncology breakthrough
- Unique design with inactive Fc differentiated from past attempts to target CD47
- Demonstrated activity in five combinations to date and a targetable CD47 biomarker
- Advancing trials in breast cancer and multiple myeloma*



ALX2004

- Highly differentiated EGFR ADC in Ph1 dose escalation in the US
- Meticulously designed and developed in-house to maximize therapeutic window
- Preclinical data support dose dependent activity and a differentiated safety profile
- Targeting EGFR-expressing tumors in Ph1 including NSCLC, CRC, HNSCC, and ESCC

* Sanofi-sponsored trial; HNSCC: head and neck squamous cell carcinoma; CRC: colorectal cancer; NSCLC: non-small cell lung cancer; ESCC: esophageal squamous cell carcinoma

ALX Is Focused on Driving Toward Multiple Inflection Points in 2026-2027

PROGRAM	INDICATION	ANTICIPATED MILESTONES
EVORPACEPT		
ASPEN-Breast Evorpacept, trastuzumab + chemotherapy	ENHERTU®-Experienced HER2-Positive Breast Cancer	**Topline data for 120 patients – 2H-2027**
ALX2004		
ALX2004 Dose-escalation and expansion	EGFR-Expressing Solid Tumors	**Safety and early efficacy data from dose escalation phase – Dec. 2026**

Projected Cash Runway through First Half of 2028

ALX Oncology is Pursuing a Focused Development Plan with Upcoming Catalysts in 2026

MODALITY /TARGET	PROGRAM	INDICATION	IND ENABLING	PHASE 1	PHASE 2	PHASE 3	STATUS
EVORPACEPT PROGRAMS							
Anti-Cancer Antibodies	**ASPEN-09-Breast** Evorpacept, Trastuzumab + chemotherapy	ENHERTU®-Experienced HER2-Positive Breast Cancer					**Enrolling, topline data for 120 patients anticipated 2H-2027**
	SARCLISA® + Dexamethasone[1] + Evorpacept	RRMM (Relapsed or Refractory Multiple Myeloma)					**Dose escalation complete, now in dose optimization**
	ASPEN-06 Evorpacept, Trastuzumab, CYRAMZA® + Paclitaxel[2]	2L or 3L Advanced HER2-Overexpressing Gastric/Gastroesophageal Junction (GEJ)					**Ph2 completed, established POC**
	Zanidatamab[3] + Evorpacept	HER2-Expressing Breast Cancer and Other Cancers					**Completed, biomarker analysis presented at ESMO Breast Cancer 2026**
ALX2004 PROGRAM							
EGFR ADC	**ALX2004** Dose-escalation and expansion	EGFR-Expressing Solid Tumors					**Enrolling, dose escalation phase safety and early efficacy data Dec. 2026**

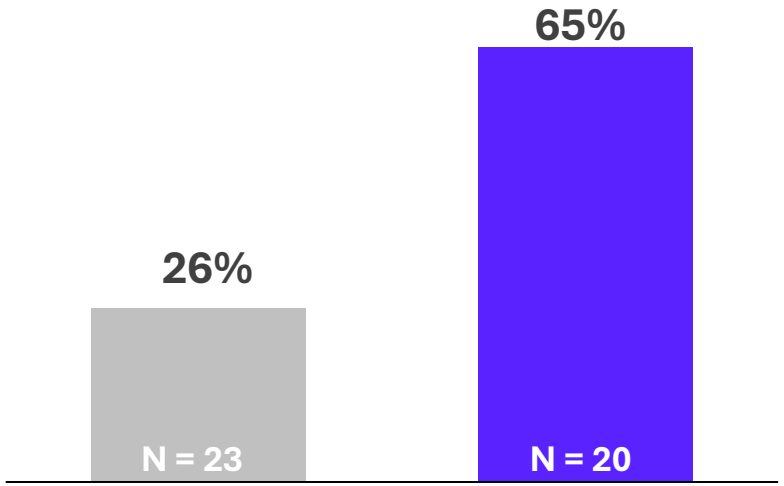
ALX-Sponsored trial ● Active Trials ● Completed Trials

ALX Oncology retains worldwide rights to evorpacept; 1. Sanofi sponsors SARCLISA® clinical trial 2. Lilly supplies CYRAMZA® for ALX Oncology's ASPEN-06 program 3. Jazz Pharmaceuticals and ALX Oncology are collaborating to conduct the zanidatamab /evorpacept clinical trial

Clinical Data Support that Evorpacept Improves Upon Anti-Tumor Activity of Standard of Care Anti-Cancer Antibodies in HER2+ Solid Tumors

HER2+ Gastric Cancer

(CD47-high expression and retained HER2+[1])



Trastuzumab + ramucirumab + paclitaxel

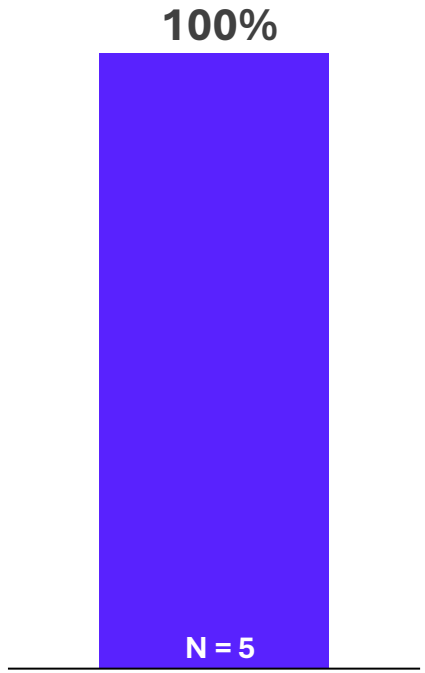
Evo + trastuzumab + ramucirumab + paclitaxel

ASPEN-06 subset, randomized Ph2

ORR

HER2+ Breast Cancer

(CD47-high expression and HER2+ by central assessment [2])



Evo + Zanidatamab[2]

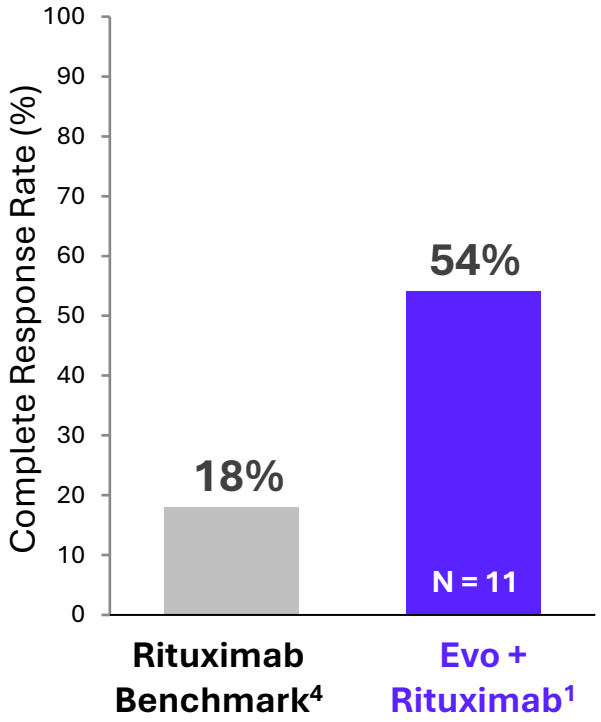
Phase 1b/2 subset

1. Wainberg et al., The 2025 Society for Immunotherapy of Cancer (SITC) Annual Meeting, November 5–9, 2025. Abstract #496. Note: Above results are based on pre-planned exploratory analysis of ASPEN-06 gastric study. CD47-high is ≥ 10% cells IHC3+; 2. ESMO Breast Cancer 2026, #72P; CD47 High = total membrane staining ≥ 20%;
ORR = overall response rate; IST = investigator-sponsored trial

Evorpacept and Rituximab-Based Regimens Have Shown a Consistent Improvement Over Historic Benchmarks in Indolent Lymphoma

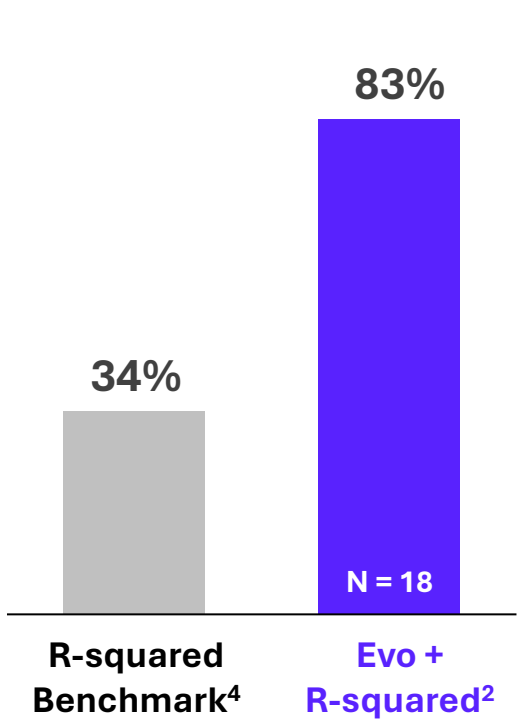
Evo + Rituximab in R/R indolent NHL[1]

(ALX-sponsored ASPEN-01 Ph1B)



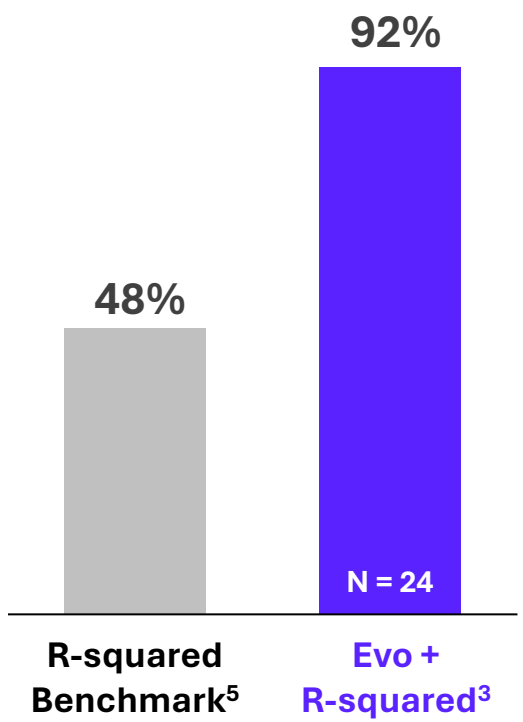
Evo + R-squared in 2L indolent NHL[2]

(MDACC Ph1 IST)



Evo + R-squared in untreated indolent NHL[3]

(MDACC Ph2 IST)



Improvement in complete response (CR) rate compared to historic benchmarks

1) ASPEN-01, Kim, *Haematologica*, 2025; 2) AACR 2024 #10285; 3) ASH 2025 #3571; 4) AUGMENT study, Leonard, *JCO*, 2019; 5) RELEVANCE study, Morschhauser, *NEJM*, 2018
Indolent lymphoma includes FL and marginal zone lymphoma; CR = complete response; IST = investigator-sponsored trial; FL = follicular lymphoma; R-squared = rituximab plus lenalidomide

