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Providing Food to Treat Adolescents at Risk for Cardiovascular Disease

Sarah D. de Ferranti, MD, MPH, **Carly E. Milliren, MPH**, **Erica Rose Denhoff, MPH**, **Nicolle Quinn, RD**, **Stavroula K. Osganian, MD, ScD**, **Henry A. Feldman, PhD**, **Cara B. Ebbeling, PhD**, and **David S. Ludwig, MD, PhD**

Department of Cardiology, (SdeF) Clinical Research Center, (CEM, ERD, SKO, HAF, NQ); Department of Medicine, (SKO, HAF); New Balance Foundation Obesity Prevention Center (CBE, DSL), Boston Children's Hospital, Boston, MA, USA.

Abstract

Objective—Diet modification is recommended to treat childhood cardiovascular (CV) risk factors; however, the optimal dietary strategy is unknown.

Methods—In a randomized trial the effect of a low-fat (LF) and a low-glycemic-load (LGL) reduced-calorie diet were examined in youth with overweight/obesity with CV risk factors. Using a novel intervention, we delivered LF or LGL meals and nutrition education to the home for 8 weeks (*Intensive Phase*), followed by 4-months *Maintenance* without food provision. Between-group differences in the change in insulin area-under-the-curve (InsAUC) by oral glucose tolerance test and other risk factors were analyzed.

Results—Overall, participants (n=27) showed substantial improvement during the *Intensive Phase*, including InsAUC (−59±18.2 μU/mL*120mins, p=0.004), total cholesterol (−9.9±3.6mg/dL, p=0.01), weight (−2.7±0.5kg, p<0.001), waist circumference (−3.1±0.8cm, p<0.001), HOMA-IR (−1.7±0.4, p<0.001), systolic BP (−5±1.4 mmHg, p=0.002) and CRP (−0.1±0.1mg/dL, p=0.04). There were minimal between-group differences; the LF group showed greater declines in HDL-C (p=0.005) and fasting glucose (p=0.01) compared to the LGL group. Improvements waned during *Maintenance*.

Corresponding address, including reprints: Sarah D. de Ferranti, MD MPH, Department of Cardiology, FA607, Children's Hospital Boston, 300 Longwood Avenue, Boston, MA 02115, t. 617 355-0955, f. 617 730-0600, ; Email: sarah.deferranti@cardio.chboston.org

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Contributors' Statement:

Dr. de Ferranti was the primary lead for the design of the study, conducted the research, drafted and edited the manuscript and has responsibility for the final content of the manuscript. Ms. Milliren conducted the statistical analysis under supervision of Dr. Henry Feldman. Ms. Denhoff recruited participants, served as the primary patient contact, and participated in the drafting and editing of the manuscript. Ms. Quinn supervised the cooking and provision of foods and beverages, the nutritional analysis, and the calculation of resting energy needs and total caloric intake. Dr. Osganian provided input with regard to study design and conduct, and participated in the editing of the manuscript. Dr. Feldman was involved in the study conceptualization, including the statistical analysis plan, and supervised the analysis. Dr. Ebbeling was involved in the design of the study, including the nutrition education component, and participated in drafting and editing the manuscript. Dr. Ludwig served as the senior mentor on the project, participating in all aspects of the study, including the design and conduct of the study, and in conceptualizing and editing the manuscript.

Conclusions—Home delivery of LF or LGL diets resulted in rapid and clinically important improvements in CV risk factors that diminished without food delivery, and did not differ based on dietary intervention. If scalable, food provision may represent an alternative nutrition treatment strategy.

Keywords

child; obesity; adolescent; cardiovascular risk factor; diet; glycemic index

INTRODUCTION

Excessive body weight, the most common pediatric chronic disease ([1]), predicts early morbidity and increased mortality ([2]). Obesity in youth carries a significant burden of cardiovascular (CV) risk factors ([3]), and is associated with premature atherosclerosis ([4]). The presence of multiple CV risk factors during childhood is associated with both adult metabolic syndrome ([5]) and CV disease events ([6]).

Dietary change is the first line therapy to reduce obesity-related CV risk ([3]) and lifestyle interventions have been shown to reduce CV risk factors in clinical trials ([7]). However, the effects of lifestyle interventions are relatively modest in the research setting, and in clinical practice ([8]). The disappointing results of these behaviorally oriented interventions may be related to challenges with adherence – including adoption and maintenance ([9]), or uncertainty about the optimal dietary prescription.

Nutrition prescriptions to reduce CV risk factors have previously focused on reducing dietary fat ([10]); however, the results of pediatric low fat (LF) diet studies are disappointing ([11]). Some clinicians and investigators have used a low glycemic load (LGL) diet to treat nutrition-related CV risk factors. Glycemic load, a dietary variable that quantifies how a food, meal, or diet affects blood glucose in the postprandial period ([12]), has been validated in physiology studies ([13]) and may be effective in adults in inducing short-term weight loss ([14]), improving CV risk factors ([15]), and reducing CV risk ([16]) and diabetes ([17]). Several pediatric studies suggest a LGL diet may reduce body weight and CV risk factors among adolescents ([18], [19]); however, these studies have not specifically focused on children with CV risk factors. This study examined the effect of LF and LGL diets during an energy-restricted *Intensive Phase* of food provision to participant homes, followed by an ad libitum *Maintenance Phase*. The aim was to disentangle the effects of dietary composition from variable compliance in the treatment of youth with CV risk factors.

