LEADING ARTICLE

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Fluid and Electrolyte Balance in Ultra-Endurance Sport

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Abstract

It is well known that fluid and electrolyte balance are critical to optimal exercise performance and, moreover, health maintenance. Most research conducted on extreme sporting endeavour (>3 hours) is based on case studies and studies involving small numbers of individuals. Ultra-endurance sportsmen and women typically do not meet their fluid needs during exercise. However, successful athletes exercising over several consecutive days come close to meeting fluid needs. It is important to try to account for all factors influencing bodyweight changes, in addition to fluid loss, and all sources of water input. Increasing ambient temperature and humidity can increase the rate of sweating by up to approximately 1 L/h. Depending on individual variation, exercise type and particularly intensity, sweat rates can vary from extremely low values to more than 3 L/h.

Over-hydration, although not frequently observed, can also present problems, as can inappropriate fluid composition. Over-hydrating or meeting fluid needs during very long-lasting exercise in the heat with low or negligible sodium intake can result in reduced performance and, not infrequently, hyponatraemia. Thus, with large rates of fluid ingestion, even measured just to meet fluid needs, sodium intake is vital and an increased beverage concentration [30 to 50 mmol/L (1.7 to 2.9g NaCl/L) may be beneficial. If insufficient fluids are taken during exercise, sodium is necessary in the recovery period to reduce the urinary output and increase the rate of restoration of fluid balance.

Carbohydrate inclusion in a beverage can affect the net rate of water assimilation and is also important to supplement endogenous reserves as a substrate for exercising muscles during ultra-endurance activity. To enhance water absorption, glucose and/or glucose-containing carbohydrates (e.g. sucrose, maltose) at concentrations of 3 to 5% weight/volume are recommended. Carbohydrate concentrations above this may be advantageous in terms of glucose oxidation and maintaining exercise intensity, but will be of no added advantage and, if hyperosmotic, will actually reduce the net rate of water absorption.

The rate of fluid loss may exceed the capacity of the gastrointestinal tract to assimilate fluids. Gastric emptying, in particular, may be below the rate of fluid loss, and therefore, individual tolerance may dictate the maximum rate of fluid intake. There is large individual variation in gastric emptying rate and tolerance to larger volumes. Training to drink during exercise is recommended and may enhance tolerance.

Water, electrolytes and carbohydrates are critical nutrients for the maintenance of normal physiological function and optimal exercise performance. As exercise endurance and intensity increase, fluid and electrolyte losses and compartmental imbalances increase, unless the losses are compensated by appropriate intakes. Not only volume but also composition is critical in ensuring whole body fluid (intra- and extracellular, vascular and interstitial) homeostasis. Carbohydrate ingestion during prolonged exercise can aid performance, not only through increased glucose oxidation but also, indirectly, through enhanced water absorption.

Unfortunately, most of our knowledge regarding fluid needs and appropriate compensatory measures is based on data from studies in which exercise duration was 3 hours or less, with most controlled studies having been conducted with 1 to 2 hours of exercise. The present article will address the fluid needs of sportsmen and women involved in ultra-endurance activities and the present state of knowledge of supplementation before, during and after exercise to optimise fluid homeostasis. This is based upon mechanisms, often elucidated from studies of exercise of moderate duration, and upon the few studies conducted with ultra-endurance exercise, those being primarily field and case studies.

For the purpose of this paper, 'ultra-endurance' sport will be defined as continuous sporting activity, of a competitive nature or at a comparable intensity, lasting >3 hours. Euhydration is defined as the state of fluid balance or equilibrium; hypohydration is defined as a state of fluid deficit and dehydration is defined as the process of becoming hypohydrated.

1. Importance of Fluid and Electrolytes for Health and Performance

With an increasing rate of muscle contraction and an increasing mass of active musculature, the metabolic demands increase as does heat production. Compensatory physiological responses include an increase in circulation and redistribution of blood flow. In particular, increases in blood flow to the muscle and periphery (skin)[1] and a decrease in abdominal splanchnic (hepatic[2] and portal vein[3]) blood flow are observed. In addition to these cardiovascular changes, an increase in sweat production occurs,[4-6] the evaporation of which provides the most efficient means of heat loss available to humans. During intensive and/or long-lasting exercise sweat losses can substantially reduce the body water. There is virtually no storage of water in the body, with the exception of perhaps water stored with muscle glycogen (which is necessary for the metabolism of that glycogen), and that in the bladder which is unavailable to contribute to fluid needs. Insensible water loss is also increased during exercise primarily through an increase in respiration and evaporation of fluid from the airways. Most water loss experienced during exercise is a result of sweating.

If fluids are ingested in sufficient amounts to offset the fluid losses, maintenance of the endogenous cooling mechanisms is achieved. However, if a fluid deficit occurs during exercise, plasma volume and stroke volume are reduced. Heart rate is increased, and cardiac output decreases at a given point, because of the inability of the heart rate to compensate the reduction in stroke volume, and core temperature is elevated.[7] In contrast, with fluid ingestion during exercise, these responses are attenuated.[8] Gonzalez-Alonso et al.[9] have shown that blood flow to the exercising muscles, as a consequence of reduced perfusion pressure and systemic flow, declines with dehydration during exercise.[9] These authors have also shown that fatigue associated with dehydration during exercise is highly correlated with high body temperature.[10] There appears to be a critical internal body temperature at which fatigue occurs despite varying initial body temperatures or rate at which heat storage occurs. However, this threshold is reduced with dehydration during exercise.[11]

Substrate metabolism is also altered as a result of dehydration during exercise; glycogenolysis and a greater reliance on anaerobic metabolism of glucose are observed.[12] However, it appears that avail-