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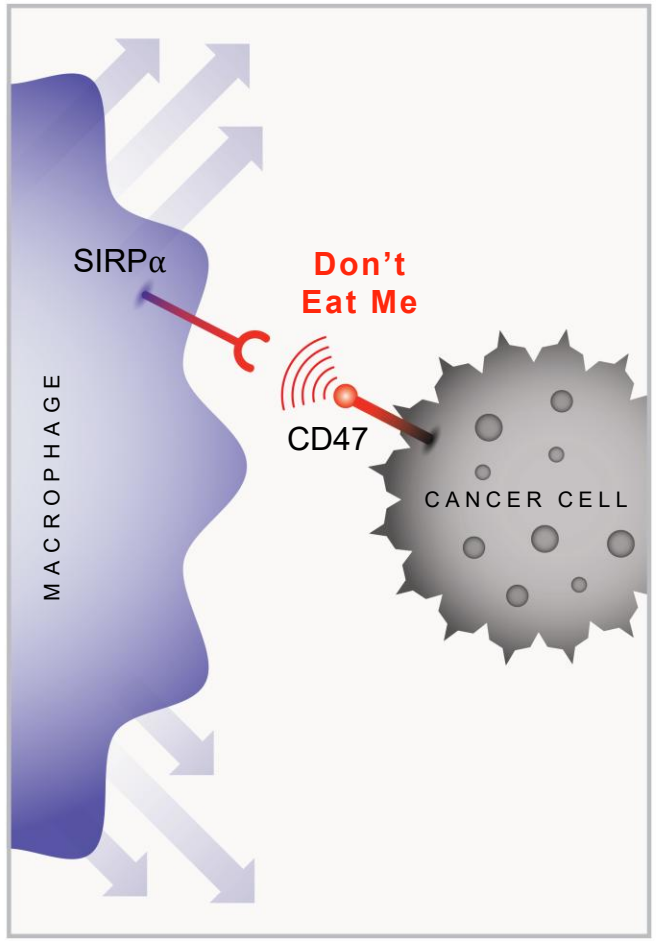
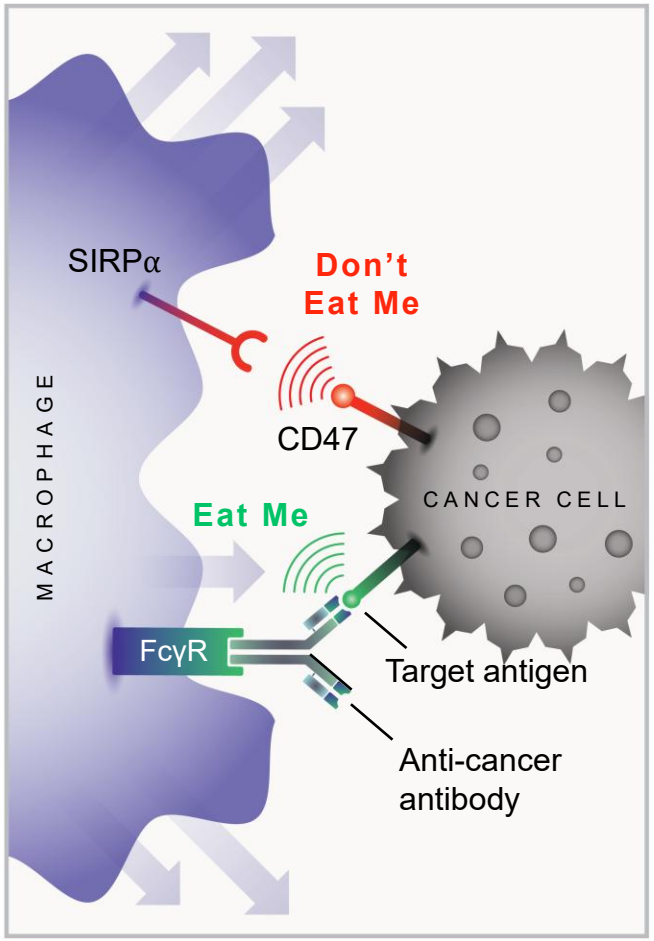
EVORPACEPT

Advancing a Synergistic Approach to Cancer Treatment Leveraging CD47

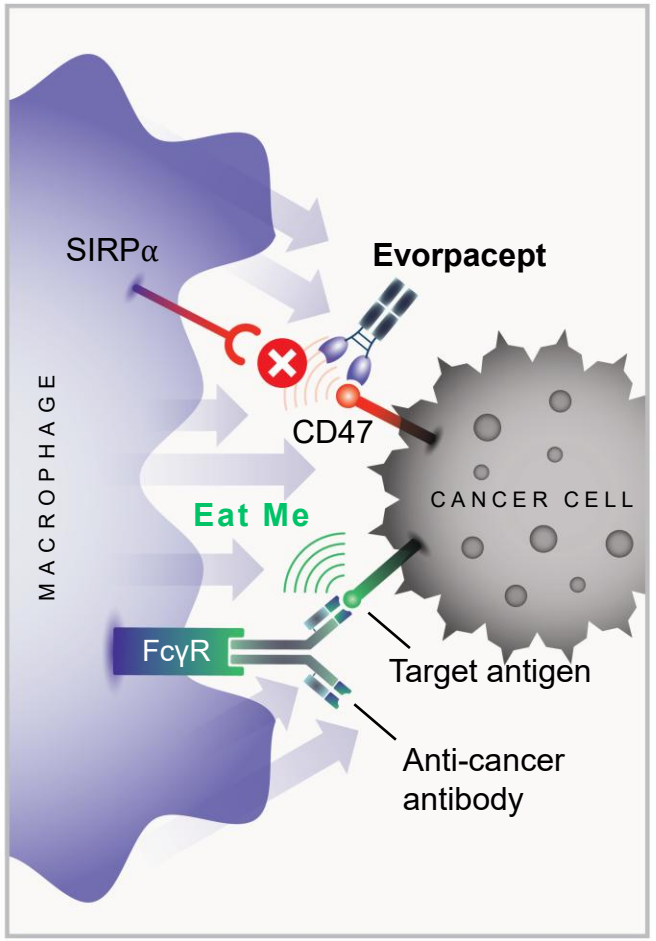
Evorpacept Blocks the CD47-SIRPα Interaction, Enhancing the Targeted ADCP of Cancer Cells when Given in Combination with Anti-Cancer Antibodies



Cancer cells overexpress CD47 in order to evade immune detection



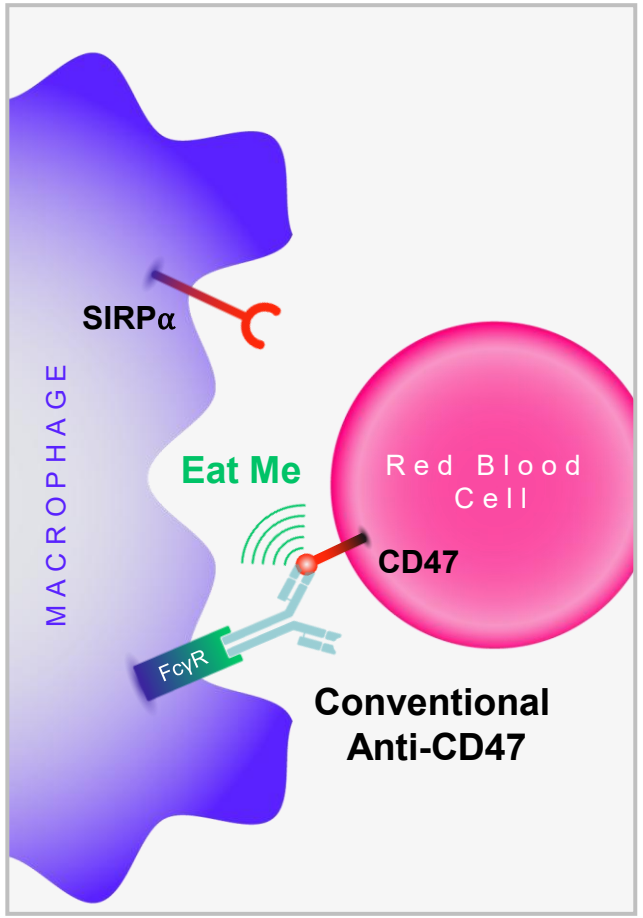
ADCP of anti-cancer antibodies is inhibited by CD47



Evorpacept blocks the "don't eat me" signal and maximizes anti-cancer activity

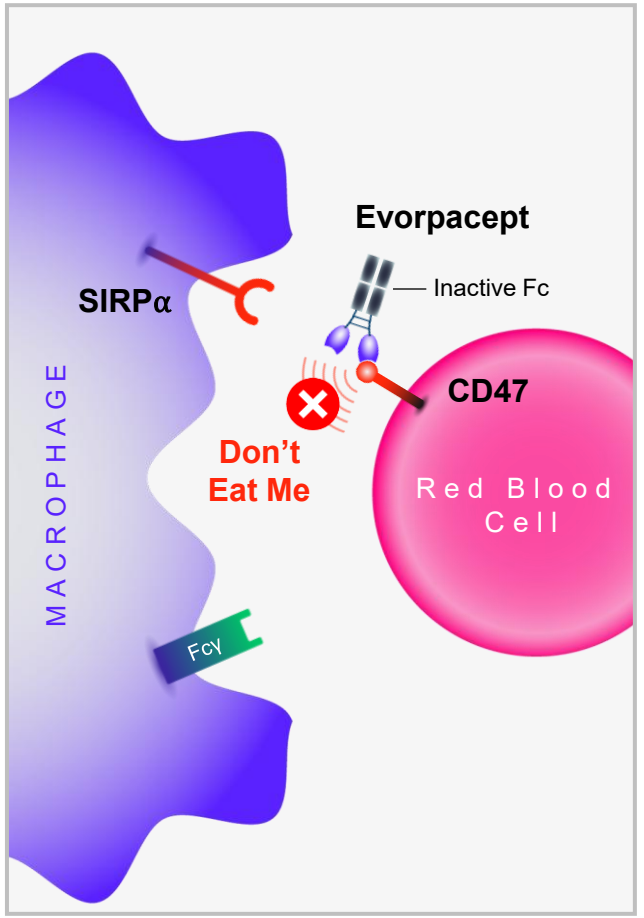
Evorpacept Is the Only CD47 Blocker with an Inactive Fc Designed to Avoid Toxicities Seen with Conventional Anti-CD47

Conventional anti-CD47 with Active Fc



Due to CD47's expression on red blood cells, this caused on-target, off-tumor toxicities

