

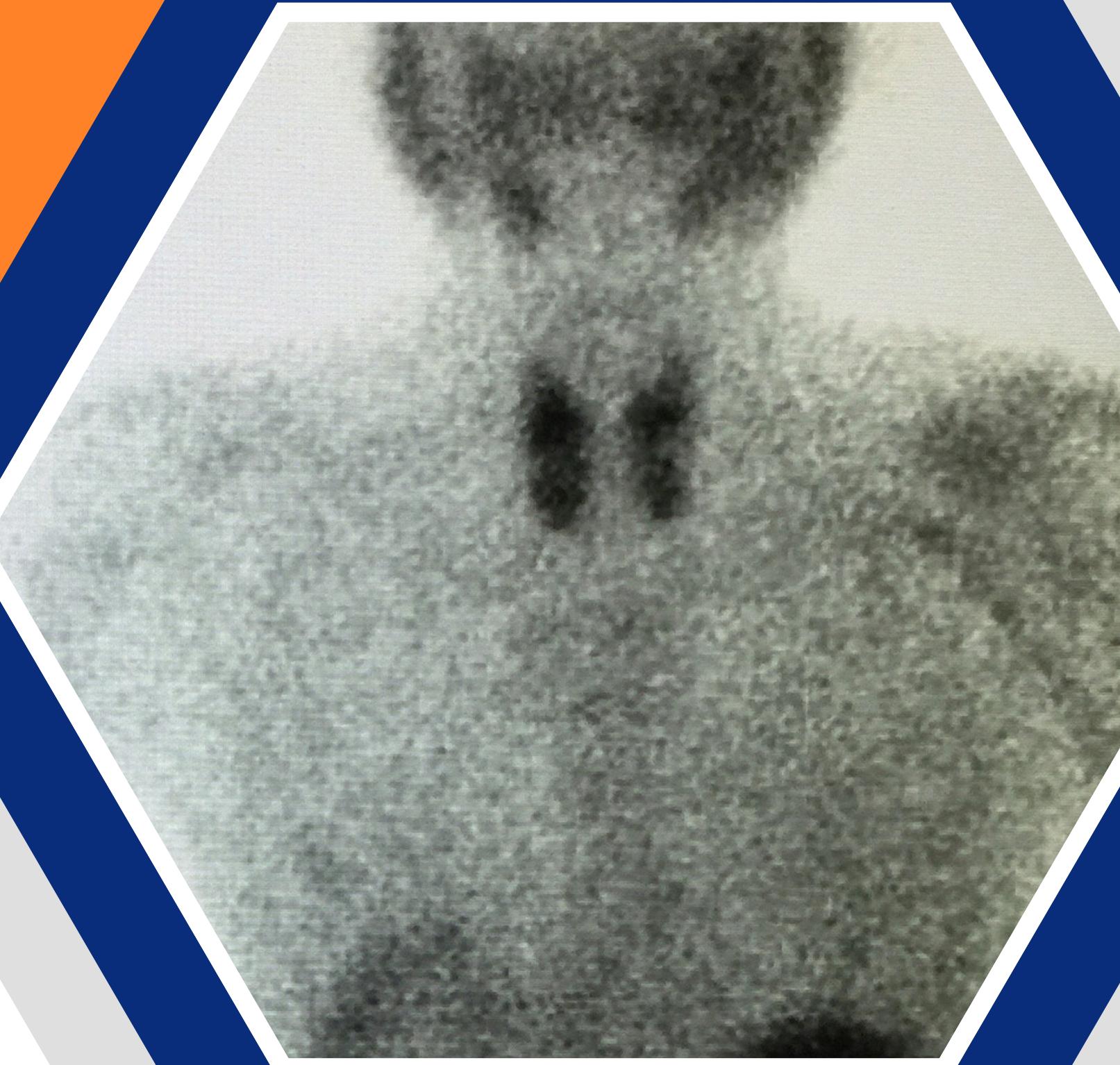
HYPERPARATHYROIDISM

NUTRITION IMPAIRMENT

Sinee Disthabanchong

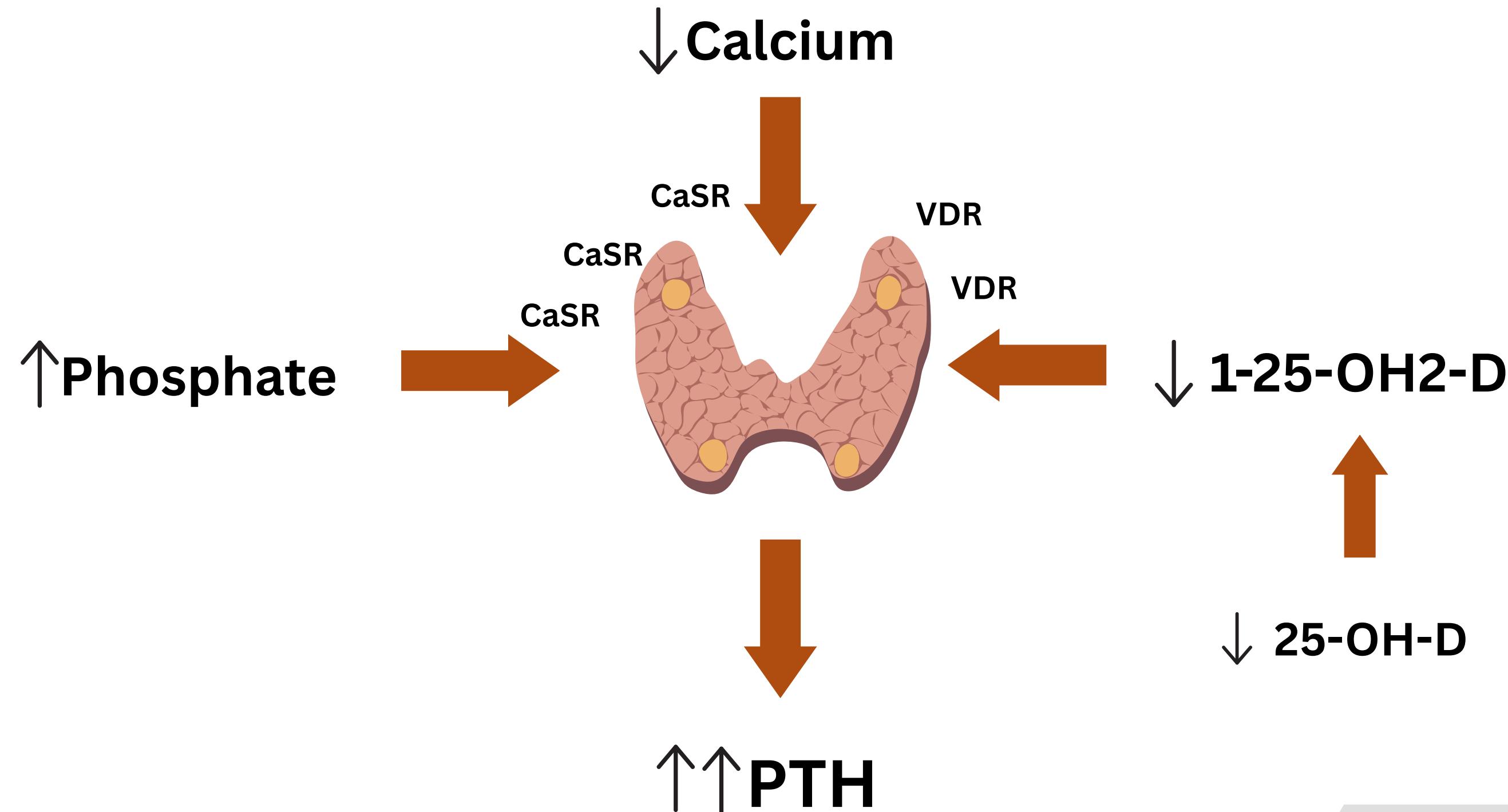
Faculty of Medicine, Ramathibodi Hospital
Mahidol University, Bangkok, Thailand

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⋮⋮⋮⋮



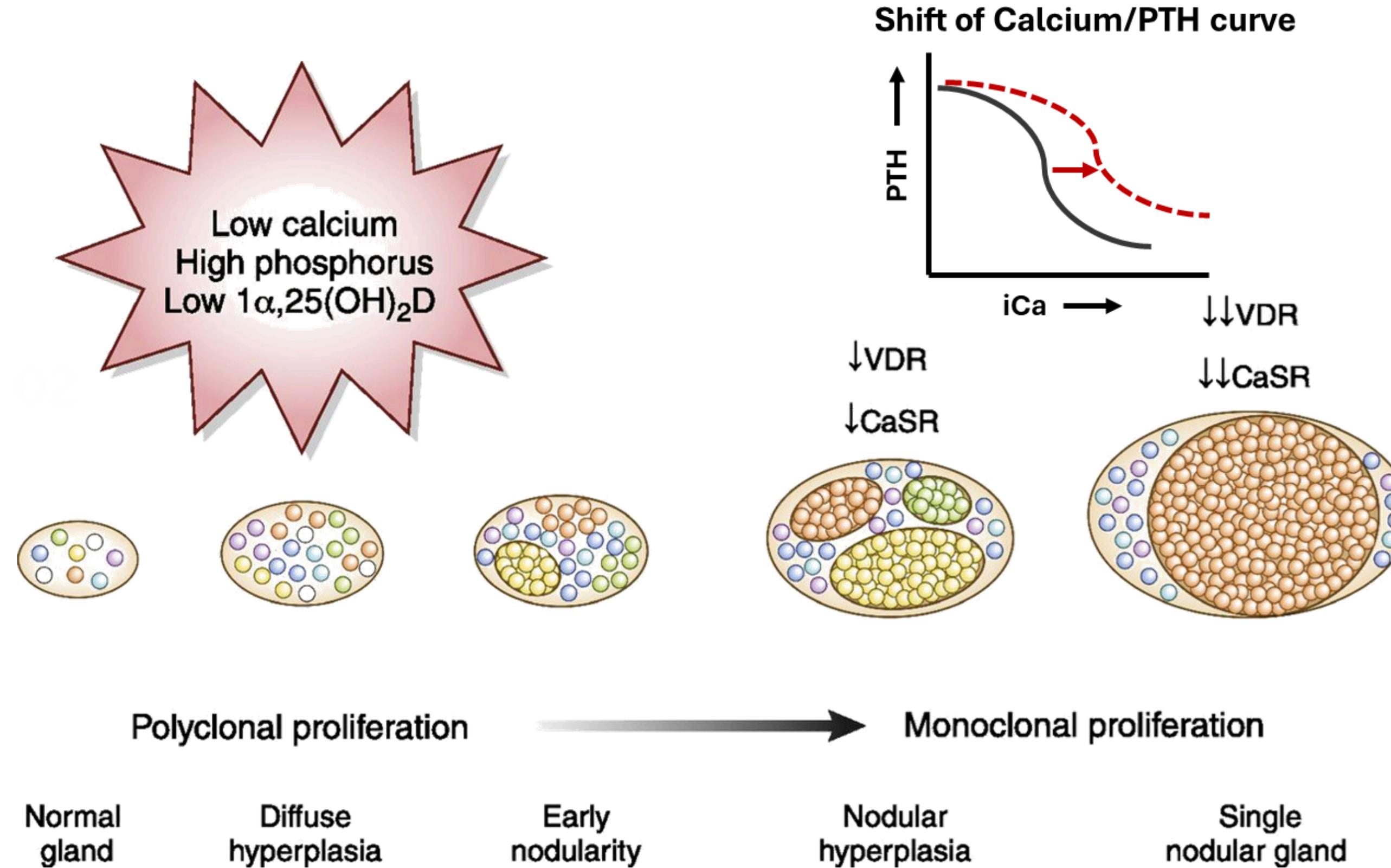


REGULATORS OF PTH SECRETION

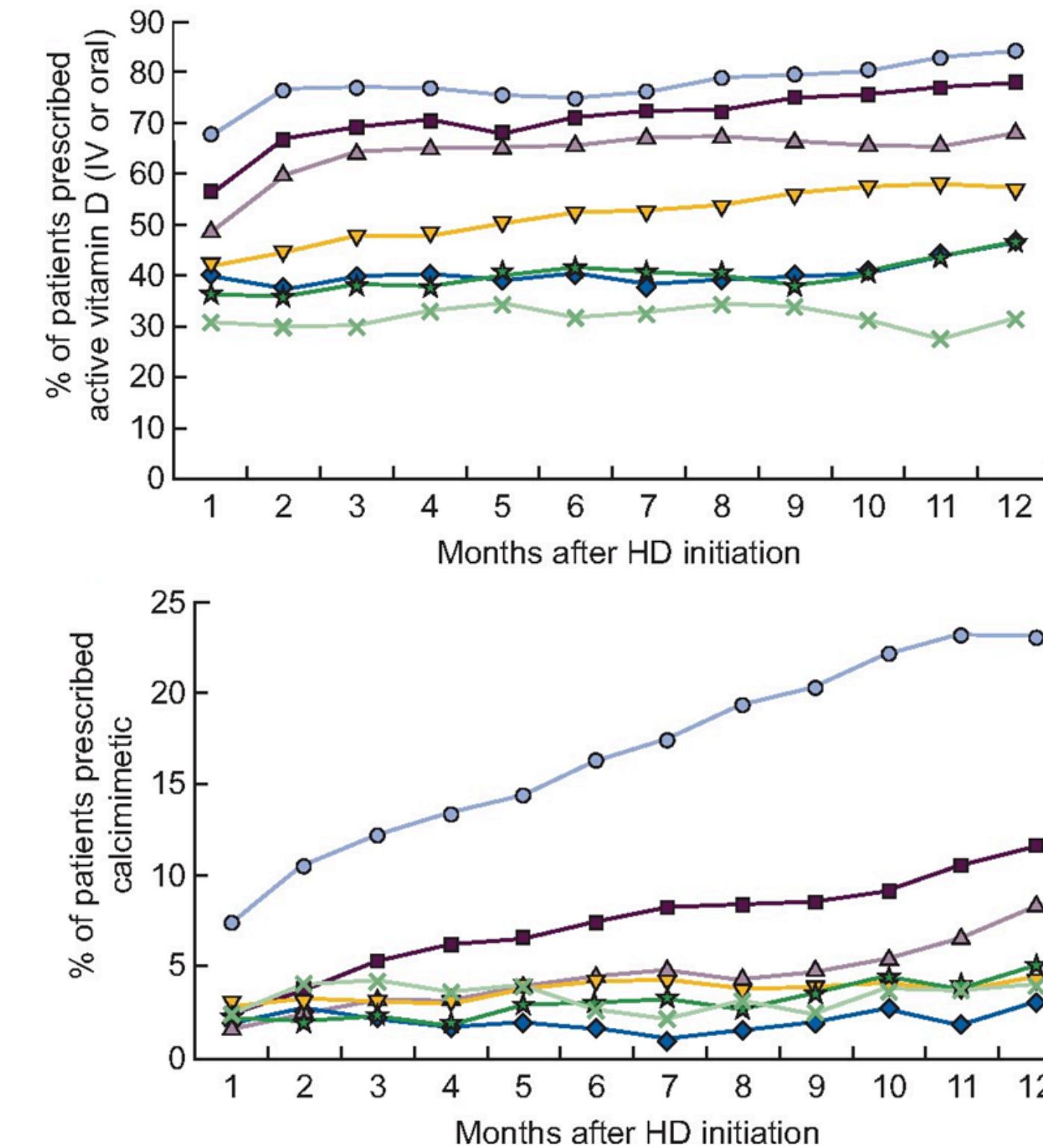
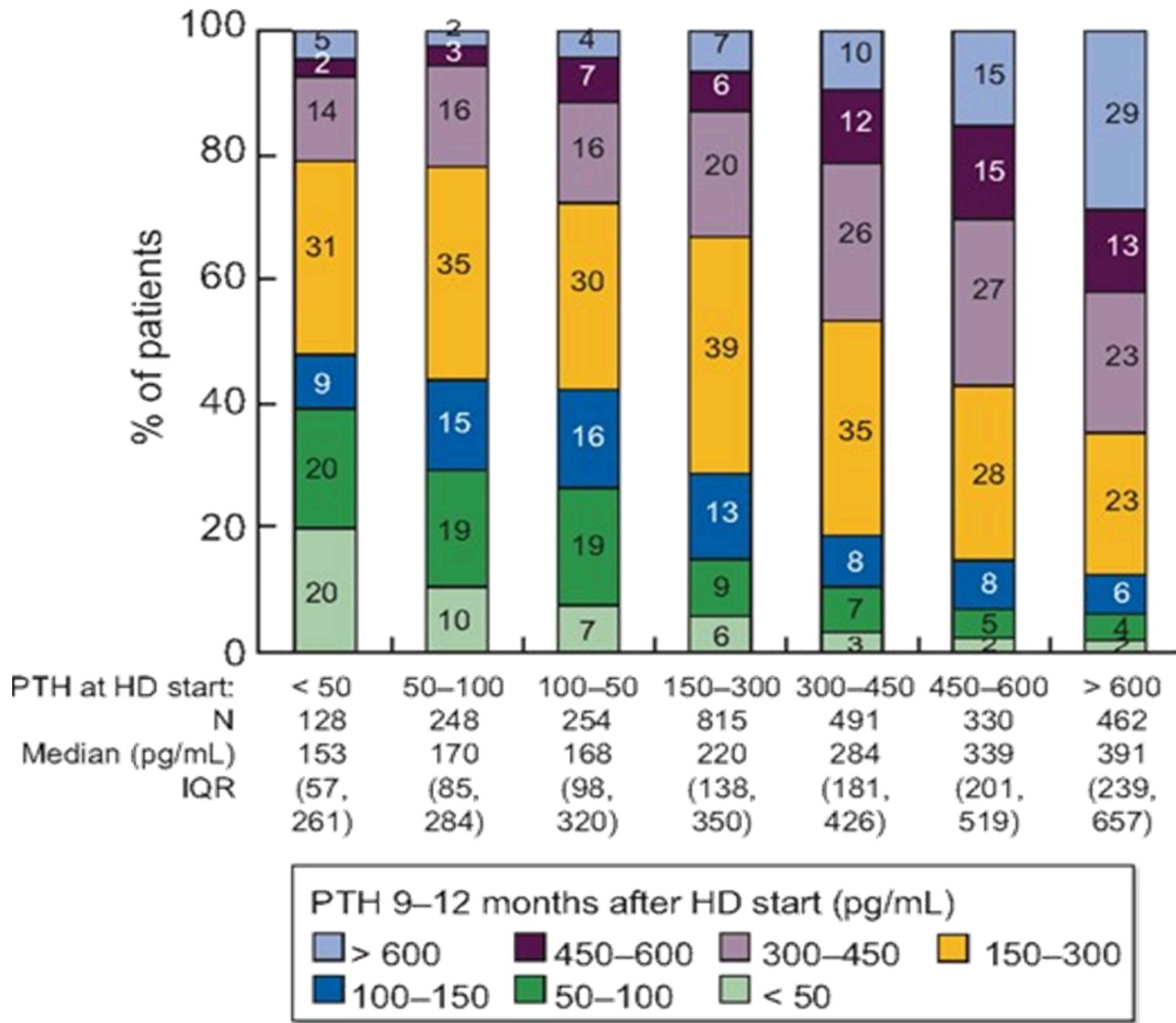