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ability of carbohydrate stores is not responsible for the fatigue associated with dehydration during exercise but rather the associated hyperthermia.[11]

The relative importance of carbohydrate and fluid supplementation during exercise can vary, as a result of variation in rates of sweating and carbohydrate utilisation. External factors such as environmental conditions as well as type, intensity and duration of exercise influence these needs. The effects of exercise intensity and ambient temperature on sweat rate are shown in figure 1, which is based on data from several labs, modelled by Sawka and Pandolf.[13] In cooler conditions, with lower sweat rates and prolonged moderate to intensive exercise, glycogen reserves can become limiting before dehydration becomes significant. Conversely, in hot and humid conditions, when sweat rates are high, fluid deficit may limit performance before carbohydrate reserves become limiting to performance. As a consequence, not only fluid but also carbohydrate may be limiting to performance in a given ultra-endurance endeavour and as such supplementation must take into account both needs. In this article, the focus is on sweat losses and the replacement of those losses.

Sweating not only results in water loss but also electrolyte losses. The level of sodium in sweat typically ranges from 20 to 80 mmol/L and that of potassium ranges from 4 to 8 mmol/L,[14-16] cited in Maughan[17] and Costill.[18] Electrolytes, and in particular sodium levels, vary dramatically in sweat and are increased by increasing sweat rate and decreased with heat acclimation and training adaptation.[18]

Sodium, as the primary extracellular cation in the extracellular space, is lost to the greatest extent. Plasma sodium levels range from 130 to 155 mmol/L,[14-16] cited in Maughan;[17] however, the clinically normal range is 136 to 145 mmol/L.[19] Potassium is the primary intracellular cation. The level of potassium in the extracellular fluid is 10 mmol/L and in the intracellular fluid 150 mmol/L. The variance in electrolyte composition between the intracellular and extracellular compartments is not caused by the membrane being impermeable, but rather by active transport by an ATP dependent, membrane bound pump (Na^+ K^+-ATPase). This pump serves to maintain an electrochemical gradient that allows for membrane excitability. Therefore, nerve conduction and initiation of cardiac and skeletal muscle contraction is dependent upon this differentiation in electrolyte composition in the extracellular and intracellular fluid. A large variance from the norm in extra- or intracellular electrolyte composition can alter the excitability and, thus, the rate of contraction and/or nervous conduction. With each contraction potassium leaks out of, and sodium moves into, the intracellular space. If this is not countered by an equal but opposite pumping of these ions, further muscle contraction is inhibited. Energy supply and the availability of ATP play a central role in maintaining the electrochemical gradient.

Thus, with large sweat losses a considerable loss of sodium and chloride and a relatively small loss of potassium and other ions (calcium, magnesium, bicarbonate, phosphate, sulphate) occurs. Costill and Miller[20] estimated that only about 1% of the

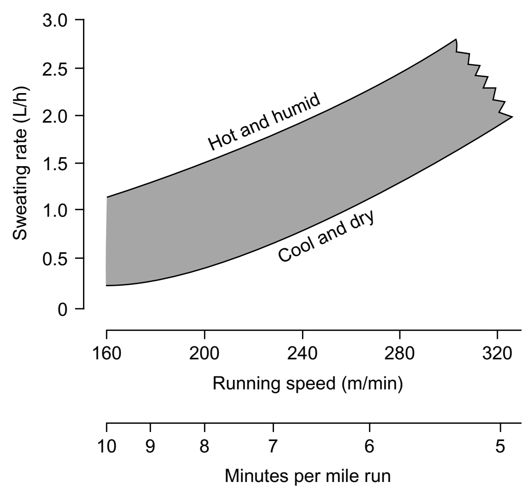

Fig. 1. The effect of exercise intensity and environmental conditions on sweat rates. Taken from Sawka and Pandolf[13] who compiled data from several laboratories for runners to approximate sweat rates (from Sawka and Pandolf,[13] with permission).

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body store of potassium is lost with dehydration to 5.8% of bodyweight. Overt potassium deficiency is rarely observed with exercise induced fluid losses. However, sodium deficiency is a more common problem associated, in particular, with long-lasting exercise and substantial fluid intakes low in sodium. A precipitous drop in plasma sodium levels during exercise is associated with impaired performance.[21] A plasma sodium level of 130 mmol/L or less (hyponatraemia) also has associated severe health risks.[22-25] It must be borne in mind that in most exercise situations fluid needs are not met and the plasma levels of sodium actually increase. This is caused by haemoconcentration, despite a net loss of sodium, since sweat is extremely hypotonic relative to plasma.

Plasma potassium levels have been observed to remain fairly stable after marathon running, even when no electrolytes were provided in supplements consumed during exercise.[26] An increase in plasma potassium levels has sometimes been observed. This is particularly the case with very high intensity, short term exercise[27] but has also been observed with ultra-endurance (67km) mountain running.[28] The inconsistency in results is most certainly related to the timing of sample taking after exercise has ceased. After exercise ceases, plasma potassium levels return to normal in a matter of minutes at rest.[29,30] The post-race samples taken several minutes after exercise, thus, may not represent the extent of potassium elevation during exercise. A portion of the increase in plasma potassium levels may be accounted for by haemoconcentration. However, most of the rise may be attributed to insufficient pumping to counteract the potassium efflux from the muscle. Release of potassium from liver and/or red blood cells might also account for (a portion of) this increase in some circumstances.[17] There is no evidence that potassium losses as a result of exercise are of a significant magnitude such that health or performance is affected.

