ORIGINAL RESEARCH

Impact of race pace on development of hyponatraemia in full- and half-marathoners*

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Abstract

Objective. Prior studies of full-marathon participants have demonstrated a higher incidence of hyponatraemia in runners with completion times of 4 hours or more. Our primary aim was to determine if slower pace is associated with increased prevalence of hyponatraemia. Secondly, we evaluated the prevalence of hyponatraemia in full-marathoners v. half-marathoners.

Methods. This observational, cross-sectional study comprised consenting runners in the 26.2 With Donna, The National Marathon to Finish Breast Cancer, in Jacksonville Beach, Florida, February 2008. On race day, participants completed a questionnaire, provided finger-stick blood samples, and were weighed both pre- and post-race.

Results. A significant negative association was found between pace and post-race sodium level ($p<0.001$). A negative correlation was found between finishing time and post-race sodium level ($p<0.001$). The prevalence of post-race hyponatraemia was 4% (4/106) among half-marathoners and 13% (12/89) among full-marathoners ($P=0.02$). An inverse correlation was found between sodium change and weight change, significant in full-marathoners ($r=-0.55$, $p<0.001$) but not half-marathoners ($r=-0.23$, $p=0.042$).

Conclusions. Slower race pace and longer finishing times were associated with lower post-race sodium levels. Full-marathoners had a significantly higher prevalence of hyponatraemia. The development of hyponatraemia was associated with weight gain. Our data indicate that the relationship between post-race sodium concentration and pace differs according to the distance of the event. We can extrapolate from this data that longer race distance with increased availability of fluid stations combined with a slower pace may increase the risk of developing exercise-induced hyponatraemia.

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Introduction

Hydration status and its role in the performance of endurance athletes remains a popular topic of debate in sports medicine. Newer recommendations, including the 2007 American College of Sports Medicine Position Stand on Exercise and Fluid Replacement, warn athletes not to lose >2% body weight during exercise as it may adversely affect performance.[1] Although the same paper recommends drinking to thirst, the unfounded fear of dehydration and/or heat illness may have prompted athletes in the USA to continue to follow protocols that promote overzealous hydration. However, more recent analysis has shown that the fastest runners (therefore highest performers)

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actually are those who lose the most weight in marathons.[2] The recognition of the potential dangers of excessive fluid consumption has initiated multiple revisions of these theories. Current hydration strategies may be based on individual sweat rate, as monitored by body weight change during exercise, but most importantly should be gauged by thirst to maximise performance.[1,3-7]

Many studies and review papers have documented the risk of overhydration during prolonged endurance events, which may result in hyponatraemia (serum sodium concentration <135 mmol/l).[1,5,8-21] The occurrence of hyponatraemia during or up to 24 hours after prolonged physical activity is known as exercise-associated hyponatraemia (EAH).[8,12] A large study from Noakes *et al.* reveals that the development of EAH occurs from three main factors: overconsumption of fluid during exercise, retention of fluid due to inadequate suppression of antidiuretic hormone (ADH), and inactivation of or failure to reactivate internal stores of sodium.[22] The confluence of these factors enhances a dilutional state and decreases the serum sodium concentration. Signs and symptoms of hyponatraemia include nausea, vomiting, confusion, and headache. As EAH progresses, more severe sequelae include seizures, pulmonary and cerebral oedema (hyponatraemic encephalophathy), and possibly death. Since its first report in a 26.2 mile (42 km) race in the 1986 Pittsburgh Marathon, EAH has been cited in multiple hospitalisations and at least 5 known deaths of marathon participants.[9,18,19] Developing science and recognition of EAH have linked multiple risk factors, including excessive drinking behaviours, female sex, failure to appropriately suppress ADH in the presence of fluid retention, lower body mass index (BMI), slower running pace, non-elite status, and prolonged exercise (>4 hours).[1,9,10,12-14,20]

Our primary aim was to determine if slower pace is associated with increased prevalence of hyponatraemia. A secondary aim was to determine if there is a significant difference in the prevalence of hyponatraemia in half- v. full-marathon participants. This study has the advantage of analysing both pre- and post-race serum sodium concentrations, as well as the weight of participants before and after exercise to assess the changes incurred during an endurance event. Furthermore, we believe the current study is the first to examine the influence of race pace on the prevalence of hyponatraemia with the benefit of data from both half- and full-marathons. With these data, we propose that EAH is, in part, a behavioural disease in which athletes are influenced by tenuous information, the availability of fluid stations, and fear of dehydration.

