

VITAMIN E AND ITS STATUS IN VEGETARIANS AND VEGANS

A.V. Galchenko

Junior Research Scientist, Federal Research Centre for Nutrition, Biotechnology and Food Safety (Moscow, Russia)

ORCID: 0000-0001-7286-5044

E-mail: gav.jina@gmail.com

R. Ranjit

Resident, Department of Oncology and Radiology, Medical Institute,
Peoples' Friendship University of Russia (RUDN University) (Moscow, Russia)

ORCID: 0000-0002-4255-4197

E-mail: rajesh.ranjit@mail.ru

Vitamin E is one of the fat-soluble vitamins. Currently, eight of its forms have been studied - these are α -, β -, γ - and δ -tocopherols and α -, β -, γ - and δ -tocotrienols. α -tocopherol has the greatest metabolic activity, since it binds most efficiently to serum carriers and is quickly delivered to the liver in order to be incorporated into lipoproteins.

Vitamin E is one of the most significant elements of the body's antioxidant defense. By preventing blood lipoproteins from free radical oxidation, it reduces the risk of atherosclerosis. Moreover, it averts platelet aggregation. In this way, vitamin E protects us from cardiovascular diseases. Vitamin E is also an immunomodulator, which has a significant effect on lymphocyte function.

Like other hydrophobic vitamins, tocopherols and tocotrienols can accumulate in tissues and cause toxic effects. The most pronounced manifestation of hypervitaminosis E is platelet dysfunction and hemorrhage.

Vegetable oils provide the majority of the dietary vitamin E. In particular, sunflower oil is one of the richest sources of vitamin E. Largely due to the fact that sunflower oil is common in Eastern Europe, vitamin E deficiency is quite rare in this region, which, however, cannot be said about population of North America or Southern Europe, where corn and olive oils are generally consumed, respectively.

Higher consumption of vegetable oils and oilseeds provides vegans with large amounts of vitamin E. Nonetheless, serum α -tocopherol concentrations are often low in those dietary groups. Primarily, this is associated with lower level of serum lipids in vegans because blood lipoproteins contain the majority of α -tocopherol. In case of vegans, the lower lipid level results lower vitamin E. Taking all these into account, the assessment of the serum α -tocopherol:cholesterol ratio comes to the fore. Even considering all the facts, there is no significant preponderance of this ratio among vegans and vegetarians. This is probably due to the fact that subjects from both plant-based and omnivorous groups in most studies had plasma lipoproteins saturated with vitamin E. However, this issue requires further research.

Key words: *tocopherol, tocotrienol, α -tocopherol:cholesterol, antioxidant, atherosclerosis, platelet aggregation, plant-based.*

For citation: Galchenko A.V., Ranjit R. Vitamin E and its status in vegetarians and vegans. Problems of biological, medical and pharmaceutical chemistry. 2022;25(6):13–21. <https://doi.org/10.29296/25877313-2022-06-02>

INTRODUCTION

Vitamin E is the collective name given for a group of fat-soluble compounds possessing distinctive antioxidant activities [1]. Vitamin E naturally exists in eight chemical forms (α -, β -, γ and δ -tocopherol; and α -, β -, γ -, and δ -tocotrienol) (Table 1) with varying levels of biological activity. α -tocopherol by so far is the most active form of vitamin E for humans [1]. This is due to the fact that although all the forms have similar antioxidant functions, only α -tocopherol has a high affinity towards hepatic α -tocopherol transfer protein [2]. It is a protein that carries α -tocopherol to the site where it is incorporated into lipoproteins and carried to other parts of the body [3].

PHYSIOLOGICAL ROLE OF VITAMIN E

Vitamin E is a fat-soluble antioxidant that scavenges peroxy radicals and takes part in the termina-

tion of the production of reactive oxygen species formed after unsaturated fatty acids undergo the oxidative process [4].

