ORIGINAL PROF-605

SERUM LIPID PROFILE;
THE EFFECTS OF DIETARY PUFA AND MUFA AN EXPERIMENTAL ANIMAL STUDY

DR. MUHAMMAD AKRAM SHAD
Associate Professor of Pathology
Nishtar Medical College Multan

DR. MUHAMMAD TAYYAB, M.PHIL
Professor of Pathology
PGMI Lahore

DR. TEHSEEN IQBAL, M.PHIL
Assistant Professor of Physiology
Nishtar Medical College Multan

ABSTRACT

Atherosclerotic vascular diseases are the biggest threat to the life and health of adult people. Hyperlipidemia, especially hypercholesterolemia is the strongest risk factor for atherosclerosis. Different types of dietary lipids have been shown to affect the serum lipid profile differently. So dietary modification is a useful measure to treat hyperlipidemia. In this study, effects of PUFA fat (corn oil) and MUFA fat (Olive oil) on serum lipid profile were compared. We used albino rats as experimental animals. Thirty six albino rats of age 8 weeks were divided into three groups of 12 each. They were fed diets containing different types of fat for 12 weeks. Serum lipid profile at 0 week and 12 weeks were compared. It was seen that both PUFA diet and MUFA diet decreased all the serum lipid fractions; PUFA diet was rather more potent in this regard. The only difference was in HDL-C fraction. HDL-C was significantly decreased in case of PUFA diet while it was significantly increased in case of MUFA diet. So MUFA diet is more advantageous for patients of atherosclerotic vascular diseases.

INTRODUCTION

Atherosclerotic vascular diseases are the biggest threat to the life and health of adult people. Among them myocardial infarction and strokes, are the most dreadful conditions. These account for at least 15 million deaths a year, many millions more are disabled by them[1]. Unfortunately their incidence is increasing in developing countries like Pakistan[2].

Hyperlipidemia, especially hypercholesterolemia is the strongest risk factor for atherosclerosis[3]. More than half of the body's cholesterol arises by synthesis and virtually all nucleated cells of the body are capable of synthesizing it[4]. Although less than half of the body's cholesterol is provided by the diet, dietary changes seem to affect its level and as suggested by WHO, drug therapy for elevated blood cholesterol should only be considered after serious attempts for modifying diet have been attempted[5].

Different types of dietary lipids have been shown to affect lipid metabolism and hence serum lipid profile, differently[6]. Dietary lipids are composed of fatty acids and dietary cholesterol. Fatty acids may be saturated, polyunsaturated or monounsaturated. Saturated fats are solid at room temperature and are derived from animal foods.

Unsaturated fats are liquid at room temperature and in

general are derived from plant foods, as vegetable oils. All vegetable oils contain either polyunsaturated fatty acids (PUFA) or monounsaturated fatty acids (MUFA). Example of PUFA fat is corn oil and MUFA fat is olive oil[7].

The two main lipids in blood are cholesterol and triacylglycerol. High levels of blood cholesterol accelerate atherogenesis. It also increases blood viscosity and makes the plaque vulnerable to rupture and thrombosis- the triggering event for myocardial infarction[8,9].

Lowering the high blood cholesterol reduces the incidence of coronary heart disease[10,11,12]. High density lipoprotein cholesterol (HDL-C) is a safety factor while low density lipoprotein cholesterol (LDL-C) is a risk factor for coronary heart disease[13,14]. Low fat diet and changes in dietary fats can reduce the serum cholesterol.

Low fat diet resulted in significant reductions in HDL-C[15,16]. Which is also harmful. PUFA fat and MUFA fat both can reduce serum cholesterol but their comparative studies are scarce. So we designed this study to compare the effects of PUFA diet and MUFA diet on the serum lipid profile using Albino rats in our experimental animal study.

MATERIAL & METHODS

Thirty six albino rats of 8 weeks age were selected. They were divided into three groups, each group having equal number of male and female rats. Animals were kept as pairs of same sex in iron cages. Three types of synthetic diets were used as shown in table 1. Each group was fed only one type of diet throughout the 12 weeks of the study period.

