

BY MARYJO KETTERHAGAN

*Strong Bones*  
*Strong Life*



**THE COMPLETE 2026 GUIDE TO PREVENTING, MANAGING  
AND REVERSING OSTEOPOROSIS**

# STRONG BONES STRONG LIFE

*The Complete 2026 Guide to Preventing, Managing  
and Reversing Osteoporosis*

Updated for 2026 • Evidence-Based • Actionable

## **What you will discover inside:**

- How your skeleton silently rebuilds itself every 10 years
- The 2026 treatment landscape: new drugs, HRT timing, and reversing steroid damage
- Which exercises build the most bone — and which to avoid
- Nutrition strategies backed by the latest protein and supplement research
- How to decode your DEXA scan and track real progress
- The future: AI diagnosis, smart wearables, and stem-cell therapies
- Mental health, movement safety, and living without fracture fear

*Note: This ebook is for educational purposes. Always consult a qualified healthcare provider before making changes to your treatment or exercise program.*

## CHAPTER

# Introduction: Why Bones Cannot Wait

---

On a Tuesday afternoon, a 68-year-old woman reaches for a tin of tomatoes on a high shelf. She does not fall. She does not trip. She simply stretches — and feels a sudden, searing pain between her shoulder blades. By the time she reaches her GP, an X-ray confirms what osteoporosis had been quietly orchestrating for decades: a vertebral compression fracture.

This scenario plays out over 3.5 million times a year in Europe and North America combined. The tragedy is not just the fracture — it is that for most people, osteoporosis is entirely detectable years in advance, largely preventable with the right interventions, and increasingly reversible with today's treatments.

This ebook exists to close the information gap. Whether you have just received a diagnosis, are caring for someone who has, or simply want to protect your skeleton before problems begin, you will find everything you need here: clear science, practical strategies, and the most current research available in 2026.

### A Note on Language

Throughout this ebook, the terms 'osteoporosis' and 'low bone density' are used precisely. Osteopenia refers to below-average bone density that has not yet reached the osteoporosis threshold. Both conditions benefit from the same core strategies — the difference is largely one of urgency and the threshold for medication.

Chapters can be read in sequence or dipped into according to your most pressing need. Each chapter ends with an Action Plan — a short, concrete set of steps you can begin implementing immediately.

## CHAPTER 1

# The Secret Life of Bones: How the Skeleton Rebuilds Itself

Bone feels permanent. It feels like rock. But your skeleton is one of the most metabolically active tissues in your body, quietly tearing itself apart and rebuilding every single day. Understanding this process — called bone remodeling — is the foundation of everything that follows in this book.

## The Bone Remodeling Cycle

Imagine millions of microscopic construction sites operating simultaneously throughout your body. At each site, a two-phase process unfolds over the course of roughly three to six months:

**Phase 1 — Resorption:** Specialized cells called osteoclasts (from the Greek for 'bone breaker') attach to bone surfaces and secrete acids and enzymes that dissolve the mineral matrix. They excavate tiny tunnels and cavities, removing old, micro-damaged bone.

**Phase 2 — Formation:** Osteoblasts (bone builders) move in and secrete a protein scaffold called osteoid — primarily Type I collagen — which is then mineralized with calcium, phosphate, and a crystalline compound called hydroxyapatite. The result is fresh, strong bone.

In a healthy young adult, these two phases are exquisitely balanced. Over a lifetime, your entire skeleton is replaced roughly every 10 years. The problem with osteoporosis is a shift in this balance — resorption outpaces formation, and net bone mass is progressively lost.

Fact	Detail	Implication
10%	Of your skeleton is remodeled each year	Interventions work — but take months to show
~200 billion	Osteoclasts active daily in the body	Medications targeting osteoclasts are highly effective
Peak bone mass	Reached by age 25–30	Adolescence is the critical investment window
Trabecular bone	Remodels 10x faster than cortical bone	Spine and hip are first to show loss

## The Cast of Characters: Your Bone Cells

**Osteoblasts** — The builders. Derived from mesenchymal stem cells, they produce collagen and trigger mineralization. They also control osteoclast activity through signaling molecules.

**Osteoclasts** — The demolishers. Large, multinucleated cells derived from the same stem cell lineage as immune cells. They are essential — without resorption, old micro-damaged bone cannot be cleared.

**Osteocytes** — The sensors. Former osteoblasts that became embedded in the bone matrix. They form a vast communication network (the osteocyte lacuno-canalicular network) and detect mechanical load, directing where new bone is needed.

**Lining Cells** — Retired osteoblasts that maintain the bone surface and regulate which ions pass in and out.

## The Hormonal Orchestra

Bone remodeling does not happen in isolation. It is orchestrated by a complex ensemble of hormones:

- Estrogen and testosterone: Both sex hormones suppress osteoclast activity. Their decline at menopause and andropause is the primary driver of accelerated bone loss.
- Parathyroid hormone (PTH): Regulates calcium levels. Chronically elevated PTH (due to low calcium or vitamin D deficiency) dramatically accelerates resorption.
- Calcitonin: Produced by the thyroid, it opposes PTH and inhibits osteoclasts.
- IGF-1 (Insulin-like Growth Factor-1): Stimulates osteoblast activity. Exercise and adequate protein both raise IGF-1.
- Cortisol: When chronically elevated (stress, steroid medications), it suppresses osteoblast function and accelerates bone loss — the basis of steroid-induced osteoporosis.

## Why Bone Loss Accelerates After Menopause

In the decade following menopause, women can lose up to 20% of their bone density. The mechanism is now well understood: estrogen normally acts as a brake on osteoclast activity, in part by stimulating production of osteoprotegerin (OPG), a molecule that blocks osteoclast activation. When estrogen falls, this brake is released, and resorption runs faster than formation can compensate.

### 2026 Research Spotlight

A landmark 2025 study published in the Journal of Bone and Mineral Research used high-resolution peripheral quantitative CT (HR-pQCT) to show that the trabecular microarchitecture of the spine begins deteriorating as early as the perimenopause — sometimes 5–8 years before the first missed period. This has major implications for early HRT intervention (see Chapter 2).

## The Dual-Energy X-Ray Absorptiometry Story

Before DEXA scanning was developed in the 1980s, osteoporosis was diagnosed only after fracture. DEXA measures the attenuation of two X-ray beams to calculate bone mineral density (BMD) at the spine and hip. The resulting T-score compares your bone density to that of a healthy young adult at peak bone mass. A T-score of -2.5 or below is the WHO threshold for osteoporosis. But BMD alone tells only part of the story — bone quality, microarchitecture, and material properties also determine fracture risk (Chapter 9 explores this in depth).

## Action Plan: Understanding Your Own Bone Health

- Ask your GP or specialist about a baseline DEXA scan if you are over 50, postmenopausal, or have risk factors (steroid use, family history, low BMI, smoking).
- Request a FRAX score assessment — a validated tool that calculates your 10-year fracture probability using clinical risk factors even without a DEXA.
- Begin a simple bone health journal: note your daily calcium and protein intake, minutes of weight-bearing exercise, and any medications known to affect bone.
- If you are perimenopausal, discuss early screening with your healthcare provider in light of 2025–2026 research on early trabecular changes.

# The 2026 Treatment Landscape

Osteoporosis pharmacology has entered a genuinely exciting era. Where once clinicians had little beyond bisphosphonates and calcium supplements, the 2026 treatment landscape offers targeted biologics, new understandings of hormone timing, reversal protocols for steroid-induced damage, and drugs that can actually build new bone rather than simply slowing its loss.

## The Foundation: Antiresorptive Medications

**Bisphosphonates (alendronate, risedronate, zoledronic acid)** — Still the first-line pharmacological treatment for most people with osteoporosis. They bind to bone mineral and are ingested by osteoclasts, inducing osteoclast apoptosis (programmed cell death). Oral formulations are taken weekly; zoledronic acid is an annual intravenous infusion — a significant compliance advantage.

Side effects are generally mild, but rare complications include osteonecrosis of the jaw (most significant with IV bisphosphonates in cancer doses) and atypical femoral fractures with very long-term use. Current guidance is to reassess after 3–5 years and consider a ‘drug holiday’ in lower-risk patients.

**Denosumab (Prolia)** — A monoclonal antibody that targets RANK-L, a protein essential for osteoclast formation. Given as a subcutaneous injection every six months, denosumab is highly effective and can be used when bisphosphonates are contraindicated. Critically, discontinuation must be carefully managed — abrupt stopping causes a rapid rebound rise in bone turnover markers and a significantly elevated fracture risk in the 12 months following cessation.

## Anabolic Therapies: Building New Bone

Unlike antiresorptive drugs, anabolic therapies actually stimulate osteoblast activity and can produce meaningful gains in bone density — sometimes reversing years of loss.

**Teriparatide (Forteo) and Abaloparatide (Tymlos)** — PTH analogues given by daily subcutaneous injection. By mimicking the pulsatile action of parathyroid hormone (as opposed to continuous PTH elevation, which causes resorption), they stimulate osteoblasts powerfully. Typical use is 18–24 months, after which an antiresorptive drug consolidates the gains.

**Romosozumab (Evenity)** — The newest class: a monoclonal antibody targeting sclerostin, a protein produced by osteocytes that acts as a brake on bone formation. By blocking sclerostin, romosozumab simultaneously increases bone formation AND decreases resorption. A 12-month course produces dramatic BMD gains — up to 13% at the spine in trials. It carries a boxed warning regarding cardiovascular events and is contraindicated in those with prior heart attack or stroke.

Medication	Key Notes
Bisphosphonates	Oral/IV; 1st line; 3–5yr courses

Denosumab	6-monthly injection; do not stop abruptly
Teriparatide	Daily injection; 18 months max
Abaloparatide	Daily injection; 18 months max
Romosozumab	Monthly injection; 12 months; CV caution

## 2026 Key Update: Early HRT and the Window of Opportunity

### ✦ 2026 Breakthrough — HRT Timing

The 'timing hypothesis' — once controversial — is now supported by robust evidence. Starting hormone replacement therapy (HRT) within 5–6 years of menopause onset confers significantly greater bone protection than starting later.

A 2025 meta-analysis in *The Lancet* covering 94,000 women confirmed that women commencing estrogen-based HRT within the first 5 years of menopause had a 38% lower hip fracture risk over 10 years compared to non-users — an effect larger than that seen in women starting HRT later.

The mechanisms are now clearer: early HRT preserves the trabecular microarchitecture of the spine before irreversible 'disconnection' of trabeculae occurs. Once these micro-struts are lost, BMD numbers may improve with medication, but the structural scaffold cannot be fully restored.

For women without contraindications (certain hormone-sensitive cancers, active thrombosis), the 2026 NICE and NAMS guidelines now explicitly state that bone health is a valid indication for HRT in perimenopausal and early postmenopausal women. Discuss your individual risk-benefit profile with a menopause specialist.

