Effect of a cereal and milk meal with or without fruits and nuts on the postprandial glycemic response in Indian men

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ABSTRACT

Background and Objectives: Some cereals, consumed at breakfast, have shown lower glycemic responses. Limited data exist in the Indian context, where the effect could be modified due to genetic or racial differences. This study aimed to investigate the effect of cereal and milk, with or without fruits/nuts, on the glycemic response in healthy Indian men. **Methods and Study Design:** A randomized cross-over study was carried out on 16 men (18 - 45 years), with 3 interventions providing equal amounts of glucose: a glucose drink (Reference), cereal and milk (CM), and cereal, milk, fruits and nuts (CMO), on separate days. Plasma glucose, serum insulin, C-peptide, ghrelin, energy expenditure (EE), substrate oxidation and appetite/satiety were measured repeatedly over 3 hours post meal. **Results:** A significant time effect and time x meal interaction between the meals, higher for the Reference meal, was observed for plasma glucose (p<0.001), insulin (p<0.001), C-peptide (p<0.001), and carbohydrate oxidation (p<0.001); while lower for satiety (p<0.001). The plasma glucose concentrations of CM and CMO meals returned to baseline 60 minutes postprandially, then remained there, unlike the Reference meal, where the plasma glucose values returned to baseline at 120 minutes and dipped significantly below baseline at 150 and 180 minutes. A significant effect of time (p<0.001) was observed for EE between meals. Ghrelin levels did not differ significantly between the test meals. **Conclusions:** Cereal with milk, along with fruits and nuts at breakfast, has a lower and stable glycemic response, along with increased satiety among healthy male subjects.

Key Words: glycemic response, cereal, breakfast, satiety, Indian men

INTRODUCTION

Breakfast, providing 20%-35% of daily energy requirement, has been considered to be the most important meal of the day.[1] Regular consumption of breakfast, particularly cereal-based breakfast, has been associated with lower body mass index (BMI), better cardio-metabolic profile[2,3] and reduction in the risk of type 2 diabetes by 15-20%.[3] Despite these health benefits, a busy working schedule, limited knowledge of food preparation, and taste preferences present some challenges for individuals to incorporate breakfast as an integral part of their daily diet.[4]

Cereals such as corn flakes and muesli,[5] consumed at breakfast along with other food groups like milk, fruits and nuts,[6] are becoming increasingly popular. Milk, which has a high protein to carbohydrate ratio and is a good source for calcium, can modify the glycemic

response to cereal and reduce post prandial glycemia.[7] Whole fruits, despite containing small amounts of free sugars, are good sources of micronutrients and dietary fibre, and also lower and flatten the glycemic response,[8] while nuts, have also been reported to blunt the post prandial glycemic response to carbohydrate meals.[9]

The South Asian phenotype is thought to be more susceptible to chronic hyperglycemia and hyperinsulinemia with a higher risk of insulin resistance and diabetes mellitus.[10] Since most of the cereal meal glycemic response studies have been conducted in Western subjects,[5,6,11,12] there is specifically a need to conduct these studies in South Asians, particularly Indians, as they exhibit a greater glycemic response to the same food when compared to Caucasians.[13] It is also important to examine the temporal nature of the glycemic response in these subjects, since a lower and persistent response offers potential benefits of a longer availability of substrate for energy production, as well as a more prolonged satiety. Therefore, this study aimed to investigate the temporal nature of the glycemic and insulinemic response of cereal (provided as cornflakes) and milk at breakfast, with or without added fruits and nuts, in a group of healthy Indian males, along with measurements of the postprandial substrate utilization and satiety.

MATERIALS AND METHODS

This was a randomized cross-over, open label study design with three arms. The study was conducted at St. John's Medical College, Bangalore, India, and volunteers were recruited from institutional student/staff population. Male subjects aged between 18 to 45 years were recruited based on inclusion criteria of weight stability for 3 months, a BMI range of 18.5 to 24.9 kg/m^2, normal fasting (80-100 mg/dL) and 2 hour post prandial blood glucose (<140 mg/dL) assessed by two sample test Oral Glucose Tolerance Test (OGTT), normal renal and liver function tests, Physical Activity Level (PAL) less than 2.0. Exclusion criteria were insulin resistance assessed by homeostatic model assessment of insulin resistance (HOMA-IR),[14] nut allergies or lactose intolerance, Hemoglobin <13 mg/dL, history of alcohol or drug abuse and a significant clinical history. Following screening, the subjects received the one of the three meals – a glucose drink (Reference), cereal and milk (CM), and cereal, milk, fruits and nuts (CMO), in random order on separate experiment days. Since all the subjects received all the treatments once, the allocation ratio was 1:1:1. In order to compare the two cereal meals (CM and CMO) with the Reference, a sample of 14 subjects was required to detect a 20% difference in glycemic response with 80% power and 2.5% level of significance.[15] The study was approved by Institutional Ethical Review Board (approval number 238/2017), and

informed consent was obtained from each participant. The study was registered in the Clinical Trials Registry of India (CTRI Ref no: REF/2017/09/015457).

