Vitamin E is a fat-soluble antioxidant that stops the production of ROS formed when fat undergoes oxidation. Scientists are investigating rapidly with oxygen to form reactive oxygen species (ROS). The body forms ROS endogenously when it converts food to energy, and damage cells and might contribute to the development of cardiovascular disease and cancer [5]. Unshared electrons are highly energetic and react Antioxidants protect cells from the damaging effects of free radicals, which are molecules that contain an unshared electron. Free radicals associated with free radicals.

In addition to its activities as an antioxidant, vitamin E is involved in immune function and, as shown primarily by in vitro studies of cells, cell signaling, regulation of gene expression, and other metabolic processes [1]. Alpha-tocopherol inhibits the activity of protein kinase C, an enzyme involved in cell proliferation and differentiation in smooth muscle cells, platelets, and monocytes [6]. Vitamin-E–replete endothelial cells lining the interior surface of blood vessels are better able to resist blood-cell components adhering to this surface. Vitamin E also increases the expression of two enzymes that suppress arachidonic acid metabolism, thereby increasing the release of prostacyclin from the endothelium, which, in turn, dilates blood vessels and inhibits platelet aggregation [6].
Recommended Intakes

Intake recommendations for vitamin E and other nutrients are provided in the Dietary Reference Intakes (DRIs) developed by the Food and Nutrition Board (FNB) at the Institute of Medicine of The National Academies (formerly National Academy of Sciences) [6]. DRI is the general term for a set of reference values used to plan and assess nutrient intakes of healthy people. These values, which vary by age and gender, include:

- **Recommended Dietary Allowance (RDA):** average daily level of intake sufficient to meet the nutrient requirements of nearly all (97%-98%) healthy people.
- **Adequate Intake (AI):** established when evidence is insufficient to develop an RDA and is set at a level assumed to ensure nutritional adequacy.
- **Tolerable Upper Intake Level (UL):** maximum daily intake unlikely to cause adverse health effects [6].

The FNB’s vitamin E recommendations are for alpha-tocopherol alone, the only form maintained in plasma. The FNB based these recommendations primarily on serum levels of the nutrient that provide adequate protection in a test measuring the survival of erythrocytes when exposed to hydrogen peroxide, a free radical [6]. Acknowledging "great uncertainties" in these data, the FNB has called for research to identify other biomarkers for assessing vitamin E requirements.

RDAs for vitamin E are provided in milligrams (mg) and are listed in Table 1. Because insufficient data are available to develop RDAs for infants, AIs were developed based on the amount of vitamin E consumed by healthy breastfed babies.

At present, the vitamin E content of foods and dietary supplements is listed on labels in international units (IUs), a measure of biological activity rather than quantity. Naturally sourced vitamin E is called d-alpha-tocopherol; the synthetically produced form is dl-alpha-tocopherol. Conversion rules are as follows:

- To convert from mg to IU: 1 mg of alpha-tocopherol is equivalent to 1.49 IU of the natural form or 2.22 IU of the synthetic form.
- To convert from IU to mg: 1 IU of alpha-tocopherol is equivalent to 0.67 mg of the natural form or 0.45 mg of the synthetic form.

Table 1 lists the RDAs for alpha-tocopherol in both mg and IU of the natural form; for example, 15 mg x 1.49 IU/mg = 22.4 IU. The corresponding value for synthetic alpha-tocopherol would be 33.3 IU (15 mg x 2.22 IU/mg).

