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Consumption of decaffeinated coffee with milk and sugar added before a high-glycemic-index meal lowers postprandial glucose surge when compared with consuming it after the meal

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ABSTRACT

This study aimed to compare the effect of consuming decaffeinated coffee at different times around a high glycemic index (GI) meal on postprandial glycemic responses. Ten healthy adults completed this randomized, crossover trial. In each session, overnight-fasted participants consumed the test drink, which was a cup of decaffeinated coffee with low-fat milk and white sugar added, at different timepoints before or after a high GI meal. Postprandial levels of glucose, insulin, and nitrotyrosine at individual timepoints, as well as the area-under-curves (AUC), were compared among the treatments using mixed-effect linear model. Results showed that consuming the test drink 30 and 60 min before the high GI meal (glucose AUC ± SE: 862.6 ± 24.8 and 861.4 ± 24.8 mmol * 120 min/L, respectively) led to significantly reduced glycemic responses when compared with consuming it after the meal (952.7 ± 24.8 mmol * 120 min/L, $p < 0.05$ compared with either treatment). Drinking the test drink at least 30 min before the meal also led to smaller insulinemic responses, while nitrotyrosine levels were not significantly different between different consumption times. These showed that consuming decaffeinated coffee with milk and sugar added at least 30 min before a meal could benefit postprandial glycemic control.

1. Introduction

Reducing the surge of postprandial glucose levels has been recognized to improve insulin sensitivity [1] and reduce systemic oxidative stress [2]. Previous studies found that pre-meal consumption of carbohydrates [3], protein [4], and decaffeinated coffee with milk and sugar added [5] could reduce the postprandial glycemic response. Nonetheless, drinking black coffees and caffeinated coffees with milk and sugar added before a meal added did not lead to remarkable differences in postprandial glycemic responses when compared with drinking water [5].

It is important to note that people drink coffee throughout the day, which could be before, after, or between main meals [6,7]. Findings from previous studies implied that the effect of drinking decaffeinated coffee at different times around meal on postprandial glycemic excursions could be different – Moisey et al. [8] found that decaffeinated coffee could benefit postprandial glycemic and insulinemic excursions when consumed three hours before a meal, while no beneficial effect was observed in a separate study when it was consumed together with the main meal [9]. Nonetheless, a direct comparison of pre-meal and post-meal consumption of decaffeinated coffee has not been made before.

Therefore, the objective of this study is to compare the effect of the different timing of the consumption of decaffeinated coffee with milk and sugar added around a high-glycemic-index (GI) meal on postprandial glucose, insulin, and nitrotyrosine levels in a group of healthy Asian adults.

Abbreviations: AUC, area under curve; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance.

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2. Materials and methods

2.1. Study design

This randomized, single-blind, crossover acute feeding study was conducted between June 2018 and April 2019 at the University of Hong Kong (HKU). Ethics approval was obtained from the Human Research Ethics Committee of the University of Hong Kong (Approval no.: EA1712012). The study was conducted according to the guidelines laid down in the Declaration of Helsinki. This trial was registered at anzctr.org.au (ID: ACTRN12618000645257). All participants provided written consent before the commencement of the study.

2.2. Participant recruitment

Participants were recruited from the staff and students of HKU through emails and face-to-face recruitment. The inclusion criteria were aged from 18 to 30 years old, with a body mass index (BMI) between 18.0 and 23.0 kg/m^2, consumed at least one cup of coffee per day in the past three months, have never smoked before, able to tolerate cow's milk and coffee with an empty stomach, and not on any regular medication (except oral contraceptives). All participants were enrolled by the first author and provided written consent before the commencement of the experiment.

2.3. Study protocol

Two days before each experimental session, participants were required to refrain from consuming alcohol, caffeine-containing food and drinks, and were advised not to take part in any vigorous physical activity. Participants were instructed to consume a standard dinner package (spaghetti with bacon and mushroom in cream sauce) on the day before the session, refrain from consuming any food and drink after dinner, fast for at least 10 h, and sleep for at least seven hours before the experiment. Female participants were advised not to take part in experimental sessions within a week before the commencement of the menstrual period and during that to avoid variation in results due to hormonal fluctuation.

