C4 Therapeutics

Protein degraded.
Disease targeted.
Lives transformed.

October 2024

Forward-looking Statements and Intellectual Property

Forward-looking Statements

The following presentation contains forward-looking statements. All statements other than statements of historical fact are forward-looking statements, which are often indicated by terms such as "anticipate," "believe," "could," "estimate," "expect," "goal," "intend," "look forward to," "may," "plan," "potential," "predict," "project," "should," "will," "would" and similar expressions. These forward-looking statements include, but are not limited to, statements regarding the therapeutic potential of C4 Therapeutics, Inc.'s technology and products. These forward-looking statements are not promises or guarantees and involve substantial risks and uncertainties. Among the factors that could cause actual results to differ materially from those described or projected herein include uncertainties associated generally with research and development, clinical trials and related regulatory reviews and approvals, as well as the fact that the product candidates that we are developing or may develop may not demonstrate success in clinical trials. Prospective investors are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date hereof. The forward-looking statements included in this presentation are subject to a variety of risks and uncertainties, including those set forth in our most recent and future filings with the Securities and Exchange Commission. Our actual results could vary significantly from those anticipated in this presentation, and our financial condition and results of operations could be materially adversely affected. C4 Therapeutics, Inc. undertakes no obligation to update or revise the information contained in this presentation, whether as a result of new information, future events or circumstances or otherwise.

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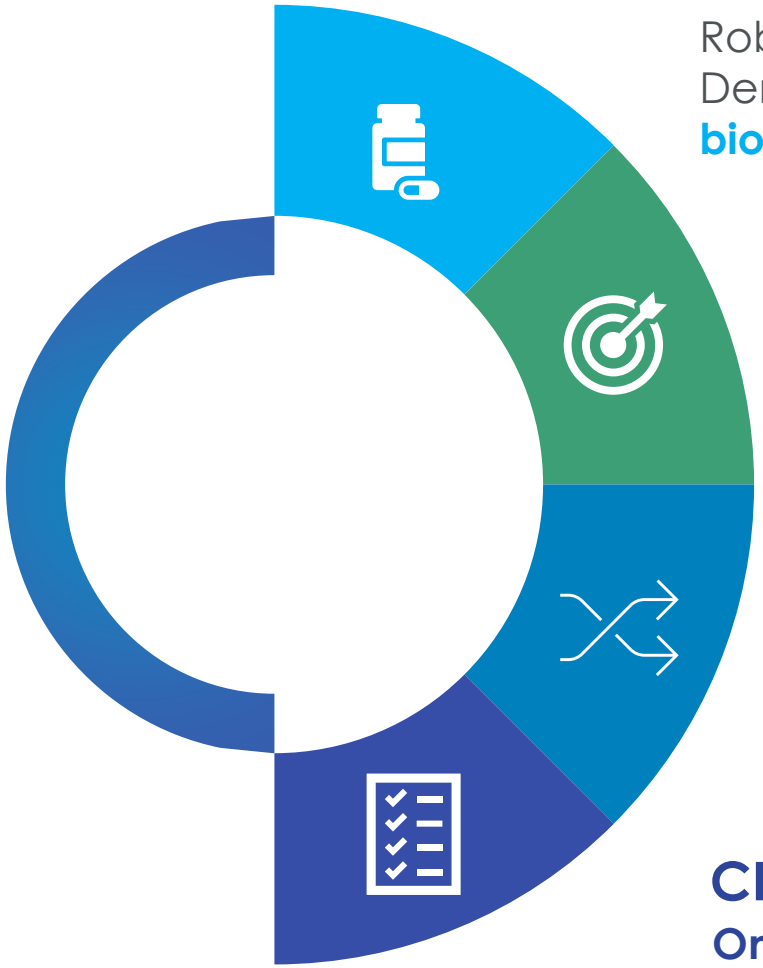
C4T is a Recognized Leader in Delivering on the Promise of Targeted Protein Degradation

Our Mission

To deliver on the promise of targeted protein degradation science to create a new generation of medicines that transform patients' lives



WORLD-CLASS DEGRADER PLATFORM

Robust patent portfolio of novel cereblon binders; Demonstrated ability to design **orally bioavailable, catalytically efficient degraders**

RIGOROUS TARGET SELECTION

Focus on targets with a **clear degrader rationale**

BROAD DEGRADER APPROACH

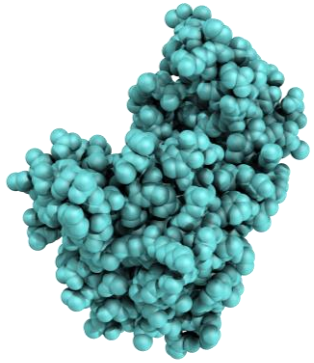
MonoDAC and **BiDAC** degraders, as well as **degrader-antibody conjugates**

CLINICAL PIPELINE

Oncology degraders against targets of high unmet need

Designed and Advanced Degraders into the Clinic Across a Range of Target Classes, Demonstrating Robust Target Degradation

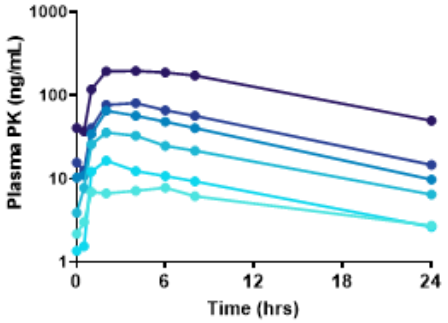
Interrogated Diverse Target Classes



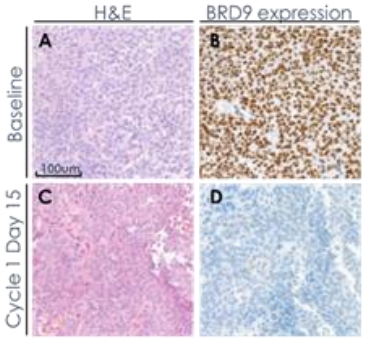
Attained IND Clearance

FDA

Achieved Desirable Drug-like Properties



Degraded Target as Predicted



- Discovered degraders and advanced **4 INDs** against a transcription factor, a chromatin modifier, and two kinases
- Have evaluated **3 programs** in the clinic, each demonstrating robust target degradation in patients
- Delivered **two development candidates** to our collaboration partner, Biogen

Investigational New Drug Applications (INDs)

C4 Therapeutics

Advancing a Broad Pipeline to Deliver Near-Term Value

Program	Target	Indications	Discovery	Preclinical	Early Phase Development	Late Phase Development	Rights
Cemsidomide	**IKZF1/3**	Multiple Myeloma & Non-Hodgkin's Lymphoma					
CFT1946	**BRAF V600 Mutant**	V600 Mutant Cancers					
CFT8919[1]	**EGFR L858R**	Non-Small Cell Lung Cancer					BETTA PHARMACEUTICALS
Discovery Stage Programs		Various Cancers					
Collaboration Programs		Autoimmune & Cancer	*2 targets*				Roche
		Cancer	*2 targets*				Merck KGaA Darmstadt, Germany
		Cancer	*1 target*				MERCK
		Autoimmune & Neurological	*2 targets*				Biogen[2]

[1]License and collaboration agreement with Betta Pharmaceuticals for development and commercialization in Greater China; [2]Delivered development candidates to Biogen in Q1 2024 and Q3 2024

C4T is On Track to Execute Across All 2024 Goals, Progressing Multiple Clinical and Preclinical Programs

Cemsidomide
IKZF1/3

- **ASH 2024 (Dec.):** Present updated data from Phase 1 dose escalation +dex trial in R/R MM
- **ASH 2024 (Dec.) :** Present data from Phase 1 dose escalation monotherapy trial in R/R NHL
- **By YE 2024:** Complete Phase 1 dose exploration in R/R MM and R/R NHL

CFT1946
BRAF V600 Mutant

- **2Q 2024:** Present preclinical data demonstrating differentiated activity in BRAF V600 mutant driven melanoma, CRC, NSCLC, and brain metastasis models at AACR
- **ESMO Congress 2024:** Present monotherapy data from Phase 1 dose escalation trial in melanoma, CRC, NSCLC and other BRAF V600 mutant driven cancers

