

RESEARCH PAPER

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Sago supplementation for recovery from cycling in a warm-humid environment and its influence on subsequent cycling physiology and performance

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ABSTRACT

This study determined whether sago porridge ingested immediately after exercise (Exercise 1) in warm-humid conditions ($30 \pm 1^\circ\text{C}$, $71 \pm 4\%$ RH; $20 \text{ km}\cdot\text{h}^{-1}$ frontal airflow) conferred more rapid recovery, as measured by repeat performance (Exercise 2), compared to a control condition. Eight well-trained, male cyclists/triathletes (34 ± 9 y, $\text{VO}_{2\text{peak}} 70 \pm 10 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, peak aerobic power 413 ± 75 W) completed two 15-min time-trials pre-loaded with 15-min warm-up cycling following >24h standardization of training and diet. Mean power output was not different between trials during Exercise 1 (286 ± 67 vs. 281 ± 59 W), however, was reduced during Exercise 2 for control (274 ± 61 W) but not sago (283 ± 60 W) that led to a significant performance decrement (vs. Exercise 1) of 3.9% for control and an improvement (vs. control) of 3.7% for sago during Exercise 2 ($P < 0.05$). Sago ingestion was also associated with higher blood glucose concentrations during recovery compared to control. These results indicate that feeding sago during recovery from exercise in a warm-humid environment improves recovery of performance during a subsequent exercise bout when compared to a water-only control. As these effects were larger than the test-retest coefficient of variation for work completed during the 15-min time-trial (2.3%) it can be confidently concluded that the observed effects are real.

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Introduction

The relationship between reduced carbohydrate (CHO) availability and the onset of fatigue has been known for some time.¹ Specifically, the progressive depletion of skeletal muscle's limited glycogen stores and reduction in circulating blood glucose as exercise progresses are linked to performance deterioration and volitional fatigue.² Following the cessation of exercise, muscle glycogen content can be restored to near pre-exercise levels within 24 hours provided adequate amounts of CHO are consumed,³ however for athletes training or competing multiple times daily or on successive days it is ideal that glycogen stores be replenished more rapidly (see ref. 4) to assist optimal rates of recovery. There exists a 'window of opportunity' as glycogen synthesis rates are at their highest during the first few hours following exercise when CHO is consumed.⁵ Therefore, it follows that the consumption of CHO early in the post-exercise period

can enhance performance in a subsequent bout of exercise;⁶ hence the consensus prescription⁷ of CHO as soon as practical post-exercise to maximise recovery between sessions. Furthermore, many competitive situations are such that only few hours separate the next bout of competitive effort, so it is important that the first CHO-containing meal is consumed as soon as possible after the initial bout, and is palatable enough to be ingested when often food intake is not desired.

Many major sporting events take place during the summer, in warm environments or at the hottest part of a day.⁸ During exercise with heat stress, there is consensus that performance is decreased and there is an increased risk of heat illness, especially with high humidity.⁹ Heat stress during exercise also results in alterations in CHO metabolism with Febbraio¹⁰ concluding that heat stress increases CHO and decreases fat utilization. For example, Yaspelkis and Ivy¹¹ demonstrated that exercise in the heat accelerated fatigue

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because of an increase in reliance upon CHO as a substrate, while Jentjens et al.¹² demonstrated that when ambient temperatures increase so does CHO oxidation during exercise largely due to an increased muscle glycogen use. Therefore, Jentjens et al.¹³ proposed that glycogen stores may be sub-optimal in athletes training or competing multiple times daily or on successive days in hot environments.

Where commercially available CHO products are not necessarily affordable or accessible to those competing in sport or exercise, there is a need to investigate local food sources as suitable alternatives which are palatable. Sago (Metroxylum sagu) palms grow all over Southeast Asia, a region with over 600,000,000 inhabitants and a year-round tropical climate. Where there is insufficient rain to grow wet rice, sago palms are used as staple foods. For example, in Malaysia sago starch is an important dietary CHO source¹⁴ with Malaysia, Indonesia and Papua New Guinea being the world's leading countries in the production of sago.¹⁵ In Sarawak, Malaysia, sago is widely used to produce sago pearls that can be boiled and consumed directly as a CHO source.

To date, there has been no investigation of sago meals ingested following exercise, although we have previously observed no performance effect of feeding 0.8 g/kg body-weight sago before (porridge) or during (gel) exercise under warm-humid conditions despite several beneficial physiological responses.¹⁶ However, in that study participants' skeletal muscle and hepatic glycogen stores were likely full due to our careful exercise and dietary control, mimicking typical pre-competition behavior i.e. reduced physical activity and a CHO-rich diet. Sago was nevertheless quite palatable and therefore would likely provide a suitable and easily obtainable meal post exercise for many in the Asian region. Further, the resting glycaemic response to sago ingestion confirms that sago is quickly

absorbed and metabolised to glucose i.e., has a high glycaemic index (GI, see refs. 16, 17), indicating that supplementing sago following exercise may be beneficial when recovering for subsequent exercise bouts, at least in terms of rapid glycogen repletion.

Therefore, the purpose of the present study was to determine whether sago ingestion in recovery between two exercise bouts under conditions of heat stress conferred a performance and/or physiological benefit(s) compared to a control condition.

