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ABSTRACT

trans Fatty acids have long been used in food manufacturing due in part to their melting point at room temperature between saturated and unsaturated fats. However, increasing epidemiologic and biochemical evidence suggest that excessive *trans* fats in the diet are a significant risk factor for cardiovascular events. A 2% absolute increase in energy intake from *trans* fat has been associated with a 23% increase in cardiovascular risk. Although Denmark has shown it is possible to all but eliminate commercial sources of *trans* fats from the diet, total elimination is not possible in a balanced diet due to their natural presence in dairy and meat products. Thus, the American Heart Association recommends limiting *trans* fats to <1% energy, and the American Dietetic Association, the Institute of Medicine, US Dietary Guidelines, and the National Cholesterol Education Project all recommend limiting dietary *trans*-fat intake from industrial sources as much as possible. The presence of small amounts of *trans* fat in hydrogenated or partially hydrogenated oils/food products will likely cause many Americans to exceed their recommended maximum. This likelihood is exacerbated by the Food and Drug Administration labeling rules, which allow products containing <0.5 g *trans* fat per serving to

claim 0 g *trans* fat. Many products with almost 0.5 g *trans* fat, if consumed over the course of a day, may approximate or exceed the 2 g maximum as recommended by American Heart Association, all while claiming to be *trans*-fat free. Accordingly, greater transparency in labeling and/or active consumer education is needed to reduce the cardiovascular risks associated with *trans* fats.

The development of the hydrogenation process in the early 20th century led to the introduction of commercial *trans* fats into the American diet. Their use expanded rapidly during the second part of the century as food manufacturers needed a replacement to respond to health recommendations to reduce saturated fat and cholesterol intake. In 1993 the Nurses Health Study reported that escalating intake of *trans* fats was associated with increased cardiovascular risk (1). Today, the American Heart Association (2,3) recommends that *trans* fats be <1% of energy, and the American Dietetic Association (4), the Institute of Medicine (5), US Dietary Guidelines (6), and the National Cholesterol Education Program (7) indicate that *trans*-fat intake should be as low as possible. Moreover, since 2006, the Food and Drug Administration (FDA) has required that *trans*-fat content be listed on product Nutrition Facts panels (8). Despite these health recommendations, significant quantities of *trans* fats remain in the diet. This review puts into perspective the uses, consumption, health implications, and regulation of *trans* fats in America in 2009 so that dietetics practitioners and other allied health professionals can stay abreast of the latest developments in this timely topic.

SOURCES OF trans FATS

Saturated fats such as lauric, myristic, palmitic, and stearic acids consist of straight chains of carbon and hydrogen (-CH₂-). They are solid at room temperature because their tight packing results in high melting points. Unsaturated fats contain carbon-carbon double bonds (-CH=CH-). Naturally occurring unsaturated fats are less tightly packed because they are generally in the *cis* configuration, which introduces a characteristic Ushaped bend (Figure 1). The *cis* fatty acids tend to be liquids or oils at room temperature. The double bonds in *trans*-fatty acids produce a more rigid configuration (Figure 1) that requires much less space than the *cis* double bond, resulting in a melting point around room temperature (between that of saturated and *cis* unsaturated fatty acids). This intermediate melting point is highly desirable in food manufacturing because it provides favorable characteristics such as texture and mouth feel. Further, *trans*-fat stability, when exposed to oxygen, enhances product shelf life (oxidation of unsaturated fatty acids is an important cause of rancidity).

There are two primary sources of dietary *trans* fats. First, *trans* fats are formed naturally by bacteria present in the rumens of ruminant animals (9). Dairy and meat products from these animals contain small amounts of *trans* fats; consequently it is impossible to completely eliminate *trans* fats from a balanced diet. Second, *trans* fats are generated from hydrogenation or partial hydrogenation of liquid vegetable oils. This commercial hydrogenation is used primarily for two reasons: to convert liquid oils to solids and to improve the oxidative stability of these fats. Commercial hydrogenation produces most of the *trans* fats in today's American diet. This process, first described by French chemist Paul Sabatier, uses a nickel catalyst to hydrogenate—or saturate—double bonds in vegetable oils. If hydrogenation is complete, the result is a

saturated fatty acid. However, partial hydrogenation forms a mixture of *cis* and *trans*-fatty acids. Trace amounts of *trans* fats are also produced during the process used to deodorize or refine vegetable oils (10). Deodorization is essentially a steam distillation process that removes the volatile compounds from fat that contribute unwanted odors and tastes. Hence, since small amounts of *trans* fat are present in vegetable fats that have not undergone hydrogenation—as well as *trans* fats from natural sources—it will be impossible to completely eliminate them from the diet, even if commercial hydrogenation ceases.

The structures of *trans* fats from commercial and natural sources are different. Biohydrogenation of 18-carbon polyunsaturated fatty acids, such as linolenic acid, forms primarily vaccenic acid (11-*trans* 18:1) and conjugated linoleic acid (9-*cis*, 11-*trans* 18:2). Chemical hydrogenation by contrast produces primarily elaidic acid (9-*trans* 18:1). The health implications of *trans*-fatty acids from commercial vs natural sources are discussed below. However, it is apparent that we consume far more commercial *trans* fats than those of natural origin. The concentration of *trans* fats in commercially produced partially hydrogenated fat is as high as 40% in some shortenings (11), compared with only 6% in natural-derived fat (12).

