The Impact of the Glycemic Index, Glycemic load and the Macronutrient intake of the Diet of overweight and obese Children on Metabolic Syndrome Development

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Abstract

Background/Objective

A significant percentage of obese children develop metabolic abnormalities. The aim of the study was to investigate whether the diet of the overweight/obese Greek children and adolescents who develop metabolic syndrome (MS) differ from the diet of those who do not concerning glycemic index (GI), glycemic load (GL) and macronutrient intake.

Methods

Three-day food diaries were analyzed from 96, randomly selected overweight and obese girls and boys, aged 5-16 years, regularly followed in the Obesity Clinic of the First Department of Pediatrics of the University of Athens. The diets were assessed for their total daily energy, protein, carbohydrate, fiber, fat content, GI and GL, using Food Processor ESHA-SQL 10.1. Data including: weight, height, pubertal stages, blood pressure, fasting plasma levels of total cholesterol, low-density and high-density lipoprotein cholesterol, triglycerides, glucose, insulin levels and calculated homeostasis model assessment index (HOMA), were collected. Results were analyzed per gender as a total and segregating per pubertal stage.

Results

In our sample of obese children, more than a third of girls and almost half of the boys had MS, with insulin resistance (HOMA) and hypertension being the most common components. Prepubertal boys with MS had a diet with a significantly greater total daily energy intake (p=0.039) and GL (p=0.032) compared to those without MS. In girls with MS there was a tendency for a greater fat intake (p=0.075), only evident in pre-pubertal girls (p=0.091). No other dietary differences were observed.

Conclusion

The higher GL of the diet and total daily energy intake were found to be the dietary factors significantly associated with the occurrence of MS in obese pre-pubertal boys. The diet of children at the pre-pubertal stage is suggested to be of great significance in the occurrence of MS. Larger prospective studies are needed to confirm these results.

Key words: Glycemic Load; Glycemic Index; Metabolic Syndrome; Children; Diet.

Introduction

Childhood and adolescent obesity is frequently associated with metabolic abnormalities, complications previously seen only in adults [1, 2]. The term metabolic syndrome (MS) is commonly used to describe the clustering of metabolic factors linked to an increased incidence of cardiovascular disease, type 2 diabetes and mortality in adults [3]. More than 15 pediatric studies on the metabolic syndrome have been conducted using different set of diagnostic criteria including the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATPIII), World Health Organization (WHO) and the European Group for the Study of Insulin Resistance (EGIR) criteria [4] as well as the International Diabetes Federation (IDF) criteria [5]. The role of the diet in the development of MS is still unclear and needs further investigation [6, 7].

The glycemic index (GI) of foods is a term that was first introduced in 1981 by Jenkins and is used to categorize carbohydrate containing foods according to their postprandial effect on blood glucose levels [8] i.e. the quality. GI is defined as the ratio of the incremental area under the glycemic response curve (AUC) elicited by the consumption of 50 grams of available carbohydrate from the test food (F) divided by the AUC after the consumption of 50 grams of the reference food (R) on separate occasions in the same subject, usually the reference food being glucose (GI = 100 x AUC_F / AUC_R). Glycemic load (GL) is the product of GI with the quantity

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of available carbohydrate consumed divided by 100 (GL = GI x the quantity of available carbohydrate in grams consumed/100) [9].

There is a growing evidence indicating the positive contribution of GI and GL in planning a healthier diet and their favorable effect on metabolic parameters such as triglycerides, HDL and insulin response [10-14]. However, the debate still continues as to their efficacy as independent factors in inducing metabolic changes [15,16].

**Aim of the Study**

The aim of this study is to investigate in a group of Greek overweight and obese children and adolescents, the impact of the GI, GL and macronutrient intake of their diet on the prevalence of MS, segregating also by pubertal stage.

**Subjects and Methods**

For the purpose of this study a sample of 108 overweight and obese girls (n=64) and boys (n=44), aged 5 to 15 years were examined from January 2007 to May 2010. This was a random sample of the children and adolescents attending the Obesity Clinic of the Division of Endocrinology, Diabetes and Metabolism of the First Department of Pediatrics of the University of Athens, in “Aghia Sofia” Children’s Hospital. The selection criteria were a BMI of overweight or obesity according to the definition of Cole et al [17], not being on a diet for the last 6 months, not taking any medication and being otherwise healthy. Twelve children, 8 girls and 4 boys, were excluded due to missing data. Of the 56 girls remaining, 52 were obese and 4 overweight and from the 40 boys, 37 obese and 3 overweight.

