Original Research

Food Intake and Electrolyte Status of Ultramarathoners Competing in Extreme Heat

Beth W. Glace, MS, Christine A. Murphy, PAC, and Malachy P. McHugh, PhD

Nicholas Institute of Sports Medicine and Athletic Trauma (B.W.G., M.P.M.), Lenox Hill Hospital (C.A.M.), New York, New York

Key words: ultramarathon, gastrointestinal distress, hyponatremia, fluid, endurance

Objective: To relate changes in laboratory indices to dietary intake during extremely prolonged running and to determine if dietary intake influences the ability of runners to finish an 160 km trail race.

Methods: We monitored intake and serum chemistries of 26 runners competing in an 160 km foot race in temperatures which peaked at 38°C. Blood was drawn pre-, mid- and post-race. Dietary intake and incidence of gastrointestinal distress or changes in mental status were determined by interview with runners approximately every 13 km. Twenty-three runners completed at least 88 kms and, of these 23 runners, 13 finished 160 km in a mean time of 26.2 ± 3.6 hours.

Results: Finishers ingested nearly 30,000 J, 19.4 ± 8.1 L of fluid and 16.4 ± 9.5 g of sodium (Na). Sodium and fluid intake per hour was estimated to be 0.6 g/hour and 0.7 L/hour, respectively. Electrolyte intake during the first half of the race was similar between those that finished the race and those that did not. Finishers ingested fluid at a greater rate than non-finishers ($p = 0.01$) and tended to meet their caloric needs more closely than did non-finishers ($p = 0.09$). Body weight was unchanged over time (ANOVA, $p = 0.52$). Serum Na concentration tended to fall from 143 to 140 mEq/L during the race ($p = 0.06$), and was inversely correlated with weight loss ($p = 0.009$). Serum Na concentration was lower mid-race in runners experiencing changes in mental status than in runners without changes ($p = 0.04$). Fluid intake was inversely correlated with serum Na concentrations ($p = 0.04$). Most of the runners experienced nausea or vomiting; these symptoms were not related to serum sodium concentration. Hyponatremia (<135 mEq/L) was seen in one runner at 88 kms, but resolved by 160 km. Urinary sodium excretion decreased ($p = 0.002$) as serum aldosterone concentration increased pre- to post-race ($p < 0.001$). From start to finish of the race plasma volume increased by 12%.

Conclusions: Food and fluid was ingested at a greater rate than described previously. Runners consumed adequate fluid to maintain body weight although dietary sodium fell far short of the recommended 1 g/hour. The rate of fluid intake was greater in finishers than in non-finishers, and finishers tended to more nearly meet their energy needs. Maintenance of body mass despite large exercise energy expenditures in extreme heat is consistent with fluid overload during a running event lasting more than 24 hours in hot and humid conditions.

INTRODUCTION

Little work has been done which prospectively and carefully examines both food and fluid intake and serum chemistries in ultra-endurance events. The choice of food and beverage becomes a crucial factor in performance during such events. Carbohydrate intake during prolonged exercise has been shown to improve time to exhaustion by providing exogenous energy [1], while sufficient fluid intake is necessary to prevent dehydration [2]. In addition, hyponatremia is well-documented during prolonged exercise [3,4]. Hyponatremic runners seeking medical assistance present with a variety of symptoms that range from nausea, weakness, confusion, incoordination to grand mal seizures and coma [5]. We have previously found that runners exercising nearly continuously for 24 hours experienced nausea and changes in mental status [6]. Sodium intake for these runners was much less than recommended (0.5 g/hour *vs.* 1–2 g/hour) [7] while fluid and total energy intake were greater than expected. Several runners had also reported profound post-exercise diuresis. These symptoms were suggestive of hyponatremia although we had not assessed serum electrolytes.

The purpose of this study was to document changes in blood

Address reprint requests to: Beth W. Glace, M.S., Nicholas Inst. Sports Medicine and Athletic Trauma, Lenox Hill Hospital, 130 East 77th Street, New York, NY 10021. E-mail: lb@nismat.org

Presented in part at the American College of Sports Medicine, Indianapolis, IN, June 2000.

Journal of the American College of Nutrition, Vol. 21, No. 6, 553–559 (2002)
Published by the American College of Nutrition

and urine electrolytes and to relate those changes to dietary intake in runners engaged in a 160 km trail race. We further sought to determine whether intake had an effect upon the ability to complete the race and upon gastrointestinal function or mental status.

