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Kristin J. Stuempfle
Gettysburg College

Martin D. Hoffman

Tamara Hew-Butler

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Abstract

Context: Gastrointestinal (GI) distress is common during ultrarunning.

Purpose: To determine if race diet is related to GI distress in a 161-km ultramarathon.

Methods: Fifteen (10 male, 5 female) consenting runners in the Javelina Jundred (6.5 loops on a desert trail) participated. Body mass was measured immediately pre-race and after each loop. Runners reported if they had nausea, vomiting, abdominal cramps, and/or diarrhea after each loop. Subjects were interviewed after each loop to record food, fluid, and electrolyte consumption. Race diets were analyzed using Nutritionist Pro.

Results: Nine (8 male, 1 female) of 15 runners experienced GI distress including nausea (89%), abdominal cramps (44%), diarrhea (44%), and vomiting (22%). Fluid consumption rate was higher ($p = .001$) in runners without GI distress ($10.9 \pm 3.2 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$) than in those with GI distress ($5.9 \pm 1.6 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$). Runners without GI distress consumed a higher percentage fat ($p = .03$) than runners with GI distress (16.5 ± 2.6 vs. 11.1 ± 5.0). In addition, fat intake rate was higher ($p = .01$) in runners without GI distress ($0.06 \pm 0.03 \text{ g} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$) than in runners with GI distress ($0.03 \pm 0.01 \text{ g} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$). Lower fluid and fat intake rates were evident in those developing GI distress before the onset of symptoms.

Conclusions: A race diet with higher percentage fat and higher intake rates of fat and fluid may protect ultramarathoners from GI distress. However, these associations do not indicate cause and effect, and factors other than race diet may have contributed to GI distress.

Keywords

gastrointestinal distress, marathon, race, diet, athlete, ultramarathon, running, hydration

Disciplines

Other Medicine and Health Sciences | Sports Sciences

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Kristin J. Stuempfle, Martin D. Hoffman, and Tamara Hew-Butler

Context: Gastrointestinal (GI) distress is common during ultrarunning. **Purpose:** To determine if race diet is related to GI distress in a 161-km ultramarathon. **Methods:** Fifteen (10 male, 5 female) consenting runners in the Javelina Jundred (6.5 loops on a desert trail) participated. Body mass was measured immediately pre-race and after each loop. Runners reported if they had nausea, vomiting, abdominal cramps, and/or diarrhea after each loop. Subjects were interviewed after each loop to record food, fluid, and electrolyte consumption. Race diets were analyzed using Nutritionist Pro. **Results:** Nine (8 male, 1 female) of 15 runners experienced GI distress including nausea (89%), abdominal cramps (44%), diarrhea (44%), and vomiting (22%). Fluid consumption rate was higher ($p = .001$) in runners without GI distress ($10.9 \pm 3.2 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$) than in those with GI distress ($5.9 \pm 1.6 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$). Runners without GI distress consumed a higher percentage fat ($p = .03$) than runners with GI distress (16.5 ± 2.6 vs. 11.1 ± 5.0). In addition, fat intake rate was higher ($p = .01$) in runners without GI distress ($0.06 \pm 0.03 \text{ g} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$) than in runners with GI distress ($0.03 \pm 0.01 \text{ g} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$). Lower fluid and fat intake rates were evident in those developing GI distress before the onset of symptoms. **Conclusions:** A race diet with higher percentage fat and higher intake rates of fat and fluid may protect ultramarathoners from GI distress. However, these associations do not indicate cause and effect, and factors other than race diet may have contributed to GI distress.

Keywords: ultrarunning, nausea, vomiting, hydration

Gastrointestinal (GI) distress is a pervasive problem in ultradistance running (Baska, Moses, Graeber, & Kearney, 1990; Glace, Murphy, & McHugh, 2002; Hoffman & Fogard, 2011; Rehrer, Brouns, et al., 1992). Nausea, vomiting, abdominal cramping, and diarrhea have been reported in 37–47% of runners participating in races 67–161 km in length (Baska et al., 1990; Glace et al., 2002; Hoffman & Fogard, 2011; Rehrer, Brouns, et al., 1992), and fecal blood loss indicating GI hemorrhage was reported in 85% of participants in a 161-km ultramarathon (Baska et al., 1990). In two 161-km ultramarathons, nausea and/or vomiting were the main reasons for dropping out among nonfinishers and were the second most common problem affecting race performance among finishers (Hoffman & Fogard, 2011).