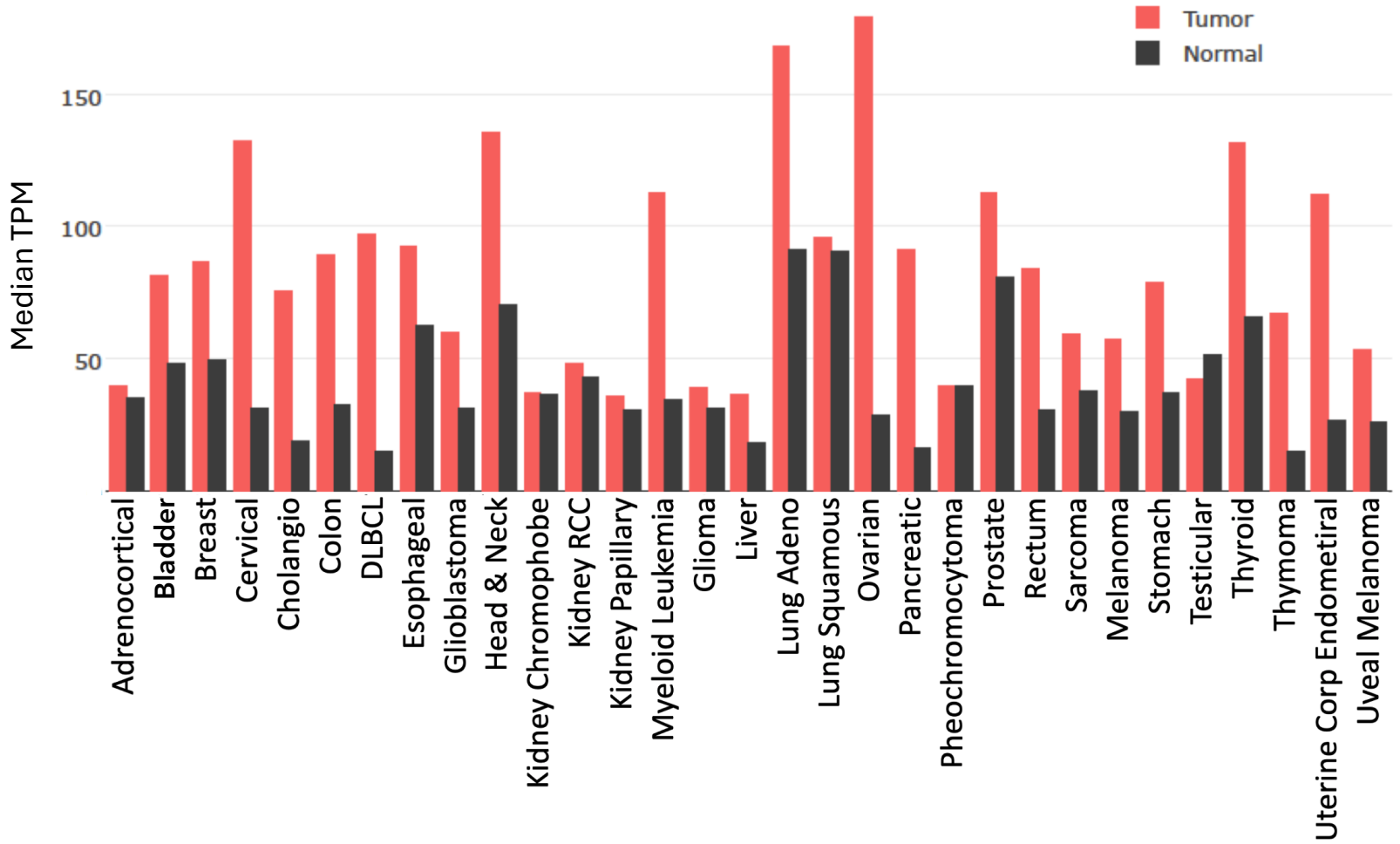
Evorpacept with Inactive Fc



Inactive Fc spares normal cells minimizing toxicity

CD47 is Overexpressed Across a Range of Solid and Heme Malignancies

CD47 Expression Levels from RNA Sequencing[1]



- As a ”marker of self”, CD47 is expressed on all cell types[2]
- Cancer cells take advantage of this by overexpressing CD47
- Due to this, the vast majority of both solid and liquid tumors utilize CD47 to evade the immune system

1) Tang, et al, GEPIA, 2017; 2) Dheilly, et al, Mol. Ther., 2017; TPM = transcripts per million

Research in CD47 Over the Last 10+ Years Provides a Strong Foundation for Utilizing CD47 as a Negative Prognostic Biomarker

In a meta-analysis of 38 cohorts across 17 publications including >7,000 patients, "CD47 overexpression correlated with shorter OS in cancer patients"*

Increased CD47 expression is correlated with poor patient outcomes in many tumor types including[1]:

- Oral squamous cell carcinoma[2]
- Nasopharyngeal carcinoma[3]
- Triple negative breast cancer[4]
- Ovarian cancer[5]
- Non-small cell lung cancer[6]
- Clear cell renal cell carcinoma[7]
- Hepatocellular carcinoma[8]
- Gastric adenocarcinoma[9]
- Colorectal adenocarcinoma[10]
- Head and neck squamous cell carcinoma[11]
- Multiple myeloma[12]

*Yang et al, *Translational Cancer Research*, 2018; 1) Huang, et al, *Scientific Reports*, 2022; 2) Pai, et al, *Cells*, 2019; 3) Wang, et al, *OncoTargets & Ther*. 2020; 4) Yuan, et al, *Oncol Lett*, 2019; 5) Li, et al, *Am J Trans Res*, 2017; 6) Barrera, et al, *Br J Cancer*, 2017; 7) Jiang, et al, *Urol Oncol*, 2022; 8) Kim, et al, *J Clin Pathol*, 2021; 9) Shi, et al, *Cancer Imm*, Imm, 2021; 10) Kim, et al, *Diagnostics*, 2021; 11) Wu, et al, *Oncoimmunology*, 2018; 12) Rastgoo, et al, *Haematologica*, 2020; OS = overall survival

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EVORPACEPT

Clinical Data Summary

HER2+ Breast and Gastric Cancer

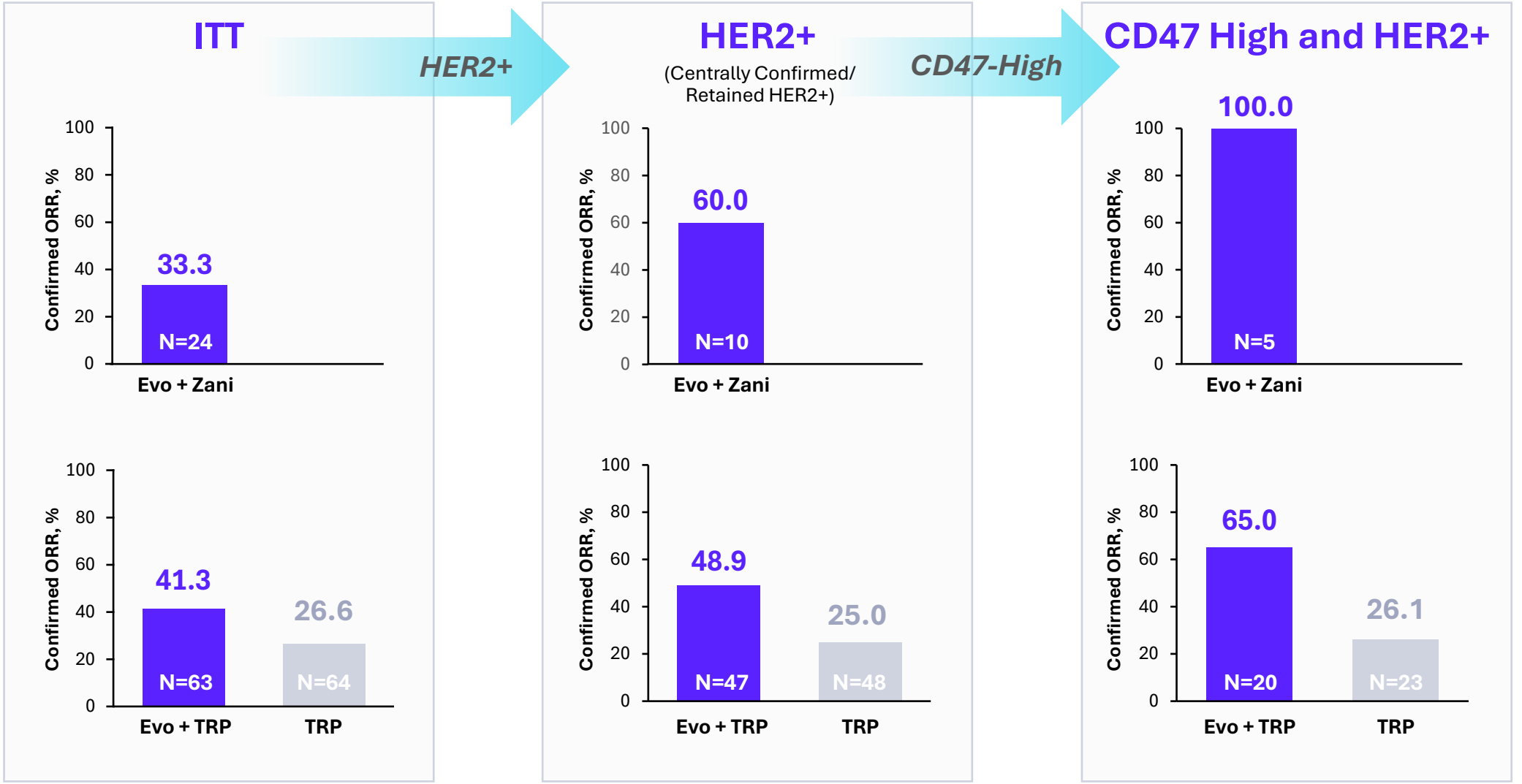
CD47 Overexpression Demonstrated Enrichment in Response Across 2 Independent HER2 Directed Trials with Evorpacept

HER2+ Breast Cancer[1]

Ph1b/2
Evo + Zani

HER2+ Gastric Cancer[2]

