Articles on K2 Posted by Vitalady

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While I am not any kind of authority on Vitamin K, my summary of the hundreds of studies I've read are summarized here. It is a fat soluble vitamin, usually included with A, D and E. Taken in dietary form (greens), it normally converts to K2 while in the INTACT intestine. As with A, D and E, people who have any form of malabsorption are missing "the converter" for K, as well.

* K2 decalcifies the blood and the extracted calcium ends up in the bones
* K2 as MK-4 (synthetic) "might" interfere with blood thinners, like Coumadin
* K2 as MK-7 is safe in doses as high as 45mg (most is sold in mcg), even for those using blood thinners
* K1 in dietary form cannot convert to K2 with any tampering of intestines, at all
* Phytonadione and Phylloquinone appear to be used interchangeably for K1

* My personal conclusion is that I will use the K1 1,000 mcg once daily for myself and Don AND the K2 (as MK-7) starting at a lower dose of 50mcg twice per day, gradually increasing dosage.
* [comments in brackets below are my own]

Vitamin K, and the Difference Between K1 and K2

Ask doctors what vitamin K does, and most will tell you it is involved in the clotting process…period!

As early as 1984, however, scientists reported that patients who suffered fractures caused by osteoporosis had vitamin K levels that were 70% lower than age-matched controls. These findings were confirmed in later studies showing diminished bone mineral density in the presence of low serum vitamin K levels. The most frightening statistic showed that women with the lowest blood levels of vitamin K had a 65% greater risk of suffering a hip fracture compared to those with the highest vitamin K levels.

One might wonder why vitamin K has such a powerful impact on bone density. The answer is quite simple. In order for calcium to bind to the bone matrix, a protein called osteocalcin is needed. Without adequate vitamin K, osteocalcin is unable to transport calcium from the blood and connect it to the bones. Tens of millions of Americans ingest calcium supplements to reduce their risk of osteoporosis and fractures. These vitamin K studies, however, show that people could still suffer the crippling effects of osteoporosis if they are vitamin K deficient because the calcium would remain in their blood and not bind to bone.

The next question is what happens to calcium if it is not taken up to form bone mass. Regrettably, in a vitamin K deficient state, the body takes calcium that is meant to form strong bones and instead deposits it into the arterial wall, thereby contributing to the process of atherosclerosis. In fact, in response to a vitamin K deficiency, the body naturally accumulates

enormous amounts of calcium in the.

This explains why so many aging individuals suffer from hardened calcified arteries, yet have brittle bones that are markedly depleted of calcium. In a huge European human clinical trial (The Rotterdam Study), doctors evaluated vitamin K intake of 4807 subjects over a 7-10 year period. After adjusting for other risk factors, coronary heart disease risk was reduced with increased intake of vitamin K2. Those who consumed the most vitamin K2 had a 57% reduction in cardiac disease compared to those who consumed the least K2.What has scientists most excited is that vitamin K2 is proving itself to be superior to K1. Health enthusiasts will be pleased to learn that a new biologically-active form of vitamin K2 has been added to the supplement formulas used by most Foundation members today.

Vitamin K1 is obtained in the diet primarily from dark leafy vegetables (lettuce, spinach, and broccoli). Unfortunately, vitamin K1 is tightly bound to the chlorophyll in green plants, meaning that aging humans are not able to benefit much from ingested K1-containing plants.10 While vitamin K1 is not absorbed particularly well from food, it is absorbed from supplements, provided that the supplements are taken with fat-containing meals.

Vitamin K2 is found in much smaller quantities in the diet, primarily in dairy products. The highest level of dietary K2 is fermented soy natto.14 Human studies show that vitamin K2 is absorbed up to ten times more than K1.15 Japanese people consume large quantities of natto, which may help explain their lower rates of heart disease and osteoporosis compared to Western populations.

Not only is K2 absorbed better, but it remains biologically active in the body far more than K1. For instance, K1 is rapidly cleared by the liver within 8 hours, whereas measurable levels of K2 have been detected 72 hours after ingestion. This means that vitamin K2 is available to facilitate transport of calcium into the bone and to protect the arterial wall against calcification much longer than is K1.

Vitamin K Impedes Atherosclerosis

In rabbits with high cholesterol levels, supplemental vitamin K2 decreased circulating cholesterol, suppressed the progression of atherosclerotic plaque, and impeded the thickening of the inner arterial lining (intima). In a rat model, supplemental vitamin K2 completely prevented calcification, whereas vitamin K1 had little effect.

In a study of 188 postmenopausal women, a group known to be at high risk for rapid decay of arterial structure, a supplement containing only 1 mg of vitamin K1 or a placebo was administered over a three-year period. In the vitamin K group, age-related arterial stiffening (as measured by carotid intima-media thickness) was completely abolished, whereas the placebo group (not receiving vitamin K) experienced a 13% worsening of arterial elasticity during the study period.18 (Please note that some vitamin K1 is converted to K2 in the intestine).

