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Biological role of Vitamin K in Human Health: A comprehensive review

Mahesh Dhola[1], Murtaza Hajoori[2]
[1]PG Student, Bhagwan Mahavir College of Basic and Applied Sciences, Bhagwan Mahavir University, Surat.
[2]Assistant Professor, Bhagwan Mahavir College of Basic and Applied Sciences, Bhagwan Mahavir University, Surat.

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ABSTRACT
Vitamin K, a crucial nutrient, is essential for various biological processes such as blood clotting and bone health. Present in two primary forms—K1 and K2—it is sourced from foods like leafy green vegetables, soybeans, meats, and cheeses. Research underscores the significant association between vitamin K deficiency and bone fractures. Adequate vitamin K intake has been shown to notably decrease the risk of fractures. Furthermore, studies indicate that maintaining optimal levels of vitamin K can result in enhanced bone health and lower rates of fractures over time, underscoring its importance in skeletal integrity. In conclusion, vitamin K's multifaceted role in the body emphasizes its value in promoting skeletal strength and overall well-being.

Keywords: Vitamin K, Fat soluble vitamin, Phylloquinone, Menaquinone

I. INTRODUCTION:

It is an important fat-solvent nutrient because it can bind to a variety of proteins found in the human body, including the coagulation factors (II, VII, IX, X, protein S and protein C), osteocalcin (a bone-shaping protein), and Matrik Gla Protein (MGP), an anti-calcification protein [1]. Vitamin K is generally present as nutrients K1 (phylloquinone) and K2 (menaquinone, MK-4 over MK-10) . Vitamin K was discovered in 1929 by Henrik Dam. Later in the 1980s, Edward Doisy identified between the many forms and structures of vitamin K. As a result of their combined efforts, they were awarded the Nobel Prize in 1943. Because vitamin K plays a fundamental role in hemostasis, deficiency may cause bleeding in a baby. The earliest depiction of bleeding brought on by vitamin K's insufficiency occurred soon after its discovery in the 1930s, when vitamin K was given to the major babies to cure clinical bleeding. Swedish scientist Jorgen Lehmann examined the use of both oral and intramuscular (IM). In the 1940s, research on vitamin K suggested that a modest dose of 0.5 to 1 mg was effective in lowering the risk of discharge in neonates. A water-soluble form of vitamin K was known and thought to offer better security in the 1950s. It shows that it has a big role in supporting the blood clot and preventing excessive bleeding [2]. This vitamin K isn't typically used as a dietetic supplement, in contrast to other vitamins. It is a compound gathering, quite literally. It appears that these mixes are extremely important sources of Vitamins K1 and K2. Leafy green foods, olive oil, and soybeans are good sources of vitamin K1. Vitamin K2 is a group of substances that are typically obtained from fermented soybean, butter, meats, cheeses, and egg yolk [3].

II. STRUCTURE OF VITAMIN K AND ITS SUBTYPES:

Vitamin K, which consists of vitamins K1 and K2, is a class of fat-soluble vitamins that are found and act in the membranes of living things. The chemical backbone of menadione, or vitamin K3, is shared by both forms; however, their lipophilic side chains are different Vitamin K2 includes unsaturated isopropyl side chains, which are referred to as MK-4 through MK-13 depending on their length, while vitamin K1 has a phenyl substituted chain [4]. The primary dietary source of vitamin K is found in vegetables, particularly in green leafy vegetables, vegetable oils, and some fruits. Fermented foods derived from animals and meals made by bacteria in the human stomach are sources of vitamin K2. MK-4 is an example since it is not frequently produced by bacterial synthesis and is thought to have animal origins due to its conversion from vitamin K1 that is specific to certain tissues [5]. However, while being called vitamin K3, menadione is not found naturally in food; rather, it is a byproduct of the breakdown of vitamin K1 and a precursor of tissue MK-4 that circulates. It would be more accurate to refer to it as a pro-vitamin for this reason [6].

