

Serotonin Is Both a Biomarker and Driver of Osteoporosis

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STORY AT-A-GLANCE

- › Osteoporosis is driven by stress-related chemical signals, not just calcium loss, and these signals begin weakening bone years before fractures appear
- › Elevated serotonin in your bloodstream acts as a hormone that directly accelerates bone breakdown while suppressing bone repair
- › Women further past menopause show higher serotonin-related markers and faster progression toward osteoporosis, even when mineral levels look normal
- › Stress hormones triggered by excess serotonin create a biological environment that dismantles bone to meet perceived survival demands
- › Calming stress chemistry through sleep, nutrition, digestion, and gentle strength work shifts bone signaling from breakdown toward preservation

You're taking your calcium. You're doing weight-bearing exercise. Your doctor says your bone scan "looks fine for your age." Then one day you sneeze, turn the wrong way, or trip on a curb – and your wrist shatters. The ER doctor tells you it's osteoporosis, but here's what no one answered: what was actually happening inside your bones for the past decade while your scans still looked acceptable?

For decades, osteoporosis has been treated as an inevitable loss of mineral – a calcium problem that stays hidden until a hip or wrist breaks. Conventional care concentrates on measuring damage after it has already occurred, offering little insight into what drives

the slow decay in the first place. That narrow focus leaves a key question unanswered: why does bone strength begin to fail long before fractures appear?

A growing body of metabolic research points to a signaling breakdown involving a molecule most people associate with mood: serotonin. While serotonin helps regulate your emotional state in your brain, approximately 90% of your body's serotonin is actually produced in your gut and circulates through your bloodstream – what researchers call "peripheral serotonin."

This peripheral serotonin has completely different effects than brain serotonin: instead of influencing mood, it acts as a stress signal that directly communicates with bone tissue. That peripheral serotonin communicates directly with bone tissue, acting less like a feel-good chemical and more like a systemic stress signal. When those signals intensify, bone integrity declines in a measurable and orderly way. This shift moves osteoporosis from inevitable consequence to preventable metabolic dysfunction.

By focusing on serotonin chemistry rather than mineral density alone, osteoporosis begins to look less like an unavoidable consequence of aging and more like a detectable metabolic state. That sets the foundation for understanding how bone deterioration develops, accelerates under stress, and leaves clues long before standard scans reveal structural damage.

Serotonin Markers Signal Bone Loss Long Before Fractures Appear

For a study published in the journal *Biomolecules and Biomedicine*, researchers investigated whether blood markers related to serotonin could identify and predict **osteoporosis** in postmenopausal women, beyond what standard bone scans reveal.¹ The team analyzed clinical data and blood samples from 287 postmenopausal women and grouped them by bone status: normal bone density, osteopenia, and osteoporosis.

Women with osteoporosis showed higher markers of bone breakdown and formation occurring at the same time – a pattern known as high bone turnover. While bone is constantly remodeling throughout life, high turnover means this process is running too fast, like a factory operating in overdrive. The problem: when the cycle speeds up under stress, breakdown consistently outpaces repair, resulting in net bone loss.

- **Serotonin-related markers rose consistently as bones weakened** – Blood levels of serotonin, its immediate precursor 5-HTP, and its breakdown product 5-HIAA were all higher in women with osteoporosis than in those with osteopenia or normal bone density. The worse their bones, the higher their serotonin markers – a progression that suggests direct causation, not coincidence.
- **Higher serotonin markers aligned with faster bone breakdown and poorer bone strength** – The researchers compared these blood markers to established bone turnover markers that show how fast bone is being broken down and rebuilt. Higher serotonin, 5-HTP, and 5-HIAA tracked with higher levels of markers that rise when bone remodeling speeds up.

At the same time, higher serotonin markers matched lower bone density in the spine and hip, the two sites most associated with **disabling fractures**. When researchers adjusted for years since menopause, bone density, and bone turnover markers, serotonin, 5-HTP, and 5-HIAA still emerged as independent risk factors for osteoporosis.

Women who were 12 years or more past **menopause** showed significantly higher levels of serotonin markers than those earlier in menopause. These elevated levels tracked with faster progression toward osteoporosis and shorter time to diagnosis.

