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Effects of Dietary Fatty Acids on Serum Lipids and Fatty Acid Composition of Serum Phospholipids in Men*

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ABSTRACT

Serum lipid levels and fatty acid composition of serum phospholipids(PL) were investigated in 24 healthy male subjects who consumed either corn oil(CO) rich in linoleic acid(LA), perilla oil(PO) rich in α-linolenic acid(α-LNA), or canola oil acid(OA) as a major fat source for 5 weeks. The PO and the CNO groups showed significant($P<0.05$) increases in serum high density lipoprotein-cholesterol(HDL-C) levels and in ratios of HDL-C/total cholesterol(TC) compared with initial values measured at the beginning of the study. Significantly($p<0.05$) increased concentrations of serum triglycerides(TG) ware observed after 5 weeks of the CO-based diet compared with both its intial value and the concentration observed after 5 weeks with the PO-based diet. Fatty acid composition of serum PL reflected changes in dietary fatty acid composition and metabolism. Compared with the initial levels, significantly increased contents of eicosapentaenoic acid(EPA) and docosahexaenoic acid(DHA) were observed in serum PL of the PO group and significantly increased contents of α-LNA and EPA were observed in the CNO group. Arachidonic acid(AA) content of serum PL did not change in the CO group during the study period, although, the increase in LA was significant($P<0.05$). Compared with the CO-based diet, both the PO and the CNO-based diets seem to have beneficial effects on atherosclerosis by influencing the serum lipid profile and fatty acid composition of serum PL. (*Korean J Nutrition* 30(4) : 415~424, 1997)

KEY WORDS : serum lipids · dietary fatty acids · serum phospholipids fatty acid · alpha-linolenic acid · eicosapentaenoic acid · linoleic acid.

Introduction

Studies on the effects of dietary n-3 and n-6 fatty acids on serum lipids have been performed by researchers worldwide[1)2)3)4)]. Many researchers reported that n-3 polyunsaturated fatty acids(PUFA) reduced serum lipids levels more effectively than n-6 PUFA[3)]. Meanwhile, some demonstrated that the hypolipidemic effect of n-3 PUFA might be due to the degree of unsaturation instead of the position of double bonds[4)]. It is because fish oil rich in eicosapentaenoic acid(EPA, C20 : 5, n-3) was usually provided as the n-3 PUFA source while vegetable oil rich in linoleic acid(LA, C 18 : 2, n-6) was used as the n-6 PUFA source. So, providing vegetable oils either rich in LA or α-linolenic acid(α-LNA, C18 : 3, n-3) would be more reasonable for the comparison of the effects of the n-3 PUFA and the n-6 PUFA on serum lipid levels, regardless of the degree of unsaturation. Beneficial effects of monounsaturated fatty acids(MUFA) on serum lipid profile were reported by some researchers[5)6)]. The fatty acid pattern in serum phospholipids(PL) is

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known to reflect the dietary intake coupled with dietary fatty acid metabolism in liver. Ingested dietary LA and α-LNA can be desaturated and elongated to arachidonic acid(AA, C20 : 4, n-6) or EPA, respectively. However, the significance of the capacity for converting LA and α-LNA to AA or EPA in man is still controversial. In this study we investigated serum lipid levels and serum PL fatty acid composition in men who consumed one of three vegetable oil-based diets with corn oil(CO), perilla oil(PO), or canola oil(CNO), which provide LA, α-LNA, and oleic acid(OA, C18 : 1, n-9), respectively, as their major fatty acids.

Methods

1. Experimental design and subjects

A 5-week metabolic study was conducted with 24 healthy male volunteers aged 20－28y. All subjects were college students who stayed in a university dormitory. In the beginning of the study, subjects were randomly assigned to the three groups and were unaware of their treatment during the experimental period. Eight subjects were in the corn-oil-based diet group, another eight were in the perilla-oil-based diet group, and the others were in the canola-oil-based diet group. Mean values of anthropometric variables of the subjects are shown in Table 1. During the study, subjects were allowed to eat only the meals prepared in a metabolic kitchen. All serving sizes were adjusted individually to provide an appropriate energy content for maintaining a constant body weight for each subject during the study. A dietitian monitored and recorded all food consumed.

2. Serum lipids

Fasting blood samples for serum lipids analysis were obtained from the subjects three times(day 1, 21, and 35) by venipuncture. Serum samples obtained by centrifugation were frozen at －70℃ until analysis. Serum triglycerides(TG)[7], total cholesterol(TC)[8], and high density lipoprotein-cholesterol(HDL-C)[9] were analyzed by enzymatic colorimetric methods(Eiken, Japan). Values for serum low density lipoprotein-cholesterol (LDL-C) were calculated using the following formula[10] :

$$LDL\text{-}C = TC - HDL\text{-}C - TG \times 0.16$$

Portions of a previously frozen serum sample pool were analyzed along with reference standards in all runs to detect day to day variation.

