Thermoregulatory Responses of Runners following a Warm-Weather Road Race

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Thermoregulatory Responses of Runners following a Warm-Weather Road Race

Thesis by

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In partial fulfillment of the requirements for the University Scholars Program and the Degree of

Bachelor’s of Science with Honors in Athletic Training

Department of Kinesiology

University of Connecticut

Storrs, CT

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Exercise in the heat presents a significant challenge to the body’s systems, especially when that exercise is in a competitive context. When environmental stress and exercise intensity overcome the body’s dissipation methods, an unsafe increase in body temperature can lead to exertional heat illnesses (EHI). This broad category of outcomes can range from the mild, heat syncope, to the potentially fatal, exertional heat stroke (EHS). The manifestation and severity of EHI is affected by both intrinsic and extrinsic factors within and beyond the athlete’s control.

Survival from EHS is dependent on accurate detection and rapid treatment. The diagnosis of EHS is based on two criteria 1) a body temperature greater than 40°C (40.5°C is alternatively used in some recent publications) and 2) central nervous system dysfunction. Therefore, a clinician’s decision to use a proven method of body temperature assessment is the first step in ensuring a patient’s survival. Subsequently rapidly cooling the EHS patient, through a proven modality such as cold-water immersion, is the primary deciding factor of the event’s outcome.

Together these two steps have ensured a 100% survival rate in documented cases. However, myriad misconceptions currently confound a clinician’s treatment decisions that otherwise would lead to survival without sequelae. Many clinicians choose inappropriate methods of temperature evaluation when assessing potential EHS, either due to an anxiety of rectal temperature measurement or misunderstanding of the validity of other devices. Ensuring that clinicians have knowledge of and access to appropriate methods of body temperature assessment can help ensure suitable treatment decisions, and ultimately protect individuals exercising in the heat.
**Detection**

When detected early and treated properly, survival from EHS is highly likely.\(^2,3\) Currently, rectal, oesophageal or gastrointestinal thermometry are the only valid measures of body temperature during exercise in the heat.\(^2\) However, the invasive nature of these methods, and a misunderstanding of how exercise affects the validity of other thermometers, deters some clinicians from correctly assessing body temperature of a potential EHS patient.\(^5,6\) The development of non-invasive technologies and techniques for the detection of EHI, such as thermal imaging, could help alleviate some of these clinicians’ inhibitions and allow for proper diagnosis and treatment.

In a series of two experiments, one indoor and one outdoor, researchers at the University of Connecticut showed that oral, temporal, axillary, aural and forehead temperature devices do not accurately reflect body temperature, when compared to rectal measurements. These commonly used methods of temperature assessment in resting individuals are misconceived to be valid during exercise. Not only did these methods not fall with in a limit of agreement of 0.5°F, the responses of these locations did not reflect concurrent changes in body temperature, therefore eliminating the ability for a correction to be applied.\(^7,8\) Meta-analyses by Huggins et al. and Mazerolle et al. have corroborated the findings that aural and oral temperatures do not reflect body temperature during exercise in hyperthermic individuals.\(^5,9\) This leads to the conclusion that use of one of these aforementioned locations for body temperature assessment may lead to inaccurate diagnostic criteria that can ultimately lead to incorrect treatment.

The only measurement from the University of Connecticut studies that demonstrated agreement with rectal thermometry during exercise in the heat was that of an ingestible thermistor.\(^7,8\) Byrne and Lim reported similar findings in a review and found that ingestible
telemetry fell within Bland and Altman’s limits of agreement with oesophageal and rectal temperatures during exercise. While this is an acceptable measurement for experimental studies, the need to ingest the pill 6-8 hours before exercise limits its applicability in acute care of EHS.

**The Falmouth Road Race**

Since its inception in 1973, the Falmouth Road Race has represented a race where heat stress is a particular challenge to participants. In an eighteen-year study, the race has averaged 2.13 cases of EHS per 1000 runners. This high incidence is likely due to combined metabolic and environmental heat stresses. A 7-mile road race is an ideal duration for the occurrence of EHI. First, the race is short enough to allow athletes to run at a high intensity, but it is also long enough to allow this high intensity exercise to create endogenous heat production that can overwhelm the body’s methods of heat dissipation. In addition, due to the high ambient temperatures and relative humidity during the August race, the body’s principal method of dissipation, evaporative cooling through sweating, is likely less efficient.

A percentage of the cases requiring medical attention each year at Falmouth are exertional heat stroke. In addition the majority of athletes requiring care finish over a relatively short period of time, approximately an hour. These two factors have created a need for a streamlined triage system that allows for rapid diagnosis and treatment. The current National Athletic Trainer’s Association’s guidelines for EHS treatment are reflective of this model developed and employed by the Falmouth Road Race. While the appropriate medical coverage of the Falmouth Road Race has led to a 100% survival rate for EHS, many sporting situations exist where appropriate knowledge and staffing are not available to prevent fatalities. The high
incidence and appropriate medical management of the Falmouth Road Race allow for the safe study of EHI in a competitive context.

