

Effects Of Dietary Saturated, Mono Unsaturated And PolyUnsaturated Fatty acids On Serum Lipids And Lipoproteins In Human Volunteers

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Citation

M Abdul Kareem, G Saayi krushna, K Lakshmi Devi. *Effects Of Dietary Saturated, Mono Unsaturated And PolyUnsaturated Fatty acids On Serum Lipids And Lipoproteins In Human Volunteers*. The Internet Journal of Nutrition and Wellness. 2008 Volume 7 Number 1.

Abstract

The current study deals with effect of different dietary oils on serum lipid profile in human volunteers. The volunteers selected in the study used to consume only one type of oil i.e., sunflower oil, ground nut oil and palm oil as their major source of dietary oil in their food preparations. Palm oil was chosen as an example of saturated fat (SFA), Ground nut oil as monounsaturated fat (MUFA) and Sunflower oil as polyunsaturated fat (PUFA). Hence the study carried out in the volunteers consuming these oils to know their effect on serum lipid profile i.e., changes in total cholesterol (TC), High density lipoprotein (HDL)-cholesterol, Low density lipoprotein (LDL)-cholesterol, Very low density lipoprotein (VLDL)-cholesterol and Triglycerides (TRI) along with changes in the atherogenic index (TC/HDL-cholesterol). The current study shows significant increase in serum TRI levels of Sunflower oil and Ground nut oil consumers. Effect of palm oil on serum cholesterol was more pronounced and the levels of HDL and LDL cholesterol are inversely proportional to one another. VLDL- cholesterol level is significantly decreased by palm oil compared to other oils used in the study. The consumption of sunflower oil and palm oil shows significant increase in TC/HDL- cholesterol levels.

INTRODUCTION

Diseases of the circulatory system account for an appreciable proportion of total morbidity and mortality in adults worldwide ¹. The Cardio vascular disease (CVD) has become a ubiquitous cause of morbidity and leading contributor to mortality in most countries. According to WHO, 16.7 million people around the world die of CVD each year. By 2020 heart disease and stroke will become the leading cause of both death and disability world wide, with number of fatalities projected to increase to more than 20 million a year and to more than 24 million a year by 2030 ². CVD is a broad, all encompassing term. Despite what its name may suggest, it is not actually a particular condition or disorder in itself. Rather CVD is a collection of diseases and conditions. CVD refers to any disorder in any of the various parts of cardiovascular system i.e., heart and blood vessels ³. The major risk factors found in several studies include hypertension, obesity, cigarette smoking, diabetes, sex/gender, age, family history, hyper cholesterolemia, life style habits, economic status, etc.

Elevated concentrations of plasma TC and LDL cholesterol

have proved to be among the major risk factors in the development of CVD ⁴. Dietary fat plays an important role in influencing blood lipid concentrations, thrombotic tendency and thus the onset of CVD ^{5,6}. Dietary saturated fat is one of the risk factors of hypercholesterolemia and CVD. Conversely, an elevated level of HDL- cholesterol is believed to confer protection. Hence, in any individual with elevated cholesterol, the primary goal is to lower the LDL- cholesterol level to reduce the risk of CVD ^{7,8}. LDL- cholesterol is recognized as the primary lipid-related risk factor and therefore the primary target for lipid-lowering therapy ^{9,10}. There are in fact several limitations of only using LDL- cholesterol as the primary risk variable ^{11,12}.

Clear relation has been evident between blood cholesterol concentration and individual risk of Coronary heart disease (CHD) ¹³. LDL- cholesterol contains the greatest amount of blood cholesterol and may be responsible for depositing cholesterol in the artery walls and these lipoproteins are atherogenic. A recent observation is HDL-cholesterol contain an enzyme, paraoxonase, which is believed to confer protection against oxidation of LDL-cholesterol in the artery

wall. The paraoxonase containing HDL-cholesterol significantly protected LDL-cholesterol from oxidation and inhibited expression of Monocyte chemotactic protein-1 (MCP-1) ¹⁴ .

