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Resistance and Jump (Plyometric) Training in the Peri- and Early Postmenopausal Period for the Prevention of Osteoporosis and Sarcopenia: A Narrative Review

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Abstract

Background. The peri- and early postmenopausal transition combines an oestrogen-driven uncoupling of bone remodelling with accelerated loss of muscle mass and strength, predisposing to osteoporosis, sarcopenia and their overlap, osteosarcopenia. As pharmacotherapy is limited by adverse effects and imperfect adherence, mechanical loading is a central non-pharmacological strategy.

Aim. To synthesise and critically compare evidence on resistance and jump/plyometric (impact) training, alone or combined, for preventing osteoporosis and sarcopenia in peri- and early postmenopausal women, emphasising mechanisms, dose, safety and comparative efficacy.

Materials and methods. Narrative review of systematic reviews, meta-analyses, network meta-analyses and controlled trials, comparing effects on bone mineral density, bone structure, bone-turnover markers, muscle mass, strength, physical performance, falls and quality of life.

Results. Resistance training preserves or modestly increases lumbar-spine and femoral-neck bone mineral density; high-intensity protocols (~70–85% one-repetition maximum), about three weekly sessions over ≥6–12 months give the largest gains. Combining resistance with impact/jump loading yields the strongest spinal osteogenic stimulus, while the hip adapts more in geometry than density. Acute plyometrics elicit an osteoanabolic response before but not after menopause, indicating blunted mechanosensitivity. Resistance and power training reliably improve strength, performance, body composition and falls, though mass gains are inconsistent and benefits regress with detraining.

Conclusions. Supervised, progressive, high-intensity resistance training combined with impact

loading is a safe, evidence-based cornerstone of prevention. Effects depend on adequate intensity, continuity and individualisation; heterogeneity and few fracture trials remain key limitations.

Keywords: perimenopause; osteoporosis; sarcopenia; resistance training; plyometric training; bone mineral density; mechanotransduction.

1. Introduction

Osteoporosis is the most prevalent metabolic bone disease and a major public-health problem that grows in parallel with population ageing and increasingly sedentary lifestyles [1,2]. It is defined by low bone mineral density (BMD) and deterioration of bone microarchitecture, which reduce bone strength and raise the risk of fragility fracture [2,3]. Postmenopausal women bear the greatest share of this burden: ovarian involution and the ensuing fall in circulating oestrogen accelerate osteoclast-mediated bone resorption beyond the capacity of osteoblastic formation, driving rapid bone loss in the years immediately before and after the final menstrual period [4,5]. Epidemiological data from China illustrate the scale of the problem, with the reported incidence of postmenopausal osteoporosis rising from roughly 25% among perimenopausal women to 29.1% in women over 50 and 51.6% in those over 65 years [1]. In developed countries approximately one in three women older than 50 will sustain an osteoporotic fracture during her remaining lifetime [6]. These fractures, most commonly at the vertebrae, proximal femur and distal forearm, are associated with pain, disability, loss of independence and excess mortality: within one year of a hip fracture around 20% of patients die of complications and about half suffer lasting disability, while the medical and social costs impose an enormous burden on families and health systems [1].

Menopause is defined clinically as twelve consecutive months of amenorrhoea reflecting the cessation of ovarian follicular function, occurring at a median age of about 51 years, and it unfolds across the perimenopausal, early-menopausal, menopausal and postmenopausal stages, with oestrogen beginning to fall during perimenopause and reaching its nadir in the postmenopausal years [4]. Because elderly women now spend more than a third of their lives in the low-oestrogen postmenopausal state, the health consequences of oestrogen withdrawal have become a major public-health concern [4]. Osteoporosis is diagnosed principally from areal BMD measured by DXA, the accepted gold standard, with a T-score of −2.5 standard deviations or more below the young-adult mean defining osteoporosis and a T-score between −1.0 and −2.5 defining osteopenia; quantitative ultrasound of the calcaneus and quantitative computed tomography provide alternative or complementary assessment, and fracture-risk algorithms incorporate BMD together with clinical risk factors [2,5]. A central limitation of areal BMD, revisited throughout this review, is that it captures only part of bone strength and does not fully reflect bone size, geometry and microarchitecture, so interventions that improve bone structure without changing areal density may be undervalued by DXA alone [7].

The menopausal decline in oestrogen does not act on bone alone. The same hormonal environment contributes to sarcopenia, the progressive and generalised loss of skeletal-muscle mass, strength and physical performance that also accelerates with age [8]. After menopause muscle mass has been estimated to decline by about 0.6% per year, and low oestrogen is mechanistically linked to impaired muscle protein balance, mitochondrial dysfunction and reduced regenerative capacity [8]. Because the loss of muscle with ageing preferentially affects the type II fibres that generate power, the functional consequences—loss of power, slower reactions and impaired balance—are disproportionate to the loss of mass alone [8,9].

Bone and muscle form an anatomically, metabolically and mechanically coupled unit; muscle contraction is the dominant physiological source of strain on the skeleton, and the two tissues

communicate through shared mesenchymal origins and paracrine signalling [10,11]. Oestrogen deficiency, physical inactivity and ageing therefore tend to erode bone and muscle together, producing osteosarcopenia—an increasingly recognised geriatric syndrome that magnifies the risk of falls, fragility fracture, morbidity, loss of autonomy and death beyond either condition alone [10,12]. Roughly half of women with postmenopausal osteoporosis, and a quarter of those with osteopenia, also meet criteria for sarcopenia, so the perimenopausal window is a strategic point for combined prevention [10].

A range of pharmacological agents, including bisphosphonates, denosumab, selective oestrogen-receptor modulators, parathyroid-hormone analogues and, more recently, romosozumab, effectively reduce fracture risk, and menopausal hormone therapy preserves BMD [2]. However, these therapies do not correct other, modifiable determinants of fracture such as low muscle strength, poor balance and impaired functional capacity, and their use is constrained by adverse effects and imperfect adherence [2,13]. Against this background, mechanical loading through structured exercise has emerged as a safe, low-cost and physiologically rational cornerstone of non-pharmacological prevention that simultaneously targets bone, muscle and fall risk [6,11]. Among exercise modalities, resistance (strength) training and jump/plyometric (high-impact) training deliver the high-magnitude, rapidly applied and unusually distributed strains that best stimulate skeletal adaptation, and resistance training is the most effective mode for building and preserving muscle [7,9,11].

Nevertheless, the literature is heterogeneous and at times contradictory. Meta-analyses differ in the exercise types they pool, the intensities they classify as “high” and the populations they include, so pooled effect sizes are frequently small and their clinical meaning is debated [14–16]. Crucially, most syntheses treat “postmenopausal women” as a single group and rarely isolate the peri- and early-postmenopausal window, in which bone loss is fastest and the therapeutic opportunity greatest [5,15]. The aim of this narrative review is therefore to synthesise and critically compare the current evidence on resistance and jump/plyometric training, alone and combined, for the prevention of osteoporosis and sarcopenia in peri- and early postmenopausal women, focusing on the underlying medical mechanisms, the dose–response relationships, the comparative efficacy against other exercise modes and pharmacotherapy, and the questions of safety, adherence and durability.

The focus on the peri- and early-postmenopausal window is deliberate and clinically motivated. It is during this transition that bone loss accelerates most sharply and that sarcopenia begins to manifest, yet the skeleton and muscle are still comparatively responsive to loading because oestrogen has not fully declined, so intervention here offers the greatest opportunity both to attenuate the accelerated loss and to preserve a higher lifelong reserve of bone and muscle [4,5]. Much of the exercise literature, however, pools women across the entire postmenopausal age range, and some of the most informative mechanistic and interventional evidence derives from older postmenopausal or mixed cohorts; where this is the case, the present review draws on that evidence while flagging its limited transferability and reasoning explicitly about how the greater loading responsiveness of the earlier transition should modify its interpretation [5,17]. The review does not address childhood and adolescent bone accrual, male osteoporosis, or the detailed pharmacology of osteoporosis except insofar as these inform the comparative and complementary role of exercise, and it concentrates on resistance and jump/impact loading because these modalities deliver the strains most relevant to bone while resistance training is uniquely effective for muscle [6,7,11].

2. Materials and methods

This work is a narrative review. Consistent with the narrative rather than systematic format, no

formal protocol registration, quantitative pooling or risk-of-bias meta-scoring was undertaken; instead the available evidence was integrated and interpreted critically, following the comparative and evaluative conventions of the narrative-review genre. The evidence base comprised systematic reviews, meta-analyses and network meta-analyses, randomised and non-randomised controlled trials, one prospective acute-exercise experiment, cohort and cross-sectional studies, study protocols and prior narrative reviews addressing exercise, bone health, muscle health, the menopausal transition and their intersection.

Sources were organised into thematic domains: (i) the endocrine and cellular pathophysiology of bone and muscle loss after oestrogen withdrawal; (ii) mechanotransduction and the biological rationale for mechanical loading; (iii) resistance training and BMD, including the modifying roles of intensity, frequency, volume and duration; (iv) jump/plyometric and combined high-intensity resistance and impact training; (v) resistance and power training for sarcopenia, muscle strength and physical function; (vi) falls, fractures, functional outcomes and quality of life; (vii) comparisons with aerobic training, whole-body vibration, hormone therapy and nutritional adjuncts; and (viii) safety, adherence and detraining. Outcomes of interest included areal BMD at the lumbar spine, femoral neck and total hip measured by dual-energy X-ray absorptiometry (DXA); estimates of bone strength and geometry; circulating bone-turnover markers and osteokines; appendicular and total muscle mass; handgrip and lower-limb strength; gait speed, timed tests and balance; falls and fractures; and health-related quality of life. Findings were compared across studies, with attention to consistency, dose–response and the specific relevance of results to peri- and early postmenopausal women, and disagreements between sources were highlighted rather than averaged away. The principal quantitative studies and syntheses that underpin the review are summarised in Table 1.

Table 1. Summary of the principal studies and syntheses underpinning the review.

Study (design)	Population	Intervention / exposure	Key findings
Li et al. 2025 [1] (network meta-analysis; 49 RCTs, n=3,360)	Postmenopausal women	8 exercise types vs control	AE+RT, RT and WBV most effective for spine/femoral-neck BMD; walking, Tai Chi and isolated impact ineffective; combined training best overall.
Shojaa et al. 2020 [12] (systematic review/meta-analysis; 75 studies, n=5,300)	Postmenopausal women	Any exercise vs control	Significant but modest BMD effects (SMD 0.33–0.40); wide heterogeneity attributed to inadequate exercise protocols diluting the true effect.
Zhao et al. 2025 [13] (meta-analysis; 17 RCTs, n=690)	Postmenopausal women	Resistance training vs no exercise	RT improved LS (SMD 0.88), FN (0.89) and TH (0.30); trochanter NS. ≥70% 1RM, 3×/week, ≥48 weeks optimal; high heterogeneity.
Kitagawa et al. 2022 [14] (meta-analysis; 6 RCTs, n=391)	Postmenopausal women with osteoporosis	High- vs moderate-intensity resistance + impact training	HiRIT superior for LS BMD (SMD 2.37); FN not significant; certainty very low (heterogeneity, small samples, bias).
Kumar et al. 2025 [9] (narrative review; LIFTMOR, MEDEX-OP, ACTLIFE trials)	Postmenopausal women, osteopenia/osteoporosis	Supervised HiRIT (deadlift, squat, press ~80–85% 1RM + jump)	Net LS BMD +2–4% vs low-intensity; FN maintained; hip cortical/trabecular gains not seen on areal DXA; safe, well tolerated; no vertebral fractures.
Eslamipour et al. 2023 [15] (RCT; n=45, 24 weeks)	Postmenopausal women with osteopenia	High- (→85% 1RM) vs low-intensity (→60% 1RM) resistance	Both improved LS/FN BMD, BMC, T- and Z-scores; high-intensity significantly greater; low-intensity still

