



Case Study

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Bone Mineral Density Recovery Patterns After Osteoporotic Hip Fracture Repair

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Abstract

Osteoporotic hip fracture repair restores mechanical continuity to the femur, but bone mineral density (BMD) at the operated and contralateral hip often continues to decline for months to years afterward unless actively addressed. This article synthesizes the literature on BMD recovery patterns following hip fracture repair, covering the epidemiological scale of the problem, the biology of post-fracture bone loss and its coupling to muscle loss in osteosarcopenia, how BMD trajectory and fracture risk are measured with DXA and FRAX in this population, and what pharmacological, nutritional, and exercise-based interventions have been shown to modify that trajectory. We review evidence that antiresorptive therapy initiated promptly after hip fracture improves fracture and mortality outcomes with a broadly consistent BMD response across subgroups, that correcting the vitamin D and calcium deficits common in this population reduces subsequent hip fracture risk, and that progressive resistance training produces a measurable, site-specific BMD gain at the hip. We also review the organizational evidence for orthogeriatric co-management and fracture liaison services, the care models most consistently associated with translating this evidence into practice. Read together, the literature supports a reasonably well-characterized biological and clinical picture of modifiable post-fracture BMD decline, while leaving direct head-to-head pharmacological comparisons in post-hip-fracture cohorts, and the implementation of secondary-prevention pathways in lower-resource, higher-growth settings, as open priorities.

Keywords: bone mineral density; hip fracture; osteoporosis; fracture liaison service; osteosarcopenia

1. Introduction

An osteoporotic hip fracture is rarely a single, self-contained event. Surgical fixation or arthroplasty restores mechanical continuity to the femur within days, but the skeleton around the repair, and the contralateral hip that shares the same systemic bone biology, continues to remodel for months to years afterward, and not always in the direction clinicians would like. A patient who is discharged walking on a well-fixed implant can still be losing bone mineral density (BMD) at the hip, the spine, and the wrist, driven by the same combination of advanced age, prior fragility, acute immobilization, and untreated osteoporosis that produced the index fracture in the first place. Understanding the trajectory of BMD after hip fracture repair, not just whether the fracture itself has healed, is therefore central to preventing the second fracture, which carries its own elevated mortality and disability burden.

This is also a genuinely global problem, and one whose scale is still growing. Recent burden-of-disease modeling projects continued increases in hip fracture incidence through 2050 as populations age, with substantial geographic and temporal disparities in who is affected and how care systems respond [1]. Region-specific projections tell a similar story outside the traditionally best-studied high-income settings: modeling for South Africa, for instance, projects a marked rise in the absolute number of hip fractures over the coming decades even under conservative incidence assumptions, a pattern that is likely to be mirrored across other low- and middle-income countries whose osteoporosis infrastructure has historically been thin [2]. Every one of those fractures initiates the same clinical question: once the hip is fixed, what happens to bone density, and what can be done about it.

This article synthesizes the literature on BMD recovery patterns after osteoporotic hip fracture repair. We are deliberately not presenting new experimental data; instead, we trace what has actually been measured and reported across five threads: the epidemiological scale and clinical stakes of the problem; what is understood about the biology of bone loss and remodeling around a healing fracture; how BMD recovery is actually measured and risk-stratified in practice; what pharmacological, nutritional, and exercise-based interventions have been shown to modify the BMD

trajectory; and the systems of care, fracture liaison services and orthogeriatric co-management chief among them, that determine whether any of this evidence reaches the patient at all. We close by naming the gaps that remain, because the honest state of this literature is that BMD "recovery" after hip fracture is unevenly defined, inconsistently measured across studies, and, for a meaningful fraction of patients, does not happen without active intervention.

It is worth being explicit about scope from the outset. Much of what is labeled "recovery" in this literature is not a return of BMD to pre-fracture levels; it is a slowing or reversal of an otherwise-expected decline, frequently under pharmacological treatment, and frequently measured against a background of profound heterogeneity in age, frailty, sex, fracture type, and surgical fixation method. Readers should treat "recovery" throughout this article as a descriptive term for the measured BMD trajectory following repair, not as a claim that any particular intervention restores skeletal health to a pre-fracture baseline.