An abundance of epidemiological evidence shows that low HDL-cholesterol acts as an independent risk factor for coronary heart disease ¹⁵ . Plasma LDL and HDL-cholesterol are important risk factors for IHD, in addition to indices such as total cholesterol /HDL cholesterol and LDL/HDL cholesterol ratios are considered to be powerful predictors of IHD ^{16,17} .

MATERIALS AND METHODS

The serum total cholesterol and HDL- cholesterol are estimated by using Allian ¹⁸ et al enzymatic kit. Where as LDL and VLDL- cholesterol were calculated by the formula of Friedewald ¹⁹ et al and Serum triglycerides were estimated by Qualigens ²⁰ diagnostic kit.

STUDY DESIGN

Subjects studied were 24 male and 24 female healthy volunteers of the age group between 35 to 45 years for each type of oil. The volunteers who consumed their diet using only one type of oil in their food preparations for more than two years and without any clinical complications such as diabetes, hypertension and smoking were chosen for the study. Participants in this study were selected from different locations in Anantapur town, Anantapur District, Andhra Pradesh, India. Before the beginning of the study the nutritional status of the subjects was studied, this work showed no worth mentioned difference in food intake of individuals.

SAMPLE COLLECTION

Fasting venous blood samples (5 ml) were collected and allowed to clot. The serum of the sample was collected by centrifugation (3000 g) and used for determination of lipid profile on the same day.

STATISTICAL ANALYSIS

The values obtained were analyzed by using DMR test. In addition values compared with control normal values given by W.H.O. Results presented in tables based on mean \pm SD, where * $P < 0.05$ was regarded statically significant.

RESULTS

Lipids being insoluble in water need a transport system made up of lipoproteins, such as chylomicrons, VLDL, LDL

and HDL ²¹ . Estimation of these lipoproteins is used as an index to measure the levels of lipids present in the serum.

In the current study (table-1) it is observed that the consumption of each oil is having its own effect on serum lipid profile. The results indicate that slight reduction in the triglyceride levels of volunteers consuming sunflower oil and ground nut oil by 0.5% and 3.8% respectively and are not significant. However, palm oil consumption showed more reduction in the triglyceride levels i.e. 22.10%, which is more significant. Compared to palm oil, consumption of sunflower oil and ground nut oil enhanced VLDL and triglyceride secretion. Among the oils used in the current study, effect of palm oil on serum cholesterol was more pronounced (0.49%). Consumption of sunflower oil and ground nut oil showed a moderate reduction, compared to normal value, i.e. 8.88% and 11.24% respectively. It is interesting to observe (table – 1) that, the consumption of sunflower oil, ground nut oil and palm oil showed a significant reduction in HDL cholesterol levels by 33.02 %, 36% and 45.9 %. The order of decrease in LDL cholesterol levels of palm oil, sunflower oil, and ground nut oil was 45.48%, 83.63% and 107.19% respectively. It is noticed that palm oil consumption enhances the LDL cholesterol levels, when compared with other oils, due to it is high SFA content.

It is evident (table – 1) that there is a pronounced influence on the levels of VLDL by sunflower oil consumption. The order of decrease of VLDL levels was 1.5% and 19.04% for ground nut oil and palm oil respectively. Compared to normal value slightly higher VLDL levels (2.23 %) are observed in the volunteers consuming sunflower oil. Data presented in table – 2 shows the changes in the ratio of TC/HDL - cholesterol of the volunteers consumed different oils. Data reveals an important antiatherogenic index regarding the consumption of ground nut oil, which shows a slight increase (0.24%) in TC/HDL - cholesterol level, where as the consumption of palm oil and sunflower oil shows significant increase (18.36% and 20.63%) compared to normal value.

DISCUSSION

Lee et al reported increase of triglycerides in rats with increasing PUFA/SFA ratio ²² . The observation of the current study was in agreement with that of Lee et al., ²² . The increase in the triglyceride levels is due to the decreased activity of LPL enzyme, and it leads to hypertriglyceridemia ²³ . If a person showing increase in the fasting triglyceride

levels, then he / she must be exposed to the increased hypertriglyceridemia during postprandial stages. In the present study blood samples were collected from fasting individuals. Prolonged exposure to triglycerides may increase hypertriglyceridemia and increases the circulating FFAs which may cause endothelial dysfunction²⁴. There is evidence that hypertriglyceridemia induced endothelial cell dysfunction plays a critical role in the pathology of atherosclerosis²⁵. Zilversmith²⁶ proposed that the simultaneous release of fatty acids during LPL mediated triglyceride hydrolysis may cause endothelial cell injury and initiate thrombotic events. Several studies supporting fatty acid mediated endothelial activation and dysfunction as a consequence of hypertriglyceridemia²⁷.

