The Effect of Sweetness on the Efficacy of Carbohydrate Supplementation During Exercise in the Heat

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Catalogue Data

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Abstract/Résumé
The aim of the present study was to investigate potential mechanisms responsible for the improvement in prolonged exercise capacity in hot environments with exogenous carbohydrate. Eight endurance-trained men (V\textsubscript{O2} max 60.5 ± 2.4 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}, mean ± SE) cycled to exhaustion on three occasions at 60% V\textsubscript{O2} max at an ambient temperature of 35 °C. They ingested either a sweet 6.4% carbohydrate solution (SC), a nonsweet 6.4% carbohydrate solution (NSC), or water (W). Exercise capacity was significantly increased with SC and NSC compared to W, the improvements corresponding to 15.8% and 11.8%, respectively. No difference in exercise capacity was seen between SC and NSC solutions. Plasma glucose concentrations were higher during the SC and NSC trials compared to W, significantly so at 10 min and at fatigue. Rates of carbohydrate oxidation were higher in the SC and NSC trials, although the rates never declined below 2.1 ± 0.2 g·min\textsuperscript{-1} in the W trial. There was no difference in the rate of rise of rectal temperature between trials, but there was a trend for subjects to fatigue at higher temperatures during the two carbohydrate trials. In conclusion, exogenous carbohydrate, independent of sweetness, improves exercise capacity in the heat compared to water alone.

Cette étude se propose d’analyser l’impact de l’apport exogène de sucre sur les mécanismes potentiels d’amélioration de la capacité de travail physique par temps chaud. Huit hommes...
entraînés à l'endurance ($\text{VO}_{2\text{max}}$ 60,5 ± 2,4 ml·kg$^{-1}$·min$^{-1}$) participent à trois épreuves de pédalage jusqu'à épuisement; l'intensité de l’effort est fixée à 60% du $\text{VO}_{2\text{max}}$ dans une pièce maintenue à 35 °C. Les sujets ont pris soit une solution d’hydrates de carbone sucrés à 6,4% (SC), soit une solution d’hydrates de carbone non sucrés (NSC), soit de l’eau (W). Comparativement à W, la capacité de travail est accrue significativement de 15,8% et de 11,8% dans les deux conditions SC et NSC, respectivement. Les différences de capacité entre les conditions SC et NSC ne sont pas significatives. Comparativement à W, les concentrations plasmatiques de glucose sont plus fortes dans les conditions SC et NSC; les différences sont significatives à la 10e min et au point de fatigue. Les taux d’oxydation des hydrates de carbone sont plus importants dans les conditions SC et NSC; dans la condition W, le taux ne baisse pas sous le niveau de 2,1 ± 0,2 g·min$^{-1}$. Dans les trois conditions, le taux d’augmentation de la température rectale ne diffère pas, mais les sujets semblaient se fatiguer davantage à plus haute température dans les conditions SC et NSC. En conclusion, un apport exogène d’hydrates de carbone, sucrés ou non, améliore la capacité de travail par temps chaud comparativement à l’eau seule.

Introduction

During prolonged exercise in cool or temperate conditions, carbohydrate (CHO) supplementation has been shown to delay the onset of fatigue and improve exercise capacity and performance (Coyle et al., 1986). The maintenance of high rates of carbohydrate oxidation during the latter stages of the exercise has been suggested as being the mechanism responsible for this improvement (Coyle et al., 1986). During exercise of a similar intensity performed in environments with a high ambient temperature, exogenous CHO has also been reported to improve exercise capacity and performance (Below et al., 1995; Carter et al., 2003; Millard-Stafford et al., 1992). However, several studies have reported no additional benefit from a CHO solution compared to water during exercise in the heat (Davis et al., 1988a; Febbraio et al., 1996; Millard-Stafford et al., 1990).

The mechanism responsible for the improvement in exercise performance with CHO supplementation during exercise in the heat is unclear. As previously mentioned, during prolonged exercise in temperate conditions, exogenous CHO maintains blood glucose concentrations and CHO oxidation rates in the latter stages of exercise (Coyle et al., 1986). However, this mechanism has received limited support in those studies reporting a beneficial effect of exogenous CHO during exercise in the heat (Davis et al., 1988b; Galloway and Maughan, 2000; Millard-Stafford et al., 1992; Murray et al., 1987; 1989) or exercise of relatively short duration (Jeukendrup et al., 1997).