The affectivity of supplementing potassium with sodium to restore intracellular as well as extracellular fluid, and associated performance effects, has not been thoroughly investigated. There has been some speculation that beverages containing potassium favour intracellular hydration.[31] Restoration of plasma volume is greatest with high sodium content in a beverage.[32] However, in terms of total body water,[33] no difference was observed in fluid balance when rehydrating with either KCl- or NaCl-containing fluids.

2. Fluid and Electrolyte Needs During Ultra-Endurance Exercise

Calculating fluid loss as a result of exercise is not simple. Typically net bodyweight changes, corrected for fluid intakes, are used to estimate the state of fluid balance, since most of bodyweight loss is water loss. Although a good correlation between the bodyweight loss (particularly when expressed as a percentage of total bodyweight) and performance[34] and thermal tolerance,[35] using raw bodyweight changes for fluid loss, results in some degree of error. In these, as in most studies on fluid balance, systemic errors are made in that all sources of bodyweight loss and gain are not taken into account. In most studies, no correction is made for the metabolism of endogenous substrates (carbohydrates, fat and protein) and subsequent loss of CO_2 over O_2 gained.

Recent research at our laboratory (unpublished observations) with indirect calorimetry combined with stabile isotopes, corrected for urea losses, has provided values for endogenous substrate loss. Trained cyclists, during steady state cycle ergometry at 50% maximal oxygen uptake ($\dot{V}O_{2max}$) and 80% $\dot{V}O_{2max}$ utilised endogenous substrates at a rate of 137 g/h (105 g/h carbohydrate, 19 g/h fat, 13 g/h protein) and 243 g/h (212 g/h carbohydrate, 17 g/h fat, 14 g/h protein), respectively. Calculated substrate use at 70% $\dot{V}O_{2max}$ was 208 g/h. The associated CO_2 loss over O_2 gain was 48 g/h and 86 g/h at 50% $\dot{V}O_{2max}$ and 80% $\dot{V}O_{2max}$, respectively, and calculated 70% $\dot{V}O_{2max}$ was 74 g/h. These values agree well with those of Mitchell et al.[36] who calculated bodyweight loss attributed to substrate utilisation during steady-state cycling at 30 to 80%

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$\dot{V}O_{2max}$ (1 to 2 g/min). The equation used to calculate this body loss (m_r) attributed to CO_2 expired over O_2 taken up is:

$$m_r = \dot{V}O_2 (R \cdot pCO_2 - pO_2)$$

where m_r is the mass loss due to respiratory exchange of CO_2/O_2 (g/min), R is the respiratory exchange ratio (i.e. $\dot{V}CO_2/\dot{V}O_2$), and pCO_2 and pO_2 are the densities of CO_2 [1.96 g/L standard temperature pressure dry (STPD)] and O_2 (1.43 g/L STPD).

Pugh et al.[37] also made estimates of bodyweight loss attributable to substrate metabolism. They used a figure of 0.12 g/kcal to calculate bodyweight loss associated with the exchange of O_2 and CO_2 when estimating fluid loss in marathon runners. The derived metabolic bodyweight loss in the first 4 finishers varied from 130g in the winner of the competition who finished the course in 158 minutes to 40g in the third-placed runner who finished in 164 minutes. These calculated values in runners are somewhat lower than our measured values in cyclists, although the relative intensity at which these runners were competing is unknown.

Some water would also be gained from substrate metabolism. However, this would not contribute substantial amounts. Calculations made in runners, (73 ± 8kg, $\dot{V}O_{2max}$ 64.5 ± 7 ml/kg/min) over 1 hour in a laboratory experiment at 56% $\dot{V}O_{2max}$ (≈36 ml/kg/min), 24°C, calculated metabolic water production of around 102 g/h and at 74% $\dot{V}O_{2max}$, 144 g/h with sweat rates of 1.2 and 1.25 kg/h, respectively, and respiratory losses of 66 and 84 g/h, respectively.[38]

From these rather sparse data it is estimated that the relative contribution of bodyweight loss and fluid gain due to substrate metabolism, in males, would be approximately –7 to –11% (calculated from unpublished data in our laboratory, 80% $\dot{V}O_{2max}$ cycling) and 8 to 11% (calculated from the data of Pivarnik et al.[38] with runners at 56 and 74% $\dot{V}O_{2max}$), respectively. It appears that these 2 nearly balance each other out but it may be debated as to whether the 'water gain' through metabolism is really a 'net' gain. Further research is needed in this area, with more data being collected to accurately quantify these gains and losses under differing conditions.

2.1 Case and Field Studies

There are few data collected from groups of athletes undertaking ultra-endurance exercise in a controlled setting. However, there are a number of case studies and a few field studies in which fluid intakes and bodyweight changes have been monitored during prolonged single or multi-day sporting events. These studies, for the most part, simply report bodyweight changes and fluid intakes, with no correction for metabolic bodyweight loss or water gain.

Buskirk and Beetham[39] conducted one of the earlier studies in which water loss as a result of endurance competition was monitored. They measured net bodyweight changes in 6 competitors in the 26.2 mile (≈42km) Boston Marathon. No measurement of fluid intake was undertaken but net bodyweight changes were monitored to estimate fluid loss. A net decrease in bodyweight of 3.84 ± 0.5kg, or 6.0 ± 0.8% bodyweight, was observed. The rate of net fluid loss was calculated as 1.52 L/h. These researchers also measured net bodyweight loss in competitors in an 18 mile (≈29km) road race. They observed a mean net loss of 4.1 ± 0.8%. As the authors pointed out, if a ratio of the distance covered in the 2 races is taken (26.2/18 = 1.46), this equals the figure obtained by taking the ratio of the percentage bodyweight loss in the 2 events (6.0/4.1 = 1.46). Thus, bodyweight loss was similar per distance covered. It follows that the net losses measured were primarily a result of fluid loss and, as such, increased with accumulated sweat loss over the distance. It can be concluded that fluid intakes were not substantial in these early events. In support of this, Pugh et al.[37] also observed a mean net bodyweight loss of 5.2 ± 1.2% bodyweight in 56 marathon runners with an intake of only 0.42 ± 0.35L.