METHODS

Subjects

Twenty-six volunteers with a mean age of 48.8 ± 8.8 years (SD) were recruited by letter three months prior to the event. To be eligible, subjects had to have an anticipated finish time of less than 24 hours by self-report. According to race results from the previous two years, this gave us a pool of approximately 85 runners to recruit from and limited us to the top 50% of competitors according to finish times. The 21 men and 5 women who agreed to participate were mailed questionnaires regarding their medical and running/training history. The research protocol was approved by the Research and Clinical Investigations Committee of Lenox Hill Hospital, and written informed consent was obtained prior to subject participation. Participants were paid $100 at study completion.

Protocol

On the evening prior to the race, before the pre-race meal, participants provided a urine sample that was placed on ice. After reviewing the medical history, body mass was determined in shorts (jog bra for women), with shoes, on a calibrated scale and body fat was determined with Skindex skinfold calipers (Caldwell, Justiss & Co., Fayetville, AR) that use a gender- and age-adjusted formula [8,9]. Blood was drawn from an antecubital vein within two minutes of being seated. An aliquot of whole blood was analyzed within three hours for hemoglobin (HemoCue, Lee Diagnostics AB, Medical) and hematocrit. The remaining sample was centrifuged and separated. The serum was frozen and later analyzed for aldosterone, sodium, potassium, osmolality and glucose. Plasma volume changes were calculated according to the methods described by Strauss *et al.* using hemoglobin and hematocrit [10]. Runners were provided with dietary intake records and instructed to record their food intake for the 12 hours preceding the race, including the pre-race meal. In addition, they were asked to record the type and amount of non-steroidal anti-inflammatory drugs (NSAIDs) pre-race.

The runners were met just prior to the 4:30 a.m. race start, where the pre-race food records were collected and reviewed. Temperatures on race day ranged from 21°–38°C. The race course consisted primarily of dirt roads and trails with 4500 meters of ascents and descents. There were 37 official food stations spaced throughout the course. Investigators manned 12 food stations, at approximately 13 km intervals. A checklist of supplied food items was used to record the type and quantity of food, beverages and NSAIDs taken by the runners at each of the observed stations. Since runners could eat from food stations other than the ones monitored by investigators and could eat foods supplied by support crews, they were also asked to recall everything they had ingested since we last interviewed them. Runners were asked to keep any wrappers from foods eaten on the trail until they next saw us, to help account for their intake. At each interview the runners were asked if they had experienced any gastrointestinal symptoms (GiSx) i.e. nausea, vomiting, diarrhea, and were observed for changes in mental status (MSx) such as confusion, disorientation, or inability to concentrate.

Blood samples were collected again at 88 km and at the finish line and analyzed or frozen, as described above. Urine collected pre- and post-race was frozen and later analyzed for electrolytes and osmolality. Body mass was assessed pre-race, at 70 km, 110 km, 133 km and immediately after the finish, after voiding and prior to taking any further food or fluid. Skinfold measurements were repeated after towel drying. Energy expenditure during the event was estimated using a stepwise calculation using body mass, mean velocity and time spent running [11].

Dietary Analysis

The NutriBase Nutrition Management Software, Clinical Version, (CyberSoft, Inc. Phoenix, AZ) was used to calculate the nutritional composition of the foods and fluids consumed. Runners were subsequently interviewed by phone if clarification was required, e.g. to obtain brand names of specialized "sport" supplements. The manufacturers were contacted to provide nutritional composition of these products, and the data was added to the nutritional software database. Where possible, food items were obtained, and the food was weighed in our laboratory according to typical serving size. For example, we standardized "handfuls" of a variety of food items by having several staff members repeatedly take a handful of the specified food. The food was then weighed, and the mean weight was used as a typical "handful". Diets were analyzed for total calories, macro- and micro-nutrients as well as for total moisture content.

Statistical Analysis

Anthropometric measurements, dietary intake and blood and urine analysis were examined. A one-way, repeated measure ANOVA was used to examine changes over the duration of the race. Two-way mixed model ANOVAs were used to examine changes overtime between runners: (1) with/without MSx; (2) finishers and non-finishers. Because non-steroidal anti-inflammatory drugs (NSAIDs) are associated with GI distress, ANCOVA with NSAID intake as a confounding variable was used to compare dietary intake between GISx and No GISx groups. Statistical significance was established for an alpha level of 0.05. Bonferroni corrections were used for all *post-hoc*

pair-wise comparisons. All results are reported as means with standard deviations.

