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The effect of type and amount of dietary carbohydrate on biomarkers of glucose homeostasis and C-reactive protein in overweight or obese adults: Results from the OmniCarb trial

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Abstract

OBJECTIVE—The glycemic index (GI) of dietary carbohydrate is thought to affect glucose homeostasis. Recently, the OmniCarb Trial reported that a low GI diet did not improve insulin sensitivity. We conducted this ancillary study of the OmniCarb Trial to determine the effects of GI and carbohydrate content on glucose homeostasis and inflammation.

RESEARCH DESIGN AND METHODS—OmniCarb was a randomized crossover feeding study conducted in overweight or obese adults without diabetes (N=163). Participants were fed each of 4 diets for 5 weeks with 2-week washout periods. Weight was held constant. Diets were: high GI (GI 65) with high carbohydrate (58% kcal), low GI (GI 45) with low carbohydrate (40% kcal), low GI with high carbohydrate; and high GI with low carbohydrate. We measured glycated albumin (GA), fructosamine, and high sensitivity C-reactive protein (CRP) at baseline and following each dietary period. These biomarkers were compared within-person between diets.

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Competing Interests Statement

The authors have no conflicts of interest to report.

Contributorship Statement

This trial is registered at clinicaltrials.gov, number: NCT00608049.

SPJ, ERM, FMS wrote manuscript, researched data. LS, RHC reviewed/edited manuscript. VJC, LJA reviewed/edited manuscript and participated in the primary trial design.

SPJ and FMS are guarantors of this work and take full responsibility for the work as a whole, including the study design, access to data, and the decision to submit and publish the manuscript.

Data Sharing

The statistical code for this analysis is available from the corresponding author, SPJ. The dataset is available from FS on request.

RESULTS—The study population was 52% female and 50% black. Mean age was 53 (SD, 11) years; mean BMI was 32 (SD, 6) kg/m². Reducing GI had no effect on GA or fructosamine, but increased fasting glucose in the setting of a high carbohydrate diet (+2.2 mg/dl; *P*=0.02). Reducing carbohydrate content decreased GA in the setting of a high GI diet (−0.2%; *P*=0.03) and decreased fructosamine in the setting of a low GI diet (−4 μmol/L; *P*=0.003). Reducing carbohydrate while simultaneously increasing GI significantly reduced both GA (−0.2%; *P*=0.04) and fructosamine (−4 μmol/L; *P*=0.009). Neither reducing GI nor amount of carbohydrate affected insulin or CRP.

CONCLUSIONS—Reducing carbohydrate, regardless of high or low GI, decreased GA and fructosamine. This suggests that reducing carbohydrate content, rather than GI, is a better strategy for lowering glycemia in adults at risk for diabetes.

Keywords

carbohydrate; diet; glycemic index; glycated albumin; fructosamine; C-reactive protein; randomized controlled trial

INTRODUCTION

Randomized clinical trials examining the effects of dietary patterns on traditional cardiovascular disease risk factors have helped inform national dietary guidelines (1,2). Recently the OmniCarb Trial tested whether modifying the amount or type of carbohydrate could reduce cardiovascular risk factors (3). It was found that at high dietary carbohydrate content, reducing glycemic index (GI) decreased insulin sensitivity and increased LDL cholesterol, but did not affect HDL cholesterol, triglycerides, or blood pressure (3). In this trial, insulin sensitivity was determined by an oral glucose tolerance test, conducted in the morning after an overnight fast; glucose and insulin were measured at 7 time points. However, the oral glucose tolerance test does not represent the overall glycemic effects of dietary patterns that vary in amount and type of carbohydrate. Glycated albumin and fructosamine, markers of 2–3 week cumulative exposure to blood glucose, may be especially well-suited for evaluating the effects of dietary carbohydrates on glycemia in an intermediate-term trial setting. Both glycated albumin and fructosamine are formed by glycation reactions during which glucose binds with intravascular proteins, including albumin (4,5) and are associated with risk of diabetes (6,7) and cardiovascular disease events (8).

