

Nutrition Update for the Ultraendurance Athlete

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Abstract

Participation in ultraendurance events has been increasing. Appropriate nutrition in training and fueling while racing within the confines of gastrointestinal tolerability is essential for optimal performance. Unfortunately, there has been a paucity of studies looking at this special population of athletes. Recent field studies have helped to clarify appropriate fluid intake and dispel the myth that moderate dehydration while racing is detrimental. Additional current nutrition research has looked at the role of carbohydrate manipulation during training and its effect on macronutrient metabolism, as well as of the benefits of the coingestion of multiple types of carbohydrates for race fueling. The use of caffeine and sodium ingestion while racing is common with ultraendurance athletes, but more research is needed on their effect on performance. This article will provide the clinician and the athlete with the latest nutritional information for the ultraendurance athlete.

Introduction

During the past 20 years, participation in ultraendurance events has been increasing in popularity. The definition of an ultraendurance event in previous literature ranges from a minimum of 4 to 6 h of continuous racing (43,60,105). In this article, we consider an ultraendurance event as any event that lasts approximately 5 h or longer, in which the majority of nutrition comes from metabolism of lipid sources, and exercise intensity averages 70% $\dot{V}O_{2max}$ or less. While the distance is not specific and is dependent on the particular sport, we consider running races to be longer than a marathon and a triathlon of at least half Ironman distance. Our understanding of the nutritional component of training, race fueling, and postrace refueling is dynamic (2,78). In this article, we will review gastrointestinal (GI) problems, hydration, and sodium and caffeine supplementation as well as focus on recent nutritional topics for the ultraendurance athlete.

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GI Concerns

The ultraendurance competitor must balance nutrition and fluid intake to successfully fuel his or her activity without suffering negative GI consequences. GI problems are some of the main presenting problems to the medical tent in the Lake Placid Ironman (personal observation). Athletes often overestimate their ability to fuel because they do not consider race day nerves and resultant mild GI discomfort or have not fueled at race intensity during training. Splanchnic blood flow is reduced to 30% to 40% of resting values at workloads of 70% $\dot{V}O_{2max}$ as blood shifts to working muscles and skin to dissipate heat (80). There is conflicting evidence on the effects of this blood flow redistribution on car-

bohydrate (CHO) absorption with some studies showing a decrease in absorption when blood flow is reduced to the intestines by 50%, while others show no negative effects of exercise intensity up to 70% $\dot{V}O_{2max}$ (32,97,100). GI problems can be difficult to overcome once they develop during an ultraendurance race because they can lead to dehydration and limit one's ability to handle the thermal effects of racing, which further limits gastric emptying (76). Once an athlete realizes he or she is having GI problems, he or she needs to slow down his or her race pace to allow proper perfusion of the GI tract and, consequently, improved substrate transit.

Race Data

Kruseman *et al.* (61) found that 90% of ultramarathon runners feel that nutrition has an influence on overall performance. Despite a high number of participants in ultraendurance events, there is a paucity of studies looking at actual race fueling (Table 1). Field research has documented hourly caloric intake ranges from less than 100 to 430 kcal·h⁻¹, with an increased ability to consume calories on the bike as opposed to running (30,59). Ultraendurance athletes should expect to finish the race with an energy intake of between 36% and 54% of energy expenditure. The predominant macronutrient athletes choose for fuel during ultraendurance races is CHO, making up as much as 90% of total caloric intake (Fig.). Although lower prerace

Table 1.
Race Fueling.

