context therapeutics

Advancing Medicines for Female Cancers and Other Solid Tumors

Corporate Presentation

January 2023

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Context Therapeutics Overview

Our Mission	• Advancing medicines for solid tumors, with a primary focus on female cancers
CTIM-76 *CLDN6 x CD3 bispecific antibody*	• Claudin 6 (CLDN6) is uniquely expressed in a broad range of solid tumors, including ovarian and endometrial • CTIM-76 is Context's CLDN6 x CD3 bispecific antibody Development Candidate • CTIM-76 is selective for CLDN6 over other CLDN proteins, reducing the risk of potential off target side effects • IND submission on track for Q1 2024
ONA-XR *oral PR antagonist*	• ONA-XR is a novel, potentially first-in-class progesterone receptor (PR) antagonist • PR signaling drives metastasis and immune evasion in breast, endometrial, and ovarian cancer • Encouraging clinical activity and safety in ongoing Phase 2 endometrial cancer (post-chemotherapy) and breast cancer (post-CDK4/6 inhibitor) trials[1,2] • Clinical collaboration in metastatic breast cancer (post-CDK4/6 inhibitor) ongoing with the Menarini Group to evaluate combination of Menarini's novel selective estrogen receptor degrader, elacestrant, with ONA-XR
Cash Guidance	• Expected cash runway into Q1 2024

1 Data cut off as of September 30, 2022; preliminary raw data
2 Kamaraju, San Antonio Breast Cancer Symposium, 2022

Pipeline Highlights

Cancer	Clinical Indication	Preclinical	Phase 1 Clinical	Phase 2 Clinical	Milestones
CTIM-76 (CLDN6xCD3 bispecific antibody)					
	CLDN6-positive cancers				**Candidate selection Q4 2022** ☑ IND submission Q1 2024
ONA-XR (PR antagonist)[1]					
Endometrial Cancer	**Recurrent PR+ Endometrioid** Combination with anastrozole in post-chemotherapy treated patients		**Phase 2 Trial OATH Trial***		**Initial data Q4 2022** ☑ Data update mid-2023
Breast Cancer	**2L/3L ER+,PR+,HER2-** Combination with elacestrant in post-CDK4/6 inhibitor treated patients		**Phase 1b/2 ELONA Trial**		**Initiated Q4 2022** ☑ Phase 1b data Q4 2023
	2L/3L ER+,HER2- Combination with fulvestrant in post-CDK4/6 inhibitor treated patients		**Phase 2 SMILE Trial***		**Initial data Q4 2022** ☑ Data update Q4 2023

1 Tyligand Biosciences Ltd licensed rights to ONA-XR in China, HK, Macau
* Investigator Sponsored Trial

2022 Highlights and Future Milestones

CTIM-76	2H 2022	1H 2023	2H 2023	1H 2024
Candidate selection	✓			
Preclinical update		●		
IND submission				●

ONA-XR	2H 2022	1H 2023	2H 2023	1H 2024
Endometrial – OATH trial Phase 2 initial data	✓			
Endometrial – OATH trial Phase 2 data update		●		
Endometrial – OATH trial Phase 2 top line data			●	
Breast – ELONA trial Phase 1b data			●	
Breast – SMILE trial Phase 2 initial data	✓			
Breast – SMILE trial Phase 2 data update			●	
Breast – SMILE PK/PD data (^{18}F-FFNP)			●	

CTIM-76

CLDN6xCD3 bispecific antibody

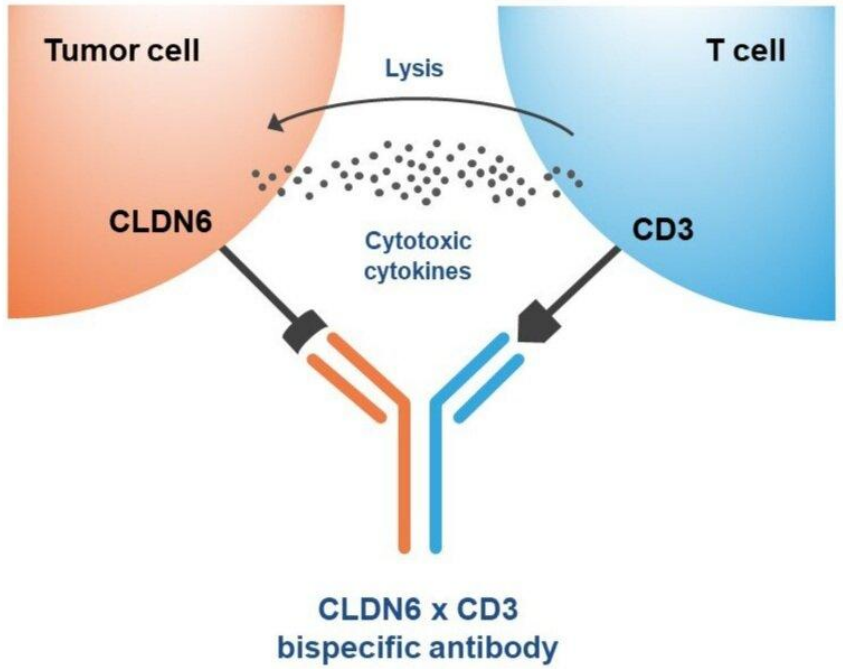
Emerging Role of Bispecific Antibodies in Treating Solid Tumors

Harnessing the Immune System to Attack Solid Tumors

- A challenge for targeting solid tumors is that many tumor-associated antigens are also expressed on normal tissues, raising concerns about “on-target off-tumor” toxicities
- Bispecific antibodies (BsAbs) are antibodies with two binding sites directed at two different targets, which can be exploited for targeting a tumor cell (e.g., CLDN6) and an immune cell (e.g., CD3)
- Compared with monoclonal antibodies, bispecific antibodies not only have stronger specificity, better targeting ability and lower off-target toxicity, but also can effectively prevent drug resistance, reduce treatment costs and improve patient access to drugs, achieving a superior therapeutic effect

Bispecific Antibodies are a Hot Field for Drug Development

- Over 50 CD3 bispecific T-cell engagers in clinical development
- Common solid cancer targets include Claudin 18.2, DLL, GPC3, HER2, PSMA
- 9 bispecific antibodies are currently approved worldwide and business development activity for BsAbs was particularly robust in 2022



Select Early-stage Bispecific Antibody Transactions in 2022[1]

Licensee	Licensor	Target	Asset	Stage	Geography	Upfront ($M)	Milestones($M)
TeneoTwo	AstraZeneca	CD19 x CD3	TNB-486	Phase 1	Worldwide	$100	$1,165
Macrogenics	Gilead	CD123 x CD3	MGD024	IND	Worldwide	$60	$1,700
LAVA	Seagen	EGFR x γδ T cell	LAVA-1223	Preclinical	Worldwide	$50	$650
Harbour	AstraZeneca	Claudin 18.2 x CD3	HBM7022	Preclinical	Worldwide	$25	$350

1 Representative transactions based on publicly available information and represents a non-head-to-head summary comparison

Claudin 6 (CLDN6) is an Ideal Target for Bispecific Antibodies

Opportunity	• CLDN6 is a **tumor-specific protein** that is present at high surface density across many adult and pediatric cancers[1] • CLDN6 is expressed at **very low levels or absent** in normal adult tissue
Challenge	• CLDN6 antigen is **conformationally dependent,** which limits access to antibody-antigen binding and antibody development • The CLDN6 antigen binding region is **highly conserved** with CLDN3, CLDN4, and CLDN9, which increases the risk of off target binding and potential side effects associated with CLDN3 (pancreas), CLDN4 (kidney, pancreas), and CLDN9 (ear, gut)
Target Validation	• BioNTech's BNT211 CAR-T cell therapy establishes **Proof of Concept**[2]: – BNT211 cell therapy evaluated in Phase 1 dose-escalation study in CLDN6-positive solid tumors – **50% response rate** (ORR) in second dosing cohort
Unmet Need	• **Selectivity:** limited off target effects • **Potency:** effective tumor killing • **Safety:** decreased risk of dangerous immune response • **Manufacturability:** ability to treat many patients

1 Faber MS, et al. Bispecific claudin-6 x CD3 antibodies AACR Annual Meeting; 2021; Virtual. Abstract 1860
2 Haanen JB, et al. BNT211: A Phase I trial. ESMO Annual Meeting; 2022; Paris, France. LBA38

CLDN6 Has the Potential to Reach a Large Patient Population

~62,500 patients per year in the United States in the Relapse/Refractory Setting

Initial indications of interest based on:

- CLDN6 prevalence
- Patient population size
- Observed clinical responses
- Eligibility for Orphan or Rare Pediatric Designation