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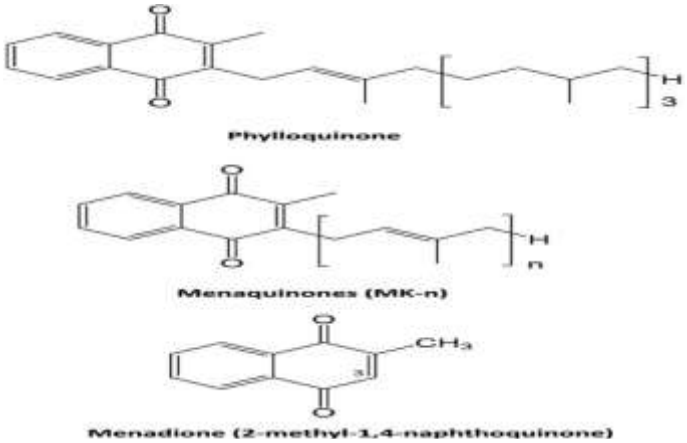

Figure1.StructureofvitaminK1(phylloquinone)andvitaminK2(menaquinone)Menadion(vitaminK3)represententhe commonK1andK2structure Menadioneisa synthetic vitaminK type [7].

III. FORMS OF VITAMIN K:

a. Vitamin K1

Vitamin K1 can now be produced chemically using a well-established method. It is employed in a variety of commercial applications, such as pharmaceutical products and human nutrition but not in cosmetics, where vitamin K1 compositions were outlawed in 2009 [8]. The main goals of improvements to vitamin K1 chemical production methods have been to reduce the creation of inactive Z-isomer and to employ less toxic compounds that are harmful to humans and the environment Removing residues of menadione is one of the main problems in chemical synthesis [9]. Furthermore, cyanobacteria species such as Anabaenacylindrica, Anabena variabilis, Spirulina spp, Nostoc muscorum, andSynechocytis spp. have been reported to possess the ability to biosynthesize and generate vitamin K1 [10].Biosynthesis of phylloquinone has been mainly associated with oxygenic photosynthetic organisms such as plants, algae, and cyanobacteria. MKs are described to be synthetized by a limited number of obligate and facultative anaerobic bacteria.Nevertheless, several species of cyanobacteria and microalgae, such as the cyanobacteria Gloeobacter violaceus [11].

b. Vitamin K2

The most researched and documented manufacturing techniques for natural vitamin K2 have been biosynthetic approaches employing bacterial fermentation, as opposed to chemical synthesis. This is mostly because many bacterial strains are easily manipulated and their growth conditions may be optimized. Microorganisms have the benefit of producing the all-trans isomer selectively. The focus of research on vitamin K2 biotechnological production has shifted in recent years from identifying the different types of bacteria that produce K2 to screening high-producing bacterial strains. These strains are frequently coupled with resistant and genetically altered mutants to produce higher K2 yields. Improved growth conditions and high-producing bacterial strains have been used to create bioengineered K2 metabolic pathways, as reported more recently [12]. For the production of vitamin K2 in a variety of bacterial species, including Flavobacterium spp, Lactic acid bacteria, Bacillus subtilis, Bacillus subtilis natto, Bacillus amyloliquefaciens, and Bacillus licheniformis, biotechnological strategies have been developed that employ either liquid or solidstate fermentation processes (LSF and SSF), as well as modifications in culture conditions such as media composition and carbon source, temperature, shaking speed, and time in culture [13].

IV. METABOLISM OF VITAMIN K

a. Absorption in the gastrointestinal tract.

Most dietary lipids can be absorbed through the intestinal route known as "pathway K," which includes the solubilization of dietary lipids in bile salt and pancreatic juice, the uptake of mixed micelles into enterocytes, the packaging of dietary lipids into CM, and their exocytosis into the lymphatic system [14]. The rate and degree to which a nutrient is absorbed and made available to the place of activity is known as its bioavailability [15]. The main source of phylloquinone in most diets is green leafy vegetables, which are followed by certain plant oils or fats that are rich in phylloquinone and found in a variety of food products [16]. A popular but finite technique to

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evaluate bioavailability is to compute the area under the curve (AUC) of plasma measurements made during the absorption phase.Plasma phylloquinone from raw spinach alone had a 10-hour AUC that was 4% of that of phylloquinone that had been detergent-solubilized (Konakion). On the other hand, butter added to spinach increased its relative bioavailability by three times [28]. Study, mean 9-hour AUC values from romaine lettuce, raw spinach, and cooked or raw broccoli consumed using a standard The test meals did not substantially differ from one another, ranging from 9 to 28% of that from a Konakion tablet. The fact that these two investigations were small and compared food absorption efficiency to medication formulations of phylloquinone is one of their drawbacks [17].

