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Effect of Vitamin E on Lipid Composition of Submandibular and Lacrimal Glands in Rats[1] (40665)

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It is generally believed that vitamin E is a natural antioxidant which prevents the lipid peroxidation of cellular and subcellular membranes (1). In vitamin E-deficient animals, there is a decrease in the content of polyunsaturated fatty acids (PUFA) in various tissues and subcellular organelles (2–5). Although changes in cholesterol and total lipid content of plasma and liver in rats have been reported (6–10), to our knowledge, there is no information in the literature on the effects of excessive intake of vitamin E on tissue fatty acid composition.

Submandibular salivary glands (SMSG) and extraorbital lacrimal glands are both exocrine secretory glands and an active lipid metabolism has been reported in SMSG (11–16). Like several other tissues, the SMSG (17, 18) and lacrimal glands (19) also undergo diet-induced modifications in their fatty acid patterns. We have previously shown that in essential fatty acid (EFA) deficiency the fatty acid composition of various lipids in these two glands is altered (18, 19). In the SMSG of EFA-deficient rats, modification of lipid composition was associated with a reduction in flow rate of pilocarpine-stimulated whole saliva (18).

The effects of vitamin E on the lipid composition of SMSG and lacrimal glands have not previously been investigated. Since modifications in the fatty acid patterns of tissue lipids, especially structural lipids, may be associated with functional changes, the present investigation was conducted to study if different levels of vitamin E will alter the fatty acid composition of SMSG and lacrimal glands.

Materials and methods. a. Animal study. Three groups of 15-days pregnant, Sprague–Dawley rats[3] were fed purified diets containing three different levels of vitamin E, 0, 250, or 2500 IU vitamin E (DL-α-tocopherol acetate) per kilogram of diet. The basal diet[4] was similar to M.I.T. No. 200 (20) but without any vitamin E added. It had the following percentage composition: lactalbumin, 20.0; sucrose, 67.0; cellulose, 6.0; salt mixture 3.0; tocopherol-stripped lard, 3.0; vitamin mixture (without vitamin E), 1.0. The fatty acid composition of tocopherol-stripped lard was determined by analyzing duplicate samples using gas chromatographic methods. It contained in percent: $C_{14:0}$, 5.2; $C_{16:0}$, 21.2; $C_{16:1}$, 5.9; $C_{18:0}$, 20.0; $C_{18:1}$, 37.4; $C_{18:2}$, 9.3; and $C_{20:0}$, 1.0.

One day after birth, the pups in each group were randomized to form foster litters, each containing 8 pups. This was done in order to minimize genetic variability between the three groups and to eliminate any possible effects of litter size on lipid composition and metabolism of the tissues. Feeding of the same diets to foster mothers was continued until the pups were weaned at 19 days of age. After weaning, young rats (20/group, 10 male and 10 female) were housed in separate, wire-bottom cages and were fed *ad libitum* the same diets which had previously been fed to their foster mothers. Rats were weighed twice a week and the food intake was occasionally measured.

Rats were killed by decapitation 4 weeks after weaning, blood samples were collected in heparinized centrifuge tubes and plasma was obtained by centrifugation. SMSG and exorbital lacrimal glands were dissected out and weighed. Tissues from 6 rats in each group were used for lipid analyses; the remaining rats were used in another study.

b. Extraction of lipids. All solvents were

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[4] All dietary ingredients except sucrose were purchased from ICN, Cleveland, Ohio.

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reagent grade and were freshly redistilled prior to use. Tissue or plasma samples were extracted for lipids with chloroform–methanol (2:1, by vol), using Folch's method (21). Washed lipid extracts were made to a volume (10 ml) and stored under nitrogen at −20°.

c. Analytical methods. Total lipids were determined in 0.2-ml aliquots of the lipid extract using Amenta's method (22) while phospholipids were determined in another aliquot of the lipid extract, using Bartlett's procedure (23). α-Tocopherol concentrations were determined using Bieri's method (24). For all these analytical procedures, duplicate samples of the total lipid extract were used.

