QUANTIFYING THE PHYSIOLOGICAL AND BIOPHYSICAL TIME-COURSE OF HUMAN HEAT ADAPTATION

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CANDIDATES CERTIFICATE

I, Grant Lynch, hereby declare that the work presented in this thesis is, to the best of my knowledge, original except as acknowledged in text. I hereby declare that I have not submitted this material, either in part or in full, for a degree at this or any other institution.

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Dated: 30th December 2022

IMPACT OF COVID-19 STATEMENT

New South Wales government mandated lockdowns and the accompanying return to safe research activity at The University of Sydney has resulted in compromises to this thesis, specifically chapters 4 and 5 where the capacity to recruit and collect data on human participants was essential. Data collection is due to continue during 2023 until both chapters are adequately powered.

In the time between receiving ethical approval to complete the studies outlined in chapters 4 and 5 (10th May 2021) and the submission of this thesis (27th December 2022) our ability to collect data has been disrupted by government mandated lockdowns (June 25th – October 11th 2021), The University of Sydney suspension of research owing to concerns around mitigating the spread of coronavirus upon return to the university (October 11th 2021– January 10th 2022) and a major chamber malfunction causing an extended block without use (27th September – November 14th 2022).

THESIS ABSTRACT

The present thesis builds on existing work done in the field of human thermoregulation, with a specific focus on the impact of repeated heat exposures to a natural (acclimatisation) or artificial (acclimation) environment on physiological adaptation and heat stress risk. The aim of the experimental study presented in chapter 3 was to model the independent influence of different environmental and personal parameters on predicted acclimatisation status throughout a representative year in Australia. Chapter 4 aimed to quantify the time course of changes in ω_{max} throughout 7 consecutive days of heat acclimation. Finally, chapter 5 aimed to develop a novel, non-invasive method to assess changes in physiological heat acclimation status in an at-home setting.

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PUBLICATIONS

Publications written during the doctoral candidature, not formally included within this thesis

1. Schranner, D., Scherer, L., **Lynch, G. P**., Korder, S., Brotherhood, J. R., Pluim, B. M., Périard, J. D., & Jay, O. (2017). In-Play Cooling Interventions for Simulated Match-Play Tennis in Hot/Humid Conditions. *Medicine and science in sports and exercise*, *49*(5), 991–998. https://doi.org/10.1249/MSS.0000000000001183
2. **Lynch, G. P**., Périard, J. D., Pluim, B. M., Brotherhood, J. R., & Jay, O. (2018). Optimal cooling strategies for players in Australian Tennis Open conditions. *Journal of science and medicine in sport*, *21*(3), 232–237. https://doi.org/10.1016/j.jsams.2017.05.017
3. Chalmers, S., Siegler, J., Lovell, R., **Lynch, G**., Gregson, W., Marshall, P., & Jay, O. (2019). Brief in-play cooling breaks reduce thermal strain during football in hot conditions. *Journal of science and medicine in sport*, *22*(8), 912–917. https://doi.org/10.1016/j.jsams.2019.04.009
4. Graham, C., **Lynch, G. P**., English, T., Hospers, L., & Jay, O. (2021). Optimal break structures and cooling strategies to mitigate heat stress during a Rugby League match simulation. *Journal of science and medicine in sport*, *24*(8), 793–799. https://doi.org/10.1016/j.jsams.2021.04.013

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CHAPTER 1: INTRODUCTION

Background

Humans attain thermal equilibrium by matching the amount of heat generated internally with the amount of heat transferred from the skin surface to the surrounding environment [1]. When the rate of heat produced exceeds the rate of heat lost to the environment, heat is stored inside the body and body temperature increases. When heat is continually stored inside the body without the means of dissipating it, body temperature can reach critical limits, causing heat illness and even potentially death [2, 3]. For different people, and for different organ systems within the same person, these limits can vary significantly. Prolonged exposure to hot weather, particularly during summer months, can cause excessive heat to be stored inside the body and is associated with excess mortality [4] and increased rates of exertional heat stroke during exercise in military personnel, occupational workers and athletes [5]. To develop effective strategies that can mitigate the negative effects of excessive heat stress requires an understanding of the inherent or developed level of individual adaptation to the heat [2].

Repeated heat exposures to a natural (acclimatization) or artificial (acclimation) environment can result in profound physiological adaptations that improve heat tolerance and minimize the strain placed on the body to reduce heat stress risk [6]. Maximum sweat rate is substantially increased through increases in the amount of active sweat glands to modify the boundary between compensable and uncompensable heat stress [7, 8]. Resting body temperature is lowered to allow more cumulative body heat storage to occur before reaching critical absolute body temperatures and plasma volume in the blood is expanded to lower heart rate and improve cardiovascular stability [9, 10].

