Food and Fluid Intake and Disturbances in Gastrointestinal and Mental Function During an Ultramarathon

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The purpose of this study was to document eating strategies employed by runners during a 160-km race, and to identify eating patterns that predispose the runner to disturbed mental or gastrointestinal functioning. We monitored intake in 19 volunteers during the 12 hours pre-race. Intake was determined by interview with runners approximately every 12 km throughout the race. The mean finish time was 24.3 hours, with 4 runners not completing the race. Body mass decreased during the race, 75.9 ± 2.3 kg to 74.4 ± 2.2 kg (p < .001). Runners ingested 2643 kcal during the 12 hours prerace (68% carbohydrate) and 3.8 L of fluid. During the race 6047 kcal, 18 L of fluid, and 12 g of sodium were consumed. Gastrointestinal distress (GI) was experienced by half of the participants, but was unrelated to food or fluid intake. Upper GI symptoms were more prevalent than lower and occurred mainly after 88 km. Runners with GI distress tended to complete fewer training miles (p = .10) and to do shorter training runs (p = .08). Half of the volunteers reported mental status changes (MSC), such as confusion or dizziness. Runners with MSC had greater intake of total calories, carbohydrate, and fluid (p < .05) than runners without MSC. They also completed shorter training runs (p = .03). Caloric and moisture intake for all runners far exceeded intakes described previously. Although intake did not match energy expenditure, it may represent the upper limit for absorption during exercise, and very high food and/or fluid intake appears to lead to perturbed mental status.

Key Words: running, hyponatremia, hydration

Introduction

Ultraendurance athletes face unique dietary challenges during their competitive events. These races commonly require physical exertion for 6 to 30 hours at a time, and may continue for many days. They include triathlons, road and trail foot races, and cycling events like the Tour de France.

Little nutritional research has been conducted on participants in ultraendurance running events. A case study of 1 male runner estimated an expenditure of 10,720
calories during 160 km of running (24). Other studies have also been based upon one or two individuals (4, 32). Rehrer et al. relied upon diet recalls to estimate intake for 172 runners participating in a 67-km race. Carbohydrate beverages provided most of the 516 calories ingested during the race (29).

Running is more likely to result in gastrointestinal distress than sports such as bicycling (37). Severe gastrointestinal discomfort will limit performance and, in fact, bowel dysfunction is reported to be the most common cause for interrupting running (37). Rehrer et al. found that 43% of runners competing over an 8–10-hour period experienced gastrointestinal distress (29). The symptomatic runners experienced significant declines in body mass and plasma volume, consistent with moderate dehydration. The likelihood of developing gastrointestinal problems has been found to be greater when hypertonic beverages or fiber rich foods are consumed (30). Rehrer also demonstrated that dietary intake just prior to, or during, exercise increases the occurrence of gastrointestinal symptoms (31).

Reports indicate that low blood sodium, or hyponatremia, is a relatively common occurrence during ultraendurance exercise (12, 26). O’Toole et al. reported that 29% of all finishers of the 1984 Hawaii Ironman were hyponatremic (26). Hyponatremic athletes present with a variety of symptoms that range from nausea, weakness, confusion, and incoordination to grand mal seizures and coma (38). Surgenor et al. described a case of hyponatremia in a 160-km race: The runner presented with acute mental status changes and eventually collapsed (38).

Adequate carbohydrate must be taken to extend performance (6), and electrolytes and fluids must be consumed to prevent hyperthermia and to maintain aerobic capacity during prolonged endurance exercise (20). The athlete must achieve a balance between the need to maintain energy intake and hydration, and the maintenance of gastrointestinal and mental functioning. Previous investigations of food intake during ultraendurance running have typically been limited to events of relatively short duration. The purpose of this study was to record food and fluid intake preceding and during a 160-km trail run and to examine the relationship of intake to mental status and gastrointestinal distress.

