REVIEW

Coconut Oil and Health Controversy : A Review

Damodaran .M. Vasudevan

Dr. D.M. Vasudevan MD FRCPath is Director – Research, R & D Centre, Agappe Diagnostics, Agappe Hills, Pattimattom, Ernakulam Dist, Kerala - 683 562, India.

Corresponding Author:
Dr. D.M. Vasudevan
E-mail:
dmvasudevan@yahoo.co.in

Various studies, including our own works have shown that coconut oil does not increase serum cholesterol level. The major fat in mother's milk is the same lauric acid as in coconut oil. Baby foods all over the world do contain lauric acid (that from coconut oil) as the prominent ingredient. The plaques in the diseased coronary arteries contain mainly long chain fatty acids (seen in other oils) and not medium chain fatty acids (of coconut oil); and this is the same irrespective of whether one takes sunflower oil or coconut oil. All these findings indicate that coconut oil is neutral with respect to atherogenicity (plaque formation and eventual heart attack). Other beneficial effects include that coconut oil increases serum HDL cholesterol; it produces very little free radicals, as opposed to other oils; it is rapidly absorbed, rapidly metabolized and so does not get deposited and it helps in resisting invading micro-organisms.

Keywords: Coconut oil, Fatty acids, Obesity, Coronary heart disease, Plaques.

INTRODUCTION

The health and nutritional benefits of coconut oil have been recognized for centuries. In India, coconut tree has been considered from time immemorial as "Kalpavriksha", literally meaning "a tree that bestows all boons". However, in recent decades, the reputation of coconut oil has been clouded by misinformation about its relation with atherosclerosis. Many health organizations advise against the consumption of high amounts of coconut oil due to its high levels of saturated fat, including US Department of Health and Human Services[1], World Health Organization[2], International College of Nutrition[3] and American Heart Association[4]. In the 1980s, a conspiracy has been made to convince the public that tropical oils (coconut and palm oils) contributed to their risk of coronary heart disease[5]. In US, successful campaigns were undertaken to force food manufacturers to remove tropical oils from their products and to replace them with hydrogenated vegetable oils, resulting in increased intakes of deleterious trans-fatty acids. In this review, the negative propaganda against coconut oil has been refuted.

ATHEROSCLEROSIS AND CHOLESTEROL

Coronary artery disease (CAD) is a condition in which the blood supply to the heart muscle is partially or completely blocked. Atherosclerosis is characterized by accumulation of lipids and cholesterol within the inside covering of the vessel wall, along with a component of vascular inflammation. The earliest pathologic lesion of atherosclerosis is the fatty streak, which may progress to form a fibrous plaque. This may further progress to luminal narrowing and compromised oxygen supply to the tissues supplied by that artery. The risk factors for the development and progression of atherosclerosis include hypercholesterolemia, hyperlipidemia, hypertension, cigarette habit and diabetes mellitus.

COCONUT OIL CONTAINS MEDIUM CHAIN FATTY ACIDS

Increased risk for CAD is attributed to elevated levels of serum cholesterol, which in turn is due to increased intake of saturated fats. However, a fear complex has been created among the general public that consumption of coconut oil results in elevated cholesterol levels. This "myth" is primarily due to equating coconut oil with saturated fat; without knowing that saturated fat in coconut oil are of the short chain and medium chain fatty acids. It is better clarified that all saturated fat are not harmful. It is to be emphasized that the fats that cause heart disease are saturated fats with long chain fatty acids. Nearly 50 % of the fat in coconut oil is lauric acid[6] which is a medium chain fatty acid (**Table 1)**.

