

Revisión

Effect of the glycemic index on lipid oxidation and body composition

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Abstract

Several studies have been conducted to evaluate the effects of the consumption of the slowly absorbed carbohydrates in a low glycemic index (GI) diet on fat oxidation, in order to obtain dietetic treatment that can favor the achievement of an adequate body composition. Therefore, the purpose of this study was to analyze studies in which the role of low GI diets on body composition, with emphasis on fat oxidation. An internet search for articles, in English or Portuguese, published since 1995, was conducted using the following key words: glycemic index, glycemic load, glycaemic index, glycaemic load, body fat, body composition, fat oxidation. Papers that described animals or humans clinical trials were selected. Data were collected from Web of Science, Science Direct, Pubmed. It was verified that the results of the majority of the analyzed studies indicated that low GI diets lead to a lower insulin response, increasing body fat oxidation. These results indicate that the consumption of low GI diet can be an important strategy to be used for the prevention and control of obesity and chronic diseases associated to it.

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Key words: *Glycemic index. Glycemic load. Insulinemia. Body fat.*

EFECTO DEL ÍNDICE GLUCÉMICO SOBRE LA OXIDACIÓN LIPÍDICA Y LA COMPOSICIÓN CORPORAL

Resumen

A lo largo de los años ha realizado diferentes estudios con el objetivo de evaluar los efectos fisiológicos resultantes del consumo de carbohidratos de lenta digestión, los llamados de bajo índice glucémico (IG) y sus efectos en la oxidación de la grasa, en el intento de encontrar estrategias dietéticas que faciliten la obtención de una composición corporal satisfactoria. De acuerdo a lo expuesto, el objetivo de la investigación fue analizar estudios en que se haya evaluado en qué medida el consumo de una dieta con bajo IG afecta a la composición corporal, dando énfasis al efecto de la oxidación de la grasa. La investigación se hizo por medio de búsqueda en Internet de artículos en inglés y/o portugués, publicados desde 1995, y para eso se usó las palabras llave *glycemic index, glycemic load, glycaemic index, glycaemic load, body fat, body composition, fat oxidation*. Fueron incluidos artículos que evaluaban ensayos clínicos en animales y/o humanos. Los datos fueron recogidos y estudios utilizando datos encontrados en los sitios: *Web of Science, Science Direct y Pubmed*. Se averiguó que los resultados en la mayoría de los estudios señalan que el consumo de una dieta de bajo IG se produce una baja respuesta insulínica, aumentando la oxidación de la grasa corporal. Estos resultados indican que la ingestión de dieta de bajo IG es una estrategia importante para ser utilizada en la prevención y control de la obesidad y también de las enfermedades crónicas a ella asociada.

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Palabras clave: *Índice glucémico. Potencialidad glucente. Carga glucémica. Insulinemia. Grasa corporal.*

Abbreviations

AUC: Area Under the Curve.
WC: Waist Circumference.

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GL: Glycemic Load.
CHO: Carbohydrate.
DCV: Cardiovascular Diseases.
DEXA: Dual Energy X-ray Absorptiometry.
G: Glucose
IBGE: Instituto Brasileiro de Geografia e Estatística.
GI: Glycemic Index.
BMI: Body Mass Index.
WHO: World Health Organization.
BW: Body Weight.
PNAD: Brazilian Research of Domiciliate Sampling.
FFQ: Food Frequency Questionnaire.
RQ: Respiratory Quotient.

IR: Insulin Resistance.
IS: Insulin Sensitivity.
ITTG: Intravenous Tolerance Test to Glucose.

Introduction

There are currently more than one billion overweight adults in the world and, at least, 300 million are obese.¹ The obesity results in an indirect cost, which is represented by the loss of working days, doctors' appointments, and the cost of disability, premature deaths and increased risk of outbreak of diseases such as diabetes and cardiovascular disease (CVD). There is also the direct cost represented by health professional cares, hospital and home attendances, as well as weight reduction drug treatment. According to the World Health Organization (WHO), the obesity indirect cost is higher than the direct cost.² This situation is aggravating because obesity is appearing much earlier in the lifetime.

