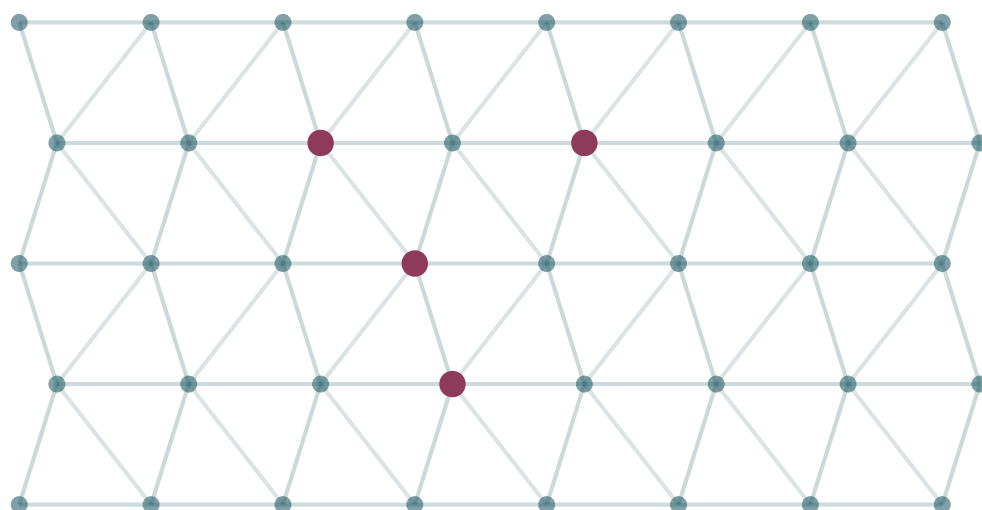


AN ILLUSTRATED GUIDE · THROUGH 2026

Understanding Bone Health

Beyond bone density — the scans that see more, and the real causes: iron, body weight, hormones & light.



An illustrated anatomy of strong bone — and how to keep it.

Written with **women's bone health at the center** —
because the menopausal years change bone fastest — and
essential for men, too.

Created on behalf of **Josi Clary Coaching**

A PLAIN-LANGUAGE DEEP DIVE · DESIGNED FOR COMFORTABLE READING · NOT MEDICAL
ADVICE

START HERE

A clearer, deeper picture of your bones

This guide is for anyone who wants to understand what keeps bones strong — and what quietly weakens them. It is written for a general reader, not a doctor, but it does not water the science down. It includes an in-depth look at **REMS**, a newer scan that can reveal things a standard DEXA misses. Where the evidence is strong, it says so. Where something is promising but still debated, it says that too.

~1 in 2

women over 50 will break a bone from fragile bone

up to 1 in 4

men over 50 — so this matters for everyone

0–100

the REMS Fragility Score for bone quality

half

of fractures are missed by density alone

WHY THIS GUIDE CENTERS ON WOMEN

After menopause, the drop in estrogen makes women lose bone faster than men, and women face more fragility fractures over a lifetime. So this guide foregrounds the female experience — menopause, estrogen, very low body weight. **♂ Relevant for men too** where a point applies equally to men, you'll see this marker, and the biology (including the iron and hormone stories) is shared by both.

How to read this guide: the evidence labels

Throughout, claims are tagged so you can tell solid ground from open questions:

- **Established** Strong, repeated evidence in humans. You can rely on this.
- **Emerging** A real biological mechanism with growing support, but the human evidence is still incomplete. Reasonable to act on the low-risk parts.
- **Promising / debated** A newer technology or idea with genuine support *and* active scientific disagreement. Worth knowing and using thoughtfully — not yet the final word.

CONTENTS

What's inside

1	Bone health is more than “density”	4
2	How your bones actually work	6
3	Seeing what DEXA misses — including REMS	7
4	What weakens bone	12
5	The iron connection (& hemochromatosis)	13
6	Body weight — the risk of being very thin	15
7	Hormones, estrogen & bone	17
8	The common thread: oxidative stress	19
9	Diet: what genuinely helps	20
10	Light, sleep & your body clock	22
11	Movement and loading	24
12	Tests worth asking about	25
13	The treatment landscape	26
✓	A simple starting checklist	28
§	Sources & further reading	29

1

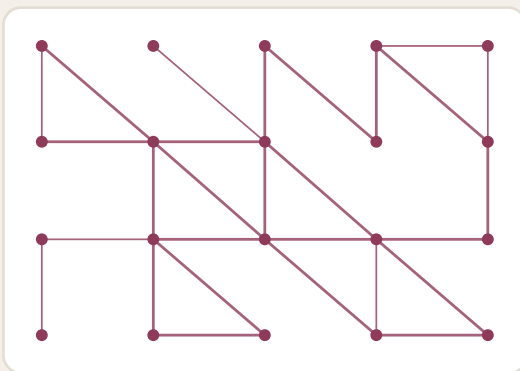
THE BIG IDEA

Bone health is more than “bone density”

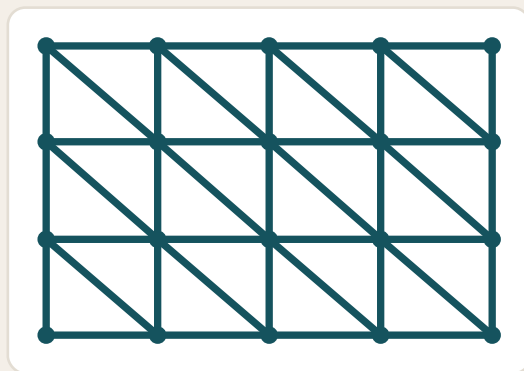
When people talk about osteoporosis, they almost always mean a low score on a **DEXA scan** — a measurement of how much mineral is packed into the bone. That number matters, but it is only half the story.

