

# Dynamics of blood glucose concentration after a food determines subsequent energy consumption

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**Summary.** Obesity and its related comorbidities such as type 2 diabetes and cardiovascular disorders are global public health challenges. It is imperative to understand patterns of the dietary composition that regulate blood glucose concentration, satiety cues, and energy consumption. *Objectives:* This study elucidated the dynamics of blood glucose concentration after a test food as a crucial determinant of appetite and energy intake at a subsequent meal. *Methods:* Both low and high glycemic index (GI) foods in liquid and solid forms or fixed available carbohydrate and in equicaloric amounts were tested in healthy female volunteers (n=14/experiment) on blood-glucose and percent energy compensation (%EC). White wheat bread (solid, high-GI) was compared with chickpeas (solid, low-GI) at 50 g available-carbohydrate in Experiment 1 and Coca-Cola (liquid, high-GI) with skim milk (liquid, low-GI) and chickpeas (solid, low-GI) at equicaloric amounts in Experiment 2. Blood glucose and appetite were measured at baseline and over time up to two hours in Experiment 1 and one hour in Experiment 2. Caloric intake was estimated from a pizza-meal at the end of the studies and %EC calculated. *Results:* Both high GI foods had the largest glucose peaks; chickpeas had an intermediate and milk the smallest peak. Blood glucose concentration before meal time was associated with energy intake. The %EC was: chickpeas (70%) > bread (7%) in Experiment 1; and chickpeas (80%) > milk (40%) > Coca-Cola (32%) in Experiment 2. *Conclusions:* Intake of foods with low GI value would prove helpful in the prevention and controlling of obesity, hyperglycemia or hyperinsulinemia.

**Key words:** Coca-Cola, milk, chickpeas, bread, energy compensation

## Introduction

Metabolic regulation of body weight relies on the hypothalamic and other parts of the brain through programmed energy balance, known as 'Body weight set point' theory (1). However, body weight regulation is more complex, implicated not solely by the genetically programmed set point, but the obesogenic environment and the hedonic temptation of the abundance and variety of food play an integral role.

Excessive body weight is considered among the major risk factors for non-communicable dietary chronic diseases including diabetes. In the past 25 years, the prevalence of Type-2 diabetes has increased 120% worldwide and is projected to increase further

(2). Health professionals are attempting hard to apprehend the mechanisms of causative dietary factors or patterns governing satiety cues and eating behaviour. It is well known that high glycemic index (GI) foods are known to encourage excess calories intake through blood glucose spike accompanying hyperinsulinemia that causes reactive hypoglycaemia, hunger and cellular fat gain (3). Nevertheless, it is argued that neither GI or insulin secretion is crucial, rather it is 'body weight set point' that regulates body fatness through the 'leptin-hypothalamus feedback loop' (4). The phenomenon to understand excessive energy consumption is further compounded by the conflicting results reported by various investigators. For example, some researchers suggest that the liquid form of foods

is less compensatory for the energy consumption at the next meal than the solid form of foods (5-7). Whereas, others reported vice versa (8-11). The reason for these controversial results may lie in the fact that most studies did not measure the blood glucose responses which could influence the feeling of hunger and consequently food consumption.

Glucose being a preferred energy source for the brain, fluctuations in the blood glucose concentration is monitored by the gut-brain axis via glucosensors in the hypothalamus (12). The blood level of glucose and its sustainability overtime is an important outcome of the type of food consumed by individuals. The post-prandial blood glucose concentration, a consequence of several factors such as the ingested food form (liquid or solid) (5-11), GI (low or high) (3, 13), or nutrient composition (14-15), however, remains controversial on appetite and energy consumption. This study was designed to evaluate some of the common dietary determinants of blood glucose concentration influencing satiety and food intake in two short-term experiments. Experiment 1 compared up to 120 min the effect of the solid form of food with low and high GI, fed at equal amounts of available carbohydrate while Experiment 2 examined up to 60 min the effect of a solid versus a liquid form of food with low and high GI, fed in equicaloric amounts. In both experiments, participants were healthy young female subjects, a population group highly susceptible to excessive weight gain with age (16).

