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Journal of Nutritional Biochemistry 13 (2002) 435–441

THE JOURNAL OF Nutritional Biochemistry

The effect of dietary menhaden, olive, and coconut oil fed with three levels of vitamin E on plasma and liver lipids and plasma fatty acid composition in rats

Ali I. Mohamed[a,*], Ahmed S. Hussein[b], Sam J. Bhathena[c], Y.S. Hafez[d]

[a]*Virginia State University, Agricultural Research Station, Petersburg, VA 23806, USA*
[b]*United Arab Emirates University, P.O. Box 17555, Al-Ain, United Arab Emirates*
[c]*BHNRC, USDA/ARS, Beltsville, MD 20705, USA*
[d]*University of Maryland Eastern Shore, Princess Ann, MD 21853, USA*

Received 8 August 2001; received in revised form 22 January 2002; accepted 4 April 2002

Abstract

The effect of dietary fats with varying degrees of unsaturation in the presence of different concentrations of vitamin E on tissue lipid levels was studied in rats. Rats were fed either menhaden oil, olive oil or coconut oil at 15% levels with either 0.1, 0.3 or 0.6 mg/g of vitamin E as α-tocopherol for four weeks. Rat serum and liver were analyzed for total cholesterol, HDL-cholesterol, triacylglycerol and phospholipids. In addition, fatty acid composition of serum lipids was also analyzed. Serum total cholesterol and triacylglycerol were significantly lower in rats fed menhaden oil than in those fed olive or coconut oil, while the HDL-cholesterol was significantly higher in serum of rats fed menhaden and olive oil than in those fed coconut oil. Levels of vitamin E in the diet had only a significant effect on serum cholesterol and liver phospholipids. The Pearson correlation coefficient showed a significant positive relationship between serum triacylglycerol and total cholesterol, and a negative correlation between triacylglycerol and HDL-cholesterol, and between total and HDL-cholesterol.

In the liver, total cholesterol was significantly higher in rats fed coconut oil than in rats fed menhaden oil. Total liver phospholipids were lower in rats fed either coconut oil or olive oil compared to those fed menhaden oil, especially with higher levels of vitamin E intake. Higher levels of vitamin E in the diet appear to increase triacylglycerol and phospholipids in livers of rats fed menhaden oil. In the liver a significant negative correlation was observed between phospholipids and cholesterol. The type and degree of unsaturation (polyunsaturated fatty acids in menhaden oil, monounsaturated fatty acids in olive oil and saturated fatty acids in coconut oil) significantly affected plasma and tissue lipids.

Keywords: Menhaden oil; Olive oil; Coconut oil; Vitamin E; Serum lipids; Liver lipids; Plasma fatty acid

1. Introduction

The composition of plasma and tissue lipids in man and animals is a reflection of the type and amount of dietary lipids consumed [1–6]. Diet modifications recommended for the general population for the prevention of cancer, heart disease, and diabetes include reductions in total dietary fat and cholesterol and replacement of saturated fat with polyunsaturated fats of both the n-3 and n-6 types. Implementing these recommendations often result in the incorporation of unsaturated fatty acyl groups in cell membrane lipids and thus modulate several physiologic processes. Polyunsaturated fatty acids (PUFA's) are readily incorporated into the cell membrane lipids of several tissues, spleen leukocytes [7], rat heart, liver, kidney and adipose tissue [7,8], and plasma and aortic plaque in swine [7]. However it is not clear whether different tissues have the same level of incorporation of the polyunsaturated fatty acids. This study therefore compares the fatty acid pattern in serum and liver of rats fed different oil and determines whether the dietary tocopherol's level will affect incorporation of fatty acids into the liver lipids.

Fish oils are relatively rich in n-3 fatty acids. The two most common, eicosapentaenoic acid (20:5n3) and docosahexaenoic acid (22:6n3), are involved in many biological processes. Fish oil supplementation has been shown to have

* Corresponding author. Tel.: +1-804-524-6715; fax: +1-804-524-5950.

