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Impact of carbohydrate nutrition
on exercise metabolism and performance

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Abstract Carbohydrate (CHO) ingestion before and during exercise has consistently been reported to increase endurance exercise capacity/performance but the mechanisms responsible and optimal dose required are still debated. Feeding CHO is believed to spare muscle glycogen, spare liver glycogen, have central neural actions, and peripheral neural effects. A combination of these mechanisms is likely and the nature of the exercise performed is key when interpreting these data. Research on the optimal dose of CHO to improve performance over a range of exercise durations and intensities has been a recent focus. Optimal doses suggested from these studies cover a range (30-80 g•h-1) that likely reflects exercise task, training status, and/or individual variation in response.

INTRODUCTION AND HISTORICAL BACKGROUND

After almost 100 years of research on CHO metabolism and exercise there are still big questions over mechanisms of action and optimal dose during exercise to maximise performance (1, 2). The purpose of the present review is to highlight these questions and cover key elements for consideration in future research and for applied sport nutrition practice. To achieve this we consider the historical background, effects of CHO on exercise performance/capacity, mechanisms of action, carbohydrate dose, and practical recommendations.

Dietary carbohydrates (CHO) were first recognised as an important fuel for muscle during moderate to high intensity exercise (defined for the purpose of the review as 50 – 90% of maximal aerobic capacity) by Krogh and Lindhard (3). They observed that participants felt tired and exercise capacity was reduced following a short term high fat diet. However, three days of a high CHO diet reversed these symptoms of fatigue (3). In addition, other authors reported that blood glucose concentration was lower in the majority of competitors immediately following the 1924 Boston Marathon (4, 5). The following year blood glucose concentration immediately after a marathon was higher in participants who had consumed confectionary during the race compared to those who consumed nothing (5). Subsequently, when fatigue was associated with low blood glucose concentration feeding a high CHO diet could extend exercise capacity (206 mins vs. 81 mins) (6). The findings led to greater consideration of the potential ergogenic impact that nutrition could have during exercise. These early observations demonstrated that CHO ingestion had an important role in regulating blood glucose and extending exercise capacity. However, the mechanisms to explain these observations with CHO feeding and elevated blood glucose had not yet been addressed.

The skeletal muscle biopsy technique, developed by the French neurologist Guillaume-Benjamin-Amand Duchenne (7), was reintroduced in the mid 1960's providing insight into muscle glycogen usage during exercise. Bergstrom and Hultman (8) demonstrated that skeletal muscle glycogen content could be increased following muscle glycogen depleting exercise if a high CHO diet was consumed. Furthermore, increasing the glycogen content of muscle improved exercise capacity above that obtained with normal muscle glycogen content. The application of this new insight was demonstrated when the consumption of a high CHO diet improved 30 km running time over that obtained on a normal diet (9). Similarly, football players covered less distance, ran slower, and walked more often during the second half of a match when starting the game with reduced muscle glycogen stores (10). It was also noted during these investigations that the consumption of carbohydrate elevated blood glucose concentration whilst sparing muscle glycogen. As such, these observations supported the notion that higher muscle glycogen content, as a result of CHO consumption in the days preceding exercise, improves the capacity to perform moderate to high intensity exercise.

In summary this early work clearly defined an association between muscle glycogen and exercise capacity. The ability to enhance muscle glycogen content via nutritional manipulation is now well established (11) and is still considered to be crucial for optimal performances (12, 13). However, CHO ingestion immediately before, and during exercise is also considered to enhance performance without any initial difference in muscle glycogen content. The remainder of the present review will focus on quantifying the size of this effect and exploring mechanisms of action and implications for applied practice.

