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Reduction in Dietary Trans Fat Intake is Associated with Decreased LDL Particle Number in a Primary Prevention Population

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Abstract

Background and Aims—Increased trans fat intake has been associated with an increased risk of cardiovascular disease (CVD). While the affect of trans fat on traditional lipids is known, it's association with LDL particle number (LDL-P), a novel marker of CVD risk, has not been established. The purpose of this study was to determine the association between trans fat intake and LDL-P over 1-year among individuals participating in a lifestyle intervention trial.

Methods and Results—Family members (n = 400, 33% male, mean age 48 ± 13) of patients hospitalized with CVD who participated in a 1-year randomized controlled primary prevention lifestyle intervention trial and had complete dietary data and LDL-P measures at baseline and 1-year. Change in trans fat as a percentage of total diet and mean absolute change in LDL-P at 1-year was assessed using multivariate adjusted linear regression models. At baseline, there was a significant positive correlation between dietary trans fat intake and LDL-P (Beta = 37, p = 0.04). For every 1 percent change in trans fat intake there was a 27 nmol/L change in LDL-P (Beta = 27, p = 0.04) over 1-year which was independent of baseline predictors and confounders (age, sex, smoking, statin use, waist size and physical activity; Beta = 30, p = 0.03).

Conclusion—A reduction in trans fat intake over 1-year was significantly associated with a reduction in LDL-P independent of potential confounders. Healthcare providers should reinforce the beneficial impact of a healthy diet, and in particular modifications in trans fat intake on improving lipid profiles.

Keywords

Prevention; Cardiovascular Disease; Nutrition

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Introduction

Over the past decade there has been increased recognition of the deleterious effect of dietary trans fat on cardiovascular disease (CVD) risk.(1) The magnitude of this association has been recently demonstrated by a meta-analysis that showed a 2% increase in dietary trans fat consumption was associated with a 23% increase in the incidence of CVD.(2) One mechanism through which increased trans fat consumption is thought to impart CVD risk is through adverse affects on traditional serum lipid measurements such as high density lipoprotein (HDL), low density lipoprotein (LDL), and total cholesterol.(3–5) In an analysis of 700 men who participated in the Normative Aging Study every unit increase in trans fat ($\log_e$ g/d) was associated with a 23% increase in LDL-C, a 4% decrease in HDL-C and a 34% increase in total cholesterol.(6) Despite these effects of trans fat on traditional serum lipid measurements, the relationship between trans fat intake and CVD is greater than that explained by the worsening of these measurements alone, suggesting that other CVD risk factors may be involved.(2, 7)

LDL particle number (LDL-P) is a novel modifiable risk factor and a marker of lipid particle burden.(8, 9) Studies have suggested that increased LDL-P leads to progression of CVD and that the predictive value of LDL-P for future CVD events are equal to or greater than more traditional lipid measurements such as LDL-C.(9–13) The relationship between dietary trans fat intake and LDL-P has not been established. The purpose of this study was to evaluate the association between dietary changes in trans fat consumption and change in LDL-P at 1-year in the context of a randomized controlled trial of lifestyle intervention among family members of patients hospitalized with CVD.

Methods

Study Population

The design was a 1-year observational follow-up evaluation of participants in The Family Intervention Trial for Heart Health (FIT Heart) which has been described previously.(14) Briefly, FIT Heart was a National Heart Lung and Blood Institute (NHLBI) sponsored randomized controlled clinical trial that enrolled 501 family members of patients who were admitted to the cardiovascular service of the New York Presbyterian/Columbia University Medical Center. Enrollment occurred between January of 2005 through June 2007 and follow up visits were conducted between January 2006 and June 2008. Participants were randomized to either a lifestyle special intervention group (SI) or to a control intervention (CI) that received general health messages. To avoid nonindependence of observations, only one family member per family was enrolled in the study and randomized. Participants in both groups followed up with their healthcare providers and initiated physician recommended therapies at their own discretion over the 1-year study period. The SI group received CVD risk factor screening results and education about diet and physical activity to prevent CVD, with regular contact and feedback by a health educator for up to 1-year. There were no significant differences in baseline clinical or biochemical characteristics between the SI and CI arms, including baseline trans fat intake and LDL-P levels. Lifestyle changes including diet were observed in both groups at 1-year (14). Both groups significantly lowered their trans fat intake during the study period; there was no significant between-group difference in this reduction.(14)

To be included in this follow-up analysis, participants had to have dietary and LDL-P data at baseline and 1-year (n = 400, 68% female, 36% racial/ethnic minority, mean age 48 ± 13 years). Reasons for exclusion were lack of dietary data at 1 year (n = 77), pregnancy (n = 5), or implausible dietary data (n = 19) defined as women < 500 kilocalories per day (kcal/day) or > 3500 kcal/day and men < 800 kcal/day or > 4000 kcal/day.(15) This study and the

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process for obtaining informed consent was approved by the Institutional Review Board of Columbia University Medical Center.