The pathophysiology of GI dysfunction in ultrarunners is not fully understood and is likely multifactorial. The mechanical pounding and jostling associated with running is widely cited as a direct cause of GI distress (de Oliveira & Burinin, 2009; Gil, Yazaki, & Evans, 1998; Simons & Kennedy, 2004; Simons & Shaskan, 2005). This mechanical theory is supported by the observation

that GI problems are more common in running events than in cycling or swimming (de Oliveira & Burinin, 2009; Peters et al., 1999). Furthermore, GI symptoms are most common during the running part of triathlon competitions (Rehrer, van Kemenade, Meester, Brouns, & Saris, 1992).

A reduction in splanchnic blood flow likely contributes to GI distress in ultrarunners, as well (de Oliveira & Burinin, 2009; Gil et al., 1998). Reduced GI blood flow results in decreased gastric emptying and intestinal absorption, which can cause GI symptoms (de Oliveira & Burinin, 2009; Gil et al., 1998; Gisolfi, 2000). As exercise duration or intensity increases, splanchnic blood flow further decreases and GI symptoms increase (Gil et al., 1998; Peters et al., 1999; Pfeiffer et al., 2012). Dehydration reduces blood volume, aggravates decreased splanchnic blood flow, and increases GI symptoms (de Oliveira & Burinin, 2009; Gil et al., 1998).

Total energy expenditure during a 161-km ultramarathon has been reported to range from 13,000 to 16,000 kcal (Cuddy, Slivka, Hailes, Dumke, & Ruby, 2009; Dumke, Shooter, Lind, & Nieman, 2006). Ingestion of fluid and fuel during a race of this duration is important to offset such large expenditures. Low fluid intake can lead to dehydration, and it has been reported that dehydrated marathon runners were more likely to suffer from GI distress (Rehrer, Janssen, Brouns, & Saris, 1989). In contrast, overconsumption of fluid may result in nausea associated with exercise-associated hyponatremia (Hew-Butler et al., 2008). Low consumption rates

Stuempfle is with the Health Sciences Dept., Gettysburg College, Gettysburg, PA. Hoffman is with the Dept. of Veterans Affairs, Northern California Health Care System, Sacramento, CA. Hew-Butler is with the Exercise Science Program, Oakland University, Rochester, MI.

of carbohydrate may result in hypoglycemia and nausea (Eberle, 2007). In contrast, concentrated carbohydrate sources can delay gastric emptying (Gisolfi, 2000), resulting in nausea and vomiting, and can cause fluid shifts into the intestines, resulting in abdominal cramping and diarrhea (Simons & Shaskan, 2005). Thus, there may be an optimal balance in fluid and fuel consumption for prevention of GI symptoms.

Although fluid and fuel ingestion is important during ultrarunning, very little field research has systematically evaluated race diet with respect to GI distress. Therefore, the purpose of the current study was to assess fluid and fuel intake during a 161-km ultramarathon and ascertain whether intake is associated with the development of GI symptoms.

Methods

This study was set at the Javelina Jundred 100 Mile Endurance Run in McDowell Mountain Regional Park near Fountain Hills, AZ. The 161-km run comprises six and a half ~25-km loops on a rolling desert trail with altitudes ranging from 549 m to 732 m. Ambient temperature during the race ranged from 15 °C to 28 °C. Aid stations on the course were stocked with various foods, fluids, and electrolyte capsules. Runners were permitted to have their own supplies at the aid stations.

Institutional review board approval was granted for this study, and all subjects signed an informed-consent document. Subjects were recruited via e-mail before the race. Body mass was measured immediately prerace and at the completion of each loop on a WW42D impedance scale (Weight Watchers, New York, NY; resolution 0.1 kg). Subjects were wearing running shoes and running attire and were not holding or wearing other items while being weighed.

