AOJ Emergency and Internal Medicine

Mini review

Open Access

Assessment of Vitamin E Bone Mass Impacts and Implications for Hip Fracture Prevention and Optimal Post Fracture Recovery [2000-2026]

Volume 1 Issue 3 – 2026

Ray Marks
[1]OARC Clinical Research and Education Director, Ontario L3T 5H3, Canada

Correspondence: Ray Marks, OARC Clinical Research and Education Director, Ontario L3T 5H3, Canada. Email doctorraymarks75@gmail.com

Received: February 17, 2026 **Published:** February 28, 2026

Citation: Marks R. Assessment of Vitamin E Bone Mass Impacts and Implications for Hip Fracture Prevention and Optimal Post Fracture Recovery [2000-2026]. AOJ Emerg and Int Med. 2026;1(3):51–60.

Abstract

Bone mass declines and related bone fracture risks and events, such as those that occur at the hip joint, have been topics of great interest for first responders and others for many decades. However, the means of preventing bone loss or the development of osteoporosis associated with fractures at any site while receiving more clinical attention has yet been quite limited and fragmented with no definitive consensus and warrants persistent attention. In particular, given the failure of medical approaches in general to build bone safely, or ensure optimal rapid healing of a fractured bone, a focus on the emergent numbers of high age often frail adults and their fracture risk and ensuing disablement who may benefit from vitamin E containing foods, and their possible promise for fostering optimal bone modeling, and averting bone resorption are quite compelling. As such, we conclude subject to further study, an optimal intake of vitamin E based nutrients may help maximize bone status as well as repair across all levels of prevention and in accord with the Haddon Matrix Injury Prevention Framework of those key pre injury, injury, and post periods where optimizing bone health is of the utmost clinical importance.

Keywords: Aging. Bone, Hip Fractures, Fracture Healing, Osteoporosis, Prevention, Rehabilitation, Vitamin E

Background

Bone, a specialized form of connective tissue vital for efficient load-bearing and mobility[1] as well as serving as a key attachment site for muscles, tendons, joint capsules, and ligaments that interact to move the body parts, also houses an intrinsic neurovascular system and cell pathways and receptors that modulates bone metabolism balances and remodeling. However, in the face of pain, an injurious fall, as well as age associated bone diseases such as osteoporosis, there may be a generalized thinning of the bones that not only reduces overall functional and structural well-being, but significantly increases the risk of sustaining a fracture that can lead to immense physical challenges as well as profound negative psychosocial and economic outcomes. With very few intervention approaches that can safely prevent or delay the onset of the increasing risk of developing this increasingly widespread prevalent costly public health condition and its related excess morbidity and premature disablement process, as well as increased fracture risk, it appears a timely review of this public health realm of challenge that is projected to worsen incrementally by 2030 is strongly indicated.

In this regard, while many articles refer to a role for vitamin D, as a bone status enhancer, here we examine the possible supplementary clinical role of vitamin E, an important antioxidant found to stimulate trabecular or cancellous bone formation,[2] one of two types of bone investigated for some time in the realm of its bone turnover impacts, whether high or low.[3] Supporting this line of inquiry is its key role in mediating optimum immune health, DNA repair, and signal transduction mechanisms that influence or regulate bone mass balances and quality[4] Moreover. its presence may avert a possible fracture risk,[5-7] as well speed up and ensure optimal post fracture healing and recovery via its anti inflammatory antioxidant properties that impact bone remodeling.[1]

However, despite a wealth of cumulative research, the efficacy of vitamin E in efforts to foster bone health and improve injury tolerance and fracture healing has not been robust or translated clinically in any realm even if this shows on balance a positive association between vitamin E and a host of bone protective and physiological associations.[9,11,12]

While it is impossible to cover this extensive literature in one report, this current overview is an attempt to consolidate those insights, especially those accrued in the past 25 years that might be helpful both clinically, as well as in the context of emergency room visits, triage, screening, and educational practices.

This topic seems especially important to investigate from a nutrition oriented biochemical view point, because vitamin E, while essential- is not synthesized by the human, and may need to depend upon external food sources. As well, research shows more than 93% of American adults may not meet the estimated average vitamin E requirement of 12 mg/day,[13] even though parallel research favors a role for vitamin E more often than not in efforts to optimize healing in non-surgical and surgical situations. Vitamin E studies further show a role for a diversity of vitamin E associated positive effects on bone biology, maintenance, repair, and regenerative processes.[14-16]

As outlined in recent work[10] it appears vitamin E presence can also mitigate osteoarthritis progression. a disease commonly associated secondarily with a hip fracture injury as a result of trauma or surgery. It can do this through strengthening the adjoining musculature, plus gene regulation, protection of the subchondral vasculature, and its anti-inflammatory properties that tend to improve the wear characteristics of highly crosslinked polyethylene and metal alloys in arthroplasty contexts post fracture that may otherwise be disabling and evoke dislocation of the prosthesis. In addition to its antioxidant and osteointegrative abilities the presence of vitamin E appears to mitigate postoperative arthrofibrosis and septic loosening often a concern in high age frail post hip fracture cases. Its presence may thus mediate or moderate otherwise high chances of incurring;

- progressive disability
- chronic pain
- a profound change in lifestyle
- risk of a second hip fracture
- a need for nursing home care
- enormous health care costs