It has been hypothesized that the associations between GI and risk of diabetes and cardiovascular disease are mediated through low-grade inflammation (9,10). Observational studies have shown that C-reactive protein, a marker of inflammation, is associated with both diabetes (11) and cardiovascular disease (12). Further, several studies have described cross-sectional associations between GI and C-reactive protein (13–16); however, this has not been observed prospectively (17).

The objectives of this paper were (1) to determine the effects of diets that vary in carbohydrate type and amount on intermediate-term markers of glycemia in an overweight and obese population using glycated albumin and fructosamine, (2) to evaluate whether risk factors associated with diabetes modify the association between diet and intermediate-term

markers of glycemia, (3) to compare the cross-sectional association of these markers with traditional diabetes risk factors, and (4) to assess whether changes in GI or amount of dietary carbohydrate affect C-reactive protein. We hypothesize that reducing glycemic index and reducing content of carbohydrate will each lower intermediate-term glycemia measured by glycated albumin or fructosamine. Our secondary hypothesis is that lowering glycemic index or content of carbohydrate would lower inflammation measured by C-reactive protein.

Research Design and Methods

The Effect of Amount and Type of Dietary Carbohydrates on Risk for Cardiovascular Heart Disease and Diabetes Study (OmniCarb) was an investigator-initiated National Heart, Lung, and Blood Institute-sponsored study, whose rationale and main results have been published (3). OmniCarb was a randomized, controlled, crossover trial, comprising 4-dietary intervention periods. Participants each consumed 4 distinct diets in random order. The 4 diets varied by GI (65 versus 45) and carbohydrate amount (40% vs. 58% kcal) as follows: high carbohydrate and high GI, high carbohydrate and low GI, low carbohydrate and high GI, or low carbohydrate and low GI. A detailed description of the diets is in Table 1. GI is a measure of the amount that blood glucose increases after a standardized amount of carbohydrates (18). It is a relative scale based on the area under the glucose curve during a 2-hour period after eating a standardized amount of a food item (18). For example, using white bread as a reference food, white rice and baked potatoes have a higher GI, while oat bran bread and long-grain rice have a lower GI (19). Currently, glucose content is the reference.

Participant Recruitment

Trial participants were adult men and women, residing in and around Boston, MA, and Baltimore, MD. Participants were aged 30 and older with systolic blood pressure (SBP) and diastolic blood pressure (DBP) ranging from 120–159 and 80–99 mmHg, respectively. Persons with a prior diagnosis of diabetes or cardiovascular disease and persons taking medications for blood pressure, lipids, or diabetes were excluded from trial participation (3). Institutional Review Boards at Johns Hopkins University, Brigham & Women's Hospital, and the Harvard School of Public Health approved the study protocol.

Controlled Feeding

Feeding by cohort began in August 2009 and was completed in September 2010. Participants were randomly assigned to 1 of 8 dietary sequences of the 4 diets and received 100% of their meals from each study center (3). Each diet was designed to be healthful like the DASH diet (1) using commonly available foods and was reduced in saturated fat, cholesterol, and sodium, but rich in fruits, vegetables, fiber, potassium, and other minerals. Both fiber and sodium content was constant across diets. Because many low GI foods are high in fiber and many high GI foods are low in fiber, we established similar fiber contents across diets by including low-fiber, low-GI foods such as pasta and high-fiber high-GI foods such as instant oatmeal and bananas, and adding unprocessed wheat bran to 3 of the breakfasts and 2–3 dinners.

Study participants underwent an 8-day run-in phase during which each of the 4 study diets was given for 2 days. Afterward, participants consumed each diet over a 5-week period followed by a 2-week washout, during which they ate a self-selected diet. Calorie targets were determined for each participant based on their body size, sex, and physical activity level. Calorie intake was adjusted throughout the trial to keep weight within 2% of participants' baseline values. Participants were encouraged to maintain the same activity levels and alcohol consumption throughout the study. Attendance at meals was recorded. Participants were required to eat one principal meal on-site and were observed while eating. Participants were sent home with the remaining two meals. Every day, participants completed a diary in which they listed their consumption of protocol and non-protocol foods. Overall, adherence was high; all study foods with no non-study foods were consumed on 96% of person-days (3). Any alcohol consumption was reported on 11% of person-days.

