UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): June 11, 2026

C4 THERAPEUTICS, INC.
(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-39567**	**47-5617627**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**
490 Arsenal Way, Suite 120 Watertown, MA		**02472**
(Address of Principal Executive Offices)		**(Zip Code)**

Registrant's Telephone Number, Including Area Code: (617) 231-0700

Not Applicable
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, $0.0001 par value per share	CCCC	The Nasdaq Global Select Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

On June 11, 2026, C4 Therapeutics, Inc. (the "Company") posted a a corporate presentation on its website at https://ir.c4therapeutics.com/events-presentations. A copy of the presentation is furnished herewith as Exhibit 99.1 to this Current Report on Form 8-K.

The information contained in Item 7.01 of this Current Report on Form 8-K and Exhibit 99.1 attached hereto is intended to be furnished and shall not be deemed "filed" for purposes of Section 18 of the Exchange Act, or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act or the Exchange Act, except as expressly set forth by specific reference in such a filing. The furnishing of this information hereby shall not be deemed an admission as to the materiality of any such information.

Item 8.01 Other Events.

On June 11, 2026, the Company posted on its website a poster presentation with further analysis from its fully enrolled Phase 1 trial of cemsidomide, a next-generation oral IKZF1/3 degrader, in combination with dexamethasone for the treatment of relapsed/refractory multiple myeloma ("RRMM"), to be presented at the European Hematology Association 2026 Congress ("EHA 2026 Congress"). A copy of the poster presentation, which has been published to the "Events & Presentations" section of the Company's website, is filed as Exhibit 99.2 to this Current Report on Form 8-K and is incorporated herein by reference.

On June 11, 2026, the Company also issued a Press Release reporting further analysis from the Phase 1 trial of cemsidomide in combination with dexamethasone for the treatment of RRMM to be presented at the EHA Congress. A copy of the Press Release is filed as Exhibit 99.3 to this Current Report on Form 8-K and is incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits. The exhibits shall be deemed to be filed or furnished, depending on the relevant item requiring such exhibit, in accordance with the provisions of Item 601 of Regulation S-K (17 CFR 229.601) and Instruction B.2 to this form.

Exhibit Number	Description
99.1	Corporate presentation of the Company dated June 2026
99.2	Poster from C4 Therapeutics, Inc.'s EHA 2026 Congress Presentation, dated June 11, 2026
99.3	Press release issued June 11, 2026
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

Sounds goofd

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

C4 Therapeutics, Inc.

Date: June 11, 2026

By: /s/ Kendra R. Adams

Kendra R. Adams

Chief Financial Officer and Treasurer

C4 Therapeutics

Protein degraded.
Disease targeted.
Lives transformed.

June 2026

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. The forward-looking statements included in this presentation speak only as of the date hereof and 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.

This presentation also contains estimates, projections and other information concerning the markets for C4 Therapeutics, Inc.'s product candidates, including data regarding the estimated size of those markets, and the incidence and prevalence of certain medical conditions and patient use of medicines. Information that is based on estimates, forecasts, projections, market research, or similar methodologies is inherently subject to uncertainties and actual events, and circumstances may differ materially from events and circumstances reflected in this information. Unless otherwise expressly stated, the Company obtained this industry, business, market and other data from reports, research surveys, clinical trials studies and similar data prepared by market research firms and other third parties, from industry, medical and general publications, from other publicly available information, and from government data and similar sources.

INTELLECTUAL PROPERTY

C4 Therapeutics, Inc., owns various registered and unregistered trademarks and service marks in the U.S. and internationally, including, without limitation, C4 THERAPEUTICS, our housemark logo, the name of our TORPEDO platform, and the names of our BIDAC and MONODAC degrader products. All trademarks, service marks, or trade names referred to in this presentation that we do not own are the property of their respective owners. Solely for convenience, the trademarks, service marks, and trade names in this presentation are referred to without the symbols ®, SM and ™, but those references should not be construed as any indicator that their respective owners will not assert, to the fullest extent under applicable law, their rights to these trademarks, service marks, or trade names.

Advancing Differentiated TPD Medicines and Building a Sustainable Pipeline of High-value Degraders To Achieve Our Vision

Potential Best-in-Class IKZF1/3 Degrader for MM

Establishing cemsidomide as a potential **foundational therapy** for the treatment of MM **across multiple lines of therapy**

Discovery Strategy Focused on INN
*(**I**nflammation, **N**euroinflammation, and **N**eurodegeneration)*

Progressing **potential first-in-class** degraders focused on **INN** diseases to build a sustainable pipeline

Platform Collaborations Expand TPD Reach

Leveraging discovery collaborations to **generate non-dilutive capital** and **to realize our full potential of TPD**

Financial Strength to Execute

Cash runway expected to **end of 2028,** beyond key value inflection points across the portfolio

Vision:
To become a ful integrated biopharmaceutic company

STRATEGY: DEVELOP BEST-IN-CLASS AND FIRST-IN-CLASS DEGRADERS. VALIDATED PATHWAYS. LARGE MARKET OPPORTUNITIES

Multiple myeloma (MM); Non-small cell lung cancer (NSCLC); Targeted Protein Degradation (TPD)

C4T is Focused on Advancing Potential Best-in-Class And First-in-Class Degraders Across Clinical Oncology Portfolio and INN Discovery Strategy

Q1 2026 Key Accomplishments:

- First patient dosed in cemsidomide Phase 2 MOMENTUM trial
- First patient dosed in cemsidomide Phase 1b trial in combination with elranatamab
- Expanded long-term partnership with Roche through new collaboration agreement focused on discovering and developing DACs[2]
- Received a $2 million milestone payment for designing and delivering a second degrader to Biogen for clinical development
- Shared plan to initiate a Phase 1b trial evaluating cemsidomide with approved multiple myeloma therapies

2026

Advance potential **best-in-class** and **first-in-class** degraders

- **Enroll 2 clinical trials** with **cemsidomide** to address 2L+ and 4L+ opportunities in MM
- **Establish combinability profile** with cemsidomide + elranatamab[1]
- **Optimize indication selection** for multiple targets across discovery portfolio

2027

Position for **regulatory success** and **pipeline build**

- **Complete enrollment** for Phase 2 MOMENTUM trial
- **Initiate additional Phase 1b trial**
- **Present two cemsidomide data readouts**:
  - Initial ORR data from Phase 2 MOMENTUM trial
  - Phase 1b data w/ elranatamab[1] to support advancement to Phase 3 trial
- **Start up activities** for **Phase 3 cemsidomide + BCMAxCD3 Bispecific**
- **Advance internal discovery pipeline** to enable INDs

2028

Unlock value across portfolio

- **Initiate** and **enroll Phase 3 trial** of cemsidomide + BCMAxCD3 Bispecific
- **Present efficacy** and **safety d** from the Phase 2 MOMENTUM trial
- **Potentially submit NDA** for cemsidomide
- **Deliver 3 potential INDs** from discovery pipeline in INN indications

[1] Pfizer supplying elranatamab (ELREXFIO®), a B-cell maturation antigen CD3 targeted bispecific antibody, to C4T for the Phase 1b trial; [2] Announced collaboration agreement on April 9, 2026 (https://ir.c4therapeutics.com/news-releases/news-release-det therapeutics-expands-long-term-partnership-roche-through-new)
Dexamethasone (dex); Inflammation, Investigational new drug (IND); New Drug Application (NDA); Overall response rate (ORR); Inflammation, Neuroinflammation, Neurodegeneration (INN); Accelerated approval (AA); Multiple myeloma (MM); Degrader conjugates (DACs)

C4 Therapeutics

Focused Pipeline Advancing Clinical Oncology Degraders and a New Discovery Strategy in Inflammation, Neuroinflammation & Neurodegeneration (INN) Diseases

	PROGRAM	TARGET	INDICATIONS	RESEARCH & PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	NEXT MILESTO
CLINICAL ONCOLOGY PORTFOLIO	Cemsidomide	IKZF1/3	4L+ Multiple Myeloma	Phase 2 MOMENTUM trial w/ dex				**Q1 2027:** Complete enrollment **2H 2027:** Present ini ORR data
			2L+ Multiple Myeloma	Phase 1b trial w/ elranatamab[2] Pfizer				**2H 2026:** Provide incremental updat **Mid-2027:** Present Phase 1b data fror cohorts
	CFT8919[1]	EGFR L858R	Non-Small Cell Lung Cancer	BETTA				
INN DISCOVERY	Discovery	*Novel targets in pathways of:* -IL-23/IL-17 -Type 1 IFN -MAPK, PI3K/AKT, NF-kB	INN *Inflammation, Neuroinflammation & Neurodegeneration*					**By year-end 2026:** Optimize indicatio selection for multip targets

[1] License and collaboration agreement with Betta Pharmaceuticals for development and commercialization in Greater China
[2] Pfizer supplying elranatamab (ELREXFIO®), a B-cell maturation antigen CD3 targeted bispecific antibody, to C4T for the Phase 1b trial
Dexamethasone (dex)