Diet Measures

Participant diet was assessed using the validated full length 1998 Gladys Block Food Frequency Questionnaire. (16, 17). Diet components, including trans fat, were expressed in percent of kilocalories per day (% of kcal/day) and were measured as the amount of kilocalories per macronutrient consumed in one day divided by the total number of kilocalories consumed in one day per participant. Change in trans fat from baseline to 1-year was expressed as the change in the percent of trans fat in kcal/day.

Laboratory Measures

Venous fasting plasma blood samples were obtained and analyzed at baseline and 1-year, stored at −70° C for up to 2 weeks and analyzed in the Columbia University Clinical and Translational Science Award Biomarker Laboratory (certified by the Centers for Disease Control and Prevention lipid quality control program). LDL-P, expressed in nanomole per liter (nmol/L) was measured by a commercially available proton NMR spectroscopic assay (LipoScience, Raleigh NC; in kind).(18) Interassay reproducibility of plasma LDL-P determined from replicate analysis was < 4%.(19) Plasma total cholesterol, HDL-C, and triglycerides, expressed in milligrams per deciliter (mg/dL), were determined spectrophotometrically on a Hitachi 912 chemical analyzer. Plasma LDL-C (mg/dL) values were assessed using a direct homogeneous enzymatic colorimetric assay when fasting triglycerides were ≤ 400 mg/dL. LDL-P was divided into quartiles based on the population risk of cardiovascular disease as defined in the Framingham Offspring Study and from the NMRLipoProfile Test™ as; Low LDL-P (< 1000 nmol/L), moderate LDL-P (1000–1299 nmol/L), borderline-high LDL-P (1300–1599 nmol/L) and high LDL-P (> 1600 nmol/L). (10, 18)

Covariate Measures

Traditional CVD risk factors and potential confounders such as demographics, medical and family history, medication usage and lifestyle were measured at baseline and 1-year using standardized questionnaires. Body Mass Index (BMI) was measured as body mass in kilograms divided by meters squared (kg/m^2). Waist Circumference was measured in inches with above goal waist circumference considered ≥ 35inches for females or ≥ 40 inches for males. Both BMI and waist circumference were measured using National Cholesterol Education Program Adult Treatment Panel III protocols.(20, 21) Physical activity and smoking were measured at baseline and at 1-year using standardized questions adapted from the validated Behavioral Risk Factor Surveillance System questionnaire.(22)

Statistical Analysis

Descriptive data were evaluated using percentages for categorical variables and means for continuous variables. Linear regression was used to evaluate the association between baseline dietary intake, cardiovascular risk factors, and baseline LDL-P. Differences in mean LDL-P stratified by categorical baseline characteristics were assessed using student t-tests of independent samples. Variables were graphed, variances computed and t-tests for unequal variances and spearman rank based correlations were used when samples were not normally distributed.

Regression analysis was used to evaluate the association between change in trans fat and change in LDL-P over 1-year adjusting for confounders and covariates. Covariates were included in the multivariate model when they were associated with LDL-P at baseline and

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when the change in the covariate over the study period was associated with change in LDL-P, or if they have been clinically shown to be associated with LDL-P in prior research (e.g. age, physical activity). Variables were excluded from the multivariate analysis if there was multicollinearity (e.g. BMI and waist size) or small sample size (< 15; e.g. change in smoking status). Changes in dietary components other than trans fat (saturated fat, mono and polyunsaturated fat, omega-3, carbohydrate and protein) that were not significantly associated with LDL-P at 1-year were not included in the final model.

A student t-test was used to compare change in trans fat intake in those that remained in the highest quartile of LDL-P after 1-year as compared to those who transitioned from the highest to a lower quartile. A paired t test was used to assess individual change in mean LDL-P from baseline to 1-year. All statistical analysis were performed using STATA 11 (StataCorp. College Station, TX). A 2-sided alpha error of < 0.05 indicated statistical significance.