Measurement of outcomes: fasting glucose, fasting insulin, glycated albumin, fructosamine, and high sensitivity C-reactive protein

Fasting laboratory specimens were collected at baseline prior to randomization and at the completion of each 5-week feeding period. The mean number of weeks between laboratory measurements in consecutive diet periods was 7.6 (SD, 2.0). Glucose and insulin were measured in serum after a brief storage period soon after specimen collection as part of the original trial protocol (3). Additional plasma specimens were stored at -70°C . Glycated albumin (Asahi Kasei Lucica GA-L; Asahi Kasei Pharma Corporation, Tokyo, Japan), fructosamine, and high sensitivity C-reactive protein were measured in year 2012 using stored plasma specimens with a Siemens Dimension Vista 1500 chemical analyzer (Siemens Healthcare Diagnostics, Glasgow, DE, Germany). Glycated albumin was assessed via an enzymatic method, involving oxidase and peroxidase reactions that yield a salt with a blue-purple pigment, measured via spectroscopy. Fructosamine was measured with a colorimetric assay to detect the rate at which ketoamines reduce nitrotetrazolium-blue (NBT) to formazan in an alkaline solution. The rate of formation of formazan is directly proportional to the concentration of fructosamine. The observed inter-assay coefficients of variation for glycated albumin and fructosamine were 7.6% (glycated albumin, mean 13.7%) and 2.0% (fructosamine, mean $309\ \mu\text{mol/L}$), while manufacturer reported performance is $<3\%$ and 2.9% , respectively. Glycated albumin was expressed as a percentage of total serum albumin according to the manufacturer's instructions: $[(\text{glycated albumin concentration in g/dL} / \text{serum albumin concentration in g/dL}) * 100 / 1.14] + 2.9$. In a general population without diabetes, glycated albumin ranges from 7.7 to 15.4%; while fructosamine ranges from 89 to $267\ \mu\text{mol/L}$ (8). High sensitivity C-reactive protein was also measured in stored plasma using an assay of polystyrene particles coated with monoclonal antibodies specific to C-reactive protein (20). The C-reactive protein assay's coefficient of variation was 2.3% (mean $3.0\ \text{mg/L}$).

Other Covariate Measurements and Definitions

Body mass index (BMI) was calculated using baseline height and weight measurements and categorized as overweight ($25\text{--}29.9\ \text{kg/m}^2$) or obese ($\geq 30\ \text{kg/m}^2$). Waist circumference (cm) was measured at the level of the umbilicus. The homeostasis model assessment index (HOMA) was calculated as follows: $\text{HOMA} = [(\text{fasting serum insulin concentration in}$

$\mu\text{U/mL}) \times (\text{fasting serum glucose concentration in mg/dL})/405$ (21). HOMA was further dichotomized based on the baseline median value of 1.48 units. Ultracentrifugation was used to prepare LDL for cholesterol measurements (3). HDL-cholesterol was measured by a precipitation method using dextran sulfate 50,000 MW and magnesium chloride (3). Cholesterol and triglycerides were measured by enzymatic assays. Triglycerides were dichotomized using the baseline median value of 83.8 mg/dL. Hypertensive status (yes or no) was determined by an average of 3 baseline blood pressure measurements for which mean systolic blood pressure was >140 mmHg or mean diastolic blood pressure was >90 mmHg.

