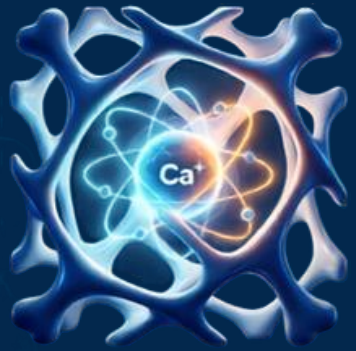
AACR American Association for Cancer Research

Research Presented in 2025 & 2026

ACRI ADVANCED CALCIUM RESEARCH INSTITUTE



Antiorbital Ionic Calcium Carbonate (AIC)

Starving **Multiple Myeloma** by Re-engineering the Bone Microenvironment

Research by

UAMS University of Arkansas for Medical Sciences

MOFFITT CANCER CENTER

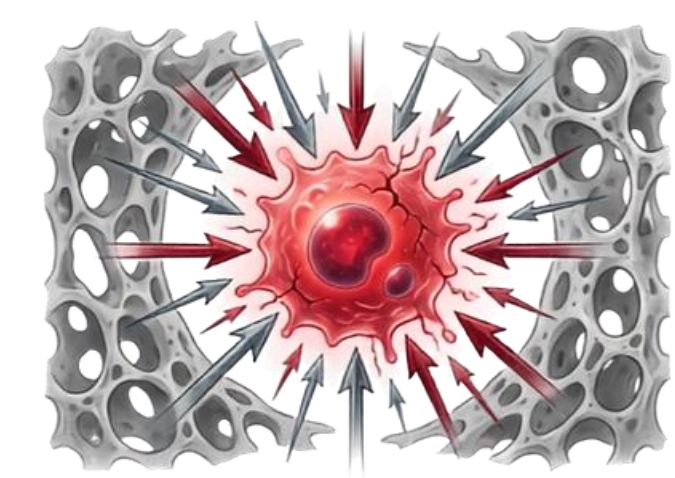
Research Funded by

NIH NATIONAL CANCER INSTITUTE

PAULA & RODGER RINEY FOUNDATION

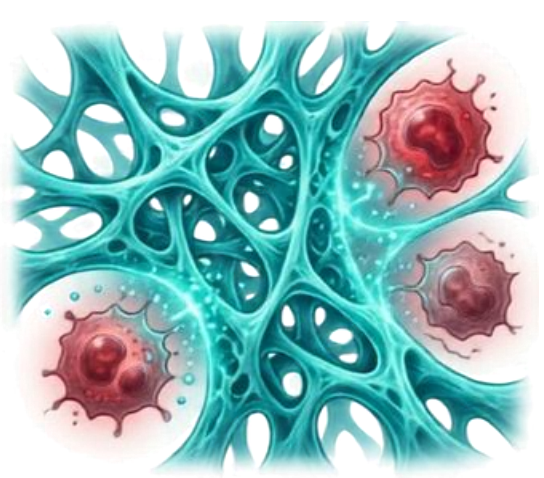
The Multiple Myeloma Paradigm: Seed vs. Soil

The Standard Approach: Attacking the Seed



Marginal effects on myeloma progression, high resistance, inevitable relapse. The soil remains hospitable to surviving cancer cells.

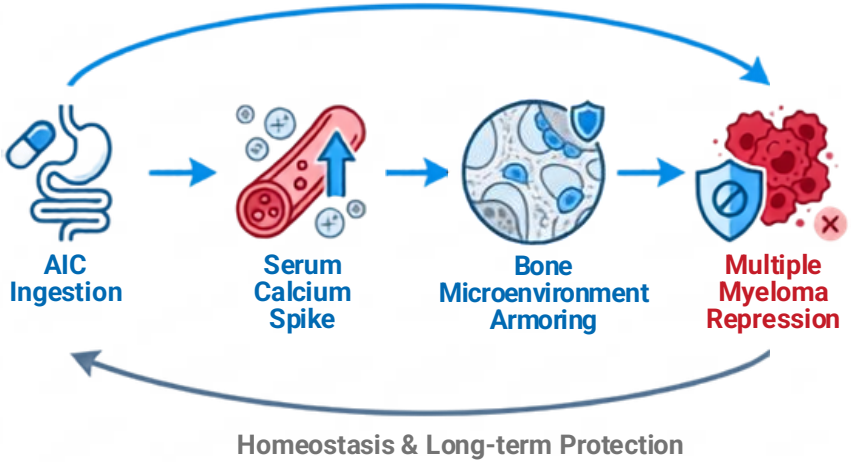
The AIC Innovation: Fortifying the Soil



Ecological modification. Fortifying the bone marrow microenvironment makes it impossible for 5TGM1 myeloma cells to take root or thrive.

