

New Study Validates Life Extension®'s Early Warning

Synthetic Alpha Tocopherol Shown to Increase Prostate Cancer Risk

We Predicted This Outcome!

Longtime members of the Life Extension Foundation® have heard our **warnings** against **synthetic alpha tocopherol** many times.

In **1997**, we announced that taking **only** the **alpha tocopherol** form of vitamin E displaces critically important **gamma tocopherol** in the body.

By displacing **gamma tocopherol**, we feared that high doses of **alpha tocopherol** could increase cancer risks.

In fact, three years after Life Extension's first warning, the **Johns Hopkins School of Public Health** released the results of a huge study (10,456 men). The findings showed that men with the highest **gamma tocopherol** blood levels had a fivefold reduction in **prostate cancer risk**. This same study showed that selenium and alpha tocopherol also reduced prostate cancer risk but **only** when **gamma tocopherol** levels were high.¹

Confirmatory studies document higher levels of **gamma tocopherol** to be strongly associated with reduced cancer risks.^{2,3,4,5}

While both alpha and gamma tocopherol are potent antioxidants, *gamma tocopherol has a unique function*. Because of its different chemical structure, **gamma tocopherol scavenges reactive nitrogen species, which can damage proteins, lipids, and DNA**.^{6,7,8}

Cancer is the end result of damage inflicted upon critical DNA genes that help regulate cellular growth and maturation. The fact that **supplementation with isolated, synthetic alpha tocopherol depletes plasma gamma tocopherol levels** means that the researchers who designed the SELECT trial created a biological catastrophe. The result of their ignorance is that men randomized to receive only synthetic alpha tocopherol suffered significant gamma tocopherol depletion and, consequently, DNA damage from reactive nitrogen species. The fact that higher prostate cancer rates were observed in the group overloaded with synthetic alpha tocopherol in the SELECT trial was predictable and expected based upon fundamental facts Life Extension understood more than a decade ago.

This costly **government-funded study** was initiated in the year 2001.

Life Extension was highly critical of this study design because it exposed healthy men to relatively **high doses** of **synthetic alpha tocopherol** without **any** supplemental **gamma tocopherol** to compensate. The results confirmed Life Extension's worst fears. Compared to a placebo, men taking **synthetic alpha tocopherol** had a **17% greater** incidence of **prostate cancer**.⁹ This is a tragedy from the standpoint of the study participants who should not have been given **alpha tocopherol** by itself. It was also a waste of tax dollars used to fund a study that was **designed to fail** from the outset.

In another arm of the study where **selenium** was given in addition to **alpha tocopherol**, there was not a statistically significant increase in prostate cancer.⁹ The study's authors commented on the protective effect of selenium but never mentioned the damage they inflicted by failing to include **gamma tocopherol** in their study. **Selenium** boosts antioxidant defenses in the body (such as glutathione peroxidase) that would help compensate for the displacement of **gamma tocopherol** by **alpha tocopherol**.¹⁰

The doctors involved in the design of the study comprise a "who's who" of the conventional cancer establishment. Virtually every major cancer center was involved in the study conception, and it seemed that virtually every pharmaceutical company had made generous payments to the study's overseers. If there was ever a greater **financial conflict of interest**, we have yet to see it. It was in the economic interest of the drug companies, the cancer centers, and the mainstream doctors to see this study **fail**, and the harsh comments against dietary supplements by the mainstream doctors reveal a strong **bias** against over-the-counter dietary supplements.