In the current study it is observed that sunflower oil (Which contains more amount of linoleic acid) consumers show an increase in the triglyceride levels, which is in agreement with several studies^{26,27}. Hypertriglyceridemia also increases the expression of endothelial receptor molecules specific for monocyte and leucocyte adhesion and stimulate adhesion of leukocyte and monocyte, especially the later to the endothelial surface²⁸. Benhard reported that postprandial hypertriglyceridemia and the simultaneous release of free fatty acids during lipoprotein lipase mediated triglyceride hydrolysis in the proximity of the endothelium can cause endothelial cell injury²⁹. This may be sufficient to cause endothelial barrier dysfunction and allows increased uptake of cholesterol-rich lipoprotein remnants into vascular tissues and thus accelerate the pathology of atherosclerosis. Aerobic exercise and weight reduction can decrease postprandial hypertriglyceridemia and thus atherogenic remnant lipoproteins. This decreases endothelial cell activation by free fatty acids and lipoprotein remnants²⁹. The cholesterol levels in the current study were significantly changed in the volunteers consumed different oils. Several studies conformed that high blood cholesterol level in young adults is a predict of CVD in later life³⁰. In general the elevated blood cholesterol level is a major risk factor for CVD. Increase in the cholesterol level was noticed in the volunteers consumed palm oil as their major source of dietary oil. Compared to the volunteers of the study a significant decrease in cholesterol levels were observed in the volunteers consuming ground nut oil and sunflower oil as their dietary source.

The results of cholesterol in this study were in agreement with that of Lu et al.,³¹ and Williams et al.,³²

Hypocholesterolemic effect of PUFA may be due to its high PUFA/SFA ratio. In sunflower oil consumers decreased levels of cholesterol and increased levels of Triglycerides were observed. The hypocholesterolemic effect of sunflower oil may be due to its high PUFA content, which acts as an inhibitor of hepatic HMG – CoA reductase, the rate limiting enzyme in cholesterol biosynthesis³³. But the increase in the cholesterol level may lead to several complications which may progress the development of CVD. By comparing the data it is clearly evident that consumption of palm oil decreases the HDL cholesterol levels, than other oils chosen in the study. It is well known that HDL cholesterol plays a vital role in reducing CVD by acting as antiatherogenic. It carries cholesterol moieties from the peripheral organs to liver³⁴ and decreases LDL oxidation as it contains peroxanase. Epidemiological cohort studies have convincingly associated with low HDL- cholesterol and increased cardiovascular risk. This strongly suggests that interventions to increase HDL-cholesterol will yield clinically significant outcome benefits³⁵. The Framingham study showed that decreased levels of HDL-cholesterol were significantly and independently associated with an increased risk of coronary death³⁶. Cohort studies have strengthened the association between low HDL-cholesterol and adverse coronary and cerebrovascular outcomes³⁷. It is noticed that palm oil consumption enhances the LDL cholesterol levels, when compared with other oils, due to it is high SFA content. Data obtained from several dietary and nutritional epidemiological studies reveals that increased LDL - cholesterol levels play an important role in the development of CVD³⁸. LDL- cholestterol can be considered as independent risk factor, due to its high probability to undergo oxidation and to be up taken by macrophage in the maturation of foam cells³⁹. The more the availability of LDL, the more oxidation may occur which may enhance the maturation of foam cells and leads to progression of CVD⁴⁰. LDL can be oxidized in the sub endothelial space which lacks many of the antioxidants present in whole blood; it is possible that LDL is oxidized by endothelial cells while passing through the endothelium⁴¹. In the current study palm oil shows a significant increase in the LDL levels compared to other oils. The data of LDL and HDL- cholesterol of the current study came in agreement with the studies of Abdullah and Rawashdeh⁴² and Clifton and Noakes⁴³.