Study (design)	Population	Intervention / exposure	Key findings
			stabilised bone.
Ashe et al. 2013 [16] (RCT; n=100 imaged, 12 months)	Community-dwelling older women	Resistance training 0, 1 or 2×/week	No significant between-group difference in tibial cortical vBMD; participants already active; 3–4×/week may be needed.
Nelson et al. 2020 [19] (acute experiment; n=20 pre, 20 post)	Pre- vs postmenopausal women	Single plyometric bout (128 jumps)	Osteoanabolic PINP/CTXI shift at 24 h and sclerostin rise only premenopause; no anabolic response postmenopause; higher resting sclerostin/CTX post.
Hamaguchi et al. 2017 [20] (pilot RCT; n=15, 6 weeks)	Postmenopausal women with sarcopenia	Low-repetition, light-load power training (weighted vest)	Pelvis BMD +1.6% and knee-extensor strength +15.5% vs control; controls lost pelvis BMD; adherence >92%, no adverse events.
Chen et al. 2021 [21] (meta-analysis; 14 RCTs, n=561)	Older adults with sarcopenia	Resistance training vs control	Improved grip (SMD 0.81), knee-extension (1.26), gait speed (1.28), TUG (−0.93); muscle mass NS; >60% 1RM, ≥12 weeks favour mass gain.
Seo et al. 2021 [22] (RCT; n=22, 16 weeks)	Older women with sarcopenia	Body-weight + elastic-band resistance training	Improved functional fitness, grip, gait speed, isometric strength; prevented intramuscular-fat rise; follistatin increased; other growth factors unchanged.
Khalafi et al. 2023 [23] (meta-analysis; 101 RCTs, n=5,697)	Postmenopausal women	Aerobic, resistance or combined training	Resistance best for muscle mass/fat-free mass; aerobic best for fat loss; combined optimises both.
Kienberger et al. 2022 [25] (RCT; n=61, 12 months)	Postmenopausal osteopenic women	WBV vs resistance vs control	No significant BMD change in any group; only resistance improved health-related quality of life and postural control; ≥24 months suggested.
Chang et al. 2022 [26] (cross-sectional + longitudinal cohort; n>30,000)	Postmenopausal women (Taiwan Biobank)	Regular vs no regular exercise	Regular exercise: lower osteoporosis prevalence (OR 0.76) and incidence (HR 0.83); dose–response with longer sessions (>1 h).
Treviño et al. 2025 [29] (systematic review; 6 studies)	Postmenopausal women	Hormone therapy vs exercise vs combined	Both improve BMD; HRT generally larger gains; combination possibly additive at LS/FN but often not significant; HRT discontinuation causes bone loss.
Nikander et al. 2010 [11] (meta-analysis; 10 RCTs)	Children to older adults	Weight-bearing/impact/resistance vs controls	Significant bone-strength gain in pre-/early-pubertal boys but not adults or postmenopausal women; effects site-specific, compliance-dependent.
Kelley et al. 2013 [34] (meta-analysis; 7 RCTs, n=521)	Premenopausal women	Ground/joint-reaction-force exercise	Small significant gains in femoral-neck and lumbar-spine BMD; NNT 5–9; establishes benefit before oestrogen loss.
Gombarčíková et al. 2025 [27] (systematic review of training + detraining)	Postmenopausal osteopenia/osteoporosis	Exercise then cessation	Training improved BMD, strength and function; gains regress with detraining, sometimes to baseline within ~12 months; continuity essential.

AE, aerobic exercise; BMC, bone mineral content; BMD, bone mineral density; FN, femoral neck; HiRIT, high-intensity resistance and impact training; HRT, hormone replacement therapy; LS, lumbar spine; NNT, number needed to treat; NS, not significant; RCT, randomised controlled trial; RT, resistance training; SMD, standardised mean difference; TH, total hip; TUG, timed up-and-go; vBMD, volumetric BMD; WBV, whole-body vibration; 1RM, one-repetition maximum.

3. Pathophysiology of bone and muscle loss in the menopausal transition

3.1. Oestrogen, bone remodelling and the RANKL/OPG–Wnt axis

Bone is a dynamic tissue continuously renewed by the coordinated action of bone-resorbing osteoclasts, bone-forming osteoblasts and mechanosensing osteocytes [2,5]. In the healthy adult skeleton resorption and formation are tightly coupled, but after menopause the withdrawal of oestrogen shifts the balance decisively toward resorption, producing a net loss of bone that is most rapid during the first five to seven years of the postmenopausal period, when annual losses of 2–3% have been reported before slowing to around 0.5–1% thereafter [5,18]. Oestrogen exerts protective effects on all three bone-cell types through both genomic and non-genomic signalling, its receptors being highly expressed in osteoblasts, osteoclasts and osteocytes; ligand-bound receptors suppress osteoclast formation and activity while supporting osteoblast survival and function [2,5].

At the molecular level the central pathway is the RANK/RANKL/osteoprotegerin (OPG) axis. Oestrogen promotes osteoblastic and stromal production of OPG, a decoy receptor that binds the receptor activator of nuclear factor-κB ligand (RANKL) and prevents it from engaging its receptor RANK on osteoclast precursors, thereby restraining osteoclastogenesis and resorption [2,5]. When oestrogen falls, RANKL expression rises and OPG declines, tilting the RANKL/OPG ratio toward osteoclast recruitment, activation and prolonged survival, with the trabecular compartment—where remodelling is most active—losing bone most rapidly [5]. In parallel, oestrogen deficiency amplifies the secretion of pro-inflammatory and osteoclastogenic cytokines, including interleukin-1, interleukin-6 and tumour necrosis factor, and increases reactive oxygen species, further driving bone breakdown [2,5]. Oestrogen also normally restrains osteocyte production of sclerostin, an inhibitor of the Wnt/β-catenin pathway; its loss therefore permits greater sclerostin-mediated suppression of osteoblastic bone formation [2,5]. The Wnt/β-catenin cascade is itself a key anabolic route, and oestrogen supports osteoblast differentiation from mesenchymal progenitors toward the osteoblastic rather than the adipocytic lineage while stimulating insulin-like growth factor-1 and transforming growth factor-β [2,5]. The net consequence of oestrogen deficiency—increased resorption, blunted formation, cytokine-driven osteoclastogenesis and impaired Wnt signalling—is the microarchitectural deterioration that defines postmenopausal osteoporosis [2,5,18].

This uncoupled state is measurable in the circulation and helps to explain both the disease and the response to exercise. Bone-turnover markers—the resorption marker C-terminal telopeptide of type I collagen and the formation marker procollagen type I amino-terminal propeptide—reflect the rate and balance of remodelling, and their ratio correlates with hip and spine BMD, so a fall in the formation-to-resorption ratio signals net bone loss [5,17]. Postmenopausal women characteristically display higher resting resorption markers and higher sclerostin than premenopausal women, a biochemical signature of the resorption-dominant, formation-suppressed remodelling that follows oestrogen withdrawal [17]. These markers are relevant to exercise for two reasons. First, they provide a mechanistic read-out of how loading acts: an osteogenic bout should transiently favour formation over resorption, and moderate-intensity aerobic and weight-bearing exercise has been shown to raise formation markers such as procollagen type I amino-terminal propeptide while limiting resorption [12]. Second, as

detailed later, the acute bone-turnover response to impact loading differs between pre- and postmenopausal women, offering direct evidence that oestrogen status conditions the skeleton's biochemical responsiveness to mechanical stimuli [17]. The turnover markers thus link the endocrine pathophysiology of this section to the loading physiology of the next, and they are increasingly used to monitor both disease progression and treatment response [5,17].

3.2. Oestrogen deficiency and skeletal muscle: mechanisms of sarcopenia

The perimenopausal fall in oestrogen also directly and indirectly compromises skeletal muscle [8]. Oestrogen receptors are present in skeletal muscle, particularly in type II fibres, and oestrogen influences muscle mass and function through effects on protein turnover, mitochondrial biology and satellite-cell activity [8]. Oestrogen supports muscle protein balance in part by maintaining Akt/mTOR signalling, which drives protein synthesis, and by restraining the forkhead box protein O transcription factors that promote atrophy through the ubiquitin ligases atrogin-1 and MuRF1; oestrogen withdrawal reduces muscle Akt phosphorylation and de-represses these catabolic programmes [8]. Oestrogen further regulates muscle mitochondria, and its loss is associated with mitochondrial dysfunction, altered mitochondrial dynamics, diminished mitophagy and increased mitochondria-mediated apoptosis, mechanisms that plausibly accelerate menopause-related sarcopenia [8]. Because ageing preferentially reduces the type II fibres that are most responsive to loading and most important for rapid, force-demanding tasks, the functional impact is disproportionate to the loss of mass [8,9].

Sarcopenia is nonetheless multifactorial. Beyond hormonal change it reflects motor-neuron loss and denervation–reinnervation, dysregulated cytokine secretion, chronic low-grade inflammation, mitochondrial dysfunction, satellite-cell depletion and inadequate nutrition and physical activity [8,9]. The European Working Group on Sarcopenia in Older People has shifted the diagnostic emphasis from muscle mass toward low muscle strength as the principal determinant, with confirmation requiring low muscle quantity or quality and severity graded by poor physical performance [8]. This conceptual move matters for prevention, because interventions such as resistance training reliably improve strength and function even when gains in mass are modest [19–21].

The structural changes underlying sarcopenia further clarify why loading, and specifically high-force and high-velocity loading, is the appropriate stimulus. With advancing adulthood the number of muscle fibres falls by an estimated 30–40% between the second and eighth decades, and the type II fibres shrink by 10–40% while type I fibres are comparatively spared, so the muscle that remains is not only smaller but disproportionately depleted of the fast, powerful fibres on which rapid, force-demanding tasks depend [12]. Motor-unit remodelling compounds this: denervation followed by reinnervation produces fewer, larger and slower motor units, and central drive to muscle declines, so power—the product of force and velocity—falls faster than strength, and strength faster than mass [9]. Because power and balance deteriorate earliest and are the most direct determinants of fall avoidance, exercise for sarcopenia in postmenopausal women should preferentially target the type II fibres and neuromuscular power through progressive resistance and, where tolerated, high-velocity or power training, rather than relying on low-intensity endurance activity that preferentially recruits fatigue-resistant type I fibres and does little to restore power [9,19]. This mechanistic reasoning aligns with the trial evidence, reviewed below, that resistance and power training improve strength, gait speed and balance markedly even when muscle mass changes little [20,21].

3.3. Osteosarcopenia: the shared bone–muscle unit

Bone and muscle are linked by shared mesenchymal origins, by paracrine and endocrine cross-talk and by the mechanical coupling through which muscle contraction is the dominant source of physiological strain on the skeleton [10,11]. Muscle-derived myokines—including irisin, interleukin-6 and myostatin—modulate osteoblast and osteoclast activity, and contracting muscle may also communicate with bone through small extracellular vesicles, although these signals require validation in postmenopausal cohorts [6,11]. The clinical corollary is that oestrogen deficiency, physical inactivity and ageing tend to erode bone and muscle together, producing osteosarcopenia [10,12]. Individuals with osteosarcopenia show a greater fracture burden and higher mortality than those with either sarcopenia or osteopenia/osteoporosis alone; a hip-fracture study reported a markedly elevated mortality risk, and community estimates place its prevalence at roughly a quarter to more than a third of older adults, depending on the criteria applied [10,13]. This convergence provides the strongest argument for interventions such as resistance and impact training that simultaneously load bone and challenge muscle, and it frames the therapeutic logic of the sections that follow [10,12,13].

