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THE CEYLON MEDICAL JOURNAL

Established 1887
The Official Publication of the Sri Lanka Medical Association
Volume 51, No. 2, December 2006
Quarterly ISSN 0009-0875

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Coconut fats

Abstract

In many areas of Sri Lanka the coconut tree and its products have for centuries been an integral part of life, and it has come to be called the “Tree of life”. However, in the last few decades, the relationship between coconut fats and health has been the subject of much debate and misinformation.

Coconut fats account for 80% of the fat intake among Sri Lankans. Around 92% of these fats are saturated fats. This has lead to the belief that coconut fats are ‘bad for health’, particularly in relation to ischaemic heart disease. Yet most of the saturated fats in coconut are medium chain fatty acids whose properties and metabolism are different to those of animal origin. Medium chain fatty acids do not undergo degradation and re-esterification processes and are directly used in the body to produce energy. They are not as ‘bad for health’ as saturated fats.

There is the need to clarify issues relating to intake of coconut fats and health, more particularly for populations that still depend on coconut fats for much of their fat intake. This paper describes the metabolism of coconut fats and its potential benefits, and attempts to highlight its benefits to remove certain misconceptions regarding its use.

Introduction

Coconut trees were known to have existed as far back as 161 BC and the existence of coconut plantations was first recorded in the 2nd century AD. Coconut kernel and kernel products such as coconut milk and coconut oil are eaten mainly for the exquisite taste that the fats in coconut convey to food.

Fats make up less than 25% of the total energy expenditure among Sri Lankans. Studies done more than 20–30 years ago have shown that coconut fats constitute about 80% or more of the total fat intake of Sri Lankans [1].

Over 95% of coconut oil is fat, and the fat content of scraped coconut is around 34% and of coconut milk around 24%. It is also true that around 92% of the coconut fat is saturated fat. However, the saturated fats in coconut and palm-kernel, oil also called “tropical oils”, differ from saturated fats in animal fats. Over 50% of the fats in coconut are medium chain triglycerides that are formed from fatty acids of chain length 8:0 to 14:0. When digested in the small intestine, these fats yield saturated medium chain free fatty acids and monoglycerides such as lauric acid (12:0). Of these, it is primarily the 14:0 fatty acids that are thought to be atherogenic. Coconut oil is the highest natural source of lauric acid. Lauric acid and its derivative monolaurin constitute around 50% of coconut fat derived lipid.

However, unlike long chain fatty acids, these medium chain free fatty acids and monoglycerides are absorbed intact from the small intestine, and

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Elsevier
A Division of Reed Elsevier India Pvt. Ltd.
Sri Pratap Udyog, 274, Captain Gaur Marg,
Sriniwaspuri,
New Delhi – 110065, India.
Tel: +91-11-2644 7160-64
Fax: +91-11-2644 7156
Website: www.elsevier.com

Printed by

Ananda Press,
Colombo 13, Sri Lanka
Tel: +94 11 2435975

This journal is indexed in BIOSIS, CAB International, EMBASE, and Index Medicus

do not undergo degradation and re-esterification processes. They are directly used in the body to produce energy, and widely used in infant formulas, nutritional drinks for athletes and intravenous lipid infusions. Since they are not stored in fat deposits, they are of high value to dieters [2]. Animal and human studies have shown that the fast rate of oxidation of medium chain free fatty acids leads to greater energy expenditure. Animal studies have also demonstrated that this greater energy expenditure results in less body weight gain and decreased size of fat depots after several months of consumption. Furthermore, both animal and human trials suggest a greater satiating effect of medium chain triglycerides compared with long chain triglycerides [3]. In fact, administration of medium chain triglycerides in the form of coconut oil along with a saturated fat diet increased oxidation of long chain fatty acids in the body [4].

Coconut fat is also totally devoid of trans-fatty acids which are now thought to be more harmful than saturated fatty acids. By virtue of almost all of its fat being saturated, it is less affected by re-use, a practice which for economic reasons is probably very prevalent in Sri Lanka. Moreover, since it does not contain trans-fatty acids, coconut oil (unlike soybean or corn oil) can be stored in clear bottles. Virgin coconut oil, made from fresh coconut kernel, dried in the sun or under low heat, and the oil expressed by a cold-press technique, also contains poly-phenolic anti-oxidants and is probably better than the commonly available coconut oil that is derived from copra.

Given the fact that the fatty acid composition of the saturated fats in coconut is very different from that of saturated fats from animal sources, how valid is the assumption that coconut fat is bad for the health?

