RESEARCH

Diabetes Research and Metabolism

Glycemic Index and Glycemic Load to the Biochemical Profile of Dyslipidemic Patients

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Abstract

Background: *The positive and negative health effects of dietary carbohydrates are of interest to both researchers and consumers. Low glycemic index (GI) and glycemic load (GL) carbohydrates have been shown to have favourable effects on blood lipid parameters.*

Objective: This study aims to investigate whether the GI and GL diet in dyslipidemic individuals.

Methods: *The subjects, 15 men and 15 women, dyslipidemic patients followed up at Hospital de Coari, were invited to answer a self-administered questionnaire for 3 days about food intake. The fasting biochemical profile was analyzed, such as total cholesterol, triglycerides and blood glucose.*

Results: *Low GI diet daily value were observed in women and men and GL diet daily value below the minimum low line for both. A correlation was found between GI and GL diet with triglycerides levels and woman and not in men. Lunch GL value were associated with blood triglycerides parameters. Biochemical profile showed an increase in fasting blood glucose only in men and in lipid levels for both.*

Conclusion: Dyslipidemic individuals might be potential influencing factors in the associations between GI and GL diet and dyslipidemia, suggesting such modifications could potentially be a useful public health recommendation.).

Keywords: *glycemic index, glycemic load, blood glucose, total cholesterol, triglycerides*

Introduction

Many factors are involved in the progress of obesity, because not only high fat, but high carbohydrate diet can also influence the pro-obesity metabolic pathways and /or changes in blood lipid and sugar levels [1]. As overweight, obesity and insulin resistance have become more prevalent, concerns for the amount and type of carbohydrate consumed may increase, rather than just decrease cardiometabolic risk [2].

Some sources of carbohydrates can be beneficial or harmful, depending on the glycemic index (GI) and fiber content [3-7]. Both the quantity and the quality of the carbohydrate influence the glycemic response [8].

Glycemic index (GI) and glycemic load (GL) are measures of carbohydrate quality [9]. The GI food measure indicates an increase in the postprandial blood glucose of the serving food compared to 50 g of carbohydrate [9-11]. GI is a valid and reproducible measure to express the glycemic response of foods [2]. High GI foods rapidly increase blood glucose levels and insulin secretion, while low GI foods increase these parameters more slowly [12].

The GL food concept takes into account the GI food and the amount of carbohydrate available in a food consumed [9,13]. The GL assesses the quality and quantity of carbohydrates and, the greater this measure, the greater the increase in the levels of blood markers responsible for the development of the metabolic syndrome [1,14].

Postprandial blood glucose and, consequently, the GI and GL diet have been linked to hyperglycemia, insulin resistance, diabetes, coronary heart disease and obesity [1,2]. Long-term consumption of a high GI / GL diet results in chronically high blood glucose and therefore in a chronically high insulin concentration [12].

Evidence supports that a low GI diet, long-term food consumption for foods with low sugar content and more soluble fibers in the diet may be associated with better glucose control and lipidemic profile [15-17]. Unlike Mediterranean type diets, low GI diets are not limited by eating specific regional foods and, therefore, can be more flexible and appropriate in different contexts [18]. Diets with foods with GI values <55, compared to 50g for glucose, are considered low GI diets, where values between 56 - 69 are average GI diets and values> 70 are high GI diets [8,11].

In summary, GI food compares equal amounts of carbohydrates, while GL food was introduced to quantify the overall glycemic effect of a portion of food [19-21]. Thus, the GL of food is the product of the amount of carbohydrate available from a food that takes into account both the quality and the amount of carbohydrate consumed [1,8,22,23].

Elevation in blood glucose and insulin was caused by high GL diets. Long-term consumption of a high GL is associated with an increased risk of diabetes and coronary heart disease [19]. GL values are classified as low (GL <10), medium (GL 11-19) and high (GL> 20) [11].

Thus, the GI proved to be a more useful nutritional concept than the chemical classification of carbohydrates, contributing to improving human health [8]. Therefore, the aim of this study is to analyze the GI and GL diet of dyslipidemic patients and their biochemical profile.

Methods

Subjects

Thirty dyslipidemic volunteer patients (15 men and 15 women), aged between 32 and 60 years, were selected to participate in the present study in the city of Coari, Brazil. The protocols were approved by the Ethics Committee of the Amazonas Federal University. Volunteers provided written informed consent prior to participating in the study.

Inclusion criteria

The volunteers were followed up at the Hospital of Coari with screening for dyslipidemia. Age between 30 and 60 years, nonsmokers and with no history of alcohol or drug abuse. In addition, only patients with a 6-month history without other diseases and without medication that could interfere with food intake were recruited.