Density tells you *how much* bone there is. It does not tell you how well that bone is *built* — the honeycomb lattice inside, the protein scaffolding the mineral sits on, and the overall shape.

Same amount of mineral — very different strength



THIN, BROKEN STRUTS
fragile — breaks under load



THICK, CONNECTED LATTICE
strong — spreads the load

Quality vs. quantity. Two bones can hold the same mineral yet differ greatly in strength. A thin, broken internal lattice fails under load; a thick, well-connected one spreads it.

KEY POINT

Relying on bone density alone misses up to half of the people who eventually fracture. In fact, more fractures happen in people in the “in-between” zone (osteopenia) than in those who meet the full definition of osteoporosis — because there are more of them, and because quality, not just quantity, decides whether a bone breaks.

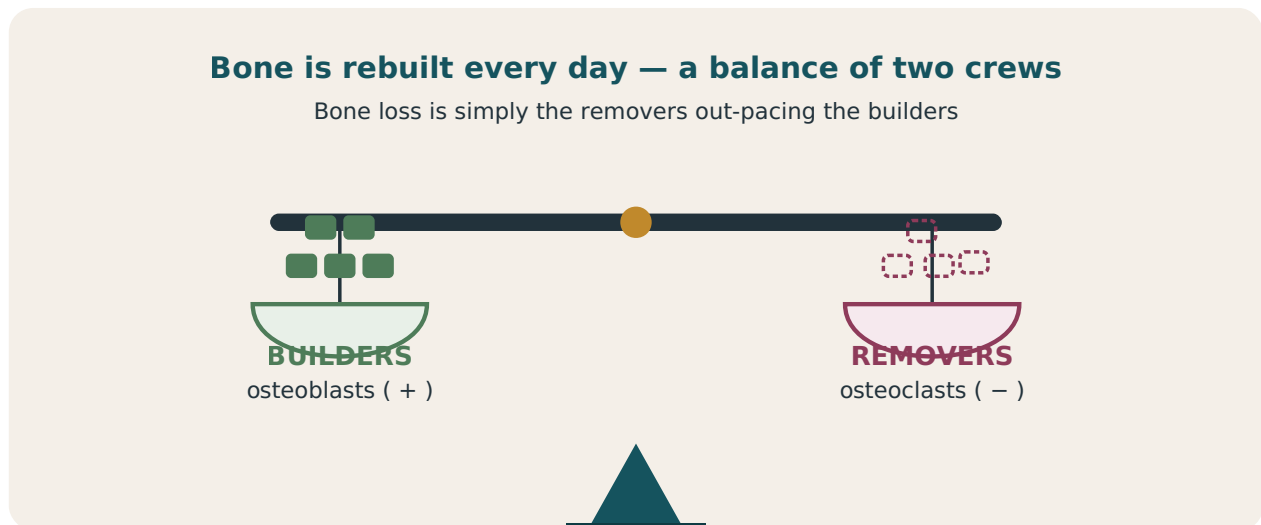
This one idea runs through the whole guide. It is also why the newer scans in Section 3 — especially REMS — have drawn so much interest: they try to measure the quality that density alone cannot see.

2

FOUNDATIONS

How your bones actually work

A quick foundation makes everything else click. Bone is not dead scaffolding — it is living tissue, constantly torn down and rebuilt in a process called **remodeling**. Two cell types run the show.



The daily balance. Builders (osteoblasts) lay down fresh bone; removers (osteoclasts) clear old bone away. In health they balance. Bone loss is the removers pulling ahead.

Almost every cause of osteoporosis works by tipping this balance: **estrogen loss** releases the brakes on the removers; **aging** slows the builders; **excess iron** (Section 5) poisons the builders while energizing the removers; and too little **loading or nutrition** starves the builders of signal and raw materials. **Established**

Keep this builder-versus-remover picture in mind — it is why the iron story and the hormone story are not separate topics, but two ways of tipping the same scale.