C4 Therapeutics

Strategic Platform Collaborations Expand Potential Reach of C4T TPD Medicines

Roche	Merck KGaA Darmstadt, Germany	Biogen
Ongoing Collaborations 1) Evaluating targets in autoimmune diseases & oncology ✓ Advanced two programs to preclinical milestones[1] 2) Discovering and developing DACs for two programs against oncology targets	Discovering targeted protein degraders against critical oncogenic proteins ✓ Achieved preclinical milestone from a project within the KRAS family	Delivered two development candidates (IRAK4 and BTK) for non-oncology targets[2] ✓ Both development candidates are now in Phase 1 clinical development

By year-end 2026: Deliver at least one development candidate to collaboration partner

[1] Earned and received preclinical milestones in Q1 2025
[2] Delivered development candidates to Biogen in Q1 2025 and Q3 2024. In Q3 2025, the IRAK4 degrader, BIIB142, entered Phase 1 clinical development and in Q1 2025, the BTK degrader, entered Phase 1 clinical development
Targeted Protein Degradation (TPD); Degrader antibody conjugates (DACs)

Cemsidomide

IKZF1/3 Degrader

Multiple Myeloma

C4 Therapeutics

Cemsidomide is Positioned for Success in Multiple Myeloma

Despite recent approval for immune-based therapies in the MM landscape, **IKZF1/3 are central drivers of MM development and progression, thus IKZF1/3 degraders** will **remain relevant across multiple lines and in combinations**

Cemsidomide has a **potential best-in-class profile** among other IKZF1/3 degraders, including CELMoDs®, in a **large** and **growing multiple myeloma market with a clinically and commercially de-risked MOA**

Two ongoing trials with a third trial expected to start next year to **support cemsidomide's potential to become a foundational MM treatment**

Multiple myeloma (MM)
CELMoDs® is a registered trademarks of BMS
C4 Therapeutics

IKZF1/3 are Transcription Factors That are Central Drivers of Multiple Myeloma Development and Progression

IMiDs® (Pomalyst (pomalidomide) capsules, Revlimid (lenalidomide) capsules), **CELMoDs®** (*Iberdomide*, *Mezigdomide*), and **cemsidomide** all degrade IKZF1/3 to drive anti-myeloma activit

Key Roles of IKZF1/3

Physiological Functions:

- **IKZF1/3** directly regulate the activity of **IRF4**, another transcription factor that regulates downstream immune cell differentiation

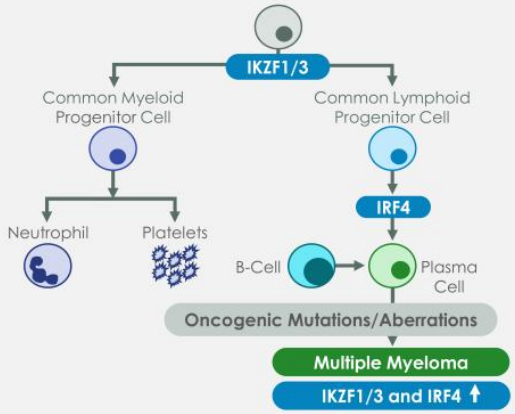
Oncogenic Functions:

- Multiple myeloma cells rely on **IKZF1/3** and **IRF4** for survival

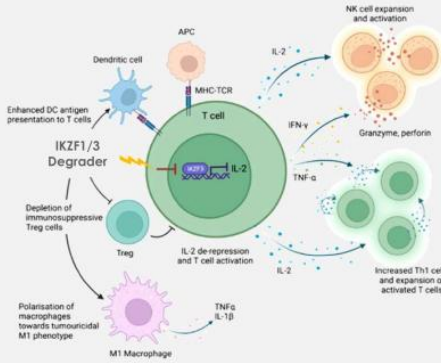
IKZF1/3 Degradation Leads to:

- Downregulation of **IRF4** promoting proliferation and myeloma cell death
- T-cell activation
- On-target neutropenia

Hematopoietic Stem Cell



T-cell Activation



Adapted from Chen and Gooding, 2022

Multiple myeloma (MM)
IMiDs® and CELMoDs® are registered trademarks of BMS

C4 Therapeutics

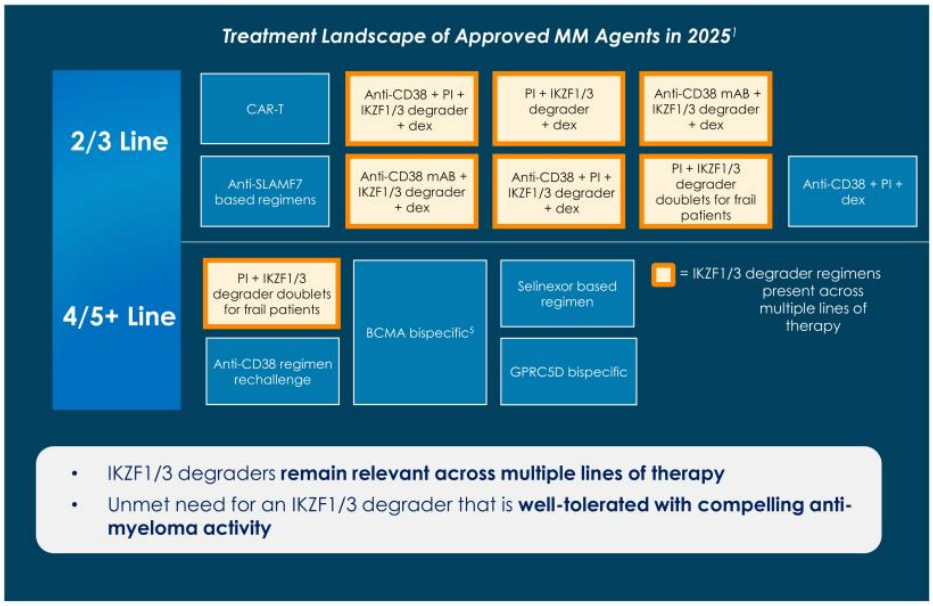
The MOA of IKZF1/3 Degraders Supports Their Role Across Lines of Treatment and Combinations

~11K
MM patient deaths expected in the U.S. in 2026[3]

~40%
MM patients do not **survive beyond five years**, despite recent treatment advances[4]

~$25.5B
Expected revenue for RRMM in U.S., Japan, EU4+UK by 2034[2]

~$59B
Total projected MM market in U.S., Japan, EU4+UK by 2034[2]



[1] NCCN guidelines, accessed in September 2025; [2] Datamonitor (accessed 5/1/2026) [3] American Cancer Society; [4] https://seer.cancer.gov/statfacts/html/mulmy.html (accessed June 2026). [5] Linovesiltamab is only approved in 5L. Multiple myeloma (MM); dexamethasone (dex)

First-generation IKZF1/3 Degraders (IMiDs®) Have Limitations Supporting the Need for Next-generation IKZF1/3 Degraders

First-generation IKZF1/3 degrader limitations:

- **High to moderate renal clearance decreasing tolerability**
  - ~50% of MM patients suffer from renal impairment[1]
- **Not as selectiveand results in off-target non hematology toxicities**[5]
  - Gastrointestinal (GI) and skin side effects are often observed[2,3]
- **Potency not optimized resulting in both modest on-target degradation, limiting anti-myeloma activity, and blockade of proliferation alone, increasing the risk of resistance mechanisms emerging**[4]

First-gen IKZF1/3 degraders' potency vs. Next-gen IKZF1/3 degraders' potency
(illustrative graphic)

Revlimid (lenalidomide) capsules · Pomalyst (pomalidomide) capsules

Next-gen IKZF1/3 Degraders:
(Iberdomide, Mezigdomide, Cemsidom

Least to Most Potent IKZF1/3 Degraders

[1]Rana 2020 Blood Advances.[2]. Tinsley S, Kurtin S, Ridgeway J Practical Management of Lenalidomide-Related Rash Clinical Lymphoma, Myeloma and Leukemia. 15, S64-S69; Dimopoulos, M., Leleu, X., Palumbo, A. *et al.*: Expert panel consensus statement on the optimal use of pomalidomide in relapsed and refractory multiple myeloma. *Leukemia* 28, 1573–1585 (2014). https://doi.org/10.1038/leu.2014.60; [4] CELMoDs May Represent Next Wave of Immunomodulation Approaches in Multiple Myeloma | OncLive [5] Developing next generation immunomodulatory drugs and their combinations in multiple myeloma - PMC
Multiple myeloma (MM); First-generation (First-gen); Next-gen (Next generation)
IMiDs® is a registered trademarks of BMS

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Phase 1 Trial of Cemsidomide + Dexamethasone Enrolled a Heavily Pre-treated Patient Population with Majority Receiving Prior CAR-T or T-cell Engager Therapy

Heavily Pre-treated Patient Population

Cemsidomide's patient population is representative of current multi-refractory patients

Characteristics	Safety Population (N=73)
Prior therapies, median (range)	7 (3-22)
Prior CAR-T therapy, n (%)	37 (51)
Prior T-cell engager therapy, n (%)	40 (55)
Prior CAR T or T-cell engager therapy, n (%)	55 (75)
Prior CAR T and T-cell engager therapy, n (%)	22 (30)
Prior BCMA therapy, n (%)	55 (75)
Triple-class exposed*, n (%)	73 (100)
Penta-drug exposed†, n (%)	59 (81)