Subjects reported to the clinical research facility at 6:30 am after an overnight fast of minimum 10 hours. They were asked to consume a usual portion sized meal of their choice and repeat similar meals before the remaining two trials. In addition to this, two days prior to each experiment subject was asked not to smoke, drink alcohol and exercise vigorously. At the beginning of each trial, a basal urine sample was collected, followed by weight and height measurement using standard methodology.[16] Body weight was measured to the nearest 0.1 kg using a calibrated electronic weighing balance (Goldtech, AE038, New Delhi, India). Height was measured using a stadiometer (Seca 213, Hamburg, Germany) to the nearest 0.1 cm. Waist circumference was measured at the narrowest part between the last rib & the iliac crest.[16] Hip circumference was measured with non-elastic tape placed around the buttocks in a horizontal plane at the level of maximum extension of the buttocks.16 Body fat was measured using bio-impedance analysis (BIA, Bodystat Quadscan 4000, Isle of Man, British Isles).[17]

After the collection of basal blood sample, one of the three test meals (Table 1) was given to the subject for ingestion within 15 min (5 min for Reference and 10-15 min for cereal meals). A Reference meal (50 g glucose dissolved in 200 mL water) was used for comparison with the other two cereal meals. The cereal used in the test meals was cornflakes. The first cereal meal (CM) contained only cereal (43 g) and milk (287 mL), while the second (CMO) contained cereal (35 g), milk (230 mL), fruit (Apple-66g) and nuts (Almonds-10g). Toned milk with 3.1% fat and moderately ripened apples were used across the trials. Each of the meals provided 50 g of glycemic (available) carbohydrate. Blood samples (3 mL) were then collected at 15, 30, 45, 60, 90, 120, 150 and 180 min from indwelling line, to measure plasma glucose, serum insulin and C-peptide, while serum ghrelin was measured up to 120 min. Plasma glucose was analysed using the Hexokinase method[18] while serum insulin and C-peptide were analyzed using Chemiluminescent Immunometric assays (IMMULITE 1000, Tarrytown, USA).[19] The intra-assay coefficient of variation (CV) was < 2% (±1.0) for glucose, < 2.2% (±0.1) for insulin and <2.6% (±0.5) for C-peptide. Serum ghrelin levels were measured by ELISA (Human GHRL Elisa Kits, Elabscience, Houston, USA) method20 with an intra-assay CV of 1.74% (±0.02). A baseline resting energy expenditure (REE, for 20 min) and post-prandial energy expenditure (EE) measurement was made at 30, 60, 90, 120, 150 and 180 min for 10 min at each time point, by indirect calorimetry with a accuracy of <2% (measured by ethanol burns). The intra-individual CV of REE was 5.4% (±0.05). Substrate oxidation rates[21] were calculated using the respiratory quotient (RQ) corrected for urinary N2

excretion (measured by Kjeldahl analysis) over the period of the postprandial measurement.[22] Visual analogue scales (VAS) were used to assess the satiety and appetite levels of the subjects to the test meals, using a scale of 100 mm,[23] to test for 'hunger', 'thoughts of food', 'urge to eat', and 'fullness of stomach'. The VAS was administered at baseline and 10, 40, 70, 100, 130, 160, 190 min after the meal.

Data are presented as mean and standard deviation (SD) or standard error of the mean (SE, in Figures) for normally distributed continuous variables. The normality of data was checked using Q-Q plot. Serum insulin and C-peptide concentrations were log transformed since they were not normally distributed. Plasma glucose, serum insulin and C-peptide responses were first calculated as the incremental area under the curve (IAUC). Next, a repeated measures (RM) ANOVA with two factors (time and type of meal) was used to compare outcomes over time by meals with interaction effects, and comparisons at specific time points were made with Bonferroni corrections for actual values and total area under the curve (TAUC) adjusted for baseline. All analyses were carried out using SPSS version 22 (Chicago, Illinois), and the significance level was set at $p<0.05$.

