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The effects of isomaltulose ingestion on gastric parameters and cycling performance in young men

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ABSTRACT

Background/Objective: Isomaltulose is a disaccharide with a low glycaemic index and plays a role in maintaining postprandial glucose. The maintenance of glucose availability during prolonged exercise has been shown to enhance exercise performance. The present study compared the effects of pre-exercise isomaltulose versus maltodextrin ingestion on gastric parameters and cycling performance in young men.

Methods: Fourteen young men (mean ± S.D., age 23 ± 2 years) performed 60 min of continuous cycling at 75% of maximum heart rate followed by a 15-min exercise performance test while ingesting a 500-mL of water containing 100 mg of ^{13}C-sodium acetate with either 50 g of isomaltulose or 50 g of maltodextrin. Gastrointestinal discomfort was assessed periodically using an 11-point visual analogue scale throughout the study. The gastric emptying rate was evaluated periodically with the ^{13}C-sodium acetate breath test. For the exercise performance test, participants were instructed to pedal a cycle ergometer, exerting as much effort as possible at a self-selected pace.

Results: Plasma glucose and insulin concentrations measured at 30 min after ingestion were lower in the isomaltulose trial than in the maltodextrin trial. There were no differences in mean power output during the exercise performance test, gastric emptying rate or the subjective feelings of gastrointestinal discomfort between both trials.

Conclusion: Under the current exercise protocol, pre-exercise ingestion of isomaltulose compared with maltodextrin provided no additional benefit relative to gastric emptying or aerobic exercise performance. Both isomaltulose and maltodextrin ingestion did not influence gastrointestinal distress during 60 min of cycling and performance test.

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Introduction

Carbohydrates are widely recognised as an important energy source during exercise from early studies.[1,2] Since muscle glycogen and blood glucose are important substrates for adenosine triphosphate resynthesis during prolonged exercise, these substrates play a major role in carbohydrate availability and unitisation for endurance exercise. Over the last three decades, the effects of consuming low-glycaemic index meals prior to an acute bout of exercise on exercise performance has received attention (for a review of these, see References.[3]). A low-glycaemic index meal before exercise is believed to be beneficial during exercise by maintaining euglycaemia, altering substrates utilisation and preserving muscle glycogen.[3]

Isomaltulose is a disaccharide composed of alpha-1,6-linked glucose and fructose and is known as a slow-digesting and fully

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digestible carbohydrate with a low glycaemic index value (i.e., 32).[4] In addition, isomaltulose plays an important role in the gastrointestinal tract, such as improving incretin responses after ingestion[5] (i.e., stimulation of glucagon-like peptide-1 and suppression of glucose-dependent insulinotropic polypeptide as compared with the effects of sucrose) and increasing lipid oxidation[6] at rest and during exercise as compared with the effects of sucrose plus glucose. The effect of pre-exercise isomaltulose intake on exercise performance has been investigated in three previous laboratory-based studies in healthy individuals.[7–9] Only one of these studies noted beneficial effects, but not statistically, on exercise performance after isomaltulose ingestion.[7] The other two studies found that unimproved or impaired exercise performance after isomaltulose ingestion compared with maltodextrin[9] or fructose-maltodextrin[8] ingestion, respectively. The timing and amount of isomaltulose ingestion, the type of exercise performance test, and participants differences are possible reasons for the inconsistent findings among studies. Thus, further research is needed in this field. Furthermore, these three previous studies were limited regarding their methods for evaluating exercise performance substrate oxidation, blood parameters and subjective feelings of abdominal discomfort were consistently used. Due to the slow rate of isomaltulose hydrolysation, which could slow gastric emptying, a high intake of this carbohydrate could lead to abdominal discomfort and adversely affect exercise performance.[8] Indeed, gastric emptying[10] and splanchnic blood flow[11] have been suggested as potential modulators of gastrointestinal symptoms such as nausea and stomach pain. This is important to address because sufficient delivery of exogenous nutrients to the working muscle may be a critical factor influencing exercise performance.[12] However, the effects of pre-exercise isomaltulose ingestion on gastric parameters remain unclear this is a research gap in the literature and no previous studies have objectively examined relevant gastric parameters.[7–9]

The purpose of the present study was to compare the effects of isomaltulose versus maltodextrin ingestion on gastric parameters and cycling performance in young men after performing 60 min of cycling exercise. We tested the hypothesis that ingesting isomaltulose, compared with ingesting maltodextrin, would lead to greater improvement in glycaemic control and better exercise performance.

