context therapeutics

Advancing T Cell Engagers for Solid Tumors

Corporate Presentation

November 2026



Forward-Looking Statements

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Pipeline Overview

PROGRAM	TARGET	LEAD INDICATION(S)	PRECLINICAL	PHASE 1	PHASE 2	ANTICIPATED MILESTONES
CTIM-76	Claudin 6	Platinum-resistant ovarian cancer (PROC)	*Fast Track Designation*			**Q1 2027:** Initiate Phase 1b **Q2 2027**: Phase 1a initial Q3W dosing data
CT-202	Nectin-4	Urothelial, colorectal, TNBC				**2H 2027**: Phase 1a initial data

Our Strategy

Target tumor antigens that are clinically validated by antibody drug conjugates (ADC) or chimeric antigen receptor T cell therapy (CAR-T)

Focus on solid tumors with high unmet need

CTIM-76: Claudin 6 x CD3

CLDN6 is overexpressed in ovarian, endometrial, lung, testicular, and other solid tumors

CTIM-76 was designed to bind selectively to CLDN6 over CLDN3, CLDN4, and CLDN9

CT-202: Nectin-4 x CD3

Nectin-4 is overexpressed in bladder, lung, breast, head and neck, and other solid tumors

CT-202 was designed to be conditionally active within the tumor microenvironment and potentially allow a wider therapeutic window while sparing effects on normal tissue

Fast Track Designation for the treatment of platinum-resistant ovarian cancer (PROC) in patients who have exhausted all standard-of-care therapies

T Cell Engaging (TCE) Bispecific Antibodies

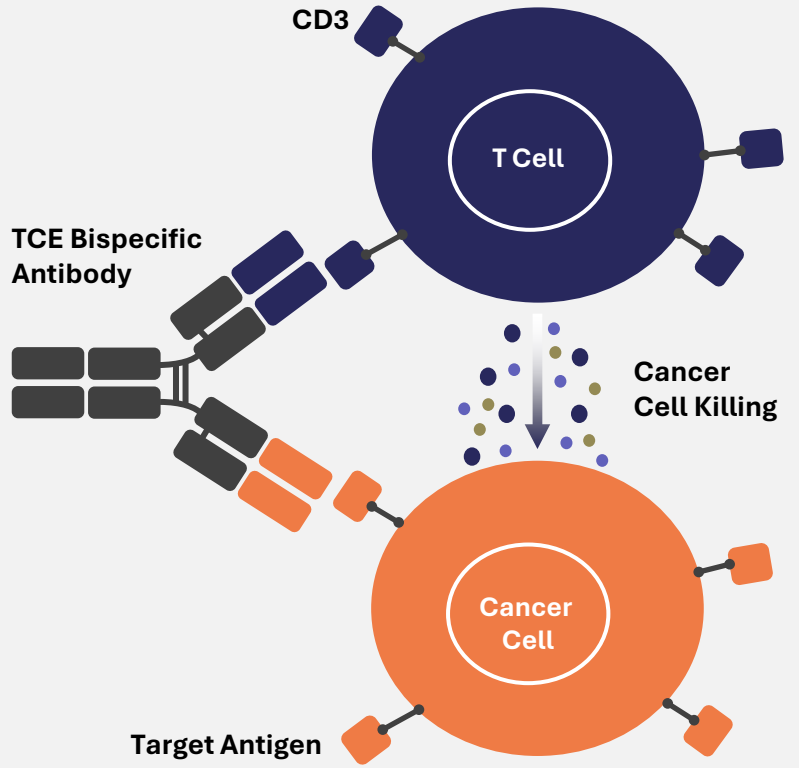
Mechanism of Action

T cell engagers are specialized medicines that help the body's own immune cells find and attack cancer.

Each one works like a bridge: one end grabs onto a marker on the cancer cell, and the other end grabs onto a T cell, one of the immune system's main fighters.

By pulling the two together, the T cell is switched on and can destroy the cancer cell.

TCE-mediated Cancer Cell Death

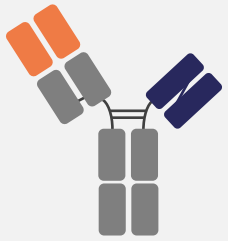


Context is Positioned to Develop the Next Generation of T Cell Engagers

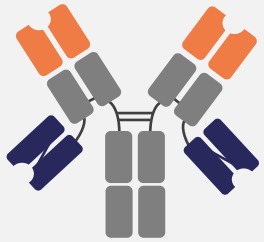
Potential to expand into early treatment lines through rational drug combinations

Near Term Development
Optimized Novel Monotherapies

CTIM-76



CT-202

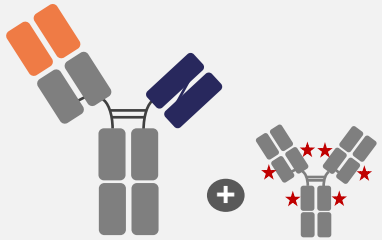


Bispecific TCE Engineered for:

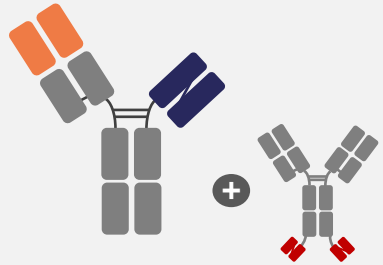
- Antitumor activity
- Reduced risk of CRS
- Target selectivity
- Clinically relevant exposure

Future Development
Combination Approaches

TCE + ADC



TCE + PD-(L)1/VEGF



Potential Opportunities for:

- Deeper and more durable responses
- Advancement into earlier lines of therapy

CTIM-76

CLDN6 x CD3 bispecific antibody



Our Initial Focus is on Platinum-resistant Ovarian Cancer (PROC)

High unmet need for new therapies that can be used after or in combination with ADCs or PD-(L)1/VEGF therapies

~75% of PROC patients are CLDN6+

No approved treatment option specifically for this biomarker selected PROC population

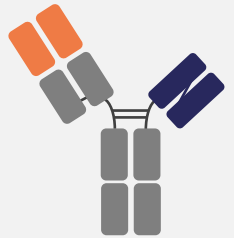
~42,000 CLDN6+ PROC patients across United States, EU7, and Japan

CLDN6 has **significant target overlap** with promising ADC targets, including B7H4, CDH6, FRα, and NaPi2b – opening the door to potential combination opportunities

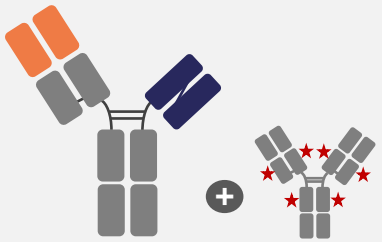
Additional opportunities in earlier lines of ovarian cancer and other tumor types with CLDN6 overexpression, including non-small cell lung (NSCLC) and endometrial cancer

Broad Use Case as Monotherapy or Combination

CTIM-76



CTIM-76 + ADC



Potential Opportunities for:

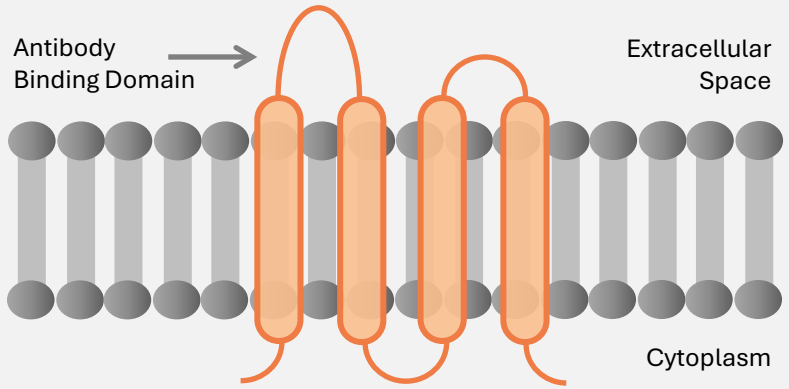
- Monotherapy development post-ADC
- Combination development with ADC, PD-(L)1/VEGF, or chemotherapy

Patient population based on multiplying NIH SEER 2026 ovarian cancer estimates in United States and prevalence estimates for EU7 and Japan by ~75% of all PROC (based on internal data)

CLDN6 is an Attractive Target for Immunotherapy

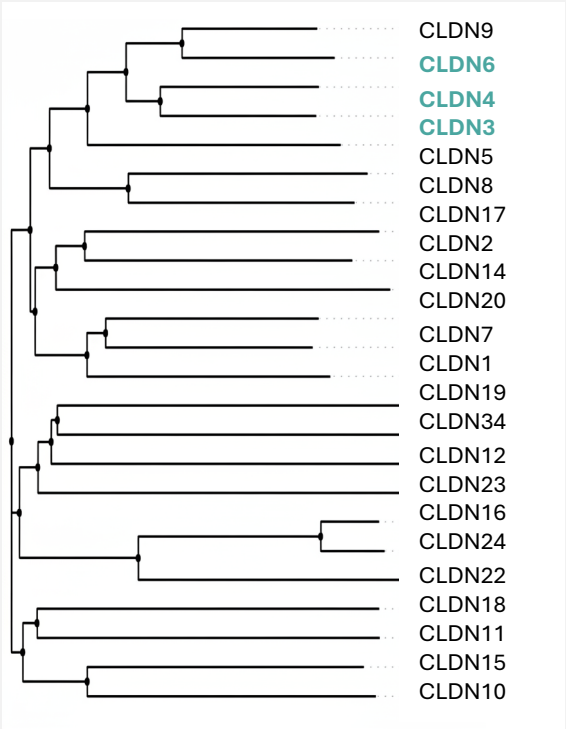
Rationale for CLDN6 Targeting

- CLDN6 is an oncofetal protein: expressed during embryonic development, silenced or very low in normal adult tissue.
- Expression increases with cancer disease stage
- Tetraspan membrane protein with low internalization, favoring a TCE over an ADC, which relies on internalization to deliver its payload