In Brazil, deaths caused by CVD, cancer and diabetes have increased from 34.4% in 1979 to 4.5% in 1998, and 48.3% in 2003. From 40 to 90% of such deaths could be potentially avoided through the ingestion of a nutritionally balanced diet.³ The results of the National Research for Home Sampling (NRHS), conducted in 2003 by the Brazilian Institute of Geography and Statistics, pointed out that 29.9% of the population had at least one type of non-transmissible chronic disease.⁴

The increase in the consumption of high energy dense diets that are rich in saturated fats and sugars, associated with low physical activity^{2,3} are the main causes of overweight. However, it has been verified that a reduction on fat consumption leads to an increase in carbohydrate (CHO) ingestion, which also induces an increase in adiposity.⁵⁻⁸

CHO is the main source of energy in human diet. According to the size of its molecule, CHO can be classified as simple or complex. However, this classification does not reflect the physiological effect of CHO on blood glucose.⁹ Thus, Jenkins and collaborators suggested the use of glycemic index (GI) as an indicator of the ability of a CHO food source to raise blood glucose levels after its consumption.¹⁰ The GI is defined as the incremental area under the blood glucose response curve after the consumption of a 50g CHO portion of a test food expressed as a percent of the response to the same amount of CHO from a standard food taken by the same subject.⁹ Although GI reflects the quality of the CHO it does not relate to the amount of CHO in the diet. Since the quantity of carbohydrate is also an important determinant of postprandial glucose increases, the concept of the glycemic load (GL) was developed. The GL is the product of a food's GI by its total available carbohydrate content.¹⁰ Several studies report the effect of GI in the glycemic control of diabetics and in the prevention of CVD.¹¹⁻¹³ The results of some studies suggest that the ingestion of low GI diets

contribute to control obesity, reducing body fat content.^{7,11,14-20} However, these beneficial effects are not confirmed by some scientists.²¹⁻²³

There are evidences that rapid intestinal absorption of glucose after the ingestion of high GI foods induces a sharp increase in blood glucose and insulin, as well as a reduction in the concentration of glucagon. Thus, there is an increase in the rate in which glucose enters the muscles, liver and adipose tissue, inducing glycogen synthesis, and stimulating lipogenesis and inhibiting lipolysis. On the other hand, the low GI foods are slowly digested and absorbed, reducing the post-prandial glycemic and insulinemic responses. These type of responses lead to a reduction in glucose oxidation and an increase in fat oxidation, resulting in a reduction in body fat content.^{7,11,24} Due to the importance of the quality of the diet consumed in the genesis of obesity and the likely beneficial effects of low GI in the prevention and control of this disease, the purpose of this study was to analyze the previously published studies on this topic. A summary of all studies discussed in this manuscript is described on table I.

Intervention studies involving animals in laboratory

Animal studies have shown more consistent results than human studies, indicating a reduction in body fat, and a higher fat oxidation after the consumption of low GI diets. In a study, Pawlak et al.¹⁴ randomly fed Wistar rats with one of the three following isocaloric diets: a) high GI diet, rich in amylopectin, b) low GI diet, rich in amylose, both with 45% CHO, 35% fat and 20% protein; and c) high fat diet with 59% fat, 20% of CHO and 21% of protein. The high and the low GI diets were ingested for 5 days and the high fat diet for 4 days. The groups did not present any change in body weight, because these diets were planned to maintain weight. However, it was verified that the low GI group had less epididymal fat than the other two groups. In addition, the consumption of the low GI diet led to lower insulin levels in the first 30 minutes of a glucose tolerance test than the consumption of the other two diets.

Kabir et al.²⁵ evaluated the effect of the chronic consumption of two types of starches, differing in GI and amylose/amylopectin contents, in rat epididymal adipocytes. Mung beans (GI 67 ± 5 and 32% amylose) and waxy cornstarch (GI 107 ± 7 and 0.5% amylose) starches were provided to two groups of diabetic and non-diabetic rats. After three weeks, adipocyte diameter was smaller in rats that consumed mung beans starch (low GI) compared to those that consumed the waxy cornstarch diet. The low GI starch increased maximal insulin-stimulated ¹⁴C-glucose oxidation, indicating an improvement in insulin sensitivity, and a reduction in lipogenesis.

