ORIGINAL ARTICLES

Weight Management by Using Low Glycemic Index Diets

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ABSTRACT

In light of widespread concern regarding the high toll of the obesity epidemic on human suffering and health care costs, development of effective weight-management strategies is a public health priority. Among the many different diet approaches to reduce body weight, conventional low-fat and low calorie diet approach has been disappointing. The glycemic index (GI) and glycemic load (GL) have been proposed as a method of ranking foods on the basis of the incremental blood glucose response they produce for a given amount of carbohydrates. The aim of this study was to investigate whether modifying the GI of obese rats diets by incorporating low-GI foods (spinach leaves, chickpea and lentil seeds) for 30 days would have a favorable effect on food intake, energy efficiency, blood glucose, serum insulin, lipids profile, body weight and body composition in obese rats. It was found that feeding on high-fat diet (positive control obese rats) caused significant increase in body weight, relative organs weight, epididymal fat pad weight, food intake, energy efficiency, blood glucose and serum insulin levels, insulin sensitivity index, triglycerides, lipids profile (TC, LDL and VLDL) levels, serum leptin and malondialdehyde (MDA) levels compared with negative control obese rats. On the other hand, feeding on that diet caused significant decrease in oxidative status relative enzymes (SOD and GPx), lipase enzyme activity, phospholipids and total lipids concentrations. Feeding obese rats on different low-GI diets showed significant improvement in the above mentioned parameters and caused reduction in body weight by 26.23%, 28.19%, 26.83% and 27.05% for rats fed on chickpea seeds, lentil seeds, spinach leaves and spinach leaves + chickpea seeds, respectively. Food intake and energy efficiency were lower by feeding on low-GI diets compared with high-fat diet. Blood glucose and serum insulin levels were significantly decreased by feeding on these diets and relative insulin resistance index (HOMA-R) was 1.8, 3.1, 2.4 and 2.7 fold less for low-GI diets. SOD and GPx enzymes were increased significantly and there was a significant decrease in MDA level, a marker of inflammation, compared with positive control rats. A significant increase in HDL-cholesterol was noticed by feeding on low-GI diets with decrease in lipids profile. Leptin levels were significantly decreased, while lipase, phospholipids and total lipids were increased by using low-GI diets. The highest improvement in these parameters was achieved by feeding on lentil seeds powder followed by spinach leaves + chickpea seeds powders, spinach leaves powder and chickpea seeds powder, respectively. Thus, it could be concluded that carefully designed low-GI diets may be a useful tool for management of body weight.

Key words: Glycemic index; Body weight; Food intake; Glucose; Insulin; Oxidative status; Leptin; Lipase.

Introduction

Obesity is one of today's most visible–yet most neglected– public health problems. Obesity is associated with a number of metabolic disorders including coronary heart disease, hypertension, stroke, certain cancers, non–insulin diabetes mellitus, gallbladder disease and dyslipidemia (AIHW, 2003). On a global level, the World Health Organization (WHO) warns of a chronic disease epidemic and projects that by 2015 there will be 2.3 billion overweight adults, plus more than 700 million obese adults. The WHO estimates that currently around one in every three of the world's adult is overweight, while nearly one in every ten is obese. According to the WHO statistics, Egypt is the fattest African country and also the 14th fattest country in the world with nearly 70% of its adult population overweight or obese. Among Egyptians above the age of 15 there are more overweight and obese females (76%) than males (64.5%). The rate of obesity in Egypt has risen markedly over the past 30 years. Egypt had the highest average body mass index in the world at 26.3 and 1.6% of 2-6 year olds, 4.9% of 6-10 year olds, 14.7% of 10-14 year olds and 13.4% of 14-18 year olds were obese (Osman, 2002 and WHO, 2012). Dietary intervention is one of the main aspects in the management of obesity. Studies have shown that dietary fat is associated with obesity and a low fat diet was effective in weight loss in some people. However, the steady increase in the obesity rate in spite of the apparent decrease in fat consumption suggests
that other dietary factors may be involved (Bray et al., 2004). It has recently been suggested the reason these low fat, high carbohydrate diets may be ineffective is that high carbohydrate diets may incorporate foods that promote a high glycemic response (a rapid increase in blood glucose concentration). Such foods may alter appetite and energy partition in a manner that is conductive to fat gain (Brand–Miller et al., 2002).

Increasing evidence is now showing that the quality and quantity of carbohydrates may play a significant role in the development of chronic diseases (Liu et al., 2001). Currently, there is much interest in the potential of low glycemic index (GI) foods in the management of obesity. The glycemic index is defined as the area under the glycemic response curve for 2 hrs after consumption of 50 g carbohydrate from a test food divided by the area under the curve for 2 hrs after consumption of 50 g carbohydrate from a control substance (either white bread or glucose). The GI is a system for classifying carbohydrate–containing foods according to how blood glucose concentrations change in the postprandial period. Vegetables, legumes and fruit generally have low–GI. The presence of fiber, protein and fat in a meal lowers the GI of co-ingested carbohydrates. Although fiber and glycemic index are not equivalent, they tend to be related because viscous dietary fibers and foods in which the natural cell wall architecture remains intact (e.g., legumes) generally have lower glycemic indexes (Jenkins et al., 2000 and Ludwig, 2002). It has been hypothesized that low–GI foods may benefit weight regulation in 2 ways: by promoting satiety and by promoting fat oxidation at the expense of carbohydrate oxidation. Studies have also shown that a low–GI diet is associated with lower fasting plasma low-density lipoprotein cholesterol, fasting triglyceride, postprandial glucose concentrations and increase in fasting plasma high-density lipoprotein concentrations (Bouche et al., 2002). It was observed that the low–GI meal resulted in lower plasma glucose, serum insulin, plasma glucagon–like peptide-1 and higher plasma glucose–dependent insulinotropic polypeptide concentrations than the high–GI meal. Ratings of fullness were slightly higher and the desire to eat something fatty was lower after the low–GI meal after 10-wk ad libitum intake compared to a high–GI meal (krog–Mikkelsen et al., 2011).

Ann et al. (2012) found that, reducing the GI or carbohydrate content of mixed meals reduces postprandial glycemia and insulinemia, and these changes can be sustained over the course of an entire day. These results demonstrate that maintaining a low–GI diet is an effective method for controlling serum glucose and insulin levels and give some support to recommendations to consume a low–GI diet. Given that overweight and obese people are at greater risk of developing diabetes and cardiovascular diseases, changing GI of foods may favorably influence their health prospect by conferring benefits to their blood glucose and lipid profiles. If this leads to a reduction in body weight, a further reduction in health risks could be seen. Therefore, the aim of this study was to investigate whether modifying the GI of obese rats diets by incorporating low–GI foods (spinach leaves, chickpea and lentil seeds) for 30 days would have a favorable effect on food intake, energy efficiency, blood glucose, serum insulin, lipids profile, body weight and epididymal fat pad weight in obese rats.

Materials And Methods

Materials:

Spinach leaves (Spinacia oleracea), lentil seeds (Lens esculenta) and chickpea seeds (Cicer arietinum) were purchased from local market of Giza Governorate, Egypt.

Methods:

Preparation of Raw Materials:

Spinach leaves were washed with tap water after removing inedible parts manually and blanched in water for 5 min. Dried lentil seeds were boiled in water for 35 min., and dried chickpea seeds were soaked in water then boiled for 35 min. Raw materials were dried in an oven at 50 ℃, ground to powder form and stored at –20 ℃ until used.