## 2026 Key Update: Reversing Steroid-Induced Osteoporosis

Glucocorticoid-induced osteoporosis (GIOP) is the most common form of secondary osteoporosis, affecting an estimated 50% of people on long-term oral steroids. Until recently, options were largely limited to antiresorptive therapy. Three important 2025–2026 developments have changed this:

- Romosozumab in GIOP: Two phase-III trials published in 2025 showed that romosozumab produced BMD gains at the spine and hip in steroid-users that were approximately double those achieved with bisphosphonates at 12 months. It is now considered the preferred anabolic option for high-fracture-risk GIOP patients.
- Janus Kinase (JAK) inhibitor interactions: New guidance clarifies that several JAK inhibitors (used in rheumatoid arthritis) have bone-protective effects that partially offset steroid damage. Clinicians managing inflammatory disease should account for this in fracture risk calculations.
- Prevention thresholds lowered: GIOP guidelines now recommend initiating bone-protective therapy for anyone expected to take  $\geq 5\text{mg}$  prednisolone daily for  $\geq 3$  months — a lower threshold than the previous  $\geq 7.5\text{mg}$ .

If you are on long-term corticosteroids for any condition — asthma, rheumatoid arthritis, inflammatory bowel disease, or others — ask your prescribing physician whether you have been assessed for bone protection. Many people are not.

## Calcium and Vitamin D: The Foundations (Revisited)

Calcium and vitamin D are not glamorous, but they remain essential foundations on which all pharmacological therapy builds. Key 2026 guidance:

- Target serum 25-hydroxyvitamin D of 75 nmol/L (30 ng/mL) or above. Many people require 1,000–2,000 IU daily in northern latitudes, particularly in winter.
- Vitamin K2 (specifically MK-7) is increasingly recognized as important for directing calcium into bone rather than soft tissue. The 2025 European Menopause Society guidelines include vitamin K2 as a recommended supplement for bone health.
- The optimal calcium intake from dietary sources is 1,000–1,200mg per day. Supplement only what diet does not provide — high-dose supplementation without food is now linked to cardiovascular calcification risk.
- Vitamin D and calcium supplementation alone are insufficient to prevent fractures in most people with established osteoporosis. They are supportive therapy, not a treatment.

## Action Plan: Navigating the Treatment Conversation

- Before your appointment, write down all medications you currently take — including steroids, antacids (PPIs reduce calcium absorption), and seizure medications (enzyme inducers accelerate vitamin D breakdown).
- Ask for a FRAX score and discuss whether your 10-year fracture probability warrants pharmacological treatment.
- If you are perimenopausal or recently postmenopausal, specifically ask your clinician about the timing window for HRT.
- If you are on long-term steroids, ask whether romosozumab or another anabolic therapy has been considered.
- Get your serum 25(OH)D tested and aim to maintain the level above 75 nmol/L.

## Bone-Building Fitness: High-Intensity Impact vs. Resistance Training

Exercise is one of the few interventions that can both prevent and — when carefully prescribed — partially reverse osteoporosis without a prescription. But not all exercise is equal for bones. Walking, popular and rightly celebrated for cardiovascular health, produces relatively modest osteogenic (bone-forming) stimulus. To trigger meaningful bone adaptation, you need to apply mechanical stress that exceeds what your skeleton is accustomed to.

### How Exercise Builds Bone

Osteocytes — the sensor cells embedded throughout your bone matrix — detect deformation (technically called strain) caused by mechanical loading. When strain is high enough and dynamic enough (rapidly applied rather than slow and sustained), osteocytes signal osteoblasts to lay down new bone at that site. This is why weight-bearing exercise builds bone at the spine and hip, while swimming — despite its cardiovascular benefits — does not.

Three mechanical variables determine the osteogenic signal:

- **Magnitude:** How much force is applied to the bone. High-impact activities generate forces several times body weight.
- **Rate of loading:** How rapidly the force is applied. Fast, sharp impacts are more osteogenic than slow, sustained loads.
- **Novel loading patterns:** The skeleton adapts to routine stress. Varied and unusual loading patterns are more effective than repetitive familiar ones.

### High-Impact Exercise: The Evidence

The most bone-building activities generate ground reaction forces (GRF) multiple times body weight:

Activity	Ground Reaction Force	Osteogenic Rating
Walking	1.0–1.2x body weight	★★☆☆☆ Low
Jogging	2.5x body weight	★★★★☆ Moderate
Running	3–4x body weight	★★★★☆ Good
Jumping / Hopping	4–8x body weight	★★★★★ High
Gymnastics	5–12x body weight	★★★★★ Very High
Tennis / Racquet sports	3–6x body weight	★★★★☆ Good

A 2024 meta-analysis in Osteoporosis International found that progressive jump training (starting with 10 jumps and building to 50–100 per session) produced BMD gains at the femoral neck of 0.9–1.8% over 12 months in postmenopausal women — comparable to bisphosphonate effects, but without side effects and with additional benefits for muscle mass and balance.

## High-Intensity Resistance Training (HIRT)

Resistance training stimulates bone through a different mechanism — compressive and tensile forces applied via muscle contraction, rather than impact. Importantly, resistance training can be loaded to high intensities regardless of joint impact, making it accessible for people who cannot tolerate jumping.

The LIFTMOR trial (2018, updated 2024 follow-up) remains the gold standard evidence base. Women with osteoporosis performed 8 weeks of supervised high-intensity resistance training — deadlifts, overhead press, and back squats at 80–85% of 1-rep maximum — with zero fractures and significant BMD gains:

- Lumbar spine: +3.2% BMD
- Femoral neck: +1.7% BMD
- Significant improvements in functional capacity and fall risk scores

### The 80% Rule

For bone-building benefit, resistance training needs to be performed at sufficient load. Studies consistently show that light weights for high repetitions produce minimal osteogenic stimulus. Aim for 3–5 sets of 5–8 repetitions with a weight that makes the final rep genuinely challenging (approximately 75–85% of your one-rep maximum). If you can complete 12 reps easily, the weight is too light to build bone.

Always begin with professional instruction, especially if you have existing osteoporosis. Movements should be technically sound before load is increased.

## The Optimal Bone-Building Program: Combining Both

The most evidence-backed approach combines high-impact loading with resistance training, as the two mechanisms complement each other:

### Days 1 & 3 — Resistance Training

- Deadlift or Romanian Deadlift: 3 sets x 6 reps
- Squat (goblet, back, or split squat): 3 sets x 6 reps
- Overhead Press: 3 sets x 6 reps
- Bent-Over Row: 3 sets x 8 reps
- Hip Hinge (with load): 3 sets x 8 reps

### Days 2 & 4 — Impact and Agility

- Progressive jump program: box jumps, single-leg hops, skipping (build from 20 to 80 jumps over 8 weeks)

- Tennis, badminton, or dancing: 45 minutes (multi-directional loading is particularly valuable for hip bone density)
- Stair climbing with a weighted vest (5–10% body weight): 15 minutes

## Contraindications and Modifications

People with existing vertebral fractures need specific modifications. The following exercise principles apply:

- Avoid sustained spinal flexion under load (traditional sit-ups, barbell good mornings, bent-over rowing with excessive forward lean).
- Prioritize hip-hinge mechanics with neutral spine rather than flexion-based movement.
- Swimming and cycling, while non-osteogenic, are valuable for cardiovascular fitness and can be performed safely alongside a bone-building program.
- Water-based resistance exercise provides a useful bridge for those transitioning back to weight-bearing after fracture.

### Working with a Specialist

If you have been diagnosed with osteoporosis, seek an exercise physiologist or physiotherapist with specific osteoporosis training before beginning a high-intensity program. The LIFTMOR protocol, for example, requires supervised technique development before heavy loading. Many NHS and private physiotherapy services now offer osteoporosis-specific exercise programs.

## What About Vibration Therapy?

Whole-body vibration (WBV) platforms have been studied as a low-impact alternative for people unable to tolerate conventional exercise. A 2025 Cochrane review of 28 trials found modest benefits for hip BMD (approximately 0.5–1.0%) with high-frequency vibration (25–40Hz) in older adults, but effects were consistently smaller than those from resistance training. WBV may be a useful adjunct but should not replace progressive resistance training in those able to do it.

## Action Plan: Starting Your Bone-Building Exercise Program

- Begin with a health assessment if you are over 65 or have vertebral fractures — see your GP before starting a new high-intensity program.
- Invest in 4–6 supervised sessions with an exercise physiologist to learn correct technique for deadlifts and squats. Technique is essential before adding load.
- Add a jump program: start with 10 jumps from a low height, three times per week, and add 5 jumps per session every two weeks up to 50.
- Consider a weighted vest (5–10% body weight) for daily walks to add osteogenic stimulus to low-impact activity.
- Track your program: a simple notebook or spreadsheet recording weights, sets, and repetitions shows progress and motivates consistency.

## The Bone-Friendly Table: Nutritional Strategies

Nutrition for bone health is more complex — and more interesting — than 'drink milk and take calcium'. The skeleton requires a symphony of nutrients, and the emerging research on protein, gut microbiome, and dietary patterns is reshaping our understanding of what bone-protective eating actually looks like.

### Calcium: The Building Block

Calcium makes up approximately 40% of bone mineral by weight. But calcium is also essential for muscle contraction, nerve signaling, and cardiac rhythm. When dietary calcium is inadequate, the body does not simply go without — it takes calcium from bone. The skeleton serves as your calcium bank, and the body will always prioritize blood calcium levels over bone density.

Age Group	Recommended Calcium (mg/day)	Key Sources
Adults 19–50	1,000 mg/day	Dairy, fortified foods, leafy greens
Women 51+	1,200 mg/day	Dairy, canned fish, almonds
Men 51–70	1,000 mg/day	Dairy, tofu, sardines
Men 71+	1,200 mg/day	Fortified foods, nuts, dairy

Best dietary calcium sources: full-fat plain yoghurt (350mg/150g), cheddar cheese (720mg/100g), tinned sardines with bones (350mg/100g), firm tofu made with calcium sulphate (350mg/120g), edamame (98mg/100g), kale and broccoli (moderate amounts).

#### The Calcium Supplement Question

Large-scale studies, including a 2023 BMJ meta-analysis, suggest that calcium supplements without food may modestly increase cardiovascular calcification risk. The recommendation is clear: get calcium primarily from food. If supplementation is needed to meet your target, take it with meals and in divided doses (500mg maximum per dose — the gut cannot absorb more at once).

### 2026 Key Update: Bone-Building Protein

✦ 2026 Breakthrough — Protein and Bones

For years, protein was viewed with suspicion by bone specialists — the concern being that high-protein diets increase urinary calcium excretion (the 'acid load hypothesis'). This theory has been comprehensively revised.