It is also worth being explicit about what this article is not. It is not a clinical practice guideline, and it does not attempt to rank interventions by strength of recommendation the way a formal guideline panel would. It is a synthesis of what the published literature has actually measured and reported about BMD behavior after hip fracture repair, organized so that the underlying evidence for each commonly cited intervention, pharmacological, nutritional, mechanical, and organizational, can be inspected on its own terms. Where a claimed effect could not be traced to a specific, verifiable figure in the cited source, we have either omitted the claim or stated it qualitatively rather than attaching an unsupported number to it.

2. Epidemiology and Clinical Burden of Osteoporotic Hip Fracture

The demographic pressure behind this topic is not subtle. Global and regional burden estimates built on standardized disease-burden frameworks project that the absolute number of hip fractures in older adults will continue climbing for decades, driven overwhelmingly by population aging rather than by any single rise in age-specific incidence, with the geographic distribution of that burden shifting toward regions whose health systems are least prepared for it [1]. The South African projection work makes the same point at national scale:

even using stable or modestly changing age-specific fracture rates, the growth of the elderly population alone is projected to substantially increase the annual count of hip fractures requiring surgical repair over the coming decades [2]. Neither of these analyses is a BMD study in the narrow sense, but both establish why BMD recovery after repair is not a niche clinical question; it scales with a growing denominator of survivors who will spend years living with the skeletal consequences of the fracture that brought them to surgery.

The clinical stakes of what happens after that surgery are high enough that hip fracture care has increasingly been organized around models explicitly designed to intervene early. Systematic reviews of post-surgical interventions after hip fracture, spanning rehabilitation, nutrition, and secondary-prevention pharmacotherapy trials, consistently frame the post-operative period as a window in which multiple, compounding forms of decline (bone, muscle, function, and independence) are all in motion simultaneously and are, to a meaningful degree, modifiable [3]. That framing matters for how BMD recovery specifically should be read: it is one strand of a broader post-fracture syndrome, not an isolated biomechanical variable, and interventions aimed at one strand (protein intake, resistance exercise, antiresorptive therapy) routinely show up in the literature on the others as well.

Guideline bodies have converged on treating this window as a mandate rather than an option. The UK's national clinical guideline for osteoporosis prevention and treatment explicitly frames the period immediately after a fragility fracture, hip fracture very much included, as the highest-yield opportunity in the entire osteoporosis care pathway, on the grounds that fracture itself is among the strongest available predictors of near-term subsequent fracture risk [4]. The European guidance for diagnosis and management of postmenopausal osteoporosis takes a structurally similar position, integrating fracture risk assessment, BMD measurement, and treatment initiation into a single continuum of care that is explicitly meant to begin at, not years after, the index fracture [5]. Both bodies of guidance treat BMD trajectory after hip fracture less as an academic outcome measure and more as the quantity that secondary-prevention care is ultimately trying to move.

3. Bone Remodeling and the Biology of

Post-Fracture BMD Loss

Two largely independent biological processes are superimposed in the months after hip fracture repair, and disentangling them is part of what makes "BMD recovery" a slippery term. The first is local fracture healing, which involves its own transient, site-specific remodeling activity around the fracture line and is largely a mechanical and biological question about the implant-bone construct rather than a systemic BMD question. The second, and the one this article is primarily concerned with, is systemic and regional bone loss driven by the same mechanisms that produced the fracture in the first place: age-related decline in bone formation relative to resorption, estrogen or androgen deficiency, secondary causes of bone loss, and, acutely, the disuse and immobility that follow major lower-limb surgery in an already frail, often bed- or chair-bound older adult. These two processes are not independent in practice, because the same patient experiencing acute disuse-related bone loss at the contralateral hip is simultaneously trying to heal the operated one, and the systemic hormonal and inflammatory environment of major surgery in an older adult affects both.

Muscle and bone loss after hip fracture are increasingly understood as coupled rather than parallel phenomena, under the umbrella term osteosarcopenia, the simultaneous presence of osteopenia or osteoporosis and sarcopenia in the same patient [6]. This coupling is mechanistically plausible and clinically consequential: skeletal muscle provides the mechanical loading that bone needs to maintain density, and the same catabolic drivers of aging, inflammation, and reduced anabolic hormone signaling, that erode muscle mass and strength also erode bone. In patients presenting with hip fracture specifically, osteosarcopenia is not a rare comorbidity but a common baseline state, and its presence at the time of fracture is associated with substantially worse downstream outcomes, including elevated mortality relative to hip fracture patients without concurrent sarcopenia [7]. Postoperative functional outcomes and the risk of a subsequent fracture are similarly worse in patients whose hip fracture occurred against a background of osteosarcopenia rather than isolated osteoporosis, which is one reason muscle-directed and bone-directed interventions are increasingly discussed together in this literature rather than as separate clinical problems [8].

