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Association between Carbohydrate Intake and Serum Lipids

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Abstract

Background—The effect of dietary carbohydrate on blood lipids has received considerable attention in light of the current trend in lowering carbohydrate intake for weight loss.

Objectives—To evaluate the association between carbohydrate intake and serum lipids.

Methods—Blood samples and 24-hour dietary and physical activity recall interviews were obtained from each subject at quarterly intervals for five consecutive quarters between 1994 and 1998 from 574 healthy adults in Central Massachusetts. Relationships between serum lipids and dietary carbohydrate factors were assessed using linear mixed models and adjusting for other risk factors known to be related to blood lipids. Both cross-sectional and longitudinal results were reported.

Results—Cross-sectional analysis results from this study suggest that higher total carbohydrate intake, percentage of calories from carbohydrate, glycemic index (GI) and/or glycemic load (GL) are related to lower high-density lipoprotein cholesterol (HDL-C) and higher serum triacylglycerol levels, while higher total carbohydrate intake and/or GL are related to lower total and low-density lipoprotein cholesterol (LDL-C) levels. In a one-year longitudinal analysis, GL was positively associated with total and LDL-C levels, and there was an inverse association between percentage of calories from carbohydrate and HDL-C levels.

Conclusions—Results suggest that there is a complex and predominantly unfavorable effect of increased intake of highly processed carbohydrate on lipid profile, which may have implications for metabolic syndrome, diabetes, and coronary heart disease. Further studies in the form of randomized controlled trials are required to investigate these associations and determine the implications for lipid management.

Keywords

dietary carbohydrates; LDL cholesterol; glycemic index; longitudinal studies

INTRODUCTION

Hyperlipidemia is established as a major risk factor for coronary heart disease (CHD) [1,2]. Dietary factors play an important role in the development of CHD through their effects on body

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weight and serum lipid levels. Because it is widely accepted that high saturated fat and cholesterol intakes increase CHD risk [3-5], investigation of dietary factors in relation to CHD has concentrated mainly on fat and cholesterol. The role of dietary carbohydrate (CHO) in determining both body weight and lipid levels has received considerably less attention. However, recently several studies have raised the issue of the relationship between carbohydrate related factors [glycemic index (GI) and glycemic load (GL)] and CHD risk factors [6]. GI is a ranking of foods containing carbohydrates according to their glycemic effect, in comparison to either white bread or glucose. In general, more refined or processed carbohydrates have a higher GI. The GL is calculated by multiplying the GI of a food by the amount of carbohydrate in one serving of the food in grams. Results from the prospective Nurses' Health Study suggest that dietary glycemic load (GL) is directly and independently associated with risk of CHD after controlling for other known CHD risk factors [6]. In addition, high intake of refined carbohydrate was found to increase the risk of stroke in this cohort [7]. These results have fueled the debate over the role of dietary carbohydrate in the development of CHD and point to the need to refine the American Heart Association's recommendation of a low-fat diet: replacing fat with carbohydrate for energy supply [8,9]. Ludwig summarized the results of thirteen experimental studies that examined the effects of dietary GI on serum lipids under macronutrient-controlled conditions [10]. Ten studies reported lower triacylglycerol, seven studies reported lower low-density lipoprotein cholesterol (LDL-C), and 10 studies reported lower ratio of total to high-density lipoprotein cholesterol (HDL-C). Six epidemiologic analyses published on the subject also demonstrated higher HDL-C levels, lower triacylglycerol levels, or lower myocardial infarction rates among individuals in the lowest category of the GI or GL distribution, as compared with those in the highest category, after adjustment for potentially confounding factors [6,11-14]. However, the long-term effect of dietary carbohydrate factors on blood lipids remains largely unknown. Further analyses from available data sets may provide insights into the relationship between dietary carbohydrate (CHO) and other known CHD risk factors.