Methods

Participants

After approval from the Institutional Ethics Committee on Human Research (Approval number: 2017-171), 14 healthy, young men gave written informed consent to participate in this study. This study was conducted in accordance with the Declaration of Helsinki. The physical characteristics of the participants (mean ± SD) were as follows: age 22.9 ± 2.5 years, height 1.72 ± 0.04 m, body mass 66.5 ± 6.3 kg, body mass index 22.5 ± 1.9 kg/m^2, systolic blood pressure 111 ± 6 mm Hg, diastolic blood pressure 76 ± 6 mm Hg and maximum oxygen uptake 49.4 ± 8.6 ml/kg/min). All participants were non-smokers and were not taking any medications, and their body masses had been stable for at least 3 months before the study.

Anthropometric measures

Body mass was measured to the nearest 0.1 kg using a digital scale (Inner Scan 50, Tanita Corporation, Tokyo, Japan) and height to the nearest 0.1 cm using a stadiometer (YS-OA, AS ONE Corporation, Osaka, Japan). Body mass index was calculated as weight in kilograms divided by the square of height in meters. Arterial blood pressure was measured twice from the right arm after 5 min of seated rest using a standard mercury sphygmomanometer (605P, Yagami Inc Aichi, Japan).

Preliminary tests

Participants participated in two preliminary exercise tests performed on a cycle ergometer (Monark 894E, Monark, Varberg, Sweden). A 16-min, four-stage, submaximal cycling test was conducted to determine the relationship between cycling workload and oxygen uptake. The initial cycling workload was set 0.5 kg. The cadence of the cycle ergometer was set at 60 rpm throughout the test. The workload was increased by 0.5 kg every 4 min. Next, maximum oxygen uptake was measured directly with an incremental protocol until the participants reached volitional fatigue. The initial workload of the cycle ergometer was set between 2.0 and 3.5 kg depending on each participant's fitness level via interview for this test. Thereafter, the workload was increased by 0.5 kg every 3 min. Oxygen uptake, carbon dioxide production and respiratory exchange ratio were measured breath-to-breath through a stationary gas analyser (Quark CPFT, COSMED, Rome, Italy). Heart rate was monitored throughout these tests using short-range telemetry (Polar RCX3, Polar Electro, Kempele, Finland). Ratings of perceived exertion (RPE) were assessed periodically during the tests by using the Borg scale.[13] Data generated from these two tests were used to determine the cycling workload at 75% of each participant's maximum heart rate (75% of HRmax 161 ± 6 bpm), and this workload was used for the main trials.

Study design and protocol

A randomised, double-blind, controlled, cross-over design was used in the present study. Each participant underwent two laboratory-based trials in random order as follows: 1) isomaltulose trial and 2) maltodextrin trial. The interval between both trials was at least 1 week. A schematic representation of the study protocol is shown in Fig. 1. Participants reported to the laboratory at 0900 h after a 10-h overnight fast. After a 30-min seated rest, fasting blood samples were collected by venipuncture at 0930 h for the measurement of circulating concentrations of triglycerides (TG) and nonesterified fatty acids (NEFA), glucose and insulin. Then, participants ingested a 500-mL of water containing 100 mg of ^{13}C-sodium acetate (Cambridge Isotope Laboratories, Inc Andover, MA, USA) with either 50 g of isomaltulose or 50 g of maltodextrin. Participants rested for 30 min in the laboratory until 1000 h, and then participants performed cycling exercise at 75% of HRmax for 60 min. In each trial, participants consumed a 100-mL of water every 15 min during a 60-min cycling exercise. Thereafter, the participants performed a 15-min cycling performance test. In this performance test, the participants were instructed to pedal a cycle ergometer (Monark 894E, Monark, Varberg, Sweden), exerting as much effort as possible at a self-selected pace. Work for each exercise performance test was calculated as the mean power output multiplied by duration (i.e., 15 min) using the Anaerobic Test Software (Monark ATS Software, Monark, Varberg, Sweden). Heart rate was monitored continuously using short-range telemetry. Gastrointestinal discomfort and RPE were assessed periodically using an 11-point (from −5 to +5) scale (i.e., the left (−5) and right (+5) ends of the scale were labelled “extreme discomfort” and “extreme comfort”) and the Borg scale,[13] respectively throughout the study. Further blood samples were collected by venipuncture at 1000 h (i.e., immediately before the 60-min cycling exercise), 1100 h (i.e., immediately after the 60-min cycling exercise) and 1115 h (i.e., immediately after the 15-min cycling performance test). The rate of gastric emptying was evaluated with the ^{13}C-sodium

