Within-subject Variation of Thermoregulatory Responses during Repeated Exercise Bouts

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Abstract

Aim: To assess the within-subject variation of thermoregulatory responses during 2 consecutive 15-km road races. Secondly, we explored whether gastrointestinal temperature (TGI) data from the first race could improve our previously established predictive model for finish TGI in the second race.

Methods: We measured TGI before and immediately after both races in 58 participants and determined correlation coefficients. Finish TGI in the second race was predicted using a linear regression analysis including age, BMI, pre-race fluid intake, TGI increase between baseline and the start of the race and finish TGI in the first race.

Results: Under cool conditions (WBGT 11–12°C), TGI was comparable between both races at baseline (37.6±0.4°C vs. 37.9±0.4°C; p=0.24) and finish (39.4±0.6°C vs. 39.4±0.6°C; p=0.83). Finish TGI correlated significantly between both races (r=0.50; p<0.001). The predictive model (p<0.001) could predict 32.2% of the finish TGI in the second race (vs. 17.1% without finish TGI in race 1).

Conclusion: Our findings demonstrate that the use of previously obtained thermoregulatory responses results in higher predictability of finish core body temperatures in future races, enabling better risk assessment for those athletes that are most likely to benefit from preventive measures.

Introduction

An elevated core body temperature (CBT) is commonly observed in athletes performing exercise and does not typically affect health or performance [1, 3]. The CBT rise is caused by the production of metabolic heat in the exercising muscle, which cannot be completely released to the environment [15]. If heat storage becomes uncompensable, athletes reduce their performance levels in anticipation of the ensuing CBT rise [24]. Interestingly, the maximal CBT that individuals reach during outdoor time trials in cool to moderate conditions varies widely, ranging from 37.3–41.5°C [13, 21, 25]. The variation in thermoregulatory responses has previously been linked to subject characteristics, e.g. age, sex, exercise intensity, body weight, body mass index (BMI), muscle/fat mass [1, 2, 7, 9, 17] and external factors (e.g. ambient temperature, wind speed, humidity) [1, 13, 27]. Predicting exercise-induced CBT rises can help athletes estimate their maximal CBT during race conditions. We demonstrated in a previous study that age, BMI, fluid intake before the race and the core body temperature change during warming-up are the primary predictors for maximal gastrointestinal temperature (TGI) in a 15-km road race under cool ambient conditions [25]. Nevertheless, the combination of these within-subject and external parameters could only predict 16.7% of finish TGI. Previous studies revealed that a history of heat illness is an independent risk factor for a future repeated event [1, 7, 17]. These findings suggest that the magnitude of exercise-induced TGI rises might be related to individually determined intrinsic factors. This would mean that, under exactly the same external conditions and with no changes in within-subject characteristics, one athlete would consistently demonstrate low CBT rises whereas another athlete will consistently demonstrate large CBT changes upon repeated equal bouts of exercise. Whether such consistent within-subject thermoregulatory responses exist in the athletic populations is currently unknown. Therefore, the aim of this study was to assess the within-subject variation of thermoregulatory responses...
responses during 2 consecutive equal exercise bouts. Secondly, we explored whether including T$_{\text{CI}}$ data from the first race edition could improve the predictability of the thermoregulatory responses during the second race edition. For these purposes, we performed T$_{\text{CI}}$ measurements in 58 participants of a 15-km running event during 2 consecutive race editions, which were held under similar environmental conditions. We hypothesized that T$_{\text{CI}}$ would strongly correlate between both exercise bouts and could improve the prediction of finish T$_{\text{CI}}$ in a subsequent race.

Materials & Methods

We recruited 58 individuals (Table 1) who participated in 2 consecutive editions of a 15-km running event (Seven Hills Run, Nijmegen, the Netherlands; organized –1 year apart). Before being included in the study, all subjects provided their written informed consent and were screened for the presence of any exclusion criteria for using the temperature capsule. The exclusion criteria were: 1. A history of obstructive or inflammatory bowel disease or prior abdominal surgery, 2. The presence of any implanted electric device, 3. A scheduled MRI scan within 1 week after the event, or 4. Pregnancy. Study procedures were approved by the Radboud University Medical Centre Ethics Committee and were in accordance with the principles of the Declaration of Helsinki. This study was conducted in compliance with the ethical standards according to Harriss et al. [16]. Study procedures and measurements were identical in both race editions. Baseline measurements were performed 2 h before the start of the race in a laboratory set up 50 m from the finish line. T$_{\text{CI}}$ was measured at baseline, 1 min before the start (i.e. after warm-up), and within 15 s after finishing. No measurements were performed during exercise, and subjects were allowed to complete the race at a self-selected pace with ad libitum fluid intake. Body height and weight (Seca 888 calibrated scale; Hamburg, Germany) were measured at baseline. Body mass index (BMI) and body surface area were calculated using the height and weight data. Body-surface area was calculated using the formula of DuBois et al. [12].

