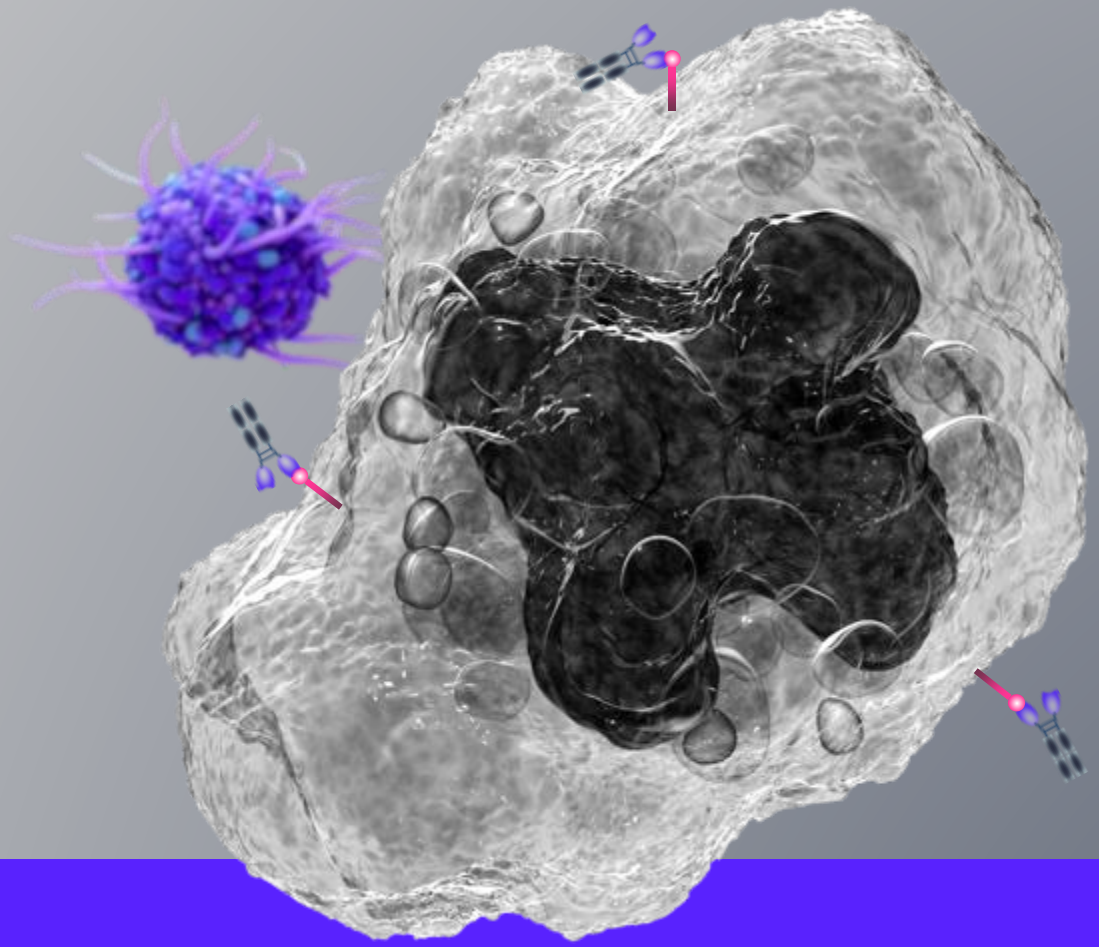


ALX™ ONCOLOGY

JP Morgan Healthcare Conference
January 2026

NASDAQ GS
ALXO

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 future M&A activity and 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.

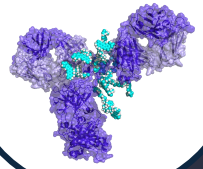
This presentation concerns product candidates that are under clinical investigation, and which have not yet been approved for marketing by the U.S. Food and Drug Administration. These product candidates are currently limited by federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated.

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.

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 now 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*

Strong Execution in 2025 Leads to a Catalyst Rich 2026

- ✓ **ASPEN-06 data at SITC '25: 2L gastric study demonstrated a 65% ORR in treatment vs 26% in control in HER2+ CD47-high patients; mPFS benefit of 18.4m vs 7.0m (HR 0.39), mOS 17.0m v 9.9m (HR 0.63)**

- ✓ **Jazz collaboration: Evo + zanidatamab demonstrated a 56% ORR in HER2+ breast cancer patients with prior Enhertu**

- ✓ **Sanofi collaboration: Evo + Sarclisa + dexamethasone in patients with previously treated multiple myeloma advanced from dose escalation into dose optimization**

- ✓ **MDACC NHL data: Evo + R2 demonstrated a 100% ORR, 92% CR rate (historical control is ~50%), and a 1-yr PFS rate of 91% in 1L indolent NHL**

- ✓ **ALX2004 enters clinic and rapidly progresses through initial dose levels with no dose-limiting toxicities**

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-Breast** Evorpacept, Trastuzumab + chemotherapy	ENHERTU®-Experienced HER2-Positive Breast Cancer					**Enrolling, interim analysis anticipated Q3 2026**
	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)					**Completed, established POC**
	Zanidatamab[3] + Evorpacept	HER2-Expressing Breast Cancer and Other Cancers					**Completed, data presented at SABCS '24**
ALX2004 PROGRAM							
EGFR ADC	**ALX2004** Dose-escalation and expansion	EGFR-Expressing Solid Tumors					**Enrolling, Initial safety data 1H 2026**

ALX-sponsored trial

Completed trial

ALX Oncology retains worldwide rights to evorpacept

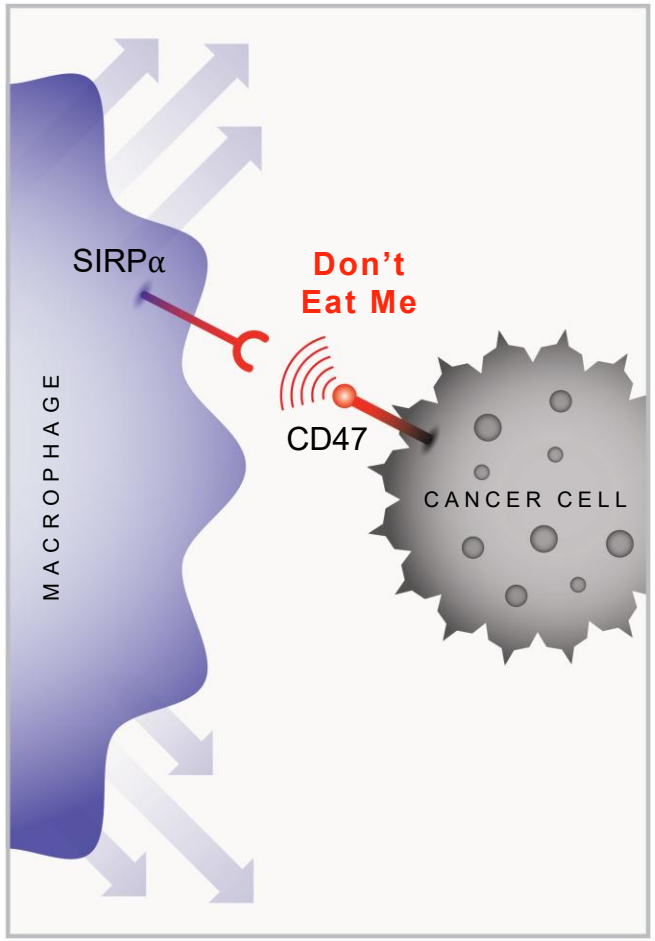
1. Sanofi sponsors SARCLISA® clinical trial. **2.** Lilly supplies CYRAMZA® for ALX Oncology's ASPEN-06 program **3.** Jazz Pharmaceuticals sponsors zanidatamab clinical trial.