### Table 1: Recommended Dietary Allowances (RDAs) for Vitamin E (Alpha-Tocopherol) [6]

<table>
<thead>
<tr>
<th>Age</th>
<th>Males</th>
<th>Females</th>
<th>Pregnancy</th>
<th>Lactation</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-6 months*</td>
<td>4 mg</td>
<td>4 mg</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(6 IU)</td>
<td>(6 IU)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7-12 months*</td>
<td>5 mg</td>
<td>5 mg</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(7.5 IU)</td>
<td>(7.5 IU)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-3 years</td>
<td>6 mg</td>
<td>6 mg</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(9 IU)</td>
<td>(9 IU)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4-8 years</td>
<td>7 mg</td>
<td>7 mg</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(10.4 IU)</td>
<td>(10.4 IU)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9-13 years</td>
<td>11 mg</td>
<td>11 mg</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(16.4 IU)</td>
<td>(16.4 IU)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14+ years</td>
<td>15 mg</td>
<td>15 mg</td>
<td>15 mg</td>
<td>19 mg</td>
</tr>
<tr>
<td></td>
<td>(22.4 IU)</td>
<td>(22.4 IU)</td>
<td>(22.4 IU)</td>
<td>(28.4 IU)</td>
</tr>
</tbody>
</table>

*Adequate Intake (AI)

Sources of Vitamin E

**Food**

Numerous foods provide vitamin E. Nuts, seeds, and vegetable oils are among the best sources of alpha-tocopherol, and significant amounts are available in green leafy vegetables and fortified cereals (see Table 2 for a more detailed list) [7]. Most vitamin E in American diets is in the form of gamma-tocopherol from soybean, canola, corn, and other vegetable oils and food products [4].

### Table 2: Selected Food Sources of Vitamin E (Alpha-Tocopherol) [7]

<table>
<thead>
<tr>
<th>Food</th>
<th>Milligrams (mg) per serving</th>
<th>Percent DV*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wheat germ oil, 1 tablespoon</td>
<td>20.3</td>
<td>100</td>
</tr>
<tr>
<td>Almonds, dry roasted, 1 ounce</td>
<td>7.4</td>
<td>40</td>
</tr>
<tr>
<td>Sunflower seeds, dry roasted, 1 ounce</td>
<td>6.0</td>
<td>30</td>
</tr>
<tr>
<td>Sunflower oil, 1 tablespoon</td>
<td>5.6</td>
<td>28</td>
</tr>
<tr>
<td>Safflower oil, 1 tablespoon</td>
<td>4.6</td>
<td>25</td>
</tr>
<tr>
<td>Hazelnuts, dry roasted, 1 ounce</td>
<td>4.3</td>
<td>22</td>
</tr>
<tr>
<td>Peanut butter, 2 tablespoons</td>
<td>2.9</td>
<td>15</td>
</tr>
<tr>
<td>Peanuts, dry roasted, 1 ounce</td>
<td>2.2</td>
<td>11</td>
</tr>
<tr>
<td>Corn oil, 1 tablespoon</td>
<td>1.9</td>
<td>10</td>
</tr>
<tr>
<td>Spinach, boiled, ½ cup</td>
<td>1.9</td>
<td>10</td>
</tr>
<tr>
<td>Broccoli, chopped, boiled, ½ cup</td>
<td>1.2</td>
<td>6</td>
</tr>
<tr>
<td>Soybean oil, 1 tablespoon</td>
<td>1.1</td>
<td>6</td>
</tr>
</tbody>
</table>
Vitamin E

Dietary supplements

Supplements of vitamin E typically provide only alpha-tocopherol, although "mixed" products containing other tocopherols and even tocotrienols are available. Naturally occurring alpha-tocopherol exists in one stereoisomeric form. In contrast, synthetically produced alpha-tocopherol contains equal amounts of its eight possible stereoisomers; serum and tissues maintain only four of these stereoisomers [6]. A given amount of synthetic alpha-tocopherol (listed on labels as "DL" or "dl") is therefore only half as active as the same amount (by weight in mg) of the natural form (labeled as "D" or "d"). People need approximately 50% more IU of synthetic alpha tocopherol from dietary supplements and fortified foods to obtain the same amount of the nutrient as from the natural form.

Most vitamin-E-only supplements provide ≥100 IU of the nutrient. These amounts are substantially higher than the RDAs. The 1999-2000 National Health and Nutrition Examination Survey (NHANES) found that 11.3% of adults took vitamin E supplements containing at least 400 IU [8].

