Physiological changes and gastro-intestinal symptoms as a result of ultra-endurance running


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Summary. One hundred and seventy-two competitors of the Swiss Alpine Marathon, Davos, Switzerland, 1988, volunteered for this research project. Of these volunteers 170 (158 men, 12 women) finished the race (99%). The race length was 67 km with an altitude difference of 1,900 m between the highest and lowest points. Mean age was 39 (SEM 0.8) years. Average finishing times were 8 h 18 min (men) and 8 h 56 min (women). Loss of body mass averaged 3.4% body mass [mean 3.3 (SEM 0.2)%; 4.0 (SEM 0.4)%; men and women, respectively]. Blood samples from a subgroup of 89 subjects (6 women and 83 men) were taken prior to and immediately after completion of the race. Changes in haemoglobin (9.3 mmol·l⁻¹ pre-race, 9.7 mmol·l⁻¹ post-race) and packed cell volume (0.44 pre, 0.48 post-race) were in line with the moderate level of dehydration displayed by changes in body mass. Mean plasma volume decreased by 8.3%. No significant changes in plasma osmolality, sodium, or chloride were observed but plasma potassium did increase by 5% (4.2 mmol·l⁻¹ pre-race, 4.4 mmol·l⁻¹ post-race). Mean fluid consumption was 3290 (SEM 103) ml. Forty-three percent of all subjects, and 33% of those who gave blood samples, complained of gastro-intestinal (GI) distress during the race. No direct relationship was found between the quantity or quality of beverage consumed and the prevalence of GI symptoms. The circulating concentration of several GI hormones was measured and several were found to be significantly elevated (P<0.05) after the race [mean values: gastrin 159.6 (SEM 17.8) ng·l⁻¹; vaso-active intestinal peptide 224.3 (SEM 20.1) ng·l⁻¹; peptide histidine isoleucine 311.1 (SEM 27.5) ng·l⁻¹; motilin 214.1 (SEM 15.1) ng·l⁻¹] but larger increases were not found to be significantly correlated with GI symptoms. Plasma cortisol, adrenaline, and noradrenaline concentrations were significantly higher after the race compared to resting values (P<0.05). There was a trend for post-race noradrenaline values to be lower in sufferers of GI disturbance. The post-race plasma noradrenaline concentration was significantly lower specifically in those runners with intestinal cramps. Also, the resting plasma cortisol concentration was significantly lower in those individuals who developed intestinal cramps during the race. Plasma creatine phosphokinase, alanine aminotransferase and aspartate aminotransferase activities were increased following the race, which may indicate that there was tissue damage. An increase in plasma potassium concentration was observed after the race in individuals with GI complaints [0.29 (SEM 0.07) mmol·l⁻¹ increase], whereas no increase was observed in individuals without GI symptoms. An inability of the Na⁺-K⁺ pump to keep pace with the needs of skeletal muscle (as well in the intestinal tract) may have accounted for the high plasma potassium values immediately following exercise and may have played a role in the development of GI disorders. However, many other sources of K⁺ release may have accounted for the elevated plasma K⁺ (skeletal muscle, liver and red blood cells) in such sufferers and the correlation between the increase in K⁺ and GI symptoms may be an indirect one.

Key words: Gastro-intestinal symptoms – Exercise – Cell damage – Catecholamines – Gastro-intestinal hormones – Electrolytes

Introduction

Over the last decade research on marathon running and endurance exercise in general has contributed greatly to the growing body of knowledge concerning normal and abnormal physiological function. Extreme situations, such as in competitive endurance events push the human body to its limits. The weak links and the intricate coupling of metabolic pathways with substrate availability can be more clearly identified when the system is stressed.
A number of physiological and biochemical changes associated with marathon running have been described. In addition to what may be considered normal responses to the physical stress of this type of exercise are abnormal changes and health risks. One such ailment which has gained some attention in the past few years is gastro-intestinal (GI) disturbance. Although there is much descriptive data, little information has been gathered regarding the aetiology of GI symptoms, or preventive measures which can be taken to avoid these problems. GI complaints are common among distance runners (Brouns et al. 1987; Fogoros 1980; Sullivan 1981). Prevalence among marathon runners has been reported to range from 30% to 50% (Keeffe et al. 1984; Rehner et al. 1989).