ALX

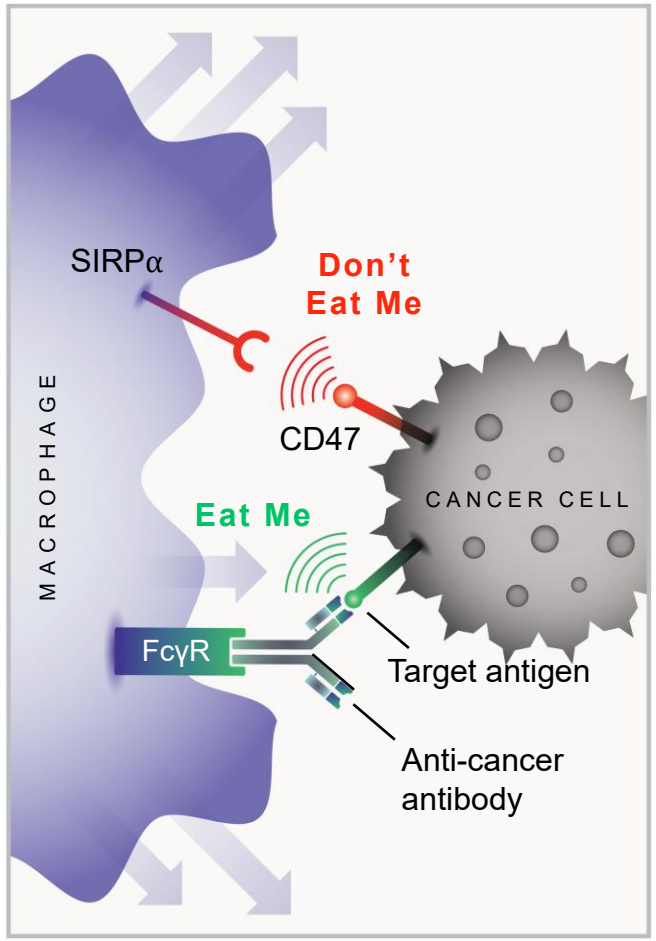
EVORPACEPT

Advancing A Synergistic Approach to Cancer Treatment

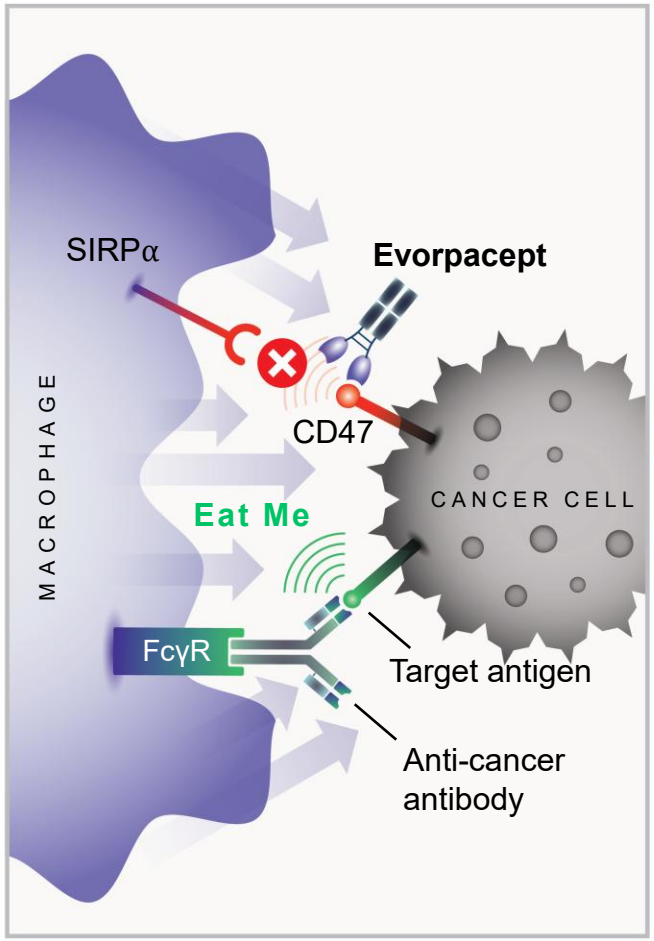
Evorpacept Blocks the CD47-SIRPα Interaction, Enhancing the Targeted ADCP of Cancer Cells when Given in Combination with Anti-Cancer Antibodies



Cancer cells overexpress CD47 in order to evade immune detection



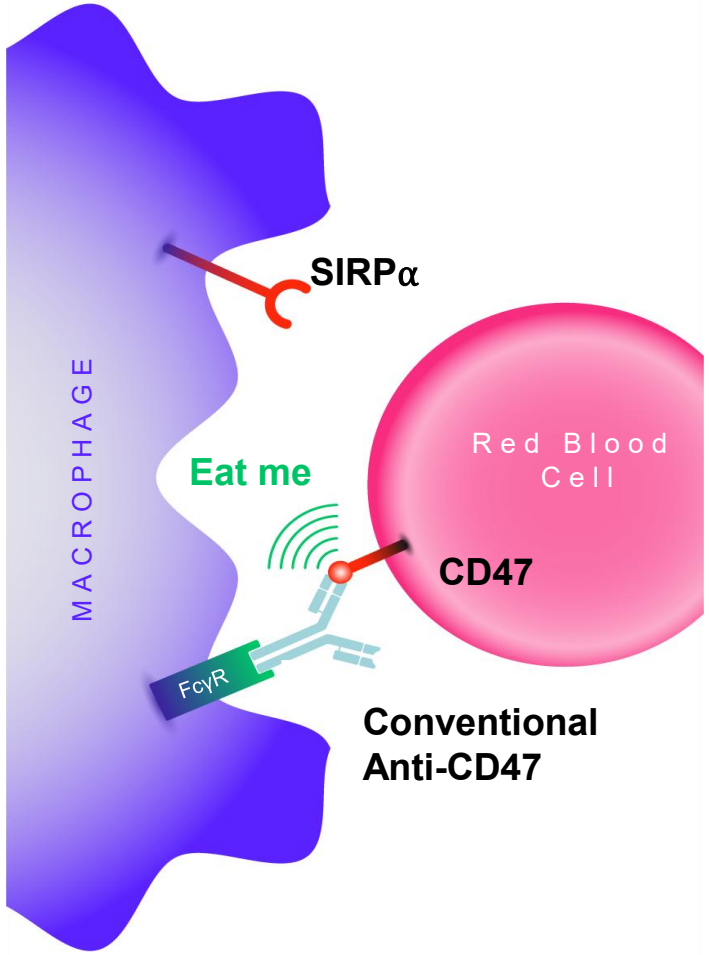
ADCP of anti-cancer antibodies is inhibited by CD47



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

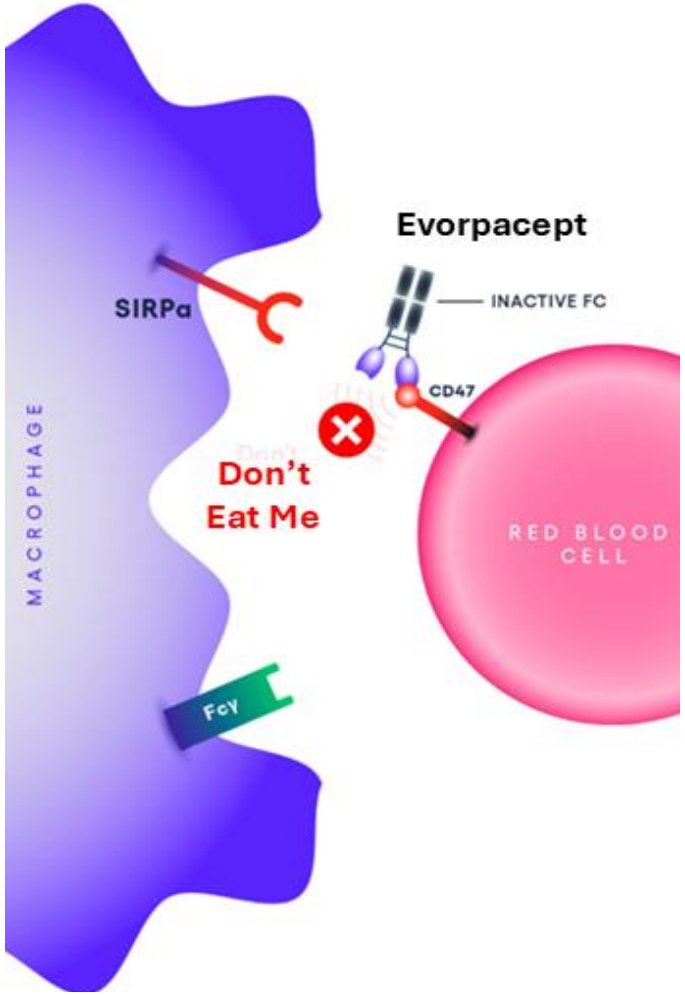
Evorpacept Is the Only CD47 Blocker with an Inactive Fc Designed to Avoid Toxicities Seen with Conventional Anti-CD47

Conventional anti-CD47 with active Fc



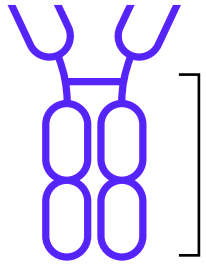
Due to CD47's expression on red blood cells, this caused on-target, off-tumor toxicities

Evorpacept with inactive Fc



Inactive Fc spares normal cells minimizing toxicity

Evorpacept's MOA with Anti-Cancer Antibodies has Demonstrated Consistent Tolerability and Robust Clinical Activity vs. Conventional Approaches



Inactive Fc domain

Evorpacept

- **evorpacept + Herceptin** — Positive randomized Ph2 in gastric cancer
- **evorpacept + Herceptin** — Positive ph1b in gastric cancer
- **evorpacept + Zanidatamab** — Positive ph1b in breast cancer
- **evorpacept + Rituxan** — Positive ph1b in 1L/ 2L NHL

Evo is the only Fc-inactive clinical-stage program and has shown consistent activity and tolerability

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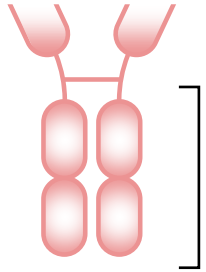


Active Fc domain

Conventional CD47 Blockers

- **Lemzo-parlimab** — Hematologic toxicity signal
- **TTI-622** — Hematologic toxicity signal
- **TTI-621** — Hematologic toxicity signal
- **Magrolimab** — Hematologic toxicity signal

Clinical trials of Fc-active programs have been mostly discontinued/ deprioritized

ALX

EVORPACEPT

Clinical data with anti-cancer antibodies

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"[1]**

Increased CD47 expression is correlated with poor patient outcomes in many tumor types including[2]:

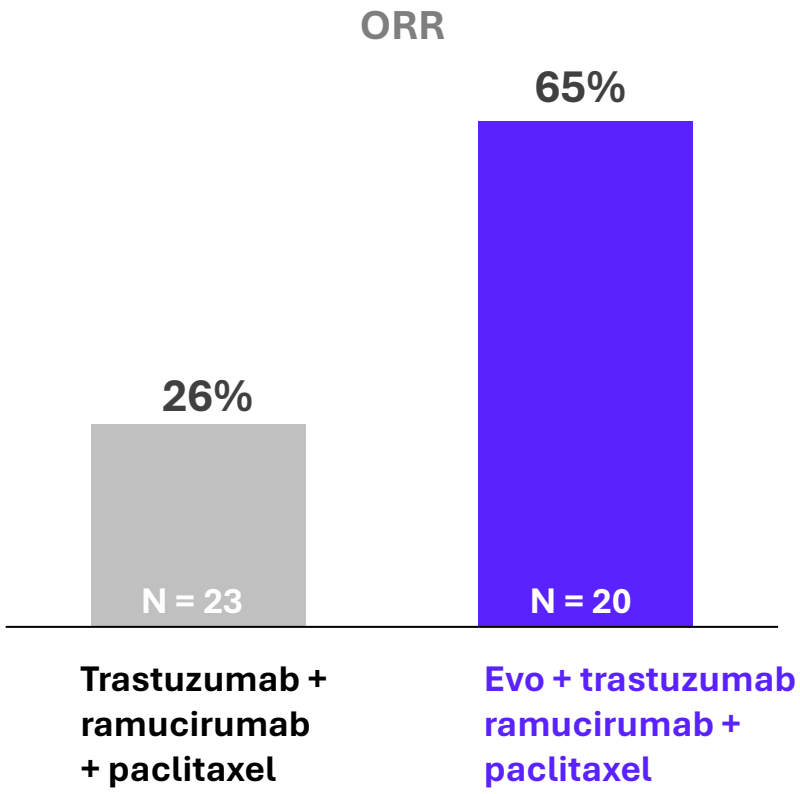
- Breast cancer[3, 14]
- Oral squamous cell carcinoma[4]
- Nasopharyngeal carcinoma[5]
- Ovarian cancer[6]
- Non-small cell lung cancer[7]
- Clear cell renal cell carcinoma[8]
- Hepatocellular carcinoma[9]
- Gastric adenocarcinoma[10]
- Colorectal adenocarcinoma[11]
- Head and neck squamous cell carcinoma[12]
- Multiple myeloma[13]