Group A (n=12) Control diet i.e. 5% fat.
Group B (n=12) MUFA diet i.e. diet containing 20% fat as olive oil.

Group C (n=12) PUFA diet i.e. diet containing 20% fat as corn oil.

The animals were kept under optimum atmospheric and hygienic conditions with food and water available at libitum. They were fed the control diet for one week prior to the start of the experimental period to make the animals acclimatized to the environment and to bring the lipid profile at the baseline.

After one week animals were weighed and blood samples were taken (0-week sample) for the estimation of serum lipid profile. Blood samples were collected by heart puncture after anaesthetizing the animal.

After 12 weeks another sample of blood was taken to find out the change in lipid profile of the animal. Total cholesterol HDL-C, LDL-C and triacylglycerol were measured enzymatically (Brox et al 1981(17). VLDL-C by nephelometry and total lipids were estimated by sulphovanillin reaction (Carroll 1962)18. Statistical significance of the comparisons was estimated by utilizing the students 't' test.

RESULTS

Table-I. Percentage Composition of various diets

Ingredients	Control diet	MUFA Diet (Olive oil)	PUFA Diet (Corn oil)
Fat	5%	20%	20%
Maize Starch	60%	45%	45%
Caseom	20%	20%	20%
Cane Sugar	5%	5%	5%
Choline & Methionine	0.5%	0.5%	0.5%
Mineral mixture	3.5%	3.5%	3.5%
Vitamin mixture	1%	1%	1%
Total	100	100	100

Table-II. Comparison of serum lipid profile in various groups at 0 and 12 weeks (values expressed as mean ±SD in mg/dl)

Parameter	Group A control diet		Group B MUFA diet		Group C PUFA diet	
	0 Wk	12 Wk	0 Wk	12 Wks	0 Wk	12 Wks
Total lipids	469±13.2	+478±13.4	**469±11.8	437.9±10.5	469.1±13.2	**428.7±14.4
Total cholesterol	87±6.7	+82.4±7	**89.6±8.1	79.2±8	89.2±6.6	**72.3±6
Triacylglycerol	94.6±10.8	+101.7±9.1	*93.7±7.6	84.2±6.7	94.3±7.7	**79.9±6
HDL-C	31.9±2.9	+30.1±2.4	*33.6±3.3	36.5±3.6	34.8±2.3	*31.2±2.5
LDL-C	34.7±6.1	+30.7±6.4	**35.6±7.3	27±6.8	36.6±5.1	**25±3.7
VDL-C	123.6±12.6	+129.3±9.4	**123.6±13	106.1±9.4	122±9.9	**100.7±7.7

+P>0.05 Non significant, *(P<0.05) significant, ** (P<0.001) Highly significant

Table-III. Percentage decrease(↓) or increase (↑) in lipid fractions in group B (MUFA diet) and group C (PUFA diet) at 12 weeks

Parameters	Group B (MUFA diet)		Group C (PUFA diet)	
Total cholesterol	↓	11.6%	↓	19%
Total lipids	↓	6.6%	↓	8.6%
TAG	↓	10%	↓	15.2%
LDL-C	↓	24%	↓	31.7%
VLDL-C	↓	14%	↓	17.4%
HDL-C	↑	8.6%	↓	10.3%

DISCUSSION

Decreasing serum cholesterol level, especially the LDL-C , is the corner stone of prevention strategy for atherosclerotic vascular diseases. At the same time, elevating serum level of HDL-C is also desirable[19,20,21,22] . Cholesterol reduction can be achieved by modifying diet, partial ileal bypass surgery, or by using pharmacological agents[23]. For the general population, non-drug, non surgical approach is most desirable[24,25]. Dietary modifications require low fat intake with change in the dietary fat. The present study reveals that both PUFA diet as well as MUFA diet can decrease the total cholesterol and other lipid fractions. Keys 1957[26], Matson and Grundy 1985[27] and Wardlaw and Snook 1990[28] demonstrated the role of PUFA diet in lowering serum lipid profile. We also found a decrease of 19% in the level of total cholesterol in the PUFA diet group. Our findings are comparable with the findings of Chong et al 1987[29], Mensik and Katen 1989[30] and Wardlaw and Snook 1990[28] who reported a decrease of 36%, 10%, and 14% respectively in the level of total cholesterol by using PUFA diet. VLDL-C and triacylglycerol lowering effect of PUFA diet was also observed by Shepherd et al1978[31]. This is due to the depressed VLDL-synthesis by the liver.