## Material and Methods

### *Subjects*

Volunteers (n=14/both experiments) were selected from a convenient sample of healthy female students, with a body mass index (BMI in kg/m<sup>2</sup>) of 20-25 and ages 17-30 years. Subjects were recruited through flyers and by word of mouth from the College of Life Sciences at Kuwait University. According to the study exclusion criteria, volunteers with high fasting blood glucose, on medication, breakfast skippers or restrained eaters (scored  $\geq 11$  on the Eating Habits Questionnaire (17) were not recruited. Furthermore, no test session was scheduled during the menstrual cycle to avoid hor-

monal effects if any, on blood glucose or appetite (18). The study was conducted in the Human Nutrition laboratories of the Department of Food Science and Nutrition. A consent form was signed by all subjects. This study was implemented in accordance with the principles established by the Declaration of Helsinki. The study was approved by the Human Subjects' Review Committee, Ethics Review Office, Kuwait University. The sample size determination was based on the results from previously reported similar research studies that detected an 80% difference in the glycemic responses among the test treatments at an alpha level of  $p < 0.05$  using the statistical package of SPSS (19).

### *Test foods*

The test foods - white wheat bread (Kuwait Flour Mills), canned chickpeas (Giant Chickpeas with Chillies, Al-Daniah, Kuwait), Coca-Cola (Classic), skim milk (Almarai Milk Company, Saudi Arabia) and bottled water (Aquafina) - were purchased from a local market. Weighed amounts corresponding to 50 g available carbohydrate (total carbohydrate - dietary fiber) of the white bread and canned chickpeas were used in Experiment 1 (Table 1). White bread was served immediately after toasting for 30 seconds and chickpeas were microwaved for 90 seconds before serving. Five gram of butter was served with both foods for taste. The water control was served at refrigerator temperature. Additional water was served with the test foods to equalize their volume in the stomach and to facilitate swallowing.

In Experiment 2, test foods were served at the temperatures they are usually consumed; Coca-Cola, skim milk and water at refrigerator temperature, and canned chickpeas were served as in Experiment 1. All test foods had contained 200 Kcal. The composition of test foods for both experiments is given in Table 1.

### *Experimental procedures*

Both experiments had a cross-over study design. Each subject consumed the test foods for each experiment in a random order. The experimental procedure followed was similar to the other studies used in our lab. Blood glucose was measured in a finger prick sample by portable blood glucose monitoring system (One Touch Ultra, Life Scan Inc and Johnson & Johnson

**Table 1.** Nutrition composition of the test preloads of Experiments 1 and 2

Experiment 1: Test preloads fed at 50 g available carbohydrate				
	Water	White Bread	Canned Chickpeas	
Energy (kcal)	0	262	307	
Available Carbs (g)	0	50	50	
Total Fat (g)	0	2.5	2.6	
Protein (g)	0	10	21	
Dietary Fiber (g)	0	0.7	20.8	
Weight (g)	500	125	342	
Water Served (mL)	500	375	158	
Total Volume (mL)	500	500	500	
Energy Density (kcal/g)	0	0.524	0.614	
Experiment 2: Test preloads fed at equicaloric amounts				
	Water	Coca Cola	Skim Milk	Canned Chickpeas
Energy (kcal)	0	200	200	200
Available carbs (g)	0	53	27	35
Total Fat (g)	0	0	2.6	2.3
Protein (g)	0	0	17	10.8
Dietary Fiber (g)	0	0	0	8.2
Weight (g)	-	-	-	226
Volume (mL)	500	493	500	261
Water Served (mL)	-	7	-	239
Total Volume (mL)	500	500	500	500
Energy Density (Kcal/g)	0	0.4	0.4	0.4

Company, USA), and subjective appetite by a Visual Analogue Scale (VAS) questionnaire (20, 21) at baseline and at every 15 min for one hour after ingestion of the test food in both experiments and then at 30 min interval for the second hour of Experiment 1. At the end of each test, a pizza meal and a bottle of water were served. The subjects were asked to eat and drink until comfortably full.

Subjects came for the study sessions between 8.00 and 10.00 am after an overnight fast of 10-12 hours. Subjects were instructed to maintain a regular pattern of food intake and physical activity throughout the study.

#### Food intake

The pizzas prepared and served as described in our earlier studies at the end of each session (19, 20) were 5-inch round containing about 200 kcal, available in two varieties Briefly, the pizzas (Four Cheeses and Deep N Delicious Vegie Pizza; McCain Foods Ltd) were baked and cut into 4 pieces, served in consecutive

trays within 6-7 minutes until the subjects refused to eat more. Food intake was assessed by weighing the cooked pizza before and after serving (left-over). The caloric consumption was calculated from the nutrition information provided by the manufacturer on the pizza labels.