**Thermoregulation during uncompensable heat stress**

The fundamentals of thermoregulation during uncompensable heat stress can be expressed through the heat balance equation, which describes the determinants of heat production and dissipation.

\[ S = (M - W) ± C ± K ± R - E \]

- \( S \) = body heat storage, \( M \) = metabolic heat production, \( W \) = external work, \( C \) = convection, \( K \) = conduction, \( R \) = radiation and \( E \) = evaporation\(^{12,16}\)

Body heat storage can be either a positive or negative value, which in turn represents heat gain, in the case of uncompensable heat stress, or heat loss respectively. The determinants of whether \( S \) is positive or negative can be divided into two groups: heat storage and heat dissipation. During rest in a thermoneutral environment, metabolic heat production represents the sole source of heat acquisition while convection (12-15\%), conduction (minimal in all conditions), radiation (60\%) and evaporation (25\%) are all methods of dissipation.\(^{16}\) This results in a situation wherein the body can easily dissipate any metabolic heat production.

The circumstances of thermoregulation change significantly with exercise in the heat. When ambient temperature increases beyond that of the body’s surface the ability to dissipate through radiation and convection decreases and dependence on evaporation of sweat predominates.\(^{12}\) Radiation is controlled by a thermal gradient between the skin and the electromagnetic environment. In fact, exposure to direct sunlight can lead to a significant heat gain through radiation.\(^{16}\) This explains the need to monitor globe temperature when evaluating risk of exertional heat stress. Convection is largely dependent upon wind speed around the
individual; for example, convective cooling plays a larger role in cycling versus running. However, when a positive thermal gradient between the skin surface and the external environment does not exist, as in the case of hot environments with limited wind, convection can contribute to heat acquisition. Finally, while evaporation is capable of dissipation at all ambient temperatures, high humidity limits its efficacy due to a diminished water vapor pressure gradient. This causes sweating to only contribute to dehydration, which can further increase exercise heat stress due to increased cardiovascular demands. As all of these methods rely on the thermal interface of the skin with the environment, highlighting the potential importance of skin temperature in understanding thermoregulation.

The factors described in the heat balance equation represent fluctuating values that differ between individuals and conditions. What determines an individual’s response to exertional heat stress and risk for EHI can be further grouped into intrinsic and extrinsic factors. Intrinsic factors represent the internal physiological, psychological and physical contributors that influence responses. Meanwhile, extrinsic factors describe the external effects of the environment and exercise structure.

**Intrinsic Factors**

Intensity is the largest determinant of endogenous heat production and risk for EHI. With muscular metabolism being only approximately 20% efficient, the remaining amount of energy is released as heat during exercise, which can create unsafe rises in core temperature. Even moderate sustained exercise in the heat can lead to unsafe endogenous heat production. Cases of EHS have reported in as low as 14°C following sustained military marching with equipment.

The rate of rise in body temperature is related to the relative workload of an individual (percentage of VO$_{2\text{max}}$). This means that regardless of absolute workload (e.g. pace), an
individual’s responses to exercise in the heat are personalized based on their abilities. A fitter individual (higher VO$_{2\text{max}}$) is hypothesized to tolerate heat stress more effectively due to decreased metabolic heat production at a given intensity and training adaptations that promote more efficient thermoregulation in the heat.

The endogenous heat produced during exercise relies on internal mechanisms in order to be transferred to the skin surface for dissipation, principally the circulatory system. During exercise in the heat three entities rely on a large amount of blood flow 1) exercising muscle for the delivery of oxygen and nutrients, 2) skin surface to allow for the evaporation of sweat and 3) circulatory system to maintain central blood pressure and venous return. However, when plasma volume decreases wherein adequate blood flow for all three systems cannot be maintained, blood flow is preferentially shunted away from the skin of the extremities in order to preserve the demands of the exercising muscle and maintain blood pressure. This in turn, compromises thermoregulatory ability and can lead to unsafe rises in core body temperature due to an ability to dissipate heat.

For this reason, hydration status has been proposed to affect exertional heat stress. Failure to replace fluid lost through sweat and respiratory losses decreases plasma volume and compromises the blood’s ability to carry heat to the skin surface. Numerous studies have demonstrated that dehydration gradually impairs aerobic performance and affects core body temperature. This drop in plasma volume also leads to increased heart rate to maintain cardiovascular output, termed cardiovascular drift.