Ph2 ASPEN-06

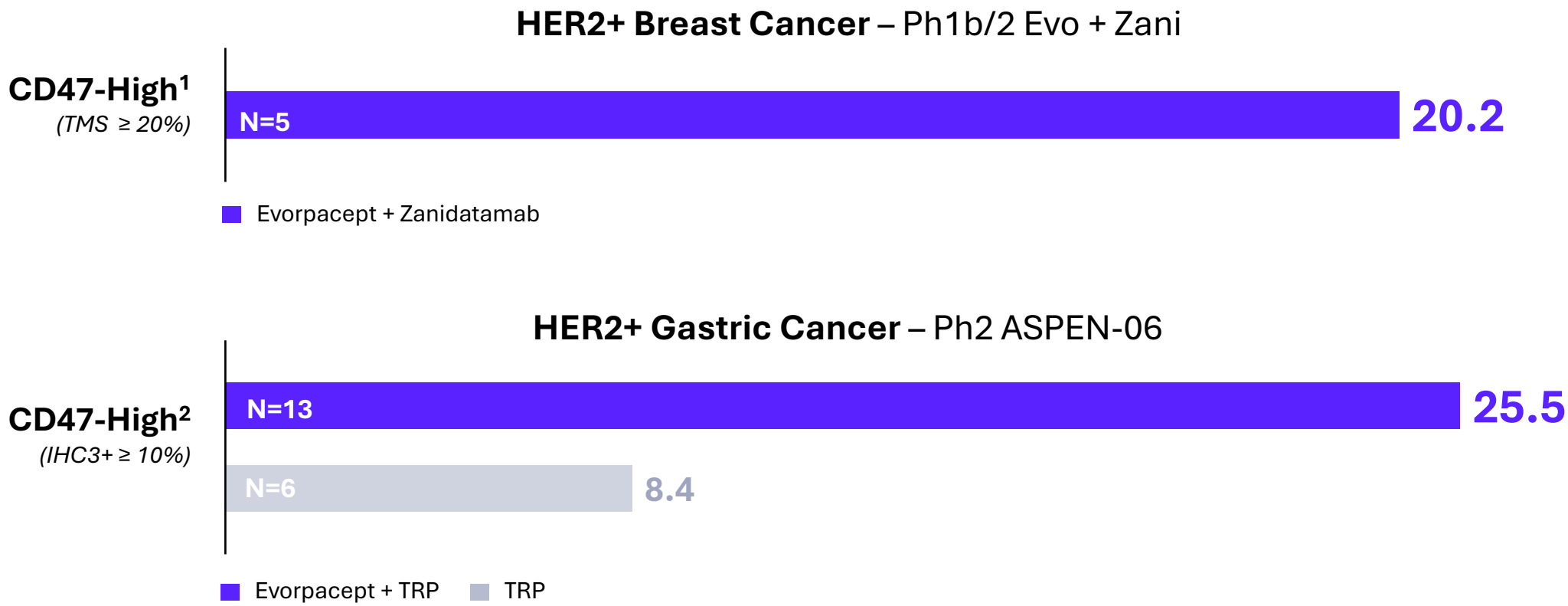


[1]Data cutoff as of August 1, 2024; HER2-positive = IHC3+, IHC2+ / ISH+; ESMO Breast Cancer 2026, #72P; CD47 High = total membrane staining ≥ 20%; * Treatment Cohort 1 was preceded by an evo dose escalation cohort at 20 mg/kg (n=3, w/ HER2+ BC), of which one patient had centrally assessed HER2+ status.

[2]Wainberg et al., The 2025 Society for Immunotherapy of Cancer (SITC) Annual Meeting, November 5–9, 2025. Abstract #496. Note: Above results are based on pre-planned exploratory analysis of ASPEN-06 gastric study. Retained HER2+ based on fresh biopsy or ctDNA amplification. Data Cutoff as of May 15, 2025. **T** = trastuzumab; **R** = ramucirumab; **P** = paclitaxel. ; CD47 High defined as (IHC3+ ≥ 10%)

Long Median DOR in Patients with CD47-High Expression was Observed Across 2 Independent HER2-Directed Trials with Evorpacept

Median Duration of Response (Months)

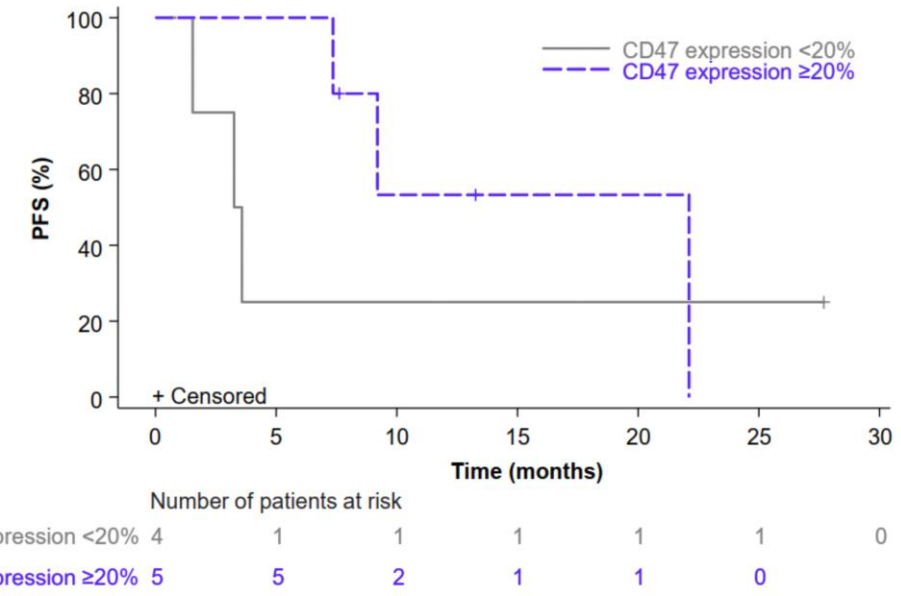


[1]Data cutoff as of August 1, 2024; HER2-positive = IHC3+, IHC2+ / ISH+; ESMO Breast Cancer 2026, #72P; CD47 High = total membrane staining ≥ 20% in confirmed HER2+ cohort; * Treatment Cohort 1 was preceded by an evo dose escalation cohort at 20 mg/kg (n=3, w/ HER2+ BC), of which one patient had centrally assessed HER2+ status.

[2]Wainberg et al., The 2025 Society for Immunotherapy of Cancer (SITC) Annual Meeting, November 5–9, 2025. Abstract #496. Note: Above results are based on pre-planned exploratory analysis of ASPEN-06 gastric study. Retained HER2+ based on fresh biopsy or ctDNA amplification. Data Cutoff as of May 15, 2025. **T** = trastuzumab; **R** = ramucirumab; **P** = paclitaxel.

Longer PFS in Patients with CD47-High Expression was Observed in Two Independent HER2 Directed Trials with Evorpacept

PFS - CD47 High vs. Low
Ph1b/2 Breast Cancer (Evo + Zani)
(Centrally confirmed HER2+ Patients)

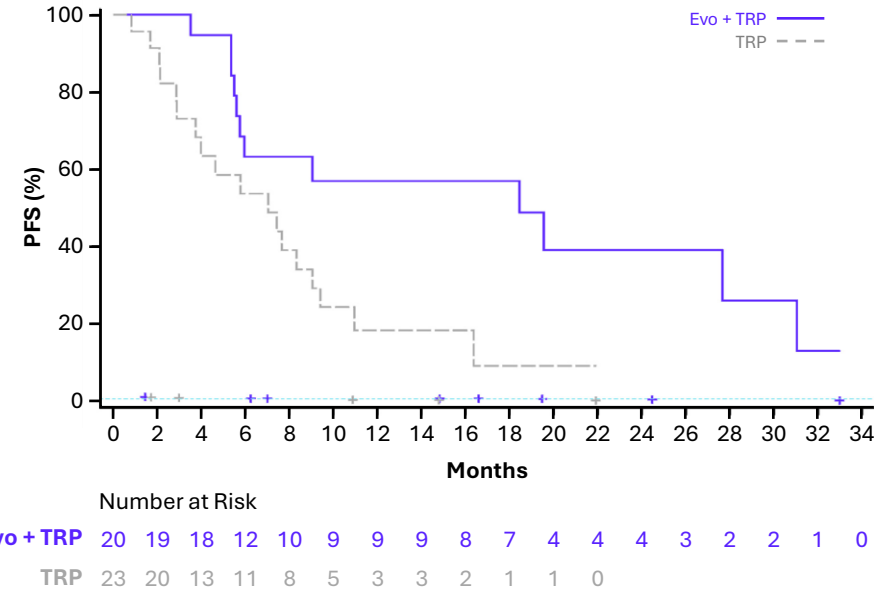


Median PFS

CD47 High (Evo + Zani): **22.1 mos**

CD47 Low (Evo + Zani): **3.4 mos**

PFS - CD47 High
Ph2 Aspen-6 Gastric Cancer
(Retained HER2+ patients)



Median PFS

CD47 High (Evo + TRP): **18.4 mos**

CD47 High (Control): **7.0 mos**

HR = 0.39 (0.17, 0.86)

ESMO Breast Cancer 2026, #72P; Data cutoff: August 1, 2024; ccHER2, centrally confirmed human epidermal growth factor receptor 2; CD47 High = total membrane staining > 20%; ; PFS, progression-free survival.

Wainberg et al., The 2025 Society for Immunotherapy of Cancer (SITC) Annual Meeting, November 5–9, 2025. Abstract #496. Note: Above results are based on pre-planned exploratory analysis of ASPEN-06 gastric study. Retained HER2+ based on fresh biopsy or ctDNA amplification; PFS by Investigator assessment. Data Cutoff as of May 15, 2025. **T** = trastuzumab; **R** = ramucirumab; **P** = paclitaxel. CD47 High defined as (IHC3+ ≥ 10%)

Evorpacept + TRP Showed Consistent Benefit Across Multiple CD47 Cut-Points in Retained HER2+ & CD47+ Patients in the ASPEN-06 Study

CD47 Cut-off[a]	% of HER2+ Subgroup (n=95)	ORR		PFS HR[b] (95% CI)	OS HR[b] (95% CI)
		Evorpacept + TRP	TRP		
No cut-off	100%	**49%** (n=47)	**25%** (n=48)	**0.72** (0.44, 1.18)	**0.95** (0.58, 1.56)
≥10% Med/High	57%	**56%** (n=25)	**24%** (n=29)	**0.40** (0.19, 0.82)	**0.75** (0.39, 1.46)
≥25% Med/High	40%	**60%** (n=20)	**22%** (n=18)	**0.36** (0.15, 0.84)	**0.65** (0.30, 1.41)
≥5% High	51%	**64%** (n=22)	**23%** (n=26)	**0.38** (0.17, 0.84)	**0.66** (0.32, 1.37)
≥10% High	45%	**65%** (n=20)	**26%** (n=23)	**0.39** (0.17, 0.86)	**0.70** (0.33, 1.47)

