



Advancing T Cell Engagers for Solid Tumors

Corporate Presentation

June 2026



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Building a Leading T cell Engager (TCE) Pipeline

Strategy



Developing potentially best-in-class TCE for solid tumors

- Tumor antigens that are clinically validated by antibody drug conjugates (ADC) or chimeric antigen receptor T cell therapy (CAR-T)
- Limited or weak competition addressing large market opportunities
- High affinity CD3 to maximize solid tumor response

Pipeline



CTIM-76: Claudin 6 (CLDN6) x CD3 bispecific antibody

- CLDN6 is overexpressed in ovarian, endometrial, lung, testicular, and other solid tumors
- CTIM-76 was designed to bind selectively to CLDN6 over similar claudin family members, including CLDN3/4/9

CT-95: Mesothelin (MSLN) x CD3 bispecific antibody

- MSLN is overexpressed in pancreatic, lung, colorectal, and other solid tumors
- CT-95 was designed to bind selectively to membrane-bound MSLN to enhance drug exposure and activity

CT-202: Nectin-4 x CD3 bispecific antibody

- Nectin-4 is overexpressed in bladder, lung, breast, colorectal, and other solid tumors
- CT-202 was designed to be conditionally active within the tumor microenvironment

Capitalization



Strong financial position with high quality investor base

- Expected cash runway into mid-2027

Pipeline Overview

PROGRAM	TARGET	ADDRESSABLE MARKET (U.S. ONLY)	PRECLINICAL	PHASE 1	PHASE 2	ANTICIPATED MILESTONES
CTIM-76	Claudin 6 (CLDN6)	> 50,000 patients				Q4 2026: QW updated data Q2 2027: Q3W dosing data
CT-95	Mesothelin (MSLN)	> 100,000 patients				Sept 2026: Preliminary Ph 1a data
CT-202	Nectin-4	> 125,000 patients				Q3 2026: Ph 1 FPI

CTIM-76: CLDN6 x CD3

Product differentiation: highly selective for CLDN6 over CLDN3/4/9

Safety: potent CD3 induction without broad cytokine activation

Potential Indications: ovarian, endometrial, lung

CT-95: MSLN x CD3

Product differentiation: avidity optimized to avoid mesothelin (MSLN) fragments

Safety: sterically hindered CD3 to avoid T cell crosslinking

Potential Indications: lung, pancreatic, ovarian, colorectal

CT-202: Nectin-4 x CD3

Product differentiation: conditionally activate in the tumor microenvironment

Safety: sterically hindered CD3 to avoid T cell crosslinking

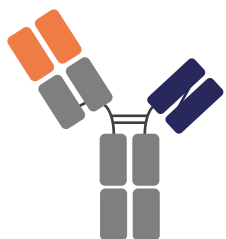
Potential Indications: bladder, colorectal, breast, lung

Context is Positioned to Develop the Next Generation of Transformative T Cell Engagers

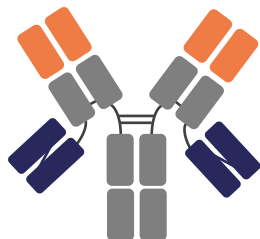
Potential to expand into early treatment lines through synergistic drug combinations

Optimized Novel Monotherapies

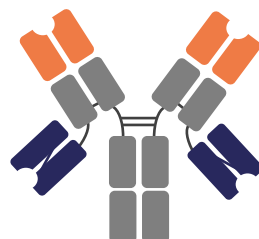
CTIM-76



CT-95



CT-202

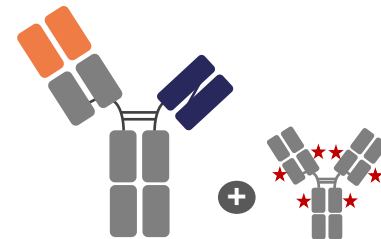


Bispecific TCE Engineered for:

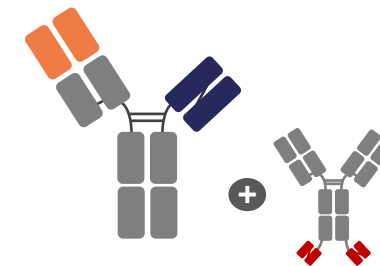
- Best-in-class efficacy
- Target selectivity
- Reduced risk of CRS
- Pharmacokinetics

Synergistic Combination Approaches

TCE + ADC



TCE + PD-1xVEGF



Potential Opportunities for:

- Complementary mechanisms to enhance activity
- Improved safety due to non-overlapping toxicities
- Synergistic immunologic effects

T Cell Engager Strategy



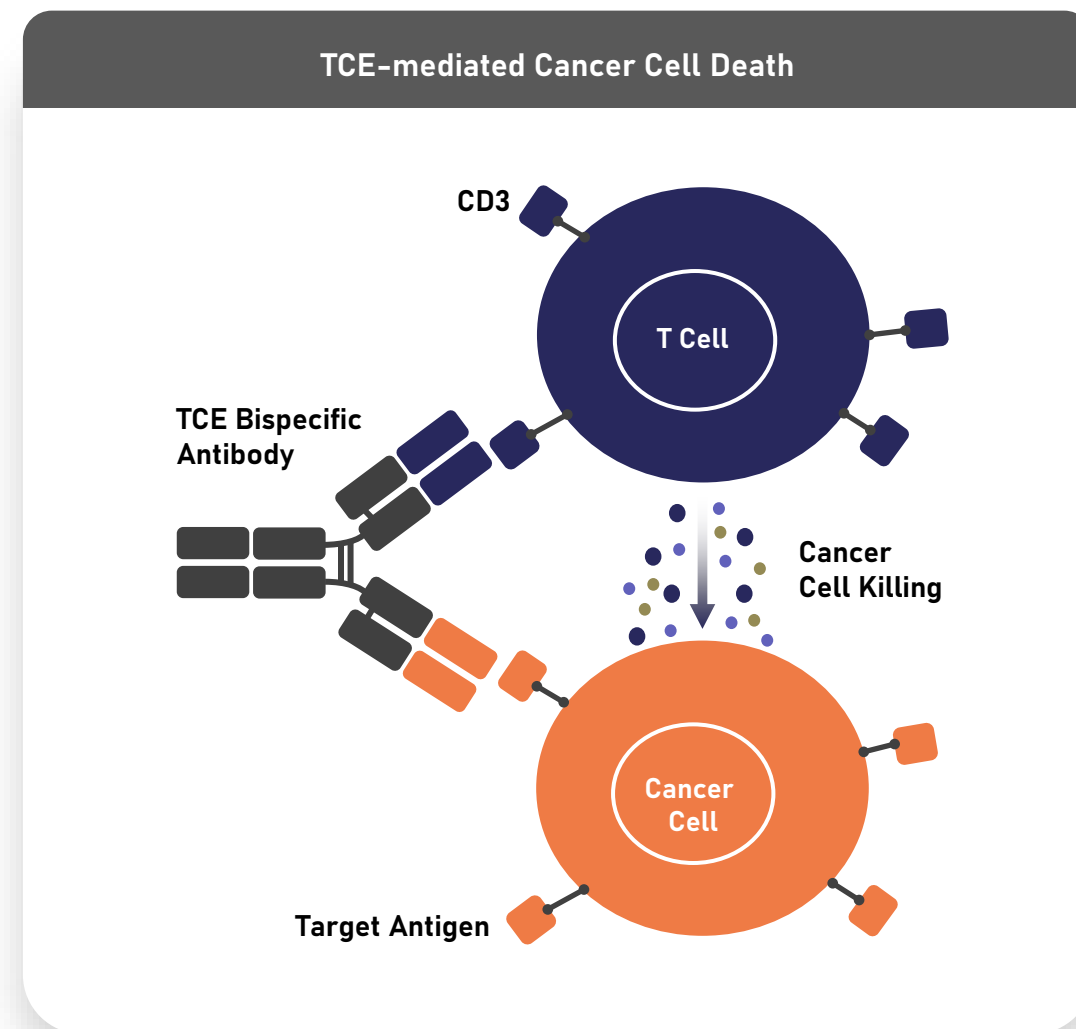
T Cell Engaging (TCE) Bispecific Antibodies

Mechanism of Action

T-cell engagers (TCEs) are bispecific antibodies designed to redirect cytotoxic T lymphocytes toward malignant cells.

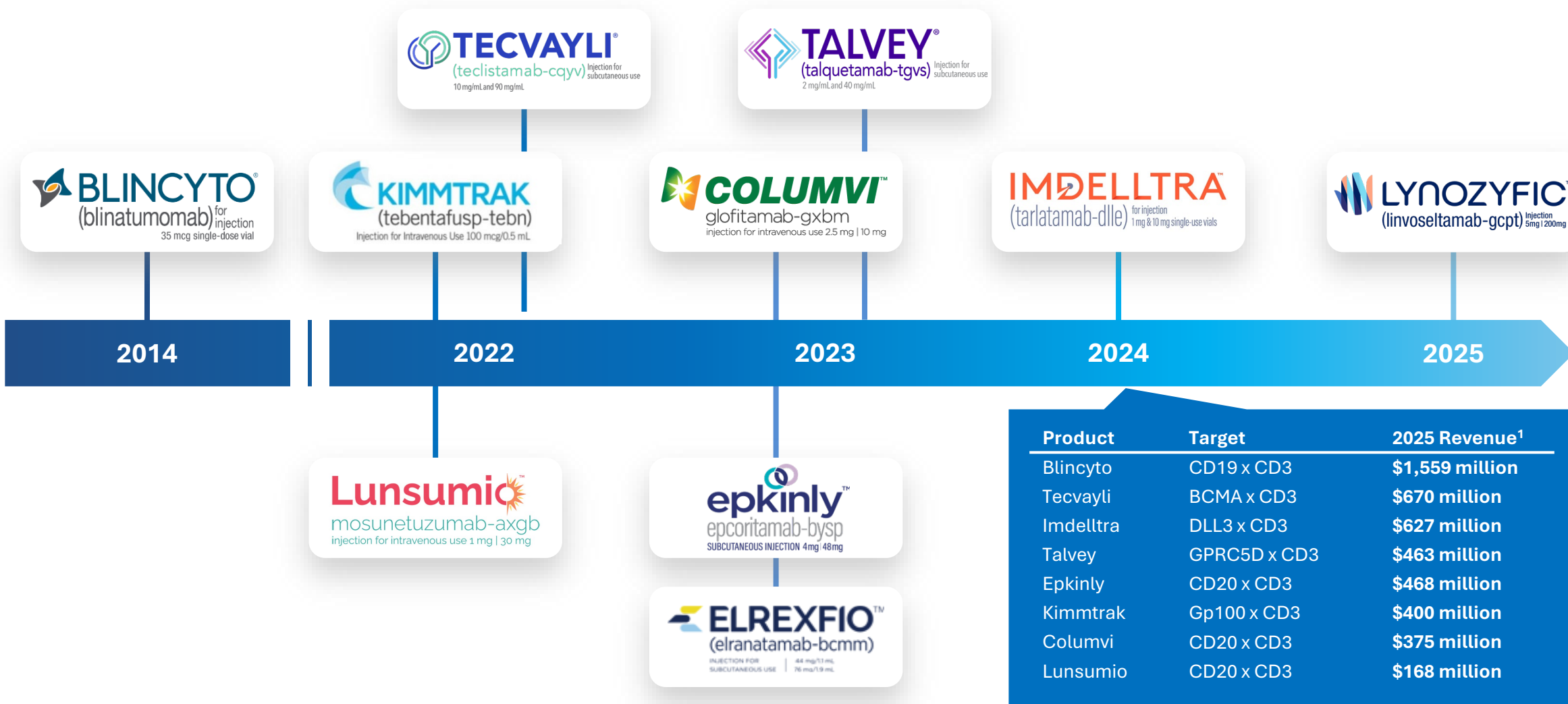
These molecules simultaneously bind to a tumor-associated antigen on the cancer cell and to CD3, a component of the T-cell receptor complex.