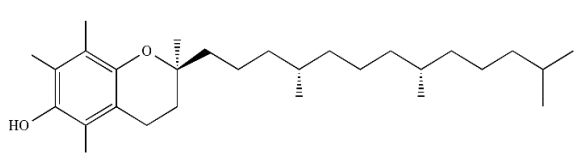
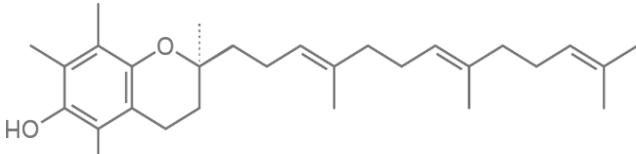
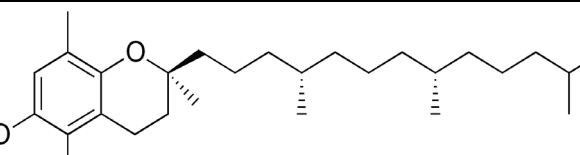
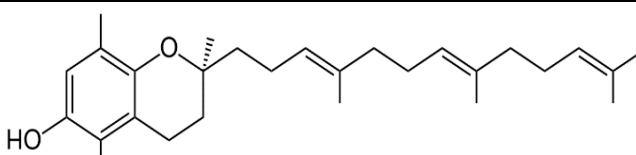
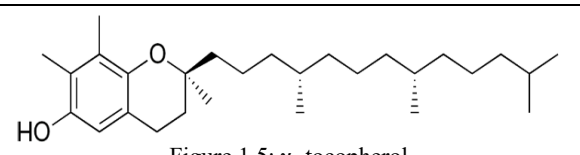
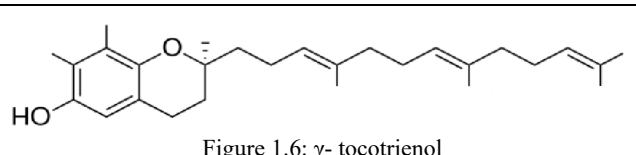
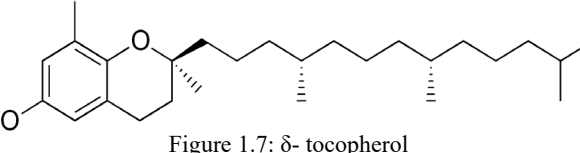
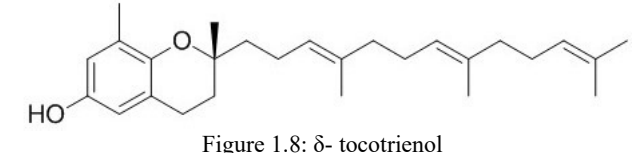
The mechanism underlying this function is that vitamin E has a higher affinity towards peroxy radicals than unsaturated fatty acids. Due to this, the chain reaction comes to an end and further oxidation of fatty acids does not occur [5]. During the process, vitamin E becomes oxidised and loses its anti-oxidant property. But vitamin C can reactivate its anti-oxidant ability by reducing vitamin E radicals [6].

Besides its obvious role as an antioxidant, vitamin E is also highly involved in immune function [1]. Vitamin E directly modulates T cell function by impacting its membrane integrity and by manipulating its system of signal transduction. Indirectly, it affects the release of inflammatory mediators produced by immune cells [7]. α -tocopherol is involved in the inhi-

bition of the activity of protein kinase C, which takes part in cell proliferation and differentiation of smooth muscle cells, platelets and monocytes [8]. It is believed that α -tocopherol increases protein kinase C- α dephosphorylation through the activation of protein phosphatase 2A, which subsequently inactivates protein kinase C by changing its phosphorylation state [9–11]. Moreover, if the inner linings of endothelial cells are rich in vitamin E, blood-cell components are

less likely to adhere to their surface. This side effect can be linked with the ability of vitamin E to reduce platelet cyclooxygenase activity and inhibit the formation of lipid peroxide, which has been directly found to inhibit platelet aggregation and release [12]. It also potentiates the arachidonic acid release, which, in turn, releases prostacyclin from the endothelium. The prostacyclin thus produced dilates blood vessels and inhibits platelet aggregation [13].

Table 1. Structures of different forms of vitamin E

Saturated phytyl tail	Unsaturated phytyl tail
 <p>Figure 1.1: α-tocopherol</p>	 <p>Figure 1.2: α-tocotrienol</p>
 <p>Figure 1.3: β-tocopherol</p>	 <p>Figure 1.4: β-tocotrienol</p>
 <p>Figure 1.5: γ-tocopherol</p>	 <p>Figure 1.6: γ-tocotrienol</p>
 <p>Figure 1.7: δ-tocopherol</p>	 <p>Figure 1.8: δ-tocotrienol</p>

HYPOVITAMINOSIS, VITAMIN E DEFICIENCY DISORDERS

There are certain groups of people who suffer from lack of vitamin E. Premature babies of very low birth weight (<1500 grams) might be at the risk of deficient in vitamin E. Vitamin E deficiency in these infants might raise some complications, such as those affecting the retina, by facilitating the accumulation of lipofuscin in the pigment epithelium of the retina [14]. In the same way, lack of vitamin E has also been linked with an increased risk of infectious diseases [15], like respiratory infections caused by viruses and bacteria [7].

Since the digestive tract requires fat to absorb fat-soluble vitamin – vitamin E, people with fat-malabsorption disorders are more likely to become de-

ficient as compared to people without such pathologies. Peripheral neuropathy, skeletal myopathy, ataxia, retinopathy, or impairment of the immune response are some deficiency symptoms [8,16]. People with cystic fibrosis, Crohn’s disease, or an inability to secrete bile from the liver into the digestive tract are more vulnerable to vitamin E deficiency [1].

Some people with abetalipoproteinemia, which is 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 problems like muscle weakness, poor transmission of nerve impulses, and retinal degeneration that leads to blindness [17]. Ataxia and vitamin E

deficiency (AVED) is another rare, inherited disorder in which the liver's α -tocopherol transfer protein is defective or absent. People with AVED have such severe vitamin E deficiency that they even develop nerve damage and lose the ability to walk unless they take very large doses of supplemental vitamin E [18]. Similarly, vitamin E deficiency has also been related to oral cancer [19].

Moreover, atherosclerosis is also associated with lack of vitamin E [20]. It is to be noted that vitamin E's circulation in the blood is accompanied by lipoproteins [21]. With the increase of serum lipid concentration, vitamin E shifts out of cellular membrane compartment into the circulating lipoproteins. In ailments, like in hepatic cholestasis and hypothyroidism, serum lipid concentration gets elevated, which consequently increases the serum level of vitamin E. In such situations, the underlying vitamin E deficiency might be missed [22, 23].

