

Consumption of a Solid Fat Rich in Lauric Acid Results in a More Favorable Serum Lipid Profile in Healthy Men and Women than Consumption of a Solid Fat Rich in *trans*-Fatty Acids¹

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ABSTRACT Solid fats are used in food manufacturing to provide texture and firmness to foods. Such fats are rich in either saturated or *trans*-fatty acids, both of which increase the risk of coronary heart disease. Epidemiological and experimental studies suggest that *trans*-fatty acids increase risk more than do saturates because they lower serum high density lipoprotein (HDL) cholesterol. However, there appear to be differences between saturates in their effect on HDL cholesterol. We investigated whether the consumption of a solid fat rich in lauric acid (C12:0) would result in a more favorable blood lipid profile than the consumption of a solid fat rich in *trans*-fatty acids. We fed 32 healthy men and women two controlled diets in a 2 × 4-wk randomized crossover design. The diets consisted of a background diet supplemented with margarines. In the *trans*-diet, 9.2% of energy was provided by *trans*-fatty acids and 12.9% by saturated fatty acids. In the Sat-diet, energy intake was 0% from *trans*-fatty acids and 22.9% from saturated fatty acids. Lauric acid composed one third of all saturates in the Sat-diet. Serum HDL cholesterol was 0.36 mmol/L lower at the end of the *trans*-diet than at the end of the Sat-diet (95% confidence interval, -0.46 to -0.26), whereas serum low density lipoprotein cholesterol and triglyceride concentrations remained stable. Serum total cholesterol was 0.31 mmol/L (95% confidence interval, -0.48 to -0.14) lower at the end of the *trans*-diet than at the end of the Sat-diet. Consumption of a solid fat rich in lauric acid gives a more favorable serum lipoprotein pattern than consumption of partially hydrogenated soybean oil rich in *trans*-fatty acids. Thus, solid fats rich in lauric acids, such as tropical fats, appear to be preferable to *trans*-fats in food manufacturing, where hard fats are indispensable. J. Nutr. 131: 242-245, 2001.

KEY WORDS: • lipoproteins • HDL • *trans*-fatty acids • saturated fatty acids • humans

Margarines and vegetable shortenings are important sources of *trans*-fatty acids in industrialized countries (Ascherio et al. 1999). The intake of vegetable shortenings exceeds that of margarines and is likely to increase even further because of their use in ready-to-eat foods (Katan 2000) and for deep-fat frying. This is an undesirable development, because the intake of *trans*-fatty acids is related to an increased risk of cardiovascular disease (Hu et al. 1997, Kromhout et al. 1995, Willett et al. 1993). Experimental studies support this relation: many metabolic studies showed that *trans*-fatty acids have an unfavorable effect on blood lipids because, like saturated fatty acids, they increase serum low density lipoprotein (LDL) cholesterol (Judd et al. 1994, Mensink and Katan 1990, Nestel et al. 1992, Zock and Katan 1992). Moreover, the replacement of saturated by *trans*-fatty acids was shown to decrease serum high density lipoprotein (HDL)³ cholesterol in many studies (Almendinger et al. 1995, Kris-Etherton and Yu 1997, Müller et

al. 1998, Sundram et al. 1997), which suggests an additional increase in the risk of cardiovascular disease (Aro et al. 1997, Ballantyne et al. 1999, Castelli et al. 1992, de Backer et al. 1998, Gordon et al. 1989, Kitamura et al. 1994, Pearson et al. 1979, Sharrett et al. 1999, Sorlie et al. 1999). In response to the negative health effects of *trans*-fatty acids, many European manufacturers have decreased the *trans*-fatty acid content of most stick margarines to <2% of energy (Katan 1995, Michels and Sacks 1995). The *trans*-fatty acid content can be decreased by reducing the degree of hydrogenation, which results in softer margarines. Alternatively, solid margarines can be produced from tropical fats instead of from partially hydrogenated vegetable oils: the amount of saturated fatty acids will then be higher but the amount of *trans*-fatty acids will be negligible (Michels and Sacks 1995). Studies suggest that the replacement of 10% of energy from *trans*-fat by saturated fat will increase serum HDL cholesterol by 0.15 mmol/L (Zock et al. 1995, Zock and Katan 1992). However, this prediction is based on studies in which palmitic acid is the major saturated fatty acid, and some, although not all (Kris-Etherton and Yu 1997), studies suggest that lauric acid might increase serum HDL cholesterol more than palmitic acid (Temme et al. 1996, Zock and Katan 1992). Palm kernel fat and coconut fat are rich sources of lauric acid (U.S. Department of Agriculture).

