45 — PHASE 03 · RECLAIM · PILLAR · MOVE

Bone Density Begins at 35

We tend to call bone loss a menopause problem. The evidence suggests it often begins earlier, in the years when cycles become less reliably ovulatory and progesterone starts to fall. A great deal of the opportunity to protect your bones for life sits in your late thirties and forties, even though support later still matters.

Dr Kirstey Holland, OMD

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i. —— THE PROBLEM

The problem

So many women come to me having been told their bloods are normal. They are tired, their cycles are changing, their sleep is broken, their breasts are tender, and yet everything supposedly looks fine. Often the tests being run are not the ones that tell us whether ovulation is still happening reliably. You can have a regular-looking period and still ovulate inconsistently, and for your bones, that distinction matters enormously.

Bone loss is silent. Most women do not think about bone density until a scan shows it has already declined, and by then years of quiet change have passed. Your late thirties and forties are a powerful window to influence your bone health for the rest of your life.

ii. —— IN THE BONE

What is actually happening in bone

Bone is living tissue. It is constantly being broken down and rebuilt, and two ovarian hormones are central to that process.

Progesterone has bone-building actions. She works on the osteoblasts, the cells that lay down new bone.[1] Estrogen is protective. She slows the osteoclasts, the cells that break bone down, which reduces the rate of loss. In plain terms, estrogen helps guard the bone you already have, while progesterone helps build new bone.

This matters because progesterone depends on ovulation. Progesterone is made by the corpus luteum, which forms only after an egg is released. From around your mid-thirties, you may begin to

have more cycles in which ovulation is delayed, shortened, or simply does not happen. A cycle without ovulation is a cycle with little or no progesterone.

That is why a regular period does not guarantee full hormonal support for your bones. Studies led by Professor Jerilynn Prior showed that women with subclinical ovulatory disturbances can lose spinal bone even when their estrogen looks perfectly normal.[2,3] In one landmark study, women with ovulatory disturbances lost on average around two per cent of spinal bone a year, and those whose cycles were not ovulating at all lost more than four per cent a year.[2] A five-year study then showed that even women with regular-looking cycles steadily lost cancellous spinal bone when ovulation was quietly disturbed.[3]

WHY PERIMENOPAUSE MATTERS SO MUCH

Perimenopause is not simply a low-estrogen state. For many women it is a time of fluctuating and often still-high estradiol, with progesterone becoming unreliable first as ovulation grows variable. That is why bone changes can begin well before periods stop.[4,5]

Professor Prior has described a meta-analysis of published longitudinal studies suggesting that spinal bone loss averages around 1.8 per cent a year in late perimenopause, against about 1.2 per cent a year in the early postmenopausal years. Those exact figures are best understood as a published synthesis she has discussed publicly rather than a single trial result, but they sit comfortably with broader cohort evidence showing that loss across the menopausal transition is more rapid than we once assumed.[4]

This is one reason the conventional focus on estrogen alone can miss part of the picture. In perimenopause, the problem is often not that estrogen is absent, but that progesterone-driven bone formation is fading. Your cycle and ovulation history across life may help shape your bone health and fracture risk for decades to come.[5]

WHY THE LANGUAGE OF TREATMENT NEEDS CARE

Menopausal hormone therapy has been studied extensively in postmenopausal women, but there is very little safety and outcome data for menopausal hormone therapy started during perimenopause itself, when estrogen is often high and erratic rather than steadily low. Much of what is done at this stage is extrapolated from postmenopausal studies.

That does not mean estrogen never has a role. It means the evidence base for this particular stage is less direct than many people assume. In a progesterone-first approach, the priority in perimenopause is to understand whether you are still ovulating and whether progesterone support is needed, with estrogen considered later if you move into a clearly low-estrogen menopausal state or have specific reasons for it.