As such, it appears safe to say that if obscured or overlooked in one or more ways either as a health determinant and functional correlate or as an anti oxidant, those older adults who remain vitamin E deficient may find themselves inadvertently at increased risk for both osteoporosis and injurious falls that culminates in a hip fracture often associated with high rates of malnutrition including too few foods containing vitamin E.[18] As such, they may experience excessively disabling and poor trauma outcomes, even if their immediate care is delivered optimally. They may experience a slower than desirable protracted recovery rate, and increasing bone losses plus declines in physical function and bone healing and thus possible future health challenges, system usage and demands.[19]

Aims

Given the strong global need to improve upon prevailing preventive and treatment strategies to counteract osteoporosis and bone fractures and to foster bone healing, in general, as indicated, and specifically at the hip joint, the present scoping review extends and incorporates a former one that covered data up to 2019. In addition to revising that data a sincere attempt was made to extract available recently published 2020-2926 English language peer reviewed literature concerning the nature of the associations that appears to exist between bone health and vitamin E levels and as applied to hip fracture prevention and treatment. The key question was whether an updated review would provide more definitive data as to whether bone status can be mediated or moderated by vitamin E, for example can rendering key vitamin E containing foods made available to those at risk for bone disease, attrition, and hip fractures prove beneficial, as well as in 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 eliminate vitamin E intake would appear reasonable to contemplate. A second aim was to examine if further research in this realm would be desirable as has been recently outlined.[10]

Methods

To establish what has unfolded over time the review targeted documents published predominantly over the past twenty five years i.e. January 1 2000-February 28, 2026. The search terms used to retrieve original studies and reviews were: *Vitamin E and Bone Health*, *Tocopherol and Osteoporosis*, *Vitamin E* and *Hip Fracture*, *Tocotrienol and Osteoporosis*, the latter terms referring to the two key structurally related sub groups of vitamin E. Databases explored were: **PUMED, Google Scholar**, **Science Direct,** and **PubMed Central.** 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 analyzed. Among those research studies deemed relevant to the current aims, no restrictions were placed on the nature of the documented research designs, provided they addressed the major themes of present interest.

After selecting the most relevant articles, the downloaded references were read carefully, and classified as being supportive of efforts to assess and intervene upon deficiencies in vitamin E or not. Studies were further categorized as being basic or clinical in nature, although the focus was on clinically conducted studies. In this regard, and after scrutinizing the available studies and their differing designs and research questions, it appeared that only a narrative overview would be suitable for reporting on these wide ranging approaches and research methods.

However, by including the most up to date findings on this topic, plus an overview of prior work on vitamin E as deduced from a series of basic study approaches, a broad picture of the issues and conclusions that prevail concerning the clinical relevance of the current topic was anticipated.

For consistency the terms used to describe the vitamin E substrate being discussed in the research were those reported by the authors, even though these were not necessarily strictly comparable across studies. 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.

Rationale

In addition to aging processes, oxidative stresses brought on by prolonged chronic diseases, as well as cases receiving glucocorticoid therapy, tends to reduce bone growth and repair, bone structure antioxidant regulatory mechanisms, and their mechanical and molecular influences on bone structural properties and qualities. Nevertheless, although this pathogenic state attributed to the excess presence of free radicals that can promote osteoclastic activity and inhibit osteoblast activity, the antioxidant *tocotrienol*- a vitamin E analogue- appears to offer protection against illnesses linked to free radicals, potentially including osteoporosis.[20] Men with adequate vitamin E levels also tend to show better hand grip strength than deficient cases that might explain why this group is less likely to sustain a fragility fracture than those with sarcopenia or muscle mass declines.[27]

Theoretical Model

To alert researchers to factors contributing to ***injury*** incidence and severity, and the timing of these factors, William Haddon Jr. an injury scientist devised a **matrix** of broad categories of factors and phases of injury that we feel are highly relevant to bone maintenance, injury prevention, and healing potential, especially in the realm of personalized medicine.

In this injury prevention approach a matrix of four columns and three rows combine to help envision the role of the host agent and environment in the three tier realm of primary, secondary, and tertiary injury prevention. More specifically, the *factors* defined by the columns in the matrix refer to the interacting factors that contribute conceptually and as closely as possible to the injury process. The host column refers to the person at risk of injury, such as a high age adult. The agent of injury refers to various possibly remediable energy impacts [for example mechanical energy impacts on bone that are transmitted to the host with low bone mass and forceful

contact with the ground]. Other factors such as personal [risky] behaviors and health status are additional potential explanatory vectors underpinning injurious bone mass disruption in this model. Physical environments and their deterministic attributes further include all the characteristics of the setting in which the injury event takes place (for example an icy sidewalk or unsafe home. Social and legal norms and practices in the culture are referred to as the social environment determinants and can include poverty, alcohol consumption, or policies about narcotic agents often given to older adults [https://www.npaihb.org/images/epicenter_docs/injuryprevention/HaddonMatrixBasics.pdf].

Through this aforementioned lens, we believe vitamin E can be seen as a life affirming mechanism that is cross cutting along all model dimensions as far as bone fragility or strength and health behaviors and the disruption of this are concerned.