Materials and methods

Participants

Eight healthy, male cyclists and/or triathletes provided their informed, written consent to participate in the study. Their mean (SD) physical characteristics were, age: 34 (9) y, height: 1.80 (0.11) m, weight: 79 (16) kg, VO_2peak : 70 (10) $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, maximal heart rate: 185 (5) $\text{beats}\cdot\text{min}^{-1}$ and peak aerobic power: 413 (75) W. All participants were regularly cycling >200 $\text{km}\cdot\text{week}^{-1}$ and participated in club-level cycling races. The study was approved by the Massey University Human Ethics Committee and performed in accordance with the 1975 Helsinki Declaration.

Experimental overview

All the participants visited the laboratory on four separate occasions: 1) preliminary submaximal and maximal tests, 2) experimental familiarization, 3–4) experimental trials. The experimental trials were completed using a counter-balanced crossover design, with these trials separated by 7 days, conducted at the same time of day (± 1 h), and following 24 h of dietary and exercise control (see below for details). Trials

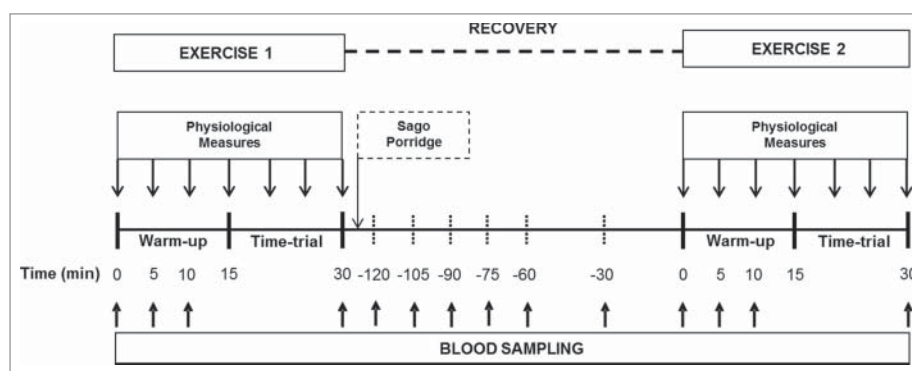


Figure 1. A schematic overview of the experimental protocol.

consisted of a Control (nothing consumed) or sago (Sago porridge consumed during recovery between exercise bouts); a schematic diagram accompanying the following sections can be seen in Figure 1. All trials were completed on an electronically-braked cycle ergometer (Lode Excalibur, The Netherlands), where participants' set-up (e.g. seat/handle bar height and horizontal position etc.) was customised and replicated for each subsequent visit.

Preliminary testing and familiarization

Following body weight and height measurement, preliminary testing was conducted in a moderate laboratory environment (18–22°C) with a fan located in front of the participants with an airflow of 20 km·h⁻¹. A submaximal test required the participant to cycle for 6 min at each of four consecutive submaximal power outputs; 100, 150, 200, and 250 W, all the while maintaining comfortable but constant cadence. Following 10 min rest, an incremental protocol increasing in workload at a rate of 45 W·min⁻¹ (beginning at 100 W) until volitional fatigue, was employed to produce a VO_{2peak} (L/min). Expiratory gases were collected continuously for the determination of VO₂, and heart rate recorded every minute. Following this, a linear relationship between the mean VO₂ (L/min) during the last 2 min of each submaximal stage and power output was determined and used to calculate a power output which would elicit 75% (time-trial) of VO_{2peak} (L/min) for each participant for the remaining two trials.

The familiarization trial was undertaken to ensure participants were accustomed to the procedures employed during the investigation and to minimise any potential learning effects during the experimental trials. These trials replicated entirely the experimental trials outlined below.

Dietary and exercise control

The twenty-four hours prior to any experimental trial was marked by abstinence from alcohol and only habitual caffeine use (as abstinence would in itself confound from withdrawal effects). On the day before any experimental trial, participants' only exercise was when they attended the laboratory to complete a standardized training ride 60 min in duration at a fixed power output that elicited ~65% of their maximum heart rate in moderate environment (18 – 22°C).

Following this, they were provided with a standardized snack (1x UP&GO[®], Sanitarium (New Zealand Health Association Ltd), New Zealand: 823 kJ providing 30.3 g carbohydrate, 8.3 g protein and 3.8 g fat) to be consumed immediately, dinner (2x Watties Snack Meals, Heinz Watties, New Zealand: 2100 kJ providing 42.0 g carbohydrate, 31.6 g protein and 22 g fat, and 1 × One Square Meal[®], Cookie Time Limited, New Zealand: 1450 kJ providing 45.1 g carbohydrate, 8.4 g protein and 11.7 g fat) and breakfast (at least 2 but not more than 4 h prior to visiting the laboratory) for the day of the trial (1x UP&GO[®]: 823 g providing 30.3 g carbohydrate, 8.3 g protein and 3.8 g fat, and 1x One Square Meal : 1450 kJ providing 45.1 g carbohydrate, 8.4 g protein and 11.7 g fat). This dietary and exercise control minimised any variation in pre-trial metabolic state and skeletal muscle glycogen level. Fluid was encouraged and available *ad libitum* to ensure adequate hydration. A euhydrated state was further ensured by instructing the participants to drink a pre-measured bolus of water (5 ml·kg⁻¹ body-weight) two hours prior to each trial.