How Vitamin K Protects Arteries from Calcification

Vitamin K controls calcium-regulating proteins that are present in vascular tissue. These vitamin K-dependent proteins (including osteocalcin and matrix G1a protein) have been shown to specifically inhibit vascular calcification, i.e. keep calcium out of the arteries.

Activation of these calcium-regulating proteins depends on the availability of vitamin K. When there is not enough vitamin K to turn on these proteins, the result is deposition of calcium into atherosclerotic plaque, thus worsening cardiovascular disease risk and leading to a condition that the lay public sometimes refers to as “hardening of the arteries.” This helps explain why patients who take anti-coagulant drugs (like Coumadin® that deplete vitamin K in the body suffer from accelerated atherosclerosis.

Increasing evidence shows that the same calcification process involved in normal bone formation also occurs in the linings of arteries when there is not enough vitamin K available to activate calcium-regulating proteins (such as matrix G1a protein, a powerful inhibitor of arterial calcification). This means that the same biological mechanism used by bone to attract and bind calcium can also pathologically occur in the linings of the arteries in the presence of inadequate vitamin K.8 This explains why patients with advanced atherosclerosis have both occluded and calcified (hardened) arteries that have lost their youthful elasticity. The inability of arteries to readily expand and contract with each heartbeat is a hallmark characteristic of hypertension.

In most individuals, vitamin K from dietary sources fills the need for proper coagulation. As people age, however, a sub-clinical vitamin K deficiency can pose severe risks to the vascular system.

Vitamin K2 Intake Associated with Reduced Arterial Disease

In the most significant human study to date, a large group of people with no history of heart disease were followed from 1990-1993 until year 2000. The incidence of coronary artery disease, all cause mortality, and severe aortic atherosclerosis was studied in relationship to the amount of vitamin K1 and/or K2 ingested over the study period.

As can clearly be seen by the table above, those who consumed the most vitamin K2 showed significant disease reductions, compared to those who ingested the least K2. In this study, intake of vitamin K1 from dietary sources was not related to these risk reductions, probably due to the poor bioavailability of K1 from plant foods. In their concluding remarks, the scientists who conducted this study stated that adequate intake of vitamin K2 could contribute to the prevention of coronary artery heart disease.

Vitamin K and Bone Fracture Prevention

A systemic review was made of all randomized controlled trials that gave adults either vitamin K1 or K2 supplements for at least six months. A total of 13 trials were identified with data on

bone loss and 7 trials that reported fracture incidences. All of these human trials except one showed that supplemental vitamin K1 or K2 reduced bone mass loss. Vitamin K2 in particular was associated with increased bone mineral density.

In all 7 trials to evaluate fracture risk, vitamin K2 proved most effective, reducing the risk of vertebral fractures by 60%, hip fractures by 77%, and an astounding reduction for all non-vertebral fractures of 81%.

For those taking calcium and vitamin D supplements to prevent osteoporosis, adequate vitamin K intake is essential to activating proteins that bind calcium to the bone. In the presence of inadequate vitamin K status, the calcium that would be used to maintain strong bones is instead deposited into the arterial wall where it accelerates the atherosclerosis process.

Summary

Since vitamin K was discovered in 1930, it was only thought to contribute to the liver's maintenance of healthy blood coagulation. Over the past 15 years, scientists have found that vitamin K plays a crucial role in arterial and bone health.

Recent studies indicate that vitamin K intake that is substantially above the government's recommended reference range can slow bone loss, reduce arterial stiffening, prevent heart attack, and reduce death rates in adult human populations.

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Conversion of Phylloquinone (Vitamin K1) into Menaquinone-4 (Vitamin K2) in
Mice

TWO POSSIBLE ROUTES FOR MENAQUINONE-4 ACCUMULATION IN CEREBRA OF
MICE*

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There are two forms of naturally occurring vitamin K, phylloquinone and the menaquinones. Phylloquinone (vitamin K1) is a major type (>90%) of dietary vitamin K, but its concentrations in animal tissues are remarkably low compared with those of the menaquinones, especially menaquinone-4 (vitamin K2), the major form (>90%) of vitamin K in tissues. Despite this great difference, the origin of tissue menaquinone-4 has yet to be exclusively defined. It is postulated that phylloquinone is converted into menaquinone-4 and accumulates in extrahepatic tissues. To clarify this, phylloquinone with a deuterium-labeled 2-methyl-1,4-naphthoquinone ring was given orally to mice, and cerebra were collected for D NMR and liquid chromatography-tandem mass spectrometry analyses. We identified the labeled menaquinone-4 that was converted from the given phylloquinone, and this conversion occurred following an oral or enteral administration, but not parenteral or intracerebroventricular administration. By the oral route, the phylloquinone with the deuterium-labeled side chain in addition to the labeled 2-methyl-1,4-naphthoquinone was clearly converted into a labeled menaquinone-4 with a non-deuterium-labeled side chain, implying that phylloquinone was converted into menaquinone-4 via integral side-chain removal. The conversion also occurred in cerebral slice cultures and primary cultures. Deuterium-labeled menadione was consistently converted into the labeled menaquinone-4 with all of the administration routes and the culture conditions tested. Our results suggest that cerebral menaquinone-4 originates from phylloquinone intake and that there are two routes of accumulation, one is the release of menadione from phylloquinone in the intestine followed by the prenylation of menadione into menaquinone-4 in tissues, and another is cleavage and prenylation within the cerebrum.

