Scholastic Orthopaedics (ISSN: 3108-1606)

Mini Review

Volume 2 Issue 4 April 2026

# Vitamin E and Its Reported Bone Tissue Protective Impacts: Efficacious or Not for Countering Frailty and its Outcomes?

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**Received:** June 22, 2026; **Published:** June 30, 2026

## Abstract

Bone, bone fractures and their causative and repair mechanisms, topics of great interest for many years, have not rendered any definitive conclusions over the years, especially in the realm of aging. When considering the role of functional foods as having a potential influence on bone health, this topic, especially the potential importance of vitamin E appears to have been relatively overlooked, despite its demonstrated role in bone genetics, resorption, and modeling processes. This mini review updates whether vitamin E in any form could prove of some clinical relevance in the realm of efforts to prevent or mitigate osteoporosis and thereby future fractures and fracture healing among aging at risk adults. Our extensive overview of the related literature that offers some favorable cumulative findings requires more resounding conclusive study, however.

**Keywords:** Bone; Bone Fractures, Fracture Healing, Osteoporosis, Prevention, Vitamin E

## Background

Bone, a highly specialized form of connective tissue, and one where predominant osteocyte (central regulators), osteoblast, and orthoclastic tissue control cells and signaling pathways commonly interact to provide an essential load-bearing and protective structure for the body is often the site of excess bone attrition. Of great concern here is the bone disease osteoporosis, a widespread disabling health condition often leading to frailty and one approaching epidemic levels among aging adults in all parts of the world. Also associated with the prolonged use of steroids or alcohol as well as sedentarity and sarcopenic muscle mass declines, osteoporosis significantly increases the risk of fracturing one or more bones, often in the face of sub-threshold, rather than severe trauma. Commonly inducing immense physical as well as profound negative psychosocial and economic outcome challenges, many years have been dedicated to exploring how to prevent or obviate this costly life disabling cycle.

Unfortunately, as of 2026 very few intervention approaches are deemed to safely prevent or delay its onset and related excess morbidity and premature disablement processes of osteoporosis, as well as its increased fracture risk. Although a possible clinical role for vitamin E, an important antioxidant found to stimulate trabecular bone formation has been investigated for some time in relation to bone turnover and metabolism [1, 2],and fracture occurrences [3], no definitive conclusion concerning its utility for mitigating bone attrition has been reached. This is despite a wealth of preclinical research studies showing nutrients such as vitamin E, a fat-soluble vitamin linked to immune health, DNA repair, and signal transduction [4] may impact bone favorably through its multiple systemic and local influences, as well as possible fracture risk and recovery via observable mechanisms of action [5-10].

However, despite a strong cumulative rationale for employing vitamin E as a possible functional food in efforts to foster bone health, and fracture healing [9-12], especially in light of its antioxidant, protective properties against illnesses linked to free radicals that could affect bone health detrimentally, this topic remains largely uncharted and theoretical rather than applied. This is despite a substantive body of evidence of its bone associated linkages, and that low serum concentrations of vitamin E have been associated with declines in physical function-a strong osteoporosis predictor- among older adults [12]. This may be because clinical evidence here is scarce and inconsistent [13], and largely focused on vitamin E benefits to the context of outcomes of arthroplasty surgery, rather than as a mitigation agent against bone attrition and fractures [14].

In this regard, we have dedicated considerable time over several years to uncover past as well as any novel insights that might be helpful both clinically, as well as in the context of future research to prevent bone fragility and its dire ramifications.

In our view, we strongly believe this topic is especially important to investigate because vitamin E found to be a strong anti-oxidative stress agent that affects the bone remodeling process [9, 15] is not synthesized by humans, and may need to depend upon external food or alternate sources, if this can be proven efficacious. It may provide an alternative to medications that are costly and often time bound and unsafe and save many lives when considering hip fracture outcomes alone. The topic is a further realm of concern given recent research showing more than 93% o f American adults may not meet the estimated average vitamin E requirement of 12 mg/day [16] despite its apparent positive effects on bone biology, maintenance, healing, repair, and regenerative processes [17-19] and research showing a possible linear association between dietary vitamin E levels and osteoporosis in a study of the older population in the United States [20].

According to Wei et al. [21] dietary supplementation of vitamin E where desired may serve as an important strategy for enhancing bone health in women, the most affected group, after menopause.

## Aims

Our prime aim was to review data pertaining to the associations between bone health and vitamin E as of mid June 2026. The key question driving the review was whether bone status can be mediated or moderated favorably in some way by vitamin E, for example by adherence to a diet containing the desired level of vitamin E nutrients, and/or rendering key vitamin E containing foods available to those at risk for bone disease and fragility fractures, as well as those who have sustained one or more fractures. On the other hand, if vitamin E is deemed to be harmful to bone, efforts to reduce or curtail vitamin E intake would appear reasonable to acknowledge. A second aim was to examine if further research in this realm would be desirable and if so in what regard.

## Clinical relevance

Osteoporosis, or bone loss, is a disease that affects many women as well as increasingly, men, both in the United States and globally as life expectancy increases. If untended, osteoporosis usually progresses and may engender a state of bone fragility also expected to reach epidemic levels by 2050 that will thereby create great suffering as well as an immense burden on the healthcare and economic sectors of all countries where older adults numbers and high ages are rising rapidly [22]. Yet, among counter strategies that could prove impactful, is the mitigation of sarcopenia, an age associated decline in muscle mass that may have a bearing on bone metabolism and protection and is a remediable factor that can be partially eliminated or attenuated to some degree in the presence of adequate dietary vitamin E [23].