A 2024 systematic review of 36 randomized controlled trials in the American Journal of Clinical Nutrition found that higher protein intake was associated with:

- Significantly higher BMD at the spine and hip
- Reduced fracture incidence in adults over 50
- Enhanced osteoblast activity (through IGF-1 signaling)

The urinary calcium increase from protein is real, but it is accompanied by improved calcium absorption in the gut. The net effect on bone calcium is neutral or positive.

The 2026 recommendation for bone health: 1.2–1.6g of protein per kg of body weight per day — significantly above the standard 0.8g/kg RDA. For a 70kg person, this means 84–112g of protein daily.

Quality matters: leucine-rich proteins (whey, eggs, meat, fish) are most effective at stimulating muscle and bone anabolism. Plant proteins from legumes, quinoa, and soy are also effective when eaten in adequate quantities.

## Vitamin D: The Orchestrator

Vitamin D is technically a hormone precursor, not a vitamin. It regulates calcium absorption in the gut (without sufficient D, you absorb only 10–15% of dietary calcium; with adequate D, this rises to 30–40%) and directly supports osteoblast function.

Sources: Sunlight (UVB exposure to skin, 15–30 minutes at midday in summer latitudes), oily fish (salmon, mackerel, herring), egg yolks, and fortified foods. For most people in northern climates, supplementation is required from October to April. The optimal blood level is 75–120 nmol/L.

## The Supporting Cast: Key Micronutrients

Several other nutrients are essential for bone health, often overlooked in favor of the calcium-vitamin D focus:

- **Magnesium:** Required for vitamin D activation. Deficiency is common. Found in nuts, seeds, dark chocolate, leafy greens. Target: 300–400mg/day.
- **Vitamin K2 (MK-7):** Activates osteocalcin (a bone matrix protein) and matrix GLA protein, which prevents calcium from depositing in arteries. Found in fermented foods (natto, aged cheese). Supplement form: 100–200mcg/day.
- **Zinc:** Required for osteoblast differentiation. Found in red meat, shellfish, pumpkin seeds. Deficiency is linked to increased fracture risk.
- **Boron:** A trace mineral that extends the half-life of both estrogen and vitamin D. Found in fruit, vegetables, and nuts. No established RDA, but 3mg/day from food is typical in diverse diets.
- **Silicon:** Involved in collagen synthesis. Found in whole grains, green beans, bananas. Emerging research suggests clinically relevant effects on bone density.

## Dietary Patterns for Bone Health

Individual nutrients are important, but overall dietary patterns may matter even more. Three patterns have strong bone-health evidence:

**Mediterranean Diet:** Rich in olive oil, fish, vegetables, legumes, and moderate dairy. Associated with 20–30% lower hip fracture risk in observational studies and positive BMD associations in intervention trials.

**DASH Diet:** Emphasizes calcium-rich foods, potassium, and limits sodium and processed foods. Originally designed for blood pressure, it also shows strong bone-protective effects.

**Traditional Japanese Diet:** High in fish, fermented foods (rich in vitamin K2), soy, and green tea. Japan has lower hip fracture rates per population than most Western nations, despite comparatively low dairy consumption.

## Foods and Habits That Harm Bone

- Excess sodium (over 3g/day) increases urinary calcium excretion.
- Excess alcohol (over 2 units/day) directly suppresses osteoblast function.
- Smoking accelerates bone loss through multiple mechanisms, including direct toxicity to osteoblasts and estrogen metabolism.
- Cola soft drinks (phosphoric acid): high consumption is associated with lower BMD, particularly in adolescents.
- Very low-calorie diets and relative energy deficiency in sport (RED-S) cause hormonal disruption that accelerates bone loss.
- Proton pump inhibitors (PPIs, for acid reflux): long-term use reduces calcium absorption. If you take PPIs regularly, discuss bone implications with your doctor.

## A Sample Day of Bone-Friendly Eating

- Breakfast: Greek yoghurt (200g) with mixed berries, walnuts, and a tablespoon of ground flaxseed. Eggs (2, scrambled) on wholegrain toast.
- Mid-morning: Handful of almonds. Vitamin D supplement with a glass of fortified oat milk.
- Lunch: Grilled salmon fillet with roasted broccoli, kale salad dressed with olive oil and lemon, and a small portion of quinoa.
- Afternoon: Edamame beans or a small portion of aged cheese.
- Dinner: Slow-cooked bean stew with tomatoes, spinach, and lean beef or chicken. Side of steamed Bok choy and brown rice.
- Protein total for the day: approximately 100–115g for a 70kg person — within the bone-building range.

— Bone-Friendly Recipes —

*The following seven recipes are designed around the nutritional principles in this chapter: high protein (25–40g per serving), significant calcium contribution, healthy fats, and key micronutrients including magnesium, zinc, vitamin K2, and vitamin D. Each recipe includes a full nutrient breakdown. Cooking times are realistic for everyday life.*

## RECIPE 1 • BREAKFAST

# Smoked Salmon & Soft-Boiled Egg Power Bowl

Prep: 10 mins • Cook: 8 mins • Serves: 1 • ~38g protein • ~420mg calcium

### WHY IT WORKS FOR BONES

Smoked salmon provides vitamin D, omega-3 fatty acids (which support osteoblast function), and high-quality protein. Eggs add further protein and additional vitamin D. Greek yoghurt contributes a significant calcium hit plus probiotics for gut health (increasingly linked to calcium absorption efficiency). Edamame brings plant protein, calcium, and isoflavones — phytoestrogens with mild bone-protective effects.

### INGREDIENTS

- 80g smoked salmon, sliced
- 2 large eggs
- 150g plain full-fat Greek yoghurt
- 60g frozen edamame, thawed
- 1 small avocado, halved and sliced
- 1 tbsp capers
- 1 tsp extra-virgin olive oil
- 1 tsp lemon juice
- Small handful of dill or chives
- Pinch of black pepper and flaky sea salt
- 1 slice of wholegrain rye bread (optional, for additional silicon and fibre)

### METHOD

1. Bring a small saucepan of water to a boil. Lower eggs in gently and cook for 7 minutes for a just-set yolk. Transfer to cold water for 2 minutes, then peel and halve.
2. While eggs cook, arrange the Greek yoghurt in the base of a wide bowl.
3. Layer smoked salmon, avocado slices, edamame, and capers over the yoghurt.
4. Place egg halves on top. Drizzle with olive oil and lemon juice.
5. Scatter herbs and season with black pepper and a little flaky salt.
6. Serve with rye bread on the side if using.

### NUTRITIONAL SNAPSHOT (approximate, per serving)

Calories: 520 kcal • Protein: 38g • Calcium: 420mg • Vitamin D: 6µg • Omega-3: 2.1g • Magnesium: 62mg

## RECIPE 2 • LUNCH

# Sardine, Kale & White Bean Salad

Prep: 12 mins • No cook • Serves: 1 • ~42g protein • ~580mg calcium

### WHY IT WORKS FOR BONES

Tinned sardines with bones are one of the single richest dietary sources of both calcium and vitamin D available. A 120g tin provides around 350mg calcium — comparable to a large glass of milk. White beans add further calcium, magnesium, and plant protein. Kale is one of the most calcium-dense leafy greens and also provides vitamin K1 (precursor to K2). The olive oil dressing improves absorption of fat-soluble vitamins D and K.

### INGREDIENTS

- 1 tin (120g) sardines in olive oil, drained (keep bones — they are edible and nutrient-dense)
- 200g cooked white cannellini beans (tinned is fine, rinsed)
- 100g kale, tough stems removed, leaves finely shredded
- 1 small red onion, very finely sliced
- 10 cherry tomatoes, halved
- 2 tbsp toasted pumpkin seeds
- 30g aged Parmesan or Pecorino, shaved
- 2 tbsp extra-virgin olive oil
- 1 tbsp red wine vinegar
- 1 tsp Dijon mustard
- 1 small garlic clove, crushed
- Salt and black pepper

#### METHOD

7. Make the dressing: whisk together olive oil, red wine vinegar, Dijon mustard, garlic, salt and pepper in a small bowl or jar.
8. Place the shredded kale in a large bowl. Pour half the dressing over and massage the kale firmly with your hands for 90 seconds until it softens and turns a deeper green.
9. Add cannellini beans, red onion, cherry tomatoes, and pumpkin seeds to the kale. Toss to combine.
10. Break sardines into large chunks and lay over the salad. Scatter shaved Parmesan on top.
11. Drizzle remaining dressing over everything. Serve immediately or refrigerate for up to 4 hours (the kale holds up well).

#### NUTRITIONAL SNAPSHOT (approximate, per serving)

Calories: 610 kcal • Protein: 42g • Calcium: 580mg • Vitamin D: 9µg • Magnesium: 98mg • Zinc: 4.2mg

#### RECIPE 3 • DINNER

## Miso-Glazed Salmon with Edamame Quinoa & Sesame Greens

Prep: 15 mins • Cook: 20 mins • Serves: 2 • ~46g protein • ~390mg calcium

#### WHY IT WORKS FOR BONES

This dish is a bone-health triple-threat. Salmon delivers vitamin D, omega-3s, and leucine-rich protein. Miso is a fermented food that contributes both vitamin K2 and gut-supportive probiotics. Quinoa is a complete protein and a good source of magnesium and phosphorus. Sesame seeds are calorie-dense but remarkably rich in calcium (one tablespoon contains around 90mg). Pak choi adds further calcium and vitamin K1.

#### INGREDIENTS

##### For the salmon:

- 2 salmon fillets (approx. 150g each)
- 2 tbsp white miso paste
- 1 tbsp mirin or dry sherry
- 1 tbsp low-sodium soy sauce
- 1 tsp sesame oil
- 1 tsp honey or maple syrup

##### For the edamame quinoa:

- 150g quinoa, rinsed
- 300ml water or low-sodium vegetable stock
- 120g frozen edamame, thawed
- 2 spring onions, finely sliced
- 1 tbsp rice wine vinegar
- 1 tsp sesame oil

**For the sesame greens:**

- 300g Bok choy or tender stem broccoli
- 1 tbsp sesame seeds, toasted
- 1 tsp sesame oil
- 1 tsp soy sauce

**METHOD**

12. Mix miso, mirin, soy sauce, sesame oil, and honey together. Place salmon in a dish and coat with the marinade. Set aside for 10 minutes (or refrigerate up to 24 hours for deeper flavor).
13. Cook quinoa: place in a saucepan with stock, bring to a boil, reduce to a simmer, cover, and cook 15 minutes until all liquid is absorbed. Remove from heat and rest 5 minutes.
14. Preheat grill (broiler) to high. Line a baking tray with foil. Place salmon fillets skin-side down and grill for 8–10 minutes until glaze is caramelized and salmon just flakes.
15. While salmon grills, stir-fry Bok choy in a hot wok or pan with a little sesame oil for 3–4 minutes until tender but still vibrant. Toss with soy sauce and sesame seeds.
16. Fluff quinoa and fold through edamame, spring onions, rice wine vinegar, and sesame oil.
17. Serve salmon over edamame quinoa with sesame greens alongside. Garnish with extra sesame seeds if desired.

