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REPORT

An Unjustified Attack on Vitamin E

by William Faloon and Angela Pirisi

In article recently published in The Journal of the American Medical Association (JAMA) showed that high doses of vitamin E did not lower urinary measurements of lipid peroxidation in healthy people. Since the primary purpose of taking vitamin E is to protect against these kinds of free radicals, the authors of this study questioned the rationale for healthy people consuming supplemental vitamin E. The media jumped all over this story and attacked the value of vitamin E supplementation.(1)

The fact that vitamin E by itself failed to demonstrate suppression of lipid peroxidation is not surprising. It has long been known that vitamin E requires other antioxidants in order to recycle itself back into a free radical scavenger.(2-6) The people who participated in this study were screened to make sure that none of them had taken a multivitamin supplement in the preceding month. During the course of the study, the subjects were not allowed to take any supplement other than the prescribed vitamin E. This meant that there were no other antioxidants available to recycle vitamin E end-products back into effective suppressors of free radicals.

Studies published many years ago showed that for vitamin E to function as a continuous antioxidant, ample levels of vitamin C must be present to regenerate (donate more electrons) to vitamin E in the body. The subjects who participated in the JAMA study were not allowed to take vitamin C or any other supplement that would have enabled the vitamin E to function as an effective antioxidant.

The critical importance of "gamma" tocopherol

In this JAMA study, the dose administered to the test subjects ranged from 400 IU to 2000 IU per day of the "alpha" tocopherol form of vitamin E. It has been established that consuming only alpha-tocopherol can lower blood levels of the more potent form of vitamin E called "gamma" tocopherol.

Scientists have shown that the average American's blood-stream is five times more rich in alpha-tocopherol than gamma-tocopherol and that difference jumps 20-fold among people who take alpha-tocopherol supplements. Since the alpha form of vitamin E displaces the critically important gamma-tocopherol, it is understandable why people given high doses of only alpha-tocopherol would not show a reduction in lipid peroxidation.(7)

In a study published in the Proceedings of the National Academy of Sciences (April 1997), researchers reported that it could be dangerous to take high levels of alpha-tocopherol without also consuming gamma-tocopherol.(8) The reason for this is that too much alpha-tocopherol could deprive the cells of the gamma form of vitamin E that is needed to neutralize existing oxidizing agents such as the peroxynitrite radical, which can be especially damaging.

The scientists who wrote the National Academy of Sciences article suggested that alpha-tocopherol vitamin E supplements should contain at least 20% gamma-tocopherol. In response to these recommendations, members of The Life Extension Foundation long ago began taking supplements that provided the critically important "gamma" form of vitamin E.

Why CoQ10 is needed by those who take vitamin E

There are additional pieces to the vitamin E puzzle. A series of groundbreaking studies by Roland Stocker and his colleagues at The Heart Research Institute in Sydney, Australia demonstrates that vitamin E (alpha-tocopherol) systematically promotes LDL oxidation. Stocker calls this pro-oxidant action of vitamin E "tocopherol-mediated peroxidation," or TMP. Through the TMP process, vitamin E amplifies mild oxidative stresses so that they do much more damage to LDL.(9-11)

The good news is that Stocker's group discovered that CoQ10 naturally present in the body protects against TMP. They showed that one molecule of CoQ10 can prevent two TMP chain reactions involving as many as 40 to 80 free radicals. In pilot studies they tested LDL from the blood of human subjects given vitamin E and/or CoQ10 supplements. CoQ10 supplements reduced TMP, while vitamin E supplements increased it. When given together, the CoQ10 supplement significantly counteracted the TMP side-effect of the vitamin E supplement.(12-14) Here is a conclusion from one of several studies that show that adequate levels of

CoQ10 are required for vitamin E to function as an efficient antioxidant in the body:

“These results demonstrate that oral supplementation with alpha-tocopherol alone results in LDL that is more prone to oxidation initiation, whereas co-supplementation with coenzyme Q not only prevents this prooxidant activity of vitamin E but also provides the lipoprotein with increased resistance to oxidation.”