Clinical relevance

A recent human study indicated a linear association between dietary vitamin E levels and osteoporosis in an older population in the United States.[28] Other attributes can be uncovered with the assistance of the Haddon Matrix as can possible benefits be projected from counter measures as summarized in the findings section below. Proposed bone mediating mechanisms of action are vitamin E and its observed ability to maintain favorable bone gene expressions and anti-resorptive host properties[25] that may influence vector effects, as well as key bone regulators favoring skeletal integrity/net bone mass increases and recovery post injury.[29]

Key Literature Search Observations

These include many pre clinical studies usually in a rodent model of bone attrition as opposed to naturally emerging osteoporosis. Clinical themes are less conclusive and highly diverse with more technical flaws than lab studies.

Laboratory Based Studies

As summarized below, vitamin E has been found to induce favorable bone building, antiosteoporotic impacts and bone remodeling processes quite consistently, regardless of model generated.[19-21] A mechanistic explanation moreover tends to support their clinical promise even if untested in man to any degree to date. In addition the data cannot be readily applied because they vary in terms of type of vitamin E compound studied, assay method, mode and duration of delivery, the nature of the bone related correlates examined,[22-24] animals examined, and their varying ages, method of producing osteoporosis and possible gender effects. Moreover, some investigate only current or short term vitamin E cellular effects in general, others investigate the specific role of either tocopherols or tocotrienols that represent one of the two subgroups of vitamin E,[25] others examined co-administration supplementary effects on bone loss, thereby possibly occluding the value of the unique vitamin E impacts.[26]

These probable impacts include favorable

- Bone remodeling/formation/regulation processes.[2,19,29,34]
- Fracture/bone healing benefits.[30-32,36, 37,40]
- Oxidative stress mitigation.[33]
- Bone calcium homeostatis.[35]
- Osteocyte membrane repair/survival.[8]
- Reductions in risk of inflicted.[37]/late life osteoporosis.[29,30,38,39-43,47]
- Tocotrienols based bone-promoting anti-inflammatory and immuno-modulatory effects.[29,44]

Clinically Related Observations

Despite a strong imperative to go beyond lab models and pursue vitamin E bone association in the clinical realm, this current broad-based search of the literature revealed very few contemporary studies translating lab data to the bedside or whether vitamin E can avert, or optimize fracture healing. Moreover, the study findings that prevail in either case must yet be viewed with caution, because unlike animal studies that can more readily control for confounding factors and bone status, and can examine the trajectory of osteoporosis from its inception, many factors including health status, gender, age, food scarcity, and health behaviors, among others that impact bone health are much more challenging to control 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 as well as dietary habits and access. Follow up is also much more susceptible to attrition or the effects of history and other factors in the human condition. However, as in pre-clinical situations, Wong.[44] who examined vitamin E bone interactions, concluded that tocotrienols, found in certain foods, do appear to have the potential to produce skeletal-promoting benefits, and do this by modulating the levels of osteocytes-derived bone-related peptides. Peng.[45] found vitamin E, - especially tocotrienol- was able to alleviate IL-1, IL-6, RANKL, iNOS and hs-CRP levels in relation to bone metabolism, denoting its anti-osteoporotic actions occur via its anti-inflammatory and immunomodulatory mechanisms. Shen.[46] who explored the impact of tocotrienol [TT] supplementation as applied over a 12-week period on various bone markers among 89 postmenopausal women with osteopenia randomly assigned to three groups:

(1) placebo (430 mg olive oil/day),
(2) low TT (430mg TT/day, 70% purity), and
(3) and high TT (860mg TT/day, 70% purity),
where the term TT referred to an extract of tocotrienol obtained from an annatto seed substrate, and that consisted of 90% delta-tocotrinol and 10% gamma-tocotrienol found the 12-week program of annatto-extracted tocotrienol supplementation decreased bone resorption and improved bone turnover rate, and that the osteoprotective effects were possibly mediated by a parallel inhibitory impact on oxidative stress.

Discussion

Years of research has shown that bone as a key structural tissue requires a variety of nutrients throughout life in order to develop and assume its vital functional roles.[57] Conversely, the idea that bone loss 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 has been put forth but with non conclusive results.[58,59] That is, despite a fairly robust health associated and emergent role for vitamin E, the body's most important antioxidant,[10] and one found to mediate a variety of anti oxidative processes that could potentially affect bone status adversely, the question of whether suboptimal vitamin E intake levels or availability or uptake or all of these factors has a bearing on human bone health, including the onset and progression of osteoporosis the issue of vitamin E supplementation remains unresolved. This is despite promising carefully controlled lab based studies generated in a number of animal models,[1] and the basic established understandings of vitamin E intake and/or health as well as bone health,[1] Its observed immunomodulatory and inflammatory actions[42] against free radical damage[1] may indeed be clinically beneficial in the face of unexpected bone trauma and should be more thoroughly examined in our estimation.[19]

Even if results are hard to aggregate because the vitamin E isomer studied, as well as its dose in some cases are diverse,[58] as are bone status indicators, in our view, vitamin E serum levels and their role in mediating bone status should be studied more carefully in the future using established bone biomarkers. At present, a beneficial bone loss prevention approach may be underutilized however, because current results are often based on poorly designed and open to bias studies that are under sized and test dissimilar associations and conditions and rarely high age adults and in reality has not improved in more than a decade as far as its frscture repair potential goes.[17]