RESULTS

The participant flow is summarized in the Figure 1. Screening was performed on 26 volunteers, out of which 8 were excluded, since they did not meet the inclusion criteria. A total of 18 subjects were recruited in the study and 16 of them completed all the three trials successfully. Out of the two drop outs, one subject did not report back after screening. The other subject started a trial, but did not like the cereal meal and could not finish the entire meal portion, therefore dropped out of the study immediately. Since, there is no biochemical or qualitative data for these two subjects, the final analysis was performed for 16 subjects. The mean BMI of the study participants was 23.1 kg/m^2 and 88% of them were between 18-30 years. The descriptive characteristics are summarized in Table 2.

Postprandial mean plasma glucose levels increased with all meals, rising to a peak value at 30 min, and returning to baseline by 60 min for the CM and CMO meals, and one hour later, at 120 min, for the Reference meal. Subsequently, the mean plasma glucose level remained at the baseline for CM and CMO meals, while for the Reference meal, it dipped significantly ($p<0.05$) below baseline values at 150 min and remained there until the end of the study. When TAUC adjusted for baseline was considered, the dip for Reference meal below baseline was significant ($p<0.05$) at 180 min. In spite of this late dip for the Reference meal, the IAUC for plasma glucose was significantly lower ($p<0.001$) in cereal meals when compared to the

Reference (CM and CMO response were lower by 53% and 52% respectively). When analyzed by time and meal, there was a significant time effect and time x meal interaction effect (p<0.001) between the test meals (Reference, CM and CMO). Post hoc tests showed that the CM meal had a significantly lower glycemic response at 30, 45, 60 and 180 min in comparison to the Reference meal, while there was slight difference in this pattern for CMO, where the significant difference was observed at 30, 45 and 150 min when compared to Reference (Figure 2a). There was no significant difference between CM and CMO meals. TAUC adjusted for baseline also confirmed these time specific significant differences (p<0.05) observed for cereal meals (CM and CMO) as compared to Reference, except at 30 min for CMO.

Postprandial mean serum insulin and C-peptide concentrations increased significantly (p<0.05) with all the test meals and returned to baseline values at 180 min for CM and Reference, while for CMO it remained significantly higher (p<0.05) even at the end of the study. A significant time effect and time x meal interaction effect (p<0.001) was noted for all the three meals. However, post hoc tests could not identify the specific time points at which the mean serum insulin or C-peptide differed between the meals (Figure 2b and 2c). In addition, a significantly lower IAUC (p<0.001) was observed for C-peptide, with 31% and 29% lower values for CM and CMO when compared to Reference. However, for insulin, the IUAC difference was lower in cereal meals but could not achieve significance as compared to Reference (CM-17% and CMO-21%). There was no significant difference between CM and CMO meals for either serum insulin or C-peptide.

A significant time effect (p<0.001) was observed in RQ and EE for all test meals, but no time x meal interaction (Table 3). The mean carbohydrate oxidation rates at each time point are presented in Table 3. When calculated as cumulative values at each time point after the meal, these values showed a significant effect of time, as well as a time x meal interaction (p<0.001) with the reference meal having the highest carbohydrate oxidation rate, however, post hoc analysis could not identify the specific time points at which the outcome differed by meal. At the end of the experiment, at 180 min, the average cumulative carbohydrate oxidation was 47.2±9.3, 46.3±9.8 and 52.3±8.5 g for the CM, CMO and Reference meals respectively. The higher cumulative carbohydrate oxidation with the Reference meal was due to its higher values from 90 to 180 min (Table 3). The mean fat oxidation rates at each time point are also presented in Table 3. For the cumulative fat oxidation, there was a significant time effect and time x meal interaction effect (p<0.001), however, post hoc tests could not identify the time at which outcome differed by meal. At the end of the study, the average

cumulative fat oxidation was 0.54±0.43, 0.54±0.32 and 0.24±0.30 g for CM, CMO and Reference meals respectively. There was no significant difference between the CM and CMO meals for RQ, EE, carbohydrate and fat oxidation.

A significant effect of time and time x meal interaction was observed for 3 of the VAS parameters i.e. thoughts of food (p<0.05), hunger (p<0.001) and fullness (p<0.05), however, post-hoc tests could not identify the time points at which these outcomes differed by meal. In contrast, for urge to eat, there was significant time effect (p<0.001) but no significant time x meal interaction. Ghrelin, which is a biomarker of hunger, did not show any significant time effect or time x meal interaction. There were no significant differences between the CM and CMO meals for any of the VAS parameters and ghrelin.