Author/Journal	Date	Location	Subjects (n)	Type of Event	Weather Conditions (°C)	Fluid (mL·h ⁻¹)	CHO (g·h ⁻¹)	Caloric Intake (kcal·h ⁻¹)	Na (mg·h ⁻¹)	Energy Intake to Utilization (%)
Lindeman <i>et al.</i> (65)/IJSNEM	1991	United States	1	Ultracycling	21 to 35	630	83	431	n/a	n/a
Clark <i>et al.</i> (16)/JADA	1992	United States	1	Ultracycling	Variable	n/a	n/a	420	n/a	n/a
Fallon <i>et al.</i> (30)/IJSNEM	1998	Australia	7	Ultramarathon	2 to 17	540	43	<100	393	n/a
Glance <i>et al.</i> (35)/IJSNEM	1992	—	19 finishers	Ultramarathon	—	741	43	248	500	45
Glance <i>et al.</i> (36)/JACN	1992	—	13 finishers	Ultramarathon	21 to 38	700	54	311	678	54
Kimber <i>et al.</i> (59)/IJSNEM	2002	New Zealand	10 male	Ironman	21	n/a	78	328	161	39
Kimber <i>et al.</i> (59)/IJSNEM	2002	New Zealand	8 female	Ironman	21	n/a	60	247	115	36
Kruseman <i>et al.</i> (61)/EJAP	2005	Switzerland	42 finishers	Ultramarathon	18 to 27	545	31	141	150	40 to 52

EJAP, *European Journal of Applied Physiology*; IJSNEM, *International Journal of Sports Nutrition and Exercise Metabolism*; JACN, *Journal of the American College of Nutrition*; JADA, *Journal of the American Dietetic Association*; n/a, data not available.

body fat consistently predicts race performance, Kruseman *et al.* found that an increased hourly rate of CHO supplementation and overall caloric intake also correlated with faster race times (36,59,61).

Train Low, Race High

In 1999, Stray-Gundersen and Levine (92) reported the adaptive endurance benefits of “live high, train low.” After a 4-wk training camp for college runners at low altitude, they randomly divided the 39 participants into three groups for subsequent training: HiHi (those who lived and trained at high altitude), LoLo (those who lived and trained at low altitude), and HiLo (those who lived at high altitude but trained at low altitude). Only the HiLo group showed improvement in low-altitude performance after training. The concept behind live high, train low is that metabolic adaptations occur because of the hypoxic stimulant of high altitude resulting in an increase in erythropoietin and red

blood cell mass while, simultaneously, quality physiologic training stress is maintained by training in a higher oxygen environment.

There is an abundance of strong evidence that beginning endurance exercise with a high muscle glycogen load and ingesting CHO during prolonged exercise will improve performance (10,19,23,41,57). But, analogous to a low-oxygen stimulus, are there benefits to purposely training in a low-glucose environment? “Train low, race high” implies periodic training with lowered blood glucose or endogenous glycogen, followed by the repletion of CHO before subsequent training or competition (40).

In 2005, Hansen *et al.* were the first to look at the value of “train low, compete high” with respect to CHO (40). In their study, seven untrained men performed leg extension during 1-h training sessions following an overnight fast 5 d·wk⁻¹ for 10 wk but had different training stimuli with each leg. On day 1, the HIGH leg was trained together with the LOW leg and then, after glycogen replenishment, was trained alone on day 2. The LOW leg was trained during a second session 2 h after its first session without allowing replenishment of glycogen stores. The LOW leg was not trained on day 2. Both legs trained an equal number of sessions, and the pattern repeated every 2 d. Posttraining biopsies revealed increased muscle glycogen concentration, increased levels of the mitochondrial enzyme 3-hydroxyacyl-CoA dehydrogenase, and a statistically significant increase in citrate synthase levels in the LOW leg compared with the HIGH. In addition, time to exhaustion was increased in the LOW compared to the HIGH leg. The authors concluded that training in a state of reduced glycogen stores caused cellular adaptation that result in increased endurance performance.

Two subsequent studies (45,103) investigated whether well-trained athletes would experience similar metabolic outcomes and performance improvements to those observed in the untrained subjects in the investigation of Hansen *et al.* (40). In these 3-wk studies, training adaptations to low glycogen stores resulted in greater cellular oxidative adaptations than training completed with normal or high glycogen stores. However, no difference in 60-min cycling time trial performance resulted from training with lower glycogen stores, with both groups increasing 10%



Figure: Aid Station at Ultraendurance Event.

from baseline due to the 3-wk training session. Of note, in the study by Yeo, the LOW group was not able to reach the same high percentage of peak power output during high-intensity training (HIT) sessions as the HIGH group during training but still achieved the same performance improvement (103).

