context therapeutics

Advancing T Cell Engagers for Solid Tumors

Corporate Presentation

January 2025

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Building a T cell Engager Pipeline

TCEs are Gaining Momentum

Recent TCE clinical data demonstrates promising efficacy and safety in solid tumors

- Clinical activity across a broad range of targets, including Claudin 18.2, DLL3, gp100, PSMA, and STEAP1
- Responses in “cold” tumors, including neuroendocrine, pancreatic, prostate, and small cell lung cancer
- Promising safety with low rate of Grade ≥ 3 cytokine release syndrome (CRS)

Potentially Best-in-Class Assets

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

- CLDN6 is overexpressed in ovarian, endometrial, lung, 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 ovarian, pancreatic, lung, 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, breast, lung, and other solid tumors
- CT-202 was designed to be conditionally active within the tumor microenvironment

Well Capitalized

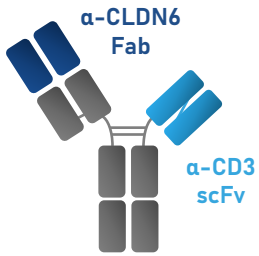
Strong financial position with high quality investor base

- Anticipated cash runway to extend into 2027

Pipeline Overview

PROGRAM	TARGET	ADDRESSABLE MARKET (U.S. ONLY)	DISCOVERY	DEVELOPMENT	PHASE 1	PHASE 2	2025 PRIORITY
CTIM-76	Claudin 6 (CLDN6)	> 50,000 patients					Dose first patient during Q1 2025, enroll Phase 1 study
CT-95	Mesothelin (MSLN)	> 100,000 patients					Dose first patient during Q1 2025, enroll Phase 1 study
CT-202	Nectin-4	> 125,000 patients					GMP campaign and IND preparation

CTIM-76: CLDN6 x CD3

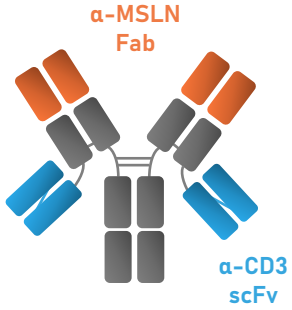


Product differentiation: highly selective for CLDN6 over CLDN3/4/9

Safety: potent CD3 induction without broad cytokine activation

Cancer Indications: ovarian, endometrial, lung, testicular

CT-95: MSLN x CD3

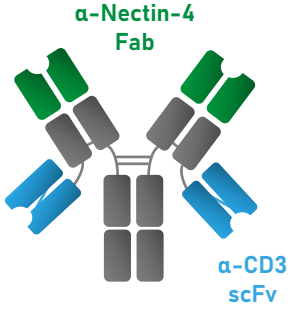


Product differentiation: avidity optimized to avoid mesothelin (MSLN) fragments

Safety: sterically hindered CD3 to avoid T cell crosslinking

Cancer indications: lung, pancreatic, ovarian, mesothelioma

CT-202: Nectin-4 x CD3



Product differentiation: conditionally activate in the tumor microenvironment

Safety: sterically hindered CD3 to avoid T cell crosslinking

Cancer indications: bladder, colon, breast

T Cell Engager Strategy

T Cell Engaging (TCE) Bispecific Antibodies

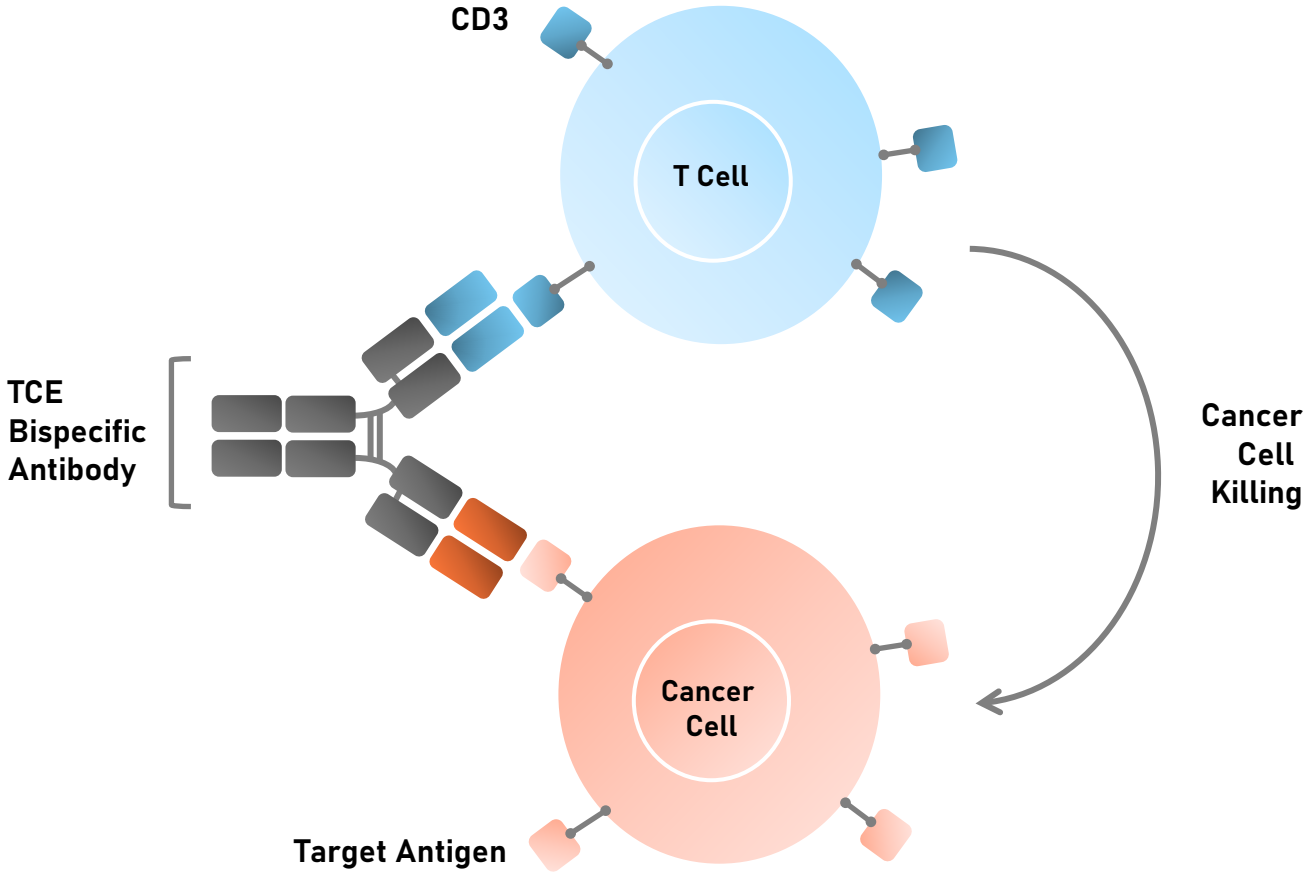
TCEs are engineered to activate an immune response against cancer cells

Mechanism of Action

T cell engagers (TCEs) are antibodies engineered to redirect the immune system's T cells to recognize and kill cancer cells

TCE bind to a target antigen expressed on a cancer cell and to an immune activator on T cells, such as CD3

This mechanism allows for the direct activation of T cells and their anti-tumor features, ultimately resulting in the killing of cancer cells



Realizing the Full Potential of T Cell Engagers (TCE)

2024 was a watershed year for TCEs

HPN328
(DLL3)

Confirmed response rate of 35% (11/31) across all tumor types (SCLC and other neuroendocrine tumors), including three complete responses

Generally well tolerated with no dose limiting toxicities at target doses[1]

$680M ACQUISITION

JANX007 / JANX008
(PSMA / EGFR)

83% (5/6) of JANX007 patients achieved PSA50 declines with first step dose ≥ 0.2mg and 56% (10/18) patients achieved PSA50 declines with the first dose ≥ 0.1mg

Early JANX008 data presented one confirmed PR and no CRS greater than Grade 1 in any cohort[2]

+$1.6B APPRECIATION

Tarlatamab /
IMDELLTRA™
(DLL3)

At 10mg, mPFS was 4.9 months with mOS of 14.3 months across 100 patients with small cell lung cancer (SCLC)

Granted Accelerated FDA Approval in May 2024[3]

$1B+ PEAK SALES OPPORTUNITY

DLL3 TCE + B7-H3 ADC[4]

DLL3 TCE + B7-H3 ADC[5]

TCR Vβ TCE + TROP2 ADC[6]

TCE + ADC
Clinical Trial Partnerships

1 Merck Press Release 8 Jan 2024; **2** Janux Press Release 26 Feb 2024; **3** Amgen Press Release 16 May 2024; **4** Daiichi Sankyo Press Release 6 Aug 2024; **5** Medilink Therapeutics Press Release 8 Oct 2024; **6** Marengo Therapeutics Press Release 13 Sept 2024. Information provided is for illustrative purposes only, is not indicative of future performance, 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.