Male rats, five weeks old, received a low GI diet for a week. Then the rats were randomly allocated into one

Table I
Main characteristics of the studies discussed

<i>Author, year</i>	<i>Studied sample</i>	<i>Type and duration of the dietetic intervention</i>	<i>Results</i>	<i>Comments</i>
Kabir et al., 1998	Diabetic and non-diabetic rats.	Diets with identical macronutrients composition: high GI or low GI, for 3 weeks.	LGI: < epididimal adipocytes, > IS, < lipogenesis from glucose.	Humans do less lipogenesis than rodents.
Pawlak et al., 2001	Wistar Rats.	High GI or low GI isocaloric diets for 5 days or high fat diet, for 4 days.	Low GI: < epididymal fat mass and insulinemia in GTT at 30 minutes.	Low GI diets: > resistant starch content may have affected the results.
Bouché et al., 2002	11 non-diabetic men, BMI 28 kg/m ² .	Isocaloric diets, identical in macronutrients: high GI and low GI (<i>crossover</i>), for 5 weeks.	Low GI: < insulinemia and body fat %.	Low GI: > fiber consumption.
Liese et al., 2005	979 subjects, 40-69 years old, non-diabetics, 33% glucose intolerants.	Habitual diet consumption assessed.	Negative correlation: GI x IS Positive Correlation: GL x WC.	FFQ was not validate to estimate diet GI/GL.
Hare-Bruun et al., 2006	376 men and women, 36-65 years old.	Cohort study, six years of duration.	Positive correlation: GI x body fat % and WC in women, mainly in sedentary women, but not in men.	Dietary data assessed only in the month before the beginning of the study.
Ebbeling et al., 2007	73 healthy obese, 18-35 years old	Low GL high fat and low fat diets, for 18 months.	Low GL diet: > weight reduction among subjects with hyperinsulinemia at 30 minutes after GTT.	Estimation of diet CL based on dietary records.
Das et al., 2007	34 overweight healthy subjects, 35 years old.	Hypocaloric diets: low GL or low fat, for 1 year.	Low GL diet: weight gain.	Low GL diet: > fat content may have favored the result.
Maki et al., 2007	86 healthy men and women, BMI 32 kg/m ² , 50 years old.	Low GL diet <i>ad libitum</i> or control diet, energy deficit of 500 to 800 kcal/day, 36 weeks of duration.	Low GL: > weight (first 12 weeks), fat mass and lean mass reductions. No difference in weight loss at the end of study.	Both groups consumed low GL diets.
Clapp & Lopez, 2007	7 women, 25-46 years old, BMI 23.3-30.9 kg/m ²	Four daily meals of high or low GI, for 20 days (<i>crossover</i>).	Low GI: > energy requirements, > energy loss through feces and urine, > weight loss, > fat oxidation, < glycemia and insulinemia and > IS.	
Scribner Pawlak & Ludwig, 2007	15 male rats, 5 weeks old	Same macronutrients and energy density: high GI or low GI ingested <i>ad libitum</i> , for 25 weeks.	Energy consumption and BW not affected. Low GI: > body fat. High GI: > insulinemia	
Scribner et al., 2008	48 male rats	Identical macronutrients: high GI or low GI <i>ad libitum</i> , for 37 weeks.	Energy consumption and BW not affected. High GI: > glycemia, insulinemia, IR, fat %, QR	
Fabrini, 2008	16 type 2 diabetics, 50 years old, BMI 29 kg/m ² .	Two daily meals of high or low GI, for 30 days.	Low GI: < fat % (women).	GI of test meals determined in laboratory.
Abete, Parra & Martinez, 2008	32 healthy obese men and women, 36 years old	Low GI or high GI hypocaloric diets (<i>crossover</i>), for 8 weeks.	Low GI: higher weight loss. Weight loss correlated with fiber ingestion.	Low GI: > fiber content. Test diets menu monotonous, especially for the low GI group.
Stevenson et al., 2009	8 healthy and sedentary women, 24 years old, BMI 21 kg/m ²	Low GI or high GI breakfast (<i>crossover</i>), 1 day.	Low GI: < glycemic and insulin response AUC, > fat oxidation and < CHO oxidation during the exercise.	Low GI: > consumption of fibers.

