

Coconut oil and cardiovascular health-Do we have enough clinical evidence?

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The debate on saturated fats started in the early 1950s and is still continuing. The fats present in coconut oil are saturated fats but is different from long chain fatty acids. Coconut oil and coconut is used widely in some parts of the country where the incidence of coronary artery disease is high compared to other states. As per the present practice, most of the physicians advice to avoid coconut oil for dietary needs and all patients are doubtful about the continuous usage of coconut oil after their cardiac event. The medium chain fatty acids [MCA] metabolism is different from the long chain fatty acids present in other fats and oil. There are only epidemiological data and small short term interventions with coconut oil and its association with CAD. Previous works of Amrita institute of Medical Sciences have proved that there were no difference in lipid profile (serum total cholesterol, triacylglycerols, and cholesterol in lipoprotein fractions) between persons taking coconut oil or sunflower oil. Higher intake of coconut oil did not cause any significant increase in the concentration of lauric acid in blood among coconut oil consumers. Moreover, serum lipid values did not show significant variation between animals (New Zealand white rabbits) fed coconut oil or sunflower oil. Coconut oil intake did not cause hypercholesterolemia or oxidative stress in rabbits. In another study, the fatty acid content of the coronary plaque (endarterectomy specimen) did not show any difference between coconut oil consumers versus sunflower oil consumers. Since these studies were done in free living subjects many confounding factors like eating outside, quantity of oil, duration of consumption and physical activity could not be assessed correctly. Another large study involving 200 coronary artery disease patients on medical treatment with two year long follow up did not record any significant difference in anthropometry, lipid profile, vasomotor function, antioxidant levels and cardiac events at the end of the second year.

The main limitation of the studies involving human are the lack of control over other nutrients which affect the serum lipids positively or negatively, non dietary factors like physical activity, life styles and the genomic factors

are likely to influence the outcomes. Most of the physician driven small clinical studies included specialized patient population like coronary artery diseases or patient with diabetes mellitus. Few of the data in humans using medium chain triglyceride are controversial as the commercially available MCT is different as it contain very small amount of lauric acid which is the main content in coconut oil and hence it is difficult to extrapolate these results with coconut oil studies. Small duration intervention with either coconut oil or virgin coconut oil is insufficient to test the hard outcome of cardiovascular system as it requires long time for the metabolites to produce favorable or unfavorable effect.

VCO is not tested in long term human clinical trials or observational studies most importantly the clinical hard end points death, myocardial infarction and stroke were not considered as end point in these studies. Many animal studies are available on coconut oil and VCO from many countries. Even though few studies showed favorable effect on lipids and oxidation redox potential, it is difficult to equate these results with human as the metabolism of fats is complex in humans and is modified by the genetic makeup.

To have clarity on coconut oil's effect (as dietary) on cardiovascular outcome, we need a long term longitudinal follow up study of a cohort or families without cardiovascular diseases or other co morbidities. Globally accepted clinical as well as biochemical outcomes should be monitored periodically. Coconut oil can be compared with any of the other commonly used oil and different ethnic population should be included in the study to assess genomic and epigenomic influences. Such a study will definitely help the medical, scientific, heart-health, governmental and intergovernmental and professional authorities to formulate the dietary recommendations.

Another area that can be explored is the HDL rising property of coconut oil. HDL is a complex lipoprotein and rather than its quantity the function of cholesterol transfer is important in cardiovascular protection. The property of the high HDL achieved by use of coconut oil can be studied. To have a definite and permanent basis for

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the argument that coconut oil has no cholesterol rising property, the metabolism of C12 from the main fatty acid lauric acid can be traced by nucleotide scanning

The beneficial effect of either oil in specific medical conditions (therapeutic effect) are explored but not in a systematic way. The disease conditions which are likely to be benefited from the use of either oil should be

identified using standard diagnostic criteria. A hypothesis on the mechanism by which coconut oil improves the disease pathology will help to search the mechanism of action and to define the clinical and laboratory criteria of treatment benefit. Finally it should undergo randomized multi centric clinical trials to have substantial level of evidence ■