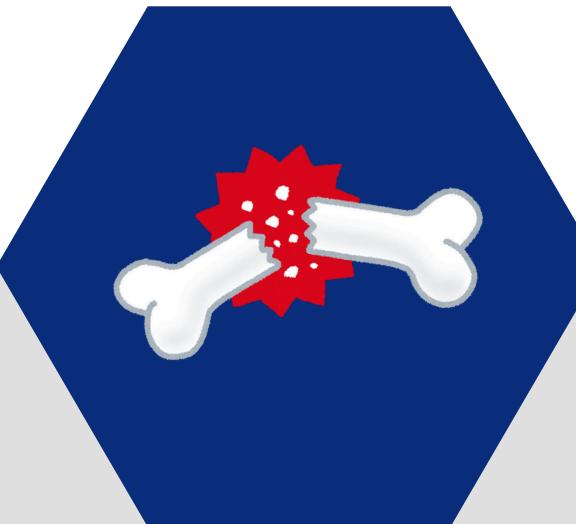
PROGRESSION OF HYPERPARATHYROIDISM



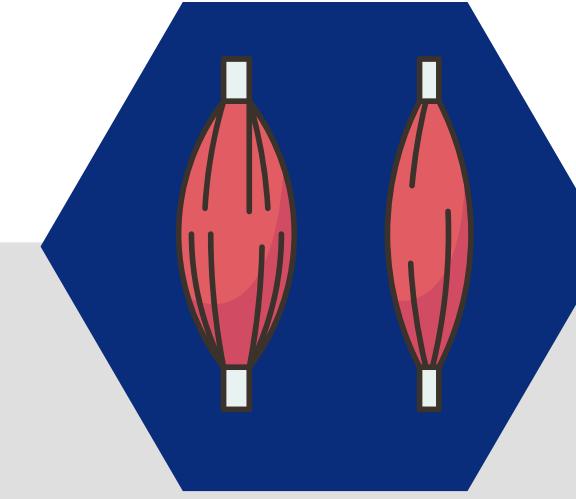
PROGRESSION OF HPT BASED ON BASELINE PTH LEVELS AT HEMODIALYSIS INITIATION



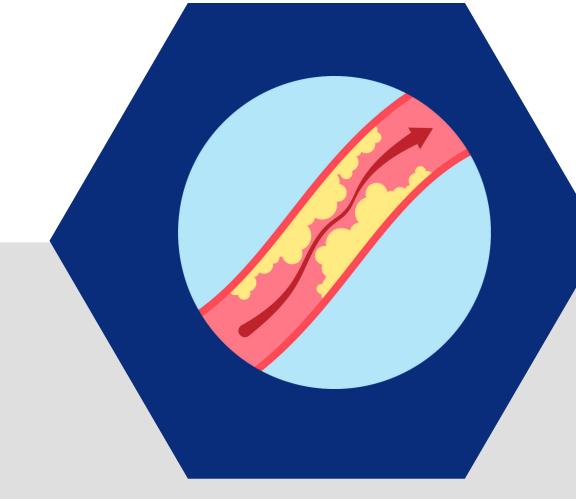
ADVERSE HEALTH EFFECTS OF HYPERPARATHYROIDISM



**Bone Loss
Fracture**



**Protein Energy
Wasting
Weight Loss**



**Vascular and
Soft Tissue
Calcification**

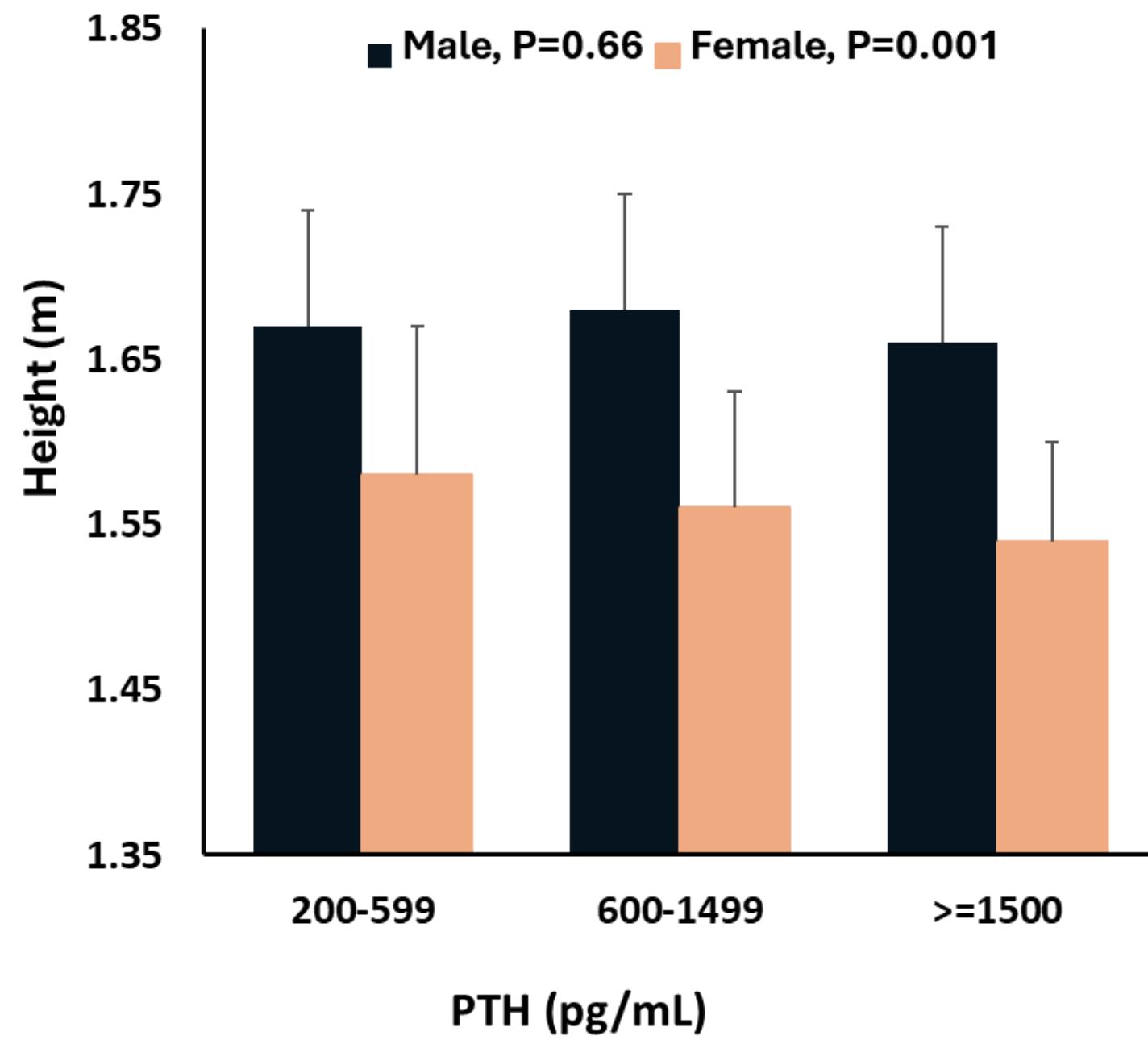


Mortality

BONE LOSS AND PROTEIN-ENERGY WASTING

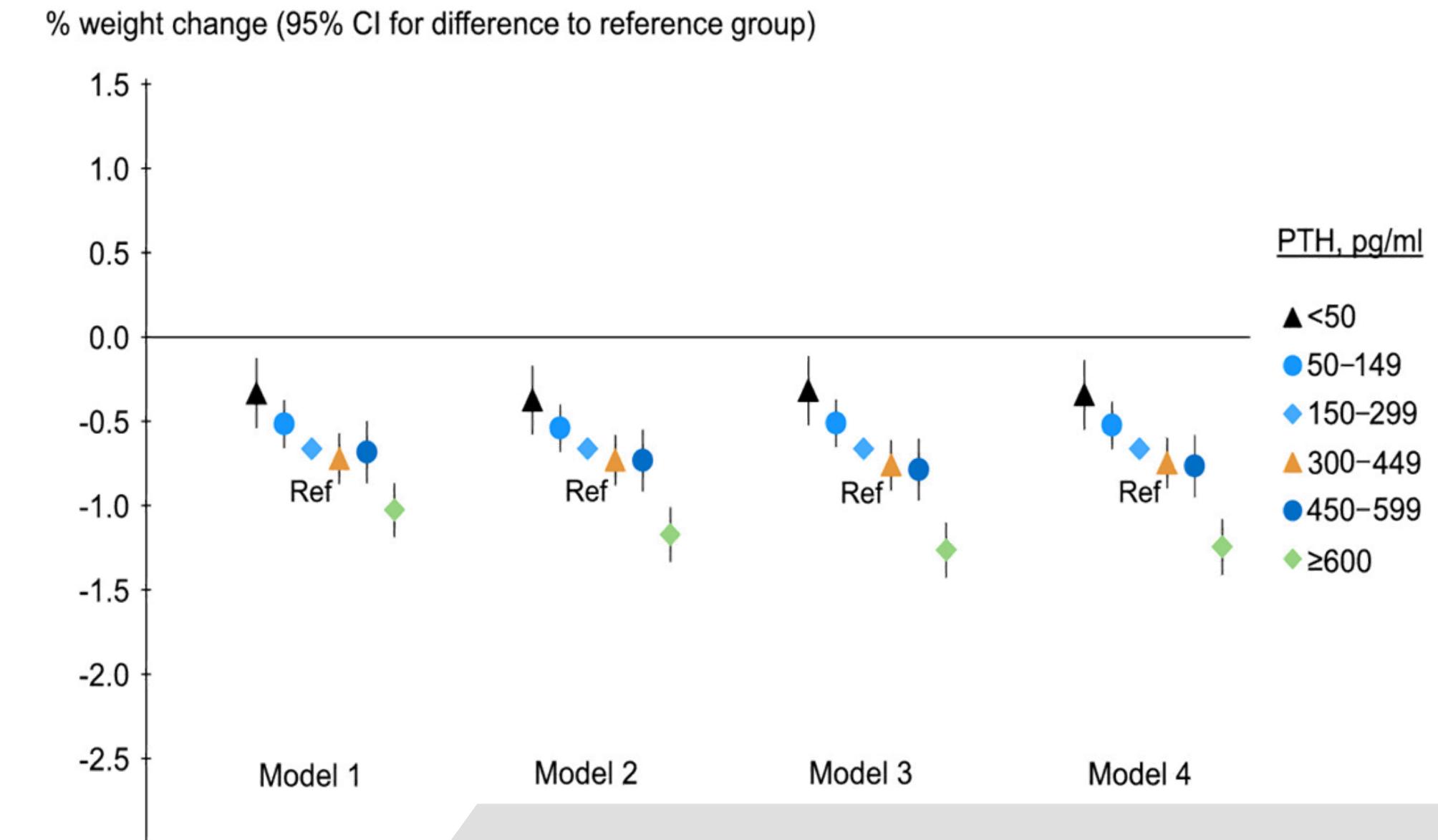
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BONE AND HEIGHT LOSS