A pragmatic implication follows directly from this biology: any account of BMD recovery after hip fracture repair that treats bone density as an isolated, purely skeletal variable is incomplete. The trajectory of BMD in the year or more after repair is entangled with mobility, nutritional status, and muscle mass in ways that make purely pharmacological or purely mechanical explanations of "recovery" insufficient on their own, a point the real-world cohort data discussed later in this article illustrates directly.

4. Measuring Recovery: DXA, FRAX, and the Trajectory of BMD After Repair

Dual-energy X-ray absorptiometry (DXA) remains the practical backbone of BMD measurement in this population, but its use after hip fracture surgery is not as straightforward as in a treatment-naïve osteoporosis patient. The operated hip itself is frequently unsuitable for reliable serial DXA measurement because of the metal implant, meaning that longitudinal tracking after unilateral hip fracture repair typically relies on the contralateral, non-operated hip, the lumbar spine, or both, introducing an implicit assumption, not always stated explicitly in the underlying studies, that the non-operated side is a valid proxy for whole-skeleton BMD trajectory. Combined with variable timing of baseline scans relative to the fracture (sometimes obtained pre-injury, sometimes only after surgery once the patient is stable enough to be positioned for imaging), the resulting BMD trajectory data in this literature carry more measurement heterogeneity than is always acknowledged.

Absolute BMD values are also, on their own, an incomplete risk signal, which is why fracture risk assessment tools have become a standard companion to DXA in this population. FRAX, the most widely used such tool, combines femoral neck BMD (when available) with clinical risk factors, age, prior fracture, glucocorticoid use, parental hip fracture history, and others, to generate an absolute ten-year probability of hip and major osteoporotic fracture, and was explicitly validated, in its original derivation and subsequent updates, for use in men and women across a range of national cohorts [9]. A recent overview of FRAX's clinical use makes the point relevant to this article directly: FRAX is most informative, and most frequently used in practice, precisely in patients who already have a fragility fracture on their history, since prior fracture is one of the tool's strongest

single risk inputs, making post-hip-fracture patients close to the paradigmatic use case for the instrument rather than an edge case [10]. In practice, this means the same patient being followed for BMD recovery after hip fracture repair is very often simultaneously being risk-stratified by FRAX, and the two measurements, a raw BMD trajectory and a composite fracture-probability estimate, are frequently reported together in the clinical secondary-prevention literature even though they answer subtly different questions.

What the trajectory itself typically looks like, when it is tracked at all, is a story of divergence rather than a single curve: patients who receive timely antiresorptive or anabolic therapy and adequate nutritional and mechanical loading tend to show stabilization or modest gains in BMD at the measured sites over the following one to two years, while patients who do not, whether because treatment was never initiated, adherence lapsed, or comorbidity and frailty precluded intervention, tend to show continued decline that mirrors or exceeds their pre-fracture rate of loss. The sections that follow examine what specifically has been shown to shift a given patient from the second group into the first.

A further practical complication is timing. FRAX and DXA are both, in principle, snapshot instruments, yet the post-hip-fracture period is anything but static: fracture risk, functional status, and nutritional status are all in flux simultaneously in the weeks and months after surgery, which means a single baseline assessment obtained too early, while the patient is still acutely unwell, or too late, after avoidable bone and muscle loss has already accrued, can under- or over-state the risk a given patient actually carries by the time treatment decisions are made. The FRAX literature itself acknowledges this limitation implicitly by recommending recalculation as clinical circumstances change rather than treating a single post-fracture FRAX score as durable for years afterward [10]. In practice, most of the secondary-prevention pathways discussed later in this article build in a second, formal reassessment at a fixed interval after the index fracture, commonly around twelve months, precisely to capture this dynamic rather than relying on the immediate post-operative snapshot alone.