Chemical Analysis:

Crude protein, ether extract, total ash and crude fiber contents of raw materials and the different experimental diets were determined as described in A.O.A.C. (2006).

Animals and Experimental Diets:

A total of 42 male Sprague–Dawley obese rats were obtained from Ophthalmology Research Institute, Giza, Egypt. All rats weighing 360 to 370 g were fed on basal diet for one week (adaptation period). The rats were divided into 6 groups, each group contained 7 rats. The experimental diets formulation was performed according
to Luke et al. (2008) with some modifications. The first group of obese rats fed on basal diet and considered as a negative control group. The other 5 groups of obese rats were fed on the following scheme for 30 days: (1) obese rats fed on high fat diet (20% corn oil instead of 10% in the basal diet) and considered as a positive control group, (2) obese rats fed on high fat diet substituted with 10% chickpea seeds powder, (3) obese rats fed on high fat diet with 10% lentil seeds powder, (4) obese rats fed on high fat diet with 10% spinach leaves powder and (5) obese rats fed on high fat diet with 5% spinach leaves + 5% chickpea seeds powders. The substitution levels of raw materials added to the diets were subtracted from corn starch and sucrose contents. Casein level was modified according to percentage of protein in raw materials. Corn oil instead of soybean oil was used as the fat source and maltodextrin was excluded from the modified diets to maximize the effect of the test low glycemic index materials. The compositional modifications of diets aimed at maintaining nonstarch ingredients consistent among the different diets and eliminating variability that could influence the results (Reeves et al., 1993). All high fat diets substituted with different low glycemic index materials were identical for macronutrient composition (protein, fat and carbohydrate contents), total energy and fiber contents, but only differed in the low glycemic index of the raw materials (Table 1). The glycemic index of the raw materials was 10 for blanched spinach leaves, 22 for boiled lentil seeds and 10 for soaked and boiled chickpea seeds as reported by Foster-Powell et al. (2002) and Internet Database (2006). From the international GI tables, the GI of all of the low glycemic index foods was estimated to be < 55 (glucose = 100). Daily food intake was determined and rats were weighed at the beginning of the dietary period and at the end of each week. At the end of the experimental period, the rats were sacrificed, liver, kidney, spleen, heart, epididymal fat pad weight and the gastrointestinal tract from stomach to rectum (cecum) were rapidly removed and weighed.

Biochemical Analysis:

Blood was collected from food-deprived rats for 12 hrs. and the serum was separated and stored at –20°C for analysis. Plasma glucose levels were determined according to the method described by Trinder (1969). Insulin levels were measured by Ins-ELISA kit (Biosource Europe S.A., B-1400 Nivelles, Belgium) according to Temple et al. (1992). Glutathione peroxidase (GPx) and superoxide dismutase (SOD) activities in serum were determined according to the methods of Daret and Ching (1996) and Marklund and Marklund (1974), respectively. Lipid peroxidation level (malondialdehyde, MDA) was estimated as described by Meltzer et al. (1997). Lipase enzyme activity was measured as mentioned by Lott (1986), while serum leptin levels were estimated by radioimmunoassay kit (Linco Research, Inc., St Charles, MO). Serum total lipids and phospholipids were determined according to the methods of Frings and Dunn (1970) and Holman (1943), respectively. Serum total cholesterol, (high-density lipoprotein cholesterol and low-density lipoprotein cholesterol), very low-density lipoprotein cholesterol and triglycerides were determined according to the methods of Roeschlau et al. (1974); Assmann (1979); Hatch and Lees (1968) and Uwajima et al. (1984), respectively. The homeostasis model assessment (HOMA) was used to assess β-cell function and insulin sensitivity index from fasting glucose and insulin concentrations. HOMA-R (insulin resistance) = glucose X insulin / 22.5, while HOMA-ß (Pancreatic β-cell function) = (20 X insulin) / (glucose – 3.5), where plasma glucose is expressed in mmol/L and serum insulin is expressed in mU/L as described by Wallace et al. (2004).

Statistical Analysis:

The standard analysis of variance procedure in a completely randomized design was applied for the present data according to Gomez and Gomez (1984). Least significant difference (LSD) test was done to compare a pair of group means. The level of statistical significance was set at P < 0.05.

Table 1: Composition of experimental diets (%).

<table>
<thead>
<tr>
<th>Diets</th>
<th>Protein</th>
<th>Fat</th>
<th>Fiber</th>
<th>Ash</th>
<th>Carbohydrates*</th>
<th>Total energy (kcal/100g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative control</td>
<td>10.34 A</td>
<td>10.75 B</td>
<td>4.39 A</td>
<td>5.33 A</td>
<td>69.19 A</td>
<td>414.87 C</td>
</tr>
<tr>
<td>Positive control</td>
<td>9.82 A</td>
<td>20.91 C</td>
<td>4.25 A</td>
<td>4.62 A</td>
<td>60.40 A</td>
<td>469.07 B</td>
</tr>
<tr>
<td>10% Chickpea seeds powder</td>
<td>10.27 A</td>
<td>20.55 C</td>
<td>4.64 A</td>
<td>5.84 A</td>
<td>58.70 A</td>
<td>460.83 C</td>
</tr>
<tr>
<td>10% Lentil seeds powder</td>
<td>10.48 A</td>
<td>20.52 C</td>
<td>4.19 A</td>
<td>6.25 A</td>
<td>58.56 A</td>
<td>460.84 C</td>
</tr>
<tr>
<td>10% Spinach leaves powder</td>
<td>9.90 A</td>
<td>20.34 C</td>
<td>5.02 A</td>
<td>6.36 A</td>
<td>58.38 A</td>
<td>456.18 B</td>
</tr>
<tr>
<td>5% Spinach + 5% Chickpea powders</td>
<td>10.35 A</td>
<td>20.50 C</td>
<td>4.57 A</td>
<td>6.17 A</td>
<td>58.41 A</td>
<td>459.54 B</td>
</tr>
<tr>
<td>L.S.D.</td>
<td>0.86</td>
<td>3.56</td>
<td>0.92</td>
<td>1.76</td>
<td>1.82</td>
<td>7.64</td>
</tr>
</tbody>
</table>

* Carbohydrates were calculated by difference.
** Numbers in the same column followed by the same letter are not significantly different at P < 0.05.