#### Data analysis

Incremental area under the blood glucose response curves (AUC), ignoring any area below fasting, was determined for each test food for each subject. For analysis of VAS appetite responses, an average appetite score was calculated for each time point using the formula:

$$\text{Average Appetite} = [\text{Question 1} + \text{Question 2} + (100 - \text{Question 3}) + \text{Question 4}] / 4.$$

Percent energy intake compensation (%EC) at the second meal for the test food calories was calculated by the following formula:

% EC = [(Kcal intake at pizza meal after the Test food - Kcal intake at pizza meal after the Water Control)/Kcal from the Test Food] \* 100.

Statistical analysis was conducted using SPSS (Statistical Package for Social Sciences). One-way repeated measures analysis of variance (ANOVA) was used to test for the effect of treatments on outcome variables, including changes from baseline in blood glucose concentrations and average appetite scores at each time point and incremental area under the curve (AUC) for these changes for the total test periods, and calorie intake and percent energy compensation at meals. Two-way repeated measures ANOVA was conducted on changes in appetite and blood glucose concentration scores at each test period to determine time and treatment effects and for a time by treatment interaction.

Tukey's posthoc tests were performed when treatment effects were statistically significant ( $p < 0.05$ ). All results presented are as mean  $\pm$  standard error of the mean (SEM). Correlation analyses were conducted using the Pearson correlation coefficient.

## Results

### Blood glucose

Blood glucose changes were affected by both treatment ( $p < 0.001$ ) and time ( $p < 0.001$ ) with a time by

treatment interaction ( $p < 0.001$ ). Peaks in blood glucose occurred at 30 min for all foods in both experiments followed by declines. In Experiment 1, white bread resulted in the most rapid increase in blood glucose, but then the levels declined by 120 min and fell below the baseline. Chickpeas reached a significantly lower rise at 30 min and maintained this rise above the baseline at 120 min, which was significantly different from white bread but not the water control. The calculated AUC for blood glucose was also significantly higher for white bread followed by chickpeas and water (Table 2).

In Experiment 2, blood glucose increases again peaked at 30 min with Coca-Cola showing significantly maximum rise than the milk, chickpeas or water and declined thereafter, with all treatments remained above the baseline at 60 min. The blood glucose AUC was significantly higher after Coca-Cola followed by chickpeas then milk and water. (Table 2).

### Average appetite

The appetite scores at the baseline were not different for all the treatments in both Experiments. The average change in appetite was affected by treatment ( $p < 0.05$ ) and time ( $p < 0.05$ ), however, no treatment by time interaction was observed ( $p > 0.05$ ). The lowest values for appetite score change (e.g. least hunger), as expected, were observed between 30-60 min after the chickpeas preloads in both Experiments compared

**Table 2.** Change in blood glucose concentration from baseline with preload of 50 g of available carbohydrate (Experiment 1) or equalcaloric amounts of low versus high GI foods (Experiment 2) as solid or liquid form

Time (min)	Experiment 1			Experiment 2			
	Water	Bread mmol/L	Chickpeas	Water	Coca Cola mmol/L	Milk	Chickpeas
15	0.04 $\pm$ 0.12 <sup>a</sup>	1.05 $\pm$ 0.12 <sup>b</sup>	0.78 $\pm$ 0.18 <sup>b</sup>	0.11 $\pm$ 0.01 <sup>a</sup>	2.00 $\pm$ 0.39 <sup>d</sup>	0.40 $\pm$ 0.41 <sup>b</sup>	1.00 $\pm$ 0.23 <sup>c</sup>
30	0.06 $\pm$ 0.10 <sup>a</sup>	1.9 $\pm$ 0.12 <sup>b</sup>	1.39 $\pm$ 0.22 <sup>c</sup>	0.10 $\pm$ 0.01 <sup>a</sup>	3.00 $\pm$ 0.24 <sup>d</sup>	0.70 $\pm$ 0.21 <sup>b</sup>	1.5 $\pm$ 0.34 <sup>c</sup>
45	0.09 $\pm$ 0.10 <sup>a</sup>	1.50 $\pm$ 0.12 <sup>b</sup>	1.22 $\pm$ 0.22 <sup>b</sup>	0.06 $\pm$ 0.02 <sup>a</sup>	2.30 $\pm$ 0.42 <sup>c</sup>	0.37 $\pm$ 0.55 <sup>a</sup>	1.48 $\pm$ 0.21 <sup>b</sup>
60	0.04 $\pm$ 0.11 <sup>a</sup>	0.94 $\pm$ 0.11 <sup>b</sup>	0.44 $\pm$ 0.15 <sup>a</sup>	0.07 $\pm$ 0.01 <sup>a</sup>	1.40 $\pm$ 0.43 <sup>b</sup>	0.30 $\pm$ 0.38 <sup>a</sup>	0.57 $\pm$ 0.27 <sup>a</sup>
90	-0.08 $\pm$ 0.11 <sup>a</sup>	0.08 $\pm$ 0.15 <sup>a</sup>	0.16 $\pm$ 0.15 <sup>a</sup>				
120	-0.19 $\pm$ 0.13 <sup>ab</sup>	-0.38 $\pm$ 0.09 <sup>b</sup>	0.07 $\pm$ 0.08 <sup>a</sup>				
AUC <sup>#</sup>	32.56 $\pm$ 10.03 <sup>a</sup>	121.68 $\pm$ 18.23 <sup>b</sup>	86.66 $\pm$ 17.30 <sup>ab</sup>	12.00 $\pm$ 2.00 <sup>a</sup>	158.11 $\pm$ 14.05 <sup>c</sup>	35.40 $\pm$ 34 <sup>a</sup>	105.43 $\pm$ 21.00 <sup>b</sup>

