CIDARA THERAPEUTICS

R&D Day

SEPTEMBER 21, 2023

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 rezafungin, CD388, and other antiviral and oncology product candidates from the Cloudbreak platform; Cidara’s potential ability to achieve milestones under its respective collaborations with Melinta, Mundipharma and Janssen, and receipt of the related milestone payments; 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 Form 10-K as most recently filed with the United States Securities and Exchange Commission ("SEC“’) under the heading “Risk Factors.”

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Agenda

Topic	Presenter
Introduction	Jeffrey Stein, PhD
Drug-Fc Conjugates (DFCs) – a novel and clinically proven therapeutic class	Jeffrey Stein, PhD
Update on JNJ 0953 (CD388): a clinical stage universal influenza DFC in Phase 2 trials	Jeffrey Stein, PhD
Cloudbreak Pipeline Discussion - translating success in infectious disease to oncology	Nicole Davarpanah, MD, JD
Who is left behind by cancer immunotherapy?	Stephen Schoenberger, PhD
Harnessing DFCs to create targeted therapies that unleash the immune system	Les Tari, PhD and Nicole Davarpanah, MD, JD
Perspective: The potential of DFCs to transform precision oncology	Ezra Cohen, MD
Wrap up and Q&A	Jeffrey Stein, PhD

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Today's Speakers

Jeffrey Stein, PhD
President & CEO
Cidara Therapeutics

Stephen Schoenberger, PhD
Professor, Immunology
La Jolla Institute for Immunology

Ezra Cohen, MD
CMO, Oncology
Tempus

Les Tari, PhD
CSO
Cidara Therapeutics

Nicole Davarpanah, MD, JD
SVP, Translational R&D
Cidara Therapeutics

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Cidara Platforms: Rezafungin & Cloudbreak

REZAFUNGIN

- Echinocandin antifungal treatment & prevention
- Approved by FDA in March 2023

Program	Indications	IND-Enab.	Phase 1	Phase 2	Phase 3	Approved	Collaborations
REZAFUNGIN	Treatment of Candidemia and Invasive Candidiasis						melinta therapeutics (U.S) / mundipharma (Ex-US/Ex-Japan)
REZAFUNGIN	Prevention of Invasive Fungal Disease in Blood & Marrow Transplant Patients						melinta therapeutics (U.S) / mundipharma (Ex-US/Ex-Japan)

CLOUDBREAK

- Novel immunotherapy platform: antiviral & oncology
- Clinical stage (influenza) – CD388; Phase 2a data 11/23
- CD73 Development candidate
- New program disclosures

Program	Indications	Discovery	Preclinical	IND-Enab.	Phase 1	Phase 2	Collaborations
CD388	Prevention of Seasonal Influenza						Janssen (Worldwide License)
CBO-421 (CD73)	Solid Tumors						
CD73/PD-1	Solid Tumors						
CCR5	Solid Tumors						

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melinta therapeutics

Launched July 31, 2023

Once-weekly

REZZAYO®

(rezafungin for injection)

Cloudbreak Programs

REZAFUNGIN

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CLOUDBREAK

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CD73/PD-1	Solid Tumors						
CCR5	Solid Tumors						

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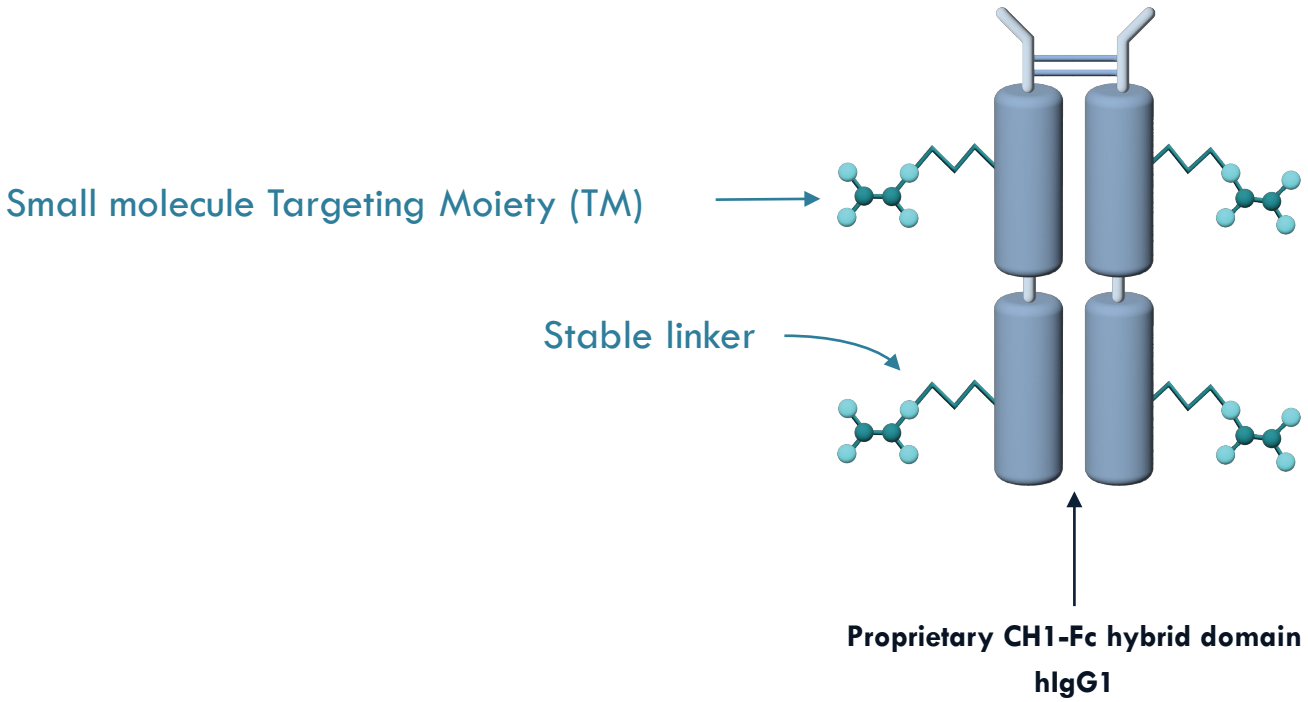
Cloudbreak® Creates a New Class of Drug Conjugates: "DFCs"

DRUG Fc CONJUGATE



DFCs are designed to engage extracellular targets

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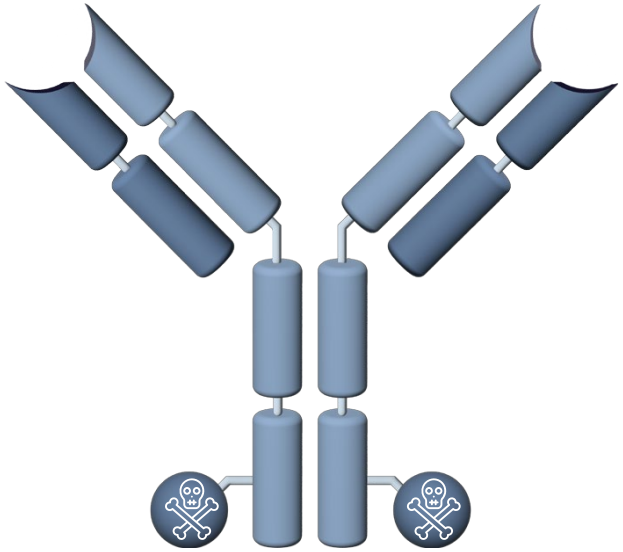
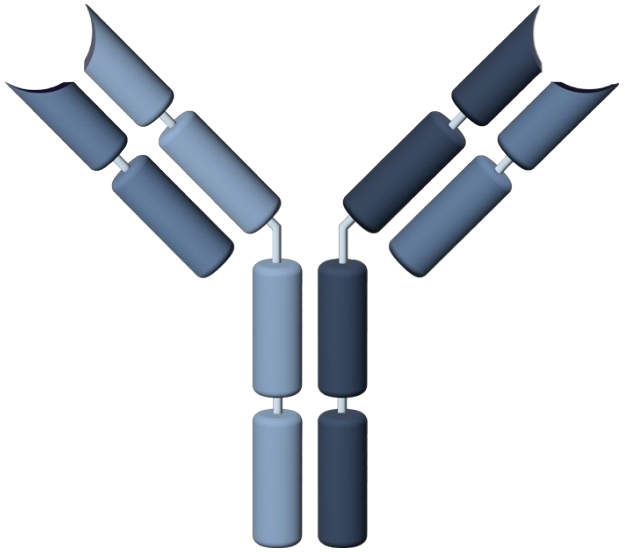
DFCs are Fundamentally Different from ADCs and Bispecifics

ADC

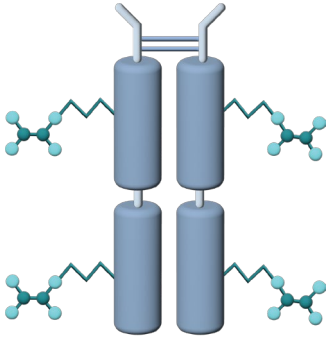


SM Cytotoxin

Bispecific mAb



DFC



SM drug conjugate

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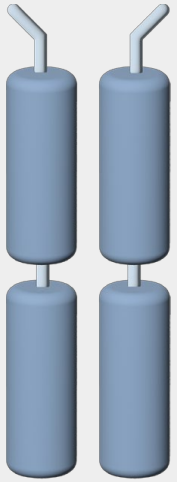
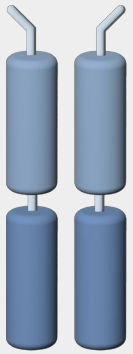
Fc Moiety is Tailored to Specific Indications

Fc MOIETY



Wild type

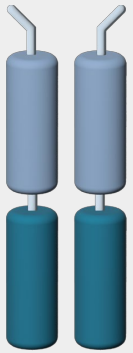
ANTIVIRALS



PK extended Fc

- IgG1
- Enhanced FcRn binding
- Increased half-life compared to wild-type

ONCOLOGY



Immune silent Fc

- IgG1 with reduced immune effector function

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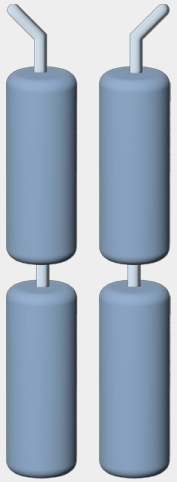
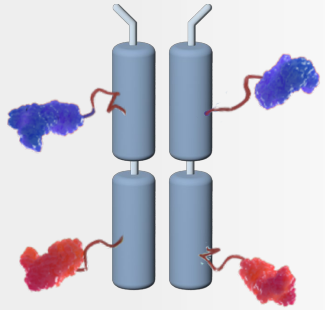
Different Targeting Moieties (TMs) Attach to the Fc Moiety

Fc MOIETY



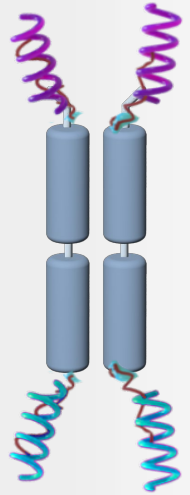
SMALL MOLECULES (SM)

Directed against surface targets
Example: Neuraminidase in CD388



PEPTIDE FUSIONS

Inhibit protein-protein interactions
Example: PD-1 peptide



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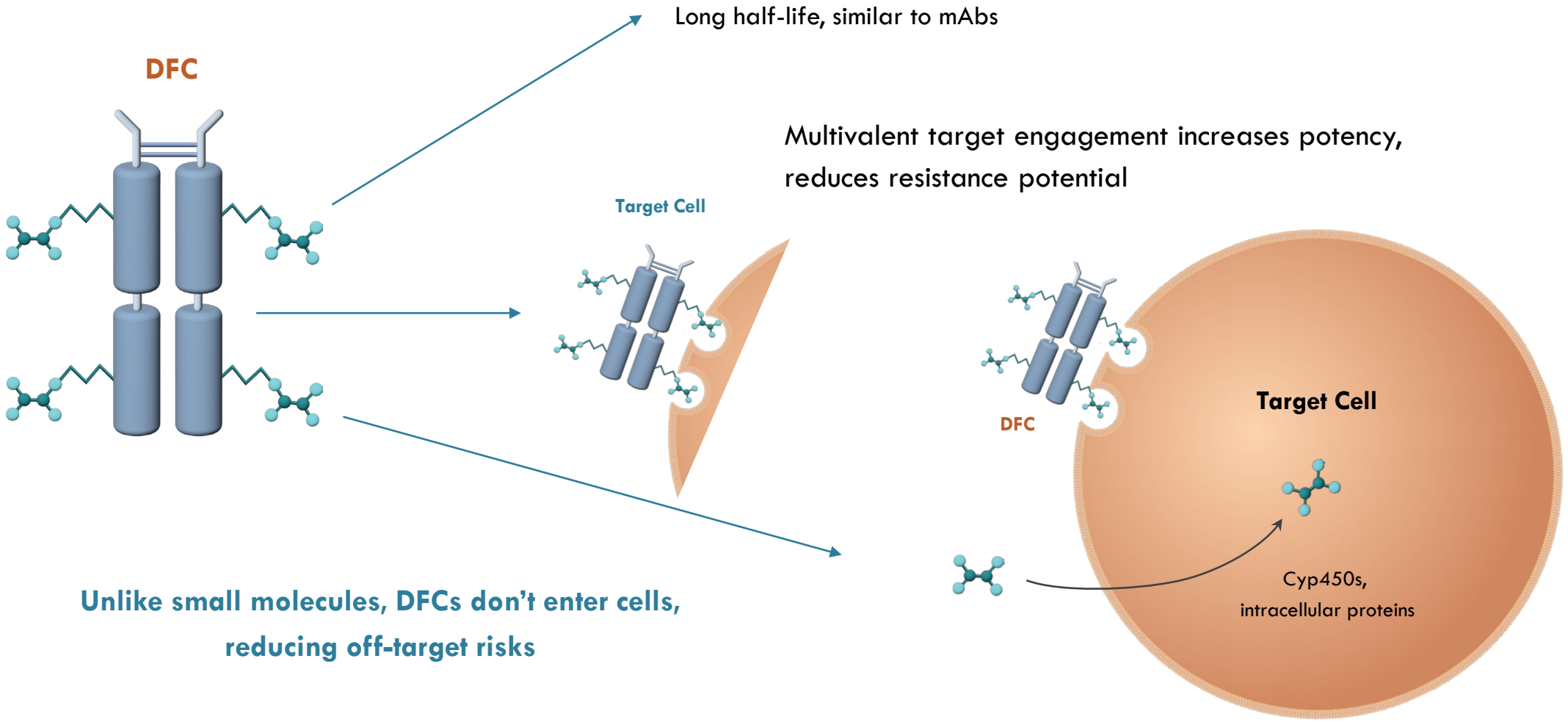
DFCs Can Improve Small Molecule Drug Potency and Safety



Unlike small molecules, DFCs don't enter cells, reducing off-target risks

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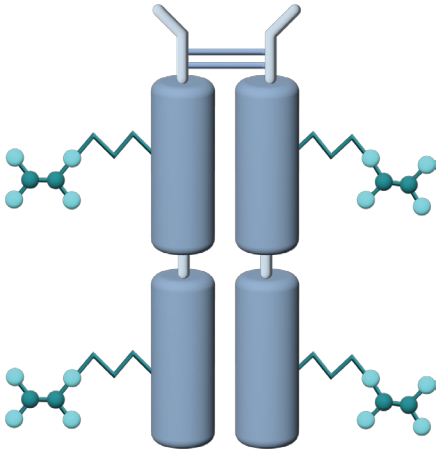
DFCs Have Reduced Potential for Off-Target Toxicity

