Trans Fat Industry Coalition
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September 24, 2004

Ms. Kathryn McMurry
Department of Health and Human Services
Office of Disease Prevention and Health Promotion
Office of Public Health and Science
1101 Wootton Parkway, Suite LL100
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Dear Ms. McMurry:

These comments pertain to the report submitted to your office by the Dietary Guidelines Advisory Committee (the Committee) and reflect the opinions of the Trans Fat Coalition (the Coalition). The Coalition is a confederation of industry associations whose memberships have considerable technical expertise regarding the nutritional properties of *trans* fat and a keen interest in the dietary fats section of the 2005 Dietary Guidelines for Americans (DGA).

Executive Summary

The Trans Fat Coalition strongly objects to the conclusion of the Dietary Guidelines Advisory Committee that intake of *trans* fat in the United States be limited to one percent of total energy or less. This conclusion is not supported by the available scientific information, is inconsistent with the position taken by other scientific panels and may have unintended public health consequences.

There are very few studies that have investigated the effect of *trans* fat on coronary heart disease (CHD) risk factors at levels at or below the current average daily intake in the U.S. of approximately 2.6% of total energy. Nevertheless, intervention trials using higher amounts of dietary *trans* fat suggest that there is no significant difference between the effect of saturated and *trans* fat on blood low-density lipoprotein cholesterol (LDL-C) concentrations at levels below approximately five percent of total energy (approximately twice the typical daily intake level). Similarly, these same trials suggest that *trans* fat does not affect serum high-density lipoprotein cholesterol (HDL-C) concentrations at levels below five percent of energy. In addition, the observational studies show that *trans* fat is only associated with CHD incidence at the highest levels of intake.

The Institute of Medicine's Daily Reference Intake (DRI) panel on macronutrients rejected the notion of a quantitative limit on *trans* fat intake, and the Nutrition Subcommittee of the Food and Drug Administration's Food Advisory Committee

concluded that there was insufficient scientific evidence to support a recommendation to limit *trans* fat intake to one percent of total energy. The National Heart, Lung and Blood Institute's National Cholesterol Education Program, the American Heart Association and the American Diabetes Association have also not made quantitative recommendations for *trans* fat intake.

Finally, the imposition of a strict quantitative limitation on *trans* fat intake by the Dietary Guidelines for Americans is likely to cause many consumers to increase saturated fat intake in an over zealous effort to eliminate sources of *trans* fat from their diets. As emphasized in the Committee's report, the current high intake of saturated fat in the U.S. dictates that it be the primary focus of dietary modification. In that regard, there are very few assurances that their *trans* fat recommendation will not have unintended public health consequences for American consumers.

The Coalition recommends that the Departments engage the appropriate stakeholders to gather additional nutritional and dietary data so that a scientifically defensible recommendation regarding *trans* fat intake can be made in the future.

Introduction

The Committee unanimously supported the following conclusive statement with respect to *trans* fat,

> The relationship between *trans* fatty acid intake and LDL cholesterol is direct and progressive, increasing the risk of CHD. *Trans* fatty acid consumption by all population groups should be kept as low as possible, which is about 1 percent of energy intake or less.

The Coalition emphatically disagrees with the Committee's recommendation to limit *trans* fat in the diet to "about 1% of energy intake or less" because it cannot be justified on the basis of available scientific information. Sustained, high intake of *trans* fat can increase the risk of coronary heart disease (CHD) by increasing the concentration of LDL-C and/or decreasing the concentration of HDL-C in the blood. However, there are very few data on the effect of *trans* fat at or below the average daily U.S. intake, and considerable evidence that a threshold exists for its effect on HDL-C.

Furthermore, the Coalition questions the Committee's conclusion that there is a progressive, dose-dependent relationship between *trans* fat intake and the LDL:HDL cholesterol ratio in the range of 0.5 to 10 percent of calories. The Committee did not provide specific literature citations to document this relationship (particularly at the low end of the range), and we are aware of no data that demonstrate intakes of *trans* fat up to 3.3 percent of total energy have a significant effect on either LDL-C or HDL-C (see discussion below).

Even if data were available to prove the existence of a linear relationship between *trans* fat intake and risk of CHD in the range typical of U.S. dietary intakes, there is currently no objective basis on which to establish a minimum recommended intake. Dietary patterns are complex, and the establishment of a quantitative limitation for *trans* fat will only promote public health if consumers respond to it by decreasing their intake of *trans* and saturated fats. However, the Committee provided no evidence that a recommendation to limit *trans* fat intake to one percent of calories or less would achieve such a reduction, or that it would be sustainable given the nature of the food supply and the consumer's capacity to understand and implement it. There is a very real possibility that consumers will focus on the elimination of *trans* fat rather than the more pressing need to reduce the intake of saturated fat. A one percent of calories cutoff level may be less effective than a two or three percent value depending on how dietary selections are affected. Until such tradeoffs are understood, the establishment of a quantitative cutoff point for *trans* fat intake is arbitrary, and its impact on public health is unknown.

Finally, the DRI macronutrient panel (Institute of Medicine, 2002) did not establish a quantitative limit on *trans* fat intake but recommended that it be "as low as possible while consuming a nutritionally adequate diet". This panel observed that efforts to eliminate dietary *trans* fat could introduce "undesirable effects" that could compromise the nutritional quality of the diet and lead to "unknown and unquantifiable health risks". This position was reiterated by the Food and Nutrition Board's Committee on Use of Dietary Reference Intakes in Nutrition Labeling (Institute of Medicine, 2003) who concluded it would be inappropriate to establish a DRI for saturated and *trans* fat until additional "experimental data on acceptable diets that contain minimal levels of these food components" are available. The Committee, in its report, did not acknowledge the IOM's concern in this area, and to the best of our knowledge no such data have become available. A quantitative recommendation in the 2005 DGA would usurp those of the IOM panels assigned to deliberate this issue.

In conclusion, we urge the Departments to avoid incorporating a quantitative recommendation for *trans* fat intake in the final DGA document. To do so at this time would be premature in light of the current scientific and consumer uncertainties. We recommend that the Departments engage the appropriate stakeholders to resolve such uncertainties so that a scientifically-defensible, consumer-beneficial recommendation can be made in a future DGA. Our rationale for this recommendation is provided below.

Individual intervention studies suggest there is a threshold for the effect of *trans* fat on serum HDL-C