WHY PROGESTERONE IS NOT THE SAME AS A PROGESTIN

When a woman is no longer making enough of her own progesterone, the most physiological replacement is body-identical oral micronised progesterone. This is different from the synthetic progestins used in earlier trials, which helped establish the principle that progestogen therapy can support spinal bone.[6,7]

That distinction matters, because much of the direct interventional bone evidence historically used medroxyprogesterone acetate rather than body-identical progesterone. Even so, the biological case for progesterone's bone-building role is strong, and combined estrogen-progestogen therapy has been shown to raise spinal bone density more than estrogen on its own.[8] Progesterone deserves far more attention in conversations about bone during perimenopause than it currently receives.

iii. — FIVE STEPS

Five steps you can begin this week

01

Find out whether you are actually ovulating

Basal body temperature charting, cycle tracking, or cycle-timed progesterone testing tell you far more than a single random blood draw. A regular bleed does not guarantee regular ovulation.

02

Move in a way that loads your bones

Walking is good for your health, but bone strengthens best in response to impact and resistance. Running, jumping, and strength training place useful load through the skeleton. Ovulation and weight-bearing exercise each appear to support spinal bone independently, and work especially well together.[9]

03

Test your vitamin D, then treat based on the result

Vitamin D is essential for calcium absorption and for bone maintenance. Many public-health guidelines treat a level around 50 nmol/L as sufficient, but Professor Michael Holick, one of the most authoritative voices in the field, has long argued that optimal status sits well above the old cutoff and that we should test and treat to a higher target rather than to the minimum.[10] In my clinic I aim for roughly 100 to 150 nmol/L, which I find better supports bone, muscle, and mood. Levels above about 150 to 200 nmol/L can raise the risk of high blood calcium, so if you supplement, test and retest rather than guessing.[11]

04

Support calcium wisely

Keep calcium coming from food where you can, such as dairy if you tolerate it, tinned salmon with the bones, tahini, almonds, and leafy greens. Vitamin K2, particularly as MK-7, is often used clinically to help direct calcium into bone, though its evidence base is not as strong as that for vitamin D, adequate calcium, exercise, and hormone status.

05

Talk to a practitioner about progesterone if your testing suggests deficiency

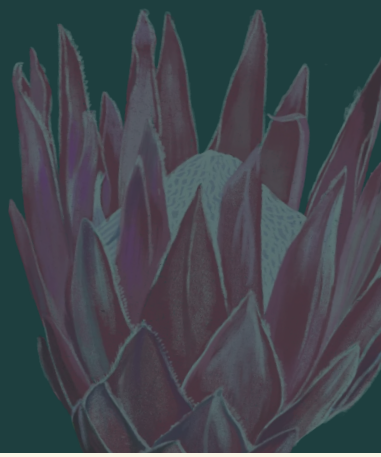
For your skeleton, this may be one of the most important conversations of your decade, particularly if your cycles are changing but you are still being told everything is normal.

A gentler reframe

Several things together decide whether you build bone or lose it: whether you are ovulating and making progesterone, the mechanical load through your skeleton, your estrogen status, your nutrition, and time. The factor most often missed in conventional care is ovulation. Protecting ovulation where we can, recognising when it is being lost, and replacing progesterone thoughtfully when it is appropriate may help restore the bone-building side of the equation that begins to weaken in perimenopause. That is why this stage matters so much. It is so often the point at which bone can still be influenced, before larger losses accumulate.

Bone health is not something to think about only on the day a scan delivers bad news. It is something you build now, especially in your

late thirties and forties, while the return on every action is still high. The question is not only how much estrogen you have. It is also whether you are still ovulating often enough to make the progesterone that lays down new bone. Think of it as strengthening the foundations while the house is still standing straight, rather than waiting for the floorboards to dip.



The evidence behind this guide

Every claim in this article is drawn from published research, most of it the lifetime work of Professor Jerilynn Prior. Here is each finding set beside the study it comes from, so you can see exactly what the evidence shows.