Exclusion criteria

Exclusion criteria included current chronic medical illness (except diabetes), pregnancy, hormone replacement therapy, lipid-lowering medication, use of steroids and other agents that can influence lipid metabolism, use of warfarin, smoking, hyper or hypothyroidism, cardiovascular events in the last six months, psychological inadequacy, major systemic diseases, gastrointestinal problems, proteinuria, liver and kidney failure.

Experimental protocol

The follow-up visit to the patients was chosen where fasting blood samples were collected by venepuncture for analysis of blood glucose and lipids at the Hospital de Coari. After that, a dietary data questionnaire [24] was applied to the patients. Volunteers were asked to write down all the food they had eaten in the past three days, using a self-administered food frequency questionnaire at main meals and snacks, including water, coffee, tea, supplements or other drinks and liquids. The fiber content of the diets was not reported in this study.

Dietary GI and GL determination

The GL and GI diets were classified as none (0-1), low (1-2), medium (2-3) and high (3-4) levels. None at absent values, low (IG <55; GL <10), medium (IG 56–69; GL 11-19) and high (IG> 70; GL> 20) [11].

Biochemical analyses

The serum and plasma were separated by low speed centrifugation at 3000 rpm for 10 minutes (Rofina 48R centrifuge). The samples were stored at -80°C until they are ready to be analyzed. Blood glucose and lipids (total cholesterol and triglycerides) were measured in plasma using an enzymatic assay kit.

Statistical analysis

Descriptive statistics was used to determine the mean and standard deviation (Mean ± SD), according to the normality pattern (parametric and nonparametric variables), applying the Kolmorogov-Smirnov test. The significance of the differences between the means was determined by the Student's t-test and the strength of the relationships was described by the Pearson product moment correlation coefficient. Statistical calculations were performed using the GraphPad Prism® version 6.00 statistical analysis program for Windows. The ANOVA method was used to verify the degree of significance ($p < 0.05$).

Results

Glycemic index

The data regarding the daily GI diet are shown in table 1.

The meal that provides the greatest GI food is breakfast for both. Meals between lunch and dinner include high GI foods for men and low GI foods for women. In addition, the meal, like the morning snack, is ignored by women, while the evening snack is ignored by men.

The low GI diet was observed for both, but did not differ significantly (Figure 1A).

The classification of the GI meals showed an average value for breakfast for both. Missing value in the morning snack for men. Low value for women and average value for men at lunch. Low value for afternoon snacks and dinner and missing value for evening snacks for women (Figure 1B).

Analysis of the results also showed that the GI diet was negatively correlated with an increase in triglycerides in female volunteers (Figure 2A). This correlation was not observed in male volunteers (Figure 2B).

Glycemic load

The data regarding the daily GL diet are shown in table 2.

In general, all daily meals reveal that men and women consume more foods with a low GL foods. Meals taken after lunch showed that women exceeded men in relation to GL. The daily GL diet was considered below the lower limit and there is no significant difference in both sexes (Figure 3A). The GL classification of daily meals showed low GL values for breakfast and lunch, absence of GL in the morning, afternoon and evening snack for men and women (Figure 3A).

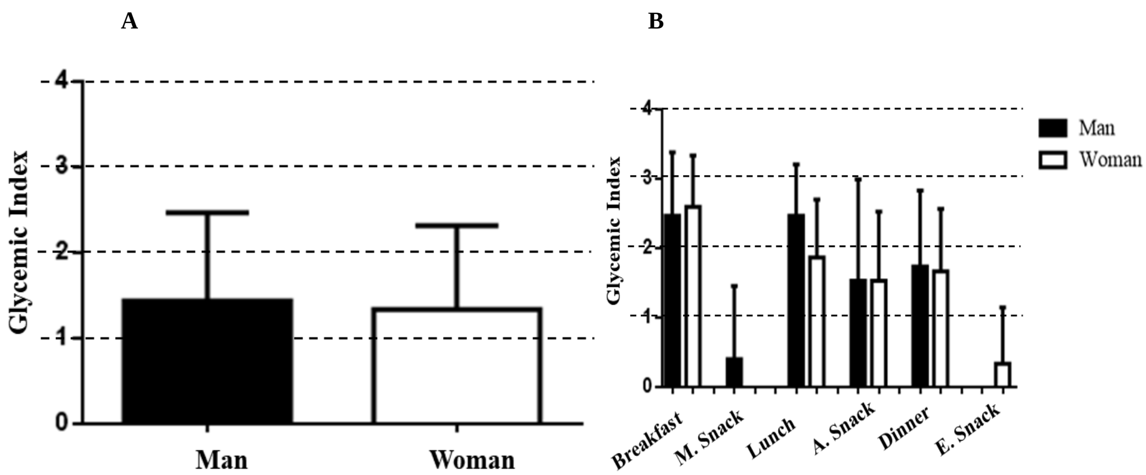
The results showed the negative correlation between the GL diet and the increase triglycerides in female volunteers (Figure 4A). This correlation was not observed in male volunteers (Figure 4B).

