Vitamins A and E and Carotenoids

Sources and Physiological Functions

Vitamins A (retinol) and E (*alpha*-tocopherol) and carotenoids are fat-soluble micronutrients. These micronutrients are also antioxidants. They are found in many foods, including some vegetables, fruits, meats, and animal products. Fish-liver oils, liver, egg yolks, butter, and cream are known for their higher content of vitamin A. Nuts and seeds are particularly rich sources of vitamin E. At least 700 carotenoids—fat-soluble red and yellow pigments—are found in nature (Britton 2004). Americans consume 40–50 of these carotenoids, primarily in fruits and vegetables (Khachik 1992). They consume smaller amounts in poultry products, including egg yolks, and in seafoods (Boylston 2007). Eight different carotenoids are easily measured in human serum: *alpha*-carotene, *cis*- and *trans*-*beta*-carotene, *beta*-cryptoxanthin, lutein, *cis*- and *trans*-lycopene, and zeaxanthin. The largest sources of carotenes are orange-colored fruits and vegetables such as carrots, pumpkins, and mangos. Lutein and zeaxanthin are also found in dark green leafy vegetables, where any orange coloring is overshadowed by chlorophyll. *Trans*-lycopene is obtained primarily from tomatoes, tomato products, and some fruits. For information on the carotenoid content of foods, see the USDA National Nutrient Database for Standard Reference, Release 28 (U.S. Department of Agriculture 2023).

Vitamin A found in foods that come from animal sources is called preformed vitamin A. Some carotenoids found in colorful fruits and vegetables are called provitamin A because they are metabolized in the body to vitamin A. Among the carotenoids, *beta*-carotene, whose structure can yield two retinol molecules upon cleavage, has the most significant provitamin A activity. Approximately 12 micrograms (µg) of dietary *beta*-carotene can provide the equivalent of 1 µg of retinol. Other provitamin A carotenoids, such as *alpha*-carotene and *beta*-cryptoxanthin, are half as active as *beta*-carotene (Institute of Medicine 2000). The bioconversion of carotenoids to vitamin A is highly variable from person to person (Krinsky 2005). Retinyl esters serve as the storage form of vitamin A and are mostly concentrated in the liver.

The absorption of fat-soluble micronutrients from the gastrointestinal tract depends on processes responsible for fat absorption or metabolism. People with conditions resulting in fat malabsorption (e.g., celiac disease, Crohn’s disease, pancreatic disorders, bariatric surgery) can develop vitamin A deficiency over time. Vitamin A also has interactions with other nutrients. Iron and zinc deficiency can affect vitamin A metabolism and the transport of vitamin A stores from the liver to body tissues (Institute of Medicine 2001). The absorption of carotenoids from foods is highly dependent on cooking techniques that break down plant cell walls and release carotenoids. Absorption also depends on dietary fat to enhance carotenoid uptake (Krinsky 2005). The liver regulates the concentration of vitamin A in circulation by releasing stored retinyl esters as needed. Only when liver reserves are nearly exhausted does serum vitamin A fall into the deficient range (Napoli 2006). Variation in serum carotenoid concentrations among people in the United States is relatively large, primarily reflecting wide-ranging differences in dietary intake (Lacher 2005).

Vitamin E activity is derived from at least eight naturally occurring tocopherols, the most potent of which is *alpha*-tocopherol. Other less active forms of vitamin E are plentiful in the U.S. diet, with *gamma*-tocopherol being the predominant form. For American adults, the most commonly consumed sources of *alpha*-tocopherol are mixed foods (spaghetti sauce, pizza and chili), fried potatoes, salad dressings, and bakery goods (Ahuja 2004). Other important sources are tomatoes, eggs, nuts and seeds, and snack foods. Plasma concentrations of tocopherols vary widely among healthy individuals and are highly correlated with plasma lipid concentrations (Ford 1999; Ford 2006).

Health Effects

Inadequate or excessive intake of vitamins A or E can lead to various disorders. For example, vitamin A deficiency, considered to be the main cause of childhood blindness in low-income countries (Roodhooft 2002), is a rare condition in the United States. Prominent signs of vitamin A deficiency include night blindness, corneal thinning, and conjunctival metaplasia. Vitamin A is also essential for proper immune function, epithelial growth and repair, bone growth, reproduction, and normal embryonic and fetal development (Tanumihardjo 2016; West 2006). Carotenemia, a common condition resulting from ingestion of excessive amounts of carrots, is a benign condition (Lascari 1981). Acute toxicity resulting from single or short-term large doses of preformed vitamin A is characterized by nausea, vomiting, headache, vertigo, blurred vision, increased cerebrospinal fluid pressure, and lack of muscular coordination. Central nervous system effects, liver abnormalities, bone and skin changes, and other nonspecific adverse effects can be indicative of chronic hypervitaminosis A. Consuming excess amounts of vitamin A during early pregnancy may lead to serious birth defects (Abadie 2023).