Table 1. Mean valus of anthropometric variables of the subjects

Variables	Co[1](n=8)	PO(n=8)	CNO(n=8)
Age(year)	23.6± 0.41[2)ab]	24.9± 0.49[b]	22.8± 0.43[a]
Height(cm)	171.1± 0.01	170.6± 0.01	170.5± 0.01
Weight(kg)	66.1± 1.02	67.6± 1.62	66.2± 1.97
BMI(kg/m^2)	22.6± 0.40	23.2± 0.55	22.7± 0.53

1) CO : Corn oil group
PO : Perilla oil group
CNO : Canola oil group
2) Mean±SEM. Values in the same row with different superscript litters are significantly different at $p<0.01$ by Tukey's test

3. Fatty acid composition of phospholipids

Total serum lipids were extracted by Folch's method[11] and PL were isolated by thin layer chromatography(TLC) using a developing solvent composed of n-hexane/diethyl ether/acetic acid(80/20/1, v/v). Methylation of fatty acids in PL was carried out using the method of Lepage and Roy[12]. Gas chromatography(HP439GC) with a capillary column and a flame-ionized detector were used to separate and quantitate fatty acid esters. Helium was the carrier gas and oven temperature was programmed to increase 1.5℃/min from 180℃ to 240℃.

4. Statistics

Repeated measures of one-way analysis of variance (ANOVA) were used to determine significance of variations in dependent variables during the study period and among the three groups. The Tukey's test was applied for multiple comparisons. Pearson correlation coefficients were used to determine the relationships between serum lipids and serum PL fatty acid composition. Statistical analysis was done with the SAS system(SAS Institute, Inc. Cary, NC).

Results

1. Consumption of diets and oils

Fatty acid compositions of corn oil, perilla oil, and canola oil used in this study are listed in Table 2. Major fatty acids of the cooking oils were LA in corn oil, α-LNA in perilla oil, and OA in canola oil. Dietary composition of each experimental diet which consumed by each group subjects during the study are shown in Table 3. Some variations in energy and nu-

Table 2. Fatty acid composition of experimental oils

Fatty acid	Experimental oils(%)		
	Corn oil	Perilla oil	Canola oil
16 : 0	12.6	7.2	4.6
18 : 0	1.6	2.1	1.1
18 : 1(n-9)	26.5	17.7	59.6
18 : 2(n-6)	57.1	13.9	21.6
18 : 3(n-3)	0.7	58.5	9.4
PUFA[1]	57.8	72.4	30.0
MUFA[2]	26.5	17.7	59.6
SFA[3]	14.2	9.3	5.7
P/S ratio[4]	4.1	7.8	5.4
n-6/n-3 ratio[5]	81.6	0.2	2.3

1) PUFA : Polyunsaturated fatty acids
2) MUFA : Monounsaturatedfatty acids
3) SFA : Saturated fatty acids
4) P/S ratio : Ratio of PUFA/SFA
5) n-6/n-3 ratio : Ratio of n-6 PUFA/n-3 PUFA

trients consumed are due to the difference in subject's body weights. Fatty acid composition of the diets prepared with the experimental oil reflected the fatty acid composition of the respective cooking oils used for the diets.

2. Serum lipids

Changes of serum lipid concentrations during the experimental period are shown in Table 4. There was no significant difference in the initial values measured at the beginning of the study for the three dietary groups. However, after 5 weeks, the CO group had significantly($p<0.05$) higher serum TG levels than did the PO group. Furthermore, in the CO group, a significantly increased serum TG level was obtained at week 5, compared with its initial value and the value at week 3.

No significant difference was observed in serum TC levels among the dietary groups or during the study period of each group. However, at week 3 and week 5, the serum TC level of the PO group was lower than the initial value.

There were some significant($p<0.05$) period differences in serum HDL-C levels in the PO and the CNO groups. In both groups, serum HDL-C levels increased significantly at week 3 from the initial values. However, the increased serum HDL-C levels observed at week 3 decreased somewhat at week 5 of the study period. Compared with the initial level, the increase in HDL-C levels appeared insignificant in the PO group while the HDL-C level was still significantly high in the CNO group after 5 weeks.

Serum LDL-C levels of the three groups did not change significantly during the study period. However, 7.5%, 14.2%, and 13.3% decreases in LDL-C levels were observed in the CO group, the PO group, and the CNO group, respectively, after 5 weeks of experimental diets.

In the CNO group, a significantly($p<0.05$) lower ratio of LDL-C/HDL-C was observed after 3 weeks of experimental diets. It appears that the low($p<0.05$) ratio is due to the significant increase in HDL-C levels observed at 3 weeks of the CNO-based diet. In the PO group, an insignificant but moderate(25%) decrease from initial valus in the ratio of LDL-C/HDL-C was obtained at week 3.

Significantly increased ratios of HDL-C/TC were obtained in the PO and the CNO groups after 3 and 5 weeks of the experimental period. No significant period difference in the ratio of HDL-C/TC was observed in the CO group.