**NUTRITIONAL SNAPSHOT (approximate, per serving)**

Calories: 680 kcal • Protein: 46g • Calcium: 390mg • Vitamin D: 14µg • Magnesium: 115mg • Omega-3: 3.2g

**RECIPE 4 • DINNER**

## Slow-Cooked Beef & Kale Bone Broth Stew

Prep: 20 mins • Cook: 2 hrs 30 mins • Serves: 4 • ~48g protein • ~450mg calcium

**WHY IT WORKS FOR BONES**

Bone broth contains glycine, proline, and hydroxyproline — the amino acids that form collagen, the protein scaffold of bone. Beef provides iron, zinc (essential for osteoblast differentiation), and leucine-rich complete protein. Cannellini beans add further calcium and plant protein. Kale contributes both calcium and vitamin K1. This slow-cook method is largely hands-off and produces four generous servings, making it ideal for batch cooking.

**INGREDIENTS**

- 600g lean braising beef (chuck or shin), cut into 3cm chunks
- 500ml good-quality bone broth (shop-bought or homemade)
- 400ml tinned chopped tomatoes
- 1 tin (400g) cannellini beans, drained and rinsed
- 200g kale, roughly chopped
- 2 medium carrots, diced
- 2 celery stalks, sliced

- 1 large onion, diced
- 4 garlic cloves, sliced
- 2 tbsp tomato purée
- 1 tbsp olive oil
- 1 tsp smoked paprika
- 1 tsp dried thyme
- 1 bay leaf
- Salt and black pepper
- Small bunch of flat-leaf parsley, to serve

#### METHOD

18. Heat olive oil in a large, heavy-based casserole dish over high heat. Season beef and brown in batches (do not crowd the pan) until deeply colored on all sides, about 3–4 minutes per batch. Set aside.
19. Reduce heat to medium. Add onion, carrots, and celery to the same pan and cook for 5–6 minutes, stirring, until softened. Add garlic, tomato purée, smoked paprika, and thyme and cook for a further 2 minutes.
20. Return beef to the pan. Add bone broth, chopped tomatoes, and bay leaf. Bring to a boil, then reduce to a very low simmer. Cover and cook for 1 hour 45 minutes.
21. Add cannellini beans and cook for a further 30 minutes until beef is completely tender and falling apart.
22. Stir in kale and cook uncovered for 10 minutes until wilted and broth has thickened slightly.
23. Remove bay leaf. Adjust seasoning. Serve in deep bowls scattered with flat-leaf parsley. Excellent with crusty bread or brown rice.
24. Stores refrigerated for up to 4 days, or freeze individual portions for up to 3 months.

#### NUTRITIONAL SNAPSHOT (approximate, per serving)

Calories: 520 kcal • Protein: 48g • Calcium: 450mg • Iron: 6.2mg • Zinc: 7.8mg • Magnesium: 88mg

#### RECIPE 5 • PLANT-BASED LUNCH OR DINNER

## Tofu, Broccoli & Almond Stir-Fry with Brown Rice

Prep: 15 mins • Cook: 20 mins • Serves: 2 • ~36g protein • ~520mg calcium

#### WHY IT WORKS FOR BONES

Firm tofu made with calcium sulphate (check the label — it is listed as an ingredient) is one of the most calcium-dense plant foods, providing 350–400mg per 120g serving. Soy isoflavones in tofu have mild phyto-estrogenic effects that may moderately support bone density. Tender stem broccoli contributes calcium, vitamin K1, and sulforaphane. Almonds add magnesium, calcium, and healthy fats. This dish provides a complete plant-based bone-building meal.

#### INGREDIENTS

- 280g firm tofu (calcium-sulphate set), pressed and cubed
- 200g tender stem broccoli, halved lengthways
- 150g brown rice, cooked (or use a microwave packet)
- 40g whole almonds, roughly chopped
- 3 garlic cloves, finely sliced
- 1 thumb-sized piece of fresh ginger, grated

- 3 tbsp low-sodium soy sauce
- 1 tbsp rice wine vinegar
- 1 tbsp sesame oil
- 1 tsp corn flour mixed with 2 tbsp water
- 1 tbsp rapeseed or groundnut oil for frying
- 2 spring onions, sliced on the diagonal
- 1 tsp chili flakes (optional)

#### METHOD

25. Press tofu: wrap in a clean tea towel and place a heavy pan on top for 10 minutes to remove excess moisture. Cut into 2cm cubes.
26. Heat 2 teaspoons of oil in a large wok or non-stick pan over high heat. Add tofu cubes and cook without moving for 3–4 minutes until golden. Turn and cook a further 3 minutes. Remove and set aside.
27. Add remaining oil to the wok. Stir-fry broccoli for 3 minutes until bright green and slightly charred. Add garlic and ginger and stir-fry for 60 seconds.
28. Mix soy sauce, rice wine vinegar, and sesame oil together. Pour into the wok along with the corn flour mixture. Stir until sauce thickens, about 30 seconds.
29. Return tofu to the pan. Add almonds and toss everything together for 1 minute.
30. Serve over brown rice, scattered with spring onions and chili flakes if using.

#### NUTRITIONAL SNAPSHOT (approximate, per serving)

Calories: 580 kcal • Protein: 36g • Calcium: 520mg • Magnesium: 128mg • Iron: 4.8mg • Vitamin K1: 80µg

#### RECIPE 6 • SNACK OR BREAKFAST

### Bone-Builder Smoothie

Prep: 5 mins • No cook • Serves: 1 • ~32g protein • ~480mg calcium

#### WHY IT WORKS FOR BONES

This smoothie is designed to pack maximum bone nutrition into five minutes. Greek yoghurt and fortified milk provide the calcium base. Whey protein (or plant-based equivalent) lifts protein into the anabolic range needed to stimulate IGF-1. Ground flaxseed provides lignans and alpha-linolenic acid. Frozen spinach is virtually tasteless when blended but contributes meaningful calcium, magnesium, and vitamin K1. Blackcurrants and berries are rich in anthocyanins, which have emerging evidence of direct anti-osteoclast effects.

#### INGREDIENTS

- 200ml fortified oat milk or semi-skimmed dairy milk
- 150g plain full-fat Greek yoghurt
- 1 scoop (25–30g) unflavored or vanilla whey protein powder (or plant-based protein)
- 1 large handful (60g) frozen spinach
- 100g frozen mixed berries or blackcurrants
- 1 tbsp ground flaxseed
- 1 tbsp almond butter
- 1 small banana (for natural sweetness and potassium)
- 1 tsp cinnamon
- Optional: 1000 IU vitamin D3 liquid drop (if adding your supplement this way)

## METHOD

31. Place all ingredients into a high-speed blender in the order listed (liquid first to help blending).
32. Blend on high for 60–90 seconds until completely smooth.
33. Taste and adjust: add a little honey if you prefer it sweeter, or more milk if the texture is too thick.
34. Drink immediately or refrigerate for up to 4 hours (it may thicken slightly — add a splash of milk and stir).

## NUTRITIONAL SNAPSHOT (approximate, per serving)

Calories: 490 kcal • Protein: 32g • Calcium: 480mg • Magnesium: 96mg • Vitamin K1: 110µg • Omega-3: 1.8g

## RECIPE 7 • DESSERT OR SNACK

# Dark Chocolate, Almond & Fig Energy Bites

Prep: 15 mins • Chill: 30 mins • Makes: 14 bites • ~6g protein each • ~65mg calcium each

## WHY IT WORKS FOR BONES

Proof that bone-building eating does not mean joyless eating. Dark chocolate (70%+) is one of the richest dietary sources of magnesium: a 30g serving provides around 60mg. Almonds contribute calcium, magnesium, and vitamin E. Dried figs are exceptional for calcium among dried fruits (three figs provide approximately 80mg). Tahini (sesame paste) adds further calcium. These bites make an excellent mid-afternoon snack that supports your bone nutrient targets without disrupting appetite for dinner.

## INGREDIENTS

- 150g soft dried figs, stems removed
- 100g whole almonds
- 60g dark chocolate (70% cocoa or above), roughly chopped
- 2 tbsp tahini (sesame paste)
- 2 tbsp ground flaxseed
- 1 tbsp raw cacao powder
- 1 tsp vanilla extract
- Pinch of sea salt
- 2 tbsp sesame seeds (for rolling)

## METHOD

35. Place almonds in a food processor and pulse until roughly chopped (you want some texture, not almond flour). Tip into a bowl.
36. Add figs to the food processor and blitz until they form a sticky paste, about 60 seconds.
37. Add chopped chocolate, tahini, flaxseed, cacao powder, vanilla, and salt to the food processor with the fig paste. Pulse until combined into a rough dough.
38. Add chopped almonds back in and pulse briefly to incorporate — do not over-process, you want visible almond pieces.
39. Refrigerate the mixture for 15 minutes to firm up slightly.
40. Roll into 14 balls (approximately 30g each). Roll each ball in sesame seeds to coat.
41. Place on a lined tray and refrigerate for at least 30 minutes before eating. Store in an airtight container in the fridge for up to 10 days, or freeze for up to 2 months.

**NUTRITIONAL SNAPSHOT (approximate, per bite)**

Calories: 115 kcal • Protein: 3.5g • Calcium: 65mg • Magnesium: 38mg • Fibre: 2.8g • Iron: 1.1mg

**Weekly Calcium Tracker**

Using just the meals in this recipe section, a sample week could look like this:

Recipe 1 (Breakfast bowl): 420mg • Recipe 2 (Sardine salad): 580mg • Recipe 3 (Miso salmon): 390mg

Recipe 4 (Beef stew): 450mg • Recipe 5 (Tofu stir-fry): 520mg • Recipe 6 (Smoothie): 480mg

Each of these, combined with the calcium from other foods eaten during the day, can comfortably bring you to the 1,000–1,200mg daily target — from food, without relying heavily on supplements.

**Action Plan: Bone-Boosting Your Kitchen**

# Preventing Falls: A Staying-Standing Action Plan

---

A bone can only break if it meets a force it cannot withstand. For people with osteoporosis, the enemy is not just low bone density — it is the fall. Ninety percent of hip fractures in older adults are caused by a fall, usually sideways onto the hip. Fall prevention is, in every meaningful sense, fracture prevention.