While the study's authors claim that there is not a "biological explanation" from their data to explain the increase in prostate cancer in the alpha tocopherol-only group, **Life Extension** long ago **predicted in writing** this study would fail. As we expected, the men

in this study who received **alpha tocopherol** experienced a **45% depletion** of vital **gamma tocopherol** during the initial 5.5-year median study period.⁹

A myriad of reports now point to the urgent need for Americans to obtain sufficient **gamma tocopherol**.^{8,11,12,13,14,15,16,17,18,19,20,21} Yet the vast majority of human clinical research focuses only on **alpha tocopherol** – as if it were the only form of vitamin E people require. This article explains the mechanisms involved in the development of **prostate cancer** and why no single supplement can be counted on alone to prevent it.>>>

Based on reports showing **antioxidants reduce** the incidence of prostate cancer, the **federal government** spent over **\$114 million** to see if **synthetic alpha tocopherol** and/or a single sourced **selenium** supplement would prevent **prostate cancer** in a large placebo-controlled trial conducted at major cancer centers throughout the United States.²²

The long-term follow-up of the study named SELECT was published in the *Journal of the American Medical Association* on October 12, 2011. Long before this study's findings were released, **Life Extension** predicted that it would **fail** and warned that men taking high doses of **alpha tocopherol** without also taking **gamma tocopherol** faced increased disease risk.

In the initial results of the SELECT study over a median 5.5-year period, men supplemented with **synthetic alpha tocopherol** experienced significant **gamma tocopherol** depletion of **45%**. Men supplemented with alpha tocopherol plus selenium experienced a **48%** depletion of gamma tocopherol. These gamma tocopherol depletions occurred by 6 months that were sustained during the course of a median trial period of 5.5 years.²²

It should be noted that serious supplement users choose natural **alpha tocopherol** because it has been shown to exert superior biological effects in the body.^{23,24} The synthetic form of alpha tocopherol is most often used in brand name multivitamins made by pharmaceutical companies that are widely advertised on national television. Organizations like **Life Extension** have resisted the cheap price of synthetic vitamin E and use the more expensive natural form of alpha tocopherol in nutrient formulations.

It should also be noted that the only form of selenium used in this study was L-selenomethionine (200 mcg). Yet scientific studies dating back to the **1970s** show that other forms of selenium might provide greater protection against cancer. That's why most Life Extension members obtain their selenium from more than one source that includes se-methylselenocysteine and/or, sodium selenite plus L-selenomethionine. ^{25,26,27,28,28a,28b,28c}

Life Extension has conducted a thorough review of the SELECT study that is now being used as a basis used to attack dietary supplements. The more of this article you read, the more you will understand why the SELECT study was **designed to fail** from the outset.

INITIAL SELECT REPORT SHOWED NO RISK OR BENEFIT

When data was first reported from the SELECT trial on December 9, 2008, it found no reduction in prostate cancer incidence in men taking alpha tocopherol or selenium over a median period of 5.5 years.²²

This was not surprising since we have known for the past 14 years that when alpha tocopherol is taken by itself, it displaces critically important gamma tocopherol in our cells.^{29,30,31,32} An abundance of evidence points to the **gamma tocopherol** form of vitamin E as the most protective against prostate cancer.^{2,33,34,35}

By supplementing aging men with only alpha tocopherol, doctors increased these men's prostate cancer risk by depriving prostate cells of critical gamma tocopherol. This is only a tiny part of the real story behind this flawed study.

The American Medical Association used the initial finding of no benefit to discredit vitamin E and selenium supplements. An editorial by the American Medical Association concluded by advising:

"... physicians should not recommend selenium or vitamin E — or any other antioxidant supplements — to their patients for preventing prostate cancer."³⁶

In January 2008, as part of our article titled "**Merv Griffin's Tragic Death from Prostate Cancer**,"³⁷ we predicted that the SELECT trial would fail. We also stated that this faulty SELECT study would be misused by the medical establishment to discredit by extrapolation other low-cost efficacious nutrients like vitamin D and fish oil.

HOW GAMMA TOCOPHEROL PROTECTS AGAINST CANCER

Gamma tocopherol exerts **anti-cancer** effects through a variety of important mechanisms, giving it an especially broad spectrum of action against a host of tumor types. At the very beginning of the cancer development process, gamma tocopherol traps reactive

nitrogen species and other free radicals that cause mutations in DNA strands and render cells vulnerable to malignant transformation.^{6,7,8} This is a crucial step in the prevention of cancer.