Methods

Subjects

This observational, cross-sectional study comprised consenting runners who participated in the first 26.2 With Donna, The National Marathon to Finish Breast Cancer Full and Half Marathons. Study design was approved by the Institutional Review Board and used written informed consent and a Health Insurance Portability and Accountability Act waiver. Race participants were approached at random during the Health Expo held on the 2 days before the race. Consenting subjects, at least 18 years of age, were identified by a brightly coloured sticker on their race bib number.

Race setting

The race took place on 17 February 2008 at Jacksonville Beach, Florida. At the start of the race, 08h30, the ambient temperature was 17.8°C (64°F) and humidity 88%. Sunny conditions prevailed at the end of the race, with an ambient temperature of 24.4°C (76°F) and 43% humidity. Hydration stations were available approximately every mile, with a sports drink (Powerade; Coca-Cola Company, Atlanta, Georgia) offered at every other station. A carbohydrate energy gel (GU Energy Gel; GU Energy Labs, Berkeley, California) was also available roughly every 3 - 5 miles after mile marker 10. Runners were provided race bags that had various advertisements, race paraphernalia, and local health magazines (www.HealthSourceMag.com). No specific instructions regarding hydration were given to race participants.

Procedures

Serum sodium concentration was measured the morning of the race and after race completion. Height was self-reported by the participants. Weight was measured on the same calibrated scale before and after the race. BMI (weight in kilograms divided by the square of height in metres) was calculated before and after the race. Runners were instructed to proceed to the Runners' Science research tent on completion of the race. They were allowed to consume fluid freely after the race while waiting for their blood to be drawn. Venous blood was collected via finger stick and tested on a tabletop analyser (Stat Profile Critical Care Xpress; Nova Biomedical, Waltham, Massachusetts). Before the marathon, set-up and implementation studies for linearity and precision were performed by the vendor to meet industry standards. In an effort to maintain impartial interpretation, the results were not reviewed at the time. Therefore, no opportunity existed to recommend for or against participation in the race on the basis of pre-race laboratory values.

Outcome measures

The primary outcome measure was the incidence of hyponatraemia (serum sodium concentration [Na^+] <135 mmol/l). Secondary outcome measures included mean changes observed and determination of any statistically significant changes in paired data sets from before and after the race. Completion times were recorded by official chip time. Race pace, reported in minutes per mile, was calculated by finish time divided by race distance (13.1 or 26.2 miles).

Sample size considerations

The target enrolment was 250 runners, based on an expected volunteer rate of 5% of the 5 000 race participants. After allowing for the loss of up to 20% of records due to inadequate blood samples, we estimated that the prevalence of electrolyte abnormalities would be estimable to within a 3 - 7% margin of error, depending on the true prevalence and assuming the use of a large-sample approximation to the 95% confidence interval. Therefore, the target sample size was expected to provide reasonable precision in the estimation of the pre- and post-race prevalence of electrolyte abnormalities.

Analysis

Continuous variables were summarised using median and range, and categorical variables were summarised using number and percentage. To evaluate the relationship between categorical variables, such as marathon distance (full v. half) and hyponatraemia status, we used the Fisher exact test. The Wilcoxon rank sum test was used to compare continuous measures, including race pace and change in serum sodium levels, between full- and half-marathoners. The Spearman correlation coefficient was used to assess the correlation between race pace or finishing time and other continuous variables. Linear regression was used to explore the relationship between post-race serum sodium level, race pace, and distance.

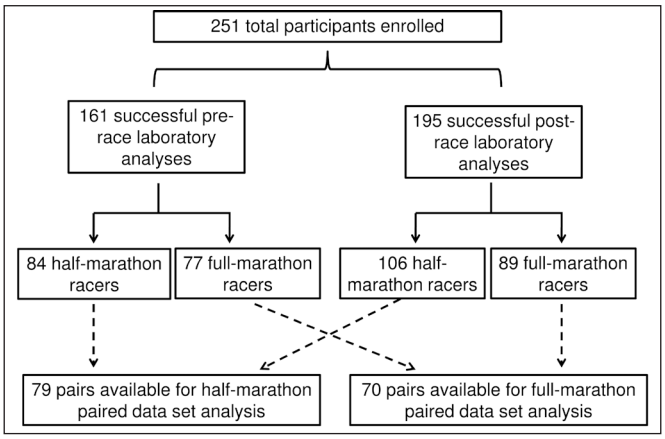

Fig. 1. Data flow in pre- and post-race settings in accordance with race distance.