## Methods

To address these aims, related documents published predominantly over the past six years i.e. January 2020-June 2026 were specifically sought, alongside data extending retrospectively to 2010. The search terms applied were: *Vitamin E and Bone Health*, *Tocopherol, and Osteoporosis*, *Tocotrienol and Osteoporosis*, the latter terms referring to the two key structurally related sub groups of vitamin E. Databases explored were: **PUMED, Google Scholar**, **PubMed Central**, **Science Direct, Embase and Scopus.** Studies that examined any form of vitamin E in the context of bone status were deemed acceptable, as were systematic reviews of related topics. No abstracts or foreign non-English articles or studies examining mechanisms of action or cellular based studies or the application of vitamin E post joint replacement surgery were specifically sought. Rather, this overview sought to focus on salient research studies deemed relevant to the current aims in the views of the author. No restrictions were placed on the nature of the documented research designs, provided they addressed the major themes of present interest. However, the material reported is not assessed quantitatively or necessarily represent the topic in its totality, which is largely constituted by preclinical data and almost no well designed clinical study.

After selecting the most relevant articles as per the viewpoint of the author, the downloaded references were read carefully, and their findings pertaining to this current line of inquiry were highlighted. Although limited to examining the degree to which vitamin E impacts bone, rather than its mechanistic explanations, we feel this brief introduces the topic as one of promise, but where the issues and conclusions that prevail remain clinically salient but non-conclusive.

For consistency, because vitamin E is a complex biological compound, the terms used herein to describe the vitamin E substrate or isomer being discussed in the research is those reported by the authors, even though these are not necessarily strictly comparable across studies or optimal. While some studies may have been overlooked, it is believed this ensuing review is inclusive of almost all, if not all, currently relevant studies and reviews on the topic of vitamin E and bone health. The topic premise that vitamin E may be a protective bone agent is supported by understandings that stem from a large multi decade long data base of studies that show vitamin E encompasses a family of lipid-soluble compounds that function primarily as antioxidants that scavenge free radicals and modulate inflammation in pathways implicated in skeletal integrity and bone remodeling. Preclinical studies in particular have demonstrated that vitamin E supplementation can preserve trabecular bone tissue connectivity, increase markers of bone formation and reduce degrading orthoclastic activity (https://www.nature.com/nature-index/topics/l4/vitamin-e-effects-on-bone-health-and-mineral-density). For a summary of prior studies readers are referred to the excellent reviews cited in references 5, 19, 21, 22, 24, 25.

## Key Observations

### *Preclinical Studies*

As a snapshot of the many diverse, albeit relevant results obtained from animal-based studies conducted to examine bone-vitamin E associations, Box 1 depicts some of these largely favorable conclusions of a high number of these studies. However, this is challenging to aggregate, and cannot conclusive in its translation to the bedside, as a small number of clinical studies, strongly contradict the possible bone building, anti osteoporotic, or bone remodeling potential of vitamin E [eg., 26].

Nonetheless, the majority of the preclinical data do tend to stem from well-designed and carefully controlled observations where almost all indicate vitamin E is of potential utility in efforts to minimize bone loss due to aging or extrinsically induced osteoporosis in multiple respects, as well as in instances of fracture healing [21] even though the data in this respect vary in terms of type of vitamin E compound studied, method of assay and delivery, the nature of the bone related correlates examined, the animals examined, and their varying ages, and method of producing osteoporosis. Most fail to examine male generated or impacted samples.

Moreover, even though definitive cellular effects imply a specific role of either tocopherols or tocotrienols that represent one of the two subgroups of vitamin E, the clinical translation of this as regards bone loss. maintenance, and healing is uncertain and clearly understudied,

even though within the laboratory, the favorable effects tend to be generalizable and apply to all four chemically distinct isoforms of vitamin E [27], namely, alpha, beta, gamma, and delta isoforms even if they differ in terms of comparable levels of bioavailability. Other salient observations are summarized below.

**Box 1:** Selected Findings From Preclinical Studies that Largely Favor a Role for Vitamin E of Various Forms in Fostering Bone Health and Bone Healing and Allaying Bone Attrition.

- May prevent imbalances in bone remodeling [28]
- Orchestrates and enhances skeletal integrity [1, 2, 29]
- Can improve fracture healing [30-32]
- Low dose tocotrienols protect osteoblasts from oxidative stress [33]
- May have beneficial effects on bone health [34]
- May foster bone calcium homeostatis [35]
- May stimulate new bone formation [36]
- May reduce the risk of male osteoporosis [37]
- May prevent late life osteoporosis [30, 38]
- Palm tocotrienol is more effective than calcium in preventing bone loss [39]
- Positively affects early and late-phase fracture healing [31, 40]
- Appears to prevents postmenopausal osteoporosis [41]
- May foster osteocyte membrane repair and survival [42]
- Prevents bone loss [43]
- Portrays potential skeletal promoting benefits [43, 44]

In addition to the above noteworthy findings, it appears tocotrienal isomers applied to osteoporotic bone has the ability to foster the preservation of its biomechanical strength and structure, as well as helping to maintain its vital inherent bone gene expression [11]. Accordingly, its anabolic as well as anti-resorptive properties of vitamin E are said to have the potential to be quite successfully utilized as a prophylactic for individuals receiving long-term glucocorticoid therapy that commonly diminishes bone density, as well as for other vulnerable older adult groups. Moreover, their ability to regulate bone genetics and preserve the biomechanical strength and structure of bone may help avert fractures and stimulate bone repair due to its osteocyte-mediated bone remodeling impacts [45-47], even in the case of alcohol-induced bone damage or age or iatrogenic associated disrupted osteocyte damage [10].