METHODS

Study design

In this pilot study, children and adolescents with overweight/obesity and additional CV risk factors were randomly assigned to a LF or a LGL diet. Food and nutrition education were delivered directly to the home during an 8-week *Intensive Phase* in a modified feeding protocol, followed by a 4-month *Maintenance Phase* that relied on nutrition education. The primary endpoint of the trial was the difference between the LF and LGL groups in the

change from baseline to 8 weeks in insulin sensitivity, represented by the insulin area under the curve (InsAUC) measured during a 2 hour oral glucose tolerance test (OGTT). Additional outcomes included homeostatic model assessment-insulin resistance [HOMA-IR] and HgA1c, fasting lipids, blood pressure (BP), and C-reactive protein (CRP). There were 4 study visits: screening, baseline, at the end of the *Intensive Phase* (8 weeks) and at the end of the *Maintenance Phase* (6 months). Study visits and food preparation were conducted at Boston Children's Hospital (BCH) Clinical and Translational Study Unit from May 2007 to March 2012. Written informed consent was obtained from participants or parents. The study was approved by the BCH Institutional Review Board; (clinicaltrials.gov registration NCT01080339).

Participants

Participants were recruited from clinical programs treating pediatric obesity and complications, Craigslist, and community practices. Individuals aged 8–21 years were eligible to participate if they had elevated body mass index (BMI) ≥ 85th percentile using CDC reference population, ([20]) a fasting insulin ≥ 10 μU/mL, and at least 2 additional CV risk factors. Additional risk factors were defined as 1) fasting triglycerides (TG) >100 mg/dL, 2) high-density lipoprotein cholesterol (HDL-C) <50 mg/dL, except for boys ages 15–19 years, in whom the cutpoint was <45 mg/dL, 3) systolic BP >90th percentile for gender, age and height ([3]), and 4) fasting glucose ≥100 mg/dL or elevated fasting insulin (>15 μU/mL). Thus all participants had a fasting insulin of at least 10 μU/mL to be considered eligible, and some had an insulin >15 μU/mL as an additional qualifying factor. Exclusion criteria included weight >275 lbs. (125 kg) due to concerns about venous access, current or anticipated pregnancy, major medical illness or medications that might significantly affect CV risk factors or weight (e.g. thyroid disorders), alcohol, tobacco, or other drug use, serious food allergy, or abnormalities at screening that indicated a need for pharmacotherapy. For this pilot study, we also excluded participants whom we anticipated would have significant difficulty following the study protocol (e.g. behavioral issues, major food restrictions or aversions) or who lived outside a reasonable driving distance.

Participants were randomized to diet groups using computer-generated assignments prepared by the BCH Clinical Research Center. Randomization was stratified by gender and performed in permutated blocks to support equal distribution between the two study diets over time. Participants were not informed of their group assignment, although some may have guessed based on the food and nutrition information provided. Participants and their families received food, dietary counseling, and parking or public transportation vouchers, iTunes credits and movie vouchers, and a voucher towards physical activity programming at study completion.

Intervention

During the *Intensive Phase* all participants received 3 customized meals and 1 snack per day prepared according to their assigned diets (LF or LGL) for 6 out of 7 days per week. On the 7th day participants were instructed to eat along the assigned dietary strategy. Foods and beverages were prepared and portioned in the metabolic kitchen and were delivered as a combination of uncooked staples, and partially and fully prepared meals and snacks

delivered in quantities to supply a caloric deficit of ~ 25% to induce modest weight loss over the course of 8 weeks. Energy requirements were calculated based on the Schofield equation for calculating resting energy expenditure ([21]); we used an activity factor of 1.2–1.5, adjusted to each participants' reported activity to calculate energy demands. Measures were taken to promote adherence including appropriately portioned planned and "emergency" snacks to prevent eating outside the dietary assignment, the use of a restaurant-style menu developed by a professionally trained chef, provision of lunches suitable for taking to school and family meals twice a week, and the allowance of one "free" day. Dietary change was reinforced by weekly in-person home nutrition counseling and weekly phone calls covering topics consistent with the participant's group assignment using an adapted nutrition curriculum ([22]).

During the *Maintenance Phase*, participants were asked to follow their assigned dietary strategy with no provision of food or in-person contact. The study dietitians continued to provide behavioral support by phone at regular intervals (weeks 10, 12, 16, and 20).

Dietary composition—The diets were designed to differ primarily in GL and macronutrient composition (percent fat/carbohydrate/protein; LGL: 40/40/20, LF: 20/60/20). The LGL diet targeted a glycemic index of 50% for each meal, calculated as product of the glycemic index of a food and the amount of carbohydrate in that food using glucose as the reference ([23], [24]). The LF diet was based on contemporary dietary guidelines (20% total fat of which 7% were saturated fat), ([10]) fulfilling recommendations for fiber, fruit and vegetables, limiting total fat, fat type, and cholesterol, and was designed to achieve a GI that reflects prevailing dietary patterns ([25]). Aside from differences in GL, total fat and saturated fat content, the study diets were designed to be as similar as possible, providing similar amounts of protein (20%), fiber (~30 g/day), and dietary cholesterol (≤200 mg/day) ([3]), as well as a similar intensity of treatment and palatability of meals. All participants were supplied with Flintstones multivitamins to minimize any potential discrepancies in micronutrients between the diets.

Physical activity—We asked participants to hold physical activity constant as much as possible to avoid confounding of the study outcomes. We assessed activity and inactivity by way of pedometers, which participants were asked to wear for 3 days in the week prior to the baseline, 8-week and 6-month study visits, and by way of self-report using questions from the Youth Risk Behavior Survey ([26]).