Ethical considerations

Restriction of participation based on pre-race laboratory data was not possible because of the blood analysis processing method. Pre-race samples were stored on ice and transported to the laboratory at the main campus. Laboratory results were not available until after the race. If a symptomatic research participant presented to the race medical tent for treatment, laboratory values were available immediately for use by the treating physician. Study investigators were notified and collected a separate blood sample of research participants presenting to the medical tent.

Results

Total enrolment of 251 was completed early on the second day of the Health Expo. Although the target enrolment was met without difficulty, 18% (46/251) and 19% (47/251) of enrolled runners did not present for pre-race and post-race finger-stick sampling, respectively. Additional records were lost during the process of laboratory analysis. See Fig. 1 for data flow illustration.

A total of 161 and 195 records remained for pre-race and post-race analyses, respectively. The 39 pre-race records lost because the quantity of blood was insufficient were deemed likely secondary to chilly morning conditions and associated peripheral vasospasm. Notably, the number of post-race analyses with insufficient blood quantity was substantially lower at 7 records, presumably because blood samples were easier to obtain in the warmer digits of runners who just completed the race. There were slightly more half-marathon participants than full-marathon participants in both the pre- and post-race environments. Approximately equal numbers of pairs, however, were available for paired analysis (79 half-marathon v. 70 full-marathon pairs).

Table 1 summarises characteristics of the subjects, according to full- or half-marathon status. There was no statistical difference in age or sex by race type. Notably, there was a strong female participation rate of 71% (178/251). The overall race participants were similarly skewed toward a higher female component of 71% (3 950/5 536). Table 1 also includes the median changes observed in serum sodium levels between the pre-race and post-race environments. Interestingly, more half-marathoners (5/84 (6.0%)) than full-marathoners (3/77

Table 1. Characteristics of the Runners' Science Study Participants according to full- or half-marathon status*

Characteristic	All participants	Full-marathoners	Half-marathoners	*p*-value
Sex (male)	73/251 (29)	40/125 (32)	33/126 (26)	0.33
Age (years)	46 (20 - 71)	46 (20 - 70)	45 (24 - 71)	0.73
Finish time (min)	240 (90.6 - 398.7)	333.7 (194.4 - 398.7)	166.1 (90.6 - 358.6)	<0.001
Pace (min/mile)	12.7 (6.9 - 27.4)	12.7 (7.4 - 15.2)	12.7 (6.9 - 27.4)	0.47
Fluid ingested (cups)	16 (1.5 - 100)	27.5 (2 - 100)	12.0 (1.5 - 64.0)	<0.001
Fluid ingested (cups/min)	0.08 (0.008 - 0.34)	0.08 (0.008 - 0.34)	0.08 (0.01 - 0.33)	0.29
Hyponatraemia				
Pre-race	8/161 (5)	3/77 (4)	5/84 (6)	0.72
Post-race	16/195 (8)	12/89 (13)	4/106 (4)	0.02
Sodium concentration (mmol/l)				
Pre-race	138.7 (105.4 - 145.8)	139 (133.2 - 145.8)	138 (105.4 - 143)	0.16
Post-race	140 (125 - 157)	140 (125 - 157)	140 (125 - 151)	0.07
Change (post-pre)	2.0 (-11.0 - 35.6)	0.5 (-11.0 - 16.0)	2.0 (-6.7 - 35.6)	0.03
Body mass index, median (range), kg/m^2				
Pre-race	24.5 (18.1 - 42.2)	24.0 (18.2 - 33.2)	25.0 (18.1 - 42.2)	0.32
Post-race	24.0 (17.7 - 36.7)	23.6 (17.7 - 32.6)	24.5 (18.1 - 36.7)	0.28
Change (post-pre)	-0.4 (-1.8 - 0.5)	-0.6 (-1.8 - 0.2)	-0.3 (-0.8 - 0.5)	<0.001
Weight, median (range), kg				
Pre-race	68.5 (43.1 - 120.1)	67.9 (43.1 - 111.1)	68.6 (50.5 - 120.1)	0.69
Post-race	67.1 (41.0 - 117.5)	67.0 (41.0 - 108.3)	67.3 (50.1 - 117.5)	0.60
Change (post-pre)	-1.1 (-5.7 - 1.4)	-1.5 (-5.7 - 0.7)	-0.8 (-2.7 - 1.4)	<0.001

