C4 Therapeutics

Protein degraded.
Disease targeted.
Lives transformed.

September 2026

Forward-looking Statements and Intellectual Property

FORWARD-LOOKING STATEMENTS

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

Potential Best-in-Class Cereblon-modulating Protein Degrader of IKZF1/3 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 fully integrated biopharmaceutical company

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

Multiple myeloma (MM);Targeted Protein Degradation (TPD)

C4 Therapeutics

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

2026 Key Accomplishments

- Declared the 75 µg cemsidomide dose level in combination with elranatamab[1] safe in the ongoing Phase 1b trial
  - Initiated the 75 µg expansion cohort and 100 µg safety cohort
- Presented additional analyses from the fully enrolled Phase 1 trial of cemsidomide with dexamethasone in RRMM at EHA 2026
- Advanced start-up activities for an additional Phase 1b trial of cemsidomide with approved multiple myeloma therapies
- Dosed first patients in cemsidomide's Phase 2 MOMENTUM trial and Phase 1b trial with elranatamab
- Further expand platform reach
  - New agreement with Roche focused on DACs[2]
  - Earned a $2 million milestone payment from Biogen for delivery of a second degrader for clinical development

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** with approved standard of care MM therapies
- **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
- **Potentially deliver 3 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-details/c4-therapeutics-expands-long-term-partnership-roche-through-new)

Dexamethasone (dex); Investigational new drug (IND); New Drug Application (NDA); Overall response rate (ORR); Inflammation, Neuroinflammation, Neurodegeneration (INN); Accelerated approval (AA); Multiple myeloma (MM); Degrader antibody conjugates (DACs)

C4 Therapeutics

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

	PROGRAM	TARGET	INDICATIONS	RESEARCH & PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	NEXT MILESTONE
CLINICAL ONCOLOGY PORTFOLIO	**Cemsidomide**	**IKZF1/3**	4L+ Multiple Myeloma	**Phase 2 MOMENTUM trial w/ dex**				**Q1 2027:** Complete enrollment **2H 2027:** Present initial ORR data
			2L+ Multiple Myeloma	**Phase 1b trial w/ elranatamab[2]** Pfizer				**Mid-2027:** Report data from all cohorts
	CFT8919[1]	**EGFR L858R**	Non-Small Cell Lung Cancer	BETTA PHARMACEUTICALS				
INN DISCOVERY	**Discovery**	*Novel targets in pathways of:* **-IL-23/IL-17** **-Type 1 IFN** **-MAPK, PI3K/AKT, NF-kB**	INN *Inflammation, Neuroinflammation & Neurodegeneration*					**2028:** Deliver up to three potential INDS

[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)

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Strategic Platform Collaborations Expand Potential Reach of C4T TPD Medicines

Roche

Active 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

Merck KGaA
Darmstadt, Germany

Active Collaborations

Discovering targeted protein degraders against critical oncogenic proteins

- ✓ Achieved preclinical milestone from a project within the KRAS family

Biogen

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

Cereblon-modulating protein degrader of IKZF1/3

Multiple Myeloma

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Cemsidomide is Positioned for Success

Continued Relevance	Despite recent approvals 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**
Generational Improvement	**Cereblon-modulating protein degraders of IKZF1/3** are designed to **overcome** the **limitations** of **first-generation IKZF1/3 degraders** (IMiDs®)
Potential best-in-class profile	Cemsidomide has a **potential best-in-class profile** among other IKZF1/3 degraders, with a **clinically de-risked MOA**
Strategic Development Path	Two ongoing trials with a third trial expected to start next year to **support cemsidomide's potential to become a backbone MM treatment**

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

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

IMiDs (Pomalyst (pomalidomide) capsules / Revlimid (lenalidomide) capsules) & Cereblon-modulating protein degraders (*Cemsidomide, Iberdomide, Mezigdomide*)

All Degrade IKZF1/3 to Drive Anti-myeloma Activity

Key Roles of IKZF1/3

Physiological Functions:

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

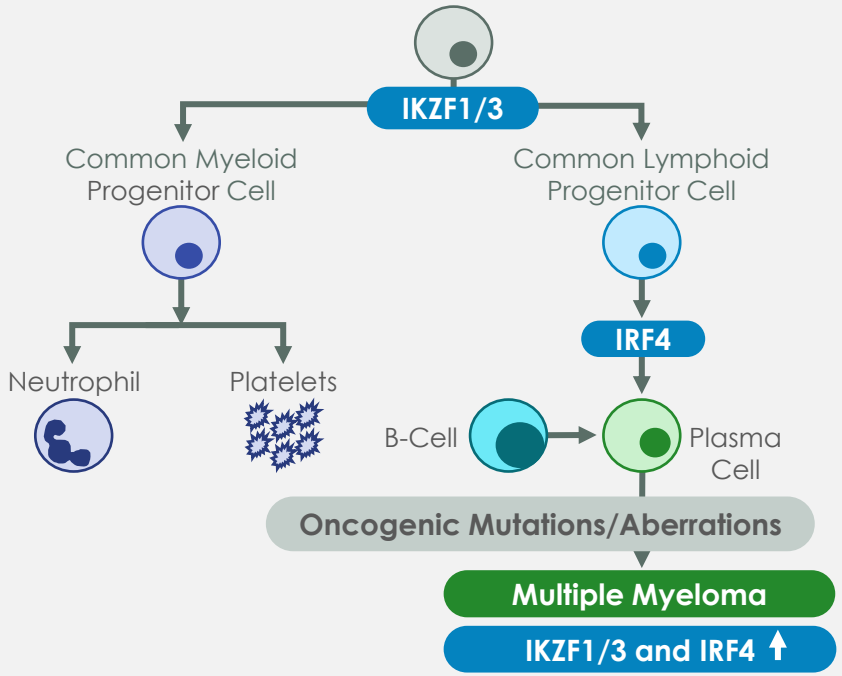
Oncogenic Functions:

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

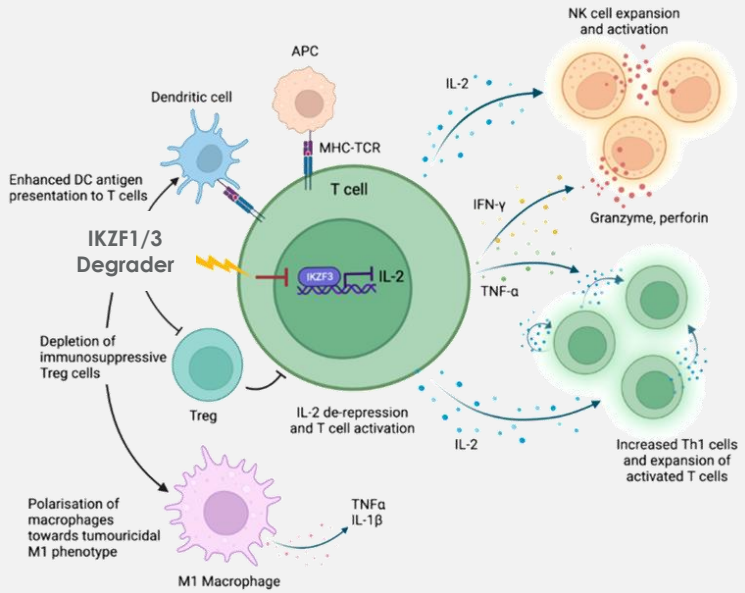
IKZF1/3 Degradation Leads to:

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

Hematopoietic Stem Cell



T-cell Activation



Adapted from Chen and Gooding, 2022

Biology of IKZF1/3 Degradation Supports Use Across Lines of Therapy and Combination Regimens