Key Insight: Multiple Myeloma heavily relies on the surrounding bone marrow microenvironment. Mice surviving initial transplantation exhibit complete resistance to tumor rechallenge. Fortified bone provides a natural protective mechanism.

Fortifying the Bone Niche: AIC Dietary Intervention to Prevent Multiple Myeloma

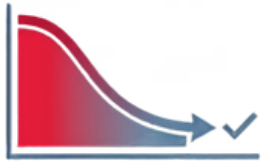


The Innovation

Antiorbital Ionic Calcium carbonate (AIC) is a uniquely engineered, highly bioavailable calcium supplement utilizing weak sigma anti-bonding.

The Mechanism

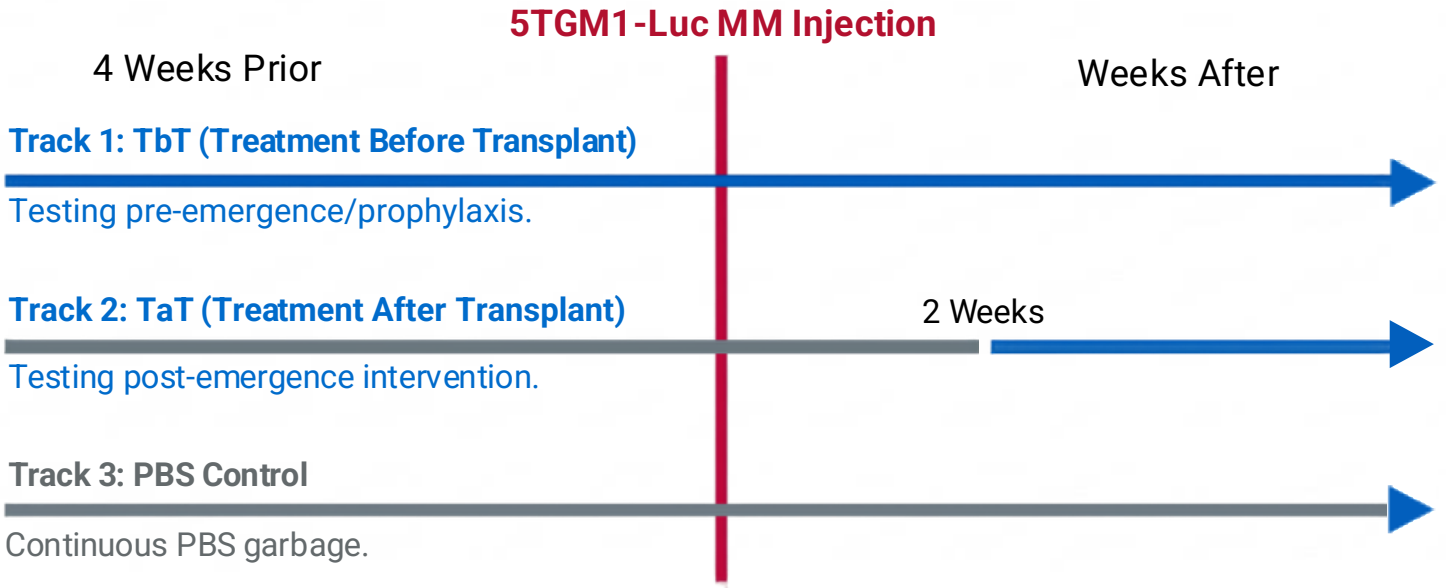
AIC drives a massive shift in the bone microenvironment, significantly elevating osteoblast differentiation while suppressing osteoclasts.