Markedly increased concentration of triglycerides followed by hike in VLDL levels with no significant changes in

cholesterol and HDL cholesterol suggest some Cardiovascular risk in sunflower oil and ground nut oil volunteers, when compared with palm oil volunteers. According to Heimberg and Wilcox PUFA and MUFA may enhance VLDL and triglyceride secretion compared with SFA⁴⁴. VLDL acts as atherogenic because it carries cholesterol from liver to other peripheral tissues and also it possesses Apo-B particle, which is membrane protein and act in the accumulation and progression of foamy lesions in the damaged endothelial cells⁴⁵.

There is good evidence that the ratio of TC/HDL - Cholesterol is a better indicator of CVD risks than either total cholesterol or LDL Cholesterol alone^{46,47,48}. Hence the current study reveals that the consumption of ground nut oil can be considered as antiatherogenic in this respect.

Figure 1

Table: 1 Changes in serum lipid profile Results based on mean \pm SD values

Control	Groundnut oil	Sunflower oil	Palm oil	Ghee
4	4.01 \pm 0.26 a	5.04 \pm 0.30 b	4.9 \pm 0.25 B	3.62 \pm 0.097 b

Each value is the mean \pm SD of volunteers and as the means of individual percentage changes \pm SD relative to control values. According to DMR Test * P<0.05.

Figure 2

Table: 2 Atherogenic index Results based on mean \pm SD

Parameter (mg/dl)	Control	Ground nut oil	Sunflower oil	Palm oil	Ghee
Triglyceride	40 - 180	173.4 \pm 1.31 a	179.1 \pm 1.90 A	147.3 \pm 2.88 c	161.4 \pm 2.98 b
Cholesterol	200	170.8 \pm 2.45 c	174.5 \pm 2.49 B	201.16 \pm 2.63 a	184.9 \pm 3.22 b
HDL cholesterol	45 - 60	44.4 \pm 2.914 b	35.18 \pm 2.9 C	41.1 \pm 2.23 b	50.7 \pm 1.09 a
LDL cholesterol	130-160	91.7 \pm 3.23 d	103.3 \pm 3.59 C	130.6 \pm 3.87 b	101.9 \pm 3.29 c
VLDL	25-35	34.6 \pm 0.26 a	35.8 \pm 0.38 A	29.4 \pm 0.57 b	32.28 \pm 0.59 a

Each value is the mean \pm SD of volunteers and as the means of individual percentage changes \pm SD relative to control values. According to DMR Test [[[*]]] P<0.05.

References

1. Jackson, KG., and Julie AL., 2002. Functional foods, blood lipids and coronary heart disease. Food Sci and Technology Bulletin, 1: 1-11.
2. Atlas of Heart Disease and Stroke, WHO, September, 2004.
3. Anonymous, 2004. Mini atlas cardiology. Jaypee Pharma Customised Imprints (P) Ltd, 1st Indian edition.

