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Postprandial Glucose and Insulin Response to Meal Sequence Among Healthy UAE Adults: A Randomized Controlled Crossover Trial

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Background: Dietary patterns that lower postprandial glycemia have been effective in preventing type 2 diabetes. Consuming macronutrients in a specific sequence within a meal has been considered as a novel strategy to reduce post-meal glucose spikes. Therefore, this study aimed to investigate the effect of meal sequence on postprandial glucose and insulin response among healthy adults in the United Arab Emirates.

Methods: Eighteen healthy adults participated in a cross-over randomized controlled trial. Two isocaloric meals were consumed separately in a different order: a standard mixed meal (SMM) vs vegetables and protein first followed by carbohydrates (VPF) meal. The postprandial glucose and insulin levels were determined at Fasting, 30, 60, and 120 min. Visual Analog Scale (VAS) rating was used to assess hunger at similar frequencies.

Results: The mean glucose and insulin levels significantly reduced ($p = 0.001$) following VPF meal compared to SMM at 30 min. The incremental area under the curve (iAUC0–120) for glucose following the VPF meal sequence was 40.9% lower ($p = 0.03$) compared with the SMM (572.83; 95% CI 157.3 to 988.2) vs (968.5; 95% CI 692.4 to 1244.8 mg/dL). Furthermore, the iAUC0–120 for insulin following the VPF meal sequence was 31.7% lower than the SMM meal sequence. (2184; 95% CI 1638.30 to 2729) vs (3196.94; 95% CI 2328.19 to 4065.69) mIU/mL × 120 min, $P = 0.023$). The feeling of fullness measured by the hunger scale showed that the feeling of fullness was higher after 60 and 120 minutes ($p = 0.05$, $p = 0.04$) with the VPF meal sequence compared to the SMM sequence. Although, there is no significant difference in the Area under the curve (AUC) for hunger rating between the two meals.

Conclusion: The VPF meal sequence could significantly reduce postprandial glucose and insulin levels and sustain fullness after a meal. Meal sequencing could be an effective dietary strategy for improving the postprandial glucose and insulin response in healthy adults.

Keywords: meal order, glucose excursions, insulin, Arab

Introduction

Type 2 diabetes mellitus (T2DM) affects approximately 462 million people globally, and its prevalence and incidence are steadily increasing.[1] Prevalence of diabetes in the UAE was reported to be 16.3% versus 9.3% globally.[2] The earliest evident metabolic change in the course of the disease is usually a loss of postprandial glucose concentration control.[2] Postprandial glucose concentration is considered an important independent contributor to macrovascular and microvascular events in T2DM.[3,4] Thus, glycemic control by controlling elevated postprandial blood glucose level is considered a crucial therapeutic goal to delay the onset of T2DM.[5] Dietary solutions to reduce postprandial glucose excursions rely on the quality and quantity of dietary carbohydrates as the main determinants of glycemic and insulinemic response.[6] However, other macronutrients, such as fat and protein, can also alter postprandial blood glucose level.[7,8]

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Previous studies have demonstrated that different eating patterns and meal sequences can alter the postprandial glycemic response. For instance, preloading main meals with fat or protein and changing the macronutrient makeup of a meal could lower the post-meal glucose level.[9–11]

A study of 11 participants with T2DM showed a significant reduction in postprandial glucose and insulin excursions when the meal sequence was protein and vegetables first, followed by carbohydrates, compared to the same meals in reversed order.[12] Similar findings were also reported in an earlier study performed on 16 participants with T2DM; the carbohydrate-last meal pattern showed a significant reduction in postprandial glucose and insulin excursions compared to the carbohydrate-first meal sequence.[13]

There are no studies on meal sequence strategies and their effects on postprandial glucose regulation or insulin response among healthy adults in the UAE with a standard meal in this region. The findings of this study would offer a simple, effective, safe, and inexpensive therapeutic approach for preventing and managing postprandial hyperglycemia.