CLDN6 Protein

CLDN3/4 Cross-reactivity is a Safety Consideration



Claudin Gene Family

CLDN6 **selectivity is required** to avoid off-target liabilities

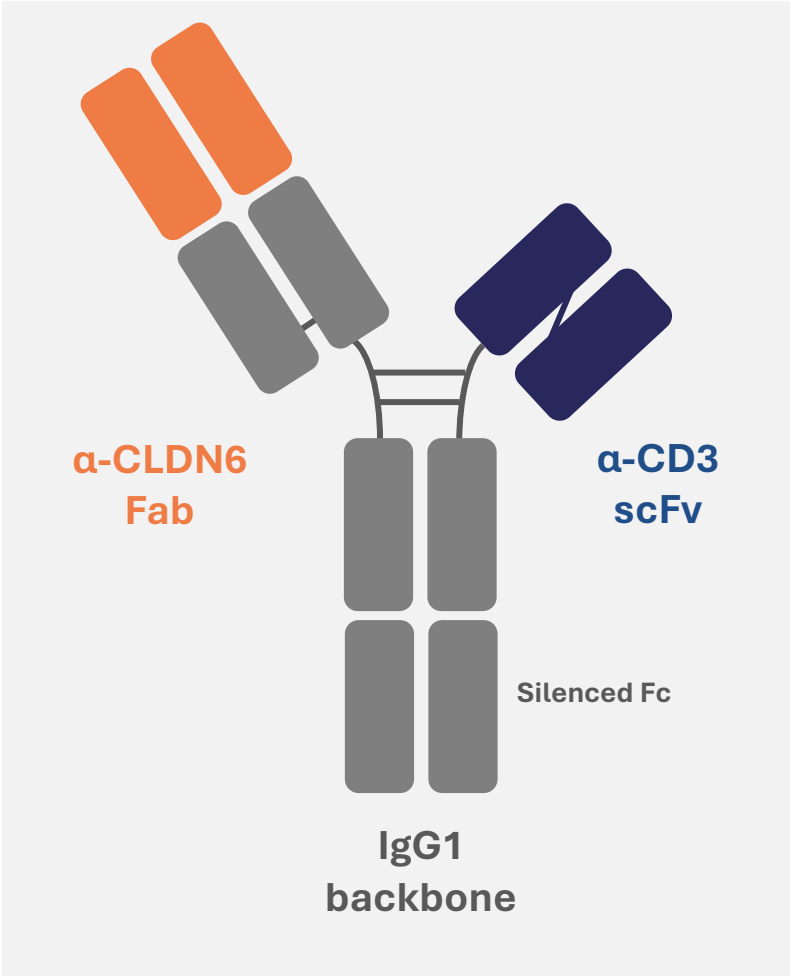
The CLDN6 antibody binding region is **highly conserved** with CLDN3 and CLDN4 – differing by only 3 amino acids[1]

CLDN3 and CLDN4 are enriched in the liver and antibody binding may result in **liver enzyme elevations**[2,3,4]

1 Screnci, Cancer Res, 2022; **2** Tanaka, J Hepatol, 2018; **3** Cordat, Physiology, 2019; **4** Li, FEBS Open Bio, 2020

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

Optimized structure for CLDN6 selectivity, potency, and manufacturability



Designed to restrict T cell activation to CLDN6-expressing target cells

- CLDN6-selective fragment antigen-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

Design features intended to improve tumor selectivity

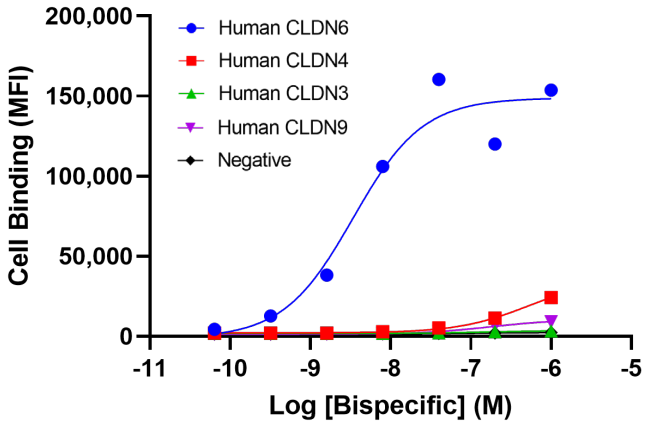
- T cell dependent cellular cytotoxicity with minimal activation of circulating cytokines

Ease of manufacturing

- IgG1 backbone is highly stable and enables high yield

CTIM-76 is a Highly Selective and Potent CLDN6 x CD3 Bispecific Antibody

Selectivity

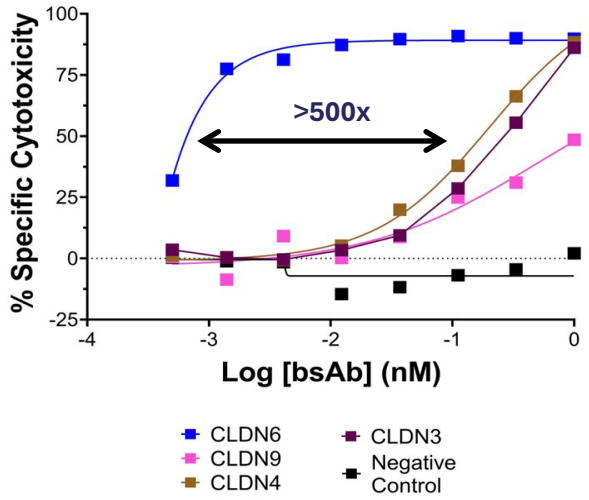


CTIM-76 CLDN6 EC50 of 3.41 nM (binding)

CTIM-76 preferentially binds to CLDN6 over CLDN3/4/9

CLDN3/4/6/9 were transiently transfected in HEK-293F cells

Potency

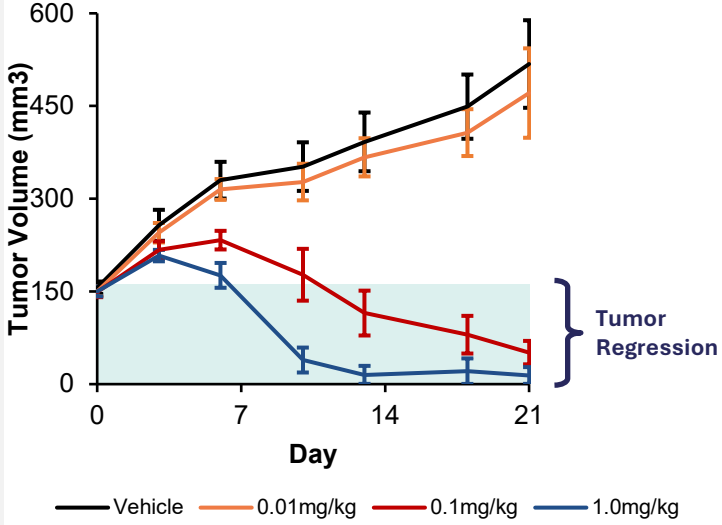


Potency assay provides a physiologically-relevant assessment for a TCE bispecific more than binding assays for off-target liabilities associated with CLDN3/4/9

CTIM-76 CLDN6 EC50 of 0.4 pM (cytotoxicity)

CTIM-76 preferentially targets CLDN6, with minimal binding and cytotoxicity against CLDN9-expressing cells

In Vivo Efficacy



CTIM-76 effectively engaged systemically administered human PBMC-engrafted mice to promote significant tumor regression and complete responses in OVCAR3 (~96,000 CLDN6 copies per cell) ovarian xenograft model

CTIM-76 was well tolerated in OVCAR3 xenograft study

NSG-b2m knockout mice (n=14/arm) engrafted with human PBMCs and bearing advanced subcutaneous OVCAR3 tumor xenografts were treated twice per week

CTIM-76 Phase 1 Study Design

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

Screening

Key Inclusion Criteria

- CLDN6-positive by IHC (≥10% 1+ staining)
- Age ≥18 years
- Measurable disease per RECIST v1.1
- ECOG 0 or 1

Trial objectives

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

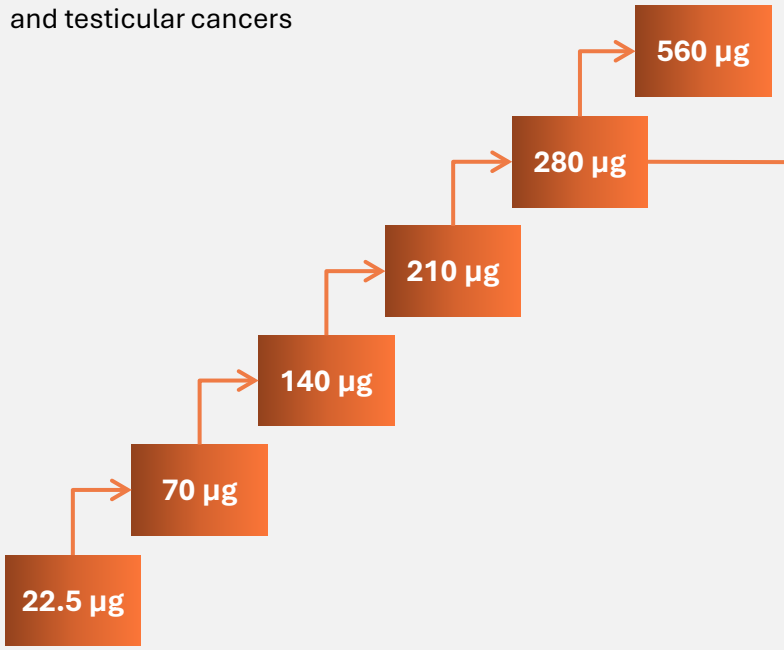
CRS Mitigation

- Step / prime dose
- Steroid prophylaxis during priming and first full dose

Phase 1a Dose Escalation (QW Dosing)

3+3 Design (Completed)

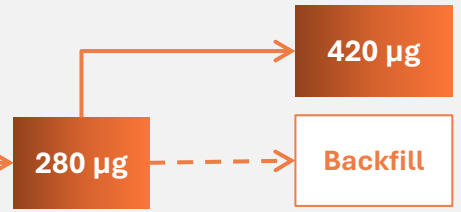
Patients with platinum resistant ovarian cancer (PROC), endometrial, and testicular cancers



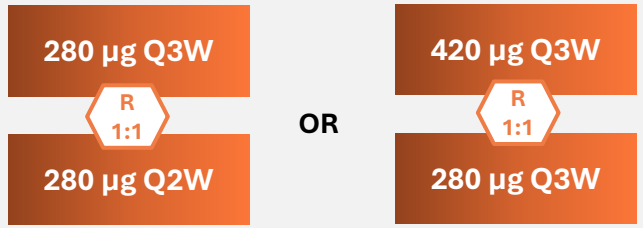
Phase 1a Dose Escalation (Q3W Dosing)

3+3 Design & Backfill (Ongoing)

Patients with PROC



Phase 1b Dose Expansion (Q1 2027)



CTIM-76-101 ClinicalTrials.gov Identifier: NCT06515613.