Results And Discussion

Effect of Diets on Body Weight in Obese Rats:
Data presented in Table (2) show the effect of diets on body weight in obese rats from zero time to the end of the experiment (30 days). Body weight of obese rats for all groups at zero time was not significantly different and also during the experimental period for negative control rats, but it was significantly different for positive control rats which gained more body weight during the experimental period compared with negative control rats as a result of feeding on high fat diet. There was a significant gradual decrease in body weight for all obese rats groups fed on the experimental diets from zero time to the end of the experiment compared with negative and positive control rats. The reduction of body weight was 26.23%, 28.19%, 26.83% and 27.05% for rats fed on chickpea seeds powder, lentil seeds powder, spinach leaves powder and spinach leaves + chickpea seeds powders, respectively. These results indicate that consuming low glycemic index foods as a part of the meal caused loss of body weight and the highest loss in body weight was observed for lentil seeds diet followed by spinach leaves + chickpea seeds diet. It was found that spinach leaves diet caused more body weight loss than chickpea seeds diet. It was suggested that lowering the dietary glycemic index through a combination of moderate carbohydrate restriction, consumption of slowly digested and absorbed forms of dietary carbohydrates or both may be advantageous for persons who are attempting to loss excess body weight. Free-living subjects also showed greater weight loss over 3-6 months with reduced-carbohydrate or low-GI diets than with traditional low-fat, portion-controlled diets (Agus et al., 2000 and Yancy et al., 2004). Ma et al. (2005) observed that, daily dietary glycemic index was positively associated with body mass index (BMI), with a five-unit increase in glycemic index being significantly associated with an increase of 0.75 units in BMI in a sample of 20-70 years old men and women. In line with this, animal study observed positive associations between dietary GI and changes in obesity measures (Pawlak et al., 2004). At the end of the initial 12-wk weight-los period, the mean weight change was -4.9 ± 0.5 kg in the reduced-GI arm and -2.5 ± 0.5 kg in the control arm. At week 12, 24 subjects (55%) in the reduced-GI group and 9 subjects (21%) in the control group had achieved a loss of ≥ 5% of body weight (Maki et al., 2007). On contrary to low-GI diets, high-GI diets increase postprandial hyperinsulinemia which favors fatty acid uptake, inhibition of lipolysis and energy storage leading to weight gain. High-GI diets also lead to lower glucose nadir and increase in counter-regulatory hormones that may explain increased hunger and increased energy intake in the post-absorptive period, presumably leading to weight gain over time (Ludwig et al., 1999). After a low-GI test meal, carbohydrate oxidation was 12% lower and fat oxidation was 118% higher than after an high-GI test meal. Neither GI nor protein had an isolated effect on body composition. However, the low protein, high-GI combination increased body fat, whereas the high protein, low-GI combination significantly decreased the percentage of obese children and was protective against obesity (Wee et al., 1999 and Papadaki et al., 2010).

Table 2: Effect of diets on body weight in obese rats.

<table>
<thead>
<tr>
<th>Time Groups</th>
<th>Body weight (g)</th>
<th>At zero time</th>
<th>After 15 days</th>
<th>After 21 days</th>
<th>At the end</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative control</td>
<td></td>
<td>365.32</td>
<td>363.74</td>
<td>361.56</td>
<td>360.66</td>
</tr>
<tr>
<td>Positive control</td>
<td></td>
<td>367.00</td>
<td>380.17</td>
<td>385.53</td>
<td>388.64</td>
</tr>
<tr>
<td>10% Chickpea seeds powder</td>
<td></td>
<td>366.73</td>
<td>340.30</td>
<td>310.07</td>
<td>270.55</td>
</tr>
<tr>
<td>10% Lentil seeds powder</td>
<td></td>
<td>369.54</td>
<td>344.45</td>
<td>320.80</td>
<td>265.38</td>
</tr>
<tr>
<td>10% Spinach leaves powder</td>
<td></td>
<td>365.55</td>
<td>349.80</td>
<td>313.61</td>
<td>267.46</td>
</tr>
<tr>
<td>10% Spinach+5% Chickpea powders</td>
<td></td>
<td>368.20</td>
<td>342.40</td>
<td>325.20</td>
<td>268.60</td>
</tr>
<tr>
<td>L.S.D.</td>
<td>15.23</td>
<td>10.14</td>
<td>17.20</td>
<td>8.17</td>
<td></td>
</tr>
</tbody>
</table>

L.S.D. for interaction between time and treatments = 9.63

Effect of Diets on Relative Organs and Fat Weights in Obese Rats:

Table (3) shows effect of diets on organs (e.g., liver, kidney, spleen, heart and cecum) and epididymal fat pad weights in obese rats at the end of the experimental period relative to final body weight. Organs weight and epididymal fat weight for negative control obese rats were significantly different and lower compared with positive control obese rats except for cecum weight which was not significantly different. Relative cecum weight of rats fed on different diets was highest in spinach leaves + chickpea seeds fed rats followed by lentil seeds, spinach leaves and chickpea seeds, respectively, as a result of feeding on slowly digested and absorbed carbohydrates (low-GI foods) which delayed transit time and increased cecum weight. Relative weight of liver, kidney, spleen, heart and epididymal fat was reduced as a result of feeding on different diets by the same trend: lentil seeds < spinach leaves + chickpea seeds < spinach leaves < chickpea seeds. Short-term studies in humans and animals have provided evidence that a high-GI diet affects hunger and nutrient partitioning in a way that promotes body fat storage. Compared with rats fed amylose (a low-GI starch), those fed amylopectin (a high-GI starch), under nutrient and energy controlled conditions for 3-5 weeks, exhibited physiologic changes that favored fat deposition, including larger adipocyte diameter, increased glucose incorporation into lipids and greater fatty acid synthase in fat tissue (Ockene et al., 1999). By 7 weeks, animals fed a high-GI diet developed increased epididymal fat mass (Pawlak et al., 2001).
Because it has been shown that increased hepatic fat oxidation is associated with lower hunger and food intake. Freely fed diets without energy restriction resulted in a greater reduction in energy intake and weight gain rate. GI (amylopectin) diet group. These results clearly demonstrate that manipulating the type of carbohydrate in ways that would favor negative energy balance which were in good agreement with the present results (Pereira et al., 2004). Of particular interest was the observation that low–GI foods increased satiety, delayed return of hunger and decreased food intake explaining the reduction in body weight and fat pad weight in obese rats fed on those diets low in glycemic index. Ludwig, (2000) summarized 16 studies that examined the effects of glycemic index on hunger, and all but one demonstrated that low–GI foods increased satiety, delayed return of hunger and decreased food intake compared with high-GI foods in humans which induce a sequence of hormonal and metabolic changes that promote excessive food intake in obese subjects. The differential losses of body weight and fat during the weight–loss phase indicate that subjects in the reduced–GI group experienced greater negative energy balance than did those in the control group. This difference must be explained by greater energy expenditure, lower energy intake or some combination of these factors. The decline in resting energy efficiency during weight loss may be attenuated with a reduced–GI diet. These findings support the view that a reduced–GI diet may affect energy efficiency and partitioning in ways that would favor negative energy balance which were in good agreement with the present results (Pereira et al., 2004). Of particular interest was the observation that low–GI starch diet (amylose) resulted in greater weight loss than high–GI starch diet (amylopectin) during energy restriction. This can be attributed largely to the expansion of the cecum that was almost 4 times heavier (full) in rats fed the amylose diet (Higgins, 2004 and Keenan, 2006). The present results are in good agreement with the results reported by Aziz et al. (2009) who mentioned that, after feeding the high fat diet for 2 wk, a distinct phenotype of body weight became obvious. The weight gain of rats fed the control diet was lower than that of diet induced obesity. By the end of the 4 wk feeding period, diet induced obesity rats had gained 233 ± 3.6 g compared with 161 ± 3.7 g and 149 ± 8.1 g for diet resistant and control rats, respectively. Total cumulative energy intake of the high fat diet over the 4 wk feeding period was higher in diet induced obesity than in diet resistant rats. Rats fed the control diet had a lower total energy intake than diet induced obesity rats. There was an effect of the amount of energy consumed and an interaction between starch type and energy consumed on weight change and on total cumulative weight gain. Diet induced obesity rats in the low–GI (amylose) diet group gained significantly less weight and had greater cecum weights than those in the high–GI (amylopectin) diet group. These results clearly demonstrate that manipulating the type of carbohydrate in freely fed diets without energy restriction resulted in a greater reduction in energy intake and weight gain rate. Because it has been shown that increased hepatic fat oxidation is associated with lower hunger and food intake in animals and humans, the reduced fat oxidation associated with hyperinsulinemia may lead to an increase in food intake. It has been demonstrated that the 30 minutes postprandial insulin level is an important determinant of weight loss on a low-GI diet, suggesting that the lower the dietary glycemic index, the greater the benefit for those who are overweight ((McLaughlin et al., 2006 and Shaw et al., 2009).

