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Glycemic and insulinemic responses to hot vs cooled potato in males with varied insulin sensitivity

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Abstract

The objective of this study was to investigate whether the temperature at which the cooked food is served affects its glycemic and insulinemic indices in subjects with varied insulin sensitivity. Two potato meals containing 50 g of carbohydrates were fed to 9 subjects with varied insulin sensitivity, at mean temperatures of 83.6 ± 2.0°C for hot potato (HP) and 26.0 ± 0.6°C for cooled potato (CP). Cooled potato resulted in a significantly lower postprandial blood glucose and area under the glucose curve (glucose AUC) as compared to HP ($P < .05$). Postprandial triglyceride values significantly decreased from fasting levels after the CP whereas an increase was observed after the HP ($P < .05$). Glycemic index of CP was significantly lower than HP ($P < .05$). After consumption of HP, greater incremental changes in glucose and insulin were observed in hyperinsulinemic as compared to normoinsulinemic subjects. These results emphasize the importance of starch temperature at consumption as a factor that influences the glycemic index and may allow patients with hyperinsulinemia and diabetes to have a wider selection of starchy foods, if consumed at the appropriate temperature.

Keywords: Diabetes mellitus; Food temperature; Glycemic index; Insulinemic index; Insulin-glucose ratio; Hyperinsulinemics

1. Introduction

Jenkins et al [1] was the first to introduce the concept of the glycemic index (GI) as a tool to classify carbohydrate foods on the basis of their ability to raise blood glucose levels, by

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comparing the postprandial rise in serum glucose to a standard of pure glucose or white bread (WB). Glycemic index is a method of ranking carbohydrates according to their postprandial blood glucose responses and has been recently strongly recommended in preventing and managing diabetes mellitus [2], obesity, and cardiovascular disease [3]. Low GI foods have also been recommended for use in insulin-resistant subjects as they not only improve the metabolic consequences of insulin resistance (IR) but also tend to reduce IR per se [4]. Of particular importance are recent studies [5] that showed that a strong correlation exists between high postprandial glucose levels and development of vascular complication in diabetes, and that modulation of postprandial glycemia plays an important role in overall glycemic control. The development of insulinemic indices (IIs) to foods has also been reported as necessary to supplement GI tables used in the dietary management of diabetes mellitus [6].

There are many factors affecting the GI of a food: the macronutrient composition, the processing and cooking methods, the physical characteristics of the carbohydrate, and the presence of other food components [7]. Many common foods contain detectable amounts of resistant starch (RS), which is the sum of starch and starch degradation products not absorbed in the small intestine of healthy individuals [8], which may vary depending on the degree of processing or cooking [9] before consumption, and which can affect the GI of the food. Previous studies centered on producing new industrially processed foods in such a way as to decrease GI; hence, the effects of processing conditions on lowering GI have already been reported [10]. However, investigating the changes in GI of the same food, under different conditions of consumption, received less attention.

When starch is subjected to heat during cooking, the starch molecules undergo structural changes. The crystallinity of the granule is disrupted, causing starch to become more digestible [11], whereas cooling may lead to resistance to enzyme hydrolysis [12]. Potatoes and other moist-heated starchy foods are incompletely digested when cold because of the retrogradation of the dispersed starch molecules. Englyst and Cummings [13] showed the digestibility of starch in cooked potato to fall around 10% after cooling. However, the effects of this on the glycemic response have not been studied. Thus, the aim of the present research is to investigate whether cooling potatoes, after they have been subjected to heat processing, will affect the glycemic and insulinemic responses of subjects with varied insulin sensitivity.

2. Methods and materials

2.1. Experimental design

Nine male volunteers with varied insulin sensitivity were recruited for the study through advertisements. Insulin-glucose ratio (IGR) was measured to assess insulin sensitivity. Insulin-glucose ratio was calculated by dividing the insulin values (mU/L) by the glucose values (mmol/L) of each subject at fasting levels. Subjects were divided into 2 groups according to the IGR; a cutoff value of IGR ≥ 4 was used to indicate IR [14]; 4 were labeled as IR and 5 as normoinsulinemic (NI) subjects. Selection criteria were male subjects aged 17 to 27 years, with stable body weights over the previous 2 months. Subjects were also screened

for fasting blood glucose and insulin and glycated hemoglobin (HbA_1c). Subjects taking medications or classified as hyperglycemic or diabetic according to the criteria of hyperglycemia and diabetes were excluded from the study [15]. The study was approved by the institution research board and all volunteers gave written informed consent.

