Chapter 1

Effect of Quality Carbohydrates on the Prevention and Therapy of Noncommunicable Diseases: Obesity and Type 2 Diabetes

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Additional information is available at the end of the chapter

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Abstract

Glycemic index (GI) is defined as "how certain meals raise blood glucose after eating, expressed as a percentage of the area under the glucose response curve when the same amount of carbohydrate was consumed as glucose or bread." Glycemic load (GL) corrects GI according to the quantity of carbohydrates ingested. Both have been related to a higher risk of developing obesity and type 2 diabetes (DM2). High GI meals have been altered to create structurally similar meals with low GI levels. Observational studies and clinical trials have been developed using subjects with DM2 and subjects with obesity undergoing bariatric surgery. It was possible to lower the GI of meals, keeping the sensory properties of the original high GI preparation. Observational studies conducted on DM2 under treatment with metformin have shown associations between GI, GL and glycated hemoglobin. However, the same has not been proven with DM2 individuals under basal insulin therapy. Another observational study in subjects with obesity undergoing bariatric surgery showed that GI affects weight loss after surgery. Regarding experimental studies, a better glucose response has been seen following low GI breakfast intake in DM2 subjects undergoing intensive insulin therapy (IIT).

Keywords: glycemic index, glycemic load, meals, obesity, type 2 diabetes

1. Introduction

The most important macronutrient in the diet is carbohydrates (CHO). Therefore, their quality may significantly affect the health status of population.

A way to evaluate the healthy quality of carbohydrates (CHO) is through its ability to affect the glycemic and insulinemic response. Jenkins et al. proposed the glycemic index (GI), which ranks foods as having a high, moderate, or low glycemic effect, according to their response after food intake [1].

The GI is a methodology in vivo and is obtained by comparing the area under the curve of glycemia produced after the intake of 50 g of available CHO from a standard food to the glycemic curve produced after the intake of the same amount of CHO from an assay food. The resulting value is expressed as a percentage. As a standard, food used is glucose or white bread, corresponding to a value of 100 [2].

The researches have defined values of GI equal to or greater than 70 as high and values equal to or lesser than 55 as low [3, 4]. If it is also considered that high GI diets are associated with putting one at a greater risk for developing obesity and diabetes, then it is concluded that foods over 70 GI are not healthy, while those that have shown a GI lower than 55 are healthy and, therefore, recommended.

The quality and quantity of CHO affect the GI. As per the definition, the GI does not consider the quantity effect, as it only concerns 50 g of CHO from the assay food and the standard food. However, the exact quantity of CHO does not represent the way they are consumed, especially when related to meals with a variety of food.

The glycemic load (GL) quantifies the glycemic effect of the available CHO in a food portion that is generally similar to a normal intake size. The GL is obtained by multiplying the quantity of available CHO from a portion by the GI of the same food and then dividing by 100 [5]. Thus, the GL introduces the quantity variable of CHO and complements the GI.

High GL is expected to produce higher glycemic incursions. Diets characterized by high GL intake in the long term are associated with a higher risk of developing noncommunicable diseases (NCDs), such as type 2 diabetes (DM2) and obesity.

The GI may be affected by a variety of factors, the speed of starch digestion in the small bowel being the most important.

The digestion speed in vitro is a methodology that determines how fast the available starch presented in food is hydrolyzed by the digestive enzymes (alpha-amylase and amyloglucosidase) in an in vitro incubation. Rapidly digestible starch is hydrolyzed after a 20-min period and slowly digestible starch after a period of 120 min. The remnants not digested after this time are referred to as resistant starch [6].

Rapidly digestible starches are gelatinized starches, meaning they are found in food under wet cook and in autoclaved and extruded foods. Slowly digestible starches limit access to hydrolytic enzymes by virtue of their molecular structure. They are found in partially ground grains and seeds and in high-density foods such as pasta and raw meals. Resistant starch is found in raw foods or in whole grains and seeds where starches are physically inaccessible to enzymes. The retrograded starch formed by cooling gelatinized starch is part of this refractory fraction to the enzymatic action. Starch classification according to speed of digestion was an idea proposed by Englyst in his 1992 work *Classification and measurement of nutritionally important starch fractions*, in which he described four types of starch resistance [6].

The culinary techniques are used to prepare foods both in homes and industrially modify the proportions of fast/slow resistance digestive starch [7]. For example, foods that are sliced, chopped, and ground accelerate the speed of enzymatic digestion by allowing for additional surface contact between the enzymes and starch [8]. Cooking, especially in water, allows for the gelatinization of the starch, which significantly decreases the time enzymes take to break down starch and initiate bowel absorption [9, 10]. On the contrary, gelatinized starch, when cooled, produces a retrogradation phenomenon, where a part of starch have become resistant, resulting in a lower impact on the glycemic response [11–13]. Furthermore, the addition of fats and proteins to starch-rich foods decreases the speed of enzymatic digestion [14]. A boiled potato, for instance, has mainly gelatinized starches, meaning that its consumption produces high glycemic curves upon intake, which is then followed by a greater glycemic drop. If oil is added to the same boiled potato, the curve is soft in both its peak and its fall.

The application of diverse culinary techniques is indispensable in that it allows for the consumption of foods that in their raw state cannot otherwise be consumed. On the other hand, this also diversifies the meals in relationship to organoleptic characteristics and increases acceptability. This strengthens adherence to diets, a fundamental issue that allows for the success of, among other things, nutrition therapy in subjects with NCDs.

NCDs are the most important health burden currently in the world. The WHO recently defined obesity as a disease, and it is now considered as of the most important causes of other NCDs such as DM2, hypertension, and cancer, among others. Obesity is the result from a positive energetic balance over time, that is, when more energy is taken than is expended. Energy intake is regulated by many factors, among them dietetic variables such as GI and GL, which take part of an important role in continuing or discontinuing a given intake [15]. The following mechanisms can be used to explain the effect of GI on intake and its potential effects on control of body weight:

— **Release of small bowel peptides related to satiety**: several peptides released from the small bowel (jejunum) in response to the presence of nutrients (especially glucose) signal satiety. Within the identified peptides, GLP-1 has the notable ability to produce satiety signals related to the CHO intake. Slow digestion associated with the intake of slow GI foods will produce a higher contact time between nutrients and bowel cells, as well as an increase in the production of GLP-1 [16].

— **Postprandial hyperinsulinemic effect**: A high GI food may trigger an increment in the postprandial glycemic curve that is two times stronger than that of food containing the same quantity and distribution of macronutrients but with low GI (e.g., white bread versus spaghetti). This hyperglycemic state triggers a hyperinsulinemic effect, which in turn raises the insulin-glucagon rate, resulting in an increase in the anabolic response and the production of expanded glycogenesis and lipogenesis [17].

— **Glucostatic theory**: the beginning of a meal is described as response behavior when the brain detects signs of energy deficiencies, the natural signal corresponds to a decrease of the glycemia. The high glycemic response of high GI meals, followed by an abundant secretion of insulin, would produce a fast fall in the postprandial glycemic curve, which

would contribute to the hungry feeling in the short term. While this theory may explain the mechanism of the effect of the GI with regard to the satiety, some research has shown weak associations between the glycemic response and food intake. Therefore, more studies are necessary to support this mechanism [18].