Table 3: Effect of diets on relative organs and epididymal fat weights in obese rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Relative organs weight (g)</th>
<th>Epididymal fat weight (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative control</td>
<td>2.357*</td>
<td>0.310*</td>
</tr>
<tr>
<td>Positive control</td>
<td>2.516*</td>
<td>0.401*</td>
</tr>
<tr>
<td>10% Chickpea seeds powder</td>
<td>3.053*</td>
<td>0.525*</td>
</tr>
<tr>
<td>10% Lentil seeds powder</td>
<td>2.724*</td>
<td>0.426*</td>
</tr>
<tr>
<td>10% Spinach leaves powder</td>
<td>2.954*</td>
<td>0.486*</td>
</tr>
<tr>
<td>5% Spinach + 5% Chickpea seeds powder</td>
<td>2.770*</td>
<td>0.454*</td>
</tr>
</tbody>
</table>

Relative organs weight was calculated by dividing weight of organs / total final body weight × 100.

Effect of Diets on Food Intake, Corrected Weight Change and Energy Efficiency in Obese Rats:

Food intake (g/rat/day) was significantly increased for positive control rats fed on high fat diet compared with negative control rats and all other groups as shown in Fig. (1). Feeding obese rats on chickpea seeds powder resulted in non significant change in food intake compared with negative control rats, but they were significantly decreased compared with positive control rats. Food intake was significantly decreased for obese rats fed on lentil seeds powder, spinach leaves + chickpea seeds powders and spinach leaves powder, respectively. Corrected weight change and energy efficiency were significantly higher (positive) for positive control obese rats fed on high fat diet compared with all other groups. Feeding obese rats on experimental diets caused a negative balance in energy efficiency which was higher for lentil seeds fed rats followed by spinach leaves + chickpea seeds, spinach leaves and chickpea seeds fed obese rats, respectively. The same trend was also noticed for corrected weight change. These results indicate that feeding obese rats on experimental diets with low–GI foods caused reduction in food intake, weight change and energy efficiency which may help explaining the reduction in body weight and fat pad weight in obese rats fed on those diets low in glycemic index. Ludwig, (2000) summarized 16 studies that examined the effects of glycemic index on hunger, and all but one demonstrated that low–GI foods increased satiety, delayed return of hunger and decreased food intake compared with high-GI foods in humans which induce a sequence of hormonal and metabolic changes that promote excessive food intake in obese subjects. The differential losses of body weight and fat during the weight–loss phase indicate that subjects in the reduced–GI group experienced greater negative energy balance than did those in the control group. This difference must be explained by greater energy expenditure, lower energy intake or some combination of these factors. The decline in resting energy efficiency during weight loss may be attenuated with a reduced–GI diet. These findings support the view that a reduced–GI diet may affect energy efficiency and partitioning in ways that would favor negative energy balance which were in good agreement with the present results (Pereira et al., 2004). Of particular interest was the observation that low–GI starch diet (amylose) resulted in greater weight loss than high–GI starch diet (amylopectin) during energy restriction. This can be attributed largely to the expansion of the cecum that was almost 4 times heavier (full) in amylose than in amylopectin fed rats. The enlargement of the cecum is due to the fermentation of resistant starch present in the amylose diet (Higgins, 2004 and Keenan et al., 2006). The present results are in good agreement with the results reported by Aziz et al. (2009) who mentioned that, after feeding the high fat diet for 2 wk, a distinct phenotype of body weight became obvious. The weight gain of rats fed the control diet was lower than that of diet induced obesity. By the end of the 4 wk feeding period, diet induced obesity rats had gained 233 ± 3.6 g compared with 161 ± 3.7 g and 149 ± 8.1 g for diet resistant and control rats, respectively. Total cumulative energy intake of the high fat diet over the 4 wk feeding period was higher in diet induced obesity than in diet resistant rats. Rats fed the control diet had a lower total energy intake than diet induced obesity rats. There was an effect of the amount of energy consumed and an interaction between starch type and energy consumed on weight change and on total cumulative weight gain. Diet induced obesity rats in the low–GI (amylose) diet group gained significantly less weight and had greater cecum weights than those in the high–GI (amylopectin) diet group. These results clearly demonstrate that manipulating the type of carbohydrate in freely fed diets without energy restriction resulted in a greater reduction in energy intake and weight gain rate. Because it has been shown that increased hepatic fat oxidation is associated with lower hunger and food intake in animals and humans, the reduced fat oxidation associated with hyperinsulinemia may lead to an increase in food intake. It has been demonstrated that the 30 minutes postprandial insulin level is an important determinant of weight loss on a low-GI diet, suggesting that the lower the dietary glycemic index, the greater the benefit for those who are overweight ((McLaughlin et al., 2006 and Shaw et al., 2009).
Fig. 1: Effect of diets on food intake (A), corrected weight change (B) and energy efficiency (C) in obese rats.

1 Corrected weight gain = final (body weight - full cecum) – initial body weight.

2 Energy efficiency (EE) = gain in total body weight divided by energy consumed over the corresponding period as reported by Levin et al. (1997).
Effect of Diets on Glucose Homeostasis in Obese Rats:

Effect of diets substituted with different low glycemic index foods on blood glucose and serum insulin levels is shown in Table (4). Blood glucose level was significantly increased by 1.13 fold in positive control obese rats fed on high fat diet compared with negative control rats at the end of the experimental period. The resistant starch content of the low–GI diet was higher than high GL diet. At the end of the experiment, blood glucose levels in obese rats fed on different low–GI diets decreased significantly compared with negative control obese rats except for obese rats fed on chickpea seeds powder which were not significantly different compared with negative control rats. The other groups of obese rats fed on low-GI diets were not significantly different compared with each other. The decrease in blood glucose levels as a result of feeding on different diets was 14.39%, 27.61%, 24.23% and 25.55% for chickpea seeds, lentil seeds, spinach leaves and spinach leaves + chickpea seeds diets, respectively. Serum insulin level of positive control obese rats fed on high fat diet was significantly higher compared with negative control obese rats and all other groups at the end of the experiment. Feeding obese rats on different diets resulted in a significant decrease in serum insulin levels and the groups were significantly different compared with each other. The reduction in insulin levels was as follows: 34.55% for chickpea seeds, 55.76% for lentil seeds, 45.49% for spinach leaves and 51.03% for spinach leaves + chickpea seeds diets. The highest reduction in blood glucose and serum insulin levels was achieved by feeding on lentil seeds followed by spinach leaves + chickpea seeds, spinach leaves and chickpea seeds diets, respectively. These results are in good agreement with the results mentioned by Brownley et al. (2012) who found that, compared with the high glycemic load meal, the low glycemic load meal was associated with lower glucose and insulin concentrations. In the present study, we used homeostasis model assessment (HOMA) to estimate relative insulin resistance and pancreatic β-cell function with higher values indicating lower insulin sensitivity and higher insulin resistance. Insulin resistance as a factor for metabolic syndrome, has been linked to many important consequences including type 2 diabetes, hypertension and dyslipidemia. Although there are some genetic causes for insulin resistance, the most common cause is an excess of nutrition. Both excess glucose and excess fat can cause insulin resistance in muscle and fat tissues and excess fat can cause insulin resistance in the liver (Sinha et al., 2002 and Jimenez-Cruz et al., 2003). From Table (4) it could be observed that, insulin resistance as calculated by HOMA index was increased significantly less in the experimental diet groups compared with positive control obese rats fed on high fat diet. Relative insulin resistance index (HOMA-R) was 1.8, 3.1, 2.4 and 2.7 fold less for low-GI diets than positive control rats and obese rats fed on chickpea seeds powder was not significantly different compared with negative control obese rats. The other diets were not significantly different compared with each other. For pancreatic β-cell function, the index was not significantly different for negative and positive control obese rats but for different diets with low-GI foods it was significantly different except for lentil seeds and spinach leaves + chickpea seeds powders which was not different. The present results are in accordance with the results reported by Fajcsak et al. (2008) who found that, besides the reduction in fasting glucose, fasting insulin values were reduced by 15% and there was a favorable reduction in HOMA-IR value after the 6-week low–GL diet. However, there was a strong negative correlation among fasting glucose and insulin levels at baseline and in the magnitude of change after the study. The higher was the baseline value the more it decreased to the end of the study. Scribner et al. (2008) found that, fasted blood glucose and plasma insulin were significantly higher for the rapidly absorbed carbohydrate group (high-GI) compared with the slowly absorbed carbohydrate group (low-GI). Insulin resistance was 2.2 fold higher for the high–GI than low–GI group, had lower fat oxidation and 40% greater body fat although net energy intake did not differ between groups. The postprandial hyperglycemia on a high–GI diet increases insulin secretion and higher insulin levels would promote glucose uptake in insulin–sensitive tissues and inhibit lipolysis in adipose tissue. These metabolic events would acutely favor oxidation of carbohydrate rather than fat and chronically lead to accumulation of body fat. In rats, the chronic consumption of an low–GI diet has been shown to increase insulin–stimulated glucose oxidation in isolated adipocytes, but to also reduce de novo lipogenesis and adipocyte diameter. A 16–week low–GI diet also ameliorates whole–body insulin resistance, thus it is hypothesized that the chronic consumption of low–GI foods could decrease adiposity and increase insulin sensitivity (Kabir et al., 1998a). The resistant starch content of the low–GI diet was higher than that of the high–GI diet which is carbohydrate that escapes digestion in the small intestine, thereby altering the colonic environment due to increased fermentation, subsequently influencing microbial flora composition and free fatty acid production. Differences in colonic bacteria have been documented in lean and obese humans and animals. These differences may play a causal role in obesity through novel mechanisms such as direct effects on fat cell metabolism. Thus, it is possible that the physiological effects of the diets were mediated not only by actions in the small intestine but also by actions in the large intestine (DiBaise et al., 2008). In insulin-resistant persons consuming high–GI foods, the liver is simultaneously exposed to hyperglycemia (deriving from higher glucose availability) and hyperinsulinemia (deriving from both hyperglycemia and insulin resistance). This combined exposure may up-regulate de novo lipogenesis and inhibit non-esterified fatty acids oxidation (Silvia et al., 2006). Hyperinsulinemia, because of its lipogenic effect, has been linked to obesity. Rizkalla et al. (2004) showed that, low-GI diets improved whole–body insulin sensitivity in individuals with type 2 diabetes. A
reduced-GI diet is likely to produce a lower average daylong insulin concentration. This is especially true for persons with insulin resistance and compensatory hyperinsulinemia, both of which are characteristic features of obesity. In persons with excess adiposity, the ability of insulin to promote glycogenesis is impaired to a greater extent than is its ability to promote glucose oxidation. In an insulin-resistant person, a high-GI diet would be expected to produce extended periods of hyperinsulinemia that would, in turn, increase whole-body carbohydrate oxidation and depress fat oxidation (Boden et al., 2005). Aston et al. (2007) showed that a weight-maintaining low-GI diet consumed over 12 weeks was associated with higher insulin sensitivity than a macronutrient and fiber-matched high-GI diet in overweight young adults and by reducing insulinemia low-GI diet may provide greater access to fatty acids as a source of fuel, promoting greater fat oxidation. The decrease in fasting blood glucose may be due to a slight improvement in insulin sensitivity when subjects consumed a low-GI breakfast compared with a high-GI breakfast for 3 weeks, an effect attributed to the increased insulin-stimulated glucose uptake by adipocytes (Pal et al., 2008).

**Table 4: Effect of diets on glucose homeostasis in obese rats.**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Glucose (mmol/L)</th>
<th>Insulin (mU/L)</th>
<th>HOMA-R</th>
<th>HOMA-β</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Zero</td>
<td>End</td>
<td>Zero</td>
<td>End</td>
</tr>
<tr>
<td>Negative control</td>
<td>5.66⁺</td>
<td>6.03⁺</td>
<td>34.07⁺</td>
<td>36.25⁺</td>
</tr>
<tr>
<td>Positive control</td>
<td>5.75⁺</td>
<td>6.8₁⁺</td>
<td>35.4₁⁺</td>
<td>48.3₀⁺</td>
</tr>
<tr>
<td>10% Chickpea seeds powder</td>
<td>5.60⁻</td>
<td>5.8₃⁻</td>
<td>34.8₀⁺</td>
<td>31.6₁⁻</td>
</tr>
<tr>
<td>10% Lentil seeds powder</td>
<td>5.₇⁸⁻</td>
<td>4.₉₂⁻</td>
<td>36.₁⁹⁺</td>
<td>21.₇⁹⁻</td>
</tr>
<tr>
<td>10% Spinach leaves powder</td>
<td>5.₆₇⁻</td>
<td>5.₁⁶⁻</td>
<td>35.₆₈⁻</td>
<td>2₆.₃₃⁻</td>
</tr>
<tr>
<td>5% Spinach +5%Chickpea powders</td>
<td>5.₇₄⁻</td>
<td>5.₀⁷⁻</td>
<td>3₄.₆₃⁻</td>
<td>2₃.₆₅⁻</td>
</tr>
<tr>
<td>L.S.D.</td>
<td>0.₄₃⁻</td>
<td>2.₂₆⁻</td>
<td></td>
<td>2.₁₁⁻</td>
</tr>
</tbody>
</table>