3

Seeing what DEXA misses — the scans that measure quality

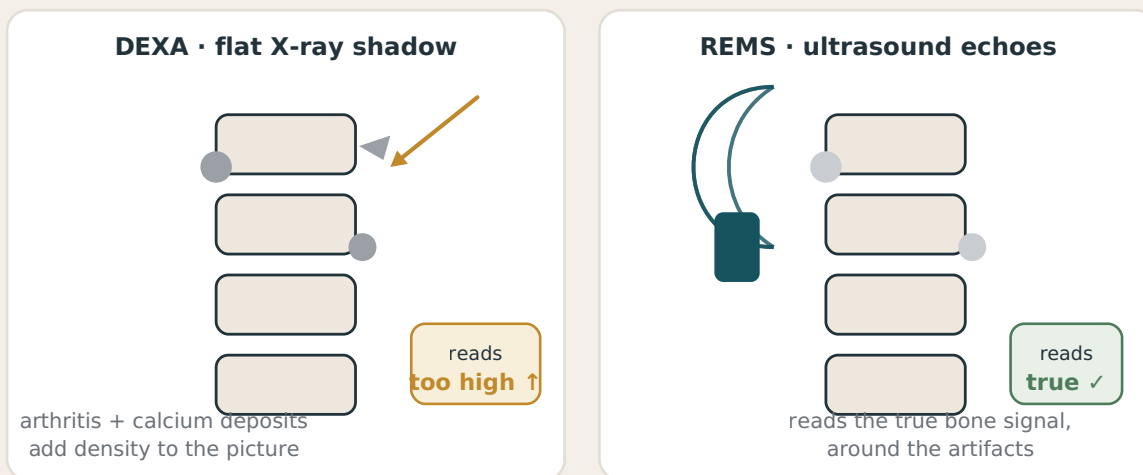
If bone strength is about quality as well as quantity, then how we measure bone matters enormously. Here are the main tools — from the familiar standard to the newer technologies your mother or friends may have heard about — with REMS covered in depth.

3.1 DEXA — the standard, and its blind spots

DEXA (dual-energy X-ray absorptiometry) is the long-standing gold standard. It uses a very low dose of X-rays to measure density at the spine and hip, and produces the **T-score** used to diagnose osteoporosis. It is fast, widely available, and the basis of most treatment guidelines. **Established** But it has real blind spots:

- It measures quantity, not quality — it cannot directly see microarchitecture.
- **It can be fooled by “artifacts.”** In older people especially, spinal arthritis, calcium in the aorta, and crush-deformed vertebrae all add density to the X-ray image — falsely raising the score.
- It uses ionizing radiation (low dose, but a consideration for frequent monitoring).
- The machine is large and stationary; frail patients must be transported and positioned on it.

Why a DEXA can read “falsely reassuring” — and REMS can see past it



The artifact trap. A flat X-ray adds up everything in its path, so arthritis and calcium can make fragile bone look stronger. REMS reads sound echoes and can work around those distortions.

3.2 TBS (Trabecular Bone Score) — quality from the DEXA image

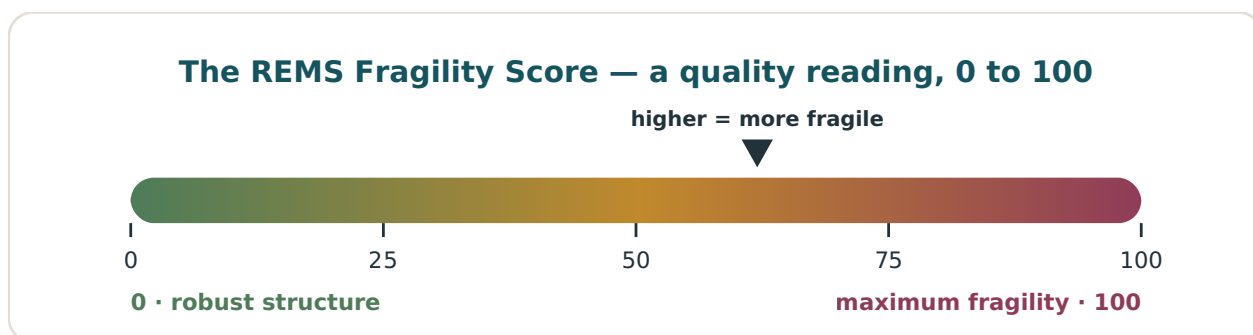
Established TBS is software that re-reads your existing DEXA spine image to estimate the internal honeycomb texture — a quality layer on top of the density number, with no extra scan or radiation. If your DEXA center offers it, it is worth requesting.

3.3 REMS — an in-depth look

REMS stands for **Radiofrequency Echographic Multi Spectrometry**. It is a radiation-free, ultrasound-based scan (the main device is made by Echolight) that assesses bone at the **lumbar spine and the femoral neck (hip)** — the same fracture-prone sites DEXA uses. That axial focus is what sets it apart from older “heel ultrasound” tests, which only measured a peripheral site.

How it works, in plain terms: a probe sends ultrasound into the bone and listens to the raw echoes that bounce back. Healthy bone and fragile bone return subtly different echo “fingerprints.” REMS compares your echoes against reference models to estimate density — and, crucially, to gauge quality. It reports the

familiar BMD, T-score and Z-score, plus one extra number that is the heart of the excitement.



The Fragility Score. A 0–100 reading of bone quality — microstructural resilience and strength — independent of density. Two people with the same density can score differently; the higher score signals higher fracture risk.

WHY PEOPLE CARE ABOUT REMS

The Fragility Score captures the **quality** a plain DEXA cannot, and REMS adds several practical advantages — listed below with honest evidence labels.

- **Measures quality, not just quantity.** **Emerging** The Fragility Score adds the microarchitecture dimension density misses.
- **Sees past the artifacts that fool DEXA.** **Emerging** Reading echoes rather than a flat shadow, REMS can give a valid result where arthritis or calcification would inflate a DEXA score — the difference between a true and a falsely reassuring reading.
- **No radiation.** **Established** Safe for frequent monitoring and for groups underserved by DEXA, including pregnant women and children. Well suited to tracking whether a treatment is working.
- **Portable.** **Established** The device can come to the bedside or a clinic, avoiding the difficult transfer DEXA needs for frail patients.
- **Useful in very thin, low-weight patients.** **Emerging** REMS has specifically been studied in low-body-weight groups, including young women with

anorexia nervosa — directly relevant to anyone at the very lean end, where DEXA is least reliable.