HEALTH IMPLICATIONS

Epidemiologic Considerations

Despite significant advances in risk reduction through decreased tobacco smoking and management of hyperlipidemia with dietary changes and statins, cardiovascular disease remains the leading cause of death in the United States (10). The incidence of other major risk factors for cardiovascular disease, including hypertension, obesity, and diabetes, also continue to rise (13). Diet is clearly an important modulator of these risk factors, especially in obesity and diabetes. These factors have been incorporated into cardiovascular risk factor models such as the Framingham risk score (14); consequently they are well recognized, even by the general population. The cardiovascular risk associated with *trans* fats is established and due, at least in part, to their effects on lipoproteins such as low-density lipoprotein (LDL) and high-density lipoprotein (HDL) cholesterol, as well as inflammatory mechanisms and interference with fat metabolism.

The landmark Nurses' Health Study showed, after adjustment for age and total energy intake, that the relative risk of coronary heart disease for those in the highest quintile of *trans*-fat intake was 1.5 times greater than for those in the lowest quintile ($P=0.001$) (1). Similar results were observed in the Boston Health Study (15). A metaanalysis of prospective studies by Mozaffarian and colleagues (16) indicated that a 2% absolute increase in energy intake from *trans* fat (equivalent to 4 g in a standard 2,000-kcal diet) was associated with a 23% increase in cardiovascular risk. These results leave no doubt that *trans* fats have a significant, adverse affect on cardiovascular risk.

Denmark has experienced a dramatic decline in cardiovascular disease of about 60% (12,17). This decline is in part due to progressively lower intake of *trans* fat from commercial sources, culminating in the passage of legislation limiting their use. Although simultaneous advances

in the prevention and treatment of cardiovascular disease have played a role in this Danish success story, the importance of eliminating *trans* fats cannot be overlooked. Mozaffarian and colleagues (16) estimated that reducing commercial *trans* fat intake from 2.1% of energy to 1.1% or 0.1% of energy could have a dramatic impact (Figure 2), potentially preventing 72,000 or 228,000 cardiovascular deaths per year in the United States, respectively.

Biochemical Considerations

Trans fats may contribute to heart disease via a number of potential mechanisms, including effects on total serum cholesterol (18) and inflammatory markers such as C-reactive protein and interleukin-6 (19). *Trans* fats have been shown to adversely affect lipoprotein metabolism, presumably through increased activity of cholesteryl-ester transfer protein (20), resulting in higher levels of LDL, very-low-density lipoprotein, triglycerides, and lipoprotein (a), as well as concomitant reductions in LDL size and HDL concentration (21,22).

Van Horn and colleagues (23) previously conducted a comprehensive review of the influence of *trans* fat on blood lipids. Here we provide a brief overview of the most important studies and analyses conducted to date, including some of the most recent research. A meta-analysis of 60 trials comprising 1,672 volunteers investigated the effects of different dietary fats and carbohydrates on blood lipids (Figure 3) (18). This analysis reported a significant increase in both the total cholesterol:HDL cholesterol ratio (0.022, $P < 0.05$; Figure 3a) and in LDL cholesterol (1.54 mg/dL [0.04 mmol/L], $P = 0.002$; Figure 3b) when 1% energy from carbohydrate was replaced with *trans* fats. This increase is contrasted with what was found when 1% of carbohydrate was replaced by saturated fats, which caused a smaller 1.24 mg/dL (0.032 mmol/L) increase in LDL cholesterol and no change in total cholesterol:HDL cholesterol ratio. Replacement with *cis* mono- and polyunsaturated fatty acids were both associated with decreased total:HDL cholesterol ratio and LDL cholesterol when replacing 1% of energy from carbohydrate. Importantly, Figure 3c shows that there was no change in HDL cholesterol with *trans* fats compared to increases with all the other fat types studied, including saturated fats (18).

Other studies have shown that *trans* fats lower HDL cholesterol. Hu and colleagues (21) showed that replacing 2% of energy from carbohydrates with *trans* fat nearly doubled the relative risk of coronary heart disease. By comparison, replacing 5% of energy from carbohydrates with saturated fat was associated with a 1.47-fold increase in relative risk; thus, on a gram-for-gram basis, *trans* fat was associated with an approximately 15 times greater risk of coronary heart disease than saturated fat. A separate analysis also found that—gram for gram—increases in *trans* fats conferred a higher cardiovascular risk than increases in saturated fats (22). These results are somewhat ironic because commercial *trans* fats were originally introduced into the diet as a means to lower the risk of cardiovascular disease from saturated fat intake. This outcome should be a sobering lesson to all of us as we consider alternatives to *trans* fats in the future. Mechanisms for testing the safety of all new fats that enter the marketplace should be established and implemented.

Cardiovascular disease is often described today as an inflammatory disease. Indeed, some studies have shown that inflammatory disease markers such as C-reactive protein are better predictors of

future cardiovascular events than lipid and lipoprotein levels alone (24). The *trans* fats have been shown to increase inflammatory markers, including C-reactive protein, interleukin-6, and tumor necrosis factor- α , possibly through modulation of monocyte and macrophage activity (19,25). Furthermore, elevated circulating levels of soluble adhesion molecules soluble intercellular adhesion molecule-1 and soluble vascular adhesion molecule-1, as well as nitric oxide-mediated endothelial cell dysfunction, have been observed in individuals consuming large quantities of *trans* fats (16,19,26). These inflammatory factors may play an important role in the development of diabetes, atherosclerosis, plaque rupture, and ultimately sudden cardiac death (16).