GI: glycemic index; GL: glycemic load; GTT: glucose tolerance test; IS: insulin sensibility; BW: body weight; IR: insulin resistance; RQ: respiratory quotient; WC: waist circumference; FFQ: food frequency questionnaire; BMI: body mass index; AUC: area under the curve; CHO: carbohydrate.

of the experimental groups and received *ad libitum* water and high or low GI diets during the next 25 weeks. While the starch in the high GI diet was 100% provided as amylopectin (n = 8), the starch in the low GI diet was composed of 60% amylose and 40% amylopectin (n = 7). These diets had identical macronutrients composition (68% CHO, 13% fat and 19% protein). In the second week, after randomization, fecal samples were collected during seven days in order to analyze the content of resistant starch of the diets and body composition was evaluated by dual energy X-ray absorptiometry (DEXA).¹⁵

Although energy consumption and body weight were similar between the groups, there was a higher energy loss in the feces of the rats that ate the low GI diet (9.66 ± 1.59 kcal vs. 2.24 ± 0.33 kcal, $p < 0.0001$). It was verified a higher body fat content in the group fed with high GI starch. Although blood glucose was not affected, insulinemia was higher in the high GI diet group, indicating that an increase on insulin secretion was necessary to keep the normoglycaemia.¹⁵ It is possible that the hyperinsulemia observed after the high GI diet stimulated the increase in the availability of oxidant and non-oxidant glucose to the cells until glycogen storage saturation, when the surplus acetyl Co-A was diverted for the synthesis of fatty acids. The increase of the fatty acids synthesis associated with the preferential oxidation of glucose in turn promotes the increased synthesis of triglycerides, resulting in higher body fat content in response to the consumption of high GI diets.

The effects (energy metabolism, adiposity and insulin sensibility) of diets differing in GI were tested in 48 male rats. In the first week, all of them received a low GI diet, which was rich in amylose. Then, these rats were randomly allocated in one of two groups, in order to continue consuming a low GI diet (60% amylose + 40% amylopectin) or to consume a high GI diet (100% amylopectin) during next 37 weeks. Both groups received food and water *ad libitum*. These diets had identical macronutrients composition (68% CHO, 13% fat and 19% protein) and energy density.²⁴

The energy intake and body weight did not differ between the groups. The group fed with high GI diet presented a higher fasting blood glucose (6.2 ± 0.5 versus 5.4 ± 0.9 mmol/l, $p = 0.0003$) and plasma insulin (114.15 ± 68.0 versus 58.8 ± 22.7 pmol/l, $p = 0.03$), as well as insulin resistance 2.2 times higher (5.4 ± 3.4 versus 2.4 ± 1.1 , $p = 0.02$) than the low GI group. On week 38, the fat percentage was 40% higher in the high GI group than in the low GI group (30.1 ± 7.6 versus 21.5 ± 3.1 , $p = 0.008$),²⁴ indicating that the effect of GI on body composition is mediated by changes on substrate oxidation and not only on energy intake.

It is possible that these results are due to postprandial hyperglycemia, leading to an increase on insulin secretion, promoting greater glucose uptake in insulin-sensitive tissues and inhibiting lipolysis in the adipose tissue. These events favor CHO oxidation instead of fat

oxidation, and in the long term leads to the increase in body fat. This hypothesis is supported by the higher respiratory quotient observed in rats fed with high GI diet during the first weeks of that study,²⁴ indicating the occurrence of lower fat oxidation than the other group. However, according to the authors²⁴ the higher content of resistant starch in the low GI diet might have contributed to the achieved results. It is worth emphasizing also that the extrapolation of these results to humans must be made with caution, since humans do less de novo lipogenesis than rodents, which may reduce the effect of the diet on body composition.

Clinical studies in humans

Epidemiologic studies

In a cohort study,²⁶ the association between GI and GL of the habitual diet versus weight and body composition was assessed during 6 years. A total of 376 Danish men and women, 36 to 65 years of age, and non-diabetics participated in the study. Anthropometric parameters and body composition (bioimpedance) were assessed at baseline and at the end of the study. The data related to the diet consumed by the participants in the previous month was evaluated through interviews. The GI and GL were calculated from the mean values published by several studies, and were based on the values presented by foods that had similar characteristics to those consumed by the participants.