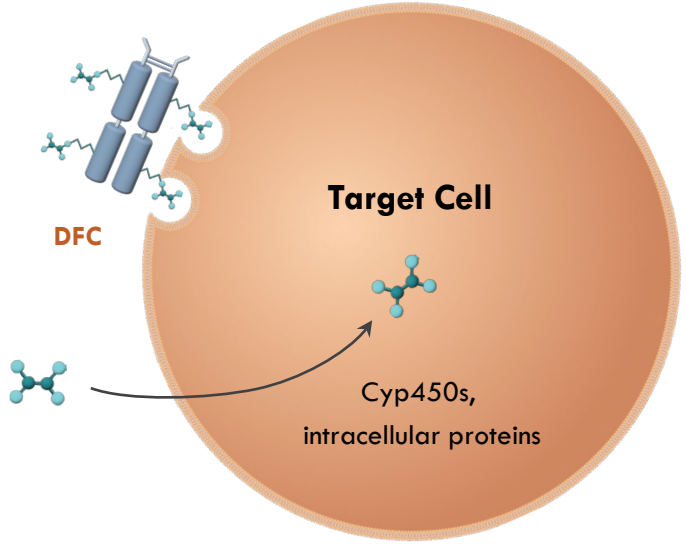
DFCs can exploit drug targets that are difficult to drug with small molecules (SMs)

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

Chemokine receptor DFC (CCR-DFC)



DFCs do not enter cells



DFCs exhibit better safety than SMs

% hERG inhibition @10μM		
SM		77
CCR-DFC		< 5

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

*EXPERT OPINION ON INVESTIGATIONAL DRUGS, 2016 VOL. 25, NO. 12, 1377–1392 http://dx.doi.org/10.1080/13543784.2016.1254615

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CD388 Phase 2A Data Confirm the TPP

INFLUENZA

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

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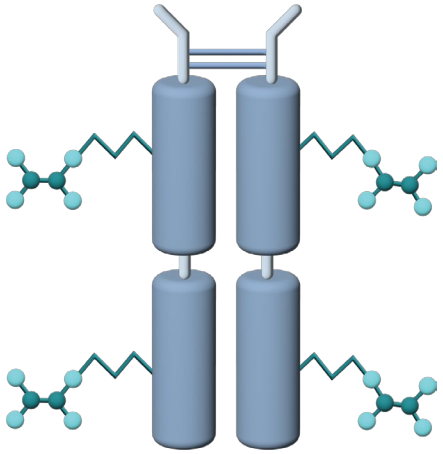
CD388 is being developed for pre-exposure prophylaxis

Neuraminidase inhibitor



Single dose / ~4-6 months

Successful Phase 2a interim data*

Data available at https://www.cidara.com/cloudbreak/influenza/ and at https://clinicaltrials.gov/ct2/show/NCT05285137

* https://www.cidara.com/news/cidara-therapeutics-announces-promising-interim-phase-2a-data-assessing-the-safety-and-efficacy-of-a-single-dose-of-cd388-in-an-influenza-challenge-model/

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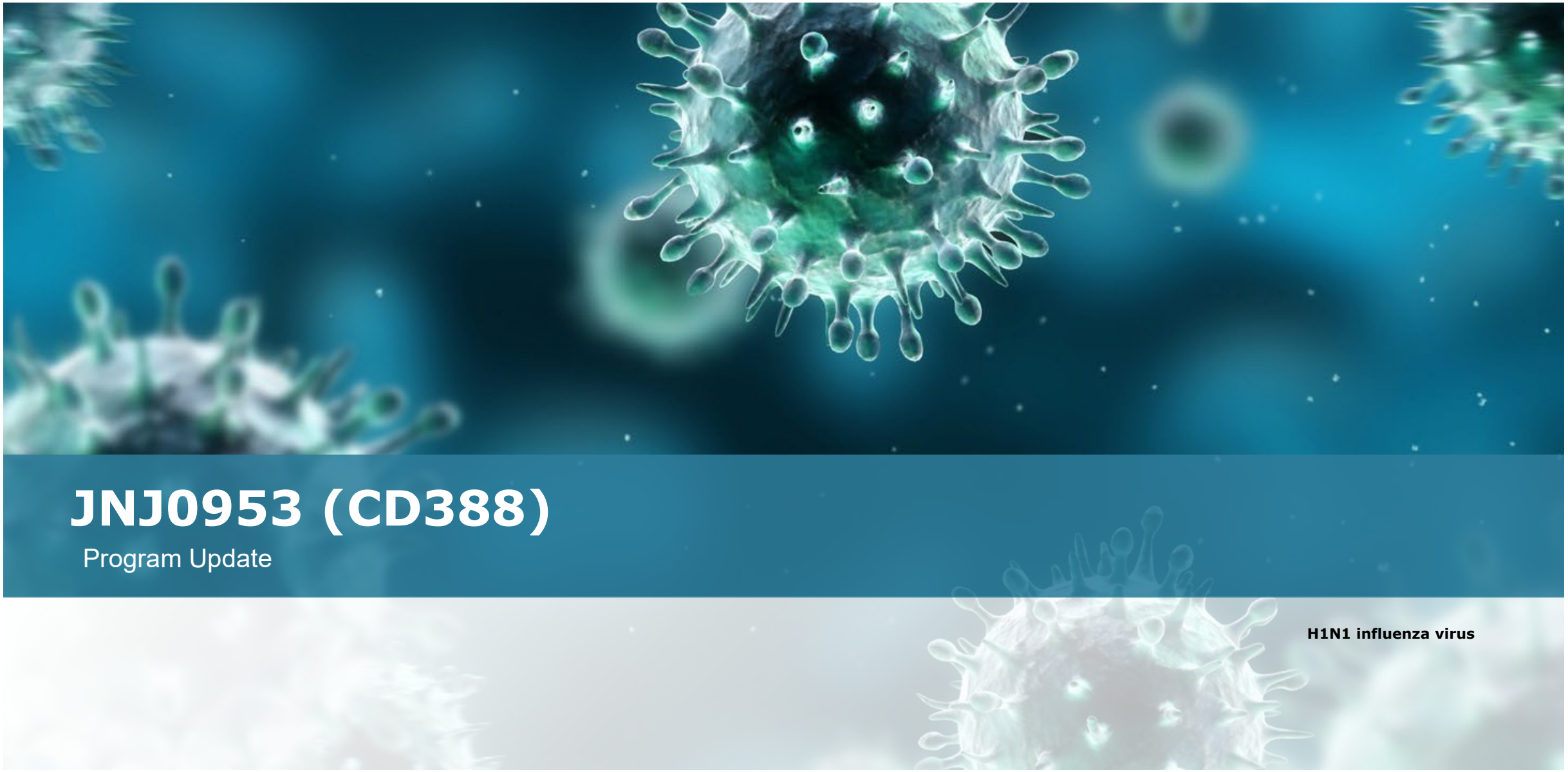


JNJ0953 (CD388)

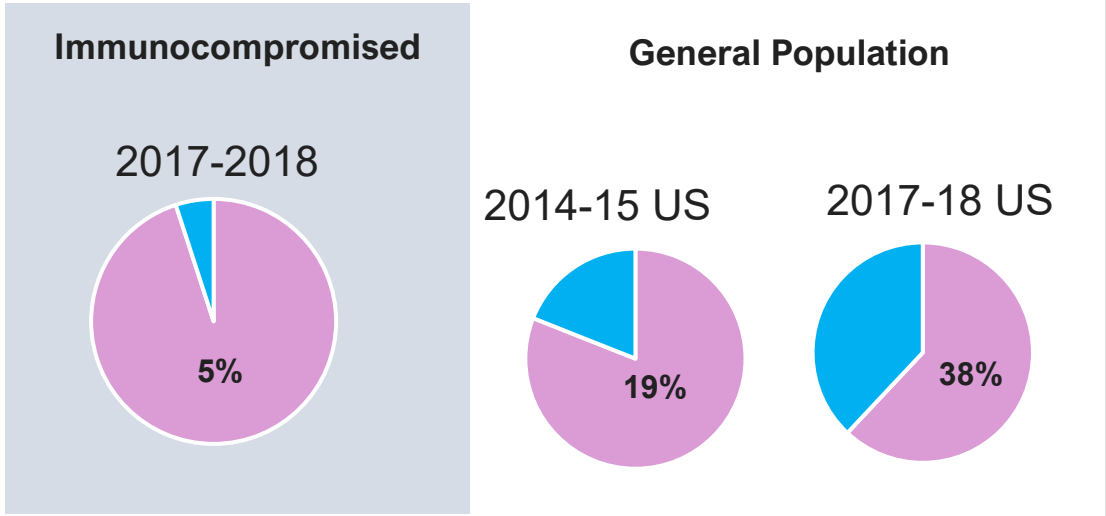
Program Update

H1N1 influenza virus

Despite influenza vaccination, a high disease burden and unmet need remains for influenza prevention in specific groups

Influenza Worldwide: 650,000 deaths/year

Influenza Vaccine Efficacy varies from year to year due to antigenic shift/drift and is consistently low in immunocompromised patients

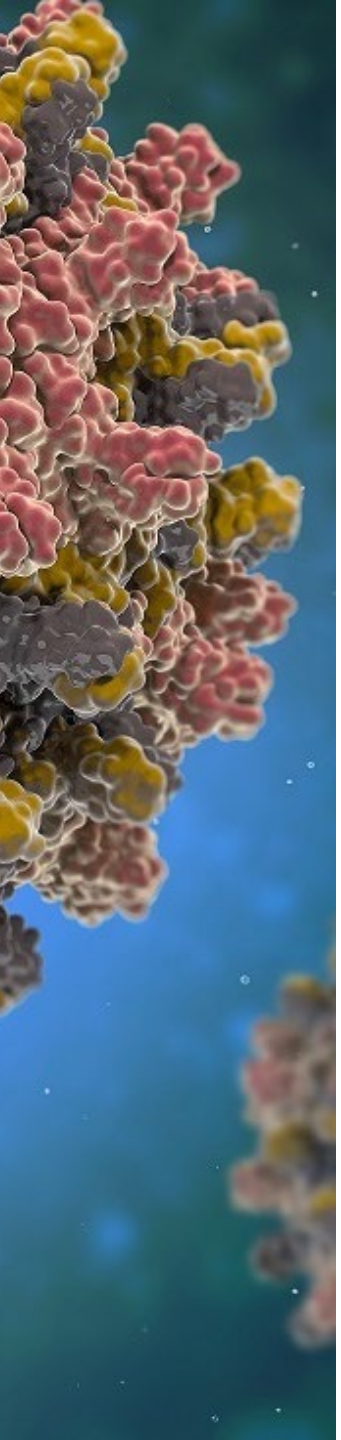


Patients with selected comorbidities are at increased risk of complications/hospitalizations of influenza-like illness despite vaccination

Unadjusted ORs (all-cause hospitalizations 30 days after influenza-like illness) against healthy controls in vaccinated individuals, Optum DS study 2022

*PrEP = Pre exposure prophylaxis

Source: https://www.cdc.gov/flu/professionals/vaccination/effectiveness-studies.htm; https://gis.cdc.gov/grasp/fluview/FluHospChars.html; Hughes et al. cid 2021:73



Objectives of Early Clinical Development

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

JNJ0953 Early clinical development studies

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

Primary endpoint: safety and tolerability

Secondary endpoints: plasma pharmacokinetics

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

Primary endpoint: safety and tolerability

Secondary endpoints: plasma pharmacokinetics

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

Primary endpoint: prophylactic efficacy: reduction of area under the viral load-time curve (VL-AUC) after influenza challenge

Secondary endpoints:

- Prophylactic efficacy: additional endpoints
- Safety and tolerability

JNJ-0953 was safe and well-tolerated up to 900 mg

Aggregated Clinical Safety Data through end August 2023 from FIH, Japanese Bridging (JBS) and Human Challenge (HCS) studies

Number of participants that received one dose of JNJ-0953 in Phase 1 and Phase 2a studies (as of Aug 2023)

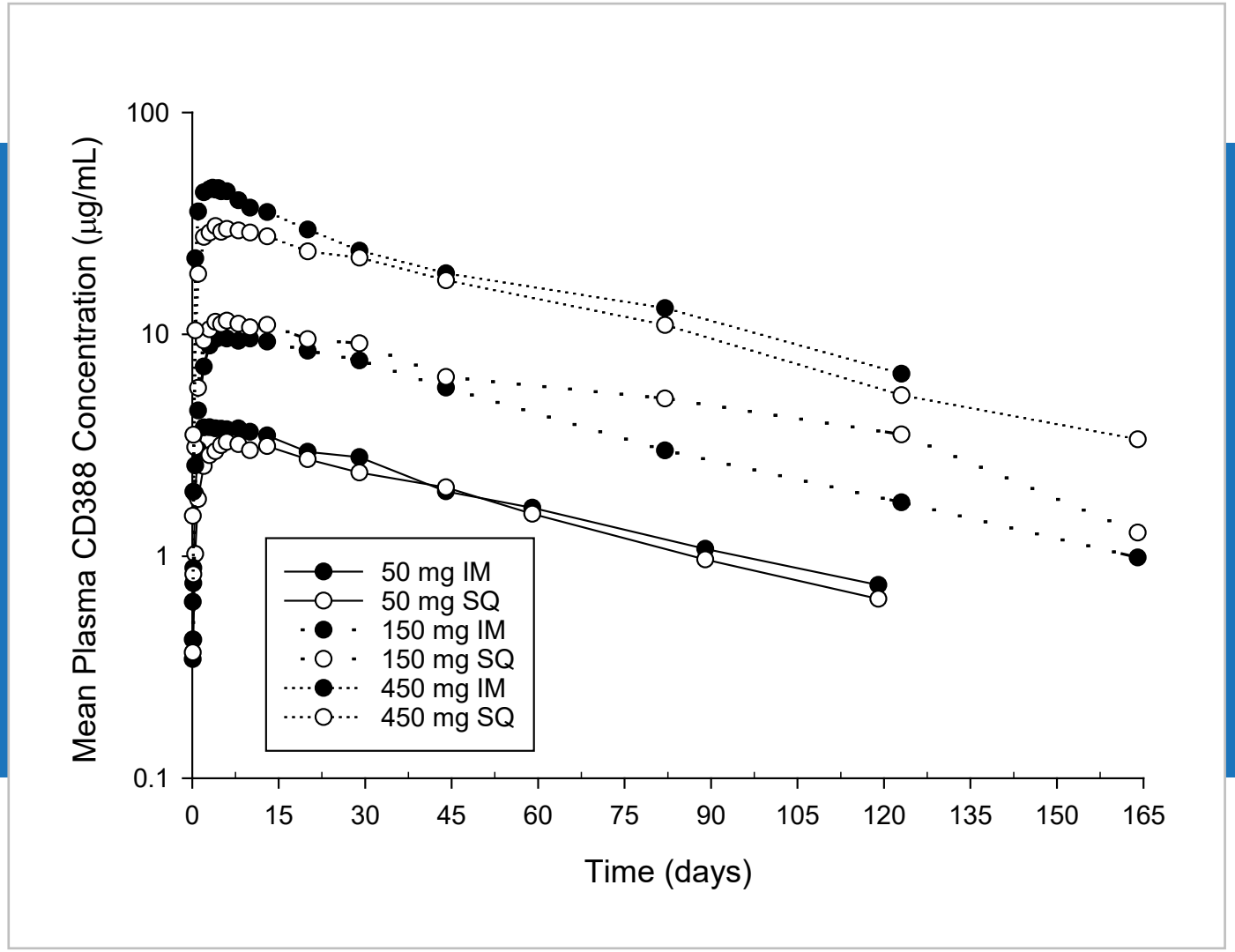
	FIH	JBS	HCS	Total
50 mg	18	7	2	27
150 mg	18	7	28	53
450 mg	18	7	0	25
900 mg	9	0	0	9
All Doses	63	21	30	**114**

The JNJ-0953 PK profile is that of a long-acting compound, potentially enabling seasonal PrEP

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 were 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

The JNJ-0953 PK profile is that of a long-acting compound, potentially enabling seasonal PrEP

