

Within-subject Variation of Thermoregulatory Responses during Repeated Exercise Bouts

Authors

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Key word

- heatstroke
- heat stress disorders
- body temperature regulation
- body temperature
- exercise physiology

Abstract



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

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

Results: Under cool conditions (WBGT 11–12°C), T_{GI} was comparable between both races at baseline ($37.6 \pm 0.4^\circ\text{C}$ vs. $37.9 \pm 0.4^\circ\text{C}$; $p=0.24$) and finish ($39.4 \pm 0.6^\circ\text{C}$ vs. $39.4 \pm 0.6^\circ\text{C}$; $p=0.83$). Finish T_{GI} 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 (T_{GI}) 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 T_{GI} . 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 T_{GI} 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

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responses during 2 consecutive equal exercise bouts. Secondly, we explored whether including T_{Cl} 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_{Cl} 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_{Cl} would strongly correlate between both exercise bouts and could improve the prediction of finish T_{Cl} in a subsequent race.

Materials & Methods

We recruited 58 individuals (see 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_{Cl} was measured at baseline, 1 min before the start (i.e. after warming-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_{Cl} measurements with fluid ingestion during testing [28]. T_{Cl} was measured using a portable telemetry system (CorTemp™ system, HQ Inc., Palmetto, USA), which has been demonstrated to safely and reliably measure T_{Cl} 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_{Cl} rise between baseline and finish was calculated by subtracting the T_{Cl} at baseline from the T_{Cl} at the finish line.

Table 1 Subject characteristics in both race editions.

	Race edition 1	Race edition 2	P-value
sex (male: female)	31: 28		–
age (years) [#]	47 ± 10		–
height (cm)	175 ± 8		–
weight (kg)	73.0 ± 12.4	73.0 ± 12.3	0.71 *
body mass index (kg/m ²)	23.6 ± 2.7	23.7 ± 2.8	0.75
body surface area (m ²)	1.88 ± 0.19	1.88 ± 0.19	1.00

[#] 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 $p \leq 0.05$. 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 measurements 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_{Cl} and the exercise-induced T_{Cl} elevation. For study aim 2 we performed a linear regression analysis with finish T_{Cl} in race edition 2 as the dependent variable, while age, BMI, fluid intake before the race, T_{Cl} change during warming-up (original model) and supplemented with finish T_{Cl} 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 ($T_{\text{Cl}} \geq 39.0^\circ\text{C}$ (yes/no), $T_{\text{Cl}} \geq 39.5^\circ\text{C}$ (yes/no), $T_{\text{Cl}} \geq 40.0^\circ\text{C}$ (yes/no)) for both race editions to explore the risk of exceeding these T_{Cl} 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 (see 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 $3\pm 3\%$). Environmental conditions were cool and comparable between race edition 1 (WBGT 11°C , $T_{\text{DRY-BULB}}$ 10.5°C , relative humidity 87%, wind speed 3.4–5.4 m/s) and race edition 2 (WBGT 12.5°C , $T_{\text{DRY-BULB}}$ 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 ($1147\pm 448\text{ mL}$ vs. $1095\pm 444\text{ mL}$; $p=0.25$), whereas fluid intake during the races was higher in race edition 2 vs. 1 ($129\pm 146\text{ mL}$ vs. $85\pm 134\text{ mL}$; $p=0.02$). Nevertheless, the percentage body weight loss was not different between either race ($-1.6\pm 0.6\%$ vs. $-1.5\pm 0.5\%$; $p=0.25$).

T_{GI} was not different at baseline ($37.6\pm 0.4^\circ\text{C}$ vs. $37.7\pm 0.4^\circ\text{C}$; $p=0.24$; Cov $1\pm 1\%$; \bullet Fig. 1), before the start ($37.8\pm 0.4^\circ\text{C}$ vs. $37.9\pm 0.5^\circ\text{C}$; $p=0.28$; CoV $1\pm 1\%$) and immediately after finishing ($39.4\pm 0.6^\circ\text{C}$ vs. $39.4\pm 0.6^\circ\text{C}$; $p=0.83$; CoV $1\pm 1\%$), and demonstrated no difference in exercise-induced T_{GI} increase in either race edition ($1.9\pm 0.8^\circ\text{C}$ vs. $1.8\pm 0.8^\circ\text{C}$; $p=0.58$). Finish T_{GI} (Pearson's $r=0.50$, $p<0.001$; \bullet Fig. 2a) and the exercise-induced increase in T_{GI} (Spearman's $r=0.40$, $p=0.002$; \bullet 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 T_{GI} 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 T_{GI} at the finish line in race 2 ($R^2=0.00$; $p=0.87$). Excluding subjects that consumed $<0.5\text{ L}$ of fluids 4 h prior to the exercise bout ($n=6$) and re-analysing the data did not affect the correlation of finish T_{GI} ($r=0.48$, $p<0.001$).