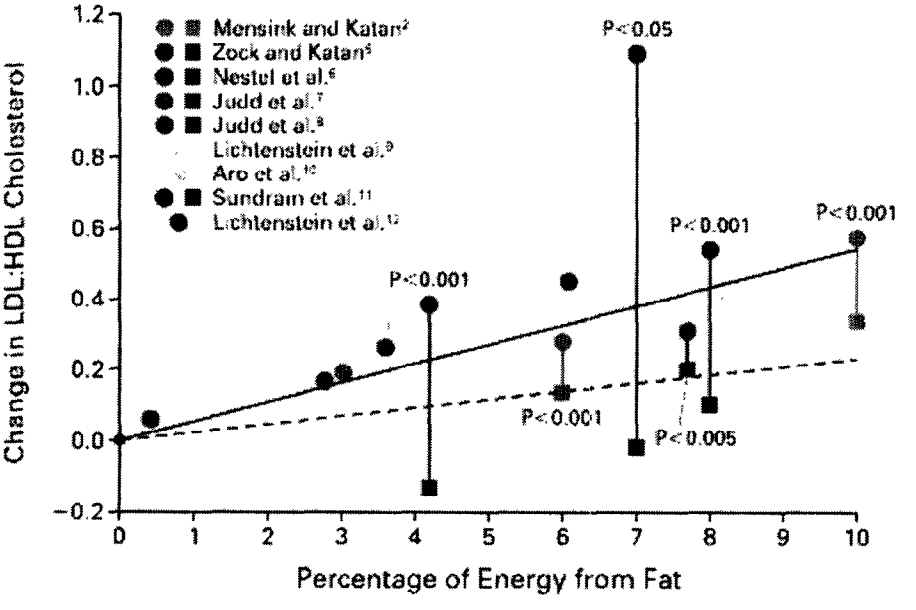
The Committee considered intervention studies cited by the macronutrient DRI panel (Institute of Medicine, 2002) as well as several more recent publications (Lovejoy *et.al.*, 2002; de Roos *et.al.*, 2001, 2002, 2003) in assessing the effect of *trans* fat on blood LDL-C and HDL-C concentrations. The interpretation of these studies appeared to be heavily influenced by a commentary published by Ascherio *et.al.* (1999). This commentary included a plot of the change in LDL:HDL ratio vs. intake of saturated and *trans* fat from

nine randomized, controlled feeding studies (see Figure 1). Linear regression analysis of these studies showed that the slope for *trans* fat was significantly greater than that for saturated fat. The Committee used this analysis as the primary source of evidence that *trans* fat is deleterious even at very low intakes.

The Coalition believes that this analysis does not provide compelling evidence that low levels of *trans* fat is detrimental, and that it cannot be used to support the recommendation to limit intake to one percent of calories or less. The regression line is heavily influenced by data from high intakes of *trans* fat because there is a paucity of data at intakes below those typical in the U.S. In addition, Ascherio *et.al.* assumed a linear relationship with no threshold by extrapolating the regression lines through the origin. Furthermore, this analysis does not consider the separate effects of dietary fatty acids on blood LDL-C and HDL-C concentrations. As the Committee observed, both *trans* and saturated fat are associated with increased LDL-C while only *trans* fat has been shown to lower HDL-C. However, by considering only the LDL:HDL ratio, it is not possible to determine how each parameter contributes to the overall effect.

Figure 1

Results of Randomized Studies of the Effects of a Diet High in *Trans* Fatty Acids (Circles) or Saturated Fatty Acids (Squares) on the Ratio of LDL Cholesterol to HDL Cholesterol.



A diet with isocaloric amounts of *cis* fatty acids was used as the comparison group. The solid line indicates the best-fit regression for *trans* fatty acids. The dashed line indicates the best-fit regression for saturated fatty acids.
Source: Ascherio *et.al.* (1999).

The Coalition believes that a much more meaningful assessment of the literature can be made by examining the separate effects of *trans* fat on serum LDL-C and HDL-C.

Table 1 summarizes the change in blood LDL-C and HDL-C in response to substitution of *trans* for *cis* fats in the randomized feeding studies included in the Ascherio *et.al.* analysis. The studies summarized in this table have been ranked according to percent of energy from *trans* fat provided in the experimental diets. The level of statistical significance reported by the authors when comparing the *trans* fat diets to their respective control groups is also provided.

Trans fat intake ranged from 0.91 to 11.0% of total energy. Only one of the experimental diets (Lichtenstein *et.al.*, 1999) used an intervention below the current estimated average U.S. daily intake of 2.6% of total energy cited by the Committee (Allison *et.al.*, 1999). The scarcity of data that reflect typical diets is evident from the fact that there are no studies between 0.91 and 3.3% of total energy from *trans* fat.

The data summarized in Table 1 strongly suggest there is a threshold for the effect of *trans* fat on blood HDL-C concentrations. Seven of the 10 experimental diets (ranging from 0.91 to 7.1% of energy from *trans* fat) reported no significant effect on HDL-C. Only diets with *trans* fat concentrations above this range consistently showed a significant effect. More recent studies not included in the Ascherio analysis by Louherantra *et.al.* (1999) and Lovejoy *et.al.* (2002) provide further evidence of such a threshold. These studies found no significant effect of *trans* fat on blood HDL-C concentrations at 5.1 and 7.3 percent of total energy, respectively.

In summary, this direct assessment of the data does not support the Committee's conclusion that low *trans* fat diets (≤1% of energy) are necessary to manage the risk of CHD. It is clear that the regression line for *trans* fat reported by Ascherio *et.al.* was heavily influenced by its effect on HDL-C at very high intake levels (up to 4.2 times the average daily U.S. intake), and that it ignored evidence of a likely threshold level for this effect at approximately five percent of total energy. Allison *et.al.* (1999) have shown that the 90th percentile intake of *trans* fat in the American diet falls below this apparent threshold – a fact that fails to support the Committee's overall conclusions.

Additional evidence of a threshold for the effect of *trans* fat on blood HDL-C concentrations was provided by a rigorous assessment of the literature commissioned by the International Life Sciences Institute (ILSI). This assessment was submitted to the Food and Drug Administration (FDA) in response to an advanced notice of proposed rulemaking (68 FR 41507, July 11, 2003) pertaining to *trans* fat labeling and is appended to this document.

This assessment utilized data from 16 randomized intervention studies in which 17 control/comparison (control) and 27 treatment *trans* fatty acid (TFA) intake levels were identified (Almendingen *et.al.*, 1995; Aro *et.al.*, 1997; Denke *et.al.*, 2000; de Roos *et.al.*, 2001; Judd *et.al.*, 1994, 1998, 2002; Lichtenstein *et.al.*, 1993, 1999; Mensink *et.al.*, 1990; Nestel *et.al.*, 1992; Noakes and Clifton, 1998; Sundram *et.al.*, 1997; Wood *et.al.*, 1993,

Table 1

Change in Blood LDL-C and HDL-C in Response to Substitution of *Trans* for *Cis* Fatty Acids

Dietary *Trans* fat (% of energy)	**Source of *Trans* fat**	**ΔLDL-C (mM)**	**Statistical Significance (p-value)**	**ΔHDL-C (mM)**	**Statistical Significance (p-value)**	**Reference**
0.91	Semi-liquid margarine (partially hydrogenated soybean oil)	0.025	NSD	0	NSD	Lichtenstein *et.al.* (1999)
3.3	Soft margarine (partially hydrogenated soybean oil)	0.13	NSD	0	NSD	Lichtenstein *et.al.* (1999)
3.8	Elaidic acid (mixed sources)	0.02	<0.05	-0.02	NSD	Judd *et.al.* (1994)
4.2	Shortening (partially hydrogenated soybean oil)	0.26	<0.05	0	NSD	Lichtenstein *et.al.* (1999)
4.2	Partially hydrogenated corn oil margarine	0.26	NSD (0.058)	-0.03	NSD (0.373)	Lichtenstein *et.al.* (1992)
4.2	Elaidic acid (mixed sources)	0.37	<0.01	-0.07	<0.01	Judd *et.al.* (2002)
6.6	Elaidic acid (mixed sources)	0.26	<0.05	-0.04	<0.05	Judd *et.al.* (1994)
6.7	Stick margarine (partially hydrogenated soybean oil)	0.36	<0.05	-0.025	NSD	Lichetnstein *et.al.* (1999)
6.9	Partially hydrogenated soybean oil	0.64	<0.05	-0.20	<0.05	Sundram *et.al.* (1997)
7.1	Elaidic acid (hardened canola/palmolein)	0.36	<0.001	0	NSD	Nestel *et.al.* (1992)
7.7	Partially hydrogenated hi-oleic sunflower oil	0.24	<0.02	-0.10	<0.02	Zock and Katan (1992)