Subjects ingested an individually calibrated telemetric temperature capsule at least 5 h (8 a.m.) before the race (start 1 p.m.) to prevent interaction of the T$_{\text{CI}}$ measurements with fluid ingestion during testing [28]. T$_{\text{CI}}$ was measured using a portable telemetry system (CorTemp™ system, HQ Inc., Palmetto, USA), which has been demonstrated to safely and reliably measure T$_{\text{CI}}$ as indicator of the subject’s CBT [6,14]. The average of 3 consecutive measurements for each time point was used for further analyses. The T$_{\text{CI}}$ rise between baseline and finish was calculated by subtracting the T$_{\text{CI}}$ at baseline from the T$_{\text{CI}}$ at the finish line.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Subject characteristics in both race editions.</th>
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<tbody>
<tr>
<td></td>
<td>Race edition 1</td>
</tr>
<tr>
<td>sex (male: female)</td>
<td>31: 28</td>
</tr>
<tr>
<td>age (years)</td>
<td>47±10</td>
</tr>
<tr>
<td>height (cm)</td>
<td>175±8</td>
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<tr>
<td>weight (kg)</td>
<td>73.0±12.4</td>
</tr>
<tr>
<td>body mass index (kg/m²)</td>
<td>23.6±2.7</td>
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<tr>
<td>body surface area (m²)</td>
<td>1.88±0.19</td>
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* Age during race edition 1 is reported
* P-value refers to a Wilcoxon signed rank test

Subjects self-reported the amount of fluid intake from the time of getting out of bed until the end of the race. Body weight was measured at baseline and within 10 min after the race, from which the relative change in body weight was calculated (expressed as percentage dehydration). Correction for fluid intake during the race was applied by adding the amount of fluids consumed to the baseline body weight and recalculating the body weight change. Subjects were allowed to drink ad libitum before as well as during the race. No restrictions were imposed on the type of fluids consumed, though subjects were requested to refrain from drinking between finishing and the second body weight measurement to avoid overestimating the post-race body weight.

Individual finish times after 15-km were obtained from the organizational measuring system (ChampionChip®, MYLAPS, Nijmegen, the Netherlands), and running speed was calculated accordingly.

Wet-bulb Globe Temperature (WBGT) was measured every 30 min throughout the day using a portable climate-monitoring device (Davis Instruments Inc., Hayward, USA) positioned in the start/finish area.

Statistical analyses were performed using the Statistical Package for the Social Sciences (IBM SPSS Statistics for Windows, Version 20.0. IBM Corp., Armonk, NY, USA). Data was reported as mean±standard deviation, with the significance level set at p<0.05. Normality distribution was examined using a Kolmogorov-Smirnov test. In case of non-Gaussian distribution, log-transformation was performed and the data was re-examined for normal distribution. If normal distribution could not be attained, non-parametric tests were applied. Differences in subject and exercise characteristics between the race editions were analysed using a Student’s t-tests. For study aim 1, a repeated measures ANOVA was used to determine whether thermoregulatory responses were comparable between race edition 1 and 2. Coefficients of variation expressed as percentage (CoV) were determined for each individual subject to gain more insight on the individual variation of thermoregulatory responses and race speed between both race editions. Subsequently, a Pearson correlation was used to determine the consistency of finish T$_{\text{CI}}$ and the exercise-induced T$_{\text{CI}}$ elevation. For study aim 2 we performed a linear regression analysis with finish T$_{\text{CI}}$ in race edition 2 as the dependent variable, while age, BMI, fluid intake before the race, T$_{\text{CI}}$ change during warm-up (original model) and supplemented with finish T$_{\text{CI}}$ in race edition 1 served as independent parameters [25]. To correct for large within-subject differences of metabolic heat production (e.g. race speed) [9,21], we performed additional analyses in which we excluded subjects who showed a >5% difference in race speed between both editions. Finally we created 3 dummy parameters (T$_{\text{CI}}$≥39.0°C (yes/no), T$_{\text{CI}}$≥39.5°C (yes/no), T$_{\text{CI}}$≥40.0°C (yes/no)) for both race editions to explore the risk of exceeding these T$_{\text{CI}}$ thresholds in the 2 consecutive road races. A Pearson’s Chi Square test was used to calculate Relative Risks (RR) and their 95% confidence intervals (CI).