A multipronged approach with built-in redundancy maximized accurate accounting of food, fluid, and electrolyte capsule intake:

1. Approximately 1 week prerace, subjects submitted via e-mail a proposed plan of the food, fluid, and electrolyte capsules they intended to consume during the race (race start to race finish). They were interviewed by a designated investigator about their submitted plan during race registration to clarify any questions the investigator had about their proposed plan and to familiarize the subjects with the detail that would be expected regarding brand, flavor, and amount of each consumed item. Volumes of bottles and hydration pack bladders were established. Food and fluid consumed before race start were not collected in this study.
2. After each loop, body mass was measured, subjects were interviewed by the same designated investigator (food, fluid, and electrolyte capsule intake; presence of nausea, vomiting, abdominal cramps, and/or diarrhea), and food wrappers were collected. Each time the runners completed a loop, they were

asked to recall any items of food they had eaten, fluids they had drunk, or electrolyte capsules they had consumed since the last observation point. The designated investigator recorded the type and quantity of food, fluid, and electrolyte capsules consumed (including brand and flavor).

3. Approximately 1 week postrace, the same designated investigator sent the subjects via e-mail the results of their individual interviews that were conducted after each loop, asking them to review the recorded consumption of food, fluid, and electrolyte capsules for completeness and accuracy and to make any corrections.

Nutritionist Pro (Axxya Systems, Stafford, TX) software was used to analyze the nutritional composition of the foods, fluids, and electrolyte capsules consumed. Nutritional information for items not included in Nutritionist Pro was obtained from the manufacturer and added to the software database. Diets were analyzed for kilocalorie, carbohydrate, fat, protein, and fluid intake.

Comparisons between runners with and without GI distress were analyzed as intake per kilogram per hour, thus normalizing for body mass and rate of intake. To evaluate changes in intake over time, the data also were analyzed by race loop through Loop 4 (before any subjects dropped out).

Analyses of categorical variables were made with Fisher's exact test. Continuous data sets were confirmed for normality using the Kolmogorov-Smirnov normality test and subsequently were compared using unpaired or paired *t* tests. Percentage change in body mass from the start through Loop 4 (before any subjects dropped out) was compared between groups across loops with a two-way (Group × Loop) repeated-measures analysis of variance (ANOVA). Follow-up analyses included one-way repeated-measures ANOVA and Scheffé's post hoc tests. Statistical significance was set at $p < .05$.

Results

Finish rate for all racers was 50% (250 starters, 125 finishers), and finish rate for our subjects was 47% (15 starters, 7 finishers). Nine of 15 subjects experienced GI distress (60%; 8 men, 1 women; 4 finishers, 5 nonfinishers) including nausea (89%), abdominal cramps (44%), diarrhea (44%), and vomiting (22%). GI distress typically started during Loop 3 (44%; km ~50–75) or Loop 4 (33%; km ~75–100). Six runners did not experience GI distress (40%; 2 men, 4 women; 3 finishers, 3 nonfinishers). GI-distress incidence was similar between nonfinishers ($n = 8$) and finishers ($n = 7$). Men (8 of 10; 80%) appeared to experience more GI distress than women (1 of 5; 20%).

Runners with and without GI distress were similar in age, running experience, distance completed, running duration, and pace in the current race (Table 1). Starting body mass was similar in runners with and without GI distress (Table 2). Runners with GI distress lost significant body mass during the race (-1.6 ± 1.9 kg), while

Table 1 Demographic Comparison of Runners With and Without Gastrointestinal (GI) Distress

