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Effect of Dietary Selenium and Fish oil on Lipid Peroxidation and Fatty Acid Profile in the Rat

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ABSTRACT

The influence of selenium deficiency and fish oil on lipid peroxidation status and fatty acid composition of tissues(plasma, aorta and liver) was studied. Male Sprague Dawley rats were fed for eight weeks semipurified diets containing 7% corn oil(by weight) or 5.5% fish oil(MaxEPA) plus 1.5% corn oil with or without selenium supplementation. The vitamin E content of all four diets was 117 IU. The indicators of selenium status(glutathione peroxidase activity and selenium levels) were significantly lower in the rats given inadequate selenium in plasma, aorta, and liver. Fish oil feeding increased the level of malondialdehyde(MDA) in the liver and aorta($p<0.002$ and $p<0.001$,respectively) but not that in plasma. Selenium supplementation decreased hepatic MDA levels($p<0.02$). Increases in the levels of 20 : 5(n-3), 22 : 5(n-3), 22 : 6(n-3), 20 : 3(n-6) and a decrease in the level of 20 : 4(n-6) were observed in plasma total lipids and aortic and hepatic phospholipids when fish oil was fed. Though selenium supplementation increased the level of n-3 fatty acids[such as 22 : 6(n-3)] in plasma and the aorta, its overall effect was smaller than the effect of fish oil feeding. These data suggest that selenium may play a significant but minor role in protecting against lipid peroxidation even when vitamin E intakes are in excess of current recommendations in both corn oil and fish oil diets.

KEY WORDS : selenium deficiency · fish oil · polyunsaturated fatty acids · lipid peroxidation.

Introduction

The incidence of cardiovascular disease(CVD), the leading cause of death in Western industrialized nations, has been shown to be modified by the quantity and composition of dietary fat. In particular, increased consumption of n-3 polyunsaturated fatty acids(PUFA), as are found in fish oils, are thought to reduce CVD[1]. However, concerns have been raised because of the tendency for the in vivo peroxidation of these fatty acids[2].

These highly unsaturated fatty acids are efficiently incorporated into cell membrane lipids of various tissues after the consumption of fish oil and are susceptible to free radical attack[3]. These can cause damage to the structure and function of the membrane. Consequently, it is thought to be prudent to increase the intake of dietary antioxidants when

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fish oils are consumed[4], although quantitative estimates have not been provided.

When the primary dietary PUFA is 18 : 2(n-6), a ratio(milligrams of D-α-tocopherol to grams of PUFA) of approximately 0.4 has been suggested for humans[5]. For rats no such suggestion has been made. The AIN-76 diet currently suggests 50IU/kg diet[6]. The content of vitamin E in the dietary oils often provides about 0.1~0.3IU/g of oil. However, in a recent workshop(AIN-76 Diet Workshop ; March 19, 1989 ; New Orleans, LA), it was suggested that the vitamin E content of the rat diet be increased to 100IU/kg diet. The necessity of increasing the intake of other antioxidants when fish oil is consumed is even less well evaluated. Selenium, as an essential component of the enzyme glutathione peroxidase(GSHPx, EC 1.11.1.9) is known to be involved in cellular antioxidant activity by reducing hydroperoxides to the less reactive alcohols[7]. It is thought that vitamin E and selenium may spare each other as antioxidants[8]. Consequently, the level of its intake may also need to be modified when fish or fish oils are consumed.

Selenium deficiency has been shown to change the fatty acid profile as the more highly unsaturated fatty acids are oxidized[9-11]. In accord with this suggestion, Witting and his colleagues[9][10] observed decreases in 20 : 5(n-3), 22 : 6(n-3), 18 : 2(n-6) and an increase in 20 : 4(n-6) in skeletal muscle and liver when rats were fed diets containing highly unsaturated fatty acids but deficient in selenium and other antioxidants for extended periods.

Thus, the level of dietary selenium as well as the type of dietary fat may potentially influence lipid peroxidation and fatty acid profile. The purpose of this study was to determine the change in the fatty acid profile, especially the changes induced in the n-6 and the n-3 fatty acids, of plasma total lipids and hepatic and aortic phospholipids and lipid peroxidation status in response to feeding diets which contained fish oil or corn oil with adequate vitamin E with or without selenium supplementation.