Statistics

The main outcomes examined in this study were plasma glycated albumin and plasma fructosamine. Serum fasting glucose and insulin were reported previously (3) but are included here as relevant outcomes to the research question. C-reactive protein was measured to examine the effects of the diets on inflammation. The primary comparison in this study was the end-of-period concentrations of the primary outcomes by diet. There were six pairs of dietary differences for each outcome: (i) high carbohydrate, high GI vs. high carbohydrate, low GI; (ii) low carbohydrate, high GI vs. low carbohydrate, low GI; (iii) high carbohydrate, high GI vs. low carbohydrate, high GI; (iv) high carbohydrate, low GI vs. low carbohydrate, low GI; (v) high carbohydrate, high GI vs. low carbohydrate, low GI; and (vi) high carbohydrate, low GI vs. low carbohydrate, high GI.

Although this 4-period crossover study could be analyzed as a factorial design, we considered that GI may have a stronger effect in a diet with high than low carbohydrate content and that carbohydrate amount may have a stronger effect when the GI is high rather than low. Therefore, a factorial analysis was considered inappropriate as described in the trial's protocol (3).

In addition, using the high GI, high carbohydrate diet for comparison, we performed stratified analyses by covariates known *a priori* to be associated with insulin resistance, namely, race (non-Hispanic black versus white), baseline hypertensive status, baseline triglycerides, baseline BMI, baseline HOMA, baseline fasting glucose (<100 mg/dL, 100 mg/dL), baseline glycated albumin (based on the population median 14.8%, $>14.8\%$), and baseline fructosamine (based on the population median 234.5 $\mu\text{mol/L}$, >234.5 $\mu\text{mol/L}$). All of the above comparisons between strata were performed via generalized estimating equation (GEE) regression models, using a Huber and White robust variance estimator (22), which assumed an exchangeable working correlation matrix. *P*-values for each stratum were generated using interaction terms.

Moreover, in the baseline period, when participants were eating their usual self-selected diets, we evaluated the association between glycated albumin and fructosamine with BMI, waist circumference, HDLc, LDLc, triglycerides, serum glucose, serum insulin, and HOMA using Pearson's coefficients and linear regression models adjusted for age, sex, and race.

All analyses were performed in Stata version 14.0 (Stata Corporation, College Station, TX, USA). Statistical significance was defined as $P < 0.05$ without Bonferroni correction (23).

We did not correct the alpha level because the new outcomes, glyated albumin and fructosamine, measure the same metabolic process; fructosamine like glyated albumin is formed during the glycation reaction with blood proteins.

During the laboratory process, it was discovered that 95 of 749 (13%) samples were slightly hemolyzed and 17 (2%) were moderately hemolyzed based on visual inspection. Both slight and moderate hemolysis were associated with significant decreases in glyated albumin (-1.2 and -6.1% ; P -values < 0.001) and significant increases in fructosamine (10.7 and 24.7 $\mu\text{mol/L}$; P -values < 0.001). As a result, we excluded these samples from all glyated albumin and fructosamine comparisons. We further excluded participants who did not have a blood sample for glyated albumin or fructosamine in at least 2 visits ($N = 3$), the minimum number of data points necessary for a comparison. Both hemolysis and other missing data specimens were found to be evenly distributed between feeding periods and diets. A sensitivity analysis excluding hemolyzed samples from glucose and insulin comparisons had virtually no effect on our analysis (results not shown).

RESULTS

Baseline characteristics

Baseline characteristics of the randomized, study population are in Table 2. Overall the mean age was 52.6 ± 11.4 years; 52% of the participants were women and 50% were non-Hispanic black race. Furthermore, 56% of the study population were obese and 26% had hypertension.

Comparison of glycemic markers between diets

Reducing GI had no effect on glyated albumin or fructosamine in the context of either high or low carbohydrate intake (Figure 1). Reducing GI increased fasting glucose in the context of a low carbohydrate diet (low GI/low carbohydrate vs. high GI/low carbohydrate: $+2.2$ mg/dL, 95% CI: 0.4, 4.0; $P = 0.02$), but not significantly in the context of a high carbohydrate diet ($+1.7$ mg/dL, 95% CI: -0.5 , 3.7; $P = 0.12$). Reducing GI had no effect on fasting insulin.