The procedures of the experiment are illustrated in Fig. 1. Participants arrived at the experiment venue with an empty stomach. In pre-meal sessions, participants gave the fasting blood sample after 15 min of resting, then they consumed the test drink, which was made by adding 2 g decaffeinated coffee granules (NESCAFÉ GOLD BLEND® decaffeinated coffee, Nestlé, Hong Kong) and 50 g low-fat cow's milk drink (Vitasoy, Hong Kong), and 7.5 g white sugar into 150 ml boiled water, providing 11 g sugar and 2.4 g protein (Supplementary Table 1). They were instructed to finish the drink in 10 min and to wait for either 15, 30, or 60 min, depending on the treatment allocation. After that, another blood sample was taken, then they were given a serve of a standard meal, which was made by adding 30 g puffed rice cereal (Rice Krispies, Kellogg's, Thailand) and 10 g glucose powder (Dextrosol®, Dextro Energy Gmbh & Co. KG, Germany) into 150 g rice milk (Original Rice Milk, Hain Celestial Group Inc., NY, USA). One serve of the standard breakfast provided 40 g of total available carbohydrate and has a glycemic index of 88. Participants were asked to finish it in 10 min. After that, blood samples were taken at 15, 30, 45, 60, 90, and 120 min after the start of breakfast. In post-meal sessions, after the participant arrived at the venue, they gave the fasting blood sample after 15 min of resting. Then they consumed a serve of the standard meal, followed by a cup of the test drink. They were instructed to finish the breakfast and the drink in 10 min. After that, blood samples were taken at 15, 30, 45, 60, 90, and 120 min after breakfast. With a total of four consumption timepoints being tested (15, 30, and 60 min before the meal and immediately after) and each timepoint being tested twice, each participant went through a total of eight sessions. Consecutive sessions were separated by a washout period of at least three days to minimize the carryover effect. The sequence of the test drink administration was randomly generated for each participant using the *Rand* function in Microsoft Excel (2016) by the first author.

2.4. Sample collection

Blood samples were collected by finger-pricking using disposable, single-use lancing devices (Accu-Chek, Roche, Indianapolis, Indiana