Several possible factors have been described which may predispose to GI disturbance. Dehydration at levels of 4%-5% loss of body mass has been found to be associated with an increased prevalence of GI distress among marathon runners (Neufer et al. 1989; Rehner et al. 1989, 1990a). During competition, specific aspects of the dietary intake of triathletes have been observed to be associated with GI symptoms. Greater fat, fibre and protein intakes (solid food) and hypertonic beverage (greater than 800 mosmol·kg⁻¹) ingestion have been associated with GI distress (Rehner et al. 1990b). It is thought that intensity of exercise and training status may increase the risk of GI disorders but well-trained athletes with more experience seem less prone to these ailments (Brouns et al. 1987).

In foot races such as the marathon (42.2 km) where sweat losses easily reach 1 l·h⁻¹ and it is difficult to drink, fluid balance is difficult to maintain. In ultramarathon competitions (longer than 42 km) one would expect that the changes and symptoms appearing as a result of marathon running would be accentuated.

The Swiss Alpine Marathon in Davos, Switzerland, is an arduous 67-km race with steep ascents, descents, and rugged terrain, making this race particularly demanding. Since the year of its conception, 1986, data from a number of participants have been gathered, in cooperation with the organisers, regarding changes in body mass and a number of plasma parameters, to monitor the health of the participants (Frey 1988). The results presented in this report come from data collected during the third Swiss Alpine Marathon in 1988. The goals of this study were to investigate further the prevalence of GI dysfunction, as well as estimates of dehydration and physiological stress, which may or may not be related to GI symptoms.

Methods

The Swiss Alpine Marathon is a 67-km ultramarathon run on a circular course with an altitude difference of 1900 m between the highest and lowest points. The profile of the course is shown in Fig. 1. On both sides of the mountain pass, reached at 50 km, are ascents and descents of as much as 23%. Drink posts are provided approximately every 7 km with water as well as beverages containing carbohydrate and electrolytes available. Competitors were recruited for this study 1 or 2 days before the 1988 race, when they came to collect their start numbers. An attempt was made to recruit as many competitors as possible.

On the day of the race at 8.00 a.m. at the start in Davos, the dry bulb temperature (TDB) was 11°C with a relative humidity of 72%. At the 50-km point, Sertig Pass, at 11.00 a.m. TDB was 7°C with a relative humidity of 64%. One hundred and seventy of the 172 runners who volunteered for the project (99%) completed the total distance within the allotted time (12 h). There were 875 participants in total (including 50 women) who finished the race within the time limit.

Blood samples (10 ml) were taken at rest from the first 101 participants who volunteered during registration, 24-48 h prior to the race. Of these 101 subjects, 89 (6 women and 83 men) finished the race and gave a blood sample after the race. On the morning of the race body mass was measured. Prior to the race (1 or 2 days), subjects were familiarized with the questionnaire requiring completion after the race. This included a list of all refreshment posts and drinks offered at each post and space to write in the number of cups of fluid consumed at each post. The maximal capacity of a cup was 150 ml. Since cups were not completely filled and spillage was common, an average content of 100 ml per cup was assumed. In addition, a list of possible gastro-intestinal ailments was printed and space was provided to mark at which kilometer(s) a problem occurred.

The subjects were met at the finishing line by a research assistant and led to a temporary laboratory 50 m away. Blood samples were taken (20 ml) and body mass was measured as quickly as possible. On the average, blood samples were taken 3-5 min after finishing the race.

