REVIEW ARTICLE

Sports Med 2005; 35 (2): 163-181
0112-1642/05/0002-0163/$34.95/0

Nutritional Considerations in Triathlon

Asker E. Jeukendrup, *Roy L.P.G. Jentjens* and *Luke Moseley*

School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Contents

Abstract

Triathlon combines three disciplines (swimming, cycling and running) and competitions last between 1 hour 50 minutes (Olympic distance) and 14 hours (Ironman distance). Independent of the distance, dehydration and carbohydrate (CHO) depletion are the most likely causes of fatigue in triathlon, whereas gastrointestinal (GI) problems, hyperthermia and hyponatraemia are potentially health threatening, especially in longer events. Although glycogen supercompensation may be beneficial for triathlon performance (even Olympic distance), this does not necessarily have to be achieved by the traditional supercompensation protocol. More recently, studies have revealed ways to increase muscle glycogen concentrations to very high levels with minimal modifications in diet and training.

During competition, cycling provides the best opportunity to ingest fluids. The optimum CHO concentration seems to be in the range of 5–8% and triathletes should aim to achieve a CHO intake of 60–70 g/hour. Triathletes should attempt to limit body mass losses to 1% of body mass. In all cases, a drink should contain sodium (30–50 mmol/L) for optimal absorption and prevention of hyponatraemia.

Post-exercise rehydration is best achieved by consuming beverages that have a high sodium content (>60 mmol/L) in a volume equivalent to 150% of body mass loss. GI problems occur frequently, especially in long-distance triathlon. Problems seem related to the intake of highly concentrated carbohydrate solutions, or hyperosmotic drinks, and the intake of fibre, fat and protein. Endotoxaemia has been suggested as an explanation for some of the GI problems, but this has not been confirmed by recent research. Although mild endotoxaemia may occur after an Ironman-distance triathlon, this does not seem to be related to the incidence of GI problems. Hyponatraemia has occasionally been reported, especially among slow competitors in triathlons and probably arises due to loss of sodium in sweat coupled with very high intakes (8–10L) of water or other low-sodium drinks.

On Wednesday September 25, 1974, the first ever triathlon was organised with 9.6km (6 miles) of running, 8km (5 miles) of cycling and 457m (500 yards) of swimming at Mission Bay in the US. One of the participants of this triathlon would become the founder of the Ironman on Long Island, Hawaii, which would catch the attention of the media and would also allow the sport to develop further. The Ironman consisted of a 3.8km swim, followed by 180km cycling and 42.2km running. More than two decades later, at the Olympic Games in Sydney in 2000, triathlon was listed for the first time as an Olympic discipline. In only one-quarter of a century, a new event had developed into a well organised and professional sport with Olympic status and worldwide participation. Triathlon is unique in that it combines three disciplines (swimming, cycling and running) and competitions last between 50 minutes (sprint distance: 500m swim, 20km cycle and 5km run) up to 14 hours (Ironman distance).

Nutritional practices and nutrition knowledge have also evolved rapidly and this review focuses on the importance of nutrition in triathlon. Obviously, the physiological demands and the nutritional challenges of triathlon depend very much on the duration of the event. Therefore, the physiological demands of triathlon at varying distances is discussed briefly and this is followed by an overview of current knowledge on preventing fatigue and health problems through nutritional measures during triathlon of varying distance.

1. Physiological and Nutritional Demands of Triathlon

Muscle glycogen and blood glucose are the most important substrates for the contracting muscle.[1-4] Fatigue during prolonged exercise is often associated with muscle glycogen depletion[5,6] and reduced blood glucose concentrations[7] and, therefore, high pre-exercise muscle and liver glycogen concentrations are believed to be essential for optimal performance. However, Noakes[8] recently suggested that it is unlikely that muscle glycogen depletion ‘alone’ limits prolonged exercise performance. Using a simulated Ironman triathlon model, Noakes predicted that after 4.5 hours of cycling at an estimated exercise intensity of 71% maximum oxygen consumption ($\dot{V}O_{2max}$), an elite male Ironman triathlete would have almost completely depleted his or her carbohydrate (CHO) stores. Interestingly, after completion of the 180km cycle, elite triathletes are able to run at a speed of 16 km/h for another 160 minutes, which represents an exercise intensity of >66% $\dot{V}O_{2max}$. At present, it is not known how it is possible that triathletes are able to complete a full marathon at such high exercise intensities when calculations suggest that muscle glycogen stores are likely to be depleted. Although speculative, it could be that other CHO sources (i.e. lactate) in addition to those in the active muscle and liver contribute to fuel oxidation during ultra-endurance exercise. It is also possible that long-distance triathletes have an increased capacity to oxidise fat, especially when muscle glycogen stores are depleted.

In addition to glycogen depletion, dehydration can also impair endurance performance.[9,10] Sweat losses occur because there is the need to dissipate the heat that is generated during exercise. Data from the Sydney Olympics report an elite triathlete generating an average of 264W over the 6-lap cycling section.[11] However, this is only a small fraction of the total energy generated by the body as gross mechanical efficiency during cycling has been observed to be around 20%.[12] Therefore, it can be estimated that the remaining ~80% or ~1100W is heat energy (discounting the proportion of energy used to maintain homeostasis). This is a large amount of heat that, if not lost from the body, would cause a large and rapid rise in deep-body temperature. One of the body's responses to this rise in temperature is the activation of sweat glands to release sweat onto the surface of the skin for evaporative cooling. Sweat rates will increase with increased metabolic rate (i.e. exercise intensity) and impaired dissipation (i.e. hot, humid, lack of wind) but can also be highly variable between individuals. The loss of body water will result in a reduction in plasma volume, stroke volume and skin blood flow, which can lead to an inability to maintain thermal homeostasis and reduced performance.[13] Therefore, the nutritional challenge is to replace body water and prevent fatigue.

The reduction in performance is thought to be due in part to reduced muscle blood flow. In addition, it has been observed that muscle glycogen breakdown is increased with increasing core body temperature,[14-16] although others have shown that fatigue may occur before muscle glycogen is depleted.[17] More recently, the effect of hyperthermia on performance has been suggested to be related to more central mechanisms, related to the attainment of a high deep body temperature.[18,19] There is general agreement that the reductions in body water need only be small for negative performance effects to occur.[9,20] Walsh et al.[21] reported that reductions in body mass of 1–2% resulted in a 44% reduction in performance while a 3% hypohydration caused by diuretics produced a 3–5% reduction in 1500–10 000m running times.[22] Severe hypohydration may reduce the rate of gastric emptying while increasing the likelihood of gastrointestinal (GI) problems,[23] therefore, hypohydration may become a self-perpetuating problem. Most importantly, while hypohydration and hyperthermia alone have negative effects on performance, their combination is particularly serious, both in terms of performance and health.[24]

2. Nutrition and Triathlon Performance

Since physiological demands can be extremely high and hyperthermia, dehydration and CHO depletion can all affect performance, it is important to combat these dangers with adequate nutritional measures. This section will discuss whether it is possible to prevent the reductions in performance and where possible, guidelines will be formulated.

2.1 Pre-Competition

2.1.1 Carbohydrate (CHO) Loading

The effect of high-CHO diets and elevated muscle glycogen levels on exercise performance has recently been summarised in a review by Hawley et al.[25] It was suggested that supercompensated muscle glycogen levels can improve performance (i.e. time to complete a predetermined distance) by 2–3% in events lasting >90 minutes. However, there seems to be little or no performance benefit of supercompensated muscle glycogen levels when the exercise duration is <90 minutes. Triathletes preparing for Olympic-distance triathlons and certainly long-distance triathlons are likely to benefit from CHO loading regimens, although this has never been tested directly.

The 'classical' glycogen supercompensation protocol as was first described by Bergström et al.[5] consisted of a glycogen-depleting exercise bout followed by a 3-day low-CHO, high-fat/protein diet and a 3-day high-CHO diet. This procedure was found to increase muscle glycogen stores by more than twice the normal glycogen concentration. More importantly, pre-exercise muscle glycogen concentration was directly related to endurance capacity (time to fatigue). Although this 'classical' glycogen supercompensation protocol was successful in elevating muscle glycogen stores, the practical significance of such a dietary-training regimen is questionable as this regimen is very strenuous and may promote both injury and GI distress.

A study by Sherman et al.[26] demonstrated that muscle glycogen stores could be supercompensated with less drastic exercise and dietary manipulations. Sherman et al.[26] showed that 3 days of a moderate CHO intake (50% of energy intake coming from CHO [En% CHO] and ~5g of CHO/kg bodyweight (BW)/day) followed by 3 days of a high-CHO intake (70 En% CHO and ~8g of CHO/kg BW/day) in combination with a tapered training regimen also resulted in supercompensated muscle glycogen concentrations.

Recently, Coyle et al.[27] demonstrated that consumption of a high-CHO diet (88 En% CHO and ~12.5g of CHO/kg BW/day) during six consecutive days of training can lead to extremely high muscle glycogen concentrations. The data of Coyle et al.[27] suggest that supercompensated glycogen levels can be achieved without performing a glycogen-depleting exercise bout first and without tapering-down of the exercise training. This is further supported by work from Fairchild et al.[28] and Bussau et al.[29] In these studies, it was shown that when a short-term high-intensity exercise bout[28] or even complete physical inactivity[29] was followed by a 1-day high CHO intake (~10.5g of CHO/kg BW/day), supercompensated muscle glycogen stores were attained within only 24 hours. This is an important finding as this 1-day CHO loading protocol allows triathletes to follow their normal training preparations up until the day prior to competition. Of note, supercompensated muscle glycogen levels can be maintained for at least 3 days when triathletes do not exercise and consume a moderate CHO intake (4–5g of CHO/kg BW/day).[30] Thus, if triathletes are not willing or not able to consume a high-CHO diet the day before competition, they could start with the 1-day CHO loading protocol several days before competition then rest afterwards while consuming a moderate-CHO diet.