Materials and Methods

Fifty-six, 30-day-old, male Sprague Dawley albino rats(Charles River Laboratories, Wilmington, MA) were housed individually in suspended, stainless steel cages in a room with controlled temperature(20~23℃) and light(8AM to 8PM) for eight weeks. They had free access to distilled-deionized water. They were divided into four groups and fed one of four semipurified diets : +SeCO, a corn oil-based diet with added selenium ; +SeFO, a fish oil-based diet with added selenium ; −SeCO, a corn oil-based diet without added selenium ; −SeFO, a fish oil-based diet without added selenium. All the diets were prepared according to the guidelines recommended by the American Institute of Nutrition[6] and contained by weight(%) : torula yeast, 30 ; oil, 7 ; mineral mix, 3.5 ; vitamin mix, 1.0 ; DL-methionine, 0.3 ; alphacel, 4.0 ; choline bitartrate, 0.2 ; and sucrose to 100. The −SeCO and +SeCO diets contained Mazola corn oil. The −SeFO and +SeFO diets contained 1.5% corn oil, which provided sufficient 18 : 2(n-6) to meet the essential fatty acid requirement, and 5.5% MaxEPA(registered trademark of Seven Seas Limited, Hull, England). The mineral mix, obtained from Teklad, Madison, WI, was the AIN-76 mineral mixture modified to contain no selenium. A selenium supplement was added to the +SeCO and +SeFO diets. Based on analysis(the method is discussed below), the −SeCO and −SeFO diets contained 0.005mg selenium/kg diet ; the +SeCO and +SeFO diets contained 0.4~0.5mg selenium/kg diet. This latter amount represents a nutritionally generous but nontoxic amount[12]. The vitamin mix, also obtained from Teklad, was the AIN-76 vitmin

mixture modified to contain no vitamin E.

The two fat sources, corn oil and MaxEPA, contained different levels of vitamin E : the corn oil contained approximately 0.46 IU/g of oil ; MaxEPA, 2 IU/g of oil. The level of vitamin E in the diets was matched at 117IU/kg diet by adding DL-α-tocopheryl acetate to the corn oil diets. From the relative weight percent of the fatty acids of the oils, the fatty acid composition of the diets was estimated(Table 1). The peroxidizability index(PI) was calculated by the following equation : PI=(% dienoic ×1)+(% trienoic ×2)+(% tetraenoic ×3)+(% pentaenoic ×4)+(% hexaenoic ×5)[13].

The diets were mixed and stored in plastic containers at −20℃ to prevent lipid peroxidation. They were replaced daily and all unconsumed material discarded. Measurement of the lipid peroxide content of the diets[14] showed that there was no difference in the peroxide levels due to type of fat, storage conditions or length of storage.

After eight weeks on their respective diets, the rats were weighed and anesthetized with ketamine hydrochloride(60mg/kg body weight)(Parke-Davis, Division of Warner-Lambert Co., Morris Plains, NJ) and xylazine(10mg/kg body weight)(Mobay Corporation, Shawnee, KS). The activity of GSHPx was assayed by a coupled enzyme procedure using hydrogen peroxide as a substrate and expressed as nmoles NADPH oxidized per min per mg protein [12]. Tissue protein concentration was determined by the method of Lowry et al.[15] The selenium concentration of plasma and liver was determined by a semiautomated fluorometric method[16][17] using an autoanalyzer II(Alphchem Corp., Clackamas, Oregon). MDA in the liver and the plasma was detected spectrometrically[18][19] and that in the aorta fluorometrically[20]. The fatty acid profile of the diets, liver, plasma and aorta were measured as described previously[21].

Data were assessed by a two way analysis of variance in a design of two dietary selenium levels and two types of dietary fat[22]. When necessay to obtain homogeneity of variance, data(selenium content in plasma and liver ; GSHPx activity in pla-

Table 1. Fatty acid composition of the diets*

Fatty Acid	Corn oil diets (−SeCO, +SeCO)	Fish oil diets (−SeFO, +SeFO)
14 : 0	−	5.93
15 : 0	−	0.50
16 : 0	10.16	17.16
17 : 0	−	0.40
18 : 0	1.77	3.07
20 : 0	0.47	0.50
21 : 0	−	0.33
22 : 0	0.13	0.13
ΣSFA	12.53	28.02
16 : 1(n-7)	0.09	6.96
18 : 1(n-9)	27.59	10.55
18 : 1(n-7)	−	4.85
20 : 1(n-9)	0.32	1.24
22 : 1(n-11)	−	0.44
22 : 1(n-9)	−	0.21
24 : 1(n-9)	−	1.04
ΣMUFA	28.00	25.29
18 : 2(n-6)t	−	1.93
18 : 2(n-6)c	58.46	13.51
18 : 3(n-3)	1.02	0.79
18 : 4(n-3)	−	2.79
20 : 2(n-6)	−	0.14
20 : 3(n-6)	−	0.11
20 : 4(n-6)	−	0.84
20 : 5(n-3)	−	14.36
22 : 5(n-6)	−	0.40
22 : 5(n-3)	−	1.73
22 : 6(n-3)	−	10.10
ΣPUFA	59.48	46.70
n-3/n-6	0.02	1.76
PI#	61	145

*Weight percent of identified fatty acid methyl esters. The notation for the fatty acids indicates chain length and number of double bonds ; c=cis isomers ; t=trans isomer.