Table 1 Fatty Acid Profile in Coconut oil

No. of Carbon atoms	Name of fatty acid	Concentration in coconut oil
C8	Caprylic acid	10%
C10	Capric acid	5%
C12	Lauric acid	55%
C14	Myristic acid	20%
C16	Palmitic acid	5%

COCONUT OIL IS EASILY DIGESTED AND ABSORBED

These medium chain fatty acids directly enter into the cells and are metabolized immediately. On the other hand, long-chain fatty acids (of other oils) require the help of lipoproteins, which are eventually deposited into various organs, including heart vessels (**Table 2**). Medium chain fatty acids are not dependent on carnitine transport[7] and they are metabolized rapidly by the body for energy requirements[8]. Thus, medium chain fatty acids are more available for oxidation and they provide a rapid source of energy and are considered to be less implicated in the accumulation of body fat[9].

COCONUT OIL DOES NOT INCREASE CHOLESTEROL LEVEL IN BLOOD

By giving coconut oil, scientists[10] found no statistically significant alteration in the serum levels of total cholesterol, HDL cholesterol or LDL cholesterol from the baseline values. They also noted a beneficial effect of adding the coconut kernel to the diet[11]. The polyphenol

Table 2 Metabolic differences between Medium chain and Long chain fatty acids		
Characteristics	**Medium chain fatty acid** (eg : Coconut oil)	**Long chain fatty acid** (eg : Other oils and fats)
For absorption, pancreatic lipase	Is not necessary	Absolutely essential
For absorption, Bile salts	Are not necessary	Absolutely essential
Absorbed as	Free fatty acid	Triglycerides
After absorption	Immediately metabolized by peripheral tissues	Transported by LDL into adipose tissue
Pathology	Not deposited in tissues	Deposited in tissues leading to plaque formation

fraction of virgin coconut oil was found to be capable of preventing LDL oxidation[12]. Sundaram *et al* fed coconut oil containing diets to healthy males[13]. Their findings indicate that a favorable alteration in serum lipoprotein balance was achieved when coconut oil was included in the diet. Coconut is the chief source of energy for populations of Polynesian islands. But vascular disease is uncommon in these populations[14]. In studies with neonatal pigs, the feeding of coconut oil did not in any way altered the blood lipid composition of animals[15]. Trautwein *et al* studied cholesterol-fed hamsters on different oil supplements[16]. Plasma cholesterol concentrations were higher for olive oil than for coconut oil. Studies conducted in some populations habitually consuming a diet rich in coconut and coconut oil have failed to establish that coconut oil increases risk for cardiovascular events[17]. In summary, studies that supposedly showed a hypercholesterolemic effect of coconut oil feeding, in fact, usually have only shown that coconut oil was not as effective at lowering the serum cholesterol as was the more unsaturated fat being compared.

LIPID PROFILE STUDIES

We have conducted a large scale study at Kochi, India[18]. Serum samples were analyzed from 302 normal healthy persons, out of which 152 were consuming coconut oil and 150 were using sunflower oil for the past 2 years or more. There were no statistically significant differences in the cholesterol, HDL or LDL levels in coconut oil consuming population versus sunflower oil consuming population (**Table 3**). Further, lipid profile in serum was analyzed in 76 coronary artery disease patients, out of which 41 were used to take coconut oil and 35 were used to take sunflower oil at least for the past 2 years. There were no differences in cholesterol levels in these two groups[18] (**Table 4**). Again, lipid profile was analyzed in 130 patients suffering from diabetes mellitus, out of which 69 were used to take coconut oil and 61 were used to take sunflower oil at least for the past 2 years (**Table 5**). From these figures it can be seen that there was no statistically significant difference in the cholesterol, HDL or LDL levels in coconut oil consuming population versus sunflower oil consuming population (**Tables 3, 4 and 5**). Plasma fatty acid composition reflected no changes with dietary fat source.