Experimental trial development

We¹⁸ have previously developed a reliable protocol for collecting both steady-state physiological and self-paced performance data under conditions of exercise heat stress an hour in duration that was successfully used¹⁶ to investigate the efficacy of sago ingestion before and during exercise. However, the combination of ambient heat stress and intensity/duration of work resulted in a rise in core temperature of 1.8–2.1°C·h⁻¹ and sweat loss of 1.2–1.7 L·h⁻¹, making recovery from such exercise for a subsequent repeated bout within two hours unlikely from a thermo- and osmo-regulatory point of view. Furthermore, competitive sporting situations do not usually require repeated bouts of such duration within this time-frame, therefore it was decided to retain the 15-min time-trial but reduce the fixed-intensity pre-load from 45 to 15 min, as this would still allow sufficient warm-up in preparation for the moderate to high intensity time-trial and the collection of some steady-state physiological data. An additional intention with the exercise protocol was to cause some disturbance to the participants' carbohydrate stores, and in that respect previous studies that have used similar duration and intensity exercise protocols have demonstrated sufficient skeletal muscle glycogenolysis i.e. ~150 mmol/kg dry wt or ~40% content

(see refs. 19, 20). Further, a one-²¹ and two-hour⁵ period of post-exercise recovery following a bolus of CHO ingestion has demonstrated an enhanced circulating plasma glucose and skeletal muscle glycogenesis, supporting our design of a 120-min recovery between the exercise bouts.

Experimental procedure

These sessions were conducted in a thermally stressful environment at a dry-bulb temperature of $30 \pm 1^\circ\text{C}$ and relative humidity of $71 \pm 4^\circ\text{C}$ with a fan located in front of the participants with an airflow of $20 \text{ km}\cdot\text{h}^{-1}$.

On arrival to the laboratory participants voided and then self-inserted a rectal thermistor 10 cm beyond the anal sphincter. A cannula was inserted into a forearm vein, secured, and kept patent with periodic saline infusion. Following measurement of body weight participants entered the environmental chamber wearing only cycling shorts, shoes and socks. Once seated on the ergometer, the heart rate monitor was positioned across the chest and four skin surface thermistors were attached to the chest, arm, thigh, and calf on the right side of the body and connected to a USB-based Temperature Measurement Device. Resting values for all measurements were recorded, including a 4ml venous blood sample.

Participants completed a warm-up consisting of 5 min fixed-intensity cycling at each of three consecutive workloads: 100, 150 and 200 W. Expired gas samples were collected during the final 2 min of every stage as was a venous blood sample, with heart rate, core and skin temperatures also recorded every 5 min throughout the trial. Potable water (kept at room temperature of $\sim 20^\circ\text{C}$) was provided to drink *ad libitum* in aliquots of $3 \text{ ml}\cdot\text{kg}^{-1}$ body weight either at pre-exercise or when requested during exercise to minimise dehydration. Immediately on completion of the 15-min warm-up, the ergometer was set to linear mode, based on the formula of Jeukendrup et al.,²² and participants were asked to complete as much work as possible in the 15 min with the only information received being when every 3 min had elapsed. Following completion of the time-trial, participants performed a low-intensity cool-down for at least 5 min where recovery was monitored. On exiting the environmental chamber, participants were allowed to towel down and weighed and then remained semi-reclined in a comfortable moderate laboratory

environment ($20\text{--}22^\circ\text{C}$) for the following 2 hours. At the start of these 2 hours, participants received either sago (0.8 g/kg body weight) or control (nothing) and were allowed/encouraged to drink water *ad libitum* during this recovery. Venous blood samples were taken at 15-min intervals (first hour) and then 30-min intervals (second hour). Following this recovery period, the above exercise protocol (and measures) were then subsequently repeated.

Sago supplementation

For Sago 0.8 g/kg bodyweight cooked sago porridge was consumed as a bolus as soon as possible following the completion of exercise, once seated comfortably (~ 5 min). A dose of 0.8 g/kg bodyweight was chosen because it could then be compared directly with sago supplementation before and during exercise¹⁶ to determine the efficacy of timing for sago supplementation. Sago is $\sim 86\%$ CHO w/w, therefore ingestion of sago at a rate of 0.8 g/kg bodyweight for a 75 kg person, equates to 52 grams of CHO. Preparation of sago followed that described previously where proximate analysis can also be found.¹⁶