\* Data presented is as Mean  $\pm$  SEM,  $n=14$ ; Different superscript letters denote a significant difference at  $p < 0.05$  in the same row (Tukey's posthoc test).

<sup>#</sup>AUC = Area under the curve (mmol min/L)

**Table 3.** Change in appetite from baseline with preload of 50 g of available carbohydrate (Experiment 1) or equicaloric amounts of low versus high GI foods (Experiment 2) as solid or liquid

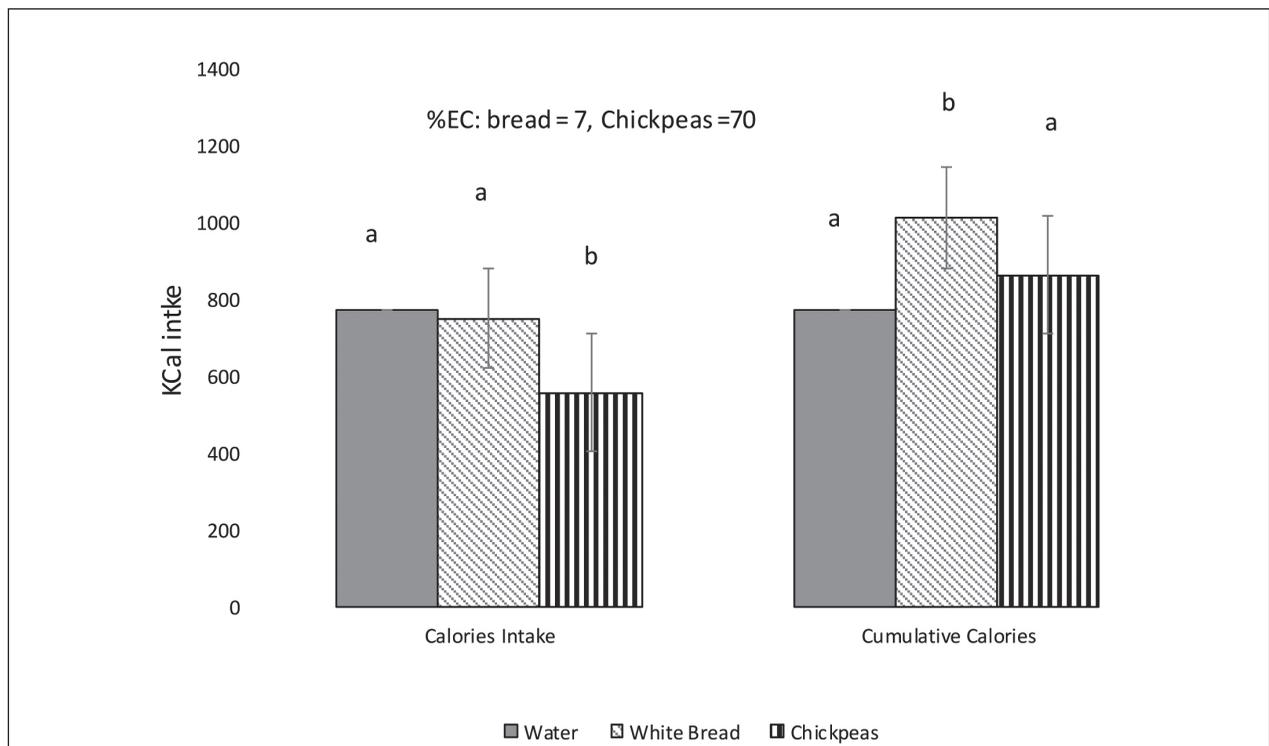
Time (min)	Experiment 1			Experiment 2			
	Water	Bread mm	Chickpeas	Water	Coca Cola	Milk mm	Chickpeas
15	-1.82±0.76 <sup>a*</sup>	-3.52±1.09 <sup>a</sup>	-4.97±0.81 <sup>a</sup>	-2.50±1.63 <sup>a</sup>	-5.00±1.04 <sup>a</sup>	-5.50±0.80 <sup>a</sup>	-6.50±0.87 <sup>a</sup>
30	-0.46±0.87 <sup>a</sup>	-1.89±0.94 <sup>ab</sup>	-4.71±0.86 <sup>b</sup>	-0.05±0.89 <sup>a</sup>	-2.00±1.95 <sup>ab</sup>	-3.00±1.90 <sup>b</sup>	-4.50±0.90 <sup>b</sup>
45	0.41±0.55 <sup>a</sup>	-0.86±0.82 <sup>a</sup>	-3.51±0.88 <sup>b</sup>	0.50±1.50 <sup>a</sup>	-0.50±0.82 <sup>a</sup>	-2.00±0.88 <sup>ab</sup>	-3.50±0.80 <sup>b</sup>
60	0.49±0.43 <sup>a</sup>	-0.32±0.89 <sup>a</sup>	-1.80±0.74 <sup>a</sup>	2.00±1.30 <sup>a</sup>	0.50±0.92 <sup>a</sup>	-0.50±0.36 <sup>ab</sup>	-2.60±0.36 <sup>b</sup>
90	1.11±0.64 <sup>a</sup>	-0.02±1.82 <sup>a</sup>	-0.77±0.78 <sup>a</sup>				
120	1.32±0.74 <sup>a</sup>	0.87±0.61 <sup>a</sup>	0.23±0.71 <sup>a</sup>				

