Korean J Nutrition 30(9) : 1073~1083, 1997

Effects of Consuming Diets Incorporating Soybean Oil, Canola Oil, and Palm Oil on Serum Lipids and Fecal Fat Excretion in Humans

Jin, Young Hee

Department of Food Science and Technology, Kimcheon Junior College, Kimcheon, Korea

ABSTRACT

This project was to compare the effects of consuming diets incorporating soybean oil, canola oil, and palm oil on blood serum lipid patterns and fecal fat excretion in healthy human adults. The project was composed of three studies that were run concurrently. Each study was composed of a 4-day pre-trial period and two, 14-day experimental periods arranged according to a cross-over design. During the pre trial period, subjects were allowed to eat self-selected diets. During the experimental period, subjects were asked to eat constant, laboratory controlled diets in which sources of dietary fat varied. Thirty healthy, young adult subjects were fed a laboratory controlled diet incorporating soybean oil, canola oil, and palm oil. Test oils provided approximately 20% of the total dietary calories from all sources. Total dietary fat from all sources was approximately 30% of consumed calories. The highest serum total cholesterol, LDL cholesterol, VLDL cholesterol, and triglyceride values occurred when palm oil diets were eaten. However, feeding on palm oil diets resulted in the same HDL cholesterol content as with soybean oil or canola oil diets. Soybean oil diets produced numerically lower blood serum total cholesterol, LDL cholesterol, and triglycerides, and numerically higher HDL cholesterol and VLDL cholesterol than did those with canola oil. Fecal fat excretion was numerically lower with soybean oil diets than with canola oil diets. The highest fecal fat excretion occurred when palm oil diets were eaten. (*Korean J Nutrition* 30(9) : 1073~1083, 1997)

KEY WORDS : serum lipids · fat excretion · soybean oil · canola oil · palm oil.

Introduction

Coronary heart disease(CHD) is one of the most frequent causes of death in the developed and developing nations. Despite substantial success in reducing CHD mortality in the past two decades, the disease is still responsible for more than 50,000 deaths annually in the United States. About 20% of hospital discharges for acute CHD are for premature disease, i. e., in patients under 55 years of age[1]. The incidence of cardiovascular disease(CVD) has been increasing rapidly in Korea[2] and the mortality rate from this disease reached 29.9% in 1990[3].

Because coronary heart disease is a multifactorial disease, many risk factors can be associated with it. Several inherited and lifestyle factors appear to influence the risk of suffering from this disease. Those considered to be most significant include genetic background, cigarette smoking, sex, obesity, hypercholesterolemia, hypertension, diabetes mellitus, physical inactivity, and, of course, diet[4]. Dietary changes have been stressed in prevention programs because of the theoretical causes and practices by which these alterations can be made.

Accepted : September 8, 1997

Epidemiological studies have identified various factors associated with both the increased and decreased incidence of coronary heart disease. Among these factors, dietary practices have been among the first considered. Positive and negative significant correlations between coronary heart disease incidence and intake of fat, carbohydrates, and protein have been found. Because the principle component of plaque is lipid, types and levels of dietary fat and cholesterol were the first, and still remain the most frequently investigated, dietary factors.

Currently, in the United States and in the world as a whole, soybean oil is the most popular plant oil in terms of total production and usage in human foods. The current leadership position of soybean oil is now being threatened by two other plant oils : palm oil and rapeseed oils(including canola)[5][6]. Palm oil represents the second largest volume of vegetable oil produced in the world[7]. Consumption of canola oil is being urged on the basis of health promotion characteristics thought to be related to its high concentrations of oleic acid, a monounsaturated fatty acid. Concurrently, health values of plant oils like soybean oil that are rich sources of polyunsaturated fatty acids are being either dismissed or downgraded. The objective of this project was to compare the effects of consuming diets incorporating soybean oil, canola oil, and palm oil on blood serum lipid patterns and fecal fat excretion in healthy human adults.

Materials and Methods

1. Study design

The project was composed of three studies that were run concurrently. Each study was composed of a 4-day pre-period and two 14-day experimental periods arranged according to a cross-over design (Table 1). During the pre-period, subjects were asked to keep complete collections of stools but were allowed to eat self-selected diets. During the experimental periods, subjects were asked to eat constant, laboratory controlled diets(Table 2) in which source of dietary fat was varied. They also had to continue keeping complete collections of stools and giving fasting blood samples. Test fats provided approximately 20% of total dietary energy from all sources. Total dietary fat from all sources was about 30% of total calories. Eggs were included to keep total cholesterol intake at approximately 300mg/day. These levels met the current U.S. Dietary Guidelines/Goals. Test fats that were used in each study

Table 1. Experimental design

Period	# Days	Diet type	Test fat source[2]
Study A			
Pre-	4	Self-selected	–
1	14	Laboratory controlled[1]	Soy
2	14	Laboratory controlled	Canola
Study B			
Pre-	4	Self-selected	–
1	14	Laboratory controlled	Soy
2	14	Laboratory controlled	Palm
Study C			
Pre-	4	Self-selected	–
1	14	Laboratory controlled	Canola
2	14	Laboratory controlled	Palm