RESULTS

Anthropometrics

Thirteen of the 26 subjects completed the 160 km course, in a mean time of 26.2 ± 0.4 hours. Of the non-finishers, all subjects completed a minimum of 45 km. Eleven of the 13 non-finishers had body mass determined at 70 km. However, most of the non-finishers dropped out of the race while on the trail; final body mass determination and blood sampling could not always be performed at the time the runner dropped out, unless they happened to stop at a monitored aid station. The mean change in weight from race start till the last time weighted during the race, or until the finish, was −0.97 ± 1.8 kg. Finishers and non-finishers did not differ in the magnitude of weight change, −0.5 ± 1.5 kg *vs.* −1.2 ± 1.8 kg ($p = 0.27$), respectively. Surprisingly, there was no significant change in body mass over time in the finishers ($p = 0.52$). Finishers decreased mass by 0.5 kg, representing a 0.6% decline from pre-race values. Although finishers tended to weigh less pre-race than non-finishers (66.5 kg *vs.* 73.8 kg, $p = 0.09$), the mean percent body fat did not differ between groups (12.8 ± 6.1% *vs.* 15.5 ± 5.9%, $p = 0.22$, respectively). There was no significant change in body fat from pre- to post-race (12.8 ± 6.1% *vs.* 11.9 ± 3.7%, $p = 0.29$).

Dietary Intake

An analysis of dietary records collected for the 12 hours pre-race revealed that 8986 joules were ingested, of which carbohydrate intake accounted for 59% (Table 1). Three liters (±1.4) of fluid and 3.2 g (±1.9) of sodium were consumed pre-race. Intake is expressed absolutely and per km/kg, in order to include runners that did not complete the entire course. Runners who completed 160 km ingested sodium, protein and fluid at a greater rate than did non-finishers. Carbohydrates provided 81% of the 29493 J ingested by those completing the race. Finishers drank a mean of 19.4 ± 5.6 L, with a range of 11.9 to 28.1 L. Sodium intake was 16.4 ± 6.8 g, with a range of 4.9 to 27.5 grams. These were consumed at rates of 0.7 L/hour and 0.6 g/hour, respectively. Notably, 16% of the total fluid intake was contributed by the moisture contained in solid foods. The greatest rates of fluid and carbohydrate intake occurred during the 2nd and 3rd quarters of the race (Fig. 1). The rate of fluid intake was greater for those runners who went on to complete the entire course, as compared to those who did not finish, when adjusted for body weight (1.8 ± 0.49 *vs.* 1.3 ± 0.4 mL/kg/km, $p = 0.011$).

The rates of energy expenditure and intake were calculated for both finishers and non-finishers. Expenditure was substantially greater than energy intake, 40,061 *vs.* 21,569 J respectively, resulting in a mean deficit of −161 J per km completed. The energy deficit per km was greater for non-finishers than for non-finishers (−190.6 *vs.* −132.1 J/km, $p = 0.01$).

Blood and Urine Analysis

Twelve of the 13 finishers and nine of the 13 non-finishers had blood drawn mid-race. Glucose concentration were 90(5.0), 97(5.4), and 95 mg/dL (5.3 mmol/L) pre-, mid- and post-race, i.e. there was no evidence of hypoglycemia. Serum potassium concentration was 4.15 mEq/L, 5.85 and 4.18 mEq/L pre-race, mid-race and post-race, respectively (effect of time $p < 0.001$). Serum sodium concentrations tended to fall from 143.9 mEq/L pre-race, to 140.8 mEq/L mid-race, and to 140.2 mEq/L post-race ($p = 0.06$). Hyponatremia, as defined by a serum Na concentration of less than 135 mEq/L, was observed in only one runner, at 90 km. The runner complained of both vomiting, which began at 12 km, and confusion, beginning at 88 km. The runner's serum Na concentrations were as follows pre-, mid-, and post-race: 144 mmol/L, 133 mmol/L and 136 mmol/L, respectively. Changes in body mass inversely paralleled the changes in serum Na; his mass was 71.2 kg pre-, 73.9 kg mid-, and 73.0 kg post-race. His mean Na and fluid intakes were 17.1 g and 24.2 L, respectively. Anecdotally, the runner reported that he had not urinated prior to his episode of confusion, but that after drinking a beer during the race he began voiding and "felt much better."