The Seasonal Variation in Blood Cholesterol Levels (SEASONS) study [15,16], an observational study designed to describe and prospectively delineate the nature and causes of seasonal variation in blood lipids, provides a unique opportunity to examine the consequences of naturally occurring seasonal changes in CHO intake on blood lipids. We have used longitudinal data from this study to evaluate the relationship between blood lipids and carbohydrate-related dietary factors (GI, GL, daily CHO intake, and percent of calories from CHO), while controlling for the effects of physical activity and other factors known to be related to blood lipids.

MATERIALS AND METHODS

Subjects

SEASONS study subjects were 641 healthy adults, aged 20 to 70 years, enrolled at the Fallon Healthcare System, a health maintenance organization (HMO) in central Massachusetts. Eligible subjects were: 1) not taking cholesterol-lowering medications; 2) not following a lipid-lowering or weight-control diet; 3) not working a night shift; 4) free from possible causes of secondary hypercholesterolemia (e.g., hypothyroidism, pregnancy); and 5) free of any chronic life-threatening illness (e.g., cancer, or renal or heart failure). Potential eligible individuals were called by the Fallon Healthcare System recruiters and were invited to participate in the study. Details of the study recruitment have been described elsewhere [15,16]. Subjects were recruited between December 1994 and February 1997. Subjects were followed prospectively for one year, during which they were assessed quarterly for serum lipids, diet, physical activity, anthropometric measures, light exposure, and psychosocial factors. The Institutional Review

Boards of the Fallon Healthcare System and the University of Massachusetts Medical School approved all subject recruitment and data collection procedures.

Of the 641 participants in the SEASONS study, 110 (17.2%) dropped from the study during the one-year follow-up. Most of the reasons for dropout are due to inability to schedule appointment (39, 35%) and loss of follow-up (33, 30%). Five hundred and seventy-four subjects (90%) had at least two visits yielding both blood lipid and CHO intake values, and were included in the analyses. Over 78% of the 574 subjects had paired blood lipid and diet data for 4 measures for the analysis, and 61% of subjects had 5 paired measures.

About half (51.7%) of subjects were men. Participants were middle-aged (average 47.8 years old), primarily white (87%), well-educated (69% with some college or more), employed (83%), non-smoking (83%), and generally overweight (63%). Details of participants' characteristics are presented in Table 1. Participants had relatively stable body weight during the study (mean weight change = −0.06 lbs, SD = 8.8).

Assessment of Demographic Characteristics

Data on demographic variables and smoking status were collected by a self-administered questionnaire at the baseline clinic visit.

Blood Sample Collection and Lipid Assays

During each visit, a 12-hour fasting blood sample was collected between 7 and 10 AM. Serum was isolated, packed in dry ice and shipped via University courier to a Centers for Disease Control Standardized Laboratory, on a weekly basis. Assays for total cholesterol, HDL-C and triacylglycerols were done in this lab. LDL-C was calculated by the Friedewald formula, i.e. LDL-C = total cholesterol − [triacylglycerol (TG)/5 + HDL-C] where all concentrations are given in mg/dl [17]. When TG exceeded 400 mg/dl, the LDL-C was not calculated.

Assessment of Diet

Serial 24-hour dietary recalls (24HR), three in each quarter of follow-up, were performed on randomly selected days to quantify dietary changes over the week (two week-days and one weekend day). The 24HR data were collected using the Nutrition Data System (NDS) data entry and nutrient database software developed and maintained by the Nutrition Coordinating Center at the University of Minnesota, Minneapolis, MN [18]. Dietary variables considered in these analyses were total energy intake (kcal/day), percent of calories from carbohydrates, alcohol, protein, saturated fat, monounsaturated fat, polyunsaturated fat (expressed as a percentage of total energy intake), as well as total grams of carbohydrate intake.