1Yang et al, *Translational Cancer Research*, 2018**;** 2) Huang, et al, *Scientific Reports*, 2022; 3)Yuan, et al, *Oncol* Lett, 2019 ; 4) Pai, et al, Cells, 2019; 5) Wang, et al, *OncoTargets & Ther.* 2020;; 6) Li, et al, *Am J Trans Res*, 2017; 7) Barrera, et al, *Br J Cancer,* 2017; 8) Jiang, et al, *Urol Oncol*, 2022; 9) Kim, et al, *J Clin Pathol*, 2021;10) Shi, et al, *Cancer Imm, Imm*, 2021; 11) Kim, et al, *Diagnostics*, 2021; 12) Wu, et al, *Oncoimmunology*, 2018; 13) Rastgoo, et al, *Haematologica,* 2020; 14) Chen, et al, J Pathol Clin Res 2023

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

HER2+ Gastric Cancer

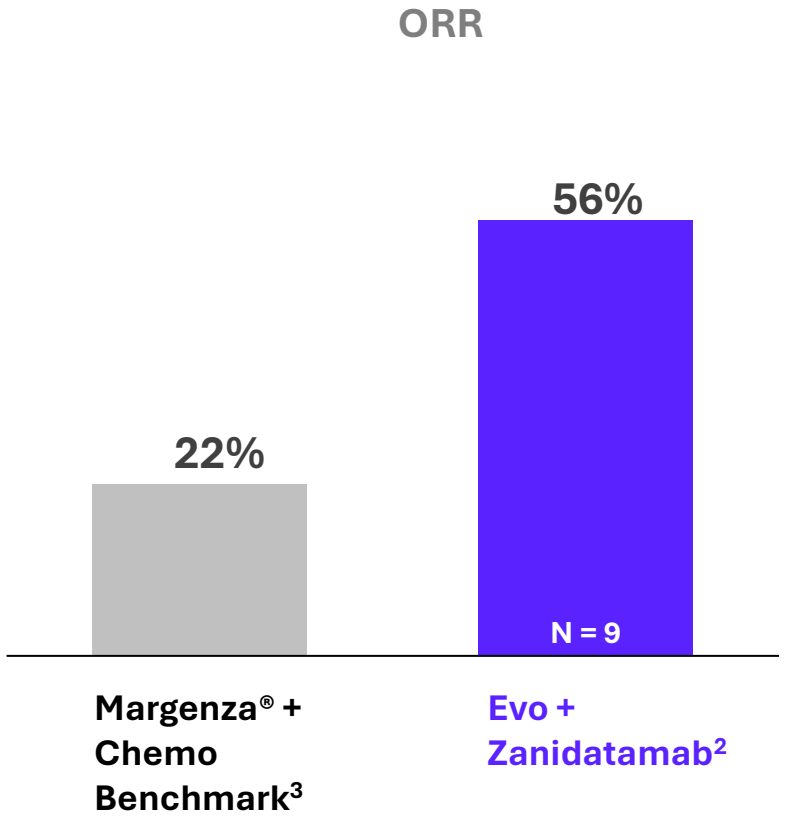
(CD47-high expression and retained HER2+[1])



ASPEN-06 subset, randomized Ph2

HER2+ Breast Cancer

(R/R HER2+ by central assessment [2])



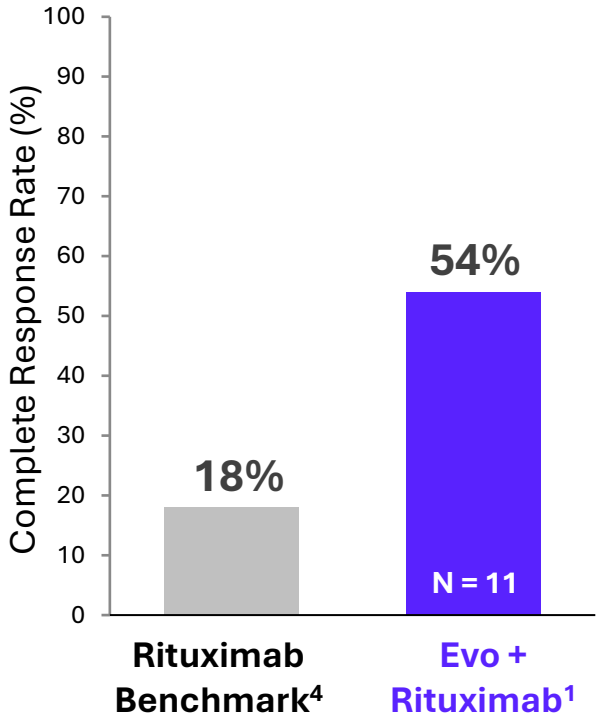
Phase 1b/2 subset – Collaboration and historic benchmark

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. SABCS 2024 #PS8-09; HER2+ by central assessment; 3. Margenza prescribing information;
ORR = overall response rate; IST = investigator-sponsored trial

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

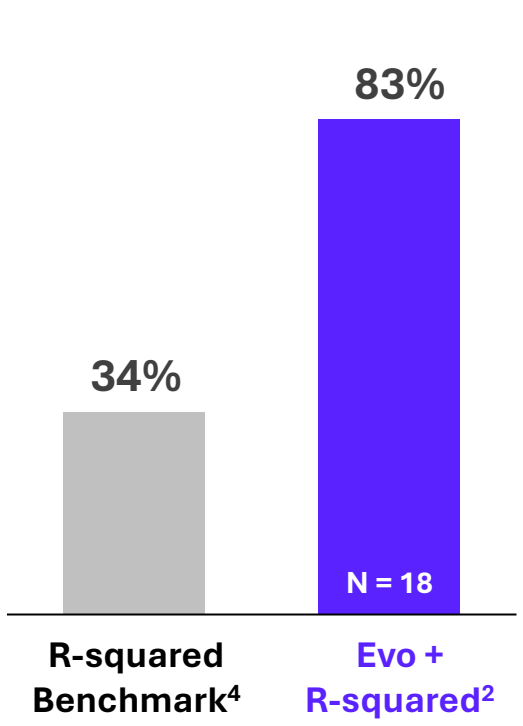
Evo + Rituximab in R/R indolent NHL[1]

(ALX-sponsored ASPEN-01 Ph1B)



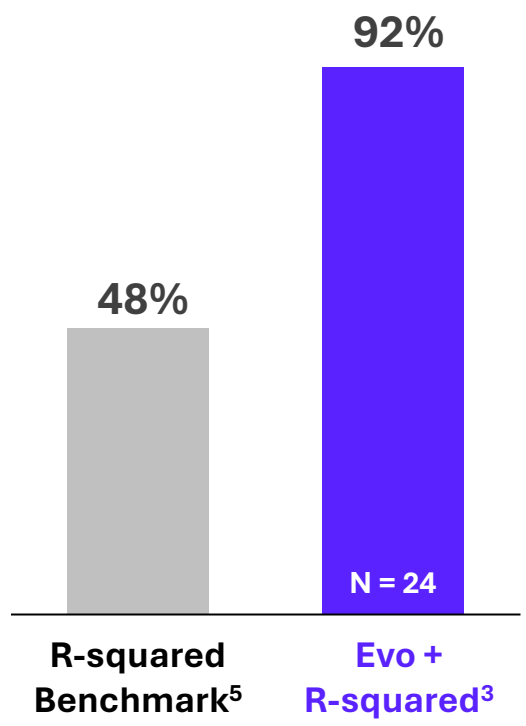
Evo + R-squared in 2L indolent NHL[2]

(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

ASPEN-06 Phase 2: Evorpacept Plus Trastuzumab + Ramucirumab + Paclitaxel (TRP) in HER2+ Advanced/Metastatic GC/GEJ Adenocarcinoma

ASPEN-06 Trial Design

Key eligibility criteria

- HER2+ GC or GEJ that has progressed on or after prior HER2-directed therapy (e.g. trastuzumab)
- 2L or 3L
- Prior trastuzumab deruxtecan (ENHERTU) and/or checkpoint inhibitors allowed
- Prior CD47-agent, anti-SIRPα, or ramucirumab excluded
- Patients enrolled with either a HER2+ fresh or archival biopsy