**Methods**

**Subjects**

Eighteen men and 1 woman were recruited by letter 3 months prior to the event. In an effort to minimize the dropout rate, eligible subjects had to have completed at least one race of 50 or more miles in the previous 12-month period and had to have an anticipated finish time of less than 23 hours. Runners agreeing to participate were mailed medical and running history questionnaires, and instructions as to what was expected of them during the study. 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.

**Protocol**

On the day prior to the race, before the pre-race meal, subjects reported to the research staff. The medical history questionnaire and consent form were collected and reviewed with the runner. A urine sample was collected, and body mass was
determined (with shoes) on a calibrated scale. Body fat percentage was estimated by
skin fold measures using the Skyndex (Caldwell, Justiss, & Company, Inc.,
Fayetteville, AR) skinfold method, utilizing generalized equations for men and
women (17, 18). The percent body fat was then used to estimate fat mass. Pre-race
diet records were provided, and the runner was instructed to record all items con-
sumed during the 12 hours preceding the race. In addition, the volume of the beverage containers to be carried during the race was verified.

Food records were collected just prior to the 4:00 AM race start. Investigators
attended 12 of the 37 food stations throughout the race so that the runners were
interviewed regarding their physical and mental condition, and regarding dietary intake, at approximately 13-km intervals. Each of the staffed food stations provided
similar foods. A checklist of supplied food items was used to record the type and
quantity of food and beverage consumed. Each time the participating runners were
seen, they were asked to recall any items of food they had eaten or beverages they
had drunk since the last observation point. Incidence of gastrointestinal distress or
changes in mental status was by self-report. Investigators interviewed each subject
as to the occurrence of any gastrointestinal distress. Runners were asked to describe
symptoms, approximately when they occurred, and duration. During each interview
the subjects were observed for alteration in mental status and were asked if they had
experienced confusion or disorientation since last questioned.

Immediately after the finish, and prior to taking any further food or fluid, the
athletes provided another urine sample, were reweighed and towel dried, and body
fat measurements were repeated. All urine samples were analyzed immediately for
glucose and protein with a urine dipstick and visual chart comparison (Multistik 10
SG reagent strip, Bayer Corp., Pittsburgh, PA). Urine dipstick analysis yields a 95–
98% accuracy rate for screening urine protein levels (34). Aliquots of the urine
sample were frozen and later analyzed for osmolality using the freezing point de-
pression method (Instrumentation Laboratories, Lexington, MA), and for sodium,
potassium, and creatinine using flame photometry (Instrumentation Laboratories,
Lexington, MA).

Dietary Analysis

The Nutritionist IV software program (N2 Computing, San Bruno, CA) was used to
calculate the nutritional composition of the foods and fluids consumed. Runners
were interviewed by phone if clarification was required (i.e., to obtain brand names
of specialized “sport” supplements). The manufacturers were subsequently con-
tacted to provide nutritional composition of their products, and the data was added to
the nutritional software database. Where possible, food was obtained and was weighed
in our laboratory according to typical serving size. For example, we standardized a
“handful” of potato chips as follows: Five staff members were asked to take a
handful of the specified food, the food was weighed, and the mean weight was used
to calculate dietary content of a typical “handful.” Diets were analyzed for total
calories, macro- and micro-nutrients, as well as total moisture content.

Total intake for the race was calculated as well as intake per kilometer com-
pleted. This allowed us to include intake data of finishers and non-finishers alike. In
order to evaluate changes in rate of intake over time, the data were also analyzed for
each quarter of the race. The division into quarters was determined by picking four
manned stations that divided the total race distance into four sections of approximately
40 km each. Energy expenditure during the event was estimated using a stepwise calculation using body mass, mean velocity, and time spent running (1).