In summary, well trained triathletes who are aiming for high muscle glycogen concentrations prior to competition should make sure that their carbohydrate intake is high (10g of CHO/kg BW/day) at least 1 day before the event and they should make sure that 1–4 days before the event, muscle glycogen concentrations have been reduced significantly. This can be achieved by a longer training session at moderate intensity[26,27] or a short-term high-intensity exercise bout.[28,29]

2.1.2 CHO Ingestion 3–4 Hours Before Exercise

Pre-exercise CHO ingestion may enhance CHO availability during prolonged exercise by increasing muscle and liver glycogen stores prior to exercise,[31] or by providing a source of glucose in the gut for later release into the bloodstream. Ingestion of CHO before exercise may be particularly important when exercise is undertaken in the morning after an overnight fast and hence liver glycogen stores are substantially depleted, or when there has been inadequate time for complete restoration of muscle and liver glycogen stores following a previous exercise session.[32] In addition, some triathletes only tolerate relatively small amounts of CHO and/ or fluids during strenuous exercise and hence they may not always be able to consume sufficient amounts of CHO 'during' exercise. Furthermore, in some cases, the availability of CHO sources (i.e. CHO supplements provided by the race organisers) may be limited and this can also lead to low CHO intakes. Therefore, ingestion of substantial amounts of CHO (i.e. 200–300g) 3–4 hours before exercise may be an effective strategy to enhance CHO availability during a subsequent exercise bout.[33,34] More importantly, CHO meals or beverages when ingested 3–4 hours prior to exercise have been shown to result in improved endurance capacity.[35,36] Therefore, it is recommended for triathletes to consume a CHO meal or snack (200–300g of CHO) 3–4 hours before the start of exercise. However, it should be noted here that triathletes must choose CHO meals that are easily digested and do not cause GI discomfort during exercise. This is even more important when CHO is consumed shortly before exercise (<60 minutes) as there is less time for (complete) digestion and absorption of foods.

2.1.3 CHO Ingestion <60 Minutes Before Exercise

Although consumption of high-CHO diets in the days before exercise[5] as well as ingestion of CHO meals 3–4 hours prior to exercise[35,36] can have positive effects on exercise performance, suggestions have been made that the intake of CHO 30–60 minutes before exercise may adversely affect performance.[37,38] Glucose ingestion in the hour before exercise can result in hyperglycaemia and hyperinsulinaemia, which is often followed by a rapid decline in blood glucose 15–30 minutes after at the onset of exercise,[37,39,40] referred to as rebound hypoglycaemia. The fall in blood glucose is most likely the result of a reduced liver glucose output and an increased muscle glucose uptake.[41] Furthermore, hyperinsulinaemia following glucose ingestion inhibits lipolysis and the availability of free fatty acids for oxidation[37,39,40] and this may subsequently lead to increased muscle glycogen utilisation. Hypoglycaemia and depletion of muscle glycogen stores have been associated with the onset of fatigue during prolonged exercise. Therefore, pre-exercise CHO feedings in the hour before exercise may have the potential to impair performance. However, only two studies have found reduced performance capacity,[37,38] while the majority of studies reported no change[42-46] or an increased performance[47-51] following pre-exercise CHO ingestion. Furthermore, a rebound hypoglycaemia in the early stage of exercise seems to be of little functional significance as this does not affect exercise performance.[42,49,52-56] This suggests that there is no need to avoid CHO intake in the hour before exercise. It is interesting to note here that rebound hypoglycaemia occurs in some triathletes but not in others.[57,58]

Kuipers et al.[58] suggested that the occurrence of rebound hypoglycaemia in trained triathletes is related to a high insulin sensitivity. However, we have recently shown that trained individuals who developed rebound hypoglycaemia did not have a higher insulin sensitivity compared with individuals who did not show rebound hypoglycaemia.[57] Therefore, it is unlikely that insulin sensitivity plays an important role in the prevalence of rebound hypoglycaemia in trained athletes. It may be argued that there are some athletes who are very ‘sensitive’ to low blood glucose levels and for them exercise-induced hypoglycaemia may be a major factor contributing to fatigue. These metabolic disturbances may be attenuated by choosing pre-exercise CHO sources with a low glycaemic index because these result in more stable blood glucose and insulin responses during subsequent exercise.[44,46,53,59] Another approach to minimise the glycaemic and insulinaemic responses during exercise is to delay CHO feeding until 5–15 minutes before the start of exercise.[55] Of note, the metabolic and performance effects of CHO ingestion shortly before exercise (<15 minutes) are very similar to those observed when CHO is fed during exercise (see section 2.2.1).

2.1.4 Fluid Ingestion Before Exercise: Euhydration or Hyperhydration

As discussed in section 1, dehydration can compromise exercise performance and it is, therefore, important to start exercise in a euhydrated state. Triathletes are generally recommended to drink about 400–600mL of fluid 2 hours before the start of exercise.[60] Consuming this amount of fluid promotes adequate hydration and allows time for the excretion of excess ingested water. Furthermore, it is believed that triathletes who have difficulty drinking sufficient amounts of fluid during exercise or who lose body water at high rates (i.e. during exercise in hot conditions), may benefit from hyperhydration. Hyperhydration has been suggested to improve thermoregulation and exercise performance, especially in the heat.[61] The thermoregulatory advantages associated with hyperhydration are reduced increases in core temperature and higher sweat rates during exercise.

Several studies have attempted to induce hyperhydration by over-drinking water with or without electrolytes. However, often hyperhydration achieved by over-drinking only produces transient expansion of body water because most of the fluid overload is rapidly excreted by the kidneys. More recently, studies have focused on the use of glycerol solutions to induce hyperhydrations. Glycerol is a naturally occurring metabolite in the human body that is evenly distributed within and between all cells at low concentrations (<1 mmol/L).[62] The rationale behind glycerol supplementation is due to its water-binding properties. Typically glycerol-mediated hyperhydration is performed by ingesting ~1 g/kg BW/day of glycerol with or followed by a large volume of fluid (~2L) 1.5–2.5 hours before exercise.[61,62] It has been shown that in resting conditions, glycerol-mediated hyperhydration can result in a 400–600mL greater fluid retention than water-mediated hyperhydration. The difference in total body water content between glycerol- and water-mediated hyperhydration at rest does not occur until 2–3 hours after glycerol ingestion.

Studies that have examined the effects of hyperhydration on thermoregulation and exercise performance (in the heat) have produced inconclusive results. This is most likely due to methodological differences between studies including variations in exercise protocol, environmental conditions, pre-exercise hydration status, and dosage and timing of water and/or glycerol supplementation. Recent research that controlled for these confounding variables has shown that when euhydration is maintained during exercise, pre-exercise hyperhydration (water or glycerol) does not further improve thermoregulation and/or performance.[61] Furthermore, there appears to be no difference in thermoregulation and performance between glycerol- and water-induced hyperhydration. The advantages of hyperhydration reported in the literature are most likely due to the fact that by the end of exercise the ‘control group’ is more dehydrated. It should be noted that triathletes often do not drink sufficient amounts of fluid to replace sweat losses during prolonged exercise and hence they may become dehydrated in the early stage of exercise.

Of interest is a recent study by Coutts et al.[63] who investigated the effects of glycerol hyperhydration on Olympic-distance triathlon performance in high ambient temperatures. Ten trained triathletes completed a 1500m swim in a 25m pool, a 40km cycle on the road and a 10km run on the road on two occasions (once in hot conditions and once in warm conditions). The group was split in two with half of them receiving glycerol and the other half water. It was found that following glycerol hyperhydration the increase in completion time between hot and warm conditions was significantly less than in the placebo group. Although this study may lack some statistical power and there may have been difficulties in supplying the triathletes with glycerol in a double-blind fashion, the results seem to indicate that glycerol has a protective effect against the negative influences of hot environmental conditions. It must be noted that not all studies found positive effects of glycerol ingestion. For example, Marino et al.[64] recently found no difference in 1-hour cycling performance in the heat following glycerol ingestion.

It is likely that triathletes who are expecting to become severely dehydrated during exercise may benefit from hyperhydration (glycerol or water). However, triathletes who wish to experiment with glycerol supplementation should be aware of the potential adverse effects that may be associated with it, such as headache and GI discomfort.[65]

2.2 During Competition

2.2.1 Maintaining CHO Supply During Exercise

It is has been known for some time that CHO ingestion during exercise improves endurance capacity by maintaining blood glucose concentrations and high rates of CHO oxidation.[7,66] Originally, it was believed that CHO feeding could only improve exercise performance when the exercise was approximately 2 hours or longer, allowing time for absorption of the CHO. However, more recently, several studies have demonstrated that even with exercise as short as 1 hour, CHO intake may have an advantage.[67-69] In a recent study by Kimber et al.,[70] the average CHO intake during an Ironman-distance triathlon was 1.0 g/kg BW/hour in female triathletes and 1.1 g/kg BW/hour in male triathletes. They achieved these CHO intakes by ingesting very large amounts of CHO during cycling (approximately 1.5 g/kg BW/hour) with most of the intake during the cycling being almost three times as high as during running leg. An interesting observation was that in male triathletes the CHO intake during the triathlon was positively correlated with finishing time. Such a relationship could not be demonstrated in females.

There appear to be some interesting differences between cycling and running that could be important in triathlon. With CHO feeding during cycling, it has repeatedly been shown that muscle glycogen breakdown is unaffected.[7,71,72] During running, however, there are suggestions that muscle glycogen breakdown is reduced in particular in type I muscle fibres.[73] Therefore, CHO feeding results in improved performance in cycling and running, although the mechanism by which this occurs may not necessarily be the same. This issue is discussed in more detail in an excellent review by Tsintzas and Williams.[74]

Studies using (stable and radioactive) isotope methodology have demonstrated that not all CHOs are oxidised at similar rates and hence they may not be equally effective. Glucose, sucrose, maltose, maltodextrins and amylopectin are oxidised at high rates. Fructose, galactose and amylose have been shown to be oxidised at rates that are generally 25–50% lower. Combinations of multiple transportable CHO may increase the total CHO absorption[75] and total exogenous CHO oxidation[76-78] but more research is needed. Increasing the CHO intake up to 1.0–1.5 g/min will increase the oxidation rate up to about 1.0–1.1 g/min.[79] A further increase of the intake rate, however, will not further increase the oxidation rates.[79] The most remarkable conclusion that was drawn in a recent review is probably that exogenous CHO oxidation rates do not exceed 1.0–1.1 g/min.[79] There is convincing evidence that this limitation is not at the muscular level but most likely located in the intestine or the liver. Intestinal perfusion studies seem to suggest that the capacity to absorb glucose is only slightly in excess of the observed entrance of glucose into the blood and the rate of absorption may thus be a factor contributing to the limitation. The liver, however, may play an additional important role, in that it provides glucose to the bloodstream at a rate of only ~1.0 (or 1.0–1.3) g/min by balancing the glucose from the gut and from glycogenolysis/gluconeogenesis. It is possible that when large amounts of glucose are ingested, absorption is a limiting factor and the liver will retain some glucose and will thus act as a second limiting factor to exogenous CHO oxidation.

