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Effect of vegetable oils on the lipid profile and antioxidant status in Wistar rats: A comparative study

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Abstract:

High level of total cholesterol, more importantly low density lipoprotein (LDL) cholesterol, is the major risk factors in coronary heart disease. Clinical trials have demonstrated that the increase in plasma LDL levels and oxygen derived free radicals have been implicated in the early development and progression of atherosclerosis. Vegetable oils are used for cooking foods by many ethnic groups. A fear and controversies have been created in the public that consumption of certain vegetable oils causes atherogenesis. Hence, the present study is designed to evaluate the effect of vegetable oils such as coconut, sunflower, palm, olive oil and vanaspati on lipid profile and antioxidant status using rat model. Reference dose of different vegetable oils were administered once daily for 90 days. Results indicate that, in all the vegetable oil treated groups, a decline of antioxidant status associated with an increase in lipid peroxidation was observed. Furthermore, total cholesterol was found to be elevated in all groups. Among the vegetable oils treated groups, coconut oil was found be comparatively better. This conclusion was made based on the non-significant mild elevation of peroxidation and the benefit of increase in high density lipoprotein (HDL).

Key words: Antioxidants, atherosclerosis, lipid profile, oxidative stress, vegetable oils

Introduction:

Coronary heart diseases are one of the most prevalent diseases of today (about 40% of world population and 50% of Indian population).[1] A major internal threat to cellular homeostasis of aerobic organisms arises from reactive oxygen metabolism. Aerobic organisms are under a continual state of "oxidative siege" by Reactive Oxygen Species (ROS). Body has extensive antioxidant defense mechanisms to counteract the damaging effects of reactive oxygen species. There is a balance between ROS and antioxidants. However, this balance may be shifted towards the pro-oxidants when production of oxygen species is increased greatly or when levels of antioxidants are diminished. This condition is "Oxidative stress" and can result in serious cell damage if the stress is massive or prolonged**.** Oxygen derived free radicals (ODFR) have been implicated in pathology of various human diseases including atherosclerosis. Susceptibility of LDL to oxidation depends both on concentration of pro-oxidant stimuli and the entity and the concentration of antioxidants.[2] So, it is imperative to study the whole profile of free radicals rather than study only lipid peroxide products like Malondialdehyde (MDA).[3]

Coconut and coconut oil have been important sources of food ingredients in Coastal Karnataka, Kerala and other coastal world for a long time. However, a fear has been developed in the public that consumption of coconut oil causes atherogenesis as coconut oil contains large concentrations of saturated fatty acids (89.5%).[4] This arose due to the contemporary popular literature of epidemiological studies, which attributed an association between saturated fatty acids and serum cholesterol level and in turn atherogenesis. This belief is primarily due to equating coconut oil with saturated fats without actually taking into consideration the fact that the saturated fats in coconut oil are mainly short chain fatty acids (Lauric acid-

43-51 %) and medium chain fatty acids (Myristic acid - 16-21 % and Capric acid 6-8 %).[4] Analysis shows that none of the saturated fatty acids that are found in atheroma were reported to be lauric acid and myristic acid.[5] Hence, evaluating the role of coconut oil against the oxidative stress and lipid profile is worthwhile to recommend its use to the public. In this manuscript, we report the role of coconut and other vegetable oils on antioxidant status and lipid profile.

Materials and Methods:

Animals

Thirty six male Wistar rats weighing about 120-150 grams were obtained from the Central animal facility of Kasturba Medical College (Manipal University), Mangalore. They were housed in institutional experimental animal laboratory. The rats were kept in cages at room temperature. They had free access to food and water. The experiment was carried out according to the guidelines of Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), Government of India and approved by Institutional Animal Ethics Committee.

Experimental design:

Animals were divided into 6 Groups (Control group n = 12 and n=6 in all other groups).

Group 1- Rat fed with Normal chow diet (Control rats)

Group 2- Rat fed with Normal chow diet + Coconut oil (2ml/day)

Group 3- Rat fed with Normal chow diet + Olive oil (2ml/day)

Group 4- Rat fed with Normal chow diet + Palm oil (2ml/day)

Group 5- Rat at fed with Normal chow diet + Sunflower oil (2ml/day)

Group 6- Rat fed with Normal chow diet + Vanaspati (2ml/day).

All the oils were administered orally using gavage once daily for ninety days. At the end of the experiment, the animals were euthanized after 12 hrs of fasting. Blood was collected by cardiac puncture.

