Designing Proteins Delivering Medicines™



Corporate Overview

November 2026

Forward-Looking Statements

Certain statements contained in this presentation, other than statements of historical fact, may constitute forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Such statements include, but are not limited to, statements regarding Xencor's development plans and timelines; potential regulatory actions; expected use of cash resources; the timing and results of clinical trials; the plans and objectives of management for future operations; and the potential markets for Xencor's product and development candidates. Forward-looking statements are based on the current expectations of management and upon what management believes to be reasonable assumptions based on information currently available to it, and involve numerous risks and uncertainties, many of which are beyond Xencor's control. These risks and uncertainties could cause future results, performance or achievements to differ significantly from the results, performance or achievements expressed or implied by such forward-looking statements. Such risks include, but are not limited to, potential delays in development timelines or negative preclinical or clinical trial results, reliance on third parties for development efforts and changes in the competitive landscape including changes in the standard of care for treatment of diseases for which we are developing product candidates, as well as other risks described in Xencor's filings with the Securities and Exchange Commission. Xencor expressly disclaims any duty, obligation or undertaking to update or revise any forward-looking statements contained herein to reflect any change in Xencor's expectations with regard thereto of any subsequent change in events, conditions or circumstances on which any such statements are based, except in accordance with applicable securities laws. For all forward-looking statements, we claim the protection of the safe harbor for forward looking statements contained in the Private Securities Litigation Reform Act of 1995.

Disclaimer: We use our trademarks and our logo in this presentation. This presentation also includes trademarks, tradenames and service marks that are the property of other organizations. Solely for convenience, trademarks and tradenames referred to in this presentation appear without the “®” and “TM” symbols, but those references are not intended to indicate, in any way, that we will not assert, to the fullest extent under applicable law, our rights or that the applicable owner will not assert its rights, to these trademarks and tradenames.

Xencor clinical innovation cycle powered by XmAb® engineering platforms

Validated XmAb® R&D platforms enable rapid, modular drug design

Xencor's world-leading protein engineering capabilities are supported by a strong financial position (~ $555.7 million*) and key partnerships

Purpose-built molecules solve complex biological engineering challenges for better drugs

Rapid prototyping and optimization of XmAb® candidates with defined therapeutic intent catalyzes clinical innovation

XmAb® Drug Development Model

Clinical strategy focuses on technical success

Experienced drug development team delivers rapid proof-of-concept clinical readouts

Data-driven discipline maximizes long-term value creation for stakeholders

Rigorous evaluation of data and competitive landscape establishes programs for commercialization or collaboration

* As of 30-Sep-2026. Includes cash, cash equivalents & marketable debt. Updated 4-Nov-2026.

Our pipeline of next-gen T-cell engagers and novel antibodies is purpose-built with defined opportunities across oncology and autoimmune disease

Program	Targets	XmAb® Platforms	Indications	Discovery	Preclinical	Phase 1	Phase 2	Phase 3
Solid Tumor Oncology: T-cell Engagers (CD3 & CD28)								
XmAb819	ENPP3 x CD3	2+1 Bispecific	ccRCC, pRCC, NSCLC, CRC					
XmAb541 + XmAb808	CLDN6 x CD3 + B7H3 x CD28	2+1 Bispecific, Xtend™	HGSOC					
XmAb Program	Undisclosed TCE	Bispecific, Xtend	Solid tumor oncology					
Immunology Programs (TL1A & CD3 B-Cell Depletion)								
XmAb942	TL1A	Xtend, FcKO	IBD (Ulcerative colitis)			Phase 2b		
XmAb412	TL1A x IL23p19	XenLock™ Bispecific, Xtend	IBD					
Plamotamab	CD20 x CD3	Bispecific	Rheumatoid arthritis			Phase 1b		
XmAb657	CD19 x CD3	2+1 Bispecific, Xtend	Idiopathic inflammatory myopathies, Sjögren's disease, Systemic sclerosis					

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ccRCC clear cell renal cell carcinoma **pRCC** papillary renal cell carcinoma **NSCLC** non-small cell lung cancer **CRC** colorectal cancer **HGSOC** high-grade serous ovarian carcinoma **FcKO** Fc knock out **IBD** Inflammatory bowel disease

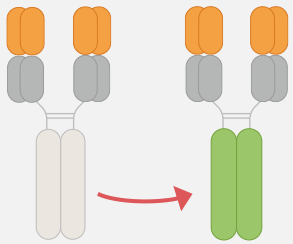
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XmAb® drug development model is validated by multiple commercialized medicines and pivotal- or late-stage programs

XmAb® toolkits enable rapid prototyping and lead optimization

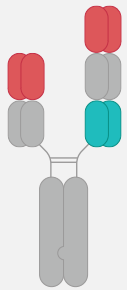
XmAb® Fc Domains

Augment native immune functions in molecules and/or control their structure, while preserving desired attributes (e.g., Xtend™ extended half-life or heterodimeric Fc domain for bispecific antibodies).



XmAb® 2+1 Formats

Designed to enable antibodies to bind more avidly and selectively kill cells with higher antigen density, potentially sparing normal cells



Continued clinical success and commercialization of partner programs supports Xencor's internal pipeline advancement

Commercial Stage

- ALEXION AstraZeneca Rare Disease — Ultomiris®
- Incyte — Monjuvi® / Minjuvi®

Pivotal Stage

- astellas — ASP2138
- AMGEN — xaluritamig
- Zenas BioPharma — obexelimab
- VIR — tobevibart
- Johnson & Johnson Innovative Medicine — JNJ-9401
- GILEAD — teropavimab + zinlirvimab

Registered trademarks Ultomiris® (Alexion Pharmaceuticals, Inc.), Monjuvi® & Minjuvi® (Incyte Holdings Corp.)

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Differentiated XmAb® antibodies developed by partners generate potential milestone and royalty revenues to offset internal development costs

Partner	Program	XmAb®	Indications[1]	Status[1]	Royalty %	Potential Milestone Payments[1]
ALEXION AstraZeneca Rare Disease	**Ultomiris®**	Xtend™	PNH, aHUS, gMG, NMOSD	**Marketed**	**Low SD[2]**	**All milestones paid**
Incyte	**Monjuvi®**	Cytotoxic	r/r DLBCL, r/r FL, 1L DLBCL	**Marketed**	**High SD to Low DD[2]**	**$195 million**
AMGEN	**Xaluritamig** STEAP1 x CD3	2+1 Bispecific	Prostate cancer, Ewing sarcoma	**Phase 3**	**Mid to High SD**	**$225 million**
Zenas BioPharma	**Obexelimab**	Immune Inhibitor	IgG4-RD, RMS, SLE	**Phase 3**	**Mid SD to Mid Teen[3]**	**$447.5 million[4]**
VIR	**Tobevibart**	Cytotoxic & Xtend	Hepatitis Delta	**Phase 3**	**Low to Mid SD[5]**	**$65 million**
astellas	**ASP2138** CLDN18.2 x CD3	2+1 Bispecific	Gastric/GEJ cancer	**Phase 3**	**High SD to Low DD**	**$207.5 million**
Johnson&Johnson Innovative Medicine	**JNJ-9401** PSMA x CD28	Bispecific	Prostate cancer	**Phase 3**	**High SD to Low DD**	**Undisclosed $640 million under collaboration**
	JNJ-1493 CD20 x CD28	Bispecific	Heme-Onc	**Phase 1**	**High SD to Low DD**	**Undisclosed $636.3 million under collaboration**

Xevudy® (sotrovimab), with Xencor's Xtend™ Fc Domain, was provided under emergency use authorization for COVID-19, but is not currently authorized in the U.S.

Registered trademarks: Ultomiris® (Alexion Pharmaceuticals, Inc.), Monjuvi® (Incyte Holdings Corp.), Xevudy® (Glaxo Group Limited), Corporate Logos (Respective Companies)

1 As of 04-Nov-2026 **2** In excess of caps as defined under agreements with OMERS, ex-U.S. **3** Dependent on geography **4** In addition, as of 30-Sep-2026, Xencor owns 1.0 million shares of common stock of Zenas **5** ex-U.S.
SD single digit **DD** double digit **PNH** paroxysmal nocturnal hemoglobinuria **aHUS** atypical hemolytic uremic syndrome **gMG** generalized myasthenia gravis **NMOSD** Neuromyelitis optica spectrum disorder
r/r relapsed or refractory **DLBCL** diffuse large B-cell lymphoma **FL** follicular lymphoma **1L** first line **IgG4-RD** IgG4-related disease **RMS** relapsing multiple sclerosis **SLE** systemic lupus erythematosus **BsAb** bispecific antibody
GEJ Gastroesophageal junction

Focused clinical execution is expected to deliver key clinical readouts in 2026 and advance programs with additional data in 2027

XmAb® Drug Candidate		Indication	1H 2026	2H 2026
Oncology Portfolio				
XmAb819	ENPP3 x CD3	ccRCC	Initiate tumor expansion cohorts for CRC, NSCLC and pRCC ✓	Present RP3D monotherapy data at ESMO 2026 ✓
XmAb541 + XmAb808	CLDN6 x CD3 + B7-H3 x CD28	CLDN6+ HGSOC		Ongoing dose escalation
Immunology Portfolio				
XmAb942	Xtend™ TL1A	IBD+	Present final Phase 1 healthy volunteer data at DDW 2026 ✓	Update on progress achieved in Phase 2b XENITH-UC study ~YE26
XmAb412	TL1A x IL23p19	IBD+	Present preclinical characterization at DDW 2026 ✓	Initiate first-in-human study ✓
Plamotamab	CD20 x CD3	Rheumatoid arthritis		Initial dose-escalation data from ongoing Phase 1b in RA at ACR ✓
XmAb657	CD19 x CD3	IIM, Sjögren's disease, Systemic sclerosis		Update on progress achieved in FIH dose-escalation study ✓

As of 09-Nov-2026 **ccRCC** clear cell renal cell carcinoma **CRC** colorectal cancer **NSCLC** non-small cell lung cancer **pRCC** papillary renal cell carcinoma **RP3D** recommended Phase 3 dose **HGSOC** high-grade serous ovarian carcinoma **IBD** inflammatory bowel disease **RA** rheumatoid arthritis **IIM** idiopathic inflammatory myopathies **FIH** first-in-human

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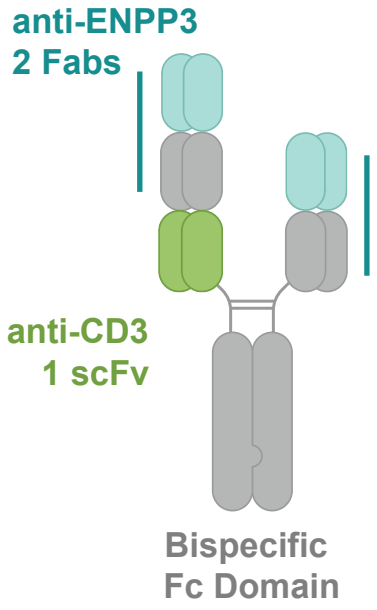
XmAb® Bispecific T-Cell Engagers

XmAb819 (ENPP3 x CD3)

Updated Phase 1 results in advanced ccRCC presented at ESMO 2026

ENPP3 x CD3 T-cell Engager in Development for ccRCC

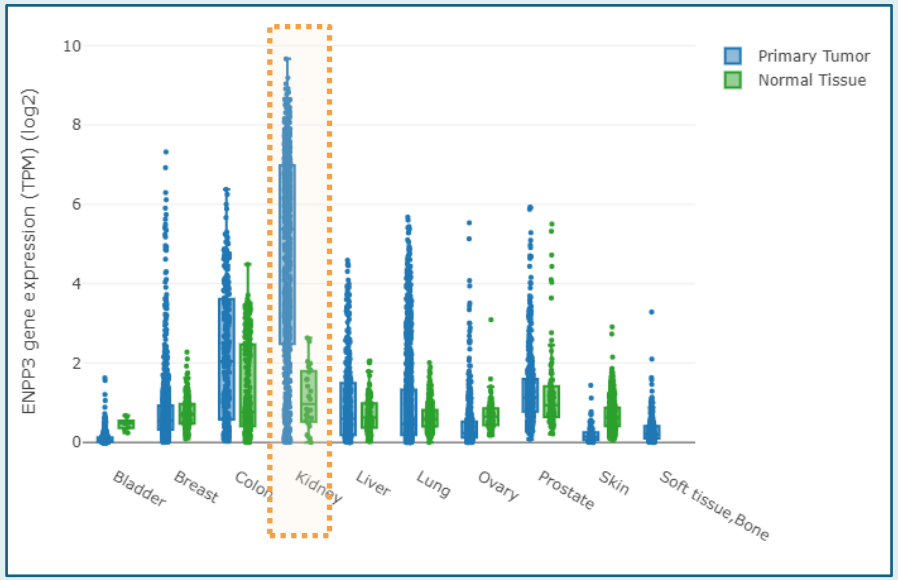
XmAb819 engages the immune system and activates T cells for highly potent and targeted lysis of tumor cells expressing ENPP3



ENPP3

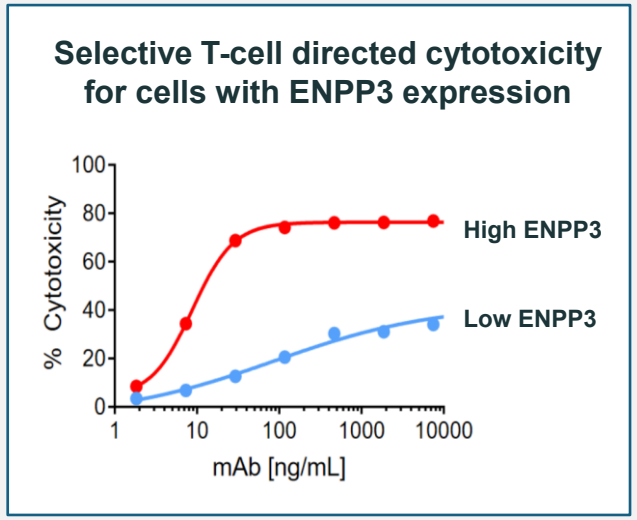
Ectonucleotide pyrophosphatase / phosphodiesterase family member 3

A differentially expressed target, with high level expression in renal cell carcinoma and low-level expression on normal tissues[1]



XmAb® 2+1

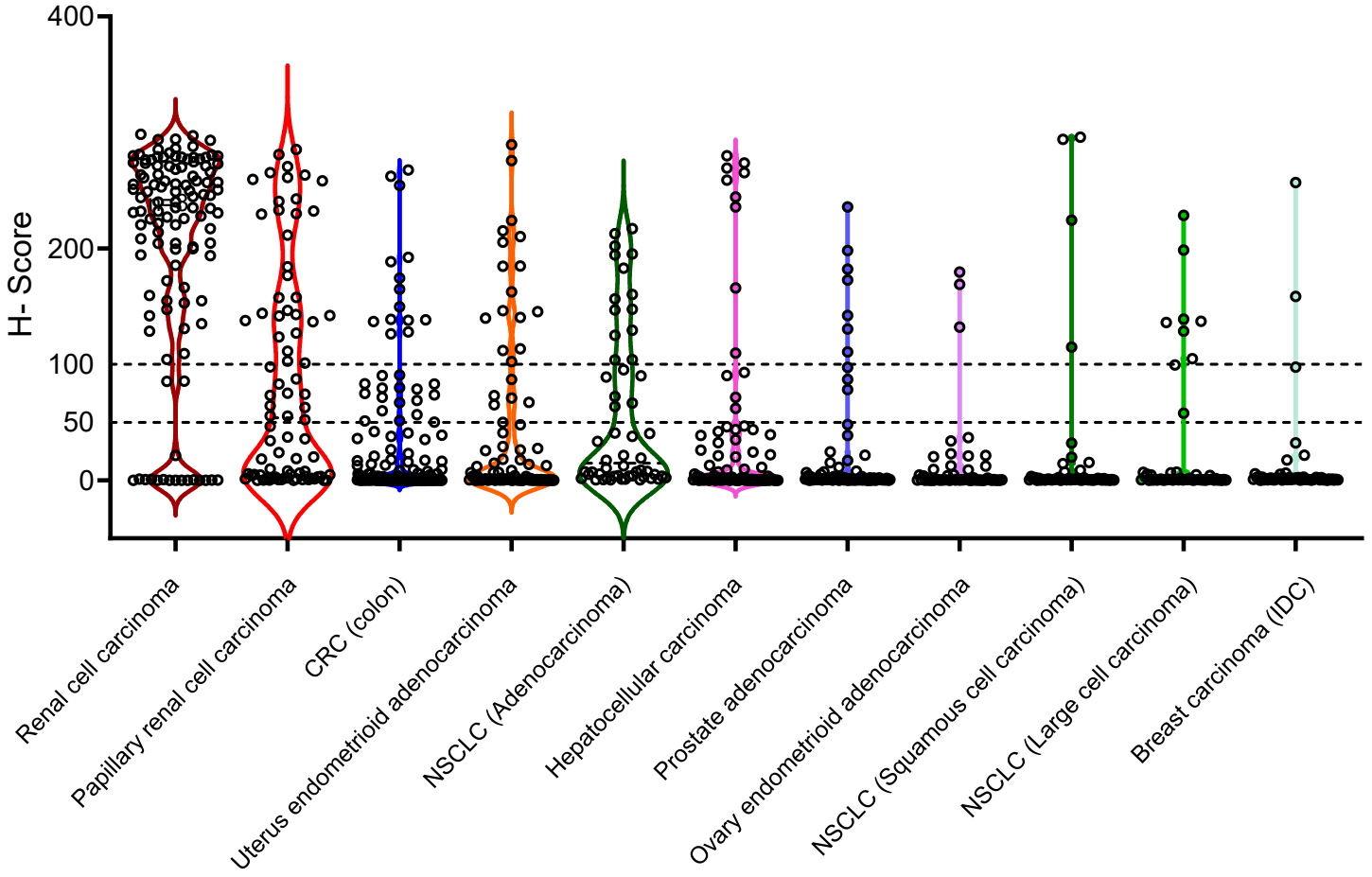
Enables antibodies to bind more avidly and selectively kill tumor cells with higher antigen density, potentially sparing normal cells

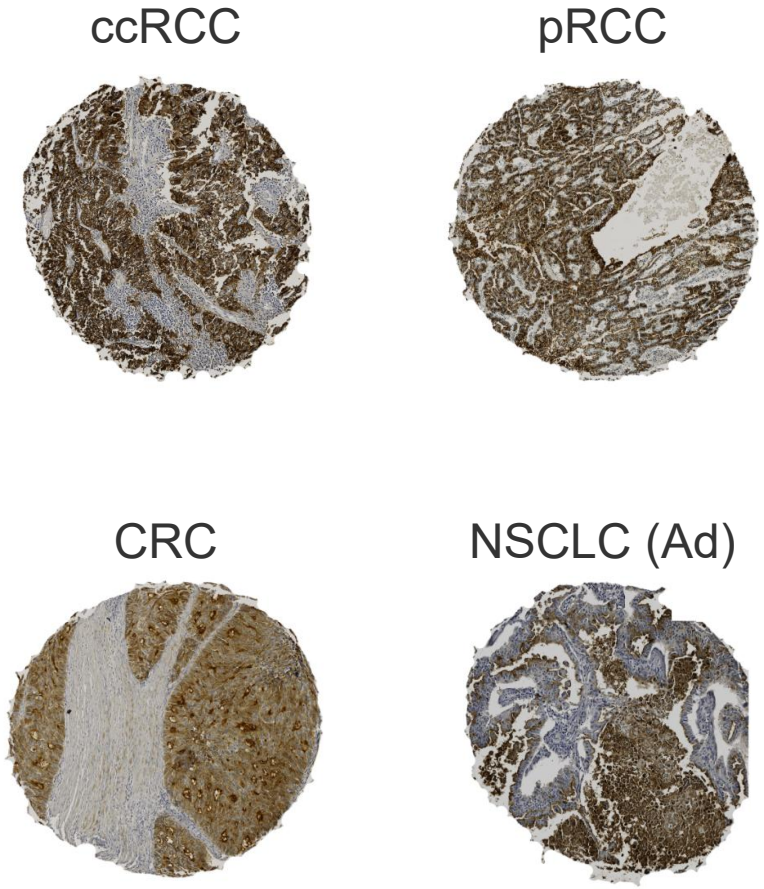


1 Doñate, Fernando, et al. Clinical Cancer Research 22.8 (2016)

XmAb819 targets ENPP3 for treatment of renal cell carcinoma and other solid tumors

ENPP3 is strongly expressed in ccRCC and in subsets of other histologies



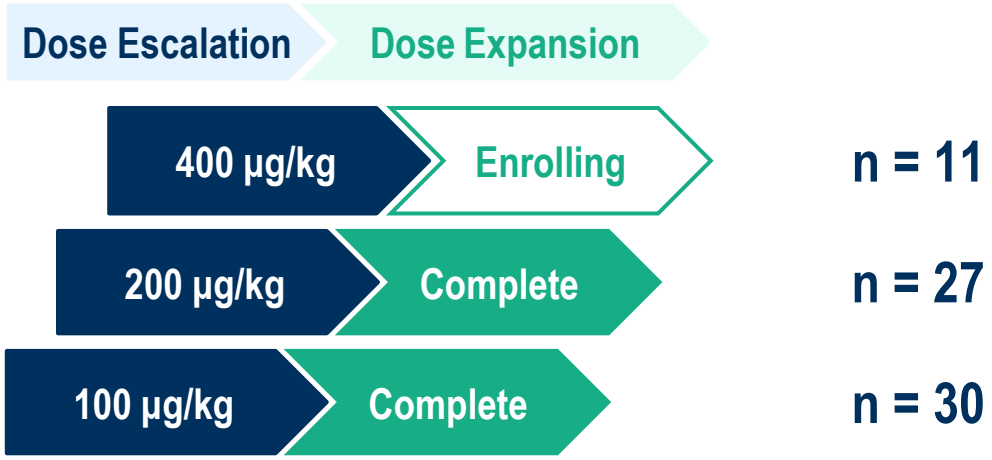


ccRCC clear cell renal cell carcinoma **CRC** colorectal cancer **IDC** invasive ductal carcinoma **NSCLC** non-small cell lung cancer **pRCC** papillary renal cell carcinoma **RCC** renal cell carcinoma

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XmAb819-01: Phase 1 study design in advanced ccRCC

Study Schema – IV Administration



Outcomes reported in current presentation.

Eight dose levels explored below expansion (n = 51)

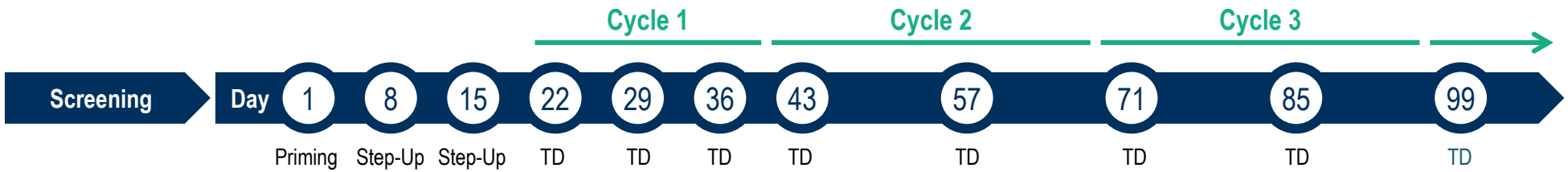
Key Eligibility Criteria

- R/R ccRCC – prior ICI & VEGFR-TKI
- Measurable disease by RECIST v1.1
- No prior anti-ENPP3 therapy

Endpoints

- Primary: Safety & Tolerability
- Secondary:
  - ORR, DOR, PFS per RECIST 1.1
  - Pharmacokinetics

NCT05433142. US & EU sites.



Pal SK, et al. Presented at ESMO Congress 2026; October 23–27, 2026; Madrid, Spain. Abstract 2684. **DOR** duration of response **ICI** immune checkpoint inhibitor **IV** intravenous **ORR** objective response rate **PFS** progression free survival **R/R** relapsed / refractory **TD** target dose **TKI** tyrosine kinase inhibitor **VEGFR** vascular endothelial growth factor receptor

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Patient characteristics

Baseline Characteristic	All IV Doses (n=119)	Expansion Doses (n=68)
Median Age, Years (range)	61 (34, 78)	62 (34, 78)
Male, n (%)	97 (81.5)	58 (85.3)
Race – White, n (%)	101 (84.9)	56 (82.4)
Time Since Disease Diagnosis, mo. (range)	52.5 (7, 287)	49.5 (7, 287)
IMDC Risk Score at Baseline[1], n (%)		
Favorable	8 (6.7)	4 (5.9)
Intermediate	81 (68.1)	46 (67.6)
Poor	30 (25.2)	18 (26.5)
Sarcomatoid Features, n (%)	16 (13.4)	13 (19.1)
≥3 Metastatic Sites, n (%)	84 (70.6)	46 (67.6)
Bone metastasis, n (%)	58 (48.7)	32 (47.1)
Liver metastasis, n (%)	48 (40.3)	27 (39.7)
Target Lesions SOD (mm), Median	90.0	90.5
ENPP3 Positive From Cases With Tissues[2] (%)	83/89 (93.3)	50/52 (96.2)

Prior Therapy	All IV Doses (n=119)	Expansion Doses (n=68)
# Prior Regimens, Median (Min, Max)	3 (1, 10)	3 (1, 10)
1, n (%)	7 (5.9)	4 (5.9)
2	35 (29.4)	22 (32.4)
3	30 (25.2)	18 (26.5)
≥4	47 (39.5)	24 (35.3)
Prior Treatments, n (%)		
Any Immune Checkpoint Inhibitor	119 (100)	68 (100)
Any VEGFR-TKI	119 (100)	68 (100)
2 or More VEGFR-TKIs	76 (63.9)	43 (63.2)
HIF2α Inhibitor	42 (35.3)	23 (33.8)
Nephrectomy, n (%)	81 (68.1)	46 (67.6)

Subjects who received ≥1 protocol-specified dose of XmAb819 were included in the baseline characteristics analysis. Data cut-off: **01 September 2026**. Excludes 1 participant who experienced approximately 10-fold higher exposure than intended at the first and only dose administered, and who subsequently developed Grade 4 CRS and discontinued treatment.

[1]IMDC risk derived at study baseline using central laboratory values; KPS <80% defined as ECOG ≥2.
[2]Tumor ENPP3 positivity was defined as a tumor proportion score (TPS) ≥1, regardless of expression intensity, by a clinical trial IHC assay (Roche Diagnostics).

Pal SK, et al. Presented at ESMO Congress 2026; October 23–27, 2026; Madrid, Spain. Abstract 2684. **HIF2α** hypoxia-inducible factor 2-alpha **IMDC** International Metastatic RCC Database Consortium **IHC** immunohistochemistry **SOD** sum of diameters **TKI** tyrosine kinase inhibitor **VEGFR** vascular endothelial growth factor receptor

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Adverse events

- Low rate of dose reductions and treatment discontinuation due to TEAEs
- Events were predominately confined to the priming dose period
- Most common Grade ≥3 TEAEs were asymptomatic, transient transaminase increases

Summary of Adverse Events

Number of Subjects With, n (%)	All IV Doses (N=119)	Expansion Doses (n=68)
Grade ≥ 3	95 (79.8)	56 (82.4)
Related TEAE Grade ≥ 3	75 (63.0)	49 (72.1)
Serious TEAE	74 (62.2)	42 (61.8)
Related Serious TEAE	64 (53.8)	37 (54.4)
TEAE Leading to Dose Reduction	7 (5.9)	1 (1.5)
Did Not Reach Target Dose	9 (7.6)	3 (4.4)
TEAE Leading to Discontinuation	8 (6.7)	5 (7.4)
Related TEAE Leading to Discontinuation	7 (5.9)	4 (5.9)
Leading to Death	0	0

Subjects who received ≥1 protocol-specified dose of XmAb819 were included in the safety analysis. Data cut-off: **01 September 2026.** Excludes 1 participant who experienced approximately 10-fold higher exposure than intended at the first and only dose administered, and who subsequently developed Grade 4 CRS and discontinued treatment. Reported TEAEs were defined per MedDRA and CTCAE; CRS graded per ASTCT[2].

[1]Combined rash term includes 10 preferred terms: rash (unspecified, maculo-papular, erythematous, macular, morbilliform, pustular, pruritic, anal), erythema, dermatitis acneiform.

[2]Lee DW, et al. Biol Blood Marrow Transplant. 2019;25:625-638.