**Effect of Diets on Oxidative Status in Obese Rats:**

Effect of different diets substituted with low-GI foods on glutathione peroxidase (GPx), superoxide dismutase (SOD) enzymes and malondialdehyde (MDA) levels in serum of obese rats is shown in Fig. (2). It was found that fatty acids released from visceral adipose tissue appear to stimulate the production of reactive oxygen species (oxidative stress), which are associated with a reduction in insulin–stimulated glucose transport in obese individuals. Moreover, as the degree of obesity increases, there is a corresponding increase in the levels of inflammatory factors (Evans et al., 2003 and Weiss et al., 2004). Excessive postprandial glycemia could decrease blood HDL–cholesterol, increase triglyceridemia and also be directly toxic by increasing protein glycation, generating oxidative stress and impaired endothelial function (Gavin, 2001). Most studies reveal a modest effect of dietary composition on some inflammatory markers in free-living adults. Significant dietary influences have been established for glycemic index and load, fiber, fatty acids composition, magnesium and flavonoids found in fruits, vegetables, legumes and grains which has shown anti-inflammatory effects (Leo, 2010). Excessive postprandial glycemia could decrease blood HDL–cholesterol, increase triglyceridemia and also be directly toxic by increasing protein glycation, generating oxidative stress and impaired endothelial function (Gavin, 2001). Most studies reveal a modest effect of dietary composition on some inflammatory markers in free-living adults. Significant dietary influences have been established for glycemic index and load, fiber, fatty acids composition, magnesium and flavonoids found in fruits, vegetables, legumes and grains which has shown anti-inflammatory effects (Leo, 2010). From Fig. (2) it could be noticed that, feeding obese rats on different low-GI diets caused significant increase in SOD and GPx enzymes activities compared with negative and positive control obese rats at the end of the experimental period. The activity of both enzymes was significantly lower for positive control obese rats compared with negative control rats as a result of feeding on high fat diet. The increase in SOD and GPx activities was in the same trend for different diets substituted with low-GI foods: lentil seeds > spinach leaves + chickpea seeds > spinach leaves > chickpea seeds. On the contrary, MDA level was significantly higher in positive control obese rats compared with negative control rats and all other obese rats fed on different diets at the end of the experiment. Feeding obese rats on different low-GI diets caused significant reduction in MDA level compared with negative and positive control obese rats. The highest reduction in MDA level was achieved by feeding obese rats on lentil seeds powder followed by spinach leaves + chickpea seeds powders. These results could be attributed to the high antioxidant activity of the studied low-GI foods. Pulses such as mung beans, chickpeas and garden peas contained between 0.12 and 0.35 mmol antioxidants per 100g. The antioxidant content increased in products such as carrots, spinach, broccoli and tomatoes during microwave cooking, steaming and boiling (Halvorsen et al., 2006). Obesity promotes oxidative stress in adipose tissue and is mechanistically linked to adipose tissue inflammation and the metabolic complications of obesity. Increased adipocyte death in obesity is suggested to reflect the elevated levels of cytotoxic stressors reported for obese adipose tissue. Higher frequencies of dead adipocytes in mice fed the high fat diet were coincident with lower gene expression of GPx3, an oxidative stress–sensitive gene in adipose tissue. Feeding these mice on diet supplemented with whole blueberry powder protects against adipose tissue inflammation and insulin resistance (Lionetti et al., 2009 and DeFuria et al., 2009). Feeding on low-GI diet for one week reduces oxidative stress more so than an high-GI diet, yet the data fail to differentially alter insulin sensitivity, suggesting that indeed longer-term interventions are required (Botero et al., 2009).
Fig. 2: Effect of diets on superoxide dismutase (A), glutathione peroxidase (B) and malondialdehyde (C) in obese rats.

Effect of Diets on Serum Lipids Profile in Obese Rats:

Feeding obese rats on high fat diet (positive control diet) resulted in a significant increase in serum triglycerides level compared with negative control obese rats (Table 5). Feeding obese rats on different low-GI diets showed a significant reduction in serum triglycerides levels compared with positive control obese rats at the end of the experimental period except for diet substituted with 10% chickpea seeds powder which was not significantly different. Serum triglycerides levels appeared to be still elevated compared with negative control obese rats because of feeding on high fat diets. Diet substituted with 10% lentil seeds powder caused the highest reduction in triglycerides level compared with other diets. Total cholesterol (TC), low-density lipoprotein
cholesterol (LDL) and very low-density lipoprotein cholesterol (VLDL) levels increased significantly in obese rats fed on high fat diet (positive control) compared with negative control obese rats. Feeding obese rats on low–GI diets caused significant reduction in the levels of lipids profile (TC, LDL and VLDL) compared with positive control obese rats. The highest reduction was achieved by feeding on 10% lentil seeds followed by 5% spinach leaves + 5% chickpea seeds, 10% spinach leaves and 10% chickpea seeds, respectively. Concerning high-density lipoprotein cholesterol (HDL) level, it was noticed from Table (5) that, HDL-cholesterol level significantly decreased as a result of feeding on high fat diet but it was increased significantly after feeding on different low–GI diets compared with negative and positive control obese rats. The highest increase was recorded for obese rats fed on 10% lentil seeds powder followed by 5% spinach leaves + 5% chickpea seeds powders. Sloth et al. (2004) found that, 10-week of the low–GI diet resulted in a 10% decrease in LDL-cholesterol whereas a small increase (2%) was seen after the high–GI diet. A tendency to a difference between groups in total cholesterol was seen as 7.2% and 1.9% decreases in the low-GI and high-GI diets, respectively. McMillan–Price et al. (2006) reported a decrease of 0.23 mmol/L (8.9 mg/dl) in total cholesterol and of 0.21 mmol/L (8.1 mg/dl) in LDL-cholesterol in 27 subjects who consumed a low–GI diet compared with a high–GI diet. However, fiber intake in the low-GI diet group was 30 g/d compared with 23 g/d in the high–GI group, which was mainly due to increased consumption of legumes and products rich in whole grains. Although these products do not provide only soluble fibers, this may explain at least a part of the effect on serum lipoprotein profile. It can be deduced that, a difference in soluble fiber intake of 7 g/d may cause a difference of about 0.28 mmol/L (10.8 mg/dl) in total cholesterol and of 0.21 mmol/L (8.1 mg/dl) in LDL-cholesterol. Although evidence suggests that low-GI and GL diets may indirectly reduce the risk of heart disease by modifying risk factors such as diabetes and obesity, evidence suggests that these diets may have additional protective effects against heart disease by modifying serum lipid levels, an effect that has not been shown with most oral antihyperglycemic drugs. However, dietary GI was inversely associated with serum HDL–cholesterol levels, caused reductions in C-reactive protein which is a marker of inflammation, serum triglycerides and LDL-cholesterol (DeRougemont et al., 2007 and Patel et al., 2008). Increasing dietary GI has been independently linked to higher prevalence of insulin resistance, the metabolic syndrome, fatty liver and metabolic risk factors including triglycerides and HDL–cholesterol (McKeown et al., 2004). Clinical studies indicate that highly digestible carbohydrate diets (high–GI) may lead to an elevation in fasting plasma triacylglycerol concentrations as a result of the accumulation of hepatic VLDL and chylomicron remnants due to altered lipoprotein secretion or clearance. Elevated fasting triacylglycerolemia and associated low HDL-cholesterol are risk factors for heart disease. In a study conducted in obese young adults over 12 months, the experimental low–GI foods caused a significant mean decline in plasma triacylglycerols than did the conventional diet (-37.2% and –19.1%, respectively). Another study using a low–GI diet in healthy French men demonstrated a reduction of fat mass and triacylglycerol after a 5-week period. Dietary glucose may stimulate intestinal resecretion and the occurrence in plasma of triacylglycerols or cholesterol in triacylglycerol–rich particles (Play et al., 2003 and Ebbeling et al., 2005). It is well documented that both excess body fat and resistance to the action of insulin impair the suppression of circulating non–esterified fatty acids in the postprandial state, which favors a greater NEFA influx into the hepatocyte and subsequent synthesis of triacylglycerols. In addition, the metabolic pathway controlling mitochondrial fat oxidation is down regulated in the presence of insulin resistance, which further contributes to intracellular fat accretion. Low–dietary GI and GL seem to be linked to favorable lipid profiles and lower levels of C-reactive protein only in overweight and obese persons, which suggests that the metabolic effects of dietary carbohydrates may be particularly important in insulin-resistant persons (Browning and Horton, 2004). However, some investigators noted favorable differences in fasting triacylglycerol and HDL–cholesterol concentrations and found a positive effect of lowering the GI or GL on HDL-cholesterol and it can be estimated that, by increasing the GL by 30 units, HDL–cholesterol will decrease by 0.08 mmol/L (3.1 mg/dl) (Foster et al., 2003 and Cuberston et al., 2009). Insulin has a stimulatory effect on VLDL synthesis. Therefore, a smaller integrated insulin response may reduce VLDL synthesis and entry into the circulation. In the absence of changes in the rates of VLDL conversion to LDL or of LDL removal from the circulation or both, a reduction in the concentration of LDL-cholesterol would be expected (Reaven, 2003).