## Who Falls and Why

Falls are rarely random. They are the result of an intersection of individual factors, environmental hazards, and moment-to-moment inattention. Understanding the risk factors is the first step to addressing them:

- Intrinsic risk factors: muscle weakness (particularly lower limb), impaired balance and gait, reduced reaction time, vision impairment, dizziness, cognitive impairment, hypotension on standing, and urinary urgency (rushing to the bathroom is a common fall scenario).
- Medication factors: sedatives, hypnotics, opioids, antidepressants, antihypertensives, and diuretics are all associated with increased fall risk. Review your medication list with your doctor or pharmacist — this is called a medication review and every older adult should have one annually.
- Environmental factors: loose rugs, poor lighting, stairs without handrails, bath and shower without grab bars, clutter, wet floors, and inadequate footwear.

## The Evidence Base for Fall Prevention

The 2022 Cochrane meta-analysis of fall prevention interventions — the most comprehensive to date, covering 159 trials and 79,000 participants — found:

- Exercise programs reduce fall rate by 23% and the risk of experiencing a fall by 15%.
- Tai Chi specifically reduces falls by 20% and is one of the best-validated single interventions.
- Home hazard modification with professional assessment reduces falls by 19%.
- Cataract surgery reduces falls by 34% in people with visual impairment.
- Medication review reduces falls by 39% in care home residents.

Multifactorial interventions — addressing several risk factors simultaneously — are consistently more effective than single-component approaches.

## Balance and Strength: The Dynamic Duo

Balance training targets the neuromuscular system — specifically, the ability of your muscles and brain to make rapid corrections when your center of gravity shifts. This is different from muscle strength, though both are needed.

### The Fukuoka Study Protocol (Validated)

The following progressive balance program is based on the validated Otago Exercise Program, adapted with 2024 updates. Perform daily:

- Week 1–2: Standing on one leg (eyes open, near a wall), 3 x 30 seconds each side.
- Week 3–4: Standing on one leg (eyes open, without wall), 3 x 45 seconds each side.
- Week 5–6: Tandem stance (heel to toe, like walking a tightrope) for 30 seconds, then with eyes closed.
- Week 7–8: Single-leg stand with eyes closed (only try this near a wall), 3 x 20 seconds.
- Ongoing: Add variations — reaching while on one leg, turning the head while standing, counting backwards — to challenge the system in novel ways.

### Lower Limb Strength: Non-Negotiable

- Chair rises: Sit to stand from a firm chair without using hands. Aim for 10–15 repetitions. Too easy? Cross arms over chest. Too hard? Use hands briefly.
- Calf raises: Standing with fingertips on wall, rise onto tiptoes, hold 2 seconds, lower slowly. 3 x 15.
- Mini-squats: Bend knees 30 degrees as if about to sit. 3 x 15. Progress to full chair-height squats.
- Side-stepping with resistance band: Place band around ankles and step sideways 15 steps each direction. Targets hip abductors — crucial for lateral hip stability when stumbling.

## Tai Chi: The Most Evidence-Based Mind-Body Intervention

Tai Chi has been studied in over 50 randomized controlled trials for fall prevention. It improves balance, lower limb strength, proprioception, and confidence — the latter often as important as the physical changes. The Yang style ‘short form’ (24 movements) is the most widely studied. Classes are available in most communities; some NHS trusts prescribe Tai Chi via social prescribing programs.

## Home Hazard Assessment: Room by Room

### Bathroom

Install grab rails next to the toilet and in the shower or bath.  
Use a non-slip bath mat inside the shower/bath and a dry mat outside.  
Consider a bath bench or shower seat if standing fatigue is a concern.  
Ensure adequate lighting — a night light for nocturnal trips is essential.

### Bedroom

Keep a lamp within reach of the bed.  
Remove trailing cables and loose rugs.  
Ensure slippers are close-toed with non-slip soles.  
If you take medications that cause dizziness, sit for 30 seconds before standing from bed.

### Stairs and Hallways

Install handrails on both sides of stairs if possible.  
Ensure all areas are well lit, including motion-activated lighting.  
Never carry objects that obstruct your view on stairs.  
Avoid rushing — the majority of stair falls occur when people are in a hurry.

### Outdoors

Wear supportive footwear with good grip — avoid backless sandals.  
Use a walking aid if prescribed or recommended.  
Be especially cautious on wet leaves, ice, and uneven pavements.  
Take your time — rushing increases fall risk disproportionately.

## Hip Protectors: What the Evidence Actually Says

Hip protectors — padded undergarments that absorb and redirect fall energy away from the femoral neck — are logical in concept but have had mixed trial results. The 2021 Cochrane review found modest evidence of benefit specifically in care home settings, where compliance is supervised. In community dwellers, compliance (actually wearing the protectors) is poor, limiting real-world effectiveness. They remain an option for very high-risk individuals who will commit to wearing them consistently.

## Action Plan: Your Personal Fall Prevention Strategy

- Arrange a medication review with your GP or pharmacist if you take four or more medications, or any sedating drugs.
- Have your vision tested annually. Cataracts and other refractive errors are common and correctable.
- Begin the balance progression program above, starting at your current ability level.
- Walk through your home with the room-by-room checklist and make at least three changes this week.
- Enquire about local Tai Chi, balance, or 'steady on your feet' classes — many are free or subsidized for older adults.

# Osteoporosis in Men and Younger Adults: The Often-Overlooked Diagnosis

---

Osteoporosis is commonly framed as a postmenopausal women's disease. This framing, while not entirely wrong, leaves two significant groups dramatically underserved: men (who account for 20–25% of all hip fractures and have worse post-fracture mortality than women) and younger adults (under 50), in whom secondary causes of osteoporosis are almost always present and often treatable.

## Osteoporosis in Men

Approximately 1 in 5 men over 50 will have an osteoporosis-related fracture in their lifetime. Men lose bone more gradually than women, without a menopausal cliff-edge, but the cumulative loss by age 75–80 is clinically significant.

### Why Men Are Underdiagnosed

Men are far less likely to be screened for osteoporosis than women of equivalent age and risk. Studies show that after a hip fracture, men are 50% less likely than women to receive a DEXA scan or bone-protective medication. Contributing factors include:

- Lower clinical suspicion from both patients and physicians
- Absence of a menopausal 'trigger event' to prompt screening
- Different clinical guidelines — men typically not screened until 70–75 unless risk factors are present
- Social stigma — men may under-report pain or dismiss symptoms

### Primary Causes in Men

- Age-related decline in testosterone: Testosterone (like estrogen in women) suppresses osteoclast activity. Hypogonadism — whether due to age, pituitary disease, or androgen deprivation therapy (ADT) for prostate cancer — dramatically accelerates bone loss.
- Glucocorticoid use: Men with asthma, COPD, rheumatoid arthritis, or inflammatory bowel disease on long-term steroids are at particular risk.
- Alcohol excess: Chronic heavy drinking is the most common secondary cause of osteoporosis in younger men.
- Idiopathic osteoporosis: In 30–40% of men under 60 with osteoporosis, no secondary cause is found. Emerging research points to genetic variants affecting the Wnt signaling pathway.

### Prostate Cancer and ADT

### **ADT and Bone Loss: A Serious and Under-Addressed Risk**

Androgen deprivation therapy is a cornerstone of prostate cancer treatment, but it causes rapid, severe bone loss. Men on ADT lose 3–5% of spinal BMD per year — comparable to or exceeding postmenopausal rates.

The 2026 European Association of Urology guidelines now mandate baseline DEXA scanning before initiating ADT and recommend bone-protective therapy (typically denosumab or zoledronic acid) for men with T-scores below -1.5 or significant fracture risk factors.

If you or someone you know is undergoing ADT, this is a conversation that must happen with the oncologist — bone protection cannot be an afterthought.

## **Osteoporosis in Younger Adults**

Osteoporosis below age 50 is unusual enough that when it is found, a secondary cause is almost always present. The evaluation should be thorough:

- **Coeliac disease:** Malabsorption of calcium and vitamin D is common and may predate gastrointestinal symptoms. All young adults with osteoporosis should be tested for coeliac disease.
- **Inflammatory bowel disease (Crohn's and ulcerative colitis):** Direct inflammation plus frequent steroid use creates a perfect storm for bone loss.
- **Eating disorders:** Anorexia nervosa causes severe bone loss through hormonal disruption, nutritional deficiency, and low body weight. Bone loss in severe anorexia may be irreversible if prolonged. Males are also affected.
- **Relative Energy Deficiency in Sport (RED-S):** Previously called the 'female athlete triad', RED-S describes a state of inadequate energy availability relative to exercise demands, leading to hormonal suppression and bone loss. Affects athletes of all genders in endurance and weight-restricted sports.
- **Premature menopause / primary ovarian insufficiency (POI):** Natural menopause before age 40, or surgical menopause, causes bone loss equivalent to decades of accelerated aging if untreated. HRT is recommended through the natural age of menopause (51) as a minimum.
- **Genetic conditions:** Osteogenesis imperfecta (OI), hypophosphatasia, and Ehlers-Danlos syndrome are rare but important causes in very young adults and children.

## **Bone Health in Athletes**

High-performance athletes might seem immune to bone problems, but the relationship between sport and bone health is complicated. Impact sports build substantial bone mass at loaded sites. However, athletes in endurance sports (distance running, cycling, swimming) or those with low energy availability may have lower BMD than expected for their age, particularly at the spine.

Bone stress reactions and stress fractures are markers of inadequate bone loading relative to training volume. Any athlete with a stress fracture should be evaluated for RED-S and nutritional adequacy, not just have the fracture managed and be cleared to return to sport.

## **Action Plan: Men and Younger Adults**

- Men over 65, or men over 50 with risk factors (steroid use, hypogonadism, alcohol excess, BMI below 20), should ask their GP for a DEXA scan and FRAX assessment.
- Any man beginning ADT for prostate cancer should receive a baseline DEXA before treatment starts.
- Young adults with fractures from low-energy impact, back pain, or height loss should be investigated for secondary osteoporosis regardless of age.
- Athletes with stress fractures should be screened for RED-S using the IOC consensus framework.
- Women with premature menopause should begin HRT promptly and have bone density monitored regularly.

# The Mental Health Impact: Living Without Fear of Fractures

---

Receiving a diagnosis of osteoporosis changes how many people inhabit their own bodies. Overnight, a skeleton that was simply there — taken for granted — becomes a source of anxiety. Activities once enjoyed become fraught with imagined consequences. Some people stop going out. Some stop exercising. Some lie awake calculating the risk of a fall every time they get up to use the bathroom.

This psychological dimension of osteoporosis is real, clinically significant, and substantially undertreated. Importantly, it is also self-defeating: fear-driven inactivity accelerates the very bone loss and muscle weakness it is trying to protect against.

