British Journal of Nutrition (2020), 123, 664–672
doi:10.1017/S0007114519003180

Breakfast consumption modulates postprandial glycaemic, insulinaemic and NEFA response in pre-diabetic Asian males

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(Submitted 22 July 2019 – Final revision received 18 November 2019 – Accepted 26 November 2019 – First published online 13 December 2019)

Abstract

Breakfast consumption is associated with a variety of nutritional and lifestyle-related health outcomes. The objective of the present study was to investigate how the consumption of breakfast affected blood glucose, insulin and NEFA profiles. A lower postprandial blood glucose, insulin and NEFA response is associated with a lower risk of development of metabolic diseases. In a randomised crossover non-blind design, thirteen pre-diabetic Chinese adult males (BMI 26·7 (SD 4·2) kg/m^2) attended two sessions where they either consumed a high-glycaemic index breakfast or no breakfast consumption. Changes in glycaemic response over 27 h periods were measured using the Medtronic MiniMed iPro^{TM}2 continuous glucose monitoring system. Blood samples were collected using a peripheral venous catheter at fixed intervals for 3 h after the test meal and 3 h after standardised lunch consumption. Postprandial glucose, insulin and NEFA response was calculated as total AUC and incremental AUC using the trapezoidal rule that ignored the area under the baseline. It was found that breakfast consumption significantly decreased postprandial glucose, insulin and NEFA excursion response at lunch time ($P = 0{\cdot}001$). Consumption of breakfast attenuated blood glucose profiles by minimising glycaemic excursions and reduced both insulinaemic and NEFA responses in pre-diabetic Asian males during the second meal. This simple dietary intervention may be a novel approach to help improve subsequent lunch glycaemic responses in Asians at high risk of developing diabetes.

Key words: **High-glycaemic index breakfast: NEFA: Blood glucose: Insulin: Continuous glucose monitoring system**

Type 2 diabetes is one of the most common and rapidly rising non-communicable diseases worldwide. In 2015, it was reported that 415 million adults had the disease, and the occurrence is expected to increase to 642 million in 2040[1]. The rising epidemic of type 2 diabetes has serious consequences for healthcare expenditure; the global health cost on diabetes was estimated to be at least USD$673 billion in 2015 and is projected to increase to USD$802 billion by 2040[1]. The public health burden of type 2 diabetes makes effective treatments and prevention strategies necessary. Much focus has been on Asia as the Asian phenotype has been shown to be more susceptible to diabetes than Caucasians. It was estimated that 56 % of the global diabetic population inhabited the South-East Asia Region or the Western Pacific Region in 2015[1–3]. Asians are also more susceptible to rapid transition from pre-diabetes to diabetes status with greater metabolic consequences[4,5]. This warrants a need to find preventive intervention methods that suit the Asian phenotype.

The American Diabetes Association recommends that in general, pre-diabetic and diabetic people should consume food products that raise the blood glucose at a slower rate. A rapid increase in postprandial blood glucose induces hormonal and metabolic changes that may affect health. A slower rise in postprandial blood glucose has been reported to significantly decrease the development of chronic diseases such as diabetes[6–9].

Glycaemic index (GI) and glycaemic response were concepts introduced to classify the blood glucose raising potential of carbohydrate foods. Some research has shown that foods with lower GI and glycaemic response are associated with a lower risk of development of type 2 diabetes[6,10]. A lower glycaemic response reduced the amount of insulin needed for the removal of glucose from the blood, this may increase insulin sensitivity, which in turn also reduced the risk of development of metabolic diseases[11–13]. Elevated fasting NEFA was also reported to be

Abbreviations: GI, glycaemic index; iAUC, incremental AUC; tAUC, total AUC.

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positively associated with the risk of development of type 2 diabetes[14–17].

Eating pattern has also been reported to have an association with the development of type 2 diabetes[18,19]. Much emphasis is placed on breakfast as regular breakfast consumption has been reported to favourably affect body composition and chronic disease risk markers. A reduction in risk factor for type 2 diabetes, such as lower BMI, was reported to be found in people who have habitual breakfast consumption[20–22]. However, much of these researches are longitudinal studies. Although there are certain studies that focus on the acute effects after breakfast consumption, they focused on the composition of the breakfast meal, rather than on the benefit of breakfast consumption or not[23,24]. Therefore, the objective of the present study was to investigate the acute effect of breakfast consumption or not on the 'second-meal effect', that is, the impact on blood glucose, insulin and NEFA at lunch in Asians. Additionally, this is also the first study that focuses on a high-GI breakfast that is commonly consumed in a traditional Asian diet[25]. Exploring the impact of breakfast consumption on glycaemic excursions throughout the day may also provide mechanistic evidence for the health benefits of breakfast observed in epidemiological research.

Methods

This trial was registered with ClinicalTrials.gov as NCT03257059.