On the other hand, its deficiency could be overestimated in diseases like cystic fibrosis and when the patient is under statins [24, 25]. This is because cholesterol concentration in blood is low under such conditions [26, 27]. That is why, for more accurate determination of vitamin E status, vitamin E:lipid ratio gives clearer picture of vitamin E concentration. A plasma tocopherol concentration of 0.5 mg/dl (11.6 mmol/l) and a tocopherol:cholesterol ratio of 2.25 has been determined to be the lowest acceptable levels of vitamin E in the body [28]. It has been assumed that

plasma concentration of vitamin E of more 30 mmol/l could decrease the risk of cancer [29].

HYPERVITAMINOSIS E, VITAMIN E TOXICITY

Any adverse effect of over-consuming natural source of vitamin E through food has not been found yet [8]. However, high doses of α -tocopherol supplements, which may increase vitamin E concentration $>5.56 \mu\text{mol}/\text{mmol}$ cholesterol [30], may cause haemorrhage and interrupt the blood coagulation system [31] in accordance with above-mentioned mechanism. Two clinical trials have found an increased risk of haemorrhagic stroke in participants taking α -tocopherol; one trial was conducted in Finnish male smokers who consumed 50 mg/day for an average of 6 years [32] and the other trial involved a large group of male physicians in the United States who consumed 364 mg every other day for 8 years [33].

The Food and Nutrition Board of the USA has well established Upper Intake Levels (ULs) for vitamin E, which are based on the propensity of haemorrhagic effects (Table 2) [8].

The ULs apply to all forms of supplemental α -tocopherol, including the eight stereoisomers present in synthetic vitamin E. Upper dose of 1000 mg/day in adults are found to be safe, although the data are limited and based on small groups of people taking at least 2000 IU for a few weeks or months. In Russia, 150 mg/day is taken as tolerable UL [34]. Vitamin E ULs for infants have not been established yet.

Table 2. Tolerable Upper Intake Levels (ULs) for vitamin E for different age groups

Age in years	Upper Intake Levels, mg	
	Institute of Medicine, the USA [8]	Scientific Committee for Food, EU [35]
1-3	200	100
4-6	300	120
7-8		160
9-10	600	220
11-13		
14	800	260
15-17		300
18		
19+ years	1,000	

SOURCES OF VITAMIN E AND ITS DAILY REQUIREMENTS

The requirement for vitamin E is directly proportional to the intake of polyunsaturated fatty acids

(PUFAs), as vitamin E is responsible for scavenging free radicals generated by lipid peroxidation of PUFAs [28, 36]. Since vegetarians consume a higher amount of PUFAs than non-vegetarians [37], they require plenty of

vitamin E in their diet. It has been established that, when the primary PUFA in the diet is linoleic acid, the ratio of intake of α -tocopherol (in milligrams) to PUFA (in grams) should be 5:2 [28, 38–40]. In case of vitamin E status in blood, the α -tocopherol:cholesterol ratio of more than 2.5 mmol/mmol is believed to be optimal [24, 41]. Zino et al. [42] concluded that neither the tocopherol:cholesterol molar ratio nor concentrations of lipids or

lipoproteins were changed by increasing the daily consumption of fruits, juices and vegetables for 8 weeks.

Many sources of food provide vitamin E. Seeds, nuts and vegetable oils are among the best sources of tocopherols (Table 3). Similarly, significant amounts of vitamin E are available in green fortified cereals and leafy vegetables [43, 44]. The Adequate Intake (AI) for vitamin E for different age groups are illustrated in Table 4.

Table 3. Sources of vitamin E (α -tocopherol) [43, 44]

Food	Milligrams (mg) per serving
Wheat germ oil, 1 tablespoon	20.3
Sunflower oil, 1 cup	16.2
Safflower oil, 1 tablespoon	4.6
Peanut butter, 2 tablespoons	2.9
Corn oil, 1 tablespoon	1.9
Spinach, boiled, 1/2 cup	1.9
Olive oil, 1 tablespoon	1.9
Broccoli, chopped, boiled, 1/2 cup	1.2
Soybean oil, 1 tablespoon	1.1
Mango, sliced, 1/2 cup	0.7
Spinach, raw, 1 cup	0.6

Note: to convert 1 mg of vitamin E to IU, it should be multiplied by 1.1 for synthetic vitamin E or by 1.5 for natural vitamin E [45]. (1 mg d- α -tocopherol equivalence (natural α -tocopherol equivalence) = 1 mg d- α -tocopherol (natural α -tocopherol) = 1.35 mg dl- α -tocopherol (synthetic α -tocopherol) = 2.5-4 mg d- β -tocopherol (natural β -tocopherol) = 10 mg d- γ -tocopherol (natural γ -tocopherol)) [46].