Dietary *Trans* fat (% of energy)	Source of *Trans* fat	ΔLDL-C (mM)	Statistical Significance (p-value)	ΔHDL-C (mM)	Statistical Significance (p-value)	Reference
8.3	Elaidic acid (mixed sources)	0.41	<0.01	-0.08	<0.01	Judd *et.al.* (2002)
8.7	Partially hydrogenated sunflower oil	0.24	<0.05	-0.20	<0.05	Aro *et.al.* (1997)
11.0	Partially hydrogenated hi-oleic sunflower oil)	0.37	<0.0001	-0.17	<0.0001	Mensink and Katan (1990)

1993a; Zock and Katan,1992). All fatty acid intakes, when not reported as percent of energy (%En), were converted to these units, thereby permitting study comparisons on a similar basis. Also, LDL-C and HDL-C values expressed as mg/dL were converted to mM.

This analysis shows that *trans* and saturated fatty acids (SFA) have similar effects on serum LDL-C concentrations and that *trans* fat does not differentially impact serum HDL-C concentrations compared to similar intakes of saturated fat. The specific bases for these conclusions are provided below:

- Intake of *trans* fat does not differentially impact serum LDL-C compared to similar intakes of saturated fat

Figures 2 and 3, respectively, plot changes in TFA intake (%En) against changes in LDL-C in relative (%) and absolute terms. Figures 4 and 5, respectively, plot changes in SFA intake (%En) against changes in LDL-C in relative (%) and absolute terms. There are two key points to note. First, in all cases the slopes of the lines are similar. This observation strongly suggests that the impact on serum LDL-C of TFA intake and SFA intake are essentially indistinguishable. Second, higher order predictive equations provide very little additional explanation of the variance, suggesting that a linear regression is a reasonable model for these data (r^2 coefficients are provided for first, second and fourth order equations as examples, though the biological relevance of a fourth order equation may be difficult to interpret).

In summary, the data do not permit a meaningful distinction between the intake of TFA and SFA with respect to any differential impact on LDL-C. Consequently, the differential effects between *trans* and saturated fat is due exclusively to their effect on blood HDL-C concentrations.

- *Trans* fat does not have a significant impact on blood HDL-C concentrations, compared to saturated fatty acids at intakes less than 5% of total calories

Figures 6 and 7, respectively, plot changes in TFA intake (%En) against changes in HDL-C in relative (%) and absolute terms. However in contrast to plots of LDL-C, higher order equations provide significantly greater predictive value, explaining a greater proportion of the variance. Most intriguing is the finding that there appears to be little impact on serum HDL-C when TFA intake is less than 5% En, when a second or fourth order equation is employed. Above this threshold, there is a clear inverse relationship, with increasing TFA intakes resulting in decreased serum HDL-C. Not surprisingly, a simple linear regression as reported by Ascherio *et.al.* (1999) has negative slope, but this analysis is a poor model of the data because the relationship is better explained by higher order equations.

SFA intake (%En) appears to show no such threshold effect on serum HDL-C, in fact showing very little effect at all (figures 8 and 9).

Figure 2

Percent change in LDL cholesterol vs. change in *trans* fat intake. Lines plotted represent first, second and fourth order equations, with r^2 coefficients presented in that order.

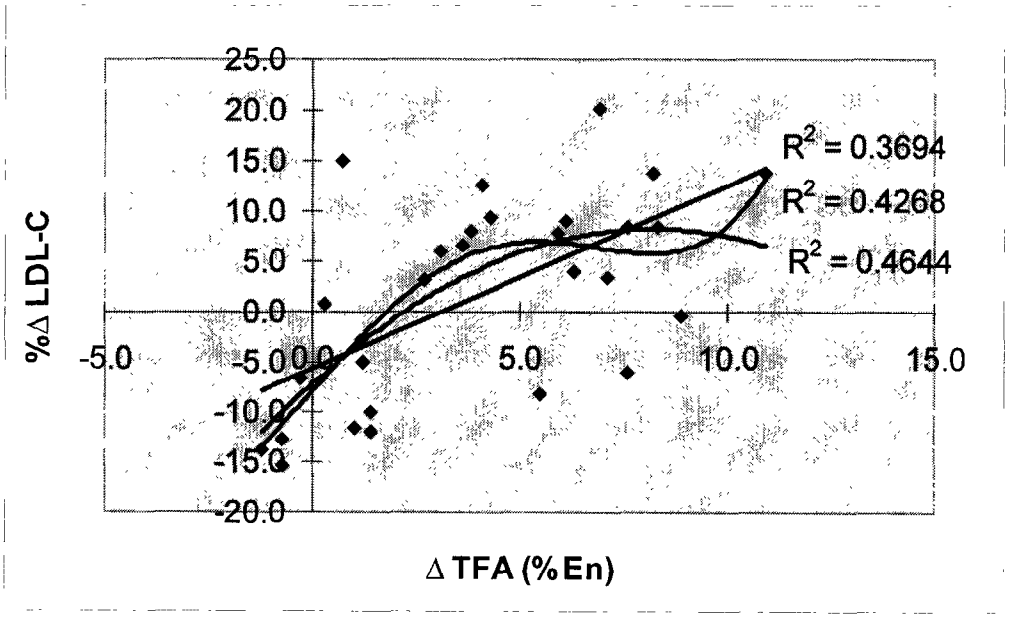


Figure 3

Absolute change in LDL cholesterol vs. change in *trans* fat intake. Lines plotted represent first, second and fourth order equations, with r^2 coefficients presented in that order.

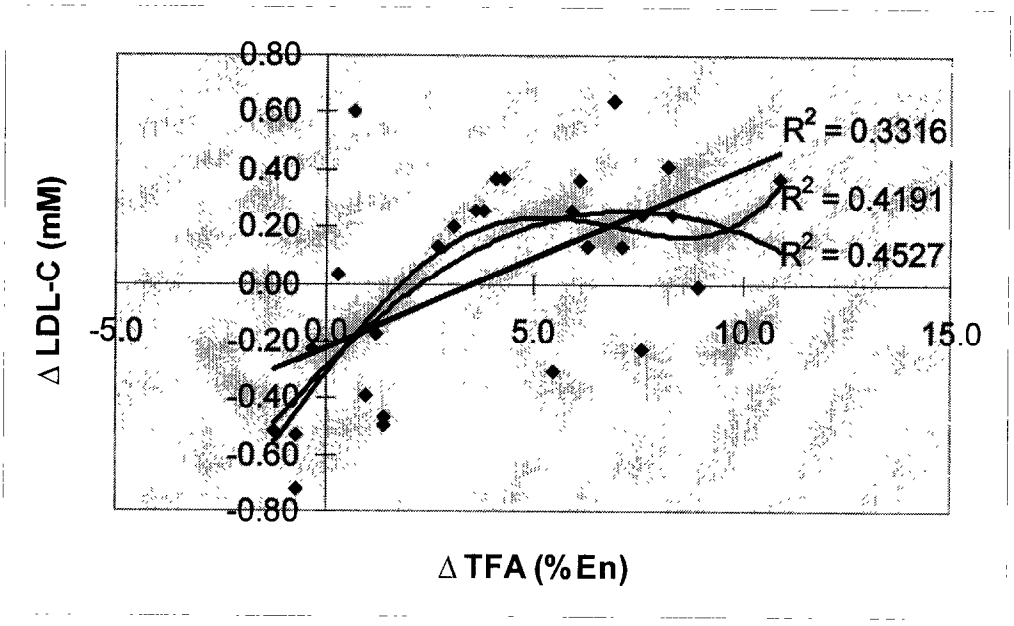


Figure 4

Percent change in LDL cholesterol vs. change in saturated fat intake. Lines plotted represent first, second and fourth order equations, with r^2 coefficients presented in that order.