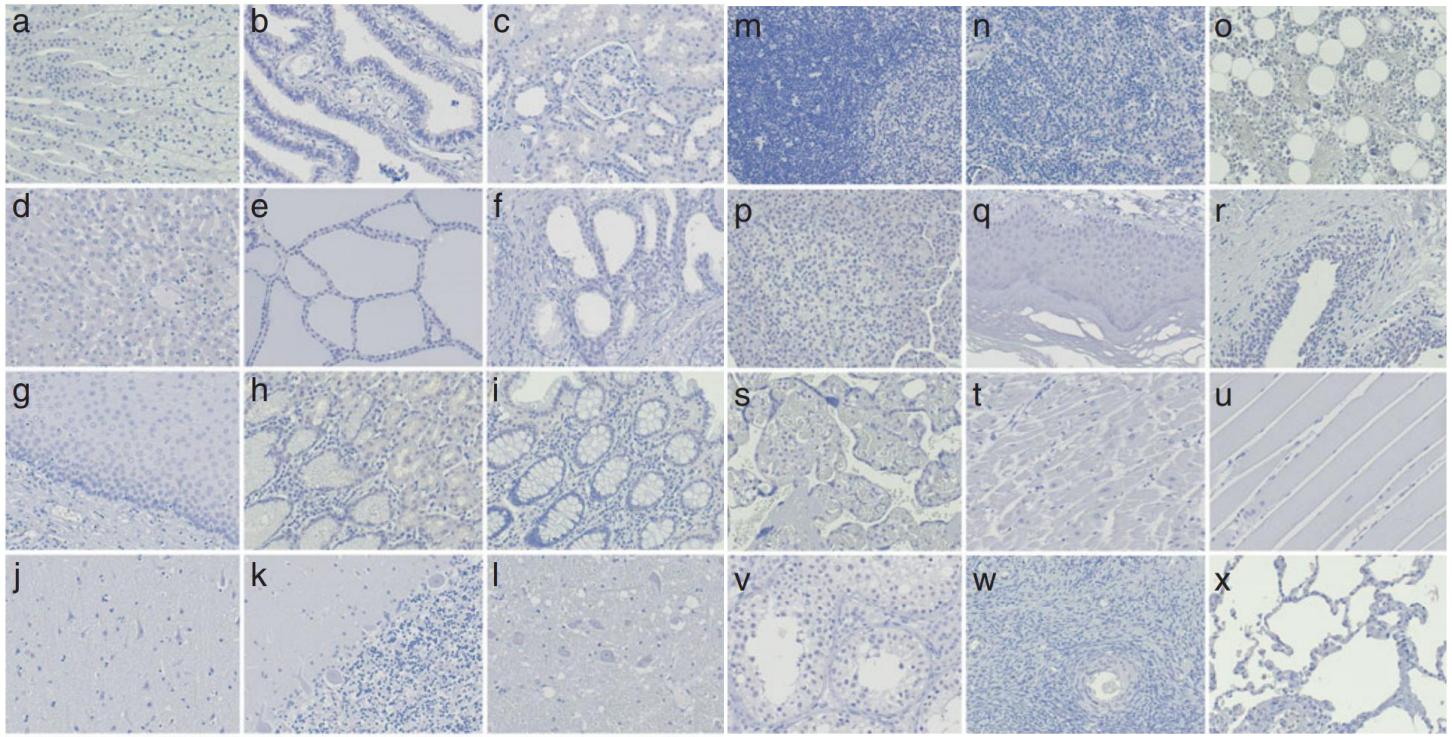
Selected Cancer indications	Incidence	Relapse / Remitting (R/R) Incidence	CLDN6 Positive	Patient Population Based on R/R Incidence
Testicular	9,910	400	95%[1]	**380**
Ovarian	19,900	12,800	54-55%[1,2]	**6,982**
NSCLC (lung)	201,229	110,653	6-50%[3,4,5]	**35,221**
Malignant Rhabdoid	50	500	29-44%[1,2,6,7]	**183**
Gastric (stomach)	26,380	11,090	13-55%[8,9]	3,771
Breast	290,600	43,800	2-41%[1,10,11]	9,417
Endometrial (uterus)	65,900	12,500	20-31%[1,12,13]	3,188
Glioma (brain)	19,000	10,000	21%[8]	2,100
Urothelial (bladder)	81,180	17,100	2-8%[1,13]	855
SCLC (lung)	35,511	19,527	2%[1]	391

1 Reinhard, Science, 2020; 2 Wang, Diagn Pathol., 2013; 3 Gao, Oncol Lett., 2013; 4 Kohmoto, Gastric Cancer, 2020; 5 Lin, Diagn Pathol., 2013; 6 Micke, Intl J Cancer, 2014; 7 Soini, Pol J Path, 2022; 8 Antonelli, Brain Pathol., 2011; 9 Sullivan, Am J Surg Pathol., 2012; 10 Jia, Intl J Clin Exp Pathol., 2019; 11 Yafang, J Breast Cancer, 2011; 12 Kojima, Cancers, 2020; 13 Ushiku, Histopath., 2012

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.

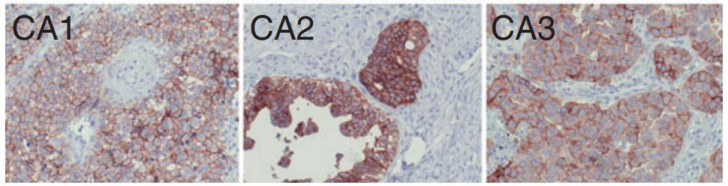
CLDN6 is Enriched in Cancer Cells vs. Non-Cancer Cells

Non-cancer, Healthy Cells



(a) adrenal gland, (b) fallopian tube, (c) kidney, (d) liver, (e) thyroid, (f) prostate, (g) esophagus, (h) stomach, (i) colon, (j) cerebrum, (k) cerebellum, (l) spinal cord, (m) thymus, (n) spleen, (o) bone marrow, (p) pancreas, (q) skin, (r) bladder, (s) placenta, (t) heart muscle, (u) striated muscle, (v) testis, (w) ovary, (x) lung

Cancer Cells

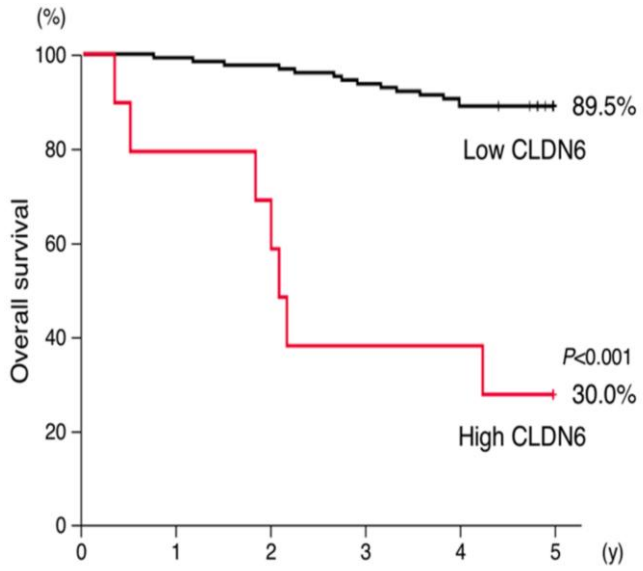


(CA1) testicular cancer, (CA2) ovarian cancer, and (CA3) lung cancer

Reinhard, Science, 2020

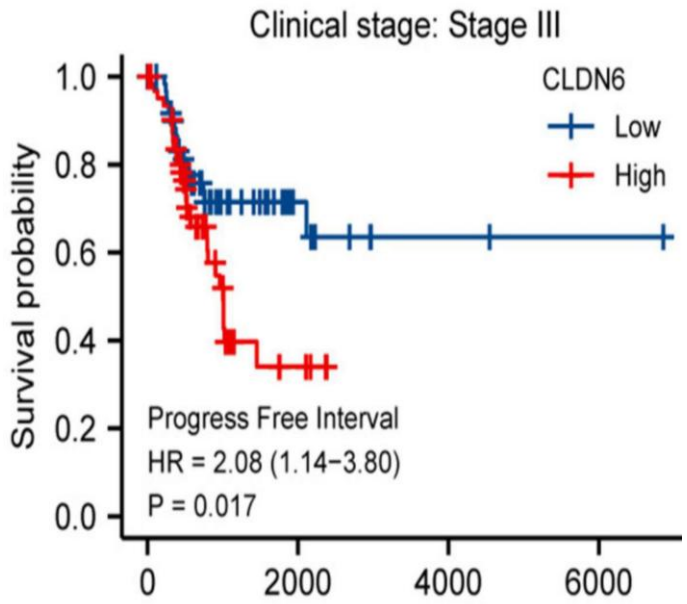
High CLDN6 Associated with a Worsened Prognosis in Cancer Patients