0955-2863/02/$ – see front matter
PII: S0955-2863(02)00196-1

a protective effect against atherosclerotic changes by lowering plasma triacylglycerol and lipoprotein (especially very-low-density lipoprotein) concentration in normal as well as in hypertriglyceridemic men [9–10]. In hyperlipidemic pigs, supplementation with cod-liver oil reduced the extent of atherosclerosis [11]. In animals fed high fat diets, n3 fatty acids prevented the development of insulin resistance in liver and skeletal muscle, the tissues involved in glucose metabolism [12]. However, fish oil may have some adverse effects in diabetic subjects [6] in that n3 fatty acids increase hepatic glucose output [13], which results in higher fasting and postprandial blood glucose concentrations [14], elevated apolipoprotein B concentration [15]. Thus, increased insulin sensitivity is offset by increased blood glucose concentrations. The beneficial effects similar to those of n3 PUFA are also observed with Mediterranean diets containing olive oil which is rich in monounsaturated fatty acid 18:1n9. The same does not appear to be true for coconut oil, rich in saturated fatty acids.

Fish oils are highly unsaturated and readily undergo peroxidation [16,17]. Thus, increased peroxidation may account for some of the adverse effects of fish oil on lipid metabolism variables. It is widely believed that administration of polyunsaturated fatty acids decreases the concentrations of vitamin E in the body [18] as a result of its antioxidant function [19]. Vitamin E supplementation is often recommended as a necessary antioxidant when large amounts of unsaturated fats are consumed [8,19]. Vitamin E may also independently affect lipid metabolism. It may promote the beneficial effects of fish oil by reducing the peroxidative damage caused by fish oil while its deficiency causes reproductive failure, nutritional myopathies and abnormalities of liver, blood, brain, pancreas and capillaries in experimental animals [20]. Vitamin E deficiency also reduces the membrane's ability to maintain normal metabolism or defend against environmental stress. A RDA of 15 IU of vitamin E daily has been established as a minimum requirement in man, with supplemental amounts recommended for pregnant and lactating women, newborn infants and older individuals. However, most American diets do not provide adequate amounts of vitamin E. However, the requirement of Vitamin E in rats is higher than Human. The vitamin E requirement for most of the frequently used strains of rats is 18 mg of α-tocopherol/Kg diet (42 μmol/Kg) when lipids compromise less than 10% of the diet [21]. This corresponds to 27 IU/Kg diet and this is equivalent to 57 μmol/Kg [21].

Most studies using fish oil concentrate or n3 fatty acids have not evaluated the addition of vitamin E or other antioxidants. Significant increases in the α- and γ-tocopherol content of red cell membranes have been reported in humans after supplementation with fish oil, even in the absence of additional vitamin E in the diet [5]. We were interested to see whether or not Vitamin E levels would modify the effect of saturated and unsaturated fatty acid on the serum and liver lipids. Hence, we added similar concentrations of vitamin E to all the dietary fats used in the present study. The data on the interaction of vitamin E with monounsaturated fatty acids and saturated fats are even more lacking. The objective of the present study was to examine the effects of interaction between unsaturation of dietary fatty acids and vitamin E at various levels on serum and liver lipids and serum lipid fatty acid composition in rats.