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³ Abbreviations used: CI, confidence interval; HDL, high density lipoprotein; LDL, low density lipoprotein.

TABLE 1

Fatty acid composition of the margarines used in the diet rich in *trans*-fatty acids and the diet rich in saturated fatty acids

Fatty acid	Margarine with <i>trans</i> fatty acids	Margarine with saturated fatty acids
	g/100 g fatty acids	
Saturated	30.5	63.1
Lauric acid (C12:0)	Not detected	24.5
Myristic acid (C14:0)	0.1	10.2
Palmitic acid (C16:0)	10.5	17.0
Stearic acid (C18:0)	18.5	7.4
Monounsaturated	59.5	21.3
<i>cis</i> -Monounsaturated	18.6	20.9
<i>trans</i> -Monounsaturated	40.9 ¹	0.3
Polyunsaturated	8.7	15.0
Linoleic acid (<i>cis,cis</i> -C18:2)	8.2	14.6
Unknown	1.3	0.6

¹ All *trans*-monounsaturated fatty acids were *trans*-C18:1.

We investigated whether replacement of a margarine rich in *trans*-fatty acids by a margarine rich in lauric acid would produce a stronger increase in serum HDL cholesterol than the 0.15 mmol/L per 10% of energy that was estimated previously.

MATERIALS AND METHODS

The protocol of the study was approved by the Medical Ethical Committee of Wageningen University. We gave each volunteer a written and oral presentation of the purpose and execution of the study, and each volunteer signed an informed consent form.

Subjects. We enrolled 11 men and 21 women with a mean age of 30 y (range, 18–69 y) in the study. All of the volunteers were nonsmokers, and all were healthy as assessed by means of a medical questionnaire. The initial serum cholesterol concentration was 5.0 mmol/L (range, 3.0 to 7.1 mmol/L), and the body mass index 22.8 ± 2.5 kg/m². The volunteers had no history of any chronic illness and were not taking any medication known to affect blood lipid metabolism. They all completed the study.

Study design. We provided two controlled diets for 4 wk each in a randomized crossover design. One diet was rich in *trans*-fatty acids (*trans*-diet), and one was diet rich in saturated fatty acids (Sat-diet). There was no washout period between the two diets. The two diets were equal except for supplemental margarines, which were given in a 28-d menu cycle. The background diet consisted of conventional food items.

The margarine that was used in the diet rich in *trans*-fatty acids was a blend of 70 parts of partially hydrogenated soybean oil, containing 44% *trans*-C18:1 (Gouda's Glorie; Van Dijk Foods, Lopik, the Netherlands); 14 parts of a vegetable oil containing 63% linoleic acid and 23% oleic acid (Becel; Unilever, Vlaardingen, the Netherlands) and 16 parts of water (Table 1). The margarine that was used in the diet rich in saturated fat was a blend of 60 parts of palm kernel fat (Loders Croklaan, Wormerveer, the Netherlands) and 40 parts of commercially available margarine made from a blend of unhydrogenated rapeseed oil, soybean oil, sunflower oil, palm kernel fat, coconut oil and palm oil (Blue Band; Van den Bergh BV, Rotterdam, the Netherlands) (Table 1). Both supplemental margarines were produced at NIZO Food Research (Ede, the Netherlands). The margarines were used as a spread, as shortening in bread and cookies and as fat in sauce and gravy. They supplied 77% of total fat in the *trans*-diet and 68% of total fat in the Sat-diet. The composition of the experimental diets was calculated using food composition tables (Anonymous 1996; Hulshof et al. 1999; van Poppel et al. 1998). To check the composition of the diets, we collected duplicates of all meals (Table 2). The analyzed values were similar to the calculated composition.

We designed menus for 14 levels of energy intake, ranging from 7 to 20 MJ/d. The subjects were allocated to an energy intake level close to their habitual energy intake, which was estimated from a food frequency questionnaire. We provided 90% of energy; all food was weighed out for each subject. The remaining 10% of energy had to be chosen from a list of low-fat food items. Subjects recorded their choice from this low-fat food list in a diary. On Monday through Friday of each week, subjects ate a hot meal under our supervision. All other foods (bread; margarine; meat and/or cheese; honey, jam or sprinkles; fruit; milk and/or yogurt) were packaged for consumption at home, as was food for the weekends. They received the diets for 21–32 d (mean 27.5 d).