Hostmark *et al* in experiments with rats, showed that coconut oil feeding produced significantly high HDL, relative to sunflower oil feeding[19]. HDL cholesterol is considered to be the "good cholesterol" in the blood. Awad[20] showed that total tissue cholesterol accumulation in animals on the safflower diet was six times greater than for animals fed the coconut oil. Mensink *et al* reported that coconut oil consumption caused increase in HDL levels[21]. Our own studies on animals at Kochi, India showed that coconut oil intake did not cause hypercholesterolemia[22]. There are many animal and human studies in world literature to disprove allegations about coconut oil enhancing the risk of a CAD[23]. At the same time, there is not even one paper in the whole literature, directly showing that coconut oil increases cardiac diseases. Willett[24] in an editorial summarized that "no relation between saturated fat intake and risk of CAD was observed in the most informative prospective study to date". 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[17].

Table 3 Lipid Profile in Serum samples of Normal individuals				
Groups	Cholesterol mg/dl	HDL-C mg/dl	LDL-C mg/dl	Triglyceride mg/dl
Coconut oil group (n = 152)	203	46	124	143
Sunflower group (n = 150)	196	44	118	140

Table 4 Lipid Profile in Serum samples of Patients from Coronary artery disease				
Groups	Cholesterol mg/dl	HDL mg/dl	LDL mg/dl	Triglyceride mg/dl
Coconut oil group (n= 41)	152	42	95	116
Sunflower group (n= 35)	141	41	84	108

Table 5 Lipid Profile in Serum samples of Patients from Diabetes mellitus				
Groups	Cholesterol mg/dl	HDL mg/dl	LDL mg/dl	Triglyceride mg/dl
Coconut oil group (n=69)	193	44	120	166
Sunflower group (n= 61)	192	42	128	152

CHOLESTEROL DOES NOT CHANGE SMALL DENSE LDL PARTICLES IN CIRCULATION

It is known that small, dense LDL particles are more atherogenic than normal LDL particles as they are more susceptible to oxidation and have decreased affinity for LDL receptor[25]. So, we conducted a study to assess apo B and LDL-cholesterol/LDL-apo B ratio as a surrogate for small, dense LDL particles among normal subjects consuming either coconut oil or sunflower oil26. Group 1 and 2 consisted of 31 control subjects each, consuming coconut oil and sunflower oil respectively. The subjects were consuming on an average 24 g of the oil and derived approximately 10% of the total calories from their cooking medium. The percentage of subjects with apo B levels >0.9 g/L did not differ significantly between groups. LDL-cholesterol/LDL-apo B ratio below 1.2, which is a surrogate for small, dense LDL particles, were seen in 43% of control subjects, but the ratio did not show significant difference on comparing control and diabetic groups. It was further observed that the proportion of healthy coconut oil consumers, with low LDL-cholesterol/LDL- apo B ratio was high compared to the sunflower oil users (48.4% vs. 38.7%). Sunflower oil consumers of our study did not show considerable reduction in apo B concentration compared to coconut oil consumers.

COCONUT OIL IS NOT DEPOSITED IN HEART VESSELS

There are a few studies on chemical analysis of the atheromatous plaques (blood clogging substance inside the artery) Felton *et al* showed that the plaques contained cholesterol esters with 74% unsaturated (41% is polyunsaturated) and only 24% saturated fatty acids[27]. Moreover, none of the saturated fatty acids were Lauric acid (fats seen in coconut oil). At Amrita Institute of Medical Sciences, Kochi, India, we have analyzed the fatty acid composition of the plaques taken from diseased arteries. A total of 71 samples of plaques were analyzed, of which 48 persons were using coconut oil and 23 persons were using sunflower oil routinely (Sabitha *et al*, unpublished). Fatty acids were extracted by chloroform and then analyzed by HPLC (high performance liquid chromatography) as per the **Table 6**. Plaques did not contain significant amounts of lauric acid or myristic acid (fatty acids from coconut oil). Instead, palmitic acid and stearic acid (long chain saturated fatty acids) were the main ingredients of these plaques. Fatty acid content of plaques from coconut oil consuming group and sunflower consuming group were the same. This clearly shows that coconut oil does not

Table 6 Fatty acid composition from Plaques obtained from Diseased Coronary artery		
Fatty acids	**Plaque from Coconut oil consumers**	**Plaque from Sunflower oil consumers**
C10, Capric acid	0.32%	0.19%
C12, Lauric acid	3.0 %	3.9 %
C14, Myristic acid	5.0 %	5.0 %
C16, Palmitic acid	46.0 %	46.0 %
C18:0, Stearic acid	34.0%	33.0 %
C18:1, Oleic acid	6.0 %	6.0 %
C18:2, Linoleic acid	6.0 %	5.0 %

have any action on plaque formation or heart disease.

