

# Thermoregulation, pacing and fluid balance during mass participation distance running in a warm and humid environment

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**Abstract** Deep body temperature ( $T_c$ ), pacing strategy and fluid balance were investigated during a 21-km road race in a warm and humid environment. Thirty-one males (age  $25.3 \pm 3.2$  years; maximal oxygen uptake  $59.1 \pm 4.2$  ml  $\text{kg}^{-1} \text{min}^{-1}$ ) volunteered for this study. Continuous  $T_c$  responses were obtained in 25 runners. Research stations at approximately 3-km intervals permitted accurate assessment of split times and fluid intake. Environmental conditions averaged  $26.4^\circ\text{C}$  dry bulb temperature and 81% relative humidity. Peak  $T_c$  was  $39.8 \pm 0.5$  ( $38.5$ – $40.7$ )  $^\circ\text{C}$  with 24 runners achieving  $T_c > 39.0^\circ\text{C}$ , 17 runners  $\geq 39.5^\circ\text{C}$ , and 10 runners  $\geq 40.0^\circ\text{C}$ . In 12 runners attaining peak  $T_c \geq 39.8^\circ\text{C}$ , running speed did not differ significantly when  $T_c$  was below or above this threshold ( $208 \pm 15$  cf.  $205 \pm 24$  m  $\text{min}^{-1}$ ;  $P = 0.532$ ). Running velocity was the main significant predictor variable of  $\Delta T_c$  at 21 km ( $R^2 = 0.42$ ,  $P < 0.001$ ) and was the main discriminating variable between hyperthermic ( $T_c \geq 39.8^\circ\text{C}$ ) and normothermic runners ( $T_c < 39.8^\circ\text{C}$ ) up to 11.8 km. A reverse J-shaped pacing profile characterised by a marked reduction in running speed after 6.9 km and evidence of an end-sprint in 16 runners was observed. Variables relating to

fluid balance were not associated with any  $T_c$  parameters or pacing. We conclude that hyperthermia, defined by a deep body temperature greater than  $39.5^\circ\text{C}$ , is common in trained individuals undertaking outdoor distance running in environmental heat, without evidence of fatigue or heat illness.

**Keywords** Core temperature · Hyperthermia · Hydration · Fatigue · Performance

## Introduction

Elevated body deep temperature ( $T_c$ ) has been implicated as a causative factor of accelerated fatigue in the heat. However, the scientific community is divided on the nature of its involvement. Evidence from laboratory trials employing fixed exercise intensities suggests that an inability to maintain the required work rate (i.e. exhaustion) occurs when a  $T_c$  of approximately  $40^\circ\text{C}$  is attained—critical internal temperature (Gonzalez-Alonso et al. 1999; Nybo and Nielsen 2001). Recent evidence from laboratory studies employing self-paced exercise protocols, with intensity free to vary according to individual volition, observe that exercise tasks are successfully completed albeit with a characteristic reduction in work rate early in the exercise and prior to marked hyperthermia (Tattersson et al. 2000; Tucker et al. 2004). This demonstrates that self-paced exercise intensity is regulated well before the attainment of critical  $T_c$ —anticipatory regulation (Marino 2004; Tucker and Noakes 2009). Systematic field studies investigating the relationship between  $T_c$  and self-selected exercise intensity are limited. Therefore, it is unknown whether athletes undertaking competitive endurance exercise in environmental heat in the field reduce their intensity

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at a critical  $T_c$  or reduce their intensity in advance of marked hyperthermia.

There is widespread evidence of  $T_c$  reaching the critical range (i.e.  $\geq 39.5^\circ\text{C}$ ) in asymptomatic individuals undertaking outdoor distance running (Byrne et al. 2006; Ely et al. 2009; Maron et al. 1977; Maughan et al. 1985; Noakes et al. 1991; Pugh et al. 1967; Robinson 1963; Wyndham and Strydom 1969). Post-race rectal temperatures of  $41.1^\circ\text{C}$  have been observed in world-class athletes following races of 4.8 and 10 km (Robinson 1963) and in the winner of a 42-km race (Pugh et al. 1967). To our knowledge, the highest observed  $T_c$  in an asymptomatic runner was reported by Maron et al. (1977), where  $T_c$  was maintained between  $41.6$  and  $41.9^\circ\text{C}$  for the final 44 min of a 42-km road race. More recently, Byrne et al. (2006) reported that 56% ( $n = 10$ ) of their sample attained  $T_c > 40.0^\circ\text{C}$  during a mass-participation 21-km road race in  $27^\circ\text{C}$  ambient temperature and 87% humidity, with two individuals attaining  $T_c$  of  $41.3$  and  $41.7^\circ\text{C}$ . That these observations arose from runners who successfully completed the exercise task, were champion athletes, or who ran faster than all other competitors despite  $T_c > 40^\circ\text{C}$ , appears to refute the hypothesis that exhaustion occurs when a critical  $T_c$  of  $\sim 40^\circ\text{C}$  is attained. A case-study observation that appears to refute the critical  $T_c$  hypothesis was provided over 30 years ago by Maron et al. (1977) who reported an end-spurt, displayed as a substantial increase in speed during the last mile of the race, in an individual with  $T_c \geq 41.6^\circ\text{C}$ .

What remains uncertain is whether these runners slowed when their  $T_c$  reached  $\sim 40^\circ\text{C}$ , since the simultaneous measurement of  $T_c$  and running speed has occurred infrequently in research involving outdoor running (Maron et al. 1977; Ely et al. 2009). The recent investigation of Ely et al. (2009) has provided the strongest evidence against the existence of a threshold  $T_c$  of  $\sim 40^\circ\text{C}$  associated with fatigue. Ely et al. measured rectal temperature and running speed in well-trained distance runners during 8 km (25–33 min) track time trials in cool ( $16.7$ – $17.8^\circ\text{C}$ ) and warm ( $29.5$ – $30.2^\circ\text{C}$ ) conditions, observed  $T_c > 40^\circ\text{C}$  in 12 runners, and reported no difference in running speed when  $T_c$  was  $< 40^\circ\text{C}$  ( $282 \pm 27 \text{ m min}^{-1}$ ) or  $> 40^\circ\text{C}$  ( $279 \pm 28 \text{ m min}^{-1}$ ;  $P = 0.82$ ). The present study extends the work of Ely et al. by investigating a longer distance (21 km) and duration (91–137 min) mass-participation event during which runners were anticipated to experience a greater duration of hyperthermia and also to experience significant dehydration. We aim to address the existence of a critical  $T_c$  threshold for fatigue and to investigate the anticipatory regulation hypothesis by examining the pacing profile of runners and the influence of  $T_c$  rate of change early in the race (i.e. 0–30 min) on subsequent running speed.