Positive correlations between GI versus body fat percentage and waist circumference were verified only in women. This effect was more pronounced in sedentary women. There was no correlation between GL versus weight and body fat. It was verified that data about body weight, fat percentage, waist and hip circumference and ingestion of fat at the beginning of the study were lower in the group that consumed high GL diet. The ingestion of CHO, energy, sugar and dietetic fiber was lower in the group that consumed low GL diet.²⁶ Therefore, these differences may have contributed to the achievement of positive results attributed to the GI and the lack of effects associated to the GL.

It must be considered, however, that the investigators evaluated only the diet consumed in the month preceding the start of the study. It seems unlikely that during six years most of participants did not change their eating habits. The authors of that study did not provide information about the type of questionnaire used obtain the dietary data. The information about the way in which foods were prepared and the types and quantities of ingredients added to the dishes consumed are important to estimate the GI/GL of the ingested diet.⁹

Non-diabetics, 40 to 69 years of age, with several ethnic group, gender and age, 33% of which were glucose intolerant, participated in a transversal study. The adiposity was evaluated through BMI and waist circumference. The data referring to habitual food con-

sumption were obtained using a validated semi-quantitative food frequency questionnaire, containing 114 items, in order to achieve information referring to food consumption in the previous year. The ingested diet presented medium GI (IG: 58 ± 4) and medium GL (GL: 128.3 ± 55.9). It was verified a negative association between GL and insulin sensitivity as well as a positive association between GL and waist circumference. These results are probably due to hyperglycemia caused by the consumption of the high GL diet, which demands a higher production of insulin, increasing glucose uptake by insulin-sensitive tissues and inhibits lipolysis in adipose tissue. These metabolic events promote CHO oxidation at the expense of fat, increasing body fat mainly in the abdominal region.²⁴

The increase in fiber intake was associated to the reduction in fasting insulinemia, waist circumference and BMI in the study participants.²⁷ The adiposity, mainly the intra-abdominal fat, assessed by BMI and waist circumference, is the main cause of insulin sensitivity impairment, due to the action of the adipocins released by the visceral adipose tissue.^{28,29} It should be noted, however, that the food frequency questionnaire used in the study was not validated to estimate the GI/GL of the diet. Furthermore, it should be emphasized that the data reported on the questionnaires may not reflect the diet ingested.^{30,31}

Intervention studies involving humans

The effect of GI on body fat was evaluated in eleven non-diabetic men, with mean BMI 28 kg/m^2 . In the crossover study, high (GI > 60) or low GI (GI < 45) breakfast and lunch meals were ingested during five weeks. Diets were prescribed according to the GI group in which participants were allocated to meet the nutritional needs of each participant. These diets had similar macronutrients composition and energy. The compliance to the prescribed diets was evaluated by the analyses of food records of the last seven days of each study session. Body composition was measured by DEXA.³²

It was observed a lower insulinemia in the low GI group. Although the weight of participants was not affected in response to the type of diet ingested, there was a reduction of approximately 700 g in total fat mass and a tendency to increase fat-free mass after the low GI diet consumption.³² These results suggest the replacement of fat tissue by the same quantity of lean mass, indicating a reduction in CHO and an increasing in fat oxidation. However, the consumption of fiber was higher in the low GI group. According to Roberts et al.,³³ a higher consumption of fiber can reduce the intestinal absorption of fat. In addition, the fermentation of soluble fibers increases the production of satiety gut hormones.³³ Therefore, the results of that study³² can not be attributed to the effect of GI presented the ingested diets only.

The effect of GI on energy substrate utilization at rest and low intensity exercise was analyzed in a crossover study, with the participation of eight sedentary and healthy women, 24 years of age and BMI 21 kg/m^2 . The meal consumed in the evening prior to the experiment was standardized. High (78) or low GI (44) breakfast presenting the same macronutrients content was consumed in fifteen minutes in the laboratory by the subjects. Participants rested in the lab for the following 3 hours. Then, they walked on a treadmill for one hour. After the exercise, volunteers received a standard lunch, which was eaten within fifteen minutes. In the following two hours, participants rested in the lab, and blood and expired air samples were collected. Resting energy expenditure was measured by an open circuit indirect calorimeter after breakfast and after lunch.³⁴

Blood glucose concentrations were lower at 30, 45, 135 and 150 minutes after the low GI breakfast. The areas under the glycemic and insulinemic response curves were lower after the low GI compared to the high GI breakfast. The free fatty acids concentrations from 120 to 180 minutes after the low GI breakfast tended to be higher, suggesting the occurrence of fat oxidation during that period of time. While fat oxidation was higher in the low GI group, CHO oxidation was higher in the high GI group during the exercise.³⁴ However, in that study the consumption of fiber once again was higher in the low GI group. According to some authors³⁵⁻³⁷ a higher consumption of fiber can reduce the post-prandial glycemic response. Therefore, the results of that study can not be attributed to the effect of GI only.