Where the evidence stands — honestly

- **Established** REMS density readings correlate strongly with DEXA across many studies, with good repeatability, and it has been FDA-cleared since 2018.
- **Emerging** A 5-year follow-up study found the Frailty Score predicted future fragility fractures — the key promise of the technology.
- **Emerging** An expert panel (ESCEO) was “strongly in favor” of REMS for certain uses (such as frail patients who cannot easily get DEXA), but only “weakly in favor” for fracture prediction, short-term monitoring, and overcoming artifacts — promising, not yet settled.

THE HONEST COUNTERWEIGHT — AN ACTIVE SCIENTIFIC DEBATE

REMS is exciting, but a balanced view protects against over-correcting. In 2025–2026, researchers noted that in one analysis **more than 90% of the variation in REMS density readings could be explained by age and weight alone** — prompting an editorial asking whether REMS density is a true independent *measurement* or substantially a *prediction* built from demographics. REMS is also operator-dependent (probe position matters), and **DEXA remains the gold standard for diagnosis and treatment decisions** — most physicians will not start or change medication on REMS alone. The fair takeaway: REMS is best used as a **valuable complement** to DEXA — especially where DEXA struggles — not a replacement. The mood in the field is genuine cautious optimism, not consensus.

3.4 HR-pQCT and QCT — the research-grade view

Established HR-pQCT is a high-resolution CT that directly images bone’s microscopic structure at the wrist or shin — the closest thing to seeing the architecture itself. QCT is a CT-based density measure. Both are mostly used in

research or specialized centers, but they are the reference against which quality tools are judged.

3.5 At a glance: how the main tools compare

Feature	DEXA	TBS	REMS
Main output	Density, T-score	Texture add-on	Density + Fragility Score
Quality info?	Limited	Yes (texture)	Yes (Fragility Score)
Radiation	Low-dose X-ray	Uses DEXA image	None (ultrasound)
Sites	Spine, hip	Spine	Spine, hip
Fooled by artifacts?	Yes (often)	Partly	Resistant
Portable?	No	—	Yes
Status	Gold standard	Accepted add-on	Promising, debated
Best role	Diagnosis & treatment	Refine risk	Complement; monitor; artifact / thin cases

The practical message: these tools are layers, not rivals. The richest picture combines a DEXA (for diagnosis) with a quality measure — TBS and/or REMS's Fragility Score — and a clinical risk estimate (FRAX). For someone very thin or with spinal arthritis, REMS's artifact resistance and radiation-free monitoring make it especially worth asking about.

4

CAUSES

What weakens bone

The familiar causes are real and worth stating plainly:

- Loss of estrogen after menopause (and low testosterone in men). Sex hormones restrain the remover cells; losing them speeds bone loss.
Established ♂ **Relevant for men too**
- Aging itself, which slows the builder cells. **Established**
- Too little calcium, vitamin D, or protein; too little weight-bearing activity.
Established
- Certain medications (notably long-term steroids) and conditions (overactive thyroid, gut malabsorption, and others). **Established**

But two causes rarely make it into the standard conversation, and both can be missed for years: **excess iron** in the body, and being **significantly underweight**. They are the focus of the next two sections.

5

The iron connection — including hemochromatosis

This is one of the most overlooked drivers of bone loss. The body needs iron, but **too much** iron is toxic to bone — and the science behind why has matured sharply in just the last two years.

How excess iron harms bone

Emerging Research through 2025 points to a process called **ferroptosis** — a form of cell death triggered by iron combined with oxidative “rusting” of cell membranes. In bone it is doubly bad: it **kills the builder cells** while **revving up the remover cells**, tilting the skeleton toward net loss. In the lab, removing the excess iron reverses much of this damage.

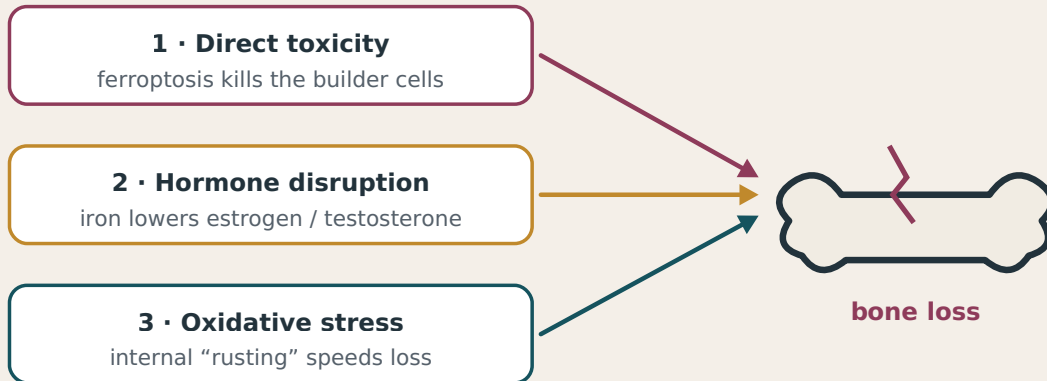
Hemochromatosis: when this is not hypothetical

Hereditary hemochromatosis is an inherited condition — most often from changes in a gene called **HFE** — in which the body stores too much iron over a lifetime. It is **not rare**: roughly 1 in 300 people of Northern European descent carry the higher-risk “double” version, and about 1 in 7 carry a single copy. [🔗](#)

Often affects men earlier

- In studied groups, roughly a quarter to a third had outright osteoporosis, and another ~40% had osteopenia. **Established**
- The more iron a person carried, the worse the bone — more than hormones or liver disease alone could explain, fitting the idea that iron itself is directly harmful.