4. Stamler J., Wentworth D, Neaton.J. 1986. Is the relationship between serum cholesterol and risk of death from coronary heart disease continuous and graded? J. Am. Med. Assoc. 256 : 2823-2828.
5. Keys A., Menotti A., Karvonen M.J. 1986. The diet and 15 year death rate in the seven countries study. Am. J. Epidemiol. 124: 903-915.
6. Hetzel B.S., Charnock J.S., Dwyer T., McLennan, P.I., 1989. Fall in the coronary heart disease mortality in USA and Australia due to sudden death: evidence for role of polyunsaturated fat. J Clin Epidemiol. 42:855-893.
7. Wilson, PW., Abbott, RD., Castelli, WP., 1988. High density lipoprotein cholesterol and mortality. The Framingham heart Study. Arteriosclerosis. 8:737-41
8. Harper's Biochemistry, 2000. 25th edition Murry, RK., Graner, DK., Mayes, PA., Rodwell, VW., (eds) Appleton and Lange, Stamford, Connecticut.
9. Grundy, SM., 2002. Low-density lipoprotein, non-high-density lipoprotein, and apolipoprotein B as targets of lipid-lowering therapy. Circulation, 106: 2526-29.
10. Genest, J., Frohlich, J., Fodor, G., McPherson, R., 2003. Working group on hypercholesterolemia and other dyslipidemias. Recommendations for the management of dyslipidemia and the prevention of cardiovascular disease: summary of the 2003 update. Can Med Assoc J., 169: 921-24.
11. Schanagl, H., Nauck, M., Wieland, H., Marz, W., 2001. The Friedewald formula underestimates LDL cholesterol at low concentrations. Clin Chem lab med, 39: 426-31
12. Walldius, G., Jungner, I., 2004. Apolipoprotein B and apolipoprotein A-I: risk indicators of coronary heart disease and target for lipid-modifying therapy. J Intern Med, 255: 188-05.
13. Cottrell, RC., 1991. Introduction: nutritional aspects of palm oil. Am J Clin Nutr, 53: 989-1009.
14. Mackness, B., Hine, D., Liu, Y., et al, 2004. Paraonase-1 inhibits oxidized LDL-induced MCP-1 production by endothelial cells. Biochem Biophys Res Commun, 318: 680-83.
15. Stampfer, MJ., Sacks, FM., Salvini, S., Willett, WC., Hennekens, CH., 1991. A prospective study of cholesterol, apolipoproteins, and the risk of myocardial infarction. N Engl J Med. 325:373-81.
16. Avogaro, P., Bittolo Bon, G., Cazzolato, G., Quinci, GB., 1979. Are lipoproteins better discriminators than lipid for atherosclerosis? Lancet. 1:910-13
17. Ishakawa, T., Fidge, N., Thelle, DS., Forde, OH., Miller, NE., 1978. The Thombo heart study: serum apolipoproteins A-I concentration in relation to future coronary heart disease. Eur J Clin Invest. 8:179-82.
18. Allian, CC., Poon, LS., Chan, CSG., Richmand, W., Fu, P., 1974. Enzymatic determination of total serum cholesterol. Clin Chem 20: 470-475.
19. Friedwold, WT., Levy, RI., Fredrickson, DS., 1972. Estimation of the concentration of low-density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. Clin Chem 18: 499-502.
20. Fossati, P., Principe, L., 1982. Serum triglycerides determined colorimetrically with an enzyme that produces hydrogen peroxide. Clin Chem 28: 2077-2080.
21. Harper's Biochemistry, 2000. 25th edition Murry, RK., Graner, DK., Mayes, PA., Rodwell, VW., (eds) Appleton and Lange, Stamford, Connecticut.
22. Lee, JH., Fukumoto, M., Niahida, H., Ikida, R., Sugano, M., 1989. The interrelated effects of n6/n3 and polyunsaturated/saturated ratios of dietary fats on the