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**********http://sciencelinks.jp/j-east/article/200304/000020030403A0074675.php
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Clarification of the conversion mechanism of vitamin K analogues to menaquinone-4(MK-4) in the rats tissues without the participation of bacterial enzyme in the intestine.

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Abstract;Phylloquinone(vitamin K1=VK1) and the menaquinones(MK-n, or vitamin K2=VK2) are naturally occurring forms of vitamin K. Most of the menaquinone series are synthesized by microorganisms, but we have reported that MK-4 is usual in being synthesized by the conversion of orally ingested VK1 or menadione(=VK3) in the major tissues of germfree rats and mice which lack their intestinal microflora. This result denies Martius' theory that described the participation of bacterial enzyme of the intestinal flora to this conversion. Recently MK-4 has been attracted the attention of researchers due to its specific physiological action such as apoptotic activity on osteoclast cells and leukemia cells, etc. The present study was undertaken to clarify the in vivo conversion of VK1,VK3,MK-6,MK-7, and MK-10 to MK-4 in Wistar rats, and also to clarify the mechanism of the conversion of VK3 or VK1 to MK-4 in tissue homogenate system(in vitro) of various organs in germfree or conventional Wistar rats, and the latter part of the study was summarized here. In vivo conversion studies revealed that the increase of MK-4 level in various tissues of Wistar rats after VK1,VK3,MK-6,MK-7, and MK-10 administration orally. In vitro conversion studies revealed that there was the enzyme activity of MK-4 formation

from VK1 and GGPP in various tissues, and no or very low activity of MK-4 formation in the gastrointestinal mucosa in germfree rats. Results obtained from the experiment using [8-ring]-14C-VK1 substrate indicate that the VK analogues such as reduced-form of VK3,VK1, and MK-4 could be candidates for intermediate products for final MK-4 formation. (author abst.)
**********http://www.healthsalon.org/213/vitamin-k-arterial-calcification-and-atherosclerosis/
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Vitamin K, Arterial Calcification and Atherosclerosis

17th April 2007 by Rett Anderson Posted in Disease <http://www.healthsalon.org/category/disease/> , Cardiovascular <http://www.healthsalon.org/category/cardiovascular/> , Nutrition <http://www.healthsalon.org/category/nutrition/>

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Researchers at the University of Maastricht in the Netherlands recently published the results of an experiment in the journal Blood showing that vitamin K deficiency causes arterial calcification and the death of the smooth muscle cells that line the blood vessel wall. They have shown in previous research that atherosclerosis is characterized by deposits of calcium salts, lipids, white blood cells, and deformed vitamin K-dependent proteins manufactured by cells that do not have enough vitamin K to meet their needs. These deposits are not random — they aggregate around the remnants of dead (”apoptotic”) smooth muscle cells.Vitamin K supports at least two proteins that are likely to protect against atherosclerosis: matrix Gla protein (MGP) and growth arrest specific gene product 6 (Gas6). MGP protects soft tissues from calcification, while Gas6 increases cell survival and helps clear away any fragments left behind by cells that do happen to die.

In this experiment, the researchers induced a vitamin K deficiency in Wistar Kyoto rats with Warfarin, an oral anticoagulant that works by inhibiting the recycling of vitamin K. Warfarin caused arterial calcification, a marked increase in the death of smooth muscle cells, a loss of blood vessel elasticity, and a decreased ability of the blood vessels to accommodate changes in pressure. High-dose vitamin K supplementation reversed these changes even after they were induced by Warfarin. Low-dose vitamin K supplementation not only did nothing to reverse the effects of warfarin but the calcification actually continued at the same rate in the animals that received the drug after it was discontinued, persisting for the entire duration of the study, showing that the vitamin K deficiency induced by oral anticoagulants persists long after their use is discontinued.

In this study and in an earlier one that the same researchers published, vitamin K1, found in plants, had no ability whatsoever to prevent warfarin-induced calcification when the drug and the vitamin were administered together, while vitamin K2, found in animal foods and fermented

plant foods, completely inhibited warfarin-induced calcification. The researchers found, however, that after warfarin was discontinued, both vitamins were equally effective at reversing calcification.

The Wistar Kyoto rats that these researchers used convert vitamin K1 to vitamin K2 with great efficiency. Warfarin not only inhibits the recycling of vitamin K, but also the conversion of vitamin K1 to vitamin K2. Although it is not clear why this would be the case and the findings should therefore be treated with caution, the results strongly suggest that only vitamin K2 protects against heart disease, and that vitamin K1 is effective only insofar as it is converted to vitamin K2. How well humans make this conversion compared to rats is unknown. In The Rotterdam Study, intakes of vitamin K2 showed a powerful inverse association between calcification of the aorta, heart disease, and heart disease mortality. Intakes of vitamin K1 by contrast — even though they were ten times higher than intakes of vitamin K2 — had no relationship to any of these endpoints at all.