Previous field-studies of 42 km running have revealed that running speed and predicted metabolic rate, particularly late in the race, can account for 12–40% of the variation in post-race  $T_c$  (Maughan et al. 1985; Noakes et al. 1991). Surprisingly, fluid ingestion has not been revealed as a significant predictor variable of  $T_c$  responses in field studies despite well-established laboratory evidence demonstrating fluid replacement to be effective in attenuating the rate of rise in  $T_c$  (Montain and Coyle 1992). During outdoor races, it has been observed that endurance athletes do not match the volumes of fluid typically consumed in laboratory studies (Noakes 2002). We have previously reported no association between estimated fluid balance variables (e.g. fluid intake, % dehydration) and the magnitude of  $T_c$  responses (Byrne et al. 2006), in agreement with other field studies (Maughan et al. 1985; Noakes et al. 1991). In our study, estimated sweat rates averaged  $1.5 \text{ l h}^{-1}$  and less than half of these losses were replaced during the race. It is noteworthy that the method of estimating race fluid intake in that study suffered from a lack of precision because it relied on runners recalling the frequency of drinks consumed. Therefore, a further aim of this study was to quantify fluid balance with precision and investigate with greater accuracy the potential contribution of body water variables to the  $T_c$  response.

## Materials and methods

### Ethical approval and participants

Thirty-one male volunteers provided both verbal and written consent to participate in this study. The study conformed to the standards set by the Declaration of Helsinki and the procedures were approved by the Institutional Review Board. Twenty-nine participants were professional soldiers recruited from the Singapore Armed Forces and the remaining two volunteers were recruited from local running clubs. All participants were naturally heat-acclimatised, due to their routine physical training in a tropical climate. Their physical characteristics are presented in Table 1.

### Race day procedures

Measurements took place on the day of the 2007 Singapore Bay Run and Army Half-Marathon, which is an annual mass participation 21 km road race. Participants arrived at an on-site research station at 0330 hours, with the race starting at 0545 hours. Nude body mass was measured after each volunteer produced a urine sample. A heart rate telemetry system was fitted (Polar Vantage, Polar Electro Oy, Kempele, Finland), and runners dressed in their running

**Table 1** Physical and physiological characteristics of the 31 participants

Variable	Mean $\pm$ SD	Range
Age (years)	25.3 $\pm$ 3.2	21–32
Stature (m)	1.72 $\pm$ 0.05	1.63–1.80
Mass (kg)	65.9 $\pm$ 6.1	53.5–77.8
Sum of six skinfolds (mm)	51 $\pm$ 13	30–77
VO <sub>2max</sub> (ml kg <sup>-1</sup> min <sup>-1</sup> )	59.1 $\pm$ 4.2	51.1–67.9
Running Economy (ml kg <sup>-1</sup> km <sup>-1</sup> )	211 $\pm$ 12	187–235
vVO <sub>2max</sub> (m min <sup>-1</sup> )	280 $\pm$ 21	238–330

singlet, shorts, socks, and shoes. Each participant was fitted with an ambulatory telemetric body core temperature data recorder worn on a lightweight harness around the waist. The total weight of the recorder and harness was 250 g. Participants then provided a pre-race venous blood sample and subsequently rested until 30 min prior to the start of the race when they performed warm up and stretching activities, followed by an approximate 500 m walk to the start line. Personnel positioned at 0, 3.0, 6.9, 9.0, 11.8, 14.7, 17.9 and 21.0 km employed chronometry (System Stopwatch S149-4A00, Seiko, Tokyo, Japan) to gain split times for each runner at these distances. Distances were determined by a Polar RS800sd running computer and s3 stride sensor<sup>TM</sup> (Polar Electro Oy, Kempele, Finland) worn by our first finishing volunteer and calibrated according to the manufacturer's instructions. The validity of this accelerometer device for measuring kinematic characteristics of running has recently been demonstrated (Hauswirth et al. 2009). Mean running speed was determined for the whole race and for each split section of the race by dividing the distance run in metres by the time taken in seconds. Pacing for each split section of the race was defined as the percentage of mean 21 km speed and calculated according to the following equation (Ely et al. 2009):

$$\text{Percentage off mean 21 km speed} = \frac{[(\text{Mean speed for split section} - \text{Mean 21 km speed}) / (\text{Mean 21 km speed})] \times 100}$$

where a positive value represents a split section run faster than mean 21 km speed and a negative value represents a split section run slower than mean 21 km speed. The assessment of variation in running speed was determined by calculating a coefficient of variation (CV) from the splits for each runner and the group average CV (Ely et al. 2009).

Upon crossing the finish line, participants walked less than 100 m to the research station where venous blood was sampled and nude body mass measured between 5 and 25 min post-race. A portable weather station

positioned at the finish area recorded dry bulb temperature, wet bulb temperature and air velocity at 1-min intervals throughout the race. Due to equipment failure, globe temperature was not logged. Indices of humidity (i.e. partial water vapour pressure, saturated water vapour pressure and relative humidity) were determined using standard equations (Parsons 2003). Prior to the actual measurements on race day, a practice trial was conducted over 15 km to familiarise runners and researchers to the experimental protocol. This trial fully replicated the experimental trial but no physiological measurements were recorded.

#### Anthropometric measures

Body mass, stature, mid-thigh circumference, maximum calf circumference, and subcutaneous skinfold thickness at the biceps, triceps, subscapular, iliac crest, anterior thigh and medial calf were measured using standard anthropometric procedures (Eston et al. 2009). Body surface area ( $A_D$ ) was estimated ( $A_D = 0.202 \text{ mass}^{0.425} \text{ stature}^{0.725}$ ; DuBois and DuBois 1916). Muscle circumferences of the thigh and calf were estimated from limb circumferences corrected for subcutaneous adipose tissue using the equation of Martin et al. (1990).

#### Measurement of deep body (gastro-intestinal) temperature

Ingestible telemetric temperature sensors and ambulatory data recorders were employed to continuously measure deep body temperature ( $T_c$ ) during the race (Byrne and Lim 2007). Approximately 6 h 15 min to 7 h 15 min before race start, each of the 31 participants ingested a telemetric temperature sensor in the presence of a researcher. Sixteen participants had  $T_c$  measured by a VitalSense<sup>®</sup> sensor and data recording system (Phillips Respironics, Bend, Oregon, USA) and 15 participants had  $T_c$  measured by a CorTemp<sup>TM</sup> sensor and data recording system (HQ Inc., Palmetto, Florida, USA). Prior to ingestion, the accuracy of the manufacturer's sensor calibration was determined for each sensor by comparing sensor temperature against a United Kingdom Accreditation Service certified calibrated mercury-in-glass thermometer (Model LR6016 BS1900, GH Zeal Ltd., London, UK) during immersion in a heated and stirred water bath (Clifton NE4-D, Nickel Electro Ltd, Weston-Super-Mare, UK) at temperatures of approximately 36, 38, 40 and 42°C. All  $T_c$  responses were scrutinised for the potential confounding effect of fluid ingestion invalidating the ingestible sensor (Wilkinson et al. 2008). Deep body temperature data are presented for 25 participants due to fluid intake during the race directly affecting the

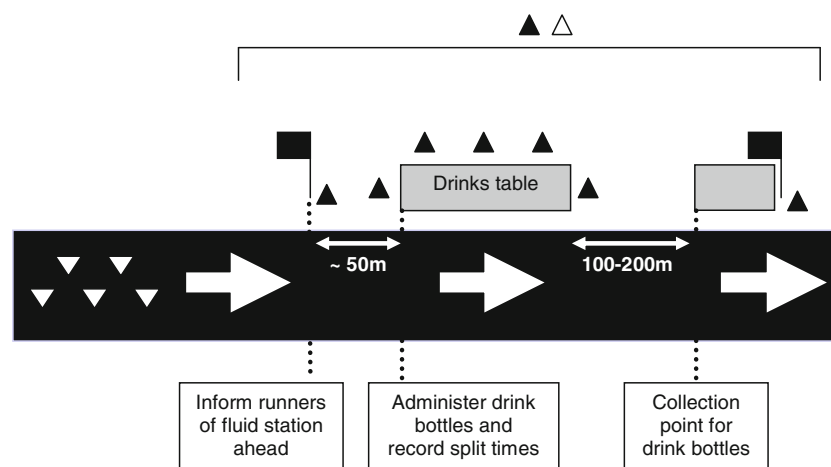
temperature recordings of five participants and data recording failure in one participant.