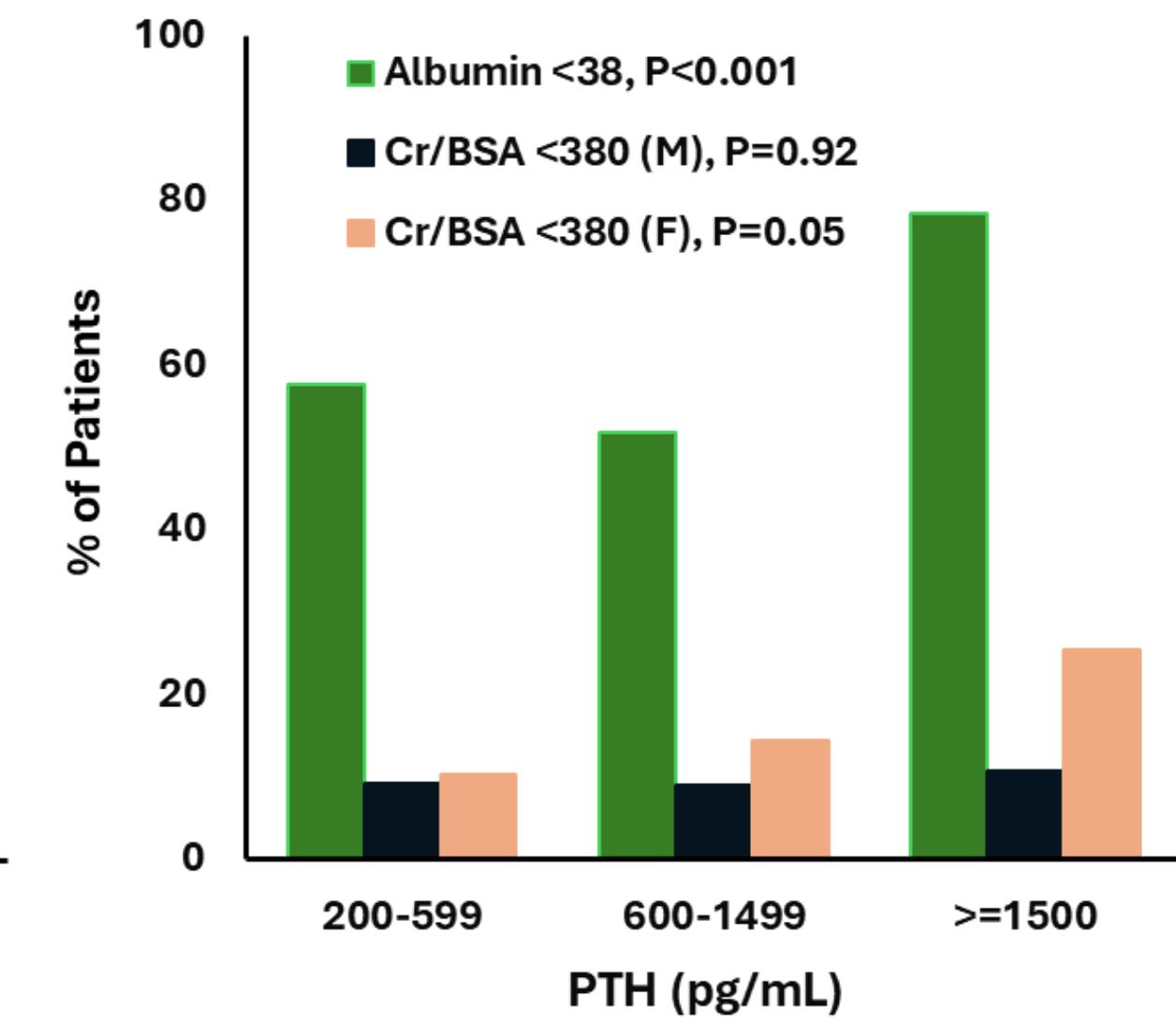
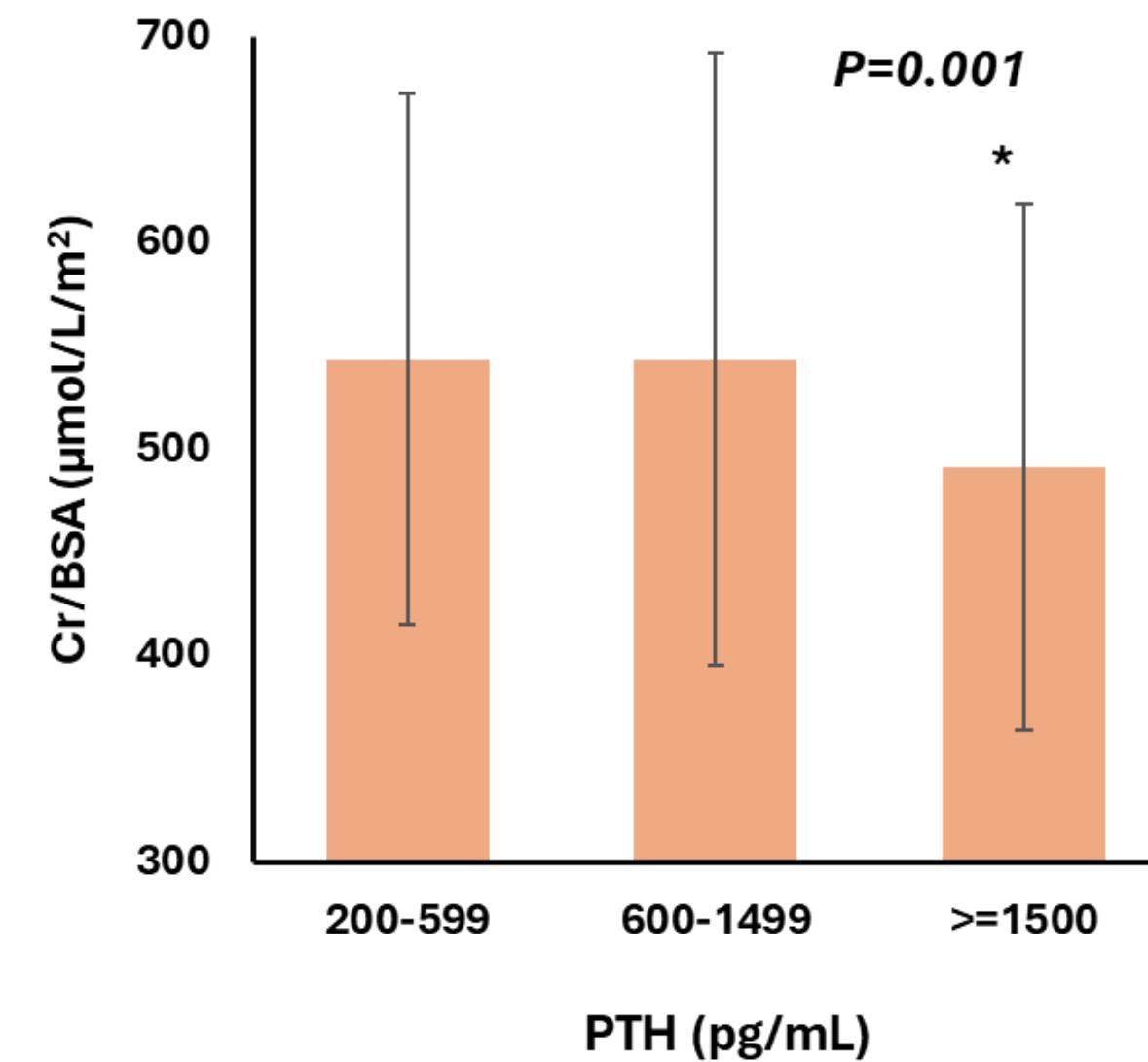
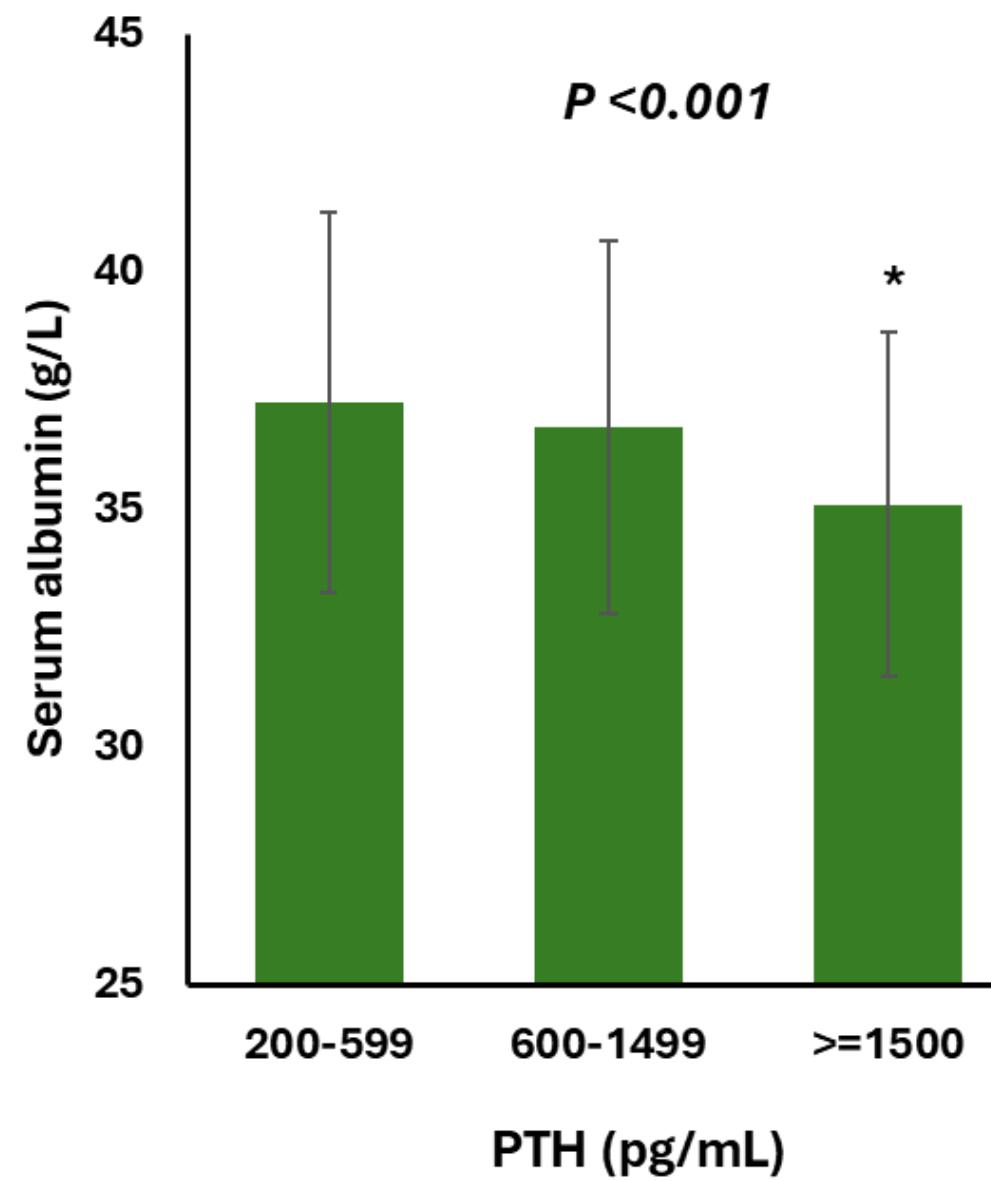


WEIGHT LOSS

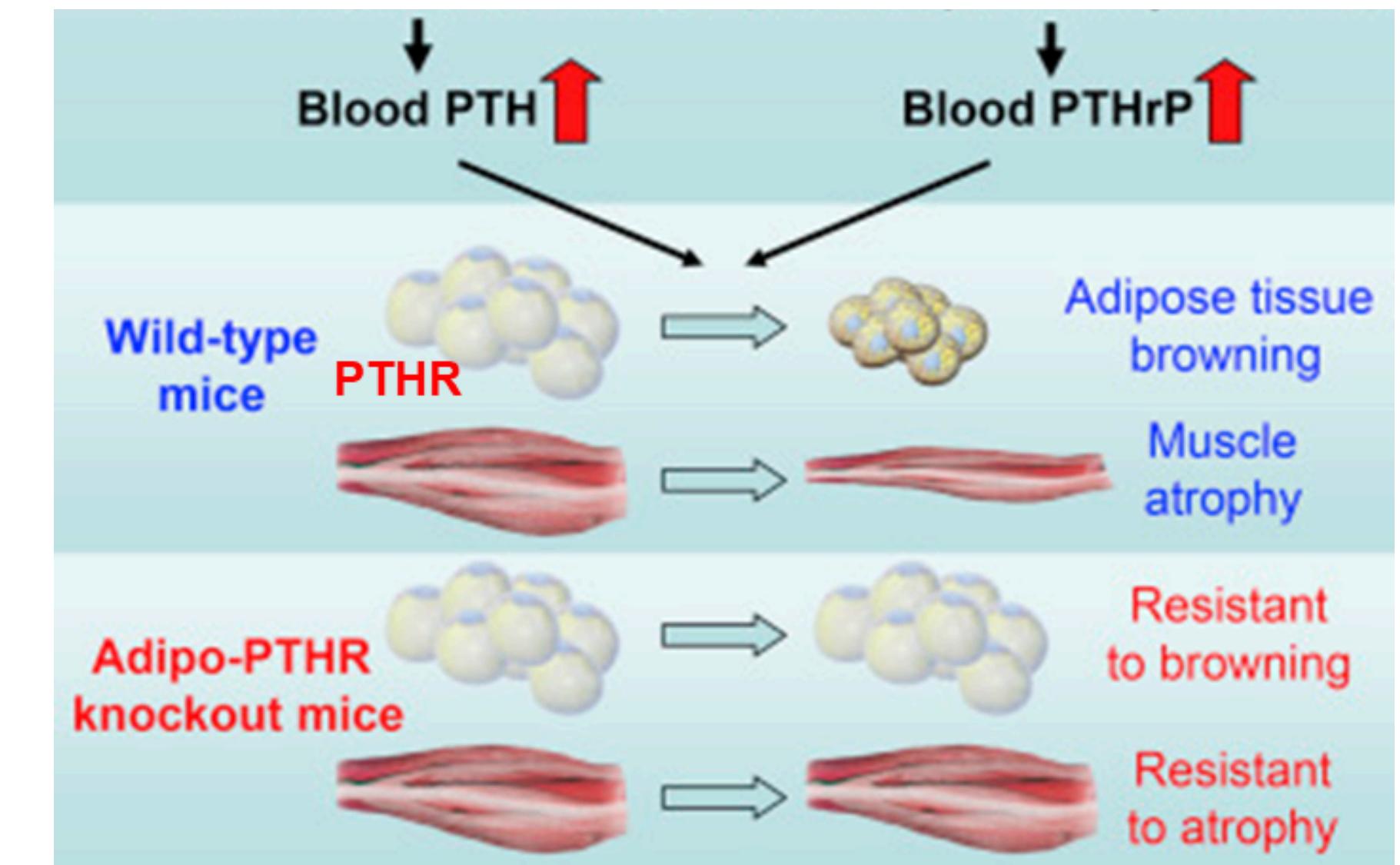
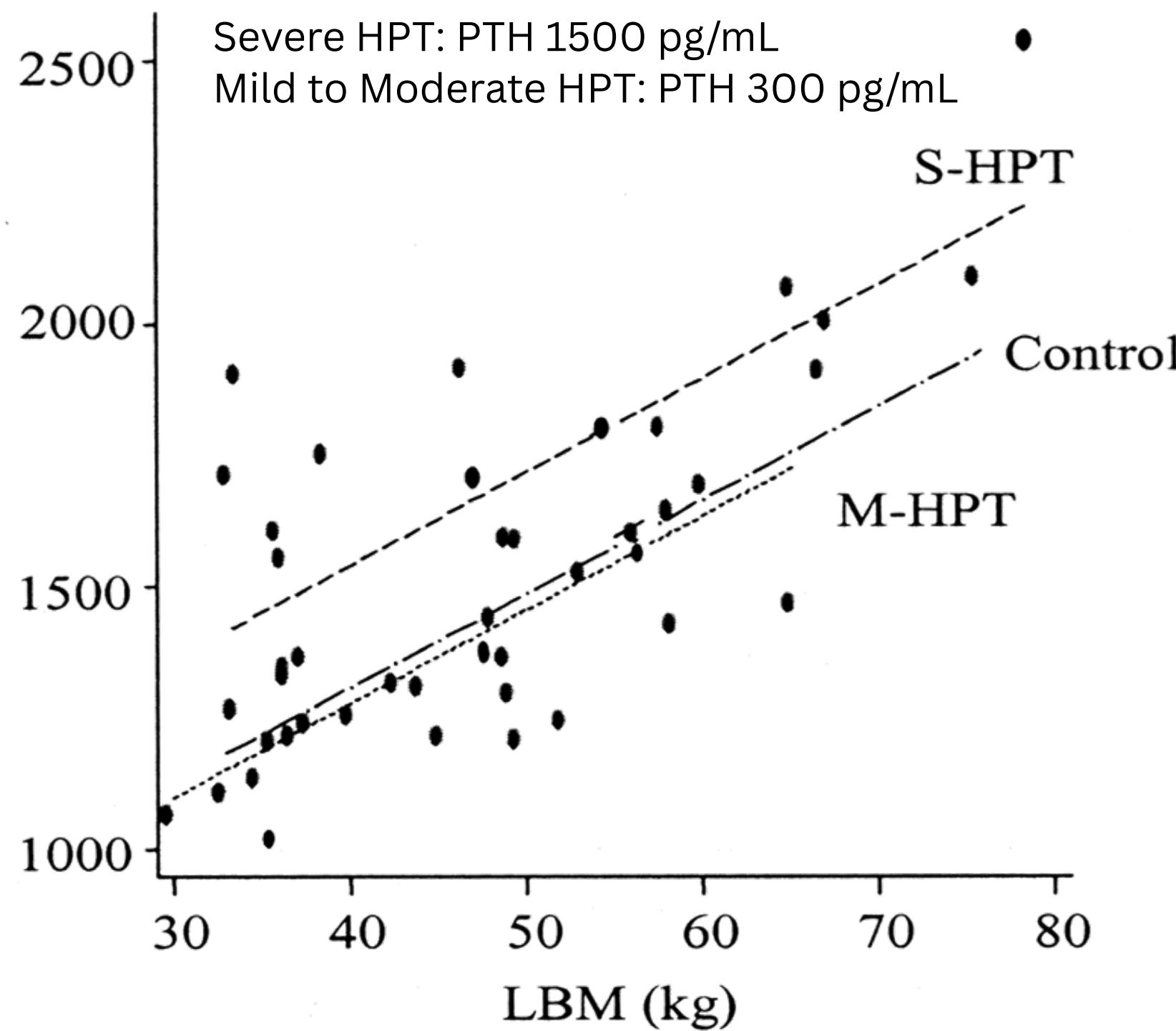
42,319 chronic HD patients from DOPPs phases 2–6
(2002–2018)