Variable	Without GI Distress		With GI Distress	
	<i>M</i> ± <i>SD</i>	Median (range)	<i>M</i> ± <i>SD</i>	Median (range)
Age, years	42.2 ± 11.1	44.0 (26.0–52.0)	49.9 ± 11.2	47.0 (37.0–67.0)
Years running	10.7 ± 8.0	8.0 (3.0–21.0)	19.6 ± 14.6	15.0 (6.0–43.0)
Previous ultramarathons completed	4.7 ± 2.9	4.5 (2.0–10.0)	25.1 ± 36.2	7.0 (3.0–113.0)
Previous 161-km races completed	1.3 ± 2.0	0.5 (0.0–5.0)	4.8 ± 7.9	1.0 (0.0–24.0)
Distance completed during race, km	137.7 ± 30.2	153.9 (99.2–161.0)	128.7 ± 30.7	123.2 (98.6–161.0)
Running duration during race, hr	22.5 ± 5.4	22.1 (17.0–28.5)	22.9 ± 5.2	23.2 (16.2–29.7)
Pace during race, km/hr	6.2 ± 1.2	5.7 (5.6–8.7)	5.6 ± 0.4	5.6 (5.2–6.1)

Table 2 Body Mass in Runners With and Without Gastrointestinal (GI) Distress, *M* ± *SD*

Variable	Without GI distress	With GI distress	Between-groups <i>p</i>
Start mass, kg	72.5 ± 18.3	78.3 ± 13.6	.49
End mass, kg	71.9 ± 17.8	76.7 ± 12.4	.54
Change in mass, kg	−0.6 ± 0.9	−1.6 ± 1.9*	.26
% mass change	−0.8 ± 1.1	−1.9 ± 2.1	.26

**p* = .03 prerace to postrace.

those without GI distress essentially maintained body mass (−0.6 ± 0.9 kg). Overall percent body-mass change did not differ between the two groups.

Figure 1 shows percent body-mass change across race loops in runners with and without GI distress. Two-way repeated-measures ANOVA showed a statistically significant main effect of loop on percent body-mass change in the analysis of start through Loop 4 (*p* < .0001). One-way repeated-measure ANOVA for runners with GI distress showed a statistically significant effect of loop (*p* < .0001), and Scheffé's post hoc tests revealed significant decreases in body mass between the start and the ends of Loops 2 (*p* = .001), 3 (*p* < .0001), and 4 (*p* = .01), as well as between Loops 1 and 3 (*p* = .01). One-way repeated-measure ANOVA for runners without GI distress did not reveal a statistically significant effect of loop on percent body-mass change (*p* = .28).

Runners consumed a variety of ordinary foods (soup, sandwiches, pizza, burritos, fruit, pretzels, chips, candy, potatoes, cookies, etc.) and foods marketed specifically to athletes (energy bars, energy gels, etc.). Fluids consumed included water, assorted sports drinks, soda, and coffee. Consumption of electrolyte capsules was prevalent (87%).

Table 3 summarizes overall race diet for runners with and without GI distress. Runners without GI distress consumed fluid (including water, sports drinks, soda, and coffee) at a higher rate (10.9 ± 3.2 ml · kg^{−1} · hr^{−1}) than runners with GI distress (5.9 ± 1.6 ml · kg^{−1} · hr^{−1}). Runners without GI distress consumed a race diet with higher percentage fat (16.5 ± 2.6) and consumed fat at a higher rate (0.06 ± 0.03 g · kg^{−1} · hr^{−1}) than runners who developed GI distress (% fat 11.1 ± 5.0; fat g · kg^{−1} · hr^{−1} 0.03 ± 0.01). Consumption rates of kilocalories, carbohydrate, and protein and percent of carbohydrate and protein in the race diet did not differ between runners with and without GI distress.

Table 4 summarizes race diet by loop (Loops 1–4, before any runners dropped out) in runners with and without GI distress. Runners without GI distress consumed fluid (including water, sports drinks, soda, and coffee) at a higher rate during Loops 1, 2, 3, and 4 than runners with GI distress. During Loop 2, runners without GI distress consumed a higher percentage fat (18.9 ± 4.2%) in the race diet and consumed fat at a higher rate (0.08 ± 0.04 g · kg^{−1} · hr^{−1}) than runners with GI distress (11.6 ± 5.5% fat, 0.04 ± 0.02 fat g · kg^{−1} · hr^{−1}).