#### Prediction of metabolic rate and exercise intensity

Individual linear regression equations for the relationships between running speed-oxygen uptake ( $\dot{V}O_2$ ) and running speed-respiratory exchange ratio ( $R$ ) were established for the prediction of  $\dot{V}O_2$ , metabolic rate (Gagge and Gonzalez 1996) and exercise intensity during the race when speed was determined from chronometry and distance. The additional oxygen cost of overcoming air resistance in calm air conditions during outdoor versus level treadmill running was accounted for by applying the equation of Pugh (1970). Approximately 4 weeks prior to the race, participants ran on a level treadmill for 4 min at four separate speeds across the range of 168–235  $\text{m min}^{-1}$ , with an increase of 16.8  $\text{m min}^{-1}$  after each 4-min stage. Treadmill running was performed in an environmental chamber (VEKZ10, Votsch Industrietechnik, Germany) maintained at 27°C dry bulb temperature and 80% relative humidity, in an attempt to simulate the environmental conditions expected during the race (Byrne et al. 2006). Oxygen uptake and  $R$  were measured using a breath-by-breath pulmonary gas analyser (MetaLyzer<sup>®</sup> 3B-R2, CORTEX Biophysik, Leipzig, Germany) and averaged over the final 60 s of each stage. Following 10 min of recovery, maximal oxygen uptake ( $\dot{V}O_{2\text{max}}$ ) was determined using a continuous incremental treadmill test to volitional exhaustion. The highest  $\dot{V}O_2$  recorded over a 60-s period was accepted as  $\dot{V}O_{2\text{max}}$ . The runners' physiological characteristics are presented in Table 1.

#### Body water variables

Urine samples were collected upon waking (approximately 0.5–2.0 h prior to reporting at the race site) and upon arrival at 0330 hours. Each urine sample was assessed for urine specific gravity using a digital refractometer (UG-1, Atago, Tokyo, Japan). Pre and post-race nude body mass were measured to the nearest 0.001 kg using a digital scale (Seca 881, Seca Vogel & Halke GmbH & Co., Hamburg, Germany). Venous blood was sampled from an antecubital vein in the standing position after remaining in this posture for 15 min. Serum was analysed in duplicate for sodium concentration using the ion selective electrode principle (9181 Electrolyte Analyser, Roche Diagnostics, Indianapolis, Indiana, USA). All subsequent fluid intake and urine output were directly measured. Prior to the race, each volunteer had free access to water from an individual drink bottle. During the race, research fluid stations (Fig. 1) were positioned at 3.0, 6.9, 9.0, 11.8, 14.7 and 17.9 km, and participants were given clear instruction to drink only from the research drink stations. At each station, participants had two individual drink bottles available: a bottle containing plain water and a bottle containing carbohydrate-electrolyte fluid (100 Plus, Fraser and Neave Limited, Singapore; carbohydrate 6.7 g  $100 \text{ ml}^{-1}$ ,  $\text{Na}^+$  21  $\text{mEq l}^{-1}$ ,  $\text{Cl}^-$  11  $\text{mEq l}^{-1}$ ). Each bottle contained ~500 ml and was labelled with the runner's number. At the end of the race, volunteers refrained from consuming any further fluid until their post-race body mass was measured. The mass of each drink bottle was determined pre and post-race, with the specific gravity of water (i.e. 1.0) and the carbohydrate-electrolyte fluid (i.e. 1.02442) used to convert the mass change into



**Fig. 1** Representation of the fluid station at every split point. *Inverted open triangle* trial participants; *filled triangle* assistant researchers; *open triangle* overall researcher in charge of the fluid station. An assistant researcher was positioned approximately 50 m ahead of the drinks table to alert incoming trial participants of the

fluid station ahead. Split timings were recorded by the researchers as the trial participants passed a pre-determined line. About 100–200 m away from the drinks table, trial participants were alerted by another assistant researcher to deposit their drink bottles

volume of fluid ingested. The temperature of ingested fluid was determined pre and during race as approximately 25°C. Urine collection containers were available pre-race, at each drink station and post-race to enable urine loss to be quantified. Respiratory evaporative water loss was estimated using the equation by Mitchell et al. (1972). Net mass loss due to substrate oxidation and metabolic water produced during substrate oxidation were estimated using the equation by Maughan et al. (2007). Sweat loss and effective body water loss were estimated from the equations by King et al. (2008). Dehydration was calculated as the effective body water loss expressed as a percentage of pre-race body mass (i.e. % dehydration) and as body mass loss expressed as a percentage of pre-race body mass (i.e. % mass loss).

#### Data handling procedures

Deep body temperature and heart rate data were recorded at 15-s intervals and subsequently averaged over 60 s for analysis. Data loss was experienced from six of the 31 attempted  $T_c$  recordings and from one of the 31 heart rate monitors. Data represent  $T_c$  responses of 25 runners, heart rate responses of 30 runners, and predicted  $\dot{V}O_2$ ,  $M$ , exercise intensity and body water responses of 31 runners. Mean values of  $M$ ,  $\dot{V}O_2$  and heart rate were determined for each split section and for the whole race.

#### Statistical analysis

Data were analysed with SPSS version 13.0 with a significance level of  $P < 0.05$  accepted for all statistical tests. Descriptive data are generally presented as mean  $\pm$  standard deviation (range). The agreement between the ingestible temperature sensors and the criterion thermometer (i.e. sensor accuracy) across four water bath temperatures (i.e.  $\sim 36$ – $42^\circ\text{C}$ ) was examined using Bland and Altman's (1986) limits of agreement method. The high number of runners and relatively wide race route at the 3.0 km mark hindered the identification of our volunteers for split time measurement. Therefore, the first split point described hereafter refers to time taken to complete the first 6.9 km. Single factor (i.e. split section) repeated measures analysis of variance were employed to investigate changes in running speed, pacing and heart rate across the six split sections. Significant main effects were examined with paired  $t$  tests using the Bonferroni procedure to protect against the inflated risk of type 1 error. Two-factor sub-sample (hyperthermic vs. normothermic) versus split-section (0–6.9, 6.9–9.0, 9.0–11.8, 11.8–14.7, 14.7–17.9, 17.9–21.0 km) repeated measures analysis of variance was employed to investigate responses of hyperthermic and normothermic  $T_c$ ,  $\Delta T_c$ ,

speed and pacing responses across split sections. Multiple regression analysis was employed as previously described (Havenith et al. 1998) to investigate significant independent predictor variables for pacing in the 6.9–9.0 km section,  $T_c$  rate of change,  $\Delta T_c$  0–6.9 km and  $\Delta T_c$  0–21 km.