In conclusion, the PO and the CNO groups showed significant($p<0.05$) increases in serum HDL-C lev-

Table 3. Composition of the experimental diet

Variables	CO[1]	PO	CNO
Energy(kal)	2313 ±34[2]	2325 ± 48	2305 ± 58
CHO[3](% of kcal)	55.5± 0.2	55.4± 0.3	55.6± 0.4
Protein(% of kcal)	14.7± 0.1	14.7± 0.1	14.7± 0.1
Fat(% of kcal)	29.8± 0.3	29.9± 0.4	29.7± 0.4
SFA[4](% of kcal)	8.6± 0.1	7.8± 0.1	7.5± 0.1
MUFA[5](% of kcal)	9.9± 0.1	8.5± 0.1	14.9± 0.4
PUFA[6](% of kcal)	11.3± 0.2	13.1± 0.3	7.3± 0.2

1) CO : Corn oil group
PO : Perilla oil group
CNO : Canola oil group
2) Mean±SEM
3) CHO : Carbohydrate
4) SFA : Saturated fatty acids
5) MUFA : Monounsaturated fatty acids
6) PUFA : Polyunsaturated fatty acids

Table 4. Changes of serum lipid levels during the experimental period

Variables	Dietary oil	Experimental period		
		Initial	3 weeks	5 weeks
TG[2](mg/dl)	CO	91.7± 9.9[1]	87.7±12.8	127.4±15.3[b#†]
	PO	100.7±11.9	105.6±15.7	93.3± 9.6[a]
	CNO	80.2±12.1	110.6±19.1	102.1±14.9[ab]
TC[3](mg/dl)	CO	137.3± 8.2	146.0±13.5	138.9±10.9
	PO	141.4±12.1	134.1±16.6	135.1± 6.9
	CNO	116.4± 7.9	135.9± 8.8	117.8±11.7
HDL-C[4](mg/dl)	CO	36.4± 3.6	40.9± 2.9	38.6± 2.4
	PO	32.8± 0.8	41.8± 3.0[#]	40.7± 3.9
	CNO	29.1± 2.2	42.1± 3.0[#]	36.9± 3.7[#]
LDL-C[5](mg/dl)	CO	86.3± 9.8	91.1±15.3	79.8±11.0
	PO	92.5± 8.2	75.4±17.7	79.4± 9.9
	CNO	74.5± 6.6	76.1± 8.4	64.6±10.4
LDL-C/HDL-C	CO	2.6± 0.4	2.4± 0.5	2.2± 0.3
	PO	2.8± 0.3	2.1± 0.7	2.3± 0.5
	CNO	2.7± 0.3	1.8± 0.2[#]	1.9± 0.5
HDL-C/TC×100(%)	CO	27.9± 4.7	31.1± 5.3	29.4± 3.3
	PO	23.7± 1.4	36.2± 6.2[#]	34.2± 3.5
	CNO	25.6± 2.3	31.4± 1.9[#]	33.3± 3.9[#]

1) Mean±SEM. Means with different letters within the same column are significantly different among diet groups. Analyzed by Repeated Measure Design
[#] Significantly different from the initial value at $p<0.05$ in the same diet group by Repeated Measure Design
[†] Significantly different from the third week's value at $p<0.05$ in the same diet group by Repeated Measure Design
2) TG : Triglycerides
3) TC : Total cholesterol
4) HDL-C : High density lipoprotein cholesterol
5) LDL-C : Low density lipoprotein cholesterol

els and ratio of HDL-C/TC, while showing a decrease in the ratio of LDL-C/HDL-C($p<0.05$) only in the CNO group, after feeding with experimental diets.

3. Fatty acid composition of serum phospholipids

Fatty acid compositions of serum PL in each dietary group were determined three times during the experimental period(Table 5, 6, 7). In the tables, we listed the major fatty acids affected by cooking oils and dietary fatty acid metabolism.

In the CO group(Table 5) concentration of LA increased 48%($p<0.05$) from the initial level after 3 weeks, but it appeared to decrease to the initial value at week 5. Similar trends were shown in concentrations of total n-6 PUFA and ratio of n-6/n-3. The CO-based diet raised concentration of total n-6 PUFA 21%, and raised the ratio of n-6/n-3 48% at week 3. However, the changes were not significant. After 5 weeks on the CO-based diet, the values of total n-6 and ratio of n-6/n-3 decreased to even below the initial values. So, concentration of total n-6 PUFA and ratio of n-6/n-3 at week 5 were significantly lower than the values at week 3. Meanwhile, no period difference was observed in the content of AA.