Moreover, heterogeneous samples that continue to be studied may not represent the high age adult needs, nor their potential health beliefs and possible confusion about categories and/or dosages of vitamin E and how these nay impact bone status as well as overall health status over time. In some cases, those patients studied who have already received vitamin E infused prosthetic femoral head joint replacement[62] may prove confounding if not excluded. As well, the influences of lifestyle, comorbid illnesses, pain status, inflammation, and genetic factors, plus the use of subjectively reported and aggregated dietary assessment data,[9] unknown supplement purity and scheduling factors, plus the cross-sectional nature of most clinical studies [generally the mode of inquiry underpinning most negative outcomes], unknown supplement type or incompatible types are some obvious additional research limitations that diminish the salience of vitamin E as a bone status mediator or moderator in humans and where data on bone mineral density may depend on the site studied.[18]

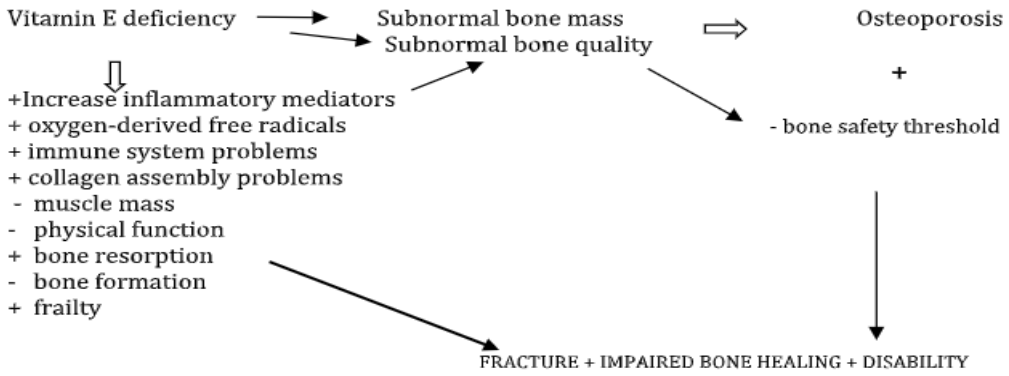

Figure 1. Envisioned interactions between vitamin E and bone [Author]

Concluding Remarks

Notwithstanding the limited number as well as quality of clinical studies that focus on vitamin E and bone in the context of osteoporosis prevention and intervention, plus fracture healing, along with their oftentimes contradictory findings,[73] 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.[4,7]
2. Despite several contrary studies in both the laboratory as well as the clinical realms, the recent findings by Shen[46] seem to provide an especially promising ray of progress in this area of clinical practice that remains challenging to otherwise address effectively at either a primary or a secondary prevention level.
3. It appears specific tocotrienol supplementation can suppress bone resorption and oxidative stress levels in osteopenic women, a finding consistent with Muhammad et al.[74]
4. In addition, research shows vitamin E may well orchestrate many fundamental bone building mechanisms at the molecular and cellular level that can account for its observed impact on bone,[7,75] as well as influencing oxidant[23,62] and inflammatory status.[4,75]
5. Rather than relying on pharmaceutical drugs, vitamin E in various forms may prevent postmenopausal osteoporosis,[74] as well nicotine-induced osteoporosis.[16]
6. Sufficient vitamin E presence has the potential to avert, help avert, or reduce hip fracture disability, and promote bone healing,[1,18,21,76-78] but may depend on food accessibility and presence of selected foods such as fats that mediate vitamin e activity.[89]

Final thoughts and implied applications

Our research leads us to believe, allied and geriatric health professionals can do much to alleviate suffering in them at aging clients at risk for bone mass declines and osteoporosis and hip fracture and with modest confidence from a vitamin E vantage point where its intake is deficient. They can carefully apply the Haddon Matrix to envision where the emphasis should be placed for an individual and examine any downside of failing to do this as outlined in Figure 1. At the same time, health related issues that have a bearing on bone status,[47] including how vitamin E analogues have to be ingested within a fat substrate to potentially foster its uptake and associated bone cell status and structural strength outcomes. They can monitor their case load and intervene as indicated and based on all we know their actions are likely to prove especially insightful and desirable.[78]

At the same time, the comparative efficacy of oral versus intramuscular vitamin E administration, and varying homologues of vitamin E on bone health, as assessed with carefully selected qualitative and quantitative bone marker variables that capture bones' static as well as its dynamic parameters will similarly prove helpful in all likelihood despite challenges in doing this. Moreover, to minimize bias and achieve more dependable personalized insights, and the role of a variety of health factors, health behaviors, and medications more specific attention is warranted.[57] Researchers can especially help by controlling for additional bone mediating factors such as gender, vitamin E dosage-associations, bone, hormonal, exercise, bone and smoking status,[86,87] plus age, within a framework of prospective studies using robust design and timely validated analytic instrumentation principles that are undergirded by the Haddon Matrix organizing principles.[88]

In the interim, we believe sufficient vitamin E evidence exists to guide and provide many older adults some degree of bone loss protection and prevention even in the pre frail adult[90] and thereby avert a possible high fracture risk and its severity and ramifications at multiple levels. In addition, even if the mechanistic data in lab models may not translate to or replicate the 'real world' situation at all closely, doing something now for the aging populations most at risk for bone fractures who 'can't wait' for assurances without risk is indicated in our estimation. Rather, clinicians who wish to prevent suffering can use both the prevailing evidence base alongside their judgment, clinical experience, and education in carefully weighing or weighting the pros of an older frail adult or younger at risk adult using vitamin E supplements or not, They can educate vulnerable adults about those foods that might influence bone remodeling and fracture risk and what can be recommended or cautioned against and can keep records of the anticipated outcomes and any indicated future adjustments.