\* Data presented is as Mean ± SEM, n=14; Different superscript letters denote a significant difference at  $p < 0.05$  in the same row (Tukey's posthoc test)

to water, at 45 min compared to bread, and at 45–60 min compared to Coco-Cola but were not different from milk preloads (Table 3). AUCs for appetite score changes did not differ by test treatment in either of the Experiments (data not included).

#### Food intake

In Experiment 1, kcal intake at 120 min was significantly lower ( $p < 0.0001$ ) after chickpeas than white bread or water (Figure 1), and cumulative energy intake was significantly higher for white bread,



**Figure 1.** Experiment 1 – Effect of a similar available carbohydrate content on energy intake, cumulative energy intake and percent energy compensation (%EC)<sup>s</sup> in healthy females

Data presented is as Mean ± SEM, n=14; Different superscript letters denote a significant difference at  $p < 0.05$  in the same group of bars (Tukey's posthoc test)

<sup>s</sup>%EC = [(Kcal intake at meal after the Control (water) preload – Kcal intake at meal after the Test preload (white bread or chickpeas) / Kcal from the Test preload] \* 100.

making the calculated %EC much higher for chickpeas. In Experiment 2, energy intake at 60 min was same for the three energy nutrients, whereas water was similar to milk and Coca-Cola, yet it was higher than the chickpeas. The cumulative energy intake was similar between milk and Coca-Cola, and both water and chickpeas were similar to each other yet significantly lower than Coca-Cola and milk ( $p < 0.001$ ) (Figure 2), making %EC the highest for chickpeas, and approximately double that of Coca-Cola and milk.