Historically, the value of fluid ingestion during exercise was not widely recognised and early rules

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for athletic competition reflect this ignorance by restricting fluid intake during competition.[40] More recently, the performance value of maintaining hydration status during exercise has become widely recognised.[41] With this growing awareness, the scope of research has also broadened to include fluid intake measurement with increasing fluid intakes during endurance events and the physiological limits and affects of rehydration during endurance exercise.

One of the earliest studies of energetic demands and nutritional practices during ultra-endurance cycling was a case study of a competitor in a 24-hour road race in Britain.[42] Data were also collected from the same cyclist before the event in the laboratory in which heart rate and oxygen consumption were measured at increasing intensities and thereby the energetic demands during competition could be estimated with the use of a heart rate monitor. Relative intensity over the event was 55% $\dot{V}O_{2max}$ (38.5 ml/kg/min) and temperatures were moderate (11 to 16.5°C). This 73kg male cyclist ingested 11.09kg of nutritional supplements during the race: 5.48kg of fluids, 3.97kg of semi solids and 1.64kg of solid food. However, he lost 1.19kg of bodyweight which was thought to reflect inadequate energy consumption based upon calculated energy needs and measured intakes. This may also be related to a fluid deficiency. Even if we assume 10kg of the ingested supplements comprised water over 24 hours, the rate of intake was only 417 ml/h.

One of the most arduous events in which nutritional data were collected is the Sydney to Melbourne footrace.[43] This is a non-stop race over 1005km and must be completed within 9 days. The participant studied completed the race in 199 hours (allowing for 3 hours of sleep per 24 hours). He consumed 11L of fluids per day (1L from ingested food, 10L from ingested fluids) in small amounts every 15 to 20 minutes. His pre-race bodyweight was identical to that observed immediately upon completion of the race. Thus, this individual was able to maintain fluid balance. The mean sodium intake was 5830 mg/day, with only 860mg coming from sports drinks. The low sodium content of sports drinks was offset by a significant amount of solid food being consumed during the event, which contributed a substantial amount of sodium to the diet. Thus, the competitor was able to complete this extremely demanding endurance event successfully and in good health.

Another case study was conducted with an ultra-endurance cyclist who competed in The Race Across America, which entails cycling from the west coast of the US, some 3000km, to the east coast.[44] The race is non-stop, with minimal down time. The individual followed scheduled rest periods of ≈2 h/day and completed the race in 10 days 7 hours and 53 minutes. His diet consisted primarily of liquid meals, sports drinks, water, sports bars and a few selected solid food items (e.g. fruit). His mean daily fluid intake was 15.7 L/day. If we calculate the mean rate of intake over the non-resting hours, this results in a rate of intake of 0.71 L/h. His sodium intake averaged 10 993 mg/day. On the first day of the event, he drank ≈28L, and although this was the longest day, and in desert conditions, he had gained 2.8kg in bodyweight at 24 hours. There are indications that this competitor over-hydrated as he had gained 1.7kg over the course of the race and was back to his pre-race bodyweight within 48 hours. Other symptoms include gastrointestinal distress, bloating, nausea and a feeling of fullness. However, it cannot be discounted that some of his symptoms might also be attributed to the composition of his foods and fluids or supplements. Nevertheless, it is highly probable that this competitor over-hydrated yet had no signs of 'water-intoxication'. The high sodium intake may account for the lack of hyponatraemia and associated overt symptoms.

Saris and co-workers[45] conducted the first dietary study of Tour de France cyclists. The Tour takes place over 22 days, covering around 4000km, with 4 to 5 hours of exercise on most days except for 1 rest day and 2 time-trial days. They observed fluid intakes of up to 11.8 L/day, with a mean intake of 6.7 ± 2.0 L/day. If we assume a mean exercise

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time of 5 h/day, a rate of intake of 1.3 L/h is obtained. Unfortunately, no measurement of bodyweight just before and after a day's racing was conducted, nor was sodium intake calculated. Some measurement of bodyweight was undertaken at 9 p.m. and ≈8 a.m. On several occasions bodyweight was actually higher (1 to 1.5kg) at 9 p.m. than in the morning. This was interpreted as indicating that any fluid deficit was more than compensated by fluid intake in the 4 hours after racing. There was also no significant net change in bodyweight over the course of the Tour and the 4 cyclists monitored all successfully completed the Tour, indicating that no overt nutritional deficiencies were experienced.

Kreider[46] reported preliminary data from a laboratory based, ultra-endurance cycling simulation. Six professional cyclists participated in two, 4-day, simulated stage races, with a mean distance of 161 km/day. Measured energy expenditure was similar to that observed in Tour de France cyclists[47] and a measured oxygen consumption ranging from 68 to 85% $\dot{V}O_{2max}$ was reported. Ambient conditions were held at 30°C and 60% relative humidity. These cyclists consumed fluids at a rate of 1.2 ± 0.3 L/h. Slightly higher losses of 1.5 L/h were measured, resulting in a 1 to 1.6% bodyweight loss in each race. Although the measured fluid needs were not met, this rate of intake may be approaching the physiological limits of gastrointestinal function, and in particular, gastric emptying, in these individuals (see section 4).