A review of 19 studies encompassing a total of 248 healthy subjects, in which meals with similar amounts of energy and macronutrients, but varying GI levels, were evaluated, proved that at following low GI food intake, satiety and satiation levels increased more than it did following high GI food intake [19]. Another prospective epidemiologic study with a follow-up of 12 years to 89,432 individuals showed that every 10-point GI increase in one's diet led to an average weight gain of 34 g per year [20]. On the other hand, a randomized clinical study that included 34 overweight and obese individuals who consumed either a high or low GI hypocaloric diet over 4 months showed that those receiving low GI diets lost significantly more weight compared to those receiving high GI diets [21].

A review published in 2007 in the *Cochrane* database that looked at six randomized controlled studies totaling 202 obese subjects who were undergoing low GL or low GI diets concluded that low GI diets have a significantly more profound effect on weight loss than do low GL diets [22].

Despite the studies cited above, the relationship between the glycemic response to food intake and weight control is not currently clear due to the lack of studies measuring glycemia, appetite, and food intake in the long term, as well as contradictory findings in researches and reviews [23].

It is known that being overweight is a risk factor for the development of DM2, making this a public health issue. DM2 affects about 366 million people worldwide, a number that is expected to reach 552 million by the year 2030 [24]. As GI and GL play an important role in the metabolic control of subjects with DM2, Ludwig proposed hypothetic metabolic pathways that explain how high GI meals may affect glucose metabolism, irrespective of its effect on the body weight. Experimental studies performed on animals and humans showed that the high postprandial glycemic curve would produce an increase in oxidative stress which directly affected the functionality of beta cells in the pancreas. These studies also showed that the postprandial hyperinsulinemic alone also affects the functioning of the beta cell. Finally, he noted that the rapid fall of the glycemia observed following the quick increase of the glycemic curve as a consequence of high GI food intake would produce the release of contra-regulation hormones in a state similar to fasting, increasing the free fatty acids in plasma which affect the beta cells as well as the insulin receptor. Therefore three conventional mechanisms are described: glucotoxicity, pancreatic overstimulation, and lipotoxicity [17].

A variety of studies indicate that dietary intake of high GI foods is associated with an increase in the risk of developing obesity and DM2 along with cardiovascular diseases seeing as the hyperglycemia and the hyperinsulinemia are related to this kind of food intake [25–27]. On the other hand, Brand Miller et al. developed a meta-analysis to determine the effect of low GI diets on the glycemic control in subjects with DM2, where the subjects studied showed positive effects to fructosamine and glycated hemoglobin (A1c) levels [28].

A systematic review from *Cochrane* that included 11 randomized controlled studies indicated that metabolic control improves significantly as the result of a low GI diet, producing a reduction of 0.5% of A1c, similar to that obtained with drug therapy [29]. This reduction is important, considering that The UK Prospective Diabetes Study (UKPDS) group found that the 1% decrease of A1c is associated with a 37% decrease in the risk of developing microvascular complications [30].

Finally, the postprandial glucotoxicity would stimulate the expression of pro-inflammatory genes through epigenetic mechanisms which respond to particular dietetic intakes, in this case, through the histone methylations which stimulate the inflammatory gene expression related to release of free radicals in the mitochondrial level [31]. The pre-inflammatory state observed in subjects with DM2 would increase insulin resistance and the loss of secretory functions in pancreas beta cells. Additionally, it has been named as a cause related to the development of vascular complications. Therefore, suggested dietetic guidelines would regulate this state throughout nutrition therapy [32].

2. Body: research methods

2.1. Glycemic index and its effect on sensorial acceptability of commonly consumed meals in Chile

There are a variety of factors that affect glycemic index (GI), such as food origin or culinary techniques used in meal preparation. On this basis, it is important to obtain the in vivo GI from commonly consumed meals in order to have more reliable data about quality of carbohydrates (CHO) consumed by the local population.

Chilean typical meals (n = 6) were measured in 10 young and healthy subjects using the techniques described by Jenkins et al. in order to calculate the GI using experimental methods. The results obtained are shown in **Table 1** [33].

The results achieved from meals composed of rice and meat show a significantly lower GI than those found in other studies [34]. It could be explained by the local culinary techniques,

Meals	Glycemic index mean ± SD
Rice-grounded meat	31.4 ± 28.4
Spaghetti-grounded meat	42.0 ± 23.4
Lentil soup	49.3 ± 29.5
Bean-spaghetti	76.8 ± 43.4
Mashed potatoes-grounded meat	51.0 ± 29.2
Carbonada (veggie beef soup)	82.1 ± 48.7

Source: Araya H, Contreras P, Vera G, Alviña M, Pak N. Eur J Clin Nutr 2002, 56:753-9.

Table 1. Glycemic index of commonly consumed meals in Chile.

as rice is fried in oil before boiling, leading to a decrease in the starch's water absorption capacity. On the other hand, the meals made from beans and pasta show much higher value than those already known [34, 35]. The reason that would explain these findings is the practice of overcooking that is commonly implemented in the Chilean kitchen.

On the other hand, a study on the basis of theoretical GI calculated for subjects with type 2 diabetes (DM2) from high acceptability meals commonly consumed in Chile showed that diet GI was 74.9 ± 11.3 in these individuals [36]. According to an unpublished study, the mean acceptability for the same meals was 6.26 on a scale of 1–7, 7 standing for "I like too much." Therefore, planning low GI meals presents a methodological challenge: keeping the organoleptic characteristics similar to those with high GI in order to ensuring good acceptability. With this aim on mind, a current study planned a low GI lunch for each traditional high GI lunch (n = 10) in an effort to keep the meals similar. These homologue meals were cooked with essentially the same ingredients but using different culinary techniques or including different

	High glycemic index lunches			**Low glycemic index lunches**		
Main food of main course	**Description**	**GI[1]**	**CHO[2] (g)**	**Description**	**GI**	**CHO (g)**
Tubers (potatoes)	Celery with boiled corn kernels	121.1	52.6	Celery with raw carrot	54.8	46.3
	Cazuela de albóndigas ("veggie and meatball soup": no skin boiled potatoes, short white pasta, and meat)			*Cazuela de albóndigas* ("veggie and meatball soup": boiled potatoes with skin, whole rice, and meat)		
	Macedonia (fruits: kiwi, banana, and orange)			*Macedonia* (fruits: pear, apple, and orange)		
Cereal (rice)	Boiled vegetables	100.3	51.9	Raw vegetables	49.0	51.9
	Meat with boiled white rice and vegetables			Meat with fried brown rice and vegetables		
	Diet jelly			Diet yogurt		
Legumes (lentils)	Tomato with boiled corn kernels	62.1	56.1	Tomato with raw onion	38.1	54.8
	Lentils with boiled potatoes and grated bread			Lentil with brown rice		
	Baked apple			Apple with beaten egg white		
Cereal (wheat)	Cucumber, tomato, and boiled fava beans	68.5	59.8	Cucumber and tomato	46.9	50.7
	Spaghetti with Bolognese sauce			Whole spaghetti with Bolognese sauce		
	Boiled pear with diet jam			Diet yogurt with apple		
Vegetables (zucchini)	Boiled corn kernels with boiled grated carrots	82.5	47.6	Cabbage with sliced raw carrots	44.7	44.7
	Zucchini pudding with grated white bread			Zucchini Soufflé (bread free)		
	Kiwi with orange juice			Diet yogurt with oat and apple		

[1]GI: glycemic index.
[2]CHO: carbohydrates.
[3]Unpublished data.