In this regard, used in other spheres and alongside other active bone modulators, vitamin E combinations may offer protection against ovariectomy-induced bone changes, as well as those observed in the presence of impaired glucose homeostasis and obesity, possibly indicating an associated ability to suppress oxidation stresses and inflammation determinants of osteoporotic bone damage [19, 49, 50]. Its fracture healing promise is also increasingly evident and attributed to the powerful immune-modulatory functions of vitamin E [19].

### *Clinically Related Observations*

Despite a fair number of preclinical animal related studies in the context efforts to understand the role of vitamin E in a variety bone health contexts as depicted above, as well as a strong imperative for doing this, this updated search of the literature revealed very few contemporary clinically oriented studies focusing on the implications of vitamin E as related to either osteoporosis prevention, or fracture healing, and prevention. Moreover, the study findings are hard to accept at face value, because unlike animal studies that can control for confounding factors and bone status, and can examine the trajectory of osteoporosis from its inception, many factors including health status, gender, age, and health behaviors, among others that impact bone health are much more challenging to control or discern in the human context. As well, vitamin E dosage delivery and outcome measures employed in animal studies may be much more challenging to pursue in humans, especially considering the variations of body size, and prevailing bone health that may exist. Follow up is also much more susceptible to attrition or the effects of history and other factors in the human condition.

Among recent studies, however, that by Wong et al. [44] who examined vitamin E bone interactions, implied tocotrienols, found in certain foods, do appear to have the potential to produce skeletal-promoting bone benefits, and do this by modulating the levels of osteocytes-derived bone-related peptides.

In a further study [45] of 52 osteopenic postmenopausal women enrolled and allocated into two groups, and where the intervention group received mixed-tocopherol 400 IU/day, while the control group received placebo tablets, it was concluded vitamin E (mixed-tocopherol) supplementation in postmenopausal osteopenic women may have a preventive effect on bone loss through its anti-resorptive activity properties.

This supported the view of the other researchers who have found vitamin E to be a strong anti-oxidative stress agent that affects the bone remodeling process favorably in most cases [eg., 50-53].

In addition, one further group found delta-tocotrienol independently enhanced bone formation, while helping to maintain bone strength in ovariectomised rats, as well as significantly improving trabecular bone structure when combined with statins, thus implying possible usage for countering postmenopausal osteoporosis [54]. A study by Magremanne et al. [55] further showed the possible dual usage of tocopherol alongside Pentoxifylline improved lesions related to osteoradionecrosis, which may be valuable to other patients who are at risk for osteonecrosis due to chemotherapy or corticosteroid usage.

In sum, preliminary studies as per below, suggest that at a minimum tocotrienal sources of vitamin E have the ability to exert protective and anabolic bone effects by influencing the activity of bone osteocytes, including shielding them from oxidative damage. *In vivo* models using ovariectomised or metabolic syndrome rats demonstrate its supplementation has the potential to modulate key osteocyte-secreted factors, including sclerostin, dentin matrix protein 1, Dickkopf-related protein 1, fibroblast growth factor 23, and receptor activator of nuclear factor κB ligand. However, the current evidence here remains limited by the use of models that may not fully represent the clinical picture of degenerative osteoporosis, and relies on restricted dose-dependent studies [10].

**Table 1:** Sample of clinically based studies examining vitamin E and bone categorized by study design and indicating a positive role for Vitamin E in bone health contexts as per data published between 2014-2026.

| STUDY MODE + RESEARCHERS | KEY FINDINGS |
|---|---|
| ***Cross-sectional studies*** | |
| Shi et al. [54] | Bone mineral density in women is related to greater consumption + serum vitamin E levels |
| Michaelson et al. [61] | Low vitamin E intake plus serum levels were associated with an increased fracture rate in elderly persons |
| Pasco et al. [62] | Vitamin E may suppress bone resorption in nonsmoking postmenopausal women |
| ***Case-control studies*** | |
| Holvik et al. [60] | Low vitamin E levels were associated with increased hip fracture risk |
| Mata-Granados et al. [63] | Lower vitamin E serum levels and osteoporosis are related |
| Sun et al. [64] | Higher dietary vitamins E intake is associated with other factors + lowers risk of hip fracture |
| Torbergsen et al. [3] | Low vitamin E concentrations increased hip fracture risk |
| ***Randomized prospective studies*** | |
| Chuin et al. [17] | Vitamin E may contribute to bone loss protection |
| D'Adamo et al. [56] | High functioning hip fracture cases had higher vitamin E concentrations |
| Shen et al. [55] | 12-weeks of tocotrienol supplementation favored bone remodeling in osteopenic women |
| Vallibhakara et al. [14] | Vitamin E mixed-tocopherol supplementation applied to in postmenopausal osteopenic women may have a preventive effect on bone loss |

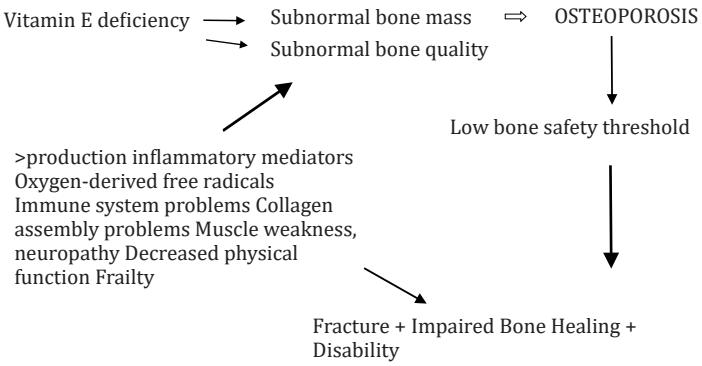
Indeed, as a whole and as outlined in the above reports it is evident that in the presence of desirable serum concentrations of both α- and γ-tocopherol one can expect better physical outcomes than not, for example after a hip fracture. This may be due to its anti inflammatory as well as its immune modifying properties [56, 57] and clearly represents a potentially modifiable factor related to the achievement of optimal recovery post fracture, regardless of its serum levels at baseline.

