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Effects of the P/S Ratio of Dietary Lipids and Antioxidant Vitamin Supplements on the Level of Serum Lipids and Liver Lipid Peroxidation in Rats Treated with DMBA*

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ABSTRACT

This study an analyzes the effects of the P/S ratio of dietary lipids and antioxidant vitamin supplements on serum lipids level and fatty acid profile, the degree of lipid peroxidation, and the antioxidant enzyme activities in the liver of rats treated with 7,12-dimethylbenz(α) anthracene(DMBA). P/S ratio of dietary lipids was made into 0.5, 1 and 2 by mixing palm oil, soybean oil, sesame oil and perilla oil at 10%(w/w) fat level and n-6/n-3 ratio was fixed to 4. Antioxidant vitamin of α-tocopherol or β-carotene was supplemented in addition to vitamin mixture which was given at 1 % of the standard diet. Female Sprague-Dawley strain rats, about 60 days old, were divided into three groups(LP ; low P/S ratio(0.5), MP ; medium P/S ratio (1.0), HP ; high P/S ratio(2.0)) and each group was sub-divided into three groups(S ; standard, T ; tocopherol supplemented, C ; carotene supplemented). Two weeks after feeding experimental diets, all groups were treated with a single dose of DMBA(2mg/100g BW) by gastric intubation and fed experimental diet for 9 week. The results were as follows ;

1) Serum total cholesterol(TC) level was not significantly influenced by diet but tended to be lower in HP groups compared to LP and MP groups. Triglyceride level was the highest in LP groups and the lowest in α-tocopherol supplemented groups.

2) Thiobarbituric acid reactive substance(TBARS) level, representing lipid peroxidation in hepatic microsome, tended to be increased as the unsaturation of dietary lipids increases. α-Tocopherol supplement significantly decreased TBARS level.

3) The activities of superoxide dismutase(SOD) and glutathione peroxidase(GSHPx) in hepatic cytosol showed the tendency to be high with increasing P/S ratio of dietary lipids. SOD activity was not significantly influenced by antioxidant vitamin, but GSHPx activity was significantly increased in α-tocopherol supplemented groups.

In summary, high polyunsaturated fat diet was effective on reducing the serum level of total cholesterol and triglyceride, while it increased unsaturation and peroxidizability of serum fatty acid. With increasing P/S ratio of dietary lipids, lipid peroxidation was increased in the liver and antioxidant enzyme system was induced to inhibit lipid peroxidation against oxidative damage. α-Tocopherol supplement was effective in lowering lipid peoxidation, but β-carotene supplement did not exhibit antioxidant effect. (*Korean J Nutrition* 31(5) : 906~913, 1998)

KEY WORDS : P/S ratio · antioxidant vitamins · α-tocopherol · β-carotene · lipid peroxidation.

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Introduction

In recent decades, the Korean diet has changed significantly in response to Western influence. As a result, the percentage of daily caloric intake from lipids has increased from 6.3 - 11.9% in 1970 to 19. 1% in 1995. Approximutely 44% of Korea house holds now consume a diet that consists more than 20% of fat. One of the more obvious repercussions of those dietary modification has been a change in disease pattern in Koreans. Incidence of cardiovascular disease and cancer had increased very rapidly since 1960 - 70[2].

Although the etiology of cancer is uncertain, it is believed that 75 - 80% of cancer-related disease is induced by environmental factors, and 30 - 40% of this is related with diet[3]. American Cancer Association has also suggested that one third of cancer is caused by dietary factors[4]. Many animal experiments has shown the strong relationship between dietary fat intake and mammary cancer. High-fat intake is believed to lead to the development of mammary tumor[5], and dietary lipid composition as well as lipid level influences the incidence of mammary carcinoma [6]. Linoleic acid has shown to have a promoting effect on mammary carcinogenesis[7], while n-3 fatty acid has shown to have a inhibiting effect[8].