Table 4. Adequate Intake (AI) for vitamin E for different age groups

Age	Institute of Medicine, the USA [8]	State sanitary and epidemiological regulation of the Russian Federation [47]	European Food Safety Authority [48]	
0–6 months	4 mg/day	3 mg/day	N/A	
7–11 months	5 mg/day	4 mg/day	5 mg/day	
1–2 years	6 mg/day		6 mg/day	
3 years		7 mg/day	9 mg/day	
4–6 years				
7–8 years				
9–10 years	11 mg/day	10 mg/day	Males – 13 mg/day	
11–13 years				
14 years	15 mg/day	12 mg/day	Females, including pregnant and lactating ♀ – 11 mg/day	
>15 years				

VITAMIN E INTAKE IN VEGANS, VEGETARIANS, AND OMNIVORES

It has been found that vegans consume the vitamin E most [49–54], which are then followed by vegetarians, and finally omnivores consume the least vitamin E [55]. However, there is also a huge disparity in the consumption of vitamin E by vegans. The

maximum intake rate was suggested by Weikert et al. [56] – 25.9 mg/day, which was followed by Rauma et al. (25 mg/day) [51], while Sanders and Roshanai [49] have calculated it to be as low as 11 mg/day. Sobiecki et al. found vegans to consume less vitamin E (12 mg/day) [55]. Larsson et al. [54] concluded intake rate to be 13 mg/day for females and 18 mg/day for males.

Studies of Elorinne et al. [53], Draper et al. [50] and Haddad et al. [52] have also come up with almost the same results, where subjects were found to consume almost 20 mg of vitamin E every day (Table 5).

The intake amounts in vegetarians do not stray too much. The lowest consumption rate in vegetarians

was found by Sobiecki et al. (13.6 mg/day) [55], while the highest rate was found by Draper et al. (16 mg/day) [50].

According to the study of Millet et al., the consumption rate for vegetarians was 15 mg/day for males and 14 mg/day for females [57].

Table 5. Intake of vitamin E in vegans, vegetarians, and omnivores

References	Type of diet	Number of participants	Vitamin E (mg)
Millet et al. [57]	Vegetarians	F*=26	15
		M**=11	14
Sanders and Roshanai [49]	Vegans	F=10	11
		M=10	14
Draper et al. [50]	Demivegetarians***	F=24	17
	Vegetarians	F=36	16
	Vegans	F=20	17
	Demivegetarians	M=13	15
	Vegetarians	M=16	16
	Vegans	M=18	23
Rauma et al. [51]	Vegans	F=20	25
	Omnivores	F=20	11
Haddad et al. [52]	Vegans	M=10	21
		F=15	17
	Omnivores	M=10	18
		F=10	23
Elorinne et al. [53] (α -tocopherol)	Vegans	22	20
	Omnivores	15	17
Larsson et al. [54]	Vegans	M=15	18
		F=15	13
	Omnivores	M=15	9.2
		F=15	7.3
Weikert et al. [56]	Vegans	36	25.9
	Omnivores	36	13.4
Sobiecki et al. [55]	Vegans	803	12.1
	Vegetarians	6673	13.6
	Omnivores	18244	16.3

Note: *F – Females; **M – Males; *** – usually avoid meat.

VITAMIN E SUPPLY IN VEGANS, VEGETARIANS, AND OMNIVORES

In the studies conducted by Helman and Darnton-Hill [58] and Millet et al. [57], the serum concentrations of α -tocopherol were significantly higher in vegetarians than in their non-vegetarian counterparts. On the other hand, in the study of Pronczuk et al. [59], it was found that male vegetarians had 30% and female vegetarians had 22% reduced

concentrations of vitamin E in plasma than the control subjects. In other studies [51, 53, 60, 61], omnivores were found to be slightly richer in vitamin E than vegetarians and vegans. The major determinant factors of serum α -tocopherol concentration in the blood are dietary and supplemental vitamin E [62–64]. Another general trend that can be observed from the results of different authors is that females have higher vitamin E level than males (Table 6).

Table 6. Blood concentration of vitamin E in vegans, vegetarians, and omnivores

Reference	Type of diet	Number of participants	Vitamin E (μmol/L)	α-Tocopherol: Cholesterol (μmol/mmol)	Percentage of subjects below threshold
Millet et al. [57]	Vegetarians and vegans	F*=26	26.5	N/A	N/A
		M**=11	22.8	N/A	N/A
	Omnivores	F=36	18.4	N/A	8
		M=33	17.2	N/A	6
Pronczuk et al. [59]	Vegetarians	F=51	16.8	4.7:1	N/A
		M=28	16.7	5.7:1	N/A
	Omnivores	F=51	20.5	4.21:1	N/A
		M=28	21.5	4.04:1	N/A
Rauma et al. [51]	Vegans	F=20	26	6.2:1	N/A
	Omnivores	F=20	29	5:1	N/A
Krajcovicova-Kudlackova et al. [66]	Vegetarians	F=38	31.4	6.59:1	N/A
		M=29	30.5	6.21:1	N/A
	Omnivores	F=37	32.7	6.13:1	N/A
		M=38	29.7	5.42:1	N/A
Krajcovicova-Kudlackova et al. [61]	Vegetarians	F=39	28.6	6.32:1	N/A
		M=42	31.4	6.97:1	N/A
	Omnivores	F=33	30.2	5.43:1	N/A
		M=29	33.2	5.83:1	N/A
Gorbachev et al. [67]	Vegetarians	46	20.43	N/A	23
Li et al. [65]	Omnivores (>285 gm meat in a day) α-tocopherol	18	12	N/A	50
	Omnivores (<285 gm meat in a day) α-tocopherol	60	12.61	N/A	65
	Vegetarians	43	12.72	N/A	50
	Vegans	18	12.05	N/A	50
Elorinne et al. [53]	Vegans	21	16.67	4.33:1	N/A
	Omnivores	18	21.1	4.66:1	N/A
Schupbach et al. [60]	Omnivores	100	26	N/A	0
	Vegetarians	53	22.7	N/A	0
	Vegans	53	22.1	N/A	3.8
Helman et al. [58]	Vegetarians	93	25.77	N/A	0
	Omnivores	37	20.66	N/A	8
Weikert et al. [56]	Vegans (with supplements)	3	32.3	7.11:1	N/A
	Vegans (without supplements)	33	28.5		N/A
	Omnivores (with supplements)	4	37.6	6.72:1	N/A
	Omnivores (without supplements)	32	33.2		N/A