TEAEs reported in >25% Subjects by Maximum Severity Grade

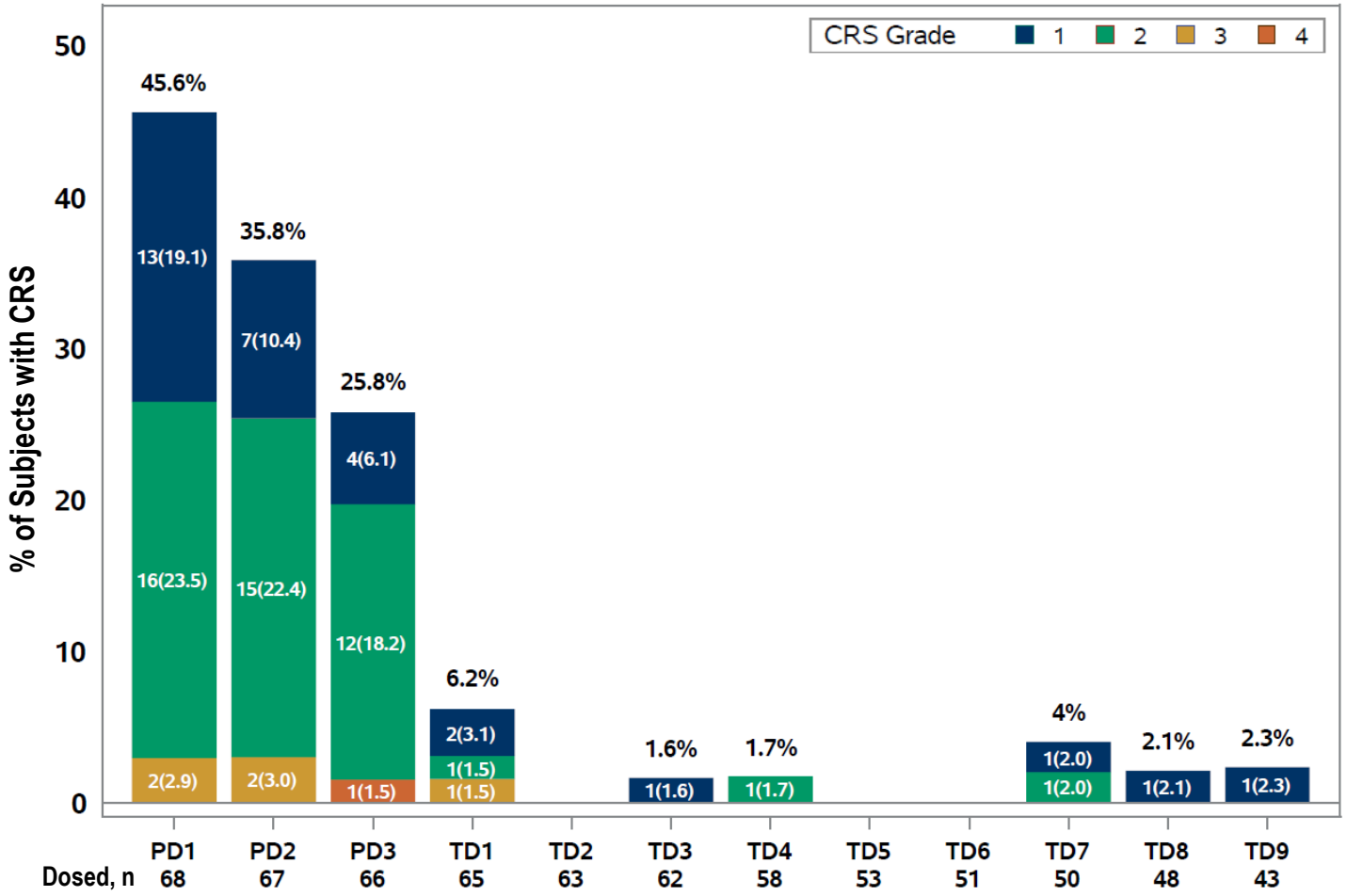
Expansion Doses (n=68)	Any Incidence		>Day 29 Onset	
Preferred Term, n (%)	All grades	Grade ≥3	All grades	Grade ≥3
Rash (combined term)[1]	62 (91.2)	15 (22.1)	20 (29.4)	2 (2.9)
Cytokine release syndrome	46 (67.6)	4 (5.9)	12 (17.6)	1 (1.5)
ALT increased	36 (52.9)	23 (33.8)	12 (17.6)	7 (10.3)
Nausea	35 (51.5)	-	9 (13.2)	-
AST increased	33 (48.5)	24 (35.3)	10 (14.7)	6 (8.8)
Oedema peripheral	31 (45.6)	-	11 (16.2)	-
Vomiting	30 (44.1)	-	7 (10.3)	-
Diarrhoea	28 (41.2)	2 (2.9)	9 (13.2)	2 (2.9)
Fatigue	27 (39.7)	-	11 (16.2)	-
Headache	23 (33.8)	-	10 (14.7)	-
Hypophosphataemia	20 (29.4)	1 (1.5)	8 (11.8)	-
Hypotension	20 (29.4)	5 (7.4)	3 (4.4)	1 (1.5)
Pruritus	19 (27.9)	-	7 (10.3)	-
Chills	18 (26.5)	-	11 (16.2)	-
Cough	18 (26.5)	-	10 (14.7)	-

Pal SK, et al. Presented at ESMO Congress 2026; October 23–27, 2026; Madrid, Spain. Abstract 2684. **ALT** alanine aminotransferase **ASTCT** American Society for Transplantation and Cellular Therapy **AST** aspartate aminotransferase **CTCAE** Common Terminology Criteria for Adverse Events **MedDRA** Medical Dictionary for Regulatory Activities **SAE** serious adverse event **TEAE** treatment-emergent adverse event

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Cytokine release syndrome (CRS)

CRS by Max Grade & Dose (n=68)



- CRS was predominately low-grade and occurred during priming
- No dose delays and one treatment discontinuation due to CRS
- Premedications for XmAb819:
  - Acetaminophen, diphenhydramine, and IV normal saline
  - Dexamethasone prior to priming doses
  - Tocilizumab prior to priming dose 1 (PD1)
- Graded per ASTCT consensus criteria; managed per institutional standards, or NCCN guidelines when local standards were absent

Subjects who received ≥1 protocol-specified dose of XmAb819 were included in the safety analysis. Data cut-off: **01 September 2026.** Excludes 1 participant who experienced approximately 10-fold higher exposure than intended at the first and only dose administered, and who subsequently developed Grade 4 CRS and discontinued treatment. Reported TEAEs were defined per MedDRA and CTCAE; CRS graded ASTCT[1]. Per-protocol PK samples were not collected for subject in plot (left) with reported Grade 4 CRS at priming dose 3; drug exposure could not be determined. Two subjects each experienced 2 events of Grade ≥3 CRS.

[1]Lee DW, et al. Biol Blood Marrow Transplant. 2019;25:625-638.

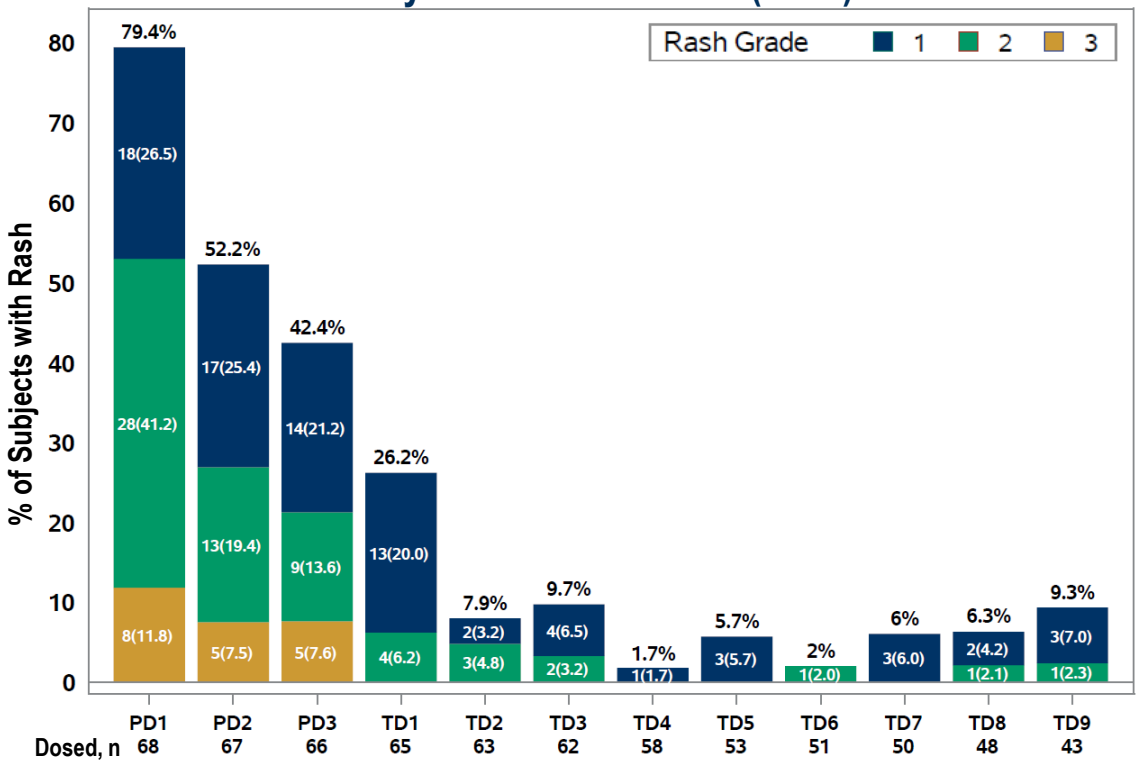
Pal SK, et al. Presented at ESMO Congress 2026; October 23–27, 2026; Madrid, Spain. Abstract 2684. **ASTCT** American Society for Transplantation and Cellular Therapy **CRS** cytokine release syndrome **CTCAE** Common Terminology Criteria for Adverse Events **MedDRA** Medical Dictionary for Regulatory Activities **NCCN** National Comprehensive Cancer Network **PD** priming dose **TEAE** treatment-emergent adverse event **TD** target dose

Cytokine-driven adverse events

- Rash, an on-target effect due to ENPP3 expression on basophils and mast cells, is well managed with daily antihistamine (oral) and steroids (topical & oral).
- Dose delays due to rash occurred in 4.4% of subjects, with no treatment discontinuations.

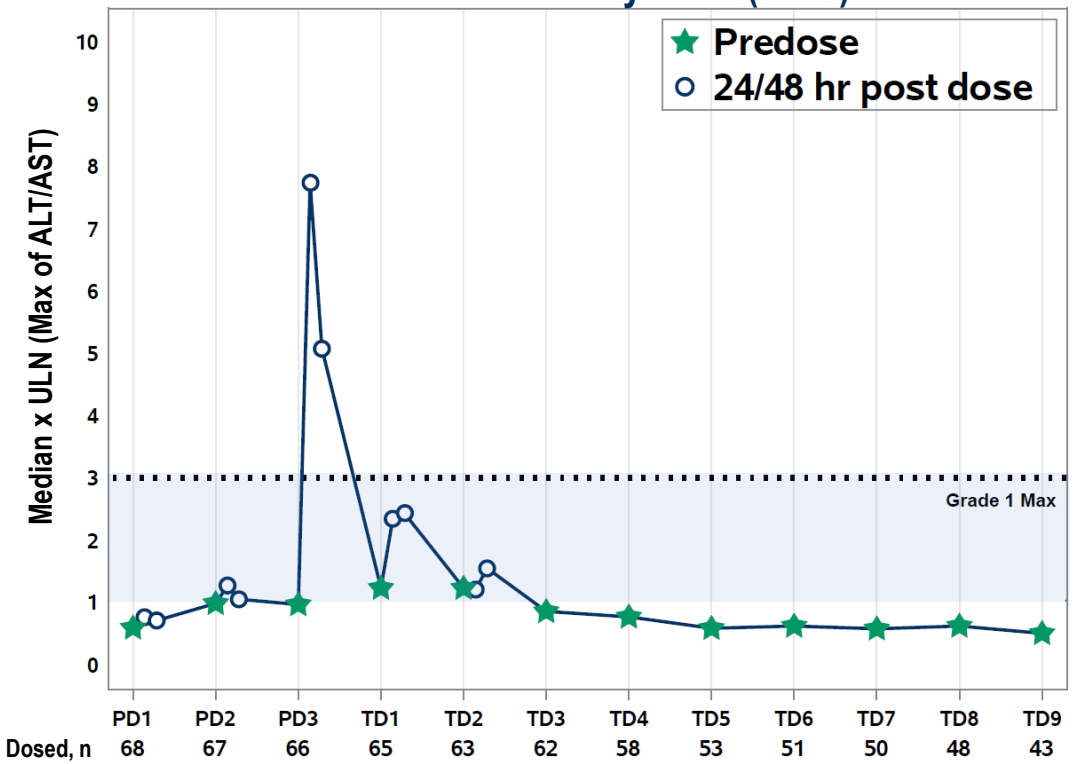
- Transaminase elevations were asymptomatic, transient, and self-limited, with a median time to resolution of 7 days and no clinical sequelae.
- Dose delays due to transaminase elevations occurred in 14.7% of subjects, with no treatment discontinuations.

Rash by Max Grade & Dose (n=68)



Combined rash terms (10 PTs): rash (unspecified, maculo-papular, erythematous, macular, morbilliform, pustular, pruritic, anal), erythema, dermatitis acneiform. Reported TEAEs were defined per MedDRA and CTCAE. Data cut-off: **01 September 2026.**

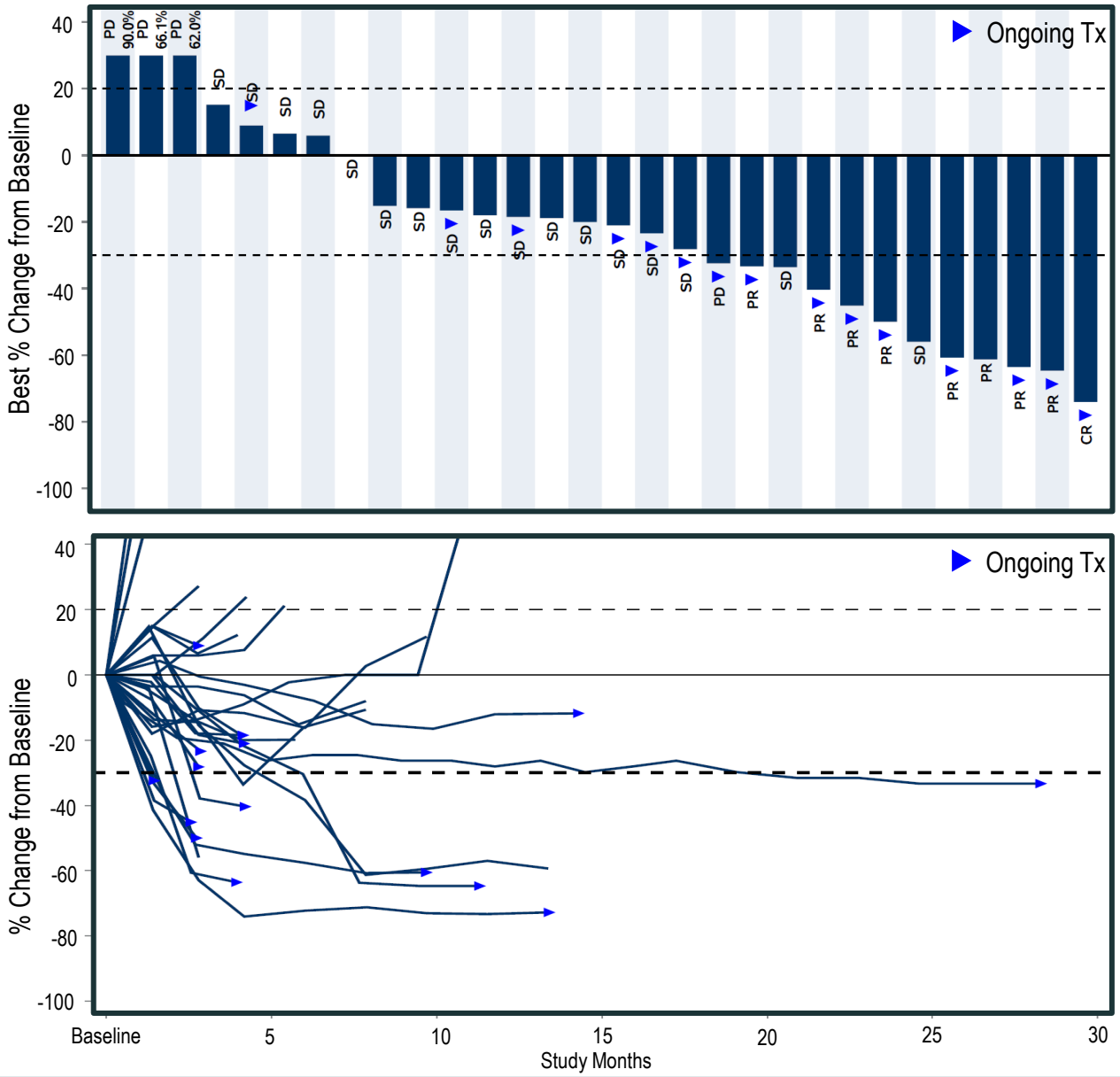
Transaminase kinetics by Dose (n=68)



Median of the per-subject maximum of ALT and AST (central laboratory), expressed as ×ULN, among subjects with an available result at each timepoint. Data cut-off: **01 September 2026.**

Pal SK, et al. Presented at ESMO Congress 2026; October 23–27, 2026; Madrid, Spain. Abstract 2684. **PD** priming dose **TD** target dose

Antitumor activity at target dose of 100 µg/kg

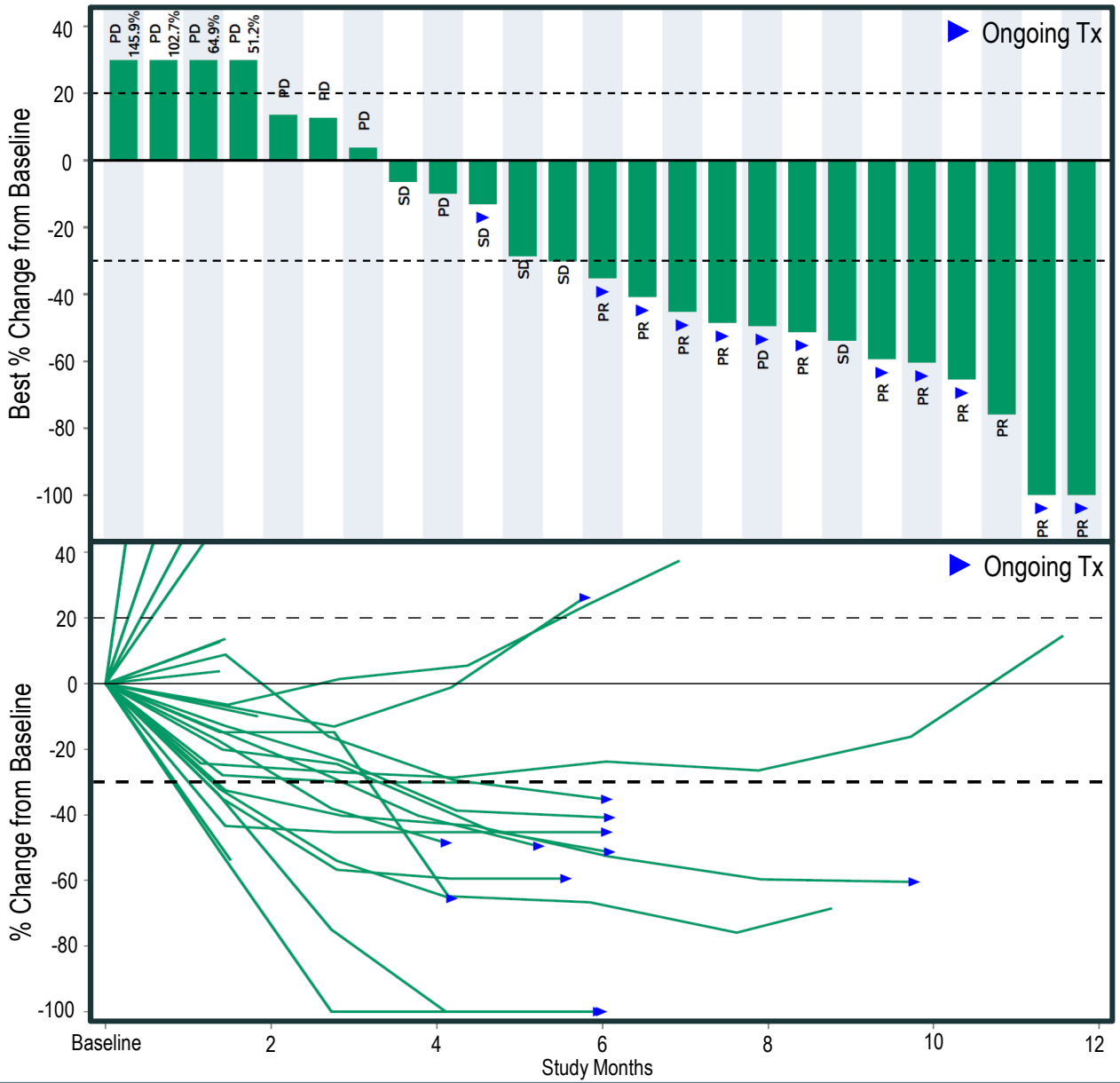


Efficacy Evaluable	100 µg/kg (n = 30)
Confirmed ORR, % (95% CI)	**30.0 (14.7, 49.4)**
BOR, n (%)	
CR	1 (3.3)
PR	8 (26.7)
SD	17 (56.7)
PD	4 (13.3)
DCR, % (95% CI)	86.7 (69.3, 96.2)
Median follow-up, months (range)	4.1 (1.1, 28.3)

Efficacy analysis includes subjects with ≥1 protocol-specified dose of XmAb819 and ≥1 post-baseline tumor assessment. Confirmed responses per investigator assessment, RECIST v1.1. Data cut-off: **01 September 2026.** CR subject has two resolved lymph node target lesions with residual mass <10 mm.

Pal SK, et al. Presented at ESMO Congress 2026; October 23–27, 2026; Madrid, Spain. Abstract 2684. **BOR** best overall response **CR** complete response **DCR** disease control rate **ORR** objective response rate **PD** progressive disease **PR** partial response **SD** stable disease **Tx** treatment

Antitumor activity at target dose of 200 µg/kg



Efficacy Evaluable[1]	200 µg/kg (n = 25)
Confirmed ORR, % (95% CI)	**44.0 (24.4, 65.1)**
BOR, n (%)	
CR	-
PR	11 (44.0)
SD	5 (20.0)
PD	9 (36.0)
DCR, % (95% CI)	64.0 (42.5, 82.0)
Median follow-up, months (range)	4.5 (0.8, 11.6)

Efficacy analysis includes subjects with ≥1 protocol-specified dose of XmAb819 and ≥1 post-baseline tumor assessment. Confirmed responses per investigator assessment, RECIST v1.1. Data cut-off: **01 September 2026.**

[1]Two subjects at 200 µg/kg discontinued treatment without a post-baseline response assessment and without documented progression.

Pal SK, et al. Presented at ESMO Congress 2026; October 23–27, 2026; Madrid, Spain. Abstract 2684. **BOR** best overall response **CR** complete response **DCR** disease control rate **ORR** objective response rate **PD** progressive disease **PR** partial response **SD** stable disease **Tx** treatment

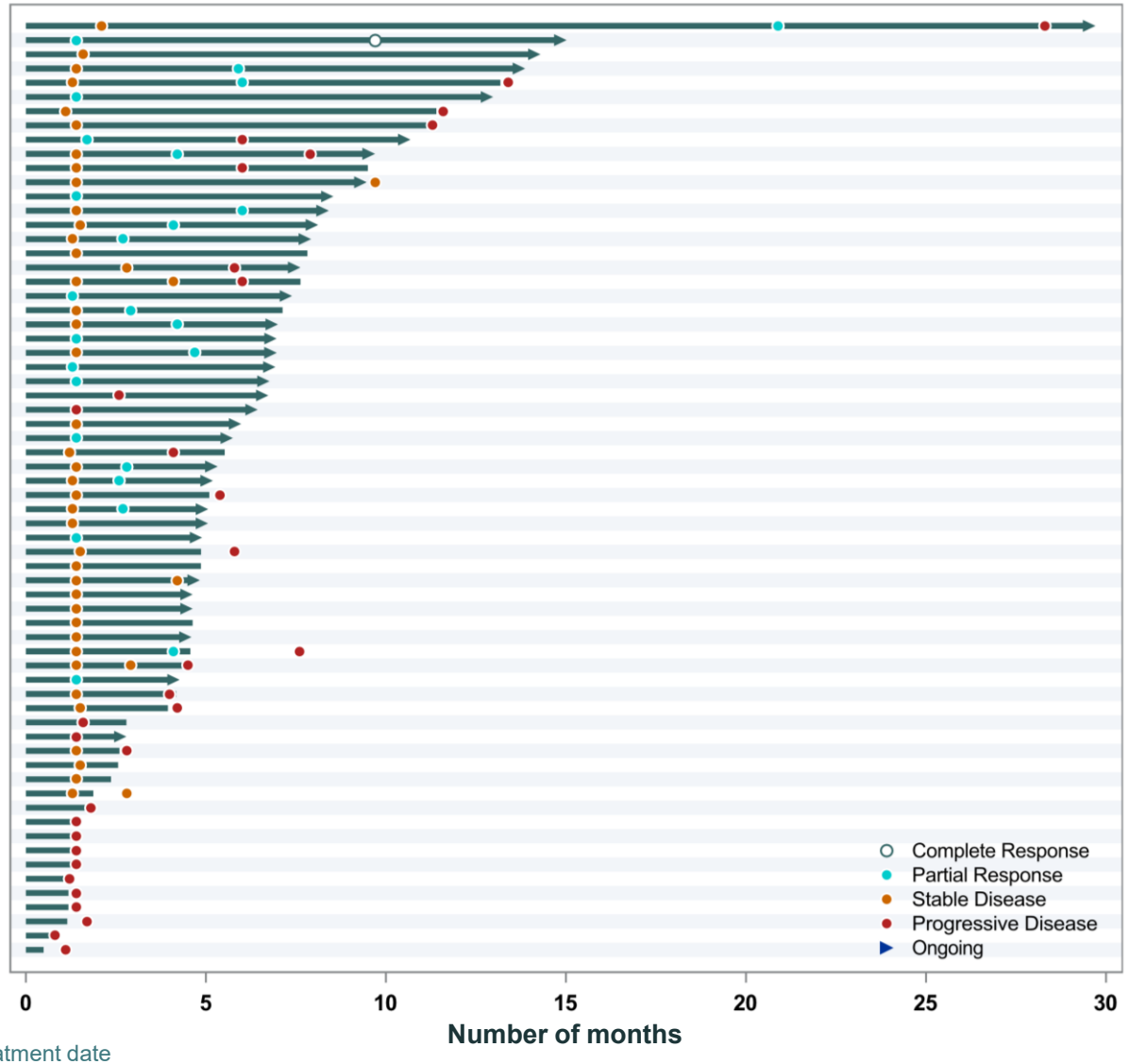
Durable disease control observed in heavily pre-treated advanced ccRCC population

7.9 month

median time-on-treatment[1]

Current follow-up for the consolidated IV target dose patient population (expanded doses: 100, 200 and 400 µg/kg)