Interestingly, recent studies from our laboratory have shown that a mixture of glucose and sucrose[77] or glucose and fructose[77] when ingested at a high rate (1.8 g/min) leads to peak oxidation rates of ~1.2–1.3 g/min and results in ~20–50% higher exogenous CHO oxidation rates compared with the ingestion of an isocaloric amount of glucose. This finding was attributed to the fact that these CHOs are absorbed, at least in part, by different intestinal transport mechanisms and hence there may be less competition for absorption. A faster rate of intestinal CHO absorption might increase the availability of exogenous CHO in the blood stream for oxidation. It remains to be investigated whether higher exogenous CHO oxidation rates will lead to improved exercise performance.

Because exogenous CHO oxidation rates from single CHOs do not exceed 1.0–1.1 g/min it has been recommended to ensure a CHO intake of 60–70 g/hour.[79] A higher CHO intake may result in GI problems, a lower intake may result in a suboptimal CHO delivery. It should be noted that during exercise in hot conditions slightly less CHO should be consumed (50–60 g/hour) as the oxidation of ingested CHO is lower (~10%) in the heat compared with a cool environment.[16]

2.2.2 Maintaining Fluid Balance During Exercise

The prevention of hypohydration during exercise is of prime importance and, in general, triathletes seem well informed about the dangers. Triathlon poses unique nutritional challenges; fluid intake during the swim phase is not possible and fluid intake during the run can cause GI problems. In addition, swimming and cycling prior to running in triathlon are reported to cause increased oxygen cost (decreased economy) and larger decreases in body mass and plasma volume compared with the run alone.[80] Water balance is not only determined by sweat losses but also affected through the saturation of air in the lungs and through the metabolism of fat and CHO stores in the body. Recently, Rogers et al.[24] attempted to account for all factors involved in water turnover during an Ironman-distance race and reported mean sweat rate to be 940 mL/hour, urinary losses at 41 mL/hour and respiratory losses to be 88 mL/hour. With a volume of water in the region of 1.1L being lost every hour, triathletes need to be aware of why and how they need to maintain fluid balance.

The cycling section represents the best opportunity to ingest fluid during a triathlon. During this stage, fluid is normally most readily available and ingestion is normally least disturbing to performance. Research has indicated that when cycling in the heat, performance is improved by ~6% when a larger volume of fluid was ingested (1330 ± 60 vs 200 ± 10mL) during exercise prior to the performance trial.[68] However, the ingestion of large volumes may not always be advisable.[81,82] Gastric emptying is thought to be negatively affected at intensities over 70% $\dot{V}O_{2max}$.[83,84] Robinson et al.[82] reported that the maximum rate of intestinal fluid absorption is 0.5 L/hour when cycling at 85% peak oxygen consumption ($\dot{V}O_{2peak}$), an intensity close to that seen during the cycling sections of Olympic-distance triathlons. In the study of Robinson et al.[82] subjects consumed ~1.5L of water in 1 hour while cycling at 85% $\dot{V}O_{2peak}$. It was estimated that ~0.9L remained in the stomach and the intestine at the end of exercise and subjects complained of abdominal fullness, and so clearly the ingestion of very large volumes may not be advantageous.

Perhaps the best advice is for triathletes to weigh themselves to assess fluid losses during training and racing and limit weight losses to 1% during exercise lasting longer than 1.5 hours.[85] In the absence of such planning, concrete advice is difficult to give as differences between individuals, race distances, course profiles and environmental conditions will confound any suggestions. However, consuming 100mL every 10 minutes would provide 600 mL/hour and would go some way to limiting the effects of dehydration.[86]

Fluids empty from the stomach in an exponential manner[87] with an initial rapid phase of emptying. In fact, one of the major stimulants of gastric emptying is the volume in the stomach, with a positive relationship between stomach volume and rate of emptying from the stomach.[83,88-90] Rehrer et al.[91] illustrated this by regular repeated ingestion of fluids, the volume in the stomach is 'topped up', therefore, maintaining the initial rapid rate of fluid delivery to the intestine. In that study, subjects consumed a bolus of fluid before exercising to prime the stomach before ingesting 150mL every 20 minutes.

The absorption of water in the intestine is primarily passive, where water passes across the intestinal membrane due to an osmotic gradient.[92] Glucose is actively transported across the intestinal membrane, a process aided by the inclusion of sodium[93] and it has been suggested that water is also co-transported during this process.[94] Indeed, isotonic CHO plus sodium solutions are absorbed more rapidly than either water and/or isotonic sodium-only solutions.[95,96] However, other authors have reported no additional effect of sodium inclusion on water absorption in an already isotonic CHO solution.[97,98] Despite this, the addition of sodium and CHO to sports drinks is widely recommended to enhance the absorption of water.[85,99]

Hypertonic solutions tend to delay water absorption in the intestine as water instead flows into the intestine to dilute the solution before water is absorbed.[85,100,101] Additionally there is contention as to whether hypertonic solutions reduce the rate of gastric emptying, Rehrer et al.[91] found repeated ingestions of a hypertonic CHO solution resulted in a reduced rate of gastric emptying after 20 minutes compared with an isotonic solution and consumption of a hypertonic beverage during triathlon competition has been related with GI problems.[100] However, the majority of studies suggest energy density is considerably more important in determining gastric emptying when solutions with an osmolality close to those normally found in sports drinks are used.[90,102,103]

The importance of beverage taste should not be underestimated. Passe et al.[104] reported that drink acceptability affected voluntary ingestion volumes during endurance exercise. While this is not surprising, even when offered the CHO drink judged least acceptable in terms of taste, subjects consumed more of the flavoured CHO drink than water. In addition, it was suggested that perception of drink acceptability depended on whether the subject was exercising or resting, underlining the importance of practising race-day strategies during training.

In summary, a balance must be met between the goals of maintaining hydration status and providing CHO to the working muscle. The rate of fluid absorption is closely related to the CHO content of the drink with high CHO concentrations compromising fluid delivery. The optimum CHO concentrations seem to be in the range of 5–8% as both fluid and CHO delivery will be high.[105-108] CHO should be ingested at a rate of 60–70 g/hour while fluid intake should aim to minimise any weight loss. In all cases, a drink should contain sodium (10–30 mmol/L[85,99]) for optimal absorption and prevention of hyponatraemia.

2.3 Recovery

2.3.1 CHO Intake After Exercise

Muscle glycogen is of primary importance for prolonged endurance exercise and hence the repletion of glycogen constitutes an important role of the post-exercise recovery process. Depending on the extent of glycogen depletion and provided that at least 8g of CHO/kg BW/day is consumed, complete restoration of these glycogen stores can occur within 24 hours.[109-111] Furthermore, it has been shown that when a high-CHO diet (9–10g of CHO/kg BW/day) is consumed between two exercise sessions separated by a 22.5-hour recovery period, intermittent[112] and endurance exercise[113] capacity is maintained or even improved. It should be noted here that muscle glycogen synthesis is impaired for several days after exercise that causes muscle damage (i.e. eccentric exercise), such as after a marathon.[114-116] Suggestions have been made that the impaired muscle glycogen synthesis following eccentric exercise is due to increased glucose uptake by inflammatory cells and hence less glucose is available for glycogen synthesis in previously exercised muscle.[115] Therefore, after extensive running, complete recovery of muscle glycogen stores may take longer than 24 hours despite the intake of a high-CHO diet.

When CHO intake is adequate (≥7.0 g/kg BW/day), co-ingestion of moderate amounts of fat and protein do not appear to have an effect on muscle glycogen storage during 24 hours of recovery after prolonged exercise.[117] Furthermore, post-exercise glycogen storage during the first 24 hours of recovery is not affected by the frequency of food intake as long as the total amount of CHO ingested is sufficient.[118] Practical issues such as appetite and the availability of food may both determine how much and how often food is consumed, and whether sufficient CHO intake is met in order to replenish glycogen stores. CHO foods (and drinks) with a moderate to high glycaemic index are highly recommended after exercise because they might result in higher glycogen synthesis rates than low glycaemic index CHO foods.[119]

From the above discussion it can be concluded that the amount of CHO consumed is the most important dietary factor influencing muscle glycogen synthesis. Furthermore, muscle glycogen concentrations can return to pre-exercise values within 24 hours after exercise when sufficient CHO (8–10 g/kg BW/day) is consumed. Since triathletes seldom compete on two consecutive days there seems to be sufficient time between races for complete recovery of muscle glycogen stores. Of note, athletes (triathletes in particular) often train more than once per day, and some events require qualification <8 hours before the actual event. Although it is unlikely that muscle glycogen stores can be completely resynthesised within hours, appropriate nutritional practices can help to optimise the rate of glycogen storage in the often limited time available for recovery. Section 2.3.2 summarises nutritional strategies to obtain maximal muscle glycogen synthesis rates in the early hours post-exercise. For more detailed information on this topic the reader is referred to a recent review by Jentjens and Jeukendrup.[120]

2.3.2 The Early Hours Post-Exercise (<8 Hours)

The rate of muscle glycogen synthesis in the hours immediately after exercise is largely dependent on the amount and frequency of CHO intake, the timing of CHO consumption and the type and form of CHO. Furthermore, suggestions have been made that the addition of certain protein and/or amino acids to a CHO supplement can increase the rate of muscle glycogen synthesis. The role of protein intake for recovery is discussed in section 2.3.3.