This study aimed to compare the effects of meal sequences vegetable and protein first followed by carbohydrates VPF meal to a standard mixed meal (SMM) on postprandial glucose response among healthy adults in the UAE. Additionally, the current study examines the effect of meal sequence on insulin response and hunger-fullness using validated Visual analog scales. We postulated that consuming a meal in a sequence (VPF) significantly reduces the postprandial glucose level and insulin response compared to a (SMM).

Methods

Subject Inclusion and Exclusion Criteria

The inclusion criteria were age of 18–59 years Arabs ethnicity; BMI: 18.5–29.9 kg/m^2; fasting blood glucose level less than 100 mg/dL; non-smoker; no genetic or metabolic diseases; and no known medical condition. The following participants were excluded: any participants with pre-existing diabetes; pregnant and breastfeeding women; participants with behaviors or/and health conditions that are known to affect glucose or insulin concentrations, such as smoking, gastrointestinal conditions, bariatric surgery, gastroparesis and reactive hypoglycemia; participants who consumed medication that might affect glucose or insulin response; and participants with reported allergy or intolerance to any of the meal components (White Basmati rice, grilled chicken, cucumber, tomato, and lettuce salad with lemon olive oil dressing).

The study complied with the Helsinki Declaration and obtained ethical approval from the Ethics and Research committee of the University of Sharjah (reference number. REC-22-10-18-S). The protocol was registered on ClinicalTrials.gov (NCT05878301, 20/08/2023).

Study Protocol

We designed a randomized controlled cross-over open-labeled study. We estimated that a sample size of 18 participants would be sufficient to detect an effect size of 25% in blood glucose response, with a significance level (α) of 0.01, two-tailed, and with a power of 95% and allowing for 10% dropout. Participants received two isocaloric test meals (SMM and VPF meal sequences) served with 250 mL water where each participant consumed the meal alone and separated by a 7–10-day washout period to minimize the effect of carryover. The isocaloric meal (Table 1) consisted of 200 g boiled flavored rice, 100 g grilled chicken fillet, and 150 g mixed vegetable Arabic salad with 20 g lemon olive oil dressing (431 kcal: 54 g carbohydrates, 34 g protein and 8 g fat) and was served to participants who arrived after 10–12 h of overnight fast. The order in which they received the interventions (the sequence) were randomized using online research randomizer. Participants were asked to restrict physical activity and avoid fast food prior to each test meal. All meals were prepared 1 h before the test and delivered to the Diabetes Center, University of Sharjah. A microwave oven (Sharp, R-28CT[S]) was used for 50 s to reheat the meals before consumption. Participants were closely monitored to ensure that all meals were fully consumed.

Table 1 Test Meal Composition

Ingredients	Amount	Kcal	Carbohydrate (g)	Protein (g)	Fat (g)
Chicken breast fillet	100g	151	0	29	3
Basmati rice long grain	200g	210	49	4.2	0
Lettuce (romaine)	50 g	8.5	1.65	0.62	0.15
Tomatoes	50g	12.5	2	0.40	0.15
Cucumber with skin	50g	8.5	1.5	0.35	0.05
Salad dressing (lemon, olive oil)	20mL	40.86	0.20	0.04	4.67
Water	250mL				
Total		431.36	54.35	34.61	8.02

Data and Blood Sample Collection

Figure 1 shows the data collection procedure. The study was conducted at the College of Medicine at the Diabetes and Research Center, University of Sharjah, between November and February 2023. Participants were recruited through an advertisement, and personal communication and selected using the convenient snow-ball sampling method. Eligibility screening was performed using a screening questionnaire that included information regarding allergies to the meal components, behavioral practices such as smoking and alcohol consumption, and any condition or medication that might affect the blood glucose readings. Furthermore, a participant information sheet was provided to explain the study procedure before signing of the consent form. After obtaining the participants' approval, an instruction sheet and invitation to the intervention site were provided. The participants were then