Reducing dietary carbohydrate in the setting of a high GI diet decreased glyated albumin (-0.2% , 95% CI: -0.4 , -0.02 ; $P = 0.03$), while reducing dietary carbohydrate in the setting of a low GI diet significantly decreased fructosamine (-3.9 $\mu\text{mol/L}$, 95% CI: -6.4 , -1.3 ; $P = 0.003$). Reducing amount of dietary carbohydrate while simultaneously increasing GI, decreased both glyated albumin and fructosamine (glyated albumin -0.2% , 95% CI: -0.4 , -0.01 ; $P = 0.04$; fructosamine -3.5 $\mu\text{mol/L}$, 95% CI: -6.2 , -0.8 ; $P = 0.01$). The decreases in glyated albumin and fructosamine from reducing dietary carbohydrates were attenuated when GI was simultaneously decreased. Reducing dietary carbohydrates did not affect fasting glucose except when GI was reduced simultaneously. Reducing dietary carbohydrates had no effect on fasting insulin regardless of low GI, high GI, increase in GI, or decrease in GI.

Stratified analyses and other sensitivity analyses

The diets' effects on glycated albumin and fructosamine by strata of factors associated with diabetes are displayed in Supplement Tables S1 and S2. Ultimately, there was little if no evidence of interactions by race, hypertension status, baseline triglycerides, BMI, HOMA, baseline fasting glucose, baseline glycated albumin, or baseline fructosamine.

The baseline measures of glycated albumin and fructosamine were correlated ($r = 0.58$) (Supplement Table S2). Lower glycated albumin and fructosamine were associated with lower BMI and waist circumference.

There was no effect from change in either GI or amount of dietary carbohydrate on high sensitivity C-reactive protein (Supplement Figure S1).

CONCLUSIONS

In this trial of adults at risk for diabetes, we found that reducing GI increased fasting glucose, particularly in the setting of a low carbohydrate diet. However, these increases in fasting glucose had little influence on markers of 2–3 week glycemia, glycated albumin and fructosamine, likely because of the opposite direction of effects conferred by reducing postprandial glycemia from the reduced GI diet. In contrast, reducing the amount of dietary carbohydrate generally reduced glycated albumin and fructosamine, suggesting that reducing dietary carbohydrate may represent a more effective strategy than reducing GI for lowering glycemia in overweight or obese adults.

In this ancillary study to the OmniCarb trial, we present the effects of type and amount of carbohydrate on glycated albumin and fructosamine. Glycated albumin and fructosamine are intermediate-term markers of glycemia that are highly associated with hemoglobin A1c (24) and are elevated prior to the development of diabetes (6). They are formed by a nonenzymatic glycation reaction in which glucose forms a covalent bond with intravascular proteins, primarily albumin (4,5), and, as observed in this study, they are correlated with each other ($r = 0.58$). The half-life of these markers ranges between 17–21 days; thus glycated albumin and fructosamine represent an average exposure to glucose concentrations in the blood in the previous 2–3 weeks (4,5,25–27). There is growing evidence that these intermediate-term markers of glycemia are more meaningful indicators of risk for diabetes, cardiovascular disease, and mortality than single glucose measurements (7,8,24,28) by representing the aggregate effects of both fasting glucose and postprandial glucose excursions (29).

GI is a measure of carbohydrate “quality,” based on the rate of glucose released into circulation after consumption of a fixed amount of carbohydrates. It is typically determined by comparing the area of the glucose curve following the consumption of 50 grams of a carbohydrate food relative to a referent 50-gram food such as white bread or glucose (18). GI has been the target of nutrition policy advocates based on numerous observational studies (30), showing that lower GI is associated with lower risk of type 2 diabetes (31), cardiovascular disease (32,33), and mortality (34). Mechanistic explanations for these relationships have included the hypothesis that a high GI diet increases late postprandial

hepatic glucose, reflected in higher fasting glucose measures (35). In our study, we found the contrary to be true, that is, low GI diets increased fasting glucose. We suspect that the reduction in GI caused a compensatory increase in gluconeogenesis and a reduction in insulin sensitivity in order to stabilize fasting glucose levels, protecting against night-time and early morning hypoglycemia. This mitigated the expected increase in glycemia from high postprandial glucose excursions during the high GI diet. As a result, there was no effect on glycated albumin or fructosamine. This is consistent with a number of trials, which found no effect from GI on fructosamine or other glycation products like hemoglobin A1c in nondiabetic populations (36).