Biochemical evaluations

Triacylglycerol (TAG), total cholesterol (TC) and high-density lipoprotein cholesterol (HDL) were determined in serum samples using commercial kit (Randox, India); very low density lipoprotein cholesterol (VLDL) was calculated as TAG/5 and low density lipoprotein cholesterol (LDL) was estimated using Friewald's formula.[6] Serum Vitamin C was determined by the method of Tietz.[7] In the RBCs, the activity of superoxide dismutase (SOD) was determined by the method of Marklund and Marklund.[8] Glutathione (GSH) level was determined using the method of Beutler et al.[9] Catalase activity was determined by the method of Aebi et al.[10] MDA level was determined using the method of Ohkawa et al.[11]

Statistical analysis

All data were represented as mean ± SD. The mean values were statistically analyzed using one-way analysis of variance (ANOVA). The significant differences between the groups were further analyzed by student 't' test. *P* values less than 0.05 were considered as significant.

Results:

Table I shows the effect of different vegetable oils on serum lipid profile.

Sunflower oil treated groups showed a significant decrease in the Serum triacylglycerol ($P<0.01$); and decrease in VLDL level ($P<0.01$). There was a significant increase in the serum LDL level in the rats treated with olive oil ($P<0.05$). Further, a significant increase in the serum LDL cholesterol level ($P<0.001$) in the Vanaspati treated rats was seen. Even though an increase in the total cholesterol and HDL level was observed in coconut oil treated groups, statistically significant increase was not observed.

Table II shows the effect of treatment of different vegetable oil on antioxidant parameters.

MDA level showed a significant increase in the oil treated groups [Olive oil and Palm oil ($P<0.05$), Vanaspati ($P<0.001$) and Sunflower

Table I: Effect of different vegetable oils on serum lipid Profile:
Values are expressed as Mean ± SD; n=6 (total number of animals in each group), Control n=12

Groups (Control n=12, Other groups n=6)	TAG (mg/dl)	Total Cholesterol (mg/dl)	HDL Cholesterol (mg/dl)	VLDL Cholesterol (mg/dl)	LDL Cholesterol (mg/dl)
Group 1: Normal diet	75.41 ± 6.23	77.08± 6.43	42.41± 5.58	15.08±1.26	19.58± 1.70
Group 2: Normal diet + Coconut oil	73.83± 6.43	82.33 ±4.80	47.17± 3.76	14.76± 1.28	20.41±0.87
Group 3: Normal diet + Olive oil	70.33± 6.31	79 ± 7.34	41.5± 4.08	14.06± 1.26	23.43± 2.50*
Group 4: Normal diet + Palm Oil	69.17± 6.24	78.33 ± 6.25	42.17± 2.74	13.83± 1.24	22.33± 2.54
Group 5: Normal diet + Sunflower oil	63± 4.69**	76.17± 7.65	43.83± 4.07	12.6± 0.93**	19.73± 2.87
Group 6: Normal diet + Vanaspati	78.67± 4.32	82.17± 6.27	38.67± 4.22	15.73± 0.86	27.76± 3.54***

* P< 0.05, **P< 0.01, ***P< 0.001; control (Rats fed with normal chow diet versus rats fed with normal chow diet + different oils

oil (P<0.001)]. A significant decrease in the GSH level was observed in the Palm oil (P<0.05), Olive oil (P<0.01), Sunflower oil and vanaspati (both P<0.001). SOD level significantly decreased in Sunflower oil treated groups (P<0.001). Further, a significant decrease in the Vitamin C level was observed in the oil treated groups - Vanaspati and Olive oil (P<0.001), Sunflower oil (P<0.01); and Palm oil (P<0.05).

Discussion:

Development of atherogenesis and coronary heart diseases has many risk factors. Hyperlipidemia is one of the major risk factor for atherosclerosis. Cigarette smoking, diabetes mellitus, stress, heredity factors are the other major risk factors. A major internal threat to cellular homeostasis of aerobic organisms arises from reactive oxygen metabolism. Aerobic organisms are under a continual state of "oxidative siege" by Reactive Oxygen Species (ROS). Body has extensive antioxidant defense mechanisms to counteract the damaging effects of reactive oxygen species. There is a balance between ROS (Reactive Oxygen Species) and antioxidants. However, this balance may be shifted towards the pro-oxidants when production of oxygen species is increased greatly or when levels of antioxidants are diminished.