The work of Stocker and his colleagues agrees with other lines of recent research suggesting that CoQ10 cooperates with vitamin E in a complex partnership that we are only beginning to understand. Indeed these “co-antioxidants” are always found together in cell membranes and LDL. CoQ10 regenerates vitamin E, which would otherwise be quickly exhausted fighting oxidative stress. Vitamin E breaks off the chain reaction of lipid peroxidation, while CoQ10 helps to prevent it from starting.

The many studies of vitamin E supplementation published over the years did not take into account the CoQ10 naturally present in the body, but we can now see that this was a crucial factor. In these studies of vitamin E, CoQ10 served as the “silent partner,” amplifying the effect of vitamin E, regenerating vitamin E as it was exhausted, and preventing TMP.

Why this study may have been flawed

Having provided reasons why vitamin E by itself may not lower urinary measurements of free radical activity in the body, we should point out a few flaws in this JAMA study that may have invalidated the findings.

First of all, the vitamin E capsules were oil-based. This was not disclosed in the JAMA article and Life Extension magazine had to call the researchers to discover this piece of information. The researcher’s were unable to tell us what type of oil was used in the capsules, nor were they able to confirm if an antioxidant like ascorbyl palmitate was added to the capsules to protect the oil from oxidizing. Here is a quote from the description of these vitamin E supplements given to us by one of the scientists who authored the JAMA study:

“To the best of my knowledge it was oil-based. It was a amber colored capsule that contained a liquid preparation.”

Serious vitamin takers prefer dry powder as opposed to oil-based alpha-tocopherol because they are concerned that unprotected oil-based alpha-tocopherol may turn rancid. While vitamin E functions as an antioxidant in the body, it does not adequately protect oil in capsules. The reason for this is that supplemental alpha-tocopherol is in an ester form and it does not become a good antioxidant until it is de-esterified in your body. Had the superior dry powder alpha-tocopherol been used, the outcome of this study may have been different.

There are a several methods to evaluate free radical activity in the body, yet the researchers who conducted this JAMA study chose to restrict their analysis to only urinary measurements. We would have preferred to see blood tests that would measure thiobarbituric acid-reactive substances, plasma lipid peroxides, malondialdehyde levels and conjugated diene formation. These blood measurements of oxidation are the gold standard by which antioxidant efficacy is measured in the body. Had these blood tests been used instead of urine tests, the results of this study may have shown some degree of protection against oxidation.

Ignoring the positive studies

As of press time, 92 studies that include vitamin E have been published in the year 2001. The vast majority of these studies showed health benefits in those who supplement with vitamin E. Out of all of these studies, the media chose to use this one JAMA report to unfairly attack the use of vitamin E supplements.

New evidence is emerging to suggest that vitamin E may play a critical role in countering the potential oxidative damage and immune function breakdown that lies at the root of many diseases, such as heart disease,(15-16) various forms of cancer,(17) as well as diabetes (complications),(18) kidney disease,(19-20) Parkinson’s,(21) retinitis pigmentosa,(22) and infertility problems and pregnancy complications.(23-25) Vitamin E has also been shown to be pivotal to the normal functioning of important cellular processes that enable the body to fight off disease.(15-48)

The exciting part of recent findings is that they are demonstrating the various mechanisms through which vitamin E seems to elicit its therapeutic effects.(26-27) Besides attenuating oxidative damage, as it does in relation to lipid peroxidation, research is now showing that vitamin E also modulates the expression and activation of signal transduction pathways.(28) In cases of malignancy, such as cancer, these pathways seem to be dysfunctional and unable to inhibit growth and promote apoptosis in cancerous cells. (29)

Meanwhile, its success in subduing lipid peroxidation, platelet aggregation and inflammation associated with atherogenesis also



seems rooted in vitamin E's ability to inhibit the activity of protein kinase C (PKC), and consequently modulating the signaling pathways in which it's involved. In addition, alpha-tocopherol has been found to regulate adhesion molecule expression and inflammatory cell cytokine production.(15) According to one study, vitamin E's inhibition of platelet aggregation may actually be modulated, at least in part, by means of a mechanism that involves inhibiting the activity of protein kinase C.(30) When researchers examined the effect of vitamin E on vascular function and platelet aggregation in animal models of endothelial dysfunction, they found that the so-called antioxidant improved the activity of endothelium-derived nitric oxide but it wasn't due to prevention of LDL oxidation. Researchers noted that its anti-platelet activity must be independent of its antioxidant capacity, seeing as platelet aggregation was still halted by isoforms of vitamin E that are devoid antioxidant activity.