We weighed the subjects twice a week and increased or decreased their energy intakes as needed to maintain stable body weights.

Biochemical analysis. We took blood samples on two separate days after d 19 of each diet. All four blood samples of each subject were analyzed in duplicate within one assay. Serum total cholesterol and triglycerides (Cholesterol Flex and Triglycerides Flex reagent cartridge; Dade Behring, Newark, NJ) and HDL cholesterol (*liquid* N-geneous HDL-C assay; Instruchemie BV, Hilversum, the Netherlands) were measured, and LDL cholesterol was calculated with the Friedewald formula. The coefficient of variation of 64 duplicate measurements was 0.4% for total cholesterol, 1.5% for triglycerides and 1.1% for HDL cholesterol.

The fatty acid composition of the margarines and the experimental diets was analyzed by gas-liquid chromatography of the fatty acid methyl esters (Metcalf 1966) and, for 18 carbon *trans*-fatty acids, by gas-liquid chromatography of fatty acid 4,4-dimethylxazoline derivatives (Fay and Richli 1991).

Statistics. We averaged the duplicate measurements in each dietary period and then calculated for each subject the difference between diets. We tested whether these differences were significantly different from zero by the Student's *t* test for paired samples. We give two-sided 95% confidence intervals (CI) for the differences.

TABLE 2

Analyzed composition of duplicate portions of the two experimental diets

Fatty acid	Diet rich in <i>trans</i> -fatty acids	Diet rich in saturated fatty acids
Carbohydrate, % of energy	48.6	45.6
Protein, % of energy	14.0	13.5
Total fat, % of energy	37.4	41.0
Saturated	12.9	22.9
Lauric acid (C12:0)	0.3	6.8
Myristic acid (C14:0)	0.8	3.8
Palmitic acid (C16:0)	5.7	7.8
Stearic acid (C18:0)	5.3	3.1
Monounsaturated, total	18.2	8.8
<i>cis</i> -Monounsaturated	8.8	8.3
<i>trans</i> -Monounsaturated	9.3	0.3
Polyunsaturated	4.7	6.9
Linoleic acid (<i>cis,cis</i> -C18:2)	4.1	5.9
Linolenic acid (<i>cis,cis,cis</i> -C18:3)	0.3	0.7
Cholesterol		
mg/MJ	27.0	26.8
mg/d	248.4	253.5
Fiber		
g/MJ	3.2	3.1
g/d	29.4	29.3
Energy		
MJ/d	9.20	9.46
kcal/d	2199	2261

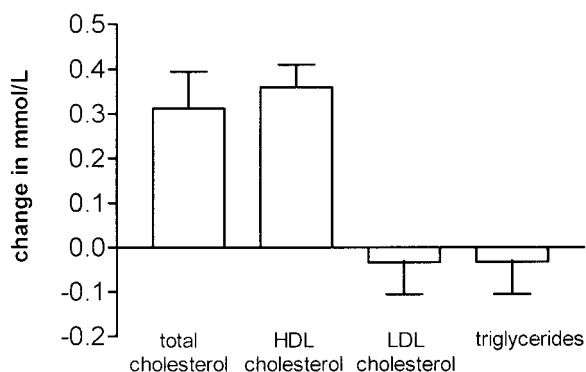


FIGURE 1 Differences in serum total cholesterol, high density lipoprotein (HDL) and low density lipoprotein (LDL) cholesterol and triglyceride concentrations in 32 fasting men and women when 9% of energy of *trans*-fatty acids in the diet was replaced with saturated fatty acids. Positive values represent higher values after the consumption of a diet rich in saturated fatty acids than after a diet rich in *trans*-fatty acids. The two diets were consumed for 21–32 d. Bars represent means with SEM. To convert values for total, HDL and LDL cholesterol to mg/dL, multiply by 38.67. To convert values for triglycerides to mg/dL, multiply by 88.54.

RESULTS

Body weight and compliance with study protocol.

Changes in body weight during the study were small and nonsignificant: -0.4 ± 0.9 kg during the *trans*-diet and -0.6 ± 0.9 kg during the Sat-diet ($P > 0.5$). We checked the diaries for deviations from the study protocol, but only minor deviations, which were unlikely to interfere with the results of the study, had been reported.