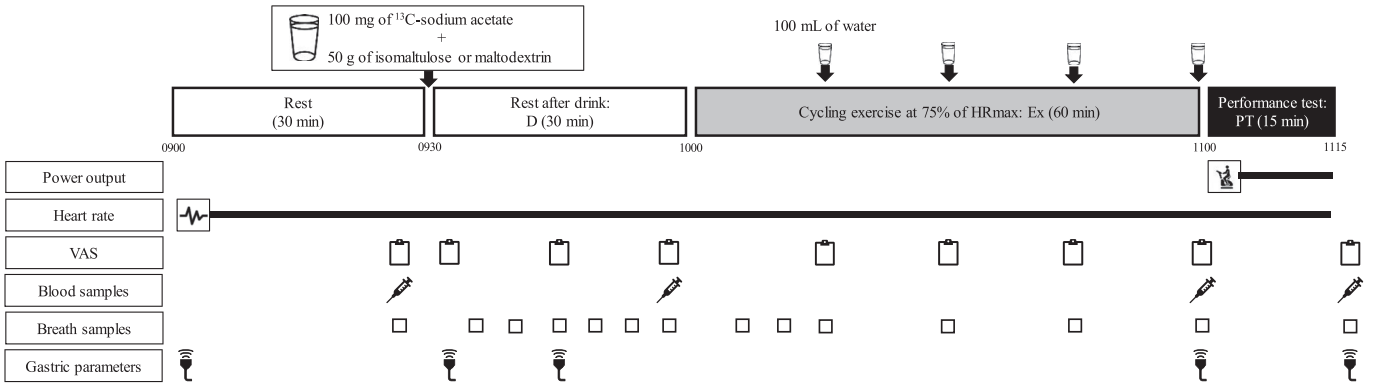

Fig. 1. A schematic representation of the study protocol. VAS, visual analogue scale.

acetate breath test.[14] Breath samples were collected at 5-min intervals during the resting period (i.e., 0930, 0935, 0940, 0945, 0950, 0955 and 1000 h), during the first 15 min of the 60-min cycling exercise (i.e., 1005, 1010 and 1015 h), and at 15-min intervals for the last 45 min of the 60-min cycling exercise (i.e., 1030, 1045 and1100 h). The final breath samples were collected immediately after the 15-min cycling performance test (i.e., 1115 h). Simultaneous pulsed and echo Doppler ultrasound flowmetry (LOGIQ3, GE Healthcare, Piscataway, New Jersey, USA) were performed to assess the change in the splanchnic blood flow, i.e., the celiac artery (CA) and superior mesenteric artery (SMA), the cross-sectional gastric antral area and gastric contractions at 0915, 0945, 1000 (i.e., before the 60-min cycling exercise), 1100 (i.e., immediately after the 60-min cycling exercise) and 1115 h (i.e., immediately after the 15-min cycling performance test). The atmospheric temperature and humidity were maintained at $23.3 \pm 0.7\,^{\circ}C$ and $47.7 \pm 2.2\%$, respectively, throughout the trials.

Analytic methods for blood, breath and gastric parameters

For serum TG and NEFA measurements, venous blood samples were collected into tubes containing clotting activators for the isolation of serum. Thereafter, samples were allowed to clot for 30 min at room temperature and then centrifuged at $1861 \times g$ for 10 min at $4\,^{\circ}C$. Serum was removed, divided into aliquots, and stored at $-80\,^{\circ}C$ for later analysis. For plasma glucose measurements, venous blood samples were collected into tubes containing sodium fluoride-EDTA. For plasma insulin measurements, venous blood samples were collected into tubes containing dipotassium salt-EDTA. Thereafter, both tubes were immediately centrifuged and treated as previously mentioned. Enzymatic, colorimetric assays were used to measure serum TG (Pure Auto S TG-N; Sekisui Medical Co. Ltd Tokyo, Japan), serum NEFA (NEFA-HR; Wako Pure Chemical Industries, Ltd Osaka, Japan) and plasma glucose (GLU-HK(M); Shino-Test Corporation, Kanagawa, Japan). Enzyme-linked immunosorbent assay (ELISA) was used to measure plasma insulin (Mercodia Insulin ELISA; Mercodia AB, Uppsala, Sweden). All analyses for each participant were completed within the same run for each measure. Intra-assay coefficients of variation were 0.72% for TG, 0.81% for NEFA, 0.70% for glucose and 8.2% for insulin.

