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ACRI ADVANCED CALCIUM RESEARCH INSTITUTE

A Paradigm Shift in Bone Health

Clinical Efficacy of Antiorbital Ionic Calcium Carbonate (AIC) in Restoring Bone Mineral Density

Based on peer-reviewed clinical data investigating the cellular reversal of osteoporotic states.

Research Presented in 2025

ASBMR The American Society for Bone and Mineral Research

Collaborative Research by

UAMS University of Arkansas for Medical Sciences

MOFFITT CANCER CENTER

SUNG KYUN KWAN UNIVERSITY 1398

The Escalating Burden

(Asian Adult Population)

The Failure of the Status Quo

Bisphosphonates

Slowing loss, but failing to halt progression. High residual fracture risk remains.

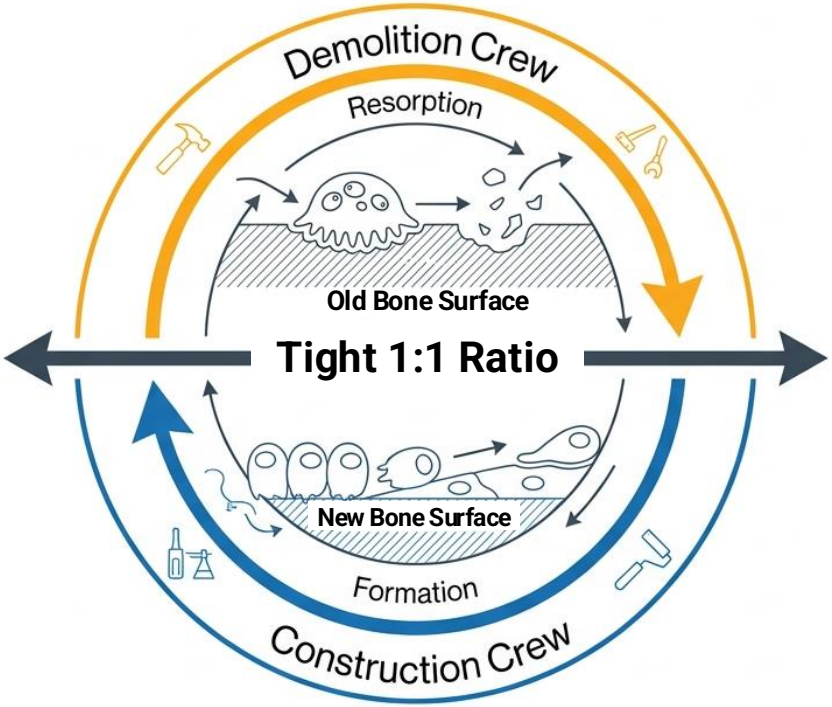
Standard Calcium

Poor bioavailability and heavy dependency on Vitamin D for absorption. Has issues with calcification and side effects.

The Biological Bottleneck

Dietary calcium rapidly binds to albumin in the bloodstream, rendering it physiologically inactive.

The Zero-Sum Game of Coupled Bone Remodeling



The Problem

In a healthy skeleton, remodeling is strictly coupled. You cannot easily build new bone without first breaking down old bone.

The Therapeutic Trap

Traditional pharmacological therapies, like bisphosphonates, merely slow down the demolition phase (osteoclasts).

Because the cycle is coupled, halting demolition eventually halts construction. This slows the decline, but struggles to achieve active, net-positive bone reversal.

