

Changes in Serum Lipid Profile of Albino Rats Fed on Canola Oil Supplemented with Atherogenic Elements: 18 weeks study

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Amongst lipid lowering factors, the dietary intervention for treating hyperlipidaemia and atherosclerosis associated ailments is physiological and gratifying. Canola is rich in monounsaturated fatty acids and is locally cultivated and cost effective. The criterion of the study was to determine the status of serum lipid profile in albino rats fed on canola oil diets for prolonged period of 24-weeks and to compare the same with those fed on canola oil supplemented with atherogenic elements. Results demonstrate that moderate amounts of canola oil (2.9% in diet) has triglyceride and cholesterol lowering effects. Both moderate and high amounts of canola oil (20%) decrease serum LDL-c profiles and resist lowering of HDL-c levels in rats fed with canola oil alongwith atherogenic elements. **Conclusions:** These findings reveal canola oil amongst the important supplement of monounsaturated fatty acids in animals and these findings may be generalized for humans.

Key Words: Canola oil diets, serum lipid profile, albino rats.

Hyperlipidaemia is virtually and universally acknowledged to be the major risk factor for atherosclerosis and other cardio-vascular diseases especially coronary artery disease (CAD) leading to the angina pectoris, acute myocardial infarction, sudden cardiac arrest, ischemic heart disease and hypertension depending upon severity of narrowing of the vessels^{1, 2}. Selection of dietary lipids is one of the factors that play an important role in regulating hyperlipidaemia. There is growing evidence that patients can improve their lipid profiles and decrease cardiovascular events by switching their diet from saturated and polyunsaturated fats or monounsaturated fats. It is because increased intake of saturated fats leads to oxidation of LDL-c and increase its uptake by macrophages, foam cell formation within the arterial intima and development of coronary artery obstruction³. The change from saturate to unsaturated diets may improve endothelial dynamics, reduce oxidation of LDL-c and atherosclerosis and enhance thrombolytic activity by decreasing platelet aggregation which is associated to thrombus formation and the risk of stroke and myocardial infarction⁴. It has been observed that people taking diet high in olive oil had lowest plasma TC and LDL-c levels in comparison to others that had used western diet (rich in saturated fatty acids⁵). Monounsaturated fatty acids are present in olive oil, certain nuts, rape-seed and canola oils.

It is reported that canola oil (n-3 fatty acid rich) diet in albino rats increases HDL-c and lowers LDL-c and preserves the myocardium more than the standard cholesterol-rich diet⁶. Further, canola oil diet enriched in monounsaturated fatty acids decreased TC and LDL-c serum levels as compared to saturated fats⁷. The long-term effects of intake of canola oil in rats are unknown. Hence the present study was carried out to observe the effects of prolonged intake of canola oil supplemented with atherogenic elements on serum lipid profiles.

Materials and Methods

An experimental study was planned and sixty albino rats

divided into five groups of twelve animals were selected (A to E). Diets were prepared for five different groups⁸. The diets were composed of wheat, starch, casein, glucose, choline/methionine, mineral and vitamin mixtures in an appropriate quantities by substitutions with different amount of canola oil for each of other four groups B, C, D and E. Group A was kept as control on routine synthetic diet. The experimental diets, consisting of 2.9 % canola oil (B), 2.9% canola oil + atherogenic element, bile salts 0.3%, propylthiouracil, 0.1% and cholesterol 1.00%, (C), 20 % canola oil (D) and 20 % canola oil + atherogenic element (E) were started. These diets were continued for 18 week and 2ml blood samples at zero and 18 weeks were collected after overnight fasting. TG, TC and HDL-c were estimated by Randox kits using enzymatic colorimetric methods. LDL-c was calculated by Friedwald formula. The statistical analysis was done by the help of student's 't' test and level of significance was determined.

Results

Results and level of significance of these groups are given in tables 1-4.

Table 1. Variations in triglycerides (mmol/L) levels (n=12,±SD).

Groups	Zero-week	18-week
A	1.139 ± 0.069	1.272 ± 0.073
B	1.111 ± 0.075	*1.103 ± 0.059
C	1.157 ± 0.071	*1.157 ± 0.049
D	1.120 ± 0.062	*1.166 ± 0.055
E	1.114 ± 0.071	*1.198 ± 0.041

A vs B,C,D,E is highly significant (p<0.001) at 18-week.

