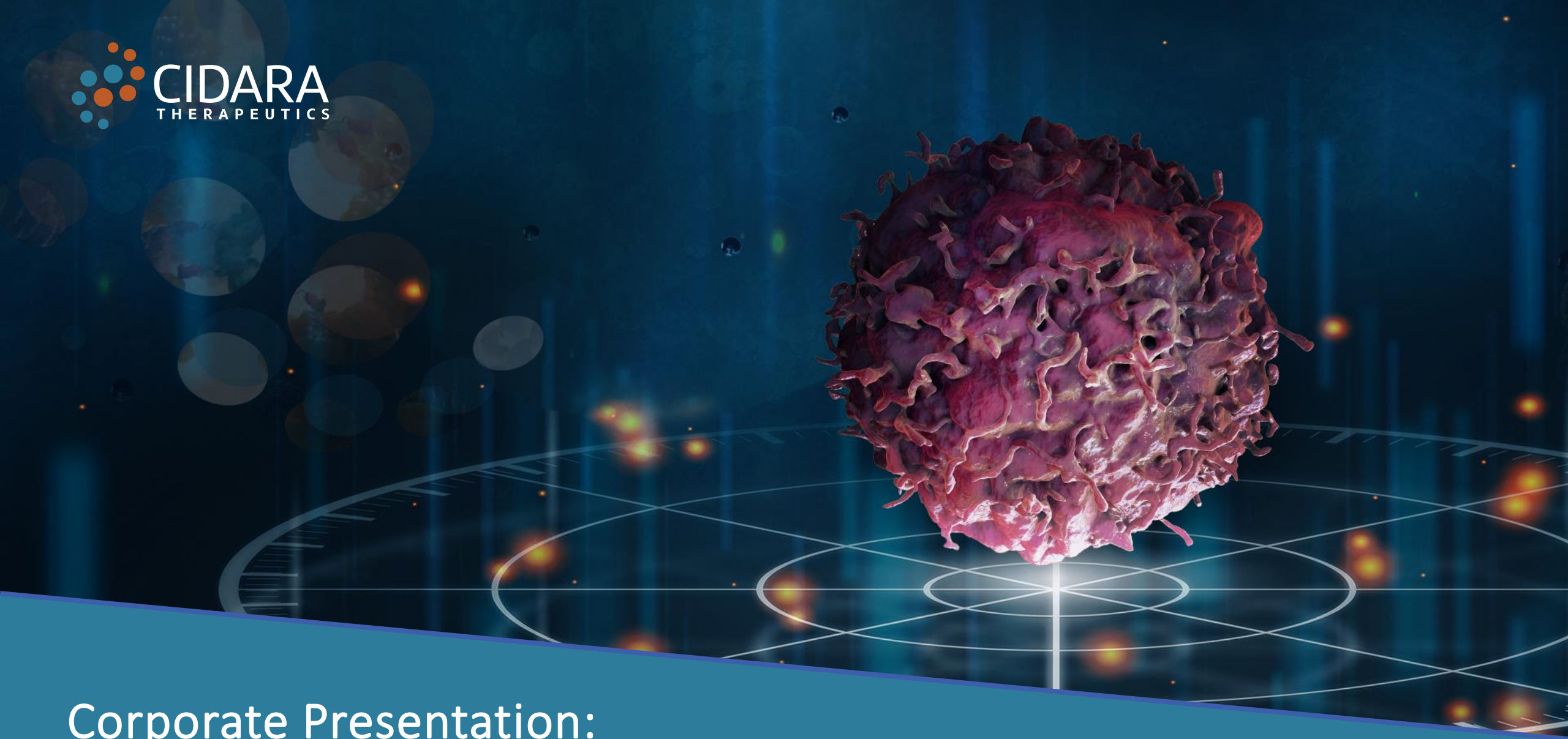


Corporate Presentation:

May 2024
NASDAQ: CDTX

Forward-Looking Statements

These slides contain forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995.

The words “may,” “will,” “estimate,” “plan”, “anticipate,” “expect,” “potential,” “could,” “project,” and similar expressions (including the negative thereof), are intended to identify forward-looking statements. Because such statements are subject to risks and uncertainties, actual results may differ materially from those expressed or implied by such forward-looking statements. Such statements include, but are not limited to, statements regarding Cidara’s research and development efforts; preclinical and clinical development activities; plans, projections and expectations for and the potential effectiveness, safety and benefits of, its product candidates, including CD388, CBO421 and other antiviral and oncology product candidates from the Cloudbreak platform; whether CD388 may have significant advantages beyond and in addition to flu vaccines; and advancement of its strategic plans

Projections, assumptions and estimates of the future performance of the markets in which Cidara operates are necessarily subject to a high degree of uncertainty and risk, including, Cidara's ability to obtain additional financing; the success and timing of Cidara’s preclinical studies, clinical trials and other research and development activities; receipt of necessary regulatory approvals for development, as well as changes to applicable regulatory laws in the United States and foreign countries; changes in Cidara's plans to develop its product candidates; Cidara's ability to obtain and maintain intellectual property protection for its product candidates; and the loss of key scientific or management personnel. These and other risks and uncertainties are described more fully in Cidara's Annual Report on Form 10-K for the fiscal year ended December 31, 2023 filed with the United States Securities and Exchange Commission ("SEC“’) on April 22, 2024, and in Cidara’s other filings with the SEC.

Additional risks and uncertainties may emerge from time to time, and it is not possible for Cidara’s management to predict all risk factors and uncertainties. Cidara cautions that the foregoing list of factors is not exclusive and not to place undue reliance upon any forward-looking statements which speak only as of the date of this presentation. Except as required by law, Cidara does not undertake any obligation to update publicly any forward-looking statements for any reason after the date of this presentation to conform these statements to actual results or to changes in its expectations.

CIDARA THERAPEUTICS

CD388 Reacquired from Janssen Pharmaceuticals

Cidara Therapeutics Reacquires Global Development and Commercial Rights to CD388 and Announces Private Placement Financing of $240 Million

- *$240 million private placement financing led by RA Capital Management with significant participation by Bain Capital Life Sciences as well as BVF Partners and Canaan Partners to fund Phase 2b clinical trial*
- *CD388, which is active against all strains of influenza A and B, is being developed for pre-exposure prophylactic treatment*

Rezafungin Divested to Mundipharma*

Approximately **$67M** in clinical development and CMC costs over next three years

Approximately **$61M** in forecasted obligations through the patent life of rezafungin

Cost savings to be applied to advancing CD388 and other Cloudbreak development programs, including CBO421

*Napp Pharmaceutical Group, an affiliate of Mundipharma.

CIDARA THERAPEUTICS

The New Cidara – Cloudbreak® Drug Fc Conjugate (DFC)Programs

CLOUDBREAK

- CD388; Phase 2a completed
- CD73 development candidate
- Dual inhibitor program
- Chemokine receptor program

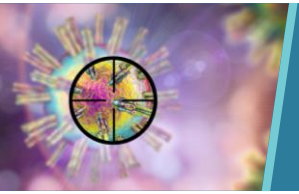
Program	Indication	Discovery	Preclinical	IND-enab.	Phase 1	Phase 2
Viral Neuraminidase	Prevention of Seasonal Influenza	CD388				
CD73	Solid Tumors, TNBC[1]		CBO421			
PD-1/ CD73	Solid Tumors					
CCR5	Solid Tumors					

Note: Additional programs in preclinical development not disclosed

1. TNBC = Triple Negative Breast Cancer

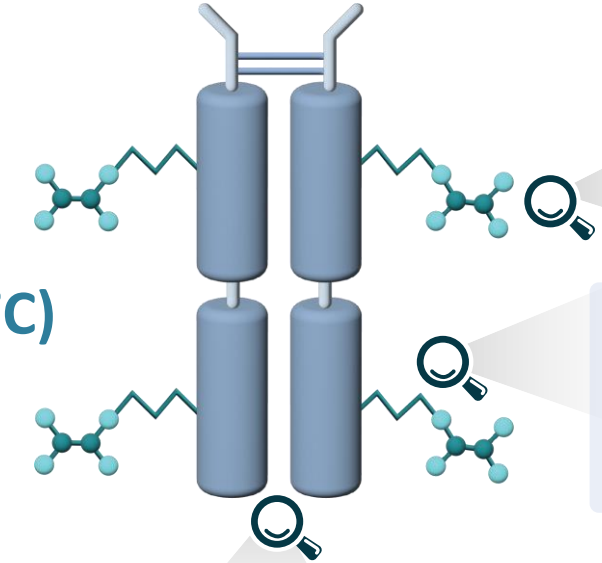
CIDARA THERAPEUTICS

Cloudbreak DFCs: A Modular Platform With Broad Applications Across Multiple Therapeutic Areas



A uniquely tunable platform that combines and expands upon the strengths of small molecule (SM) and antibody therapeutics

DRUG Fc CONJUGATE (DFC)



Small molecule Targeting Moiety (TM)

All the strengths of mAbs with several potential advantages

- *Efficient targeting of cryptic sites and small molecule receptors*
- *Tunable valency to exploit avidity for improved potency*
- *Multiple routes to low molecular weight multispecific agents*

Non-cleavable linker

No intracellular exposure = Superior safety vs small molecule drugs

- *Greater freedom to optimize TMs for target potency*
- *Potential to inhibit "undruggable" targets*

Proprietary hIgG1 CH1-Fc hybrid domain

Multiple tunable attributes

- *Immune effector function*
- *Half-life extension*

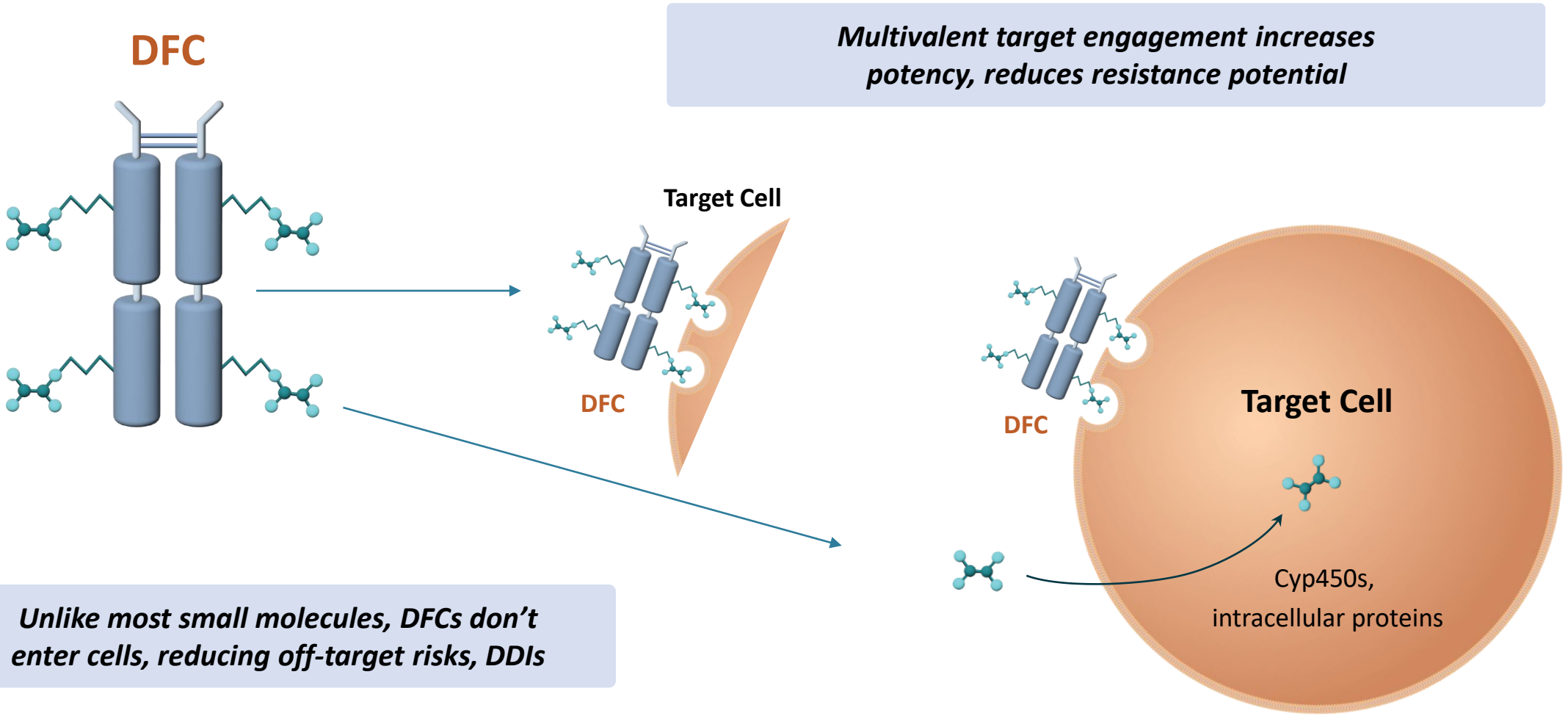
2.5x smaller than mAbs

- *Superior tissue/tumor penetration*

mAbs = Monoclonal Antibodies

CIDARA THERAPEUTICS

DFCs May Improve Small Molecule Drug Potency and Safety



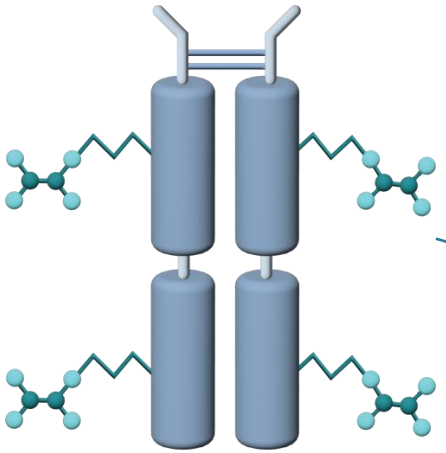
DDIs = Drug-drug Interactions

DFCs Have Reduced Potential for Off-Target Toxicity

DFCs can exploit drug targets that are difficult to drug with small molecules (SMs)

Many SM programs have failed to advance due to intracellular off-target toxicity (e.g., hepatotoxicity)*

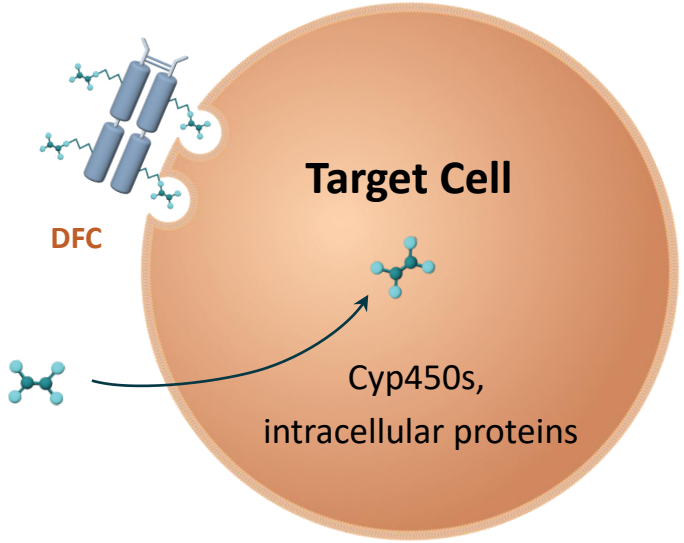
DFC



DFCs do not enter cells

DFCs exhibit better safety than SMs

Cardiovascular toxicity due to hERG inhibition has impaired clinical development in many SM programs*



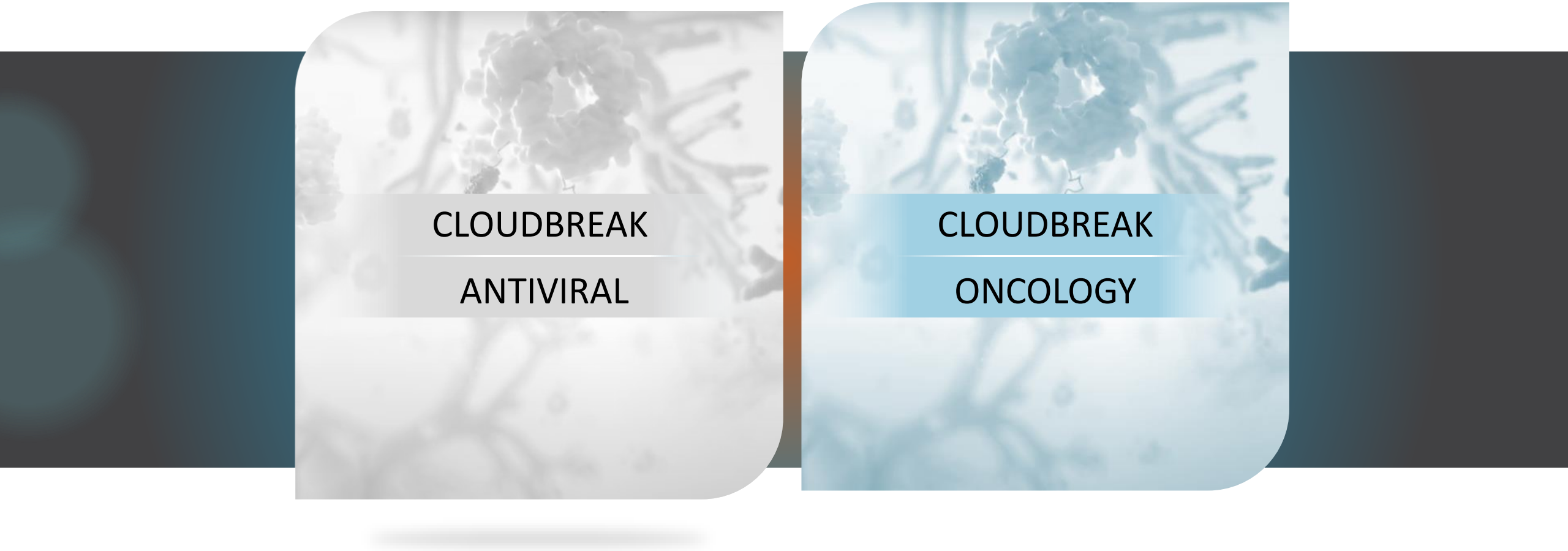
% hERG inhibition @10mM	
SM	77
DFC	< 5

*Kim, et. Al., "CCR5 receptor antagonists in preclinical to phase II clinical development for treatment of HIV". Expert Opinion on Investigational Drugs, 2016 VOL. 25, NO. 12, 1377–1392. http://dx.doi.org/10.1080/13543784.2016.1254615

hERG = human ether-a-go-go-related gene

CIDARA THERAPEUTICS

Cidara's Pipeline Targets Multiple Unmet Medical Needs

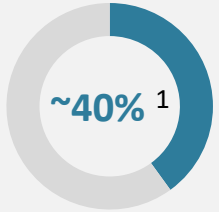


CIDARA THERAPEUTICS

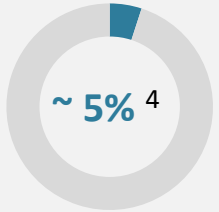
The Problem: A "Universal" Flu Vaccine Does not Exist

Influenza worldwide death rates >650,000 per year [2]

Low vaccine effectiveness



General population
(5-year flu season average) [1]



Immunocompromised [4]

Antigenic drift

Few viable programs in development

2020-2024 Flu season average [3]

Co-morbidities at higher risk of hospitalization:

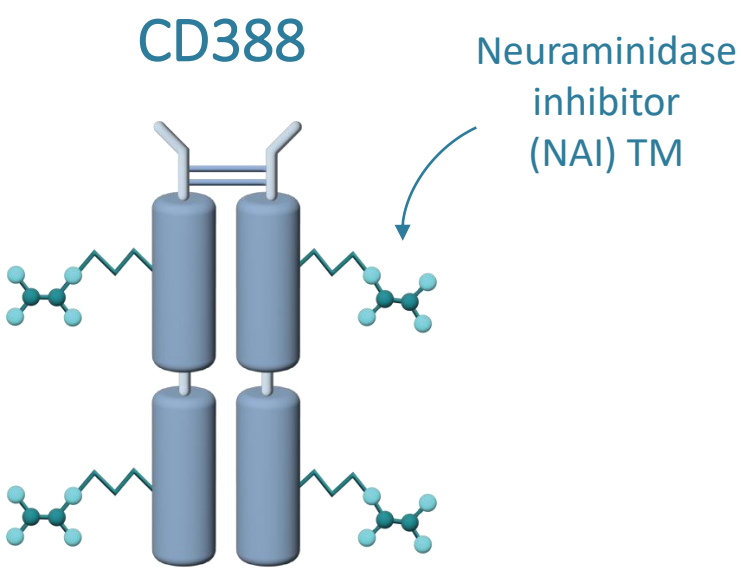
- **Chronic lung disease:** 9-fold increase
- **Kidney disease:** 5-fold increase
- **Cardiovascular Disease:** 12-fold increase

1. https://www.cdc.gov/flu/vaccines-work/past-seasons-estimates.html
2. Estimates of global seasonal influenza-associated respiratory mortality: a modelling study. Lancet. 2018 Mar 31;391(10127):1285-1300.
3. Laboratory-Confirmed Influenza Hospitalizations (cdc.gov)
4. Hughes K, Middleton DB, Nowalk MP, et al. Effectiveness of Influenza Vaccine for Preventing Laboratory-Confirmed Influenza Hospitalizations in Immunocompromised Adults. Clin Infect Dis. 2021;73(11): e4353-e4360.