Measurements

The subject's height and weight were measured using a stadiometer (Seca, Germany; accurate to 0.1cm) and scale (Jadever, Taiwan; accurate to 0.01kg). The calibrated skin thermistors (Grant Instruments Ltd., Cambridgeshire, UK; accurate to 0.2°C) were secured in place with Transpore Surgical Tape (3 M Healthcare, St. Paul, Minnesota, USA). The skin and calibrated rectal (Covidien Mon-a-Therm, USA; accurate to 0.1°C) thermistors were then connected to a USB-based Temperature Measurement Device and displayed using TracerDAQ[®] software. Weighted mean skin temperature was calculated according to the equation of Ramanathan.²³ Expiratory gases were collected and recorded via Turbofit (VacuMed Vista Turbofit, USA) metabolic software for determination of minute ventilation, oxygen uptake and carbon dioxide production and hence the respiratory exchange ratio (RER); all values as standard temperature, pressure, dry. Prior to each experimental trial, the instrument received a 2-point calibration using a zero and a known gas mixture (β -standard: O_2 15.01%, CO_2 5.02%) and volume (VacuMed 3L Calibration Syringe, USA). Substrate oxidation rates ($\text{g}\cdot\text{min}^{-1}$) were

calculated from indirect calorimetry measurements using the stoichiometric equation proposed by Jeukendrup and Wallis,²⁴ assuming a non-protein contribution. For venous blood sampling the cannula (BD Venflon I.V Cannula, Sweden) was kept patent by regular flushing with 3 ml of sterile saline (sodium chloride 0.9% IV-IM; Multichem NZ Ltd., New Zealand). At each time-point, the initial 2 ml drawn was discarded and then 4 ml blood was collected into a lithium heparin containing vacutainer (Becton-Dickinson, UK). The whole blood was then centrifuged at 4°C and 805 g for 15 min. Following this, aliquots of plasma were transferred into Eppendorf tubes (Genuine Axygen Quality, USA) and stored at -80°C until further analysis. Plasma glucose, lactate, sodium and potassium concentrations were measured using an automated analyzer (ABL FLEX, Radiometer, Denmark) with a repeatability of ≤ 0.1 mmol/L.

Data and statistical analyses

All statistical analyses were performed with SPSS software for windows (IBM SPSS Statistics 20, NY, USA). Descriptive values were obtained and reported as means and standard deviation (SD) unless stated otherwise. A Shapiro-Wilk test was used to ensure data did not differ substantially from a normal distribution. Data were analyzed by two-way (trial x time: work completed, water consumed and sweat lost) or three-way (trial x bout x

time: all other measures) ANOVA for repeated measures. Where main or interaction effects occurred *post-hoc* pairwise analyses were performed using a paired samples *t*-test (Bonferroni correction if appropriate), with statistical significance set at $P < 0.05$. Sphericity was assessed and where the assumption of sphericity could not be assumed, adjustments to the degrees of freedom were made ($\epsilon > 0.75 =$ Huynh-Feldt; $\epsilon < 0.75 =$ Greenhouse-Geisser). Our experimental design, and exercise and dietary control, allowed us to assess the test-retest reliability in time-trial performance (work completed) for Exercise 1 between Control and Sago. Several measures were calculated according to Hopkins;²⁵ these were the mean difference (change in mean), intra-class correlation coefficient (ICC), and the typical error of measurement as a coefficient of variation (CV) between trials.

Results

All eight participants were able to complete all experimental trials. Unfortunately, freezer plasma samples for time-point 15-min were destroyed due to freezer malfunction.

Time trial reliability

As can be seen in Table 1A, the amount of work completed for the time-trial during the familiarization,

Table 1. Individual performances (A) and measures of test-retest reliability (B) for work completed (kJ) during the 15-min cycling time trial following a 15-min warm-up. Trials 1 and 2 refer to Exercise 1 for Control and Sago.

A			
Participant	Familiarization	Trial 1	Trial 2
1	284.6	296.9	286.0
2	278.2	278.1	287.5
3	244.1	245.6	241.9
4	335.1	347.7	321.9
5	202.9	177.0	181.6
6	308.5	308.8	302.1
7	217.0	207.2	208.7
8	200.5	195.8	196.0
Mean \pm SD	259 \pm 51	257 \pm 61	253 \pm 53
B			
Measure of Reliability	Familiarization – Trial 1	Trial 1 – Trial 2	
Intra-class Correlation Coefficient	0.98	1.00	
Lower 95% confidence limit	0.88	0.99	
Upper 95% confidence limit	1.00	1.00	
Change in Mean (%)	-1.8	-1.9	
Lower 95% confidence limit	-7.1	-4.4	
Upper 95% confidence limit	3.7	0.8	
Typical Error as a CV (%)	4.3	2.3	
Lower 95% confidence limit	2.8	1.5	
Upper 95% confidence limit	9.8	4.7	

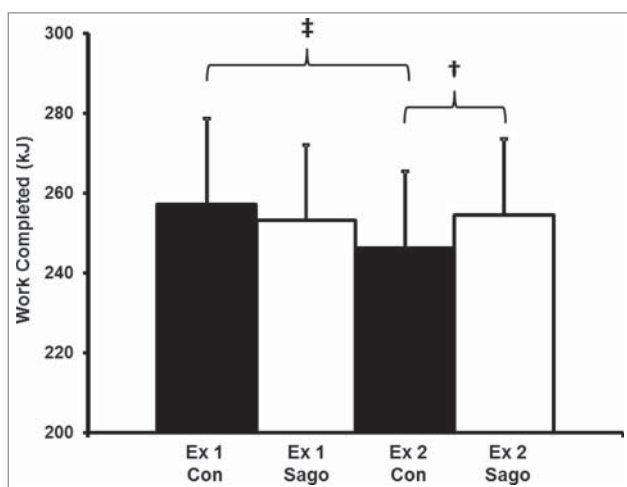


Figure 2. Mean total work completed (kJ) during the 15-min time trial for Control and Sago trials before (Ex 1) and after (Ex 2) a 2-h recovery. ‡ indicates significantly different to Ex 1. † indicates significantly different to Con. Data are expressed as mean \pm SE.