This dual engagement facilitates T-cell activation, immune synapse formation, and targeted cytotoxicity of tumor cells.












10 FDA Approvals for TCE for Solid and Liquid Tumors

Recently approved TCE have had strong initial commercial launches



TCE Success in Solid Tumors

									
Asset	Tarlatamab (AMG757)	HPN328	QLS31905	IBI389	VIR-5500	JANX007	JNJ-78278343	Xaluritamig (AMG509)	Ubatamab (REGN4018)
Target x Effector	DLL3 x CD3	DLL3 x CD3	CLDN18.2 x CD3	CLDN18.2 x CD3	PSMA x CD3	PSMA x CD3	KLK2 x CD3	STEAP1 x CD3	MUC16 x CD3
Cancer Indication	Small Cell Lung	Small Cell Lung	Pancreatic	Pancreatic	Prostate	Prostate	Prostate	Prostate	Ovarian
Patients (n)	100	73	12	27	11	8	33	21	13
Efficacy	ORR: 40% PFS: 4.9 mos.	ORR: 55% DoR: 10.8 mos.	ORR: 25% PFS: 3.9 mos.	ORR: 38%	PSA50: 100% ORR: 36% PFS: ~6 mos.	PSA50: 100% ORR: 13% PFS: 7.9 mos.	PSA50: 42% ORR: 8% PFS: 7.9 mos.	PSA50: 50% ORR: 20% PFS: 7.8 mos.	ORR: 31%
Grade ≥ 3 CRS	1%	1%	3%	0%	2%	8%	0%	2%	0%
Reference	Ahn 2023	ESMO 2025	ASCO 2025	ASCO 2024	ASCO GU 2026	Dec 2025 data cutoff	ASCO 2025	ESMO 2024	ESMO 2022

TCE efficacy in cold tumors with a low rate of cytokine release syndrome (CRS)

CTIM-76

CLDN6 x CD3 bispecific antibody



CLDN6 Therapies Have the Potential to Reach a Large Patient Population

>50,000 patients per year in the United States in Relapse/Refractory (R/R) Setting

Initial indications of interest based on:

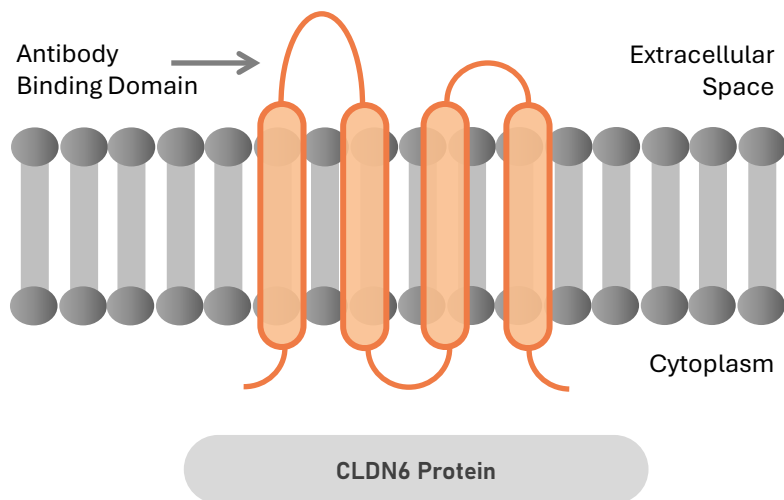
- CLDN6 prevalence
- Patient population size
- CLDN6 target validation

Selected Cancer indications	Incidence (US Only)	R/R Incidence	CLDN6 Positive	CLDN6 Med/High	Patient Population Based on R/R Incidence
Ovarian	21,010	19,500	75% ¹	35% ¹	14,625
Endometrial	68,270	17,000	50% ¹	22% ¹	8,500
Testicular	9,810	750	100% ³	>95% ³	750
Non-Small Cell Lung	199,586	122,500	26% ²	6% ²	31,850
Colon	158,850	36,600	43% ³	0% ³	15,738
Breast	321,910	66,000	40% ³	0% ³	26,400

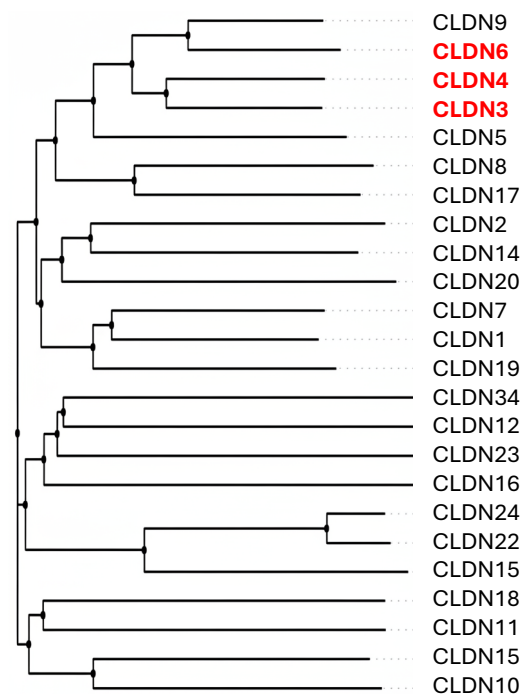
CLDN6 is an Attractive Target for Immunotherapy

CLDN6 is an Ideal TCE Target

- CLDN6 is an oncofetal protein. Normally present at higher levels during embryonic development
- Turned off or have low levels of expression in adult tissues
- Expression increases with cancer disease stage
- Tetraspan protein; does not readily internalize



Avoiding CLDN3 and CLDN4 is a Critical Safety Determinant



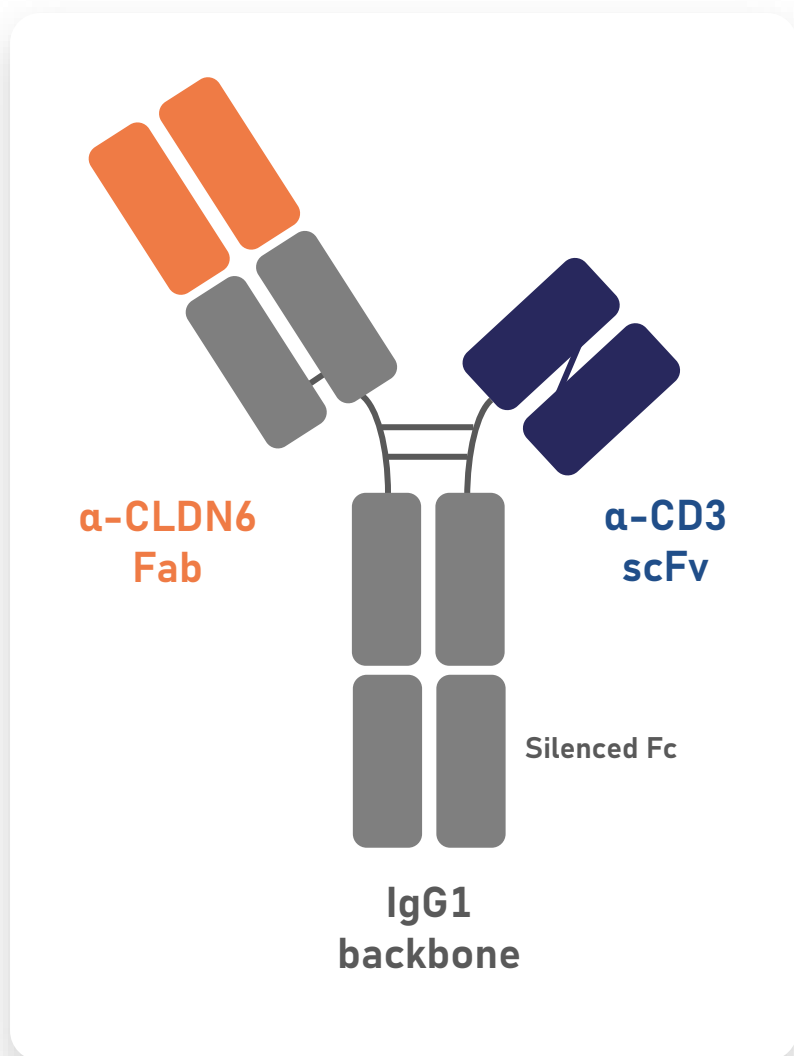
Claudin Gene Family

CLDN6 selectivity is required to avoid off-target liabilities

The CLDN6 antibody binding region is **highly conserved** with CLDN3 and CLDN4 – differing by only 3 amino acids¹

CLDN3 and CLDN4 are enriched in the liver and antibody binding may result in **liver enzyme elevations**^{2,3}

CTIM-76: Claudin 6 x CD3 T cell Engaging (TCE) Bispecific Antibody



Optimized structure for CLDN6 selectivity, potency, and manufacturability

- Highly selective CLDN6 binding fragment antibody-binding (Fab) arm
- Immunostimulatory CD3 binding single-chain fragment variable (scFv) domain is functionally monovalent to avoid aberrant T cell activation
- Silenced Fc domain to avoid off target immune cell activation

Potentially wide therapeutic window

- T cell dependent cellular cytotoxicity with no or minimal activation of circulating cytokines
- Humanized CLDN6 and CD3 binding domains

Ease of manufacturing

- IgG1 backbone is highly stable and enables high yield

Positive Phase 1a Clinical Data Observed for CTIM-76

CTIM-76 has been granted FDA Fast Track Designation



Pan-PROC Target

~75% of platinum resistant ovarian cancer (PROC) patients are CLDN6+

Potential for Rapid Clinical Enrollment



Clinical Activity in Late Line PROC

7 (5-16) prior lines of therapy

29% confirmed ORR
57% confirmed DCR
66% of patients on therapy

Potential to Address ADC Resistance in PROC



Low Rate of CRS in PROC

11% Grade 1 CRS at target dose levels

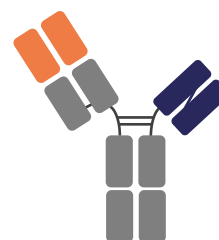
Potential for Outpatient Dosing

Significant Market Opportunity in High Unmet Need CLDN6+ Post-ADC PROC

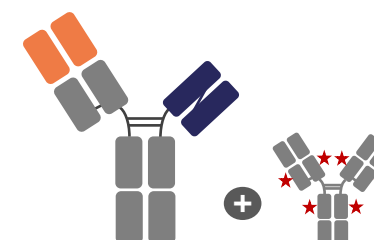
- **~75%** of PROC patients are CLDN6+
- **No approved treatment option** specifically for this biomarker selected PROC population
- **~42,000** CLDN6+ PROC patients globally (US, EU7, JP)
- CLDN6 has **significant target overlap** with promising ADC targets, including FR α , CDH6, NaPi2b – opening the door to potential combination opportunities
- **Additional opportunities** in earlier lines of ovarian cancer and other tumor types with CLDN6 overexpression, including non-small cell lung (NSCLC) and endometrial cancer

Broad Use Case as Monotherapy or Combination

CTIM-76



CTIM-76 + ADC



Potential Opportunities for:

- Monotherapy development post-ADC
- Combination development with ADC, VEGF, or chemotherapy