Table 2. High-g3lycemic-index lunches commonly consumed in Chile and their homologue low-glycemic-index lunches[3].

amounts of each ingredient based on the digestive speed of its starch. All preparations consisted of salad, a main course, and a dessert, with all of the components providing between 50–60 g of available CHO together, an amount theoretically calculated using exchange portions and the chemical composition table from Chilean food pyramid [37, 38]. The GI values were obtained from Brand Miller et al. using white bread as standard food [34].

Table 2 (unpublished data) shows some homologue lunches and their GI values. It is possible to identify that the most significant changes to the meals are reflected in alterations made to salads and desserts. The amounts of ingredients in homologue meals were modified at convenience if a high or low GI lunch was being planned. White cereals were replaced for whole-grain cereals; in addition, fruits and tubers with skin were included based on their insoluble fiber content as this type of fiber acts as a physical obstacle to enzymatic action, meaning digestion speed may be slowed down [35]. Whole foods were replaced by sliced, ground, or grated foods in order to achieve lower GI as these culinary techniques increase surface contact between food and digestive enzymes, subsequently increasing speed of starch digestion and thereby increasing the GI of meals [39]. In addition, in an attempt to decrease the speed of digestion, oil was added when the starch of certain foods suffered gelatinization. Furthermore, wet boiled was used to cook high GI meals in a longer period than low GI meals. Conversely, in order to keep the low GI of meals, dry cooking (e.g. baking) or brief boiling was implemented. Additionally, low GI foods replaced certain similar high GI foods; uncooked oats, for example, replaced bread.

The high GI lunches measured between 62 and 121, while the low GI lunches were between 38 and 55.

In order to verify if low GI lunch acceptability equaled that of the high GI lunches, a seven-point hedonic scale was applied among a non-probabilistic sample of 25 women similar to female population with DM2. As noted in **Table 3** (data not published), an average value

Similar lunches[1]	**Lunch acceptability**		
	High GI[2,3]	**Low GI**	**P**[4]
Salad, *Cazuela de Vacuno* (*veggie and meat soap*), fruit with milk dessert	6.5 ± 0.7	6.7 ± 0.6	NS
Salad, *Cazuela de Albóndigas* (*veggie and meat balls soap*), *Macedonia* (variety of Mediterranean fruits)	6.6 ± 0.5	6.5 ± 0.8	NS
Boiled vegetables, fish with potatoes/brown rice, fruit	6.3 ± 0.9	6.2 ± 0.8	NS
Salad, meat with mashed potatoes/mashed potatoes and pumpkins, fruit	6.0 ± 1.0	5.8 ± 0.9	NS
Boiled vegetables/raw vegetables, zucchini pudding, fruit/oat	6.3 ± 0.8	6.6 ± 0.6	NS
Salad, spaghetti, fruit/diet yogurt with fruit	6.2 ± 0.7	6.6 ± 0.7	NS
Salad, chicken with boiled vegetables, fruit/oat	6.5 ± 0.7	5.9 ± 0.8	0.008
Tomato, lentils, apple	6.2 ± 0.6	6.1 ± 1.2	NS
Potatoes salad, fish pudding, milk dessert	6.5 ± 0.6	6.5 ± 0.8	NS
Boiled vegetables/raw vegetables, rice with meat, diet dessert	6.4 ± 0.9	6.5 ± 0.8	NS

[1]Slash (/): divide foods used in low or high GI lunches.
[2]GI: glycemic index.
[3]Data are means ± SD.
[4]Wilcoxon test, significant differences at $p < 0.05$.
[5]Unpublished data.

Table 3. Acceptability of similar high- and low-glycemic-index lunches, according to hedonic scale (n = 20)[5].

of six points was achieved by the lunches evaluated, representing moderate to high acceptability. In most cases, no significant differences were found between high and low GI meals.

2.2. Relationship between glycemic index, glycemic load, and weight control in subjects with obesity

Glycemic index (GI) and glycemic load (GL) are two dietetic factors that affect food intake and, therefore, explain the weight control effect. Subjects undergoing bariatric surgery (BS) generally see a decrease in the rate of weight loss 6 months after surgery and generally start to regain weight one year following surgery [40]. Therefore, it is important to ask the following: Which factors impact the speed of weight loss or weight regain in obese subjects that have undergone BS?

A prospective descriptive study was conducted to evaluate the relationship between the carbohydrate (CHO) intake and weight loss after surgery in individuals undergoing BS. Twenty women undergoing gastric bypass (GBP) (BMI, 42.02 ± 3.9 kg/m^2; age, 36.9 ± 8.4 years) and twenty women undergoing sleeve gastrectomy (BMI, 37.4 ± 2.9 kg/m^2; age: 33.4 ± 8.5 years) were studied 6 and 12 months following surgery. GI and GL averages were assessed using a 3-day dietary record, and the percentages of excess weight loss (PEWL) and weight loss compared to that lost before surgery (ΔW) were measured. GI and GL averages found at 6 months after surgery were 47.6 ± 6.4 and 45.7 ± 16.6 g, respectively. Twelve months following surgery, the GI of the diet was 52.7 ± 6.7 and GL 63.5 ± 22.3 g. Data showed significant weight loss at 6 months after surgery (ΔW, 27.6 ± 4.6%; PEWL, 67.1 ± 14.6%) and at 12 months after surgery (ΔW, 32.3 ± 7.3%; PEWL, 78 ± 20.1%). As suggested in **Figure 1** (unpublished data), the GI diet showed a significant correlation with PEWL at 6 months after surgery. **Figure 2** (unpublished data) describes the association between the GI diet and PEWL at 12 months after surgery.

The GI showed a significant negative correlation with ΔW ($r = -0.42$; $p = 0.008$) six months after surgery, but this correlation is nevertheless annulated 12 months after surgery [41]. No considerable correlation was found when GL and ΔW were analyzed 6 and 12 months after surgery. It would appear that the quality of CHO impact is stronger than CHO quantity over the weight control, according to *Cochrane* review [22].

When performing multiple regression analysis including all independent variables studied (energy sufficiency, CHO, GI, CHO of high GI, GL) as well as dependent variables PEWL and ΔW six and twelve months after surgery, the only variable that showed association was diet GI. This index explains 19% of the variability of PEWL six months after surgery ($r^2 = 0.189$, $p = 0.005$) and 17% of the variability of ΔW ($r^2 = 0.172$, $p = 0.008$). On the other hand, GI explains 11% of the variability of PEWL one year after surgery ($r^2 = 0.106$, $p = 0.04$).

In conclusion, in this group of women undergoing BS that was evaluated 6 and 12 months after surgery (SG and GBP), GI of the diet showed a negative significant association with weight loss, a trend that was most notable 6 months after surgery. On the other hand, the quantity of CHO and GL in the diet did not show any association at all with weight loss in either of the periods studied [41].