Table 1. Nutrient Intake

	Joules	Fluid (L)	Carb (g)	Fat (g)	Protein (g)	Fiber (g)	Na (g)	K (g)
Pre-Race (n = 26)	8986 ± 3359	3.2 ± 1.4	318 ± 144	70 ± 39	67 ± 66	31.7 ± 8.7	3.2 ± 1.9	1.9 ± 1.0
Race All (n = 26)	21589 ± 12803	14.1 ± 6.5	1084 ± 609	95 ± 73	84 ± 62	23 ± 16	11.4 ± 7.4	5.1 ± 3.3
Race Finishers (n = 13)	29493 ± 3481	19.4 ± 5.6	1419 ± 622	137 ± 75	128 ± 56	32 ± 15	16.4 ± 6.8	7.2 ± 3.1
Rate of Intake		(mL)	(g)	(mg)	(mg)	(mg)	(mg)	(mg)
All /kg/km (n = 26)	2.15 ± 1.07	1.57 ± 0.5	0.13 ± 0.07	0.01 ± 0.007	0.009 ± 0.006	0.003 ± 0.002	1.3 ± 0.7	0.06 ± 0.03
Non-Finishers /kg/km (n = 13)	2.15 ± 0.99	1.33* ± 0.41	0.12 ± 0.07	0.008 ± 0.007	0.006* ± 0.004	0.002 ± 0.002	0.94* ± 0.58	0.06 ± 0.04
Finishers Per /kg/km (n = 13)	2.15 ± 1.18	1.8* ± 0.49	0.14 ± 0.07	0.01 ± 0.007	0.01* ± 0.006	0.003 ± 0.001	1.60* ± 0.69	0.06 ± 0.03

* Finishers and non-finishers, $p \leq 0.01$. Values are ± SD.

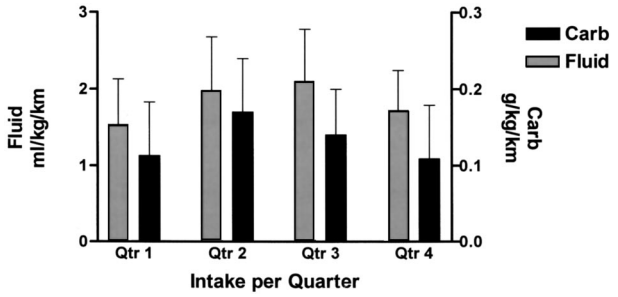


Fig. 1. Intake per quarter. Fluid: effect of time, $p = 0.04$; Carbohydrate: effect of time, $p = 0.004$. Values are ± SD.

There was no relationship between total sodium intake and post-race serum (r = 0.07, $p = 0.83$) or urinary sodium (r = 0.41, $p = 0.17$). Post-race serum sodium was affected, however, by fluid intake during the second half of the race (r = −0.58, $p = 0.04$) (Fig. 2a); high fluid intakes were correlated with lower post race serum sodium. Post-race urinary sodium also tended to be inversely correlated with 2nd half fluid intakes (r = −0.53, $p = 0.06$), (Fig. 2b). No relationship was seen between dietary potassium intake and serum or urinary potassium levels.

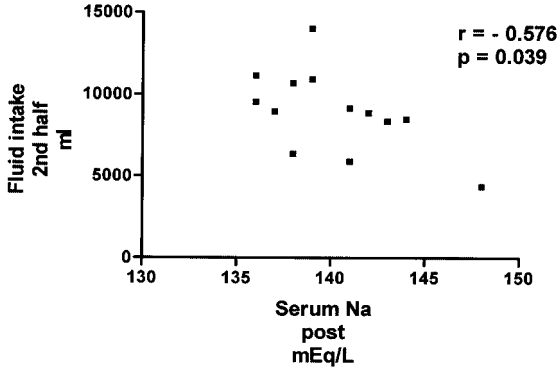
Loss of body weight was inversely correlated with changes in serum sodium (r = 0.58, $p = 0.009$). There was no change in serum osmolality from pre- to post-race (290 ± 11 *vs.* 283 ± 12 mOsm/kg H_2O, $p = 0.15$). Serum aldosterone increased significantly during the race, from 8.3 ± 4.9 to 49.2 ± 3.1 ($p = 0.001$), and mean sodium excretion fell from 74.1 mEq/L pre-race to 19.4 mEq/L post-race ($p = 0.002$). A significant difference in plasma volume changes was observed between finishers and non-finishers mid-race. By 90 km non-finishers experienced a 5% decrease in plasma volume while those who went on to complete the course had expanded plasma volume by 3% (Fig. 3). Overall, finishers increased plasma volume by 11.8% from pre- to post-race.