Clinical Methodology: An 8-Month Evaluation/Analysis

82 Asian Participants

Aged 30 - 70+

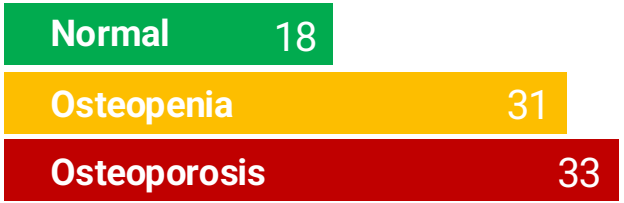
Dosing: 7.6 mg of **AIC** 2x Daily

Duration: 8 months

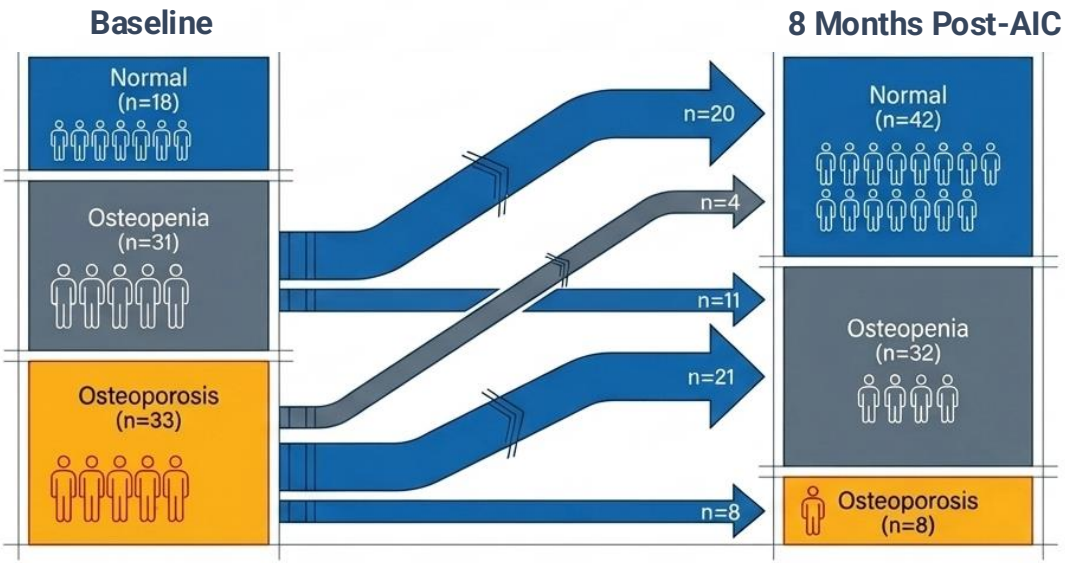
Distal Radius Ultrasound

MiniOmni™ System
Tracking T-score, BMD, and SOS

Baseline Clinical Status



Eight Months to Uncouple the Remodeling Cycle



65% of Osteopenia patients improved to **Normal.**

64% of Osteoprosis patients transitioned up to **Osteopenia.**

12% of Osteoprosis patients fully **normalized**.

Across an 82-patient Asian cohort, a daily 7.6mg x 2 intervention of Antiorbital Ionic Calcium (AIC) triggered active bone reversal across all age groups and genders. These results indicate a fundamental uncoupling of the remodeling cycle.

Transformative Efficacy Across All Primary Bone Health Metrics

BMD
(Bone Mineral Density)

+16% Increase

0.95 → 1.10 g/cm²

p<0.0001

T-Score

+1.15

-2.0 → -0.85

p<0.0001

SOS
(Speed of Sound)

+133

3,946 → 4,079

p<0.0001

After 8 months, the average participant migrated from a baseline average of clinical osteopenia (-2.0) well into the normal range (-0.85), just above the osteopenia threshold.

Universal Efficacy Across Gender and Menopause Status

Male Cohort

Highest overall T-Score improvement.