The Clinical Outcome

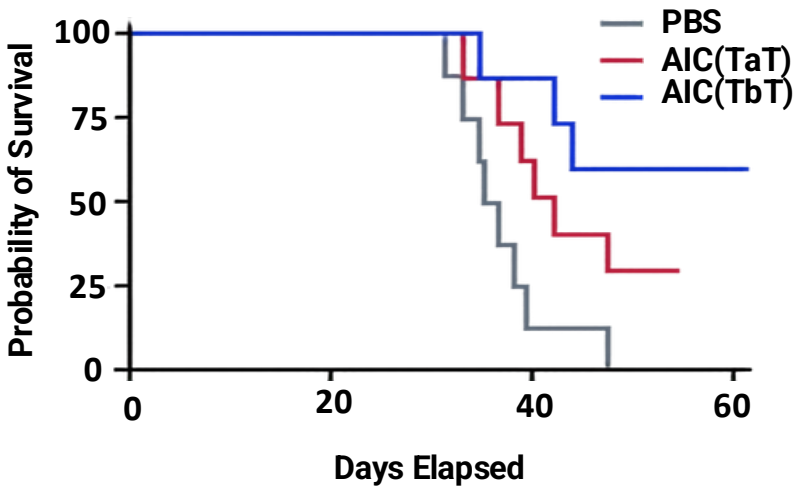
Prophylactic AIC treatment radically delays Multiple Myeloma (MM) engraftment and tumor progression in vivo, extending median survival indefinitely in murine models.

Clinical Regimen Design: Pre-Emergence vs. Post-Emergence



Methodology Note: 5TGM1-Luc cells injected via tail vein into NSG (immunodeficient) mice, monitored weekly via bioluminescence.

Survival Outcomes: Complete Prevention of Mortality in NSG Models



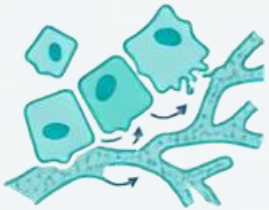
PBS Control
- 100% mortality.
- Median survival = 39 days.

AIC TaT
- Delayed mortality.
- Median survival = 45 days (p = 0.0437).

AIC TbT
- Unprecedented survival.
- Median survival =Undefined (p = 0.007).

THE CONCLUSION: Pre-fortifying the bone environment with AIC before cancer introduction essentially nullifies the lethality of the 5TGM1 MM cells in this model. Even post-emergence intervention showed statistically significant benefit (p=0.0437); however, the prophylactic window is clearly the optimal use case.

The Hostile Soil Cross-Section: Shifting the Cellular Balance



Extracellular Ca^2 triggers rapid osteoblast differentiation and bone matrix protein expression (elevated P1NP).

Significant Proliferation ($p<0.003$)

Total Suppression ($p<0.0001$)

Reversal of the typical myeloma-induced osteolysis.

The extreme ratio shift physically occupies the space and overrides the biochemical signals that 5TGM1 myeloma cells require to survive, effectively starving the cancer niche.

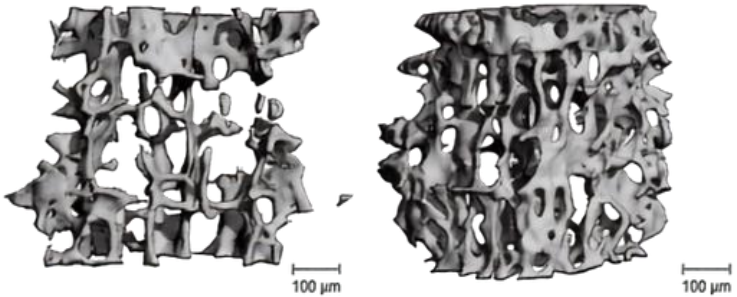
Osteoblasts
(The Builders)

Osteoclasts
(The Destroyers)

Structural Remodeling: The Bone Microenvironment Dashboard

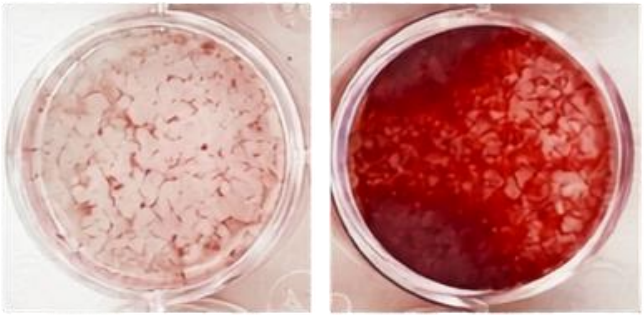
Visual Proof: 3D Micro-Architecture

Control (PBS) | AIC (TbT)



Biochemical Proof: Mineralization

PBS Control | AIC Sustained



Key Metrics (In Vivo Structural Osteogenesis)

Bone Mineral Density (BMD)

Significant Increase
(p=0.030)

Bone Volume (BV/TV)