CIDARA THERAPEUTICS

CD388 Development Data Confirm the Target Product Profile (TPP)

CD388 is being developed for pre-exposure prophylaxis for influenza



Single dose /~ 4-6 months

Promising Phase 2a data

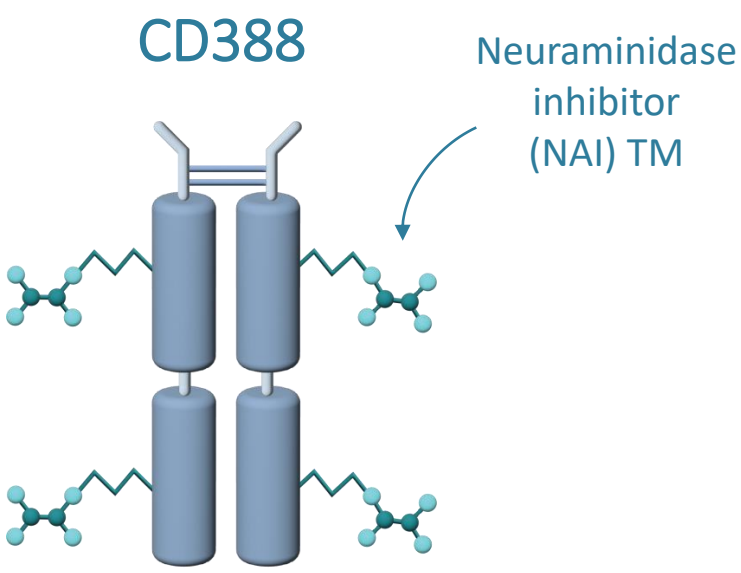
	DFCs
Potential for Universal protection	Yes
Potential to protect high-risk groups	High
Potential for prevention and treatment	Yes
Scale and cost	Attractive

Study summary at https://clinicaltrials.gov/ct2/show/NCT05285137

CIDARA THERAPEUTICS

Opportunity: First "Universal" Influenza Preventative

CD388 has the potential to be the first therapeutic to provide season-long, universal influenza protection



CIDARA THERAPEUTICS

- Reacquired Global Development and Commercial Rights from Janssen April'24
- Phase 2a results achieved safety, efficacy and PK objectives
- Coverage of diverse panels of seasonal and pandemic strains[1]
- Single SQ or IM dose per flu season
- Streamlined, low-cost manufacturing process, low cost of goods (COGs)
- High concentration formulations, compatible with SQ or IM dosing
- Low immunogenicity
- Well-tolerated in Phase 1 and 2a

Phase 2a clinical proof of concept supports advancement to pivotal studies

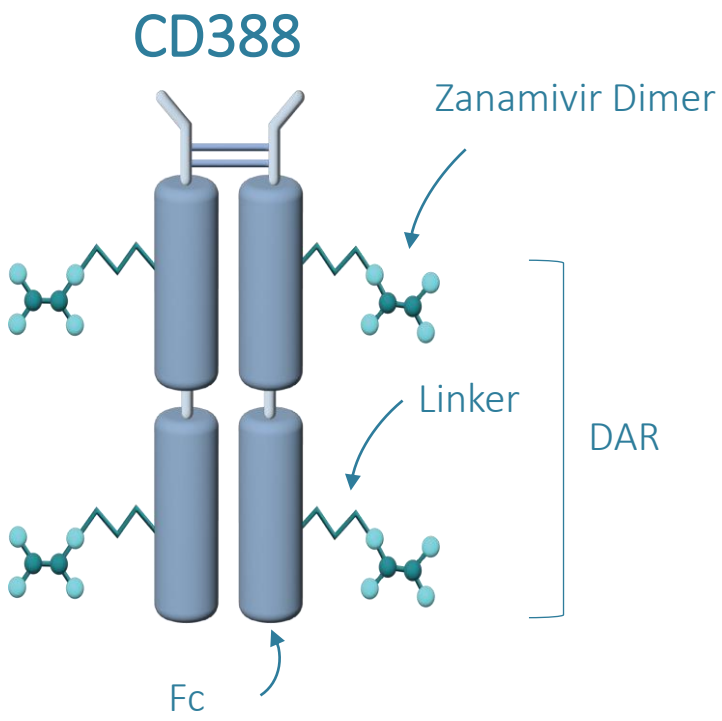
1. Based on preclinical data.

SQ = Subcutaneous; IM = Intramuscular

CIDARA THERAPEUTICS

Comprehensive Global IP Protects CD388 Exclusivity

Two Issued US Composition of Matter Patents – US Exclusivity to Sept. 2039 (+ PTE)



Portfolio Highlights:

- Global Portfolio Pending in 31 Jurisdictions
- Eight Patent Families Directed to:
  - CD388 Composition of Matter*
  - Fc Construct Composition of Matter
  - Conjugation Methods*
  - Dosing Regimens
- CD388 Composition of Matter Coverage Includes:
  - Zanamivir Conjugation Point Optionality*
  - Multiple Covalent Linker Options (length, composition, dimer spacer)*
  - Wide Drug-Antibody Ratio (DAR) Range*

As biologic agents, DFCs are not subject to the IP limitations of the Inflation Reduction Act (IRA)

*Included in issued patents
PTE = Patent Term Extension

CIDARA THERAPEUTICS

CD388 Is Differentiated From Other Influenza Prophylactics

CD388 is the most advanced small molecule or peptide biologic conjugate in clinical development[1]

Companies in Space	CIDARA THERAPEUTICS	sanofi GSK Pfizer moderna Others[3]	moderna Pfizer NIH National Institutes of Health Others[3,4]	VIR / GSK Others[5]
Modality	**DFC (CD388)**	Vaccines – strain specific (mRNA, conventional e.g. Fluzone)	Vaccines – Universal (mRNA, protein-based)	Monoclonal antibodies
Stage of Development	**P2**	Approved/P3	P2/3	Failed/Terminated
Spectrum	**Universal[2]**	Strain specific	Universal	Influenza A[5]
Efficacy in High-Risk Populations	**Yes[2]**	Low/None[3]	Low/None	Yes
Route of Administration	**SC/IM**	SC/IM	SC/IM	IV[5]

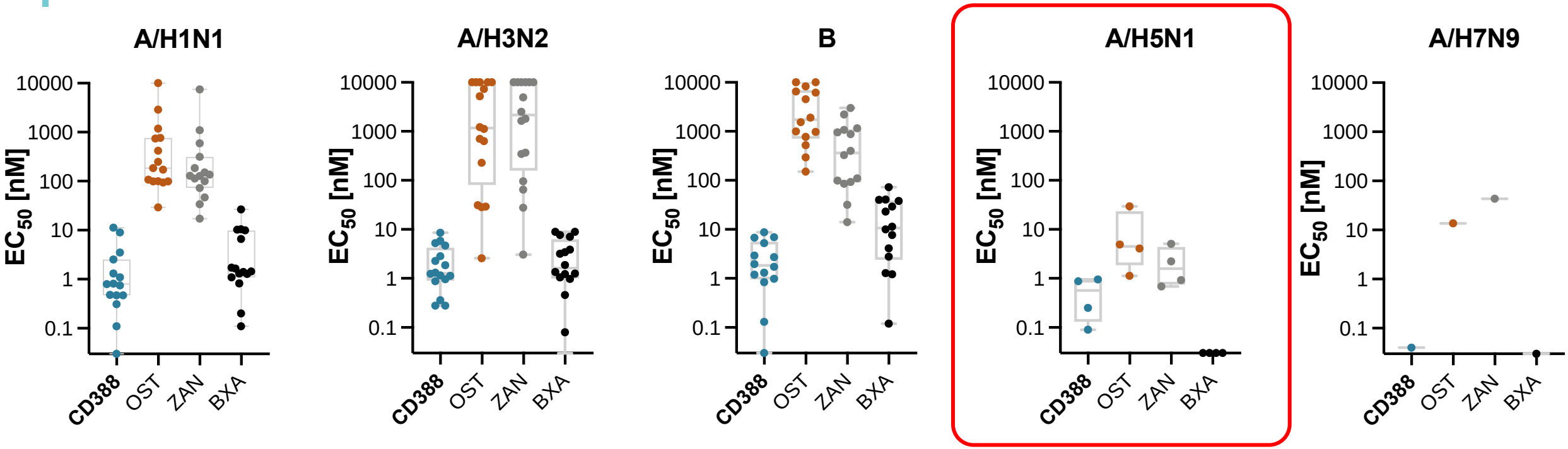
1. Based on Clinicaltrials.org.
2. Based on preclinical data.
3. Moderna quadrivalent mRNA vaccine (mRNA 1010) may perform better than conventional vaccines in elderly adults based on Phase 3 topline data https://s29.q4cdn.com/435878511/files/doc_downloads/program_detail/2024/flu-11-02-23.pdf.
4. Gupta and Mohan Journal of Genetic Engineering and Biotechnology (2023) 21:154 https://doi.org/10.1186/s43141-023-00581-y
5. Includes: Celltrion mAb cocktail (CT-P27) (NCT03511066), Medimmune (MEDI8552) (NCT02603952), NIAID/Crucell (CR6261, CR8020) (NCT01992276), Roche (MHAA45498) (NCT02293863), Visterra (Vis410) (NCT03040141 – only tested as treatment). Vir 2482 was IM dosed in a phase 2b prophylaxis study, but failed to meet efficacy endpoints (NCT05567783)

CIDARA THERAPEUTICS

CD388: Potential First "Universal" Influenza Preventative

CD388 retains potent antiviral activity across diverse seasonal and high pathogenicity strains, including H5N1

Cytopathic Effect (CPE) Activity Versus Influenza Strain Panels



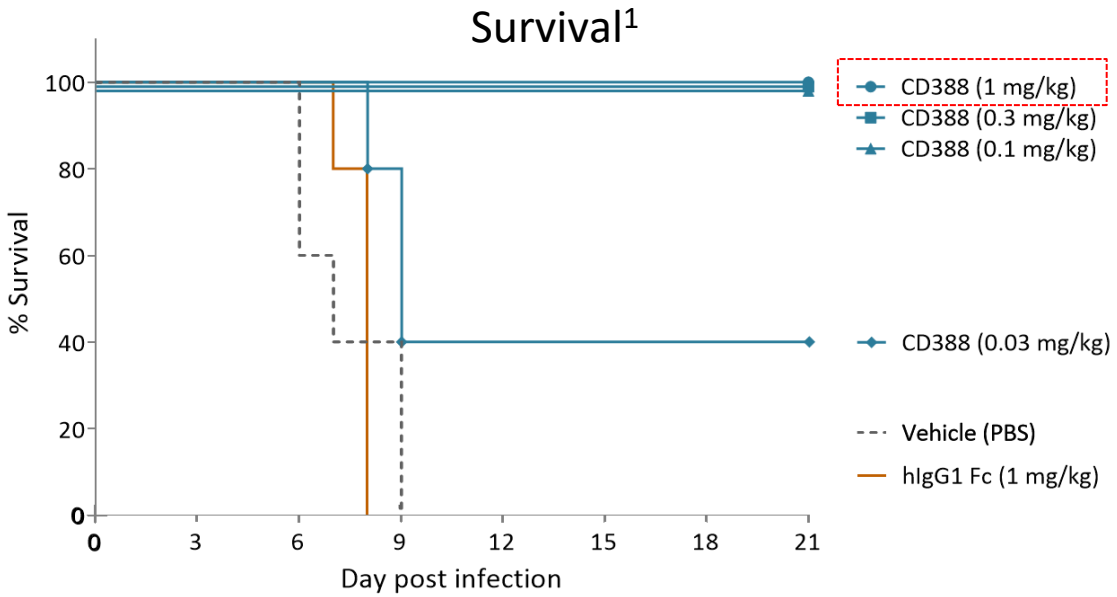
OST = Oseltamivir carboxylate; ZAN = Zanamivir; BXA = Baloxavir acid

CIDARA THERAPEUTICS

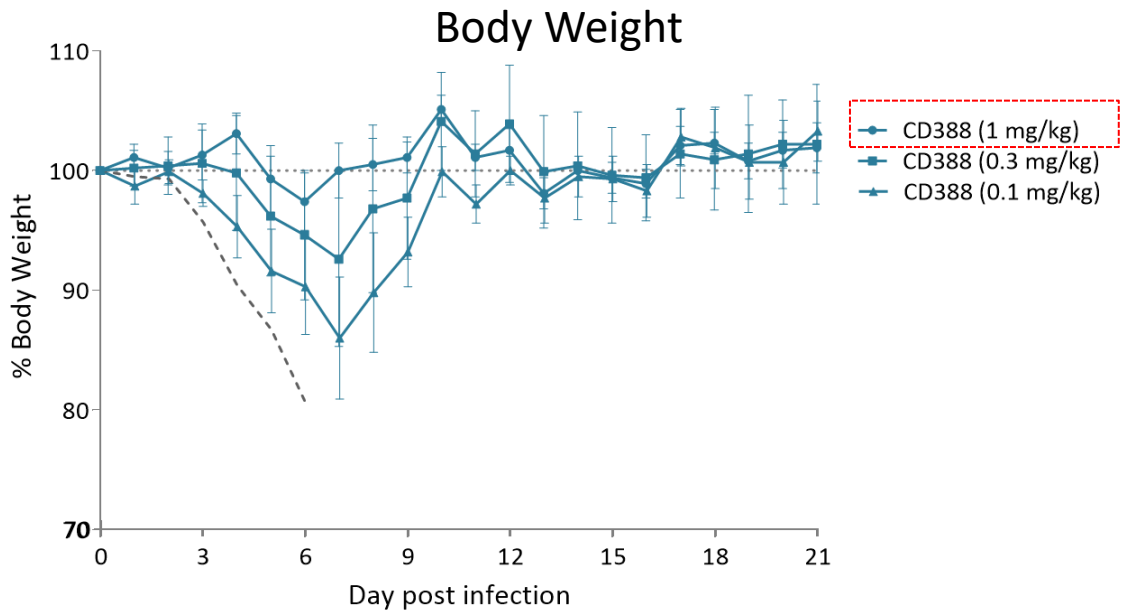
Potential for Single Dose, Long-Acting Prevention in Lethal Models

CD388 has the potential to be the first therapeutic to provide season-long, universal influenza protection

100% survival across broad dose range



Protection against body weight loss



Exposure selected for clinical development

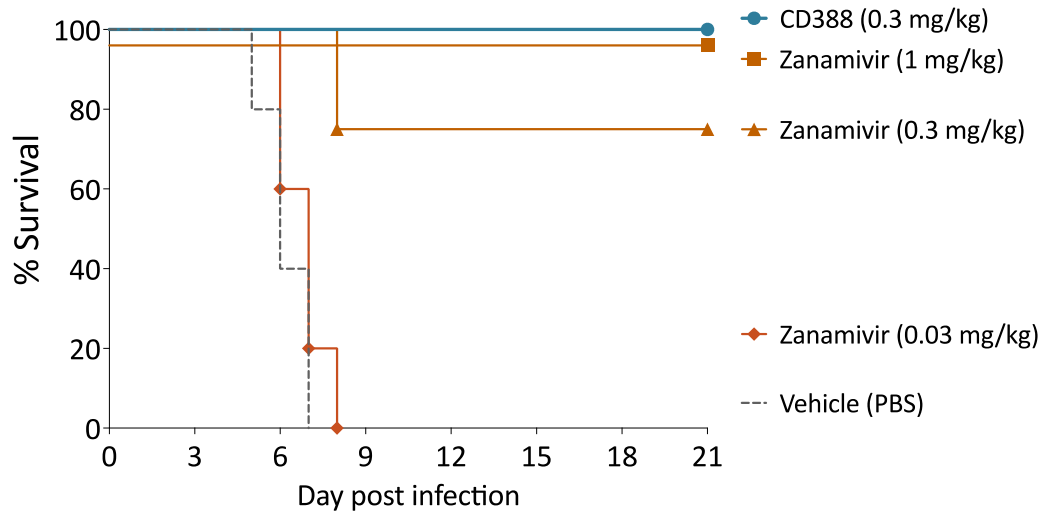
CD388 protected mice from lethal infection across broad panels of influenza H1N1, H3N2, B/Vic and B/Yam strains at doses **≤ 1 mg/kg**

1. Treatment initiated 2 hours post viral challenge. A/Puerto Rico/8/1934(H1N1). Single IM dose.

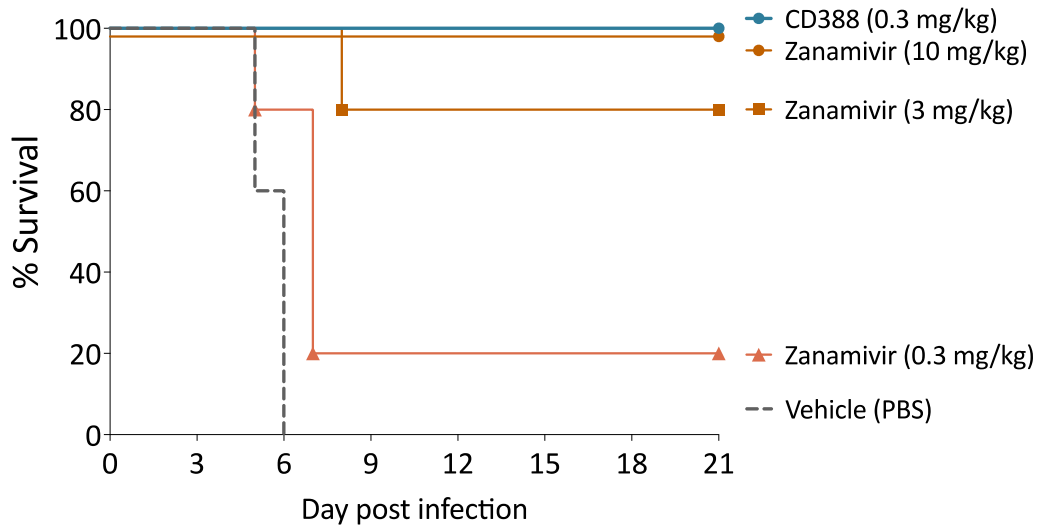
CD388 Retains In Vivo Activity Against Highly NAI Resistant Strains

Protective dose of CD388 does not shift in mice infected with matched NAI sensitive and NAI resistant strains

B/Laos/0080/2016 - NAI Sensitive[1]



B/Laos/0654/2016 - H134N[1]



Strain	Neuraminidase inhibition IC_{50} (nM)		Protective dose (mg/kg)	
	CD388	Zanamivir	CD388	Zanamivir
B/Laos/0080/2016 **H134 (NAI-S)**	7.44	2.61	0.3	1
B/Laos/0654/2016 **H134N (NAI-R)**	4.66	310.8	0.3	10