Non-Menopausal Female

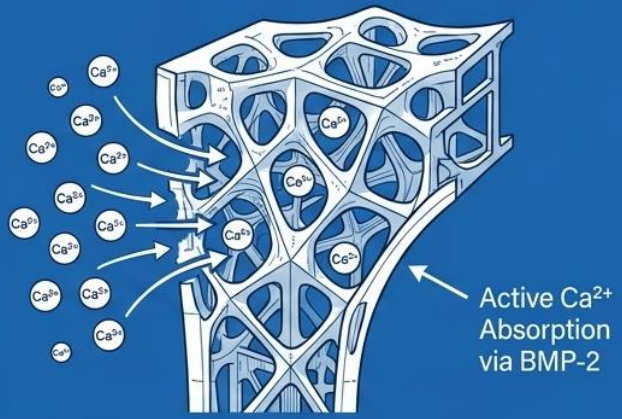
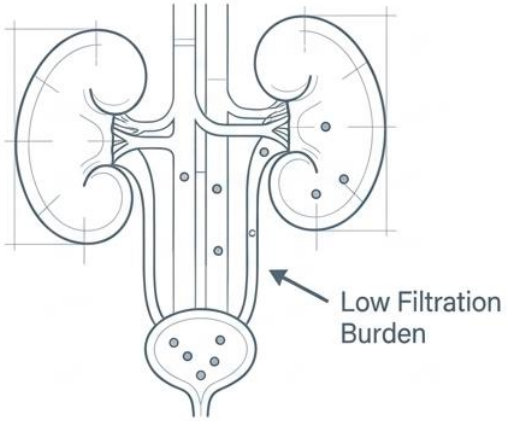
8.1% average BMD increase.

Menopausal Female

15.1% average BMD increase.

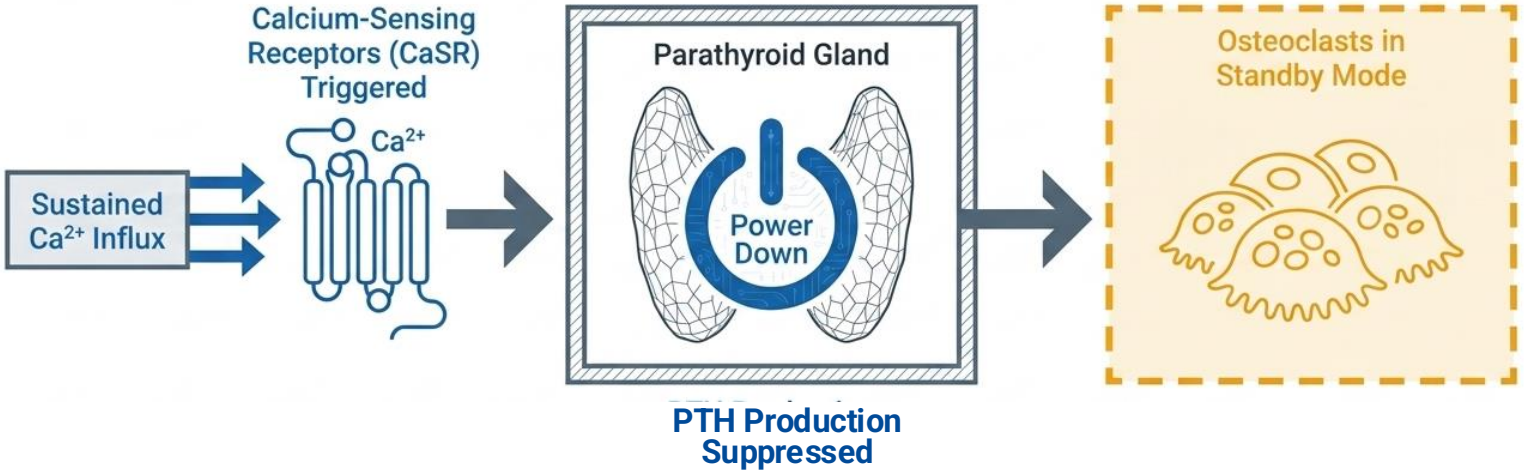
Estrogen deficiency during menopause typically accelerates calcium outflow. Remarkably, the menopausal cohort demonstrated a near-double percentage increase in BMD compared to non-menopausal females (15.1% vs 8.1%, p=0.028).

Achieving Homeostasis Without Nephrotoxicity

The Skeletal Sponge	The Protected Kidneys
	

"Conventional calcium supplements often rely on massive doses that over-saturate the body, putting a heavy filtration burden on the kidneys and risking arterial calcification. AIC therapy completely avoids this toxicity. It utilizes a highly efficient dual mechanism: minimizing bone loss while triggering BMP-2 to actively draw new ionic calcium directly into the skeletal matrix. By providing an immediate destination for the calcium, the skeleton rapidly clears it from circulation, sparing the kidneys from stress. Best of all, because AIC achieves this through a precise, targeted daily dose of only 16 mg, it safely restores bone health without overloading the body."