Measurements

Laboratory testing—The primary study outcome was the change from baseline to 8 weeks in insulin sensitivity measured by calculating the area under the curve (AUC) of glucose and insulin from a 2-hour oral glucose tolerance test (OGTT); samples were collected at −20, −10, 0, +10, +20, +30, +60, +90, and +120 minutes after a 75-gram oral dose of Trutol. HOMA-IR was calculated using averaged fasting glucose and insulin values from the −20, −10, and 0 time points. A fasting lipid panel was measured at all study visits according to standard methods. Additional measures included CRP, liver function tests (ALT, AST), and HgA1C.

Anthropometrics and blood pressure—Anthropometrics were measured three times including weight (nearest 0.1 kg), height and waist circumference (nearest 0.1 cm) and the average of each was used in the analysis. Percent body fat was measured in the supine position in the fasting state using bioelectrical impedance (Quantum II, RJL Systems, Inc.). Study nurses measured systolic and diastolic BP by auscultation three times in the right arm in a quiet room after five minutes of rest, according to standard methods ([3]), and averaged.

Process measures—Adherence with the dietary assignment was assessed by unannounced dietary recalls interviews conducted by telephone at baseline, 8 weeks and 6 months. Three interviews were done per participant at each time point on two randomly selected weekdays and one weekend day. Dietary intake data were collected using Nutrition Data System for Research (2006–2011, Nutrition Coordinating Center, Minneapolis, MN).

Statistical methods

Descriptive statistics at baseline by diet group and overall are reported as mean (SD) or frequency (%). Differences between treatment groups on demographic and clinical factors at baseline were assessed using two-sample t-tests for continuous variables and Fisher's exact test for categorical variables. Descriptive statistics for primary and secondary outcome measures at each visit are reported as mean±SE. Variables were assessed for normal distribution, and log-transformed if not normally distributed. Paired t-tests were used to assess pre-post changes from baseline to 8 weeks or 6 months within diet. Differences between diets in pre-post changes from baseline at each visit were assessed using independent t-tests. Selected lipid parameters (where noted in tables) demonstrated skewed distributions and were log transformed for analysis using parametric tests and re-transformed to natural units for reporting. All tests were performed at two-sided alpha-level of 0.05. SAS (version 9.3, Cary, NC) was used for all analyses. Analysis of primary and secondary outcomes was performed with the intention-to-treat principle. The baseline observation was carried forward to impute information for two subjects who missed visits. One subject dropped out prior to the 8 week visit and another dropped out after the 8 week and prior to the 6 month.

The study was designed to recruit 46 patients in order to complete data collection on 40 patients, 20 per group, which would have produced 80% power to detect a difference in mean change of 0.7 multiplied by the coefficient of variation in the primary outcome variables, assuming a pre-post (baseline-8 week) correlation of 0.7 and 5% Type I error rate. The baseline coefficient of variation was 55% for insAUC and 44% for HOMA-IR, making the estimated detectable effects 37% and 30%, respectively.

RESULTS

Participants

We approached 383 potentially eligible children and adolescents. Of these, 356 were excluded (Figure 1). Common primarily for medications or medical illness (n=104), normalization of CV risk factors at baseline (n=22), and factors that would make it difficult to comply with the requirements of the study (n=56) such as highly restrictive food

preferences or major food allergies; some were excluded for multiple reasons. This left 27 participants for randomization. The study was stopped due to slow enrollment. The study reached 59% of target enrollment, and 65% of the number necessary to complete 8 weeks of data collection estimated in the original sample size calculation.

Baseline characteristics

Table 1 describes baseline characteristics of all study participants. By design, participants had high rates of CV risk factors, including adiposity (mean BMI 98th percentile and Z score 2.05, percent body fat 35.3%). Many came from families with a history of CV risk factors and events. No differences were detected between the two dietary groups with regard to sociodemographic and anthropometric characteristics, or CV risk factors.

Intensive Phase

Measures of glucose homeostasis – fasting insulin, insAUC, HOMA-IR, and glycosylated hemoglobin (A1C) – improved in the study participants overall (Table 2 and Figure 2). In analyses by group, the LF group showed a greater decrease in fasting glucose (−8.0 ±3.2 mg/dL) than the LGL group (1 ±1.8 mg/dL, p= 0.01 for difference between groups). There were no between-group differences in the change from baseline to 8 weeks in InsAUC, the primary outcome, or in other measures of glucose homeostasis.

Lipid parameters improved over the course of the intervention in the group overall (Table 2). The only difference in lipid changes between the two diets was in HDL, which declined/worsened in the LF group by −3.7 ± 1.4 mg/dL, but not in the LGL participants (1.5 ± 1.1 mg/dL; p=0.005 for difference between groups). Improvements in other lipid measures were not statistically significant. CRP declined in the group overall; there was no difference between the LF and LGL groups.

In the group overall (N=27), body weight (−2.7 ± 0.5 kg), BMI (−1.4 ± 0.21 kg/m^2) and waist circumference (−3.1 ± 0.8 cm) decreased during the 8-week *Intensive Phase* (Table 3). There were no significant differences between the LF and LGL groups for mean change in any anthropometric measures. SBP decreased significantly among all participants combined and for the LF group, while DBP decreased significantly in the LGL group. However, between-group comparisons for changes in SBP and DBP were not statistically significant.