Other possible adverse effects of *trans* fats include inhibition of the incorporation of other fatty acids into cell membranes, interference with elongation and desaturation of essential fatty acids, increased platelet aggregation, decreased birth weight, increased body weight, decreased serum testosterone, and abnormal sperm morphology (27). No studies have shown that commercial *trans* fats are needed in the diet, or that they confer any health benefit.

Industrial vs Natural *trans* Fat

As detailed above, the *trans* fats derived from natural and commercial sources have different structures. Three recent studies attempted to determine whether *trans* fats from these two sources had different effects on plasma lipids and lipoproteins (28-30). The first of these studies by Motard-Belanger and colleagues (29) determined that diets enriched in either natural or commercial *trans* fats had similar effects on HDL and LDL cholesterol, as well as on the total cholesterol:HDL cholesterol ratio. In the *TRANSFACT* study (28), a statistically significant decrease in both HDL and LDL cholesterol was observed in women consuming commercial but not natural *trans* fat; no change was seen in men. A third study indicated that both industrial and natural *trans* fats had a similar adverse affects on the total cholesterol to HDL cholesterol ratio (30). Taken together these studies provide a growing body of evidence to indicate that there is no clinical difference in the effects of *trans* fats from natural and commercial sources. The most important difference between commercial and natural *trans* fats is in the amount—rather than the type—consumed. The relatively small amount of *trans* fat in the diet from natural sources is unlikely to have important adverse sequelae relative to the much larger quantities of commercial *trans* fats consumed. In fact, there is some suggestion that conjugated linoleic acid available from natural sources has a beneficial affect on atherosclerosis and adiposity (31,32). Further research is needed to determine the health implications of *trans* fats from natural sources. The American Dietetic Association, in its recommendation to minimize dietary *trans* fats, specifically refers to limiting *trans* fats from industrial sources (4).

ALTERNATIVES TO trans FATS

During the past 10 years, a number of alternatives to *trans* fats have been promulgated (reviewed by Eckel and colleagues [2]), although questions remain about most of them. One of the most common modern-day techniques to prepare alternatives to *trans* fats is interesterification, a process that repositions the fatty acids on triglyceride molecules. This process does not actually reduce *trans* fats if they are already present before interesterification, and the health implications of this new commercial fat—much like the early days of *trans* fats—are not yet known. Some

trans-fat alternatives have limited commercial availability; others such as corn and soybean oils are being used as biofuels rather than in the food supply. One of the more attractive options employed by some food manufacturers is the use of genetically modified plants that produce low-linoleic, mid-oleic, or high-oleic oils. Frito-Lay (Pepsico, Purchase, NY), for example, now uses mid-oleic sunflower oil for its potato chips. Developing a reliable supply of these oils has been a challenge due to lower crop yields.

Sundram and colleagues (33) described a fat blend that minimizes *trans* fats while simultaneously optimizing HDL cholesterol:LDL cholesterol ratios. This blend, a 1:1.3:1 ratio of saturated fat:monounsaturated fat:polyunsaturated fat (8.1% energy from saturated fat), yielded a higher HDL cholesterol:LDL cholesterol ratio than diets with low saturated fat (<2% energy from saturated fat) or low polyunsaturated fat/high saturated fat (12%energy from saturated fat). Because only natural oils were used (with no hydrogenation employed), only trace amounts of *trans* fat were present from the deodorization process. This study reminds us that saturated fat should not be employed to replace *trans* fat, with the high saturated fat diet having an adverse affect on blood lipid chemistry. It should be noted that the optimal fat blend proposed by Sundram and colleagues (3,7) exceeded the current recommended target of <7% energy from saturated fat. This approach must be reconciled with dietary recommendations. A recent study found that efforts to reduce *trans* fats in foods have resulted in increases in not only saturated fats, but monounsaturated and polyunsaturated fats as well (34).

As summarized by Eckel and colleagues (2), a number of companies have now taken innovative steps to reduce *trans* fats in their food products. These companies must consider not only the fat composition of their products, but also taste, texture, cost, and availability of materials when reformulating their comestibles. These companies currently include Campbell Soup Co (Camden, NJ), ConAgra Foods (Omaha, NE), General Mills (Golden Valley, MN), The Hershey Company (Hershey, PA), The J.M. Smucker Co (Orrville, OH), Johnson & Johnson (New Brunswick, NJ), Kellogg Co (Battle Creek, MI), Kraft Foods (Northfield, IL), Nestle SA (Vevey, Switzerland), PepsiCo (Purchase, NY), Procter & Gamble (Cincinnati, OH), Sara Lee Corp (Downers Grove, IL), The Schwan Food Co (Marshall, MN), and Unilever (London, UK) (2), as well as GFA Brands, Inc (Boulder, CO), which formulates products based on the fat blend described above (33).

REGULATION

Although development of *trans*-fat alternatives generally emanate from manufacturers, such developments should have regulation based on relevant science and be embraced by the health care community to direct reductions in the amounts of *trans* fats allowed. Figure 4 shows the recommendations of the American Heart Association, the American Dietetic Association, the Institute of Medicine, US Dietary Guidelines, and the National Cholesterol Education Program Adult Treatment Panel (2-7). These influential groups acknowledge that there is no medical rationale for allowing commercial *trans* fats in the diet and recommend limiting their consumption as much as possible. No federal regulation limits *trans* fats in the United States. Instead, since 2006 FDA has required that all products state the amount of *trans* fat per serving on the Nutrition Facts panel (8); no percent daily value is given because there is no nutritional requirement for *trans* fats in the diet. Importantly, *trans* fats with <0.5 g/serving can be listed as

containing 0 g. Food manufacturers have responded by reducing *trans*-fat content in many products to <0.5 g. A number of health care institutions such as Cleveland Clinic, Kaiser Permanente facilities, and a number of other hospitals have followed suit by banning products with *trans* fat on the label (35). These institutions have led by example and have provided direction for other institutions to quickly become *trans*-fat free (35). Many would argue that the label requirement has been effective in reducing *trans*-fat use. Indeed, Eckel and colleagues (36) recently showed that awareness of *trans* fats increased between March 2006 and May 2007. However, these same authors concluded that consumer knowledge about *trans* fat and its sources remained low, highlighting the need for consumer education in this area (36).