TCEs are Starting to Have Consistent Success in Solid Tumors

Tumor shrinkage with low rate of Grade ≥ 3 cytokine release syndrome (CRS)

	AMGEN	HARPOON Therapeutics	Innovent	Janux	VIR	AMGEN
Asset	**Tarlatamab (AMG757)**	**HPN328**	**IBI389**	**JANX007**	**VIR-5500**	**Xaluritamig (AMG509)**
Target x Effector	DLL3 x CD3	DLL3 x CD3	CLDN18.2 x CD3	PSMA x CD3	PSMA x CD3	STEAP1 x CD3
Cancer Indication	Small Cell Lung	Small Cell Lung	Pancreatic	Prostate	Prostate	Prostate
Normal tissue expression	Brain	Brain	Gastrointestinal (GI)	Endocrine, GI, pancreas, skin, marrow	Endocrine, GI, pancreas, skin, marrow	Brain, respiratory, prostate, smooth muscle
Patients (n)	100	19	27	16	12 (1st dose >120 µg/kg)	21
Efficacy	**ORR: 40% mPFS: 4.9 months**	**ORR: 32%**	**ORR: 38%**	**PSA50: 100% PSA90: 63% ORR: 50%**	**PSA50: 58% PSA90: 9%**	**PSA50: 50% PSA90: 28% ORR: 20%**
Grade ≥ 3 CRS	**1%**	**3%**	**0%**	**6%**	**0%**	**2%**
Reference	Ahn 2023	ESMO 2023	ASCO 2024	15 Nov 2024 data cutoff	13 Nov 2024 data cutoff	ESMO 2024

Information provided in the table above as of January 9, 2025. Information provided 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.

Context's Approach to TCEs

Historical TCE Challenges

- ✗ Cytokine release syndrome
- ✗ Weak clinical activity
- ✗ Broadly expressed antigens
- ✗ Poor durability

Context's TCE Approach

- ✓ **Premedication + step-dosing**
- ✓ **High affinity CD3 binding**
- ✓ **Tumor restricted**
- ✓ **Combination with ADC or Checkpoint**

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
- Observed clinical responses
- Potential accelerated pathway

Selected Cancer indications	Incidence (US Only)	R/R Incidence	CLDN6 Positive	CLDN6 Med/High	Patient Population Based on R/R Incidence
Endometrial	65,900	14,000	51%[1]	22%[1]	**7,140**
Ovarian	19,900	12,800	44%[1]	25%[1]	**5,632**
Testicular	9,910	400	94%[1]	90%[1]	**376**
Non-Small Cell Lung	201,229	110,653	26%[1]	6%[1]	28,769
Colon	152,810	53,010	40%[2]	0%[2]	21,204
Breast	290,600	43,800	40%[2]	0%[2]	9,417
Sarcoma	17,100	12,390	20%[2]	10%[2]	2,478
Gastric	26,380	11,090	9%[1]	7%[1]	998

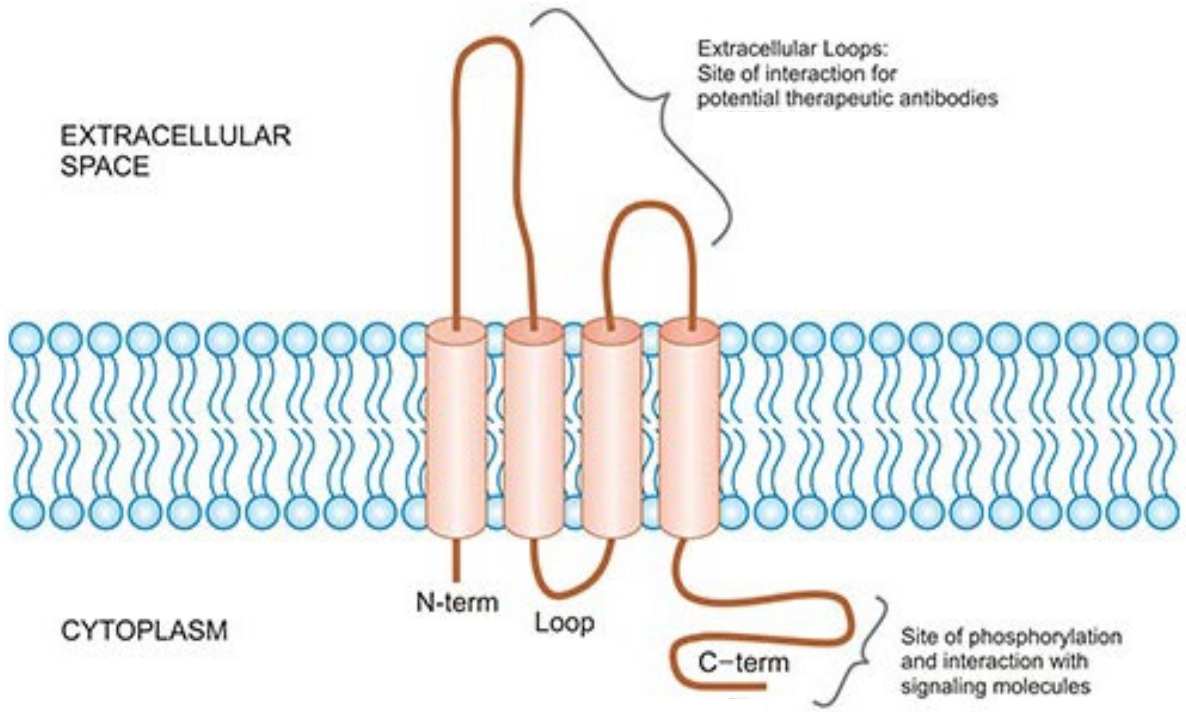
1 Context internal data; **2** Mackensen, Nature Medicine, 2023. Incidences based on public estimates; Relapsed/refractory (R/R) or last-line patient population approximated by annual mortality; CLDN6 target prevalence is based on IHC or RNAseq from published reports. Patient population derived from midpoint of CLDN6 positive population multiplied by R/R incident population.

CLDN6 is an Oncofetal Protein

Oncofetal proteins are considered favorable candidates for immunotherapy

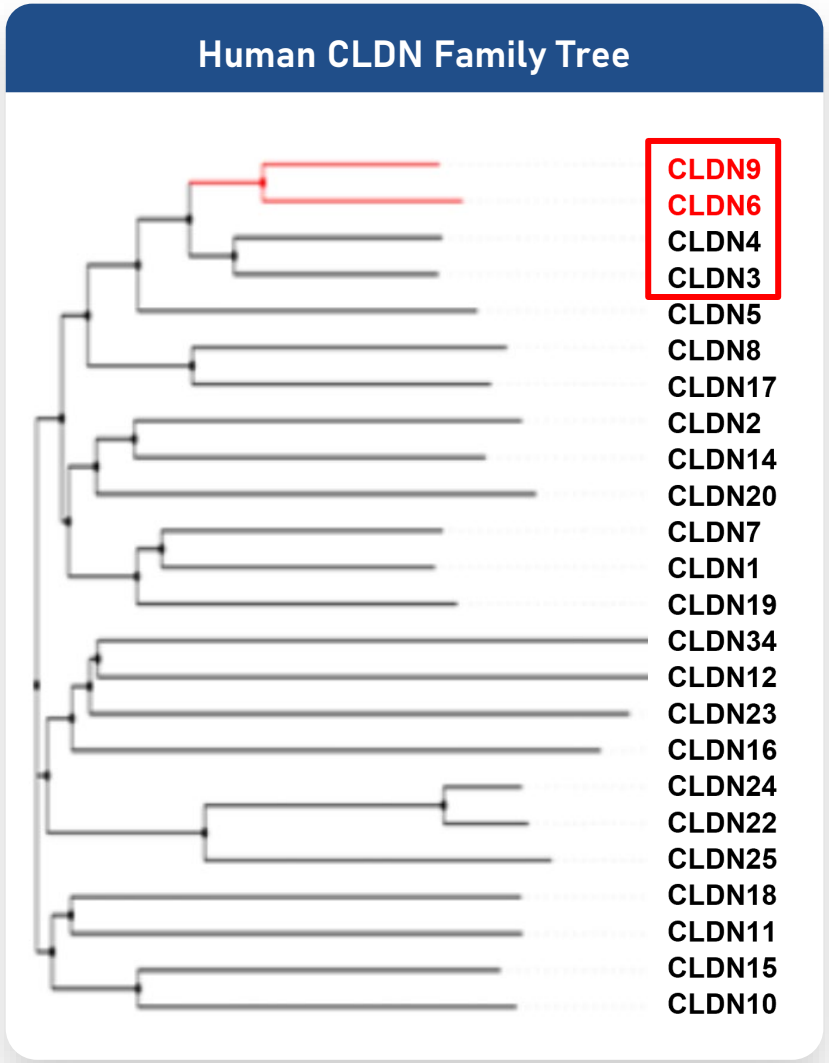
Oncofetal Characteristics of CLDN6

- Normally present at higher levels during embryonic development
- Turned off or have low levels of expression in adult tissues
- Increased expression across many solid tumors