**Extrinsic Factors**

Environmental conditions affect an individual’s responses to exercise in the heat primarily by decreasing the efficacy of the body’s methods of dissipation as described
previously. High ambient temperatures diminish the ability of radiation and convection to
dissipate heat, while high humidity decreases the efficiency of evaporation. Data from the
Falmouth Road Race and Twin City Marathon have shown a correlation between ambient
conditions and incidence of EHI. 11,23

**Skin Temperature Responses**

With its predominant role in the dissipation of endogenous heat, the skin surface plays a
key role in the understanding of how the body reacts to exertional heat stress. Skin temperature
may decrease during compensable heat stress, allowing for exercising muscle to receive
sufficient blood flow.24,25 However, during uncompensable heat stress, skin temperature
gradually increases as cutaneous blood flow increases to allow for heat dissipation.20 Through
the action of cutaneous thermal afferents, vasodilators are activated which effectively increases
the thermal conductance of the skin.20 When endogenous heat is brought to the skin surface these
thermal afferents continue to activate vasodilation until the heat can either be dissipated or the
heat stress is removed. This process of heat dissipation is limited by sufficient plasma volume to
allow for the skin circulation to be perfused for dissipation of heat. However as shown
previously, during prolonged exercise or cases of hypohydration blood can be shunted away
from the skin surface to maintain blood flow to the heart and exercising muscles. Therefore, skin
temperature has been suggested to affect both performance and safety during exercise in the heat.
It has also been shown that skin temperature might be modulated differently in fit and unfit
individuals, wherein less fit individuals have higher skin temperatures during exercise due to less
optimized thermoregulatory abilities.26,27 The temperature of the skin can be concluded to
represent both the afferent thermoregulatory input for the CNS and to a lesser degree the thermal
load of the body’s principal dissipation vehicle.
Skin temperature responses do not directly reflect body temperature, however, they may play a role in an individual’s thermal sensation and auto-regulation of exercise intensity.\(^{28}\) Sawka et al. have proposed that high skin temperatures lead to aerobic performance decrements rather than a high core temperature.\(^{22}\) This is likely due to input from cutaneous thermal sensors that participate in regulatory feedback via the hypothalamus.\(^{28}\) The input from these cutaneous temperature sensors could be affected by aerobic training and heat acclimatization, thereby allowing individuals to tolerate higher levels of heat stress. In other words, individuals who are more aerobically fit and heat acclimatized may be able to tolerate higher skin temperatures without performance decrements due to regulatory and physiological adaptations.

While skin temperature has been shown to not correlate with core temperature, thereby limiting its ability as a diagnostic tool for EHI, analysis of skin temperature can provide insight on the physiological responses to exercise in the heat. Pandolf et al. first suggested that convergence of skin temperature and core temperature predicts the limits of heat tolerance.\(^{29}\) As the thermal gradient between the core and skin diminishes, the amount of heat that can be dissipated decreases proportionally. The point at which core temperature is equivalent to skin temperature would represent the point where endogenous heat can no longer be dissipated effectively and thermoregulatory failure is eminent. This limit has been suggested to exist in uncompensable heat stress conditions, particularly high ambient temperatures with subjects wearing clothing or equipment that would limit heat dissipation.\(^{29}\) Cheuvront et al. resurfaced this idea in 2010 adding that skin temperature can be affected by environmental conditions, helping explain the mechanisms for increased risk of EHI in hot environments.\(^{17}\)

Skin temperature plays a key role in thermoregulatory responses during exercise in the heat. In order to apply this concept into clinical and experimental practice researchers have taken
several different approaches to quantify skin temperatures. The calculation of mean skin
temperature, based on several measurement points across the body surface, has been the most
commonly used method since it was described by Ramanathan in 1964.  

While improvements in the formula have been made since this method’s introduction,
fundamental problems still exist. Even with calculations that account for 7 or 14 sites, the
assumption that each of these points is representative of the temperature an entire area is over
simplified. Temperature over an area of skin surface is not uniformly controlled, nor does
dissipation of the heat in the area occur uniformly. For example only one measurement in the
method described by Ramanathan accounts for the arm.  

However, skin temperature is very
likely to be different between the surface of the upper arm and the axilla. Livingstone et al. has
demonstrated these differences in regional skin temperature through point measurements and
thermographic mapping of the chest. Their finding that point measurement of chest skin
temperature can differ from mean chest skin temperature by as much as 3°C calls for a closer
examination of the accuracy of this method.  

Skin temperature, like all physiological measurements, represents a fluctuating
expression that is controlled by several different inputs and feedback mechanisms. For this
reason the use of a uniform formula despite changes in external environments and internal
physiological events likely diminishes its validity. In addition to the previous issues noted with
mean skin temperature calculations, Livingstone et al. found that body fat percentage and
environmental conditions have effects on the measurements obtained from mean skin
temperature calculations.
Thermal Imaging

Thermal imaging has been proposed as an alternative to mean skin temperature calculations due to its ability to map the entire skin surface rather than single point measurements from thermistors. Modern cameras use infrared lenses to detect thermal energy, which in turn is represented as temperature through color gradients. The research performed during exercise with thermography has predominantly focused on skin temperature mapping during exercise in a thermoneutral environment and only recently has been applied to the study of exercise in hot environments, with no cohort study evaluating outdoor heat stress.