Significant($p<0.05$) increases of as much as 20% in total PUFA, 120% in total n-3 PUFA, 212% in docosahexaenoic acid(DHA, C22 : 6, n-3), and 164% in ratio of(EPA+DHA)/AA were observed after 5 weeks on the PO-based diet(Table 6). 20–50% decreases in MUFA($p<0.05$), ratio of the n-6/n-3, and OA were observed after 5 weeks on the PO-based diet. Significant($p<0.05$) increases in total PUFA and EPA,

Table 5. Fatty acid composition of serum phospholipids in the corn oil group(area %)

Fatty acid	Initial(n=8)	3 weeks(n=8)	5 weeks(n=8)
PUFA[1]	26.18±4.43[2]	30.21±1.24	24.29±2.48
MUFA[3]	17.98±5.74	16.77±2.02	21.59±1.12#
SFA[4]	55.84±6.48	53.02±2.89	54.13±2.14
P/S ratio[5]	0.47±0.08	0.59±0.07	0.47±0.07
n-6 PUFA	21.54±3.72	26.10±1.05	19.47±1.98†
n-3 PUFA	4.63±0.96	4.10±0.58	4.81±0.88
n-6/n-3 ratio[6]	5.13±0.75	7.60±1.37	4.62±0.70†
C18 : 1(n-9)	10.23±1.09	13.07±1.77	12.95±0.95
C18 : 2(n-6)	13.37±2.32	19.75±1.69#	13.21±1.81†
C18 : 3(n-3)	0.35±0.23	0.23±0.05	0.65±0.12†
C20 : 4(n-6)	4.69±1.07	4.16±0.76	4.56±1.35
C20 : 5(n-3)	0.63±0.25	1.66±0.50	1.00±0.12
C22 : 6(n-3)	2.09±0.83	1.54±0.83	1.60±0.16
(EPA+DHA)A/A[7]	0.47±0.13	1.35±0.58	0.90±0.18

1) PUFA : Polyunsaturated fatty acids
2) Mean±SEM
Significantly different from the initial value at $p<0.05$ in the same row by Repeated Measure Design
† Significantly different from the third week's value at $p<0.05$ in the same row by Repeated Measure Design
3) MUFA : Monounsaturated fatty acids
4) SFA : Saturated fatty acids
5) P/S ratio : Ratio of PUFA/SFA
6) n-6/n-3 ratio : Ratio of n-6 PUFA/n-3 PUFA
7) EPA : Eicosapentaenoic acid, C20 : 5(n-3) DHA : Docosahexaenoic acid, C22 : 6(n-3)
AA : Arachidonic acid, C20 : 4(n-6)

Table 6. Fatty acid composition of serum phospholipids in the perilla oil group(area %)

Fatty acid	Initial(n=8)	3 weeks(n=8)	5 weeks(n=8)
PUFA[1]	20.10±3.04[2]	27.98±0.82#	24.71±3.02#
MUFA[3]	24.29±2.49	16.25±0.38#	16.77±1.96#
SFA[4]	55.59±1.81	55.77±1.05	58.52±1.88
P/S ratio[5]	0.37±0.07	0.50±0.02	0.44±0.07
n-6 PUFA	16.79±2.56	23.29±0.64	17.43±2.57
n-3 PUFA	3.31±0.55	4.68±0.62	7.28±0.85#
n-6/n-3 ratio[6]	5.53±0.85	6.02±1.26	2.75±0.56#
C18 : 1(n-9)	15.71±0.96	11.81±0.36#	12.10±1.31#
C18 : 2(n-6)	10.33±2.72	17.90±0.63#	8.87±1.96†
C18 : 3(n-3)	0.73±0.21	0.75±0.07	0.78±0.07
C20 : 4(n-6)	3.81±0.72	3.14±0.34	4.20±1.31
C20 : 5(n-3)	1.05±0.30	3.14±0.62#	1.65±0.34
C22 : 6(n-3)	1.46±0.43	0.73±0.09	4.56±0.39#
(EPA+DHA)A/A[7]	0.75±0.13	1.41±0.29	1.98±0.29#

1) PUFA : Polyunsaturated fatty acids
2) Mean±SEM
Significantly different from the initial value at $p<0.05$ in the same row by Repeated Measure Design
† Significantly different from the third week's value at $p<0.05$ in the same row by Repeated Measure Design
3) MUFA : Monounsaturated fatty acids
4) SFA : Saturated fatty acids
5) P/S ratio : Ratio of PUFA/SFA
6) n-6/n-3 ratio : Ratio of n-6 PUFA/n-3 PUFA
7) EPA : Eicosapentaenoic acid, C20 : 5(n-3) DHA : Docosahexaenoic acid, C22 : 6(n-3)
AA : Arachidonic acid, C20 : 4(n-6)

Table 6. Fatty acid composition of serum phospholipids in the canola oil group(area %)