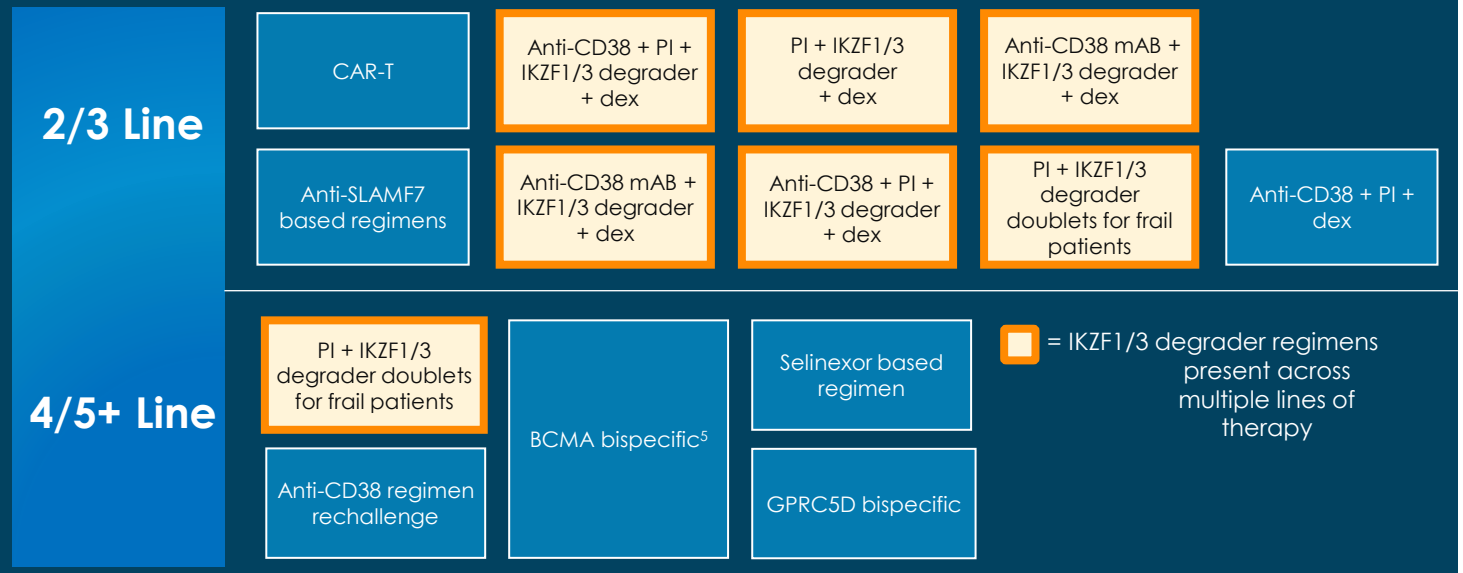
~11K
MM patient deaths expected in the U.S. in 2026[3]

~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]

Treatment Landscape of Approved MM Agents in 2025[1]



- Degradation of IKZF1/3 **remains relevant across multiple lines of therapy**
- Unmet need for a cereblon-modulating protein degrader of IKZF1/3 that is **well-tolerated with compelling anti-myeloma activity**

[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] Linovesltamab is only approved in 5L Multiple myeloma (MM); dexamethasone (dex)

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Discovery of IKZF1/3 Degradation Mechanism Advanced Next-Generation Degrader Development

Foundation
2003 – 2013

- Lenalidomide & pomalidomide developed as IMiDs
- Lenalidomide approved for MM (2006)
- CRBN identified as substrate receptor of CRL4^{c}CRBN E3 ubiquitin ligase (2010)
- Pomalidomide approved for RRMM (2013)

MOA unclear at approval

Mechanism Revealed
2013 – 2015

- Scientific papers confirm lenalidomide and pomalidomide degrade IKZF1/3 via CRBN
- Discovered targeting IKZF1/3 drives MM cell death and prevents proliferation
- Lenalidomide and pomalidomide not optimized for potency[1]
- Molecular glue mechanism discovered: IKZF1/3 recruited to CRL4^{c}CRBN complex

Limited Potency Only Prevents Proliferation, Resulting in Rise of Resistance Mechanisms

Development of Next-generation IKZF1/3 Degraders
2015 – 2020s

- Mezigdomide, iberdomide and cemsidomide designed to be more selective and potent vs. IMiDs
- Mezigdomide & iberdomide developed using chemistry based on IMiDs
- Cemsidomide developed using novel CRBN-binder chemistry

Increased Potency Drives Cell Death AND Blocks Proliferation

Next-generation IKZF1/3 Degraders Advance
2020s – Present

- Cemsidomide demonstrated potential best-in-class profile in a Phase 1 trial in heavily pre-treated RRMM patients
- Cemsidomide advancing in Phase 2 MOMENTUM trial in 4L+ & Phase 1b combo w/ elranatamab[1]
- Mezigdomide and iberdomide in late-stage trials: SUCCESSOR-1; SUCCESSOR-2; EXCALIBER NDMM
- FDA granted AA of iberdomide in combination with daratumumab and dexamethasone (2026)
- T-cell activation data emerging; Synergistic MOA with immune-based regimens