DISCUSSION

This study aimed to assess the glycemic response to a cereal and milk meal, with or without added fruits and nuts, against a Reference meal of glucose. The lower postprandial excursion of plasma glucose for cereal meals was supported by an earlier systematic review on similar cereal meals, which also reported their postprandial plasma glucose lowering effect,[5] however the earlier return to baseline with the cereal meals and the dip below baseline for the Reference meal, is of interest.

The observed lower glycemic response for the cereal meals could be attributed to partially digestible starch or complex carbohydrate, formed during the processing of the test cereal. This can be substantial, as a previous study reported a 40% lower glycemic response for partially digestible starch in comparison to a completely digestible starch.[24] The other factor is the gastric emptying time, since it is influenced by several meal based factors such as physical state of the meal (solid/liquid), portion size, caloric density, osmolality and nutrient composition.[25] The co-ingestion of protein and fat along with carbohydrate also reportedly delays gastric emptying leading to a blunted glycemic response.[9,26] Addition of milk, which has high protein to carbohydrate ratio (1.0:1.3), could also have suppressed the glycemic response in both CM and CMO.7 Furthermore, a complex interdependent relationship exists between gastric emptying, the incretin axis and postprandial glycemia.[27] Delayed gastric emptying slows the nutrient absorption from the small intestine with subsequent release of incretins, glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic peptide (GIP), which stimulates pancreatic insulin release, and attenuates postprandial hyperglycemia.[27] A greater volume (Reference – 250 g, CM – 330 g, CMO – 340 g) and

higher calorie density (Reference - 0.8 kcal/g, while that of the CM and CMO - 1 kcal/g) of the cereal meals, may also have had an additive effect.[28]

The end of the glycemic response to the meals was judged by a disappearance of significantly different postprandial plasma glucose values in comparison to the baseline. Thus, in CM and CMO meals, plasma glucose returned to its baseline concentration at 60 minutes post-meal and remained there until 180 min. In contrast, the plasma glucose concentration of Reference meal rose higher than the cereal meals, returned to baseline at 120 min and then dipped below baseline at 150 and 180 min. This pattern in reference meal can be explained by inspecting the carbohydrate oxidation rates, which remained higher in the second half of the study, perhaps due to the higher insulin response along with higher hunger and other VAS parameters. An additional factor to consider is that after 150 min, both cereal meals effectively had a higher glycemic response in comparison to the Reference meal, even though their plasma glucose concentrations were not significantly different from the baseline. This could be due to a combination of effects arising from a continuing absorption of glucose from the cereal meals, as well as a lower serum insulin response and therefore lower carbohydrate disposal rates.[29] The latter effect could partly be due to a lower total glucose concentration per unit mass of the meal[30] (Reference meal: 0.2 g/g, CM and CMO meal: 0.15 g/g), compounded by slower gastric emptying. Of interest is that the mean cumulative carbohydrate oxidation over 180 min was about 50 g in all meals, which corresponded to the amount of carbohydrate provided in the meals. Given that the serum insulin concentrations remained elevated above baseline for the entire postprandial period, it is unlikely that the oxidized carbohydrate could have come from endogenous hepatic glucose production.[31]

All three meals (Reference, CM and CMO) showed the peak glycemic excursion within 30 min post-meal. Such pattern for the cereal meals could be explained by the evidence of greater number of copies of the salivary amylase gene (AMY1), and a higher secretion of salivary amylase in South Asians, followed by faster digestion of ingested starch.[32] Indian subjects, habituated to high starch diets, could also have higher AMY1 copy numbers, but this is not confirmed to our knowledge.

The addition of fruit and nut to the cereal and milk meal did not make a difference to the glycemic and other outcomes measured. Probably the usual consumption amount of fruit and nut along with a cereal meal was not sufficient enough to show a lower glycemic response. Studies have reported a flattened glycemic response only after the inclusion of 30 grams of fat (from almonds)[9] and 10 grams of dietary fibre;[33] which was 5 and 7 fold higher than the amount provided in the present study. A significant attenuation in the glycemic response to a

typical cereal meal has been observed when half of the test carbohydrate was replaced with fructose[34] which was more than 3 times of the amount provided (from apple) in the present study. Equally, it is possible that the addition of fruits like cherry, grapefruit, orange, peach, pear, or watermelon with a lower glycemic load could be better options for lowering post-prandial glycemic response.[35] A varying protein and fat content in different fruits and nuts could also exert an altered glycemic and insulinemic response.[36]