An isotope ratio mass spectrometer (POCOne; Otsuka Electronics Co. Ltd Osaka, Japan) was used to measure the $^{13}CO_2/^{12}CO_2$ ratio of breath samples. The CO_2 production was assumed to be 300 mmol per square meter of body surface per hour. Body surface area was calculated from body mass and height.[15] Time courses of the percentage of $^{13}CO_2$ recovery per hour and the cumulative percentage of $^{13}CO_2$ recovery were determined. Tmax represents the time taken to reach the maximum rate of pulmonary $^{13}CO_2$ excretion, and half time represents the time taken for half the contents of the stomach to empty. Tmax and half time were calculated based on a standard analytical method.[16]

CA supplies blood flow to the stomach, liver and spleen, while the SMA supplies the entire small intestine, proximal portions of the colon and the pancreas. Simultaneous pulsed and echo Doppler ultrasound flowmetry (LOGIQ3, GE Healthcare, Piscataway, New Jersey, USA) were used to measure blood velocity and vessel diameters of CA and SMA, and cross-sectional gastric antral areas, as in previous studies.[17–22] To maintain precision of these measurements, a curved-array Doppler-scan probe was operated at a pulsed Doppler frequency of 3.3 MHz, and the Doppler beam insonation angle relative to the blood vessel was maintained at $\leq 60^{\circ}$. After obtaining these signals for measuring blood velocity for 1 min, a cross-sectional image of the vessel and antral area were recorded for 30 s and 1 min, respectively. The B-mode images sent from the Doppler monitor were recorded to enable later measurement of the vessel diameters and cross-sectional gastric antral areas using image-editing software (ImageJ 1.47, Wayne Rasband, National Institute of Mental Health, USA). Heart rate and blood velocity were sampled at 20 kHz using an A/D converter (PowerLab 4/26, ADInstrument, Bella Vista, NSW, Australia). The spectra of the blood velocity signals were analysed offline with our own Doppler signal processing software, and beat-by-beat blood velocity values were calculated. For improving the sensitivity, blood velocity was determined by averaging the ten largest values in each minute so as to eliminate respiration-induced data variations.[17–21] Blood velocities in CA and SMA were calculated as $\pi \cdot r^2 \cdot$blood velocity $\times 60$, where r is the radius of the artery.

Calculations and statistical analysis

Data were analysed using the Predictive Analytics Software (PASW) version 23.0 for Windows (SPSS Japan Inc Tokyo, Japan). The Shapiro-Wilk test was used to check for normality of distribution all parameters were found to be normally distributed. The paired *t*-test was used to assess differences in mean power output during the 15-min exercise performance test, and in Tmax and half time between the trials. Repeated-measures two-factor ANOVA was used to examine differences between the trials over time for blood metabolites, mean power output during the cycling exercise performance test, $^{13}CO_2$ excretion, splanchnic circulations (blood velocity, vessel diameter and blood flow in the CA and SMA), subjective feelings of gastrointestinal discomfort, the cross-sectional gastric antral area and frequency of gastric contractions. Where significant trial - time interactions and trial effects were found, the

values were subsequently analysed using a Bonferroni multiple comparison test. Statistical significance was accepted at the 5% level. Results are reported as mean ± S.D.

Results

Blood metabolites

Plasma glucose and insulin concentrations during each trial are shown in Fig. 2. Plasma glucose concentrations did not differ between the isomaltulose and maltodextrin trials (Fig. 2A). There was a difference in the pattern of response between the trials (trial × time interaction, $p < 0.05$). Post-hoc analyses indicated that the rate of increase in plasma glucose concertation was lower in the isomaltulose trial than in the maltodextrin trial at 30 min after drink ingestion. Plasma insulin concentrations were lower in the isomaltulose trial than in the maltodextrin trial (main effect of trial, $p < 0.05$) (Fig. 2B). There was a difference in the pattern of response between the trials (trial × time interaction, $p < 0.05$). Post-hoc analyses indicated that the rate of increase in plasma insulin concentration was lower in the isomaltulose trial than in the maltodextrin trial at 30 min after drink ingestion. Serum TG and NEFA concentrations did not differ between the isomaltulose and maltodextrin trials (data not shown).