**Statistical Analysis**

Subjects were classified according to symptoms of gastrointestinal distress, such as nausea, vomiting, or diarrhea (GI) and according to alterations in mental status, such as confusion, disorientation, or inability to concentrate (MSC). The absence of symptoms is indicated as No GI and No MSC, respectively. Changes over time were analyzed using a 2-way analysis of variance (group × time) with repeated measures on time. The data collected from the 8 monitoring stations were grouped in pairs and reported as intake per quarter. When significant main effects of time were found, Student’s t tests with Bonferroni corrections were used to identify significant differences between intake per kilometer during each quarter of the race. Since these were 4 × 2 comparisons (4 times and 2 groups), a significant interaction term indicates that changes in intake over time were different between groups. Because non-steroidal anti-inflammatory drugs (NSAIDS) are associated with GI distress, ANCOVA with NSAID intake as a confounding variable was used to compare intake between GI and No GI groups. Changes in anthropometrics and urine chemistries were also analyzed using 2-way ANOVA. Bivariate correlations were used to examine the relationship between body mass, body composition, and finish time. Statistical significance was established for an alpha level of .05.

**Results**

The mean weight of the 19 subjects that began the race was 75.9 kg, and the mean body fat was 14.6%. Their typical weekly training distance during the 3 months preceding the race was 98.3 km/week, with their longest training run during the same period averaging 88 km. There was a significant correlation between body mass and composition and finish time (i.e., slower runners weighed more and were fatter than faster runners; p < .05). Fourteen of the men completed the 160-km course in a mean finish time of 24.3 (±0.67) hours. During the race, runners lost 1.6 kg of body mass (p < .001), with body fat accounting for 1.13 kg of the weight loss according to skinfold measures (p < .05). Loss of lean body compartments (protein and fluid mass), then, is estimated to have been 0.47 kg. Pre-race urine osmolality was 488.1 mOsm/kg H₂O (±103.7), with 8 of 19 runners presenting with urine osmolality greater than 500 mOsm/kg. There was no difference in pre-race urine osmolality in finishers as compared to non-finishers (536 ± 98 vs. 390 ± 169 mOsm/kg; p = .55); neither was there a difference in urine osmolality pre versus post race among the finishers (536 ± 98 vs. 745 ± 69 mOsm/kg, p = .11, pre vs. post race, respectively).

Food intake for the 12 hours pre-race is summarized in Table 1. Runners ingested approximately 4 L of moisture, including that contained in food items, and 2642 kcal, of which 67% came from carbohydrate, 21% from fat, 10% from protein, and 2% from alcohol. Those who finished the race consumed 6047 kcal during the race, with carbohydrates contributing 82% of the energy. Nearly 18 L of fluid were ingested during the race, with moisture contained in solid foods accounting for nearly 30% of total fluid consumption. Sodium intake was only 12.5 g, or approximately 0.5 g per hour. Four runners did not finish the race; in order to consider their
Table 1  Pre-race Dietary Intake 12 hours

<table>
<thead>
<tr>
<th>Group</th>
<th>Kcal</th>
<th>Carbo (g)</th>
<th>Fat (g)</th>
<th>Pro (g)</th>
<th>Fiber (g)</th>
<th>Fluid (ml)</th>
<th>Na (mg)</th>
<th>K (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All (n = 19)</td>
<td>2642 ± 403</td>
<td>450 ± 78</td>
<td>62 ± 10</td>
<td>63 ± 9</td>
<td>13 ± 2</td>
<td>3872 ± 534</td>
<td>3128 ± 548</td>
<td>2541 ± 394</td>
</tr>
<tr>
<td>MSC (n = 11)</td>
<td>3413* ± 620</td>
<td>592* ± 123</td>
<td>83* ± 15</td>
<td>79 ± 15</td>
<td>16 ± 4</td>
<td>4493 ± 855</td>
<td>4115 ± 887</td>
<td>3207 ± 630</td>
</tr>
<tr>
<td>No MSC (N = 8)</td>
<td>1776* ± 308</td>
<td>390* ± 57</td>
<td>39* ± 9</td>
<td>45 ± 7</td>
<td>10 ± 2</td>
<td>3172 ± 560</td>
<td>2016 ± 334</td>
<td>1794 ± 300</td>
</tr>
<tr>
<td>GI (n = 11)</td>
<td>2374 ± 448</td>
<td>395 ± 69</td>
<td>63 ± 15</td>
<td>56 ± 15</td>
<td>10 ± 2</td>
<td>3728 ± 645</td>
<td>3035 ± 792</td>
<td>1971 ± 393</td>
</tr>
<tr>
<td>No GI (N = 8)</td>
<td>2881 ± 666</td>
<td>498 ± 137</td>
<td>62 ± 15</td>
<td>69 ± 12</td>
<td>16 ± 4</td>
<td>3999 ± 865</td>
<td>3209 ± 801</td>
<td>3049 ± 629</td>
</tr>
</tbody>
</table>