GI and GL Calculation

GI and GL were computed from the 24HR. Food items devoid of carbohydrate were excluded when the GI and GL values were calculated. From an initial 1,482 foods, 1,261 carbohydrate-containing food items remained for these analyses. Dishes containing more than one food item were broken down into individual food components and matched to the International Table of Glycemic Index [19] using NDS (version 5.0_35). Using these food components, we calculated each food's GL by multiplying the carbohydrate content (computed from NDS) by the food's GI value [6,20,21]. For example, if a subject consumed a serving (1 slice) of white bread: carbohydrate amount (grams) = 12 g; GI for white bread = 1 (100%); therefore, the GL of this serving would be 12 × 1 = 12. Each unit of dietary GL represents the equivalent of 1 g carbohydrate from white bread. In order to calculate the daily GL, we added all the individual GL for each food item consumed in a 24-hour period. The dietary GL essentially matches carbohydrate content gram-for-gram and thus reflects the overall quality and quantity of

carbohydrate intake in an entire diet. On the other hand, the overall daily GI, a variable ranking the quality of daily carbohydrate intake, was calculated by dividing the daily GL by the total amount of carbohydrate consumed per day. The formulas for the calculation of daily GL and GI are the following:

$$\text{Overall Dietary GL} = \sum_{i=1}^{n} \text{GI}_i \times \text{CHO}_i$$

$$\text{Overall Dietary GI} = \sum_{i=1}^{n} \text{GI}_i \times \text{CHO}_i \; / \; \sum_{i=1}^{n} \text{CHO}_i$$

where GI_i is the GI for food "i", CHOi is the carbohydrate content in food "i" (g per day), and "n" is the number of foods eaten per day. The GI for each food was established by a matching and ranking process utilizing the food items listed in the International Tables of GI [19] and in the updated online database maintained by the University of Sydney [http://www.glycemicindex.com/]. The NDS program provided the carbohydrate content of foods necessary for GL calculation. The GI value for the food was multiplied by 1.43 to convert from glucose to white bread as the referent GI value [19]. We excluded foods with very low carbohydrate content since their GI values cannot be tested. The cutoff point was set at 3.5 grams of carbohydrate per serving [22]. For this reason, we also ignored beer, wine, and spirits in the calculation of GI and GL [23]. Of the total 1261 food items reported in the 24HR, 424 foods were excluded. These 424 foods contributed only 5.4% (12.5 grams) to the total daily average carbohydrate consumption of 232 grams. The methodology we used for dealing with GI calculation has been widely cited [20,22], it also has been used in our previous publication [24].

The average GI and GL values in our study are 84 and 198, which is comparable to those reported in several studies using a food frequency questionnaire (FFQ) as a dietary assessment tool [6,12-14,25]. For example, the GI was 75 (SD = 5.0) and GL was 166 (SD = 32) in the Women's Health Study [26].

Physical Activity

Data on physical activity, which were used as control variables in analyses, were collected using the same telephone-administered 24HR session employed to assess diet [27,28]. Subjects were asked to recall the amount of time they spent in light, moderate, vigorous, and very vigorous activities in household, occupational, and leisure-time activity domains. Estimates of physical activity energy expenditure in metabolic equivalent task hours (MET-hour) were calculated according to methods developed by Ainsworth and colleagues [29]. The 24HR was validated against both accelerometers and standard questionnaires [27]. Results were comparable to published data from other short-term activity assessments employing the Baecke Questionnaire and activity monitors as criterion measures [27].

Statistical analyses

Descriptive analyses were conducted to evaluate the range and distribution of all demographic and clinical outcome variables of interest (e.g., serum lipids, CHO, GI, GL, etc.). All clinical outcomes under analysis except triacylglycerols were approximately normally distributed. Triacylglycerols were highly skewed to the right and log-transformed in further analysis.

For the final analysis, data from all five time-points were fitted into a linear mixed model, with a random intercept for each subject, to examine the relationship between carbohydrate-related factors and serum lipids. Total cholesterol, LDL-C and HDL-C, as well as log-transformed triacylglycerols, were the dependent variables, and each was analyzed separately. Relevant clinical predictors included carbohydrate factors, including daily GI, GL, grams of CHO, and

percentage of calories from carbohydrate. Since analytic approaches were similar for all the lipid outcomes, the statistical modeling is illustrated using LDL-C.