Probably the most important factor determining the rate of muscle glycogen synthesis is the quantity of CHO consumed after exercise. When no CHO is ingested after exercise, muscle glycogen synthesis rates are very low. However, very high muscle glycogen synthesis rates are observed when 1.0–1.2g of CHO/kg BW/hour is ingested at frequent intervals during a 3- to 5-hour recovery period.[121-123] From the data available in the literature, it seems reasonable to conclude that maximal glycogen synthesis rates occur at a CHO intake of ~1.2 g/kg BW/hour (or 75–90g of CHO per hour). It should be noted that the highest rates of muscle glycogen synthesis rates have been found in studies in which CHO supplements were provided at regular intervals (every 15–30 minutes). Thus, in the early hours post-exercise, consumption of small repetitive CHO feedings appears to be more beneficial for high muscle glycogen synthesis rates than ingestion of one or two larger CHO feedings.[120,123]

The pattern of muscle glycogen synthesis following glycogen-depleting exercise occurs in a biphasic manner. Initially, there is a rapid phase of glycogen synthesis (insulin-independent phase), which generally lasts between 30–60 minutes. Following this rapid phase of glycogen synthesis, muscle glycogen synthesis occurs at a much slower rate (slow phase or insulin-dependent phase) and in the presence of CHO availability and high insulin concentrations this phase can last for several hours.[124] A study by Ivy et al.[125] clearly demonstrated that muscle glycogen synthesis rates were almost twice as high when a CHO supplement was ingested immediately post-exercise compared with 2 hours later. It is, therefore, recommended that triathletes consume a CHO as soon as possible after exercise as this may increase the rate of muscle glycogen storage.

Studies have found similar rates of muscle glycogen synthesis after glucose and sucrose ingestion.[126] However, ingestion of an equal amount of fructose results in much lower glycogen synthesis rates.[126] This is most probably due to a slower absorption rate of fructose from the intestine[127,128] and the fact that fructose requires conversion to glucose by the liver before it can be metabolised in the skeletal muscle.[128-130] It is recommended to consume CHO foods with a moderate to high glycaemic index because this might result in higher glycogen synthesis rates than ingestion of CHO foods with a low glycaemic index. Whether the CHO supplement is in solid or liquid form does not seem to affect the rate of muscle glycogen synthesis.[109,131] CHO beverages are often recommended to triathletes because they also provide a source of fluid that may be beneficial for rapid rehydration (see section 2.3.4). Furthermore, when appetite is suppressed immediately after exercise, there may be a preference for drinking fluids rather than eating solid foods.

2.3.3 Protein and Amino Acids

Several studies have shown that the addition of certain proteins and/or amino acids to a CHO supplement can increase muscle glycogen synthesis rates by 40–100%, most probably as a result of an enhanced insulin response.[123,132] Insulin stimulates muscle glucose uptake and activates glycogen synthase,[133] the rate-limiting enzyme in glycogen synthesis. However, we have recently demonstrated that when the total CHO intake is high (1.2 g/kg BW/hour) the presence of a protein-amino acid mixture does not further increase the rate of muscle glycogen synthesis despite a much higher insulin response.[121] This suggests that when sufficient CHO is consumed (1.2 g/kg BW/hour) there is no need for protein and/or amino acid ingestion as this does not lead to higher muscle glycogen synthesis rates. In addition, most studies have been performed with unpalatable protein hydrolysates and amino acids, which triathletes would not normally consume. When such protein and amino acids are added to a CHO supplement this might refrain the triathlete from sufficient CHO intake and hence optimal muscle glycogen synthesis rates are not reached.

It should be noted that there is some evidence that amino acid ingestion in combination with[134] and without[135] CHO may increase post-exercise protein synthesis and net muscle protein balance (protein synthesis minus protein degradation).[136] An increased protein accretion and an increased availability of essential amino acids might contribute to faster tissue growth and repair. Prolonged eccentric exercise, such as marathon running or downhill running, is known to induce severe muscle damage and hence nutritional supplements that would speed up the recovery of damaged muscle could be of benefit to the triathlete. The importance of post-exercise amino acid and/or protein ingestion to stimulate net muscle protein anabolism and the consequence this might have on the repair of exercise-induced muscle damage remain to be elucidated.

2.3.4 Restoration of Fluid Balance

The restoration of fluid balance after exercise is an important part of the recovery process and this is even more important following exercise in hot and humid conditions. It has been suggested that effective rehydration after exercise can only be achieved when both sweat loss and the sodium lost in sweat are replaced.[137]

Shirreffs et al.[138,139] suggested that at least 150% of the amount of fluid lost during exercise is needed to ensure complete rehydration. When smaller volumes of fluid are consumed, equal to sweat losses, optimal rehydration may not be achieved due to ongoing urine production. Of note, when the sodium content of the drinks is low (23 mmol/L) even ingestion of large drink volumes (equal to 1.5- to 2-times the sweat loss) is not adequate to restore fluid balance.[139] There seems be an inverse relationship between the sodium content of the ingested fluid and urine production, which suggests that more fluid is retained when beverages are ingested with a moderate to high sodium content (>50 mmol/L). Numerous studies have indicated that water is not the most effective rehydration beverage.[140-143] Costill and Sparks[140] illustrated that ingestion of plain water increased urine output and resulted in less effective restoration of net fluid balance compared with including glucose and electrolytes in the beverage.[140] Maughan and Leiper[144] compared the effect of either 2, 26, 52 or 100 mmol/L of sodium in drinks given after dehydrating exercise. Subjects were exercised in the heat until they had lost 1.9% of their body mass. After resting for 30 minutes, subjects consumed a volume equivalent to 1.5-times of the mass lost over the next 30 minutes. All urine produced over the 5.5 hours following drink ingestion was collected. Urine production was inversely related to sodium content, the drink with the largest amount of sodium resulting the least urine production and the greatest net water gain. Only the 56 and 100 mmol/L drinks resulted in a restoration of whole-body sodium content, with the 100 mmol/L drink increasing whole-body sodium above pre-exercise levels. Plasma volume restoration was also related in a similar fashion to beverage sodium concentration.

The mechanism by which sodium exerts a positive effect on net fluid balance during post-exercise rehydration has been proposed to be 2-fold.[145] The first effect of sodium is to stimulate glucose absorption in the small intestine as described in section 2.2.2. The second proposed effect of sodium is to prevent the dilution of plasma sodium that would otherwise occur with the ingestion of plain water. Low plasma sodium concentrations inhibit the production of anti-diuretic hormone (ADH, vasopressin) and result in increased urine volume. Sodium-containing drinks may also enhance post-exercise fluid balance compared with water alone by increasing the sensation of thirst and, therefore, voluntary fluid intake.[142,146]

While it seems clear that the inclusion of sodium prevents plasma sodium dilution and is a positive factor in post-exercise rehydration, the inclusion of potassium does not seem to have a similar effect. Sodium is the major ion of the extracellular space and thus inclusion of sodium in beverages prevents the plasma sodium dilution that would occur if water alone were drunk. Potassium, however, is the major ion of the intracellular space, and it has been theorised that inclusion of potassium in a rehydration beverage might increase the retention of fluid within the intracellular space. Experimental evidence, however, does not support this hypothesis. Maughan et al.[147] investigated the effect of glucose alone, potassium alone, sodium alone or all three in combination, on rehydration following dehydration by ~2% from exercise in a hot environment. The addition of either sodium or potassium alone increased net fluid balance compared with glucose alone but there was no further improvement with all three in combination. Additional evidence regarding the lesser importance of other electrolytes compared with sodium comes from the examination of the electrolyte concentration of sweat and urine. The concentration of potassium, chloride and magnesium are more dilute in sweat than their concentration in their primary body compartment and, therefore, tend to become more concentrated due to sweating.[148] Costill[148] reported that even repeated days of heavy sweating did not result in magnesium or potassium deficits. In contrast, some researchers have suggested that hypomagnesaemia may be a problem in long-distance triathlon.[149] At present, however, research does not seem to indicate additional benefit of including additional electrolytes other than sodium in rehydration beverages.

It is clear from the above discussion that sodium is an important ingredient in rehydration drinks. However, one disadvantage of beverages that contain high sodium concentrations is that these drinks are not very palatable and this may prevent the triathlete from consuming sufficient amounts of fluid. Palatability is a major issue when large volumes of fluid need to be consumed. Therefore, the triathlete should choose a rehydration drink that contains sufficient amounts of sodium and does not compromise fluid intake. Addition of moderate amounts of CHO will improve palatability and may increase the rate of intestinal uptake of sodium and water. However, beverages with high CHO concentrations (>10%) will reduce fluid absorption and hence the availability of fluid for rapid rehydration. When an triathlete is severely dehydrated after exercise, the restoration of fluid balance has the priority above glycogen restoration and hence a more diluted CHO beverage should be consumed.

There is some evidence that the ingestion of solid food in combination with plain water may be of additional benefit than ingestion of a similar volume of sports drink. In a study of Maughan et al.[150] the volume of fluid in a solid food plus water trial was equivalent to that in a fluid-only trial but the volume of urine produced in the solid-food trial was smaller, resulting in improved net fluid balance. However, the solid food contained greater sodium and potassium than the sports drink and it is likely that this factor was responsible for the differences seen between trials.[145] Fluid retention seems positively related to sodium content of the beverage[144] and it may be easier to achieve high intakes of sodium by consuming some form of salty solid food after exercise.

Although in the majority of cases dehydration may be a problem, there is also a risk of drinking too much. Fluid ingested above what is required may result in GI discomfort or, more seriously, hyponatraemia.[151-153] These problems will be discussed in section 2.4.

2.4 Preventing Nutrition-Related Medical Problems

Probably the most common medical problem in triathlon is hyperthermia and heat-related illness. This can usually be combated by adjusting the drinking pattern and strategies to do this have already been discussed in sections 2.1.4 and 2.2.2. There are, however, quite a few other complications that may be nutrition related such as GI problems, endotoxaemia and hyponatraemia. These will be discussed in sections 2.4.1–2.4.3 of this review.

2.4.1 Gastrointestinal Problems

There is a very high prevalence of GI complaints during exercise among long-distance runners, triathletes and athletes involved in other types of strenuous long-lasting exercise.[154-156] The symptoms include dizziness, nausea, stomach or intestinal cramps, vomiting and diarrhoea. Prevalences of 30–50% have been reported among marathon runners.[157-159] In an attempt to evaluate the prevalence and the nature of the GI symptoms during triathlon, a study was carried out by the French Medical Society.[160] The study included 25 640 competitors participating in 101 triathlon events. It was found that 8.9% of the triathletes reported gastric symptoms such as nausea, epigastric pain or vomiting, and 8% of the competitors reported intestinal problems such as diarrhoea or abdominal pain.[160]

Rehrer et al.[100] reported a link between nutritional practices and GI complaints during a half Ironman-distance triathlon. It was found that GI problems were more likely to occur with the ingestion of fibre, fat, protein and concentrated CHO solutions during the triathlon. In particular, beverages with very high osmolalities seemed to be responsible for some of the reported complaints.