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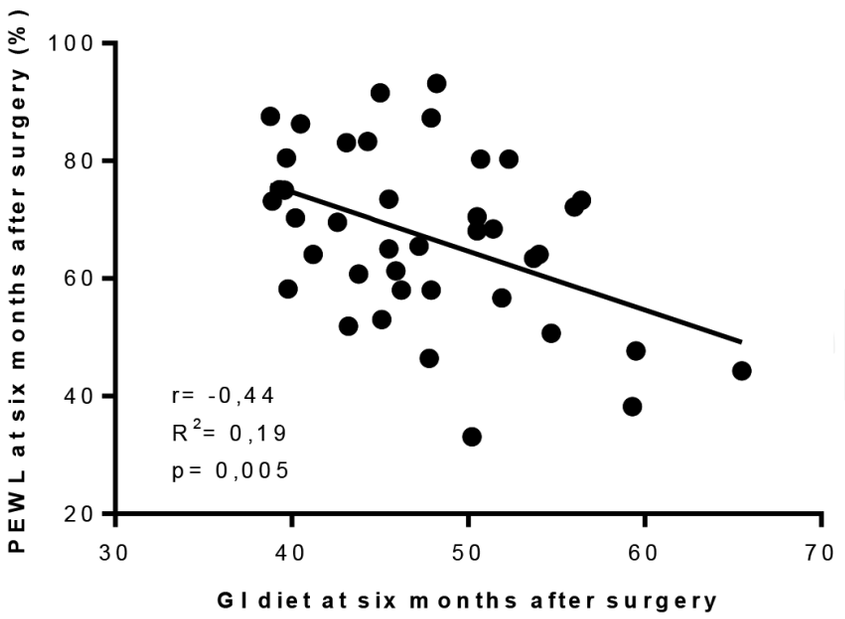
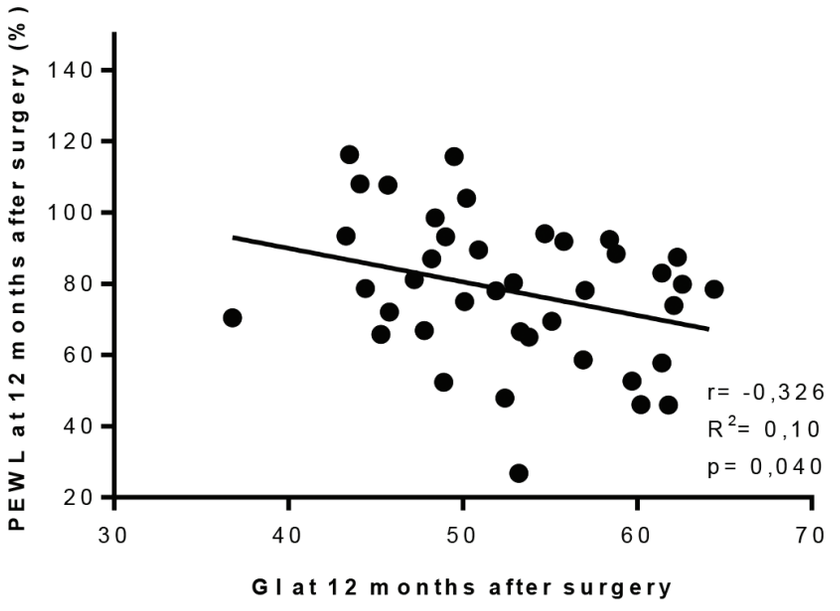


PEWL: percentage of excess weight loss GI: glycemic index
Pearson´s correlation test
[1]Unpublished data

Figure 1. Association between glycemic index diet and percentage of weight loss 6 months after surgery (n = 40)[1].



PEWL: percentage of excess weight loss GI: glycemic index
Pearson´s correlation test
[1]Unpublished data

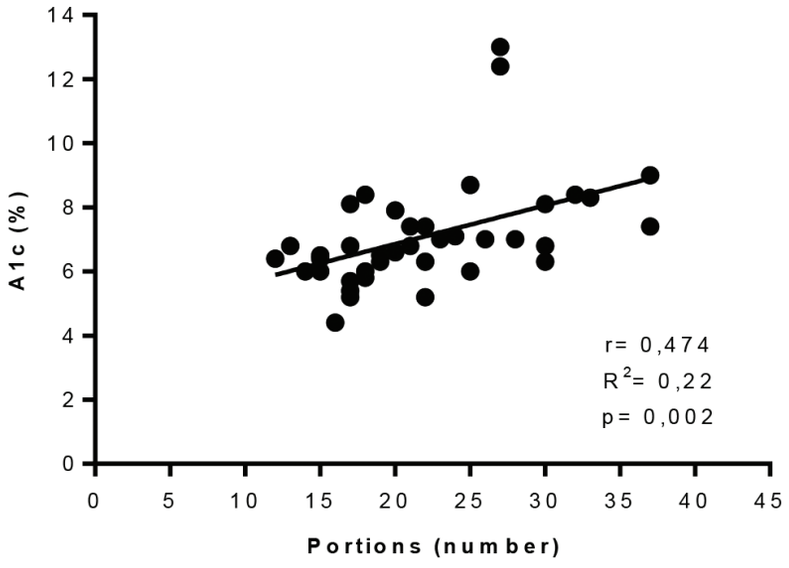
Figure 2. Association between glycemic index diet and percentage of weight loss 12 months after surgery (n = 40)[1].

2.3. Relationship between glycemic index, glycemic load, and metabolic control in subjects with type 2 diabetes

Nutrition therapy plays a key role in metabolic control of type 2 diabetes (DM2) and in the prevention of future diabetes-related complications [42]. Considering the controversial findings shown by previous studies regarding the usefulness of the glycemic index (GI) or glycemic load (GL) in nutrition therapy for DM2 and in order to determine the impact of the quality of carbohydrates (CHO) in the Chilean diet on metabolic parameters in subjects with DM2 under metformin therapy, two transversal descriptive studies were developed. The first one studied 40 individuals with DM2 (age, 58.6 ± 9.5 years; BMI, 32.5 ± 5.8; A1c, 7.08 ± 1.6), in which the subjects were examined through modified frequency food surveys to measure the average amount of usual high GI food portion intake. The findings showed an intake of 403.8 ± 110.7 g of CHO per day. The average GI and GL found were 78.5 ± 3.5 and 317.5 ± 88.3, respectively. The amount of high GI food intake was 21.8 ± 6.5. The association between glycated hemoglobin (A1c) and the amount of high GI food intake is shown in **Figure 3** [43].

The second descriptive study evaluated 108 subjects with DM2 under metformin therapy (age, 53.6 ± 9.45 years; BMI, 30.8 ± 5.8 kg/m^2; A1c, 7.3 ± 1.3 %), all of which were examined through two separate 24-h recall surveys to measure the GI and GL amount in the diet. The findings showed an average CHO intake of 219.8 ± 27.0 grams of CHO per day. Diet GI was 74.9 ± 11.3 and diet GL was 164.0 ± 22.04 g per day. Positive significant associations were found between GI diet and A1c (r, 0.7; $p < 0.05$) and GL and A1c (r, 0.225; $p < 0.05$) [36].