Note: *F – Females; **M – Males.

Only the outlier to this trend is the result made by Krajcovicova-Kudlackova et al. [61].

Vegans' vitamin E concentrations widely vary from author to author. The lowest level was calculated by Li et al. [65] (12.05 $\mu\text{mol/L}$). On the other hand, Rauma et al. [51], have found it to be up to 26 $\mu\text{mol/L}$. Schupbach et al. [60] and Elorinne et al. [53] have reported it to be 22.1 $\mu\text{mol/L}$ and 16.67 $\mu\text{mol/L}$ respectively.

In the case of vegetarians, Krajcovicova-Kudlackova et al. [61,66] found the highest level of vitamin E (31.4 $\mu\text{mol/L}$), whereas Li et al. [65] found it to be as low as 12.72 $\mu\text{mol/L}$. Pronczuk et al. [59] has also found it to be low (16.7 $\mu\text{mol/L}$ for males and 16.8 $\mu\text{mol/L}$ for females). The results of Helman et al. [58], Schupbach et al. [60] and Gorbachev et al. [67] are pretty close to each other, ranging from 20.43 $\mu\text{mol/L}$ to 25.77 $\mu\text{mol/L}$.

As far α -tocopherol:cholesterol ratio is concerned, there are slight variations in results. Pronczuk et al. [59] have found the least α -Tocopherol:Cholesterol ratio. According to their study, α -Tocopherol:Cholesterol was as low as 4.04:1. Pronczuk et al. and Elorinne et al. [53] have also found relatively small ratio α -Tocopherol:Cholesterol in all the dietary groups (4.33:1 and 4.66:1 for vegans and omnivores, respectively). The results of the other researches were a bit higher. For instance, Weikert et al. [56] have recorded the highest α -Tocopherol:Cholesterol ratio in vegans (7.11:1). Other data have shown the ratio in different dietary groups to be between 5:1 – 6.97:1 [61, 66].

CONCLUSION

Based on the data available to date, we can conclude that vegans are the leaders in vitamin E intake. Omnivores consume the least amount of it. At the same time, most studies show that there is no significant difference between serum vitamin E concentrations between vegans, vegetarians, and omnivores. More importantly, serum level of vitamin E does not provide clear information about its status in the body. So, the vitamin E:cholesterol ratio has been considered a better indicator for assessing actual vitamin E status, because the blood concentration of the vitamin is directly proportional to the lipid present in blood. However, in different researches, the vitamin E:cholesterol ratio among the same dietary group does not significantly stray. Similarly, there is not any significant difference among different dietary groups. In order to get definitive results, more researches have to be conducted.

Foundation

The research work to prepare the manuscript was subsidized and conducted within the framework of the State assignment under the Program of scientific research work of the State Academies of Sciences for 2022-2024 (theme № 0410-2020-0005).

Conflict of interest

Authors declare no conflicts of interest.