Too much iron attacks bone three ways at once



The “triple hit.” Excess iron damages bone three ways at once: it poisons builder cells, it lowers protective sex hormones, and it drives oxidative stress — which is why iron overload can be so damaging.

The simple tests that can settle it

If iron is even a question in your family, the workup is inexpensive and informative. Ask a doctor specifically about:

- **Serum ferritin** (stored iron — though it can rise with inflammation, so it is imperfect alone).
- **Transferrin saturation** (a more specific signal of iron overload).
- **HFE genetic testing** (for the C282Y and H63D variants) if iron numbers are high or there is family history.

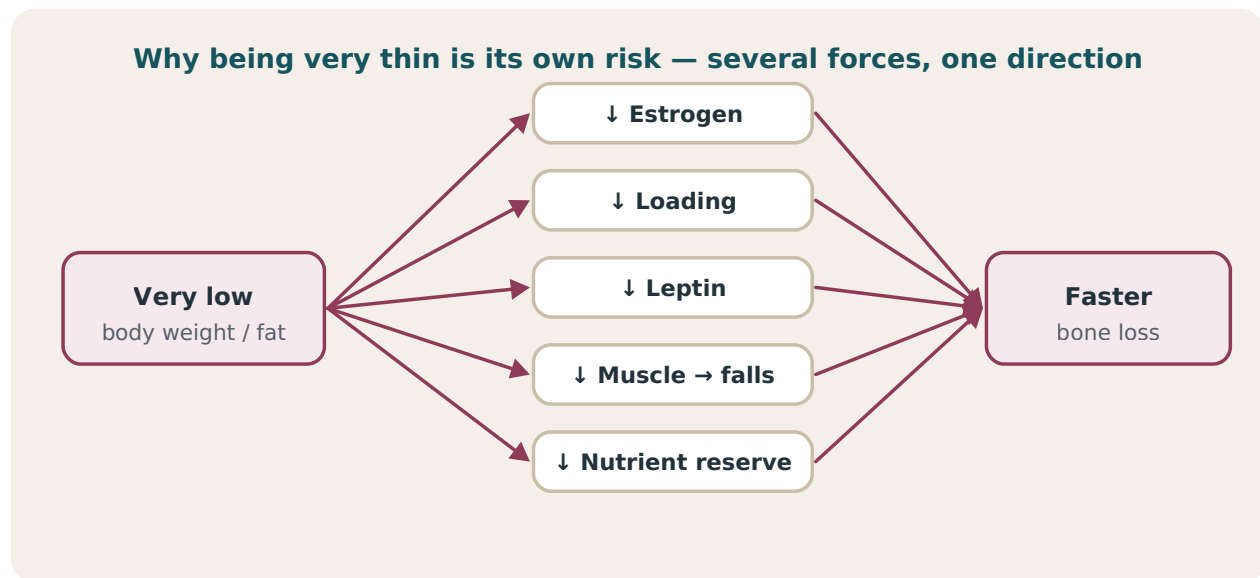
As more than one case report has put it: a simple blood panel can reveal the cause of bone loss that would otherwise go unexplained for years.

6

BODY WEIGHT

Why being very thin carries its own risk

Most public conversation about weight focuses on the risks of carrying *too much*. For bone, the opposite end of the scale deserves equal attention. Being significantly **underweight** is a well-established, independent risk factor for fracture — and almost everything that makes higher body weight protective of bone is missing in a very thin person.



One direction, several forces. Very low body weight removes estrogen, mechanical loading, leptin, muscle, and nutritional reserve — all of which push the same way: toward faster bone loss.

- **Less estrogen.** **Established** After menopause, body fat becomes a main source of estrogen. A very thin woman has little of this tissue left, so she sits near the bottom of the estrogen range — and estrogen protects bone.
- **Less mechanical loading.** **Established** Bone strengthens in response to force; less body weight means a weaker daily “build” signal.
- **Less muscle (sarcopenia).** **Established** Low weight tracks with muscle loss — more falls, and less cushioning over the hip when a fall happens.
- **Less nutritional reserve.** **Established** You cannot build strong bone without enough protein, minerals, and energy.

- **Lower signaling hormones.** **Emerging** Fat tissue makes hormones such as leptin that appear to support bone; very low fat means low levels.

AN IMPORTANT NUANCE ABOUT SCANS IN VERY THIN PEOPLE

In a very lean person, a DEXA score may actually **understate** true fracture risk — partly a measurement quirk, partly because there is little soft-tissue padding to protect the hip in a fall. A number that looks “not too bad” should not be falsely reassuring. This is exactly where a quality measure (TBS or REMS’s Fragility Score) and a FRAX estimate — which counts low body weight — become valuable. REMS’s radiation-free monitoring also makes close tracking easier.