#Peroxidizability index (see text).

sma, aorta and liver) were transformed into natural logs before statistical analysis. The linear relationship between two variables was determined from the Pearson product moment correlation(r) statistic [22]. Differences were considered significant at $p \leq 0.05$. Statistical calculations were done with STATGRAPHICS(Statistical Graphics Corporation, Rockville, MD) and SAS(SAS Institute, Cary, NC) computer programs.

Results

Although the diets had similar levels of monounsaturated fatty acids(MUFA), the fish oil diet had a greater content of saturated fatty acids(SFA) and PUFA. This latter fact resulted in greatly different ratios of n-3 to n-6 fatty acids and in the PI. The diets both had much higher ratios of vitamin E to PUFA than the 0.4 currently recommended for humans ; in both diets this ratio was about 3.

The weight gain over the 8 week treatment period was satisfactory for each group. There were no significant differences in food intake and the final weight among the dietary groups. The data are given in a previous publication[21].

The effect of the four diets on indicators of selenium status is given in Table 2. As expected, selenium supplementation significantly increased the selenium content of plasma and liver and GSHPx activity in plasma, liver, and the aorta. The effect of the type of dietary fat on indicators of selenium status was not significant. However, in the selenium-supplemented groups, all indicators of selenium status tended to be lower in the rats that had been fed fish oil. This was especially true of GSHPx activity of liver from rats fed fish oil ; the activity was 22% lower in this group.

The effect of the diets on tissue levels of MDA as an indicator of lipid peroxidation is shown in Table 3. Plasma levels were not affected by the dietary treatments. However, significant effects on the level of MDA in the aorta and liver were produced. The concentration of MDA in the aorta of the fish oil group(−SeFO and +SeFO)(68.5± 3.3nmole/g)

Table 2. Effect of level of dietary·selenium and type of dietary fat on tissue selenium concentration and GSHPx activity*

	Plasma Se (ng Se/ml)	Plasma GSHPx+	Aorta GSHPx+	Liver Se (ng Se/g wet wt)	Liver GSHPx+
−SeCO	41.1± 4.3	4.95± 0.47	25.4± 1.2	32.6± 2.9	8.00± 0.86
	(7)	(7)	(7)	(7)	(7)
−SeFO	43.9± 3.3	4.37± 0.67	24.4± 2.2	35.0± 2.8	7.69± 0.97
	(7)	(5)	(6)	(7)	(7)
+SeCO	572± 22	91.2± 9. 4	301± 14	1190± 40	1100± 50
	(7)	(5)	(6)	(6)	(6)
+SeFO	511± 17	84.1± 7.7	291± 19	1100± 30	859± 84
	(6)	(6)	(7)	(7)	(7)
P-values#					
Se	0.001	0.001	0.001	0.001	0.001
Oil	NS	NS	NS	NS	NS
Se×Oil	NS	NS	NS	NS	NS

*Values are means SEM. ( ) : The number of rats per group.
+Activity is expressed as nanomoles NADPH oxidized per min per mg protein.
#NS : Not significant at $p<0.05$.

Table 3. Effect of level of dietary selenium and type of dietary fat on tissue malondialdehyde level*

	Plasma (nmole/ml)	Aorta (nmole/g)	Liver (nmole/g)
−SeCO	1.19± 0.14(5)	41.5± 1.6(7)	449± 27(6)
−SeFO	1.16± 0.16(5)	70.7± 6.2(7)	515± 32(6)
+SeCO	1.13± 0.09(5)	41.5± 3.0(7)	380± 11(6)
+SeFO	1.01± 0.05(5)	66.3± 2.5(7)	424± 11(6)
P-values#			
Se	NS	NS	0.02
Oil	NS	0.001	0.002
Se×Oil	NS	NS	NS

*Values are means SEM.
( ) : The number of rats per group.
#NS : Not significant at $p<0.05$.

was 65% higher than that in the corn oil group (−SeCO and +SeCO)(41.5± 6.6nmole/g)($p<0.001$). In liver its level(469± 21nmole/g) was 13% higher in the fish oil group than in the corn oil group(414± 17nmole/g)($p<0.002$). In addition, in the liver of rats fed selenium the level of lipid peroxidation was 20% lower than in those not given selenium($p<0.02$). There was a negative correlation between the level of MDA and selenium concentration and the level of MDA and GSHPx activity in the liver($r=-0.55$, $p<0.01$; $r=-0.56$, $p<0.02$; respectively).