Endometrial Cancer[1]



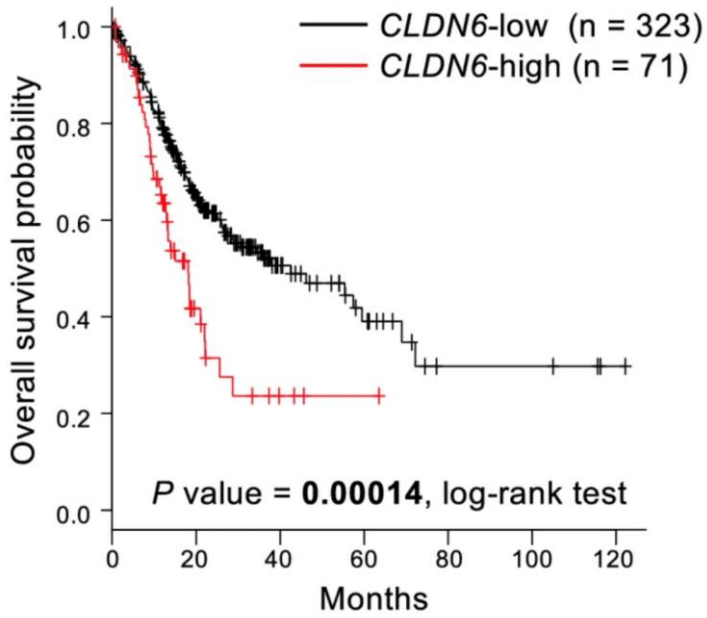
Overexpression of CLDN6 is associated with worse overall survival in endometrial cancer patients

Bladder Cancer[2]



Overexpression of CLDN6 is associated with worse overall survival and higher disease Stage (more aggressive) in bladder cancer patients

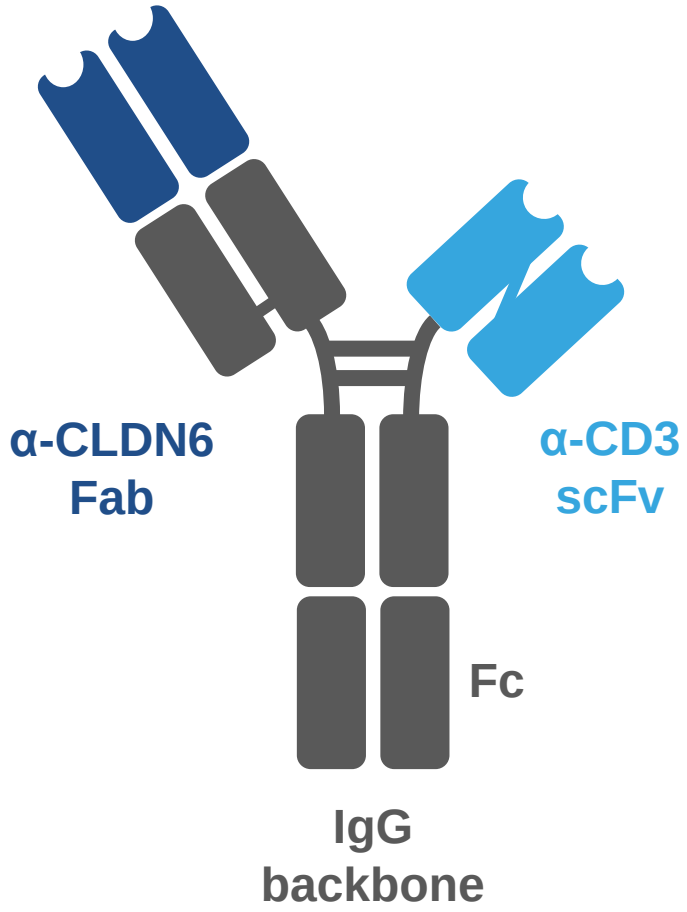
Stomach Cancer[3]



Overexpression of CLDN6 is associated with worse overall survival in stomach cancer patients

1 Kojima, Cancers, 2020
2 Zhang, Front. Cell Dev. Biol., 2021
3 Kohmoto, Gastric Cancer, 2020

CTIM-76: Claudin 6 x CD3 Bispecific Antibody



Wide therapeutic window

- Highly selective CLDN6 binding fragment antibody-binding (Fab) arm
- Immunostimulatory CD3 binding single-chain fragment variable (scFv) domain is designed to be functionally monovalent to avoid aberrant T-cell activation, potentially enhancing safety profile
- The fragment crystallizable region (Fc region) is the tail region of an antibody that interacts with cell surface receptors called Fc receptor. A mutation has been inserted into Fc domain to silence the Fc domain function and avoid T-cell activation by Fc-gamma receptor positive cells

Convenient dosing with low immunogenicity risk

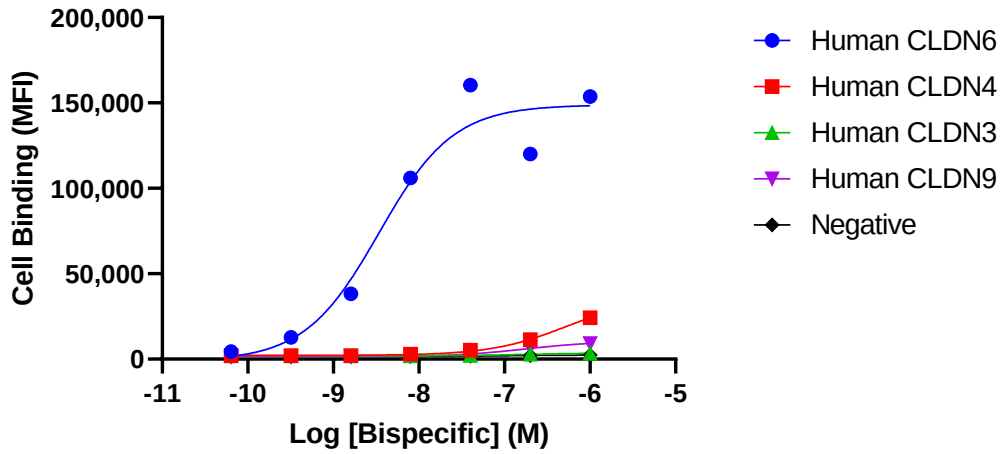
- T-cell dependent cellular cytotoxicity with no or minimal activation of circulating cytokines
- Humanized CLDN6 and CD3 binding domains

Ease of manufacturing

- IgG backbone is highly stable and enables high yield

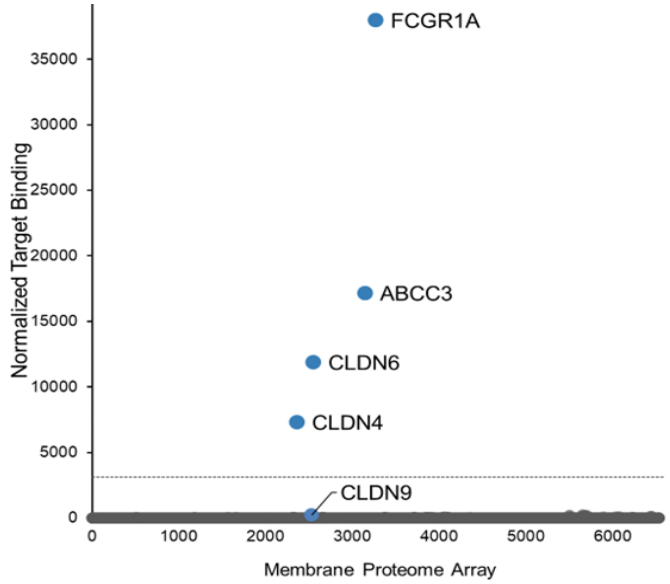
CTIM-76 Exhibits Excellent Selectivity and Specificity

CLDN6 Selectivity



- CTIM-76 **CLDN6 EC50 of 3.41 nM**
- CTIM-76 **preferentially binds** to CLDN6 over CLDN3/4/9
- CLDN3/4/6/9 were transiently transfected in HEK-293F cells (4:1 Target:GFP)