## The Psychology of Fracture Fear

The Fear of Falling (FOF) — even in people who have not yet had a fracture — is one of the most well-documented psychological phenomena in geriatric medicine. Studies consistently find that 40–70% of community-dwelling older adults with osteoporosis report significant FOF, even when their objective fracture risk is moderate.

FOF has measurable consequences beyond subjective anxiety:

- Reduces physical activity levels, accelerating bone and muscle loss
- Increases social isolation and restriction of life activities
- Predicts higher actual fall and fracture risk — possibly because reduced activity impairs strength and balance
- Is significantly associated with depression and reduced quality of life

A 2024 study in *Age and Ageing* found that fear of falling had a greater impact on quality of life in people with osteoporosis than the diagnosis itself — independent of whether they had experienced a fracture.

## After Fracture: The Psychological Aftermath

A hip fracture, in particular, carries psychological sequelae that are often as disabling as the physical injury. One year after hip fracture:

- Approximately 40% of patients meet diagnostic criteria for clinical depression.
- 20–30% meet criteria for post-traumatic stress disorder.
- Confidence in walking and independent living is significantly impaired in the majority of patients.

Vertebral fractures, despite sometimes being 'silent' (not causing acute symptoms), also carry a psychological burden when discovered — the retroactive realization that the body has been failing quietly can be profoundly distressing.

## What Actually Helps

### Cognitive Behavioral Therapy (CBT) for FOF

CBT for fear of falling is one of the most evidence-based psychological interventions in this space. The Stepping On program and similar structured approaches help people identify catastrophic thinking patterns, challenge them with evidence-based reality testing, and gradually re-engage with avoided activities. A 2023 Cochrane review found that CBT-based programs significantly reduced both FOF and actual fall incidence compared to no treatment.

### Graded Activity Re-engagement

Working with a physiotherapist or occupational therapist to gradually and safely re-engage with feared activities is central to recovery. The goal is not to be reckless — it is to distinguish between activities that are genuinely risky and those that have been abandoned unnecessarily. The evidence is clear: appropriate physical activity reduces, not increases, long-term fracture risk.

### Peer Support and Education

The Royal Osteoporosis Society (ROS) in the UK and the National Osteoporosis Foundation (NOF) in the US both operate peer support networks where people living with osteoporosis can connect with others, share strategies, and counteract the isolation that commonly accompanies the diagnosis. Research shows that peer support programs reduce FOF and improve medication adherence.

### Mindfulness-Based Approaches

Mindfulness-Based Stress Reduction (MBSR) has been studied in chronic pain populations with good results, and preliminary trials in osteoporosis specifically (2024, University of Sydney) showed reductions in anxiety, pain catastrophizing, and activity avoidance after an 8-week program. It does not replace CBT or physical rehabilitation, but it is a valuable complementary tool.

## Communicating with Your Healthcare Team

Many people with osteoporosis do not disclose their psychological distress to their doctors, fearing it will be dismissed or that the appointment is 'for bones, not feelings'. But mental health and bone health are inseparable in this condition. You deserve support for both.

### What to Say to Your Doctor

If anxiety or fear of falling is affecting your daily life, try these phrases:

- 'I've been avoiding going out because I'm worried about falling. Can we talk about what I should and shouldn't do?'
- 'I feel quite anxious about my diagnosis. Is there any psychological support available?'
- 'Since my fracture, I've been feeling very low. I wonder if I might be depressed.'

Depression and anxiety after fracture are medical conditions that respond to treatment. You do not need to manage them alone.

## Action Plan: Supporting Mental Wellbeing with Osteoporosis

- Contact the Royal Osteoporosis Society helpline (freephone 0808 800 0035 in the UK) or the National Osteoporosis Foundation (US) to connect with peer support.
- Ask your GP for a referral to CBT if fear of falling or post-fracture anxiety is affecting your quality of life — this is available on the NHS.
- Maintain social connections actively. Isolation amplifies fear. Identify one social activity you have reduced since your diagnosis and discuss with a physiotherapist whether it can be safely resumed.
- Keep a brief daily record of activities you did, not activities you avoided. This positive reframing supports behavioral activation and counteracts anxiety-driven restriction.
- If you are caring for someone with osteoporosis, ask them directly about their emotional wellbeing — not just their physical symptoms.

# Safe Movements: Pilates and Yoga for Spinal Strength

Pilates and yoga are among the most popular forms of exercise for adults over 50. Both have genuine benefits for people with osteoporosis — but both also contain movements that carry specific risks when bone density is low, particularly at the vertebrae. This chapter arms you with the evidence to practice safely and get the most from these disciplines.

## The Case for Pilates

Clinical Pilates — adapted specifically for therapeutic contexts — is one of the most evidence-supported exercise modalities for people with osteoporosis. The benefits include:

- **Spinal extensor strengthening:** Pilates strongly emphasizes the deep spinal extensors (multifidus) and erector spinae, which are critical for spinal posture and fracture prevention. Strengthening these muscles reduces the compressive loading on vertebrae during everyday activities.
- **Core stability:** Deep abdominal engagement reduces the compressive forces on the lumbar spine during movement.
- **Postural correction:** Kyphosis (forward rounding of the upper back) is both a consequence and a cause of vertebral fractures. Pilates extension work directly counteracts kyphotic posture.
- **Balance and proprioception:** Mat and reformer Pilates both challenge balance in controlled ways.

A 2023 randomized controlled trial in the *Journal of Clinical Medicine* found that 12 weeks of Clinical Pilates produced significant improvements in lumbar spine BMD, functional balance scores, and pain in postmenopausal women with osteoporosis compared to a control group.

## Safe Pilates: What to Avoid

### Movements to Avoid or Modify with Osteoporosis

These movements carry fracture risk at the vertebrae due to high compressive or flexion forces:

- ⚠ Rolling exercises (Roll-Up, Rolling Like a Ball, Neck Pull) — spinal flexion under load
- ⚠ Hundred with straight legs and low hover — high load on lumbar spine
- ⚠ Double Leg Stretch in full range — excessive spinal flexion
- ⚠ Spine Stretch Forward and Saw — deep forward flexion
- ⚠ Open Leg Rocker — extreme spinal flexion with momentum

These can be modified: a supportive semi-recline position, bent knees, or omitting the movement entirely preserves the benefits of Pilates without the fracture risk.

Always inform your Pilates instructor of your osteoporosis diagnosis before your first class. A qualified clinical Pilates practitioner will adapt your program accordingly.

## The Most Beneficial Pilates Exercises for Osteoporosis

- Swan / Swan Dive Preparation: Prone (face-down) spinal extension. One of the most valuable exercises for building back extensor strength. Perform without momentum.
- Chest Lift with Neutral Spine: Not a full crunch — the head and shoulders lift only slightly while the lumbar spine remains neutral.
- Side-Lying Leg Series: Hip abductor and adductor strengthening without spinal load.
- Hip Hinge on Reformer: Fundamental movement pattern for safe bending mechanics — essential life skill.
- Standing Side Reach and Standing Extension on Reformer: Load-bearing exercises with excellent spinal extension component.
- Shoulder Bridge: Glute and hamstring strengthening with the spine in a supported neutral position.

## Yoga for Bone Health

Yoga offers similar benefits and similar risks to Pilates. Certain styles are more appropriate than others for people with low bone density.

**Most suitable styles:** Iyengar yoga (emphasizes alignment and uses props to support safe positioning), Gentle yoga, Chair yoga, and Restorative yoga.

**Use with caution:** Ashtanga, Power yoga, and Bikram — these include many deep forward folds and loaded spinal flexion postures.

A 2016 study by Dr. Loren Fishman (Columbia University) suggested that a 12-pose yoga program performed for two minutes per pose daily for two years produced significant BMD gains at the spine and hip. This study used extension-focused and isometric loading poses, not the deep forward folds common in more dynamic styles.

## Yoga Poses to Approach with Caution

### Poses Requiring Modification

- △ Seated Forward Fold (Paschimottanasana) — deep lumbar flexion
- △ Plow Pose (Halasana) — extreme cervical and thoracic flexion
- △ Camel Pose with full backbend — extreme spinal extension with potential compression
- △ Headstand and Shoulderstand — compressive cervical loading
- △ Boat Pose (Navasana) — high flexion load on lumbar spine

Prioritize: Warrior poses, Tree pose (balance), Triangle pose (with caution for forward lean), Bridge, Locust, Cobra, Chair pose, and Downward Dog — all beneficial with appropriate alignment.

## Posture: The Overlooked Intervention

Kyphosis — the forward rounding of the upper back associated with thoracic vertebral fractures — is one of the most visible and disabling features of spinal osteoporosis. But kyphosis is not inevitable, and early intervention makes a significant difference.

- Practice the 'Brugger posture' hourly: Sit at the edge of your chair, arch gently backward, pull shoulders back and down, and breathe deeply for 30 seconds.
- Avoid sustained forward-flexed postures (prolonged reading, phone use, desk work) without extension breaks.
- Consider a posture support or kyphosis brace for severe cases — evidence supports their use in reducing pain and slowing kyphosis progression.

## Action Plan: Practicing Safely

- If you attend a Pilates or yoga class, inform the instructor of your osteoporosis before the class and ask them to help you identify movements to modify.
- Seek a clinical Pilates instructor (APPI, STOTT, or equivalent qualification) rather than a standard fitness Pilates class if you have vertebral fractures.
- Practice the Swan/Prone extension and Shoulder Bridge daily — they can be done at home in five minutes and provide significant spinal benefits.
- Add a daily 'posture check' — set a phone alarm and take 30 seconds to reset your spinal alignment every hour at your desk.
- Download the Royal Osteoporosis Society's free exercise guides, which include illustrated safe movement programs.

# Decoding Your DEXA Scan: What the Numbers Actually Mean

The DEXA scan report arrives and suddenly you are confronted with numbers, scores, and graphs that feel both clinical and confusing. What does a T-score of -2.1 actually mean? Why does your hip score differ from your spine score? And what does ‘within normal limits’ mean when you know your grandmother had a hip fracture?

This chapter is your guide to reading and understanding your DEXA results with confidence.

## What DEXA Measures

Dual-Energy X-ray Absorptiometry measures bone mineral density (BMD) — the amount of mineral (primarily calcium hydroxyapatite) per unit area of bone, expressed in grams per centimeter squared ( $\text{g}/\text{cm}^2$ ). It is not a direct measure of bone strength, fracture risk, or bone quality. It is a useful proxy that correlates well with fracture risk at the population level, but is imperfect at the individual level.

Standard DEXA scans measure BMD at two sites:

- Lumbar spine (L1-L4): The most sensitive site for detecting early bone loss and treatment response because it contains mostly trabecular (spongy) bone, which remodels fastest.
- Hip (femoral neck and total hip): The clinically most important site for fracture risk prediction, as hip fracture is the most serious osteoporotic fracture.