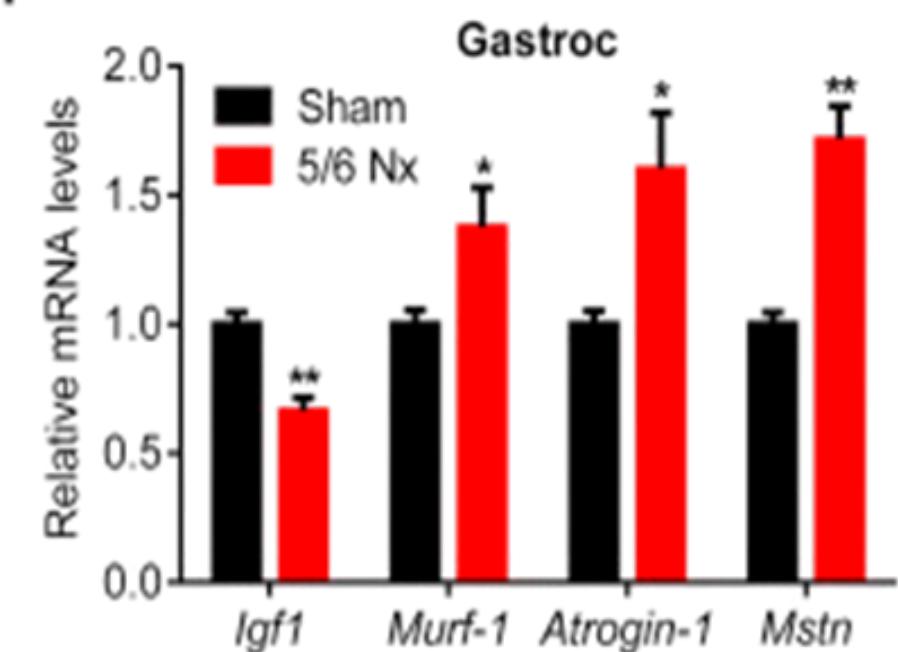
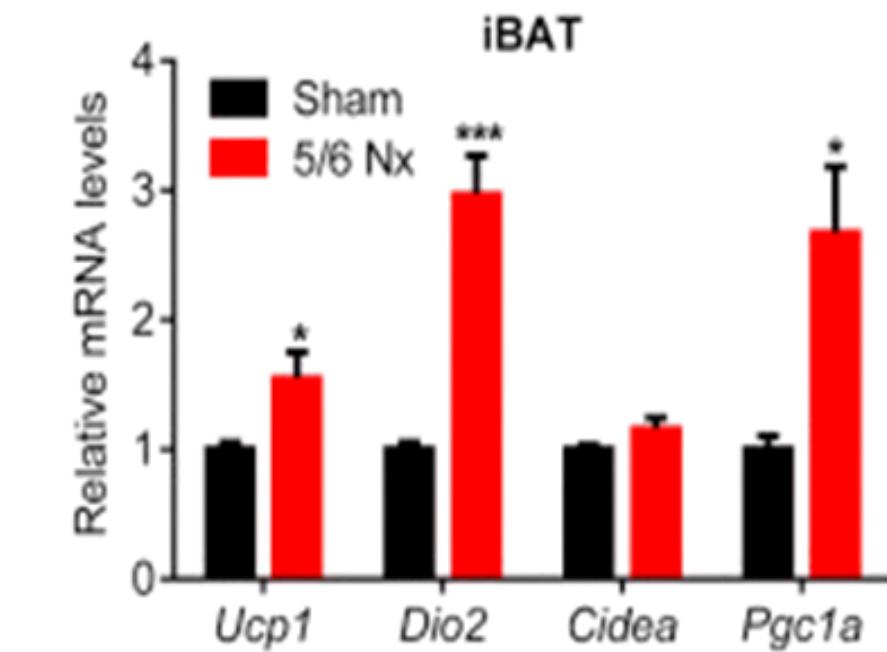
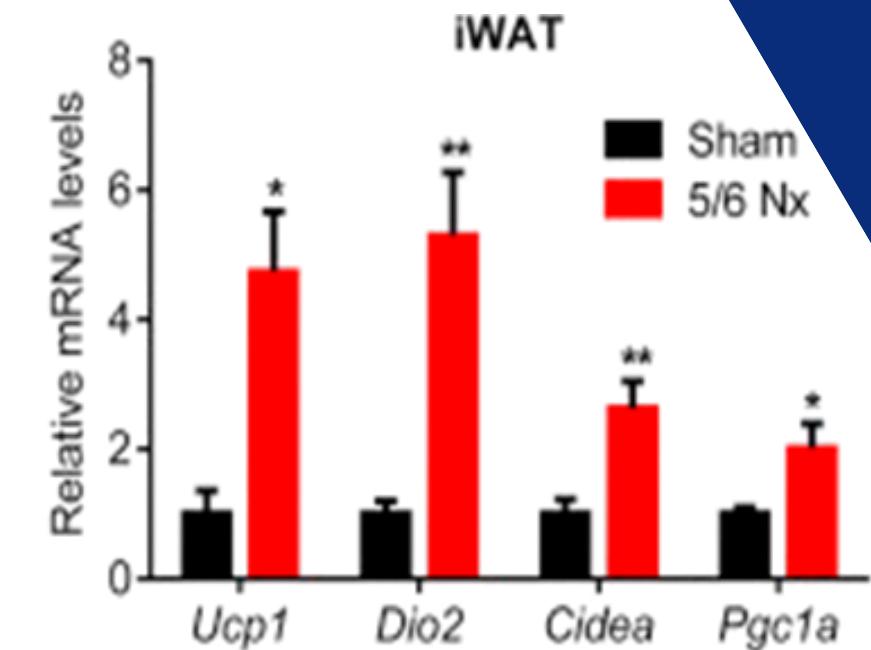
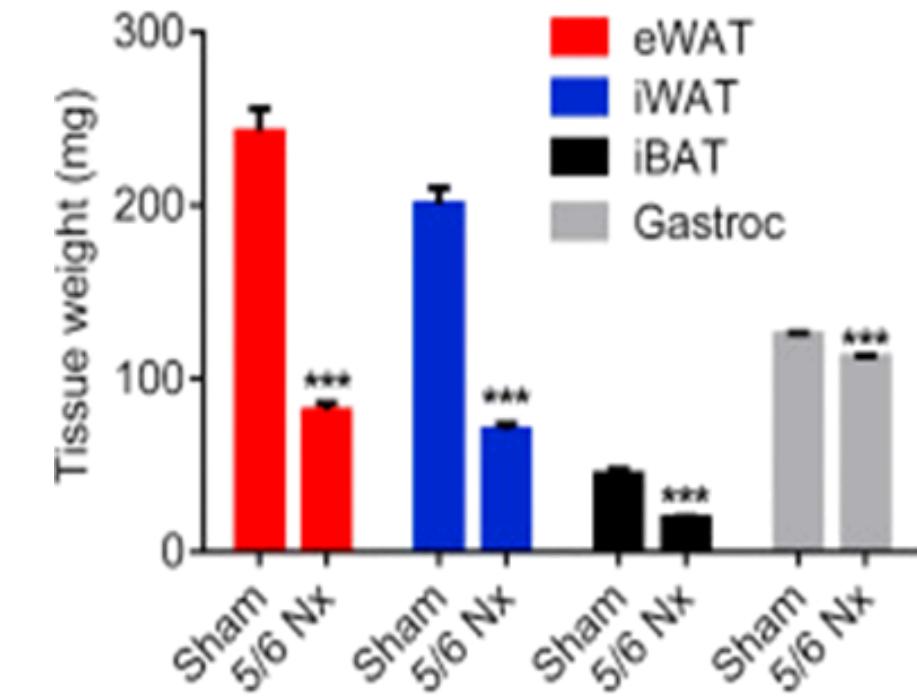
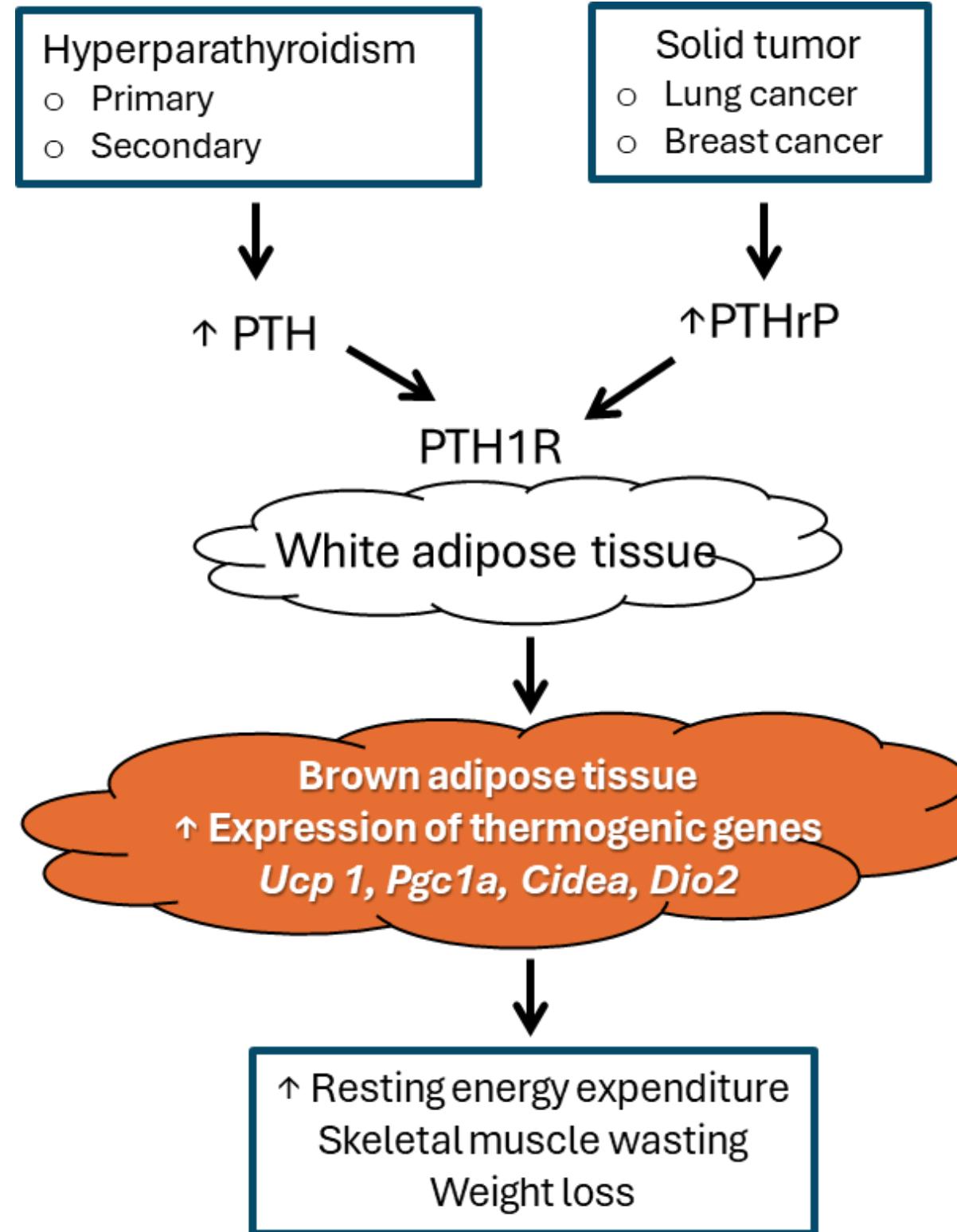
PROTEIN-ENERGY WASTING



INCREASED RESTING ENERGY EXPENDITURE IN HYPERPARATHYROIDISM



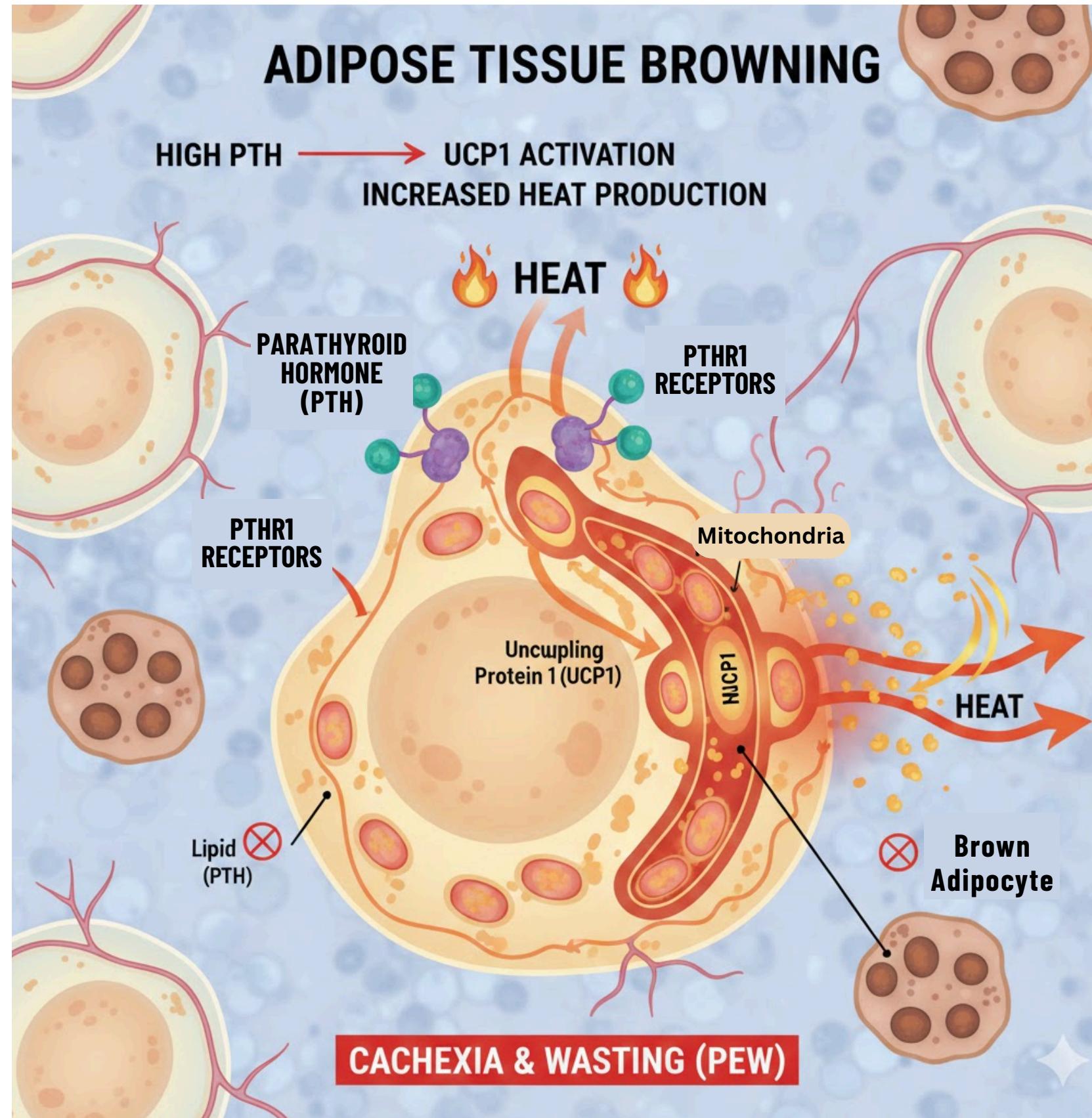
ADIPOSE TISSUE BROWNING



eWAT: Epidymal white adipose tissue (Visceral fat)
iWAT: Inguinal white fat (Subcutaneous fat)
iBAT: Intercapsular brown fat
Gastro: Gastrocnemius muscle