Studies specifically using thermal imaging for evaluating thermal stress while exercising in a hot environment are limited. Most of these studies have focused on qualitatively describing the skin temperature responses to exercising in the heat. In addition, many of these studies lack sufficient sample sizes to allow for generalization of results. While this analytical method is useful in terms of our understanding of the body’s response to exercise in the heat, development of this technology and its analysis could aid our ability to diagnose exertional heat illnesses.

Recent research using thermal imaging during exertional heat stress has focused on the ability of infrared imagery to detect elevated core body temperatures based on areas of the skin surface. For example, it has been suggested that the skin temperature of inner canthus of the eye may predict core body temperature. However, an investigation by Teunissen et al. revealed no relationship between oesophageal and inner canthus temperature during rest, exercise or passive heating. The authors also cite a large amount of inter-subject variability at rest, further questioning its diagnostic validity.
Bourlai et al. examined facial skin temperature, assessed via thermal imagery, and body temperature of firefighters during treadmill walking in the heat. They found that mean facial temperature demonstrated a moderate correlation ($r=0.411$) with body temperature assessed via ingestible thermistor. However, this study included only 6 subjects, performing 45 minutes of walking with firefighting gear, elevating their body temperature to 38.3°C. While these results may apply to occupational situations, the intensity of the exercise performed did not mimic athletic situations in which metabolic heat production overwhelms thermoregulatory ability. More likely the increase in body temperature was due to diminished dissipation from the amount of protective firefighting gear worn.

The current evidence does not support the use of thermal imagery for the direct evaluation of body temperature. This is more likely due to anatomical and physiological issues than technological inadequacies. Skin temperature acts as one of the principal inputs for determining thermoregulatory behavior during rest and exercise, thereby leading to the body’s ability to modify core temperature. The effects exercise in the heat has on skin temperature are driven through the responses of vasodilators and vasoconstrictors determining blood flow to the skin, which in turn modulates skin temperature. These mechanisms are independent of those that determine core temperature and are affected by factors beyond endogenous heat production.

However, based on our current understanding of skin temperature, thermal imaging may aid in determination of individuals who should be evaluated further for EHI. As suggested by Pandolf et al. and Cheuvront et al., the convergence of skin temperature and body temperature likely represents a point at which an overwhelming of thermoregulation is imminent. Therefore skin temperature analysis through the use of thermal imaging may be able to act as a
screening process for the determination of EHI risk, wherein individuals with skin temperatures at or near deep body temperatures need further evaluation.
INTRODUCTION

The ability to treat EHI depends on a clinician’s ability to accurately evaluate body temperature. However, the invasive nature of the currently validated methods, rectal, gastrointestinal and esophageal temperatures, may deter some clinicians from assessing body temperature for suspected EHI. The development of novel non-invasive screening methods could help clinicians make better diagnostic and treatment decisions for the care of EHS. Thermal imaging, as an assessment of skin temperature, has been proposed to be an useful tool for the evaluation of thermoregulatory responses and my be useful for the diagnosis of exertional heat illnesses.

Skin temperature has been established to play a role in the regulation and dissipation of body heat during exercise in warm environments. While there is no established direct relationship between skin temperature and body temperature, Pandolf et al. suggested that a convergence of skin temperature and deep body temperature might represent the upper limit for heat tolerance. Cheuvront et al. added to this paradigm, indicating that hypohydration may further exacerbate the conditions of thermal fatigue. This concept would indicate that elevated skin temperatures might reflect high deep body temperatures when thermoregulatory collapse is imminent, creating a context where thermal imaging may be appropriate for the assessment of EHI.

Besides body temperature, disturbances to the central nervous system are the other diagnostic criteria for exertional heat stroke. Ely et al. demonstrated that with acute heat stress, mood changes are likely to be the first noticeable changes in nervous system function.

Extreme hyperthermia has been documented at the Falmouth Road Race, where a retrospective study found a mean of 15.2 exertional heat strokes per year. The nature of the
11.2 km race seems to be an ideal circumstance for uncompensable heat stress. The race is short enough that metabolic heat production, as a by-product of intensity, is great, while the race is also long enough that this endogenous heat load can contribute to increases in body temperature.