Massive Expansion
(p<0.018)

Trabecular Thickness (Tb.Th)

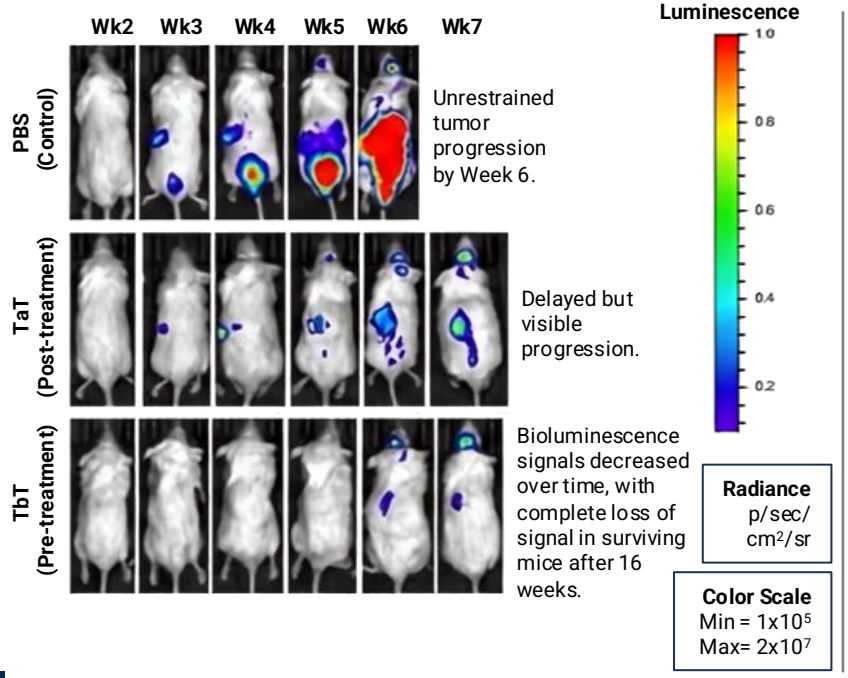
Structural Fortification
(p<0.22)

Takeaway: Elevated systemic calcium directly drives massive in vivo and in vitro structural osteogenesis, transforming porous environments into impenetrable mineral matrices.

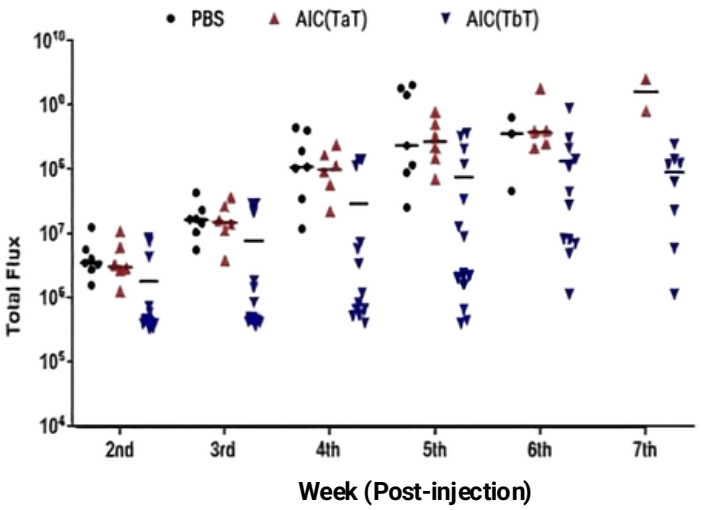
Disease Progression: AIC Dramatically Delays Tumor Burden

In the NSG immunodeficient mouse model, control subjects exhibited rapid MM engraftment and progression. Conversely, pre-treatment with AIC suppressed tumor growth entirely or delayed it massively.

Bioluminescence Imaging: Tumor Engraftment and Progression



Quantitative Analysis: Total Flux Scatter Plot

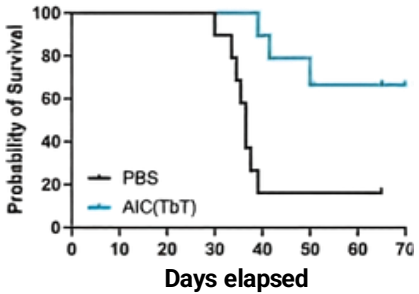


Validation: Success in an Immune-Competent Model

To ensure results were not anomalies of immunodeficient mice, the study was replicated in the C57BL/KaLwRij conventional MMBD model—known for pronounced osteolytic lesions and immune competence.