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 T_{GI} of race edition 2 (\bullet Table 2). Supplementing the model with finish T_{GI} of race edition 1 as an independent variable resulted in a higher predictive capacity of the regression model ($R^2=0.32$, F-score 4.66, $p=0.001$; \bullet Table 2). Interestingly, correction for subjects with a $>5\%$ difference in race speed resulted in an even stronger predictive model ($R^2=0.47$, $p<0.001$). Lastly, reanalysing our data after exclusion of subjects that consumed $<0.5\text{ L}$ of fluids 4 h prior to exercise did not affect our predictive model ($R^2=0.31$, $p<0.01$).

Lastly, runners who demonstrated a finish $T_{\text{GI}} \geq 39.0^\circ\text{C}$ in race 1, had a 3.7 times greater chance (CI: 1.0–14.0) of exceeding this T_{GI} threshold again in race edition 2 compared to athletes who had a finish T_{GI} lower than 39.0°C in race 1. Likewise, runners with a finish $T_{\text{GI}} \geq 39.5^\circ\text{C}$ and $\geq 40.0^\circ\text{C}$ in race edition 1 had an elevated risk of exceeding these T_{GI} levels again in race edition 2 (RR: 6.5, CI: 2.0–21.0 and RR: 6.0, CI: 1.5–24.5 respectively).

Discussion

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 T_{GI} was not different across the 2 exercise bouts at baseline, start and finish, and show that both finish T_{GI} ($r=0.50$) as well as the exercise-induced T_{GI} increase ($r=0.40$) correlated significantly between the 2 race editions. Moreover, by supplementing our predictive model with the finish T_{GI} from the first race edition, we improved the predictive capacity of finish T_{GI} from 17.1 to 32.2%. Lastly, our results demonstrate that the chance of attaining a high T_{GI} was significantly greater if that subject demonstrated previous high

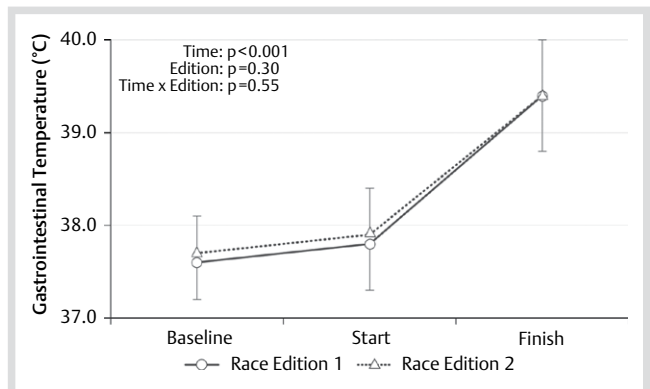


Fig. 1 Gastrointestinal temperature (T_{GI}) at baseline, 1 min before the start and immediately after finishing in race edition 1 (solid line, circles) and race edition 2 (dotted line, triangles). T_{GI} was not different at all time points ($p=0.30$) and increased significantly over time ($p<0.001$).