CTIM-76 Phase 1a Dose Escalation Trial

Target population

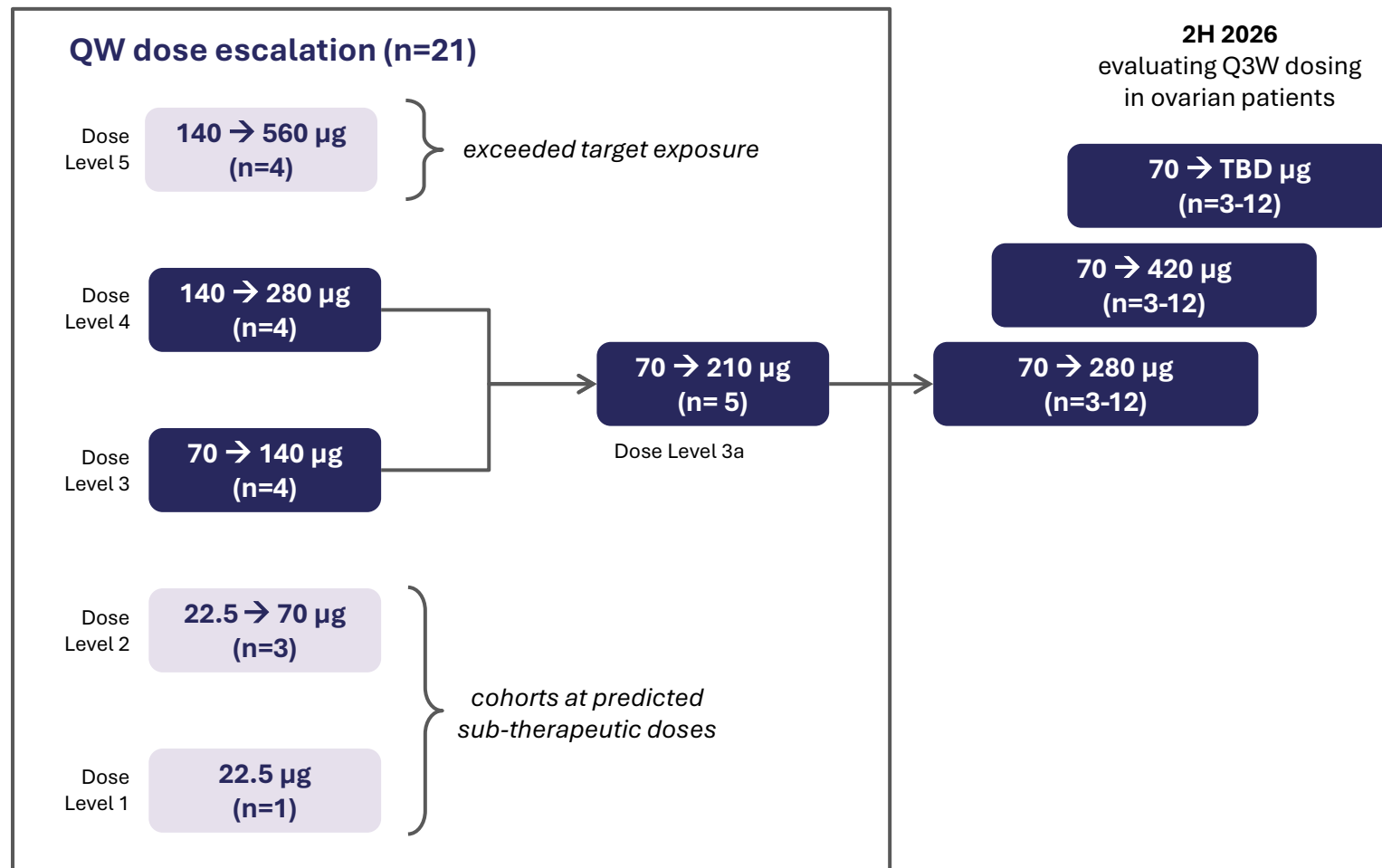
- Ovarian, endometrial and testicular cancer relapsed to standard of care (all comers)
- CLDN6+ positive via IHC ($\geq 10\%$ 1+ staining)

Trial objectives

- Assess safety and tolerability
- Pharmacokinetic and pharmacodynamic data
- Evaluate preliminary anti-tumor activity

Dosing and Administration

- Step dosing
- Pretreat patients with 16 mg dexamethasone 1 hour prior to C1D1 and C1D8 doses



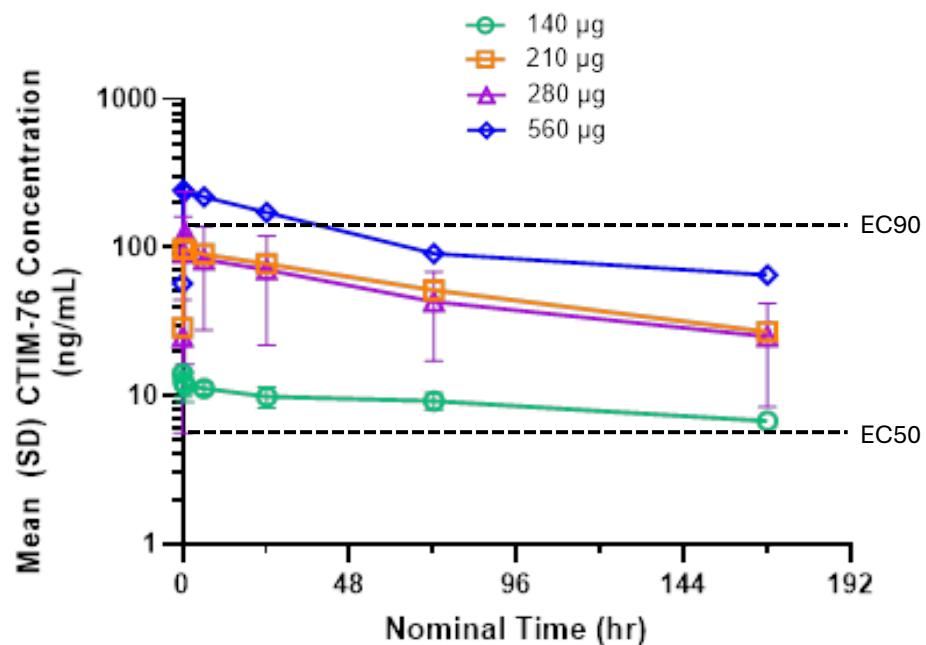
Path to the identification of optimal prime dose, full dose, and dosing schedule

CTIM-76 Exhibits Compelling Pharmacokinetic (PK) Properties

Weekly Dosing (QW)

210 and 280 μ g doses displayed optimal PK properties, including C_{max} below target saturation (EC₉₀) and C_{min} above activity threshold (EC₅₀)

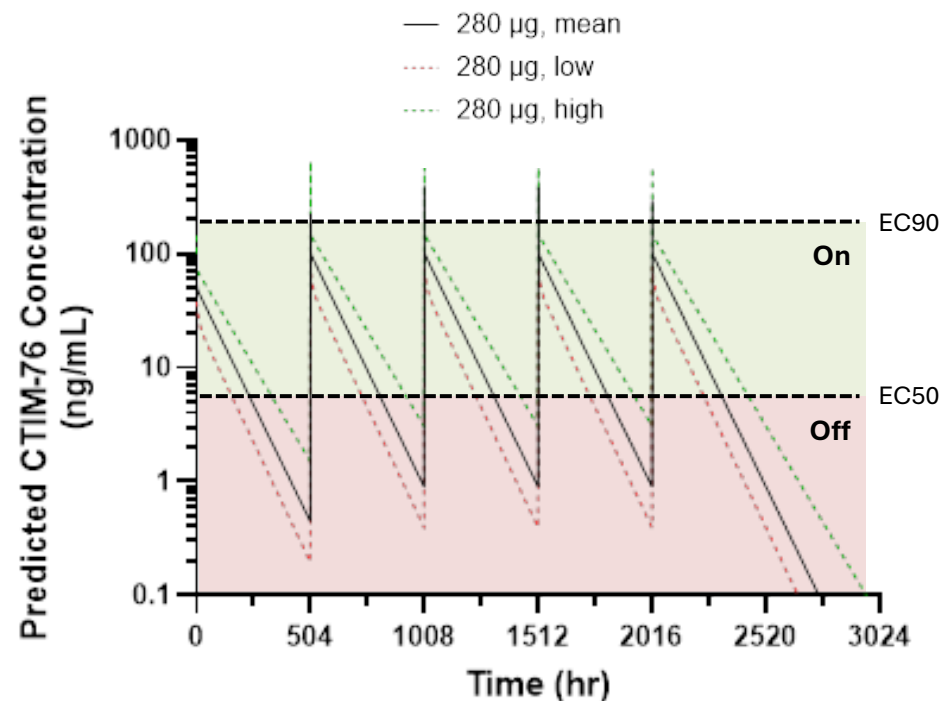
560 μ g resulted in target saturation and was associated with rapid T cell exhaustion



Every Three Week (Q3W) PK Simulation

Reducing TCE dose frequency has been shown to reduce T cell exhaustion, resulting in improved treatment response and durability¹

Q3W dosing of CTIM-76 may optimize T cell activation and recovery through on/off cycling



Patient Demographics at Target Doses of 140 to 280 µg

CTIM-76 evaluated in heavily pre-treated patients with high tumor burden

Baseline Characteristics	All Comers
N	13
Age, n (range)	61 (29-72)
ECOG, n (range)	1 (0-1)
Ovarian, n (%)	9 (69)
Testicular, n (%)	3 (23)
Endometrial, n (%)	1 (8)
Prior therapies, median (range)	6 (3-16)
1, n (%)	0 (0)
2	0 (0)
3	3 (23)
4	1 (7)
≥5	9 (69)

Baseline Characteristics	Ovarian
N	9
Age, n (range)	63 (53-74)
ECOG, n (range)	0 (0-1)
Sum of longest dimension (mm)	71 (39-114)
Liver metastases, n (%)	4 (44)
H-Score, median (range)	145 (25-300)
Prior therapies, median (range)	7 (5-16)
Checkpoint Inhibitor, n (%)	5 (55)
ADC	8 (89)
DNA Repair	7 (78)
VEGF	9 (100)