A fair caveat for balance: across the whole population the weight–fracture link is not perfectly simple, and higher weight brings its own fracture risks (more falls, certain sites). **Both ends of the scale carry risk — through different mechanisms.** But for someone markedly underweight, the forces above all push the same direction.

WORTH SAYING GENTLY

Significant, unexplained thinness in an older adult is itself worth a medical check — it can be the visible sign of something treatable (such as a problem absorbing nutrients) that is also harming bone. Addressing the root cause matters as much as the bone itself.

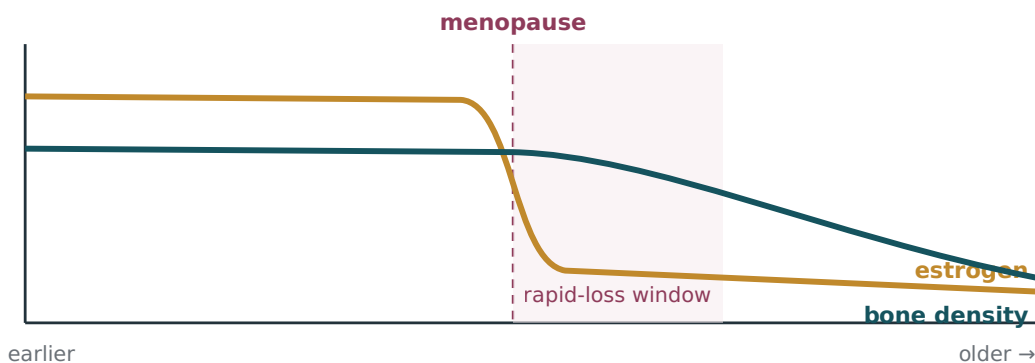
7

HORMONES

Hormones, estrogen & bone

Because estrogen keeps the remover cells in check, the menopausal drop in estrogen is the single biggest reason women lose bone rapidly in the years right after menopause. This is why hormone-based approaches exist — and why they are nuanced. **Established**

After menopause: estrogen drops, then bone declines



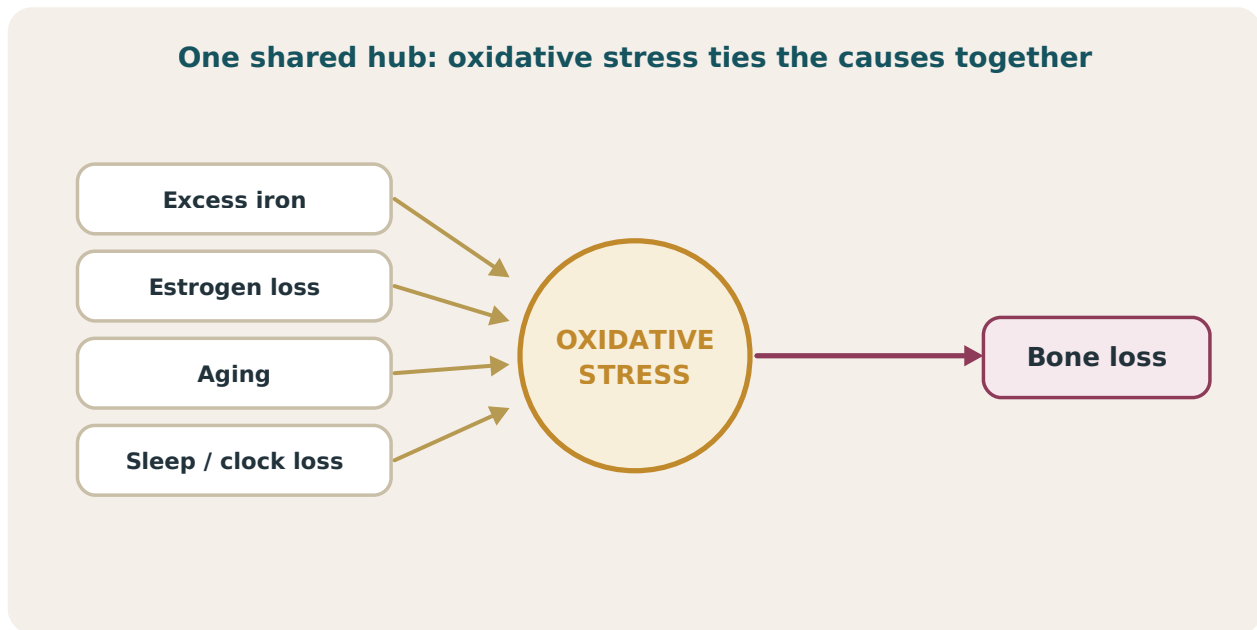
The menopause window. Estrogen falls first; bone density then declines, fastest in the early post-menopausal years — the window where protection matters most.

- **Menopausal hormone therapy** (estrogen, sometimes with progesterone) reliably preserves bone and reduces fractures. Whether it suits you depends on age, time since menopause, history, and how it is delivered — a decision to make with a physician, not from a guide. **Established**
- **Delivery route matters.** **Emerging** Transdermal (patch/gel) and oral estrogen differ in their risk profiles, and FDA-approved “body-identical” hormones differ from custom compounded preparations. Worth raising specifically.
- **In men**, low testosterone similarly accelerates bone loss — part of why the iron-hormone link in Section 5 matters for men too. **♂ Relevant for men too**

The point is not to recommend or discourage any hormone approach — it is that the hormone conversation is central to bone health and deserves a careful, individualized discussion.