*Note. Data reported ± SEM. MSC, runners experiencing changes in mental status during race; No MSC, runners not experiencing changes in mental status; GI, runners experiencing nausea, vomiting, or diarrhea during race; No GI, runners not experiencing GI symptoms during race. *Students t tests: MSC > No MSC, p < .05.
**Table 2  Rate of Dietary Intake During Race Per Kilometer**

<table>
<thead>
<tr>
<th>Group</th>
<th>Kcal (±SEM)</th>
<th>Carbo (g) (±SEM)</th>
<th>Fat (g) (±SEM)</th>
<th>Pro (g) (±SEM)</th>
<th>Fiber (g) (±SEM)</th>
<th>Fluid (ml) (±SEM)</th>
<th>Na (mg) (±SEM)</th>
<th>K (mg) (±SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All (n = 19)</td>
<td>33.5 ± 3.7</td>
<td>6.8 ± 0.62</td>
<td>0.47 ± 0.08</td>
<td>0.49 ± 0.06</td>
<td>0.16 ± 0.02</td>
<td>103.5 ± 5.7</td>
<td>66.3 ± 8.3</td>
<td>42.0 ± 409</td>
</tr>
<tr>
<td>MSC (n = 10)</td>
<td>39.8*± 5.0</td>
<td>8.1*± 1.2</td>
<td>0.57 ± 0.13</td>
<td>0.53 ± 0.09</td>
<td>0.18 ± 0.03</td>
<td>108.0 ± 9.3</td>
<td>69.0 ± 12.2</td>
<td>42.3 ± 6.2</td>
</tr>
<tr>
<td>No MSC (n = 9)</td>
<td>26.0*± 1.9</td>
<td>5.6*± 0.45</td>
<td>0.37 ± 0.09</td>
<td>0.45 ± 0.05</td>
<td>0.14 ± 0.02</td>
<td>98.9 ± 6.8</td>
<td>64.0 ± 11.2</td>
<td>36.1 ± 5.6</td>
</tr>
<tr>
<td>GI (n = 11)</td>
<td>37.0 ± 5.6</td>
<td>7.5 ± 1.2</td>
<td>0.52 ± 0.14</td>
<td>0.51 ± 0.06</td>
<td>0.16 ± 0.02</td>
<td>106.0 ± 8.1</td>
<td>67.2 ± 11.2</td>
<td>41.5 ± 6.2</td>
</tr>
<tr>
<td>No GI (n = 8)</td>
<td>30.4 ± 3.7</td>
<td>6.2 ± 0.63</td>
<td>0.43 ± 0.09</td>
<td>0.47 ± 0.09</td>
<td>0.16 ± 0.02</td>
<td>100.0 ± 8.7</td>
<td>65.7 ± 1.2</td>
<td>36.6 ± 5.6</td>
</tr>
</tbody>
</table>

*Note.* Includes both finishers and non-finishers. Data reported ±SEM. MSC, runners experiencing changes in mental status during race; No MSC, runners not experiencing changes in mental status; GI, runners experiencing nausea, vomiting, or diarrhea during race; No GI, runners not experiencing GI symptoms during race. *Students t* tests: MSC > NO MSC, p < .05.
Figure 1 — Intake per quarter. Energy and fluid intake expressed per kilometer during each quarter of the 160-km race. Values represent mean $\pm$ SEM. Kcal: ANOVA with Bonferroni corrections: effect of time ($p < .01$), 2nd quarter > 1st or 4th quarters. Fluid: ANOVA with Bonferroni corrections: effect of time ($p < .01$), 1st quarter < 2nd, 3rd, or 4th quarters.