Enrollment was completed in September 2025

*Defined as exposed to ≥1 immunomodulatory agent, ≥ 1 proteasome inhibitor, and 1 anti-CD38 monoclonal antibody; †Defined as exposed to ≥2 immunomodulatory agents, ≥ 2 proteasome inhibitors, and 1 anti-CD38 monoclonal antibody

C4 Therapeutics

Cemsidomide Phase 1 data cutoff as of 2/27/2026; Source: C4T data on file, Poster presentation at EHA 2026 (https://ir.c4therapeutics.com/static-files/00811021-bc0d-4e7f-bdb9-9a01e95ed6eb)

Cemsidomide + Dexamethasone Demonstrated a Well-tolerated Profile With Minimal Dose Reductions and Discontinuations

Hematologic and Infection TEAEs, n (%)	All Grades (N=73)	Grade 3 (N=73)	Grade 4 (N=73)	Grade 5 (N=73
Neutropenia	45 (62)	16 (22)	26 (36)	0
Infections	46 (63)	21 (29)	1 (1)	1 (1)
Pneumonia	13 (18)	11 (15)	0	0
URTI	13 (18)	2 (3)	0	0
Septic Shock	1 (1)	0	0	1 (1)
Sepsis	2 (3)	2 (3)	0	0
PML*	1 (1)	0	1 (1)	0
Anemia	28 (38)	17 (23)	1 (1)	0
Leukopenia	22 (30)	10 (14)	8 (11)	0
Thrombocytopenia	14 (19)	5 (7)	3 (4)	0
Lymphopenia	12 (16)	7 (10)	1 (1)	0
Febrile Neutropenia	4 (6)	3 (4)	1 (1)	0

Neutropenia was manageable with majority of events occurring in the first two cycles[3]

45% of patients received G-CSF across all doses

Limited grade 3/4 non-hematology side effects

Minimal dose reductions
- TEAEs leading to dose reductions: 5/73 (7%)[1]

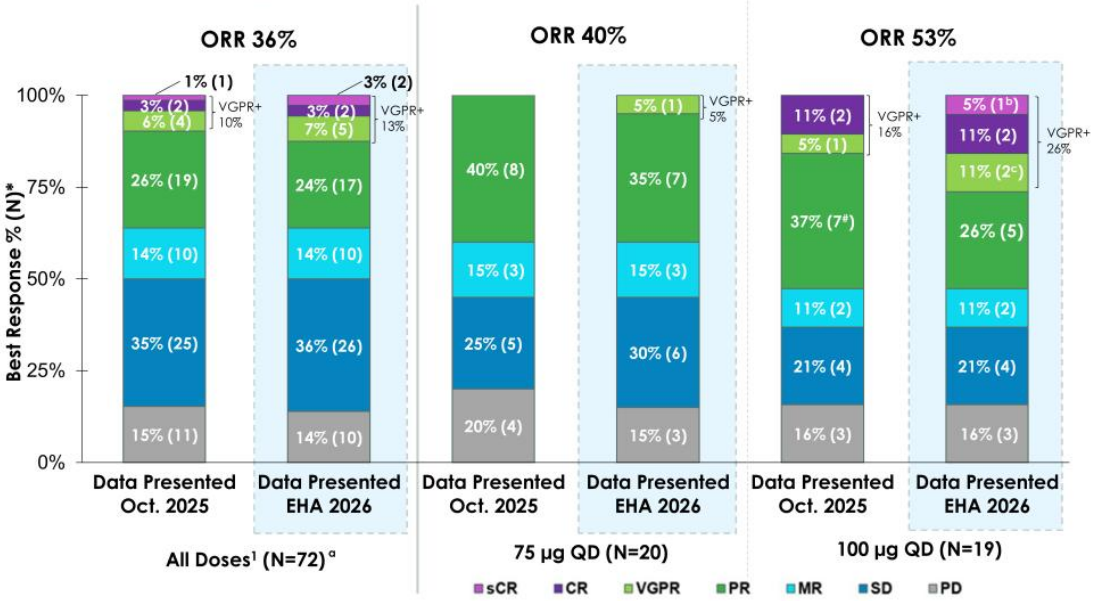
No discontinuations related to cemsidomide
- 3 TEAEs led to discontinuation, unrelated to cemsidomide[2]

- *Grade 4 PML considered possibly related but occurred in the setting of pre-existing chronic lymphopenia and prior exposure to immunosuppressive therapies, including therapies that have been associated PML. Patient had recurrent seizures in the setting of a brain lesion with a negative CSF for PML. After withdrawal of care due to recurrent seizures and ultimately death, autopsy report indicated a brain le consistent with PML diagnosis.
- 4 patients experienced grade 5 AEs (septic shock, subdural hematoma, T-Cell lymphoma and partial seizures), all deemed unrelated to cemsidomide

[1]Dose Reductions: A patient in the 75 µg cohort had grade 4 thrombocytopenia possibly related to cemsidomide resulting in dose reduction; A patient in the 100 µg cohort had grade 3 pneumonia; Another patient at 100 µg had grade 3 neutropenia, both AEs possibly related to cemsidomide resulting in dose reduction; a patient in the 100 µg cohort had a dose reductions after an AE of arthralgia, deemed possibly related to cemsidomide; a patient in the 100 µg cohort had two dose reductions after two events of pseudomonal bacteremia, deemed unrelated to cemsidomide. [2]3 patients discontinued due to a grade 5 AE of septic shock, grade 5 AE of T cell lymphoma, grade 5 AE of partial seizures, all deemed unrelated to cemsidomide [3] C4T data on file, presented at IMS September 2025
Treatment emergent adverse events (TEAEs)

Cemsidomide Phase 1 data cutoff as of 2/27/2026; Source: C4T data on file. Poster presentation at EHA 2026 (https://ir.c4therapeutics.com/static-files/00811021-bc0d-4e7f-bdb9-9a01e95ed6eb)

Cemsidomide + Dexamethasone Demonstrated Deep and Durable Responses Across the Highest Two Dose Levels With Some Responses Deepening Over Time



- At 75 µg: 1 patient who previously achieved a PR **deepened to a VGPR**
- At 100 µg: 1 patient who previously achieved a PR **deepened to a sCR**; 1 pati who previously achieved **deepened to a VGPR**
- At 100 µg: **Two patients** wh achieved a sCR and CR a **achieved MRD negativity**
- **mPFS across all doses:** 3.9 months (95% CI, 3.2 – 5.6)
- **mDOR across all doses:** 7.9 months (95% CI, 3.0 - NE)

*Investigator assessed response; [1] In the Phase 1 cemsidomide + dexamethasone trial evaluated doses of 50 µg MWF, 37.5 µg MWF, 62.5 µg QD, 75 µg QD, 100µg QD; [a]1 patient in the 62.5 µg cohort did not have a post-baseline assessment; #2 patients in th cohort had an unconfirmed PR in the October 2025 dataset ; [b]After the 2/27/26 data cutoff one patient went from VGPR to sCR; [c]After the 2/27/26 data cutoff one patient went from PR to VPGR
Clinical benefit rate (CBR); Dexamethasone (dex); Minimal response (MR); Minimal residual disease (MRD); Objective response rate (ORR); Progressive disease (PD); Partial response (PR) ; Once daily (QD); Relapsed/refractory multiple myeloma (RRMM); Stri complete response (sCR); Stable disease (SD); Very good partial response (VGPR); Confidence Interval (CI)

For EHA 2026 data: Cemsidomide Phase 1 data cutoff as of 2/27/2026;. Poster presentation at EHA 2026 (https://ir.c4therapeutics.com/static-files/00811021-bc0d-4e7f-bdb9-9a01e95ed6eb)
For Oct. 2025 data: C4T data on file as of 9/10/2025 (0a4b569a-ab8f-4cbd-9b99-cc6a9ff19d0b)

ORR was Consistent Across Key Subgroups in the Phase 1 Trial of Cemsidomide + Dexamethasone

ORR across key subgroups:

All Doses	Responders/ Patients	ORR % (95% CI)
Prior CAR-T or T-cell engager therapy	20/54	37.0% (24.3, 51.3)
Prior BCMA	18/54	33.3% (21.1, 47.5)
> 5 Prior Lines of Therapy	16/48	33.3% (20.4, 48.4)

At 100 µg (RP2D)	Responders/ Patients	ORR % (95% CI)
Prior CAR-T or T-cell engager therapy	9/17	52.9% (27.8, 77.0)
Prior BCMA	7/15	46.6 % (21.3, 73.4)
> 5 Prior Lines of Therapy	7/15	46.7% (21.3, 73.4)

Overall response rate (ORR); Recommended Phase 2 Dose (RP2D)

Cemsidomide Phase 1 data cutoff as of 2/27/2026; Source: C4T data on file. Poster presentation at EHA 2026 (https://ir.c4therapeutics.com/static-files/00811021-bc0d-4e7f-bdb9-9a01e95ed6eb)

Cemsidomide Has the Potential to Be a Foundational Treatment Across Multiple Lines of Multiple Myeloma

3 strategic paths to capture multi-billion dollar opportunities

Late-line Opportunity
Combination with dexamethasone

RATIONALE

- Only next-generation IKZF1/3 degrader with a label-enabling development strategy for the 4L+
- Unmet need for an all-oral treatment regimen that is both well-tolerated and efficacious for patients who have exhausted all options
- Near-term value