More recently an alternative explanation has been proposed concerning the effects of carbohydrate (Carter et al., 2004a; 2004b; Jeukendrup et al., 1997). It has been suggested that carbohydrate may have a more central effect in the brain that may affect performance or the perception of effort, independent of substrate delivery. Such an effect could be mediated through receptors for sweetness or specific receptors for CHO that are located in the oral cavity or elsewhere in the gastrointestinal tract (Carter et al., in press-b).

In view of the above, the aim of the present study was to investigate exogenous CHO and prolonged endurance exercise in the heat. The present study involved
three different drinks: plain water, a nonsweet CHO solution, and an artificially sweetened CHO. The nonsweet CHO solution was included to determine the additional benefit, if any, of sweetness per se on endurance capacity in the heat. Previous studies which have controlled CHO and PLA solutions for sweetness have reported an improvement in exercise performance or capacity with only exogenous CHO (Below et al., 1995; Coyle et al., 1986; Jeukendrup et al., 1997). Therefore the purpose of the work described here was to test the idea that feeding CHO during exercise improves endurance capacity in the heat, and to confirm our hypothesis that there is no additional benefit of sweet CHO compared to nonsweet CHO.

Methods

SUBJECTS

Eight endurance-trained male volunteers gave their informed consent to participate in the study, which was approved by the local ethics committee. Their mean age, height, weight, and VO$_2$ max were 24 ± 4 yrs, 181 ± 7 cm, 76 ± 8 kg, and 60.5 ± 6.7 ml·kg$^{-1}$·min$^{-1}$ (mean ± SD). All subjects had previously been involved in studies involving cycle ergometry at this intensity and in similar conditions and were fully familiar with the experimental procedures.

EXPERIMENTAL DESIGN

All exercise tests were carried out on an electrically braked cycle ergometer (Lode Excalibur, Gröningen, The Netherlands) set in the cadence-independent mode. The protocol consisted of 5 visits. Visit 1 involved an incremental exercise test to determine maximum oxygen uptake (VO$_2$ max), peak power output (PPO), and the workload needed to elicit 55% PPO. Visits 2–5 involved exercising to volitional fatigue in a heat chamber maintained at 35 °C and a relative humidity of 30%. Visit 2 was an habituation ride; subjects exercised at 55% PPO to exhaustion and consumed water. During the remaining 3 visits they exercised at 55% PPO, consuming either a sweetened 6.4% maltodextrin solution (SC), a nonsweet 6.4% maltodextrin solution (NSC), or water (W). The sweetness was achieved with a 0.2% aspartame solution after a pilot study involving a range of aspartame concentrations determined 0.2% to be the best concentration. The subjects were told they would receive 3 drinks differing in composition and taste: some would contain CHO and some would not; some would taste sweet and some would not. Furthermore, they were told that all 3 drinks had previously been shown to be beneficial to performance during exercise in the heat. The study was carried out in a randomised blind fashion, with each visit separated by 7 days.

VISIT 1

Subjects underwent an incremental exercise test to volitional fatigue at a self-selected cadence on a cycle ergometer. The appropriate seat position, handlebar height, and orientation were used during testing and replicated in each subsequent visit. The initial workload was 95 watts and was increased by 35 watts every 3 minutes until fatigue. Expired gas was recorded continuously throughout (Oxycon Pro, Jaeger, Germany), as was heart rate (Polar Vantage NV, Polar Electro, Oy, Finland).
Subjects arrived at the laboratory in the morning following an overnight fast, having abstained from exercise, alcohol, caffeine, and tobacco for the previous 24 hrs. To minimise differences in starting muscle glycogen concentrations, each subject was asked to record his diet in the 24-hr period prior to the second visit. This record was copied and returned to him with the instruction to follow the same diet before each subsequent visit. Upon arrival, each subject inserted a rectal thermister 10 cm beyond the anal sphincter before being weighed nude. He then rested in a supine position, during which time a cannula (20 G Venflon) was inserted into a superficial forearm vein and kept patent with saline (Baxter). Following this, a baseline blood sample was taken with the subject in a seated position, skin thermisters were attached, and the subject drank a bolus (8 ml·kg\(^{-1}\)) of either W, SC, or NSC. He was then transferred to the heat chamber and cycled to exhaustion at the prescribed workload. There was a 5-min interval between bolus consumption and the start of exercise.