Figure 2 — Gastro-intestinal symptoms and mental status changes. Upper gastrointestinal symptoms (nausea or vomiting); lower gastrointestinal symptoms (diarrhea). Mental status changes are defined as dizziness, confusion, or disorientation.
Intake During a 160-km Trail Run

Data, we also expressed intake adjusted per kilometer completed (Table 2). Caloric intake was greatest during the second quarter of the race (45 to 88 km) when participants ingested 46 kcal/km (Figure 1). Runners drank the most fluid during the second and third quarters of the race, consuming a peak of 133 ml/km (Figure 1). ANOVA did reveal an effect of distance, with intake of fluids, Na, K, CHO, fat, protein, and calories greatest during the middle portion of the race.

Nine runners experienced gastrointestinal symptoms at some point during the race (Figure 2). Upper gastrointestinal symptoms were more prevalent than lower gastrointestinal symptoms. One runner experienced upper GI symptoms in the first half of the race; 8 runners experienced nausea or vomiting during the second half. Nausea and vomiting, which persisted from 88 km through 133 km, forced one runner out of the race. Only two men suffered with lower gastrointestinal distress. Finish times did not differ between those who did/did not suffer GI disturbances, 24.1 versus 22.8 hours, respectively ($p = .42$).

We did not observe an effect of dietary intake/kilometer on the development of GI symptoms (Table 2). A strong trend was noted for non-steroidal anti-inflammatory drugs (NSAIDS) to contribute to gastrointestinal symptoms ($p = .08$). However, ANCOVA with NSAIDS as the confounding variable did not demonstrate a significant difference in dietary intake between GI and NoGI. There was no significant difference in weight loss, or prerace urine osmolality between those who did and did not suffer from GI symptoms. Runners who experienced GI tended to have trained less: 85 versus 103 km/week ($p = .10$). Additionally, there was also a trend for runners with GI to have done shorter training runs, 76 km versus 93 km ($p = .08$).

The incidence of MSC was also high: Ten runners reported confusion and/or lightheadedness. Two runners cited confusion as the reason for not finishing the race. Among those runners who completed the entire race, final time was not different between those who did or did not experience MSC ($p = .63$). Most MSC were reported after 88 km and were described as lightheadedness, disorientation, or “bonking” (inability to concentrate, confusion; Figure 2). Runners experiencing MSC during the race consumed nearly twice as many kilocalories (fat and carbohydrate) pre-race as compared to those without symptoms. Those runners complaining of MSC did not differ in body weight from the asymptomatic runners, although they had averaged significantly fewer miles in training (84 vs. 108 km/week, $p = .03$). Runners experiencing MSC continued the pattern of increased food intake throughout the race, consuming significantly more kilocalories, carbohydrate, and fluid than did the No MSC group, and tending to eat more protein and fat (Figures 3, 4).

**Discussion**

The mean finish time for our volunteers was very similar to that of the entire field of competitors, 24.3 versus 24.2 hours. The finish rate for the total field of participants in the 160-km race was approximately 67%, while the finish rate for our sample population was 74%. These finishers consumed in excess of 6000 kcal during the 24-hour race, similar to the daily intakes reported for cyclists during the Tour de France (33) and more per kilometer than values previously reported for ultramarathoners (11, 29). Fallon et al. reported a caloric intake of 10 kcal/km during a 100-km race as compared to our observed intakes of approximately 33 kcal/km. Runners in Fallon’s study averaged 10 km/h, while ours covered only 6.6 km/h. The slower pace may have permitted greater intakes.
Figure 3 — Calories per quarter. Calories per kilometer from protein, fat, and carbohydrate, expressed per quarter of the race. MSC, mental status changes. NMSC, no mental status changes. Kcal: ANOVA with Bonferroni corrections, effect of time and condition, $p < .001$. Carbohydrate: ANOVA with Bonferroni corrections, effect of time and condition, $p < .001$. Fat: ANOVA with Bonferroni corrections, effect of condition, $p = .056$. Protein: ANOVA with Bonferroni corrections, effect of time $p < .01$, effect of condition, $p = .08$.