Phase 1a Clinical Data Observed with CTIM-76

Weekly Dosing (QW)

22.5–560 µg QW

Dose range evaluated with a single priming dose

Heavily pretreated patients with ovarian, endometrial, or testicular cancer

QW PROC Experience*

22% ORR
56% DCR
5.6-month PFS

Late-line PROC:
median 7 prior therapies (range, 5–16)

Every 3 weeks (Q3W)

Evaluating ≥280 µg Q3W

Completed:
Dose escalation: 280 µg (n=6 patients)

Currently enrolling:
Dose escalation: 420 µg (n=3–6)
Cohort backfill: 280 µg (n=6)

Ovarian cancer focus

Rationale: high prevalence of CLDN6 expression and unmet need following antibody–drug conjugate (ADC) therapy

Biologically active doses

Doses of 140 to 280 µg QW

Anti-tumor activity correlated with peak cytokine induction

Manageable tolerability with limited low-grade cytokine release syndrome (CRS)

Q3W dosing strategy

Intended to deepen responses by allowing T cells to recover between doses while keeping peak cytokine activation high

Enroll earlier-line patients, who typically have lower tumor burden, more effector T cells, and better immune fitness[1]

CTIM-76-101 ClinicalTrials.gov Identifier: NCT06515613. *RECIST-evaluable PROC population (n=9 patients at target doses) is defined as having measurable disease documented at baseline according to RECIST v1.1 criteria and at least one follow-up tumor assessment after a full cycle of treatment. Disease Control Rate: Patients achieving confirmed stable disease, partial response, or complete response. 1 2026 ESMO DeLLphi-305 Data as of October 30, 2026.

CTIM-76 Patient Demographics

CTIM-76 evaluated in heavily pretreated patients with high tumor burden

Baseline Characteristics	All Patients
Dose Levels	22.5–560 µg
N	27
Age, median (range)	63 (29-76)
ECOG, median (range)	0 (0-1)
Ovarian, n (%)	20 (74.1)
Testicular, n (%)	4 (14.8)
Endometrial, n (%)	3 (11.1)
Prior therapies, median (range)	6 (3-16)
1, n (%)	0 (0)
2	0 (0)
3	6 (22.2)
4	2 (7.4)
≥5	19 (70.4)

Baseline Characteristics	Ovarian (QW)	Ovarian (Q3W)
Dose Levels	140–280 µg	280 µg
N	9	6
Age, median (range)	63 (53-72)	67 (43-76)
ECOG, median (range)	1 (0-1)	0 (0-1)
Sum of longest dimension (mm)	71 (39-114)	155 (28-205)
Liver metastases, n (%)	4 (44)	4 (67)
H-Score, median (range)	135 (25-300)	95 (20-150)
Prior therapies, median (range)	7 (5-16)	6 (3-6)
Checkpoint Inhibitor, n (%)	5 (56)	2 (33)
ADC	8 (89)	4 (67)
≥ 2 ADC	3 (33)	2 (33)
Topotecan	4 (44)	0 (0)
DNA Repair: Chk1, PARP, Wee1	7 (78)	4 (67)
VEGF or PD-1xVEGF	9 (100)	5 (83)

CTIM-76-101 ClinicalTrials.gov Identifier: NCT06515613. Data as of October 30, 2026. All treated patients (≥1 dose). Age and ECOG at screening; prior therapies = number of prior anticancer regimens; Ovarian includes fallopian tube (n=1) and primary peritoneal (n=1). Sum of longest dimension = screening target lesions; liver metastases = liver target/non-target lesion at screening. H-score = CLDN6 IHC, latest biopsy.

CTIM-76 Safety Summary

In biologically active dose cohorts, events were asymptomatic, transient and largely priming-related

			Biologically Active Dose Cohorts (n=19)					
Dose Level	**22.5 µg**	**70 µg**	**140 µg**	**210 µg**	**280 µg**	**280 µg**	**560 µg**	**Total**
Schedule	**QW**	**QW**	**QW**	**QW**	**QW**	**Q3W**	**QW**	
Patients	**N=1**	**N=3**	**N=4**	**N=5**	**N=4**	**N=6**	**N=4**	**N=27**
Events	**n (%)**	**n (%)**	**n (%)**	**n (%)**	**n (%)**	**n (%)**	**n (%)**	**n (%)**
TEAE	1 (100.0)	3 (100.0)	4 (100.0)	5 (100.0)	4 (100.0)	5 (83.3)	4 (100.0)	**26 (96.3)**
TEAEs Related to CTIM-76	1 (100.0)	2 (66.7)	4 (100.0)	5 (100.0)	4 (100.0)	5 (83.3)	4 (100.0)	**25 (92.6)**
Serious TEAE	1 (100.0)	1 (33.3)	3 (75.0)	1 (20.0)	2 (50.0)	3 (50.0)	3 (75.0)	**14 (51.9)**
Serious TEAEs Related to CTIM-76	0 (0.0)	0 (0.0)	1 (25.0)	0 (0.0)	1 (25.0)	2 (33.3)	3 (75.0)	**7 (25.9)**
TEAE Leading to Dose Delay	0 (0.0)	0 (0.0)	2 (50.0)	3 (60.0)	2 (50.0)	1 (16.7)	4 (100.0)	**12 (44.4)**
TEAE Leading to Dose Reduction	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (25.0)	**1 (3.7)**
TEAE Leading to Discontinuation	1 (100.0)	1 (33.3)	0 (0.0)	0 (0.0)	0 (0.0)	1 (16.7)	1 (25.0)	**4 (14.8)**
Related TEAE Leading to Discontinuation	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (16.7)	1 (25.0)	**2 (7.4)**
TEAEs of Special Interest	0 (0.0)	0 (0.0)	0 (0.0)	1 (20.0)	1 (25.0)	1 (16.7)	2 (50.0)	**5 (18.5)**
CRS	0 (0.0)	0 (0.0)	0 (0.0)	1 (20.0)	1 (25.0)	1 (16.7)	2 (50.0)	**5 (18.5)**
ICANS	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	**0 (0.0)**

CTIM-76-101 ClinicalTrials.gov Identifier: NCT06515613. Data as of October 30, 2026. Each patient is counted once per row. TEAE = treatment-emergent adverse event, meaning a side effect or health problem that started on or after the first day of treatment; related = related or possibly related to the study drug; dose delay / discontinuation = the patient's record shows the drug was delayed or paused ("Drug Delayed/Interrupted") or stopped ("Drug Withdrawn"); dose reduction = a lower dose was recorded on the dosing form. Biologically active dose cohorts = tumor reduction and T-cell cytokine induction with manageable safety.

Grade ≥ 3 Adverse Events by Dose Cohort

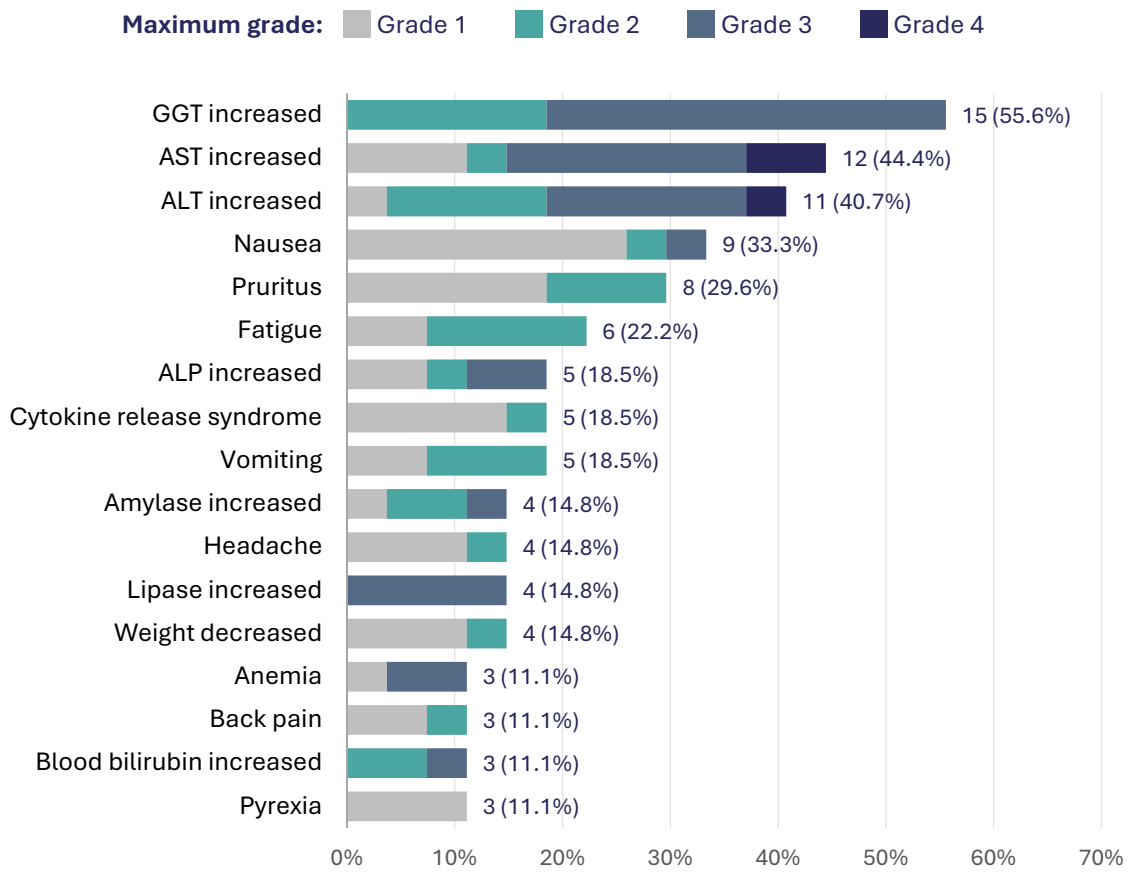
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			Biologically Active Dose Cohorts (n=19)					
Dose Level	**22.5 µg**	**70 µg**	**140 µg**	**210 µg**	**280 µg**	**280 µg**	**560 µg**	**Total**
Schedule	**QW**	**QW**	**QW**	**QW**	**QW**	**Q3W**	**QW**	
Patients	**N=1**	**N=3**	**N=4**	**N=5**	**N=4**	**N=6**	**N=4**	**N=27**
Events	**n (%)**	**n (%)**	**n (%)**	**n (%)**	**n (%)**	**n (%)**	**n (%)**	**n (%)**
Any Adverse Event	1 (100.0)	1 (33.3)	4 (100.0)	5 (100.0)	3 (75.0)	4 (66.7)	4 (100.0)	**22 (81.5)**
GGT increased	0 (0.0)	0 (0.0)	1 (25.0)	2 (40.0)	1 (25.0)	3 (50.0)	3 (75.0)	**10 (37.0)**
AST increased	0 (0.0)	0 (0.0)	0 (0.0)	1 (20.0)	2 (50.0)	1 (16.7)	4 (100.0)	**8 (29.6)**
ALT increased	0 (0.0)	0 (0.0)	0 (0.0)	1 (20.0)	2 (50.0)	1 (16.7)	2 (50.0)	**6 (22.2)**
Lipase increased	0 (0.0)	0 (0.0)	0 (0.0)	2 (40.0)	0 (0.0)	3 (50.0)	0 (0.0)	**5 (18.5)**
Anemia	0 (0.0)	1 (33.3)	1 (25.0)	0 (0.0)	1 (25.0)	0 (0.0)	1 (25.0)	**4 (14.8)**
ALP increased	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (16.7)	1 (25.0)	**2 (7.4)**
Amylase increased	0 (0.0)	0 (0.0)	0 (0.0)	1 (20.0)	0 (0.0)	1 (16.7)	0 (0.0)	**2 (7.4)**
Fatigue	1 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (16.7)	0 (0.0)	**2 (7.4)**
Pleural effusion	0 (0.0)	0 (0.0)	1 (25.0)	1 (20.0)	0 (0.0)	0 (0.0)	0 (0.0)	**2 (7.4)**
Syncope	0 (0.0)	0 (0.0)	1 (25.0)	0 (0.0)	1 (25.0)	0 (0.0)	0 (0.0)	**2 (7.4)**