## Results

### Environmental conditions and race topography

Mean (range) dry bulb, wet bulb, ambient vapour pressure, relative humidity and wind velocity were 26.4 (26.1–27.3) °C; 23.9 (23.7–24.4) °C; 2.80 (2.77–2.85) kPa; 81 (79–82) %; and 0.3 (0.0–1.1)  $\text{m s}^{-1}$ , respectively. The race started at 0545 hours with environmental conditions remaining constant for approximately 75 min followed by subtle increases in temperature and water vapour pressure associated with sunrise at 0703 hours. The course consisted of flat road surfaces at sea-level with the only topographical feature being a road bridge occurring after 4.5 km, consisting of a 2.9% uphill gradient for approximately 870 m and a corresponding return to sea-level. Runners returned to level running approximately 500 m prior to the first split time at 6.9 km.

### Running speed, metabolic rate, heart rate and pacing

All volunteers finished the race in a mean duration of  $107 \pm 9$  (91–137) min, corresponding to a mean speed of  $197 \pm 16$  (153–230)  $\text{m min}^{-1}$ . Predicted mean oxygen uptake was  $42 \pm 4$  (35–49)  $\text{ml kg}^{-1} \text{min}^{-1}$ ; relative exercise intensity was  $71 \pm 5$  (59–86) % $\dot{V}O_{2\text{max}}$ ; and metabolic rate was  $530 \pm 54$  (448–634)  $\text{W m}^2$ . Mean heart rate was  $172 \pm 7$  (159–187)  $\text{beats min}^{-1}$  or  $89 \pm 3$  (84–94) % $\text{HR}_{\text{max}}$ , and peak HR was  $185 \pm 7$  (175–196)  $\text{beats min}^{-1}$  or  $96 \pm 3$  (90–100) % $\text{HR}_{\text{max}}$ .

Table 2 illustrates mean running speed and pace (% off mean 21 km speed) across the six split sections. Pacing demonstrated a reverse J-shaped profile characterised by mean 0–6.9 km speed and pace being significantly higher than all but the 17.9–21.0 km speed, and mean 6.9–9.0 km speed being significantly lower than all other split sections. This response was very consistent at the individual level with 18 runners (60%) achieving a pace faster than their individual CV during the 0–6.9 km split, and 27 (90%) achieving a pace slower than individual CV during the 6.9–9.0 km split. Sixteen runners (52%) demonstrated an end-spurt by increasing mean speed over the final 3.1 km by  $4.2 \pm 3.8$  (0.1–12.2) % compared with mean 21 km speed, but only five of these exceeded their individual CV. Fifteen runners reduced mean speed over the final 3.1 km by



**Table 2** Running speed and pace for each split section of the race

	Race section (km)					
	0–6.9	6.9–9.0	9.0–11.8	11.8–14.7	14.7–17.9	17.9–21.0
Running speed ( $\text{m min}^{-1}$ )	$208 \pm 17^\dagger$	$181 \pm 17^\ddagger$	$194 \pm 15$	$196 \pm 16$	$193 \pm 21$	$198 \pm 23$
Hyperthermic ( $n = 12$ )	$218 \pm 15^{**}$	$189 \pm 20^*$	$200 \pm 17^*$	$202 \pm 21$	$201 \pm 28$	$206 \pm 26$
Normothermic ( $n = 13$ )	$200 \pm 14$	$172 \pm 12$	$188 \pm 10$	$192 \pm 11$	$187 \pm 14$	$192 \pm 23$
Pace (% off mean 21 km velocity)	$5.7 \pm 4.8^\dagger$	$-8.4 \pm 4.7^\ddagger$	$-1.8 \pm 2.9$	$-0.7 \pm 3.9$	$-2.2 \pm 4.8$	$0.3 \pm 6.1$
Hyperthermic ( $n = 12$ )	$6.6 \pm 5.6$	$-7.9 \pm 3.2$	$-2.4 \pm 2.9$	$-1.8 \pm 2.0$	$-2.4 \pm 6.2$	$-0.10 \pm 1.3$
Normothermic ( $n = 13$ )	$4.9 \pm 5.0$	$-9.5 \pm 6.7$	$-1.6 \pm 2.8$	$0.8 \pm 5.3$	$-1.8 \pm 4.1$	$0.40 \pm 8.2$

$^\dagger$  Value for the section is significantly different than all but the 17.9–21.0 km value,  $P < 0.01$ .  $^\ddagger$  Value for the split section is significantly different than all other split section values,  $P < 0.01$ . Hyperthermic value is significantly higher than normothermic value,  $* P < 0.05$ ;  $** P < 0.01$

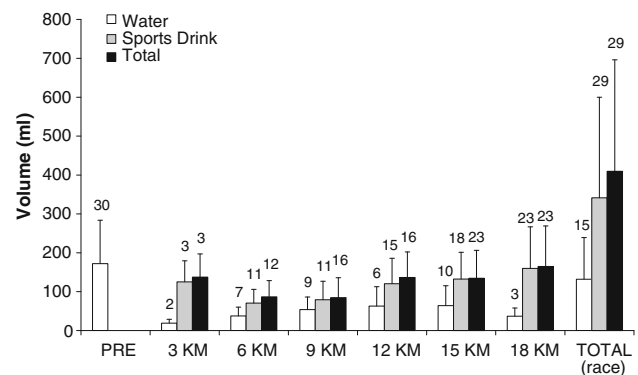
$4.3 \pm 4.8$  (0.2–17.9) %, with only two of these exceeding their individual CV.

### Fluid balance

Urine specific gravity upon waking and arrival for the race were  $1.014 \pm 0.008$  (1.002–1.029) and  $1.015 \pm 0.007$  (1.001–1.026) units, respectively. Three of the runners had urine specific gravity that exceeded 1.020 before the race. Serum sodium concentration increased significantly ( $P < 0.01$ ) from  $140 \pm 2$  (137–145) pre-race to  $146 \pm 2$  (142–149)  $\text{mmol l}^{-1}$  post-race. Mean body mass loss after accounting for urine output was  $2.47 \pm 0.63$  (1.33–4.46) kg, corresponding to  $3.7 \pm 0.9$  (1.9–6.2) % of mass loss. Sweat loss was  $2.58 \pm 0.59$  (1.51–4.68) l and occurred at a rate of  $1.45 \pm 0.32$  (0.83–2.42)  $\text{l h}^{-1}$ . Effective body water loss was  $2.22 \pm 0.61$  (1.10–4.17) l, resulting in  $3.4 \pm 0.8$  (1.5–5.8) % of effective dehydration. Race fluid intake replaced  $15 \pm 11$  (0–44) % of sweat loss. When pre-race intake was also considered, this increased slightly to  $21 \pm 12$  (0–56) % of sweat loss replaced. Figure 2 depicts the type (water and sports drink), volume and frequency of fluid intake before and during the race. During the race, the sports drink was ingested by a greater proportion of the runners (94 cf. 48%), in greater volume ( $341 \pm 259$  cf.  $132 \pm 107$  ml) and at a higher rate ( $187 \pm 138$  cf.  $75 \pm 62$   $\text{ml h}^{-1}$ ).