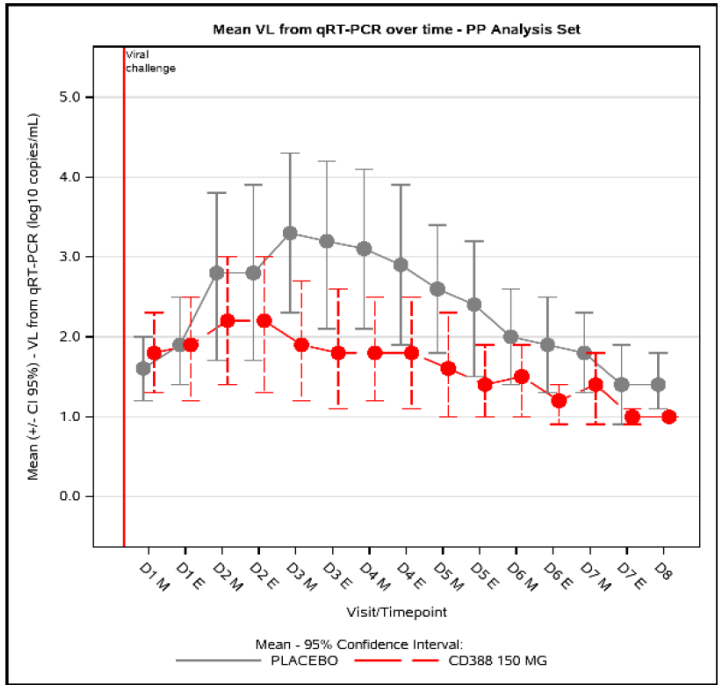


JNJ-0953 demonstrates prophylactic efficacy in reducing viral replication in the URT and the incidence of PCR-confirmed influenza infection

Phase 2a Prophylactic Efficacy IA Results

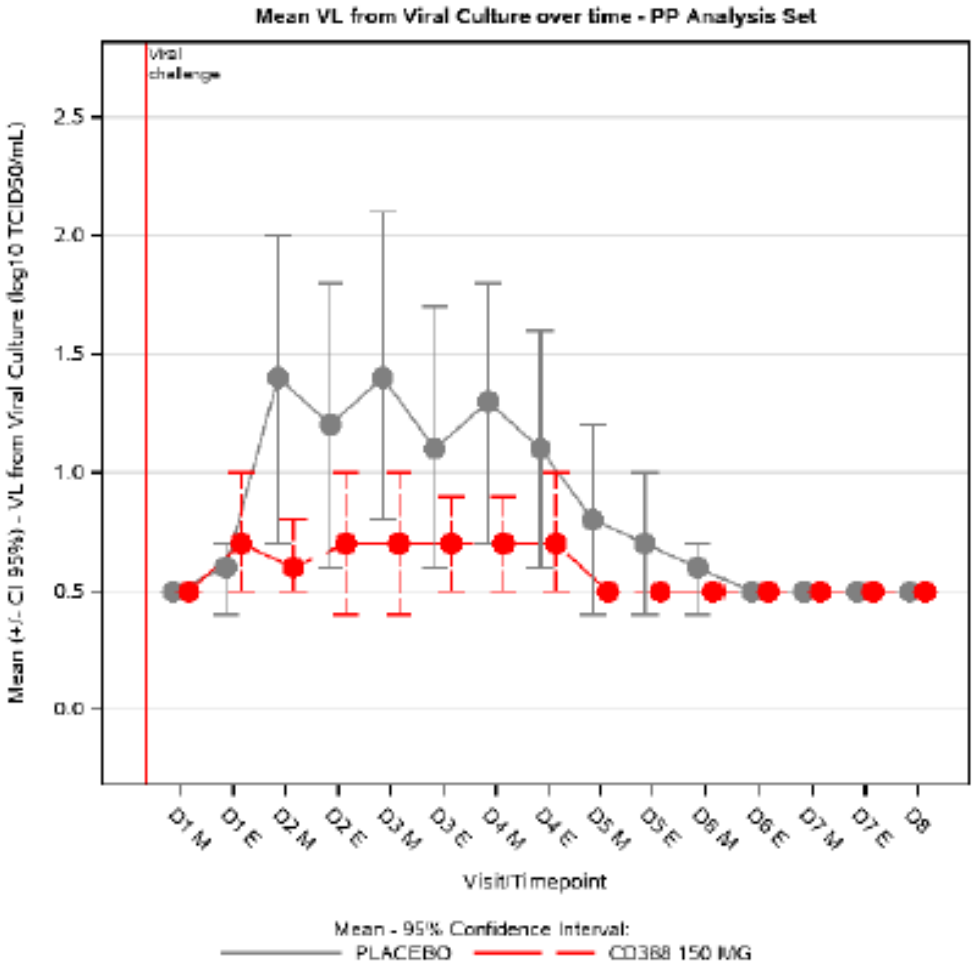
endpoint	Placebo N=28	JNJ-0953 150 mg N=28	P-value
qRT-PCR confirmed influenza infection *(“attack rate” see Placebo data)*	14 (50%)	6 (21%)	0,0248
qRT-PCR confirmed symptomatic influenza infection	9 (32%)	4 (14%)	0,1023
qRT-PCR confirmed moderately to severe symptomatic influenza infection	7 (25%)	3 (11%)	0,1477

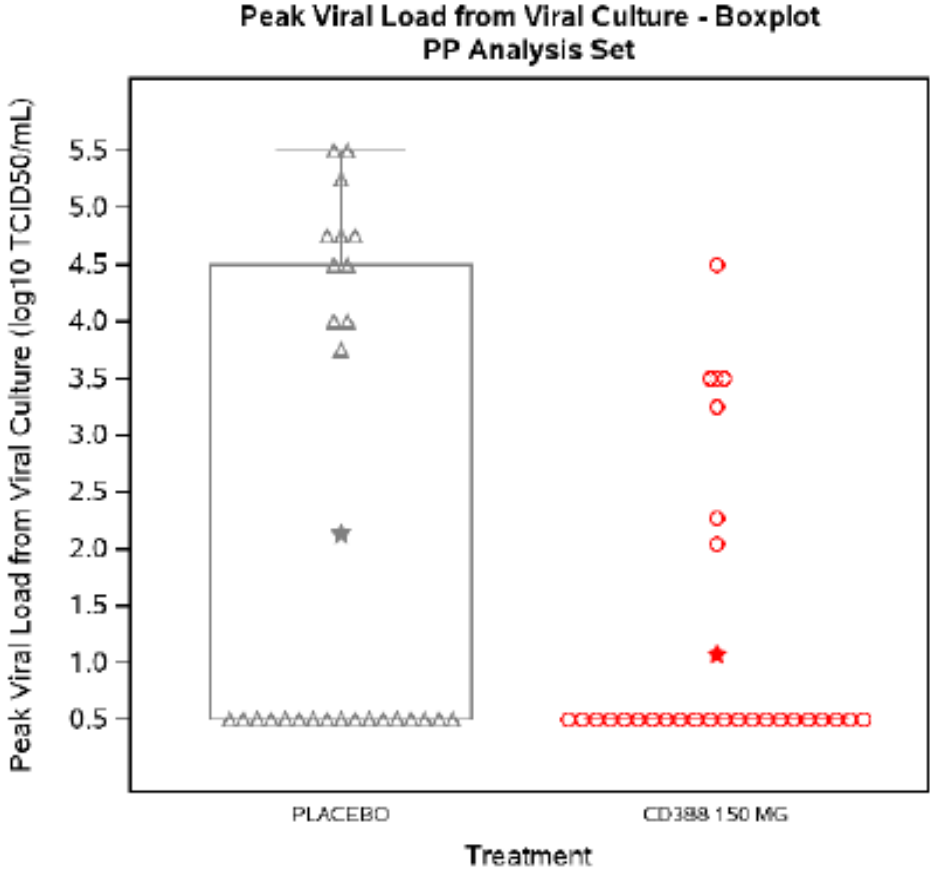
Primary endpoint: AUC viral load-time_ qRT-PCR



One sided p-value Wilcoxon rank sum test: 0.0390

Viral culture data confirmed efficacy seen in early analyses





Summary

First of its class antiviral conjugate, targeting influenza PrEP

High recognition of unmet need

Preclinical and clinical data show efficacy and long-acting properties

- Activity against all tested influenza viruses
- FIH studies prove long-acting properties
- Human Challenge Study demonstrates efficacy in preventing influenza infection
- Safe and well tolerated

CD388 (JNJ-0953) Program Update

CIDARA THERAPEUTICS ANNOUNCES JANSSEN'S ELECTION TO PROCEED UNDER ITS LICENSE AGREEMENT RELATING TO NOVEL DRUG-FC CONJUGATES TARGETING INFLUENZA

September 6, 2023

Decision follows promising interim efficacy and safety data from ongoing Phase 1 and 2a trials

Cidara to receive a $7 million milestone payment and is eligible to receive an additional $685 million in milestones, plus royalties

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A Track Record of Forging Partnerships

Over $1.8 Billion in Potential Value* from Existing Licenses

Rezafungin

2019 — mundipharma

Rights: ex-US / Japan

~$568M

Phase 2 data

- $30M upfront
- $9M equity investment
- $42M in development support
- $487M clin/reg/comm milestones
- Double-digit royalties in the teens

2022 — melinta therapeutics

Rights: US

~$460M

Phase 3 data/ NDA Filed

- $30M upfront
- $60M regulatory milestones
- $370M commercial milestones
- Low double-digit to mid-teens royalties

Cloudbreak (CD388)

2021 — janssen

Program:
Influenza--CD388 | Rights: Global

~$780M

Preclinical data

- $27M upfront
- $58M in R&D support
- $695M clin/reg/comm milestones
- Mid to high single digit royalties

* Does not include royalties.

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Cloudbreak Oncology DFC Programs

CD73: CBO421 Development Candidate

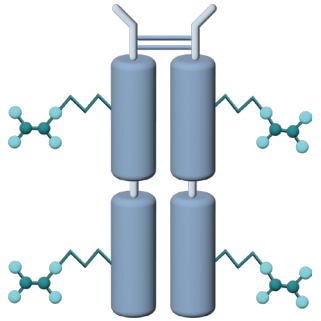
- Potential Best-in-Class
- Complete responders in combination with PD-1 inhibitors
- Excellent safety and COGS

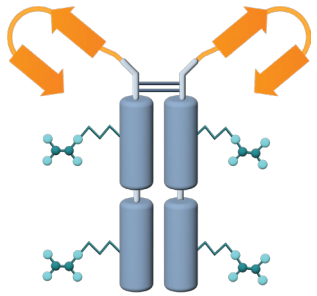
CD73/PD-1 Discovery Program

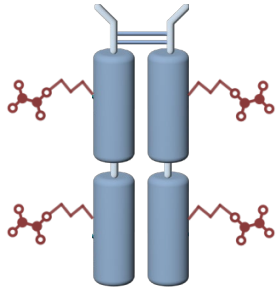
- Unprecedented dual inhibitor of CD73 and PD-1
- Compelling preclinical data
- Potential for more efficient clinical development

CCR5 Discovery Program

- Overcomes limitations of prior approaches
- Proof of concept data against “undruggable” target
- Opportunity to expand to other CCR targets







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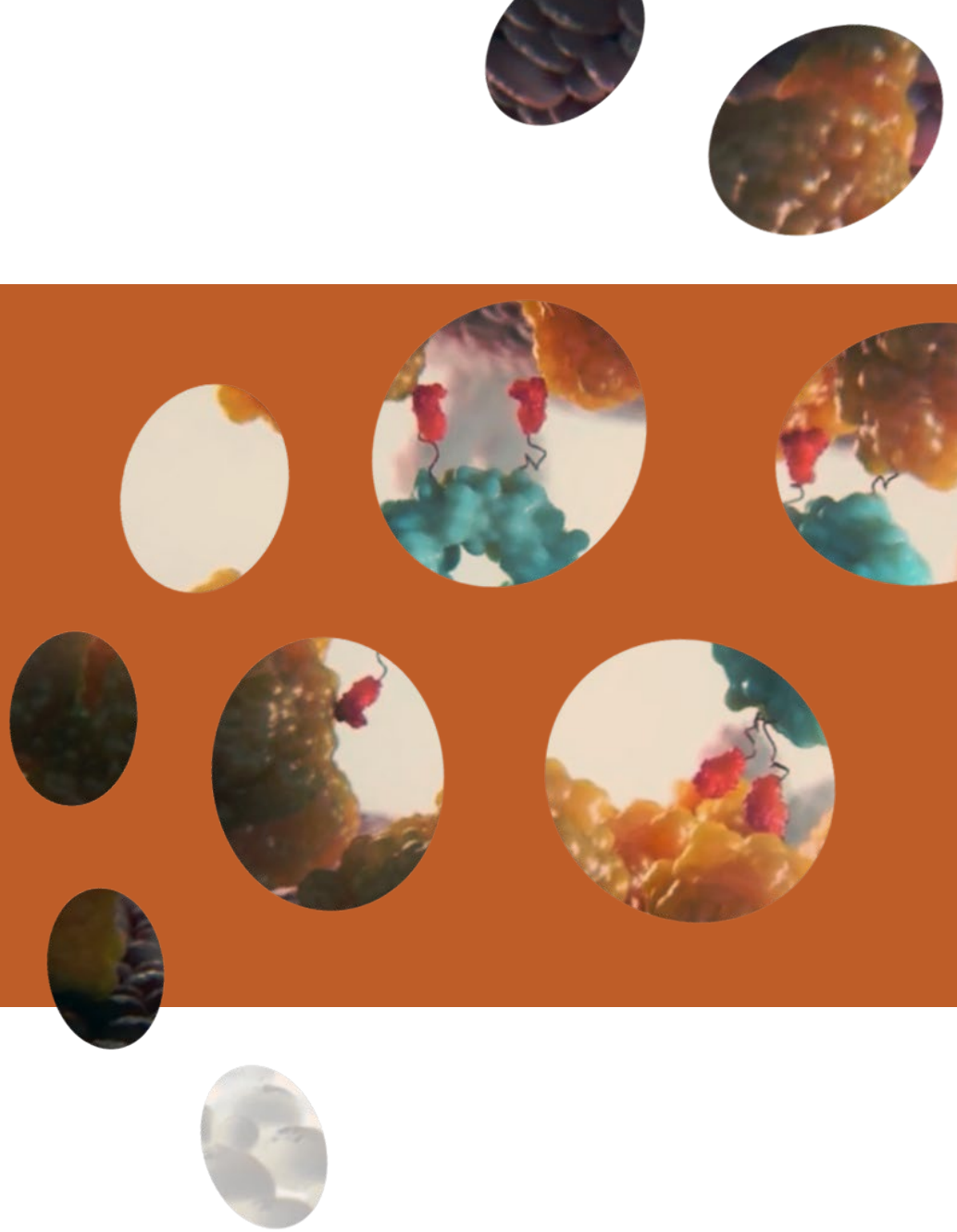
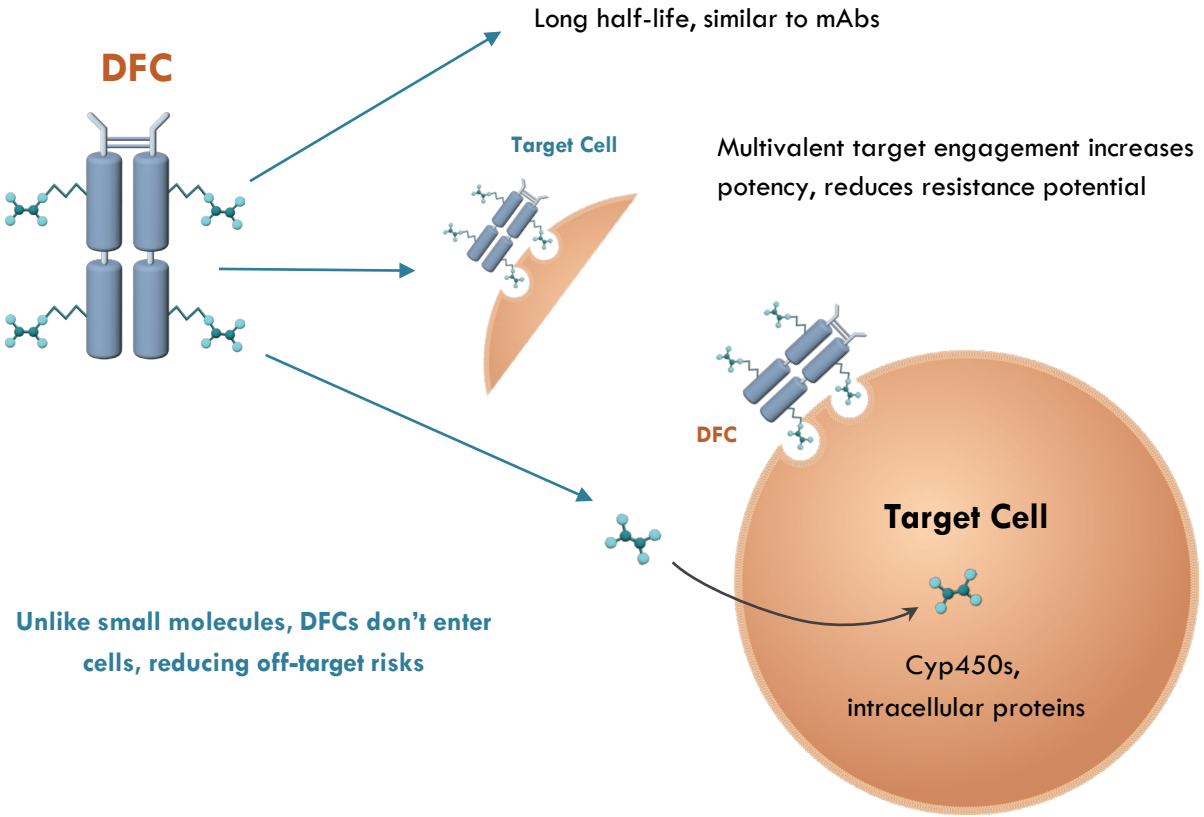