- **The relationship worked in both directions, strengthening confidence in the finding** – As serotonin-related markers increased, bone density decreased. As bone density decreased, bone turnover markers increased. These relationships held across every measurement, every analysis, every comparison – the kind of consistency that suggests a fundamental biological mechanism rather than statistical noise.

- **Higher marker levels translated into faster progression toward osteoporosis** – When researchers divided women into high and low marker groups using defined cutoffs, those with higher serotonin-related markers developed osteoporosis sooner. Women with **elevated serotonin** markers showed roughly two- to three-fold higher risk over time compared to those with lower levels.
- **Peripheral serotonin acts on bone cells** – The study described how serotonin produced outside your brain interferes with osteoblasts, the cells responsible for building new bone. At the same time, it supports processes that increase bone resorption, tipping the balance toward net bone loss. In simple terms, higher peripheral serotonin tells bone to break down faster than it rebuilds.

These findings document the what – serotonin markers predict and track osteoporosis progression. But they stop short of fully explaining the why. How exactly does elevated serotonin in your bloodstream translate into weakened bones? This is where bioenergetic researcher Georgi Dinkov's analysis becomes important, as he traces the specific hormonal cascade that connects high serotonin to active bone destruction.²

Serotonin Activates Stress Pathways That Break Bone

In his commentary on the Biomolecules and Biomed study, Dinkov described age-related bone loss as a stress-driven process. He framed the results through the lens of chronic hormonal strain rather than mineral deficiency, emphasizing that the **serotonin** elevations observed in postmenopausal women act upstream of bone breakdown.

His central point was that serotonin is not just associated with osteoporosis in the study – it provides a mechanistic explanation for why **bone loss** accelerates with aging and menopause.

- **Serotonin stimulates the hypothalamic-pituitary-adrenal (HPA) axis, which raises cortisol levels** – Dinkov explained that the serotonin elevations documented in the study activate what's called the HPA axis – a chain reaction that starts in your brain

and ends with your adrenal glands pumping out cortisol, your body's primary stress hormone.

Think of it as your body's emergency broadcast system, designed to mobilize resources during genuine threats. Activation of this axis increases **cortisol**, and cortisol is a catabolic hormone that breaks down tissue. In other words, elevated serotonin doesn't just correlate with bone loss – it causes bone loss by triggering the exact hormonal cascade that dismantles skeletal tissue.

- **Cortisol directly weakens bone structure over time** – Dinkov linked the high-serotonin profiles observed in the study to sustained cortisol exposure, which suppresses bone-forming cells while increasing bone resorption. He noted that this mechanism mirrors what's seen in cortisol-excess conditions such as Cushing's syndrome, offering a clear biological explanation for the faster bone turnover and lower bone density reported in the paper.
- **Blocking serotonin interrupts this destructive loop** – Using evidence from existing drug research, Dinkov explained that blocking serotonin signals shuts down the cortisol response that breaks bone, and in experimental studies this leads to bone loss slowing or reversing. This directly supports the study's conclusion that serotonin is not merely a biomarker of osteoporosis but a driver of the process identified in postmenopausal women.
- **This framework explains why bone loss worsens with time since menopause** – Dinkov emphasized that the longer serotonin remains elevated – as shown in women further from menopause in the study – the longer cortisol acts on bone tissue. This explains why your bone scan can look "borderline" for years before suddenly showing osteoporosis – the damage has been accumulating invisibly all along, driven by stress chemistry that standard scans never measure.

Dinkov pointed out that serotonin measurements reflect active biological stress on bone, whereas bone density scans identify damage only after it has accumulated. In the context of the Biomolecules and Biomedicine findings, this suggests a way to

identify and monitor osteoporosis risk earlier and more safely.

- **Serotonin signals from the brain directly influence bone cells** — Separate research published in *Molecular and Cellular Endocrinology* explains that bone is not an inert structure but is richly supplied with nerve fibers that actively regulate bone-building and bone-breaking cells.³

The researchers describe functional pathways that allow bone tissue to both respond to and regulate serotonin uptake, meaning serotonin acts as a neural signal that directly alters bone metabolism rather than working only through hormones or circulation. This finding shows that changes in brain signaling, mood regulation, and drugs that affect serotonin do not stay confined to your nervous system — they transmit instructions straight to bone cells, shaping bone mass across the lifespan.