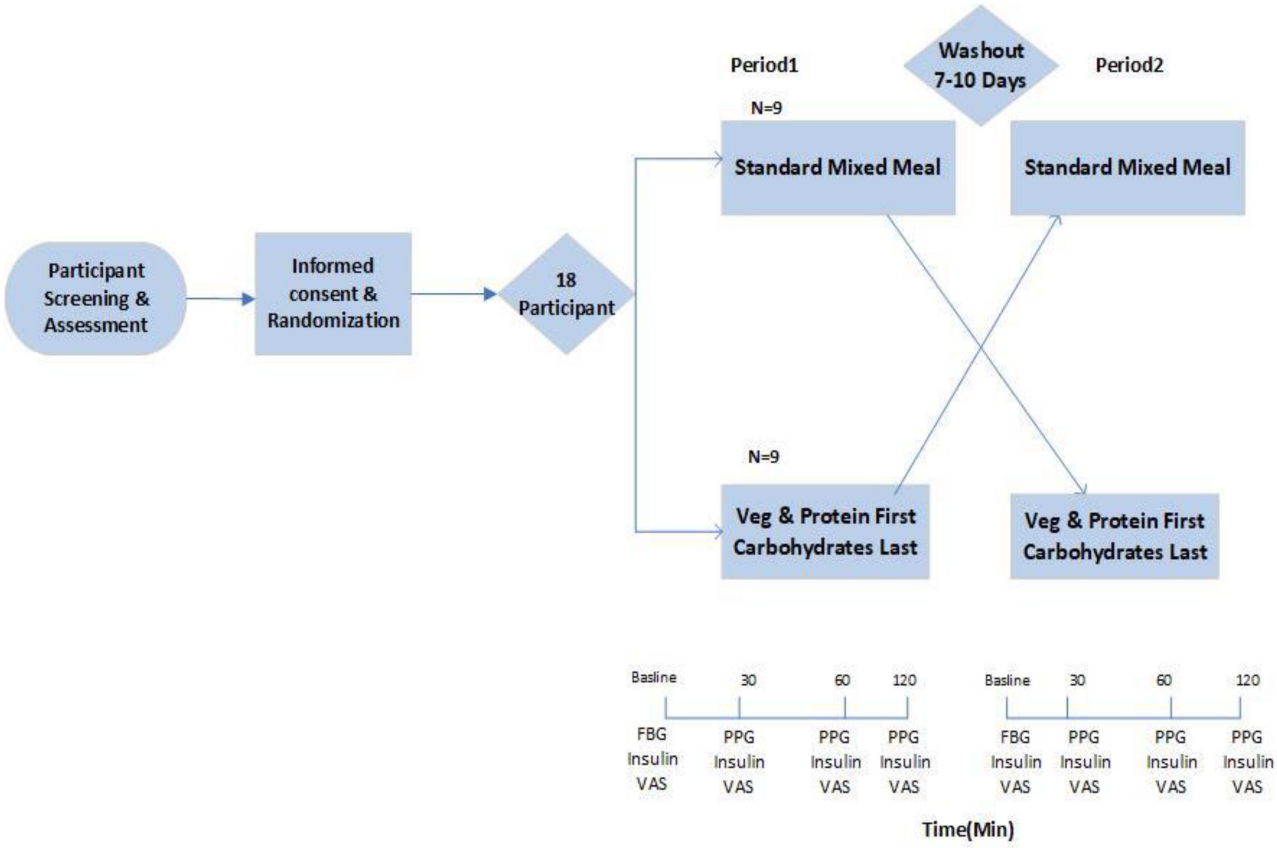

Figure 1 Data collection flow diagram.