Table 5: Effect of diets on lipids profile in obese rats (mg/dl).

<table>
<thead>
<tr>
<th>Groups</th>
<th>Triglycerides</th>
<th>Total cholesterol</th>
<th>HDL-cholesterol</th>
<th>LDL-cholesterol</th>
<th>VLDL-cholesterol</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Zero</td>
<td>End n</td>
<td>Zero</td>
<td>End</td>
<td>Zero</td>
</tr>
<tr>
<td>Negative control</td>
<td>85.60 A</td>
<td>89.67 B</td>
<td>215.19 A</td>
<td>225.74 A</td>
<td>63.34 B</td>
</tr>
<tr>
<td>Positive control</td>
<td>85.46 A</td>
<td>126.34 B</td>
<td>219.30 A</td>
<td>264.16 B</td>
<td>64.30 B</td>
</tr>
<tr>
<td>10% Chickpea seeds</td>
<td>87.55 A</td>
<td>120.41 B</td>
<td>218.94 A</td>
<td>192.13 B</td>
<td>63.75 B</td>
</tr>
<tr>
<td>10% Lentil seeds</td>
<td>85.93 A</td>
<td>101.84 C</td>
<td>216.80 A</td>
<td>171.63 C</td>
<td>62.56 B</td>
</tr>
</tbody>
</table>

*Note: Values are mean ± SE. Significant differences at p < 0.05 by Duncan’s multiple range test. A, B, C, D, E and F indicate significant differences among the groups.
Lipoprotein–catabolizing enzymes. Insulin regulation of adipose tissue lipase enzyme has been demonstrated in starch diet may also increase circulating lipid levels through an effect on lipase enzyme, one of the major nutritional controls to achieve lipid homeostasis. In addition to an effect on lipid biosynthesis, dietary high–GI factors involved in the regulation of lipogenesis such as insulin, glucagon and thyroid hormone interact with structural components. This pathway is regulated by complex nutritional and hormonal controls. Hormonal lipase in both normal and noninsulin–dependent diabetic rats was studied by Hillgartner.

Low–GI starch diets on some lipid storage related enzymes regulated by insulin, i.e., fatty acid synthase and levels, which could have induced a reduction in fat depot (Boivin et al., 1994). The mismatch between hypothalamic gene expression and circulating leptin levels are suggestive of central leptin resistance. Defective leptin signaling is likely mediated by increases in hypothalamic gene expression, which contributes to leptin resistance in mice and rats fed a high–fat diet (White et al., 2009).

Lipase enzyme activity is important because of its ability to hydrolyze triglycerides. The metabolic activities of human adipocytes has focused on plasma triacylglycerol hydrolysis and the uptake of fatty acids in adipose tissue by lipase enzyme. Alterations in insulin sensitivity might alter lipid metabolism through the action of insulin on lipase. Actually, the lipase enzyme level in adipose tissue is positively correlated with tissue by lipase enzyme. Alterations in insulin sensitivity might alter lipid metabolism through the action of insulin on lipase. Actually, the lipase enzyme level in adipose tissue is positively correlated with

Effect of Diets on Serum Leptin, Lipase, Phospholipids and Total Lipids Concentrations in Obese Rats:

Data presented in Table (6) show effect of different diets substituted with low–GI foods on serum leptin, lipase enzyme activity, phospholipids and total lipids concentrations in obese rats. Serum leptin levels in obese rats were significantly unchanged for negative control obese rats from zero time to the end of the study period. Leptin level for positive control obese rats fed on high fat diet was significantly increased (28.62%) compared with zero time level and it was significantly higher than all other groups at the end of the experimental period. Feeding obese rats on different low–GI diets caused a significant decrease in serum leptin levels compared with negative and positive control rats. The highest decrease was achieved by feeding on lentil seeds powder (45.36%) followed by spinach leaves + chickpea seeds powders (43.26%). The four diets were not significantly different for serum leptin levels compared with each other. Concerning lipase enzyme activity in serum of obese rats, it was found that there is a significant decrease for positive control obese rats at the end of the study period compared with lipase activity for negative control rats both at zero time and the end. Feeding on chickpea seeds, spinach leaves, spinach leaves + chickpea seeds and lentil seeds caused a gradual increase in serum lipase enzyme activity by 5.99%, 11.13%, 17.55% and 26.52%, respectively, compared with negative control obese rats at zero time. From data presented in Table (6) it could be observed that, phospholipids and total lipids concentrations in serum of positive control obese rats were significantly decreased compared with negative control rats at the end of the experimental period. A significant increase in serum phospholipids and total lipids concentrations of obese rats was observed after the low–GI diets feeding with the highest increase achieved by using lentil seeds powder (20.35% and 22.62%) for phospholipids and total lipids levels, respectively) compared with negative control obese rats at zero time. Also, there is a gradual increase in concentrations of phospholipids and total lipids by using chickpea seeds, spinach leaves and spinach leaves + chickpea seeds powders, respectively. Caro et al. (1996) found that, serum leptin decreased more rapidly and to a greater extent during the low–GI diet than during the high–GI diet. By day 6, leptin had decreased by 50.0 ± 5.5% from baseline with the low–GI diet. The decrease in leptin levels may be explained by the lower insulin concentrations associated with low–GI diets, because insulin is a leptin secretagogue, or by decreased adipocyte glucose metabolism. A positive association between carbohydrate consumption and leptin concentration during energy restriction was observed. The lower leptin concentration with the low-GI diet occurred without evidence of increased hunger (ad libitum food intake was actually lower with this diet), suggesting a functional improvement in the leptin resistance associated with obesity (Sivitz et al., 1998). Diet had significant effects on changes in leptin levels, which decreased more in high carbohydrate, low-GI diet, with a significant interaction between GI and carbohydrate content. Hence, lowering the GI resulted in a larger decrease in leptin levels when the carbohydrate intake was high. However, on an individual basis, the absolute decrease in leptin levels correlated significantly with change in fat mass, with no additional effect of GI or carbohydrate content. Changes in fat mass were also correlated with change in fasting insulin level and changes in insulin sensitivity (McMillan-Price et al., 2006). Weight loss resulted in lower serum leptin concentrations and there were effects of starch type and amount of energy consumed as well as an interaction between them on serum insulin and leptin concentrations in food–deprived rats. The mismatch between hypothalamic gene expression and circulating leptin levels are suggestive of central leptin resistance. Defective leptin signaling is likely mediated by increases in hypothalamic gene expression, which contributes to leptin resistance in mice and rats fed a high–fat diet (White et al., 2009).