Symptomatology

Gastrointestinal symptoms were experienced by 17 of the 26 participants. Upper Gi symptoms were far more prevalent than lower, with 15/26 describing nausea or vomiting but only 3/26 runners describing diarrhea or intestinal cramping. Runners with GiSx had greater energy intake, and specifically greater carbohydrate intake, than did runners not experiencing symptoms (Table 2). The rate of intake of other nutrients was similar between GiSx and No GiSx groups. Most runners used NSAIDs (18/26); however, their use was not associated with GiSx ($p = 0.50$). There were no differences in body mass changes or in serum sodium changes over time between those who did and those who did not use NSAIDs. Runners without GiSx had completed longer runs during training or racing than had runners with symptoms, 112 *vs.* 68 km ($p = 0.02$).

Nine of 26 runners complained of MSx, which ranged in severity from mild confusion to hallucinations. Dietary intakes were not different between runners with and without MSx. Of the finishers, mid-race serum Na was lower in those reporting MSx (137.5 mg/dL) compared to those with no MSx (141.9

a



b

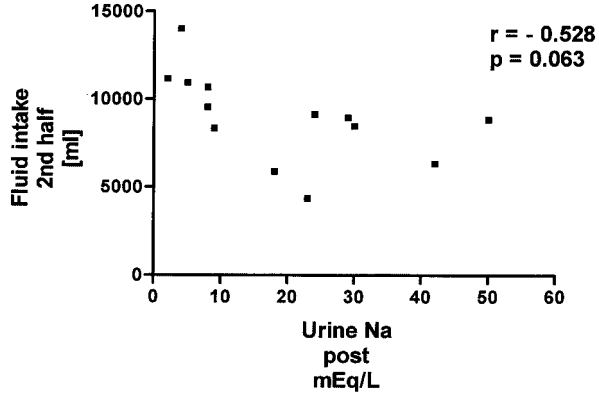


Fig. 2. Relationship of fluid intake to post-race serum and urinary sodium concentrations.

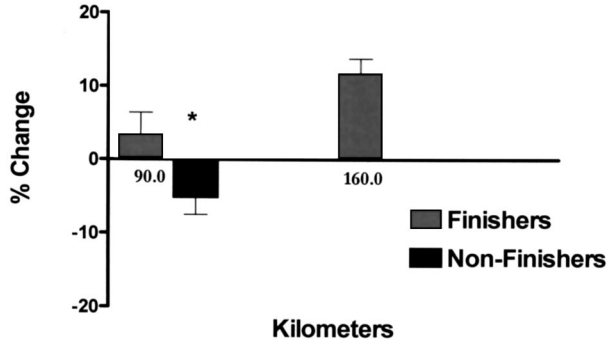


Fig. 3. Percent change in plasma volume from pre-race to mid-race for finishers and non-finishers *$p = 0.047$, and from pre- to post-race. Values are ± SD.

Table 2. Nutrient Intake and Gastrointestinal Distress

	Joules (J/kg/km)	CHO (g/kg/km)	Fat (g/kg/km)	Protein (g/kg/km)	Fluid ($mL/kg^{-1} \cdot km^{-1}$)	Na ($mg \cdot kg^{-1} \cdot km^{-1}$)
GI	0.61 ± 0.06#	0.15 ± 0.02*	0.01 ± 0.002	0.009 ± 0.002	1.6 ± 0.1	1.4 ± 0.2
No GI	0.33 ± 0.08#	0.09 ± 0.02*	0.01 ± 0.002	0.009 ± 0.002	1.5 ± 0.1	1.1 ± 0.2

Groups significantly different, $p = 0.005$.

* Groups significantly different, $p = 0.02$. Values are ± SD.

mg/dL, $p = 0.04$). The rate of serum sodium change did not differ between MSx (−0.037 mg/dL/km) and no MSx (−0.038 mg/dL/km). The rate of dietary potassium intake was lower in seven runners who complained of muscle cramping (27.2 mg/km) compared to those without cramping (46.8 mg/km, $p = 0.046$). There was no association between cramping and sodium or fluid intake prior to, or during, the race.

DISCUSSION

While other studies have examined dietary intake during prolonged exercise, many have used small sample populations [12,13], were limited by using diet recall methods immediately following the race [12] or used dietary data collected during subsequent telephone surveys [14]. While conveniently applied, these methods are associated with inaccuracies in dietary data and might be expected to be even less reliable after physically exhausting exercise. Furthermore, the ultra-endurance events described were of far shorter duration than the event being reported upon here [13,14]. This study is unique in that we repeatedly interviewed athletes regarding, and observed, food intake throughout more than 24 hours of continuous exercise.