Participants

The inclusion criteria were Asian Chinese males aged between 40 and 65 years without any metabolic diseases (such as diabetes, hypertension, etc.) and known glucose-6-phosphate dehydrogenase deficiency. They must also be self-reported regular breakfast consumers, have regular sleep/wake patterns, non-smoker and BMI above 23 kg/m^2. Only individuals with a fasting blood sugar level from 100 to 125 mg/dl (5·6–7·0 mmol/l) were recruited in accordance with the definition of pre-diabetics by the American Diabetes Association[26]. Only males were included to eliminate potential variations due to hormonal changes during menstrual cycles in women. In addition, individuals with medical conditions and/or taking medications known to affect glycaemia (glucocorticoids, thyroid hormones, thiazide diuretics), intolerances or allergies to foods, partake in sports at the competitive and/or endurance levels, intentionally restrict food intake or have been on antibiotics at any time 3 months before the study period were excluded from the study as all of these were reported to have an impact on glucose and lipid metabolism of the body[27].

The study was conducted in accordance with the guidelines laid down in the Declaration of Helsinki, and all procedures involving human participants were approved by the Domain Specific Review Board of National Healthcare Group, Singapore (reference no. 2017/00537). The study protocol was explained to the subjects, and they gave their written informed consent before participation.

Study design

The study was a randomised crossover trial, where participants were subjected to two test sessions: breakfast consumption or no breakfast consumption in a randomised order with a minimum of 3 d (washout period) between the two test sessions. The blinding of participants to the two test sessions was not possible in the present study; the participants were told that the purpose of our study was to investigate the health effects of different breakfast meals.

Volunteers who expressed interests and met all inclusion criteria attended a screening session in the morning after an overnight fast, where written informed consent was obtained from the participants. The screening session and the test sessions were all held in Clinical Nutrition Research Centre, Singapore. During the screening session, participants will receive an informed consent form and given ample time to go through it and rectify any queries they have. If they decide to take part in the study, they will be asked to sign the informed consent form. Baseline measurements including fasting blood glucose, anthropometric measurements and blood pressure will be collected from each participant in order to determine their eligibility. Height was measured using a stadiometer (Seca Limited), blood pressure with an Omron blood pressure monitor (Model HEM-907; Omron Healthcare Singapore) and body weight and fat percentage with a bioelectrical impedance scale (Tanita BC-418). Blood glucose level was measured using the HemoCue 201$^+$ Glucose RT analyser (HemoCue Ltd). The first two drops of expressed blood were discarded, and the next drop was collected directly into a microcuvette for blood glucose analysis. The Hemocue is a reliable method that has intra- and inter-assay CV of 1·2 and 1·3 %, respectively, for capillary blood glucose analysis, and it is an accepted method for blood glucose assessments by the FAO/WHO[28]. Participants will then be scheduled for the two test visits, interspaced by a minimum of 3 d.

Each test session spanned three consecutive days, and the iPro™2 continuous glucose monitoring system (iPro™2 Professional CGM-Medtronic MiniMed) was used to obtain continuous interstitial glucose readings throughout the 3 d.

On day 1 of each test session, participants came to the centre for the insertion of the continuous glucose monitoring system and the collection of their standardised dinner, which was to be consumed at 19.30 hours. On day 2 of each test session, participants came to the centre after fasting for 10 h and subjected to either breakfast consumption or no breakfast consumption treatment. Baseline venous blood was collected by cannulation into Vacutainers® (Belton Dickinson Diagnostics) containing disodium EDTA for the analyses of plasma glucose, insulin and NEFA concentrations. The tubes were centrifuged at 1500 ***g*** for 10 min at 4°C (Sorvall™ ST 16 Centrifuge, Thermo Fisher Scientific). Plasma was aliquoted into Eppendorf tubes and stored at −80°C until analysis. The indwelling catheter was kept patent by flushing with 3 ml non-heparinised saline. After collecting the baseline venous blood samples, participants consumed either the test breakfast or did not consume any test breakfast and a 3 h postprandial venous blood collection commenced at 15, 30, 60, 90, 120, 150 and 180 min after consumption. At 3 h after the consumption of breakfast or no breakfast, participants consumed a standardised lunch and a 3 h postprandial venous blood collection commenced after lunch at 30, 60, 90, 120, 150 and 180 min. This enabled the measurements of 6 h plasma profiles of insulin and NEFA. Participants were also given