Control and Sago (both Exercise 1) was not different ($p > 0.05$). However, from Table 1B it can be seen that the reliability of the 15-min time-trial was still improved when a familiarization was performed. These data also indicate an acceptable reliability both with and without a familiarization being performed, and put in the context of the reliability of the physiological variables between trials i.e., a CV and ICC of

2.9 % and 0.89, and 5.2 % and 0.79 for the exercise responses of heart rate and VO_2 , respectively.

Time trial performance

The average work completed in the 15-min time-trials for both trials before and following recovery can be seen in Figure 2. A significant interaction effect for work completed ($P < 0.05$) indicates that during Control, work completed during the Exercise 2 (246 ± 55 kJ) was less ($3.9 \pm 3.7\%$, $P < 0.05$) than the Exercise 1 (257 ± 61 kJ), whereas for Sago work completed during Exercise 2 (255 ± 54 kJ) was no different ($0.6 \pm 4.4\%$, $P > 0.05$) than Exercise 1 (253 ± 53 kJ). Therefore, sago supplementation at the start of a 2-h recovery between exercise bouts maintained performance (vs. Exercise 1) and Control did not (difference equating to $3.7 \pm 5.1\%$, $P < 0.05$).

Metabolic responses

Figure 3 shows the plasma glucose and lactate responses during exercise and recovery for both trials. Main effects of time and time*trial were observed for glucose (both $P < 0.05$) although interestingly no effect of the exercise bout was observed (i.e. Exercise 1 vs. Exercise 2). During Exercise 1,

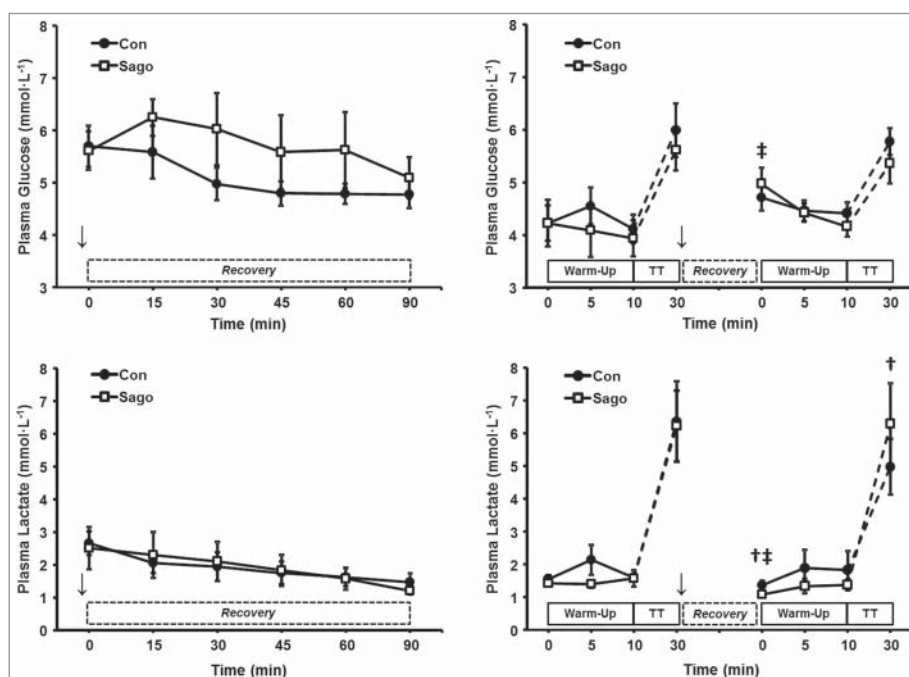


Figure 3. Plasma glucose and lactate concentrations during recovery (left panels) and Exercise 1 and 2 (right panels) for Control and Sago trials. TT: 15-min time-trial. Arrow indicates Sago ingestion. Data are expressed as mean \pm SE. ‡ indicates significantly different to corresponding Exercise 1 time-point. † indicates significantly different to Con at that time-point.

glucose concentrations had only increased above resting at the end of the time-trial in both trials. As far as the recovery glucose response is concerned, a divergent response was observed; Control concentrations began to decrease toward pre-exercise values from 30 minutes onwards whereas for Sago concentrations were maintained elevated throughout this period. This led to a higher glucose concentration at pre-exercise for Exercise 2 than 1 with Sago. During Exercise 2, glucose concentrations decreased below resting in both trials, before being elevated above resting at the end of the time-trial for the Control trial only. Therefore, representative glucose concentrations were: start (Control: 4.2 ± 1.0 , Sago: 4.2 ± 1.2 mmol.L⁻¹) and end (Control: 6.0 ± 1.4 , Sago: 5.6 ± 1.1 mmol.L⁻¹) of Exercise 1, start (Control: 5.7 ± 1.1 , Sago: 5.6 ± 1.0 mmol.L⁻¹) and middle (Control: 4.8 ± 0.5 , Sago: 5.6 ± 2.0 mmol.L⁻¹) of recovery and start (Control: 4.7 ± 0.7 , Sago: 5.0 ± 0.9 mmol.L⁻¹) and end of Exercise 2 (Control: 5.8 ± 0.7 , Sago: 5.4 ± 1.1 mmol.L⁻¹).