It is also been suggested that the high intake of polyunsaturated fatty acid(PUFA) increases the peroxidation of lipid by free radical at the cell membrane, consequently cell damage leads to the promotion of cancer[9][10]. Lipid peroxidation by free radical can be inhibited by antioxidant vitamin and antioxidant enzyme system. Therefore, it is necessary to balance the level of dietary PUFA and antioxidant vitamin intakes.

In this study, the effect of P/S ratio of dietary lipids and antioxidant vitamin supplement on lipid metabolism were examined in DMBA treated rats.

Materials and Methods

1. Animals and experimental diets

Ninety weanling female Sprague-Dawley rats housed in stainless-steel wire-bottomed cages, were maintained at temperature of 22±2℃ with a 12h light and dark cycle. All rats were fed a commercial diet (Samyang Co.) for 4 weeks. The animals were then randomly assigned to nine groups(LP-S, LP-T, LP-C, MP-S, MP-T, MP-C, HP-S, HP-T, HP-C) and each were fed a specific experimental diet for 11 weeks

Table 1. Composition of experimental diets (g/kg diet)

Ingredients	LP-S[1,2]	LP-T	LP-C	MP-S	MP-T	MP-C	HP-S	HP-T	HP-C
Casein	150	150	150	150	150	150	150	150	150
Corn starch	200	200	200	200	200	200	200	200	200
Sucrose	450	450	450	450	450	450	450	450	450
Palm oil	77	77	77	50	50	50	25	25	25
Soybean oil	15	15	15	30	30	30	40	40	40
Sesame oil	3	3	3	13	13	13	25	25	25
Perilla oil	5	5	5	7	7	7	10	10	10
DL-methionine	3	3	3	3	3	3	3	3	3
Choline chloride	2	2	2	2	2	2	2	2	2
α-Cellulose	50	50	50	50	50	50	50	50	50
Vitamin mix.	10	10	10	10	10	10	10	10	10
Mineral mix.	35	35	35	35	35	35	35	35	35
dl-α-Tocopheryl acetate[3]	-	2	-	-	2	-	-	2	-
β-Carotene[4]	-	-	0.14	-	-	0.14	-	-	0.14

1) LP-S : Low P/S ratio(0.5) and Standard, LP-T : Low P/S ratio(0.5) and Tocopherol supplemented, LP-C : Low P/S ratio(0.5) and Carotene supplemented, MP-S : Medium P/S ratio(1.0) and Standard, MP-T : Medium P/S ratio(1.0) and Tocopherol supplemented, MP-C : Medium P/S ratio(1.0) and Carotene supplemented, HP-S : High P/S ratio(2.0) and Standard, HP-T : High P/S ratio(2.0) and Tocopherol supplemented, HP-C : High P/S(2.0) ratio and Carotene supplemented

2) Standard, based on AIN-76 diet 3) 2,000IU dl-α-tocopheryl acetate/kg diet 4) 240,000IU β-carotene/kg diet

(Table 1). Casein, DL-methionine, choline chloride, α-cellulose, dl-α-tocopheryl acetate, and β-carotene were purchased from Sigma Chemical Co. Corn starch(Poongjeon), sucrose(Samyang Co.), vitamin and mineral mixture(ICN Biomedicals Inc.) were used to prepare diet. Diets, and tap water were provided ad libitum.

The P/S ratios of dietary lipids were controlled to 0.5, 1 and 2(LP, MP, HP) by mixing palm oil(Lotte-Samgang Co., soybean oil(Haepyo), sesame oil(Ottugi) and perilla oil(Gohyangnonsan) at 10%(w/w) of the diet. The n-6/n-3 ratio was fixed to 4. Fatty acid composition of each fat source was analysed by gas chromatography(Shimadzu GC 17-A).

Antioxidant vitamin of α-tocopherol(T) or β-carotene(C) was supplemented to the standard diet(S). Since S diet already contained a vitamin mixture which included 4000 IU Vit.A/kg and 50 IU VE/kg, the T group diet was set so that it contained 40 times the amount of Vit.E of the S diet and 2000 IU dl-α-tocopheryl acetate/kg diet. The C group diet was set so that it contained 20 times the Vit.A activity of S diet and 240,000 IU β-carotene/kg diet.

