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EFFECTS OF CARBOHYDRATE INGESTION DURING EXERCISE

Physiology

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ABSTRACT

To understand the pathway of ingested carbohydrate from 'mouth to mitochondria' culminating in energy production in skeletal muscles during exercise. The intestinal mucosa has carbohydrate sensors that stimulate the release of two 'incretin' hormones (GIP and GLP-1) whose actions range from the secretion of insulin to appetite regulation. Most of the ingested carbohydrate is taken up by the liver resulting in a transient inhibition of hepatic glucose release in a dose-dependent manner. Receptors in the oral cavity detect ingested carbohydrate and activate reward and other centers in the brain that can improve sports performance

KEYWORDS

INTRODUCTION

Strategies to increase pre-exercise muscle glycogen stores include increasing daily dietary CHO intake]and ingesting CHO-rich meals prior to exercise. The consumption of easy-to-digest CHO-rich meals 3 h before exercise increases liver and muscle glycogen concehntrations by 11–15% [5, 6]. Commencing exercise with elevated liver and muscle glycogen contents can improve endurance capacity, as has been consistently shown with running and cycling exercise . Wen a pre-exercise CHO-rich meal is combined with the ingestion of CHO during exercise, then the improvements in endurance capacity during prolonged cycling and during running tasks are greater than when either of these CHO interventions is adopted separately. Endurance time-trial performance (~ 1 h>70% V□ O2max) has also been reported to beneft from CHO ingestion during running and cycling exercise However, the magnitude of beneft likely depends on the pre-exercise endogenous CHO storage levels of an individual . The central response to ingesting CHO has been investigated using functional magnetic resonance imaging (fMRI). In one study, participants ingested either 300 mL of water (control), a glucose solution, an aspartame (sweet taste) solution, or a maltodextrin (non-sweet CHO) solution. Both sweet taste and energy content appeared to produce a hypothalamic response]. The hypothalamic response was reported to be dose dependent on CHO, specifcally, in relation to changes in circulating insulin concentrations]. Both glucose (sweet) and glucose polymers (non-sweet) in the mouth activate regions in the brain associated with reward, such as the insula/frontal operculum, orbitofrontal cortex, and striatum. These fndings suggest that there may be a class of, so far unidentifed, oral receptors that respond to CHO independently of sweetness]. Regions of the brain associated with reward are also believed to mediate behavioral responses to rewarding stimuli, such as taste]. Receptors on the tongue also extract information about the texture and temperature of food. This processing prepares the GI system for compounds in the mouth by causing the organism to salivate, masticate, swallow, or expel, as well as to release a cascade of post-prandial hormones such as insulin and other peptides . In humans, simply tasting food stimulates the release of insulin from the pancreas, known as the cephalic insulin release (CPIR). Under fasting conditions, both nutritive (sucrose) and non-nutritive sweetener (saccharin) solutions have been shown to induce CPIR, when mouth-rinsed for 45 s and expectorated without ingestion]. However, the magnitude of change in insulin secretion from the pancreas following the CPIR is negligible (1–2 mU/L) compared to the ~80-fold increase in response to elevated blood glucose concentrations. Although it is not known if the CPIR persists during exercise, it is unlikely to impact on carbohydrate metabolism or performance.