Schurgers LJ, Spronk HMH, Soute BAM, Schiffers PM, DeMey JGR, Vermeer C. Regression of warfarin-induced medial alastocalcinosis by high intake of vitamin K in rats. Blood. 2006; [Epub ahead of print].

Remember, vitamin D uses vitamin K2, so you need to make sure you get enough of both.
**********http://www.menaq7.com/index.php?s=Links
or
http://tinyurl.com/5c3rwy

There are two forms of vitamin K2 available as supplements, Natural Vitamin K2 as Menaquinone-7, (MK-7) and Synthetic Menaquinone-4 (Mk-4). There are significant differences between these two forms of vitamin K2.

<http://www.menaq7.com/img/wykres_top.gif>
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* A single dose of 1mg of Synthetic MK-4 and Natural MK-7 were given to healthy volunteers
* The maximum absortion levels in serum were found at 2 hours for MK4 and 4 hours for MK-7; MK-4 is excreted in approximately 8 hours
* While peak heights are comparable, MK-7 demonstrates a much longer half-life in the blood, thus available to the body 24h/day with daily dosing

<http://www.menaq7.com/img/contact_bot.gif>

Synthetic vitamin K2 as menatetrenone (MK-4)

* Quickly appears but also disappears from the blood. Half life 1 hour! (see graph)
* As a result, high pharmacological doses (mg) are necessary; and multiple daily doses are

needed
* Importantly, large doses interact negatively with persons on blood-thinning medications (warfarin) – very dangerous and medical supervision is necessary

Natural Vitamin K2 as Menaquinone-7 (MK-7) from Natto

* Highly bioavailable, highly bioactive and longest serum half life (Schurgers, et al. Blood 2007). Half life 3 days!
* Long half life will result in building up a buffer of MK-7, and supplies MK-7 to ALL tissues 24 hours a day.
* More effective at doses that do not exceed the present recommendations for daily vitamin K intake
* At 45mcg/daily, provides levels shown to be the highest consumption in the Rotterdam study
* Not likely to interact negatively with blood-thinning medications (warfarin) at 45 mcg/day!

Summary: VitaK research at the University of Maastricht, experts in vitamin K for more than 30 years, studied the bioavailability and bioactivity of the three K-vitamins (K1, MK-4 & MK-7) currently available for human consumption, both in supplements and in functional foods. They have clearly demonstrated at regular intake (once daily) of nutritional doses (20-150 mcg/day) of MK-7, its long half-life may lead to accumulation in blood and extra-hepatic tissues (e.g. bone and vasculature) to levels that may only be reached with more frequent supplementation (e.g. three times a day) and much higher doses of either K1 or MK-4.

Clarification of MK-4 Claims: True or False?

Claim: MK4 is the superior form of Vitamin K2.

* MK-4 bioavailability was shown to be at least 4 times greater than MK-7 in bone

This point is not true: the study they refer to (Yamaguchi et al., 1999) was performed by giving first high dose of either MK-4 or MK-7. In the high dose, both vitamins accumulated equally well in plasma and bone. All the studies currently performed with MK-4 are done with extremely high doses. Nothing is known about the “normal” physiological intake of MK-4. Next, they studied the accumulation of low dose of MK-7 (combination of natural natto + extra “pure” MK-7) in plasma and bone. THEY DID NOT STUDY LOW DOSE OF MK-4. They found that MK-7 accumulated very well in the plasma (what we see in the human study being published in Blood April 2007). However, they found that MK-4 was the result in bone. This is due to the conversion of MK-7 into MK-4 (same as the conversion of K-1 into MK-4). The carboxylation (activation) of osteocalcin was good in case of the low MK-7. When the conversion and why and how the conversion of MK-7 (or K-1 or even MK-4.) into MK-4 is taken place, nobody knows. We have, however, human nutritional data in the normal range from Hodges et al (1993, JBMR) who describe that in the bones K-1 and MK-7, 8, &9 accumulate to

the same extent, but not MK-4. Recently, Dr. Sato and colleagues have performed a rat-study, submitted recently to a Japanese journal in which he proves that at the low dose ONLY MK-7 increase vitamin K2 in the bone, whereas MK-4 does NOT. Also in this case he finds that some conversion of MK-7 into MK-4 takes place (thus he only finds MK-4 after MK-7 intake whereas he does not find MK-4 after MK-4 intake in the bone...). Taken together, we have to conclude that at the aimed physiological range MK-7 is the most potent K-vitamin.