However, these results differ from those observed in another study,³⁸ in which the effect of consumption of two daily meals of high or low GI on weight and body composition was assessed in sixteen type 2 diabetics. The participants had a mean age of 50 years and a BMI of 29 kg/m^2 . During thirty consecutive days, the low or high GI breakfast and lunch were consumed in the laboratory. The energy density, macronutrients proportion and fiber content of the test meals were similar. The other meals were consumed under free living conditions, and the subjects were instructed to select food that had GI value according to the group in which they were allocated. The GI of 28 types of test meals was determined in a pilot-study. The mean GL and GI of the subjects' habitual diet and of the diet consumed during the study were based on food records information.

The weight and body composition of the diabetics were not affected during the study. There was, however, a significant reduction in the fat percentage among the women of low GI group.³⁸ It is possible that such result occurred due to the higher body fat presented at baseline by the women than the men. However, the mechanism responsible for the observed effect in this study it is not clear and it should be investigated in future studies. It is worth emphasizing that in the previously mentioned study,³⁸ the GI of the test

meals was determined in the laboratory, differently from what was done in several other studies,^{22,32,34,37,40,41} in which the GI estimation was based on the International GI tables. According to Flint et al.,⁴² the GI presented in these tables may not reflect the value presented by a given food type.

In another crossover study, Clapp and Lopez⁴³ compared the GI effect on blood lipids profile, energy requirement, fat oxidation and insulin sensitivity in 7 adult women, aged between 25-46 years and presenting BMI from 23.3-30.9 kg/m². During 20 days, the participants performed physical activity during 20 minutes, three times a week, and ingested four daily low (59 ± 2) or high GI (92 ± 3) meals, which provided sufficient energy to maintain their body weight. There was a higher energy consumption (p < 0.03) and the energy requirements were 11% higher (p < 0.03) during the low GI session. There was also a higher energy loss through the feces and urine (p < 0.002) during the low GI compared to the high GI session. At the end of the study, a significant weight loss in response to the consumption of the low GI meals (p < 0.03) was observed. While fat oxidation in fasting state and at rest was equivalent to 45 ± 4% during the low GI session, this type of oxidative response corresponded to 28 ± 5% in the high GI session. Furthermore, blood glucose and insulinemia were 45% lower and the insulin sensitivity was 20% higher in response to the consumption of the low GI diet.

During eight consecutive weeks, fourteen women and eighteen men healthy and obese, 36 years-old, consumed high (60-65) or low (40-45) GI hypocaloric diets. Food intake was assessed on the week before the beginning of the study and on the last week of the study. In the low GI diet, most of the protein were originated from legumes, and in the high GI diet, the protein came from animal sources. The low GI group had a significant reduction in weight compared to the other group.⁴¹ However, the weight loss was correlated with fiber intake, which was higher in the low GI diet. It should be noted also that the menu used in the study was very monotonous, especially for the low GI group, which might have affected compliance. Thus, other factors could have contributed to the results obtained for the low GI group.

The effect of the consumption of low GL high fat and low fat diets on insulin secretion and body fat was evaluated during 18 months in healthy obese subjects. The experimental diets were prepared by the participants themselves and consumed *ad libitum*. Dietary records were used to assess food intake. A total of 73 adults, aged 18 to 35 years, participated in the study. Participants received intense guidance in the first six months, followed by one year of monitoring. GI and GL of the foods were obtained from the 2002 International GI Tables,⁴⁴ considering glucose as the referenced food. There was not difference in the weight and percentage of body fat (bioimpedance) after the consumption of these two test diets. However, when the

subjects were stratified by insulin concentration thirty minutes after the ingestion of an oral glucose dose, a significant reduction in the body weight (-1.2 ± 0.5 kg for each two-fold increase in insulin concentration at 30 minutes only in the low GL group, p = 0.02) and body fat (p = 0.01) was observed in the group with insulin concentration higher than the average (57.5 UI/mL) presented by all of the participants. Among the subjects that presented high insulin concentration, the group that ingested low GL diet lost weigh faster during the six first months (-1.0 *versus* 0.4 kg/month; p < 0.0001) and lost more weight after eighteen months (-5.8 *versus* -1.2 kg; p < 0.004) than the group that ingested the low fat diet.⁴⁰