Before each test, subjects fasted for 12 hours overnight, during which free consumption of water was allowed. On 3 test mornings, subjects were randomly given 1 of 3 meals: 2 test meals, hot potato (HP) and cooled potato (CP), and 1 standard meal, WB. The time interval between test mornings ranged from 1 to 5 weeks. To avoid the "second-meal effect" [16], subjects were instructed not to eat legumes in the meal preceding the fast. In addition, a meal standardized in its composition was recommended for all subjects to be taken in the evening before each of the 3 test mornings. They were also instructed not to do any strenuous physical exercise throughout the study period. On the day of the experiment, subjects were allowed to bring in with them any form of entertainment not requiring physical effort.

For calculation of GI, WB of a locally consumed brand (Pain D'or) was chosen as the reference food because it follows a standardized recipe [17]. It was purchased 1 day before the experiment from a bakery shop and kept at room temperature overnight. The average weight of bread consumed was equivalent to 50 g carbohydrates. Fresh potatoes were purchased from a retail supermarket in Beirut. They were peeled and freshly boiled each morning before the test, until soft. The temperature of potato meals was recorded when consumed hot and after cooling. The cooling parameter was constant and the potatoes were cooled to a predetermined temperature of 26°C selected to fit the participants' preference. Mean temperature (°C) for consumption of CP and HP is indicated in Table 1. Each potato meal was also calculated to contain 50 g carbohydrates, that is, starch and sugars according to their tabulated composition per 100 g edible portions [18]. Water was the adjunct taken with every meal in quantities to make up the overall meal volume to approximately 350 mL. Table 1 lists information about the weight of meals ingested, the added water, their nutrient composition, and the temperature at which they were consumed.

2.2. Blood analysis

On the morning of each test, 5-mL fasting blood samples were collected to determine baseline glucose and insulin levels. Each subject then consumed the meal within 15 minutes. After completion of each test meal, 5-mL venous blood samples were collected by a registered nurse at time intervals of 15, 30, 45, 60, 90, 120, and 180 minutes.

Serum glucose was assayed using the UV test based on the hexokinase/G6P-DH method. This kit was provided by Boehringer-Mannheim, Germany. Insulin was measured by using an antiinsulin antibody radioimmunoassay provided as a kit by CIS Bio-international, France.

Total cholesterol and triglycerides (TGs) were analyzed by an enzymatic colorimetric test provided as a kit by Boehringer-Mannheim (Cat No. 816370). High-density lipoprotein cholesterol (HDL-C) was determined colorimetrically by using the HDL separation tab (Union Carbide Corp, Pleasantville, NY, for in vitro diagnosis use). Low-density lipoprotein cholesterol (LDL-C) was calculated according to the Friedwald equation [19].

Table 1
Nutrient composition of the 3 meals (mean ± SEM, n = 9)

Meal type	Wt of meal (g)	Added water (mL)	Food's water content (mL)	Energy (kcal)	Carbohydrate (g)	Protein (g)	Fat (g)	Fiber (g)	Temp (°C)
Bread (WB)	100.8 ± 0.2	311.4 ± 0.4	37.3 ± 0.09	262.1 ± 0.6	48.4 ± 0.1	8.1 ± 0.02	4.03 ± 0.09	1.9 ± 0.01	–
HP	251.8 ± 0.4	157.1 ± 2.8	193.9 ± 0.3	214.5 ± 0.4	50.4 ± 0.09	3.7 ± 0.07	Tr	3.8 ± 0.01	83.6 ± 2.0
CP	252.1 ± 0.6	161.2 ± 0.3	194.1 ± 0.4	214.8 ± 0.5	50.4 ± 0.1	3.7 ± 0.01	Tr	3.8 ± 0.09	26.0 ± 0.6

Tr indicates trace amounts.

Table 2
Clinical characteristics and blood parameters of the 9 subjects

Characteristics	Mean ± SEM, n = 9
Age (y)	20.9 ± 0.7
Body mass index (kg/m^2)	30.6 ± 3.4
Fasting plasma glucose (mg/dL)	91.7 ± 2.8
Fasting plasma insulin (mU/L)	28.0 ± 4.9
IGR	5.4 ± 0.7
Total cholesterol (mg/dL)[a]	173.5 ± 15.4
LDL-C (mg/dL)[a]	113.5 ± 9.3
HDL-C (mg/dL)[a]	35.9 ± 2.0
TGs (mg/dL)	125.8 ± 31.9
HbA_1c (%)	5.0 ± 0.2

[a] Values represent means of 3 different values taken on 3 different test mornings.