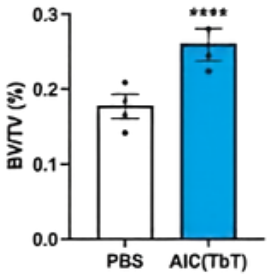
Validation Dashboard

1. Survival



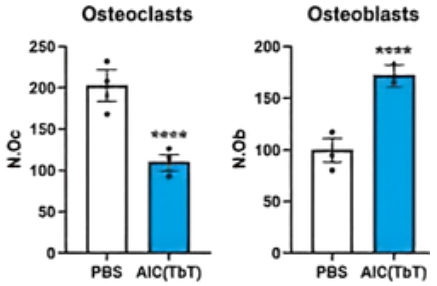
TBT median survival remains **Undefined** vs 46 days for control (p=0.007).

2. Bone Volume



Massive, statistically **significant increase** in trabecular bone volume (p=0.0001).

3. Cellular Shift



The **exact same biological mechanism** was observed: increased osteoblasts, decreased osteoblasts.

Takeaway: The efficacy of AIC is robust and translatable across diverse biological systems.

Clinical Implications & The Future of Preventative Care

Highly Tolerable Intervention

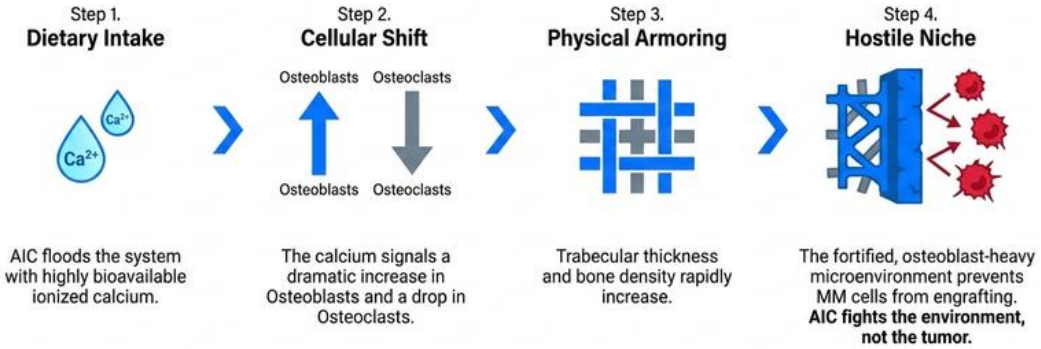
As a dietary supplement, AIC acts with extreme potency but zero observed renal toxicity or crystallization, unlike high-dose standard calcium.

The Prophylactic Paradigm

Data explicitly proves that pre-treatment (T1B1) is vastly superior to post-treatment. Altering the bone niche before cancer emerges is a viable new therapeutic philosophy.

Target Populations

AIC represents a breakthrough, low-toxicity regimen specifically suited for high-risk patients with pre-emerging MM (MGUS/SMM) or for maintaining deep remission and preventing relapse.

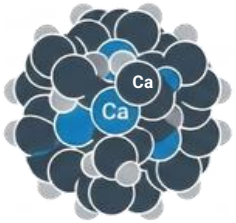


THE PARADOX:

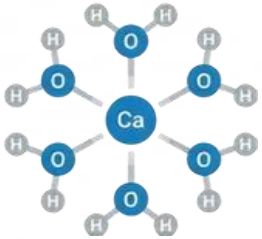
In Vitro, AIC has marginal direct effects (<10% inhibition) on isolated myeloma cells. Yet, In Vivo, it stops the cancer entirely. **Why?**

The AIC Innovation: Weak Sigma Anti-Bonding

Conventional Calcium



AIC Calcium



- Independent of Vitamin D for bloodstream absorption.
- Bypasses Albumin protein binding.
- Remains as bioavailable ionized calcium.

Antiorbital Ionic Calcium Carbonate (AIC-CaCO3) fundamentally alters the absorption pathway by altering the molecular bond, maintaining a highly reactive, ionized state in the body. AIC sustains elevated serum ionic calcium levels, significantly restoring calcium homeostasis at both the systemic and cellular levels by stimulating bone formation and cellular decalcification.

Introducing Antiorbital Ionic Calcium Carbonate (AIC)

The Origin

Extracted from natural oyster shells.

