Lipids (2013) 48:1197–1205
DOI 10.1007/s11745-013-3834-y

ORIGINAL ARTICLE

The Effect of an Energy Restricted Low Glycemic Index Diet on Blood Lipids, Apolipoproteins and Lipoprotein (a) Among Adolescent Girls with Excess Weight: a Randomized Clinical Trial

Mohammad Hossein Rouhani · Roya Kelishadi · Mahin Hashemipour · Ahmad Esmaillzadeh · Leila Azadbakht

Received: 8 April 2013 / Accepted: 15 August 2013 / Published online: 3 September 2013

Abstract Some studies focused on the effect of the dietary glycemic index on lipoproteins and apolipoproteins in adults; however, little evidence exists among adolescents regarding the effect of a low glycemic index (LGI) diet on apolipoproteins and lipoprotein (a) (Lpa). This study was conducted to evaluate the effect of an LGI diet on the lipid profile, apolipoproteins and Lpa among overweight and obese adolescent girls. For this parallel designed randomized clinical trial, 50 healthy overweight/obese girls at pubertal ages were randomly allocated to an LGI or a healthy nutritional recommendations (HNR) based diet. Equal macronutrient distributed diets were prescribed to both groups. Biochemical measurements included lipid profile, apolipoprotein A, apolipoprotein B and Lpa were conducted before and after 10 weeks of intervention. Forty one adolescent girls completed the study. The dietary glycemic index in the LGI group was 42.67 ± 0.067. There were no differences in the mean of blood lipid indices baseline and after intervention between two groups. There were no significant differences between the two groups regarding lipid profiles, apolipoproteins and Lpa. There were no significant differences in lipid profiles, apolipoproteins and Lpa between the LGI diet and the HNR-based diet and the impact of these two diets on lipid profile was equal in this trial. Trial registry code: IRCT201109272839N4.

Keywords Carbohydrate quality · Hyperlipidemia · Obesity

Abbreviations

GI	Glycemic index
HDL	High density lipoprotein(s)
HGI	High glycemic index
HNR	Healthy nutritional recommendations
LDL	Low density lipoprotein(s)
LGI	Low glycemic index
Lpa	Lipoprotein (a)
TAG	Triacylglycerol(s)

Introduction

Cardiovascular diseases (CVD) are known to be a major cause of mortality, worldwide [1]. According to the results from the Framingham and INTERHEART studies, dyslipidemia is a CVD risk factor [2]. There is an upward trend for prevalence of dyslipidemia among Americans [3], Chinese [4] and Iranian populations [5]. The incidence of dyslipidemia among obese children is high [6]. Treatment and prevention programs for dyslipidemia will be more effective if they were exerted before adulthood [7]. Dietary intervention could play an important role in this program [8]. Carbohydrate is a principal component of our diet. A

M. H. Rouhani · A. Esmaillzadeh · L. Azadbakht (✉)
Food Security Research Center and Department of Community Nutrition, School of Nutrition and Food Science, Isfahan University of Medical Sciences, PO Box 81745, Isfahan, Iran
e-mail: azadbakht@hlth.mui.ac.ir

R. Kelishadi · M. Hashemipour
Department of Pediatrics, Child Growth and Development Research Center, and School of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran

M. Hashemipour
Department of Pediatric Endocrinology and Metabolism Diseases, Endocrinology and Metabolism Research Center, School of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran

number of dietary intervention studies focused on the glycemic index (GI) as a carbohydrate quality indicator. Two to four hours after ingesting a high GI food, the free fatty level will be increased [9]. So, several studies have examined the effect of dietary GI in relation to dyslipidemia among different groups of adults [10, 11]; there is limited evidence for adolescents. One previously conducted study reported a significant decrease in triacylglycerols (TAG) and total cholesterol following a low glycemic index (LGI)-high carbohydrate modified-lipid diet consumption in children [12]. However, another study showed that high carbohydrate diets had adverse effects on the lipid profile among adults [13]. The effect of an LGI diet on the lipid profile among children was examined in previous studies [14–16]. Although the sample size, dietary intervention process and design of these studies were appropriate, in some cases apolipoprotein levels were not evaluated. Apolipoproteins contribute to regulating the plasma cholesterol level and they are also known as a predictor of CVD [17]. It should be kept in mind that previous studies showed that there is a strong association between dyslipidemia and obesity among children [18, 19]. There is little evidence regarding the effect of an LGI diet on lipid profiles, especially apolipoproteins and lipoprotein (a) (Lpa) among children and adolescents. So, the present paper reports the results of a parallel RCT (randomized controlled trial) designed to examine the effects of an LGI diet on the lipid profile, apolipoproteins and Lpa among healthy obese girls in pubertal ages in comparison to the healthy nutritional recommendations (HNR) based diet.