Free radicals play a major role in development and progression of atherogenesis. Oxidized LDL is considered as one of the most important factor in the development of atherogenesis. The free radicals oxidize normal LDL to oxidized LDL, which is taken up by macrophages. Excessive uptake of modified macrophages causes the transformation of these cells into foam cells, which participates in formation of atherosclerotic plaques.[12] Susceptibility of LDL to oxidation depends both on concentration of pro-oxidant stimuli and the entity and concentration of antioxidants.[2]

Table II: Effect of different vegetable oils on antioxidant parameters:
Values are expressed as Mean ± SD; n=6 (total number of animals in each group), Control n=12

Groups (Control n=12, Other groups n=6	MDA (nmol/g Hb)	GSH (µmol/g Hb)	SOD (U/g Hb)	Catalase (U/g Hb)	Vitamin C (mg/dl)
Group 1: Normal diet	30.50±2.72	10.24± 0.70	5567.22± 342.70	25102.25± 3740.82	1.12± 0.06
Group 2: Normal diet + Coconut oil	32.08±2.18	9.62± 0.33	5342.70 ±216.49	23548.62± 1203.60	1.06± 0.05
Group 3: Normal diet + Olive oil	* 36.02±4.17	** 9.29 ±0.46	5166.41± 384.42	24466.43± 1476.35	*** 0.98± 0.06
Group 4: Normal diet + Palm Oil	* 36.8± 3.46	* 9.42± 0.14	5286.12± 273.16	23467.320± 1166.30	* 1.02±0.04
Group 5: Normal diet + Sunflower oil	*** 48.42±4.13	*** 8.76± 0.29	*** 4842.44± 273.15	22428.73± 1205	*** 1.01±0.04
Group 6: Normal diet + Vanaspati	*** 42.52±4.95	*** 8.94 ± 0.24	5134.39± 260.62	21862.61±1546.27	*** 0.92±0.08

* P< 0.05, **P< 0.01, ***P< 0.001; control (Rats fed with normal chow diet versus rats fed with normal chow diet + different oils

Many biological effects of the free radicals results from their attack on lipids (of membranes), nucleic acids, proteins and carbohydrates. Administration of vegetable oils in rat daily for 90 days, significantly changes the antioxidant status as evident from the results of this study.

GSH is the major antioxidant non-protein thiol present in the body. Administration of coconut oil could decrease the GSH level when compared to that of the normal group. A statistically decreased value was found in the sunflower oil, vanaspati, olive oil and palm oil treated group. The decrease of GSH among the vegetable oil treated group was sunflower oil> vanaspati> olive oil> palm oil>coconut oil. The decrease of SOD among the vegetable oil treated group was sunflower oil> vanaspati> olive oil> palm oil>coconut oil, significantly lowered only in sunflower oil treated group. Similarly, catalase and vitamin C were decreased in the vegetable oil treated group. The decrease in SOD and vitamin C was lowest in the coconut oil treated group. The increased level of lipid peroxidation was observed in the sunflower treated group than the other vegetable oil treated group. This can be ascribed to the low GSH level in this group. Among the vegetable oil treated animals, coconut oil did not produce any significant increase in the MDA level. This indicates that the saturated medium chain fatty acids in coconut oil are not vulnerable to the peroxidation at the physiological level of ROS. This could be further supported by the significantly increased MDA level in other vegetable oil treated group, maximum in sunflower oil indicating that polyunsaturated fatty acids in sunflower oil are more vulnerable to the peroxidation at the physiological level of ROS. Increased level of lipid profile was found in the vegetable oil treated groups. Though the coconut oil increased the total cholesterol level, it effectively increased the HDL cholesterol too. Sunflower oil also increases the HDL; palm oil did not change its level. Furthermore, total

cholesterol level was found to be increased in all the vegetable oil treated groups except in the group treated with sunflower oil. The decrease in TAG was found maximum in the sunflower oil treated group.

Furthermore, oxidized LDL is considered in the development of atherogenesis. Conversion of LDL to oxidized LDL (OxLDL) depends both on concentration of oxidants and pro-oxidants and the concentration of antioxidants.[2] OxLDL is taken up by macrophages. The modified macrophages cause the transformation into foam cells and formation of atherosclerotic plaques, as well**.**

Hence, the result of this study concluded that vegetable oils increase the total cholesterol and decrease the antioxidant status. In all the vegetable oil treated groups, a decline of antioxidant status associated with an increase in lipid peroxidation was observed. However, among the oil treated groups, coconut oil was found to be comparatively better. This conclusion is made based on the non-significant mild induction of peroxidation and the increased level of HDL. Further, study on the effect of these vegetable oils in rats fed with high fat diet is warranted to support this conclusion.

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