2. Materials and methods

2.1. Experimental design

Ninety weaning male Sprague-Dawley rats were divided into nine groups of ten each. The rats were fed 15g/100g fat diet containing either menhaden oil (rich in long-chain PUFA), olive oil (rich in monounsaturated fatty acids, oleic acid) or coconut oil (rich in saturated fatty acids), with three levels of vitamin E as α-tocopherol at 0.1, 0.3 and 0.6 g/100 g diet. Each ten-replicate group was randomly assigned to each of nine dietary treatments. The treatments consisted of nine diets containing three types of fat and three levels of added vitamin E in a complete factorial arrangement. All diets contained 15g fat per 100g diet, plus 40g defatted soybean meal, 17.5g starch, 16g dextrose, 5g cellulose, 4g AIN-93 mineral mix and 2g AIN-93 vitamin mix. Fatty acid composition of oils fed to rats is given in Table 1. The rats were fed the test diets for four weeks. The entire protocol was approved by the University of Maryland Eastern Shore Animal Care and Use Committee. Rats were housed individually in stainless steel screen bottom cages (18 $\times$ 28 $\times$ 18 cm) in a room maintained on a 12-hr light and dark cycle (light on: 07.00–19.00 hr) with temperature controlled at 18–26°C and relative humidity at 40–50%. Rats were given free access to food and distilled, deionized water. Food intake and body weight were determined weekly.

2.2. Blood and liver collection

At the end of the experimental feeding period, rats were fasted 12 hr, anesthetized with diethyl ether, and the blood collected from the central retinal artery. Serum was obtained by centrifugation at 5°C at 1500 $\times$ g for 30 min, then removed, aliquoted, and stored at -70°C for subsequent analysis. The liver was quickly removed, weighed, placed in a plastic bag, frozen on dry ice and stored at -70°C.

2.3. Chemical analyses

Serum samples were analyzed for total cholesterol [22], HDL cholesterol [23], and triacylglycerol [24]. Liver was analyzed for total lipids [25], total cholesterol [26], triacylglycerol [27] and phospholipids [28]. Serum and liver lipids were extracted and purified as described by John and Bell [29]. Fatty acid methyl esters (FAME) were prepared as

Table 1
Fatty acid composition of oils

Fatty acid %	Oil		
	Menhaden	Olive	Coconut
C6:0	–	–	1.3
C8:0	–	–	12.2
C10:0	–	–	8.0
C12:0	–	–	48.8
C14:0	9.7	–	14.8
C15:0	1.4	–	–
C16:0	20.5	11.3	6.9
C16:1	12.6	0.8	–
C16:2	1.6	–	–
C16:3	2.2	–	–
C16:4	1.7	–	–
C18:0	3.3	2.6	2.0
C18:1	11.1	78.4	4.5
C18:2ω6	1.8	5.7	1.4
C18:3ω3	1.6	0.5	–
C18:4ω3	2.9	–	–
C20:0	0.4	0.4	0.1
C20:1	1.0	0.2	–
C20:4ω6	2.1	–	–
C20:5ω3	13.7	–	–
C22:1	0.8	–	–
C22:5ω3	2.6	–	–
C22:6ω3	9.2	–	–
Total Saturated FA	35.1	14.3	94.1
Total Monounsaturated	23.6	79.4	4.5
Total Polyunsaturated	39.1	6.2	1.4
ω3/ω6	7.8	0.1	–
P:S Ratio	1.1	0.4	0.1

described in details in our previous report [30] and was analyzed by capillary gas chromatography as previously described [30].

2.4. Statistical analysis

Data were analyzed by a two-way ANOVA to measure differences between dietary fats and vitamin E concentration, and by linear regression analysis for the various measurements using SAS programs, version 6.12 [31]. Duncan's multiple range test [32] was used to determine differences in model-classified composition data when significant differences were observed using ANOVA. Differences with P values less than 0.05 were considered to be statistically significant.

3. Results

3.1. Serum lipids

The type of dietary fat had no significant effect on body weight gain of rats, but significantly ($p < 0.009$) affected feed intake. Rats fed diets containing coconut oil had higher average feed intake (21.86 g/day) than rats fed diets containing menhaden or olive oil, 20.76, 20.41 g/day, respectively. However, vitamin E levels had no significant effect on either body weight gain or feed intake (Table 2).