CLDN6 Specificity



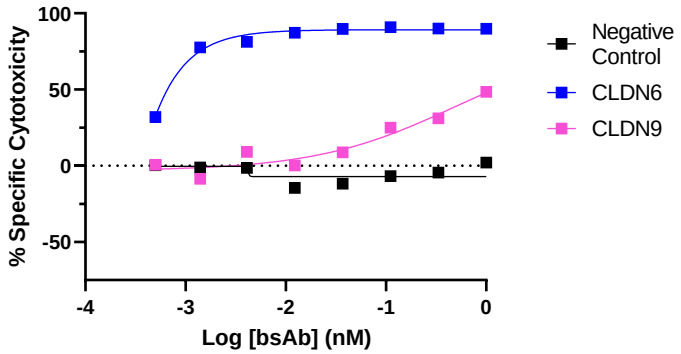
- IM301 Mab (CLDN6 Fab arm of CTIM-76) screened for specificity using Integral Molecular Membrane Proteome Array, consisting of **~6,000 human membrane proteins** in their native state in unfixed cells
- IM301 Mab was cross-reactive for internal control FCGR1A (Fc gamma) and nonrelevant intracellular epitope of ABCC3

CTIM-76 Preferentially Targets CLDN6 Over Other Claudin Family Proteins

- There is high sequence homology between CLDN6 and CLDN9 in the extracellular loops
- CTIM-76 preferentially targets CLDN6, with minimal activity against CLDN9-expressing cells
- No binding is observed to other CLDN family proteins (CLDN3 and CLDN4) that have <85% homology in the extracellular loops

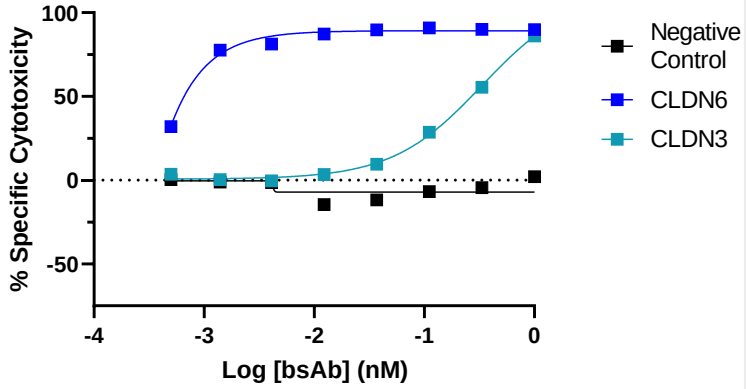
CLDN6:CLDN9

Activity Gap ~1192x



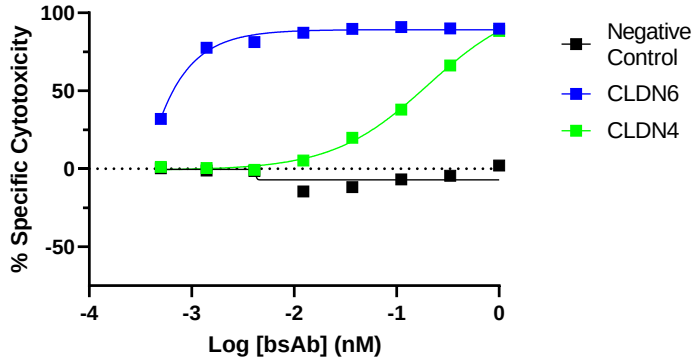
CLDN6:CLDN3

Activity Gap ~836x



CLDN6:CLDN4

Activity Gap ~504x



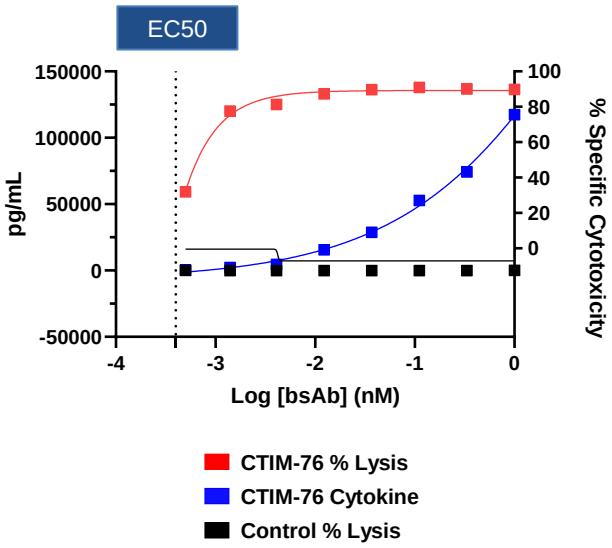
Study Design: K562 cells stably over-expressing CLDN3, CLDN4, CLDN6, or CLDN9 were co-cultured with human T-cells at an E:T ratio of 10:1 for 48 hours. Cytotoxicity was determined by luminescence imaging.

CTIM-76 has the Potential for a Wide Therapeutic Window

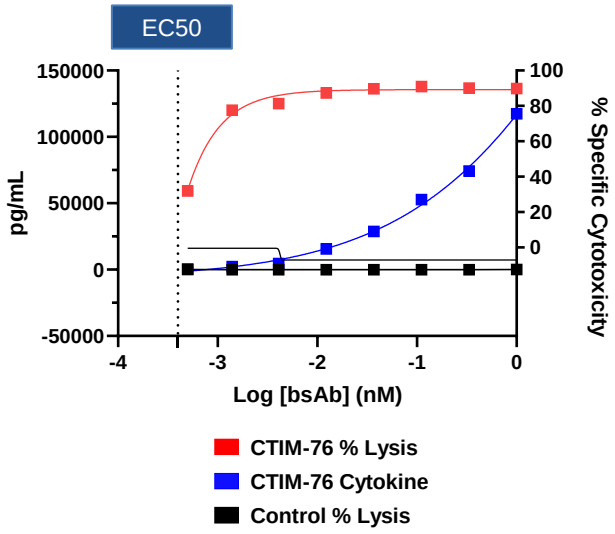
- Data supports potential to dose at levels that promote cancer cell killing but have manageable levels of free cytokine production, thereby potentially reducing the risk of cytokine release syndrome
- Cytokine production evaluated in exogenous (CLDN6-K562) cell line model at 48 hours
- Cytokine production happens well above the concentration of maximal killing (TDCC EC50 = 0.0004 nM)

Comparison of T cell-dependent cellular cytotoxicity (TDCC) to Cytokine Production

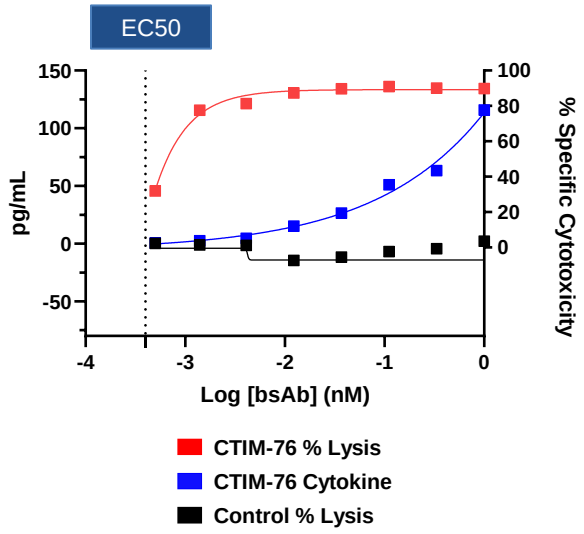
TDCC vs IFNg



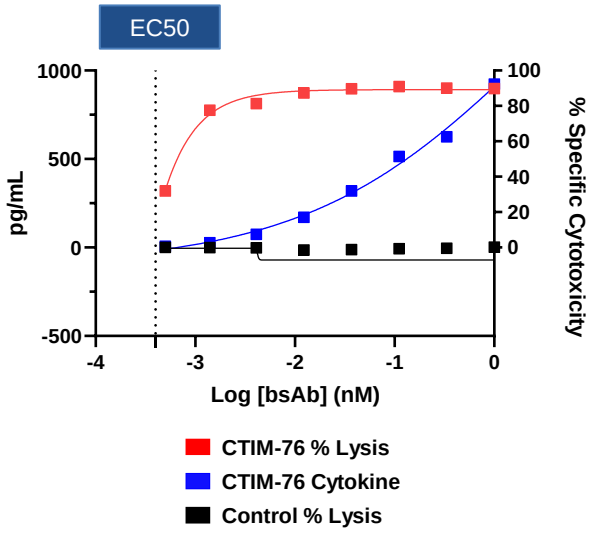
TDCC vs IL2



TDCC vs IL10



TDCC vs TNFα



Study Design: K562 cells stably over-expressing CLDN6 and luciferase were co-cultured with human T cells at an E:T ratio of 10:1 for 48 hours. Cytotoxicity was determined by luminescence imaging.