Results

Baseline characteristics of the study population are listed in Table 1. The majority of the participants were less than 65 years old (87%), female (68%), white (64%), obese/overweight (62%), and physically inactive (physical activity ≤ 3 days/week; 77%). A small proportion of participants were on HMG-CoA reductase inhibitors (statins) (15%).

Baseline mean trans fat intake was 2.5% of kcal/day and all participants had trans fat in their diet. Nearly all participants had greater than 1% of kcal/day as trans fat intake and a quarter consumed greater than 3% per day. Two thirds of participants consumed greater than 35% of their kcal/day as total fat, and 56% of participants consumed greater than 10% of kcal/day as saturated fat. Mean baseline LDL-P was 1186 ± 373 nmol/L; 33% of participants were classified as low LDL-P, 34% moderate LDL-P, 18% borderline-high LDL-P and 14% had high LDL-P.

Baseline Analysis of Participant Characteristics, Dietary Composition, CVD Risk Factors and LDL-P

Table 2 shows the linear association between baseline characteristics, dietary intake, CVD risk factors and LDL-P. There was a significant positive association between baseline dietary trans fat intake and baseline LDL-P (Beta = 37, p = 0.04). Those who consumed diets with higher compositions of monounsaturated fats (Beta = −13, p = 0.02) and protein (Beta = −16, p = 0.01) had lower baseline LDL-P levels.

Table 3 presents mean baseline LDL-P stratified by participant characteristics. Participants who were male, overweight/obese and active smokers all had higher baseline LDL-P. Baseline statin use was associated with a significantly lower LDL-P.

Change in Trans Fat and LDL-P over 1-Year

In univariate analysis, every 1% change in trans fat intake (% of kcal/day) was associated with a 27 nmol/L change in LDL-P (Beta=27, p = 0.04) over 1-year. Multivariate linear regression analysis of the association between change in trans fat and change in LDL-P over 1-year is shown in Table 4. In the multivariate model the association between change in trans fat and change in LDL-P over 1-year remained significant after adjustment for potential confounders including new statin use (n=15), waist size, and physical activity over 1-year (Table 4). The beta estimate for the association was not altered when baseline monounsaturated fat, protein, BMI and change in BMI were added into the model and therefore the covariates were not included in the final model.

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Sub-Group Analysis of Change in Trans Fat and LDL-P over 1-Year

Among participants who were initially classified as having a high LDL-P (n = 54, 7%), 27% (n = 15) transitioned from the highest LDL-P quartile to the borderline high (n = 10) or moderate (n = 5) quartile at the end of 1-year. Mean trans fat intake of these individuals in % of kcal/day decreased from 2.5% to 1.8% (p = < 0.001). When compared to participants who remained in the highest LDL-P quartile, those who transitioned to a lower LDL-P quartile at the end of 1-year had significantly lower trans fat intake (Table 5).

Among participants who increased their trans fat intake over 1 year (n = 174, 43%) mean LDL-P increased from 1116 nmol/L to 1162 nmol/L (p = 0.01). Those who increased their transfat intake less than 1% had a mean LDL-P increase of 36 nmol/L (n = 129, p = 0.08). Those who increased their trans fat intake 1% or more showed a mean LDL-P increase of 82 nmol/L (p=0.04).

Discussion

These data document that a decrease in dietary trans fat intake over 1-year was associated with a significant decrease in LDL-P, independent of confounders and other changes in lifestyle. For every 1% change in trans fat intake (%kcal/day) there was a 27 nmol/L change in LDL-P. When participants were stratified by LDL-P risk quartiles, transitioning to a lower quartile was associated with a greater reduction in trans fat as opposed to those who remained in the same quartile throughout the course of the 1-year study.

To our knowledge this is one of the first studies that has examined the affects of trans fat on LDL-P. Our findings are similar to studies that have evaluated surrogate markers of LDL-P such as apolipoprotein B (apoB). ApoB is found primarily in LDL particles and is considered a marker of lipid particle burden.(9) In a meta-analysis of eight clinical trials that evaluated the association between change in dietary trans fat intake and change in lipids and lipoprotiens (LDL-C, HDL-C, triglycerides, total cholesterol, apoA, apoB), change in trans fat correlated the highest with change in apoB.(23) Our study corroborates this result by demonstrating that LDL-P is associated with dietary trans fat intake.