STATUS

✓ **Enrolling Phase 2 MOMENTUM Trial**
- Cemsidomide + dexamethasone

- Data from the Phase 1 trial of cemsidomide + dexamethasone demonstrated a potential best-in-class profile[5]

Novel Combination
Combination with BCMAxCD3 Bispecific

RATIONALE

- For use in earlier lines
- Goal is to establish cemsidomide as an IKZF1/3 degrader of choice for novel combinations
- Complementary MOA via T-cell activation with potential to drive potent anti-myeloma effect

STATUS

✓ **Enrolling Phase 1b Trial**
- Cemsidomide + dexamethasone + elranatamab[3]

- Data from MagnetisMM-30 trial[1] demonstrates proof-of-concept for combination with opportunity to improve depth of response

IMiD® Replacement Across Line
Combination with a PI or CD38 antibody

RATIONALE

- Opportunity to improve upon first-generation IKZF1/3 degraders
- Establish dose of cemsidomide for potential standard of care combination approaches

STATUS

○ **Initiation of Phase 1b Trial w/ Two Arms Expected in 1H 2027**
- Cemsidomide + dexamethasone + PI
- Cemsidomide + dexamethasone + CD38 antibody

- Upcoming data from the EXCALIBER RRMM trial[2] and SUCCESSOR-1 trial[4]

GOAL: Develop a potential best-in-class IKZF1/3 degrader to become partner of choice for MM treatment

[1]Clinical trial evaluating elranatamab in combination with iberdomide in RRMM; [2]EXCALIBER RRMM trial is a Phase 3 trial comparing iberdomide, daratumumab and dexamethasone versus daratumumab, bortezomib, and dexamethasone [3]- Pfizer suppl elranatamab (ELREXFIO®), a B-cell maturation antigen CD3 targeted bispecific antibody. to C4T for the Phase 1b trial. [4] SUCCESSOR-1 is a Phase 3 trial evaluating mezigdomide, bortezomib, dexamethasone versus pomalyst, bortezomib, dexamethasone IMiDs® are registered trademarks of BMS [5] Cemsidomide Phase 1 data cutoff as of 2/27/2026; Source: C4T data on file. Poster presentation at EHA 2026 (https://ir.c4therapeutics.com/static-files/00811021-bc0d-4e71-bdb9-9a01e95ed6eb)

Phase 2 MOMENTUM Trial of Cemsidomide + Dexamethasone in 4L+ MM Now Enrolling Patients

Enrollment Expected to Complete in Q1 2027

Phase 2 MOMENTUM

Cemsidomide + dex (single arm) 4L+

N = ~100

Dose: 100 µg QD

Potential for accelerated approval

2H 2027: Phase 2 initial ORR data

PHASE 2 MOMENTUM TRIAL DESIGN:

Endpoints:

ORR per IMWG response criteria assessed by independent review committee

- 20% increase over a background rate of 20%

RP2D: 100 µg

Schedule: QD 14/14

Dexamethasone (dex); Overall response rate (ORR); Fourth line (4L); Recommended Phase 2 dose (RP2D); Overall response (ORR); International Myeloma Working Group (IMWG); Once daily (QD)

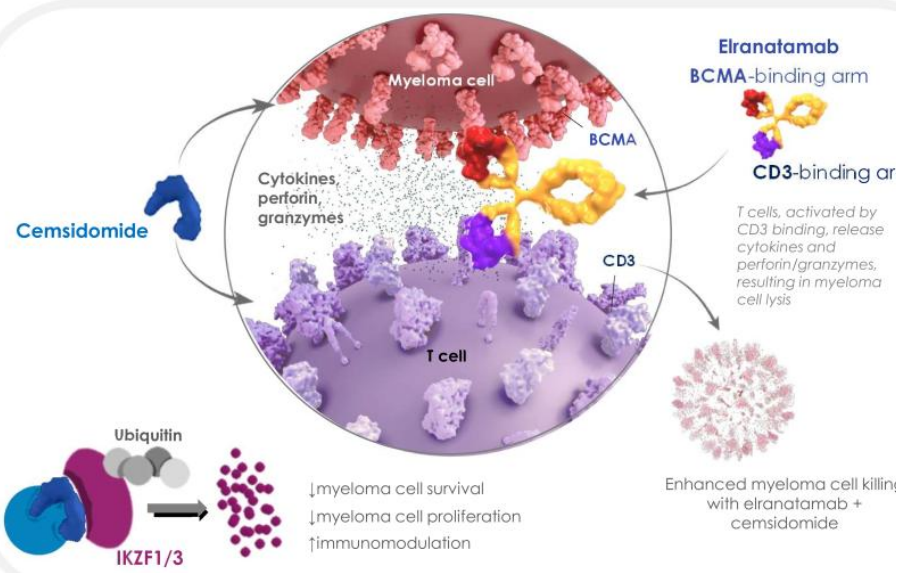
Based on Complementary Mechanisms of Action, Cemsidomide in Combination with Elranatamab Has Potential to Provide Additional Benefit to Patients

Elranatamab is a BCMAxCD3 Bispecific approved as a monotherapy for patients with RRMM who have received ≥1 IMiD®, ≥1 PI, and ≥1 anti-CD38 mAb[1-2]

Cemsidomide is an oral IKZF1/3 degrader, advancing through clinical development, with a potential best-in-class profile:

- Demonstrated t-cell activation across clinically relevant doses as a monotherapy and in combination w/ dexamethasone[3]

Elranatamab + cemsidomide + dexamethasone may provide additional benefit to patients with RRMM based on the complementary mechanisms of action



[1]Elrexfio (elranatamab-bcmm). Prescribing information. Pfizer Inc; 2025.; [2]Elrexfio (elranatamab-bcmm). Summary of product characteristics. Pfizer Europe MA EEIG; 2024; [3] C4T data on file: https://ir.c4therapeutics.com/static-files/39670c4f-0806-41b6-8813-ef7adcf04207 ; https://ir.c4therapeutics.com/static-files/ec59b02e-3074-484d-ad88-e81831bf37ed

B-cell maturation antigen (BCMA); Immunomodulatory drug (IMiD); Monoclonal antibody (mAb); Proteasome inhibitor (PI); Relapsed or refractory multiple myeloma (RRMM); Cereblon (CRBN)

IMiDs® are registered trademarks of BMS

Early IKZF1/3 Degrader + BiTE Data Provide Proof of Concept for Cemsidomide with Opportunity For Improvement

Currently CAR-Ts demonstrate higher ORR and ≥CR than BiTEs alone[1,2]

Early data from IKZF1/3 degrader + BiTE combo **support POC for similar anti-myeloma activity to CAR-Ts with better overall profile**, but **opportunity to improve depth of response**

- Combination is safe
- Early evidence of anti-myeloma activity

Opportunity to improve BiTE response rate including depth of response

CEMSIDOMIDE DEVELOPMENT RATIONALE IN 2L+ IN COMBO WITH A BITE

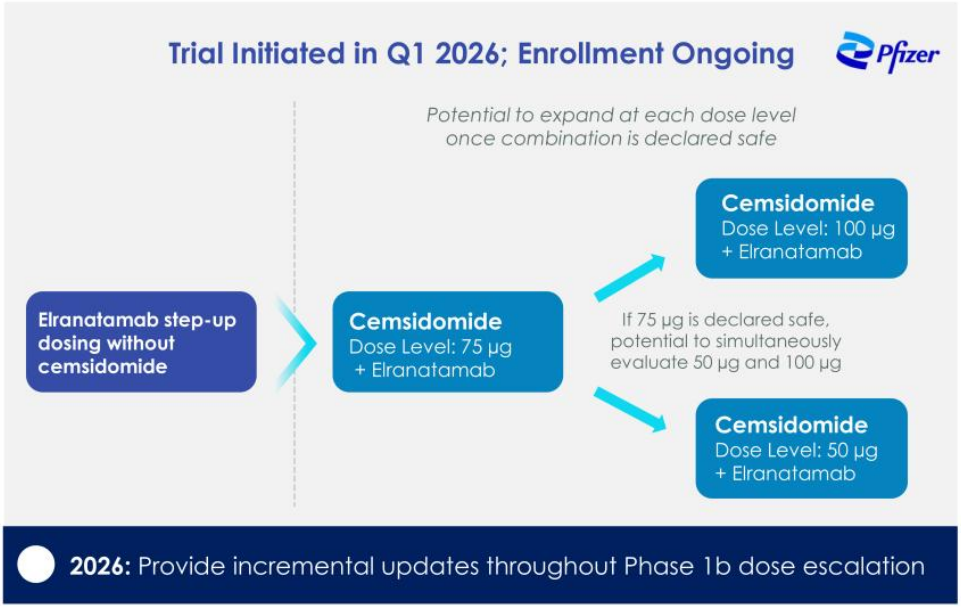
- Differentiated safety profile
- Compelling anti-myeloma activity across the highest 3 doses
- T-cell activation observed across all cemsidomide dose levels
- Phase 1b trial with elranatamab[4] will evaluate MRD negative responses

Cemsidomide is well-positioned to provide further differentiation to BiTE combination