2. Mammary cancer induction

After 2 weeks of feeding the assigned diets, rats were fasted for 24 hours. DMBA(Sigma Cemical Co.) dissolved in soybean oil was administered intragastrically in amount of 2.0mg/100 g b.w. Subsequently, each experimental diet was continued to be provided for an additional 9 weeks. DMBA, used in this experiment as a carcinogen is known to induce in animals mammary carcinoma similar to human breast cancer[11].

3. Blood and tissue sampling

After 11 weeks of feeding these experimental diets, the rats were sacrificed. Blood samples were obtained through heart puncture and serum were seperated at 1,000×g for 15min. The livers were removed, washed with saline and weighed. Serum and liver were stored at −75℃ until analysis.

4. Analysis

(1) Serum lipid concentration

Serum total cholesterol and triglyceride concentration were analyzed by enzymatic procedures using kits(Youngdong Co.).

(2) Serum fatty acid composition

To analyze the fatty acid composition with gas chromatography, a serum was prepared by modifying methods of Fletcher et al.[12] and Lepage & Roy[13]. The Instruments and operating conditions of GC were as follows : Schimadzu GC-17A ; Flamable Ionization Detector ; 30×0.53mm×1μ DB wax capillary column; Carrier gas He; Injection temperature 250℃; Detection temperature 260℃; Column temperature 211℃, isothermal run.

(3) TBARS(thiobarbituric acid reactive substance) concentration in liver

To analyse the amount of peroxidation, liver TBARS concentration was measured by the modified method of Buckingham[14].

(4) Enzyme activities in liver

To measure the antioxidation related enzyme activity, liver SOD(superoxide dismutase) and GSHPx (glutathione peroxidase) activities were analysed by

Table 2. Fatty acid composition of experimental diets[1]

Fatty acids	Dietary groups		
	LP	MP	HP
C12 : 0	0.62	0.40	0.21
C14 : 0	0.94	0.63	0.34
C16 : 0	36.30	26.95	18.22
C16 : 1	0.14	0.11	0.08
C18 : 0	4.09	4.00	3.94
C18 : 1 n9	35.56	32.74	30.58
C18 : 2 n6	17.24	27.70	36.58
C18 : 3 n3	4.32	6.64	9.21
C20 : 0	0.26	0.28	0.30
unknown	0.53	0.55	0.55
Total SFA	42.21	32.27	23.00
Total MUFA	35.70	32.84	30.66
Total PUFA	21.56	34.34	45.79
P/S ratio	0.51	1.06	1.99
n-6/n-3 ratio	3.99	4.17	3.97
PI[2]	26.77	41.80	55.77

1) Expressed as % distribution of fatty acid methyl esters
2) Peroxidizability index(PI)=monoenoic acid×0.025+dienoic acid×1+trienoic acid×2+tetraenoic acid×4+pentaenoic acid×6+hexaenoic acid×8

modified method of McCord & Fridovich[15] and Flohe & Gunzler[16].

(5) Statistical analysis

The results were presented as a mean±SEM and analyzed by analysis of one-way ANOVA. Interaction of dietary P/S ratio and antioxidant vitamin supplement were analysed by two-way ANOVA. Statistical evaluations were determined by Tukey's multiple range test at $\alpha=0.05$. Pearson's correlation coefficients were used to determine the relationship between parameters.

Results and Discussion

1. Food intake and weight gain

Food intake, weight gain and FER(food efficiency ratio) are shown in Table 3. There are no significant differences in food intake among groups. Weight gain was significantly lower in MP groups(P/S=1) than LP groups(P/S=0.5). FER of LP groups(P/S =0.5) was the highest.