Neutral lipids and phospholipids were separated by silicic acid chromatography (25). Aliquots of total lipid extract, neutral lipids and phospholipids were saponified under nitrogen by heating at 100° for 5 min with 0.5 *N* methanolic-NaOH and the methyl esters of the fatty acid were prepared using 14% BF_3-methanol (26). The methyl esters of fatty acids were extracted with hexane and the fatty acid composition was determined using gas–liquid chromatography as previously described (18).

All data were statistically evaluated using analysis of variance and Newman–Keuls' test (27). Since there was no difference in the lipid and fatty acid composition of the glands between male and female rats, the data from six rats in each group were combined.

Results. Food intake determinations made on two separate occasions during the study showed no significant differences in the three groups. Body weights and gland weights of rats fed different levels of vitamin E are shown in Table I. Final body weights of rats fed 250 IU of vitamin E/kg of diet were slightly higher as compared to the other two groups. This difference in body weight was not, however, statistically significant. The absolute weights of SMSG and lacrimal glands were also slightly higher in the rats fed 250 IU vitamin E/kg of diet. However, when expressed in terms of body weight, there was no difference in either SMSG or lacrimal gland weights in any of the three groups.

The data on total lipids and total phospholipids of the lacrimal gland and SMSG are shown in Table II. As compared to the lacrimal gland, the concentrations of total lipids in SMSG were generally higher. There was no significant difference in total lipids of either type of glands whether 0, 250, or 2500 IU/kg of diet of vitamin E were fed. Total lipid phosphorus content was the highest in SMSG and lacrimal glands of rats fed 0% dietary vitamin E as compared with the other two groups.

Table III shows the fatty acid composition of total lipids of the lacrimal glands and SMSG as affected by different dietary vi-

TABLE I. EFFECT OF DIFFERENT LEVELS OF VITAMIN E ON BODY AND TISSUE WEIGHTS

Group	Dietary vitamin E (IU/kg diet)	I. B. Wt.[a] (g)	Final B. Wt. (g)	SMSG		Lacrimal glands	
				mg	mg/100 g B. Wt.	mg	mg/100 g B. Wt.
I	0	48.4 ± 0.1[b]	166.1 ± 10.8	202.0 ± 6.8	115.5 ± 3.5	95.6 ± 3.8	54.9 ± 2.3
II	250	52.7 ± 0.8	191.1 ± 7.2	219.0 ± 8.5	114.9 ± 2.3	99.1 ± 3.4	52.4 ± 1.6
III	2500	51.8 ± 1.1	180.4 ± 5.6	203.4 ± 7.3	112.2 ± 2.7	94.3 ± 2.3	52.7 ± 1.5

[a] Body weight at the time of weaning.
[b] Values are means ± SEM of 19–20 rats/group.

TABLE II. EFFECT OF FEEDING DIFFERENT LEVELS OF VITAMIN E ON TOTAL LIPIDS AND LIPID PHOSPHORUS IN SMSG AND LACRIMAL GLANDS OF RATS

	Lacrimal gland			SMSG		
	dietary vitamin E (IU/kg diet)			dietary vitamin E (IU/kg diet)		
	0	250	2500	0	250	2500
Total lipids (%)	2.96 ± 1.18[a]	2.22 ± 0.42	2.32 ± 0.66	3.78 ± 0.51	3.39 ± 0.42	3.63 ± 0.29
Total lipid P (μmole/g)	24.8 ± 1.79	21.6* ± 1.54	20.8* ± 2.11	28.2 ± 3.77	20.8* ± 2.82	20.7* ± 1.69

[a] Values are mean ± SD of six rats/group. All values are expressed on the basis of fresh weight of the tissue.
* Significantly different as compared with 0 vitamin E group ($P < 0.01$).