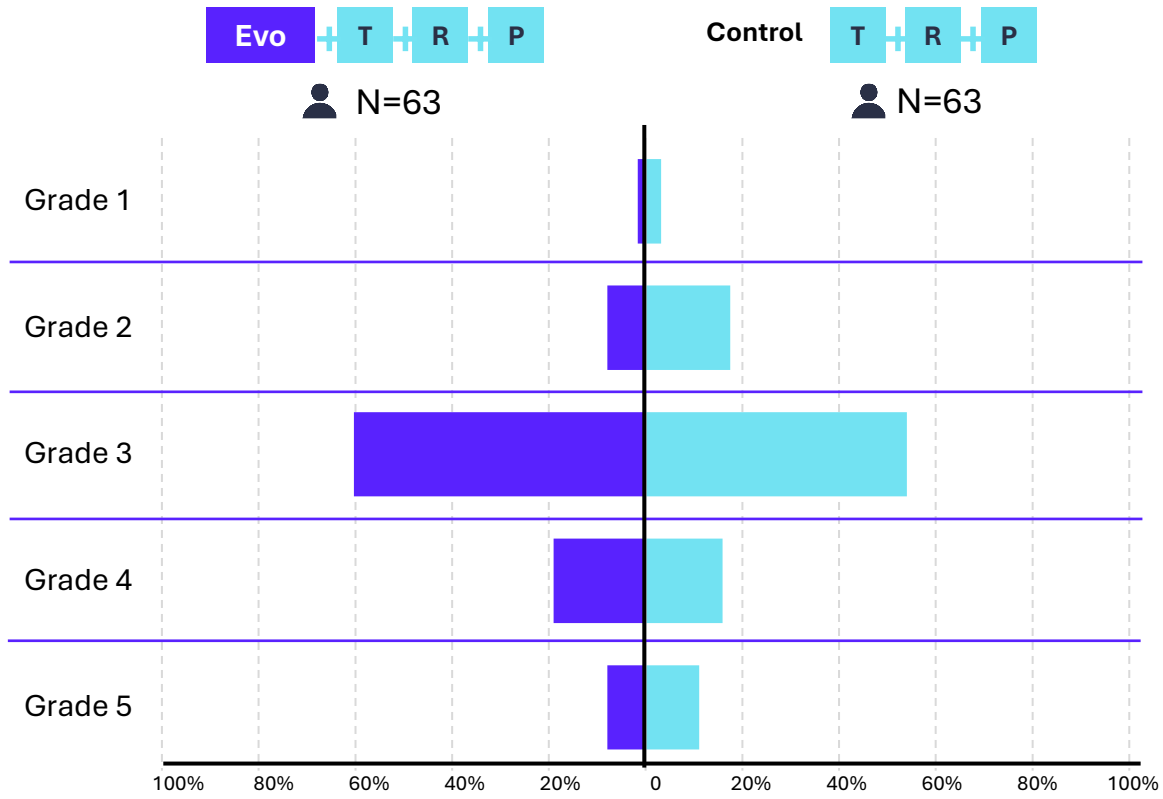
[a]CD47 cut-off defined by cell staining intensity: Medium = IHC2+; High = IHC3+.

[b]The HR is estimated from a Cox proportional hazards model with the treatment, region=Asia (Yes/No) and the use of prior T-DXd (Yes/No) as covariates

Wainberg et al., The 2025 Society for Immunotherapy of Cancer (SITC) Annual Meeting, November 5–9, 2025. Abstract #496. Note: Above results are based on pre-planned exploratory analysis of ASPEN-06 gastric study. Retained HER2+ based on fresh biopsy or ctDNA amplification; Data Cutoff as of May 15, 2025. **T** = trastuzumab; **R** = ramucirumab; **P** = paclitaxel.

Evorpacept Demonstrated a Manageable Safety Profile in ASPEN-06 and Consistently Across Trials

All causality adverse events, by grade



- The incidence of adverse events due to any cause was comparable by arm
- There were 12 patients with Grade 5 treatment emergent adverse events (5 for ETRP; 7 for TRP), only three of which were deemed to be treatment related: esophageal perforation and acute respiratory failure (ETRP), and pneumopathy (TRP). Neither event in ETRP was attributable to evorpacept.

Evorpacept has been studied in >750 patients treated to date, with the overall safety profile being characterized by generally manageable and reversible adverse events

Evo Evorpacept T Trastuzumab R Ramucirumab P Paclitaxel

All G5 TEAEs: ETRP (N=5): sepsis N=2, esophageal perforation N=1, respiratory failure N=1, acute respiratory failure N=1. TRP (N=7): sepsis N=1, pneumonia/pneumopathy/respiratory infection N=1 each, sudden death N=1, death from unknown cause N=1, esophageal hemorrhage N=1; data cutoff as of 15May 2025
Two G5 TEAEs due to disease progression were not included for ETRP

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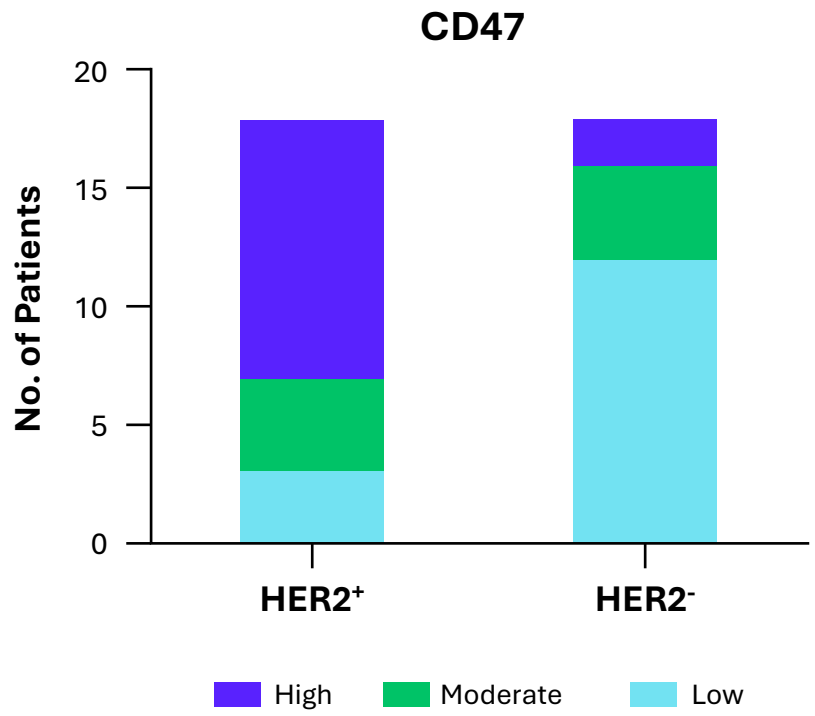
EVORPACEPT

Program Overview

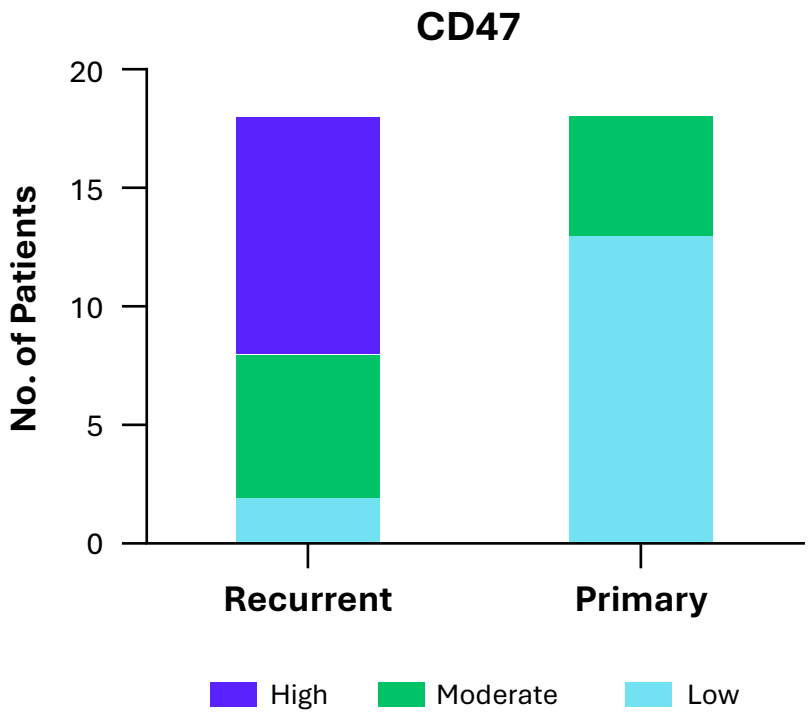
HER2+ Breast Cancer

CD47 Expression in Breast Cancer is Higher in HER2+ Disease, More Common in Resistant Cancer

CD47 Expression is Higher on HER2+ BC Cells vs HER2- and...



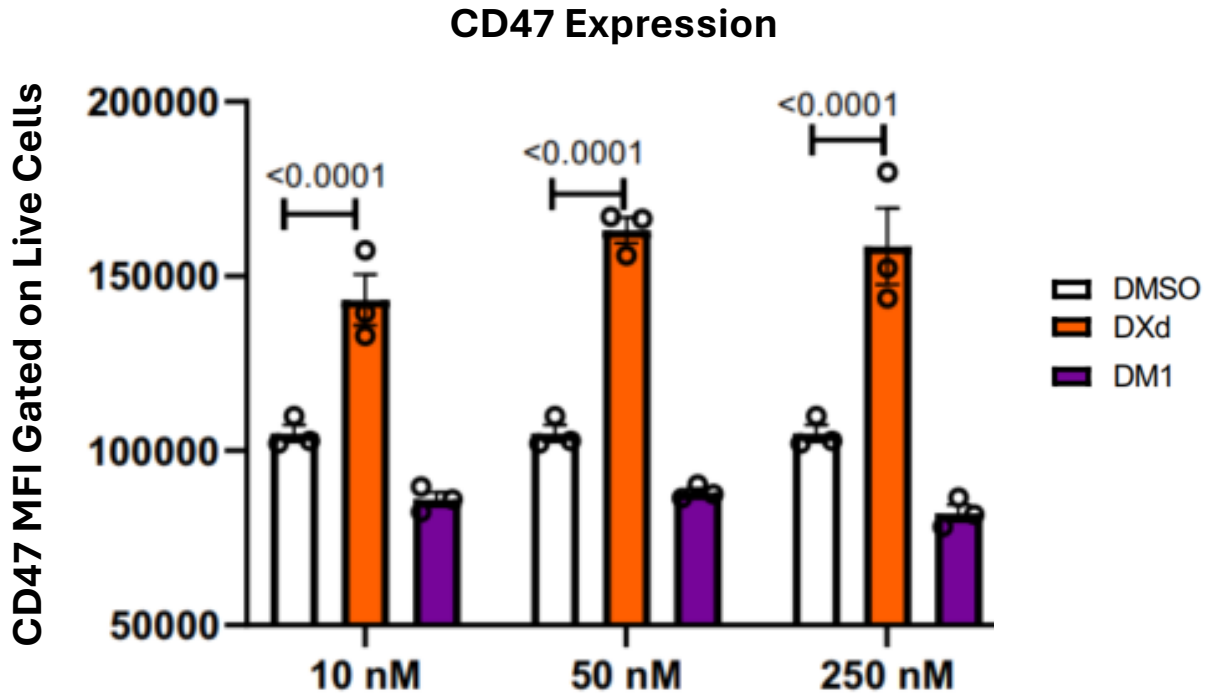
... CD47-High Cells are More Common in Recurrent HER2+ BC



Candas-green, et al, *Nature Communications* 2020

CD47 is Upregulated in Response to T-DXd (ENHERTU) Treatment in HER2-Positive Breast Cancer Cell Lines

T-DXd (Enhertu) Exposure Increases CD47 Expression



Flow cytometry assessment of surface CD47 expression on Au565 cells after 2 days of treatment with Enhertu's payload (DXd) or Kadcyla's payload (DM1) as compared to control (DMSO)

- Per Tsao, Nature Communications, 2025 : "Interestingly, we found that DXd treatment raised surface CD47 levels in HER2 + BC cells."
- Provides validation that CD47 is a key mode of Enhertu evasion and resistance in HER2+ breast cancer patients