Results

Subject characteristics (i.e. baseline body weight, BMI and body surface area) did not differ between race 1 and 2 (Table 1). All subjects successfully completed both races at comparable running speeds (11.8±1.9 km/h vs. 11.7±1.9 km/h, range 8.1–
16.5 km/h; p = 0.78; CoV 3 ± 3%). Environmental conditions were cool and comparable between race edition 1 (WBGT 11 °C, TDRY-BULB 10.5 °C, relative humidity 87%, wind speed 3.4–5.4 m/s) and race edition 2 (WBGT 12.5 °C, TDRY-BULB 11.5 °C, relative humidity 88%, wind speed 3.4–7.9 m/s). Pre-race fluid intake was not different between either race edition (1147 ± 448 mL vs. 1095 ± 444 mL; p = 0.25), whereas fluid intake during the races was higher in race edition 2 vs. 1 (129 ± 146 mL vs. 85 ± 134 mL; p = 0.02). Nevertheless, the percentage body weight loss was not different between either race (-1.6 ± 0.6% vs. -1.5 ± 0.5%; TGI p = 0.25).

Lastly, runners who demonstrated a finish TGI lower than 39.0 °C in race 1. Likewise, runners with a finish TGI ≥ 39.0 °C and ≥ 40.0 °C in race edition 1 (x-axis) and race edition 2 (y-axis; Fig. 2a) and the exercise-induced TGI increase (Spearman's r = 0.40, p = 0.002; Fig. 2b) correlated significantly between both races. Correction for subjects with a > 5% (n = 14) difference in race speed between both race editions improved the correlation of finish TGI between race edition 1 and 2 (Pearson's r = 0.59, p < 0.001). Lastly, a linear regression analysis revealed that the higher fluid intake in race 2 did not significantly influence TGI at the finish line in race 2 (R² = 0.00; p = 0.87). Excluding subjects that consumed < 0.5L of fluids 4h prior to the exercise bout (n = 6) and re-analysing the data did not affect the correlation of finish TGI (r = 0.48, p < 0.001).

By applying our original linear regression model to the present subject population we were able to predict 17.1% (F-score 2.58, p < 0.05) of the finish TGI of race edition 2 (Table 2). Supplementing the model with finish TGI of race edition 1 as an independent variable resulted in a higher predictive capacity of the regression model (R² = 0.32, F-score 4.66, p = 0.001; Table 2). Interestingly, correction for subjects with a > 5% difference in race speed resulted in an even stronger predictive model (R² = 0.47, p < 0.001). Lastly, reanalysing our data after exclusion of subjects that consumed < 0.5L of fluids 4h prior to exercise did not affect our predictive model (R² = 0.31, p < 0.01).

This study assessed the within-subject variation of thermoregulatory responses in athletes participating in 2 consecutive editions of a 15-km road race under comparable environmental conditions. Our results demonstrate that TGI was not different across the 2 exercise bouts at baseline, start and finish, and show that both finish TGI (r = 0.50) as well as the exercise-induced TGI increase (r = 0.40) correlated significantly between the 2 race editions. Moreover, by supplementing our predictive model with the finish TGI from the first race edition, we improved the predictive capacity of finish TGI from 17.1 to 32.2%. Lastly, our results demonstrate that the chance of attaining a high TGI was significantly greater if that subject demonstrated previous high TGI (r = 0.50) as well as the exercise-induced TGI increase (r = 0.40) correlated significantly between the 2 race editions. Moreover, by supplementing our predictive model with the finish TGI from the first race edition, we improved the predictive capacity of finish TGI from 17.1 to 32.2%. Lastly, our results demonstrate that the chance of attaining a high TGI was significantly greater if that subject demonstrated previous high TGI (r = 0.50).
exercise-induced thermoregulatory responses (relative risk varying from 3.7 to 6.5). These results suggest that CBT responses are not different within subjects over consecutive exercise bouts. Therefore, individual CBT data are valuable for improving the predictability of exercise-induced thermoregulatory responses and identifying which athletes are most likely to benefit from cooling strategies.