CFT8919
EGFR L858R

- **2024:** Support trial start-up activities related to Betta's Phase 1 dose escalation trial in China

Discovery

- **1Q 2024:** Collaboration with Merck KGaA, Darmstadt, Germany to discover two targeted protein degraders against critical oncogenic proteins
- **2024:** Deliver development candidate to collaboration partner

Runway into 2027, Beyond Value Inflection Milestones

Relapsed or refractory multiple myeloma (R/R MM); Relapsed or refractory non-Hodgkin lymphoma (R/R NHL); Colorectal cancer (CRC); Non-small cell lung cancer (NSCLC)

Cemsidomide

Targeting IKZF1/3

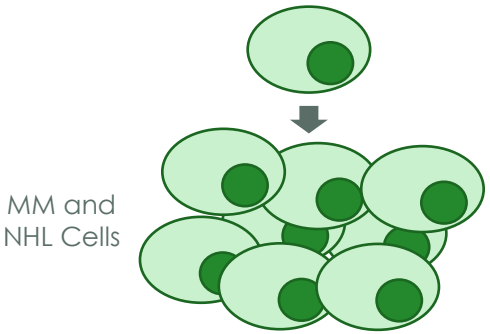
Multiple Myeloma (MM)
& Non-Hodgkin's Lymphoma (NHL)

C4 Therapeutics

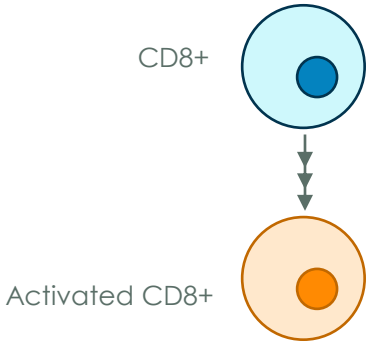
IKZF1/3 Degradation Drives Three Distinct Areas of Hematopoietic Biology; Degrading IKZF1/3 is a Validated Therapeutic Strategy in MM and NHL

IKZF1 / IKZF3 Transcription Factors

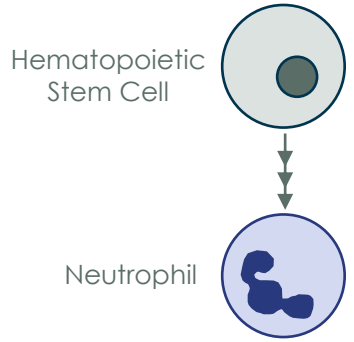
Drive MM and NHL Cell Growth and Survival



Activate Fully Differentiated T-cells



Regulate Hematopoietic Stem Cell Differentiation



Consequences of IKZF1/3 Degradation:

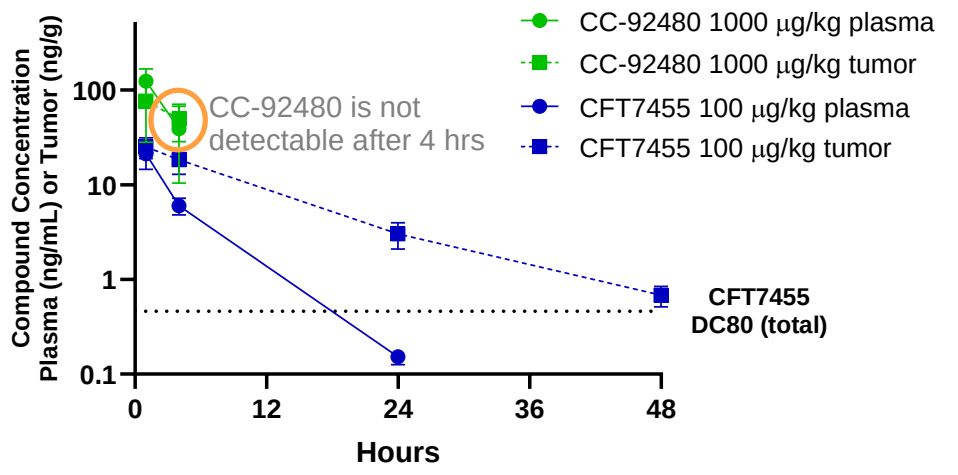
- **MM and NHL Cell Death**
- **T-cell Activation**
- **On-target Neutropenia**

Ikaros Family Zinc Finger proteins 1 and 3 (IKZF1/3); Multiple Myeloma (MM); Non-Hodgkin's Lymphoma (NHL)

Differentiated PK and Class-leading Catalytic Activity of Cemsidomide Leads to Sustained Degradation Compared to Other Agents in this Class

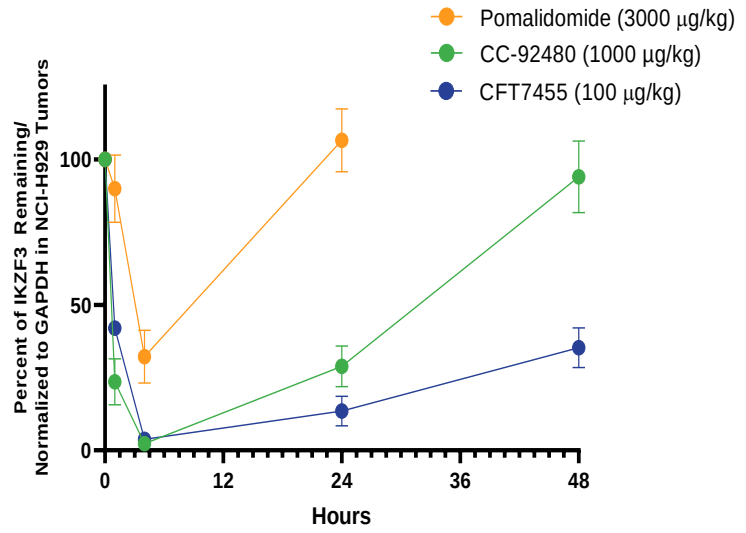
Extended Plasma and Tumor Exposure

In Vivo Tumor PK



Leads to Optimized Degradation Kinetics

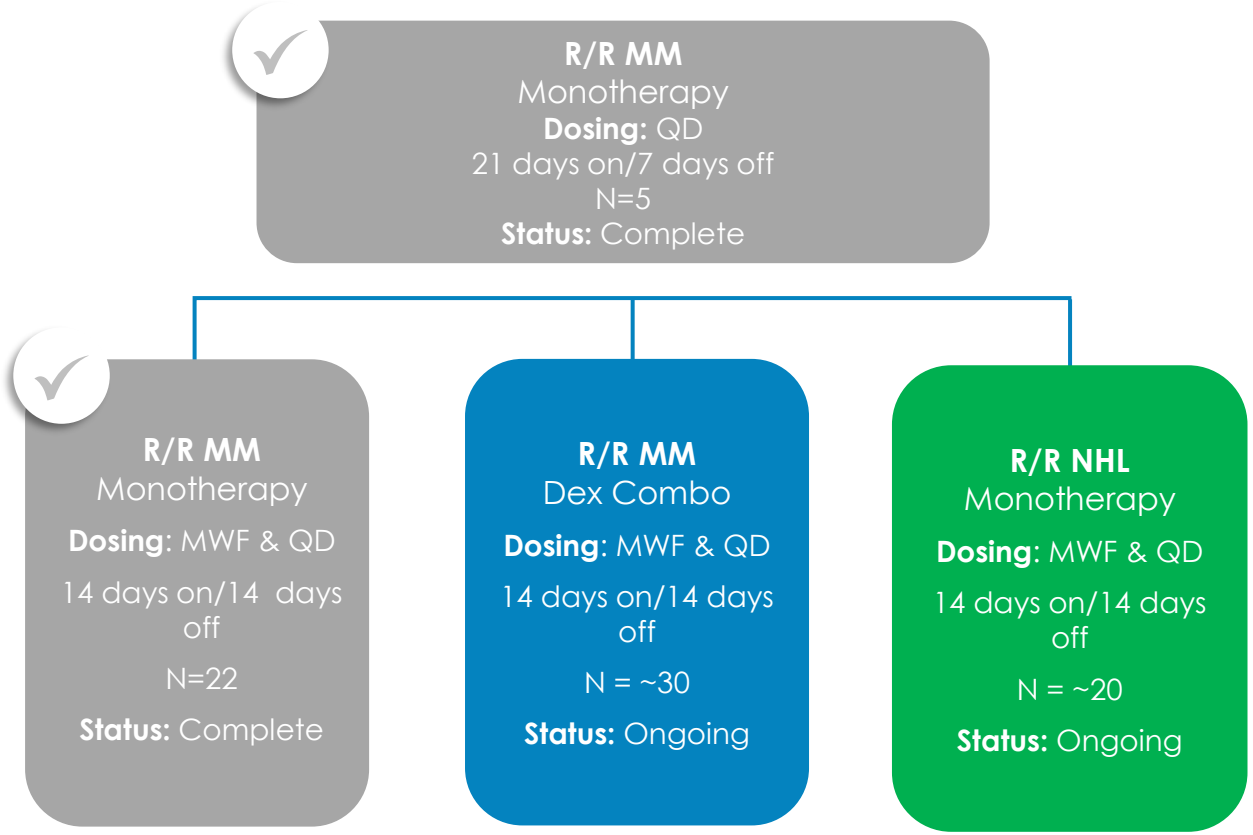
In Vivo Degradation Kinetics (48 hrs.)