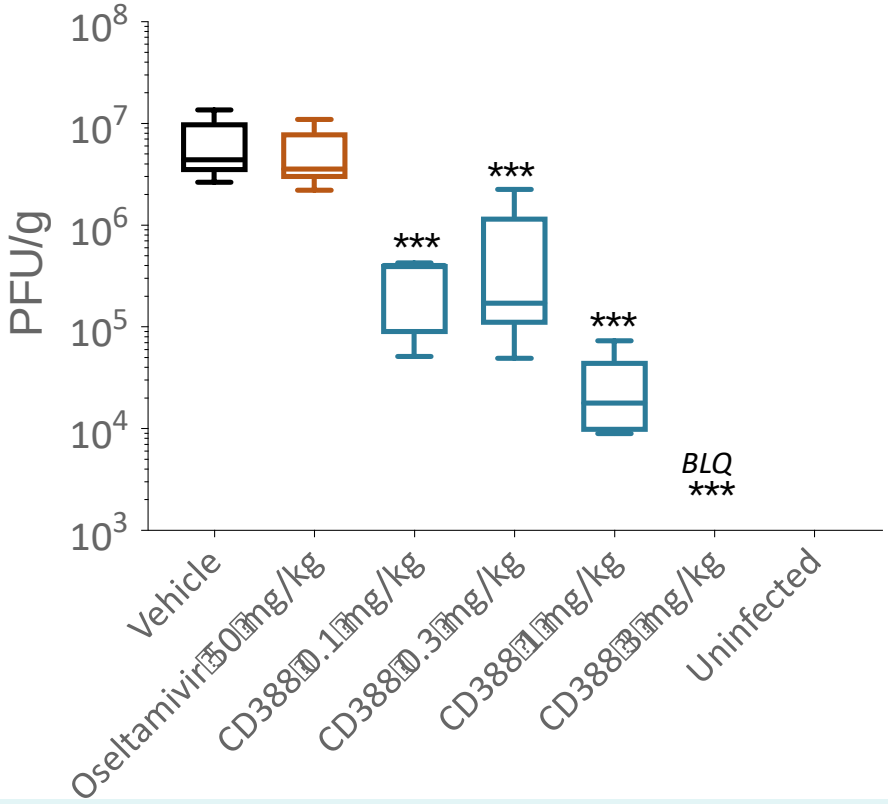
1. 5 animals/group treated a single IM dose of CD388 2-hours after viral challenge. Zanamivir dosed IN once daily for 5-days starting 2-hours after infection. Survival monitored for 21 days

CIDARA THERAPEUTICS

CD388 Reduces Viral Burden in Lung

Unlike oseltamivir, CD388 demonstrates robust, dose dependent viral clearance in lungs

Viral burden in lung



Study Details:

- Mouse adapted A/Puerto Rico/8/1934 (H1N1) (n=5 mice/arm)
- Treatment initiated 2 hours post infection
- Oseltamivir dosed twice daily (10x human equivalent dose)
- CD388 dosed once
- Viral burden assessed four days after lethal challenge

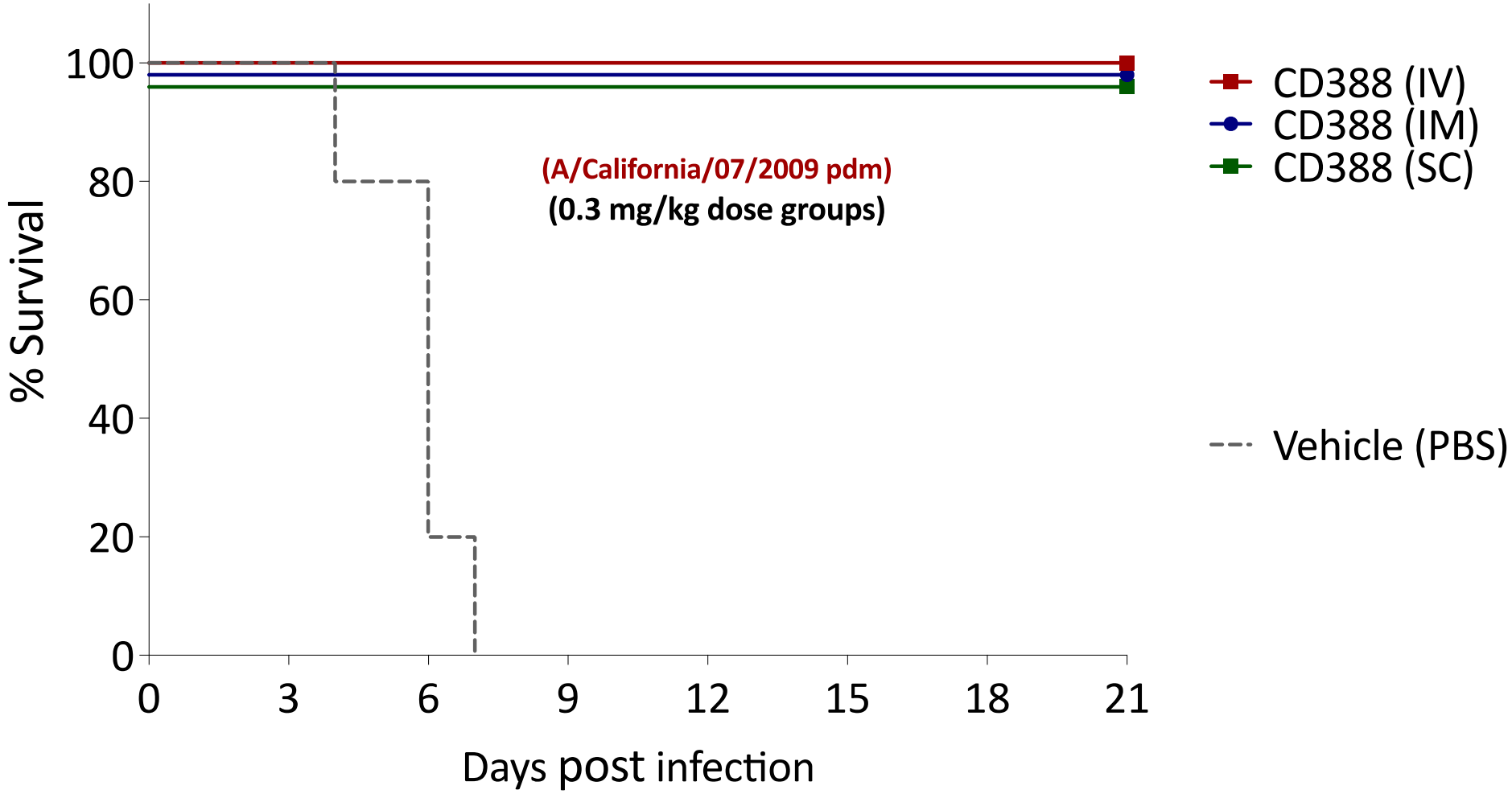
CD388 potently inhibits NAI resistant strains with improved pharmacodynamics

*** P < 0.001

CIDARA THERAPEUTICS

CD388 Properties Enable Flexible Dosing Options

Equivalent efficacy whether dosed via IV, subcutaneous or intramuscular injection[1]



1. Treatment initiated 2 hours post viral challenge. A/Puerto Rico/8/1934(H1N1)

CD388 Nonclinical Summary

Data support potential for single dose "universal" prevention and treatment

Efficacy & PK	• Potent in-vitro and in-vivo activity against all tested seasonal and pandemic strains of influenza A and B, including strains resistant to zanamivir and tamiflu (neuraminidase inhibitors) • Equivalent exposure and efficacy via IV, subcutaneous or intramuscular administration • Equivalent efficacy in immune-competent and immune compromised mouse models • Supports potential for single dose of 1.0 mg/kg to provide protection for flu season
Safety	• Well-tolerated at doses up to 500 mg/kg (vs projected 1.0 mg/kg efficacious dose for 100% protection in lethal models) • Anti-drug antibodies directed to the human Fc fragment, not the targeting moiety (TM) • No concerning findings in reproductive or genotoxicity studies

CIDARA THERAPEUTICS

Objectives of Early Clinical Programs

Pharmacokinetics	Can CD388 be used for seasonal prophylaxis of influenza?
Safety	Is CD388 well-tolerated and amenable for prophylactic use?
Efficacy	Does CD388 exhibit activity against influenza in the respiratory tract of humans?

CIDARA THERAPEUTICS

Phase 1 and Phase 2a Clinical Studies

First in human Phase 1 study	A Phase 1, Randomized, Double-Blind, Single-Dose and Repeat Single Dose, Dose-Escalation Study to Determine the Safety, Tolerability, and Pharmacokinetics of CD388 Intramuscular or Subcutaneous Administration in Healthy Subjects (63 subjects)
Japan bridging Phase 1 study	A Phase 1, Randomized, Double-Blind, Single Ascending Dose Study to Determine the Safety, Tolerability, and Pharmacokinetics of CD388 Subcutaneous Administration in Healthy Japanese Subjects (21 subjects)
Phase 2a study	A proof-of-concept, randomized, double-blind, placebo-controlled, Phase 2A study to assess the prophylactic antiviral activity against influenza, safety, tolerability, and pharmacokinetics of CD388 via a human viral challenge model (30 subjects)

CIDARA THERAPEUTICS

CD388 Was Well-Tolerated for Up To 900 MG

Safety data from First-in-Human, Japanese Bridging and Human Challenge studies

Number of participants that received one dose of CD388 in Phase 1 and Phase 2a studies (as of Aug 2023)

Dose	FIH	JBS	HCS	Total
50 mg	18	7	2	27
150 mg	18	7	28	53
450 mg	18	7	0	25
900 mg	9	0	0	9
All Doses	63	21	30	**114**

FIH- First in Human study; **JBS-** Japanese Bridging study;
HCS- Human Challenge study

Safety Summary

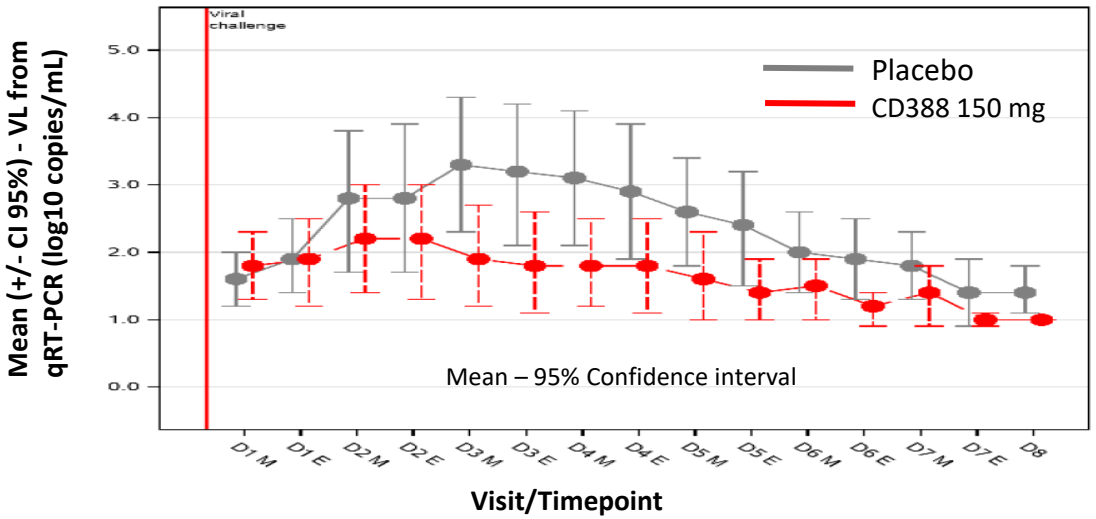
- No treatment-emergent SAEs and no discontinuation of study drug or withdrawals due to safety findings
- No consistent AE patterns
- No hypersensitivity reactions
- Most TEAEs Grade 1 (90%), few Grade 2, all resolved
- Incidence of TEAE not dose-dependent
- Few injection site events (pain, IM route mainly), Grade 1, all resolved spontaneously
- No clinically relevant ECG, vital signs or physical exam abnormalities

SAEs = Serious Adverse Events; TEAE= Treatment Emergent Adverse Events

CD388 Demonstrated Efficacy in Phase 2a

Endpoint	Placebo N=28	CD388 150 mg N=28	P-value
qRT-PCR confirmed influenza infection *	14 (50%)	6 (21%)	0.0248
qRT-PCR confirmed symptomatic influenza infection **	9 (32%)	4 (14%)	0.1023
qRT-PCR confirmed moderately to severe symptomatic influenza infection ***	7 (25%)	3 (11%)	0.1477

Mean VL from qRT-PCR



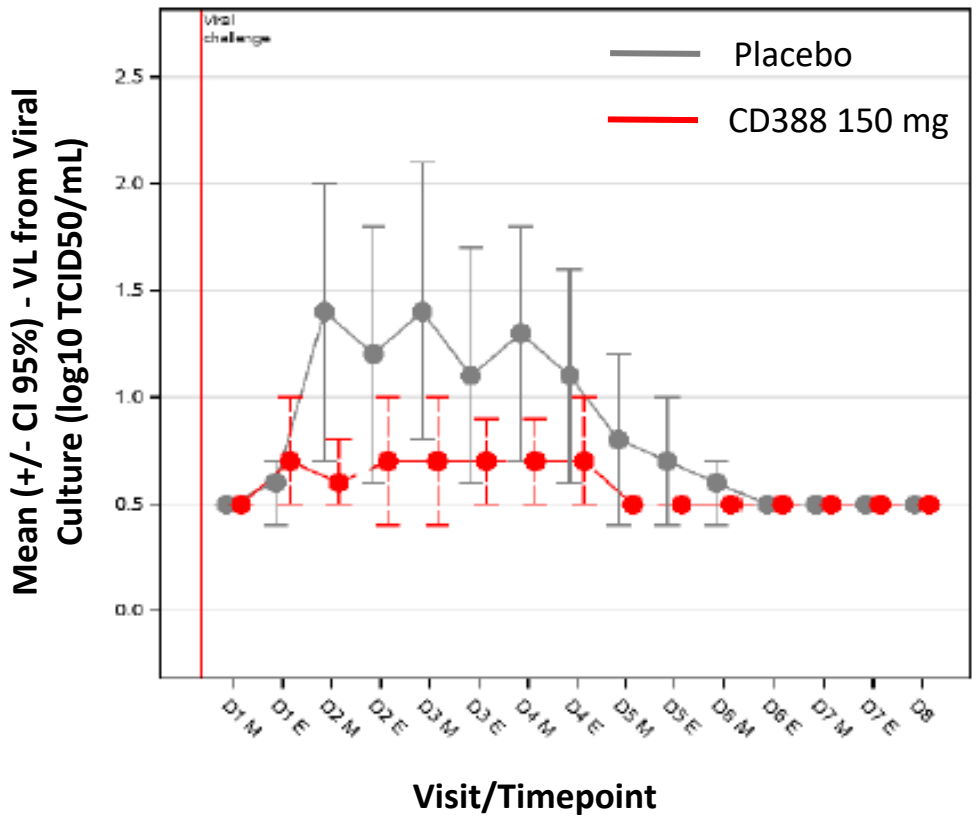
Primary endpoint: AUC viral load-time_ qRT-PCR

One sided p-value Wilcoxon rank sum test: 0.0390

*RT-PCR-confirmed influenza infection: 2 quantifiable (≥ lower limit of quantification [LLOQ]) qRT-PCR measurements (reported on 2 or more independent samples over 2 days), from Day 1 (pm) up to Day 8 (am).
**RT-PCR-confirmed symptomatic influenza infection: RT-PCR-confirmed influenza infection (2 quantifiable [≥LLOQ] qRT-PCR measurements [reported on 2 or more independent samples over 2 days]), from Day 1 (pm) up to Day 8 (am), AND symptoms ≥2 at a single time point.
***RT-PCR-confirmed moderate to severe symptomatic influenza infection: RT-PCR confirmed influenza infection (2 quantifiable [≥LLOQ] qRT-PCR measurements [reported on 2 or more independent samples over 2 days]), from Day 1 (pm) up to Day 8 (am), AND any symptoms of grade ≥2 at a single time point.

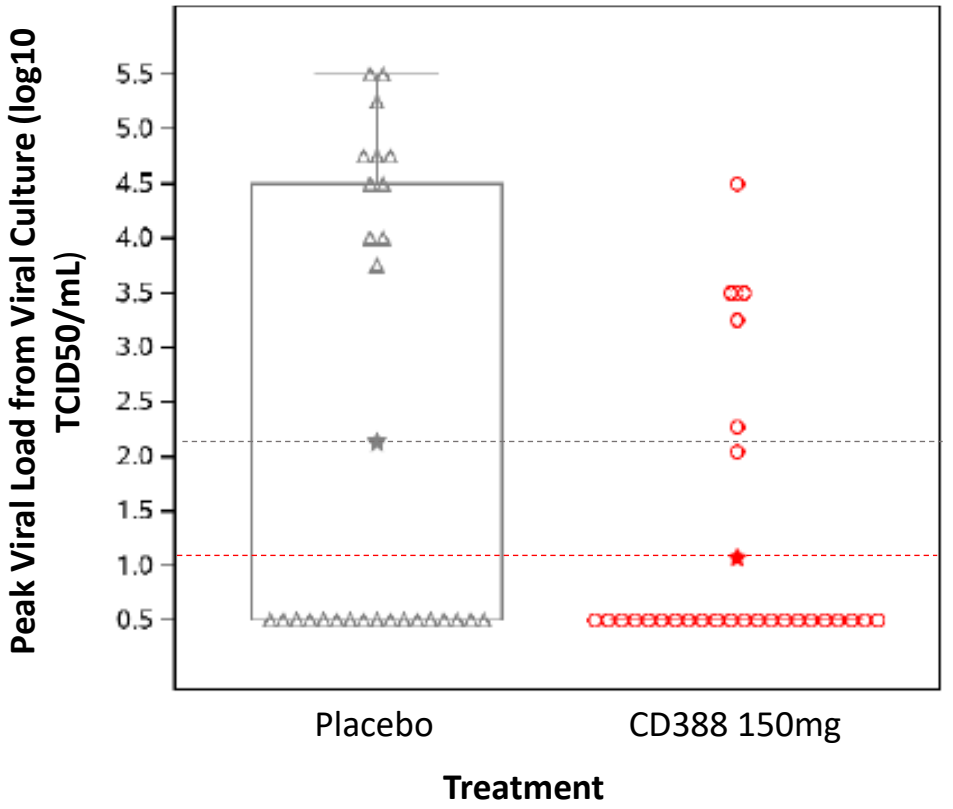
Viral Culture Data Confirmed Efficacy Seen in Early Analyses

Mean VL from Viral Culture



Mean – 95% Confidence interval

Peak Viral Load from Viral Culture



*, * = mean.
Placebo median = 0.50; CD388 150 mg median = 0.50

Phase 2b CD388 Influenza Prevention Study Planned in Fall 2024

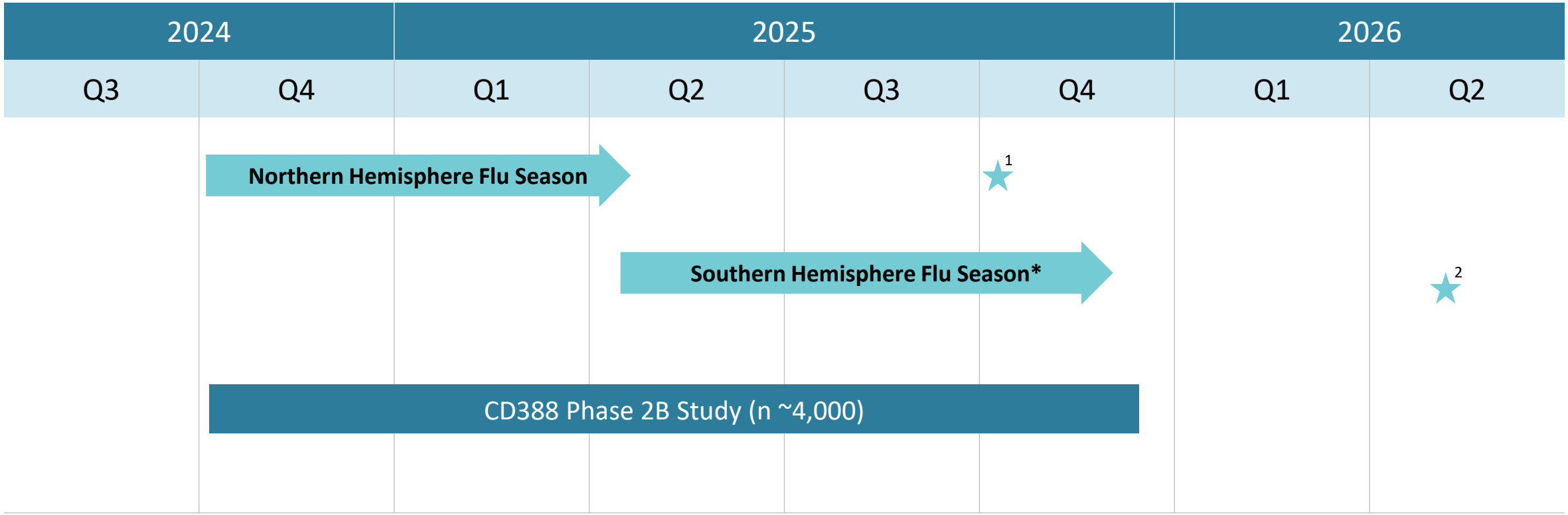
Phase 2b Double-Blind RCT for the Efficacy/Safety of CD388 vs Placebo in the Prevention of Influenza

- **Study Design:** Blinded, randomized, controlled trial with single doses of CD388 or placebo administered at the beginning of the influenza season with subjects followed for the entire influenza season to monitor for breakthrough cases of influenza
- **Primary Endpoint:** To compare the rates of laboratory-confirmed clinical influenza between different single doses of CD388 and placebo over an influenza season
- **Study Population:** Generally healthy adults (patients at high-risk of complications from influenza excluded)
  - Seasonal influenza vaccines also excluded
- **Study Size:** Target of 4000 subjects with possible increase to a maximum of 6000 subjects (depending on the rate of influenza infection) with three CD888 dose groups and 1 placebo group randomized in a 1:1:1:1 ratio

Planned Phase 3 study in subjects at high risk of developing complications from influenza

RCT = Randomized Clinical Trial

CD388 Phase 2b Estimated Development Timeline

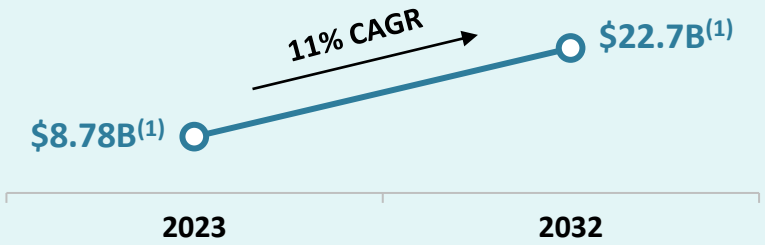


Actual timelines may differ materially due to severity of flu season, rate of patient enrollment etc.