Data have also been collected from a large group of ultra-endurance runners competing in the Swiss Alpine Marathon, a 67km race in Davos, Switzerland that includes a net climb and descent of 1900m. Bodyweights were recorded for 91 of the participants (5 women and 86 men) the morning of the race and immediately after the race. A mean (± standard error of the mean) decrease in bodyweight of 3.3 ± 0.2% in men and 4.0 ± 0.4% in women was observed despite a concerted effort to encourage and optimise fluid availability by organisers. Mean fluid intake over the course of the race was 3.3 ± 0.1L in men and 2.7 ± 0.3L in women. Based on mean running times of 8 hours 18 minutes (men) and 8 hours 56 minutes (women) estimated rates of fluid intake were 398 and 302 ml/h, respectively.[28]

In a recent study in New Zealand during The Goldrush 2000, multi-day, multi-sport event, data were collected from 5 individuals. In this event participants completed 11 stages of kayaking, road cycling, mountain biking and running, for a total of 344km over 3 days. This was not a continuous event, but each day had allocated stages. Once a day's stages were completed, competitors had time to rest before the next day's stages were to begin. Over the 3 days, among our participants, a mean (± standard deviation) daily bodyweight loss of 2.1 ± 0.8kg, representing 2.9 ± 1.1% bodyweight was observed (unpublished observations). This is not dissimilar from that observed in ultra-marathon running. During a 42km marathon competition, a mean decrease of 3.2 ± 1.0% in bodyweight has also been observed.[48] However, the mean fluid intake observed during one day's competition in the multi-sport, ultra-endurance race was much larger (3.5 ± 1.3L, rate 592 ± 176 ml/h) than that observed with marathon running (577 ± 46ml, rate 155 ml/h) or ultra-marathon running. In this multi-day, multi-sport race, over the 3 days, each participant began the race with a decreased bodyweight from the start of the previous day, despite substantial fluid intake after each day's racing (2.2 ± 0.9L). Rehydration to pre-race levels may not have been achieved because of low sodium intakes. The sodium intakes were very low in comparison with recommendations and sweat sodium loss based upon calculated sweat rates and estimated sweat sodium levels. A sodium deficit of 3 ± 1.3 g/day was calculated, based on dietary intakes and bodyweight changes, with an assumed sweat sodium level of 40 mmol/L. Maughan and Leiper[49] have shown very clearly the reduced rate of rehydration that occurs when sodium-poor fluids are ingested, caused by increased urine production, despite a state of dehydration.

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However, there are problems (in multi-day racing in particular), with calculating fluid balance based on bodyweight changes, as part of the bodyweight change may more reflect energy imbalance and tissue wasting and/or net loss of glycogen or fat.

Although the hours of exercise conducted per day during The Goldrush are similar to those observed in the Tour de France, the distance covered per day, not taking into account rest or time trial days, in the Tour was greater (187km). Despite the fact that the multi-sport event contained running, kayak and mountain bike stages, in which the distance covered per time was lower than with road cycling, it may be assumed that the relative intensity was greater in these professional cyclists. As a result, it may be speculated that heat production and sweat loss in these cyclists may have been greater. The professionalism of these cyclists, however, would be expected to have resulted in larger fluid intakes. The availability of fluids by support wagons and ease of consumption while road cycling would also contribute to optimal fluid consumption.

It can be concluded from the studies that have been performed with ultra-endurance athletes that sodium losses and the necessity of replacement is much more of a concern than for those exercising for shorter durations. The exact magnitude of fluid losses and, hence, needs, is difficult to measure. Much more research is required in this area to be able to quantify the bodyweight loss attributed to substrate utilisation and, thus, more accurately quantify fluid losses.

3. Effects of Dietary Intake and Exercise on Fluid Balance

The composition of the diet, and particularly the remnants of the diet that must be excreted, can influence fluid balance. The excretion of solutes from the kidney is always accompanied by water excretion caused by osmotic drag.[50] With increased protein ingestion or catabolism, urea production is increased. This increase in urea production increases urea output and hence urinary water loss.[51] Preliminary results of a study in which substrate utilisation was monitored with and without carbohydrate ingestion during intensive exercise (80% $\dot{V}O_{2max}$ cycling) indicate that protein catabolism is greater when no carbohydrate is fed (unpublished observations). Additionally, the urinary output was greater. Furthermore, when initial muscle glycogen levels are low, a greater reliance on protein to supply energy exists and a greater urea excretion is observed.[52]

Leiper et al.[53] also observed an effect of carbohydrate content of the diet on exercise induced plasma volume increase. This study involved participants walking 37km on 4 consecutive days. Fluid intake was *ad libitum*. Each individual conducted the experiment twice, once on a high carbohydrate diet (85% energy) and once on a low carbohydrate diet (25% energy). Plasma volume increased by 19 ± 4% (± standard error of the mean) on the high carbohydrate diet on day 4, but no increase was observed on the low carbohydrate diet. Both urinary sodium and urea excretion were significantly greater with the low carbohydrate diet.

The make-up of the long term diet also affects metabolic water production. A high fat diet will result in lower metabolic water production than a predominantly carbohydrate diet. Meat diets also result in acid formation in contrast to alkaline-producing, vegetarian diets.[54] This and the possible associated ketosis that can result from a high protein/fat diet of primarily meat can also increase urinary water loss. Ketosis can also be increased during or immediately subsequent to exercise, particularly in less well-trained individuals.[37] It is also increased when carbohydrate availability is limited and fatty acid metabolism and the resultant acetyl CoA cannot be metabolised quickly enough within the Krebs cycle.[55] These altered metabolic states resulting from diet and exercise can have a profound effect on fluid balance. Thus, not only exercise and acute dietary interventions during exercise, but also the long term diet can influence fluid losses.