5. Pharmacological Modulation of BMD Recovery

Antiresorptive therapy initiated after hip fracture has the

best-established evidence base for altering the post-fracture BMD and fracture-risk trajectory, and the pivotal trial in this specific population, initiating treatment after, not merely alongside, hip fracture, is the HORIZON Recurrent Fracture Trial. In that trial, patients who had recently undergone surgical repair of a hip fracture were randomized to annual intravenous zoledronic acid or placebo, and the treatment arm showed a reduction in new clinical fractures and in all-cause mortality relative to placebo over the follow-up period [11]. The trial's significance for this article is less about the topline fracture and mortality numbers, striking as they are, and more about what it established methodologically: that initiating potent antiresorptive therapy in the immediate post-hip-fracture window, rather than waiting for a subsequent formal osteoporosis diagnosis and referral pathway, is both feasible and effective, a finding that reshaped secondary-prevention guidelines for this population going forward.

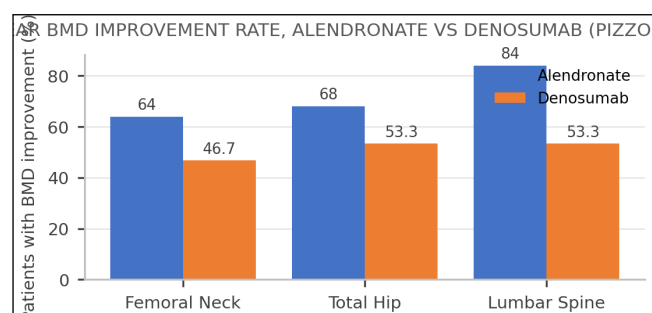


Figure 1: Proportion of hip fracture patients showing osteo-metabolic (bone mineral density) improvement at one-year follow-up under alendronate versus denosumab, by measurement site, in a prospective real-world cohort of very old adults with osteosarcopenia [14].

Subsequent analysis of the same trial population examined how the BMD response to zoledronic acid varied across patient subgroups, an important question given how heterogeneous the post-hip-fracture population is in age, frailty, and baseline bone status, and found that the direction and general magnitude of BMD benefit held across most examined subgroups, supporting zoledronic acid's use as a broadly applicable rather than narrowly targeted intervention in this population [12]. This subgroup consistency is clinically reassuring precisely because the post-hip-fracture population is not a homogeneous group of otherwise-healthy osteoporosis patients; it includes the oldest-old, patients with multiple comorbidities, and patients for whom trial enrollment criteria elsewhere in the osteoporosis literature might have excluded them altogether.

Denosumab, a fully human monoclonal antibody against RANKL with a fundamentally different mechanism from bisphosphonates, has its own pivotal evidence base in the FREEDOM trial, which, while conducted in postmenopausal women with osteoporosis rather than a purely post-hip-fracture cohort, established that denosumab reduces vertebral, hip, and nonvertebral fracture risk relative to placebo and is now a standard second-line, and increasingly first-line, option in the same secondary-prevention pathway that follows hip fracture repair [13]. Direct, real-world comparisons between an oral bisphosphonate (alendronate) and denosumab specifically in the post-hip-fracture population, rather than the broader postmenopausal osteoporosis population the pivotal trials enrolled, remain comparatively scarce, but the data that do exist are informative precisely because of that scarcity. A prospective cohort of very old adults with osteosarcopenia followed for one year after hip fracture, comparing alendronate and denosumab, found a numerically higher proportion of patients achieving BMD improvement in the alendronate group across all three measured sites, femoral neck, total hip, and lumbar spine, though the authors explicitly characterized the difference as non-significant given the small completer sample, while also noting a favorable trend in a skeletal muscle index measure favoring denosumab, a hint that the two drug classes may not be interchangeable once muscle outcomes are considered alongside bone outcomes in this specific, very frail population [14]. Figure 1 summarizes the reported BMD improvement rates from that cohort. The honest reading of this single, small, real-world study is not that one agent is clearly superior, but that treatment choice in the oldest and most osteosarcopenic post-hip-fracture patients may need to weigh bone and muscle outcomes jointly, a question the larger pivotal trials were not designed to answer because they did not enroll this specific, high-frailty subgroup at this specific post-fracture time point.