No study to date has evaluated the applicability of thermal imaging for evaluating thermal stress in a competitive environment. Additionally, no study has evaluated the skin temperature responses of hyperthermic and near-hyperthermic runners. The purpose of this study was to examine the relationship of skin temperature assessed via infrared thermography, physiological and perceptual variables following intense exercise in the heat. Our specific aims were: 1) evaluate the skin temperature responses of runners following a warm-weather road race, 2) determine the relationship between skin temperature, gastrointestinal temperature and other physiological variables during outdoor heat stress and 3) identify the relationship between skin temperature and perceptual symptoms of heat stress. We hypothesized that skin temperature would increase concurrently with gastrointestinal temperature during the race, due to the progressive increased strain on the thermoregulatory system. We also hypothesized that individuals with higher skin temperatures would demonstrate increased thermal strain and mood disturbances.
METHODS

Study Overview

Race data collection occurred at the Falmouth Road Race held in Falmouth, MA on August 11\textsuperscript{th}, 2013. The Falmouth Road Race is an 11.2 km point-to-point race, held at 10 AM annually. Subjects presented for data collection at two time points, pre-race (PRE) and post-race (POST). An additional subset of subjects completed preliminary fitness testing at the UConn Human Performance Laboratory. The University of Connecticut Institutional Review Board approved this study.

Participants

Subjects were recruited via e-mail recruitment materials and on-site recruitment prior to the race. Subjects were limited to individuals already registered for the race, between the ages of 18 and 60, and those who planned on finishing the 7-mile race in less than 55 minutes. Additionally subjects were excluded if they had a history of the following conditions: chronic health problems, a history of cardiovascular, metabolic or respiratory disease, a fever or other illness at the time of the race, an exertional heatstroke within 3 years, or any of the contraindications to the use of an ingestible thermistor, including but not limited to obstructive bowel disease and gastrointestinal mobility disorders.

A total of 20 male subjects were included in the study. One female was excluded from the data analysis due to the innate differences in thermal imaging analysis for clothed males and females. Subject demographics are presented in table 1.
Table 1. Subject Demographics

<table>
<thead>
<tr>
<th></th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Weight</td>
<td>73.53 ± 10.89 kg</td>
</tr>
<tr>
<td>Finish Time</td>
<td>49.41 ± 8.34 min</td>
</tr>
<tr>
<td>VO\textsubscript{2max}</td>
<td>52.93 ± 8.39 ml·kg\textsuperscript{-1}·min\textsuperscript{-1} (N=10)</td>
</tr>
<tr>
<td>Speed at VO\textsubscript{2max}</td>
<td>15.5 ± 1.5 km·h\textsuperscript{-1} (N=10)</td>
</tr>
<tr>
<td>Speed at Lactate Threshold</td>
<td>14.8 ± 1.5 km·h\textsuperscript{-1} (N=10)</td>
</tr>
</tbody>
</table>

Preliminary Testing

A subset of subjects (N=10) conducted preliminary fitness testing at the UConn Human Performance Laboratory within 40 days of the race. Testing comprised of VO\textsubscript{2max} and lactate threshold testing. Subjects were asked to observe a 24-hour alcohol, 12-hour caffeine and 3-hour food fast prior to testing. Upon arrival to the laboratory, subjects provided a urine sample to be analyzed for hydration status via refractometer (Model A 300 CL, A. Daigger & Company, Lincolnshire, IL). Subjects with a urine specific gravity less than 1.025 were allowed to proceed with testing. Subject’s body mass was then obtained.

Following a 10-minute self-selected intensity warm up, subjects were fitted with a heart-rate telemetry strap (Timex Heart Rate Monitor, TIMEX INC., Middlebury, CT) to allow for monitoring during testing. All subjects began with VO\textsubscript{2max} testing, which consisted of a graded exercise protocol to volitional exhaustion. Subjects completed three-minute intervals of running at a 1% grade beginning at an approximation of their 10k pace until the test was completed. Upon the completion of each stage speed was increased either 0.8 or 1.6 km/h approximated to complete the test in 12 minutes. During the test expired gases were captured via indirect calorimetry and analyzed using a metabolic cart (model CPX/D, Medical Graphics Corporation, St. Paul, MN). VO\textsubscript{2max} was considered to be obtained upon volitional fatigue, a respiratory
exchange ratio greater than 1.10, a heart rate greater than 90% a subject’s age predicted maximum (220-age), or a plateau in VO$_2$ or heart rate despite an increase in intensity.

Following a 30-minute break subjects completed a five-minute self-selected intensity warm up to prepare for lactate threshold testing. An initial finger-prick lactate measurement was obtained and analyzed using a handheld lactate meter (Lactate Plus, nova biomedical, Waltham, MA). Subjects then began a graded exercise protocol, wherein intensity increased by 0.8 km/h each stage. Stages consisted of three minutes running at a given intensity at a 1% treadmill grade. Following each stage subjects straddled the treadmill for 1 minute to allow for collection of a finger-prick blood sample to measure lactate. Lactate threshold was considered to have been achieved if a non-linear increase in lactate measurement occurred between 2 stages.