The common thread: oxidative stress

Here is a connection rarely drawn but clarifying. Excess iron, the loss of estrogen, ordinary aging, and a disrupted body clock all converge on one thing inside bone: **oxidative stress** — a kind of internal “rusting” from unstable molecules that damage cells.



The hub. Several separate-seeming causes feed the same fire — oxidative stress — which drives bone loss. Much of what protects bone works by keeping this fire low.

- Iron directly fuels it (the heart of ferroptosis).
- Estrogen is itself a natural antioxidant in bone, so losing it raises the “rusting.”
- This is why several experimental bone-protective compounds work mainly by calming oxidative stress — and why the sleep hormone melatonin (Section 10) does the same.

Emerging You need not act on this directly. It is the unifying idea: much of what protects bone — hormones, good sleep, not overloading on iron — can be seen as keeping oxidative stress in check.

Diet: what genuinely helps

For someone who is underweight, the priorities flip from the usual “eat less” messaging. The dominant problem is shortage, not excess.

- **Enough total food.** **Established** For a very thin person, simply eating enough so the body is not breaking down its own muscle and bone comes before everything else.
- **Protein.** **Established** Bone is roughly half protein by volume. Guidance for older adults at risk of frailty suggests aiming higher than the bare minimum — on the order of 1.0–1.2 grams per kilogram of body weight per day, spread across meals. Higher protein intake is linked with better density and fewer spine fractures over time.
- **Calcium and vitamin D.** **Established** The foundation; vitamin D lets the body absorb calcium. Deficiency turns up again and again in people with bone loss.
- **Supporting cast.** **Emerging** Magnesium, vitamin K2 (which helps steer calcium into bone rather than soft tissue), and adequate — not excessive — vitamin A.

THE IRON PARADOX — PLEASE READ BEFORE ADDING IRON

This is the single most important caution in this guide, and the easiest to get wrong. A thin, tired-looking older person is often told to “build up” with iron-rich red meat and vitamin C. But if that person has iron overload or hemochromatosis, this advice **flips and becomes harmful to bone**. In that case the sensible direction is the reverse:

- Do not take iron supplements unless a deficiency is confirmed.
- Go easy on heme iron (red and organ meats) rather than loading up.
- Avoid pairing vitamin C with iron-rich meals (vitamin C boosts iron absorption).
- Calcium, coffee, and tea with meals reduce iron absorption — normally a nuisance, here potentially helpful.

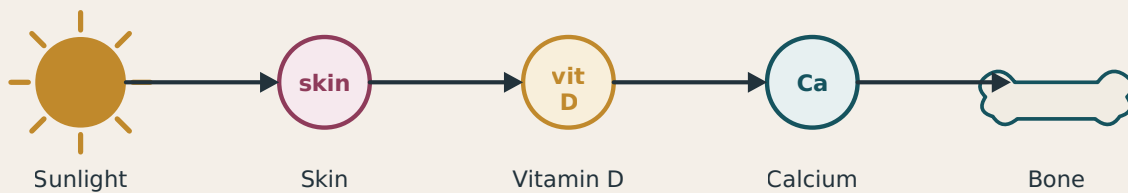
And the standard treatment for iron overload — **therapeutic phlebotomy** (giving blood to lower iron) — is plausibly good for bone as well. The bottom line: get the iron blood tests *before* anyone starts pushing iron-rich food or pills.

Because feeding an underweight older adult well — enough energy *and* protein *and* the bone nutrients, while respecting any iron issue — takes some skill, working with a **registered dietitian** is genuinely worth it rather than guessing alone.

Light, sleep & your body clock

Light affects bone in one well-proven way and several interesting-but-unsettled ways. Keep them separate.

The proven light-bone pathway: sun → vitamin D → calcium → bone



The proven route. Sunlight on skin makes vitamin D, which lets the gut absorb calcium to mineralize bone. For many older or mostly-indoor people, a supplement is the practical path.

ESTABLISHED

Sunlight (UVB) lets the body make vitamin D, essential for absorbing calcium and mineralizing bone. This is the solid, non-negotiable link between light and bone. For many people — especially the frail or mostly-indoor — a vitamin D supplement is the practical route, worth discussing with a doctor.

EMERGING

Bone is partly governed by the body's 24-hour clock. Bone turnover rises and falls over the day, and disrupting the clock — shift work, poor sleep, constant light at night — is associated with worse bone. The sleep hormone melatonin appears to protect bone in lab and animal studies, partly by calming oxidative stress. Low-risk takeaways: get morning daylight, protect your sleep, keep nights genuinely dark. Melatonin is not yet an approved osteoporosis treatment.

PROMISING / DEBATED

Some popular frameworks claim specific light wavelengths or precise sun-timing directly “power” the body’s cells and bones beyond vitamin D. The underlying ingredients — that cellular energy, oxidative stress, and the body clock matter — are legitimate and overlap with the cutting-edge science here. But the specific protocols and strong claims are not proven. Take the curiosity seriously; hold the specific prescriptions loosely.