The Transformation

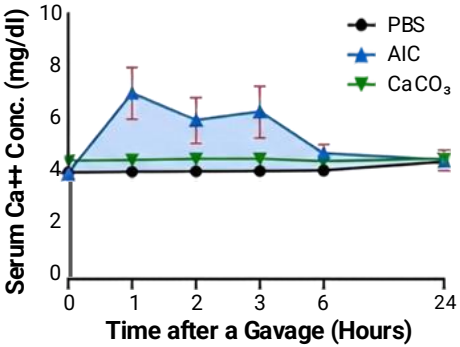
Structurally modified to alter energetic barriers and electron orbital states.

The Result

A weak-bonding, potent dietary calcium supplement that exists in a highly bioavailable, ion-like state in aqueous conditions.

AIC vs. Conventional Calcium Carbonate (CaCO3)

Dimension	$CaCO_3$	AIC
Molecular Bond	Stable Ionic	**Sigma Anti-Bonding**
Solubility	Low	**~200x more soluble**
Reaction Rate	0.035 mol/L per sec	**0.160 mol/L per sec (4.57x faster)**
In Vivo Ionization	Negligible serum change	**Rapid, sustained increase in serum Ca++**

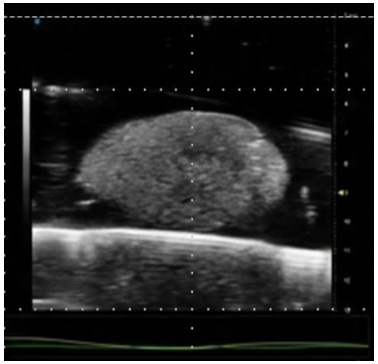


Long-Term Maintenance

Continuous twice-daily intake yields a sustained elevation of serum calcium ion levels across multiple mouse strains (NSG and C57BL6)

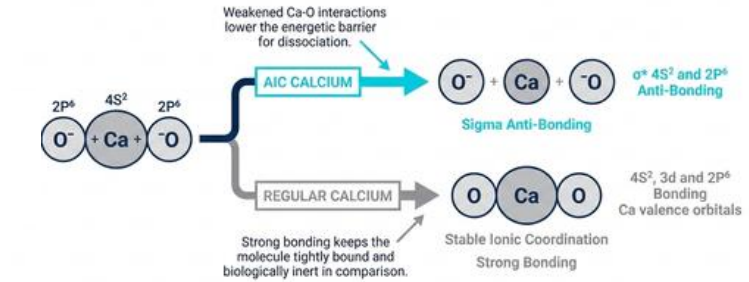
Key AIC Triggers

- PTH inhibition
- BMP-2 Activation
- Calcitonin Secretion



Elevated urinary calcium is safely excreted. *Ex vivo* ultrasound confirms absolutely zero crystallization or kidney stone formation after prolonged AIC regimens.

The Molecular Blueprint: Sigma Anti-Bonding



The anti-bonding orbital configuration creates exceptional water molecule solvation-driven coordination, allowing AIC to instantly flood the system with active calcium ions.

Kyung Hee University
Department of Pharmaceutical Biochemistry

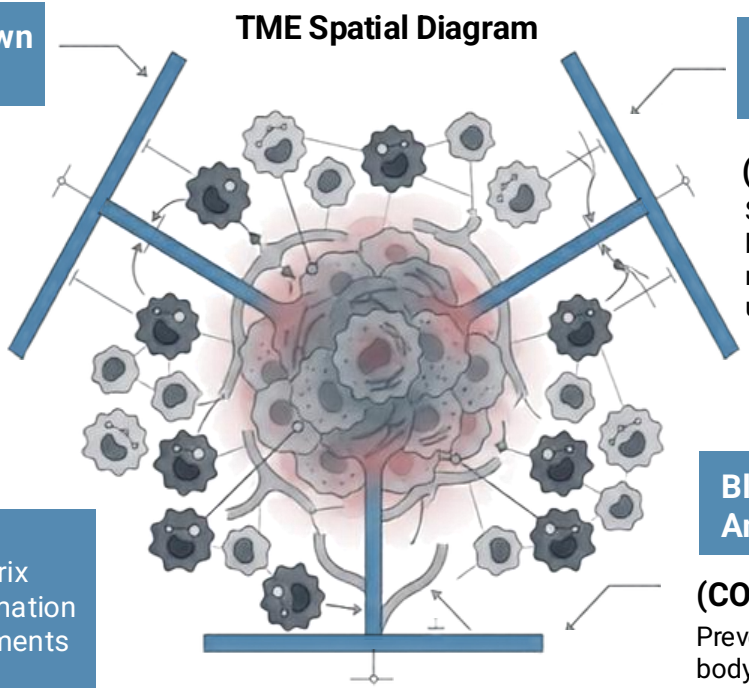
Oncology: Normalizing the Tumor Microenvironment

AIC does not act as indiscriminate cytotoxic chemotherapy; it alters the biochemical “soil” so the cancer “seed” cannot thrive.