Another research conducted in our department evaluated 40 subjects with DM2 (age, 61.8 ± 8.03 years; BMI, 29.9 ± 4.7 kg/m^2; A1c, 8.6 ± 1.8%) under basal insulin therapy in order



Pearson´s correlation test A1c: glycated hemoglobin
Source: Varela N, Valenzuela K, Vega C. Arch Latinoam Nutr 2012; 62(1):24-29

Figure 3. Correlation between glycemic index food portion intake and A1c in subject with type 2 diabetes under metformin therapy.

to find the relationship between quality of available CHO in every mealtime and metabolic parameters. Individuals were evaluated using 3-day dietary record surveys to identify mealtime GI and GL levels in the diet. Mean fasting glycemia of 163 ± 70 mg dl^{-1} and pre- and postprandial glycemia of 180 ± 73 and 209 ± 83 mg dl^{-1} were found, respectively, and bedtime glycemia was 221 ± 81 mg dl^{-1}. GI and GL of meal time are shown in **Table 4** [44].

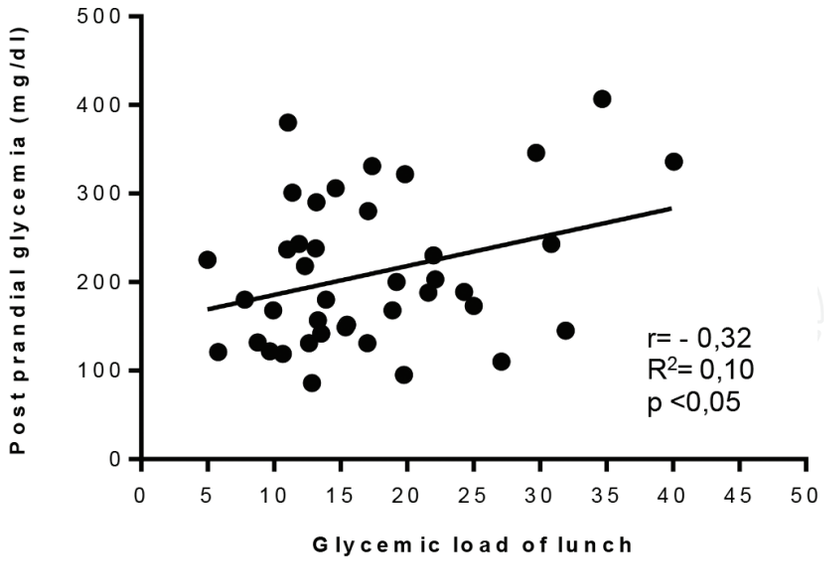
The significant associations found in this study are shown in **Figures 4** and **5**. Nonsignificant associations were found for other analyzed variables.

On the basis of these studies, it is possible conclude that, as is widely accepted, the 24-h recall surveys tend to underestimate the food intake and, conversely, the food frequency surveys tend to overestimate the food intake. On the other hand, the GI and GL are associated with metabolic parameters related to DM2 control; nevertheless, this association is weaker when applied in subjects with DM2 under basal insulin therapy.

Variable	Breakfast (n = 40)	Lunch (n = 40)	"Tea time" (n = 38)	Dinner (n = 25)	Snacks (n = 33)
Glycemic index	64.1 ± 8.8[a]	44.7 ± 7.6[b]	*69.3[a] (4.1–4.2)	49.6 ± 9.0[c]	45.6 ± 11.8[bc]
Glycemic load (g)	26.2 ± 11.9[a]	17.3 ± 8.1[b]	32.6 ± 18.7[c]	11.0 ± 8.5[d]	13.1 ± 9.3[d]

Data are expressed as mean ± SD. *Median (Q1–Q3).
A repeated measure ANOVA was used to achieve differences between meal times.
a, b, c, and d: different upper letters show significantly difference between every mealtime ($p < 0.05$).
Source: Sambra et al. [44].

Table 4. Quality of diet carbohydrates in every meal time.



Pearson´s correlation test
Source: Sambra V, Tapia C, Vega C. Nutr Hosp 2015; 31(4):1566-1573

Figure 4. Correlation between postprandial glycemia and glycemic load of lunch in subject with type 2 diabetes under basal insulin therapy (n = 40).

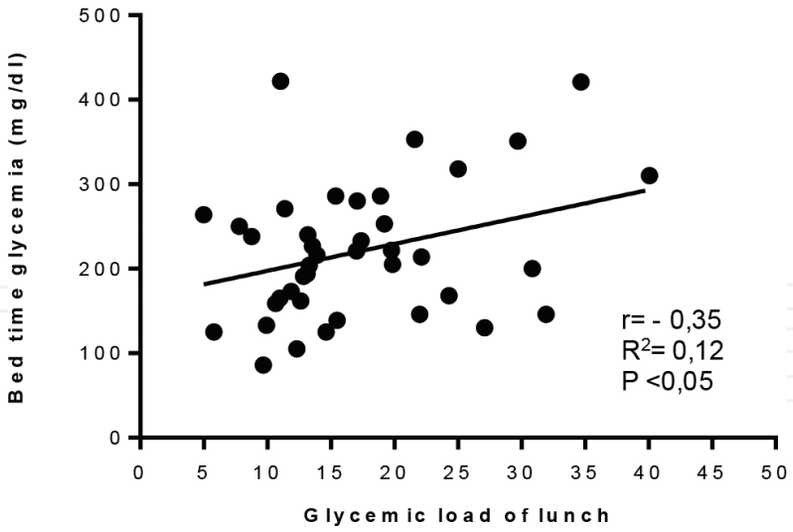


Figure 5. Correlation between bedtime glycemia and lunches glycemic load in subject with type 2 diabetes under basal insulin therapy (n = 40).

2.4. Effect of different GI meal intake over glycemic response and satiety in subjects with obesity, insulin resistance, and type 2 diabetes

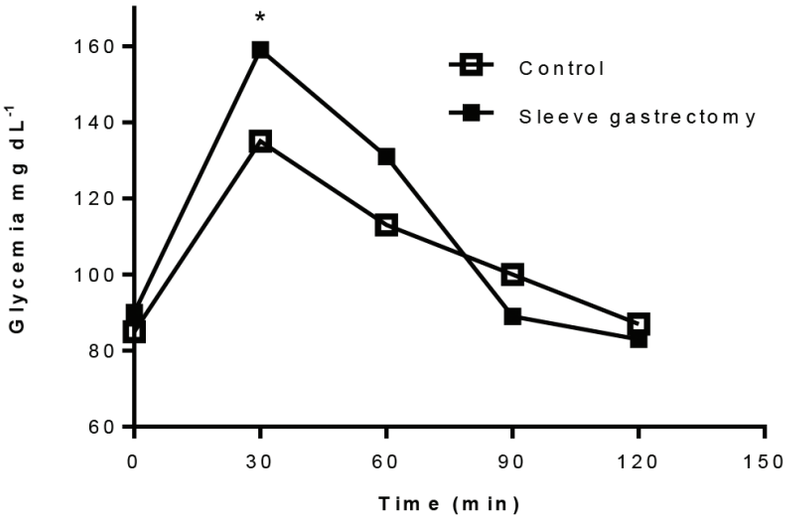
2.4.1. Effect of high glycemic index food intake on obese subjects undergoing sleeve gastrectomy