The results showed the negative correlation between GL lunch and the increased triglycerides in female volunteers (Figure 5A). This correlation was not observed in male volunteers (Figure 5B).

GI (%)	Breakfast	M. snack	Lunch	A. snack	Dinner	E. snack
Men						
None	0,0	86,7	0,0	40,0	13,3	0,0
Low	26,7	0,0	13,3	13,3	33,3	0,0
Medium	0,0	0,0	26,7	0,0	20,0	0,0
High	73,3	13,3	60,0	46,7	33,3	0,0
Women						
None	0,0	0,0	0,0	13,3	0,0	80,0
Low	13,3	0,0	40,0	40,0	60,0	13,3
Medium	13,3	0,0	33,3	26,7	13,3	0,0
High	73,3	0,0	26,7	20,0	26,7	6,7

M. snack: morning snack, A. snack: afternoon snack, E. snack: evening snack. Values are means ± SD

Table 1. *Glycemic index daily diet classification (%)*



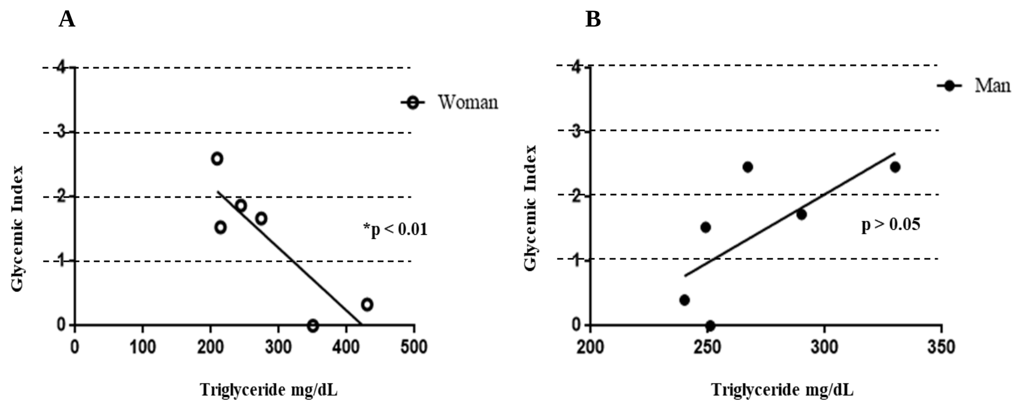
(A): Glycemic index diet; (B): Glycemic index meal. M. snack: morning snack, A. snack: afternoon snack, E. snack: evening snack. GI levels: none (0-1), low (1-2), medium (2-3) and high (3-4). Values are means ± SD, $p > 0.05$.

Figure 1. *Glycemic index*

GL (%)	Breakfast	M. snack	Lunch	A. snack	Dinner	E. snack
Men						
None	0,0	86,7	0,0	40,0	13,3	0,0
Low	80,0	13,3	73,3	60,0	86,7	0,0
Medium	20,0	0,0	20,0	0,0	0,0	0,0
High	0,0	0,0	6,7	0,0	0,0	0,0
Women						
None	0,0	0,0	0,0	13,3	0,0	80,0
Low	80,0	0,0	93,3	80,0	86,7	20,0
Medium	6,7	0,0	0,0	6,7	13,3	0,0
High	13,3	0,0	6,7	0,0	0,0	0,0