The GI for each food was calculated according to the formula of Jenkins et al [1]. The II of foods was calculated according to the method of Chew et al [20].

2.3. Calculations and statistical analysis

The areas under the curves (AUCs) were calculated geometrically as the incremental areas above the fasting glucose, insulin, and TGs values using PRISM (version 3.0, Graph Pad Software, San Diego).

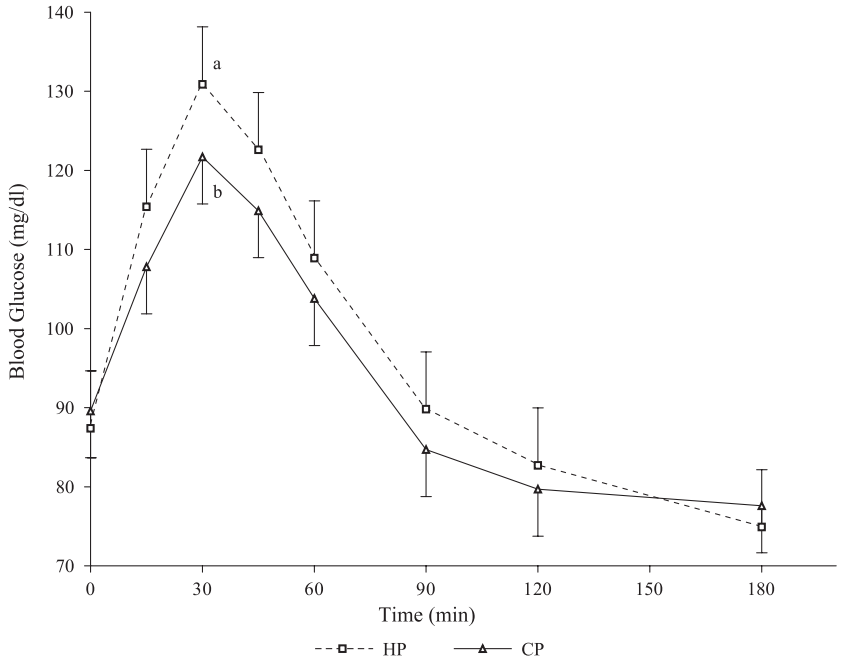

Fig. 1. Mean postprandial glucose responses after ingestion of HP and CP. Means with different superscripts (a and b) are significantly different at $P < .05$.

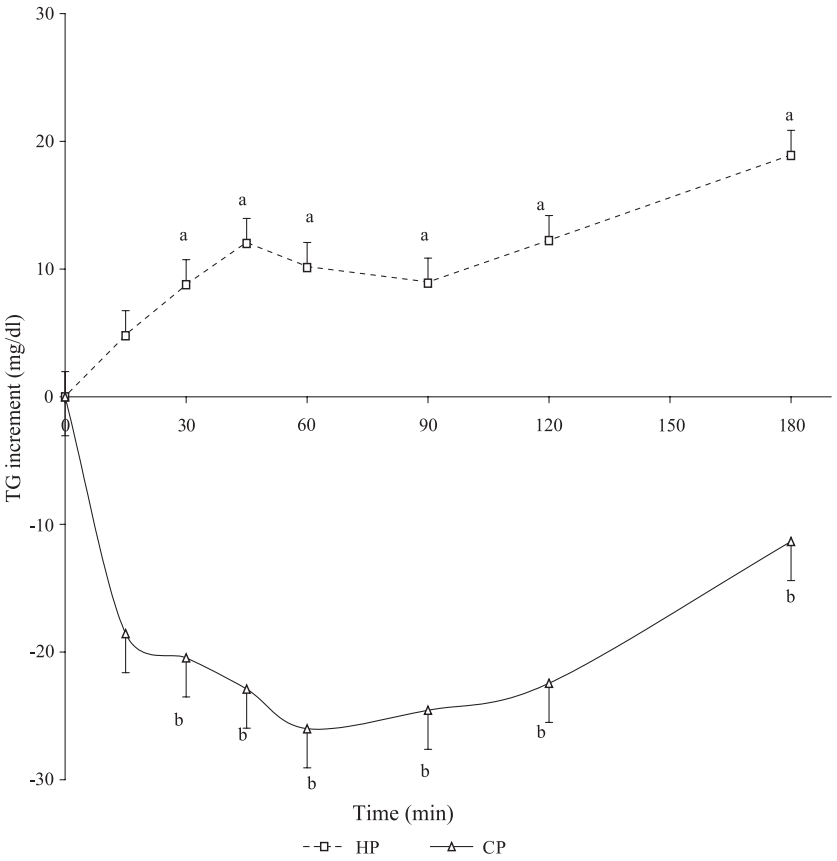

Fig. 2. Mean postprandial TG responses after ingestion of HP and CP. Values with different superscripts (a and b) are significantly different at $P < .05$.