Subjects ingested fluid at a rate of 3 ml·kg\(^{-1}\) every 15 min during the ride. They wore lycra shorts and clip-in shoes. If a subject needed to urinate during the test, he stopped pedaling and passed urine into a container while remaining on the ergometer. A fan was used to circulate the air in the chamber; the fan was set at the same position and speed between trials. Following exercise, the subject was weighed nude to allow for estimation of sweat loss after correcting for urinary loss (collected throughout the ride), respiratory changes (Snellen, 1966), metabolic changes (Mitchell et al., 1972), and fluid intake.

**Blood Collection.** Venous blood samples (8 ml) were collected into EDTA tubes at rest, at 10-min intervals throughout the ride, and at the time of fatigue. During blood collection each subject’s arm was relaxed and positioned flat across the handlebars. One milliliter of blood was separated and analysed for haemoglobin and haematocrit. The remaining whole blood was centrifuged within 10 min of collection and the plasma was separated for determination of lactate and glucose concentrations. Samples were stored at \(-15\, ^\circ\text{C}\) and all assays were completed, in duplicate, within 6 weeks of the exercise.

**Blood Analyses.** Plasma lactate concentrations were determined by spectrophotometry using a quantitative enzymatic (lactate oxidase and peroxidase) method (Sigma Kits, Gillingham, Dorset, U.K.). Plasma glucose concentrations were determined by spectrophotometry using a quantitative enzymatic (hexokinase) method (Sigma Kits). Whole blood was used for the quantitative, colorimetric determination of haemoglobin (Sigma Kits), and microcentrifugation was used to determine haematocrit. Percent changes in plasma volume were calculated from haemoglobin and haematocrit values (Dill and Costill, 1974).

**Gas Analysis.** Expired air was collected for 5 minutes at 15, 45, and 75 min using Douglas bags. The volume of expired air in the bags was measured with a dry gas meter (Harvard, Kent, U.K.). The O\(_2\) and CO\(_2\) concentrations were determined using Servomex 1400B4 analysers (Sussex, U.K.). CHO oxidation rates were calculated from indirect calorimetry (Frayn, 1983).

**Temperature Measurement.** Ambient temperature during each ride was measured using a wet and dry bulb mercury thermometer (Brannan, Cumbria, U.K.)
and relative humidity was calculated from the wet and dry bulb thermometer differential. Thermistors were placed on four skin locations from which mean skin temperature ($T_{\text{msk}}$) was calculated (Nielsen and Nielsen, 1984). The rectal and skin thermistors were connected to a Squirrel data logger (Grant Instruments, Cambridge, U.K.) and values were recorded every 2.5 min and at fatigue.

**Perceived Exertion, Heart Rate, and Thermal Discomfort.** Rating of perceived exertion (RPE) was recorded every 10 minutes using the 6–20-point Borg scale (Borg, 1982), and thermal discomfort was recorded simultaneously using the Frank scale (Frank et al., 1999). Heart rate was measured continuously by telemetry (Polar Vantage NV).

**Data and Statistical Analysis.** Data are reported as mean and standard error (mean ± SEM) unless otherwise stated. A two-way repeated-measures analysis of variance was used to compare the data from the W, SC, and NSC trials. Specific differences were determined using a Tukey’s HSD post hoc test.

**Results**

All 8 subjects successfully completed the study. Volitional fatigue was the reason for termination of exercise in all trials. Seven subjects were able to exercise longer with SC than with W, and 6 subjects exercised longer with NSC than with W. Time to fatigue in the W trial was $128.0 \pm 14.1$ min compared to $152.0 \pm 18.3$ min in the SC trial and $145.1 \pm 12.1$ min in the NSC trial, an average improvement of $15.8 \pm 2.9\%$ and $11.8 \pm 1.4\%$, respectively. In both the SC and NSC trials there was a significant increase in exercise capacity compared to the W trial, $p < 0.05$; no differences were found between SC and NSC trials.

