Within-Subject Variation of Thermoregulatory Responses During Repeated Exercise Bouts

Matthijs TW Veltmeijer, MD[1]; Dick HJ Thijssen, PhD[1,2]; Maria TE Hopman, MD, PhD[1] ; Thijs MH Eijsvogels, PhD[1,3]

[1] Department of Physiology, Radboud University Medical Center, Nijmegen, the Netherlands;

[2] Research Institute for Sport and Exercise Science, Liverpool John Moores University, Liverpool, United Kingdom;

[3] Henry Low Heart Centre, Department of Cardiology, Hartford Hospital, Hartford, USA

Total Word Count: 2711

Word Count Abstract: 190

Total Number of Figures: 2

Total Number of Tables: 2

Key-words: Heatstroke, Heat Stress Disorders, Body Temperature Regulation, Body Temperature, Exercise Physiology

Author for correspondence:

Dr. Thijs Eijsvogels, Dept. of Physiology (392), Radboud University Medical Center, PO Box 9101, 6500 HB Nijmegen, the Netherlands. E-mail: Thijs.Eijsvogels@Radboudumc.nl, Telephone: +31 24 361 3426, Fax: N/A.

ABSTRACT

Aim: To assess the within-subject variation of thermoregulatory responses during two 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±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 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 T_{GI} in the second race (versus 17.1% without finish T_{GI} 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 to 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 small CBT changes upon repeated equal bouts of exercise. Whether such consistent within individual 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 during two consecutive equal exercise bouts. Secondly, we explored whether including T_{GI} data from the first race edition could improve the predictability if the thermoregulatory responses during the second race edition. For these purposes, we performed T_{GI} measurements in 58 participants of a 15-km running event during two consecutive race editions, which were held under similar environmental conditions. We hypothesized that T_{GI} would strongly correlate between both exercise bouts and could improve the prediction of finish T_{GI} in a subsequent race.

Materials & Methods

We recruited 58 individuals (Table 1) that participated in two 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 a written informed consent and all subjects were screened for the presence of any exclusion criteria for using the temperature capsule: 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 accorded to the principles of the Declaration of Helsinki. This study was conducted in agreement 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 hours before the start of the race in a laboratory set up 50 meters from the finish line. T_{GI} was measured at baseline, 1 minute before the start (i.e. after warming-up), and within 15 seconds 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 five hours (8 a.m.) before the race (start 1 p.m.) to prevent interaction of the T_{GI} measurements with fluid ingestion during testing [28]. T_{GI} was measured using a portable telemetry system (CorTemp™ system, HQ Inc., Palmetto, USA), which has been demonstrated to safely and reliably measure T_{GI} as indicator of the subject's CBT [6,14]. The average of three consecutive measurements for each time point was used for further analyses. The T_{GI} rise between baseline and finish was calculated by subtracting the T_{GI} at baseline from the T_{GI} at the finish line.

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 minutes 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 minutes 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 was 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 normality 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 if 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 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_{GI} and the exercise-induced T_{GI}

elevation. For study aim 2 we performed a linear regression analysis with finish T_{GI} in race edition 2 as the dependent variable, and age, BMI, fluid intake before the race, T_{GI} change during warming-up (original model) and supplemented it with finish T_{GI} in race edition 1 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 that showed a >5% difference in race speed between both editions. Finally we created 3 dummy parameters (T_{GI} ≥39.0°C (yes/no), T_{GI} ≥39.5°C (yes/no), T_{GI} ≥40.0°C (yes/no)) for both race editions to explore the risk for exceeding these T_{GI} thresholds in the two 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 *versus* 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, $T_{DRY-BULB}$ 10.5°C, relative humidity 87%, wind speed 3.4–5.4 m/s) and race edition 2 (WBGT 12.5°C, $T_{DRY-BULB}$ 11.5°C, relative humidity 88%, wind speed 3.4–7.9 m/s). Pre-race fluid intake was not different between both race editions (1147±448 mL *versus* 1095±444 mL; p=0.25), whereas fluid intake during the races was higher in race edition 2 *versus* 1 (129±146 mL *versus* 85±134 mL; p=0.02). Nevertheless, the percentage body weight loss was not different between both races (-1.6±0.6% *versus* -1.5±0.5%; p=0.25).