We observed a reduction of 10.3% in the level of HDL-C in PUFA diet group. This finding is in agreement with the findings of Shepherd et al (1980)[32] and Carg et al 1988[33] .This is linked to the decreased hepatic biosynthesis of HDL-C apoprotein i.e, Apo-A.

In our study, MUFA diet decreases the level of total cholesterol and LDL-C. Shepherd et al[11], Mattson[12] and Grundy[24] showed similar findings. This effect of MUFA diet is due to the increased activity of LDL-C receptors. In our study, MUFA diet also increased the level of HDL-C. This is in agreement with the findings of Garg et al[33] and Jacottot et al 1988[34]. This effect may either be due to the enhanced biosynthesis of Apo-A or due to its reduced catabolism. In our study both PUFA diet and MUFA diet produced reduction in various fractious of serum lipids except HDL-C levels which are decreased in PUFA diet and increased in case of MUFA diet. As compared with MUFA diet, PUFA diet is more effective in reducing the total cholesterol and LDL-C. MUFA diet has advantage over PUFA diet in its HDL-C raising

capacity. So in atherosclerotic vascular diseases, MUFA diet may be more beneficial and it is said that its protective effects may last up to four years[35].

REFERENCES

1. WHO. Measuring Health In. The World Health Report 1998. Life in the 21st century. A vision for all. WHO Geneva 39-112; 1988.

2. Bhopal R, Unwin N, White M, Walker L, Alberti KGMM et al. Heterogeneity of coronary heart disease risk factors in India. Pakistani, Bangladeshi and European origin populations. Cross-sectional study. Br. Med. J, 319: 215-220; 1999.

3. Chandrasoma P and Taylor CR. The Heart III. Myocardium and pericardium. In concise Pathology. 3rd ed, Appleton and Lange: pp 360-367; 1998.

4. Murray RK, Granner DK, Mayes PA and Rodwell VW. Cholesterol synthesis, transport and excretion. In Harper's Biochemistry, 24th ed, Appleton and Lange, 271-283; 1996.

5. WHO. The double burden. In The world Health Report 1999. Making a difference. WHO, Geneva 13-22, 85-110; 1999.

6. Grundy SM. Comparison of monolunsaturated fatty acids and carbohydrates for lowering plasma cholesterol N Engl J Med 314: 745-748; 1986.

7. American Dietetic Association; http://www.eatright.org.

8. Sloop G.D. A critical analysis of the role of cholesterol in atherogenesis. Atherosclerosis 142: 265-272; 1999.

9. Castelli WP. The new pathophysiology of coronary artery disease. AM J Cardiol 82(10B): 60T-64T; 1998.

10. Monsen ER. New Dietary Reference Intakes proposed to replace the recommended dietary allowances. J Am. Diet Assoc 96: 754-759; 1996.

11. Shepherd J et al. Prevention of coronary heart disease with pravastatin in men with hypercholesterolemia-West of Scotland Coronary Pravastatin Study Group. N Eng. J. Med 333: 1301-1308; 1995.

12. Scandinavian Simvastatin Survival Study Group. Randomized trial of cholesterol lowering in 4444 patients with coronary heart disease, the Scandinavian, Simvastatin Survival study(4S) Lancet 344: 1383; 1994.

13. Carlson LA and Bottiger LE. Islamic heart disease in relation to fasting values of plasma triacylglycerides and cholesterol. Lancet; 1: 865-868.

14. Castelli WP, Doyle JT, Gordon T et al. Cholesterol and other dietary lipids in coronary heart disease. Circulation 55: 767-772; 1977.