Dietary recalls suggested successful implementation of the dietary intervention (see Supplemental Table). The LGL diet produced a lower GL than the LF diet, as measured by recall at 8 weeks (47.9 ± 2.4 and 78.1 ±1.4 g/1000 kcal respectively). The LGL group experienced a substantial decline in glycemic load from baseline to 8 weeks of −22.4 ±3.4 g/1000 kcal (p= <0.001), as expected based on diet design, while there was no change in GL in the LF group (0.84 ±3.6, p=0.82). Added sugars declined in the LGL group (−23.8±4.6, p<0.0001), but not in the LF group (−8.9±4.2 g/1000 kcal, p=0.06). The difference between diets was significant for both GL and added sugars (p<0.0001 and p=0.03, respectively). Similarly, percent of total energy intake from saturated fat differed at 8 weeks between the two diets, LGL 10.3 ±0.4% and LF 5.7 ±0.7%. Total fat and saturated fat as a percent of total energy intake declined from baseline to 8 weeks in the LF group (−7.0 ±1.9%, p=0.004 and −3.2 ±0.6%, p=0.010). Percent of total energy intake from protein increased in the LF

group (4.0 ±1.5%) and remained stable in the LGL group (p=0.048 for difference). There were significant group differences in change from baseline to 8 week visit between the two diets in reported carbohydrate as a percent of total energy intake (p=0.02), percent total fat (p<.001), and in mono- (p=0.001), poly- and saturated fat. Physical activity by pedometer and self-report did not change from baseline to 8 weeks in the group overall and there were no between-group differences (data not shown).

Maintenance Phase

Outcomes tended to return toward baseline during the *Maintenance Phase*, although some benefits persisted. Compared to baseline measures, pooled analysis of all participants showed the group as a whole sustained small benefits in BMI (−0.8 ±0.24, p=0.003), BMI Z-score (−0.13 ±0.03, p<.001), waist circumference (−2.6 ±0.8 cm, p=0.005), percent body fat (−1.8 ±0.5%, p<.001), and SBP (−3 ±1.4 mmHg, p=0.04) at the 6 month visit. Improvements in the group overall were also maintained in HDL (2.1 ±0.7 mg/dL, p=0.005), TC/HDL −0.3 ±0.1, p=<0.001) and TG/HDL (−0.6 ±0.2, p=0.03). Dietary recalls suggested the participants retained improved dietary quality compared to baseline, including lower GL (−6.1±3.1 g/1000 kcal, p=0.06), lower added sugars (−7.52 ±3.8 g/1000 kcal, p=0.06), higher percent calories from protein (2.3 ±1.2, p=0.07); none of these changes reach statistical significance. Energy intake declined in the group overall (−221.6 ±63.4, p=0.002). There were no significant differences between the two diet groups in any of these parameters (Supplemental Table).

Discussion

Home delivery of a calorie-restricted diet combined with nutrition education over 8 weeks produced important improvements in CV risk factors in high-risk children and adolescents; notably, no consistent differences was demonstrated between the LF and LGL dietary strategy in this pilot study. Improvements in insulin resistance (HOMA-IR), adiposity, lipid measures, and blood pressure from this intensive intervention waned during the maintenance phase when food delivery stopped.

This study was designed to assess difference in insulin sensitivity between the LF and LGL diets. Weight loss studies in adults demonstrate no differential benefit of LF or LGL diets with regard to weight loss in unselected populations ([15], [27]), although individuals with high insulin secretion may experience more weight loss on LGL diets ([28], [29]). Some studies in adults suggest differential effects of diet on cardiometabolic risk factors ([30]–[32]), with LF diets tending to lower TC and LDL ([30]) but these improvements may not necessary translate into significant reductions in CV events ([33]). Insulin resistance, a key pathophysiologic process in obesity, may be improved by a LGL diet ([27], [34]–[37]), and LGL diets may be associated with fewer CV events, at least in women ([16]). Pediatric studies are also mixed, with some reporting benefits of a LGL diet for weight loss and insulin resistance compared to LF dietary advice ([22], [38]), and other trials showing no benefit ([19], [39]). None of the previous pediatric studies employed high intensity interventions, and compliance was variable.

This study did not demonstrate superiority of a LF or a LGL diet for CV risk factor reduction, either because of a true lack of differential efficacy, a dominating effect of weight loss on CV risk factors, the short-term nature of the intervention, and/or limited power. When we considered change from baseline in the 25 subjects who completed the study, there was an observed difference of 5% for the primary outcome, resulting in post-hoc power to demonstrate significance for differences of the observed magnitude of only 5–6%. Therefore, a much larger sample would be required for adequate power to test the primary hypothesis. This study faced significant difficulty with recruitment, which not only contributed to low power but also may limit the generalizability of these findings. Interpretation of these results may also be limited by self-reported measures of physical activity and diet, although these measures were complemented by more objective measures (BMI, WC and pedometer data).

Despite limited power to assess differences between diet groups, this intervention produced potentially important improvements in CV risk factors in only 8 weeks, on par with changes produced by pharmacologic therapies ([3]), that if sustained, could meaningfully impact the development of cardiometabolic disease among children at risk. We developed this novel intervention strategy as a feasible alternative to a conventionally implemented feeding study design that requires patients to come frequently to a metabolic kitchen, which is logistically challenging for adults and impractical for children. Home food provision was combined with complementary nutrition education to treat children at risk for early atherosclerosis. Implementation of the intervention was successful with very low dropout rates, (n=1 during the *Intensive Phase*), suggesting this model holds promise for future nutrition research. The results of this study are in contrast to nutrition education approaches to reducing CV risk in children and adolescents that have modest and diminishing effects in research studies, and may not produce meaningful change in real-life settings ([7, 8]).