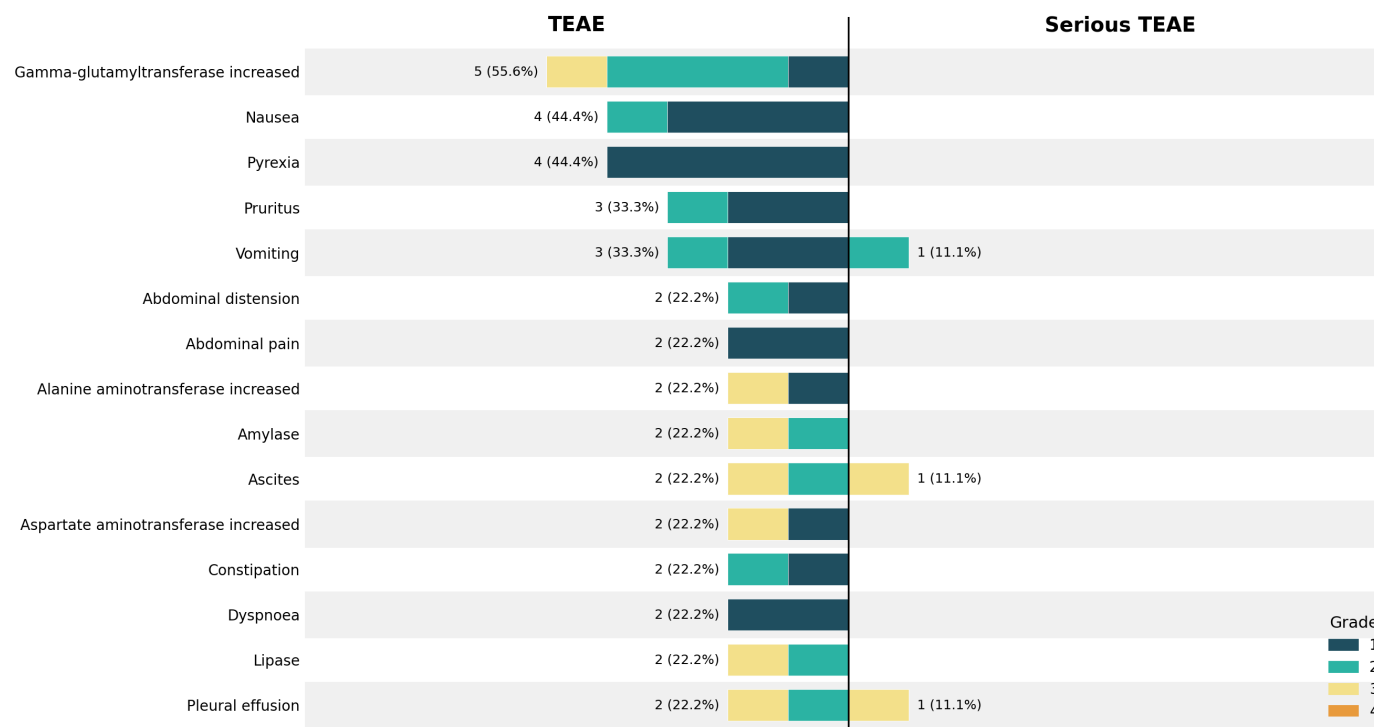
Safety Analysis at Target Doses of 140 to 280 µg

Observed to be well-tolerated with a favorable safety profile

- Adverse events (AE) generally occurred during the first or second dose
- Most events were low grade, of short duration, and were reversible with standard management
- Low rate of cytokine release syndrome (CRS)

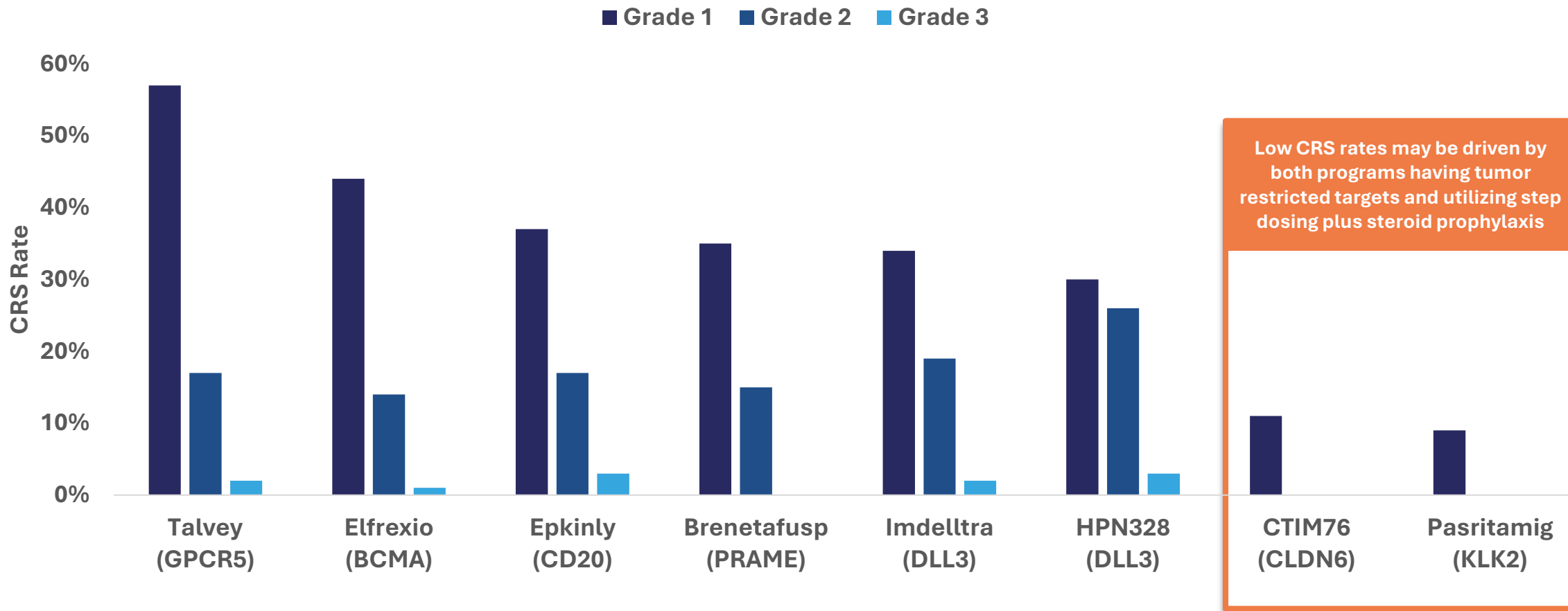
Treatment Emergent Adverse Events (TEAE)	All Comers	Ovarian
N (%)	13	9
Any	13 (100)	9 (100)
Related	13 (100)	9 (100)
Serious	5 (39)	3 (33)
Related Serious	2 (15)	1 (11)
Grade 1 CRS	2 (15)	1 (11)
Grade ≥2 CRS	0 (0)	(0)
Dose Reduction	0 (0)	0 (0)
Discontinuation	0 (0)	0 (0)

Ovarian: overall TEAE and Serious TEAE for by Maximum Severity Grade (N=9)



CRS in PROC Patients at Target Dose Levels

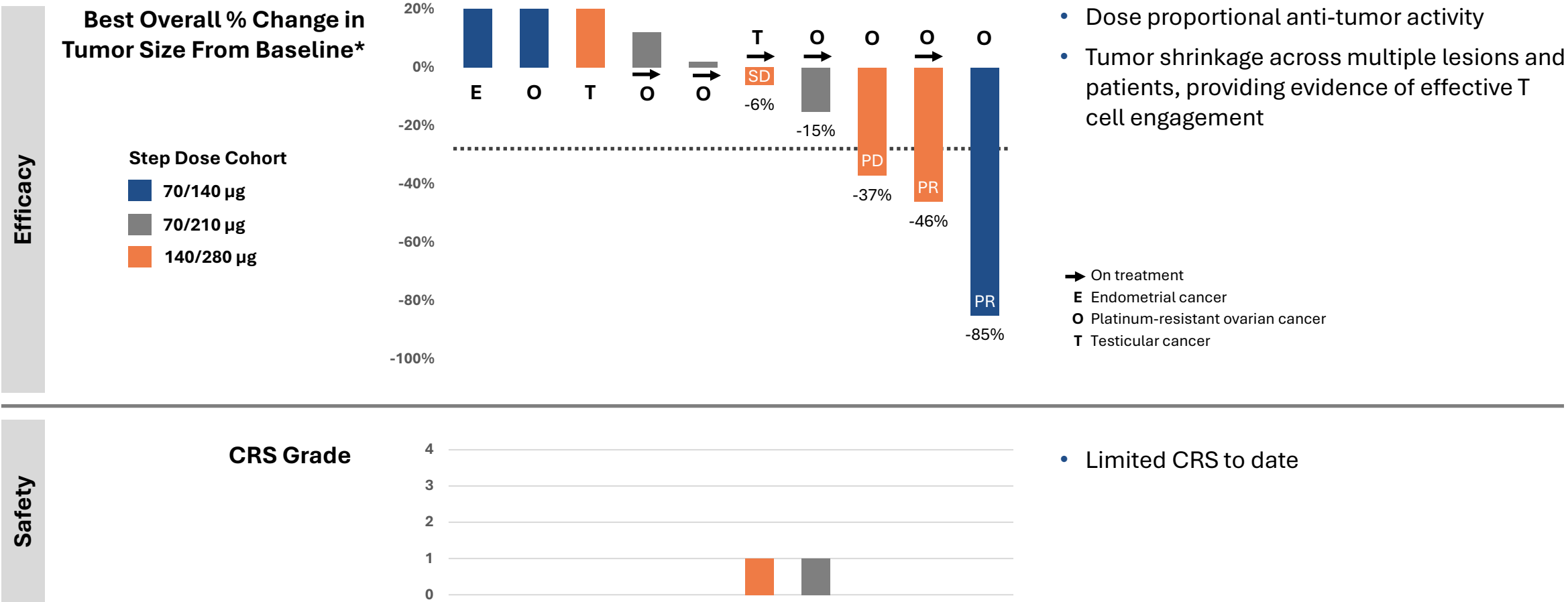
Benchmarking CRS profiles across approved and experimental TCE



CTIM-76-101 ClinicalTrials.gov Identifier: NCT06515713. Data as of May 29, 2026. CTIM-76 CRS derived from 16% Grade 1 CRS in all comers patients (n=13). Cross-trial data interpretation should be considered with caution as it is limited by differences in study population, sample size, inclusion and exclusion criteria and many other factors. Imdelltra; Epkinly; Elfrexio; Talvey: Prescribing label. HPN329: Beltran ASCO 2024. Brenetafusp: Freidman ESMO 2024. Pasritamig: Autio ESMO 2025, Stein ASCO 2025

CTIM-76 Interim Phase 1a Data at Target Doses

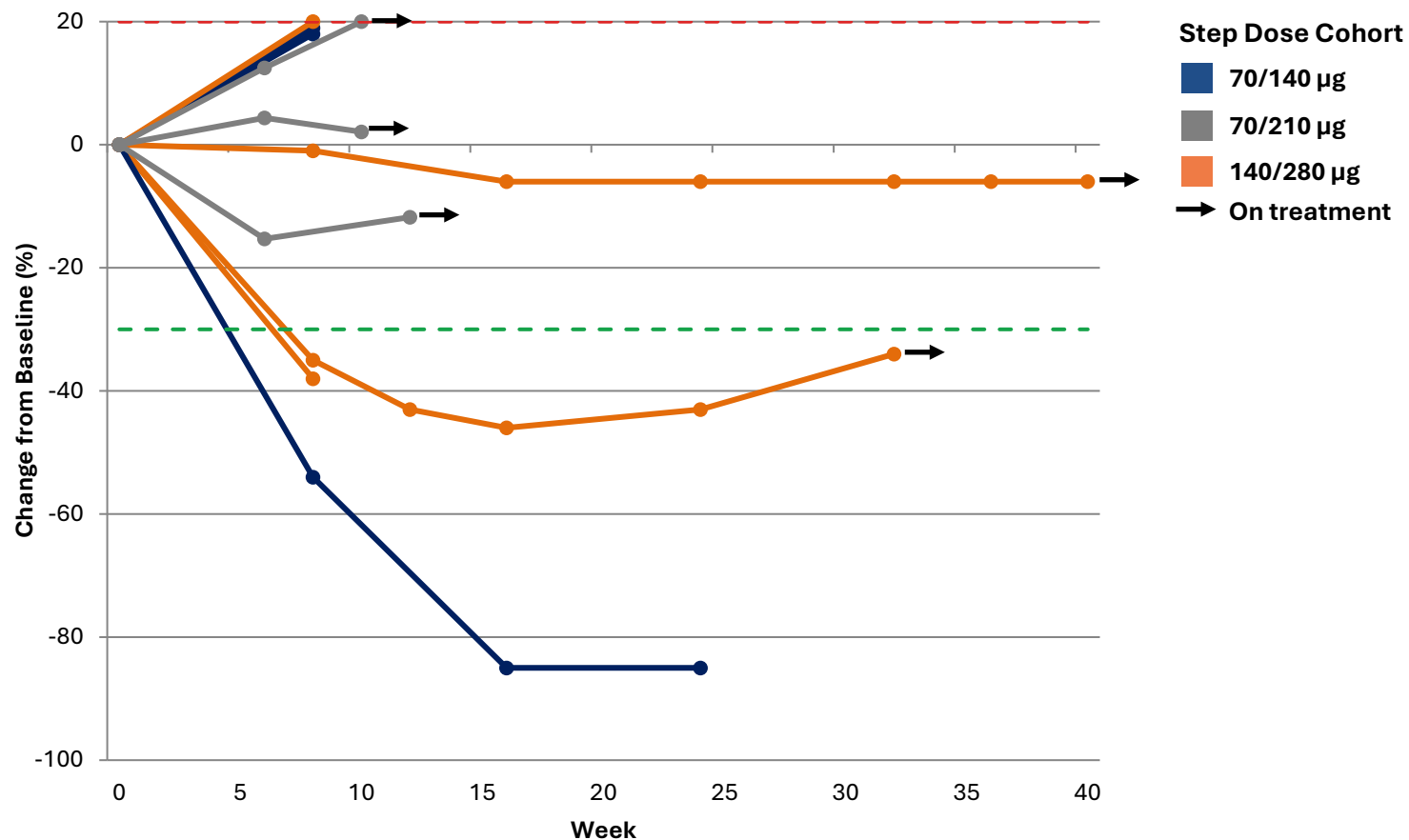
Tumor reduction combined with manageable CRS profile supports continued clinical development