mezigdomide (CC-92480); Ikaros family zinc finger protein (IKZF3); multiple myeloma (MM); pharmacodynamics (PD); pharmacokinetics (PK); once daily (QD)
Source: AACR 2022 presentation

Cemsidomide Phase 1 Dose Escalation Trial's Goal is to Define the Safety Profile and Identify Signs of Anti-Tumor Activity in R/R MM and R/R NHL

Phase 1 Dose Escalation Trial



Endpoints

Primary:

- Safety and tolerability
- Determine the maximum tolerated doses

Secondary:

- Estimate anti-tumor activity
- Assess PK

Exploratory:

- Characterize target engagement
- Assess kinetics, depth, recovery and consistency of target engagement
- Assess immunomodulation

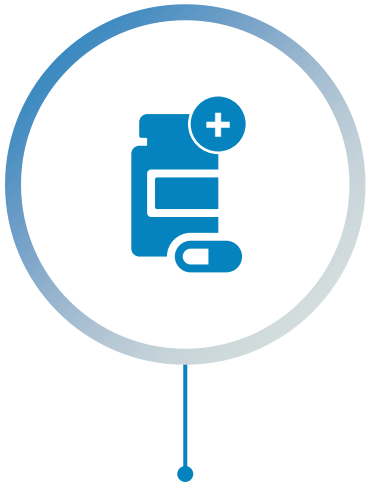
Pharmacokinetic (PK); Monday, Wednesday, Friday dosing (MWF); once daily (QD); Relapsed refractory multiple myeloma (R/R MM); Relapsed refractory non-Hodgkin's lymphoma (R/R NHL); Dexamethasone (Dex)

C4 Therapeutics

Schedule Adjustment Yielding Expected Results for Cemsidomide as a Potential MM Therapy

Established Safety Profile and Dosing Schedule

- Cemsidomide is well tolerated with no DLTs resulting in treatment discontinuations
- The 14 days on/14 days off schedule is optimal



Demonstrated Monotherapy Activity

- Anti-myeloma activity and immunomodulatory effects observed at well tolerated doses
- Opportunity in combination with novel MM agents for early-line patients and as a maintenance therapy option

Promising Responses with Cemsidomide + Dexamethasone

- Multiple patients achieved IMWG responses at low doses with best responses in patients refractory to BCMA therapies
- Opportunity in combination with dexamethasone for multi-refractory patients

Cemsidomide is a **potential treatment for multi-refractory MM patients** with the ability **to move into earlier lines** with numerous combination opportunities

Dose Limiting Toxicities (DLTs); Multiple myeloma (MM); B cell maturation antigen (BCMA); International Myeloma Working Group (IMWG)
Source: C4T data on file as 11/28/2023

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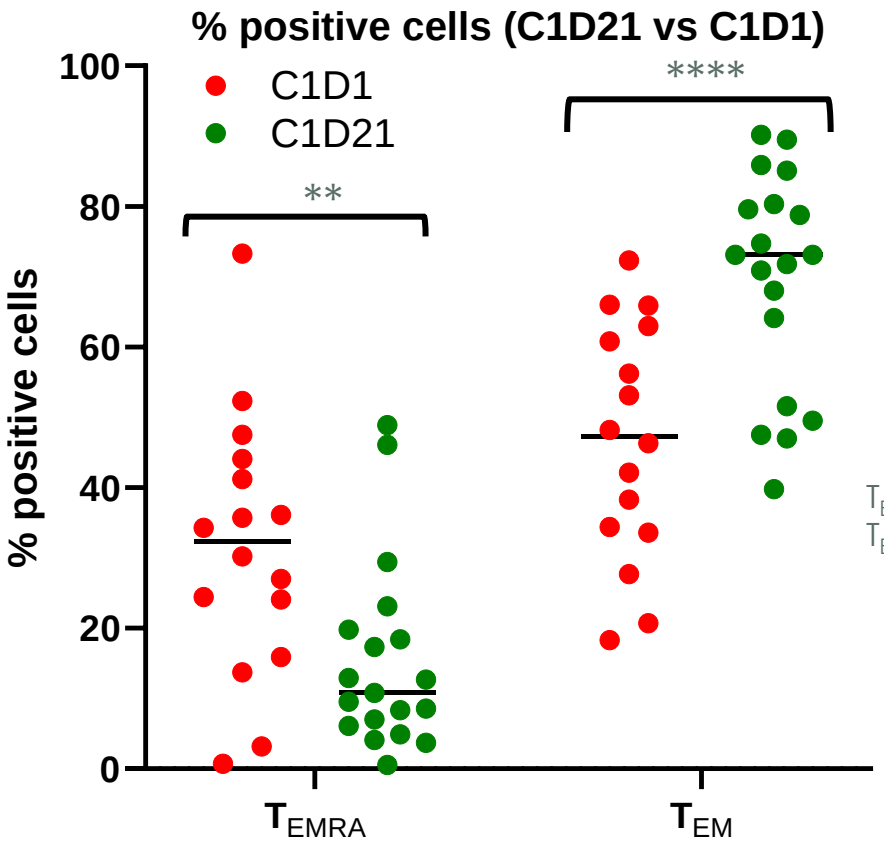
Clinical Evidence of Immune T-cell Activation with Cemsidomide Monotherapy



T_{EMRA}: Terminally differentiated T-cells
T_{EM}: Effector memory T-cells

- 19 patient samples (PBMCs) analyzed by flow cytometry
- Aggregate data of 25 µg, 50 µg, and 75 µg MWF and QD

Supports potential of cemsidomide as a maintenance therapy option and in combination with novel MM agents to improve efficacy:

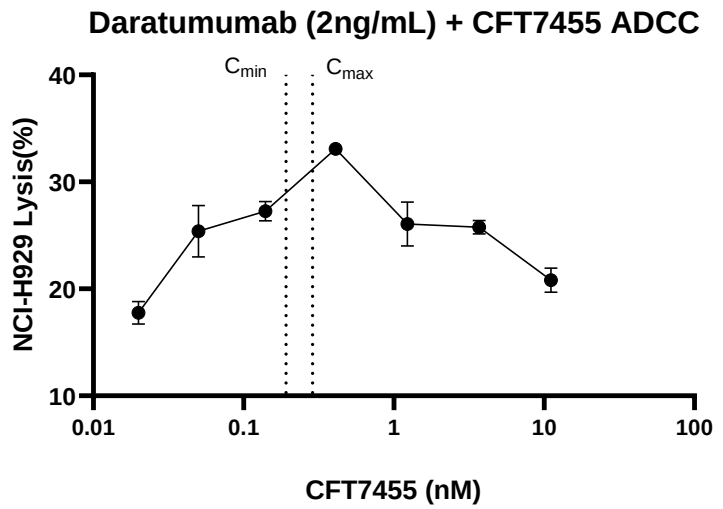
- ✓ Cemsidomide induces CD8+ T-cell activation by increasing effector memory T-cell subset
- ✓ T-cell activation is observed at well tolerated monotherapy clinical doses
- ✓ The clinical data consistent with the preclinical *in vitro* data reported for cemsidomide