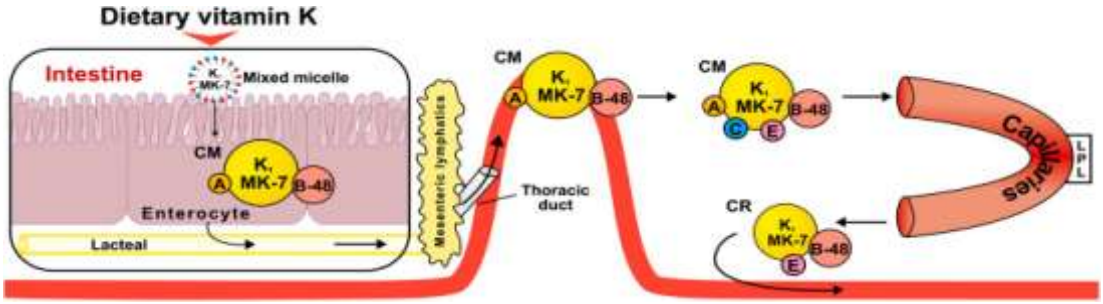

Figure 2: "Intestinal Absorption Pathways of Dietary Phylloquinone (K1) and MK-7: A Comprehensive Overview"[18].

Over the past 10 years, techniques for examining the bioavailability of tracer levels of phylloquinone using stable isotope technology have been developed [19]. This method in conjunction with Compartmental modeling is presently improving our understanding of phylloquinonone absorption, distribution, and metabolism in humans and offers clear benefits in terms of specificity and sensitivity. Specifically, the ability to label phylloquinone with deuterium or during plant growth makes it possible to analyze phylloquinone bioavailability in environments that closely mimic the average digestive process. After consuming isotopically tagged uncooked kale (with 30 g oil), compartmental analysis of the plasma distribution of labeled phylloquinone revealed a mean bioavailability of only 5% [20]. This is significantly less than ~80% absorption efficiency for free-radicalized phylloquinone that balance tests have found [21].

b. Transport in the bloodstream

Using radiolabeled phylloquinone, early research revealed that following intestinal absorption, vitamin K initially manifests in lymph before entering the bloodstream and being linked to CM [22].Subsequent studies on lipoprotein transport following the injection of stable isotope-labeled or unlabeled phylloquinone revealed that TRL was linked to most of the phylloquinone throughout the postprandial phase of absorption. Following a fat-rich meal with unlabeled phylloquinone (w100–3000 mg), plasma concentrations of the compound peaked at 6 hours, with phylloquinone making up 75–90% of the total retinoic fraction at that time [23]. After vitamins A and E were administered, plasma retinal esters showed a similar response. A number of theories were proposed to account for this relative vitamin K delay in comparison to TG. In addition to a potential delayed absorption at the intestinal absorption level, it is possible that CM undergo lipolysis in the capillary endothelium, which is facilitated by LPL, and that the resultant CR retain a significantly higher amount of their phylloquinone cargo than TG. Another theory is that the liver reabsorbs CR and resecretes it along with VLDL, which has a higher phylloquinone payload than TG [24].

c. Activation and recycling in the liver

The mechanism of dietary vitamin K's hepatic uptake has not been directly studied, but based on general fat absorption principles,it can be assumed that most vitamin K is transported to the liver in the form of CR produced during the postprandial phase of intestinal absorption. Different Apo proteins on the surface of lipoproteins, cell surface lowaffinity binding sites of HSPG, and high affinity lipoprotein receptors that mediate internalization of the lipoprotein particles are all involved in the complex absorption process[25].ApoE is primarily relevant to the transport and cellular internalization of vitamin K