Novel aspects of our intervention that may have contributed to success included a pragmatic approach to food provision (into the home), allowing for meal choice within the prescribed diets, oversight from a professional chef, and frequent responsive interaction between participants and the research team. As has been seen with most other obesity interventions, benefits to adiposity and other CV risk factors were not sustained and additional support would be required to promote the maintenance of the healthy diet. Home delivery of prepared meals with dietary counseling is not likely to be a feasible intervention for all youth with obesity, but might be useful for high-risk youth who might otherwise be candidates for pharmacologic management or bariatric surgery. Trials of home food delivery in adults show promise ([40]). Modifications of this intervention to reduce cost and promote scalability could include delivering staples instead of some of the home meals, the use of the internet or phone to facilitate counseling, and a more skills-based *Maintenance Phase* that includes grocery shopping and cooking demonstrations. Cost effectiveness research is needed to compare such an approach to other available treatments for children with overweight/obesity at risk for CV disease.

In conclusion, this pilot study showed LGL and LF dietary strategies were equally beneficial to children and adolescents in reducing adiposity and other CV risk factors when delivered to the home along with nutrition education. This novel intervention strategy – utilizing home

delivery of meals to supplement conventional nutrition education in a medical or public health setting – shows early promise but requires additional research.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

AAP	American Academy of Pediatrics
ALT	alanine aminotransferase
BCH	Boston Children's Hospital
BMI	Body mass index
BP	Blood pressure
CRP	C-reactive protein
CV	Cardiovascular
InAUC	Insulin area under the curve
HDL	high-density lipoprotein
HOMA-IR	homeostatic model assessment-insulin resistance
LDL	low-density lipoprotein
LF	Low fat
LGL	Low glycemic load
NIDDK	National Institute of Diabetes and Digestive and Kidney Diseases
NHLBI	National Heart, Lung and Blood Institute

TG	Triglyceride

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What is Known on This Subject

- Dietary improvement is recommended to treat cardiovascular (CV) risk factors during childhood.
- Low saturated fats were primarily recommended in the past; more recently a low glycemic load approach has been advised.
- The optimal dietary strategy for improving CV risk factors in youth is not known.

What This Study Adds

- Neither dietary strategy was superior; both produced significant weight loss and improvement in CV risk factors.
- Home delivery of food and nutrition education holds promise as an alternative to conventional approaches to nutrition research and for the treatment of high-risk youth.

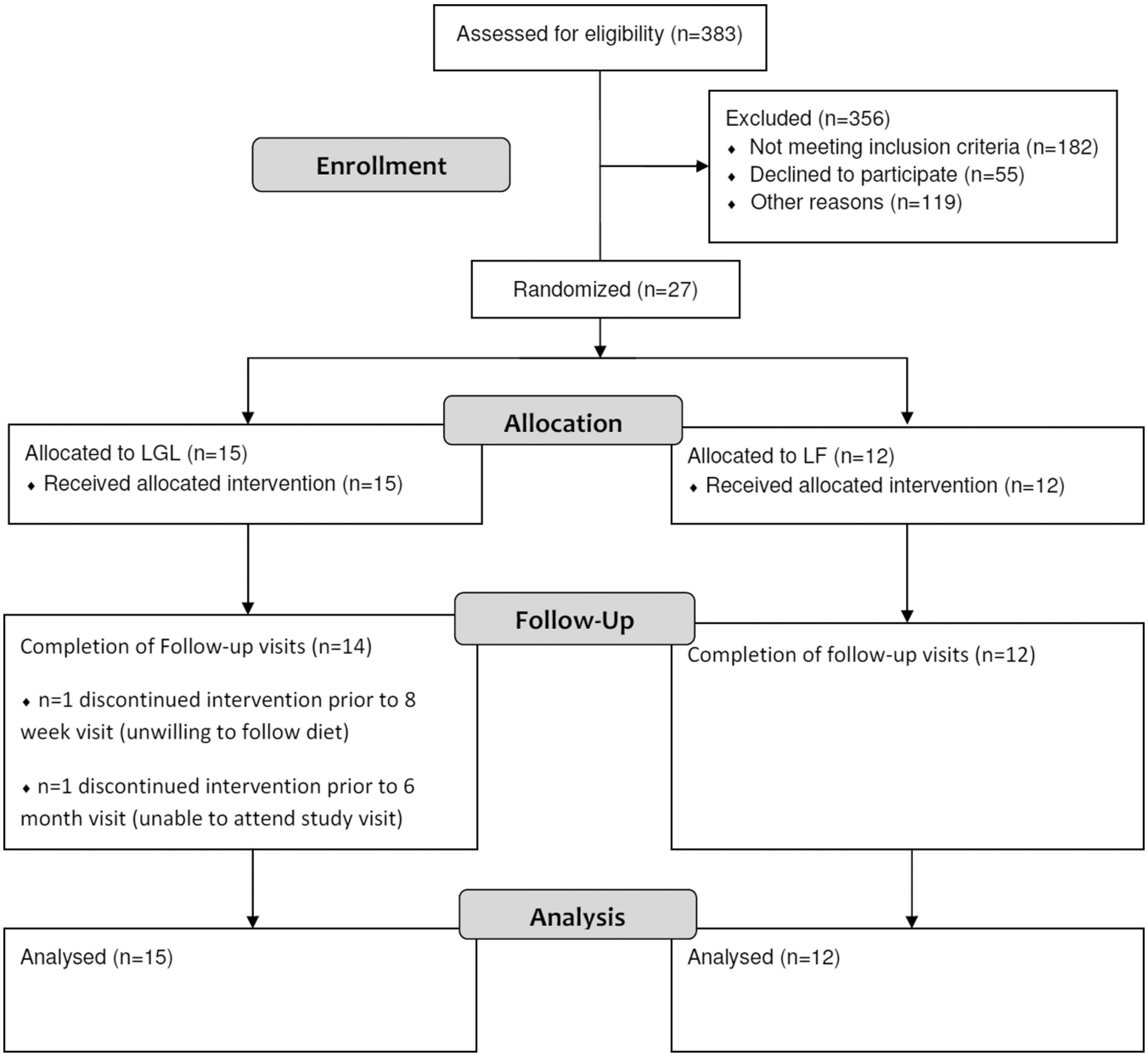

Figure 1.