Peripheral Blood Mononuclear Cells (PBMCs); Daily dosing (QD); Monday, Wednesday, Friday Dosing Schedule (MWF);Multiple Myeloma (MM)
Source: C4T data on file as of 11/28/2023

Cemsidomide Combined with Novel MM Agents Demonstrated Enhanced Immune Cell Lysis in Non-clinical Translational Models

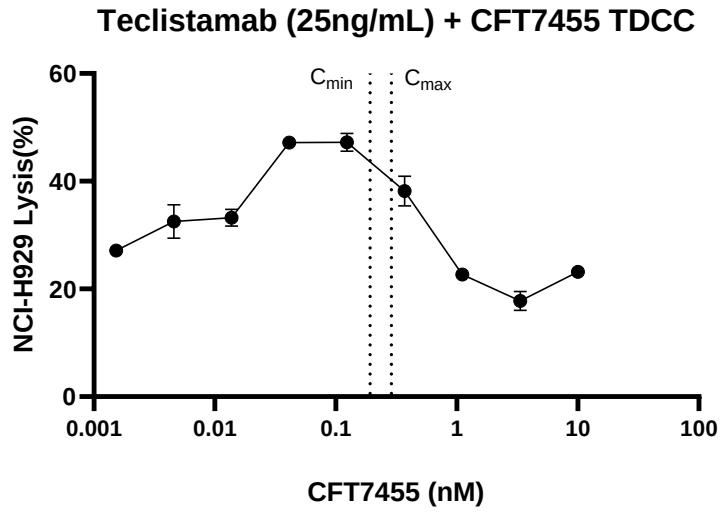
Daratumumab (Anti-CD38) Combo

Antibody-Dependent Cell-Mediated Cytotoxicity Assay (ADCC)



Teclistamab (BCMA BiTE) Combo

T-cell Dependent Cellular Cytotoxicity Assay (TDCC)



C_{min} and C_{max} represent human plasma concentrations for a 50 µg dose of cemsidomide

Bispecific T-Cell Engager (BiTE)
Source: C4T data on file as of 11/28/2023

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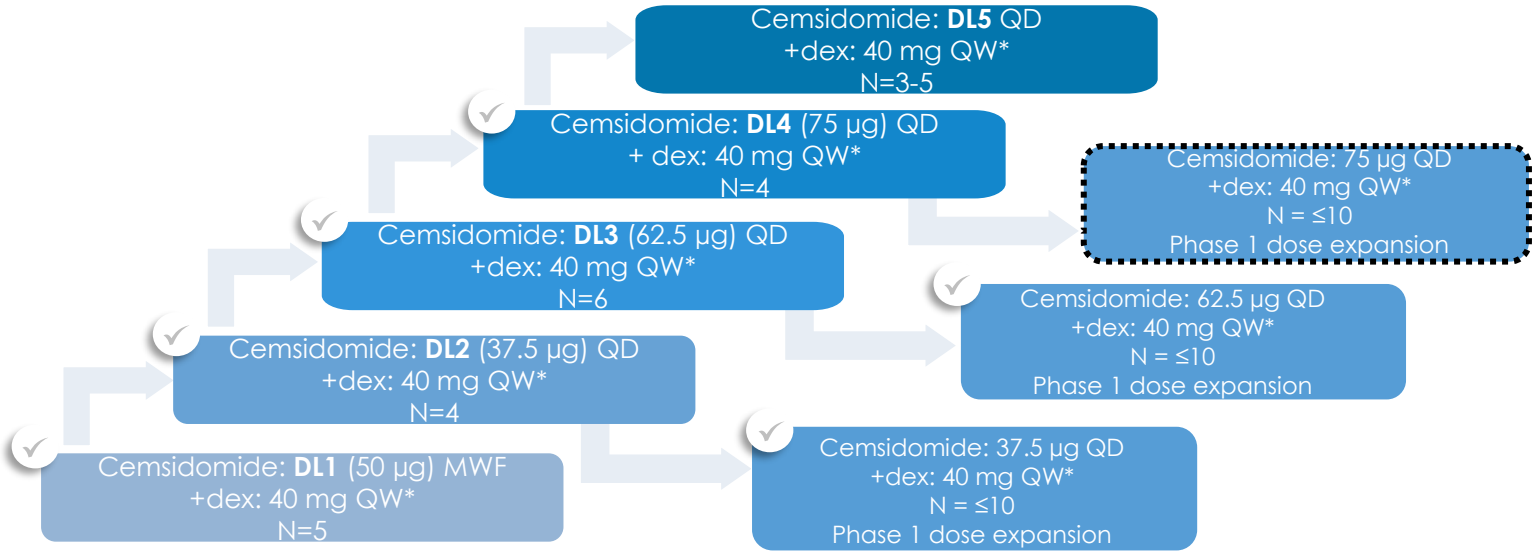
Cemsidomide + Dexamethasone Dose Escalation in R/R MM Continues to Progress

KEY INCLUSION CRITERIA

- Adults with R/R MM, at least 3 prior lines that have included lenalidomide, pomalidomide, a proteasome inhibitor, a glucocorticoid, and an anti-CD38 monoclonal antibody
- Nonresponsive to or progressed within 60 days of prior therapy
- Measurable disease
- Adequate bone marrow function (ANC ≥1000, Hgb ≥8.0, platelets ≥75,000)
- Creatinine clearance ≥40 mL/min
- ECOG ≤2

Phase 1: Dose Escalation + Dexamethasone 14 Days On/14 Days Off



- 75 µg dose has been declared safe
- Additional patients are enrolling at the 75 µg expansion cohort
- Dose escalation continues as maximum tolerated dose has not yet been reached

Phase 2

Cohort Expansion N=~30

Eastern Cooperative Oncology Group (ECOG); Monday, Wednesday, Friday dosing (MWF); Daily Dosing (QD); Relapsed/Refractory multiple myeloma (R/R MM); Absolute neutrophil count (ANC); Hemoglobin (Hgb); Dexamethasone (Dex); Dose level (DL)
*+Dex is dosed on days 1, 8,15, and 22 and dose is reduced for older patients.

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Cemsidomide + Dexamethasone is Well Tolerated and Best Responses in Patients Refractory to BCMA Therapies

Anti-myeloma Activity:

Dosing Schedule	Dose Level	EMD Status	Prior CAR-T or TCE	# of Prior Lines	Cycle 1	Cycle 2	Cycle 3	Cycle 4	Cycle 5	Cycle 6	Cycle 7	Cycle 8	Cycle 9	Cycle 10	Cycle 11
14 days on/ 14 days off	Cemsidomide 50 µg MWF +dex: 40 mg QW	No	No	6		PD									
		No	Yes	4		SD				PD					
		No	No	5		SD	MR		PR	→					
		Yes	Yes	12		PD									
		No	No	6		SD			■						
	Cemsidomide 37.5 µg QD +dex: 40 mg QW	No	Yes	5		VGPR	sCR		→						
		No	Yes	9		PR		→							
		Yes	No	7		SD	■								
		Yes	Yes	7		NE	→								

- Stringent Complete Response (sCR)
- Very good partial response (VGPR)
- Partial Response (PR)
- Minimal Response (MR)
- Stable Disease (SD)
- Progressive Disease (PD)
- Not Evaluable (NE)
- → Ongoing
- ■ Withdrawal of consent or physician decision

Safety:

- Cemsidomide + dexamethasone is well tolerated
- Consistent with the monotherapy safety signal
- No AEs have led to dose reductions, discontinuations or DLTs