Serum lipids. Serum total cholesterol was 0.31 mmol/L (95% CI, 0.14–0.48 mmol/L, $P = 0.0007$) or 12.0 mg/dL lower at the end of the *trans*-diet period than at the end of the Sat-diet period (Fig. 1). This difference was mainly due to a difference in HDL cholesterol, which was 1.89 ± 0.46 mmol/L (73.1 ± 17.8 mg/dL) at the end of the Sat-diet and 1.46 ± 0.33 mmol/L (56.5 ± 12.8 mg/dL) at the end of the *trans*-diet, for a difference of 0.36 mmol/L (95% CI, 0.26–0.46, $P < 0.0001$) (Fig. 1). Serum LDL cholesterol did not differ between the diet periods and was 3.05 ± 0.81 mmol/L at the end of the Sat-diet and 3.04 ± 0.80 mmol/L at the end of the *trans*-diet ($P = 0.64$). Serum triglycerides were slightly lower at the end of the Sat-diet, with a concentration of 0.90 ± 0.36 mmol/L, than at the end of the *trans*-diet, when the concentration was 0.98 ± 0.41 mmol/L ($P = 0.66$) (Fig. 1). The order of consumption of the two diets did not affect the change in HDL cholesterol: the mean change was 0.33 ± 0.26 mmol/L in subjects who switched from the *trans*-diet to the Sat-diet and 0.39 ± 0.31 mmol/L in the subjects who received the diets in the reverse order ($P = 0.5$).

The LDL/HDL ratio was significantly higher after the *trans*-diet, with a ratio of 2.2, than after the Sat-diet, when the ratio was 1.8 (difference -0.41 , 95% CI -0.54 to -0.27 , $P < 0.0001$).

DISCUSSION

We investigated whether the replacement of a margarine rich in *trans*-fatty acids with a margarine rich in lauric acid would produce a stronger increase in serum HDL cholesterol than the 0.15 mmol/L per 10% of energy that was estimated before (Mensink and Katan 1992). Indeed, serum HDL cholesterol was 0.36 mmol/L higher at the end of a 4-wk period in

which 9.2% of energy was provided by *trans*-fatty acids and 12.9% was provided by saturated fatty acids than after a 4-wk period in which energy intake from *trans*-fatty acids was 0% and that from saturated fatty acids was 22.9%. Although the diets did not provide equal amounts of polyunsaturated fatty acids (a difference of 2.2% energy), this difference could only account for ~ 0.015 mmol/L in HDL cholesterol according to the Mensink and Katan (1992) equation. Thus, lauric acid (or C12:0) appears to increase serum HDL cholesterol more than does myristic (or C16:0) and palmitic acid (or C14:0) (Sundram et al. 1997, Temme et al. 1996, Zock and Katan 1992). The major *trans*-fatty acids in our study were the (n-10), (n-9) and (n-11) isomers of C18:1, as is usual in partially hydrogenated soybean oil. These *trans*-fatty acids were also the major *trans*-fatty acids that were used in many other studies (Almendingen et al. 1995, Judd et al. 1994, Lichtenstein et al. 1993, Mensink and Katan 1990).

The difference in serum HDL cholesterol at the end of the two diets is in line with results of epidemiological studies that suggest that the risk of cardiovascular disease is increased more by the consumption of *trans*-fatty acids than by the consumption of saturated fatty acids (Ascherio et al. 1999). For example, in the Health Professionals follow-up study, the multivariate relative risk for myocardial infarction was 1.12 (95% CI, 0.97–1.28) for each 5% increase in intake of saturated fatty acids and 1.36 (95% CI, 1.03–1.81) for each 2% increase in intake of *trans*-fatty acids (Ascherio et al. 1996). In the Nurses' Health Study, the intake of foods rich in *trans*-fatty acids, such as margarines, was also significantly associated with a higher risk of coronary heart disease (Willett et al. 1993). In the same study, each increase of 5% in energy intake from saturated fat was associated with a multivariate relative risk of coronary heart disease of 1.17 (95% CI, 0.97–1.41). This was less than the relative risk associated with a 2% increase in energy intake from *trans*-fatty acids, which was 1.93 (95% CI, 1.43–2.61) (Hu et al. 1997). These studies show that the intake of saturated and especially *trans*-unsaturated fatty acids should be reduced to reduce the risk of coronary heart disease. Moreover, we found that the LDL/HDL ratio was significantly higher after the diet rich in *trans*-fatty acids than after the diet rich in saturated fatty acids, indicating a higher risk of coronary heart disease.

The consumption of saturated fatty acids and *trans*-fatty acids should not be encouraged. However, in products that require solid fats for their texture or firmness, the replacement of *trans*-fat with solid, tropical fats rich in lauric acids appears to be prudent.

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