Fatty acid	Initial(n=8)	3 weeks(n=8)	5 weeks(n=8)
PUFA[1)]	22.90±2.51[2)]	32.63±4.24	21.26±1.04
MUFA[3)]	22.97±1.92	16.80±2.61	26.08±1.60
SFA[4)]	55.22±2.35	50.57±2.19	52.79±1.53
P/S ratio[5)]	0.55±0.17	0.59±0.10	0.40±0.03
n-6 PUFA	20.33±1.85	29.12±3.94	15.53±1.23#†
n-3 PUFA	2.57±0.91	3.52±0.83	5.73±0.88
n-6/n-3 ratio[6)]	12.23±2.59	11.21±2.65	3.57±0.92#†
C18 : 1(n-9)	15.43±0.68	18.22±1.59	17.44±1.15
C18 : 2(n-6)	8.85±1.22	13.15±1.75#	8.77±1.37†
C18 : 3(n-3)	0.16±0.07	0.37±0.07	1.00±0.12#†
C20 : 4(n-6)	4.16±0.60	6.05±1.53	3.18±0.31
C20 : 5(n-3)	0.40±0.20	0.93±0.31	1.58±0.32#
C22 : 6(n-3)	1.76±0.61	1.99±0.66	3.24±0.58
(EPA+DHA)A/A[7)]	0.44±0.12	0.63±0.16	1.49±0.58

1) PUFA : Polyunsaturated fatty acids
2) Mean±SEM
Significantly different from the initial value at $p<0.05$ in the same row by Repeated Measure Design
† Significantly different from the third week's value at $p<0.05$ in the same row by Repeated Measure Design
3) MUFA : Monounsaturated fatty acids
4) SFA : Saturated fatty acids
5) P/S ratio : Ratio of PUFA/SFA
6) n-6/n-3 ratio : Ratio of n-6 PUFA/n-3 PUFA
7) EPA : Eicosapentaenoic acid, C20 : 5(n-3) DHA : Docosahexaenoic acid, C22 : 6(n-3)
AA : Arachidonic acid, C20 : 4(n-6)

and significant($p<0.05$) decreases in MUFA and OA were also observed after 3 weeks on the PO-based diet. In the PO group, concentration of LA increased by 73%($p<0.05$) after 3 weeks, and then it decreased to even below the initial value after 5 weeks of this diet. No significant period difference was observed in α-LNA concentration of serum PL in the PO group (Table 6).

Significant($p<0.05$) changes in several fatty acids were observed, after 5 weeks of the CNO-based diet (Table 7). Concentrations of α-LNA, EPA, and the ratio of(EPA+DHA)/AA increased($p<0.05$) 525%, 295%, and 239%, respectively. Significant($p<0.05$) decreases in concentration of total n-6 PUFA(24%) and in the ratio of n-6/n-3(71%) were observed(Table 7).

4. Relationship between serum lipids and serum phospholipids fatty acids

Significant relationships between serum lipids and serum PL fatty acids are listed in Table 8. Negative relationships between serum TG levels and ratios of (EPA+DHA)/AA were obtained in all dietary groups. In the PO group, serum HDL-C showed a negative relationship with a ratio of n-6/n-3, and positive relationships with the ratio of(EPA+DHA)/AA and EPA. In the CNO group, serum TC was negatively correlated with SFA, n-3 PUFA, and DHA, whereas PUFA, OA, and n-6 series PUFA were positively correlated with this variable. Oleic acid was positively correlated with both serum HDL-C and LDL-C. Negative relationships were observed between HDL-C and α-LNA and between LDL-C and DHA. In the CO group, there was no significant relationship beetween serum lipids and serum PL fatty acids.

Discussion

There has been a large variation and much inonsistency in the results of studies on the effects of dietary fatty acids on serum lipids in human and animal subjects[1)]. Our results are consistent with previous findigs that n-3 PUFA-based diets have more protective effects than n-6 PUFA-based diets on the process of atherogensis by influencing serum lipid lev-

Table 8. Significant relationships between serum lipids and serum phospholipids fatty acids

Diet group	Factors*		r[1]	Period
All[2]	TG	× (EPA+DHA)/AA	−0.4737#	5 week
CO[3]	none			
PO[4]	HDL-C	× n-6/n-3 ratio	−0.8112#	3 week
		EPA	0.4837#	All
		(EPA+DHA)/AA	0.4937#	All
CNO[5]	TC	× PUFA	0.8632#	3 week
		SFA	−0.9245†	3 week
		P/S ratio	0.8975#	3 week
		n-6 PUFA	0.8636#	3 week
		LA	0.8397#	3 week
		n-3 PUFA	−0.9458#	5 week
		DHA	−0.9109†	5 week
		OA	0.6018#	All
	HDL-C	× OA	0.8114#	3 week
		α-LNA	−0.8639#	3 week
	LDL-C	× SFA	−0.9057†	3 week
		P/S ratio	0.8025#	3 week
		DHA	−0.8531#	5 week
		n-6 PUFA	0.5651#	All
		OA	0.5424#	All

1) Pearson correlation coefficients
#$p<0.05$
†$p<0.01$
2) All : all three groups
3) CO : Corn oil group
4) PO : Perilla oil group
5) CNO : Canola oil group
* Listed abbraviations in Factors :
TG : triglycerides, EPA : Eicosapentaenoic acid, 20 : 5(n-3), DHA : Docosahesaenoic acid, 22 : 6(n-3), AA : Arachidonic acid, 20 : 4(n-6), HDL-C : High density lipoprotein cholesterol, n-6/n-3 ratio : Ratio of n-6 PUFA/n-3 PUFA, TC : Total cholesterol, PUFA : Polyunsaturated fatty acids, SFA : Saturated fatty acids, P/S ratio : Ratio of PUFA/SFA, LA : Linoleic acid, 18 : 2(n-6), OA/Oleic acid, 18 : 1(n-9), α-LNA : α-Linolenic acid, 18 : 2(n-3)

els, especially influncing serum TG and serum HDL-C levels[13]. In addition, the MUFA-based diet of this study did not differ from n-3 PUFA based diet as far as influence on serum lipid levels are concerned.