Other studies evaluating the effect of decreasing exogenous glucose availability on training adaptation revealed equivocal results (1,27,70,96). Although metabolic adaptations increasing oxidative capacity were increased in the LOW groups in several of these studies, no overall difference in performance benefits such as $\dot{V}O_{2\max}$, 60-min time trial, and time to exhaustion were found (1,27,70,96). Morton *et al.* assessed the difference in stimulation of increased heat shock protein production of controls versus groups with lower muscle glycogen via a prior same-day training session and low exogenous CHO availability by limiting CHO ingestion during exercise (66). They found an increase only in oxidative enzymes but not in heat shock proteins (which are involved in cell remodeling processes such as mitochondria biogenesis) or exercise performance (66).

A study by Cox *et al.* (21) deserves specific attention because unlike many studies that use atypical laboratory fabricated training stimuli, it looked at endurance-trained male triathletes and cyclists and used a typical cyclist's training program. The 16 subjects were paired based upon fitness level and divided randomly into a high-CHO group and an energy-matched low-CHO group. Subjects performed individually determined training programs $6 \text{ d}\cdot\text{wk}^{-1}$ that consisted of time trials, hill rides, and HIT sessions, as well as a standard weekly long ride of 3 to 5 h. The results did not show a difference in training-induced increase in performance between the groups (Table 2).

It would seem that training in a low-CHO state and resultant demonstrated increase in oxidative enzymes would be especially beneficial for ultraendurance competitions in which exercise intensity is usually below 70% $\dot{V}O_{2\max}$ and most of fuel utilization is free fatty acids (75,79). However, there have been no studies looking at ultraendurance performance after training with low CHO. Further research is needed to evaluate and determine if there is an appropriate titration where the metabolic gains of training low exceed the losses from the lower training intensity stimulus. It is likely that many endurance athletes already incorporate a portion of their training with reduced CHO availability either intentionally or unintentionally, due to busy training, work, or school schedules and/or limited nutrition and fueling knowledge (42).

Fluids

Fluid intake is a balance between fluid deficit, which has been shown in laboratory studies to result in a decrease in work capacity and to contribute to the development of hyperthermia, and fluid overload that can result in exercise-associated hyponatremia (EAH) (11,13,24,34,69,73,81). In 1969, Wyndham and Strydom published a report on runners who ran Sugar's Marathon in Johannesburg, South Africa, on two fall days in 1969, which helped shape fluid consumption recommendations for the next 30 years (18,101). In the study, there was a linear correlation between weight loss and increasing rectal temperatures at

the finish of the marathon. The authors noted a rise in temperature to $>102^{\circ}\text{F}$ occurred more frequently in individuals with $>3\%$ loss in body weight and felt the increase in temperature could lead to heat stroke (although none of the runners developed heat stroke). They concluded that thirst was not a good gauge of hydration status and stated, "The ideal regimen of water drinking is to take about 300 ml every 20 minutes or so. This should start right at the beginning of the race." Of interest is that the runner with the greatest degree of dehydration and rectal temperature won the race on both days.

Results from more recent studies have refuted Wyndham and Strydom's concerns that thirst is not a good indicator for hydration status and that $>3\%$ weight loss from evaporation of sweat will result in heat stroke. Looking at competitors in the South African Ironman, Sharwood *et al.* (87) showed that pre- to postrace changes in body weight were not related to postrace rectal temperatures. Some of the ultraendurance triathletes they studied sustained a 6% loss in body weight without a resultant increase in medical problems (87,88). In addition, body weight loss of 3% in the Ironman race did not lead to thermoregulatory failure (64).

In half-marathon runners and ultramarathoners, body mass alone may not be an accurate indicator of hydration status. Tam *et al.* (94) looked at runners of 21.1- and 52-km races and found they lost body mass (from a combination of substrate utilization, sweat evaporation, and insensible fluid loss) but overall gained total body water while preserving serum sodium and potassium and only had a small change in serum osmolality. Despite not drinking as much as sweat losses, they attributed the increase in total body water from release of water stored with glycogen that was released during muscle glycogen metabolism and possibly from fluid stores in the GI tract. Kao *et al.* (56) evaluated body weight changes before, at 4-h intervals during, and immediately after 12- and 24-h ultramarathons and found a positive correlation between weight loss and performance in the 24-h race.