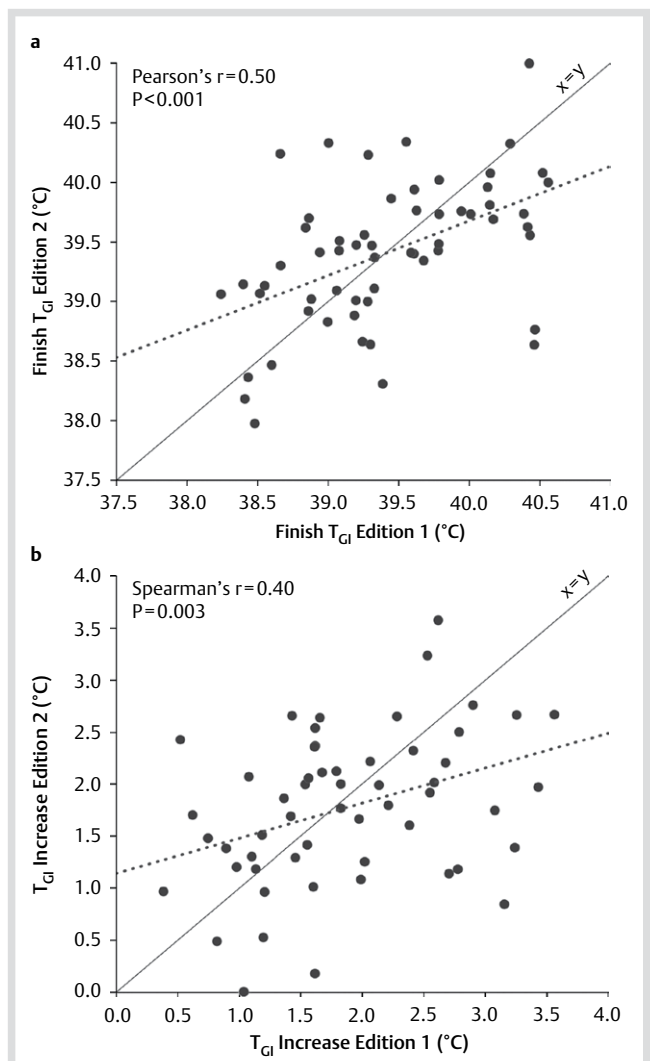


Fig. 2 Correlation between finish gastrointestinal temperature in race edition 1 (x-axis) and race edition 2 (y-axis; a) and correlation between the gastrointestinal temperature increase (baseline to finish) between race edition 1 (x-axis) and race edition 2 (y-axis; b). The regression analysis revealed that gastrointestinal temperature in race edition 1 accounted for 25% of the finish gastrointestinal temperature in race edition 2. The dotted lines refer to the correlation coefficients, while the solid lines refer to the line of identity ($x=y$).

Table 2 Predictor characteristics for finish T_{Cl} of race edition 2 using our previously established predictive model (upper section) and our new model which was supplemented with finish T_{Cl} in race edition 1 as potential predictive factor.

Variable	Linear regression analysis		
	B	95% CI	β
Original model *			
constant	39.5	38.2–40.8	
age	-0.02	-0.03–0.00	-0.29 ^{NS}
BMI	0.04	-0.02–0.10	0.18 ^{NS}
T_{Cl} rise after warming-up	0.30	0.05–0.54	0.31 ^B
fluid intake before race	0.00	-0.00–0.00	-0.18 ^{NS}
New model **			
constant	25.0	16.1–33.9	
age	-0.01	-0.03–0.00	-0.21 ^{NS}
BMI	0.03	-0.03–0.08	0.14 ^{NS}
T_{Cl} rise after warming-up	0.19	-0.05–0.42	0.20 ^{NS}
fluid intake before race	0.00	-0.00–0.00	-0.17 ^{NS}
finish T_{Cl} race 1	0.37	0.15–0.59	0.41 ^A

* R for original model = 0.41; $R^2 = 0.17$; F-score of change: 2.58, $p < 0.05$ / ** R for new model = 0.57; $R^2 = 0.32$; F-score of change: 4.66, $p = 0.001$

^A $p < 0.005$; ^B $p < 0.05$; ^{NS} not significant

CI = confidence interval; β = standardized B

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 T_{Cl} in the same subjects performing 2 similar exercise bouts without applying any intervention. Previous studies that measured T_{Cl} 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 T_{Cl} . To summarize, the significant correlations of finish T_{Cl} ($r = 0.50$) and T_{Cl} 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 T_{Cl} confirms previous findings (16.7 % predictive capacity in a different study cohort) [25]. By adding the finish T_{Cl} from race 1 to this model to predict finish T_{Cl} 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 T_{Cl} ($R^2 = 0.47$). Furthermore, we demonstrated that individuals who developed a finish $T_{\text{Cl}} \geq 39.0^\circ\text{C}$ during the first edition were 3.7 times more likely to attain a similar or higher T_{Cl} during a second exercise bout compared to subjects who finished with a $T_{\text{Cl}} \leq 39.0^\circ\text{C}$. This likelihood was even greater if higher cut-off values were chosen. Subjects finishing with a $T_{\text{Cl}} \geq 40.0^\circ\text{C}$ 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 T_{Cl} responses within subjects in combination with the large variation in thermoregulatory responses between subjects (T_{Cl} increase ranging 0.4–3.6 $^\circ\text{C}$) 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 ~0.5L of fluids 4h 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 T_{Cl} 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 T_{Cl} in both editions. Nevertheless, we still found a significant correlation of 0.50 in finish T_{Cl} 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 $T_{\text{Cl}} \geq 40.0^\circ\text{C}$ 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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