We agree as well with Chin et al. [25] that tocotrinal vitamin E sources of palm oil and annatto bean origin has immense potential as well as demonstrated efficacy in improving skeletal and joint health in numerous conditions that lead to bone loss and that may be applicable to analogous results achieved by costly pharmaceutical interventions to counter osteoporosis in humans. Mechanistic studies in particular suggest the above studies in Table 1 results are not spurious but that these reflect vitamin E attributes that can be shown to exert their bone mediating effects through its antioxidant, anti-inflammatory, Wnt-suppressive, and mevalonate-bone cellular modulating mechanisms. Vitamin E also shows promise in terms of fostering bone self-repair mechanisms, but is not well studied when compared to the wealth of recent studies favoring vitamin E as a parallel beneficial strategy post joint replacement surgery. However, human clinical trials in this field appear highly desirable given the potent bone restorative properties of vitamin E that may yet await discovery and refinement [58] and validation in favor of this possible adjunctive osteoporosis mitigation and treatment option as outlined by several others [eg., 3, 54, 59, 61-64].

Indeed, the potential negative impact of vitamin E insufficiency on bone status is increasingly reported, and the research largely implies sufficient vitamin E levels are more likely than not to promote bone growth, and enhance bone metabolism. It can also produce better static and dynamic histomorphometric bone parameters, while reducing bone resorption [1] and fracture risk independent of bone mineral density [12, 15, 18, 19].

Its observed bone building efficacy is reportedly attainable regardless of level of intake dosage, a factor often cited in the clinical literature against vitamin E supplementation, but an attribute that may allow more aging adults to avoid bone attrition and frailty. Vitamin E also has proven health benefits other than those apparently supporting bone health directly, such as its ability to positively influence aging muscle contractile force and to possibly help avert falls and fracture risk in steroid users and others [4], and even in the presence of chronic osteoporosis, or other conditions such as metabolic syndrome and androgen deprivation therapy [41, 48, 69]. It may also help build bone from an early age [1], while preserving bone in instances when skeletal unloading is indicated [70].

Research further shows vitamin E is capable of averting negative hormonal states, while beneficially influencing the cardiovascular system, in addition to aiding in the treatment of age-related osteoporosis and fracture healing and recovery. Preventing vitamin E deficiency may also foster rather than impair bone calcium status, a crucial bone-mediating factor [23, 29, 30, 35, 39] as per Figure 1 below.



However, since a number of researchers have reported either a null effect or a noxious effect of vitamin E on bone status, and very few randomized controlled intervention studies prevail, and the preclinical support which is largely solid and of great promise may not equate with the clinical manifestations of bone loss in the elderly, we urge researchers to consider this understudied realm of possible high clinical relevance, sooner rather than later.

## Discussion

Over the years, ample research has demonstrated that bone in its various forms requires a variety of nutrients throughout life in order to develop and assume an array of optimal structural and functional attributes. Conversely, bone density losses leading to an increased fracture and disability risk among many older individuals may be significantly affected by a variety of nutritional factors, such as vitamin E as outlined above. Based on current research to examine the validity of this idea, this review not only noted insufficient numbers of well-designed clinical studies in this regard, but some divergent conclusions that clearly preclude any definitive implications for clinical practice at the present time. That is, despite a fairly robust health associated role for vitamin E based on laboratory study, and that vitamin E is the body's most important antioxidant, and one found to mediate a variety of anti-oxidative processes that could potentially affect bone status, the question of whether suboptimal vitamin E intake levels or availability or uptake or all of these factors that has a bearing on human bone health, including the onset and progression of osteoporosis, and related fracture risk and fracture recovery remains inconclusive. Indeed, even though several reasonably favorable investigative reports on this topic have been generated over time in a sizeable number of animal models [eg 1], no specific report to date has yielded any irrefutable evidence for regarding vitamin E intake as a crucial anti-osteoporotic and/or bone health moderating agent despite a strong case for its possible role in light of its anti-oxidative, immune-modulatory and anti-inflammatory actions against free radical damage [1]. To support these claims it appears more supportive observations which emanate from osteoporotic models that closely emulate the human condition more authentically than the rat model widely used are indicated. In addition, more definitive isomer studies, and dose dependent analyses, baseline vitamin E serum levels and bone status measures that are more uniform, more carefully construed and designed and controlled for are clearly indicated.

Carefully selected qualitative and quantitative bone marker variables that can capture bones' static as well as its dynamic parameters, including bone thickness, will similarly be very helpful in broadening and solidifying the relevance of vitamin E as a potentially highly effective bone health mediator or moderator. Moreover, to minimize bias and achieve more dependable conclusive study results, the role of a variety of health factors, health behaviors, and medications appears to warrant more specific attention. Controlling for additional bone mediating factors such as gender, vitamin E dosage-associations, hormonal, and smoking status, plus age is also strongly indicated.