1 Topline Data if NH only
2 Topline Data if NH & SH
* If needed

CD388: A Potential Multi-Billion Dollar Market Opportunity

Global Influenza Vaccine Market



Market Growth and Expansion Expected

- **Growth** due to rise of more effective vaccines
- **Expansion** due to:
  - Next generation premium flu vaccines
  - Better prophylactic agents
  - New therapeutic modalities with improved efficacies

CD388 Target Product Profile & Positioning

- Potential for universal protection
- Potential to protect all high-risk groups
- Potential for prevention and treatment
- Attractive scale and cost

% Market Share of Influenza High-Risk Groups [2] (18 and older)

- Adults 18 and older
- Immunocompromised or weakened immune systems (e.g., HIV, cancer or on immunosuppressive drugs)
- People with certain health conditions {diabetes, lung disease, asthma, heart disease, sickle cell anemia, kidney or liver disease, metabolic disorders, and disorders than may cause breathing problems (e.g., muscle, nerve disorders)}
- People who are overweight - Body Mass Index (BMI) 40 or over
- People living in nursing homes and other care facilities
- Those who are in close contact with people at high risk of complications (e.g., healthcare workers)

% Market Share of Traditional and Premium Vaccine Market

+

+

% Market Share of Worldwide Opportunity

1 Source: Influenza Global Flu market till 2032 (2023-2032 CAGR 11.07%).
https://www.globenewswire.com/en/news-release/2024/03/05/2840453/0/en/Global-Influenza-Vaccine-Market-to-Attain-Valuation-of-USD-22-71-Billion-By-2032-Astute-Analytica.html
2. https://www.nyc.gov/site/doh/providers/health-topics/influenza-high-risk-groups.page;
CAGR = Compounded Annual Growth Rate; HIV = human immunodeficiency virus.

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Benchmarks of Estimated Peak Sales for Selected Prophylactic Flu Programs

	CIDARA THERAPEUTICS	moderna	VIR / GSK	GSK / CUREVAC	Pfizer / BIONTECH	Traditional Vaccines [1]
Estimated Peak Sales [2]	**Multi-billion dollar potential**	**$5.6bn**	**$5.2bn**	**$2.9bn**	**$3.9bn**	**$5.9bn[1]**
Modality	DFC	mRNA Vaccine	mAb	mRNA Vaccine	Quadrivalent mod RNA Vaccine (mRNA vaccine)	Quadrivalent Vaccine (egg and Cell based)
Current Status	Phase 2	Phase 2/3	Terminated	Phase 2	Phase 3	Commercial
Broker / Source	N/A	William Blair 7/24/2023	Needham 5/5/2023	Goldman Sachs 12/11/2023	HSBC 7/2023	CDC

1. FY 2022 total revenue estimates based on CDC and individual Company data; includes Flumist, Fluarix, FluLaval, Fluzone, Fluzone HD, Flubloc, Afluria, Flucelvax & Fluad.
2. Estimated Peak Sales at 100% POS. Patient population and addressable market for each program may not be similar.

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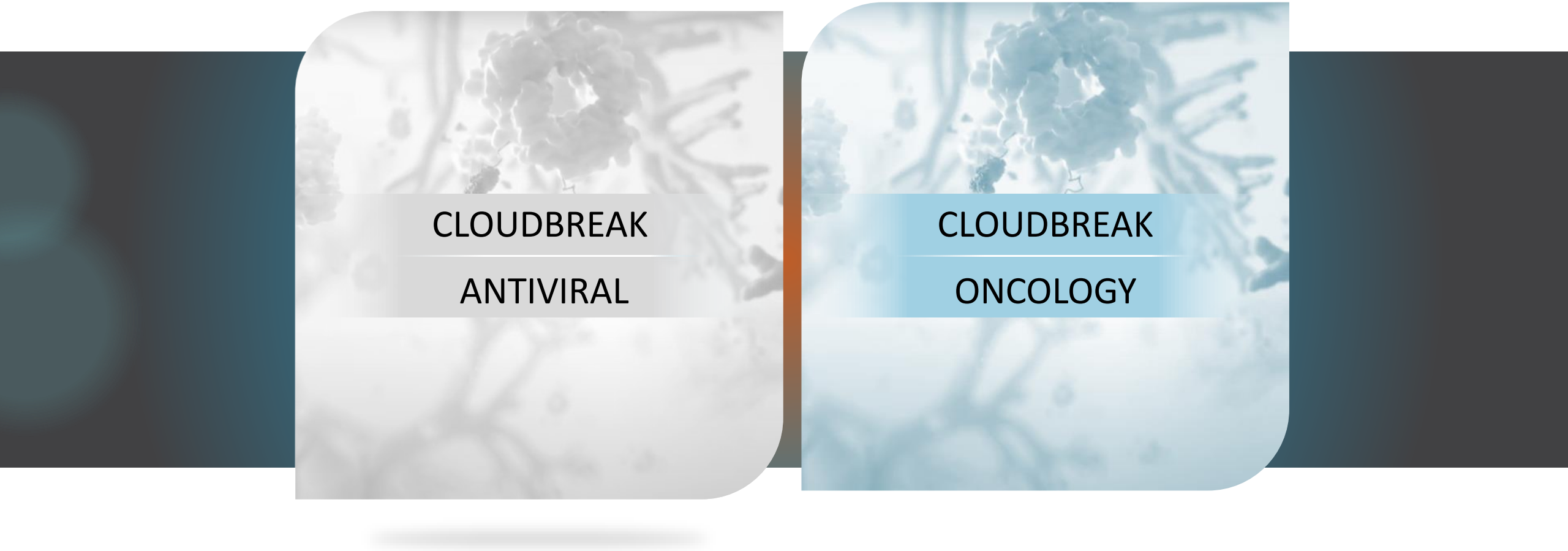
Cidara's Pipeline Targets **Multiple Unmet Medical Needs**



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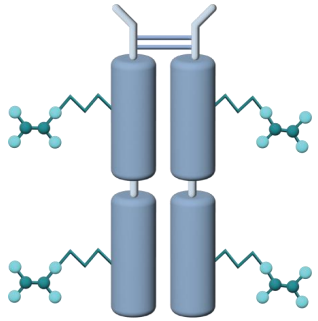
Cloudbreak Oncology DFC Programs

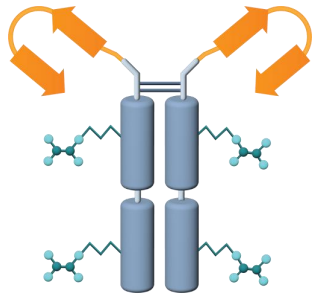
CD73: CBO421 Development Candidate

- Differentiated from existing SM and mAb CD73 inhibitors
- Complete responders in combination with PD-1 inhibitors
- Excellent safety and COGS



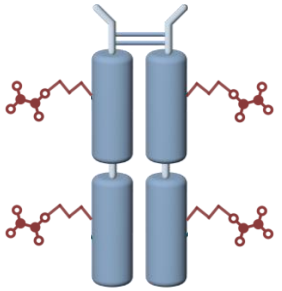
PD-1/CD73 Discovery Program

- Unique dual inhibitor of CD73 and PD-1
- Compelling preclinical data
- Potential for more efficient clinical development



CCR5 Discovery Program

- Overcomes limitations of prior approaches
- Proof of concept data against “undruggable” target
- Potential for improved safety over SM antagonists



mAbs = Monoclonal Antibodies; SM= Small Molecule

CIDARA THERAPEUTICS

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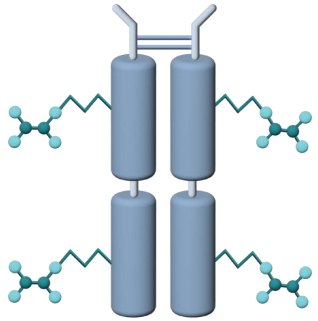

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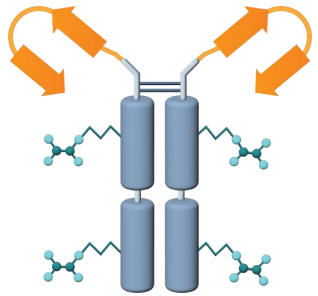

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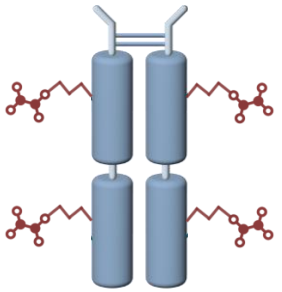

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Adenosine is an Important Mediator of Tumor Immune Evasion

Rationale for CD73

- Adenosine creates an immune suppressive Tumor Microenvironment (TME) by reprogramming multiple immune cell classes
- Dying tumor cells release ATP into the TME which subsequently gets converted into adenosine by CD73
- CD73 inhibitors are an ideal partner for chemotherapy

Tumor Immunosuppression

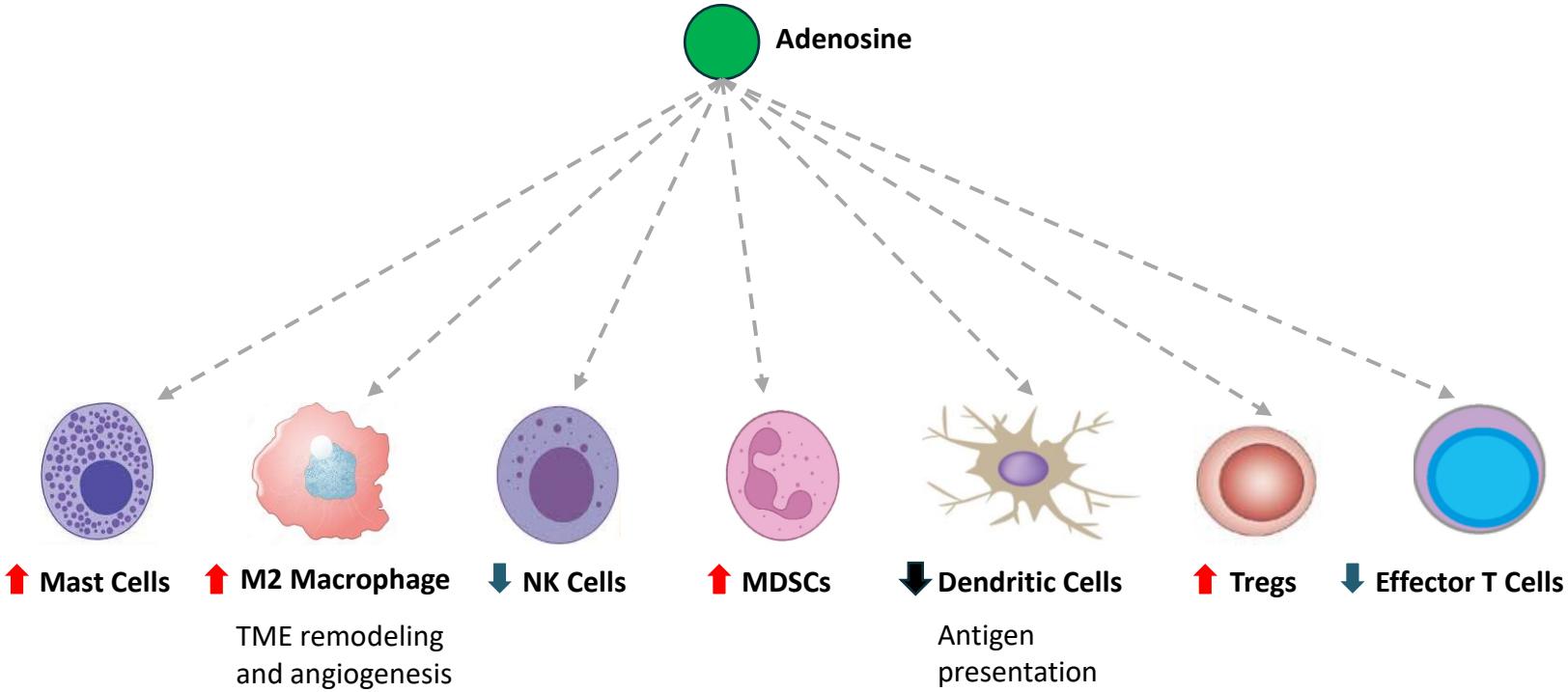

Image reprinted from Harvey JB, Phan LH, Villarreal OE and Bowser JL (2020) CD73's Potential as an Immunotherapy Target in Gastrointestinal Cancers. Front. Immunol. 11:508.

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CD73 Clinical Data To Date Has Been Disappointing

Is CD73 a valid target?

Therapy	Drug Class	Safety Profile	Single agent activity	Combination Efficacy
Oleclumab (AstraZeneca)	mAb	Benign	None[1]	With anti-PD-L1 in stage III NSCLC[2]: • ORR 30% (vs. 17.9%) • No additional CR's achieved beyond PD-L1 single agent activity
NZV930 (Novartis)	mAb	Benign	None; monotherapy trial halted due to poor efficacy	Studies in progress in combo with other immune therapies[3]
ORIC-533 (Oric)	SM	Benign	Study in progress[4]	No studies found

- No Approved Drugs
- Significant potential to improve CD73 inhibitors

Cidara's next generation inhibitor combines robust catalytic inhibition of soluble and cell anchored CD73 with receptor internalization for potential enhanced performance in the clinic

1. Kondo S et al. Int. J. Clin. Oncol., 27 (2022), pp. 1795-1804
2. Herbst R et al. J Clin Oncol. (2022) Oct 10;40(29). NSCLC = Non-Small Cell Lung Cancer.
3. https://doi.org/10.1158/1538-7445.AM2022-CT503
4. https://doi.org/10.1182/blood-2023-173730;
   mAbs = Monoclonal Antibodies; SM= Small Molecule

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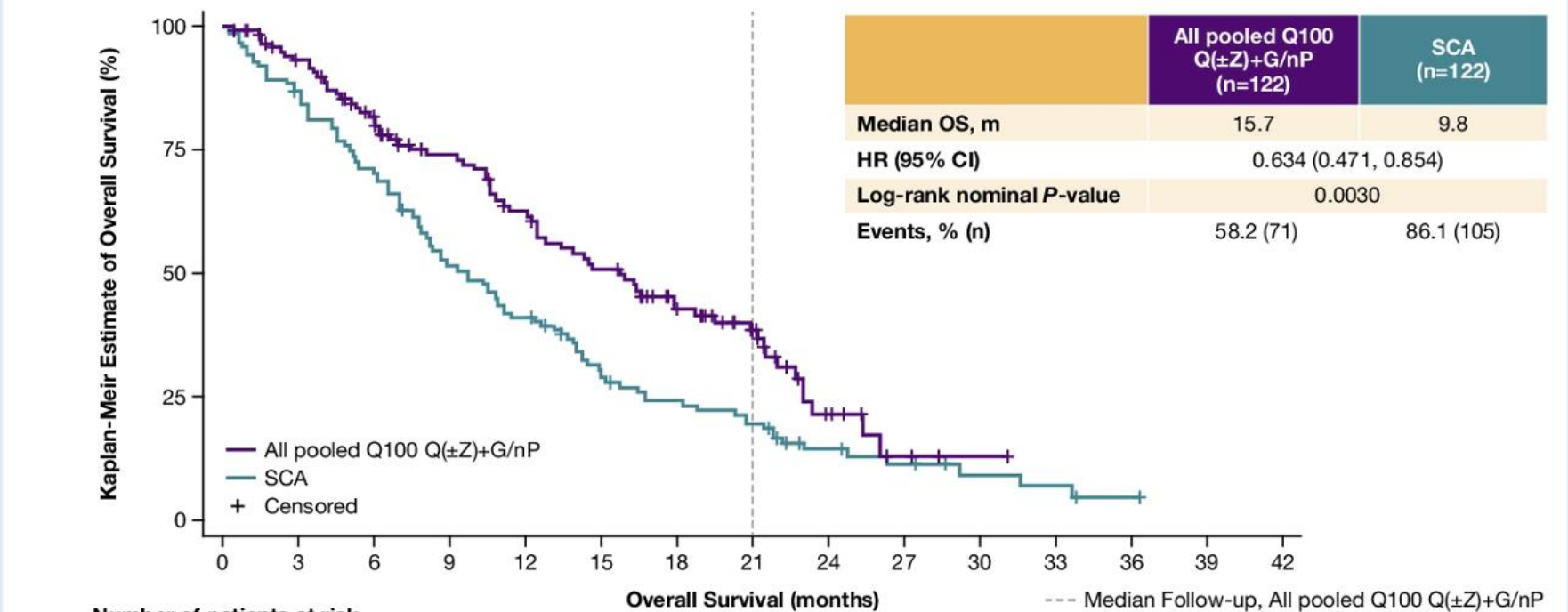
Recent Phase 1bData Supports CD73 as a Valid Target: Improved Overall Survival Combining CD73 Inhibitor With Chemotherapy

Despite selection of an inferior standard of care chemotherapy partner in the ARC-8 study

Figure 4. Overall Survival: Pooled Q(±Z)+G/nP vs SCA



	All pooled Q100 Q(±Z)+G/nP (n=122)	SCA (n=122)
Median OS, m	15.7	9.8
HR (95% CI)	0.634 (0.471, 0.854)	
Log-rank nominal *P*-value	0.0030	
Events, % (n)	58.2 (71)	86.1 (105)

Number of patients at risk

All pooled Q100 Q(±Z)+G/nP	122	108	89	72	59	47	34	23	8	3	1	0		
SCA	122	104	85	61	49	32	26	21	12	7	4	3	1	0

G/nP, gemcitabine/nab-paclitaxel; OS, overall survival; Q, quemliclustat; SCA, synthetic control arm; Z, zimberelimab.