Nicole Davarpanah, MD, JD

SVP, Translational Research & Development

Cloudbreak Oncology

Significant Opportunity to Address Unmet Drug Development Challenges and Clinical Needs in Immuno-Oncology



Immuno-Oncology

- Only 1/5 patients show an initial response
- Amongst responders, 85% develop resistance within 6m
- Urgently need multi-modal therapies that address TME and mechanisms of immune evasion

Small Molecules

- Low response rates
- Off-target binding contributes to toxicity, DDIs
- Inadequate pharmacokinetics
- Drug resistance

Monoclonal Antibodies

- Poor tumor penetration
- Inhibition of small molecule receptors is challenging
- High cost

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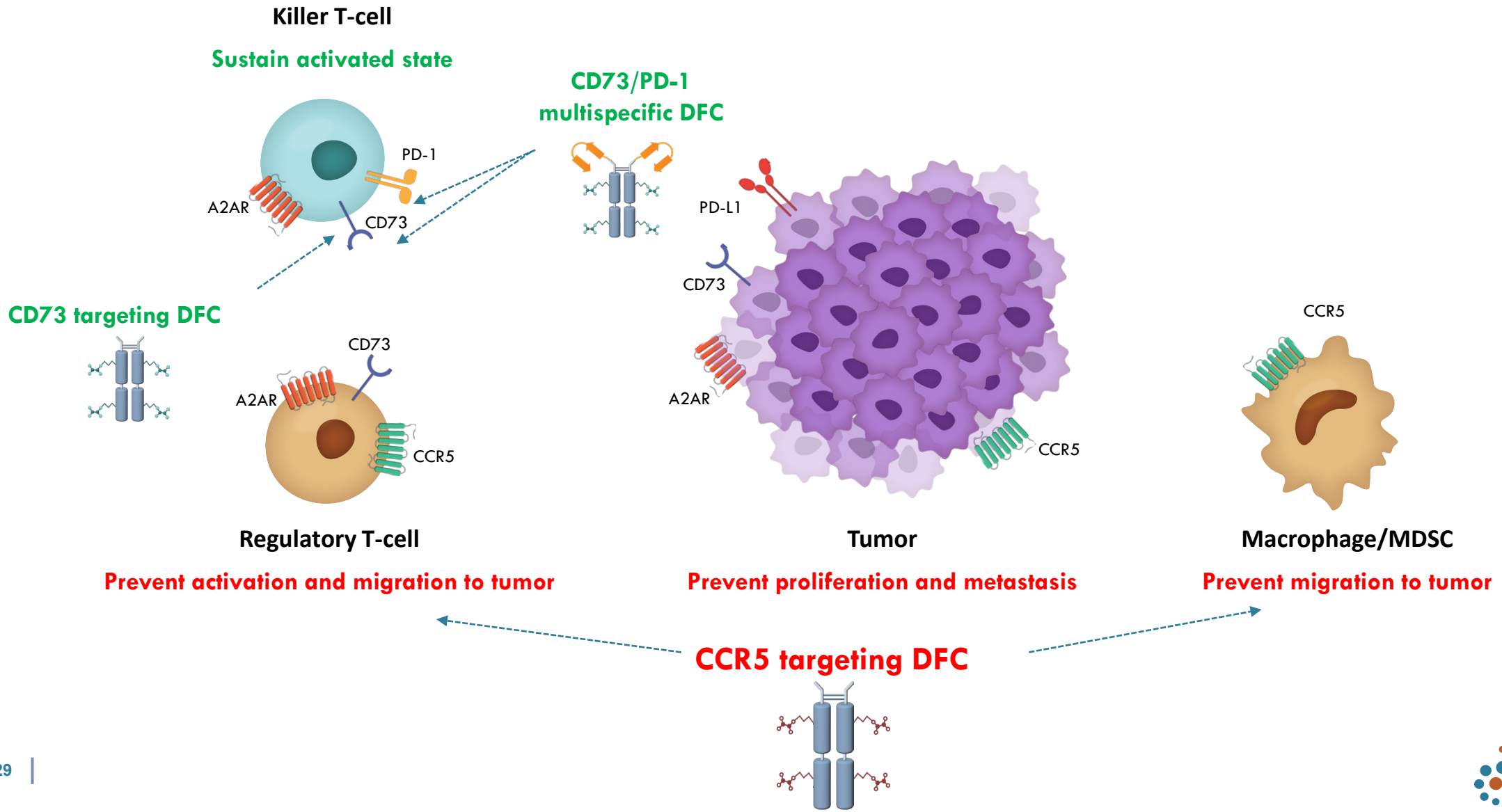
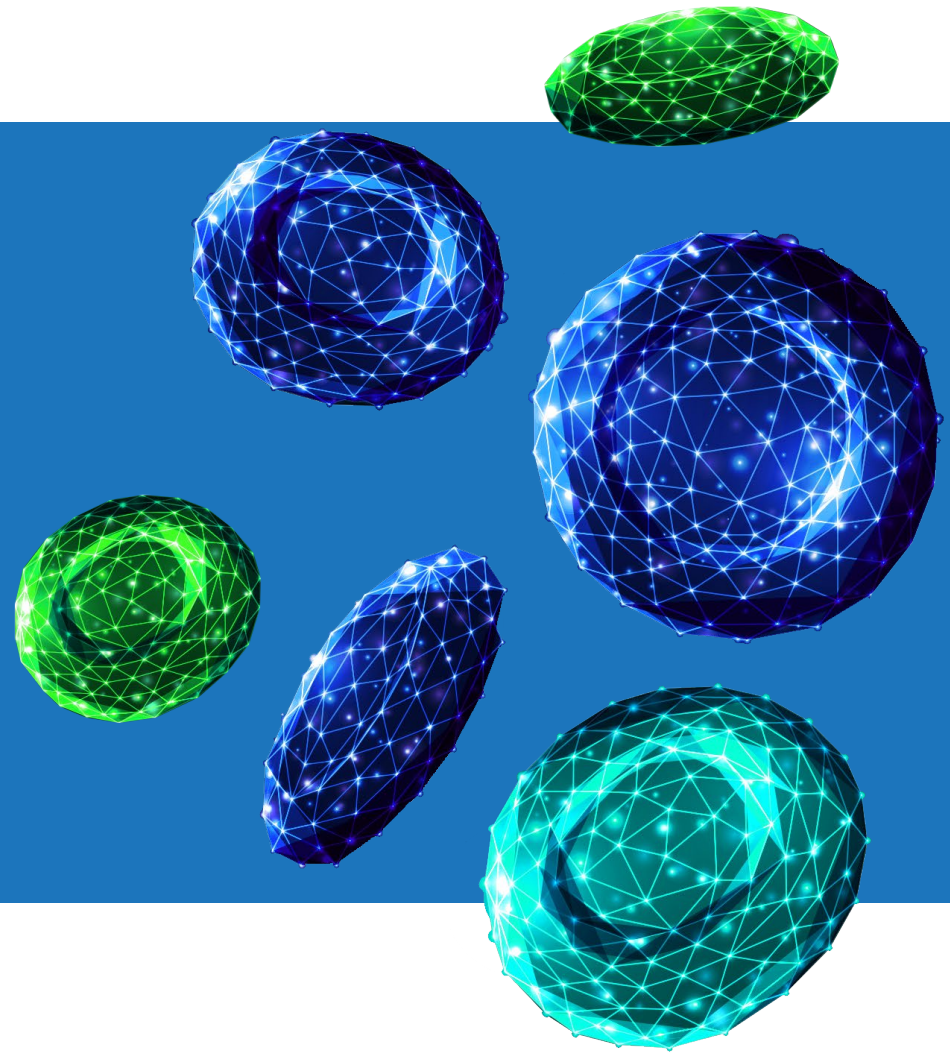
DFCs Inhibit Multiple Tumor Immune Evasion Mechanisms



Stephen Schoenberger, PhD

Who is left behind in cancer immunotherapy?



Stephen P. Schoenberger, PhD

Professor, Center for Immunotherapy, La Jolla Institute for Immunology

Adj. Professor, Division of Hematology & Oncology, UCSD Moores Cancer Center

Co-Director, San Diego Center for Precision Immunotherapy

IMAGINE
LIFE WITHOUT DISEASE

CIDARA THERAPEUTICS

Stephen P. Schoenberger, Ph.D

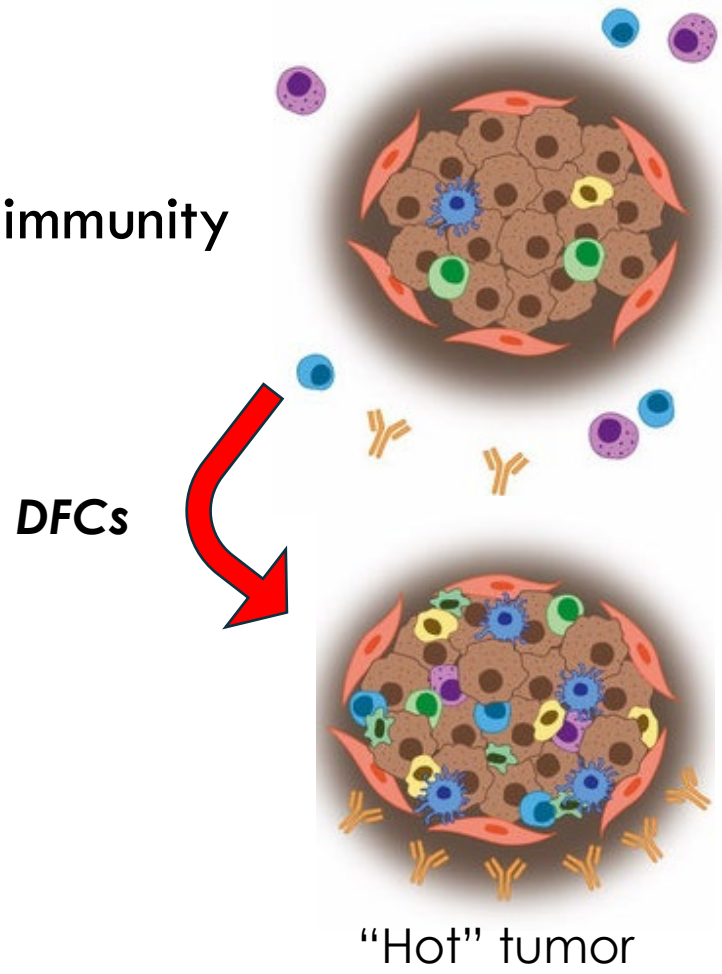
Professor, Center for Immunotherapy, La Jolla Institute for Immunology
Adj. Professor, Division of Hematology & Oncology, UCSD Moores Cancer Center
Co-Director, San Diego Center for Precision Immunotherapy

- Research Focus: Translational Immuno-Oncology
  - Personalized Cancer Vaccines
  - Adoptive Cell Therapy
  - Preclinical and Clinical immune and biomarker analysis
  - Fundamental T cell biology

1) Developed NeoAg ID platform & 1st/2nd gen. vaccines
2) Phase Ib NeoAg vaccine trial (NCT03568058)
3) Phase Ib NeoAg ACT (TIL)trial (NCT03991741)
4) Phase I/II NeoAg vaccine trial (NCT05153304)

Unmet needs / strategic opportunities in Immuno-Oncology

- Overcoming adaptive resistance to natural and induced tumor immunity
  - Ex 1: Treg, M2 Mf, MDSC (Adenosine, TGF-beta, Arginase, etc)
  - Ex 2: T cell inhibition by PD-1, CTLA-4, TIGIT, LAG-3, etc
- Therapeutic modification of TME
  - Ex: Deliver adjuvants, antigens, or chemo- or cytokines where needed



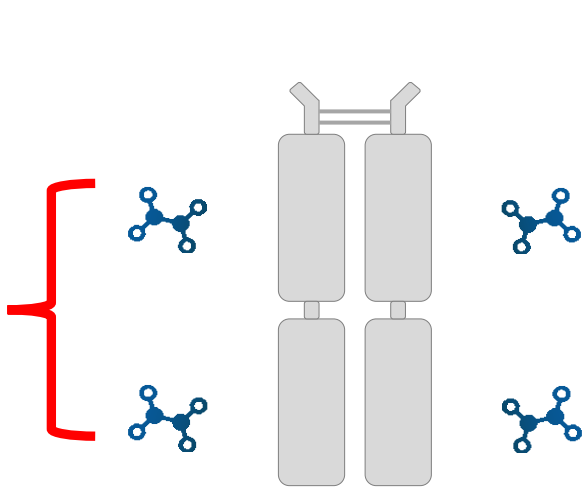
Unmet needs / strategic opportunities

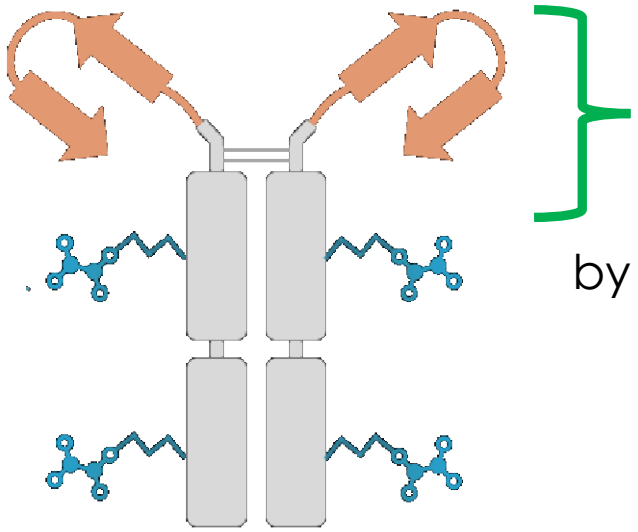
- Which patients/cancer types? -

- Incomplete ICB: Lung, Skin, RCC, HNSCC (20-40% ORR)
- Little or no ICB signal: CRC, BrCa, PrCa, Liver, others

- Leverages a proven “winner” in immunobiology protein structure with many impressive features (T1/2, bioavailability, neglible immune response, etc.)
- Unique biology and target interaction features
- Facile and efficient conjugation/derivatization platform
- Unique PK/ADME characteristics
- Proven efficacy in humans in influenza setting and impressive preclinical data in oncology models

Functional Payload
Inhibitors
Agonists
Peptides





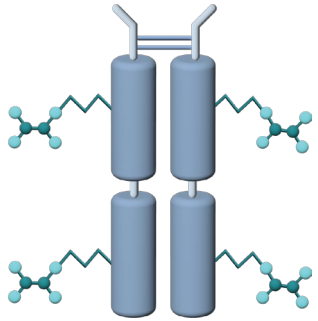
Targeting Moeity
Tumor
APC
Accessory cell
by *specific molecular target*

The DFC platform offer an unprecedented capacity for delivering custom small molecule response modifiers to cell surface targets with unique and desirable pharmacological properties that are well-suited to the IO setting