Steps That Target the Root Cause of Bone Loss

If you're reading this and recognizing symptoms — anxiety, digestive issues, sleep problems, osteopenia — understand that these aren't separate conditions requiring separate treatments. They're all downstream effects of the same metabolic dysfunction. The conventional approach treats each symptom in isolation: [antacids](#) for digestion, [SSRIs for anxiety](#), and [bisphosphonates](#) for bones.

But if elevated serotonin is the common driver, that approach is like mopping the floor while the faucet is still running. What follows are interventions that address the source — not by adding more serotonin (which most [antidepressants](#) do), but by addressing the stress chemistry that's elevating it in the first place.

1. **Shut down chronic stress signals that keep your bones in breakdown mode** — If you're surviving on adrenaline and coffee, waking up exhausted, or lying awake at 3 a.m. with racing thoughts, your stress hormones aren't just elevated — they're actively cannibalizing your skeleton to fund your body's emergency operations — and this directly prevents bone repair. Your body is prioritizing immediate survival over long-term structural maintenance.

The solution isn't complicated: establish regular sleep and wake times, get morning sunlight exposure on your bare skin (this resets your [circadian rhythm](#)), and eat at consistent, predictable times. These aren't relaxation tips – they're metabolic interventions that directly shut down the stress cascade dismantling your bones. As your stress chemistry normalizes, those bone-destroying signals quiet down, and your body finally gets the message that it's safe to rebuild.

- 2. Boost calming brain chemistry to counter excess serotonin** – Here's what most doctors won't tell you: when you're experiencing anxiety, irritability, impulsiveness, or constant worry, your serotonin levels are likely running high while your calming neurotransmitter gamma-aminobutyric acid (GABA) is running low. This matters tremendously for your bones.

Here's the key relationship your doctor has likely never mentioned: [GABA](#) and serotonin operate like a seesaw in your brain. When GABA is high, you feel calm and emotionally stable – and serotonin naturally stays in its healthy range. But when stress depletes GABA, serotonin floods your system unchecked.

This isn't just about mood: that excess serotonin is simultaneously triggering anxiety in your mind and dismantling your skeleton. The women developing osteoporosis fastest aren't necessarily calcium-deficient – they're GABA-deficient. Both natural and supplemental GABA sources show promise. GABA-rich foods like fermented foods and certain teas provide a natural way to increase intake, while supplements offer more precise dosing options.

- 3. Eat in a way that stops gut-based serotonin production at the source** – When you eat hard-to-digest carbohydrates – like grains, legumes, or fibrous vegetables – with an irritated gut, they pass through your stomach incompletely broken down. Once they reach your intestines, they become food for gram-negative bacteria – the problematic species that thrive in an unhealthy gut. As these bacteria multiply, they release endotoxin (specifically lipopolysaccharide, or LPS) into your intestinal wall.

Your body treats endotoxin as an emergency signal, and one of its primary responses is converting the amino acid tryptophan into serotonin right there in your gut lining. This gut-derived serotonin then enters your bloodstream as peripheral serotonin – the bone-destroying hormone we've been discussing.

The solution? Eat meals that digest fully and comfortably in your stomach. This means choosing **easily digestible carbohydrates** to start, including fruit and white rice. Eat slowly, chew thoroughly, and pay attention to how your body responds. When digestion happens where it should – in your stomach – you dramatically reduce endotoxin production and shut off serotonin synthesis at its primary source.

Further, Lactobacillus and Bifidobacterium probiotic strains produce GABA directly in your gut. This creates another mechanism linking gut health to bone health: a healthy microbiome both reduces endotoxin-driven serotonin production and increases GABA availability.

4. Maintain consistent energy and nutrient intake to prevent metabolic panic – If you avoid carbohydrates, you're sending your body an emergency signal. This immediately raises stress hormones like cortisol, which then elevates serotonin signaling – and your bones pay the price. Your body interprets carbohydrate restriction as famine and begins breaking down bone tissue to liberate stored minerals and amino acids – a survival mechanism that's destroying your structural foundation.