CARBOHYDRATE AND ENDURANCE EXERCISE PERFORMANCE / CAPACITY

The ergogenic effect of CHO feeding during moderate to high-intensity exercise has been extensively investigated and summarised in 3 recent meta-analyses (14-16) that are worth consideration. The first meta-analysis investigated the duration of exercise where CHO was having the largest effect. The analysis used Cohen's effect sizes as a means of inferring the magnitude of effect and can be interpreted as; 0.2 small; 0.5 moderate; 0.8 large effects respectively (14). The authors reported that in the 72 studies examined, CHO ingestion had a moderate positive effect size (ES) of ~0.42 on exercise performance / capacity. Feeding CHO during exercise bouts of > 2 h had a significantly greater effect (ES ~0.5) than on those between 30 min and 2 h (ES ~0.35). Interestingly, the effect size was similar in running and cycling (ES ~0.4), and with feeding glucose alone or mixed monomers (ES ~ 0.4). CHO feeding during exercise has been shown to increase power output during a 40km time trial (TT) lasting ~1 h (17), during the latter stages of a 60min TT (18), during an all-out 15 min TT task following a 45 min submaximal ride (19), and during Wingate sprint performance following a 50 min sub maximal ride (80% VO_{2max}). However, Mitchell et al (20) reported no difference in 10 km running time when participants ingested a range of CHO doses (0, 34, 39, 50 $g.h^{-1}$). Equally it has been reported that time to exhaustion (TTE) at 80% VO_{2max} did not change whether CHO or water was consumed prior to or during exercise (21). Interestingly, these two studies both examined shorter exercise durations (20-30 mins) than other studies included in the analysis. Consequently, it is generally considered that exercise tasks are required to be > 40 min, irrespective of CHO type or dose, to achieve a moderate beneficial effect on performance / exercise capacity. Nonetheless, new evidence indicates that acute CHO provision may have a beneficial effect on very short task durations (i.e., <10 min) suggesting a need for further study within this context (22).

In the second meta-analysis fifty studies were grouped to illuminate the effects of the exercise protocol, pre-exercise nutritional status (i.e., fed or overnight-fasted), training status, and gender on the efficacy of CHO feeding during exercise (15). Four exercise categories were considered: time to exhaustion (TTE), time trial (TT), submaximal preload with TTE (submax+TTE) and submaximal preload with TT (submax+TT). The inclusion criteria for studies with multiple CHO trials was limited to the single highest glucose concentration trial with ingestion rates between 30 and 80 $g.h^{-1}$ and solution concentration not exceeding 8%. The results from this analysis are shown in Table 1.

The results from this meta-analysis imply that CHO intake in line with current intake guidelines (30 to 80 $g \cdot h^{-1}$) during exercise ≥ 1 h, moderately improves exercise performance/ capacity irrespective of the exercise protocol used. Additionally, the pre exercise nutritional status of participants (i.e. fasted >8 h, and non-fasted <6 h) appears to have no effect on the subsequent exercise performance / capacity achieved. However, the scope of this meta-analysis was limited and did not comprehensively address all feeding strategies employed in the wider literature, potentially leading to an over-simplified view.

To provide greater scope the third meta-analysis we will consider pooled 88 randomised cross over studies investigating the effects of CHO consumption during exercise (16). Of the studies included 83% used cycling as the mode of exercise and all studies measured exercise capacity with time to exhaustion (mean duration 106 min) and exercise performance via time trial (mean duration 47 min). The mean percentage change in capacity / performance from control conditions was deduced by compiling the reported additive performance effects of components included in the model. However, a -2% (impairment) to a +6% (improvement) was reported across all studies included in the meta-analysis. Interestingly, for some individuals consuming CHO caused a reduction in exercise performance / capacity. The range in performance outcome is not surprising considering the variety of feeding strategies, exercise interventions, and participant characteristics included in the analysis. Nonetheless, some recent studies report considerable individual variation in performance measures even when the same exercise intervention is used to determine the effect of CHO feeding. Taken together the variability reported highlights the importance of acknowledging individual responses to CHO intake during exercise (23, 24). Overall, there seems to be a clear moderate positive effect of CHO feeding on exercise performance/capacity throughout the three meta-analyses considered. Several mechanisms have been proposed and investigated to explain the ergogenic action of CHO feeding during sustained moderate-high intensity exercise.

Mode of Exercise	Number of studies	Grouped mean Cohen's ES*	95% CI of ES, (p-value)	Weighted mean improvement (%)
TTE	19	0.47	0.32 – 0.62, p < 0.01	15.1
Submax+TTE	3	0.44	0.08 – 0.8, p = 0.017	54.2
TT	11	0.30	0.07 – 0.53, p=0.011	2.0
Submax+TT	17	0.53	0.37 – 0.69, p < 0.001	7.5

Table 1. Number of studies, effect size (ES) outcomes, and mean percentage improvement in performance from placebo/control as presented in the meta-analysis from Temesi et al (2011). *0.2 small; 0.5 moderate; 0.8 large effect.