Table 3. Food intake, weight gain, food efficiency ratio (FER)[1]

Group	Food intake (g/d)	Weight gain (g/d)	FER
LP-S	18.18±1.26	2.40±0.17	0.132±0.034[b2]
LP-T	18.59±1.14	2.48±0.21	0.132±0.055[b]
LP-C	18.80±1.13	2.41±0.18	0.128±0.047[ab]
MP-S	19.02±1.57	2.20±0.22	0.116±0.043[ab]
MP-T	18.56±0.79	2.04±0.17	0.110±0.074[a]
MP-C	16.24±0.88	1.93±0.14	0.119±0.057[ab]
HP-S	17.34±0.66	2.12±0.12	0.122±0.038[ab]
HP-T	16.62±0.76	2.06±0.01	0.124±0.011[ab]
HP-C	18.29±1.23	2.15±0.17	0.118±0.043[ab]
P/S ratio			
LP	18.52±0.66	2.43±0.11[B3]	0.131±0.003[B]
MP	18.00±0.68	2.06±0.10[A]	0.115±0.003[A]
HP	17.41±0.53	2.11±0.	0.121±0.002[A]
Vit supplement			
Standard	18.18±0.69	2.24±0.10	0.123±0.002
α-to-	17.92±0.54	2.20±0.10	0.122±0.003
β-carotene	17.83±0.65	2.17±0.10	0.121±0.003
P/S*Vit	NS	NS	NS

1) Mean±SE
2) Values with different superscripts are significantly different at $\alpha=0.05$ level by Tukey's multiple range test
3) Statistical significance was calculated at the $\alpha=0.05$ level by 2-way ANOVA
P/S : Main effect of P/S ratio of dietary lipid
Vit : Main effect of vitamin supplement
P/S*Vit : Interaction between P/S ratio of dietary lipid and vitamin supplement

2. Serum lipid concentration

Serum total cholesterol(TC) and triglyceride(TG) concentration are shown in Table 4. TC concentration tended to be lowered in high P/S ratio groups (HP groups). TG concentration was the highest in LP groups(P/S=0.5). α-Tocopherol supplemented groups showed the lowest TG level compared to the standard vitamin groups and β-carotene supplemented groups.

The hypocholesterolemic effect of polyunsaturated fatty acids has been reported in human and animal experiments[17][18]. Inconsistant results have reported monounsaturated fatty acid[19], meanwhile it is well known that saturated fatty acids increases serum cholesterol level[20].

Polyunsaturated fatty acids are known to decrease plasma cholesterol by disturbing absorption in the small intestine, decreasing reabsorption of bile acids, increasing secretion of cholesterol into the intestinal tract, decreasing synthesis of cholesterol in the liver, increasing conversion of cholesterol into bile acids

Table 4. Total cholesterol and triglyceride concentration in serum[1]

Group	Total cholesterol (mg/dl)	Triglyceride (mg/dl)
LP-S	144.64±16.20	178.30±21.40[c2]
LP-T	108.28±12.09	117.79±26.03[ab]
LP-C	150.67±20.24	141.67±12.58[bc]
MP-S	135.84±19.96	101.96±16.18[ab]
MP-T	144.40±17.77	73.83±23.42[ab]
MP-C	119.41±13.12	124.08±18.04[ab]
HP-S	110.42± 9.25	133.07±15.69[bc]
HP-T	109.44±11.92	50.86± 8.23[a]
HP-C	118.46±13.10	123.94±11.52[ab]
P/S ratio		
LP	134.53± 9.84	145.92±12.58[B3]
MP	133.22± 9.77	101.82±11.50[A]
HP	112.77± 6.47	101.10± 9.98[A]
Vit supplement		
Standard	130.30± 9.19	137.42±11.51[B]
α-tocopherol	120.71± 8.49	78.52±12.08[A]
β-carotene	129.51± 9.26	129.45± 8.53[B]
P/S*Vit	NS	NS

1-3) See the explanation of Table 3

Table 5. Fatty acid composition in serum[1] (Relative weight %)