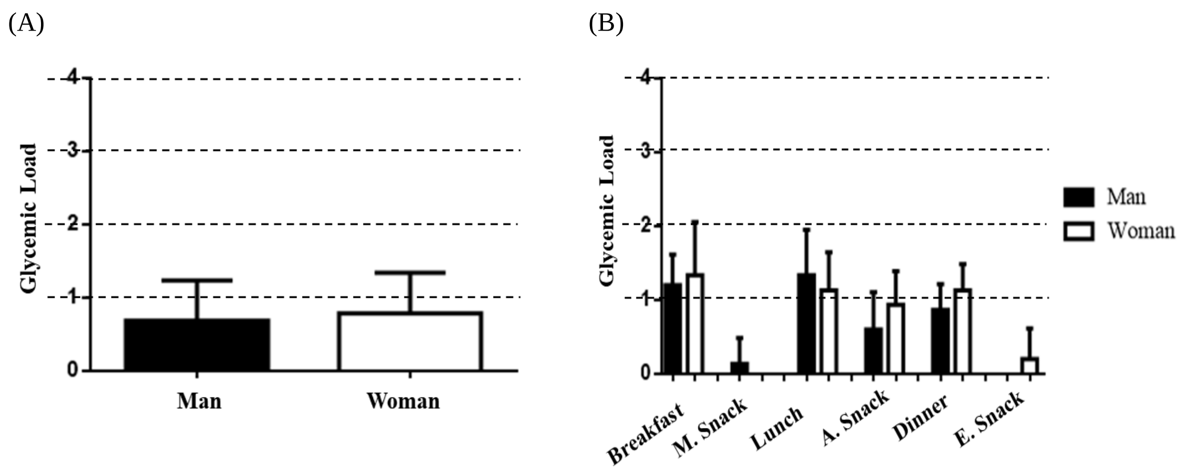
M. snack: morning snack, A. snack: afternoon snack, E. snack: evening snack. Values are means ± SD

Table 2. *Glycemic load daily diet classification (%)*



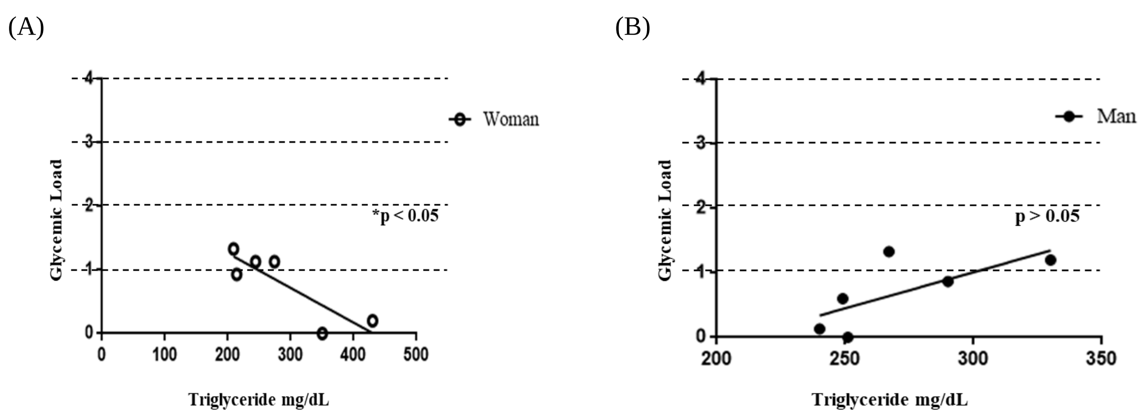
(A): Women; (B): Men. GI levels: none (0-1), low (1-2), medium (2-3) and high (3-4). Values are means ± SD, *p < 0.05.

Figure 2. *Glycemic index and triglyceride levels correlation*



(A): Glycemic load diet; (B): Glycemic load meal. M. snack: morning snack, A. snack: afternoon snack, E. snack: evening snack. GL levels: none (0-1), low (1-2), medium (2-3) and high (3-4). Values are means ± SD, p > 0.05.

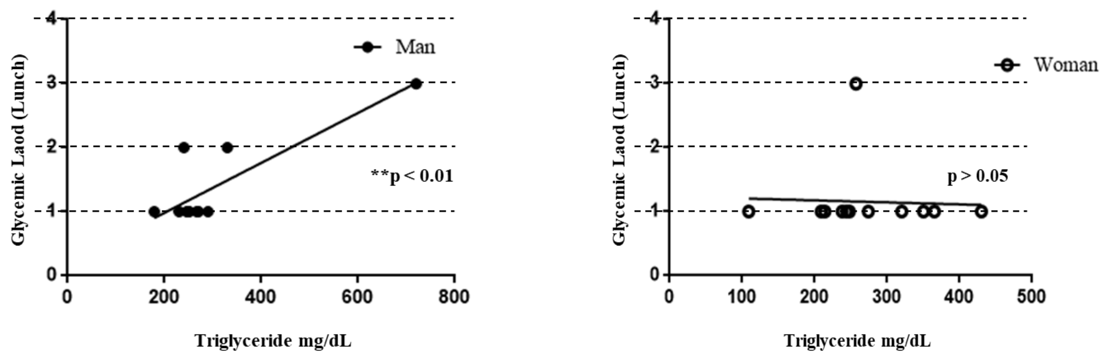
Figure 3. *Glycemic load*



*(A): Women; (B): Men. GI levels: none (0-1), low (1-2), medium (2-3) and high (3-4). Values are means ± SD, *p < 0.05.*

Figure 4. *Glycemic load and triglyceride levels correlation*

(A) (B)



(A): Women; (B): Men. GI levels: none (0-1), low (1-2), medium (2-3) and high (3-4).
Values are means ± SD, *p < 0.05.