6. Nutrition, Muscle, and Non-Pharmacological Determinants of Recovery

Pharmacotherapy does not act on a blank slate, and the nutritional and mechanical environment a patient is in after hip fracture surgery substantially shapes whether antiresorptive or anabolic treatment can do its expected work. Vitamin D deficiency is strikingly common in this population; a recent review synthesizing multiple

cohorts of hip fracture patients found hypovitaminosis D prevalence estimates ranging from roughly 46% to 92%, depending on the specific threshold used to define deficiency and the population studied, meaning that a large fraction of patients entering the post-fracture recovery window are doing so with inadequate substrate for the calcium-handling and bone-mineralization processes that any antiresorptive or anabolic drug ultimately depends on [15]. That same review situates calcium and vitamin D co-supplementation as an evidence-based intervention in its own right rather than merely a supportive adjunct: meta-analytic evidence in community-dwelling and nursing-home older adults associates combined calcium and vitamin D supplementation with an approximately 25 to 30 percent reduction in hip fracture risk, and a cluster-randomized trial using dairy-food fortification as the delivery vehicle reported a 46 percent reduction in hip fractures, a 33 percent reduction in total fractures, and an 11 percent reduction in falls within a relatively short intervention window [15]. These are fracture-outcome numbers rather than direct BMD-trajectory numbers, but they bear directly on this article's subject because they describe the same nutritional substrate that determines whether a given patient's bone-remodeling machinery can respond to mechanical loading or pharmacological stimulation in the first place.

Table 1: Representative reported effects of pharmacological, nutritional, and exercise-based interventions relevant to bone mineral density recovery after osteoporotic hip fracture repair.

Intervention	Source	Population / Design	Reported Effect
Zoledronic acid (annual infusion)	Lyles et al. [11]	HORIZON-RFT, post hip-fracture patients	Reduced new clinical fractures and all-cause mortality versus placebo
Denosumab	Cummings et al. [13]	FREEDOM trial, postmenopausal osteoporosis	Reduced vertebral, hip, and nonvertebral fracture risk versus placebo
Alendronate vs denosumab	Pizzonia et al. [14]	Prospective cohort, 1-year post hip fracture (n=40 completers)	BMD improvement in 64-84% (alendronate) vs 46.7-53.3% (denosumab) across sites
Progressive resistance training	O'Bryan et al. [16]	Meta-analysis, 14 RCTs, older adults	Femur/hip BMD +2.77% (p=0.02); lumbar spine +1.60% (not significant)

Intervention	Source	Population / Design	Reported Effect
Calcium + vitamin D supplementation	Cianferotti et al. [15]	Meta-analyses, community and nursing-home older adults	~25-30% reduction in hip fracture risk; dairy-fortification trial: 46% hip fracture reduction

Mechanical loading, delivered through structured exercise, is the second major modifiable lever, and here the evidence is more directly BMD-specific. A systematic review and meta-analysis of fourteen randomized controlled trials of progressive resistance training in older adults found a statistically significant mean increase in femur or hip BMD of 2.77 percent relative to control conditions, alongside a smaller, non-significant increase of 1.60 percent at the lumbar spine [16]. The site-specific pattern, hip responding more robustly than spine, is mechanistically sensible given that hip-region BMD is more directly loaded by the resistance exercises typically studied (squats, leg press, hip-extension movements) than the lumbar spine is, and it is also clinically convenient, because the hip is precisely the site whose density is of most direct concern after hip fracture repair. It is worth being cautious about generalizing this meta-analysis, conducted in community-dwelling older adults broadly rather than in a post-hip-fracture-surgery population specifically, directly onto the post-surgical rehabilitation setting; the exercise tolerance, fall risk, and surgical precautions applicable to someone weeks or months removed from hip fracture repair differ substantially from the trial populations underlying this figure, and post-surgical rehabilitation protocols necessarily start from a much lower baseline of tolerated load. Nonetheless, the underlying principle, that mechanical loading through resistance exercise produces a measurable, site-specific BMD response even in older adults, provides a biological rationale for why structured rehabilitation programs following hip fracture are increasingly designed with explicit progressive-loading components rather than mobility restoration alone, a pattern reflected in the broader post-surgical intervention literature for this population [3]. Table 1 places the resistance-training and nutritional-supplementation evidence alongside the pharmacological findings discussed above, to make the relative magnitude of these different intervention categories easier to compare directly.