Athletes should have a basic hydration plan prior to entering competition that they have developed and tested during their training sessions. The 2002 International Marathon Medical Directors Association guidelines, although targeted at marathon runners, are a good starting point for athletes in ultraendurance events (68). These advise that marathon runners drink approximately $400 \text{ to } 800 \text{ mL}\cdot\text{h}^{-1}$, with increased rates for the faster, heavier runners, competing in warm conditions, but no more than $800 \text{ mL}\cdot\text{h}^{-1}$. Although faster competitors often finish with more significant dehydration, they often have greater experience and may be able to safely tolerate a higher degree of dehydration without suffering negative medical consequences (68,74,101). Pre- and postworkout weights can help athletes assess fluid needs for specific training loads, and monitoring urine color (aim for light yellow) and frequency can help athletes predict needs during events. On race day, athletes need to be flexible in their fluid plans to account for race day environmental conditions.

Ingestion of Multiple Different Carbohydrates

In 2004, Jentjens *et al.* and Jeukendrup and Jentjens at the University of Birmingham Human Performance Laboratory

Table 2.
Restricting CHO availability during training, selected studies.

Author	Date	Location	Subjects	Training Stimulus	Type of CHO Restriction	Main Outcomes
Hansen <i>et al.</i>	2005	Denmark	Seven untrained males	10 wk, five sessions per week of 1-h knee extensor exercise	Half of the training completed in a reduced glycogen state	Compared with training with normal glycogen levels, training with lowered glycogen stores: <ul style="list-style-type: none"> • increased resting muscle glycogen and levels of mitochondrial enzymes • doubled the magnitude of the increase in posttraining time to exhaustion
Yeo <i>et al.</i>	2008	Australia	14 well-trained male cyclists and triathletes	Once- or twice-a-day cycling for 100 min AT followed 1 to 2 h later by HIT for 3 wk	Half of the training was completed in a reduced glycogen state	Training with lowered glycogen stores resulted in: <ul style="list-style-type: none"> • increased resting glycogen • increased adaptation related to fat oxidation and increased whole body fat oxidation • no difference in performance
De Bock <i>et al.</i>	2008	Belgium	20 moderately active males	6-wk endurance cycling (2 h, 3 d·wk ⁻¹ at ~75% $\dot{V}O_{2peak}$)	Trained in a fasted state while withholding exogenous carb	The carb-restricted group experienced the following: <ul style="list-style-type: none"> • a decrease in exercise-induced glycogen breakdown • increased levels of proteins involved in fat metabolism but no increase in total fat oxidation
Akerstrom <i>et al.</i>	2009	Denmark	Nine physically active males	10-wk knee extensor exercise program	Trained in a fasted state while withholding exogenous carb	Exogenous glucose ingestion during training did not alter the following: <ul style="list-style-type: none"> • adaptation related to substrate metabolism • mitochondrial enzyme activity • glycogen content • performance outcomes
Nybo <i>et al.</i>	2009	Denmark	15 untrained males	8-wk endurance and high-intensity cycling	Exogenous carb restricted	<ul style="list-style-type: none"> • The carb-restricted group had increased levels of GLUT-4, mitochondrial enzymes, and resting muscle glycogen • The CHO-supplemented group only had increased lean body mass

(Continued on next page)

Table 2.
Continued.

Author	Date	Location	Subjects	Training Stimulus	Type of Carb Restriction	Main Outcomes
Hulston <i>et al.</i>	2010	United Kingdom	14 well-trained male cyclists and triathletes	3-wk endurance and high-intensity cycling	Half of the training was completed in a reduced glycogen state	Training with reduced glycogen: <ul style="list-style-type: none"> • lowered self-selected training intensity • increased muscle triglyceride oxidation and mitochondrial enzyme activity • did not improve cycling time trial performance
Yeo <i>et al.</i>	2010	Australia	12 well-trained male cyclists and triathletes	Single bout of aerobic cycling (100-min ride) followed by HIT (8 × 5-min work bouts with 1-min rest)	Withheld carb repletion between aerobic and HIT training	Single HIT training session with low glycogen stores resulted in a greater magnitude of increase in phosphorylation of 5'AMP-activated protein kinase
Cox <i>et al.</i>	2010	Australia	16 well-trained male cyclists and triathletes	28-d endurance and high-intensity cycling	Exogenous carb withheld 2 h before and during training	No clear effects of the carb restriction on resting muscle glycogen, GLUT-4 activity, or performance
Morton <i>et al.</i>	2010	United Kingdom	23 recreationally active males	6 wk of high-intensity run training	Reduced glycogen stores coupled with either withholding or providing exogenous carb during HIT	Training with reduced exogenous and endogenous carb availability resulted in enhanced stimulus for inducing oxidative enzyme adaptation but did not lead to performance enhancement
Van Proeyen <i>et al.</i>	2011	Belgium	Physically active males	6-wk endurance cycling	Training completed in fasted state. No exogenous carb provided during training.	The fasted subjects experienced increased: <ul style="list-style-type: none"> • contribution of intramyocellular lipid to energy • levels of oxidative enzymes • blood glucose stability No difference in performance was seen between the two groups