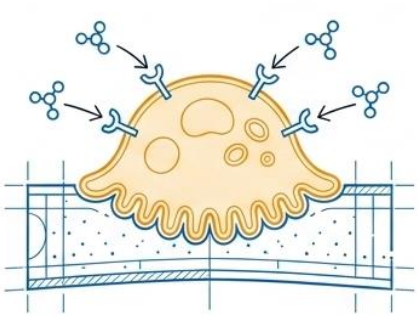
Pathway I: Suppressing Parathyroid Hormone to Halt Demolition



PTH is the body's primary calcium retriever. By maintaining elevated active calcium, AIC continuously triggers parathyroid CaSR. The body registers absolute abundance, **deeply suppressing PTH**. This cuts off the primary fuel for bone breakdown, effectively putting the osteoclast demolition crews on prolonged standby.

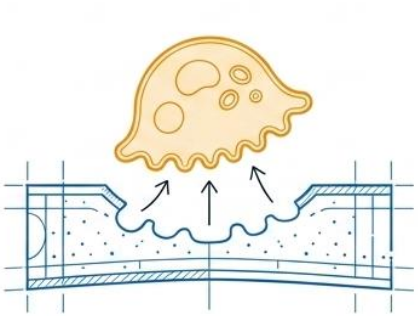
Pathway II: Calcitonin Release Immobilizes Existing Osteoclasts

1.Binding



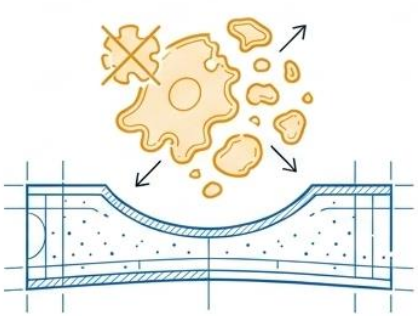
Calcitonin binds to active osteoclast receptors