The associations between lipids and carbohydrate related factors, as determined using scatter plots and fractional polynomial regression, were approximately linear. The associations between lipid outcomes and predictors were assessed using linear mixed models [30-32] while accounting for within-subject correlations. To develop the "best" model, we included socio-demographic and physiological variables that are known to be associated with LDL-C (such as those listed in Table 1, plus physical activity and dietary variables). We forced gender, leisure-time physical activity, BMI, and age into the model, and then used a backward stepwise process to eliminate non-significant ($p > 0.10$) covariates. As the serial autocorrelation was estimated in the neighborhood of 0.80, we specified the serial correlation between observations of the same subject as autoregressive of order one. Seasonal variation in blood lipids [16] was accounted for by fitting season of the year, categorized using the light-season definition, centered at the equinoxes (Winter: November 6—February 4; Spring: February 5—May 6; Summer: May 7—August 5 and Fall August 6—November 5). This maximized variation in light exposure. We distinguished cross-sectional (between-subject) effects from longitudinal effects (temporal change within subject) of predictors [33,34]. For example, concurrent GI was separated into two components: 1) the subject-specific average and 2) within-subject quarterly deviations from this average. Both the cross-sectional and longitudinal effects were included in the model. This method was used in our previous analyses in examining the association between dietary carbohydrate and body weight [24] and accounts for missing data points.

Because GI, total CHO intake, percent of calories from CHO, and GL are related to each other, similar models were conducted for each variable separately to avoid collinearity. In order to incorporate the effect of carbohydrate on total and HDL-C into a composite lipid profile, further analyses were conducted between TC/HDL-C ratio and carbohydrate.

We also conducted similar analyses stratified by gender.

RESULTS

Participants' average blood lipids, dietary carbohydrate factors and physical activity from all time points are presented in Table 2. Subjects had moderately elevated serum lipids and ate a fairly typical American diet. There was no significant age difference between males and females. Males had higher LDL-C, triacylglycerols, caloric intake, glycemic load, and total and occupational physical activity than females, while females had higher values of HDL-C, percentage of calories from carbohydrates, glycemic index, and household physical activity.

Table 3 presents the association between carbohydrate factors and blood lipids. Results of analyses using both cross-sectional and longitudinal data, and unadjusted and adjusted for covariates, are shown in the table. In general, analyses of unadjusted cross-sectional data produced results consistent with effects of carbohydrate-related factors on study outcomes. Cross-sectionally, primarily inverse associations (i.e., as one value increases, the other decreases) were observed in the unadjusted analyses between carbohydrate-related parameters and cholesterol-related outcomes. However, positive associations (i.e., as one value increases, the other also increases) were observed with triacylglycerols and the TC:HDL-C ratio. With adjustment, most results persisted; however, the effect of % of calories from carbohydrate on both triacylglycerols and the TC:HDL-C ratio became significant in the adjusted analyses. Longitudinally, unadjusted analyses produced strikingly different, and many fewer, significant results. When covariates were included in the model, only four results were significant at the nominal $\alpha = 0.05$. These included the associations of GL with TC and LDL-C, and % of calories from carbohydrate with HDL-C and TC:HDL-C ratio.

We also fit models that included CHO (percentage of calories from carbohydrate or daily grams of carbohydrate) and GI in the model simultaneously. Adjustment for the other variable did not substantially change the relationships between CHO or GI and blood lipids (data not shown), suggesting that the effects of CHO and GI are independent from each other. The associations of blood lipids and carbohydrate related factors, stratified by gender, were similar to results obtained with both genders together (data not shown).

DISCUSSION

Findings from the cross-sectional analyses are consistent with results from several studies suggesting that higher total carbohydrate intake, percentage of calories from carbohydrate, GI and/or GL are related to lower HDL-C and higher serum triacylglycerols levels [12,13,35]. An interesting finding from our study revealed that higher total carbohydrate intake, GL, or both are also related to lower total and LDL-C levels. To our knowledge, these associations have not been reported in previous studies [12,13,35]. Although the inverse relationship between dietary carbohydrate and total cholesterol and LDL-C may appear to be beneficial, the overall impact on the lipid profile is still unfavorable because of a proportionally greater decrease in HDL-C, resulting in a relative increase in the TC:HDL-C ratio. On the other hand, this analysis confirmed a clearly unfavorable relationship between higher carbohydrate intake and elevated triacylglycerol levels [14]. Both decreased HDL-C levels and increased triacylglycerol levels are related also to the development of metabolic syndrome and diabetes. Longitudinal analysis showed a significant increase in total cholesterol and LDL-C related to increasing GL, and a significant decrease in HDL-C and increase in total cholesterol/ HDL-C ratio related to percentage of calories from carbohydrates. Nonetheless, these observations also suggest that dietary CHOs have a complex relationship with serum lipids, which need to be further elucidated.