CTIM-76 Anti-tumor Activity at Target Dose Levels

Emerging durability signal, despite weekly dosing during escalation phase

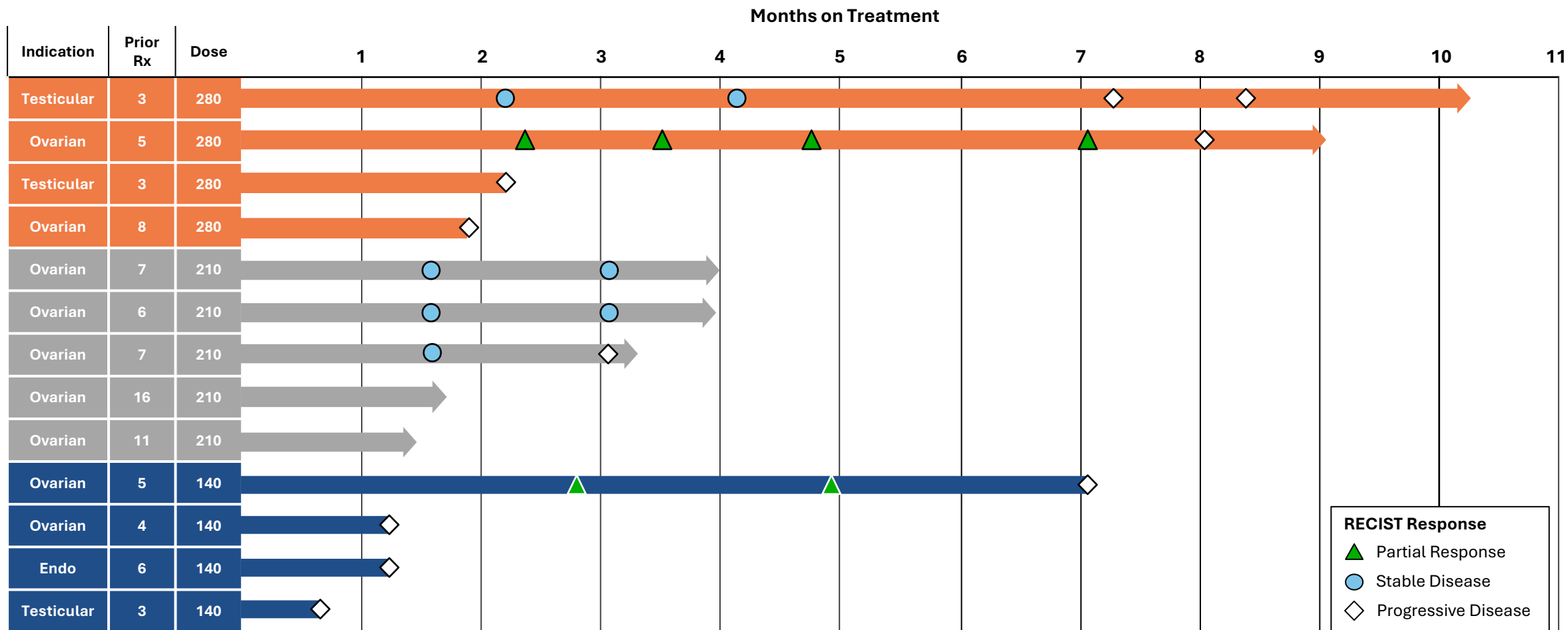
Spider Plot



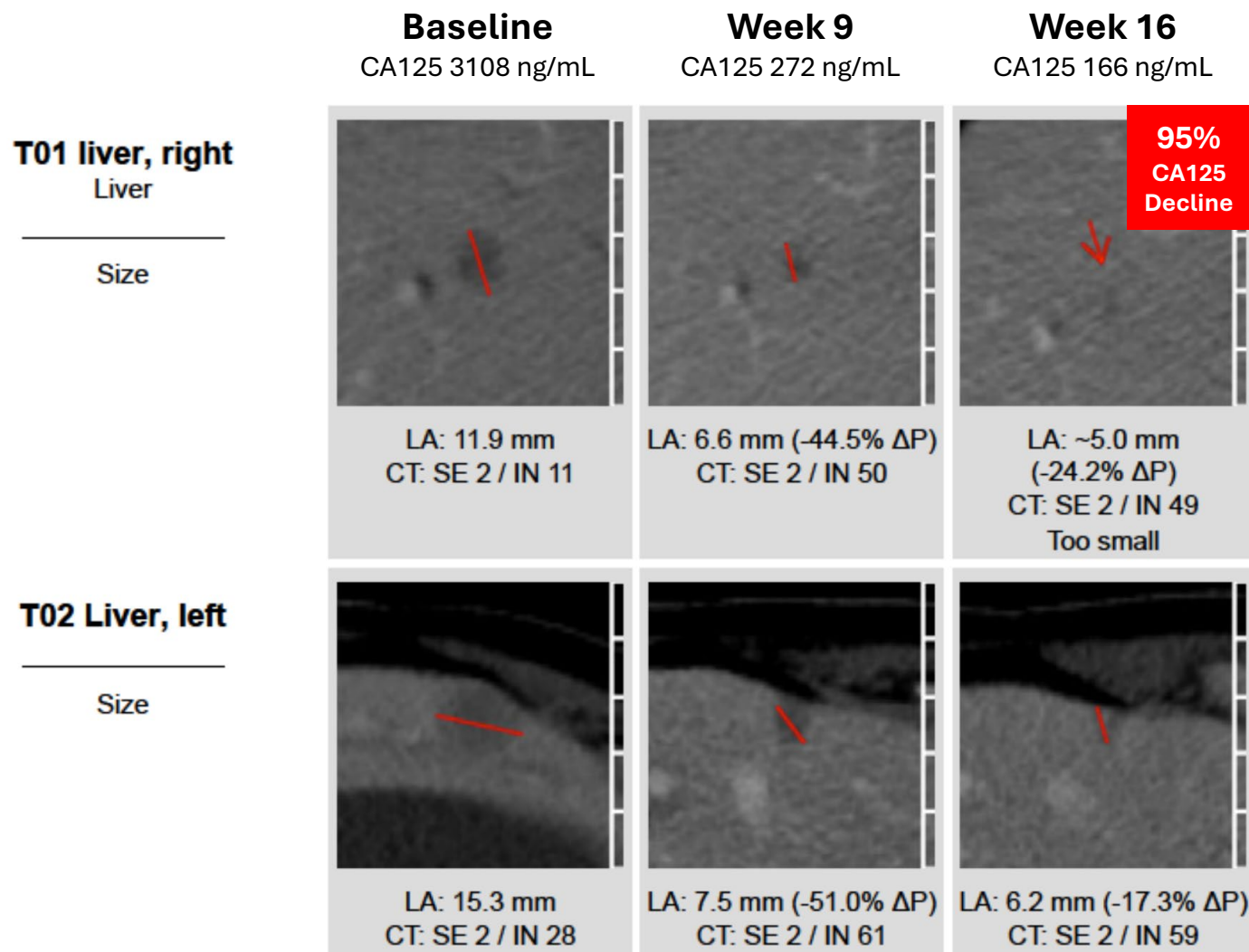
	All Comers	Ovarian
Patient enrolled, n	13	9
RECIST evaluable, n	10	7
Not RECIST evaluable, n	1	0
Pending 1st Scan	2	2
Overall Response Rate (ORR), n (%)	2 (20)	2 (29)
Stable Disease (SD), n (%)	3 (30)	2 (29)
Disease Control Rate (DCR), n (%)	5 (50)	4 (57)

CTIM-76 Anti-tumor Activity at Target Dose Levels

Emerging durability signal with the potential to further improve with less frequent CTIM-76 dosing