The muscle side of the osteosarcopenia coupling described earlier reinforces why these two levers,

nutrition and loading, are usually discussed together rather than separately in this literature. Because osteosarcopenia at the time of hip fracture predicts worse mortality and functional outcomes, and because adequate protein and vitamin D status is a prerequisite for skeletal muscle to respond to resistance training in the first place, nutritional optimization is increasingly framed not as a supportive afterthought to exercise-based rehabilitation but as a co-requisite for it to work at all [6,15].

7. Care Models and Secondary Fracture Prevention Systems

None of the pharmacological or lifestyle evidence reviewed above reaches patients automatically; it depends on a care system that reliably identifies post-hip-fracture patients, initiates appropriate treatment, and follows them long enough to know whether BMD is actually recovering. Two overlapping organizational models dominate this part of the literature: orthogeriatric co-management, in which geriatric medicine specialists are embedded in the surgical care pathway from admission onward, and fracture liaison services (FLS), which are specifically tasked with secondary-prevention follow-up, osteoporosis treatment initiation, and monitoring after the acute surgical episode concludes.

Systematic review and meta-analytic evidence on orthogeriatric care models finds consistent improvement across multiple outcome domains, including function and process-of-care measures, when geriatric co-management is embedded in hip fracture surgical pathways compared with standard orthopedic-only care, and identifies this integration as one of the more consistently reproducible findings across the health-services literature on hip fracture [17]. An earlier systematic review and meta-analysis reached a structurally similar conclusion, finding that orthogeriatric care models were associated with improved outcomes and shorter hospital stays relative to conventional orthopedic management, while also noting that the heterogeneity of co-management model design across studied institutions made precise quantitative pooling of effect sizes across all outcomes difficult [18]. Taken together, these two reviews, published roughly eight years apart and drawing on substantially different sets of underlying studies, represent a rare case of a hip-fracture-care intervention whose benefit has proven

durable and reproducible as the evidence base has matured, rather than shrinking under closer scrutiny as so often happens with early promising findings in this field.

Fracture liaison services extend that logic past hospital discharge into the specific secondary-prevention window this article is concerned with. A three-year outcomes analysis of a fracture liaison service found that structured follow-up, incorporating DXA-based reassessment, osteoporosis treatment initiation, and adherence monitoring, was associated with meaningful reductions in subsequent fracture incidence over the follow-up period relative to what would be expected from historical, non-FLS-managed comparators [19]. A separate evaluation of an inpatient rehabilitation-embedded FLS model, initiating the secondary-prevention pathway even earlier, during the inpatient rehabilitation stay rather than at outpatient follow-up, reported improved uptake of osteoporosis treatment and secondary-prevention process measures relative to usual care, reinforcing the general finding in this literature that earlier initiation of structured secondary prevention, rather than any single drug choice, is the single largest lever available for improving population-level outcomes after hip fracture [20].

The guideline literature has absorbed this evidence directly into its recommendations. Both the UK national osteoporosis guideline and the European guidance for postmenopausal osteoporosis management explicitly recommend structured secondary-prevention pathways, whether formally branded as FLS or delivered through equivalent orthogeriatric-embedded processes, as standard of care following any fragility fracture, hip fracture representing the highest-priority case given its associated mortality and disability burden [4,5]. Read alongside the pharmacological and nutritional evidence discussed earlier, the picture that emerges is one in which the biological tools for improving post-hip-fracture BMD trajectory are reasonably well established, and the primary remaining bottleneck is organizational: whether a given health system reliably delivers those tools to the patients who need them, in the narrow window after surgery when they are most likely to be effective.

8. Open Challenges

Several gaps recur across this literature and deserve to be stated plainly rather than left implicit. First,

measurement heterogeneity: because the operated hip is frequently unsuitable for reliable serial DXA, most longitudinal BMD data after hip fracture repair rely on the contralateral hip or lumbar spine as a proxy, an assumption that is rarely validated within the same study and that likely introduces unquantified noise into reported recovery trajectories across the literature as a whole.

Second, the post-hip-fracture population is precisely the population most likely to be underrepresented in the pivotal trials whose results are used to guide its treatment. The HORIZON Recurrent Fracture Trial is a partial exception in that it explicitly enrolled patients after hip fracture [11], but much of the broader antiresorptive and exercise evidence base, including the resistance-training meta-analysis discussed above, is drawn from community-dwelling older adults rather than patients in the acute post-surgical recovery window specifically [16], leaving open questions about how directly those effect sizes transfer to a frailer, more recently operated population.