Descriptive statistics (mean ± SEM) were estimated. Nonparametric Wilcoxon paired test was used to test for significant differences between HP and CP for glucose, TG, glycemic, and insulinemic indices, areas under the curves. For this analysis, the Statistical Packages for the Social Sciences (SPSS version 11.0, Chicago, Ill) was used. When the sample was stratified by IGR, the PRISM Graph Pad software was used to compare the IR (n = 4) to NI (n = 5) curves as far as their responses to HP and CP (unmatched analysis), and the effect of

Table 3
Mean glucose, insulin, and TG area under curve after ingestion of the HP and CP (mean ± SEM)

	HP (n = 9)	CP (n = 9)
Glucose AUC ([mg min]/dL)	2343.9 ± 347.6[a]	1484.1 ± 235.1[b]
Insulin AUC ([mU min]/L)	6416.3 ± 2261.4	4478.3 ± 821.5
TG AUC ([mg min]/dL)	1499.7 ± 627.0[a]	81.7 ± 66.3[b]

Means in the same row with different superscripts (a and b) are significantly different at $P < .05$.

Table 4
Mean glycemic and insulinemic indices of HP and CP (mean ± SEM)

Meal	HP (n = 9)	CP (n = 9)
GI (%)	122.4 ± 18.2[a]	77.5 ± 12.3[b]
II (%)	117.0 ± 41.3	81.7 ± 15.0

Values in the same row with different superscripts (a and b) are significantly different at $P < .05$.

HP vs CP on postprandial glucose and insulin response curves in IR (n = 4) subjects (matched analysis) [21]. Results were considered significant at $P < .05$.

3. Results

The baseline characteristics and blood parameters of the 9 subjects who participated in the study are shown in Table 2.

Fig. 1 demonstrates glucose responses after consumption of potato meals and bread. Mean postprandial glucose values at 30 minutes were significantly greater after HP consumption than CP ($P < .05$). There were no significant differences in insulin between HP and CP at any time.

Fig. 2 demonstrates a net incremental increase in TGs that occurred after consuming HP, whereas a net decrease was observed after CP consumption. Significant postprandial