Exercise performance

There was no difference in mean power output during the cycling exercise performance test between the isomaltulose and maltodextrin trials (Fig. 3). The mean power output in the isomaltulose and maltodextrin trials was 190 ± 21 W and 187 ± 24 W, respectively.

Gastric parameters

$^{13}CO_2$ excretion during each trial is shown in Fig. 4. $^{13}CO_2$ excretion did not differ between the isomaltulose and maltodextrin trials (Fig. 4). There was no difference in the pattern of response between the trials. Tmax (20 ± 4 min versus 20 ± 4 min) and half time (30 ± 5 min versus 32 ± 6 min) of $^{13}CO_2$ excretion did not differ between the isomaltulose and maltodextrin trials. The blood flows in CA and SMA during each trial is shown in Fig. 5. CA blood flow did not differ between the isomaltulose and maltodextrin trials (Fig. 5A). SMA blood flow was lower in the isomaltulose trial than in the maltodextrin trial (main effect of trial, $p < 0.05$) (Fig. 5B). There was a difference in the pattern of response between the trials (trial × time interaction, $p < 0.05$). Post-hoc analyses indicated that the rate of increase in SMA blood flow was lower in the isomaltulose trial than in the maltodextrin trial at 15 min after drink ingestion and at 60 min after the cycling exercise. SMA blood velocity was lower in the isomaltulose trial than in the maltodextrin trial (main effect of trial, $p < 0.05$) (Table 1). There was a difference in the pattern of response between the trials (trial × time interaction, $p < 0.05$). Post-hoc analyses indicated that the rate of increase in SMA blood velocity was lower in the isomaltulose trial than in the maltodextrin trial between 15 min after drink ingestion and 60 min after the cycling exercise. CA blood velocities and vessel diameters in CA and SMA did not differ between the isomaltulose and maltodextrin trials (Table 1). The cross-sectional gastric antral area, the frequency of gastric contractions and the subjective

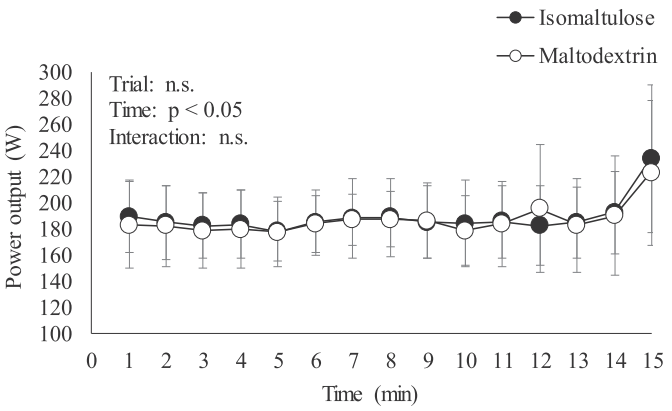

Fig. 3. The mean power output during a 15-min exercise performance test in the isomaltulose and maltodextrin trials. Filled and open circles denote data for the isomaltulose trial and maltodextrin trials, respectively.

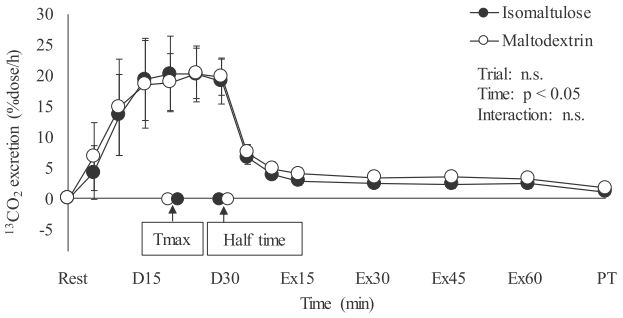

Fig. 4. $^{13}CO_2$ excretion in the isomaltulose and maltodextrin trials. Filled and open circles denote data for the isomaltulose and maltodextrin trials, respectively.

