Lipid profile and antioxidant enzymes in coconut oil consumers

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The consumption of coconut oil, as a part of routine diet, may not contribute to the risk for CAD, directly by affecting the lipid profile or indirectly by aggravating oxidative stress. It may not be the type of cooking oil, rather its quantity that may be contributing to the risk of Coronary Artery Disease (CAD).

Abstract

In this study, we compared the lipid profile and antioxidant enzymes of normal and diabetic subjects consuming coconut oil and sunflower oil. 70 normal healthy persons and 70 patients with diabetes were studied. Each group was further subdivided into two subgroups of 35 subjects each, consuming coconut oil and sunflower oil respectively as cooking medium. Samples of blood were analyzed for serum total cholesterol, triacylglycerols, and cholesterol in lipoprotein fractions. Total glutathione and glutathione peroxidase were measured in erythrocytes and superoxide dismutase in serum. Though lipid profile parameters and oxidative stress were high in diabetic subjects compared to controls, no pronounced changes for these parameters were observed between the subgroups (coconut oil vs. sunflower oil).

Introduction

Coconut has been an important component of the diet of Kerala population for decades. Coconut oil contains approximately 90% saturated fats. Saturated fats are known to contribute to coronary artery disease (CAD) by causing hypercholesterolemia, an established risk factor for CAD. However, most of the saturated fats of coconut oil are medium chain fatty acids having 10 to 12 carbon atoms, which are preferentially transported through the portal venous system to the liver. Thus, medium chain fatty acids are more available for oxidation and they provide a rapid source of energy (1) and are considered to be less implicated in the accumulation of body fat (2). Great propaganda that coconut oil is a major contributor to the rise in the incidence of CAD among the Kerala population have made the people shift to alternate cooking oils like sunflower oil. Long chain fatty acids like linoleic acid are incorporated into chylomicrons and follow the lymphatic system. In the hepatocytes, they form triacylglycerols and esterify cholesterol to give cholesterol esters (3). Most of the recent investigations conducted in animals as well as human beings show that coconut oil does not increase the risk of atherosclerosis and heart disease (4,5). Though the effect of coconut oil on lipid profile has been studied in detail by many investigators, studies on its effect on oxidative stress are scanty (6) and have not been understood properly. In this study, we compare the lipid profile and anti oxidant enzymes (total

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glutathione, glutathione peroxidase and superoxide dismutase) of subjects who were consuming coconut oil (saturated, medium chain fatty acid) and those subjects consuming sunflower oil (polyunsaturated, long chain fatty acid).

Materials and Methods

Male subjects aged from 35 to 65 years, who came to the hospital for routine health check or for diabetic evaluations, were recruited for the study.

Group 1 consisted of 35 control subjects consuming coconut oil

Group 2 consisted of 35 control subjects consuming sunflower oil

Group 3 consisted of 35 subjects with Type 2 diabetes consuming coconut oil

Group 4 consisted of 35 subjects with Type 2 diabetes consuming sunflower oil.

All these persons were consuming the respective oil as the predominant cooking medium for over a period of six years. The subjects derived approximately 13 to 20% of their total calories from the oil considered. Diabetic subjects were on insulin or oral hypoglycemic agents, but none of them had previously diagnosed CAD.

Serum was analyzed for lipid profile, including Total cholesterol, HDL cholesterol, LDL cholesterol and triaclyglycerol (TAG) concentrations. Erythrocytes were washed thrice with ice-cold physiological saline, lysed with deionized water and used for determination of glutathione (GSH) and glutathione peroxidase (GPx). GSH was estimated in erythrocytes using the method of Beutler *et al* (7). GPx was assayed in erythrocytes according to Paglia and Valentine (8), as modified by Lawrence and Burk (9). Superoxide dismutase (SOD) was estimated in the serum, based on the method of Marklund and Marklund(10).

Statistical analysis was carried out in SPSS, version 11.0. Independent 't' test was used to compare normal analytes and "Mann Whitney U test" was applied to compare non-normal analytes. The differences were considered significant if the p value was <0.05. Comparisons were drawn between coconut oil and sunflower oil consumers (between groups 1 & 2, 3 & 4, 1 & 3 and 2 & 4).

Results

Total cholesterol showed significant difference only between control subjects consuming sunflower oil and diabetics consuming sunflower oil (between groups 2 and 4) (see Table 1). The HDL cholesterol levels also showed the same pattern of statistically significant difference between groups 2 and 4. Triacylglycerols, LDL cholesterol and VLDL cholesterol levels were high for the diabetic subjects of both groups (groups 3 and 4) compared to their respective controls (groups 1 and 2).

GSH and GPx values were showing significant decrease for diabetic subjects (groups 3 and 4) compared to their controls (groups 1 and 2), while SOD values showed significant variation between coconut oil consuming groups only (groups 1 and 3).

Table 1. Mean values and standard deviations of serum lipid levels of subjects

	Group 1 Control Coconut oil	Group 2 Control Sunflower oil	Group 3 Diabetic Coconut oil	Group 4 Diabetic Sunflower oil
Mean age	44.9 ± 7	45.2 ± 8.6	55.1 ± 8.6	53.6 ± 7.4
Plasma lipidsTotal cholesterol (mg/dL)	161.3 ± 30.7	157.1 ± 28	172.4 ± 35.9	179.1 ± 32.3*
Triacylglycerols (mg/dL)	136.5 ± 44.7	125.2 ± 38.3	162.1 ± 47.9*	151.2 ± 37.4*
HDL cholesterol (mg/dL)	47.8 ± 10	44.3 ± 8.5	43.8 ± 10.3	39.9 ± 9.8*
LDL cholesterol (mg/dL)	78.3 ± 24.2	82.6 ± 26.9	108.2 ± 35.4*	121.7 ± 34.9*
VLDL cholesterol (mg/dL)	27.3 ± 8.9	25.00 ± 7.7	32.4 ± 9.6*	30.2 ± 7.5*

Values are mean ± SD, * p < 0.05 compared to controls. No significant changes were observed between subgroups (Groups 1& 2 and groups 3& 4).