Subjects

For this parallel designed RCT, potential participants were chosen from Isfahanian student health booklets. Then the students' parents were invited to an information visit and the study was explained to them. Informed written consent forms were signed by volunteer adolescents and one of their parents. Moreover, we used flyer advertisements to recruit more participants. This study was conducted in Isfahan, Iran in 2011. Inclusion criteria were: being female, <18 years old, overweight or obese, menstruating and not using medications. Low adherence to recommendations or using lipid profile-related medications were considered to be exclusion criteria. For defining overweight and obesity, the table of the body mass index (BMI) for age released by the WHO was used [20]. BMI values between 85th and 95th percentiles were considered as being overweight and those with a BMI greater than the 95th percentile were defined as obese. Age, menstruation status and medication history were assessed by oral questions. Fifty adolescents were enrolled to the study (Fig. 1). The study was approved by the Research Council and Ethical Committee of School of Nutrition and Food Science, Isfahan University of Medical Science, Isfahan, Iran and Food Security Research Center, Isfahan University of Medical Science, Isfahan, Iran. This RCT was registered in the Iranian Registry of Clinical Trials (IRCT201109272839N4).

Study Procedure

Adolescents were randomized to either an LGI ($n = 25$) or an HNR-based diet ($n = 25$) for a 10-week period. In our previous study, we calculated the healthy eating index (HEI) score for 819 Tehranian adults [21]. After stratifying scores to three categories (poor diet, needs improvement and good diet), results showed that only 23 % of the study population consumed a good diet as defined by the HEI. This finding demonstrated that unhealthy diet is prevalent among our study population. The HNR-based diet was substituted for their usual diet. Because of the dietary intervention, volunteers were not blinded. All assistants in the biochemical laboratory were blinded. The intervention groups were matched for gender, pubertal status and age.

Individual counseling visits were established every 18 days. All volunteers were free-living. Dietary interventions were comprehensively explained to the adolescents and their parents. All the meals were provided by parents.

Dietary Intervention

We calculated the total energy needs individually, based on the formula suggested by the US Institutes of Medicine [22]. Because the effect of LGI or HNR diets on obesity was not confirmed previously, a moderate calorie subtraction (200 kcal) was exerted for obese adolescents to provide a benefit to these volunteers. Similar macronutrient composition (53–56 % en carbohydrate, 16–18 % en protein, and 27–30 % en fat) was prescribed for both groups. We defined LGI as GI <50. In the intervention group, adolescents were directed to select carbohydrate containing foods included fruits, vegetables, dairy and grains from a list of LGI foods. Furthermore, they were advised to curtail the intake of high glycemic index (HGI) foods (GI > 50). Regarding food choices of meat and fat, adolescents were given a standard food exchange list. Individual sessions were established for teaching the prescribed diet. The participants in the HNR group were given food lists emphasizing the limitation of unhealthy food such as fast foods, French fries, fried foods, industrial beverages and unhealthy fats, they were advised to drink 1.5–2 l of water, consume a large amount of fruit and vegetables of different

