Coconut oil and the diet/heart hypothesis

For the past several decades you have heard about animal and human studies feeding coconut oil that purportedly showed increased indices for cardiovascular risk. Blackburn et al (1988) have reviewed the published literature of "coconut oil's effect on serum cholesterol and atherogenesis" and have concluded that when "...[coconut oil is] fed physiologically with other fats or adequately supplemented with linoleic acid, coconut oil is a neutral fat in terms of atherogenicity." The question then is, how did coconut oil get such a negative reputation The answer quite simply is, initially, the significance of those changes that occurred during animal feeding studies were misunderstood. The wrong interpretation was then repeated until ultimately the misinformation and disinformation took on a life of its own.

The problems for coconut oil started four decades ago when researchers fed animals hydrogenated coconut oil that was purposefully altered to make it completely devoid of any essential fatty acids. The hydrogenated coconut oil was selected instead of hydrogenated cottonseed, corn or soybean oil because it was a soft enough fat for blending into diets due to the presence of the lower melting medium chain saturated fatty acids. The same functionality could not be obtained from the cottonseed, corn or soybean oils if they were made totally saturated, since all their fatty acids were long chain and high melting and could not be easily blended nor were they as readily digestible. The animals fed the hydrogenated coconut oil (as the only fat source) naturally became essential fatty acid deficient; their serum cholesterol levels increased. Diets that cause an essential fatty acid deficiency always produce an increase in serum cholesterol levels as well as an increase in the atherosclerotic indices. The same effect has also been seen when other essential fatty acid deficient, highly hydrogenated oils such as cottonseed, soybean or corn oils have been fed; so, it is clearly a function of the hydrogenated product, either because the oil is essential fatty acid (EFA) deficient or because of trans fatty acids (TFA).

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