Table 2. Mean values and standard deviations of antioxidant enzyme levels of subjects

	Group 1 Control Coconut oil	Group 2 Control Sunflower oil	Group 3 Diabetic Coconut oil	Group 4 Diabetic Sunflower oil
GSH (nmoles/g Hb)	7.14 ± 0.7	6.88 ± 0.73	5.5 ± 0.87*	5.26 ± 0.95*
GPx (nmol of NADPH oxidized/minute/g Hb)	18.3 ± 1.8	18.7 ± 2.1	16.8 ± 2.2*	17 ± 1.6*
SOD (U/ml serum)	5.59 ± 1.14	5.22 ± 1.22	4.67 ± 0.98*	5 ± 1.1

Values are mean ± SD, * p < 0.05 compared to controls. No significant changes were observed between subgroups (Groups 1& 2 and groups 3& 4).

Most importantly lipid profile or oxidative stress parameters did not show significant changes between coconut oil and sunflower oil groups.

Discussion

Atherogenic dyslipidemia is a major risk factor for CAD. This condition is characterized by borderline high LDL cholesterol (130-160 mg/dL), low HDL cholesterol (<35 mg/dL), high triacylglycerols (>150 mg/dL) and increased small dense LDL particles (11). Lipid profile parameters of the diabetic subjects of the present study showed considerable variation from the controls. 11.4% of our diabetic subjects had atherogenic lipid profile compared to 2.8% of the control subjects. LDL cholesterol level was significantly high for diabetics compared to the controls. Oxidized LDL induces the development of atherosclerosis (12). There are numerous animal as well as human studies in which lipid profile parameters on consuming coconut oil and other dietary fats were compared. The results obtained are not uniform. Some studies have failed to find any association of coconut oil with adverse lipid profile changes (13) and some that showed that coconut oil consumption has beneficial effects compared to other dietary fats (14). The effectiveness of polyunsaturated fatty acids in reducing serum cholesterol and LDL cholesterol has been well documented (15). But several studies reported that polyunsaturated fats also lower HDL cholesterol and therefore may not be as good as it was thought previously (16,17). HDL cholesterol concentrations were low for sunflower oil groups compared to coconut oil groups in the present study also. Results from Table 1 fails to provide any indications that coconut oil consumers have undesirable lipid profile pattern or increased risk for CAD compared to sunflower oil consumers. This present finding is in harmony with an earlier study conducted in Kerala population, which indicates that habitual consumption of coconut and coconut oil has no specific role in the causation of coronary heart disease in Kerala population (18).

Coronary artery disease is now considered as an inflammatory disease and accumulating evidence is now available to suggest that oxidative stress may contribute or aggravate the process of atherosclerosis (19). Enzymatic inactivation of reactive oxygen species is achieved by glutathione peroxidase and superoxide dismutase (20). Results of recent studies indicate that oxidative stress occurs in patients with CAD despite being clinically stable and under medical treatment (21). Diabetes is a major risk factor for atherosclerosis and diabetes is exacerbated by oxidative stress (22). In the present study, there is a significant reduction in the GSH and GPx levels among the diabetic subjects compared to the controls (Table 2). This clearly indicates that, their anti oxidant defenses are weak compared to the healthy population. A reduction in serum SOD activity was noticed for diabetic subjects in this study (Table 2) and earlier studies have reported similar results (23).

The role played by various dietary fats on oxidative stress in humans has not been elucidated so far. The formation of the promutagenic, exocyclic DNA adducts in the liver of rats, which are markers for DNA damage by lipid peroxidation, was found to be highest in sunflower oil fed rats when compared to coconut oil, olive oil or rapeseed oil (24). It was found that rats fed with coconut oil have low susceptibility to lipid peroxidation compared to olive or sunflower oil diets (25). Since there was no noticeable variation for the anti oxidant enzymes of the subjects consuming either of the oils, it may be concluded that the type of dietary fat consumed may not be a major contributory factor to oxidative stress in this population.

Hence, it may be concluded that the consumption of coconut oil, as a part of routine diet, may not contribute to the risk for CAD, directly by affecting the lipid profile or indirectly by aggravating oxidative stress. It may not be the type of cooking oil, rather its quantity that may be contributing to the risk of CAD.

In continuation to this small study, a prospective study with larger sample size is now in progress with the financial assistance from the Coconut Development Board.

Analysis of fatty acid composition of plaque and plasma is now under investigation in our department.

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VCO in capsules more effective - Philippines

The capsulized version of the highly acclaimed Virgin Coconut Oil (VCO) is more effective than its liquid version, said General Surgeon Dr. Ed Lalusis, Medical Director and CEO of Growrich Manufacturing Inc., Philippines. According to the latest studies on VCO products, the sales of coconut oil in capsule form registered a significant increase than its bottled VCO counterparts.Describing VCO as the "miracle oil," Lalusis said the VCO is considered the healthiest dietary oil on earth and has earned a good following among Filipino overseas contract workers and foreigners in different countries all over the world. He said that overseas workers before encountered problems in bringing VCO in cold climate countries and drinking it there. "Those working in countries with cold climates usually get frustrated when the VCO liquid freezes or hardens," said Dr. Lalusis. "But OFWs who take with them the capsulized VCO product, do away with these storage problems." Another reason for encapsulating virgin coconut oil is the growing concern of doctors on the oxidation that VCO liquid goes through once it is exposed to air. *(http://www.malaya.com.ph)*

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