<table>
<thead>
<tr>
<th>Diet</th>
<th>Serum Leptin (µg/ml)</th>
<th>Serum Lipase (U/ml)</th>
<th>Serum Phospholipids (mg/dl)</th>
<th>Total Lipids (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>8.83±0.12</td>
<td>21.65±1.23</td>
<td>84.72±2.34</td>
<td>112.25±0.89</td>
</tr>
<tr>
<td>Low-GI</td>
<td>7.13±0.08</td>
<td>21.03±1.12</td>
<td>88.33±2.45</td>
<td>119.67±0.92</td>
</tr>
<tr>
<td>Spinach leaves</td>
<td>6.63±0.06</td>
<td>20.55±1.08</td>
<td>84.72±2.34</td>
<td>112.25±0.89</td>
</tr>
<tr>
<td>Spinach leaves + Chickpea seeds</td>
<td>3.11±0.03</td>
<td>7.13±0.08</td>
<td>88.33±2.45</td>
<td>119.67±0.92</td>
</tr>
<tr>
<td>Lentil seeds</td>
<td>2.03±0.02</td>
<td>17.67±1.02</td>
<td>84.72±2.34</td>
<td>112.25±0.89</td>
</tr>
</tbody>
</table>

Data presented in Table (6) show effect of different diets substituted with low–GI foods on serum leptin, lipase enzyme activity, phospholipids and total lipids concentrations in obese rats. Serum leptin levels in obese rats were significantly unchanged for negative control obese rats from zero time to the end of the study period. Leptin level for positive control obese rats fed on high fat diet was significantly increased (28.62%) compared with zero time level and it was significantly higher than all other groups at the end of the experimental period. Feeding obese rats on different low–GI diets caused a significant decrease in serum leptin levels compared with negative and positive control rats. The highest decrease was achieved by feeding on lentil seeds powder (45.36%) followed by spinach leaves + chickpea seeds powders (43.26%). The four diets were not significantly different for serum leptin levels compared with each other. 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Changes in fat mass were also correlated with change in fasting insulin level and changes in insulin sensitivity (McMillan-Price et al., 2006). Weight loss resulted in lower serum leptin concentrations and there were effects of starch type and amount of energy consumed as well as an interaction between them on serum insulin and leptin concentrations in food–deprived rats. The mismatch between hypothalamic gene expression and circulating leptin levels are suggestive of central leptin resistance. Defective leptin signaling is likely mediated by increases in hypothalamic gene expression, which contributes to leptin resistance in mice and rats fed a high–fat diet (White et al., 2009). Lipase enzyme activity is important because of its ability to hydrolyze triglycerides. The metabolic activities of human adipocytes has focused on plasma triacylglycerol hydrolysis and the uptake of fatty acids in adipose tissue by lipase enzyme. Alterations in insulin sensitivity might alter lipid metabolism through the action of insulin on lipase. Actually, the lipase enzyme level in adipose tissue is positively correlated with hyperinsulinemia. Consistent with the reduction of postprandial insulinemia and triacylglycerol, after an low-GI diet, there was a significant reduction in lipase enzyme expression in adipose tissue and an increase in serum levels, which could have induced a reduction in fat depot (Boivin et al., 1994). The effect of a high against a low–GI starch diets on some lipid storage related enzymes regulated by insulin, i.e., fatty acid synthase and lipase in both normal and noninsulin–dependent diabetic rats was studied by Hillgartner et al. (1995) who found that, fatty acid synthesis is a major metabolic pathway for the provision of energy reserves and cellular structural components. This pathway is regulated by complex nutritional and hormonal controls. Hormonal factors involved in the regulation of lipogenesis such as insulin, glucagon and thyroid hormone interact with nutritional controls to achieve lipid homeostasis. In addition to an effect on lipid biosynthesis, dietary high–GI starch diet may also increase circulating lipid levels through an effect on lipase enzyme, one of the major lipoprotein–catabolizing enzymes. Insulin regulation of adipose tissue lipase enzyme has been demonstrated in
vitro and insulin is known to stimulate lipoprotein lipase activity in adipose tissue, an effect blunted in glucose-intolerant, obese subjects (Coppack et al., 1992). The presence of large adipocytes produced by consuming high-GI diets reduced lipase enzyme activity because these cells were less sensitive to insulin effect (Kabir et al., 1998b). During the high-GI period, a more insulin-resistant state than the low-GI period, the ability of insulin to suppress fatty acid release from adipose tissue might be impaired. Moreover, the pathway of fatty acid trapping (adipocyte uptake of fatty acids liberated from plasma triacylglycerol by lipase enzyme in adipose tissue) could also be defective, therefore, adding to impaired buffering of fatty acids and their accumulation in the circulation (McGarry, 2002). The study by Kallo et al. (2007) makes a significant contribution to the potentially major effects of dietary composition on the hormone-sensitive lipase which has been proposed to affect body weight and metabolic variables. Phospholipids concentrations did not differ in normal and diabetic rats fed on waxy corn starch (high-GI) or mung bean starch (low-GI) diets, thus phospholipids were not significantly affected by diet treatment. Phospholipids are substances composed of lecithin, choline and inositol which help transport and metabolize fatty acids and cholesterol. This make phospholipids ideal in protecting against cardiovascular disease (Kabir et al., 1998a and Hongu and Sachan, 2000). Total lipids composed of glycerides, phospholipids, free fatty acids and unsaponifiable matters including sterols, hydrocarbons, tocopherols and pro-vitamins E and A. Phospholipids and non-specific esterases must be at optimal contents and a significant increase in total lipids in serum indicates beneficial effects on health (Ficek and Stankiewicz, 1987).

Table 6: Effect of diets on leptin, lipase, phospholipids and total lipids concentrations in obese rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Leptin (µg/L)</th>
<th>Lipase (U/L)</th>
<th>Phospholipids (mg/dl)</th>
<th>Total lipids (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zero</td>
<td>12.20</td>
<td>5.91</td>
<td>36.93</td>
<td>377.80</td>
</tr>
<tr>
<td>End</td>
<td>13.87</td>
<td>4.62</td>
<td>35.50</td>
<td>370.30</td>
</tr>
<tr>
<td>Positive control</td>
<td>12.93</td>
<td>5.43</td>
<td>36.65</td>
<td>375.30</td>
</tr>
<tr>
<td>10% Chickpea seeds</td>
<td>14.19</td>
<td>5.91</td>
<td>36.93</td>
<td>377.80</td>
</tr>
<tr>
<td>10% Lentil seeds</td>
<td>13.86</td>
<td>5.43</td>
<td>36.65</td>
<td>375.30</td>
</tr>
<tr>
<td>10% Spinach leaves</td>
<td>13.06</td>
<td>5.43</td>
<td>36.65</td>
<td>375.30</td>
</tr>
<tr>
<td>5%Spinach+5%Chickpea</td>
<td>13.06</td>
<td>5.43</td>
<td>36.65</td>
<td>375.30</td>
</tr>
<tr>
<td>Negative control</td>
<td>12.20</td>
<td>5.91</td>
<td>36.93</td>
<td>377.80</td>
</tr>
<tr>
<td>Zero</td>
<td>13.87</td>
<td>4.62</td>
<td>35.50</td>
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</tr>
</tbody>
</table>

From the above mentioned results it could be concluded that, foods which had a low-glycemic response could be useful in reducing body weight, improving some metabolic parameters and lipids profile. Using lentil seeds achieved the highest improvement in these parameters followed by spinach leaves + chickpea seeds. Cheap ingredients which used in traditional Egyptian diets such as lentil, chickpea and spinach showed promising benefits as a low-glycemic index materials and could improve human health.

References