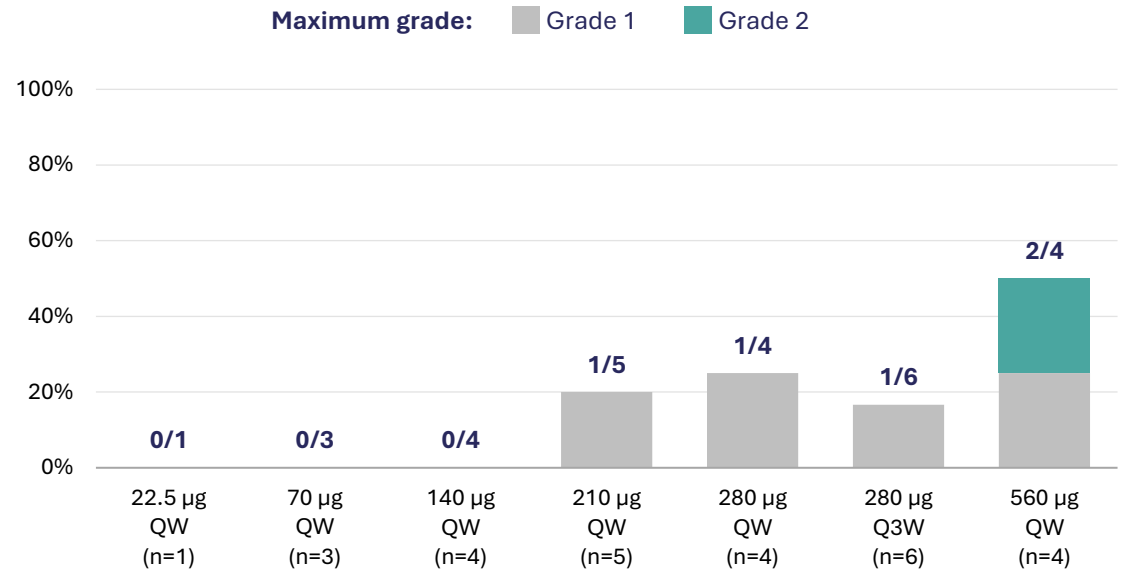
CTIM-76-101 ClinicalTrials.gov Identifier: NCT06515613. Data as of October 30, 2026. Grade ≥ 3 TEAEs by MedDRA preferred term, patients counted once per term.

CTIM-76 Demonstrates a Manageable Safety Profile, With CRS Limited to Grade 1–2

CTIM-76-related TEAEs in ≥ 10% of patients



Cytokine release syndrome by dose level



CRS in 5 of 27 patients (18.5%)

- Grade 2 CRS in 1 patient at 560 µg QW; all other CRS grade 1
- CRS onset predominantly associated with first prime dose

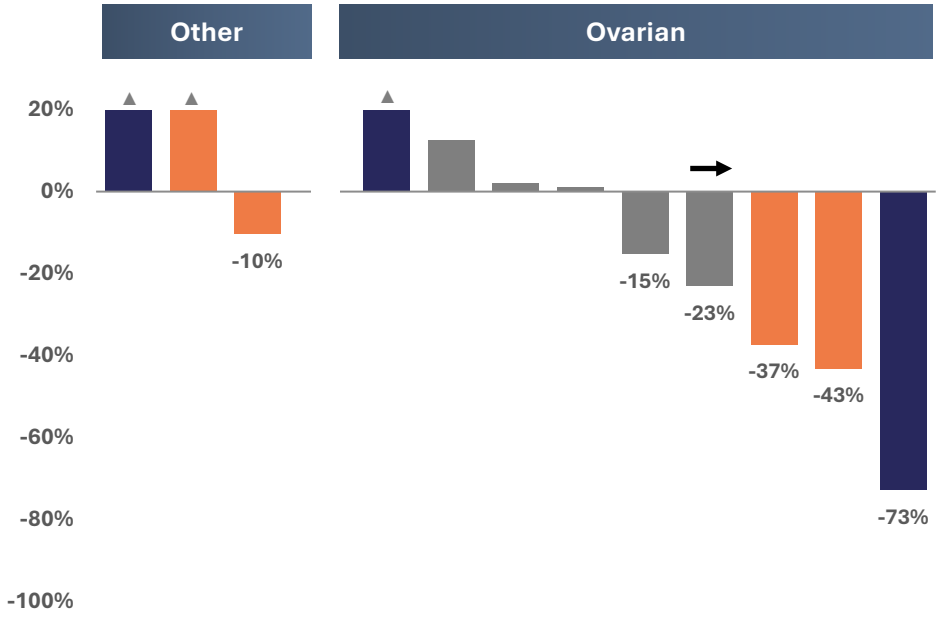
CTIM-76-101 ClinicalTrials.gov Identifier: NCT06515613. TEAE = adverse event starting on or after the first CTIM-76 dose; related = related or possibly related to CTIM-76 (investigator). Patients counted once per term at their maximum grade; terms reported in ≥ 10% of all treated patients shown, % of n=27. Dose level = enrolled cohort (target dose; Q3W = Part 1a-B); CRS = MedDRA PT cytokine release syndrome, grade on the AE form. Data as of October 30, 2026.

Promising Antitumor Activity in QW Biologically Active Dose Cohorts

Early efficacy signal in a heavily pretreated population supports advancing the QW biologically active doses

Efficacy

Best Overall % Change in Tumor Size From Baseline*



Tumor size reduction in 5 of 9 heavily pretreated PROC patients with QW dosing, including 2 confirmed partial responses (−73%, −43%)

Step Dose Cohort

- 70/140 µg QW
- 70/210 µg QW
- 140/280 µg QW

→ On treatment

Other Endometrial or testicular cancer

Safety

Cytokine Release Syndrome (CRS) Grade

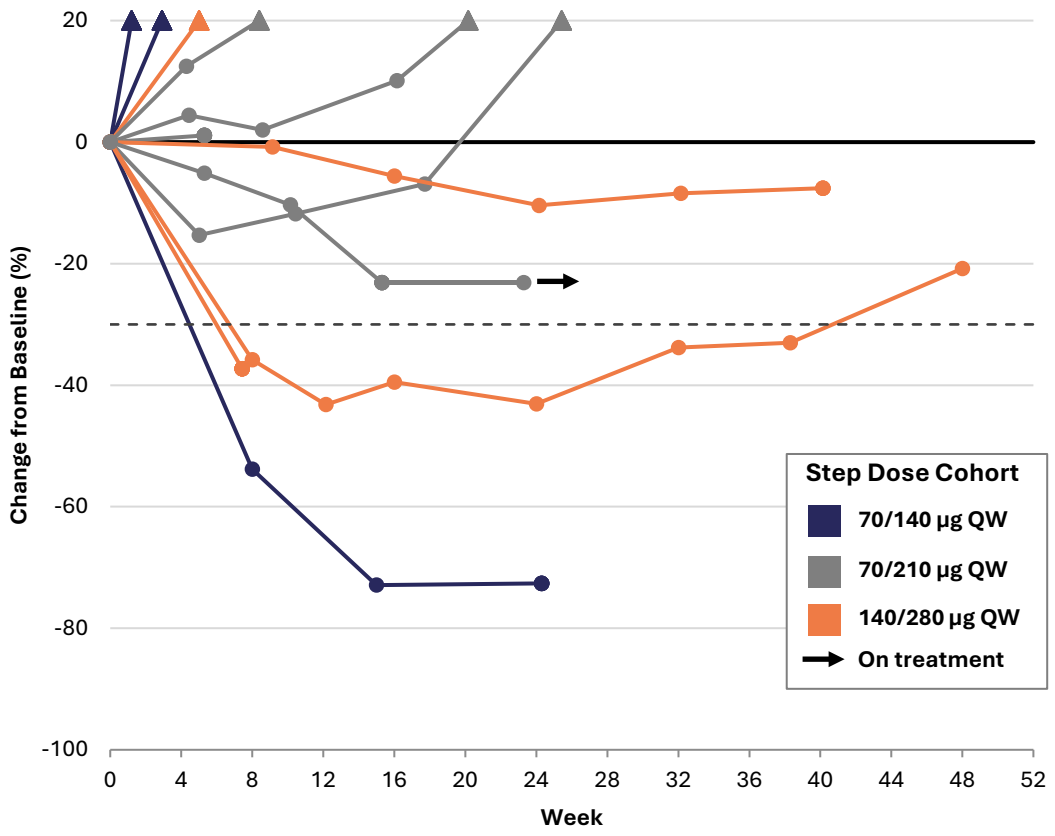


Dose-proportional cytokine release (Slide 23) with no severe CRS supports a manageable tolerability profile

CTIM-76-101 ClinicalTrials.gov Identifier: NCT06515613. Data as of October 30, 2026. RECIST-evaluable population (n=12 patients at biologically active doses, QW) is defined as having measurable disease documented at baseline according to RECIST v1.1 criteria and at least one follow-up tumor assessment after a full cycle of treatment. *Best tumor reduction at any time point in evaluable patients. Bars truncated at +20%. ▲ = best change >+20%. CRS = maximum grade on the AE form.