*Continuous variables are reported as median (range), with comparisons between full- and half-marathoners performed using the Wilcoxon rank sum test. Categorical variables are reported as fraction (percentage), with comparisons between full- and half-marathoners performed using the Fisher exact test.

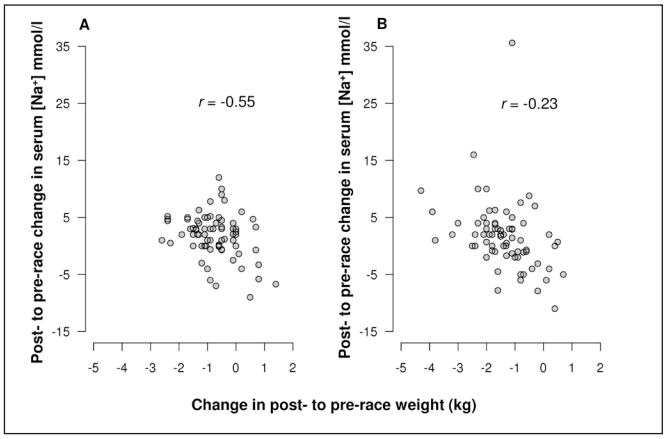

Fig. 2. Scatterplots of the change in weight (kg) v. change in serum sodium concentration (mmol/l) from post- to pre-race in (A) full- and (B) half-marathon participants.

(3.9%)) presented with hyponatraemia before the race, with 1 asymptomatic half-marathoner in a state of laboratory-classified severe hyponatraemia (serum sodium 105 mmol/l). However, this difference was not statistically significant (p=0.72). At completion of the race, 3 times as many full-marathoners as half-marathoners were hyponatraemic (12/89 (13.5%) and 4/106 (3.8%), respectively; p=0.02). There was a significant difference in the change in sodium levels between half- and full-marathon runners. The median increase was 2.0 mmol/l for half-marathon runners (range, -6.7 - 35.6 mmol/l) and 0.5 mmol/l for full-marathon runners (range, -11.0 - 16.0 mmol/l) (p=0.03, Wilcoxon rank sum test). Therefore, full-marathon runners experienced a smaller increase in sodium over the course of the race, and significantly more full-marathoners than half-marathoners were hyponatremic after the race (p=0.02).

Data of the change in weight (kg) and calculated in BMI (kg/m^2) among runners indicated that full-marathoners lost significantly more weight during the race (-1.5 kg, -0.6 kg/m^2) than half-marathoners (-0.8 kg, -0.3 kg/m^2) (p<0.001, p<0.001). An inverse correlation was found between sodium change and weight change, significant in full-marathoners (Spearman correlation r=-0.55, p<0.001) but not half-marathoners (r=-0.23, p=0.042) (Fig. 2). See Table 2 for full data on hyponatraemic participants according to change in pre- and post-race sodium concentration (mmol/l) and weight (kg). Of the 16 participants who were hyponatraemic post-race, 6 showed an increase in weight. The mean weight change for the hyponatraemic marathoners was -0.07 kg^2 (range, -0.9 - 1.4 kg).

The median race pace was 12.7 min/mile (range, 6.9 - 27.4 min/mile) for half-marathoners and 12.7 min/mile (range, 7.4 - 15.2 min/mile) for full-marathoners. A significant negative association was found between pace and post-race serum [Na^+] (Spearman correlation r=-0.30, p<0.001) (Fig. 3). Median finishing times for half- and full-marathoners were 166.1 minutes (range, 90.6 - 358.6 minutes) and 333.7 minutes (range, 194.4 - 398.7 minutes), respectively. Finishing

Table 2. Reporting pre- and post-race hyponatraemic participants according to change in sodium concentration (mmol/l) and weight (kg) post-pre-race (*value not obtained)