Serum or plasma concentrations of carotenoids are considered among the best biological markers for fruit and vegetable intake. The strongest dietary predictors of serum carotenoid concentrations are fruits (sources of *beta*-cryptoxanthin), carrots and root vegetables (sources of carotenes), and tomato products (sources of *trans*-lycopene) (Al-Delaimy 2005). Research studies have shown inconsistencies in the relationship between carotenoid intake and protection from cancer. Carotenoids in foods, even when consumed over long periods and in large amounts are not known to produce adverse health effects. However, results of intervention studies of smokers who used 20–30 milligrams (mg) of *beta*-carotene per day showed that this group had more lung cancers than placebo-treated groups (Albanes 1996; Redlich 1998). Higher dietary intakes of vitamins E and C and beta-carotene have been shown to reduce the risk of type 2 diabetes (Lampousi 2024). The progression of age-related macular degeneration slowed with the use of high-dose supplementation containing vitamins C and E, beta-carotene, and zinc, while there was no effect on the development or progression of age-related lens opacities (Age-Related Eye Disease Study Research Group 2001a and 2001b).

Vitamin E deficiency occurs only rarely in people, and symptoms of overt deficiency in people consuming low vitamin E diets have never been described (Institute of Medicine 2000). The main manifestation of vitamin E deficiency is peripheral neuropathy characterized by the degeneration of the large-caliber axons of sensory neurons (Institute of Medicine 2000). The upper limit (UL) for vitamin E intake (1,000 mg/day) is based on hemorrhagic effects; however, a causal association between excess *alpha*-tocopherol intake in apparently healthy individuals and adverse health outcomes has not consistently been shown (Institute of Medicine 2000). Studies evaluating tocopherols to reduce the risk for cardiovascular disease demonstrated inconsistent findings (Moss and Ramji 2016). The American Heart Association advises that antioxidant supplements (such as vitamins E and *beta*-carotene) should not be used for primary or secondary prevention of cardiovascular disease (U.S. Preventive Services Task Force 2022). Nevertheless, the American Heart Association recommends consuming food sources of antioxidant nutrients, principally from a variety of plant-derived foods such as fruits, vegetables, whole grains, and vegetable oils.

Intake Recommendations

The National Academy of Sciences has established dietary reference intake values for vitamins A and E by determining the Adequate Intake (AI) for infants and the Recommended Dietary Allowance (RDA) for older age groups (Institute of Medicine 2000 and 2001). The RDA for vitamin A in retinol equivalents is 900 µg/day for men and 700 µg/day for women. For children and adolescents (1–18 years), the RDA ranges from 300–900 µg/day. For infants (0–12 months), the AI is set at 400–500 µg/day of retinol equivalents. The Tolerable Upper Intake Level (UL) for adults is set at 3,000 µg/day of preformed vitamin A, whereas the UL for infants (600 µg/day), younger children 1–8 years (600–900 µg/day), older children 9–13 years (1,700 µg/day) and adolescents 14–18 years (2,800 µg/day) are age-dependent. For adults, the RDA for vitamin E is 15 mg/day of *alpha*-tocopherol; for children and adolescents (1–18 years), the RDA ranges from 6–15 mg/day. There is no RDA for other forms of vitamin E, such as *gamma*-tocopherol. The UL for vitamin E, which applies to all eight stereoisomers of *alpha*-tocopherol, is 1,000 mg/day for adults. The Institute of Medicine could not establish a UL for infants and so only recommended food and formula sources of dietary intake. The UL for vitamin E for children and adolescents ranges from 200–800 mg/day. Although no quantitative recommendations are available for the intake of carotenoids, existing recommendations support increased consumption of carotenoid-rich fruits and vegetables. For those consuming a 2,000 kcal/day diet, current public health guidelines advise a daily intake of three servings of vegetables and two servings of fruit to ensure adequate nutrient intake (U.S. Department of Agriculture and U.S. Department of Health and Human Services 2026).