Subjects. The experimental group included 158 men and 12 women. Mean finishing time for the men was 8 h 18 min and 8 h 56 min for the women. Included among the subjects were the first woman (6 h 45 min) and fifth man to finish (5 h 41 min), as well as individuals who finished in just under 12 h. Ninety-one percent of the subjects had run a marathon (42.2 km) or a longer race previously and 58% had run the Swiss Alpine Marathon before. In general the subjects appeared to be experienced and well-trained, based upon previous race experience and the high percentage of finishers. The group surveyed can be considered representative of the total group of competitors, based upon finishing times and

![Fig. 1. Profile of the race course](attachment:image_url)
places. The mean age of the men was 40 years (range 20–72 years) and of the women 35 years (range 26–46 years). Body masses were obtained from 6 of the 12 women and 87 of the 158 men prior to the race and from 5 and 86 of these subjects, respectively, after the race. Mean pre-race masses were 60.7 (SEM 3.2) kg and 71.7 (SEM 8.9) kg for women and men, respectively.

Blood analyses. A blood sample was taken from a forearm vein and a portion of whole blood was analysed for packed cell volume (PCV) and haemoglobin (Hb) immediately afterwards. A 2-ml sample of blood was collected in separate tubes for GI hormone analyses. Plasma glucose, osmolality, sodium, potassium, chloride, the enzymes creatine phosphokinase (CK), aspartate aminotransferase (ASAT) and alanine aminotransferase (ALAT), the GI hormones vasoactive intestinal polypeptide (VIP), motilin, gastrin, peptide histidine isoleucine (PHI), as well as catecholamines and cortisol were measured. Details of sample preparation and analyses for GI hormones are published elsewhere (Riddoch 1990). For catecholamine analyses, gluthathione was added to the plasma. Plasma samples were kept on ice until being put into deep-freeze storage (−70°C) until further analyses could be conducted.

The Hb was determined by the met-Hb technique and PCV by the standard microcapillar centrifuge method. Plasma volume change was calculated using Hb and PCV values according to the formula of Dill and Costill (1974). Data has been presented as actual concentrations measured uncorrected for plasma volume changes. This has been done because we were interested in the specific effects of the concentration of a particular substance or metabolite and not in the production or disappearance rate of these substances. Only for the GI hormones did we also correct for plasma volume changes to evaluate the rate of production or disappearance.

Plasma osmolality was determined by the freeze-point method (Osmometer 830, Gonotec, Berlin, Germany). Plasma sodium and potassium were determined by flame photometry (Instrumentation Laboratory 243, Lexington, Mass., USA). Catecholamines were analysed with high performance (pressure) liquid chromatography (Smedes et al. 1982). The CK, ASAT, and ALAT were determined using commercial enzyme kits (Boehringer, Mannheim, Germany). Plasma glucose was determined by the GOD-perid method (Boehringer, Mannheim, FRG). Cortisol was determined by radio-immunoassay (Diagnostic Products, Los Angeles, Calif., USA).

Statistical analyses. Non-parametric analyses were used since the data was not normally distributed. Unless otherwise stated, all results are presented as mean and SEM. The Mann-Whitney analysis was applied to variables where a difference in subjects with GI symptoms compared to those without was expected. A similar analysis was also completed for each specific GI disorder, i.e. nausea, vomiting, diarrhoea, etc., in comparing postrace and prerace values and the differences (post-race minus pre-race) in the sufferers compared to non-sufferers, as well as the prevalence of such complaints in men compared to women. To compare pre- and post-race values the Wilcoxon's signed rank test for paired data was used. Simple linear regression was performed when looking for a correlation between variables with a range of values. A 95% confidence interval was defined in testing for significant differences between means.

Results

Body mass changes

Body mass losses of 2.4 (SEM 0.1) kg (3.3% of body mass) in men and 2.3 (SEM 0.2) kg (4.0%) in women were measured. The average loss of mass, including both men and women, was less than that which had been observed in the two previous years' competitions (1986, 5.5%; 1987, 6.6%; compared to 1988, 3.4%).