Participant	Distance run	Pre-race sodium (mmol/l)	Pre-race hyponatraemia?	Post-race sodium (mmol/l)	Post-race hyponatraemia?	Post - pre sodium (mmol/l)	Pre-race weight (kg)	Post-race weight (kg)	Post-pre-weight (kg)
A	Half	*		134.0	Yes	*	67.6	67.6	0
B	Half	141.0	No	134.3	Yes	-6.7	61.8	63.2	1.4
C	Half	139.8	No	134.0	Yes	-5.8	62.7	63.5	0.8
D	Full	139.0	No	133.0	Yes	-6.0	60.3	59.4	-0.9
E	Full	139.0	No	132.0	Yes	-7.0	64.5	63.8	-0.7
F	Full	142.0	No	133.0	Yes	-9.0	91.5	92.0	0.5
G	Full	136.0	No	130.0	Yes	-6.0	53.7	53.8	0.1
H	Full	139.0	No	133.0	Yes	-6.0	61.5	60.7	-0.8
I	Full	138.0	No	132.0	Yes	-6.0	96.5	95.7	-0.8
J	Full	136.0	No	131.0	Yes	-5.0	65.9	66.6	0.7
K	Full	136.1	No	133.0	Yes	-3.1	49.2	49.0	-0.2
L	Full	136.0	No	125.0	Yes	-11.0	54.2	54.6	0.4
M	Full	138.0	No	133.0	Yes	-5.0	83.4	82.6	-0.8
N	Full	141.9	No	134.0	Yes	-7.9	65.3	65.1	-0.2
O	Half	130.7	Yes	125.0	Yes	-5.7	75.5	*	
P	Full	134.0	Yes	133.0	Yes	-1.0	84.2	83.6	-0.6
Q	Half	134.0	Yes	136.0	No	2.0	75.3	75.3	0
R	Half	105.4	Yes	141.0	No	35.6	54.2	53.1	-1.1
S	Half	134.7	Yes	138.0	No	3.3	76.5	77.2	0.7
T	Half	1330	Yes	135.0	No	2.0	51.5	51.7	0.2
U	Full	134.0	Yes	137.0	No	3.0	64.6	62.9	-1.7
V	Full	133.2	Yes	142.0	No	8.8	50.2	49.7	-0.5

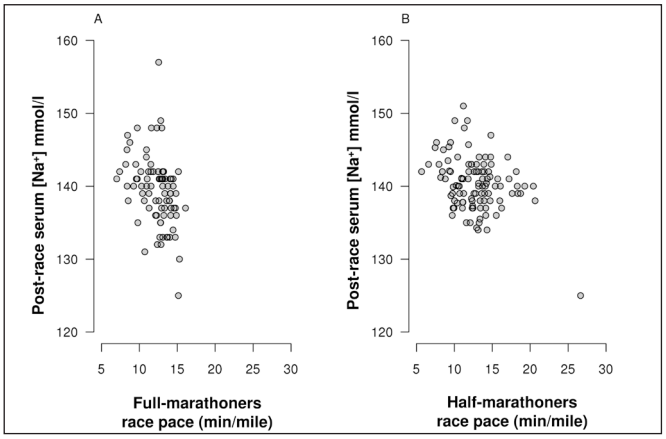

Fig. 3. Scatterplots of post-race serum sodium levels v. race pace for full-marathoners and half-marathoners.

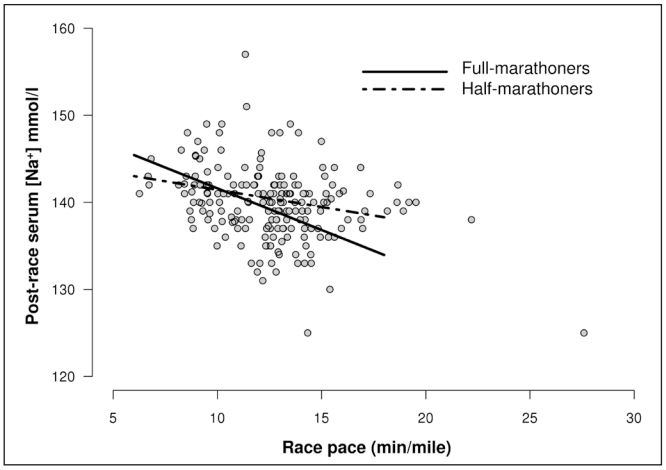

Fig. 4. Scatterplot of the inverse relationship between post-race serum sodium level and race pace, with overlaid linear regression lines for full- and half-marathoners (p=0.03).

time and post-race sodium level had a significant negative correlation as well (Spearman correlation r=-0.28, p=0.001).