In addition to this, food quality and access as well as joint protection may prove important considerations in helping the most vulnerable adults who are likely to be malnourished and depressed, such as the recurrent faller and an actual hip fracture case. Vitamin E also has other health benefits other than supporting bone health directly, such as positively influencing aging muscle mass, strength and frailty risk that might otherwise impact bone health adversely.[55] Vitamin E may also mitigate bone loss, improve bone formation in animal models of osteoporosis,[26,63,64] as well as positively protecting bone and osteoblast mineralization in addition to cases of steroid induced osteoporosis,[1,44] and bone loss in men undergoing androgen deprivation therapy.[65] It may also help build bone from an early age,[1] and preserve bone in instances where

skeletal unloading is indicated.[66] Jn all the above scenarios, as per the need to preserve bone mass across many interacting forces, a brief one page understandable Fact Sheet on vitamin E and bone and best sources and mode of obtaining results may prove helpful to the client as well as their caretakers.

Researchers in the interim can further explore vitamin E and its molecular actions as regards distinct bone cell pathways and measureable molecular and bone tissue impacts and what appears efficacious for averting negative bone impacted cardiovascular and hormonal states,[65,67] as well as in aiding the treatment of age-related osteoporosis[68] and bone fracture prevention and repair.[83]

Testing whether preventing vitamin E deficiency may foster rather than impair bone calcium status,[69] a crucial bone-mediating factor, and the potential for removing free radicals via vitamin E infusion at time of surgery is hypothesized to preserve the artificial joint lining and inserts mechanical properties and wear behavior in vivo. Indeed, excellent polyethylene wear behavior has been noted and should be studied further as perhaps bone itself can be directly infused in a similar way[62] even if a null or noxious effect of vitamin E on bone status has been previously reported.[57,70-73]

Acknowledgement

None.

Conflicts of Interest

None.

Funding

None.