Addresses limitations of IMiDs and remains a foundational MOA

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Cereblon (CRBN); Relapsed refractory multiple myeloma (MM); Mechanism of action (MOA)

[1] CELMoDs May Represent Next Wave of Immunomodulation Approaches in Multiple Myeloma | OncLive

Cemsidomide is a Potential Best-in-Class Cereblon-modulating Protein Degrader of IKZF1/3 in Multiple Myeloma

Clinical Pharmacology

Highly potent & selective
IKZF1/3 degrader

High target specificity
Does not degrade off-target neosubstrates[1], including CK1α and GSPT1

No renal clearance
Relevant for ~50% of patients with renal impairment[2]

Low protein binding
Maximizes free drug availability

Design Elements

~2 day half life

Slower clearance sustains free drug at therapeutic concentrations, while **maintaining efficacy** and **allowing neutrophils to recover**

Results in an effective and convenient **14 days on / 14 days off dosing schedule**

Dosing Schedule + Safety Profile + Sustained Efficacy = Ideal for Combination Regimens

Development Strategy

Initial **differentiated label-enabling strategy** from other IKZF1/3 degraders focused on evolving landscape

Goal is to potentially establish cemsidomide as a **backbone therapy across multiple lines of MM therapy**

[1] Source: C4T data on file; [2] Rana 2020 Blood Advances

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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

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/0081f021-bc0d-4e7f-bdb9-9a01e95ed6eb)

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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

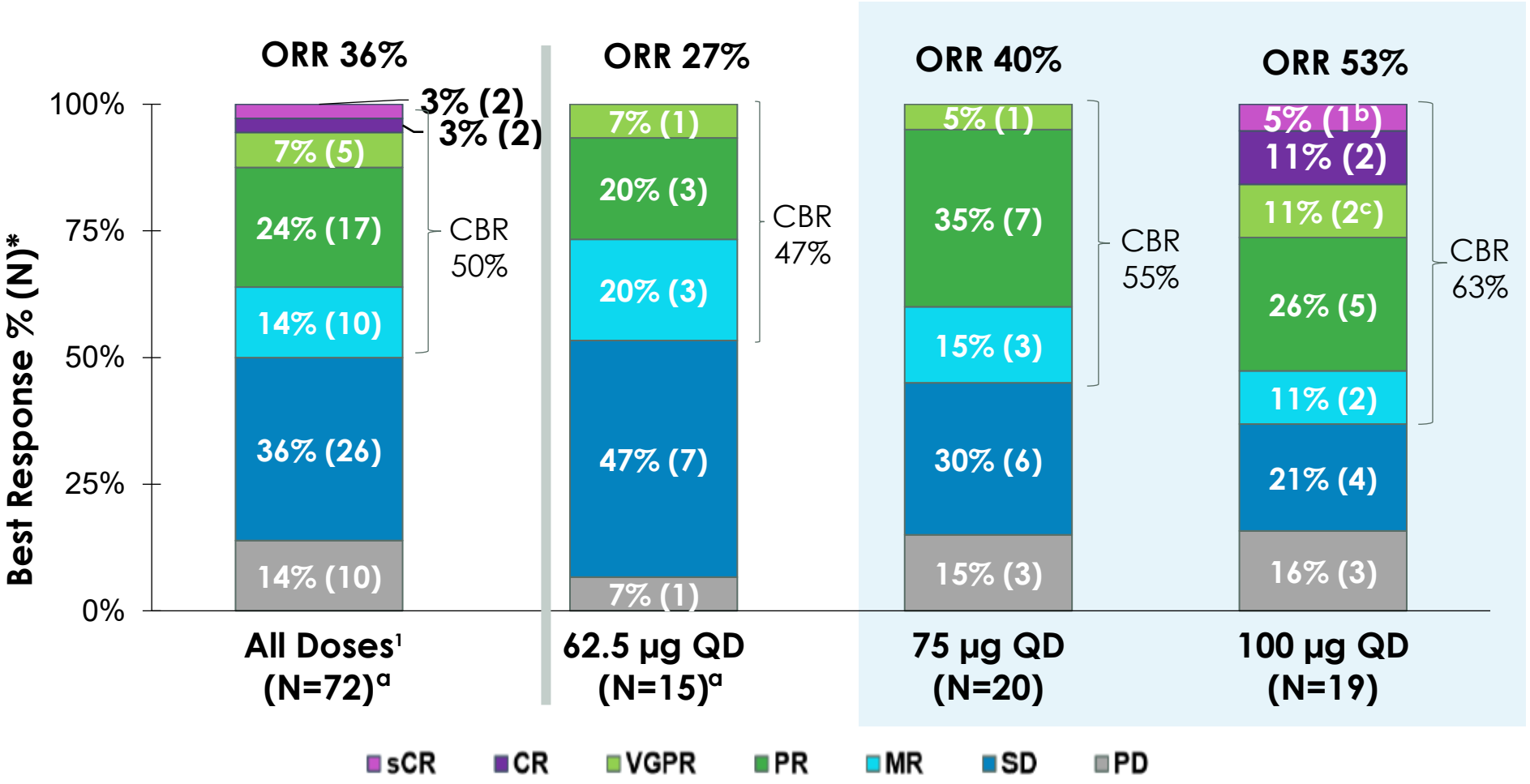
Minimal dose reductions
- TEAEs leading to dose reductions: 5/73 (7%)[1]