T_{GI} was not different at baseline (37.6±0.4°C *versus* 37.7±0.4°C; p=0.24; Cov 1±1%, Figure 1), before the start (37.8±0.4°C *versus* 37.9±0.5°C; p=0.28; CoV 1±1%) and immediately after finishing (39.4±0.6°C *versus* 39.4±0.6°C; p=0.83; CoV 1±1%), and demonstrated no difference in exercise-induced T_{GI} increase in both race editions (1.9±0.8°C *versus* 1.8±0.8°C; p=0.58). Finish T_{GI} (Pearson's r=0.50, p<0.001; Figure 2A) and the exercise-induced increase in T_{GI} (Spearman's r=0.40, p=0.002; Figure 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 <0.5L of fluids 4 hours 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 (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; 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, re-analysing our data after exclusion of subjects that consumed <0.5L of fluids 4 hours prior to exercise did not affect our predictive model (R^2=0.31, p<0.01).

Lastly, runners that demonstrated a finish T_{GI} ≥39.0°C in race 1, had a 3.7 times larger chance (CI: 1.0 – 14.0) to exceed 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_{GI}≥39.5°C and ≥40.0°C in race edition 1 had elevated risks to exceed 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 two consecutive editions of a 15-km road race in comparable environmental conditions. Our results demonstrate that T_{GI} was not different across both 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 two 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 larger if that subject demonstrated previous high exercise-induced thermoregulatory responses (relative risk varying from 3.7 – 6.5). These results suggest that CBT responses are not different within subjects over consecutive exercise bouts. Therefore, individual CBT data are valuable to improve the predictability of exercise-induced thermoregulatory responses and to identify which athletes are most likely to benefit from cooling strategies.

To our knowledge, this is the first study to directly compare and correlate T_{GI} in the same subjects performing two similar exercise bouts without applying any intervention. Previous studies that measured T_{GI} 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 whilst 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 will therefore unlikely have influenced our results. Although fluid intake during the race was significantly higher in the second exercise bout (129 ± 146 mL *versus* 85 ± 134 mL), absolute differences between race editions were small (44 ± 150

mL), body weight changes were comparable (-1.6±0.6 *versus* -1.5±0.5% of total body weight), and regression analysis showed no impact of fluid intake on finish T_{GI}. To summarize, the significant correlations of finish T_{GI} (r=0.50) and T_{GI} 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, whilst the coefficients of variation are low within subjects.

Our model that demonstrated a 17.1% predictive capacity for finish T_{GI} confirms previous findings (16.7% predictive capacity in a different study cohort) [25]. By adding the finish T_{GI} from race 1 to this model to predict finish T_{GI} 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_{GI} (R^2=0.47). Furthermore, we demonstrated that individuals, who developed a finish T_{GI} ≥39.0°C during the first edition, were 3.7 times more likely to attain a similar or higher T_{GI} during a second exercise bout compared to subjects who finished with a T_{GI} ≤39.0°C. This chance was even greater if higher cut-off values were chosen; subjects finishing with a T_{GI} ≥40.0°C had a 6.0 times greater chance for exceeding this threshold again during a subsequent race. These findings may help to identify athletes that benefit from cooling interventions preceding and during exercise [4].

The limited variation of exercise-induced T_{GI} responses within subjects, in combination with the large variation in thermoregulatory responses between subjects (T_{GI} 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]. Whether the genetic variation also affects thermoregulatory responses and/or the capacity of heat dissipating mechanisms is currently unknown. Likewise, there is evidence that CBT responses are related to exercise-induced changes of the hypothalamic setpoint [5,19]. Potentially,

the 'high-responders' in our study 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 4 hours prior to exercise should ensure euhydration at the start of the exercise [23]. Whilst 52 subjects met this criterion, 6 subjects did not. Re-analysis of our data without these subjects did not affect the correlation of finish T_{GI} 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_{GI} in both editions. Nevertheless, we still found a significant correlation of 0.50 in finish T_{GI} 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 two 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 that showed a finish $T_{GI} \geq 40.0°C$ had a 6.0 times greater chance for exceeding 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 that are the most likely to benefit from cooling interventions preceding and during exercise.

Acknowledgements: We thank Joan Merx and Ronald Veerbeek of the 'Seven Hills Run' for providing facilities to set up our on-site laboratory. This study was funded by the Department of Physiology of the Radboud University Medical Center.

Dr. Eijsvogels received a Rubicon Grant (825.12.016). Dr. Thijssen is financially supported by the Netherlands Heart Foundation (E. Dekker-stipend 2009T064).

This study was presented during the Aspetar Training and Competing in the Heat Conference in Doha, Qatar (March 24th 2014).