1. Wainberg et al. GI ASCO 2024 poster https ://doi.org/10.1200/JCO.2024.42.3_suppl.665

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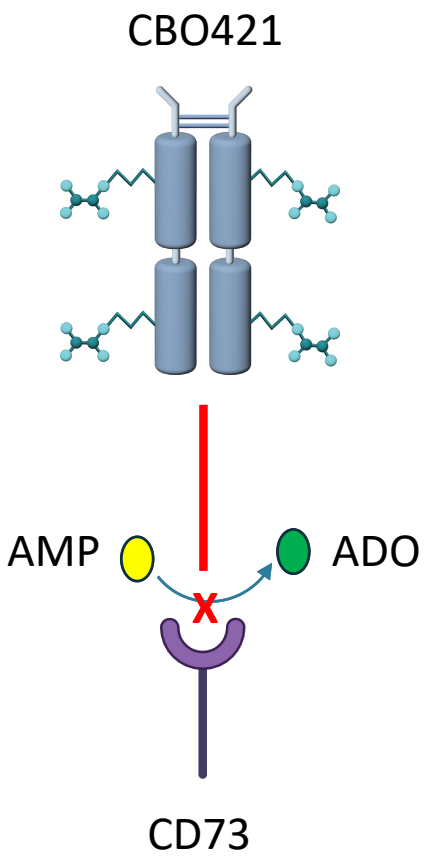
CBO421 Emerging as a Differentiated CD73 Therapy



AACR American Association for Cancer Research
ANNUAL MEETING 2024 • SAN DIEGO
APRIL 5-10
AACR.ORG/AACR2024
#AACR24

Session LBPO.IM02 - Late-Breaking Research: Immunology 2

LB131 / 19 - CBO421, a novel drug Fc-conjugate, inhibits the enzymatic activity of CD73 and triggers CD73 internalization

- Differentiated from existing SM and mAb CD73 inhibitors
- Highly stable with excellent pharmaceutical properties
- ~65 kDa (vs > 150 kDa for mAbs) – better tumor penetration
- Inhibits both membrane bound and soluble CD73, downregulates CD73 via internalization
- Ideal combination partner agent with PD-1/L1 axis inhibitors and chemotherapy
- Robust manufacturing process

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CBO421 Exhibits Differentiated Preclinical Performance

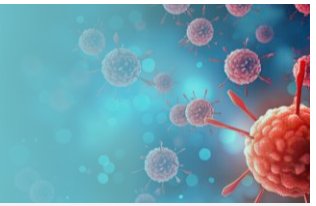
Differentiated from Existing Adenosine Inhibitors

PBMC rescue assay (ATP) vs clinical stage adenosine pathway inhibitors

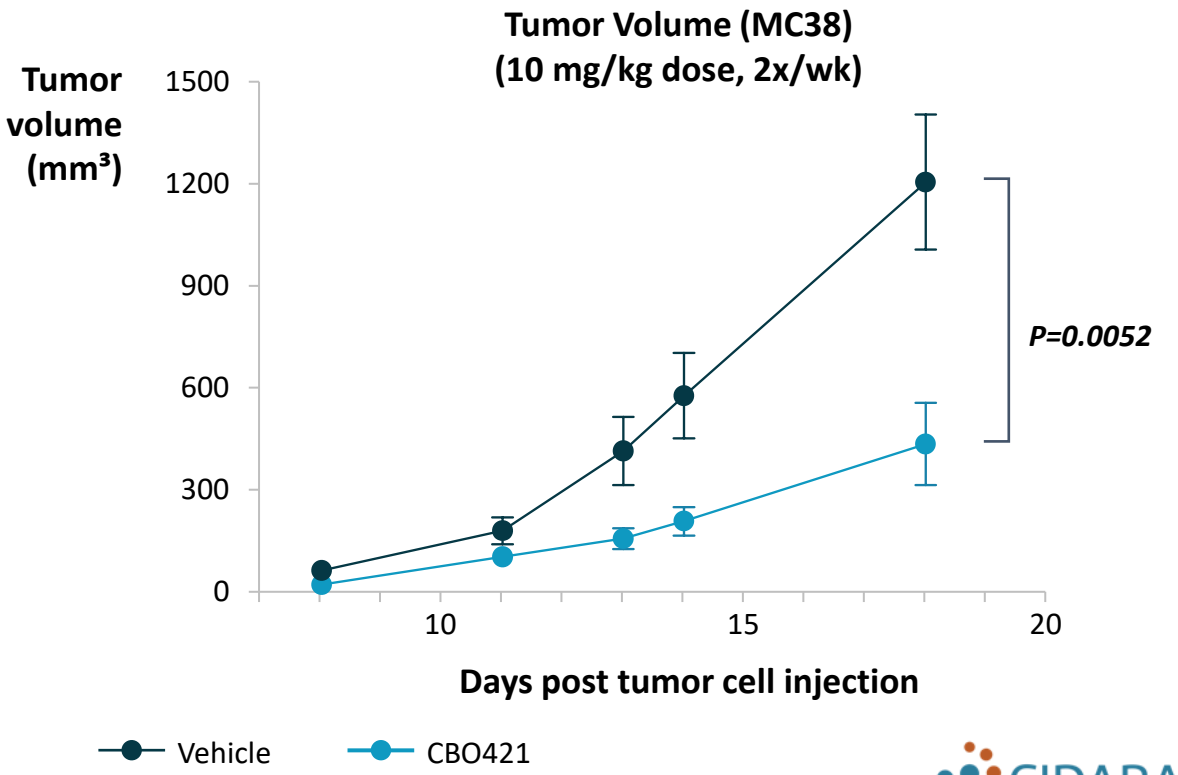
Test article	Target/Class	EC_{50} [nM]	
		$CD4^+CD25^+$	$CD8^+CD25^+$
CBO421	**CD73/DFC**	**13**	**51**
AB680*	CD73/small molecule	39	73
Oleclumab	CD73/mAb	>1,000	>1,000
IPH5201	CD39/mAb	>1,000	>1,000
AB928	A2AR/small molecule	>1,000	>1,000
CPI-444		>1,000	>1,000

AB680	Oleclumab	IPH5201	AB928	CPI-444
ARCUS BIOSCIENCES	AstraZeneca biosimilar	innate pharma biosimilar	ARCUS BIOSCIENCES	CORVUS PHARMACEUTICALS
CD73 inhibitor	CD73 inhibitor	CD39 inhibitor	A2AR inhibitor	A2AR inhibitor

Potent Anti-Tumor Activity as Monotherapy

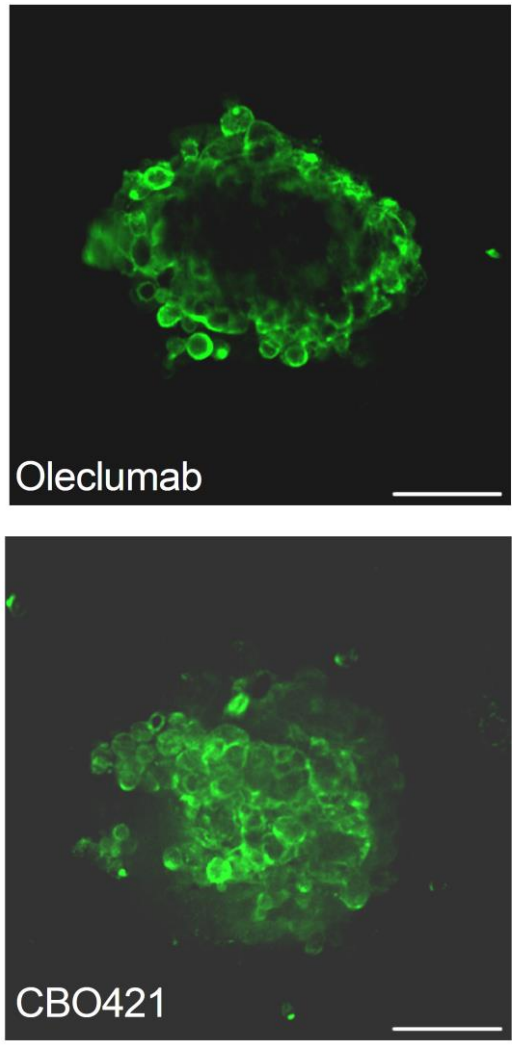


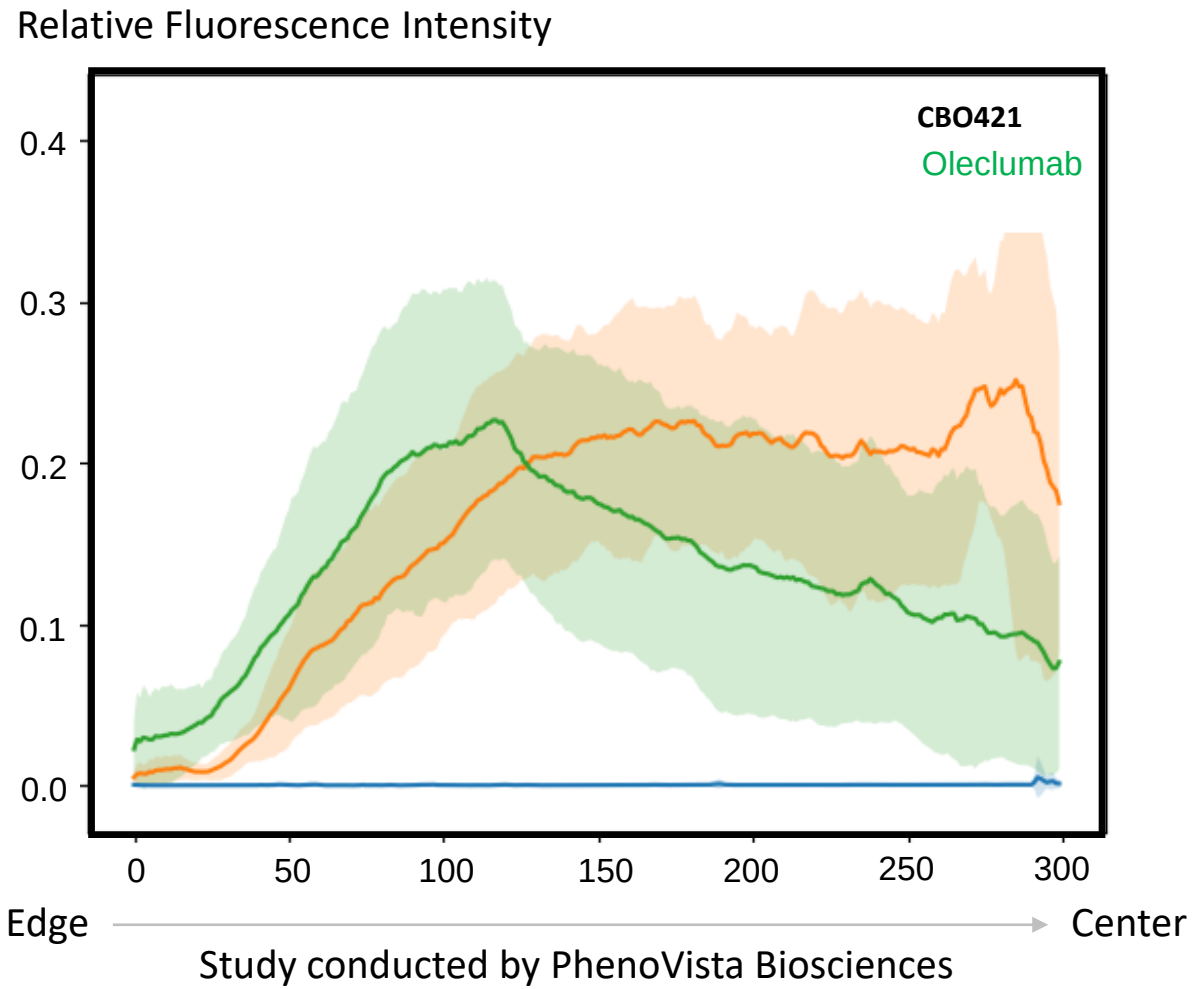
MC38 – murine colorectal carcinoma

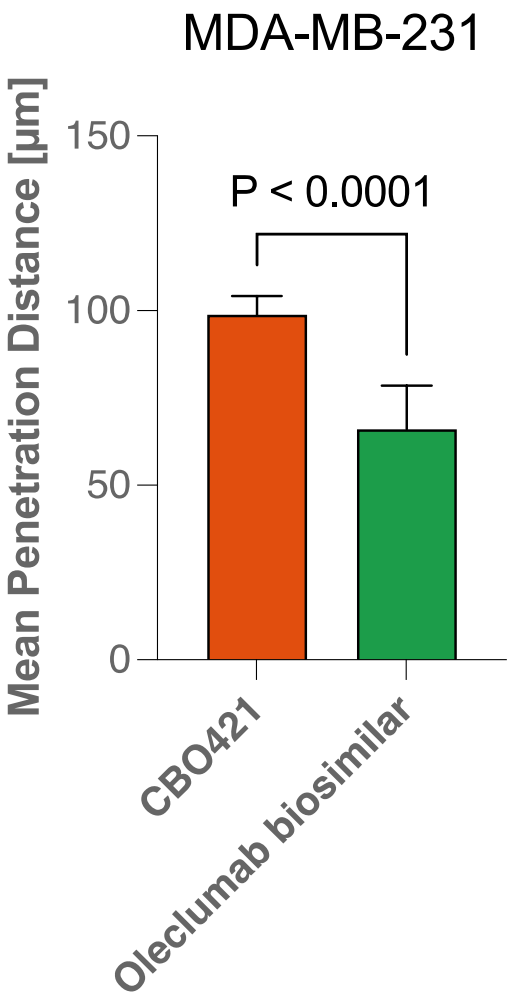


Tumor Control: CBO421 Exhibits Superior Tumor Penetration vs. mAbs

MDA-MB-231* Spheroids







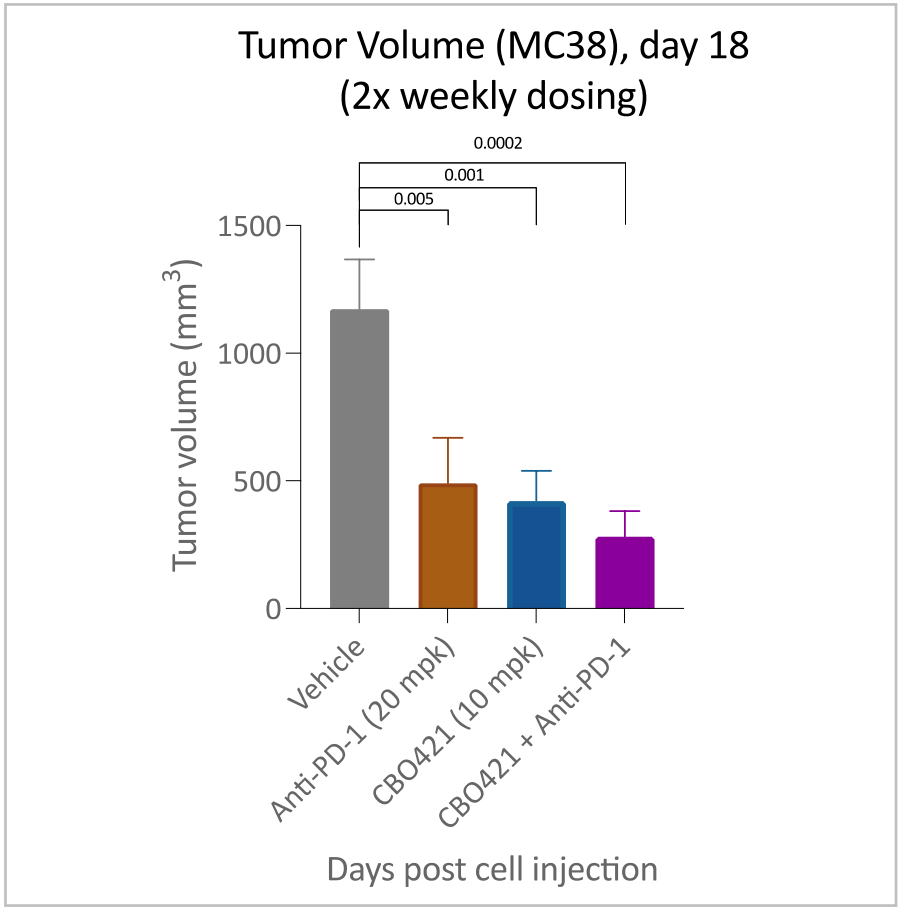
MDA-MB-231 cell line was isolated at MD Anderson, is ER, PR, and E-cadherin negative, and expresses mutated p53. It is commonly used to represent a model of TNBC.

CBO421 Enhances Anti-tumor Activity Of PD-1 Inhibitors

CBO421 + Anti-PD-1 combination improves response rates versus monotherapy

MC38 – murine colorectal carcinoma

Study Arm	% Responders[2]
Vehicle	0
CBO421	27
Anti-PD-1[1]	47
CBO421 + Anti-PD-1[1]	**60**



1. *RMP1-14
2. Defined as mice that demonstrate cessation of tumor growth or reduction in tumor volume across two or more timepoints.

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CBO421 Elicits Complete Responses with PD-1 Inhibitors

CBO421 + Anti-PD-1 combination improves survival and induces immunological memory

Study start (D+0)

Last dose (D+22)

40-day washout period

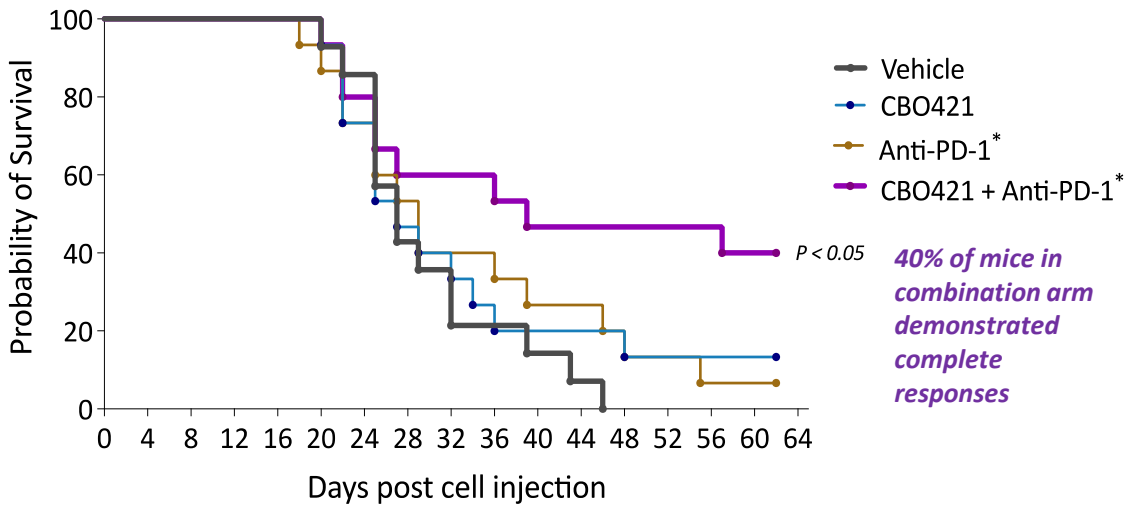
D+62 (re-challenge)

D+73

EMT6 – murine mammary carcinoma

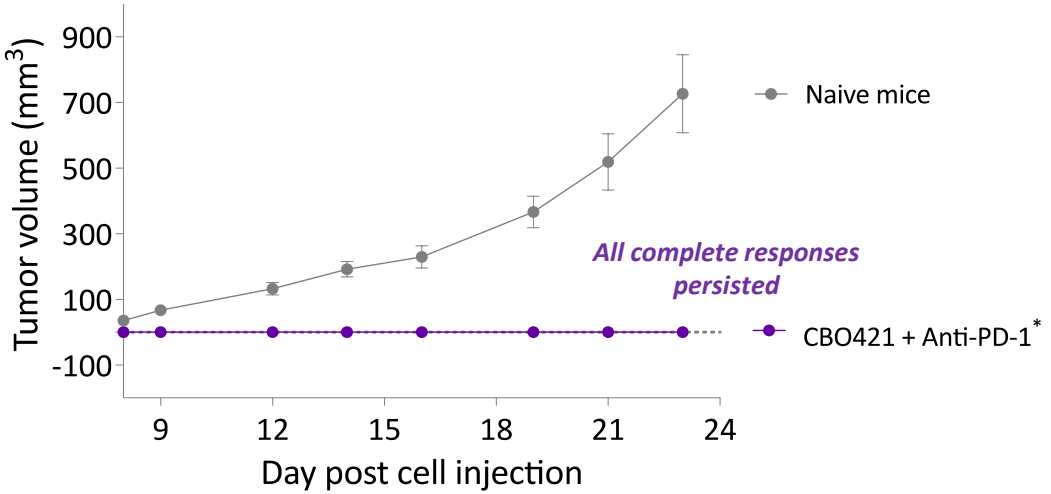
Survival (Day 62)