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 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

[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/0081f021-bc0d-4e7f-bdb9-9a01e95ed6eb)

Cemsidomide + Dexamethasone Demonstrated Deep and Durable Responses Across the Highest Two Dose Levels



- At 100 µg: **Two patients** who achieved a sCR and CR also **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)
- **ORR was consistent across key subgroups** *(prior CAR-T or T-cell engager therapy, prior BCMA, > 5 prior lines of therapy)*

*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; [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); Stringent complete response (sCR); Stable disease (SD); Very good partial response (VGPR); Confidence Interval (CI)

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/0081f021-bc0d-4e7f-bdb9-9a01e95ed6eb)

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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 cereblon-modulating protein degrader of IKZF1/3 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[4]

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[2]

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

IMiD® Replacement Across Lines
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 + daratumumab
- cemsidomide + dexamethasone + carfilzomib

- Data from SUCCESSOR-1 trial[3] has potential to further validate next-generation IKZF1/3 degraders

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] Pfizer supplying elranatamab (ELREXFIO®), a B-cell maturation antigen CD3 targeted bispecific antibody, to C4T for the Phase 1b trial. [3] SUCCESSOR-1 is a Phase 3 trial evaluating mezigdomide, bortezomib, dexamethasone versus pomalyst, bortezomib, dexamethasone
[4]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/0081f021-bc0d-4e7f-bdb9-9a01e95ed6eb)

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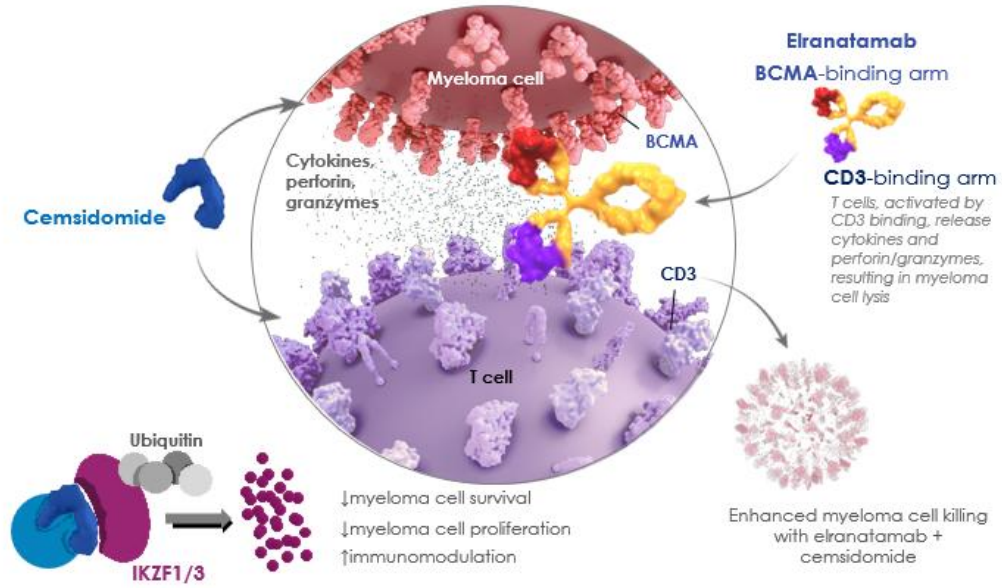
Preliminary Phase 1b Biomarker Data is Supportive of Cemsidomide's Mechanistic Rationale for Combining with Immune-Based Therapies