References

1. Armstrong LE, Casa DJ, Millard-Stafford M, Moran DS, Pyne SW, Roberts WO. American College of Sports Medicine position stand. Exertional heat illness during training and competition. Med Sci Sports Exerc 2007; 39: 556-572
2. Bedno SA, Li Y, Han W, Cowan DN, Scott CT, Cavicchia MA, Niebuhr DW. Exertional heat illness among overweight U.S. Army recruits in basic training. Aviat Space Environ Med 2010; 81: 107-111
3. Binkley HM, Beckett J, Casa DJ, Kleiner DM, Plummer PE. National Athletic Trainers' Association Position Statement: Exertional Heat Illnesses. J Athl Train 2002; 37: 329-343
4. Bongers CC, Thijssen DH, Veltmeijer MT, Hopman MT, Eijsvogels TM. Precooling and percooling (cooling during exercise) both improve performance in the heat: a meta-analytical review. Br J Sports Med 2014, DOI: 10.1136/bjsports-2013-092928:
5. Bradford CD, Cotter JD, Thorburn MS, Walker RJ, Gerrard DF. Exercise can be pyrogenic in humans. Am J Physiol Regul Integr Comp Physiol 2007; 292: R143-149
6. Byrne C, Lim CL. The ingestible telemetric body core temperature sensor: a review of validity and exercise applications. Br J Sports Med 2007; 41: 126-133
7. Casa DJ, Armstrong LE, Kenny GP, O'Connor FG, Huggins RA. Exertional heat stroke: new concepts regarding cause and care. Curr Sports Med Rep 2012; 11: 115-123
8. Casa DJ, Clarkson PM, Roberts WO. American College of Sports Medicine roundtable on hydration and physical activity: consensus statements. Curr Sports Med Rep 2005; 4: 115-127
9. Cheuvront SN, Haymes EM. Thermoregulation and marathon running: biological and environmental influences. Sports Med 2001; 31: 743-762
10. Coris EE, Mehra S, Walz SM, Duncanson R, Jennings J, Nugent D, Zwygart K, Pescasio M. Gastrointestinal temperature trends in football linemen during physical exertion under heat stress. South Med J 2009; 102: 569-574

11. Daanen HA, Jonkman AG, Layden JD, Linnane DM, Weller AS. Optimising the acquisition and retention of heat acclimation. Int J Sports Med 2011; 32: 822-828
12. DuBois D DE. A formula to estimate the approximate surface area if height and weight are known. Arch Intern Med 1916; 17: 869-871
13. Ely BR, Ely MR, Cheuvront SN, Kenefick RW, Degroot DW, Montain SJ. Evidence against a 40 degrees C core temperature threshold for fatigue in humans. J Appl Physiol (1985) 2009; 107: 1519-1525
14. Gant N, Atkinson G, Williams C. The validity and reliability of intestinal temperature during intermittent running. Med Sci Sports Exerc 2006; 38: 1926-1931
15. Gonzalez-Alonso J, Quistorff B, Krustrup P, Bangsbo J, Saltin B. Heat production in human skeletal muscle at the onset of intense dynamic exercise. J Physiol 2000; 524 Pt 2: 603-615
16. Harriss DJ, Atkinson G. Ethical standards in sport and exercise science research: 2014 update. Int J Sports Med 2013; 34: 1025-1028
17. Howe AS, Boden BP. Heat-related illness in athletes. Am J Sports Med 2007; 35: 1384-1395
18. Martin L, Doggart AL, Whyte GP. Comparison of physiological responses to morning and evening submaximal running. J Sports Sci 2001; 19: 969-976
19. Mauger AR, Taylor L, Harding C, Wright B, Foster J, Castle P. Acute acetaminophen (paracetamol) ingestion improves time to exhaustion during exercise in the heat. Exp Physiol 2013, DOI: 10.1113/expphysiol.2013.075275:
20. Maughan RJ, Leiper JB, Thompson J. Rectal temperature after marathon running. Br J Sports Med 1985; 19: 192-195
21. Noakes TD, Myburgh KH, du Plessis J, Lang L, Lambert M, van der Riet C, Schall R. Metabolic rate, not percent dehydration, predicts rectal temperature in marathon runners. Med Sci Sports Exerc 1991; 23: 443-449

22. Periard JD, Cramer MN, Chapman PG, Caillaud C, Thompson MW. Neuromuscular function following prolonged intense self-paced exercise in hot climatic conditions. Eur J Appl Physiol 2011; 111: 1561-1569

23. Sawka MN, Burke LM, Eichner ER, Maughan RJ, Montain SJ, Stachenfeld NS. American College of Sports Medicine position stand. Exercise and fluid replacement. Med Sci Sports Exerc 2007; 39: 377-390

24. Tucker R, Rauch L, Harley YX, Noakes TD. Impaired exercise performance in the heat is associated with an anticipatory reduction in skeletal muscle recruitment. Pflugers Arch 2004; 448: 422-430