CLDN6 Competitive Landscape[1]

	Candidate	IND	Phase 1
Antibody Drug Conjugate (ADC)	**GB-7008-01** CLDN6/CLDN9 + MMAE **UCLA-23-ADC** CLDN6 + MMAE		**DS-9606a** CLDN6 + DXd
Bispecific Antibody	**NBL028** Undisclosed CLDN6x4IBB **Undisclosed** 2+1 bsAb CLDN6xCD3 **CTIM-76** bsAb CLDN6xCD3 **TJ-46CB** 2+2 bsAb CLDN6x4IBB		**AMG794** BiTE CLDN6xCD3 **BNT142** mRNA encoded BsAb CLDN6xCD3
Cell Therapy			**CAR-NK** CAR-NK + IL7 secreting vector **BNT211** CAR-T + CARVac

1 Analysis based on publicly available information compiled as of January 1, 2023

Clinical Experience for CLDN6 Therapies is Nascent

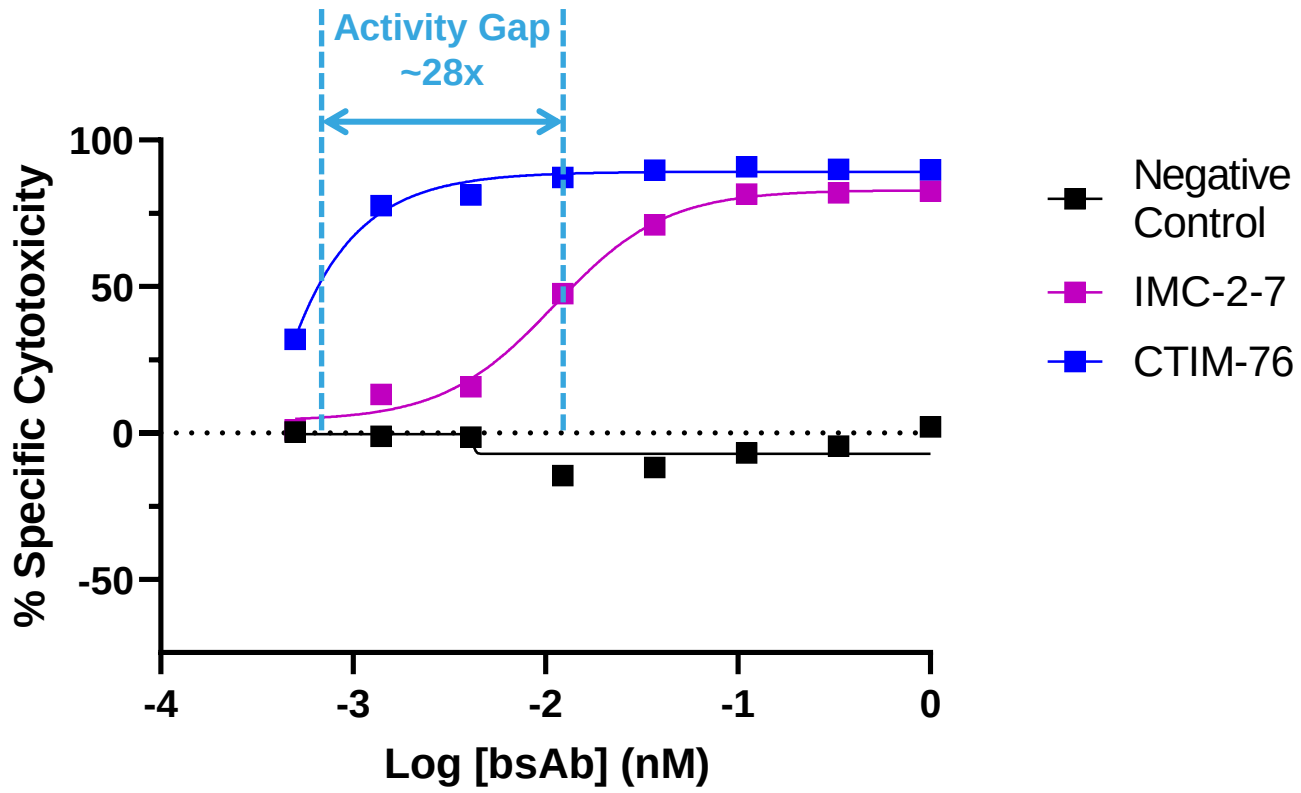
	Company	Program (Development Stage)	Description / Details[3]
Active Programs	BioNTech	**BNT211:** CLDN6CAR-T + CARVac (Phase 1)	Initial data for BNT211 were presented April 2022 (AACR), with an update in Sept 2022 (ESMO). Received PRIME Designation for testicular cancer June 2022
		BNT142: CLDN6 mRNA encoded bsAb (Phase 1)	Initiated Phase 1 development for BNT142 in mid-2022
	Amgen	**AMG794:** CLDN6 BiTE (Phase 1)	AMG794 candidate were presented April 2022 (AACR), trial is not yet recruiting
	Guangzhou Medical University	**Undisclosed:** CAR-NK + multiple gene edits (Phase 1)	Engineered to express IL7/CCL19 and/or SCFVs against PD1/CTLA4/Lag3, initiated Phase 1 development in mid-2022
	Daiichi	**DS-9606a:** CLDN6 + DXd (Phase 1)	Initiated Phase 1 development for DS-9606a in mid-2022
	I-Mab	**TJ-46CB:** CLDN6 bsAb CLDN6x4IBB (Preclinical)	Initial data were presented April 2021 (AACR), IND filing is expected in 2H 2023
	Xencor	**Undisclosed:** CLDN6 bsAb CLDN6xCD3 (Preclinical)	Initial data were presented April 2021 (AACR), no timeline to IND provided
Notable Deprioritized Programs	**Astellas/Ganymed**	**IMAB027/ASP1650:** CLDN6 mAb (Phase 2)	Lack of single agent activity in Phase 2 trial relapse/refractory testicular germ cell tumors[1]
	Abbvie/Stemcentryx	**SC004:** CLDN6/9 ADC (Phase 1)	Dose-limiting toxicity observed in Phase 1 in patients with ovarian cancer, potentially attributed to CLDN9 binding[2]

1 Adra, Invest New Drugs, 2022
2 Hamilton, Cancer Res, 2020

3 Analysis based on publicly available information compiled as of January 1, 2023

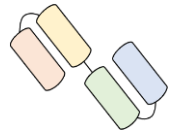
Role of Bispecific Format in Activity

CTIM-76 format demonstrates superior potency compared to a traditional BiTE molecule (e.g., AMG-794)

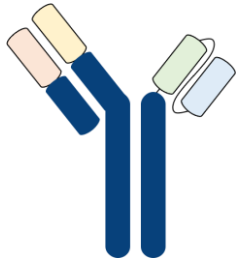


CTIM-76 vs BiTE

IMC-2-7
Tandem scFvs (BiTE)



CTIM-76
scFv-Fab IgG



Study Design: K562 cells stably expressing CLDN6 and luciferase were co-cultured with human T-cells at an E:T ratio of 10:1 for 48 hours. Cytotoxicity was determined by luminescence imaging.