Emerging Evidence of Durable Activity in QW Biologically Active Dose Cohorts

Biologically Active Dose Range, Percent Change from Baseline (n=12)



	All Comers	All Ovarian	Ovarian QW Dosing		
			140 µg	210 µg	280 µg
Patient enrolled, n	13	9	2	5	2
RECIST evaluable, n	12	9	2	5	2
Not RECIST evaluable, n	1	0	0	0	0
ORR, n (%)	2 (17)	2 (22)	1 (50)	0 (0)	1 (50)
SD, n (%)	4 (33)	3 (33)	0 (0)	3 (60)	0 (0)
DCR, n (%)	6 (50)	5 (56)	1 (50)	3 (60)	1 (50)

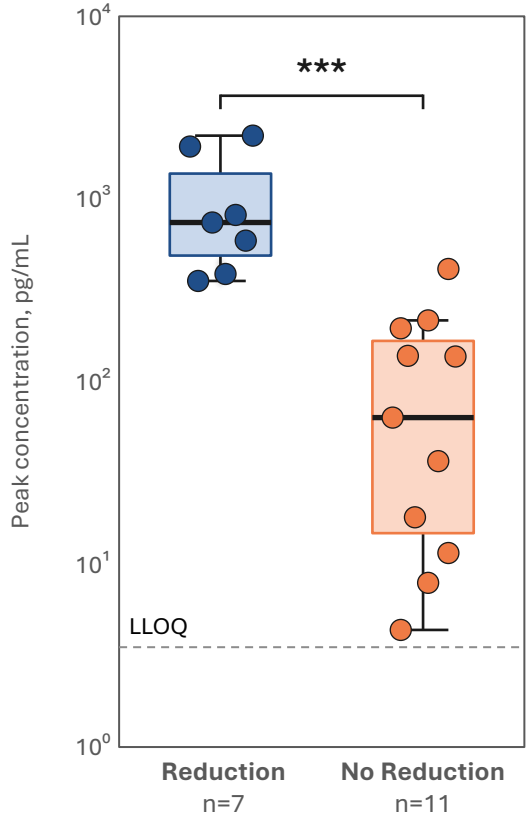
CTIM-76-101 ClinicalTrials.gov Identifier: NCT06515613. Data as of October 30, 2026. RECIST-evaluable population (n=12 patients at target doses, QW) is defined as having measurable disease documented at baseline according to RECIST v1.1 criteria and at least one follow-up tumor assessment after a full cycle of treatment. ORR = confirmed PR. Disease Control Rate: patients achieving confirmed stable disease, partial response, or complete response. Spider: % change in sum of target-lesion diameters at each complete re-assessment, by week from C1D1. Y axis shown from +20% to −100%; lines rising above +20% are truncated at the axis edge (▲).

Greater T-Cell Activation With Tumor Reduction, Consistent With Target-Dependent Activity

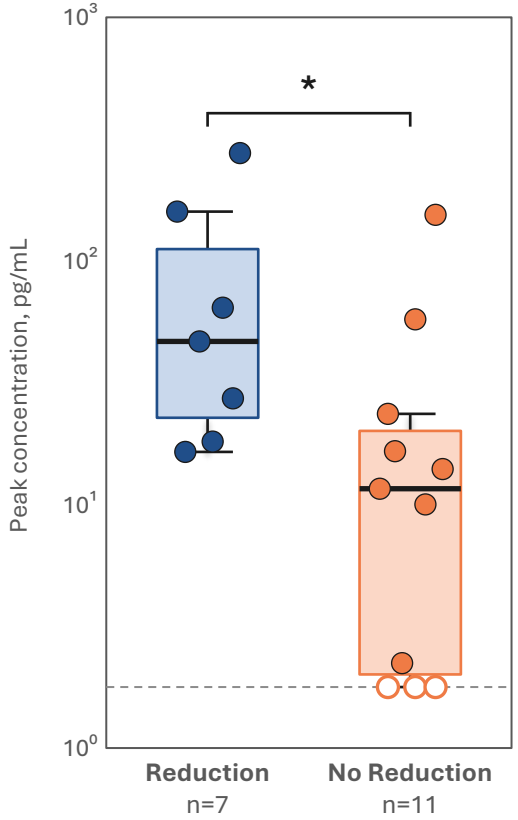
Tumor reduction associated with higher IFN-γ, IL-2 and CD69+ T cell responses

Peak serum cytokines

IFN-γ

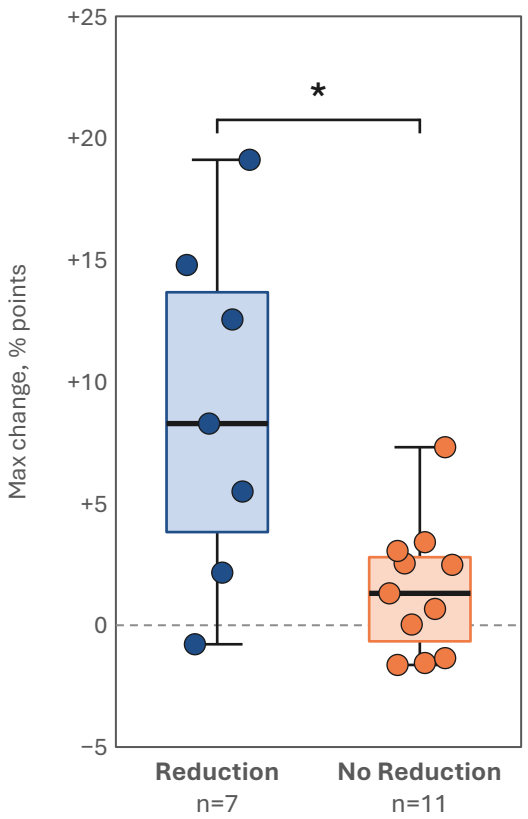


IL-2



Activated CD8 T cells

CD69+



Greater T cell activation in patients with tumor reduction

- Higher median IFN-γ and IL-2 peaks in patients with best target-lesion change
- Larger maximal increase from baseline in CD69+ frequency among CD8 T cells

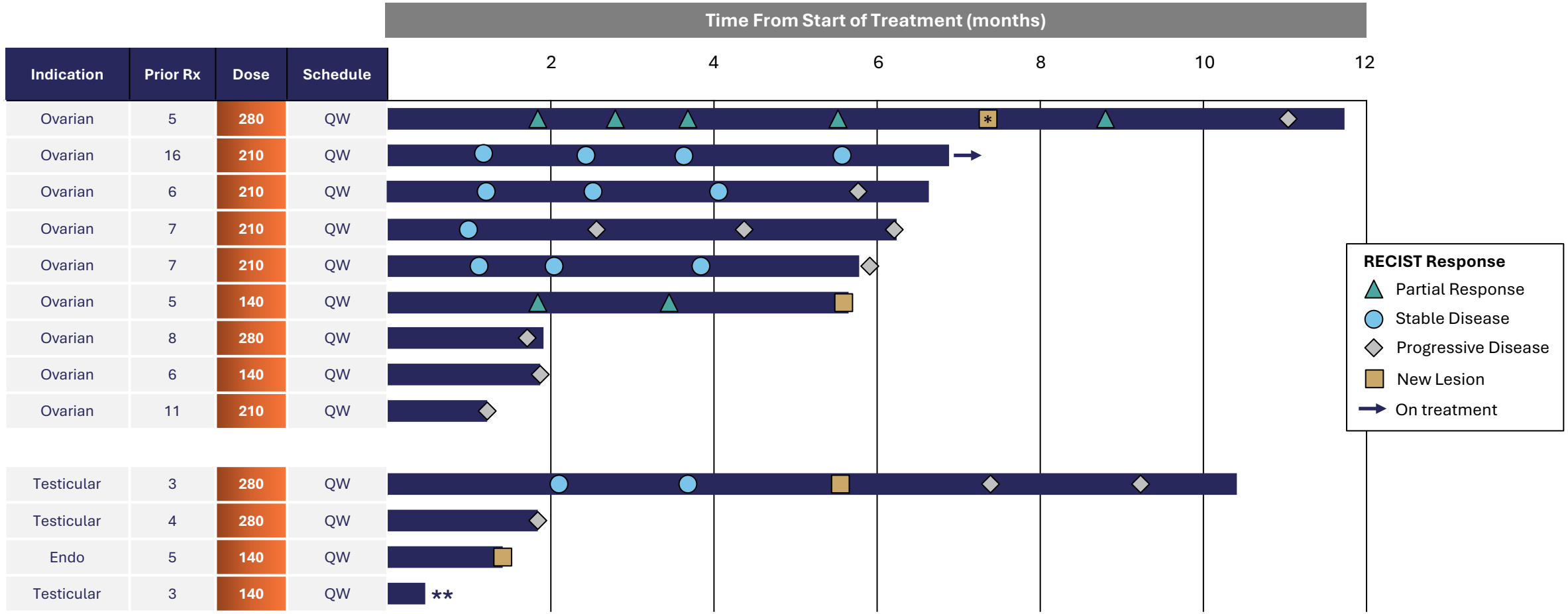
Support for mechanism of action

- Biomarker evidence of T cell activation correlated with tumor reduction
- Supports the proposed mechanism of CLDN6-directed T cell engagement

CTIM-76-101 ClinicalTrials.gov Identifier: NCT06515613. Exploratory analysis as of October 30, 2026. Patients in QW cohorts with ≥1 sample of that biomarker ≥7 days after the first target dose at any visit. Mann–Whitney, unadjusted; whiskers 1.5× IQR. Cytokines: peak serum level. Open dot = below LLOQ. CD69+: highest change from C1D1 pre-dose, % of CD8 T cells. Reduction = best target-lesion change <0. Box = IQR · line = median · *p<0.05 ***p<0.001.