Biochemical Indicators and Cutoff Values

The Biomarkers of Nutrition for Development (BOND) project published a comprehensive review covering aspects of vitamin A biology and biomarkers (Tanumihardjo 2016). Vitamin A status assessment is not trivial for several reasons: serum retinol concentrations are under homeostatic control and they are depressed during infection and inflammation because retinol binding protein (RBP) is a negative acute-phase reactant. There are several biochemical markers of vitamin A status: serum retinol, RBP, breast-milk retinol, dose-response tests, isotope dilution approaches, and serum retinyl esters. However, the most commonly used indicator is serum retinol. The gold standard to determine inadequate vitamin A status is through hepatic biopsy, but this invasive procedure is unsuitable for population studies. RBP, the carrier of retinol, is sometimes used as a surrogate for retinol in population studies. However, RBP concentrations can be influenced by inflammation, iron status, pregnancy, obesity, and protein-energy malnutrition (Engle-Stone 2011). Thus, when assessing vitamin A status based on measured RBP concentrations, serum retinol concentrations should be determined in a subset of the population to assess the ratio of retinol to RBP, which could then be used to predict serum retinol from serum RBP (Tanumihardjo 2016).

A diagnosis of vitamin A or E deficiency is supported by measuring serum biomarkers. People with serum retinol concentrations of less than 20 µg/dL are considered vitamin A deficient, and those with serum concentrations of less than 10 µg/dL are considered severely deficient (Institute of Medicine 2001; WHO 2011). Serum retinol values do not always reflect total body status because of homeostatic control. Therefore, the values are often not useful for assessing the vitamin A status of individuals. Additional tests may be required to confirm vitamin A deficiency when 20 µg/dL is used as a cutoff (Gibson 2005). The distribution of serum retinol concentrations in a population together with the prevalence of individuals with serum retinol values below a given cutoff provide important information about the vitamin A status of a population. The World Health Organization recommends using the prevalence of serum retinol concentrations of less than or equal to 20 µg/dL (0.7 µmol/L) to define public health problems involving vitamin A as mild (2–9%), moderate (10–19%) or severe (≥20%) (WHO 2011). In chronic hypervitaminosis A, serum concentrations are generally greater than 100 µg/dL (Bendich 1989). Carotenoid deficiency has no defined serum concentrations. The laboratory diagnosis of vitamin E deficiency is based on serum concentrations of *alpha*-tocopherol (less than 500 µg/dL or less than 0.8 mg of *alpha*-tocopherol per gram of total lipids) (Johnson 2025). Such concentrations are based on *in vitro* hydrogen peroxide-induced red blood cell lysis and not on clinical deficiency symptoms (Institute of Medicine 2000).

Analytical Methods

Serum or plasma retinol and vitamin E are measured using high-performance liquid chromatography (HPLC) with ultraviolet (UV) detection. RBP is measured through enzyme-linked immunosorbent assay (ELISA). Serum or plasma concentrations of carotenoids are measured using HPLC and visible light (450 nm) absorbance.

Clinical laboratories typically use conventional units for serum concentrations of these fat-soluble micronutrients (microgram per deciliter, [µg/dL]). For vitamin A, conversion factors from conventional to International System of Units (SI) are 1 µg/dL = 0.0349 micromole per liter (µmol/L).

For vitamin E, conversion factors from conventional to SI units are 1 µg/dL = 0.02322 µmol/L. Depending on its molecular weight, each carotenoid has a specific conversion factor.

International reference materials for vitamins A and E and carotenoids are available from the U.S. National Institute of Standards and Technology (SRM 968 Fat-soluble vitamins, carotenoids, cholesterol, and 25(OH)D_3 in human serum). For most laboratories participating in the NIST external quality assurance program, standardized HPLC methods for measuring fat-soluble micronutrients showed consistent agreement for values (Duewer 2022). The U.S. College of American Pathologists offers an external quality assurance program for serum vitamin A and E concentration measurements (Bone Markers and Vitamins survey).

Findings from NHANES

The National Health and Nutrition Examination Survey (NHANES) is the only source for nationally representative data on vitamins A and E and carotenoids for the U.S. population (Pfeiffer 2026a). Since 1971, various fat-soluble micronutrients have been measured in the serum of participants in NHANES. In NHANES III (1988–1994), clinically low concentrations of serum retinol were uncommon in U.S. residents aged 4 years and older, although racial/ethnic and socioeconomic differences existed (Ballew 2001). Researchers found variations in serum carotenoid concentrations by ethnicity and sex for adults, children, and adolescents (Ford 2000; Ford 2002). Sociodemographic variations in serum concentrations of *alpha*-tocopherol among U.S. adults in NHANES III (Ford 1999) and *alpha*- and *gamma*-tocopherol in NHANES 1999–2000 (Ford 2006) have also been documented. Applying the most common cutoff value for serum *alpha*-tocopherol concentrations in NHANES 1999–2000 (500 µg/dL), demonstrated a low prevalence of vitamin E deficiency, despite the fact that the U.S. Department of Agriculture-estimated dietary intakes of vitamin E were low and that most of the U.S. population did not meet dietary intake recommendations (Ford 2006). After adjusting for demographic changes over time, investigators found no or small changes (<2 percentage points) in the prevalence of vitamin A and E deficiency (vitamin A <20 µg/dL; vitamin E <500 µg/dL) and vitamin A excess (vitamin A >100 µg/dL) between NHANES 1999–2000 and 2017–2018 (Pfeiffer 2026b).