Sources: [1]Packaging Insert for each product (carvykti – accessed 8/26/25) [2] Labels from tecvayli; elrexfio; lynozyfic - accessed 2/27/26 - the data is not a head-to-head trial; [3]2025 ASH ORR data at each dose level from Phase 1b MagnetismMM-30 trial evaluating iberdomide + elranatamab [4]Pfizer supplying elranatamab (ELREXFIO®), a B-cell maturation antigen CD3 targeted bispecific antibody, to C4T for the Phase 1b trial
BiTE: Bispecific T-cell engager; ORR: Overall response rate; CR: Complete response; Combo: Combination; MRD: Minimal residual disease

C4 Therapeutics

Phase 1b Trial is Evaluating Safety and Tolerability of Cemsidomide in Combination With Elranatamab, With Data From All Cohorts Expected in Mid-2027



PHASE 1b TRIAL DESIGN:

Primary Objectives:

Characterize the safety and tolerability of cemsidomide in combination with elranatamab

Dosing Regimen:

- Cemsidomide: QD 14/14
- Dexamethasone: QW through cycle 4
- Elranatamab[1]

Key Differentiators:

- Evaluated with dex, which ma help manage neutropenic complications
- Focused on evaluating MRD negativity rates to demonstrat depth of response

[1] Pfizer will supply elranatamab (ELREXFIO®), a B-cell maturation antigen CD3 targeted bispecific antibody, to C4T for its upcoming Phase 1b trial
Dexamethasone (dex): Once daily (QD); Once weekly (QW)

C4 Therapeutics

Discovery

Inflammation, Neuroinflammation, & Neurodegeneration (INN)

C4 Therapeutics

New Discovery Strategy Focused on Inflammation, Neuroinflammation & Neurodegeneration (INN) with First-in-Class Potential in Clinically Validated Pathways Uniquely Suited for TPD

Leveraging C4T's success

C4T HAS CONSISTENTLY DEVELOPED ORALLY BIOAVAILABLE HIGHLY CATALYTIC HETEROBIVALENT DEGRADERS THAT…

- **Penetrate the blood brain barrier** to **achieve high central nervous system exposures** and **compelling efficacy** in central nervous system models
- Control target protein levels through **finely-tuned degrader kinetics**

Maximizing value through target selection

TARGET-TO-DISEASE LINK:

- Selecting targets that modulate **clinically validated pathways** in inflammation, neuroinflammation, and neurodegeneration **(INN)** to enhance efficacy
- **Focusing on early clinical validation** with opportunity to **grow value** through indication expansion

STRONG DEGRADER RATIONALE:

- **Strong competitive positioning**
- **Clear** and **compelling advantage** for a degrader **over an inhibitor**

EXPANDED CAPABILITIES:

- **Extended capabilities** to identify molecular glue degraders for targets with and without G- and RT-loops by **utilizing DNA-encoded library (DEL) technology**

Deliver degraders with first-in-class potential that are CNS penetrant

Focused on Inflammation, Neuroinflammation & Neurodegeneration (INN) to Address High Unmet Needs in a Large Patient Population with a Clear TPD Advantage

Degraders have the potential to **outperform inhibitors** in **efficacy** and **safety** in CNS diseases[1]

Fast path to clinical proof-of-concept, including **early validation** based on PD markers in healthy volunteers

Normalize elevated protein levels without the need for complete elimination of the target

Large market opportunities with high **unmet medical needs**

Deploying TPD where the MOA is uniquely positioned to have an advantage over inhibitors to help be patients in a large market

Central nervous system (CNS), Pharmacodynamic (PD); Targeted Protein Degradation (TPD); Mechanism of action (MOA)
[1]Based on preclinical evidence and working hypothesis

Potential for Degraders To Be the Optimal Therapeutic Modality for CNS Diseases Over Inhibitors

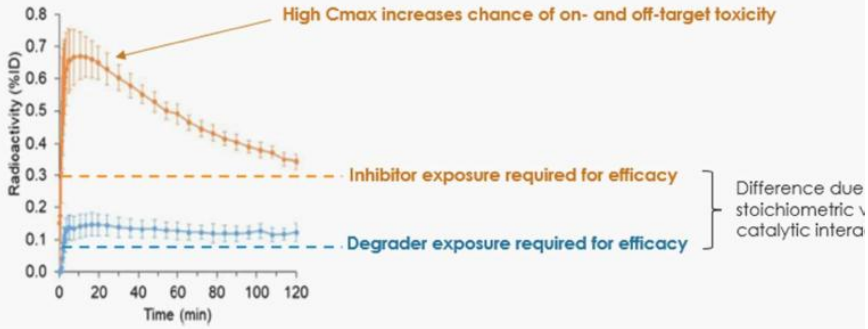
Lower exposure levels for highly catalytic degraders are required for efficacy versus inhibitors to **achieve efficacious** results in CNS diseases

Pharmacokinetics of inhibitors is associated with high Cmax driving toxicities vs. **degraders have consistent and sustained levels resulting in lower toxicity issues**

Theoretical Inhibitor and Degrader Brain PK Curves for Molecules With Similar Efficacy*

(Illustrative graphic)

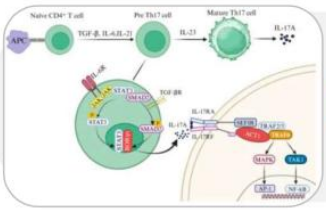
**For target proteins with a long resynthesis rate*



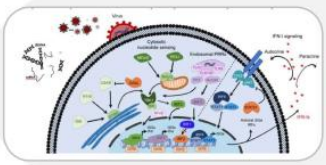
Sources: Drug Discov Today. 2019 May;24(5):1067-1073. doi: 10.1016/j.drudis.2019.01.015; Pharm Res. 2022 Jul;39(7):1321-1341. doi: 10.1007/s11095-022-03246-6

Central nervous system (CNS); Pharmacokinetic (PK)

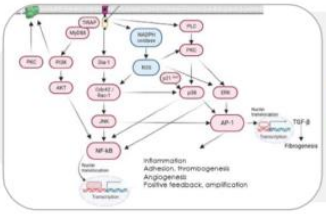
Pursuing Targets in Validated Pathways With Application to a Broad Set of Indications



IL-23/IL-17 Pathway



Type 1 IFN Pathway



MAPK, PI3K/AKT, NF-kB Pathways

POTENTIAL INDICATIONS

- Alzheimer's Disease*
- Psoriasis
- Multiple Sclerosis*
- Down Syndrome*
- Parkinson's Disease*
- Rheumatoid Arthritis
- Multiple Myeloma
- Lupus Nephritis
- Systemic Lupus Erythematosus
- Inflammatory Bowel Disease
- Asthma
- Autosomal Dominant Polycystic Kidney Disease
- Chronic Kidney Disease
- Metabolic Dysfunction Associated Steatohepatiti
- Idiopathic Pulmonary Fibrosis

*Highlights indications that are central nervous system diseases

Image [1] Zheng M-Y, Luo L-Z Int. J. Mol. Sci. 2025; Image [2] Lukhele S, et al. Semin Immunol 2019; Image [3] Liu T, et al. Sig. Transduct. Target. Ther. 2017

C4T is Focused on Advancing Potential Best-in-Class And First-in-Class Degraders Across Clinical Oncology Portfolio and INN Discovery Strategy

2026

Advance potential **best-in-class** and **first-in-class** degraders

- **Enroll 2 clinical trials** with **cemsidomide** to address 2L+ and 4L+ opportunities in MM
- **Establish combinability profile** with cemsidomide + elranatamab[1]
- **Optimize indication selection** for multiple targets across discovery portfolio

2027

Position for **regulatory success** and **pipeline build**

- **Complete enrollment** for Phase 2 MOMENTUM trial
- **Initiate additional Phase 1b Trial**
- **Present two cemsidomide data readouts**:
  - Initial ORR data from Phase 2 MOMENTUM trial
  - Phase 1b data w/ elranatamab[1] to support advancement to Phase 3 trial
- **Start up activities** for **Phase 3 cemsidomide + BCMAxCD3 Bispecific**
- **Advance internal discovery pipeline** to enable INDs

2028

Unlock value across portfolio

- **Initiate** and **enroll Phase 3 trial** of cemsidomide + BCMAxCD3 Bispecific
- **Present efficacy** and **safety data** from the Phase 2 MOMENTUM trial
- **Potentially submit NDA** for cemsidomide
- **Deliver 3 potential INDs** from discovery pipeline in INN indications

[1] Pfizer supplying elranatamab (ELREXFIO®), a B-cell maturation antigen CD3 targeted bispecific antibody, to C4T for the Phase 1b trial
Dexamethasone (dex); Inflammation, Investigational new drug (IND); New Drug Application (NDA); Overall response rate (ORR); Inflammation, Neuroinflammation, Neurodegeneration (INN); Accelerated approval (AA); Multiple myeloma; Degrader antib conjugates (DACs)

Updated Results of a Phase 1 First-in-Human Study of Cemsidomide, a Novel MonoDAC® Degrader, wi Dexamethasone (DEX) in Patients (pts) with Relapsed/Refractory Multiple Myeloma (RRMM)