1) See Table 2
2) 20% of total Calories

Table 2. Basal diet

Food item	Amount(g/day)
Breakfast	
Orange juice	261
Milk(skim)	287
Bread[1]	
Soybean oil	139
Canola oil	137
Palm oil	139
Lunch	
Bread[1]	
Soybean oil	139
Canola oil	137
Palm oil	139
Milk(skim)	287
Chicken soup	45
Tomato juice	249
Potato chips	37
Fruit cup(diced peaches)	29
Supper	
Bread[1]	
Soybean oil	139
Canola oil	137
Palm oil	139
Spaghettio's	214
Egg	54
Fruit punch	257
Yogurt	136

1) Bread made with soybean, canola or palm oil
Jelly and life savers are optional for calorie adjustment

were as follows : Study A-soybean oil and canola oil, Study B-soybean oil and palm oil, Study C-palm oil and canola oil. Test oils were incorporated into a bread product(Table 3). The fatty acid composition of the test oils is shown in Table 4. Soybean oil, canola oil, or palm oil provide linoleic acid(C18 : 2), oleic acid(C18 : 1), and palmitic acid(C16 : 0), respectively, as their major fatty acids.

2. Subjects

Thirty healthy adult subjects participated in the three studies. All subjects were required to eat only food provided by the studies, preferably in the Ruth Leverton Hall diet laboratory(one meal/day had to be eaten there although other meals could be carried out). Subjects maintained normal daily activities with the exception of eating controlled diets, making complete fecal collections, and donating blood samples during the studies. All subjects were determined to be healthy and received medical clearance from the University Health Services. All signed a required consent form before participation. This project was reviewed and given approval from the University of Nebraska Institutional Committee on Investigations Involving Human Subjects.

Table 3. Bread recipe

Ingredient	Amount	Method
Whole wheat flour	568g	Combine dry ingredients in mixed bowl.
Unenriched flour	596g	
Sugar	170g	
Salt	57g	
Baking powder	57g	
Eggs, beaten	270g	Combine eggs, milk, fat.
Milk	1818g	Add to dry ingredients.
Fat[1](melted, cooled)	170g	Mix(low speed) only until ingredients are moistened. Spread into greased baking pan. Bake at 400° F., 35 minutes.

1) Soybean oil(320 loaves) or Canola oil(320 loaves) or Palm oil(320 loaves)

Table 4. Typical fatty acid composition of test oils(%)

Fatty acid	Fat		
	Soybean oil	Canola oil	Palm oil
Lauric(12 : 0)	–	–	0.1
Myristic(14 : 0)	0.1	0.1	1.0
Palimitic(16 : 0)	10.6	4.1	44.4
Palmitoleic(16 : 1)	0.1	0.3	0.2
Margaric(17 : 0)	0.1	0.1	0.1
Stearic(18 : 0)	4.0	1.8	4.1
Oleic(18 : 1)	23.2	60.9	39.3
Linoleic(18 : 2)	53.7	21.0	10.0
Linolenic(18 : 3)	7.6	8.8	0.4
Arachidic(20 : 0)	0.3	0.7	0.3
Gadoleic(20 : 1)	–	1.0	–
Behenic(22 : 0)	0.3	0.3	0.1
Erucic(22 : 1)	–	0.7	–
Linoceric(24 : 0)	–	0.2	–

3. Sample collections and laboratory analysis

Fasting blood samples were drawn at the beginning and after each experimental period in studies A, B, and C by technicians at the University of Nebraska-Lincoln Health Center. Serum total cholesterol, LDL cholesterol, HDL cholesterol, VLDL cholesterol, triglyceride, and phospholipid contents were analyzed by Roche Biomedical Laboratories, Inc., Kansas City, MO.

Complete fecal collections were made daily by each subject for the duration of the study. Fecal collections were made in opaque polyethylene freezer containers. Feces were divided into period lots representing food eaten during periods by the appearance of fecal dye(brilliant blue) which was given orally at the beginning of each period. Feces were weighed, composited, homogenized into period lots, and freeze dried. Fecal fat excretion was analyzed for using the AOAC modification of the Goldfisch method for fat extraction.

4. Statistical analysis

Statistical analysis of data was completed by use of the Statistical Analysis System(SAS). The Analysis of Variance procedure was performed to detect possible variation resulting from dietary treatment. To determine possible differences between mean responses, Duncan's Multiple Range test was performed.

Results and Discussion

1. Serum lipids

Mean fasting serum lipid values are shown on Tables 5 through 7. Mean fasting serum total cholesterol concentrations for subjects consuming self-selected, soybean oil, and canola oil diets in study A were 186±12, 201±14, and 203±16mg/dl, respec-

Table 5. Mean fasting serum lipid values for humans consuming diets incorporating soybean oil and canola oil, Study A[1]

Serum lipids (mg/dl)	Diet treatment			
	Pre-	Soybean	Canola	
Total cholesterol	186±12	201±14	203±16	NS[2]
LDL cholesterol	115± 9	128±11	136±14	NS
HDL cholesterol	47± 5	45± 4	41± 3	NS
VLDL cholesterol	24± 6	28± 9	26± 6	NS
Triglyceride	121±19	132±23	144±24	NS
Phospholipid	200±10	200±10	222±10	NS

1) Mean±S.E.
2) Not significant at $p<0.05$ by Duncan's multiple range test

Table 6. Mean fasting serum lipid values for humans consuming diets incorporating soybean oil and palm oil, Study B[1]