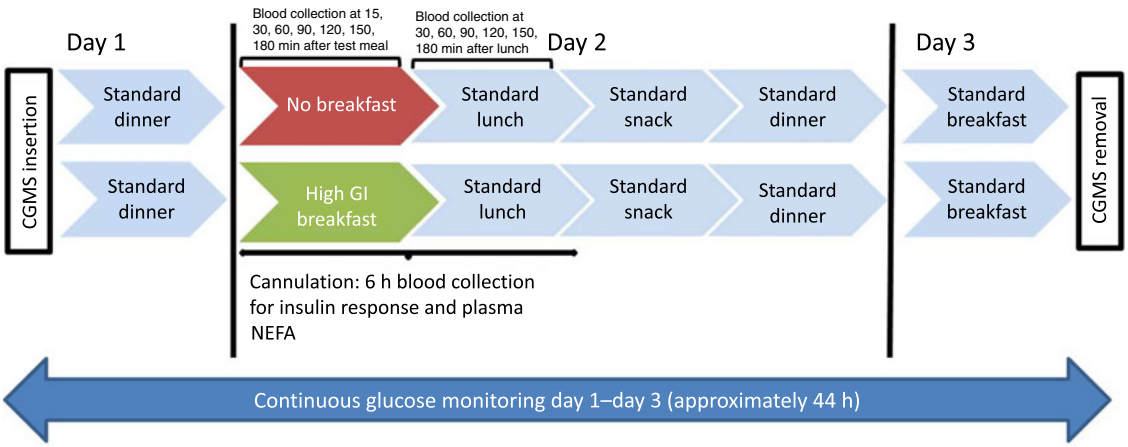

Fig. 1. Schematic representation of study protocol. CGMS, continuous glucose monitoring system; GI, glycaemic index.

Table 1. Nutritional composition of test foods and standardised meals

Food	Portion	Energy (kcal*)	Carbohydrate (g)	Sugars (g)	Fibre (g)	Protein (g)	Total fat (g)
Test breakfast							
New Moon Premium Glutinous Rice	100·6 g	356·1	811·4		0·2	7·24	0·2
Standardised breakfast							
Vitasoy Soya Bean Packet Drink	One packet (250 ml)	104	16	15·8	0	3·8	2·8
Jacob's Wheat Crackers – Weetameal	One packet (30 g)	142	20·7	4·2	1	2·2	5·6
Standardised lunch							
Spaghetti with Chicken Sauce (CP)	One packet (320 g)	352	52·2		2·6	17·3	8·3
CP Shrimp Wonton (bowl)	One packet (145 g)	132	18	2·8	2·8	5·6	2·1
Naspac Pudding with Nata De Coco Cup-Mango	One packet (108 g)	85	19			0	0
Vitasoy Soya Bean Packet Drink	One packet (250 ml)	104	16	15·8	0	3·8	2·8
Standardised snack							
FairPrice Roasted Cashew Nuts	One packet (40 g)	251	10·9	2·2	1·4	7·3	19·7
Ribena Blackcurrant Fruit Packet Drink – Regular	One packet (200 ml)	86	21·2	21		0	0
Standardised dinner							
Teriyaki chicken with rice (CP)	One packet (320 g)	510	85		0	19	10
Milo Chocolate Malt UHT Packet Drink	One packet (200 ml)	124	19		1	3·4	3·4
Naspac Pudding with Nata De Coco Cup-Mango	One packet (108 g)	85	19			0	0

* To convert kcal to kJ, multiply by 4·184.

a standardised snack to be consumed after 6 h and a standardised dinner to be consumed at 19.30 hours. They were also given a standardised breakfast to be consumed the next day at 08.00 hours. On day 3 of each test session, participants consumed the standardised breakfast at 08.00 hours and arrived at the centre at 12.30 hours for the removal of the continuous glucose monitoring system. A schematic representation of study flow is presented in Fig. 1. The study session terminated after the removal of the continuous glucose monitoring system, and they repeated the second study session (with 3 d washout period). The participants were encouraged to minimise physical activity during the whole test period of 3 d as physical activity was reported to result in changes in both glucose and lipid metabolism[27].

The test breakfast consisted of 100·6 g of high-GI rice (New Moon Premium glutinous rice, GI: 92, Diabetic Specialties Pte Ltd). The high-GI rice was cooked in 250 ml of water, with 2 g of chicken stock (Knorr Chicken Stock). Rice was cooked for 10 min in an electric rice cooker. Lunch, snacks and dinner were standardised and provided by the researchers. The nutritional composition of the test breakfast and standardised meals provided is shown in Table 1.

Continuous glucose measurement

The continuous glucose monitoring system provides continuous real-time glucose readings (at every 5 min interval) based on the electrochemical detection of glucose in the subcutaneous interstitial fluid. It is a commonly used method in clinical practices for glucose management in pre-diabetic and diabetic patients[29,30]. For each test session, the continuous glucose monitoring sensor was inserted in the side of the abdominal area of the body on day 1 and removed on day 3 of the study by a trained professional in the research centre. The continuous glucose monitoring system records and stores glucose readings

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at every 5 min interval when worn by the participants. The sensor was calibrated four times per d (before breakfast, before lunch, before dinner and before going to bed) using the OneTouch® Ultra®2 blood glucose meter (LifeScan, Inc.) during the whole intervention period by the subjects. This was done using finger prick capillary blood glucose. The continuous glucose monitoring sensor was worn unobtrusively from day 1 to day 3 of the study, and the data were collated and processed using an online software (Medtronic Diabetes CareLink iPro; https://carelink.minimed.eu). As the study aims to investigate the relationship between breakfast consumption and glucose homeostasis afterwards, only data obtained from day 2 at the start of the test meal were used for a 24- and 27-h analysis.