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4. Gastrointestinal Limitations and Fluid Absorption During Exercise

Little research has been conducted on gastrointestinal function during ultra-endurance exercise, but from the numerous controlled, laboratory studies with steady-state, endurance exercise we can gain an understanding of the effect of exercise on the digestive processes and factors that influence fluid assimilation. Although most of this laboratory-based research has been conducted on exercise periods of <2 hours, the effects seem consistent over time and factors which influence gastrointestinal function are assumed to continue to play a role as exercise duration increases. However, it is not inconceivable that hormonal, metabolite and electrolyte changes as well as fluid deficit and substrate depletion may be more severe as a result of very long-lasting exercise and as a consequence gastrointestinal dysfunction may be more common.

It is acknowledged that gastric emptying can limit the rate at which fluids are available for absorption and, hence, incorporation into bodily fluids.[17] Factors that can inhibit gastric emptying of fluids include carbohydrate levels, and other nutrients including protein and fat, and to a lesser extent osmolarity, pH and intensive exercise (for review see references[17,56-58]). Increasing the volume of fluid ingested increases the gastric emptying rate; however, there appears to be an upper limit beyond which discomfort, a reduction in gastric emptying and gastric intolerance can occur. Mitchell and Voss[59] observed increasing gastric emptying rates of fluid ingestion with increasing volumes from 11.5 to 17.1 to 23 ml/kg/h, equating to 800, 1200 and 1600 ml/h, respectively, for a 70kg individual. Although mean emptying rate over a 2-hour period increased as the volume ingested increased (12, 15 and 19 ml/min, respectively), the percentage of ingested beverage emptied decreased (84, 73 and 67%, respectively). Subjective stomach fullness was also greater with the larger volume, correlating with the residual gastric volume. Half of the participants presented general gastrointestinal discomfort with the medium volume and all participants experienced some gastrointestinal discomfort with the largest volume. Two individuals presented severe gastrointestinal dysfunction (nausea and diarrhoea) with the largest volume. The lowest volume ingested ranged from 760 to 920 ml/h. Based upon residual stomach volumes and fullness ratings, this lower volume was deemed to be the most appropriate of the 3 volumes. There were, however, large inter-individual differences in gastric emptying rates and some individuals emptied the medium volume (1200 ml/h) readily and experienced no distress. A large variation in gastric emptying rate between individuals has been observed in most studies of gastric emptying. There may be an adaptation to ingestion of large volumes. It is known that there is an adaptation to glucose ingestion such that, after a high intake of glucose, the effect of glucose to reduce the gastric emptying rate is abated.[60] The large inter-individual difference that has been observed may be a result of this adaptation in some individuals. Anecdotal evidence suggests that some trained individuals may tolerate up to around 2 L/h during 2 to 3 hours of moderate exercise. This is an area worthy of further investigation.

As stated earlier, highly intensive exercise decreases gastric emptying of fluids.[61,62] Hypoxia has also been shown to decrease gastric emptying and secretion.[63] The mechanism or hormonal link has yet to be elucidated; however, it is speculated that hypoxia is the stimulus for reduction of gastric emptying with highly intensive exercise.

Typically there is feedback from the gut to the stomach and gastric emptying and intestinal absorption are coordinated such that the rate at which fluids and nutrients are provided to the intestine does not exceed the capacity for absorption. Some research indicates that exercise can decrease the absorption rate of fluids.[64,65] However, the evidence is limited and inconclusive. Maughan et al.[65] utilised deuterium-labelled water as a tracer. The resulting concentrations of deuterium in plasma, however, were not only affected by the rate of absorption but were also the result of gastric emptying. Furthermore, Fordtran and Saltin[61] ob-

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served no effect of exercise on intestinal absorption using the same triple lumen perfusion technique that Barclay and Turberg[64] used.

Exercise is known to decrease blood flow to the splanchnic area including the intestinal tract.[2,3,66-69] However, it is also known that carbohydrate ingestion increases the flow.[70] In our studies,[71] employing Doppler ultrasound, we also demonstrated that the decrease in flow observed during exercise was ameliorated when carbohydrate-containing fluids were ingested throughout exercise. This may explain the disparity in results on the effect of exercise on intestinal absorption presented above. In the study of Fordtran and Saltin[61] they fed glucose-containing fluids, whereas Barclay and Turnberg[64] fed a water and electrolyte solution. The reduction in intestinal absorption sometimes observed with exercise may be a result of decreased blood supply and hypoxia; if carbohydrates are fed, the blood supply could be better maintained. It must also be borne in mind that the absorption of glucose is an active process, coupled to sodium transport. If the levels of glucose and osmolality of the solution are not too high the absorption of water is enhanced via osmotic drag (for review see Leiper & Maughan[72]). This affect of carbohydrate may have overshadowed any direct effect of exercise in the study of Fordtran and Saltin.[61]

With exercise-induced hyperthermia and dehydration, the integrity of the gastrointestinal mucosa can become compromised, due to ischaemia, which can lead to gastrointestinal bleeding and, in the worst case scenario, endotoxaemia.[73] It is unknown if the risks are greater for ultra-endurance athletes than other sportsmen and women, but one may speculate that, if proper rehydration strategies are not put into place, the ultra-endurance athlete has a higher risk of becoming dehydrated, simply because of the length of exercise time.