### Accuracy of ingestible temperature sensors

Limits of agreement analysis for the 25 successful sensors versus the criterion thermometer revealed a non-significant ( $P = 0.36$ ) systematic bias of  $0.00^\circ\text{C}$  with 95% of differences within  $\pm 0.04^\circ\text{C}$ . The maximum difference for any single comparison was  $0.07^\circ\text{C}$ , which fell within the manufacturers' accuracy assurance of  $\pm 0.10^\circ\text{C}$ . Individual linear regression equations were developed for the relationship between ingestible sensor and criterion thermometer temperature, which enabled the minor correction of raw



**Fig. 2** Volume, frequency and type (water or sports drink) of fluid intake before and during the race. Only water was available before the race. Data labels denote the frequency of volunteers ingesting fluid at each drink station. Total volume consumed relates only to fluid intake during the race

sensor data to validated  $T_c$ . These relationships were highly linear and significant (mean  $R^2 = 1.0 \pm 0.0$ ,  $\text{SEE} = 0.02 \pm 0.01^\circ\text{C}$ ,  $P < 0.01$ ).

### Deep body temperature responses

Peak  $T_c$  was  $39.8 \pm 0.5$  (38.5–40.7)  $^\circ\text{C}$  and peak  $\Delta T_c$  was  $2.4 \pm 0.5$  (1.4–3.2)  $^\circ\text{C}$ . Twenty-four of the 25 runners achieved  $T_c > 39.0^\circ\text{C}$ , 17 runners  $\geq 39.5^\circ\text{C}$ , and 10 runners  $\geq 40.0^\circ\text{C}$ . The latter 10 runners achieved a  $T_c$  of  $40^\circ\text{C}$  after  $81 \pm 17$  (54–105) min and maintained  $T_c \geq 40^\circ\text{C}$  for  $17 \pm 12$  (0–37) min. This corresponded to  $79 \pm 18$  (44–100) % and  $17 \pm 13$  (0–41) % of race duration, respectively. Deep body temperature increased at a rate of  $3.5 \pm 0.7$  (2.2–4.6)  $^\circ\text{C h}^{-1}$  during the initial 30 min of running with  $\Delta T_c$  after 6.9 km [duration =  $33 \pm 3$  (29–43) min] accounting for  $67 \pm 10$  (51–91) % of  $\Delta \text{peak } T_c$ .

A two-component regression model incorporating running speed and lean body mass (LBM) explained 38% of the variance in  $T_c$  rate of change from 0 to 30 min. The standardised beta values indicate that as mean speed over

the initial 6.9 km increases by  $17 \text{ m min}^{-1}$ ,  $T_c$  rate of change increases by  $0.50^\circ\text{C h}^{-1}$  ( $\beta = 0.50$ ,  $R^2 = 0.25$ ,  $P = 0.007$ ), and as LBM increases by 4.8 kg,  $T_c$  rate of change increases by  $0.39^\circ\text{C h}^{-1}$  ( $\beta = 0.39$ ,  $P = 0.002$ ). A single variable model consisting of mean 0–6.9 km metabolic rate ( $W$ ) explained 27% of the variance in  $\Delta T_c$  0–6.9 km: as  $M$  increases by 118 W,  $\Delta T_c$  0–6.9 km increases by  $0.08^\circ\text{C}$  ( $\beta = 0.52$ ,  $P = 0.01$ ). A two-component model incorporating mean 21 km speed and thigh muscle circumference ( $C_m$ ) explained 50% of the variance in  $\Delta T_c$  0–21 km: as speed increases by  $16 \text{ m min}^{-1}$ ,  $\Delta T_c$  0–21 km increases by  $0.47^\circ\text{C}$  ( $\beta = 0.67$ ,  $R^2 = 0.42$ ,  $P < 0.001$ ), and as thigh  $C_m$  increases by 2.4 cm,  $\Delta T_c$  0–21 km increases by  $0.22^\circ\text{C}$  ( $\beta = 0.31$ ,  $P = 0.043$ ).

The sample was divided into hyperthermic (i.e. individual peak  $T_c \geq$  mean peak  $T_c$  of  $39.8^\circ\text{C}$ ,  $n = 12$ ) and normothermic (i.e. individual peak  $T_c <$  mean peak  $T_c$  of  $39.8^\circ\text{C}$ ,  $n = 13$ ) runners. Peak  $T_c$  for the hyperthermic subgroup was  $40.2 \pm 0.3$  ( $39.8\text{--}40.7^\circ\text{C}$ ). As illustrated in Table 3,  $T_c$  for hyperthermic versus normothermic runners was not significantly different at race start but was significantly higher by 6.9 km and remained significantly higher at 9.0, 11.8, 14.7, 17.9 and 21.0 km. Table 3 also illustrates that  $\Delta T_c$  was significantly greater in hyperthermic runners from 0 to 6.9, 6.9 to 9.0, and 9.0 to 11.8 km, but not thereafter. Hyperthermic runners displayed a trend for greater  $T_c$  rate of change during the initial 30 min of running ( $3.7 \pm 0.6$  cf.  $3.2 \pm 0.7^\circ\text{C h}^{-1}$ ;  $P = 0.067$ ). Hyperthermic runners displayed a significantly faster mean 21 km speed than normothermic runners ( $205 \pm 20$  cf.  $190 \pm 20 \text{ m min}^{-1}$ ;  $P = 0.027$ ). Table 2 illustrates that mean speed was significantly faster for hyperthermic runners from 0 to 6.9, 6.9 to 9.0, and 9.0 to 11.8 km. There were no significant differences in pace (% off mean 21 km speed) across splits between hyperthermic and normothermic runners. Hyperthermic runners displayed higher  $\text{VO}_{2\text{max}}$  values ( $62 \pm 4$  cf.  $57 \pm 4 \text{ ml kg}^{-1} \text{ min}^{-1}$ ;  $P = 0.014$ ), higher predicted mean 21 km  $\text{VO}_2$  ( $45 \pm 4$  cf.  $41 \pm 3 \text{ ml kg}^{-1} \text{ min}^{-1}$ ;  $P = 0.012$ ) and metabolic rate ( $569 \pm 49$  cf.  $508 \pm 46 \text{ W m}^2$ ;  $P = 0.004$ ). Hyperthermic

and normothermic runners did not differ with regard to any anthropometric or fluid balance variable.