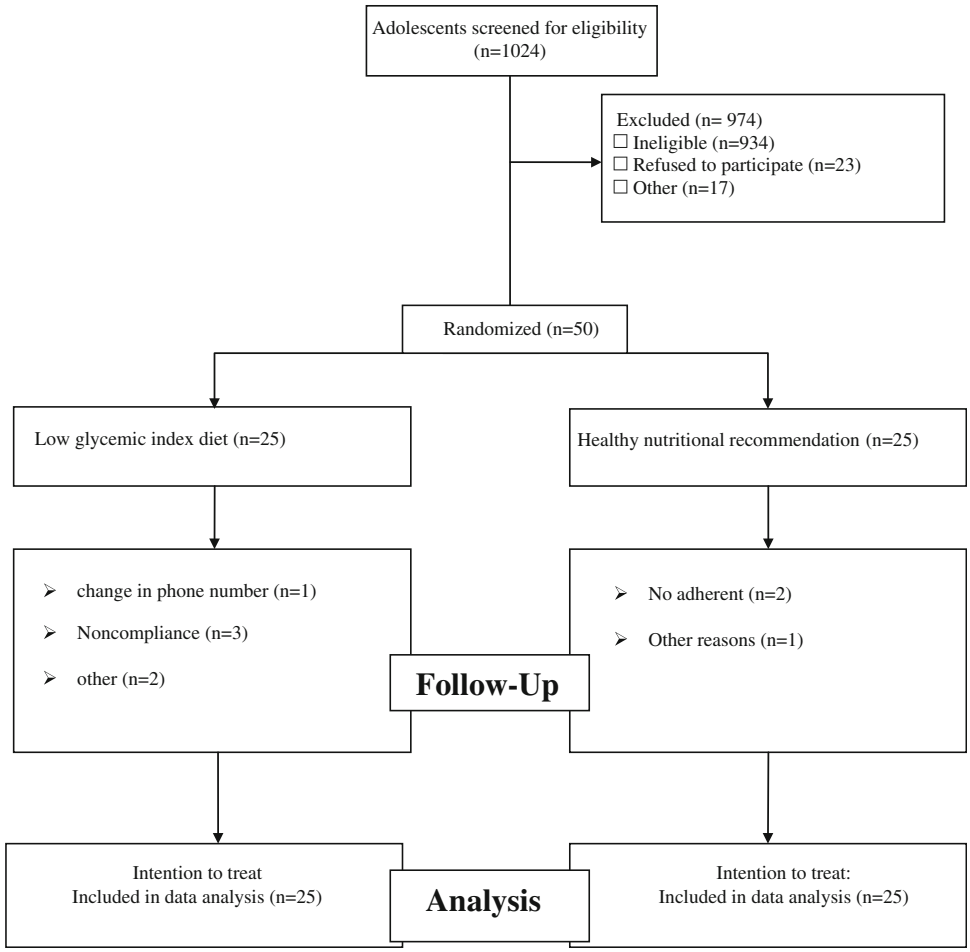

Fig. 1 Study procedure and volunteers' randomization

varieties, low fat dairy and whole grains. We provided a complete food exchange list for selecting the foods in the HNR group. Volunteers completed a 1-day weighed dietary record and a 1-day physical activity record in weeks 2.5, 5, 7.5 and 10 of the study. The participants completed a 4-day food record and a 4-day physical activity record to include 1 weekend and 3 week days. In individual sessions, records were evaluated and clarified. At each visit, one dietitian answered participants' queries. A range of 1.35–2.39 × basal metabolic rate ratio was defined as acceptable reported energy intake [23]. For evaluating compliance in the HNR group, reported quantity (% en) of carbohydrate, protein and fat in food records was compared with the prescribed diet. This comparison demonstrated that good compliance with the prescribed diets and food records (P values for the difference between macronutrients in prescribed diet and the amount of consumed macronutrients in the diet resulted from the food records: $P = 0.202$ for carbohydrate, $P = 0.148$ for protein and $P = 0.172$ for fat). So, according to the results, the non-intervention group had a good compliance because there were no significant differences between macronutrients intake resulted from the analysis of what subjects consumed and the prescribed amounts.

Compliance in the intervention group was defined by GI less than 50.

Published native GI tables were used for GI values [24]. GI values not included in native tables were extracted from International Tables [25]. The GI of mixed foods was calculated based on their carbohydrate-containing components. Other GI values were estimated from similar foods. The mean GI of diet was computed by the following formula [11]:

$$\mathrm{GI}_{\mathrm{mean}} = \sum \left(\left(C_{\mathrm{food}} / C_{\mathrm{total}} \right) \times \ \mathrm{GI}_{\mathrm{food}} \right)$$

where mean GI, gram of carbohydrate in food, gram of total carbohydrate in diet and GI of food were presented by $\mathrm{GI}_{\mathrm{mean}}$, C_{food}, C_{total} and $\mathrm{GI}_{\mathrm{food}}$, respectively. Food record analysis showed that the mean ± SE of GI in the intervention group was 43.22 ± 0.54. So, it shows that the intervention group complied with their diet as their calculated dietary GI was lower than the prescribed dietary GI.

Biochemical Measurements

For assessing biochemical variables, we drew one blood sample after >12 h overnight fasting from each participant at baseline and at the end of week 10. For separating serum, samples were centrifuged at 3,000×*g* for 10 min after coagulating. Enzymatic colorimetric tests were used to measure the level of the TAG and total cholesterol. The high density lipoprotein (HDL) concentration was assessed by photometric methods in which LDL, very low density lipoprotein (VLDL) and chylomicrons were blocked by antibodies and finally the HDL concentration was evaluated by enzymatic measurement. The LDL level was measured after blocking HDL, VLDL and chylomicrons by enzymatic colorimetric tests. The immunoturbidimetry method, in which stimulated antibodies banded to a specific marker, was used to assess the concentration of the Apo B, Apo A1 and Lpa. Inter- and intra-assay coefficients were less than 5 % for all assessments. All biochemical kits were provided by Pars Azmoon, Iran.

Statistical Analysis

For estimating the sample size, we used a parallel study sample size formula [26] $N = 2[(Z_{1-\alpha/2} + Z_{1-\beta})^2 \times S^2]/d^2$ where type one (α) and type two error (β) were 0.05 and 0.20 (power = 80 %), respectively. Based on a previous study, the variance of low density lipoprotein (LDL) was 1.2 [15]. We also considered 1.2 as the difference in the mean (d) of LDL. The formula showed that the current study needed 16 subjects in each group for 80 % of the power of the study. The normal distribution of the variables was tested by using a histogram curve and a Kolmogorov–Smirnov test. Based on the results, the distribution of Lpa was not normal. So, we reported geometric means for this variable. We also used the Student t test for comparing nutrient intake, baseline and endpoint values as well as the percentage changes in groups. Percentage changes were calculated using the following formula: $[(E - B)/B] \times 100$ in which end values and baseline values were shown by E and B, respectively. For evaluating the differences between end and baseline values within each group, we used the paired t test. In the adjusted model in which the effect of age was satisfied, analysis of the covariance (ANCOVA) was performed. We reported all values as means ± SE. P_{time}, P_{group} and $P_{time \times group}$ were calculated for all variables. Time × age interaction was also calculated because age was significantly different between the two groups. Intention to treat analysis was performed. The statistical significant level was defined as $P < 0.05$. SPSS for Windows software (SPSS Inc., Chicago, IL) version 10 was used for statistical analysis.