Duration of Treatment and Response in QW Biologically Active Dose Cohorts

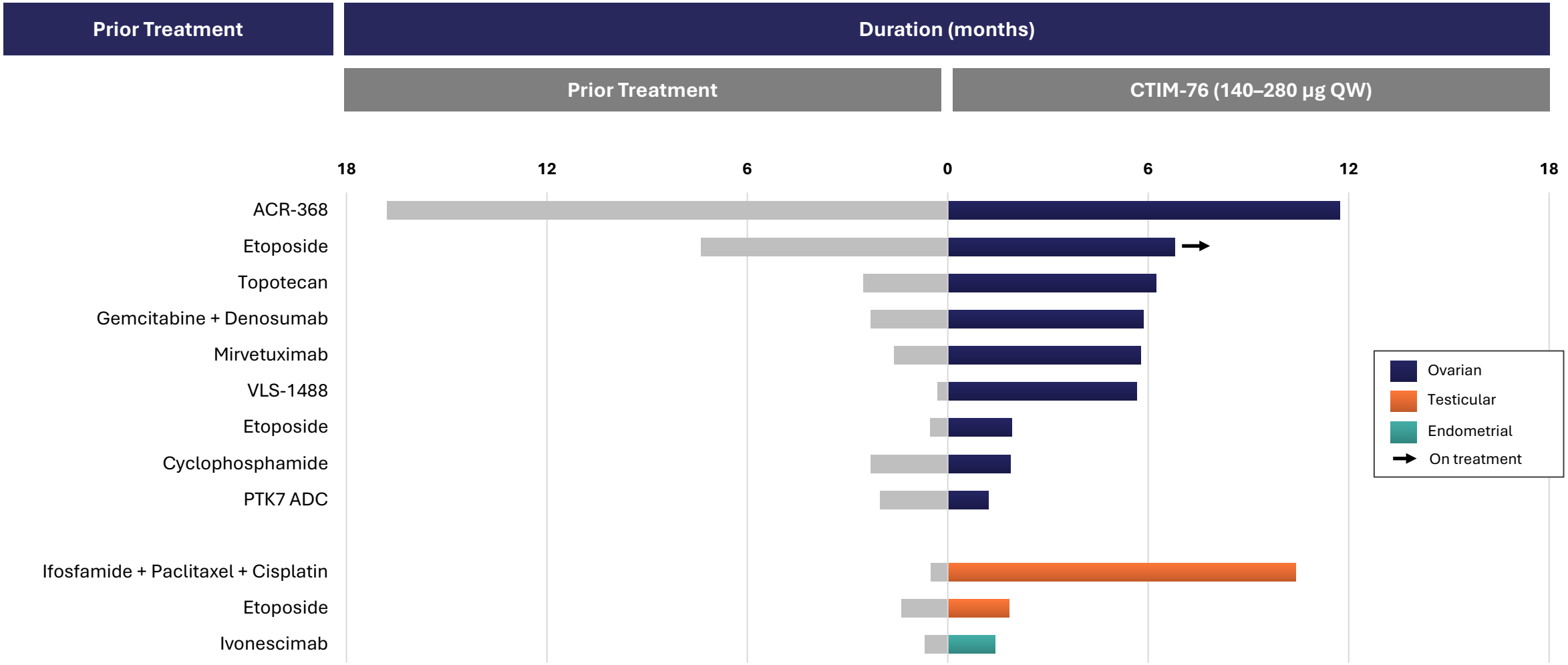
5.6 month median PFS (95% CI: 1.2-7.4 months) in late-line ovarian cancer



CTIM-76-101 ClinicalTrials.gov Identifier: NCT06515613. Data as of October 30, 2026. Swimmer plot includes all comers at Biologically Active Dose levels with QW dosing (n=13). Bar = C1D1 to end of treatment (ongoing: last recorded visit). Median PFS: Kaplan–Meier, ovarian QW (n=9); event = first RECIST 1.1 progression (incl. new lesion), censored at last assessment. * New lesion not found on subsequent scans. **Withdrew consent due to clinical progression before completing the DLT period.

Treatment Duration With CTIM-76 vs Prior Therapy

Time on CTIM-76 exceeded the most recent prior regimen in the majority of patients



CTIM-76-101 ClinicalTrials.gov Identifier: NCT06515613. Data as of October 30, 2026. RECIST-evaluable patients at biologically active doses, QW (n=12); Prior Rx = most recent regimen before C1D1 (latest end date), start to end of regimen; CTIM-76 = C1D1 to end of treatment (ongoing: last visit).

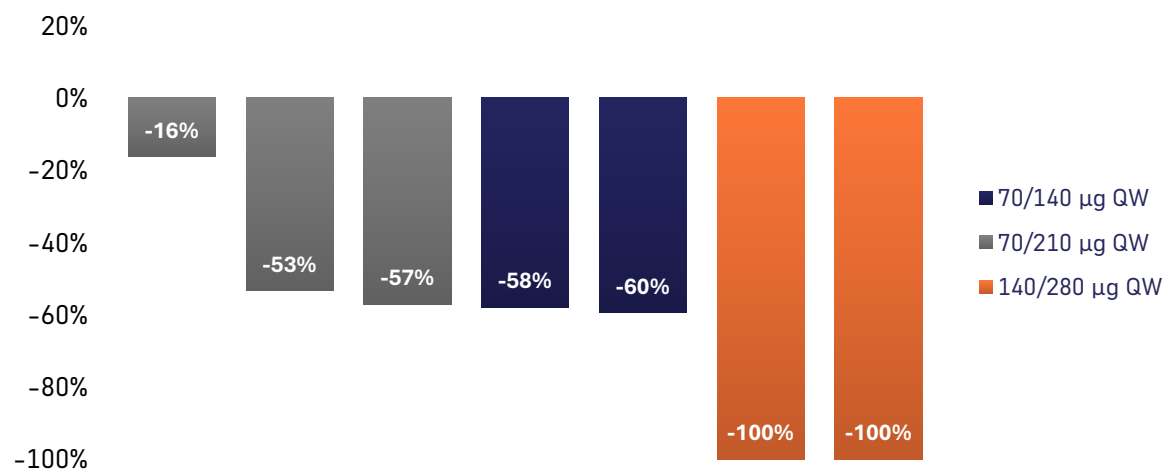
Decrease in Tumor Size Across Diverse Metastatic Sites, With Strong Responses in Liver

Tumor size reduction by Location
Target lesions with any decrease

Liver	7 of 7 lesions
Peritoneum / mesentery	5 of 14 lesions
Lymph node	3 of 5 lesions
Lung / pleura	1 of 2 lesions
Vaginal cuff	0 of 2 lesions

Liver Target Lesion Size
Best % Change from Baseline Per Target Lesion

7 of 7 liver target lesions decreased across all 4 patients with liver mets (6 of 7 by ≥50%)



Liver metastases are common and hard to treat with immunotherapy
The liver is among the most frequent metastatic sites in solid tumors[1] and the most common distant site in ovarian cancer[2], and liver metastases predict shorter PFS and OS on checkpoint inhibitors[3]

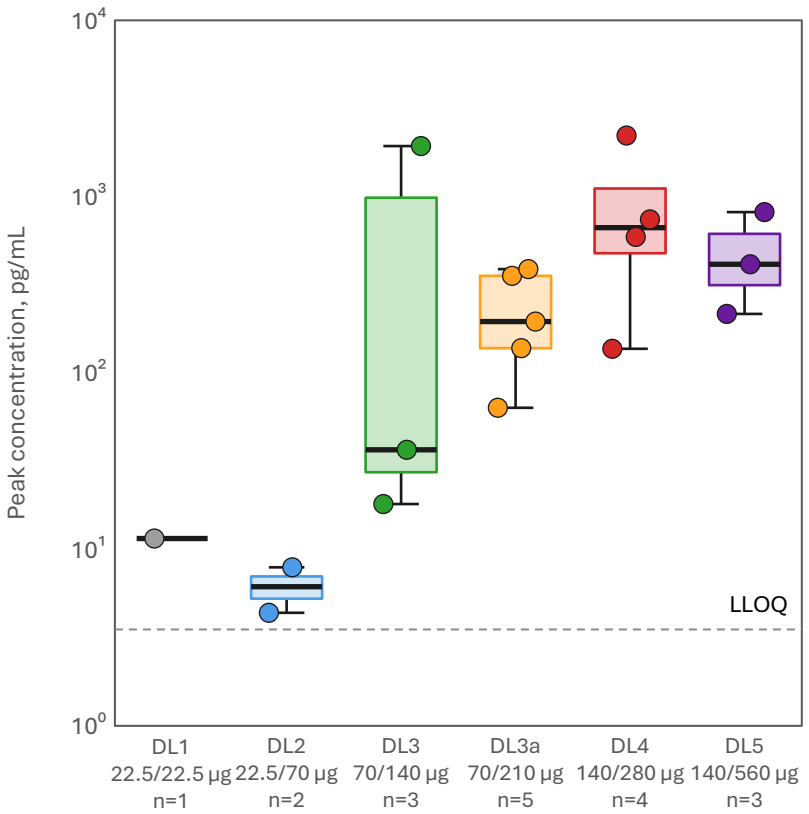
CTIM-76-101 ClinicalTrials.gov Identifier: NCT06515613. Data as of October 30, 2026. All target lesions (n=30) in RECIST-evaluable ovarian patients at biologically active target doses, Best % change per lesion = lowest post-baseline measurement vs screening (lymph nodes: short axis); Reduction = any decrease. Lesions too small to measure were generally recorded as 5 mm; −100% = lesion no longer measurable (0 mm). Lesion-level changes are exploratory and are not RECIST overall response. Refs: 1. Hess KR, et al. Cancer. 2006;106:1624-1633. 2. Zhao H, et al. Front Oncol. 2020;10:571671. 3. Tian BW, et al. Clin Exp Metastasis. 2023;40:255-287.

Cytokine Response Helps Inform Dose and Schedule Selection

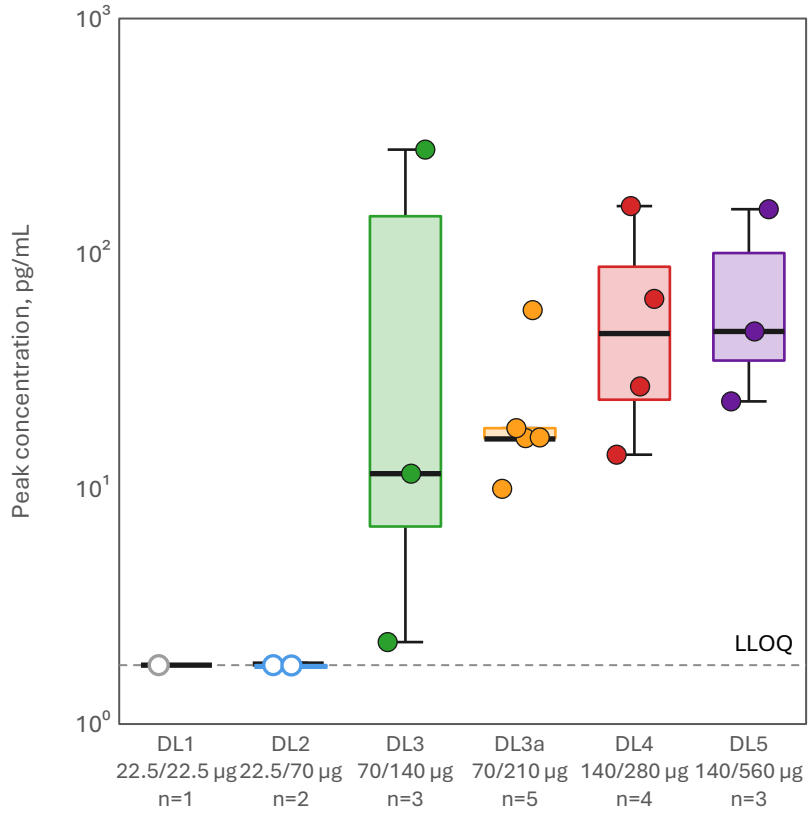
Dose-dependent cytokine release consistent with T cell activation

Peak serum cytokine by Dose Level

IFN-γ



IL-2



Response Rises With Dose

- Cytokine release starts at 140 µg given weekly (QW)
- Median IFN-γ and IL-2 peaks are highest at ≥280 µg QW

Response Levels Off

- No further increase in cytokine response is observed at 560 µg QW
- This may reflect a QW schedule-dependent ceiling

Actively exploring ≥280 µg with every 3 Weeks (Q3W) dosing

- Longer dosing interval may allow greater T-cell recovery
- Higher doses may help maintain effective exposure between doses
- Q3W therefore provides an opportunity to explore greater and/or more sustained antitumor activity

CTIM-76-101 ClinicalTrials.gov Identifier: NCT06515613. Exploratory analysis as of October 30, 2026. Peak = highest serum value from first dose through Day 37, measured ≥7 days after the first target dose. Whiskers 1.5× IQR. Box = IQR · line = median · open symbol = below LLOQ.