MECHANISMS OF ACTION

Muscle glycogen sparing

As indicated in the Introduction fatigue has frequently been associated with low muscle glycogen (8, 25, 26). Nutritional strategies used to increase muscle glycogen stores prior to exercise can lead to enhanced performance and an increase in exercise capacity (27, 28). Similarly, nutritional strategies utilising CHO during exercise may also have a positive effect on endurance performance/ capacity by providing an alternative fuel source for muscle energy metabolism and therefore limiting the amount of muscle glycogen utilised (26-31). An overview of the studies investigating the effect of CHO provision before, and during, exercise on working muscle glycogen utilisation (measured from skeletal muscle biopsy samples) is shown in Table 2. 50% of the 16 studies listed report a sparing of muscle glycogen with CHO supplementation during exercise. Further, some reports indicate muscle glycogen sparing may be fibre type specific (26, 27) although conflicting evidence has emerged (26). The variability in outcomes may reflect differences in exercise protocol (e.g., continuous vs intermittent running and cycling), location of muscle biopsy, method of analysis (e.g., biochemical vs. histochemical, mixed muscle vs. fibre

Study	Exercise mode and duration	Intensity	CHO intervention	MG sparing
Glycogen Sparing				
Berstrom and Hultman (1967)(8)	Running, 1 h	950 kpm/min	Glucose infusion (3.5 g.min^{-1})	20% total reduction
Bjorkman et al (1984)(28)	Cycling. TTE	68% VO_{2max}	70 g·h^{-1} glucose	Rate of utilisation decreased from 2.3 to 1.3 mmol kg d.w.$^{-1}$ min^{-1}
Errikson et al (1987)(32)	Cycling, 90 min	65-70% VO_{2max}	1 g·kg BM	31.8% reduction
Hargreaves et al (1984)(30)	Cycling, 4 h	50% VO_{2max} + intermittent sprints/30 min.	43 g·h^{-1} solid CHO + 400ml H_2O	26% total reduction
Stellingwerf et al (2007)(26)	Cycling, 3 h	63% VO_{2max}	0.7 g·CHO·h^{-1}	19% total reduction
Tsintzas et al (1995)(27)	Running, 1 h	70% VO_{2max}	5.5% mixed CHO	28% total reduction
Tsintzas et al (2001)(31)	Running TTE (matched)	70% VO_{2max}	8 ml·kg BM of 5.5% CHO solution	56% reduction in Type I fibres
No Glycogen Sparing				
Arkinstall et al (2001)(33)	Running and cycling 1 h	Lactate threshold	8 ml·kg BM pre ex, 2 ml·kg BM every 20 min 6.4% solution	No difference
Chryssanthopoulos et al (2002)(34)	Running 1 h	70% VO_{2max}	Pre ex meal and 46 g CHO during	No difference
Coyle et al (1986)(35)	Cycling TTE	71% VO_{2max}	2.0 g·kg BM @ 20min, 4 g·kg.BM each 20 min thereafter.	No difference
Coyle et al (1991)(36)	Cycling 2 h	73% VO_{2max}	Hyperglycemic infusion clamp (~10 mmol)	No difference
Febbraio et al (1996)(37)	Cycling 2 h + 15 min performance	70% Peak O_2 consumption	Pre ex CHO meal	No difference
Febbraio (2000)(38)	Cycling 2 h + 15 min performance	70% Peak O_2 consumption	Pre ex CHO meal	No difference
Flynn et al (1987)(39)	Cycling 2 h	Complete as much work as possible	Mixed CHO/concentration	No difference
Hargreaves et al (1988)(40)	Cycling 2 h	70% VO_{2max}	30 g CHO pre and every 30 min	No difference
Mitchell et al (1989)(41)	Cycling 2 h	105min @ 70% VO_{2max}	6, 12 and 18 g·100ml	No difference

Table 2. Summary of studies reporting muscle glycogen usage and carbohydrate ingestion during exercise

specific), and inherent variability in the measurement of muscle glycogen. Many of the studies which report muscle glycogen sparing also report an increase in circulating plasma glucose concentration as a major factor in explaining this phenomenon. It is possible that circulating plasma glucose has a role to play but the mechanisms of action continue to be elusive. As such, muscle glycogen sparing can occur in some contexts but this is not always observed leading to the suggestion that there may be other mechanisms to explain the positive effects of CHO feeding.