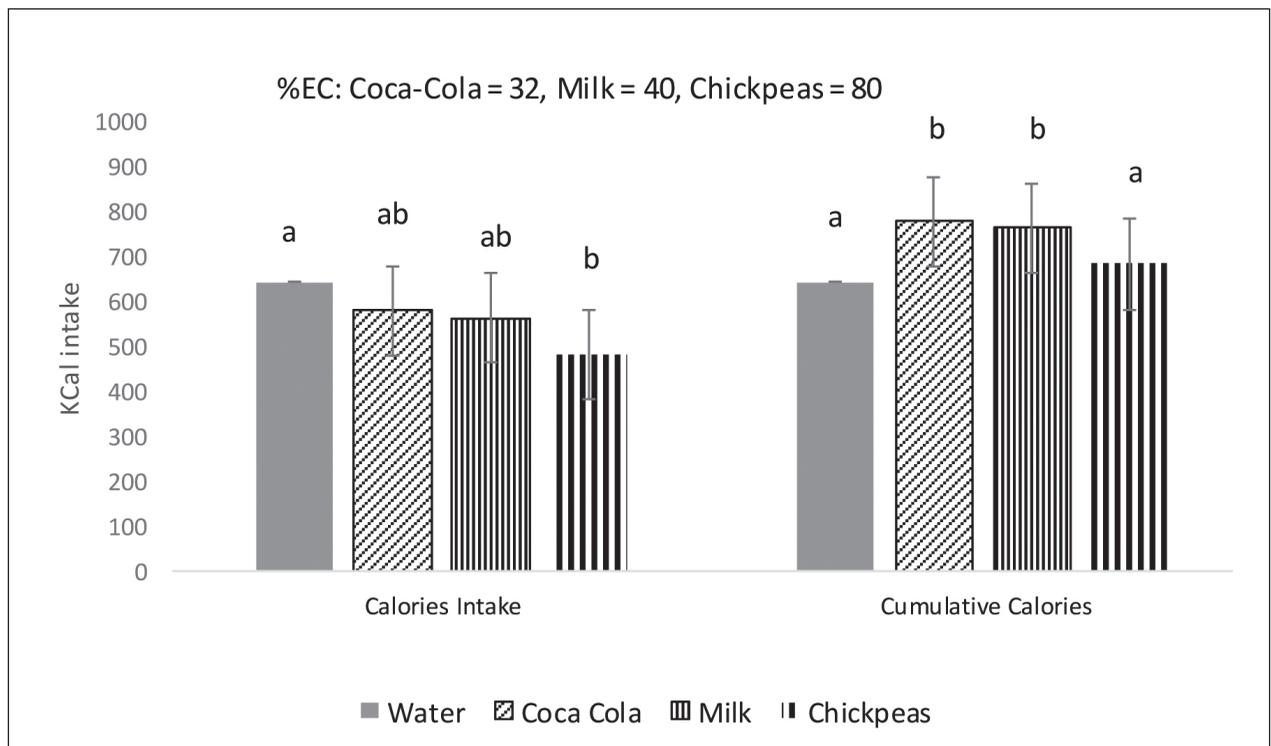
#### Relations among dependent measures

The average appetite AUC in each experiment had a positive correlation with Kcal intake ( $r = 0.352$ ,  $p < 0.05$  and  $r = 0.330$ ,  $p < 0.01$ ) in Experiment 1 and 2, respectively, supporting an increase in food intake with average increase in appetite scores as would be expected. On the other hand, blood glucose AUC for

test foods in both experiments was not correlated with food intake or with average appetite AUC. However, the correlation between the final blood glucose before the meal and calories intake from the meal was time dependent, for example, there was a negative correlation at 120 min in Experiment 1 and no correlation at 60 min in Experiment 2

#### Discussion

These results demonstrate greatest energy compensation after the chickpeas in both experiments; the largest difference (70% vs. 7%) being with white bread of comparable carbohydrate content. When Kcal and preload energy density were equated, the energy intake after the three preloads was not significantly different, yet the differences in energy compensation were still



**Figure 2.** Experiment 2 - Similar caloric density preload effect on energy intake, cumulative energy intake and percent energy compensation (%EC)<sup>s</sup> in healthy females.

Data presented is as Mean  $\pm$  SEM,  $n = 14$ ; Different superscript letters denote a significant difference at  $p < 0.05$  in the same group of bars (Tukey's posthoc test).

<sup>s</sup>%EC = [(Kcal intake at meal after the Control (water) preload - Kcal intake at meal after the Test preload (coca-cola, milk or chickpeas) / Kcal from the Test preload] \* 100.

reasonably high for chickpeas when compared with milk (a liquid, low GI food) (80% vs 40%) and Coca-Cola (a liquid, high GI food) (80% vs 32%). This suggests that cooked chickpeas with their low GI are better in compensating energy intake when fed at equicaloric or available carbohydrate levels and whether the comparable food is a solid or a liquid. Chickpeas are low GI and reportedly are more satiating because of their high fiber and protein content and, therefore, with slow rate of digestion and absorption sustaining euglycemia for an extended period of time (22). When the two equicaloric liquid foods, milk and Coca-Cola, with low and high GI based on differences in their protein, fat and carbohydrate contents were compared, milk did not produce a significantly lower energy intake than Coca-Cola, despite its low GI (Figure 2). Other workers have also demonstrated no difference between milk and Coca-Cola in subsequent energy intake (15, 23, 24).