5. Renal Function and Hormonal Control of Fluid Balance

Ultra-endurance athletes are not different from other endurance athletes in that they do not consume fluids sufficiently to offset fluid (primarily sweat) losses. When fluid losses are not fully compensated, blood volume decreases and plasma osmolality increases. The increased osmolality (or sodium increase) is sensed by central ‘cephalic’ as well as peripheral receptors that stimulate vasopressin (antidiuretic hormone) release from the posterior pituitary. Vasopressin, in turn, increases water reabsorption in the distal and collecting tubules of the kidney. A decrease in NaCl/extracellular fluid/blood volume and, hence, arterial blood pressure stimulates the renin-angiotensin-aldosterone system. Angiotensin II stimulates aldosterone production from the adrenal cortex which increases Na^+ reabsorption from the kidney distal and collecting tubules. The end result is an increase in salt retention, which in turn increases water retention and elevates arterial pressure. Additionally, angiotensin II also stimulates vasopressin release and increases thirst and arterial vasoconstriction. Both angiotensin II and aldosterone also increase appetite for sodium.

Atrial natriuretic peptide (ANP), conversely, is released when arterial pressure increases. It acts to decrease reabsorption of sodium in the kidneys and, hence, decrease fluid retention (see Johnson[74] for review). During exercise angiotensin II, aldosterone, vasopressin and catecholamines are increased with decreasing plasma volume, all which serve to increase blood volume and stroke volume. This response is exacerbated with dehydration. These hormones are increased in relation to the exercise intensity[75,76] and the magnitude of the water deficit.[77] Although ANP is also increased, its effects are offset during exercise by the above-mentioned hormones and may play a more important role in recovery after exercise.[78] In dehydrated rats, plasma ANP levels increase significantly within minutes after allowing them to rehydrate. This may be responsible for the diuretic (natriuretic) response often seen in humans who rapidly attempt to rehydrate after exercise. Nose et al.[79] also have observed a decrease in vasopressin levels with rapid rehydration, which would also result in diuresis.

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It is uncertain why, during ultra-endurance exercise, some individuals develop hyponatraemia. Not all cases involve fluid overload, but simply low sodium intakes. It has been shown that the development of hyponatraemia is associated with a reduced urinary output.[21] Zambraski[78] speculated that the redistribution in blood flow to the renal medulla could decrease the medullary osmolality and decrease the concentrating capacity in urine production.

With exhaustive exercise in dehydrated states, athletes can present with proteinuria and haematuria that can be associated with renal failure. During exercise, total renal blood flow is reduced and hypohydration and hyperthermia can reduce the flow by an additional 20 to 30%.[78] Acute renal failure can occur if O_2 supply is reduced to critical levels. With severe hypohydration and hyperthermia during exercise rhabdomyolysis or haemolysis can cause myoglobin or haemoglobin release and can also contribute to kidney dysfunction.

Renal function and hormones associated with fluid and sodium retention can be altered during exercise. This is associated with a reduction in blood flow to the kidneys that can, in extreme cases, become life threatening. Every effort must be taken to maintain fluid and electrolyte balance to maintain blood flow and avoid hypohydration and hyperthermia.

6. Effects of Specific Drugs on Fluid Balance

Glycerol with water ingestion before exercise has been touted as a regimen to achieve hyperhydration, possibly by enhancing plasma volume.[80] This would be of particular advantage to athletes who have difficulty drinking during their chosen sport or who lose body water at a rate that is greater than the maximal gastric emptying rate. However, the empirical evidence supporting the benefits of glycerol ingestion in terms of enhanced thermoregulation or endurance exercise performance is not conclusive. Typically 1 g/kg bodyweight of glycerol is recommended with around 1.5L of water 1 to 2 hours before exercise. A review of the studies conducted with glycerol revealed conflicting findings. Some studies demonstrated an enhanced thermoregulatory response whereas others did not.[81] Some showed an improvement in performance, but without any alteration in thermoregulatory measures, and others showed no improvement whatsoever.[81] It must be borne in mind, however, that the research to date has been done with exercise of relatively short duration (<2 hours). As Wagner[81] suggests, it may be that with ultra-endurance exercise or multi-stage events over several days, particularly in hot, humid conditions, the benefits may be more consistent and substantial. Headaches (R.J. Maughan, personal communication) and gastrointestinal complaints, however, are not uncommon[81] and any benefits must be weighed against possible ergolytic effects.

Other commonly consumed beverages include caffeine and alcohol. Both are diuretics and can inhibit rehydration attempts. Alcohol suppresses vasopressin secretion and thereby promotes free water clearance and increased urine production despite a body water deficit.[50] Caffeine, even in amounts contained in cola, is also a diuretic and can similarly thwart attempts to rehydrate by increasing urinary output.[82] Anecdotally, it has been observed that athletes often combine caffeine-containing beverage ingestion with carbohydrate and electrolyte-containing sports drink ingestion during competition. It is unknown if in this combination caffeine still induces diuresis.

7. Recommendations

Ultra-endurance athletes, as with most other endurance athletes, do not typically consume fluids in quantities to offset fluid losses during exercise. Thirst is not a good indicator of needs during exercise[83] (for review see Hubbard et al.[84]). It is frequently recommended that athletes monitor their urine colour, and if it is pale in colour the assumption is that they have consumed fluids in sufficient quantity to be in fluid balance. Kovacs et al.,[85] however, have shown that urine colour is

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not a good indicator of hydration status for up to 6 hours after dehydrating exercise. The volume of fluid that should be ingested must be determined individually and adjusted to tolerance, attempting to meet needs. Individual needs should be assessed by bodyweight changes, correcting for fluid intakes and adjusting for metabolic bodyweight losses and metabolic water production when feasible. These measurements should be made in pre-trails, conducted under conditions as similar as possible to the competitive situation. The fluid regimen determined to meet fluid needs should be attempted in practice runs, increasing the volume on sequential runs as there may be some adaptation to larger volumes. There is no advantage to ingesting small volumes. Maintenance of a large volume in the stomach, with repeated topping up, will enhance gastric emptying, providing it is tolerated.