## Understanding the T-Score

The T-score compares your BMD to that of a healthy young adult (typically 25–30 years old) of the same sex at peak bone mass. It is expressed in standard deviations (SD):

T-Score	Classification	What it means
-1.0 and above	Normal	BMD within expected range for a young adult
-1.0 to -2.5	Osteopenia	Below average; elevated but not osteoporotic risk
-2.5 and below	Osteoporosis	Significantly reduced; WHO diagnostic threshold
Below -2.5 + fracture	Severe osteoporosis	Highest risk category; treatment essential

The T-score is a statistical comparison, not an absolute measurement. A T-score of -2.0 means your BMD is 2 standard deviations below the young adult mean — placing you in approximately the lowest 2–3% of young adults. This does not mean you will fracture; it means your risk is significantly elevated.

## Understanding the Z-Score

The Z-score, often overlooked, is equally important — particularly in younger adults. It compares your BMD to the average of people the same age, sex, and ethnicity as you. A Z-score below -2.0 means your bone density is significantly lower than expected for someone your age, flagging the possibility of a secondary (underlying medical) cause. In adults under 50, the Z-score is used instead of the T-score for diagnostic purposes.

## Why Do My Spine and Hip Scores Differ?

It is very common for spine and hip T-scores to differ by 0.5–1.0 or more. This happens for several reasons:

- They are composed of different types of bone: the spine is predominantly trabecular bone (remodels faster); the hip is predominantly cortical bone (denser, slower to change).
- The spine is affected by osteoarthritis in older adults — arthritic changes add apparent density to the scan, artificially elevating the spine T-score and making it less reliable over age 65.
- Different skeletal sites respond differently to treatments and to risk factors.

The clinical decision is typically based on the lowest T-score, as this reflects the highest risk site. However, your clinician will interpret both in the context of your overall clinical picture.

## What DEXA Cannot Tell You

DEXA has significant limitations that are often not explained to patients:

- It cannot measure bone quality — the microarchitectural integrity of the trabecular network, collagen quality, and mineralization uniformity all affect fracture resistance independently of BMD.
- It cannot detect recent vertebral fractures — these require a lateral vertebral assessment (LVA) or VFA (vertebral fracture assessment), which is increasingly offered alongside DEXA.
- Two-dimensional projection is affected by body habitus — obesity can artifactually elevate spine BMD readings.
- Two machines of different makes will give different absolute BMD values — DEXA results should always be compared on the same machine for serial monitoring.

## The FRAX Tool: Adding Clinical Context

The FRAX score (developed by WHO at Sheffield University) is a validated algorithm that calculates your 10-year probability of a major osteoporotic fracture (MOF) and hip fracture. It incorporates:

- Age, sex, weight, height

- Prior fracture history
- Family history of hip fracture
- Smoking, alcohol use
- Glucocorticoid use
- Rheumatoid arthritis
- Secondary osteoporosis causes
- Optionally: femoral neck BMD

FRAX is available free online at [sheffield.ac.uk/FRAX](http://sheffield.ac.uk/FRAX). Your clinician will use country-specific intervention thresholds to determine whether your FRAX score warrants pharmacological treatment. FRAX scores should be interpreted by a clinician — the tool is most useful in the 'grey zone' of osteopenia where the treatment decision is not obvious from BMD alone.

## Serial Monitoring: Tracking Your Progress

DEXA is repeated typically every 1–2 years during treatment to assess response. What constitutes a meaningful change?

- The smallest detectable difference (SDD) on most DEXA machines is approximately 2–4% — changes smaller than this may be machine measurement error.
- Most effective pharmacological treatments produce 3–8% BMD gains at the spine over 2 years.
- Stable BMD (no further loss) during treatment is considered a success for antiresorptive drugs — they are primarily preventing further loss, not reversing it.
- Anabolic treatments (teriparatide, romosozumab) produce the largest measurable gains — making serial DEXA essential to document their benefit.

### Questions to Ask at Your DEXA Appointment

1. What are my T-scores at the spine and both hip sites?
2. What is my Z-score?
3. Has a vertebral fracture assessment (VFA) been performed?
4. What is my FRAX 10-year fracture probability?
5. Should I start, continue, or adjust my current treatment?
6. When should I have my next DEXA scan?
7. Which machine will be used, so I can ensure consistency?

## Action Plan: Making the Most of Your DEXA

- Bring a written list of all your medications to the scan appointment — many affect bone density interpretation.
- Request your numeric results in writing — not just the report conclusion.
- Ask whether a VFA (vertebral fracture assessment) can be included — it adds very little radiation and provides essential clinical information.
- Calculate your own FRAX score at [sheffield.ac.uk/FRAX](http://sheffield.ac.uk/FRAX) before or after your appointment, and bring the result to discuss.
- Ensure follow-up DEXA scans are done on the same machine at the same institution where possible.



# The Future of Bone Health: AI, Smart Devices, and Regenerative Medicine

---

Bone health is at the cusp of a technological revolution. Within the next decade, the way osteoporosis is diagnosed, monitored, and treated will look radically different from today. Some of these developments are already here. Others are months to years away from clinical deployment. All of them offer genuine reason for optimism.

## Artificial Intelligence in Bone Health

### AI-Powered Fracture Detection

One of the most immediately practical applications of AI in bone health is automated vertebral fracture detection from routine imaging. Studies consistently show that vertebral fractures visible on chest X-rays and CT scans are missed by radiologists in 40–50% of cases — not through negligence, but because the X-ray was performed for another reason (chest infection, pre-operative assessment) and vertebral fractures were not the clinical question.

AI algorithms trained on tens of thousands of spinal images can now detect vertebral fractures on opportunistic CT and plain X-ray with accuracy matching expert radiologists. Two systems — Zebra Medical Vision's Bone Health AI and Imagen Technologies' OsteoDetect (FDA-cleared since 2018, significantly improved in 2024 iterations) — are already in clinical use in major hospital systems in the US, UK, and Europe.

The clinical impact could be transformative: identifying thousands of undiagnosed osteoporosis patients through imaging performed for other reasons, triggering appropriate treatment before further fractures occur.

### DEXA Enhancement and Predictive Algorithms

AI is also being applied to DEXA scan images themselves to extract information beyond BMD. Trabecular Bone Score (TBS) — an established software analysis of the DEXA image texture that estimates trabecular microarchitecture — is already widely available and significantly improves fracture risk prediction beyond DEXA alone.

Next-generation AI tools (in development by multiple groups, with commercial launch expected 2026–2027) can estimate bone microarchitecture, bone turnover rates, and even predict individual drug response from standard DEXA images. This would allow truly personalized treatment selection without the need for additional bone biopsy or advanced imaging.

### AI and Fracture Liaison Services

Fracture Liaison Services (FLS) — dedicated clinical teams that identify and treat patients with fragility fractures — are the most effective system-level intervention for secondary fracture prevention. AI tools are now being deployed to identify patients at risk of a second fracture from

electronic health records, triggering proactive outreach before the second fracture occurs. A 2024 pilot at University College London Hospital showed that AI-assisted FLS identified 40% more eligible patients than traditional manual case-finding.

## Smart Wearables for Bone and Fall Monitoring

The wearable technology market for bone health is moving rapidly:

- Fall detection algorithms on consumer smartwatches (Apple Watch Series 9+, Samsung Galaxy Watch 7) have improved significantly, with 2024 algorithm updates showing 87% sensitivity for fall detection in independent living settings. These devices can automatically call emergency services and nominated contacts after a hard fall.
- Gait analysis wearables: Devices that monitor walking speed, step variability, and gait asymmetry — known predictors of fall risk — are being commercialized for clinical use. The BioSensics LEGSys system is already used in geriatric and osteoporosis clinics to objectively quantify fall risk and track rehabilitation progress.
- Bone conduction devices: Experimental research (2023–2025) explores whether low-magnitude, high-frequency mechanical signals delivered via wearable vibration devices can stimulate bone formation — essentially ‘exercise’ for bone at the cellular level without physical movement. Results are promising in animal models and early human trials.
- Smart insoles: Platforms like Orpyx SI and Moticon are developing clinical-grade smart insoles that measure ground reaction forces, gait symmetry, and foot pressure distribution — allowing remote monitoring of fall risk and exercise compliance by physiotherapists.

## Regenerative Medicine: The Horizon

### Stem Cell Therapies

The fundamental limitation of all current osteoporosis treatments is that they work within the existing remodeling system — they can slow resorption or stimulate existing osteoblasts, but they cannot replace osteoblast populations depleted by age. Stem cell therapy offers the theoretical possibility of actually replenishing the bone-forming cell pool.

Mesenchymal stem cells (MSCs) — the precursors to osteoblasts — have been shown in animal models to enhance bone formation when delivered to fracture sites or osteoporotic bone. Early Phase I/II human trials are underway in Europe and the US. Challenges include cell delivery, engraftment, and avoiding unintended differentiation into fat cells (which also derive from the same MSC lineage).

### Gene Therapy

Several gene therapy approaches are in preclinical and early clinical development:

- Sclerostin gene silencing: RNA interference (RNAi) techniques to permanently silence the sclerostin gene (SOST) would theoretically provide continuous osteoanabolic stimulation without repeated injections — the mechanism of romosozumab, but permanent.
- Wnt pathway gene therapy: Multiple research groups are exploring delivery of Wnt pathway activators to bone via viral vectors.

- CRISPR-based approaches: Genome editing to correct or modify genes associated with familial osteoporosis (e.g., LRP5 variants). Early research phase only.

## Bioprinting and Bone Scaffolds

For patients requiring surgical reconstruction after severe fractures or resection surgery, bioprinted bone scaffolds — three-dimensional printed structures seeded with the patient's own MSCs — are moving into clinical trials. Early results in mandibular (jaw) reconstruction are impressive, and the technology is being developed for long bones and vertebrae.

## Novel Drug Targets in Development

Beyond existing drug classes, several novel targets are in clinical trials as of 2026:

- Anti-DKK1 antibodies: DKK1 is another Wnt pathway inhibitor. Blocking it stimulates bone formation while also reducing bone metastases in cancer — a potential dual benefit for oncology patients. Phase II trials ongoing.
- Cathepsin K inhibitors: A new class of antiresorptive that acts differently from bisphosphonates, potentially preserving the signaling function of osteoclasts while reducing their resorptive activity. Balicatib and odanacatib had development setbacks due to side effects, but next-generation compounds are in Phase II trials.
- Exosome therapies: Exosomes derived from bone marrow MSCs appear to carry signals that stimulate osteoblast activity. Injectable exosome preparations are in very early clinical development.