Children were asked to keep a 3-day food diary, prior to their initial evaluation. The food diaries were analyzed for carbohydrate, fat, protein, fiber, energy, GI and GL content. Food models were used for better estimation of portion sizes. The study’s protocol has been approved by the ethical committee of the Children’s Hospital and children and families were voluntarily involved in the study, signing an informed consent.

Anthropometric measurements were assessed including weight, height, BMI, blood pressure, and Tanner stages of puberty[18]. Pubertal Tanner stage 1 was used to describe pre-pubertal girls and boys, and girls and boys with Tanner stages ≥ 2 were grouped as “pubertal”. In addition, blood was withdrawn after a 12 hour overnight fast and plasma levels of total cholesterol, LDL, HDL, triglycerides, glucose and insulin levels were determined. Wherever indicated, an oral glucose tolerance test (OGTT) was undertaken. The insulin resistance HOMA (Homeostasis Model Assessment) index (HOMA-IR) was calculated according to the initial formula (glucose mmol/l x insulin m U/L / 22.5) [19]. The dietary analysis was performed using The Food Processor ESHA-SQL 10.1 program, which includes GI values of foods based on the International table of glycemic index and glycemic load values [20] and the website of the University of Sidney (www.glycemicindex.com).

For the purpose of this study the MS definition used, as in Weiss et al [21] was based on the modified National Cholesterol Education Program’s Adult Treatment Panel (ATPIII) [22] adapted using our national percentile curves modified ATP III, based on the Greek population: childhood obesity using the definition by Cole et al. [17] in the Greek children population BMI charts [23], Increased blood pressure, either increased systolic or diastolic blood pressure, > 95th percentile for age and gender [24], increased triglycerides (> 90th percentile) based on the percentiles for lipid levels in Greek children [25], selecting the 90th percentile as the cut-off point which is closer to the universal adult cut-off of 150mg/dl as chosen for the identification of cut-off points for hyperlipidemia in US children and adolescents [21, 26]. Similarly, low HDL (< 10th percentile) for age and sex; high LDL >90th percentile. Impaired glucose tolerance was diagnosed as a 2hour glucose value in the OGTT of greater than 140 and less than 200 mg/dl or impaired fasting glucose with a fasting value of greater than 100 mg/dl [21, 27]. For the foods that did not have a GI assigned to them, a GI was estimated based on the methodology used for this estimation as described in the Methodology for adding glycemic load values to the National Cancer Institute (NCI) Diet History Questionnaire Database[28] and in the Development of a Glycemic Index Database for Food Frequency Questionnaires (FFQ) used in Epidemiological Studies [29].

**Statistical Analysis**

For the statistical analysis the SPSS 21.0 package was used, with the significance level set at p≤0,05. Both t-test and Mann-Whitney test were used, depending on whether the parameter analyzed was following a normal distribution or not, respectively.

**Results**

More than a third of the girls included in our study and almost half of the boys were diagnosed with MS. The most common components of the MS observed in our sample were insulin resistance, diagnosed in about 55 % of the girls and 41 % of the boys and hypertension in more than 30% of the girls and close to 50% of the boys (Table 1).

The comparison of the diet of girls and boys with MS to those without, irrespective of pubertal stage, showed no significant differences in the GI/ GL, carbohydrate, fiber and protein intake, expressed as grams or percentages (%) of total daily energy intake (p≥ 0,05). Only a tendency for greater fat gram intake in girls with MS (p=0,075) was observed (Table 2).

However, when the samples were analyzed according to pubertal stage (Table 3), there was a significant difference in the diet of pre-pubertal boys with MS, having a greater energy (p=0,039) and GL (p=0,032) intake as compared to those without MS. This was not observed in pubertal boys. In girls no significant differences were observed when the sample was analyzed per pubertal stage. The tendency towards a greater fat intake observed in the total number of girls persisted only in pre-pubertal girls (p=0,091) (Table 3).