TABLE III. EFFECT OF DIFFERENT LEVELS OF VITAMIN E ON THE FATTY ACID COMPOSITION OF TOTAL LIPIDS IN SMSG AND LACRIMAL GLANDS OF RATS

	Lacrimal gland			SMSG		
	dietary vitamin E (IU/kg diet)			dietary vitamin E (IU/kg diet)		
Fatty acid	0	250	2500	0	250	2500
14:0	1.4 ±0.22*	1.4 ±0.20	1.2 ±0.10	0.8 ±0.20	0.8 ±0.17	0.7 ±0.15
Iso 16:0**	1.3 ±0.17	1.4 ±0.17	1.4 ±0.17	1.7 ±0.34	1.7 ±0.15	1.6 ±0.15
16:0	24.4 ±1.49	25.8 ±0.54	24.7 ±1.59	19.5 ±0.32	19.5 ±0.56	20.0 ±0.47
16:1	14.3 ±3.09	9.6 ±3.31	11.4 ±3.87	4.4 ±0.56	4.0 ±0.64	4.3 ±0.71
18:0	9.1 ±1.71	9.65 ±0.34	10.2 ±0.51	12.9 ±0.59	12.8 ±0.44	12.9 ±0.64
18:1	23.8 ±3.67	21.2 ±2.89	22.6 ±1.15	23.6 ±1.52	22.5 ±0.78	22.9 ±1.20
18:2	6.1 ±0.34	7.4 ±0.76	7.3 ±1.30	6.3^{a} ±0.61	6.7 ±0.27	7.2^{a} ±0.54
20:1	1.5 ±0.34	1.48 ±0.59	1.8 ±0.47	1.3 ±0.17	1.3 ±0.17	1.2 ±0.17
20:2	1.0 ±0.37	1.40 ±0.66	1.1 ±0.27	0.7 ±0.10	0.8 ±0.17	0.8 ±0.27
20:3 ω9	4.42^{a} ±0.98	4.65^{b} ±0.71	$3.35^{a,b}$ ±0.59	6.1^{c} ±0.61	5.3^{a} ±0.81	$4.1^{a,c}$ ±1.15
20:3 ω6	$1.98^{a,b}$ ±0.39	2.65^{a} ±0.47	2.87^{b} ±0.61	$3.9^{c,d}$ ±0.29	4.6^{d} ±0.47	5.0^{c} ±0.32
20:4	9.2^{a} ±0.44	12.2^{a} ±1.69	10.8 ±1.94	15.9 ±0.73	17.0 ±1.67	17.2 ±1.86
20:3 ω9 / 20:3 ω6	2.22^{c} ±0.29	1.79^{b} ±0.34	$1.17^{b,c}$ ±0.39	$1.57^{a,c}$ ±0.22	1.17^{a} ±0.29	$0.82^{a,c}$ ±0.27

* Values are mean (% by weight) ± SD of six rats/group. Values sharing a common superscript letter are significantly different using analysis of variance, a and b indicating $P < 0.05$, c and d, $P < 0.01$.

** Tentative identification.

tamin E levels. The level of $C_{20:3\ \omega6}$ in the total lipids increased with an increase in dietary vitamin E. However, the levels of $C_{20:3\ \omega9}$ changed in the opposite direction, i.e., with an increase in dietary vitamin E, the levels of this fatty acid decreased significantly. As a result of these changes in the fatty acid composition, the $C_{20:3\ \omega9}$ to $C_{20:3\ \omega6}$ ratio decreased in the SMSG as well as lacrimal gland total lipids as the level of vitamin E was increased from 0 to 2500 IU/kg of diet. As compared to the SMSG, the lacrimal gland lipids had two- to threefold higher proportions of $C_{16:1}$.

Pooled samples of neutral lipids and phospholipids from each of the three groups were also analyzed for their fatty acid composition. Similar changes were observed in both SMSG and lacrimal glands as were found in their total fatty acid composition, i.e., the ratios of $C_{20:3\ \omega9}$ to $C_{20:3\ \omega6}$ fatty acids were reduced as the level of vitamin E in the diet was increased (data not shown).