2. Detachment



Osteoclast shrinks and detaches from bone matrix

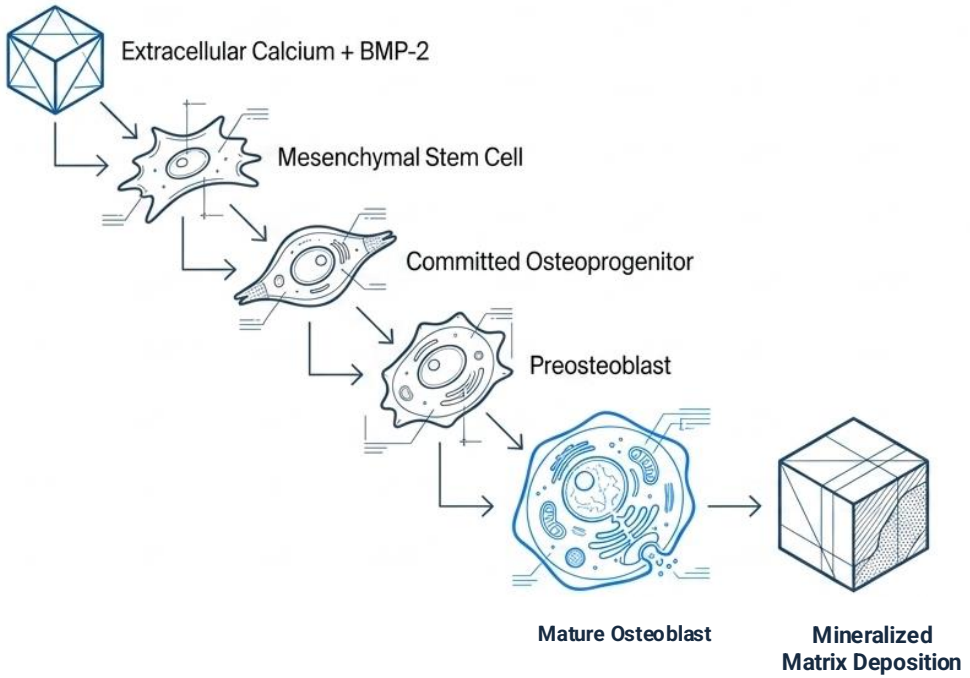
3. Apoptosis



Cell undergoes programmed destruction

While low PTH prevents the birth of new bone-destroying cells, the sustained calcium influx triggers the thyroid's C-cells to release calcitonin—a physical lock on existing demolition. Active osteoclasts possess specific calcitonin receptors. Upon binding, these cells instantly shrink, stop secreting bone-dissolving acid, and detach from the skeletal surface, eventually undergoing apoptosis (cell death) within hours.

Pathway III: BMP-2 Activation Forces Independent Construction



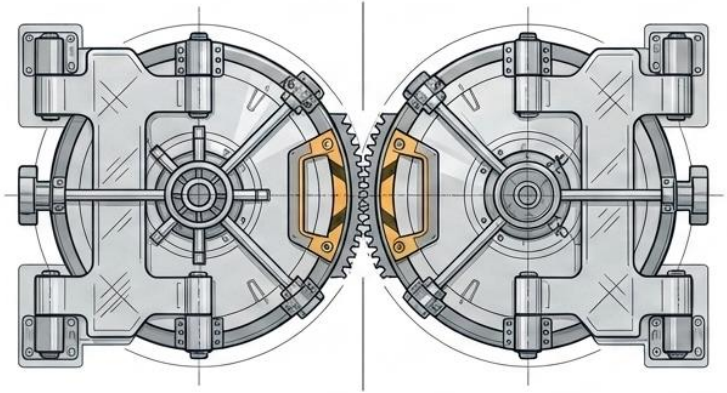
Typically, bone construction slows when demolition stops. AIC upregulates this coupled cycle to favor bone formation over resorption.

High local concentrations of extracellular Ca^{2+} upregulate Bone Morphogenetic Protein-2 (BMP-2). This acts as an independent command, forcing bone marrow mesenchymal stem cells to differentiate into mature osteoblasts. They begin aggressively depositing a new mineralized collagen matrix, surpassing already down-regulated osteoclast activity.