Alpha-tocopherol in dietary supplements and fortified foods is often esterified to prolong its shelf life while protecting its antioxidant properties. The body hydrolyzes and absorbs these esters (alpha-tocopheryl acetate and succinate) as efficiently as alpha-tocopherol [6].

Vitamin E Deficiency

Frank vitamin E deficiency is rare and overt deficiency symptoms have not been found in healthy people who obtain little vitamin E from their diets [6]. Premature babies of very low birth weight (<1,500 grams) might be deficient in vitamin E. Vitamin E supplementation in these infants might reduce the risk of some complications, such as those affecting the retina, but they can also increase the risk of infections [11].

Because the digestive tract requires fat to absorb vitamin E, people with fat-malabsorption disorders are more likely to become deficient than people without such disorders. Deficiency symptoms include peripheral neuropathy, ataxia, skeletal myopathy, retinopathy, and impairment of the immune response [6,12]. People with Crohn’s disease, cystic fibrosis, or an inability to secrete bile from the liver into the digestive tract, for example, often pass greasy stools or have chronic diarrhea; as a result, they sometimes require water-soluble forms of vitamin E, such as tocopheryl polyethylene glycol-1000 succinate [1].

Some people with abetalipoproteinemia, a rare inherited disorder resulting in poor absorption of dietary fat, require enormous doses of supplemental vitamin E (approximately 100 mg/kg or 5-10 g/day) [1]. Vitamin E deficiency secondary to abetalipoproteinemia causes such problems as poor transmission of nerve impulses, muscle weakness, and retinal degeneration that leads to blindness [13]. Ataxia and vitamin E deficiency (AVED) is another rare, inherited disorder in which the liver’s alpha-tocopherol transfer protein is defective or absent. People with AVED have such severe vitamin E deficiency that they develop nerve damage and lose the ability to walk unless they take large doses of supplemental vitamin E [14].

Vitamin E and Health

Many claims have been made about vitamin E’s potential to promote health and prevent and treat disease. The mechanisms by which vitamin E might provide this protection include its function as an antioxidant and its roles in anti-inflammatory processes, inhibition of platelet aggregation, and immune enhancement.

A primary barrier to characterizing the roles of vitamin E in health is the lack of validated biomarkers for vitamin E intake and status to help relate intakes to valid predictors of clinical outcomes [6]. This section focuses on four diseases and disorders in which vitamin E might be involved: heart disease, cancer, eye disorders, and cognitive decline.

Coronary heart disease

Evidence that vitamin E could help prevent or delay coronary heart disease (CHD) comes from several sources. In vitro studies have found that the nutrient inhibits oxidation of low-density lipoprotein (LDL) cholesterol, thought to be a crucial initiating step for atherosclerosis [6]. Vitamin E might also help prevent the formation of blood clots that could lead to a heart attack or venous thromboembolism [15].
Several observational studies have associated lower rates of heart disease with higher vitamin E intakes. One study of approximately 90,000 nurses found that the incidence of heart disease was 30% to 40% lower in those with the highest intakes of vitamin E, primarily from supplements [16]. Among a group of 5,133 Finnish men and women followed for a mean of 14 years, higher vitamin E intakes from food were associated with decreased mortality from CHD [17].

However, randomized clinical trials cast doubt on the efficacy of vitamin E supplements to prevent CHD [18]. For example, the Heart Outcomes Prevention Evaluation (HOPE) study, which followed almost 10,000 patients at high risk of heart attack or stroke for 4.5 years [19], found that participants taking 400 IU/day of natural vitamin E experienced no fewer cardiovascular events or hospitalizations for heart failure or chest pain than participants taking a placebo. In the HOPE-TOO followup study, almost 4,000 of the original participants continued to take vitamin E or placebo for an additional 2.5 years [20]. HOPE-TOO found that vitamin E provided no significant protection against heart attacks, strokes, unstable angina, or deaths from cardiovascular disease or other causes after 7 years of treatment. Participants taking vitamin E, however, were 13% more likely to experience, and 21% more likely to be hospitalized for, heart failure, a statistically significant but unexpected finding not reported in other large studies.