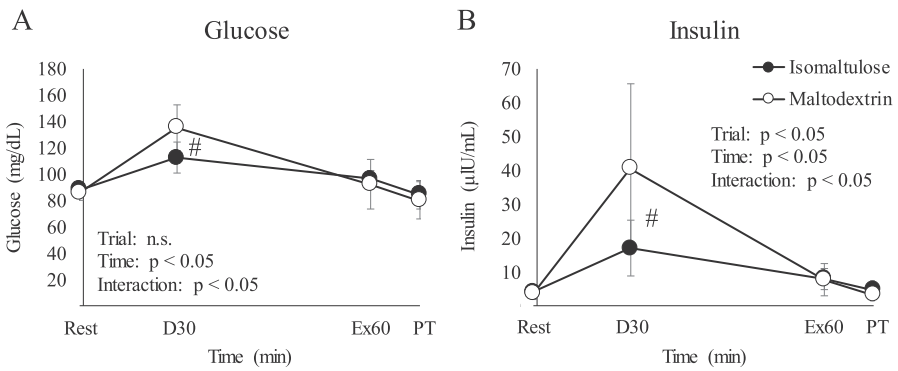

Fig. 2. Plasma glucose (A) and insulin (B) concentrations in the isomaltulose and maltodextrin trials. Filled and open circles denote data for the isomaltulose and maltodextrin trials, respectively. $^{\#}P < 0.05$, difference between trials.

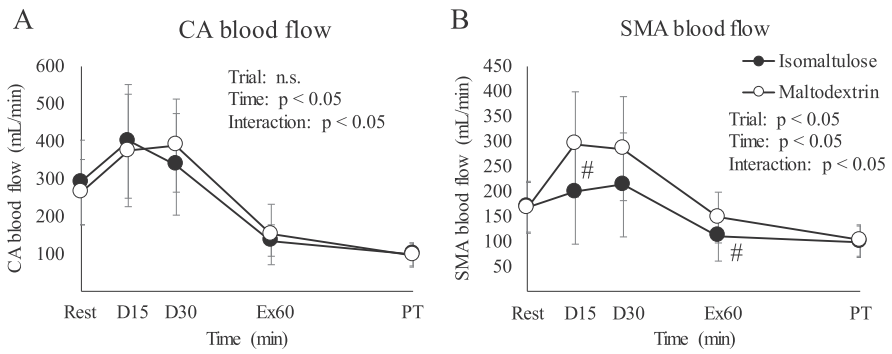

Fig. 5. The celiac artery blood flow (A) and the superior mesenteric artery blood flow (B) in the isomaltulose and maltodextrin trials. CA, celiac artery; SMA, superior mesenteric artery. Filled and open circles denote data for the isomaltulose and maltodextrin trials, respectively. $^{\#}P < 0.05$, difference between trials.

feelings of gastrointestinal discomfort did not differ between the isomaltulose and maltodextrin trials (Table 1).

Discussion

In the present study, although isomaltulose ingestion did not appear to improve cycling performance, the rates of gastric emptying determined by the ^{13}C-sodium acetate breath test may indicate that gastrointestinal distress was similar between isomaltulose and maltodextrin ingestion. These findings were also confirmed by the subjective feelings of gastrointestinal discomfort None of the participants reported any gastrointestinal discomfort during and after exercise in response to both isomaltulose and maltodextrin ingestion.

In accordance with a previous study,[7] the present study demonstrates that pre-exercise isomaltulose ingestion was effective in attenuating postprandial glucose concentrations measured 30 min after ingestion compared with pre-exercise maltodextrin ingestion. Although simultaneous reductions in glucose and insulin concentrations were observed before exercise in the present study both glucose and insulin concentrations returned to similar values in both trials at 60 min post-exercise. This contrasts with a previous study by König and collogues[7] that reported higher glucose concentrations during 90 min of cycling after ingesting 75 g/750 mL of isomaltulose compared with 75 g/750 mL of maltodextrin. Since we were unable to assess blood glucose and insulin concentrations during 60-min cycling and resting glycogen content, it is not known if pre-exercise isomaltulose ingestion maintained prolonged blood glucose availability in the present study. Further controlled studies are needed to clarify the influence of isomaltulose intake on carbohydrate availability during exercise.