randomly assigned to receive the test meal; the test meal was to be consumed in different sequences after 10–12 h fast on two separate days (7–10 days apart) in a randomized order. The two isocaloric test meal food sequences were as follows: standard mixed meal (mix of rice, chicken, and vegetable) consumed over 15 min and intervention meal (protein and vegetables first over 10 min, a 10-min rest interval, and then carbohydrates over 10 min). Participants were closely monitored to assure that all meals were fully consumed within the allotted time. Blood samples (total 12 mL: 3 mL at 0 (fasting), 30, 60, and 120 min) were drawn from the left- and right-antecubital veins using a butterfly needle at baseline (before meal ingestion) and 30-min intervals up to 120 min after the start of the meal. Plasma glucose and serum insulin concentration were analyzed using the Roche Cobas 6000 Chemistry Analyzer (Roche, Hitachi, USA) at the SRL diagnostic laboratory, Dubai Healthcare City. Intra-assay and inter-assay coefficient of variations were 1% and 2.08%, respectively.

Statistical Analysis

Data were analyzed using Stata Statistical Software Release 13 (StataCorp LP; College Station, TX. Data were checked for normal distribution, and continuous data, such as age, BMI, and blood pressure were expressed as mean ± SD, and categorical data, such as sex, nationality, and profession, were expressed as counts and percentages. Homeostasis model assessment-estimated insulin resistance (HOMA-IR) was computed as follows: [fasting insulin (μIU/mL) × fasting glucose (mg/dL)/405]. Homeostasis Model Assessment of β-Cell Function (HOMA-B) was calculated using the following formula: [360 × fasting insulin (μIU/mL)/fasting glucose (mg/dL) − 63]. Insulinogenic Index (IGI) was calculated from the change in insulin/change in glucose over the first 30 min after the ingestion of the test meal (IGI = Δ insulin [30–0 min] [μIU/mL]/Δ glucose [30–0 min] [mg/dL]).14 Paired *t*-test was used to find the difference between the two test meals. The Incremental Area Under the Curve (IAUC) and Area Under the Curve (AUC) of each measurement was calculated according to the trapezoidal rule and analyzed using the paired *t*-test. P values ≤0.05 were accepted as statistically significant.

Results

The clinical and demographic characteristics of the study participants are presented in Table 2; 18 healthy adults (7 male and 11 female individuals) completed the study. The participants consumed the test meals after signing the informed

Table 2 Clinical and Demographic Characteristics of the Study Participants (N = 18)

Variable	N=18
Sex Females, n (%)	11 (61)
Males, n (%)	7 (39)
Arab Ethnicity, n (%)	15 (83)
Emirati, n (%)	3 (16)
Age (years)	31.1 ± 8.6
Systolic blood pressure (mm Hg)	111 ± 13
Diastolic Blood Pressure (mm Hg)	73± 5
Weight (Kg)	71.7 ± 9.6
Height (cm)	167.7 ± 8.6
Body mass index (kg/m^2)	25.4 ± 2.3
Waist circumference (CM)	84.8 ± 7.8
Fat mass (%)	29±7.7
Fat free mass (kg)	51±10.2

Note: Data are n, n (%), or mean ± SD.

consent. The participants' average age was 31.1 years (range 20–45 years), and the mean BMI was 25.4 kg/m2 (50% normal bobweight and 50% overweight, range: 21–28.8 kg/m2). The mean fasting blood glucose level and systolic and diastolic blood pressure were within the normal range. Majority of the participants (83%) were of Arab ethnicity (3 Jordan, 3 Palestine, 2 Sudan, 1 Algeria, 1 Tunisia, 1 Egypt, and 1 Yemen), and 16% were Emirati nationals. Among the participants, 22.2% were students from the University of Sharjah and 77% were University staff; 39% were undergraduate students and 61% were postgraduate students. Waist circumference ranged from 72–91 cm for female individuals and 88–103 cm for male individuals. The mean (HOMA_IR) and (HOMA-B) values were within normal range 1.8 and 121, respectively.[14,15]

The postprandial responses to the SMM and VPF meal sequences over 120 min are presented in Figure 2. Postprandial mean glucose concentrations were significantly decreased by 20.9% (P = 0.001) at 30 min following the VPF meal sequence compared with that following the SMM sequence. The (iAUC0–120) following the VPF meal sequence was 40.9% lower (572.83; 95% CI 157.3 to 988.2) vs (968.5; 95% CI 692.4 to 1244.8) mg/dl × 120 min, P = 0.035) compared with that following the SMM sequence. There were no significant differences between the two meal sequences at baseline fasting glucose concentrations or at 60 min and 120 min.