Results

Forty-one participants (19 in LGI and 22 in HNR) of 50 enrolled subjects completed the study (Fig. 1). In the LGI group, six subjects withdrew from the study because of poor compliance, missing phone number and other reasons. In the HNR group, three adolescents withdrew due to poor compliance and other reasons. An intention to treat analysis was used and data of all 50 subjects were analyzed. The mean of

Table 1 Daily nutrient intake of participants on a low glycemic index diet and a healthy nutrition recommendations based diet on the food diaries

Variables	Low glycemic index group[a] ($n = 25$)	Healthy nutrition recommendations group[b] ($n = 25$)	P value[c]
Energy (kcal)	1,503.09 ± 48.39[d]	1,532.66 ± 61.57	0.707
Carbohydrate (g)	192.06 ± 8.42	194.98 ± 8.29	0.806
Protein (g)	73.85 ± 3.09	69.15 ± 3.84	0.347
Fat (g)	51.57 ± 3.8	55.93 ± 5.2	0.505
Fiber (g)	18.51 ± 1.76	18.44 ± 1.18	0.975
Saturated fatty acids (g)	13.78 ± 1.50	14.73 ± 1.55	0.662
Polyunsaturated fatty acid (g)	14.33 ± 1.17	16.93 ± 2.32	0.325
Monounsaturated fatty acid (g)	13.99 ± 1.31	15.11 ± 1.57	0.588
Cholesterol (mg)	206.03 ± 29.45	223.92 ± 37.01	0.707

[a] Low glycemic index diet defined as glycemic index less than 50

[b] Healthy nutrition recommendations emphasized on limiting foods with high content of fats, fast foods, French fries, fried foods, industrial beverages and unhealthy fats, drinking 1.5–2 l of water, consuming the large amount of fruits and vegetable with different varieties, low fat dairy and whole grains

[c] P values were computed by independent t test

[d] Values are presented as means ± SE

the age in the HNR group was significantly greater than the LGI group (13.18 ± 0.21 vs. 13.98 ± 0.27 years; $P = 0.031$). According to the analysis of the food diaries (Table 1), dietary intakes included total energy ($P = 0.707$), carbohydrate ($P = 0.806$), protein ($P = 0.347$), fat ($P = 0.505$), fiber ($P = 0.975$), saturated fatty acids ($P = 0.662$), polyunsaturated fatty acid ($P = 0.325$), monounsaturated fatty acid ($P = 0.588$) and cholesterol ($P = 0.707$) were not significant different between treatment groups. The dietary GI in the LGI and HNR groups were 43.22 ± 0.54 and 46.70 ± 1.03, respectively ($P = 0.005$). The analysis of the physical activity records showed no differences between the two groups (LGI group: 1.10 ± 0.01 MET h/day vs. HNR group: 1.12 ± 0.02 MET h/day; $P = 0.429$).

The mean values of the lipid profile components, apolipoproteins and Lpa at baseline and after 10 weeks in LGI and HNR groups are illustrated in Table 2. The comparison between the two groups showed no differences in baseline values. After intervention, lipid values were not significantly different between treatment groups. Moreover, within-group comparison of baseline and after intervention values for each variable showed that in the HNR group, Apo A1 was significantly decreased after intervention (146.3 ± 5.8 vs. 127.7 ± 5.0 mg/dl; $P = 0.043$). Further, end point values of ApoB/ApoA were increased in both groups compared to baseline values ($P = 0.039$ for the LGI group and $P = 0.011$ for the HNR group). Other variables had no significant differences. Group and time × group are not significantly different for all variables. Although time and time × age was statistically significant for HDL ($P = 0.012$ and $P = 0.010$, respectively), other values were not significantly different. The concept of P_{time} is whether or not time is responsible for observed differences. The concept of P_{group} is whether or not grouping is responsible for observed differences. The concept of $P_{time \times group}$ is whether or not time × group interaction is responsible for observed differences. The concept of $P_{time \times age}$ is whether or not time × age interaction is responsible for observed differences. Within- and between-groups analysis did not show any significant difference between baseline and end point values of HDL. So, the values of P_{time}, P_{group}, $P_{time \times group}$, $P_{time \times age}$ were not statistically important.

The comparison of the percentage changes of variables between two groups is presented in Table 3. Percentage changes in lipid profile components, apolipoproteins and Lpa between LGI diet and HNR were not significantly different between treatment groups.

Discussion

The main finding of the current study was a lack of differences in changes of lipid profiles, apolipoproteins and Lpa between the LGI diet and the HNR-based diet. The effect of the LGI diet on the lipid profile was evaluated in previous studies of adults [27–30], but little evidence exists for adolescents. To our knowledge, this is the first comparison between an LGI diet and HNR among female adolescents in which lipid profiles, apolipoproteins and Lpa were evaluated.

A comparison between an LGI (GI = 60) diet and an HGI (GI = 90) diet demonstrated that the LGI diet provided more favorable changes in TAG than the HGI diet ($P < 0.05$ for both) over a long-term period among 22 obese girls and boys [16]. Another study examined the effect of an LGI diet on the lipid profile in eight healthy children [14]. The results of this before–after designed study demonstrated a slight reduction in TAG and an increase in HDL, but changes in apolipoproteins were not assessed. There are several differences between our study and these reported studies such as gender, LGI definition, study duration and comparison diet. We used an HNR-based diet for the non-LGI group while an HGI diet was used by Parillo et al. [16]. An HNR-based diet typically is prescribed for overweight and obese adolescents. Therefore, comparing the effects of an LGI diet vs. an HNR-based diet may be more appropriate. Therefore, differences in the study design may explain the differences in findings.