25. Veltmeijer MT, Eijsvogels TM, Thijssen DH, Hopman MT. Incidence and predictors of exertional hyperthermia after a 15-km road race in cool environmental conditions. J Sci Med Sport 2014, DOI: 10.1016/j.jsams.2014.04.007:

26. Wallace RF, Kriebel D, Punnett L, Wegman DH, Wenger CB, Gardner JW, Gonzalez RR. The effects of continuous hot weather training on risk of exertional heat illness. Med Sci Sports Exerc 2005; 37: 84-90

27. Wendt D, van Loon LJ, Lichtenbelt WD. Thermoregulation during exercise in the heat: strategies for maintaining health and performance. Sports Med 2007; 37: 669-682

28. Wilkinson DM, Carter JM, Richmond VL, Blacker SD, Rayson MP. The effect of cool water ingestion on gastrointestinal pill temperature. Med Sci Sports Exerc 2008; 40: 523-528

FIGURE AND TABLE LEGENDS

Figure 1: Gastrointestinal temperature (T_{GI}) at baseline, 1 minute 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 ($p<0.001$).

Figure 2: Correlation between finish gastrointestinal temperature in race edition 1 (x-axis) and race edition 2 (y-axis; Figure 2A) and correlation between the gastrointestinal temperature increase (baseline to finish) between race edition 1 (x-axis) and race edition 2 (y-axis; Figure 2B). 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 and the solid lines refer to the line of identity ($x = y$).

Table 1: Subject characteristics in both race editions.

Table 2: Predictor characteristics for finish gastrointestinal temperature of race edition 2 using our previously established predictive model (upper section) and our new model, which was supplemented with finish gastrointestinal temperature in race edition 1 as potential predictive factor.

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

Values are presented as mean ± standard deviation

[#] Age during race edition 1 is reported.

* P-value refers to a Wilcoxon Signed Rank test.

Table 2: Predictor characteristics for finish CBT of race edition 2

Variable	**Univariate Analysis**			**Multivariate Analysis***		
	B	95% CI	β	B	95% CI	β
Constant				25.0	16.1 – 33.9	
Age	-0.01	-0.03 – 0.01	-0.15 [NS]	-0.01	-0.03 – 0.00	-0.21 [NS]
BMI	0.01	-0.05 – 0.08	0.06 [NS]	0.03	-0.03 – 0.08	0.14 [NS]
CBT rise after warming-up	0.26	0.01 – 0.51	0.27 [C]	0.19	-0.05 – 0.42	0.20 [NS]
Fluid intake before race	0.00	-0.00 – 0.00	-0.19 [NS]	0.00	-0.00 - 0.00	-0.17 [NS]
Finish CBT race 1	0.49	0.26 – 0.72	0.49 [A]	0.67	0.15 – 0.59	0.41 [B]