Huan, Mol Med Reports, 2021

Developing a Highly Selective CLDN6 Antibody is Challenging



- CLDN6 antigen is **conformationally dependent**, which limits access to antibody-antigen binding
- Antigen binding region is **highly conserved** with CLDN3, CLDN4, and CLDN9, making CLDN6-selective binding a challenge[1]
- CLDN6 **selectivity is required** to avoid off-target liabilities identified in murine knockout and knockdown studies with CLDN3 (intestine)[2], CLDN4 (liver, pancreas)[3], and CLDN9 (liver, ear)[4]

1 Screnci, Cancer Res, 2022; **2** Tanaka, J Hepatol, 2018; **3** Cordat, Physiology, 2019; Li, FEBS Open Bio, 2020; **4** Nakano, PLoS Genet, 2009

CLDN6 Target Validation via ADC and CAR-T

CTIM-76 is designed to potentially address limitations of TORL-1-23 (ADC) and BNT211 (CAR-T)

High Response Rates with CLDN6 ADC and CAR-T

BIONTECH

Basket[1]
51% ORR (n=17/33)

Ovarian Cancer[1]
58% ORR (n=7/12)

Testicular Cancer[1]
41% ORR (n=5/12)

Lung Cancer[1]
1 partial response

IHC Cutoff = 50% 2+/3+ staining

TORL BIOTHERAPEUTICS

Basket[2]
33% ORR (n=15/45)

Ovarian Cancer[2]
45% ORR (n=9/20)

IHC Cutoff = >30% 1+ staining

CTIM-76 Addresses Limitations of ADC and CAR-T

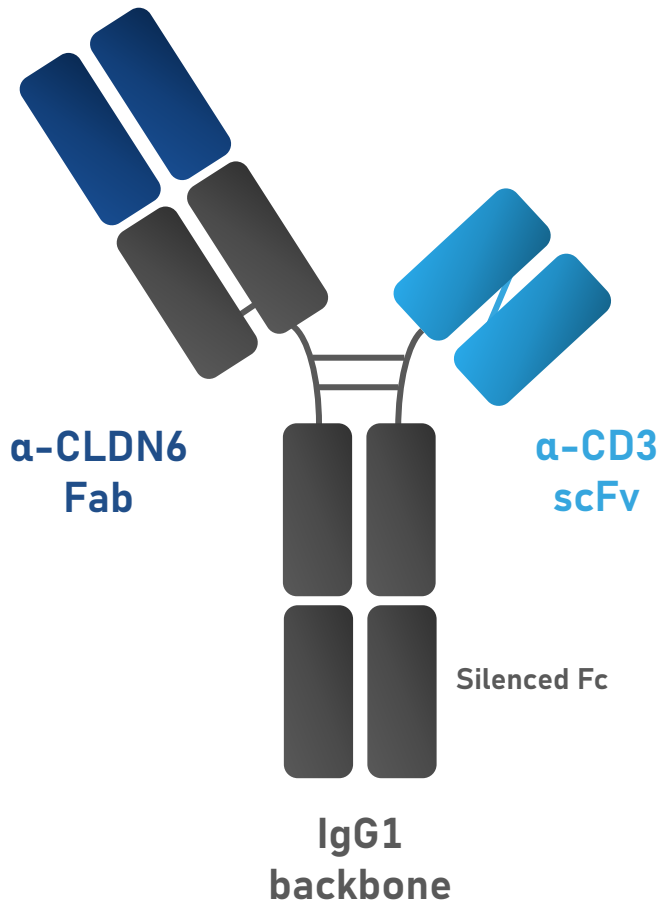
	CTIM-76	BNT211[1]	TORL-1-23[2,3]
High Potency	✓	✓	✗
Low Expression Cutoff	✓	✗	✓/✗
Scalable manufacturing	✓	✗	✓

CTIM-76 is ~50-100x more potent than TORL-1-23

CTIM-76 targets low / med / high CLDN6 expressing cells

1 Haanen, ESMO 2024; **2** Konecny, ESMO 2024; **3** Context SITC 2023. Information provided 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.

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

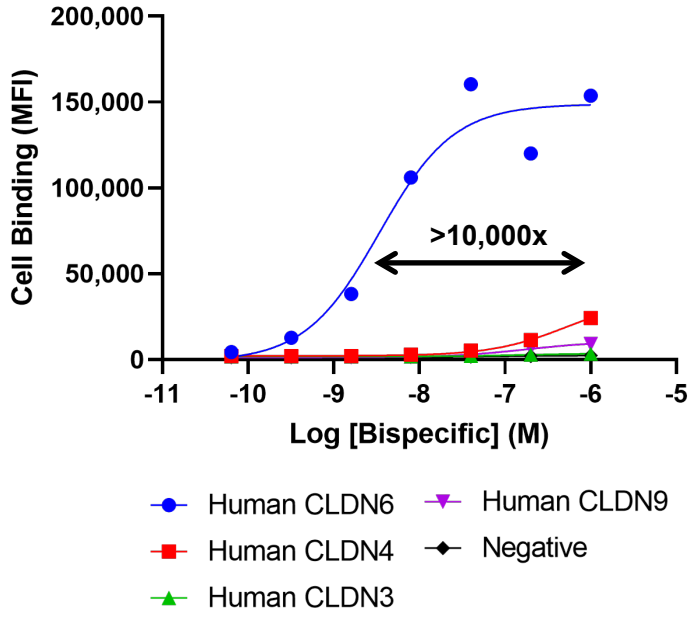
Ease of manufacturing

- IgG1 backbone is highly stable and enables high yield

Phase 1 first patient dosed anticipated in Q1 2025

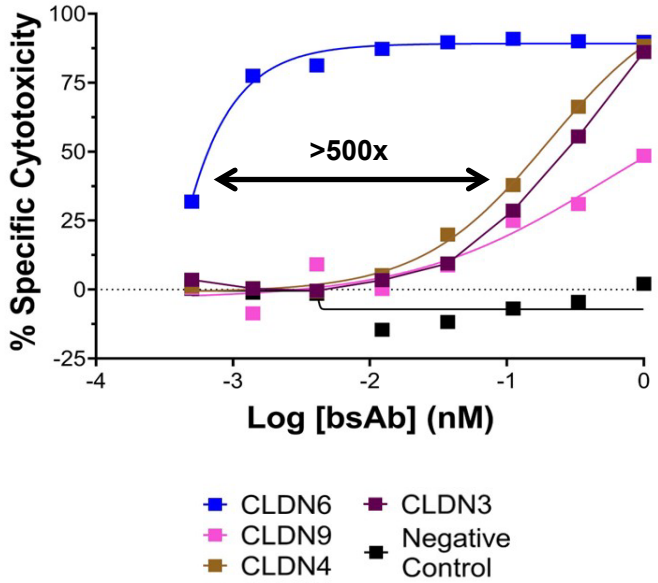
CTIM-76 is a Highly Selective and Potent CLDN6 x CD3 Bispecific Antibody