To maximise the net rate of water absorption, the beverage should contain glucose or glucose-containing carbohydrates (e.g. sucrose, maltose and maltodextrins) at around 3 to 5% weight/volume. These should preferably be hypo-osmotic (<290 mOsm/L), or iso-osmotic (290 to 300 mOsm/L), to enhance net fluid absorption in the gut, with minimal effect on gastric emptying. Higher concentrations of carbohydrate solutions may be used, for example, 7 to 9%, when they are predominantly made of sucrose, maltose and/or maltodextrins so that the osmolality still remains low.

An advantage of a higher carbohydrate concentration is that carbohydrate oxidation can be enhanced, thereby improving endurance performance. It appears that a rate of carbohydrate ingestion of 30 to 60 g/h is required to achieve this (for review see Coyle[86]). This rate of carbohydrate ingestion is difficult to obtain if low concentrations are used unless very large volumes are ingested.

For ultra-endurance exercise it is recommended that the sodium concentration of beverages be around 30 to 50 mmol/L (1.7 to 2.9 g/L NaCl).[17] This may affect palatability and as the palatability may affect intake, the sodium content may have to be adjusted to tolerance. Again, the beverage must be trialed in an exercise situation, not at rest. What is undesirable at rest may be well tolerated during exercise and vice versa. This level of sodium inclusion is higher than that recommended by the American College of Sports Medicine in their position stand on fluid replacement.[41] They recommended inclusion of 0.5 to 0.7 g/L (8.6 to 12 mmol/L) NaCl ‘during exercise lasting more than 1 hour’. However, during very long-lasting exercise with large sweat losses the risk of hyponatraemia increases. In figure 2, a proposed model of rates of carbohydrate ingestion, and fluid and sodium concentrations suggested for supplementation during exercise lasting from 3 to 24 hours is presented.

Even with a sodium concentration of 18 mmol/L, a decrease in plasma sodium levels has been observed with exercise in the heat, albeit, less than that observed with plain water ingestion.[21] It should be noted that this occurred without overhydration. However, when only a small portion of fluid losses are replaced (50%) there is no advantage to adding sodium to the beverage.[87] After exercise or between bouts/stages, if dehydration occurs, rehydration and restoration of plasma volume is best achieved with sodium-containing beverages as urinary output occurs at a higher rate, even before positive fluid balance is achieved, with sodium-free fluid ingestion.[49] It should be taken into account that some of the sodium requirement may be met during ultra-endurance exercise and during recovery by the sodium content of consumed solid foods. Salt tablets are not recommended because of possible increases in gastric and intestinal secretions and possible gastrointestinal symptoms. The resulting hypertonicity of luminal contents could attract water into the lumen and aggravate the state of dehydration, as could hyperosmotic fluids in general.

8. Conclusion

There is still much unknown concerning the body’s tolerance and adaptation to ultra-endurance physical endeavours and the limitations to supplementation during such events. Research into this

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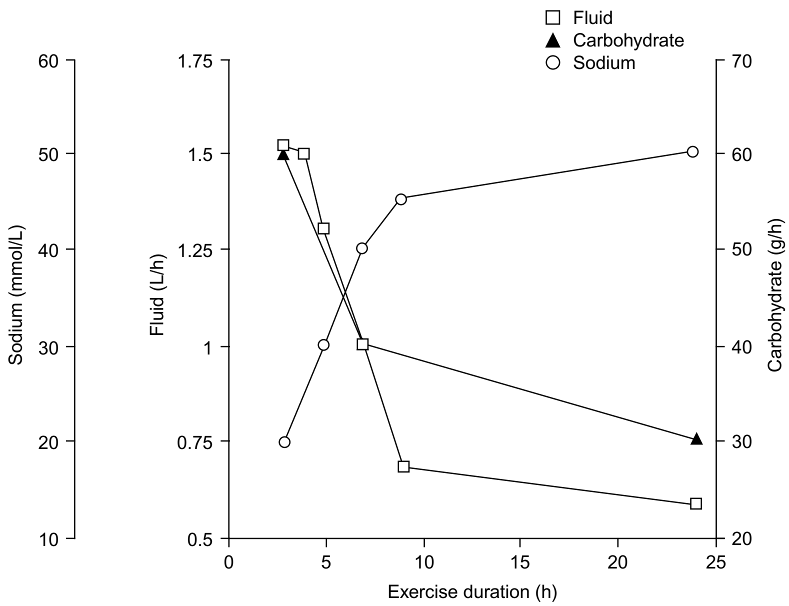

Fig. 2. Suggested rates of fluid and sodium intake and carbohydrate levels for supplementation during exercise of varying duration. These are approximate values based on data compiled from various studies presented in this article with regards to fluid loss. The suggested rates of carbohydrate ingestion are based upon data from performance studies summarised by Coyle[86] and with respect to decreasing rates of fluid loss and ingestion observed with increasing duration of exercise and the effect that would have on carbohydrate levels. Rates of sodium ingestion are based on Maughan[17] and reflect a greater sodium need in a supplement with increasing exercise duration because of reduced solid food intake and sodium from this source. These are general recommendations; varying environmental conditions, exercise intensity and individual variation will alter requirements.

type of extreme event is needed with various fluid supplementation regimens being trialed, and thermoregulatory processes as well as changes in fluid compartments being monitored.

There is wide individual variation in the physiological response to exercise and extreme conditions. Future research directed at the magnitude of adaptation to various supplementation regimens during training will be of great value to ultra-endurance athletes themselves and will add to our understanding of the processes influencing fluid homeostasis.

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