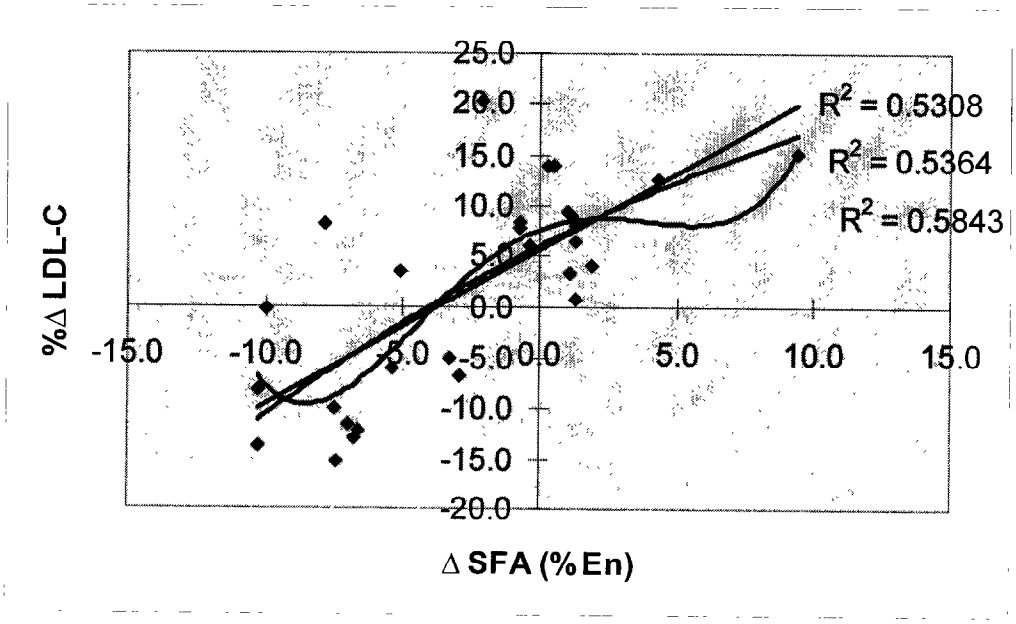


Figure 5

Absolute change in LDL cholesterol vs. change in saturated fat intake. Lines plotted represent first, second and fourth order equations, with r^2 coefficients presented in that order.

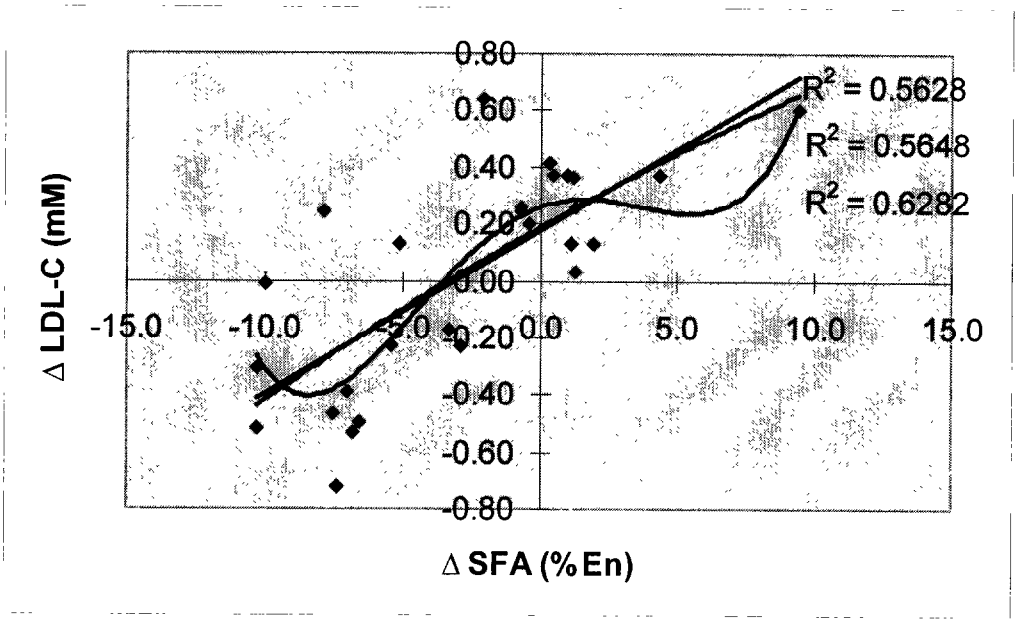


Figure 6

Percent change in HDL cholesterol vs. change in *trans* fat intake. Lines plotted represent first, second and fourth order equations, with r^2 coefficients presented in that order.

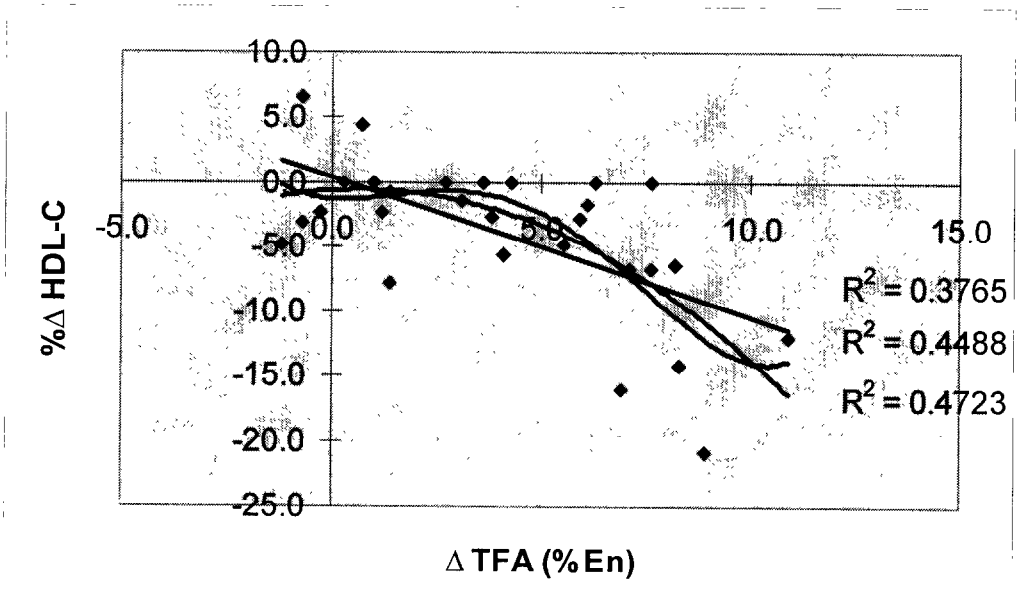


Figure 7

Absolute change in HDL cholesterol vs. change in *trans* fat intake. Lines plotted represent first, second and fourth order equations, with r^2 coefficients presented in that order.