To further elucidate why the two solid foods of different GI, bread and chickpeas, fed at the same available carbohydrates and volume load responded differently on food intake at a test meal, but why the two liquid foods of different GI, milk and Coca-Cola, fed at the same caloric level and volume load responded similarly in food intake, we examined time trends in blood glucose patterns. Being low GI, both chickpeas and milk, resulted in a lower peak than for the high GI foods, white bread or Coca-Cola; yet at 120 min the blood glucose was significantly higher after the chickpeas compared to that after bread (Table 2), and was associated with less hunger and thus a lower energy intake. At 60 min the glucose level remained above the baseline after all four treatments (Table 2), the energy intake from the meal was not different among them except for the chickpeas that was lower only compared with the water control. Cumulative energy intake was, however, significantly lower after both chickpeas and the control. These results are supported by the higher blood glucose concentration after chocolate milk (sweeter) and not plain milk (not sweet) 30 min before meal that lead to reduced food intake (14).

When correlations performed, the final blood glucose response before meal time was implicated in the correlation of feeling of hunger and food intake; in Experiment 1, there was a positive correlation

with both, but no correlation in Experiment 2. White bread, with blood glucose dropping below the baseline, showed only 7% EC, whereas the blood glucose after the Coca-Cola remained well above the baseline at one hour resulted in a much higher, 32% EC compared to white bread. Similarly, blood glucose concentration remaining above the baseline after milk preload resulted in 40% EC, only 8% more than after the Coca-Cola despite its low GI. Dove et al. (8) reported 8.5% compensation in energy consumption after skim milk compared to fruit juice. The pattern of blood glucose concentration and its influence on caloric intake might have changed after the high GI, Coca-Cola preload if the experiment time had extended beyond 60 min to allow a postprandial dip below baseline that would have triggered hunger signals by the brain to initiate eating.

The manipulating effect of blood glucose concentration on satiety and energy intake at the second meal therefore, emphasizes the need to measure blood glucose concentration simultaneously when measuring satiety and food intake. The concept of glucostatic theory presented earlier by Mayer (25) states that the transient decline in the blood glucose concentration when occurs at a correct magnitude and a correct time course is detected by the brain glucoreceptors, thus initiates feeding and when the blood glucose concentration increases to a certain high level, feeding is terminated. The reason Maersk et al. (23) could not explain why they were not able to reproduce the results of a similar study conducted by Dove et al. (8) despite the same study design and testing of the same foods for the same intermeal interval was probably that Dove et al served the preloads with breakfast which contributed more calories and sustained blood glucose for longer while Maersk et al served the preloads alone (8, 23). But since the blood glucose concentration was not measured in either study, the differences in their results were hard to explain reflected in a conflicting message for the public. Likewise, lack of blood glucose measurements could not accurately explain why chocolate milk and Coca-Cola showed no difference in EC when food intake measured at 30 min or when 1% milk was compared with Coca-Cola and fruit juice tested at 145 minutes before lunch (15, 26).

## Conclusions and recommendations

In conclusion, our results emphasize that it is not just the form of the foods such as solid or liquid, GI of a food such as low or high, its macronutrient composition or the energy density, it is rather the pattern of glucose that is imparted to the blood by a type of food consumed that might be influencing the feelings of hunger and thus the amount of calories consumption at the subsequent meal. Foods with low glycemic index containing dietary fiber and good quality of protein would encourage a sustained yet low peak glucose supply to the blood circulation as was seen in these two experiments. In order to prevent the occurrence of diabetes, monitoring body weight gain is imperative. Thus Consumption of foods with low GI value would prove beneficial in the prevention and management of obesity, hyperglycemia or hyperinsulinemia.

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### Ethical considerations:

Ethical issues (Including plagiarism, Informed Consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, etc.) have been completely observed by the authors.