Figure 4 — Fluid intake. Fluid per kilometer, expressed per quarter of the race. Values represent mean $\pm$ SEM. MSC, mental status changes. NMSC, no mental status changes. ANOVA with Bonferroni corrections, effect of time and condition, $p < .01$. 
Total energy expenditure was estimated to be approximately 13,560 kcal, while intake averaged only 6047, thus creating an energy deficit of nearly 7513 kcal in a 24-hour period. The unmet caloric needs would be expected to result in a loss of about 1-kg body fat if all the caloric needs were met at the expense of body fat stores (19). In keeping with the caloric deficit, we did estimate a body fat loss of approximately 1 kg from start to finish of the race using skin fold methods.

Kreider suggests that the potential energy available from carbohydrate feeding during exercise is 60–200 kcal/hour (20). The volunteers that completed the entire 160 km consumed 176 kcal/hour from carbohydrate sources, nearing the proposed limit for carbohydrate intake during exercise. Although their total energy intake fell well short of their estimated caloric expenditure, it may have approached the physiologic maximum for absorption during exercise.

Several runners later admitted that they had withheld food and fluid until their body mass could be determined the day prior to the race. Competitors are eliminated from the race if their weight falls to < 93% of their pre-race weight. They, therefore, attempted to weigh in as light as possible, hoping for greater leeway for weight change during the run. Our pre-race weights, therefore, might be somewhat skewed downward. Runners who had withheld fluids stated that they hoped to rehydrate prior to the race, and on average they did ingest nearly 4 L of moisture during the 12 hours immediately preceding the race. Given the high rate of fluid consumption, urine produced just prior to the 4 AM race start may have been less concentrated than the sample we collected. However, some of the competitors may not have been able to fully restore fluids and may have begun the race with marginal or poor hydration status.

Fluid intake during the race was far greater than previously described for ultramarathoners. Rehrer et al. (29) estimated fluid intake to be 56.8 ml/km during a 67-km race, as compared to approximately 100 ml/km in our study. Our volunteers consumed a large amount of solid food, which contributed significant moisture content to the diet and may in part explain the larger fluid volume observed. Runners in this study ingested sodium at a rate of 0.5 g/h, far less than the 1–2 g of sodium per hour recommended for ultraendurance athletes (13).

**Gastrointestinal Symptoms**

Most investigations of eating practices during long-distance running have focused upon events lasting less than 12 hours (11, 31). Our study is unique in that it monitored volunteers who exercised nearly continuously for 24 hours. Rehrer et al. reported that approximately 17% of the participants in a 67-km race reported moderate to severe upper GI distress (i.e., nausea or vomiting) as compared to 47% in our 160-km race. It is likely that the duration of the event contributed to the observed differences, since we noted most GI problems after 88 km.

We were unable to find any significant relationship between food intake and GI distress in runners exercising for 24 hours. Factors other than diet were likely responsible for the high incidence of nausea and vomiting documented. Neuroendocrine changes, stress, non-steroidalals, dehydration, and endotoxemia have all been postulated to contribute to GI dysfunction during exercise (25, 27, 29). Dehydration decreases GI blood flow and has been demonstrated to be associated with GI symptoms during exercise in some studies (30). However, runners in our study apparently maintained body weight fairly well while covering 160 km. Dehydration, then, was
not likely responsible for the GI symptoms observed. Those runners with GI and without GI symptoms lost similar amounts of weight.

Importantly, more highly trained runners tended to report less GI problems, consistent with the contention put forth by Brouns et al. that training decreases the risk of GI symptoms during races (3). Brocke-Utne et al. found that nausea, vomiting, and diarrhea were associated with endotoxemia during an ultramarathon race (2). The authors suggested that endotoxemia increases with increasing exercise duration and that training may impart a protective effect by increasing anti-endotoxins. These explanations are consistent with the protective effect of training that we observed. It is possible that leakage of endotoxins into portal circulation during the latter stages of the 160-km race promoted nausea and vomiting.