The HOPE and HOPE-TOO trials provide compelling evidence that moderately high doses of vitamin E supplements do not reduce the risk of serious cardiovascular events among men and women ≥50 years of age with established heart disease or diabetes [21]. These findings are supported by evidence from the Women’s Antithrombotic Vitamin and Estrogen study, in which 423 postmenopausal women with some degree of coronary stenosis took supplements with 400 IU vitamin E (type not specified) and 500 mg vitamin C twice a day or placebo for >4 years [22]. Not only did the supplements provide no cardiovascular benefits, but all-cause mortality was significantly higher in the women taking the supplements.

The latest published clinical trial of vitamin E’s effects on the heart and blood vessels of women included almost 40,000 healthy women ≥45 years of age who were randomly assigned to receive either 600 IU of natural vitamin E on alternate days or placebo and who were followed for an average of 10 years [23]. The investigators found no significant differences in rates of overall cardiovascular events (combined nonfatal heart attacks, strokes, and cardiovascular deaths) or all-cause mortality between the groups. However, the study did find two positive and significant results for women taking vitamin E: they had a 24% reduction in cardiovascular death rates, and those ≥65 years of age had a 26% decrease in nonfatal heart attack and a 49% decrease in cardiovascular death rates.

The most recent published clinical trial of vitamin E and men’s cardiovascular health included almost 15,000 healthy physicians ≥50 years of age who were randomly assigned to receive 400 IU synthetic alpha-tocopherol every other day, 500 mg vitamin C daily, both vitamins, or placebo [24]. During a mean followup period of 8 years, intake of vitamin E (and/or vitamin C) had no effect on the incidence of major cardiovascular events, myocardial infarction, stroke, or cardiovascular morality. Furthermore, use of vitamin E was associated with a significantly increased risk of hemorrhagic stroke.

In general, clinical trials have not provided evidence that routine use of vitamin E supplements prevents cardiovascular disease or reduces its morbidity and mortality. However, participants in these studies have been largely middle-aged or elderly individuals with demonstrated heart disease or risk factors for heart disease. Some researchers have suggested that understanding the potential utility of vitamin E in preventing CHD might require longer studies in younger participants taking higher doses of the supplement [25]. Further research is needed to determine whether supplemental vitamin E has any protective value for younger, healthier people at no obvious risk of CHD.

**Cancer**

Antioxidant nutrients like vitamin E protect cell constituents from the damaging effects of free radicals that, if unchecked, might contribute to cancer development [7]. Vitamin E might also block the formation of carcinogenic nitrosamines formed in the stomach from nitrates in foods and protect against cancer by enhancing immune function [26]. Human trials and surveys that attempted to associate vitamin E intake with cancer incidence have generally been inconclusive.

Some research links higher intakes of vitamin E with a decreased incidence of breast and prostate cancers [27], but the evidence is inconsistent. For example, an examination of the impact of dietary factors, including vitamin E, on the incidence of postmenopausal breast cancer in >18,000 women found no benefit from the vitamin [28]. Similarly, a prospective cohort study of >29,000 men found no association between dietary or supplemental vitamin E intake and prostate cancer risk, with one exception: among current smokers and men who had quit, vitamin E intakes of more than 400 IU/day were associated with a statistically significant 71% reduction in the risk of advanced prostate cancer [29]. A large randomized clinical trial began in 2001 to determine whether 7-12 years of daily supplementation with synthetic vitamin E (400 IU), with or without selenium (200 mcg), reduces the number of new prostate cancers in healthy men. The trial was discontinued in October 2008 when an analysis of the supplements, taken alone or together for an average of 5 years, did not prevent prostate cancer [30]. Study staff members will continue to monitor participants’ health for an additional 3 years.