As previously discussed, required limitations on dietary *trans* fats in Denmark have nearly eliminated *trans* fats from commercial sources and has contributed to continued reductions in cardiovascular disease–related deaths (12,17). Canada is also moving to limit *trans* fats in the diet, and is currently asking manufacturers to voluntarily reduce *trans* fats (37). A number of states and municipalities (eg, New York City) have introduced or passed legislation either to limit *trans* fat use or to enforce stricter labeling (38). However, it is too soon to evaluate the effectiveness of these initiatives.

THE NUTRITION FACTS PANEL

Although the FDA labeling requirement has led to some reduction in dietary *trans* fats, the labels may mislead many consumers. Because products with <0.5 g/serving may be labeled as having 0 g *trans* fat, an individual may ingest significant quantities of *trans* fats while believing they have consumed none. This possibility is especially important when considering the American Heart Association recommendation to limit *trans* fat to <1% energy (or 2 g based on a 2,000-kcal diet) (2,3). For example, the Nutrition Facts panel for crème filled sponge cakes show 0 g *trans* fat per serving (a serving is one cake), even though the US Department of Agriculture Nutrient Database lists these products as having 0.459 g *trans* fat per serving (11). Similarly, microwave popcorn (94% fat free) contains 0.251g *trans* fat in a single 1-oz serving (11). It should be noted that manufacturers frequently alter the oils and other ingredients in foods, so these examples are merely illustrative. Food labels provide consumers with some information on the *trans* fats for food eaten at home. However, food eaten outside the home may be a major source of *trans* fats for which no labeling information is readily accessible. Historically, one of the major uses of hydrogenated oils was for butter alternatives, such as shortening and oleo. A recent analysis of currently available buttery spreads (*trans*-fat–free margarine), which the National Cholesterol Education Program Adult Treatment Panel recommends be used in place of butter, stick margarine, or shortening (7) (Figure 4) , showed that *trans*-fat content ranged from 0.07 to 0.47 g per 14-g serving (1 T) despite stating 0 g on the Nutrition Facts panel (personal communication, GFA Brands, Inc., January 2009).

Serving or portion size is another important consideration. It has been reported that consumers consistently underestimate portion size (39) and consume oversized portions when they are presented (40-42). Consequently, an individual may ingest significant quantities of *trans*

fats when consuming a single portion of a product labeled 0 g *trans* fat. Taken together this highlights the need for consumer education to help them fully understand the Nutrition Facts panel, in terms of assessing *trans*-fat and saturated fat intake, as well as portion size.

Cost is another important consideration in elimination of *trans* fats. A recent study found an inverse correlation between *trans*-fat and saturated fat content and price; that is, products low in *trans* fats cost more than those high in *trans* fats (43). In the current economic climate, the additional cost associated with the lower *trans*-fat products could be a barrier to elimination of *trans* fats from the diet.

Stender and colleagues (12) estimated that almost 50 g *trans* fats could be ingested in the United States from a single high-fat meal and snacks comprising large chicken nuggets with fries, 100 g cookies, cakes, or wafers, and 100 g microwave popcorn. Although such high-fat menus contain more *trans* fat than anywhere else in the world, the total *trans* fat intake in the United States is currently unknown. The most recent available data from National Health and Nutrition Examination Survey indicated that *trans* fat comprised 2% of energy intake (twice current recommendations) (44). However, these estimates may be spuriously low, due to underestimation of portion size, and many individuals consume much more *trans* fat than 2% of their daily energy intake.

Collectively, these data suggest the need for more accurate labeling of *trans* fats to protect against cardiovascular disease. Such labeling could take the form of a lower threshold for the *trans*-fat-free claim, listing of actual *trans*-fat values for all products, or noting the presence of added *trans* fats, including for products that are currently listed as 0 g *trans* fat. However, there may be limitations on the ability to further regulate *trans* fats because few clinical data are available to determine the cut points for the upper level of dietary *trans*-fat intake.

CONCLUSIONS

Trans fats are believed to be nutritionally unnecessary. Epidemiologic evidence has shown that they are an important risk factor for cardiovascular disease; a metaanalysis of these studies demonstrated that a 2% increase in daily energy intake from *trans* fat was associated with a 23% increase in cardiovascular disease risk (15). *Trans* fats have also been shown to have a significant adverse influence on serum lipids and lipoproteins, increasing cardiovascular disease risk to a greater extent than saturated fat (18,21). A number of mechanisms for the effects of *trans* fats have been proposed, including increased activity of cholesteryl-ester transfer protein and elevated levels of inflammatory markers. It is clear from these studies that dietary *trans* fats should be minimized. However, the presence of *trans* fats in dairy and meat products will make complete elimination from a balanced diet impossible.