Table 2 Variations in total cholesterol (mmol/L) contents (n=12±SD).

Groups	Zero-week	18-week
A	2.041 ± 0.123	2.267 ± 0.135
B	2.028 ± 0.108	2.168 ± 0.122
C	2.038 ± 0.135	2.173 ± 0.145
D	2.082 ± 0.147	*2.013 ± 0.081
E	2.080 ± 0.124	*2.090 ± 0.098

A vs D,E is highly significant (p<0.001) at 18-week.

Table 3. Variations in LDL-c (mmol/L) contents (n=12,±S.D.).

Groups	Zero-week	18-week
A	0.923 ± 0.057	1.057 ± 0.079
B	0.960 ± 0.083	1.007 ± 0.105
C	0.901 ± 0.142	1.048 ± 0.143
D	0.965 ± 0.141	*0.788 ± 0.048
E	0.995 ± 0.141	*0.923 ± 0.061

A vs D,E is highly significant (p<0.001) at 18-week.

Table 4. Variations in HDL-c (mmol/L) levels (n=12,±S.D.).

Groups	Zero-week	18-week
A	0.615 ± 0.038	0.635 ± 0.041
B	0.583 ± 0.028	0.638 ± 0.041
C	0.611 ± 0.031	0.612 ± 0.033
D	0.615 ± 0.039	*0.711 ± 0.048
E	0.614 ± 0.038	0.632 ± 0.041

A vs D is highly significant (p<0.001) at 18-week.

Discussion

Hyperlipidaemia is progressive, largely irreversible and primarily dependent on life style factors especially on diet and is difficult to manage effectively with drug therapy. Restriction of the intake of the fat and selective use of fat or oils may be first line intervention for hyperlipidaemia. Saturated fatty diets usually cause elevations of serum TC and LDL-c levels which contribute to the development of atherosclerosis that leads to CHD. Amongst various serum lipids, TC has received too much importance because of its strong and consistence association with CHD. It has been demonstrated that the significant elevation of LDL-c level is positively associated while elevation of HDL-c is negatively associated with the development of CHD. High TC and LDL-c levels increase the risk of CHD, while raised HDL-c is a powerful protective factor against CHD¹⁰.

The effects of dietary constituents on hypercholesterolaemia and atherosclerosis have received much attention because vegetable proteins polyunsaturated fatty acids and dietary fiber appeared to lower the blood TC and events of atherogenic progress¹¹. There are growing evidences that patients can improve lipids levels and may avoid cardiovascular events by using in their diets monounsaturated fatty acids rather than saturated. Because polyunsaturated fatty acids intake increases the oxidation of LDL-c which enhances the uptake of LDL-c by macrophages leading to foam cell formation phenomenon and causing CHD³, the change from the advancing use of polyunsaturated fatty acids towards monounsaturated fatty acids may decrease the risk of stroke and MI⁴. Among the cost effective and monounsaturated fatty acids rich oils, canola oil was used to assess the serum lipid profile.

Serum TG levels lowered in experimental groups (canola oil diet groups) at 18 week. Our findings are in consistent with the published work^{5,12}. The rise of TG in high canola vs low canola may be due to increased concentration of high fat (20 % canola vs 2.9 % canola) and this is in confirmation with the literature¹³. Serum TC

levels lowered in canola oil diets groups suggesting that monounsaturated fats cause hypocholesterolaemia as observed by Keys et al 1970¹⁴. TC levels lower in high canola groups because of cholesterol lowering effects of oleic acid by enhancing the LDL-c receptors. Similar profiles were shown by Matheson et al 1996¹⁵ using canola oil in human volunteers.

Serum HDL-c levels at 18 weeks were increased in high canola oil diet group without atherogenic supplement but lowered in atherogenic diet groups as demonstrated^{16,17} suggesting that monounsaturated fatty acids favourably increase HDL-c levels. Serum LDL-c levels at 18 week were decreased non-significantly. High canola oil diet showed decrease at 18 weeks. Similar findings are documented by and consistent with literature^{18,19}. These studies demonstrate that prolonged intake of canola oil in albino rats is effective way to reverse the existing atherosclerotic lesions and hyperlipidaemia as a physiological dietary intervention.