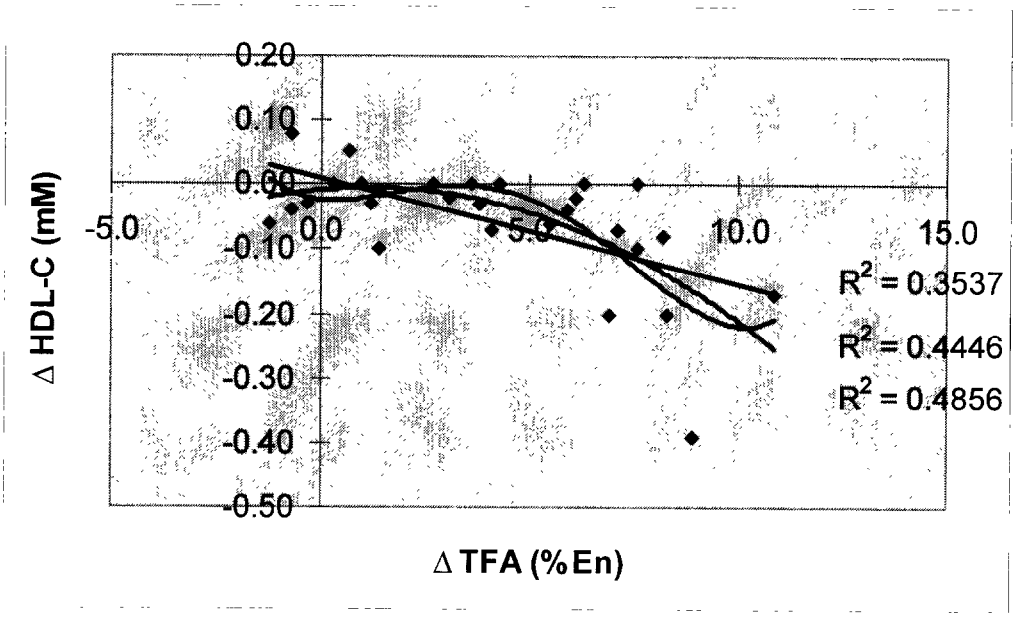


Figure 8

Percent change in HDL cholesterol vs. change in saturated fat intake. Lines plotted represent first, second and fourth order equations, with r^2 coefficients presented in that order.

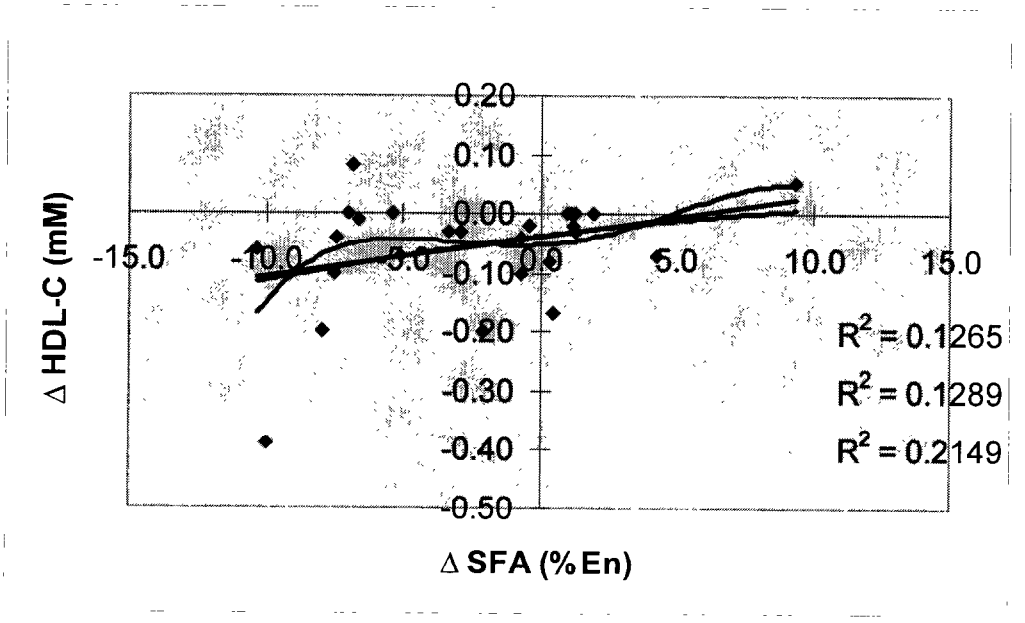
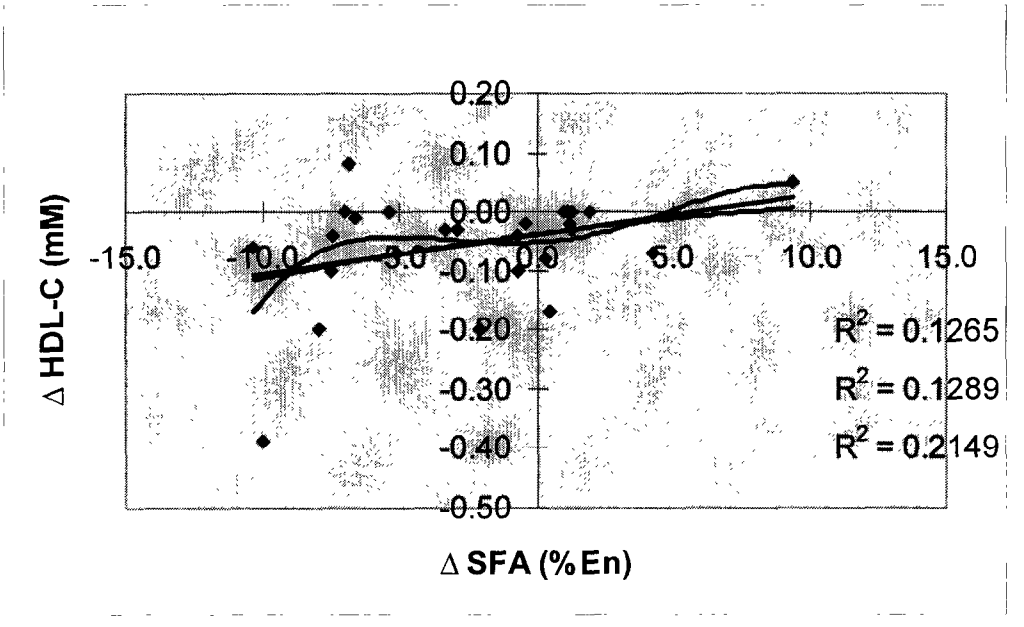


Figure 9

Absolute change in HDL cholesterol vs. change in saturated fat intake. Lines plotted represent first, second and fourth order equations, with r^2 coefficients presented in that order.



In summary, the data do not permit a meaningful distinction between the intake of TFA and SFA with respect to any differential impact on HDL-C, when TFA intake is less than 5% of total energy intake (5% En). In addition, as noted previously, the current U.S. average daily intake of *trans* fat (2.6 % En) acknowledged by the Committee (Allison *et.al.*, 1999) is substantially below the threshold suggested by the available data.

The Coalition strongly believes that this thorough analysis of the existing scientific information fails to support the Committee's recommendation to limit *trans* fat intake to one percent of calories or less, and we urgently recommend that the 2005 DGA not provide a quantitative recommendation with respect to *trans* fat intake.