Gamma tocopherol inhibits cancer cell growth in culture through a number of different mechanisms.³⁵ It down-regulates control molecules known as cyclins, which trap cancer cells in the midst of their reproduction cycle and prevent them from reproducing and spreading.³⁴ This anti-cancer effect appears to be based on a mechanism separate from the vitamin's well-known antioxidant powers.

A cell membrane receptor called PPAR-gamma is a promising target for anti-cancer therapies because it affects genes that control cancer cell growth and death.³⁸ This is why PPAR-activating drugs are being researched and developed by pharmaceutical companies as anti-cancer drugs. Gamma tocopherol is more powerful than alpha tocopherol at stimulating PPAR-gamma activity, especially in colon cancer cells.^{11,39} In prostate cancer cells, PPAR-gamma stimulation by gamma tocopherol resulted in a complete cessation of cancer cell growth.¹¹

Once cancerous transformation has taken place, there are still biological opportunities to prevent full-blown tumor development. One of these ways is the induction of deliberate cell death through built-in genetic programs, a process called apoptosis. In a variety of cancer tissues, gamma tocopherol has been found to be superior to alpha tocopherol at inducing apoptosis, triggering a number of cell-death-inducing pathways.^{2,40} In prostate cancer cells, gamma tocopherol induced cell death by blocking synthesis of important cell membrane components.⁴¹ Gamma tocopherol also reduced the development of new blood vessel formation in tumors, depriving them of the nutrients they need to thrive.⁴²

To date, all of these mechanisms have been shown to inhibit cancers of the colon, prostate, breast, and lung in animal models, with many more under active investigation.⁴³

A study found that women who consumed the most vitamin E from food sources had a **60% reduction** in the risk of breast cancer compared to women with the lowest consumption. The form of vitamin E that strongly predominates in food sources is **gamma tocopherol**.⁴⁴

In the **December 2000** issue of the *Journal of the National Cancer Institute*, researchers at the *Johns Hopkins School of Public Health* published results of a huge study of 10,456 men showing that those with the highest **gamma tocopherol** blood levels had a **fivefold reduction** in prostate cancer risk. This same study showed that selenium and alpha tocopherol also reduced prostate cancer risk but **only** when gamma tocopherol levels were high.⁹

These findings should have been glaringly apparent to those involved in the SELECT human clinical trial that began one year later (in 2001), yet the SELECT study design called for men to be given a high dose of **synthetic alpha tocopherol** that resulted in depletion of vital **gamma tocopherol** by **45%** during the initial 5.5-year median trial period! This is one reason why we believe the SELECT study was "**designed to fail**."

We know that free radical-induced damage to DNA genes can cause cancer, but there are *other* risk factors beyond oxidative stress to blame for most prostate tumors.

PROSTATE CANCER IS INITIATED EARLY IN LIFE

While prostate cancer is not usually diagnosed until men reach older ages, it can be initiated 15–25 years prior to clinical manifestation. In fact, there is convincing evidence that the initiating DNA damage inflicted by **estrogen** to prostate cells can occur before a man is even born.⁴⁵

Studies show that as early as the second and third trimester of life, exposure to elevated estrogens in the womb can initiate prostate cancer that may not manifest for 80 years.^{46,46,47,48,49,50,51} A man's lifetime exposure to higher than normal estrogen may be a contributing factor to prostate cancer development. There is no evidence that antioxidants like alpha tocopherol and selenium would protect against this kind of prostate cancer induced by prolonged excess estrogen exposure.

Please don't feel helpless about this, as it requires more than mere **initiation** for cancer to fully develop. Dietary and other lifestyle factors have an enormous impact on whether men will develop prostate cancer, even if they are genetically predisposed.