Extramedullary Disease (EMD); T-Cell Engager (TCE); Daily Dosing (QD); One Weekly (QW); Monday, Wednesday, Friday Dosing (MWF); Dose Limiting Toxicity (DLTs); Dexamethasone (dex); B cell maturation antigen (BCMA); Adverse events (AEs)
Source: C4T data on file as of 11/28/2023

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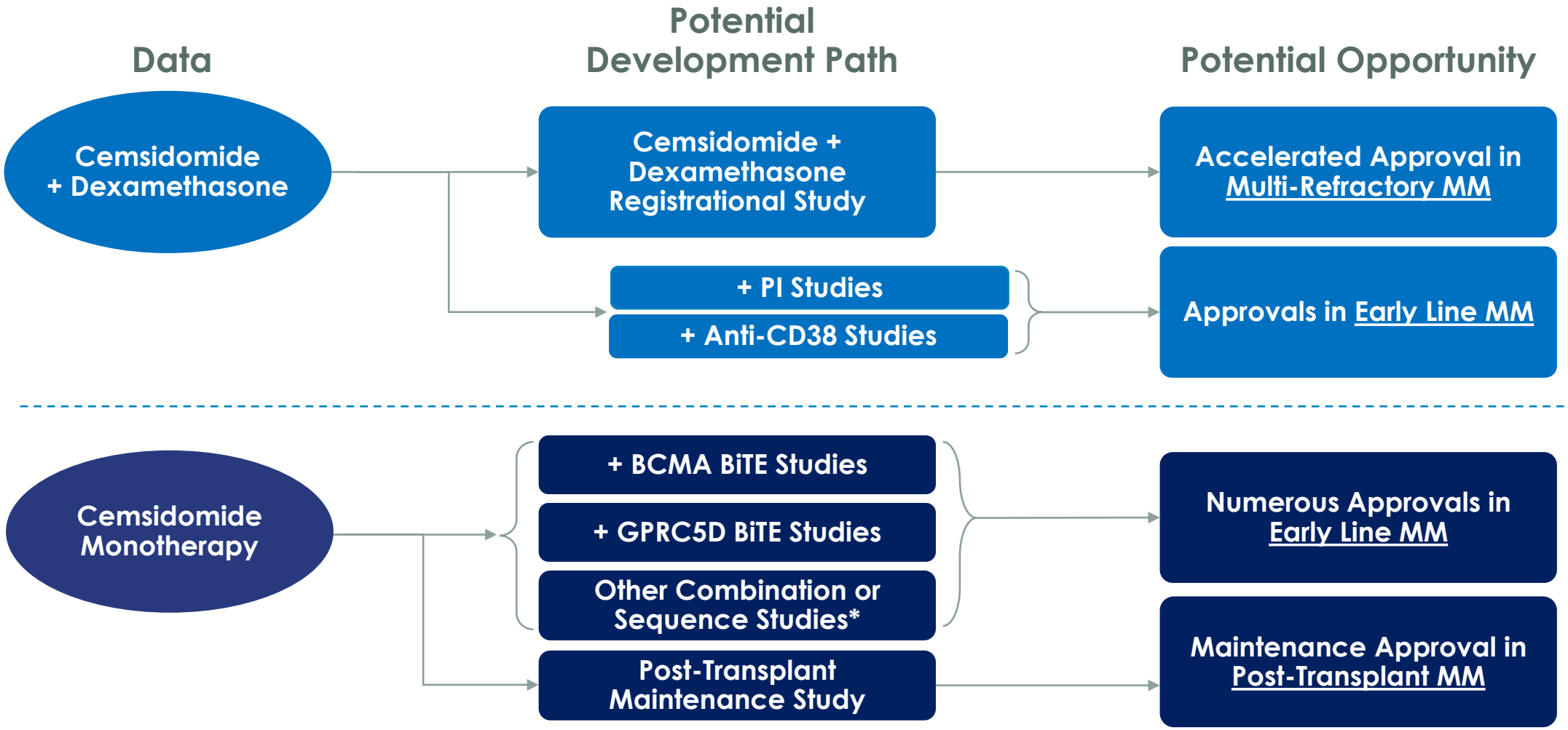
Cemsidomide Profile Supports Multiple Opportunities across MM Landscape



* Other combination opportunities may include CAR-T, anti-SLAMF7, XPO1 inhibitors, FcRH5 BiTE, among others.

Bi-specific T-cell Engager (BiTE); Proteasome Inhibitors (PI); Multiple myeloma (MM); B cell maturation antigen (BCMA); G protein–coupled receptor, class C, group 5, member D (GPRC5D)

CFT1946

Targeting BRAF V600 Mutant

Melanoma, Colorectal (CRC)
& Non-Small Cell Lung Cancer (NSCLC)

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CFT1946 has the Potential to Overcome Several Shortcomings Seen with Inhibitors for BRAF V600X Cancers

Key Limitations of Approved BRAF Inhibitors:

- **Durable and deep responses are often not seen** in melanoma, NSCLC and CRC patients, due to **MAPK pathway resistance**
- **Poor tolerability**, such as high-rates of cutaneous adverse events
- Often **combined with a MEK inhibitor** to **enhance both efficacy and minimize side effects resulting from paradoxical activation** by BRAF inhibitors
- **Limited approved treatment options** for BRAF V600 **patients who do not have a BRAF V600E or V600K mutation**

Despite limitations, current BRAF inhibitor market is **~$2B**[2]

BRAF inhibitor market is estimated to grow to

~$3B by 2028[2]

Potential Advantages of CFT1946, a Novel, Oral, BRAF V600 Mutant BiDAC degrader:

- ✓ Prevents BRAF V600 mutant **mono/heterodimer formation**[1]
- ✓ **Avoids paradoxical activation** seen with approved inhibitors[1]
- ✓ **Addresses MAPK pathway alterations** resulting from BRAF inhibitor resistance (e.g., BRAF splice variants, BRAF amplification)[1]
- ✓ **Specifically targets BRAF V600 mutations**, which includes BRAF V600 mutations beyond BRAF V600E
- ✓ Spares wild-type BRAF[1], likely **avoiding AEs associated with inhibition of wild-type BRAF**
- ✓ Enables deep elimination of mutant BRAF signaling to **create potential durable responses** through degrader molecule recycling and catalytic effect

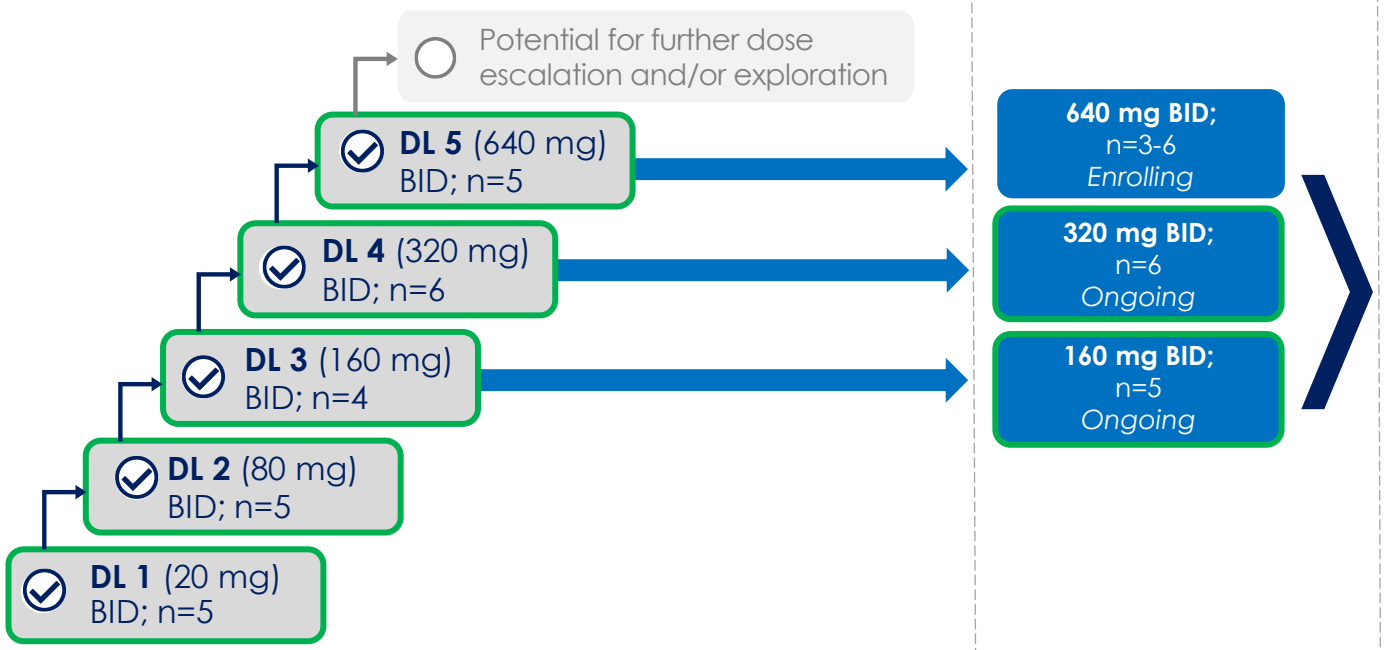
[1]Kreger B et al. Abstract 1658, AACR 2024; [2]Evaluate Pharma 2023
Adverse event (AE); Mitogen-activated protein kinase (MAPK)