Observational data also fail to support the recommendation to limit *trans* fat to one percent or less of total calories

Observational studies provide weaker evidence than randomized, controlled intervention studies because they are not capable of demonstrating a cause and effect relationship. Nevertheless, such studies are useful for identifying associations in free-living populations and were considered by the Committee in developing their recommendations in the area of *trans* fat and CHD.

The Committee noted that six cohort studies cited by the DRI Macronutrient Committee (Institute of Medicine, 2002) suggest that "high *trans* fat intake is associated with an increased risk of coronary artery disease" (Ascherio *et.al.*, 1996; Gilman *et.al.*, 1007; Hu *et.al.*, 1997; Kromhout *et.al.*, 1995; Pietinen *et.al.*, 1997; Willett *et.al.*, 1993). Two case-control studies (Ascherio *et.al.*, 1994; Tavini *et.al.*, 1997) were also included in the IOM table.

Table 2 summarizes the studies noted above that reported relative risk (RR) ratios for *trans* fat intake and incidence of CHD. Without exception, these studies show that the significant associations occur only at the highest level of *trans* fat consumption after adjustment for common CHD risk factors. This observation is consistent with the analysis of dietary intervention studies discussed above that shows high levels of dietary *trans* fat is necessary before serum HDL-C concentrations are affected, and that the effect of *trans* and saturated fat is similar below this threshold. Clearly, the epidemiological data do not provide a compelling justification for the Committee's recommendation that *trans* fat intake be restricted to one percent of energy or less.

Positions taken by other outside groups

The Coalition believes there is little precedent for the Committee's recommendation to restrict intake of *trans* fat to one percent of calories or less from other organizations. As the Committee observed, the Macronutrient DRI Committee (Institute of Medicine, 2002) did not issue a quantitative benchmark (i.e. Upper Level) to limit *trans* fat consumption.

Table 2

Summary of Observational Studies that Reported Relative Risks for CHD Incidence by Trans Fatty Acid Consumption Category

Reference	*Trans* fatty acid intake of upper category	Adjustments for confounding variables	Relative Risk of Myocardial Infarction (95% Confidence Interval)				
			1	2	3	4	5
Ascherio *et.al.* (1993)	6.51 g/d	Age, gender	1.0	0.59 (0.31, 1,12)	1.18 (0.66, 2.13)	1.41 (0.79, 2.50)	2.14* (1.24, 3.68)
		Multiple CHD risk factors	1.0	0.73 (0.37, 1.44)	1.24 (0.66, 2.32)	1.63 (0.88, 3.0)	2.28 (1.28, 4.08)
		Above plus other fatty acids	1.0	0.63 (0.31, 1.30)	1.03 (0.53, 2.00)	1.35 (0.69, 2.63)	2.02 (1.03, 3.93)
Ascherio *et.al.* (1996)	4.3 g/d	Age	1.0	1.24 (0.97, 1.59)	1.33 (1.04, 1.70)	1.40 (1.10, 1.78)	1.57 (1.24, 1.98)
		Multiple CHD risk factors	1.0	1.20 (0.93, 1,54)	1.24 (0.97, 1.60)	1.27 (0.99, 1.63)	1.40 (1.10, 1.79)
		Above plus dietary fiber	1.0	1.12 (0.86, 1.44)	1.12 (0.87, 1.46)	1.12 (0.86, 1.46)	1.21 (0.93, 1.58)
Hu *et.al.* (1997)	2.9 % En	Age	1.0	1.07 (0.86, 1.32)	1.21 (0.98, 1.49)	1.21 (0.99, 1.49)	1.32 (1.09, 1.64)
		Multiple CHD risk factors	1.0	1.07 (0.86, 1.33)	1.10 (0.89, 1.37)	1.13 (0.91, 1.39)	1.27 (1.03, 1.56)
		Above plus other fatty acids	1.0	1.09 (0.87, 1.37)	1.16 (0.91, 1.47)	1.24 (0.96, 1.60)	1.53 (1.16, 2.02)
Pietinen *et.al.* (1997)	6.2 g/d	Age	1.0	1.13 (0.95, 1.34)	1.02 (0.86, 1.22)	1.14 (0.96, 1.34)	1.19 (1.00, 1.41)
		Multiple CHD risk factors	1.0	1.10 (0.93, 1.31)	0.97 (0.82, 1.16)	1.07 (0.90, 1.28)	1.14 (0.96, 1.35)

Tavani *et.al.* (1997)	Undefined margarine consumption categories	None	1.0	1.5 (1.0. 2.2)			
Willett *et.al.* (1993)	5.7 g/d	Age	1.0	1.15 (0.85, 1.56)	1.03 (0.74, 1.42)	1.16 (0.85, 1.59)	1.50 (1.12, 2.00)
		Multiple CHD risk factors	1.0	1.12 (0.82, 1,52)	0.97 (0.71, 1.36)	1.12 (0.82, 1.54)	1.35 (1.00, 1.82)
		Above plus other fatty acids	1.0	1.15 (0.83, 1.59)	1.03 (0.72, 1.48)	1.22 (0.83, 1.78)	1.57 (1.05, 2.34)
		Above plus other fatty acids and multiple vitamin use	1.0	1.12 (0.81, 1.55)	0.99 (0.69, 1.43)	1.16 (0.80, .170)	1.47 (0.98, 2.20)

*Shaded cells indicate relative risk ratios that are statistically significant ($p<0.05$).

Similarly, although the desirability of reducing *trans* fat intake was recognized, quantitative recommendations were not issued by the National Cholesterol Education Program (2002) in its Adult Treatment Panel III report, or by the American Diabetes Association (Franz *et.al.*, 2004) or the American Heart Association (Kraus *et.al.*, 2000).

Furthermore, we believe the Committee egregiously mischaracterized the recent conclusions of the Nutrition Subcommittee of FDA's Food Advisory Committee. The statement regarding limiting *trans* fat quoted in the Committee's report was a secondary position passed after the Subcommittee concluded that there was insufficient scientific information to justify a strict, quantitative recommendation. The formal question that was addressed according to the transcript of this group's April 28, 2004 meeting reads,

> The Dietary Guidelines Committee may suggest that less than 1 percent of energy should be obtained from trans fat (2 grams per day for a 2,000 kcal diet). Does the scientific evidence support this level?

The Subcommittee voted "no" to this question by a vote of 5 to 3.

As the Committee observed, the Danish Nutrition Council (Stender and Dyerbery, 2003) recommended that the use of "industrially produced" *trans* fat be discontinued based largely on the prospective observational studies and the Ascherio *et.al.* (1999) analysis of nine dietary intervention trials. As discussed above, the Coalition believes this interpretation of the data is incorrect. In addition, the Committee has already rejected the notion that "industrial sources" of *trans* fat be eliminated because it would be impractical (if not impossible) to do so given the constraints of food technology.

The Dietary Guidelines Committee report cites the World Health Organization Report (WHO, 2003) as recommending a quantitative (i.e. 1% of energy) limit for *trans* fat intake. This report included a general review of the observational and dietary intervention literature, but did not provide a specific rationale for their quantitative recommendation. The evidence cited by the WHO report has the same scientific limitations as that used by the Committee. These limitations contributed to the macronutrient DRI panel's decision not to establish quantitative benchmarks for *trans* fat intake.

