



## Invited Commentary

# Commentary on 'Influence of virgin coconut oil-enriched diet on the transcriptional regulation of fatty acid synthesis and oxidation in rats – a comparative study' by Sakunthala Arunima and Thankappan Rajamohan

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For decades, dietary saturated fats have been synonymous with atherogenic diets, in part, due to their serum cholesterol-raising effect compared with PUFA. Saturated fats were initially equated with ruminant animal fats, specifically dairy and beef, and later with fats from pork produced from pigs fed saturated fat diets and contrasted with PUFA-rich vegetable oils. Later, the concept of 'tropical oils' joined animal fats, specifically coconut oil and palm kernel oil (the latter not to be confused with palm oil). Dozens of studies in rats and rabbits produced concordant data, showing that both these plant fat sources dramatically increase cholesterol levels in the liver and serum so reliably that they are routinely used as positive controls in comparative animal feeding studies. Adverse increases in serum cholesterol levels were found after ingestion of these saturated oils whether they were hydrogenated or not, showing that the major artifactual *trans*-monoene in hydrogenated oil, elaidic acid, was not the primary cause, a hypothesis recently confirmed<sup>(1)</sup>.

A serious inconsistency with the atherogenic saturated fat hypothesis was found with highly saturated cocoa butter, a tropical oil without atherogenic properties, which led to studies indicating that stearic acid is rapidly desaturated to oleic acid and behaves more like a monoene<sup>(2)</sup>. Though 40% of cocoa butter is the presumed atherogenic palmitic acid (16:0), chocolate is now considered anti-atherogenic, a property ascribed to its phytochemical content, which, apparently, is generally assumed to overcome its saturated fat character. Moreover, where they are produced and widely consumed for centuries, staple tropical oils and the plants that produce them have enjoyed a much different reputation. Criticism of coconut and its oil by Western nutritional science has been widespread, boarding on frank puzzlement at the reputation of this staple food in many tropical and subtropical countries such as India, The Philippines, Indonesia and Sri Lanka. Indeed, early epidemiology (in the 1970s) of Sri Lankans whose lipid intake is 80% coconut fat via fresh coconut supports low, not high, incidence rates of CVD<sup>(3)</sup>.

Coconut oil nearly disappeared from the Western food supply in the latter 1990s, but has recently made a stunning comeback as a favourite in natural and organic health food stores, at least in the USA. Although the fatty acid profile of coconut oil has not changed, the method of production has. In particular, virgin coconut oil is produced from fresh coconut under mild conditions with gentle heating and no solvent extraction or harsh chemical treatment to obtain a flavourful product. In contrast, the industrial method of refined–deodorised–bleached (RDB) coconut oil production involves solvent extraction of dried coconut (known as 'copra'), extended heating of the oil above 200°C to drive off volatiles, and bleaching, yielding a bland, white fat suitable for use as a shortening in many processed foods. It is versions of the latter product, RDB coconut oil, that cause changes in lipid profiles associated with atherogenesis.

In this issue of the *British Journal of Nutrition*, Arunima & Rajamohan<sup>(4)</sup> of the University of Kerala in South India present a head-to-head comparison of the influence of virgin coconut oil and copra oil on fatty acid oxidation and lipid parameters in rats. Serum and liver cholesterol and TAG levels were dramatically reduced in the virgin coconut oil-fed group compared with the copra oil-fed group and compared with the oleic acid-rich olive oil-fed group and even the linoleic acid-rich sunflower oil-fed group. Mitochondrial and peroxisomal fatty acid oxidation was highest in the virgin coconut oil-fed group, as determined by direct measurement, and consistent with relevant gene expression. The fatty acid profiles of copra oil and virgin coconut oil were identical and thus any difference in their effects must be due to other components. The authors suggest that lower levels of polyphenols and other antioxidants in copra oil compared with virgin coconut oil may be responsible for the differential effects, as has been also suggested by others. The generation of bioactive components during processing of copra oil that mediate adverse effects on lipid metabolism is also plausible.

Notably, saturate-rich virgin coconut oil resulted in lower serum cholesterol levels compared with linoleate-rich

sunflower oil. PUFA, in general, and linoleate, in particular, lower serum cholesterol levels when substituted for monounsaturates and indeed in the present study resulted in lower mean serum cholesterol levels compared with olive oil. However, systematic reviews of human studies conclude that substitution of unspecified PUFA for saturates results in lower serum cholesterol levels, which is not consistent with the results of the present study showing that saturate-rich coconut oil may result in both higher, and lower, serum cholesterol levels compared with sunflower oil. That is, the results of the present study indicate that factors other than the relative sums of saturates or PUFA control serum cholesterol levels. We note, for completeness, that key human randomised controlled trial data appearing in the *British Medical Journal* show that higher linoleic acid intake lowers serum cholesterol but increases CVD<sup>(5)</sup>, calling into question the mechanistic relationship between serum cholesterol and CVD, at least at moderate serum cholesterol levels widely found in Western populations.

Arunima and Rajamohan's study adds to the evidence for a re-evaluation of the nearly universal presumption that saturated fats are mechanistically atherogenic compared with monounsaturated or polyunsaturated fats. At least equally plausible and more consistent with the available basic preclinical science such as the present study is that the totality of oil composition, in large part determined by production methods, is the key determinant of the healthfulness of any particular oil.

Both authors report no conflicts of interest.

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