**Discussion**

Metabolic syndrome is common in obese children and adolescents and is associated with cardiovascular disease. In this study of
Table 1: Percentage of subjects presenting metabolic abnormalities in a sample of overweight and obese girls and boys

<table>
<thead>
<tr>
<th></th>
<th>Ob/ Owt</th>
<th>High TG</th>
<th>Low HDL</th>
<th>High SBP/DBP</th>
<th>High LDL</th>
<th>HOMA &gt; 3</th>
<th>IFG/IGTF</th>
<th>&gt; 3 factors Excluding HOMA, LDL</th>
<th>&gt; 3 factors including HOMA, LDL</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Girls (N=56)</strong></td>
<td>92.8% Ob 7.2% Owt</td>
<td>10.7%</td>
<td>39.3%</td>
<td>32.1%</td>
<td>16.1%</td>
<td>55.3%</td>
<td>21.4%</td>
<td>36.3%</td>
<td>53.6%</td>
</tr>
<tr>
<td><strong>Boys (N=40)</strong></td>
<td>92.5% Ob 7.5% Owt</td>
<td>21.9%</td>
<td>24.4%</td>
<td>48.8%</td>
<td>22.5%</td>
<td>41.4%</td>
<td>12.2%</td>
<td>48.8%</td>
<td>52.5%</td>
</tr>
</tbody>
</table>

Abbreviations/Notes: Ob= Obese, Owt= Overweight, %ile = percentile, BMI = Body Mass Index, TG= triglycerides, hypertriglyceridemia defined as fasting plasma levels > 90th % ile for age and gender, LDL = Low density Lipoprotein with hyperlipidemia defined as fasting plasma LDL levels > 90th %ile for age and gender, HDL = High density Lipoprotein with low levels defined as fasting plasma levels < 10th %ile, SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure with hypertension defined as SBP or DBP > 95th % ile, HOMA = Homeostasis Model Assessment with insulin resistance defined as HOMA > 3, IFG = Impaired fasting glucose defined as > 100mg/gl, IGT = Impaired Glucose Tolerance, defined as 2 hour glucose levels after an Oral Glucose Tolerance Test of > 140mg/dl and < 200 mg/dl

Table 2: Comparison of the GI, GL and macronutrient intake of the diet of girls and boys with versus without MS irrespective of pubertal status

<table>
<thead>
<tr>
<th>Variable</th>
<th>Girls without MS</th>
<th>p value</th>
<th>Girls with MS</th>
<th>p value</th>
<th>Boys without MS</th>
<th>p value</th>
<th>Boys with MS</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI</td>
<td>58,06</td>
<td>0,556</td>
<td>57,36</td>
<td>0,949</td>
<td>58,51</td>
<td>127,2</td>
<td>58,98</td>
<td>0,213</td>
</tr>
<tr>
<td>GL</td>
<td>105,31</td>
<td>0,163</td>
<td>105,82</td>
<td>0,163</td>
<td>122,7</td>
<td>2154</td>
<td>2374,8</td>
<td>0,101</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>75,6</td>
<td>0,0173</td>
<td>74,6</td>
<td>0,137</td>
<td>91</td>
<td>16,9</td>
<td>93,8</td>
<td>0,542</td>
</tr>
<tr>
<td>% energy</td>
<td>16,8</td>
<td>0,743</td>
<td>15,9</td>
<td>0,0173</td>
<td>16,9</td>
<td>17,0</td>
<td>17,0</td>
<td>0,836</td>
</tr>
<tr>
<td>Carbohydrates (g)</td>
<td>185</td>
<td>0,599</td>
<td>201,5</td>
<td>0,549</td>
<td>236,5</td>
<td>45,1</td>
<td>261,4</td>
<td>0,159</td>
</tr>
<tr>
<td>% energy</td>
<td>44,6</td>
<td>0,221</td>
<td>42,7</td>
<td>0,431</td>
<td>41,6</td>
<td>13,9</td>
<td>44,3</td>
<td>0,931</td>
</tr>
<tr>
<td>Fiber (g)</td>
<td>12,9</td>
<td>0,0173</td>
<td>12,9</td>
<td>0,431</td>
<td>13,9</td>
<td>14,3</td>
<td>14,3</td>
<td>0,749</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>74,1</td>
<td>0,075</td>
<td>83,3</td>
<td>0,221</td>
<td>94,4</td>
<td>38,4</td>
<td>40,1</td>
<td>1,00</td>
</tr>
<tr>
<td>% energy</td>
<td>39,4</td>
<td>0,221</td>
<td>41,6</td>
<td>0,221</td>
<td>38,4</td>
<td>40,1</td>
<td>40,1</td>
<td>1,00</td>
</tr>
</tbody>
</table>