Table 2 summarizes serum total cholesterol, HDL-cholesterol and triacylglycerol concentrations in rats fed different dietary fats and different levels of vitamin E. The average serum total cholesterol level was significantly ($p < 0.001$) lower in rats fed menhaden oil (1.37 mmol/L) compared to those fed either olive (1.73 mmol/L) or coconut oil (1.96 mmol/L). Similar trends were observed with serum triacylglycerol levels. Rats fed diets containing menhaden oil had significantly ($p < 0.001$) lower levels of triacylglycerol (0.40 mmol/L) compared to those rats fed diets with olive oil (0.82 mmol/L) or with coconut oil (1.13 mmol/L). Also, serum HDL-cholesterol levels were significantly ($p < 0.001$) higher in rats fed either menhaden (1.37 mmol/L) or olive oil (1.35 mmol/L) compared to those rats fed coconut oil (0.90 mmol/L).

Vitamin E levels had a significant ($p < 0.002$) effect only

Table 2
Weight gain, feed intake, serum cholesterol, HDL-cholesterol, and triglycerides in rats fed different dietary fats and vitamin E for 4 weeks*

Oil Type	Vitamin E mg/Kg	Weight Gain g	Feed Intake, g/day	Cholesterol, mmol/L	HDL, mmol/L	Triacylglycerol mmol/L, as trilinolein
Menhaden	100	330.12 ± 7.70	20.52 ± 0.71	1.35 ± 0.08	1.40 ± 0.15	0.38 ± 0.03
	300	342.83 ± 4.81	21.59 ± 0.71	1.44 ± 0.12	1.40 ± 0.05	0.37 ± 0.10
	600	332.90 ± 9.68	20.18 ± 0.09	1.33 ± 0.14	1.54 ± 0.07	0.42 ± 0.08
Olive	100	337.83 ± 10.03	20.18 ± 1.8	1.49 ± 0.08	1.37 ± 0.12	0.78 ± 0.10
	300	337.99 ± 7.31	20.47 ± 0.91	1.89 ± 0.07	1.29 ± 0.08	0.77 ± 0.13
	600	319.65 ± 12.8	20.59 ± 0.56	1.76 ± 0.08	1.40 ± 0.06	0.89 ± 0.15
Coconut	100	344.57 ± 8.55	21.90 ± 0.82	1.67 ± 0.13	1.04 ± 0.15	1.14 ± 0.12
	300	340.94 ± 7.71	21.95 ± 0.72	1.96 ± 0.08	0.57 ± 0.01	1.16 ± 0.17
	600	353.24 ± 8.61	21.86 ± 0.74	2.21 ± 0.12	1.05 ± 0.12	1.19 ± 0.11
Significance						
Oil		NS	0.009	0.001	0.001	0.001
Vitamin E		NS	NS	0.002	NS	NS
Oil X Vitamin E		NS	NS	NS	0.096	NS
Vitamin.EEE						

* Each value represnets the mean ± standard error mean of 8 to 10 samples.

Table 3
Liver total lipids, triacylglycerol, cholesterol, phospholipids and C/P ratio in rats fed different fats and vitamin E for 4 weeks*

Oil type	Vitamin E mg/kg	Total Lipids g/100g	Triacylglycerol:mol/g (Trilinolein)	Cholesterol:mol/g	Total Phospholipids: mol/g	C:P ratio**
Menhaden	100	4.19 ± 0.36	33.53 ± 2.37	11.81 ± 0.50	12.70 ± 0.93	0.93
	300	4.82 ± 0.43	43.46 ± 4.07	11.63 ± 0.77	19.26 ± 2.02	0.60
	600	4.94 ± 0.42	47.27 ± 2.94	11.39 ± 0.64	20.89 ± 0.97	0.54
Olive	100	4.46 ± 0.33	39.72 ± 2.93	17.97 ± 1.73	13.06 ± 1.79	1.38
	300	4.46 ± 0.36	36.38 ± 3.73	14.54 ± 0.59	14.37 ± 0.49	1.01
	600	5.15 ± 0.40	40.95 ± 5.24	13.67 ± 0.97	16.68 ± 1.70	0.82
Coconut	100	4.92 ± 0.32	39.12 ± 2.63	16.15 ± 0.83	10.18 ± 1.00	1.59
	300	4.91 ± 0.36	42.58 ± 4.10	18.47 ± 1.64	10.59 ± 0.91	1.74
	600	4.67 ± 0.34	41.26 ± 3.46	17.46 ± 1.28	12.07 ± 0.99	1.44
Significance						
Oil		NS	NS	0.001	0.001	
Vitamin E		NS	NS	NS	0.002	
Oil X Vitamin E		NS	NS	0.006	NS	