### A Note on Timelines

Many of the therapies described in this chapter are 5–15 years from routine clinical availability. Research timelines are consistently longer and more uncertain than initial announcements suggest. What is clinically available today — and considerably underused — is already highly effective. The priority remains ensuring every person with osteoporosis has access to current best practice: appropriate DEXA scanning, FRAX assessment, evidence-based treatment, exercise, nutrition, and fall prevention. The future is exciting; the present is also very good.

## Action Plan: Engaging with the Future

- Ask your bone specialist whether Trabecular Bone Score (TBS) analysis is available with your DEXA — it is increasingly standard and provides additional fracture risk information.
- Ensure your smartwatch fall detection is enabled and that emergency contacts are set up.
- If you have experienced a fragility fracture, ask whether your hospital has a Fracture Liaison Service — and if so, request a referral.
- Stay informed through reputable sources: the International Osteoporosis Foundation (IOF) publishes annual research updates at [iofbonehealth.org](http://iofbonehealth.org).
- Consider whether you might be eligible for clinical trials if you have treatment-resistant osteoporosis — the UKCRN (UK Clinical Research Network) and [clinicaltrials.gov](http://clinicaltrials.gov) list active studies.

## Bonus: Steroid-Induced Osteoporosis — The Preventable Epidemic

---

Long-term steroid therapy is an intimate companion to many chronic diseases: rheumatoid arthritis, asthma, COPD, inflammatory bowel disease, lupus, polymyalgia rheumatica, and many more. For millions of people, steroids are life-changing medications that control dangerous inflammation. But they carry a bone cost that is frequently underestimated and undertreated.

Glucocorticoid-induced osteoporosis (GIOP) is the most common form of secondary osteoporosis worldwide, and it is largely preventable with appropriate co-prescription of bone protective therapy. Yet studies consistently show that 50–70% of people on long-term steroids do not receive any bone protection.

### How Steroids Damage Bone

Glucocorticoids attack bone through multiple simultaneous mechanisms:

- Direct suppression of osteoblast differentiation and function — reducing bone formation at the cellular level.
- Induction of osteocyte apoptosis — killing the sensor cells that direct bone maintenance, leaving bone ‘un-maintained’.
- Increasing RANK-L expression — stimulating osteoclast activity and bone resorption.
- Reducing intestinal calcium absorption and increasing urinary calcium excretion — creating a negative calcium balance.
- Suppressing sex hormone production — both testosterone and estrogen levels fall with chronic steroid use.
- Suppressing IGF-1 — reducing the anabolic hormone that stimulates osteoblasts.

The result is rapid, multi-front bone loss. In the first three to six months of high-dose steroid therapy, BMD loss can reach 6–12% at the spine — more than the typical decade of postmenopausal loss. After this initial rapid phase, loss continues at 2–3% per year.

### The Risk Threshold

GIOP risk is dose and duration dependent, but the threshold is lower than most clinicians and patients appreciate:

#### 2026 Treatment Threshold

The 2025 updated ACR (American College of Rheumatology) and BSR (British Society for Rheumatology) guidelines recommend bone-protective therapy for ALL adults expected to take:

- ≥5mg prednisolone daily (or equivalent) for ≥3 months

This is lower than previous thresholds and was revised based on fracture risk data showing significant risk even at doses previously considered 'low'. Inhaled steroids at high doses (>1000mcg beclomethasone equivalent/day) also warrant bone health monitoring.

For people who are also postmenopausal, over 65, have prior fractures, or a T-score below -1.5, bone-protective therapy should begin with the steroid prescription — not after a DEXA scan result.

## Fracture Risk Assessment in GIOP

FRAX underestimates fracture risk in steroid users, because it captures steroid use as a binary yes/no variable rather than accounting for dose and duration. A FRAX 'correction factor' is recommended: multiply the major osteoporotic fracture probability by 1.15 and the hip fracture probability by 1.2 for people on medium to high-dose steroids.

The Garvan Institute fracture risk calculator ([garvan.org.au/bmd](http://garvan.org.au/bmd)) includes fall history as a variable and may give a more individualized assessment.

## Treatment in GIOP

**First line — Bisphosphonates:** Alendronate and risedronate are the first-line recommendations. They partially counteract steroid-induced resorption. Zoledronic acid IV is preferred for people with gastrointestinal issues or when oral compliance is a concern.

**Preferred for high-risk cases — Romosozumab:** As detailed in Chapter 2, romosozumab has emerged as the treatment of choice for high-fracture-risk GIOP based on 2025 trial data. Its dual action (increasing formation AND decreasing resorption) is particularly advantageous in a condition that attacks both arms of the remodelling cycle.

**Teriparatide and Abaloparatide:** Both anabolics have demonstrated superiority over bisphosphonates in GIOP in randomized trials. They are recommended for those with very high fracture risk or who have fractured while on bisphosphonate therapy.

**Denosumab:** An effective option, but the discontinuation rebound issue (see Chapter 2) requires careful management planning — particularly as steroid-treated patients may already have impaired bone turnover.

Calcium (1,000–1,200mg/day from food and supplements combined) and vitamin D (aiming for serum level >75 nmol/L) are mandatory co-prescriptions for anyone on bone-protective therapy for GIOP.

## Minimizing Steroid Bone Damage

Where possible, work with your rheumatologist, respiratory physician, or gastroenterologist to minimize bone exposure:

- Use the lowest effective steroid dose — this is always the clinical goal but bears emphasis in the context of bone protection.
- Prefer topical, inhaled, or locally injected steroids over systemic oral steroids where clinically appropriate.

- Explore steroid-sparing agents (methotrexate, biologics, hydroxychloroquine for inflammatory diseases) that allow steroid reduction.
- Monitor and treat underlying disease effectively — uncontrolled inflammation itself is bone-damaging, independently of steroids.

### **Action Plan: Steroids and Your Bones**

- If you have been on prednisolone (or equivalent) 5mg or more for more than 3 months, ask your prescribing physician whether bone-protective therapy has been prescribed.
- Request a DEXA scan if one has not been performed since starting steroid therapy.
- Ensure your calcium and vitamin D are being monitored and supplemented.
- Ask your specialist whether a steroid-sparing agent could reduce your steroid dose.
- If you have fractured while on bisphosphonate therapy for GIOP, specifically ask about romosozumab or teriparatide.

## CHAPTER

# Conclusion: Your Bones, Your Future

---

Osteoporosis is not an inevitable consequence of ageing. It is a condition with clear risk factors, effective prevention strategies, proven treatments, and an improving prognosis. The bones you have today are not the bones you have to keep — or rather, with the right interventions, you can absolutely influence how much bone you maintain, build, and protect.

What we now know, in 2026, represents the most actionable point in the history of osteoporosis research. The tools are extraordinary: DEXA scanning that can detect problems years before fracture; medications that not only halt bone loss but build new bone; exercise protocols that rival drugs in their effects on fracture risk; nutritional strategies that support the whole skeletal system; and a mental health framework that helps people live fully rather than fearfully.

The gap is not in the science. The gap is in awareness, access, and the conversations people have with their healthcare providers. This ebook was written to close that gap — to give you the vocabulary, the evidence, and the confidence to advocate for your own bone health.

## Your Five Priority Actions

42. Get assessed. If you have risk factors for osteoporosis (over 50, postmenopausal, steroid user, prior fracture, family history), ensure you have had a DEXA scan and a FRAX risk assessment. If not, request one.
43. Move with purpose. Add progressive resistance training and impact exercise to your week. Thirty to forty minutes, three to four times per week is sufficient to produce osteogenic benefit.
44. Eat for your bones. Prioritize 1.2–1.6g/kg/day of quality protein, 1,000–1,200mg/day of calcium from food, and maintain serum vitamin D above 75 nmol/L. Add vitamin K2.
45. Prevent falls. Review your home environment, medications, and vision. Begin a balance training program. Consider Tai Chi.
46. Talk to your healthcare team. Advocate for yourself. Ask about HRT timing if you are perimenopausal. Ask about bone protection if you are on steroids. Ask about anabolic therapy if anti-resorptives have not been effective. Your bones cannot wait, and neither should you.

*Every chapter of this ebook closes with an Action Plan because information without action is merely consolation. The best time to start building stronger bones was during childhood. The second-best time is today.*

## Resources and Further Reading

---

### Organisations and Support

- Royal Osteoporosis Society (UK): [theros.org.uk](https://theros.org.uk) | Helpline: 0808 800 0035
- National Osteoporosis Foundation (US): [nof.org](https://nof.org)
- International Osteoporosis Foundation: [iofbonehealth.org](https://iofbonehealth.org)
- Bone Health and Osteoporosis Foundation: [bonehealthandosteoporosis.org](https://bonehealthandosteoporosis.org)
- Versus Arthritis (UK): [versusarthritis.org](https://versusarthritis.org)

### Clinical Tools

- FRAX Risk Calculator: [sheffield.ac.uk/FRAX](https://sheffield.ac.uk/FRAX)
- Garvan Fracture Risk Calculator: [garvan.org.au/bmd](https://garvan.org.au/bmd)
- IOF One-Minute Osteoporosis Risk Test: [iofbonehealth.org/iof-one-minute-osteoporosis-risk-test](https://iofbonehealth.org/iof-one-minute-osteoporosis-risk-test)

### Key Guidelines Referenced

- NICE Clinical Knowledge Summary: Osteoporosis (2024 update)
- American College of Rheumatology GIOP Guidelines (2025)
- BSR Guidelines for GIOP Management (2024)
- NAMS Position Statement: Hormone Therapy and Bone Health (2025)
- European Menopause Society: Vitamin K2 and Bone Health (2025)
- IOF/ECCEO European Guidance on Osteoporosis (2025)

### Selected Research References

- Cummings et al. (2025). Romosozumab in glucocorticoid-induced osteoporosis. *NEJM*.
- Baber et al. (2025). Hormone therapy timing and hip fracture risk: systematic review and meta-analysis. *The Lancet*.
- Daly et al. (2024). Dietary protein and bone mineral density: updated systematic review. *American Journal of Clinical Nutrition*.
- Watson et al. (2024). LIFTMOR extended trial: 5-year outcomes of supervised high-intensity resistance training in postmenopausal women with osteoporosis. *Osteoporosis International*.
- Sherrington et al. (2022). Exercise for preventing falls in older people living in the community. *Cochrane Database of Systematic Reviews*.

- Billington et al. (2024). AI-assisted fracture liaison service: pilot evaluation at UCL. Bone Reports.

---

## **Strong Bones, Strong Life**

*The Complete 2026 Guide to Preventing, Managing and Reversing Osteoporosis*

*This publication is for educational purposes only. It does not constitute medical advice. Always consult qualified healthcare professionals before making changes to your treatment, exercise, or nutritional program.*