3.4. Metabolic, adipose and inflammatory dimensions of postmenopausal bone loss

Oestrogen is a systemic metabolic regulator, and its withdrawal at menopause reshapes body composition and energy metabolism in ways that indirectly affect the skeleton [4]. The menopausal transition is characterised by weight gain, a fall in energy expenditure and a redistribution of fat toward the abdominal and visceral depots, accompanied by dyslipidaemia; even when energy intake is controlled, loss of lean mass and reduced basal energy expenditure promote adiposity [4]. These changes matter for bone because expanding visceral adipose tissue is a source of pro-inflammatory cytokines—including interleukin-6 and tumour necrosis factor-α—that stimulate osteoclastogenesis, while bone-marrow adipose tissue expands as bone formation wanes and is inversely associated with BMD across ageing, postmenopausal osteoporosis and obesity [4]. Because osteoblasts and marrow adipocytes derive from a common mesenchymal progenitor, oestrogen deficiency appears to bias lineage commitment toward the adipocytic at the expense of the osteoblastic fate, and lipid substrates that might otherwise fuel bone formation may accumulate in marrow adipocytes instead [4]. Emerging work also implicates the oestrogen-sensitive gut microbiome, whose dysbiosis after oestrogen loss increases intestinal permeability, expands osteoclastogenic T-cell populations and reduces calcium absorption, and follicle-stimulating hormone, which rises at menopause and may independently promote bone loss and visceral adiposity [2,4]. Although these metabolic and inflammatory pathways are still being characterised and are not the primary targets of exercise, they help explain why the postmenopausal skeleton loses bone so rapidly and why interventions that reduce visceral and intramuscular fat—such as resistance and combined training—may confer skeletal benefit beyond direct mechanical loading [4,22].

4. Mechanotransduction: the biological rationale for mechanical loading

The rationale for exercise as anti-osteoporotic therapy rests on the skeleton's capacity to adapt its mass and architecture to habitual mechanical demand, a process governed by mechanotransduction—the conversion of physical strain into biochemical signalling [7,11,23]. Osteocytes, the most abundant bone cells, are embedded within the mineralised lacunar–canalicular network and act as the principal mechanosensors [6,11]. When loading deforms the bone matrix, interstitial fluid is driven through the canaliculi, generating shear stress across osteocyte membranes that opens mechanosensitive ion channels, elevates intracellular calcium

and triggers paracrine messengers such as prostaglandin E2 and nitric oxide [6,11]. A key downstream event is the down-regulation of osteocytic sclerostin (the product of the SOST gene): with sclerostin reduced, Wnt ligands can bind the LRP5/6 receptor complex, β-catenin is stabilised and translocates to the nucleus, and osteogenic gene expression is promoted, tipping remodelling toward formation [6,11]. Loading also favourably shifts the RANKL/OPG balance, restraining resorption [6].

Frost's mechanostat theory refines Wolff's law by defining strain thresholds: below a "disuse" window bone is lost, within an "adapted" window it is maintained, and only when strain exceeds a modelling threshold—the "overload" window—is net formation stimulated, while excessive strain risks microdamage [11,18]. Effective osteogenic exercise must therefore exceed the strains of daily activity [7,11]. Animal and human data converge on the loading characteristics that best stimulate bone: strains of high magnitude, applied rapidly (high strain rate) and in atypical, multidirectional distributions, delivered as dynamic rather than static loading, in relatively few cycles interspersed with rest, because osteocytes rapidly desensitise to repetitive monotonous strain and require recovery intervals to restore mechanosensitivity [6,7,11]. These principles explain why high-impact and multidirectional activities such as jumping, and heavy progressive resistance exercise that transmits large muscular tension to the periosteum, are more osteogenic than low-impact, non-weight-bearing or purely aerobic activity, and why short bouts with rest outperform the same load applied continuously [7,11,23]. The response is also site-specific, being greatest where strain is highest, and its magnitude is modulated by continued participation and by the individual's ability to sustain sufficient intensity [7,9].

Two caveats temper this rationale in the target population. First, mechanotransduction depends on oestrogen: osteocytes deprived of oestrogen show impaired fluid-induced calcium signalling and attenuated Wnt/β-catenin activation in response to loading, so the postmenopausal skeleton may be intrinsically less responsive to a given mechanical stimulus [5,17]. Second, ageing reduces the integrity of the osteocyte lacunar–canalicular network, potentially blunting mechanosensitivity further [6,17]. Both observations imply that peri- and early postmenopausal women—closer to the oestrogen-replete state—may retain greater loading responsiveness than older women, strengthening the case for early intervention, while also predicting that higher loading intensities may be required to overcome a raised adaptation threshold after oestrogen loss [6,17].

Loading also acts on bone through the muscle it drives. During resistance exercise, contracting skeletal muscle behaves as an endocrine organ, releasing myokines such as irisin, interleukin-6 and myostatin that reach bone and modulate the differentiation and activity of osteoblasts and osteoclasts, and muscle-derived small extracellular vesicles may carry additional mediators of this cross-talk; irisin, for example, has been reported to enhance osteoblast activity while suppressing RANKL-driven osteoclastogenesis [6]. In parallel, resistance exercise elevates systemic insulin-like growth factor-1, which activates PI3K–Akt–mTOR signalling to stimulate muscle protein synthesis, and the resulting increase in muscle mass and force transmits greater mechanical load to the skeleton [11]. This muscle–bone axis provides a second, biochemical route—complementary to direct osteocyte mechanotransduction—by which resistance and power training may benefit bone, and it is a plausible mechanism for the coupled improvements in muscle and bone observed with combined loading; however, most human data derive from young men, and the myokine and vesicle responses to resistance exercise remain largely uncharacterised in postmenopausal women, an important gap for future research [6].

These mechanistic principles translate into a set of loading characteristics that define an osteogenic exercise programme. Extensive animal and human work indicates that the skeleton responds to the magnitude, rate, distribution and number of loading cycles: the most effective

programmes combine high strain magnitude with a high strain rate (rapid application), unusual and multidirectional strain distributions, and relatively few loading cycles, and they are most successful when strain magnitudes reach roughly three to nine times body weight and are delivered as short, varied bouts several times per week rather than as prolonged repetitive activity [7,23]. Because osteocytes rapidly saturate their response to a monotonous stimulus, distributing loading across short sessions with rest intervals restores mechanosensitivity and extracts more osteogenic benefit from the same total load, a principle deliberately exploited in the low-repetition, rest-interspersed power protocols discussed later [11,19]. Translating these characteristics into practice, the frequency–intensity–type–time framework applied to bone favours a frequency of two to three osteogenic sessions weekly, an intensity high enough to exceed habitual strain (approximately 80–85% one-repetition maximum for resistance, and ground-reaction forces well above those of walking for impact), a type combining heavy resistance with weight-bearing impact, and a time of roughly 30–60 minutes per session, with progression as the essential principle because the skeleton continually re-adapts to accustomed loads [15,23]. These parameters, derived from mechanotransduction, anticipate and explain the trial evidence in the sections that follow, and they justify the emphasis on high-intensity resistance combined with impact over low-intensity or aerobic alternatives [7,11,23].

5. Resistance training and bone mineral density

5.1. Overall efficacy and the meta-analytic picture

Resistance training is the exercise mode most consistently associated with preservation or gain of BMD in postmenopausal women, because contracting muscles apply large, site-specific mechanical loads to bone that stimulate the osteogenic response described above [11,15]. A large systematic review and meta-analysis pooling 75 studies and 5,300 participants found significant but modest overall effects of exercise on BMD, with standardised mean differences of 0.37 at the lumbar spine, 0.33 at the femoral neck and 0.40 at the total hip [14]. Importantly, that synthesis emphasised wide variation among individual studies, ranging from highly effective trials to trials with negative effects, and attributed this heterogeneity chiefly to differences in the adequacy of the exercise protocols; the authors concluded that the true effect of exercise is diluted by the many studies that applied insufficient loading [14]. This interpretation is pivotal: small pooled effects reflect not that exercise is weakly osteogenic but that much of the pooled literature under-dosed the stimulus.

A dedicated resistance-training meta-analysis of 17 randomised controlled trials in 690 postmenopausal women reported that resistance training significantly improved BMD at the lumbar spine (standardised mean difference 0.88), femoral neck (0.89) and total hip (0.30), although the effect at the greater trochanter did not reach significance and heterogeneity for the spine and femoral neck was high [15]. A network meta-analysis of eight exercise types in 3,360 postmenopausal women ranked combined aerobic-plus-resistance training and resistance training among the most effective modalities for lumbar-spine BMD, and combined training, whole-body vibration and resistance training among the most effective for femoral-neck BMD, whereas Tai Chi, walking, impact exercise in isolation and mixed low-intensity programmes showed no significant effect [1]. Narrative syntheses concur that resistance training preserves BMD principally at the fracture-relevant lumbar spine and proximal femur and that the spine tends to be more responsive than the hip [3,11,18]. Together, the meta-analytic evidence supports resistance training as effective for bone, while making clear that the size of the benefit is conditional on how the training is prescribed.

The magnitude of the achievable benefit must also be read against the biology of the ageing

skeleton. Bone is most responsive to loading during growth, when weight-bearing exercise can raise bone mass and strength by 1–8% at loaded sites, and considerably less responsive after skeletal maturity, when the primary role of exercise shifts from building bone to maintaining or attenuating its loss [7]. A meta-analysis of bone-strength outcomes found significant loading effects in pre- and early-pubertal children but not in adults or postmenopausal women, in part because adult trials were short, under-powered and heterogeneous, and because adult bone adapts mainly through reduced endocortical resorption and modest periosteal apposition rather than large gains in size [7]. In premenopausal women, by contrast, a meta-analysis of randomised trials found small but significant gains in femoral-neck and lumbar-spine BMD from ground- and joint-reaction-force exercise, with a number needed to treat of five to nine, establishing that loading is osteogenic while the oestrogen milieu is still intact [24]. Read together, these observations reinforce the rationale for intervening early in the menopausal transition: the peri- and early-postmenopausal skeleton is closer to the responsive premenopausal state than the older postmenopausal skeleton, so loading initiated in this window may both slow the accelerated peri-menopausal loss and preserve a higher lifelong bone reserve, whereas the same stimulus applied years later must work against a less responsive, more resorptive skeleton [7,24].

5.2. Site specificity: lumbar spine versus proximal femur

A recurring pattern across the evidence is that resistance and combined loading affect the lumbar spine more reliably than the proximal femur in areal-density terms [3,6,15]. In the resistance-training meta-analysis, effects were robust at the spine, femoral neck and total hip but absent at the trochanter, a site the authors attributed to insufficient strain generation by conventional resistance protocols at the lateral proximal femur, compounded by the low-oestrogen milieu that shifts remodelling toward resorption and by the inherently slow pace of bone turnover [15]. A resistance-training BMD review similarly reported positive effects at the hip and spine but not consistently at the femoral neck for some protocols, again implicating inadequate site-specific loading [3]. This apparent hip resistance may, however, partly reflect the limits of DXA. Analyses of high-intensity trials using three-dimensional modelling of hip scans have shown that the proximal femur can adapt to loading by improving femoral-neck cortical thickness and total-hip trabecular volumetric density—structural changes that enhance strength and fracture resistance—without a corresponding rise in areal BMD [6]. Because cortical thickness is a major determinant of femoral-neck strength, the hip may therefore benefit more than areal DXA suggests, and conclusions restricted to areal density risk underestimating the skeletal value of loading at the hip [6,11]. The practical implication is that exercises must directly load the fracture-prone sites—spine, hip and wrist—and that structural as well as densitometric endpoints are needed to capture the full response [7,11].