Confounding variables in the meantime in clinical studies appear to include a failure to account for other key bone, medication usage in supplement or serum based measurement oriented studies, and other related health mediating factors, such as steroid exposure. Moreover, those that prevail are diverse in design and sampling, do not always produce conclusions that concur, do not necessarily examine similar samples under similar conditions, fail to exclude smokers, alcohol dependent persons or bone attrition inducing hormones, and to control for supplement usage. Most fail as well to include sufficient numbers of subjects selected at random, while vague or dissimilar inclusion criteria produce heterogeneous rather than well defined samples that cannot be aggregated readily. Age and gender attributes and health sub groupings, alongside dosages and modes of vitamin E administration as well as differing bone status are additional validation challenges. As well, the influences of lifestyle, comorbid pain status, inflammation, kinesiophobia, and genetic factors, plus the use of subjectively reported and aggregated dietary assessment data, unknown blinding factors, plus the limited numbers of clinical studies that include men are some obvious additional challenges to deriving sound clinical recommendations as regards the salience of vitamin E as a bone status mediator or moderator in humans at risk for excess bone resorption or in those who have suffered a fracture. In other cases, the independent effects of vitamin E isomers on bone mineral density may depend on the bone site studied [75, 76].

In this regard, it may be necessary to conceptualize a new approach to examining this issue, since most prevailing laboratory as well as clinical studies may not replicate the 'real world' situation quite accurately. To this end, it appears the specific influences of vitamin E and its isomers on cortical, versus cancellous or trabecular bone needs to be examined more comprehensively, as does the possible utility of micronutrient sources of vitamin E delivery post hip fracture surgery on outcomes, and bone effects in cases who experience bone building medication side effects.

In the interim, as per Mulligan et al. [53] dietary vitamin E intake may play a vital role in explaining musculoskeletal health differences among older and younger adults, as well as men and women, and can be achieved by eating a varied and balanced diet, including the consumption of foods rich in vitamin E, such as oily seeds and their derivatives, nuts and cereals rich in vitamin E, including fortified breakfast cereals. The presence of vitamin E sufficiency may also improve overall health and multiple musculoskeletal correlates that decline with age particularly muscle mass, as well as mitigating inflammation and oxidative stresses that underlie many musculoskeletal disorders and more focus here is highly recommended [eg., 51, 57].

On the contrary, without further study, health providers may continue to face uncertainty in this sphere, especially given the unknown interaction effects of caffeine use on bone in the absence of vitamin E, calcium related impacts, and food quality mechanisms that can serve to activate or inhibit vitamin E bone mass metabolic effects. Moreover, the oftentimes concomitant lack of blinded baseline or follow up alongside reliable serum vitamin E assays, and bone tomographic and radiologic measures, the lack of attention to temporal, historic or maturation factors and others that may affect vitamin E bioavailability, are additional confounders.

To overcome the limitations listed above, more thorough prospective study of large diverse high age populations are strongly recommended in light of the degree to which failure to do this can predictably fail to avert a high degree of undue suffering and health costs by the elderly.

Positively influencing aging muscle strength and frailty risk through vitamin E based micronutrients that might well have a marked impact on bone metabolism, as well as in cases of steroid induced osteoporosis, metabolic syndrome [29, 44], nicotine addiction, and post operative hip fracture surgery bone losses [51] should be specifically explored. Vitamin E based diets may also help build bone from an early age [1],thus helping to preserve bone in later life as well as in instances when aging adults are deemed osteosarcopenic, or where skeletal unloading is indicated [5, 70, 73].

## Key Conclusions

Notwithstanding the limited number as well as quality of clinical studies that focus on vitamin E and bone in the context of osteoporosis and bone fragility prevention and intervention, plus fracture healing, along with their oftentimes contradictory findings, we conclude:

1. A wide array of health promoting factors including those that influence bone are seemingly strongly influenced biologically by one or more vitamin E analogues.
2. Despite several studies to the contrary in both the laboratory as well as the clinical realms, the recent findings seem to provide an especially promising ray of progress in solidifying the value of further pursuing this area of clinical practice at either a primary or a secondary prevention level.
3. Ample data reveal tocotrienol supplementation may clearly suppress bone resorption and oxidative stress levels and may well orchestrate many fundamental bone building mechanisms at the molecular and cellular level that can account for its observed important impacts on bone status [70- 75].
4. Rather than relying on anti-osteoporotic pharmaceutical drugs, vitamin E in various forms may help prevent postmenopausal osteoporosis, as well nicotine-induced osteoporosis, hip fracture occurrences and excess disability outcomes.
5. Its potential in allaying suffering in aging adults at high risk for osteoporosis appears highly promising and crucial to pursue.

Indeed we further conclude carefully examining related issues mentioned above, plus how vitamin E analogues are absorbed, transported, taken up and depleted to impact bone status outcomes, is likely to prove of high benefit to many.

## Final Thoughts

Examining the role of dietary factors such as vitamin E in the context of bone health may offer new insights as regards opportunities to avert or minimize the considerable undue suffering and related costs associated with osteoporosis, especially among those adults receiving steroids, those with malabsorption conditions, plus smokers and alcohol dependent persons and those unable to afford or access or process vitamin E containing foods. To arrive at more generalizable conclusions, and clinical recommendations, the value of efforts to routinely measure vitamin E serum levels in all adults, as well as educating the general public, as well as the at risk population regarding the importance of consuming a consistent intake of vitamin E containing foods, along with the dangers of excess vitamin E intake, including the use of supplements should be examined. Studies that examine the role of natural vitamin E sources versus synthetic vitamin E products, plus the ensuing anti-osteoporotic and fracture healing outcomes associated with vitamin E in conjunction with other agents, as well as the unique role of tocotrienols on bone biomechanics, structure, and inflammatory pathways are also strongly indicated to strengthen the established evidence base.