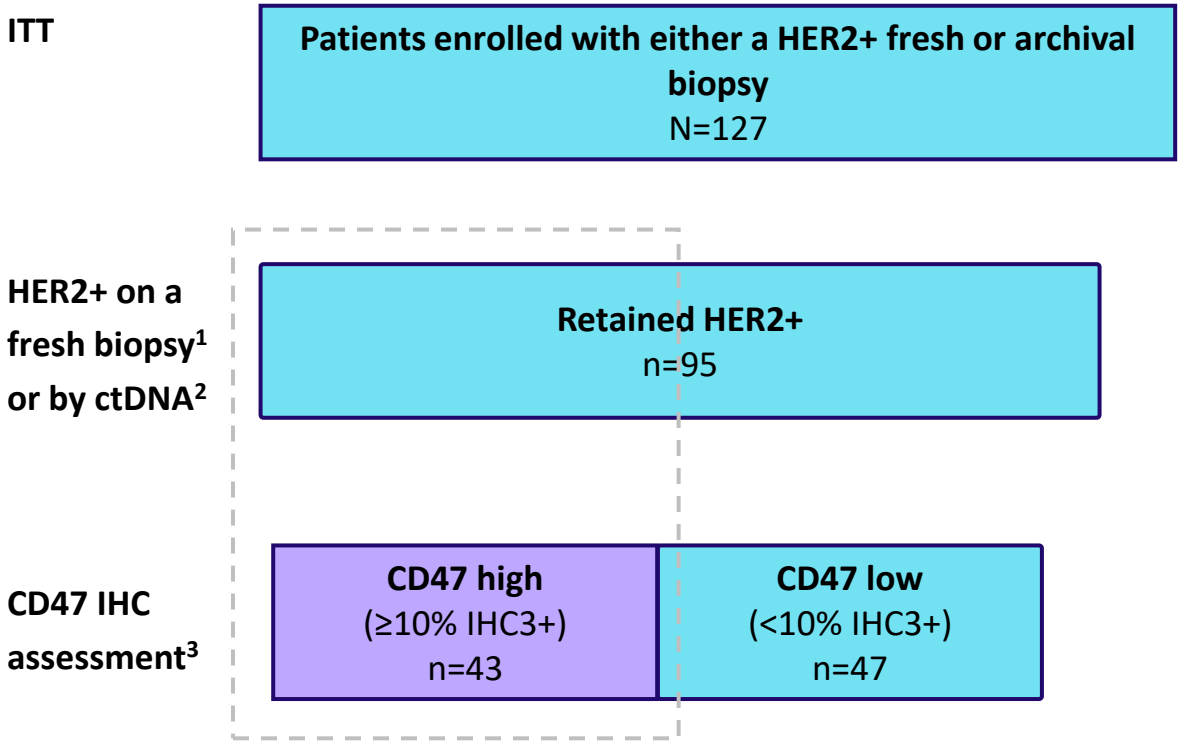
1:1

Evorpacept + T + R + P

VS.

Control T + R + P

Study Flow Diagram



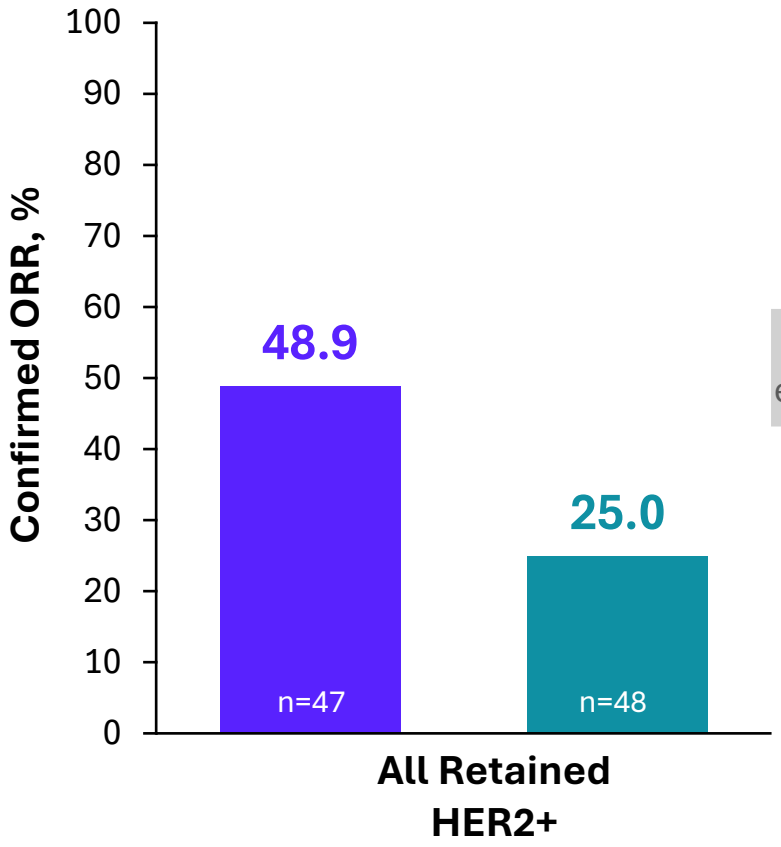
43 patients had retained HER2+ and were CD47-high

Evo Evorpacept (30 mg/kg Q2W) T Trastuzumab (6 mg/kg > 4 mg/kg Q2W) R Ramucirumab (8 mg/kg Q2W) P Paclitaxel (80 mg/m^2 on day 1, 8, 15 of 28-day cycle)

GC- gastric cancer, GEJ- gastroesophageal junction, TRP- trastuzumab, ramucirumab, paclitaxel. Retained HER2+: (1) Fresh HER2-positive is defined as biopsies that were HER2-positive after receiving prior HER2-targeted treatment, and (2) HER2 (ERBB2) plasma gene amplification from Guardant360® analysis; (3) 6 patients with confirmed HER2+ had missing CD47 samples or non-evaluable samples

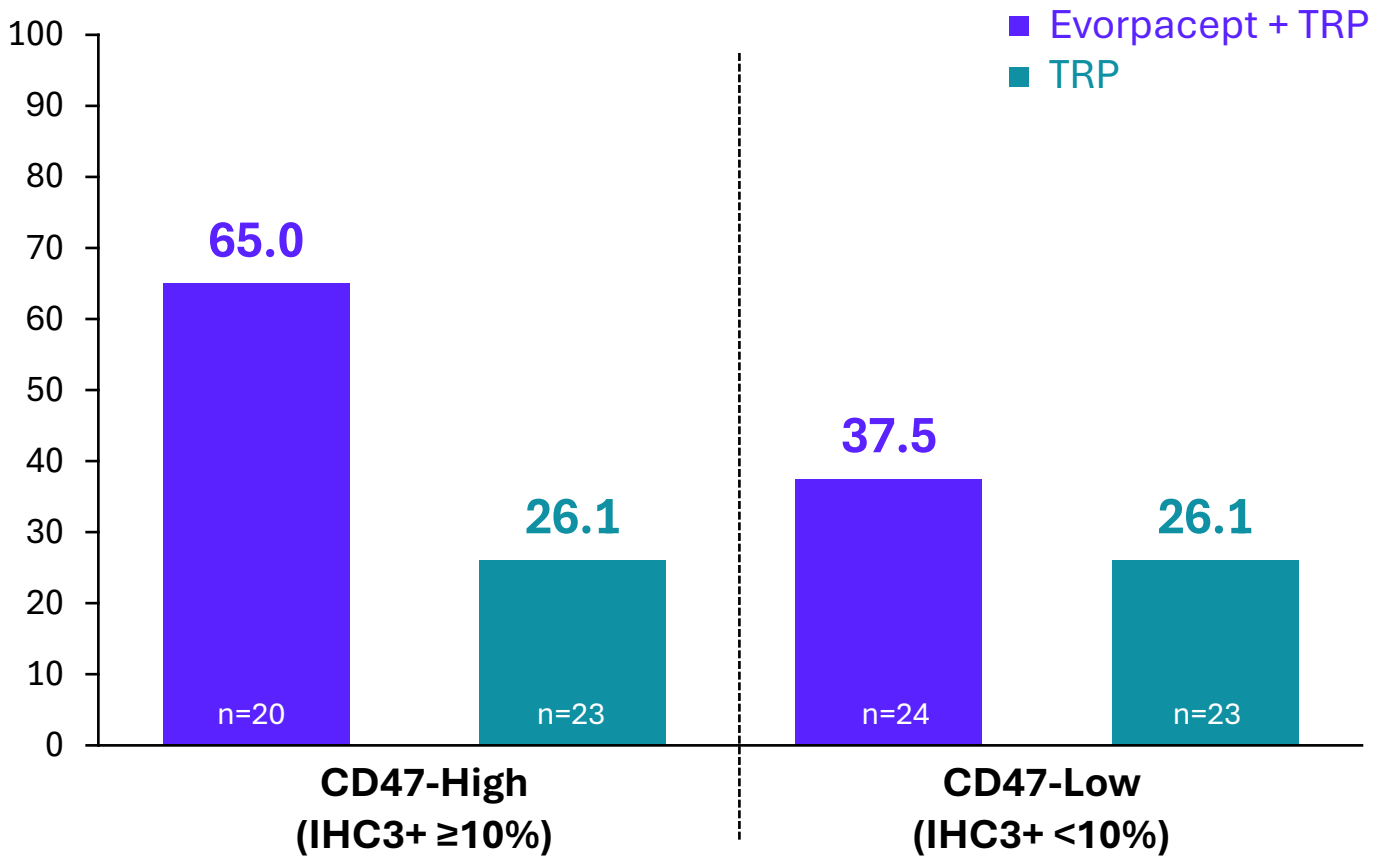
CD47 Expression Acts as a Predictive Biomarker for Durable Patient Benefit from Evorpacept in the ASPEN-06 Trial

ORR in retained HER2+ (n=95)