Increased carbohydrate consumption and intake of food with a high GI produces higher postprandial glucose and insulin concentrations [36]. Ultimately, this may decrease insulin sensitivity [37], raising fasting triacylglycerol concentrations and reducing HDL-C levels, a profile that increases the likelihood of CHD [38,39]. Cross-sectional associations of carbohydrate factors with HDL-C and triacylglycerol concentrations are supported by findings reported in previous studies. Four of five epidemiological cross-sectional analyses published on the subject, including the Nurses' Health Study and examination of NHANES III data, demonstrated higher HDL-C levels and lower triacylglycerol levels among individuals in the lowest category of GI or GL as compared with those in the highest category, after adjustment for potentially confounding factors [11-14]. Two of these studies compared GI and GL directly with respect to serum lipid concentrations. One found that GL had a greater effect [14]; while in the other, both GI and GL had similar effects [12]. The fifth observational study found no significant association between GI and metabolic risk factors for heart disease [25]. Cochrane system review of 15 RCTs for low GI diet intervention also found little association between GI and serum lipids [40].

In contrast to the cross-sectional analyses, there are many fewer associations observed in the longitudinal analyses: only a significant increase in total cholesterol and LDL-C related to increasing GL, and a significant decrease in HDL-C and increase in total and HDL-C ratio related to percentage of calories from carbohydrate. It is well known that even well-established predictors of serum lipids require relatively large changes in order to effect group-level change. This may also be true of carbohydrate factors, though this field is far from producing predictive equations of the type developed for dietary fat [41,42].

Unlike intensive intervention trials, there were only small changes in either CHO intake or blood lipid levels over the one-year observational period [i.e., the change in LDL-C was—0.91

mg/dl (SD = 22.8), grams of CHO was 0.54 (SD = 80.4), and GI was 0.42 (SD = 8.0)]. Therefore, the associations between CHO and blood lipids from this study were mainly encountered in the cross-sectional (between-subject), and not in the longitudinal (within-subject) analyses. This is essentially the converse of what has been reported in large-scale intervention trials regarding the temporal relationship between total cholesterol and dietary fat intake, where relatively predictable changes in serum cholesterol values are observed with changes in dietary fat intake, but there is no observable difference cross-sectionally [43].

We found that GL was associated with a decrease of total cholesterol and LDL-C cross-sectionally, and an increase of total cholesterol and LDL-C in the longitudinal analyses. Acute increases in carbohydrate consumption will increase blood insulin concentrations, thereby perhaps increasing total cholesterol and LDL-C production [38,39]. However, chronic increases in GL intake (e.g., evident cross-sectionally) may decrease insulin sensitivity, thereby decreasing LDL-C production [44,45].

HDL-C and LDL-C are the major focus in the US National Cholesterol Education Program guidelines [46,47]. Our results showed an inverse relation between HDL-C and carbohydrate predictors. According to the Framingham Study, for each 1 mg/dl decrease in HDL-C CHD risk increases by ~3% in women and ~2% in men [48]. Based on results from this study, a ten-unit increase in GI is associated with a decrease of 1.6 mg/dl in HDL, which in turn would translate into a 5.1% increase in CHD risk. Also, LDL-C and total cholesterol are important determinants of CHD. In general, a 10 mg/dl change in LDL-C is associated with a 5 to 7 percent change in CHD risk over 14 years, independent of the other major coronary risk factors [5]. Our data indicated inverse relations of LDL-C and total cholesterol concentration with grams of carbohydrates and GL, therefore, grams of carbohydrate and GL may be associated with CHD, supporting results from Liu and colleagues [6].