## References

1. Mehat MZ, Squid AN, et al; (2010 "Beneficial effects of vitamin E isomer supplementation on static and dynamic bone histomorphometry parameters in normal male rats". *J Bone Min and Metab*;28(5): pp503-509.
2. Xu H, Watkins BA, Seifert MF et al: (1995) "Vitamin E stimulates trabecular bone formation and alters epiphyseal cartilage morphometry". *Calcific Tissue Int*;57(4): pp293-300.
3. Torbergsen AC et al; (2017) "Micronutrients and the risk of hip fracture: case-control study". Clin Nutr;36(2): pp438-443.
4. Rondanelli M, Faliva MA, et al; (2015) "Focus on pivotal role of dietary intake (diet and supplement) and blood levels of tocopherols and tocotrienols in obtaining successful aging". *Int J Molecular Sci*;16(10): pp23227-23249.
5. Li H, Cao W, et al; (2025) "The role of fat-soluble vitamins on bone metabolism and osteoporosis: a literature review". *Ann Med*;57(1): pp2533429.
6. Brooks R, Kalia P, et al; (2011) "Direct inhibition of osteoclast formation and activity by the vitamin E isomer gamma-tocotrienol". *Int J Vitamin Nutr Res*;81(6): pp358-367.
7. Wong SK, Mohamad NV et al; (2019) "The molecular mechanism of vitamin E as a bone-protecting agent: a review on current evidence". *Int J Molecular Sci*;22; 20(6). pp E1453.
8. Ibrahim N', et al; (2015) "The effects of targeted deliveries of lovastatin and tocotrienol on ossification-related gene expressions in fracture healing in an osteoporosis rat model". *Int J Environ Res Publ Hlth*;12(10): pp12958-76.
9. Shahriarpour Z, Nasrabadi B, et al; (2021) "Oxidative balance score and risk of osteoporosis among postmenopausal Iranian women". *Arch Osteoporos*;16(1): pp43.
10. Zahanordin NN, Ng PY, Chin KY et al: (2026) "Modulation of tocotrienol's bone effects by osteocytes: a perspective". *Front Pharmacol*; 17: pp1773544.
11. Ramli ESM, Ahmad F,et al; (2025) "Comparative bone-protective effects of tocotrienol isomers from palm and annatto in dexamethasone-induced osteoporotic male rats". *Int J Mol Sci*;26(20): pp10206.
12. D'Adamo CR, et al; (2011) "Serum vitamin E concentrations among highly functioning hip fracture patients are higher than in nonfracture controls". *Nutr Res*;31(3): pp205-14.
13. Chin KY, Ima-Nirwana S. et al; (2014) "The effects of α-tocopherol on bone: a double-edged sword?". *Nutrients*;6(4): pp1424-1441.
14. Sköldenberg O, Mukka S, Magneli M. et al; (2024) "6-year follow-up on migration outcomes: a randomised clinical trial of cemented vitamin E-stabilised highly crosslinked versus standard polyethylene cup in total hip arthroplasty". *Hip Int*;34(6): pp695-700.
15. Vallibhakara SA, et al; (2021) "Effect of vitamin E supplement on bone turnover markers in postmenopausal osteopenic women: A double-blind, randomized, placebo-controlled trial". *Nutrients*;13(12): pp4226.
16. Fulgoni VL, et al; (2011) "Foods, fortificants, and supplements: where do Americans get their nutrients?" *J Nutr*;141(10): pp1847-1854.
17. Chuin A, Labonté M, et al; (2009) "Effect of antioxidants combined to resistance training on BMD in elderly women: a pilot study". *Osteoporos Int*. Jul;20(7): pp1253-1258.
18. Abukhadir SS, et al; (2012) "Effects of palm vitamin e on bone-formation-related gene expression in nicotine-treated rats". *Evid Based Complement Alternat Med*; 2012: pp656025.
19. Viswanathan VK, et al; (2025) "Evaluation of vitamin E in trauma and orthopaedics: a narrative synthesis". *Indian J Orthop*;59(9): pp1364-1380.

20. Zhuang R, Hou W, et al; (2024) "Association between dietary vitamin E and osteoporosis in older adults in the United States". *Front Endocrinol (Lausanne)*;15: pp1410581.

21. Wei Y, Lei C, et al; (2025) "Effects of dietary supplements on bone turnover markers in women after menopause: a network meta-analysis". *PeerJ*;13: pp19882.

22. Liang G, Kow ASF, et al; (2022) "Ameliorative effect of tocotrienols on perimenopausal-associated osteoporosis-a review". *Antioxidants (Basel)*;11(11): pp2179.

23. Otsuka Y, Iidaka T, et al; (2021) "Dietary intake of vitamin e and fats associated with sarcopenia in community-dwelling older Japanese people: a cross-sectional study from the fifth survey of the ROAD Study". *Nutrients*;13(5): pp1730.

24. Guralp O. et al; (2014) "Effects of vitamin E on bone remodeling in perimenopausal women: mini review". *Maturitas*;79(4): pp476-80.

25. Chin KY, Ima-Nirwana S. et al: (2019) "The role of tocotrienol in preventing male osteoporosis-a review of current evidence". *Int J Mol Sci*;20(6). ppE1355.

26. Zhang J, Hu X, Zhang J. et al; (2017) "Associations between serum vitamin E concentration and bone mineral density in the US elderly population". *Osteoporos Int*;28(4): pp1245-1253.