Jeukendrup et al.[161] found an even higher prevalence of symptoms during a long-distance triathlon. Thirty triathletes were asked to report their complaints during swimming, cycling and running and 92% of them reported at least one complaint during one of the disciplines. Severe symptoms included vomiting and diarrhoea and occurred mainly during running. It has been suggested that the problems occur especially during running because of the movements of the gut. However, because running is the final event of the triathlon it could also be because of the duration of the exercise. Peters et al.[162] investigated this question by adding a cycling leg after the run and still observed more GI symptoms during running compared with cycling.

Symptoms are often mild and may not even affect performance. Some of the symptoms, however, can be very serious and will not only affect performance but are also health threatening. For example, marathon runners and long-distance triathletes occasionally have blood loss in faeces in the hours following a marathon. Schaub et al.[163] observed epithelial surface changes known to occur during ischaemia upon colonoscopic inspection of one such triathlete following a marathon and suggested that ischaemia of the lower GI tract induced the problems. Øktedalen et al.[164] reported increased intestinal permeability after a marathon, indicating damage to the gut and impaired gut function. Despite the high prevalence of symptoms, mild or severe, the aetiology of these GI complaints in endurance athletes is still incompletely understood.

2.4.2 Endotoxaemia

It has been suggested that endotoxaemia may be responsible for some of the reported GI problems. Prolonged exercise at high intensities leads to a quantitative redistribution of blood flow. The blood flow to the exercising muscle is increased (exercise hyperaemia) in proportion to the energy demand in order to increase the supply of oxygen and substrates. In addition, during intense exercise the blood flow to the skin is increased to facilitate heat dissipation. As a consequence, the blood flow to central tissues (gut and liver) is reduced during exercise by almost 80%.[165-167] The blood flow to the gut may even be further reduced during exercise in the heat when plasma volume may be further reduced.[168] A similar redistribution of blood flow is seen in patients with major trauma and/or sepsis and various forms of shock. In this situation a serious underperfusion of the gut often leads to shock-induced mucosal damage and invasion of Gram-negative intestinal bacteria and/or their toxic constituents (endotoxins) into the blood circulation.[169] Increased circulating lipopolysaccharide (LPS) levels in patients lead to various symptoms such as fever, shivering, dizziness, nausea, various GI complaints such as vomiting and diarrhoea, and ultimately sepsis,[170] symptoms similar to those often reported by ultra-endurance athletes.

Endotoxaemia after strenuous ultra-endurance exercise has been reported. Brock-Utne et al.[171] reported that 81% of 89 ultra-marathon runners in the Comrades marathon (90km) demonstrated elevated plasma endotoxin concentrations. Two percent showed endotoxin (LPS) concentrations >1000 pg/mL, a value reported in patients with meningococcal sepsis and considered to be extremely high if one considers a value of 5 pg/mL the limit for endotoxaemia to predict or exclude oncoming sepsis.[172] However, in that study, resting levels of LPS also were in the range usually observed in critically ill septic patients. In another study, LPS concentrations increased and the anti-LPS IgG levels markedly decreased after a triathlon (3.2km swim, 140km cycle, 42.2km run).[173] Again, the reported resting levels of LPS were in the range usually observed in critically ill septic patients, which raises doubt about the validity of those results. Other studies utilising subjects with post-exertional illness after a 161km (100-mile) cycle ride in the heat[174] and after a marathon[175] showed only minor or no systemic endotoxaemia.

Thirty of the triathletes competing in the 1996 Embrun long-distance triathlon volunteered to take part in a study investigating the potential relationship between endotoxaemia and GI problems. One-third of the triathletes reported stomach problems, 21% reported nausea, 7% experienced dizziness, six triathletes (21%) vomited and two had diarrhoea.[161] Two triathletes had to abandon the race because of severe GI distress (vomiting and diarrhoea). Although there was a high incidence of GI complaints, including several severe symptoms, only mild endotoxaemia was observed in the athletes investigated (i.e. LPS just above the 5 pg/mL threshold used to define endotoxaemia). The degree of endotoxaemia was not related to the incidence or the severity of the complaints. The occurrence of endotoxaemia in this study was not related to the fluid or food intake in these athletes.[161] Although there were considerable differences in fluid intake (ranging from 400 mL/hour to 1.2 L/hour) and weight loss varied from 0–6kg this also did not seem to be related to the occurrence of endotoxaemia.[161]

In summary, it seems unlikely that endotoxaemia is responsible for the observed GI problems or the sometimes occurring post-exercise fever, shivering, dizziness and nausea, and there is currently no evidence to suggest a link between nutritional practices and endotoxaemia.

2.4.3 Hyponatraemia

An electrolyte imbalance, commonly referred to as ‘water intoxication’ that results from hyponatraemia (low plasma sodium) due to excessive water consumption has occasionally been reported in long-distance triathletes.[153,176-178] This appears to be most common among slow competitors in triathlons and ultra-marathon races and probably arises due to loss of sodium in sweat coupled with very high intakes (8–10L) of water or other low-sodium drinks.[179] The symptoms of hyponatraemia are similar to those associated with dehydration and include mental confusion, weakness and fainting. Such symptoms are usually seen at serum sodium concentrations of 126–130 mmol/L. Below 126 mmol/L, seizures, coma and death may occur. Because the symptoms of hyponatraemia are so similar to those of dehydration, there can be a danger of misdiagnosis of this condition when it occurs in individuals participating in endurance races. The usual treatment for dehydration is administration of fluid both orally and intravenously. If this treatment were to be given to a hyponatraemic individual, the consequences could be fatal.

Often, however, triathletes may develop hyponatraemia without displaying the symptoms. Hyponatraemia may occur in a state of euhydration or even dehydration but is generally associated with fluid overload.[177] To prevent hyponatraemia, it is recommended to avoid overhydration and to inform athletes about the potential dangers of drinking too much water or sodium-free beverages. Vrijens et al.[180] investigated the effect of replacing sweat losses by a sodium-containing drink compared with water and found that the sodium-containing drink attenuated the fall in plasma sodium. Interestingly, Speedy et al.[181] recently investigated the effects of sodium ingestion on the development of hyponatraemia. Thirty-eight athletes competing in an Ironman-distance triathlon were given salt tablets (700 mg/hour) to ingest during the race. Data collected from these athletes were compared with data from athletes not given salt. Sodium ingestion was associated with a decrease in the extent of weight loss during the race. There was no evidence that sodium ingestion significantly influenced changes in plasma sodium concentration or plasma volume more than fluid replacement alone in this study. The authors, therefore, suggested that sodium supplementation was not necessary to prevent the development of hyponatraemia in these athletes. The triathletes in this study lost weight, indicating that they had only partially replaced their fluid during the Ironman triathlon.

3. Conclusions

Triathlon is a sport that combines three disciplines (swimming, cycling and running) and competitions last between 1 hour 50 minutes (Olympic distance) and 14 hours (Ironman distance). The causes of fatigue in short- versus long-distance triathlons are likely to be different; however, independent of the distance, dehydration and carbohydrate depletion are the most likely causes of fatigue in triathlon. Contrary to traditional beliefs, studies have demonstrated that it is possible to increase muscle glycogen concentrations to very high levels with minimal modifications in diet and training. Cycling provides the best opportunity to ingest fluids to reduce fluid losses. Based on extensive research, detailed guidelines have been formulated. Much less is known about the causes of gastrointestinal problems that frequently occur, especially in long-distance triathlon. Endotoxaemia has been suggested as an explanation for some of the gastrointestinal problems, but this has not been confirmed by recent research. Although mild endotoxaemia may occur after an Ironman-distance triathlon, this does not seem to be related to the incidence of gastrointestinal problems. Hyponatraemia has occasionally been reported and is often associated with very large fluid intakes. Future studies should look into the hazards of dehydration versus hyponatraemia and guidelines should be formulated that take into account the potential risk of both dehydration and hyponatraemia.

Acknowledgements

The research of the authors is funded by GlaxoSmithKline, Consumer Healthcare.