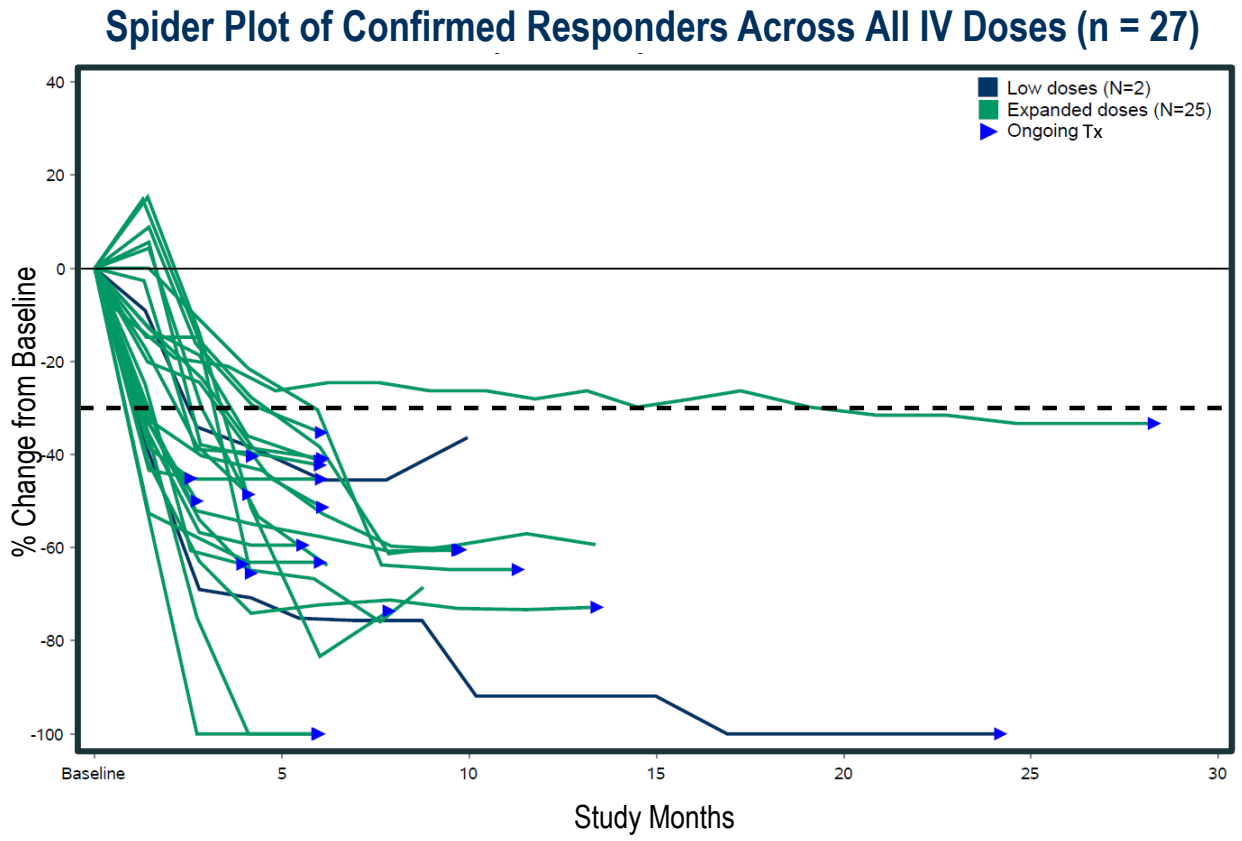
n=66; 95% CI: 5.8, 13.4



Data-cut: 01-Oct-2026 **1** Kaplan-Meier estimate of time-on-treatment defined as censor as of last treatment date or first event of progressive disease or death **ccRCC** clear cell renal cell carcinoma **CI** confidence interval

Deepening of response and new responses over time were common

- Consistent with sustained T-cell activation and low adverse event rate after priming period
- Combination of rapid disease control with potential for longer term deepening of response
- Response profile matured over several cycles: 25% of responders >4.2 months TTR

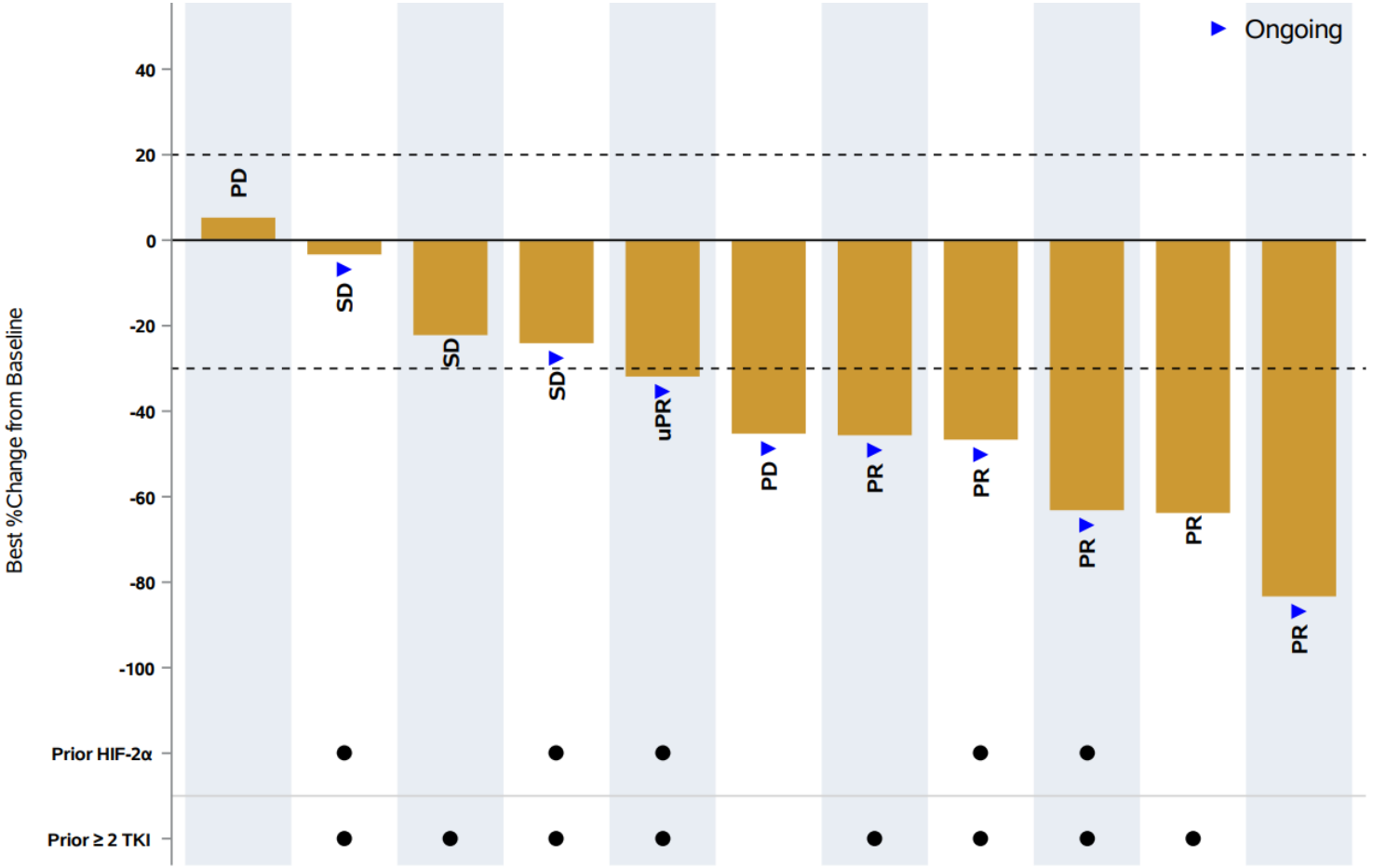


Time to Response (TTR)	% of Responders
>1.4 mo	75
>2.7 mo	50
>4.2 mo	25

TTR analysis for n=25 patients in Expanded Doses.
Data cut-off: **01 September 2026.**

400 µg/kg target dose is the proposed IV RP3D for XmAb819

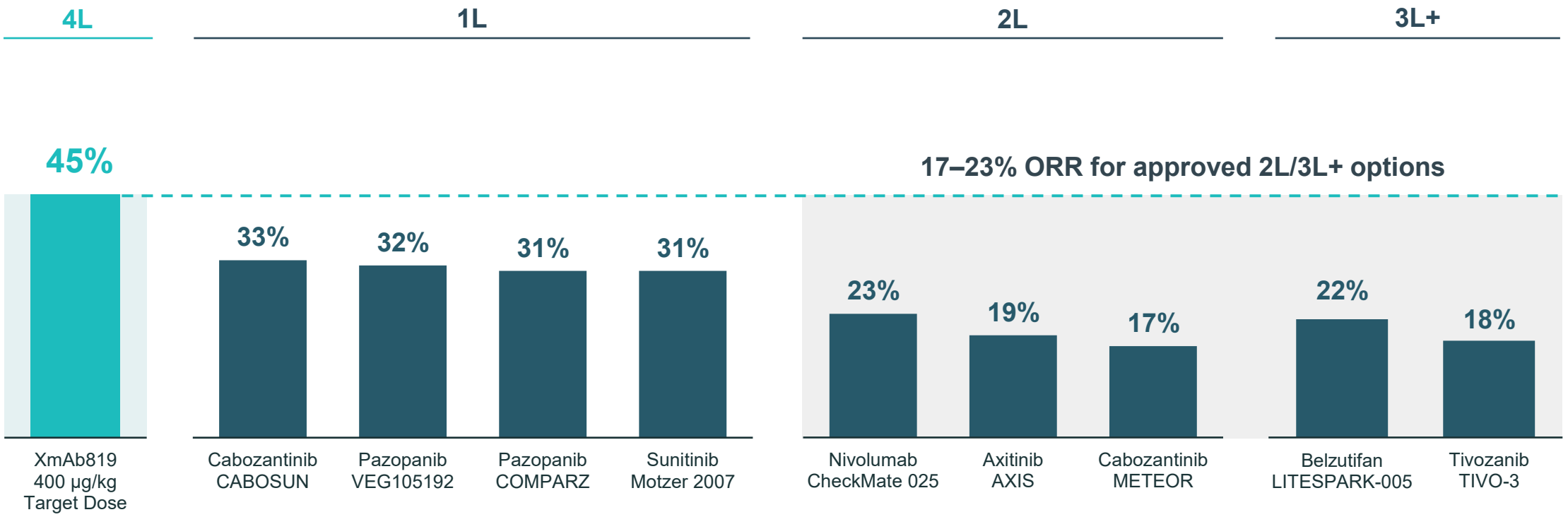
- 45% cORR plus additional week 42 conversion of a stable disease patient to uPR (uORR 55%)
- >80% DCR, low rate of primary progressive disease
- >70% of patients with treatment ongoing
- ~82% 6-month PFS K-M estimate at follow-up
- Median PFS is Not Reached
- Safety results: 400 µg/kg consistent with 200 µg/kg and 100 µg/kg target dose cohorts
- Enrollment complete in 400 µg/kg expansion cohort
- Data from the full 400 µg/kg IV analysis set expected during 1H2027 (20+ patients)



Data-cut: 01-Oct-2026 **cORR** confirmed objective response rate **DCR** disease control rate **IV** intravenous **K-M** Kaplan-Meier **PFS** progression free survival **RP3D** recommended Phase 3 dose **uORR** unconfirmed objective response rate **uPR** unconfirmed partial response

45% cORR in advanced ccRCC exceeds monotherapy benchmarks despite a more heavily pre-treated patient population

ORR for XmAb819 compared to NCCN guideline recommended monotherapy treatments for advanced ccRCC[1]



1 Cross-trial comparison for illustration only and is not based on head-to-head studies; trials differ in design, population and response assessment, and strong caution should be exercised when comparing data across unrelated trials. Comparators: **AXIS** Rini Lancet 2011 **CABOSUN** Choueiri JCO 2017; investigator-assessed, 20% by IRC **CheckMate 025** Motzer Cancer 2020 **COMPARZ** Motzer NEJM 2013; Motzer NEJM 2007 **LITESPARK-005** Choueiri NEJM 2024; 1–3 prior regimens **METEOR** Choueiri Lancet Oncol 2016 **TIVO-3** Rini Lancet Oncol 2020 **VEG105192** Sternberg JCO 2010, treatment-naïve subgroup **ccRCC** clear cell renal cell carcinoma **XmAb819** XmAb819-01 Phase 1, 400 µg/kg IV target dose (**Data cut Oct 01, 2026**) **cORR** confirmed objective response rate **NCCN** National Comprehensive Cancer Network **ORR** objective response rate

XmAb819 demonstrated favorable balance of response to primary progressive disease despite enrolling a poorer prognosis patient population

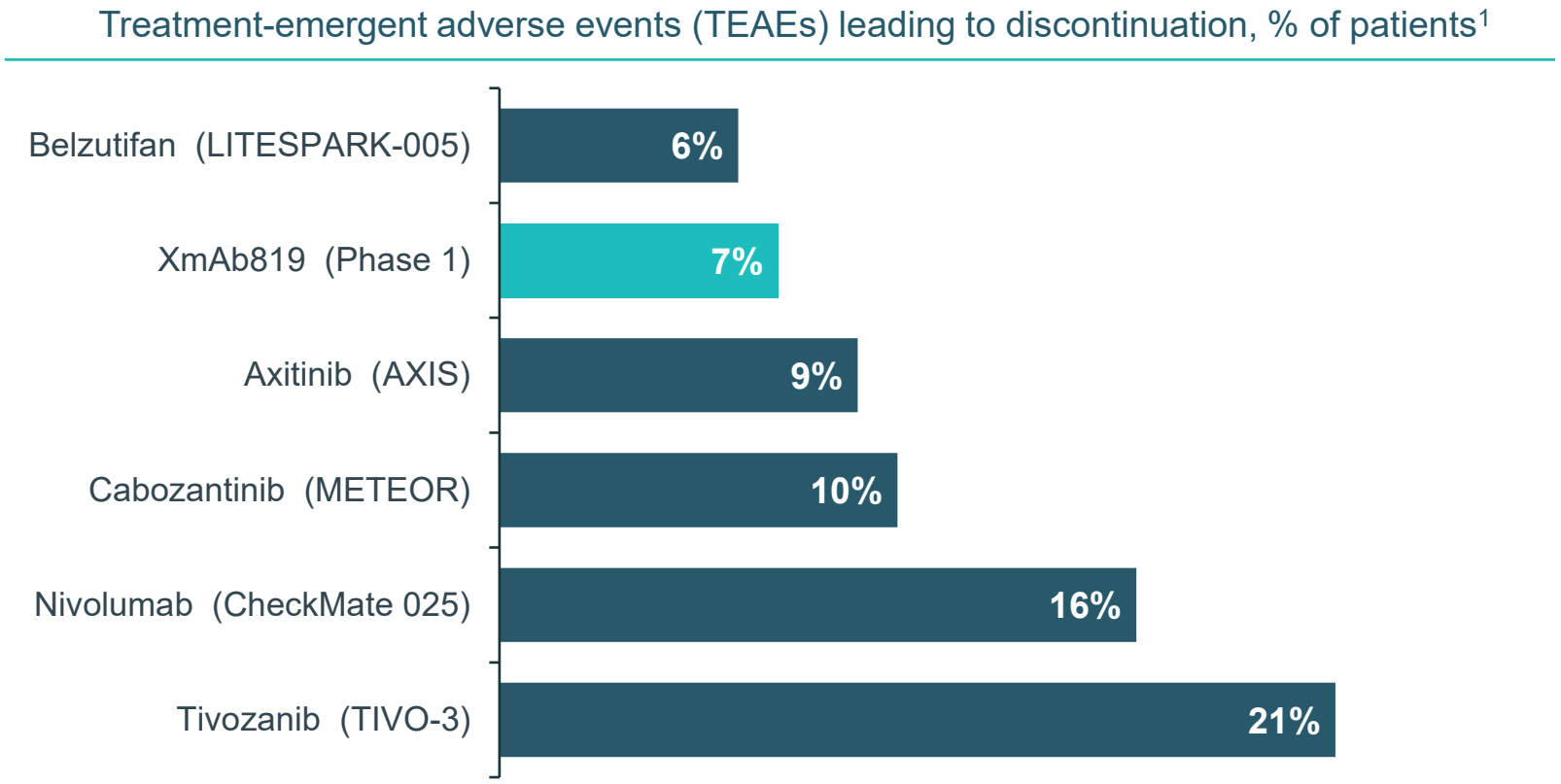
Agent / trial[1]	Intermediate-to-poor risk[2]	◄ Primary progression	Objective response ►	Net (ORR − PD)
XmAb819 Phase 1 study, 400 µg/kg IV	**82%**	18%	45%	**+27%**
Cabozantinib METEOR	57%	14%	17%	**+3%**
Axitinib AXIS	70%	22%	19%	**-3%**
Tivozanib TIVO-3	80%	22%	18%	**-4%**
Belzutifan LITESPARK-005	78%	34%	22%	**-12%**
Nivolumab CheckMate 025	65%	35%	23%	**-12%**

XmAb819 high objective response rate relative to primary progression supports earlier line development as a monotherapy treatment

1 Cross-trial comparison for illustration only and is not based on head-to-head studies; trials differ in design, population and response assessment, and strong caution should be exercised when comparing data across unrelated trials **2** Risk group per IMDC or MSKCC as reported by each trial Comparators: **AXIS** Rini Lancet 2011 **CheckMate 025** Motzer NEJM 2015 **LITESPARK-005** Choueiri NEJM 2024 **METEOR** Choueiri NEJM 2015 **TIVO-3** Rini Lancet Oncol 2020 **XmAb819** XmAb819-01 Phase 1, all treated patients (**Data-cut 01-Oct-2026**)

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XmAb819 was well tolerated with a low rate of discontinuations due to adverse events compared to third party studies of approved monotherapy ccRCC therapies

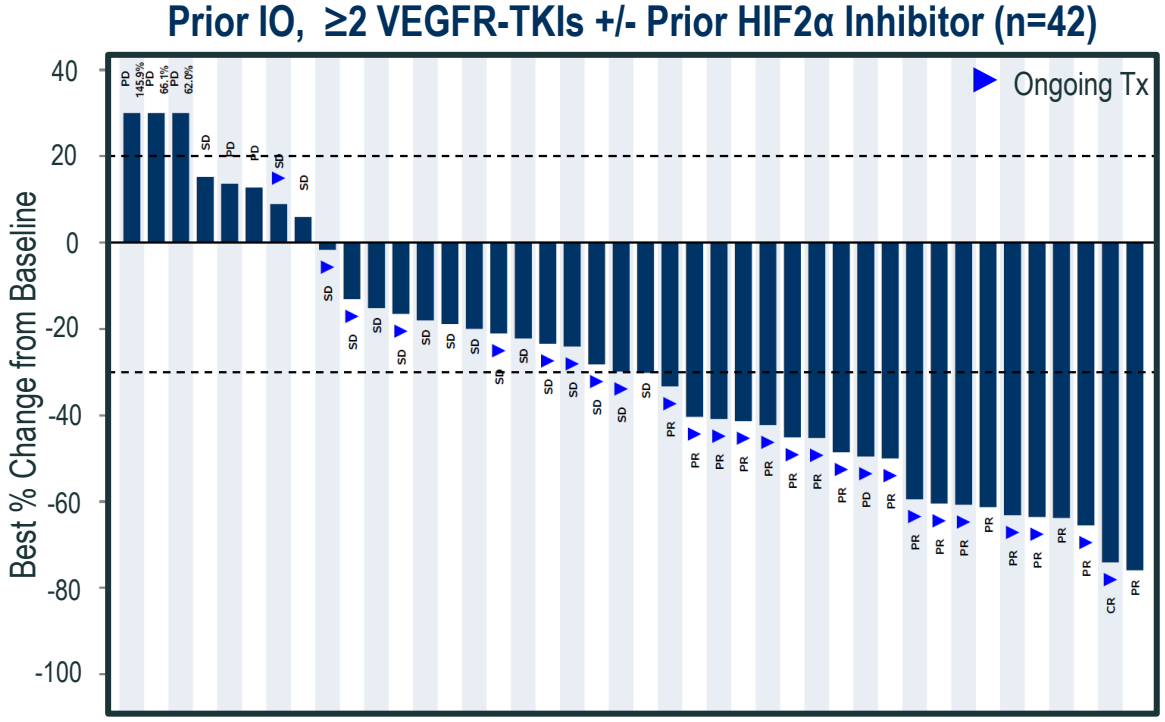


Fewer discontinuations compared with those reported in certain studies of most approved later-line therapies, with no dose reductions required for toxicity management at target dose

1 Cross-trial comparison for illustration only and is not based on head-to-head studies; trials differ in design, population, exposure duration and AE attribution (any-cause vs. treatment-related), and strong caution should be exercised when comparing data across unrelated trials. Comparators: **AXIS** Rini Lancet 2011 **CheckMate 025** Motzer NEJM 2015 **LITESPARK-005** Choueiri NEJM 2024 **METEOR** Choueiri NEJM 2015 **TIVO-3** Rini Lancet Oncol 2020 **XmAb819** XmAb819-01 Phase 1, all treated patients (**Data-cut 01-Oct-2026**) **ccRCC** clear cell renal cell carcinoma

Xencor plans to discuss accelerated approval pathway for XmAb819 based on efficacy results in key sub-groups

- No guideline-recommended treatments exist post PD-1/PD-L1 inhibitor plus ≥2 VEGFR-TKIs, or post PD-1/PD-L1 inhibitor, VEGFR-TKI, and HIF2α inhibitor (triple-class exposed)
- Estimated addressable population of ~9,200 US patients who were treated post PD-1/PD-L1 inhibitor and ≥2 prior VEGFR-TKIs in 2025[1]
- Triple-class exposed population is smaller but growing, driven by approval of belzutifan + lenvatinib post PD-1/PD-L1 inhibitor (LITESPARK-011)



Expanded Doses	Prior HIF2α (n = 23)	≥2 VEGFR-TKIs (n=42)
Confirmed ORR, % (95% CI)	**34.8 (16.4, 57.3)**	**45.2 (29.9, 61.3)**
DCR, % (95% CI)	78.3 (56.3, 92.5)	85.7 (71.5, 94.6)

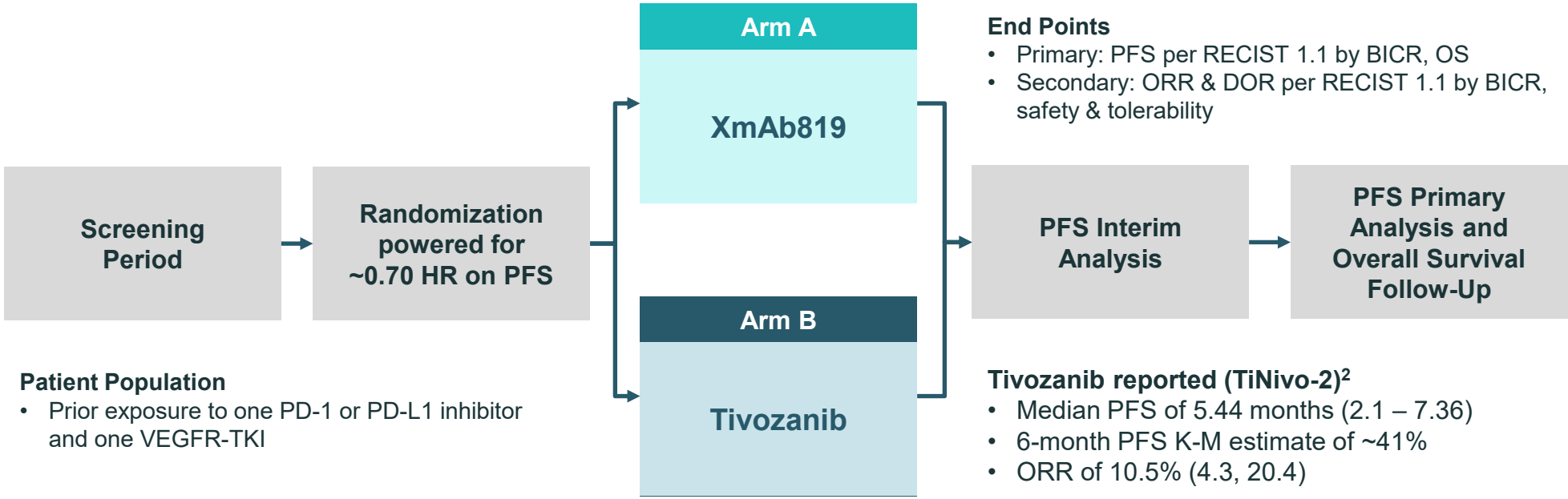
Efficacy analysis includes subjects with ≥1 protocol-specified dose of XmAb819 and ≥1 post-baseline tumor assessment. Confirmed responses per investigator assessment, RECIST v1.1. Data cut-off: **01 September 2026.** CR subject has two resolved lymph node target lesions with residual mass <10 mm.

DCR, disease control rate; **HIF2α,** hypoxia-inducible factor 2-alpha; **ORR,** objective response rate; **TKI,** tyrosine kinase inhibitor; **Tx**, treatment; **VEGFR,** vascular endothelial growth factor receptor

1 Komodo Health claims data; nationally weighted U.S. patient estimates for 2025

Global randomized control trial with a tivozanib control arm could potentially support BLA submission seeking a post IO-TKI label[1]

Proposed Phase 3 study design to evaluate XmAb819 compared to tivozanib in post IO-TKI patient population



End Points
- Primary: PFS per RECIST 1.1 by BICR, OS
- Secondary: ORR & DOR per RECIST 1.1 by BICR, safety & tolerability

Patient Population
- Prior exposure to one PD-1 or PD-L1 inhibitor and one VEGFR-TKI

Tivozanib reported (TiNivo-2)[2]
- Median PFS of 5.44 months (2.1 – 7.36)
- 6-month PFS K-M estimate of ~41%
- ORR of 10.5% (4.3, 20.4)

3L sub-group analysis of TiNivo-2 provides a basis for expectations of standard dose tivozanib outcomes in a contemporary post IO-TKI patient population

1 Study design and registrational potential subject to further interactions with FDA **2** Final analysis of the TiNivo-2 phase 3 trial: Long-term outcome of Tivozanib (Tivo) in patients with metastatic renal cell carcinoma (mRCC); Motzer and Choueiri et al; ASCO 2026. **BICR** blinded independent central review **BLA** biologics license application **DOR** duration of response **HR** hazard ratio **IO** immuno-oncology **K-M** Kaplan-Meier **ORR** objective response rate **OS** overall survival **PFS** progression free survival **QD** every day **RECIST** response evaluation criteria in solid tumors **TKI** tyrosine kinase inhibitor **VEGFR** VEGF receptor

Development plan for XmAb819 to become the backbone of ccRCC treatment

Establish XmAb819 Monotherapy Post IO-TKI

Phase 1 Part	Description	Patient population	U.S. Patient opportunity[1]	Status	Expected Milestone or Readout
A & B	IV monotherapy	**Post-IO + TKI**	**~23.9K**	Ongoing	End of Phase 1 meeting 1H 2027

Post Front-line IO-IO for IMDC Intermediate – Poor Risk Patients

Phase 1 Part	Description	Patient population	U.S. Patient opportunity[1]	Status	Expected Milestone or Readout
D	2L ccRCC	**Post-1L nivolumab/ipilimumab (TKI-naïve)**	**~9.4K**	Ongoing	Proof of concept data 2027

PD-1 Proof-of-concept Combination for Future 1L study

Phase 1 Part	Description	Patient population	U.S. Patient opportunity[1]	Status
F	Nivolumab combination	**1L opportunity for novel IO-TCE combination**	**~30.1K**	Planned

Increase Clinical Convenience

Phase 1 Part	Description	Patient population	Status	Expected Milestone or Readout
A & B	SC monotherapy	Post-IO + TKI	Ongoing	End of Phase 1 meeting 1H 2027
A & E	Q3W IV / reduced hospitalization	Post-IO + TKI	Planned	–

1 Komodo Health claims data; nationally weighted U.S. patient estimates for 2025 **ccRCC** clear cell renal cell carcinoma **IO** immuno-oncology **IV** intravenous **TCE** T-cell engager **TKI** tyrosine kinase inhibitor **SC** subcutaneous **Q3W** every 3 weeks **1H** first-half

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Global RCC drug market is an estimated ~$11.7B annualized opportunity

TKI Class	IO Class	HIF2α Class
~$5B[1]	~$5.6B[2]	~$1.1B[3]
cabozantinib, lenvatinib, axitinib, sunitinib, pazopanib, tivozanib	nivolumab, pembrolizumab, ipilimumab, avelumab	belzutifan



1 Estimate based on 2025 cabozantinib net product revenue and RCC market share internal estimates from Xencor **2** Estimate based on annualized Q2'26 global nivolumab sales and Komodo RCC market claims data **3** Estimate based on annualized Q2'26 global belzutifan sales **IO** immuno-oncology **RCC** renal cell carcinoma **TKI** tyrosine kinase inhibitor

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ENPP3+ expansion programs add significant development opportunities

U.S. 2030 TAM in ENPP3+[1] papillary RCC, MSS colorectal cancer and NSCLC **+$4.5B**



2L/3L Papillary RCC

U.S. 2030 TAM

~$0.5B

> 50% ENPP3+[1]

SoC: cabozantinib



3L MSS Colorectal Cancer

U.S. 2030 TAM

+$1B

> 50% ENPP3+[1]

SoC: regorafenib, fruquintinib or FTD/TPI + bevacizumab



2L/3L NSCLC

U.S. 2030 TAM

+$3B

> 30% ENPP3+[1]