Intakes

All subjects drank during the course of the race. During the competition fluid intake was 3335 (SEM 107) ml in men and 2746 (SEM 338) ml in women. Water was the most commonly consumed beverage. From the 3290 ml consumed (combined average men and women) 1723 ml was water, 479 ml was a beverage containing 18% carbohydrate [312 kJ·100 ml⁻¹; 380 mosmol·kg⁻¹ (Perform)] and 569 ml was a beverage containing 7% carbohydrate [138 kJ·100 ml⁻¹; 298 mosmol·kg⁻¹ (Isostar)]. Based on the average intakes of these two beverages, mean carbohydrate intake was at least 129 g. A wide variety of other beverages provided by the athletes themselves was consumed. The intake of all these other beverages combined averaged 518 ml. No estimation of the additional carbohydrate contribution from these drinks was made since they were frequently home-made and no quantitative or qualitative estimation of the ingredients was possible. Solid foodstuffs, mostly pieces of fruit, were sporadically consumed by 85% of the subjects.

Gastro-intestinal symptoms

Gastro-intestinal distress was reported by 43% of the runners surveyed. One or more GI symptom was reported by 42% of the men and 57% of the women. Diarrhoea was more common among the women and there was a trend for the women to have intestinal cramps more frequently than the men. The frequency of specific disorders is shown in Table 1.

Blood parameters

Increases in PCV and Hb concentration were observed (Table 2) which are in line with the moderate level of dehydration indicated by loss of body mass. Based upon PCV and Hb values before and after the race, the plasma volume decrease was calculated at 8.3%. Plasma

Table 1. Runners reporting GI problems

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>All</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>Nausea</td>
<td>25</td>
<td>14.7</td>
<td>22</td>
</tr>
<tr>
<td>Vomiting</td>
<td>4</td>
<td>2.4</td>
<td>4</td>
</tr>
<tr>
<td>Stomach ache</td>
<td>19</td>
<td>11.2</td>
<td>19</td>
</tr>
<tr>
<td>Side ache</td>
<td>32</td>
<td>18.8</td>
<td>27</td>
</tr>
<tr>
<td>Intestinal cramps</td>
<td>15</td>
<td>8.8</td>
<td>12</td>
</tr>
<tr>
<td>Diarrhoea*</td>
<td>10</td>
<td>5.9</td>
<td>7</td>
</tr>
</tbody>
</table>

* Significant difference between men and women (P<0.05)
Table 2. Blood parameters

<table>
<thead>
<tr>
<th></th>
<th>Prerace</th>
<th>Postrace</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td>SEM</td>
</tr>
<tr>
<td>Packed cell volume</td>
<td>0.444</td>
<td>0.003</td>
</tr>
<tr>
<td>Haemoglobin (mmol-l⁻¹)</td>
<td>9.2</td>
<td>0.1</td>
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<tr>
<td>Osmolality (mosmol·kg⁻¹)</td>
<td>305.3</td>
<td>0.8</td>
</tr>
<tr>
<td>Sodium (mmol·l⁻¹)</td>
<td>139.1</td>
<td>0.4</td>
</tr>
<tr>
<td>Potassium (mmol·l⁻¹)</td>
<td>4.24</td>
<td>0.03</td>
</tr>
<tr>
<td>Chloride (mmol·l⁻¹)</td>
<td>103.4</td>
<td>0.2</td>
</tr>
</tbody>
</table>

* Significant post-race increase (P<0.05)