References

- Lusis, A.J. Atherosclerosis. Nature. 2000. 407: 233-241.
- Levenson JW, Skerrett PJ and Gaziano JM.. Reducing the global burden of cardiovascular disease: the role of risk factors. Prev Cardiol. 2002; 5(4): 188-199.
- Reaven P, Parthasarathy S, Grasse BJ, Moller E, Almazan F et al. Feasibility of using an oleate-rich diet to reduce the susceptibility of low density lipoprotein to oxidative modification in humans. Am J Clin Nutr 1991; 54: 701-6.
- Renaud S, Godsey F, Dumont E, Thevenon C, Orchanian E, Martin JL. Influence of long term diet modification on platelet function and composition in Moselle farmers. Am J Clin Nutr 1986; 43: 136-50.
- Mahley RW, Palaoghe KE, Atak Z, Dawson Pesin J et al. Turkish Heart study: lipids. Lipoproteins and apolipoproteins. J Lipid Res. 1995; 36(4): 839-59.
- Aquila MB, Rodrigues-Apfel MI, Mandarim-de-Lacerda CA. Serology of the myocardium and blood biochemistry in aged rats fed with a cholesterol rich and canola oil diet (n-3 fatty acid rich). Brasil: Basic-Res-Cordioli 1998; 93(3): 182.
- Schwab US, Vogel S, Kammi-Keefe CJ, Schaefer EJ, Li Z, Ausman LM et al. Lipid Metabolisms laboratory. Boston, USA: J Nutr, 1998; 128(10): 1703-9.
- Welhe WH. The Laboratory Rat. Ist Ed. London: CV Mosby, 1983; 309-29.
- Lasser LN, Roheim PS, Edelstein D. Serum lipoprotein of normal and cholesterol fed rats. J Lipid Res, 1973; 14: 1-8.
- McNamara DJ, Howell WH. Epidemiologic data linking diet to hyperlipidaemia and arteriosclerosis. Seminars in Liver Disease. 1992; 12: 347-55.
- Chi MS, Koh ET, Stewart TJ. Effect of garlic on lipid metabolism in rats fed cholesterol or lard. J Nutr. 1984; 112: 241-81.
- Sattar A. The effects of *Nigella sativa* on serum and tissue lipids in albino rats fed on palm oil and atherogenic diet for prolonged period. (M.Phil. Thesis): Univ of the Punjab, Lahore. 1996: 168.
- Grundey SM. Monounsaturated fatty acid, plasma cholesterol and coronary heart disease. Am J Clin Nutr. 1987; 45: 1168.
- Keys A, Menotti A, Karvonen MJ, et al. The diet and 15

- year death rate in the seven countries study. *Am J Epidemiol* 1970; 124(6): 603-15.
15. Matheson B, Walker KZ, Taylor DM, Peterkin R, Lugg D and Dea KO. Effect on serum lipids of monounsaturated oil and margarine in the diet of Antarctic Expedition. *Am J Clin Nutr* 1996; 63: 933-8.
 16. Sahito MM, Effects of Long term administration of various dietary fats on serum and tissue lipids in the rat. (M.Phil. Thesis): University of the Punjab, Lahore. Pakistan. 1993; 239-42.
 17. Shad MA. Quantitative effects of olive oil on serum lipid profile in albino rats. (M.Phil. Thesis): Univ of the Punjab, Lahore. Pakistan. 1991: 145-47.
 18. Wardlaw G, Snook JT, Lin MC, Puangco MA and Kwon SS. Serum lipid and apolipoproteins concentrations in healthy men on diets enriched in either canola oil or Safflower oil. *Am J Clin Nutr* 1991; 51: 104-10.
 19. Dreon DM, Vramizan KM, Krauss RM, Austin MA, Wood PD. The effects of polyunsaturated fats vs monounsaturated fat on plasma lipoproteins. *JAMA*. 1990; 263: 2462-66.