References

1. Coyle EF. Substrate utilization during exercise in active people. Am J Clin Nutr 1995; 61: 968-79
2. Holloszy JO, Kohrt WM. Regulation of carbohydrate and fat metabolism during and after exercise. Ann Rev Nutr 1996; 16: 121-38
3. Ivy JL. Role of carbohydrate in physical activity. Clin Sports Med 1999; 18: 469-84
4. Romijn JA, Coyle EF, Sidossis LS, et al. Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity. Am J Physiol 1993; 265: E380-91
5. Bergström J, Hermansen L, Hultman E, et al. Diet, muscle glycogen and physical performance. Acta Physiol Scand 1967; 71: 140-50
6. Hultman E. Physiological role of muscle glycogen in man, with special reference to exercise. Circ Res 1967; 10: I99-I114
7. Coyle EF, Coggan AR, Hemmert MK, et al. Muscle glycogen utilization during prolonged strenuous exercise when fed carbohydrate. J Appl Physiol 1986; 61: 165-72
8. Noakes TD. Physiological models to understand exercise fatigue and the adaptations that predict or enhance athletic performance. Scand J Med Sci Sports 2000; 10: 123-45
9. Coyle EF. Fluid and fuel intake during exercise. J Sports Sci 2004; 22: 39-55
10. Sawka MN, Pandolf KB. Effects of body water loss in physiological function and exercise performance. In: Lamb DR, Gisolfi CV, editors. Perspectives in exercise science and sports medicine: fluid homeostasis during exercise. Indianapolis (IN): Benchmark Press, 1990: 1-38
11. Bentley DJ, Millet GP, Vleck VE, et al. Specific aspects of contemporary triathlon: implications for physiological analysis and performance. Sports Med 2002; 32: 345-59
12. Moseley L, Jeukendrup AE. The reliability of cycling efficiency. Med Sci Sports Exerc 2001; 33: 621-7
13. Sawka MN. Physiological consequences of hypohydration: exercise performance and thermoregulation. Med Sci Sports Exerc 1992; 24: 657-70
14. Febbraio MA, Snow RJ, Stathis CG, et al. Effect of heat stress on muscle energy metabolism during exercise. J App Physiol 1994; 77: 2827-31
15. Fink WJ, Costill DL, Van Handel PJ. Leg muscle metabolism during exercise in the heat and cold. Eur J App Physiol 1975; 34: 183-90
16. Jentjens RL, Wagenmakers AJ, Jeukendrup AE. Heat stress increases muscle glycogen use but reduces the oxidation of ingested carbohydrates during exercise. J Appl Physiol 2002; 92: 1562-72
17. Pitsiladis YP, Maughan RJ. The effects of exercise and diet manipulation on the capacity to perform prolonged exercise in the heat and in the cold in trained humans. J Physiol 1999; 517: 919-30
18. Gonzales-Alonso J, Teller C, Andersen SL, et al. Influence of body temperature on the development of fatigue during prolonged exercise in the heat. J Appl Physiol 1999; 86: 1032-9
19. Nielsen B, Hales JRS, Strange NJ, et al. Human circulatory and thermoregulatory adaptations with heat acclimation and exercise in a hot, dry enviroment. J Physiol 1993; 460: 467-85
20. Cheuvront SN, Carter III R, Sawka MN. Fluid balance and endurance exercise performance. Curr Sports Med Rep 2003; 2: 202-8
21. Walsh RM, Noakes TD, Hawley JA, et al. Impaired high-intensity cycling performance time at low levels of dehydration. Int J Sports Med 1994; 15: 392-8
22. Armstrong LE, Costill DL, Fink WJ. Influence of diruetic-induced dehydration on competitive running performance. Med Sci Sports Exerc 1985; 17: 456-61
23. Rehrer NJ, Beckers EJ, Brouns F, et al. Effects of dehydration on gastric emptying and gastrointestinal distress while running. Med Sci Sports Exerc 1990; 22: 790-5
24. Rogers G, Goodman C, Rosen C. Water budget during ultra-endurance exercise. Med Sci Sports Exerc 1997; 29: 1477-81

25. Hawley JA, Schabort EJ, Noakes TD, et al. Carbohydrate loading and exercise performance: an update. Sports Med 1997; 24 (2): 73-81
26. Sherman WM, Costill DL, Fink WJ, et al. Effect of exercise-diet manipulation on muscle glycogen and its subsequent utilisation during performance. Int J Sports Med 1981; 2: 114-8
27. Coyle EF, Jeukendrup AE, Oseto MC, et al. Low-fat diet alters intramuscular substrates and reduces lipolysis and fat oxidation during exercise. Am J Physiol Endocrinol Metab 2001; 280: E391-8
28. Fairchild TJ, Fletcher S, Steele P, et al. Rapid carbohydrate loading after a short bout of near maximal-intensity exercise. Med Sci Sports Exerc 2002; 34: 980-6
29. Bussau VA, Fairchild TJ, Rao A, et al. Carbohydrate loading in human muscle: an improved 1 day protocol. Eur J Appl Physiol 2002; 87: 290-5
30. Goforth HW, Arnall DA, Bennett BL, et al. Persistence of supercompensated muscle glycogen in trained subjects after carbohydrate loading. J Appl Physiol 1997; 82: 342-7
31. Coyle EF, Coggan AR, Hemmert MK, et al. Substrate usage during prolonged exercise following a preexercise meal. J Appl Physiol 1985; 59: 429-33
32. Burke LM, Hawley JM. Carbohydrate and exercise. Curr Opin Clin Nutr Metab Care 1999; 2: 515-20
33. Hargreaves M. Metabolic responses to carbohydrate ingestion: effects on exercise performance. In: Lamb DR, Murray R, editors. Perspectives in exercise science and sports medicine: the metabolic basis of performance in exercise and sport. Carmel: Cooper Publishing Group LLC, 1999, 93-124
34. Hargreaves M, Hawley JA, Jeukendrup AE. Pre-exercsie carbohydrate and fat ingestion: effects on metabolism and performance. J Sports Sci 2004; 22: 31-8
35. Sherman WM, Brodowicz G, Wright DA, et al. Dernbach. Effects of 4h preexercise carbohydrate feedings on cycling performance. Med Sci Sports Exerc 1989; 21: 598-604
36. Wright DA, Sherman WM, Dernbach AR. Carbohydrate feedings before, during, or in combination improve cycling endurance performance. J Appl Physiol 1991; 71: 1082-8
37. Foster C, Costill DL, Fink WJ. Effects of preexercise feedings on endurance performance. Med Sci Sports 1979; 11: 1-5
38. Keller K, Schwarzkopf R. Preexercise snacks may decrease exercise performance. Phys Sportsmed 1984; 12: 89-91
39. Costill DL, Coyle E, Dalsky G, et al. Effects of elevated plasma FFA and insulin on muscle glycogen usage during exercise. J App Physiol 1977; 43: 695-9
40. Koivisto VA, Karonen SL, Nikkila EA. Carbohydrate ingestion before exercise: comparison of glucose, fructose, and sweet placebo. J Appl Physiol 1981; 51: 783-7
41. Marmy-Conus N, Fabris S, Proietto J, et al. Preexercise glucose ingestion and glucose kinetics during exercise. J Appl Physiol 1996; 81: 853-7
42. Chryssanthopoulos C, Hennessy LC, Williams C. The influence of pre-exercise glucose ingestion on endurance running capacity. Br J Sports Med 1994; 28: 105-9
43. Febbraio M, Stewart K. CHO feedings before prolonged exercise: effect of glycemic index on muscle glycogenolysis and exercise performance. J Appl Physiol 1996; 81: 1115-20
44. Febbraio MA, Keenan J, Angus DJ, et al. Preexercise carbohydrate ingestion, glucose kinetics, and muscle glycogen use: effect of the glycemic index. J Appl Physiol 2000; 89: 1845-51
45. Hargreaves M, Costill DL, Fink WJ, et al. Effect of pre-exercise carbohydrate feedings on endurance cycling performance. Med Sci Sports Exerc 1987; 19: 33-6
46. Sparks MJ, Selig SS, Febbraio MA. Pre-exercise carbohydrate ingestion: effect of the glycemic index on endurance exercise performance. Med Sci Sports Exerc 1998; 30: 844-9
47. Gleeson M, Maughan RJ, Greenhaff PL. Comparison of the effects of pre-exercise feeding of glucose, glycerol and placebo on endurance and fuel homeostasis in man. Eur J Appl Physiol 1986; 55: 645-53
48. Kirwan JP, O'Gorman D, Evans WJ. A moderate glycemic meal before endurance exercise can enhance performance. J Appl Physiol 1998; 84: 53-9
49. Sherman WM, Peden MC, Wright DA. Carbohydrate feedings 1h before exercise improves cycling performance. Am J Clin Nutr 1991; 54: 866-70
50. Speedy D, Kelly M, O'Brien M. The effect of pre-exercise feeding on endurance exercise performance. NZ J Sports Med 1998; 26: 34-7
51. Thomas DE, Brotherhood JR, Brand JC. Carbohydrate feeding before exercise: effect of glycemic index. Int J Sports Med 1991; 12: 180-6
52. Jentjens RL, Cale C, Gutch C, et al. Effects of pre-exercise ingestion of differing amounts of carbohydrate on subsequent metabolism and cycling performance. Eur J Appl Physiol 2003; 88: 444-52
53. Jentjens RL, Jeukendrup AE. Effects of pre-exercise ingestion of trehalose, galactose and glucose on subsequent metabolism and cycling performance. Eur J Appl Physiol 2003; 88: 459-65
54. Mitchell JB, Braun WA, Pizza FX, et al. Pre-exericse carbohydrate and fluid ingestion: influence of glycemic response on 10-km treadmill running performance in the heat. J Sports Med Phys Fitness 2000; 40: 41-50
55. Moseley L, Lancaster GI, Jeukendrup AE. Effects of timing of pre-exercise ingestion of carbohydrate on subsequent metabolism and cycling performance. Eur J Appl Physiol 2003; 88: 453-8
56. van Zant RS, Lemon PWR. Preexercise sugar feeding does not alter prolonged exercise muscle glycogen or protein catabolism. Can J Appl Physiol 1997; 22: 268-79
57. Jentjens RL, Jeukendrup AE. Prevalence of hypoglycemia following pre-exercise carbohydrate ingestion is not accompanied by higher insulin sensitivity. Int J Sport Nutr Exerc Metab 2002; 12: 398-413
58. Kuipers H, Fransen EJ, Keizer HA. Pre-exercise ingestion of carbohydrate and transient hypoglycemia during exercise. Int J Sports Med 1999; 20: 227-31
59. Wee SL, Williams C, Gray S, et al. Influence of high and low glycemic index meals on endurance running capacity. Med Sci Sports Exerc 1999; 31: 393-9
60. Convertino VA, Armstrong LE, Coyle EF, et al. American College of Sports Medicine position stand: exercise and fluid replacement. Med Sci Sports Exerc 1996; 28: i-vii
61. Latzka WA, Sawka MN. Hyperhydration and glycerol: thermoregulatory effects during exercise in hot climates. Can J Appl Physiol 2000; 25: 536-45
62. Robergs RA, Griffin SE. Glycerol: biochemistry, pharmacokinetics and clinical and practical applications. Sports Med 1998; 26 (3): 145-67
63. Coutts A, Reaburn P, Mummery K, et al. The effect of glycerol hyperhydration on olympic distance triathlon performance in high ambient temperatures. Int J Sport Nutr Exerc Metab 2002; 12: 105-19
64. Marino FE, Kay D, Cannon J. Glycerol hyperhydration fails to improve endurance performance and thermoregulation in