Within-group analysis of our study confirmed the results of a previous study conducted on 26 obese children (aged 7–13) [14]. Although changes between groups were not compared, within-group analysis showed that baseline values of cholesterol, HDL and TAG had no significant changes after prescribing an LGI diet [14].

Although some investigators reported the effect of GI on apolipoproteins in adults [8, 31], data for children and adolescent is lacking. Studies [10, 31] were conducted on diabetic men, so their results are not comparable with the present study. Apo B to Apo A ratio reflects the ratio of atherogenic to atheroprotective cholesterol containing lipoproteins [32]. It also has a direct relationship with CVD [33]. A 3-year follow up of the trend of Apo A1 changes among adolescent girls showed that the lowest concentration of Apo A1 had been observed at 13 years old [34]. Previous study reported an inverse association between Apo A1 concentration and BMI among Chinese vegetarian and omnivore adults [35]. Therefore, it seems that the observed reduction in Apo A1 among our subjects may be due to obesity and age-induced hormonal changes. Apo A1 reduction was observed in both groups in the current study, but it was not significant in the LGI group.

We could not observe any statistical difference in within-group and between-group analysis for non-HDL cholesterol and the total to HDL cholesterol ratio. Non-HDL cholesterol which is calculated by total cholesterol minus HDL cholesterol is a better CVD predictor than LDL

Table 2 Lipid profiles, apolipoproteins and lipoprotein (a) values among low glycemic index diet and healthy nutrition recommendations at baseline and after 10 weeks of study

Variables	Low glycemic index group[a] ($n = 25$)	Healthy nutrition recommendations group[b] ($n = 25$)	P overall[c]	P_{time}[d]	P_{group}[e]	$P_{time \times group}$[f]	$P_{time \times age}$[g]
TAG (mg/dl)							
Before	109.97 ± 8.31[i]	114.14 ± 12.53	0.412	0.731	0.716	0.310	0.675
After	112.37 ± 8.66	114.74 ± 13.48	0.883				
P[h]	0.227	0.988	–				
TC (mg/dl)							
Before	162.07 ± 5.74	169.19 ± 4.51	0.334	0.927	0.622	0.433	0.944
After	166.07 ± 4.23	166.89 ± 5.56	0.907				
P[h]	0.460	0.645	–				
HDL (mg/dl)							
Before	44.02 ± 1.65	46.21 ± 1.51	0.336	0.012	0.326	0.368	0.010
After	43.48 ± 1.36	44.86 ± 1.29	0.467				
P[h]	0.802	0.366	–				
LDL (mg/dl)							
Before	92.60 ± 4.00	96.42 ± 3.59	0.481	0.976	0.861	0.358	0.952
After	93.55 ± 3.09	93.37 ± 3.88	0.972				
P[h]	0.678	0.290	–				
Apo A1 (mg/dl)							
Before	137.42 ± 5.14	145.79 ± 5.12	0.255	0.704	0.293	0.322	0.829
After	129.43 ± 3.15	127.83 ± 4.39	0.769				
P	0.460	0.043	–				
Apo B (mg/dl)							
Before	101.99 ± 5.41	109.31 ± 6.32	0.383	0.370	0.210	0.481	0.359
After	105.12 ± 4.06	108.28 ± 4.09	0.587				
P[h]	0.616	0.887	–				
Lpa (mg/dl)							
Before	20.39 ± 2.94	24.91 ± 3.74	0.348	0.226	0.122	0.609	0.220
After	21.04 ± 2.68	25.04 ± 2.48	0.280				
P[h]	0.310	0.340	–				
TAG/HDL							
Before	2.48 ± 0.26	2.66 ± 0.38	0.689	0.233	0.966	0.304	0.203
After	2.77 ± 0.29	2.75 ± 0.38	0.960				
P[h]	0.366	0.774	–				
ApoB/ApoA							
Before	0.73 ± 0.01	0.74 ± 0.02	0.761	0.372	0.302	0.724	0.240
After	0.81 ± 0.02	0.86 ± 0.03	0.269				
P[h]	0.039	0.011	–				
TC/HDL							
Before	3.78 ± 0.18	3.76 ± 0.17	0.937	0.035	0.658	0.289	0.030
After	3.89 ± 0.15	3.81 ± 0.18	0.715				
P[h]	0.514	0.685					
Non-HDL cholesterol (mg/dl)							
Before	118.04 ± 5.70	122.98 ± 5.01	0.519	0.242	0.853	0.075	0.559
After	122.59 ± 4.22	122.03 ± 5.93	0.939				

Table 2 continued

Variables	Low glycemic index group[a] ($n = 25$)	Healthy nutrition recommendations group[b] ($n = 25$)	P overall[c]	P_{time}[d]	P_{group}[e]	$P_{time \times group}$[f]	$P_{time \times age}$[g]
P[h]	0.384	0.841					