Serum lipids (mg/dl)	Diet treatment			
	Pre-	Soybean	Palm	
Total cholesterol	184±13	181±16	194±11	NS[2]
LDL cholesterol	113±10	118±11	124±11	NS
HDL cholesterol	49± 7	47± 7	47± 6	NS
VLDL cholesterol	21± 5	22± 6	22± 5	NS
Triglyceride	105±18	122±18	124±19	NS
Phospholipid	231±11	238±12	237±11	NS

1) Mean±S.E.
2) Not significant at $p<0.05$ by Duncan's multiple range test

Table 7. Mean fasting serum lipid values for humans consuming diets incorporating canola oil and palm oil, Study C[1]

Serum lipids (mg/dl)	Diet treatment			
	Pre-	Canola	Palm	
Total cholesterol	191±18	188±20	200±16	NS[2]
LDL cholesterol	120±17	120±19	130±17	NS
HDL cholesterol	46± 4	42± 4	42± 4	NS
VLDL cholesterol	25± 4	26± 5	27± 5	NS
Triglyceride	126±13	133±15	137±18	NS
Phospholipid	237±16	235±18	246±14	NS

1) Mean±S.E.
2) Not significant at $p<0.05$ by Duncan's multiple range test

tively. Total cholesterol levels in study B during self-selected, soybean oil, and palm oil periods were 184 ±13, 181±16, and 194±11mg/dl, respectively. These in study C during self-selected, canola oil, and palm oil periods were 191±18, 188±20, and 200± 16mg/dl, respectively. Mean serum total cholesterol concentrations did not differ significantly among treatments. As with mean serum total cholesterol concentrations, highest total cholesterol occurred when palm oil diets were eaten. Serum total cholesterol concentrations were numerically lower for soybean oil than for canola oil. Although alterations in total cholesterol appear to be relatively small, according to the Lipid Research Clinic's report[8] a 1 percent fall in total serum cholesterol(approximately 2mg/dl) is associated with a 2.4 percent decrease in the risk of coronary heart disease. Many studies have reported that polyunsaturated fatty acids have a hypocholesterolemic effect in humans and animal models. Palm oil, which is very rich in palmitic acid, increased the serum cholesterol concentration more than safflower oil, but the degree of the elevation was moderate compared with that of olive oil[9].

Dietary fiber provided by the basal diet was lower in this study than is customarily used in studies of this type. In other studies conducted in this laboratory using considerably higher amounts of dietary fiber diets of canola oil produced lower serum total cholesterol and lower LDL cholesterol levels than did diets of soybean oil[10]. It may be that effects of dietary fiber on blood serum lipid levels are influenced by the degree of saturation of dietary fat[10].

Mean fasting low density lipoprotein(LDL) cholesterol concentrations in study A for pre-, soybean oil, and canola oil were 115±9, 128±11, and 136±14mg/dl, respectively. In study B, LDL levels for pre-, soybean oil, and palm oil were 113±10, 118±11, and 124±11mg/dl, respectively. In study C the LDL cholesterol concentrations for pre-, canola, and palm oil were 120±17, 120±19, and 130±17mg/dl, respectively. Mean low density lipoprotein(LDL) cholesterol concentrations did not differ significantly among treatments. As with mean serum LDL cholesterol concentrations, highest LDL cholesterol levels occurred with palm oil diets. Serum LDL cholesterol concentrations were numerically lower for soybean oil than for canola oil. This was also true with serum total cholesterol concentrations.

A considerable body of experimental evidence has shown that blood cholesterol concentration can be modulated in individuals by alterations in the fatty acid content of the fat in their diets[11)12)]. An increase in saturated fatty acids in the diet in experiments generally leads to an increase in blood total cholesterol content and LDL cholesterol. Some saturated fatty acids are more effective than others[13)14)], however, and there is a marked variation among individuals in the response observed[15)]. The effects of saturated fats on plasma cholesterol are seen only with saturated fatty acids having chain lengths of 12, 14, and 16 carbon atoms ; all increase total cholesterol mainly by increasing the concentration of LDL[16)]. This was true in this study.

The mechanisms whereby saturated fatty acids raise LDL levels are not completely understood. A strong probability, however, is that they interfere with LDL receptor-mediated clearance of LDL[17)]. The serum cholesterol-raising action of saturated fatty acids is manifest largely in the LDL fraction. These fatty acids generally do not raise triglyceride concentrations, as might be expected if they were to stimulate the synthesis of VLDL, the precursor of LDL. Isotope-kinetic studies of LDL apolipoprotein B-100(apoB) in humans indicate that the LDL-raising action of saturated fatty acids is due mainly to impaired removal of LDL from the circulation[18)]. In accord, in laboratory animals, diets high in saturated fatty acids seemingly suppress LDL-receptor-mediated clearance of LDL[19)20)]. Nonetheless, saturated fatty acids theoretically could act by a second mechanism, namely, by enhancing synthesis of apoB-containing lipoproteins. Research in humans and animals does not yield unequivocal evidence that the only mechanism for the increase in LDL levels is reduced clearance through the LDL-receptor pathway. Action of saturated fatty acids on synthesis of lipoprotein will therefore remain a possibility until excluded by definitive experiment.