Perilla oil, a commonly used cooking oil in Korea, contains as much as 60% α-LNA of its total fatty acids. In this study the PO-based diet lowered serum TG levels significantly compared with the CO-based diet that provides LA. Several studies, including clinical trials and epidemiological observations, have demonstrated a pronounced effect of n-3 PUFA in lowering serum TG levels[2][14]. Dietary n-3 PUFA, especially in fish oil, have been known to be more hypotriglyceridemic than equivalent amounts of n-6 PUFA[3]. Earlier studies have demonstrated the mechanisms involved in the hypotriglyceridemic effect of n-3 PUFA. Dietary n-3 PUFA, especially in fish oil, decrease the VLDL-TG pool by suppressing TG synthesis and limiting VLDL secretion, and possibly enhancing the clearance of VLDL-TG by peripheral tissues or the liver[15][16]. Even though the mechanism has not been examined, this study demonstrated the hypotriglyceridemic effect of a vegetable oil rich in n-3 PUFA compared with an n-6 PUFA containing oil.

The effects of the consumption of n-6 or n-3 PUFA or MUFA on serum LDL-C and HDL-C levels conflict with the results of previous studies because of differences in dosage, composition of the dietary fatty acids, plasma lipid concentration of subjects, and duration of the study[17]. The concentration of LDL

and HDL are reported to rise, fall, or not change after feeding n-6 PUFA, n-3 PUFA, or MUFA-based diets[1]. Failor et al.[18] observed a significant reduction in TC and LDL-C levels in hyperlipidemic subjects consuming n-3 or n-6 PUFA in place of saturated fatty acid(SFA). A clinical trial by Mattson and Grundy[5] showed that OA was as effective as LA in lowering LDL-C levels, after feeding OA and LA-based diets in place of palmitic oil to hypercholesterolemic patients. However, they observed that LA lowered serum HDL-C levels, too. Meanwhile, Wardlaw et al.[6] showed that both MUFA and n-6 PUFA reduced LDL-C significantly with no significant change in HDL-C concentration. In this study, the CNO-based diet and the PO diet significantly elevated serum HDL-C levels and the ratio of HDL-C/TC compared with the initial values. Canola oil provides OA as its major fatty acid and 10% α-LNA of its total fatty acids. There were no significant period differences in serum TC, LDL-C, and HDL-C levels of the subjects consuming the CO-based diet.

Therefore, the PO and the CNO-based diets of this study seem to have more beneficial effects than CO-based diets on the process of atherogenesis by mediating serum lipid levels.

It has been reported by many researchers[19] that serum PL fatty acid patterns reflected dietary fatty acid composition as well as fatty acid synthesis and modification occurring in the liver. In this study, fatty acid patterns in serum PL reflected fatty acid composition of the cooking oil used for the diets and dietary fatty acid metabolism in the body(Table 5, 6, 7). Significant decreases from initial values in the ratio of n-6/n-3 and significant increases in EPA and ratio of(EPA+DHA)/AA were observed in both the PO and the CNO groups. Also, significant increases in DHA and total n-3 PUFA were observed in the PO group, and significant decreases in total n-6 PUFA and insignificant but moderate increase in α-LNA were observed in the CNO group after feeding with the oil-based diets. In the CO group, significant increases in LA and insignificant but moderate increases in total n-6 PUFA content were observed 3 weeks after initiation of the diet.

The capacity for converting α-LNA to its derivatives, such as EPA or DHA, in man is controversial. Many researchers observed significant increases in longer chain n-3 fatty acid concentrations in humans when a diet rich in α-LNA was consumed[20]. Lassere et al.[21] noted that higher values of EPA were observed in serum PL when subjects consumed a diet containing canola oil rather than a vegetable oil rich in LA. However, Dyerberg et al.[22] did not find any significant conversion of α-LNA to EPA and DHA. In this study α-LNA appeared to be converted to EPA or DHA in significant amounts by the subjects who consumed the PO or the CNO-based diets. In the PO group, significant increases in EPA at week 3 and significant increases in DHA at week 5 were observed without significant changes in α-LNA content (Table 6). The increase in EPA and α-LNA, and decrease in DHA of serum PL observed at 3 week of the PO-based diet may result from the acute effect ; that is, the increased EPA converted from α-LNA displaced DHA in serum PL. However, at week 5 on the PO-based diet, the DHA content is elevated significantly while the EPA content returned to the initial value. It has been demonstrated in healthy human subjects that the delta-4 desaturase converts EPA to DHA, and DHA is known as a reservior form of EPA in plasma and cell PL[23]. The conversion of α-LNA to EPA may occur within 3 weeks after ingestion of α-LNA in the PO group, and then the conversion of EPA to DHA occurs later. So, a significant increase in DHA content of serum PL observed after 5 weeks of the PO-based diet is regarded as a relatively long-term effect of n-3 PUFA metabolism. Meanwhile, in the CNO group, significant increases in α-LNA and EPA and insignificant but moderate(84%) increase in DHA were obtained after 5 weeks of the CNO-based diets(Table 7). Thus, α-LNA in CNO also increased EPA and DHA concentration of serum PL even though degree of the conversion of α-LNA to EPA or DHA was less than what occurred in the PO group. Due to this significant conversion, significantly increased ratios of(EPA+DHA)/AA were observed in both the PO and the CNO groups after 5 weeks of the oil-based diets.