Third, direct head-to-head BMD-trajectory comparisons between different antiresorptive or anabolic agents specifically in post-hip-fracture cohorts remain scarce; the single real-world cohort comparing alendronate and denosumab in this article's Figure 1 is illustrative precisely because it is one of relatively few such comparisons, and its small completer sample and high dropout rate limit how far its findings can be generalized [14]. Larger, adequately powered, post-hip-fracture-specific comparative trials would meaningfully strengthen this evidence base.

Fourth, the osteosarcopenia framework, while conceptually compelling and consistently associated with worse outcomes [6,7,8], has not yet produced a correspondingly mature evidence base of interventions specifically designed and tested for the combined bone-and-muscle phenotype in the post-hip-fracture population, as opposed to bone-focused and muscle-focused interventions studied largely in parallel.

Fifth, and more of a systems gap than a biological one: even where the organizational evidence for orthogeriatric co-management and fracture liaison services is strong [17,18,19,20], implementation remains inconsistent across health systems, particularly in the lower-resource, higher-projected-growth settings that

burden modeling identifies as facing the largest coming increase in hip fracture volume [1,2]. Closing that implementation gap, rather than generating further proof-of-concept evidence for the underlying interventions, may be the single highest-value next step for this field.

Sixth, reporting standards for BMD outcomes themselves are inconsistent across this literature in ways that make cross-study comparison harder than it should be. Some studies report BMD as an absolute density value, others as a T-score or Z-score, others as a percentage change from a stated baseline, and still others report only the categorical proportion of patients meeting a threshold for "improvement" or "stabilization" without disclosing the underlying continuous values at all, as in the real-world alendronate-versus-denosumab cohort discussed above [14]. A field this clinically important would benefit from a more standardized minimum reporting set for post-hip-fracture BMD studies, specifying measurement site, timing relative to surgery, the metric used, and, where feasible, both the continuous and threshold-based versions of the outcome, so that future systematic reviews and meta-analyses are not forced to discard otherwise-relevant studies purely because their outcome was reported in an incompatible format.

9. Conclusion

The literature synthesized here supports a reasonably specific set of conclusions about BMD recovery after osteoporotic hip fracture repair. Left to run its unmodified course, the post-fracture trajectory in this population tends toward continued bone loss, compounded by disuse, by the frailty and comorbidity that predisposed the patient to fracture in the first place, and, in a large fraction of patients, by previously unaddressed vitamin D and calcium insufficiency [15]. That trajectory is modifiable: antiresorptive therapy initiated promptly after hip fracture repair improves both fracture and mortality outcomes and produces a broadly consistent BMD response across patient subgroups [11,12], nutritional correction of the vitamin D and calcium deficits common in this population is independently associated with reduced hip fracture risk [15], and progressive resistance exercise produces a measurable, site-specific BMD gain at the hip even in older adults broadly [16]. None of these interventions functions in isolation from the others; the coupling

between bone and muscle captured by the osteosarcopenia framework means that nutritional, mechanical, and pharmacological interventions are best understood as complementary components of a single recovery process rather than competing treatment options [6,8].

What ultimately determines whether an individual patient experiences this evidence-based trajectory rather than the unmodified default decline is, overwhelmingly, whether the care system around them reliably initiates and sustains these interventions. Orthogeriatric co-management and fracture liaison services are the two organizational models with the clearest evidence of closing that gap [17,18,19,20], and guideline bodies have accordingly made structured secondary prevention, not merely the availability of effective drugs, the standard of care after hip fracture [4,5]. Given the scale of growth in hip fracture incidence projected over coming decades, concentrated disproportionately in regions with the least-developed secondary-prevention infrastructure [1,2], the central unresolved question for this field is no longer primarily biological. It is whether health systems, particularly those facing the steepest projected increases in caseload, can deliver the reasonably well-established package of pharmacological, nutritional, and mechanical interventions reviewed here to the patients who need them, in the narrow post-surgical window when they matter most.

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Conflict of Interest

The authors declare that they have no competing interests relevant to the content of this article.

Data Availability Statement

This article is a literature synthesis and did not generate new primary data. All findings discussed are drawn from the cited published sources, and readers should consult those original publications for their respective data and code availability statements.

Ethical Approval

Not applicable. As a literature synthesis, this work did not involve human participants, animal subjects, or the collection of primary clinical data, and therefore did not require ethical committee review.

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