Cloudbreak Oncology DFC Programs

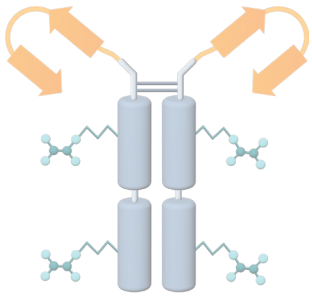
CD73: CBO421 Development Candidate

- Potential Best-in-Class
- Complete responders in combination with PD-1 inhibitors
- Excellent safety and COGS



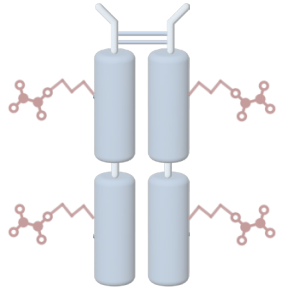
CD73/PD-1 Discovery Program

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



CCR5 Discovery Program

- Overcomes limitations of prior approaches
- Proof of concept data against “undruggable” target
- Opportunity to expand to other CCR targets



CIDARA THERAPEUTICS

CBO421 Is Cidara's First DFC Oncology Development Candidate

CBO421 Targets CD73, which mediates Immunosuppression and Resistance via Adenosine (Ado) Production

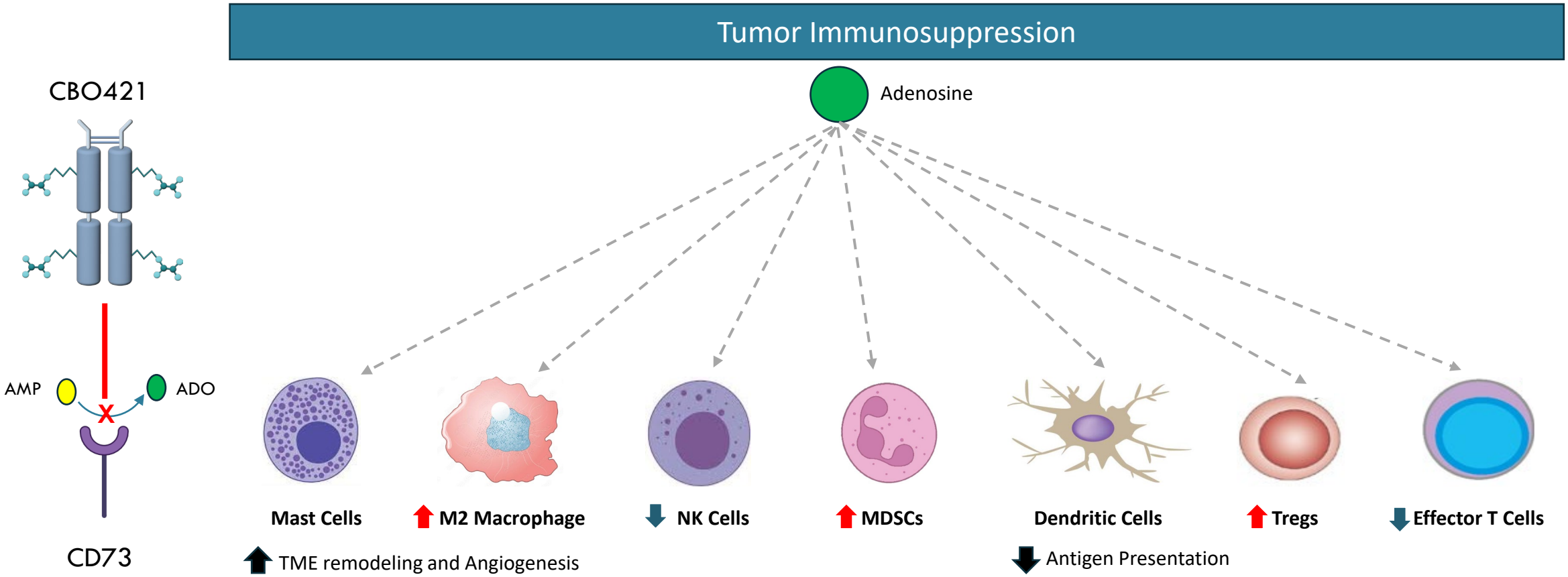
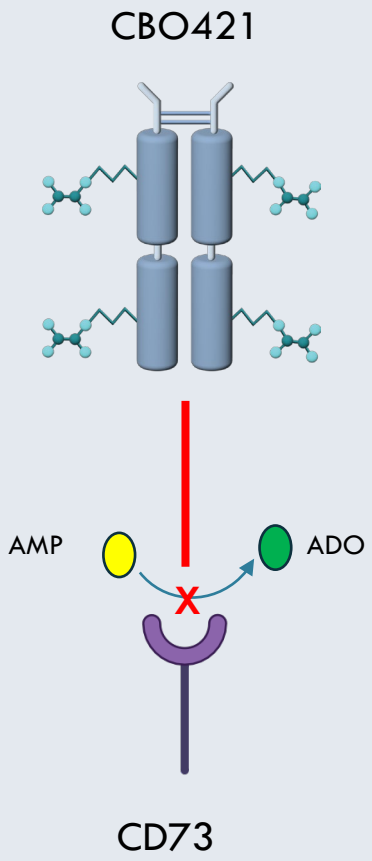


Image reprinted from Harvey JB, Phan LH, Villarreal OE and Bowser JL (2020) CD73's Potential as an Immunotherapy Target in Gastrointestinal Cancers. Front. Immunol. 11:508.

Les Tari, PhD

Chief Scientific Officer

CBO421 demonstrates outstanding preclinical performance



- Best-in-class activity
- 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
- Robust manufacturing process

CBO421 Exhibits Exceptional Preclinical Performance

Potential Best-in-Class Activity

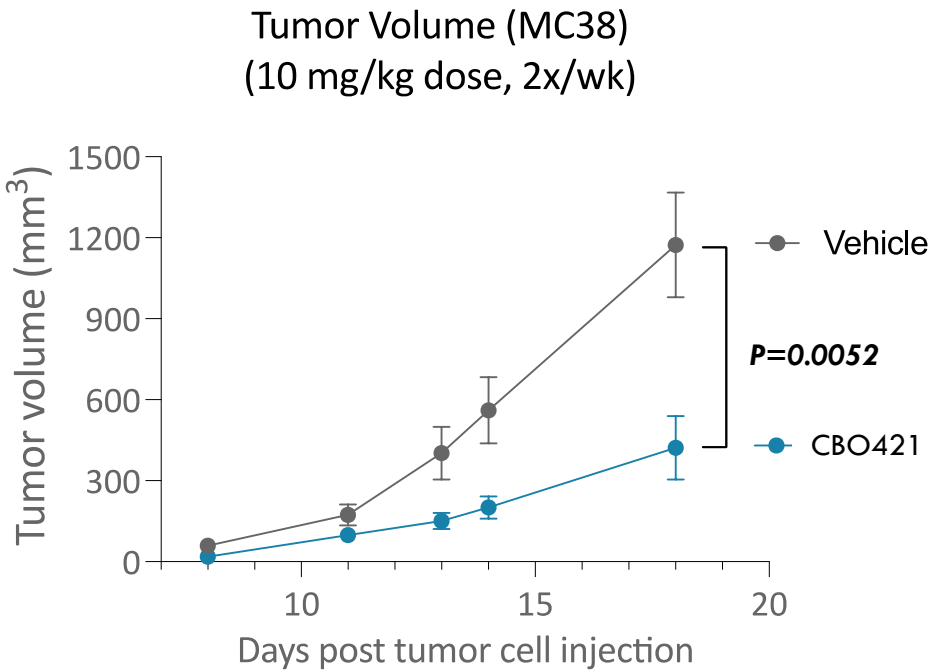
PBMC rescue assay (ATP) vs clinical stage adenosine pathway inhibitors

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	A2AR/small molecule	>1,000	>1,000

AB680 – Arcus Biosciences CD73 inhibitor
Oleclumab – Astra Zeneca biosimilar CD73 inhibitor
IPH5201 – Innate Pharma biosimilar CD39 inhibitor
AB928 – Arcus Biosciences A2AR inhibitor
CPI-444 – Corvus A2AR inhibitor

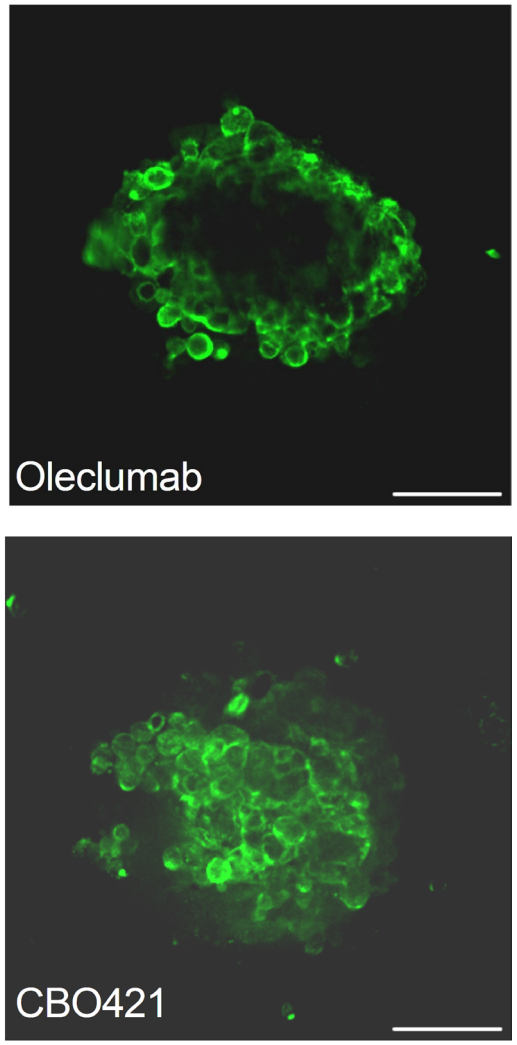
Potent Tumor Activity

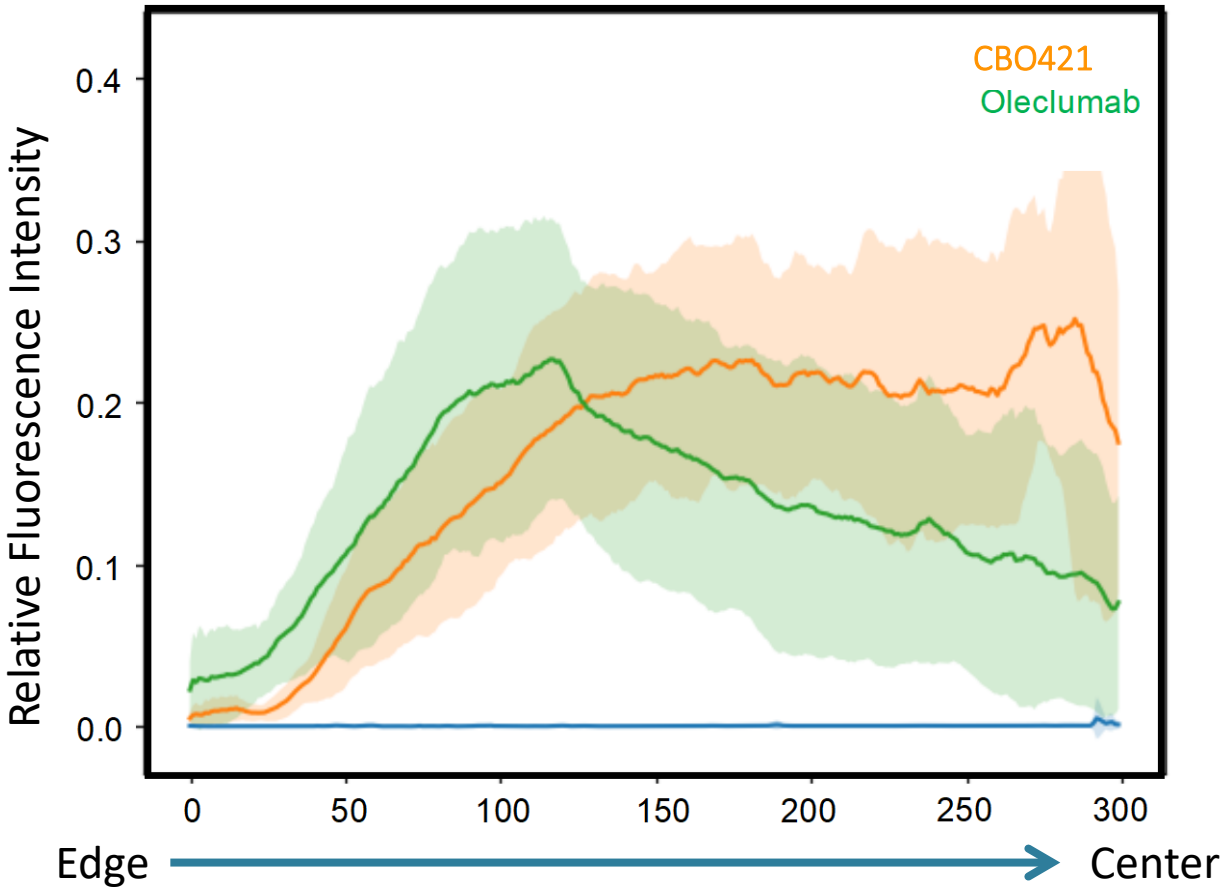
MC38 – murine colorectal carcinoma

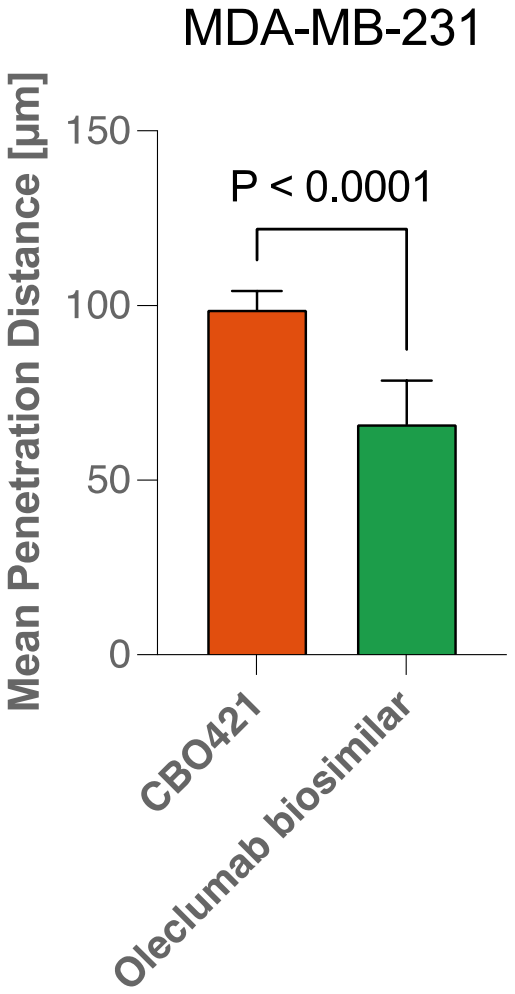


Tumor Control: CBO421 Exhibits Superior Tumor Penetration Vs. mAbs

MDA-MB-231 Spheroids







Study conducted by PhenoVista Biosciences

CIDARA THERAPEUTICS

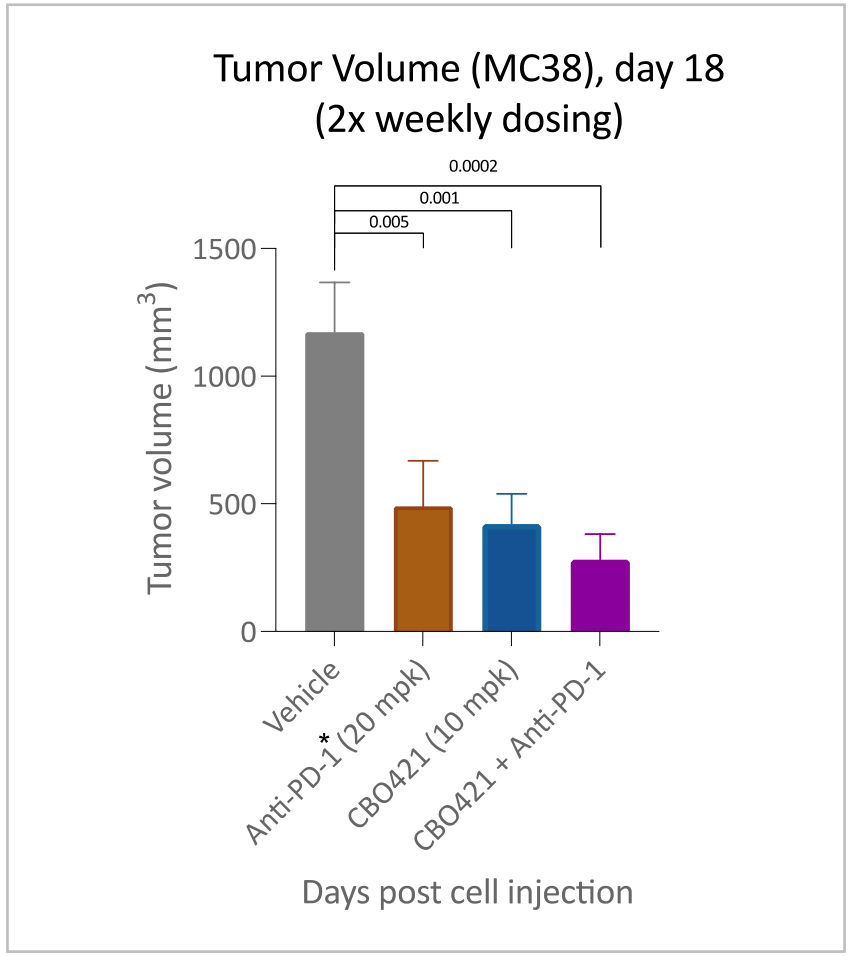
CBO421 Enhances Anti-tumor Activity Of PD-1 Inhibitors