## References

1. Yu YH, Vasseli JR, Zhang Y, Mechanick JI, Komer J, Peterli R. Metabolic vs. hedonic obesity: a conceptual distinction and its clinical applications. *Obs Rev* 2015; 16: 234-247.
2. Guariguata L, Whiting D, Hambleton I, Beagley J, Linnenkamp U, Shaw J. Global estimates of diabetes prevalence for 2013 and projections for 2035. *Diabetes Research and Clinical Practice* 2014; 103: 137-149 <http://dx.doi.org/10.1016/j.diabres.2013.11.002>.
3. Ludwig DS. Clinical update: the low-glycemic-index diet. *The Lancet* 2007; 369: 890-892.
4. Guyenet SJ, Schwartz MW. Regulation of food intake, energy balance, and body fat mass: implications for the pathogenesis and treatment of obesity. *J Clin Endocrinol Metab* 2012; 97: 745-755.
5. Cassidy BA, Considine RV, Mattes RD. Beverage consumption, appetite, and energy intake: what did you expect? *Am J Clin Nutr* 2012; 95: 587-593.
6. Mourao DM, Bressan J, Campbell WW, Mattes RD. Effects of food form on appetite and energy intake in lean and obese young adults. *Int J Obes (Lond)* 2007; 31: 1688-1695.
7. Mattes RD, Campbell WW. Effects of food form and timing of ingestion on appetite and energy intake in lean young adults and in young adults with obesity. *J Am Diet Assoc* 2009; 109: 430-437.
8. Dove ER, Hodgson JM, Puddey IB, Beilin LJ, Lee YP, Mori TA. Skim milk compared with a fruit drink acutely reduces appetite and energy intake in overweight men and women. *Am J Clin Nutr* 2009; 90: 70-75.
9. Almiron-Roig E, Flores SY, Drewnowski A. No difference in satiety or in subsequent energy intakes between a beverage and a solid food. *Physiol Behav* 2004; 82: 671-7.
10. Almiron-Roig E, Chen Y, Drewnowski A. Liquid calories and the failure of satiety: how good is the evidence? *Obes Rev* 2003; 4: 201-212.
11. Anderson GH, Catherine NL, Woodend DM, Wolever TM. Inverse association between the effect of carbohydrates on blood glucose and subsequent short-term food intake in young men. *Am J Clin Nutr* 2002; 76: 1023-1030.
12. Fournel A, Marlin A, Abot A, et al. Glucosensing in the gastrointestinal tract: impact on glucose metabolism. *Am J Physiol Gastrointest Liver Physiol* 2016. Doi:10.1152/ajpgi.00015.201.
13. Mattes R. Fluid calories and energy balance: the good, the bad, and the uncertain. *Physiol Behav* 2000; 89: 66-70.
14. Panahi S, Luhovyy BL, Liu TT, Akhavan T, ElKhoury D, Goff HD, Harvey AG. Energy and macronutrient content of familiar beverages interact with pre-meal intervals to determine later food intake, appetite and glycemic response in young adults. *Appetite* 2013; 60: 154-161.
15. Harper A, James A, Flint A, Astrup A. Increased satiety after intake of a chocolate milk drink compared with a carbonated beverage, but no difference in subsequent ad libitum lunch intake. *Br J Nutr* 2007; 97: 579-83.
16. Badr HE, Shah NM, Shah MA. Obesity among Kuwaitis aged 50 years or older: prevalence, correlates and comorbidities. *The Gerontologist* 2012; 53: 555-566.
17. Herman CP. Restrained Eating. *Psychiatric Clinics of North America* 1978; 1: 593-607.
18. Escalante PJM, Alpizar SM. Change in insulin sensitivity, secretion and glucose effectiveness during menstrual cycle. *Arch Med Res* 1999; 30: 19-22.
19. Zafar TA, Kabir Y. Chickpeas suppress postprandial blood glucose concentration and appetite and reduce energy intake at the next meal. *J Food Sci Technol* 2017; 54: 987-94.
20. Zafar TA, Kabir Y, Ghazaii C. Low glycemic index foods suppress glycemic response, appetite and food intake in young Kuwaiti females. *Kuwait J Sci Eng* 2011; 38: 111-23.
21. Flint A, Raben A, Blundell JE, Astrup A. Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *Inter J Obesity* 2000; 24: 38-48.

22. Foster-Powell K, Holt SH, Brand-Miller JC. International table of glycemic index and glycemic load values. *Am J Clin Nutr* 2002; 76: 5-56.
23. Maersk M, Belza A, Holst JJ, Fenger-Gron M, Pedersen SB, Astrup A, Richelsen B. Satiety scores and satiety hormone response after sucrose-sweetened soft drink compared with isocaloric semi-skimmed milk and with non-caloric soft drink: a controlled trial. *European Journal of Clinical Nutrition* 2012; 66: S23-S29.
24. Soenen S, Westerterp-Plantenga MS. No difference in satiety or energy intake after high-fructose corn syrup, sucrose, or milk preloads. *Am J Clin Nutr* 2007; 86: 1586-1594.
25. Mayer J. Glucostatic mechanism of the regulation of food intake. *N Engl J Med* 1953; 249: 13-6.
26. Almiron-Roig E, Drewnowski A. Hunger, thirst, and energy intakes following consumption of caloric beverages *Physiol Behav* 2003; 79: 767-73.

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