**CADENCE, RPE, AND THERMAL COMFORT**

Cadence was maintained at a steady rate up to 80 minutes in all 3 trials: $83 \pm 5$ rpm, $84 \pm 4$ rpm, and $81 \pm 5$ rpm for W, SC, and NSC, respectively, before declining to values at fatigue of $71 \pm 4$ rpm, $69 \pm 4$ rpm, and $69 \pm 4$ rpm for W, SC, and NSC respectively. No significant differences were observed between trials for cadence at any point, $p > 0.05$. RPE increased throughout the exercise in all 3 trials (Figure 1). There was no difference in values for RPE between the 3 trials at any time point, $p > 0.05$. Rating of thermal discomfort rose steadily during exercise, with no difference between W, SC, and NSC trials, $p > 0.05$.

**PHYSIOLOGICAL AND TEMPERATURE MEASUREMENTS**

CHO supplementation, either SC or NSC, had no effect on heart rate response to exercise, $p > 0.05$, which increased gradually with exercise during all 3 trials (Figure 1). Measures of hydration status were largely unaffected by drink condition. Mean sweat rate was $1301 \pm 56$ ml·hr$^{-1}$, $1250 \pm 72$ ml·hr$^{-1}$, and $1277 \pm 94$ ml·hr$^{-1}$ for W, SC, and NSC, respectively, $p > 0.05$. Mean urine production and mean weight loss were similar between trials. Urine output was $224 \pm 109$ ml, $273 \pm 132$ ml, and $246 \pm 103$ ml for W, SC, and NSC, respectively, $p > 0.05$. Values for weight loss were $513 \pm 363$ g, $800 \pm 408$ g, and $550 \pm 282$ g for W, SC, and NSC,
respectively, \( p > 0.05 \). The rate of fluid ingestion did not differ between trials, \( p > 0.05 \), and equaled 1232 ± 176 ml, 1185 ± 154 ml, and 1185 ± 1153 ml for W, SC, and NSC, respectively. Plasma volume decreased in all trials (Figure 2), but this fall was not different between trials, \( p > 0.05 \).

Plasma glucose concentrations were the same before exercise in the 3 trials (Figure 3). There was a trend for plasma glucose concentrations to be higher in both CHO trials compared to W throughout the exercise period. However, values in the SC and NSC differed significantly from W only at 10 min and at fatigue (Figure 3, \( p < 0.05 \)). Plasma lactate concentrations were similar between conditions at rest and did not differ significantly between trials during exercise (Figure 3, \( p > 0.05 \)). \( \dot{V}O_2 \) remained steady during exercise and was similar between drink conditions, \( p > 0.05 \). The work rate corresponded to 57.5 ± 3.6% \( \dot{V}O_2 \)\text{max} in the W trial, 57.2 ± 2.4% \( \dot{V}O_2 \)\text{max} in the SC, and 58.0 ± 2.3% \( \dot{V}O_2 \)\text{max} in the NSC. There was a trend for CHO oxidation rates to be higher during both CHO trials compared to W. This trend was significant after 45 and 75 min (Figure 4, \( p < 0.05 \)). There was no difference between CHO oxidation rates during the SC and NSC trials.
Figure 2. Plasma volume change during all trials ($N = 8$).

Figure 3. Plasma lactate and plasma glucose concentrations during all trials. *Significant difference between SC/NSC and W at that time point, $p < 0.05$ ($N = 8$).
T_rec and T_msk responses were similar between W, SC, and NSC at all time points, \( p > 0.05 \). T_msk increased initially in all 3 trials before remaining steady from 10 min until fatigue. T_rec increased gradually throughout all 3 trials (Figure 5) before reaching values at fatigue of 38.7 ± 0.1°C in the W trial, and 39.0 ± 0.2 °C in both the SC and NSC trials.