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

MC38 – murine colorectal carcinoma

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

*Defined as mice that demonstrate cessation of tumor growth or reduction in tumor volume across two or more timepoints



CBO421 Elicits Complete Responses with PD-1 Inhibitors

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

Study start (D+0)

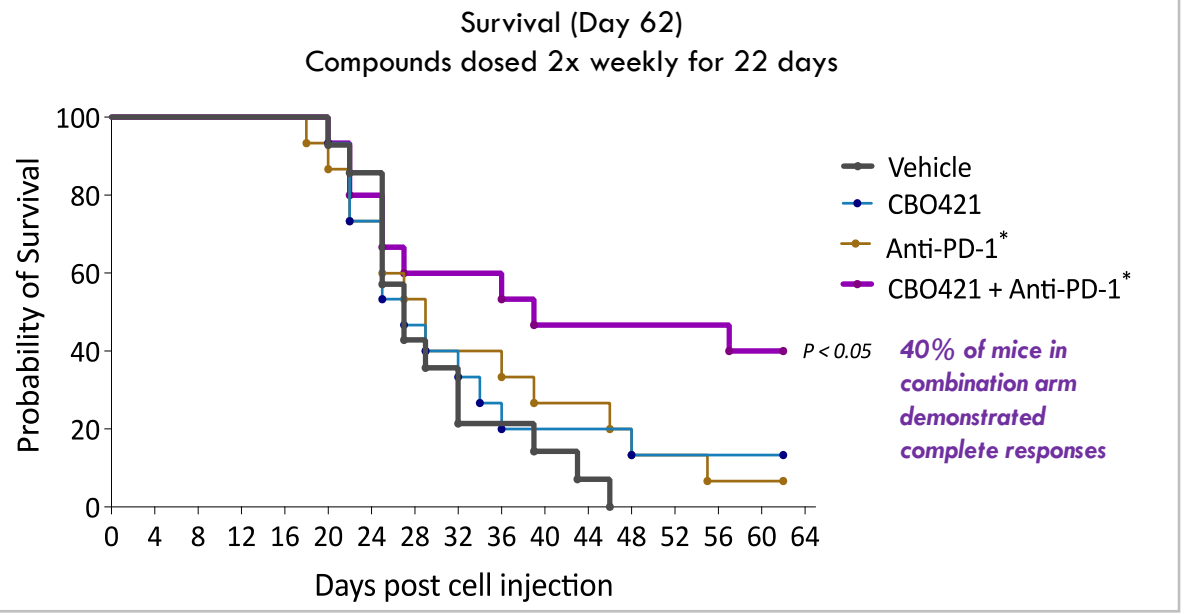
Last dose (D+22)

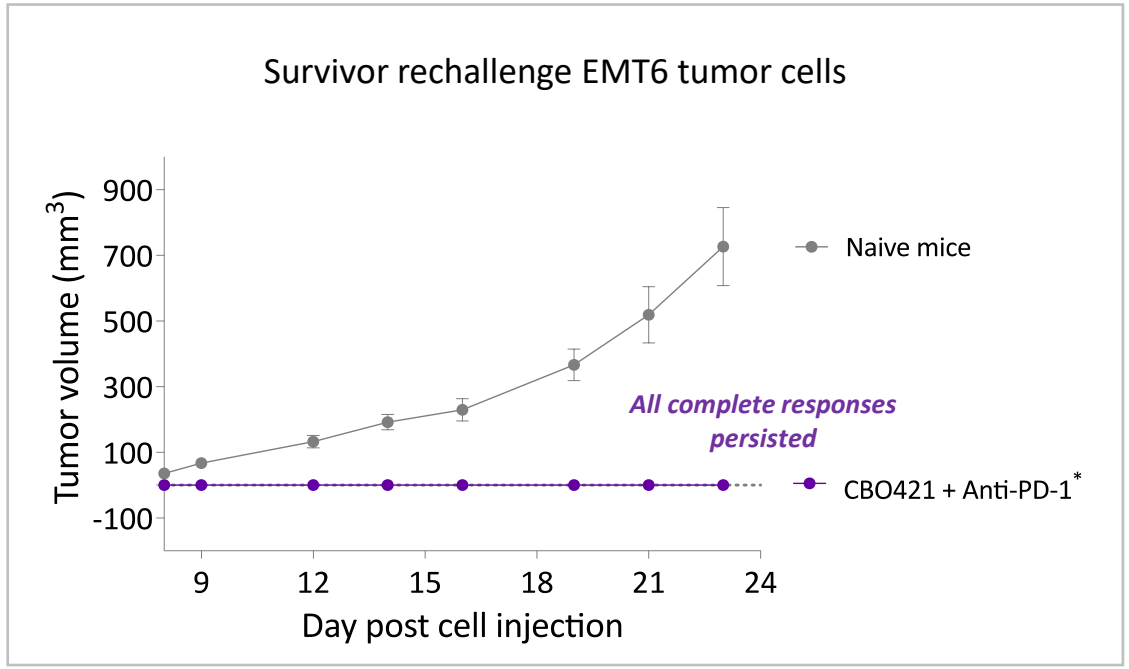
40-day washout period

D+62 (re-challenge)

D+73

EMT6 – murine mammary carcinoma





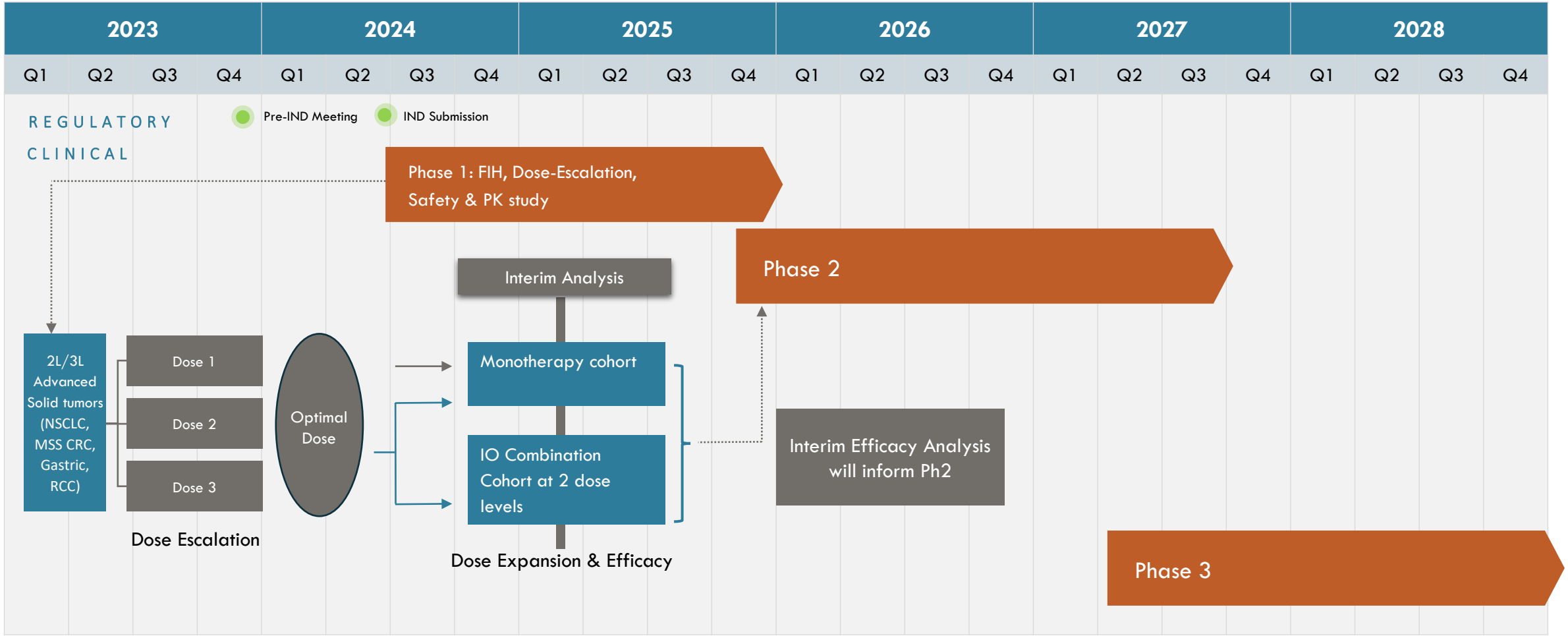
*RMP1-14

CBO421 Preclinical Summary

Superior specificity vs clinically available molecules	Potential for minimal off-target effects Potential for improved toxicity profile
Enhanced tumor penetration vs. mAbs	Potential to fully eradicate tumor Potential to control micro-metastatic disease
Demonstrate formation of immunologic memory	Potential to control disease with fewer doses Potential to achieve durable responses – large unmet need in IO
Ease of manufacturing	Optimized manufacturing process Low COGS
Modularity of DFC platform	Ability to optimize avidity, size, and pharmacokinetic properties Multispecific DFCs
Broad clinical development	Lower toxicity = patient centric; ability to achieve efficacious dose Lower DDI potential = better combinability with chemotherapy Enhanced efficacy with IO inhibitors

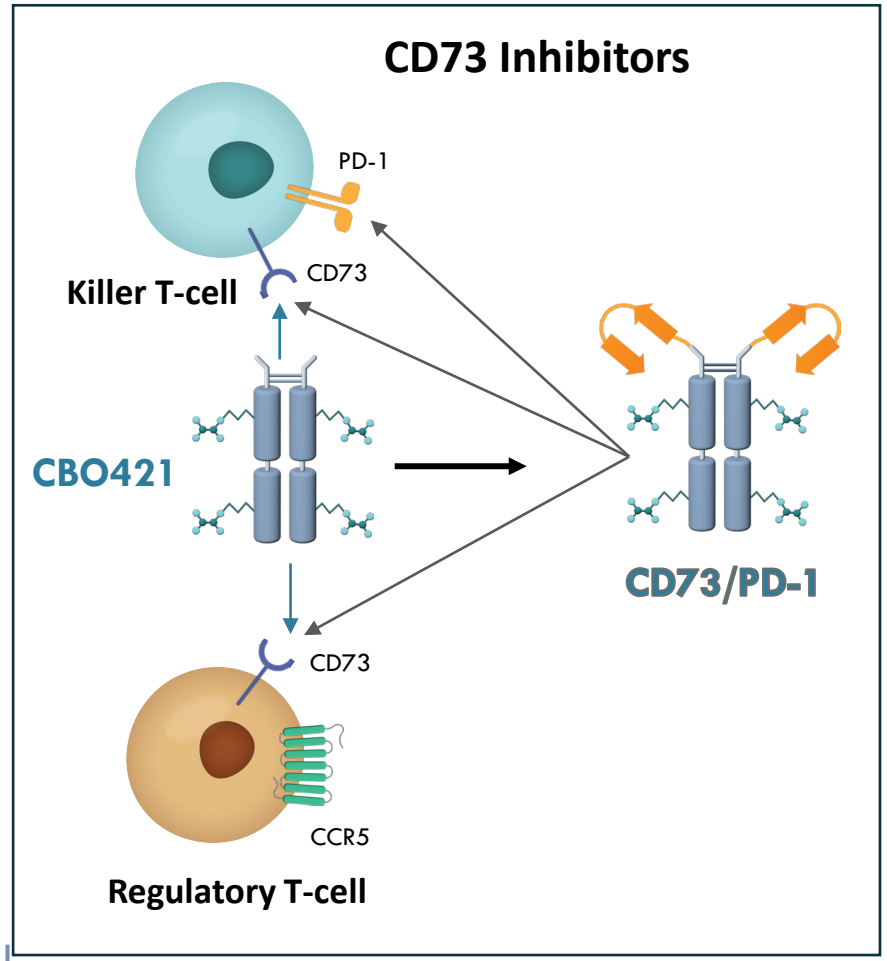
CIDARA THERAPEUTICS

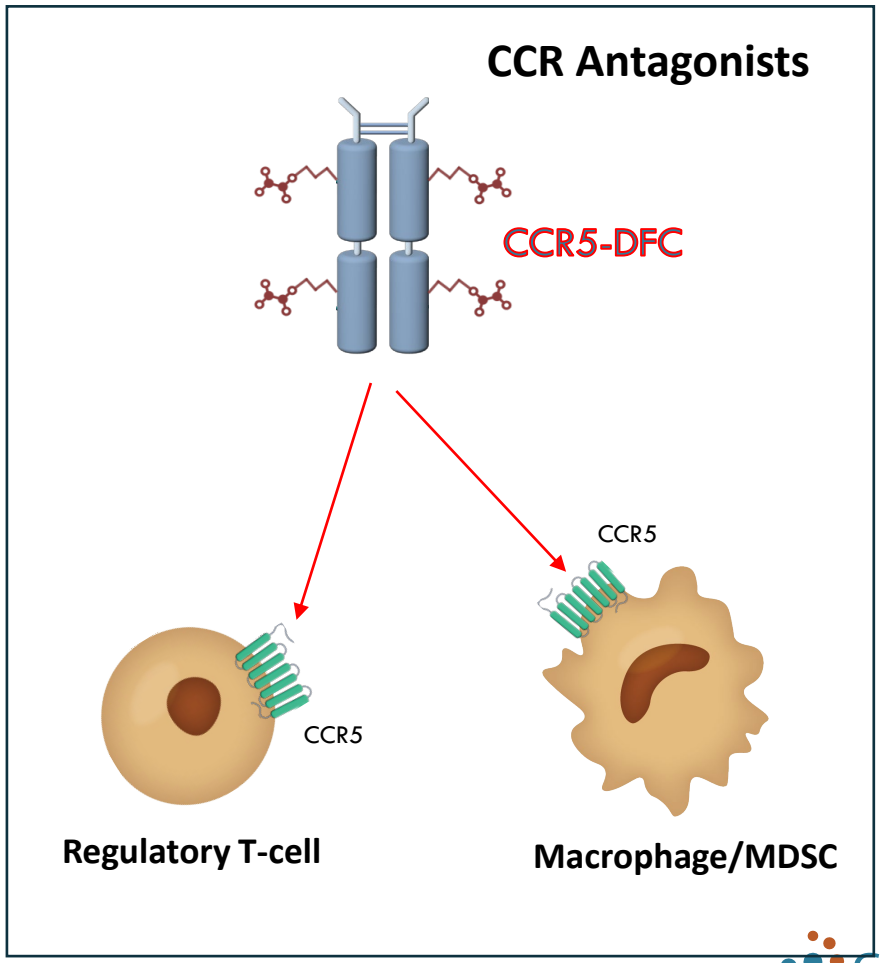
CBO421 Clinical Development Timeline



Expanding The DFC Platform:

Modularity and properties of DFCs allows for inhibition of multiple tumor immune evasion mechanisms, novel combinations, and exploitation of 'difficult to drug' targets

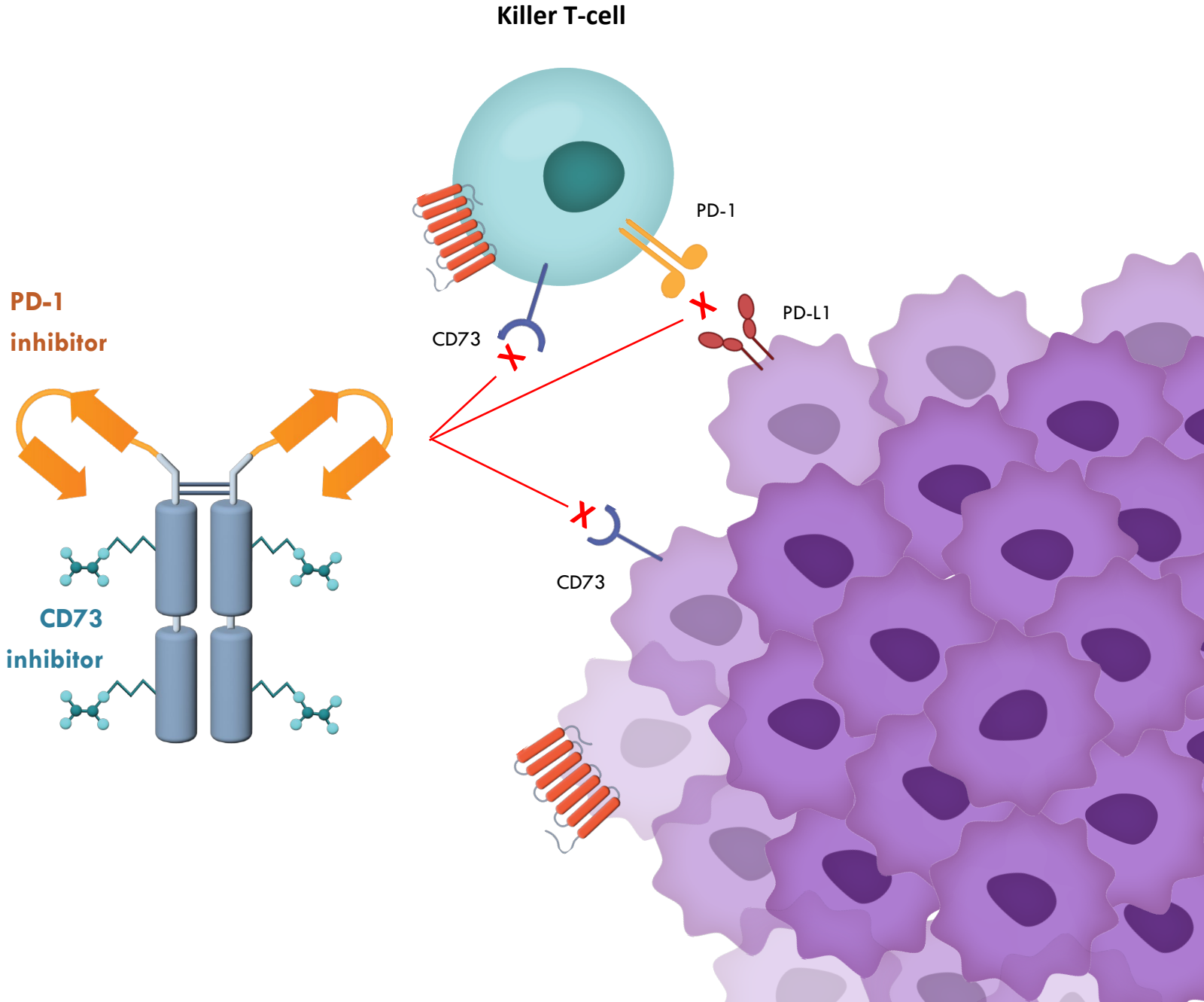




CIDARA THERAPEUTICS

Cidara's first multispecific DFC (CD73/PD1) is a **first-in-class** dual inhibitor

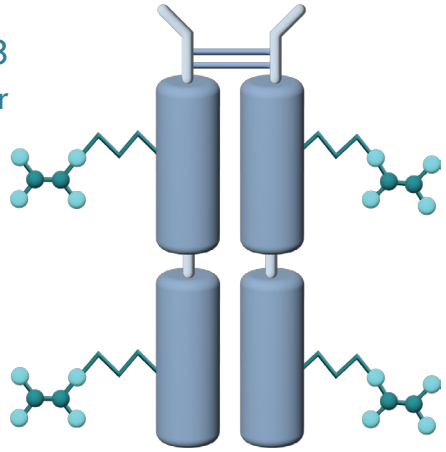
Engineered to improve efficacy by disabling a key resistance mechanism to PD-1 inhibitors



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

CD73 DFC

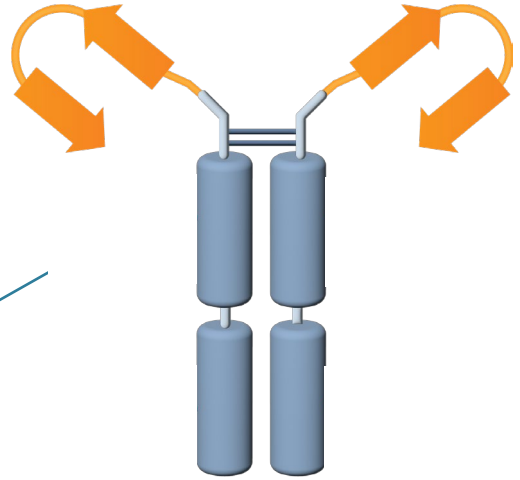
CD73 inhibitor



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

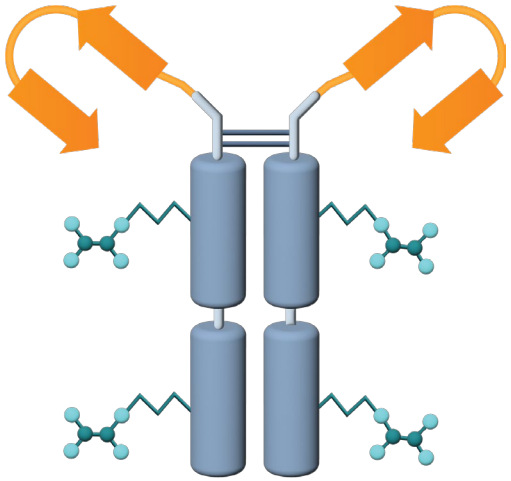
CTP-PD1

PD-1 inhibitor



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

CD73/PD-1 DFC

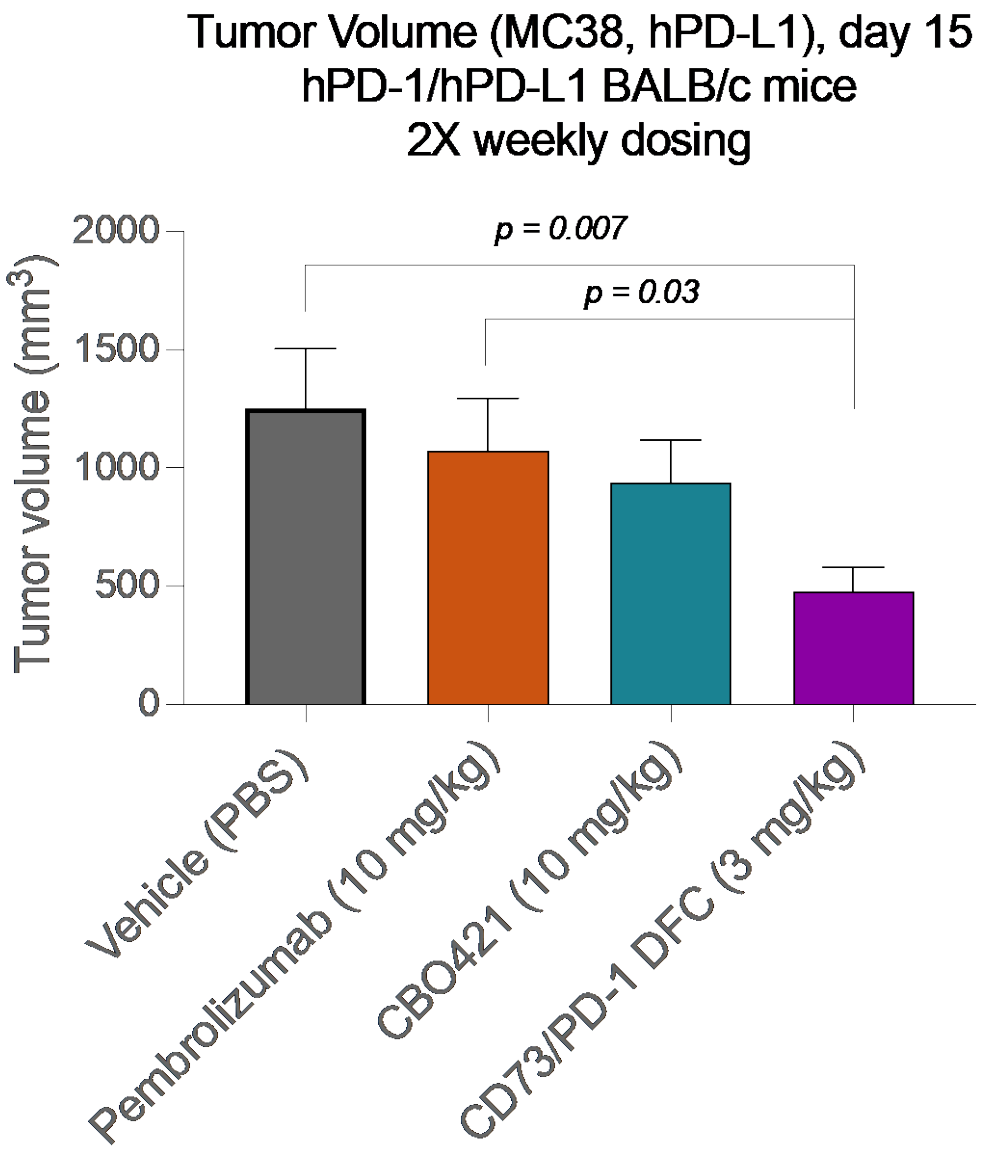


PD-1 Binding (IC_{50}, nM) – **0.064**

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

CIDARA THERAPEUTICS

CD73/PD-1 DFC Outperforms Monotherapies In Humanized Tumor Models



CIDARA THERAPEUTICS

Cloudbreak Oncology DFC Programs

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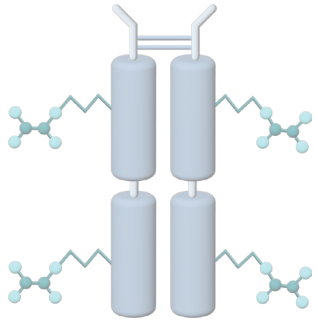

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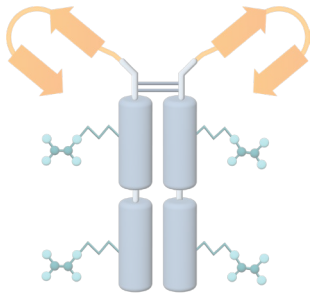
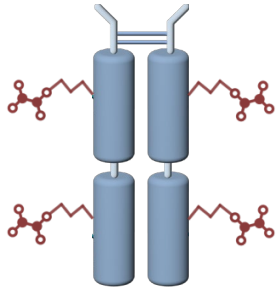
- Potential for more efficient clinical development



CCR5 Discovery Program

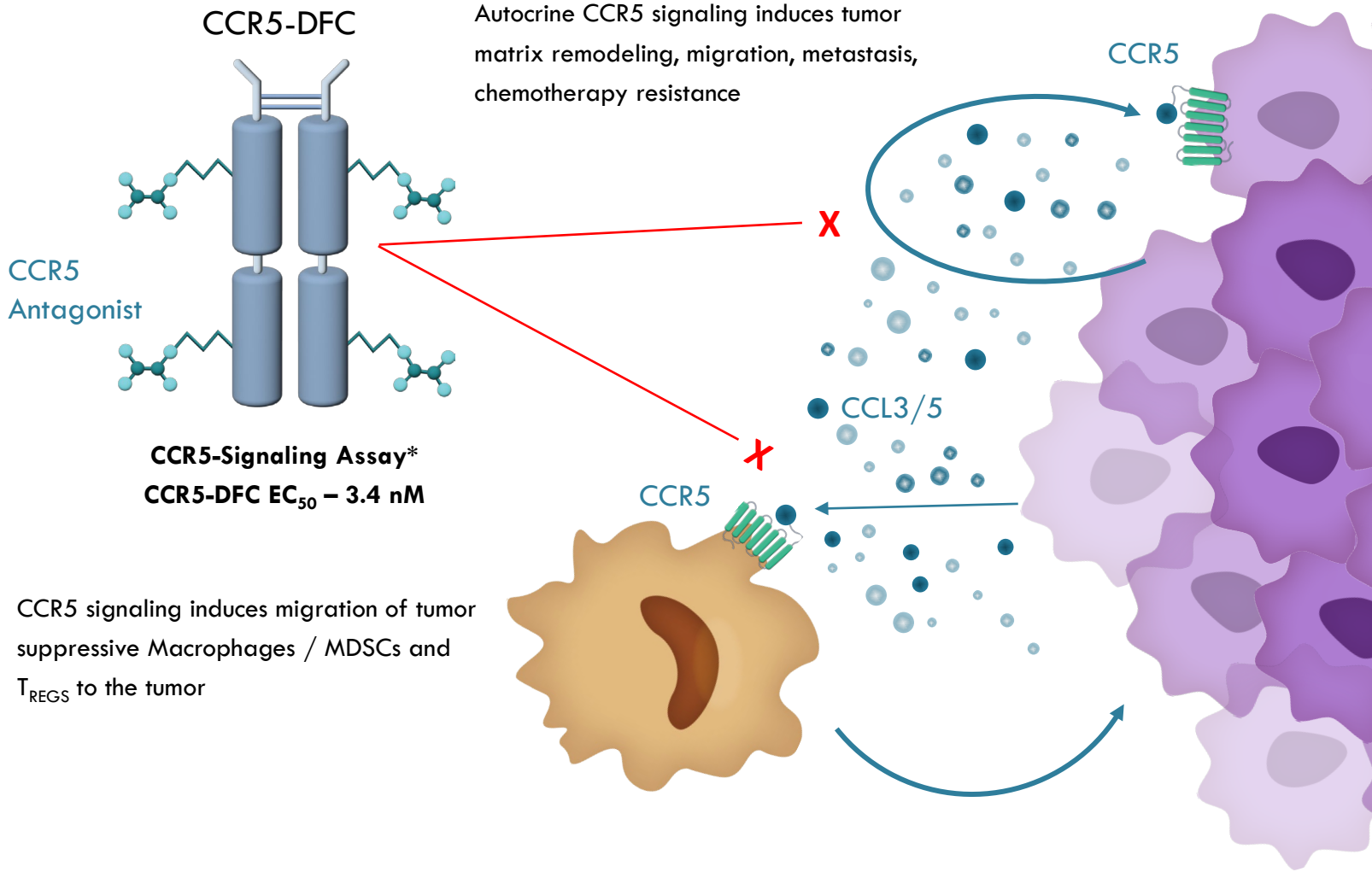
- Overcomes limitations of prior approaches
- Proof of concept data against “undruggable” target
- Opportunity to expand to other CCR targets