* Each value represents the mean ± standard error mean of 8 to 10 samples.
** C:P ratio: Cholesterol:phospholipids ratio. These values were calculated and mean was reported without carrying out the statistical analysis.

on serum total cholesterol, where it was higher in rats fed the upper levels of vitamin E 0.6 and 0.3 mg/g (1.67 and 1.68 mmol/L), respectively, compared to those rats fed the lower level of vitamin E (0.1 mg/g) 1.51 mmol/L.

The Pearson correlation coefficient between serum triglycerides and total cholesterol was 0.655 ($p < 0.001$), between serum triglycerides and HDL-cholesterol was 0.568 ($p < 0.001$), and between serum cholesterol and HDL-cholesterol was 0.731 ($p < 0.001$).

3.2. Liver lipids

Table 3 summarizes the liver lipids profile of total lipids, triacylglycerol, cholesterol, total phospholipids and cholesterol/phospholipids ratio in rats fed different dietary fats and different levels of vitamin E. The type of dietary fat had no significant effect on liver total lipids or triacylglycerol. Liver cholesterol and total phospholipids levels in rats fed different dietary fats were qualitatively similar to those observed in serum. Cholesterol levels were significantly ($p < 0.001$) lower (11.59 mmol/L) in rats fed menhaden oil compared to those fed olive oil (15.42 mmol/L) or coconut oil (17.45 mmol/L), and the level of cholesterol in olive oil fed rats was significantly lower than that found in rats fed coconut oil. A similar trend was observed regarding the effect of the type of oil on liver phospholipids. The level of phospholipids was significantly ($p < 0.001$) higher in rats fed menhaden oil (16.81 mmol/L) compared to those fed olive oil (14.80 mmol/L) or coconut oil (11.01 mmol/L). However, rats fed olive oil had significantly higher levels of phospholipids than those fed coconut oil.

The levels of vitamin E in the diets had no significant effect on liver total lipid, triacylglycerol or total cholesterol. However, total phospholipids was significantly ($p < 0.002$) affected by vitamin E levels in the diets. Liver total phospholipids concentration was significantly higher (16.55 and 15.14 mmol/L) in rats fed the higher levels of vitamin E, 0.6 and 0.3 mg/g, respectively, compared to those rats (11.98 mmol/L) fed the lowest level of vitamin E (0.1 mg/g).

The Pearson correlation coefficient between liver triglycerides and total lipids was 0.941 ($p < 0.001$) and between liver phospholipids and liver cholesterol was -0.238 ($p < 0.08$). The negative correlation between cholesterol and phospholipids levels in rats fed dietary oil with different unsaturation is reflected in the C:P ratio.

3.3. Fatty acid composition of serum lipids

Table 4 summarizes the fatty acid pattern of serum lipids in rats fed different dietary fats and different levels of vitamin E. Medium chain saturated fatty acids, especially myristic acid (14:0) and palmitic acid (16:0), were significantly higher in serum lipids of rats fed coconut oil than those fed either menhaden or olive oil. Eighteen carbon fatty acids, especially stearic acid (18:0), were higher in serum lipids of rats fed olive or coconut oil than those fed menhaden oil. Oleic acid (18:1n9) levels were higher in serum lipids of rats fed olive oil compared to those fed menhaden or coconut oil. It is interesting to note that linoleic acid (18:2n6) was significantly higher in serum lipids of rats fed coconut oil than those fed either olive oil or menhaden oil. Similarly, arachidonic acid (20:4n6) was also higher, though not significantly, in serum lipids of rats fed coconut oil compared to those fed menhaden oil especially with higher doses of Vitamin E. As expected, linolenic acid (18:3n3) was higher in serum lipids of rats fed menhaden oil than those fed olive oil or coconut oil. Long chain fatty acids, both saturated (24:0) and unsaturated fatty acids, especially 22:2, 22:5 and 22:6 were significantly higher in serum lipids of rats fed menhaden oil than those fed olive or coconut oil. Interestingly, 24:0 was higher in serum lipids of rats fed coconut oil than those fed olive oil.