Group	SFA	MUFA	PUFA	PUFA/SFA	PI[4]
LP-S	39.46±0.55	26.00±1.38[c2]	35.37±1.01[a]	0.90±0.03[a]	95.69±3.04[a]
LP-T	38.95±1.32	25.03±2.21[c]	36.86±1.53[ab]	0.96±0.05[abc]	99.61±6.24[a]
LP-C	39.59±0.28	24.29±1.44[bc]	37.04±1.32[ab]	0.93±0.03[ab]	102.63±3.86[ab]
MP-S	39.80±0.82	18.56±1.35[ab]	41.65±1.09[abc]	1.05±0.04[abcd]	115.06±3.51[ab]
MP-T	38.45±0.71	16.97±1.34[a]	45.30±1.71[c]	1.16±0.06[cd]	121.03±5.28[b]
MP-C	39.22±0.69	19.73±1.16[abc]	41.05±1.31[abc]	1.05±0.04[abcd]	107.60±4.01[ab]
HP-S	38.54±1.23	17.53±0.93[a]	44.72±1.39[c]	1.15±0.06[cd]	111.09±5.51[ab]
HP-T	39.06±1.19	17.10±0.94[a]	44.67±1.33[c]	1.15±0.07[bcd]	111.98±4.48[ab]
HP-C	37.74±0.53	16.91±1.24[a]	45.35±1.47[c]	1.21±0.05[d]	110.69±3.74[ab]
P/S ratio					
LP	39.31±0.50	25.10±0.99[B3]	36.44±0.75[A]	0.93±0.02[A]	99.32±2.69[A]
MP	39.18±0.43	18.42±0.75[A]	42.67±0.85[B]	1.09±0.03[B]	114.57±2.62[B]
HP	38.39±0.56	17.18±0.59[A]	44.92±0.78[B]	1.17±0.03[B]	111.23±2.58[B]
Vit supplement					
Standard	39.31±0.51	20.62±1.00	40.62±0.98	1.03±0.03	107.56±2.79
α-tocopherol	38.80±0.61	19.89±1.18	42.11±1.15	1.09±0.04	110.79±3.53
β-carotene	38.84±0.35	20.29±0.91	41.15±0.99	1.07±0.03	107.01±2.25
P/S*Vit	NS	NS	NS	NS	NS

1-3) See the explanation of Table 3

4) Peroxidizability index(PI)=monoenoic acid×0.025+dienoic acid×1+trienoic acid×2+tetraenoic acid×4+pentaenoic acid×6+hexaenoic acid×8

and redistribuing of cholesterol from plasma into liver and other tissues. Kim[21] reported that serum, TG, TC, LDL-C level were decreased when P/S ratio was increased by mixing various dietary lipids. It has been also reported that vitamin E as well as dietary lipids influenced the serum lipids levels. In a short-term experiment, vitamin E lowered serum lipid and cholesterol concentration[22]. Nam and Park[23] studied the effect of polyunsaturated fat with vitamin E supplement on serum lipids and found that highly unsaturated fat as well as vitamin E supplement lowered serum TC and TG level.

3. Serum fatty acid composition

The serum fatty acid composition is shown in Table 5. The serum fatty acid composition related very well with the dietary lipids composition, but the vitamin supplement did not affect the serum fatty acid composition. Since SFA can be synthesized in the body, serum SFA level was similar in all groups and not influenced by dietary lipid. Serum MUFA level was high in LP groups those dietary P/S ratio was 0.5, while serum PUFA level was high in HP groups those dietary P/S ratio was 2. Serum P/S ratio reflected well of dietary P/S ratio.

Peroxidizability index(PI) was significantly lower in LP groups than others. PI represents oxidizability according to the degree of fatty acid unsaturation[24]. Nam and Park[25] also reported increased PI value in highly unsaturated high fat diet and suggested the possibility of impairment in membrane integrity.

In this experiment, serum P/S ratio and PI were increased by increasing the P/S ratio of dietary lipid, however antioxidant vitamin supplement did not influence the serum P/S ratio or PI.