	WHAT IT SHOWS	STUDY
1	Progesterone is a bone-building hormone, acting directly on the osteoblasts to lay down new bone.	Prior JC. Progesterone as a bone-trophic hormone. Endocr Rev. 1990;11(2):386-98.
2	Women with disturbed ovulation lose spinal bone even when their estrogen is normal, around 2 per cent a year, and over 4 per cent a year when not ovulating at all.	Prior JC, Vigna YM, Schechter MT, Burgess AE. Spinal bone loss and ovulatory disturbances. N Engl J Med. 1990;323(18):1221-7.
3	Even women with regular-looking cycles steadily lose cancellous spinal bone over five years when ovulation is quietly disturbed.	Prior JC, Vigna YM, Barr SI, Kennedy S, Schulzer M, Li DK. Ovulatory premenopausal women lose cancellous spinal bone: a five year prospective study. Bone. 1996;18(3):261-7.
4	Bone loss is more rapid in perimenopause than in the years after menopause, with falling progesterone-driven bone formation contributing.	Seifert-Klauss V, Prior JC. Progesterone and bone: actions promoting bone health in women. J Osteoporos. 2010;2010:845180.
5	A woman's cycle and ovulation history across life foreshadows the future health of her bones.	Kalyan S, Prior JC. Bone changes and fracture related to menstrual cycles and ovulation. Crit Rev Eukaryot Gene Expr. 2010;20(3):213-33.
6	Cyclic progesterone-type treatment built spinal bone where placebo lost it, in a randomised controlled trial.	Prior JC, Vigna YM, Barr SI, Rexworthy C, Lentle BC. Cyclic medroxyprogesterone treatment increases bone density. Am J Med. 1994;96(6):521-30.
7	Markers of new bone formation rose during progesterone-type therapy, confirming its bone-building action.	Prior JC, Vigna YM, Wark JD, et al. Premenopausal ovariectomy-related bone loss: a randomised one-year trial of conjugated estrogen or medroxyprogesterone acetate. J Bone Miner Res. 1997;12(11):1851-63.
8	Adding a progestogen to estrogen produces a significantly greater gain in spinal bone density than estrogen on its own.	Prior JC, Seifert-Klauss VR, Giustini D, Adachi JD, Kalyan S, Goshtasebi A. Estrogen-progestin therapy causes a greater increase in spinal bone mineral density than estrogen therapy. J

	WHAT IT SHOWS	STUDY
		Musculoskelet Neuronal Interact. 2017;17(3):146-54.
9	Ovulation and weight-bearing exercise each independently build spinal bone, and work best together.	Petit MA, Prior JC, Barr SI. Running and ovulation positively change cancellous bone in premenopausal women. Med Sci Sports Exerc. 1999;31(6):780-7.

A NOTE ON THE FIGURES

— The figures of around 1.8 per cent spinal bone loss a year in perimenopause and 1.2 per cent in early menopause are as Professor Prior has described them publicly, drawing on her synthesis of longitudinal studies, rather than the result of a single named trial. Vitamin D guidance in this article draws additionally on Holick (2007) and Kennel and colleagues (2010), listed in the references below.

REFERENCES

1. Prior JC. Progesterone as a bone-trophic hormone. Endocr Rev. 1990;11(2):386-98.
2. Prior JC, Vigna YM, Schechter MT, Burgess AE. Spinal bone loss and ovulatory disturbances. N Engl J Med. 1990;323(18):1221-7.
3. Prior JC, Vigna YM, Barr SI, Kennedy S, Schulzer M, Li DK. Ovulatory premenopausal women lose cancellous spinal bone: a five year prospective study. Bone. 1996;18(3):261-7.
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controlled trial in active women with menstrual cycle disturbances. Am J Med. 1994;96(6):521-30.

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11. Kennel KA, Drake MT, Hurley DL. Vitamin D deficiency in adults: when to test and how to treat. Mayo Clin Proc. 2010;85(8):752-7.

> This article is educational and is not a substitute for individual medical advice. Hormone therapy, including bioidentical progesterone, is a prescription decision to be made with your own practitioner, based on your history and testing.

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This resource is for educational purposes and is not a substitute for personalised clinical advice. Every woman's biochemistry is unique. If you would like individualised support, you are warmly welcome inside the Vitality Clinic.

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