27. Nicoll R. et al: (2014) "Vitamin E and bone health: not just alpha-tocopherol".*Mediterranean J Nutrit Metab*;7: pp87-93.

28. Chin KY, et al; (2014) "Annatto tocotrienol improves indices of bone static histomorphometry in osteoporosis due to testosterone deficiency in rats". *Nutrients*;6(11): pp4974-4983.

29. Wong SK, et al; (2019) "The effects of tocotrienol on bone peptides in a rat model of osteoporosis induced by metabolic syndrome: the possible communication between bone cells". *Int J Environ Res Publ Hlth*;16(18). pp E3313.

30. Shuid AN, Mehat Z, et al; (2010) "Vitamin E exhibits bone anabolic actions in normal male rats". *J Bone Miner Metab*;28(2): pp149-56.

31. Durak K, et al; (2003) "Histological assessment of the effect of alpha-tocopherol on fracture healing in rabbits". *J Int Med Res*;31(1): pp26-30.

32. Paskalev MD, Goranov NV, et al; (2011) "Antioxidant and bone healing effect of vitamin E in an experimental osteotomy model in dogs". *Comparative Clin Pathol*;20(4): pp403–408

33. Nizar AM, et al; (2011) "Low dose of tocotrienols protects osteoblasts against oxidative stress". *Clin Ter*;162(6): pp533-538.

34. Kasai S, Ito A, et al; (2015) "High-dose α-tocopherol supplementation does not induce bone loss in normal rats". *PLoS One*;10(7): pp0132059.

35. Norazlina M, Chua CW, et al; (2004) "Vitamin E deficiency reduced lumbar bone calcium content in female rats". *Med J Malaysia*;59(5): pp623-630.

36. Akçay H, et al; (2019) "Vitamin E promotes bone formation in a distraction osteogenesis model". *J Craniofac Surg*. Jun 20.

37. Casati L, Pagani F et al; "Beneficial effects of δ-tocotrienol against oxidative stress in osteoblastic cells: studies on the mechanisms of action". *Eur J Nutr*. Jul 6.

38. Abdul-Majeed S, Mohamed N, Soelaiman IN et al: (2015) "The use of delta-tocotrienol and lovastatin for anti-osteoporotic therapy". *Life Sci*;125: pp42-48.

39. Suhaimi F, Muhammad N, Soelaiman IN et al: (2012) "The effects of alpha-tocopherol supplementation on fracture healing in a postmenopausal osteoporotic rat model". *Clinics (Sao Paulo)*;67(9): pp1077-1085.

40. Turk C, Halici M, et al; (2004) "Promotion of fracture healing by vitamin E in rats". *J Int Med Res*;32(5): pp507-512.

41. Mohamad S, Shuid AN, et al: (2012) "The effects of alpha-tocopherol supplementation on fracture healing in a postmenopausal osteoporotic rat model". *Clinics (Sao Paulo)*;67(9): pp1077-1085.

42. Hagan ML, Bahraini A, et al; (2019) "Inhibition of osteocyte membrane repair activity via dietary vitamin E deprivation impairs osteocyte survival". *Calcific Tissue Int*;104(2): pp224-234.

43. Wong SK, Chin KY, et al; (2018) "The effects of vitamin E from elaeis guineensis (oil palm) in a rat model of bone loss due to metabolic syndrome". *Int J Environ Res Publ Hlth*;15(9). Pp E1828.

44. Wong SK, et al; (2018) "Exploring the potential of tocotrienol from Bixa orellana as a single agent targeting metabolic syndrome and bone loss". *Bone*;116: pp8-21.

45. Vakili S, Zal F, et al; (2021) "Quercetin and vitamin E alleviate ovariectomy-induced osteoporosis by modulating autophagy and apoptosis in rat bone cells". *J Cell Physiol*;236(5): pp3495-3509.

46. Ekeuku SO, et al; (2025) "Palm tocotrienol preserves trabecular osteocyte indices and modulates the expression of osteocyte markers in ovariectomized rats". *Biomedicines*;13(5): pp1220.

47. Abdullah Sani N, Kamaruddin NA, et al; (2025) "Palm tocotrienol activates the Wnt3a/β-Catenin signaling pathway, protecting MC3T3-E1 osteoblasts from cellular damage caused by dexamethasone and promoting bone formation". *Biomedicines*;13(1): pp243.

48. Mohamed N, Ahmed Abukhadir SS, et al; (2024) "Vitamin E improves cellular and structural bone histomorphometry in an alcohol-induced osteoporosis rat model". *Pharmaceuticals (Basel)*;17(12): pp1730.

49. Ibrahim N', Mohd Noor H' et al; (2021) "Osteoprotective Effects in Postmenopausal osteoporosis rat model: oral tocotrienol vs. intraosseous injection of tocotrienol-poly lactic-co-glycolic acid combination". *Front Pharmacol*;12: pp706747.

50. Shen CL, Wankhade UD, et al; (2024) "Effects of statin and annatto-extracted tocotrienol supplementation on glucose homeostasis, bone microstructure, and gut microbiota composition in obese mice". *In Vivo*;38(4): pp1557-1570.

51. Melhus H, Michaëlsson K, et al; (1999) "Smoking, antioxidant vitamins, and the risk of hip fracture". *J Bone Miner Res*;14(1): pp129-35.

52. Peng YL, Wang ZY, et al; "Lower risk of low bone mineral density in high vitamin E level in older people: a cross-sectional study". *Clin Nutr ESPEN*;61: pp316-21.