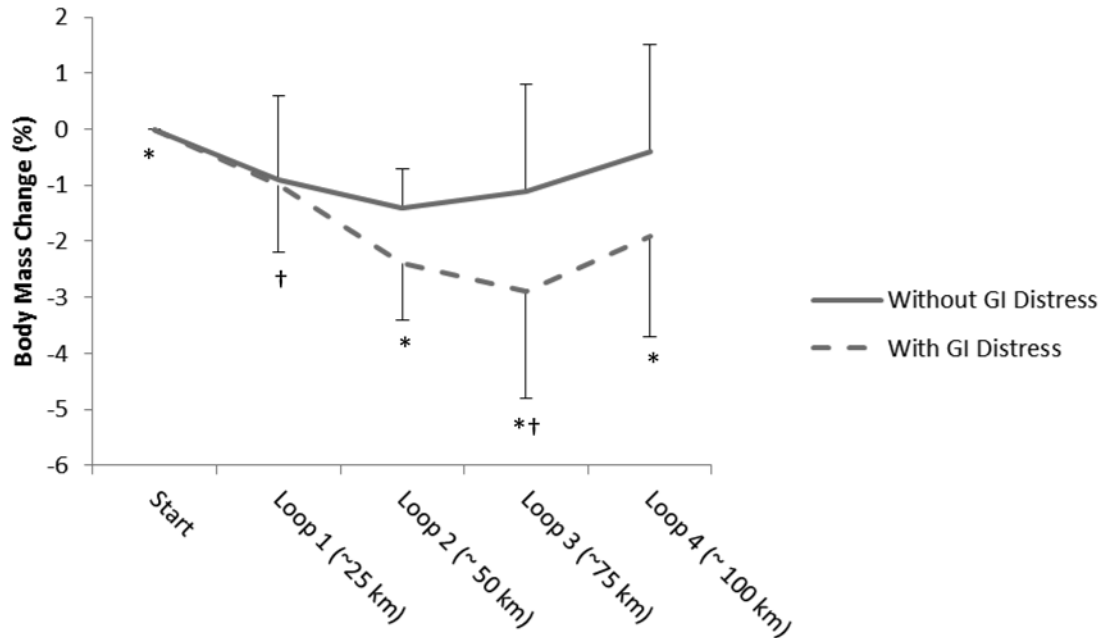


Figure 1 — Body-mass change (%) from prerace values by loop in runners with and without gastrointestinal (GI) distress. Significant differences ($p < .05$) were present for runners with GI distress between the start and Loops 2, 3, and 4 (*) and between Loops 1 and 3 (†). Brackets represent 1 SD.

Table 3 Overall Race Diet in Runners With and Without Gastrointestinal (GI) Distress, $M \pm SD$

Variable	Without GI distress	With GI distress	p
Energy, $\text{kcal} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$	3.4 ± 1.7	2.3 ± 0.9	.11
% carbohydrate	74.2 ± 5.2	79.5 ± 7.9	.17
Carbohydrate, $\text{g} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$	0.65 ± 0.38	0.45 ± 0.21	.22
% fat	16.5 ± 2.6	11.1 ± 5.0	.03
Fat, $\text{g} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$	0.06 ± 0.03	0.03 ± 0.01	.01
% protein	9.3 ± 3.1	9.3 ± 4.8	.99
Protein, $\text{g} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$	0.07 ± 0.02	0.05 ± 0.02	.13
Fluid, ^a $\text{ml} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$	10.9 ± 3.2	5.9 ± 1.6	.001

^aIncludes water, sports drinks, soda, and coffee.

Discussion

This study confirms that GI distress is common in ultra-running. The 60% incidence rate in this study is higher than a report from a 67-km run (43%; Rehrer, Brouns, et al., 1992) and within the range of other 161-km ultramarathons (37–75%; Glace et al., 2002; Hoffman & Fogard, 2011; Stuempfle, Hoffman, Weschler, Rogers, & Hew-Butler, 2011). We found that nausea was by far the most common GI symptom (89%), followed by abdominal cramps (44%), diarrhea (44%), and vomiting (22%).