AT, aerobic training.

used radioisotope methodology to show that CHO oxidation can be increased beyond the previous believed ceiling of $1 \text{ g} \cdot \text{min}^{-1}$ for a single CHO to a new level of $1.26 \text{ g} \cdot \text{min}^{-1}$ when glucose and fructose are coingested (50,54). Eight trained male cyclists or triathletes performed four exercise trials of 120 min of cycling at 50% W_{max} (defined as last completed work rate plus fraction of time spent in the final noncompleted work rate, multiplied by the work rate

increment during a graded exercise test to exhaustion). Fluids consumed during the trials consisted of 8.7% glucose drink (Med-Glu), a 13% glucose drink (High-Glu), an isoenergetic fructose + glucose drink (Fruc + Gluc) (in fructose-to-glucose ratio of 1:2), and plain water. There was an increase in total exogenous CHO oxidation in the combination group ($1.26 \text{ g} \cdot \text{min}^{-1}$) compared with the groups with a single CHO fuel source ($0.8 \text{ g} \cdot \text{min}^{-1}$). Interestingly, the

rate of glucose oxidation alone in the Fruc + Gluc trial was equal to the rate of the Med-Glu and the High-Glu, and the amount of fructose oxidation was equivalent to the overall difference in CHO oxidation between the groups. Follow-up studies looking at high glucose/fructose ingestion rate; CHO mixtures of glucose, fructose, and sucrose; and combination of maltodextrin (a nonsweet tasteless glucose polymer of approximately 10 glucose units long) and fructose have all supported increased exogenous CHO oxidation with multiple CHO sources compared with a single CHO source or water (49,51,99).

In one of the few nutrition studies that looked at exercise duration within the ultraendurance range, Jeukendrup and Chambers evaluated the CHO oxidation rates of eight endurance-trained men who cycled at 50% W_{max} for 5 h, while ingesting glucose solution, glucose + fructose, or water (55). The glucose + fructose trial had increased exogenous CHO oxidation and increased cycling cadence at end of time trial and a decreased RPE for the workload. While it is assumed that increased exogenous CHO oxidation and sparing of endogenous glycogen stores will translate to improved performance, it was not looked at until 2008. Currell and Jeukendrup (25) tested eight trained male cyclists in the laboratory with three exercise trials where subjects ingested a glucose-only beverage, a glucose + fructose solution, and plain water. The protocol involved 2 h cycling at 55% W_{max} followed by a time trial in which subjects had to complete a set amount of work as quickly as possible. The glucose + fructose group had an 8% faster time than the glucose group and a 19% faster time than the water trial.

Glucose is absorbed in the intestine by the sodium-dependent glucose SGLT1 receptor, while fructose is transported from the intestinal lumen via the sodium-independent GLUT5 transporter (31). Fueling with glucose beyond the level of complete saturation of the SGLT1 transporters will result in GI distress and not increased absorption. We advise ultraendurance athletes to fuel with a combination of different CHO to support maximum oxidation. A practical consideration during training and racing also includes optimal delivery of low concentrations of glucose and fructose in a manner that will not interfere with hydration (e.g., mixing concentrated drinks), usually <6%. GI upset often experienced by athletes ingesting fructose and other concentrated “simple” sugars during competition may be mitigated potentially by the use of maltodextrin. Its use can result in higher CHO availability without impairing hydration and possibly less GI distress. Posttraining or event repletion, especially in the first hour after exercise, when the body has an increased ability to replenish glycogen stores, offers more flexibility and tolerability when mixing sugars and maximizing absorption and utilization.