REFERENCES

1. *Traber M.* vitamin E. In: Shils M, Shike M, Ross A, Caballero B, Cousins R, editors. Modern nutrition in health and disease. 10th ed. Baltimore: Philadelphia: Lippincott Williams & Wilkins, 2006; 396–411.
2. *Hosomi A., Arita M., Sato Y., et al.* Affinity for alpha-tocopherol transfer protein as a determinant of the biological activities of vitamin E analogs. *FEBS Lett.* 1997; 409(1): 105–108.
3. *Lim Y., Traber M.G.* Alpha-Tocopherol Transfer Protein (α -TTP): Insights from Alpha-Tocopherol Transfer Protein Knockout Mice. *Nutr Res Pract.* 2007; 1(4): 247.
4. *Pham-Huy L.A., He H., Pham-Huy C.* Free radicals, antioxidants in disease and health. *Int. J. Biomed. Sci.* 2008; 4(2): 89–96.
5. *Traber M.* Vitamin E regulatory mechanisms. *Annu. Rev. Nutr.* 2007; 27: 347–362.
6. *Niki E.* Interaction of ascorbate and alpha-tocopherol. *Ann. N.Y. Acad. Sci.* 1987; 498(1): 186–199.
7. *Lewis E.D., Meydani S.N., Wu D.* Regulatory role of vitamin E in the immune system and inflammation. *IUBMB Life.* 2019; 71(4): 487–494.
8. Institute of Medicine. Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids. *Krinsky N.I.*, editor. Washington, D.C.: National Academies Press, 2000; 528.
9. *Ricciarelli R., Tassinato A., Clément S., Ozer N., Boscoboinik D., Azzi A.* alpha-Tocopherol specifically inactivates cellular protein kinase C alpha by changing its phosphorylation state. *Biochem. J.* 1998; 334(Pt 1): 243–249.
10. *Traber M., Atkinson J.* Vitamin E, antioxidant and nothing more. *Free Radic. Biol. Med.* 2007; 43(1): 4–15.
11. *Zingg J.* Vitamin E: A Role in Signal Transduction. *Annu. Rev. Nutr.* 2015; 35(1): 135–173.
12. *Steiner M.* Influence of vitamin E on platelet function in humans. *J. Am. Coll. Nutr.* 1991; 10(5): 466–473.
13. *Chan A.C., Wagner M., Kennedy C., et al.* Vitamin E up-regulates arachidonic acid release and phospholipase A2 in megakaryocytes. *Mol. Cell. Biochem.* 1998, 1891, 1998; 189(1): 153–159.
14. The Effect of Deficiency of Vitamins E and A on the Retina. *Nutr. Rev.* 1980; 38(11): 386–389.
15. *Brion L.P., Bell E.F., Raghuvver T.S.* Vitamin E supplementation for prevention of morbidity and mortality in preterm infants. *Cochrane Database Syst. Rev.* 2003; (4): CD003665.
16. *Kowdley K.V., Mason J.B., Meydani S.N., Cornwall S., Grand R.J.* Vitamin E deficiency and impaired cellular immunity related to intestinal fat malabsorption. *Gastroenterology.* 1992; 102(6): 2139–2142.
17. *Tanyel M.C., Mancano L.D.* Neurologic findings in vitamin E deficiency. *Am Fam Physician.* 1997; 55(1): 197–201.