A higher satiety level, as assessed by the VAS, was observed for both cereal meals, 3 hours post-meal. Satiety is primarily dependent on composition and volume of ingested meals. A higher meal volume causes gastrointestinal distension and vagal stimulation resulting in anorexic signaling to the arcuate nucleus of hypothalamus, thereby reducing food intake.[37] Additionally, fat,[37] protein[38] and calcium[39] content of the test meals could also have suppressed the hunger sensation. Ghrelin, an appetite stimulator, reflects degree of hunger.[40] Similar studies with cereal-based breakfast have shown suppressed serum ghrelin concentration,[41-43] which in turn reduces food intake later in the day.[43] However, in the present study, no significant difference observed in postprandial ghrelin concentration for the cereal meals as compared to Reference, could be attributed to the high inter-individual variability (41%), comparable to the findings (10 - 47%) of previous studies.[38,42,43] Moreover, sample size of the present study may not have been sufficient to observe significant differences in serum ghrelin between the meals.

One limitation of this study was that it was conducted only in males; it is known that females could have a different glycemic response pattern.[44] The measurement of GLP-1, GIP and glucagon concentrations could have clarified mechanisms involved in the observed glycemic, insulinemic and satiation responses. The role of cereal alone, on the glycemic response could not be evaluated, since the study did not have cereal as an independent group. The VAS questionnaire used in the present study was not validated for Indians. The body fat mass of the study participants was measured using BIA and could have been underestimated.[45] The strengths of this study lie in the strict control on recruitment, additional measurements of EE and hormones, and randomly paired observations of the study.

Conclusion

The present study suggests that breakfast time cereal consumption with milk and added fruits and nuts, results in a lower and stable glycemic response, and improves satiety among healthy male subjects. Further studies are needed to understand the underlying mechanisms, which could enable targeted lifestyle recommendations for glycemic control.

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CONFLICT OF INTEREST AND FUNDING DISCLOSURE

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Table 1. Nutrient composition of the interventions provided in the study

Ingredients	Amount (g)	Energy (kcal)	Available carbohydrate (g)	Protein (g)	Fat (g)	Fibre (g)
Reference	50	200.0	50.0	-	-	-
Dextrose	50	200.0	50.0	-	-	-
CM		335.7	50.1	12.3	9.3	1.1
Cornflakes†	43		36.29	2.86	0.43	1.1
Milk	287		13.78	9.47	8.9	-
CMO		378.4	50.1	12.0	13.7	3.6
Cornflakes†	35		29.54	2.33	0.35	0.9
Milk	230		11.04	7.59	7.13	-
Apple§	66		9.21	0.20	0.35	1.4
Almonds§	10		0.30	1.84	5.85	1.3

Reference: Glucose drink; CM: Cereal and Milk; CMO: Cereal, Milk, Fruits and Nuts.
†Calculated as per the nutritive values provided by the manufacturer.
‡Calculated as per the nutrition information given on the packet of milk.
§Calculated using Indian Food Composition Tables (IFCT), 2017.

Table 2. Baseline characteristics of the study population (n=16)

Variable	Mean±SD
Age (y)	24.7±5.4
Anthropometric measurements	
Weight (kg)	65.1±5.2
Height (cm)	168±5.3
BMI (kg/m^2)	23.1±2.1
Waist circumference (cm)	81.8±6.3
Hip circumference (cm)	96.4±3.4
MUAC (mm)	28.8±2.6
BF% from BIA	18.2±3.4
Physical activity pattern	
PAL	1.65±0.22
TEE (kcal/d)	2759.3±366.2
BMR (kcal/d)	1674.3±64.5
Habitual dietary intake†	
Energy (kcal/d)	2144.9±984.2
Protein (g/d)	74.8±41.7
Fat (g/d)	65.0±36.3
Carbohydrate (g/d)	316.4±129.8
Dietary fibre (g/d)	9.3±4.2

BMI: Body Mass Index; MUAC: Mid-Upper Arm Circumference; BF%: Body Fat Percentage; BIA: Bio-electrical Impedance. Analysis; PAL: Physical Activity Level; TEE: Total Energy Expenditure; BMR: Basal Metabolic Rate.
†Assessed by triple pass 24 hour dietary recall.