The insulin response to different meal sequences is presented in Figure 3. The VPF meal sequence resulted in 60.8% (P = 0.001) lower postprandial mean serum insulin response at 30 min and significantly lower iAUC0–120 by 31.7% (2184; 95% CI 1638.30 to 2729) vs (3196.94; 95% CI 2328.19 to 4065.69) mIU/mL × 120 min, P = 0.023) compared with the SMM sequence. There were no significant differences in baseline fasting insulin concentrations, 60 min and 120 min between the two meal sequences on the 2 days of trial visits. No significant difference was found between the insulinogenic index measured in the SMM and VPF meal sequences (2.22; 95% CI 1.56 to 2.88) vs (2.89; 95% CI −2.2 to 8.04), P = 0.78).

Figure 4 shows the mean hunger scale rates. There were no significant differences at baseline in the hunger scale between the SMM and VPF meal sequences. There were slight differences in mean postprandial fullness at 30 min after the VPF meal sequence compared to that after the SMM sequence; however, it is not significant (P = 0.086). Fullness at 60 and 120 min after the VPF meal sequence was significantly higher compared to that after the SMM sequence (P = 0.05 and P = 0.04, respectively). There were no significant differences in (AUC) for hunger scale between the SMM and VPF meal sequences (P = 0.017).