Researchers at the Mayo Clinic in Rochester, Minnesota wanted to test the theory that hypercholesterolemia and its health consequences may in part be mediated by pro-oxidative activity in plasma and tissue. The study investigators used cholesterol-fed pigs to examine the idea. Pigs were divided into two groups, both of which were fed a high cholesterol diet for 12 weeks. But only one of the groups was supplemented daily with 100 international units/kilogram vitamin E and 1 gram of vitamin C, while the other group served as a control. At the end of the diet study period, the activity of free radical scavengers was decreased and LDL oxidation showed an increase in the controls, while the group on the vitamin E and C-enhanced diet had normalized levels of oxidation and antioxidant status.(31)

In terms of heart disease, new evidence is weighing in to support vitamin E's usefulness as a therapeutic agent. According to researchers who reviewed five large, prospective clinical trials of alpha-tocopherol, four of them clearly vouch for vitamin E's ability to decrease lipid peroxidation, platelet aggregation, and reduce inflammation.(32-33) "The totality of evidence based on epidemiological data, in vitro studies and animal models, and the clinical trials appears to support a benefit for alpha-tocopherol supplementation in patients with pre-existing cardiovascular disease." Although, add the authors, it may be too soon to make definitive recommendations in view on ongoing clinical trials, which will bear more solid evidence yet.

Cancer, however, is another area of study with regards to vitamin E that is similarly yielding positive tidings about the nutrient's role in both treatment and prevention. A study by German researchers, for example, now shows that alpha-tocopheryl succinate (alpha-TOS), an esterified vitamin E analogue without antioxidant properties, can selectively kill malignant cells without harming normal cells, unlike the toxic effects of the popular chemotherapeutic drug adriamycin (doxorubicin).(34) Earlier research by the same investigators had shown that alpha-tocopheryl succinate could induce apoptosis in Jurkat T lymphoma cells and human colon cancer cells. In the latest study, a number of cancer cell lines, as well as several kinds of normal cells, were subjected to treatment with alpha-tocopheryl succinate. Results showed that all of the malignant cell types tested were susceptible to alpha-tocopheryl succinate. A dose of 50 micromoles per liter for a 12-hour period induced apoptosis in 28%-65% of the cells, including leukaemic cell lines, B and T lymphoma cells, as well as lung, breast and colorectal cancer cells. Meanwhile, no apoptotic effect was observed in normal cell types, such as haematopoietic cells, fibroblasts, endothelial cells, cardiomyocytes, hepatocytes and smooth muscle cells. While the research to date doesn't show how alpha-tocopheryl succinate may exert its pro-apoptotic activity on malignant cells while sparing healthy cells, the study authors suspect that its selectivity may be related to a role of the cell cycle, and an inhibitory effect on the cancer cell-proliferating activity of protein kinase C (PKC). The investigators conclude that alpha-tocopheryl succinate may present a new option for cancer prevention and/or treatment that does not pose significant side effects, and that its potential use as a stand-alone or adjuvant drug should be explored in animal studies looking at tumorigenesis and leukaemia.