Cross-Trial Comparison of CTIM-76 and Other TCEs in PROC

Encouraging tumor response, durability, and CRS rate

Company	Context Therapeutics	Xencor	Regeneron	Immunocore
Asset	CTIM-76	XmAb541	Ubamatamab	Brenetafusp
Target	CLDN6	CLDN6	MUC16	PRAME
Prior ADC (%)	**89**	n.d.	n.d.	11
Prior Lines, median	**7**	n.d.	4.5	5
PFS, median (months)	**5.6**	n.d.	3.0	3.3
ORR (%)	**22**	14	14	6
Reference	30 October 2026 Data Cut	Q2 2026 Earnings Release	ESMO 2022	ESMO 2024

Data are not from head-to-head studies. 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. CTIM-76-101 ClinicalTrials.gov Identifier: NCT06515613. Data as of October 30, 2026.

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 Relapsed/Refractory (R/R) Setting

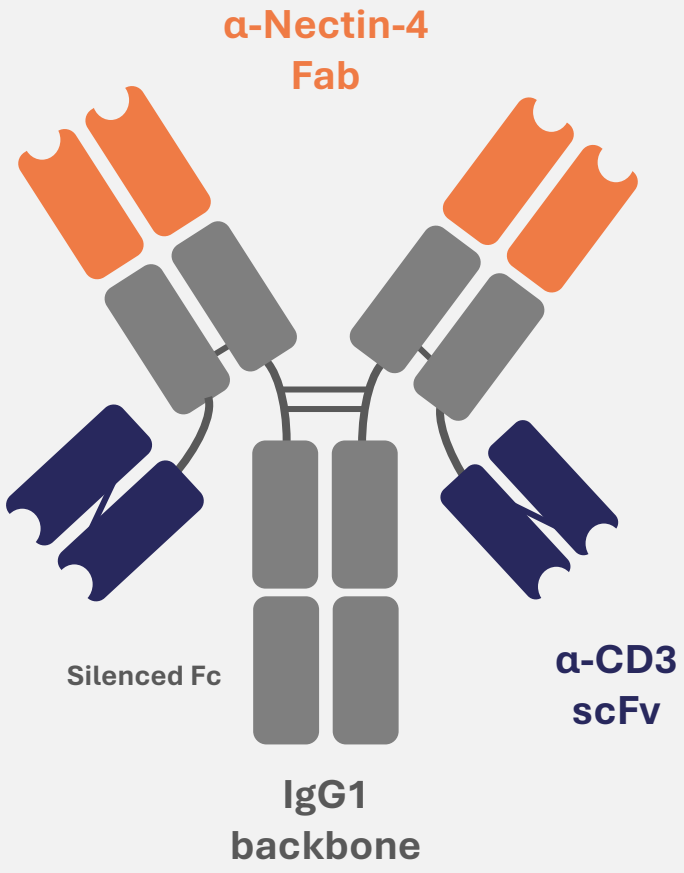
Initial indications of interest based on:

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

Cancers	Incidence (US Only)	R/R Incidence	Nectin-4 Positive	Nectin-4 Med/High	Patient Population Nectin-4-positive R/R Incidence
Colon	158,850	36,600	39%[1]	9%[1]	**14,274**
Bladder (urothelial)	84,530	18,765	60%[2]	36%[2]	**11,259**
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
Gastric	31,510	11,200	94%[7]	60%[4]	10,528
Esophageal	22,530	16,290	55%[3]	24%[3]	8,960
Head and Neck	72,680	12,000	59%[3]	18%[3]	7,080
Cervical	12,960	4,500	89%[8]	67%[8]	4,005

Incidences 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 Context Internal Data; **2** Klumper, Clin Cancer Res, 2023; **3** Challita, Cancer Res, 2016; **4** Zhang, Oncol Lett, 2018; **5** Zeindler, Front Med, 2019; **6** Takano, Mol Bio, 2009; **7** Muro, ESMO Open, 2025; **8** Jian, 9MW2821 SGO 2024

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
- CT-202 incorporates pH-dependent binding to Nectin-4 and CD3, designed to favor activity in acidic tumor environments

Preclinical tolerability and pharmacokinetics

- Well tolerated at high dose in nonhuman primates
- Pharmacokinetics may support Q2W or Q3W dosing

Ease of manufacturing

- IgG1 backbone is highly stable and enables high yield

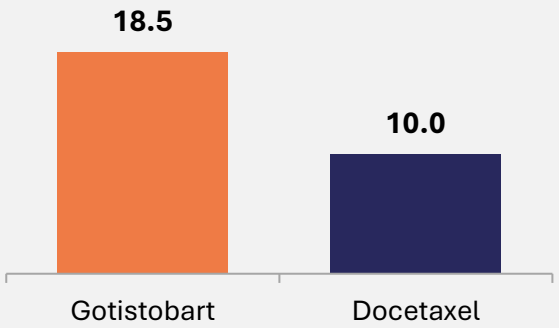
AACR 2026, “Targeting solid tumors with pH-dependent dual-specific TCEs: First-in-Human development of CT-202”

Clinical Precedent De-Risks pH-Dependent Binding for CT-202

Gotistobart and Ultomiris provide the strongest precedent for CT-202's design

	Gotistobart BNT316 / ONC-392 — anti-CTLA-4	Ultomiris® ravulizumab-cwvz — anti-C5	PRESERVE-003 Stage 1 Gotistobart vs docetaxel, 2L+ sqNSCLC
Challenge addressed	• Systemic CTLA-4 blockade drives immune-related AEs, narrowing the therapeutic index	• Frequent infusions; C5 antibody lost to target-mediated clearance	
pH-dependent design	• Binds CTLA-4 in acidic tumor microenvironment; releases at neutral pH • Confines Treg depletion to the tumor	• LS Fc mutations raise FcRn affinity at pH 6.0; release at pH 7.4 • pH-sensitive C5 release enables recycling	
Clinical & regulatory validation	• Phase 3 Stage 1: median OS 18.5 vs 10.0 months; Grade ≥3 TRAEs 44.4% vs 48.8% • FDA Fast Track (2022); Orphan Drug (2026); Stage 2 ongoing	• ~4x longer half-life than eculizumab; Q8W dosing • FDA-approved, marketed product	

Median overall survival (months)



HR 0.572

43% lower risk of death vs docetaxel

n=87 squamous pts (1:1); non-pivotal stage; no new safety signals

PRESERVE-003 (NCT05671510) Stage 1; Source: BioNTech/OncoC4, IASLC WCLC 2026 (Sept 14, 2026). Gotistobart and Ultomiris are not Context programs; cross-trial comparisons are not head-to-head.

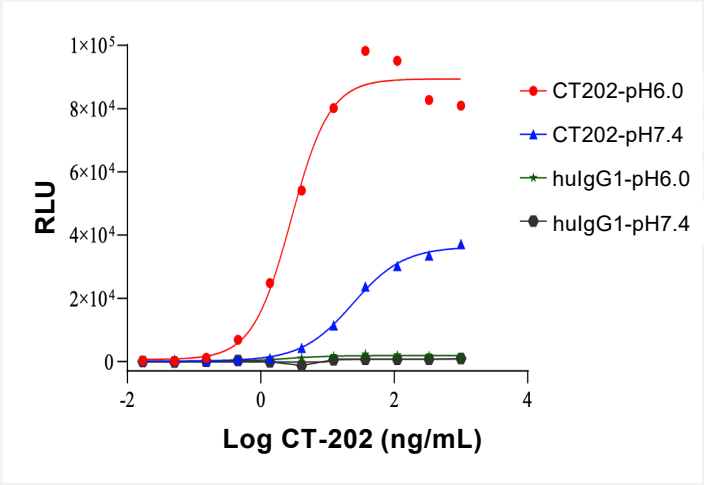
CT-202 is Designed to Optimize Tumor Selectivity and Minimize Off-tumor Toxicity

Minimize Activity in Normal Tissues at pH7.4

T cell activation at low pH(6.0) and normal pH(7.4) with OVCAR3 ovarian cancer cell targets

CT-202 preferentially activated T cells at low pH, which should maximize potency in the TME and minimize on-target/off-tumor liabilities of a Nectin-4 directed therapeutic in normal tissues

T cell Activation Assay

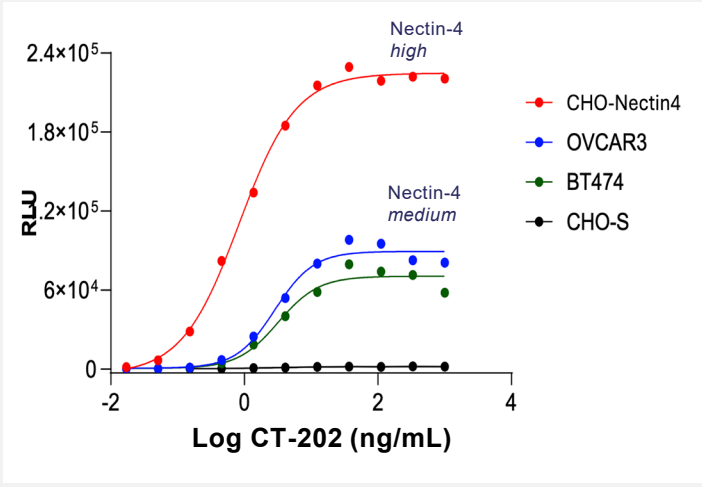


Potency is Correlated With Target Density

T cell activation by cells with varying levels of Nectin-4

CT-202 mediated robust T cell activation in the presence of the Nectin-4+ cells but not in the presence of the CHO-S cells lacking Nectin-4. Activation was proportional to the level of Nectin-4 on the surface of the target cells.