Other studies have shown increased LA concent-rations but decreased or constant AA concentration after eating LA-rich diets[24][25]. A signigicant increase in LA, but not in AA, was also oberved after 3 weeks of

the CO-based diet(Table 5). Even though it is well established that dietary LA is converted to AA in vivo[21], the capacity for this conversion in man is controversial. Mantzioris et al.[25] demonstrated significant conversion of dietary α-LNA to EPA in men. However, they did not detect conversion of dietary LA to AA in plasma and blood cells, like neutrophils, mononuclear cells, and platelets of healthy human subjects who consumed either α-LNA or LA rich diets. We also observed significantly increased LA levels of serum PL after 3 weeks of the PO-based and the CNO-based diets(Table 6, 7). We assumed this result was due to increased biosynthesis of EPA from α-LNA and suppressed biosynthesis of AA from LA after feeding on α-LNA rich diets. The same enzymes are involved and compete in the biosynthesis of AA and EPA from LA and α-LNA, respectively, and the relative rate of enzymatic elongation and desaturation of C18 fatty acid series was reported to be in the following order : n-3 > n-6 > n-9[26]. In general, increased concentrations of cellular EPA have been known to be beneficial in atherosclerosis. Eicosanoids derived from EPA have greater antiaggregatory and vasodilatory effects than those from AA.

Usually, changing from one type of diet to another causes an exaggerated response in metabolic parameters like serum lipid concentration for a short-term period. However, after a certain period of adaptation to a diet, there is a tendency to obtain a relatively less significant difference. In this study, effects of dietary fatty acids on the fatty acid composition of serum PL were observed after 3 weeks. At week 5 after initiation of the oil-based diet, the dietary induced effects on serum PL fatty acid composition became smaller, or the composition returned to initial levels. So, five weeks may be an intermediate period in which neither an acute effect nor a long-term effect of diet modification on the fatty acid composition of serum PL is shown. Popp-snijders and Blonk[19][27] noted that fatty acid patterns in serum PL reflected short-term intake of dietary fatty acids. They demonstrated that fatty acid composition in adipose tissue reflected long-term intake of dietary fatty acids[27].

Compared with n-6 PUFA, longer chain n-3 PUFA, such as EPA and DHA, have beneficial effects on atherosclerosis and serum lipid profiles(Table 8). EPA, DHA, (EPA+DHA)/AA, or total n-3 PUFA were negatively correlated with serum TC or LDL-C, while they were positively correlated with serum HDL-C. Meanwhile, n-6 PUFA, LA, or the ratio of n-6/n-3 showed positive relationships with serum TC and LDL-C, and negative relationships with serum HDL-C. Considering the relationships between serum lipids and serum PL fatty acids, the beneficial effects of the PO-based diets and the CNO-based diets on serum lipid profiles seem to related to increased concentrations of EPA and DHA in serum PL of both groups.

In this study, PO rich in α-LNA appears to allow an effective method for increasing serum HDL-C levels as well as EPA and DHA concentrations of serum PL. Fish oil rich in EPA has been reported to have beneficial effects on atherosclerosis by influencing the serum lipid profile and fatty acid composition of the cells[16]. So, PO, which is a vegetable oil containing n-3 PUFA, may replace or be used as an adjunct to fish oil treatment taken to reduced the risk of atherosclerosis. This study also shows the beneficial effects of dietary MUFA on the process of atherosclerosis by increasing serum HDL-C levels without changing LDL-C.