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The cancer studies

While vitamin E's ability to inhibit cancer growth such as pancreas, breast and prostate has been shown by in vitro studies, now researchers at the University of California (UCLA) have found that the micronutrient may also serve a purpose in warding off gastric cancer too. An in vitro study looked at the effects of treating gastric cancer cells with doses of 25, 50 or 100 micrograms/milliliter of vitamin E. When researchers analysed the outcome after 24, 48 and 72 hours of incubating the treated malignant cells, they found that all three doses of vitamin E worked equally well at 72 hours to inhibit tumor growth, but that the 50 and 100 microgram/milliliter doses worked better at all time points. The authors concluded from their results that vitamin E effectively inhibits gastric carcinoma cell growth in vitro in a dose- and time-dependent manner.(35)



But vitamin E is not only proving effective at inhibiting cancer cell growth,(29) as it has also been shown to be among the antioxidant agents that can enhance the efficacy of chemotherapeutic agents, as well as decrease the side effects related to their cytotoxicity. For example, consider a recent study that looked at how vitamin E might assist in increasing the susceptibility of colon carcinoma cells to the chemotherapeutic agent, 5-fluorouracil (5 FU). Vitamin E, in conjunction with N-acetylcysteine (NAC), reduced intercellular oxidant levels by 500%, which increased pro-apoptotic bax protein expression and apoptotic response to a non-toxic dose of 5 FU in two colorectal cancer cell lines (colo 201 and colo 205).(36) Meanwhile, in another study, vitamin E was shown to oppose the toxic effects of the chemotherapeutic agent doxorubicin in the skin (as evidenced by increased theoredoxin reductase activity in skin, as well as increased glutathione peroxidase activity in the erythrocytes) and may therefore also alleviate cytotoxicity associated with chemotherapy treatment.(37)

Fertility

Apart from heart disease and cancer, vitamin E has also been recognized in terms of its ability to improve sperm function and promote healthy pregnancy, although this role was

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demonstrated several decades ago. In Japan, researchers demonstrated that vitamin E protects trophoblastic cells (these mediate fetal implantation and form a thin membrane to cover the fetus) against oxidative stress in the labyrinthine region of the placenta during its development.(25) Alpha-tocopherol transfer protein (alpha-TTP) is known to bind alpha-tocopherol and is associated with vitamin E deficiency, as has been shown in patients with ataxia. In essence, the alpha-tocopherol transfer gene expression modulates circulating levels of alpha-tocopherol. The researchers examined the effect of the protein in male mice fertility and in pregnant female mice, finding that alpha-tocopherol transfer protein did not affect male fertility, its expression related to severe impairment of the placentas that culminated in the death of the embryos at mid-gestation. Investigators then tried to administer excess vitamin E or a synthetic antioxidant supplement to pregnant female mice. In this case, results showed that the extra vitamin E, or antioxidant supplementation, prevented placental failure and allowed the mice to reach full-term. Other findings suggest that vitamin E also helps to protect sperm from oxidative insult that would tamper with its structural integrity and normal functioning.(23) Such findings trace back to vitamin E's humble beginnings, before any relation to heart disease and cancer was made. Keep in mind that vitamin E was discovered in the 1920s after it was found that rats fed a vitamin E deficient diet could not reproduce. The term tocopherol now used to identify vitamin E comes from the Greek word meaning "to bear offspring." Of course, as we are finding out, that name does not appropriately encompass the multi-faceted role that vitamin E is proving to play in overall health.

Conclusion

What the JAMA report helped confirm is the fact that taking a high dose of a single antioxidant may not be an effective way to prevent lipid peroxidation. Since most serious supplement consumers take vitamin C and CoQ10 along with their vitamin E, the results of this study would not appear to pertain to them. Those who take the gamma-tocopherol form of vitamin E should be especially comforted to know that they may be obtaining optimal protection against a wide range of toxic free radicals that induce lipid peroxidation.

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Scientists suggest that those who take alpha-tocopherol vitamin E supplements should also supplement with at least 20% gamma-tocopherol. In response to these recommendations, Foundation members have been taking one capsule a day of a supplement called Gamma E Tocopherol that provides 210 mg of gamma-tocopherol in each capsule. This same amount of gamma-tocopherol (210 mg) is also included in the Life Extension Booster formula. Foundation members obtain additional protection in the Super CoQ10 softgel caps that are fortified with a tocotrienol complex that provides the gamma tocotrienol vitamin E fraction.

Since the average member takes between 400 IU and 1000 IU a day of alpha-tocopherol vitamin E, the 210 mg of gamma-tocopherol found in either Gamma E Tocopherol or Life Extension Booster more than fulfills this 20% gamma-tocopherol requirement.

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