Tsao, Nature Communications, 2025

ASPEN-09 Phase 2: Evorpacept with Trastuzumab and Chemotherapy in HER2+ Breast Cancer - Currently Enrolling

Key Eligibility Criteria

- HER2+ mBC (IHC3+ or IHC2+/ISH+)
- Measurable disease per RECIST 1.1
- Prior ENHERTU (trastuzumab deruxtecan; T-DXd)
- All approved treatments are allowed post T-DXd therapy
- ECOG 0-1

N=120

Evorpacept

\+ Trastuzumab

\+ Physician's Choice Chemo[1]

Key Objectives

Primary

- ORR in CD47+ subpopulation

Secondary

- Efficacy in CD47+ sub-population by HER2 ctDNA status
- CBR, DOR, PFS, OS, and safety

Exploratory

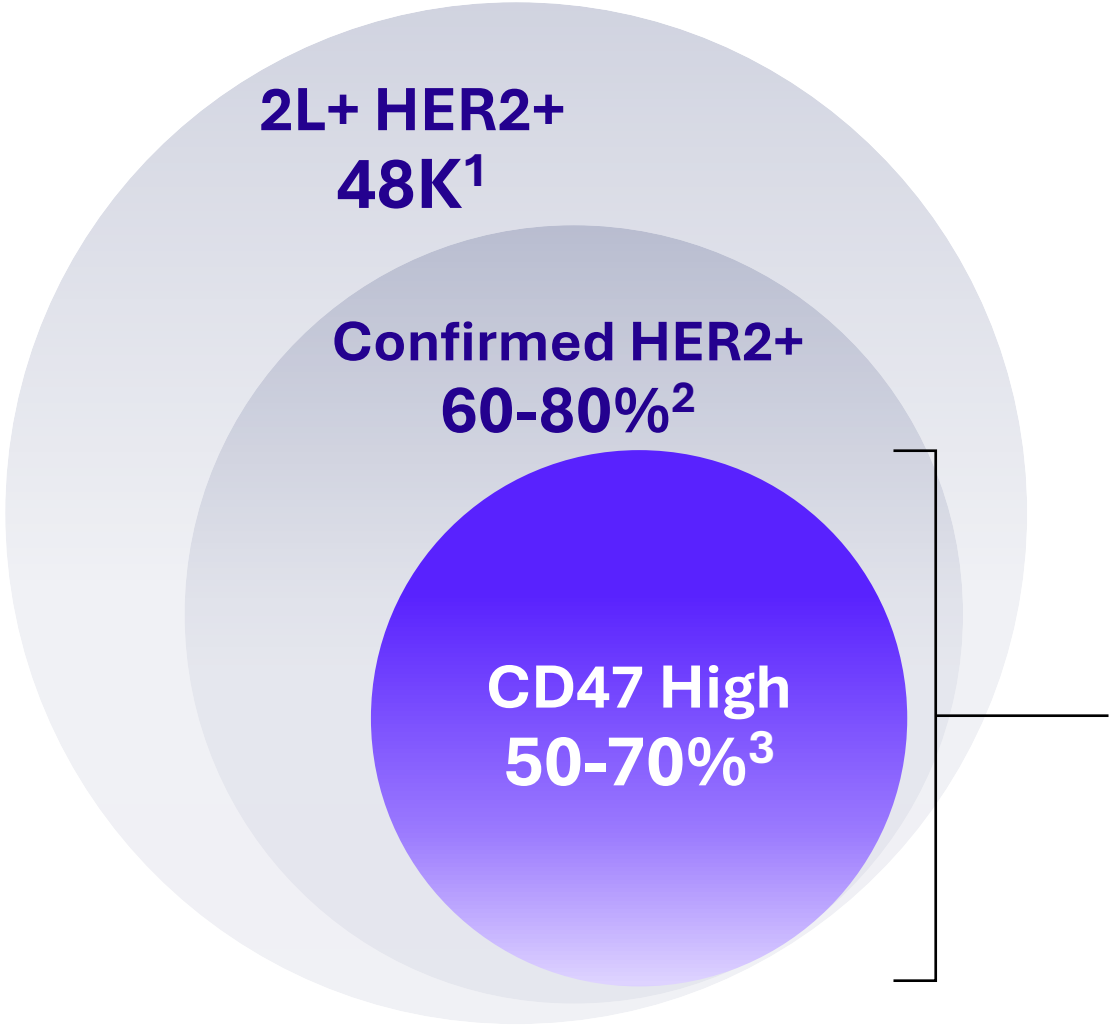
- Efficacy by CD47-status and ctDNA status

- Inclusion of both CD47-high and CD47-low patients enables evaluation of the value of CD47 as a biomarker for evorpacept and will inform the design of a registrational study

Topline data for 120 patients anticipated 2H-2027

#NCT07007559; 1. Capecitabine, eribulin, gemcitabine, paclitaxel, or vinorelbine

HER2+ and CD47-High 2L+ BC Represents a Significant Initial Commercial Opportunity with Potential to Move into Earlier Lines of Therapy



- ~20K addressable patients are CD47-high
- Represents $2-4B market opportunity in CD47-high, HER2+ 2L+ BC[4]

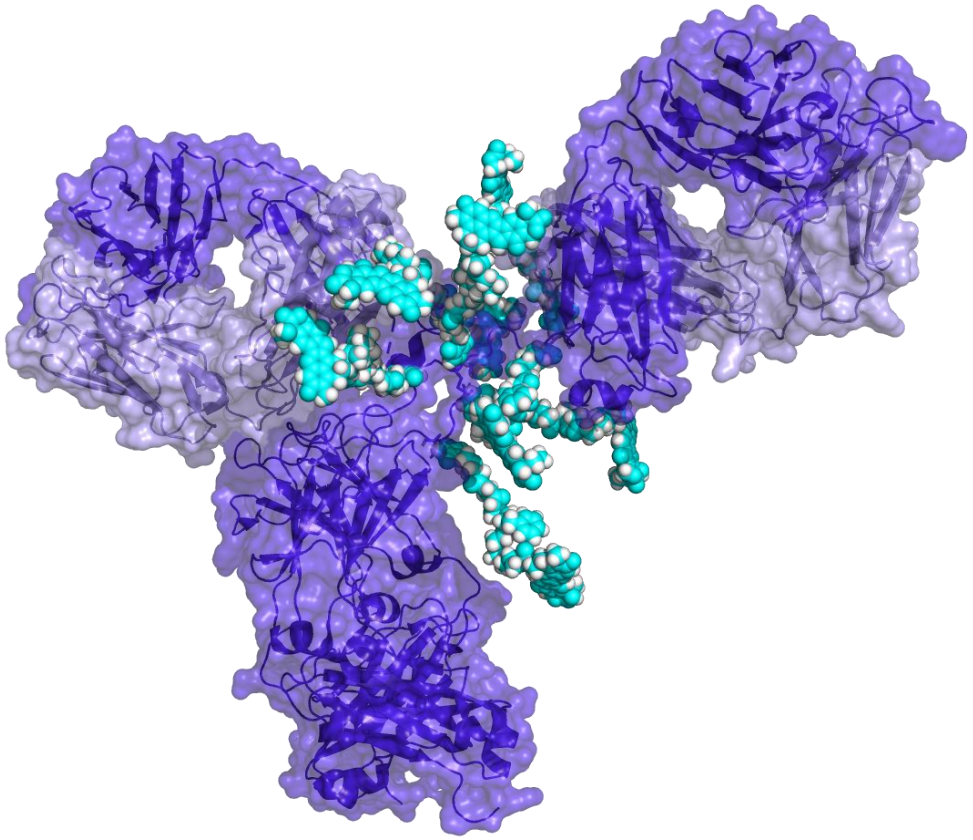
Annual market opportunity based on: 1. US, EU5, JPN addressable patients; ~18k patients in the US; 2. ALX advisory board feedback on breast cancer trial; 3. ALX analysis of Alhanafy, 2024; Sun, 2022; Kosaka, 2021; Chen, 2022; Yuan, 2019 and Tsao, 2025; 4. Monthly price estimate is based on benchmarks in US and extrapolated to core markets.

ALX

ALX2004

EGFR ADC

ALX2004 was Designed to Maximize the Therapeutic Window and Has the Potential to Establish Proof-of-Concept Early in Development Cycle



ALX2004

EGFR-targeted ADC
DAR 8 topoisomerase I
Inhibitor payload (Top1i)

EGFR Antibody

Matuzumab-derived EGFR antibody selected to minimize off-tumor skin toxicity and to maximize therapeutic window

Epitope distinct from that of FDA-approved EGFR antibodies

Proprietary Linker-Payload

Lysosomal cleavage like deruxtecan ADCs with improved linker-antibody stability to minimize off-tumor payload release

Proprietary top1i Payload, DAR 8

Top1i with similar direct cytotoxic potency and enhanced bystander activity compared to deruxtecan

Preclinical Data Support Dose Dependent Activity and Differentiated Safety Profile

ANTI-TUMOR ACTIVITY

- **Dose-dependent activity** across a range of tumors, EGFR expression levels, and mutations
- **Potent anti-tumor activity** in clinically relevant xenograft models
- Demonstrated dose-dependent **activity in patient-derived CRC model**

SAFETY

Safety profile in **NHP toxicity studies support clinical development** plans

- Does **not show EGFR-related skin toxicity** at clinically relevant doses
- **No evidence of payload-related ILD** in NHP toxicity studies, potentially due to linker stability

NHP: Non-human primate; ILD: interstitial lung disease; CRC: colorectal cancer

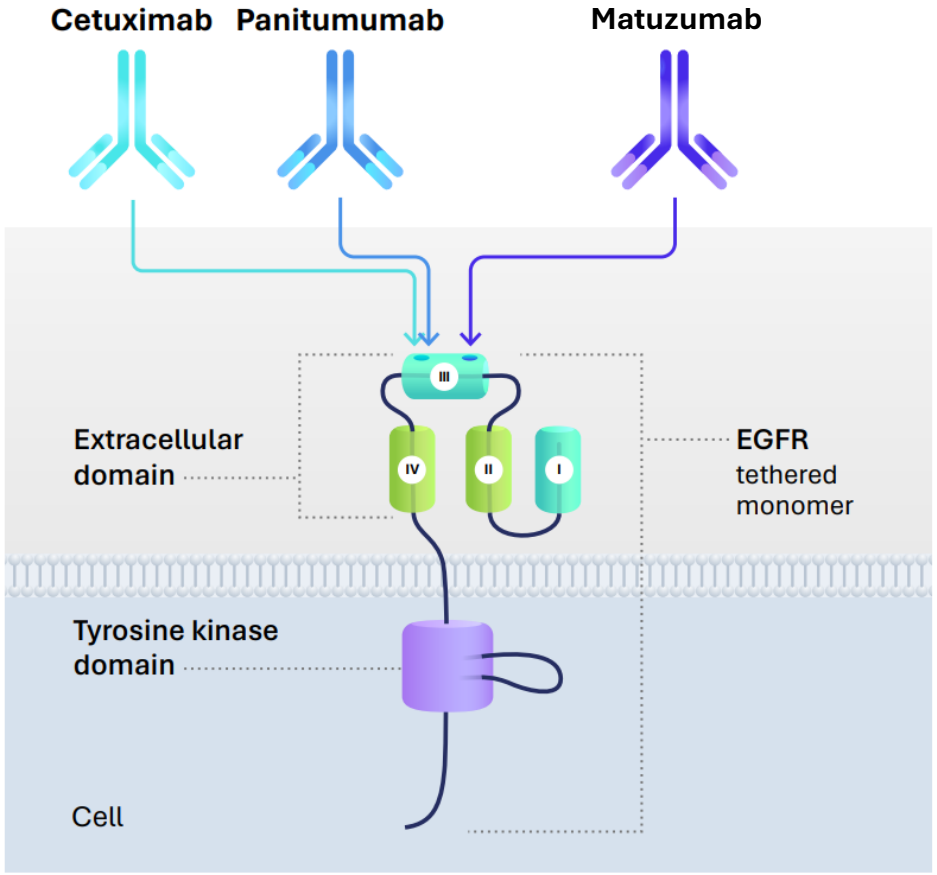
Highly Differentiated Design for ALX2004 Optimizes Validated Payload and Antibody to Maximize the Potential for Success