To our knowledge, this is the first study to directly compare and correlate TGI in the same subjects performing 2 similar exercise bouts without applying any intervention. Previous studies that measured TGI during repeated exercise bouts reported variable results, but are difficult to compare to the present study as they all imposed different kinds of potentially confounding interventions, including diurnal variation [18], variable environmental conditions [11], variable heat load [10] or variable exercise protocols [22]. By performing measurements in the same subjects who twice completed the same 15-km run under similar conditions, we were able to directly compare thermoregulatory responses while limiting the chance of confounders. Indeed, our results showed that BMI [26], running speed [20] and hydration status [8], which are known to influence CBT during exercise, were all similar across both exercise bouts and are therefore unlikely to have influenced our results. Although fluid intake during the race was significantly higher in the second exercise bout (129 ± 146 mL vs. 85 ± 134 mL), absolute differences between race editions were small (44 ± 150 mL), body weight changes were comparable (−1.6 ± 0.6 vs. −1.5 ± 0.5 % of total body weight), and regression analysis showed no impact of fluid intake on finish TGI. To summarize, the significant correlations of finish TGI (r = 0.50) and TGI increase (r = 0.40) between both race editions suggest that the correlation of CBT at the finish line between two 15-km road races is moderate, while the coefficients of variation are low within subjects.

Our model that demonstrated a 17.1 % predictive capacity for finish TGI confirms previous findings (16.7 % predictive capacity in a different study cohort) [25]. By adding the finish TGI from race 1 to this model to predict finish TGI in race 2, we were able to improve the predictive capacity from 17.1 to 32.2 %. Interestingly, correcting our model for changes in exercise intensity (<5 % difference in finish time between race 1 and 2), further improved the predictability of finish TGI (R² = 0.47). Furthermore, we demonstrated that individuals who developed a finish TGI ≥ 39.0 °C during the first edition were 3.7 times more likely to attain a similar or higher TGI during a second exercise bout compared to subjects who finished with a TGI ≤ 39.0 °C. This likelihood was even greater if higher cut-off values were chosen. Subjects finishing with a TGI ≥ 40.0 °C were 6.0 times more likely to exceed this threshold again during a subsequent race. These findings may help to identify athletes who benefit from cooling interventions prior to and during exercise [4].

The limited variation of exercise-induced TGI responses within subjects in combination with the large variation in thermoregulatory responses between subjects (TGI increase ranging 0.4–3.6 °C) raises questions regarding the underlying mechanisms that are responsible for this observation. In addition to anthropometric factors such as age [7], sex [17] and BMI [2, 7], inherited intrinsic factors might play an important role. For example, several genes have been linked to the development of heat illness [7]. It is currently unknown whether the genetic variation also affects thermoregulatory responses and/or the capacity of heat dissipating mechanisms. Likewise, there is evidence that CBT responses are related to exercise-induced changes of the hypothalamic setpoint [5, 19]. The ‘high-responders’ in our study may have potentially demonstrated a larger increase in the CBT setpoint compared to the ‘low-responders’. Since our study did not include measurements of these intrinsic factors, future studies focussing on the potential underlying mechanisms are warranted.

This study was limited by the fact that we did not measure hydration status prior to the start of the exercise, which could mask differences in hydration status between both exercise bouts. However, previous literature recommended that the consumption of −0.5 L of fluids 4 h prior to exercise should ensure euhydration at the start of the exercise [23]. While 52 subjects met this criterion, 6 subjects did not. Reanalysis of our data without these subjects did not affect the correlation of finish TGI or our predictive model. We therefore believe that differences in hydration status did not impact on our findings. Furthermore, this study was also limited by the fact that both race editions were separated by a ~1 year time span. This could have potentially lead to the occurrence of within-subject differences that could not have been accounted for (e.g. changes in health status, training status, etc.), possibly resulting in a suboptimal comparison between finish TGI in both editions. Nevertheless, we still found a significant correlation of 0.50 in finish TGI between both races. Therefore, given that a smaller time span between both exercise bouts might have resulted in a higher correlation, our results likely only underestimate the actual within-subject variation of thermoregulatory responses.

In conclusion, exercise-induced thermoregulatory responses significantly correlated within subjects performing 2 consecutive conditions of a 15-km road race under cool environmental conditions, demonstrated a moderate within-subject variability and a low coefficient of variation. Athletes who showed a finish TGI ≥ 40.0 °C were 6.0 times more likely to exceed this threshold again during a subsequent race. More importantly, the use of previously obtained thermoregulatory responses improves the predictability of finish core body temperatures in future races. Our findings enable identification of athletes who are the most likely to benefit from cooling interventions preceding and during exercise.
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Conflict of interest: The authors have no conflict of interest to declare.

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