* Most of the published human studies supporting Vitamin K2's role in bone health and vascular health used MK-4

This is absolutely true: However, even more evidence is present on K-1. This is only due to the following reason: First K-1 was discovered and all the research focused on K-1. Later, MK-4 was discovered and even more importantly PRESENT as synthetic compound and therefore most of the K-2 research was done with MK-4. Behind this, of course the industry was a major driving factor: the company which produces MK-4 is Eisai Co., Tokyo, Japan and they sponsored most of the MK-4 studies. Only since the last decade, MK-7 as natural product from Natto was recognized as being important for vascular health and bone health. The limited data available, however, point towards MK-7 as most potent K-vitamin.

* MK-4 is the form the body creates from plant-source Vitamin K1

This is only partly true. MK-4 is formed out of K-1, but also out of MK-7 and even out of MK-4 itself. Why, how, and where is not known. Therefore, we cannot draw any conclusions from this. Furthermore, no evidence is available if the converted MK-4 (out of plant derived K-1) has an effect on bone health or vascular health. Additionally, if the MK-4 out of the plant derived K-1 would have an effect, all the data demonstrating that K2 is better than K-1 would not be true since K-1 also creates MK-4. This is definitely NOT the case since we find major advantages of K-2 over K-1.

* Vitamin K2 MK-4 is the truly natural form of Vitamin K-2. It is the form your body uses.

Not true: All forms of vitamin K are used by the body: K-1, and all K-2 vitamins. Moreover, the definition of a vitamin is that it needs to be taken by the food because the body cannot produce it. The MK-4 we receive is via the food (at least with the present knowledge we have, nobody knows what the contribution is of the converted MK-4 out of K-1), and this is via the food products meat and eggs. The chickens and cows receive the highly toxic vitamin K3 (menadion, not allowed for human consumption.) and they convert MK-4 out of this K3. It accumulates in the meat and in the eggs, and this is the food source of MK-4. As mentioned above, the low range intake of MK-4 has never been investigated, and the contribution to the vitamin K status is rather questionable.

Further, the only supplemental form of MK-4 is the synthetic Menatetrenone. Menatetrenone at high doses poses a risk for the general population because any form of supplemental vitamin K daily at a dose over 100-150mcg will impact those using the common K-antagonist blood

thinning medications, including coumarin / warfarin. Rather, menaquinone-7 at 45mcg daily is recommended to optimally activate vitamin K dependent proteins at levels well below this threshold.

* MK-4 benefits bone strength and protects against cardiovascular calcification

Yes, this is correct, but only is true for the high doses: 45 mg in humans, or very high doses in animals. No physiological dose of MK-4 has demonstrated a beneficial effect on bone health or cardiovascular health whereas this has been shown for MK-7: the Rotterdam study was mainly based on 45mcg of the long-chain MKs (MK-7, 8, 9) and also the beneficial effect of natto (= MK-7) on bone health has been accepted.

* XXX Company's Mk-4 provides a stable Vitamin K2 MK-4 in a protected beadlet.

True, but not necessary: all K-vitamins are very stable and only degrade in the UV light or under alkaline conditions.
**********http://www.truestarhealth.com/Notes/3260001.html
or
http://tinyurl.com/3oj6k8

[meds that interact with Vit K]

Vitamin K

Also indexed as: Phylloquinone, Phytonadione

Drug Interactions

Certain medicines interact with vitamin K
<http://www.truestarhealth.com/Notes/2932003.html> : Some interactions may increase the need for vitamin K (Beneficial <http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>), other interactions may be negative (Avoid <http://www.truestarhealth.com/Notes/Images/dnicon_Avoid.gif>) and indicate vitamin K should not be taken without first speaking with your physician or pharmacist, others may require further explanation (Check <http://www.truestarhealth.com/Notes/Images/dnicon_Check.gif>). Refer to the individual drug article for specific details about an interaction.

Note: The following list only includes the generic or class name of a medicine. To find a specific brand name, use the Medicines <http://www.truestarhealth.com/Notes/2411003.html> index.