Compounds dosed 2x weekly for 22 days



40% of mice in combination arm demonstrated complete responses

Survivor rechallenge EMT6 tumor cells

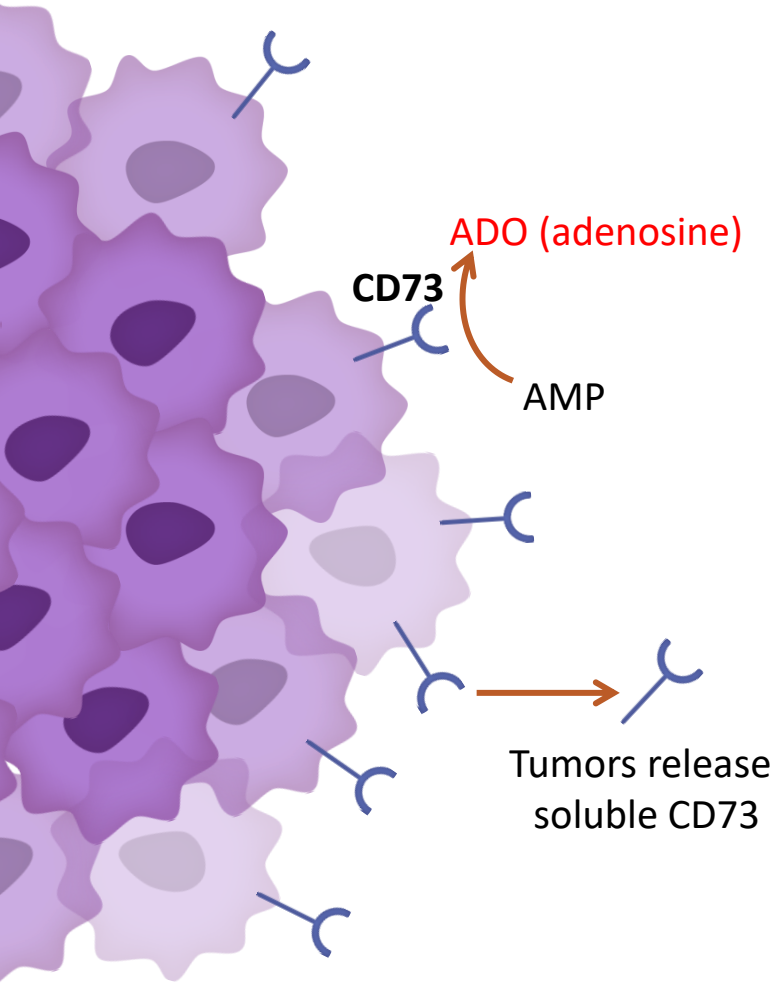


All complete responses persisted

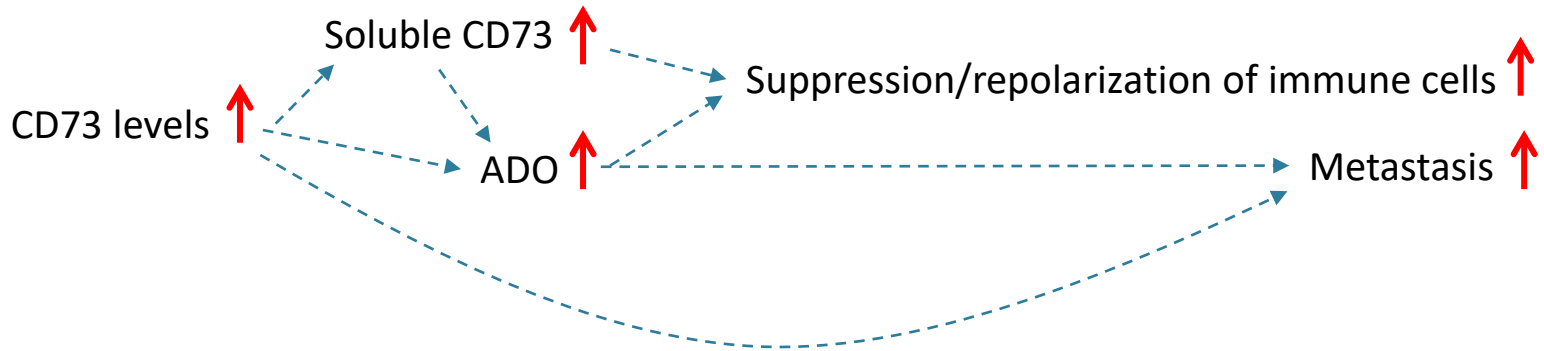
*RMP1-14

Chemotherapy Creates a Feedback Loop That Can Overwhelm Current CD73 Inhibitor Classes

CD73 inhibitors potentiate chemo, but effects can be attenuated by tumor adaptations



CD73 expression increases in response to chemotherapy, creating a pro-tumorigenic environment[1]

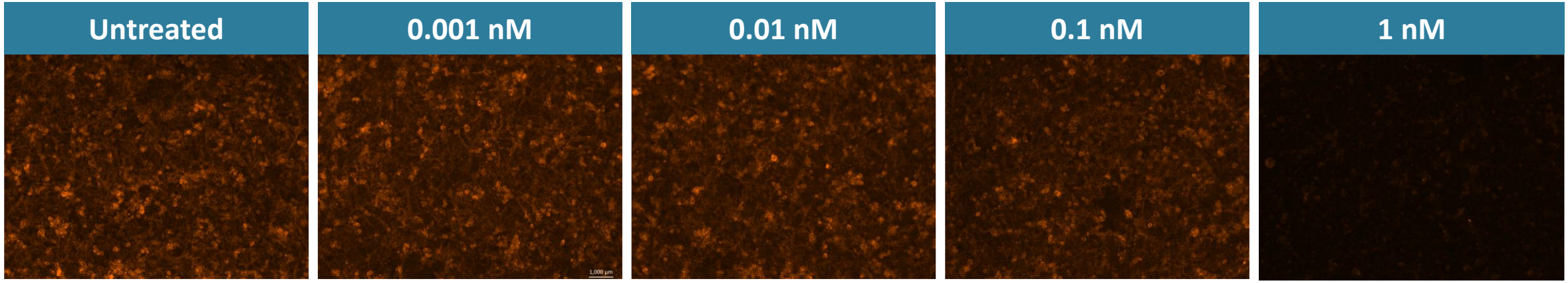


- *Small molecule CD73 inhibitors have no effect on CD73 levels, and can be overwhelmed by increasing ADO levels induced by chemotherapy*
- *mAb CD73 inhibitors are limited by inferior catalytic inhibition but some reduce CD73 levels via receptor internalization*
- ***CBO421 uniquely counteracts tumor adaptations to chemotherapy by combining potent catalytic inhibition and receptor internalization***

1. Bach, N. et al. Int. J. Mol. Sci. 2023, 24, 11759. https://doi.org/10.3390/ijms241411759

CIDARA THERAPEUTICS

Low CBO421 Concentrations Rapidly Reduce CD73 Levels on CD73+ TNBC Cells (MDA-MB-231)*



≥ 1 nM concentrations, CBO421 removes detectable cell anchored CD73 from the cancer cell surface

*MDA-MB-231 cell line is ER, PR, and E-cadherin negative and expresses mutated p53 and commonly used to represent TNBC.

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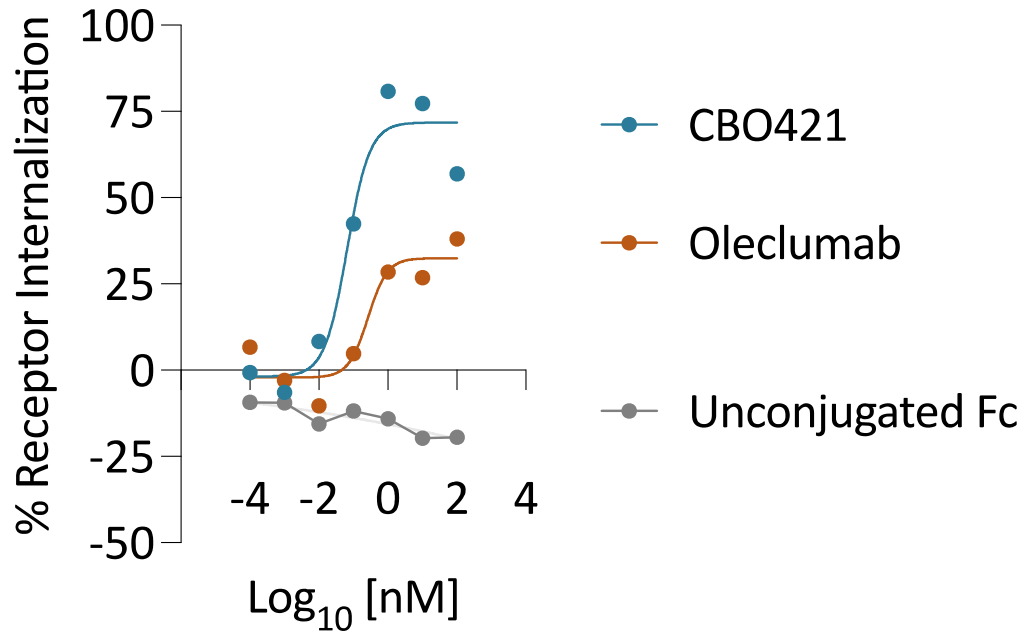
Combination of Receptor Internalization and Potent Catalytic Inhibition Positions CBO421 as an Ideal Chemotherapy Partner

CD73 Internalization on MDA-MB-231 tumor cells by flow cytometry (4 hours post incubation)



Test article	Target/Class	Maximum % internalization	EC_{50} [nM]
CBO421	**CD73/DFC**	**81**	**0.07**
AB680*	CD73/small molecule	0*	NA
Oleclumab#	CD73/mAb	38	0.27

CBO421 combines potent CD73 inhibition and CD73 downregulation, distinguishing it from small molecule and mAb CD73 inhibitors in clinical trials

°MDA-MB-231 cell line was isolated at MD Anderson, is ER, PR, and E-cadherin negative, and expresses mutated p53. It is commonly used to represent a model of TNBC.
*AB680 – Arcus Biosciences CD73 inhibitor, internalization data not shown
#Oleclumab – Astra Zeneca biosimilar CD73 inhibitor

CIDARA THERAPEUTICS

CBO421 Development for High Unmet Need TNBC Populations Leverages Potential Benefit of CD73/Chemo Combination Therapy

Triple Negative Breast Cancer (TNBC):

Lacks ER, PR, and HER2[1]: no targeted or hormonal therapies available

Young patients: majority diagnosed before age 50[2]

10% present with metastatic disease; another 30-50% develop metastatic disease after initial treatment[3]

Standard of care is chemotherapy backbone which provides median survival of 12-15m[3,4]

- For PD-L1+ (CPS ≥ 10): SOC is pembrolizumab + chemotherapy but only 30% are eligible[5]
- For BRCA-mutation+, SOC is PARP-I or chemotherapy but only 10-15% are eligible[6]
- High unmet need: approximately 50% of patients are *neither* CPS10+ *nor* BRCA+
- Chemotherapy remains backbone of every effective treatment for TNBC[7]

TNBC expresses high levels of soluble CD73 with correlation to poor prognosis and resistance to chemotherapy[8]

An opportunity for CBO421 to dramatically change the course of disease

1. Wolff et al. 138, 241–256. 10.5858/arpa.2013-0953.
2. McGuire et al. Cancers 7, 908–929. doi:10.3390/cancers7020815
3. Dent et al. Clin Cancer Res. 2007; 13: 4429-4434
4. Kim et al. Lancet Oncol. 2017 18, 1360–1372
5. Flavia et al. Cancers (Basel). 2023 Jun; 15(11): 2933
6. Gonzales-Angulo et al. Clin Cancer Res. 2011;17(5):1082–1089
7. Zeichner et al. Breast Cancer (Auckl). 2016; 10: 25-36
8. Loi et al. Proc. Natl. Acad. Sci. U.S.A. 2013: 110, 11091–11096

ER = Estrogen Receptor; PR= Progesterone Receptor

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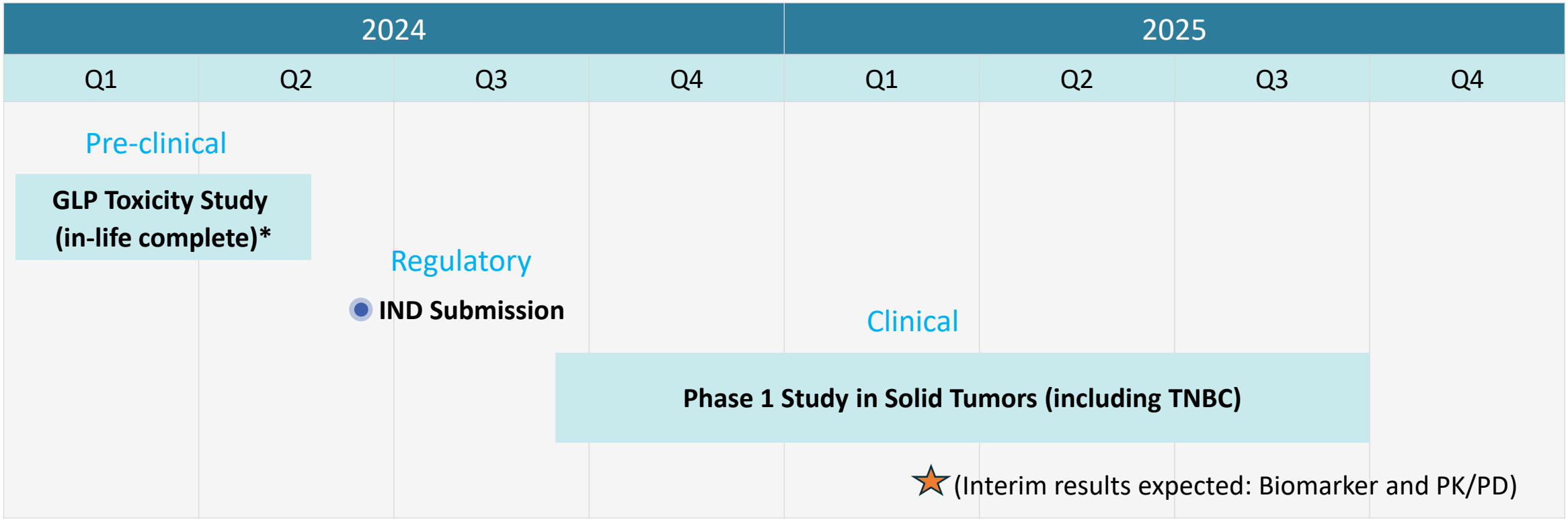
Planned First-in-Human Clinical Trials for CBO421



Actual timelines may differ materially due to timing of site initiation, patient enrollment, etc.

*Based on current results, NOAEL = 500 mg/kg/week

Cloudbreak CD73/PD-1 Bispecific DFCs

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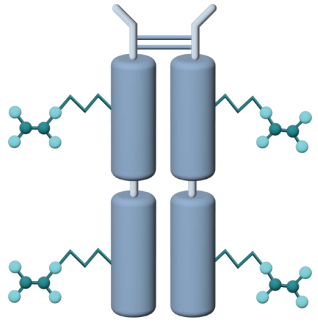

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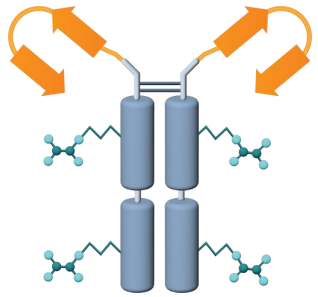

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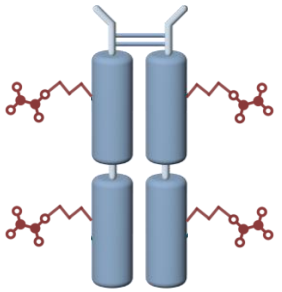

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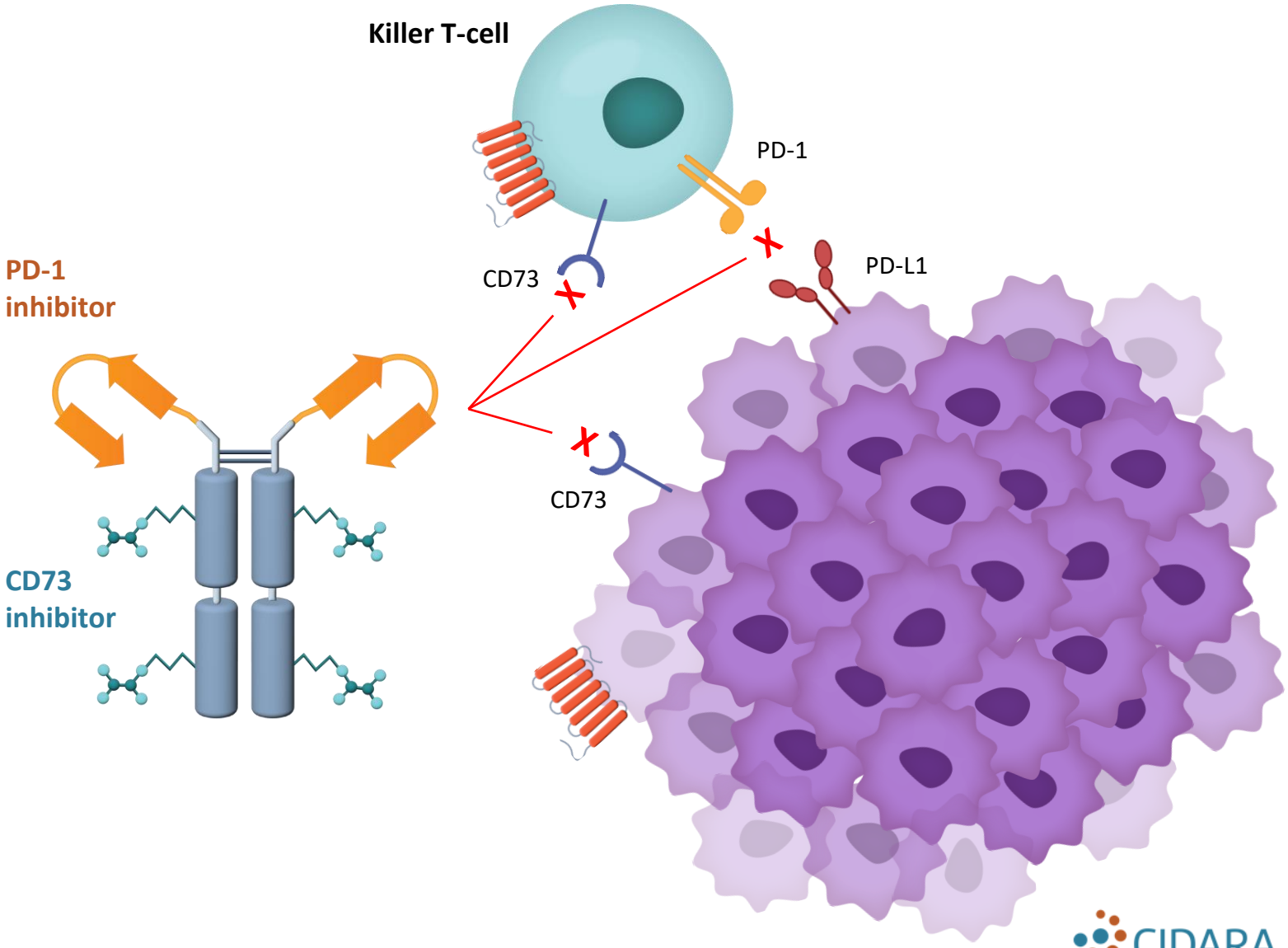
Potential First-in-Class PD-1/CD73 Inhibitor Disables Two Major Tumor Immune Escape Mechanisms in a Single Molecule