Claims regarding the effect of a low-carbohydrate diet or lowering GI or GL on blood lipids are premature. Some randomized controlled trials (RCTs) using a low-carbohydrate or a low GI diet have been done recently; however, interventions have been limited due to small sample size and short duration. For example, Sloth and colleagues found reduced LDL-C after 10 weeks of low GI diet in 23 healthy overweight women [49]. While low carbohydrate intakes have been associated with lowering serum triacylglycerol levels, the effect on HDL-C and LDL-C levels is still controversial [50-52]. The long-term impact of dietary CHO intake on CHD is even less clear; a recent meta-analysis of 15 RCTs for low GI diet indicated that no study reported an effect of low GI diet on CHD mortality or morbidity [40]. Long-term intervention studies in diverse populations are needed to clarify the relationship between various carbohydrate factors, other aspects of diet, and physical activity in relation to lipid profiles and CHD.

There are several strengths to our investigation. Previously, GI and GL had been computed from food frequency questionnaires (FFQs) querying either the past month or previous year [12-14]. The 24HR utilized in the present study provide more accurate data for computing GI and GL [53]. Because they allow much greater specificity, the 24HR provide a better measure of factors related to the GI, including a great variety (i.e., tens of thousands) of foods, portion sizes, cooking, and other preparation methods. By contrast, FFQs must estimate nutrient intakes from a composite listing of only about a hundred foods, rely on a small number of comparisons (usually 3) with standard portion sizes, and they are generally self-administered (and thus do not allow for careful probing). By utilizing data pooled from three 24HR, the nutrient intake is a more stable point estimate approximating true intake than one 24HR. The current study also collected information on many possible confounding factors, including BMI, smoking, alcohol intake, and physical activity, which were controlled for in the analysis. Finally, two to

five time point measures of dietary factors and lipid variables provided more reliable values for the analyses than a single measurement.

On the other hand, our study also has several potential limitations. Though the 24HR is the self-report method associated with the lower overall error compared with FFQ [54-57], information on diet and physical activity was still obtained from self-report and this measurement is subject to information bias. Also, there is a potential limitation in generalizing our study results. Participants in this study included highly motivated men and women between the ages of 20 and 70 years, who were predominantly well-educated, employed full time, and white. In addition to issues around selection bias, the physiological mechanism underlying the relationship between carbohydrate intake and serum lipids may vary across race and ethnicity, which is suggested by the greater prevalence of metabolic syndrome and diabetes among African-American and Hispanic populations [58,59]. Caution should be taken when generalizing to populations that have different baseline characteristics and/or dietary patterns with average carbohydrate intake values different from the range found in this study. The most consistent findings from this study derive from cross-sectional associations. As such, they are less likely to be considered “causal”.

CONCLUSION

From cross-sectional data, we found that percentage of calories from carbohydrates, as well as total intake of carbohydrates and glycemic load, were inversely associated with total cholesterol, LDL-C and HDL-C, moderately increasing the overall TC:HDL-C ratio. GI was found to be inversely associated only with HDL-C levels. Percentage of calories from carbohydrate is related to higher serum triacylglycerol levels. From the longitudinal analysis, GL was positively associated with total cholesterol and LDL levels, and there was an inverse association between percentage of calories from carbohydrate and HDL-C levels. Elevated triacylglycerols and decreased HDL-C serum levels are both known risk factors for developing CHD, as well as metabolic syndrome and diabetes. Further studies in the form of clinical trials are required to investigate these associations and to disentangle the relationship between dietary carbohydrate intake and serum lipids and its implications for cardiovascular disease and diabetes prevention.

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Table 1

Characteristics of Study Participants (N = 574), Seasonal Variation of Blood Cholesterol Study, Worcester, Massachusetts, 1994–1998

Variable	Frequency	%
Categorical variable		
Gender		
Male	297	51.7
Female	277	48.3
Ethnicity/Race		
White	470	86.9
Hispanic	39	7.2
Other	32	5.6
Education		
Less than high school	32	5.8
HS or vocational degree	138	25.0
Some college or Associates degree	170	30.8
College/graduate	211	38.3
Employment		
Full-time	371	67.2
Part-time	85	15.4
Unemployed/retired	96	17.4
Current smoking		
Yes	92	17.0
No	450	83.0
BMI classification		
Normal (18.5–24.9)	206	37.4
Overweight (25–29.9)	210	38.1
Obese (≥ 30)	135	24.5
	Mean	Standard Deviation
Continuous variable		
Age (years)	47.8	12.2
BMI (kg/m^2)	27.4	5.4

Due to missing values the total number of subjects differs.