T cell Activation Assay at Low pH(6.0)

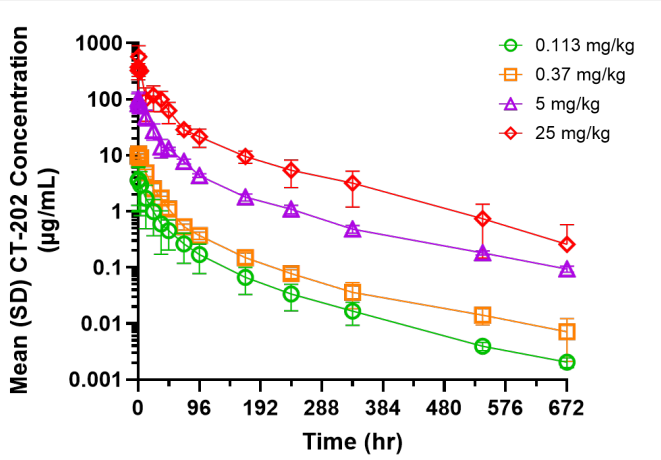


Preclinical Tolerability and Pharmacokinetics

MTD was not identified and PK supports initial biweekly (Q2W) dosing

No notable test-article related in-life effects at any dosage. HNSTD is the highest administered dosage of 25.0 mg/kg/dose

Single dose PK following IV administration in NHP



AACR 2026, "Targeting solid tumors with pH-dependent dual-specific TCEs: First-in Human development of CT-202"

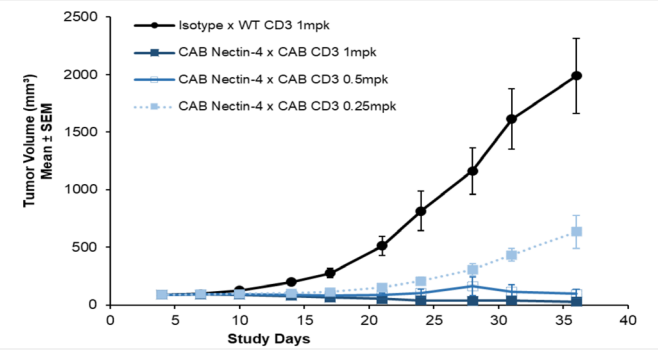
CT-202 is Highly Active and Well Tolerated Across In Vivo Models

Complete Tumor Regressions

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

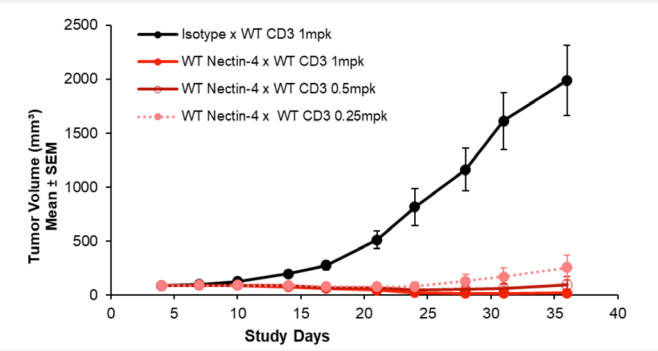
pH conditional

CT-202



Not pH conditional

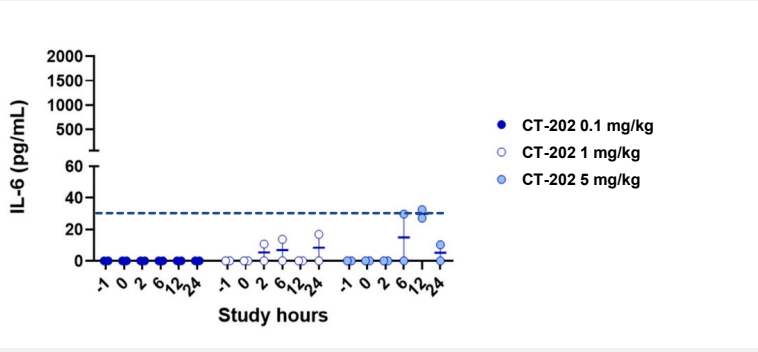
Enfortumab TCE



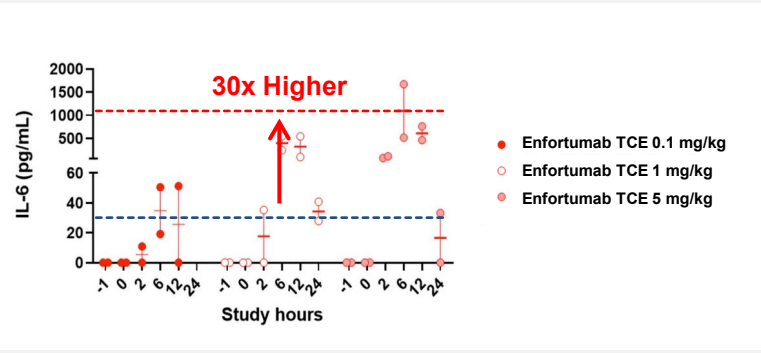
Lower Cytokine Induction

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

CT-202



Enfortumab TCE



CT-202 Phase 1 Study Design

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

Screening

Key Inclusion Criteria

- Patients with advanced metastatic urothelial, colorectal, and triple negative breast cancers
- Age ≥18 years
- Measurable disease per RECIST v1.1
- ECOG 0 or 1

Biomarker Stratification

- Nectin-4 positive (≥10% of cells 1+ IHC)

CRS Mitigation

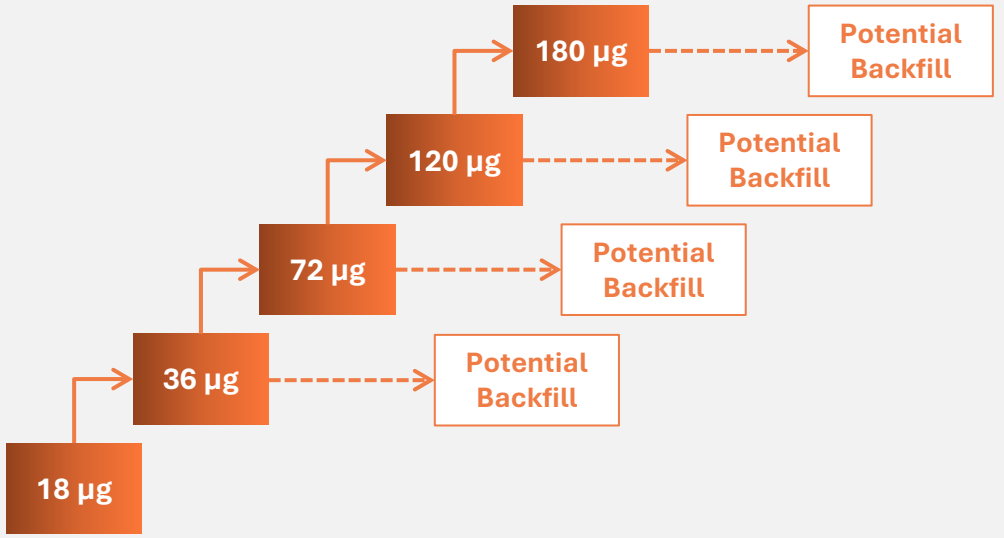
- Step / prime dose
- Steroid prophylaxis on W1D1 and W2D8

Phase 1a Dose Escalation (Q2W Dosing)

Bayesian Optimal Interval (BOIN) Design & Backfill

Study Update

- 6 trial sites open in Australia
- Cohort 1 is ongoing
- US IND filing by YE 2026



Backfill ≥1 dose levels meeting protocol-defined safety and activity criteria

CT202-101 ClinicalTrials.gov Identifier: NCT07545122. Data as of October 30, 2026.

Selected Nectin-4-directed Immune-engaging Programs

Programs differ in molecular format, immune effector, and conditional-activation strategy

Company	Context Therapeutics	Bicycle Therapeutics	Rondo Therapeutics	Marengo Therapeutics	LabGenius
Asset	CT-202	BT7480[1,2]	RNDO-564[3]	TriSTAR0701[4]	LGTX-101[5]
Format	2 + 2 (pH dependent)	1 + 2 (Bicycle)	1 + 1 (Fixed light chain)	1 + 1 (+wt IL2)	3 + 1
Conditionally active	✓	✗	✗	✗	✗
Avidity enhanced	✓	✗	✗	✗	✓
Immune Effector	**CD3**	4-1BB	CD28	CD3	CD3
Program Status	Phase 1 (FPI September 2026)	Phase 1 (completed)	Phase 1 (FPI Dec 2025)	Preclinical	Preclinical

1 Bicycle Therapeutics Corp Presentation 2024; **2** Hurov, J Immunother Cancer, 2021; **3** PEGS Boston 2024 ; **4** SITC 2025; **5** ESMO 2026. Information provided in the table above is for illustrative purposes only and is not a head-to-head comparison. Differences exist between study or trial designs and subject characteristics, and caution should be exercised when comparing data across studies.

Corporate



Experienced Leadership Team



Martin Lehr
CEO and Director

Praesidia BIOLOGICS
Cure Duchenne
OSAGE UNIVERSITY PARTNERS



Karen Chagin, MD
Chief Medical Officer

Adaptimmune
TMUNITY
GSK



Jennifer Minai
Chief Financial Officer

MILLENDO THERAPEUTICS
parexel
EY



Alex Levit, Esq
Chief Legal Officer

optinose
teva
ReedSmith



Jennifer Dashnau, PhD
SVP Technical Operations

CENTURY THERAPEUTICS
MERCK



Jill Conwell, MBA
Consulting
Chief People Officer

idera
Shire
accenture

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

Investment Highlights (Nasdaq: CNTX)

Investment Rationale

Large Unmet Need	Solid tumors and ADC resistance
High-Value Targets	Claudin 6 and Nectin-4
Strong Team	Deep oncology experience Focus on clinical execution

Anticipated Milestones

Q2 2027	**CTIM-76** Phase 1a initial Q3W data
2H 2027	**CT-202** Phase 1a initial data

Expected cash runway to early Q4 2027

context therapeutics

Advancing T Cell Engagers for Solid Tumors

© CONTEXT THERAPEUTICS 2026

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
FIH	First-in-human
FPI	First Patient In (dosed)
FRα	Folate receptor alpha
GPI	Glycosylphosphatidylinositol
IHC	Immunohistochemistry
IND	Investigational new drug
IV	Intravenous
LLOQ	Lower limit of quantitation
MABEL	Minimum anticipated biologic effect level
MoA	Mechanism of action
MTD	Maximum tolerated dose
N.D.	Not disclosed
ORR	Objective 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
TNBC	Triple-negative breast cancer
YE	Year End