The sleeve gastrectomy is a surgery procedure that aims to decrease gastric capacity [45]. The weight regained one year after surgery is normally found in these kinds of individuals due to an increase in gastric capacity and, hence, a decrease in satiety [40]. Consequently, it is important to consider the qualitative indexes diet, especially those from carbohydrates (CHO) because, according to the last food intake survey (2010–2011) conducted in Chile, this is the main component of energy intake [46]. In order to describe and compare the glycemic response (GR) and glycemic index (GI) in 10 subjects with obesity one year after sleeve gastrectomy versus 10 healthy subjects, a research was conducted. **Figure 6** shows a higher glycemic response 30 min after instant mashed potato intake among subjects undergoing bariatric surgery, as opposed to healthy subjects (SG, 159.8 ± 25.9 mg dl^{-1}; healthy subjects, 135.3 ± 17.3 mg dl^{-1}; p = 0.023). However, the GI value calculated from instant mashed potatoes was similar in both studied groups (instant mashed potatoes GI in subjects undergoing SG, 119; instant mashed potatoes GI in healthy subjects, 120; p = 0.974) [47].

In conclusion, instant mashed potatoes, despite its similar GI in both groups studied, produce greater glycemic response; therefore, the intake of this food achieves an outcome contrary to the surgery objectives, as it stimulates weight regain.

2.4.2. Effect of white or whole-wheat pasta intake in subjects with insulin resistance

The insulin resistance (IR) is a metabolic condition that allows for the maintenance of normal postprandial glycemia at the expense of a raise in insulin synthesis from the pancreas. In this condition, pro-inflammatory factors, free fatty acids in plasma or ectopic fat, generate damage to the insulin receptor [48]. Additionally, there is a genetic predisposition that contributes



* p<0.05 by one-way ANOVA

Source: Fuentes G, Del Valle M, Vega C. Nutr Hosp 2014; 30(6):1263-1269

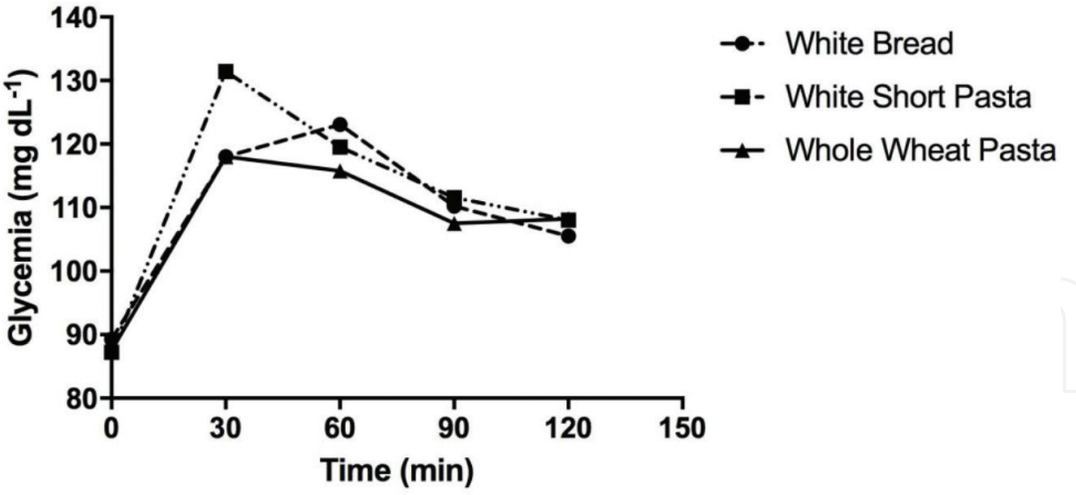
Figure 6. Instant mashed potato glycemic response in subject undergoing sleeve gastrectomy (n = 20, control group, 10; Sleeve gastrectomy, 10).

to the development of this condition. In order to assess the glycemic response and GI after white and whole-wheat pasta intake, 10 healthy subjects and 10 individuals with insulin resistance were studied. All participants ate 50 g of available CHO from two types of pasta (white or whole wheat) with equal morphologic characteristics (short pasta) that were subjected to the same culinary technique. No considerable differences in the glycemic response between groups were found in this study for either type of short pasta. On the other hand, **Figure 7** shows a substantial difference found 30 min after intake of white versus whole-wheat pasta which only manifests itself among subjects with insulin resistance (unpublished data) [49].

These findings prove that the insulin-resistance condition meets the metabolic demands of foods, regardless of their GI values. Nevertheless, individuals with insulin resistance would benefit from whole-wheat pasta intake as it invokes a significantly lower glycemic response than does white pasta.

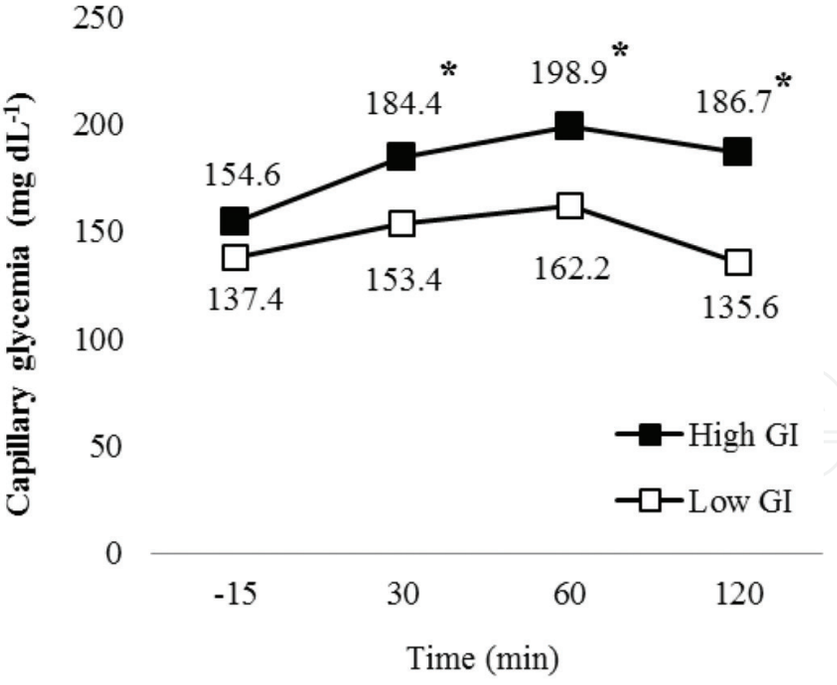
2.4.3. Effect of low and high glycemic breakfast on glycemic response and satiety in subjects with type 2 diabetes under intensive insulin therapy

There is not currently enough consistent evidence about the effect of low GI meal intake in individuals with DM2 under intensive insulin therapy (IIT). A controlled, crossover, single-blind study was conducted in order to determine the effect of low GI breakfast on glycemic response and satiety in 10 subjects (age, 57 ± 8 years; BMI, 34.9 ± 2.0; A1c, 8.9 ± 0.8) with DM2 under IIT. The findings described in **Figure 8** show that low GI breakfast intake achieves a postprandial glycemia significantly lower than high GI breakfast intake (unpublished data) [50].



