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Vitamin E intake from food and supplements and the association with plasma α-tocopherol and γ-tocopherol concentrations

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A review of randomised controlled trials found that vitamin E supplementation is associated with increased all-cause mortality[(1)]. Supplementation of vitamin E tends to be mainly α-tocopherol which causes γ-tocopherol concentrations to decrease and this might affect antioxidant and anti-inflammatory capacity[(2)]. We quantified, in a general population consuming commonly available supplement doses, how total food and supplement intake (total nutrient intake, TNI) of vitamin E were associated with plasma α-tocopherol and γ-tocopherol concentrations and explored threshold effects.

The Norfolk based European Prospective Investigation into Cancer (EPIC-Norfolk) recruited men and women, between 40–79 years old. They attended a health examination, at which height, weight and blood samples were taken. Plasma α-tocopherol and γ-tocopherol were determined by high performance liquid chromatography in a subsample ($n = 7482$). Participants completed a 7-day diet diary, from which TNI, expressed as α-tocopherol equivalents (α-TE) was calculated[(3)] forming three subgroups: non-supplement users (NSU, $n = 4166$), those consuming vitamin E in supplement form (SU + E, $n = 1307$) and those consuming a supplement which did not contain vitamin E (SU-E, $n = 1243$). Median (M) and Interquartile Range (IQR) of nutrient intakes and plasma concentrations were calculated for participants with complete data ($n = 6716$), followed by linear regression on log-transformed plasma concentrations, adjusted for sex, age, cholesterol concentration, body mass index, smoking, alcohol and energy intake. Obtained betas were exponentiated, representing the percentage increase or decrease of plasma concentrations per 10 mg α-TE increase in intake.

SU + E consumers had the highest α-tocopherol, but the lowest γ-tocopherol concentrations. TNI in the SU + E group attenuated the association with plasma, indicating that more extreme intakes did not increase plasma concentrations. This was confirmed by restricting analysis to intakes below 17 mg/d (NSU: $n = 3888$, SU-E: $n = 1144$, SU + E: $n = 547$), the intake below which linear associations have been established in vitamin E depleted participants[(4)]. This strengthened associations among SU + E and changed the direction with γ-tocopherol.

	Food only NSU		Food only SU-E		Food only SU + E		Food + Suppl SU + E	
Descriptive analysis (crude)	M	IQR	M	IQR	M	IQR	M	IQR
Vitamin E intake (mg α-TE/d)	9·3	7·0, 12·0	9·6	7·4, 12·3	9·5	7·4, 12·5	18·8	13·9, 30·8
α-tocopherol (μmol/L)	25·4	21·5, 29·9	26·5	22·6, 31·3	28·7	24·1, 35·2	28·7	24·1, 35·2
γ-tocopherol (μmol/L)	1·7	1·3, 2·3	1·6	1·3, 2·2	1·3	0·9, 1·9	1·3	0·9, 1·9
% Difference (Δ) in biomarker per 10 mg α-TE/d increase in vitamin E intake								
	Δ	95% CI	Δ	95% CI	Δ	95% CI	Δ	95% CI
α-tocopherol	+14	+12, +16	+11	+7, +14	+10	+5, +14	+1	+1, +1
γ-tocopherol	−7	−10, −4	−6	−12, +1	−5	−12, +3	−3	−3, −2
% Difference (Δ) in biomarker per 10 mg α-TE/d increase in vitamin E intake (max. 17 mg)								
	Δ	95% CI	Δ	95% CI	Δ	95% CI	Δ	95% CI
α-tocopherol	+17	+14, +20	+15	+10, +20	+10	+1, +20	+10	+3, +18
γ-tocopherol	−10	−15, −6	−5	−14, +4	+18	−1, +42	−11	−21, +3

In conclusion, supplement sources of vitamin E attenuated the association with biomarker data, indicating that a plateau level was reached. However, not including supplement sources reversed the direction of the association between intake and γ-tocopherol. The possible health effects of lower γ-tocopherol concentrations will be studied further.

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