By CD47 expression

ORR by CD47 expression level in retained HER2+



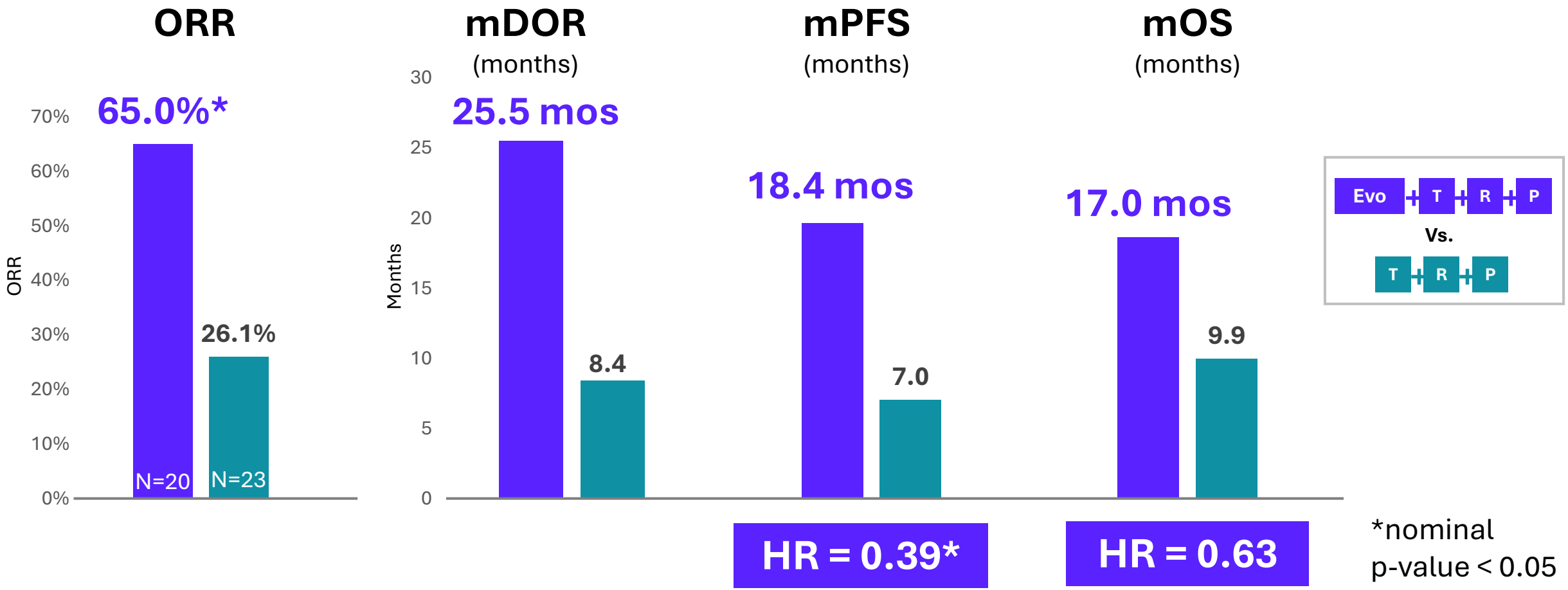
Wainberg et al., The 2025 Society for Immunotherapy of Cancer (SITC) Annual Meeting, November 5–9, 2025. Abstract #496. Note: Above results are based on pre-planned exploratory analysis of ASPEN-06 gastric study. Retained HER2+ based on fresh biopsy or ctDNA amplification. Data Cutoff as of May 15, 2025.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

Potential for Evorpacept to Drive Transformational Benefit Across All Key Efficacy Parameters in Patients with High CD47 Expression

ASPEN-06 Gastric / GEJ Trial

Patients with CD47-High Expression and Retained HER2+

(N=43/127)

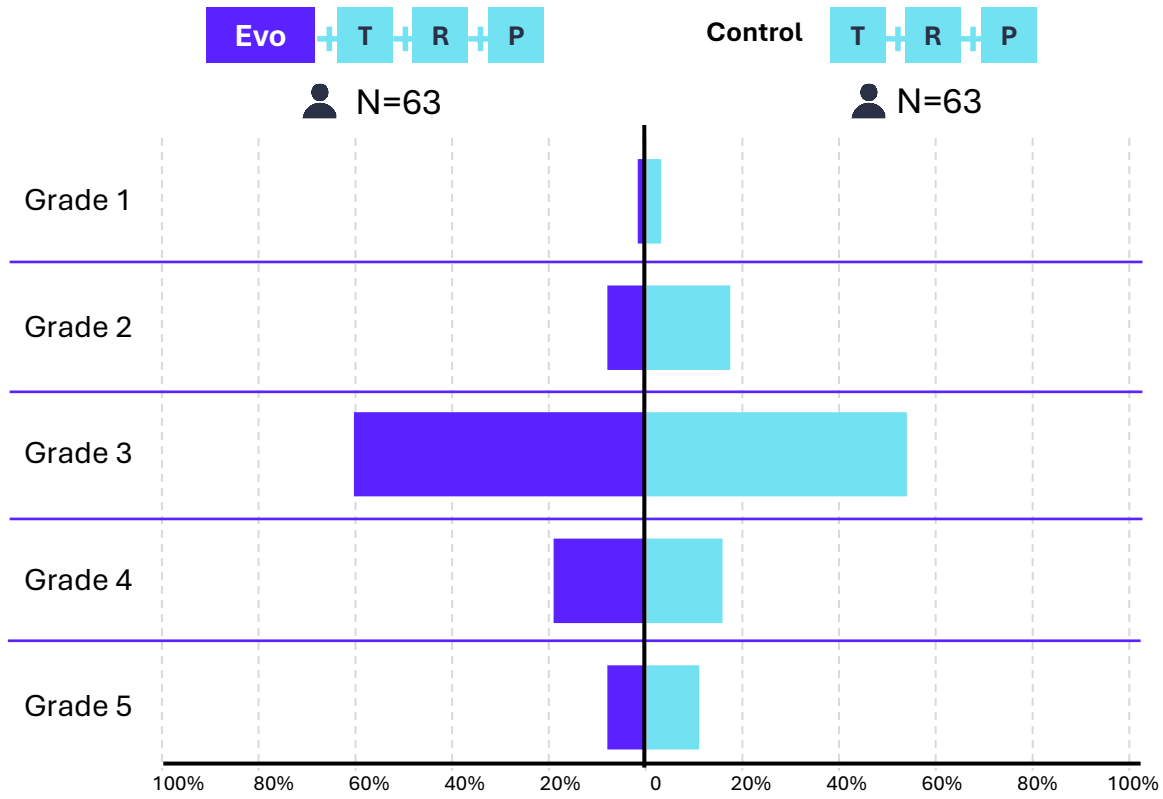


*nominal
p-value < 0.05

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. ORR per investigator. **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

ALX

EVORPACEPT

Breast Cancer

Phase 1b/2 Trial Evaluating Safety and Efficacy of Evorpacept Plus Zanidatamab in Patients Who Have Progressed on Prior HER2-Directed Therapy

Key eligibility criteria: Cohort 1

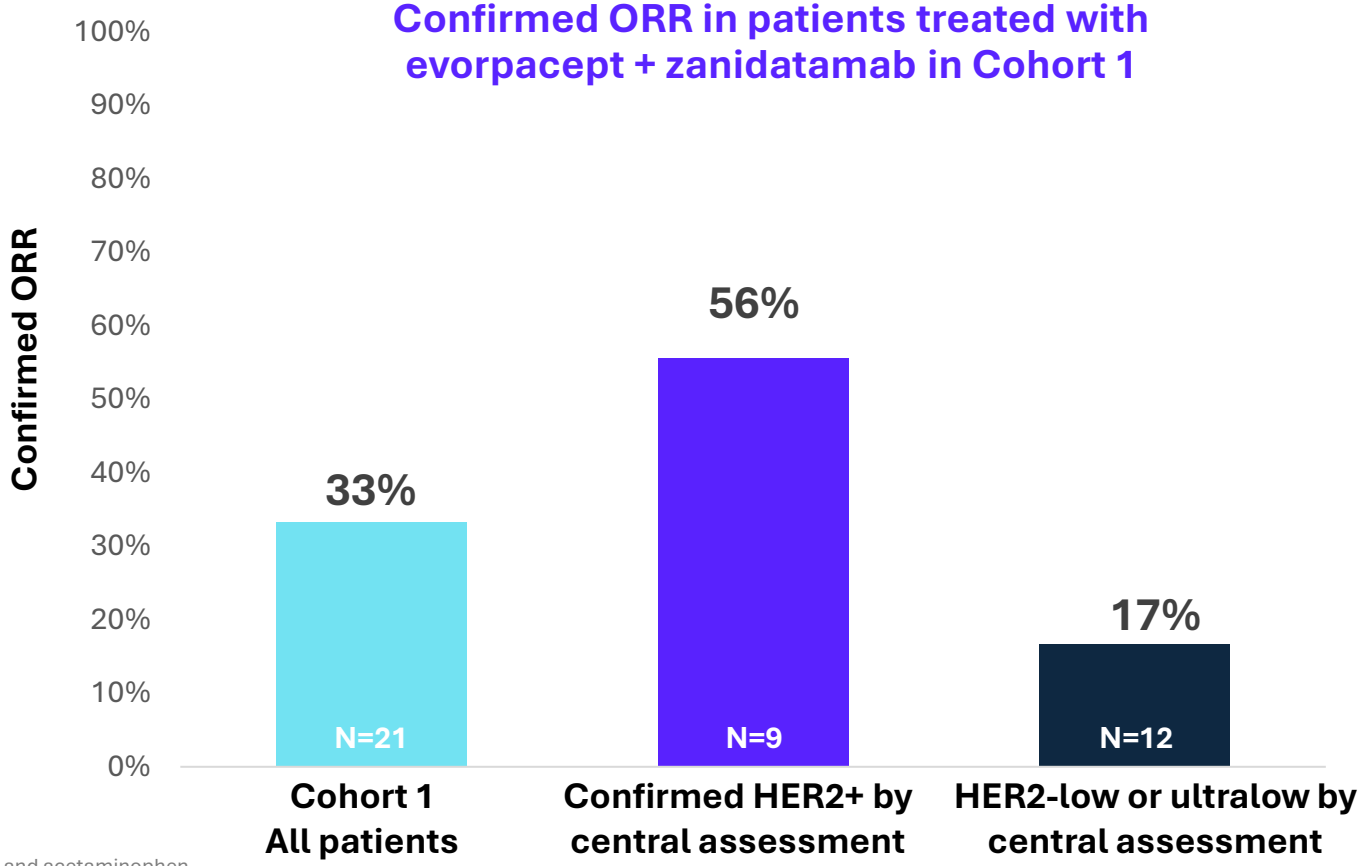
- HER2-positive breast cancer (IHC 3+ or IHC 2+/ISH+)
- ≥3 prior regimens, must include trastuzumab, pertuzumab and either T-DM1, tucatinib, or T-DXd
- Data were analyzed for all patients enrolled and based on central assessment

Treatment[1]