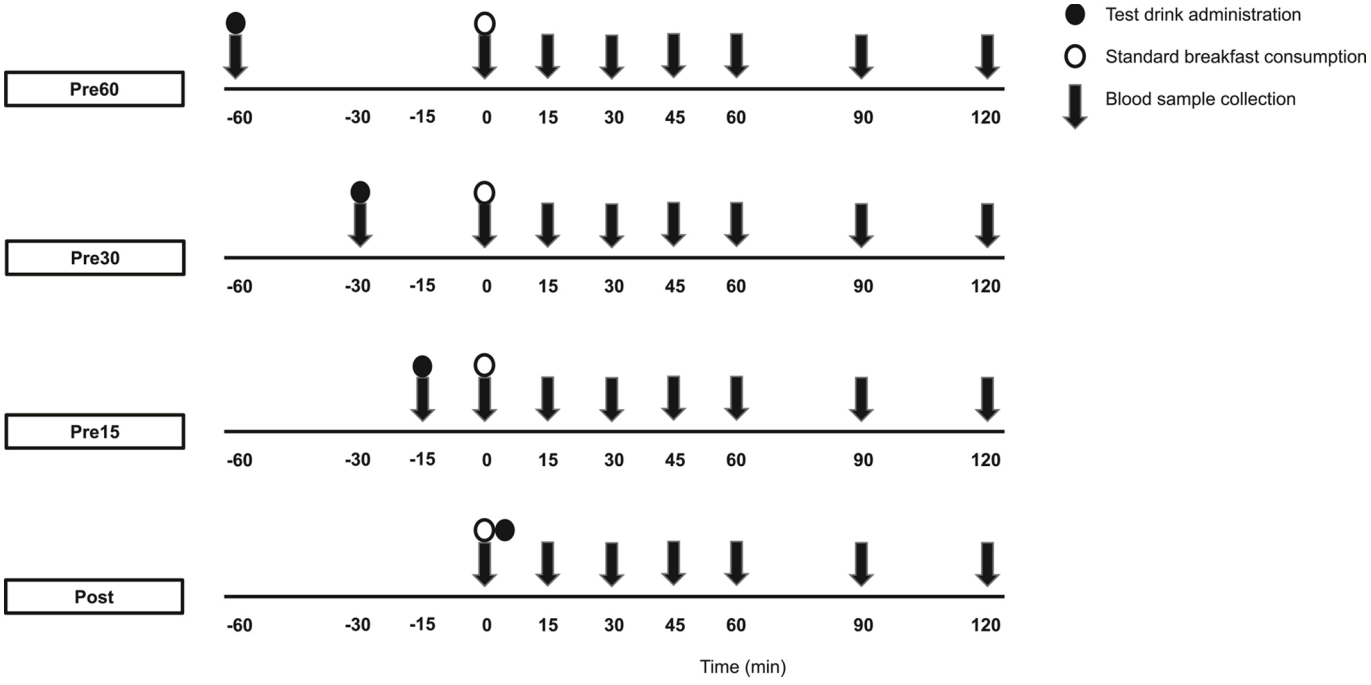

Fig. 1. Procedures of the experimental sessions. Pre60, drinking the test drink (i.e. decaffeinated coffee with milk and sugar added) 60 min before the meal. Pre30, drinking the test drink 30 min before the meal. Pre15, drinking the test drink 15 min before the meal. Post, drinking the test drink immediately after the meal.

USA), and were collected into Eppendorfs coated with heparin. A total of 1 ml of whole blood was collected at each time-point. All samples were stored on ice and centrifuged (3000 *g*, 2 min) within one hour after collection. Plasma was collected and stored in aliquots at −80 °C until analysis.

2.5. Anthropometry measurement

Height and weight were measured to the nearest 0.1 cm and 0.1 kg respectively, both using the Seca 769 electronic column scale (Seca, Hamburg, Germany). Body-mass-index (BMI) was calculated by dividing body weight (kg) by the square of height (m).

2.6. Biochemistry measurement

Concentrations of plasma glucose were measured by the glucose oxidase method (Stanbio Glucose LiquiColor, Stanbio Laboratory, Boerne, TX). Concentrations of both plasma insulin and nitrotyrosine were measured by enzyme-linked immunosorbent assays (ELISA) (insulin: ImmunoDiagnostics Limited, Shatin, Hong Kong; nitrotyrosine: Cell Biolabs Inc., San Diego, CA). The Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) [10] was calculated for each participant to serve as an indirect measurement of insulin resistance, using the fasting glucose and insulin obtained in their first experiment sessions.

2.7. Sample size calculation

Power calculation was done using the G*Power calculator (version 3.1.9.2, Heinrich-Heine-Universität Düsseldorf). Using data from a previous trial [11], a sample size of 10 was determined *a priori* to be able to detect an effect size of 0.68 with 80% power using a randomized crossover design ($\alpha = 0.05$, two-tailed, control group: mean 9·06 (SD 0·42) mmol/l; coffee group: mean 9·41 (SD 1·04) mmol/l, correlation: 0·5). This translates to a 1.1 mmol/L difference in peak glucose levels.

2.8. Statistical analysis

The primary outcome was the peak level of plasma glucose after the meal. The secondary outcomes were the peak insulin level, glucose and insulin measurements at individual time-points, area-under-curve (AUC) from 0 min to 120 min of both glucose and insulin, as well as nitrotyrosine levels at 0, 60, and 120 min in differences from fasting levels. The AUCs were calculated using the trapezoidal method.

Statistical analyses were performed using SPSS (version 23.0, SPSS Inc., Armonk, New York USA). The timepoint measurements, except the fasting measurements, of all biomarkers in all sessions were estimated by mixed-effect linear models using the MIXED procedure. Individual timepoints and time of test drink consumption were specified as the main effects while fasting measurements and baseline coffee consumption frequency (in cups/week) were included as covariates. Participants were specified as a random factor and individual timepoints were specified to have a covariance structure of a first-order autoregressive model. The fasting measurements were estimated using the same method, with the time of test drink consumption as the main effect and baseline coffee consumption frequency as the covariate.