Selectivity



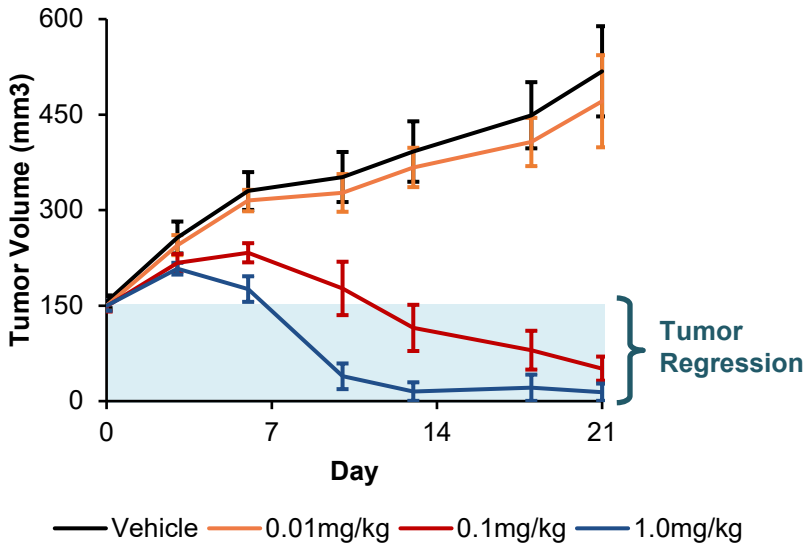
- CTIM-76 CLDN6 EC50 of 3.41 nM (binding)
- CTIM-76 preferentially binds to CLDN6 over CLDN3/4/9
- CLDN3/4/6/9 were transiently transfected in HEK-293F cells

Potency



- Potency assay provides a better assessment for a TCE bispecific than binding assays for off-target liabilities associated with CLDN3, CLDN4, or CLDN9
- CTIM-76 CLDN6 EC50 of 0.0004 nM (cytotoxicity)
- CTIM-76 preferentially targets CLDN6, with minimal binding and cytotoxicity against CLDN9-expressing cells

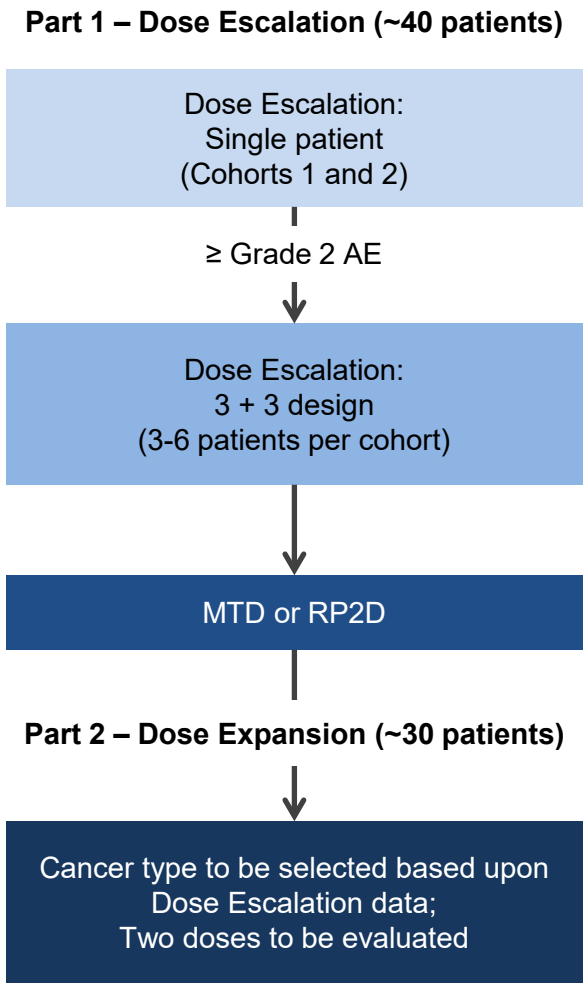
In Vivo Efficacy



- CTIM-76 effectively engaged systemically administered human PBMC cells to promote significant tumor regression and complete responses in OVCAR3 (~96,000 CLDN6 copies per cell) ovarian xenograft models in mice
- 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 1a/b Study

An open-label, multi-center, dose escalation / expansion, safety, and PK study (NCT06515613)



- **Target population**
  - Platinum resistant ovarian cancer
  - Endometrial and testicular cancer relapsed to standard of care
- **Biomarker stratification**
  - CLDN6+ positive (10% ≥ 1+) ovarian and endometrial
  - Due to high CLDN6 prevalence, testicular cancer does not require prospective screening
- **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 starting at 22.5 µg, corresponding to MABEL dose
  - Premedication (steroid + NSAID) and step dosing to manage cytokine release syndrome (CRS)

MABEL = minimum anticipated biological effect level; **MTD** = maximum tolerated dose; **RP2D** = recommended Phase 2 dose

CTIM-76 Competitive Landscape

CLDN6 T Cell Engaging Bispecifics

	Active					Discontinued
	CTIM-76	XmAb541	ARC101	SAIL66	NBL-028	AMG794
Company	Context	Xencor	Third Arc Bio	Chugai	NovaRock	Amgen
Stage	Ph 1	Ph 1	Ph 1	Ph 1	Ph 1 (China)	Ph 1 (Discontinued July 2024)[1]
Bispecific Format	1 + 1	2 + 1	n.d.	Dual Specific Fab	1 + 1	HLE Bite
CLDN6 Selectivity	**High[1]**	Moderate / High[2]	n.d.	Moderate[3]	Moderate[4]	High[5]
Preclinical Tolerability	**Well tolerated**	Well tolerated	n.d.	Poor tolerability	n.d.	Poor tolerability
Immune Effector	CD3	CD3	n.d.	CD3 / 4-1BB	4-1BB	CD3

Clinical trials.gov accessed on Sept 9, 2024 **1** Rucker, SITC 2023 **2** Faber, AACR 2021; Patent US11739144; **4** Kamikawa, SITC 2023; Patent WO2021006328 **5** Tong, AACR 2022; **5** Patent WO2022096700. **N.D.**= not disclosed. 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.

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

Initial indications of interest based on:

- MSLN prevalence
- Patient population size
- Potential accelerated pathway

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	201,229	110,653	**55%**	**36%**	**60,859**
Pancreatic	66,440	51,750	**80%**	**61%**	**41,400**
Ovarian	19,900	12,800	**90%**	**80%**	**11,520**
Mesothelioma	3,000	2,500	**70%**	**60%**	**1,750**
Colon	152,810	53,010	41%	17%	**21,734**
Esophageal	22,370	16,130	41%	26%	**6,613**
Endometrial	65,900	14,000	45%	23%	**6,300**
Gastric	26,380	11,090	49%	23%	**5,434**
Breast (TNBC)	62,054	15,500	30%	18%	**4,650**
Cervical	13,820	4,360	42%	21%	**1,831**

Incidences based on public estimates; Relapsed/refractory (R/R) or last-line patient population approximated by annual mortality; MSLN target prevalence is based on Simon et al, Biomedicines, 2021. Patient population derived from MSLN positive population multiplied by R/R incident population.

Mesothelin (MSLN) Target Validation via ADC and CAR-T

CT-95 has the potential to be used after RC88 and HBM-9033, or in combination

Recent Investor and Strategic Interest in MSLN

December 2023

Pfizer licensed ex-Asia rights to HBM-9033 for $53 million upfront and up to $1.05 billion in milestone payments

June 2024

Phase 1 data in MSLN-high for RC88[1]:

- **45% ORR** in platinum-resistant ovarian cancer
- **33% ORR** in cervical cancer
- **31% ORR** in non-small cell lung cancer

August 2024

$144 million Series B financing led by RA Capital to fund OPB-101 lead MSLN CAR-T

CT-95 Use Case

- Potential to be used as a monotherapy or in combination with MSLN ADC or FRα ADC
- Target validation via RC88 (ADC) in MSLN-high population
- ADC treatment debulks tumor, resulting in clonal selection for low/medium MSLN cells
- TCE is ~50-100x more potent than ADC, making it ideally suited to treat low/medium expressors
- TCE and ADC have non-overlapping mechanisms of action, safety, and resistance mechanisms

1 Remegen Biosciences, Press Release, 3 June 2024. Information provided is for illustrative purposes and is not indicative of future performance.