Our sample population was fairly representative of the field of entrants: the mean finish times were 26.2 *vs.* 25.1 hours, respectively. In previous years the finish rate for this race has been approximately 75%; in 1999 the rate of completion was only 44% for the overall field of entrants and 50% for those runners for whom we collected data. The attrition rate was likely due to the extreme environmental conditions of high humidity and temperature.

Our most important finding was that body mass was very well-maintained throughout the race, despite extreme energy expenditure and thermal stress. A 3% to 4% decrease in body mass during ultra-marathons has been reported previously [15]. It has been suggested that failure to lose approximately 2 kg might actually represent fluid overload since the utilization of fat and glycogen stores should result in net loss of mass. The estimated energy deficit during this race ought to have resulted in a loss of approximately 0.64 kg of body mass, if all of the unmet energy needs came at the expense of body fat [17]. The estimated expense of body fat would account for all of the measured change in body mass and would suggest that runners experienced a slight relative gain in body fluids. Recommendations for fluid consumption during ultra-marathons have varied from 0.5 to 1 L/hour [18] to 0.6 to 2.4 L/hour [2]. Our data demonstrates that an intake of 0.74 liters of moisture per hour was more than sufficient to maintain body weight during such prolonged but low-intensity exercise and supports recommendations of fluid consumption of no more than 1 L/hour in ultra-marathons.

Fluid overload has been suggested to be the primary factor contributing to low serum sodium levels [19]. It has been estimated that ultra-distance athletes would need to lose about 4% of their body weight in order to maintain serum sodium [19, 20]. We observed a strong indirect relationship between weight change and serum sodium. Weight maintenance may represent an inappropriate retention of fluid in the vascular space, as suggested by the expansion of blood volume. Despite the cooler night temperatures and decreased pace during the latter stages of the race, fluid intake was similar between the first and second halves of the event. It is feasible that 19 liters of fluid ingested in one day exceeded sweat rate and the kidney's capacity to excrete excess fluid in some individuals.

Goldberger has recommended that athletes engaged in very prolonged exercise ingest 1 g/hour of sodium [7]. The athletes we observed ate approximately half that amount. Serum sodium concentrations overall tended to fall throughout the race, and plasma volume was expanded under environmental conditions which would be expected to result in a contraction of plasma volume and in hemoconcentration [21]. While serum sodium would typically be expected to drive plasma expansion, Gastmann *et al.* have suggested that plasma expansion during 24 hours of exercise may result from a carbohydrate-induced osmotic gradient [22]. Serum Na concentrations declined independently of serum osmolality; the high rate of carbohydrate intake may have contributed to serum osmolality and to the retention of fluid in the vascular space [19, 23, 24]. In a previous study at the same race we found that runners ingested nearly identical amounts of moisture and sodium while competing under far more mild conditions. We suspect that had conditions been less severe plasma volume may have expanded, and serum Na concentration declined, to an even greater degree.

Another important finding of this study is that runners seemed willing and able to ingest far more food than has been described. We believe that the extreme duration of exercise,

and therefore the reduced intensity of exercise, greatly influenced the amount and type of foods ingested. Running pace averaged 6.5 km/hour overall and only 5.7 km/hour in the second half, as compared to that of participants in a 100 km run who averaged nearly 10 km/hour [13]. While runners engaged in higher intensity exercise often rely heavily upon the carbohydrate contained in liquids or fruits to provide energy [25], runners in this study ate substantial quantities of solid food, including items such as fried chicken and peanut butter sandwiches, obtaining 17% of total energy from fats. Finishers consumed approximately 30,000 Joules, with a range of 11,832 to 47,359 Joules, at a mean rate of 176 J/km. This is similar to the data collected during a previous 160 km race [6], during the Alaskan Iditasport [12] and the Tour de France [26]. However, the observed rate we observed was fourfold greater than described by others during running races [13].

Dietary intake may have affected the ability of runners to finish the race. The deficit between estimated energy expended and energy consumed tended to be larger in non-finishers compared to finishers: finishers tended to more nearly meet their energy needs. Rehrer *et al.* estimated fluid intake to be 56.8 mL/km during a 67 km race, or half the rate we observed [14]. Our volunteers consumed a large amount of solid food which contributed a significant amount of moisture and may, in part, explain the high fluid intakes.