One difficulty with the concept that saturated fatty acids suppress the activity of LDL receptors is that this putative action does not fit into known schemes whereby synthesis of LDL receptors is down-regulated. The major regulator of LDL-receptor synthesis is the amount of cholesterol within a cell[21)22)], or, more likely, the amount of an oxygenated sterol derived from cholesterol[23)]. Clearly, experimental evidence shows that factors that increase the cellular content of cholesterol, and hence oxysterols, suppress LDL-receptor synthesis. If dietary saturated fatty acids likewise suppress LDL-receptor synthesis, perhaps they exert their action by redistributing cholesterol among various cellular compartments to favor its inhibitory action on receptor synthesis. If this mechanism is in operation, saturated fatty acid diets might be expected to reduce the abundance of mRNA for LDL receptors, as occurs with cholesterol diets[24)]. A reduced mRNA for LDL cholesterol receptors was found with feeding of saturated fatty acids to baboons[25)] in one investigation but not in another with African green monkeys[24)]. Additional research, therefore, is needed to determine with certainty whether saturated fatty acids alter transcription of the LDL-receptor gene.

An alternate mechanism for the cholesterol-raising action of saturated fatty acids has been postulated by Loscalzo[26)] from in vitro studies with human blood cells. The activity of the LDL receptor may be lowered by saturated fat if these fatty acids are incorporated into cell membrane phospholipids, possibly by reducing binding or internalization of circulating LDL.

Monounsaturated fatty acid has been considered neutral in its influence on total cholesterol levels. All studies nonetheless indicate that oleic acid lowers

plasma cholesterol when substituted for palmitic acid [27)28)]. By convention, however, palmitic acid is designated a cholesterol-raising fatty acid, whereas oleic acid is called neutral because it affects total cholesterol levels similarly to the effect of carbohydrates. If LDL-receptor activity is the prime variant, oleic acid presumably allows for the natural expression of receptor activity, whereas palmitic acid actively reduces the activity. But Mattson[29)] and Chan[30)] reported that the experimental diets were equally effective in lowering total and LDL cholesterol and apolipoprotein B concentration in plasma. This indicates that dietary oleic acid, linoleic acid, and linolenic acid were equally hypocholesterolemic in normolipidemic men. Neither oleic acid nor linoleic acid had striking effects on lipoprotein levels of hypertriglyceridemic patients[29)]. However, the Mata[31)] study showed that a monounsaturated-rich diets resulted in a less atherogenic total cholesterol and LDL cholesterol than polyunsaturated-rich diets. There are some unexpected and inexplicable ano-malies in these studies, however, that need to be resolved before clear conclusions can be drawn.

For many years, linoleic acid was considered a cholesterol-lowering fatty acid. This concept grew out of early studies of Kinsell[32)] and Ahrens[33)] which showed that vegetable oils rich in linoleic acid lower the serum cholesterol level when substituted for dietary saturated fatty acids. Once the concept was established that linoleic acid reduces total cholesterol and LDL cholesterol concentrations, the question naturally arose as to the mechanism for this action.

A mechanism whereby polyunsaturates might lower LDL levels is by inhibition of hepatic synthesis of apoB-containing lipoproteins. Decreased synthesis of VLDL-apoB, for example, would ultimately reduce the formation of LDL apoB. High intake of polyunsaturates may decrease serum triglyceride concentrations[34)35)] and can decrease production of VLDL-apoB[36)]. This change apparently occurs in some individuals, but in many others polyunsaturates have little or no effect on VLDL levels. LDL-lowering by linoleic acid, therefore, probably cannot be due primarily to an inhibition of hepatic secretion of VLDL particles.

Other evidence suggests that substitution of linoleic acid for saturated fatty acids causes an increase in LDL-receptor activity. In accord, polyunsaturates enhance the fractional clearance rate for LDL when they are substituted for saturates in the diets of humans[37)]. Although some individuals may show a reduced conversion of VLDL to LDL on polyunsaturates[38)], this too could be the result of enhanced direct removal of VLDL remnant via VLDL receptors. Studies with laboratory animals[39)] further indicate that exchange of linoleate for saturated acids raises receptor-mediated uptake of LDL. These observations, however, do not prove that linoleic acid actively stimulates LDL-receptor activity, analogous to HMG CoA reductase inhibitors[40)]. In fact, when linoleic acid is fed in large quantities to monkeys, there is no evidence for an increase in mRNA abundance for LDL receptors[24)]. The effects of linoleic acid, therefore, could be entirely passive, which is to say, saturated fatty acids may actively suppress activity or function of LDL receptors by mechanisms yet to be determined, whereas linoleic acid is neutral. If so, linoleate would have no greater effect on LDL-receptor function than oleic acid. Ventura[41)] found that both dietary fish oil and safflower oil increased LDL receptor activity in rat livers, although fish oil produced a greater effect than safflower oil[42)]. Polyunsaturated fat may increase the secretion of bile acids and cholesterol in bile[43)] thereby causing up-regulation of hepatic LDL receptors.

One final mechanism for the observed effect of dietary fat on plasma LDL must be considered. LDL modified by oxidation is rapidly removed from the plasma by scavenger receptors. If dietary unsaturated fat leads to more oxidation products in LDL, more LDL may be scavenged, which could lower the LDL level in plasma. There is, however, no experimental evidence for such a mechanism. To the contrary, Drevon[44)] reported that LDL from subjects supplemented with fish oil or with corn oil showed similar susceptibility to copper-catalyzed lipid peroxidation in vitro.