Table 3. Gastro-intestinal hormones

<table>
<thead>
<tr>
<th></th>
<th>Prerace</th>
<th>Postrace</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td>SEM</td>
</tr>
<tr>
<td>Gastrin (ng·l⁻¹)</td>
<td>71.6</td>
<td>11.9</td>
</tr>
<tr>
<td>Vasactive intestinal polypeptide (ng·l⁻¹)</td>
<td>28.8</td>
<td>2.6</td>
</tr>
<tr>
<td>Motilin (ng·l⁻¹)</td>
<td>146.4</td>
<td>13.0</td>
</tr>
<tr>
<td>Peptide histidine isoleucine (ng·l⁻¹)</td>
<td>37.7</td>
<td>2.5</td>
</tr>
</tbody>
</table>

* Significant post-race increase (P<0.05)

![Fig. 2. Plasma skeletal muscle and liver enzyme activities. All post-race concentrations were significantly elevated (P<0.05). CK, creatine phosphokinase; ASAT, aspartate aminotransferase; ALAT, alanine aminotransferase](image)

![Fig. 3. Plasma adrenaline and noradrenaline concentrations. Post-race concentrations of both catecholamines were significantly increased (P<0.05)](image)

![Fig. 4. Plasma cortisol concentrations. Post-race concentration was significantly increased (P<0.05)](image)

Osmolality did not change significantly (305 mosmol·kg⁻¹ pre-race; 304 mosmol·kg⁻¹ post-race). Concentrations of sodium and chloride remained unchanged while plasma potassium concentration increased significantly (Table 2). The CK, ASAT and ALAT activities were all significantly increased immediately following the race compared with resting values. These changes are shown in Fig. 2.

Plasma glucose was not significantly altered after the race [5.7 (SEM 0.1) mmol·l⁻¹] compared with resting, pre-race values [5.2 (SEM 0.1) mmol·l⁻¹]. The GI hormone concentrations in plasma (uncorrected for plasma volume changes) are shown in Table 3. All GI hormones assayed were observed to be significantly increased after the race, even if corrected for plasma volume changes. The post-race concentrations of these hormones approached levels that at rest would be considered pathological. Post-race plasma adrenaline, noradrenaline (Fig. 3) and cortisol concentrations (Fig. 4) were significantly increased. Post-race plasma cortisol concentrations were significantly correlated with lower (faster) finishing times.
Correlation of parameters with GI symptoms

No correlation was observed between change of body mass and prevalence of GI distress. There was an apparent trend for mean plasma volume to be decreased to a greater degree in those with GI complaints [11 (SEM 3)%] than in those without [6 (SEM 2)%] but this was not significant. There was no correlation found between the amount of fluid ingested during the course of the race and GI symptoms. Also, no correlation was observed between the quantity of any one of the carbohydrate-containing sports drinks offered and the prevalence of GI complaints. Neither was there a direct correlation of total grams CHO ingested, from the two sources provided by the race organisers, with GI complaints observed. Since the composition of other beverages consumed which were not provided by the race organisers varied widely, no analysis of the effects of these beverages could be carried out. The increase in GI hormones in the plasma after the race (measured as the difference from the resting value) was not different in the sufferers compared to the non-sufferers.

There was a trend for post-race noradrenaline to be less in sufferers (2047 SEM 139) than in non-sufferers (2559 SEM 190) (P=0.08) and the difference from the resting value to be less in sufferers (P=0.06). Individuals with intestinal cramp had significantly lower pre-race cortisol (Fig. 5) as well as post-race noradrenaline (Fig. 6).

![Fig. 5. Cortisol concentrations in individuals with and without intestinal cramp. Pre-race concentration was lower in individuals with cramp (P<0.05). ■ With intestinal cramp; □ Without intestinal cramp](image)

![Fig. 6. Noradrenaline concentrations in individuals with and without intestinal cramp. Post-race concentration was lower in individuals with cramp (P<0.05). For explanation of the symbols compare Fig. 5](image)

Greater post-race plasma potassium concentration was also observed in the group which experienced GI distress. This difference was significant if individuals with "side-ache" were excluded (Fig. 7). No difference in plasma sodium or chloride changes was observed between sufferers and non-sufferers.