Table 4
Effects of dietary oils on fatty acid composition of serum lipids*

FA Pattern	Oil Type									Significance		
	Menhaden			Olive			Coconut					
	Vitamin E, mg/Kg			Vitamin E, mg/Kg			Vitamin E, mg/Kg					
	100	300	600	100	300	600	100	300	600	Oil	Vit E	Oil X Vit E
14:0	2.8	3.6	3.4	2.4	1.8	1.8	4.3	4.8	4.6	0.001	NS	NS
16:0	18.4	16.1	17.0	17.6	16.8	16.2	18.3	18.5	18.6	NS	NS	NS
16:1ω7	5.7	5.0	4.8	2.1	3.5	1.9	2.3	2.9	3.1	0.015	NS	NS
18:0	6.6	6.9	6.4	7.4	7.3	8.2	9.6	7.1	7.2	NS	NS	NS
18:1ω9	13.3	11.6	9.5	28.2	25.6	24.9	18.2	15.5	15.6	0.001	NS	NS
18:2ω6	6.4	6.9	5.8	7.9	7.7	9.5	12.5	11.9	9.7	0.001	NS	NS
18:3ω3	1.2	0.8	0.9	0.4	0.4	0.3	0.6	0.6	0.7	NS	NS	NS
20:0	7.5	7.3	6.2	9.7	8.7	8.2	6.2	6.9	7.5	NS	0.001	0.001
20:1ω9	1.6	1.6	1.8	0.4	0.6	0.3	0.8	1.6	1.7	NS	NS	NS
20:2ω9	1.0	2.0	2.5	0.9	1.5	1.4	0.8	1.6	1.7	NS	NS	NS
20:3ω6	0.5	1.1	1.3	1.0	1.1	0.7	0.8	1.1	1.3	NS	NS	NS
20:4ω6	9.9	11.1	11.4	13.5	15.0	15.4	12.9	15.5	15.1	0.020	NS	NS
20:5ω3	8.2	9.8	11.5	0.8	0.9	0.5	0.3	0.8	1.0	0.001	NS	0.005
20:0	3.1	4.4	3.8	2.2	2.2	2.7	2.2	3.4	3.5	NS	NS	NS
22:0	3.1	4.4	3.8	2.2	2.2	2.7	2.2	3.4	3.5	NS	NS	NS
22:2ω6	4.0	3.4	4.7	1.8	2.0	2.4	5.4	2.2	2.1	0.001	NS	0.006
22:5ω3	4.6	2.5	2.3	0.3	1.4	2.4	0.4	0.5	0.7	0.003	0.003	0.001
22:6ω3	3.4	3.4	3.8	1.1	1.8	2.0	2.5	2.8	2.8	NS	NS	NS
24:00	3.3	3.6	2.6	2.3	1.8	1.5	1.9	2.3	2.9	0.050	NS	NS
T. Sat. FA	41.7	41.8	39.4	41.5	38.7	38.6	42.5	43.1	44.4	0.065	0.004	0.005
T. Monounsat FA	38.1	41.0	44.3	27.8	31.8	34.4	36.2	37.0	35.3	0.044	0.003	0.005
T polyunsat FA	38.1	41.0	44.3	27.8	31.8	34.4	36.2	37.0	35.3	0.044	0.003	0.005
ω3/ω6	1.9	1.5	1.6	0.2	0.4	0.4	0.2	0.3	0.4	NA	NA	NA
P:S ratio	0.9	1.0	1.1	0.7	0.8	0.9	0.9	0.9	0.8	NA	NA	NA

* Each value represents the mean of 8 to 10 samples.