The notion that linoleic acid has unique LDL-lowering properties was called into question by Mattson and Grundy[29)]. They compared three diets that were relatively high in palmitic acid, linoleic acid, or oleic acid. Each diet used a different fat : palm oil, high-

linoleic safflower oil, or high-oleic safflower oil. These diets were all liquid formula diets having 40% of calories as fat. In this investigation, substitution of oleic acid for palmitic acid resulted in a lowering of LDL cholesterol levels. The LDL cholesterol concentration, however, was not lower on the high-linoleate diet than on the high-oleate diet. This unexpected finding suggested that the unique cholesterol-lowering property of linoleic acid may not extend to LDL cholesterol levels. Mensink and Katan[45] also found that oleic acid and linoleic acid have essentially identical effects on LDL cholesterol levels. Two preliminary reports[46][47] suggested the same identity of LDL response to dietary oleic and linoleic acid. In addition to comparisons of linoleate with oleate, studies by Brussaard[48], Weisweiler[49], and Grundy[28] have cast doubts on claims that linoleic acid is more hypocholesterolemic than carbohydrate. Thus, linoleic acid may not uniquely lower the LDL cholesterol level as was previously thought. The significance of this in terms of CHD risk has been debated[50]. However, it appears that among all treatment oils, soybean oil(high in linoleic acid, 18 : 2 n-6) is the most capable of depressing serum total and LDL cholesterol values and elevating serum HDL cholesterol values at the same time. Reduced serum total and LDL cholesterol, along with increased HDL cholesterol, correspond to a decreased risk of CHD.

Mean serum high density lipoprotein(HDL) cholesterol levels in study A were 47±5 at pretreatment stage, 45±4 with soybean oil, and 41±3mg/dl with canola oil. Mean values in study B were 49±7mg/dl at pretreatment, 47±7, with soybean oil and 47±6mg/dl with palm oil. Mean values in study C were 46±4mg/dl at pretreatment, 42±4mg/dl with canola oil, and 42±4mg/dl with palm oil. In this fraction of total cholesterol, soybean oil diets resulted in numerically higher HDL cholesterol values than with canola oil. Palm oil produced the same HDL cholesterol concentration as soybean oil or canola oil. In populations consuming diets high in saturated fatty acids, levels of both LDL and HDL tend to be high[51]. In metabolic ward investigations, HDL cholesterol concentrations typically are the highest when the diet is rich in both total fat and saturated fatty acids[28][52]. Concerning the type of saturated acid, similar levels of HDL cholesterol are obtained whether the predominant fatty acid is palmitic acid or stearic acid[14]. A high level of HDL cholesterol in populations consuming diets high in saturates, nevertheless, does not protect individuals from CHD if LDL cholesterol concentrations are concomitantly increased[51]. This is one argument that dietary alterations of HDL levels do not modify coronary risk, whether they raise or lower HDL concentrations. Nonetheless, even in those populations having relatively high concentrations of both LDL and HDL, individuals with the lowest concentration of HDL are those at greatest risk for CHD[53].

Feeding of canola oil did not lower HDL cholesterol levels when substituted for palm oil in this study. Dietary monounsaturated fatty acids do not reduce HDL cholesterol levels when substituted for saturated fatty acids[28][29]. Since exchange of oleic acid for saturated acids reduces concentrations of LDL cholesterol, the result of this exchange is a decrease in LDL/HDL ratio. This favorable modification of the lipoprotein ratios theoretically should decrease coronary risk and low rates of CHD in populations consuming a lot of oleic acid provide support for this concept[54].

Diets of soybean oil, which is rich in linoleic acid, resulted in higher HDL cholesterol concentrations than did canola oil in this study. In contrast to this study, high intake of n-6 polyunsaturates reduce HDL cholesterol concentrations[29][55]. Some workers postulate that decreased intake of polyunsaturates will not evoke this change[48]. A review of the literature suggests that HDL cholesterol levels are reduced about 1% for every 2% of total calories in which polyunsaturated fatty acids substitute for saturated or monounsaturated fatty acids[29][55]. In large populations, however, even a small reduction in HDL cholesterol levels could produce a slight increase in CHD risk.

Mean blood serum very low density lipoprotein (VLDL) cholesterol concentrations in study A were 24±6, 28±9, and 26±6mg/dl when subjects were fed the control, soybean oil, and canola oil diets, respectively. Mean values obtained in study B were 21±5, 22±6, and 22±5mg/dl with pretrial diets,

soybean oil, and palm oil diets, respectively. Mean values obtained in study C were 25±4, 26±5, and 27±5mg/dl for self-selected diets, canola oil, and palm oil diets, respectively. Mean blood serum VLDL cholesterol concentrations did not differ significantly among treatments. Numerically lower serum VLDL cholesterol values were obtained with canola oil in comparison to the values of either soybean or palm oil. In blood, VLDL is converted to IDL, which is taken up by the liver via LDL receptors, or metabolized further to LDL, mainly by the activity of hepatic lipase. However, it is difficult to explain why canola oil produced relatively higher levels of blood serum LDL cholesterol and lower levels of blood serum VLDL cholesterol. The production of VLDL is dependent on TG synthesis in the liver. As de novo synthesis of fatty acids in human liver is normally low, VLDL production depends on the balance between influx of free fatty acids to the liver and their oxidation within the liver.

