

Eating the Right Kind of Fats

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Despite the wide belief that saturated fat intake is associated with the risk of coronary heart disease, authors from a recent study published in the *Annals of Internal Medicine*, analyzed data from 72 unique studies with over 600,000 participants from 18 nations and concluded: "current evidence does not clearly support cardiovascular guidelines that encourage high consumption of polyunsaturated fatty acids and low consumption of total saturated fats [1]." Other recent studies have similarly created doubt in the link between dietary saturated fat and heart disease. A study published in the *American Journal of Clinical Nutrition*, which evaluated this association, does not support the notion that saturated fats increase the risk of coronary heart disease, stroke, or peripheral vascular disease [2]. Another study published in the same journal found that the replacement of saturated fats with high glycemic index carbohydrates significantly increases the risk of heart attacks [3]. These recent findings suggest that the dietary intake of butter and animal fats is not as bad as previously believed.

The hypothesis that saturated fats raise serum cholesterol levels and high blood cholesterol causes obstructive plaques in arteries (atherosclerosis) was based on the early research of Nikolaj Anitschkow (1885-1964). In 1913 Anitschkow fed rabbits high doses of animal fat and showed that cholesterol caused atherosclerotic changes in the rabbits' arterial intima like that seen with human atherosclerosis [4]. Over several decades, other investigators also did atherosclerosis research on cholesterol-fed rabbits, which they cited in support of the diet-cholesterol theory of heart disease. Some skeptics contend that rabbits, being herbivores, are not designed to digest animal fat and cholesterol, so when they are fed high doses of cholesterol, they are not able to handle its disposal like other animals, and the cholesterol gets stuck in the walls of their blood vessels. However, studies have shown that if the level of cholesterol rises high enough, all animal species are capable of inducing atherosclerotic lesions. Other evidence in the support of the fat hypothesis of heart disease came in 1953 from the investigation of Dr. Ancel Keys. Keys obtained the dietary data and vital statistics of men in 6 countries and compared the total deaths from all forms of heart disease with the percentage of fat calories consumed in those countries, as derived from data supplied by the United Nations. He concluded: "dietary fat somehow is associated with cardiac disease mortality, at least in middle age [5]." In an effort to validate this conclusion, Yerushalmy and Hilleboe [6] reviewed data from 22 countries, not just the 6 that were selected by Keys, and found that the suggested association between the 2 variables was present but not as compelling as in Keys' more narrow selection of countries. In the late 1950s, Keys embarked on his extraordinary long-term Seven Countries Study in which he reported an association between blood cholesterol level and coronary heart disease. The study data has elicited intriguing conclusions and controversy. Interestingly, dietary studies of indigenous people including Maasai (tribes in Tanzania), Inuits (tribes in North America), Rendille (tribes in Kenya), and Tokelau (tribes in New Zealand) revealed diets comprised of 60-75% saturated fats, but the incidence of coronary heart disease in these tribes is dramatically low. Furthermore, lower rates of heart disease are associated with a higher intake of saturated fats in the diets of people from Austria, Finland, Belgium, Iceland, Netherlands, Switzerland, and France, whereas higher rates of heart disease are associated with a lower intake of saturated fats in people from Georgia, Tajikistan, Azerbaijan, Moldova, Croatia, Macedonia, and Ukraine [7]. This data suggest an

inverse correlation with saturated fat consumption and the rate of heart disease. In keeping with dietary guidelines designed to prevent cardiovascular disease (CVD), saturated fatty acid consumption in the US has drastically decreased since 1980, and the consumption of polyunsaturated fatty acids and carbohydrates has increased. Ironically, since 1980 there has also been a dramatic increase in the prevalence of CVD, obesity, insulin resistance, and diabetes. In fact, a study from the 1990s showed that dietary polyunsaturated fatty acids directly influence aortic plaque formation, and no such associations were found with saturated fatty acids [8]. Earlier studies had suggested reconsidering the effort to increase polyunsaturated fatty acids in the diet [9,10], but these suggestions were largely ignored. Furthermore, the evidence that dietary saturated fat elevates LDL cholesterol, and thus the risk of CAD, is not strong [11]. Other studies have shown that lowering saturated fat intake by dietary intervention does not lower coronary disease or total mortality [12].