### Deep body temperature and running performance

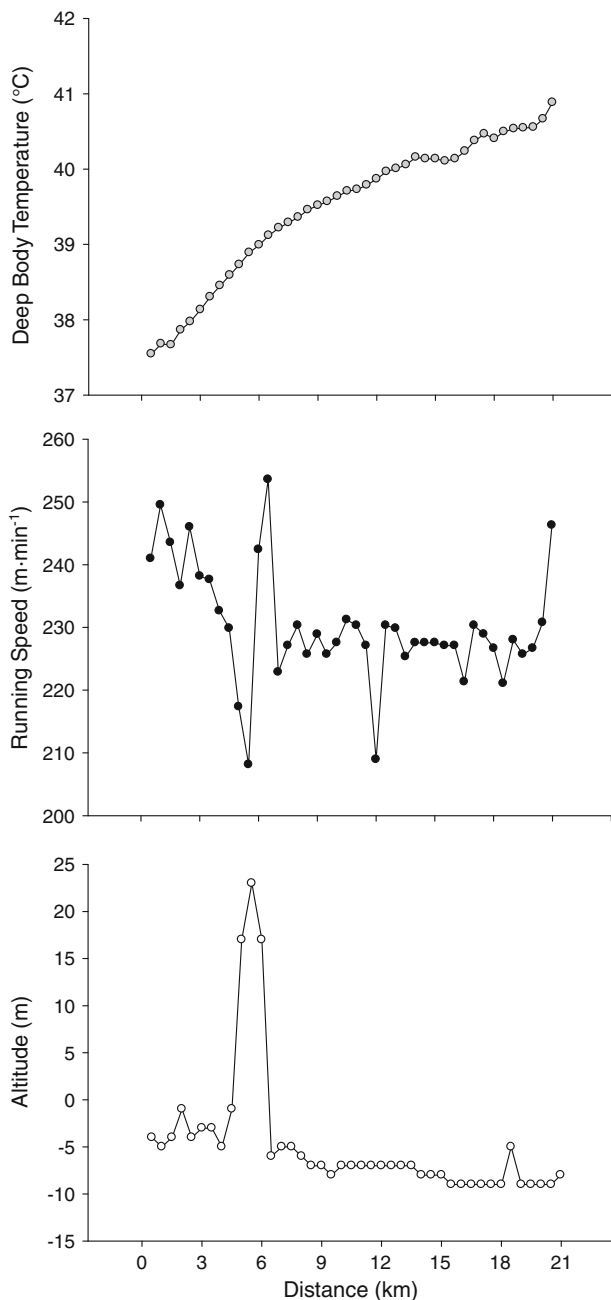
The greatest and most consistent change in pace was observed from 0–6.9 to 6.9–9.0 km (Table 2). Fifty-eight per cent of the variation in the percentage reduction in speed from 0–6.9 to 6.9–9.0 km was explained by a three-component regression model comprising of mean  $\% \text{VO}_{2\text{max}}$  over 0–6.9 km, thigh muscle circumference and  $T_c$  at 6.9 km. The standardised beta values indicate that as mean  $\% \text{VO}_{2\text{max}}$  over 0–6.9 km increased by 5%, speed over 6.9–9.0 km decreased by 3.7% ( $\beta = 0.62$ ,  $P < 0.001$ ); as thigh muscle circumference increased by 3.1 cm, speed decreased by 3.0% ( $\beta = 0.51$ ,  $P = 0.002$ ); and as  $T_c$  at 6.9 km increased by  $0.30^\circ\text{C}$ , speed increased by 3.0% ( $\beta = -0.51$ ,  $P = 0.002$ ). No significant relationship was observed between  $T_c$  rate of rise (0–30 min) and 6.9–9.0 km pace ( $R^2 = 0.02$ ,  $P = 0.846$ ).

Within the hyperthermic group, running speed was not significantly different when  $T_c$  was  $<39.8^\circ\text{C}$  versus  $\geq 39.8^\circ\text{C}$  ( $208 \pm 15$  cf.  $205 \pm 24 \text{ m min}^{-1}$ ;  $P = 0.532$ ). Individual responses reveal that in the presence of a  $39.8^\circ\text{C}$   $T_c$ , six of the 12 hyperthermic runners increased running speed by  $6 \pm 6$  ( $1\text{--}16$ )  $\text{m min}^{-1}$  and six reduced speed by  $11 \pm 14$  ( $1\text{--}39$ )  $\text{m min}^{-1}$ . If one of the latter runners (who suffered cramp, adopted a walk-run strategy and reduced speed by  $39 \text{ m min}^{-1}$ ) is excluded, the remaining five runners reduced speed by  $5 \pm 4$  ( $1\text{--}10$ )  $\text{m min}^{-1}$ . These changes in speed fell within the individual CV for all but one runner who increased speed to an extent greater than his individual CV. Greater pacing resolution was available for the fastest runner in our sample who also recorded the highest  $T_c$ . Figure 3 illustrates  $T_c$ , running speed and course elevation at 0.5 km intervals for this runner. It should be noted that this runner exhibited a very conservative pacing profile as illustrated by a low individual CV across splits in comparison with the sample mean (i.e. 1.6 cf. 5.9%), and less pronounced changes in pace in comparison with the sample mean during the 0–6.9 km (i.e. 2.4 cf. 5.7%) and 6.9–9.0 km (i.e.  $-1.6$  cf.

**Table 3** Deep body temperature responses for each split section of the race

	Race distance (km)						
	0	6.9	9.0	11.8	14.7	17.9	21.0
Deep body temperature ( $^\circ\text{C}$ )							
Hyperthermic ( $n = 12$ )	$37.4 \pm 0.2$	$39.2 \pm 0.1^{**}$	$39.4 \pm 0.2^{**}$	$39.6 \pm 0.3^{**}$	$39.7 \pm 0.2^{**}$	$39.9 \pm 0.3^{**}$	$40.1 \pm 0.4^{**}$
Normothermic ( $n = 13$ )	$37.4 \pm 0.2$	$38.8 \pm 0.3$	$38.9 \pm 0.3$	$38.9 \pm 0.3$	$38.9 \pm 0.4$	$39.1 \pm 0.4$	$39.2 \pm 0.4$
$\Delta$ Deep body temperature ( $^\circ\text{C}$ )							
Hyperthermic ( $n = 12$ )		$1.78 \pm 0.17^*$	$0.21 \pm 0.12^*$	$0.22 \pm 0.24^*$	$0.13 \pm 0.15$	$0.20 \pm 0.32$	$0.24 \pm 0.16$
Normothermic ( $n = 13$ )		$1.38 \pm 0.26$	$0.09 \pm 0.14$	$0.02 \pm 0.13$	$0.09 \pm 0.15$	$0.11 \pm 0.13$	$0.11 \pm 0.13$

Hyperthermic value is significantly higher than normothermic value, \*  $P < 0.05$ ; \*\*  $P < 0.01$



**Fig. 3** Deep body temperature, running speed and elevation versus race distance for the fastest runner

–8.4%) splits. At 20 km when  $T_c$  was  $40.6^\circ\text{C}$  and mean speed was  $227\text{ m}\cdot\text{min}^{-1}$ , this individual initiated an end-sprint by increasing speed to  $231\text{ m}\cdot\text{min}^{-1}$  over 20.0–20.5 km and  $246\text{ m}\cdot\text{min}^{-1}$  over 20.5–21.0 km. Deep body temperature increased to  $40.7^\circ\text{C}$  at 20.5 km and  $40.9^\circ\text{C}$  at 21.0 km.