ADIPOSE TISSUE BROWNING



White adipose tissue (WAT) is primarily for energy storage. Brown adipose tissue (BAT) are specialized fat cells that generate heat by burning energy (thermogenesis). = Browning

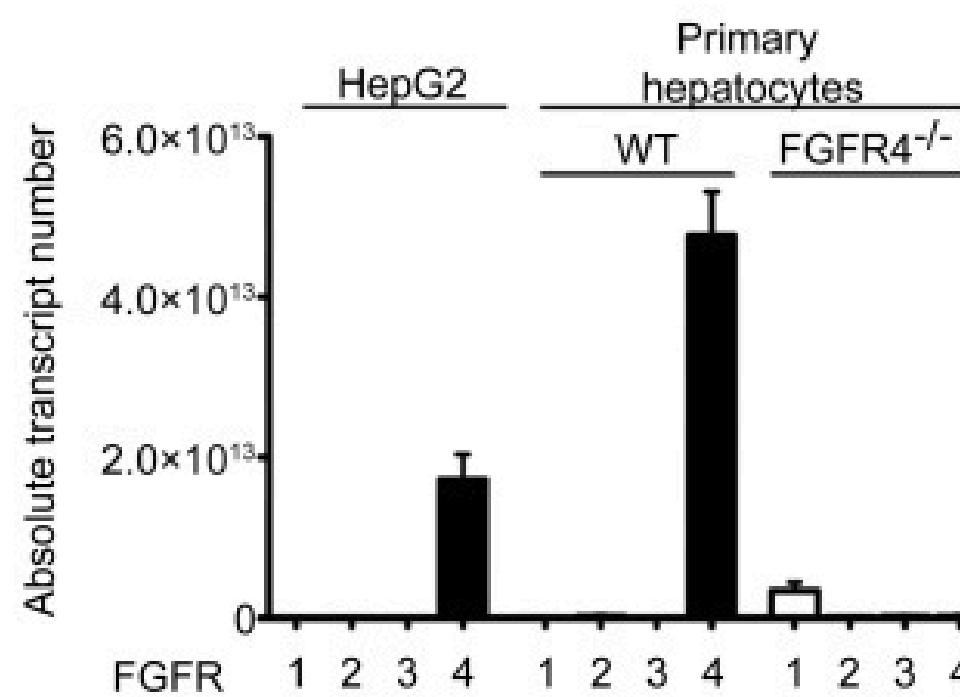
High concentrations of PTH (and related peptides like PTHrP) activate PTHR1 on the white adipocytes. This activation triggers signaling pathways that promote the expression of key thermogenic proteins, most notably Uncoupling Protein 1 (UCP1).

UCP1 uncouples the process of oxidative phosphorylation in the mitochondria, meaning the energy from food is dissipated as heat instead of being stored as ATP or converted into new fat/muscle mass.

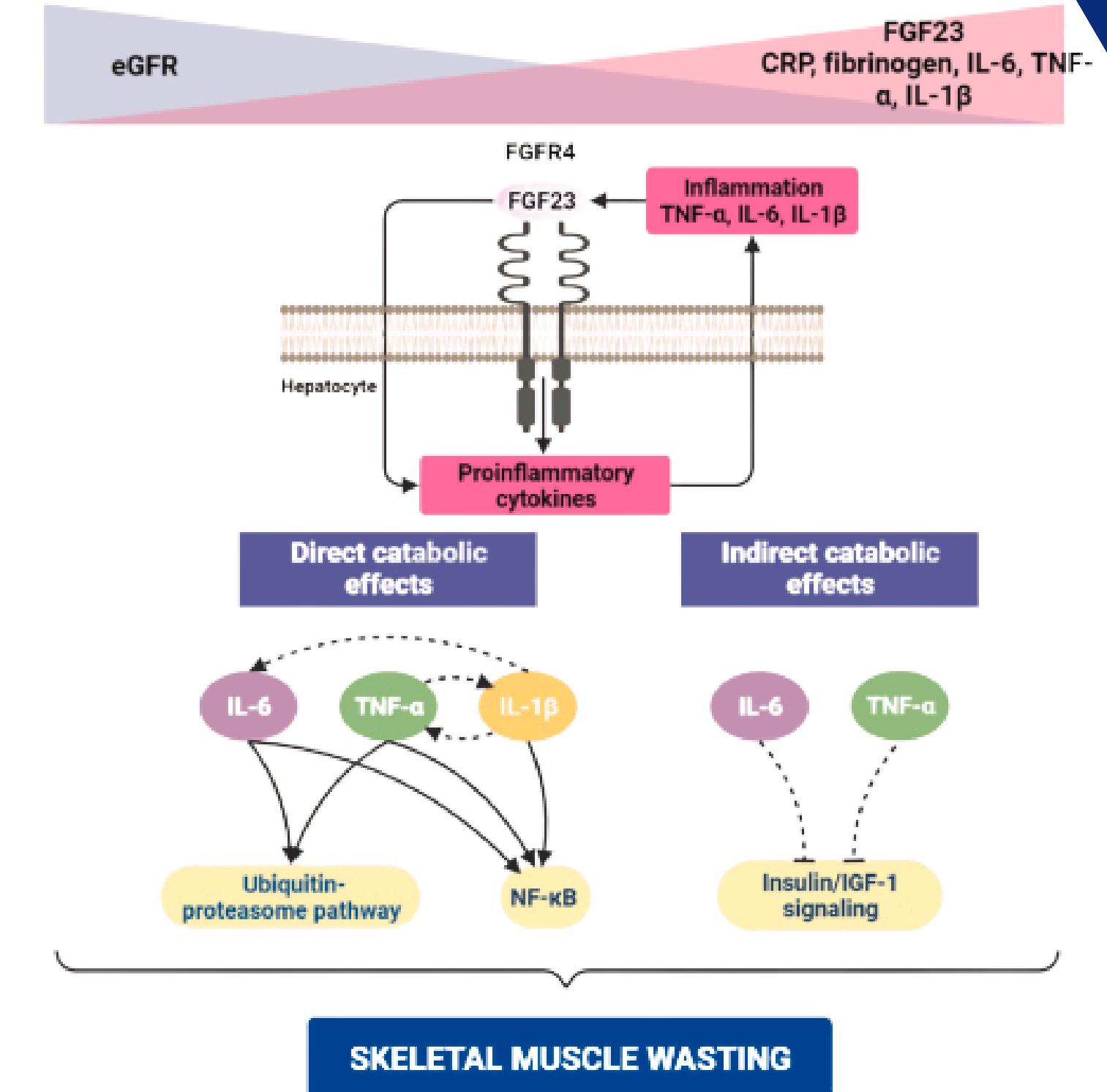
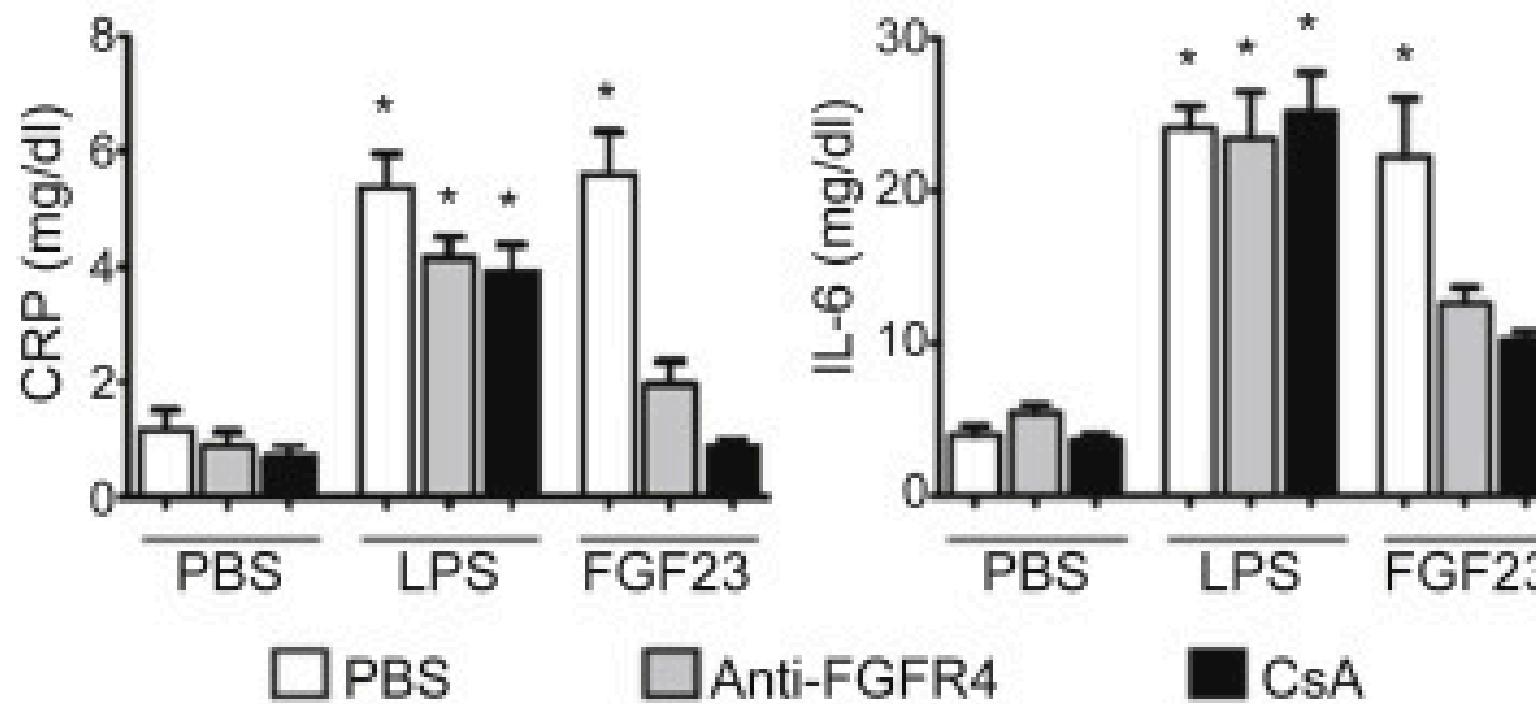
The net effect is a significant and persistent increase in the body's resting energy expenditure (REE). The body burns calories unnecessarily for heat, leading to a negative energy balance.

This high energy demand contributes directly to the breakdown of both fat and muscle stores, resulting in cachexia and wasting (PEW).