humans in a warm humid environment. Pflugers Arch 2003; 446: 455-62
65. Rehrer NJ. Fluid and electrolyte balance in ultra-endurance sport. Sports Med 2001; 31 (10): 701-15
66. Coggan AR, Coyle EF. Reversal of fatigue during prolonged exercise by carbohydrate infusion or ingestion. J Appl Physiol 1987; 63: 2388-95
67. Anantaraman R, Carmines AA, Gaesser GA, et al. Effects of carbohydrate supplementation on performance during 1h of high intensity exercise. Int J Sports Med 1995; 16: 461-5
68. Below PR, Mora-Rodríguez R, Gonzáles Alonso J, et al. Fluid and carbohydrate ingestion independently improve performance during 1h of intense exercise. Med Sci Sports Exerc 1995; 27: 200-10
69. Jeukendrup A, Brouns F, Wagenmakers AJ, et al. Carbohydrate-electrolyte feedings improve 1h time trial cycling performance. Int J Sports Med 1997; 18: 125-9
70. Kimber NE, Ross JJ, Mason SL, et al. Energy balance during an ironman triathlon in male and female triathletes. Int J Sport Nutr Exerc Metab 2002; 12: 47-62
71. Jeukendrup AE, Raben A, Gijsen A, et al. Glucose kinetics during prolonged exercise in highly trained human subjects: effect of glucose ingestion. J Physiol 1999; 515: 579-589, 1999
72. Jeukendrup AE, Wagenmakers AJ, Stegen JH, et al. Carbohydrate ingestion can completely suppress endogenous glucose production during exercise. Am J Physiol 1999; 276: E672-83
73. Tsintzas OK, Williams C, Boobis L, et al. Carbohydrate ingestion and glycogen utilisation in different muscle fibre types in man. J Physiol 1995; 489: 243-50
74. Tsintzas K, Williams C. Human muscle glycogen metabolism during exercise: effect of carbohydrate supplementation. Sports Med 1998; 25 (1): 7-23
75. Shi X, Summers RW, Schedl HP, et al. Effects of carbohydrate type and concentration and solution osmolality on water absorption. Med Sci Sports Exerc 1995; 27: 1607-15
76. Adopo E, Peronnet F, Massicotte D, et al. Respective oxidation of exogenous glucose and fructose given in the same drink during exercise. J Appl Physiol 1994; 76: 1014-9
77. Jentjens RL, Moseley L, Waring RH, et al. Oxidation of combined ingestion of glucose and fructose during exercise. J Appl Physiol 2004; 96: 1277-84
78. Jentjens RL, Venables MC, Jeukendrup AE. Oxidation of exogenous glucose, sucrose, and maltose during prolonged cycling exercise. J Appl Physiol 2004; 96: 1285-91
79. Jeukendrup AE, Jentjens R. Oxidation of carbohydrate feedings during prolonged exercise: current thoughts, guidelines and directions for future research. Sports Med 2000; 29 (6): 407-24
80. Guezennec CY, Vallier JM, Bigard AX, et al. Increase in energy cost of running at the end of a triathlon. Eur J App Physiol 1996; 73: 440-5
81. Backx KK, van Someren A, Palmer GS. One hour cycling performance is not affected by ingested fluid volume. Int J Sport Nutr Exerc Metab 2003; 13: 333-42
82. Robinson TA, Hawley JA, Palmer GS, et al. Water ingestion does not improve 1h cycling performance in moderate ambient temperatures. Eur J Appl Physiol 1995; 71: 153-60
83. Costill DL, Saltin B. Factors limiting gastric emptying during rest and exercise. J App Physiol 1974; 37: 679-83
84. Leiper JB, Broad NP, Maughan RJ. Effect of intermittent high-intensity exercise on gastric emptying in man. Med Sci Sports Exerc 2001; 33: 1270-8
85. Rehrer NJ, Brouns F, Beckers EJ, et al. The influence of beverage composition and gastrointestinal function on fluid and nutrient availability during exercise: a review. Scand J Med Sci Sports 1994; 4: 159-72
86. Dennis SC, Noakes TD, Hawley JA. Nutritional strategies to minimize fatigue during prolonged exercise: fluid, electrolyte and energy replacement. J Sport Sci 1997; 15: 305-13
87. Hunt JN, Spurrell WR. The pattern of emptying of the human stomach. J Physiol 1958; 113: 157-68
88. Hunt JN, Smith JL, Jiang CL. Effect of meal volume and energy density on the gastric emptying of carbohydrates. Gastroenterology 1985; 89: 1326-30
89. Mitchell JB, Voss KW. The influence of volume on gastric emptying and fluid balance during prolonged exercise. Med Sci Sports Exerc 1991; 23: 314-9
90. Noakes TD, Rehrer NJ, Maughan RJ. The importance of volume in regulating gastric emptying. Med Sci Sports Exerc 1991; 23: 307-13
91. Rehrer NJ, Brouns F, Beckers EJ, et al. Gastric emptying with repeated drinking during running and bicycling. Int J Sports Med 1990; 11: 238-43
92. Parsons DS, Wingate DL. The effect of osmotic gradients on fluid transfer across rat intestines in vitro. Biochem Biophys Acta 1961; 46: 107-83
93. Olsen WA, Inglefinger FJ. The role of sodium in intestinal glucose absorption in man. J Clin Invest 1968; 47: 1133-42
94. Loo DD, Zeuthen T, Chandy G, et al. Cotransport of water by the Na+/glucose cotransporter. Proc Natl Acad Sci U S A 1996; 93: 13367-70
95. Gisolfi C, Summers R, Schedl H, et al. Intestinal water absorption from select carbohydrate solutions in humans. Med Sci Sports Exerc 1992; 24: S939
96. Gisolfi CV, Spranger KJ, Summers RW, et al. Effects of cycle exercise on intestinal absorption in humans. J App Physiol 1991; 71: 2518-27
97. Gisolfi CV, Summers RD, Schedl HP, et al. Effect of sodium concentration in a carbohydrate-electrolyte solution on intestinal absorption. Med Sci Sports Exerc 1995; 27: 1414-20
98. Hargreaves M, Costill DL, Burke L, et al. Influence of sodium on glucose bioavalability during exercise. Med Sci Sports Exerc 1994; 26: 365-8
99. Maughan RJ. The sports drink as a functional food: formulations for successful performance. Proc Nutr Soc 1998; 57: 15-23
100. Rehrer NJ, van Kemenade M, Meester W, et al. Gastrointestinal complaints in relation to dietary intake in triathletes. Int J Sport Nutr 1992; 2: 48-59
101. Ryan AJ, Lambert GP, Shi X, et al. Effect of hypohydration on gastric emptying and intestinal absorption during exercise. J Appl Physiol 1998; 84: 1581-8
102. Brouns F, Senden J, Beckers EJ, et al. Osmolarity does not affect the gastric emptying rate of oral rehydration solutions. JPEN J Parenter Enteral Nutr 1995; 19: 403-6
103. Murray R, Eddy DE, Bartoli WP, et al. Gastric emptying of water and isocaloric carbohydrate solutions consumed at rest. Med Sci Sports Exerc 1994; 26: 725-32
104. Passe DH, Horn M, Murray R. Impact of beverage acceptability on fluid intake during exercise. Appetite 2000; 35: 219-29
105. Applegate E. Nutritional concerns of the ultraendurance triathlete. Med Sci Sports Exerc 1989; 21: S205-8
106. Coyle EF, Montain SJ. Carbohydrate and fluid ingestion during exercise: are there trade-offs? Med Sci Sports Exerc 1992; 24: 671-8
107. Laursen PB, Rhodes EC. Factors affecting performance in an ultraendurance triathlon. Sports Med 2001; 31 (3): 195-209