The incidence of mental status changes was quite high during the race. We found that mental status changes were associated with lower serum Na concentration and that serum Na concentrations were associated with high fluid intakes. Serum sodium concentration was lower mid-race in those athletes experiencing MSx, but there was no difference in dietary intake of sodium between those that did and did not report MSx. While high fluid intake increased the likelihood of complaints of mental status change, finishers did ingest fluid at a greater rate than those who dropped out of the race. The mental status changes, then, were not typically severe enough to result in withdrawal from the competition. In a previous study of ultra-marathoners competing on the identical course in less severe conditions we similarly found that those with mental status changes ingested more fluid than those without symptoms, but that overall finish time was not affected by the occurrence of mental status change [6].

Although dehydration has been postulated to contribute to GI distress [14], we found no relationship between markers of dehydration (i.e. weight loss, osmolality) and GI symptoms. The lack of a relationship could be due to the minimal dehydration experienced by runners in this study. Hypertonic carbohydrate beverages have been shown to increase risk of GI problems in triathletes [25]. We did find an increased risk of developing GI symptoms with increasing caloric intake, specifically of carbohydrate intake. The rate of carbohydrate intake was 54 g/hour, plus 5 g/hour of fat and protein, an intake which exceeds the limit suggested by Kreider *et al.* during ultra-distance running, of 15–50 g/hour [2]. As with the mental status change, however, most of the GI symptoms were not severe enough to warrant withdrawal from the race.

Rehrer *et al.* and Keefe *et al.* reported that lower GI symptoms were far more prevalent than upper GI symptoms in shorter ultra-marathon events [14,27]. However, we found a much greater incidence of moderate to severe upper GI distress, i.e. nausea or vomiting, than of lower GI symptoms. The high incidence of upper GiSx may be a function of race duration [6]. Better trained runners were less likely to experience GI distress. A protective effect of training on the incidence of GI symptoms has been reported previously [6,28]. Brocke *et al.* found that ultra-distance athletes often experience endotoxemia during very prolonged exercise with symptoms of nausea and vomiting, secondary to increased intestinal permeability [29]. Interestingly, better trained runners were found to have better immunity to endotoxins and fewer symptoms. The authors speculated that training may confer a protective effect by increasing antibodies to endotoxins.

CONCLUSION

We studied food and moisture intake, markers of serum electrolyte status and disturbances in gastrointestinal and mental status in runners engaged in 21 to 30 hours of near continuous exercise. The slow pace in this long duration race may have resulted in low sweat rates, while simultaneously allowing for greater food and fluid consumption than previously reported. Runners ate more than has been previously described in running races and ingested sufficient fluid to maintain body weight. Although high fluid intakes were associated with decreased serum sodium and increased risk of mental status change, finishers ingested fluid at a greater rate than those who did not complete the race and more closely met their energy requirements. Collectively, we observed a minimum of weight loss, a high incidence of upper gastrointestinal distress with high carbohydrate intake and an expansion in plasma volume despite extreme environmental conditions and exercise stress.

ACKNOWLEDGMENT

The authors wish to thank Maria Pagano for her enthusiastic assistance despite severe sleep deprivation, and Dr. Maria Devita for lending her expertise in renal handling of fluids and electrolytes.

REFERENCES

1. Coyle E, Coggan A, Hemmert M, Ivy J: Muscle glycogen utilization during prolonged strenuous exercise when fed carbohydrate. J Appl Physiol 61:165–172, 1986.