Role of liver glycogen

Moderate to high rates of CHO intake during exercise have been reported to reduce endogenous hepatic glucose output to basal levels (42) or completely inhibit endogenous hepatic glucose output altogether (43). Thus, exogenous CHO oxidation could replace hepatic glycogen as a fuel source. Coyle et al (1986) reported an enhanced maintenance of blood glucose during prolonged exercise with the ingestion of CHO. The authors attributed an enhanced blood glucose concentration with elevated rates of CHO oxidation throughout the bout. Consequently, feeding CHO should allow more liver glycogen to be available towards the latter stages of competitive exercise when exercise intensity, and the requirement for CHO oxidation, are both increased. Casey et al (44) investigated liver glycogen depletion during exercise and its subsequent repletion over 4 hours post exercise when feeding different types of CHO. Liver glycogen content was measured using ^{13}C magnetic resonance spectroscopy following an overnight fast prior to exercise, and following each glycogen resynthesis trial. A weak but positive association between liver glycogen content following resynthesis and subsequent TTE occurred. Therefore, CHO feeding prior to and during exercise, which increases liver glycogen content and/or reduces the rate it is utilised, may be one factor to help explain the mechanisms of action that feeding CHO can have on exercise performance / capacity. However, this mechanism of action may only be relevant when feeding stops but exercise continues. Nevertheless, it is likely the liver has a key regulatory role when achieving optimal performances with CHO supplementation and warrants further investigation.

Peripheral neural effects

A limited amount of research has been conducted on the peripheral neural effects of CHO ingestion and muscle activity. One study reported attenuation in the rise in muscle neural activity during the latter stages (> 45 min) of exercise at 84% VO_{2max} when ingesting a 6.4% CHO solution (45). Additionally, Abbiss and Peiffer et al (46) highlighted a correlation between muscle activation of the VL and power output sustained during a 16.1 km TT task. However, CHO feeding had no effect on the percentage activation of the muscle when CHO was consumed. This highlights that it was not central factors influencing fatigue during the trial. Additionally, when CHO was not consumed; muscle activation level was maintained even with a reduction in the power output produced. Taken together these findings suggest a peripheral metabolic nature of the fatigue in the TT task. As such, CHO intake during exercise may be able to directly affect the contractile properties of the active muscle, helping to promote force production and subsequently improve performance, though the exact mechanism to explain the effect is unclear. It should be noted that this interpretation is based on only a few studies, often with poor methodological control of muscle activity recordings, highlighting a need for future research on this possible mechanism. In contrast, research investigating the potential central neural effects of CHO administration has been much more active.

Central neural effects (oral glucose sensing)

There is now robust evidence to indicate that CHO can be detected in the oral cavity (47-49) which is thought to directly affect the brain (50, 51). Carter et al (52) demonstrated that detection of CHO in the oral cavity also has an effect on exercise performance. Participants completed a simulated TT (~1 h) as fast as possible when mouth rinsing either a CHO or placebo solution. CHO mouth rinsing caused a significant (2.9%) increase in performance compared to the placebo condition. To isolate the effect of the gastrointestinal (GI) tract Carter et al then infused CHO

intravenously to bypass the GI tract and maintain exogenous CHO availability. CHO was infused at 1 g·min^{-1} but had no effect on exercise performance compared to placebo (53). These observations highlighted the potential importance of oral CHO sensing for performance enhancement during short duration activity. Furthermore, there is great interest in this work since endogenous CHO availability for short duration high intensity efforts is not typically considered a limiting factor for exercise performance in this context.
The outcomes from these studies have led to speculation regarding the underlying mechanisms explaining CHO mouth sensing, and also CHO ingestion during short duration tasks (<1 hr). Some have proposed that oral cavity CHO sensitive receptors exist which detect exogenous CHO and directly act upon the central nervous system (50). An increase in the corticomotor output in the fasted state to both rested and fatigued skeletal muscle when CHO is rinsed in the mouth supports this hypothesis (50). Similarly, CHO mouth rinsing significantly improved performance over that of a saccharin sweetened placebo demonstrating that oral sensors are caloric sensitive (51). Additionally, Chambers reported increases in neural activity in brain regions involved in reward and motor control when mouth rinsing a CHO solution. The authors proposed that the increase in brain activity in these regions supports the increase in performance. However, not all studies report an increase in performance with CHO rinsing (54, 55). The duration of the pre-exercise fast has led some to speculate that nutritional status (fed vs. fasted) of the participant influences the impact of the effect (56).

CARBOHYDRATE DOSE RESPONSE – WHAT IS THE OPTIMAL INGESTION RATE?