Significant RECIST Response in Ovarian Patient with Liver Lesions

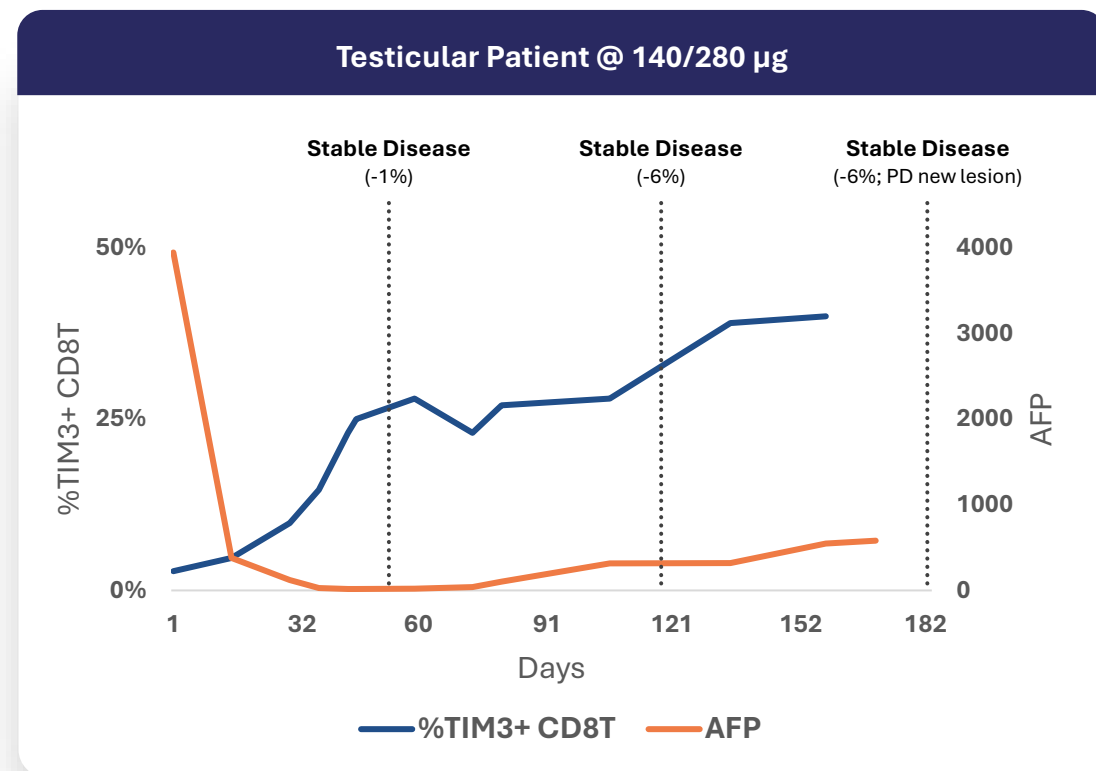
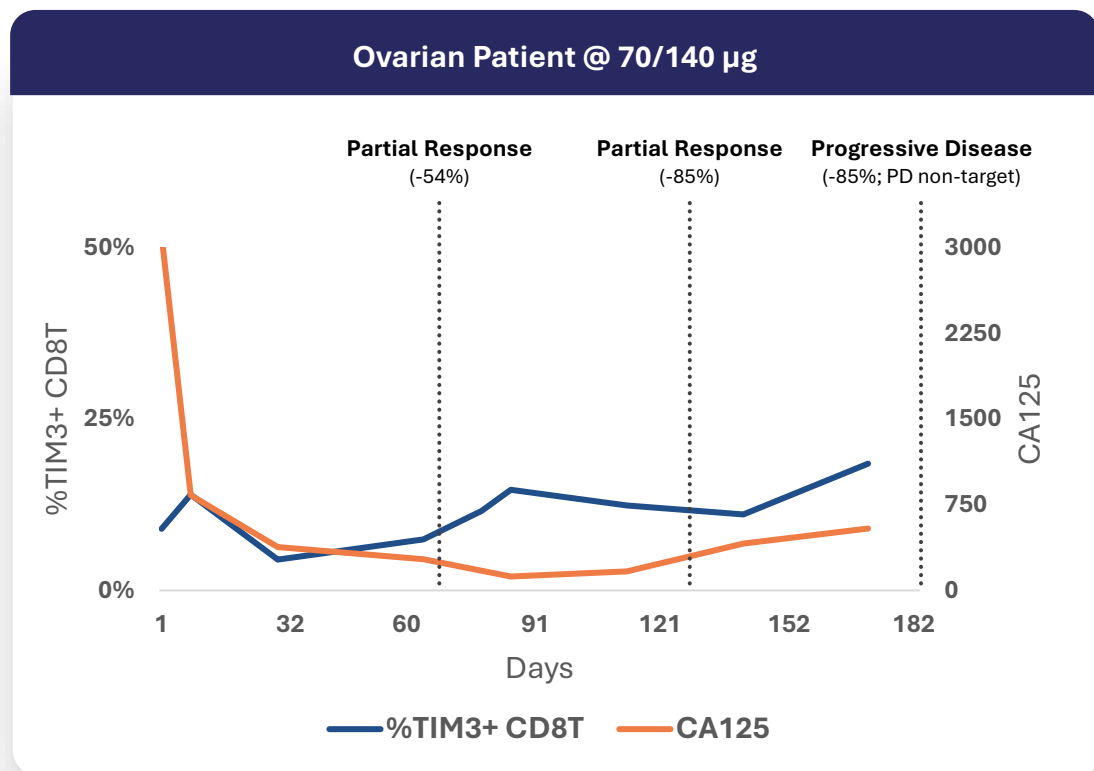


Patient Case Study Detail

- PROC patient administered 140μg
- 53-year-old female
- High disease burden; liver and peritoneum
- 5 prior lines of treatment and treatments included mirvetuximab, pembrolizumab, olaparib, VLS-1488
- Rapid progression on two prior therapies
- Confirmed PR, 85% decrease in tumor diameter
- Disappearance of peritoneal target lesion
- On treatment for 162 days; progression due to new lesions

T Cell Exhaustion is a Critical Determinant of Tumor Response Durability

Q3W dosing may delay T cell exhaustion, improve treatment durability, and be more convenient for patients¹



- Response profiles for two patients with similar tumor burden and H-scores were compared
- Markers of T cell exhaustion (%TIM3+ CD8T) were dose proportional

Competitive Profile for CTIM-76 in PROC

Encouraging tumor response and safety profile in heavily pretreated / ADC-experienced patient population

	CTIM-76	Multiple	Nab-Paclitaxel	Azenosertib	Brenetafusp
Target	CLDN6	CDH6, FR α , NaPi2b	Microtubule	Wee1 (CCNE1 amplified)	PRAME
Format	TCE	ADC	Chemotherapy	Small Molecule	TCE
Prior ADC (%)	89	3-20	3	15	11
Prior Lines, median	7	3-5	2	3	4
ORR (%)	29	46-55	30	33	6
Trial	NCT06515713	REJOICE Ovarian-01, Rainfol-01, NAPISTAR	ROSELLA	DENALI Part 1b	ESMO 2024

CT-95

MSLN x CD3 bispecific antibody



MSLN Therapies Have the Potential to Reach a Large Patient Population

>100,000 patients per year in the United States in Relapse/Refractory (R/R) Setting

Selected Cancer indications	Incidence (US Only)	R/R Incidence	MSLN Positive	MSLN Med/High	Patient Population Based on R/R Incidence
Non-Small Cell Lung	199,586	122,500	55%	36%	67,375
Pancreatic	67,530	47,850	80%	61%	38,280
Colon	158,850	36,600	41%	17%	15,006
Ovarian	19,900	19,500	90%	80%	17,550
Mesothelioma	3,000	2,500	70%	60%	1,750
Esophageal	22,530	16,290	41%	26%	6,679
Endometrial	68,270	17,000	45%	23%	7,650
Gastric	31,510	11,200	49%	23%	5,488

Initial indications of interest based on:

- MSLN prevalence
- Patient population size
- MSLN target validation

MSLN Target Biology

Shed mesothelin (sMSLN) in tumor microenvironment requires a creative solution to overcome

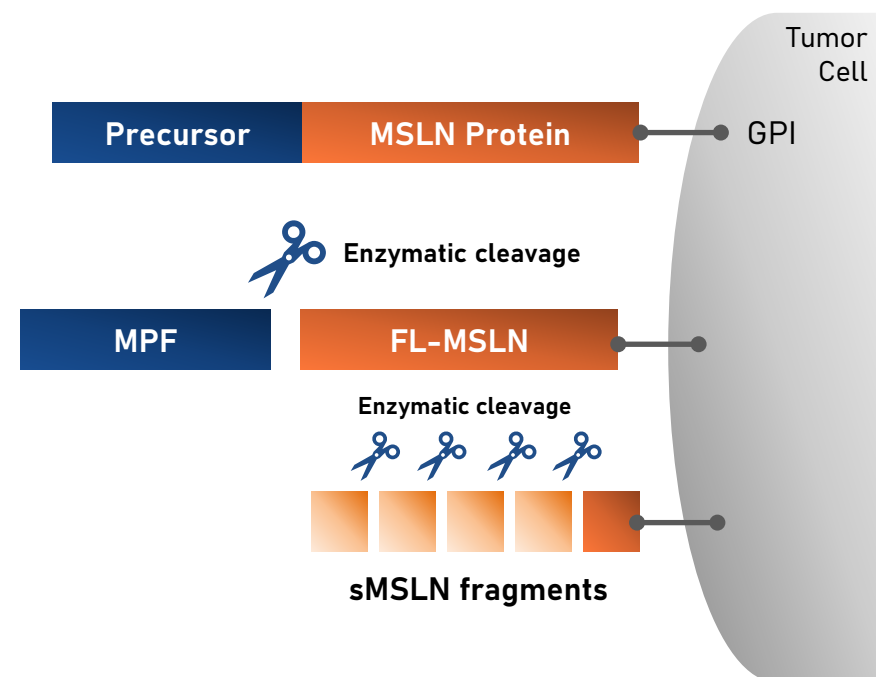
Overcoming Fragmented MSLN in the Tumor Microenvironment

MSLN is bound to tumor cells via a GPI-anchor

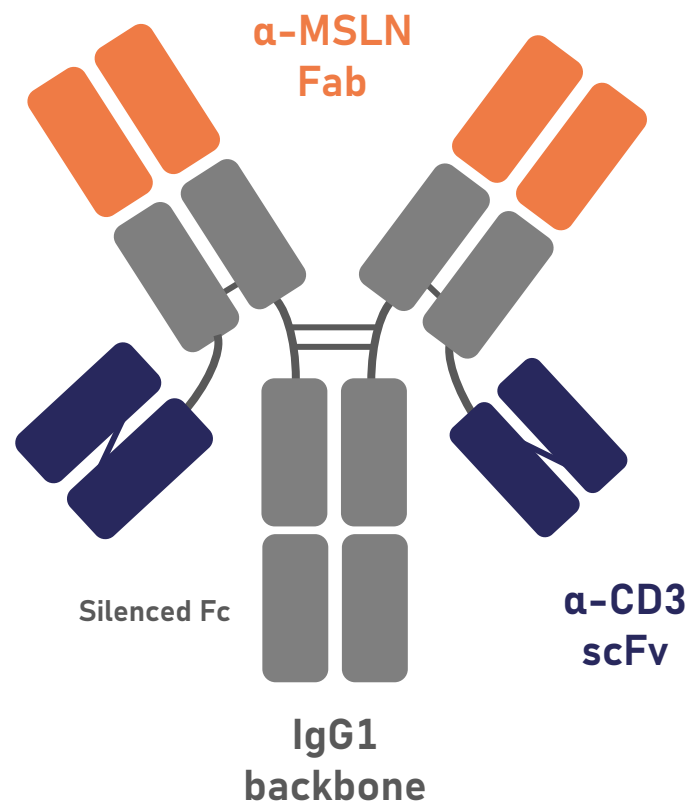
Like many GPI-anchored proteins, MSLN can be cut into smaller fragments^{1,2}

The MSLN gene encodes a precursor that is cleaved into two products: a soluble N-terminal protein called megakaryocyte potentiating factor (MPF), and a membrane-bound fragment called full length mesothelin (FL-MSLN)

sMSLN serves as a competitive sink, preventing antibodies from binding to the tumor, which can lead to suboptimal drug exposure and efficacy



CT-95: MSLN x CD3 T cell Engaging (TCE) Bispecific Antibody



Novel design to overcome mesothelin (MSLN) sink

- Binds to membrane-proximal MSLN epitope
- Affinity tuned MSLN binding
- Cooperative binding results in high affinity binding of CT-95 to tumor

Potentially wide therapeutic window

- No crosslinking by shed MSLN, mitigating off-tumor T cell activation
- Cooperative binding of MSLN on tumor surface crosslinks CD3, activating T cells