Main effects of time, trial and time*trial were observed for lactate (all $P < 0.05$), although akin to plasma glucose, no main effect of the exercise bout was observed. During Exercise 1, lactate concentrations had only increased above resting at the end of the time-trial in both trials. As far as the recovery lactate response is concerned, lactate concentrations began to decrease toward pre-exercise values from 15 (Control) and 30 (Sago) minutes onwards. At the start of Exercise 2, lactate concentrations were lower for Sago than Control and also lower than Exercise 1 for Sago only. During Exercise 2, lactate concentrations only increased above resting at the end of the time-trial in both trials where values for Sago were significantly higher than Control.

The RER and substrate oxidation rates can be seen in Table 2. As expected, a main effect of time was observed for both RER and carbohydrate oxidation rates (both $P < 0.05$) - specific *post-hoc* results can be seen in Table 2 - however, no effects of trial or exercise bout were evident (both $P > 0.05$).

Thermoregulatory responses

The rectal and mean skin temperatures can be seen in Figure 4. A main effect of time was observed for rectal temperature ($P < 0.05$) such that values increased progressively at each time point, although no effect of trial or the exercise bout was observed i.e., Exercise 1 vs. Exercise 2. Therefore, representative values were: start (Control: 37.1 ± 0.4 , Sago: $37.0 \pm 0.5^\circ\text{C}$) and end (Control: 38.3 ± 0.4 , Sago: $38.0 \pm 0.3^\circ\text{C}$) of Exercise 1, and start (Control: 36.9 ± 0.4 , Sago: $37.0 \pm 0.4^\circ\text{C}$) and end of Exercise 2 (Control: 38.2 ± 0.3 , Sago: $38.4 \pm 0.3^\circ\text{C}$). Main effects of time and time*trial were observed for mean skin temperature (both $P < 0.05$) although no effect of the exercise bout was observed i.e. Exercise 1 vs. Exercise 2. During both exercise bouts and both Control and Sago, values increased progressively until 15 minutes, plateauing thereafter. Therefore, representative values were: start (Control: 32.4 ± 0.7 , Sago: $32.1 \pm 1.2^\circ\text{C}$) and 15 min (Control: 33.2 ± 1.0 , Sago: $33.1 \pm 1.1^\circ\text{C}$) during Exercise 1, and start (Control: 32.5 ± 0.6 , Sago: $32.3 \pm 1.0^\circ\text{C}$) and 15 min during Exercise 2 (Control: 33.3 ± 0.9 , Sago: $33.1 \pm 1.1^\circ\text{C}$).

There was no difference in the volume of water consumed (0.48 ± 0.26 L) or sweat lost (0.93 ± 0.23 L) during exercise between trials or exercise bouts (both $P > 0.05$), which resulted in a fluid deficit of $0.45 \pm$

Table 2. Carbohydrate and fat oxidation rates (g·min⁻¹) and RER during warm-up/steady-state exercise.

	Time (min)					
	Exercise 1			Exercise 2		
	5	10	15	5	10	15
Carbohydrate						
Control	1.1 ± 0.4	$2.2 \pm 0.7^*$	$2.6 \pm 0.5^*$	0.8 ± 0.7	$1.7 \pm 0.6^*$	$2.3 \pm 1.0^{*\#}$
Sago	1.2 ± 0.4	$2.2 \pm 0.7^*$	$2.3 \pm 1.1^*$	1.3 ± 0.5	1.6 ± 0.6	2.0 ± 0.6
Fat						
Control	0.6 ± 0.2	0.5 ± 0.3	0.6 ± 0.2	0.7 ± 0.4	0.6 ± 0.3	0.7 ± 0.5
Sago	0.6 ± 0.3	0.5 ± 0.3	0.7 ± 0.5	0.5 ± 0.3	0.7 ± 0.3	0.8 ± 0.3
RER						
Control	0.87 ± 0.03	0.88 ± 0.04	$0.91 \pm 0.05^*$	0.83 ± 0.03	0.85 ± 0.04	$0.87 \pm 0.04^*$
Sago	0.86 ± 0.05	$0.90 \pm 0.05^*$	$0.92 \pm 0.06^*$	0.87 ± 0.05	0.87 ± 0.04	$0.89 \pm 0.05^{*\#}$

Data are presented as mean \pm SE; $N = 8$; * denotes different to 5 min; # denotes different to 10 min.