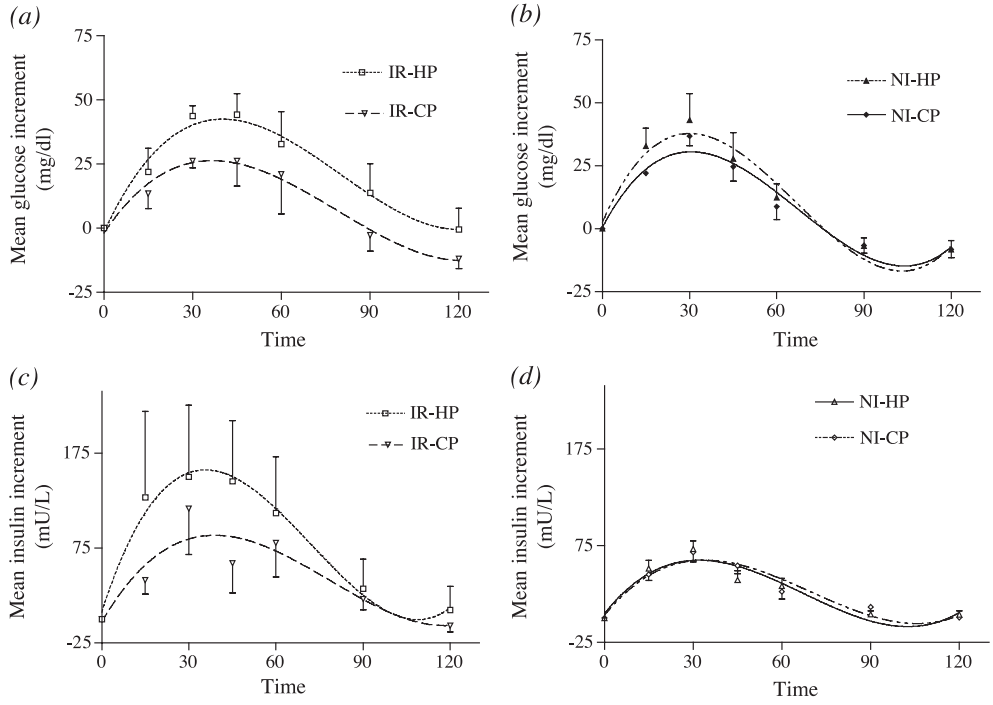

Fig. 3. Mean incremental changes with respect to time in glucose and insulin after consumption of HP and CP for both the hyper (IR) and NI subjects.

differences in TGs between potato meals were seen at all times: 30, 45, 60, 90, and 120 minutes.

The areas under the glucose, insulin, and TG curves after consuming HP and CP were also calculated (Table 3). Mean glucose and TG AUC after the ingestion of HP was found to be significantly greater than CP. Although the mean insulin AUC for HP appeared greater than that for CP, the levels did not reach statistical significance.

The GI value obtained after consuming HP was significantly greater than that of CP at $P < .01$. As for II, there was no statistically significant difference between HP and CP (Table 4).

When subjects were divided into 2 groups according to IGR, a cutoff value of IGR ≥ 4 was used to indicate IR [19]: 4 were labeled as IR and 5 as NI subjects. Fig. 3 demonstrates the incremental changes with respect to time in glucose and insulin for both IR and NI subjects after consumption of HP and CP.

Within IR subjects (Fig. 3a, c), a greater postprandial glucose and insulin incremental responses were observed after HP as compared to CP consumption. Global fitting of the data showed that the differences were statistically significant.

Within the NI group (Fig. 3b, d), no differences were observed after HP as compared to CP consumption.

Comparing IR to NI subjects (data not shown), postprandial glucose and insulin incremental peaks were higher in IR as compared to NI group after HP consumption; however, the difference did not reach statistical significance. There was no difference in postprandial incremental glucose and insulin levels between the 2 groups after CP ingestion.

4. Discussion

This study provides new evidence that the temperature at which potato (starch-containing food) is consumed can affect the postprandial glycemic and lipid profile. We report in this study on the effect of cooling potatoes on the GI and the lipid responses, and whether hyperinsulinemia can affect the postprandial glycemia, glycemic and insulinemic indices of HP vs CP. This is particularly significant because modulation of postprandial plasma glucose levels has been shown to play an important role in glycemic control especially in subjects with diabetes. Meal-associated hyperglycemia has been shown to be correlated with development of macro- and microvascular complications. Evidence of a strong correlation between high postprandial glycemic levels and development of vascular complications underscores the significance of treating mealtime glycemia with drugs [5].

After ingestion of CP, a lower 30-minute glucose peak and lower glucose AUC were observed as compared to HP. This may be due to molecular changes that occur in potatoes in response to cooling. Previous studies have reported changes in potato structure in response to heating and cooling. Application of both heat and moisture during cooking caused the starch to be gelatinized and more susceptible to enzyme hydrolysis [22], whereas upon cooling, the amylose chains retrograde and become more resistant to amylase [13]. This resistance was associated with the production of more RS of the subtype RS3 (retrograded amylose), and studies have reported its recovery in ileostomy effluent to be twice that obtained after freshly cooked HPs [13,23]. In our study, whether further cooling of potatoes would have an additional effect on RS and consequently the GI needs to be investigated.

Recent studies have addressed this issue of starch digestibility and showed that increased cooking is associated with increased glycemic responses to potato [9,24]. Vaaler et al [25] also showed that cooking was responsible for the rapid rise in blood glucose after potato ingestion. This study revealed that although both CP and HP were processed in the same manner, it was the effect of temperature of the potato before consumption that led to the resulting decrease in peak glucose value and glucose AUC.

In accordance with this concept, our GI measurements revealed a significant difference in the GI of potato, depending on its temperature at consumption. Cooling the potato after cooking produced a significantly lower GI value than HP, which could be due to the higher RS content of potatoes due to cooling. Bjorck et al [4] combined data from various studies showing the inverse correlation between GI and RS content of foods. Food factors that reduce the rate of starch digestion such as retrogradation of the amylose component seem to render the starch fraction resistant to amylases, which inversely affects the GI of the food. Hence, it is evident that the amount of carbohydrate content in a food is not the only factor determining the GI of the food and that temperature should be regarded as an influencing factor.