Table 3. Respiratory Quotient (RQ), Energy Expenditure (EE; kcal/min), carbohydrate and fat oxidation rates (g/30 min) across the time points for interventions (n=16)

Group	Time points							*p* value time effect	*p* value interaction effect
	Basal	30 min	60 min	90 min	120 min	150 min	180 min		
Respiratory Quotient									
Reference	0.90±0.05	0.93±0.04	0.93±0.04	0.94±0.04	0.93±0.04	0.92±0.04	0.93±0.05		
CM	0.89±0.05	0.90±0.04	0.92±0.03	0.89±0.03*	0.89±0.04	0.90±0.04	0.91±0.04	<0.001	0.195
CMO	0.88±0.05	0.91±0.03	0.91±0.05	0.90±0.04	0.90±0.04	0.90±0.03	0.89±0.04		
Energy Expenditure (kcal/min)									
Reference	1.06±0.15	1.21±0.16	1.21±0.14	1.91±0.15	1.14±0.15	1.14±0.11	1.17±0.14		
CM	1.07±0.13	1.27±0.11	1.27±0.10	1.23±0.11	1.21±0.10	1.18±0.09	1.20±0.11	<0.001	0.220
CMO	1.03±0.11	1.25±0.12	1.25±0.11	1.20±0.10	1.19±0.09	1.18±0.10	1.19±0.13		
Carbohydrate oxidation rates (g/30 min)									
Reference	5.04±1.49	7.87±1.80	8.06±1.61	8.21±1.79	7.76±2.02	7.43±2.42	7.92±2.00		
CM	5.18±1.65	7.23±2.08	7.97±1.73	6.64±1.46	6.61±2.19	6.49±1.69	7.06±1.87	<0.001	0.225
CMO	5.00±2.07	7.62±1.47	7.54±2.14	6.84±2.07	6.57±2.05	6.46±1.14	6.31±1.89		
Fat oxidation rates (g/30 min)									
Reference	0.10±0.05	0.03±0.06	0.02±0.06	0.01±0.06	0.02±0.07	0.03±0.09	0.02±0.07		
CM	0.10±0.07	0.08±0.08	0.05±0.08	0.09±0.06	0.08±0.09	0.08±0.07	0.06±0.08	<0.001	0.031
CMO	0.10±0.07	0.06±0.04	0.06±0.07	0.07±0.07	0.08±0.07	0.08±0.05	0.09±0.06		

Data presented as Mean±SD; Reference: Glucose Drink; CM: Cereal, Milk; CMO: Cereal, Milk, Fruits and Nuts.
*Significant interaction effect ($p<0.05$).