COMPLEMENTARY MECHANISMS OF ACTION

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 cereblon-modulating protein degrader of IKZF1/3, advancing through clinical development



BIOMARKER DATA FROM THE FIRST TWO PATIENTS IN THE ONGOING PHASE 1B TRIAL[3]

Cemsidomide in Combination with Elranatamb[5]:

Activation and expansion of CD8+ effector memory T cells
Increased HLA-DR expression with Granzyme-B

Observed T-cell activation despite the two patients harboring different immunophenotypes at baseline

Prevented T-cell exhaustion
Measured PD-1, TIM-3 or LAG3 exhaustion markers did not increase when cemsidomide was added

Builds upon broader immunomodulatory data
In the Phase 1 trial, cemsidomide demonstrated robust T-cell activation as a monotherapy and in combination with dexamethasone[4]

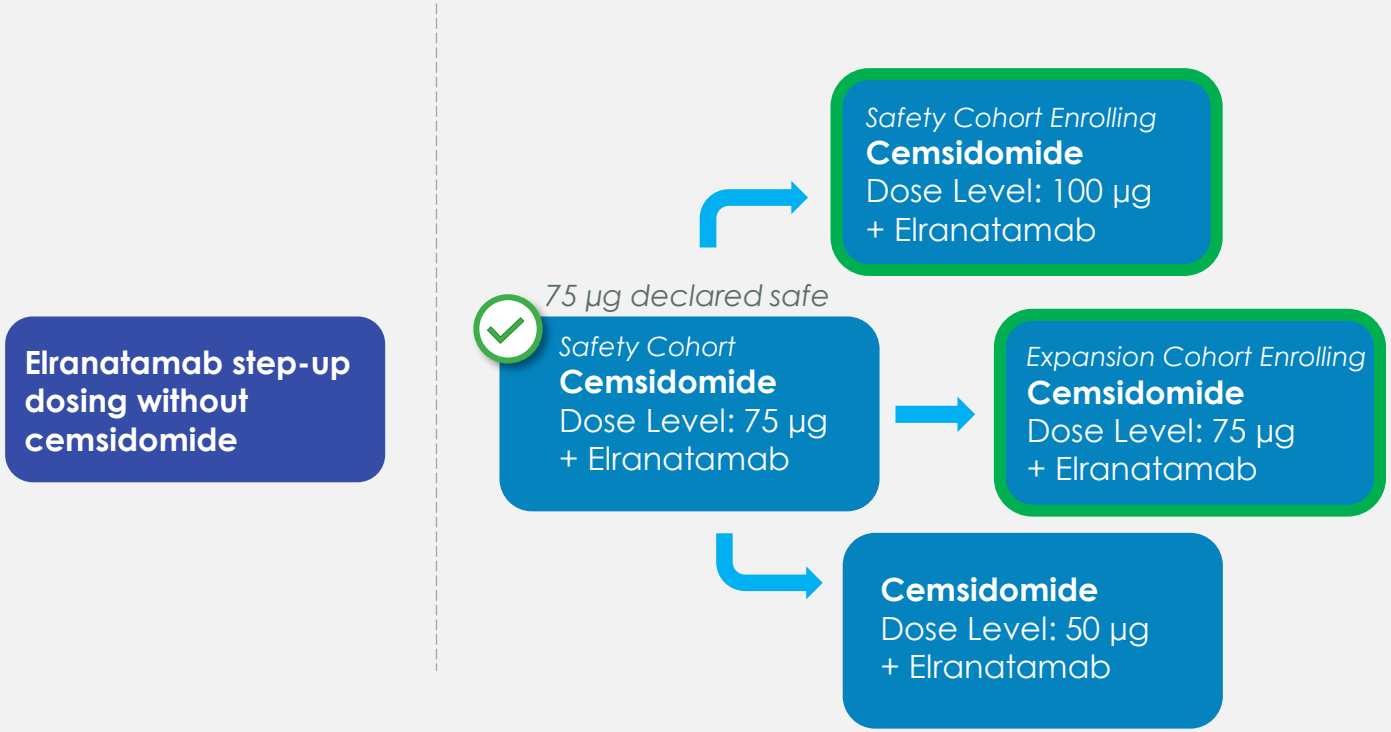
Totality of the data support cemsidomide as a potential combination partner for immune-based therapies