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because it functions as a ligand to promote lipoproteins' high-affinity binding to the LDLR and other LDLR family members, such as the LRP and the VLDLR. The process known as secretion capture is the mechanism by which CR binds to members of the LDLR family that are present on the surface of the same target cells by acquiring apoE from HDL and from cells in their immediate surroundings. Cell surface HSPG interacts with CR to enhance its binding and subsequent liver internalization [26]. A connection with cell surface HSPG facilitates the liver's binding and subsequent internalization of CR [27].

V. BIOLOGICAL FUNCTION OF VITAMIN K IN HUMAN

a. Role of Vitamin K in Blood Coagulation:

Nameof Protein	Function
Prothrombin, factorsVII,IXandX	Haemostasis(pro-coagulantfunction)
ProteinsS,C,andZ	Haemostasis(anticoagulantfunction)
matrix Gla protein (MGP)	Inhibit arterialcalcification
Osteocalcin	Bone metabolism
Growtharrestsequence6 protein(Gas6)	Regulationofcellgrowth
Periostin	Bonemetabolism,cell migration,angiogenesis
Gla rich protein (GRP)Periostin like factorFourtransmembraneGlaprotein	Functionunknown

Table 1:The kinds and purposes of the 17 Gla-proteins[28].

b. Role in bone metabolism and mineralization

Vitamin D and K are essential for the proper metabolism of bone. Three vitamin K-dependent proteins were separated from the three components make up bone: S protein, matrix Gla protein (MGP), and osteocalcin. Osteocalcin is produced by osteoblasts, which are cells that build bone, and it plays a role in the mineralization of bone following collagen. The most important protein incorporated into the bone matrix is osteocalcin. Osteocalcin is activated by vitamin K through the carboxylation of three of its glutamic acid residues, whilst vitamin D increases the quantity of calcium and improves osteocalcin synthesis. In bones, calcium (hydroxylated calcium phosphate) will be bound and stored by this active form of osteocalcin. Bone density loss may result from inadequate osteocalcin carboxylation (caused, for example, by a vitamin K shortage) [29]. Children with hereditary protein S deficiencies have less problems with low bone density and enhanced coagulation. Because their levels of estrogen decrease after menopause, postmenopausal women are more vulnerable to osteoporosis and bone loss [30]. A large dose of phylloquinone (1000ug/day) combined with vitamin D, magnesium, calcium, and zinc over a period of three years was shown to reduce bone loss on the femoral neck but not on the lumber spine in women between the ages of 50 and 60. This effect of phylloquinone treatment on bone loss in postmenopausal women has been assessed in a number of random trails [31].

c. Potential effects on cardiovascular system

One of the primary processes leading to cardiovascular disease (CVD), the leading cause of death worldwide, is calcification of the blood arteries. It is well known that proteins dependent on vitamin K activate the defense system to stop blood vessel calcification from occurring. The activation of matrix Gla protein (MGP) by a carboxylation reliant on vitamin K is thought to potentially decrease the amount of calcium deposited on the lining of blood vessels. Additionally, a high blood concentration of inactive, undercarboxylated MGP

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is thought to be a likely sign of early-stage atherosclerosis [32]. According to a case control study, giving 500 micrograms of vitamin K1 daily for three years could postpone the onset of early coronary artery calcification in elderly individual's.In addition to its impact on MGP, vitamin K may use an anti-inflammatory mechanism to prevent blood vessel calcification. Proinflammatory cytokines like TNF-α, oncostatin M, IL-6, and IL-1β are produced when macrophages are activated during the chronic inflammatory process known as vascular calcification. These pro-inflammatory cytokines promote the estrogenic differentiation of smooth muscle cells in blood arteries. By preventing NF-kB singles transduction, vitamin K has an anti-inflammatory action and slows the calcification of blood vessels [33]. This is particularly true when elderly individuals use vitamin K antagonists to lower blood clotting [34].