Like GI, the proportion of calories derived from carbohydrates has been associated with risk of diabetes and cardiovascular disease in observational studies (31,37,38) and clinical trials (39–41). Unlike GI, we found that reducing the amount of dietary carbohydrates was associated with reductions in either glycated albumin (high GI diets) or fructosamine (low GI diets). Interestingly, there were significant reductions in both glycated albumin and fructosamine when GI was increased while the amount of carbohydrates was decreased, but not when GI was decreased as amount of carbohydrates was decreased. This likely reflects the fact that decreasing GI increased fasting glucose, which would oppose the effect of reducing carbohydrates on glycated albumin and fructosamine. Overall, these results suggest that dietary carbohydrate is more important than GI as a dietary factor influencing aggregate glycemic exposure. Whether these differences in the effect of dietary carbohydrate on glycemia affect progression to diabetes or CVD should be a focus of subsequent research.

Prior studies have shown that a higher GI diet is associated with higher levels of C-reactive protein (13–16), a marker of inflammation associated with risk for diabetes or cardiovascular disease (11,12). This has led to the hypothesis that inflammation may mediate the association of GI with cardiovascular disease (9,10). In this ancillary study, we found no effect from either GI or amount of carbohydrates on C-reactive protein levels. This suggests that carbohydrates do not act via inflammation with regards to the pathogenesis of diabetes or cardiovascular disease.

This study has several limitations. The feeding periods were too brief for the observation of clinical events, making it necessary to examine surrogate markers as outcomes; however, we consider the feeding periods sufficient to determine effects on the surrogate outcomes. Based on our experience with prior feeding studies (1,2,42), effects of diet are observed soon after the initiation of feeding with the majority of effects taking place within the first two weeks (43). Thus, we are confident that even those with a short feeding period would still manifest an effect from GI if one existed. We reviewed the literature on duration for effects of GI, and if anything find a diminution of effects on risk factors with duration, especially beyond 6 months (44). In a systematic review and meta-analysis that included 421 participants in 4 trials with at least 6 months duration, reducing GI had no effect on hemoglobin A1C (mean difference -0.1% , $P=0.7$) (45). Finally, 15% of the plasma specimens were hemolyzed and subsequently excluded from our analyses of glycated albumin and fructosamine, which reduced statistical power.

With regards to strengths, our study was a randomized, controlled feeding study that employed a crossover design to assess dietary effects on glucose homeostasis. Diets were highly regulated and isocaloric with virtually no weight change over the course of the trial, reducing the impact of weight on our outcomes. Furthermore, we utilized non-traditional markers of glucose homeostasis that reflect intermediate-term glycemic exposure, permitting more accurate assessments of diet-induced changes in 2–3 week glucose homeostasis. Finally, our study population was quite diverse with 50% of participants being black, a population at risk for pre-diabetes and diabetes.

In conclusion, we found that a lower GI diet increases fasting glucose, while diets with a lower amount of dietary carbohydrates reduce glycated albumin and fructosamine, markers of 2–3 week glycemia. Together these findings suggest that a low carbohydrate diet most effectively lowers glycemia in adults at risk for diabetes. Additional research is needed to determine the long-term effects of these physiologic changes in glucose homeostasis on clinical outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations used

OmniCarb	The Effect of Amount and Type of Dietary Carbohydrates on Risk for Cardiovascular Heart Disease and Diabetes Study
BMI	body mass index
LDL	low density lipoprotein
HDL	high density lipoprotein
HOMA	homeostatic model assessment
DASH	Dietary Approaches to Stop Hypertension
GEE	generalized estimating equation
CI	confidence interval