[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 as presented at IMS September 2026 based on available biomarker data, as of June 22, 2026, from the first two patients to complete cycle 1: https://c4therapeutics.com/our-science/scientific-presentations-publications/ ; [4] C4T data on file: https://ir.c4therapeutics.com/static-files/39670c4f-0806-41b6-8813-ef7adcf04207; [5] Pfizer supplying elranatamab (ELREXFIO®), a B-cell maturation antigen CD3 targeted bispecific antibody, to C4T for the Phase 1b trial
B-cell maturation antigen (BCMA); Immunomodulatory drug (IMiD); Monoclonal antibody (mAb); Proteasome inhibitor (PI); Relapsed or refractory multiple myeloma (RRMM); Cereblon (CRBN)

First Cemsidomide Dose Level (75 µg) in Phase 1b Trial with Elranatamab Declared Safe; Advancing Trial to Dose Expansion and Escalation Cohorts

Trial Initiated in Q1 2026; Enrollment Ongoing

Potential to expand at each dose level once combination is declared safe



Mid-2027: Data from all cohorts evaluated

PHASE 1b TRIAL DESIGN:

Primary Objectives:

Characterize the safety and tolerability of cemsidomide in combination with elranatamab[1]

Dosing Regimen:

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

Key Differentiators:

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

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

Phase 2 MOMENTUM Trial of Cemsidomide + Dexamethasone in 4L+ MM Continues to Progress

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

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)

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

Targeted Protein Degradation (TPD); Central nervous system (CNS)

C4 Therapeutics

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 benefit 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

C4 Therapeutics

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

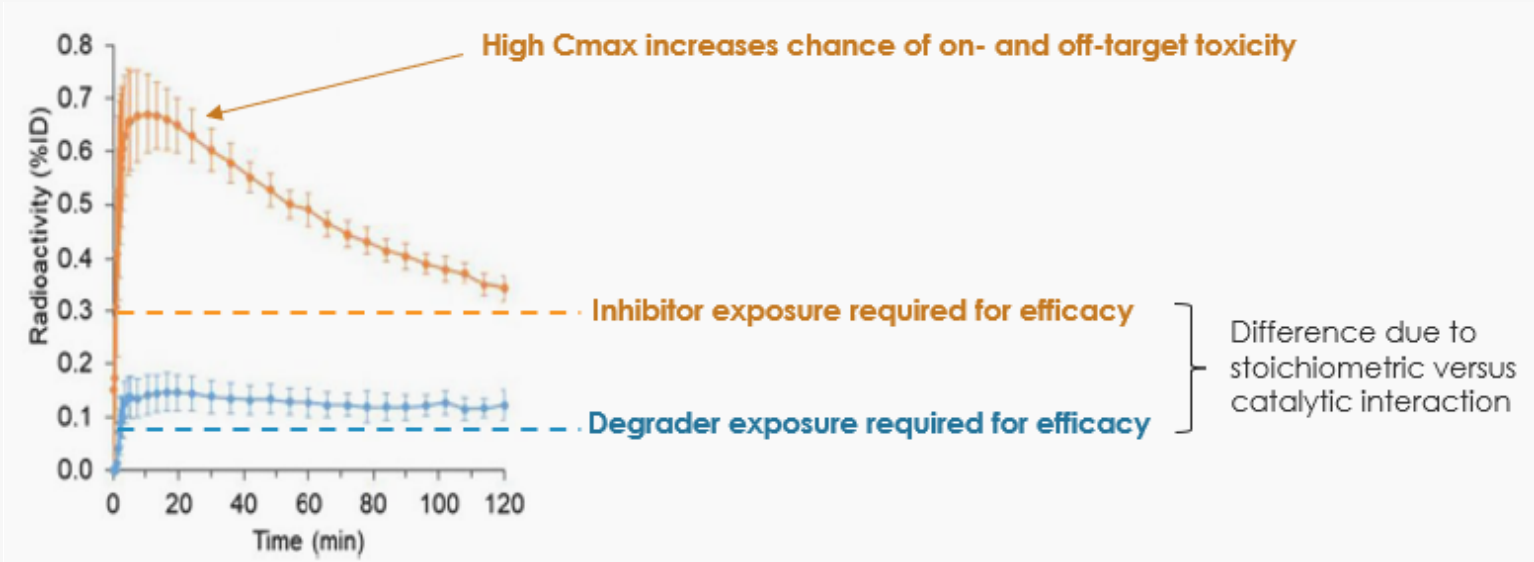
Lower exposure levels for highly catalytic degraders are required for efficacy versus inhibitors to **achieve efficacious** results in CNS diseases