TAG triacylglycerol, *TC* total cholesterol, *HDL* high density lipoprotein, *LDL* low density lipoprotein, *Apo A1* apolipoprotein A1, *Apo B* apolipoprotein B, *Lp (a)* lipoprotein (a)

[a] Low glycemic index diet defined as having a glycemic index of less than 50

[b] Healthy nutrition recommendations emphasized on limiting foods with high contents of fats, fast foods, French fries, fried foods, industrial beverages and unhealthy fats, drinking 1.5–2 l of water, consuming a large amount of fruits and vegetable with different varieties, low fat dairy foods and whole grains

[c] P values present a comparison baseline and end point values between two groups (computed by independent samples t test)

[d] P values demonstrate the effect of time (computed by analysis of the covariance)

[e] P values represent the effect of grouping (computed by analysis of the covariance)

[f] P values represent the time × group interaction (computed by analysis of the covariance)

[g] P values represent the time × age interaction (computed by analysis of the covariance)

[h] P values present comparison baseline and end point values within each group (computed by paired sample t test)

[i] All values are means ± SE except for lipoprotein (a) that is the geometric mean ± SE

cholesterol [36]. The total to HDL cholesterol ratio is considered to be a stronger predictor of coronary artery disease than total or lipoprotein cholesterol concentrations [37]. Therefore, an elevated level of either non-HDL cholesterol or total to HDL cholesterol ratio is related to higher risk of CVD. These ratios are strongly influenced by hormones, especially in females [38]. Estrogen, the main sex hormone in the female, has an increasing effect on HDL [38]. Researchers should focus on the link between biochemistry and physiological pattern of the body, especially among pubertal females.

The difference in GI between the LGI group and the HNR group may be not physiologically valuable. Indeed, the subjects in the HNR group did not consume an HGI diet. So, the results did not show any differences between the two groups. As shown in the Iranian native GI table [24], Iranian staple foods such as white rice and most kinds of white bread are not categorized as HGI foods. These staple foods were responsible for attenuating the physiological difference in GI values between groups.

One possible reason for a lack of change in the blood lipids in both the LGI diet group and the HNR-based diet group is that the adolescents' blood lipids were in the normal range at baseline. These outcomes may have been different if the subjects' lipid homeostases were disturbed.

Some physiological mechanisms supported the reduction in the GI of diet; insulin secretion is rapidly increased following an HGI diet [9]. After 4–6 h, hypoglycemia occurs and counterregulatory hormones secretion increases [9]. Counterregulatory hormones stimulate free fatty acid releasing from adipose tissue [9]. Long-term HGI diet consumption may lead to insulin resistance [39]. An increased free fatty acid concentration resulted from insulin resistance and increases in counterregulatory hormones, stimulates very low density lipoprotein, an LDL precursor, production [27]. Insulin resistance also decreases HDL concentration [40].

Limitations

A major limitation of present study is the absence of an HGI group for comparing the HGI and LGI diets. Nonetheless, the effect of a LGI diet on blood lipids was not examined in comparison to the HNR-based diet in previous studies. This study was conducted on overweight or obese adolescents who had normal levels of lipid profiles. The same intervention should ideally be repeated with subjects with elevated lipid profiles in future studies. As reports show some unfavorable dietary behaviors among the Iranian population [41, 42], conducting interventional dietary research to clarify suitable diet is necessary.

Several points should be considered as strengths of the current study such as a statistically adequate sample of one specific sex over a narrow age range, equal macro- and micro-nutrient distribution between two groups, an adequate study duration, comparison of an LGI diet and an HNR-based diet, and comprehensive lipid profiles from several biochemical measurements.

In summary, the results of our study showed that an LGI diet had no significant effect on blood lipids compared to an HNR-based diet and that the impact of these two diets on lipid profiles was equal in this trial. In practice, dietitians can obtain the benefits of an LGI diet on blood lipids by prescribing an HNR-based diet. Future studies should focus on the influence of GI and HNR on the lipid profiles of adolescents with dyslipidemia.

Table 3 Percentage changes of lipid profiles, apolipoproteins and lipoprotein (a) for the low glycemic index diet and for the healthy nutrition recommendations at baseline and after 10 weeks of study

Variables	Low glycemic index group[a] ($n = 25$)	Healthy nutrition recommendations group[b] ($n = 25$)	P[c]
TAG	25.10 ± 13.02[d]	7.36 ± 9.26	0.273
TC	4.77 ± 3.40	−0.30 ± 3.19	0.282
HDL	0.82 ± 3.06	−1.60 ± 2.70	0.555
LDL	3.47 ± 3.42	−2.07 ± 2.92	0.225
Apo A	−1.36 ± 4.34	−8.85 ± 4.56	0.241
Apo B	9.79 ± 5.45	6.93 ± 6.49	0.738
Lpa	62.79 ± 30.35	94.98 ± 41.47	0.689
Apo B/Apo A	12.51 ± 3.54	18.49 ± 5.23	0.349
TAG/HDL	29.06 ± 13.96	12.03 ± 9.88	0.325
TC/HDL	6.40 ± 5.54	2.09 ± 3.03	0.499
Non-HDL cholesterol	7.45 ± 5.04	0.55 ± 4.12	0.295