d. Potential effects on immune system

In vivo studies have revealed a role for vitamin K2 in immunomodulation that was previously unknown. First, it has been demonstrated that MK-7 altered the expression of IL-1β, TNF alpha, and IL-1alpha. In addition, K2 reduces T-cell proliferation in persons in good health, while vitamin K1 remains unaffected. T-cells from a large number of children with atopic dermatitis and healthy children, as well as data from additional studies including dialysis patients, have also confirmed it [35]. K2 has been demonstrated in both experiments to decrease the quantity and growth of activated T-cells. Thus, a body of research indicates that K2 now plays a different role as an immune-suppressive substance. This needs more explanation; thus far, it's possible that vitamin K2 can increase immunomodulation through a distinct physiological mechanism, but more research is needed [36].

VI. CAUSES OF VITAMIN K DEFICIENCY

While 8% to 31% of generally healthy persons may frequently exhibit vitamin K insufficiency, bleeding that is clinically serious is rarely the result. Those who have liver disease, malabsorption disorders, or are taking drugs that affect vitamin K metabolism are more likely to experience bleeding or hemorrhage [37]. Every newborn has reduced vitamin K levels. The first accounts of classic VKDB describe a bleeding condition that manifests on the second or third day of life and date back to 1894. When sepsis-related bleeding was added, the frequency increased to 600 cases per 100,000 newborns, translating into a 62% mortality rate. Mothers who were taken anticonvulsants or other drugs that interfere with vitamin K have been associated with early VKDB. Without preventive neonatal vitamin K, the risk of early VKDB can reach 12%. The current incidence of classic VKDB is estimated to be between 0.25% and 1.7% in the absence of vitamin K therapy. Asian people have the highest frequency of late VKDB, which affects 4.4 to 72 infants per 100,000 births. Infants who are exclusively breastfed are at a higher risk of developing the condition. For late-onset VKDB, the death rate varies from 20% to 50%. Furthermore, cerebral bleeding contributes to a notable neurological morbidity rate in late VKDB[38]. Less than 30 cases of VKCFD have been documented globally, making it an uncommon autosomal recessive condition that equally affects boys and females[39].

VII. IMPACT OF DIET ON VITAMIN K STATUS

a. Dietary sources of Vitamin K1 and K2

Since vertebrates, including humans, cannot synthesize vitamin K, they must get it through food in order to meet their daily requirement. Furthermore, the body's store of vitamin K is quickly depleted in the absence of a regular diet[40]. All photosynthetic organisms, such as plants, algae, and cyanobacteria, contain vitamin K1, which is a byproduct of the shikimate pathway in the process of photosynthesis[41]. Green leafy vegetables including kale, romaine lettuce, broccoli, cabbage, and spinach are the primary dietary sources of vitamin K[42]. Moreover, fruits, grains, meat, and dairy items have smaller concentrations of K1[43]. Vitamin K1 is included in many common Japanese foods, including vegetables (raw perilla has the greatest amount at 1007μg/100 g), dried seaweed (Sargassum fusiform, hijiki, 175μg/100 g), and dry wakame (Undaria pinnatifida, 1293μg/100 g)[44].

A tissue-specific conversion from vitamin K1 in mammals can produce MK-4, however bacterial production is the primary source of vitamin K2. The UbiAprenyltransferase domain-containing 1 enzyme is responsible for catalyzing this process[45]. It has been reported that the human gut's bacterial flora produces a number of long-chain MKs. The main forms of K2 that have been detected in the human large intestine, such as MK-6, MK-7, MK-8, MK-10, and MK11, are

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produced by a variety of enterobacteria, includingVeillonella, Eubacterium lentum, Bacteroides, and Enterobacteria[46].