Literature cited

1) Nestel PJ. Effects of n-3 fatty acids on lipid metabolism *Annu Rev Nutr* 10 : 149-167, 1990
2) Renaud S, de Lorgeril M. Dietary lipids and their relation to ischemic heart disease : from epidemiology to prevention. *J Intern Med* 225 : 39-46, 1989
3) Harris WS, Connor WE, McMurry MP. The comparative reductions of the plasma lipids and lipoproteins by dietary polyunsaturated fats : Salmon oil versus vegetable oils. *Metabolism* 32 : 179-184, 1983
4) Ide T, Tanaka T, Sugano M. Regulation by dietary fats of 3-hydroxy-3-methyl glutaryl CoA reductase in rat liver. *J Nutr* 109 : 601-612, 1978
5) Mattson FH, Grundy SM. Comparison of effects of dietary saturated, monounsaturated, and polyunsaturated fatty acids on plasma lipids and lipoproteins in man. *J Lipid res* 26 : 194-202, 1985
6) Wardlaw GM, Snook JT, Lin MC, Puangco MA, Kwon JS. Serum lipid and apolipoprotein concentrations in healthy men on diets enriched in either canola oil or safflower oil. *Am J Clin Nutr* 54 : 104-110, 1991

7) Bucolo G, David H. Quantitative determination of serum triglycerides by the use of enzymes. *Clin Chem* 19 : 476-482, 1973

8) Allain CC, Poon LS, Chan CGS, Richmond W, Fu PC. Enzymatic determination of serum total cholesterol. *Clin Chem* 20 : 470-475, 1974

9) Allen JK, Hensley WJ, Nichols AV, Whitfield JB. An enzymatic and centrifugal method for estimating high-density lipoprotein cholesterol. *Clin Chem* 25 : 325-327, 1979

10) Fridwald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low density lipoprotein cholesterol in plasma without use of the preparative ultracentifuge. *Clin Chem* 18 : 499-502, 1972

11) Folch J, Lees M, Stanley GHS. A simple method for the isolation and purification of total lipids from animal tissue. *J Biol Chem* 226 : 497-509, 1957

12) Lepage G, Roy GC. Direct trans-esterification of all classes of lipids in one step reaction. *J lipid Res* 27 : 114-129, 1986

13) Kestin M, Clifton P, Bellin GB, Nestel PJ. N-3 fatty acids of marine origin lower systolic blood pressure and triglycerides but raise LDL-cholesterol compared with n-3 and n-6 fatty acids from plants. *Am J Clin Nutr* 51 : 1028-1034, 1990

14) Herold P, Kinsella JE. Fish oil consumption and decreased risk of cardiovascular disease ; A comparison of findings from animal and human feeding trials. *Am J Clin Nutr* 43 : 566-573, 1986

15) Harris WS, Connor WE, Inkeles SB, Illingworth DR. Dietary omega-3 fatty acids prevent carbohydrate-induced hypertriglycerdmia. *Metabolism* 33 : 1016-1019, 1984

16) Harris WS. Fish oils and plasma lipid and lipoprotein metabolism in humans : Critical review. *J Lipid Res* 30 : 785-807, 1989

17) Kinsella JE. Requirements and sources of n-3 polyunsaturated fatty acids in the human diet. In : Karel M, Lees R, eds. Proceedings of the MIT conference on fish oils. New York : Marcel Dekker. 1989

18) Failor RA, Childs MT, Berman E. The effects of ω-3 and ω-6 fatty acid enriched diets on plasma lipoprotein and apoproteins in familial combined hyperlipidemia. *Metabolism* 37 : 1021-1027, 1988

19) Popp-Snijders C. Assessment of compliance to changes in dietary fatty acids. *Diabetes Nutr Metab* 4 : 155-163, 1991

20) Sanders TAB, Younger KM. The effect of dietary supplements of ω-3 polynsaturated fatty acids on the fatty acid composition of platelets and plasma choline phosphoglycerides. *Br J Nutr* 45 : 613-616, 1981

21) Lassere M, Mendy F, Spielmann D, Jacotot B. Effects of different dietary intake of essential fatty acids on C20 : 3 ω6 and C20 : 4 ω6 serum levels in human adults. *Lipids* 20 : 227-233, 1985

22) Dyerberg J, Bang HO, Aagaard O. Alpha-linolenic acid and eicosapentaenoic acid. *Lancet* 1 : 199, 1980

23) Von Schacky C, Weber PC. Metabolism and effects on platelet function of the purified eicosapentaenoic and docosahexaenoic acids in human. *J Clin Invest* 76-2446-2450, 1985 :

24) Renaud S, Godsey F, Dumont E, Thevenon C, Ortchanian E, Martin JL. Influence of long-term diet modification on platelet function and composition in Moselle farmers. *Am J Clin Nutr* 43 : 136-150, 1986

25) Mantzioris E, James MJ, Gibson RA, Cleland LG. Differences exist in the relations between dietary linoleic and alpha-linolenic acids and their respective long-chain metabolites. *Am J Clin Nutr* 61 : 320-324, 1995

26) Siguel EN, Maclure M. Relative activity of unsaturated fatty acid metabolism pathways in humans. *Metabolism* 36 : 664-669, 1987

27) Popp-Snijders C, Blonk MC. Omega-3 fatty acids in adipose tissue of obese patients with non-insulin-dependent diabetes mellitus reflect long-term dietary intake of eicosapentaenoic acid docosahexaenoic acid. *Am J Clin Nutr* 61 : 360-365, 1995