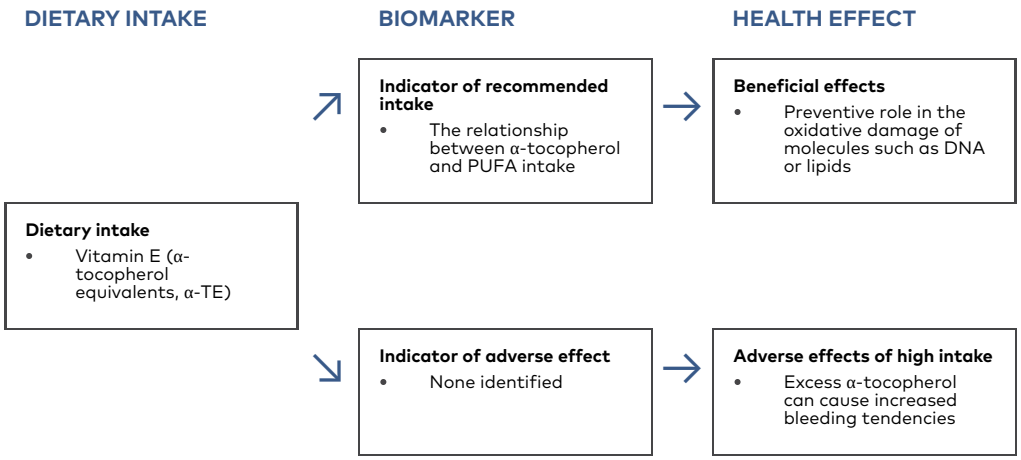
Vitamin E



	Females	Males
Provisional AR (α-TE/d)	8	9
AI (α-TE/d)	10	11

For more information about the health effects, please refer to the background paper by Essi Marjatta Hantikainen and Ylva Trolle Lagerros (Hantikainen & Lagerros, 2023).

Dietary sources and intake. Vitamin E is used as a generic term for molecules that possess the biological effects of α-tocopherol, of which four tocopherols (α-, β-, γ-, and δ) and four tocotrienols (α-, β-, γ-, and δ) occur naturally. In NNR2023, vitamin E activity is confined to α-tocopherol, since α-tocopherol is the only form that is recognized to meet human requirements. The naturally occurring α-tocopherol in foods is the stereoisomer RRR-α-tocopherol (Hantikainen & Lagerros, 2023). Food sources of vitamin E are vegetable oils, vegetable oil-based spreads, nuts, seeds, and egg yolk. The average vitamin E intake ranges from 7.8 to 14.9 mg /d (Lemming & Pitsi, 2022).

Main functions. Vitamin E is a fat-soluble antioxidant that also exhibits non-antioxidant activities, such as modulation of gene expression, inhibition of cell proliferation and regulation of bone mass. The main biochemical function of α-tocopherol is antioxidant activity. α-tocopherol is present in cell membranes. It has a significant preventive role in the oxidative damage of molecules such as

DNA or lipids by neutralizing free radicals and breaking the chain reaction in the oxidation of PUFA. Increased dietary intake of PUFA decreases vitamin E concentrations in plasma and tissues (Hantikainen & Lagerros, 2023).

Indicator for recommended intake. EFSA found that there was insufficient data on markers of a-tocopherol intake/status/function to derive the requirement, and instead set AIs based on observed dietary intakes in healthy populations with no apparent α-tocopherol deficiency (EFSA, 2015g). The IOM based the adult requirements for vitamin E on prevention of hydrogen peroxide–induced haemolysis (Hantikainen & Lagerros, 2023; Raederstorff et al., 2015).

Main data gaps. Some of the evidence related to chronic diseases relies on findings from observational studies only, rather than RCTs. The effect of vitamin E can therefore not be fully separated from other nutritional factors. In addition, several studies suggest that besides α-tocopherol, other tocopherols and tocotrienols might have important functions and beneficial effects on various chronic disease outcomes.

Deficiency and risk groups. Vitamin E deficiency due to low dietary intake has not been described in healthy adults. However, deficiency can be caused by prolonged fat malabsorption due to genetic defects in lipoprotein transport or in the hepatic α-tocopherol transfer protein, or fat malabsorption syndromes, such as cholestatic liver disease or cystic fibrosis. Vitamin E deficiency is more frequently found in children, likely due to limited stores and rapid growth. Specifically, premature and very low birth weight infants are at risk and symptoms such as haemolytic anaemia, thrombocytosis, and oedema have been reported.

Dietary reference values. To estimate the AI for vitamin E the NNR Committee considered a basal vitamin E requirement (4 mg) plus a factor based on the dietary intake of PUFA. The recommended intake of PUFA is 5–10 E%. For calculating the AI, the lower value of this range is used (i.e., 5 E%). The estimated optimal vitamin E:PUFA ratio, which ranges from 0.4 to 0.6 mg RRR-α-tocopherol/g of PUFA in the diet, suggests that a ratio of 0.5 mg α-TE/g of PUFA can reasonably be used (Hantikainen & Lagerros, 2023; Raederstorff et al., 2015). The AI is set to 10 α-TE/day in females and 11 α-TE/day in males. The provisional AR is set to 8 α-TE/day (females) and 9 α-TE/day (males). The UL of vitamin E is 300 mg/d.