TABLE IV. THE CONCENTRATIONS OF α-TOCOPHEROL IN PLASMA, SMSG, AND LACRIMAL GLANDS OF RATS FED DIFFERENT LEVELS OF VITAMIN E[a]

Group	Dietary vitamin E (IU/kg diet)	Plasma (mg/100 ml)	SMSG[b] (μg/g)	Lacrimal glands[b] (μg/g)
I	0	0.14^{a} ±0.05	1.3	2.7
II	250	1.02^{a} ±0.15	33.1	28.2
III	2500	3.65^{a} ±0.42	72.4	74.3

[a] Values are mean ± SD of six rats/group. Values sharing a common superscript letter are significantly different, $P < 0.01$.

[b] Pooled sample from six rats. Wet weight of the tissue.

The concentrations of α-tocopherol in plasma, SMSG, and lacrimal glands of rats from this study are shown in Table IV. The plasma levels of α-tocopherol were the lowest

in the group fed vitamin E-deficient diet. The concentrations of α-tocopherol in plasma increased from 0.14 to 1.02 to 3.65 mg/100 ml as the diet contained increasing amounts of vitamin E. Similarly, the concentrations of α-tocopherol in the lacrimal gland and SMSG increased from 27- to 56-fold with an increase in dietary vitamin E levels.

Discussion. The lower level of vitamin E (250 IU/kg) used in our study is about eightfold the requirement of vitamin E for rats (28). However, comparable levels of vitamin E, 200–500 IU/kg, have been used by other investigators in their control diets to study the effects of vitamin E on tissue lipid composition (29) and on liver microsomal fatty acid desaturases (30) in rats.

The excessive intake of vitamin E has been reported to result in decreased thymus weight and increased adrenal weight in rats (31), and adrenal degeneration and growth inhibition in chickens (32). Reduced body weights, increased relative heart, and spleen weights have also been described as some of the adverse effects of excess vitamin E in rats (33). The lack of significant changes in body weights and SMSG and lacrimal gland weights in groups fed excess vitamin E in our study may be due to comparatively shorter experimental duration and differences in vitamin E levels. Whereas, we fed the different diets for a total of about 7 weeks (1 week during pregnancy, 19 days of lactation, and 4 weeks postweaning), in some of the studies which have reported changes in body weights and organ weights of animals, much longer experimental periods of up to 16 months and higher levels of vitamin E, 10,000 and 25,000 IU/kg of diet have been used (10).

Dietary level of vitamin E did not have an effect on the total lipid content of the SMSG and lacrimal gland. This differs with the results obtained on liver (7) where an increase in dietary level of vitamin E decreased the total lipids.

Increasing the dietary levels of vitamin E resulted in higher concentrations of vitamin E in plasma, SMSG, and lacrimal glands. Increases were proportional to the dietary levels of vitamin E. Similar findings have been reported in other tissues such as liver (10) and red blood cells (34). It would be interesting to study how changes in the level of vitamin E in SMSG and lacrimal glands influence the excretion of this vitamin in saliva or lacrimal fluid.

Although we did not use criteria such as susceptibility of red blood cells to hemolysis *in vitro* to assess vitamin E deficiency, rats fed 0 IU vitamin E had very low levels of vitamin E in plasma (0.14 mg/100 ml) and in the SMSG and lacrimal glands, thus indicating that these rats were indeed deficient in vitamin E since plasma levels less than 0.5 mg/100 ml are generally associated with vitamin E deficiency and those above 1 mg/100 ml are known to reflect adequate vitamin E nutrition (35).

In vitamin E-deficient animals, the levels of PUFA in various tissues and subcellular organelles is decreased (2–5). This may result in an increased tendency for lipid peroxidation of the membranes, resulting in cellular damage. What effect does an excess of vitamin E have on the profiles of PUFA has not, however, been investigated. Our studies indicate that, at least in the SMSG and lacrimal glands of rats, excess vitamin E can modify PUFA composition. There was an increase in the levels of $C_{20:3\ \omega6}$ and a decrease in $C_{20:3\ \omega9}$ levels as the dietary vitamin E was increased. This resulted in a reduction in the ratio of $C_{20:3\ \omega9}$ to $C_{20:3\ \omega6}$. Since our basal diet was marginally deficient in EFA which provided 0.7% of the total calories rather than a minimum of 1% for the rat (36), it might have influenced the fatty acid composition of the lipids such as relatively high 20:3 ω9 levels which were observed in all the three groups.