THE CAUSE OF ALL CANCERS

Cancer can be defined in one sentence as follows:

"CANCER IS THE ACCUMULATION OF MUTATIONS IN GENES THAT REGULATE CELLULAR PROLIFERATION."⁵²

All cancers are caused by gene mutations. Every time a cell divides, there are slight mutations to one's genes. Oxidative stress accelerates gene mutation but is by no means the primary factor. While selenium and vitamin E reduce some types of oxidative stress, the aged men in the SELECT study had already sustained considerable genetic mutations that are not reversible by taking antioxidants.

Fortunately, there are nutrients that have been shown to favorably reverse the gene alterations involved in cancer initiation and progression. One promising nutrient is vitamin D, which has been shown to slash prostate cancer risk in some studies.⁵³ Serum levels of vitamin D were not assessed in the SELECT study, so it was not possible to know which men had protective levels of vitamin D and which had insufficient or even deficient levels. If men in the placebo group had even slightly higher vitamin D status, they should have been less likely to contract prostate cancer.

What researchers fail to comprehend is that giving aged men a single antioxidant like alpha tocopherol is not going to reverse seven decades of genetic damage to prostate DNA. Fortunately, we know of other mechanisms that fuel prostate cancer progression that can be mitigated.

EATING YOUR WAY TO PROSTATE CANCER

Cancer cells lurk in the prostate glands of most aging men, yet only one in six men are ever diagnosed with prostate cancer. If one looks at what is required for a single cancer cell to develop into a detectable tumor, it becomes obvious that natural barriers exist to protect people against full-blown cancer.^{2,41,42,43,44}

Unfortunately, the dietary choices of most men living in the modern Western world circumvent the body's natural **protective barriers**. The end result is that most men unwittingly provide **biological fuel** for existing prostate cancer cells to propagate and metastasize.

Fortunately, an understanding of the biological roles of diet and specific nutrients can enable aging men to achieve a considerable amount of control over whether isolated cancer cells in their prostate gland will ever show up as a clinically diagnosed disease.

The impact of the food we ingest on cell growth and death is so pronounced that it can be identical to the effects displayed by anti-cancer drugs. As it relates to the SELECT study on alpha tocopherol and/or selenium, the study participants' diet was not taken into consideration. This fact alone could have rendered the findings highly suspect. Read on to see what we mean.

THE FIRST LINE OF DEFENSE: OMEGA-3 FATTY ACIDS

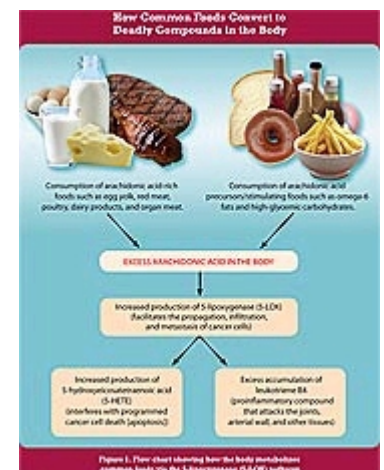
Diets high in **omega-6 fats** and **saturated fats** are associated with greater prostate cancer risk whereas increased intake of **omega-3 fats** from fish has been shown to reduce risk. Based on consistent epidemiological findings across a wide range of human populations, scientists have sought to understand why eating the wrong kinds of fat (saturated and omega-6 fats) provokes a stimulatory effect on prostate cancer.^{54,55}

To ascertain what happens after we eat bad fats, all one has to do is look at the metabolic breakdown pathways that these fats follow in the body, as shown in the chart on *Figure 1*. For example, let us assume that for dinner, you eat a steak (a source of saturated fat) and a salad, along with a typical salad dressing of soybean and/or safflower oils (sources of omega-6 fats).