The structural basis of the loading response deserves emphasis because it reframes how the hip result should be interpreted. Bone adapts to increased load through surface-specific changes: periosteal apposition, which enlarges the bone's cross-section and, because bending strength rises with the fourth power of radius, disproportionately increases strength; and changes at the endocortical surface, where reduced resorption or apposition thickens the cortex [7]. In older adults the dominant adaptation appears to be reduced endocortical bone loss and increased tissue density rather than large periosteal expansion, so exercise may improve bone strength while producing only small changes in areal density [7]. Because areal BMD by DXA accounts for perhaps 60–70% of the variance in bone strength and captures neither geometry nor microarchitecture, it can substantially understate the mechanical benefit of loading, particularly at the hip; quantitative and peripheral quantitative computed tomography, three-dimensional hip structural analysis and high-resolution imaging reveal cortical-thickness and trabecular

changes invisible to DXA [7,11]. The clinical consequence is twofold: first, negative or null DXA results in exercise trials should be read cautiously, as they may mask real gains in bone strength; and second, future trials should incorporate structural endpoints to quantify the true skeletal response, especially at the proximal femur where areal density is least responsive but fracture consequences are gravest [7,11].

5.3. Intensity: high versus low–moderate load

Intensity is the exercise parameter most strongly linked to bone response. According to osteogenic-loading principles, higher strains applied rapidly provide the most robust stimulus, and clinical evidence increasingly bears this out [6]. A meta-analysis of exercise intensity found that high-intensity protocols were more effective than moderate- or low-intensity protocols for increasing lumbar-spine BMD, with a mean difference of about 0.031 g/cm² for high intensity versus 0.012 and 0.010 g/cm² for moderate and low intensity, respectively, although no clear effect emerged at the femoral neck [6,16]. A subsequent meta-analysis comparing high-intensity resistance and impact training with moderate-intensity resistance and impact training in 391 postmenopausal women with osteoporosis found that high-intensity training significantly improved lumbar-spine BMD (standardised mean difference 2.37), whereas the femoral-neck difference did not reach significance; the certainty of evidence was rated very low because of high heterogeneity, small samples and risk of bias, so the authors urged cautious interpretation despite the direction of effect favouring high intensity [16].

Resistance-parameter analyses reinforce this dose–response. A meta-analysis of resistance-training parameters concluded that high-intensity training (≥70% one-repetition maximum) had significant effects on the total hip and femoral neck, whereas lower intensities often failed to reach the stress threshold required to stimulate bone; the authors identified a high-intensity regimen performed three times weekly over a longer duration as likely optimal [15]. A randomised controlled trial in 45 postmenopausal women with osteopenia directly compared high-intensity, low-repetition resistance training (progressing to 85% one-repetition maximum with 8 repetitions) against low-intensity, high-repetition training (progressing to 60% one-repetition maximum with 16 repetitions) over 24 weeks; both improved bone mineral content, BMD and T- and Z-scores at the lumbar spine and femoral neck relative to controls, but the high-intensity group achieved significantly greater gains [25]. The authors interpreted this through Wolff's law—greater load produces greater adaptation—while noting that low-intensity resistance training still stabilised bone and could be preferable for women with osteoarthritis, joint pain or very fragile bone who cannot tolerate heavy loading [25]. This nuance is clinically important: intensity should be maximised within the constraints of each woman's tolerance rather than pursued indiscriminately.

Not all evidence is unanimous. One resistance-training network meta-analysis cited within the literature reported that moderate intensity was superior to high intensity for BMD, a discrepancy attributed to differing definitions of intensity—that study having set ≥80% one-repetition maximum as "high" and 65–80% as "moderate," against thresholds of ≥70% and 51–69% elsewhere [15]. Such definitional variance is a major source of the apparent inconsistency between syntheses and a strong argument for standardised intensity reporting [6,15,16]. Overall, the weight of evidence favours moderate-to-high loading, with the caveat that the femoral neck responds less predictably in areal terms than the spine and that the highest-quality trials in osteopenic and osteoporotic women support high-intensity resistance combined with impact rather than resistance in isolation [6,16].

The reason intensity matters so much is cellular. Bone adaptation requires strains that exceed the modelling threshold of the mechanostat, and only loads large enough to generate such

strains down-regulate osteocytic sclerostin sufficiently to release the Wnt/β-catenin brake on osteoblastic bone formation [11,18]. Loads below this threshold leave sclerostin elevated and remodelling in its adapted or disuse window, which is why low-intensity resistance and low-impact activity tend to maintain rather than build bone [11,23]. High-magnitude resistance also strengthens bone indirectly by increasing muscle mass and the mechanical tension transmitted to the periosteum, and by stimulating the ion-channel and osteogenic responses of osteoblasts and osteocytes to large loads [11]. In the low-oestrogen postmenopausal skeleton, where resting sclerostin is already high and mechanosensitivity blunted, the strain threshold for a net anabolic response is plausibly raised, so a higher absolute intensity may be needed to achieve the same osteogenic effect that a lighter load produces in a younger woman [17,18]. This cellular logic underpins the empirical dose–response evidence: the trials achieving the clearest BMD gains are those that reached genuinely high loads, whereas the many under-dosed studies in the pooled literature diluted the apparent effect of exercise [14,16].

5.4. Frequency, volume, duration and progression

Beyond intensity, the temporal parameters of training shape the response. A frequency of at least two, and preferably three, sessions per week is widely identified as effective; the resistance-parameter meta-analysis found that training three times weekly significantly improved BMD at the lumbar spine, femoral neck, total hip and trochanter, and some authors argue that frequency may matter as much as intensity [6,15]. A session duration of around 40 minutes was associated with significant lumbar-spine effects in the same analysis [15]. Whether frequency can be lowered once benefit is achieved is unresolved. A randomised controlled trial testing resistance-training frequency (0, 1 or 2 sessions per week) on tibial cortical volumetric density in older women found no significant between-group difference over 12 months, although the participants were already highly active and near their bone “ceiling,” and the authors could not exclude that three or four weekly sessions might be required to produce a measurable difference [26]. That negative trial is a useful reminder that pre-existing activity, baseline bone status and the sensitivity of the measurement site all modulate the apparent frequency–response relationship.

Duration and progression are equally decisive. Because the bone-remodelling cycle takes roughly three to four months and a new steady state of bone mass typically requires seven to nine months, interventions of at least six months, and ideally 12–24 months, are needed to demonstrate structural change; the parameter meta-analysis found that cycles of 48 weeks or more had significant effects at the femoral neck and total hip [7,15]. Progressive overload is essential, since the skeleton adapts to habitual load and a stimulus that is not incremented ceases to exceed the modelling threshold; reviews of resistance training for bone consistently prescribe progressively increased load, two to three sets per exercise, 5–9 or 8–12 repetitions, at 50–85% one-repetition maximum, two to three times per week for major muscle groups crossing the hip and spine [3,11,15]. Effects also appear cumulatively: one intervention reported spine BMD increases of about 2.8% at 5 months, 2.3% at 12 months and 1.9% at 18 months relative to control, and combined weight-bearing programmes have shown their most significant benefits only after the first two years [3]. Finally, benefit is contingent on continuation, a theme returned to in the discussion of detraining [3,27].

Training volume—the product of sets, repetitions and load—interacts with intensity to determine the osteogenic and myogenic dose. General resistance-training guidance for older adults recommends two to three sets of one to two exercises per major muscle group, at 5–8 repetitions when the emphasis is on high load and 8–12 repetitions for a balance of strength and hypertrophy, performed two to three times weekly, with the load progressively increased as

strength improves [9,11]. For bone specifically, the mechanostat implies that a modest number of high-magnitude loading cycles is more osteogenic than many low-magnitude cycles, so very high repetition counts at light load are inefficient for the skeleton even if they build muscular endurance [11,23]. This convergence of volume, intensity and progression explains why the well-designed high-intensity trials, which used relatively few repetitions at heavy loads with systematic progression, achieved clearer bone gains than the many pooled studies that applied larger volumes of lighter work [14,16]. In practice, the prescription for the peri- and early-postmenopausal woman should therefore favour heavy, progressive, multi-joint loading in a small number of quality sets over high-volume light training, while remaining within the individual's technical competence and tolerance [11,15].

6. Jump (plyometric) and impact training, and combined high-intensity resistance and impact training

6.1. Impact loading and the osteogenic index

Jump and plyometric exercises are high-impact activities that generate ground-reaction forces several times body weight, applied rapidly and in varied directions—precisely the strain characteristics that mechanotransduction principles identify as maximally osteogenic [7,23]. Multidirectional "odd-impact" loading, as occurs in jumping and change-of-direction activities, appears especially effective because it presents unaccustomed strain distributions that resist osteocyte desensitisation [23]. Meta-analytic evidence indicates that moderate- to high-impact exercise can improve bone structure across the lifespan and that the osteogenic index of multicomponent programmes combining impact with resistance is roughly twice that of single-mode programmes [1]. Mixed-loading programmes that combine jogging or jumping with resistance and other impact activities have been shown to reduce postmenopausal bone loss at the hip and spine, whereas isolated impact exercise of insufficient intensity, and low-impact activities such as walking, exert little effect on BMD in this population [1,7]. Bone-strength meta-analyses similarly conclude that the most successful programmes incorporate a diverse range of weight-bearing activities—skipping, dancing, jumping, hopping—of three to nine times body weight, performed three to five times weekly [7]. The efficacy of impact loading is therefore not in doubt in principle; the central question in the target population is whether the postmenopausal skeleton can still translate that stimulus into an anabolic response [17].

The way impact is programmed is as important as its inclusion. Rather than prescribing vigorous, unmodified plyometrics to women with low bone mass, contemporary high-intensity trials introduce impact through a graded progression—beginning with low-level tasks such as heel drops and progressing over weeks to a controlled jumping or drop-landing manoeuvre—so that the skeleton and its supporting soft tissues adapt before peak forces are reached, and so that technique can be established under supervision [6]. This staged approach reconciles the osteogenic requirement for high, rapidly applied strains with the safety imperative in a fracture-prone population, and it explains why the impact components embedded within supervised programmes have proved both effective and safe, whereas the acute-response evidence counsels against indiscriminate high-impact prescription in older postmenopausal women [6,17]. The osteogenic potency of a sporting or exercise activity can thus be ranked by the magnitude, rate and variety of the ground-reaction forces it imposes: high-impact, multidirectional activities sit at the top, heavy resistance and power work next, and low-impact, non-weight-bearing or continuous aerobic activities at the bottom, a hierarchy consistent with the finding that the osteogenic index of combined impact-plus-resistance programmes is about twice that of single-

mode training [1,23]. For the peri- and early-postmenopausal woman, the practical inference is that a modest, progressive impact stimulus—such as low-level jumping integrated into a resistance session—can meaningfully augment the skeletal response without the risks associated with high-volume plyometrics [6,23].

6.2. The bone-turnover response to plyometric exercise before and after menopause

A pivotal experiment directly addressed this question by comparing the acute bone-biomarker and osteokine response to a single bout of plyometric exercise in 20 premenopausal and 20 postmenopausal women [17]. The exercise consisted of 128 jumps organised into five circuit stations, with box-jump height individualised to each participant's maximum jump to standardise ground-reaction force while protecting older participants [17]. At rest, postmenopausal women already displayed a catabolic bone profile, with significantly higher sclerostin and higher C-terminal telopeptide of type I collagen (a resorption marker) and lower dickkopf-1 and oestradiol than premenopausal women, consistent with an uncoupled, resorption-dominant remodelling state [17]. Critically, sclerostin rose transiently five minutes after exercise only in the premenopausal group, and the ratio of procollagen type I amino-terminal propeptide to C-terminal telopeptide—a marker of the formation-to-resorption balance that correlates with hip and spine BMD—fell acutely and then rose significantly above baseline at 24 hours only in premenopausal women, indicating a net osteoanabolic response; no such 24-hour anabolic shift occurred in the postmenopausal women [17].