References

1. Mehat MZ, Shuid AN, Mohamed N, et al. Beneficial effects of vitamin E isomer supplementation on static and dynamic bone histomorphometry parameters in normal male rats. *J Bone Min Metab*. 2010:28(5);503-509.
2. Smith BJ, Lucas EA, Turner RT, et al. Vitamin E provides protection for bone in mature hindlimb unloaded male rats. *Calcif Tissue Int*. 2005;76(4):272-279.
3. Torbergsen AC, Watne LO, Wyller TB, et al. Micronutrients and the risk of hip fracture: case-control study. *Clin Nutr*. 2017;(2);438-443.
4. Rondanelli M, Faliva MA, Peroni G, et al. Focus on pivotal role of dietary intake (diet and supplement) and blood levels of tocopherols and tocotrienols in obtaining successful aging. *International J Molecular Sci.* 2015;16(10);23227-23249.
5. Zhang J, Hu X, Zhang J. Associations between serum vitamin E concentration and bone mineral density in the US elderly population. *Osteoporos Int*. 2017;28(4);1245-1253.
6. Brooks R, Kalia P, Ireland DC, et al. Direct inhibition of osteoclast formation and activity by the vitamin E isomer gamma-tocotrienol. International Journal of Vitamin *Nutrition Research*. 2011:81(6);358-367.
7. Guralp O. Effects of vitamin E on bone remodeling in perimenopausal women: mini review. *Maturitas*. 2014;79(4):476-480.
8. Hagan ML, Bahraini A, Pierce JL, et al. (2019) Inhibition of osteocyte membrane repair activity via dietary vitamin E deprivation impairs osteocyte survival. *Calc Tissue Int.* 2019;104(2);224-234.
9. Ilesanmi-Oyelere BL, Brough L, Coad J, et al. The relationship between nutrient patterns and bone mineral density in postmenopausal women. *Nutrients*. 2019;11(6).pii:E1262.
10. Viswanathan VK, Jain VK, Iyengar KP, et al. Evaluation of Vitamin E in Trauma and Orthopaedics: A Narrative Synthesis. *Indian J Orthop*. 2025;59(9):1364-1380.
11. Hampson G, Edwards S, Sankaralingam A, et al. Circulating concentrations of vitamin E isomers: association with bone turnover and arterial stiffness in post-menopausal women. *Bone*. 2015:81;407-412.
12. Abdullah Sani N, Kamaruddin NA, Soelaiman IN, et al. Palm Tocotrienol activates the Wnt3a/β-Catenin Signaling Pathway, protecting MC3T3-E1 osteoblasts from cellular damage caused by dexamethasone and promoting bone formation. *Biomedicines*. 2025;13(1):243.
13. Fulgoni VL, 3rd, Keast DR, Bailey RL, et al. Foods, fortificants, and supplements: where do Americans get their nutrients? *J Nutr*. 2011:141(10);1847-1854.
14. Kurklu M, Yildiz C, Kose O, et al. Effect of alpha-tocopherol on bone formation during distraction osteogenesis: a rabbit model. *J Orthop Traumatol*. 2011;12:153-158.
15. Chin KY, Mo H, Soelaiman IN. A review of the possible mechanisms of action of tocotrienol - a potential antiosteoporotic agent. *Curr Drug Targets*. 2013;14(13):1533-541.
16. Abukhadir SS, Mohamed N, Makpol S, et al. Effects of palm vitamin R on bone-formation-related gene expression in nicotine-treated rats. *Evid Based Complement Alternat Med*. 2012;2012:656025.
17. Borhanuddin B, Mohd Fozi NF, Naina Mohamed I. Vitamin E and the healing of bone fracture: the current state of evidence. *Evid Based Complement Alternat Med*. 2012;2012:684510.
18.Torbergsen AC, Watne LO, Frihagen F, et al. Effects of nutritional intervention upon bone turnover in elderly hip fracture patients. Randomized controlled trial. *Clin Nutr ESPEN*. 2019;29:52-58.
19. Ferrucci L, Magaziner J, Shardell MD. Serum vitamin E concentrations and recovery of physical function during the year after hip fracture. *J Gerontol A Biol Sci Med Sci*. 2011;66(7):784-93.
20. Ramli ESM, Ahmad F, Kamaruddin NA, et al. Comparative bone-protective effects of tocotrienol isomers from palm and annatto in dexamethasone-induced Osteoporotic Male Rats. *Int J Mol Sci*. 2025;26(20):10206.
21. Ibrahim N', Mohamed N, Soelaiman IN, et al. 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 Public Health*. 2015;12(10):12958-76.
22. Chin KY, Ima-Nirwana S. The effects of α-tocopherol on bone: a double-edged sword? *Nutrients*. 2014;6(4):1424-41.
23. Casati L, Pagani F, Limonta P, et al. Beneficial effects of δ-tocotrienol against oxidative stress in osteoblastic cells: studies on the mechanisms of action. *Eur J Nutr*. 2019.
24. Iwaniec UT, Turner RT, Smith BJ, et al. Evaluation of long-term vitamin E insufficiency or excess on bone mass, density, and microarchitecture in rodents. *Free Radic Biol Med*. 2013;65:1209-1214.
25. Li H, Cao W, Tao L, et al. The role of fat-soluble vitamins on bone metabolism and osteoporosis: a literature review. *Ann Med*. 2025;57(1):2533429.
26. Abdelkarem HM, Fadda LH, El-Sayed EM, et al. Potential role of l-arginine and vitamin E against bone loss induced by nano-zinc oxide in rats. *J Diet Suppl*. 2018;15(3):300-310.
27. Wu D, Wang H, Wang W, et al. Association between composite dietary antioxidant index and handgrip strength in American adults: data from National Health and Nutrition Examination Survey (NHANES, 2011-2014). *Front Nutr*. 2023;10:1147869.
28. Zhuang R, Hou W, Zhang T, et al. Association between dietary vitamin E and osteoporosis in older adults in the United States. *Front Endocrinol (Lausanne)*. 2024;15:1410581.
29. Wong SK, Chin KY, Ima-Nirwana S. 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 Public Health*. 2019;16(18). pii: E3313.
30. Shuid AN, Mehat Z, Mohamed N, et al. Vitamin E exhibits bone anabolic actions in normal male rats. *J Bone Miner Metab*. 2010;28(2):149-156.