Sodium Supplementation

There are two potential roles for supplemental sodium during ultraendurance events: prevention of EAH and prevention of exercise-associated muscle cramping (EAMC). Speedy *et al.* looked at competitors in the 2000 South African Ironman who received supplemental sodium tablets

and compared them to controls who did not receive supplemental tablets (sodium intake was not regulated otherwise) (89). The sodium ingestion group showed a decrease in weight loss compared with controls (3.30 vs 3.92 kg), but there was no difference in serum sodium concentration or plasma volume between the two groups, and there were no cases of hyponatremia in either group. The study was repeated with similar results at the following year’s race, but the test group was given salt tablets, and the control group received placebo pills (44). The authors concluded that sodium supplementation was of no additional benefit beyond limiting excessive fluid intake in the prevention of EAH.

EAMC is defined as “a painful, involuntary contraction of skeletal muscle that occurs during or immediately after exercise” (83). The two prevailing theories regarding its etiology are sodium deficit and neuromuscular excitability, but they are not mutually exclusive (9,82). Occupational medicine literature has shown that sodium supplementation can be an effective treatment for and prevention of cramps in individuals who work in extreme heat (28). Bergeron reported on a cramping tennis player with sweat loss of 50 g of sodium during a 4-h exercise session who eliminated EAMC with appropriate salt supplementation (8). Higher sweat sodium losses have been shown in football players with a history of EAMC as opposed to those without a history of EAMC (90,91).

Swellnus *et al.* from South Africa have done excellent research comparing ultraendurance racers who developed EAMC with controls in multiple studies and found that the EAMC group had an increased height, an increased weight, a history of tendon and/or ligament injury, family history of EAMC, faster predicted race time, and a history of previous EAMC (84,86). In addition, the EAMC group had faster race times and faster cycling times, despite both groups having similar training volume and intensity and similar personal bests in previous endurance races (84,86). The groups had similar serum electrolyte measurements and weight change. The authors concluded that serum electrolytes and hydration status were not associated with EAMC but more likely were due to athletes competing at an overreaching intensity level, leading to fatigue, altered neuromuscular control, and, ultimately, EAMC. However, a closer look at their data might suggest a contributing role of sodium. In their study, evaluating runners at the Two Oceans Ultramarathon, prerace serum sodium was similar for the cramping and noncramping runners (139.2 vs 139.3 $\text{mmol}\cdot\text{L}^{-1}$). However, there was a significantly lower postrace serum sodium in the group of athletes who suffered from EAMC compared with the noncramping group (139.8 vs 142.3 $\text{mmol}\cdot\text{L}^{-1}$) (85). This observation was repeated in a follow-up study evaluating Ironman triathletes in which the postrace serum sodium of the athletes with EAMC was statistically lower than that of the control group (140 vs 143 $\text{mmol}\cdot\text{L}^{-1}$) (93). It also is possible that faster racers and racers with an elevated body mass index were not overreaching race day but simply losing more sodium due to sweat evaporation and that intravascular sodium levels were preserved at the expense of the interstitial compartment (95). It would enhance our understanding to perform follow-up studies evaluating individual sweat rate and

sodium concentrations in athletes with EAMC compared with controls.

When Jordan Rapp set the course record for Ironman Arizona in 2009, he consumed $1,532 \text{ mg}\cdot\text{h}^{-1}$ of sodium on the bike and an additional $2,131 \text{ mg}\cdot\text{h}^{-1}$ on the run (personal communication). Elite ultraendurance athletes have learned by trial and error that sodium supplementation while racing may prevent EAMC. Whether science ultimately will support current practice is unclear. However, the risk of increased sodium ingestion is minimal except for mild GI discomfort (95). The authors are unaware of any reports of ultraendurance athletes developing hyponatremia from excessive sodium supplementation. We encourage ultraendurance athletes, especially athletes who are larger, faster, or known high-volume or high-sodium sweaters to ingest sodium beyond what is in manufactured sports drinks, gels, and bars. A reasonable starting place is a total of 500 to $1,000 \text{ mg}\cdot\text{h}^{-1}$. Individual experimentation during training is recommended to determine GI tolerance and effectiveness in preventing EAMC.