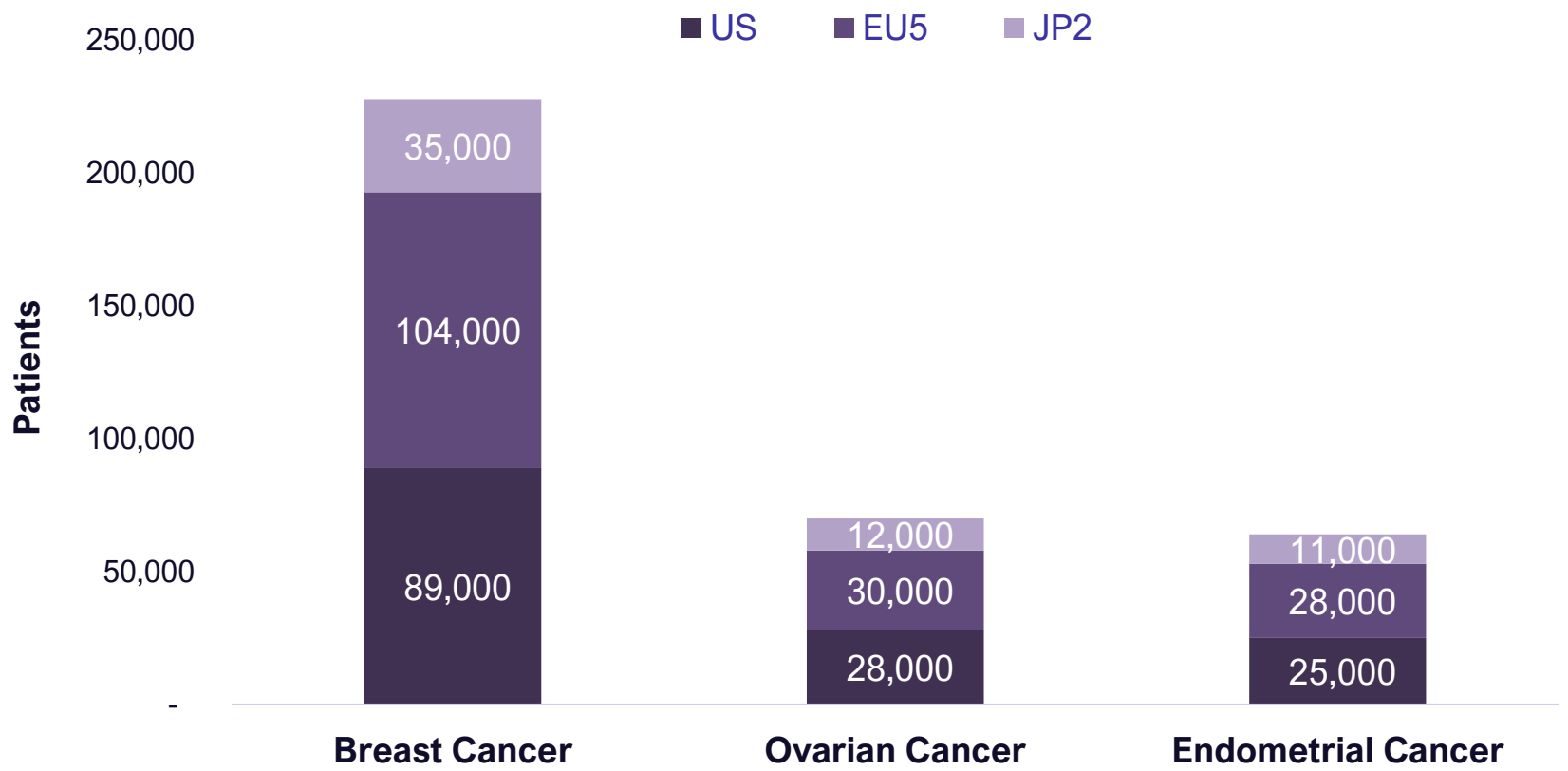
ONA-XR

Progesterone Receptor Antagonist for Female Cancers

Unmet Need in Female Cancers

Prevalence of Metastatic Female Cancers in EU5, Japan, and US

Within the G7 countries, it is estimated that there are over 355,000 patients living with metastatic breast, ovarian, or endometrial cancer



Source: secondary epidemiologic estimates, 2020 estimates

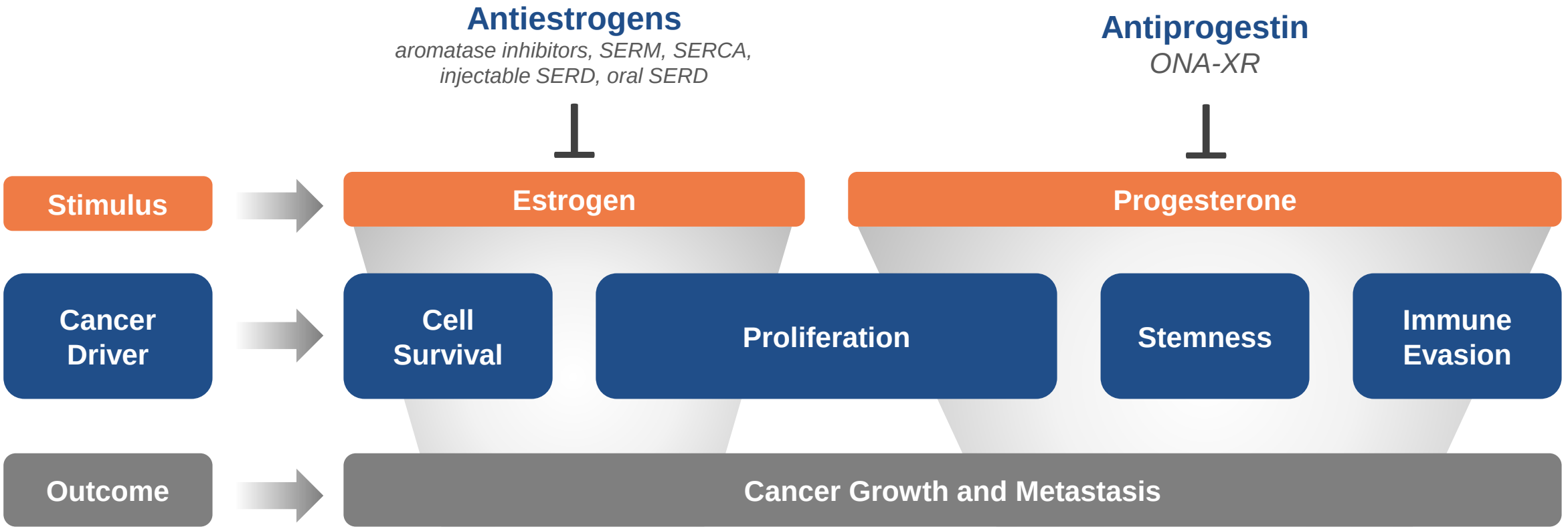
Onapristone Extended Release (ONA-XR)

- Progesterone Receptor (PR) oncogenic signaling is associated with female cancers and is a potential resistance mechanism to standard of care treatments including antiestrogens and CDK4/6 inhibitors
- Onapristone (ONA) is a progesterone receptor (PR) antagonist that suppresses PR oncogenic signaling
- Over 150 patients treated to date across female cancers
- Encouraging Phase 2 clinical data in ongoing breast (SMILE) and endometrial (OATH) cancer trials

Most Complete PR Antagonist	Easy Administration	Minimal Side Effects	Broad Activity	CNS Activity
Blocks both ligand-dependent and ligand-independent PR activation	Attractive pharmacokinetic profile; 50 mg orally administered at morning and night with or without food	Favorable clinical tolerability and safety as monotherapy and in combination with antiestrogens (anastrozole, fulvestrant)	Meaningful antitumor activity in both wild-type and mutant (*ESR1*, *PIK3CA*) preclinical models and durable benefit in clinical settings	Brain metastases are common with breast cancer; ONA-XR is CNS penetrant with demonstrated activity in nonclinical meningioma studies

Mechanism of Action

- Antiestrogen therapy is the backbone treatment for female cancers, whereas there are no FDA-approved antiprogestin therapies approved for cancer
- Estrogen and progesterone play unique roles in regulating the drivers of cancer growth and spread
- Combining antiestrogen and antiprogestin therapy may more completely block cancer drivers and ultimately improve patient outcomes



Completed Clinical Trials

Summary of select clinical trials evaluating onapristone with immediate (IR) or extended release (XR) formulation

Onapristone Treatment	Stage	Patients (n)	Clinical Indication	Prior Treatments Median (range)	Biomarker	Data
IR (100 mg QD)	Ph 2	19	Breast Cancer First line (1L) advanced or metastatic	Hormone naïve		**56% ORR[1]** **67% CBR** **14.0 month PFS**
IR (100 mg QD)	Ph 2	101	Breast Cancer Second line (2L) advanced or metastatic	1 (1-2)		**10% ORR[2]** **48% CBR** **4.0 month PFS**
XR (50 mg BID)	Ph 2	14	Granulosa Cell Tumor of Ovary Advanced or Metastatic	4 (2-17)	PR+	**35% CBR[3]** **12 month PFS rate of 20%**
XR (10-50 mg BID)	Ph 1	13	Ovarian Cancer Advanced or Metastatic	4 (2-10)	PR+	**8% ORR[4]** **6 month PFS rate of 31%**
XR (10-50 mg BID)	Ph 1	20	Breast Cancer Advanced or Metastatic	9 (2-14)	PR+	**25% DCR[4]** **6 month PFS rate of 15%**

IR = immediate release; XR = extended release

1 Robertson, Eur J Cancer, 1999

2 Jonat, Endocrine Therapy of Breast Cancer, 2002

3 Grisham, ASCO Annual Meeting 2022

4 Cottu, PLoS One, 2018

Key Ongoing Clinical Trials

Treatment	Clinical Indication	Stage	Patients (n)	Key Inclusion and Exclusion Criteria	Ongoing Trial Data	Completed Trials / Historical Data[2]
ONA-XR + Anastrozole[1]	Endometrial Cancer OATH Trial	Ph 2	25	• Must have received at least one prior treatment with a platinum plus taxane chemotherapy	• **12 patients enrolled** • **4-month PFS rate of 77%** • **12-month PFS rate of 33%** • **No treatment-related SAE**	Chemotherapy (KEYNOTE-775)[3] • 3.8 month PFS • 72% Grade 3 or higher AE Anastrozole (PARAGON)[4] • 2.8 month PFS
ONA-XR + Fulvestrant[1]	Breast Cancer (2L/3L) SMILE Trial	Ph 2	39	• Must have received prior CDK4/6 inhibitor therapy • One line of prior chemotherapy in metastatic setting allowed	• **10 patients enrolled** • **4-month PFS rate of 44%** • **No treatment-related SAE**	Elacestrant (EMERALD)[6] • 2.8 month PFS Fulvestrant (EMERALD)[6] • 1.9 month PFS
ONA-XR + Elacestrant	Breast Cancer (2L/3L) ELONA Trial	Ph 1b/2	67	• Must have received prior CDK4/6 inhibitor therapy • ≥50% patients with ESR1 mutant • No prior chemotherapy in metastatic setting	• **Initiated Q4 2022** • **First patient enrolled Jan 2023** • **Ph 1b data expected Q4 2023**	Elacestrant (EMERALD)[6] • 2.8 month PFS Fulvestrant (EMERALD)[6] • 1.9 month PFS