Table 2

Average Blood Lipids, Dietary Carbohydrate Factors and Physical Activity, Seasonal Variation of Blood Cholesterol Study, Worcester, Massachusetts, 1994–1998 (N = 574)

Variables	Overall (n = 574) Mean (SD)	Males (n = 297) Mean (SD)	Females (n = 277) Mean (SD)
Blood lipids			
Total cholesterol (mg/dl)	218 (40)	221 (39)	216 (42)
LDL-C (mg/dl)*	143 (35)	146 (34)	143 (35)
HDL-C (mg/dl)*	47 (12)	43 (10)	52 (12)
Triacylglycerols (mg/dl)†	143 (119)	167 (146)	118 (72)
Log (Triacylglycerols)*	4.7 (0.58)	4.9 (0.60)	4.6 (0.52)
TC/HDL-C*	4.9 (1.6)	5.5 (1.6)	4.4 (1.3)
Dietary factors			
Energy intake (kcal per day)*	1966 (569)	2268 (559)	1643 (367)
% of calories from carbohydrate*	51 (7.5)	49 (7.7)	53 (6.8)
Total Carbohydrate (grams per day)	247 (74)	277 (78)	216 (53)
Glycemic index*	84 (5.1)	84 (5.5)	85 (4.8)
Glycemic load*	198 (63)	222 (68)	172 (46)
Physical activity			
Total MET-h/d*	30 (4.6)	32 (5.4)	29 (2.9)
Leisure MET-h/d	1.9 (2.0)	2.1 (2.2)	1.7 (1.8)
Occupational MET-h/d*	4.6 (5.7)	6.0 (6.8)	3.1 (3.7)
Household MET-h/d*	4.7 (3.2)	4.4 (3.5)	5 (2.9)

Abbreviations used in the table: SD: standard deviation, LDL-C: low-density lipoprotein cholesterol, HDL-C: high-density lipoprotein cholesterol, TC: total cholesterol, and MET-h/d metabolic equivalent task hours per day.

* $p < 0.05$ for testing the mean difference between males and females.

† comparison between males and females was not made due to the highly skewed distribution of this value.

Table 3

Carbohydrate factors in predicting blood lipids, Seasonal Variation of Blood Cholesterol Study, Worcester, Massachusetts, 1994–1998 (N = 574)