Thus Plaques from coronary artery does not contain fatty acid from coconut oil. Fatty acid content of plaques from coconut oil group and sunflower group are the same. This clearly shows that coconut oil does not have an effect to produce plaque or heart disease **(Table 6).**

ANTIOXIDANT STATUS IN COCONUT OIL CONSUMERS

In a parallel 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. Total glutathione and glutathione peroxidase were measured in erythrocytes and superoxide dismutase in serum (**Table 7**). Though oxidative stress parameters were high in diabetic subjects compared to controls, no pronounced changes for these parameters were observed between the subgroups (coconut oil vs. sunflower oil)[22].

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). Most importantly lipid profile or oxidative stress parameters did not show significant changes between coconut oil and sunflower oil groups. Although scanty, available previous studies on the effect of coconut oil on oxidative stress confirm our findings28. Results from Table 7 fail 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[29].

Table 7 Mean and Standard deviations of Antioxidant enzyme levels of Subjects				
Antioxidant enzyme levels	**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, * Significant Difference - $p < 0.05$ compared to controls, No significant changes were observed between subgroups (Groups 1& 2 and Groups 3& 4).

RANDOMIZED CLINICAL STUDIES

A large scale randomized single blind case control study comparing the impact of coconut oil versus sunflower oil as a cooking medium on the cardiovascular events and risk factors is ongoing at Amrita Institute of Medical Sciences. 200 patients with heart diseases were enrolled for a 2 years follow up. All the patients are getting their usual drugs, while one group (100 patients) is using only coconut oil, the

other 100 patients are using only sunflower oil. At the end of the 2 year study, all conventional atherosclerotic risk factors such as total cholesterol, LDL cholesterol, triglycerides etc were remained the same in both groups. Antioxidant markers (lipid peroxidase, glutathione reductase, glutathione-S-transferase, superoxide dismutase, catalase) were again shown to be the same in both groups. High Sensitive CRP (hsCRP) is an important inflammatory marker which is associated with atherosclerosis and predicts cardiovascular events. The hsCRP was also found to be the same level in both coconut oil users and sunflower oil users (Unpublished data).

PUFA, IN EXCESS, MAY BE HARMFUL

Poly unsaturated fatty acids (PUFA) can definitely reduce cholesterol level in blood. This finding led to clinicians advising patients to refrain from the use of coconut oil, and to use vegetable oils such as sunflower oil. Eventually, general public came to the conclusion that PUFA are safe. However, PUFA are notorious for lipid peroxidation and generation of toxic free radicals. The starting point of atheroma formation is the deposition of peroxide laden LDL in the arteries. High intake of omega-6 oils will cause lowering of HDL, elevation of plasma triglycerides, and will promote platelet aggregation; all of which favor heart attacks. Vegetable oils (e.g., sunflower oil), containing PUFA are rich in omega-6 variety; while coconut oil and butter are low in omega-6. Normal Indian diet (cereals, pulses and vegetables) contains about 10 g of PUFA per day; out of which about 2 g is omega-3 and the rest 8 g is omega-6. Further intake of omega-6, as sunflower oil or other vegetable oil may be harmful. The optimal ratio for omega-6 to omega-3 varieties in diet is 4:1. In an average Indian diet, this is about 30:1. In sunflower oil, this value is 160:1, and therefore, unnecessary addition of such vegetable oils will further deteriorate the condition. In coconut oil, the omega 6 to 3 ratio is 3:1, and therefore superior to sunflower oil in this respect.