***Figure 5**. Glycemic load lunch and triglyceride levels correlation*

(mg/dL)	**Men**	**Women**	**Reference value**
(fasting)			
Total Cholesterol	243,0+31,08	250,3+22,99	< 190
Trygliceride	297,5+145	269,4+81,31	< 150
Blood glucose	111,1+61,69	92,00+46,00	< 100

Values are means ± SD

***Table 3.** Biochemical profile in dislypidemic patients.*

Biochemical profile

Clinical biochemical measures of the volunteers who completed the study are shown in Table 3.

The biochemical profile showed dyslipidemia due to cholesterol above 190 mg / dL and triglyceride above 150 mg / dL in fasting, as recommended by the [25]. Fasting blood glucose above 100 mg / dL was verified in male volunteers. According to the Brazilian Diabetes Society (2019), fasting blood glucose> 100 mg / dL is pre-diabetes or increased risk of diabetes [26].

Discussion

The present study was designed to assess the dietary effects of GI and GL in dyslipidemic patients. Evidence on associations between GI or GL and risk of cardiometabolic disease has been described separately, as first risk factors for type 2 diabetes, followed by risk of cardiovascular disease [11]. The biochemical profile of this study reported the traditional risk factors for dyslipidemia (cholesterol and triglycerides).

Some studies have suggested that while the general GL of a diet is mainly associated with high-carbohydrate foods and therefore is closely related to total carbohydrate intake, GI is associated not only with high consumption of high-carbohydrate foods, but also low consumption of some low-carbohydrate foods, including fruits, dairy products, vegetables and legumes [27-29]. Thus, the dietary GI, unlike GL, may reflect more dimensions of the diet than just carbohydrates [30].

Although the standard methodology for assessing the GI of a food involves measuring blood glucose over a period of 2 h period [31], there are no standard protocols for assessing the postprandial effects of different GI foods on other results. In general, low GI meals resulted in lower blood glucose [32-35] and insulin levels [32]. In addition, lower blood glucose and / or insulin secretion were observed in the context of higher dietary fiber content in meals [34,35]. Interestingly, the women in this study showed a negative correlation between GI and GL daily with increased blood triglyceride levels. Althougth, studies have indicated that diets rich in GI or GL may be associated with type 2 diabetes [17,36-40] a study have been confirmed that low GL in both men and women, with greater risk reduction in diabetic women [17]. Therefore, a decrease in fasting blood glucose can be beneficial in preventing the development of metabolic diseases, especially in high-risk groups, as represented in this study.

The results showed that, regardless of gender, they behave the same way in relation to the GI and GL diet. In addition, the highest peaks of these measures were found at breakfast, lunch and dinner, but did not differ significantly between men and women. Interestingly, higher GI and GL were observed in the first part of the day (until lunch) for men, while women in the second part of the day (after lunch), showing different eating behaviors in both. An old review showed that foods with lower GI lead to decreased hunger, increased satiety or less energy consumption [41]. In another study, participants had lower fasting blood glucose and reported greater satiety after consuming breakfast with low GI [42].

In general, this study found no difference between the value of the GI and GL diet between men and women, but a negative correlation was observed in women between the GI and GL lunch with the increase in blood triglyceride levels, while men did not show this same correlation.

Concerning lipid metabolism, low GI diet study tended to decrease fasting plasma cholesterol and decreased triacylglycerol levels after the lunch meal [43]. Studies have also shown that a low GI diet is associated with lower fasting plasma LDL cholesterol, fasting triglyceride, and postprandial glucose concentrations and increase in fasting plasma HDL concentrations [31-35,44,45]. Also another study, reduced triglycerides, total-cholesterol, and LDL-cholesterol but with no effect on HDL-cholesterol was reported in a medium-term study, which involved daily consumption of low GI meals for 3 months [46].

On the other hand, another short-term study carried out through a 5-week intervention did not observe significant effects on lipid parameters in three weeks [43]. These results suggest that changes in fasting cholesterol concentrations resulting from modification of the GI diet may require a timerframe [42]. However, the greatest weight loss with the low-GL diet was not accompanied by significant changes in cholesterol total, LDL- or HDL-cholesterol or triglycerides levels [47].