Other studies have examined the relationship between total fat and LDL-P and yielded similar findings in the setting of secondary prevention.(24–26) In a study of 146 patients with CVD, a low fat diet (< 10% of kcal/day from fat) was associated with an 8% reduction in LDL-P (p = 0.04, 1437 nmol/L to 1317 nmol/L) over 1-year.(24) In a prospective cohort study of 131 participants on a dietary intervention to reduce total fat over 3 months, LDL-P was reduced from 1145 nmol/L to 1128 nmol/L (p = 0.001).(26) Our findings suggest that it may be reduction in trans fat that is contributing to the reduction in LDL-P.

The association between increased LDL-P and CVD risk has been shown; subjects with higher LDL-P can have upwards of two to three times the risk of CVD independent of LDL-C.(10, 11, 13, 27) In our study, subjects who reduced their trans fat by almost 1% over 1-year also moved to a lower LDL-P quartile. This suggests that reduced LDL-P may be one mechanism through which reduced trans fat confers reduced CVD risk, but this must be replicated in future studies.

Strengths of this study include the participation of diverse free living individuals followed over 1-year. There were also limitations to consider. Mean baseline trans fat intake was 2.5% in the study population, which may have limited the potential for reduction in dietary intake over one year. Statistical power to detect an association between trans fat and LDL-P was limited, therefore the possibility that the observed associations were due to chance cannot be excluded. Food frequency questionnaire data may be subject to interviewer or

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response biases. However, food frequency data was validated against food records in this study population, and interviewers were trained using standardized methods and overseen for quality assurance supporting that any error in the measure of trans fat intake would be non-differential by participant characteristic.(14) There was a trans fat ban initiated in New York City during the last year of the study.(28) However, this would be unlikely to affect the observed association between trans fat intake and LDL-P.

In conclusion, a dietary decrease in trans fat over 1-year was associated with a significant decrease in LDL-P independent of confounders and covariates. Healthcare providers should reinforce the beneficial impact of a healthy diet, and in particular modifications in trans fat intake on improving lipid profiles.

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Table 1

Baseline Characteristics (N = 400)

Characteristic	N (%)
Age ≥ 65	51 (13)
Female Sex	270 (68)
Race or Ethnic Group	
White	255 (64)
Black	24 (6)
Hispanic	100 (25)
Overweight/Obese	247 (62)
Above Goal Waist Circumference[a]	163 (41)
Statin[b] Use	61 (15)
Smoker	40 (10)
Physical Activity ≤ 3 days/week	309 (77)
Dietary Intake (% of kcal/day)[c]	Mean ± SD
Trans Fat	2.5 ± 1
Total Fat	37.8 ± 7
Saturated Fat	10.6 ± 3
Monounsaturated Fat	15.0 ± 3
Polyunsaturated Fat	9.2 ± 3
Omega-3 Fatty Acids	0.8 ± 0.3
Carbohydrate	45.6 ± 8
Protein	16.2 ± 3
Alcohol	3.5 ± 4.8
Serum Lipid Measures	Mean ± SD
LDL Particle Number (nmol/L)	1185 ± 37
Total Cholesterol (mg/dl)	202.6 ± 38
Direct LDL (mg/dl)	128.1 ± 35
Triglycerides (mg/dl)	114.2 ± 64
HDL (mg/dl)	
Men	48.2 ± 13
Women	65.0 ± 19

[a] Men ≥ 40 inches or women ≥ 35 inches;

[b] HMG-CoA reductase inhibitor (statin);

[c] Dietary intake in percent of kilocalories (% of kcal) per day was measured as the amount of kilocalories per macronutrient consumed in one day divided by the total number of kilocalories consumed in one day per participant.