Mean serum triglyceride levels in study A were 121±19, 132±23, and 144±24mg/dl when subjects received diets containing self-selected foods, soybean oil, and canola oil, respectively. Mean values obtained in study B were 105±18, 122±18, and 124±19mg/dl when subjects received diets containing self-selected foods, soybean oil, and palm oil. Mean values obtained in study C were 126±13, 133±15, and 137±18mg/dl when subjects received diets containing self-selected foods, canola oil, and palm oil, respectively. Again, mean serum triglyceride levels did not differ significantly among treatments. As with mean triglyceride levels, the highest serum triglyceride levels occurred with palm oil diets. Serum triglyceride levels were numerically lower for soybean oil than for canola oil. Diets high in oleic acid did not increase serum TG levels in either normotriglyceridemic or hypertriglyceridemic subjects, when they are compared to saturated fatty acids[14)29)]. Linoleic acids have been reported to reduce serum TG in some patients with hypertriglyceridemia[34)35)]. When TG lowering does occur in response to dietary linoleic acid, perhaps high intake of n-6 polyunsaturates reduces hepatic synthesis of VLDL TG[34)]. Alternatively, serum TG containing polyunsaturated fatlipase than those high in saturated fatty acids ; this too could account for some TG lowering.

Mean phospholipid contents of blood in study A were 200±10, 200±10, and 222±10mg/dl with intake of self-selected, soybean oil, and canola oil diets, respectively, Mean values in study B were 231±11, 238±12, and 237±11mg/dl for self-selected, soybean oil, and palm oil diets, respectively. Mean values in study C were 237±16, 235±18, 246±14mg/dl for self-selected, canola oil, and palm oil diets, respectively. There were no significant differences in phospholipid content among soybean oil, canola oil, and palm oil diets. The relationship between phospholipids and atherosclerosis involves the transportation of cholesterol. Phospholipids aid in solubilization of lipids in the bloodstream as components of lipoproteins.

2. Fat excretion

Mean fecal fat excretions for subjects are found in Table 8. Mean fecal fat excretions for subjects consuming soybean oil and canola oil in study A were 8.97±1.98 and 9.35±1.80g/day, respectively. Mean values for subjects consuming soybean oil and palm oil in study B were 10.05±2.14 and 10.50±3.84g/day, respectively. Mean values for subjects consuming canola oil and palm oil in study C were 11.86±2.25 and 12.03±1.80g/day, respectively. Mean fecal fat excretion did not differ significantly among treatments. As with mean fecal fat excretion, highest fecal fat excretion occurred when palm oil diets were eaten. Fecal fat excretion was numerically lower for soybean oil than for canola oil. This might imply that decreases in fat absorption and/or reabsorption of cholesterol might cause a hypocholesterolemic ef-

Table 8. Mean fecal fat excretion(g/day) for humans consuming diets incorporating soybean oil, canola oil, and palm oil[1)]

Fecal fat(g/day)	Diet treatment		
	Soybean	Canola	
Study A	8.91±1.98	9.35±1.80	NS[2)]
	Soybean	Palm	
Study B	10.05±2.14	10.50±3.84	NS
	Canola	Palm	
Study C	11.86±2.25	12.03±1.80	NS

1) Mean±S.E.

fect for palm oil. However, palm oil did not produce lower serum cholesterol values than soybean oil or canola oil diets in the present study. It is unlikely that absorption would cause a cholesterol lowering effect with palm oil. However, endogenously produced cholesterol excreted in the bile is also present in the intestine of both humans and animals. Serum triglyceride values were higher on the palm oil diet, which may be reflective of greater intestinal absorption. Because only total, not fractionated, fat excretion was measured, it is difficult to obtain a clear relationship between intestinal absorption and serum lipid patterns.

In conclusion, soybean oil diets produced numerically lower blood serum total cholesterol, LDL cholesterol, and triglyceride, and numerically higher HDL cholesterol and VLDL cholesterol than did canola oil. The highest serum total cholesterol, LDL cholesterol, VLDL cholesterol, and triglyceride values were obtained when palm oil diets were fed. However, palm oil diets resulted in the same HDL cholesterol content as those values achieved by soybean oil or canola oil diets. Fecal fat excretion was numerically lower for soybean oil diets than for diets of canola oil. The highest fecal excretions were obtained when palm oil diets were consumed. In general, oils rich in polyunsaturated fatty acids produced more favorable responses in decreasing cardiovascular disease than did those containing large amounts of monounsaturated or saturated fatty acids.