In diabetic individuals, it is generally found that chronic consumption of a low GI diet improves plasma glucose and lipid profiles [48]. An old review showed that a low GI diet resulted in a decrease in the glycemic area under the curve and in plasma levels of cholesterol and triacylglycerol [49]. On the other hand, one study did not report differences in fasting lipids at the end of periods of low or high GL diet [50].

However, although no difference was observed in the GI or GL diet with increased blood glucose levels in the participants, another study showed that the GL diet has been a stronger predictor of postprandial glycemia and insulin response than the GI [51,52]. This correlation was probably not observed due to the response of insulinemic activity.

An important limitation of this study is the use of food frequency questionnaires to characterize the dietary GI. Although this method is useful for classifying individuals according to relative intake in a large study population, this method used was not designed specifically to assess dietary GI. On the other hand, the validity of the GI of carbohydrate foods added to a mixed meal is doubtful [23].

Conclusions

The negative relationship between the reduction of GI and GL with increased levels of triglycerides in non-diabetic women in this study appears to be associated with an adequate insulin response, different from what occurs in pre-diabetic men.

The choice of foods with low GI and GL should be recommended, as a whole diet, even in healthy people. These results suggest that a modification of the GL diet is a good public health recommendation, as opposed to the modification of the GI diet of all meals during the day.

This study describes the potential limitation of the GI and GL criteria and suggests the importance of comprehensively assessing the historical dietary profile of dyslipidemic patients. Further studies are needed to determine the role of these measures in the lipidemic in this population.

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References

1. Patel Brand-Miller JC, Holt SHA, Pawlak DB, McMillan J. Glycemic index and obesity. *Am J Clin Nutr*. 2002; 76: 281S-5S.
2. Augustin LSA, Kendall CWC, Jenkins DJA, Willett WC, Astrup A, et al. Glycemic index, glycemic load and glycemic response: An International Scientific Consensus Summit from the International Carbohydrate Quality Consortium (ICQC). *Nutr Metab Cardiovasc Dis*. 2015; 25: 795–815.
3. Ludwig DS. The glycemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. *JAMA*. 2002; 287: 2414–2423.
4. Jakobsen MU, Dethlefsen C, Joensen AM, Stegger J, Tjønneland A, et al. Intake of carbohydrates compared with intake of saturated fatty acids and risk of myocardial infarction: importance of the glycemic index. *Am J Clin Nutr*. 2010; 91: 1764–1768.
5. Jenkins DJA, Kendall CWC, McKeown-Eyssen G, Josse RG, Silverberg J, et al. Effect of a low-glycemic index or a high-cereal fiber diet on type 2 diabetes: a randomized trial. *JAMA*. 2008; 300: 2742–2753.
6. Astrup A, Dyerberg J, Elwood P, Hermansen K, Hu FB, et al. The role of reducing intakes of saturated fat in the prevention of cardiovascular disease: where does the evidence stand in 2010? *Am J Clin Nutr*. 2011; 93: 684–688.
7. Mirrahimi A, de Souza RJ, Chiavaroli L, Sievenpiper JL, Beyene J, et al. Associations of glycemic index and load with coronary heart disease events: a systematic review and meta-analysis of prospective cohorts. *J Am Heart Assoc*. 2012; 1: e000752.
8. Foster-Powell K, Holt SHA, Brand-Miller JC. International table of glycemic index and glycemic load values: 2002. *Am J Clin Nutr*. 2002; 76: 5–56.
9. Schlesinger S, Chan DSM, Vingeliene S, Vieira AR, Abar L, et al. Carbohydrates, glycemic index, glycemic load, and breast cancer risk: a systematic review and dose-response meta-analysis of prospective studies. *Nutr Rev*. 2017; 75: 420–441.
10. Jenkins DJ, Wolever TM, Taylor RH, Barker H, Fielden H, et al. Glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr*. 1981; 34: 362–366.
11. Vega-López S, Venn BJ, Slavin JL. Relevance of the Glycemic Index and Glycemic Load for Body Weight, Diabetes, and Cardiovascular Disease. *Nutrients*. 2018; 10.
12. Turati F, Galeone C, Augustin LSA, La Vecchia C. Glycemic Index, Glycemic Load and Cancer Risk: An Updated Meta-Analysis. *Nutrients*. 2019; 11.
13. Salmerón J, Manson JE, Stampfer MJ, Colditz GA, Wing AL, et al. Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. *JAMA*. 1997; 277: 472–477.