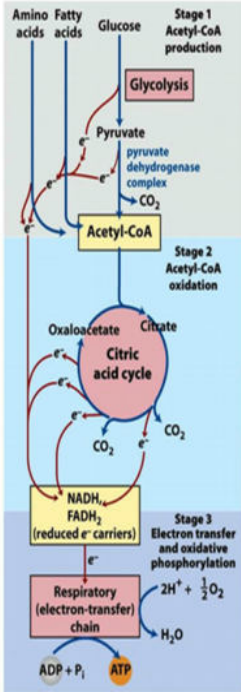
The intestinal mucosa is also involved in CHO sensing. The potential for the gut to sense glucose was established when it was observed that glucose ingestion produces a greater insulin response when compared to intravenous glucose infusion, matched for circulating plasma glucose concentrations . This response has been termed the incretin efect and is primarily a result of the secretion of two incretin hormones: glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide-1 (GLP-1). The absorption of CHO via the SGLT1 transporter is thought to be a key intestinal glucose-sensing mechanism. Substrates for SGLT1 such as glucose, galactose, and glucose analogues stimulate GIP secretion, and these responses can be abolished by the addition of phloridizin, a competitive inhibitor of SGLT1]. These incretin hormones display a variety of physiological efects, from insulin secretion to appetite regulation, yet their roles in exercise metabolism are currently unclear. In addition to hormonal signaling, intestinal sensing of glucose may also signal via vagal aferent pathways], yet the implications of such sensing for exercise metabolism remains to be elucidated. Following intestinal absorption, ingested CHO are transported to the liver via the portal vein, where glucoreceptors are present. These glucoreceptors are innervated by vagal aferent fbers and are important in the I. Rollo et al. sympathoadrenal response to hypoglycaemia . Information from peripheral glucose sensors along the GI tract converges in the nucleus solitarius of the caudal medulla of the brain. The information provided is used by the medulla to generate appropriate oropharyngeal and autonomic motor responses, and can be relayed to the hypothalamus and taste/ visceral cortex via the lateral parabranchial nucleus in the pons.

Glucose is transported to and taken up by the active muscle via GLUT1 (SLC2A1) and (primarily) GLUT4 (SLC2A4) through facilitated difusion. GLUT4 is translocated from an intracellular microsomal GLUT4 pool to the cell membrane following (CHO-induced) insulin release and/or muscle contraction, thereby enabling a rapid increase in plasma glucose uptake within skeletal muscle tissue . Whereas lactate appears to be predominantly oxidized within the active muscle, glucose (after being phosphorylated by hexokinase into glucose-6-phosphate) has at least two distinct metabolic fates during exercise. Glucose within contracting muscle can be directly oxidized via a series of enzymatic reactions (i.e., glycolysis, TCA cycle, and oxidative phosphorylation) to generate adenosine triphosphate (ATP). It has been well established that when (only) glucose is ingested during exercise, that exogenous glucose can be oxidized at a maximal rate of~1.0–1.2 g·min−1. However, these rates can be increased further to up to ~ 1.75 g·min−1 when a mixture of glucose (polymers) and fructose is ingested . Therefore, the additional available (fructose-derived) glucose and lactate in the systemic circulation appear to be oxidized to generate ATP during exercise and maintain exercise performance.

CONCLUSION

It is widely acknowledged that ingested CHO can improve endurance capacity during prolonged exercise of moderateto-high intensity by

providing ample substrate for energy metabolism in skeletal muscle tissue. However, CHO is also the main substrate for energy metabolism in the brain and central nervous system . Ingested CHO is frst detected by receptors in the oral cavity and on the tongue. Its presence is relayed to reward and other centers in the brain resulting in a series of actions that include the release of insulin and enhanced endurance performance. After digestion, glucose is transported across the intestine into the systemic circulation in association with the active transport of Na+. The uptake of glucose in the GI tract seems to be limited by its intestinal uptake via SGLT1. As fructose is transported over the intestinal membrane via a diferent transporter protein, it has been reported that the combined ingestion of glucose and fructose can further increase the capacity to absorb exogenous CHO. Consequently, combining the ingestion of fructose and glucose can augment intestinal CHO uptake, increase post-prandial glucose availability, and increase exogenous carbohydrate oxidation rates by 40–50%. The hepatic glucose output resulting from the ingested CHO is distributed to both the brain and skeletal muscles so as to prevent hypoglycaemia and improve endurance performance capacity of individuals.



TCA Cycle

ETC

overall we are seeing on
how we convert carbohydrates ⇒ ATP

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