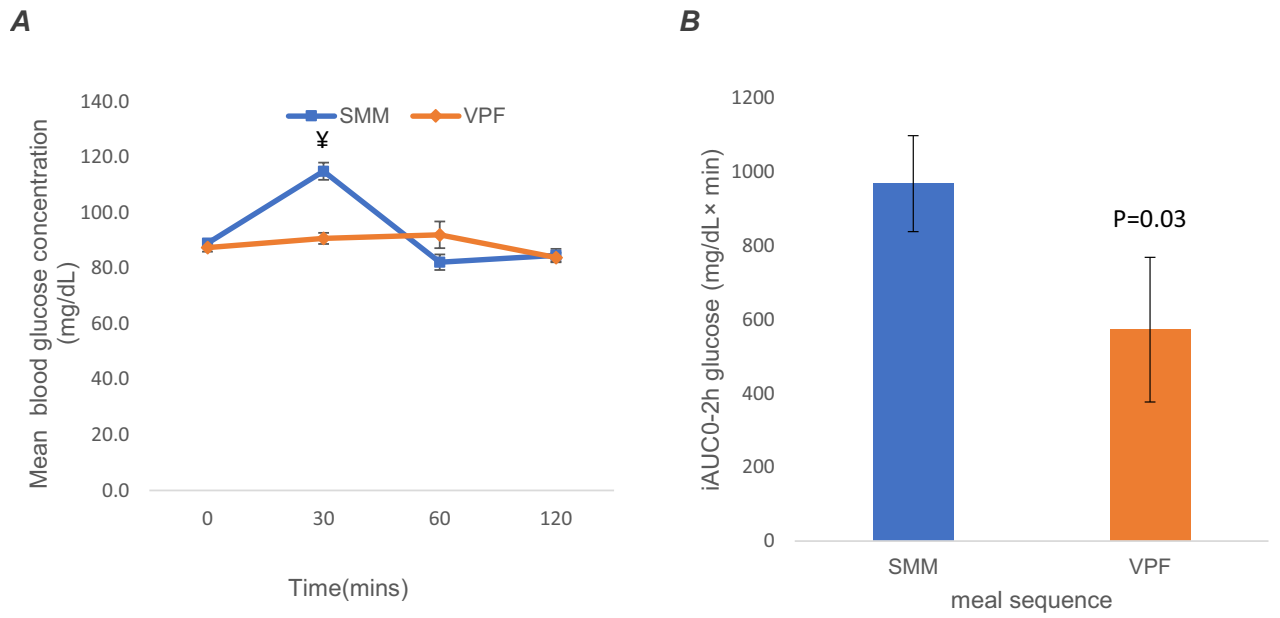

Figure 2 Mean glucose response and IAUC (glucose). Mean change from baseline postprandial glucose response following the standard mixed meal (SMM) and vegetable protein first (VPF) meal sequences over 120 min after the meal (**A**). iAUC for blood glucose concentration following the SMM and VPF meal sequences over 120 min after the meal (**B**). (n = 18). Error bars shows standard error. ¥Significant differences (P ≤ 0.05) between the SMM and VPF meal sequences.

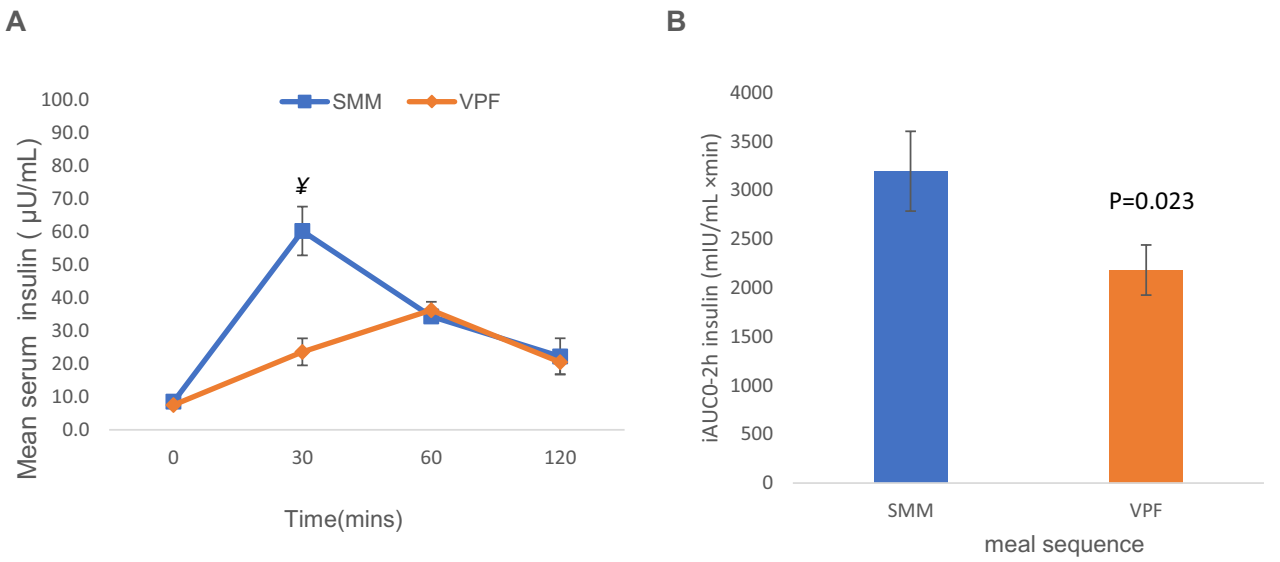

Figure 3 Mean change in insulin response and IAUC (insulin). Mean change from baseline postprandial insulin response following the standard mixed meal (SMM) and vegetable protein first (VPF) meal sequences over 120 min after the meal (**A**). iAUC for blood insulin concentration following SMM and VPF meal sequences over 120 min after the meal (**B**). (n=18). Error bars shows standard error. ¥Significant differences (p≤0.05) between the SMM and VPF meal sequences.