Enrollment Flow Diagram showing the number of participants assessed for eligibility, enrolled, randomized, followed and analyzed.

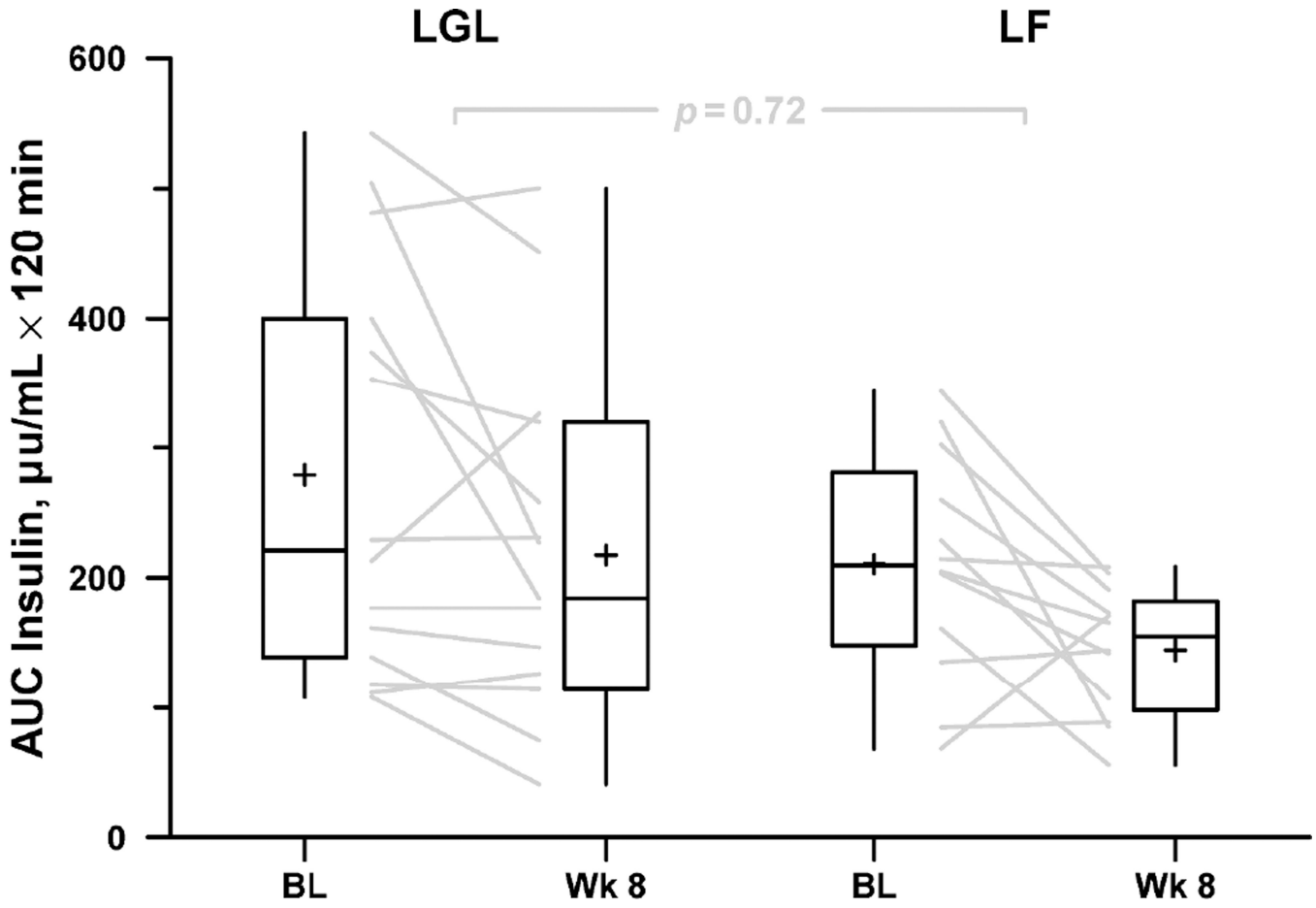

Figure 2.

Results of the InsAUC measured by oral glucose tolerance test over 120 minutes are shown by dietary assignment at baseline and at 8 weeks. There was no difference between groups in the change in InsAUC from baseline (p=0.72). InsAUC – Insulin area under the curve; BL – baseline; LGL – low glycemic load; LF – low fat.

Table 1

Baseline participant characteristics

Data are reported for all study participants as n (%), or mean (SD). Family history and sociodemographic characteristics were collected from the parent at the time of the baseline visit.

Participant Characteristics		(n=27)
Gender	Male	9 (33%)
Age (years)		13.2 (2.5)
Race/ethnicity	White	13 (48%)
	Black	5 (19%)
	Other/Multiracial	9 (33%)
	Hispanic	7 (26%)
High risk family history:		
Hypertension		6 (22%)
Lipid disorder		4 (15%)
Heart disease		13 (48%)
Obesity		8 (30%)
Type 2 Diabetes		7 (26%)
Income	Less than $40,000	3 (11%)
	$40,000 – $79,999	8 (30%)
	$80,000 or Above	9 (33%)
	Declined to Answer	7 (26%)
Highest Level of Education	High School or Below	3 (11%)
	Some College or Trade School	5 (19%)
	College Grad or Above	19 (70%)
Cardiovascular Risk Factors		
BMI		29.8 (3.9)
BMI z-score *		2.0 (0.3)
SBP (mm Hg)		105 (7.7)
Fasting insulin (μU/mL)		21 (9.2)
Fasting glucose (mg/dL)		88 (8.3)
TG (mg/dL)		131 (91.3)
HDL (mg/dL)		36.6 (5.9)

* BMI z score was calculated based CDC growth charts.([20])

BMI – body mass index.