MSLN Target Biology

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

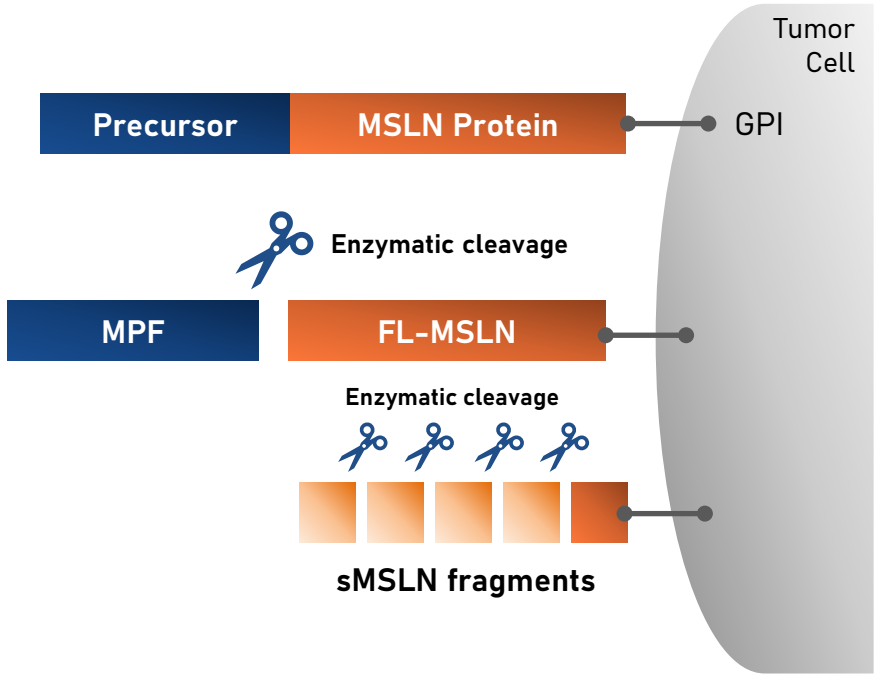
Overcoming Fragmented MSLN in the Tumor Microenvironment

MSLN is bound to tumor cells via a GPI-anchor

Like many GPI-anchored proteins, MSLN can be cut into smaller fragments[1,2]

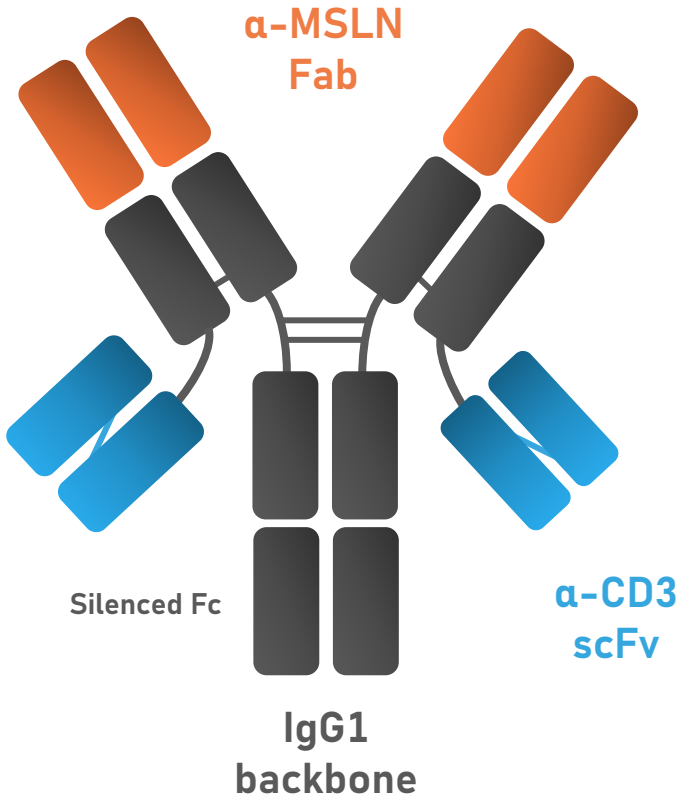
The MSLN gene encodes a precursor that is cleaved into two products: a soluble N-terminal protein called megakaryocyte potentiating factor (MPF), and a membrane-bound fragment called full length mesothelin (FL-MSLN)

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



1 Zhang, Transl Oncol, 2022; **2** Liu, Commun Biol, 2020; **GPI** = Glycosylphosphatidylinositol

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



Novel design to overcome mesothelin (MSLN) sink

- Binds to membrane-proximal MSLN epitope
- 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

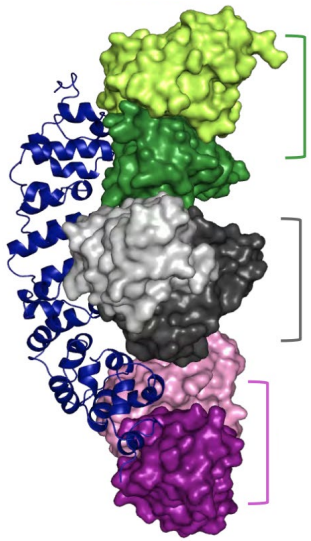
- IgG1 backbone is highly stable and enables high yield
- Drug product ready for Phase 1 trial

Phase 1 first patient dosed anticipated in Q1 2025

Two-Pronged Approach to Overcoming Soluble MSLN Sink Challenge

Binds MSLN Epitope Close to Cell Surface

Far
From cell surface



Amatuximab
MORAb-009 mAb

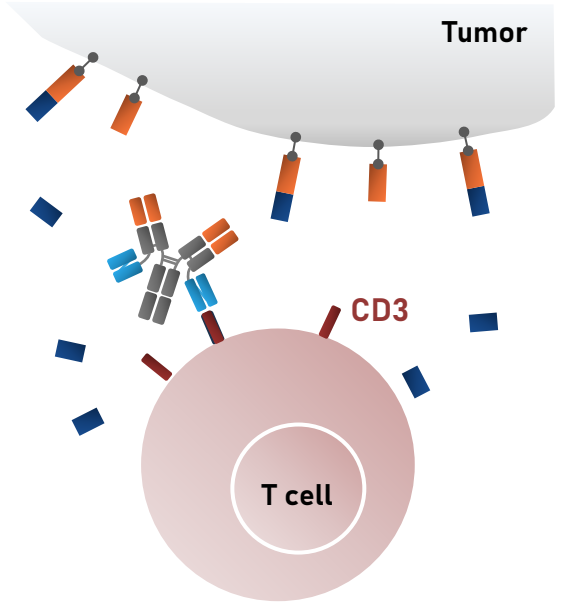
Anetumab
BAY 94-9343 ADC

CT-95
Binds membrane-proximal epitope

Close
To cell surface

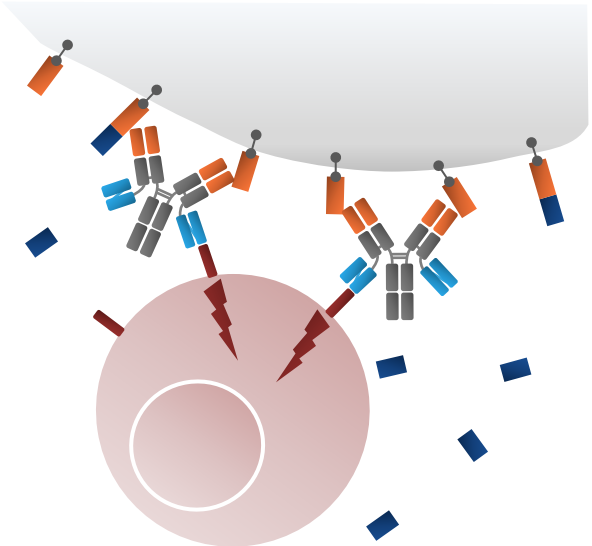
Activates T cells Through Cooperative Binding