TAG triacylglycerol, *TC* total cholesterol, *HDL* high density lipoprotein, *LDL* low density lipoprotein, *Apo A1* apolipoprotein A1, *Apo B* apolipoprotein B, *Lpa* lipoprotein (a)

[a] Low glycemic index diet defined as having a glycemic index of less than 50

[b] Healthy nutrition recommendations emphasized on limiting foods with high content of fats, fast foods, French fries, fried foods, industrial beverages and unhealthy fats, drinking 1.5–2 l of water, consuming the large amount of fruits and vegetable with different varieties, low fat dairy and whole grains

[c] P values present percent changes between two groups (computed by independent samples t test)

[d] All values are mean ± SE

Acknowledgments The Department of Community Nutrition, School of Nutrition and Food Science, Isfahan University of Medical Sciences, Isfahan, Iran has supported this study with a grant for which we are grateful.

Conflict of interest The authors have no conflicts of interests.

References

1. Ejim EC, Okafor CI, Emehel A, Mbah AU, Onyia U, Egwuonwu T et al (2011) Prevalence of cardiovascular risk factors in the middle-aged and elderly population of a Nigerian rural community. J Trop Med 2011:308687
2. Da-Conceição Ferreira CC, Peixoto Mdo R, Barbosa MA, Silveira EA (2010) Prevalence of cardiovascular risk factors in elderly individuals treated in the Brazilian public health system in Goiânia. Arq Bras Cardiol 95:621–628
3. Goff DC Jr, Bertoni AG, Kramer H, Bonds D, Blumenthal RS, Tsai MY et al (2006) Dyslipidemia prevalence, treatment, and control in the multi-ethnic study of atherosclerosis (MESA): gender, ethnicity, and coronary artery calcium. Circulation 113:647–656
4. Ruixing Y, Fengping H, Shangling P, Dezhai Y, Weixiong L, Tangwei L et al (2006) Prevalence of hyperlipidemia and its risk factors for the middle-aged and elderly in the Guangxi Hei Yi Zhuang and Han populations. J Investig Med 54:191–200
5. Ramezani M, Azizi F (2005) Prevalence of hyperlipidemia in 3-19 years old children and adolescent of Tehran in 2000. IJEM 7:249–253
6. Kelishadi R, Hashemi M, Mohammadifard N, Asgary S, Khavarian N (2008) Association of changes in oxidative and proinflammatory states with changes in vascular function after a lifestyle modification trial among obese children. Clin Chem 54:147–153
7. Moayeri H, Aghighi Y (2006) Prevalence of hyperlipidemia in a group of obese adolescents in Tehran. IRCMJ 8:30–125
8. Kelly RB (2010) Diet and exercise in the management of hyperlipidemia. Am Fam Phys 81:1097–1102
9. Levitan EB, Cook NR, Stampfer MJ, Ridker PM, Rexrode KM, Buring JE et al (2008) Dietary glycemic index, dietary glycemic load, blood lipids, and C-reactive protein. Metabolism 57:437–443
10. Rizkalla SW, Taghrid L, Laromiguiere M, Huet D, Boillot J, Rigoir A et al (2004) Improved plasma glucose control, whole-body glucose utilization, and lipid profile on a low-glycemic index diet in type 2 diabetic men: a randomized controlled trial. Diabetes Care 27:1866–1872
11. de Rougemont A, Normand S, Nazare JA, Skilton MR, Sothier M, Vinoy S (2007) Beneficial effects of a 5-week low-glycaemic index regimen on weight control and cardiovascular risk factors in overweight non-diabetic subjects. Br J Nutr 98:1288–1298
12. Morales LM, Semprún-Fereira M, Ryder E, Valbuena H, Rincón E, Fernandez V et al (1997) Improved triglyceride control with low glycaemic index-high carbohydrate modified-lipid diet in a hypertriglyceridaemic child. Acta Paediatr 86:772–774
13. Acheson KJ (2010) Carbohydrate for weight and metabolic control: where do we stand? Nutrition 26:141–145
14. Iannuzzi A, Licenziati MR, Vacca M, De Marco D, Cinquegrana G, Laccetti M et al (2009) Comparison of two diets of varying glycemic index on carotid subclinical atherosclerosis in obese children. Heart Vessel 24:419–424
15. Fajcsak Z, Gabor A, Kovacs V, Martos E (2008) The effects of 6-week low glycemic load diet based on low glycemic index foods in overweight/obese children—pilot study. J Am Coll Nutr 27:12–21
16. Parillo M, Licenziati MR, Vacca M, De Marco D, Iannuzzi A (2011) Metabolic changes after a hypocaloric, low-glycemic index diet in obese children. J Endocrinol Invest