Literature cited

1) National Cholesterol Education Program. *Nutr Today* 60 : 3-8, 1991
2) Choe EH, Byun YC. Recent pattern of mortality in Korea. *J Popul Associ Korea* 8(2) : 67, 1985
3) National Bureau of Statistics, Economic Planning Board, Republic of Korea. Annual report on the cause of death statistics, 1982-1990
4) Mormot MG. Life style and national and international trends in coronary heart disease mortality. *Postgrad Med J* 60 : 3-8, 1984
5) Anonymous. Production exceeds 52 million MT. *J Am Oil Chem Soc* 65 : 1232-1258, 1988
6) International Association of Seed Crushers. Vancouver Congress, June 8-10, 1988. L London : International Association of Seed Crushers, 58-59, 1988
7) Whelan E. American Council on Science and Health views on tropical oils. *Manuf Confect* 69 : 14-15, 1989
8) Lipid Research Clinics Program. The lipid research clinics coronary primary prevention trial results : I. Reduction in incidence of coronary heart disease. *JAMA* 251 : 351-364, 1984
9) Sugano M, Imaizumi K. Effect of palm oil on lipid and lipoprotein metabolism and eicosanoid production in rats. *Am J Clin Nutr* 53 : 1034-1038, 1991
10) Shayesteh MR. Omega-3 fatty acid contents of human and rat diets : physiological effectsion lipid metabolism and food acceptability. Doctors Dissertation, Univ. Nebr., *1989*
11) Keys A, Anderson J, Grande F. Serum cholesterol response to changes in the diet. *Metabolism* 14 : 747-787, 1965
12) Reiser R. Saturated fat in the diet and serum cholesterol concentration : critical examination of the literature. *Am J Clin Nutr* 26 : 524-555, 1957
13) Hegsted DM, McGandy RB, Myers ML, Stare FJ. Quantitative effects of dietary fat on serum cholesterol in man. *Am J Clin Nutr* 17 : 281-295, 1965
14) Bonanome A, Grundy SM. Effect of dietary stearic acid on plasma cholesterol and lipoprotein levels. *N Engl J Med* 318 : 1244-1248, 1988
15) Ahrens EH. Dietary fats and coronary heart disease : unfinished business. *Lancet* 1 : 1345, 1979
16) Grundy SM, Denke MA. Dietary influences on serum lipids and lipoproteins. *J Lipid Res* 31 : 1149-1172, 1990
17) Norum RR. Dietary fat and blood lipids. *Nutr Rev* 50 : 30-37, 1992
18) Shepherd J, Packard CJ, Grundy SM, Yeshurun D, Gotto AM, Taunton OD. Effects of saturated and polyunsaturated fat diets on the chemical composition and metabolism of low density lipoproteins in man. *J Lipid Res* 21 : *91-99, 1980*
19) Spady DK, Woollett LA. Interaction of dietary saturated and polyunsaturated triglycerides in regulating the processes that determine plasma low density lipoprotein receptors in the hamster. *Proc Natl Acad Sci USA* 82 : 4526-4530, 1985
20) Nicolosi RJ, Stucchi AF, Kowala MC, Hennessy LK, Hegsted DM, Schaefer EJ. Effect of dietary fat saturation and cholesterol on LDL composition and metabolism. *Arteriosclerosis* 10 : 119-128, 1990
21) Kovanen PT, Brown MS, Basu SK, Bilheimer DW, Goldstein JL. Saturation and suppression of hepatic lipoprotein receptors : a mechanism for the hypercholesterolemia of cholesterol-fed rabbits. *Proc Natl Acad Sci USA*

78 : 1396-1400, 1981

22) Russell DW, Yamanoto T, Schneider WJ, Slaughter CJ, Brown MS, Goldstein JL. CDNA cloning of the bovine low density lipoprotein receptor : feedback regulation of a receptor mRNA. *Proc Natl Acad Sci USA* 80 : 7501-7505, 1983

23) Dawson PA, Hofmann SL, Westhuyzen VD, Sudfof TC, Brown MS, Goldstein JL. Sterol-dependent repression of low density lipoprotein receptor promoter mediated by 16-bass pair sequence adjacent to binding site for transcription factor Spl. *J Biol Chem* 263 : 3372-3379, 1988

24) Sorci-Thomas M, Wilson MD, Johnson FL, Williams DL, Rudel LL. Studies on the expression of genes encoding apolipoproteins B100 and B48 and the low density lipoprotein receptor in nonhuman primates. *J Biol Chem* 264 : 9039-9045, 1989

25) Fox JC, Mcgill HC, Carey KD, Getz GS. In vivo regulation of hepatic LDL receptor mRNA in the baboon : differential effects of saturated and unsaturated fat. *J Biol Chem* 262 : 7014-7020, 1987

26) Loscalzo J, Fredman J, Rudd RM, Barsky-Vasserman, Vaughan DE. Unsaturated fatty acids enhance low density lipoprotein uptake and degradation by peripheral blood mononuclear cells. *Arteriosclerosis* 7 : 450-455, 1987

27) Baggio G, Pagnan A, Muraca M, Martini S, Opportuno A, Bonanome A. Olive oil-enriched diet : effect on serum lipoprotein levels and biliary cholesterol saturation. *Am J Clin Nutr* 47 : 960-964, 1988

28) Grundy SM, Florentin L, Nix D, Whelan MF. Comparison of monounsaturated fatty acids and carbohydrates for reducing raised levels of plasma cholesterol in man. *Am J Clin Nutr* 47 : 965-969, 1988

29) 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

30) Chan JK, Bruce VM, McDonald BE. Dietary-linolenic acid is as effective as oleic acid and linoleic acid in lowering blood cholesterol in normolipidemic in men. *Am J Clin Nutr* 53 : 1230-1234, 1991

31) Mata P, Garriso JA, Ordovas JM, Blazquez E, Alvarez-Sala LA, Rubio MJ, Alonso R. Effect of dietary monounsaturated fatty acids on plasma lipoproteins and apolipoproteins in women. *Am J Clin Nutr* 56 : 77-83, 1992