No crosslinking with shed MSLN



No T cell activation

MSLN on tumor crosslinks CD3



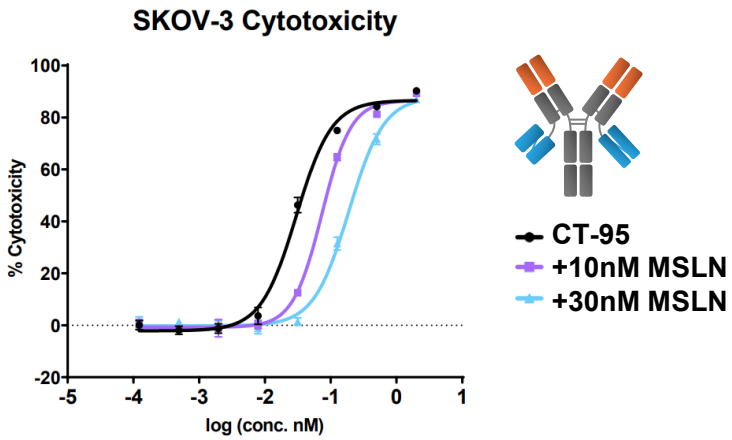
Potent T cell activation

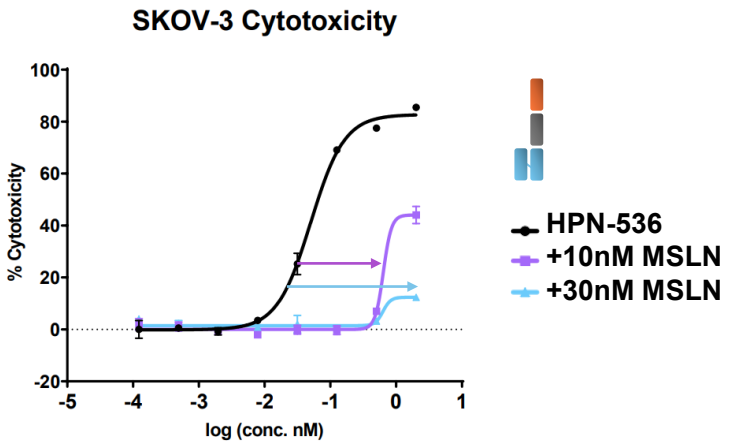
CT-95 Intended to Overcome MSLN Sink

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

Low MSLN Expression

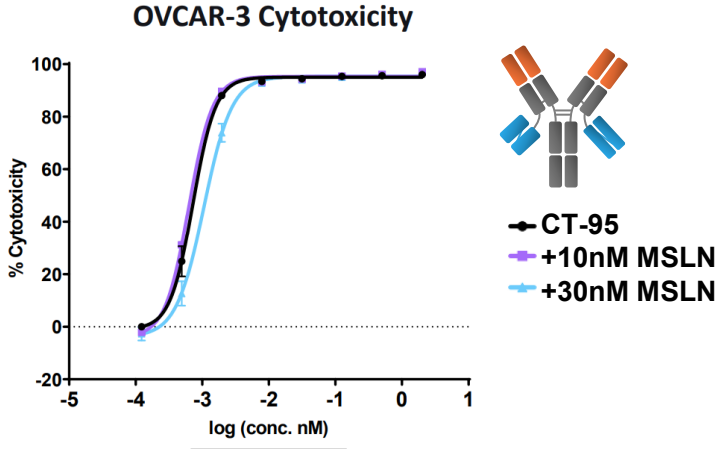
4k copies per cell

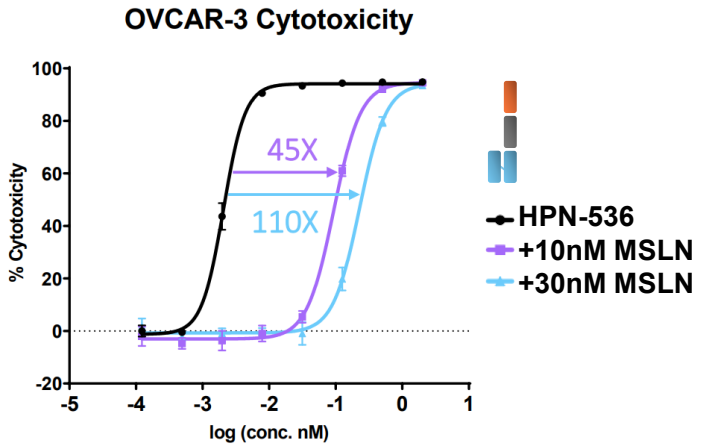




Medium MSLN Expression

27k copies per cell





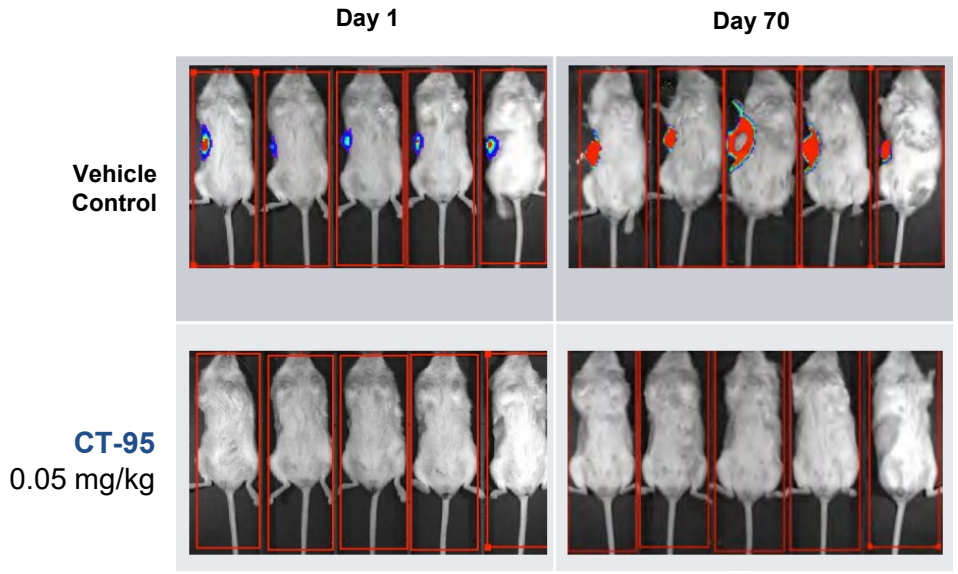
HPN-536 clones are not derived from the original manufacturer and were produced for this research study based on the published sequence of their antibody variable chains; thus, the clones used in this study are biosimilars and may not be identical to the antibodies formulated for clinical development.