Ease of manufacturing

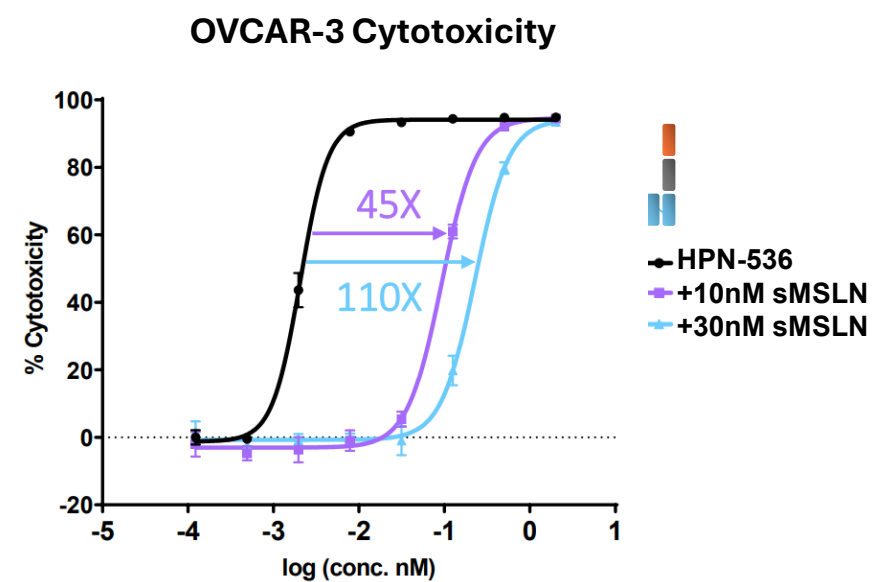
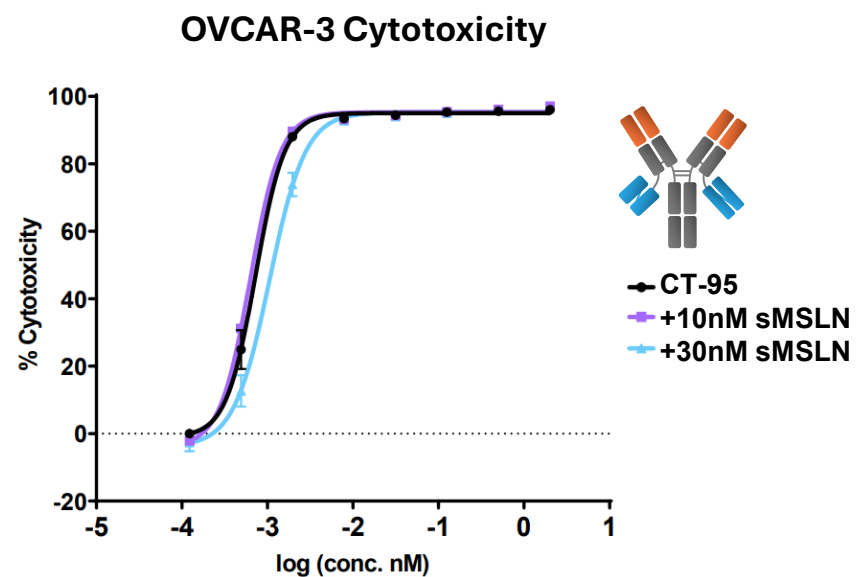
- IgG1 backbone is highly stable and enables high yield

Anticipate Preliminary Phase 1a Data in September 2026

CT-95 Intended to Overcome MSLN Sink

HPN-536 (Harpoon Therapeutics) binds to soluble MSLN (sMSLN) in a dose proportional manner, limiting therapeutic exposure

Cytotoxicity in the Presence of Soluble MSLN



CT-95 Phase 1a/b Study

An open-label, multi-center, dose escalation / expansion, safety, and PK study (NCT06756035)

Biomarker stratification

- Ovarian, pancreatic, and mesothelioma do not require prospective screening
- All other indications require prospective MSLN screening via IHC ($\geq 10\%$ 1+ staining)

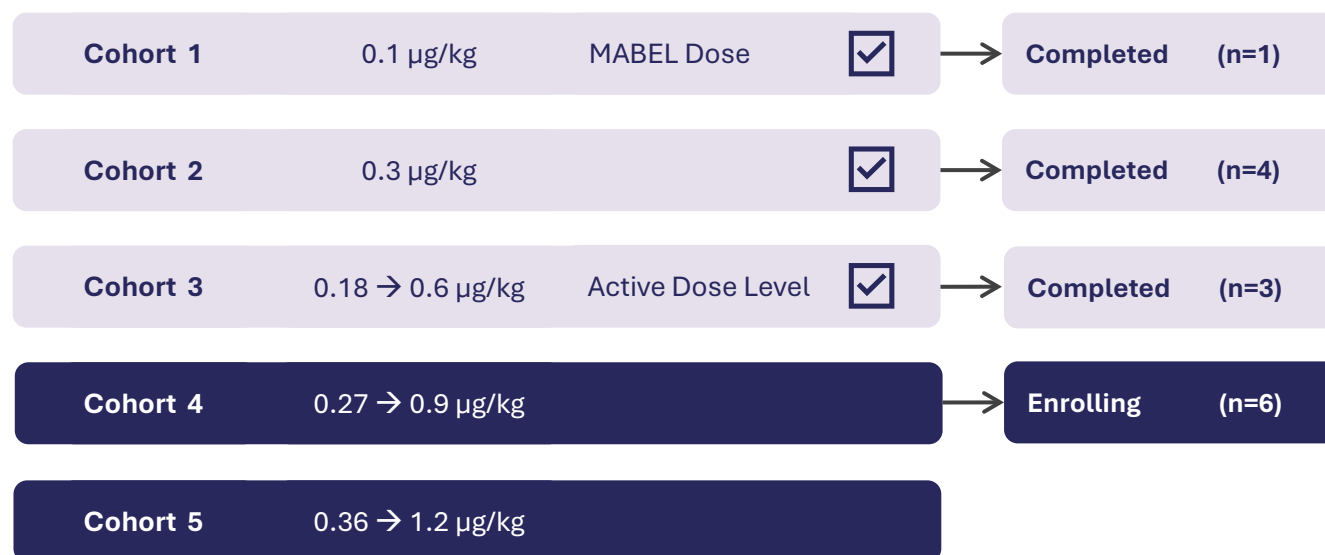
Trial objectives

- Assess safety and tolerability at increasing dose levels
- Pharmacokinetic and pharmacodynamic data
- Evaluate preliminary anti-tumor activity

Dosing and Administration

- Weekly IV infusion
- Single step dose
- Pretreat patients with 10 mg dexamethasone 1 hour prior to C1D1 and C1D8 doses

Phase 1a Dose Escalation



CT-95 Competitive Landscape

CT-95 is avidity enhanced, affinity tuned, and binds to membrane-bound MSLN

	CT-95	AMG-305	HPN-536	JNJ-79032421	ZW171
Company	Context	Amgen ⁴	Harpoon ¹	JNJ	Zymeworks ^{2,3}
Format	2 + 2	1 + 1 + 2 CDH3 + MSLN dBiTE	TriTAC	1+1	2 + 1
Avoids sMSLN	✓	X	X	✓	X
Affinity Tuned	✓	X	X	X	X
High Affinity CD3	✓	X	✓	X	X
Program Status	Phase 1 FPI Apr 2025	Phase 1 FPI Oct. 2023	Phase 1 Discontinued	Phase 1 Discontinued	Phase 1 Discontinued



CT-202

Nectin-4 x CD3 bispecific antibody

Nectin-4 Therapies Have the Potential to Reach a Large Patient Population

>125,000 patients per year in the United States in Relapse/Refractory (R/R) Setting

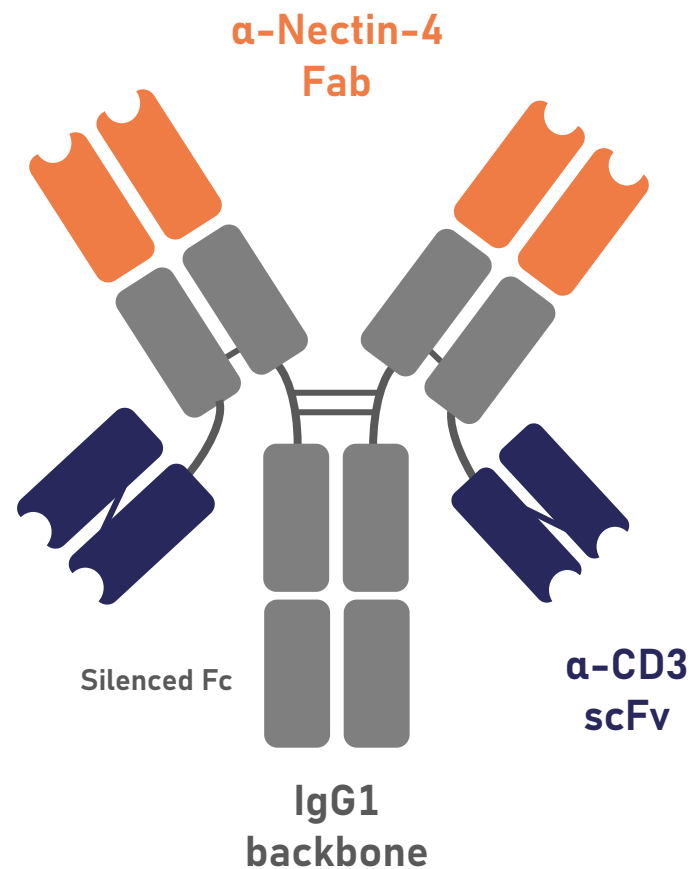
Initial indications of interest based on:

- Nectin-4 prevalence
- Patient population size
- Target validation via antibody-drug conjugates (ADCs)

Selected Cancer indications	Incidence (US Only)	R/R Incidence	Nectin-4 Positive	Nectin-4 Med/High	Patient Population Based on R/R Incidence
Colon	158,850	36,600	87% ¹	78% ²	31,842
Bladder (urothelial)	84,530	18,765	83% ³	60% ³	15,575
Breast (TNBC)	56,334	11,550	78% ³	58% ⁵	9,009
Non-Small Cell Lung	199,586	122,500	64% ³	58% ⁶	78,400
Pancreatic	67,530	47,850	71% ³	37% ³	33,974
Head and Neck	72,680	12,000	59% ³	18% ³	7,080
Esophageal	22,530	16,290	55% ³	24% ³	8,960
Gastric	31,510	11,200	94% ⁷	60% ⁴	10,528

Incidence based on public estimates; Relapsed/refractory (R/R) or last-line patient population approximated by annual mortality; Patient population derived from Nectin-4 positive population multiplied by R/R incident population. ¹ Kobecki, Int J Mol Sci, 2023; ² Nikanjan, Can Let, 2025; ³ Challita, Can Res, 2016; ⁴ Zhang, Oncol Lett, 2018; ⁵ Zeindler, Front Med, 2019; ⁶ Takano, Mol Bio, 2009; ⁷ Muro, ESMO Open, 2025

CT-202: Nectin-4 x CD3 T cell Engaging (TCE) Bispecific Antibody



Novel design incorporating logic gating to spare Nectin-4 in normal tissue

- Because of its expression in healthy epidermal keratinocytes, sweat glands, and hair follicles, Nectin-4 targeted treatments are associated with dermatological side effects
- CT-202 uses pH dependent binding to both Nectin-4 and CD3 to minimize binding to healthy tissues and maximize binding and T cell activation within the tumor microenvironment

Avidity optimized to mitigate CRS risk

- Bivalent Nectin-4 binding to reduce T cell crosslinking in the absence of target
- Steric hindrance of CD3 binding by Fc domain prevents T cell crosslinking by single CT-202 molecules