The AUCs of all biomarkers in all pre-meal sessions were also estimated by mixed-effect linear models using the MIXED procedure. Time of test drink consumption was specified as the main effect while fasting measurements and baseline coffee consumption frequency were specified as covariates. Participants were specified as a random factor and the time of test drink consumption was specified to have a covariance structure of compound symmetry.

Post-hoc comparisons were carried out to determine differences of all outcome measures between the test drinks and estimated marginal means were reported. As previously suggested [12], both results with and without correction for multiple comparisons were presented. *Post-hoc* comparisons without correction were done by the Fisher's Least Significant Difference method and adjustments for multiple comparisons were done by the Bonferroni procedure. For all statistical tests, $p < 0.05$ was considered statistically significant.

3. Results

The recruitment flowchart is shown in Supplementary Fig. 1. A total of 10 participants, all of whom were of Asian ethnicity, completed the whole experiment protocol and their baseline characteristics were shown in Table 1. All of them had BMIs within the range 18–23 kg/m^2 and all had normal glucose tolerance, as defined by HOMA-IR below 1.0.

Glycemic excursions in the different experiment sessions are shown in Fig. 2. Glucose levels of all sessions rose and peaked at T = 30 min. Consuming the test drink 30 min before the meal (pre30) led to a significantly lower peak (mean ± SEM: 8.6 ± 0.3 mmol/L) than either having it 15 min before the meal (pre15: 9.5 ± 0.3 mmol/L) or immediately after (post-meal: 10.1 ± 0.3 mmol/L, both $p < 0.05$ when compared with pre30), while the peak postprandial glucose level for having the test drink 60 min before the meal (pre60) was similar. From T = 30–60 min, post-meal consumption led to significantly higher glucose levels than both pre30 and pre60. Moreover, glucose levels of pre15 were also significantly higher than pre30 at T = 30–60 min. After adjusting for multiple comparisons (Supplementary Fig. 2), the differences between pre15 and other treatments were no longer statistically significant at T = 60 min.

Glucose AUCs of pre-meal and post-meal sessions are shown in Fig. 3. The glucose AUC of the post-meal session was 10% higher than those of both pre60 and pre30 (both $p < 0.05$). Similarly, glucose AUC of pre15 was 9% higher than those in these two sessions (both $p < 0.05$). These differences remain statistically significant after adjusting for multiple comparisons (Supplementary Fig. 3).

Postprandial insulin excursions of pre-meal and post-meal sessions are shown in Fig. 4. Peak insulin levels of all sessions appeared at T = 30 min, with post-meal sessions having the highest peak (mean ± SEM, post: 34.8 ± 2.3 μU/ml) and were significantly higher than those of all pre-meal sessions. On the other hand, pre60 had the lowest insulin peak (24.9 ± 2.3 μU/ml) among all sessions, which was significantly lower than those of pre15 ($p = 0.029$) and post ($p < 0.001$). Insulin levels in the post-meal session remained significantly higher than those of all other sessions at T = 45 and 60 min. After adjusting for multiple comparisons (Supplementary Fig. 4), the difference in peak insulin levels between pre60 and pre15 was no longer statistically significant.

For overall insulin excursions (Fig. 5), the post-meal session had the highest insulin AUC among all sessions, which was 38% higher than that of pre60 ($p < 0.001$) and 24% higher than that of pre30 ($p = 0.008$). The insulin AUC of pre15 was also 14% higher than that of pre60 ($p = 0.007$). These differences remained statistically significant after adjusting for multiple comparisons (Supplementary Fig. 5).

Nitrotyrosine results are shown in Supplementary Fig. 6. No significant difference was found in any timepoint among all treatments.

Table 1
Characteristics of study participants.

Variables	Values
Male, n (%)	3 (30)
Age, years	20.8 ± 1.1
BMI, kg/m^2	20.3 ± 0.9
Fasting glucose, mmol/L	5.4 ± 0.3
Fasting insulin, μU/mL	5.0 ± 3.0
HOMA-IR	0.6 ± 0.4

Values are mean ± SD unless otherwise stated. BMI, body mass index. HOMA-IR, Homeostatic Model Assessment of Insulin Resistance.