One study of women in Iowa provides evidence that higher intakes of vitamin E from foods and supplements could decrease the risk of colon cancer, especially in women ≤65 years of age [31]. The overall relative risk for the highest quintile of intake (≥35.7 IU/day) was 0.32. However, prospective cohort studies of 87,998 women in the Nurses’ Health Study and 47,344 men in the Health Professionals Follow-up Study failed to replicate these results [32].

The American Cancer Society conducted an epidemiologic study examining the association between use of vitamin C and vitamin E supplements and bladder cancer mortality. Of the almost one million adults followed between 1982 and 1998, adults who took supplemental vitamin E for 10 years or longer had a reduced risk of death from bladder cancer [33]; vitamin C supplementation provided no protection.

Both the recently published HOPE-TOO Trial and Women’s Health Study evaluated whether vitamin E supplements might protect people from cancer. HOPE-TOO, which followed men and women ≥55 years of age with heart disease or diabetes for 7 years, found no significant differences in the number of new cancers or cancer deaths between the groups taking 400 IU/day vitamin E or a placebo [20]. In the Women's Health Study, in which healthy women ≥45 years of age received either 600 IU vitamin E every other day or a placebo for 10 years, the supplement did not reduce the risk of developing any form of cancer [23].

The inconsistent and limited evidence precludes any recommendations about using vitamin E supplements to prevent cancer.

**Eye disorders**

http://ods.od.nih.gov/factsheets/vitamine/
Age-related macular degeneration (AMD) and cataracts are among the most common causes of significant vision loss in older people. Their etiologies are usually unknown, but the cumulative effects of oxidative stress have been postulated to play a role. If so, nutrients with antioxidant functions, such as vitamin E, could be used to prevent or treat these conditions.

Prospective cohort studies have found that people with relatively high dietary intakes of vitamin E (e.g., 30 IU/day) have an approximately 20% lower risk of developing AMD than people with low intakes (e.g., <15 IU/day) [34,35]. However, two randomized controlled trials in which participants took supplements of vitamin E (500 IU/day dl-alpha-tocopherol in one study [36] and 111 IU/day dl-alpha-tocopheryl acetate combined with 20 mg/day beta-carotene in the other [37]) or a placebo failed to show a protective effect for vitamin E on AMD. The Age-Related Eye Disease Study (AREDS), a large randomized clinical trial, revealed that participants with early-stage AMD could slow the progression of their disease by taking a daily supplement of vitamin E (400 IU dl-alpha-tocopheryl acetate), vitamin C (500 mg), beta-carotene (15 mg), zinc (80 mg), and copper (2 mg) for an average of 6.3 years compared to participants taking a placebo [38].

Several observational studies have revealed a potential relationship between vitamin E supplements and the risk of cataract formation. One prospective cohort study found that lens clarity was superior in participants who took vitamin E supplements and those with higher blood levels of the vitamin [39]. In another study, long-term use of vitamin E supplements was associated with slower progression of age-related lens opacification [40]. However, in the randomized AREDS study, the use of the vitamin E-containing supplement package had no apparent effect on the development or progression of cataracts over 7 years [41].

Overall, the available evidence is inconsistent with respect to whether vitamin E supplements, taken alone or in combination with other antioxidants, can reduce the risk of developing AMD or cataracts. However, the formulation of vitamin E, other antioxidants, zinc, and copper used in AREDS holds promise for slowing the progression of AMD in people with early-stage disease. Additional information about the dietary supplements used in AREDS is available at http://www.nei.nih.gov/areds2. AREDS 2, a followup study, will determine whether a modified combination of dietary supplements can further slow the progression of vision loss from AMD; further information is available at http://www.nei.nih.gov/areds2.