17. Sonoyama K, Nishikawa H, Kiriyama S, Niki R (1994) Cholestyramine and a fat-free diet lower apolipoprotein A-IV mRNA in jejunum and cholestyramine lowers apolipoprotein A-I mRNA in ileum of rats. J Nutr 124:621–627
18. Ahmed N, Anwar W, Waqas H (2009) Obesity, hyperlipidemia, and hyperuraecemia in young and old hypertensive patients. J Ayub Med Coll Abbottabad 21:53–56
19. Newfield RS, Dewan AK, Jain S (2008) Dyslipidemia in children with type 2 diabetes vs. obesity. Pediatr Diabetes 9:115–121
20. World Health Organization (2011) BMI-for-age girls. http://www.who.int/entity/growthref/bmifa_girls_5_19years_per.pdf. Accessed September 2011
21. Azadbakht L, Mirmiran P, Hosseini F, Azizi F (2005) Diet quality status of most Tehranian adults needs improvement. Asia Pac J Clin Nutr 14:163–168
22. Institute of Medicine (US) Energy (2005) In: Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids, 1st edn. The National Academies Press, Washington, p 217
23. Azizi F, Esmaillzadeh A, Mirmiran P (2005) Correlates of under- and over-reporting of energy intake in Tehranians: body mass index and lifestyle-related factors. Asia Pac J Clin Nutr 14:54–59
24. Azam-Taleban F, Esmaeili M (1999) Glycemic index of Iranian foods, 1st edn. National Nutrition and Food Technology Research Institute, Tehran (in Farsi)
25. Foster-Powell K, Holt SH, Brand-Miller JC (2002) International table of glycemic index and glycemic load values: 2002. Am J Clin Nutr 76:5–56
26. Fleiss JL (1986) The design and analysis of clinical experiments. John Wiley, London, pp 263–271
27. Zhang Z, Lanza E, Kris-Etherton PM, Colburn NH, Bagshaw D, Rovine MJ et al (2010) A high legume low glycemic index diet improves serum lipid profiles in men. Lipids 45:765–775
28. Bukkapatnam RN, Berglund L, Anuurad E, Devaraj S, Hyson D, Rafii F et al (2010) Postprandial metabolic responses to dietary glycemic index in hypercholesterolemic postmenopausal women. Prev Cardiol 13:29–35
29. Shikany JM, Phadke RP, Redden DT, Gower BA (2009) Effects of low- and high-glycemic index/glycemic load diets on coronary heart disease risk factors in overweight/obese men. Metabolism 58:1793–1801
30. Wolever TM, Gibbs AL, Mehling C, Chiasson JL, Connelly PW, Josse RG et al (2008) The Canadian trial of carbohydrates in diabetes (CCD), a 1-y controlled trial of low-glycemic-index dietary carbohydrate in type 2 diabetes: no effect on glycated hemoglobin but reduction in C-reactive protein. Am J Clin Nutr 87:114–125
31. Kabir M, Oppert JM, Vidal H, Bruzzo F, Fiquet C, Wursch P et al (2002) Four-week low-glycemic index breakfast with a modest amount of soluble fibers in type 2 diabetic men. Metabolism 51:819–826
32. Marcovina S, Packard CJ (2006) Measurement and meaning of apolipoprotein AI and apolipoprotein B plasma levels. J Intern Med 259:437–446
33. Jung CH, Hwang JY, Shin MS, Yu JH, Kim EH, Bae SJ et al (2013) Association of apolipoprotein b/apolipoprotein A1 ratio and coronary artery stenosis and plaques detected by multidetector computed tomography in healthy population. J Korean Med Sci 28:709–716
34. Garcés C, Oya ID, Lasunción MA, López-Simón L, Cano B, de Oya M (2010) Sex hormone-binding globulin and lipid profile in pubertal children. Metabolism 59:166–171
35. Nikolic D, Katsiki N, Montalto G, Isenovic ER, Mikhailidis DP, Rizzo M (2013) Lipoprotein subfractions in metabolic syndrome and obesity: clinical significance and therapeutic approaches. Nutrients 5:928–948
36. Lau VW, Journoud M, Jones PJ (2005) Plant sterols are efficacious in lowering plasma LDL and non-HDL cholesterol in hypercholesterolemic type 2 diabetic and nondiabetic persons. Am J Clin Nutr 81:1351–1358
37. Mensink RP, Zock PL, Kester AD, Katan MB (2003) Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. Am J Clin Nutr 77(5):1146–1155
38. Jousilahti P, Vartiainen E, Tuomilehto J, Puska P (1999) Sex, age, cardiovascular risk factors, and coronary heart disease: a prospective follow-up study of 14 786 middle-aged men and women in Finland. Circulation 99:1165–1172
39. Buyken AE, Cheng G, Günther AL, Liese AD, Remer T, Karaolis-Danckert N (2008) Relation of dietary glycemic index, glycemic load, added sugar intake, or fiber intake to the development of body composition between ages 2 and 7 y. Am J Clin Nutr 88:755–762
40. Reaven GM (1995) Pathophysiology of insulin resistance in human disease. Physiol Rev 75:473–486
41. Azadbakht L, Esmaillzadeh A (2008) Dietary and non-dietary determinants of central adiposity among Tehrani women. Public Health Nutr 11:528–534
42. Azadbakht L, Mirmiran P, Esmaillzadeh A, Azizi F (2006) Dietary diversity score and cardiovascular risk factors in Tehranian adults. Public Health Nutr 9:728–736