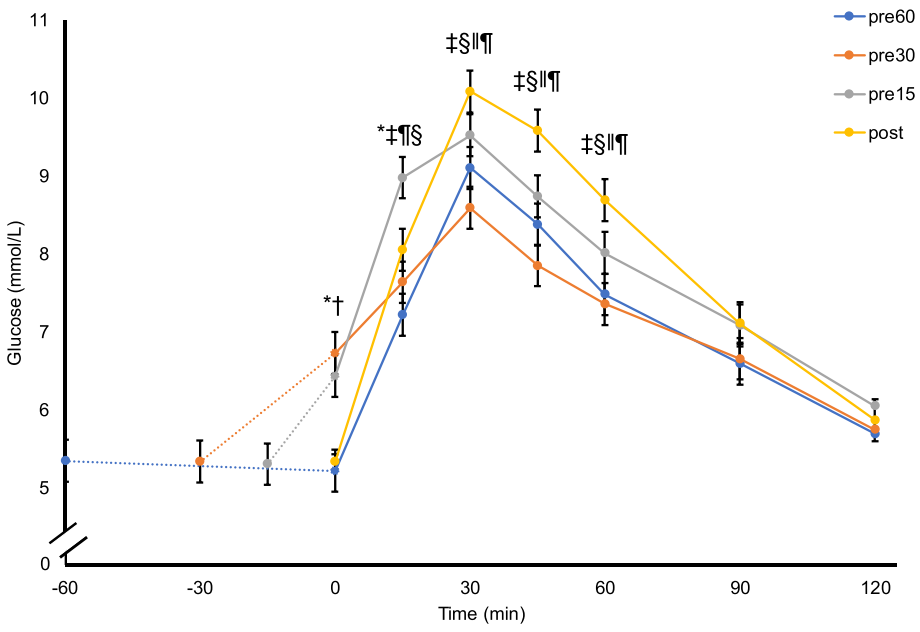

Fig. 2. Glucose levels in pre-meal and post-meal sessions without adjustments for multiple comparisons. Data at each timepoint were estimated using a mixed-effect linear model, with fasting data adjusted for baseline coffee consumption frequency and those at later timepoints further adjusted for fasting levels. All values are estimated marginal means and error bars depict SEM. The following symbols depict significant differences ($p < 0.05$) between indicated sessions: *, pre60 and pre15; †, pre60 and pre30; ‡, pre30 and pre15; §, post-meal and pre60; ‖, post-meal and pre30; ¶, post-meal and pre15. Broken lines were used whenever the excursions between two timepoints were not measured. Pre60, drinking the test drink (i.e. decaffeinated coffee with milk and sugar added) 60 min before the meal. Pre30, drinking the test drink 30 min before the meal. Pre15, drinking the test drink 15 min before the meal. Post, drinking the test drink immediately after the meal.