14. Atkinson FS, Foster-Powell K, Brand-Miller JC. International tables of glycemic index and glycemic load values: 2008. *Diabetes Care*. 2008; 31: 2281–2283.
15. Wolever TM, Jenkins DJ, Vuksan V, Jenkins AL, Wong GS, et al. Beneficial effect of low-glycemic index diet in overweight NIDDM subjects. *Diabetes Care*. 1992; 15: 562–564.
16. Jenkins DJA, Kendall CWC, Augustin LSA, Franceschi S, Hamidi M, et al. Glycemic index: overview of implications in health and disease. *Am J Clin Nutr*. 2002; 76: 266S-273S.
17. Livesey G, Taylor R, Hulshof T, Howlett J. Glycemic response and health--a systematic review and meta-analysis: relations between dietary glycemic properties and health outcomes. *Am J Clin Nutr*. 2008; 87: 258S-268S.
18. Evans CE, Greenwood DC, Threapleton DE, Gale CP, Cleghorn CL, et al. Glycemic index, glycemic load, and blood pressure: a systematic review and meta-analysis of randomized controlled trials. *Am J Clin Nutr*. 2017; 105: 1176–1190.
19. Bornet FR, Costagliola D, Rizkalla SW, Blayo A, Fontvieille AM, et al. Insulinemic and glycemic indexes of six starch-rich foods taken alone and in a mixed meal by type 2 diabetics. *Am J Clin Nutr*. 1987; 45: 588–595.
20. Wolever TMS, Vuksan V, Relle LK, Jenkins AL, Josse RG, et al. Glycaemic index of fruits and fruit products in patients with diabetes. *International Journal of Food Sciences and Nutrition* Internet.. 1993 Jan 1 cited 2020 Jul 6; 43: 205–212.
21. Brand-Miller JC, Allwan C, Mehalski K, Brooks D. The glycaemic index of further Australian foods. *Proc Nutr Soc Aust.* 1998; 22: 110.
22. Liu S, Willett WC, Stampfer MJ, Hu FB, Franz M, et al. A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. *Am J Clin Nutr*. 2000; 71: 1455–1461.
23. Stevens J, Ahn K, Juhaeri null, Houston D, Steffan L, et al. Dietary fiber intake and glycemic index and incidence of diabetes in African-American and white adults: the ARIC study. *Diabetes Care*. 2002; 25: 1715–1721.
24. Bueno AL, Czepielewski MA. O recordatório de 24 horas como instrumento na avaliação do consumo alimentar de cálcio, fósforo e vitamina D em crianças e adolescentes de baixa estatura. Revista de Nutrição Internet 2010; 2020; 23: 65–73.
25. Aa F, Mco I, Jfk S, Apm C, Ht B, AA, et al. Atualização Da Diretriz Brasileira De Dislipidemias E Prevenção Da Aterosclerose – 2017 Internet.. Vol. 109, Arquivos brasileiros de cardiologia. Arq Bras Cardiol; 2017 cited 2020 Jul 6.
26. Sociedade Brasileira de Diabetes. Parte 1 – Princípios básicos: avaliação, diagnostico e metas de tratamento do diabetes mellitus Classificação e diagnóstico do diabetes mellitus. DIRETRIZES. 2019-2020.
27. Du H, van der A DL, van Bakel MME, van der Kallen CJH, Blaak EE, et al. Glycemic index and glycemic load in relation to food and nutrient intake and metabolic risk factors in a Dutch population. *Am J Clin Nutr.* 2008; 87: 655–661.
28. Mendez MA, Covas MI, Marrugat J, Vila J, Schröder H. Glycemic load, glycemic index, and body mass index in Spanish adults. *Am J Clin Nutr*. 2009; 89: 316–322.
29. Murakami K, McCaffrey TA, Livingstone MBE. Associations of dietary glycaemic index and glycaemic load with food and nutrient intake and general and central obesity in British adults. *Br J Nutr*. 2013; 110: 2047–2057.
30. Schulz M, Liese AD, Mayer-Davis EJ, D'Agostino RB, Fang F, et al. Nutritional correlates of dietary glycaemic index: new aspects from a population perspective. *Br J Nutr*. 2005; 94: 397–406.
31. Wolever TM, Jenkins DJ, Jenkins AL, Josse RG. The glycemic index: methodology and clinical implications. *Am J Clin Nutr*. 1991; 54: 846–854.
32. Makris AP, Borradaile KE, Oliver TL, Cassim NG, Rosenbaum DL, et al. The individual and combined effects of glycemic index and protein on glycemic response, hunger, and energy intake. *Obesity (Silver Spring).* 2011; 19: 2365–2373.