The fix is straightforward but crucial: eat steady, adequate calories every day, and include quality carbohydrates at each meal. Aim for 250 grams of healthy carbs daily. This signals safety to your body and keeps cortisol and serotonin in their proper ranges, allowing your bones to maintain their integrity instead of being cannibalized for survival.

In addition, while stress chemistry drives bone loss, three nutrients work downstream to support bone rebuilding once that stress cascade is addressed: magnesium, vitamin D3, and vitamin K2. **Magnesium** deserves first mention

because it does double duty – it directly dampens the stress response while also being required for vitamin D activation and calcium regulation, and chronic stress depletes it faster than any other mineral.

Without adequate magnesium, your body can't properly use vitamin D or direct calcium into bone. **Vitamin D** – ideally from proper sun exposure – is required for calcium absorption and bone mineralization, but supplementation without adequate magnesium can worsen soft tissue calcification.

Vitamin K2 activates proteins that bind calcium to bone matrix and prevent calcium deposition in arteries and soft tissues. These three nutrients work synergistically – none can compensate for deficiency in the others, and none can overcome chronic cortisol elevation – but once you've addressed the stress chemistry driving bone breakdown, they become essential for directing the rebuilding process properly.

- 5. Use gentle strength work to signal safety to bone tissue** – Your bones respond to consistent mechanical signals, not punishment. Loading them gently but regularly sends the message: "This structure is needed. Maintain and reinforce it." Gentle, consistent resistance training – such as body-weight movements, light weights, **KAATSU**, or resistance bands – is ideal for this purpose.

This type of movement lowers stress chemistry rather than amplifying it, especially when paired with adequate fuel and recovery. If you're rebuilding after bone loss, regular low-intensity strength work sends a powerful message: the environment is stable, load is appropriate, and bone preservation is required.

When serotonin levels drop, stress chemistry settles, and both your gut and nervous system stabilize, your bones receive a clear biochemical signal to stop breaking down and start holding their ground. This isn't about swallowing more calcium while your stress chemistry continues eroding your skeleton from the inside. This isn't about waiting until a fracture forces you to finally address what's been breaking down for years.

This is about addressing the metabolic dysfunction that's driving bone loss in the first place. Your bones aren't separate from your metabolism — they're actively participating in it. When you calm chronic stress, heal your gut, and restore neurotransmitter balance, you're not "treating osteoporosis." You're removing the signals that were commanding your bones to break down in the first place. The rest happens automatically.

FAQs About Serotonin and Osteoporosis

Q: What does serotonin have to do with osteoporosis?

A: Serotonin is widely known for its role in mood, but most of it circulates outside your brain as a hormone. Research shows that higher levels of this peripheral serotonin send stress signals directly to bone tissue, accelerating bone breakdown and reducing bone density over time.

Q: Why isn't calcium the main issue in osteoporosis?

A: Calcium matters for bone structure, but it doesn't explain why bone loss accelerates under stress. Studies show that bone density declines even when calcium and phosphorus levels are normal. The deeper issue is signaling — specifically stress-related chemistry that tells bone cells to break down faster than they rebuild.

Q: How does menopause affect serotonin and bone loss?

A: After menopause, stress signaling becomes more dominant. Research shows that women further from menopause have higher serotonin-related markers and faster progression toward osteoporosis. The longer these signals stay elevated, the

more bone erosion accumulates.

Q: Why are standard bone scans not enough?

A: Bone density scans detect damage only after it has already occurred. They don't reveal the biological signals driving bone loss. Serotonin-related blood markers reflect active stress on bone tissue, offering insight into risk much earlier in the process.

Q: What daily habits help lower serotonin-driven bone loss?

A: Regular sleep, consistent meals, adequate carbohydrate intake, calm digestion, and gentle strength training all reduce stress chemistry. These habits lower excess serotonin signaling, stabilize cortisol, and create an internal environment where bone maintenance and repair resume.

Sources and References

- ¹ [Biomolecules and Biomedicine 2025 Jun 11;25\(11\):2519–2527](#)
- ² [To Extract Knowledge from Matter January 28, 2026](#)
- ³ [Molecular and Cellular Endocrinology November 2005, 242\(1-2\): 1-9](#)