18. Cavalier L., Ouahchi K., Kayden H.J., et al. Ataxia with Isolated Vitamin E Deficiency: heterogeneity of Mutations and Phenotypic Variability in a Large Number of Families. *Am. J. Hum Genet.* 1998; 62(2): 301–310.
19. Ranjit R., Takalloabdali S., Galchenko A.V. Importance of micronutrients in the oral cavity. *Acta Stomatol Naissi.* 2021; 37(83): 2186–2202.
20. Meydani M. Vitamin E and atherosclerosis: beyond prevention of LDL oxidation. *J. Nutr.* 2001; 131(2).
21. Barron J. Fat soluble anti-oxidant vitamins A, E and carotenoids. *CPD Bull Clin Biochem.* 2001; 3(3): 82–86.
22. Pearce E. Hypothyroidism and dyslipidemia: modern concepts and approaches. *Curr Cardiol Rep.* 2004; 6(6): 451–456.
23. Nemes K., Åberg F., Gylling H., Isoniemi H. Cholesterol metabolism in cholestatic liver disease and liver transplantation: From molecular mechanisms to clinical implications. *World J. Hepatol.* 2016; 8(22): 924.
24. Horwitt M., Harvey C., Dahm C., Searcy M. Relationship between tocopherol and serum lipid levels for determination of nutritional adequacy. *Ann. N.Y. Acad. Sci.* 1972; 203(1): 223–236.
25. Vasankari T., Ahotupa M., Viikari J., et al. Effect of 12-month statin therapy on antioxidant potential of LDL and serum antioxidant vitamin concentrations. *Annals of Medicine.* 2009; 36(8): 618–622.
26. Figueroa V., Milla C., Parks E.J., Schwarzenberg S.J., Moran A. Abnormal lipid concentrations in cystic fibrosis. *Am. J. Clin. Nutr.* 2002; 75(6): 1005–1011.
27. Stroes E. Statins and LDL-cholesterol lowering: an overview. *Curr. Med. Res. Opin.* 2005; 21(Suppl 6): S9–S16.
28. Commission of the European Communities. Report of the scientific committee for food nutrient and energy intakes for the European community. commission of the European communities. Brussels; 1993.
29. Gey K.F. Vitamins E plus C and interacting conutrients required for optimal health. *BioFactors.* 1998; 7(1–2): 113–174.
30. Pastori D., Carnevale R., Cangemi R., et al. Vitamin E Serum Levels and Bleeding Risk in Patients Receiving Oral Anticoagulant Therapy: a Retrospective Cohort Study. *J. Am. Hear Assoc Cardiovasc Cerebrovasc Dis.* 2013; 2(6): e000364.
31. Leppälä J., Virtamo J., Fogelholm R., et al. Controlled trial of alpha-tocopherol and beta-carotene supplements on stroke incidence and mortality in male smokers. *Arterioscler Thromb Vasc. Biol.* 2000; 20(1): 230–235.
32. The Alpha-Tocopherol and Beta Carotene Cancer Prevention Study Group. The Effect of Vitamin E and Beta Carotene on the Incidence of Lung Cancer and Other Cancers in Male Smokers. *N. Engl. J. Med.* 1994; 330(15): 1029–1035.
33. Sesso H.D., Buring J.E., Christen W.G., et al. Vitamins E and C in the Prevention of Cardiovascular Disease in Men. *JAMA.* 2008; 300(18): 2123.
34. United sanitary-epidemiological and hygienic requirements for goods subjected to sanitary-epidemiological supervision (control) of the EurAsEC Customs Union.
35. Norwegian Scientific Committee for Food Safety (VKM). Assessment of vitamin E intake in relation to tolerable upper intake levels. Norway; 2017.
36. Ulatowski L., Thakur V., Manor D., Parker R. Vitamin E protects against PUFA-induced behavioral and motor deficits (596.8). *FASEB J.* 2014; 28(S1): 596.8.
37. Resnicow K., Barone J., Engle A., et al. Diet and serum lipids in vegan vegetarians: a model for risk reduction. *J. Am. Diet. Assoc.* 1991; 91(4): 447–553.
38. National Research Council. Recommended Dietary Allowances. 10th ed. Washington, D.C.: National Academies Press; 1989.
39. Bieri J., Everts R.P. Tocopherols and fatty acids in American diets. The recommended allowance for vitamin E. *J. Am. Diet. Assoc.* 1973; 62(2): 147–151.
40. Witting L.A., Lee L. Dietary levels of vitamin E and polyunsaturated fatty acids and plasma vitamin E. *Am. J. Clin. Nutr.* 1975; 28(6): 571–576.
41. Thurnham D.I., Davies J.A., Crump B.J., Situnayake R.D., Davis M. The use of different lipids to express serum tocopherol: lipid ratios for the measurement of vitamin E status. *Ann. Clin. Biochem.* 1986; 23(pt 5): 514–520.
42. Zino S., Skeaff M., Williams S., Mann J. Randomised controlled trial of effect of fruit and vegetable consumption on plasma concentrations of lipids and antioxidants. *BMJ.* 1997; 314(7097): 1787–1787.
43. U.S. Department of Agriculture, Agricultural Research Service. 2019.
44. Food and Data Central. 2020. Accessed on 01.11.2021: <https://fdc.nal.usda.gov>.
45. Traber M.G., Manor D. Vitamin E. *Adv. Nutr.* 2012; 3(3): 330.
46. Australian Government. Vitamin E. *Natl. Heal. Med. Res. Counc.* 2006.
47. State sanitary and epidemiological regulation of the Russian Federation. Norms of physiological needs for energy and nutrients for various groups of the population of the Russian Federation. Moscow; 2021 july. (Accessed on 01.03.2022). Available from: <https://base.garant.ru/402816140/>
48. European Commission. Scientific Opinion on Dietary Reference Values for vitamin E as α -tocopherol. *EFSA J.* 2015; 13(7): 4149.
49. Sanders T.A., Roshanai F. Platelet phospholipid fatty acid composition and function in vegans compared with age- and sex-matched omnivore controls. *Eur. J. Clin. Nutr.* 1992; 46(11): 823–831.
50. Draper A., Lewis J., Malhotra N., Wheeler L.E. The energy and nutrient intakes of different types of vegetarian: a case for supplements? *Br. J. Nutr.* 1993; 69(1): 3–19.
51. Rauma A.L., Törrönen R., Hänninen O., Verhagen H., Mykkänen H. Antioxidant status in long-term adherents to a strict uncooked vegan diet. *Am. J. Clin. Nutr.* 1995; 62(6): 1221–1227.
52. Haddad E.H., Berk L.S., Kettering J.D., Hubbard R.W., Peters W.R. Dietary intake and biochemical, hematologic, and immune status of vegans compared with nonvegetarians. *Am. J. Clin. Nutr.* 1999; 70(3 Suppl): 586S–593S.
53. Elorinne A.L., Alfihan G., Erlund I., et al. Food and nutrient intake and nutritional status of Finnish vegans and non-vegetarians. *PLoS One.* 2016; 11(2): e0148235.
54. Larsson C.L., Johansson G.K. Dietary intake and nutritional status of young vegans and omnivores in Sweden. *Am. J. Clin. Nutr.* 2002; 76(1): 100–106.
55. Sobiecki J.G., Appleby P.N., Bradbury K.E., Key T.J. High compliance with dietary recommendations in a cohort of meat eaters, fish eaters, vegetarians, and vegans: Results from the European Prospective Investigation into Cancer and Nutrition-Oxford study. *Nutr. Res.* 2016; 36(5): 464–477.
56. Weikert C., Trefflich I., Menzel J., et al. Vitamin and Mineral Status in a Vegan Diet. *Dtsch Arztebl Int.* 2020; 117(35–36): 582.
57. Millet P., Guillard J.C., Fuchs F., Klepping J. Nutrient intake and vitamin status of healthy French vegetarians and nonvegetarians. *Am. J. Clin. Nutr.* 1989; 50(4): 718–727.