As seen in the (*Figure 1*) flow chart, saturated and omega-6 fats convert to arachidonic acid in the body. The meat itself contains arachidonic acid. One way that the body rids itself of excess arachidonic acid is provoking a dangerous metabolizing pathway through 5-lipoxygenase (5-LOX). Studies show that 5-LOX products directly stimulate prostate cancer cell proliferation via several well-defined mechanisms.^{56,57,58,59,60,61,62} Arachidonic acid is metabolized by 5-LOX to 5-hydroxyeicosatetraenoic acid (5-HETE), a potent survival factor that prostate cancer cells use to escape destruction.^{58,63,64}

Figure 1 clearly demonstrates how consuming a diet of foods rich in arachidonic acid directly provokes the production of dangerous 5-LOX products, which can promote the progression of prostate cancer. In addition to 5-HETE, 5-LOX also metabolizes arachidonic acid into leukotriene B4, a potent pro-inflammatory agent that causes destructive reactions throughout the body and inflicts severe damage to the arterial wall.^{65,66,67,68,69,70,71}

One reason that fish oil supplements have become so popular is that their beneficial EPA/DHA fatty acids can help reduce the production of arachidonic acid-derived eicosanoids in the body.^{72,73,74,75,76,77} As shown in *Figure 1* above, if arachidonic acid levels are reduced, there would be a corresponding suppression of the 5-LOX products 5-HETE and leukotriene B4.



[Click Here for Figure 1](#)

Click Here for Figure 1

5-LOX IS OVEREXPRESSED IN PROSTATE CANCER

Based on studies showing that consumption of foods rich in arachidonic acid is greatest in regions with high incidences of prostate cancer,^{56,57,62,82} scientists sought to determine how much of the 5-LOX enzyme is present in malignant versus benign prostate tissues.⁶⁰

Using biopsy samples taken from living human patients, the researchers found that 5-LOX mRNA levels were an astounding **sixfold greater** in malignant prostate tissues compared with benign tissues. This study also found that levels of 5-HETE were 2.2-fold greater in malignant versus benign prostate tissues.⁶⁰ The scientists concluded this study by stating that selective inhibitors of 5-LOX may be useful in the prevention or treatment of patients with prostate cancer.

5-LOX PROMOTES TUMOR GROWTH FACTORS

As the evidence mounts that ingesting "bad fats" increases prostate cancer risk, scientists are evaluating the effects of 5-LOX on various growth factors involved in the progression, angiogenesis, and metastasis of cancer cells.

One study found that 5-LOX activity is required to stimulate prostate cancer cell growth by **epidermal growth factor** (EGF) and other cancer cell proliferating factors produced in the body. When 5-LOX levels were reduced, the cancer cell stimulatory effect of EGF and other growth factors was diminished.⁵⁶

In a mouse study, an increase in 5-LOX resulted in a corresponding increase in **vascular endothelial growth factor**, a key growth factor that tumor cells use to stimulate new blood vessel formation (angiogenesis) into the tumor. *5-Lipoxygenase* inhibitors were shown to reduce tumor angiogenesis along with a host of other growth factors.⁸³ In both androgen-dependent and androgen-independent human prostate cancer cell lines, the inhibition of 5-LOX has consistently been shown to induce rapid and massive apoptosis (cancer cell destruction).^{57,82,84,85}

DAILY USE OF ASPIRIN MAY DECREASE PROSTATE RISKS

Researchers studied 2,447 men over 12 years, examining them every other year. After adjusting for age, diabetes, hypertension, and other factors, they found that men who took a daily aspirin or another NSAID (like ibuprofen) reduced their risk of moderate or severe urinary symptoms by 27% and lowered their risk of an enlarged prostate by 49%. Even more intriguing was the finding that men who consumed aspirin or another NSAID were 48% less likely to have an elevated level of prostate-specific antigen (PSA), the protein measured in the blood that helps detect prostate cancer.⁷⁸

Aspirin inhibits the cyclooxygenase (COX-1 and COX-2) enzymes, which are also involved in the arachidonic acid inflammatory pathway.^{79,80} Like 5-lipoxygenase, COX-2 is known to promote the proliferation of prostate cancer cells.⁸¹

In the SELECT study used to discredit alpha tocopherol-selenium, the use of aspirin or ibuprofen by the placebo group may have reduced the prostate cancer risk more than what could be expected in those receiving alpha tocopherol and selenium who may not have been taking as much aspirin or other NSAID.