It is known that potatoes are among the most commonly consumed high-GI foods [4] and that they are also the one food that has shown variable blood glucose responses. Glycemic index values for potatoes as listed in the literature have ranged from 67 in boiled potatoes to 137 in baked potatoes [17,26-28]. This made it difficult to assign a GI for potatoes and compare results. The GI values have varied across studies depending on the type of potato tested, the degree of maturity, and the cooking procedure applied. Investigators differed in their conclusions regarding the impact of cooking methods on the GI of potatoes. In some studies, there were no significant differences in GI values whether potatoes were boiled, oven-baked, microwaved, or mashed [27]. In others, baked potatoes produced a significantly lower incremental glycemic response compared with boiled potatoes [29]. In addition, the method of consuming potatoes with reported GIs has not been well defined in many studies, and notably, the temperature at which potatoes were consumed has not been documented. This may partly explain the large variability observed in studies for the GI values of potatoes. In this study, the type of potato and the eating method were controlled, and temperature was the only variable.

Postprandial insulin results indicated a lower insulin peak after consumption of CP as compared to HP, but the difference did not reach statistical significance. This may be due to the high individual variability in insulin levels, because 4 of the subjects were insulin resistant according to the IGR classification [14]. Similar results were also found in the AUC calculations for insulin. Although the insulin AUC obtained after CP consumption appeared smaller than that after HP consumption, the difference was not statistically significant. Our II findings also showed no significant difference in the II value of HP vs CP. The sample of subjects ($n = 9$) who participated in this study was not normally distributed in respect to their insulin levels, and this may explain the lack of significance. These findings however are partly in disagreement with some studies that have suggested insulin responses to parallel glucose responses [30-32]. However, Indar-Brown et al [33] and Wolever et al [34] did not show a correlation between glucose response and insulin response areas. Also, Wolever et al [16] suggested that an increased insulin secretion did not account for the reduced glycemic responses produced by low GI foods. Further studies are needed to elucidate this issue.

Triglyceride analysis revealed a significant difference postprandially between the HP and CP. There was a net incremental increase in TG concentration after consuming the HP, whereas a net decrease was observed after the CP. In addition, the TG AUC after consuming HP was significantly greater than the area obtained after CP. A possible mechanism is that a higher insulin response was evoked after the HP (high-GI), which stimulated the hepatic production of very low-density lipoprotein [35]. This finding is in agreement with the literature that showed that high-GI meals elicit increased postprandial TG concentrations [36-38], whereas low-GI foods have been shown to decrease TGs [39-41], thus, eliciting their beneficial effects on lipemia, a risk factor of cardiovascular disease and metabolic syndrome. Other longer term studies have also noted the benefits of low-GI diets in lowering TGs [41].

It thus seems logical to propose that low-GI diets, which reduce insulin secretion, may also be of use in the treatment of hyperlipidemia. This is the first recorded observation that the temperature of the carbohydrate meal at consumption affects the TG response.

When the subjects were grouped according to IGR (IGR $\pm$ SEM, 8.6 $\pm$ 3.1 and 3.4 $\pm$ 0.2 for IR and NI, respectively), the IR demonstrated a tendency toward higher glucose and insulin incremental changes after the ingestion of HPs compared to CP; however, this effect was not observed in the NI subjects. We speculate that in NI subjects, the first phase of insulin release is intact and may prevent an exaggerated glycemic response to high carbohydrates. In insulin-resistant subjects, the first phase of insulin release is lost causing plasma glucose levels to rise sharply after a meal, precipitating an increased stimulation of second-phase insulin release. Hence, in this study, a high level of available carbohydrates and glycemic response did not result in an exaggerated insulin response characteristic of insulin-resistant subjects.

The lower postprandial glycemia, insulinemia, and TG levels associated in this study with the consumption of low GI CP provide, in accordance with the literature, further evidence on the benefits of consuming a low GI diet.

It seems logical to deduce that subjects with hyperinsulinemia or diabetes, who are insulin resistant, may benefit from substituting cooled starchy meals (lower GI) in place of hot (higher GI) meals. Yet, this observation needs to be evaluated further in studies involving a higher number of insulin-resistant patients.

Further research is also needed to evaluate the GI of starch-containing foods and whether it changes when consumed at cooled temperatures. In addition, the effect of further cooling below the ambient temperature used in this study could be tested to observe if a further decrease in the glycemic response can be achieved.

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