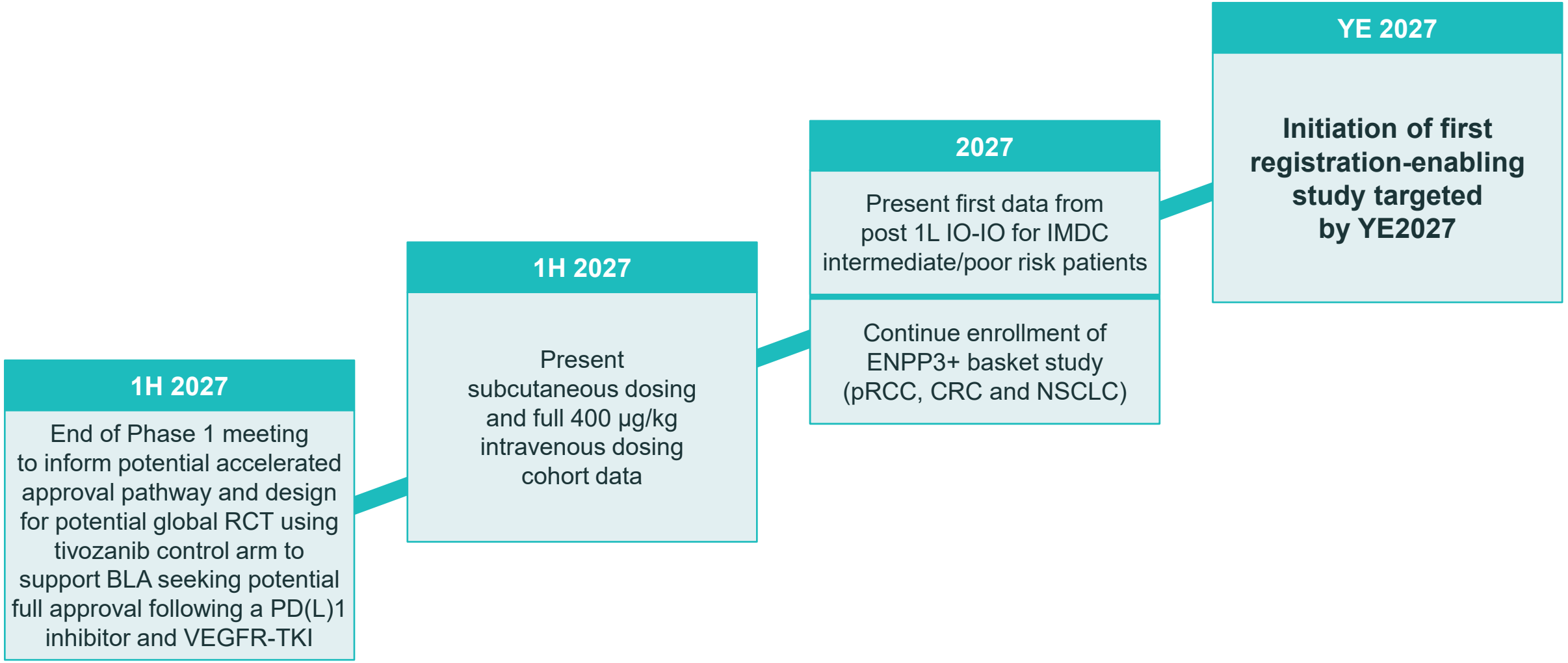
SoC: docetaxel post IO and/or targeted therapy for actionable mutations

Enrolling patients with pRCC, MSS CRC and NSCLC in Part C of the ongoing Phase 1 study

1 Estimates for U.S. ENPP3+ population based on H-Score of ≥25 cutoff **CRC** colorectal cancer **FTD/TPI** trifluridine/tipiracil **IO** immuno-oncology **MSS** microsatellite stable **NSCLC** non-small cell lung cancer **SoC** standard of care **TAM** total addressable market

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Expected 2027 plans include first potential registration-enabling study in ccRCC and further data from ongoing Phase 1 study cohorts



As of 04-Nov-2026. **ccRCC** clear cell renal cell carcinoma **CRC** colorectal cancer **IMDC** International mRCC Database Consortium **IO** immuno-oncology **NSCLC** non small cell lung cancer **pRCC** papillary renal cell carcinoma **RCT** randomized control trial **TKI** tyrosine kinase inhibitor **VEGFR** VEGF receptor **YE** year-end **1H** first-half

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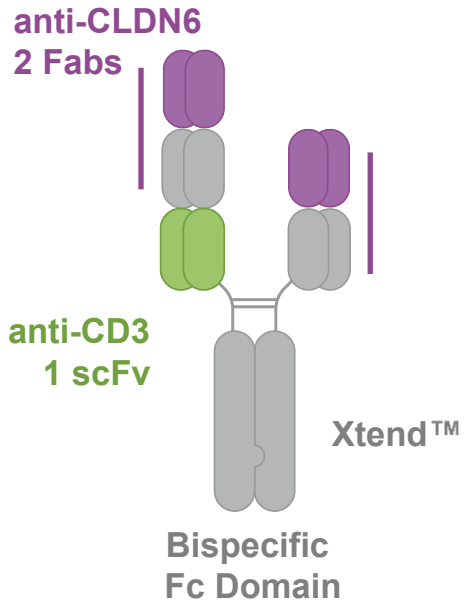
XmAb® Bispecific T-Cell Engagers

XmAb541 (CLDN6 x CD3)
+ XmAb808 (B7-H3 x CD28)

Phase 1 in CLDN6+ HGSOC

XmAb541 (CLDN6 x CD3) development has been prioritized in combination with XmAb808 (B7-H3 x CD28)

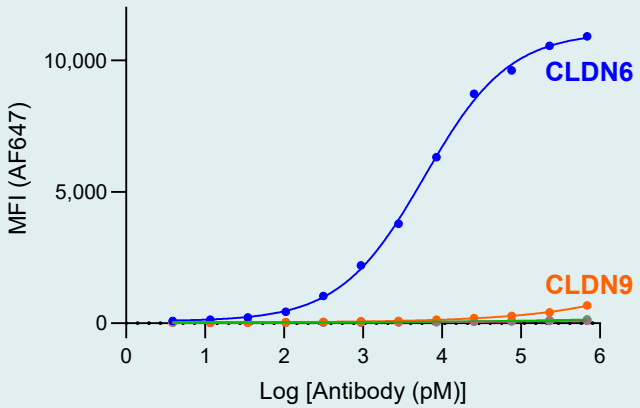
XmAb541 engages the immune system and activates T cells for highly potent and targeted lysis of tumor cells expressing CLDN6



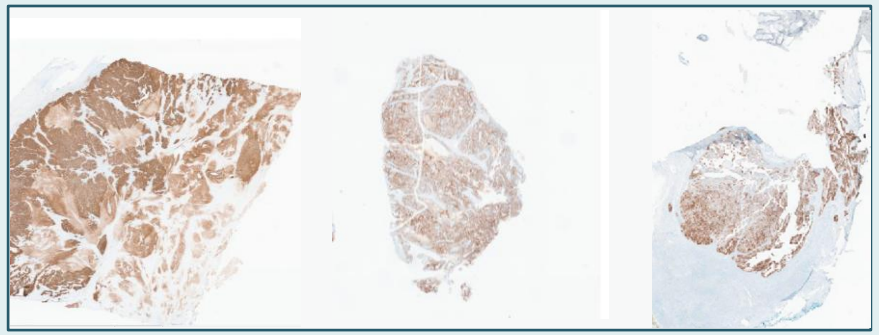
CLDN6 *Claudin-6*

- Differential expression in cancerous tissue presents CLDN6 as an intriguing target
- CLDN family members, which are small membrane proteins, have high sequence identity, complicating antibody design

Highly selective for CLDN6 over CLDN9



Representative IHC of CLDN6



GCT | Endometrial | Ovarian

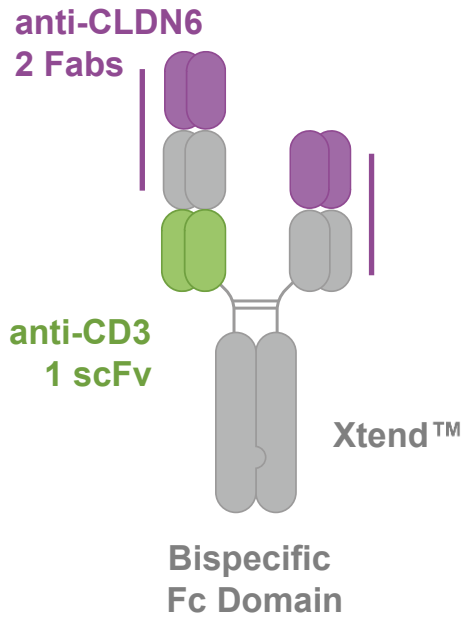
- XmAb541 engineered for CLDN6 selectivity over similar CLDN9, CLDN3 and CLDN4

GCT germ cell tumor **MFI** mean fluorescence intensity **CDx** companion diagnostic **IHC** immunohistochemistry

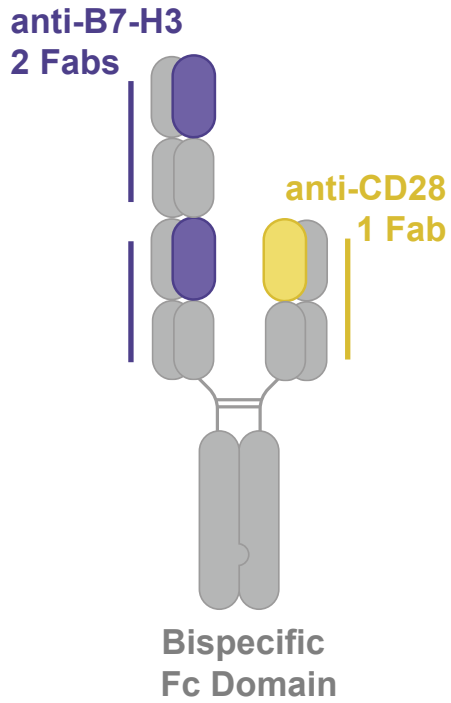
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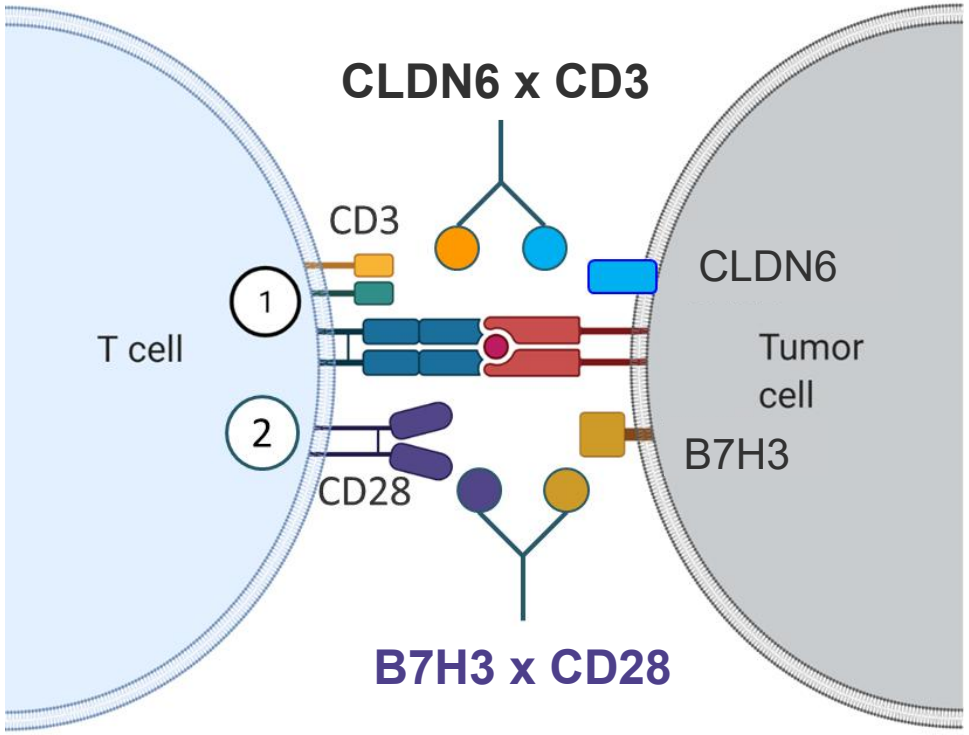
XmAb541 (CLDN6 x CD3) + XmAb808 (B7-H3 x CD28) in ovarian cancer

XmAb541 activates
T cells via **Signal 1** provided by
CD3 engagement



XmAb808 is designed to deliver tumor-targeted **Signal 2**,
CD28 co-stimulation, to complement Signal 1





XmAb541 monotherapy data support XmAb541 + XmAb808

Phase 1 dose-escalation study of XmAb541

- At the putative RP3D, emerging monotherapy data show clinical activity in heavily pretreated patients
  - ~14% ORR in ovarian cancer
  - ~28% ORR in germ cell tumors
- Additional anti-tumor activity was observed at doses above 60 mg, but reversible hearing impairment limited exposure due to dose interruptions and reductions
  - Hearing impairment is potentially on-target for CLDN6, which is expressed on cochlear hair cells
- Cytokine release syndrome (CRS) has been low grade, with no Grade ≥3 CRS reported at any dose level; at the putative RP3D, Grade 1 CRS was observed in ~14% of patients and Grade 2 CRS in ~17%
- No other clinically significant safety signals have been observed, and the overall profile supports future outpatient administration

Based on monotherapy data, the U.S. FDA granted Fast Track designation to XmAb541 for the treatment of germ cell tumors in patients who have relapsed following two or more lines of platinum therapy or were refractory to prior platinum therapy.

XmAb541 Monotherapy Study Status

Monotherapy expansion cohorts at the putative RP3D (60 mg Q3W) in high-grade serous ovarian carcinoma (TPS ≥50) and germ cell tumors are expected to complete enrollment by year-end 2026

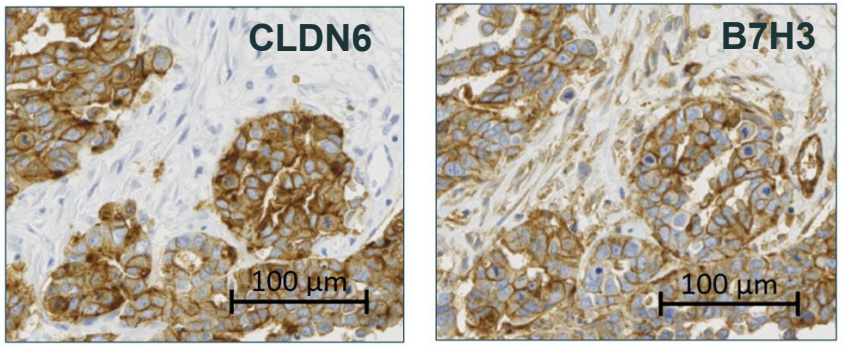
XmAb541 + XmAb808 Combination Study Status

The Phase 1 dose-escalation study of XmAb541 and XmAb808 is ongoing and is expected to reach the combination's target dose range in 2027

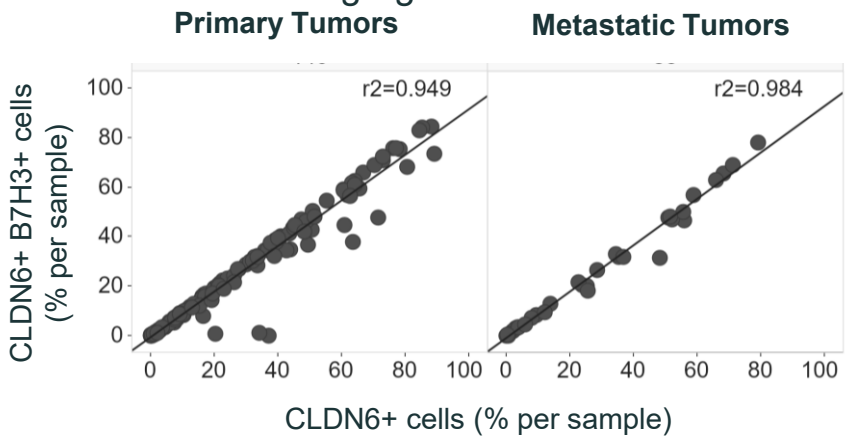
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XmAb® CD28 bispecific antibodies provide a tumor-specific activation boost to CD3 bispecifics

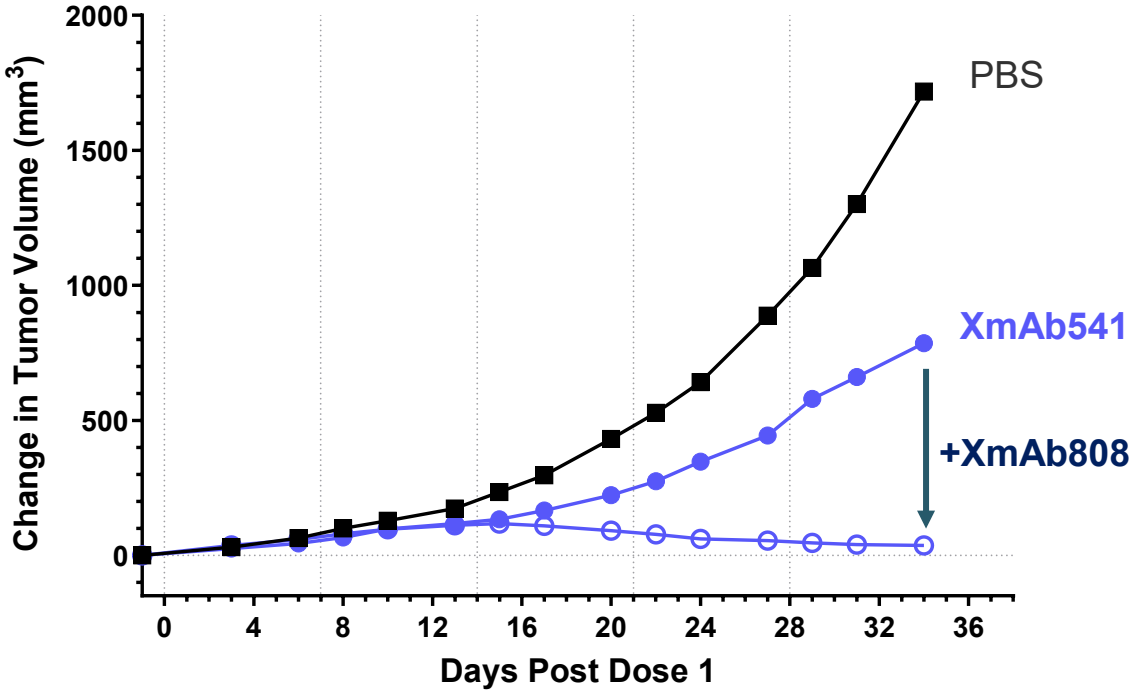
CLDN6 and B7H3 are co-expressed in ovarian tumors



Ovarian High-grade Serous Carcinoma



XmAb808 (B7H3 x CD28) enhances activity of XmAb541 *in vivo*



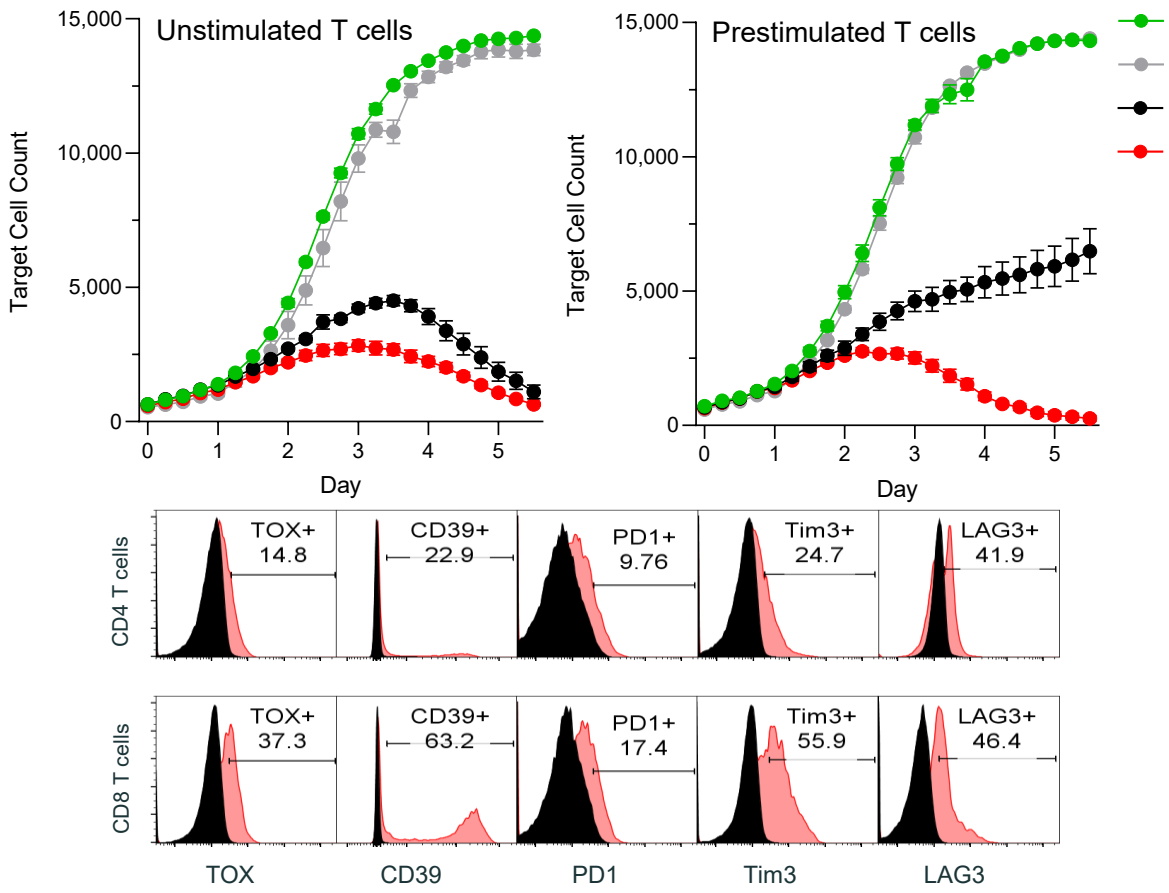
0.03 mg/kg CLDN6 x CD3

CLDN6 and B7-H3 have low expression overlap on normal tissues, potentially localizing T-cell co-stimulation to tumor cells and supporting the ongoing clinical evaluation of the XmAb541 + XmAb808 combination.

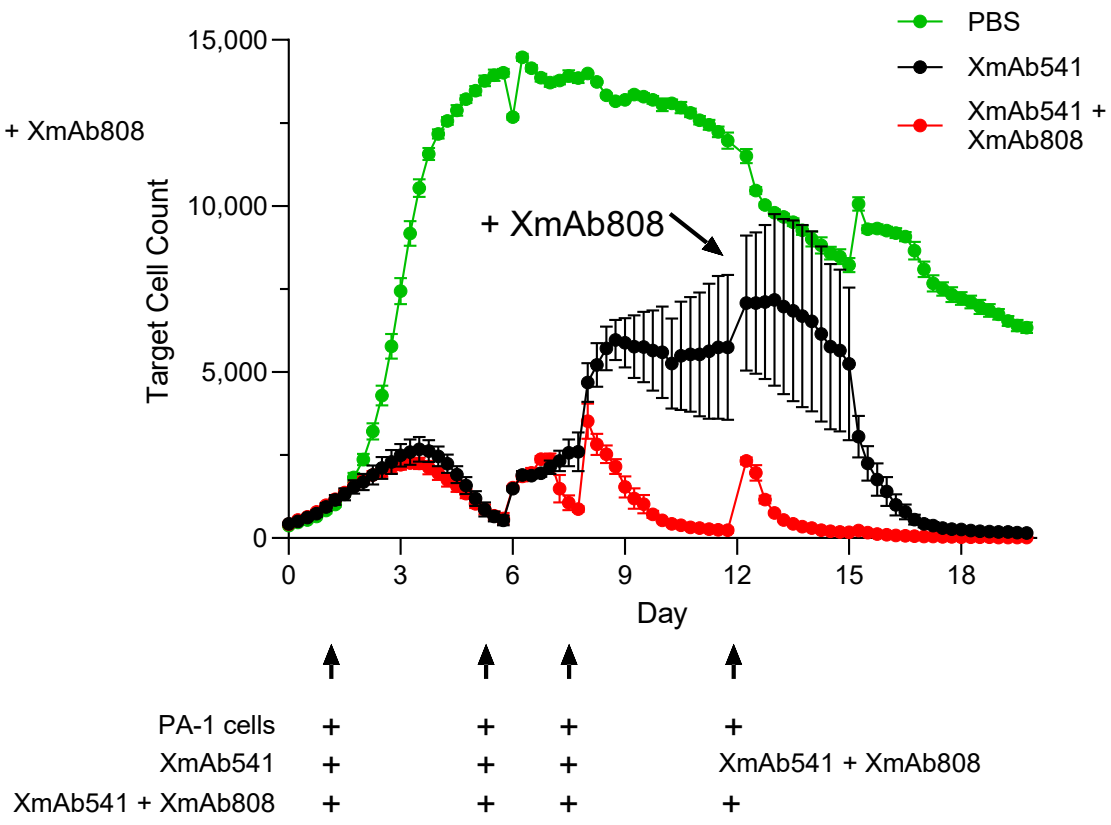
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XmAb808 resists T-cell exhaustion and promotes durable killing by XmAb541 *in vitro*

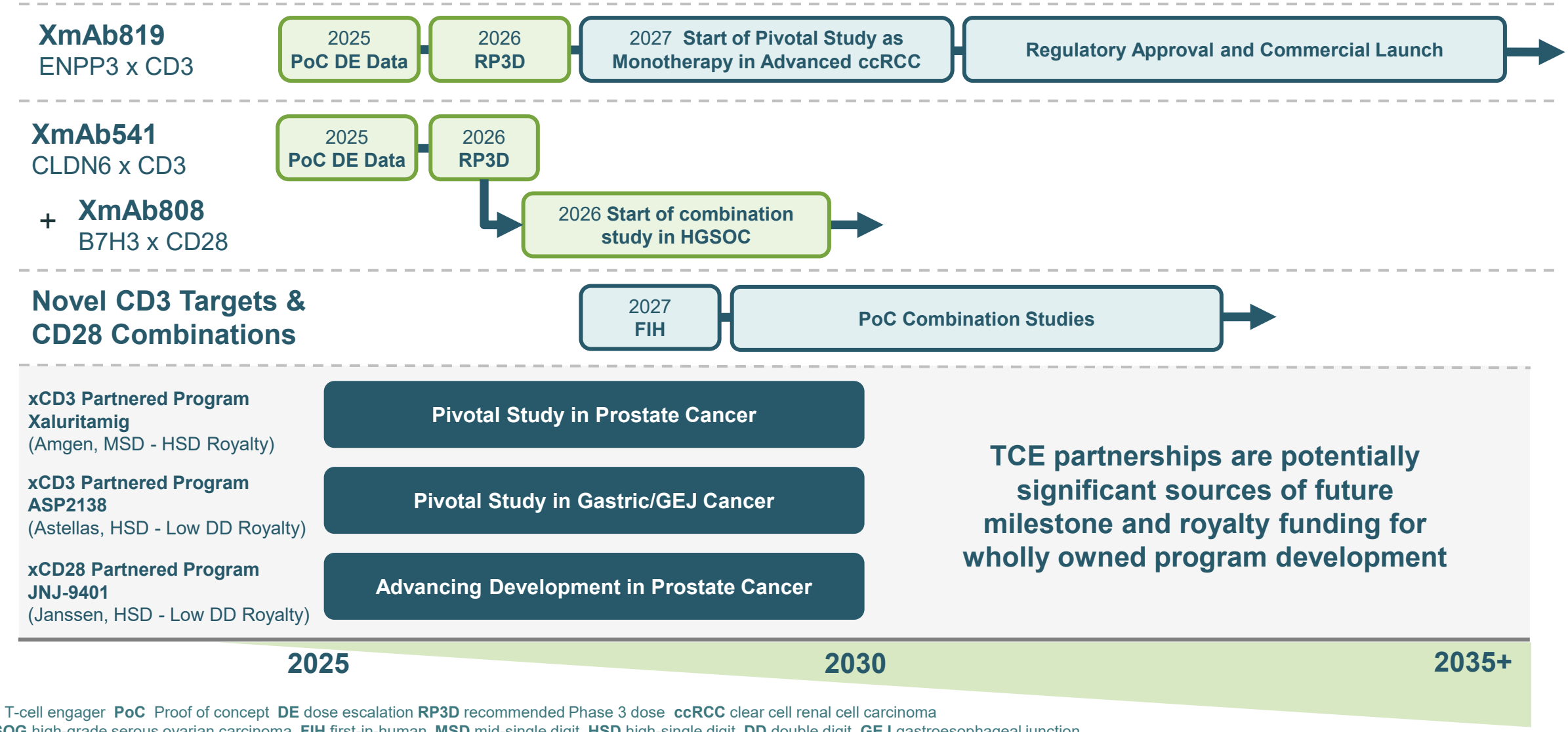
Combination re-activates exhausted T cells