## Discussion

There are limited studies providing intra-race measurements of  $T_c$  during competitive events. In this field study,  $T_c$

was continuously and simultaneously profiled in 25 competitive runners during a mass participation 21 km road race in a tropical climate, where environmental conditions are known to be unfavourable for peak performance (Galloway and Maughan 1997). By positioning research stations along the route, we attempted to derive split time and to accurately quantify fluid intake during the race. The measurement of the former, together with the continuous profiling of  $T_c$ , provides insights to pacing in relation to  $T_c$  during a competitive half marathon race in the heat. The latter permits precise data describing fluid intake during a half marathon race to ascertain its influence on  $T_c$  during outdoor distance running, as prior studies with accurate fluid intake quantification were not conducted in actual races (Burke et al. 2005; Passe et al. 2007). Although logistical challenges, lack of control and other limitations exist in field research, its key ecological advantage is that volunteers are motivated to perform to their maximum capacity during actual racing, in contrast to laboratory investigations. In addition, it is almost impossible to create conditions associated with a real-life mass participation event. Our data do not support the hypothesis that a critical  $T_c$  in the range of  $39.5\text{--}40.4^\circ\text{C}$  is associated with fatigue or exhaustion. Two-thirds of the  $T_c$  responses in this study were  $\geq 39.5^\circ\text{C}$  and none of these runners succumbed to exhaustion. Mean running speed of the 12 hyperthermic runners (i.e. individual peak  $T_c \geq$  mean peak  $T_c$  of  $39.8^\circ\text{C}$ ) was not significantly different before and after the attainment of a  $39.8^\circ\text{C}$   $T_c$ . The greater resolution of pacing data for our fastest runner illustrated the initiation of a marked increase in speed over the final kilometre, which commenced when  $T_c$  was  $40.6^\circ\text{C}$  and rose to  $40.9^\circ\text{C}$  at the finish. Collectively, these findings refute the association of a critical  $T_c$  in the range of  $39.5\text{--}40.4^\circ\text{C}$  with an acute reduction in exercise intensity. Our data are in direct agreement with the recent field-based observational study of Ely et al. (2009) who reported no significant difference in running speed with  $T_c$  below or above  $40.0^\circ\text{C}$  during an 8-km track time trial. Our data also support observations that individuals can increase exercise intensity when their  $T_c$  is in the critical range (Ely et al. 2009; Maron et al. 1977). Finally, our data complement the evidence base of observational field studies reporting  $T_c \geq 39.5^\circ\text{C}$  in asymptomatic runners completing distance races successfully without undue fatigue or exhaustion (Byrne et al. 2006; Ely et al. 2009; Maron et al. 1977; Maughan et al. 1985; Noakes et al. 1991; Pugh et al. 1967; Robinson 1963; Wyndham and Strydom 1969). The current evidence suggests that in trained individuals, a  $T_c > 39.5^\circ\text{C}$  per se is not associated with accelerated fatigue or heat stroke. These phenomena would appear to involve hyperthermia and further unidentified parameters that have yet to exert meaningful effects in this study or any of the aforementioned observational studies.



There is a scarcity of data on the pacing strategies employed by endurance runners. Pacing responses revealed in the current study had a reverse J-shaped profile, which is consistent with pacing strategies observed in diverse athletic competitions (Abbiss and Laursen 2008). Comparison with mean 21 km speed revealed a faster pace (5.7%) during the first 6.9 km and a slower pace (−8.4%) during the 6.9–9.0 km split section. Although our pacing data suffer from low resolution, the reduction in pace after ~30% of the race is in agreement with previous laboratory trials of self-paced exercise in heat demonstrating an early reduction in exercise intensity (Tattersson et al. 2000; Tucker et al. 2006). For example, Tucker et al. (2006) observed a progressive reduction in power output after 30% of a 20-km cycling time-trial in heat and Tattersson et al. (2000) observed a decline in power output after 20% of a 30-min cycling time-trial in heat. A unique pacing feature in the current study was the marked reduction in pace during the 6.9–9.0 km split and a rebound increase in pace from 9.0 km onwards, albeit slower than the 0–6.9 km pace. The change in pace after 6.9 km occurred during level running, although this followed approximately 1.7 km of uphill and downhill running, and occurred in the presence of low and unchanged wind resistance and unchanged running surface. This would not appear to represent a readjustment to level running, since a short transient increase in speed is observed when level running is preceded by downhill running (Townshend et al. 2010). When greater resolution was available (Fig. 3), a progressive decrease in pace was evident from much earlier in the race (i.e. 1–2 km), followed by the maintenance of a slower pace. These field data support laboratory observations of an early reduction in exercise intensity during self-paced endurance exercise in heat (Tattersson et al. 2000; Tucker et al. 2006).

The  $T_c$  associated with the most marked and consistent reduction in running speed in this study (i.e. at 6.9 km) was  $39.0 \pm 0.3$  (38.1–39.4) °C and below the range commonly associated with hyperthermia-induced fatigue. Based on greater resolution from our fastest runner (Fig. 3), we hypothesise that reductions in running speed began before the 6.9 km mark and therefore, at lower levels of  $T_c$  than stated above. The early reduction in speed prior to marked hyperthermia (i.e.  $\geq 39.5^\circ\text{C}$ ) is consistent with the ‘anticipatory regulation’ model of exercise intensity regulation (Marino 2004; Tucker and Noakes 2009). Proponents of the anticipatory model suggest that the rate of heat storage early in exercise is associated with subsequent regulation of exercise intensity (Marino 2004; Tucker et al. 2006). That is, the higher the  $T_c$  rate of rise, the greater the reduction in exercise intensity (Tucker et al. 2006). Our data do not support a significant relationship between  $T_c$  rate of rise in the first 30 min of running and the

subsequent relative change in running speed from 6.9–9.0 km ( $R^2 = 0.02$ ,  $P = 0.846$ ) occurring after 29–43 min. Nevertheless, we were able to explain 58% of the variance in 6.9–9.0 km pace with a three-component regression model consisting of % $\text{VO}_{2\text{max}}$  over 0–6.9 km ( $\beta = 0.62$ ), lean thigh circumference ( $\beta = 0.51$ ) and  $T_c$  at 6.9 km ( $\beta = -0.51$ ). That sustaining a higher relative intensity in the first 6.9 km was associated with a greater reduction in subsequent speed is consistent with a greater early physiological stress influencing negatively on subsequent pacing. A higher lean thigh circumference being associated with greater reductions in subsequent running speed possibly represents a refinement of the basic dependency of performance in heat on body mass (Hayward et al. 1986). Finally, the association of a higher  $T_c$  at 6.9 km with a lower reduction in subsequent running speed appears contradictory to predictions of the anticipatory regulation hypothesis. In summary, our data support the anticipatory regulation hypothesis in that intensity is reduced in advance of marked hyperthermia but do not reveal a role for  $T_c$  rate of change influencing subsequent exercise intensity.

Our data confirm the strong positive association between running speed and the magnitude of  $T_c$  elevation (Noakes et al. 1991). Mean 21 km running speed explained 42% of the variance in  $\Delta T_c$  at 21 km. Speed was also found to be higher up to 11.8 km in hyperthermic runners (i.e.  $\geq 39.8^\circ\text{C}$ ). The direct linear relationship between running velocity and metabolic rate (Margarita et al. 1963) and the limited evaporative capacity of the warm, humid environment (Havenith et al. 1998) possibly account for the greater strength of association between speed and final  $T_c$  in the current study (i.e.  $R^2 = 0.42$ ) compared with previous studies in cool or temperate environments (i.e.  $R^2 = 0.11$ –15).