Blood analysis

To determine insulin and NEFA responses to test meals, venous blood samples were collected at fixed intervals in Vacutainers® (Belton Dickinson Diagnostics) with disodium EDTA, centrifuged at 1500 ***g*** for 10 min at 4°C (Sorvall™ ST 16 Centrifuge, Thermo Fisher Scientific), and plasma was then aliquoted into Eppendorf tubes and stored at −80°C until analysis. Plasma insulin was determined on the immunoassay chemistry analyzer Cobas e411 (Roche, Hitachi), which has an intra-assay CV < 5 % and inter-assay CV < 6 %. Plasma NEFA concentrations were measured using commercial enzymatic colorimetric assay kits (LabAssay™ NEFA, Wako Pure Chemical Industries), with intra- and inter-assay CV of <0·8 and <5 %, respectively. All CV of assays were provided by the manufacturers.

Statistical analyses

All statistical analyses were performed using the Statistical Package for Social Sciences software (IBM SPSS version 23.0; IBM Corp.), and statistical significance was set at $\alpha = 0{\cdot}05$, two-tailed. Participants' baseline characteristics and the outcomes of test sessions are reported as mean values and standard deviations unless otherwise stated.

Prior to statistical analysis, the normality of the data was confirmed using the Shapiro–Wilk test. No data transformation was performed before the non-parametric analysis. The data obtained were expressed as total AUC (tAUC) using the trapezoidal rule for postprandial glucose, insulin and NEFA response. Temporal changes in postprandial glucose, insulin and NEFA response were tabulated as changes from baseline fasting values, and they were compared using the general linear model for repeated-measures ANOVA (GLM RMANOVA) with Bonferroni correction. The baseline fasting value for glucose was obtained based on the mean glucose readings over a 1 h (12 × 5 min intervals) period prior to breakfast consumption or no breakfast consumption using the iPro™2 continuous glucose monitoring system. Using these changes, the postprandial glucose, insulin and NEFA response was expressed as the incremental AUC (iAUC) calculated using the trapezoidal rule that ignored the area under the baseline[31].

The primary aim of the study was to investigate the effects of breakfast on the glyceamic response of the subsequent meal. Based on a previous study using similar study design, but on type 2 diabetic patients, incremental area under the glycaemic curve for the second meal following breakfast improved significantly with an effect size of 2·2[32]. We assumed the correlation between the repeated measures to be 0·5. To determine a difference with an effect size of 2·2 with 80 % power and an α of 0·05 in a cross-over design, the minimum sample size required is at least 4. In addition, based on the recommendations by the FAO/WHO, a minimum of ten subjects are required during the analysis of glycaemic and insulinaemic responses in humans to take into account the inter-individual variations. By taking into account the attrition rate of 20–30 %, a total sample size of 14 was used for the present study.

Results

Fourteen participants were recruited but one participant withdrawn as he was unable to commit to the study schedule. Hence, no data were available for subsequent analysis for that subject. Complete data were gathered for both breakfast consumption and no breakfast consumption groups for the remaining thirteen participants who completed the study. Fig. 2 shows the number of participants throughout the study starting from recruitment, and Table 2 summarises the baseline characteristics of the study participants. The fasting glucose 6·40 mmol/l ($P = 0{\cdot}917$), insulin 11·92 μU/ml ($P = 0{\cdot}125$) and NEFA 0·27 μU/ml ($P = 0{\cdot}161$) concentrations were not significantly different on the first test day for both treatments.

The average 24 h glucose profile between breakfast consumption and no breakfast consumption is graphically presented in Fig. 3(a) and the change in glucose profile is present in Fig. 3(b). Fig. 3(b) shows that participants who consumed breakfast had a significantly lower glucose peak following lunch compared with participants who did not consume breakfast ($P < 0{\cdot}001$). Although there was a higher peak in glucose response after the test breakfast for participants who consumed breakfast, this was compensated by a lower glucose response following the standard lunch.