As food manufacturers and the food industry seek alternatives to *trans* fats, hurdles include supply of ingredients and unknown health sequelae of new processes. *Trans* fats gained popularity as a means of replacing saturated fats in the diet. However, we now know that *trans* fats have greater adverse health implications than the saturated fats they sought to replace. Eliminating *trans* fats by returning to a high-saturated-fat diet is inappropriate.

Consumers are not fully aware of the well-established health consequences of *trans* fats. Indeed, many are confused as to what fats they should or should not be eating. Many are likely consuming *trans* fats in excess of the maximum intake recommended by the American Heart Association (3). The current FDA labeling requirements are a good first step in providing consumers with information on *trans* fats. However, given the recommendation that *trans* fat intake be as low as possible, allowing all products with <0.5 g *trans* fats to claim 0 g *trans* fats can be misleading to many consumers. Eating four or five daily servings of foods with close to 0.5 g *trans* fat can mean an individual who believes he/she is consuming a healthful, balanced diet is actually exceeding 1% total energy from *trans* fats. Greater transparency is required to allow consumers to restrict dietary *trans* fats more effectively. Average consumers do not understand the Nutrition Facts label, or its relation to actual portion size (45,46). Consumer education is extremely important. In the interim, educational programs targeted at these consumers must be developed to help them determine which foods likely contain *trans* fats based on the presence of hydrogenated or partially hydrogenated oils in the ingredient list, as well as to more accurately estimate their portion size relative to standardized values on the Nutrition Facts panel.

STATEMENT OF POTENTIAL CONFLICT OF INTEREST

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REFERENCES

1. Willett WC, Stampfer MJ, Manson JE, Colditz GA, Speizer FE, Rosner BA, Sampson LA, Hennekens CH. Intake of *trans* fatty acids and risk of coronary heart disease among women. *Lancet*. 1993;341:581- 585.
2. Eckel RH, Borra S, Lichtenstein AH, Yin-Piazza SY. Understanding the complexity of *trans* fatty acid reduction in the American diet: American Heart Association *trans* Fat Conference 2006: Report of the *trans* Fat Conference Planning Group. *Circulation*. 2007;115:2231- 2246.
3. American Heart Association Nutrition Committee, Lichtenstein AH, Appel LJ, Brands M, Carnethon M, Daniels S, Franch HA, Franklin B, Kris-Etherton P, Harris WS, Howard B, Karanja N, Lefevre M, Rudel L, Sacks F, Van Horn L, Winston M, Wylie-Rosett J. Diet and lifestyle recommendations revision 2006: A scientific statement from the American Heart Association Nutrition Committee. *Circulation*. 2006;114:82-96.
4. Kris-Etherton PM, Innis S. Position of The American Dietetic Association and Dietitians of Canada: Dietary fatty acids. *J Am Diet Assoc*. 2007;107:1599-1611.
5. Letter Report on Dietary Reference Intakes for *trans* Fatty Acids. Published 2002. Institute of Medicine Web site. <http://www.iom.edu/CMS/5410.aspx>. Accessed March 4, 2009.
6. *Nutrition and Your Health: Dietary Guidelines for Americans*, 2005. 6th ed. Washington, DC: US Government Printing Office; 2005.
7. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third Report

- of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106:3143-3421.
8. Questions and answers about *trans* fat nutrition labeling. Published 2006. FDA Center for Food Safety and Applied Nutrition, Labeling and Dietary Supplements Web site. http://vm.cfsan.fda.gov/_dms/qatrans2.html. Accessed May 18, 2009.
 9. Khanal RC, Dhiman TR. Biosynthesis of conjugated linoleic acid (CLA): A review. *Pakistan J Nutr*. 2004;3:72-81.
 10. Tasan Y, Demirci N. *Trans* FA in sunflower oil at different steps of refining. *J Am Oil Chem Soc*. 2003;80:825-828.
 11. US Dept of Agriculture, Agricultural Research Service. USDA National Nutrient Database for Standard Reference, Release 21. Nutrient Data Laboratory Home Page, <http://www.ars.usda.gov/nutrientdata>. Accessed March 18, 2009.
 12. Stender S, Astrup A, Dyerberg J. Ruminant and industrially produced *trans* fatty acids: Health aspects. *Food Nutr Res*. Published March 12, 2008. Accessed February 10, 2010. doi:10.3402/fnr.v52i0.1651.
 13. Kung HC, Hoyert DL, Xu JQ, Murphy SL. Deaths: Final data for 2005. *Natl Vital Stat Rep*. 2008;56:1-121.
 14. Wilson PW, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB. Prediction of coronary heart disease using risk factor categories. *Circulation*. 1998;97:1837-1847.
 15. Ascherio A, Hennekens CH, Buring JE, Master C, Stampfer MJ, Willett WC. *Trans*-fatty acids intake and risk of myocardial infarction. *Circulation*. 1994;89:94-101.
 16. Mozaffarian D, Katan MB, Ascherio A, Stampfer MJ, Willett WC. *Trans* fatty acids and cardiovascular disease. *N Engl J Med*. 2006; 354:1601-1613.
 17. Leth T, Jensen HG, Mikkelsen AA, Bysted A. The effect of the regulation on *trans* fatty acid content in Danish food. *Atheroscler Suppl*. 2006;7:53-56.
 18. Mensink RP, Zock PL, Kester AD, Katan MB. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: A meta-analysis of 60 controlled trials. *Am J Clin Nutr*. 2003;77:1146-1155.
 19. Lopez-Garcia E, Schulze MB, Meigs JB, Manson JE, Rifai N, Stampfer MJ, Willett WC, Hu FB. Consumption of *trans* fatty acids is related to plasma biomarkers of inflammation and endothelial dysfunction. *J Nutr*. 2005;135:562-566.
 20. van Tol A, Zock PL, van Gent T, Scheek LM, Katan MB. Dietary *trans* fatty acids increase serum cholesterylester transfer protein activity in man. *Atherosclerosis*. 1995;115:129-134.
 21. Hu FB, Stampfer MJ, Manson JE, Rimm E, Colditz GA, Rosner BA, Hennekens CH, Willett WC. Dietary fat intake and the risk of coronary heart disease in women. *N Engl J Med*. 1997;337:1491-1499.
 22. Ascherio A, Katan MB, Zock PL, Stampfer MJ, Willett WC. *Trans* Fatty acids and coronary heart disease. *N Engl J Med*. 1999;340:1994-1998.
 23. Van Horn L, McCoin M, Kris-Etherton PM, Burke F, Carson JA, Champagne CM. The evidence for dietary prevention and treatment of cardiovascular disease. *J Am Diet Assoc*. 2008;108:287-331.