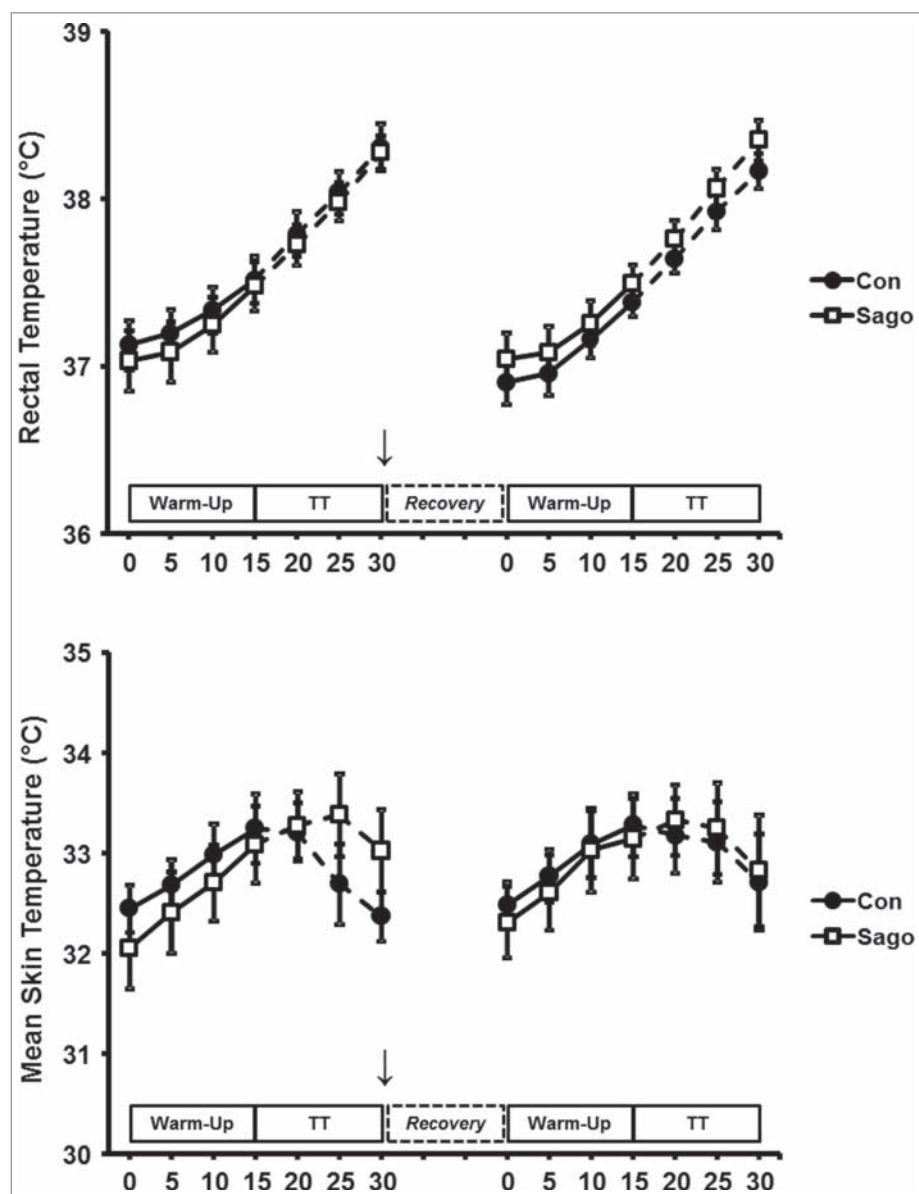


Figure 4. Rectal and mean skin temperatures during Exercise 1 and 2 for Control and Sago trials. TT: 15-min time-trial. Arrow indicates Sago ingestion. Data are expressed as mean \pm SE.

0.36 L (0.6 ± 0.4 % of body mass) by the end of exercise. This was, however, restored with *ad libitum* water consumption during the recovery period as pre-exercise body weight was similar ($P > 0.05$) between Exercise 1 and 2 (77.9 ± 15.0 vs. 78.4 ± 15.0 kg).

A main effect of time only ($P < 0.05$) was observed for plasma concentrations of sodium and potassium, such that concentrations increased from start (sodium: 133 ± 2 mmol·L⁻¹, potassium: 4.2 ± 0.1 mmol·L⁻¹) to end (sodium: 138 ± 2 mmol·L⁻¹, potassium: 4.9 ± 0.1 mmol·L⁻¹) of Exercise 1. Concentrations then decreased and returned to resting levels mid-way during the recovery period (sodium: 133

± 3 mmol·L⁻¹, potassium: 4.3 ± 0.1 mmol·L⁻¹) before increasing again from start (sodium: 134 ± 2 mmol·L⁻¹, potassium: 4.3 ± 0.1 mmol·L⁻¹) to end (sodium: 135 ± 2 mmol·L⁻¹, potassium: 4.7 ± 0.1 mmol·L⁻¹) of Exercise 2.

Cardiorespiratory responses

During the warm-up stages, participants were exercising at an intensity eliciting 40 ± 6 , 52 ± 6 and 63 ± 8 %VO_{2peak} ($P < 0.05$) with no effects of trial or exercise bout (both $P > 0.05$). Similarly, an effect of time but not trial or exercise bout was

observed for ventilation such that values increased ($P < 0.05$) from 38 ± 5 to 50 ± 5 and 59 ± 10 L·min⁻¹ during the warm-up stages, respectively. Finally, an effect of time, time*trial and exercise bout (all $P < 0.05$) was observed for heart rate such that values increased progressively throughout exercise from 71 ± 4 beats·min⁻¹ at rest to 143 ± 5 beats·min⁻¹ at the end of the warm-up and 174 ± 4 beats·min⁻¹ at the end of the time-trial, respectively, with values during Exercise 2 being 3 ± 1 beats·min⁻¹ higher than Exercise 1.