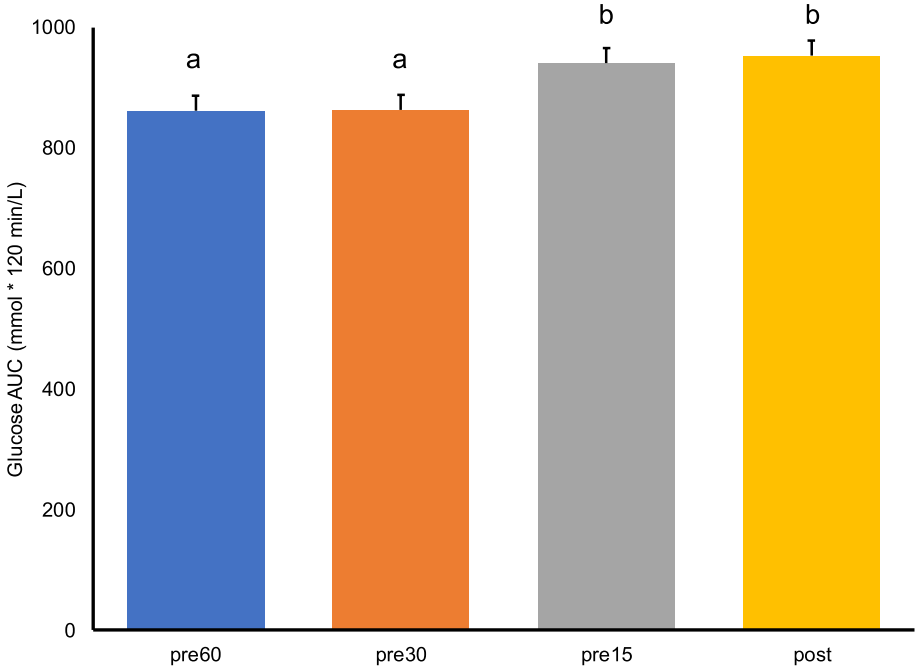

Fig. 3. Glucose AUCs of pre-meal and post-meal sessions without adjustments for multiple comparisons. Comparisons between sessions were done using a mixed-effect linear model with fasting levels and baseline coffee consumption frequency as covariates. Values are estimated marginal means and error bars depict SEM. Unlike alphabets depict statistically significant differences ($p < 0.05$). Pre60, drinking the test drink (i.e. decaffeinated coffee with milk and sugar added) 60 min before the meal. Pre30, drinking the test drink 30 min before the meal. Pre15, drinking the test drink 15 min before the meal. Post, drinking the test drink immediately after the meal.

4. Discussion

Our results showed that by having a small load of sugar, protein, and decaffeinated coffee at least 30 min before the main meal, the postprandial rise in glucose was significantly lower than that observed after consuming the drink and the meal together. This observation did not come with an enhancement in insulin secretion since the overall insulinemia in pre-meal sessions was lower than the post-meal session.

While in previous studies the blunted glycemic and insulinemic responses were observed after a 60-min gap between the test drink consumption and the main meal consumption [5,13], findings from this work showed that these changes could occur with a smaller time gap (30 min). Meanwhile, when the test drink was consumed 15 min before the test meal, the postprandial glucose and insulin levels were higher than when having the test drink 30 and 60 min before the meal. On the other hand, measurements obtained in pre15 were similar to those observed in the post-meal session. This may mean that either the protective effect takes time to build up, or the carbohydrate load in the decaffeinated coffee was still contributing to the postprandial glucose and insulin rises. The design of the study by Bonuccelli et al. [14], which observed improved insulin sensitivity and suppressed endogenous glucose release in participants receiving two consecutive oral glucose tolerance test (OGTT) that were three hours apart, is similar to that of the current work and thus are probable explanations of the mechanisms behind the findings of this study. However, the amount of the two nutrient loads and the time gap between them were both much smaller in this study (current study *vs.* previous studies: 11 g sugar *vs.* 75 g glucose; 2.4 g *vs.* 50 g protein; ≥ 30 min *vs.* 180 min). Future studies that monitor the