CT-95 is Highly Active and Well Tolerated Across In Vivo Models

Complete tumor regressions in mice at doses ≤ 0.05 mg/kg

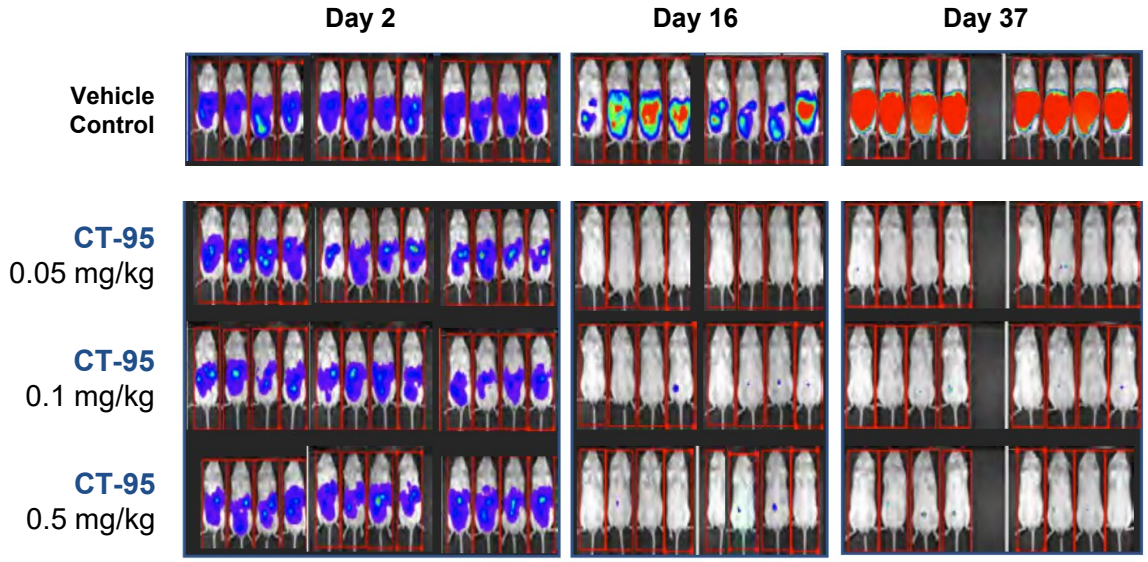
Primary Lesion Model

Ovarian cancer line OVCAR3 flank implantation tumor model



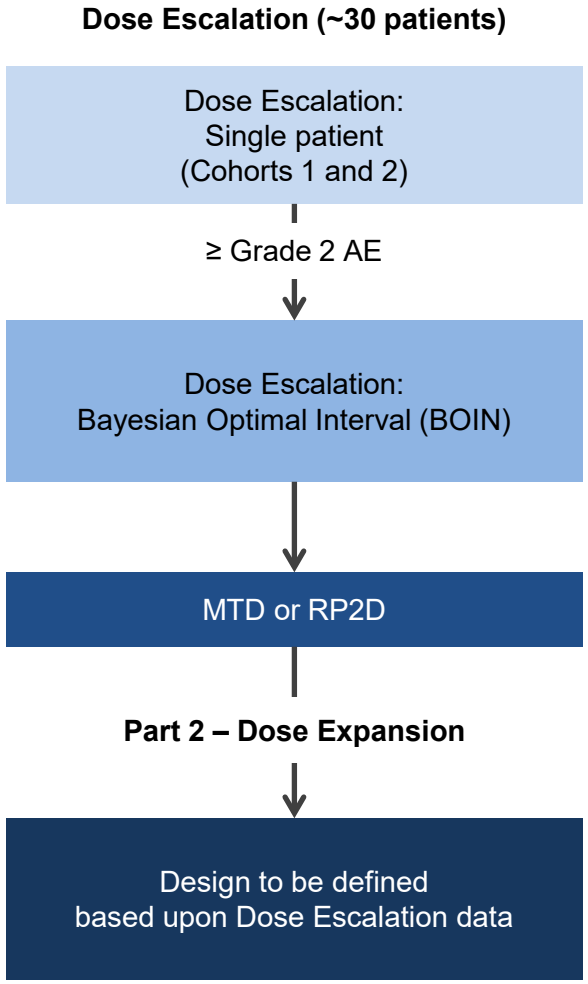
Metastatic Lesion Model

OVCAR3 pre-passaged in mice to generate aggressive, metastatic tumor model



CT-95 Phase 1 Study

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



- **Target population**
  - Platinum resistant ovarian cancer
  - Mesothelioma, pancreatic, and lung cancer
- **Biomarker stratification**
  - Due to high MSLN prevalence, ovarian cancer does not require prospective screening
  - All other indications require prospective MSLN screening via IHC
- **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 starting at 0.05 µg/kg, corresponding to MABEL dose
  - Premedication (steroid + NSAID) and step dosing to manage cytokine release syndrome (CRS)

CT-95 Competitive Landscape

1st generation MSLN T cell engagers (TCE) were discontinued due to poor efficacy

- **HPN-536:** poor drug exposure due to binding to shed MSLN and albumin[1]
- **ABBV-428:** 0% overall response rate at highest dose tested (3.6 mg/kg)[2]

	Active					Discontinued		
	CT-95	JNJ-79032421	ZW171	AMG-305	NAV-003	HPN-536	ABBV-428	NM28-2746
Company	Context	JNJ	Zymeworks[3,4]	Amgen[5]	Navrogen[6]	Harpoon	AbbVie	Numab[7]
Format	2 + 2	1 + 1	2 + 1	2 +2 CDH3 + MSLN dBiTE	2 + 2	TriTAC	2 + 2	Trispecific
PK Enhancement	Fc	Fc	Fc	Truncated Fc	Fc	Albumin	Fc	Albumin
Avoids MSLN sink	✓	✓	✗	✓	✓	✗	✗	✓
High potency TCE	✓	✗	✗	✓	✓	✓	✗	✓
Program Status	Phase 1 Opened Dec 2024	Phase 1 Opened Feb 2024	Phase 1 FPI Oct. 2024	Phase 1 FPI Oct. 2023	Preclinical Candidate	Phase 1	Phase 1	Phase 1 (China)

1 Harpoon Therapeutics Corporate Presentation, 4 June 2021; **2** Fong, J Immunother Cancer, 2021; **3** Piscitelli, PEGS Boston, 2023; **4** Zymeworks R&D Day, 2022; **5** WO2022096716A2; **6** Kline, Eur J Immunol, 2023; **7** Urech, Oncoimmunology, 2023. 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.

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

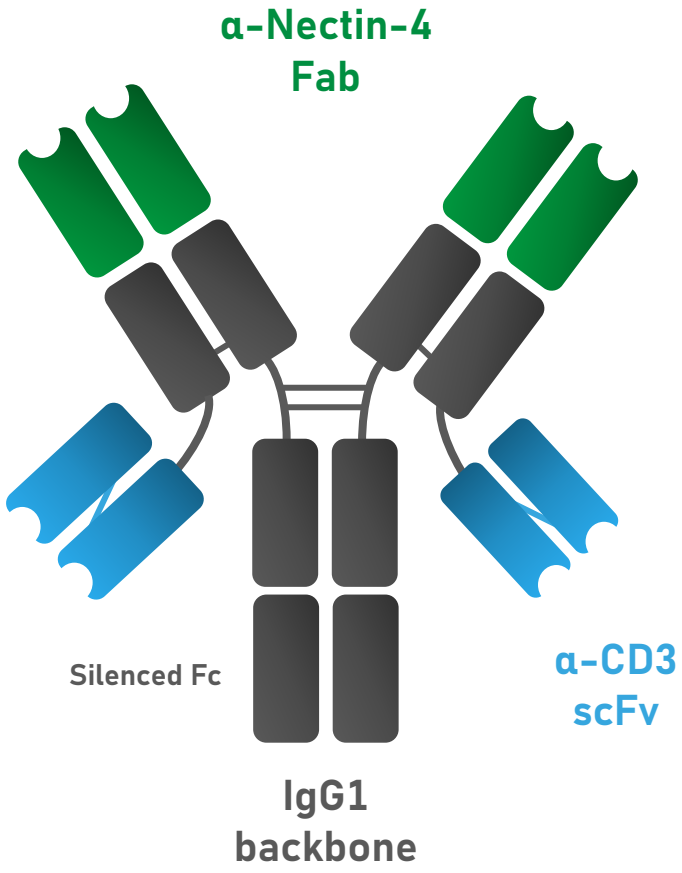
Initial indications of interest based on:

- 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
Non-Small Cell Lung	201,229	110,653	64%[1]	30%[1]	**70,818**
Colon	152,810	53,010	87%[1]	78%[1]	**46,119**
Pancreatic	66,440	51,750	71%[1]	37%[1]	**36,743**
Bladder (urothelial)	83,190	20,000	83%[1]	60%[1]	**16,600**
Breast (TNBC)	62,054	15,500	69%[1]	53%[1]	**10,695**
Head and Neck	54,000	12,000	59%[1]	18%[1]	**7,080**
Esophageal	22,370	16,130	55%[1]	24%[2]	**8,872**
Gastric	26,890	12,000	71%[3]	60%[3]	**8,520**
Ovarian	19,900	12,800	57%[4]	2%[4]	**7,296**

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** Challita, Can Res, 2016; **2** Zhang, Oncol Lett, 2018; **3** Derycke, Am J Clin Pathol, 2010; **4** Kobecki, Int J Mol Sci, 2023

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

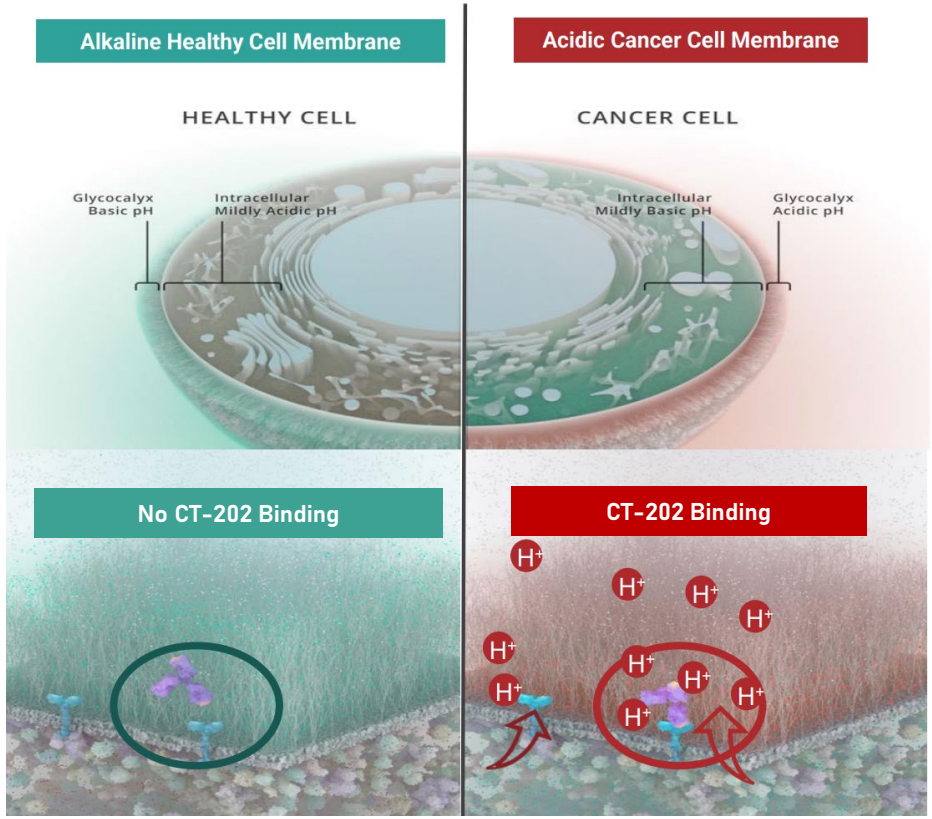
- IgG1 backbone is highly stable and enables high yield