Once one understands the lethal metabolic cascades that occur in response to poor dietary choices, it is easy to see why people who excessively consume foods rich in arachidonic acid, and/or those who do not reduce the production of excessive arachidonic acid metabolites, are setting themselves up for prostate cancer and a host of inflammatory diseases (including atherosclerosis). A chart appearing later in this article clearly shows the destructive cascade initiated by excess arachidonic acid.

Men in the SELECT study who took alpha tocopherol-selenium supplements but consumed foods high in arachidonic acid and not enough omega-3s would be more likely to develop prostate cancer. The researchers who designed this study should have known to correct for this critical confounding factor, i.e., dietary patterns.

SUPPRESSING ARACHIDONIC ACID BYPRODUCTS

Health-conscious people take nutrients like **fish oil**, **curcumin**, and **lycopene** that help to lower 5-LOX activity in the body.^{82,86,87,88,89,90,91,92,93,94}

A rat study showed that **gamma tocopherol**, but not **alpha tocopherol**, exhibited potent reduction of PGE2 and leukotriene B4, powerful pro-inflammatory factors, end-products of the COX-2 and 5-LOX-pathways, respectively.⁹⁵ A review of several studies indicates that combinations of alpha and gamma tocopherol optimally reduce end products (such as PGE2 and leukotriene B4) of arachidonic acid breakdown in the body.^{17,96,96}

Extracts from the boswellia plant selectively inhibit 5-lipoxygenase (5-LOX)^{97,98} A novel boswellia extract has been developed that is **52% more bioavailable compared to standard boswellia extracts**,^{99,100} thus providing a greater opportunity to suppress deadly 5-LOX and other cancer-promoting byproducts of **arachidonic acid**.

As humans age, over-expression of the enzymes **5-LOX** and **COX-2** typically occurs. For maturing males, excess levels of these pro-inflammatory enzymes may contribute to the epidemic of prostate cancer observed after the age of 60.¹⁰¹

Based on the cumulative knowledge that 5-LOX, COX-2, and their breakdown products can promote the invasion and metastasis of prostate cancer cells, it would appear advantageous to take aggressive steps to suppress these lethal enzymes.

For the unfortunate men who received only *alpha tocopherol* in the SELECT study, the suppression of gamma tocopherol that occurred in their bodies presumably exposed them to higher levels of cancer-promoting byproducts of arachidonic acid. Interestingly, selenium has shown 5-LOX-inhibiting effects, which may partially explain why men receiving selenium and alpha tocopherol-alone did not show a statistically significant increase in prostate cancer.

MULTIPLE DANGERS OF EXCESS ARACHIDONIC ACID

In response to arachidonic acid overload, the body increases its production of enzymes like 5-lipoxygenase (5-LOX) to degrade arachidonic acid. Not only do 5-LOX products directly stimulate cancer cell propagation,^{66,67,68,69,70,71,72} but the breakdown products that 5-LOX produces from arachidonic acid (such as leukotriene B4, 5-HETE, and hydroxylated fatty acids) cause tissue destruction, chronic inflammation, and increased resistance of tumor cells to apoptosis (programmed cell destruction).^{66,102,103,104,105,106,107}

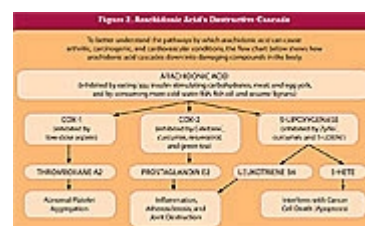
It is important to understand that 5-LOX is not the only dangerous enzyme the body produces to break down arachidonic acid. As seen in *Figure 2 below*, both cyclooxygenase-1 and cyclooxygenase-2 (COX-1 and COX-2) also participate in the degradation of arachidonic acid.