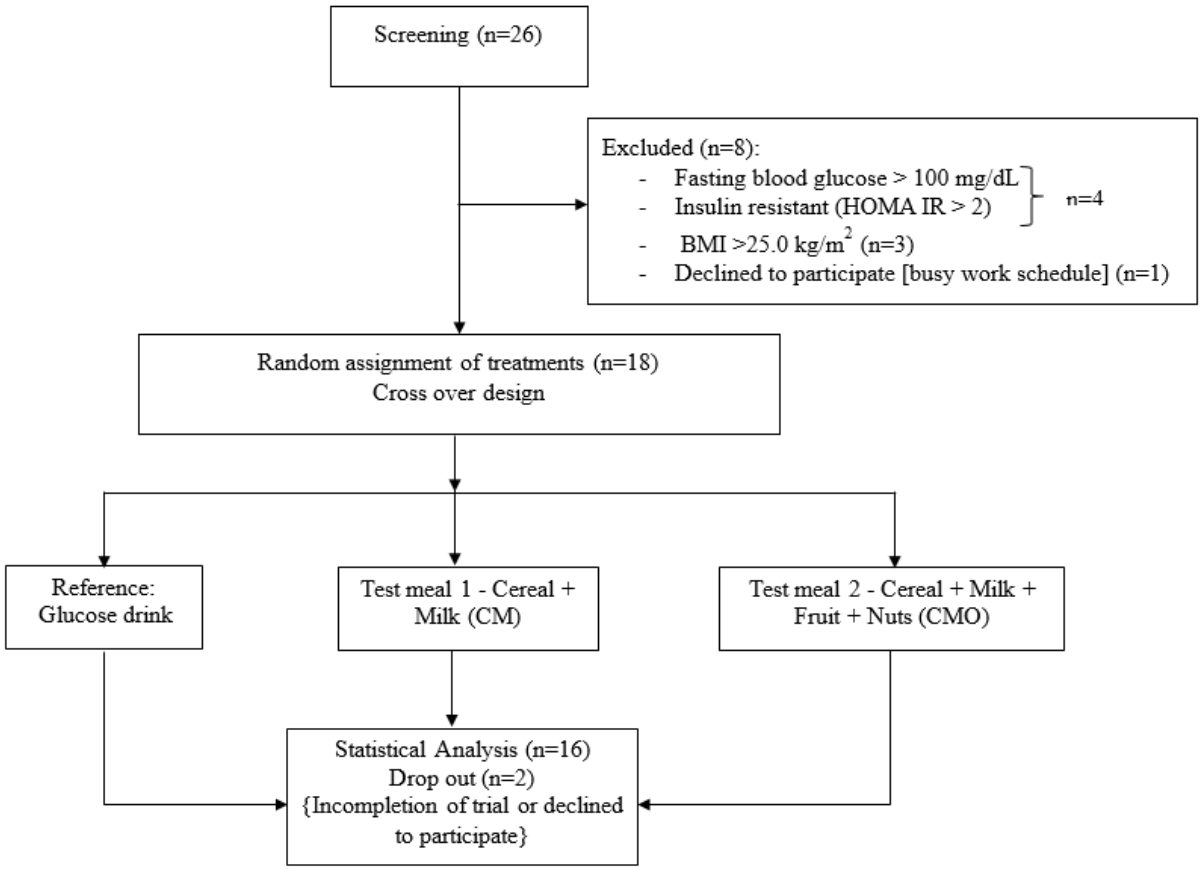

Figure 1. Participant flow diagram.