Combination overcomes drug-induced resistance



Integrated pipeline of next generation precision immunotherapy



TCE T-cell engager **PoC** Proof of concept **DE** dose escalation **RP3D** recommended Phase 3 dose **ccRCC** clear cell renal cell carcinoma
HGSOG high-grade serous ovarian carcinoma **FIH** first-in-human **MSD** mid-single digit **HSD** high-single digit **DD** double digit **GEJ** gastroesophageal junction

XmAb® TL1A Pipeline

XmAb942 (Xtend™ TL1A)

Ongoing Phase 2b in Ulcerative Colitis

XmAb412 (TL1A x IL-23p19)

Ongoing Phase 1 Study in Healthy Participants

Xencor is positioned with best-in-class drug candidates targeting TL1A in large and growing market for biologic therapies to treat IBD

$24.6b Global annual IBD spend by 2030[1]

3.3m Estimated IBD diagnoses in the U.S. and EU (CD + UC)

1/3 Proportion of the advanced therapy market addressable by TL1A mechanism[2]

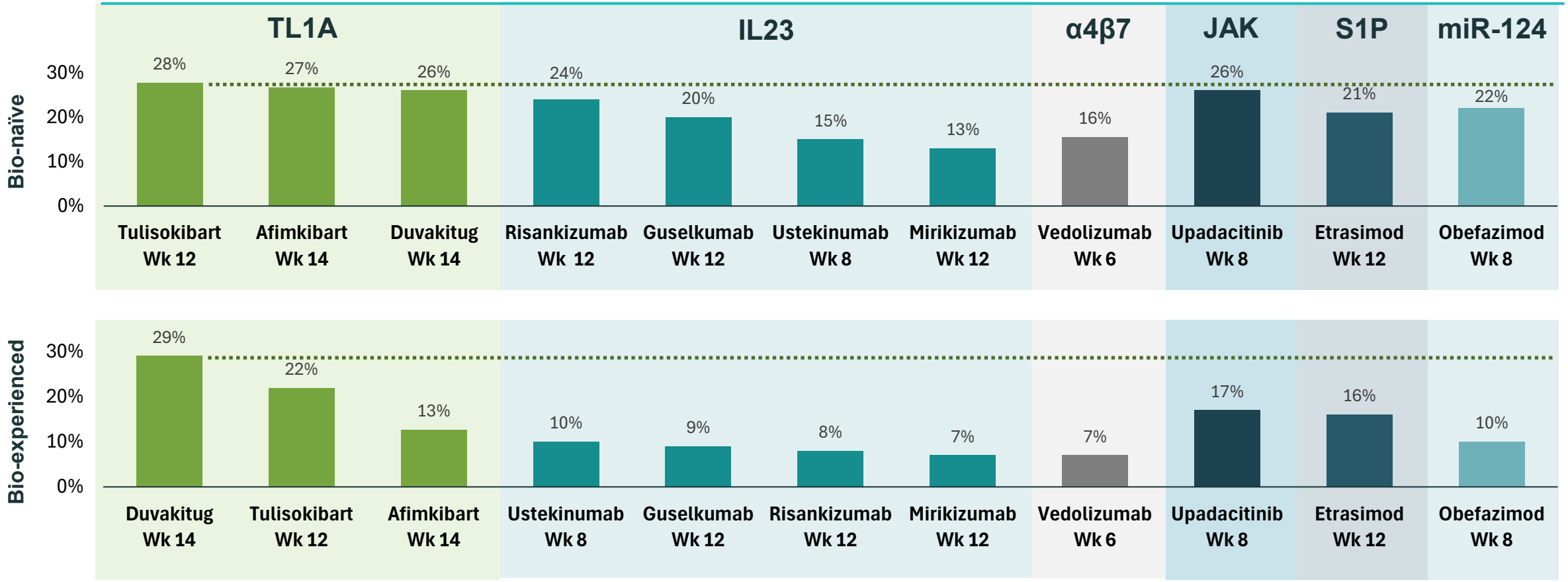
9/10 Gastroenterologists expect anti-TL1A as 1st or 2nd line[3]

Program	Target	2025 Net Sales (All Indications)[1]
Top biologics indicated for treating moderately to severely active UC + CD		
Skyrizi® (risankizumab)	IL-23p19	**$17.6b**
Entyvio® (vedolizumab)	α4β7	**$6.4b**
Stelara® (ustekinumab)	IL-23p40	**$6.1b**
Tremfya® (guselkumab)	IL-23p19	**$5.2b**
Omvoh® (mirikizumab)	IL-23p19	**$0.3b**
Emerging class of first generation anti-TL1A biologics		
Afimkibart Tulisokibart Duvakitug	TL1A	Ongoing Phase 3 studies
Xencor biologics designed to improve clinical efficacy and clinical convenience		
XmAb942	TL1A	Ongoing XENITH-UC Phase 2b study
XmAb412	TL1AxIL23p19	FIH study start 3Q26

1 GlobalData. Projected peak IBD sales are illustrative estimates; clinical data pending. **2** Xencor-sponsored survey; ~34% patients with moderately to severely UC eligible for anti-TL1A therapy per Phase 2 data
3 Xencor-sponsored survey of 27 U.S.-based gastroenterologists **4** Company earnings reports

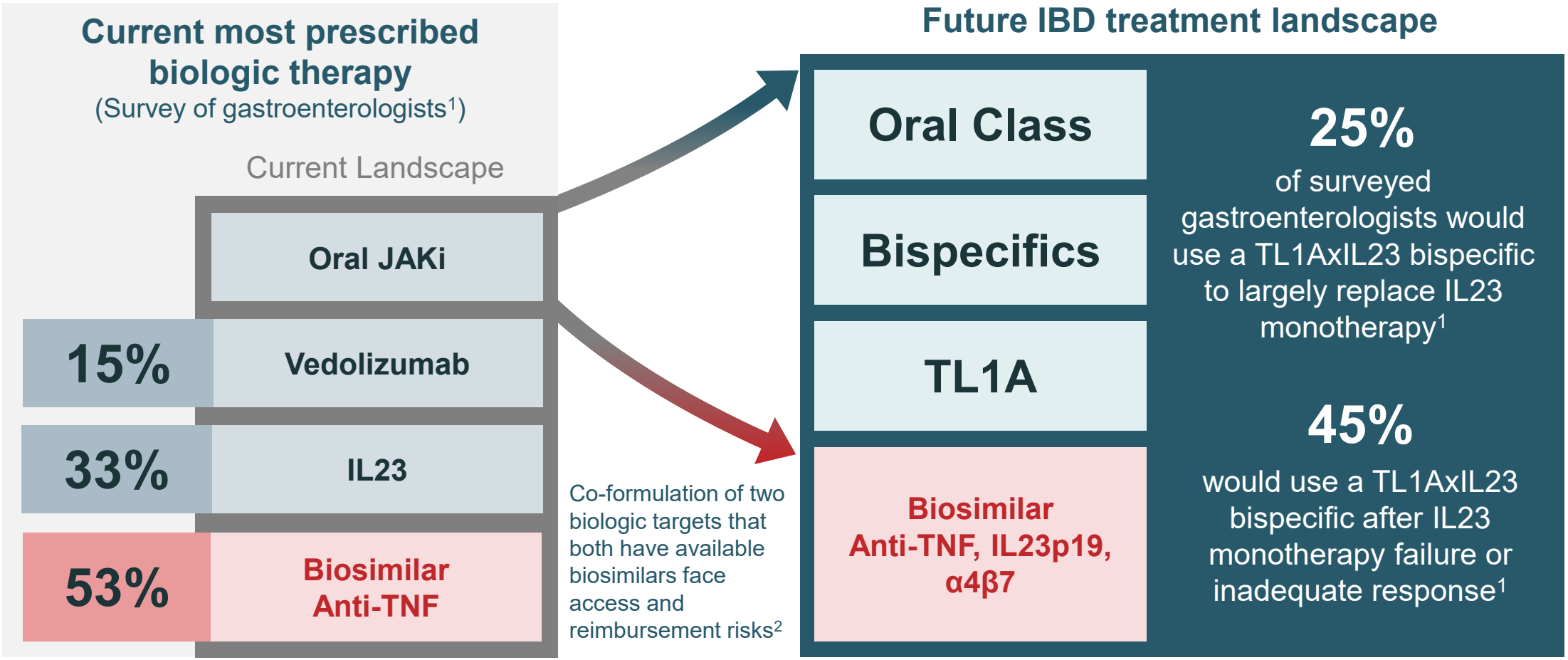
More than half of patients with moderately to severely active UC do not adequately respond to biologic therapy

Advanced therapy clinical remission rates in ulcerative colitis (placebo adjusted)



Source: **Tulisokibart** Sands BE, et al, N Engl J Med. 2024 Sep 26;391(12):1119-1129. **Afimkibart** Danese S, et al, The Lancet Gastroenterology & Hepatology, 2025; 10, 882-895. **Duvakitug** Reinisch W, et al, Journal of Crohn's and Colitis, Volume 19, Issue Supplement_1, January 2025, Pages i79–i80. Per FDA labels for Skyrizi® (**risankizumab**), Tremfya® (**guselkumab**), Stelara® (**ustekinumab**), Omvoh® (**mirikizumab**), Rinvoq® (**upadacitinib**), Velsipity® (**etrasimod**) **Vedolizumab** Feagan BG, et al. Clinical Gastroenterology and Hepatology. Volume 15, Issue 2, February 2017, Pages 229-239.e5. **Obefazimod** Sands BE, et al. UEG Week 2025, Berlin.

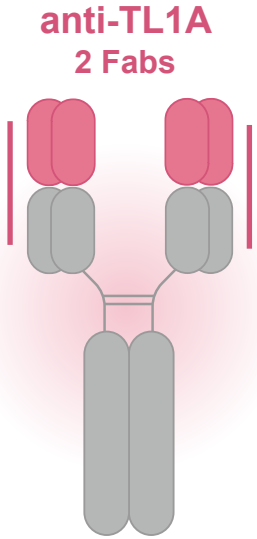
Xencor's TL1A clinical strategy is aligned with the future of IBD treatment



1 Xencor-sponsored survey of 40 U.S.-based gastroenterologists covering treatment of >9000 patients with IBD annually **2** Per AbbVie 2025 10-K: The United States composition of matter patents covering risankizumab are expected to expire in 2033 (AbbVie 2025 10-K). ENTYVIO (vedolizumab) will face loss of regulatory exclusivity in the latter half of this decade and certain patents covering various aspects of this product are expected to expire in 2032 (Takeda 2025 Annual report).

XmAb® protein engineering enables differentiated, potentially best-in-class treatment options for autoimmune and inflammatory diseases

Design Objectives for a **Novel Next-Gen Anti-TL1A**



Xtend + FcKO

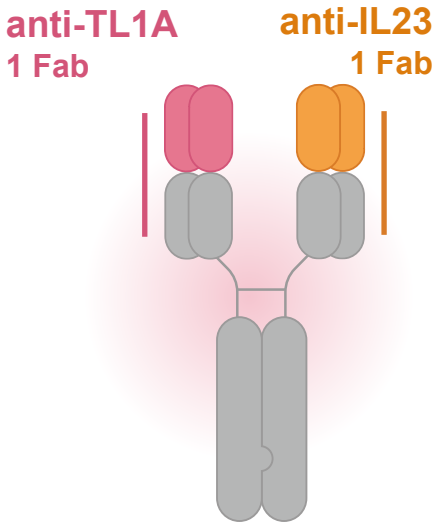
Class-leading potency for superior inhibition of TL1A within the GI tract

XmAb stability and solubility engineering for high concentration formulation and lower immunogenicity risk

Long half-life from Xtend™ Fc domain designed to enable extended subcutaneous dosing intervals in maintenance

XmAb942 (anti-TL1A)

Design Objectives for a **Novel TL1A x IL-23p19 Bispecific**



XmAb Bispecific Fc Domain

Xtend + FcKO

Highly stable monovalent format to allow subcutaneous formulation and avoid large immune complexes

Very high affinity TL1A and IL23p19 binders to deliver equivalent target inhibition as monospecific antibodies

- Blocking IL23p19 gives consistently superior clinical outcomes across indications versus IL23p40[1,2]

Long half-life from Xtend Fc domain

XmAb412 (TL1A x IL23p19)

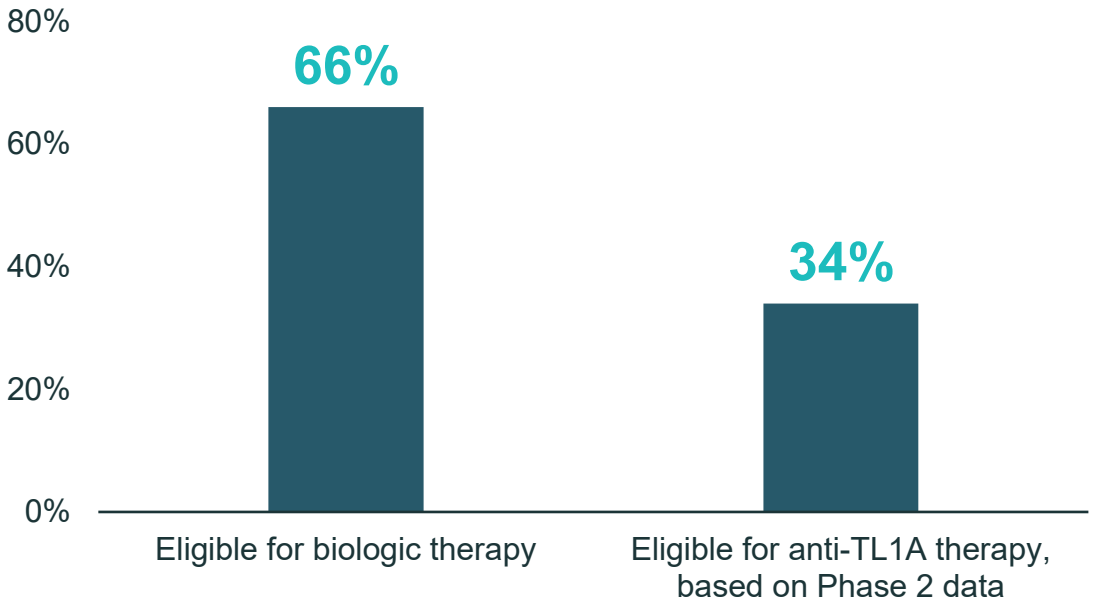
1 Week 47 Efficacy of Guselkumab and Ustekinumab in Crohn's Disease Based on Prior Response/Exposure to Biologic Therapy: Results from the GALAXI 2 & 3 Phase 3 Studies; Danese and Rubin et al; JNJ Presentation. **2** Comparing Efficacy of Guselkumab versus Ustekinumab in Patients with Psoriatic Arthritis: An Adjusted Comparison Using Individual Patient Data from the DISCOVER and PSUMMIT Trial; Thilakarathne and Hassan et al.; Rheumatol Ther. 2024 Feb 28.

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XmAb942 positioned as best-in-class anti-TL1A mAb in a rapidly expanding market targeting TL1A across autoimmune and inflammatory diseases

TL1A Expected to be Cornerstone of Advanced Therapy in IBD & TAM Expanding to AID Broadly

Anti-TL1A class could capture ~1/3 of total market for advanced therapy in IBD[1]



Multi-indication immune franchise opportunities with several ongoing proof-of-concept studies representing ~$86b in global TAM by 2030E[12]

Indication	Trial sponsors[11]
Moderate-to-severe rheumatoid arthritis[3,4,5]	■ ■ ■
Moderate-to-severe atopic dermatitis[6]	■
Advanced MASH liver fibrosis[8]	■
Axial spondyloarthritis[3,9]	■ ■
Psoriatic arthritis[3,10]	■ ■
Hidradenitis suppurativa[7]	■ ■

1 Percent of moderately to severely active UC patients; Xencor-sponsored survey of 27 U.S.-based gastroenterologists covering treatment of >6000 patients with ulcerative colitis annually. **2** GlobalData. **3** Clinicaltrials.gov NCT07148414 4 Clinicaltrials.gov NCT07137598 **5** Clinicaltrials.gov NCT07176390 **6** Clinicaltrials.gov NCT06863961 **7** Clinicaltrials.gov NCT06956235 **8** Clinicaltrials.gov NCT06903065. **9** Clinicaltrials.gov NCT07133633 **10** Clinicaltrials.gov NCT07486960 **11** Active clinical trials sponsored by Merck (green), Roche (blue) and Spyre (magenta), as of 01-May-2026 **12** Global Data estimates.

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XmAb942 exhibits best-in-class clinical profile versus 1st gen TL1A mAbs

Class Leading Drug Exposure ❯ Higher consistency of adequate drug exposure driven by potency and low rates of immunogenicity

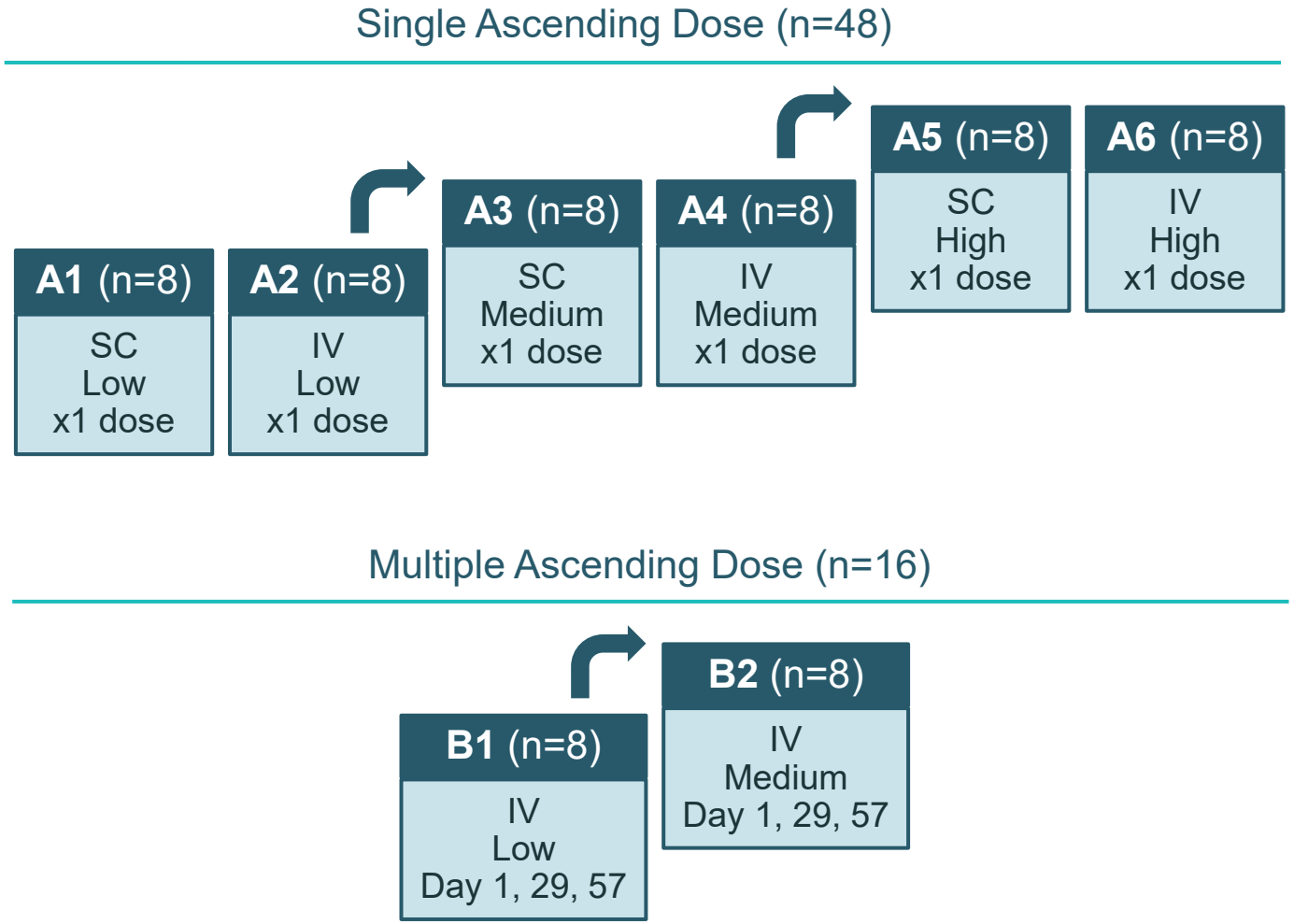
Class Leading Clinical Convenience ❯ Single subcutaneous injection maintenance dose every 12-weeks

Program[1]	Potent	TL1A Suppression[2]	Convenient SC Dosing	Q12W Dosing	Half-Life Extension	Low Immunogenicity
XmAb942[8]	✓	✓	✓	✓	✓	✓
Tulisokibart[3,4]	–	–	✓	✗	✗	✓
Afimkibart[5,6]	✓	✓	✓	✗	✗	✗
Duvakitug[7]	✓	✓	–	✗	✗	✓

1 No head-to-head trial has been conducted evaluating XmAb942 against other data included herein. Differences exist between clinical trial design, patient populations and the product candidates themselves, and caution should be exercised when comparing data across trials **2** As predicted by quantitative systems pharmacology (QSP) modeling based on human and non-human primate (NHP) pharmacokinetic (PK)/ pharmacodynamic (PD) data **3** PRA023 Progress Update (Prometheus presentation) **4** Feagan et al. Journal of Crohn's and Colitis, 2023;17:Supplement_1, i162-i164 **5** Banfield et al. Br J Clin Pharmacol. 2020;86:812–824 **6** Clarke et al. mAbs. 2018;10:4, 664-677 **7** Danese et al. Clin Gastroenterology and Hepatology. 2021;19:11, 2324-32.e6 **8** Osterman MT, et al. Abstract Mo1538. Presented at: Digestive Disease Week (DDW) 2026; May 2–5, 2026; Chicago, IL.

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The Phase 1 first-in-human study in healthy participants efficiently leveraged the known safety profile of the anti-TL1A class[1]



Study Design Elements

- Randomized, double-blind, placebo-controlled
- SAD (Part A) and MAD (Part B) cohorts
- SC and IV administration in SAD cohorts
- 6 active: 2 placebo per cohort

Population

- Healthy participants

Endpoints

- Primary: Safety
- Secondary: Pharmacokinetics
- Exploratory: Immunogenicity, PD profile

1. NCT06619990 **SC** subcutaneous administration **IV** intravenous administration

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XmAb942 is safe and well tolerated in healthy participants

All treatment emergent adverse events (TEAEs) were mild or moderate.

Rates of overall TEAEs were similar in XmAb942 and placebo: 75% (36/48 participants) vs. 69% (11/16 participants).

There were no serious or severe TEAEs, and no TEAEs led to drug or study discontinuation.

Headache was the most common TEAE and occurred in 33% of participants administered XmAb942 vs. 38% of participants administered placebo.

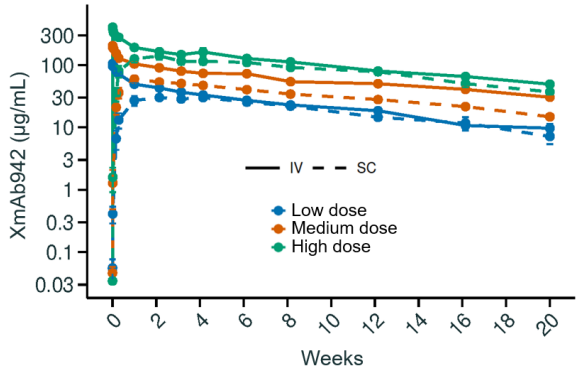
There were only 2 definite treatment-related AEs: both mild (1 injection site reaction, 1 administration site bruise) and occurred in the highest SC dose.

Osterman MT, et al. Abstract Mo1538. Presented at: Digestive Disease Week (DDW) 2026; May 2–5, 2026; Chicago, IL.

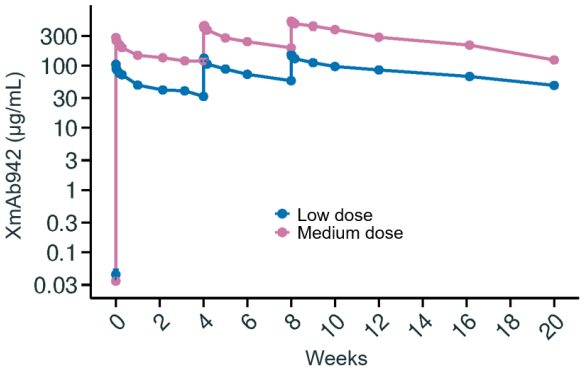
XmAb942 extended serum exposure and target engagement support the 12-week dosing interval during maintenance in XENITH-UC

Drug Concentration

Single Dose (SAD) IV and SC

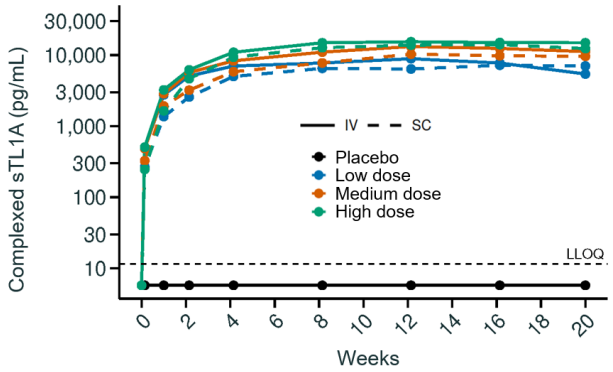


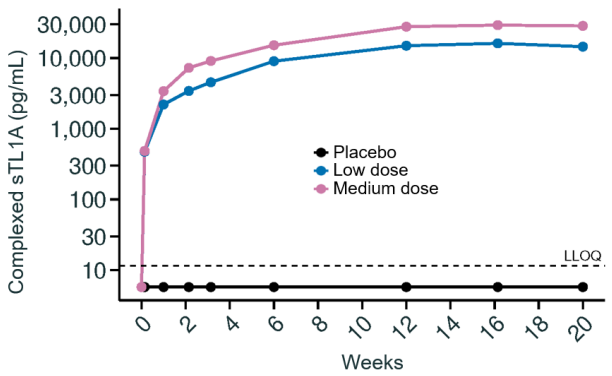
Multiple Dose (MAD) IV



Estimated terminal half-life is 74.1 days from pooled analysis of single-dose cohorts

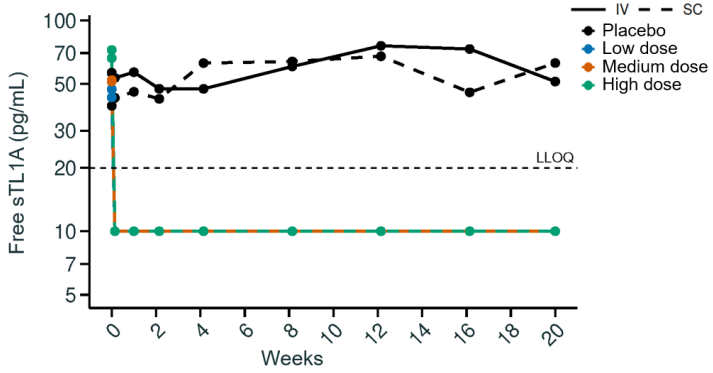
Complexed Soluble TL1A

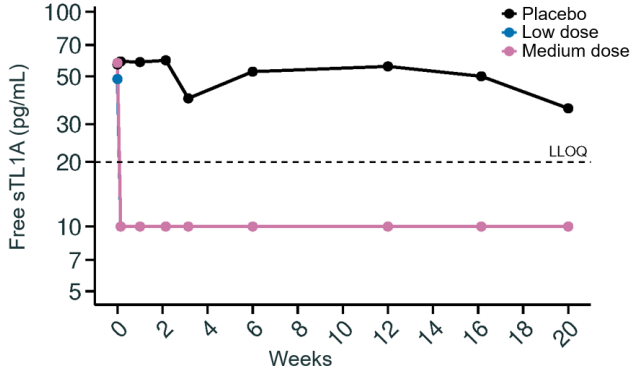




Dose-dependent and durable increases in target engagement for at least 20 weeks after a single dose of XmAb942