Discussion

This study is the first of which we are aware to examine the influence of race pace on the prevalence of hyponatraemia with the benefit of data from both half- and full-marathons. Prior studies have shown an increased incidence of hyponatraemia in marathon runners with completion times of more than 4 hours.[8,11-13,17,21] No studies found in a review of the literature have looked at actual race pace as a predictor of hyponatraemia. The consensus statement from the Second International Exercise-Associated Hyponatraemia Consensus Development Conference 2007 named slower running or performance pace to its list of athlete-related risk factors.[12] However, of the 4 articles cited after this statement,[8,11,17,21] only Almond *et al.*,[8] reporting data from the 2002 Boston Marathon, commented on pace; the others focused on longer finishing times, generally more than 4 hours. Furthermore, Almond *et al.*[8] reported only a slower training pace as statistically significant (self-reported by runners on pre-race surveys), but did not calculate pace on the actual day of the race. As EAH is nearly non-existent in some countries, such as South Africa and New Zealand, our data also implicate the perpetuation of fear of dehydration that may continue to promote overconsumption of fluids in the USA.

Hew *et al.*[17] reported that longer finishing times and overconsumption of fluids were the main risk factors associated with the development of hyponatraemia. Specifically, they state finishing times of more than 4 hours 20 minutes resulted in the lowest serum sodium levels. Although a slower pace can be inferred from this statement, with only a 26.2 mile marathon on which to base their calculations, they were not able to consider distance in relation to change in sodium.

Current global data have established a prevalence of pre-race hyponatraemia of 0 - 2%.[23] Our results demonstrate a pre-race incidence of 6.0% (5/84) for half-marathoners and 3.9% (3/77) for full-marathoners. Higher pre-race incidence of hyponatraemia in the USA raises the question of race participant education. Race packets were picked up at the expo 1 - 2 days prior to the race. Among advertisement of sponsors, HealthSource Magazine (HealthSourceMag.com) was included, which may have included advice from local nutritionists. For example, the February 2010 edition contained an article entitled 'A runner's diet'. Recommendations included: 'To avoid hitting the wall ... you need to focus on your diet and hydration many days before the event. Be sure to increase intake of all fluids and be in a well hydrated state. Dehydration adversely affects athletic performance.'[24] These messages may have encouraged runners to overhydrate prior to the race. The post-race hyponatraemia prevalence of 13% (12/89) in full-marathoners is in agreement with cohorts evaluated in Boston (13%)[8] and London (12%).[23] In contrast, EAH has yet to be reported in a marathon runner in South Africa or New Zealand, and only occasional cases occur in longer ultramarathon races in South Africa (T D Noakes, personal communication).

Our data support the prior finding that a significant inverse relationship exists between total finishing time and serum [Na^+] (p<0.001). Additionally, with the benefit of data from two different distances, we were able to demonstrate the importance of the pace-distance interaction with change in sodium. In Fig. 4, a linear regression model of post-race sodium with pace, distance, and the pace-distance interaction as independent variables found a significant interaction between pace and distance (p=0.03). As interactions are difficult to detect, this model indicates that the relationship between post-race sodium and pace indeed differs according to the distance of the run.

Davis *et al.*[11] reported a retrospective analysis of marathon participants in the 1998 and 1999 Suzuki Rock 'N' Roll Marathon in San Diego, California, who presented within 24 hours after the conclusion of the race to a local emergency department. To define unconditioned v. conditioned athletes, the patients were stratified into those finishing in more or less than 4 hours, respectively. None of the hyponatraemic patients finished the race in less than 4 hours, suggesting that unconditioned athletes, or those who run slowly, are at risk for the development of EAH. Davis *et al.*[11] also discussed the impact of post-race hyperhydration as a major contributor to the development of EAH. A possible limitation to our study design is that post-race blood samples were collected directly after the race; therefore, we only measured a minimum occurrence and may have missed the development of EAH in those participants who then continued to hydrate aggressively over the next 24 hours.