Discussion

This is the first study to determine whether sago, a starch staple found across Southeast Asia and prepared through boiling pearls into porridge, influences cycling performance after recovering from an exercise bout 2 h previous under conditions that simulate a tropical environment. The main finding is that feeding sago after such exercise maintains performance and thus enhances recovery compared to a control and this is likely due to the enhanced supply of exogenous carbohydrate. Furthermore, as this protocol under these conditions is highly reliable (a test-retest coefficient of variation of 2.3%), it can be said with confidence that the observed differences in time-trial performance in this study (3.9% for Control Exercise 2 vs. Exercise 1, and 3.7% for Sago Exercise 2 vs. Control Exercise 2) are real.

For athletes training or competing multiple times daily or on successive days (i.e., <24 h recovery between exercise bouts) the consensus recommendation for post-exercise CHO consumption (meal/snack) is 0.8–1.0 g·kg⁻¹ bodyweight·h⁻¹ within 30 minutes of exercise cessation.⁷ However, these recommendations are based on rates of skeletal muscle glycogen re-synthesis, not exercise performance, and do take into consideration the possible additive effect of a hot ambient environment.¹³ Therefore, when making recommendations based on performance of a repeated exercise bout (usually following a 4-h recovery within the literature) it appears that exercise performance is enhanced when a bolus of CHO (≥ 50 g) is consumed within 30 minutes of exercise cessation above rehydration alone (see refs. 6 and 26) but that a bolus²⁷ or serial feeding²⁸ with greater CHO content confers no additional performance benefit; additionally, the form (e.g., liquid vs. solid) that the CHO takes has no

effect.²⁹ Thus, our observations of an improved performance following recovery from a previous exercise bout having consumed ~60 g CHO supports previous studies and extends the available literature i) as the exercise was performed under conditions of humid heat, ii) was performed using a more face-valid self-paced (cf. fixed-intensity endurance) model, iii) the recovery period was only 2 h (compare 4 h), and iv) the CHO source was a palatable whole food (cf. CHO-electrolyte solution) that is easily sourced across Southeast Asia.

The mechanisms responsible for this performance maintenance (from Exercise 1) and improvement (from Control) with sago almost certainly concern an enhanced, or at least maintained, supply of CHO within the system as demonstrated by a higher than resting blood glucose for longer during recovery (Fig. 3 upper left panel), elevated blood glucose at the start of Exercise 2 compared to Exercise 1 (Fig. 3 upper right panel), and greater blood lactate upon completion of the time-trial with Sago (Fig. 3 lower left panel). Unfortunately, it was beyond the resources of the current study to be able to partition the source of this CHO i.e. exogenous vs. endogenous (hepatic vs. muscular vs. circulating), however this would be valuable in future investigations. That we observed no differences between exercise bouts or trials for substrate oxidation (Table 2) indicates no 'real' effect of repeated exercise or this CHO intervention, a lack of sensitivity with this measure or perhaps that an assumption of the methodology has been violated, for example gluconeogenesis (see refs.^{24,30}); the former may be as a result of the additional "stress" that ambient heat presents to hepatic glucose output (see refs.^{31,32}). It is also possible that improved CHO status has a central ergogenic effect that is not related to or detectable by whole body (indirect) calorimetry.

As far as the timing of ingestion is concerned, whether consumed before, during or following exercise, sago exerts no detrimental effects (beyond an elevated heart rate of ~5 beats·min⁻¹ likely related to the additional digestive load) but is associated with increased fluid retention and an attenuated rise of rectal temperature,¹⁶ and in the current study with an improved exercise performance.

The primary aim with the current study was to identify whether there was any benefit of supplementing sago during recovery from exercise, therefore we used a control condition where nothing was consumed

other than water to maintain similar levels of hydration. This was performed in order to compare these results with our previous.¹⁶ The next logical step would be to assess sago against a suitable and known whole-food (e.g. pasta) or CHO-electrolyte fluid to determine relative efficacy, as it has previously been shown that a solution high in waxy starch for 12h following glycogen-depleting exercise restores muscle glycogen and influences work completed in a subsequent 30-min time-trial similarly as equicaloric solutions with glucose or maltodextrin.³³ Including a whole-food or fluid placebo would also be worthwhile, as a placebo effect has been demonstrated previously with CHO,³⁴ something that cannot be determined in the present study.

In summary, the present study has shown that consuming 0.8 g/kg bodyweight cooked sago porridge upon completion of an initial exercise bout confers a performance advantage during a second exercise bout following a 2-h recovery when compared to a control condition.

Abbreviations

CHO	Carbohydrate
CV	Coefficient of variation
GI	Glycemic index
ICC	Intraclass correlation coefficient
RER	Respiratory exchange ratio
VO ₂	Volume of O ₂ consumed

Disclosure of potential conflicts of interest

No potential conflicts of interest were disclosed.

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