Cognitive decline

The brain has a high oxygen consumption rate and abundant polyunsaturated fatty acids in the neuronal cell membranes. Researchers hypothesize that if cumulative free-radical damage to neurons over time contributes to cognitive decline and neurodegenerative diseases, such as Alzheimer's disease, then ingestion of sufficient or supplemental antioxidants (such as vitamin E) might provide some protection [42]. This hypothesis was supported by the results of a clinical trial in 341 patients with Alzheimer's disease of moderate severity who were randomly assigned to receive a placebo, vitamin E (2,000 IU/day dl-alpha-tocopherol), a monoamine oxidase inhibitor (selegiline), or vitamin E and selegiline [42]. Over 2 years, treatment with vitamin E and selegiline, separately or together, significantly delayed functional deterioration and the need for institutionalization compared to placebo. However, participants taking vitamin E experienced significantly more falls.

Vitamin E consumption from foods or supplements was associated with less cognitive decline over 3 years in a prospective cohort study of institutionalization compared to placebo. However, participants taking vitamin E experienced significantly more falls.

In summary, most research results do not support the use of vitamin E supplements by healthy or mildly impaired individuals to maintain cognitive performance or slow its decline with normal aging [46]. More research is needed to identify the role of vitamin E, if any, in the management of cognitive impairment [47].

Health Risks from Excessive Vitamin E

Research has not found any adverse effects from consuming vitamin E in food [6]. However, high doses of alpha-tocopherol supplements can cause hemorrhage and interrupt blood coagulation in animals, and in vitro data suggest that high doses inhibit platelet aggregation. Two clinical trials have found an increased risk of hemorrhagic stroke in participants taking alpha-tocopherol; one trial included Finnish male smokers who consumed 50 mg/day for an average of 6 years [48] and the other trial involved a large group of male physicians in the United States who consumed 400 IU every other day for 8 years [24]. Because the majority of physicians in the latter study were also taking aspirin, this finding could indicate that vitamin E has a tendency to cause bleeding.

The FNB has established ULs for vitamin E based on the potential for hemorrhagic effects (see Table 3). The ULs apply to all forms of supplemental alpha-tocopherol, including the eight stereoisomers present in synthetic vitamin E. Doses of up to 1,000 mg/day (1,500 IU/day of the natural form or 1,100 IU/day of the synthetic form) in adults appear to be safe, although the data are limited and based on small groups of people taking at least 2,000 IU for a few weeks or months. Long-term intakes above the UL increase the risk of adverse health effects [6]. Vitamin E ULs for infants have not been established.

### Table 3: Tolerable Upper Intake Levels (ULs) for Vitamin E [6]

<table>
<thead>
<tr>
<th>Age</th>
<th>Male</th>
<th>Female</th>
<th>Pregnancy</th>
<th>Lactation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-3 years</td>
<td>200 mg (300 IU)</td>
<td>200 mg (300 IU)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4-8 years</td>
<td>300 mg (450 IU)</td>
<td>300 mg (450 IU)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9-13 years</td>
<td>600 mg (900 IU)</td>
<td>600 mg (900 IU)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14-18 years</td>
<td>800 mg (1,200 IU)</td>
<td>800 mg (1,200 IU)</td>
<td>800 mg (1,200 IU)</td>
<td>800 mg (1,200 IU)</td>
</tr>
<tr>
<td>19+ years</td>
<td>1,000 mg (1,500 IU)</td>
<td>1,000 mg (1,500 IU)</td>
<td>1,000 mg (1,500 IU)</td>
<td>1,000 mg (1,500 IU)</td>
</tr>
</tbody>
</table>
Two meta-analyses of randomized trials have raised questions about the safety of large doses of vitamin E, including doses lower than the UL. These meta-analyses linked supplementation to small but statistically significant increases in all-cause mortality. One analysis found an increased risk of death at doses of ≥400 IU/day, although the risk began to increase at 150 IU [49]. In the other analysis of studies of antioxidant supplements for disease prevention, the highest quality trials revealed that vitamin E, administered singly (dose range 10 IU-5,000 IU/day; mean 569 IU) or combined with up to four other antioxidants, significantly increased mortality risk [50].