Many attempts have been made to recommend 'optimal' doses of CHO for endurance performance. However, considerable debate remains as to how much is enough? Some have suggested there may be a dose response relationship between CHO ingestion and endurance exercise performance. Smith et al (14) fed 15, 30, and 60 g·h^{-1} during a 2 h submaximal ride prior to a 20 km TT. All CHO conditions significantly improved performance compared to the placebo condition. However, a lack of statistical power (n=12) meant only the 60 g·h^{-1} ingestion rate significantly improved performance over that achieved with 15 g·h^{-1}. Additionally, Watson and Shirreffs et al (57) reported that exercise capacity was improved to the same extent with 32 and 47 g.h^{-1} of CHO compared to placebo suggesting that a saturation in additional performance gains may occur around 30 g·h^{-1}. Both of these studies suggested a trend for a dose response relationship, but the low sample sizes have made the existence of a dose response relationship difficult to confirm.
Smith et al (2013) increased sample size using a multi-centre study. Fifty five participants spread across four sites consumed CHO during a 2 h submaximal ride followed by a 20 km TT task. Each participant completed 4 trials, one placebo and three CHO treatments, between 10 and 120 g·h^{-1} (10 g·h^{-1} increments). Following some statistical modelling of their data the authors reported an optimal dose of 78 g·h^{-1} for performances during the TT. However, the linear regression model used for the determination of the optimal feeding strategy utilised was not significant. There are also some concerns over the study design and allocation of treatments across multiple sites. Smith et al report a 1.7% improvement in performance between 30 and 80 g·h^{-1} and a rather trivial 0.7% improvement in performance when dose is increased from 40 to 80 g·h^{-1}. Taken together these studies suggest increasing amounts of CHO may result in diminishing returns with respect to performance gains. From a practical perspective, it is important to further clarify the dose-response relationship. There is a need to discriminate between the dose increments where the largest gains in performance are observed, and dose increments where performance gains are marginal but negative effects (such as gastro-intestinal distress) are increased.
As such, we recently conducted a study where participants ingested 0 (water control) 20, 39 and 64 g.h^{-1} of glucose during

a 2 h submaximal ride which was followed by simulated time trial (~ 35 min). Our unpublished data indicate that 39 and 64 $g.h^{-1}$ were equally effective at improving endurance exercise performance (6.1% and 6.5% respectively) over that of a water control. Additionally, although not statistically significant, consuming 20 $g.h^{-1}$ led to a meaningful mean improvement of 3.7% (~1 min) when compared to the control trial. The results of our study are in line with that of Watson et al (57) highlighting a probable saturation in performance gains with increasing rates of CHO ingestion. Similarly, as shown in other studies, there is considerable variability in the inter-individual response to the different CHO rates ingested. As such, a focus is required on the underlying mechanisms to understand why some individuals respond positively to CHO supplementation and some do not. This may involve individual variation in the tolerability of the solution ingested and in the ability to oxidise the ingested CHO available, in addition to differences in the putative central or peripheral neural mechanisms of CHO feeding outlined above.

PRACTICAL APPLICATION

CHO is crucial for optimal performances and maintaining glycogen stored during heavy periods of training. Consuming a high CHO diet will ensure elevated muscle and liver glycogen contents prior to exercise which may facilitate an increase in exercise performance. When using CHO during exercise recent studies on well-trained but not elite athletes demonstrate the effect of diminishing returns in exercise performance gains with increasing amount of CHO ingested. As such, well-trained individuals can be recommended to consume more modest amounts (30-40 $g \cdot h^{-1}$) of CHO without fear of a reduction in performance gains. Elite athletes may well tolerate higher ingestion rates and can start to increase the amount of CHO they are consuming should they respond positively to increasing amounts of CHO. However, it should be noted that not all individuals will respond positively to CHO intake and practitioners and nutritionists alike should be aware of this possibility.

CONCLUSION

Carbohydrate ingestion during exercise has been positively associated with increases in exercise capacity and performance for a number of years. We are only now starting to understand the mechanisms underpinning these physiologically enhancing effects. The mechanisms are not purely metabolic in nature with peripheral and central neural effects being reported with CHO intake. The optimal dose of CHO for endurance performance is not well defined but appears to be around 40 $g \cdot h^{-1}$. However, this value is likely to be highly individual specific so athletes should take an active role in determining what is optimal for them.

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