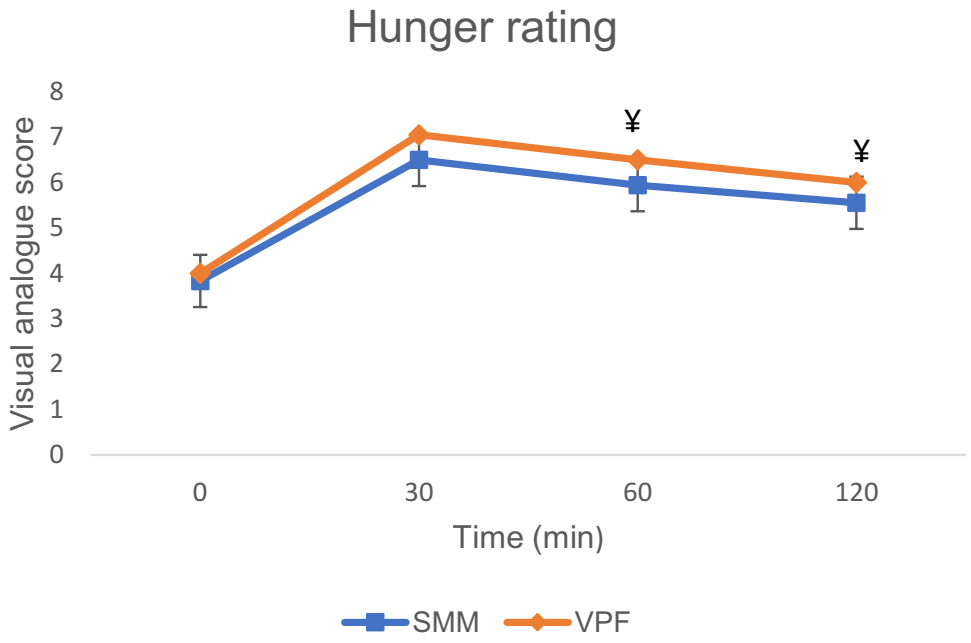

Figure 4 Mean change in visual analog scale hunger scale from baseline following standard mixed meal (SMM) and vegetable protein first (VPF) meal sequences over 120 min after the meal (n = 18). ¥Significant differences (p≤0.05) between the SMM and VPF meal sequences.

Discussion

In this study, we demonstrated that, in healthy adults, manipulating meal sequences significantly impacts postprandial glucose and insulin levels and hunger rating. Compared with the SMM sequence, the VPF meal sequence decreased postprandial glucose, required less insulin secretion, and resulted in a higher post-meal fullness rate. The findings in the current study suggested that eating vegetables and proteins before carbohydrates could be a simple and new behavioral strategy to decrease postprandial glycemic spikes without any required increase in insulin secretion in healthy individuals.

Diabetes and cardiovascular disease are significantly influenced by postprandial glycemia. [16] Blood glucose spikes after meals are more closely associated with atherosclerosis than fasting plasma glucose or HbA1c levels.[17] Several factors may affect blood glucose response including gastric emptying (GE) rate, meal components, the type of carbohydrate and metabolic reactions of glucose in the liver.[18,19]

In addition, we were comparing the standard eating pattern to meal sequence and the duration difference of eating the sequenced meal could be one of the factors that have contributed to the positive effect on glucose and insulin and fullness.

This finding is consistent with the result of a previous study by Shukla et al (2017) which found a significant reduction in glucose level and insulin response in patients with T2DM following a meal sequence of vegetables and protein first, followed by carbohydrates, and suggested that less insulin is required in controlling carbohydrates with this meal pattern. The effect of carbohydrate-last meal sequence could be explained by the slower absorption of carbohydrates and the lower GE rate.[13] This finding supports the rationale that altering the rate of nutrient absorption is a key therapeutic principle in diabetes.

The rate at which nutrients leave the stomach and are transported to the small intestine is largely determined by GE.[19] Consumption of dietary fiber has been shown to delay GE in healthy individuals.[20] The vegetables used in the current study weighed 150 g and contained around 2 g of dietary fiber. This suggested that even a small preload of vegetables before carbohydrate intake reduced postprandial glucose level and insulin responses in a healthy individual. This result was confirmed in a previous study in which a small amount of dietary fiber was effective in reducing postprandial glucose and insulin spikes.[21] This is due to the effect of dietary fiber in inhibiting glucose diffusion and delaying carbohydrate digestion and absorption.[22] The result in the current study showed that dietary carbohydrates ingested after vegetables were digested slowly and needed less insulin to be metabolized.