2. Kreider R: Physiologic considerations of ultraendurance performance. Int J Sports Nutr 1:3–27, 1991.
3. Backer H, Shopes E, Collins S, Barkan H: Exertional heat illness and hyponatremia in hikers. Am J Emerg Med 17:532–539, 1999.
4. Frizzel R, Lang G, Lowance D, Laham S: Hyponatremia and ultramarathon running. JAMA 255:772–774, 1986.
5. Surgenor S, Uphold R: Acute hyponatremia in ultra-endurance athletes. Am J Emerg Med 12:441–444, 1994.
6. Glace B, Murphy C, McHugh M, Nicholas S: Food and fluid intake: Relationship to gastrointestinal and mental function During an ultramarathon. Int J Sports Nutr, in press, 2002.
7. Goldberger E: "A Primer of Water, Electrolyte and Acid-Base Syndromes." Philadelphia: Lea and Febinger, 1986.
8. Jackson AS, Pollock ML, Ward A: Generalized equations for predicting body density of women. Med Sci Sports Exerc 12:175–181, 1980.
9. Jackson AS, Pollock ML: Generalized equations for predicting body density of men. Br J Nutr 40:497–504, 1978.
10. Strauss M, Davis J, Rosenbaum J, Eoaamwial E: "Water diuresis" produced during recumbency by the intravenous infusion of isotonic saline solution. J Clin Invest 30:862–868, 1951.
11. "ACSM: Guidelines for Exercise Testing and Prescription," 3rd ed. Philadelphia, PA: Williams and Wilkins, p 277, 1995.
12. Case S, Evans D, Tibbets G, Case S, Miller D: Dietary intakes of participants in the IditaSport Human Powered Ultra-Marathon. Alaska Med. 37:20–24, 1995.
13. Fallon K, Broad E, Thompson M, Reull P: Nutritional and fluid in a 100-km ultramarathon. Int J Sports Nutr 8:24–35, 1998.
14. Rehrer N, Brouns F, Beckers E, Frey W, Villiger B, Riddoch C, Menheere P, Saris W: Physiologic changes and gastrointestinal symptoms as a result of ultra-endurance running. Eur J Appl Physiol 64:1–8, 1992.
15. Speedy DB, Noakes TD, Kimber NE, Rogers IR, Thompson JMD, Boswell DR, Ross JJ, Campbell RGD, Gallagher PG, Kuttner JA: Fluid balance in the Ironman triathlon. Clin J Sport Med 11:44–50, 2001.
16. Speedy D, Campbell R, Mulligan G, Robinson D, Walker C, Gallagher P, Arts J: Weight changes and serum sodium concentrations after an ultradistance multisport triathlon. Clin J Sports Med 7:100–103, 1997.
17. Katch FI, McArdle WD: "Nutrition, Weight Control, and Exercise." Philadelphia, PA: Lea & Febiger, pp 155–178, 1988.
18. Burke LM: Fluid balance during team sports. J Sports Sci 15:287–295, 1997.
19. Speedy D, Noakes T, Rogers I, Thompson J, Campbell R, Kuttner J, Boswell D, Wright S, Hamlin M: Hyponatremia in ultradistance triathletes. Med Sci Sports Exerc 31:809–815, 1999.
20. Speedy DB, Rogers IR, Noakes TD, Wright S, Thompson JM, Campbell R, Hellemans I, Kimber NE, Boswell DR, Kuttner JA, Safih S: Exercise-induced hyponatremia in ultradistance triathletes is caused by inappropriate fluid retention. Clin J Sport Med 10: 272–278, 2000.
21. Hiller W: Dehydration and hyponatremia during triathlons. Med Sci Sports Exerc 21(5S):S219–S221, 1989.
22. Montain S, Laird J, Latzka W, Sawka M: Aldosterone and vasopressin responses in the heat: hydration level and exercise intensity effects. Med Sci Sports Exerc 29:661–668, 1997.
23. Gastmann U, Dimeo F, Huonker M, Bocker J, Steinacker J, Petersen K, Wieland H, Keul J, Lehmann M: Ultratriathlon-related blood-chemical and endocrinological responses in nine athletes. J Sports Med Phys Fitness 38:18–23, 1998.
24. Noakes T, Norman R, Buck R, Godlonton J, Stevenson K, Pittaway D: The incidence of hyponatremia during prolonged ultraendurance exercise. Med Sci Sports Exerc 22:165–170, 1990.
25. Rehrer N, van Kemenade M, Meester W, Brounds F, Saris W: Gastrointestinal complaints in relation to dietary intake in triathletes. Int J Sports Nutr 2:48–59, 1992.
26. Saris W: The Tour de France: food intake and energy expenditure during extreme sustained exercise. Cycling Sci 12:17–28, 1990.
27. Keefe E, Lowe D, Goss J, Wayne R: Gastrointestinal symptoms of marathon runners. West J Med 141:481–482, 1984.
28. Brouns F, Beckers E. Is the gut an athletic organ? Digestion absorption and exercise. Sports Med 15:242–257, 1993.
29. Brocke-Utne J, Gaffin L, Wells M, Gathiram P, Sohar E, James M, Morell D, Norman R: Endotoxaemia in exhausted runners after a long-distance race. S Afr Med J 73:533–536, 1988.
30. Knochel J: Clinical complications of body fluid and electrolyte imbalance. In Buskirk E, Puhl S (eds): "Body Fluid Balance Exercise and Sport." Boca Raton, FL: CRC Press, pp 297–317, 1996.

Received December 24, 2001; revision accepted July 3, 2002.