4. TBARS concentration in liver

TBARS concentration was used as an indicator of the amount of malondialdehyde(MDA) which is the product of lipid peroxidation. Table 6 shows that TBARS contents tended to be lower in tocopherol supplemented groups than in standard and β-carotene supplemented groups.

Many studies about lipid peroxidation were reported in terms of quantity and quality of dietary lipid. At a low fat diet, MDA levels were not significantly different among various lipid sources[21][25]. But at a high fat diet, MDA levels increased with per-

illa oil and fish oil compared to beef tallow and corn oil[25)26)]. Meanwhile Nam and Park[25)] explained the reason of the non-significant difference of MDA levels among dietary lipids was that the amount of tocopherol in the diet was sufficient enough to inhibit oxidative damage due to high unsaturation.

Table 6. Lipid peroxide(TBARS) level and superoxide *dismutase* and *glutathione* peroxidase activity in liver[1)]

Group	Lipid peroxide (TBARS) (nmole/g liver)	Superoxide dismutase[4)] (Unit/mg protein)	Glutathione peroxidase[5)] (Unit/mg protein)
LP-S	2.77 ± 0.10^{ab2}	85.16 ± 7.82	2.09 ± 0.17
LP-T	2.44 ± 0.09^{a}	82.73 ± 7.51	2.29 ± 0.08
LP-C	2.61 ± 0.12^{ab}	83.94 ± 5.62	2.19 ± 0.07
MP-S	2.72 ± 0.09^{ab}	82.95 ± 4.76	2.23 ± 0.08
MP-T	2.52 ± 0.07^{ab}	81.34 ± 5.70	2.35 ± 0.13
MP-C	2.82 ± 0.11^{ab}	99.08 ± 9.51	2.22 ± 0.09
HP-S	2.82 ± 0.09^{ab}	87.96 ± 8.80	2.12 ± 0.06
HP-T	2.57 ± 0.08^{ab}	92.22 ± 8.00	2.54 ± 0.13
HP-C	2.90 ± 0.10^{b}	92.51 ± 7.33	2.39 ± 0.11
P/S ratio			
LP	2.61 ± 0.06	83.94 ± 3.81	2.19 ± 0.06
MP	2.68 ± 0.06	87.79 ± 4.15	2.26 ± 0.06
HP	2.76 ± 0.06	90.89 ± 4.54	2.35 ± 0.07
Vit supplement			
Standard	$2.77\pm0.05^{B3)}$	85.27 ± 3.99	2.15 ± 0.06^{A}
α-Tocopherol	2.51 ± 0.05^{A}	85.62 ± 4.07	2.40 ± 0.07^{B}
β-Carotene	2.77 ± 0.07^{B}	91.80 ± 4.47	2.26 ± 0.05^{AB}
P/S*Vit	NS	NS	NS

1-3) See the explanation of Table 3
4) Superoxide dismutase activities were expressed as Units/min/mg protein
(1 Unit was defined as the inhibition of cytochrome C reduction by 50%)
5) Glutathione peroxidase activities were expressed as μmoles of NADPH oxidized/min/mg protein

Vitamin E is known to serve as a lipid soluble chain-breaking antioxidant, to neutralize free radical, to reduce formation of various free radical species, to inhibit the lipid peroxidation and to protect the cell membrane from oxidative stress[27)]. Buckingham[14)] studied the effect of various P/S and vitamin E level on lipid peroxidation. When vitamin E was deficient or supplemented at 10IU/kg diet, TBARS concentrations were significantly increased in all levels of dietary P/S. But when vitamin E was supplemented at 40, 100 IU/Kg diet, lipid peroxidation was inhibited regardless of dietary P/S ratio. These results were similar to the results of this experiment, suggesting that TBARS contents were affected by tocopherol supplement more sensitively than by dietary P/S level.