The average tAUC and iAUC glucose for post-meal, 24 and 27 h are presented in Fig. 4(a) and (b), respectively. Using the Wilcoxon signed-rank test, it was determined that the tAUC and iAUC glucose values after breakfast consumption ($P < 0{\cdot}001$; $P = 0{\cdot}001$) and standardised lunch consumption ($P < 0{\cdot}001$; $P < 0{\cdot}001$) for participants who consumed breakfast were significantly different from those observed for participants who did not consume breakfast. Breakfast consumption led to a 62 % decrease in iAUC of the postprandial glucose response over 3 h after lunch consumption compared with no breakfast consumption. However, there was no significant differences in tAUC and iAUC values during standardised snack ($P = 0{\cdot}212$; $P = 0{\cdot}463$) and standardised dinner ($P = 0{\cdot}279$; $P = 0{\cdot}753$) between the two treatments. Similarly, the overall 24 and 27 h iAUC values measured from test breakfast were all not significantly different between the two treatments. These results suggest that breakfast consumption has no effect on attenuating glucose excursions later in the day, after the second meal.

The average 6 h insulin profile of breakfast consumption and no breakfast consumption group is graphically represented in

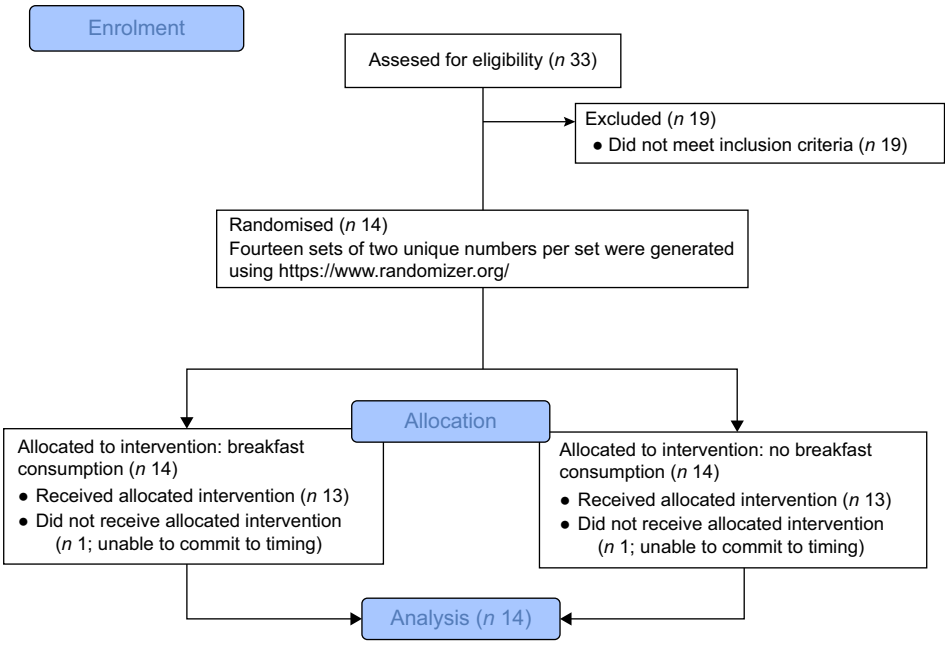

Fig. 2. Schematic flow diagram of number of participants at each recruitment process.

Table 2. Baseline characteristics of participants (*n* 13) (Mean values and standard deviations)

Anthropometric and physiological parameters	Mean	SD
Age (years)	54	2
Height (cm)	171·3	1·9
Weight (kg)	78·4	3·1
BMI (kg/m^2)	26·8	1·1
Fasting blood glucose (mmol/l)	6·40	0·55
Fasting plasma insulin level (μU/ml)	11·92	5·53
Fasting plasma lipid level (mmol/l)	0·66	0·22

Fig. 5(a), and the change in insulin profile is also presented in Fig. 5(b).

Similar to glucose response as observed in Fig. 3(b), as expected, breakfast consumption resulted in an increase in blood insulin concentration as compared with no breakfast consumption (Fig. 5(b)). Using the paired *t* test, it was found that the insulin response to the standardised lunch was more rapid for participants who consumed breakfast as compared with participants who did not consume breakfast, whereby the time in which the insulin level peaked was attained significantly ($P = 0{\cdot}025$) earlier for participants who consumed breakfast (at 210 min) than those who did not consume breakfast (at 270 min).

Using the Wilcoxon signed-rank test, it was found out that the tAUC and iAUC insulin values for post breakfast period ($P < 0{\cdot}001$; $P = 0{\cdot}001$) and overall 6 h post treatment ($P < 0{\cdot}001$; $P = 0{\cdot}001$) were significantly different between the two treatments, as observed from Fig. 6(a) and (b), respectively. However, only the iAUC insulin values for post standardised lunch period ($P < 0{\cdot}001$) were significantly different between the two treatments. Breakfast consumption led to a 59 %