Coconut consumption and lipid patterns in Sri Lanka

A recent study has shown that in Sri Lanka, whereas 91% of the poor still consume coconut as their main source of fat, only 70% of those earning >Rs. 5,000/- use coconut oil. The amount of coconut consumed is less than 5 years ago for 75% of those surveyed [5]. This study also found that one of the reasons for reduced coconut consumption was a misconception that coconut fats are bad. The misconception has arisen due to the fact that coconut fat is mainly saturated fat, and saturated fats are synonymous with atherosclerosis, ischaemic heart disease and cerebrovascular disease.

There remains the question whether coconut fats have actions other than those in relation to the lipid hypothesis of atherosclerosis, ischaemic heart disease and cerebrovascular disease which need to be considered when answering the above question.

To answer that one would have to first look at coconut consumption and possible relationship to the prevalence of ischaemic heart disease and cerebrovascular disease in Sri Lanka. Kaunitz states that the Demographic Yearbook of the United Nations (1978) reported that Sri Lanka has the lowest death rate from ischaemic heart disease. Sri Lanka was quoted as being the only country giving reliable data where coconut oil is the main dietary fat [6]. This was at a time when average coconut consumption was around 130 nuts/person/year. On the other hand, according to Abeywardena, the incidence of ischaemic heart disease and cerebrovascular disease in Sri Lanka is increasing at an alarming rate and is similar to that of the developed world [7]. Central Bank of Sri Lanka reports show that the coconut consumption has now declined to around 100–110 nuts/person/year.

Under the circumstances, are coconut fats the culprit or have they been given a bad name simply by virtue of its fat being >90% saturated fat?

Epidemiologic studies both in Sri Lanka and elsewhere, have failed to clearly establish a relationship between coconut fats, atherosclerosis and ischaemic heart disease. The classic study by Prior among Pacific islanders

whose diet contained large amounts of coconut showed a low prevalence of heart disease, cancer, diabetes and arthritis [8]. Mendis has attributed this to a 'protective effect' of oemga-3 fatty acids from the fish these people ate [1]. Other, more recent studies on small population samples have failed to establish a clear link between consumption of coconut products and atherosclerosis, ischaemic heart disease and cerebrovascular disease [9,10]. Case control studies comparing the diet of patients with ischaemic heart disease and controls have come out both for and against a causative role for coconut fats [11,12].

Coconut fats and lipids

The possible role of coconut fats in disturbances of lipid metabolism is also not well established. Many allopathic medical practitioners seem to think that, as coconut fats are saturated, they elevate plasma lipids in the same manner as the saturated fats from animal sources. Adding to the misconception is the fact that many of the animal studies on the role of coconut fats in lipid metabolism have used hydrogenated coconut oil. This is an unnatural form of coconut oil that is purposely altered to make it completely devoid of any essential fatty acids. The conclusion that can be drawn from such animal research is that feeding hydrogenated coconut oil devoid of essential fatty acids enhances the formation of atherosclerosis markers [13]. However, coconut oil, unlike much of the soybean and corn oil consumed both here and abroad, is free of hydrogenated fat and hence trans-fatty acids. In human feeding studies, coconut fats without doubt elevate high density lipoprotein (HDL) cholesterol. The effect on total cholesterol and low density lipoprotein (LDL) cholesterol is probably neutral. It is also possible that women may react differently to men when fed a coconut diet [14]. A Sri Lankan study found that the risk of coronary heart disease as assessed by the body mass index, ratios of total cholesterol to HDL-cholesterol, and LDL-cholesterol to HDL-cholesterol, was significantly lower in subjects in rural areas, who were agricultural workers with a high degree of physical activity, subsisting on a diet consisting mainly of plant food, despite a higher consumption of coconut, a saturated fat. This study also suggested that hyperlipidaemia is more common among urban dwellers than among the rural population [15]. This difference was attributed to lower fruit, vegetable and fibre content in the diet and lower physical activity among the urban population. Yet from Peiris' study we know the rural poor consume more coconut than city dwellers [5]. Abeywardena has proposed that a low fat diet with predominantly coconut fat may lead to the 'metabolic syndrome' and thus be associated with the increasing prevalence of ischaemic heart disease and cerebrovascular disease [7]. However, this needs to be proven by good epidemiologic studies.

Virgin coconut oil when fed to rats is reported to lower lipid levels in serum and tissues, and LDL oxidation. This property of virgin coconut oil is attributed to the biologically active polyphenol components present in the oil [16]. Other studies have shown that consumption of a solid fat rich in lauric acid gives a more favorable serum lipoprotein pattern than consumption of partially hydrogenated soybean oil rich in trans-fatty acids [17].

Other effects of coconut oil

What of the claim that coconut fats decrease platelet adhesiveness? Hard data are not readily available. A 1993 study comparing a high fat diet (50% of total energy) to a low fat diet (20%) suggested that the high fat diet might increase blood thrombogenicity by virtue of augmented postprandial activation of factor VII [18]. A more recent 2003 study showed that a lesser increase in FVIIa occurred after the consumption of saturated fats, than after unsaturated test fats [19].