CIDARA THERAPEUTICS

CCRs are a historically difficult to drug receptor family

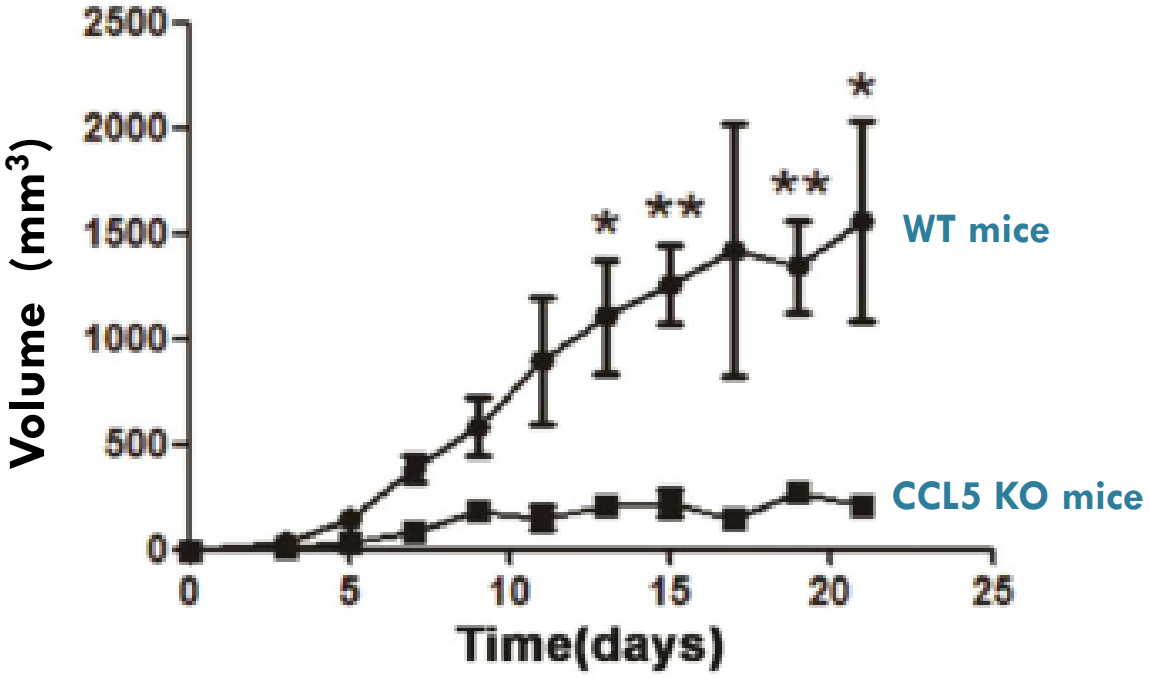
Next generation DFCs allow us to exploit validated but difficult targets like CCR5 which promote metastasis, invasiveness, migration, recruitment of immunosuppressive cells to TME, and render tumors resistant to DNA damaging agents



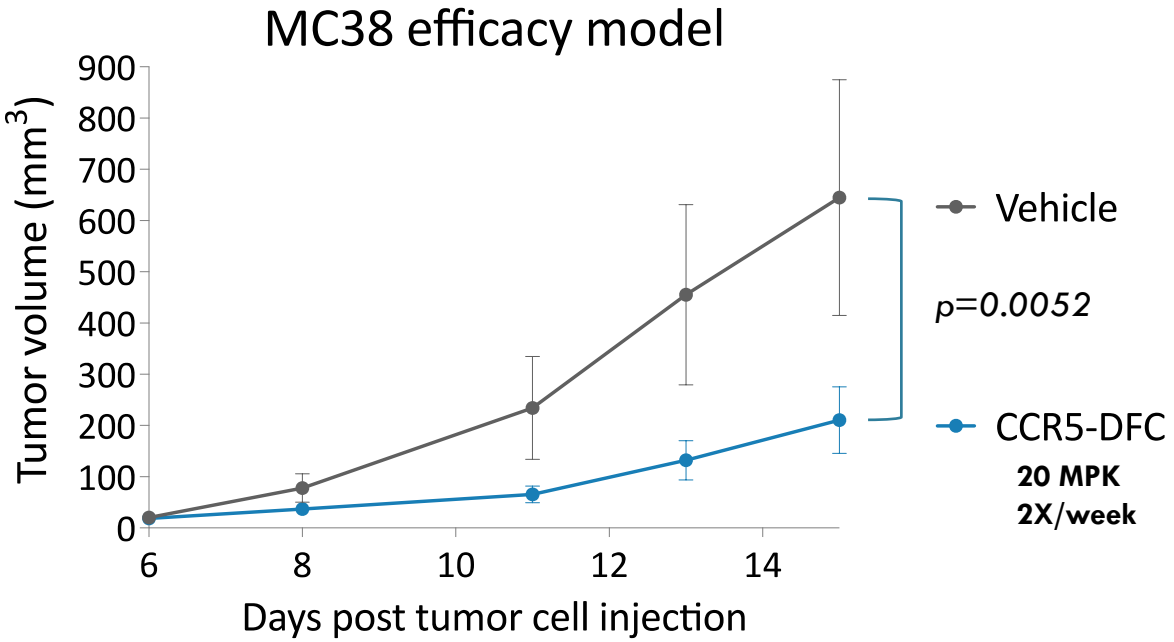
* Pathhunter® eXpress β-arrestin CCR5 GPCR assay

CCR5-DFC Shows Strong Tumor Control In Preclinical Model

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



CCR5-DFC treatment accomplishes a similar degree of tumor reduction



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

CIDARA THERAPEUTICS

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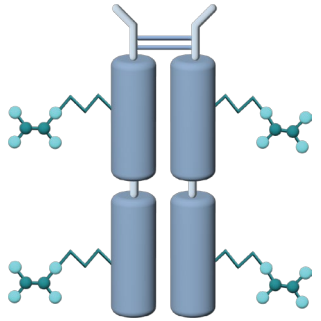
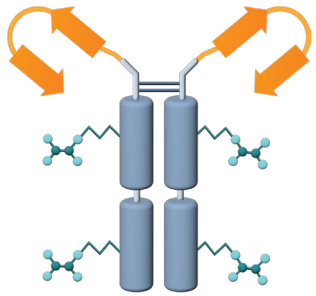
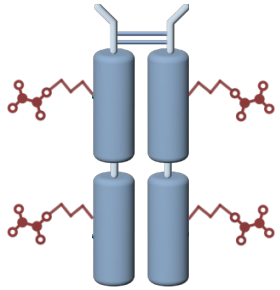
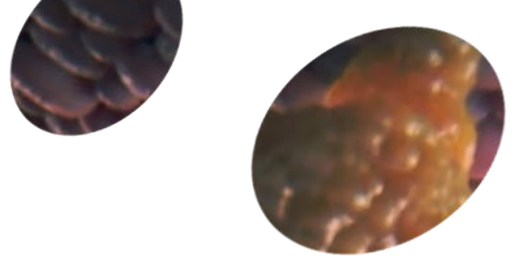






CIDARA THERAPEUTICS

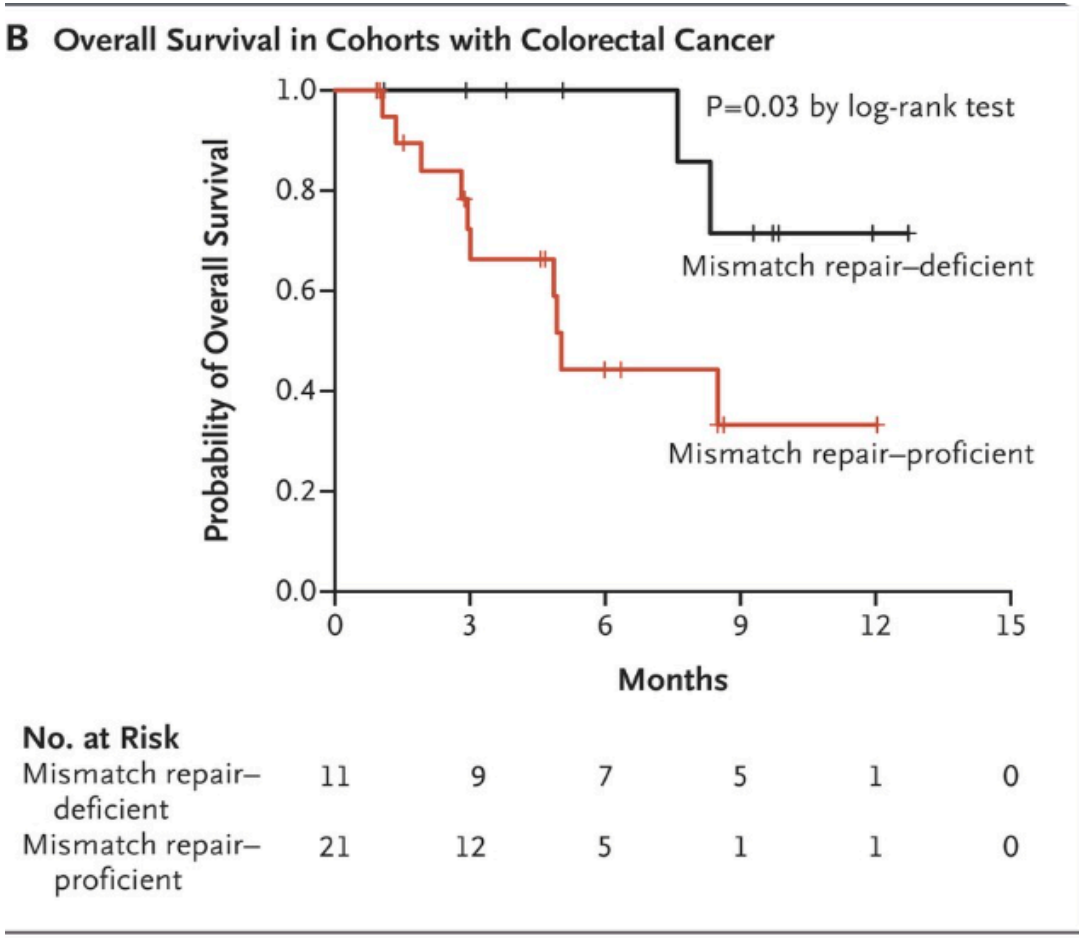


Ezra Cohen, MD

Perspective: The Potential of DFCs to Transform Precision Oncology

Spotlight on Colorectal Carcinoma (CRC)

- 3rd most common cancer and the 2nd cause of cancer-related deaths worldwide. *Bray et al. CA Cancer J Clin. (2018) 68:394–424*
- OS remains poor for metastatic disease ~ 30 months.
- Mortality has increased by 0.5%–3% annually in individuals younger than 50 years. *American Cancer Society, 2021*
- IO therapy was recently approved for refractory MMR-deficient/microsatellite instability high (MSI-H) CRC but applies to less than 5% of metastatic diagnoses.
- Great unmet need remains, particularly for MMR-proficient/ microsatellite stable (MSS) CRC.



Le DT, Uram JN, Wang H, et al. PD-1 blockade in tumors with mismatch-repair deficiency. N Engl J Med. 2015;372:2509-2520.

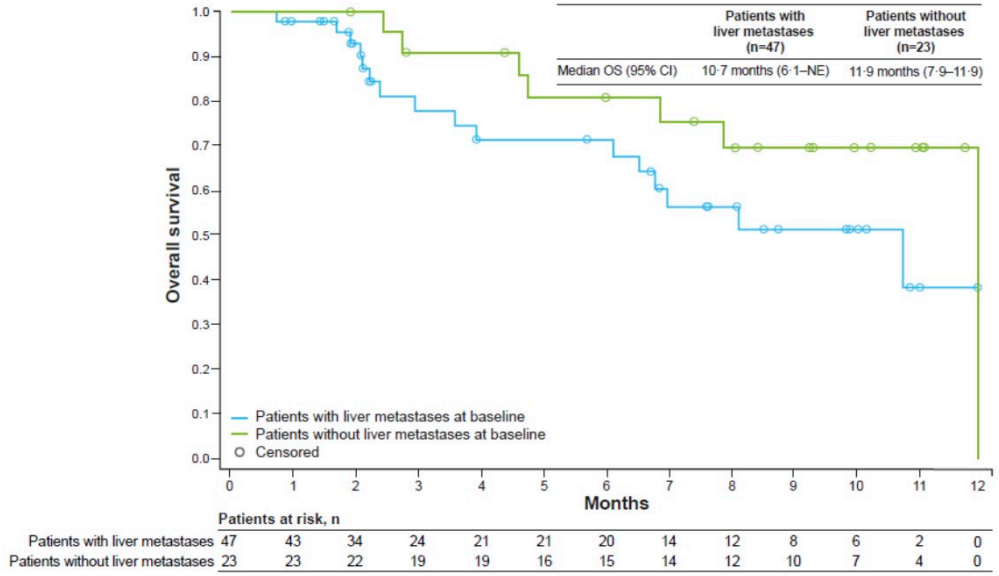
CIDARA THERAPEUTICS

MSS Metastatic Colon Cancer is Particularly Adept at Immune Escape

Immunotherapy and tyrosine kinase inhibitor combinations in MSS CRC have resulted in limited clinical efficacy, particularly in patients with liver metastases (>50% of patients)

Study	Benefit
CM-142 (Nivolumab + Ipilimumab)[1]	PFS 1.4m
Cancer Trial Group CO.26 study (Durvalumab + Tremelimumab)[2]	PFS 1.8m vs. 1.9m (BSC)
IMblaze-370 (Atezolizumab vs. Atezolizumab + Cobimetinib vs. Regorafenib)[3]	OS 7.1m vs. 8.9m vs. 8.5m
Leap-017 (Pembro + Lenvatinib)[4]	OS 9.8m vs 9.3m (SOC)
Nivolumab + Regorafenib ph2[5] (*liver metastasis subgroup data below*)	ORR 7% (*p=0.27*)
Ph2 Nivolumab + Relatlimab (LAG3 inhibitor)[6]	In progress

Overall Survival in MSS CRC patients with and without liver metastases after nivolumab + regorafenib therapy[5]



1Overman MJ, Kopetz S, McDermott RS, et al. Nivolumab ± ipilimumab in treatment (tx) of patients (pts) with metastatic colorectal cancer (mCRC) with and without high microsatellite instability (MSI-H): CheckMate-142 interim results. J Clin Oncol; 2016;34:15s (supplement)

2Chen EX, Jonker DJ, Loree JM, et al. Effect of combined immune checkpoint inhibition vs best supportive care alone in patients with advanced colorectal cancer: The Canadian Cancer Trials Group CO. 26 Study. JAMA Oncol. 2020;6:831-838.

3 Eng C, Kim TW, Bendell J, et al; IMblaze370 Investigators. Atezolizumab with or without cobimetinib versus regorafenib in previously treated metastatic colorectal cancer (IMblaze370): a multicentre, open-label, phase 3, randomised, controlled trial. Lancet Oncol. 2019;20:849-861.

4 https://doi.org/10.1016/j.annonc.2023.04.015 (ESMO 2023 abstract)

5Fakih M et al. Regorafenib plus nivolumab in patients with mismatch repair-proficient/microsatellite stable metastatic colorectal cancer: a single-arm, open-label, multicentre phase 2 study. EClinicalMedicine. 2023 Apr 6;58:101917 (supplement)

6 NCT03642067

DFCs offer opportunity to uniquely target tumor microenvironment of MSS CRC which is distinct from its MSI-H counterpart

Only CD73 biologic that targets both soluble and membrane-bound CD73

High levels of soluble CD73 in patients with CRC liver metastases linked to shorter survival[1]

Preclinical model CCR5-DFC accomplished similar degree of tumor reduction as CCR5 deficient CRC cell

First CCR5 targeted molecule specifically designed for oncology needs

Preclinical model demonstrated enhanced tumor penetration compared to mAbs

Potential to achieve complete responses and prevent seeding of micro-metastatic disease

DFC platform allows for multi-targeted mechanisms of action

Potential for both monotherapy and synergistic activity in combination with approved checkpoint inhibitors

Preclinical models demonstrated immunologic memory

Durability of response significantly lacking in current treatment regimens

Promising non-GLP animal safety data

Anticipate benign toxicity profile

[1]Messaoudi N et al. Prognostic value of CD73 expression in resected colorectal cancer liver metastasis. Oncoimmunology. 2020 Apr 23;9(1):1746138.