FGF-23 AND INFLAMMATION

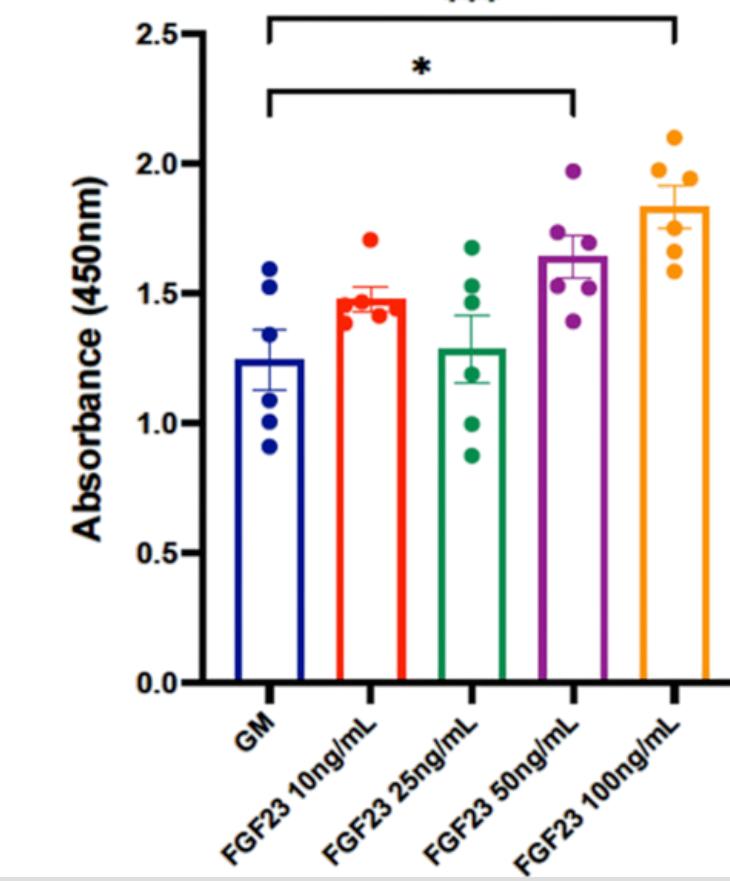
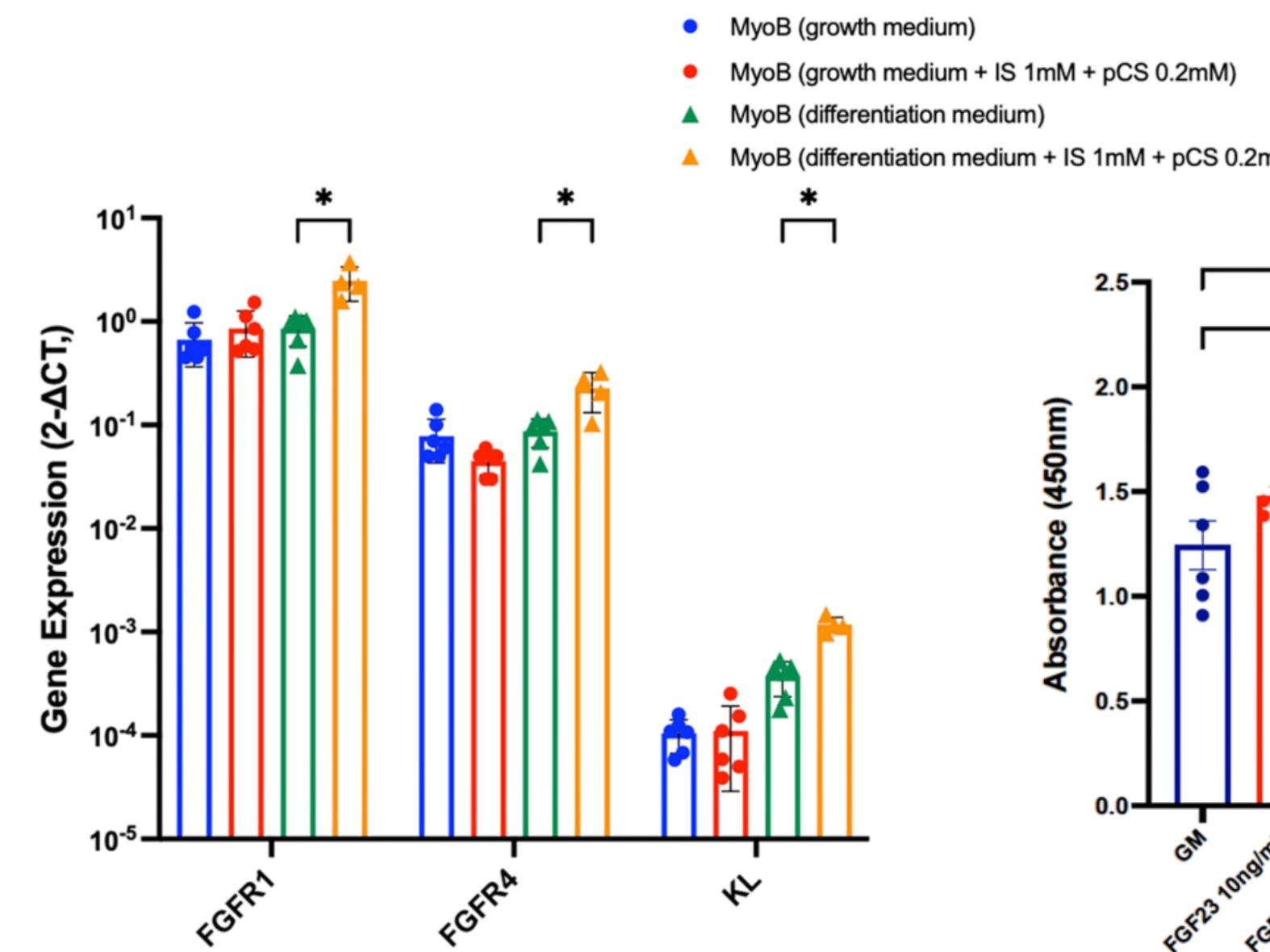
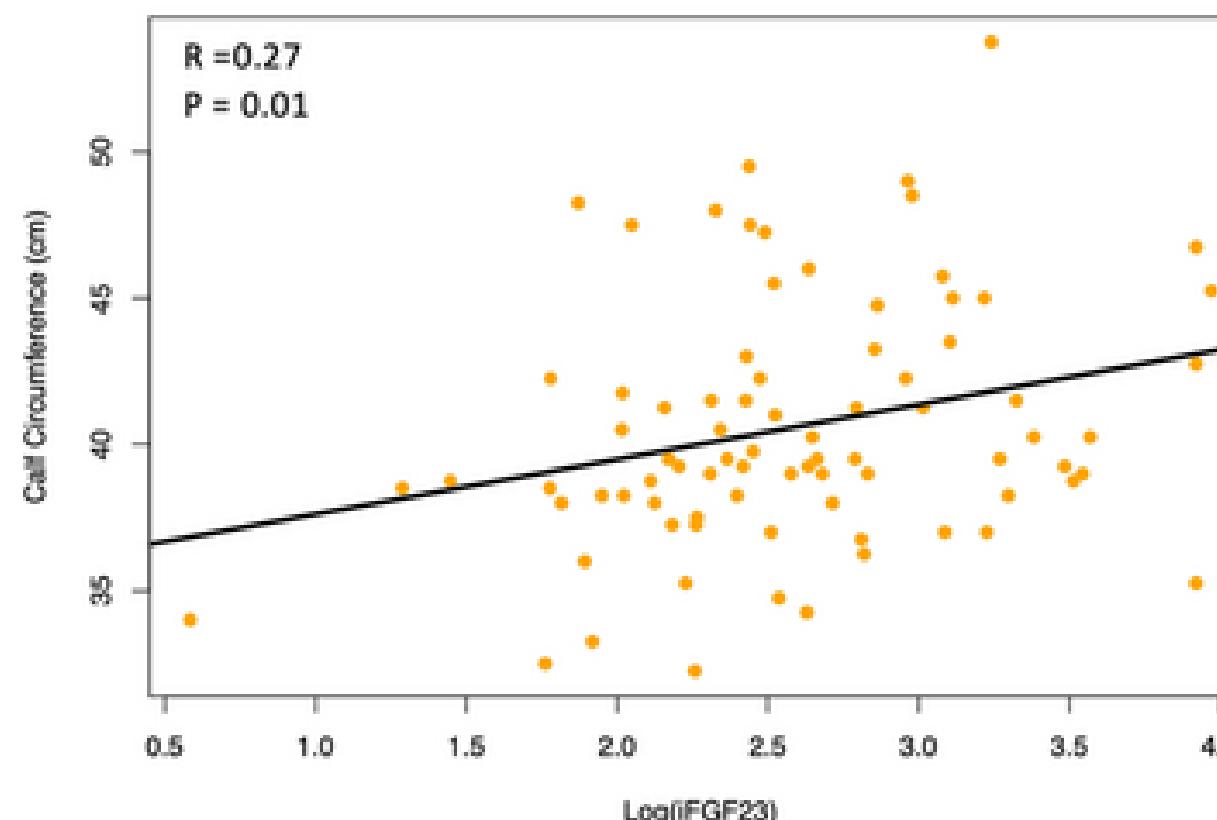
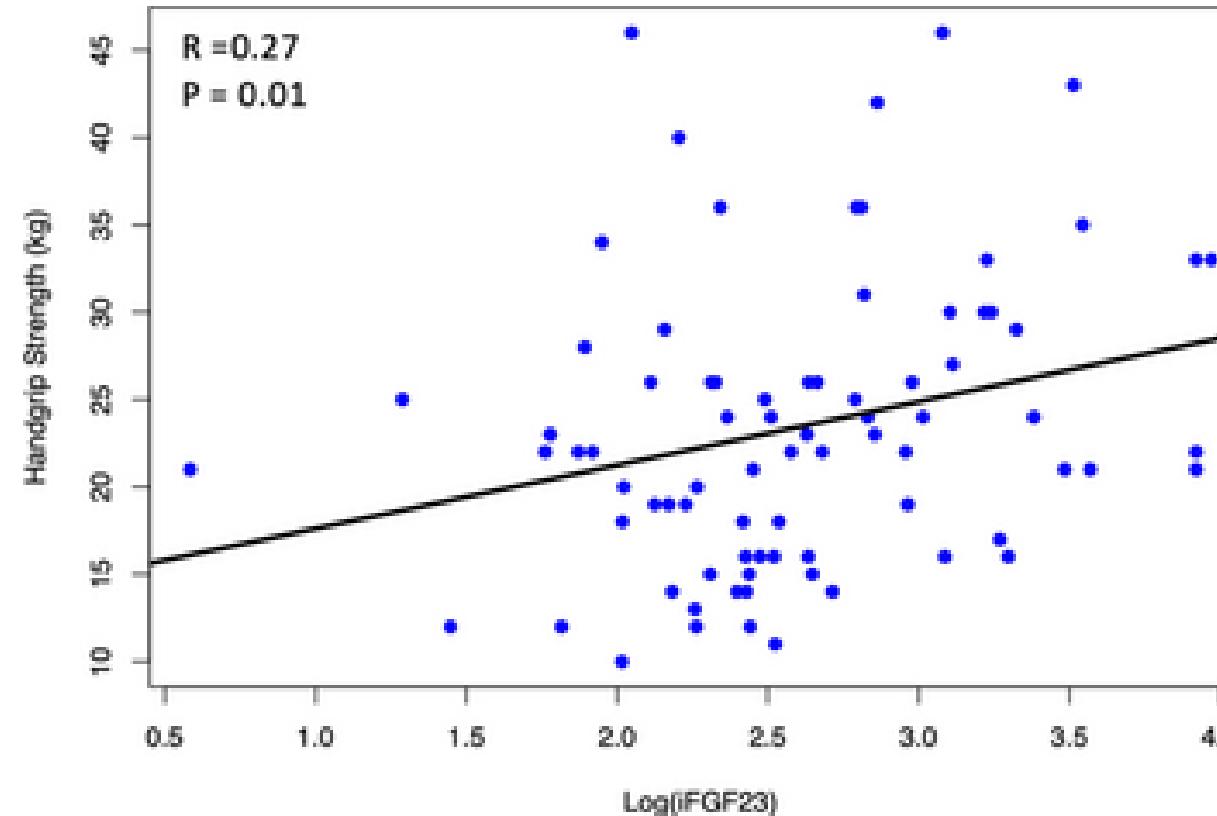


FGF23 can directly induce cytokine production from hepatocytes through activation of FGFR4 expressed on hepatocytes, through PLC-Calcineurin-NFAT pathway



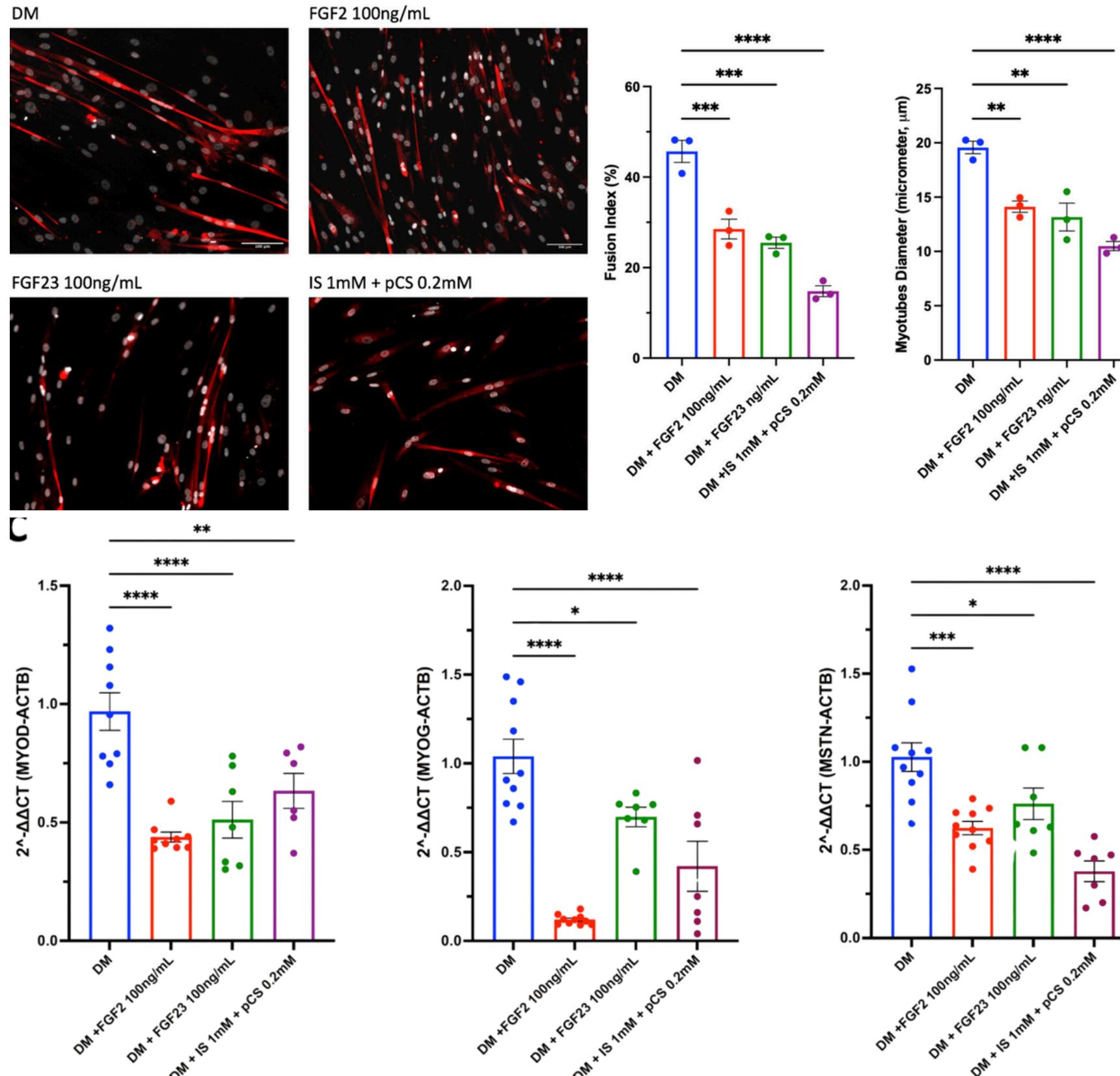
FGF-23 AND SARCOPENIA

- FGF-23 level is associated with muscle mass and strength in HD patients
- FGFR1, FGFR4, and klotho are present in adult human skeletal muscle myoblasts
- Increasing dose of FGF-23 promotes myoblast proliferation but repressing myogenic differentiation