31. Durak K, Sonmez G, Sarisozen B, et al. Histological assessment of the effect of alpha-tocopherol on fracture healing in rabbits. *Int Med Res*. 2003;31(1):26-30.
32. Paskalev MD, Goranov NV, Krastev SJ, et al. Antioxidant and bone healing effect of vitamin E in an experimental osteotomy model in dogs. *Comp Clin Path*. 2011;20(4):403-408.
33. Nizar AM, Nazrun AS, Norazlina M, et al. Low dose of tocotrienols protects osteoblasts against oxidative stress. *Clin Ter*. 2011;162(6):533-8.
34. Kasai S, Ito A, Shindo K, et al. High-dose α-tocopherol supplementation does not induce bone loss in normal rats. *PLoS One*. 2015;10(7):e0132059.
35. Norazlina M, Chua CW, Ima-Nirwana S. Vitamin E deficiency reduced lumbar bone calcium content in female rats. *Med J Malaysia*. 2004;59(5):623-30.
36. Savvidis M, Papavasiliou K, Taitzoglou I, et al. Postoperative administration of alpha-tocopherol enhances osseointegration of stainless steel implants: an in vivo rat model. *Clin Orthop Relat Res*. 2020;478(2):406-419.
37. Mohamed N, Ahmed Abukhadir SS, Syed Hashim SA, et al. Vitamin E Improves cellular and structural bone histomorphometry in an alcohol-induced osteoporosis rat model. *pharmaceuticals (Basel).* 2024;17(12):1730.
38. Abdul-Majeed S, Mohamed N, Soelaiman IN. The use of delta-tocotrienol and lovastatin for anti-osteoporotic therapy. *Life Sci*. 2015;125:42-48.
39. Suhaimi F, Muhammad N, Soelaiman IN. The effects of alpha-tocopherol supplementation on fracture healing in a postmenopausal osteoporotic rat model. *Clinics (Sao Paulo).* 2012;67(9):1077-1085.
40. Turk C, Halici M, Guney A, et al. Promotion of fracture healing by vitamin E in rats. *J Int Med Res*. 2004;32(5):507-512.
41. Mohamad S, Shuid AN, Mohamed N, et al. The effects of alpha-tocopherol supplementation on fracture healing in a postmenopausal osteoporotic rat model. *Clinics (Sao Paulo).* 2012;67(9):1077-1085.
42. Nazrun Shuid A, Das S, Mohamed IN. Therapeutic effect of Vitamin E in preventing bone loss: An evidence-based review. *Int J Vitam Nutr Res.* 2019;Mar:1-14.
43. Wong SK, Chin KY, Suhaimi FH, et al. 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 Public Health*. 2018;15(9). pii: E1828.
44. Kohno K, Yamada W, Ishitsuka A, S et al. Tocotrienol-rich fraction from annatto ameliorates expression of lysyl oxidase in human osteoblastic MG-63 cells. *Biosci Biotechnol Biochem*. 2020;84(3):526-535.
45. Peng YL, Wang ZY, Wang XJ, et al. Lower risk of low bone mineral density in high vitamin E level in older people: a cross-sectional study. *Clinical Nutrition ESPEN.* 2024;61:316-21.
46. Shen CL, Yang S, Tomison MD, et al. Tocotrienol supplementation suppressed bone resorption and oxidative stress in postmenopausal osteopenic women: a 12-week randomized double-blinded placebo-controlled trial. *Osteoporos Int*. 2018;29(4):881-891.
47. Skalny AV, Aschner M, Tsatsakis A, et al. Role of vitamins beyond vitamin D 3 in bone health and osteoporosis. International Journal of Molecular Medicine. 2024;53(1):1;26(20):10206.
48. Magremanne M, Reychler H. Pentoxifylline and tocopherol in the treatment of yearly zoledronic acid-related osteonecrosis of the jaw in a corticosteroid-induced osteoporosis. *J Oral Maxillofac Sur*g. 2014;72(2):334-337.
49. Shi WQ, Liu J, Cao Y, et al. 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*. 2016;115(1):113-120.
50. Michaëlsson K, Wolk A, Byberg L, et al. Intake and serum concentrations of α-tocopherol in relation to fractures in elderly women and men: 2 cohort studies. *Am J Clin Nutr*. 2014;99(1):107-114.
51. Pasco JA, Henry MJ, Wilkinson LK, et al. Antioxidant vitamin supplements and markers of bone turnover in a community sample of nonsmoking women. *J Womens Health (Larchmt)*. 2006;15(3):295-300.
52. Holvik K, Gjesdal CG, Tell GS, et al. Low serum concentrations of alpha-tocopherol are associated with increased risk of hip fracture. A NOREPOS study. *Osteoporos Int.* 2014;25(11):2545-54.
53. Mata-Granados JM, Cuenca-Acebedo R, Luque de Castro MD, et al. Lower vitamin E serum levels are associated with osteoporosis in earlypostmenopausal women: a cross-sectional study. *J Bone Miner Metab.* 2013;31(4):455-460.
54. Sun LL, Li BL, Xie HL, et al. Associations between the dietary intake of antioxidant nutrients and the risk of hip fracture in elderly Chinese: a case-control study. *Br J Nutr.* 2014;112(10):1706-1714.
55. Mulligan AA, Hayhoe RP, Luben RN, et al. 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*. 2021;10(2):159.
56. Chuin A, Labonté M, Tessier D, et al. Effect of antioxidants combined to resistance training on BMD in elderly women: a pilot study. *Osteoporos Int*. 2009;20(7):1253-258.
57 D'Adamo CR, Miller RR, Shardell MD, et al. Higher serum concentrations of dietary antioxidants are associated with lower levels of inflammatory biomarkers during the year after hip fracture. *Clin Nutr*. 2012;31(5):659-665.
58. Shen CL, Klein A, Chin KY, et al. Tocotrienols for bone health: a translational approach. *Ann N Y Acad Sci*. 2017;1401(1):150-165.
59. Norazlina M, Chua CW, Ima-Nirwana S. Vitamin E deficiency reduced lumbar bone calcium content in female rats. *Med J Malaysia*. 2004;59(5):623-630.
62. Thoen PS, Lindalen E, Nordsletten L, et al. Low wear with vitamin E-infused highly cross-linked polyethylene : 14 years' follow-up of a randomized radiostereometric analysis study. *Bone Joint J*. 2025;107-B(11):1182-1188.
63. Wan Hasan WN, Abd Ghafar N, Chin KY, et al. Annatto-derived tocotrienol stimulates osteogenic activity in preosteoblastic MC3T3-E1 cells: a temporal sequential study. *Drug Des Devel Ther*. 2018 ;12:1715-1726.
64. Johnson SA, Feresin RG, Soung do Y, et al. Vitamin E suppresses ex vivo osteoclastogenesis in ovariectomized rats. *Food Funct*. 2016:1628-1633.
65. Mohamad NV, Soelaiman IN, Chin KY. Effects of tocotrienol from Bixa orellana (annatto) on bone histomorphometry in a male osteoporosis model induced by buserelin. *Biomed Pharmacother*. 2018;103:453-462.
66. Smith BJ, Lucas EA, Turner RT, et al. Vitamin E provides protection for bone in mature hindlimb unloaded male rats. *Calcif Tissue Int*. 2005;76(4):272-279.
67. Wong SK, Chin KY, Ima-Nirwana S. 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 Public Health*. 2019;16(18); pii: E3313.
68. Ruiz-Ramos M, Vargas LA, Fortoul Van der Goes TI et al, Supplementation of ascorbic acid and alpha-tocopherol is useful to preventing bone loss linked to oxidative stress in elderly. *J Nutr Health Aging*. 2010;14(6):467-472.
69. Norazlina M, Ima-Nirwana S, Abul Gapor MT, et al. Tocotrienols are needed for normal bone calcification in growing female rats. *Asia Pac J Clin Nutr*. 2002;11(3):194-199.
70. Vallibhakara SA, Nakpalat K, Sophonsritsuk A, et al. Effect of vitamin E supplement on bone turnover markers in postmenopausal osteopenic women: a double-blind, randomized, placebo-controlled trial. *Nutrients*. 2021;13(12):4226.
71. Fujita K, Iwasaki M, Ochi H, et al. Vitamin E decreases bone mass by stimulating osteoclast fusion. *Nature Med*. 2012:18(4);589-594.
72. Tennant KG, Leonard SW, Wong CP, et al. High-dietary alpha-tocopherol or mixed tocotrienols have no effect on bone mass, density, or turnover in male rats during skeletal maturation. *J Med Food*. 2017;20(7):700-708.
73. Lu Y, Yang Z, Duan J, et al. Associations of serum vitamins, carotenoids, and retinyl esters with risk of hip and spine fractures. *Nutr Hosp*. 2025;45(5):1016-1030.
74. Muhammad N, Luke DA, Shuid AN, et al. Tocotrienol supplementation in postmenopausal osteoporosis: evidence from a laboratory study. *Clinics (Sao Paulo).* 2013;68(10):1338-1343.
75. Kanchi MM, Shanmugam MK, Rane G, et al. Tocotrienols: the unsaturated sidekick shifting new paradigms in vitamin E therapeutics. *Drug Discov Today*. 2017;22(12):1765-1781.
76. Muhammad N, Luke DA, Shuid AN, et al. Two different isomers of vitamin E prevent bone loss in postmenopausal osteoporosis rat model. *Evid Based Complement Alternat Med*. 2012;2012:161527.
77. Zhang J, Munger RG, West NA, et al. Antioxidant intake and risk of osteoporotic hip fracture in Utah: an effect modified by smoking status. *Am J Epidemiol*. 2006;163(1):9-17.

