

Editorial

Dietary fat and cardiovascular disease?

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Dietary saturated fat (SF) intake has been shown to increase low density lipoprotein (LDL) cholesterol and therefore has been associated with increased risk of cardiovascular disease (CVD). This evidence coupled with inferences from epidemiologic studies and clinical trials, had led to longstanding public health recommendations for limiting SF intake as a means of preventing CVD. However the relationship between SF and CVD risk remains controversial, due at least in part to the intrinsic limitations of clinical studies that have evaluated this relationship.⁽¹⁾ A recent meta analysis showed that current evidence does not clearly support cardiovascular guidelines that encourage high consumption of polyunsaturated fatty acids (PUFA) and low consumption of total SF.⁽²⁾ They found weak positive associations between circulating palmitic and stearic acids (found largely in palm oil and animal fats, respectively) and CVD, whereas circulating margaric acid (a dairy fat) significantly reduced the risk of CVD.^(2,3) Saturated fat are not associated with all cause mortality, CVD, CHD, ischemic stroke or type 2 diabetes, but the evidence is heterogenous with methodological limitations.⁽⁴⁾

Dietary lipids are important regulators of cardiac function through their role in membrane phospholipids, as signaling molecules and ligands for nuclear receptors, and as the predominant oxidative substrate for cardiac mitochondria. Moreover, long chain n3 PUFA decrease very low density lipoprotein assembly and secretion reducing triacylglycerol

production. Eicosanoids derived from n6 PUFA have proinflammatory actions, while eicosanoids from n3 PUFA have anti inflammatory ones. Previous studies showed that inflammation contributes to both the onset and progression of atherosclerosis actually, atherosclerosis is predominantly a chronic low grade inflammatory disease of the vessel wall.⁽⁵⁾ Studies in rodents show that in the absence of obesity, replacing refined carbohydrate with fat can attenuate or prevent ventricular expansion and contractile dysfunction in response to hypertension, infarction or genetic cardiomyopathy. Clinical studies generally support high intake of n 3 PUFA from marine sources to prevent and treat heart failure. This effect is associated with decreased inflammation and improved resistance to mitochondrial permeability transition.⁽⁶⁾

Dietary guidelines must carefully consider the health effect of recommendations for alternative macronutrient to replace SF.⁽³⁾ Saturated fat intake of around 20 g/day (approximately 10% of total energy) may be optimal, which corresponds to 200 g of milk a day and 150 g of meat every other day.⁽⁷⁾ Epidemiologic studies and randomized clinical trials have provided consistent evidence that replacing SF with PUFA but not carbohydrates is beneficial for CHD.⁽¹⁾ In practice, reducing red meat and dairy products in a food supply and increasing intakes of nuts, fish, soy products and non hydrogenated vegetables oils will improve the mix of FA and have a markedly beneficial effects on rates of CHD.⁽⁴⁾



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