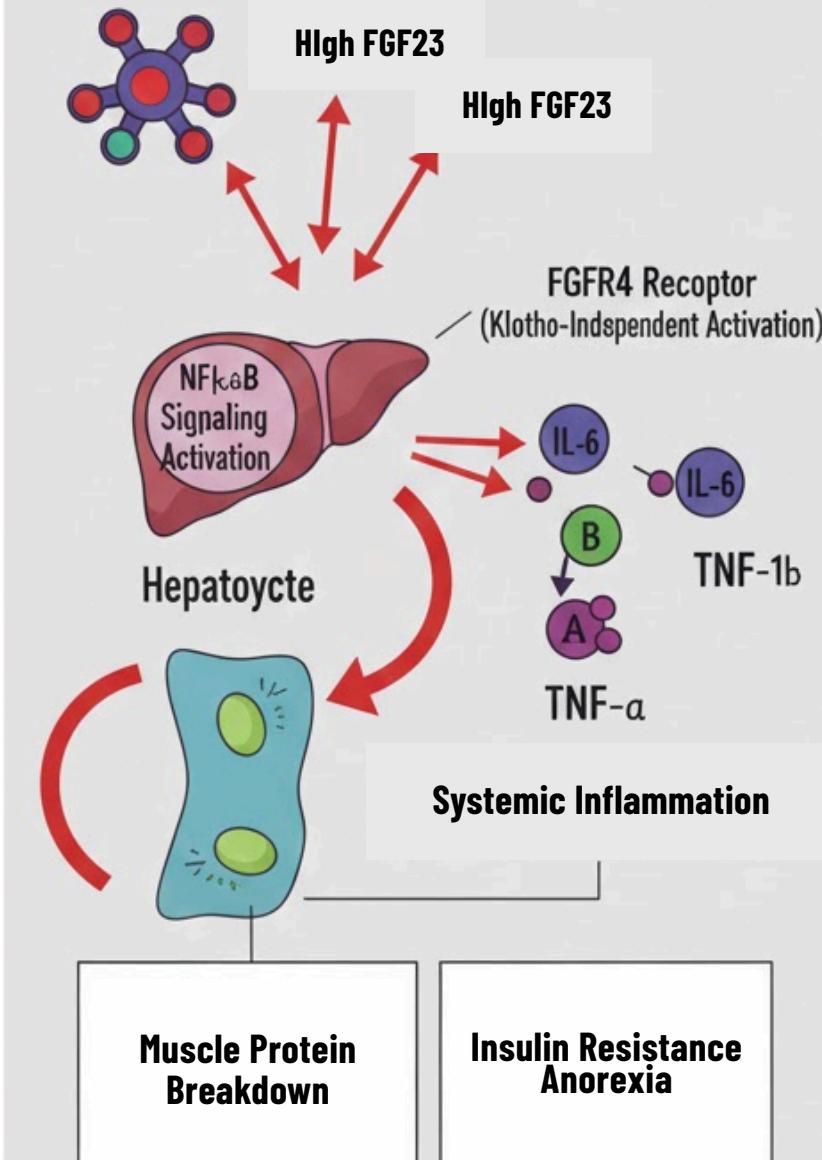
FGF-23 AND SARCOPENIA

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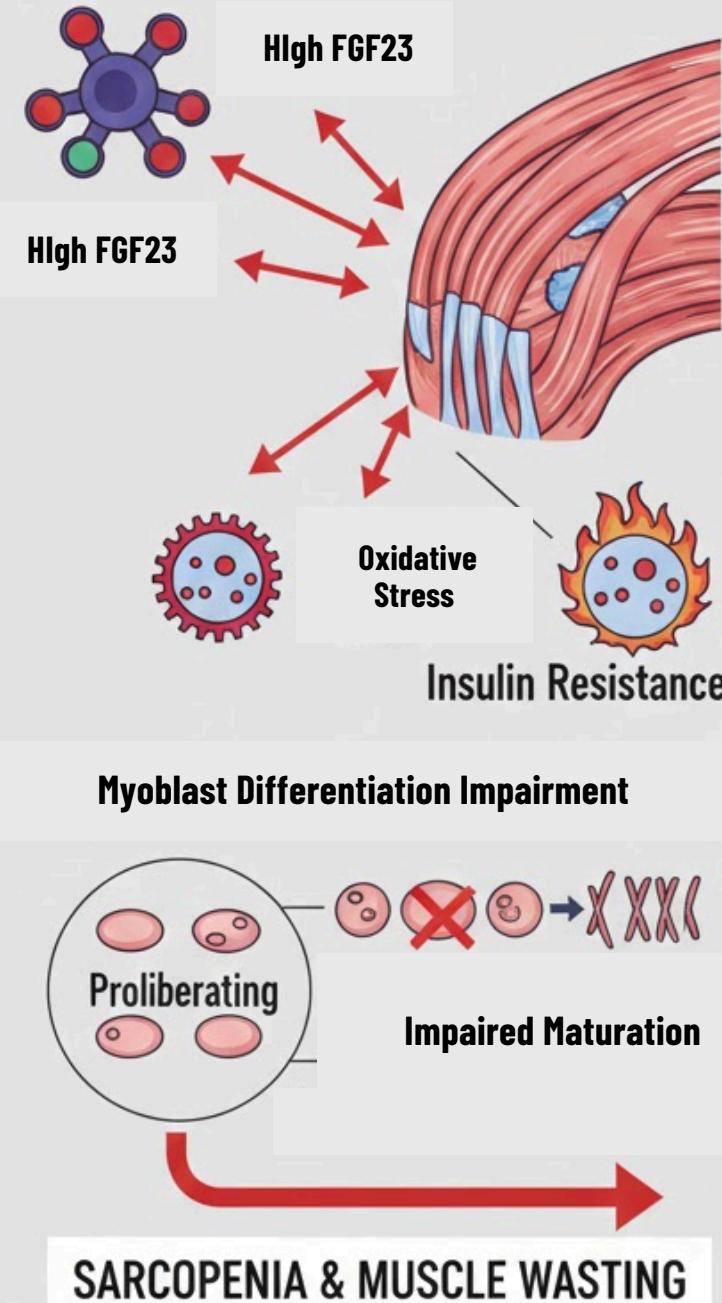


Reduced myosin heavy chain (red staining), fusion indices and myotubes diameter after FGF-23 treatment. The expressions of MYOD, MYOG and MSTN were reduced in myoblasts cultured in differentiated medium (DM) in the presence of FGF-23

1. Systemic Inflammation



2. Muscle Toxicity & Sarcopenia



HIGH FGF23 DRIVES INFLAMMATION & MUSCLE DYSFUNCTION, ACCELERATING PEW IN CKD-MBD.

Wong L. J Cachexia Sarcopenia Muscle. 2025 Jun;16(3):e13848

MANAGEMENT OF HYPERPARATHYROIDISM

MULTIMODAL TREATMENT IS
THE KEY TO EFFECTIVE
MANAGEMENT

Phosphate Restriction
Phosphate Binders

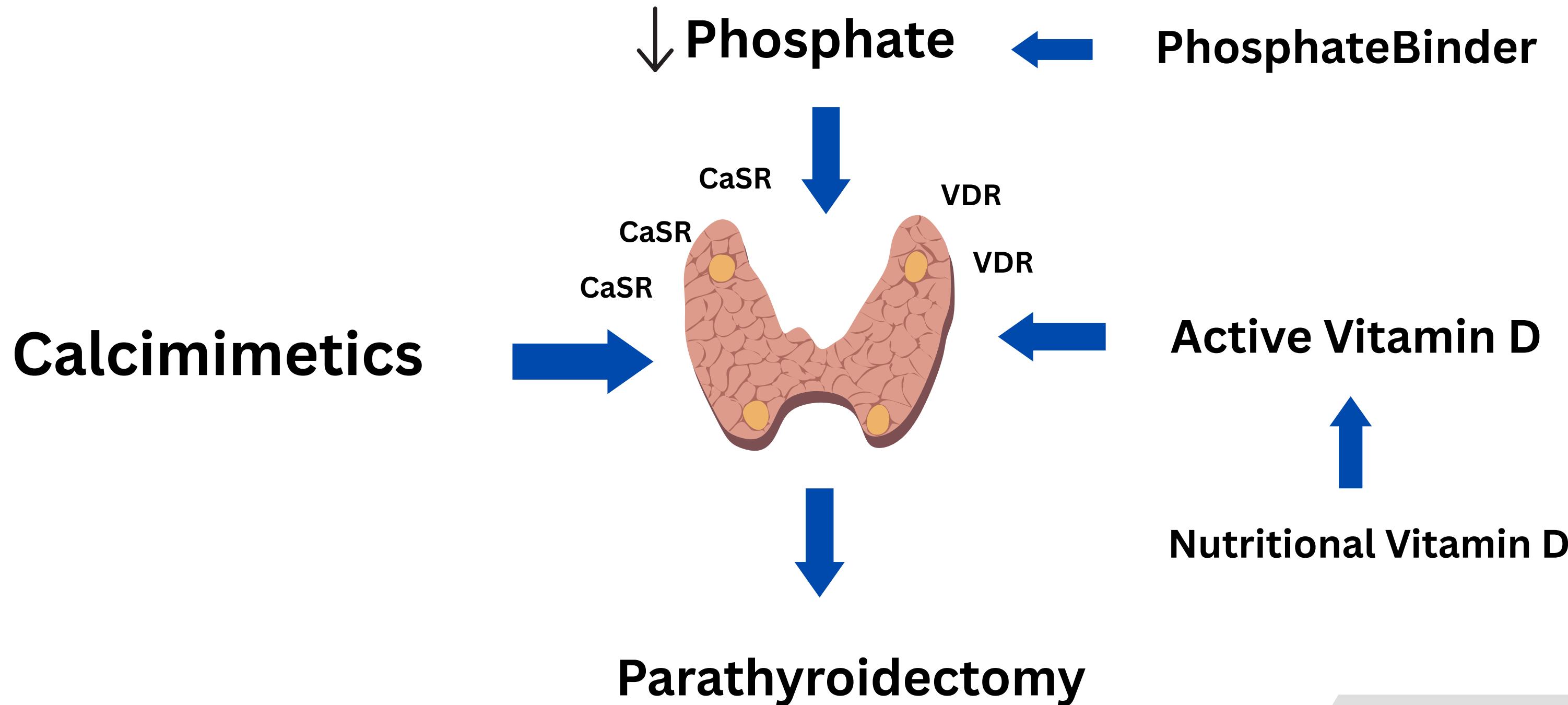
Active Vitamin D

Calcimimetics

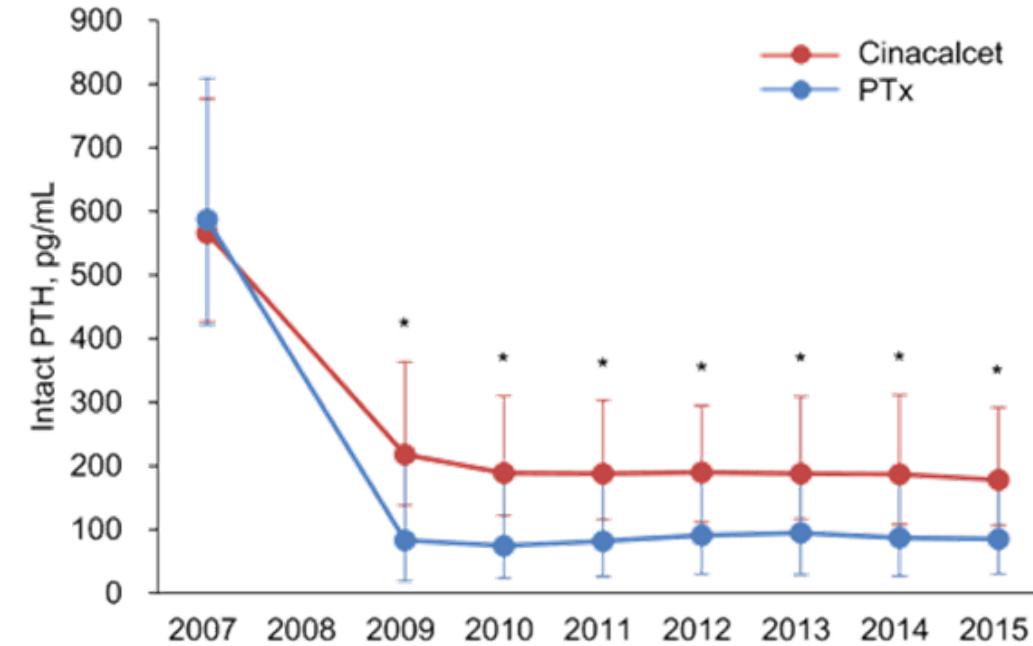
Parathyroidectomy



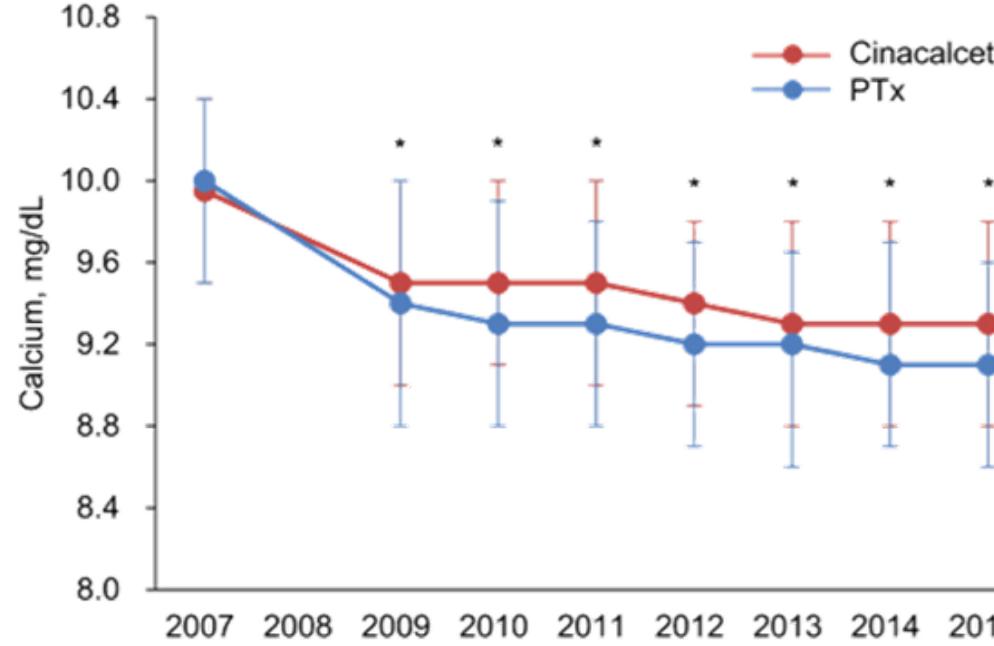
TREATMENT OPTIONS FOR HYPERPARATHYROIDISM



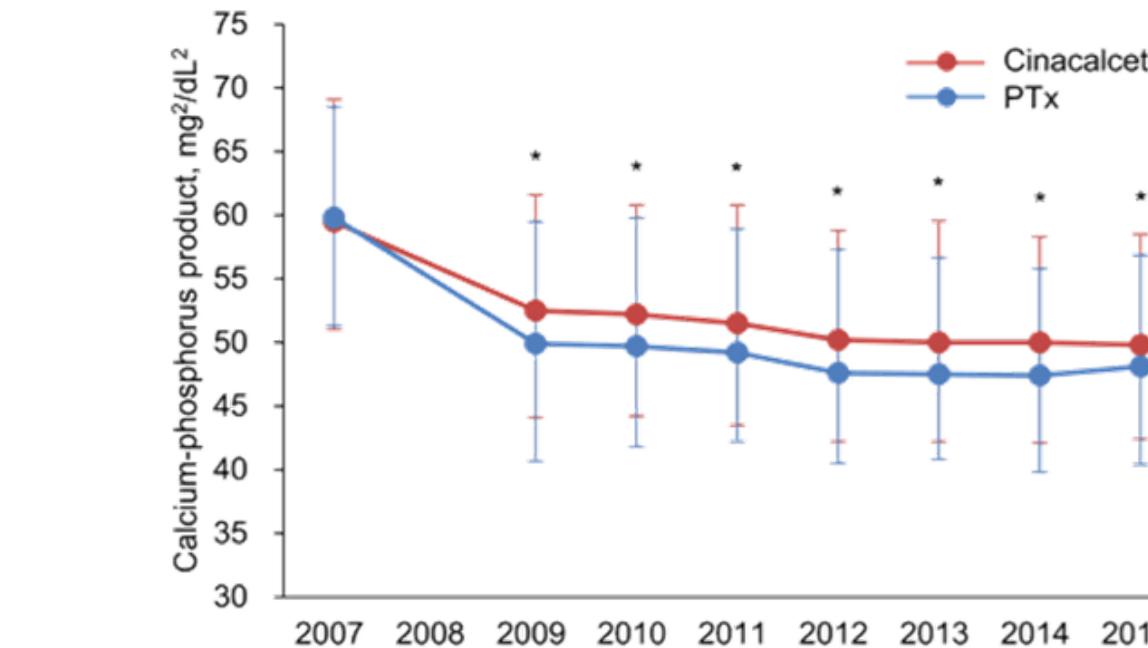
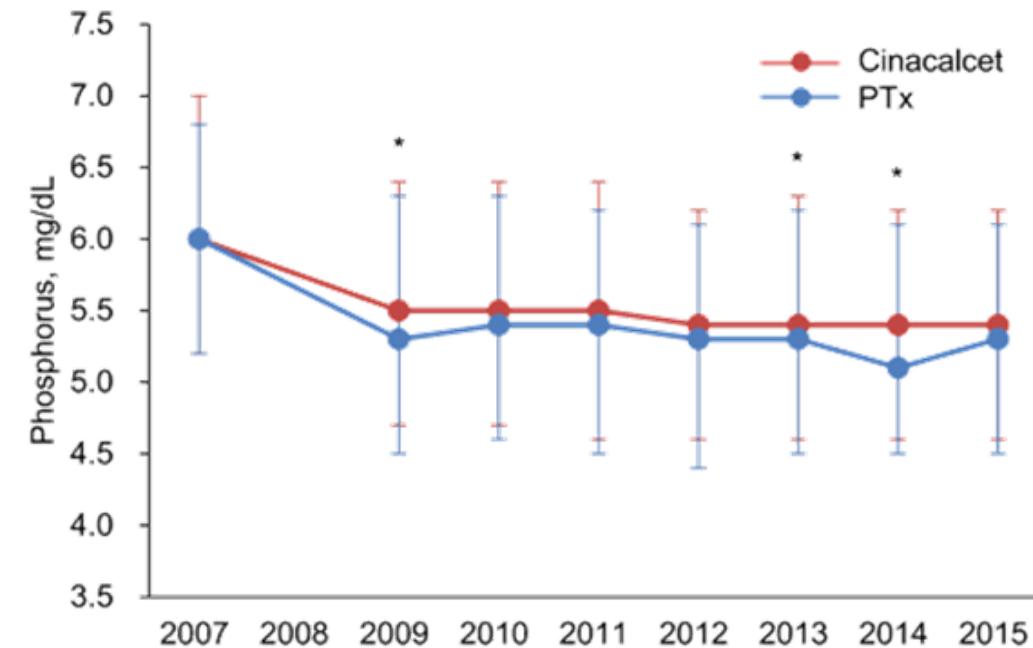
PARATHYROIDECTOMY AND NUTRITION