1 Data cut off as of September 30, 2022; preliminary raw data
2 Analysis based upon publicly available information and represents a non-head-to-head summary comparison
3 Makker, NEJM, 2022
4 Mileshkin, Gyn Onc, 2019
5 Grisham, ASCO 2022
6 Bidard, JCO, 2022

ONA-XR

Recurrent PR+ Endometrial Cancer

Endometrial Cancer

- **Endometrial cancer is the 4th most common cancer in women**
  - Endometrial cancer is on the rise and is linked to obesity[1,2]
  - 12,500 patient deaths per year in the US[3]
  - Market is projected to grow from $1.5bn in 2020 to $5.1bn in 2029[5]
- **Hormone signaling is a driver of endometrial cancer**
  - Endometrial cancer is thought to be caused by excess hormone production that leads to endometrial hyperplasia and cancer
- **Chemotherapy and surgery remain first-line treatments**
  - First-line treatment includes surgical removal of uterus, ovaries, and fallopian tubes followed by platinum/taxane chemotherapy
  - PD-1 antibodies (Keytruda, Jemperli) were recently approved in MSI-H and dMMR genetic subpopulations post-chemotherapy (~13-30% of population)[5]
  - Lenvima + Keytruda combination therapy is approved post-chemotherapy, however, tolerability can be challenging for patients[6]
- **Antiestrogen therapy is currently used off-label**
  - Hormonal therapy is an alternative treatment for patients who wish to preserve their fertility, and for those with metastatic or recurrent disease without curative options

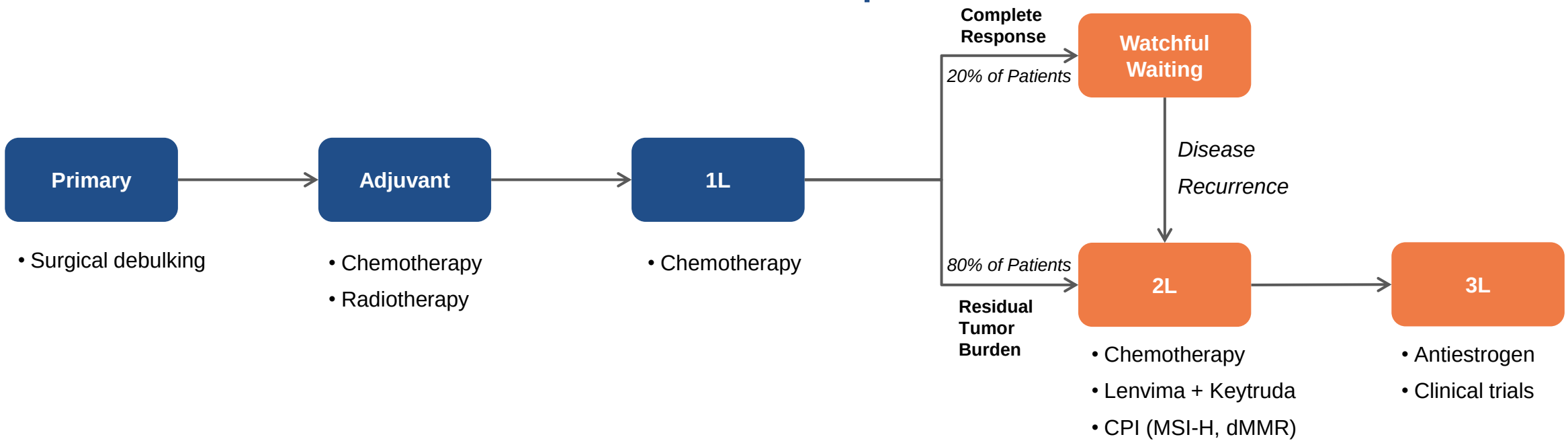
12,500 patients have recurrent endometrial cancer that cannot be fully removed via surgery [2]

34%

of endometrial cancer patients are PR+[4]

1 American Cancer Society, Endometrial Cancer Risk Factors. (accessed Nov. 4, 2022)
2 Epic Oncology (Incidence, 1st/ 2nd line treated); epic Oncology physician survey 2019
3 Nation Cancer Institute, Endometrial Cancer Incidence Rising in the US and Worldwide (accessed Nov. 4, 2022)
4 Høgdall, Oncol Rep, 2007
5 Vinuesa and Webster, Nat Rev Drug Disc, 2022
6 Makker, NEJM, 2022

Endometrial Cancer Patient Treatment Landscape[1]



1L First Line (1L)

- Standard of Care (SOC) is carboplatin + paclitaxel
- mPFS of ~12 months

2L Second Line (2L)

- Treatment goal is disease stabilization for 4-6 months
- Lenvima + Keytruda poor tolerability associated with high discontinuation rate in patients

M Maintenance Line

- No approved therapies
- Treatment goal is disease stabilization for ≥4 months and to provide a high quality of life

3L Third Line (3L)

- Limited treatment options

Potential Target Indications for ONA-XR

1 National Comprehensive Cancer Network Clinical Practice Guidelines in Oncology. Uterine Neoplasms. v4.2021

ONA-XR + Anastrozole in PR+ Endometrial Cancer[1]

- **Ongoing Phase 2 Trial**
  - Investigator-initiated, open-label, multi-center trial (the “OATH” trial) evaluating ONA-XR 50 mg BID in combination with the antiestrogen anastrozole 1 mg QD administered orally to treat women with ER+/PR+ endometrial adenocarcinoma who have received at least one prior platinum/taxane-based chemotherapy regimen
  - Co-primary endpoints: 4-month PFS and ORR
  - Secondary endpoints: DCR, DoR, safety, and quality of life

- **Efficacy**
  - The study has enrolled 12 of 25 planned patients
  - 9 evaluable patients; completed at least one month of treatment
  - 4-month PFS rate was 77%
  - 12-month PFS rate was 33%
  - 7 patients remain on the trial

- **Safety**
  - There have been no treatment-related serious adverse events reported

- **Updated data anticipated in mid-2023**

Treatment Goal in Endometrial Cancer

We seek to deliver chemotherapy-like clinical activity without debilitating toxicity

1 Data cut off as of September 30, 2022; preliminary raw data

Preliminary Data vs Historical Trials[1]

	ONA-XR + Anastrozole	ONA-XR	Anastrozole	Chemotherapy
Trial	OATH (ongoing)	Cottu 2018[2]	PARAGON 2019[3]	KEYNOTE-775 2022[4]
Patients (n)	12 (9 evaluable)	12	54	416
Lines of Prior Chemotherapy, n (%) **1** **≥2**	 8 (66) 4 (33)	 4 (33) 8 (66)	 50 (93) 4 (7)	 277 (67) 139 (33)
Treatment free interval (TFI) ≥6 months, n (%)	4 (33)	1 (8)	36 (70)	ND
4-month PFS rate, n (%)	7 (77)	4 (33)	ND	ND
12-month PFS rate, n (%)	3 (33)	1 (8)	4 (7)	18 (4)
mPFS (95% CI), months	NE	2.0 (1.7-5.3)	2.7 (1.9-4.5)	3.8 (3.6-4.2)
Side Effects	Well tolerated	Well tolerated	Well tolerated	72% experienced Grade 3 or higher AE

1 Analysis based upon publicly available information and represents a non-head-to-head summary comparison. Data cut off as of September 30, 2022; preliminary raw data