The effect of the 8 week treatment on the fatty acid composition of hepatic phospholipids is given in Table 4. The consumption of fish oil compared to corn oil produced the expected changes[3)4)]. Most of the fatty acids in the n-6 family[18 : 3(n-6), 20 : 2(n-6), 20 : 4(n-6), 22 : 4(n-6), and 22 : 5(n-6)] were decreased ; only the level of 20 : 3(n-6) increased. In the n-3 family, in addition to the increase in 20 : 5(n-3) and 22 : 6(n-3), the levels of 22 : 5(n-3) and 18 : 3(n-3) were also increased ($p<0.001$). These changes resulted in significant differences in Σ(n-6), Σ(n-3), and their ratio. Because the changes produced in plasma total lipids and aortic phospholipids by the consumption of fish oil were very similar, the data are not shown.

The changes in the levels of the fatty acids from selenium deficiency are shown in Table 5. Only fatty acids in which there was a significant effect are given. Fewer fatty acids were affected by the level of dietary selenium than by the type of dietary fat. In the animals given the low selenium diet, the level of the n-6 fatty acids, 18 : 3(n-6) and 20 : 2(n-6), increased significantly in plasma. For the n-3 family, the level of 22 : 4(n-3) was increased and 22 : 6(n-3) decreased in the low selenium group. In the aorta, only two fatty acids were affected by the level of dietary selenium. The lack of selenium decreased the weight percent of 22 : 5(n-3) and 22 : 6(n-3). These changes caused a small but significant decrease in the ratio of the n-3 to n-6 fatty acids. The fatty acid composition of the hepatic phospholipids from the selenium-supplemented and selenium-deficient groups was very similar. The only fatty acid in which the level changed was 24 : 1(n-9) ; it decreased a small amount with selenium deficiency.

Discussion

There was a profound effect produced on the selenium content of plasma and liver and on the GSHPx activity of plasma, aorta and liver by the level of selenium in the diet, as has been shown repeatedly[12)23)24)]. The lack of an effect of dietary selenium on these variables is in accord with observation by Nalbone et al.[4)].

As n-3 polyunsaturated fatty acids are oxidatively unstable, an increase in membrane unsaturation induced by fish oil intake increases the potential for membrane peroxidation. This increased oxidation can be reflected in the MDA content of tissues. The elevation of the level of MDA in the livers and aorta from rats fed fish oil supports this concept

Table 4. Effect of level of dietary selenium and type of dietary fat on the fatty acid composition of hepatic phospholipids*