CFT1946 Phase 1/2 Dose Escalation Trial Continues to Progress Across BRAF V600 Mutant Driven Solid Tumors

KEY INCLUSION CRITERIA[1]

- Evidence of BRAF V600 mutation obtained from tumor tissue or liquid biopsy
- BRAF V600 mutant measurable solid tumors with ≥1 prior line of SoC therapy for unresectable locally advanced or metastatic disease
- Melanoma patients must have received prior BRAF inhibitor therapy
- CRC, ATC, NSCLC or other non-CNS solid tumors: prior BRAF inhibitor therapy unless not available per SoC
- No patient with CNS involvement (primary tumor or metastatic disease), except if clinically stable

MONOTHERAPY DOSE ESCALATION



PK, PD, ANTI-TUMOR ACTIVITY EVALUATION[2]

- **640 mg BID;** n=3-6 *Enrolling*
- **320 mg BID;** n=6 *Ongoing*
- **160 mg BID;** n=5 *Ongoing*

PRIMARY ENDPOINTS

- Safety and tolerability
- Determine RP2D/MTD

SECONDARY ENDPOINTS

- Estimate anti-tumor activity
- Assess PK and PD

Exploratory Expansion: CFT1946 monotherapy in melanoma 640 mg BID *Enrolling*

Exploratory Expansion: CFT1946 monotherapy in melanoma 320 mg BID *Ongoing*

Phase 1B: CFT1946 in combination with cetuximab in CRC 160 mg BID *Enrolling*

Phase 1B: CFT1946 in combination with trametinib for melanoma and NSCLC *Pending*

[1]NCT05668585. www.clinicaltrials.gov. Accessed 01/09/2024; [2]Evaluating additional patients for pharmacodynamic assessment pre- and post-drug exposure biopsies
Colorectal cancer (CRC); Anaplastic thyroid cancer (ATC); Non-small cell lung cancer (NSCLC); Central nervous system (CNS); Standard of care (SoC); Dose Level (DL); Twice daily (BID); Recommended Phase 2 dose (RP2D); Maximum tolerated dose (MTD); Pharmacokinetic (PK); Pharmacodynamic (PD)

CFT1946 Monotherapy Phase 1 Data Demonstrate Proof of Mechanism and Provide Early Evidence of Proof of Degrader Concept

Proof of Mechanism

- **Well tolerated** and **highly selective degrader**, results in **no Grade ≥ 3 cutaneous adverse events**, which are commonly seen with wild-type BRAF inhibition
- **Increased drug exposure** observed with dose escalation
- **Degraded BRAF V600E** protein in all available post-treatment biopsies collected to date

Proof of Degrader Concept

- Early evidence of monotherapy **anti-tumor activity** in patients who progressed after treatment with BRAF inhibitors
- Anti-tumor activity seen **across multiple BRAF V600 mutants**
- Degradation of mutant BRAF protein overcomes resistance mechanisms and results in potentially **deeper** and more **durable responses than BRAF inhibitors**

CFT1946 has the potential to **disrupt the treatment landscape** and become an **important option for patients** with **BRAF V600 mutant driven solid tumors**

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No Discontinuations, Dose Interruptions or Reductions Due to CFT1946 Treatment-related Adverse Events

	20 mg BID (n=5) n (%)	80 mg BID (n=5) n (%)	160 mg BID (n=9) n (%)	320 mg BID (n=12) n (%)	640 mg BID (n=5) n (%)	Total (n=36) n (%)
Patients with any TEAEs	4 (80)	4 (80)	7 (78)	11 (92)	5 (100)	31 (86)
Grade ≥ 3 TEAEs	3 (60)	2 (40)	3 (33)	3 (25)	3 (60)	14 (39)
TEAEs related to CFT1946	0	1 (20)	3 (33)	9 (75)	3 (60)	16 (44)
Grade ≥ 3 TEAEs related to CFT1946	0	0	0	0	1 (20)*	1 (3)
Any TESAEs	1 (20)	3 (60)	1 (11)	2 (17)	2 (40)	9 (25)
TESAEs related to CFT1946	0	0	0	0	0	0
TEAEs leading to CFT1946 discontinuation	1 (20)	1 (20)	1 (11)	0	0	3 (8)
TEAEs leading to CFT1946 interruption	1 (20)	2 (40)	2 (22)	2 (17)	2 (40)	9 (25)
TEAEs leading to CFT1946 reduction	0	0	1 (11)	0	0	1 (3)
TEAEs leading to death	0	1 (20)#	0	0	0	1 (3)
TRAEs leading to CFT1946 discontinuation, interruption, reduction or death	0	0	0	0	0	0
Patients with DLTs	0	0	0	0	0	0

*Grade 3 hypertension possibly related to CFT1946 with no dose change #Adverse event of cerebrovascular accident leading to death, which was not related to CFT1946
Treatment-emergent adverse events (TEAEs); Treatment-emergent serious adverse event (TESAEs); Treatment-related adverse event (TRAE); Dose limiting toxicities (DLTs); Twice daily (BID)
Source: ESMO Congress 2024; C4T data as of 07/19/2024

Well Tolerated Monotherapy Safety Profile, Consistent with BRAF V600 Mutant Selectivity Design of CFT1946

- No DLTs
- Majority of TEAEs observed were mild to moderate
- No treatment-related SAEs
- No Grade ≥ 3 treatment-related cutaneous adverse events
- No new primary malignancies

