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
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.

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 fat is also very different from that of saturated fats from animal sources, since it contains medium chain triglycerides [3]. In fact, administration of medium chain triglycerides results in 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 significantly different from that of saturated fats from animal sources, since 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 omega-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 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

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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 Association 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

Selection of students to medical school

Selection criteria must be based on valid predictors of performance in medical school

A recent leading article in the Ceylon Medical Journal that deals with an important policy issue emphasizes that the ultimate goal of selection of students to a medical school must be to identify those who are likely to contribute towards meeting the health needs of society [1]. In discussing the pros and cons of the district quota system used for the selection of medical students in Sri Lanka since the 1970s, this leading article mentions the need for studies that examine the correlation between admission criteria and medical school performance.

We have recently carried out several such studies. The first showed that although the raw aggregate score from the GCE Advanced Level examination did show a statistically significant correlation with outcome (three summative examinations conducted in the medical school) at Ragama, it was a weak predictor of performance [2]. The second study, which extended the scope of the analysis to all six medical schools in the country, confirmed this finding [3]. It also showed that students who sat the A Level examination as school candidates (in their 1st or 2nd attempt at the examination) did much better in medical school than private candidates (who were probably in their 3rd attempt).

The leading article [1] also stresses the need for subject-wise standardisation, with Z-scores, rather than raw aggregate scores, as the basis for selection. This change came into effect in Sri Lanka in 2000, along with a change of subjects. The first intake of students selected according to Z-scores has not yet graduated from medical school, so a comprehensive analysis is not possible. However, we have just completed a correlational study similar to the two mentioned previously, using performance at the first summative examination in medical school as the outcome measure.

The study sample comprised medical students in Colombo (n=299) and Ragama (n=196), entering university on the basis of their A Level examination results in 2001 (n=195) and 2002 (n=300). 70.1% of these students passed the first examination in medical school at the first attempt; 35.6% with honours. We examined the predictive value of the Z-score in determining examination outcome, using logistic regression in a model very similar to that described previously [3]. We found that although the Z-score was significantly associated with exam outcome, both for passing and obtaining honours, it explained only 4% of the variation in outcome in passing the examination at first attempt, and 5% of the variation in obtaining honours. The equivalent values for the aggregate score were 2% and 7% [3]. Thus it appears that the change from using raw aggregate scores to Z-scores may not have improved matters a great deal for selecting better medical students, perhaps because the subject combination has remained unchanged. Also, valid use of the Z-score depends on the assumption that


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