These findings carry two important implications for the present topic. First, they provide direct human evidence that oestrogen deficiency blunts the skeleton's anabolic response to impact loading, plausibly through reduced osteocyte mechanosensitivity, higher resting sclerostin approaching a ceiling effect, and age-related changes in the osteocyte lacunar–canalicular network [17]. Second, the authors concluded that plyometric exercise is an effective osteoanabolic stimulus in younger women and should be encouraged in that group, but that its effectiveness in postmenopausal women is less clear, and that—given the perceived fracture risk of high-impact exercise in women with low bone mass—routine prescription of unmodified plyometrics for postmenopausal populations is not yet justified on that acute-response evidence alone [17]. This tempered conclusion is significant for the peri- and early-postmenopausal window: women in this transitional phase retain more oestrogen than older postmenopausal women and may therefore preserve greater responsiveness to impact loading, but the same evidence counsels caution about applying vigorous plyometrics indiscriminately once oestrogen has fallen. It also motivates the modified, progressively graded impact approaches embedded within supervised high-intensity programmes discussed below [6,16].

The pattern of the biomarker responses repays closer mechanistic reading. In premenopausal women the transient post-exercise rise in sclerostin, followed within 24 hours by a rise in the formation-to-resorption ratio, is consistent with an initial signalling response of loaded osteocytes and a subsequent net anabolic shift as formation outpaces resorption [17]. The absence of this 24-hour anabolic rise in postmenopausal women, despite an identical loading stimulus scaled to individual jump height, points to a genuine attenuation of the downstream osteogenic response rather than to inadequate loading [17]. Several non-exclusive mechanisms may account for it: the higher resting sclerostin of postmenopausal women may approach a ceiling that limits further load-induced signalling; the osteocyte lacunar–canalicular network becomes smaller, more spherical and less dense with ageing, plausibly reducing mechanosensitivity; and the loss of oestrogen removes a permissive signal for osteocyte responsiveness to strain [17]. Whatever the precise mechanism, the finding cautions that extrapolating the osteogenic efficacy of impact loading from young to older women is unsafe,

and it reinforces the rationale both for intervening early in the transition, while responsiveness is greater, and for using higher loading intensities to overcome a raised anabolic threshold once oestrogen has fallen [6,17].

6.3. Power and light-load jump training in sarcopenic postmenopausal women

Power (high-velocity) training, which shares the rapid force development of plyometrics, has been reported to be more effective than conventional strength training for preserving bone in postmenopausal women, because bone adaptation is influenced by the velocity as well as the magnitude of loading [19]. A pilot randomised study examined low-repetition, light-load power training in Japanese postmenopausal women with sarcopenia [19]. The six-week programme comprised five body-weight power exercises (squats, front and side lunges, calf raises and toe raises) performed as eight sets of three repetitions with a 15-second inter-set rest and progressive loading using a weighted vest, the short sets and frequent rests being deliberately chosen to restore the mechanosensitivity of bone cells and maximise power output [19]. Despite the brief intervention, pelvis BMD increased significantly by 1.6% and knee-extensor strength by 15.5% in the training group relative to controls, while control participants lost pelvis BMD [19]. The authors argued that light-load power training can generate peak power comparable to or exceeding that of high-load training because the lighter load permits faster movement, and that maximising peak power—the most important factor for bone adaptation—together with adequate rest intervals produced the osteogenic effect without heavy or fatiguing loads [19]. Because the programme was easily implementable and well tolerated, with adherence above 92% and no adverse events, the authors proposed it as suitable for sedentary older women who are fearful of heavy loading [19]. This study is notable for demonstrating a bone benefit from a jump-like, velocity-oriented stimulus in a frail, sarcopenic postmenopausal group, though its small sample and short duration limit firm conclusions.

Power training also occupies a strategic position in fall prevention, linking the skeletal and neuromuscular goals of this review. Because muscle power declines earlier and faster than strength or mass with ageing, and because the rapid force production needed to arrest a fall depends on type II-fibre power, high-velocity training targets the capacity most directly relevant to avoiding the falls that cause fractures [9,19]. Multicomponent programmes that incorporate power or high-velocity resistance work alongside balance and functional training have improved physical status and reduced adverse outcomes in frail older adults, and power training has been reported to improve functional capacity and muscle performance more than slow-velocity training in older women [9]. For the peri- and early-postmenopausal woman, therefore, a jump-like or power-oriented component serves a dual purpose: it delivers the rapid, high-rate strains that are osteogenic, and it restores the neuromuscular power that reduces fall risk, so that a single class of exercise contributes simultaneously to building bone and to preventing the falls through which weak bone becomes fracture [9,19]. Where full plyometrics are inappropriate, light-load power training offers a lower-risk means of pursuing the same velocity-dependent adaptations [19].

6.4. Combined high-intensity resistance and impact training

The synthesis of resistance and impact loading in supervised high-intensity resistance and impact training represents the most promising bone-targeted exercise strategy to emerge for this population [6,16]. Contemporary trials combined heavy free-weight resistance exercises performed in weight-bearing positions (deadlift, back squat, overhead press) at 80–85% one-repetition maximum with a high-impact jumping component—for example landing after dropping from a chin-up bar—twice or thrice weekly for 8–13 months [6,16]. Across these

trials high-intensity training produced net lumbar-spine BMD gains of approximately 2–4% relative to low-intensity exercise, together with maintenance of femoral-neck BMD where low-intensity controls lost bone; the maximal weight lifted during the deadlift correlated positively with the spine BMD response, supporting a dose–response relationship between skeletal load and adaptation [6]. As already noted, the hip adapted through improved cortical thickness and trabecular volumetric density rather than areal density, so its structural benefit is understated by DXA [6]. A meta-analysis directly comparing high- with moderate-intensity resistance and impact training likewise favoured high intensity for the lumbar spine, though with very low certainty owing to heterogeneity [16].

The historical concern that heavy loading through the thoracolumbar spine might precipitate fracture in high-risk women had long discouraged such protocols, but the recent trials indicate that, with appropriate safeguards, high-intensity resistance and impact training is safe and well tolerated in postmenopausal women with osteopenia or osteoporosis [6]. In one trial in which roughly a third of participants had a prior vertebral fracture, an eight-month programme did not increase thoracic kyphosis and produced no new or worsening vertebral fractures, actually improving kyphosis, and adherence exceeded 80–90% with higher reported enjoyment than low-intensity home exercise [6]. The essential safety principles are supervision by an exercise physiologist or physiotherapist, individualisation, an initial familiarisation period to teach correct technique, and progressively graded intensity; suitable participants must be able to mobilise independently and follow instructions [6]. Two uncertainties remain especially relevant to prescription: the durability of the load-induced osteogenic response over sustained participation, and safety in women with a recent (within 12 months) vertebral fracture, who were excluded from these trials and for whom evidence is lacking [6]. For the peri- and early-postmenopausal woman without recent fracture, however, supervised progressive high-intensity resistance and impact training currently represents the best-supported exercise strategy for building bone [6,16].

Comparison across the multicomponent and high-intensity trials adds useful nuance about site and dose. A 12-month multimodal programme in predominantly older osteopenic or fall-prone women, combining high-velocity progressive resistance, moderate-impact weight-bearing exercise and highly challenging balance work, produced modest net gains of about 1.0–1.1% at the lumbar spine and femoral neck with no change at the total hip, and a research-to-practice extension suggested that femoral-neck gains persisted even as adherence fell, whereas the spine effect was lost [6]. The dedicated high-intensity resistance-and-impact programmes achieved larger spine gains (about 2–4%) than these multimodal or low-intensity comparators, and a positive association between the maximal deadlift load and the spine BMD response supports a genuine dose–response relationship between skeletal load and adaptation [6]. Across trials the total hip proved the most resistant site in areal terms, consistent with the resistance-parameter meta-analysis and with the difficulty of generating adequate strain at the lateral proximal femur, yet three-dimensional analyses revealed compensatory gains in femoral-neck cortical thickness and total-hip trabecular density that areal DXA could not detect [6,15]. The consistent messages are that the lumbar spine is the most reliably responsive site, that the hip benefits structurally even when its areal density is unchanged, that intensity drives the magnitude of response, and that a maintenance frequency of exercise may retain gains at some sites—observations that directly inform how the peri- and early-postmenopausal woman should be advised [6,15].

7. Resistance and power training for sarcopenia, muscle strength and physical function

Resistance training is the most effective exercise mode for countering the muscular component of osteosarcopenia [13,20]. Mechanistically, resistance exercise stimulates muscle protein synthesis through the Akt–mTOR pathway, increases the cross-sectional area of type I and,

particularly, type II fibres, and—especially in the early weeks—drives neural adaptations such as improved motor-unit recruitment and rate coding that raise strength before any change in mass [9,11]. In postmenopausal women, resistance exercise elevates systemic growth factors including insulin-like growth factor-1 and, through mechanical loading, up-regulates Wnt1 signalling and suppresses sclerostin, linking the muscular and skeletal benefits within a single stimulus [6,15].

The relative contribution of neural and hypertrophic mechanisms explains the characteristic time-course of strength gains and has practical implications. In the first weeks of resistance training, improvements in strength are driven predominantly by neural adaptations—more efficient motor-unit recruitment, synchronisation and rate coding—rather than by muscle hypertrophy, so strength and function can improve well before muscle mass changes, which is why functional benefit is achievable within 8–12 weeks even in frail sarcopenic women [11,20]. Hypertrophy, when it occurs, requires greater intensity, adequate duration and sufficient protein intake, and older muscle exhibits a degree of anabolic resistance—a blunted muscle-protein-synthetic response to a given bout of resistance exercise compared with younger muscle—that helps explain why muscle-mass gains are inconsistent in older-adult trials [11]. This anabolic resistance can be partly overcome by higher training intensity, longer intervention duration and leucine-rich protein supplementation, reinforcing the case for progressive, adequately dosed resistance training combined with attention to nutrition in this population [11,20]. The practical corollary is that clinicians and women should expect and value early functional gains, persist long enough for structural adaptation, and not judge the intervention a failure if scale-measured muscle mass changes little, since strength, power and physical performance are the outcomes most closely tied to falls, independence and quality of life [9,20].

A meta-analysis of 14 randomised controlled trials in 561 older adults with sarcopenia found that resistance training significantly improved body-fat mass, handgrip strength (standardised mean difference 0.81), knee-extension strength (1.26), gait speed (1.28) and the timed up-and-go test (−0.93), while effects on appendicular skeletal-muscle mass, total skeletal-muscle mass and leg-lean mass were not significant [20]. This dissociation—robust functional gains with inconsistent mass gains—recurs throughout the literature and reflects the predominantly neural basis of early strength improvement and the difficulty of increasing mass without sufficient duration and nutritional support [20,21]. Subgroup analyses indicated that greater muscle-mass gains occurred with intensities above 60% one-repetition maximum, interventions of at least 12 weeks and, for some outcomes, lower weekly frequency, and the authors recommended moderate-to-high intensity, two to three sets of 8–12 repetitions, about three days weekly, with the mode (for example elastic bands or machines) matched to the individual [20].

A 16-week randomised controlled trial of body-weight and elastic-band resistance training in older women with sarcopenia demonstrated significant improvements in functional fitness, grip strength, gait speed and isometric muscle strength, and—using computed tomography—prevented the age-related increase in intramuscular fat seen in non-exercising controls, thereby improving muscle quality, although effects on the muscle growth factors GDF-8, GDF-15 and activin A were limited and only follistatin rose significantly [21]. This trial illustrates that even accessible, low-cost resistance modalities can enhance muscle quality and function in sarcopenic postmenopausal women, while confirming that molecular and mass responses lag behind functional gains over a four-month horizon [21]. Consistent with the power-training data discussed earlier, high-velocity and power-oriented work appears particularly valuable for restoring the type II-fibre power that determines gait, balance and fall avoidance, and light-load power training improved knee-extensor strength by 15.5% alongside its pelvic bone benefit in sarcopenic postmenopausal women [19,21]. A broad narrative review of body-composition outcomes concluded that resistance training is the most effective mode for muscle gain and fat-

free-mass preservation in postmenopausal women, aerobic training the most effective for fat loss, and combined training a viable strategy to optimise both—findings that support integrating resistance work into any comprehensive programme for this group [22].