Table 3: Analysis per pubertal stage comparing the diet of girls and boys with MS to those without

<table>
<thead>
<tr>
<th>Variables</th>
<th>Pre-pubertal girls¹</th>
<th>p values</th>
<th>Pubertal girls²</th>
<th>p values</th>
<th>Pre-pubertal boys ³</th>
<th>p values</th>
<th>Pubertal boys ⁴</th>
<th>p values</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI</td>
<td>0,471</td>
<td>0,332</td>
<td>0,404</td>
<td>0,776</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GL</td>
<td>0,437</td>
<td>0,919</td>
<td>0,039</td>
<td>0,891</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Energy (kcal)</td>
<td>0,506</td>
<td>0,640</td>
<td>0,032</td>
<td>0,921</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein (g)</td>
<td>0,618</td>
<td>0,823</td>
<td>0,227</td>
<td>0,630</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>0,824</td>
<td>0,670</td>
<td>0,070</td>
<td>0,527</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fiber (g)</td>
<td>0,890</td>
<td>0,248</td>
<td>0,364</td>
<td>0,952</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat (g)</td>
<td>0,091</td>
<td>0,670</td>
<td>0,156</td>
<td>0,921</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notes:
¹ Prepubertal girls with MS (N=12) versus without MS (N=14)
² Pubertal girls with MS (N= 12) versus without MS (N=18)
³ Prepubertal boys with MS (N=13) versus without MS (N=16)
⁴ Pubertal boys with MS (N= 8) versus without MS (N=3)
overweight and obese children the incidence MS was higher compared to that (16.5-35.7%) found in the obese European children study [30] probably due to the fact that this is a clinic population, that may inherently have a motivation to seek assistance in comparison to the general population of overweight children. The most common obesity related traits encountered in our sample were insulin resistance as measured by HOMA index and hypertension, which are in accordance with previous findings [31-33], demonstrating the influence of the “westernized type” lifestyle and obesity in children.

There are few cross-sectional studies, up to date, addressing the macronutrient composition including GI and GL in relation to MS development in children and adolescents. The available evidence suggests that total energy, fat and protein are predictors of parameters of MS in 6 to 14 year old children [32]. In addition, a higher GI, GL and carbohydrate diet in adolescents, was associated with higher odds for IDF defined MS [7]. Furthermore, the prevalence of MS in the Framingham Offspring Cohort was positively associated with GI and negatively with whole grain and cereal fiber intake [34].

In accordance, our data suggest an association of total energy intake and GL to prevalence of MS, dependent on pubertal stage and gender. Specifically, it was shown that pre-pubertal obese boys, who have already developed metabolic abnormalities fulfilling criteria for MS, were following a significantly higher GI and energy containing diet compared to their obese peers without MS. These findings agree with the data from adolescents in the Dortmund Nutritional and Anthropometric Longitudinally Designed Study (DONALD) where a high GI diet at puberty onset, was associated with a tendency for a higher % body fat and BMI standard deviation scores in overweight but not normal weight adolescents. This may be preliminary evidence for the importance of entering puberty on a lower GI and GL diet, as being protective factors against overweight and MS. This association was not observed in the adolescents of developing MS have a higher energy intake and higher GL in their diet in comparison to their age-matched counterparts not demonstrating MS. Thus childhood may be an important phase of life, where dietary interventions may mostly have an impact on future health. Entering adolescence with a lower GI and GL diet, providing adequate but not excessive energy intake, may be crucial for the prevention of MS and protection against increased body fat in adolescence and adulthood. If our results are confirmed in larger multinational prospective studies, a low GL, energy reduced diet could be a useful tool in targeting specifically pre-pubertal overweight and obese boys for prevention of MS.

**Acknowledgement**

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**Reference**


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