Aminoglycoside <http://www.truestarhealth.com/Notes/1312007.html>
Antibiotics Beneficial <http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Amoxicillan–Potassium <http://www.truestarhealth.com/Notes/4496006.html>
Clavulanate Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Amoxicillin <http://www.truestarhealth.com/Notes/1315009.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Ampicillin <http://www.truestarhealth.com/Notes/1317001.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Antibiotics <http://www.truestarhealth.com/Notes/1081002.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Anticonvulsants <http://www.truestarhealth.com/Notes/1082009.html>
Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Azithromycin <http://www.truestarhealth.com/Notes/1094000.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Bile Acid <http://www.truestarhealth.com/Notes/1322001.html> Sequestrants
Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Cephalosporins <http://www.truestarhealth.com/Notes/1344006.html>
Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Chlorhexidine <http://www.truestarhealth.com/Notes/1104006.html>
Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Ciprofloxacin <http://www.truestarhealth.com/Notes/1109001.html>
Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Clarithromycin <http://www.truestarhealth.com/Notes/1113008.html>
Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Clindamycin Oral <http://www.truestarhealth.com/Notes/1115001.html>
Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Clindamycin Topical <http://www.truestarhealth.com/Notes/1116000.html>
Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Colestipol <http://www.truestarhealth.com/Notes/1125006.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Cycloserine <http://www.truestarhealth.com/Notes/1128008.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Dapsone <http://www.truestarhealth.com/Notes/1131009.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Dicloxacillin <http://www.truestarhealth.com/Notes/1351002.html>
Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Doxycycline <http://www.truestarhealth.com/Notes/1366004.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Erythromycin <http://www.truestarhealth.com/Notes/1134001.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Gabapentin <http://www.truestarhealth.com/Notes/1390003.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Gentamicin <http://www.truestarhealth.com/Notes/1392006.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Isoniazid <http://www.truestarhealth.com/Notes/1404000.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Levofloxacin <http://www.truestarhealth.com/Notes/1419004.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Loracarbef <http://www.truestarhealth.com/Notes/1426004.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Macrolides <http://www.truestarhealth.com/Notes/1430001.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Mineral Oil <http://www.truestarhealth.com/Notes/1156003.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Minocycline <http://www.truestarhealth.com/Notes/1157007.html> Beneficial <http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Neomycin <http://www.truestarhealth.com/Notes/1442006.html> Beneficial <http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Nitrofurantoin <http://www.truestarhealth.com/Notes/1445008.html> Beneficial <http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Ofloxacin <http://www.truestarhealth.com/Notes/1263005.html> Check <http://www.truestarhealth.com/Notes/Images/dnicon_Check.gif>

Oral Corticosteroids <http://www.truestarhealth.com/Notes/1346008.html> Check <http://www.truestarhealth.com/Notes/Images/dnicon_Check.gif>

Penicillin V <http://www.truestarhealth.com/Notes/1454006.html> Beneficial <http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Penicillins <http://www.truestarhealth.com/Notes/1455007.html> Beneficial <http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Phenobarbital <http://www.truestarhealth.com/Notes/1460006.html> Beneficial <http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Quinolones <http://www.truestarhealth.com/Notes/1478001.html> Beneficial <http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Sulfamethoxazole <http://www.truestarhealth.com/Notes/1503009.html> Beneficial <http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Sulfasalazine <http://www.truestarhealth.com/Notes/1504003.html> Beneficial <http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Sulfonamides <http://www.truestarhealth.com/Notes/1505002.html> Beneficial <http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Tetracycline <http://www.truestarhealth.com/Notes/1514007.html> Beneficial <http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Tetracyclines <http://www.truestarhealth.com/Notes/1515008.html>
Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Tobramycin <http://www.truestarhealth.com/Notes/1522000.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Trimethoprim <http://www.truestarhealth.com/Notes/1530004.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Trimethoprim/Sulfamethoxazole
<http://www.truestarhealth.com/Notes/1531000.html> Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Valproic Acid <http://www.truestarhealth.com/Notes/1535009.html>
Beneficial
<http://www.truestarhealth.com/Notes/Images/dnicon_Beneficial.gif>

Warfarin <http://www.truestarhealth.com/Notes/1539003.html> Check
<http://www.truestarhealth.com/Notes/Images/dnicon_Check.gif>
**********http://www.nutriherb.net/vitamin_K.html
or
http://tinyurl.com/6x49dg

Vitamin K

Also Known as: Phylloquinone, Phytonadione, Menadiol

Vitamin K Facts
Vitamin K is a fat-soluable vitamin that is stored in the liver in minute amounts. It derives its name from the term "koagulation vitamin" due to its role in blood clotting. Natural forms of this vitamin come from the chlorophyll in plants that give them their green color. The body does not store Vitamin K in large amounts, but deficiencies are rare among healthy individuals.

How Vitamin K Works
Vitamin K helps the body transport calcium, to be utilized for bone formation and normal blood clotting. It is responsible for setting in motion the blood clotting process as soon as a wound occurs.

Possible Benefits
* Reduces hemorrhaging risks and protects against bleeding after surgery
* Useful treatment for osteoporosis; decreases risk of fractures
* Aids normal liver functioning
* May help with cancer prevention

* Radiation therapy support
* Aids vitality and longevity
* May reduce LDL cholesterol and reduce build-up of arterial plaque
* May help with excessive menstrual bleeding
* May reduce LDL cholesterol and reduce build-up of arterial plaque
* Helpful with morning sickness
* May aid in Chrohn's disease and Cystic Fibrosis

Usage Guidelines
RDA for Vitamin K is 80 mcg per day. Common dosages found in multivitamins range from 65 - 80 mcg. Take with meals to enhance absorption. Side effects are rare.