	Units	Unadjusted				Adjusted*			
		Cross Sectional		Longitudinal		Cross Sectional		Longitudinal	
		β (SE)	p	β (SE)	p	β (SE)	p	β (SE)	p
Dependent Variable = Total cholesterol (mg/dl)									
% Carbohydrate†	5	−2.95 (1.04)	<0.01	−0.86 (0.34)	0.01	−1.27 (2.94)	0.67	−1.72 (2.80)	0.54
Grams Carbohydrate	20	−1.70 (0.42)	<0.01	0.52 (0.16)	<0.01	−1.61 (0.74)	0.03	0.52 (0.61)	0.39
Glycemic Index	5	−0.79 (1.54)	0.61	0.43 (0.35)	0.23	−0.39 (1.52)	0.80	0.48 (0.38)	0.21
Glycemic Load	25	−2.42 (0.62)	<0.01	0.78 (0.19)	<0.01	−1.85 (0.74)	0.01	1.04 (0.36)	0.004
Dependent Variable = LDL-C (mg/dl)									
% Carbohydrate†	5	−2.15 (0.93)	0.02	−0.54 (0.31)	0.08	0.54 (2.70)	0.84	−0.84 (2.58)	0.75
Grams Carbohydrate	20	−1.55 (0.37)	<0.01	0.22 (0.14)	0.13	−1.72 (0.68)	0.01	0.14 (0.58)	0.81
Glycemic Index	5	0.06 (1.40)	0.96	0.31 (0.32)	0.33	-6.4×10^{-2} (1.41)	0.96	0.23 (0.35)	0.51
Glycemic Load	25	−2.09 (0.55)	<0.01	0.42 (0.18)	0.02	−1.73 (0.68)	0.01	0.64 (0.33)	0.05
Dependent Variable = HDL-C (mg/dl)									
% Carbohydrate†	5	−0.55 (0.30)	0.07	−0.20 (0.10)	0.06	−3.50 (0.86)	<0.01	−1.86 (0.84)	0.03
Grams Carbohydrate	20	−0.73 (0.12)	<0.01	0.10 (0.05)	0.04	−0.49 (0.21)	0.02	0.12 (0.18)	0.52
Glycemic Index	5	−1.02 (0.44)	0.02	1.4×10^{-2} (0.11)	0.90	−0.89 (0.38)	0.02	3.1×10^{-2} (0.12)	0.79
Glycemic Load	25	−1.15 (0.17)	<0.01	0.13 (0.06)	0.03	−0.77 (0.20)	<0.01	0.20 (0.11)	0.07
Dependent Variable = Log (Triacylglycerols)									
% Carbohydrate†	5	-1.7×10^{-2} (1.5×10^{2})	0.26	2.4×10^{-3} (5.2×10^{-3})	0.65	0.09 (0.04)	0.047	0.04 (0.04)	0.36
Grams Carbohydrate	20	1.3×10^{-2} (6.1×10^{-3})	0.03	7.2×10^{-3} (2.4×10^{-3})	<0.01	1.1×10^{-2} (1.1×10^{-2})	0.31	6.0×10^{-3} (9.5×10^{-3})	0.52
Glycemic Index	5	8.9×10^{-3} (2.2×10^{-2})	0.69	-4.6×10^{-5} (5.4×10^{-3})	0.99	2.5×10^{-2} (2.0×10^{-2})	0.22	4.7×10^{-3} (6.0×10^{-3})	0.44
Glycemic Load	25	1.8×10^{-2} (9.0×10^{-3})	0.04	7.8×10^{-3} (3.0×10^{-3})	0.01	9.6×10^{-3} (1.0×10^{-2})	0.35	2.1×10^{-3} (5.6×10^{-3})	0.71
Dependent Variable = TC/HDL-C ratio									
% Carbohydrate†	5	−0.006 (0.008)	0.48	0.0005 (0.002)	0.81	0.38 (0.10)	<0.001	0.20 (0.09)	0.03
Grams Carbohydrate	20	0.002 (0.0008)	0.008	0.0002 (0.0003)	0.34	3.0×10^{-2} (2.0×10^{-2})	0.22	1.4×10^{-2} (2.0×10^{-2})	0.48
Glycemic Index	5	7.9×10^{-2} (6.0×10^{-2})	0.19	4.6×10^{-3} (1.2×10^{-2})	0.70	9.1×10^{-2} (5.2×10^{-2})	0.22	1.6×10^{-2} (1.3×10^{-2})	0.22
Glycemic Load	25	7.3×10^{-2} (2.4×10^{-2})	<0.01	5.9×10^{-3} (6.5×10^{-3})	0.36	4.1×10^{-2} (2.6×10^{-2})	0.11	3.7×10^{-3} (1.2×10^{-2})	0.76

Abbreviations used in the table: Units: number changed in carbohydrate factors, β: regression coefficient, SE: standard error, LDL-C: low-density lipoprotein cholesterol, HDL-C: high-density lipoprotein cholesterol, and TC: total cholesterol:

*Adjusted for gender, BMI, smoking status, age, energy intake, % saturated fat intake, % alcohol intake, % protein, % monounsaturated fat, % polyunsaturated fat, dietary cholesterol, leisure time physical activity (met-hr/day), race/ethnicity, education, and season of year at lipids assessment.

†energy intake is not included in the adjusted model to avoid over-controlling.