OTHER HEALTH BENEFITS OF COCONUT OIL

The Lauric acid in coconut oil is used by the body to make the anti-microbial derivative monolaurin[30]. Coconut oil inhibits various microorganisms including bacteria, yeast, fungi, and enveloped viruses[31]. Coconut oil-treated wounds healed much faster, as indicated by a decreased time of complete epithelization[32]. The medium chain fatty acids in coconut oil offer positive health benefits for patients with irritable bowel syndrome and other digestive disorders, by reducing inflammation in the intestinal tract and helping to combat unhealthy microorganisms[33,34]. Coconut oil was found to reduce the protein loss in hair35. Coconut oil is shown to be anti-inflammatory in animal models[36]. Virgin coconut oil (VCO) is growing in popularity as functional food oil and the public awareness of it is increasing. VCO has been shown to have excellent antioxidant properties[37].

COCONUT OIL AND BODY WEIGHT

Overweight is an important health problem in developed countries, and it is becoming so in India too. Overweight increases the risk for osteoarthritis, diabetes, heart disease, stroke and early death. The usual advice given to overweight individual is to reduce food intake, particularly fat intake. However, on such a diet, the person will feel hungry and feel depressed. In such circumstances, they stop the dieting and end up in rebound weight gaining. Coconut oil has an advantage in these types of settings. Overweight person taking coconut oil, containing medium chain fatty acids, gradually over the months lose weight effortlessly[38,39]. Replacing long chain fatty acids with medium chain fatty acids result in a decrease in body weight and a reduction in fat deposition[40-42]. Coconut-oil-enriched diet was found to be effective in producing a decrease in fat stores[43]. Cleary *et al*[44] found that the safflower oil-fed animals had more fat cells than the coconut oil-fed. It may be a paradoxical finding that overweight person taking coconut oil is losing weight. The explanation is that long chain fats will almost always go into fat stores; this will eventually make the person overweight and will cause dyslipidemia. However, as explained

previously, the medium chain fatty acids will be immediately utilized for energy purposes, and will not be deposited in the body. This will help to reduce weight[45]. Obesity being a great problem, particularly among growing children, this feature of coconut oil may be of immense help in curbing the onset of obesity at a very early age.