The current sample of predominantly professional elite soldiers recorded a mean finish time (i.e.  $107 \pm 9$  min) that was 11 min less than our previous study on predominantly national service soldiers (Byrne et al. 2006). This mean finish time was 12 min longer than recreational runners in the 21-km field study by Williams and Nute (1983), and 13 and 33 min longer than slow and fast recreational runners, respectively, in the 21-km field study by Selley et al. (1995). Despite our sample having a comparable  $\text{VO}_{2\text{max}}$  with the Williams and Nute sample (i.e.  $59.1 \pm 4.2$  cf.  $58.1 \pm 5.5$  ml  $\text{kg}^{-1}$   $\text{min}^{-1}$ ), our runners’ predicted mean  $\text{VO}_2$  (i.e.  $42 \pm 4$  cf.  $46 \pm 5$  ml  $\text{kg}^{-1}$   $\text{min}^{-1}$ ) and relative exercise intensity (i.e.  $71 \pm 5$  cf.  $79 \pm 5\%$   $\text{VO}_{2\text{max}}$ ) during the race were lower. Our runners’  $\text{VO}_{2\text{max}}$  values also compared favourably with the slow ( $55.8 \pm 2.9$  ml  $\text{kg}^{-1}$   $\text{min}^{-1}$ ) and fast ( $61.0 \pm 2.6$  ml  $\text{kg}^{-1}$   $\text{min}^{-1}$ ) runners in Selley et al. (1995). We suggest that the relative inferior performance of our runners is due to a lower task-specific training status and the deleterious effects of the warm and humid environment

(Tatterson et al. 2000; Tucker et al. 2006). Intensity of effort, determined by measured mean heart rate responses (i.e.  $89 \pm 3\%HR_{\max}$ ), was similar to those observed for slow ( $89 \pm 2\%HR_{\max}$ ) and fast ( $91 \pm 1\%HR_{\max}$ ) runners in the study by Selley et al. (1995). We suggest that our sample is representative of a trained sample and produced an intensity of effort comparable to previous observations on competitive distance running.

Limited studies have accurately assessed fluid balance during the mass participation endurance races. Some have done so via simulated races (Burke et al. 2005; Passe et al. 2007), but it is known that actual race settings affect behavioural strategies in consuming fluids (Passe et al. 2007). One of the earliest reports was by Wyndham and Strydom (1969), but that study was conducted in cool conditions. In contrast to previous studies (Burke et al. 2005; Passe et al. 2007) where runners replaced  $\sim 30\%$  of fluid loss, our runners ingested about 0.21 l of fluid hourly when average sweat rate was 1.45 (0.83–2.42)  $l\ h^{-1}$ , and replaced  $\sim 15$  (0–44) % of sweat loss. This observation extends previous notions that the inadequacy of fluid intake to match sweat loss may be greater during a half marathon in a warm and humid environment. In comparison with unflavoured water, voluntary drink intake is increased when carbohydrate-electrolyte drinks are given ad libitum during exercise in the heat (Wilk and Bar-Or 1996). Total race intake of water in our runners was  $60 \pm 100$  ml (0–350 ml), which was 81% less than the volume of sports drink ingested ( $320 \pm 260$  ml; 0–1210 ml). This failure to match fluid loss with intake is unsurprising, as it has long been shown that ad libitum drinking results in dehydration when exercising in the heat (Rothstein et al. 1947), and marathon runners frequently choose drink volumes that result in  $>2\%$  dehydration despite accessibility to fluid stations (Cheuvront and Haymes 2001). Based on pre and post-body mass, our runners experienced  $3.7 \pm 0.9$  (1.9–6.2) % dehydration. It is noteworthy that our fastest runner did not consume any fluid in the 2 h before the race and during the race. As suggested by Burke et al. (2005), the need to slow down at aid stations, a disruption of race rhythm and the potential of gastro-intestinal discomfort (especially with increasing exercise intensity) could explain this behavioural strategy. Regulation of plasma osmolality between 280 and 295  $mosmol\ kg^{-1}$  triggers the main controller of thirst. The increased fluid intake by our runners as the race progressed may imply an increased urge (thirst response) to ingest fluid with concomitant increase in blood osmolality (not measured). The increased frequency and volume of sports drink ingested, in comparison with water, are likely attributable to its enhanced palatability and the well-known benefits of its consumption.

Position Stands and Statements on fluid replacement during exercise (Hew-Butler et al. 2006; Sawka et al. 2007)

suggest, “the goal of drinking during exercise is to prevent excessive ( $>2\%$  body mass loss from water deficit) dehydration”. Our results in heat-acclimatised and mainly professional soldiers participating in a competitive 21 km road race with plentiful access to fluids and drinking ad libitum revealed no ill effects of dehydration up to 6.2% body mass loss. This observation does not support a causative relationship between dehydration and heat-related illnesses. However, whether performance could have been improved if efforts were made to increase fluid intake and prevent such substantial dehydration could not be ascertained from the study design. Dugas et al. (2009) suggests that fluid intake greater than ad libitum neither reduces thermal strain nor enhances performance in the heat (33°C with 50% relative humidity). In that study, six male cyclists completed 80 km time trials on separate occasions, commencing with ad libitum drinking on the first trial to estimate sweat loss. During subsequent trials, the cyclists replaced 0, 33, 66 or 100% of body mass lost. The authors concluded that ad libitum fluid ingestion is sufficient to prevent the negative consequences of under- and over-drinking.

There is sufficient scientific evidence from studies conducted in well-controlled conditions in laboratories to show the inverse relationship between fluid ingestion and  $T_c$ . However, the difference in volume consumed during crossover trials (Montain and Coyle 1992) may not be translatable to actual field settings. Ecological validity can be ensured by extending the often practised discretions in identifying differences between laboratory and field based studies, inclusive of but not limited to exercise intensity, ambient environment and mode of exercise, to typical fluid volumes consumed in actual settings. Full replacement of fluid loss during prolonged exercise in hot environments is rare and difficult (Sawka et al. 2007). The inverse relationship between fluid ingestion and  $T_c$  cannot be extrapolated to real situations when the differences in volume consumed are minute in comparison with the larger differences induced between experimental trials in laboratory studies. In addition, Armstrong et al. (1997) has shown via a laboratory study that  $T_c$  responses with ad libitum and no fluid intake during a heat-stress test were similar in pre-test euhydrated men. The volume of fluid ingested ad libitum was similar to that in our study ( $\sim 0.21\ l\ h^{-1}$ ). However, raised  $T_c$  and heart rates were observed when volunteers were hypohydrated pre-test and not provided fluid, implying that fluid ingestion had a significant influence only on physiological responses of hypohydrated individuals. Utilising a urine specific gravity of 1.020 as the threshold for hypohydration (Casa et al. 2000), only three of our runners were hypohydrated ( $1.015 \pm 0.007$ ; 1.001–1.026). The majority of our sample was euhydrated prior to the race and this may explain the negligible effect of fluid intake on  $T_c$ .

Our observations echo the comments by Costill et al. (1970), who attributed the lack of association between dehydration and marathon running performance to the limited amount of fluids ingested in actual field settings. Taken together, our findings do not refute the efficacy of fluid ingestion on attenuation of  $T_{c}$ , but suggest that the large amount of fluids ingested in laboratory based studies is seldom achieved during actual field races, and hence the physiological benefits derived from water consumption during actual field settings is likely to be small.

In conclusion, our study findings suggest that hyperthermia, defined by a body core temperature greater than 39.5°C, is common in trained individuals undertaking outdoor distance running in environmental heat, without evidence of fatigue or heat illness. Running velocity was the main significant predictor variable of  $\Delta T_{c}$  at 21 km and was the main discriminating variable between hyperthermic and normothermic runners up to 11.8 km. Fluid volumes ingested during competitive races have no detectable effect on any of the variables relating to  $T_{c}$  or performance.

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