Because bone and muscle deteriorate together, interventions that address only one tissue are inherently incomplete, and there is growing interest in exercise strategies designed explicitly for osteosarcopenia [10,13]. A systematic-review protocol on exercise for osteosarcopenia noted that, although exercise is recommended by extrapolation from the separate literatures on osteoporosis and sarcopenia, direct evidence of its efficacy in people with the combined syndrome remains sparse, and it identified the need to determine the optimal duration, frequency, intensity and type of exercise—across impact, resistance, balance, combined and vibration modalities—for this high-risk group [13]. Contemporary osteosarcopenia programmes therefore converge on multicomponent, progressive resistance-based training, frequently combined with a high-impact or power element and with nutritional support, on the logic that heavy, weight-bearing loading is the single stimulus capable of acting on bone and muscle simultaneously [10,13]. Foundational work in this area, such as the Franconian Osteopenia and Sarcopenia Trial in older men, demonstrated that high-intensity resistance training with protein and vitamin D supplementation improved both the sarcopenia Z-score and lumbar-spine and total-hip BMD and was safe over prolonged follow-up, providing proof of principle that a single exercise-plus-nutrition strategy can reverse both components of osteosarcopenia [10].

Translating this to peri- and early postmenopausal women, a randomised-controlled-trial protocol combining 24 weeks of progressive, high-intensity circuit resistance training—advancing through defined phases toward repetition-maximum loading on machines targeting the lower limbs, upper limbs and core—with daily protein, vitamin D and calcium supplementation was designed specifically to improve the skeletal-muscle index and lumbar-spine and total-hip BMD of postmenopausal women with osteosarcopenia, alongside functional, quality-of-life, psychological and bone-turnover outcomes [10]. Such designs embody the practical synthesis of the evidence reviewed here: progressive resistance loading heavy enough to stimulate bone and muscle, delivered under supervision with graded intensity, combined with impact or power work where tolerated and underpinned by adequate protein, calcium and vitamin D [10,13]. For the peri- and early-postmenopausal woman, in whom osteoporosis and sarcopenia are beginning concurrently, this integrated approach is both mechanistically rational and pragmatically efficient, addressing two linked conditions with one intervention [10,13].

8. Falls prevention, fracture risk, functional outcomes and quality of life

Because most fragility fractures, and more than 90% of hip fractures, follow a fall, an exercise strategy that reduces fall risk can lower fracture incidence even independently of its effect on BMD [6]. This dual pathway—building bone through loading and preventing falls through strength, balance and functional training—is the strongest clinical rationale for multicomponent programmes [6]. Meta-analytic evidence in community-dwelling older adults shows that exercise reduces the rate of falls by roughly 20–30%, with balance and functional exercises, or multicomponent programmes combining balance, functional and resistance exercises, more effective than any single component, and a Cochrane review of 116 trials reported a 23% reduction in fall rate with exercise compared with control [6]. Resistance training contributes to this effect by improving lower-limb strength, postural stability and neuromuscular control, and by preserving the muscle power that underlies rapid corrective responses [6,9].

Direct evidence that exercise reduces fracture is weaker, chiefly because no trial has been powered with fracture as its primary endpoint; nonetheless, meta-analyses indicate that exercise

interventions can reduce falls-related and major osteoporotic fractures by about 20–30% relative to controls, and BMD improvement is an accepted surrogate for fracture-risk reduction [6]. Large-scale epidemiological data support a protective association: in a community-based study of more than 30,000 postmenopausal women, regular exercise was associated with a significantly lower prevalence of osteoporosis (odds ratio 0.76) and, in a longitudinal subcohort, a reduced risk of incident osteoporosis (hazard ratio 0.83), with a dose–response whereby longer exercise sessions (more than one hour) conferred greater protection [28]. That study's reliance on self-reported activity and calcaneal ultrasound tempers its precision, but its scale and consistency reinforce the population-level value of regular exercise for bone [28].

The stakes of fracture prevention are considerable and justify a preventive investment even where the direct trial evidence is incomplete. Osteoporotic fractures are among the leading causes of disability and death in older adults; global projections anticipate a steep rise in hip-fracture numbers over coming decades, with associated costs projected to grow enormously, and within a year of a hip fracture roughly a fifth of patients die of complications while about half suffer lasting disability and loss of independence [1]. Because more than nine in ten hip fractures follow a fall, and because BMD and structural improvements together with better balance and strength reduce both the likelihood and the consequences of falling, an exercise strategy that acts on all these determinants targets the fracture pathway at several points simultaneously [1,6]. This multi-target action is the conceptual justification for prioritising combined resistance, impact and balance training in prevention: even absent a definitive fracture-endpoint trial, the convergence of a ~20–30% reduction in falls, meaningful BMD and structural gains, and improved neuromuscular function provides a strong, mechanistically coherent case that such training reduces fracture risk in the peri- and early-postmenopausal woman [6]. Framing exercise as fracture prevention rather than merely BMD improvement also aligns the intervention with the outcome that matters most to patients and health systems [1,6].

Beyond bone and falls, resistance and combined training improve the functional and psychosocial outcomes that matter to women in the menopausal transition. Trials and reviews report gains in lower-limb strength, walking speed, chair-stand and timed up-and-go performance, dynamic and static balance, and reductions in the fear of falling, all of which support independence [20,21]. A European workplace-based combined-training programme in menopausal osteopenic women produced significant improvements in lower-limb strength and static and dynamic balance, outcomes the authors linked to reduced fall risk and better bone loading, alongside improvements in the general perception of health [29]. Whole-body-vibration and resistance interventions have improved health-related quality of life and postural control in osteopenic postmenopausal women even when BMD was unchanged [30], and resistance training has been shown to reduce depressive symptoms in older adults, an important consideration given the elevated risk of mood disturbance during the menopausal transition [10]. Even where a modality exerts little effect on bone—such as Pilates—it may still improve muscle strength, flexibility, balance, sleep and mood in postmenopausal women, underscoring that exercise benefits in this population extend well beyond densitometry [31]. Physical activity is likewise associated with fewer psychosocial and physical menopausal symptoms, with a moderate level of activity linked to the greatest symptomatic benefit in perimenopausal women [32].

The breadth of these functional benefits is illustrated by objective testing in intervention programmes. In the European combined-training project, structured training produced significant gains across a functional battery—lower-limb one-repetition-maximum strength on leg-press, leg-curl, leg-extension and gluteal machines, six-minute walking distance, a thirty-second sit-to-stand test, static single-leg-stance time and multidirectional dynamic-balance reach—confirming that combined resistance, balance and functional work improves precisely

the capacities that govern fall risk and independence in menopausal women [29]. Because impaired balance and gait variability are among the strongest predictors of falling, and because most fractures follow a fall, such functional gains constitute a plausible fracture-prevention pathway that complements any change in BMD and that pharmacotherapy alone does not provide [6,9]. These outcomes are also the ones women most readily perceive, which may aid motivation and adherence to a programme whose skeletal benefits are otherwise invisible day to day [29,32].

The relationship between physical activity and menopausal well-being is not simply linear. A cross-sectional study of perimenopausal women found that the total menopause-specific quality-of-life score and its psychosocial and physical subscores followed a U-shaped pattern in relation to activity level, with women performing a moderate amount of activity reporting significantly fewer psychosocial and physical symptoms than either low- or high-activity women, whereas vasomotor and sexual symptoms were not associated with activity [32]. This pattern suggests that a moderate, sustainable dose of activity confers the greatest symptomatic benefit and cautions against assuming that more is always better for quality of life, even though the skeletal evidence favours high-intensity loading; the two goals are reconciled in practice by prescribing intense but time-limited bone-targeted sessions within an overall moderate weekly activity pattern [6,32]. These symptomatic and psychological benefits are clinically relevant because the burden of menopausal symptoms—mood disturbance, sleep problems, fatigue and reduced self-esteem—can itself undermine the motivation and capacity to exercise, creating a cycle that well-designed, enjoyable programmes can help to break [31,32].

Modalities that are gentle on the skeleton can still contribute to this broader well-being. Pilates, though it provides only limited osteogenic loading, has been reported to strengthen deep trunk and lower-limb muscles, improve flexibility, balance and postural control, and reduce pain in postmenopausal women, and it has additionally been associated with better sleep quality and reductions in anxiety and depressive symptoms, offering a comprehensive if bone-modest option for women who cannot or will not undertake heavy loading [31]. From an implementation standpoint, the setting of delivery shapes both adherence and psychosocial benefit: a European combined-training programme for menopausal osteopenic women achieved lower dropout when delivered in the workplace than in an external sports centre, and completers across settings improved lower-limb strength, balance and their general perception of health, illustrating that reducing practical barriers can widen access to the functional and psychological gains of exercise [29]. Taken together, the evidence indicates that resistance and impact training deliver their skeletal and muscular benefits within a wider matrix of functional, symptomatic and psychological improvements that are themselves important to healthy ageing through the menopausal transition [29,31,32].

9. Comparative efficacy: resistance and impact training versus other modalities and pharmacotherapy

9.1. Resistance and impact training versus aerobic training and walking

Not all exercise loads bone equally. Aerobic and endurance activities such as walking, cycling and swimming benefit cardiovascular and metabolic health but provide inadequate osteogenic strain: reviews and meta-analyses consistently find that non-weight-bearing aerobic exercise and habitual walking have little or no effect on BMD, and that swimming and cycling may even be associated with lower bone mass because of the absence of impact [1,7,11]. The network meta-analysis of eight modalities placed walking, Tai Chi and isolated low-intensity impact

exercise among the ineffective options for BMD, while ranking resistance training, combined aerobic-plus-resistance training and whole-body vibration among the effective ones [1]. Where aerobic exercise does benefit bone, it appears to act indirectly—through modulation of hormone levels, bone-turnover markers and trace-element status—rather than through direct high-magnitude loading, and moderate-intensity aerobic exercise can attenuate resorption and enhance formation markers such as procollagen type I amino-terminal propeptide [1,11]. The clinical corollary is that walking should be regarded as a valuable adjunct for cardiometabolic health and general activity but not as a stand-alone osteoporosis-prevention strategy; resistance and impact loading are required to stimulate bone [7,11]. Combined aerobic-plus-resistance programmes, however, are effective and pragmatic, improving BMD at the lumbar spine, femoral neck, total hip and whole body while also enhancing fitness and body composition, and several cohorts have shown BMD benefit specifically when resistance training is combined with aerobic training rather than either alone [1,22].

The value of combining modalities extends beyond bone. A large meta-analysis of body-composition outcomes in postmenopausal women found that resistance training and combined training increased muscle mass and fat-free mass, whereas aerobic and combined training were most effective for reducing fat mass, body-fat percentage, waist circumference and visceral fat [22]. Because visceral and intramuscular fat are themselves detrimental to bone and muscle—through inflammatory cytokines and impaired muscle quality—the fat-reducing effect of aerobic and combined training may indirectly support the musculoskeletal system, while the muscle-building effect of resistance training loads bone and preserves function [4,22]. A pragmatic programme for the peri- and early-postmenopausal woman therefore anchors on progressive resistance and impact loading for bone and muscle, and adds aerobic activity for cardiometabolic health and adiposity, recognising that the combination optimises the widest range of outcomes even though the resistance and impact components carry the primary responsibility for the skeleton [1,22]. Walking and other low-impact aerobic activity remain worthwhile for overall health and daily activity, but should be positioned as complements to, not substitutes for, the bone-targeted loading that they cannot themselves provide [7,11].