COX-1 causes the production of thromboxane A2, which can promote abnormal arterial blood clotting (thrombosis), resulting in heart attack and stroke.^{108,109,110,111,112} COX-2 is directly involved in cancer cell propagation,^{113,114,115,116} while its breakdown product (prostaglandin E2) promotes chronic inflammation.^{107,117,118} Most health-conscious people already inhibit the COX-1 and COX-2 enzymes by taking low-dose aspirin,^{110,119,120,121,122} curcumin,^{90,123,124,125,126,127,128,129,130,131,132,133,134} green tea,^{90,135,136} and various flavonoids such as resveratrol.^{90,136,137}

A more integrative approach to this problem, however, would be to also reduce levels of arachidonic acid, which is the precursor of 5-HETE and leukotriene B4.

SOY, LIGNANS, AND CRUCIFEROUS VEGETABLES

Men who regularly consume certain plant foods have sharply lower rates of prostate cancer. Studies show that cauliflower, broccoli, flax lignans, and soy isoflavones^{137,138,139,140,141,142,143,144,145,146} protect against a host of diseases, including prostate cancer. If the men in the SELECT placebo group ate an even slightly healthier diet, then they would be expected to enjoy a lower rate of prostate cancer compared with men who took the alpha tocopherol-selenium supplements but ate fewer cancer-preventing plant foods.



[Click Here for Figure 2 Chart](#)

LOW TESTOSTERONE INCREASES PROSTATE CANCER RISK

In the book *Testosterone for Life*, authored by Harvard University experts, detailed findings are presented that dispel a misleading notion about testosterone causing prostate cancer.¹⁴⁷ These researchers meticulously document their observations that men with low levels of testosterone have higher prostate cancer risks.

This finding provides another confounding factor that skews the results of the SELECT trial that only used alpha tocopherol and/or selenium. If men receiving the supplements had lower testosterone levels, they would conceivably have a higher rate of prostate cancer.

TOO MANY FACTORS INVOLVED IN PROSTATE CANCER CAUSATION

The SELECT study was designed based on prior studies showing sharply lower risks of prostate cancer in men who consumed vitamin E and selenium.^{148,149,150,151,152} It was also based on the premise that protecting genes against oxidative stress would reduce prostate cancer incidence in aged men.

We now know of dozens of factors involved in the development of full-blown prostate cancer. One could not expect that taking just one or two nutrients would result in less prostate cancer developing in these older study subjects. There are too many other causes that should have been factored in when the SELECT study was originally designed.

It is encouraging that a plethora of new research findings have identified definitive ways for aging men to drastically slash their risk of developing prostate and other cancers.

CONCLUSION

Life Extension long ago warned of **increased** disease risk in those who only took **alpha tocopherol** supplements without also taking **gamma tocopherol**.

Leaving out gamma tocopherol is not the only flaw in the SELECT study. It is rather conspicuous, however, since men supplemented with alpha tocopherol and alpha tocopherol plus selenium experienced a **45–48% depletion** in **gamma tocopherol** levels by six months that was sustained during the course of the initial 5.5-year median trial period.

The facts revealed in this rebuttal to the SELECT study identify a fundamental problem confronting researchers who seek to "prove" whether a certain supplement prevents a disease. There are too many "other" factors involved in the development and progression of prostate cancer including low levels of testosterone, increased levels of estrogen, co-existing diabetes or metabolic syndrome, low vitamin D intake, and increased dietary saturated fats.¹⁵³ These confounding factors therefore make it difficult to study just one or two compounds and expect to come up with a valid finding.

To emphasize today's sense of urgency, the aging population will contract prostate cancer at epidemic levels unless aggressive changes are implemented immediately. That's because mutated cells in the prostate glands of aging males are already on the verge of maturing into full-blown cancer.

This is why we encourage Life Extension® members to consume healthy diets and supplements that have been shown to sharply reduce prostate cancer incidence. There is not enough time left in our generation's projected life spans to withstand the kind of scientific design flaws seen in the SELECT study, the medical establishment's bias against supplements, and arbitrary standards set by the pharmaceutical monopoly.

If you have any questions on the scientific content of this article, please call a Life Extension Health Advisor at 1-800-226-2370.

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