Free Soluble TL1A



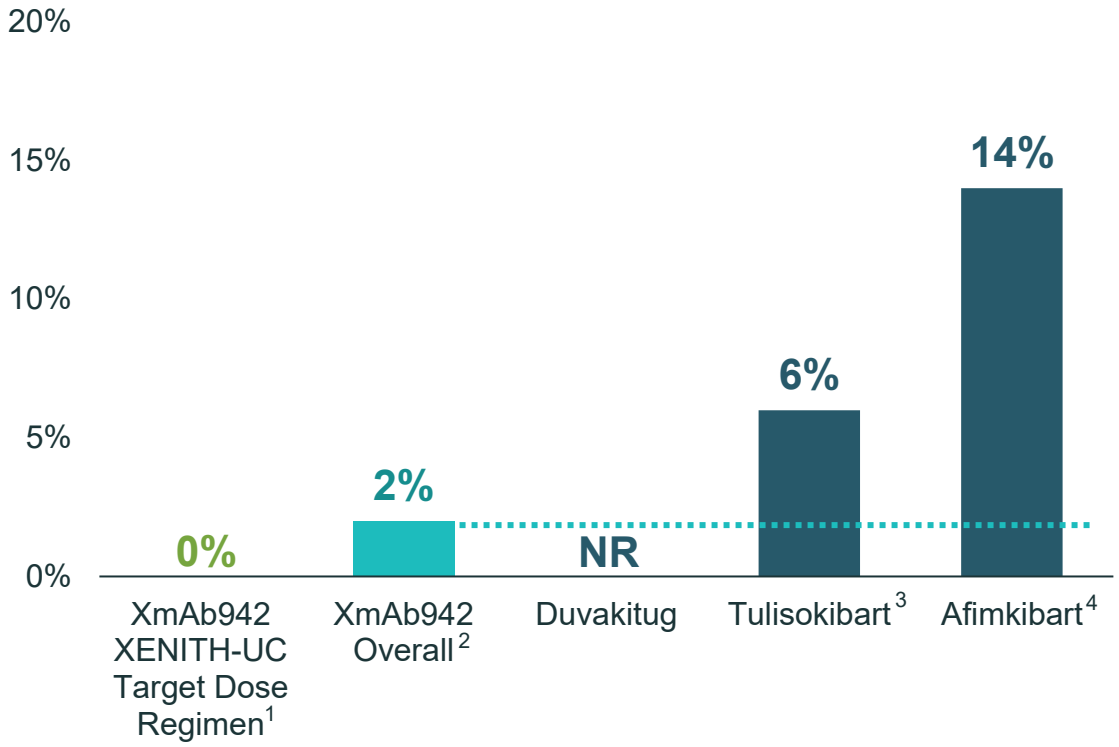


Rapid and sustained reduction of free soluble TL1A achieved below LLOQ for at least 20 weeks after a single dose of XmAb942

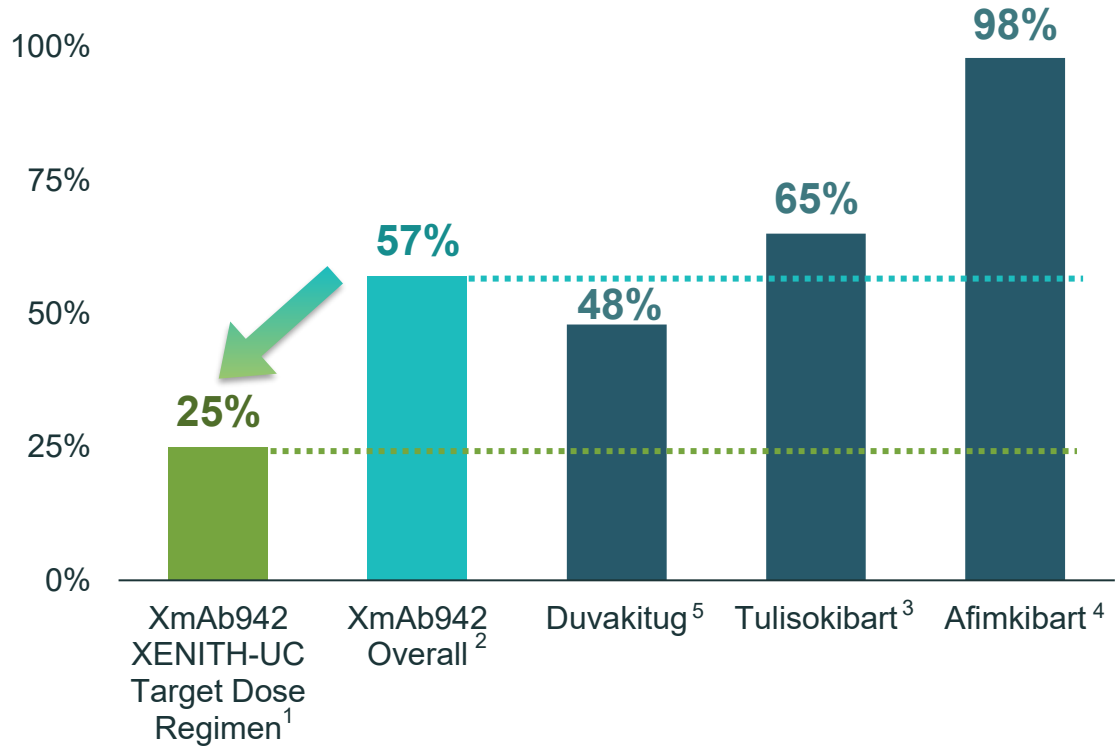
Osterman MT, et al. Abstract Mo1538. Presented at: Digestive Disease Week (DDW) 2026; May 2–5, 2026; Chicago, IL. Median value per cohort **LLOQ** lower limit of quantification (values plotted as LLOQ/2 per convention)

XmAb942 has a favorable immunogenicity profile compared to 1st generation anti-TL1A class

Reported rate of NAb+ in healthy participants*



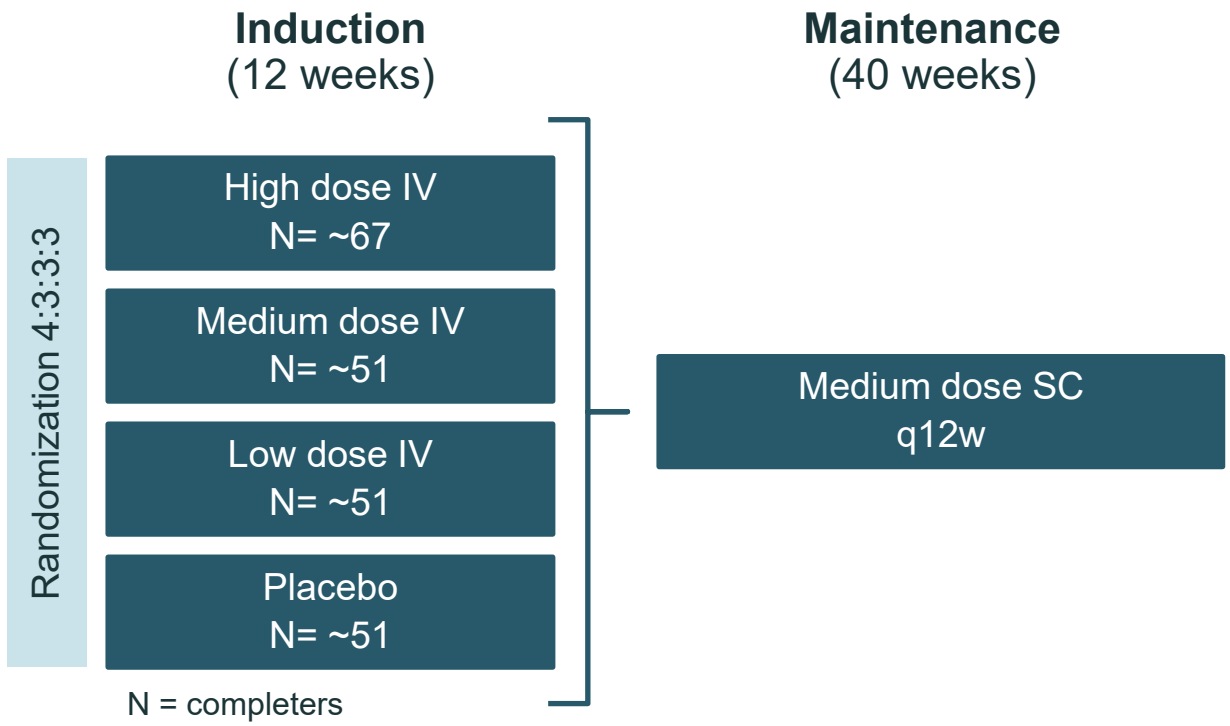
Reported rate of ADA+ in healthy participants**



* XmAb942 reported rate from all healthy participants dosed and evaluable from SAD/ MAD cohorts. Tulisokibart reported as NAb+ healthy participants dosed at 1000 mg SAD, 200mg and 500 mg MAD. Afimkibart calculated as NAb+ healthy participants dosed at 100mg or higher in SAD/MAD cohorts. ** Reported rate of ADA+ healthy participants from all SAD/MAD cohorts receiving study drug reported for XmAb942, duvakitug, tulisokibart, and afimkibart.

1 Xencor internal data. XENITH-UC Target Dose Regimen is the highest IV dose induction regimen arm of the Phase 2b study and the single SC dose used for maintenance in all cohorts **2** Osterman MT, et al. Abstract Mo1538. Presented at: Digestive Disease Week (DDW) 2026; May 2–5, 2026; Chicago, IL. **3** PRA023 Progress Update, Phase 1 Data & Announcement on New Indication, Prometheus Biosciences, 2021. **4** Banfield, et al. BJCP, April 2019. **5** Balyan et al., ECCO 2024.

XENITH-UC Study: Phase 2b design



Study Design Elements

- Double-blind, placebo-controlled
- IV administration in induction
- SC administration in maintenance

Population

- Moderate to severely active ulcerative colitis
  - Failed ≥1 conventional or advanced therapy
- N=~220, randomized 4:3:3:3 active:placebo

Primary Endpoint

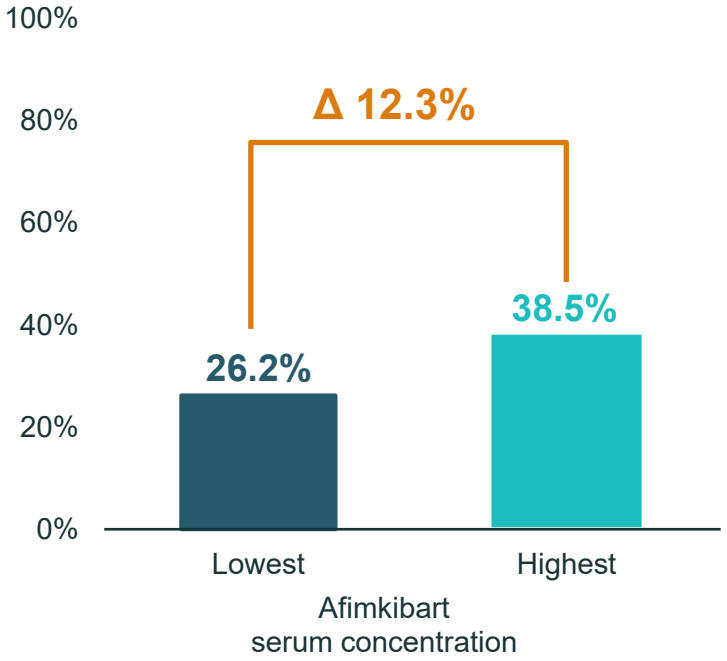
- Remission at Week 12 per modified Mayo score

- Drug exposure maximized to potentially achieve greater induction efficacy than observed in competitor trials
- Long half-life supports every 12-week dosing (q12w) during maintenance
- Asymmetric randomization ratio will minimize number of participants receiving induction placebo
- All induction placebo participants will receive active treatment during maintenance

Consistent relationship of exposure-response across biologic targets in ulcerative colitis studies is the pharmacological thesis for XENITH-UC

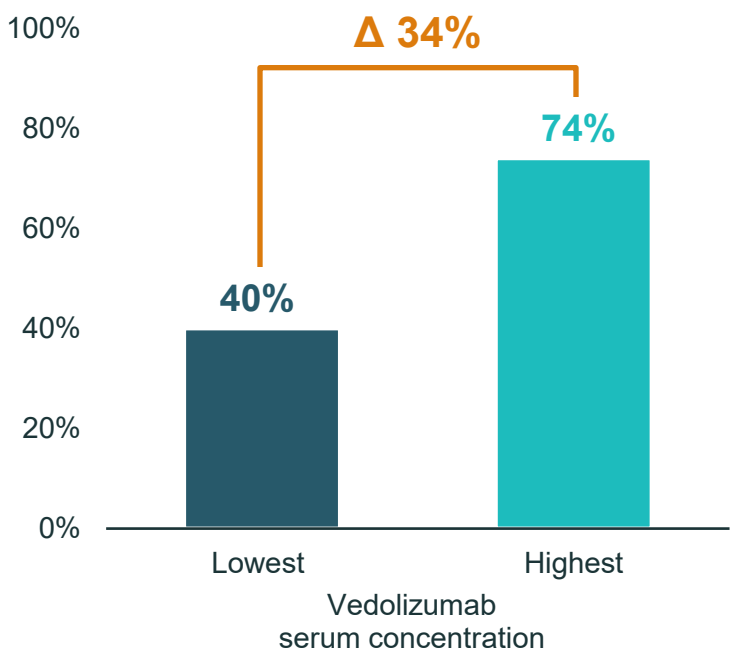
Anti-TL1A
Afimkibart

Clinical remission exposure-response at week 14 by 14-week C_{ave} tertiles[1]



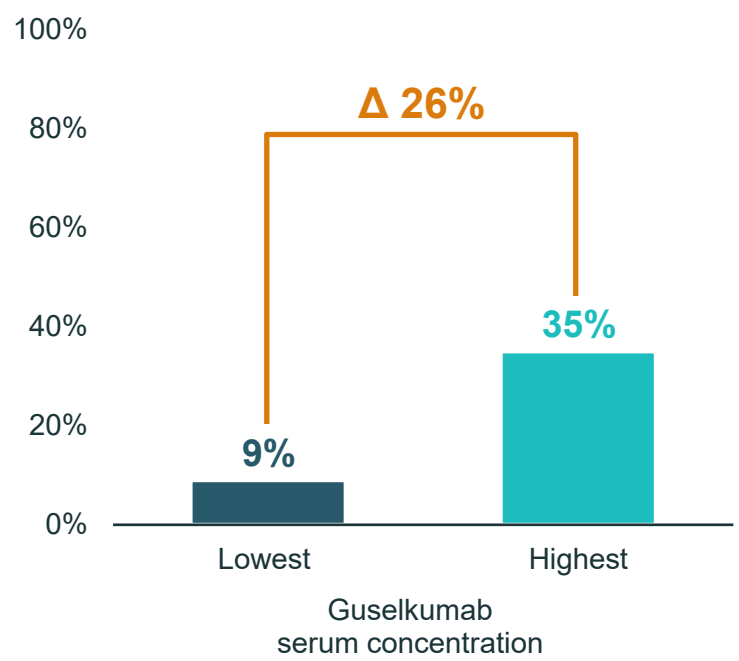
Anti-α4β7
Vedolizumab

Clinical remission exposure-response at week 14 by 6-week C_{ave} quartiles[2]



Anti-IL23
Guselkumab

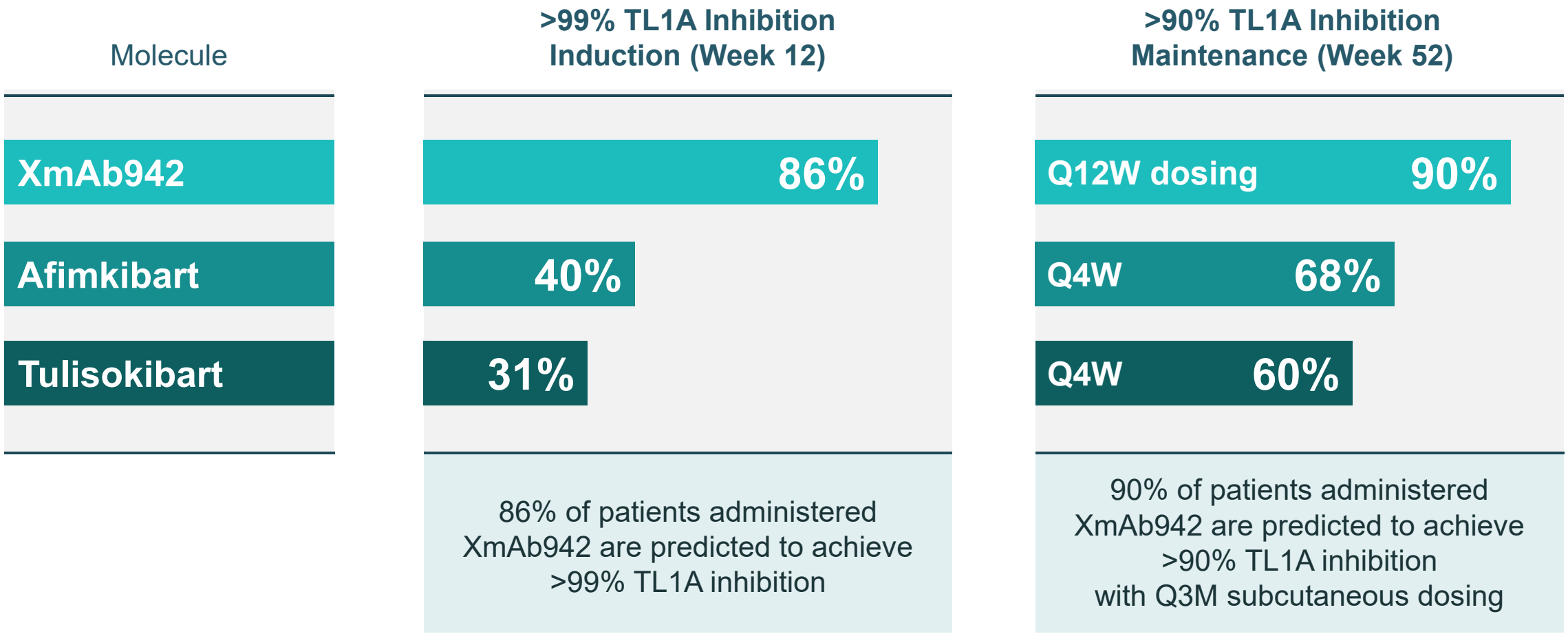
Clinical remission exposure-response at week 12 by 12-week C_{ave} quartiles[3]



1 Danese S, et al. *Lancet Gastroenterol Hepatol*. 2025;published online July 21. doi:10.1016/S2468-1253(25)00129-3. Supplementary appendix. **2** Osterman MT, et al. *Aliment Pharmacol Ther*. 2019;49:408–418. doi:10.1111/apt.15113. **3** Peyrin-Biroulet L, et al. Poster presented at: American College of Gastroenterology (ACG) Annual Meeting; October 27–29, 2025; Phoenix, AZ. Poster P5307.

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XmAb942 is predicted by quantitative pharmacology model to exceed 1st gen benchmarks of TL1A target inhibition in XENITH-UC

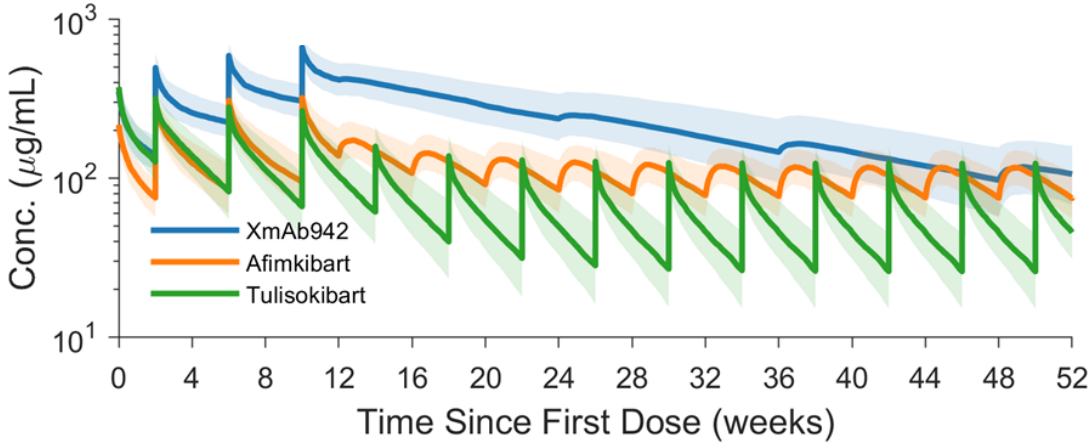


A unified quantitative systems pharmacology (QSP) model integrating clinical and published data for XmAb942, afimkibart, and tulisokibart was developed and extended to support virtual population simulations and comparative population-level PK/PD predictions across compounds.

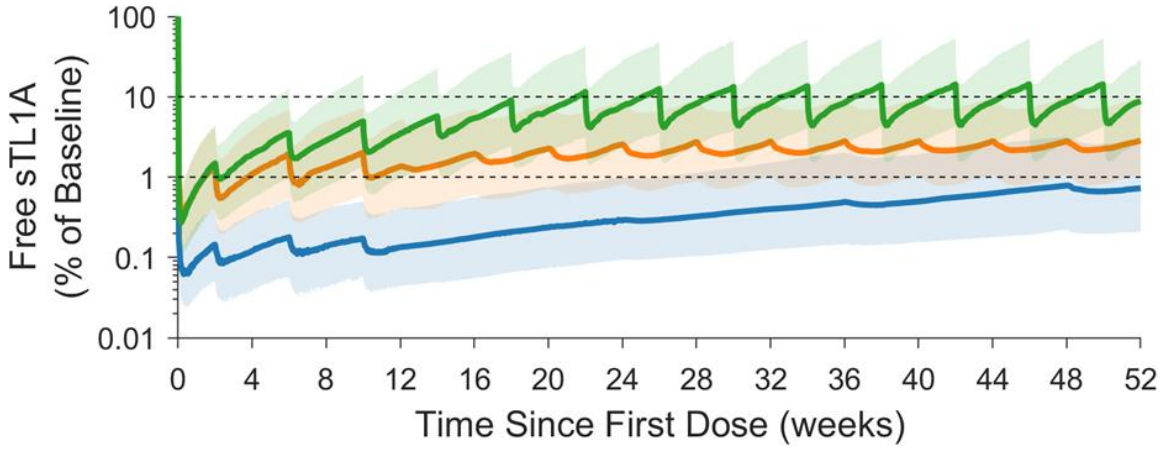
Source Osterman MT, et al. Abstract Mo1538. Presented at: Digestive Disease Week (DDW) 2026; May 2–5, 2026; Chicago, IL.

XmAb942 is predicted to maintain higher exposure compared to other TL1A antibodies during both induction and maintenance with fewer injections

Model-predicted pharmacokinetics (PK)



Model-predicted pharmacodynamics (PD) in tissue



A unified quantitative systems pharmacology (QSP) model integrating clinical and published data for XmAb942, afimkibart, and tulisokibart was developed and extended to support virtual population simulations and comparative population-level PK/PD predictions across compounds.

Source Osterman MT, et al. Abstract Mo1538. Presented at: Digestive Disease Week (DDW) 2026; May 2–5, 2026; Chicago, IL.

Favorable XmAb942 clinical convenience in XENITH-UC, with a significant reduction of injection burden in maintenance period



Source Based upon clinicaltrial.gov listings and publicly available presentations from Merck, Roche and Teva

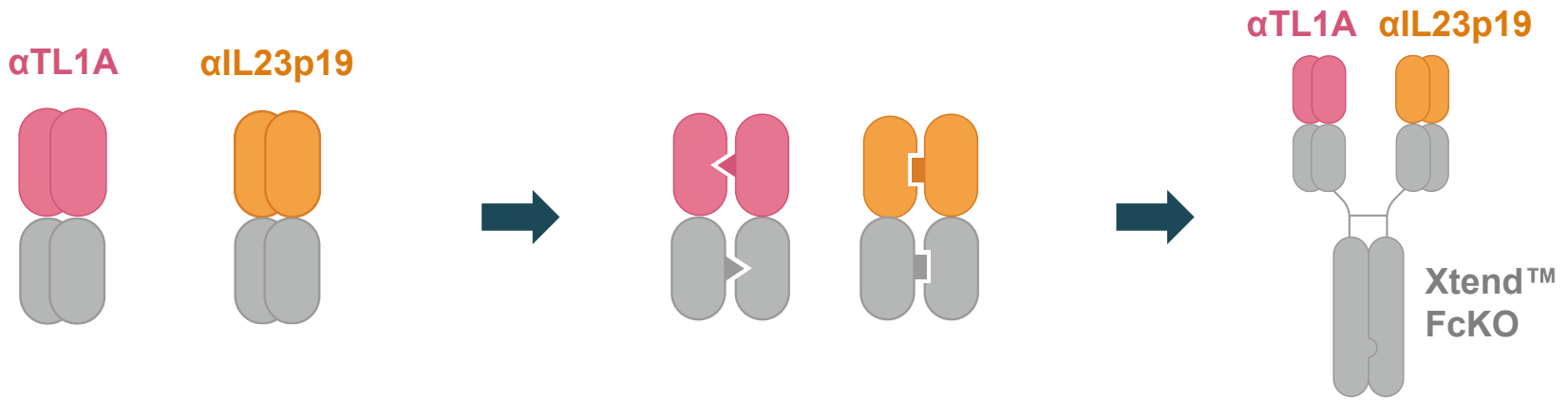
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New XenLock™ bispecifics are designed to meet the high bar for patient convenience, efficacy and immunogenicity in autoimmune disease

Fabs optimized separately
Ultra-high affinity
High stability, developability

With **XenLock™ Fab domains**, each pair of light and heavy chains **pair distinctly**

XmAb412 (TL1A x IL23p19) is the first XmAb® bispecific engineered with XenLock™ Fab domains



Modular building blocks
- 2 XenLock™ Fab domains
- 1 heterodimer Fc domain
- Xtend™ and FcKO mutations

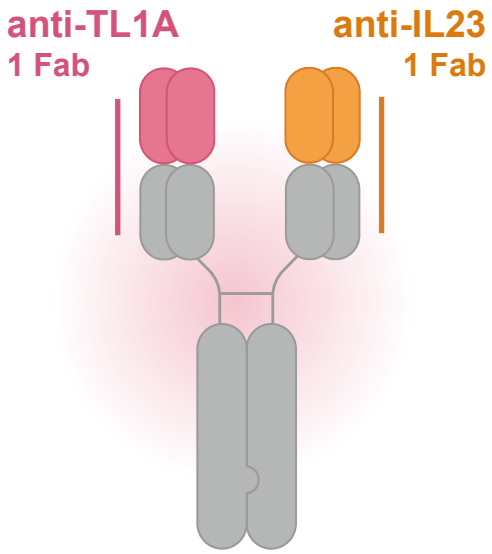
Efficient assembly

Manufactured at scale

- Bispecifics with an IgG format minimize molecular size, allowing for easier **high concentration formulation** and 1+1 valency, which **reduces immunogenicity potential**
- **XenLock enables sub-picomolar affinities** needed to reach sufficient potency (vs. CLC or Vhh solutions)
- **XenLock opens access to the full suite of antibody optimization tools** and **full variable domain diversity**

XmAb412 poised to be best-in-class biologic for IBD and deliver PoC for new XenLock™ bispecific platform for autoimmune and inflammatory disease

XenLock™ Fab Domains

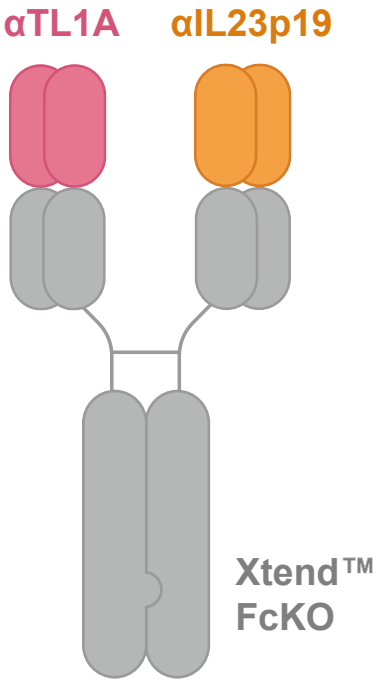


XmAb® Bispecific Fc Domain
Xtend™ + FcKO

- **XmAb412 in healthy participants initiated enrollment in third quarter of 2026.**
- XmAb412 demonstrated IC_{50} values comparable or superior to clinical-stage TL1A antagonists and approved IL23 antagonists.
- XmAb412 is predicted to have a half-life of 60 to 70 days in humans. In NHPs, XmAb412 achieved a half-life exceeding 20 days, with similar target engagement to monospecific antibodies.
- XmAb412 supports high-concentration, low viscosity, citrate-free formulation suitable for subcutaneous dosing.
- Next XenLock autoimmune program is in preclinical development