**Race Day Testing**

The day before the race subjects reported to the research staff to obtain their ingestible thermistor (CorTemp, HQ Inc., Palmetto, FL). They were instructed to take the ingestible thermistor prior to sleeping the night before the race to ensure proper placement within the gastrointestinal tract.

On the morning of the race, subjects presented for PRE variable collection between one and three hours before the race. Subjects first provided a urine sample that was analyzed for USG. PRE gastrointestinal temperature ($T_{GI}$) was then assessed for determination of a baseline measure and to ensure proper placement of the ingestible thermistor. Next subjects were weighed and completed perceptual indices. Perceptual indices consisted of the Profile of Mood States (POMS), Environmental Symptoms Questionnaire-14 (ESQ), thermal sensation and thirst. Subjects then proceeded to a shaded area where two anterior thermal images (superior, inferior)
were obtained. Thermal images were obtained using an infrared camera (ICI 7320P, Infrared Cameras Inc., Beaumont, TX) at a standardized distance, against a white background.

During the race, wet-bulb globe thermometer, ambient temperature and humidity readings were collected at regular intervals at mile 2, 4, 6 and the finish line. Environmental conditions during the race are presented in Table 2.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mile 2</th>
<th>Mile 4</th>
<th>Mile 6</th>
<th>Finish Line</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry Bulb</td>
<td>25.9 ± 2.7</td>
<td>27.8 ± 1.6</td>
<td>23.6 ± 0.5</td>
<td>31.1 ± 1.9</td>
<td>26.5 ± 3.1</td>
</tr>
<tr>
<td>(°C)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Humidity</td>
<td>48.1 ± 4.5</td>
<td>35.1 ± 3.8</td>
<td>45.4 ± 2.8</td>
<td>21.8 ± 3.4</td>
<td>39.2 ± 9.7</td>
</tr>
<tr>
<td>(%rh)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WBGT</td>
<td>21.9 ± 1.7</td>
<td>25.3 ± 1.2</td>
<td>25.2 ± 1.2</td>
<td>27.0 ± 1.1</td>
<td>24.6 ± 2.3</td>
</tr>
<tr>
<td>(°C)</td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

Immediately after the race subjects were brought to the testing area for POST data collection. TC\text{GI} was measured, followed by a finger-prick measurement of blood lactate (Lactate Plus, nova biomedical, Waltham, MA). Subjects then proceeded to a shaded area where two anterior thermal images (superior, inferior) were obtained. Thermal images were obtained at a standardized distance, against a white background. Subjects were then weighed and provided a urine sample that was analyzed for USG. Finally subjects completed POMS, ESQ, thermal sensation and thirst indices.

**Thermal Imaging Analysis**

Subjects who presented for thermal imaging greater than 15 minutes after they completed the race were excluded from analysis. Thermographs were analyzed using IR Flash software (Infrared Cameras Inc., Beaumont, TX). Thermal images were corrected for emissivity, reflection and transmission as appropriate. Images were then divided into 11 regions (Figure 1): Head (H), Superior Torso (ST), Inferior Torso (IT), Left Upper Arm (LUA), Right Upper Arm
(RUA), Left Forearm (LFA), Right Forearm (RFA), Left Thigh (LT), Right Thigh (RT), Left Lower Leg (LLL) and Right Lower Leg (RLL). Average and maximum temperature was obtained for each region for PRE and POST. Mean weighted skin temperature (MWST) was calculated based on an estimate of the body surface area represented via the “Rule of 9’s” for each region.  

![Regions of Interest for Thermal Imaging Analysis](image)

**Figure 1. Regions of Interest for Thermal Imaging Analysis.**

**Data Analysis**

All data were analyzed using IBM SPSS Statistics 21 (IBM SPSS Statistics, Chicago, IL). Data are presented as Mean ± SD in tables and figures unless specified otherwise. Perceptual variables are presented as Median (IQR). Paired samples t-tests were performed to compare PRE and POST values. Wilcoxon signed ranks test was used for the comparison of non-parametric PRE and POST values. Correlation analysis was performed to evaluate the relationship between $T_{GI}$, hydration markers, fitness parameters and MWST. Spearman’s rho was performed to evaluate the relationship between MWST and perceptual variables. An alpha level of 0.05 was set a-priori for all tests.
**RESULTS**

$T_{GI}$ and MWST changes are shown in Figure 2. Three subjects had a POST $T_{GI}$ greater than 40°C. USG decreased from PRE to POST (PRE=1.015 ± 0.014, POST= 1.006 ± 0.005; $p=0.020$), despite significant body mass losses ($\Delta$PRE-POST= -1.5 ± 0.6 kg; $p<0.001$). Skin temperature changes for each region of interest are presented in Figure 3. PRE and POST thermal Images for subjects with the lowest (A), median (B) and highest (C) POST MWST are shown in Figure 4.