Some Natural Sources
Spinach, kale, collards, broccoli, swiss chard, turnip greens, spring onions, brussel sprouts, alfalfa, kelp, liver, soybeans, pistachios, soybean oil, olive oil, cottonseed oil, canola oil, safflower oil, fish liver oils, and in lesser amounts - dairy products, fruits, cereals, meats, and eggs
**********[use of vitamin K + D3 for 2 bone density studies below shows positive result of this combo. Please note that the low doses mentioned here are for people whose intestinal arrangement is still in original condition and would not apply to malabsorptive people]

http://www.druglib.com/abstract/sc/schaafsma-a_eur-j-clin-nutr_20000800.html
or
http://tinyurl.com/6dgjvr

Vitamin D(3) and vitamin K(1) supplementation of Dutch postmenopausal women with normal and low bone mineral densities: effects on serum 25-hydroxyvitamin D and carboxylated osteocalcin.

Author(s): Schaafsma A, Muskiet FA, Storm H, Hofstede GJ, Pakan I, Van der Veer E

Affiliation(s): Department of Research & Development Leeuwarden, Friesland Coberco Dairy Foods, Leeuwarden, The Netherlands. SchaafsA@FDF.NL

Publication date & source: 2000-08, Eur J Clin Nutr., 54(8):626-31.

Publication type: Clinical Trial; Randomized Controlled Trial

OBJECTIVE: Improvement of vitamin D and K status of about 60 -y-old postmenopausal Dutch women.
DESIGN: In a randomized study postmenopausal women with normal (T-score >-1; n=96) and low (T-score< or =-1; n=45) bone mineral density (BMD) of the lumbar spine, were supplemented with 350-400 IU vitamin D(3), 80 microg vitamins K(1) vitamins K(1)+D(3), or

placebo for 1 y. Serum 25-hydroxyvitamin D [25(OH)D] and percentage carboxylated osteocalcin (%carbOC) were measured at baseline and after 3, 6 and 12 months. RESULTS: Baseline %carbOC of the entire study population was positively correlated with BMD of the lumbar spine and femoral neck. Correspondingly, women with low BMD had lower %carbOC at baseline than women with normal BMD but this difference disappeared after 1 y of supplementationwith vitamin K(1) ((mean+/-s.d.) 68+/-11% (95% CI, 64. 5-71.2%) vs 72+/-6% (95% CI, 70.1-72.9%), respectively). One year of supplementation with vitamin D(3) showed maximum increases in 25(OH)D of 33+/-29% (95% CI, 24.8-41.8%) and 68+/-58% (95% CI, 50.1-84.6%) in women with normal and low BMD, respectively. During winter, however, a 29% decline in maximum 25(OH)D levels was not prevented in women with low BMD. CONCLUSION: Daily supplementation of Dutch postmenopausal women with >400 IU vitamin D(3) is indicated to prevent a winter decline in 25(OH)D and to control serum parathyroid hormone levels. Daily supplementation with 80 microg vitamin K(1) seems to be necessary to reach premenopausal %carbOC levels. A stimulatory effect of calcium and/or vitamin D on %carbOC cannot be excluded. European Journal of Clinical Nutrition (2000) 54, 626-631.
**********[specifically K2 in this study]

http://www.druglib.com/abstract/ad/adams-j_am-j-health-syst-pharm_20050801.html
or
http://tinyurl.com/5z2bcx

Vitamin K in the treatment and prevention of osteoporosis and arterial calcification.

Author(s): Adams J, Pepping J

Affiliation(s): Castle Medical Center, Kailua, HI 96814, USA.

Publication date & source: 2005-08-01, Am J Health Syst Pharm., 62(15):1574-81.

Publication type: Review

PURPOSE: The role of vitamin K in the prevention and treatment of osteoporosis and arterial calcification is examined. SUMMARY: Vitamin K is essential for the activation of vitamin K-dependent proteins, which are involved not only in blood coagulation but in bone metabolism and the inhibition of arterial calcification. In humans, vitamin K is primarily a cofactor in the enzymatic reaction that converts glutamate residues into gamma-carboxyglutamate residues in vitamin K-dependent proteins. Numerous studies have demonstrated the importance of vitamin K in bone health. The results of recent studies have suggested that concurrent use of menaquinone and vitamin D may substantially reduce bone loss. Menaquinone was also found to have a synergistic effect when administered with hormone therapy. Several epidemiologic and intervention studies have found that vitamin K deficiency causes reductions in bone mineral density and increases the risk of fractures. Arterial calcification is an active, cell-controlled process that shares many similarities with bone metabolism. Concurrent arterial calcification and osteoporosis have been called the "calcification paradox" and occur frequently in

postmenopausal women. The results of two dose-response studies have indicated that the amount of vitamin K needed for optimal gamma-carboxylation of osteocalcin is significantly higher than what is provided through diet alone and that current dosage recommendations should be increased to optimize bone mineralization. Few adverse effects have been reported from oral vitamin K. CONCLUSION: Phytonadione and menaquinone may be effective for the prevention and treatment of osteoporosis and arterial calcification.
**********http://www.nowfoods.com/?action=itemdetail
<http://www.nowfoods.com/?action=itemdetail&item_id=73282> &item_id=73282
or
http://tinyurl.com/5av34r

Vitamin K1, Phytonadione

By Nilesh Patel, NOW Quality Assurance Dept.