Ease of manufacturing

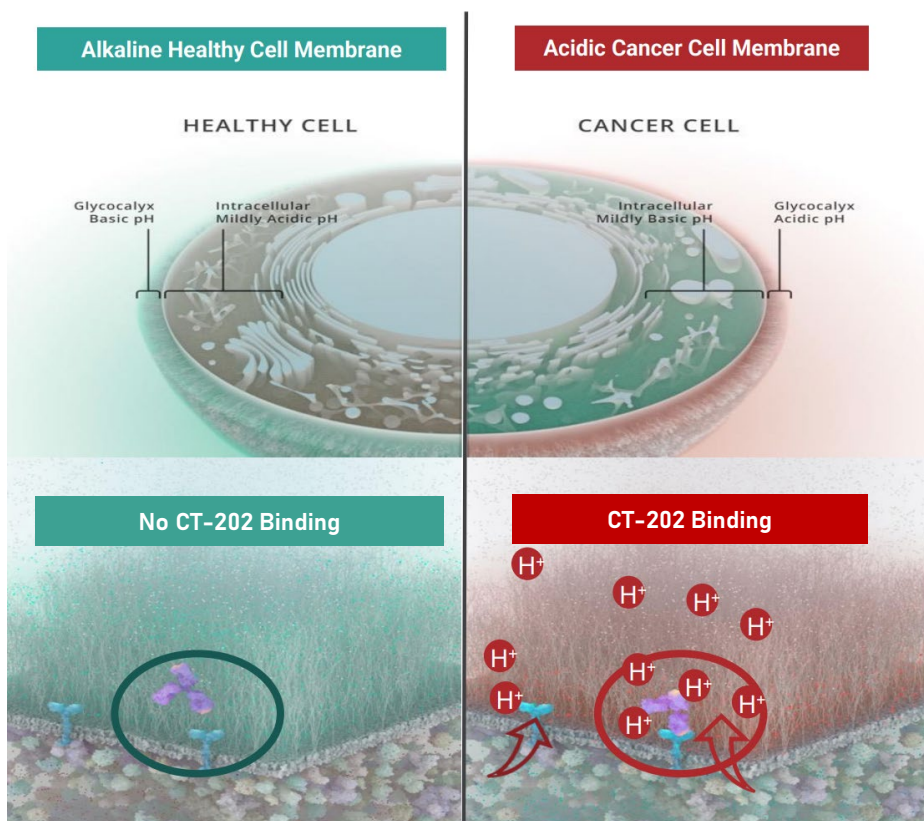
- IgG1 backbone is highly stable and enables high yield

Anticipate Phase 1 First Patient Dosed in Q3 2026

Two-Pronged Approach to Overcoming Nectin-4 Expression in Skin

Logic Gating Through pH Dependency

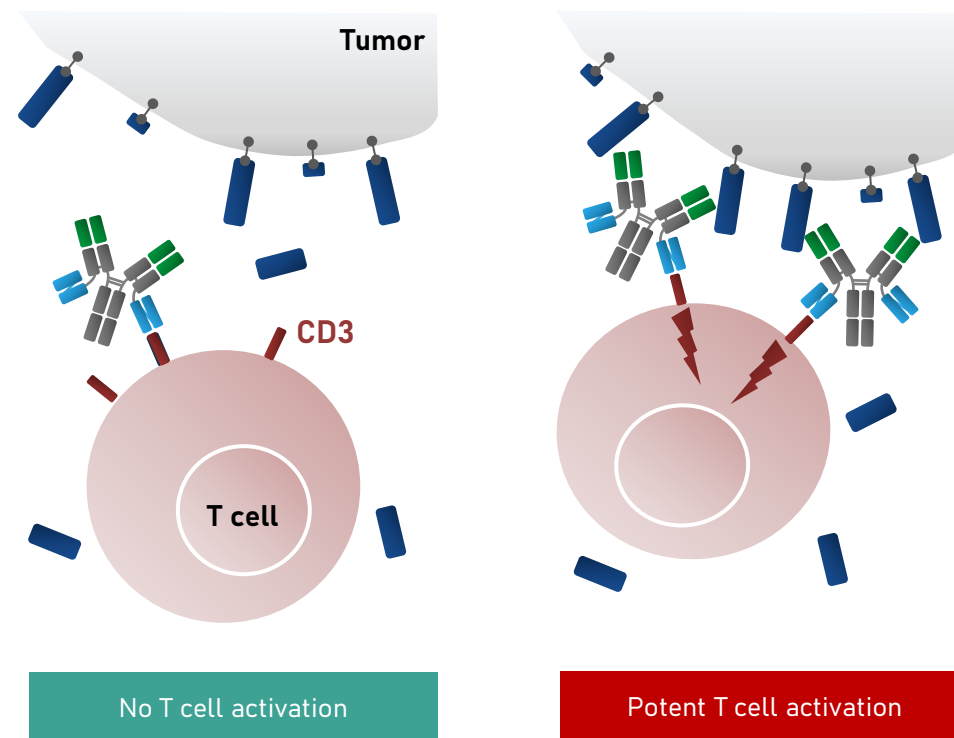
30x gain of activity in acidic tumor microenvironment versus healthy cells¹



Increased Target Selectivity Through Avidity

High on-/off-rate when bound to Nectin-4 monomer

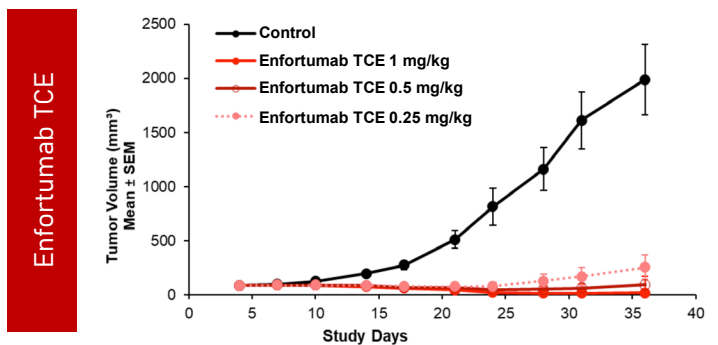
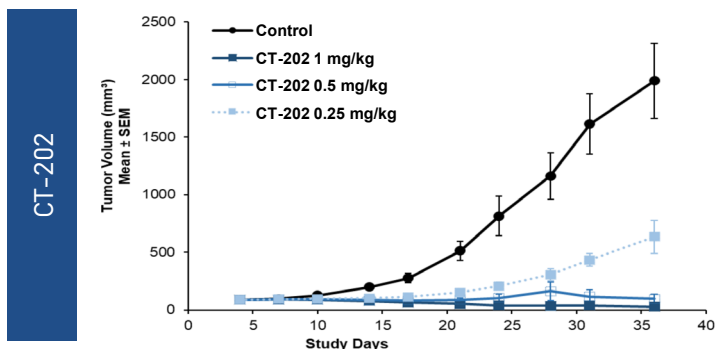
Avidity-enhanced Nectin-4 binding



CT-202 is Highly Active and Well Tolerated Across In Vivo Models

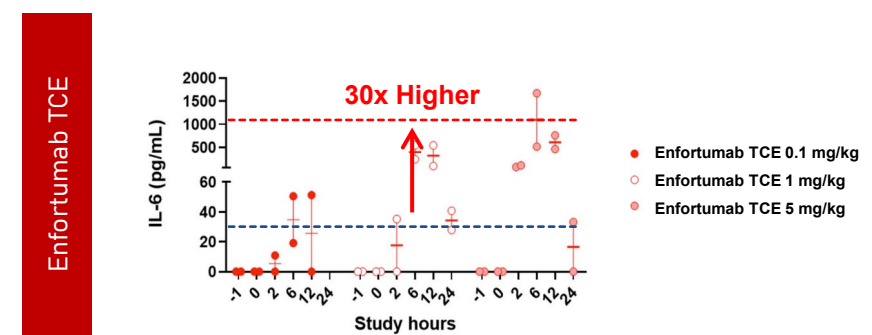
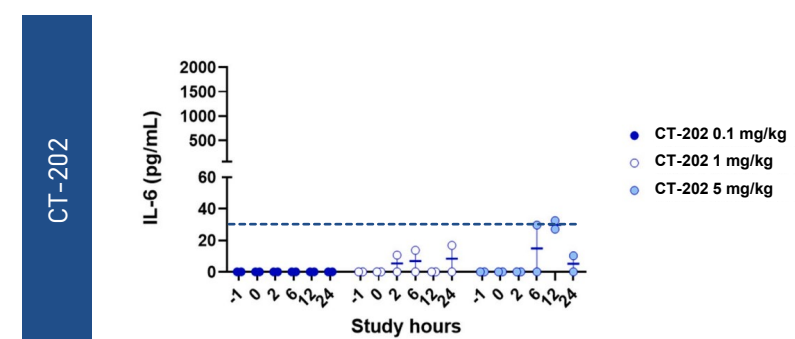
Complete Tumor Regressions

CT-202 demonstrated similar efficacy in BT474 breast cancer xenograft compared to enfortumab TCE control antibodies in mice



Reduced Cytokine Release

CT-202 treatment resulted in significantly lower IL-6 induction compared to enfortumab TCE benchmark antibodies in NHP



CT-202 Competitive Landscape

Competitor TCE programs lack conditional activation, avidity enhancement, and high potency immune activator

Company	Context Therapeutics	Bicycle Therapeutics	Rondo Therapeutics
Asset	CT-202	BT7480 ^{1,2}	RNDO-564 ³
Format	2 + 2 (pH dependent)	1 + 2 (Bicycle)	1 + 1 (Fixed light chain)
Conditionally active	✓	✗	✗
Avidity enhanced	✓	✗	✗
High Affinity CD3	✓	✗	✗
Program Status	Preclinical (Ph 1 FPI expected Q3 2026)	Phase 1 (completed)	Preclinical (Ph 1 late 2025)



Corporate

Anticipated Key Milestones in 2026-2027

- Multiple data inflection points over the next 12 months
- Current cash runway into mid-2027

PROGRAM <i>target</i>	1H 2026	2H 2026	1H 2027	2H 2027
CTIM-76 <i>Claudin 6 (CLDN6)</i>	Ph 1a Dose Escalation	Ph 1a Q3W Dose Evaluation	Ph 1b Dose Expansion	
CT-95 <i>Mesothelin (MSLN)</i>	Ph 1a Dose Escalation		Ph 1b Dose Expansion	
CT-202 <i>Nectin-4</i>			Ph 1a Dose Escalation	

Experienced Leadership Team



Martin Lehr
CEO and Director



Karen Chagin, MD
Chief Medical Officer



Jennifer Minai
Chief Financial Officer



Alex Levit, Esq
Chief Legal Officer



Jennifer Dashnau, PhD
SVP Technical Operations



Chris Beck, MBA
SVP Operations



Focus on Execution

Experienced management team

Clinical team has developed T cell therapies

Our management team is supported by a Board with deep oncology experience



Advancing T Cell Engagers for Solid Tumors

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Glossary

ADC	Antibody drug conjugate
AE	Adverse event
CAR-T	Chimeric antigen receptor T cell therapy
CD3	Cluster of differentiation 3
CLDN	Claudin
CRS	Cytokine release syndrome
DLT	Dose limiting toxicity
Fab	Fragment antigen-binding region
FIH	First-in-human
FPI	First Patient In (dosed)
FRα	Folate receptor alpha
GPI	Glycosylphosphatidylinositol
IHC	Immunohistochemistry
IND	Investigational new drug
IV	Intravenous
Mabel	Minimum anticipated biologic effect level
MoA	Mechanism of action
MSLN	Mesothelin

MTD	Maximum tolerated dose
N.D.	Not disclosed
ORR	Overall response rate
PFS	Progression free survival
PK	Pharmacokinetic
PR	Partial Response
PROC	Platinum resistant ovarian cancer
QW	Every week
Q3W	Every three weeks
scFv	Single chain variable fragment
SD	Stable Disease
TCE	T cell engager
YE	Year End