53. Mulligan AA, Hayhoe RP, et al; (2021) "Positive associations of dietary intake and plasma concentrations of vitamin E with skeletal muscle mass, heel bone ultrasound attenuation and fracture risk in the EPIC-Norfolk cohort". *Antioxidants*;10(2): pp159.

54. Shi WQ, et al; (2016) "Association of dietary and serum vitamin E with bone mineral density in middle-aged and elderly Chinese adults: a cross-sectional study". *Br J Nutr*;115(1): pp113-20.

55. Magremanne M, Reychler H. et al; (2014) "Pentoxifylline and tocopherol in the treatment of yearly zoledronic acid-related osteonecrosis of the jaw in a corticosteroid-induced osteoporosis". *J Oral Maxillofac Surg*;72(2): pp334-7.

56. D'Adamo CR, Miller RR, et al; (2011) "Serum vitamin E concentrations and recovery of physical function during the year after hip fracture". J Gerontol A Biol Sci Med Sci;66(7): pp784-93.

57. D'Adamo CR, Miller RR, et al; (2012) "Higher serum concentrations of dietary antioxidants are associated with lower levels of inflammatory biomarkers during the year after hip fracture". *Clin Nutr*;31(5): pp659-665.

58. de Morais RPL, de Aguiar AWPB, et al; (2024) "Is the use of Pentoxifylline and Tocopherol effective in the treatment of Osteoradionecrosis of the jaws or for the treatment of medicationosteonecrosis of the jaw? An overview". *J Stomatol Oral Maxillofac Surg*;125(12 Suppl 2): pp101959.

59. Shen CL, Klein A, et al; (2017) "Tocotrienols for bone health: a translational approach". *Ann N Y Acad Sci*;1401(1): pp150-165.

60. Holvik K, Gjesdal CG et al; (2014) "Low serum concentrations of alpha-tocopherol are associated with increased risk of hip fracture. A NOREPOS study". *Osteoporos Int*;25(11): pp2545-2554.

61. Michaëlsson K, Wolk A, et al; (2014) "Intake and serum concentrations of α-tocopherol in relation to fractures in elderly women and men: 2 cohort studies". *Am J Clin Nutr*;99(1): pp107-14.

62. Pasco JA, Henry MJ, et al; (2006) "Antioxidant vitamin supplements and mrkers of bone turnover in a community sample of nonsmoking women". *J Womens Health* (Larchmt);15(3): pp295-300.

63. Mata-Granados JM, Cuenca-Acebedo R, et al; (2013) "Lower vitamin E serum levels are associated with osteoporosis in early postmenopausal women: a cross-sectional study". *J Bone Miner Metab*;31(4): pp455-60.

64. Sun LL, Li BL, et al; (2014) "Associations between the dietary intake of antioxidant nutrients and the risk of hip fracture in elderly Chinese: a case-control study". *Br J Nutr*;112(10): pp1706-1714.

65. Turan B, Balcik C, Akkas N et al: (1997). "Effect of dietary selenium and vitamin E on the biomechanical properties of rabbit bones". *Clin Rheumatol*;16(5): pp441-9.

66. Norazlina M, Chua CW, Ima-Nirwana S. et al; (2004) "Vitamin E deficiency reduced lumbar bone calcium content in female rats". *Med J Malaysia*; 59(5): pp623-630.

67. Saud Gany SL, Chin KY, Tan JK et al; (2023), "Aminuddin A, Makpol S. Preventative and therapeutic potential of tocotrienols on musculoskeletal diseases in ageing". *Front Pharmacol*;14: pp1290721.

68. Ikegami H, Kawawa R, Ichi I, Ishikawa T, Koike T, et al; (2017) "Excessive vitamin E intake does not cause bone loss in male or ovariectomized female mice fed normal or high-fat diets". *J Nutr*;147(10): pp1932-1937.

69. Mohamad NV, Soelaiman IN, Chin KY. Et al; (2018) "Effects of tocotrienol from Bixa orellana (annatto) on bone histomorphometry in a male osteoporosis model induced by buserelin". *Biomed Pharmacother*;103: pp453-462.

70. Smith BJ, Lucas EA, et al; (2005) "Vitamin E provides protection for bone in mature hindlimb unloaded male rats". *Calcif Tissue Int*;76(4): pp272-279.

71. Norazlina M, Ima-Nirwana S, et al; (2002) "Tocotrienols are needed for normal bone calcification in growing female rats". *Asia Pac J Clin Nutr*;11(3): pp194-199.

72. Shen CL, Klein A, Chin KY, Mo H, Tsai P, et al: (2017) "Tocotrienols for bone health: a translational approach". *Ann N Y Acad Sci*;1401(1): pp150-165.

73. Ahmadieh H, Arabi A. et al; (2011) "Vitamins and bone health: beyond calcium and vitamin D". *Nutr Rev*. Oct;69(10): pp584-598.

74. Wan Hasan WN, et al; (2018) "Annatto-derived tocotrienol stimulates osteogenic activity in preosteoblastic MC3T3-E1 cells: a temporal sequential study". *Drug Des Devel Ther*;12: pp1715-1726.

75. Iwaniec UT, Turner RT, et al; (2013) "Evaluation of long-term vitamin E insufficiency or excess on bone mass, density, and microarchitecture in rodents". *Free Radic Biol Med*;65: pp1209-1214.

76. Nguyen TTH, Eo MY et al; (2019) "Effects of pentoxifylline and tocopherol on a rat-irradiated jaw model using micro-CT cortical bone analysis". *Eur Arch Otorhinolaryngol*;276(12): pp3443-3452.