This pattern is identical to that reported for a 67-km run (Rehrer, Brouns, et al., 1992). In the current study, GI symptoms began for most runners between ~50 and 100 km. This agrees with the report from another 161-km run (Glace et al., 2002). The incidence of GI distress was similar among finishers (57%) and nonfinishers (63%) in the current study, which supports the findings of a study conducted at two 161-km runs (Hoffman & Fogard, 2011). Previous reports from marathons have suggested that GI distress is more common in women than men (Halvorsen, Lyng, Glomsaker, & Ritland, 1990;

Table 4 Race Diet by Loop in Runners With and Without Gastrointestinal (GI) Distress, $M \pm SD$

	Energy, kcal · kg ⁻¹ · hr ⁻¹	CHO, g · kg ⁻¹ · hr ⁻¹	% CHO	CHO, g · kg ⁻¹ · hr ⁻¹	% fat	Fat, g · kg ⁻¹ · hr ⁻¹	% protein	Protein, g · kg ⁻¹ · hr ⁻¹	Fluid, ^a ml · kg ⁻¹ · hr ⁻¹
Loop 1 (~25 km)									
without GI distress	2.9 ± 1.3	83.2 ± 5.8	0.58 ± 0.3	12.7 ± 5.3	0.05 ± 0.04	4.1 ± 1.8	0.03 ± 0.02	10.7 ± 5.3	
with GI distress	2.5 ± 0.9	85.7 ± 13.6	0.52 ± 0.2	8.1 ± 9.8	0.02 ± 0.03	6.2 ± 6.4	0.04 ± 0.05	6.0 ± 2.1	
<i>p</i>	.43	.69	.63	.32	.23	.45	.57	.03	
Loop 2 (~50 km)									
without GI distress	4.2 ± 2.4	73.3 ± 7.0	0.79 ± 0.53	18.9 ± 4.2	0.08 ± 0.04	7.8 ± 4.2	0.06 ± 0.02	14.0 ± 5.6	
with GI distress	2.9 ± 1.1	81.8 ± 10.6	0.58 ± 0.27	11.6 ± 5.5	0.04 ± 0.02	6.7 ± 6.8	0.04 ± 0.03	7.40 ± 2.0	
<i>p</i>	.18	.11	.34	.02	.01	.74	.22	.01	
Loop 3 (~75 km)									
without GI distress	3.6 ± 1.3	85.2 ± 4.9	0.75 ± 0.30	10.3 ± 2.7	0.04 ± 0.02	4.6 ± 3.4	0.04 ± 0.02	15.44 ± 3.78	
with GI distress	2.9 ± 1.4	83.0 ± 8.5	0.61 ± 0.31	9.9 ± 7.3	0.04 ± 0.03	7.1 ± 5.2	0.05 ± 0.03	8.62 ± 3.04	
<i>p</i>	.40	.58	.40	.92	.67	.32	.40	.002	
Loop 4 (~100 km)									
without GI distress	3.7 ± 1.9	72.2 ± 4.1	0.67 ± 0.41	17.0 ± 4.2	0.06 ± 0.03	10.9 ± 2.0	0.10 ± 0.05	12.2 ± 2.51	
with GI distress	2.0 ± 1.5	62.2 ± 17.9	0.34 ± 0.32	18.7 ± 10.5	0.04 ± 0.03	19.2 ± 11.5	0.08 ± 0.05	4.74 ± 2.38	
<i>p</i>	.07	.21	.10	.72	.07	.11	.44	<.0001	

Note. CHO = carbohydrate.

^aIncludes water, sports drinks, soda, and coffee.

Keeffe, Lowe, Goss, & Wayne, 1984; Riddoch & Trinick, 1988), although the reason for this difference is unknown (Halvorsen et al., 1990). In contrast, the incidence of GI distress appeared to be lower in women (1 of 5, 20%) than in men (8 of 10, 80%) in the current study. Due to the low number of women, it is difficult to determine the reasons for this difference, but hormonal influences, race diet, or a slower pace may have been contributing factors.