b. Microbial source source of Vitamin:

i. Gut Microbiota

The term "gut microbiota" describes all of the microorganisms that live in the intestines. It consists of a huge range of viruses, bacteria, fungus, Achaea, and protozoans. The phyla Firmicutes and Bacteroidetes make up the majority of a healthy gut microbiota, followed by Actinobacteria and Verrucomicrobia. These phyla don't change, although there is noticeable wide variation in the genera' qualitative and quantitative makeup in every section of the intestines. The following species are found in the gut microbiota: Fusobacteria spp., Lachnospira spp., Roseburiaspp., Butyrivibrio spp., Faecalibacterium spp., Pseudomonas spp., Rothia spp., Veillonella spp., Clostridium spp., Porphyromonas spp., Eubacterium spp., Ruminococcus spp., Enterobacter spp., Enterobacter spp., Lactobacillus spp., Peptostreptococcus spp., Fusobacteria spp., Lachnospira spp., Roseburia spp., Butyrivibrio spp., Streptococcus spp.[47].

These microorganisms are typically mutualists or commensalists, and they primarily support nutrition metabolism by fermenting carbohydrates, among other things. As a result, the short chain fatty acid butyrate is synthesized (SCFA), a crucial source of energy for colonocytes. Its anti-inflammatory and anti-cancer qualities are well-known. Lipid metabolism is also positively impacted by intestinal microbes. They improve the metabolism of proteins by the action of microbial proteinases and peptidases. Additionally, they are essential for the production of conjugated linoleic acids (CLA), vitamin K, vitamin B12, and biotin. They participate in the degradation of different polyphenols and convert bile acids [48].

ii. Other bacterial sources

Menaquinones of different chain lengths are produced by a number of other gut bacteria, including Bacteroides ovatus, Enterococcus faecalis, Escherichia coli, Prevotellabuccae, Staphylococcus epidermidis, and Staphylococcus haemolyticus. Serratia marcescens, Bacteroides spp., Citrobacter freundii, Enterobacter agglomerans, Enterococcus faecium, Staphylococcus capitis, and Staphylococcus warneriare among the bacterial strains that were isolated from the feces of infants who were exclusively fed formula and were reported to produce vitamin K [49].

VIII. HEALTH BENEFITS AND THERAPEUTIC POTENTIAL

a. Effectof vitaminKoncardiovasculardiseases

There is evidence that the prevalence of coronary artery calcium (CAC) rises with declining kidney function. In fact, it has been shown that 13% of cases with CAC prevalence 'Healthy' individuals without renal illness, patients without chronic kidney disease (40%) who are not receiving dialysis, patients beginning dialysis (57%), and patients receiving long-term dialysis (83%) [50]. Vitamin K deficient diets can hasten the onset of vitamin K deficiency in as little as seven days. Furthermore, vitamin K in subclinical form deficit is not unusual, particularly in warfarin-using patients. Higher levels of vitamin K2 (menaquinone) have been linked to a decreased risk of coronary heart disease (CHD), CHD mortality, all-cause mortality, and severe aortic calcifications, according to cross-sectional and cohort data [51]. Phylloquinone, the main dietary source of vitamin K, did not demonstrate this effect when vitamin K1 was consumed.26 27 In order to prevent arterial calcifications and/or lower the risk of eventual cardiovascular events and death, dietary vitamin K1 intake alone may not be adequate in the absence of vitamin K2. It has been suggested that vitamin K in the menaquinone form is superior to vitamin K1 in terms of preventing and treating arterial calcifications. According to undercarboxylated osteocalcin and MGP, a significant number of seemingly healthy persons may be subclinically vitamin K deficient, which would likely raise their risk of vascular calcifications, cancer, and osteoporosis [52]. Increased vascular calcifications are linked to low vitamin K status (shown by undercarboxylated (MGP), which can be raised with efficient vitamin K supplementation.28–32 For a long time, it was thought that vitamin K was only necessary for the formation of coagulation factors, or for the maintenance of hemostasis. Nevertheless, the functionality of certain additional vitamin-K-dependent proteins, such as those containing γ-carboxyglutamate or Gla, depends on vitamin-K carboxylation [53].

b. **Effects on bone health and osteoporosis**

Osteoporosis is a major factor in fractures globally, accounting for around 8.9 million fractures every year. Also, an estimated 200 million