Cidara's first multispecific DFC (PD-1/CD73) is a **unique** dual inhibitor

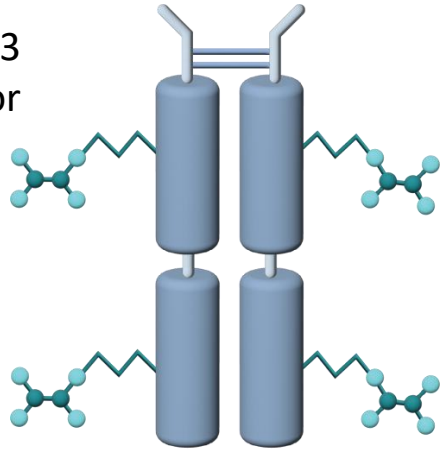
Engineered to improve efficacy by disabling a key resistance mechanism to PD-1 inhibitors



PD-1/CD73 DFC Potently Inhibits Both PD-1 and CD73 Receptors

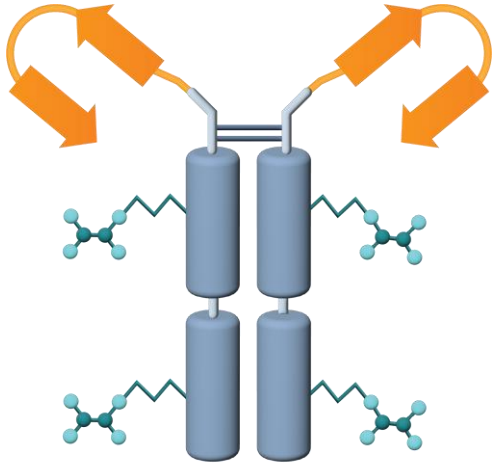
CD73 DFC

CD73 inhibitor



T-cell reactivation (EC_{50}, nM) – **12**

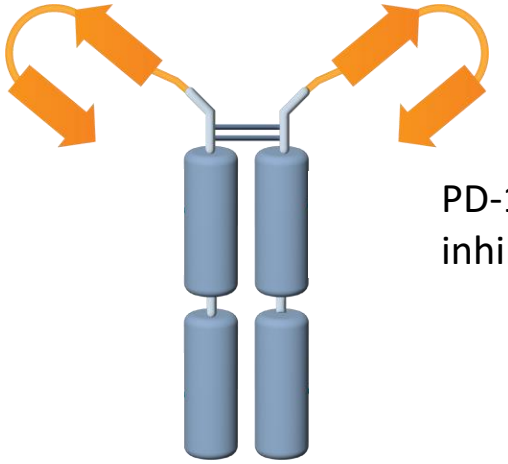
PD-1/CD73 DFC



PD-1 Binding (IC_{50}, nM) – **0.064**

T-cell reactivation (EC_{50}, nM) – **9**

CTP-PD-1



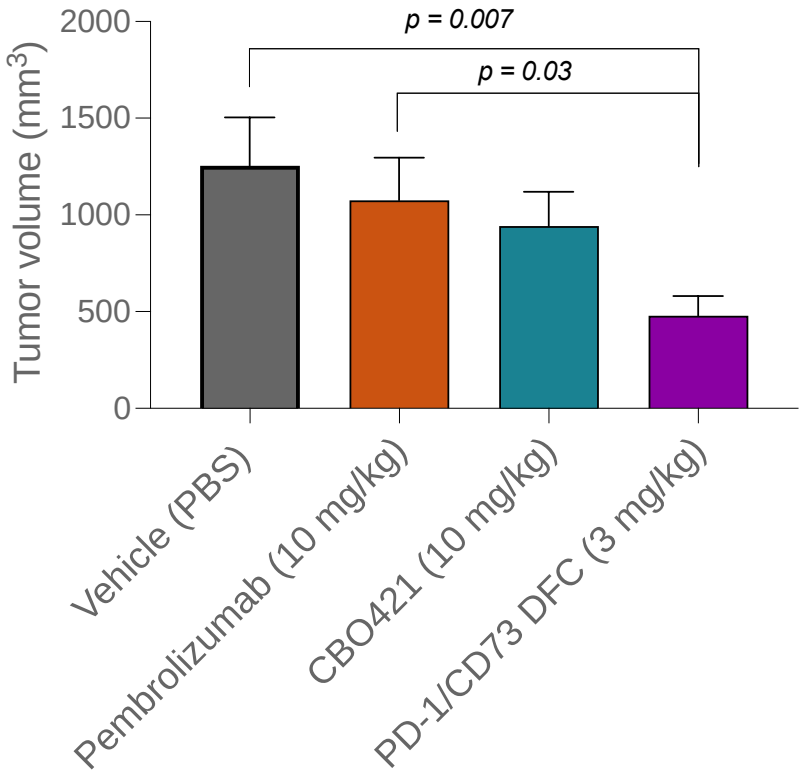
PD-1 inhibitor

PD-1 Binding (IC_{50}, nM) – **0.056**

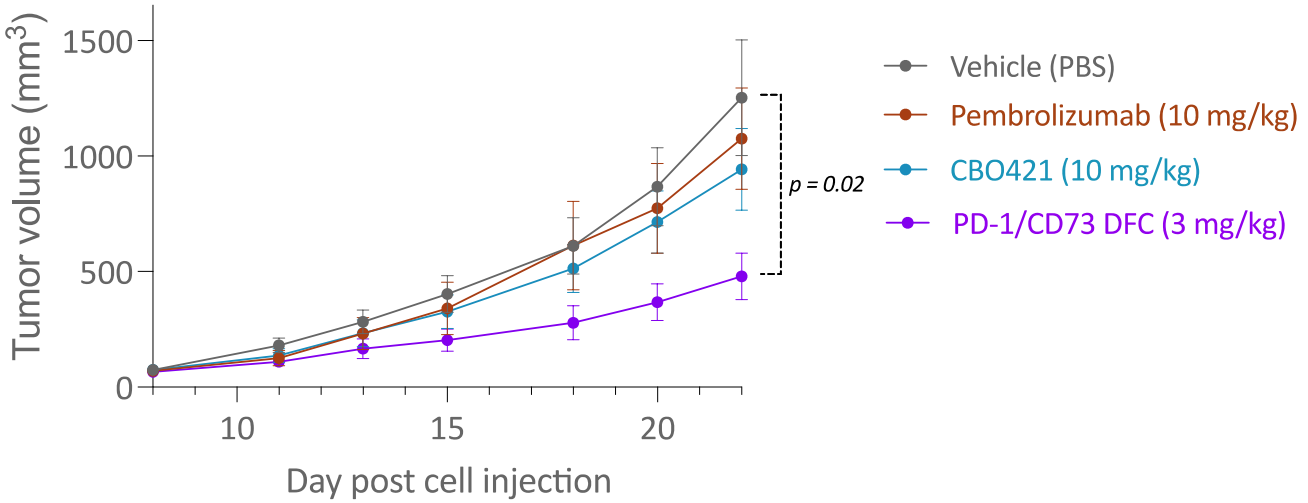
CIDARA THERAPEUTICS

PD-1/CD73 DFC Demonstrates Superior Tumor Growth Inhibition vs Monotherapies in Humanized Tumor Models

Tumor Volume (MC38, hPD-L1), day 22
hPD-1/hPD-L1 C57BL/6 mice – 2X weekly dosing



Tumor Volume (MC38, hPD-L1), day 22
hPD-1/hPD-L1 C57BL/6 mice – 2X weekly dosing

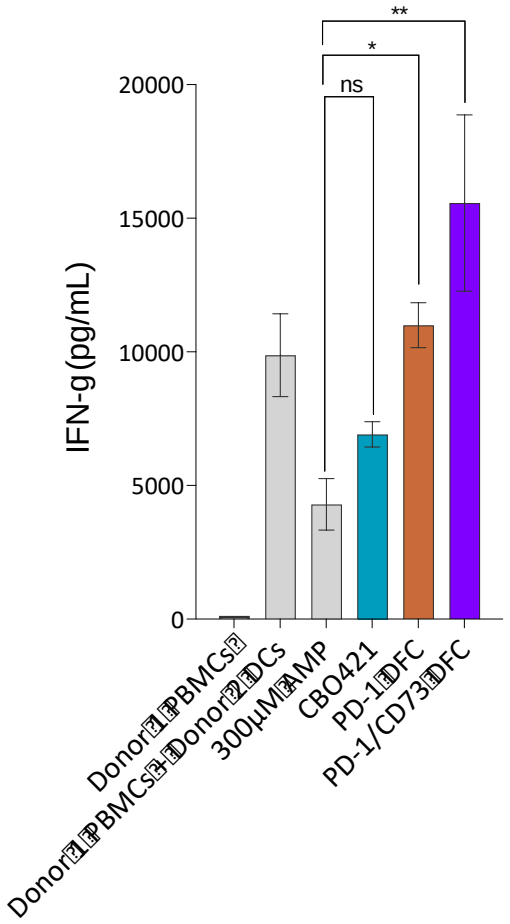


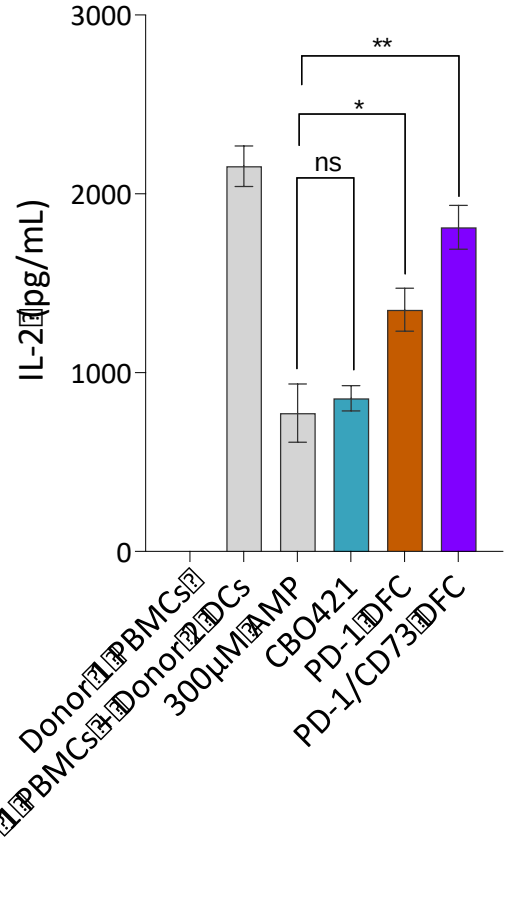
CIDARA THERAPEUTICS

PD-1/CD73 Improves Release of Immune Suppression

PD-1/CD73 DFC releases immune suppression mediated by adenosine and PD-1/L1 in 1-way mixed lymphocyte reaction assay

Dendritic cells (DCs) express CD73 and PD-L1/L2



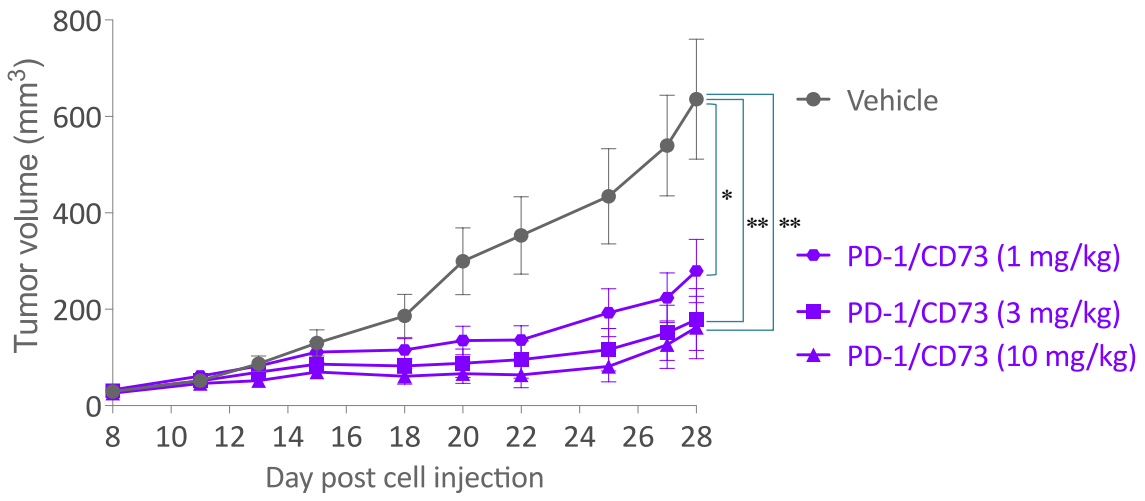


** P < 0.01
* P < 0.05

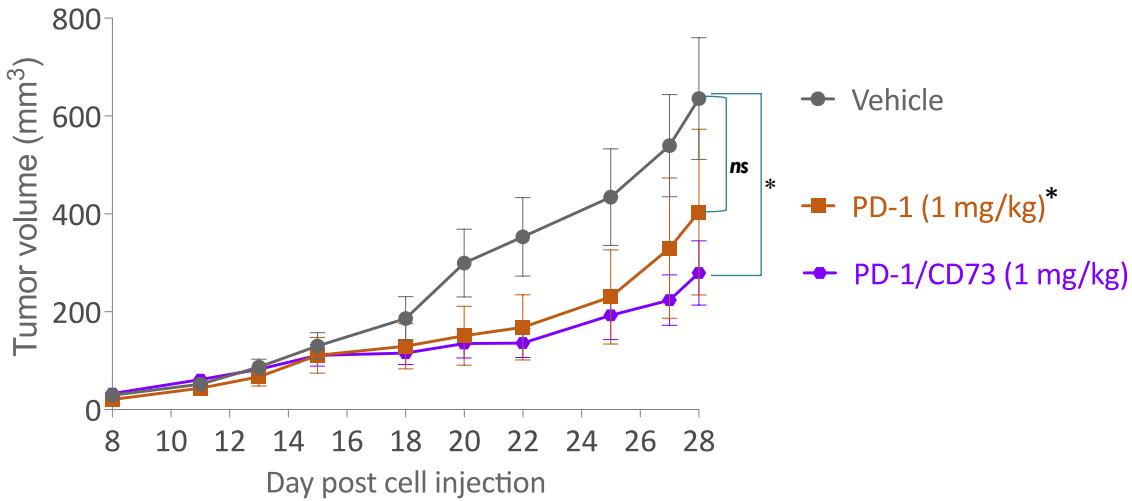
PD-1/CD73 DFC Demonstrates Robust Tumor Growth Inhibition

PD-1/CD73 DFC retains statistically significant tumor growth control at lower doses

Tumor Volume (MC38, hPD-L1)
hPD-1/hPD-L1 C57BL/6 mice – 2X weekly dosing



Tumor Volume (MC38, hPD-L1)
hPD-1/hPD-L1 C57BL/6 mice – 2X weekly dosing



**PD-1 is the proprietary PD-1 inhibitor used in the multispecific PD-1/CD73 DFC*

*** P < 0.01*
** P < 0.05*

Cidara's PD-1/CD73 DFC is Differentiated

Cidara's DFC and Akeso's bispecific mAb appear to be the most advanced PD-1/CD73 multispecific agents in development

Cidara PD-1/CD73 DFC – Preclinical

Akeso bispecific mAb (AK131) – Phase 1a opened (Dec 2023)

Attribute	PD-1/CD73 DFC	AK131	Potential for differentiation with PD-1/CD73 DFC
Size	< 125 kDa	> 200 kDa	Superior tumor penetration, better potential for high concentration formulations
Inhibition of CD73 catalytic activity	Full	Partial*	Superior inhibition of CD73
Retention of component monotherapy activity in multispecific format	Full	Partial*	Efficacy at lower doses

*Based on data presented in: *Cancer Res* (2022) 82 (12_Supplement): 5526. https://doi.org/10.1158/1538-7445.AM2022-5526

CIDARA THERAPEUTICS

PD-1/CD73 DFC Offers Advantages Over Both Cytotoxic ADCs and Bispecific mAbs*

Attribute	ADC	Bispecific mAb	DFC	Advantage of DFC
Example	Sacituzumab Govitecan Enfortumab Vedotin	PD-1/LAG3 PD-1-CTLA4 PD-1/CD73	**PD-1/CD73**	
Size	**>**150kDa	> 200 kDa	**< 125 kDa**	Smaller size allows enhanced tumor penetration
Cytotoxic component	Yes	No	**No**	No chemotherapy component allows enhanced safety profile
Linker	Cleavable	Stable	**Stable**	MOA not restricted to internalization only Not limited by lower therapeutic efficiency in case of inefficient intracellular trafficking

DFCs allow for use of combined targeting modalities (peptides and small molecules) offering unique combinations, smaller size, tunable multivalent binding and enhanced safety

*Based on data from Shim et al. Biomolecules. 2020 Feb 26;10(3):360

PD-1/CD73 Clinical Development Strategy – A First in Class Opportunity

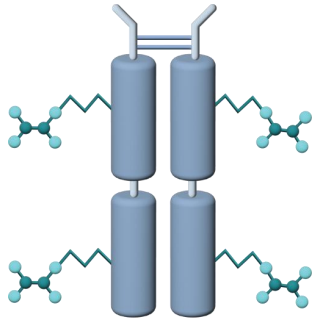
- Potential first-in-class, novel molecule: clinical trials historically enroll faster
- Single infusion of multi-specific DFC vs. sequential infusions of 2 separate therapeutics: limits infusion chair time; patient-friendly; oncology office friendly*
- Opportunity to be evaluated in earlier lines of development where PD-1/L1 agents are established SOC
- PD-1/CD73 clinical development may be expedited due to having already shown contribution of components with CBO421
- **Development candidate selection anticipated mid-2024**

*Sugalski JM, Kubal T, Mulkerin DL, et al: National Comprehensive Cancer Network Infusion Efficiency Workgroup study: Optimizing patient flow in infusion centers. J Oncol Pract 15:e458-e466, 2019

Cloudbreak CCR5 DFC Program

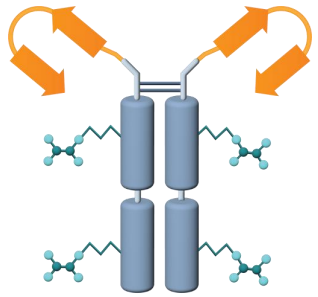
CD73: CBO421 Development Candidate

- Differentiated from existing SM and mAb CD73 inhibitors
- Complete responders in combination with PD-1 inhibitors
- Excellent safety and COGS



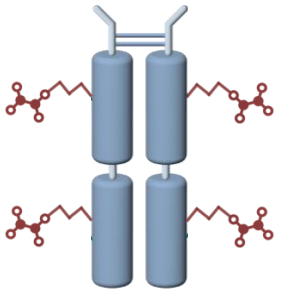
PD-1/CD73 Discovery Program

- Unique dual inhibitor of CD73 and PD-1
- Compelling preclinical data
- Potential for more efficient clinical development