Fatty acid	−SeCO (6)	−SeFO (7)	+SeCO (6)	+SeFO (7)	P-values# Se	Oil	Se×Oil
14 : 0	0.19± 0.03	0.20± 0.01	0.21± 0.04	0.24± 0.03	NS	NS	NS
15 : 0	0.21± 0.01	0.24± 0.01	0.19± 0.03	0.25± 0.03	NS	NS	NS
16 : 0	17.50± 0.38	20.53± 0.57	16.73± 0.46	20.43± 0.39	NS	0.001	NS
16 : 1(n-7)	0.56± 0.08	1.19± 0.06	0.57± 0.11	1.09± 0.11	NS	0.001	NS
17 : 0	0.77± 0.04	0.82± 0.04	0.78± 0.08	0.90± 0.09	NS	NS	NS
18 : 0	23.22± 0.71	21.80± 0.51	23.40± 0.58	22.66± 0.51	NS	NS	NS
18 : 1(n-9)	2.90± 0.13	3.78± 0.10	3.01± 0.17	3.63± 0.25	NS	0.001	NS
18 : 1(n-7)	3.27± 0.34	2.77± 0.07	3.19± 0.25	2.86± 0.16	NS	NS	NS
19 : 0	0.12± 0.03	0.12± 0.01	0.13± 0.02	0.14± 0.01	NS	NS	NS
18 : 2(n-6)	12.61± 0.49	13.20± 0.21	12.66± 0.25	12.86± 0.50	NS	NS	NS
18 : 3(n-6)	0.02± 0.02	0.01± 0.01	0.05± 0.03	–	NS	0.05	NS
20 : 0	0.18± 0.02	0.10± 0.02	0.16± 0.02	0.14± 0.01	NS	0.03	NS
18 : 3(n-3)	–	0.06± 0.02	0.01± 0.01	0.06± 0.02	NS	0.001	NS
20 : 1(n-9)	0.24± 0.03	0.13± 0.04	0.17± 0.01	0.11± 0.04	NS	0.03	NS
18 : 4(n-3)	–	0.03± 0.03	–	0.01± 0.01	NS	NS	NS
21 : 0	0.02± 0.01	0.13± 0.02	–	0.16± 0.05	NS	0.001	NS
20 : 2(n-6)	0.49± 0.04	0.15± 0.01	0.41± 0.02	0.17± 0.01	NS	0.001	0.05
20 : 3(n-6)	0.70± 0.11	1.17± 0.06	0.52± 0.11	1.15± 0.07	NS	0.001	NS
22 : 0	2.07± 0.15	1.40± 0.30	1.98± 0.20	1.29± 0.24	NS	0.01	NS
20 : 4(n-6)	29.10± 0.34	12.68± 0.31	29.66± 0.49	12.42± 0.26	NS	0.001	NS
22 : 1(n-9)	–	–	–	0.13± 0.10	NS	NS	NS
20 : 5(n-3)	0.06± 0.01	4.80± 0.32	0.06± 0.02	4.67± 0.34	NS	0.001	NS
24 : 0	0.90± 0.04	0.67± 0.04	0.93± 0.05	0.73± 0.03	NS	0.001	NS
22 : 4(n-6)	0.83± 0.02	–	0.81± 0.03	–	NS	0.001	NS
24 : 1(n-9)	–	0.59± 0.01	–	0.67± 0.04	0.05	0.001	NS
22 : 5(n-6)	0.66± 0.10	0.20± 0.01	0.60± 0.05	0.21± 0.01	NS	0.001	NS
22 : 4(n-3)	0.07± 0.02	0.03± 0.01	0.05± 0.01	0.05± 0.01	NS	NS	NS
22 : 5(n-3)	0.58± 0.03	2.26± 0.08	0.59± 0.05	2.10± 0.14	NS	0.001	NS
22 : 6(n-3)	2.78± 0.16	10.94± 0.21	3.16± 0.18	10.94± 0.47	NS	0.001	NS
ΣPUFA	47.82± 0.43	45.39± 0.37	48.55± 0.44	44.58± 0.31	NS	0.001	NS
Σn-6	44.40± 0.42	27.40± 0.42	44.69± 0.30	26.80± 0.67	NS	0.001	NS
Σn-3	3.48± 0.18	18.13± 0.44	3.87± 0.22	17.78± 0.86	NS	0.001	NS
n-3/n-6	0.08± 0.00	0.66± 0.02	0.09± 0.00	0.67± 0.05	NS	0.001	NS

*Expressed as the weight percent of the fatty acid methyl esters. Values are means± SEM.
( ) : The number of rats per group. #NS is not significant at $p<0.05$.

Table 5. Effect of level of dietary selenium on the fatty acid composition in plasma total lipids, aortic and hepatic phospholipids*

Fatty Acid	Low Se (−SeCO & −SeFO)	High Se (+SeCO & +SeFO)	P-values
Plasma			
18 : 3(n-6)	0.26± 0.08	0.15± 0.05	0.02
20 : 2(n-6)	0.23± 0.05	0.13± 0.04	0.03
22 : 4(n-3)	1.07± 0.19	0.43± 0.07	0.004
22 : 6(n-3)	5.82± 1.54	6.54± 1.63	0.04
Aorta			
22 : 5(n-3)	2.34± 0.44	2.53± 0.46	0.02
22 : 6(n-3)	3.47± 0.59	3.90± 0.71	0.05
(n-3)/(n-6)	0.41± 0.09	0.45± 0.10	0.01
Liver			
24 : 1(n-9)	0.32± 0.09	0.36± 0.10	0.05

*Only fatty acids which were affected by the level of dietary Se are listed. Expressed as the weight percent of the fatty acid methyl esters. Values are means± SEM ; n=10 rats per group for plasma ; n=13 rats per group for aorta and liver.

and agrees with the data of Kobatake et al.[25] and Mouri et al.[26]. It is possible that the elevations in MDA seen in the fish oil-fed rats could be due to the ingestion of peroxide-rich oils. However, analysis of our diets did not show a higher level of MDA in the fish oil diets.