The present study demonstrated reduced SMA blood flow after isomaltulose ingestion compared with pre-exercise maltodextrin ingestion, indicating hypoperfusion of small intestinal areas which could have led to increased blood supply to the working muscle. Despite such potential physiological benefits, pre-exercise isomaltulose intake (a 500-mL of drink containing 50 g of isomaltulose: 10% *w/v*) does not appear to improve cycling performance determined by work performed during a fixed time period in healthy active, but untrained men. One possible explanation for this is that circulating concentrations of glucose and NEFA were similar at the end of a 60-min cycling exercise between the isomaltulose and maltodextrin trials. These findings suggest that the exercise duration we used may not have been sensitive enough to modulate substrate utilisation during steady-state exercise. Indeed, a previous study directly comparing the isoenergetic ingestion of isomaltulose and maltodextrin reported that pre-exercise isomaltulose intake transiently maintained glucose concentrations to a greater extent than pre-exercise maltodextrin intake during a 90-min cycling exercise and was accompanied by a tendency for beneficial time trial in trained cyclists.[7] It is, however, worth noting that persistent prolonged blood glucose and/or NEFA availability may not always coincide with improved exercise performance.[8,9] Differences in carbohydrate fluid (i.e., comparing isomaltulose with higher glycaemic carbohydrates), postprandial exercise volume (i.e., intensity duration and frequency of exercise), performance-measurement protocols and participants are likely to further confound the interpretation of findings among the studies. Therefore, further prudent research is required when examining the effect of pre-exercise isomaltulose intake on exercise performance.

In accordance with previous studies directly comparing pre-exercise isoenergetic intake with higher glycaemic carbohydrates,[7,9] ingesting a 500-mL of drink containing 50 g of isomaltulose did not affect the subjective feelings of gastrointestinal discomfort during 60 min of cycling and the cycling performance test. In addition, similar $^{13}CO_2$ excretion, which is an indicator of gastric emptying rate, was observed between isomaltulose and maltodextrin ingestion in the present study. Thus, these objectively assessed findings support the subjectively assessed gastrointestinal findings. Since CA supplies blood to the stomach, liver and spleen, representing digestive activities,[23] our CA blood flow data further support $^{13}CO_2$ excretion data, suggesting similar motility and delivery of absorbed isomaltulose and maltodextrin between the trials. In contrast to our findings, Oosthuyse and collogues[8] reported that isomaltulose intake (63 g/h) before and during 120 min of cycling followed by a 16-km time trial resulted in subjective feelings of gastrointestinal discomfort despite providing a recommended dose of carbohydrate (i.e., 90 g/h for exercise lasts ≥ 120 min).[24] Thus, the high amount and frequent isomaltulose intake used in the study by Oosthuyse and collogues[8] may have impaired the subjective feelings of gastrointestinal comfort compared with the present study. Collectively, these findings need practical validation to ascertain whether isomaltulose should be mixed with other transportable carbohydrates (i.e., glucose or fructose) in order to avoid gastrointestinal distress during and after prolonged endurance exercise.

The present study has some strengths. The rate of gastric emptying determined by the ^{13}C-sodium acetate breath test was objectively used to evaluate the gastrointestinal distress. Given the unique nature of physiological effect after ingesting isomaltulose, a slower rate of absorption may lead to an increase in the subjective feelings of gastrointestinal discomfort during exercise.[8] This was not the case in our experimental protocol as ingested isomaltulose amount and timing were 50 g/500 mL and 30 min, respectively, prior to a 60-min cycling exercise, respectively. One limitation of

Table 1
The gastric parameters and subjective feelings of gastrointestinal discomfort in the isomaltulose and maltodextrin trials.