NSAIDS are known to induce ulceration and perforation of the gastrointestinal tract, which is associated with bleeding, nausea, and vomiting (6). There was a strong trend between NSAID intake, and nausea and vomiting. Although NSAIDs may not have directly caused GI symptoms in our subjects, they may have exacerbated mucosal damage.

The ultramarathoners we studied were better able to tolerate solid foods, concentrated beverages, and fiber than athletes working at higher intensities over much shorter distances (28, 29). Runners competing in events of very long duration typically work at only 45–50% of maximum aerobic capacity (8). The GI disturbances we observed during 160 km of relatively low intensity running seem more likely related to stress-mediated metabolic changes or to changes in mucosal integrity than to food intake per se.

Mental Status Changes

The incidence and severity of mental status changes were unexpected. Runners described the changes as “bonking,” dizziness, inability to concentrate, confusion, or as being “out of it.” Symptoms of dehydration-induced heat exhaustion include dizziness, incoordination, and confusion (9). However runners experiencing MSC consumed more fluid per kilometer than asymptomatic runners, making dehydration an unlikely explanation. This suggests that over-consumption of fluid may have contributed to impaired mental status. Anecdotally, one participant quit the race at 100 km, complaining that he could not concentrate, was feeling “spacey,” and had not urinated during the race despite copious fluid intake. After the race he experienced severe nausea, followed by the frequent need to urinate. This subject’s intake of sodium per kilometer was approximately half that of the other volunteers’. The high incidence of disturbed mental functioning we observed may have been related to over-consumption of fluids relative to sodium intake.

Hyponatremia, frequently observed in ultraendurance athletes (26, 38), is marked by disorientation, confusion, dizziness, and incoherent speech (9, 22). Incomplete restoration of sodium or over-consumption of fluid might result in reductions in serum sodium. Hiller et al. has suggested that 1–2 g/h of sodium would prevent hyponatremia; runners in this study consumed far less: 0.63 g/L (13). Fluid overload has been cited as a mechanism for hyponatremia (21, 36). Ironman triathletes were predicted to require a 4% loss in body mass in order to maintain serum sodium levels, and those athletes who maintained or gained mass while racing were found most likely to be hyponatremic. Hyponatremia was more common in slower runners, perhaps because slower runners had lower rates of fluid loss and greater
opportunity to consume fluid (36). The slow pace of athletes in our study (7 km/h) may also have allowed them to over-ingest fluid: symptomatic runners drank 0.8 L/h, similar to the rate of fluid ingestion previously observed in hyponatremic athletes (15). In general, runners in our study consumed less sodium than recommended and maintained body weight very well; additionally, those with MSC consumed more fluid than those without symptoms. Although we may speculate that some of the runners describing MSC may have been hyponatremic, we did not draw blood and have no way of confirming the condition.

Hypoglycemia, a condition marked by tremulousness, confusion, fatigue, and cognitive failure (7), may also have been responsible for some of the changes in mental status observed. The MSC group ate 50% more kcal and carbohydrates than the asymptomatic group. Carbohydrate intake increases insulin during exercise of less than 50% of VO$_{2\text{max}}$ (16). Runners in this race averaged just 6.6 km/hour; the exercise may have been of insufficient intensity to suppress insulin. Hyperinsulinemia, in conjunction with the heightened insulin-sensitivity seen in trained individuals, may have triggered a reactive hypoglycemia, similar to that seen when carbohydrates are taken close to the initiation of exercise (10).

**Conclusions**

Food and fluid intake during a 160-km race was far greater than previously reported for ultramarathoners, although sodium intake was less than recommended. The majority of runners experienced impairments in mental status, and gastrointestinal distress occurred in nearly half the runners. Intake was not different between those who did/did not experience GI symptoms. It appears that GI distress in very prolonged events may be more closely related to metabolic factors than to food intake. Food and fluid intake was greater for runners experiencing changes in mental status, compared to asymptomatic runners. Future research should focus on the effect of food and fluid intake on serum electrolytes and glucose, and the relationship of these values to maintenance of gastrointestinal function, mental status, and performance.

**References**


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