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Table 2

Linear Association between Baseline Participant Characteristics, Dietary Composition, Cardiovascular Risk Factors and LDL Particle Number

	Baseline LDL Particle Number (nmol/L)		
Baseline Variables	**Beta**	**SE**	**P-Value**
Age (y)	0.98	1.39	0.48
Female Sex	−139.08	39.34	< 0.001
BMI (Kg/m^2)	18.02	3.03	< 0.001
Waist Circumference (inches)	18.6	3.2	< 0.001
Statin[a] Use	−100.05	55.42	0.07
Smoker	161.76	61.88	0.01
Physical Activity (days/week)	−3.64	9.6	0.71
Trans Fat (% of kcal/day)	36.55	17.46	0.04
Total Fat (% of kcal/day)	−4.64	2.73	0.09
Saturated Fat (% of kcal/day)	2.31	7.32	0.75
Monounsaturated Fat (% of kcal/day)	−12.64	5.4	0.02
Polyunsaturated Fat (% of kcal/day)	−9.05	6.7	0.18
Omega-3 Fatty Acids (% of kcal/day)	−50.61	59.65	0.4
Carbohydrate (% of kcal/day)	4.14	2.2	0.06
Protein (% of kcal/day)	−15.95	5.68	0.01
Alcohol (% of kcal/day)	2.68	3.9	0.5

[a] HMG-CoA Reductase inhibitor (statin)

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Table 3

Baseline Mean LDL Particle Number Stratified by Baseline Characteristics

Characteristic (N)	Mean ± SD (nmol/L)	P-Value
Age		
Age ≥ 65 (y) (N = 51)	1146 ± 36	
Age < 65 (y) (N = 349)	1191 ± 21	0.41
Sex		
Male (N = 130)	1280 ± 36	
Female (N = 270)	1140 ± 21	0.001
BMI		
BMI ≥ 25 kg/m^2 (N = 247)	1275 ± 24	
BMI < 25 kg/m^2 (N = 153)	1040 ± 27	< 0.001
Waist Circumference		
Above Goal[a] (N = 163)	1277 ± 28	
At Goal (N = 237)	1122 ± 24	< 0.001
Statin[b] Use		
Yes (N = 61)	1091 ± 45	
No (N = 338)	1202 ± 20	0.03
Smoker		
Yes (N = 40)	1332 ± 67	
No (N = 360)	1169 ± 19	0.02
Physical Activity		
≤ 3 days/week (N = 309)	1196 ± 21	
> 3 days/week (N = 90)	1152 ± 42	0.31

[a] Men ≥ 40 inches or women ≥ 35 inches;

[b] HMG-CoA reductase inhibitor (statin).

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Table 4

Multivariable Linear Regression Analysis: The Association between Change in Trans Fat and Change in LDL Particle Number Over One Year

	Change in LDL Particle Number (nmol/L) Over 1-Year		
	Beta	**SE**	**P-Value**
Change in Trans Fat (% of kcal/day)	26.91	12.41	0.03
Age (y)	0.92	1.07	0.39
Female Sex	−23.36	28.85	0.42
Baseline Statin Use	47.94	38.65	0.22
Started Statin During Trial[a]	−573.96	66.77	< 0.001
Baseline Smoker	−10.7	44.29	0.81
Above Goal Waist Circumference[b]	8.99	27.42	0.74
Decrease in Waist Circumference[c]	−17.75	29.57	0.55
Low Baseline Physical Activity Level[d]	78.15	31.72	0.01
Increase in Physical Activity[e]	−3.42	27.53	0.9

[a] Participants who were started on HMG-CoA reductase inhibitors (statin) during the 1-year study period;

[b] Men ≥ 40 inches or women ≥ 35 inches;

[c] Participants who decreased their waist circumference over the study period;

[d] ≤ 3 days/week;

[e] Participants who increased their level of physical activity over the study period; 10 participants were excluded due to missing LDL-P data at 1-year.

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Table 5

Association between Change in Trans Fat Intake and Change in LDL Particle Number Quartile Over 1-Year

Change in LDL-P Quartile[b]	Baseline Trans Fat Intake[a] (Mean ± SD)	Final Trans Fat Intake (Mean ± SD)	Change in Trans Fat (Mean ± SD)
Reduction from Highest to Other LDL-P Quartile			
(yes vs. no, N = 15)	2.52 ± 0.97	1.83 ± 0.94	−0.69 ± 0.82
Remains in Highest Quartile			
(yes vs. no, N = 39)	2.78 ± 1.12	2.45 ± 1.05	−0.33 ± 0.63
Students T test (P – Value)	P = 0.40	P = 0.045	P = 0.14

[a] Trans fat in % kcal/day;

[b] LDL-P quartiles: Low LDL-P (< 1000 nmol/L), moderate LDL-P (1000–1299 nmol/L), borderline-high LDL-P (1300–1599 nmol/L), high LDL-P (> 1600 nmol/L).