The Triad of Reversal: Creating a Net-Positive Window

The Dual Lockdown

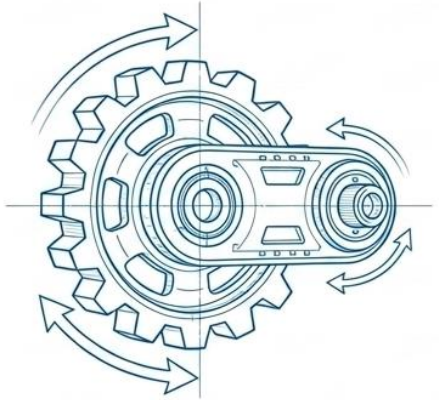
PTH Suppression + Calcitonin Release



Plummeted Osteoclast Activity

The Independent Engine

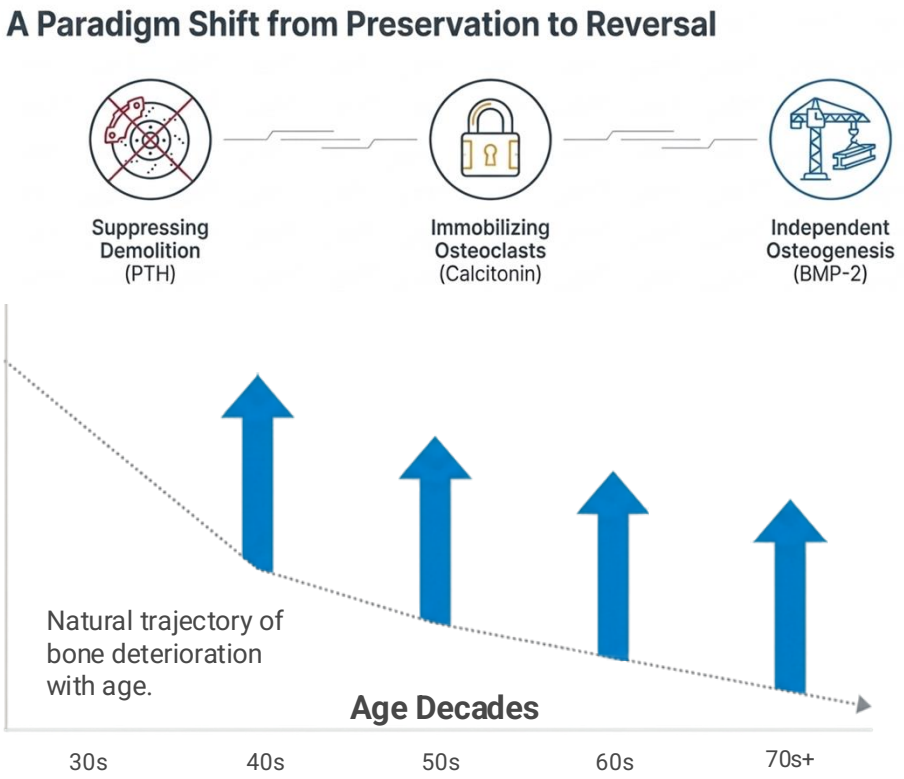
BMP-2 Activation



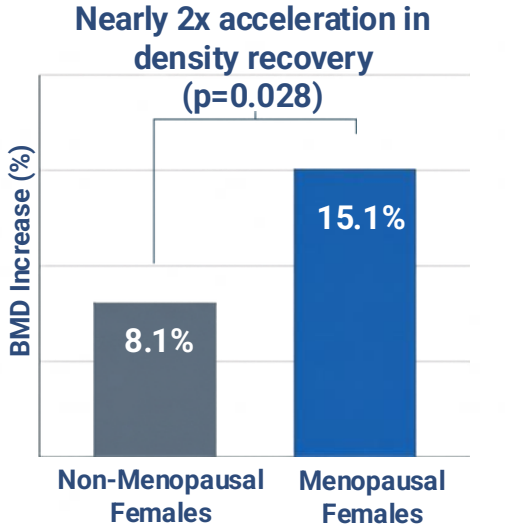
Supercharged Osteoblast Activity

AIC therapy introduces a powerful shift in bone kinetics. By triggering a systemic hormonal response (low PTH and high calcitonin) that suppresses widespread bone loss, AIC establishes an environment heavily weighted toward growth. Within this quieted landscape, locally activated BMP-2 signaling efficiently channels ionic calcium into the bone matrix, ensuring that newly deposited minerals are effectively retained and integrated into strong skeletal architecture.

Overcoming the Natural Deterioration of Age

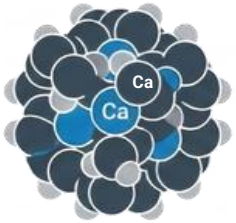


Efficacy was observed across all age groups equally. The downward trajectory of aging does not limit the bone-building capacity of AIC intervention.

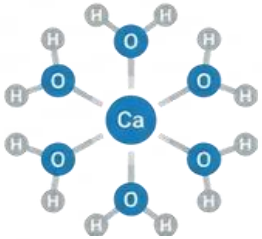


The AIC Innovation: Weak Sigma Anti-Bonding

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

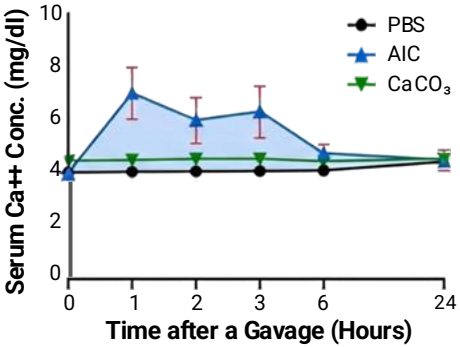
Structurally modified to alter energetic barriers and electron orbital states.