32) Kinsell LW, Michaels GD, Partridge FW, Boling LA, Ballch HE, Cochrane GC. Effect upon serum cholesterol and phospholipids of diets containing large amounts of vegetable fat. *J Clin Nutr* 1 : 224-231, 1953

33) Ahrens AH, Blomstrand R, Hirsch J, Tsaltas TT, Peterson ML. The influence of dietary fats on serum lipid levels in man. *Lancet* 1 : 943-953, 1957

34) Chait A, Onitiri A, Nicoll A, Rabaya E, Davies J, Lewis BB. Rdduction of serum triglyceride levels by polyunsaturated fat. Studies on the mode of action and on very low density lipoprotein composition. *Atherosclerosis* 20 : 347-364, 1974

35) Grundy SM. Effects of polyunsaturated fats on lipid metabolism in patients with hypertriglyceridemia. *J Clin Invest* 55 : 269-282, 1975

36) Cortese C, Levy Y, Janus ED, Turner PR, Rao N, Mlller NE, Lewis B. Modes of action of lipid lowering diets in man : consumption and dietary fatty acid consumption. *Eur J Clin Invest* 13 : 79-85, 1983

37) Connor WE, Cerqueira MT, Connor RW, Wallace RB, Malinow MR, Casdorph HR. The plasma lipids, lipoproteins and diet of the Tarahumara Indians of Mexico. *Am J Clin Nutr* 31 : 1131-1142, 1978

38) Turner JD, Le NA, Brown WV. Effect of changing dietary fat saturation on low density lipoprotein metabolism in man. *Am J Physiol* 241 : E57-63, 1981

39) Kovanen PT, Brown MS, Basu SK, Bilheimer Dw, Goldstein JL. Saturation and suppression of hepatic lipoprotein receptors : a mechanism for the hypercholesterolemia of cholesterol-fed rabbits. *Proc Natl Acad Sci USA* 78 : 1396-1400, 1981

40) Grundy SM, Vega GL. Hypertriglyceridemia : causes and relation to coronary heart disease. *Semin Thromb Hemostasis* 14 : 149-164, 1988

41) Ventura MA, Woollett LA, Spady DK. Dietary fish oil stimulates hepatic low density lipoprotein transport in the rat. *J Clin Invest* 84 : 528-537, 1989

42) Spady DK, Woollett LA. Interaction of dietary saturated and polyunsaturated triglycerides in regulating the processes that determine plasma low density lipoprotein concentrations in the rat. *J Lipid Res* 31 : 1809-1819, 1990

43) Sturdevant RAL, Pearce ML, Dayton S. Increased prevalence of cholelithiasis in men ingesting a serum cholesterol-lowering diet. *N Engl J Med* 288 : 24-27, 1973

44) Drevon CA. Marine oils and their effects. *Nutr Rev* 50 : 3845, 1992

45) Mensink RP, Katan MB. Effect of a diet enriched with monounsaturated or polyunsaturated fatty acids on levels of low density and high density lipoprotein cholesterol in healthy women and men. *N Engl J Med* 321 : 436-441, 1989

46) Berry EN, Kaufmann Y, Friedlander Y, Eisenberg S, Stein Y. The effect of dietary substitution of monounsaturated fatty acids on lipoprotein levels structure, and function in free-living population. *Circulation* 80 : 11-85, 1989

47) Valsta LM, Mutanen M, Jauhiainen M, Aro A, Katan MB.

Effects of a turnip rape oil-based MUFA diet and a sunflower oil-based PUFA diet on serum lipids and apolipoproteins in normocholesterolemic men and women. x International symposium on drugs affecting lipid metabolism. Houston, Nov. 8-11, 1989

48) Brussaard JH, Katan MB, Groot PH, Havekes LM, Hautvast JG. Serum lipoproteins of healthy persons fed a low-fat diet or a polyunsaturated fat diet for three months. A comparison of two cholesterol-lowering diet. *Atherosclerosis* 42 : 205-219, 1982

49) Weisweiler P, Janetschek P, Schuandt P. Influence of polyunsaturated fats and fat restriction on serum lipoproteins in humans. *Metabolism* 34 : 83-87, 1985

50) Nestel PJ. Polyunsaturated fatty acids(n-3, n-6). *Am J Clin Nutr* 45 : 1161-1167, 1987

51) Knuiman JT, West CE, Katan MB, Hautvast JGAJ. Total cholesterol and high density lipoprotein cholesterol levels in populations differing in fat and carbohydrate intake. *Arteriosclerosis* 7 : 612-619, 1987

52) Grundy SM. Cholesterol and coronary heart disease. *JAMA* 256 : 2849-2858, 1986

53) Kukita H, Imamura Y, Hamada M, Joh T, Kokbu T. Plasma lipids and lipoproteins in Japanese male patients with coronary artery disease and in their relatives. *Atherosclerosis* 42 : 21-29, 1982

54) Keys A, Menotti A, Karvonen MJ, Aravanis C, Blackburn H, Buzimna R, Djordjevic BS, Contas AS. The diet and 15-year death rate in the Seven Countries Study. *Am J Epidemiol* 124 : 903-915, 1986

55) Jackson RL, Kashyap ML, Barnhart RL, Allen C, Hogg E, Glueck CJ. Influence of polyunsaturated and saturated fats on plasma lipids and lipoproteins in man. *Am J Clin Nutr* 39 : 589-597, 1984