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people worldwide suffer from osteoporosis. women globally (about 1/10th of women in their 60s, 1/5th in their 70s, 2/5th in their 80s, and 2/3rd in their 90s). Among those over 50, one in three women and one in five men will suffer an osteoporotic fracture. Furthermore, women account for 61% of all osteoporotic fractures. According to predictions, the incidence of hip fractures would rise by 240% for women and 310% for men by 2050; as a result, osteoporosis will likely have a major financial impact[54]. Research indicates that osteoporosis is more common in hospital days than diabetes, heart attacks, or breast cancer.It is also a major cause of disability, which has been demonstrated to be higher than that caused by asthma, rheumatoid arthritis, and heart disease associated to high blood pressure, as well as greater than that caused by cancer (except from lung cancer). About 20% of people die overall in the first year following a hip fracture. Being greater in males than females.In addition, men account for 20–25% of hip fractures15 and are thought to have a 30% lifetime risk of developing osteoporotic bone disease chance of getting prostate cancer[55].

c. Anti-inflammatory and antioxidant properties

A substance that can prevent or delay the oxidation of a crucial biomolecule while working at a concentration far lower than the oxidizable substrate is known as a biological antioxidant[56]. The first category of compounds that suppress the creation of free radical species affects both free radical species and their initiators, and this is where the full explanation of defense strategies is found. Antioxidants, which perform their scavenging function by electron donation, inhibiting chain start, or breaking chain propagation, make up the second line of defense. Repair antioxidants are part of the third line of defense; they take action subsequent to the onset of free radical damage. The fourth line of defense's antioxidants takes advantage of adaptive processes. A free radical species' signal directs the production of an antioxidant species at the right location [57]. Exogenous and endogenous antioxidants are taken into account in a source-based antioxidant classification [58].

Exogenous antioxidants mostly fall into two categories: vitamins, including C and E. Carotenoids, which include lutein, zeaxanthin, beta-carotene, lycopene, and others; - Phenolic antioxidants, which include flavonoids and non-flavonoids such phenolic acids, which include gallic acid, cafeic acid, chlorogenic acid, and others, or stilbene derivatives.Isoflavones (genistein, daidzein, glycitein), flavonols (quercetin, kaempferol, myricetin), flavanones (naringenin, eriodictyol, hesperetin), flavones (luteolin, apigenin), and flavonols (proanthocyanidins, catechins). Trace elements zinc and selenium are known as anthocyanins. Anthocyanidins (malvidin, cyanidin, and pelagonidin) are flavone cations that have a flavylium cation as their main structural component [59].

IX. RECENT RESEARCH

The makeup and distribution of vitamin K metabolites in organs other than the liver are beginning to show similarities. The comparable conversion of phylloquinone and other menaquinones, both separately and together, to MK4 in nonhepatic organs, such as the brain, was found using stable isotopes in animal models.It has been determined that UbiAprenyltransferase domain containing 1 (UBIAD1) is the only enzyme in charge of converting different quinones to MK4 through the application of CRISPR/Cas9 (clustered regularly interspaced short palindromic repeats/CRISPR-associated protein 9) technology [60]. Mice lacking UBIAD1 do not survive because UBIAD1 is an essential enzyme in the route leading to the manufacture of cholesterol. Though MK4 is involved in unrelated physiological roles that affect these animals' ability to thrive, these UBIAD1-deficient mice do not exhibit overt signs of vitamin K deficiency. This has been interpreted as a sign that these animals obtain enough vitamin K to support carboxylation of the hepatic vitamin K–dependent coagulation protein. An exceptional chance to clarify the functions of MK4 other than the carboxylation of vitamin K-dependent proteins is provided by the UBAID1-deficient mouse model [61].

X. CONCLUSION

In conclusion, vitamin K plays a critical role in supporting various aspects of health, including bone health, blood clotting, cardiovascular function, and potentially cognitive function. Ensuring an adequate intake of vitamin K through a balanced diet rich in vitamin K sources is essential for maintaining optimal health and reducing the risk of associated health conditions. Further research into the mechanisms of action and benefits of vitamin K may provide valuable

insights for improving health outcomes and developing targeted interventions in the future.

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