24. Ridker PM, Rifai N, Rose L, Buring JE, Cook NR. Comparison of C-reactive protein and low-density lipoprotein cholesterol levels in the prediction of first cardiovascular events. *N Engl J Med*. 2002;347:1557-1565.
25. Han SN, Leka LS, Lichtenstein AH, Ausman LM, Schaefer EJ, Meydani SN. Effect of hydrogenated and saturated, relative to polyunsaturated, fat on immune and inflammatory responses of adults with moderate hypercholesterolemia. *J Lipid Res*. 2002;43:445-452.
26. de Roos NM, Bots ML, Katan MB. Replacement of dietary saturated fatty acids by *trans* fatty acids lowers serum HDL cholesterol and impairs endothelial function in healthy men and women. *Arterioscler Thromb Vasc Biol*. 2001;21:1233-1237.
27. Simopoulos AP. The importance of the omega-6/omega-3 fatty acid ratio in cardiovascular disease and other chronic diseases. *Exp Biol Med (Maywood)*. 2008;233:674-688.
28. Chardigny JM, Destailats F, Malpuech-Brugere C, Moulin J, Bauman DE, Lock AL, Barbano DM, Mensink R.P, Bezelgues JB, Chaumont P, Combe N, Cristiani I, Joffre F, German JB, Dionisi F, Boirie Y, Sebedio JL. Do *trans* fatty acids from industrially produced sources and from natural sources have the same effect on cardiovascular disease risk factors in healthy subjects? Results of the *trans* Fatty Acids Collaboration (TRANSFACT) study. *Am J Clin Nutr*. 2008;87:558-566.
29. Motard-Belanger A, Charest A, Grenier G, Paquin P, Chouinard Y, Lemieux S, Couture P, Lamarche B. Study of the effect of *trans* fatty acids from ruminants on blood lipids and other risk factors for cardiovascular disease. *Am J Clin Nutr*. 2008;87:593-599.
30. Katan M, Wanders A, Brouwer I, Siebelink E. Impact of animal vs industrial *trans* fatty acids on lipoproteins in humans [abstract 51]. *Atherosclerosis* 2009;10(suppl 2):e23.
31. Campbell B, Kreider RB. Conjugated linoleic acids. *Curr Sports MedRep*. 2008;7:237-241.
32. Mitchell PL, McLeod RS. Conjugated linoleic acid and atherosclerosis: Studies in animal models. *Biochem Cell Biol*. 2008;86:293-301.
33. Sundram K, Hayes KC, Siru OH. Both dietary 18:2 and 16:0 may be required to improve the serum LDL/HDL cholesterol ratio in normocholesterolemic men. *J Nutr Biochem*. 1995;6:179-187.
34. Stender S, Astrup A, Dyerberg J. What went in when *trans* fats went out? *N Engl J Med*. 2009;361:314-316.
35. Aase S. Taking *trans* fats of the menu: What you can learn from *trans*-fat bans at Sheikh Khalifa Medical City and the Cleveland Clinic. *J Am Diet Assoc*. 2009;109:1148-1151.
36. Eckel RH, Kris-Etherton P, Lichtenstein AH, Wylie-Rosett J, Groom A, Stitzel KF, Yin-Piazza S. Americans' awareness, knowledge, and behaviors regarding fats: 2006-2007. *J Am Diet Assoc*. 2009;109:288-296.
37. L'Abbe MR, Brown S. TRANSforming the food supply—Report of the *Trans* Fat Task Force. In: Nutrition Evaluation Division, Health Canada. Ottawa, Ontario, Canada; 2006:1-120. http://www.hc-sc.gc.ca/fn-an/nutrition/gras-trans-fats/tf-ge/tf-gt_rep-rap-eng.php. Accessed February 10, 2010.
38. *Trans* fat and menu labeling legislation. Published 2008. National Conference of State Legislatures Web site. <http://www.ncsl.org/programs/health/transfatmenulabelingbills.htm>. Accessed January 9, 2009.
39. Kelly MT, Rennie KL, Wallace JM, Robson PJ, Welch RW, Hannon-Fletcher MP, Livingstone, MB. Associations between the portion sizes of food groups consumed and measures of adiposity in the British National Diet and Nutrition Survey. *Br J Nutr*. 2009;101:1413-1420.