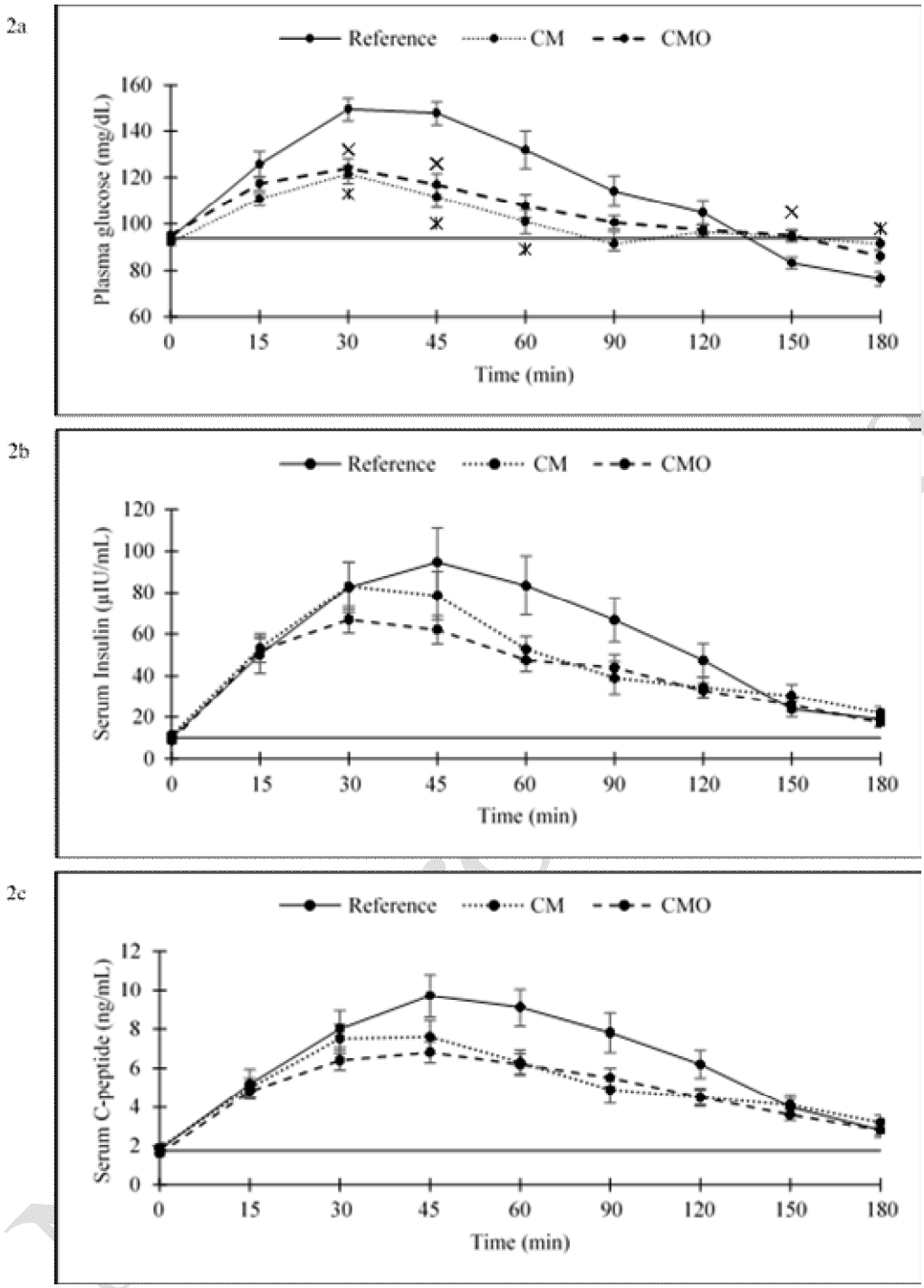

Figure 2. Blood parameters across the time points for interventions (a) Plasma glucose values (mg/dL); (b) Serum Insulin values (µIU/mL); (c) Serum C-peptide values (ng/mL); (n=16). Data presented as Mean ± SE. Reference: Glucose Drink; CM: Cereal, Milk; CMO: Cereal, Milk, Fruits and Nuts. Shows significant interaction effect between CM and Reference. Shows significant interaction effect between CMO and Reference. In each plot, the average baseline values for all meals have been extended as a horizontal line.

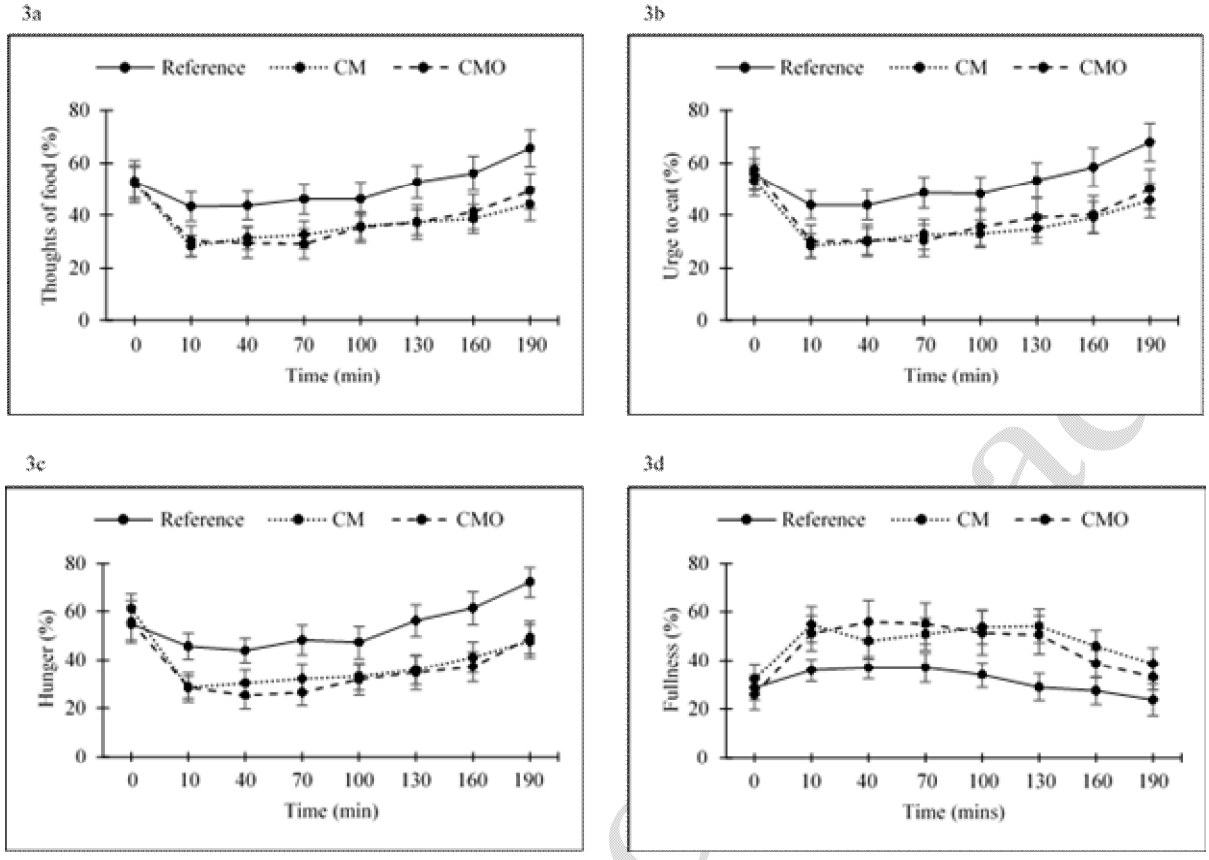

Figure 3. Visual Analogue Scale (VAS) scores (%) across the time points of appetite assessment for interventions (a) Thoughts of food (b) Urge to eat (c) Hunger (d) Fullness (n=16). Data presented as Mean ± SE. Reference: Glucose Drink; CM: Cereal, Milk; CMO: Cereal, Milk, Fruits and Nuts.