	ALX2004	EGFR ADCs (monospecific)	EGFR ADCs (bispecific)
Payload tolerability	✓ *Proprietary Top1i payload*	✗ *Mostly MMAE or eribulin (↑ toxic)*	✓ *Majority utilize Top1i payload*
Validated drug target	✓ *Validated EGFR target*	✓ *Validated EGFR target*	✗ *Unvalidated secondary target / combination*
Optimized antibody	✓ *Differentiated epitope and affinity*	✗ *Mostly cetuximab based*	✗ *Bispecific complexity*

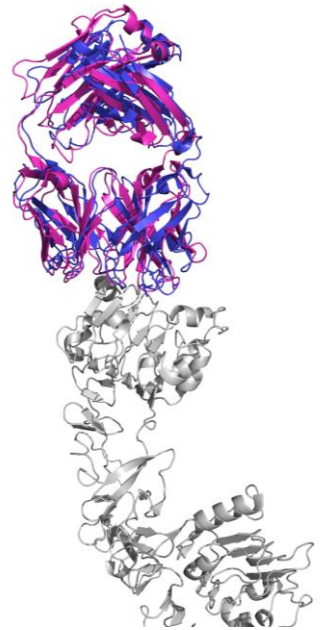
ALX2004 Design Approach

- **Select optimal linker and payload. Top1i most validated and tolerable payload**
- **Use validated targets and drug designs**
- **Maximize therapeutic window through binding epitope and affinity**

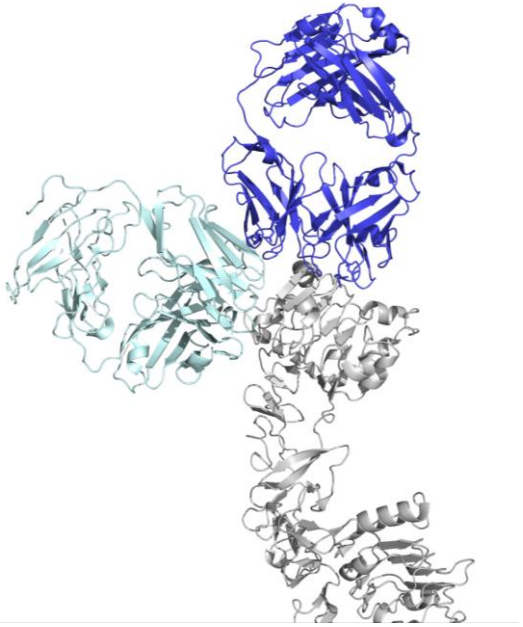
ALX2004's Matuzumab Derived Antibody Binds to a Non-Overlapping EGFR Epitope Compared to Cetuximab and Panitumumab



Blue: Cetuximab
Magenta: Panitumumab
Grey: EGFR domain III



Blue: Cetuximab
Cyan: Matuzumab
Grey: EGFR domain III



Matuzumab vs. cetuximab or panitumumab for an ADC backbone

- Unique binding domain selected for differentiated safety profile
- More likely to be active in Ph1 trials where patients have prior cetuximab or panitumumab exposure

ALX2004 Linker-Payload Designed to Deliver Payloads to Tumors, Not the Periphery

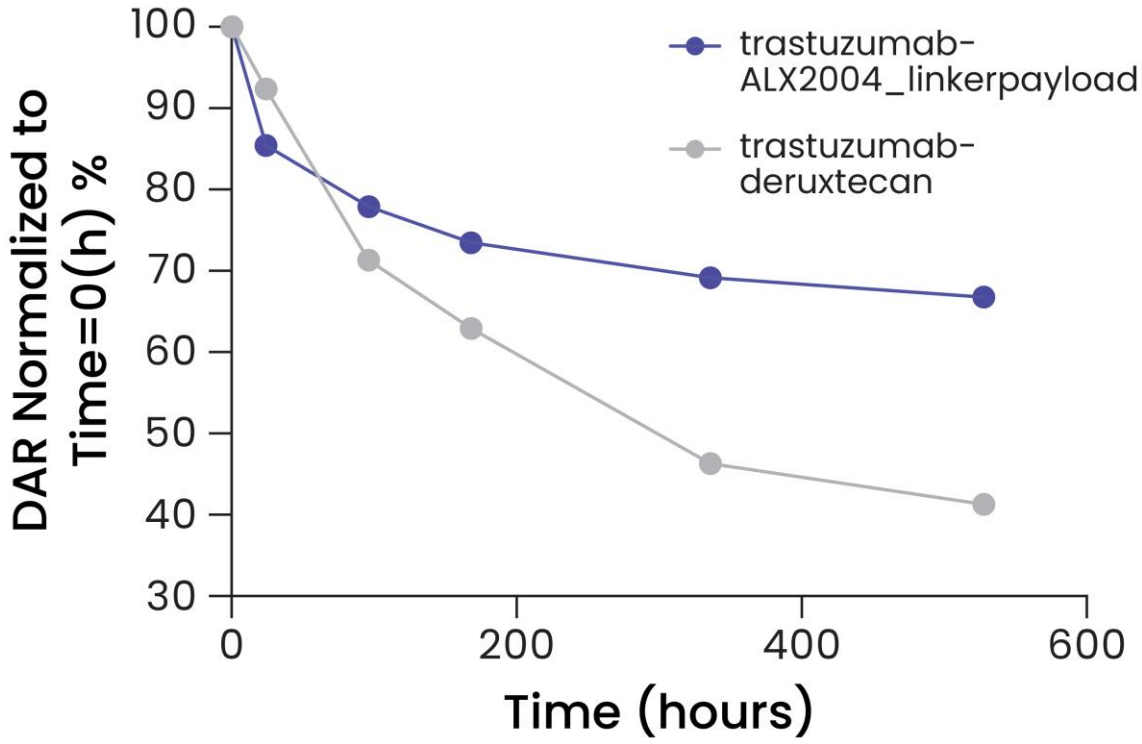
Less drug delivered to off-tumor, off-target tissues

- ALX2004 linker designed with improved stability in circulation to minimize off-tumor linker-payload release

More drug delivered to the tumor

- ALX2004 linker-payload shows improved extracellular stability over industry-standard deruxtecan linker-payload

Analysis of drug-to-antibody ratio over time in NHP model

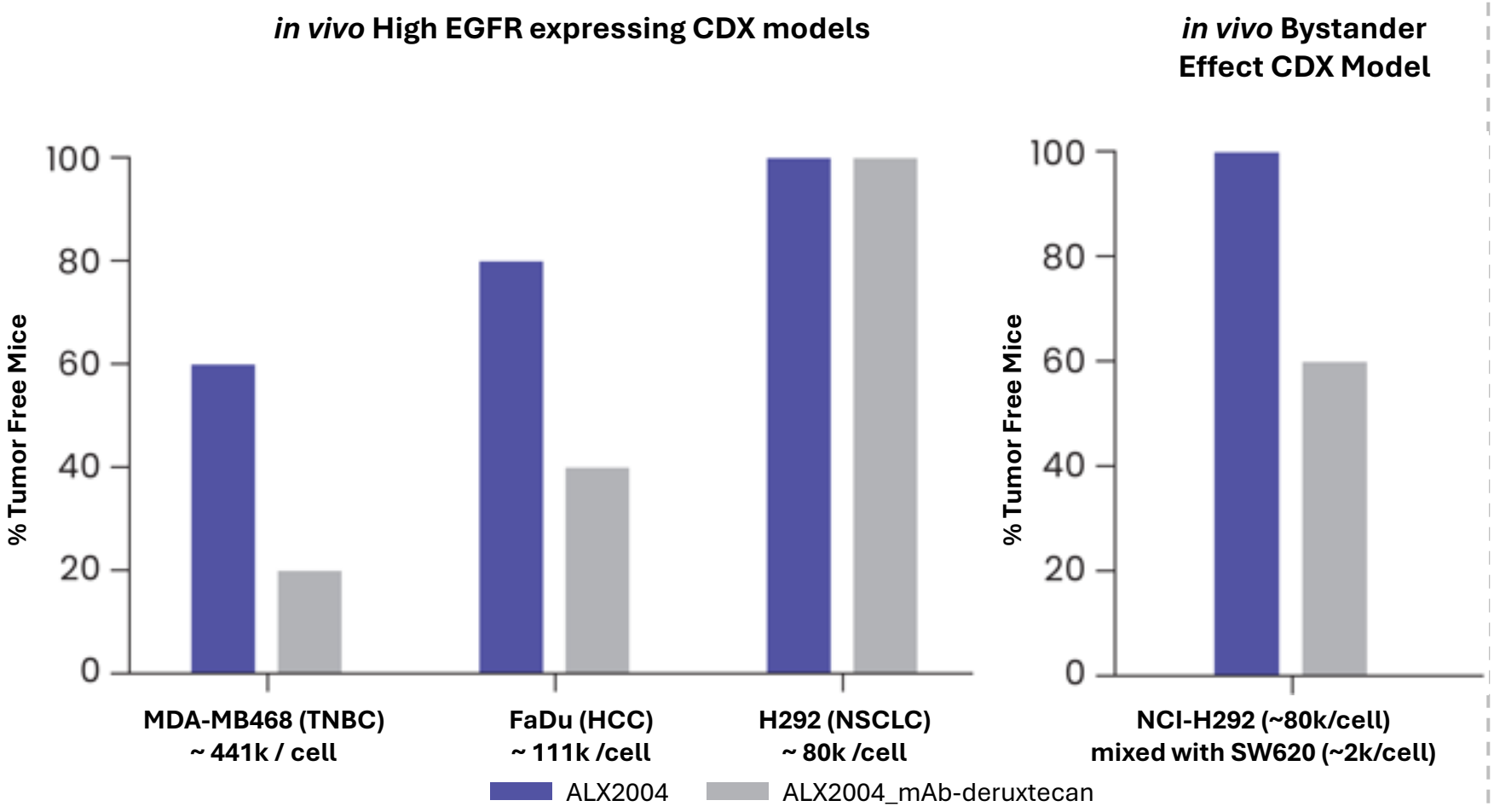


ALX's proprietary linker-payload conjugated to trastuzumab shows improved stability compared to in-house generated trastuzumab-deruxtecan