CONFLICTS OF INTEREST

None declared

REFERENCES

1. US Department of Health and Human Services: Dietary Guidelines for Americans, 7th edition, 2010". www.dietaryguidelines.gov
2. World Health Organization : Avoiding Heart Attacks and Strokes. 2005
3. Singh RB, Mori H, Chen J, Mendis S, Moshiri M, Zhu S et al. Recommendations for the prevention of coronary artery disease in Asians: a scientific statement of the International College of Nutrition. J Cardiovasc Risk. 1996; 3 (6): 489 - 494.
4. American Heart Association: "Tropical Oils", 2011 http://www.heart.org/HEARTORG/GettingHealthy/FatsAndOils/Fats101/Tropical-Oils_UCM_306031_Article.jsp
5. McNamara DJ: Palm oil and health. A case of manipulated perception and misuse of science. Journal of the American College of Nutrition. 2010; 29(3 Suppl): 240S–244S.
6. Riegel ER, Kent JA. Riegel's Handbook of Industrial Chemistry. Springer. 2003.
7. Foster DW. From glycogen to ketones and back. Diabetes.1984; 33:1188-1199.
8. Guillot E, Vaugelade P, Lemarchal P, Rerat A. Intestinal absorption and liver uptake of medium chain fatty acids in non-anaesthetized pigs. Br J Nutr. 1993; 69: 431-442.
9. Tsuji H, Kasai M, Takeuchi H, Nakamura M, Okazaki M, Kondo K. Dietary medium- chain triglycerides suppress body fat accumulation in a double blind, controlled trial in healthy men and women. J Nutr .2001; 131: 2853-2859.
10. Kurup PA, Rajmohan T. II. Consumption of coconut oil and coconut kernel and the incidence of atherosclerosis. Coconut and Coconut Oil in Human Nutrition, Proceedings. Symposium on Coconut and Coconut Oil in Human Nutrition. Coconut Development Board, Kochi, India, 1995.
11. Mini S, Rajamohan T. Influence of coconut kernel protein on lipid metabolism in alcohol fed rats: Indian Journal of Experimental Biology. 2004; 42: 53-57.
12. Nevin KG, Rajamohan T. Beneficial effects of virigin coconut oil on lipid parameters and in vitro LDL oxidation: Clinical Biochemistry. 2004; 37: 830-835.
13. Sundram K, Hayes KC, Siru OH. Dietary palmitic acid results in lower serum cholesterol than does a lauric-myristic acid combination in normolipemic humans. American Journal of Clinical Nutrition. 1994; 59, 841-846.
14. Prior IA, Davidson F, Salmond CE, Czochanska Z. Cholesterol, coconuts, and diet on Polynesian atolls; a natural experiment: the Pukapuka and Tokelau island studies. American Journal of Clinical Nutrition. 1981; 34: 1552-1556.
15. McWhinney VJ, Pond WG, Mersmann HJ. Ontogeny and dietary modulation of 3-hydroxy-3-methylglutaryl-CoA reductase activities in neonatal pigs. Journal of Animal Science. 1996; 74: 2203-2210.
16. Trautwein EA, Kunath-Rau A, Dietrich J, Drusch S, Erberdobler HF: Effect of dietary fats rich in lauric, myristic, palmitic, oleic or linoleic acid on plasma, hepatic and biliary lipids in cholesterol-fed hamsters. British Journal of Nutrition. 1997; 77: 605-620.
17. Lipoeto NI, Agus Z, Oenzil F, Masrul M, Wattanapenpaiboon N, Wahlqvist ML. Contemporary Minangkabau food culture in West Sumatra, Indonesia. Asia Pac J Clin Nutr.2001; 10: 10-16.
18. Sabitha P, Vaidyanathan K, Vasudevan DM, Kamath P. Comparison of lipid profile and antioxidant enzymes among South Indian men consuming coconut oil and sunflower oil: Indian Journal of Clinical Biochemistry. 2009; 24: 76-81.
19. Hostmark AT, Spydevold O, Eilertsen E. Plasma lipid concentration and liver output of lipoproteins in rats fed coconut fat or sunflower oil. Artery. 1980; 7:367-383.
20. Awad AB. Effect of dietary lipids on composition and glucose utilization by rat adipose tissue. Journal of Nutrition. 1981; 111:34-39.
21. Mensink RP, Zock PL, Kester AD, Katan MB. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. Am. J. Clin. Nutr. 2003; 77 (5): 1146–1155.
22. Sabitha P, Vasudevan DM, Kamath P. Effect of high fat diet without cholesterol