40. Greenwood JL, Stanford JB. Preventing or improving obesity by addressing specific eating patterns. *J Am Board Fam Med*. 2008;21:135- 140.
41. Fisher JO, Arreola A, Birch LL, Rolls BJ. Portion size effects on daily energy intake in low-income Hispanic and African American children and their mothers. *Am J Clin Nutr*. 2007;86:1709-1716.
42. Jeffery RW, Rydell S, Dunn CL, Harnack LJ, Levine AS, Pentel PR, Baxter JE, Walsh EM. Effects of portion size on chronic energy intake. *Int J Behav Nutr Phys Act*. 2007;4:27.
43. Albers MJ, Harnack LJ, Steffen LM, Jacobs DR. 2006 marketplace survey of *trans*-fatty acid content of margarines and butters, cookies and snack cakes, and savory snacks. *J Am Diet Assoc*. 2008;108:367-370.
44. Harnack L, Lee S, Schakel SF, Duval S, Luepker RV, Arnett DK. Trends in the *trans*-fatty acid composition of the diet in a metropolitan area: The Minnesota Heart Survey. *J Am Diet Assoc*. 2003;103:1160-1166.
45. Borra S. Consumer perspectives on food labels. *Am J Clin Nutr*. 2006;83(suppl):1235S.
46. Rolls BJ, Morris EL, Roe LS. Portion size of food affects energy intake in normal-weight and overweight men and women. *Am J Clin Nutr*. 2002;76:1207-1213.
47. Stampfer MJ, Sacks FM, Salvini S, Willett WC, Hennekens CH. A prospective study of cholesterol, apolipoproteins, and the risk of myocardial infarction. *N Engl J Med*. 1991;325:373-381.
48. Oh K, Hu FB, Manson JE, Stampfer MJ, Willett WC. Dietary fat intake and risk of coronary heart disease in women: 20 years of follow-up of the nurses' health study. *Am J Epidemiol*. 2005;161:672-679.

Figure 1. Structure and melting point of saturated, *cis* (18:1) and *trans* (18:1) fatty acids.