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*

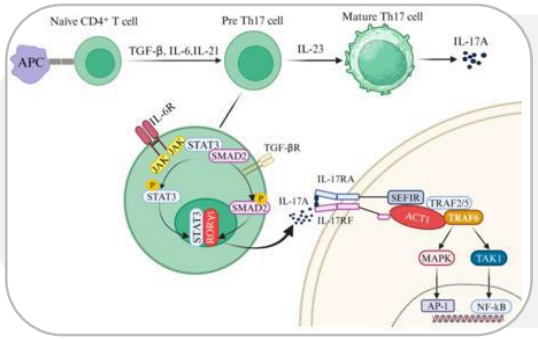


Sources: Drug Discov Today. 2019 May;24(5):1067-1073. doi: 10.1016/j.drudis.2019.01.015; Pharm Res. 2022 Jul;39(7):1321-1341. doi: 10.1007/s11095-022-03246-6

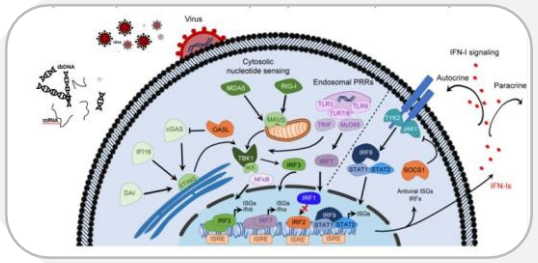
Central nervous system (CNS); Pharmacokinetic (PK)

C4 Therapeutics

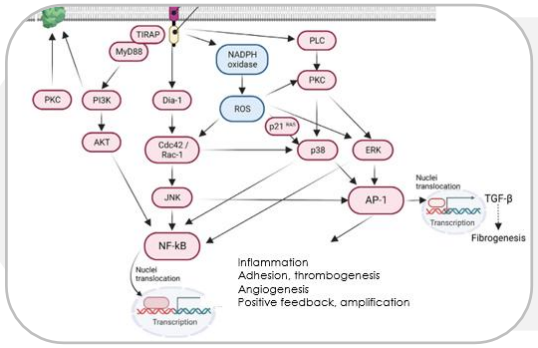
Pursuing Targets in Validated Pathways With Application to a Broad Set of Indications



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 Steatohepatitis
- 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

C4 Therapeutics

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

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

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

- **Complete enrollment** for Phase 2 MOMENTUM trial
- **Initiate additional Phase 1b Trial** with approved standard of care MM therapies
- **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

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
- **Potentially deliver 3 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); Investigational new drug (IND); New Drug Application (NDA); Overall response rate (ORR); Inflammation, Neuroinflammation, Neurodegeneration (INN); Accelerated approval (AA); Multiple myeloma (MM); Degrader antibody conjugates (DACs)

C4 Therapeutics