The current study demonstrated that the meal sequence of vegetables and protein before carbohydrates had a positive result in extending satiety compared to that of the SMM sequence. This might be due to the fact that high-protein diets induce more satiety compared to a high-carbohydrate or high-fat diet.[23] Particularly, 25–30 g of protein per meal can increase satiety and reduce energy intake (per calorie).[24] The protein used in our study was 100 g of chicken breast, which contained approximately 30 g of protein. In addition, there is a positive association between satiety, reduction of food intake, and gut hormones such as GIP-1, and dietary protein is crucial in stimulating these hormones.[24,25] Previous studies performed on the meal sequence of vegetables and protein before carbohydrates demonstrated that this meal sequence improved the release of gut hormone compared with other meal sequences in both patients with diabetes and healthy individuals.[13,21,26,27]

There is clear evidence that the incretin maintains glucose homeostasis. The proposed direct effects of incretin hormone, primarily GLP-1, include the stimulation of insulin release, inhibition of glucagon production that reduces food intake, and the slowing of GE rate.[28] Unfortunately, incretins response was not evaluated in the current study. However, one study using a similar meal sequence did not find a significant difference in measured hunger and fullness.[21]

There is a large spike in glucose levels and insulin responses at 30 min following the SMM sequence, whereas no such changes were seen following the VPF meal sequence. Recent publications on the same meal sequences have demonstrated a similar increase in postprandial glucose and insulin levels when all meal components were ingested together.[13,21,29]

The VPF meal sequence reduced glucose IAUC by 40.9%, which is considered comparable to the effect observed in the use of blood glucose-lowering pharmacological agents, such as acarbose and nateglinide, in which iAUC was reduced by 31% and 64%, respectively, compared with placebo.[30,31]

Limitation and Strength of the Study

Potential limitations of the study glucose levels and insulin responses were only analyzed after 120 min of meal consumption; thus, it is possible that the results do not reflect the full picture of how meals affect glucose and insulin levels. Our current study has several strengths, including a robust study design and adequate power. In addition, the use of real-world meals that resemble Arab eating habits in both quali quantity could have positive implications in dietary recommendations to the Arab population in the UAE. However, the results may not be applicable to other populations with different eating habits.

It is recommended that future research be conducted on the effects of meal sequencing strategies on participants with impaired glucose tolerance and diabetes. It may be useful to study the effects of meal sequencing on other metabolic

markers in long-term clinical care interventions for pre-diabetic and diabetic patients. To improve dietary recommendations in clinical practice, other meal sequences in different cultures should be measured.

In addition, measuring the incretin hormone responses following the meal sequence, such as GLP-1, GIP, satiety hormones, and GE, could add value to the physiological changes associated with meal sequencing. Further studies with longer follow-up beyond 120 min to delineate the full impact, including the delayed effects and the mechanism underlying the glycemic effect of meal sequence, are required.

Conclusion

In conclusion, this study showed that eating carbohydrates after vegetables and protein portions improved postprandial glycemic spikes, insulin secretion, and satiety in healthy individuals. This study adds evidence to the theory that order of food intake could reduce postprandial glycemic response in a straightforward and inexpensive manner. These results suggest that meal sequencing could reduce T2DM risk by improving postprandial glucose spikes and could be considered in developing a local guideline for T2DM.

Data Sharing Statement

The data used to support the findings of this study are restricted by the University of Sharjah Research Ethics Committee – UAE, in order to protect participant privacy. Data are available from Ayah Shaheen, Aya.n.shaheen@gmail.com for researchers who meet the criteria for access to confidential data.

Informed Consent Statement

Written informed consent was obtained from all participants involved in the study.

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Disclosure

The authors report no competing interests in this work.

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