2 Cottu, PLoS One, 2018
3 Mileshkin, Gyn Onc, 2019
4 Makker, NEJM, 2022

ONA-XR

2L/3L Metastatic Breast Cancer

Hormone Receptor-positive Breast Cancer

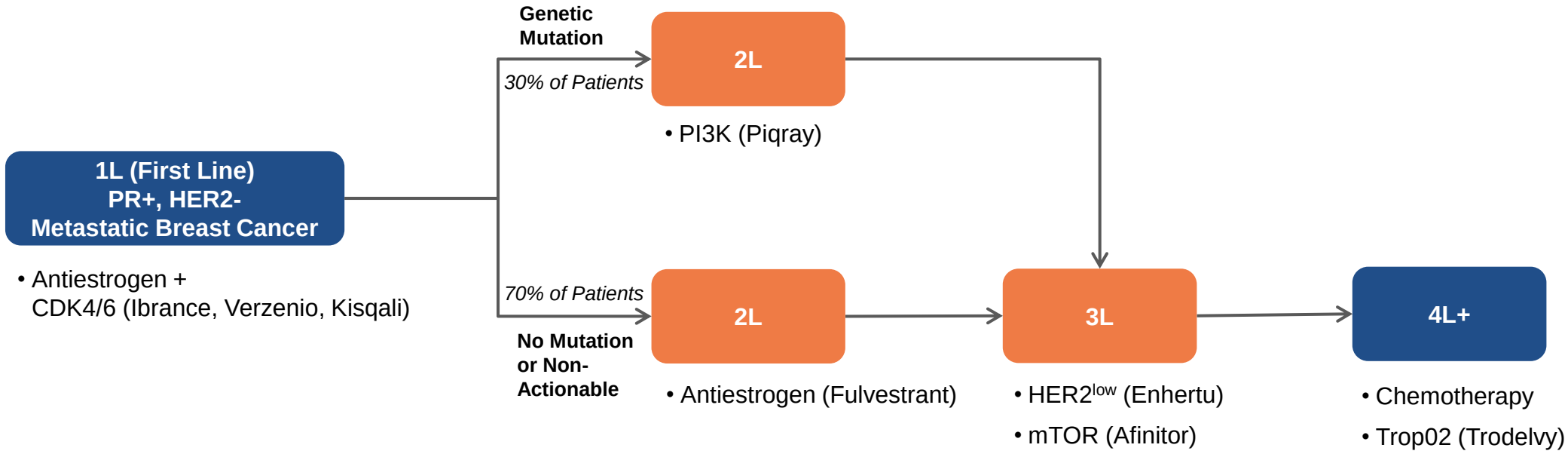
- **Breast Cancer is the 2nd most common cancer in women**
  - 2L/3L+ therapy, could represent a $3-5B U.S. segment of $20B endocrine therapy market
  - Potential to expand ONA-XR into earlier treatment lines
- **Hormone signaling is a driver of breast cancer**
  - Approximately 75% of breast cancer patients have hormone-driven, also known as hormone receptor-positive, breast cancer
  - For these patients, antiestrogen therapy is the backbone treatment due to excellent tolerability
- **Antiestrogen + CDK4/6 inhibitor is first-line treatment in metastatic setting**
  - Patients generally respond well to this treatment and are often on therapy for years
  - Upon first-line relapse (i.e., second-line), there are limited FDA approved treatment options for patients
- **Second and third-line metastatic hormone-driven breast cancer is evolving**
  - Potential FDA approvals in 2023 for new antiestrogen (elacestrant) and pan-AKT inhibitor (capivasertib)

~43,250 women succumb to metastatic breast cancer in the United States per year

~75%

of breast cancer patients are progesterone receptor positive (PR+)

ONA-XR has the Potential to be Used Across Many Lines of Treatment[1]



1L First Line

- Standard of Care (SOC) is CDK4/6 inhibitor plus antiestrogen
- mPFS of 14-18 months

2L Second Line

- Treatment goal is disease stabilization for >4 months
- Potential new treatments pending FDA approval include antiestrogen (elacestrant) and AKT inhibitor (capivasertib)

3L Third Line

- mTOR associated with significant side effects
- Enhertu anticipated to be approved in HER2^{low} and/or HER2-

Initial Target Indications for ONA-XR

4L Fourth Line

- Limited treatment options

1 National Comprehensive Cancer Network Clinical Practice Guidelines in Oncology. Uterine Neoplasms. v4.2021

Our Development Strategy in 2L/3L HR+,HER2- Metastatic Breast Cancer

- Estimated 115,000 treatment eligible patients in United States and EU5
- Unmet need for new therapies that can improve antiestrogen response after CDK4/6 inhibitor therapy without adding significant toxicity
- New treatments, including antibody drug-conjugate (Enhertu) therapy, expected to be used once patients are no longer responsive to antihormonal therapy

Ongoing Context Clinical Trials in Post-CDK4/6 Treatment Line

	SMILE Trial	ELONA Trial
Patients (n)	39	67
Indication	2L/3L ER+,HER2- mBCa	2L/3L ER+,PR+, HER2- mBCa
Treatment	ONA-XR + fulvestrant	ONA-XR + elacestrant
Key Inclusion / Exclusion	• Must have received prior CDK4/6 inhibitor therapy • One line of prior chemotherapy in metastatic setting allowed	• Must have received prior CDK4/6 inhibitor therapy • ≥50% patients with ESR1 mutant • No prior chemotherapy in metastatic setting
Next Expected Data Milestone	Q4 2023	Q4 2023

Preliminary Data vs Historical Trials[1]

	ONA-XR + Fulvestrant	Fulvestrant	Elacestrant
Trial	SMILE (ongoing)[2]	EMERALD 2021[3]	EMERALD 2021[3]
Patients (n)	10 (9 evaluable)	238	239
Key Demographics			
Prior CDK4/6 inhibitor	100%	100%	100%
ESR1 mutation	ND	48%	47%
4-month PFS rate, %	44.4	NE	NE
mPFS, months	Trial ongoing	1.91	2.79
Side Effects	Well tolerated	Well tolerated	Well tolerated

1 Analysis based upon publicly available information and represents a non-head-to-head summary comparison. Data cut off as of September 30, 2022; preliminary raw data

2 Kamaraju, SABCS 2022

3 Bardia, SABCS 2021

Corporate

Experienced Leadership Team

Martin Lehr
CEO and Director

Praesidia Biologics
Cure Duchenne
Osage University Partners

Jennifer Minai, CPA
Chief Financial Officer

Millendo Therapeutics
parexel
EY

Chris Beck, MBA
SVP Operations

Galera Therapeutics
Shire
Merck

Alex Levit, Esq
Chief Legal Officer

optinose
teva
ReedSmith

Tarek Sahmoud, MD, PhD
Chief Medical Officer

Novartis
H3 Biomedicine
Celgene

Priya Marreddy, MS
VP Clinical Operations

Spark Therapeutics
teva
PRA Health Sciences

Focus on Execution

Experienced team with deep oncology experience

Our CMO led the clinical development of multiple blockbuster drugs for female cancers, including Kisqali, Arimidex, and Afinitor

Our management team is supported by a Board with strong public company operating and governance experience

Investment Highlights (Nasdaq: CNTX)

Large Unmet Need

Solid Tumors, Primary Focus on Female Cancers

High-Value Targets

Progesterone Receptor and Claudin 6

Near-Term Milestones

Multiple Data Readouts in 2023

Strong Team

Deep Domain Experience, Track Record of Success

Financial Strength

Expected Cash Runway into Q1 2024

context therapeutics

Advancing Medicines For Female Cancers and Other Solid Tumors

Abbreviations

Clinical Trial Efficacy	
CBR (CR+PR+ SD ≥6 mos)	Clinical benefit rate
CR	Complete response
DCR (CR+PR+ SD)	Disease control rate
DoR	Duration of response
mPFS	Median PFS
ORR (CR+PR)	Overall response rate
PFS	Progression free survival
SD	Stable disease
95% CI	95% confidence interval

Clinical Trial Safety	
AE	Adverse event
DLT	Dose-limiting toxicity
TRAE	Treatment-related adverse event
SAE	Serious adverse event

Diseases	
BC	Breast cancer
GCT	Granulosa cell tumor
NSCLC	Non-small cell lung cancer
SCLC	Small cell lung cancer

Other Terms	
1L	First Line
2L	Second Line
BID	Twice per day
CPI	Checkpoint inhibitor
dMMR	DNA mismatch repair
ER	Estrogen receptor
mAb	Monoclonal antibody
MSI-H	Microsatellite instability high
ND	Not determined
NE	Not evaluable
PK	Pharmacokinetics
PR	Progesterone receptor
QD	Once per day
QOL	Quality of life
SOC	Standard of care
TFI	Treatment free interval

Approved Drugs Mentioned	
Jemperli	Dostarlimab-gxly (GSK)
Lenvima	Lenvatinib (Eisai)
Keytruda	Pembrolizumab (Merck)

Medical Organizations / Conferences	
AACR	American Association for Cancer Research
ASCO	American Society of Clinical Oncology
ESMO	European Society for Medical Oncology
SABCS	San Antonio Breast Cancer Symposium