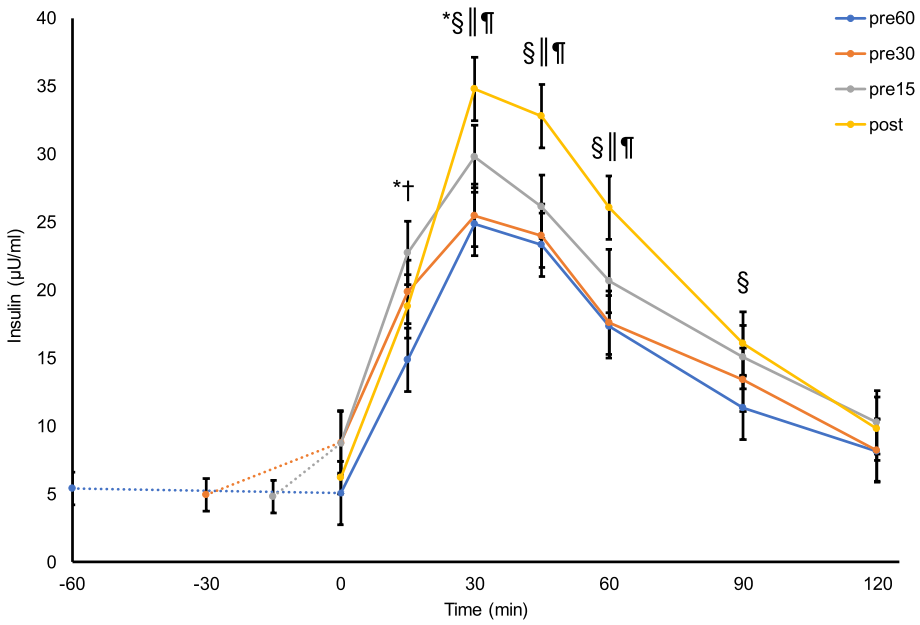

Fig. 4. Insulin levels in pre-meal and post-meal sessions without adjustments for multiple comparisons. Data at each timepoint were estimated using a mixed-effect linear model, with fasting data adjusted for baseline coffee consumption frequency and those at later timepoints further adjusted for fasting levels. All values are estimated marginal means and error bars depict SEM. The following symbols depict significant differences ($p < 0.05$) between indicated sessions: *, pre60 and pre15; †, pre60 and pre30; ‡, pre30 and pre15; §, post-meal and pre60; ‖, post-meal and pre30; ¶, post-meal and pre15. Broken lines were used whenever the excursions between two timepoints were not measured. Pre60, drinking the test drink (i.e. decaffeinated coffee with milk and sugar added) 60 min before the meal. Pre30, drinking the test drink 30 min before the meal. Pre15, drinking the test drink 15 min before the meal. Post, drinking the test drink immediately after the meal.

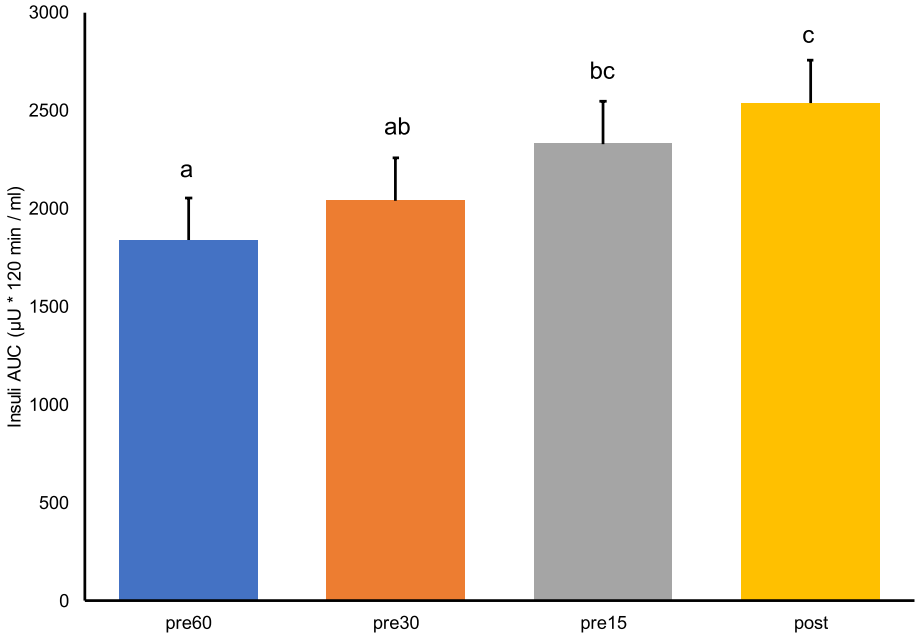

Fig. 5. Insulin AUCs of pre-meal and post-meal sessions without adjustments for multiple comparisons. Comparisons between sessions were done using a mixed-effect linear model with fasting levels and baseline coffee consumption frequency as covariates. Values are estimated marginal means and error bars depict SEM. Unlike alphabets depict statistically significant differences ($p < 0.05$). Pre60, drinking the test drink (i.e. decaffeinated coffee with milk and sugar added) 60 min before the meal. Pre30, drinking the test drink 30 min before the meal. Pre15, drinking the test drink 15 min before the meal. Post, drinking the test drink immediately after the meal. AUC, area under curve.

glucose and insulin dynamics inside the participants' circulations could provide more insight in this regard.