Wong et al., AACR-NCI-EORTC 2025. Abstract #A119

ALX Proprietary Linker-Payload Shows Superior Activity Compared to Deruxtecan ADCs in CDX Mouse Models

Percent of tumor-free mice in models with varying levels of EGFR expression
(N=5 mice / bar)



- ALX2004 performed as well or better vs. deruxtecan comparator in high-mid EGFR-expressing mouse models
- ALX2004 outperformed deruxtecan comparator in bystander effect model
- Improved bystander effect also demonstrated in cell-based bystander effect assay

Comparator is an in-house generated ADC comprising the ALX2004 antibody conjugated to the deruxtecan linker-payload; Wong et al., AACR-NCI-EORTC 2025. Abstract #A119

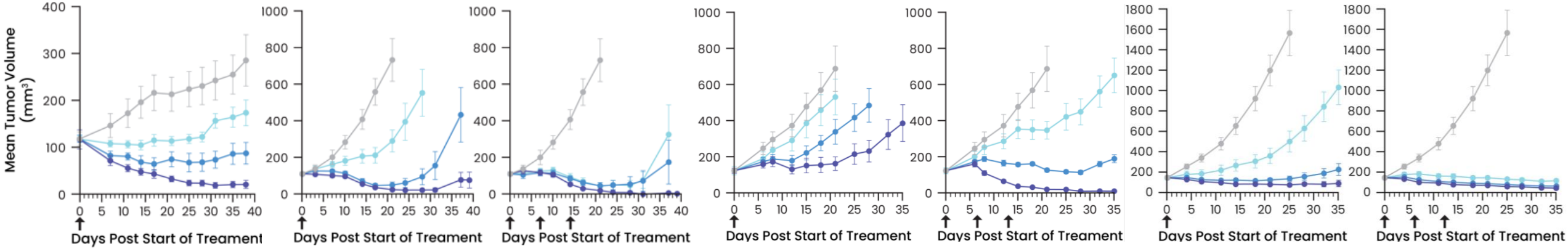
ALX2004 Shows Potent Anti-Tumor Activity Across Multiple Tumor Types, Varying Levels of EGFR Expression and Mutational Status

HCC827 (NSCLC)
EGFRdel19 mt
EGFR: 145,000/cell surface

NCI-H1975 (NSCLC)
EGFR L858R/T790M mt,
EGFR: 50,000/cell surface

COLO205 (CRC)
wt EGFR, BRAF V600E
EGFR: 12,000 /cell surface

HCT116 (CRC)
wt EGFR, KRAS G12D
EGFR: 20,000/cell surface



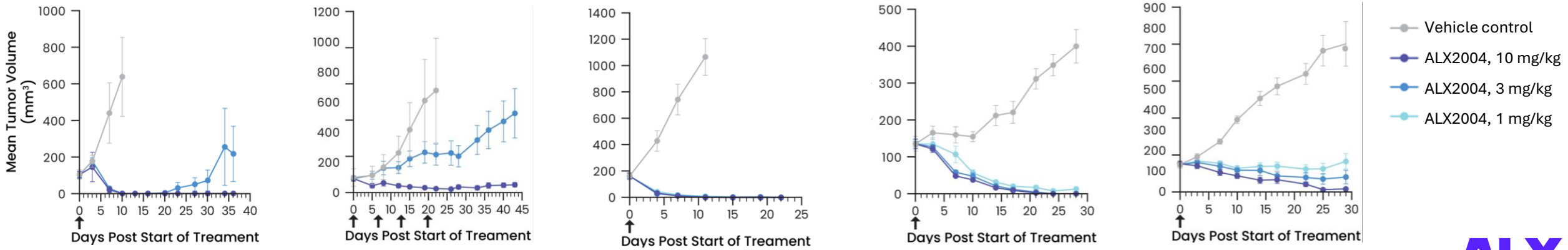
NCI-H292 (NSCLC)
wt EGFR
EGFR: 80,000 / cell surface

A549 (NSCLC)
wt EGFR, KRAS G12S
EGFR: 27,000 / cell surface

FaDu (HNC)
wt EGFR P53 R248L mutation
EGFR: 111,000/cell surface

MDA-MB-468 (TNBC)
wt EGFR, P53 R273C
EGFR: 441,000 EGFR /cell surface

CFPAC-1 (PDAC)
wt EGFR, KRAS G12V
EGFR: 62,000/cell surface



Wong et al., AACR-NCI-EORTC 2025. Abstract #A119

Safety Profile Findings in NHP Toxicity Support Clinical Development Plans

GLP NHP Toxicology Study

Design

6-week repeat dose (Q3W dosing) with 6-week recovery period at 5, 10 and 20 mg/kg

Key Findings

10 mg/kg dose (n=10)
NOAEL (*No Observed Adverse Effect Level)*

20 mg/kg dose (n=10)
HNSTD (*Highest Non Severely Toxic Dose*)

- All findings are minimal to moderate and fully recoverable
- No dose limiting major target organ toxicity, including on-target toxicity (i.e. skin or other EGFR expressing cells)
- No evidence of ILD

Wong et al., AACR-NCI-EORTC 2025. Abstract #A119; NHP = non-human primate

Phase 1 Clinical Development Plan in EGFR-Expressing Tumors

Safety and Early Efficacy from Dose Escalation in December 2026

Dose Finding (Phase 1a) ALX2004 IV Q3W in NSCLC, HNSCC, CRC, and ESCC

Dose Escalation

BOIN Dose Escalation

Additional Dose Levels Ongoing
≥4 mg/kg Q3W*

Dose Level 2
2 mg/kg Q3W
n=3

Dose Level 1
1 mg/kg Q3W
n=3

Dose Exploration
(optional)

Explore cohorts at different doses and schedules to identify the RDE(s)

RDE(s)

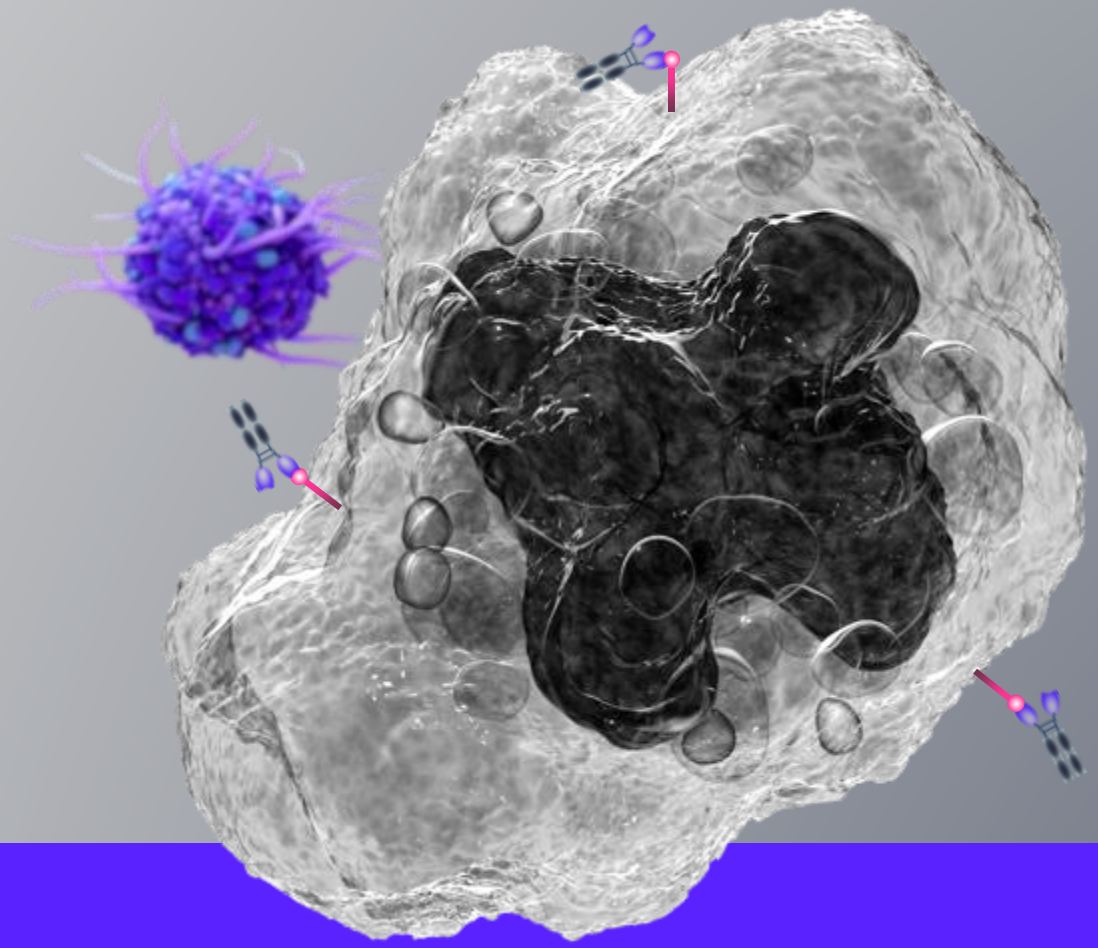
Potential to Expand into Phase 2 Single-Arm Study to Support Accelerated Approval

Dose Expansion (Phase 1b) n = up to 80

1 or 2 tumor types selected from Dose Escalation at up to 2 different doses and/or schedules

For each selected tumor, if 2 doses or schedules are selected, patients will be randomized to the 2 groups

HNSCC: head and neck squamous cell carcinoma; CRC: colorectal cancer; NSCLC: non-small cell lung cancer; ESCC: esophageal squamous cell carcinoma; RDE: recommended dose for expansion; * Additional dose levels and dosing regimens may also be tested.



NASDAQ – ALXO

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ALX is Rapidly Advancing Two Novel Cancer Treatments with Multiple Near-Term Catalysts

1. ALX is focused on **driving toward multiple inflection points in 2026 and 2027** across both programs – evorpacept and ALX2004

2. Evorpacept biomarker strategy validated by data from both the Ph2 ASPEN-06 gastric cancer and the Ph1b/2 breast cancer trials, supporting potential to drive compelling benefit in the ongoing **HER2+ breast cancer trial of evorpacept with trastuzumab and chemo with topline, N=120 data expected in 2H 2027**

3. ALX2004 is a **highly differentiated ADC** in development for EGFR-expressing solid tumors enrolling in a phase 1 trial which is on track with initial safety and efficacy results from approximately 40 patients expected in December 2026

4. **$150M gross proceeds** from February 2026 strengthens balance sheet and is sufficient to fund planned operations through the first half of 2028

Thank You.

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