**Discussion**

In the present study, the delay in time to fatigue with both SC and NSC compared to water provides further support for the benefit of CHO feeding during exercise in the heat. Although not unanimous in their findings, the majority of previous studies examining the efficacy of CHO supplementation immediately before and dur-
ing prolonged exercise in the heat have reported a beneficial effect. These benefits correspond to improvements in both exercise capacity (Carter et al., 2003; Galloway and Maughan, 2000) and exercise performance (Davis et al., 1988b; Millard-Stafford et al., 1992; Murray et al., 1987).

There was no additional benefit reported in the SC compared to the NSC trial. Therefore the improvement in exercise capacity appears to be related to the CHO content per se and not to the sweetness of the ingested solution. The report of no additional benefit of sweetness is in agreement with previous studies which have matched the CHO and PLA solutions for sweetness, flavour, and color, and in which an improvement in exercise performance with only exogenous CHO was demonstrated (Below et al., 1995; Coyle et al., 1986; Jeukendrup et al., 1997).

The improvement in exercise capacity of ~16% for the SC trial and ~12% for the NSC trial in the present study was similar to the improvement of ~15% reported in a previous study in our laboratory (Carter et al., 2003). In the present study, which examined moderate intensity exercise in the heat with CHO supplementation, the mechanism responsible for the improvement was unclear. This is in accord with a number of other studies (Galloway and Maughan, 2000; Murray et al., 1987; 1989). A mechanism that receives considerable interest, and which has been shown to be important in delaying volitional fatigue during prolonged exercise in temperate conditions, is the maintenance in plasma glucose concentrations and of CHO oxidation rates during the latter stages of exercise (Coyle et al., 1986).

Some support for this mechanism is provided in the present study. There was a trend for a higher plasma glucose concentration throughout exercise in both CHO trials compared to water, which was significant after 10 min and at fatigue, and a higher rate of CHO oxidation was found in the CHO trials compared to the water trial after 45 and 75 min (Figure 4). Previous studies have reported similar findings concerning glucose concentrations and oxidation rates during exercise in the heat with CHO feeding, and these studies have cited increased substrate delivery as the mechanism responsible for the improvement in exercise performance (Davis et al., 1988b; Millard-Stafford et al., 1992).

In the present study, if exogenous CHO exerted its beneficial effect through an enhancement of CHO oxidation rates, then plasma glucose concentrations, at least in the water trial, would be expected to approach hypoglycaemia at fatigue. During exercise in temperate conditions, plasma glucose concentrations at fatigue have been reported to be ≤3 mmol·L\(^{-1}\) (Coyle et al., 1986). However, in the present study plasma glucose concentrations at fatigue in all 3 trials were unlikely to be limiting (≥4 mmol·L\(^{-1}\), Figure 3). This concentration is similar to that of ~4-6 mmol·L\(^{-1}\) at fatigue during the PLA trials of previous studies on exercise capacity in the heat with CHO supplementation (Carter et al., 2003; Febbraio et al., 1996; Galloway and Maughan, 2000). Since plasma glucose in the W trial did not fall to concentrations that would be limiting to exercise, and since CHO oxidation was not impaired, it seems unlikely that endogenous CHO supply to the working muscles was ever limiting. According to the catastrophe models, hypoglycaemia should be the factor terminating exercise, but in this study exercise was terminated without catastrophic limitation, which is more in keeping with the central governor theory. Similar conclusions have been reported in other studies (Nielsen et al., 1990; Parkin et al., 1999).
As far as can be determined from the methods used, there were no obvious indications of peripheral fatigue nor homeostatic failure during any of the trials in the present study suggesting that termination of exercise was dictated by a central mechanism. Together with the absence of hypoglycaemia, plasma lactate concentrations during exercise were low and constant throughout the exercise (Figure 3). A reduction in blood flow to the active muscles has been previously linked to fatigue during exercise in the heat, although this only occurs in combination with dehydration (Gonzalez-Alonso et al., 1998). In the present study there was no indication of dehydration in either the CHO or water trials, as assessed by plasma volume change (Figure 2), heart rate response (Figure 1), or rate of rise of $T_{rec}$ (Figure 5). Increased cardiovascular strain is recognised as an important factor during exercise in the heat (Rowell, 1983), but there was no evidence that it was either the cause of fatigue or that it differed between exercise trials. Heart rate (Figure 1), ventilation, and $V_{O_2}$ never reached limiting values in any condition. Thus it appears that the subjects stopped exercising while in a homeostatic state in all 3 trials in the present study.