Evorpacept 30 mg/kg Q2W

\+ **Zanidatamab** 1200 mg (<70 kg) OR 1600 mg (≥ 70kg) Q2W

Confirmed ORR in patients treated with evorpacept + zanidatamab in Cohort 1



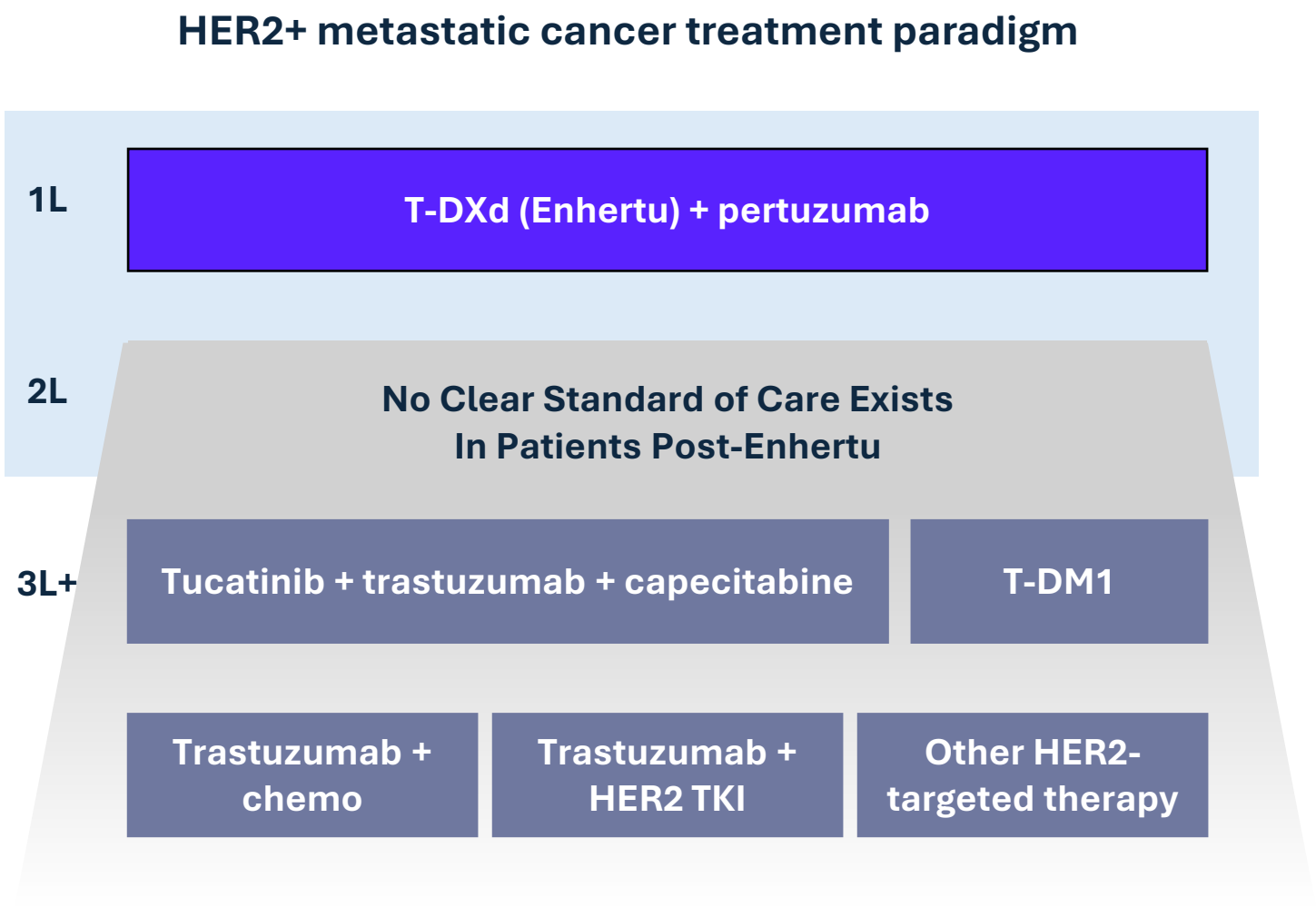
1. Mandatory IRR prophylactic treatment included corticosteroids, antihistamines, and acetaminophen.

Study conducted by Jazz Pharmaceuticals; Median follow-up (range) was 9.6 (0.6, 29.7) months, with six patients on treatment at data cutoff as of August 1, 2024; HER2-Low/Ultralow = IHC1+, IHC2+ / ISH-, IHC 0

Montero AJ et al., San Antonio Breast Cancer Symposium, December 2024, PS8-09

This study provides clinical data supporting further development of evorpacept with HER2-targeted agents in patients with breast cancer

No Clear Standard of Care Exists for Patients Post Enhertu



- Significant unmet need exists and will increase for patients that have progressed on T-DXd
- Evorpacept has demonstrated activity in post-Enhertu patients
  - Evorpacept + zanidatamab showed promising antitumor activity in patients with heavily pretreated HER2-positive mBC including after progression on prior T-DXd

Adapted from NCCN guidelines v5.2025 and FDA label for Enhertu

Strong Expected Benefit in CD47-High and HER2+ Patients Enables Evorpacept-Attributable Benefit in Single-Arm Study

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=80

Evorpacept

\+ Trastuzumab

\+ Physician's Choice Chemo[1]

Key objectives

Primary

- ORR in HER2 ctDNA+ subpopulation

Secondary

- ORR in HER2 ctDNA+ subpopulation by level of CD47 expression
- CBR, DOR, PFS, OS, and safety

Exploratory

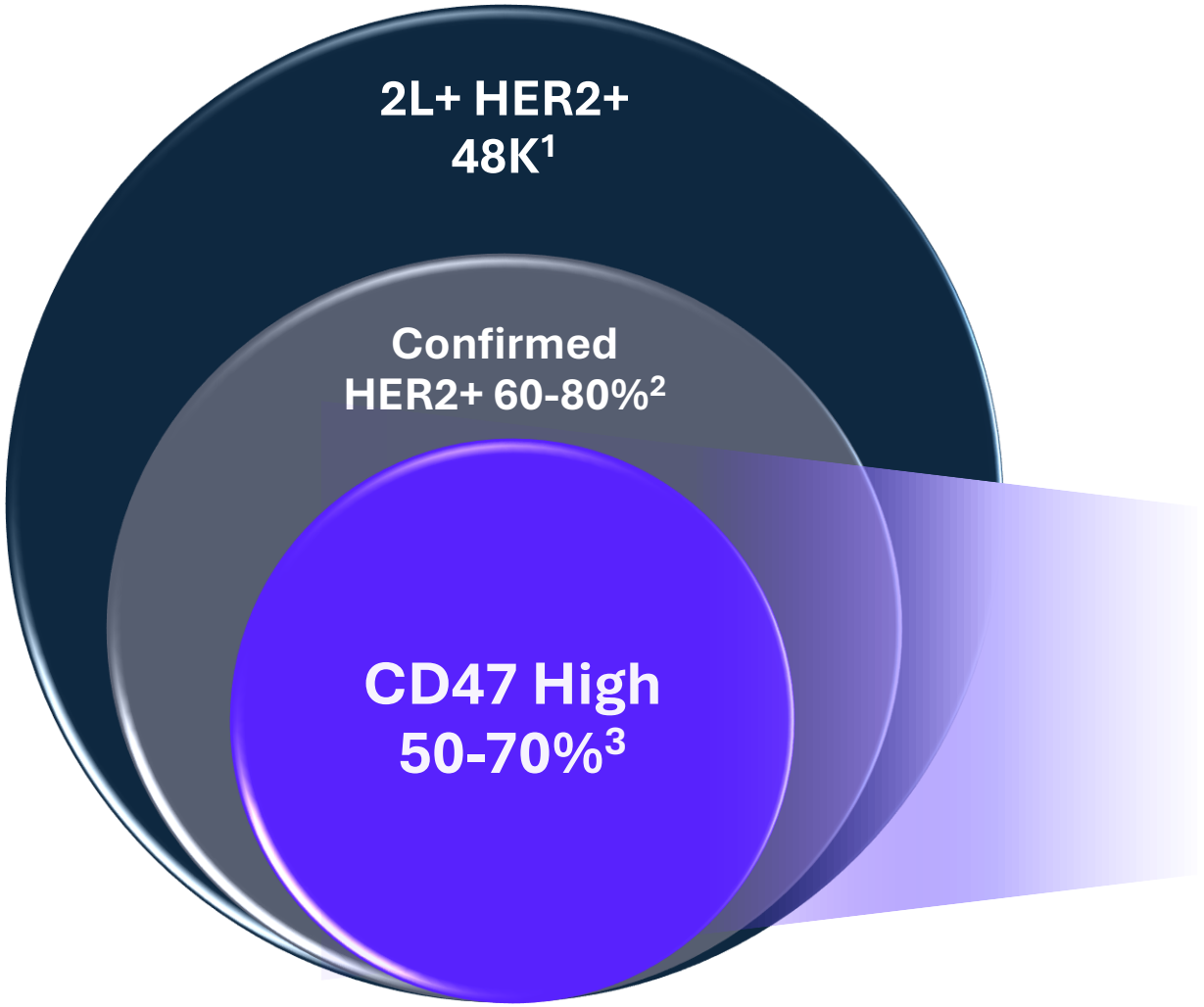
- ORR in HER2 ctDNA-negative subpopulation