*p<0.05 at 30 minutes after food intake by repeated measures ANOVA.
[1]Unpublished data

Figure 7. Glycemic response after intake of white and whole-wheat short pasta in subject with insulin resistance (n = 10)[2].



*p<0.05 by paired Student´s test. GI: glycemic index
[1]Unpublished data

Figure 8. Capillary glycemic response after intake of low or high glycemic index breakfast in subjects with type 2 diabetes under intensive insulin therapy (n = 10)[2].

Variable[1,3]	High GI breakfast	Low GI breakfast	P[2]
Hungry	0.0–1.9 (0.80)	0.0–1.0 (0.80)	NS
Satiation	5.2–10.0 (9.20)	9.1–10.0 (9.20)	NS
Fullness	5.1–10.0 (9.00)	8.3–10.0 (9.20)	NS

[1]Data are expressed as range and median. GI, glycemic index.
[2]Paired t-Student's test.
[3]Unpublished data.

Table 5. Satiation in subjects with type 2 diabetes under intensive insulin therapy after low- or high-glycemic-index breakfast intake (n = 10).

Variable[1,3]	High GI breakfast	Low GI breakfast	P[2]
Hungry	0.0–4.0 (1.00)	0.0–5.0 (0.80)	NS
Satiety	2.9–10.0 (8.10)	5.0–10.0 (9.20)	NS
Fullness	4.9–10.0 (8.10)	1.9–10.0 (9.20)	NS

[1]Data are expressed as range and median. GI, glycemic index.
[2]Paired t-Student's test.
[3]Unpublished data.

Table 6. Satiety of subjects with type 2 diabetes under intensive insulin therapy after low- or high-glycemic-index breakfast intake (n = 10).

After evaluating satiety through an analogue visual scale of 10 cm, it was found that subjects who ate a low GI breakfast reported fewer signs of hunger and more fullness immediately after eating than did subjects who consumed a high GI breakfast intake. Nevertheless no significant differences were found (**Table 5**, data no published). Two hours after low GI breakfast intake, individuals stated feeling less hunger and more satiety than high GI intake, although no significant differences were found (**Table 6**, unpublished data).

Regarding the findings described, it is possible to conclude that the low GI breakfast intake achieves significantly lower postprandial glycemic responses, greater satiety, and less hunger than does high GI breakfast intake in individuals with DM2 under intensive insulin therapy.

3. Conclusion: key results

There is currently limited data that describes in vivo GI values of meals commonly consumed in Chile. According to the above findings, it is possible to conclude that local culinary techniques affect GI values, as this study established a difference with the data published until the present. This finding ratifies the importance of reporting local values in order to account authentically data regarding the quality of carbohydrates consumed by population and its corollary effect over health.

With regard to high/low GI homologue meals, it has proven possible to produce a strong impact on the theoretically estimated GI value of various meals by making adjustments regarding the type and proportions of ingredients, as well as culinary techniques applied. These interventions maintained the organoleptic properties of the high GI meal in order to ensure adherence, as was demonstrated by findings acquired.

With regard to the effect of GI and GL on obesity, it is possible to conclude that GI shows a greater impact than does GL in weight loss in obese subjects undergoing bariatric surgery, a trend that is more notable 6 months following surgery than it is 12 months following surgery. In type 2 diabetes (DM2) under metformin therapy, both GI and GL affect the metabolic control in subjects studied. Those findings are different in subjects with DM2 under basal insulin therapy, where GL showed a greater impact on glycemic control that did GI.

Finally, the clinical trials developed establish that high GI meals/food achieves greater glycemic response in subjects with obesity undergoing sleeve gastrectomy, insulin resistance, or DM2 under intensive insulin therapy, especially 30 min following food intake.

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References

[1] Jenkins D, Wolever T, Taylor R, et al. Glycemic index of food a physiological basis for carbohydrate exchange. Am J Clin Nutr 1981;**34**:362-366.

[2] Mathers J, Wolever T. Digestion and metabolism of carbohydrates. In: Gibney M, Voster H, Kok F, editors. Introduction to Human Nutrition. 1st ed. Oxford, UK. Blackwell Science; 2002. pp. 69-80.

[3] Levitan E, Mittleman M, Hakansson N, Wolk A. Dietary glycemic index, dietary glycemic load, and cardiovascular disease in middle-age and older Swedish men. Am J Clin Nutr. 2007;**85**:1521-1526.

[4] Hare-Bruun H, Flint A, Heitmann BL. Glycemic index and glycemic load in relation to changes in body weight, body fat distribution, and body composition in adult Danes. Am J Clin Nutr. 2006;**84**:871-979.

[5] Brand-Miller JC. Glycemic load and chronic disease. Nutr Rev. 2003;**61**:S49-S55.

[6] Englyst HN, Kingsman SM, Cummings JH. Classification and measurement of nutritionally important starch fractions. Eur J Clin Nutr. 1992;**46**:S33-S50.

[7] Osorio-Díaz P, Bello-Perez LA, Sáyago-Ayerdi SG, Benítez-Reyes MP, Tovar J, Paredes-Lopez O. Effect of processing and storage time on in vitro digestibility and resistant starch content of two bean (*Phaseolus vulgaris* L). J Sci Food Agric. 2003;**83**:1283-1288.

[8] Aguilera JM. Why food microstructure? J Food Eng. 2005;**67**:3-11.

[9] Chung H, Lim HS, Lim S. Effect of partial gelatinization and retrogradation on the enzymatic digestion of waxy rice starch. J Cereal Sci. 2006;**43**:353-359.

[10] Shin SI, Kim HJ, Ha HJ, Lee SH, Moon TW. Effect hydrothermal treatment on formation and structural characteristics of slowly digestible non-pasted granular sweet potato starch. Starch-starke. 2005;**57**:421-430.

[11] Srikaeo K, Furst JE, Ashton JF, Hosken RW. Microstructural changes of starch in cooked wheat grains as affected by cooking temperatures and times. Food Sci Technol-LEB. 2006;**39**:528-533.

[12] Giacco R, Brighenti F, Parillo M, et al. Characteristics of some wheat-based foods of Italian diet in relation to their influence on postprandial glucose metabolism in patients with type 2 diabetes. Brit J Nutr. 2001;**85**:33-40

[13] Rosini MP, Lajolo MF, Menezes WE. Measurement and characterization of dietary starches. J Food Comp Anal. 2002;**15**:367-377.

[14] Berti C, Riso P, Monti LD, Porrini M. In vitro starch digestibility and in vivo glucose response of gluten-free foods and their gluten counterparts. Eur J Nutr. 2004;**43**:198-204.

[15] Galgani J and Ravussin E. Principles of human energy metabolism. In: Ahima R S, editor. Metabolic Basis of Obesity. 1st ed. Philadelphia, USA: Springer; 2011. pp. 1-24.

[16] Gutzwiller JP, Goke B, Drewe J et al. Glucagon-like peptide-1: a potent regulator of food intake in humans. Gut. 1999;**44**(1):81-86.

[17] Ludwig D. The glycemic index physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. JAMA. 2002;**287**(18):2414-2423.