In another study, diets differing in GI were consumed during one year, by 34 overweight and healthy adults, aged 35 years. The study was conducted in three phases: phase one, seven weeks of duration, when participants were guided to keep their weight by the ingestion of their habitual diet. On phase two, the subjects randomly consumed low or high GL diets, both providing 70% of their energy needs. In this phase, participants received ready to eat foods, during six months. Participants were instructed to consume the provided food only and to return any food that was not consumed. However, they were asked to report the consumption of any other food not provided. In the following six months, subjects were guided to prepare their own food, in order to keep the same diet of phase two. The test diets had the following composition: high GI (GI ≈ 85 and GL ≈ 118): 60% CHO, 20% fat and 30% protein; low GI (GI ≈ 52 and GL ≈ 45): 40% CHO, 30% fat and 30% protein. To evaluate compliance, the energy intake was measured by double labeled water. At the end of the study, weight gain was verified among the subjects that consumed the low GI diet²². However, the higher fat content of the low GL diet may have favored the result. It should be emphasized that the number of men and women that participated in the study was not indicated by the authors. Other investigators^{26,38} have observed that there seems to be differences in the effect of GI on body composition of men and women.

Healthy men (n = 58) and women (n = 28), with mean BMI 32 kg/m² and 50 years of age participated in a 36 weeks study when a low GL *ad libitum* diet or a control diet was consumed. The test diets presented an energy deficit of 500 to 800 kcal/day. During the first two weeks (phase 1), the low GL group was advised to eat a low CHO and low saturated and trans fat diet to satisfy their hunger. Certain types of foods (high in starch, fruits and alcohol) were not included in that diet. From week two to twelve (phase 2), low GI foods were reintroduced into the diet and a moderate consumption of alcohol was allowed. After this phase, participants from both groups could choose to consume a weight maintenance diet or to keep ingesting the weight loss diet until week 24, when all of them started to consume a weight maintenance diet until the end of the study. Body composition was measured by DEXA.

The GI and GL of the diet consumed during the study were estimated from a food frequency questionnaire data.³⁹

In the first twelve weeks, the low GL group lost more weight than the control group (-4.9 ± 0.5 kg versus -2.5 ± 0.5 kg, p adjusted = 0.002). On week 12, 55% of the low GL group and 21% of control group lost at least 5% of their body weight ($p = 0.002$). At the end of the study, the weight lost did not differ between the groups. Although on week 12 both groups lost body fat and fat free mass, the reduction of these two compounds was higher in the low GL group (-1.9 ± 0.3 kg versus -0.9 ± 0.3 kg, $p = 0.016$ for fat mass and for fat free mass (-2.2 ± 0.2 kg versus -1 ± 0.3 kg, $p < 0.001$). On week 36, the reduction of fat free mass occurred only in the low GL group (-2.1 ± 0.3 kg versus -0.9 ± 0.3 kg, $p = 0.004$).³⁹ Among the studies analyzed in this review paper, this was the only one that verified a reduction in fat free mass after the consumption of low GI diet. However, according to the GI classification cut points proposed by Brand-Miller et al.⁴⁵ both groups consumed low GI diets. Considering that GL is obtained by the product of GI versus the amount of CHO ingested, it appears that lower the GL was obtained by reducing the intake of CHO.

Conclusion

Although there are divergences in the results of the studies analyzed in this paper, the results of most studies suggest that the consumption of a low GI diet leads to a lower insulin response, which induces an increase in fat oxidation, resulting in the reduction of body fat. These beneficial effects are mainly observed in studies involving laboratory animals, because in this case there is a greater control of factors that can interfere in the GI effects on the parameters evaluated.

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