Caffeine

A current review of four leading sports fuel and energy drink manufacturers reveals that more than half of their gels and many of their other fueling products contain caffeine. Caffeine, a methylxanthine that blocks adenosine receptors, has actions on essentially all human tissues, peaking in serum 15 to 120 min after ingestion, with a half-life ranging from 2.5 to 6 h (4,37). The effects of caffeine can persist for up to 6 h or more after ingestion (58,77). The predominant mechanism of the ergogenic benefits of caffeine is still unknown. However, various mechanisms of action have been proposed: inhibition of phosphodiesterase with a subsequent increase in lipolysis and preservation of glycogen; mobilization of intracellular calcium via release from the sarcoplasmic reticulum, which improves muscle contractility; antagonism of central adenosine receptors; and an increase in β -endorphin and cortisol release, which may alter the athlete's perception of pain and fatigue (20,26,33,63).

Caffeine has been shown to reduce the onset of fatigue in sustained workload studies, as well as to improve time trial performance (20,22,39,38,46,71,104). However, we are unaware of studies on caffeine use with exercise lasting longer than 3 h — well below the ultraendurance level. Cox *et al.* tried to simulate race conditions in the laboratory with appropriate prerace glycogen loading and CHO fueling during a 2-h steady-state ride at 70% $\text{VO}_{2\text{max}}$ followed by a time trial that lasted <27 min (22). Twelve highly trained athletes in two different studies ingested $6 \text{ mg}\cdot\text{kg}^{-1}$ of either caffeine or placebo 1 h prior to their time trial in trial 1 and ingested a $5\text{-mg}\cdot\text{kg}^{-1}$ cola beverage at two intervals prior to their time trial or an equal caloric CHO placebo in trial 2. Caffeine supplementation resulted in >3% improved time trial performance, but there was no benefit with cola ingestion compared to CHO noncaffeinated substrate (postulated by the authors to be from the low total caffeine intake in the cola group). Taking caffeine with CHO may be additive for performance enhancement. Yeo *et al.* noted increased exogenous glucose oxidation when caffeine doses of $5 \text{ mg}\cdot\text{kg}^{-1}$ were ingested with glucose during 2 h of sub-

maximal cycling, perhaps due to increased intestinal absorption (104). Pedersen *et al.* showed that caffeine might be useful for recovery as well as performance (72). They studied seven endurance trained cyclists/triathletes who trained 12 to $15 \text{ h}\cdot\text{wk}^{-1}$ and were not habitual caffeine users and found that a ride to volitional exhaustion followed by ingestion of CHO with $8 \text{ mg}\cdot\text{kg}^{-1}$ of caffeine immediately following the ride resulted in 66% higher muscle glycogen levels than CHO alone 4 h later. Perhaps another important effect of caffeine in ultraendurance athletes is enhanced mental focus late in the race as concentration becomes more difficult (personal experience).

Common side effects of caffeine include nausea, stool urgency and frequency, diarrhea, jitteriness, palpitations, anxiety, elevated blood pressure, headaches, insomnia, physiologic addiction, and withdrawal symptoms. While 3 to $6 \text{ mg}\cdot\text{kg}^{-1}$ may be beneficial, higher doses may be more harmful than helpful. Caffeine consumption over $15 \text{ mg}\cdot\text{kg}^{-1}$ can cause headaches and more profound nervousness, and $200 \text{ mg}\cdot\text{kg}^{-1}$ can invoke seizures and death (4). Hypokalemia and dehydration requiring hospitalization of cyclists ingesting very high levels of caffeine in competition have been reported (77). Consumption of $>7 \text{ mg}\cdot\text{kg}^{-1}$ does not have a greater ergonomic advantage over 3 to $6 \text{ mg}\cdot\text{kg}^{-1}$ but results in greater urinary concentrations. Benefits may level off due to adenosine receptor and hepatic caffeine metabolism saturation (29,39,71). Despite warnings that caffeine has negative effects on hydration status, electrolyte balance, and thermoregulation, Armstrong *et al.*, in a review of 75 years of literature, found that the evidence does not support these concerns (3).