Table 2

Laboratory Parameters

Results are shown at Baseline, 8 week and 6 month study visits shown as mean (SD) and change from baseline to 8 weeks. P-values reflect change from baseline and comparison between the groups.

		Mean ± SE			Change BL to 8 Weeks		
Parameter	**Dietary Assignment**	**Baseline**	**8 Weeks**	**6 Months**	**Mean ± SE**	**p-value[a]**	**p-value (diff)[b]**
Fasting Insulin (μU/mL)	All	21 ±1.8	14 ±1.6	18 ±2.0	−8 ±1.9	<.001	
	LGL	24 ±2.8	15 ±2.7	20 ±3.2	−9 ±3.1	0.017	0.582
	LF	18 ±1.5	12 ±1.4	16 ±1.6	−6 ±1.6	0.002	
Fasting Glucose (mg/dL)	All	89 ±1.6	86 ±2.1	89 ±1.4	−3 ±1.9	0.163	
	LGL	89 ±2.6	91 ±1.9	91 ±2.0	1 ±1.8	0.468	0.013
	LF	88 ±1.5	80 ±3.4	87 ±1.9	−8 ±3.2	0.030	
AUC Insulin (μU/mL * 120 mins)	All	248 ±26.0	185 ±21.3	-	−59 ±18.2	0.004	
	LGL	279 ±42.2	218 ±34.8	-	−52 ±26.7	0.072	0.720
	LF	211 ±25.4	145 ±14.5	-	−66 ±25.3	0.025	
AUC Glucose (mg/dL * 120 mins)	All	251 ±8.1	239 ±6.5	-	−12 ±9.5	0.225	
	LGL	245 ±11.0	242 ±8.7	-	−3 ±8.4	0.767	0.262
	LF	260 ±11.9	235 ±10.1	-	−24 ±19.2	0.232	
HOMA-IR (mg/dL*μU/mL)	All	4.7 ±0.4	3.0 ±0.4	4.2 ±0.5	−1.7 ±0.4	<.001	
	LGL	5.3 ±0.7	3.5 ±0.6	4.6 ±0.8	−1.8 ±0.7	0.023	0.778
	LF	3.9 ±0.3	2.4 ±0.3	3.5 ±0.4	−1.5 ±0.4	0.001	
Hemoglobin A1C (%)	All	5.6 ±0.1	5.5 ±0.1	5.7 ±0.1	−0.1 ±0.1	0.032	
	LGL	5.7 ±0.1	5.6 ±0.1	5.8 ±0.1	−0.1 ±0.1	0.104	0.922
	LF	5.5 ±0.1	5.4 ±0.1	5.5 ±0.1	−0.1 ±0.1	0.183	
Total Cholesterol (mg/dL)	All	158 ±6.9	148 ±7.3	155 ±7.2	−9.9 ±3.6	0.012	
	LGL	160 ±10.1	155 ±10.7	161 ±10.0	−4.5 ±4.8	0.360	0.102
	LF	155 ±9.4	138 ±9.4	148 ±10.5	−16.5 ±5.1	0.008	
HDL (mg/dL)	All	36.6 ±1.1	35.8 ±1.2	38.7 ±1.1	−0.8 ±1.0	0.419	
	LGL	36.7 ±1.3	38.2 ±1.3	39.1 ±1.4	1.5 ±1.1	0.168	0.005
	LF	36.6 ±2.0	32.9 ±1.8	38.3 ±2.0	−3.7 ±1.4	0.020	
LDL (mg/dL) *	All	92 ±5.2	85 ±5.8	88 ±5.5	−7.5 ±0.3	0.023	