CCR5 Discovery Program

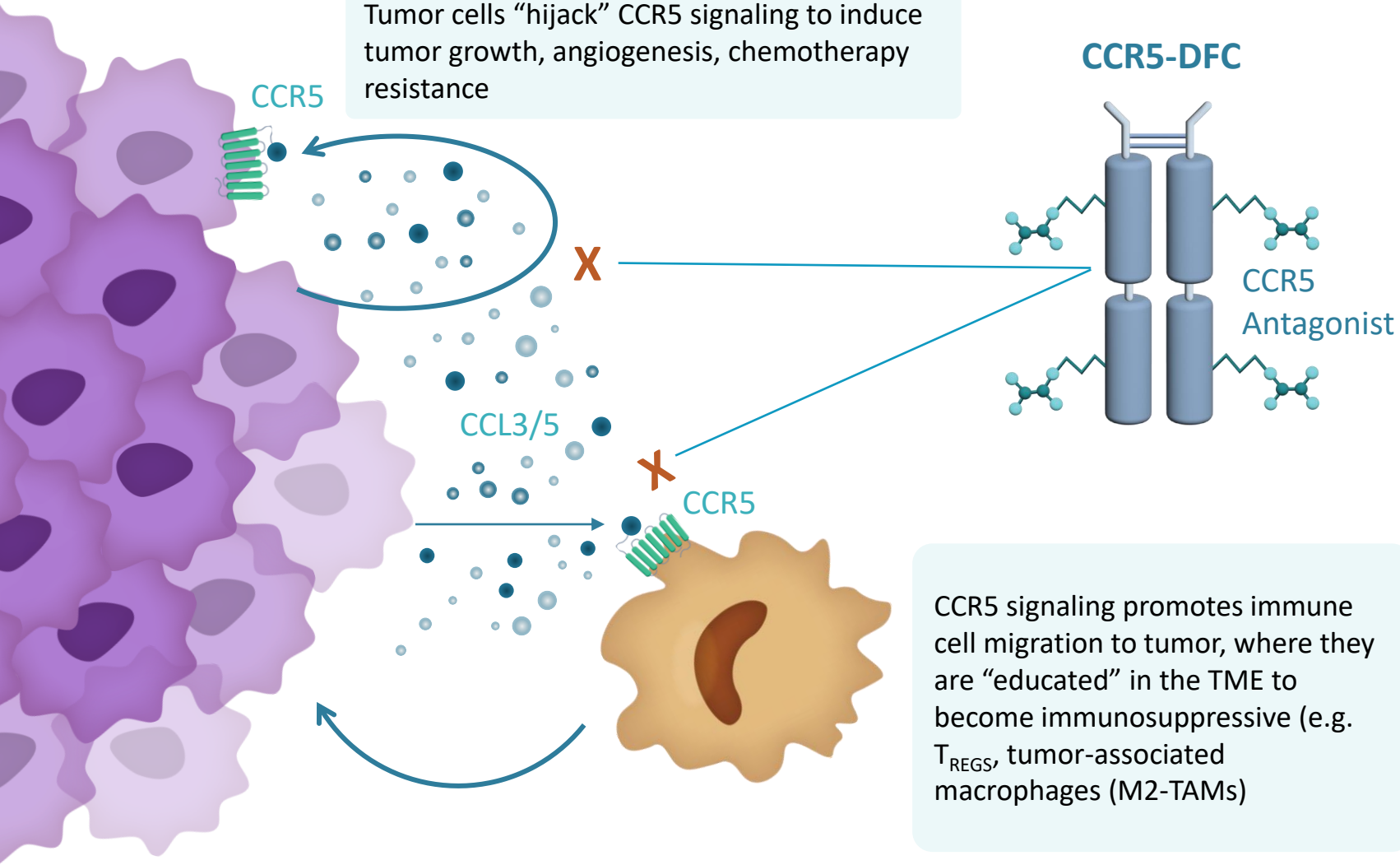
- Overcomes limitations of prior approaches
- Proof of concept data against “undruggable” target
- Potential for improved safety over SM antagonists



CIDARA THERAPEUTICS

CCR5: Compelling Target That Drives Progression of Hard-to-Treat Cancers

Tumor cells "hijack" CCR5 signaling to induce tumor growth, angiogenesis, chemotherapy resistance



CCR5 signaling promotes immune cell migration to tumor, where they are "educated" in the TME to become immunosuppressive (e.g. T_{REGS}, tumor-associated macrophages (M2-TAMs)

CCL5/CCR5 is an important driver of tumor growth, immune evasion and metastasis in several difficult to treat cancers*

- Breast
- Pancreatic
- Ovarian
- Prostate

*Int. J. Mol. Sci. 2022 https://doi.org/10.3390/ijms232214159

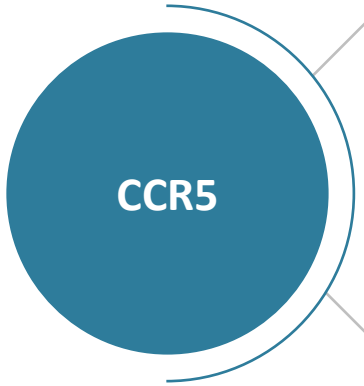
CIDARA THERAPEUTICS

CCR5 is a Compelling Oncology Target

Development candidate nomination anticipated in mid-2024

Acts as a "GPS" system that promotes metastatic disease and migration of immune suppressive cells to tumor[1,2]

- CCR5+ Circulating Tumor Cells (CTCs) and Cancer Associated Macrophage Like cells (CAMLs) promote metastatic disease – good efficacy biomarker
- Promotes Treg migration to tumor and macrophage repolarization to promote immune evasion



CCR5 signaling upregulates DNA repair, angiogenesis in tumors[3]

- CCR5 signaling reduces the effectiveness of chemotherapy
- Chemotherapy upregulates CCR5 expression

CCR5 antagonists could significantly augment chemotherapy and checkpoint blockade therapy[4]

- TNBC, ovarian, prostate and colon cancer

1. Hamid, R. et al. Cells 2023, 12, 2237. https://doi.org/10.3390/cells12182237
2. Raghavakaimal et al. Breast Cancer Research (2022) 24:35 https://doi.org/10.1186/s13058-022-01528-w
3. Jiao et al. Breast Cancer Research (2021) 23:11 https://doi.org/10.1186/s13058-021-01391-1
4. EXPERT OPINION ON THERAPEUTIC TARGETS 2021, VOL. 25, NO. 4, 311–327 https://doi.org/10.1080/14728222.2021.1902505

CIDARA THERAPEUTICS

Two Competitor CCR5 Programs with Known Liabilities

Anti-CCR5	Drug Class	Clinical Data	Notes
Leronlimab (CytoDyn)	mAb	Pooled analysis 3 studies in combo with chemotherapy TNBC (n=28): mOS 12+ months[1]	2019: Fast Track Designation for TNBC **2022: FDA clinical hold due to CMC and data quality concerns – lifted on March 1, 2024[2]**
Maraviroc (Pfizer)	Small Molecule	PICASSO-1 ph1b (n=22) in combo with pembrolizumab mCRC: modest efficacy; G3/G4 AEs noted[3]	Limited by off-target toxicity and DDI potential[4] **No further development planned in oncology**

CCR5 monotherapy and combination DFCs

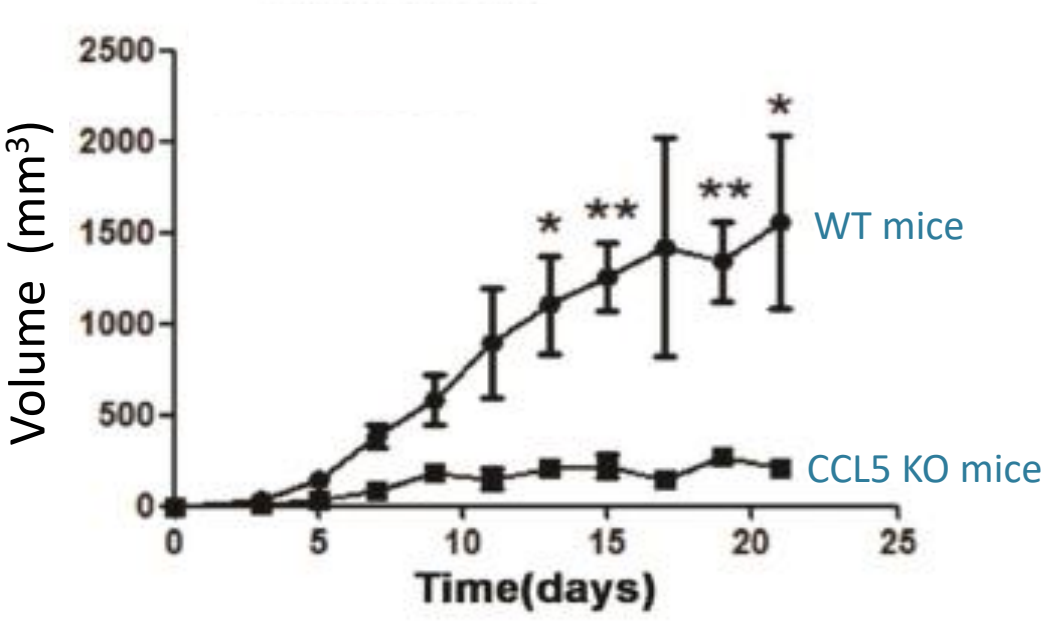
- Enhanced safety profile and compatibility with chemotherapy vs small molecule antagonists
- Potential as a combination agent with SOC chemotherapy in TNBC
- Potential for development of first in class multispecific therapies
- Potential to develop for long-acting HIV retroviral therapy (with partner)

1. Journal of Clinical Oncology Volume 40, Number 16_suppl https://doi.org/10.1200/JCO.2022.40.16_suppl.e130
2. https://www.cytodyn.com/newsroom/press-releases/detail/618/cytodyn-announces-fda-has-lifted-clinical-hold
3. https://doi.org/10.1016/j.ejca.2022.03.017
4. Drug Metabolism and Disposition May 2019, 47 (5) 493-503; DOI: https://doi.org/10.1124/dmd.118.085241

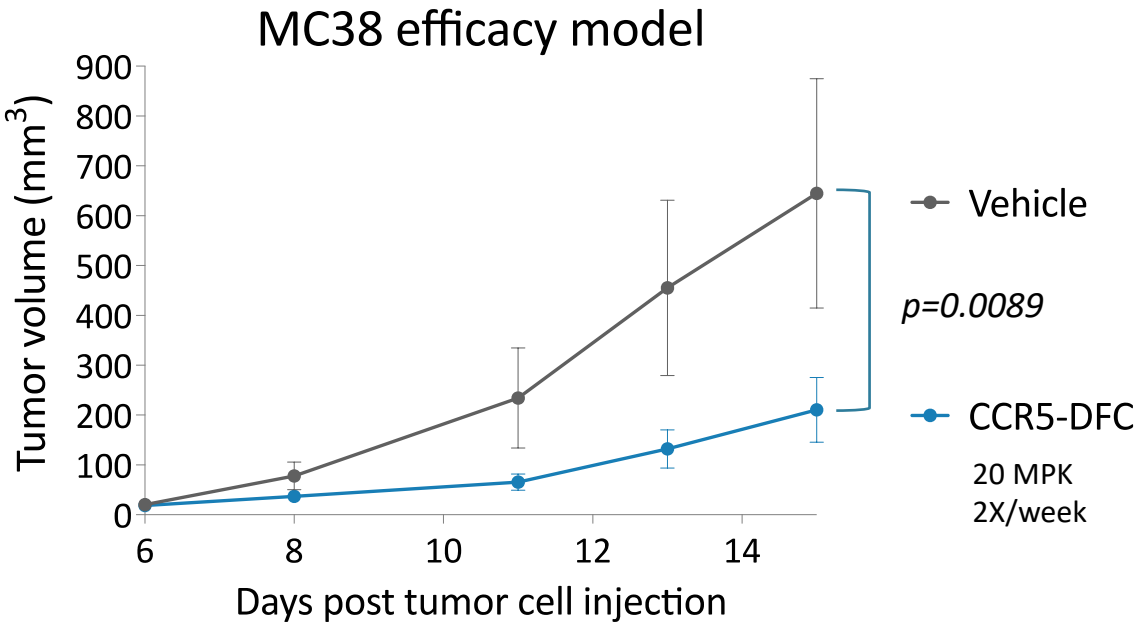
CIDARA THERAPEUTICS

CCR5 DFC Induces Tumor Reduction Commensurate With a CCL5 KO Model in Colorectal Carcinoma

MC38 tumors are unable to proliferate in CCL5 KO mice (MC38 = murine colorectal carcinoma)



CCR5 DFC treatment potently inhibits tumor growth



First CCR5 targeting therapy designed for oncology

1. Zhang et al. Cell Death and Disease (2018) 9:766 DOI 10.1038/s41419-018-0796-2

CIDARA THERAPEUTICS

Cloudbreak Oncology DFC Programs

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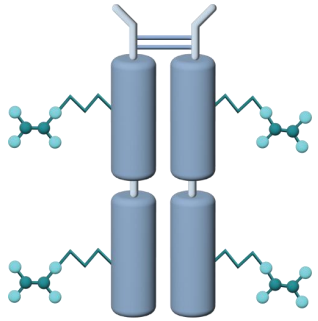

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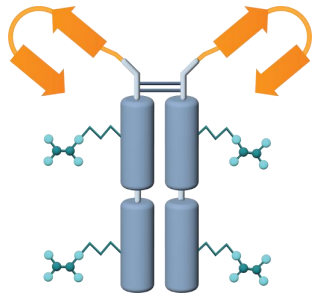

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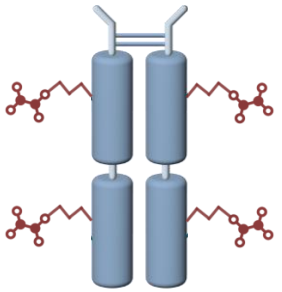


CIDARA THERAPEUTICS

Financial Overview

Important Information	December 31, 2023[1]
Cash and Cash Equivalents	$35.8M
Common Stock Outstanding	90,601,999
Common Equivalent Shares Outstanding[2]	111,646,719

Summary Consolidated *Cash Flow Information*	**Rolling two-quarter period ended December 31, 2023[3]**
Operating Cash Burn	$(31.9)M
Mundipharma Reimbursements	$0.4M
Janssen Reimbursements & Milestones	$15.4M
Melinta Milestones & Sales Receipts	$1.5M
Net Cash Provided by Operations, Investing & Financing (excluding ATM Proceeds)	$0.6M
ATM Proceeds Less Offering Costs	$0.0M
Net Cash Decrease	$(14.6)M

On April 24, 2024, the Company raised an additional $240 million in gross proceeds in a private placement led by RA Capital Management with significant participation by Bain Capital Life Sciences as well as BVF Partners and Canaan Partners.

1. Information listed here is as of December 31, 2023 (as disclosed in our Form 10-K).
2. Includes (i) 90,601,999 shares of common stock and (ii) 21,044,720 shares of common stock issuable upon the conversion of Series X Convertible Preferred stock, both as of December 31, 2023. Each share of Series X Convertible Preferred is convertible into 10 shares of common stock.
3. Amounts reflect a rolling two-quarter period ending on the date noted. Amounts shown are historical and may not be indicative of future results.

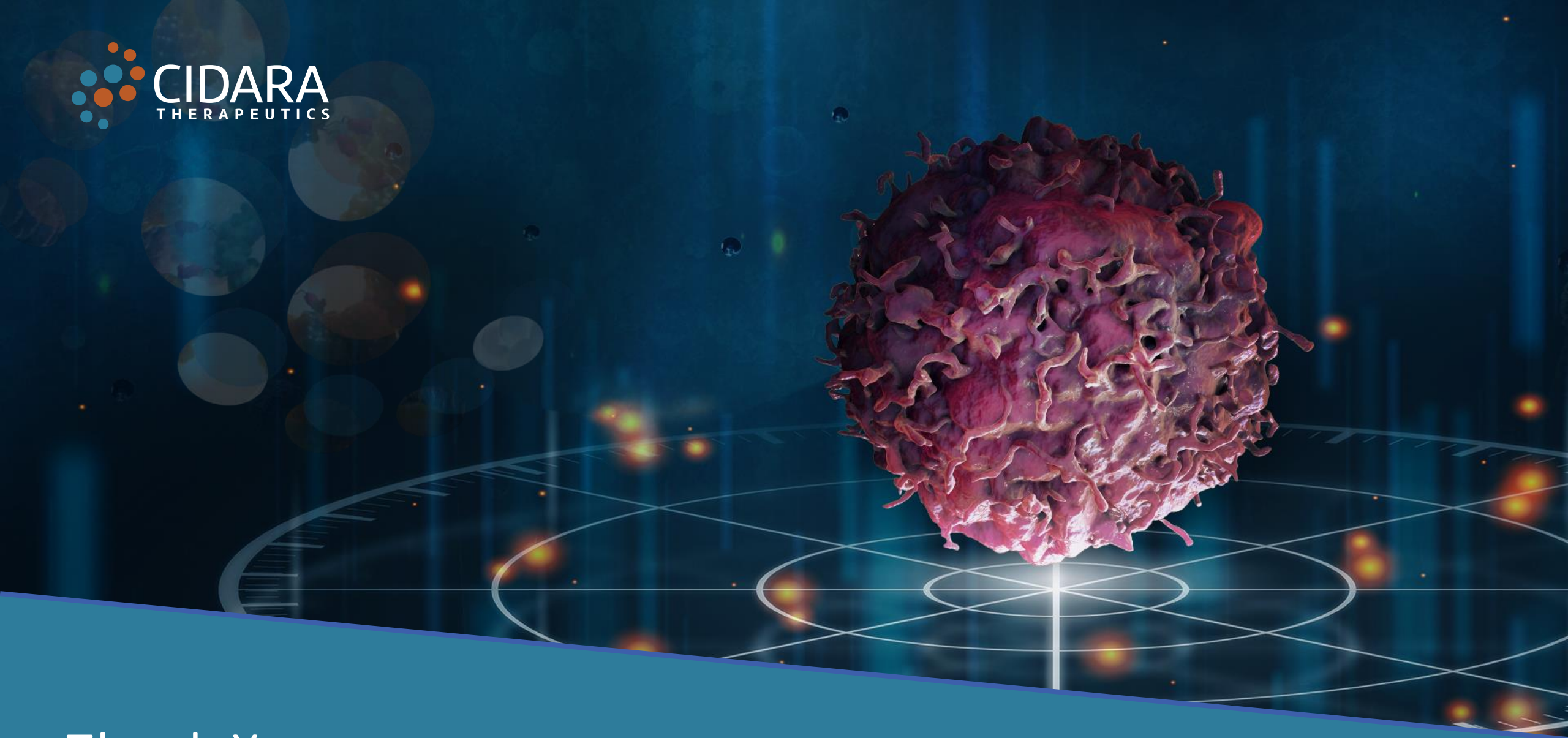
CIDARA THERAPEUTICS

Capital Structure and Share Information

All shares in millions	12/31/23 [1] (as filed 10-K)	4/24/24 Split -Adjusted (1:20)[5]	4/24/24 PIPE Financing (Split Adjusted 1:20)[5]	4/24/24 Pro-forma PIPE Financing & Reverse Split (1:20)[5]
Common Shares Outstanding	90,601,999	4,530,100		4,530,100
Series X Convertible Preferred stock (as converted) [2]	21,044,720	1,052,236		1,052,236
Common stock options, RSUs and PRSUs issued and outstanding	12,764,068	638,203		638,203
Fully Diluted Common Shares Outstanding	124,410,787	6,220,539		6,220,539
240,000 shares of Series A Convertible Voting Preferred Stock (as converted) [3]			16,901,408	16,901,408
Pro-forma Fully Diluted Common Shares Outstanding				**23,121,948[6]**
Average VWAP Post Reverse Spilt 4/24/24 – Current[4]				**$13.73**
Implied Pro-forma Fully Diluted Equity Value / Market Cap				**$317.5 million**
Cash and Cash Equivalents	$35.8 million		$240.0 million	**$275.8 million[7]**
Debt	-			**-**

1. Information listed here is as of December 31, 2023 (as disclosed in our Form 10-K).
2. 21,044,720 shares of common stock issuable upon the conversion of Series X Convertible Preferred stock, both as of December 31, 2023. Each share of Series X Convertible Preferred is convertible into 10 shares of common stock.
3. 240,000 shares of Series A Convertible Voting Preferred Stock, par value $0.0001 per share (the "Series A Preferred Stock"). Each share of Series A Preferred Stock is, subject to stockholder approval and certain beneficial ownership conversion limitations, automatically convertible into shares of common stock, par value $0.0001 per share, at a conversion price of $14.20 per share, rounded down to the nearest whole share.
4. As of 4/29/24.
5. Reverse Split-adjusted numbers are approximate based on a ratio of 1:20.
6. Subject to stockholder approval of an increase in authorized common stock.
7. Pro-forma as of 12/31/23 and PIPE financing on 4/24/24.

CIDARA THERAPEUTICS



Thank You

May 2024
NASDAQ: CDTX