15. Knopp RH et al. Long term cholesterol lowering effects of 4 fat restricted diets in hyper-cholesterolemic and combined heper-lipidemic men. The Dietary Alternatives Study. JPMA; 278: 1509-1515; 1997.

16. Nishimura PM, Lowe S, Verstuyft J, Naggert JK, Kuypers FA, Paigen B. Effects of dietary fats from animal and plant sources on diet induced fatty streak lesions in C57bl/6J mice. J. Lipid Res, 34: 1413-1422; 1993.

17. Brox JH, Kille JE, Gunnes S, Norday A. The effect of Cod liver oil and corn oil on platelets and vessel wall in man. Thromb Ha Emost 46: 604-611; 1981.

18. Carroll KK. Studies on the mechanism by which Erucic and acid affects cholesterol metabolism. Canad J Biochem & Physiology 40: 1115-1121; 1962.

19. Kennel WB. Overview of atherosclerosis. Clinical Therapeutics 20 (Suppl B): B2-B17; 1998.

20. Bhakdi S. Pathogenesis of atherosclerosis, an alternative hypothesis. Herz 23(3): 163-167 [Abstract]; 1998.

21. Beil FU, Windler E. Goals and procedure of lipid intervention in coronary heart disease Herz 22(3): 134-140 (Abstract); 1997.

22. Maher V, Sinfuego J, Chao P, Parekh J. Primary prevention of coronary heart disease. What has WOSCOPS told us and what questions remain? Drugs 54(1): 1-8; 1997.

23. Slark EM. Review of the major intervention trials of lowering coronary artery disease risk through cholesterol reduction. AM J cardiol 78(Suppl 6a):

13-19; 1997.

24. Grundy SM. Primary prevention of coronary heart disease. Role of cholesterol control in the United States. J Int Med 241(4): 295-306; 1997.

25. Maccaron DA, Oparil S, Chait A, Haynes RB, Krisetherton P, Sterm JS et al. Nutritional management of cardiovascular risk factors. A Randomized clinical trial. Arch Int Med 157(2): 169-177; 1997.

26. Keys A. Diet and the epidemiology of CHD. JAMA 164: 1912; 1957.

27. Mattson FH and Grundy SM> Comparison fo effects of dietary saturated, mono-unsaturated and poly-unsaturated fatty acids on plasma lipids and lipoproteins J lipid Res 26: 194-202; 1985.

28. Wardlaw GM and Snook JT. Effect of diets higher in butter, corn oil or high oleic acids, sunflower oil on serum lipids and lipo-protein in men. Am J Clin Nutr 51: 815-821; 1990.

29. Chong KS, Nicolsi RJ, Rodger RF et al, Effect of dietary fat saturation on plasma lipo-proteins and HDL metabolism of the rhesus monkey. J. Clin Invest 79: 675-683; 1987.

30. Mensik RP and Katen MB. Effecxt of fatty acids on cholesterol levels. N Engl J Med 321: 436-441; 1989.

31. Shepherd J, Packard CJ, Patsch JR et al. Effects of dietary polyunsaturated and saturated fat on properties of HDL. J Clin Invest 60: 1582-1592; 1978.

32. Shepherd J, Packard CJ, Grundy SM et al. Effect of saturated and polyunsaturated fat diet on the chemical composition and metabolism of LDL in man. J. lipid Res. 21:90-92; 1980.

33. Garg A, Bonamone A, Grundy SM et al. Comparison of high carbohydrate diet with a monounsaturated fat diet in patients with non insulin dependent diabetes mellitus. N. ngl J Med 319: 829-834; 1988.

34. Jacottot B, Baudet MF, Lasserre M et al. Olive oil and the lipoprotein metabolism. Rev Fr Corps Gras 35: 51-55; 1988.

35. Lorgeril M et al. Mediterranean diet, traditional risk factors and the rate of cardiovascular complications after myocardial infarction: Final report of the Lyon Diet Heart Study. Circulation 99: 779-815 (Abstract); 1999.

Believe in yourself;
You can do the best

Shuja Tahir