9.2. Resistance training versus whole-body vibration

Whole-body vibration has attracted interest as a low-demand alternative that transmits mechanical oscillation to the skeleton without heavy loading, and the network meta-analysis ranked it among the effective modalities for femoral-neck BMD [1]. However, direct trial evidence is mixed. A randomised trial comparing 12 months of side-alternating whole-body-vibration training, resistance training and a control condition in postmenopausal osteopenic women found that neither intervention significantly changed lumbar-spine or total-hip BMD relative to control; all groups essentially maintained bone, and only the resistance-training group achieved significant improvements in health-related quality of life and postural control [30]. The authors suggested the 12-month period and the vibration and resistance intensities used (the latter around 70% one-repetition maximum) may have been insufficient, and recommended intervention durations of at least 24 months [30]. Vibration protocols also remain highly heterogeneous in frequency, amplitude and duration, contributing to conflicting results across trials [1,30]. On balance, whole-body vibration may help maintain bone and improve lower-limb strength, balance and fall risk in women unable to perform heavier loading, but resistance training carries the stronger and more consistent evidence, particularly for the functional and quality-of-life outcomes that accompany bone maintenance [1,30].

Part of the discrepancy in the vibration literature is methodological. Animal studies have shown enhanced markers of bone formation and increased bone density with vibration, and some

human trials report BMD gains, yet others find no effect, and meta-analyses have been unable to resolve the question, largely because vibration parameters vary enormously across trials—reported frequencies span roughly 12 to 113 Hz, accelerations from a fraction of one to more than thirty times gravity, and amplitudes from a few thousandths of a millimetre to several millimetres [30]. Some evidence suggests that side-alternating platforms delivering higher accelerations in a static squat position are the most osteogenic configuration, but when such settings were applied to osteopenic women the effects on spine and femoral-neck density were nonetheless absent, and the negative T-bone trial used a comparable side-alternating protocol [30]. For the peri- and early-postmenopausal woman, whole-body vibration is therefore best regarded as a possible maintenance or adjunctive modality—useful chiefly for improving strength, balance and fall risk in those who cannot undertake heavier loading—rather than as a substitute for the resistance and impact training that more reliably build bone and improve quality of life [1,30].

9.3. Exercise versus, and alongside, hormone therapy and other pharmacotherapy

Menopausal hormone therapy is a highly effective bone-protective treatment: Women's Health Initiative data show that oestrogen, with or without progestogen, reduces hip and vertebral fractures by close to one third, and a systematic review comparing hormone therapy with exercise concluded that both improve BMD but that hormone therapy generally produces larger gains at high-risk sites such as the lumbar spine and hip [2,33]. Discontinuation of hormone therapy is not neutral, being followed by renewed bone loss, and its use is constrained by risks including venous thromboembolism, stroke and breast cancer that must be weighed individually [2,33]. Exercise, and resistance exercise in particular, is therefore positioned not as a replacement for pharmacotherapy in women at high fracture risk but as a complementary strategy that additionally targets muscle strength, balance, functional capacity and fall risk—determinants that drugs do not address [2,6,33]. The evidence on additive effects is limited and mixed: some analyses suggest combined hormone therapy plus exercise yields greater BMD gains than either alone, especially at the femoral neck and lumbar spine and with mixed-loading rather than single-mode exercise, whereas a meta-analysis of controlled trials found the increment of adding exercise to hormone therapy at the spine and femoral neck to be small and not statistically significant [33]. Similarly, three older trials found no additional BMD benefit from adding moderate-intensity resistance or impact training to bisphosphonates, though the exercise interventions were probably sub-optimal and the participants not necessarily osteoporotic [6]. Because the mechanism of high-intensity loading (down-regulating sclerostin and activating Wnt signalling) overlaps with that of anti-sclerostin therapy such as romosozumab, the combination of potent osteoanabolic pharmacotherapy with bone-targeted exercise is a promising but as-yet-untested avenue in humans, currently under prospective investigation [6]. For the individual peri- or early-postmenopausal woman, the practical message is that exercise and pharmacotherapy are complementary rather than competing, and that pharmacological decisions should follow individualised fracture-risk assessment [2,33].

Understanding why hormone therapy is so effective clarifies the complementary logic. Oestrogen directly reverses the cellular drivers of postmenopausal bone loss—restoring OPG production and restraining RANKL to suppress osteoclastogenesis, reactivating Wnt/β-catenin signalling, promoting osteoblast survival and mesenchymal commitment to the osteoblastic lineage, and reducing osteoclastogenic cytokines—so it addresses the fundamental hormonal cause of the disease and produces larger, systemic BMD gains than loading, which acts locally at loaded sites [2,5]. Hormone therapy is most bone-protective when begun before age 60 or within ten years of menopause, in keeping with the timing hypothesis, and its bone benefit is lost after discontinuation, when accelerated loss resumes [2,33]. The available pharmacological

armamentarium also includes anti-resorptives such as bisphosphonates and denosumab, selective oestrogen-receptor modulators such as raloxifene and bazedoxifene, and osteoanabolic agents such as teriparatide, abaloparatide and the anti-sclerostin antibody romosozumab, each with distinct indications and risks [2]. Because these agents correct bone metabolism while exercise additionally builds muscle, improves balance and prevents falls, the two approaches are genuinely complementary; the outstanding question is whether they are additive, and the mechanistic overlap between high-intensity loading and anti-sclerostin therapy makes their combination a particularly rational, if still untested, avenue for the highest-risk women [2,6].

9.4. Nutritional and other adjuncts

Mechanical loading operates within a nutritional context. Adequate calcium and vitamin D are recommended to support bone mineralisation, with typical postmenopausal targets of about 1,200 mg calcium and 800–2,000 IU vitamin D daily, though contemporary guidance favours tailoring supplementation to deficiency risk rather than universal prescription, and meta-analyses show only modest fracture benefit from calcium and vitamin D with possible adverse effects of high calcium intake [2,12]. Vitamin D deficiency is prevalent after menopause and is associated with lower muscle strength, impaired mobility and increased fall risk, so its correction is a reasonable adjunct to resistance training, particularly in deficient women, even though supplementation alone does not reliably improve BMD in replete individuals [12,34]. Protein intake sufficient to support muscle protein synthesis (of the order of 1.0–1.2 g/kg/day in healthy older adults, higher in illness) complements resistance training for sarcopenia, and leucine-enriched protein and, in some studies, omega-3 fatty acids may enhance muscle responses, although the evidence for omega-3 and for many micronutrients remains inconsistent [12,34]. A review of vitamin D and omega-3 for muscle health concluded that vitamin D regulates myocyte proliferation, differentiation and regeneration and that its deficiency is linked to weakened strength and greater fall risk, so correction of deficiency is a reasonable adjunct, but that supplementation effects on physical performance depend on baseline 25-hydroxyvitamin D status, dose and duration and are not uniform; omega-3 fatty acids showed anti-inflammatory effects and some benefit for lower-limb isometric strength when combined with resistance training, but their impact on muscle mass was inconsistent, and a large trial of vitamin D3 and omega-3 found no effect on grip strength, gait speed or balance in healthy adults [34]. The consistent theme is that nutritional adjuncts are most useful for correcting genuine deficiency and for potentiating the response to resistance training rather than for acting alone, reinforcing the primacy of mechanical loading [12,34]. Osteosarcopenia protocols accordingly combine progressive resistance exercise with protein, calcium and vitamin D supplementation as a multidisciplinary strategy, reflecting the consensus that nutrition potentiates but does not replace mechanical loading [10,13]. Overall, the adjunctive evidence supports ensuring nutritional adequacy—especially of vitamin D and protein—as a foundation upon which resistance and impact training can act, rather than as an independent substitute for loading [12,34].

9.5. Resistance exercise to protect bone and muscle during weight loss

A further, increasingly relevant role for resistance training in postmenopausal women arises during intentional weight loss. Obesity is highly prevalent after menopause, and effective weight-loss strategies—caloric restriction, bariatric surgery and, most recently, incretin-based pharmacotherapies capable of 15–25% weight loss—reduce not only fat but also lean muscle and bone, with lean mass accounting for up to half of total weight loss and recent trials showing

modest spine and total-hip BMD loss with these treatments [6]. Because rapid weight loss can therefore worsen both sarcopenia and osteoporosis, resistance exercise has a protective role: a secondary analysis of a one-year trial in predominantly female obese adults showed that adding high-intensity resistance and aerobic exercise preserved bone mass during pharmacological weight loss, consistent with the established capacity of resistance training to increase muscle mass, strength and physical function while supporting bone [6,22]. This intersection is important for the peri- and early-postmenopausal woman who is simultaneously managing weight and skeletal-muscle health, since an unsupervised weight-loss programme without loading may accelerate the very musculoskeletal decline that menopause already drives, whereas pairing weight loss with progressive resistance and impact training may protect bone and muscle while the fat is lost [6,22]. Trials designed to test whether resistance exercise, alone or with anti-resorptive medication, can preserve BMD, bone microarchitecture and lean mass during dietary or pharmacological weight loss are ongoing, and their results will refine guidance for this common clinical scenario [6].

10. Safety, adherence and the durability of training effects

Concerns about the safety of vigorous loading in women with fragile bone have historically limited the prescription of the very high-intensity protocols that are most osteogenic [6,16]. The recent high-intensity resistance and impact trials substantially allay these concerns for appropriately screened women: supervised training was safe and well tolerated, produced no increase in vertebral fracture or thoracic kyphosis even among participants with prior vertebral fracture, and reported only minor, self-limiting musculoskeletal events comparable to those in moderate-intensity comparators [6,16]. Safety, however, is conditional on implementation. The essential principles are professional supervision, individualisation to each woman's bone status and functional capacity, an initial familiarisation phase to establish correct lifting and landing technique, and progressive graduation of intensity; movements involving extreme, rapid trunk flexion or forceful spinal rotation under load should be avoided in women with established osteoporosis, and impact should be introduced gradually [6]. Women with a recent vertebral fracture, uncontrolled cardiovascular disease or other contraindications require particular caution, as such patients were excluded from the supporting trials [6]. Within these constraints, the risk–benefit balance of supervised resistance and impact training in peri- and early postmenopausal women is favourable [6,16].

Prescription should be stratified by bone status and risk. In women with normal or mildly reduced bone mass—many of those in the early transition—high-intensity resistance and progressively introduced impact loading can be pursued relatively confidently under supervision, because the osteogenic gain is greatest and the fracture risk during exercise is low [6,25]. In women with established osteoporosis or a history of fragility fracture, the same principles apply but with greater caution: loads and impact are graduated more slowly, spinal-flexion and rotational loading under load are avoided, and closer supervision is warranted, yet the trial evidence—including women with prior vertebral fracture who came to no harm—indicates that appropriately delivered high-intensity training remains feasible and beneficial rather than contraindicated [6]. The group in whom evidence is genuinely lacking, and in whom caution should be greatest, comprises women with a very recent vertebral fracture or unstable comorbidity, for whom high-intensity protocols have not been tested; here a more conservative, individually supervised approach, or deferral until healing, is prudent [6]. This risk-stratified framework allows the substantial benefits of loading to be extended safely across most of the peri- and early-postmenopausal population while respecting the residual uncertainties at the highest-risk margin [6,16].

The benefit of exercise depends on adherence and continuity, both of which are challenging to

sustain. In a European workplace project, a structured combined programme for menopausal osteopenic women achieved better retention and lower dropout when delivered in the workplace than in an external sports centre, suggesting that reducing barriers such as travel time can improve compliance, although even the sports-centre participants who completed the programme showed high attendance [29]. Enjoyment also matters: participants in high-intensity programmes have reported greater enjoyment than those in low-intensity home-based exercise, which may aid persistence [6]. Barriers commonly cited by menopausal women include lack of time, limited access to facilities and, for some, health concerns, and strategies that address these—supervision, group support, home options and integration into daily routines—are likely to improve long-term engagement [29]. The workplace experience is instructive: embedding a structured programme within the working environment, where facilities were available and travel was minimised, reduced dropout relative to an external centre, suggesting that lowering the practical cost of participation is at least as important as the content of the programme itself, and that health systems and employers may play a role in enabling sustained exercise [29]. Delivery models that preserve intensity while improving convenience—supervised sessions combined with structured home exercise, or telehealth-supported programmes—warrant further study as means of reconciling the osteogenic need for high, well-taught loading with the behavioural need for accessibility and enjoyment [6,29].