* R for model = 0.57; R^2 = 0.32

[A] p <0.001; [B] p <0.005; [C] p <0.05; [NS] not significant

CI = confidence interval; β = standardized B

Figure 1

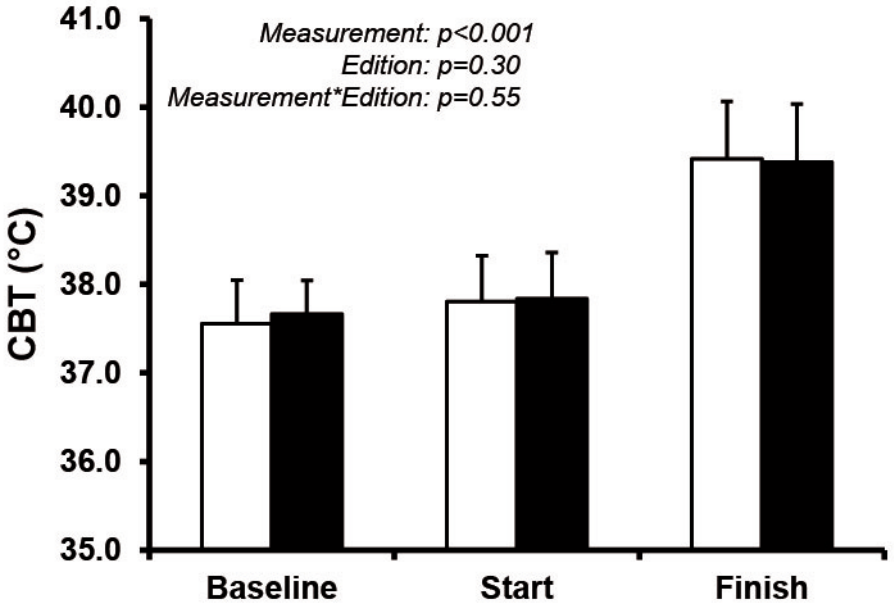
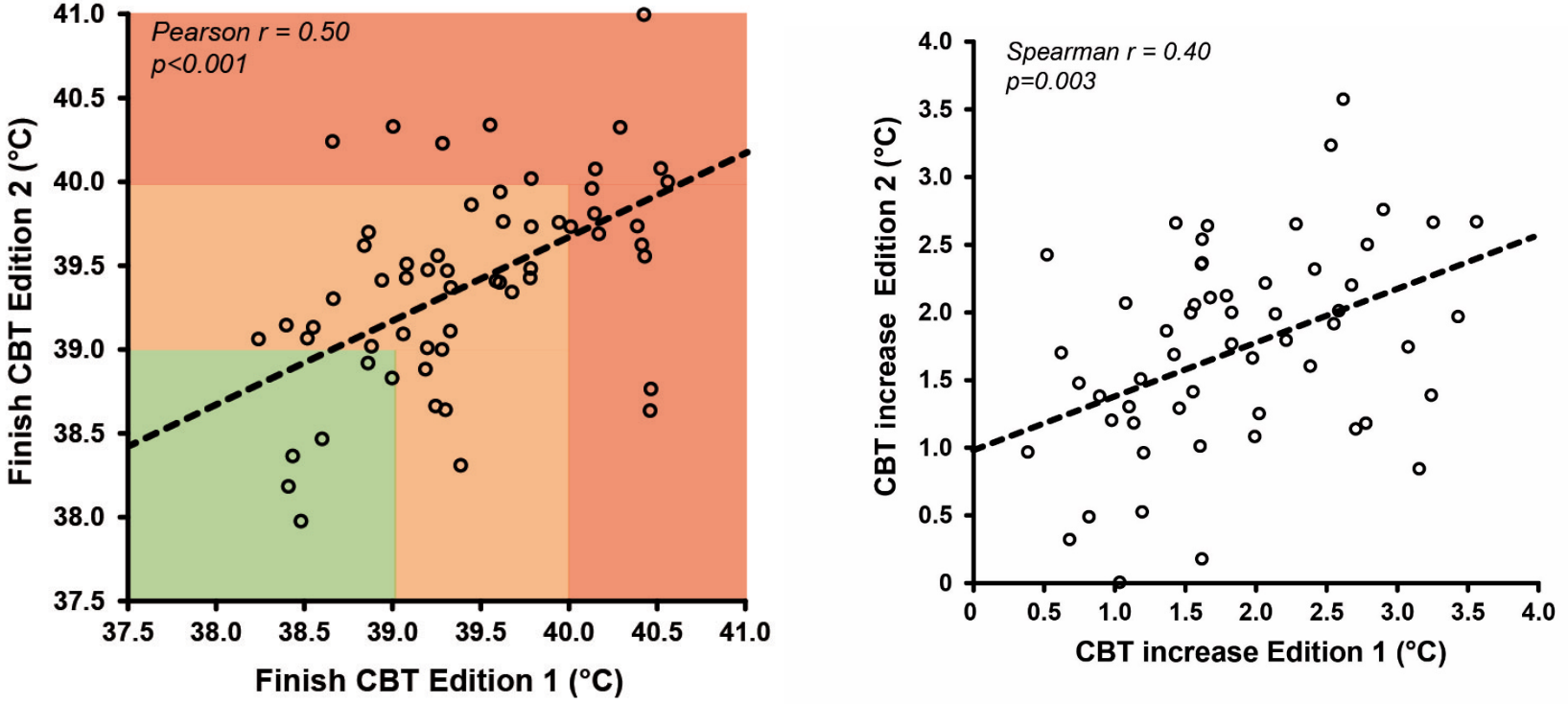

Measurement: p<0.001
Edition: p=0.30
Measurement*Edition: p=0.55
CBT (°C)
41.0
40.0
39.0
38.0
37.0
36.0
35.0
Baseline
Start
Finish

Figure 2


Pearson r = 0.50
p<0.001
Finish CBT Edition 2 (°C)
Finish CBT Edition 1 (°C)
41.0
40.5
40.0
39.5
39.0
38.5
38.0
37.5
37.5
38.0
38.5
39.0
39.5
40.0
40.5
41.0
Spearman r = 0.40
p=0.003
CBT increase Edition 2 (°C)
CBT increase Edition 1 (°C)
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0
0
0.5
1.0
1.5
2.0
2.5
3.0
3.5
4.0