![Figure 2](image-url)

**Figure 2.** Gastrointestinal temperature and mean weighted skin temperature changes in a warm-weather road race.

* Indicates significant difference from PRE value. ($p<0.05$)
Figure 3. Skin Temperature Changes by Region.

*Indicates significant difference from pre-race value (p<0.05).
Figure 4. Thermal images of the lowest (A), median (B) and highest (C) POST MWST.

POMS total mood disturbance (TMD) (PRE=−10 (19.5), POST= 6 (29); p=0.022), ESQ (PRE=3 (2), POST=10(12); p=0.001), Thirst (PRE=3 (2) POST=5 (1.5); p=0.022), and thermal sensation (PRE= 4 (1), POST= 5(1.25); p=0.003) all increased from PRE to POST.
The relationship between MWST and T_{Gi} is shown in Figures 5 and 6. Neither of these relationships was significant. The relationship between finish time and MWST is shown in Figure 7. Correlation coefficients between specific regions and POST T_{Gi} are shown in Table 3. POST MWST was not significantly correlated with POST USG (r=0.108, p=0.659), changes in body mass (r=-0.227, p=0.471), VO_{2max} (r=0.201, p=0.578), speed at VO_{2max} (r=0.081, p=0.823), speed at lactate threshold (r=0.257, p=0.473) or POST lactate (r=-0.382, p=0.144). \Delta MWST was not significantly correlated with changes in body mass (r=0.069, p=0.774).

POST MWST was not significantly correlated with POST ESQ (\rho=0.269, p=0.296), POST Thirst (\rho=-0.199, p=0.459), POST TMD (\rho=0.305, p=0.233), or POST thermal sensation (\rho=0.136, p=0.616). \Delta MWST was not significantly correlated with \Delta ESQ (\rho=0.386, p=0.93), \Delta TMD (\rho=0.243, p=0.383), \Delta thermal sensation (\rho=0.387 p=0.139).

Figure 5. Mean weighted skin temperature and gastrointestinal temperature after a warm-weather road race.
Figure 6. Change in mean weighted skin temperature and change in gastrointestinal temperature in a warm-weather road race.

Figure 7. Mean weighted skin temperature and finish time in a warm-weather road race.
Table 3. Correlations of Regions of Interest with POST T_{GI}.

<table>
<thead>
<tr>
<th>Region</th>
<th>H</th>
<th>ST</th>
<th>IT</th>
<th>RUA</th>
<th>RFA</th>
<th>LUA</th>
<th>LFA</th>
<th>RT</th>
<th>RLL</th>
<th>LT</th>
<th>LLL</th>
</tr>
</thead>
<tbody>
<tr>
<td>T_{GI}</td>
<td>0.154</td>
<td>0.258</td>
<td>0.357</td>
<td>0.130</td>
<td>0.190</td>
<td>0.052</td>
<td>0.196</td>
<td>-0.005</td>
<td>-0.059</td>
<td>0.073</td>
<td>0.009</td>
</tr>
</tbody>
</table>

* Indicates significant correlation. (p<0.05)
DISCUSSION

Thermal imaging has been proposed to be useful in the detection of EHI during exercise in the heat. However, due to numerous anatomical and physiological factors, skin temperature would likely only converge with deep body temperature when thermoregulatory failure is imminent. It is for that reason that we evaluated the thermal images of runners in a warm-weather road race where exertional heat stroke is frequently documented. In this field setting we sought to relate thermal images to both the physiological and perceptual signs of exertional heat illnesses to evaluate the diagnostic potential of this modality.

The primary finding of this study was that despite moderate levels of hyperthermia, skin temperature assessed via thermal imaging was not associated with $T_{GI}$. POST MWST was not related to either POST $T_{GI}$ or $\Delta T_{GI}$. Additionally, both MWST and skin temperature for each region of interest decreased significantly from PRE to POST. POST MWST was not significantly correlated with any of the perceptual indices (POMS, ESQ or thermal sensation). These findings contrast our hypotheses that increased MWST would be associated with higher $T_{GI}$ and increased perceptual heat stress.

Our results corroborate Teunissen et al. wherein we found that no single point approached $T_{GI}$ at either time point. Additionally, we found that POST regional skin temperatures analyzed separately did not correlate with $T_{GI}$. These findings contrast those of Bourlai et al. who found that facial skin temperature assessed with thermal imaging was correlated with core temperature. The discrepancy between these studies might be partially accounted for the fact that Bourlai et al. utilized a heat stress that was compounded by the wearing of a firefighting uniform, whereas both our study and Teunissen et al. relied on exercise heat stress in sport clothes. This can be supported by Armstrong et al. who found that football
uniforms had an effect on differences in post-exercise skin temperature of the forearm and neck.\textsuperscript{40}