Saturated fat, although cast as a villain, has many important biological activities. For example, butyric acid (4:0), the shortest saturated fatty acid, is a well-known modulator of genetic regulation [13,14], and it also plays a role in cancer prevention [15]. The medium chain fatty acids, including caprylic acid (8:0) and capric acid (10:0), exhibit antiviral activity against HIV [10,16]. Lauric acid (12:0), also a medium chain fatty acid, demonstrates antibacterial [17], antiviral [18], and antiplaque activities [19], whereas consumption of the long-chain stearic acid (18:0) lowers thrombogenic and atherogenic risk factors [20]. In addition, saturated fatty acids are the preferred energy substrate for the heart [21] and play a role in protecting cells. Cell membranes contain nearly 50% long-chain fatty acids [22]. The major fatty acid in the lung (a 16-carbon-long saturated fatty acid) is a major component of the lipoprotein complex of surfactants, which play an essential role in preventing asthma and other breathing disorders [23]. Saturated fats also protect against the adverse effects of alcohol in the liver [24].

Similarly, polyunsaturated and monounsaturated fatty acids have many important biological activities, too, particularly omega-3 polyunsaturated fatty acids, which have been regarded as heart healthy, as shown by our own research [25-27]. During cooking at high temperatures, however, saturated fatty acids are more stable than mono- or polyunsaturated fatty acids. Unsaturated fatty acids generate toxic products from their oxidative degradation and hydrogenation when they are heated at high temperatures [28-30]. Trans fatty acids, strongly associated with cardiovascular disease, are the hydrogenated byproducts of unsaturated fatty acids [31]. The trans fats are commonly present in bakery products, snacks, imitation

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cheese, and processed foods and are banned in most of the European countries. Our research has highlighted some of the mechanisms by which trans fats affect cardiovascular diseases [32-34]. Perhaps some of the complications associated with fats can be avoided by selecting an appropriate source of fat based on its use. For example, coconut oil, palm oil, butter, tallow and lard are better for high-heat cooking such as frying and baking. Avocado oil, macadamia nut oil, olive oil, peanut oil, and rice bran oil are good for low-heat cooking. Safflower oil, sesame seed oil, canola oil, sunflower oil, corn oil, and soybean oil should be avoided for cooking, as they are very unstable when heated at high temperatures. In conclusion, choosing the right fat rather than replacing the carbohydrate intake over the fat intake may be the key to avoiding heart disease. It may be time for the regulatory agencies to reconsider the fat intake recommendation to reverse the upward trend in obesity and metabolic syndrome. Lipidomics data also needs to be generated for non-enzymatic oxidative products from saturated, monounsaturated, and polyunsaturated fatty acid degradation to determine their toxic effects in humans.