Summary of TEAEs ≥ 10% of 36 patients treated with CFT1946

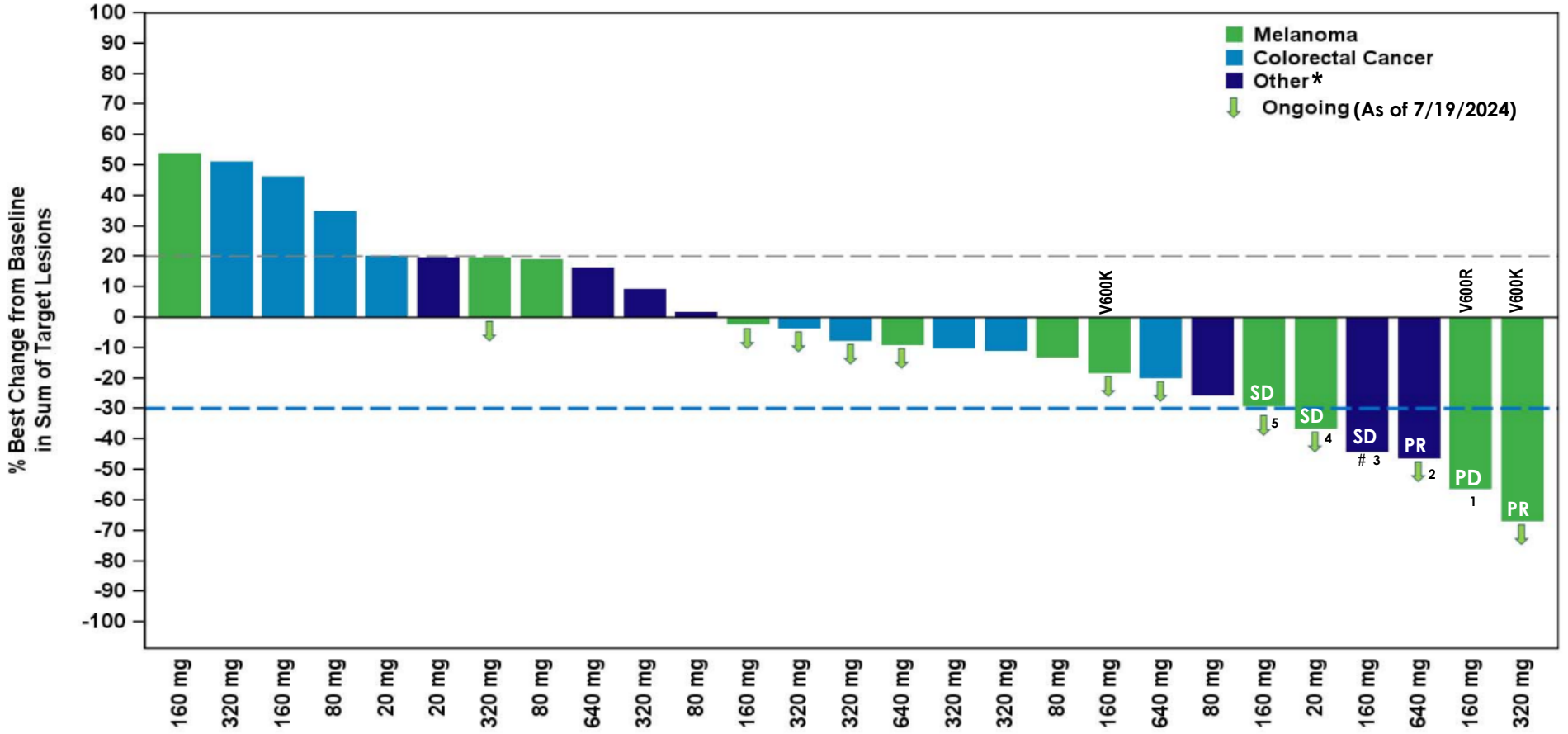
Preferred Term	Grade 1 n (%)	Grade 2 n (%)	Grade 3 n (%)	Grade 4 n (%)	Grade 5 n (%)	Total (n=36) n (%)
Patients with any TEAEs^	3 (8)	14 (39)	11 (31)	2 (6)	1 (3)#	31 (86)
Anemia	1 (3)	4 (11)	2 (6)	0	0	7 (19)
Abdominal pain	4 (11)	1 (3)	2 (6)	0	0	7 (19)
Peripheral edema	5 (14)	1 (3)	0	0	0	6 (17)
Pyrexia	4 (11)	2 (6)	0	0	0	6 (17)
Fatigue	1 (3)	4 (11)	0	0	0	5 (14)
Lipase increased	3 (8)	2 (6)	0	0	0	5 (14)
Back pain	1 (3)	2 (6)	1 (3)	0	0	4 (11)
Hypophosphatemia	1 (3)	3 (8)	0	0	0	4 (11)
Constipation	1(3)	2 (6)	0	0	0	4 (11)*

^A patient is only counted once with the highest severity and preferred term
#Patient had a fatal cerebrovascular accident not related to CFT1946
CTCAE v5.0 grading criteria; *Grade missing for 1 patient with TEAE

Serious adverse events (SAEs); Dose limiting toxicities (DLTs); Treatment-related adverse events (TRAES); Treatment-emergent adverse events (TEAEs)

Source: ESMO Congress 2024; C4T data as of 7/19/2024

Early Signs of Anti-tumor Activity: 59% (16/27) Patients Demonstrated Target Lesion Tumor Reductions with 11 Efficacy Evaluable Patients Continuing Treatment



*Other tumor types include cholangiocarcinoma, non-small cell lung cancer, pancreatic carcinoma, and small intestine cancer; BRAF V600 mutation is V600E unless otherwise specified; #This patient did not receive prior BRAF inhibitor therapy, all other patients received prior BRAF inhibitor therapy. Dotted lines represent partial response (-30%, blue line) and progressive disease (20%, gray line) per RECIST v1.1.

[1] Patient on 160 mg BID had 56.2% reduction on target lesion, progression on non-target lesion and a new lesion, hence assessed as PD for overall response; [2] Patient on 640 mg BID had PR confirmed after data cut off; [3] Patient on 160 mg BID had PD following first PR (-43.9%), hence assessed as SD for overall response; [4] Patient on 20 mg BID had unconfirmed PR, hence assessed as SD for overall response; [5] Patient on 160 mg BID had -29% reduction on target lesion, hence assessed as SD

Source: ESMO Congress 2024; C4T data on file as of 7/19/2024

CFT1946 has the Potential to Address Multiple Tumor Types with BRAF V600X Mutations Where BRAF Inhibitors are Insufficient

	BRAF V600X Mutation Rate	2023 U.S. Incidence of BRAF V600X Patients[4]	Approved BRAF Inhibitors	BRAF Inhibitor Regimen mPFS[5]
 Melanoma	~35%[1]	~35,000	• Dabrafenib • Encorafenib • Vemurafenib *All used in combination with MEK inhibitors*	**11.4 months** (dabrafenib + trametinib in 1L+)
 Colorectal Cancer	5-10%[2]	~11,000	• Encorafenib *Used in combination with cetuximab (anti-EGFR)*	**4.2 months** (encorafenib + cetuximab in 2L+)
 Non-Small Cell Lung Cancer	1-2%[3]	~3,000	• Dabrafenib • Encorafenib *Both used in combination with MEK inhibitors*	**15.2 months** (dabrafenib + trametinib in 2L+)

Sources: 1. Owsley 2021 Exp Biol Med. 2. Paik 2011 J Clin Oncol. 3. Bylsma 2020 Cancer Med. 4. NCI SEER, consulting work done by Health Advances. 5. FDA Labels

On Track for Multiple CFT1946 Milestones in 2025

2024

- ✓ Initiated monotherapy melanoma expansion cohort at 640 mg
- Continue dose escalation beyond 640 mg if absorption/exposure data supports
- Initiate Phase 1b portion of the trial evaluating CFT1946 in combination with trametinib for melanoma by **year-end**

2025

- Complete CFT1946 monotherapy dose escalation portion of ongoing Phase 1 trial and present full data in **2025**
- Data from Phase 1 expansion cohorts evaluating CFT1946 monotherapy for melanoma expected in **2025**
- Data from Phase 1b portion of the trial evaluating CFT1946 in combination with cetuximab for CRC expected in **2025**