- **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**

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

First patient dosed with interim data anticipated Q3 2026

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

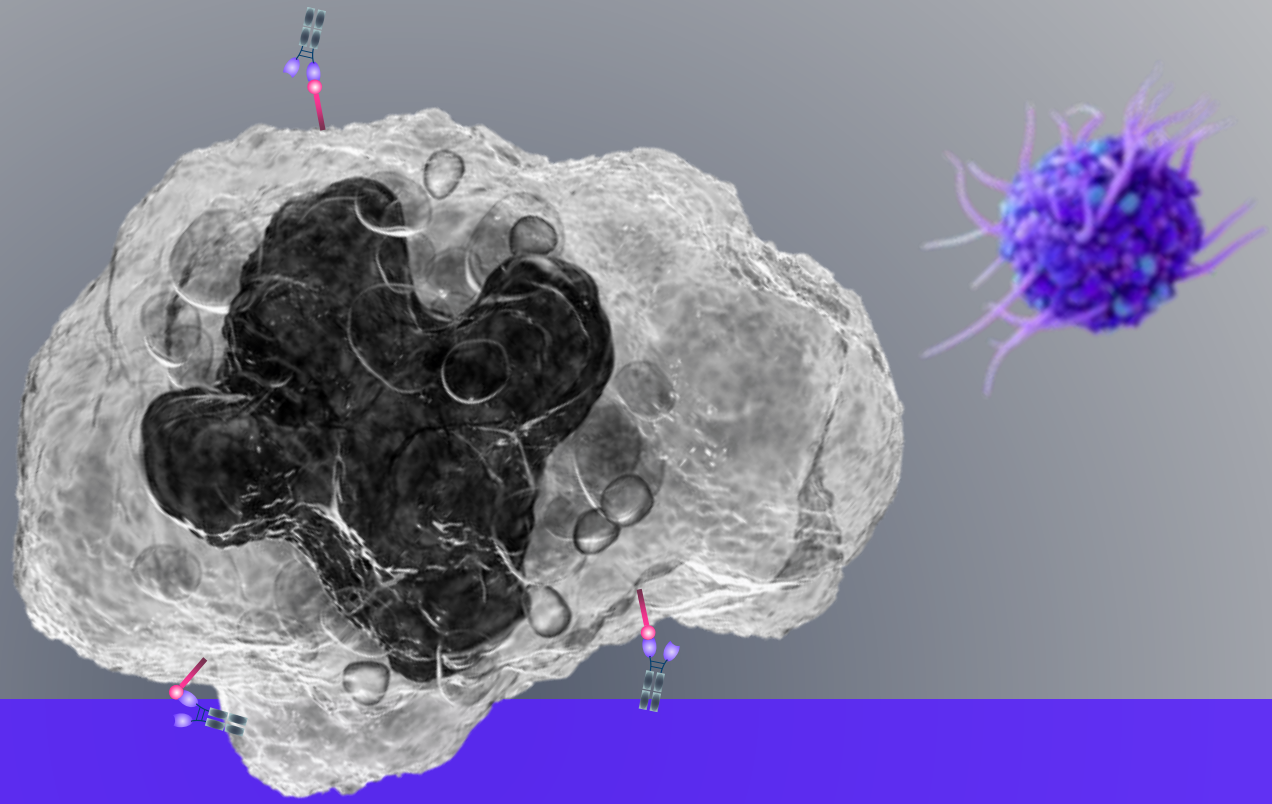


~20K addressable patients are CD47-high

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.

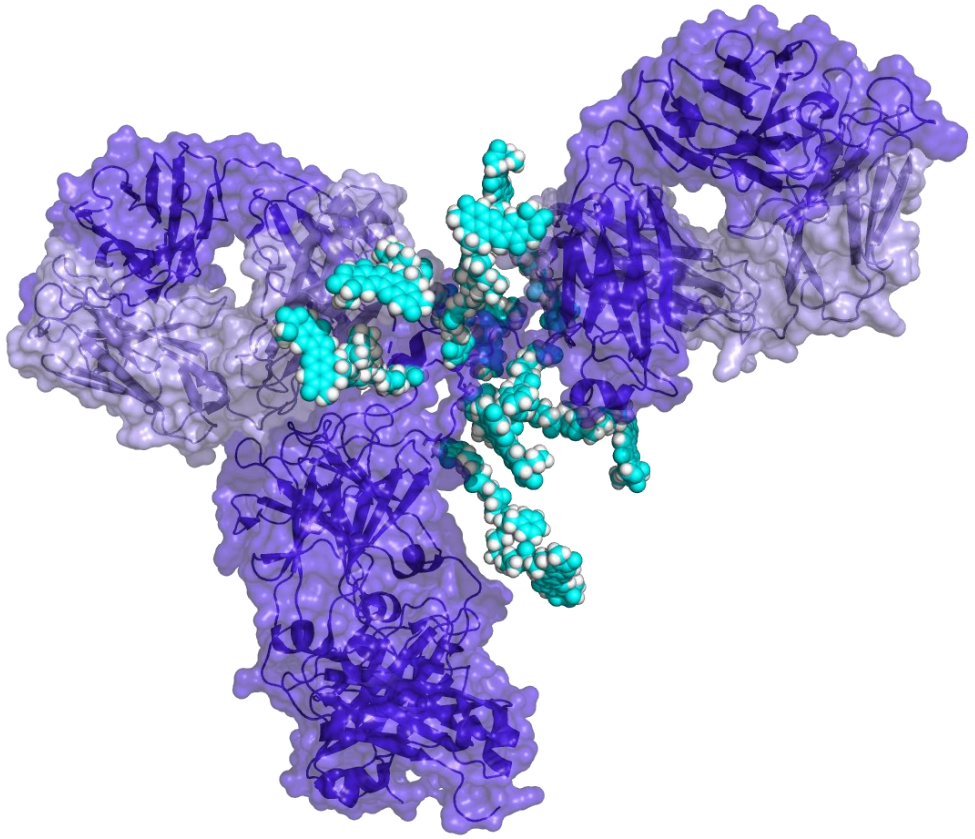
ALX



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

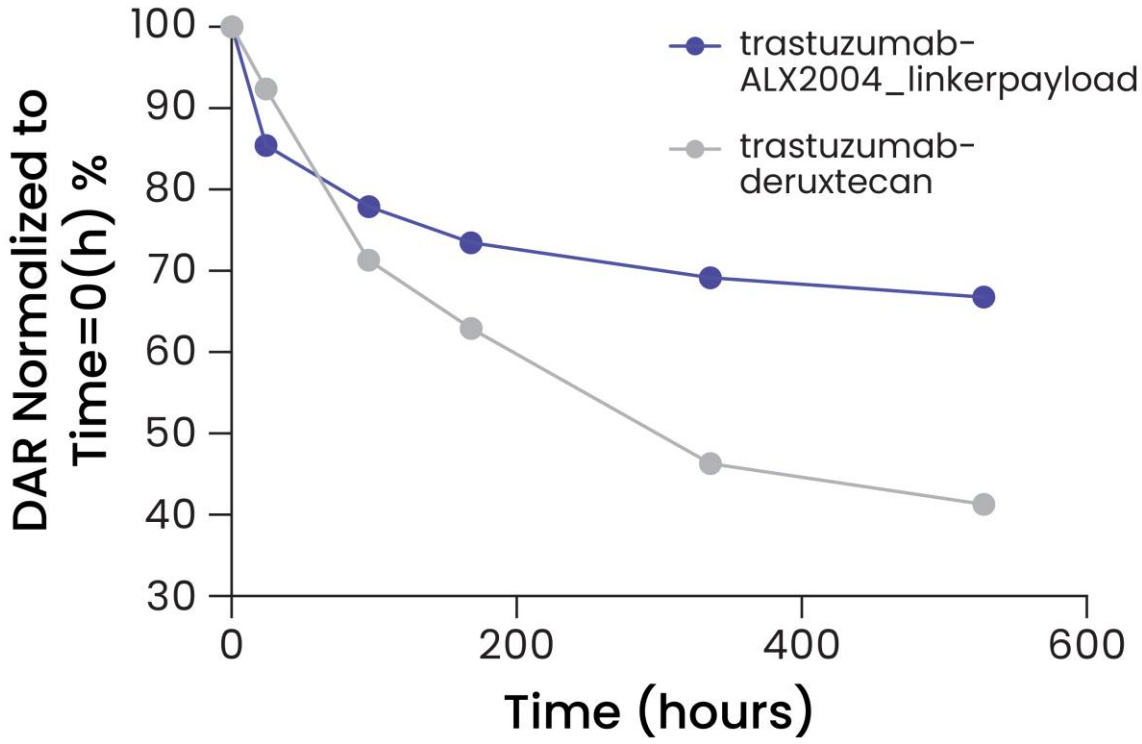
Less drug delivered to off-tumor, off-target tissues