	Isomaltulose trial								Maltodextrin trial							
	Rest	D0	D15	D30	Ex15	Ex30	Ex45	Ex60	PT	Rest	D0	D15	D30	Ex15	Ex30	Ex45	Ex60	PT
CA blood velocity (m/s)	0.32 ± 0.10	-	0.41 ± 0.13	0.35 ± 0.11	-	-	-	0.15 ± 0.04	0.12 ± 0.04	0.31 ± 0.10	-	0.40 ± 0.14	0.41 ± 0.13	-	-	-	0.15 ± 0.05	0.11 ± 0.03
SMA blood velocity (m/s)	0.18 ± 0.06	-	0.23 ± 0.08#	0.23 ± 0.10#	-	-	-	0.13 ± 0.05#	0.12 ± 0.04	0.19 ± 0.05	-	0.30 ± 0.11#	0.31 ± 0.12#	-	-	-	0.16 ± 0.05#	0.12 ± 0.04
CA diameter (mm)	4.3 ± 0.4	-	4.5 ± 0.3	4.5 ± 0.4	-	-	-	4.4 ± 0.4	4.3 ± 0.3	4.2 ± 0.4	-	4.4 ± 0.3	4.5 ± 0.3	-	-	-	4.5 ± 0.5	4.3 ± 0.3
SMA diameter (mm)	4.4 ± 0.4	-	4.3 ± 0.4	4.4 ± 0.5	-	-	-	4.3 ± 0.6	4.2 ± 0.5	4.3 ± 0.3	-	4.6 ± 0.3	4.4 ± 0.2	-	-	-	4.5 ± 0.4	4.2 ± 0.3
Cross-sectional gastric antral area (cm^2)	4.1 ± 1.9	-	14.7 ± 5.1	13.2 ± 4.2	-	-	-	11.6 ± 4.2	7.6 ± 3.3	4.7 ± 1.5	-	16.2 ± 4.5	13.6 ± 4.1	-	-	-	10.7 ± 4.5	6.7 ± 3.7
The frequency of gastric contractions (times)	1.1 ± 0.9	-	1.9 ± 1.1	2.7 ± 0.7	-	-	-	3.1 ± 0.9	2.1 ± 1.3	1.6 ± 1.0	-	1.7 ± 1.3	2.8 ± 1.0	-	-	-	2.6 ± 0.6	2.6 ± 1.3
Subjective feelings of gastrointestinal discomfort (point)	0.4 ± 1.2	−0.3 ± 1.3	0.0 ± 1.2	0.2 ± 1.1	0.3 ± 1.0	0.1 ± 1.2	0.0 ± 1.1	−0.1 ± 1.1	−0.3 ± 1.2	0.3 ± 1.7	−0.2 ± 2.1	0.1 ± 1.9	0.4 ± 1.7	0.2 ± 1.5	0.1 ± 1.4	−0.1 ± 1.4	−0.1 ± 1.4	−0.3 ± 1.5

CA, celiac artery; SMA, superior mesenteric artery. #P < 0.05, difference between trials.

the present study includes comparing only isomaltulose versus maltodextrin. Although commercially available sports beverages often contain maltodextrin, other sources of carbohydrates such as fructose and sucrose are also contained along with maltodextrin. Indeed, the greatest rates of exogenous carbohydrate oxidation during exercise reported a ratio of 1–1.2 for maltodextrin to 0.8–1.0 for fructose.[25] In addition, we recruited only young untrained men - athletic status may be a factor affecting the exercise performance test.[26,27] Thus, more studies are needed to examine whether pre-exercise isomaltulose ingestion offers metabolic[28] and performance advantage over these carbohydrates in individuals with different training statuses.

It is worth noting that circulating concentrations of glucose and insulin were different between trials although there was no difference in $^{13}CO_2$ excretion in the present study. We have no firm explanation for these findings. Nonetheless, breath testing is based on oxidation of the ingested substrate, and this includes systemic and cellular metabolic processing.[29] Thus, it is not only a reflection of gastrointestinal processing. Indeed, a previous study has shown that the rate of gastric emptying determined by the ^{13}C breath test was similar for different solid meals despite the different circulating concentrations of glucose in these meals.[30]

Conclusion

The present study demonstrates that although isomaltulose ingestion attenuates elevated circulating concentrations of glucose and insulin, it did not improve exercise performance compared to maltodextrin. However, isomaltulose ingestion, compared with maltodextrin ingestion, did not disturb the subjective feelings of gastrointestinal discomfort during 60 min of cycling and cycling performance test.

Declaration of interest

M.S K.M. and Y.N. are employees of Mitsui Sugar Co Ltd. and were not involved in the data acquisition, statistical analysis and writing of the manuscript - M.S K.M. and Y.N. were involved in the study design, the interpretation of the results and the decision to submit the manuscript for publication. M.M. has no professional relationships with the company involved in this study. M.M. received a research grant from Mitsui Sugar Co Ltd.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jesf.2019.06.001.

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