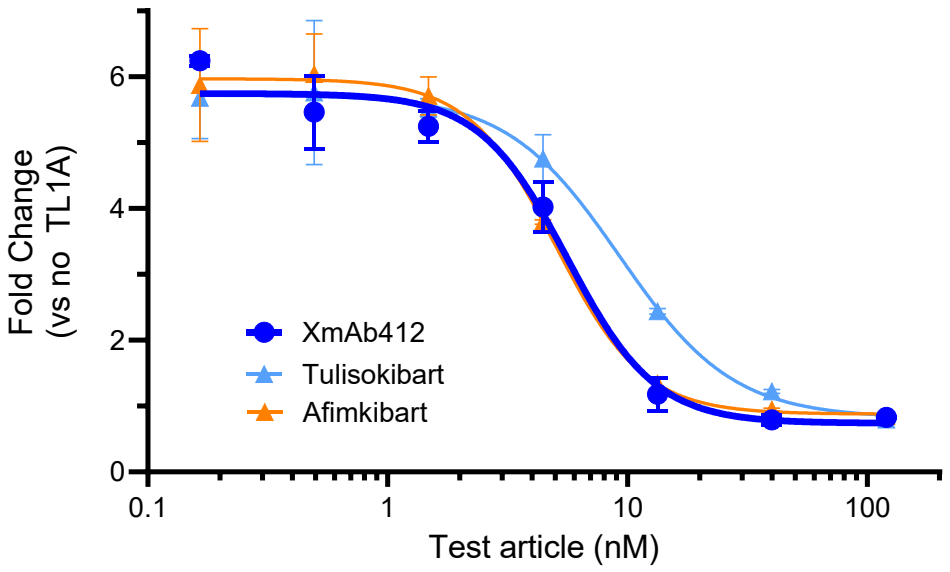
PoC proof of concept

XmAb412 suppresses two important inflammatory axes – TL1A and IL23 with equal potency to best-in-class monospecific mAbs



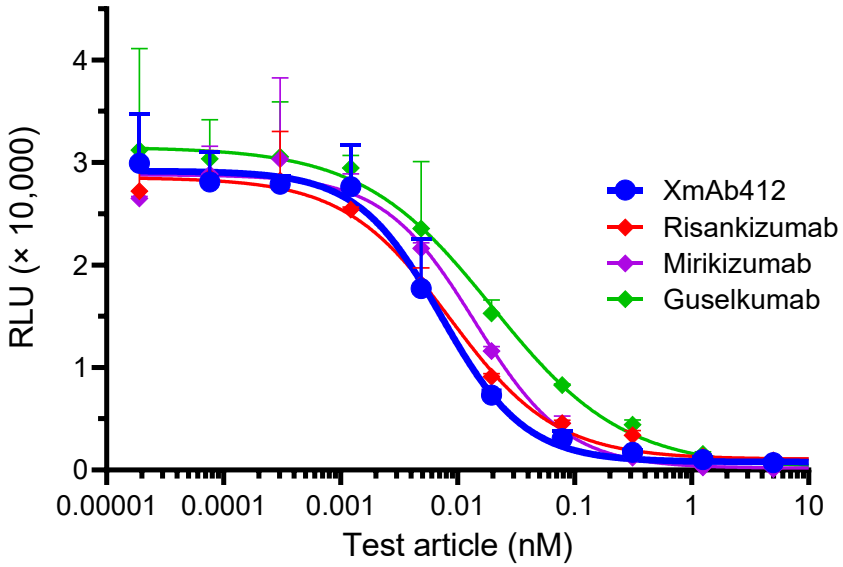
Requires half the antibody concentration as equivalent co-formulation doses

XmAb412 inhibits TL1A-induced apoptosis similar to a first-generation monospecific anti-TL1A antibody



TF1 cycloheximide assay

XmAb412 inhibits IL23 activity more potently than marketed monospecific IL23 antibodies



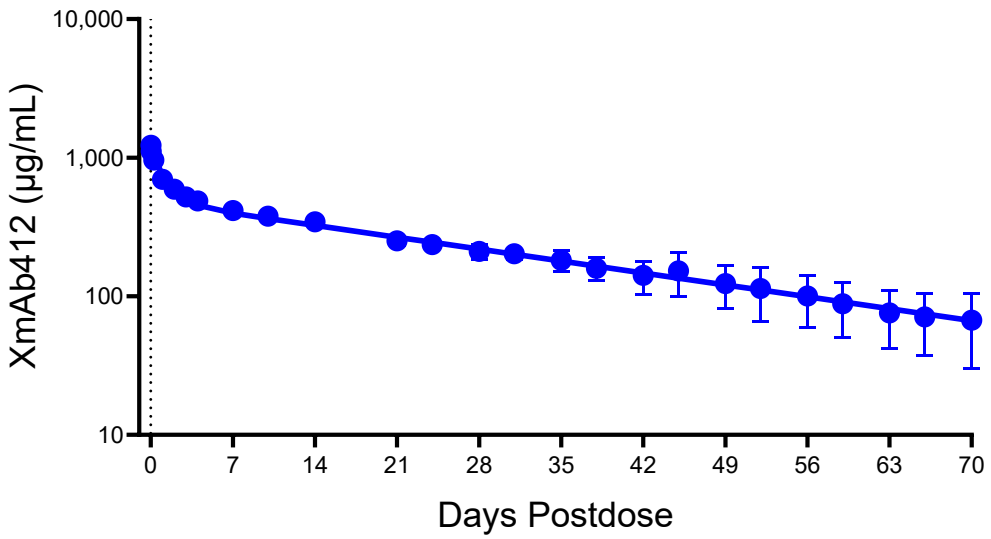
Reporter assay

RLU relative light unit

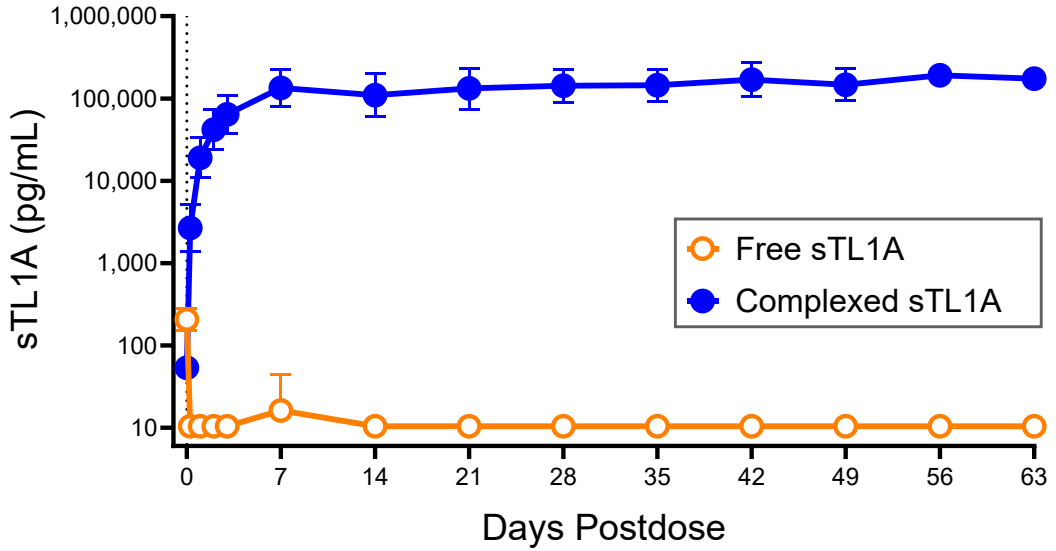
Faber MS, Avery KN, et al. Abstract Tu1468. Presented at: Digestive Disease Week (DDW) 2026; May 2–5, 2026; Chicago, IL.

Data support a 60- to 70-day half-life in humans, enabling a potential dosing interval of every 8 to 12 weeks or longer[1]

NHP half-life > 20 days



Durable engagement in NHPs



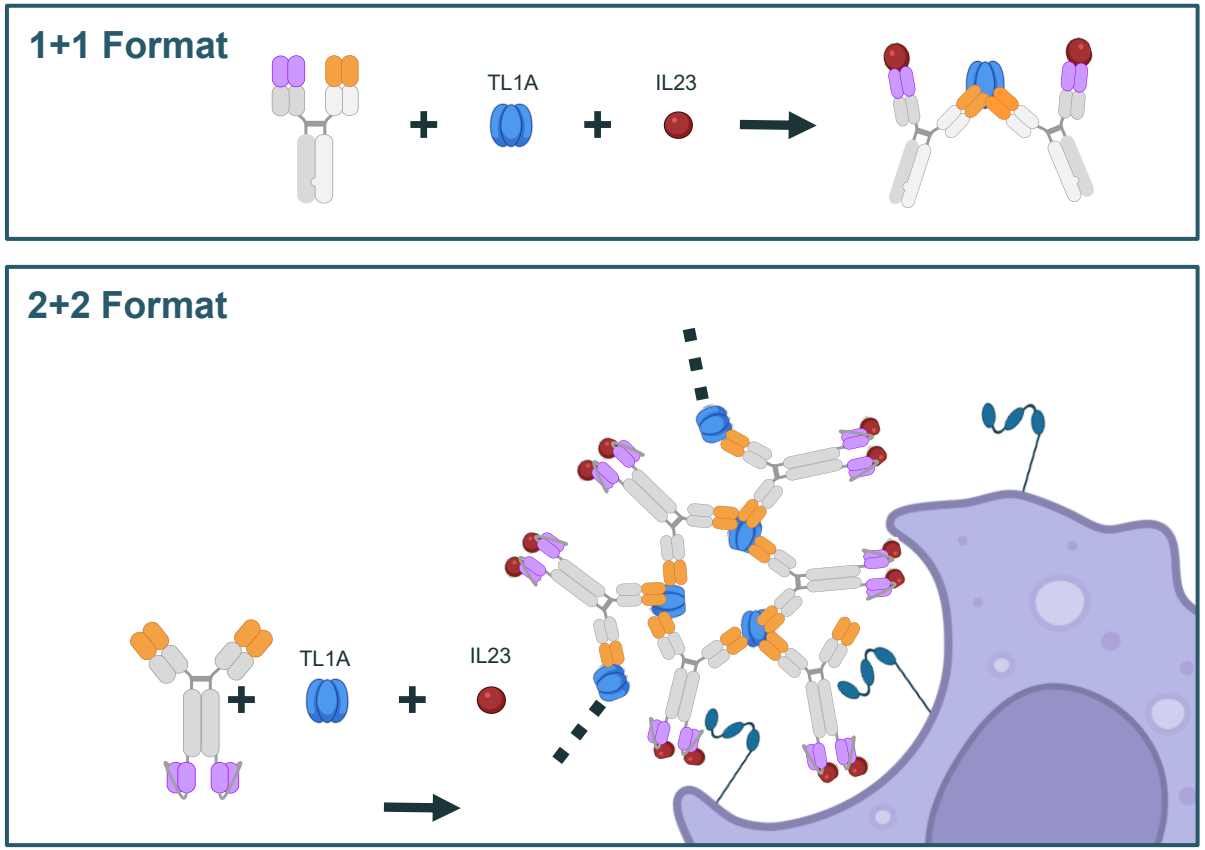
Cynomolgus monkeys received a single intravenous injection of XmAb412. Concentration vs time data (left plot) were analyzed by 2-compartment analysis. Data are means ± SEM. Free or XmAb412-complexed soluble (s) TL1A was measured (right plot). Data are geometric means ± geometric SD. The vertical lines indicate the day of dosing.

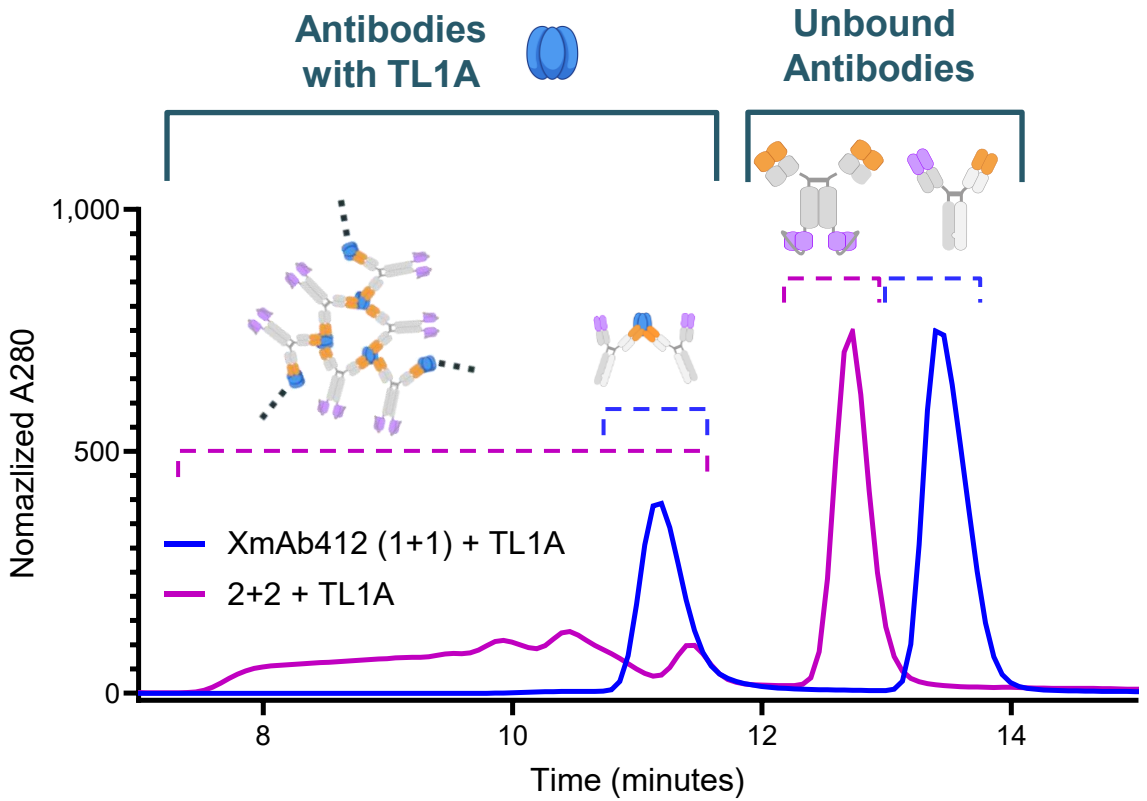
NHP non-human primate **1** Allometric scaling adjusted for half-life engineered antibodies. Haraya K, Tachibana T. *BioDrugs.* 2023;37(1):99-108.
Faber MS, Avery KN, et al. Abstract Tu1468. Presented at: Digestive Disease Week (DDW) 2026; May 2–5, 2026; Chicago, IL.

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XmAb412 avoids large immune complex formation seen in 2+2 formats, which reduces potential immunogenicity impact to clinical outcomes

Possible Immune Complexes



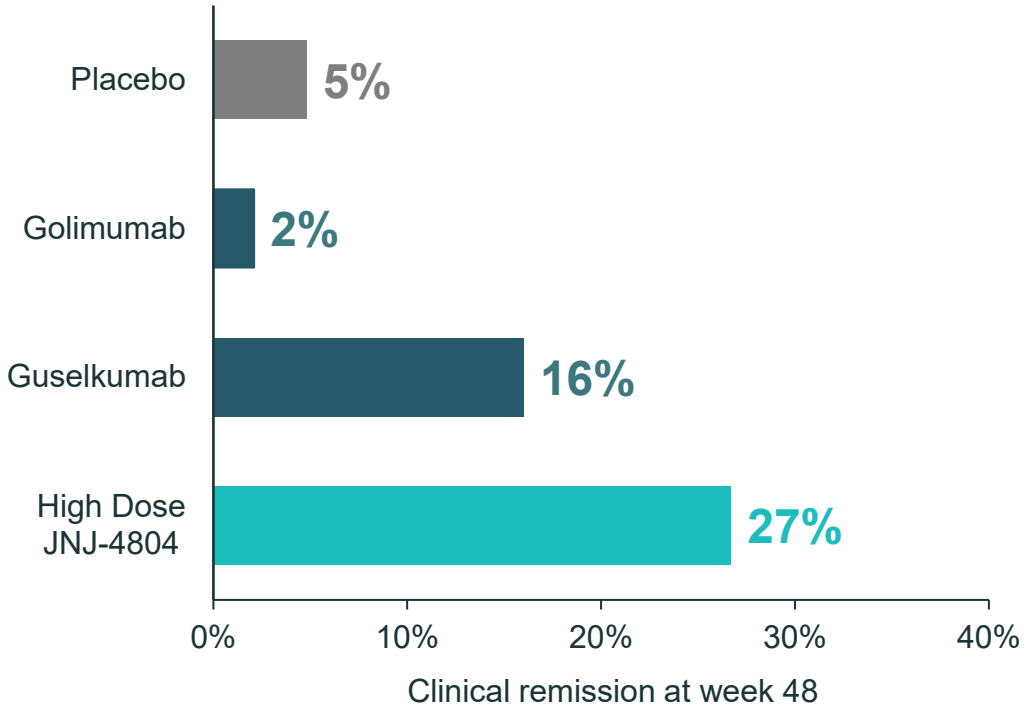


Antibody immune complex size as determined by size exclusion chromatography.

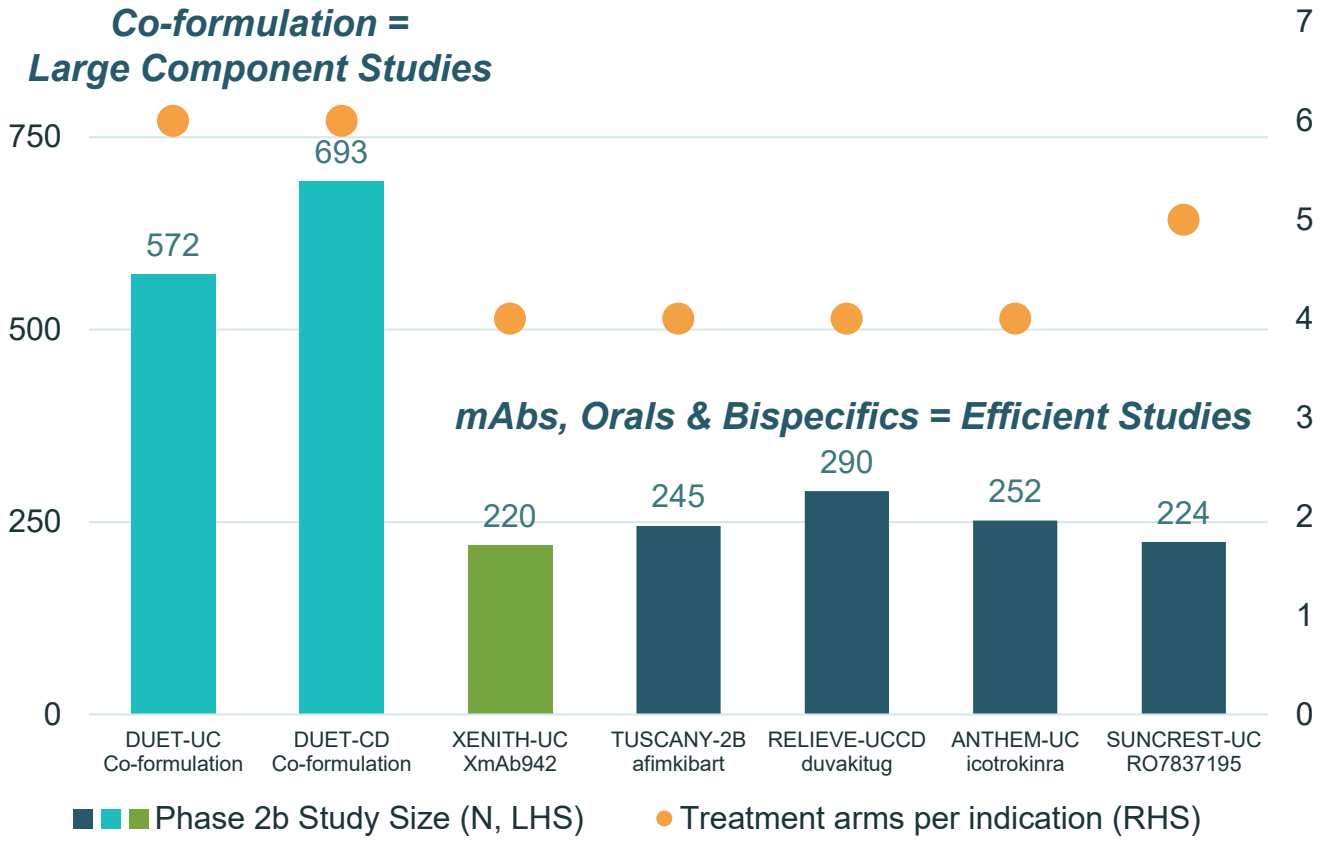
Faber MS, Avery KN, et al. Abstract Tu1468. Presented at: Digestive Disease Week (DDW) 2026; May 2–5, 2026; Chicago, IL.

XmAb412 is designed to deliver the promise of dual targeted therapy for IBD but avoid complex development of co-formulation approaches

DUET-UC Study[1]
(Inadequate Response to ≥2 ST Classes)



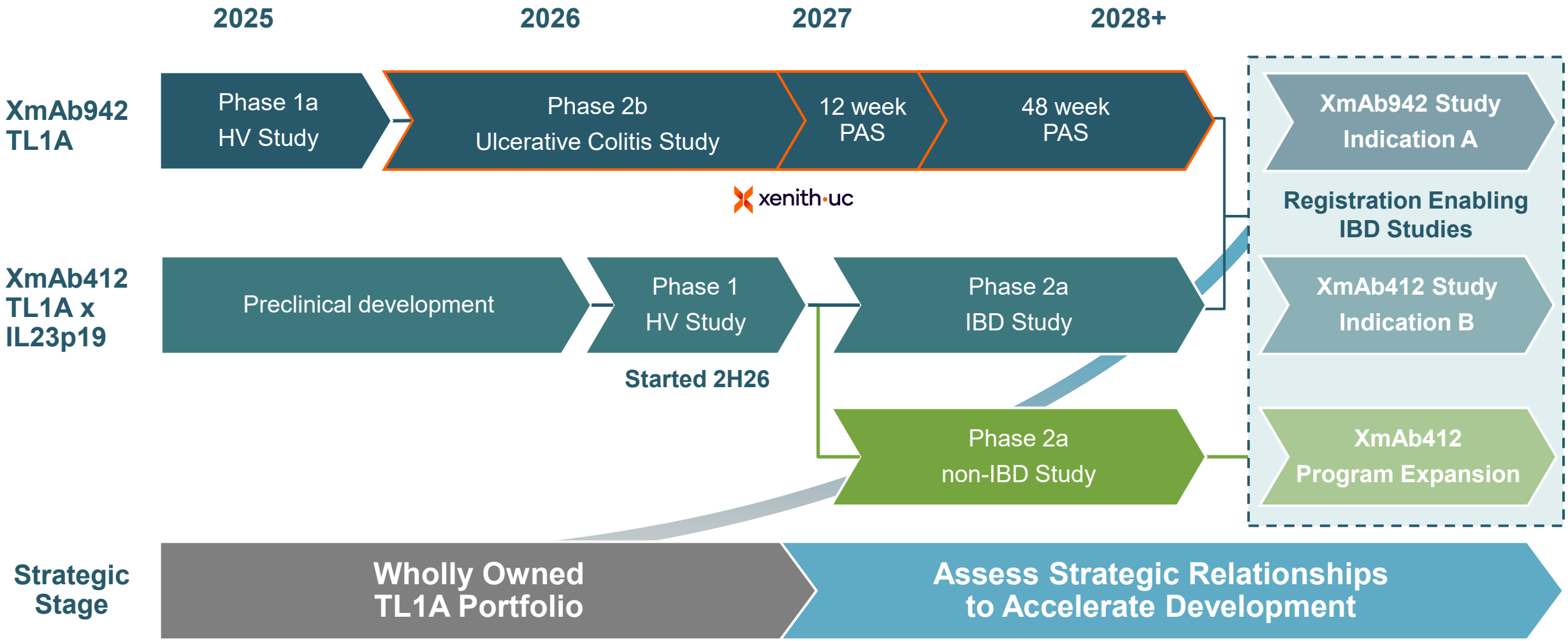
Comparison of IBD study sizes[2]



1 Abreu MT, et al. Abstract 1058d. Presented at: Digestive Disease Week (DDW) 2026; May 2–5, 2026; Chicago, IL. **2** Clinical trial information on size and number of treatment arms is based on trial listings on Clinicaltrials.gov.

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Xencor's TL1A development roadmap integrates XmAb942 and XmAb412 decision points to define registration enabling studies in IBD



PAS primary analysis set

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Next clinical development milestones for Xencor's TL1A pipeline

✓	Start of Phase 1 first-in-human study of XmAb412
YE26	Update on enrollment and blinded interim analysis of XENITH-UC
1H27	Interim results of the Phase 1 first-in-human study of XmAb412
2H27	12-week induction primary endpoint of XENITH-UC

XmAb® Bispecific T-Cell Engagers

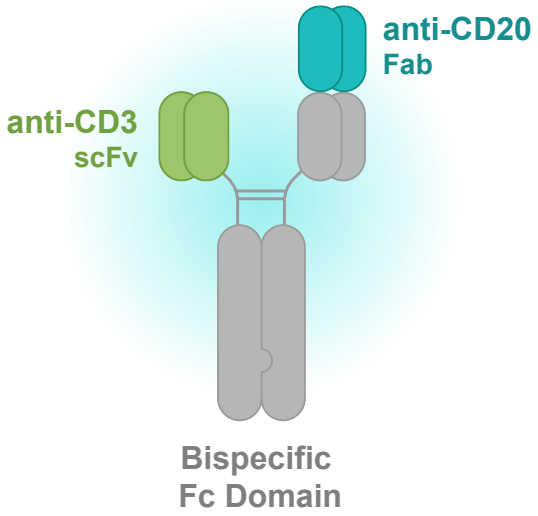
B-cell depleting bispecific antibodies in autoimmune disease



Plamotamab is a subcutaneous CD20 x CD3 T-cell engager in Phase 1b study

Proof-of-concept for the T-cell engager class in autoimmune and inflammatory disease

XmAb® CD20 x CD3 Bispecific Design



- Plamotamab designed in a 1+1 format and selected for extended activity and favorable tolerability observed in NHPs
- Human half-life ~18 days; estimated 80% SC bioavailability
- Robust manufacturing process with high yield and excellent formulation stability data

Positioned for Success

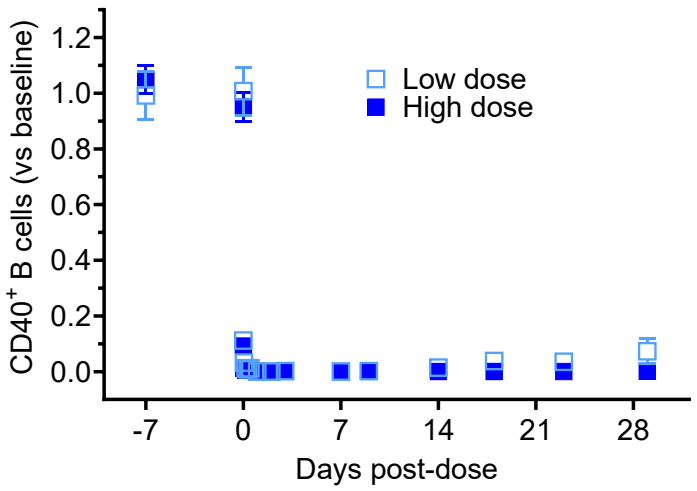
- N=154 from dose escalation and expansion cohorts with both IV and SC formulations in B-cell cancers
- Comparable preliminary efficacy data to leading commercial CD20 x CD3 in patients with prior CAR-T
- SC dosing regimen with improved CRS data vs. leading commercial CD20 x CD3[1]
- Existing inventories of drug product and drug substance for seamless integration into the next phase of clinical development

BsAb bispecific antibody **IV** intravenous **SC** subcutaneous **NHP** non-human primate **CRS** cytokine release syndrome **1** No head-to-head trial has been conducted evaluating plamotamab against other data included herein. Differences exist between clinical trial design, patient populations and the product candidates themselves, and caution should be exercised when comparing data across trials.