58. Helman A.D., Darnton-Hill I. Vitamin and iron status in new vegetarians. *Am. J. Clin. Nutr.* 1987; 45(4): 785–789.
59. Pronczuk A., Kipervarg Y., Hayes K.C. Vegetarians have higher plasma alpha-tocopherol relative to cholesterol than do nonvegetarians. *J. Am. Coll. Nutr.* 1992; 11(1): 50–55.
60. Schüpbach R., Wegmüller R., Berguerand C., Bui M., Herter-Aeberli I. Micronutrient status and intake in omnivores, vegetarians and vegans in Switzerland. *Eur. J. Nutr.* 2017; 56(1): 283–293.
61. Krajcovicova-Kudlackova M., Šimončič R., Béderová A., Klvanová J., Brtková A., Grančičová E. Lipid and antioxidant blood levels in vegetarians. *Nahrung. Food.* 1996; 40(1): 17–20.
62. Willett W.C., Stampfer M.J., Underwood B.A., Taylor J.O., Hennekens C.H. Vitamins A, E, and carotene: effects of supplementation on their plasma levels. *Am. J. Clin. Nutr.* 1983; 38(4): 559–566.
63. Stryker S.W., Kaplan L.A., Stein E.A., Stampfer M.J., Sober A., Willett W.C. The relation of diet, cigarette smoking, and alcohol consumption to plasma beta-carotene and alpha-tocopherol levels. *Am. J. Epidemiol.* 1988; 127(2): 283–296.
64. Jacques P.F., Sulsky S.I., Sadowski J.A., Phillips J.C., Rush D., Willett W.C. Comparison of micronutrient intake measured by a dietary questionnaire and biochemical indicators of micronutrient status. *Am. J. Clin. Nutr.* 1993; 57(2): 182–189.
65. Li D., Sinclair A.J., Mann N.J., Turner A., Ball M.J. Selected micronutrient intake and status in men with differing meat intakes, vegetarians and vegans. *Asia Pac. J. Clin. Nutr.* 2000; 9(1): 18–23.
66. Krajcovicova-Kudlackova M., Simonic R., Babinska K., et al. Selected vitamins and trace elements in blood of vegetarians. *Ann Nutr Metab.* 1995; 39(6): 334–339.
67. Gorbachev D.O., Sazonova O.V., Gilmiyarova F.N., et al. Characteristics of the nutritional status of vegetarians. *Profil Meditsina.* 2018; 21(3): 51–56.

Поступила 5 января 2022 г.

ВИТАМИН Е И ЕГО СТАТУС СРЕДИ ВЕГЕТАРИАНЦЕВ И ВЕГАНОВ

© А.В. Гальченко, Р. Ранджит, 2022

А.В. Гальченко

мл. науч. сотрудник, отделение реабилитационной диетотерапии,
ФГБУН «ФИЦ Питания, биотехнологии и безопасности пищи» (Москва, Россия)
ORCID: 0000-0001-7286-5044
E-mail: gav.jina@gmail.com

Р. Ранджит

ординатор, кафедра онкологии и рентгенодиагностики, Медицинский институт,
Российский университет дружбы народов (Москва, Россия)
ORCID: 0000-0002-4255-4197
E-mail: rajesh.ranjit@mail.ru

Витамин Е – один из жирорастворимых витаминов. В настоящее время изучено восемь его форм – это α-, β-, γ- и δ-токоферолы и α-, β-, γ- и δ-токотриенолы. Наибольшую метаболическую активность имеет α-токоферол, так как он наиболее эффективно связывается с сывороточными переносчиками и быстрее доставляется в печень для того, чтобы быть включенным в состав липопротеинов.

Витамин Е представляет собой один из наиболее значительных элементов антиоксидантной защиты организма. Защищая липопротеины крови от свободнорадикального окисления, он препятствует развитию атеросклероза. Более того, он снижает агрегацию тромбоцитов. Таким образом, витамин Е снижает риск многих сердечно-сосудистых заболеваний. Витамин Е также является иммуномодулятором, оказывая значительное влияние на функцию лимфоцитов.

Как и другие гидрофобные витамины, токоферолы и токотриенолы могут накапливаться в тканях и вызывать токсические эффекты. Наиболее выраженным проявлением гипervитаминоза Е является тромбоцитарная дисфункция и геморрагические явления.

Основной пищевой источник витамина Е – растительные масла. В частности, подсолнечное масло является одним из лидеров по его содержанию. Во многом благодаря тому, что подсолнечное масло является наиболее распространенным на территории Восточной Европы, дефицит витамина Е встречается довольно редко в этом регионе, чего нельзя, однако, сказать о странах Северной Америки или Южной Европы, где основными являются кукурузное и оливковое масла.

Большее потребление растительных масел и масличных культур обеспечивает веганов достаточными количествами витамина Е. Однако сывороточные концентрации α-токоферола у них довольно часто оказываются более низкими. Связано это в первую очередь с более низким у веганов уровнем сывороточных липидов, в которых и заключено основное количество α-токоферола. В этом свете на первый план выходит оценка отношения сывороточного α-токоферола к холестерину. Однако и в этом случае не наблюдается значительного перевеса в показателе этого отношения у веганов и вегетарианцев. Вероятно, это связано с тем, что испытуемые как из «растительных» групп, так и из групп смешанного питания в большинстве исследований имели насыщенные витамином Е плазменные липопротеины. Данный вопрос требует дальнейшего исследования.

Ключевые слова: токоферол, токотриенол, α-токоферол/холестериновый индекс, антиоксидант, атеросклероз, агрегация тромбоцитов, растительного происхождения.

Для цитирования: Гальченко А.В., Ранджит Р. Витамин Е и его статус среди вегетарианцев и веганов. Вопросы биологической, медицинской и фармацевтической химии. 2022;25(6):13–21. <https://doi.org/10.29296/25877313-2022-06-02>