108. Millard-Stafford M, Sparling PB, Rosskopf LB, et al. Carbohydrate-electrolyte replacement during a simulated triathlon in the heat. Med Sci Sports Exerc 1990; 22: 621-8
109. Keizer H, Kuipers H, van Kranenburg G. Influence of liquid and solid meals on muscle glycogen resynthesis, plasma fuel hormone response, and maximal physical working capacity. Int J Sports Med 1987; 8: 99-104
110. Kochan RG, Lamb DR, Lutz SA, et al. Glycogen synthase activation in human skeletal muscle: effects of diet and exercise. Am J Physiol 1979; 5: E660-6
111. Starling RD, Trappe TA, Parcell AC, et al. Effects of diet on muscle triglyceride and endurance performance. J Appl Physiol 1997; 82: 1185-9
112. Nicholas CW, Green PA, Hawkins RD, et al. Carbohydrate intake and recovery of intermittent running capacity. Int J Sport Nutr 1997; 7: 251-60
113. Fallowfield JL, Williams C. Carbohydrate intake and recovery from prolonged exercise. Int J Sports Nutr 1993; 3: 150-64
114. Asp S, Rohde T, Richter EA. Impaired muscle glycogen resynthesis after a marathon is not caused by decreased muscle GLUT4 content. J Appl Physiol 1997; 83: 1482-5
115. Costill DL, Pascoe DD, Fink WJ, et al. Impaired muscle glycogen resynthesis after eccentric exercise. J Appl Physiol 1990; 69: 46-50
116. Widrick JJ, Costill DL, Fink WJ, et al. Carbohydrate feedings and exercise performance: effect of initial glycogen concentration. J Appl Physiol 1993; 74: 2998-3005
117. Burke L, Collier GR, Beasley SB, et al. Effect of coingestion of fat and protein with carbohydrate feedings on muscle glycogen storage. J Appl Physiol 1995; 78: 2187-92
118. Burke L, Collier GR, Davis PG, et al. Muscle glycogen storage after prolonged exercise: effect of the frequency of carbohydrate feedings. Am J Clin Nutr 1996; 64: 115-9
119. Burke LM, Collier GR, Hargreaves M. Muscle glycogen storage after prolonged exercise: effect of glycemic index of carbohydrate feedings. J Appl Physiol 1993; 75: 1019-23
120. Jentjens R, Jeukendrup A. Determinants of post-exercise glycogen synthesis during short-term recovery. Sports Med 2003; 33 (2): 117-44
121. Jentjens RL, van Loon LJ, Mann CH, et al. Addition of protein and amino acids to carbohydrates does not enhance postexercise muscle glycogen synthesis. J Appl Physiol 2001; 91: 839-46
122. van Hall G, Shirreffs SM, Calbet JA. Muscle glycogen resynthesis during recovery from cycle exercise: no effect of additional protein ingestion. J Appl Physiol 2000; 88: 1631-6
123. van Loon LJ, Saris WH, Kruijshoop M, et al. Maximizing postexercise muscle glycogen synthesis: carbohydrate supplementation and the application of amino acid or protein hydrolysate mixtures. Am J Clin Nutr 2000; 72: 106-11
124. Ivy JL. Muscle glycogen synthesis before and after exercise. Sports Med 1991; 11 (1): 6-19
125. Ivy JL, Lee MC, Brozinick JT, et al. Muscle glycogen storage after different amounts of carbohydrate ingestion. J Appl Physiol 1988; 65: 2018-23
126. Blom PCS, Høstmark AT, Vaage O, et al. Effect of different post-exercise sugar diets on the rate of muscle glycogen resynthesis. Med Sci Sports Exerc 1987; 19: 491-6
127. Fujisawa T, Mulligan K, Wada L, et al. The effect of exercise on fructose absorption. Am J Clin Nutr 1993; 58: 75-9
128. Henry AW, Crapo PA, Thorburn AW. Current issues in fructose metabolism. Ann Rev Nutr 1991; 11: 21-39
129. Chen M, Whistler RL. Metabolism of D-fructose. Adv Carbohydr Chem Biochem 1977; 34: 265-343
130. Mayes PA. Intermediary metabolism of fructose. Am J Clin Nutr 1993; 58: 754S-65S
131. Reed JM, Brozinick JT, Lee MC, et al. Muscle glycogen storage postexercise: effect of mode of carbohydrate administration. J Appl Physiol 1989l; 66: 720-6
132. Zawadzki KM, Yaspelkis III BB, Ivy JL. Carbohydrate-protein complex increases the rate of muscle glycogen storage after exercise. J Appl Physiol 1992; 72: 1854-9
133. Ivy J. Glycogen resynthesis after exercise: effect of carbohydrate intake. Int J Sports Med 1998; 19: S142-5
134. Rasmussen BB, Tipton KD, Miller SL, et al. An oral essential amino acid-carbohydrate supplement enhances muscle protein anabolism after resistance exercise. J Appl Physiol 2000; 88: 386-92
135. Tipton KD, Ferrando AA, Phillips SM, et al. Postexercise net protein synthesis in human muscle from orally administered amino acids. Am J Physiol 1999; 276: E628-34
136. Tipton KD, Wolfe RR. Protein and amino acids for athletes. J Sports Sci 2004; 22: 65-79
137. Maughan R, Leiper J, Shirreffs S. Factors influencing the restoration of fluid and electrolyte balance after exercise in the heat. Br J Sports Med 1997; 31: 175-82
138. Shirreffs SM, Armstrong AA, Cheuvront SN. Fluid and electrolyte needs for preparation and recovery from training and compitition. J Sports Sci 2004; 22: 57-63
139. Shirreffs SM, Taylor AJ, Leiper JB, et al. Post-exercise rehydration in man: effects of volume consumed and drink sodium content. Med Sci Sports Exerc 1996; 28: 1260-71
140. Costill DL, Sparks KE. Rapid fluid replacement following thermal dehydration. J App Physiol 1973; 34: 299-303
141. Gonzalez-Alonso J, Heaps CL, Coyle EF. Rehydration after exercise with common beverages and water. Int J Sports Med 1992; 13: 399-406
142. Nose H, Mack GW, Shi X, et al. Role of osmolality and plasma volume during rehydration in humans. J Appl Physiol 1988; 65: 325-31
143. Nose H, Mack GW, Shi X, et al. Shift in body fluid compartments after dehydration in humans. J App Physiol 1988; 65: 318-24
144. Maughan RJ, Leiper JB. Sodium intake and post exercise rehydration in man. Eur J App Physiol 1995; 71: 311-9
145. Shirreffs SM. Rehydration and recovery after exercise. In: Maughan RJ, editor. IOC encyclopaedia of sports medicine: nutrition in sport. Oxford: Blackwell Science, 2000: 73-84
146. Maughan RJ, Leiper JB. Post-exercise rehydration in man: effects of voluntary intake of four different beverages. Med Sci Sports Exerc 1993; 25 Suppl.: S2
147. Maughan RJ, Owen JH, Shirreffs SM, et al. Post-exercise rehydration in man: effects of electrolyte addition to ingested fluids. Eur J Appl Physiol 1994; 69: 209-15
148. Costill DL. Sweating: its composition and effects on body fluids. Ann NY Acad Sci 1984; 301: 106-74
149. O'Toole M, Douglas PS. Applied physiology of a triathlon. Sports Med 1995; 19 (4): 251-67
150. Maughan RJ, Leiper JB, Shirreffs SM. Restoration of fluid balance after exercise-indued dehydration: effects of food and fluid intake. Eur J App Physiol 1996; 73: 317-25
151. Armstrong LE, Curtis WC, Hubbard RW, et al. Symptomatic hyponatremia during prolonged exercise in heat. Med Sci Sports Exerc 1993; 25: 543-9

152. Irving RA, Noakes TD, Buck R, et al. Evaluation of renal function and fluid homeostasis during recovery from exercise-induced hyponatremia. J App Physiol 1991; 70: 342-8

153. Speedy DB, Rogers IR, Noakes TD, et al. Diagnosis and prevention of hyponatremia at an ultradistance triathlon. Clin J Sports Med 2000; 10: 52-8

154. Brouns F, Saris WHM, Rehrer NJ. Abdominal complaints and gastrointestinal function during long-lasting exercise. Int J Sports Med 1987; 8: 175-89

155. Rehrer NJ, Brouns F, Beckers EJ, et al. Physiological changes and gastro-intestinal symptoms as a result of ultra-endurance running. Eur J Appl Physiol 1992; 64: 1-8

156. Rehrer NJ, Janssen GME, Brouns F, et al. Fluid intake and gastrointestinal problems in runners competing in a 25-km race and a marathon. Int J Sports Med 1989; 10: S22-5

157. Keeffe EB, Lowe DK, Goss JR, et al. Gastrointestinal symptoms of marathon runners. West J Med 1984; 141: 481-4

158. Riddoch C, Trinick T. Gastrointestinal disturbances in marathon runners. Br J Sports Med 1988; 22: 71-4

159. Sullivan SN. The gastrointestinal symptoms of running [letter]. N Eng J Med 1981; 304: 915

160. Lopez AA, Preziosi JP, Chateau P, et al. Digestive disorders and self medication observed during a competition in endurance athletes: prospective epidemiological study during a championship of triathlon. Gastroenterol Clin Biol 1994; 18: 317-22

161. Jeukendrup AE, Vet-Joop K, Sturk A, et al. Relationship between gastro-intestinal complaints and endotoxaemia, cytokine release and the acute-phase reaction during and after a long-distance triathlon in highly trained men. Clin Sci 2000; 98: 47-55

162. Peters HP, van Schelven WF, Verstappen PA, et al. Exercise performance as a function of semi-solid and liquid carbohydrate feedings during prolonged exercise. Int J Sports Med 1995; 16: 105-13

163. Schaub N, Spichtin HP, Stalder GA. Ischemic colitis as a cause of intestinal bleeding after marathon running [in German]. Schweiz Med Wochenschr 1985; 115: 454-7

164. Øktedalen O, Lunde OC, Opstad PK, et al. Changes in gastrointestinal mucose after long-distance running. Scand J Gastroenterol 1992; 27: 270-4

165. Bradley SE. Variations in hepatic blood flow in man during health and disease. N Engl J Med 1949; 240: 456-61

166. Clausen JP. Effect of physical training on cardiovascular adjustments to exercise in man. Physiol Rev 1977; 57: 779-815

167. Rowell LB, Blackmon JR, Bruce RA. Indocyanine green clearance and estimated hepatic blood flow during mild to maximal exercise in upright man. J Clin Invest 1964; 43: 1677-90

168. Rowell LB, O'Leary DS, Kellogg DL. Integration of cardiovascular control systems in dynamic exercise. In: Rowell LB, Shephard JT, editors. Handbook of physiology: regulation and integration of multiple systems. New York: Oxford Press, 1996: 770-838

169. van Deventer SJH, Gouma D. Bacterial translocation and endotoxin transmigration in intestinal ischaemia and reperfusion. Curr Opinion Aneasth 1994; 7: 126-30

170. van Leeuwen PA, Boermeester MA, Houdijk AP, et al. Clinical significance of translocation. Gut 1994; 35: S28-34

171. Brock-Utne JG, Gaffin SL, Wells MT, et al. Endotoxaemia in exhausted runners after a long distance race. S Afr Med J 1988; 73: 533-6

172. van Deventer SJH, Buller HR, ten Cate JW, et al. Endotoxaemia: an early predictor of septicaemia in febrile patients. Lancet 1988; I (8586): 605-8

173. Bosenberg AT, Brock-Utne JG, Gaffin SL, et al. Strenuous exercise causes systemic endotoxemia. J Appl Physiol 1988; 65: 106-8

174. Moore GE, Blair Holbein ME, Knochel JP. Exercise-associated collapse in cyclists is unrelated to endotoxemia. Med Sci Sports Med 1995; 27: 1238-42

175. Camus G, Poortmans J, Nys M, et al. Mild endotoxaemia and the inflammatory response induced by exercise. Clin Sci 1997; 92: 415-22

176. Hiller WDB, O'Toole ML, Fortess EE, et al. Medical and physiological considerations in triathlon. Am J Sports Med 1987; 15: 164-7

177. Speedy DB, Noakes TD, Rogers IR, et al. Hyponatremia in ultradistance triathletes. Med Sci Sports Exerc 1999; 31: 809-15

178. Speedy DB, Rogers IR, Noakes TD, et al. Exercise-induced hyponaremia in ultradistance triathletes is caused by inappropriate fluid retention. Clin J Sports Med 2000; 10: 272-8

179. Noakes TD, Goodwin N, Rayner BL, et al. Water intoxication: a possible complication during endurance exercise. Med Sci Sports Exerc 1985; 17: 370-5

180. Vrijens DM, Rehrer NJ. Sodium-free fluid ingestion decreases plasma sodium during exercise in the heat. J Appl Physiol 1999; 86: 1847-51

181. Speedy DB, Thompson JM, Rodgers I, et al. Oral salt supplementation during ultradistance exercise. Clin J Sport Med 2002; 12: 279-84

Correspondence and offprints: Dr *Asker E. Jeukendrup*, Human Performance Laboratory, School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, B15 2TT, UK.
E-mail: A.E.Jeukendrup@bham.ac.uk