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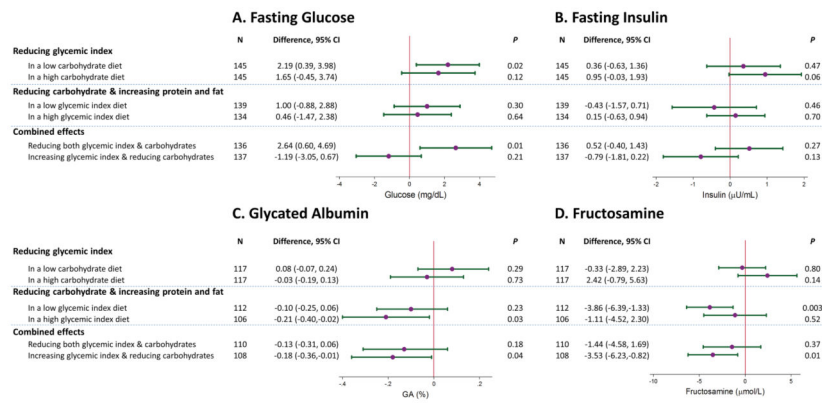


Figure 1. (A) Fasting serum glucose (mg/dL), (B) fasting serum insulin (μ U/mL), (C) plasma glycated albumin (%), and (D) plasma fructosamine (μ mol/L) measured at the end of each feeding period: between diet comparisons, mean and 95% confidence intervals. The feeding periods are grouped by glycemic index comparisons (low versus high glycemic index), carbohydrate proportion (low versus high proportion), and changes in both glycemic index and amount of carbohydrates, i.e. reductions in both or an increase in glycemic index while decreasing amount of carbohydrate. Hemolyzed samples comprising 15% of the total were removed from the glycated albumin and fructosamine comparisons.

Table 1

Nutrient composition of the four diets used in the OmniCarb*

Dietary Pattern	High Carbohydrate/High Glycemic Index	High Carbohydrate/Low Glycemic Index	Low Carbohydrate/High Glycemic Index	Low Carbohydrate/Low Glycemic Index
Energy, kcal	2011	1998	2011	1993
Glycemic Index	66	41	65	40
Carbohydrates, %kcal	58	57	41	40
Protein, %kcal	16	16	23	23
Fat, % kcal	27	27	37	37
Saturated, % kcal	6	6	7	7
Monounsaturated, % kcal	12	13	18	19
Polyunsaturated, % kcal	7	8	10	10
Animal Protein, g	42	46	78	81
Vegetable Protein, g	39	38	39	39
Fiber, g	32	37	29	33
Fructose, g	48	40	26	28
Cholesterol, mg	90	89	170	163
Calcium, mg	1032	1051	993	995
Potassium, mg	3963	4103	3949	4026
Sodium, mg	2245	2211	2305	2199
Magnesium, mg	462	429	468	440

* Estimated from food analysis software (ESHA Food Processor SQL, version 10.2, ESHA Research). Note that macronutrient estimates may not sum to 100% due to rounding.

Table 2

Baseline characteristics of trial participants (N = 163), mean (SD) or No. (%)

Age, y	52.6 (11.4)
Women, %	85 (52)
Race, %	
Non-Hispanic White	66 (40)
Non-Hispanic Black	82 (50)
Hispanic	11 (7)
Asian	4 (2)
Body mass index, kg/m ²	32.3 (5.5)
Body mass index, %	
25–29.9	71 (44)
30	92 (56)
Waist circumference, cm	104.4 (13.5)
Homeostasis model assessment (HOMA), units	1.9 (1.6)
Triglycerides, mg/dL	104.6 (67.1)
HDL cholesterol, mg/dL	58.3 (16.0)
LDL cholesterol, mg/dL	153.0 (42.1)
Systolic blood pressure, mm Hg	132.0 (9.1)
Diastolic blood pressure, mm Hg	80.0 (7.5)
Baseline hypertensive status [*] , %	
Non-hypertensive	120 (74)
Hypertensive	43 (26)

^{*} Defined as baseline SBP ≥ 140 or DBP ≥ 90 mmHg

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