Discussion

The prevalence of GI disturbance among ultramarathon runners surveyed was similar to that reported among marathon runners (Keeffe et al. 1984; Rehrer et al. 1989). Similarly, the losses of body mass measured as a result of this 67-km foot-race were similar to those reported to result from marathon competition (Rehrer et al. 1989), despite the fact that the time to run this race was approximately double that taken to run a marathon. Two factors accounted for this discrepancy. One was that fluid intakes were greater during this endurance race, approximately 3 l, versus the 0.5 l which has been measured in marathon runners when losses of mass were similar (Rehrer et al. 1989). Another difference was that running speed was lower during the ultramarathon which would, under similar weather conditions, have resulted in decreased sweat rates. Overt hyperthermia was not observed; the (cool) weather and reduced running intensity probably also contributed to the maintenance of normal body temperature regulation.

Despite similar losses of mass, the relationship noted in marathon runners between loss of body mass (level of dehydration) and GI distress was not found in this group of ultramarathon runners. Other factors, which may or may not be related to fluid balance (e.g. plasma volume and blood flow changes) may also have influenced the prevalence of GI symptoms. Plasma sodium was not increased after the race, whereas plasma potassium was increased significantly. The difference might have been caused by several factors, among them glucose deprivation and increments of atrial natriuretic peptide (ANP).

If glucose is limited at a membrane or tissue level the ionic pump may be impaired, thus sodium collects intracellularly and potassium extracellularly from active muscle tissue where glycogen is depleted. Intracel-
lular reaccumulation of potassium may not keep pace with the potassium released through repolarisation. Since a 3–5 min delay occurred before blood sampling and since samples were taken from a peripheral vein, these electrolyte values may be lower than what would be found during or immediately after cessation of exercise at a sampling site close to the largest mass of exercising muscle. Return to normal plasma potassium values after exercise has been shown to occur very quickly after exercise is stopped: plasma potassium values ranging from 8–10 mmol·L⁻¹, have returned to pre-exercise values in 3 min (Sejersted and Medbo 1989). The quick recovery after the cessation of exercise is accounted for by an increased blood flow and uptake in inactive muscles which coincides with a decreased potassium leak when exercising muscles cease exercising. Thus, the plasma potassium concentrations measured in the present experiment may not have reflected the extent of the increase that may have been present during the race.

Several other explanations are possible for the significant increase in plasma potassium concentration observed in subjects with GI distress but not in those without. An increased utilization of liver and muscle glycogen in those individuals may have contributed to the plasma potassium increase since potassium is stored with glycogen (Fenn 1940). Also, skeletal muscle, liver, or red blood cell damage (Davidson 1969) may have contributed. In the present study, however, CK, ASAT, and ALAT activities, indicating tissue damage, were not found to be increased in those individuals with GI disturbance. Thus damage to skeletal muscle or liver is unlikely to be the cause. Further, increments in ANP after exercise have been reported by several authors (Bittner et al. 1986; Somers et al. 1986; Toft et al. 1990). This hormone has been shown to increase natriuresis (Genest and Cantin 1988) but not potassiumuresis.

The relationship between the presence of GI distress and the increased plasma potassium concentration remains unclear. This finding may have been related to the fact that potassium has been shown to have a concentration dependent effect on the circular and longitudinal muscles of the duodenum (Kimura et al. 1984). It is also possible that the increased plasma potassium concentrations observed in sufferers were not directly causing the GI distress. It may be that the metabolic milieu (e.g. glycogen store, training status, exercise intensity) in these individuals was such that both increased plasma potassium concentration and increased risk of GI disturbance occurred.

Carbohydrate consumption was rather low (approximately 124 g, based upon mean intakes of the two carbohydrate-containing drinks at designated aid stations). This may have contributed to a faster glycogen depletion in working muscles and a localized glucose shortage. Although plasma glucose concentrations were maintained this did not necessarily reflect the substrate availability at a cellular or tissue level.