Anticipated IND filing in mid 2026

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

Logic Gating Through pH Dependency

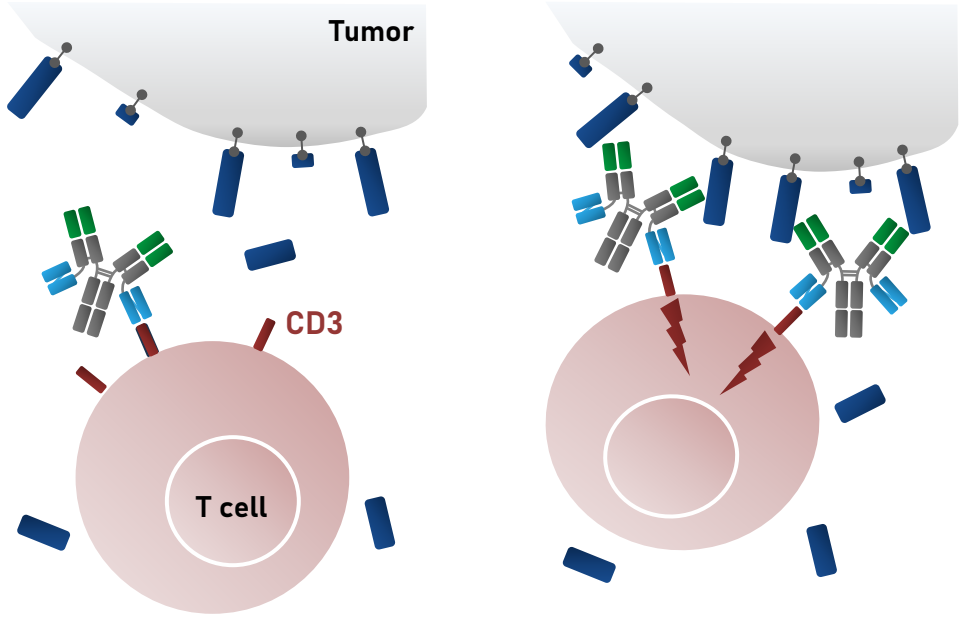
30x gain of activity in acidic tumor microenvironment versus healthy cells[1]



Increased Target Selectivity Through Avidity

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

Avidity-enhanced Nectin-4 binding



No T cell activation

Potent T cell activation

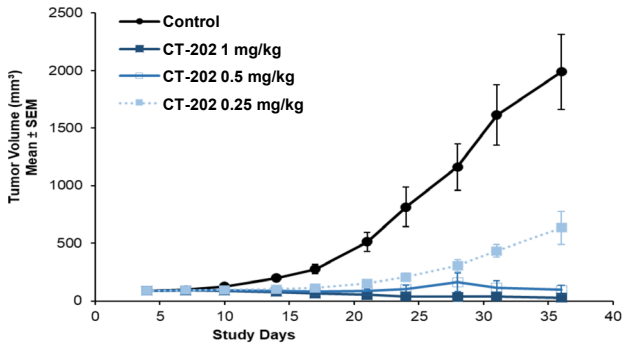
1 Chang, PNAS, 2021

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

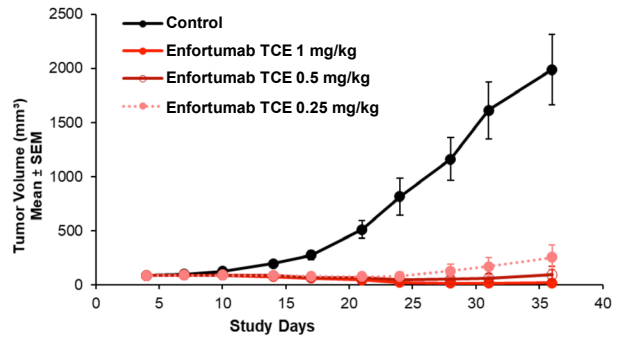
Complete Tumor Regressions

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

CT-202



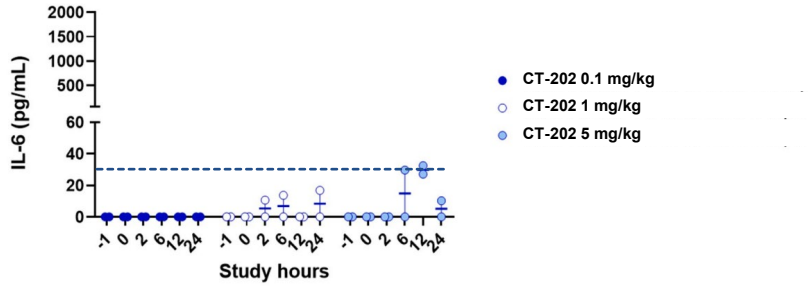
Enfortumab TCE



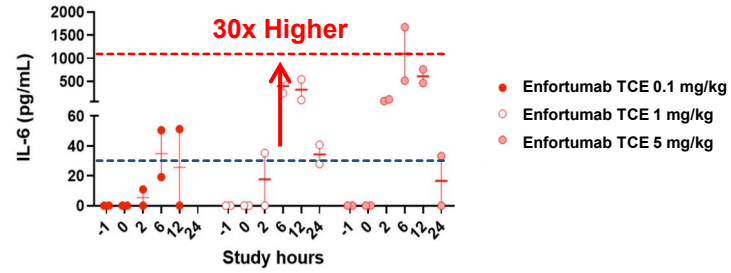
Reduced Cytokine Release

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

CT-202



Enfortumab TCE



Padcev = enfortumab vedotin

CT-202 Competitive Landscape

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

- **BT7480:** 2 partial responses out of 33 patients treated in a Phase 1 dose escalation trial, pursuing combination studies going forward[1]
- **RNDO-564:** detuning CD28 may limit potency in tumor cells with low or moderate Nectin-4 expression[3]

Company	Context Therapeutics	Bicycle Therapeutics	Rondo Therapeutics	Henlius
Asset	CT-202	BT7480[2]	RNDO-564[3]	HLX-309
Format	2 + 2 (pH dependent)	1 + 2 (Bicycle)	1 + 1 (Fixed light chain)	n.d.
Conditionally active	✓	✗	✗	**n.d.**
Avidity enhanced	✓	✗	✗	**n.d.**
Immune Effector	**CD3**	**4-1BB**	**CD28**	**4-1BB**
Program Status	Preclinical (IND filing Mid 2026)	Phase 1 (completed)	Preclinical (Ph 1 late 2025)	Preclinical

1 Bicycle Therapeutics R&D Day, Dec 2023; **2** Hurov, J Immunother Cancer, 2021; **3** PEGS Boston 2024. 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

Claudio Dansky Ullmann, MD
Chief Medical Officer

AVENGE BIO
MaxCyte
Infinity PHARMACEUTICALS

Jennifer Minai
Chief Financial Officer

MILLENDO THERAPEUTICS
parexel
EY

Alex Levit, Esq
Chief Legal Officer

optinose
teva
ReedSmith

Chris Beck, MBA
SVP Operations

Galera Therapeutics, Inc
Shire
MERCK

Karen Andreas, MS
VP, Clinical Operations

AVENGE BIO
MERRIMACK
BOSTON pharmaceuticals

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)

Large Unmet Need

Solid Tumors

+

ADC Resistance

High-Value Targets

Claudin 6

+

Mesothelin

+

Nectin-4

Anticipated Milestones

CTIM-76
initial data
1H 2026

CT-95
initial data
Mid 2026

CT-202
IND filing
Mid 2026

Strong Team

Deep oncology experience

+

Focus on clinical execution

Cash Runway

Expected cash runway into 2027

context therapeutics

Advancing T Cell Engagers for Solid Tumors

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
Fab	Fragment antigen-binding region
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
RP2D	Recommended Phase 2 dose
TCE	T cell engager
TRAE	Treatment-related adverse event
scFv	Single chain variable fragment