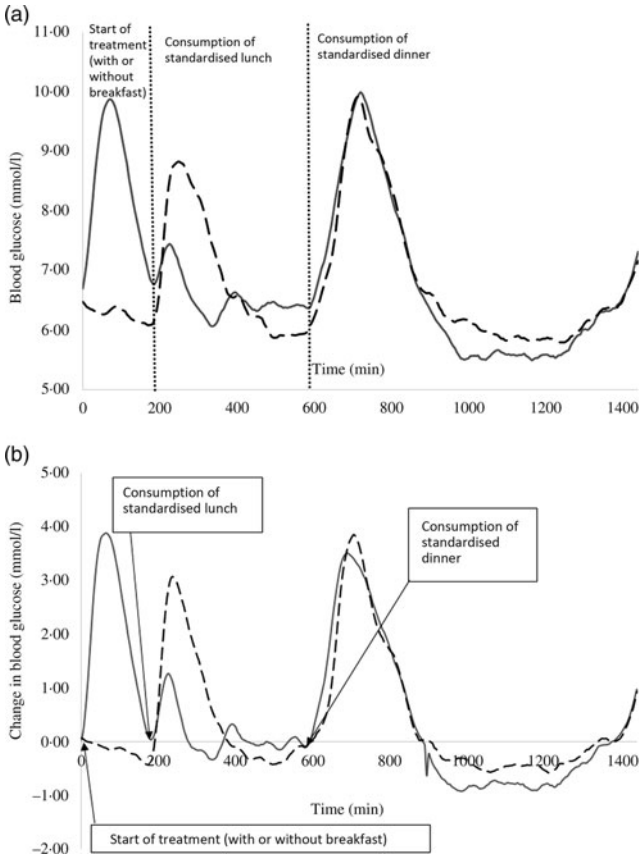

Fig. 3. (a) Mean 24 h glucose responses for breakfast consumption and no breakfast consumption treatment group (*n* 13). (b) Mean 24 h change in glucose responses for breakfast consumption and no breakfast consumption treatment group (*n* 13). ——, With breakfast; — –, without breakfast.

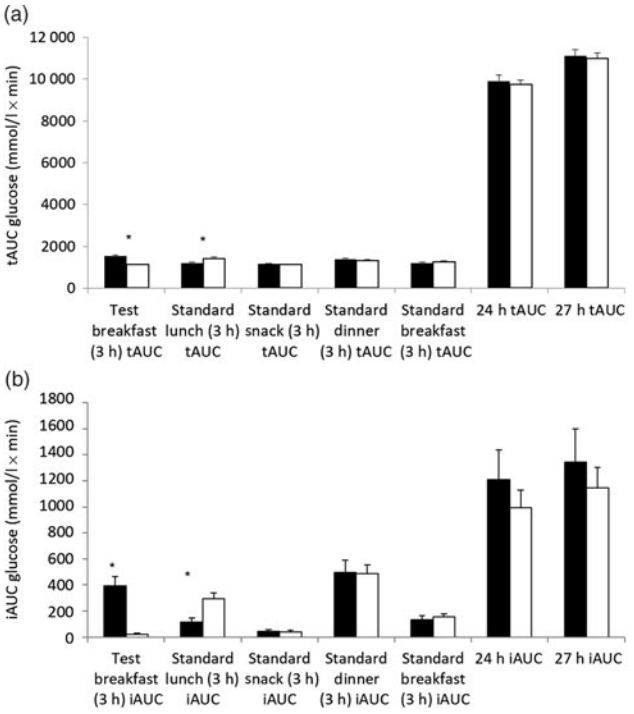

Fig. 4. (a) Total AUC (tAUC) values of glucose responses for the various treatments (n 13). (b) Incremental AUC (iAUC) values of glucose responses for the various treatments (n 13). Values are means, with standard deviations represented by vertical bars. * Significant difference between the two treatments ($P < 0{\cdot}05$). ■, With breakfast; □, without breakfast.