In contrast, although the level of MDA in plasma and serum was shown to increase as a result of fish oil feeding in the studies by Panganamala et al.[27] and Kobatake et al.[25], such an increase was not observed in our experiment. The diets in our study contained more than two times the amount of vitamin E than was present in the experiment of Kobatake et al.(117IU vs 50IU). This higher level of vitamin E in the diet might prevent peroxide levels in plasma from increasing as a result of fish oil feeding. It does not appear to be sufficient, however, to avoid increased lipid peroxide levels in the aorta and liver.

Unlike earlier work, in our study selenium deficiency affected lipid peroxidation only in the liver. Masukawa et al.[28] observed an increase in the level of MDA in aorta from rats fed diets deficient in selenium. Our failure to see such an effect may be due to the presence of sufficient quantities of dietary vitamin E and other antioxidants such as methionine, vitamin C, copper, and zinc. Hafeman and Hoekstra[29] also did not see a difference in lipid peroxidation in selenium-deficient rats and suggested that in the presence of a rather high dose of vitamin E(200IU/kg), there is sufficient protection to prevent peroxidation. It may also reflect the fact that the aorta is less sensitive to lipid peroxidation than the liver. Kornburst and Mavis[30] showed that heart microsomes were peroxidized at a much lower rate than liver microsomes.

Increased oxidation of fatty acids, as indicated by MDA, can also be reflected in a decreased concentration of PUFA. Although plasma and aortic MDA levels indicated that selenium deficiency caused no differences in lipid peroxidation, its level still affected fatty acid composition. These changes were more pronounced in plasma than in aorta. The percentage of 22 : 6(n-3) decreased in plasma and aorta from animals fed the low selenium diet, agreeing with studies by Witting and Horwitt[9]. In aorta, along with 22 : 6(n-3), 22 : 5(n-3) was decreased in the selenium-deficient groups. Though selenium supplementation decreased lipid peroxidation in the liver, its fatty acid composition was minimally affected.

In conclusion, selenium deficiency appears to cause modest changes to peroxidation and in the fatty acid profile at the level of vitamin E used in this study. However, the decreased level of MDA in the liver of selenium-supplemented rats suggests that even at levels of vitamin E thought to be in excess of need, selenium might help to limit lipid peroxidation. The lack of a significant interaction between fish oil and the level of dietary selenium

indicates that at the level of vitamin E used in this study, the sparing effect of selenium is not dependent upon the extent of unsaturation in the dietary oils.

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=국 문 초 록=

식이 셀레늄 수준과 식이 지방산 조성이 쥐의 지질과산화 상태와 조직의 지방산 조성에 미치는 영향

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식이 셀레늄(Se)의 수준과 식이지방산의 조성이 조직(혈장, 동맥, 간)의 지질과산화 상태와 조직의 지방산 조성에 미치는 영향에 대해 연구하였다. Sprague Dawley종 수컷쥐들을 식이 Se 수준(0.005 mg/kg diet, 0.5mg/kg diet)과 식이 지방산의 주요 급원(옥수수유, 어유)에 따라 4군으로 나누어 8주간 실험하였다. 식이에 함유된 vitamin E의 함량은 117IU정도이다.

Se 영양상태의 지표인 glutathione peroxidase 활성도와 Se 수준은 Se이 결핍된 식이를 공급시키면 식이지방 종류에는 상관없이 낮게 나왔다. Se이 충분히 공급된 쥐에게서는 어유군이 옥수수유군보다 위 지표가 낮게 나왔다. 어유공급은 간과 동맥의 MDA를 증가시키나 Se을 공급시킨 쥐의 간 MDA수준은 감소되었다. 어유를 공급시키면 혈장 총지질, 동맥 과 간의 인지질에서 20 : 5(n-3), 22 : 5(n-3), 22 : 6(n-3), 20 : 3(n-6) 수준은 증가하였으며 20 : 4(n-6)수준은 감소되었다. 비록 Se공급이 n-3 지방산 수준 [22 : 6(n-3)]을 증가시키나 어유공급 효과에 비하면 그 효과가 적었다. 본 실험결과에 의하면 vitamin E 가 현재 권장되는 양보다 충분히 많게 공급되어도 Se이 지질과산화를 방지하는데 중요한 역할을 하고 있으므로 적절한 Se공급이 바람직하다.