Public health concerns

The ultimate goal of the DGA is to provide the American population with the best information possible to assist in the selection of a healthy diet. Goals should be established that will have the greatest impact on public health. It is well known that both *trans* fat and saturated fat increases serum LDL-C. Although data are limited *trans* fat intakes above a threshold of approximately five percent of energy appear to have a negative impact on HDL cholesterol while intakes below this level have little impact. Published data (Allison *et.al.*, 1999) indicate that the average intake of *trans* fat in the American diet is 2.6% of energy and the 90^{th} percentile falls below five percent of total energy.

As noted previously, even if data in the range of *trans* fat intakes typical in the U.S. were available to show that there is a linear relationship with risk of CHD, we believe it would be premature to establish a quantitative dietary recommendation at this time. The cutoff point for such a recommendation cannot be objectively determined without a more thorough understanding of how it would affect consumer behavior in the context of the food supply.

The Coalition is concerned that the attention that will be directed toward *trans* fat if the Committee's recommendation is enacted will distract from the well-established need to address saturated fat intake. For example, replacing butter with *trans* fat-containing margarine results in an improvement in serum lipid profiles (Chisholm *et.al.*, 1996; Denke, *et.al.*, 2000; Judd *et.al.*, 1998). However, paranoia about *trans* fat generated by the DGA could easily prompt consumers to make poor dietary choices. The Committee also expressed concern regarding this issue,

> Although intakes of saturated fat, *trans* fat and cholesterol all should be decreased, because saturated fat consumption is proportionately much greater than that of these other fats, saturated fat should be the primary focus of dietary modification.

We agree completely with this concern, and believe that the Committee's recommendation for limiting intake of *trans* fat will exacerbate it.

Food Industry Actions to Reduce *Trans* Fat in the Diet

The U.S. food industry has been very responsive to public health concerns about *trans* fat, rapidly developing many new food products that contain no *trans* fat or significantly reduced *trans* fat. These products are the result of considerable investment in product reformulations and new processing techniques. These efforts continue on a fast track. Other methods by which dietary *trans* fat will be further reduced in the future include the development of new oilseed varieties, the oils from which will be more stable and not require hydrogenation to make them technologically acceptable. But the promise of these oils is not yet reality. It would be a major step backward for public health and an enormous disservice to the American consumer if *trans* fat was simply replaced with greater levels of saturated fat in the diet.

Care must be taken to avoid unintended consequences in making dietary choices. The Committee clearly emphasized a much higher relative concern about reducing the intake of saturated fat (as noted above), and there are very few assurances that a quantitative limitation of *trans* fat intake will not be counterproductive from a public health perspective.

Efforts by industry to remove, or otherwise minimize to the extent possible, *trans* fat in food products along with quantitative labeling pursuant to a final rule on *trans* fat labeling will have a much more positive impact on consumer nutrition than will the confusion created, including the likely consumption of higher levels of saturated fat, if inappropriate and scientifically-unsupportable, quantitative *trans* fat levels are included in the 2005 Dietary Guidelines for Americans.

Summary and conclusions

The Trans Fat Coalition respectfully recommends that the 2005 Dietary Guidelines for Americans refrain from providing a quantitative recommendation for limiting intake of *trans* fat. This recommendation is based on the following facts:

- There are virtually no experimental data on the effect of *trans* fat on serum lipid biomarkers at levels at or below current average daily intakes in the United States.

- The prospective observational studies consistently show a positive association between *trans* fat and CHD, but only at the upper intake levels.

- Informal inspection and more rigorous statistical analysis of the existing dietary intervention studies suggest that *trans* fat does not lower blood HDL-C concentrations when substituted for saturated fatty acids unless intakes substantially exceed current estimated amounts.

- The Nutrition Subcommittee of FDA's Food Advisory Committee concluded that there is insufficient science to support limiting *trans* fat to one percent of energy or lower.

- Two recent National Academies Institute of Medicine panels concluded that it is inappropriate to establish a quantitative benchmark for *trans* fat intake until more data are available on the nutritional adequacy of diets largely devoid of *trans* fat-containing foods. Such data should be made publicly available for thorough review.

- The Committee's proposed quantitative recommendation regarding *trans* fat is likely to have unintended consequences by detracting from consumer's efforts to reduce the intake of saturated fat.

- The Coalition recommends that the Departments spearhead an effort to resolve the current uncertainties regarding the scientific evidence and consumer response so that a well-considered quantitative recommendation for *trans* fat can be made in the future.

The Coalition very much appreciates the opportunity to provide these comments, and would be pleased to answer any questions the Departments may have.

Respectfully submitted,

American Bakers Association
Grocery Manufacturers of America
Institute of Shortening and Edible Oils
National Association of Margarine Manufacturers

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REFERENCES

Allison, B.D., Egan, S.K., Barraj, L.M., Caughman, C., Infante, M. and Heimbach, J.T. 1999. Estimated intakes of *trans* fatty acids in the US population. *J. Am. Diet. Assn.* 99:166.

Almendingen, K., Jordal, O., Kierulf, P., Sandstad, B. and Pedersen J.I. 1995. Effects of partially hydrogenated fish oil, partially hydrogenated soybean oil, and butter on serum lipoproteins and Lp(a) in men. *J. Lipid Res.* 36:1370.

Aro A., Jauhiainen M., Partanen R., Salminen, I. and Mutanen, M. 1997. Stearic acid, trans fat, and dairy fat: effects on serum and lipoprotein lipids, apolipoproteins, lipoprotein(a), and lipid transfer proteins in healthy subjects. *Am. J. Clin. Nutr.* 65:1419.

Ascherio, A., Hennekens, C.H., Buring, J.E., Master, C., Stampfer, M.J. and Willett, W.C. 1994. *Trans*-fatty acids intake and risk of myocardial infarction. *Circulation* 89:94.

Ascherio, A., Rimm, E.B., Spiegelman, D. and Stampfer, M. 1996. Dietary fat and risk of coronary heart disease in men: cohort follow-up study in the United States. *Br. Med. J.* 313:84.

Ascherio, A., Katan, M.B., Zock. P.L., Stampfer, M.J. and Willett, W.C. 1999. Trans fatty acids and coronary heart disease. *New Eng. J. Med.* 340:1994.

Chisholm, A., Mann, J., Sutherland, W., Duncan, A., Skeaff, M and Frampton, C. 1996. Effect on lipoprotein profile of replacing butter with margarine in a low fat diet: randomized crossover study with hypercholesterolaemic subjects. *Br. Med. J.* 312:931.

Denke, M.A., Adams-Huet, B. and Nguyen, B.S. 2000. Individual cholesterol variation in response to a margarine- or butter-based diet--a study in families. *J. Am. Med. Assn.* 284:2740.

de Roos, N.M., Bots, M.L. and Katan, M.B. 2001. Replacement of dietary saturated fatty acids by *trans* fat lowers serum HDL Cholesterol and impairs endothelial function in healthy men and women. *Arterioscler. Thromb. Vasc. Biol.* 21:1233.

de Roos, N.M., Schouten, E.G., Scheek, L.M., van Tol, A. and Katan, M.B. 2002. Replacement of dietary saturated fat with *trans* fat reduces serum paraoxonase activity in healthy men and women. *Metabolism* 51:1534.

de Roos, N.M., Schouten, E.G. and Katan, M.B. 2003. Trans fat, HDL-cholesterol, and cardiovascular disease. Effects of dietary changes on vascular reactivity. *Eur. J. Med. Res.* 8:355.