supplementation on oxidative stress and lipid peroxidation in New Zealand White Rabbits: J. Atherosclerosis and Thrombosis. 2010; 17, 213-218.
23. Blackburn GL, Kater G, Mascioli EA, Kowalchuk M, Babayan VK, Bistrian BR. A reevaluation of coconut oil's effect on serum cholesterol and atherogenesis. The Journal of the Philippine Medical Association. 1989; 65:144-152.
24. Willett W. Editorial: Challenges for public health nutrition in the 1990s. American Journal of Public Health.1990; 80:1295-1298.
25. Austin MA, Edwards KL. Small, dense low density lipoproteins, the insulin resistance syndrome and noninsulin-dependent diabetes. Curr Opin Lipidol.1996; 7, 167-171.
26. Sabitha P, Vasudevan DM. Assessment of small dense LDL particles among subjects onsuming coconut oil or sunflower oil as cooking medium by using LDL-cholesterol/LDL-apoB ratio as a surrogate marker: J. Food Biochem, accepted, 2013
27. Felton CV, Crook D, Davies MJ, Oliver MF. Dietary polyunsaturated fatty acids and composition of human aortic plaques. Lancet. 1994; 344:1195-1196.
28. Nevin KG, Rajmohan T. Virgin coconut oil supplemented diet increases the antioxidant status in rats. Food Chem. 2006; 99: 260-266.
29. Kumar PD. The role of coconut and coconut oil in coronary heart disease in Kerala, South India. Trop Doct. 1997; 27: 215-217.
30. Enig MG. Diet, serum cholesterol and coronary heart disease, in Mann GV (ed): Coronary Heart Disease: The Dietary Sense and Nonsense. Janus Publishing, London.1993.
31. Kabara JJ. Inhibition of Staphylococcus aureas in The Pharmacological Effect of Lipids II (JJ Kabara, ed) American Oil Chemists' Society, Champaign IL. 1985.
32. Nevin KG, Rajamohan T. Effect of topical application of virgin coconut oil on skin components and antioxidant status during dermal wound healing in young rats. Skin Pharmacol Physiol. 2010; 23(6):290-297.
33. Gorard DA. Enteral nutrition in Crohn's disease: fat in the formula. Eur J Gastroenterol Hepatol. 2003; 15:115-118.
34. Shea JC, Bishop MD, Parker EM, Gelrud A, Freedman SD. An enteral therapy containing medium-chain triglycerides and hydrolyzed peptides reduces postprandial pain associated with chronic pancreatitis. Pancreatology. 2003; 3:36-40.
35. Rele A, Mohile R. Effect of mineral oil, sunflower oil, and coconut oil on prevention of hair damage. Journal of cosmetic science. 2003; 54: 175–192.
36. Zakaria ZA, Somchit MN, Mat Jais AM, Teh LK, Salleh MZ, Long K. In vivo Antinociceptive and Anti-inflammatory Activities of Dried and Fermented Processed Virgin Coconut Oil. Medical Principles and Practice 2011; 20 (3): 231–236.
37. Marina AM, Che Man YB, Amin I. Virgin coconut oil: emerging functional food oil. Trends in Food Science & Technology. 2009; 20: 481–487.
38. Baba N. Enhanced thermogenesis and diminished deposition of fat in response to overfeeding with diet containing medium-chain triglycerides. American Journal of Clinical Nutrition. 1982; 35, 379.
39. Bach AC, Babayan VK. Medium-chain triglycerides; an update: American Journal of Clinical Nutrition. 1982; 36, 950-962.
40. Bray GA. Weight gain of rats fed medium-chain fatty triglycerides is less than rats fed long chain fatty acids. International Journal of Obstetrics. 1980; 4:27-32.
41. Geliebter A. Overfeeding with medium-chain triglycerides diet results in diminished deposition of fat. American Journal of Clinical Nutrition. 1983; 37, 1-4.
42. Hasihim SA, Tantibhedyangkul P. Medium chain triglycerides in early life; effects on growth of adipose tissue. Lipids. 1987; 22, 429.
43. Portillo MP, Serra F, Simon E, del Barrio AS, Palou A. Energy restriction with high-fat diet enriched with coconut oil gives higher UCP1 and lower white fat in rats. International Journal of Obesity and Related Metabolic Disorders, 1998; 22, 974-979.
44. Cleary MP, Phillips FC, Morton RA. Genotype and diet effects in lean and obese Zucker rats fed either safflower or coconut oil diets: Proceedings of the Society for Experimental Biology and Medicine. 1999; 220, 153-161.
45. Hill JO, Peters JC, Yang D, Sharp T, Kaler M, Abumrad NN et al. Thermogenesis in humans during overfeeding with medium-chain triglycerides. Metabolism. 1989; 38: 641–648.