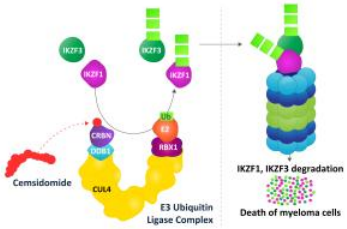
Sagar Lonial, MD[1], Andrew J. Yee, MD[2], Paul G. Richardson, MD[3], Sikander Ailawadhi, MD[4], Saurabh Chhabra, M.B.B.S[5], Wilson Gonsalves, MD[6], Jesus G. Berdeja, MD[7], Shambavi Richard, MD[8], Jeffrey V. Matous, MD[9], Urvi A. Shah, MD[10], Mark A. Schroeder, MD[11], Sumedha Javalikar, MPH[12], Amro Ali, PharmD[12], Leah Leahy, BS[12], Uma Chandrasekaran, PhD[12], Riadh Lobbardi, PhD[12], Rong Chu, PhD[12], Eunju Hurh, PhD[13], Leonard Reyno, MD[12], and Binod Dhakal, MD[13]

[1]Winship Cancer Institute, Emory University, Atlanta, GA [2]Massachusetts General Hospital Cancer Center, Boston, MA; [3]Dana-Farber/Boston Children's Cancer and Blood Disorders Ctr, Boston, MA; [4]Division of Hematology, Mayo Clinic, Jacksonville, FL; [5]Mayo Clinic Arizona, Phoenix, AZ; [6]Mayo Clinic Rochester, Saint Oncology, Nashville, TN; [8]Department of Medicine, Hematology and Medical Oncology, Icahn School of Medicine at Mount Sinai, New York, NY; [9]Sarah Cannon Research Institute, Colorado Blood Cancer Institute, Denver, CO; [10]Memorial Sloan Kettering Cancer Center, New York, NY; [11]Washington University School of M [12]C4 Therapeutics, Inc., Watertown, MA; [13]Medical College of Wisconsin, Milwaukee, Wisconsin

Introduction

- Cemsidomide is a novel, highly potent, cereblon-based, IKZF1/3 MonoDAC® degrader, having a similar mechanism of action as BMS's IMiD® or CELMoD® degraders for MM
- Cemsidomide displays catalytic activity enabling rapid and deep target degradation with high binding affinity to overcome resistance due to low cereblon levels
- Cemsidomide binds to cereblon to facilitate the recruitment and ubiquitination of IKZF1/3 leading to the proteasomal degradation of both proteins (Figure 1)
- IKZF1/3 degradation induces multiple myeloma cell death, activation of fully differentiated T-cells which prevents T-cell exhaustion and promotes secretion of key immune stimulating cytokines[1]

Figure 1: Mechanism of Action of Cemsidomide[2]



IKZF1/3 Degradation Induces:

- Multiple Myeloma cell death
- Immune stimulation
  - Activates fully differentiated T-cells, preventing T-cell exhaustion
  - Promotes secretion of key immune stimulating cytokines (e.g., IL-2)
- On-target neutropenia
  - Disrupts hematopoietic stem cell differentiation

Methods

CFT7455-1101 Study Design[3]

- Open-label, multicenter, phase 1/2 clinical trial with dose escalation and expansion phases (NCT04756726)*
- Dose escalation phase, beginning with a starting oral dose of 50 µg MWF 14 days on/14 days off, following a Bayesian logistic regression model until determination of the MTD and/or RP2D
  - Escalation cohorts enrolled 3-6 patients, once dose was declared safe by SRC, additional patients were eligible to enroll at the dose deemed safe
    - G-CSF and transfusions were not allowed in cycle 1 for dose escalation subjects
    - Once a dose was declared safe, additional patients at each dose level were allowed G-CSF use at any timepoint

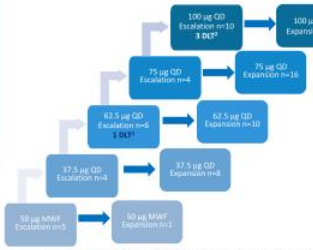
KEY INCLUSION CRITERIA

- Adults with MM, R/R to at least 3 prior lines of therapy 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
- Creatinine clearance ≥40 mL/min
- ECOG ≤2

Phase 1 Study Endpoints

- Primary: assess safety, tolerability and define the RP2D/MTD
- Secondary: assess PK, PD, and preliminary anti-tumor activity

Figure 2: Phase 1 Dose Escalation Cemsidomide 14/14 + Dex*



*CFT7455 administered as 14 days on/14 days off in a 28-day cycle; Dex was dosed on days 1, 8, 15, and 22 at doses of 40 mg orally for patients ≤75 years old and 20 mg orally for patients >75 years old; †DLT in the 62.5 µg QD was due to grade 4 neutropenia lasting >7 days; ‡Three patients in the 100 µg QD escalation had 5 DLT events (G4 neutropenia, G3 pneumonia in 2 subjects, G3 ALT increase, G3 febrile neutropenia)

European Society of Hematology 2026

Results

Patients

- At the data cutoff date (February 27, 2026), 73 patients had received cemsidomide + dexamethasone
- Baseline characteristics are shown in Table 1, and prior therapies are shown in Table 2
  - Patients were heavily pre-treated, having received a median of 7 prior lines of therapy (range 3-22)
  - 75% of patients had received a CAR-T or TCE and 75% had received a prior BCMA therapy

Table 1: Baseline Characteristics

Characteristics	Safety Population (N=73)
Age, median (range)	67 (38-90 years)
Male, n (%)	43 (59)
Time since initial diagnosis, median (range)	7 (2-22 years)
ECOG performance status, n (%)	
0	18 (25)
1	52 (71)
2	3 (4)
Asian	1 (1)
Black or African American, n (%)	15 (21)
White, n (%)	50 (69)
Other, n (%)	7 (10)
Revised ISS at screening, n (%)	
Stage 1	24 (33)
Stage 2	31 (43)
Stage 3	9 (12)
Missing	9 (12)
Presence of EMD, n (%)	23 (32)

Table 2: Prior Therapies

Characteristics	Safety Population (N=73)
Prior therapies, median (range)	7 (3-22)
3L, n (%)	3 (4)
4L, n (%)	13 (15)
≥ 5L, n (%)	59 (81)
Prior stem cell transplant, n (%)	45 (62)
Prior lenalidomide, n (%)	73 (100)
Prior pomalidomide, n (%)	72 (99)
Prior anti-CD38 mAb, n (%)	73 (100)
Prior CAR-T therapy, n (%)	37 (51)
Prior T-cell engager therapy, n (%)	40 (55)
Prior CAR T or T-cell-engager therapy, n (%)	55 (75)
Prior CAR T and T-cell engager therapy, n (%)	22 (30)
Prior BCMA therapy, n (%)	55 (75)
Prior GPRC5D therapy, n (%)	35 (48)
Triple-class exposed*, n (%)	73 (100)
Penta-drug exposed†, n (%)	59 (81)

*Defined as exposed to ≥1 immunomodulatory agent, ≥ 1 proteasome inhibitor, and 1 anti-CD38 monoclonal antibody; †Defined as exposed to ≥2 immunomodulatory agents, ≥ 2 proteasome inhibitors, and 1 anti-CD38 monoclonal antibody

Table 3: Treatment Disposition

Patient Disposition, n (%)	Safety Population (N=73)
Ongoing	7 (10)
Discontinued	66 (90)
Progressive disease	51 (70)
Withdrawal of consent	8 (11)
Adverse event	2 (3)*
Death	2 (3)*
Physician Decision	2 (3)
Other	1 (1)†

*2 patients discontinued due to an AE of septic shock and an AE of partial seizures (unrelated to cemsidomide)
†Death in a 62.5 µg cohort patient was due to subdural hematoma (related to a fall), unrelated to cemsidomide; death in a 100 µg cohort patient was due to T cell lymphoma (unrelated to cemsidomide); ‡A patient in the 50 µg MWF cohort was transferred to hospice, did not meet IMWG definition of progressive disease

- Treatment disposition is shown on Table 3 with treatment ongoing for 7 patients (10%)
- The primary reason for discontinuation was progressive disease (51/66)
- Median time on treatment was 3.4 months

Safety

Table 4: Overall Treatment Emergent Adverse Events

Adverse Events, n (%)	50 µg MWF (N=4)	37.5 µg QD (N=12)	62.5 µg QD (N=16)	75 µg QD (N=20)	100 µg QD (N=19)	Total (N=73)
TEAEs	6 (100)	12 (100)	16 (100)	20 (100)	19 (100)	73 (100)
TEAEs possibly related to cemsidomide	3 (50)	11 (92)	12 (75)	14 (70)	16 (84)	56 (77)
TESAEs	3 (50)	6 (50)	7 (44)	7 (35)	9 (47)	32 (44)
TESAEs possibly related to cemsidomide	0	4 (33)	3 (19)	5 (25)	4 (21)	16 (22)
Any grade ≥3 TEAEs	5 (83)	8 (67)	12 (75)	18 (90)	16 (84)	59 (81)
Any grade ≥3 TEAEs possibly related to cemsidomide	3 (50)	8 (67)	9 (56)	12 (60)	13 (68)	45 (62)
TEAEs leading to discontinuation	0	0	0	1 (5)	2 (11)	3 (4)*
TEAEs leading to reduction	0	0	0	1 (5)†	4 (21)‡	5 (7)

*3 patients discontinued due to a grade 5 AE of septic shock, grade 5 AE of T cell lymphoma, grade 5 AE of partial seizures, all deemed unrelated to cemsidomide
†A patient in the 75 µg cohort had grade 4 thrombocytopenia possibly related to cemsidomide resulting in dose reduction. ‡A patient in the 100 µg cohort had grade 3 pneumonia and another patient at 100 µg had grade 3 neutropenia, both AEs possibly related to cemsidomide resulting in dose reduction, a patient in the 100 µg cohort had a dose reductions after an AE of arthralgia, deemed possibly related to cemsidomide. a patient in the 100 µg cohort had two dose reductions after two events of pseudomonal bacteremia, deemed unrelated to cemsidomide.