11

MOVEMENT

Movement and loading

Established Bone is living tissue that strengthens when loaded. **Weight-bearing activity** (walking, stair-climbing) and especially **progressive resistance / strength training** signal bone to maintain and build itself, while building the muscle that prevents falls. For someone thin and short on muscle, supervised strength work is one of the highest-value things they can do — ideally guided by a physiotherapist who can keep it safe.

Balance training (and removing fall hazards at home) deserves equal weight: many fractures are ultimately a fall problem as much as a bone problem.

12

Questions & tests worth raising with a doctor

None of this replaces a clinician who can see the actual numbers. But these are concrete, low-cost things you can ask about by name:

- A **DEXA scan** — and whether **TBS** (the bone-quality read-out) can be added to it.
- A **REMS scan** (Echolight) where available — especially for radiation-free monitoring, its Fragility Score, and accuracy when spinal arthritis or very low body weight would limit DEXA. Use it to complement DEXA, not to replace treatment decisions.
- A **FRAX** fracture-risk estimate, which factors in low body weight and other risks density alone misses.
- **Iron studies:** ferritin and transferrin saturation; HFE genetic testing if those are high or there is family history.
- **Vitamin D** level, calcium, and a review of the hormone (estrogen / testosterone) picture. (♂ Relevant for men too)
- For marked or unexplained thinness, a broader check for causes such as nutrient malabsorption.
- A referral to a **registered dietitian** for safe weight and nutrition support.

13

The current treatment landscape (through 2026)

Medicines do not replace bone in a simple way; they tip the build-versus-remove balance, and the goal is to reduce fractures. A doctor matches the choice to the person's risk.

How the medicines work: build more, or remove less



Two levers. Some drugs build more bone (anabolics; romosozumab); others slow removal (antiresorptives; romosozumab does both). For very high risk, building first, then locking in gains, is often preferred.

- **Antiresorptives** (bisphosphonates, denosumab) slow bone breakdown — the long-standing backbone. **Established**
- **Bone-building (anabolic) agents** (teriparatide, abaloparatide) actively stimulate new bone. **Established**
- **Romosozumab** both builds bone and slows breakdown. For very high risk, the current approach is often to build first, then follow with an antiresorptive. It carries a heart-related warning, so cardiovascular history must be reviewed first — relevant if hemochromatosis (which can affect the heart) is in the picture. **Established**
- **On the horizon.** **Emerging** Researchers are pursuing broader bone-formation pathways and — tying back to Section 5 — treatments aimed at the iron /

oxidative-stress process itself. A late-2025 regulatory change in how trials are judged is expected to speed new drugs along.



TAKE ACTION

A simple starting checklist

If you take nothing else from this guide, these steps capture the heart of it:

- **Get the iron blood tests first** — cheap, and they can change the entire plan, including the diet.
- **Ask for bone quality, not just density** — add TBS to a DEXA, consider a REMS Fragility Score where available, and get a FRAX estimate.
- **Treat nutrition as adequacy, not restriction** — enough food and protein, plus calcium and vitamin D; hold iron until the tests are back; involve a dietitian.
- **Build muscle and balance** — supervised strength training does double duty for bone and fall prevention.
- **Protect sleep and get morning daylight** — free, low-risk, biologically sensible.
- **Have the hormone conversation** — individualized, with a physician.
- **Bring all of it to a doctor** — this guide is for asking better questions, not for self-treating.

“Density tells you how much bone there is. Quality tells you whether it will hold. Aim to understand both.”



Sources & an important note

This guide summarizes peer-reviewed research in plain language. Key sources include:

- REMS in the diagnosis and management of osteoporosis: state of the art. *Aging Clinical and Experimental Research*, 2024.
- Radiofrequency Echographic Multi Spectrometry — a novel tool: a systematic review, 2025 (incl. ESCEO GRADE recommendations; FDA clearance 2018).
- Fragility Score: a REMS-based indicator for prediction of incident fragility fractures at 5 years. *Aging Clin Exp Res*, 2023; 5-year follow-up, *Bone*, 2020.
- REMS can overcome internal artifacts and evaluate bone fragility accurately. *Calcified Tissue International*, 2024.
- Editorial: Is REMS-BMD truly a measured parameter? *Osteoporosis International*, 2025–2026 (age/weight critique and rebuttals).
- Targeting ferroptosis: emerging insights into osteoporosis mechanisms. *Biology*, 2025; related ferroptosis reviews, 2025.
- Iron overload and osteoporosis in hereditary hemochromatosis; BMD in HFE mutation carriers. *Osteoporosis International*.
- Underweight and risk of fractures in adults (nationwide cohort). *Scientific Reports*, 2023; obesity/aromatase/leptin reviews.
- Protein recommendations for muscle and bone health in older adults. *Frontiers in Nutrition*, 2024.
- Trabecular Bone Score and bone quality beyond DEXA; Bone Strain Index. *Reviews*, 2020–2025.
- Melatonin: a potential therapy for osteoporosis. *Journal of Pineal Research*, 2025; circadian rhythm and bone reviews.
- Novel therapies in osteoporosis — clinical update, 2025; FDA surrogate-endpoint qualification, Dec 2025.

IMPORTANT

This document is for general education only and is not medical advice. It cannot account for any individual's full health picture. Please use it to inform a conversation with a qualified physician, who can interpret your own results and tailor any plan to you. Evidence labels reflect the general state of research and may evolve.

Prepared on behalf of **Josi Clary Coaching**