33. Louie JCY, Markovic TP, Ross GP, Foote D, Brand-Miller JC. Timing of peak blood glucose after breakfast meals of different glycemic index in women with gestational diabetes. *Nutrients*. 2012; 5: 1–9.
34. Silva FM, Kramer CK, Crispim D, Azevedo MJ. A high-glycemic index, low-fiber breakfast affects the postprandial plasma glucose, insulin, and ghrelin responses of patients with type 2 diabetes in a randomized clinical trial. *J Nutr.* 2015; 145: 736–741.
35. Lobos DR, Vicuña IA, Novik V, Vega CA. Effect of high and low glycemic index breakfast on postprandial metabolic parameters and satiety in subjects with type 2 diabetes mellitus under intensive insulin therapy: Controlled clinical trial. *Clin Nutr ESPEN*. 2017; 20: 12–16.
36. Brand-Miller J, Hayne S, Petocz P, Colagiuri S. Low-glycemic index diets in the management of diabetes: a meta-analysis of randomized controlled trials. *Diabetes Care*. 2003; 26: 2261–2267.
37. Anderson JW, Randles KM, Kendall CWC, Jenkins DJA. Carbohydrate and fiber recommendations for individuals with diabetes: a quantitative assessment and meta-analysis of the evidence. *J Am Coll Nutr*. 2004; 23: 5–17.
38. Barclay AW, Petocz P, McMillan-Price J, Flood VM, Prvan T, et al. Glycemic index, glycemic load, and chronic disease risk--a meta-analysis of observational studies. *Am J Clin Nutr*. 2008; 87: 627–637.
39. Halton TL, Liu S, Manson JE, Hu FB. Low-carbohydrate-diet score and risk of type 2 diabetes in women. *Am J Clin Nutr*. 2008; 87: 339–346.
40. Greenwood DC, Threapleton DE, Evans CEL, Cleghorn CL, Nykjaer C, et al. Glycemic index, glycemic load, carbohydrates, and type 2 diabetes: systematic review and dose-response meta-analysis of prospective studies. *Diabetes Care*. 2013; 36: 4166–4171.
41. Ludwig DS. Dietary glycemic index and obesity. *J Nutr*. 2000;130: 280S-283S.
42. Pal S, Lim S, Egger G. The effect of a low glycaemic index breakfast on blood glucose, insulin, lipid profiles, blood pressure, body weight, body composition and satiety in obese and overweight individuals: a pilot study. *J Am Coll Nutr*. 2008; 27: 387–393.
43. Bouché C, Rizkalla SW, Luo J, Vidal H, Veronese A, et al. Five-week, low-glycemic index diet decreases total fat mass and improves plasma lipid profile in moderately overweight nondiabetic men. *Diabetes Care*. 2002; 25: 822–828.
44. Torrance GW, Feeny D, Furlong W. Visual analog scales: do they have a role in the measurement of preferences for health states? *Med Decis Making*. 2001; 21: 329–334.
45. Strader AD, Woods SC. Gastrointestinal hormones and food intake. *Gastroenterology*. 2005; 128: 175–191.
46. Jenkins DJ, Wolever TM, Kalmusky J, Guidici S, Giordano C, et al. Low-glycemic index diet in hyperlipidemia: use of traditional starchy foods. *Am J Clin Nutr*. 1987; 46: 66–71.
47. Philippou E, McGowan BMC, Brynes AE, Dornhorst A, Leeds AR, et al. The effect of a 12-week low glycaemic index diet on heart disease risk factors and 24 h glycaemic response in healthy middle-aged volunteers at risk of heart disease: a pilot study. *Eur J Clin Nutr*. 2008; 62: 145–149.
48. Miller JC. Importance of glycemic index in diabetes. *Am J Clin Nutr*. 1994; 59: 747S-752S.

49. Behall KM, Scholfield DJ, Yuhaniak I, Canary J. Diets containing high amylose vs amylopectin starch: effects on metabolic variables in human subjects. *Am J Clin Nutr*. 1989; 49: 337–344.
50. Botero D, Ebbeling CB, Blumberg JB, Ribaya-Mercado JD, Creager MA, et al. Acute effects of dietary glycemic index on antioxidant capacity in a nutrient-controlled feeding study. *Obesity (Silver Spring)*. 2009; 17: 1664–1670.
51. Bao J, Atkinson F, Petocz P, Willett WC, Brand-Miller JC. Prediction of postprandial glycemia and insulinemia in lean, young, healthy adults: glycemic load compared with carbohydrate content alone. *Am J Clin Nutr*. 2011; 93: 984–996.
52. Brand-Miller J, Buyken AE. The glycemic index issue. *Curr Opin Lipidol*. 2012; 23: 62–67.

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