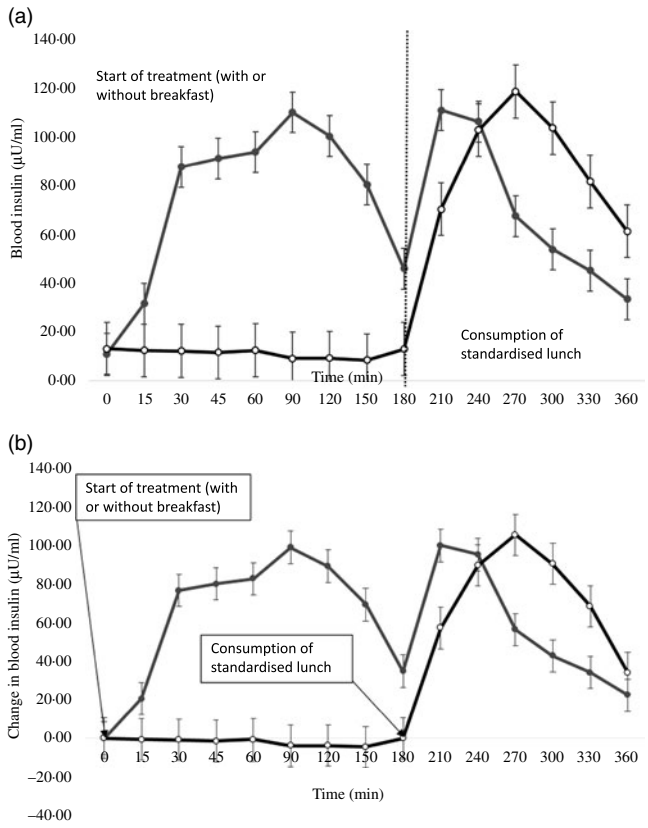

Fig. 5. (a) Blood insulin responses for breakfast consumption and no breakfast consumption treatment group for 6 h (n 13). (b) Change in blood insulin responses for breakfast consumption and no breakfast consumption treatment group for 6 h (n 13). Values are means, with standard deviations represented by vertical bars. —●—, With breakfast; —○—, without breakfast.

decrease in iAUC of the postprandial insulin response over 3 h after lunch consumption compared with no breakfast consumption.

The average 6 h NEFA profile and the changes in NEFA values for the two treatments are graphically represented in Fig. 7(a) and (b), respectively.

Using the Wilcoxon signed-rank test, it was found out that the negative tAUC and iAUC of NEFA (representing suppression of NEFA) during breakfast period ($P < 0{\cdot}001$; $P = 0{\cdot}001$) and during standardised lunch period ($P < 0{\cdot}001$; $P = 0{\cdot}001$) were significantly different between the two groups. NEFA suppression was significantly greater for participants who consumed breakfast compared with those who did not consume breakfast during the postprandial period following standardised lunch.

Discussion

The results from the present study showed how the consumption of breakfast, even a high-GI breakfast, serves as a pre-load to favourably attenuate postprandial glycaemic, insulinaemic and NEFA responses during subsequent lunch postprandial period, a phenomenon known as the 'second-meal effect'. Such a phenomenon was also observed consistently in earlier studies in both healthy participants[33,34] and people with type 2 diabetes[32]. Consuming foods that elicit the second-meal effect may help with the maintenance of low blood glucose concentrations in the short–medium term and thereby reduce demands on the insulin-mediated blood glucose regulatory systems. The importance of modulating the glucose response during the 'second-meal' effect has been widely recognised as an important precursor for improving glucose homeostasis, hence reducing the risk of type 2 diabetes[35,36]. The lowering of glycaemic excursion and glucose variability is now widely considered as the major treatment in diabetic patients[9,37].

One possible reason for the metabolic response differences between the two treatments could be that insulin release is influenced by β-cell memory from previous glucose exposure (e.g. the previous meal). Therefore, the absence in glucose elevation during no breakfast consumption may have decreased β-cell responsiveness and delayed the insulin release after lunch. This can be observed from Fig. 5(b) where it shows that the postprandial insulin peaks more rapidly after the standardised lunch period for participants who consumed breakfast as compared with those who did not consume breakfast. This thus suggests an improved β-cell responsiveness as well as better insulin action (sensitivity) during the lunch period (second-meal effect) for people who consumed breakfast as compared with those who do not consume breakfast.

This correlates with a lower iAUC insulin values during the post standardised lunch period for participants who consumed breakfast as compared with participants who did not consume breakfast (Fig. 6(b)). A greater response in β-cell during the

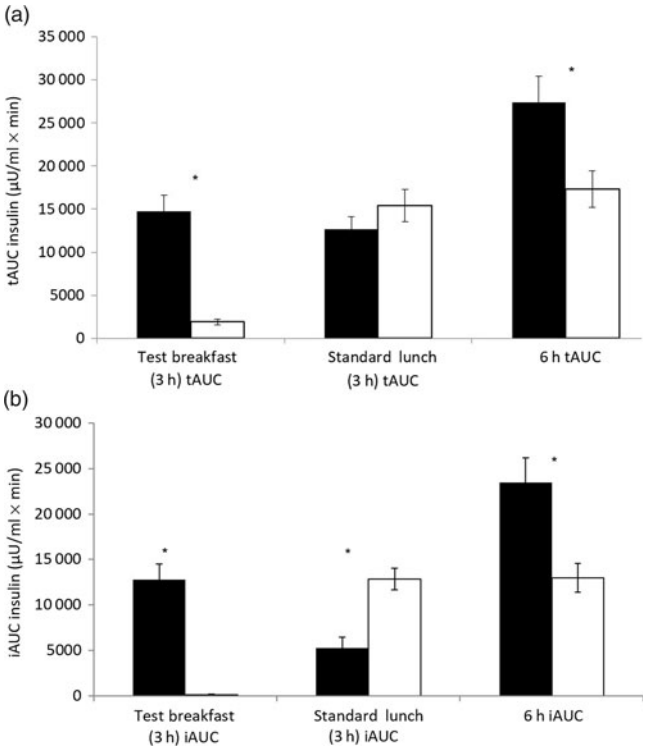

Fig. 6. (a) Total AUC (tAUC) values of insulin responses for the various treatments (*n* 13). (b) Incremental AUC (iAUC) values of insulin responses for the various treatments (*n* 13). Values are means, with standard deviations represented by vertical bars. * Significant difference between the two treatments ($P < 0{\cdot}05$). ■, With breakfast; □, without breakfast.