78. Casati L, Pagani F, Maggi R,. Food for bone: evidence for a role for delta-tocotrienol in the physiological control of osteoblast migration. *Int J Mol Sci*. 2020;21(13):4661.
79. Mohamad S, Shuid AN, Mohamed N, et al. Therapeutic effect of vitamin E in preventing bone loss: an evidence-based review. *Int J Vitam Nutr Res*. 2019:1-14.
80. Radzi NFM, Ismail NAS, Alias E. Tocotrienols regulate bone loss through suppression on osteoclast differentiation and activity: a systematic review. *Curr Drug Targets*. 2018;19(9):1095-1107.
81. Chin KY, Ima-Nirwana S. The role of tocotrienol in preventing male osteoporosis-a review of current evidence. *Int J Mol Sci*. 2019;20(6). pii: E1355.
82. Ha H, Lee JH, Kim HN, et al. α-Tocotrienol inhibits osteoclastic bone resorption by suppressing RANKL expression and signaling and bone resorbing activity. *Biochem Biophys Res Commun*. 2011;406(4):546-551.
83. Zhou P, Shao R, Wang H, et al. Dietary vitamin A, C, and E intake and subsequent fracture risk at various sites: A meta-analysis of prospective cohort studies. *Medicine (Baltimore).* 2020;99(35):e20841.
84. Feresin RG, Johnson SA, Elam ML, et al. Effects of vitamin E on bone biomechanical and histomorphometric parameters in ovariectomized rats. *J Osteoporos*. 2013;2013:825985.
85. Zakaria S, Mat-Husain SZ, Ying-Hwey K, et al. Vitamin E improved bone strength and bone minerals in male rats given alcohol. *Iran J Basic Med Sci.* 2017;20(12):1360-1367.
86. Pedrinolla A, Isanejad M, Antognelli C, et al. Randomised controlled trial combining vitamin E-functionalised chocolate with physical exercise to reduce the risk of protein-energy malnutrition in predementia aged people: study protocol for Choko-Age. *BMJ Open.* 2023.
87. Shuid AN, Mohamad S, Muhammad N, et al. Effects of α-tocopherol on the early phase of osteoporotic fracture healing. *J Orthop Res*. 2011.
88. Haddon W Jr. The changing approach to the epidemiology, prevention, and amelioration of trauma: the transition to approaches etiologically rather than descriptively based. 1968. *Inj Prev*. 1999;5(3):231-235.
89. Yang J, Wang A, Shang L, et al. Study on the association between dietary habits, patterns and frailty of the elderly: a cross-sectional survey from communities in China. *Clin Interv Aging*. 2022;17:1527-1538.
90. Kochlik B, Stuetz W, Pérès K, et al. Associations of fat-soluble micronutrients and redox biomarkers with frailty status in the FRAILOMIC initiative. *J Cachexia Sarcopenia Muscle*. 2019;10(6):1339-1346.