The implications of these analyses for the potential adverse effects of high-dose vitamin E supplements are unclear [51-54]. Participants in the studies included in these analyses were typically middle-aged or older and had chronic diseases or related risk factors. These participants often consumed other supplements in addition to vitamin E. Some of the studies analyzed took place in developing countries in which nutritional deficiencies are common. A review of the subset of studies in which vitamin E supplements were given to healthy individuals for the primary prevention of chronic disease found no convincing evidence that the supplements increased mortality [55].

**Interactions with Medications**

Vitamin E supplements have the potential to interact with several types of medications. A few examples are provided below. People taking these and other medications on a regular basis should discuss their vitamin E intakes with their healthcare providers.

**Anticoagulant and antiplatelet medications**

Vitamin E can inhibit platelet aggregation and antagonize vitamin K-dependent clotting factors. As a result, taking large doses with anticoagulant or antiplatelet medications, such as warfarin (Coumadin®), can increase the risk of bleeding, especially in conjunction with low vitamin K intake. The amounts of supplemental vitamin E needed to produce clinically significant effects are unknown but probably exceed 400 IU/day [56].

**Simvastatin and niacin**

Some people take vitamin E supplements with other antioxidants, such as vitamin C, selenium, and beta-carotene. This collection of antioxidant ingredients blunted the rise in high-density lipoprotein (HDL) cholesterol levels, especially levels of HDL2, the most cardioprotective HDL component, among people treated with a combination of simvastatin (brand name Zocor®) and niacin [57,58].

**Chemotherapy and radiotherapy**

Oncologists generally advise against the use of antioxidant supplements during cancer chemotherapy or radiotherapy because they might reduce the effectiveness of these therapies by inhibiting cellular oxidative damage in cancerous cells [59,60]. Although a systematic review of randomized controlled trials has called this concern into question [61], further research is needed to evaluate the potential risks and benefits of concurrent antioxidant supplementation with conventional therapies for cancer.

**Vitamin E and Healthful Diets**

According to the 2005 *Dietary Guidelines for Americans*, "nutrient needs should be met primarily through consuming foods. Foods provide an array of nutrients and other compounds that may have beneficial effects on health. In certain cases, fortified foods and dietary supplements may be useful sources of one or more nutrients that otherwise might be consumed in less than recommended amounts. However, dietary supplements, while recommended in some cases, cannot replace a healthful diet."

The *Dietary Guidelines for Americans* describes a healthy diet as one that:

- Emphasizes a variety of fruits, vegetables, whole grains, and fat-free or low-fat milk and milk products.
  - Vitamin E is found in green leafy vegetables, whole grains, and fortified cereals.
- Includes lean meats, poultry, fish, beans, eggs, and nuts.
  - Nuts are good sources of vitamin E.
- Is low in saturated fats, trans fats, cholesterol, salt (sodium), and added sugars.
  - Vitamin E is commonly found in vegetable oils.
- Stays within your daily calorie needs.


**References**

Vitamin E

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Disclaimer

Reasonable care has been taken in preparing this document and the information provided herein is believed to be accurate. However, this information is not intended to constitute an "authoritative statement" under Food and Drug Administration rules and regulations.

About ODS

The mission of the Office of Dietary Supplements (ODS) is to strengthen knowledge and understanding of dietary supplements by evaluating scientific information, stimulating and supporting research, disseminating research results, and educating the public to foster an enhanced quality of life and health for the U.S. population.

General Safety Advisory

Health professionals and consumers need credible information to make thoughtful decisions about eating a healthful diet and using vitamin and mineral supplements. These Fact Sheets provide responsible information about the role of vitamins and minerals in health and disease. Each Fact Sheet in this series received extensive review by recognized experts from the academic and research communities. The information is not intended to be a substitute for professional medical advice. It is important to seek the advice of a physician about any medical condition or symptom. It is also important to seek the advice of a physician, registered dietitian, pharmacist, or other qualified health professional about the appropriateness of taking dietary supplements and their potential interactions with medications.

[References]

Natural Medicines Comprehensive Database. Vitamin E. http://www.NaturalDatabase.com

http://ods.od.nih.gov/factsheets/vitamine/