- 4 DLTs: 1 patient at 62.5 µg had grade 4 neutropenia >7 days; 3 patients at 100 µg had 5 DLT events (grade 4 neutropenia >7 days, grade 3 ALT increase, grade 3 febrile neutropenia, grade 3 pneumonia in 2 subjects)
- No patient had a related TEAE leading to cemsidomide discontinuation

Table 5: Treatment Emergent Adverse Events by Grade

Hematologic and Infection TEAEs, n (%)	All Grades (N=73)	Grade 3 (N=73)	Grade 4 (N=73)	Grade 5 (N=73)
Neutropenia	45 (62)	16 (22)	26 (36)	0
Infections	46 (63)	21 (29)	1 (1)	1 (1)
Pneumonia	13 (18)	11 (15)	0	0
LRTI	13 (18)	2 (3)	0	0
Septic Shock	1 (1)	0	0	1 (1)
Sepsis	2 (3)	2 (3)	0	0
PML*	1 (1)	0	1 (1)	0
Anemia	28 (38)	17 (23)	1 (1)	0
Leukopenia	22 (30)	10 (14)	8 (11)	0
Thrombocytopenia	14 (19)	5 (7)	3 (4)	0
Lymphopenia	12 (16)	7 (10)	1 (1)	0
Febrile Neutropenia	4 (6)	3 (4)	1 (1)	0

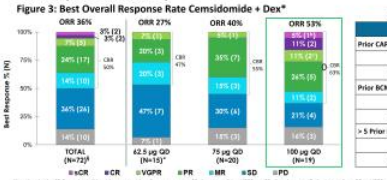
TEAEs (>20%) and Events of Interest, n (%)	All Grades (N=73)	Grade 3 (N=73)	Grade 4 (N=73)	Grade 5 (N=73)
Fatigue	29 (40)	0	0	0
Diarrhea	26 (36)	1 (1)	0	0
Cough	21 (29)	1 (1)	0	0
Insomnia	20 (27)	1 (1)	0	0
Constipation	19 (26)	0	0	0
Pyrexia	19 (26)	0	0	0
Nausea	18 (25)	0	0	0
Arthralgia	18 (25)	0	0	0
Dyspnea	17 (23)	2 (3)	0	0
Hypertension	17 (23)	5 (7)	1 (1)	0

*Grade 4 PML considered possibly related but occurred in the setting of pre-existing chronic lymphopenia and prior exposure to immunosuppressive therapies, including therapies that have been associated with PML. Patient had recurrent seizures in the setting of a brain lesion with a negative CSF for PML. After withdrawal of care due to recurrent seizures and ultimately death, autopsy report indicated a brain lesion consistent with PML diagnosis.

- 4 patients experienced grade 5 AEs (septic shock, subdural hematoma, T-Cell lymphoma and partial seizures), all deemed unrelated to cemsidomide
- G-CSF support was not allowed during cycle 1 for patients in dose escalation cohorts
- 42/73 (58%) of patients experienced grade 3/4 neutropenia, an anticipated on-target effect of IKZF1/3 degradation
  - Neutropenia was manageable with treatment interruptions and G-CSF use when permitted
  - Across all doses, 45% (33/73) of patients received G-CSF

Anti-Myeloma Activity

Figure 3: Best Overall Response Rate Cemsidomide + Dex*

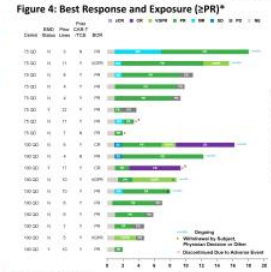


*1 patient in the 62.5 µg cohort did not have a post-baseline assessment; †Patient went from VGPR to sCR after data cut; ‡Patient went from PR to VGPR after data cut.

Table 6 : (All Dose Lev

Re
Prior CAR-T or TCE
All doses
100 µg (RP2D)
Prior BCMA
All doses
100 µg (RP2D)
> 5 Prior Lines of Therapy
All doses
100 µg (RP2D)

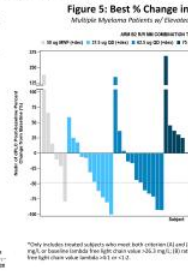
Figure 4: Best Response and Exposure (≥PR)*



*Investigator assessed response

Figure 5: Best % Change in

Multiple Myeloma Patients w/ Elevate



*Only includes treated subjects who meet both criterion (A) and (ng/L or baseline lambda free light chain value >26.3 mg/L; (B) no free light chain value lambda <9.1 or >1.2.

*Investigator assessed response
†Patient at 75µg discontinued due to grade 5 AE of septic shock, deemed unrelated to cemsidomide; ‡Patient at 100 µg discontinued due to grade 5 recurrent seizures, deemed u VGPR to sCR after data cut; §Patient went from PR to VGPR after data cut.

- ORR at the highest dose level of cemsidomide (100 µg) was 53% with a clinical benefit rate of 63% (Fig
  - MRD negativity achieved in 2 patients who achieved a sCR and CR at the highest dose level of cems
- ORR is consistent across subgroups irrespective of prior therapies (Table 6)
- Median PFS across all dose levels was 3.9 months (95% CI, 3.2–5.6)
- Median DOR across all dose levels was 7.9 months (95% CI, 3.0–NE)
- Figure 5 represents the greatest dFLC reduction from baseline
  - Cemsidomide + Dex induced a ≥ 50% decrease in dFLC in 50% (32/64) of patients
  - Cemsidomide demonstrated anti-myeloma activity across a broad range of doses

Conclusions

- Cemsidomide 14/14 plus Dex demonstrated durable anti-myeloma activity at increasing pretreated patient population
  - A 53% ORR was observed at the highest dose of 100 µg QD, with a 36% ORR observed acros
  - 100 µg 14/14 QD was established as the RP2D and MTD
  - Responses were durable and continued to deepen over time at the 75 µg and 100 µg dose l
  - **ORR was consistent across subgroups, with patients receiving prior CAR-T or TCE having a**
- Cemsidomide 14/14 plus Dex was well tolerated across dose levels
  - TEAEs were manageable with minimal treatment discontinuations or reductions
- Cemsidomide data strongly support further development across multiple lines of treatm with other anti-myeloma agents, including PIs, CD-38 mAB, ADCs, and TCEs
- Based on these results, cemsidomide 14/14 plus Dex is currently being assessed in the p study in the 4L+ patient population and in a phase 1b study in combination with elranata

Abbreviations

References

Acknowledgmen

C4 Therapeutics Presents Phase 1 Data at European Hematology Association (EHA) 2026 Congress Highlighting Cemsidomide as a Potential Best-in-Class IKZF1/3 Degrader for Multiple Myeloma in Heavily Pretreated Relapsed/Refractory Population

At the 100 µg Recommended Phase 2 Dose (RP2D), Cemsidomide Demonstrated a 53% Overall Response Rate, Including Complete Responses and Minimal Residual Disease (MRD) Negative Status

Cemsidomide Was Well Tolerated With Minimal Discontinuations and Dose Reductions Related to Safety or Tolerability

Data Further Support Development Strategy Across Lines of Therapy and in Combination With Approved Therapies, Positioning Cemsidomide as a Potential Foundational Therapy for Relapsed/Refractory Multiple Myeloma

WATERTOWN, Mass., June 11, 2026 (GLOBE NEWSWIRE) -- C4 Therapeutics, Inc. (C4T) (Nasdaq: CCCC), a clinical-stage biopharmaceutical company dedicated to advancing targeted protein degradation (TPD) science, will present further analysis from its fully enrolled Phase 1 trial of cemsidomide, a next-generation oral IKZF1/3 degrader, in combination with dexamethasone for the treatment of relapsed/refractory multiple myeloma (RRMM) in a poster presentation at the European Hematology Association (EHA) 2026 Congress on Friday, June 12, 2026 at 6:45 pm CEST / 12:45 pm ET.

The analysis is consistent with previous data disclosed from the Phase 1 clinical trial and highlights cemsidomide's anti-myeloma activity and differentiated safety profile, further supporting its development as a potential best-in-class IKF1/3 degrader. The poster will be presented by Sagar Lonial, M.D., FACP, FASCO, chief medical officer at the Winship Cancer Institute at Emory University, and an investigator in the cemsidomide clinical trials.

“Despite advances in multiple myeloma therapies, IKZF1/3 degradation remains a foundational treatment strategy across lines of therapy because it is the only approach that addresses the underlying biology of the disease and has the built-in ability to stimulate the immune function, becoming a natural partner for immune therapies. Next-generation IKZF1/3 degraders are expected to help advance treatment regimens for this persistent disease, given data demonstrating their efficacy and tolerability,” said Dr. Lonial. “The clinical activity and safety profile of cemsidomide are highly encouraging for patients with relapsed/refractory multiple myeloma as they continue to seek disease-altering treatment options. The data from the ongoing Phase 1 study support the continued development of cemsidomide for patients with relapsed/refractory multiple myeloma who may benefit from IKZF1/3 degradation.”