Vitamins are, by definition, essential for human growth and health. They are usually found in the foods we eat. Vitamin K is necessary for normal clotting of the blood. Although Vitamin K deficiencies are rare, they can lead to problems with blood clotting and excessive bleeding.

Fat-soluble vitamins [for example: Vitamin A (trans-Retinol), Vitamin D (Calciol), Vitamin E (á-tocopherol), and Vitamin K (Phylloquinone)] are all polyprenyl compounds (similar to steroids) synthesized from a five-carbon molecule (isoprene). Phylloquinone ($C_{31}H_{46}O_2$), the most common form of Vitamin K, is found in plants as a photosynthetic electron carrier (mostly isolated from alfalfa, commercially), and is the preparation of choice marketed under the generic name of phytonadione. In other words, Phylloquinone is also called phytonadione; both terms refer to the fat-soluble natural Vitamin K (Vitamin K1) used for nutritional supplementation.

(Technical note: The term Vitamin K refers to a group of 2-methyl-1,4-napthoquinone derivatives which can fulfill an essential co-factor function in humans, aiding in the biosynthesis of a number of calcium-binding proteins, some of which are essential for homeostasis
(normal biological equilibrium). The natural forms are substituted in position 3 with an alkyl side chain. Vitamin K1 (phylloquinone) has a phytyl side chain in position 3, where as Vitamin K2 has an isoprenyl side chain at position 3.)

Vitamin K is essential since the 1,4-napthoquinone compound cannot be synthesized in the body. In nature, Vitamin K occurs as phylloquinone in plants and as menaquinones produced by bacteria, another significant source. There are three notable forms of Vitamin K:

K1 (phytonadione/ phylloquinone/ phytonactone);

K2 (menaquinones), which can be formed naturally by the bacteria in the intestines (note: K1 is converted to K2 in the body); and

K3 (menadione), which is the most active of the synthetic forms of Vitamin K and is a water-

soluble vitamin not used as a prophylaxis (preventative vitamin) because of its potential to cause hemolytic anemia with jaundice.

Absorption of K1 is from the gut (duodenum and jejunum) via the lymphatic system. Thus, conditions that impair the fat absorption will also affect the absorption of Vitamin K. Antibiotics destroy the beneficial bacteria in the intestine needed for Vitamin K synthesis. All forms of Vitamin K have a common basic structure that acts as a cofactor for the enzymes essential for normal blood clotting, namely, in forming the blood clotting chemical prothrombin (Factor II).

In the liver, Vitamin K plays an important role in the actions of coagulation (blood clotting) factors [Proconvertin (Factor VII), Stuart-Power factor (Factor X), & Christmas factor (Factor IX)]. The following are also dependent on Vitamin K: protein C, protein S, and protein Z, along with anti-coagulants proteins C, S, and Z. Phytonadione (K1) is an analogue of Vitamin K, but it has the quickest onset of action, the most prolonged duration, and is the most potent of all the Vitamin K forms. K1 is safer than menadione (K3) to use on newborns. Vitamin K3 is the only form that could cause toxicity, causing hemolytic anemia (due to dying red blood cells), causing jaundice, liver damage and severe neurological damage.

Natural Vitamin K is required for two bone matrix proteins: osteocalcin and matrix-Gla (gamma carboxyglutamic acid). Gla is an amino acid that is part of certain proteins that control calcium. Vitamin K is shown to be beneficial in bone health because it helps to produce the natural protein osteocalcin. Vitamin K adds carboxyl groups to osteocalcin and other proteins that build and maintain bones by binding to (“chelating”) calcium. This is a self-limiting process (similar to hydroxylation of collagen by Vitamin C). Calcium needs these proteins to crystallize and strengthen bone tissue. Vitamin K has been approved for the treatment of osteoporosis in Japan. Thus, Vitamin K is required for proper bone formation and blood clotting. In both cases, Vitamin K accomplishes the job by assisting the body in transporting calcium*.

Green leafy vegetables (spinach, kale, parsley, cilantro, and broccoli) and certain vegetable oils (soybean oil, olive oil, cottonseed oil and canola oil) are good food sources of Vitamin K1. Hydrogenation of oils produces a biologically non-active form of Vitamin K called dihydrophylloquinone. The RDA for Vitamin K ranges from about 65mcg for adult females to 80mcg for adult males, per day.

Deficiency symptoms of Vitamin K manifest as decreased clotting, nosebleeds, increased blood pressure, hemorrhages, and diarrhea.

Precautions: Vitamin K should not be taken without prior medical consultation if pregnancy is suspected or if one is breast-feeding. Antibiotics and certain steroidal preparations may deplete or interfere with Vitamin K. Blood-thinning medications like warfarin and dicumarol will adversely interact with Vitamin K. Vitamin K inhibitors include: x-rays, radiation, aspirin, mineral oil, and laxatives. High amounts of Vitamins A and E can actually block Vitamin K, necessitating supplementation to compensate.

Selected References:

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10 http://www.merck.com/product/usa/pi_circulars/m/mephyton/mephyton_pi.pdf