No. of patients	
Cinacalcet	2,682
PTx	894



No. of patients	
Cinacalcet	2,570
PTx	863



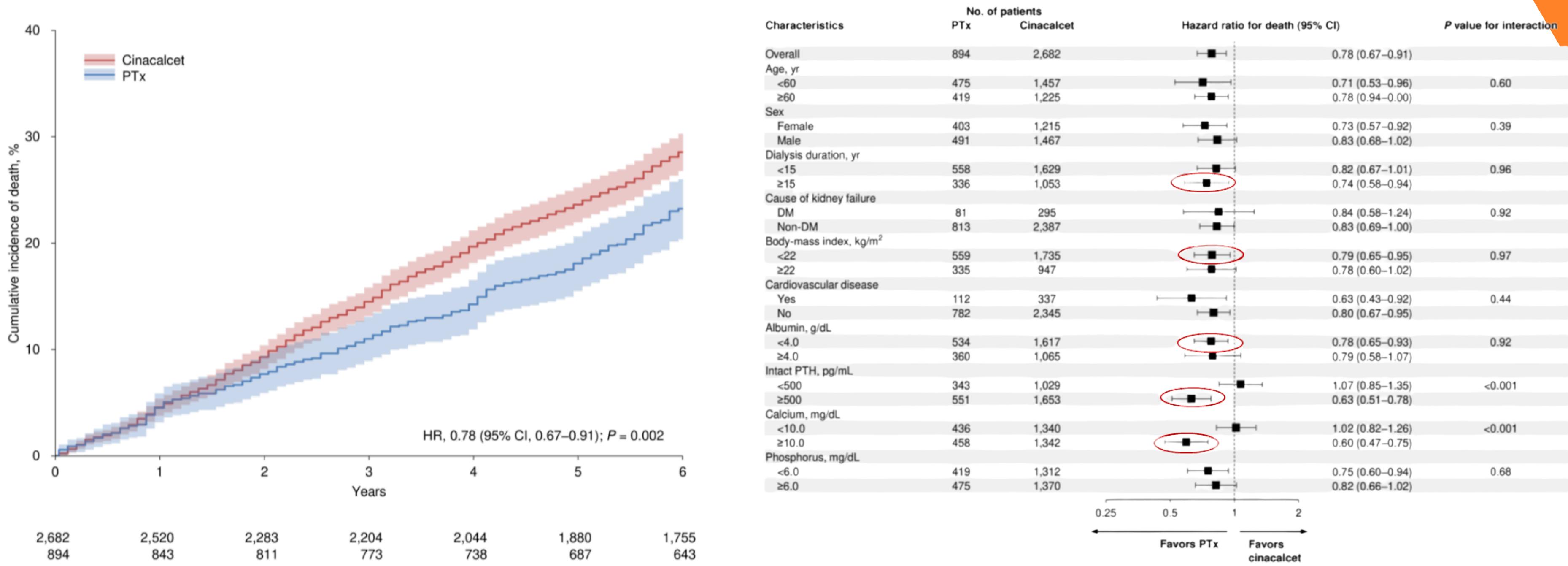
No. of patients	
Cinacalcet	2,570
PTx	863

- Patients with iPTH ≥ 300 pg/mL underwent PTx or started treatment with cinacalcet were matched by propensity score 1:3 ratio
- Those who underwent PTx had lower serum calcium, phosphate, and PTH levels



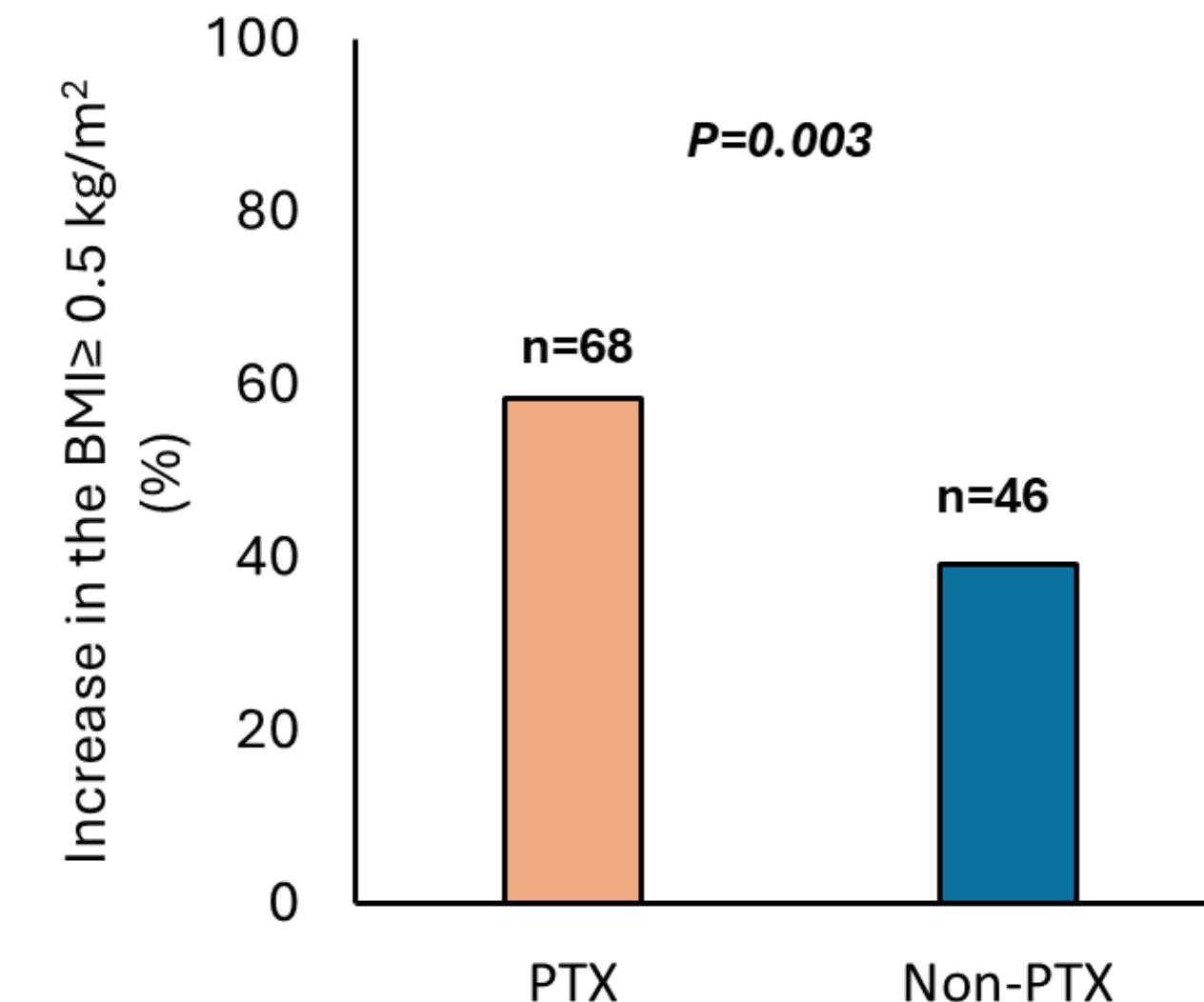
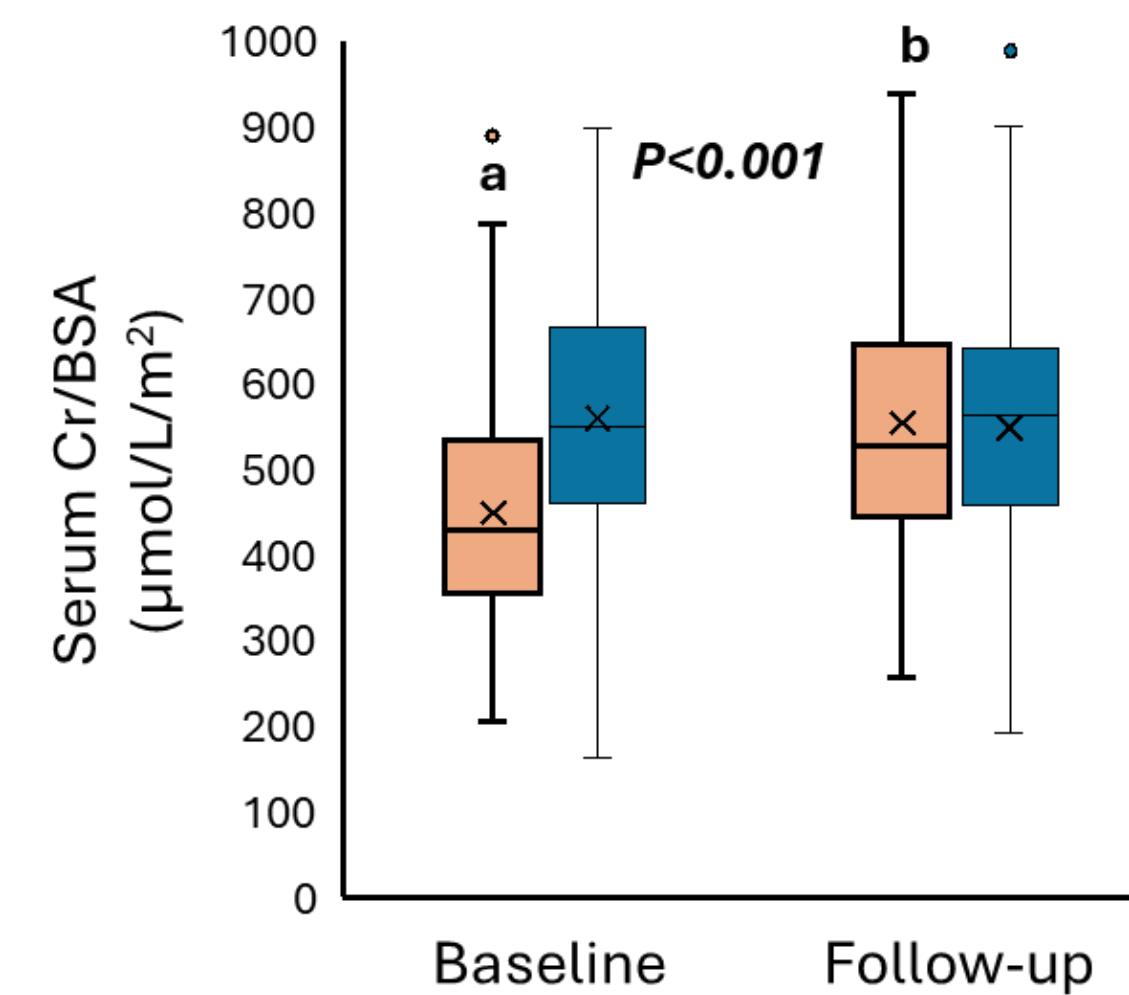
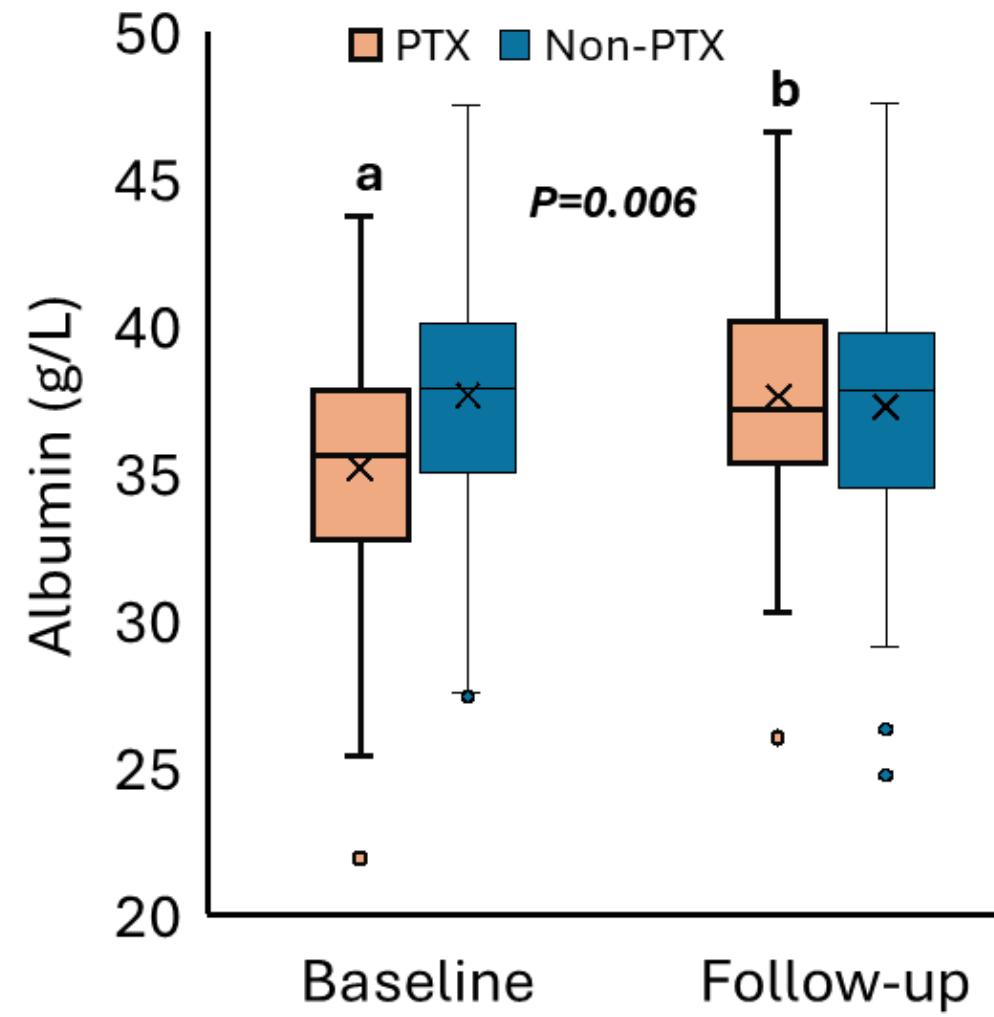
PARATHYROIDECTOMY AND NUTRITION

The reduction in mortality was more pronounced among patients with more severe HPT and impaired nutrition



NUTRITIONAL IMPROVEMENT AFTER PTX

- HD patients who underwent PTX were matched 1:1 to non-PTX and Pre-PTX HD patients
- Serum albumin, Cr/BSA, and BMI increased after PTX



SUMMARY

01

ADVERSE HEALTH CONSEQUENCES OF SEVERE HPT

Bone loss, protein-energy wasting, weight loss, vascular and soft tissue calcification, dementia, and death.

02

MECHANISMS OF PROTEIN ENERGY WASTING

Adipose tissue browning and increased energy expenditure
Enhanced muscle proteolysis and impaired energy production
Systemic inflammation and hormonal imbalance

03

MANAGEMENT

Phosphate restriction/phosphate binders, active vitamin D, calcimimetics, and parathyroidectomy when medications fail.

**THANK YOU
FOR YOUR ATTENTION**