Both Hew *et al.*[17] and Davis *et al.*[11] commented on the inverse relationship of serum sodium to time of presentation. These data were retrospectively reviewed from medical tents and emergency department visits. Our study was geared specifically to screen for abnormal serum sodium concentrations that developed during the

endurance race. Recruited participants were instructed to present to the Runners' Science tent before and after the race. Consequently, we were unable to report on time of presentation in relation to serum sodium except with regard to race finish time. Of note, 360 ml (12 oz) water bottles were available at the finish line, and race participants could drink at will while waiting to have blood drawn. Therefore, it is plausible to assume that out of fear of dehydration, those who used the 'drink as much as possible' philosophy may have continued to overhydrate after the race and thus have had lower serum sodium levels than if they had hydrated to thirst.

Our study is in agreement with the Consensus Statement on EAH[12] that weight gain during a race is a risk factor for hyponatraemia. Of the 16 participants who were hyponatraemic after the race, 6 showed an increase in BMI with a mean change of 0 kg/m^2 (range, -0.3 - 0.5 kg/m^2). Although more full-marathoners developed hyponatraemia during the race, full-marathoners also lost significantly more weight than their counterparts in the half-marathon. This can be explained by a possible 1 - 2% decrease in body weight that can occur during a 26.2 mile marathon without a change in total body water. This scenario may lead to a dilutional hyponatraemic state with a net loss or neutral BMI.

Our results also show that, despite an equal median pace of 12.7 min/mile in both half- and full-marathoners, the latter had a significantly higher prevalence of hyponatraemia (p=0.02). This finding further supports the importance of the performance pace-distance interaction. It may be proposed that a difference between the half- and full-marathon race courses is the number of hydration stations. Along our race course, the availability of hydration stations at each mile marker essentially doubled in the full- v. half-marathon. Reid *et al.*[25] reported that the limitation of fluid availability with placement of aid stations every 5 km has been shown to be associated with absence of hyponatraemia in a standard marathon. We plan to recommend this arrangement of fluid stations for future races.

Our study design is not without its limitations. First, selection bias may have occurred with race participants who are more interested in health outcomes associated with endurance racing. Second, both data sets were incomplete, limiting paired data set analysis. Also, as mentioned above, by collecting blood directly after the race, we lost the ability to detect development of hyponatraemia in the subsequent 24 hours. Finally, our study, as well as race participants overall, may have some gender bias toward a higher female component, 71% (178/251) compared with 34.4% female finishers reported by Hew *et al.*[17] This may be attributable to the nature of the race to benefit breast cancer research and women living with breast cancer. Given that the consensus statement on EAH[12] names female sex as a risk factor, our study population may have been predisposed to development of EAH. Finally, we acknowledge the role of certain confounding factors on the data. Although our figures clearly suggest that slower pace increases the risk of hyponatraemia, we are unable to determine if this is a true physiological difference due to pace. Rather, it may be due to the impact of outside influence of education favouring excessive hydration out of fear of dehydration. Or perhaps, when moving at a slower pace, athletes simply have more time to drink at fluid stations.

In summary, recommendations on fluid replacement during endurance races continue to evolve. Currently at the forefront is drinking *ad libitum* (according to thirst) with the goal to replace fluid lost as sweat. Runners should be encouraged to individualise their hydration strategy on the basis of their particular sweat rates to optimise rather than maximise fluid intake during running.[11] It is imperative that the sports medicine community promote this strategy rather than continue to uphold the unsubstantiated fear of dehydration. Hyponatraemia occurred significantly more frequently in full-marathoners than in half-marathoners. With the benefit of data from half- and full-marathons, we have illustrated the intricate relationship among race pace, distance, and serum sodium concentration. If EAH truly is a behavioural disease based in overconsumption of fluids, then appropriate education and behaviour modification is the key for prevention.

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Please note a subsection of these data has been published in *Sports Health* March/April 2011 issue. Similar graphics and tables were used.

Mohseni M, Silvers S, McNeil B, et al. Prevalence of hyponatraemia, renal dysfunction, and other electrolyte abnormalities among runners before and after completing a marathon or half marathon. Sports Health: A Multidisciplinary Approach March 2011 3:145-151 [http://dx.doi.org/10.1177/1941738111400561].

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