Parameter	Dietary Assignment	Mean ± SE			Change BL to 8 Weeks		
		Baseline	8 Weeks	6 Months	Mean ± SE	p-value[a]	p-value (diff)[b]
Triglycerides (mg/dL) *	LGL	94 ±8.0	90 ±9.9	95 ±7.9	−3.4 ±0.2	0.446	0.131
	LF	91 ±6.8	79 ±6.1	80 ±7.4	−12.2 ±0.6	0.013	
	All	107 ±13.9	93 ±10.2	104 ±11.0	−13.8 ±1.1	0.091	
VLDL (mg/dL) *	LGL	104 ±17.4	89 ±11.7	99 ±14.1	−14.4 ±1.4	0.131	0.880
	LF	112 ±24.8	99 ±19.5	111 ±19.0	−13.1 ±2.0	0.389	
	All	20 ±2.4	19 ±2.1	21 ±2.2	−2.5 ±0.2	0.124	
Non-HDL Cholesterol (mg/dL)	LGL	21 ±3.6	18 ±2.4	20 ±2.8	−2.8 ±0.3	0.151	0.817
	LF	20 ±3.6	20 ±3.9	22 ±3.8	−2.0 ±0.3	0.481	
	All	121 ±6.9	112 ±7.3	116 ±7.1	−9.1 ±3.2	0.010	
Total Cholesterol/HDL Ratio	LGL	123 ±10.2	117 ±10.8	122 ±9.9	−6.1 ±4.5	0.200	0.312
	LF	118 ±9.2	105 ±9.6	109 ±10.1	−12.8 ±4.6	0.018	
	All	4.4 ±0.2	4.2 ±0.2	4.1 ±0.2	−0.2 ±0.1	0.171	
Triglyceride/HDL Ratio	LGL	4.4 ±0.3	4.1 ±0.3	4.2 ±0.3	−0.3 ±0.2	0.076	0.214
	LF	4.3 ±0.3	4.3 ±0.4	3.9 ±0.3	0.0 ±0.2	1.000	
	All	3.8 ±0.6	3.2 ±0.4	3.2 ±0.4	−0.6 ±0.3	0.116	
ALT (U/L)	LGL	3.5 ±0.6	2.7 ±0.4	3.0 ±0.5	−0.8 ±0.4	0.058	0.374
	LF	4.1 ±1.0	3.9 ±0.9	3.5 ±0.7	−0.2 ±0.6	0.728	
	All	27 ±5.5	23 ±3.8	23 ±4.4	−3.7 ±2.3	0.116	
Fibrinogen (mg/dL)	LGL	34 ±9.6	27 ±6.6	28 ±7.7	−6.1 ±3.9	0.146	0.267
	LF	18 ±1.6	17 ±2.1	17 ±1.9	−0.8 ±1.4	0.576	
	All	327 ±12.5	321 ±12.8	316 ±12.8	−12.6 ±9.1	0.178	
CRP (mg/dL)	LGL	328 ±20.2	332 ±16.3	332 ±19.1	−6.3 ±9.8	0.533	0.483
	LF	326 ±14.3	308 ±20.3	293 ±12.1	−19.5 ±15.9	0.249	
	All	0.3 ±0.1	0.2 ±0.1	0.2 ±0.1	−0.1 ±0.1	0.040	
	LGL	0.4 ±0.2	0.2 ±0.1	0.2 ±0.1	−0.2 ±0.1	0.079	0.271
	LF	0.2 ±0.1	0.1 ±0.0	0.1 ±0.1	−0.1 ±0.0	0.280	

*Log-transformed for analysis, re-transformed to standard units.

[a]Testing for zero mean change within diet group.

[b] Testing for equal mean change between diets.

ALT – alanine aminotransferase, AUC – area under the curve, CRP – C-reactive protein, HDL – high-density lipoprotein, HOMA-IR – homeostatic model assessment-insulin resistance, LDL – low-density lipoprotein.

Table 3

Anthropometrics and Blood Pressure

Data are presented as mean ± standard error (SE). Baseline, 8 week and 6 month study visits shown as mean (SD) and change from baseline to 8 weeks. P-values reflect change from baseline and comparison between the groups.

		Mean ± SE			Change BL to 8 Weeks		
Anthropometric Parameter	**Dietary Assignment**	**Baseline**	**8 Weeks**	**6 Months**	**Mean ± SE**	**p-value[a]**	**p-value (diff)[b]**
BMI[c]	All	29.8 ±0.74	28.4 ±0.79	29.0 ±0.77	−1.4 ±0.21	<.001	
	LGL	30.7 ±1.22	29.3 ±1.30	30.0 ±1.27	−1.4 ±0.32	<.001	0.972
	LF	28.7 ±0.61	27.4 ±0.65	27.8 ±0.59	−1.4 ±0.29	<.001	
BMI z-score[c]	All	2.05 ±0.05	1.89 ±0.06	1.92 ±0.05	−0.16 ±0.03	<.001	
	LGL	2.09 ±0.06	1.93 ±0.09	1.98 ±0.08	−0.16 ±0.04	0.003	0.976
	LF	1.99 ±0.09	1.83 ±0.08	1.84 ±0.06	−0.16 ±0.03	<.001	
Weight (kg)	All	79.3 ±3.5	76.6 ±3.6	79.0 ±3.5	−2.7 ±0.5	<.001	
	LGL	82.0 ±4.9	79.2 ±5.1	81.7 ±5.1	−2.8 ±0.7	0.002	0.873
	LF	76.0 ±5.0	73.3 ±5.1	75.5 ±4.8	−2.6 ±0.7	0.002	
Waist Circumference (cm)	All	99.8 ±1.8	96.7 ±1.9	97.2 ±1.8	−3.1 ±0.8	<.001	
	LGL	100.6 ±2.7	97.3 ±2.9	98.1 ±2.9	−3.4 ±0.9	0.002	0.721
	LF	98.7 ±2.3	96.0 ±2.3	96.1 ±2.1	−2.8 ±1.5	0.084	
Percent Body Fat	All	35.3 ±0.6	34.2 ±0.6	33.5 ±0.7	−1.1 ±0.4	0.004	
	LGL	36.3 ±0.9	35.5 ±0.9	34.7 ±1.0	−0.9 ±0.5	0.071	0.461
	LF	34.2 ±0.8	32.7 ±0.7	32.2 ±0.8	−1.4 ±0.6	0.030	
Systolic BP (mm Hg)	All	105 ±1.5	101 ±1.4	102 ±1.3	−5 ±1.4	0.002	
	LGL	106 ±2.0	102 ±1.6	102 ±1.5	−4 ±1.8	0.052	0.443
	LF	105 ±2.3	99 ±2.3	103 ±2.4	−6 ±2.1	0.017	
Diastolic BP (mm Hg)	All	69 ±1.4	67 ±1.1	67 ±1.2	−2 ±1.3	0.147	
	LGL	70 ±2.2	66 ±1.7	67 ±1.2	−4 ±1.6	0.028	0.078
	LF	67 ±1.3	68 ±1.3	67 ±2.3	1 ±1.9	0.756	

[a] Testing for zero mean change within diet group.

[b] Testing for equal mean change between diets.

[c] BMI and z scores were calculated based on CDC growth charts.([20])

BMI – body mass index, BP – blood pressure