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++**

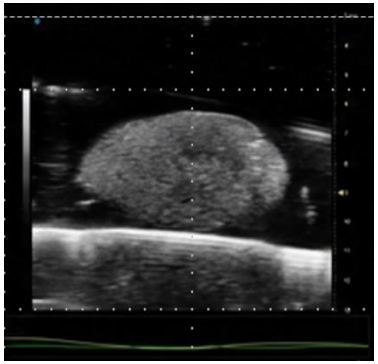


Long-Term Maintenance

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

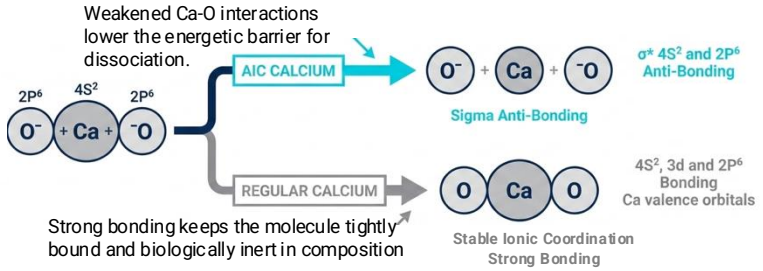
Key AIC Triggers

- PTH inhibition
- BMP-2 Activation
- Calcitonin Secretion

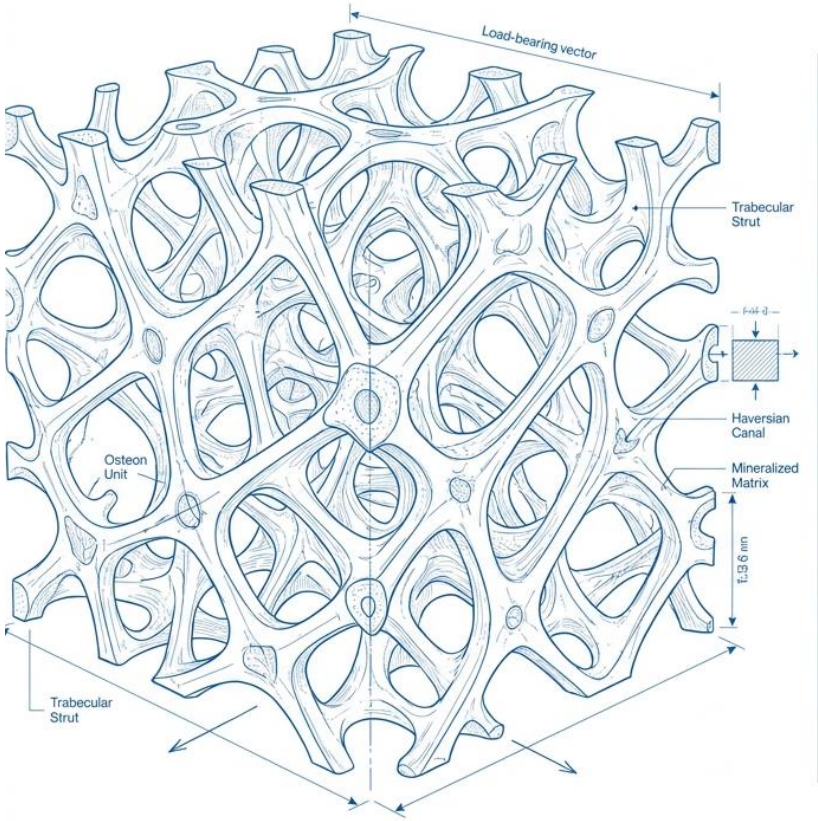


Elevated urinary calcium is safely excreted. *Ex vivo* ultrasound confirms absolutely zero crystallization or kidney stone formation after prolonged AIC regimens.

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.



Reversing Bone Loss Through Cellular Homeostasis

The biological mechanism and clinical efficacy of Antiorbital Ionic Calcium (AIC) therapy in active osteoporosis reversal.

A comprehensive decoding of the PTH, Calcitonin, and BMP-2 pathways driving a paradigm shift from bone preservation to net-positive osteogenesis.

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

To study more research and clinical cases of AIC therapy.

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