“The totality of cemsidomide data, particularly data showing that patients have experienced a deepening response over time, continue to demonstrate its potential to deliver a tolerable therapy that can provide sustained benefit for patients who have progressed through other treatment options,” said Len Reyno, M.D., chief medical officer of C4 Therapeutics. “We remain focused on advancing our clinical development strategy to capitalize upon cemsidomide’s differentiated profile in hopes patients at various stages of their treatment journey may be able to benefit from this important investigational therapeutic regimen.”

The poster presentation includes data on 73 patients with a data cutoff of February 27, 2026. Patients were heavily pretreated, receiving a median of seven prior lines of therapy. Fifty-five patients (75%) received prior BCMA therapy, and 55 patients (75%) received prior CAR-T or T-cell engager therapy (TCE).

At the RP2D and maximum tolerated dose (100 µg,) cemsidomide achieved a 53% overall response rate (ORR). At the 75 µg dose level, cemsidomide achieved a 40% ORR. Across all doses evaluated, cemsidomide achieved a 36% ORR.

Key new data include:

- Responses deepened over time across the cemsidomide 75 µg and 100 µg dose levels:
  - At 75 µg, one patient whose best response was previously a partial response (PR) deepened to a very good partial response (VGPR).
  - At 100 µg, several patients achieved a deeper response:
    - One patient whose best response was previously a PR deepened to a stringent complete response (sCR)
    - One patient whose best response was previously a PR deepened to a VGPR
  - Minimal residual disease (MRD) negativity was achieved in two patients who achieved a sCR and complete response (CR) at 100 µg.
- ORR was consistent across key subgroups:

	ORR % (95% confidence interval (CI))
All Doses	
Prior CAR-T or TCE	37% (24, 51)
Prior BCMA	33% (21, 48)
Prior Lines of Therapy > 5 Lines	33% (20, 48)
100 µg (RP2D)	
Prior CAR-T or TCE	53% (28, 77)
Prior BCMA	47% (21, 73)
Prior Lines of Therapy > 5 Lines	47% (21, 73)

- Durable responses were observed across all dose levels:
  - Patients experienced a median duration of response of 7.9 months (95% CI, 3.0 – non-evaluable).
  - Seven patients remain on treatment currently.

Cemsidomide in combination with dexamethasone was generally well tolerated. Incidences of on-target neutropenia remained manageable; 42 patients (58%) experienced Grade 3/4 neutropenia. All treatment emergent adverse events were manageable with no discontinuations deemed related to cemsidomide and minimal dose reductions (five patients; 7%).

UPCOMING INVESTOR EVENTS

- **June 18, 2026 at 9 am ET:** C4T will host an educational webinar with Nisha Joseph, M.D., associate professor at the Winship Cancer Institute at Emory University and investigator in the cemsidomide clinical trials to discuss the evolving multiple myeloma landscape, the role of IKZF1/3 degradation in treating multiple myeloma, and cemsidomide's profile.

About Cemsidomide

Cemsidomide is an investigational, next-generation orally bioavailable MonoDAC® degrader (molecular glue) of IKZF1/3, transcription factors foundational to multiple myeloma biology. Data from the fully enrolled Phase 1 trial show cemsidomide's differentiated safety and tolerability profile and potentially class-leading anti-myeloma activity that support the potential for durable outcomes.

About Multiple Myeloma

Multiple myeloma is a blood cancer that affects plasma cells in the bone marrow. It is the second most common blood cancer, with approximately 36,000 people in the United States diagnosed each year. Multiple myeloma is characterized by cycles of remission and relapse, which leads to patients needing multiple lines of therapy to manage this persistent disease. More than 175,000 patients in the United States are estimated to be living with or in remission from myeloma. However, despite treatment advances, approximately 40% of patients do not survive beyond five years.

About IKZF1/3 Degradation

Targeted degradation of IKZF1 (Ikaros) and IKF3 (Aiolos) is a foundational therapeutic strategy to treat multiple myeloma, a blood cancer affecting plasma cells. IKZF1/3 degradation leads to downregulation of IRF4, which promotes myeloma cell death. IKZF1/3 degradation also activates T-cells, which contributes to broader anti-myeloma response. For decades, IKZF1/3 degradation has been used in approved therapies for multiple myeloma. Next-generation IKZF1/3 degraders are being developed to leverage advances in targeted protein degradation research while continuing to address the biology foundational to multiple myeloma.

About the MOMENTUM Trial

MOMENTUM (Multi-center trial Of cemsidoMidE iN relapsed/refracTory mUltiple Myeloma) is a Phase 2, open-label, single-arm study to evaluate the efficacy, safety, pharmacokinetics and pharmacodynamics of cemsidomide in combination with dexamethasone in patients with relapsed/refractory multiple myeloma. Data from the Phase 1 trial identified 100 µg as the recommended Phase 2 dose. The primary endpoint is overall response rate per International Myeloma Working Group response criteria, as assessed by an independent review committee. Approximately 100 patients who have received at least three prior anti-myeloma regimens that must have included an IKZF1/3 degrader, a proteasome inhibitor, an anti-CD38 antibody, and a

T-cell engager or CAR-T therapy will be enrolled in the trial. More information is available at clinicaltrials.gov (NCT07284758).

About Cemsidomide in Combination With Elranatamab (ELREXFIO®)
The Phase 1b trial is designed to evaluate the safety, tolerability and preliminary efficacy of cemsidomide and dexamethasone in combination with elranatamab, an FDA-approved B-cell maturation antigen CD3 targeted bispecific antibody. Data generated from the cemsidomide Phase 1 trial in relapsed/refractory multiple myeloma demonstrate robust T-cell activation and cytokine expression across multiple doses. By activating immune T-cells, cemsidomide, when combined with a BCMAxCD3 bispecific such as elranatamab, may amplify the anti-myeloma immune response and lead to deeper and more durable responses. The study will evaluate different cemsidomide dose levels (beginning with 75 µg, with the opportunity to simultaneously explore 50 µg and 100 µg) in patients who have received one to four prior lines of therapy, which must have consisted of at least one IKZF1/3 degrader. Exclusion criteria for patients include those who have received prior treatment with a BCMA-directed T-cell engager or BCMA-directed CAR-T therapy. More information is available at clinicaltrials.gov (NCT07280013).

About C4 Therapeutics
C4 Therapeutics (C4T) (Nasdaq: CCCC) is a clinical-stage biopharmaceutical company dedicated to delivering on the promise of targeted protein degradation science to create a new generation of medicines that transforms patients' lives. C4T is progressing targeted oncology programs through clinical studies and leveraging its TORPEDO® platform to efficiently design and optimize small-molecule medicines to address difficult-to-treat diseases. C4T's degrader medicines are designed to harness the body's natural protein recycling system to rapidly degrade disease-causing proteins, offering the potential to overcome drug resistance, drug undruggable targets and improve patient outcomes. For more information, please visit www.c4therapeutics.com.

Forward Looking Statements
This press release contains "forward-looking statements" of C4 Therapeutics, Inc., within the meaning of the Private Securities Litigation Reform Act of 1995. These forward-looking statements may include, but may not be limited to, express or implied statements regarding our ability to develop potential therapies for patients; the safety, tolerability, design and potential efficacy of our therapeutic approaches and product candidates; the predictive capability of our TORPEDO® platform in the development of novel, selective, orally bioavailable BiDAC™ and MonoDAC® degraders; the potential initiation, timing, design, results and advancement of our preclinical studies and clinical trials, including the potential timing for and receipt of regulatory authorization and guidance related to clinical trials and other clinical development activities including clinical trial commencement or cohort initiation; the potential for regulatory approval, including accelerated approval, of our product candidates; our ability and the potential to successfully manufacture and supply our product candidates for clinical trials; regulatory developments in the United States and foreign countries; our ability to replicate results achieved in our preclinical studies or clinical trials in any future studies or trials; and our ability to fund our future operations. Any forward-looking statements in this press release are based on management's current expectations and beliefs of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from

those set forth in or implied by such forward-looking statements. These risks and uncertainties include, but are not limited to: uncertainties related to the initiation, timing, advancement and conduct of preclinical and clinical studies and other development requirements for our product candidates; the risk that any one or more of our product candidates will cost more to develop or may not be successfully developed and commercialized; the risk that our product candidates will not receive accelerated approval or that we will need to redesign our regulatory strategy; the risk that sufficient capital to fund our future operations will be available to us on acceptable terms or at the times required; and the risk that the results of preclinical studies and/or clinical trials will or will not be predictive of results in connection with future studies or trials. For a discussion of these and other risks and uncertainties, and other important factors, any of which could cause our actual results to differ from those contained in the forward-looking statements, see the section entitled "Risk Factors" in C4 Therapeutics' most recent Annual Report on Form 10-K and/or Quarterly Report on Form 10-Q, as filed with the Securities and Exchange Commission. All information in this press release is as of the date of the release and C4 Therapeutics undertakes no duty to update this information unless required by law.

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