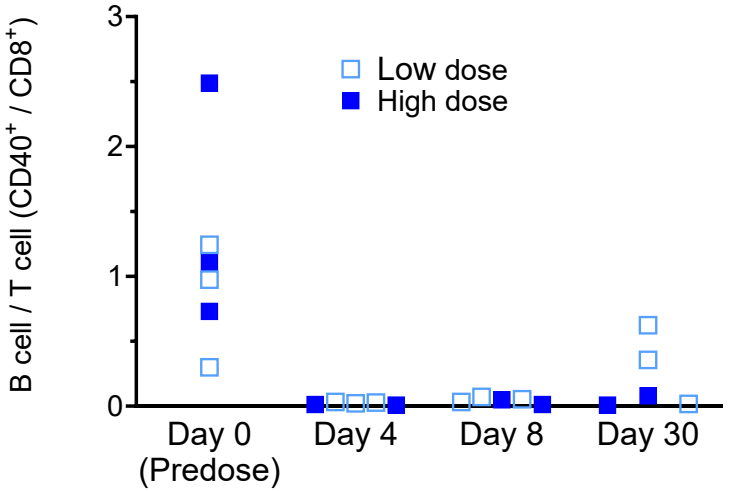
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Durable B-cell depletion was observed in blood and lymphoid organs with a single dose of plamotamab in NHPs

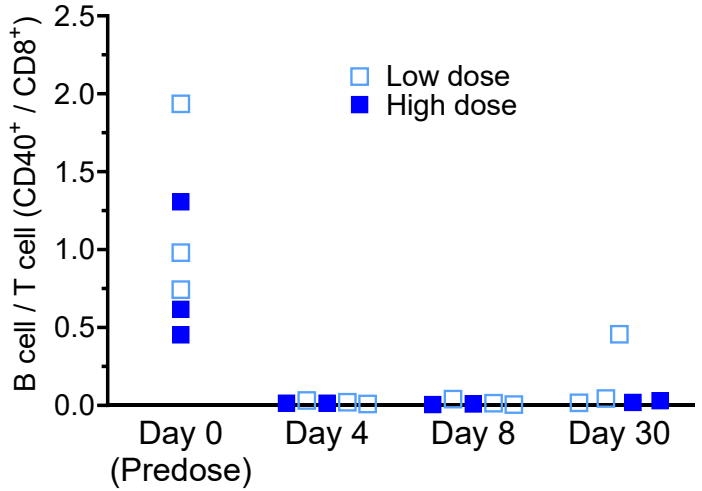
Blood B cells



Bone marrow B cells



Lymph node B cells

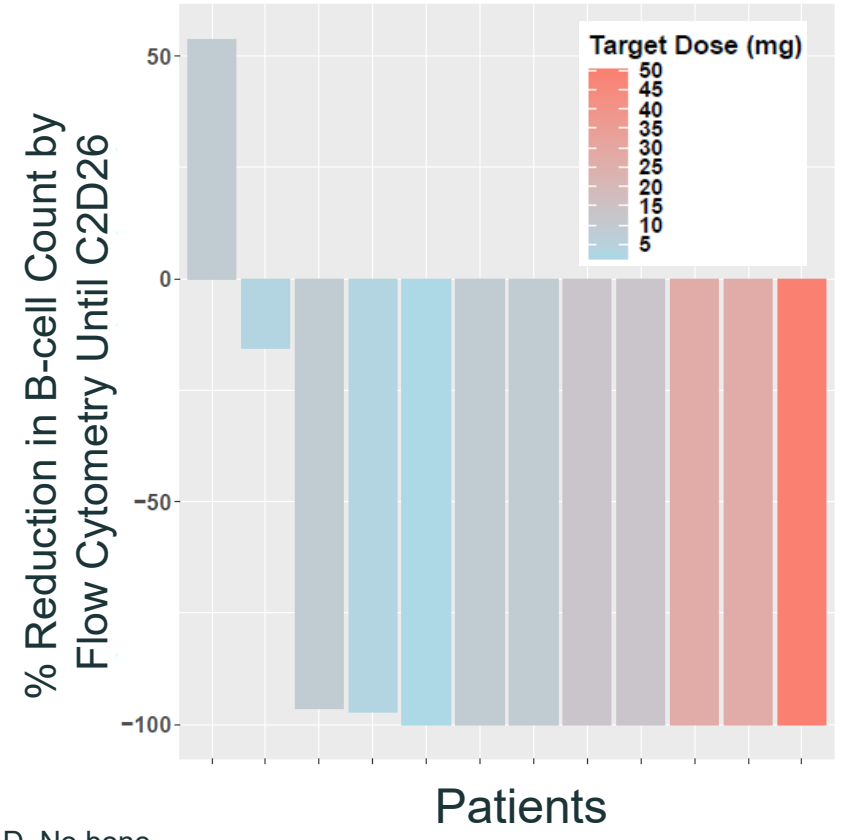


>95-99% peripheral B-cell depletion was observed in lymphoma patients with IV & SC dosing in the Phase 1 monotherapy study

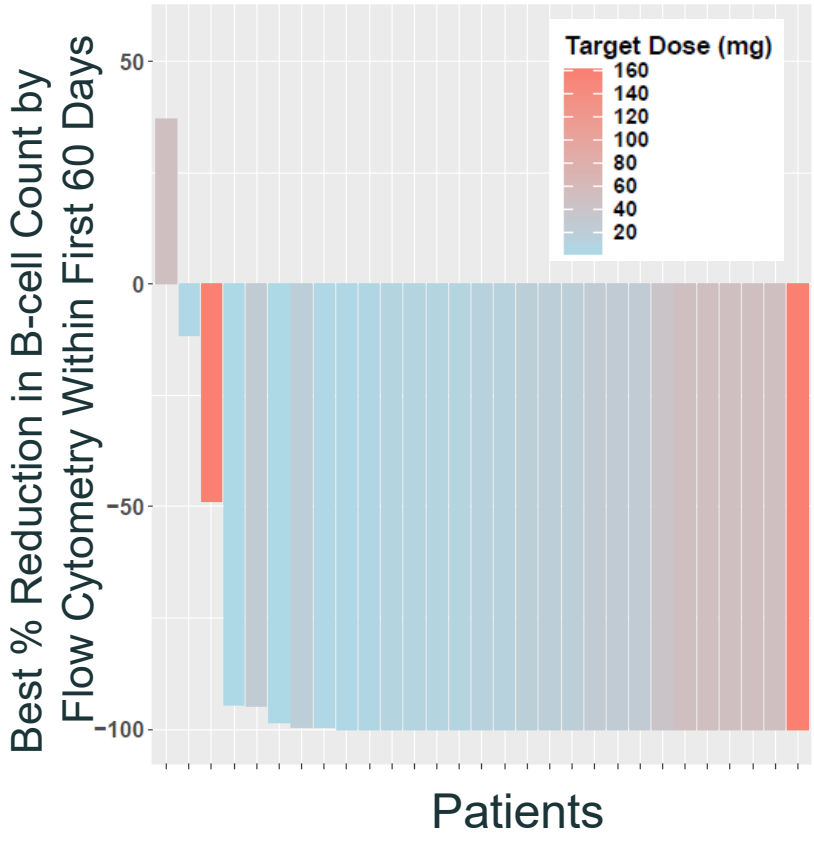
- Absolute CD19+ B-cell count in bone marrow (on C1D1 and C1D26) and whole blood (on C1D1 and timepoints up to C5D1) measured by flow cytometry
- >90% decrease in B cells in both bone marrow (baseline vs post-dose) and whole blood across the dose cohorts

Significant Reduction in B-cell Count

Percent Reduction of B Cells in Bone Marrow



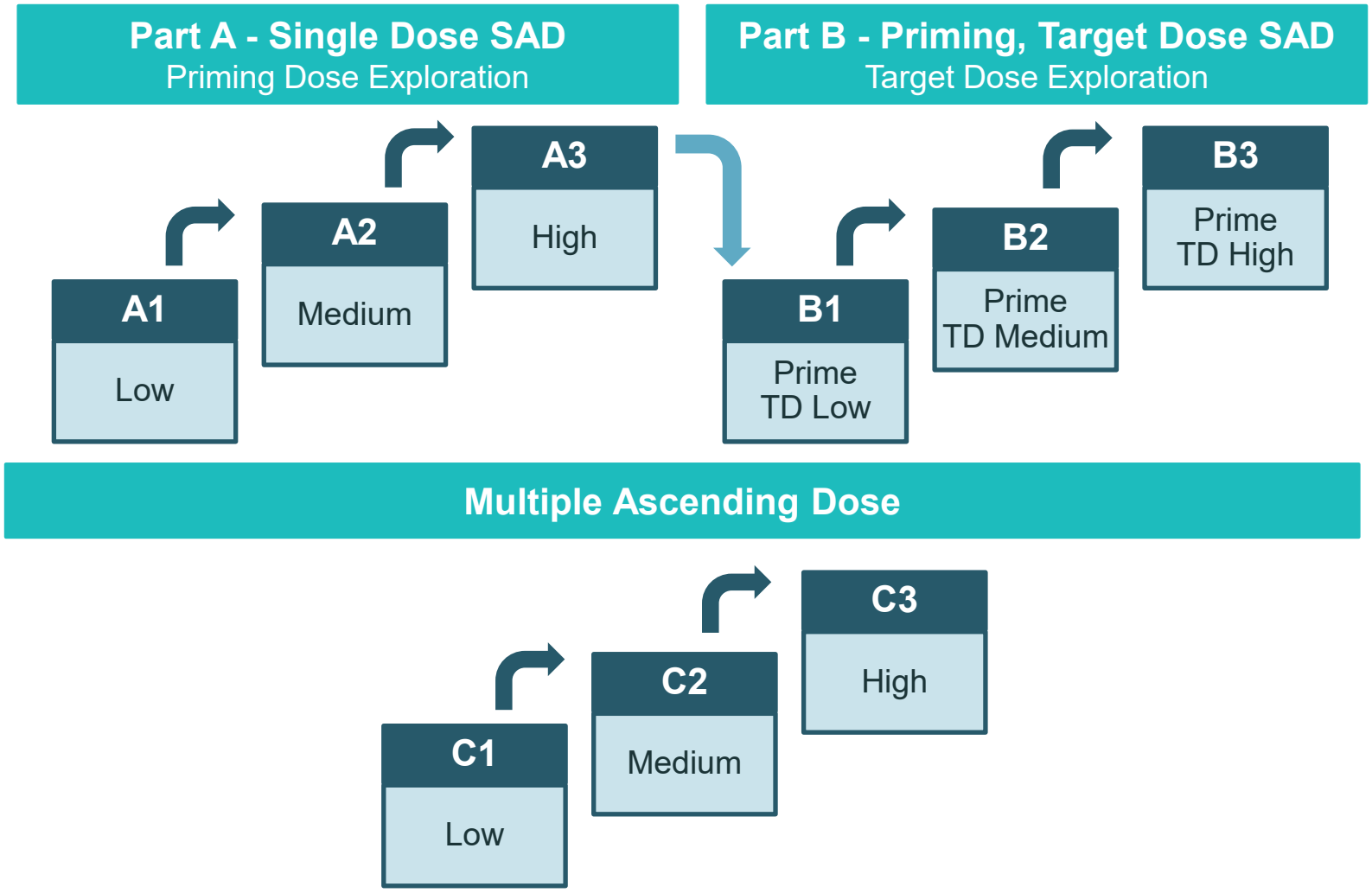
Percent Reduction of B Cells in Whole Blood



C = cycle; D = day. Data includes patients from Parts A-D. No bone marrow samples were collected in Part D. Patients with LBCL were included if their baseline B-cell count was >10 cell/uL.

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Ongoing Phase 1b open-label study design in rheumatoid arthritis (RA)



Highly efficient study design leverages known oncology exposure profile

Population

- Rheumatoid Arthritis (RA)

Study Design Elements

- Evaluate safety, tolerability, pharmacokinetic (PK), and pharmacodynamics (PD), and immunogenicity
- SAD and MAD cohorts
- SC administration

Endpoints

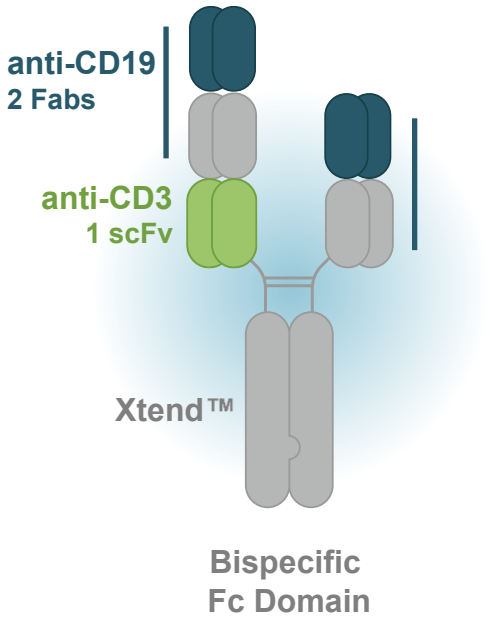
- Primary: Safety
- Secondary: Pharmacokinetics
- Exploratory: Immunogenicity, PD profile

NCT07230353

SC subcutaneous administration **TD** target dose

XmAb657 is a CD19 x CD3 T-cell engager optimized for autoimmune disease

Rational XmAb® Design



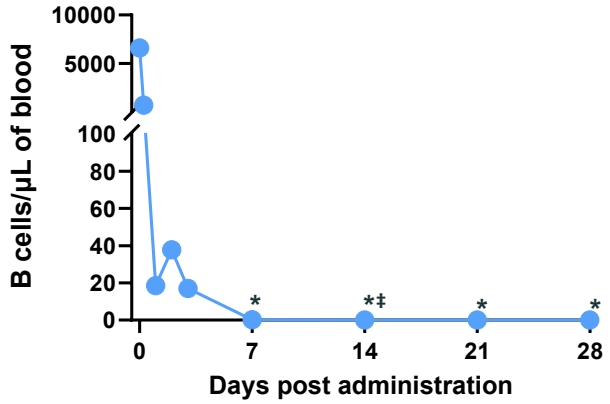
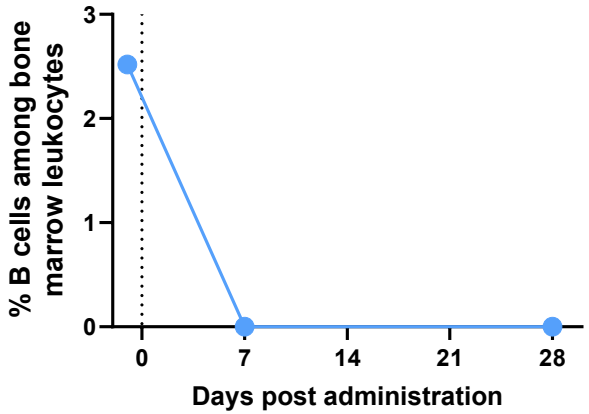
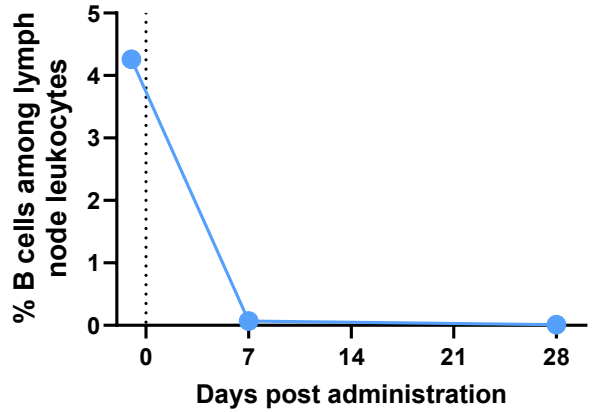
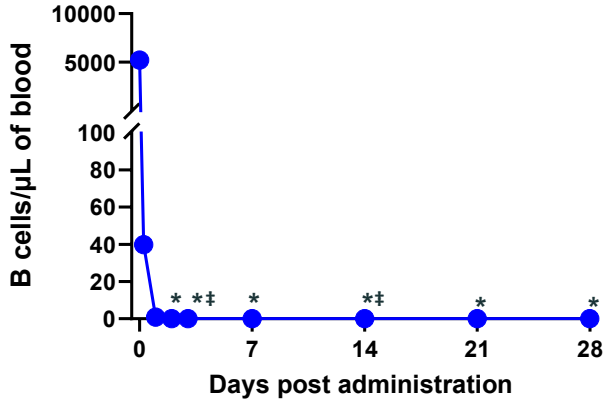
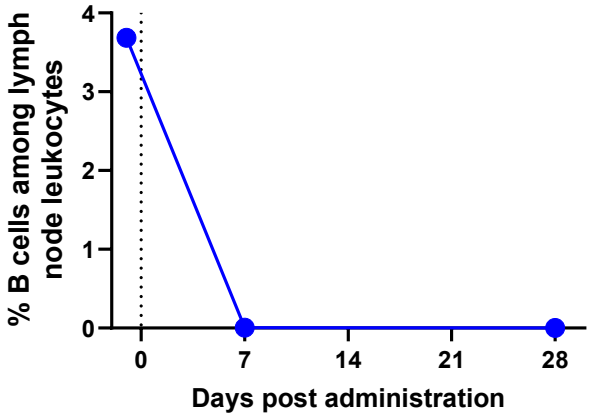
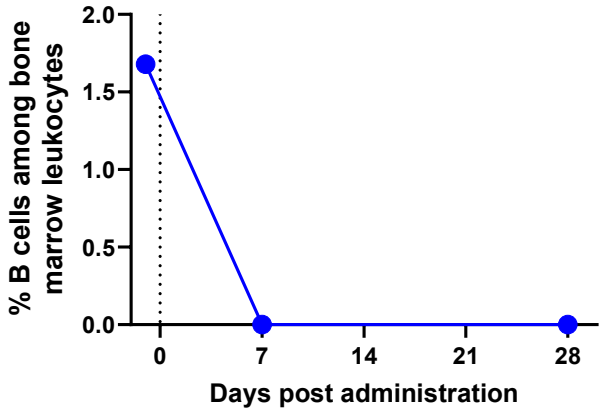
- High affinity and stability anti-CD19 binder
  - Bivalent to efficiently target B cells expressing very low levels of CD19 (e.g., plasma cells and plasmablasts)
- Affinity-tuned and highly stable anti-CD3 binder
- Uses Xencor's clinically validated 2+1 format
- Heterodimeric Fc domain engineered to abrogate effector function and improve half-life
- Xtend™ Fc for long half life

Positioned for Success

NHP studies have shown effective B-cell depletion with single dose

- Broad opportunity set of disease indications supports multiple development pathways for success
- CD19 CAR-T clinical data highlights potential issues with CAR-T approach on efficacy and safety for patients
- Rational design of XmAb657 supports best-in-class potential for clinical outcomes
- **FIH study initiated in Q4'25 puts Xencor on-track to be a leading CD19 x CD3 program within autoimmune disease**

Deep B-cell depletion was sustained for at least 28 days with a single dose in NHPs

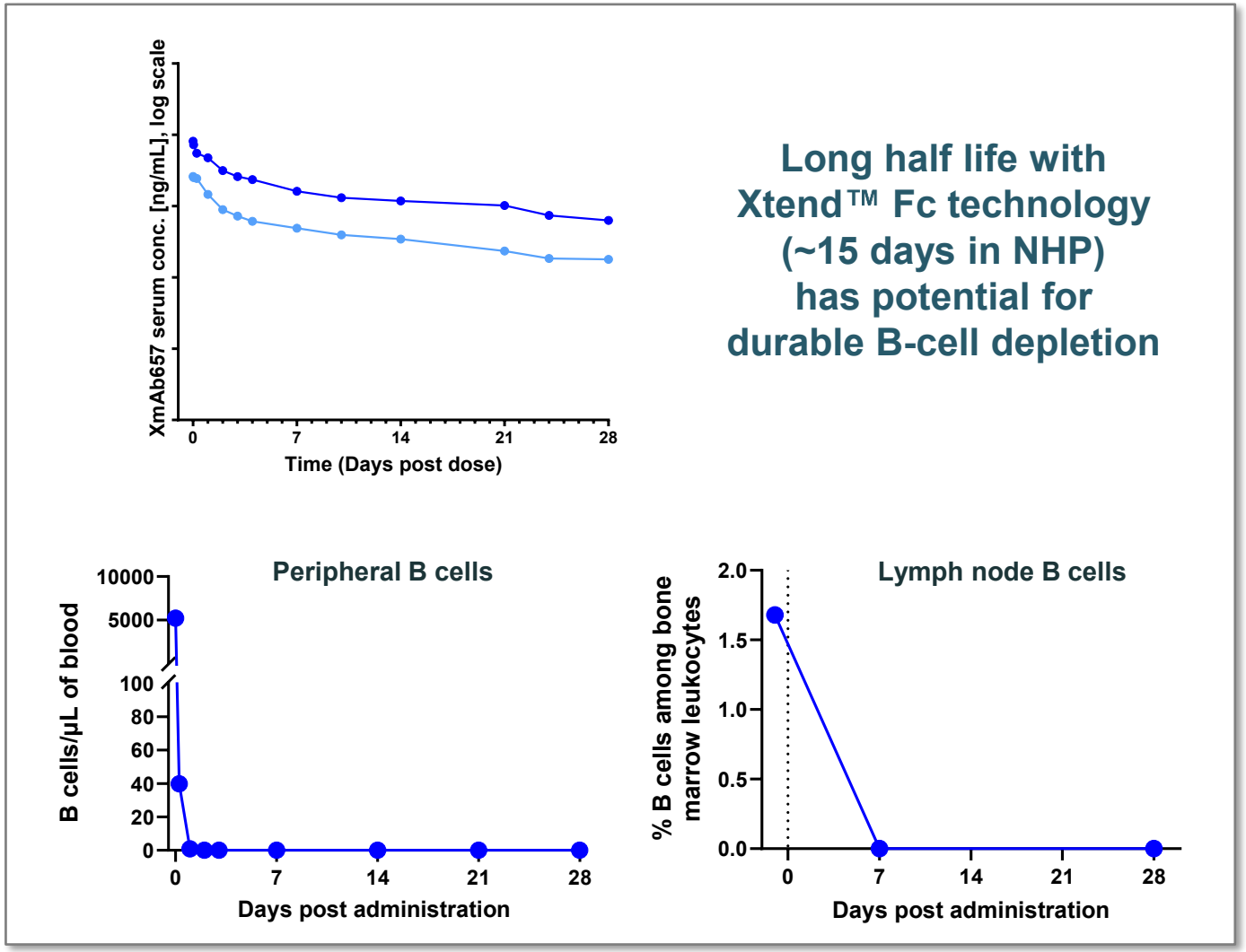
	Peripheral blood	Bone marrow	Lymph nodes
XmAb 657 Single IV Dose (low)			
XmAb657 Single IV Dose (high)			

*peripheral B cells <1 B cell per µL;
‡this data point is zero B cells per µL

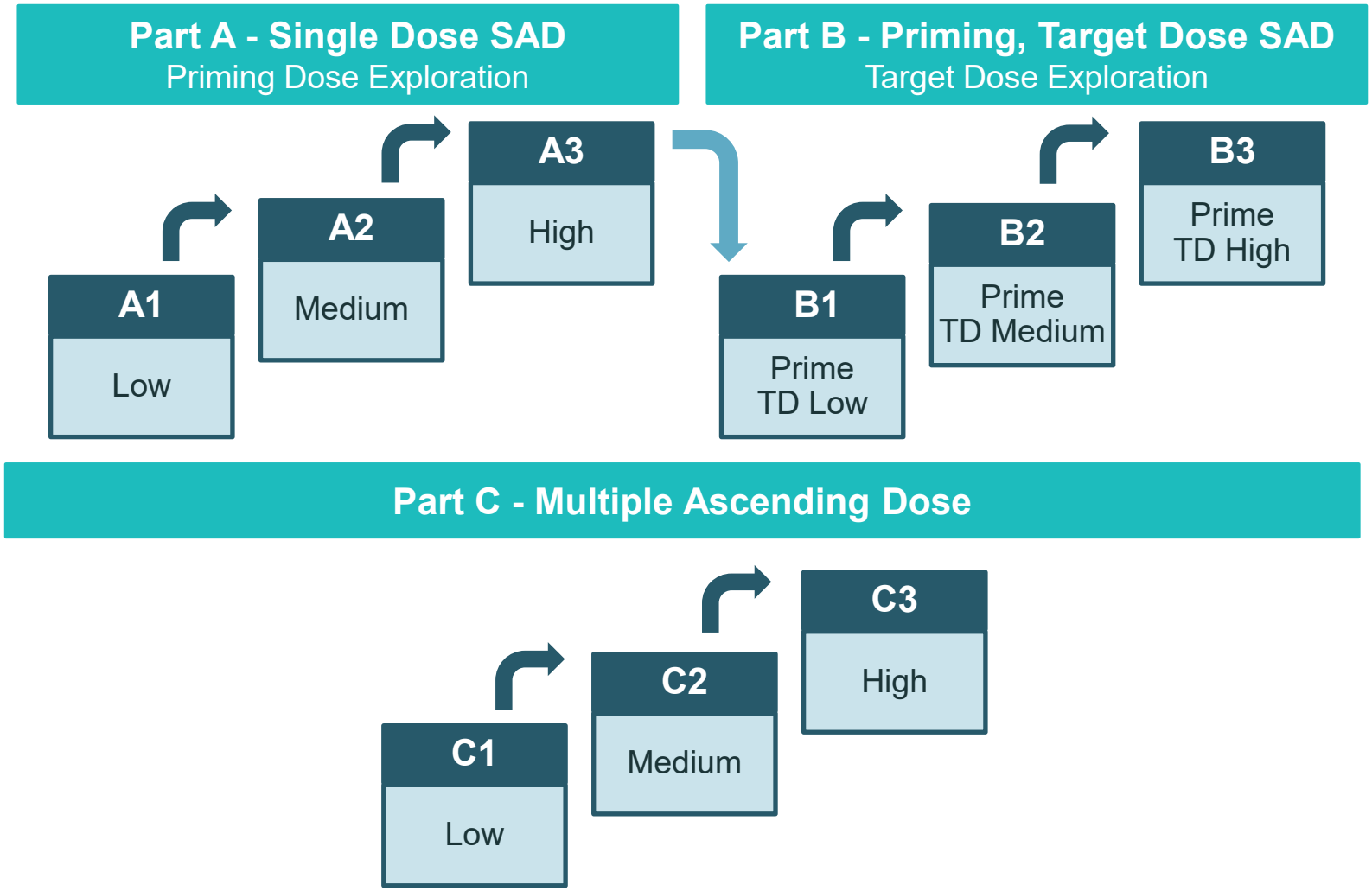
B cells were gated as CD45+CD2-/lowCD20+CD4-CD8a-CD159a-

XmAb657 is rationally designed for autoimmune disease, and a Phase 1 study was initiated in Q4 2025

- Has been observed to demonstrate deep and durable B-cell depletion in NHPs, enabled by potentially best-in-class pharmacokinetics
- Has been well tolerated in NHP with no clinical signs of CRS
- **Initiated Phase 1 study in healthy volunteers, idiopathic inflammatory myopathies (IIM), Sjögren's disease and systemic sclerosis in Q4 2025**



Phase 1 open-label study hybrid design of HV and AID patients



Population

- Healthy volunteers (HV) and autoimmune diseases (AID): idiopathic inflammatory myopathies (IIM), Sjögren's disease, systemic sclerosis

Study Design Elements

- Evaluate safety, tolerability, pharmacokinetic (PK), and pharmacodynamics (PD), and immunogenicity
- SAD and MAD cohorts
- SC administration

Endpoints

- Primary: Safety
- Secondary: Pharmacokinetics
- Exploratory: Immunogenicity, PD profile

SC subcutaneous administration **TD** target dose

Focused clinical execution is expected to deliver key clinical readouts in 2026 and advance programs with additional data in 2027

XmAb® Drug Candidate		Indication	1H 2026	2H 2026
Oncology Portfolio				
XmAb819	ENPP3 x CD3	ccRCC	Initiate tumor expansion cohorts for CRC, NSCLC and pRCC ✓	Present RP3D monotherapy data at ESMO 2026 ✓
XmAb541 + XmAb808	CLDN6 x CD3 + B7-H3 x CD28	CLDN6+ HGSOC		Ongoing dose escalation
Immunology Portfolio				
XmAb942	Xtend™ TL1A	IBD+	Present final Phase 1 healthy volunteer data at DDW 2026 ✓	Update on progress achieved in Phase 2b XENITH-UC study ~YE26
XmAb412	TL1A x IL23p19	IBD+	Present preclinical characterization at DDW 2026 ✓	Initiate first-in-human study ✓
Plamotamab	CD20 x CD3	Rheumatoid arthritis		Initial dose-escalation data from ongoing Phase 1b in RA at ACR ✓
XmAb657	CD19 x CD3	IIM, Sjögren's disease, Systemic sclerosis		Update on progress achieved in FIH dose-escalation study ✓

As of 09-Nov-2026 **ccRCC** clear cell renal cell carcinoma **CRC** colorectal cancer **NSCLC** non-small cell lung cancer **pRCC** papillary renal cell carcinoma **RP3D** recommended Phase 3 dose **HGSOC** high-grade serous ovarian carcinoma **IBD** inflammatory bowel disease **RA** rheumatoid arthritis **IIM** idiopathic inflammatory myopathies **FIH** first-in-human

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