Levels of vitamin E had no significant effect on short and medium chain fatty acids (C18 and lower) (Table 4). The level of vitamin E had an inconsistent effect on longer chain fatty acids (C20 and higher) of serum lipids. Saturated fatty acids, 20:0 and 24:0, decreased with increasing vitamin E concentrations in rats fed menhaden oil and olive oil but the reverse was observed in rats fed coconut oil. The 22:0 increased with increasing levels of vitamin E in rats regardless of type of dietary fat.

4. Discussion

Several studies have shown the antioxidative and protective effects of vitamin E on lipid peroxidation in humans and animals [8,19,20] when fed diets containing polyunsaturated fatty acids such as fish oil. However, studies on the effect of vitamin E on lipid peroxidation or on tissue lipids in humans and animals fed diets rich in saturated or monounsaturated fatty acids, e.g. coconut oil and olive oil, are lacking. We therefore evaluated the effects of the interaction between level of dietary fat unsaturation and varying levels of vitamin E on serum and tissue lipids and serum lipid fatty acid composition in rats.

Our results showed that the modification of dietary lipid resulted in small but statistically significant changes in serum and liver lipids. Changes in both serum and liver cholesterol were dependent on the saturation of the dietary fats. Thus, the level was highest in rats fed coconut oil, intermediate in rats fed olive oil and lowest in those fed menhaden oil. Several studies have reported cholesterol-lowering effects of unsaturated fats [2,6,9–11]. Few studies have shown that the nature and amount of unsaturation is not important for hypolipidemic effect, i.e., monounsaturated fatty acid, oleic acid, and linoleic acid are equally effective as long-chain polyunsaturated fatty acids like eicosapentaenoic acid and docosahexaenoic acid [6]. In the present study, however, we observed a significantly greater effect of menhaden oil than olive oil on serum and liver cholesterol. Though serum and liver cholesterol and triacylglycerol were significantly altered by dietary fat, there was no significant effect on total lipid levels. As reported in several studies, we also observed a negative relationship between triglycerides and HDL-cholesterol [1,6]. The negative correlation between cholesterol and phospholipid levels in liver in rats fed dietary oils with different unsaturation is reflected in the C:P ratio, as discussed earlier.

In the present study we observed a higher level of linoleic acid and arachidonic acid in serum lipids in rats fed coconut oil than those fed menhaden or olive oil. Linoleic

acid is an essential fatty acid not synthesized in the body, and therefore coming only from the diet. Arachidonic acid is synthesized in the body from linoleic acid. It is therefore surprising to see higher levels of both linoleic acid and arachidonic acid in tissues from rats fed coconut oil. No plausible explanation is available for this observation. It's important to note that fatty acids of the n-6 series compete with those of the n-3 series. It's been reported that in rats, dietary n-3 fatty acids decrease arachidonic acid concentration in the brain but not in the liver [33]. In humans fed salmon rich in EPA and DHA, there was a marked increase of these fatty acids in all blood components with a concomitant decrease in linoleic and arachidonic acids [34].

Long chain highly unsaturated fatty acids are more prone to oxidation and, therefore, increased levels of antioxidants such as vitamin E are recommended with the use of fish oils [8,19,20] which are rich in polyunsaturated fatty acids. It is widely believed that the administration of PUFA decreases the concentration of vitamin E in the body [18] as a result of its antioxidative function [19]. Fish oils are highly unsaturated and readily undergo peroxidation [16,17]. Vitamin E protects lipoproteins and the vascular endothelium from oxidative damage. Diabetic subjects are more prone to the oxidative damage and therefore vitamin E may be more effective in these subjects. Plasma α-tocopherol levels are higher in diabetic subjects than in controls, despite elevated lipid peroxidation. Plasma α -tocopherol is also higher in streptozotocin-treated rats than in control rats. The increase α-tocopherol may be an adaptive mechanism to protect biological systems against oxidative stress [35].