- 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

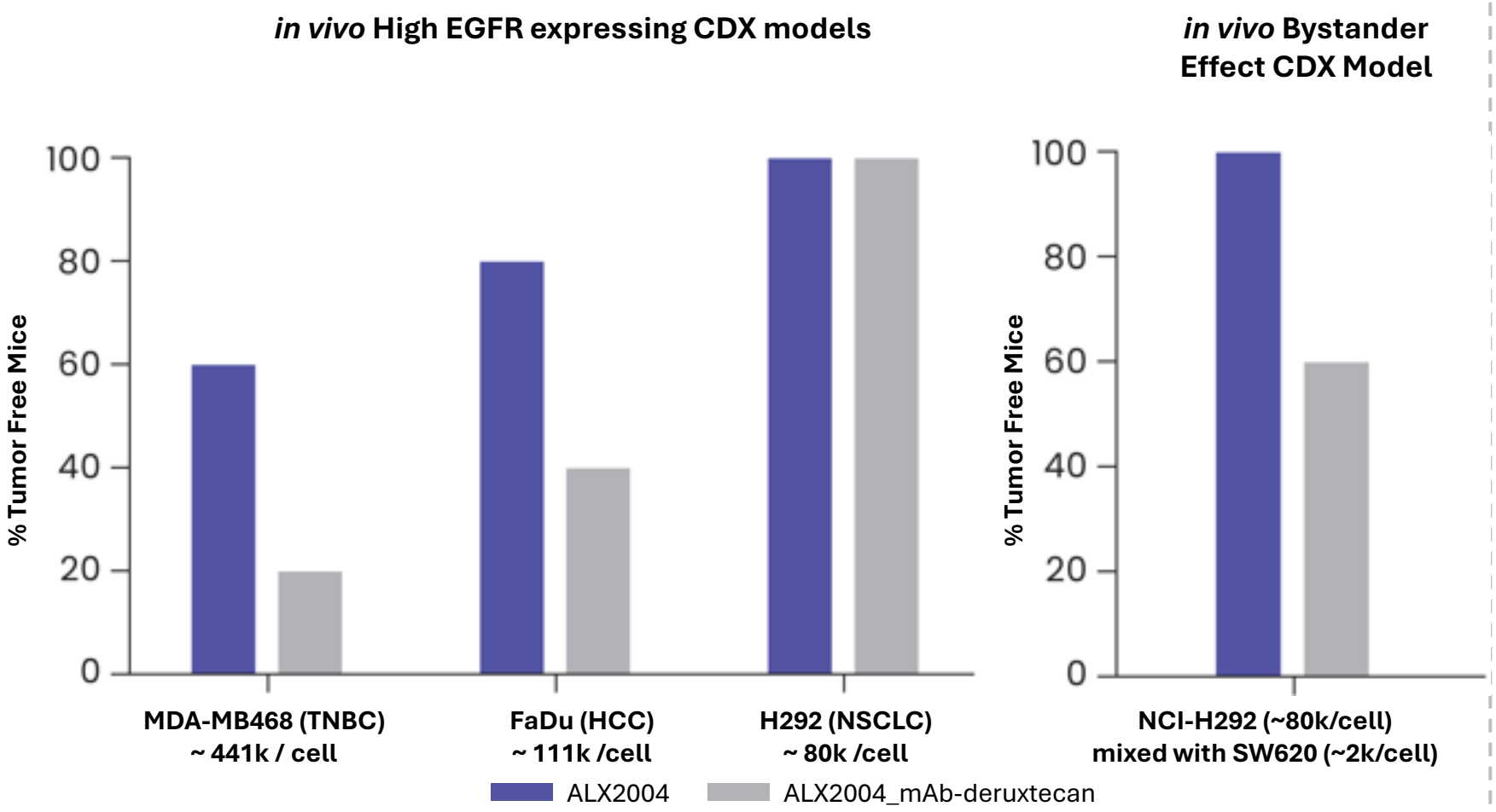


ALX's proprietary linker-payload conjugated to trastuzumab shows improved stability compared to in-house generated trastuzumab-deruxtecan

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

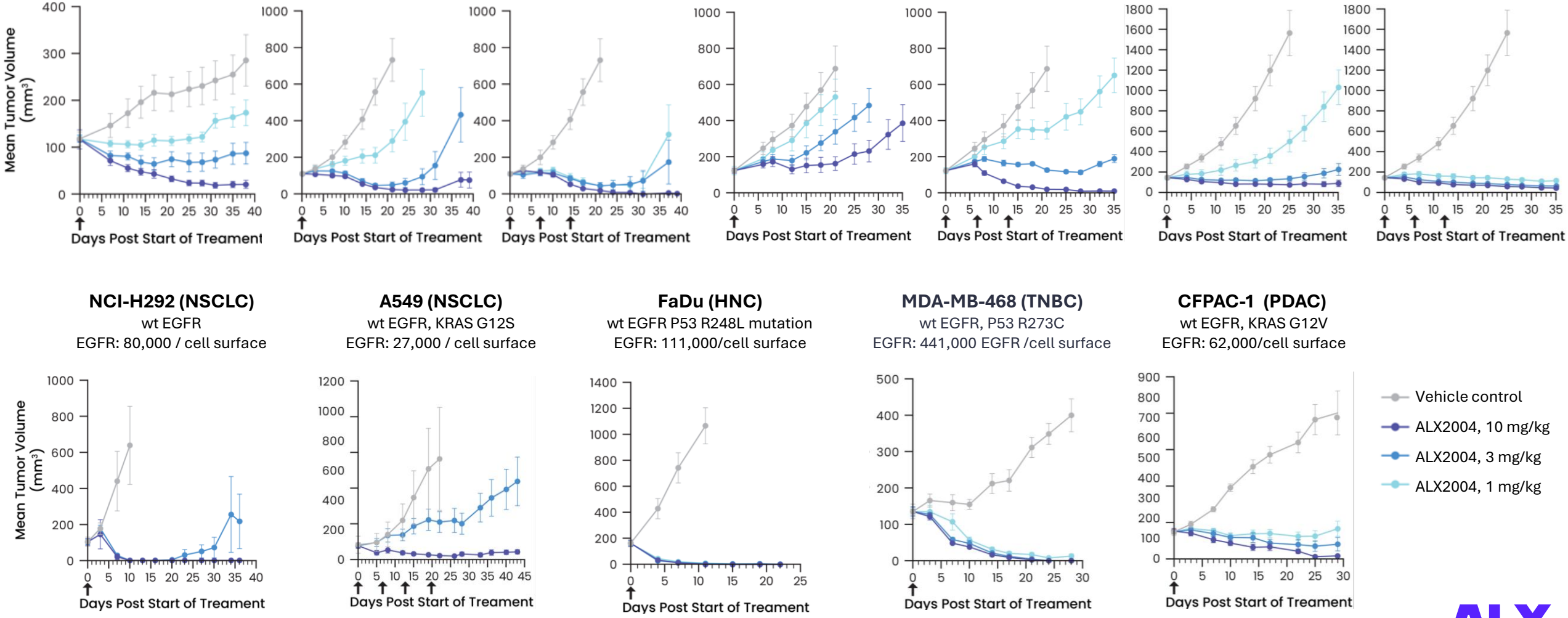
NCI-H292 (NSCLC)
wt EGFR
EGFR: 80,000 / cell surface

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

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

Dose Escalation

BOIN Dose Escalation

- **Dose Level 5** n=3+
- **Dose Level 4** n=3+
- **Dose Level 3** 4 mg/kg Q3W n=3+
- **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)

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

Initial safety data anticipated 1H 2026

Potential to expand into Phase 2 single-arm study to support accelerated approval

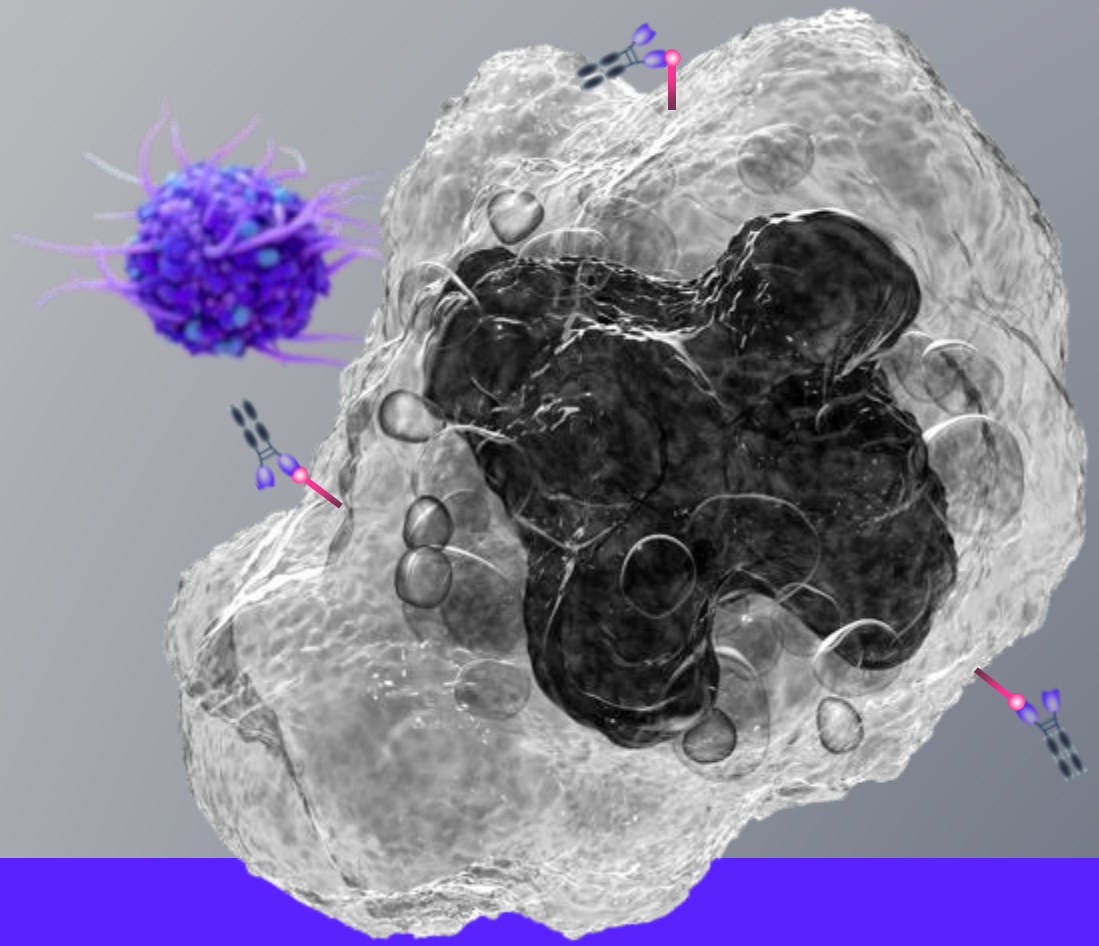
HNSCC: head and neck squamous cell carcinoma; CRC: colorectal cancer; NSCLC: non-small cell lung cancer; ESCC: esophageal squamous cell carcinoma; RDE: recommended dose for expansion

Conclusion

Path Forward and 2026 Catalysts

ALX is Rapidly Advancing Two Novel Cancer Treatments with Multiple Near-Term Catalysts in 2026

1	**ALX is focused on *driving toward multiple inflection points in 2026* across both our programs – Evorpacept and ALX2004**
2	**The addition of Evorpacept led to a compelling benefit for patients with high CD47 expression and retained HER2+ gastric cancer with *the potential to translate to HER2+ breast cancer when combining with Trastuzumab and chemo***
3	**ALX2004 is a *highly differentiated ADC* in development for EGFR-expressing solid tumors now enrolling in a phase 1 trial**
4	**Our projected cash runway extends into Q1 2027 driving key milestones: *ALX2004 initial safety (1H'26), ASPEN-Breast data (Q3'26)***



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