- regulation of lipid metabolism in rats. *J Nutr*, 119: 1893-99.
23. Picard, F., Kapur, S., Perreault, M., Marette, A., Deshaies, Y., 2001. Nitric oxide mediates endotoxin-induced hypertriglyceridemia through its action on skeletal muscle lipoprotein lipase. *The FASEB J*, 15: 1828-30.
24. Chon, J.S., 1998. Postprandial lipemia: emerging evidence for atherogenicity of remnant lipoproteins. *Can J Cardiol* 14(supl B):18B-27B.
25. Boquist, S., Ruotolo, G., Tang, A., 1999. Alimentary lipemia, postprandial triglyceride rich lipoproteins, and common carotid intima-mediated thickness in healthy, middle-aged men. *Circulation* 100:723-728.
26. Zilversmit, D.B., 1973. A proposal linking atherogenesis to the interaction of endothelial lipoprotein lipase with triglyceride-rich lipoproteins. *Circ Res*, 33: 633-38.
27. Henning, B., Toborek, M., McClain, C.J., Diana, J.N., 1996. Nutritional implications in vascular endothelial cell metabolism. *J Am Coll Nutr*, 15: 345-5
28. de Grijter, M., Hoogebrugge, N., van Rijn, M.A., Koster, J.F., Sluiter, W., Jongkind, J.A., 1991. Patients with combined hypercholesterolemia-hypertriglyceridemia show an increased monocyte-endothelial cell adhesion in vitro: triglyceride level as a major determinant. *Metabolism*, 40: 1119-21.
29. Benhard, H., Toborek, M., Craig, J., McClain, 2001. High-energy diets, fatty acids and endothelial cell function: Implications for atherosclerosis. *J Amer Coll Nutr*, 20 (2): 97-105.
30. Myers, L., Coughlin, S.S., Webber, L.S., Srinivasan, S.R., Berenson, G.S., 1995. Prediction of adult cardiovascular multifactorial risk status from childhood factor levels. The Bogalusa heart study. *Am J Epidemiol* 142:918-24.
31. Lu, Z., Hendrich, S., Shen, N., White, P.J., Cook, L.R., 1997. Low linolenate and commercial soybean oils diminish serum HDL cholesterol in young free living adult females. *J Am College Nutr*, 16: 562-9.
32. Williams, P.J., Blanche, P., Cavanagh, A., Holl, L., Austin, M., 1993. Lipoprotein subclasses in genetic studies: the Berkeley data set. *Gen Epidemiol* 10:523-28.
33. Yaqoob, S., and Peterson, J., 1995. An interpretation on: Effect of PUFA on atherosclerosis. *Am J Med*, 24:246-51.
34. Philip, B., 2005. The role of HDL-cholesterol in preventing atherosclerotic disease. *Europ heart J Suppl*, 7: F4-F8.
35. James, S., 2005. Raising HDL-cholesterol and lowering CHD risk: does intervention work? *Europ heart J Suppl*, 7: F15-F22.
36. Gordon, T., Castelli, W.P., Hjortland, M.C. et al., 1977. High density lipoprotein as a protective factor against coronary heart disease. The Framingham study. *Am J Med*, 62: 707-14.
37. Tanne, D., Yaari, S., Goldbourt, U., 1997. High-density lipoprotein cholesterol and risk of ischemic stroke mortality. A 21-year follow-up of 8000 men. *Arterioscler Thromb Vasc Biol*, 17: 107-113.
38. Keys, A., 1980. Seven countries: a multivariate analysis of death and coronary heart disease. Harvard University press, Cambridge Massachusetts.
39. Marshall, 1998. Text book clinical Biochemistry with clinical correlations. 2:430-32.
40. Witztum, J.L., 1993. Role of oxidized low density lipoprotein in atherogenesis. *Br Heart J* 69:s12-s18.
41. Steinberg, D., Parthasarathy, S., Carew, T.E., Khoo, J.C., Witztum, J.L., 1989. Beyond cholesterol: modification of low density lipoprotein that increase its atherogenicity. *N Engl J Med*, 320: 915-924.
42. Abdullah, Y., Rawashdeh, A., 2003. Comparison between the effects saturated and unsaturated oils on serum cholesterol of Jordanian volunteers. *Pakistan J Biol Sci*, 6 (6): 580-86.
43. Clifton, P.M., Noakes, M., Nestel, P.J., 1998. LDL particle size and LDL and HDL cholesterol changes with dietary fat and cholesterol in healthy subjects. *The J Lipid Res*, 39: 1799-1804.
44. Heimberg, M., Wilcox, H.G., 1972. The effect of palmitic and oleic acids on the properties and composition of the very low density lipoprotein secreted by the liver. *J Biol Chem*, 247: 875-80.
45. Philip, B., 2005. The role of HDL-cholesterol in preventing atherosclerotic disease. *Europ heart J Suppl*, 7: F4-F8.
46. Kinoshita, B., Glick, H., Garland, G., 1994. Cholesterol and coronary heart disease: predicting risks by levels and ratios. *Ann Intern Med*, 121:641-7.
47. Asmann, G. and Schutle, H., 1992. Relation of high density lipoprotein cholesterol and triglycerides to incidence of atherosclerotic coronary artery disease (the PROCAM). *Am J Cardiol* 70:733-37.
48. Manninen, V., Tenkanen, L., Koshinen, P., Huttunen, J.K., Manttari, M., Heinonen, O.P., Frick, M.H., 1992. Joint effect of serum triglyceride and LDL and HDL cholesterol concentrations on coronary heart disease risk in the Helsinki Heart study: implications for treatment. *Circulation* 85:37-45.

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