One mode of action of vitamin E in terms of its ability to protect membranes from peroxidative damage is that it can form a complex with arachidonic acid residues of membrane phospholipids. It has been proposed (37) that the two methyl groups at C_4' and C_8' of the phytyl side chains of vitamin E can fit in the pockets created by the cis double bonds at C-5 and C-11 of arachidonic acid, $C_{20:4\ \omega6}$. It is possible that other PUFA with similar structural features may also interact with vitamin E. Our data give some indirect evidence that this type of interaction is likely to take place between vitamin E and $C_{20:3\ \omega6}$ since the levels of this fatty acid increased in both SMSG and lacrimal glands as the levels of vitamin E in the diet and consequently in

the glands were increased. Since $C_{20:3\ \omega6}$ and $C_{20:4\ \omega6}$ are structurally similar in terms of having cis double bonds at carbons 8, 11, and 14 and the levels of both of these fatty acids in the glands increased with an increase in the diet-induced tissue levels of vitamin E, it is rather tempting to speculate that the structural requirements for complex formation between the two methyl groups of vitamin E at C_4' and C_8' of the phytyl side chain and the cis double bonds of the unsaturated fatty acid are not at C-5 and C-11 as proposed by Lucy (37) but rather at C-8 and C-14 of the PUFA.

Another mechanism whereby the different levels of dietary vitamin E could have affected the lipid composition of the tissues observed in our present investigation is by influencing the circulating thyroid hormone levels. Although we did not measure the plasma thyroid hormone levels in these rats, it has been shown that excessive intake of vitamin E suppresses thyroid activity in chicken (38) and reduces the serum thyroid hormone levels in man (39). A number of studies have indicated that thyroid hormones markedly affect lipid metabolism in man and in several species of animals. Thyroxine administration to normal rats increases the hepatic concentrations of all fatty acids, especially those in phospholipid and free fatty acid fractions (40). Therefore, it is possible that the lower phospholipid concentrations observed by us in the SMSG and lacrimal glands of rats fed vitamin E-supplemented diets may be due to lower thyroid hormone levels.

The activities of microsomal Δ9 and Δ6 desaturases are modified in different ways by the dietary components. Whereas, the 9-desaturase activity is increased by a high carbohydrate diet, a high protein diet increases the 6-desaturation (41–44). *In vitro* stimulation of microsomal Δ9 desaturation activity has been observed in thyroxine-treated rats (45, 46). The Δ6 desaturase activity seems to be inhibited by thyroxine (46). In case the activities of these two desaturases in the SMSG and the lacrimal glands are also similarly effected by thyroid hormones it is quite possible that this may have resulted in decreased synthesis of 20:3 ω9 and increased synthesis of 20:3 ω6, thus resulting in lower proportions of 20:3 ω9 to 20:3 ω6, with an increase in vitamin E levels. There is also some evidence that the activity of hepatic microsomal Δ9 desaturase is decreased in rats fed for 10 weeks diets deficient in vitamin E or containing supplemental levels of vitamin E (30).

Summary. The effects of different levels of vitamin E on SMSG and lacrimal gland lipids were studied in rats. Feeding of purified diets containing 0, 250, or 2500 IU vitamin E/kg of diet was initiated during gestation and continued throughout lactation and then to the pups until 1 month after weaning. SMSG and lacrimal glands were extracted for lipids and the effects of vitamin E on lipid and fatty acid composition were examined. Total lipid content was not affected by different levels of vitamin E. Total lipid phosphorus was, however, decreased with an increase in dietary vitamin E. Vitamin E content of the SMSG and lacrimal glands was increased by severalfold depending upon the level of dietary vitamin E. The fatty acid composition of total lipids, neutral lipids, and phospholipids showed that the levels of $C_{20:3\ \omega9}$ were reduced and those of $C_{20:3\ \omega6}$ were increased as the dietary levels of vitamin E were increased. This resulted in a decrease in the ratio of $C_{20:3\ \omega9}$ to $C_{20:3\ \omega6}$.

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