β-Carotene is known to be one of the most effective quenchers of singlet oxygen[28)]. But effect of β-carotene on the lipid peroxidation is not clear. There are some reports[29)] showing that β-carotene delayed the MDA production in cell membrane and liposome, but other reports[30)] showed that β-carotene accelerated lipid peroxidation and increased oxidative enzyme activity. Ayres et al.[31)] compared the antioxidant activity of Vitamin E and β-carotene and found that vitamin E reduced LDL oxidation and TBARS production. However β-carotene showed no antioxidant effect. About the lack of antioxidant effect of β-carotene supplement, Alam and Alam[32)] suggested that the transfer of β-carotene across the in-

Table 7. Correlation coefficient between serum lipid level and composition, liver lipid peroxide level and antioxidant enzyme activities

	TC	TG	SFA	MUFA	PUFA	PUFA /SFA	PI	TBARS	SOD	GSHPx
TC	-									
TG	0.44**	-								
SFA	-0.10	-0.14	-							
MUFA	0.29**	0.79**	-0.17	-						
PUFA	-0.25*	-0.72**	-0.32**	-0.84**	-					
PUFA/SFA	-0.22*	-0.49**	-0.65**	-0.64**	0.92**	-				
PI	-0.05	-0.71**	-0.04	-0.80**	0.84**	0.66**	-			
TBARS	-0.16	-0.06	-0.07	-0.10	0.09	0.12	0.00	-		
SOD	-0.10	-0.22	0.02	-0.17	0.16	0.13	0.15	0.27*	-	
GSHPx	-0.28**	-0.44**	-0.12	-0.38**	0.41**	0.46**	0.31**	-0.02	0.15	-

* : Pearson correlation is significant at $p<0.05$ ** : Pearson correlation is significant at $p<0.01$

testine in rats was to be quite inefficient, therefore, the tissue and plasma levels of β-carotene were not increased enough to exert a possible antioxidant effect. Conflicting evidence was shown to function as an antioxidant, a prooxidant, or neither.

Results of this experiment showed no effect of β-carotene on TBARS production.

5. Antioxidant enzyme activities in liver

Activities of liver superoxide dismutase(SOD) and glutathione peroxidase(GSHPx) are shown in Table 6. Table 7 showed the correlation coefficient between serum lipid level and composition, PI, TBARS, and antioxidant enzyme activities.

As cellular defenses against free radical damage, antioxidant enzyme systems as well as antioxidant vitamins have evolved scavenge superoxide, hydrogen peroxide and lipid peroxide[33]. SOD activity was not significantly influenced by P/S ratio of dietary lipid and vitamin supplement. But it showed the tendency to increase in high P/S groups(HP-S, HP-T, HP-C), and β-carotene supplemented group. SOD activity showed significant positive correlation with TBARS content. GSHPx activity tended to be high in high P/S groups and significantly higher in α-tocopherol supplemented groups than other vitamin groups. GSHPx activity showed a negative correlation with serum TC, TG, MUFA level and a positive correlation with serum PUFA, P/S and PI.

Kim[21] reported that SOD and GSHPx activities were not changed by dietary lipid pattern. Nam and Park[25] showed the importance of tocopherol rather than SOD activity to lower the production of lipid peroxide. Meydani et al.[34] observed that GSHPx activity was decreased in Vit E deficiency, but other investigators[35][36] observed no effect of vitamin E status on the GSHPx activity.

In conclusion, high intake of unsaturated fat was effective on reducing total cholesterol and triglyceride concentration in serum, but resulted in the increase of unsaturation of fatty acid profile in serum and lipid peroxidation in liver. Consequently which increased the need of vitamin E and antioxidant enzyme activity to reduce the formation and accumulation of the free radicals and lipid peroxides. When polyunsaturated fat intake increased drastically, to protect from lipid peroxidation, the function of antioxidant vitamins became more important than antioxidant enzymes because the increase of enzyme activity was within the limit of physiologic condition. As a antioxidant vitamin, α-tocopherol efficiently inhibited lipid peroxidation reaction, but β-carotene did not.

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