It has been suggested that fatigue, and therefore performance, is under the influence of a central neural governor that controls the proportion of active muscle recruited during exercise (Kayser, 2003; Noakes et al., 2004). This “central governor” is thought to act as a safety guard, reducing or terminating physiological activity before serious physiological damage can occur (e.g., myocardial ischaemia, cerebral hypoxia, hypoglycaemic coma, or hyperthermia). Concurrent with this is the observation that muscle ATP stores are protected during exercise so that muscle rigor never occurs (Fitts, 1994; Noakes et al., 2004). Toward the latter stages of strenuous exercise the motor cortex reduces skeletal muscle activation, causing the work output to fall and heralding the onset of fatigue (Noakes and St Clair Gibson, 2004; Noakes et al., 2004). Therefore, the improvement in exercise capacity with CHO feeding in the present study may be explained by an extension of the homeostatic state in the CHO trials.

The RPE data between trials was similar at the end of exercise, suggesting that fatigue occurred at the same RPE regardless of the metabolic state. However, the duration of exercise was significantly longer in both CHO trials. This implies that the rate of rise of RPE during exercise was attenuated by CHO feeding, a finding that has been reported by others and attributed to increased blood glucose concentrations and better maintenance of CHO oxidation rates (Coyle et al., 1986; Utter et al., 1997; 1999).

Receptors for glucose are well known to exist throughout the body, including the brain (Ritter, 2000). Thus it is possible that glucose in the circulation was acting as a signal, as well as a substrate, providing feedback to the brain concerning the athlete’s physiological state. In the CHO trials at the point where volitional fatigue occurred in the water trial, the brain permitted the continuation of exercise due to a more favourable metabolic state, as reflected by higher blood glucose concentrations and rates of CHO oxidation. A recent study from our lab, however, in which glucose was infused during exercise suggests that high blood glucose concentrations and higher rates of glucose uptake do not result in better performance (Carter et al., in press-a). So we have demonstrated that glucose ingestion improves performance or exercise capacity (Carter et al., 2003; Jeukendrup et al.,
1997), but glucose infusion does not (Carter et al., in press-a). So the effect of CHO may be related to taste perception or CHO binding to a receptor that sends a signal to the brain.

From the present study we would have to conclude that sweetness is not an important factor, as a nonsweet CHO still resulted in improved exercise capacity. Interestingly, in another study in which subjects rinsed their mouth with a CHO solution (but did not swallow it), we found improved performance (Carter et al., in press-b). This suggests there are receptors in the oral cavity which, once stimulated by CHO, send a signal to the brain that may reduce the perception of effort. More research is needed to investigate the exact mechanisms.

An increased body core temperature appears to be an important factor responsible for fatigue during exercise in the heat (Bruck and Olschewski, 1987; Gonzalez-Alonso et al., 1999; Nielsen et al., 1993). In the present study, \( T_{rec} \) values at fatigue, although not significant, were 0.3 °C higher in the CHO trials compared to the water trial. This is in exact agreement with \( T_{rec} \) values at fatigue in our previous study (Carter et al., 2003). Whether a rising core temperature was involved in the decision to stop exercising in the present study, and by what means, and whether CHO ingestion improved the tolerance to this rising core temperature are questions beyond the scope of the present study. However, it should be pointed out that core temperature values at fatigue during all 3 trials (\( \leq 39 \) °C) were somewhat lower than those (\( \geq 39.5 \) °C) reported in previous studies citing a high core temperature as responsible for fatigue (Gonzalez-Alonso et al., 1999; Nielsen et al., 1993).

In conclusion, it is clear that CHO supplementation immediately before and during exercise in the heat delays the onset of fatigue compared to the ingestion of water alone. The mechanism responsible for this improvement is unclear from the present results, but is independent of sweetness. In combination with the results of other studies in our laboratory, these results suggest there are receptors in the oral cavity which, once stimulated by CHO (but not sweetness), send a signal to the brain that may reduce the perception of effort. The present study, along with other studies from our lab, provides preliminary evidence that CHO ingestion not only has a metabolic effect during exercise but also a central effect in the brain.

References


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