Ingesting 3 to $6 \text{ mg}\cdot\text{kg}^{-1}$ of caffeine with CHO 15 to 60 min prior to exercise followed by dosing at regular intervals (perhaps every 2 to 5 h) for longer events may enhance performance and appears to be safe. If competitors in ultraendurance races wish to consume caffeine while racing, we advise practicing with caffeine during training since responses vary and caffeine-naïve individuals often have more adverse effects but possibly a greater ergogenic effect (7).

CHO Rinse

A CHO rinse consists of ingesting CHO solution into the mouth, swishing it around, and then spitting the solution out (as opposed to passing the solution into the stomach) in an effort to stimulate CHO taste receptors in the mouth without exogenous CHO oxidation. CHO feedings improve exercise performance in prolonged moderate-intensity exercise due to maintenance of blood glucose levels and increased exogenous CHO oxidation (17,23). In a glycogen-loaded athlete, one might not expect a performance benefit from CHO supplementation during high-intensity exercise of 1 h in duration due to sufficient endogenous CHO stores to maintain blood glucose levels and the time required for the body to metabolize ingested substrate and distribute to working muscles. Surprisingly, Jeukendrup *et al.* found that CHO ingestion during a fixed workload cycling time trial of approximately 1 h resulted in 1 min better performance despite an estimate of only 15 g of exogenous CHO oxidation (52). In a follow-up study, glucose intravenous infusion in a similar time trial did not show an improved performance compared with saline (15).

Carter *et al.* took the next logical step and looked at the response of CHO rinse on exercise performance (14). Seven male and two female endurance-trained athletes (mean $\dot{V}O_{2\max} = 63.2 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) performed two 1-h cycling time trials after a 4-h fast: one with 6.4% maltodextrin rinse and one with water. In both trials, participants rinsed with the trial solution for 5 s then spit into a bowl. There was a statistically significant improvement in the maltodextrin rinse time trial performance compared with placebo with no increased heart rate or RPE. However, in a follow-up study with a similar 1-h time trial, there was no benefit to CHO rinses when athletes began exercises following a standardized breakfast 2 h prior to testing (6). Functional magnetic resonance imaging studies have demonstrated that CHO ingestion (glucose, maltodextrin, and sucrose) activates reward centers in the brain that are not activated with artificial sweetener (saccharin) (52).

Baron *et al.*, Lambert *et al.*, and Noakes have proposed that the brain has a functional entity termed the “central governor” that receives afferent information from the body and surrounding environment and determines the appropriate pace of exercise that will allow the individual to optimize performance and prevent injury to the body’s vital organs (5,62,67). Jeukendrup and Chambers hypothesized that CHO rinse can potentially override the brain’s central governor (53). It is possible that the afferent messages sent from oral CHO receptors indicate to the central governor that the body’s CHO supplies are plentiful, in a sense, “tricking” the central governor and lessening the athlete’s perceived sense of fatigue.

We recommend that, if an ultraendurance racer is unable to temporarily consume fuel during a race due to GI discomfort, he or she consider rinsing with CHO while resting the GI tract. However, this attempt to override normal physiologic feedback mechanisms should be done with caution because obscuring protective indicators of exertion may have deleterious outcomes.

Summary

Appropriate fueling is essential for optimal performance and safety in ultraendurance events. Athletes should practice their race nutrition during training sessions (if possible with the nutrition that will be offered at their upcoming races). A reasonable goal for caloric intake is to replenish some of used CHO but not to expect to completely replace all utilized glycogen stores. We encourage fueling with different CHO sources to maximize oxidation, ingest supplement sodium to possibly prevent EAMC, and consider adding caffeine either before or during the race. Recent evidence suggests there may be a role for CHO manipulation in training to maximize CHO oxidative capacity on race day. Individuals who are having problems fueling on race day might want to consider rinsing with CHO in an effort to stimulate central drive while their GI tract recovers. Thirst is a good indicator of appropriate hydration status for the given race, and athletes should expect to finish the race dehydrated.

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