Franz, M.J., Bantle, J.P., Beebe, C.A., Brunzell, J.S., Chiasson, J.L., Garg, A., Holzmeister, L.A., Hoogwerg, B., Mayer-Davis, E., Mooradian, A.D., Purnell, J.Q. and Wheeler, M. 2004. American Diabetes Association. Nutrition principles and recommendations in diabetes. *Diabetes Care* 27:S36.

Gillman, M.W., Cupples, A., Gagnon, D., Millen, B.E., Ellson, R.C. and Castelli, W.P. 1997. Margarine intake and subsequent coronary heart disease in men. *Epidemiology* 8:144.

Hu, F.B., Stampfer, M.J., Manson, J.E., Rimm, E., Colditz, G.A., Rosner, B.A., Hennekens, C.H. and Willett, W. C. 1997. Dietary fat intake and the risk of coronary heart disease in women. *New Eng. J. Med.* 337:1491.

Institute of Medicine, National Academy of Sciences. 2002. "Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein and Amino Acids." National Academies Press, Washington, DC.

Institute of Medicine, National Academy of Sciences. 2003. "Dietary Reference Intakes: Guiding Principles for Nutrition Labeling and Fortification. National Academies Press, Washington, DC.

Judd, J.T., Clevidence, B.A., Muesing R.A., Wittes, J., Sunkin, M.E. and Podczasy, J.J. 1994. Dietary trans fat: effects on plasma lipids and lipoproteins of healthy men and women. *Am. J. Clin. Nutr.* 59:861.

Judd, J.T., Baer, D.J., Clevidence, B.A., Muesing, A., Chen, S.C., Weststrate, J.A., Meijer, G.W., Wittes, J., Lichtenstein, A.H., Vilella-Bach, M. and Schaefer, E.J. 1998. Effects of margarine compared with those of butter on blood lipid profiles related to cardiovascular disease risk factors in normolipemic adults fed controlled diets. *Am. J. Clin. Nutr.* 68:768.

Judd, J.T., Baer, D.L., Clevidence, BA, Kris-Etherton, P., Muesing, R.A. and Iwane, R.A. 2002. Dietary cis and trans monounsaturated and saturated fatty acids and plasma lipids and lipoproteins in men. *Lipids* 37:123.

Krauss, R.M., Eckel, R.H., Howard, B., Appel, L.J., Daniels, S.R., Deckelbaum, R.J., Erdman, J.W. Jr., Kris-Etherton, P., Goldberg, IlJ., Kotchen, T.A., Lichtenstein, A.H., Mitch, W.E., Mullis, R., Robinson, K., Wylie-Rosett, J., St Jeor, S., Suttie, J., Tribble, D.L. and Bazzarre, T.L. 2000. AHA Dietary Guidelines: revision 2000: A statement for healthcare professionals from the Nutrition Committee of the American Heart Association. *Circulation* 102:2284.

Kromhout, D., Menotti., A., Bloemberg, B., Aravanis, C., Blackburn, H., Buzina, R., Dontas, A.S., Fidanza, F., Giampaoli, S., Jansen, A., Karvonen, M., Katan, M., Nissinen, A., Nedeljkovic, S., Pekkanen, J., Pekkarinen, M, Punsar, S., Räsänen, L., Simic, B. and Toshima, H. 1995. Dietary saturated and *trans* fatty acids and cholesterol and 25-year mortality from coronary heart disease: The Seven Countries Study. *Prev. Med.* 24:308.

Lichtenstein, A.H., Ausman, L.M., Carrasco, W., Jenner, J.L., Ordovas. J.M. and Schaefer, E.J. 1993. Hydrogenation impairs the hypolipidemic effect of corn oil in humans. Hydrogenation, trans fat, and plasma lipids. *Arterioscler. Thromb.* 13:154.

Lichtenstein, A.H., Ausman, L.M., Jalbert, S.M. and Schaefer, E.J. 1999. Effects of different forms of dietary hydrogenated fats on serum lipoprotein cholesterol levels. *New Eng. J. Med.* 340:1933.

Lovejoy, J.C., Smith, S.R., Champagne, C.M., Most, M.M., Lefevre, M., DeLany, J.P., Denkins, Y.M., Rood, J.C., Veldhuis, J. and Bray, G.A. 2002. Effects of diets enriched in saturated (Palmitic), monounsaturated (Oleic), or *trans* (Elaidic) fatty acids on insulin sensitivity and substrate oxidation in healthy adults. *Ciabetes Care* 25:1283.

Mensink, R.P. and Katan, M.B. 1990. Effect of dietary trans fat on high-density and low-density lipoprotein cholesterol levels in healthy subjects. *New Eng. J. Med.* 323:439.

National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). 2002. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults final report. *Circulation* 106:3143.

Nestel, P.J., Noakes, M., Belling, G.B., McArthur, R., Clifton, P., Janus, E. and Abbey, M. 1992. Plasma lipoprotein lipid and Lp(a) changes with substitution of elaidic acid for oleic acid in the diet. *J. Lipid Res.* 33:1029.

Noakes, M. and Clifton, P.M. 1998. Oil blends containing partially hydrogenated or interesterified fats: differential effects on plasma lipids. *Am. J. Clin. Nutr.* 68:242.

Pietinen, P., Ascherio, A., Korhonen, P., Hartman, A.M., Willett, W.C., Albanes, D. and Virtamo, J. 1997. Intake of fatty acids and risk of coronary heart disease in a cohort of Finnish men. The alpha-tocopherol, beta-carotene cancer prevention study. *Am. J. Epi.* 145:876.

Stender, S. and Dyerbery, J. 2003. *The influence of trans fat on health.* A Report from the Danish Nutrition Council.

Sundram, K., Ismail, A., Hayes, K.C., Jeyamalar, Rl and Pathmanathan, R. 1997. Trans (Elaidic) fatty acids adversely affect the lipoprotein profile relative to specific saturated fatty acids in humans. *J. Nutr.* 127:514S.

Tavani, A., Negri, W., Avanzo, B.D'. and LaVecchia, C. 1997. Margarine intake and risk of nonfatal acute myocardial infarction in Italian women. *Eur. J. Clin. Nutr.* 51:30.

WHO Technical Report No. 916. 2003. *Diet, Nutrition and the Prevention of Chronic Diseases.* Geneva, Switzerland.

Willett, W.C., Stampher, M.J., Manson, J.E., Colditz, G.A., Speizer, F.E., Rosner, B.A., Sampson, L.A. and Hennekens, C.H. 1993. Intake of *trans* fatty acids and risk of coronary heart disease among women. *Lancet* 341:581.

Wood, R., Kubena, K., Tseng, S., Martin, G. and Crook, R. 1993. Effect of palm oil, margarine, butter, and sunflower oil on the serum lipids and lipoproteins of normocholesterolemic middle-aged men. *J. Nutr. Biochem.* 4:286.

Wood, R., Kubena, K., O'Brien, B., Tseng, S. and Martin, G. 1993a. Effect of butter, mono- and polyunsaturated fatty acid-enriched butter, trans fatty acid margarine, and zero trans fatty acid margarine on serum lipids and lipoproteins in healthy men. *J. Lipid Res.* 34:1.

Zock, P.L. and Katan, M.B. 1992. Hydrogenation alternatives: effects of trans fatty acids and stearic acid versus linoleic acid on serum lipids and lipoproteins in humans. *J. Lipid Res.* 33:399.