Others have attempted to study the effects of individual fatty acids on whole blood aggregation and concluded that compared to oleic acid, lauric, myristic or palmitic acids do not affect in vitro whole blood aggregation induced by collagen ADP-induced aggregation [20]. Compared to a high unsaturated fat or high polyunsaturated fat diet, a coconut oil-based diet lowers postprandial t-PA antigen concentration, and this may favourably affect the fibrinolytic system and the Lp (a) concentration [21].

What of other benefits that may accrue from eating coconut oil? Many readers may not be aware of the close similarity among the medium chain triglycerides in coconut fats, human breast milk and the secretion of sebaceous glands, all rich in lauric acid. Monolaurin and even lauric acid have been shown to be bactericidal, particularly against *Helicobacter pylori*, *Vibrio cholerae*, *Salmonella typhi*, *Shigella sonnei* and enterotoxigenic *Escherichia coli* [22]. Coconut oil also helps the body to increase absorption of calcium and magnesium ions. It has been suggested that coconut oil is used to supplement treatment of rickets in poorly developed countries, alongside infant formulae supplemented with these inorganic ions [2]. No work has been done with coconut fats per se. However, it is interesting to speculate whether monolaurin and lauric acid released by pre-gastric lipase may contribute to the reported low incidence of *Helicobacter pylori* infection in Sri Lanka compared to other south Asian countries [23].

Current understanding based on the effect of dietary lipid manipulation upon immune system function indicates that fatty acids are involved in the modulation of the immune response through complex pathways. The problem with many animal studies is that they use hydrogenated coconut oil. One study where non-hydrogenated coconut oil was used showed that lipopolysaccharide-stimulated TNF-alpha production by macrophages decreased with increasing unsaturated fatty acid content of the diet [24]. There are anecdotal claims to the effect that using coconut oil improves the action of anti-HIV drugs. Many other unsubstantiated

claims on the benefits of coconut products such as its anti-oxidant effects etc, are gaining popularity among health food enthusiasts.

A plea for further research

Clearly more work, both epidemiological and experimental, needs to be done to establish whether coconut fats are indeed associated with atherogenesis, ischaemic heart disease and cerebrovascular disease. Population studies should attempt to remove the possible effect of other confounding variables such as increased consumption of animal fats along with coconut, smoking, alcohol, and in the Sri Lankan context, illicit brews that could contribute to the increasing incidence of ischaemic heart disease and cerebrovascular disease in Sri Lanka. In the absence of convincing evidence against the continued use of coconut fats in relation to atherosclerosis and ischaemic heart disease, one has to ask whether the recommendation of the Asian Heart Assoication issued in 1996, "An intake of 400 g/day of fruit, vegetables and legumes, mustard or soybean oil (25 g/day) instead of hydrogenated fat, coconut oil or butter, in conjunction with moderate physical activity (1255 kJ/day), cessation of tobacco consumption and moderation of alcohol intake may be an effective package of remedies for prevention of coronary artery disease in Asians" is valid in toto [25]. Should we be encouraging the use of mustard, soybean, corn or sunflower oils all of which will need to be imported, or emphasize the need for eliminating smoking, excessive use of alcohol, consumption of animal fats and other life style changes instead?

Finally, one is left with the question—given the importance of coconut not only as a major dietary component in our diet but also because of its export potential, why is it that there has been little or no investment in research in Sri Lanka on the health effects of coconut products?