The fact that in this study the dehydration level (loss of body mass) was not correlated with GI dysfunction may have reflected the difference in rehydration solutions consumed. In an early study of marathon runners where this relationship was observed (Rehrr et al. 1989), supplementation was almost exclusively with a beverage containing carbohydrate and electrolytes. In the present study more than half of the fluid consumed was plain water. Although it has been shown that dehydration alters GI physiology and may play a role in the development of GI distress, particularly when it results in hyperthermia, the influence may not be a direct one. As Hubbard et al. (1987) have outlined in the “energy depletion model for hyperthermia”, the effects observed may be brought about by insufficiency of the ionic pump and energy depletion at a cellular level. Ionic pumping [Na⁺/K⁺ in skeletal muscle and intestinal epithelia, and H⁺/K⁺ in gastric mucosal membrane (Schrijen 1981)] maintains the function and integrity of cells. It has been proposed that increased temperature increases the energy demand and increases the demand for ionic pumping. When ionic pumping cannot meet the demands it may further result in insufficient glucose transport and energy deficit whereby the rate of pumping is then further reduced. Alternatively, when available glucose is limited the rate of ionic pumping is also limited. This may explain why muscle fatigue has been found to be concomitant with an increased extracellular (plasma) potassium concentration (Sejersted and Medbo 1989; Sjøgaard 1989). As Costill (1977) has noted, during prolonged exercise, the ratio of intramuscular to extramuscular potassium concentration can be significantly altered, in which case changes in the muscle cell membrane may occur.

The trend for post-race noradrenaline values to be less in sufferers than in non-sufferers may have been a result rather than a cause of GI disorder. Runners experiencing these symptoms may have slowed their pace and thus noradrenaline concentrations later in the race may not have been as high as those who maintained running at a higher intensity since noradrenaline concentrations are known to be higher as exercise intensity increases (Galbo et al. 1977). There is also the possibility that these subjects were overtrained, which may have resulted in depression of the pituitary-adrenocortical system. It is also possible to speculate that the difference in GI symptoms relative to the catecholamine response may have been related to the fact that noradrenaline and adrenaline stimulate absorption of glucose and sodium in the intestine (Aulsebrook 1965), so that those with a normal catecholamine response were better able to utilise ingested fluids and carbohydrate.

The significance of the fact that the runners who suffered from intestinal cramp had lower resting plasma cortisol concentrations is uncertain and one can only speculate on the cause. Individuals with lower cortisol concentrations may have been less able to adapt to a high level of energy expenditure. Fat utilization as well as liver glycogenolysis and gluconeogenesis have been shown to be stimulated by cortisol (Galbo 1983) which have been found to potentiate catecholamine functions (Lamberts et al. 1975). The GI symptoms may have arisen in response to a reduction in available substrate to
GI tract tissues. Supporting this idea is the fact that strength and work capacity have been observed to be decreased with adreno-cortico-steroid insufficiency (Korge and Roosson 1975). In individuals with Addison’s disease abdominal pain and diarrhoea are common. These symptoms have been attributed to cortisol deficiency (Jensen 1976). The difference in cortisol concentrations at rest in our two groups may also reflect a difference in training status. Trained subjects, at the same relative exercise intensity, often have been found to have higher cortisol concentrations in plasma (Bloom et al. 1976). Training status has been pointed out as one of the factors influencing the prevalence of GI symptoms in runners, less well-trained runners having more problems (Brouns et al. 1987). On the other hand, it must also be considered that the subjects with low plasma cortisol concentrations before the race may have been over-trained. The over-training syndrome has been characterised by lower plasma cortisol concentrations and a decreased cortisol response to exercise, among other metabolic adaptations (Kuipers and Keizer 1988). Thus it may be that the lower pre-race cortisol concentrations in runners with intestinal cramps are less well-trained or over-trained, and that the cramp is not a direct consequence of the plasma cortisol concentrations. A reduction in hypothalamic-pituitary-adrenal axis stimulation may also be related to the high plasma potassium concentrations due to a reduction in aldosterone secretion.