A multiple regression analysis of NHANES 2003–2006 vitamin A, vitamin E, and carotenoid biomarkers showed that together, sociodemographic (age, education, income, race-ethnicity, and sex) and lifestyle (alcohol consumption, body mass index, dietary supplement use, physical activity, and smoking) variables explained from 10% (carotenes) to 23% (vitamin E) of the biomarker variability (Schleicher 2013). Although lipid adjustment (using lipid-altering medications and adjusting for total cholesterol) explained additional variability for these biomarkers, it appeared to be largely independent of sociodemographic and lifestyle variables. Age, sex, and race and Hispanic origin differences in these biomarker concentrations observed in crude univariate analysis generally remained significant after adjusting for sociodemographic and lifestyle variables. Use of dietary supplements was an important correlate of these biomarkers. Smoking and BMI were also important correlates of carotenoids (Schleicher 2013). A second multiple regression analysis of NHANES 2003–2006 showed that after controlling for demographic variables, smoking, supplement use, fasting, inflammation, and renal function, fasting was associated with significantly higher vitamin E concentrations (3.7%), whereas inflammation was associated with significantly lower vitamin A (-8.2%), carotenoid (-18.3%), and xanthophyll (-20.6%) concentrations. Impaired renal function was associated with significantly higher vitamin A (20.2%) concentrations (Haynes 2013). Pregnancy (in women 20–49 years of age) was associated with significantly higher vitamin E (22.2%), carotenoid (11.9%), and xanthophyll (32.3%) concentrations and significantly lower vitamin A (-21.5%) concentrations (Haynes 2013). A third multiple regression analysis of NHANES 2003–2006 evaluated sociodemographic, lifestyle, and physiologic factors as potential confounders or effect modifiers of the relationship between biomarkers and intake. The investigation demonstrated that dietary supplement use explains more variance in serum vitamin A and E concentrations than 24-hour dietary intake from food only (Sternberg 2026).

For more information on these fat-soluble micronutrients, see the Institute of Medicine's Dietary Reference Intake reports (Institute of Medicine 2000 and 2001) and the vitamin fact sheets from the National Institutes of Health, Office of Dietary Supplements (https://ods.od.nih.gov/factsheets/list-VitaminsMinerals/).

Data in the 2026 tables

Data presented are from univariate analysis that was not adjusted for demographic variables (e.g., age, sex, race and Hispanic origin) or other blood concentration determinants (e.g., dietary intake, supplement use, smoking, BMI). Data for the fat-soluble micronutrients (vitamins A, E, and carotenoids) were available from different NHANES cycles and population subgroups. They were measured by a single assay panel employing HPLC separation and using UV or visible light detection (HPLC-UV/vis).

Fat-soluble micronutrient biomarker	**NHANES cycle and population subgroup**
Vitamin A (retinol)	1999–2002: persons ≥3 years of age 2005–2006: persons ≥6 years of age 2017–2018: persons ≥6 years of age
Retinyl palmitate and retinyl stearate	1999–2002: persons ≥3 years of age 2005–2006: persons ≥6 years of age 2017–2018: persons ≥6 years of age
Vitamin E (*alpha*-tocopherol)	1999–2002: persons ≥3 years of age 2005–2006: persons ≥6 years of age 2017–2018: persons ≥6 years of age
gamma-Tocopherol	1999–2002: persons ≥3 years of age 2005–2006: persons ≥6 years of age 2017–2018: persons ≥6 years of age
Carotenoids: • *alpha*-Carotene • *trans-beta*-Carotene • *cis-beta*-Carotene • *beta*-Cryptoxanthin • *alpha*-Cryptoxanthin (only for 2017–2018) • Lutein and zeaxanthin • *trans*-Lycopene • Total lycopene *(cis*- and *trans*-) (not for 2001–2002)	2001–2002: persons ≥3 years of age 2005–2006: persons ≥6 years of age 2017–2018: persons ≥6 years of age

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