To enable a better comparison, we extrapolated the glucose and insulin data of drinking water 60 min before the test meal in this group of participants. This was done by calculating the mean percentage differences of the glucose and insulin data between consuming water and the test drink 60 min before breakfast at each timepoint obtained in a previous study conducted by the authors [5]. The percentage differences were then applied to the glucose and insulin data of consuming the same test drink 60 min before breakfast for each participant in this study, thereby extrapolating a set of glucose and insulin data of the potential water session. The results of these comparisons are shown in Supplementary Figs. 7-10. While pre-meal water consumption led to peak glucose and insulin levels similar to pre60 and pre30, both glucose and insulin levels after T = 45 min were higher than both pre60 and pre30 till 2 h after meal consumption. Furthermore, both glucose and insulin AUC of the extrapolated water data were higher than those of pre60. These observations mean that consuming decaffeinated coffee with milk and sugar added could lead to lower postprandial glycemic and insulinemic responses than drinking water before the meal. Nonetheless, this extrapolation cannot replace a true treatment control and these findings require confirmation in future studies.

Among the main constituents of the test drink used in this study, both milk [4] and sugar [3,14] were able to lower glycemic responses when consumed together with the meal or before it. Interestingly, insulin levels were found to be unchanged [14] or reduced together with glucose in some studies [4,15], suggesting pathways not involving insulin might be in effect. Boer et al. [16] speculated that a smaller increase in postprandial glucose levels could lead to a reduction in the direct glucose stimulation of pancreatic beta-cells, thereby leading to a smaller insulin release. This may mean that reduced glycemic excursions did not come at the expense of an increased workload of the pancreatic

beta-cells, and factors other than insulin variations were contributing to the blunt in postprandial glucose levels observed. It should be noted that black decaffeinated coffee was not tested in this study because it was shown in our previous study that black decaffeinated consumption before a meal was not able to lower the postprandial glycemic and insulinemic responses [5]. In light of this finding, we decided to use the only test drink that showed significant changes (i.e. decaffeinated coffee with milk and sugar added) as the only test drink in this study and vary only the time of test drink consumption. Therefore, the observed effect could be due to sugar, milk, or the combination of decaffeinated coffee with these two items.

Nitrotyrosine levels, a surrogate marker of oxidative stress in the circulation [2], were expected to be different since a previous study found that it was positively correlated with glucose level [17]. Nonetheless, no significant difference was detected between different consumption times in this study, which is similar to the findings of our previous study [5]. It is possible that the rich antioxidant content in the decaffeinated coffee suppress the rise in nitrotyrosine levels, or the variations would be apparent at a longer observation period, as other studies did [2,17,18]. Future studies might need to monitor nitrotyrosine levels for a longer period to observe clear changes.

One strength of this study is that the results were adjusted for fasting levels, thus avoiding the bias in baseline differences when assessing the outcomes. This study also implemented tight control in the experimental conditions of the participants, such as standardizing the dinner on the previous day, resting period, and physical activity levels before each experimental session.

On the other hand, caution should be exercised when interpreting the results of this study due to the lack of control measurements in the experiment in the same subjects. To account for this, we made use of data from the previous research [5] to extrapolate the results of a possible control session for better comparison. However, we acknowledge that this could not replace the use of a proper control. Furthermore, the results of this study cannot be generalized to non-habitual coffee drinkers and people of other ethnicities. Besides, the results of this study could be due to the consumption of sugar, dairy milk, decaffeinated coffee, or a combination of the above.

5. Conclusions

To conclude, consuming a cup of decaffeinated coffee with cow's milk drink and white sugar added before a high glycemic index meal led to lower glucose and insulin surges when compared with drinking it after a meal. This observation implied that optimizing the time of decaffeinated coffee consumption with milk and sugar added around a meal could benefit mealtime glycemic control without changing the total carbohydrate intake of the main meal. However, further studies are needed to investigate the mechanisms involved.

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Ethics approval

Ethics approval was obtained from the Human Research Ethics Committee of the University of Hong Kong (Approval no.: EA1712012).

Authors' contributions

JCYL designed the project; TWHT, WHS, and IMYT ran the experiments, collected samples, and carried out measurements; JCYL and TWHT analyzed the data; TWHT drafted the manuscript; TWHT, JCYL, and JMFW revised the manuscript; JCYL and JMFW oversaw the whole project. All authors read and approved the final manuscript.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.hnm.2021.200124.

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