When the finishing time was used as an indication of exercise intensity, there was no difference between those with and those without GI symptoms. Finishing time, however, is a measure of absolute intensity and it may be that relative intensity was different in the two groups.

Concentrations of all GI hormones measured increased after the race, as has been observed in 30 km (Sullivan et al. 1984) and marathon runners (Riddoch 1990). There was, however, no correlation between the increase in any of the GI hormones and the occurrence of GI symptoms. The increase in GI hormones may have been more metabolic, in response to the energy demands, rather than being specifically related to the alteration in GI function. For example, VIP is known to stimulate lipolysis (Frandsen and Moody 1973), glycogenolysis and gluconeogenesis (Matsumura et al. 1972). Supporting the idea that energy demands stimulate this increase is the fact that increases in plasma VIP concentrations have been observed during starvation and energy deficiency as well as during prolonged exercise (Oktealdean et al. 1983). Also supporting the metabolic role of VIP is the lack of change in plasma concentration found during short term moderate to intense physical exercise (60%-100% VO_2max) but the significant increase seen in long term exercise (Galbo et al. 1979). Although VIP is known to be a powerful secretagogue and is known to induce diarrhoea, no relationship between plasma concentrations of VIP and the prevalence of diarrhoea was observed. PHI also has been found to induce intestinal secretions and reduce net absorption of water and electrolytes, but its role in stimulating glycogenolysis and gluconeogenesis in the liver (observed in rats; Feliz and Marco 1983) may be related more to its increase during endurance exercise. Gastrin is involved in gastric acid secretion, via histamine. The concentrations of gastrin observed were at the same level as those observed in marathon runners following competition, as were the other hormones (Riddoch 1990). As has been noted by Riddoch (1990), this increase in gastrin concentration is similar to that found in the presence of gastrin-producing pancreatic tumours. Although adrenaline is known to stimulate gastrin secretion, Sullivan et al. (1984) have observed no relationship between the changes in circulating adrenaline concentration and changes in gastrin. Motilin initiates the interdigestive migrating motor complex of the stomach and intestine. How this increase in concentration during exercise is related to gastro-intestinal function is uncertain. Other factors which were not taken into consideration in this study, such as dopamine and endorphin concentrations, which are known to influence gastro-intestinal function, may also play a role in the development of GI symptoms. It has been found that these substances are elevated during extreme exercise (Bortz et al. 1981).

Plasma activities of the enzymes CK, ASAT, and ALAT were all significantly elevated after the race, indicative of tissue damage. The CK activities observed 3–5 min after finishing this ultramarathon were comparable to values found by Janssen (1988) 24 h after a marathon. It may be assumed that these plasma activities would have continued to increase for 24 h after the race (Apple et al. 1985). The high enzyme activities can be attributed to the large eccentric component of running downhill and the length of the race. Even running on a level surface includes an eccentric component, which, if carried on for long enough and at a high enough intensity, results in an increased in the plasma activity of these enzymes. Also, the steepest and largest amount of downhill running occurred after 50 km in the Davos race. It can be assumed that glycogen deposits in the dominant working muscle fibres were depleted by this time. This may have increased the amount of tissue damage occurring as a result of the continued (downhill) running (Thomson et al. 1975). The question remains whether greater carbohydrate supplementation during exercise can prevent or decrease the amount of tissue damage, possibly via maintenance of intramuscular/extramuscular electrolyte balance and muscle cell membrane integrity. Furthermore, it is questioned whether the relationship observed between higher post-race plasma potassium concentration and GI distress reflected another consequence of cellular energy depletion and if endogenous carbohydrate supplementation could have reduced the prevalence of these symptoms.

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