[18] Mayer J. Glucostatic mechanism of the regulation of food intake. N Engl J Med. 1953;**249**:13-16.

[19] Bornet FR, Jardy-Gennetier AE, Jacquet N, Stowell J. Glycaemic response to foods: impact on satiety and long-term weight regulation. Appetite. 2007;**49**(3):535-553.

[20] Du H, van der A DL, van Bakel MME, et al. Dietary glycaemic index, glycaemic load and subsequent changes of weight and waist circumference in European men and women. Int J Obes. 2009;**33**:1280-1288.

[21] Wolever, TMS, Mehling C. Long-term effect of varying the source or amount of dietary carbohydrate on postprandial plasma glucose, insulin, triacylglycerol, and free fatty acid concentrations in subjects with impaired glucose tolerance. Am J Clin Nutr. 2003;**77**(3):612-621.

[22] Thomas D, Elliott EJ, Baur L. Low glycaemic index or low glycaemic load diets for overweight and obesity. In: Cochrane Database of Systematic Reviews. 3rd ed. John Wiley and Sons, Ltd; 2007. p. CD005105.

[23] Anderson GH, Woodend D. Effect of glycemic carbohydrates on short-term satiety and food intake. Nutr Rev. 2003;**61**(Suppl. 5):S17-S26.

[24] Aguiree F, Brown A, Cho NH et al. IDF Diabetes Atlas. 6th ed. Basel, Switzerland: International Diabetes Federation; 2013. 155p.

[25] Salmeron J, Manson JAE, Stampfer MJ. Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. JAMA. 1997;**277**(6):472-477.

[26] Maki KC, Rains TM, Kaden VN, Raneri KR, Davidson MH. Effects of a reduced-glycemic-load diet on body weight, body composition, and cardiovascular disease risk markers in overweight and obese adults. Am J Clin Nutr. 2007;**85**(3):724-734.

[27] Liu S, Manson JE, Stampfer MJ, et al. Dietary glycemic load assessed by food-frequency questionnaire in relation to plasma high-density-lipoprotein cholesterol and fasting plasma triacylglycerols in postmenopausal women. Am J Clin Nutr. 2001;**73**(3):560-566.

[28] 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**(8):2261-2267.

[29] Thomas D, Elliot E. Low glycaemic index, or low glycaemic load, diets for diabetes mellitus. In: Cochrane Database of Systematic Reviews, editor. The Cochrane Library. John Willey and Sons; 2009.

[30] United Kingdom Prospective Diabetes Study (UKPDS). 13: Relative efficacy of randomly allocated diet, sulphonylurea, insulin or metformin in patients with newly diagnosed non-insulin dependent diabetes followed for three years. Brit Med J. 1995;**310**:83-88.

[31] Gogebakan O, Kohl A, Osterhoff M et al. Effects of weight loss and long-term weight maintenance with diets varying in protein and glycemic index on cardiovascular risk factors. Circulation. 2011;**124**:2829-2838.

[32] Goldberg RB. Cytokine and cytokine-like inflammation markers, endothelial dysfunction, and imbalanced coagulation in development of diabetes and its complications. J Clin Endocrinol Metab. 2009;**94**(9):3171-3182.

[33] Araya H, Contreras P, Vera G, Alviña M, Pak N. A comparison between and in vitro method to determine carbohydrate digestión rate response in Young men. Eur J Clin Nutr. 2002;**56**(8):735-739.

[34] Foster Powell K, Holt S, Brand Miller J. International table of glycemic index and glycemic load values. Am J Clin Nutr. 2002; **76**(1):5-56.

[35] Venn BJ, Mann JI. Cereal grains, legumes and diabetes. Eur J Clin Nutr. 2004;**58**:1443-1461.

[36] Pincheira D, Morgado R, Alviña M, Vega C. Quality of carbohydrates in the diet and its effect on metabolic control in subjects with type 2 diabetes mellitus. Arch Latinoam Nutr. 2014;**64**(4):241-247.

[37] FAO/WHO. Carbohydrate in Human Nutrition: Report of a Joint FAO/WHO Expert Consultation. Paper No. 66 ed. Rome, Italy: FAO. Food and Nutrition; 1998.

[38] Jury G, Urteaga C, Taibo M. Exchange portions and chemical composition of chilean foods pyramid. 1st ed. Santiago, Chile: Institute of Nutrition and Food Thecnologies, Center of Human Nutrition, Faculty of Medicine, Universidad de Chile; 1997. 129p.

[39] Parada JA, Rozowski J. Relationship between glycemic response of starch and its microstructure state. Rev Chil Nutr. 2008;**35**(2):84-92.

[40] Sjöström L, Narbro K, Sjöström CD et al. Effects of bariatric surgery on mortality in Swedish obese subjects. N Engl J Med 2007;**357**:741-752.

[41] Vega C. Effect of carbohydrates and proteins quality on weight loss and metabolic parameters n subjects undergoing bariatric surgery [thesis]. Santiago, Chile: Universidad de Chile; 2013. 68p.

[42] American Diabetes Association. Nutrition therapy recommendations for the management of adults with diabetes. Diabetes Care. 2014;**37**(1):S120-S143.

[43] Varela N, Vega C, Valenzuela K. Relationship of consumption of high glycemic index diet and levels of A1c in subjects with type 2 diabetes under metformin or nutrition therapy. Arch Latinoam Nutr. 2012;**62**(1):23-29.

[44] Sambra V, Tapia C, Vega C. Effect of fragmentation and quality of carbohydrates on metabolic control parameters in subjects with type 2 diabetes under basal insulin therapy. Nutr Hosp. 2015;**31**(4):1566-1573.

[45] Yousseif A, Emmanuel J, Karra E et al. Differential effects of laparoscopic sleeve gastrectomy and laparoscopic gastric bypass on appetite, circulating acyl-ghrelin, peptide YY3-36 and active GLP-1 levels in non-diabetic humans. Obes Surg. 2014;**24**(2):241-252.

[46] Amigo H, Bustos P, Pizarro M. National Survey of Food intake: final report. [Internet]. 1st ed. Santiago, Chile: february 02, 2016. [updated march 02, 2016; cited july 12, 2016]. Available in: http://web.minsal.cl/encadescarga/]

[47] Fuentes G, Deel Valle M, Vega C. Comparative analysis of the glycemic response and glycemic index of instant mashed potatoes in subjects undergoing laparoscopic sleeve gastrectomy and control subjects. Nutr Hosp. 2014;**30**(6):1263-1269.

[48] Matsuzawa Y, Funahashi T, Nakamura T. The concept of metabolic syndrome: contribution of visceral fat accumulation and its molecular mechanisms. J Atheroscler Thromb. 2011;**18**:629-639.

[49] Del Valle M, Teran C. Comparative studies of the glycemic response and glycemic index of two high carbohydrates foods, in subjects with insulin resistance [thesis]. Valparaiso, Chile: Universidad de Valparaíso; 2013. 90p.

[50] Lobos D, Vicuna I. Effect of a sucralose preload and low glycemic index breakfast intake in metabolic post prandial parameters in subjects with type 2 diabetes under intensive insulin therapy [thesis]. Valparaíso, Chile: Universidad de Valparaíso; 2015. 90p.

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