In this study, runners with GI distress completed 128.7 ± 30.7 km in 22.9 ± 5.2 hr with an average pace of 5.6 ± 0.4 km/hr, whereas runners without GI distress completed 137.7 ± 30.2 km in 22.5 ± 5.4 hr with an average pace of 6.2 ± 1.2 km/hr (Table 1). These low to moderate running paces are similar to reports from other 161-km runs (5.7 – 6.6 km/hr; Glace et al., 2002; Stuempfle et al., 2011). Running at low or moderate intensity (~ 25 – 70% VO_{2max}) has been reported to not change (Carrio et al., 1989) or to increase gastric emptying (Neufer et al., 1986; Neufer, Young, & Sawka, 1989), whereas high-intensity running ($>75\%$ VO_{2max}) decreases gastric emptying (Leiper, Nicholas, Ali, Williams, & Maughan, 2005; Neufer et al., 1989). Therefore, it is unlikely that decreased gastric emptying due to high-intensity exercise contributed to the nausea reported in the current study, as the intensity of effort in our cohort was modest over a prolonged period of time.

Race diet appears to be an important factor in the development of GI distress. The overall fluid (including water, sports drinks, soda, and coffee) consumption rate in runners without GI symptoms was almost double that of runners with GI symptoms (Table 3), and this difference was apparent from race start. Runners who never developed GI symptoms consumed fluid at significantly higher rates in Loops 1, 2, 3, and 4 (Table 4). For the runners who developed GI symptoms, the symptoms primarily started during Loops 3 and 4. This suggests that early fluid consumption may help prevent GI distress later in the race. These findings support a report showing a trend for greater GI distress in triathletes with lower fluid consumption than that of asymptomatic triathletes (Rehrer, van Kemenade, et al., 1992). Furthermore, in a case-controlled study, an elite ultraendurance athlete with a history of nausea prevented that symptom by increasing his fluid intake (Bowen, Adams, & Myburgh, 2006).

Previous reports have indicated that dehydrated runners were more likely to experience GI distress (Rehrer, Beckers, Brouns, Hoor, & Saris, 1990; Rehrer et al., 1989). In the current study, overall percent body-mass change was similar in runners with and without GI distress (Table 2). However, when percent body-mass change was analyzed by loop, only the GI-distress group showed a significant change from the start through Loop 4. It is notable that body mass decreased among those developing GI distress before the onset of the distress. This finding coincides with our determination that fluid intake rates were lower in Loops 1–4 among those developing GI distress. Taken together, these findings suggest that runners who developed GI distress were not drinking fluid at a high enough rate to maintain their body mass.

Runners in this study without GI distress also consumed an overall race diet with higher percentage fat and consumed fat at a higher rate than did symptomatic runners (Table 3). Loop analysis revealed that this greater percentage fat consumption and intake rate was present in Loop 2 (Table 4), before those becoming symptomatic started developing GI distress during Loops 3 and 4. Compared with all-carbohydrate foods, foods that contain fat and/or protein slow gastric emptying, digestion, and absorption (Gisolfi, 2000). Furthermore, although exercise decreases splanchnic blood flow, eating during exercise helps maintain it (Qamar & Read, 1987). Therefore, the ingestion of food during an ultramarathon may attenuate the decreased splanchnic blood flow induced by exercise and help prevent GI symptoms (Gil et al., 1998).

We found the overall percentage of carbohydrate in the race diet and carbohydrate intake rates to be comparable in runners with and without GI distress (Tables 3 and 4). These findings are similar to those of another 161-km ultramarathon (Glace et al., 2002). In addition, a study with various endurance athletes showed no relationship between carbohydrate intake and GI distress (Pfeiffer et al., 2012).

Conclusion

Runners with GI distress consumed fluid (including water, sports drinks, soda, and coffee) and fat at lower rates than asymptomatic runners, and these lower intake rates were evident before GI symptoms developed. These findings suggest that fluid and fat consumption may protect ultramarathoners from GI distress. However, it should be emphasized that this study reveals associations between race diet and GI symptoms and does not indicate cause and effect. Factors beyond those examined in this study may have contributed to the development of GI distress.

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