Stearic Acid



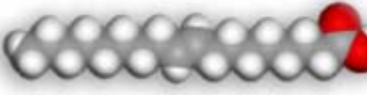
Solid: Melting Point = +70°C

Oleic Acid



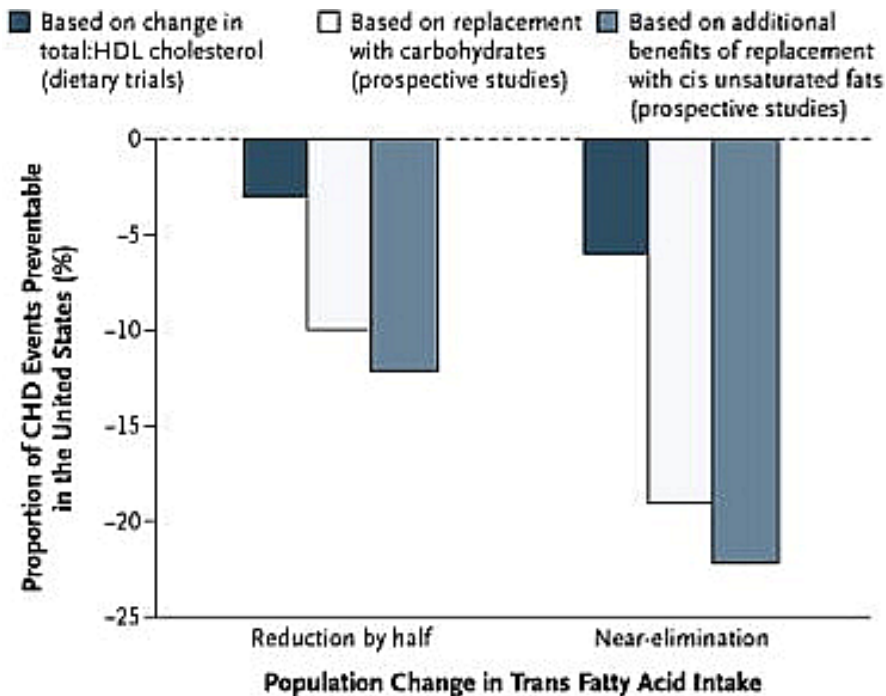
Liquid: Melting Point = -5°C

Elaidic Acid



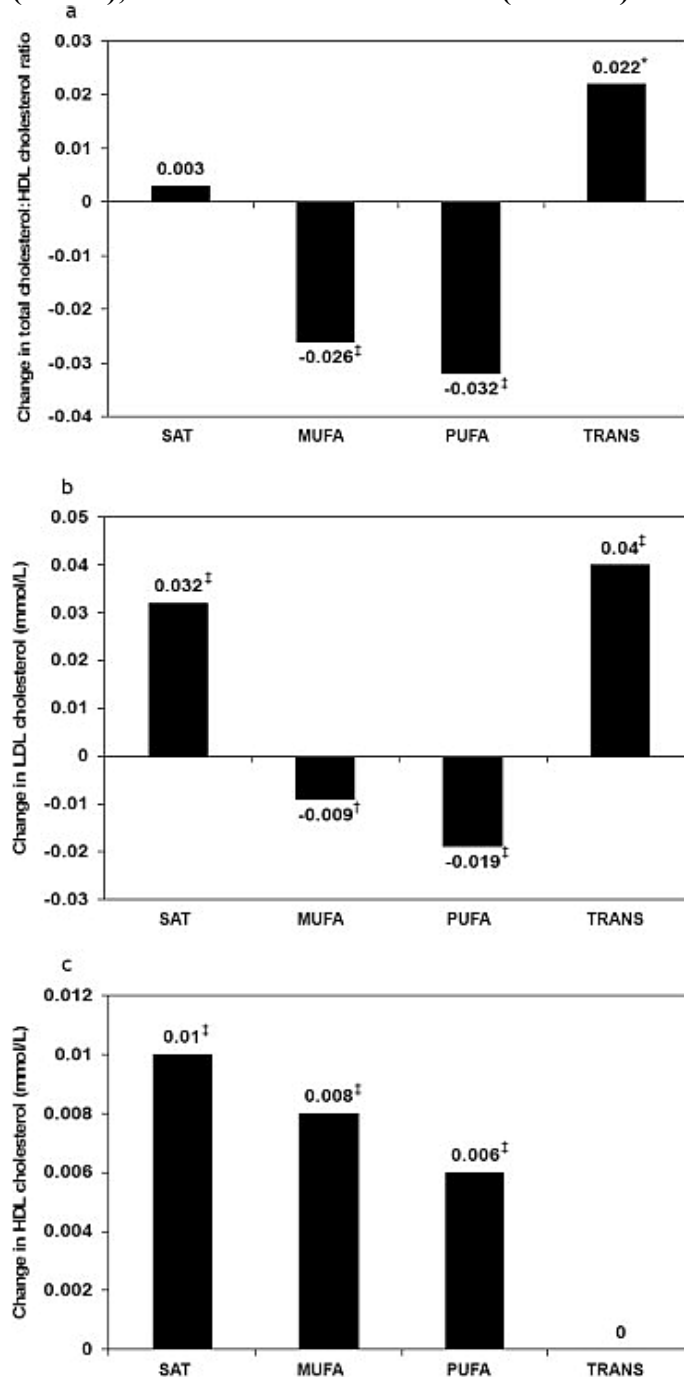
Solid: Melting Point = 42°C

Figure 2. Estimated effects of reducing the consumption of commercially produced *trans*-fatty acids on the incidence of coronary heart disease (CHD) (nonfatal myocardial infarction or death from CHD) in the United States.



HDL=high-density lipoprotein. Population-attributable risks were calculated for a reduction by approximately half in the percent of energy intake (from 2.1% to 1.1%) or the near elimination (from 2.1% to 0.%) of *trans*-fatty acid intake. Three effects were estimated: based on the effects of isocaloric replacement of *trans* fats with *cis* mono- or polyunsaturated fats (averaged effect) on the ratio of total to HDL cholesterol in controlled trials and the relation of this ratio to the incidence of CHD (46); based on the reported relation of *trans*-fatty acid intake, substituted for carbohydrate intake, with the incidence of CHD in a pooled analysis of prospective studies; and based on the additional potential benefits if *trans* fats were replaced with *cis* mono- or polyunsaturated fats (averaged effect), as calculated from the pooled analysis of the prospective studies and the difference in relative risk resulting from *trans* fats being replaced by carbohydrates as compared with *cis* unsaturated fats in updated 2005 analyses from two cohorts (47,48). Reprinted with permission from reference (16): Mozaffarian D, Katan MB, Ascherio A, Stampfer MJ, Willett WC. *Trans* fatty acids and cardiovascular disease. *N Engl J Med.* 2006;354:1601-1613. Copyright ©2006 Massachusetts Medical Society. All rights reserved.

Figure 3. Predicted changes in the ratio of serum total cholesterol to high-density lipoprotein (HDL) cholesterol (a), Low-density lipoprotein(LDL) cholesterol (b), and HDL-cholesterol concentrations when carbohydrates constituting 1% of energy are replaced isoenergetically with saturated (SAT), cis monounsaturated (MUFA), cis polyunsaturated (PUFA), or trans monounsaturated (TRANS) fatty acids (c).



*P_{0.05}; †P_{0.01}; ‡P_{0.001}. Adapted with permission from Mensink et al (18): *Am J Clin Nutr.* (2003;77:1146-1155), American Society for Nutrition.

Figure 4. Position of major professional societies on *trans* fat intake. TFA_ *trans*-fatty acid. SFA_ saturated fatty acid. AHA_ American Heart Association. LDL_ low-density lipoprotein. CHD_ coronary heart disease.

American Dietetic Association (4)	“Foods containing commercially derived TFA should be minimized. . . . TFA replacement strategies (should) not result in a higher TFA and SFA.”
American Heart Association (2,3)	“A recent meta-analysis . . . found that a 2% increase in energy intake from <i>trans</i> fatty acids was associated with a 23% increase in the incidence of coronary heart disease.” “As a set of goals, the AHA recommends intakes of _7% of energy as saturated fat, _1% of energy as <i>trans</i> fat, and _300 mg cholesterol per day.”
Institute of Medicine (5)	“There is a positive linear trend between <i>trans</i> fatty acid intake and total and LDL cholesterol concentration, and therefore increased risk of CHD, thus suggesting a Tolerable Upper Intake Level (UL) of zero.”
US Dietary Guidelines (6)	“. . . keep <i>trans</i> fatty acid consumption as low as possible.”
National Cholesterol Education Program: ATP III (7)	“Intakes of <i>trans</i> fatty acids should be kept low. The use of liquid vegetable oil, soft margarine, and <i>trans</i> fatty acid-free margarine are encouraged instead of butter, stick margarine, and shortening.”