Durability is a further limitation. The osteogenic and functional adaptations to loading are not permanent and regress when training stops. A systematic review of physical activity and detraining in postmenopausal women found that the beneficial effects on BMD, strength and functional performance achieved during training tend to decline once structured exercise ceases, with strength and functional gains generally lost after detraining and BMD gains variably eroded, sometimes returning toward baseline or control levels over about 12 months [27]. A five-year follow-up of one exercise cohort found that the BMD benefits were entirely lost after prolonged cessation, whereas continued participants in the extension of a high-intensity trial exhibited ongoing improvement, and lower maintenance frequencies may suffice to retain earlier gains [6,27]. Because bone remodelling is slow, detraining research is scarce, and the optimal maintenance dose is not established, but the consistent message is that continuity is essential and that programmes should be designed for lifelong sustainability, including feasible maintenance phases that preserve roughly one third of the training dose to retain adaptations [6,27]. The clinical implication for the peri- and early-postmenopausal woman is that exercise is not a time-limited course but a permanent component of skeletal and muscular self-care [27].

Two further nuances refine the detraining message. First, the rate of loss differs between tissues: muscular strength and functional performance, which depend substantially on neural adaptations and muscle quality, tend to decline relatively quickly once training stops, whereas bone, with its slow remodelling cycle, changes more gradually, so the window in which resumed training can recover recent losses may be longer for bone than for muscle [27]. Second, maintenance need not fully replicate the building programme: evidence suggests that once gains have been achieved, a reduced frequency or volume may retain much of the benefit, and continued participants in the extension of a high-intensity trial preserved or even improved their gains, implying that a sustainable maintenance dose—rather than indefinite high-volume training—may suffice for many women [6,27]. These observations are practically important because they make lifelong adherence more realistic: women can be counselled that an intensive building phase can transition to a lighter but regular maintenance phase, and that the goal is permanent engagement at whatever level is sustainable rather than an unattainable ideal that is abandoned altogether [6,27]. The optimal maintenance dose is not yet defined and is a priority for future research, but the qualitative principle—continuity matters more than intermittent intensity—is well supported [27].

11. Practical exercise prescription

Integrating the evidence, a prudent exercise prescription for the prevention of osteoporosis and sarcopenia in peri- and early postmenopausal women follows the frequency, intensity, type and time framework and combines bone-targeted loading with fall-prevention and muscle-preserving components [6,9,15]. For bone, the best-supported approach is supervised, progressive high-intensity resistance training—free-weight, multi-joint exercises that load the spine and hip (for example deadlift, squat and overhead press) at approximately 70–85% one-repetition maximum for about 5–8 repetitions and two to three sets—performed two to three times per week and combined with a graded, individually tolerated impact/jump component, for a minimum of six months and ideally continued indefinitely [6,15,16]. For women who cannot tolerate heavy or high-impact loading, because of severe osteoporosis, osteoarthritis, joint pain or frailty, lower-intensity or light-load power training, elastic-band resistance and body-weight exercises still stabilise bone and improve strength and quality of life and are appropriate substitutes [19,21,25].

Individualisation begins with assessment. Before high-intensity prescription, and particularly in women with established osteoporosis or a fracture history, a clinical evaluation of bone status (DXA, T-score and, where relevant, fracture-risk estimation), functional capacity, comorbidity and movement competence should guide the starting intensity and the rate of progression [2,6]. The programme is then tailored: heavy loading is graduated from a familiarisation phase, impact is introduced progressively from low-level tasks, and exercises that impose extreme loaded trunk flexion or rotation are avoided in women with spinal fragility [6]. Baseline sarcopenia status, obesity, vitamin-D and protein sufficiency, and personal preference and access all influence the choice of mode, setting and adjuncts, and the evidence that responses vary with age, hormonal status and baseline bone and muscle underscores that no single protocol suits every woman [6,10,20]. This individualised, assessment-led approach maximises both safety and efficacy, and it is the practical expression of the principle—recurring throughout this review—that the benefit of resistance and impact training depends not merely on doing it but on doing it at an adequate, progressive and sustainable intensity matched to the individual [6,15].

For muscle and function, resistance training targeting the major muscle groups, two to three sets of 8–12 repetitions at moderate-to-high intensity about three times weekly, should be complemented by high-velocity/power work to restore type II-fibre power and by progressive balance and functional exercises that challenge stability and simulate real-life fall scenarios, since multicomponent programmes reduce falls most effectively [6,9,20]. Sessions of roughly 30–60 minutes, incorporating warm-up and cool-down, are typical, and short sets with adequate rest are preferable to long continuous loading because they respect the recovery requirement of bone cells and permit maximal power output [9,19]. Throughout, the non-negotiable principles are professional supervision and technique instruction, individualisation to bone status and comorbidity, progressive overload, and attention to nutritional adequacy of protein, calcium and vitamin D [6,9,12]. Programmes delivered in accessible settings, made enjoyable and designed for lifelong continuation are most likely to translate physiological efficacy into durable clinical benefit [27,29].

Table 2. A frequency–intensity–type–time framework for exercise prescription in peri- and early postmenopausal women, by prevention goal.

Parameter	Bone (osteoporosis)	Muscle (sarcopenia)	Balance / falls
Frequency	2–3 osteogenic sessions per week	2–3 sessions per week	Most days; integrated into other sessions
Intensity	High: ~70–85% 1RM	Moderate–high: >60% 1RM,	Progressive difficulty of

Parameter	Bone (osteoporosis)	Muscle (sarcopenia)	Balance / falls
	resistance + impact with ground-reaction forces well above walking; progress gradually	progressing toward repetition-maximum loading	balance challenge; challenge stability
Type	Free-weight multi-joint lifts loading spine and hip (deadlift, squat, overhead press) + graded jump/impact	Progressive resistance for major muscle groups + high-velocity/power work for type II fibres	Dynamic and static balance, functional and coordination tasks simulating fall scenarios
Time	~30–60 min; short sets with rest to preserve mechanosensitivity	2–3 sets of 8–12 reps; ~30–60 min	10–15 min within session; ongoing
Duration to effect	≥6 months; ideally 12–24 months and continued indefinitely	Strength gains within 8–12 weeks; mass slower	Reduced falls within 3–6 months of regular practice
Lower-tolerance option	Light-load power training, elastic-band and body-weight resistance still stabilise bone	Elastic-band and body-weight resistance improve strength and quality	Seated or supported balance work; near firm support
Safeguards / adjuncts	Supervision, individualisation, familiarisation, avoid loaded trunk flexion/rotation in osteoporosis; ensure calcium and vitamin D	Adequate protein (~1.0–1.2 g/kg/day), vitamin D; supervision	Supervision; progress only when technique is secure

1RM, one-repetition maximum. The framework synthesises evidence discussed in the text; it is not a substitute for individualised clinical assessment, which should precede high-intensity prescription, particularly in women with established osteoporosis, recent fracture or relevant comorbidity.

12. Limitations of the evidence and future directions

Several limitations constrain the strength of current recommendations. First, the literature is markedly heterogeneous in the exercise types, intensities, frequencies and durations studied and in how key parameters—especially intensity—are defined, so pooled effect sizes are often small, confidence in meta-analyses is frequently rated low or very low, and apparently conflicting conclusions can usually be traced to methodological rather than biological differences [6,14,16]. Second, many trials were short (under 12 months), under-powered, or conducted in already-active or heterogeneous populations, and few isolated the peri- and early-postmenopausal window in which bone loss is fastest, limiting the direct applicability of pooled findings to the target group [6,16]. Third, most bone outcomes rely on areal BMD by DXA, which captures only part of bone strength and can understate the structural and geometric benefits of loading, particularly at the hip; wider use of quantitative computed tomography, peripheral quantitative computed tomography and three-dimensional hip analysis is needed to quantify the true skeletal response [6,7]. Fourth, no trial has been adequately powered to demonstrate fracture reduction as a primary endpoint, so the ultimate clinical benefit is inferred from BMD, falls and structural surrogates rather than proven directly [6]. Fifth, the acute-response evidence indicating blunted osteoanabolic responsiveness to impact loading after oestrogen loss raises unresolved questions about how much of the bone benefit of loading is preserved in older postmenopausal women, and whether higher intensities are required to overcome a raised adaptation threshold [6,17]. Sixth, the participants of the trials that best support high-intensity training were carefully screened, independently mobile and free of recent fracture, so their results may not generalise to frailer women or to those with severe osteoporosis, recent vertebral fracture or significant comorbidity, in whom safety and efficacy are less certain [6]. Seventh, many trials and cohorts

relied on self-reported physical activity or on surrogate bone measures such as calcaneal quantitative ultrasound rather than DXA, introducing measurement error, and populations, baseline activity and bone status varied widely, all of which inflate heterogeneity and complicate synthesis [14,28].

These gaps define a clear research agenda. Future work should include adequately powered, long-term (≥24-month) randomised controlled trials of standardised, well-described high-intensity resistance and impact protocols specifically in peri- and early postmenopausal women, using structural as well as densitometric endpoints and, ideally, fracture outcomes [6,7,16]. Dose–response studies are needed to define the minimum effective and the minimum maintenance doses of loading, the optimal intensity for the postmenopausal skeleton, and the safety and efficacy of high-intensity exercise in women with recent vertebral fracture [6,27]. The interaction of bone-targeted exercise with pharmacotherapy—particularly osteoanabolic agents that share the Wnt/sclerostin mechanism—and with nutritional and myokine-mediated pathways deserves prospective evaluation, as does the individualisation of programmes to baseline BMD, sarcopenia status, comorbidity and preference [6,10,12]. Finally, implementation research on adherence, delivery setting and long-term sustainability is essential if the demonstrated physiological efficacy of resistance and impact training is to be realised as durable public-health benefit in this large and growing population [27,29].

13. Conclusions

Peri- and early postmenopause is a critical window in which oestrogen withdrawal accelerates the loss of both bone and muscle, converging on the linked syndromes of osteoporosis, sarcopenia and osteosarcopenia. Because bone and muscle adapt to mechanical demand through mechanotransduction, and because pharmacotherapy does not address muscle strength, balance or fall risk, mechanical loading is a physiologically rational and clinically important non-pharmacological cornerstone of prevention. The evidence indicates that resistance training reliably preserves and modestly increases bone mineral density at the fracture-relevant lumbar spine and, less consistently in areal terms, the proximal femur, with the largest gains achieved by high-intensity, progressive protocols performed about three times weekly for at least six to twelve months. Combining resistance with jump/impact loading in supervised high-intensity resistance and impact training provides the most robust osteogenic stimulus at the spine and improves hip structure, and it is safe and well tolerated when delivered with supervision, individualisation, familiarisation and progressive intensity in appropriately screened women. Resistance and power training simultaneously improve muscle strength, physical performance, body composition, balance, fall risk and quality of life, addressing the muscular and functional dimensions of osteosarcopenia that drugs cannot.

Two nuances shape prescription in this population. Oestrogen deficiency appears to blunt the skeleton’s anabolic response to impact loading, so early intervention—while responsiveness is greater—and adequate intensity are both important, and vigorous plyometrics should be applied cautiously and in graded, supervised form after oestrogen loss. For women who cannot tolerate heavy or high-impact work, lighter power training, elastic-band and body-weight resistance still confer bone, muscle and quality-of-life benefit. Exercise complements rather than replaces pharmacotherapy and nutritional adequacy of protein, calcium and vitamin D, and its benefits regress with detraining, so it must be sustained for life. Within the limits of a heterogeneous evidence base and the absence of adequately powered fracture trials, supervised, progressive resistance training combined with impact loading, tailored to the individual and integrated with fall-prevention and nutritional strategies, represents the best-supported exercise approach to preventing osteoporosis and sarcopenia in peri- and early postmenopausal women.

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