Blockade 1: Shutting Down Survival Signals

(IL-6 / TNF-α / IL-1β)
Prevents Tumor-Associated Macrophages (TAMs) from turning on anti-apoptosis switches that keep cancer cells alive.

TME Spatial Diagram



Blockade 3: Halting Metastatic Escape

(MMPs)
Strengthens structural tissue barriers, disabling the molecular scissors cancer uses to enter the bloodstream.

Blockade 2: Blocking Angiogenesis Fuel Lines

(COX-2)
Prevents the tumor from signaling the body to build dedicated blood vessels for nutrient supply.

Kyung Hee University’s AIC research on extracellular matrix (ECM) protection and inflammation reduction effectively complements our MM study.

Human Case Study #1: Initiation of AIC Following Discontinuation of Conventional Treatments

Patient 01

Female, Age 80, South Korea

- Diagnosed with Multiple Myeloma in 2012.
- Commenced AIC Therapy in Dec. 2013.

Cancer Remission (Hematology)

Baseline (Nov 2013)
WBC 2,190 K/uL

UP

Current (Jan 2015)
WBC 4,760 K/uL

Platelets and Neutrophils fully normalized.

Renal Recovery

Baseline (Nov 2013)
GFR 12

UP

Current (Nov 2015)
GFR 62

Reversal of chemo-induced damage.

Bone Density (Osteoporosis Reversal)

Baseline (Nov 2013)
T-Score -3.6

UP

Current (Nov 2015)
T-Score -0.9

(Normal Range: Above -1.0).

This clinical case was provided by ACRI. The patient achieved a normal CBC within 14 months of initiating AIC therapy. Furthermore, her previously declining kidney function normalized, and her osteoporosis was fully reversed.

Human Case Study #2: Initiation of AIC for 5 months Prior to Conventional Treatments

Patient 02
Female, Age 67
USA

- Diagnosed Nov. 2023. Presented with diffuse lytic lesions and multiple compression fractures. Refused to see an oncologist.
- No conventional MM treatments received for 5 months, including Zometa. Maintained exclusively on AIC Therapy (3-4x daily) and pain medication.

Hypercalcemia Resolution

Baseline (Nov 2023):
Calcium 11.2 mg/dL

DOWN

Current (Apr 2024):
Calcium 9.1 mg/dL

Return to Normal Range (8.4-10.5).

Renal Function Restoration

Recent (Mar 2024):
E-GFR 62 mL/min

UP

Current (Apr 2024):
E-GFR 83 mL/min

Strong function (Normal: >=60).
Total Urine Protein dropping sharply (24 to 9.6).

Systemic Resilience

Recovery trajectory maintained despite severe intervening infections between Jan-Feb 2024 (Meningitis, Pneumonia, C Diff).

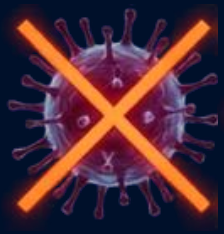
This clinical case was provided by ACRI. The patient began conventional treatments (Valcade and Darzalex, without Zometa) in May 2024, following improvement with AIC therapy. As of 2026, on her oncologist's recommendation, she has discontinued all treatments except for a daily maintenance dose of AIC therapy. She continues to be actively monitored.

The Synthesis:

The AIC Microenvironment Framework

Chemistry: Sigma Anti-Bonding Configuration creates a massive influx of bioavailable Ca^{2+} ions.

Physiology: Ion flood triggers an Osteoclast-to-Osteoblast cellular shift, radically increasing trabecular density.



Outcome: Tumor starvation, failed engraftment, and undefined survival extension.

Oncology: The remodeled, structurally dense bone niche becomes fundamentally **hostile** to multiple myeloma colonization.

To book a Zoom session with ACRI research team and to learn more about AIC therapy.

To study more research and clinical cases of AIC therapy.

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