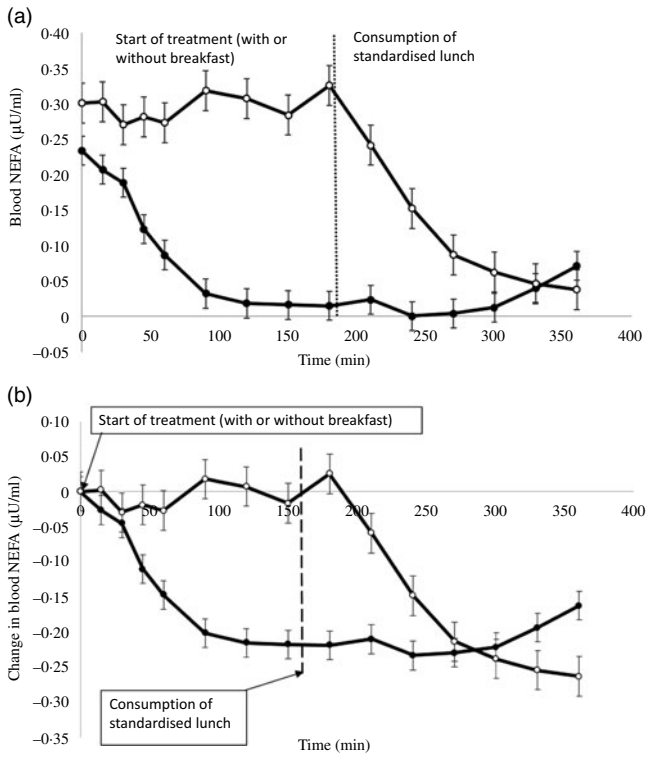

Fig. 7. (a) Blood NEFA responses for breakfast consumption and no breakfast consumption treatment group for 6 h (*n* 13). (b) Change in blood NEFA responses for breakfast consumption and no breakfast consumption treatment group for 6 h (*n* 13). Values are means, with standard deviations represented by vertical bars. —●—, With breakfast; —○—, without breakfast.

second meal was also observed in both diabetic[32] and nondiabetic healthy participants[33].

The suppression of NEFA concentration during the postprandial lunch period for participants who consumed breakfast (Fig. 7(a) and (b)) also seems to be associated with the second-meal effect. A reduction of NEFA was said to improve insulin secretion as prolonged elevation of NEFA level results in β-cell lipotoxicity, impairing insulin secretion[38,39]. Similar results were observed in healthy people where a suppression of NEFA concentration after the first meal improves insulin action, facilitating the second-meal effect[34,40]. Overall, these findings support the importance of breakfast, even a high-GI breakfast, for the improvement in glucose homeostasis.

In the present study, both the tAUC and iAUC values for the postprandial responses were calculated for glucose, insulin and NEFA. However, more emphasis is placed on iAUC comparison as the baseline values are different for tAUC; hence, the use of iAUC values better reflects physiological responses to the ingestion of test food in humans[41].

A significant strength and novelty of the study is its focus on a high-GI breakfast, which is commonly consumed in traditional Asian diets, and not done previously in pre-diabetic Asians to our knowledge. Another strength of the study is that it was conducted in controlled living conditions whereby all the meals were given to the participants and only male subjects of comparable age and BMI were recruited to minimise biological variability and also to reduce any confounders such as the menstrual cycle. However, one limitation in the present study is that food diaries from volunteers were not collected. Therefore, we were unable to determine whether volunteers ate more or less energy at subsequent meals after day 3 following the two treatments. Such analysis of food diaries could be done in future studies.

Conclusion

Breakfast consumption serves as a pre-load to attenuate both postprandial glycaemic and insulinaemic responses during subsequent lunch postprandial period (i.e. second-meal effect). Greater suppression of postprandial plasma NEFA was also observed during breakfast consumption, indicating an improved β-cell responsiveness as well as better insulin action (sensitivity). Given the importance of postprandial glycaemic and insulinaemic responses, our results indicate that the provision of breakfast resulted in an improvement in glucose homeostasis and this advocates for the regular consumption of breakfast.

Acknowledgements

The authors warmly thank the volunteers for taking the time to participate in the present study, and we would also like to acknowledge the assistance from research nurse, Susanna Lim.

The present study was funded by Singapore Institute for Clinical Sciences, grant no. H/17/01/a0/A11 and partially supported by General Mills Inc., grant no. GMI-783996.

All authors contributed to the study design, data analysis and interpretation, and the writing of the manuscript.

The authors declare no conflicts of interest.

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