References

- Chowdhury R, Warnakula S, Kunutsor S, Crowe F, Ward HA, et al. (2014) Association of dietary, circulating, and supplement fatty acids with coronary risk: a systematic review and meta-analysis. *Ann Intern Med* 160: 398-406.
- Siri-Tarino PW, Sun Q, Hu FB, Krauss RM (2010) Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease. *Am J Clin Nutr* 91: 535-546.
- Jakobsen MU, Dethlefsen C, Joensen AM, Stegger J, Tjønneland A, et al. (2010) Intake of carbohydrates compared with intake of saturated fatty acids and risk of myocardial infarction: importance of the glycemic index. *Am J Clin Nutr* 91: 1764-1768.
- (1983) Classics in arteriosclerosis research: On experimental cholesterol steatosis and its significance in the origin of some pathological processes by N. Anitschkow and S. Chalutow, translated by Mary Z. Peliass, 1913. *Arteriosclerosis* 3: 178-182.
- KEYS A (1953) Atherosclerosis: a problem in newer public health. *J Mt Sinai Hosp N Y* 20: 118-139.
- Yerushalmy J, Hilleboe He (1957) Fat in the diet and mortality from heart disease; a methodologic note. *N Y State J Med* 57: 2343-2354.
- Petersen S, Peto V, Rayner M, Leal J, Luengo-Fernandez R, et al. (2005) European cardiovascular disease statistics.
- Felton CV, Crook D, Davies MJ, Oliver MF (1994) Dietary polyunsaturated fatty acids and composition of human aortic plaques. *Lancet* 344: 1195-1196.
- Pinckney ER (1973) The potential toxicity of excessive polyunsaturates. Do not let the patient harm himself. *Am Heart J* 85: 723-726.
- West CE, Redgrave TG (1974) Reservations on the use of polyunsaturated fats in human nutrition. *Search* 5: 90.
- Ravnskov U (1998) The questionable role of saturated and polyunsaturated fatty acids in cardiovascular disease. *J Clin Epidemiol* 51: 443-460.
- Hooper L, Summerbell CD, Higgins JP, Thompson RL, Capps NE, et al. (2001) Dietary fat intake and prevention of cardiovascular disease: systematic review. *BMJ* 322: 757-763.
- Smith JG, German JB (1995) Molecular and genetic effects of dietary derived butyric acid. *Food Technol* 49: 87-90.
- Smith JG, Yokoyama WH, German JB (1998) Butyric acid from the diet: actions at the level of gene expression. *Crit Rev Food Sci Nutr* 38: 259-297.
- German JB (1999) Butyric acid: a role in cancer prevention. *Nutr Bull* 24: 293-299.
- Neyts J, Kristmundsdóttir T, De Clercq E, Thormar H (2000) Hydrogels containing monocaprin prevent intravaginal and intracutaneous infections with HSV-2 in mice: impact on the search for vaginal microbicides. *J Med Virol* 61: 107-110.
- Dawson PL, Carl GD, Acton JC, Han IY (2002) Effect of lauric acid and nisin-impregnated soy-based films on the growth of *Listeria monocytogenes* on turkey bologna. *Poult Sci* 81: 721-726.
- Sun CQ, O'Connor CJ, Robertson AM (2003) Antibacterial actions of fatty acids and monoglycerides against *Helicobacter pylori*. *FEMS Immunol Med Microbiol* 36: 9-17.
- Schuster GS, Dirksen TR, Ciarlone AE, Burnett GW, Reynolds MT, et al. (1980) Anticaries and antiplaque potential of free-fatty acids in vitro and in vivo. *Pharmacol Ther Dent* 5: 25-33.
- Kelly FD, Sinclair AJ, Mann NJ, Turner AH, Abedin L, et al. (2001) A stearic acid-rich diet improves thrombogenic and atherogenic risk factor profiles in healthy males. *Eur J Clin Nutr* 55: 88-96.
- Lawson LD, Kummerow FA (1979) beta-Oxidation of the coenzyme A esters of elaidic, oleic, and stearic acids and their full-cycle intermediates by rat heart mitochondria. *Biochim Biophys Acta* 573: 245-254.
- Abbott SK, Else PL, Atkins TA, Hulbert AJ (2012) Fatty acid composition of membrane bilayers: importance of diet polyunsaturated fat balance. *Biochim Biophys Acta* 1818: 1309-1317.
- Cockshutt AM, Absolom DR, Possmayer F (1991) The role of palmitic acid in pulmonary surfactant: enhancement of surface activity and prevention of inhibition by blood proteins. *Biochim Biophys Acta* 1085: 248-256.
- Ronis MJ, Korourian S, Zippeman M, Hakkak R, Badger TM (2004) Dietary saturated fat reduces alcoholic hepatotoxicity in rats by altering fatty acid metabolism and membrane composition. *J Nutr* 134: 904-912.
- Siddiqui RA, Harvey KA, Ruzmetov N, Miller SJ, Zaloga GP (2009) n-3 fatty acids prevent whereas trans-fatty acids induce vascular inflammation and sudden cardiac death. *Br J Nutr* 102: 1811-1819.
- Siddiqui RA, Harvey KA, Zaloga GP (2008) Modulation of enzymatic activities by n-3 polyunsaturated fatty acids to support cardiovascular health. *J Nutr Biochem* 19: 417-437.
- Zaloga GP, Ruzmetov N, Harvey KA, Terry C, Patel N, et al. (2006) (N-3) long-chain polyunsaturated fatty acids prolong survival following myocardial infarction in rats. *J Nutr* 136: 1874-1878.
- Marnett LJ (1999) Lipid peroxidation-DNA damage by malondialdehyde. *Mutat Res* 424: 83-95.
- Muller FL, Lustgarten MS, Jang Y, Richardson A, Van Remmen H (2007) Trends in oxidative aging theories. *Free Radic Biol Med* 43: 477-503.
- Ostrea EM Jr, Cepeda EE, Fleury CA, Balun JE (1985) Red cell membrane lipid peroxidation and hemolysis secondary to phototherapy. *Acta Paediatr Scand* 74: 378-381.
- Mozaffarian D, Katan MB, Ascherio A, Stampfer MJ, Willett WC (2006) Trans fatty acids and cardiovascular disease. *N Engl J Med* 354: 1601-1613.
- Harvey KA, Arnold T, Rasool T, Antalis C, Miller SJ, et al. (2008) Trans-fatty acids induce pro-inflammatory responses and endothelial cell dysfunction. *Br J Nutr* 99: 723-731.
- Harvey KA, Walker CL, Xu Z, Whitley P, Siddiqui RA (2012) Trans fatty acids: induction of a pro-inflammatory phenotype in endothelial cells. *Lipids* 47: 647-657.
- Zaloga GP, Harvey KA, Stillwell W, Siddiqui R (2006) Trans fatty acids and coronary heart disease. *Nutr Clin Pract* 21: 505-512.