Based on our findings, it can be surmised that the elevation of body temperature in our subjects likely was not due to a convergence of skin temperature and deep body temperature diminishing heat dissipation, as suggested by Pandolf et al.\textsuperscript{29} Rather, the observed rise in $T_{GI}$ is likely attributed to the intensity of the 11.2 km race creating a significant metabolic demand. When considering the exertional heat stress paradigm of competitive blood flow, both endogenous heat production and increased muscular perfusion resultant of the high intensity can contribute to a hyperthermic $T_{GI}$ increase with slight changes in skin temperature. As noted by Cheuvront et al. a model of the upper limits of heat tolerance wherein the convergence of skin temperature and deep body temperature represent thermoregulatory failure would be exacerbated by body fluid losses secondary to hypohydration.\textsuperscript{17} In our study USG decreased non-significantly from PRE to POST indicating that the additional physiological stress of hypohydration did not play a role in body temperature rises.

Within the context of uncompensable exertional heat stress two divergent paradigms for body temperature rise would explain our finding that $T_{GI}$ increased without concurrent changes in skin temperature. In the case of our study, large amounts of endogenous heat production and high muscular blood flow demands that supersede cutaneous blood flow created a relatively rapid rise in body temperature. On the contrary, a uniformed or equipment laden exertional heat stress, with a diminished ability to dissipate heat could also create similar levels of hyperthermia at lower intensities or longer durations. This is shown by Armstrong et al. who found that time-to-exhaustion was less and rate of rise of rectal temperature was greater in individuals wearing a full football uniform compared to individuals in shorts and a t-shirt.\textsuperscript{40} In this second case the
microenvironment of a uniform minimizes the interaction of the skin and the environment to creating a thermal load. In this situation, cutaneous blood flow demands can be met and skin temperature would rise until a point of thermoregulatory failure is met.

When put within the context of these heat stress concepts the disparities between the findings of Bourlai et al., Teunissen et al. and our study can be reconciled. Due to the fact that Bourlai et al. used uniformed firefighters the rises in deep body temperature could fall into the second situation described above. This impaired heat dissipation model may represent a situation where elevated skin temperature can be associated with elevated deep body temperature.

Interestingly, skin temperature was found to decrease from PRE to POST for all regions of interest. This is likely due to a normal decrease that occurs in compensable heat stress. These changes were significant for the torso and upper arms. These were the regions that not only had the primary muscles of locomotion active during the race but also were likely covered by clothing, minimizing their interface with the environment. Since the thermal gradient between the skin surface and the environment is a method of heat dissipation, the drop in skin temperature for these regions would indicate that they maintained a potential for heat exchange greater than both the exercising muscles of the leg and the face and forearms that were exposed to the environment. This adds support to the recommendations to cover as little skin surface as possible during exercise in the heat.

Skin temperature is also known to be a primary effector input regarding perception of heat. However, we found that POMS, ESQ and thermal sensation were not correlated with MWST. This is likely attributed to the variability in MWST. Additionally, since the variables were measured after the race, in a field setting, establishing a connection between the effects of thermal stress and those of the race day environment are difficult.
Limitations

One of the chief limitations of this study is that we only evaluated anterior thermal images. It is well understood that the interface of the anterior surface of the body and the posterior surface have different interactions with the environment during outside running due to convective air flow. Therefore it is likely that skin temperature between these two surfaces would also differ.

Additionally, we were limited due the fact that not all images were taken immediately after runners had completed the race. Images that were taken greater than 15 minutes after a subject finished the race were excluded from analysis. However, even in these 15 minutes post-exercise drops in skin temperature could occur, confounding measurement. Thermal imaging remains a new technology for the assessment of skin temperature and standardized methods of analysis have not been established.

Due to the nature of this field study, subjects’ actions during the race could not be controlled. We were limited to taking PRE measures 1-hour before the start with some subjects having measurements 3 hours prior to the race. Theses results are limited to our specific study population, and would be difficult to apply to females or different duration races.

Future Research

A key area for future research in examining thermal imaging measurement of skin temperature is in an alternative paradigm for hyperthermia, wherein rises in body temperature are due to diminished heat dissipation, such as exercise in uniform or with equipment. These studies may find a greater connection between body temperature and skin temperature increases because of the compromised heat dissipation. Future studies should also examine females in comparison with males to understand differences in physiological responses to exercise in the heat. Finally,
further studies should be done to evaluate the relationship between skin temperature and deep body temperature at differing levels of hydration.

Conclusions

The purpose of this study was to evaluate the relationship between skin temperature assessed via thermal imaging, physiological and perceptual responses following a warm-weather road race. In the context of this 11.2 km road race, skin temperature assessed via thermal imaging was not related to $T_{GI}$ or any perceptual indices. This indicates that in this setting thermal imaging may not be appropriate for the detection of exertional heat illnesses.
REFERENCES


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