References

1. Mendis S. Heart attacks: exploring new preventive strategies. *Ceylon Medical Journal* 1998; **43**: 210–8.
2. Pehowich DJ, Gomes AV, Barnes JA. Fatty acid composition and possible health effects of coconut constituents. *West Indian Medical Journal* 2000; **49**: 128–33.
3. St-Onge MP, Jones PJ. Physiological effects of medium-chain triglycerides: potential agents in the prevention of obesity. *American Society for Nutritional Sciences Journal Nutrition* 2002; **132**: 329–33.
4. Papamandjaris AA, White MD, Raeini-Sarjaz M, Jones PJ**.** Endogenous fat oxidation during medium chain versus long chain triglyceride feeding in healthy women. *International Journal of Obesity Related Metabolic Disorders* 2000; **24**: 1158–66.
5. Peiris TSG, Fernando MTN, Samarajeeva S. Factors influencing the use of coconut oil by householders in Sri Lanka and their policy relevance to popularize the consumption of coconut oil. *Cord* 2004; **20**: 12–20.
6. Kaunitz HJ. Medium chain triglycerides (MCT) in aging and arteriosclerosis. *Environmental Pathology, Toxicology and Oncology* 1986; **6**: 115–21.
7. Abeywardena MY. Dietary fats, carbohydrates and vascular disease: Sri Lankan perspectives. *Atherosclerosis* 2003; **171**: 157–61.
8. Prior I.A, Davidson F. Cholesterol, coconuts, and diet on Polynesian atolls: a natural experiment: the Pukapuka and Tokelau island studies. *American Journal of Clinical Nutrition* 1981; **34**: 1552–61.
9. Lindeberg S, Berntorp E, Nilsson-Ehle P, Terent A, Vessby B. Age relations of cardiovascular risk factors in a traditional Melanesian society: the Kitava Study. *American Journal of Clinical Nutrition* 1997; **66**: 845–52.
10. Lipoeto NI, Agus Z, Oenzil F, Wahlqvist M, Wattanapenpaiboon N. Dietary intake and the risk of coronary heart disease among the coconut-consuming Minangkabau in West Sumatra, Indonesia. *Asia Pacific Journal of Clinical Nutrition* 2004; **13**: 377–84.
11. Kumar PD. The role of coconut and coconut oil in coronary heart disease in Kerala, South India. *Tropical Doctor* 1997; **27**: 215–7.
12. Zhang J, Kesteloot H. Differences in all-cause, cardiovascular and cancer mortality between Hong Kong and Singapore: role of nutrition. *European Journal Epidemiology* 2001; **17**: 469–77.
13. Garcia-Fuentes E, Gil-Villarino A, Zafra MF, Garcia-Peregrin E. Influence of fasting status on the effects of coconut oil on chick plasma and lipoprotein composition. *Journal of Physiological Biochemistry* 2003; **59**: 101–10.
14. Cox C, Mann J, Sutherland W, Chisholm A, Skeaff M. Effects of coconut oil, butter, and safflower oil on lipids and lipoproteins in persons with moderately elevated cholesterol levels. *Journal of Lipid Research* 1995; **36**: 1787–95.
15. Atukorala TM, Jayawardene MI. Lipid patterns and dietary habits of healthy subjects living in urban, suburban and rural areas. *Ceylon Medical Journal* 1991; **36**: 9–16.
16. Nevin KG, Rajamohan T. Beneficial effects of virgin coconut oil on lipid parameters and in vitro LDL oxidation. *Clinical Biochemistry* 2004; **37**: 830–5.
17. de Roos N, Schouten E, Katan M. Consumption of a solid fat rich in lauric acid results in a more favorable serum lipid profile in healthy men and women than consumption of a solid fat rich in trans-fatty acids. *Journal of Nutrition* 2001; **131**: 242–5.
18. Marckmann P, Sandstrom B, Jespersen J. Dietary effects on circadian fluctuation in human blood coagulation factor VII and fibrinolysis. *Atherosclerosis* 1993; **101**: 225–34.
19. Tholstrup T, Miller GJ, Bysted A, Sandstrom B. Effect of individual dietary fatty acids on postprandial activation of blood coagulation factor VII and fibrinolysis in healthy young men. *American Journal of Clinical Nutrition* 2003; **77**: 1125–32.

20. Temme EH, Mensink RP, Hornstra G. Comparison of the effects of diets enriched in lauric, palmitic, or oleic acids on serum lipids and lipoproteins in healthy women and men. *American Journal of Clinical Nutrition* 1996; **63**: 897–903.

21. Muller H, Lindman AS, Blomfeldt A, Seljeflot I, Pedersen JI. A diet rich in coconut oil reduces diurnal postprandial variations in circulating tissue plasminogen activator antigen and fasting lipoprotein (a) compared with a diet rich in unsaturated fat in women. *Journal of Nutrition* 2003; **133**: 3422–7.

22. Bergsson G, Steingrimsson O, Thormar H. Bactericidal effects of fatty acids and monoglycerides on *Helicobacter pylori*. *International Journal of Antimicrobial Agents* 2002; **20**: 258–62.

23. de Silva M. Prevalence of *Helicobacter pylori* infection in patients with functional dyspepsia. *Ceylon Medical Journal* 1999; **44**: 118–9.

24. de Pablo MA, Ortega E, Gallego AM, Alvarez C, Pancorbo PL, et al. The effect of dietary fatty acid manipulation on phagocytic activity and cytokine production by peritoneal cells from Balb/c mice. *Journal of Nutritional Science and Vitaminology (Tokyo)* 1998; **44**: 57–67.

25. Singh RB, Mori H, Chen J, Mendis S, Moshiri M, et al. Recommendations for the prevention of coronary artery disease in Asians: a scientific statement of the International College of Nutrition. *Journal of Cardiovascular Risk* 1996; **3**: 489–94.

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