The present study used 100–600 mg/Kg vitamin E. Other researchers [36,37] reported no adverse effects from a 200–600 mg vitamin E/day dosage in humans but, higher doses of vitamin E in streptozotocin-treated rats were shown to decrease plasma triacylglycerol [38]. In the present study a small but significant increase was observed in serum cholesterol concentration with increasing doses of vitamin E in rats fed either coconut or olive oil, but not in those fed menhaden oil. Since vitamin E has antioxidative activity, it may have prevented a rise in cholesterol in menhaden oil-fed rats that were given higher doses of vitamin E. Since less or no oxidation may occur in rats fed olive oil or coconut oil, higher doses of vitamin E may not reduce cholesterol, and may actually increase the levels. Liver phospholipid concentrations were higher at the highest dose of vitamin E compared to lower doses, regardless of type of dietary fat.

Dietary fats as demonstrated in the present study often modify tissue fatty acids. Inclusion of dietary fatty acids is apparently species and tissue specific. Thus, different tissues will have quantitatively different responses. Jones et al. [39] observed that in rats adipose tissue was more responsive than liver, which in turn was more responsive than heart tissue. Feeding rabbits [4] menhaden oil resulted in a significant effect on fatty acyl composition of plasma and tissue phospholipids. However, there were substantial differences in the n-3 fatty acids incorporation into plasma lipoproteins on the one hand, and liver and heart on the other. Bourre et al. [33] observed greater incorporation of EPA and lower levels of arachidonic acid in brain lipids in rats fed fish oil. Croset and Kinsella [40] reported incorporation of docosahexaenoic acid into cardiac organelles. Increases in eicosapentaenoic and docosahexaenoic acids were reported in human blood cells [41] after feeding fish oil compared to olive oil.

Supplementation with fish oil or purified n-3 fatty acids has been reported to affect several metabolic and physiologic processes in humans and animals. Hypolipidemia induced by fish oil is usually associated with triacylglycerol, however the effects on blood cholesterol are still uncertain [9–11]. De Schrijver et al [42] studied the effects of fish oil, beef tallow and peanut oil on tissue lipids in rats. Though both fish oil and peanut oil significantly decreased plasma triacylglycerol, only fish oil decreased plasma cholesterol; peanut oil had no significant effect. Similar results were obtained by Bourre et al. [33] while others [9–11] have observed lower plasma cholesterol and very low-density lipoproteins after feeding fish oil. In the present study rats fed menhaden oil also had significantly lower plasma cholesterol and triacylglycerol and higher HDL-cholesterol than those fed either olive oil or coconut oil. Menhaden oil-fed rats also had significantly higher levels of liver phospholipids compared to those fed other fats, but total lipids and triacylglycerol concentrations in liver were not significantly affected by the type of dietary fat. However, liver C : P ratio was significantly lower in rats fed menhaden oil than those fed coconut oil. In humans, significantly lower plasma triacylglycerol after fish oil supplementation was observed, but plasma cholesterol was not significantly affected [43]. The addition of vitamin E at 200 mg/day had no further effect on plasma triacylglycerol. In another study, feeding a salmon diet to humans lowered plasma triacylglycerol and increased high-density lipoproteins while very low-density lipoproteins only marginally decreased [44].

In conclusion, data from the present and several other studies indicate that the type of dietary fat, and possibly vitamin E supplementation, affect serum and tissue lipids and serum lipid fatty acid composition. However, how vitamin E affects changes in lipid parameters in liver and plasma remains unexplained. The present study suggests some interesting possibilities such as an antioxidative effect of vitamin E as one possible explanation. Clearly more definitive work needs to be done to elucidate the mechanisms and significance of these observations.

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