2026

Initiate registrational trial(s) to position CFT1946 as BRAF therapy of choice

CFT8919

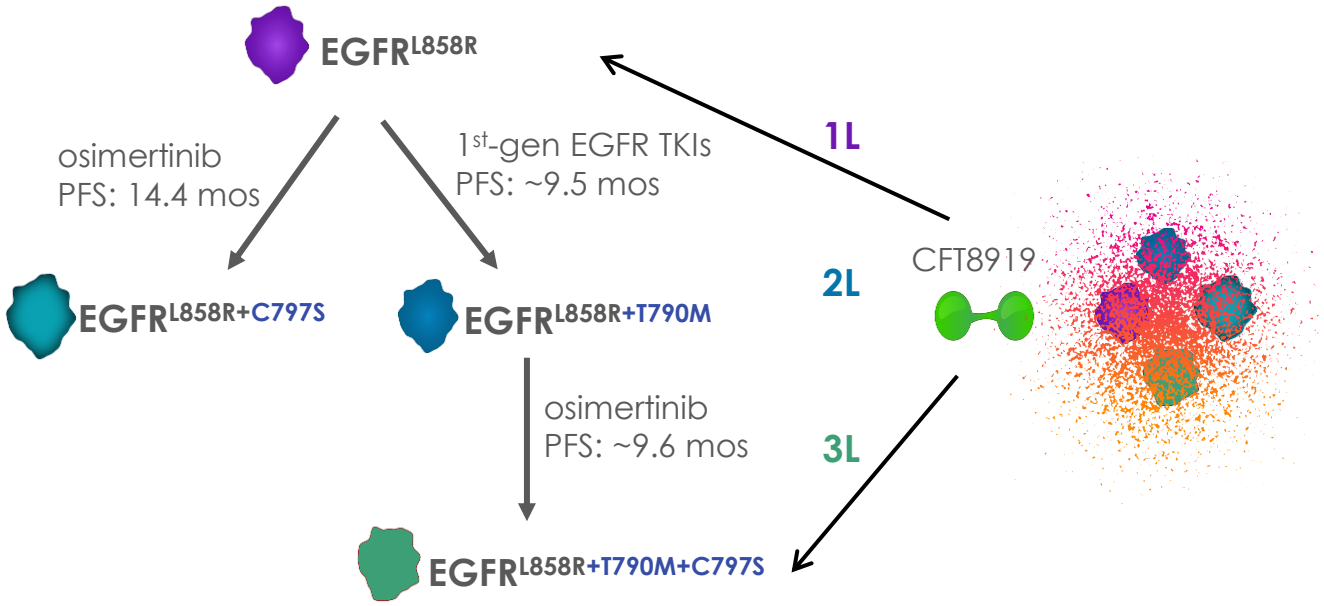
Targeting EGFR L858R

Non-Small Cell Lung Cancer (NSCLC)

C4 Therapeutics

Potential for CFT8919 to Improve Outcomes for NSCLC Patients with EGFR L858R Mutations

Strong Rationale for an EGFR L858R Degrader



Osimertinib and other inhibitors provide suboptimal responses in L858R mutant NSCLC compared to other mutations of NSCLC

Osi: 14.4 months PFS versus 21.4 months PFS
(L858R) (Exon 19 deletion)

CFT8919 Key Properties

- Orally bioavailable
- Potent and selective against L858R, regardless of secondary mutations
- Allosteric binding

Market Size

- ~$6B approved EGFR inhibitor market[1]

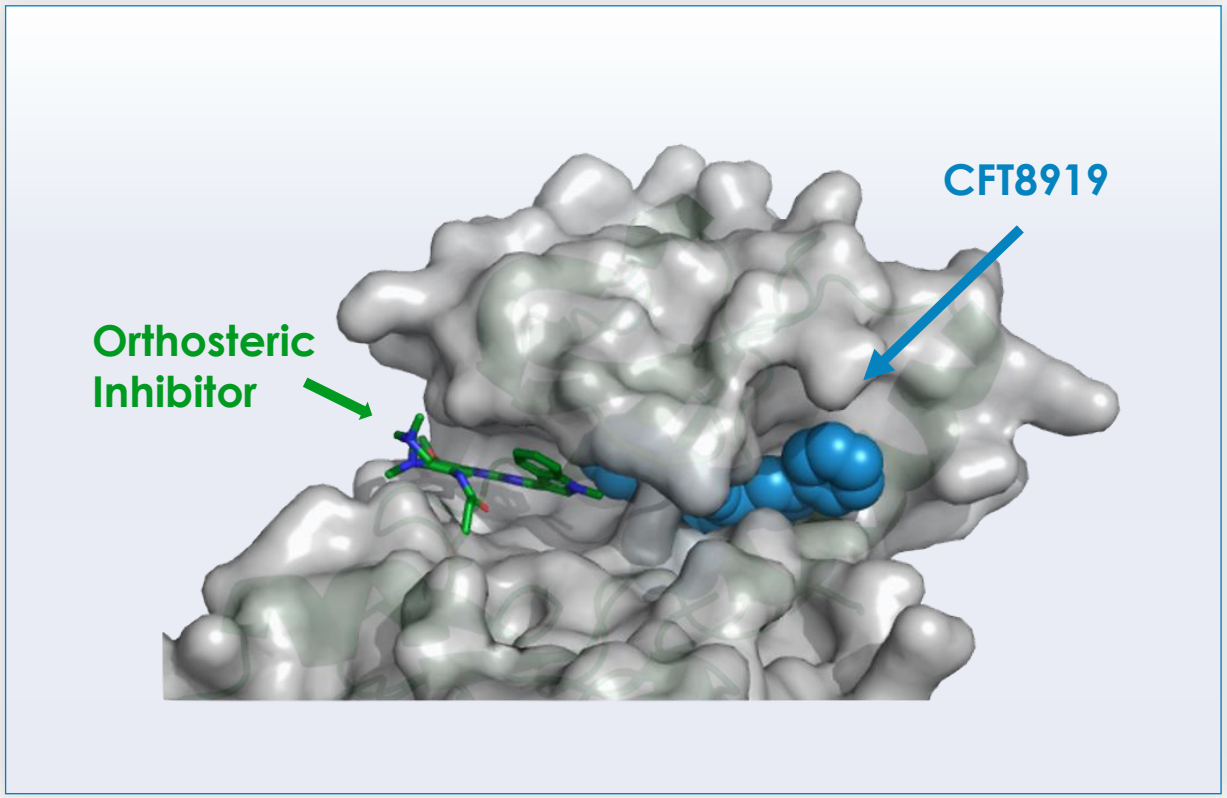
Progress to Date

- Achieved FDA clearance of U.S. IND
- Betta received CTA clearance from China's NMPA

Non-small cell lung cancer (NSCLC); Tyrosine Kinase Inhibitor (TKI); Osimertinib (Osi); Investigational New Drug (IND); Clinical Trial Application (CTA)
Sources: Soria, J.C. et al. NEJM 378, 113–125 (2018); Sher, T. et al, Mayo Clin. Proc. 83, 355-367 (2008); 1. 2023 market size from EvaluatePharma.

C4 Therapeutics

CFT8919 is a Potent, Oral, Allosteric, Mutant-selective Degrader of EGFR L858R



- CFT8919 exploits **allosteric binding site**, close to L858R activating mutation
- Allosteric binding site avoids known resistance-causing mutations in **orthosteric binding site**
- Allosteric binders do not require covalent binding through C797S and do not compete with orthosteric binding

Allosteric binding avoids resistance mutations, wild-type activity, and is combinable with orthosteric inhibitors

C4T is On Track to Execute Across All 2024 Goals, Progressing Multiple Clinical and Preclinical Programs

Cemsidomide
IKZF1/3

- **ASH 2024 (Dec.):** Present updated data from Phase 1 dose escalation +dex trial in R/R MM
- **ASH 2024 (Dec.) :** Present data from Phase 1 dose escalation monotherapy trial in R/R NHL
- **By YE 2024:** Complete Phase 1 dose exploration in R/R MM and R/R NHL

CFT1946
BRAF V600 Mutant

- **2Q 2024:** Present preclinical data demonstrating differentiated activity in BRAF V600 mutant driven melanoma, CRC, NSCLC, and brain metastasis models at AACR
- **ESMO Congress 2024:** Present monotherapy data from Phase 1 dose escalation trial in melanoma, CRC, NSCLC and other BRAF V600 mutant driven cancers

CFT8919
EGFR L858R

- **2024:** Support trial start-up activities related to Betta's Phase 1 dose escalation trial in China

Discovery

- **1Q 2024:** Collaboration with Merck KGaA, Darmstadt, Germany to discover two targeted protein degraders against critical oncogenic proteins
- **2024:** Deliver development candidate to collaboration partner

Runway into 2027, Beyond Value Inflection Milestones

Relapsed or refractory multiple myeloma (R/R MM); Relapsed or refractory non-Hodgkin lymphoma (R/R NHL); Colorectal cancer (CRC); Non-small cell lung cancer (NSCLC)