For public health officials and organisations, a key aspect of managing heat stress risk is characterising the level of physiological protection to the heat workers may have at different times throughout the year from repeated exposures to the natural environment [11, 12]. The concept of natural acclimatization to the heat is a widely held conviction by many epidemiologists [13-15], however, it remains unclear if the stimulus necessary for physiological heat acclimatization through exposure to the natural climate, individual physical activity and clothing, is sufficient to induce adaptation to the heat. If acclimatization does occur, the timeline of how it develops and to what extent it offers protective benefits is also unknown. In Australia, to our knowledge, only one study has demonstrated the independent influence of natural acclimatization, with sweat rates during 35 minutes of cycling (40% $\dot{V}O_{2\,max}$) higher in summer compared to winter months for male outdoor workers of different trades in a hot, dry climate (Perth, Western Australia) [16]. However, given the large differences in prevailing climates across the various regions of Australia, the increasing prevalence of air-conditioning units for personal (e.g. home, car) and industry (e.g. office building) use [17], and the heat exposure levels necessary to fully physiologically adapt people to the heat [8, 18-20], it remains unclear whether the combination of environmental heat exposure, physical activity and clothing worn are sufficient to induce an adaptation to the heat. Even if the combination of environmental and personal factors is sufficient to elicit a natural physiological adaptation to the summer heat, the time course of the acquisition, maintenance and decay of acclimatization is unknown.

Our understanding of how humans respond to artificial heat exposure (heat acclimation) is well established [21-23]. Typically, exposures require an individual to exercise for >60-100 minutes in hot ambient conditions, often coupled with high humidity, for a minimum of 7 consecutive days. Full heat acclimation usually requires 10 to 14 days of consecutive exposure [22]. A key physiological adaptation with heat acclimation is the

elevation of an individual's maximum evaporative capacity (E_{max}). This elevation is mediated by an increase in the maximum proportion of skin surface that can be covered in sweat (i.e., maximal skin wettedness (ω_{max})) that theoretically ranges from zero sweating with only trans-epidermal diffusion of moisture (0.06) to all skin surfaces completely saturated with sweat (1.00). The importance of estimates of ω_{max} are that they are used to model heat stress risk for active people in hot environments (e.g., occupational settings) using rational heat stress indices. The best example is the current international standard for predicting occupational heat strain (ISO 7933), which uses values of 0.85 and 1.00 to characterize "unacclimated" and "acclimated" individuals to the heat, respectively [24]. Recent work has questioned these values, reporting unacclimated ω_{max} values as low as 0.72, that increases to 0.84 with 8 weeks of aerobic training, and further increases to 0.95 with 8 consecutive days of subsequent heat acclimation in a hot-humid environment [8]. What remains unclear is the time course of how a person transitions from "unacclimated" to "acclimated", making an accurate assessment of an individual's heat stress risk *during* the acclimation process difficult to ascertain.

While the importance of minimizing individual heat stress risk through inducing a physiological heat adaptation cannot be overstated, the process of evaluating adaptation status typically involves access to highly specialised, expensive equipment coupled with measurement techniques that are invasive in nature. This limitation has implications for occupational workers, athletes, and the general population, where knowing the level of adaptation would help tailor strategies to best prepare for work or competition in the heat [25-27] and understanding the naturally acquired protection through summer months would require a test that is broadly scalable and can be taken into people's homes. Heat tolerance tests are objective screening tools used to determine suitability to return to work or competition following a bout of heat-related illness by measuring physiological responses during a standard exercise protocol in the heat. However, current iterations require a climate

chamber and are not designed to be sensitive to acute changes in acclimation status [28, 29]. A practical, cost-effective alternative are passive heat stress tests, with saunas and hot water immersion shown to be as sensitive to changes in acclimation status as exercise focused assessments [30, 31]. Additionally, physical techniques typically used to measure adaptation status are usually not suitable in the field or at-home. Of the standard measures taken in a laboratory when quantifying heat exposure and subsequent adaptation, core temperature requires thermistors inserted in either the rectum or esophagus and local measures of sweating involve either body sweat mapping [32] or ventilated capsules [33] to perform accurately. The combination of both a highly responsive measure of core temperature and continuous measurement of the amount of moisture on the skin are used to identify an onset threshold for sweating. With the challenges around the practicality of the test administered and the measures necessary to detect changes in acclimation status, it remains unclear whether a non-invasive, easily employable testing battery could detect changes in acclimation/acclimatization status, which would be a valuable tool to track adaptation and predict vulnerability to extreme heat.

Aims

Study #1 (Chapter 3)

Model the time course and magnitude of the potential for physiological heat acclimatization throughout a typical summer across all states and territories of Australia.

Study #2 (Chapter 4)

To quantify the time course of changes in ωmax throughout 7 consecutive days of heat acclimation by comparing critical environmental limits on alternate days.

Study #3 (Chapter 5)

To develop a novel, non-invasive method to assess changes in physiological heat adaptation status in an at-home setting.

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CHAPTER 2: LITERATURE REVIEW

Human Heat Balance

At rest, our internal body temperature is ~37°C and this is maintained by ensuring that the rate of heat that we lose to the surrounding environment equals the rate of heat produced inside the body. This balance between heat production and heat loss can be expressed using the fundamental law of human heat balance

$$M - W = \pm C \pm K \pm R + E \pm S\,(W) \quad (1)$$

where: M is metabolic energy expenditure; W is external work; C is convective heat transfer; K is conductive heat transfer; R is radiative heat transfer; E is evaporative heat loss from the surface of the skin and S is the net heat storage within the body.

Metabolic Heat Production

Estimating the heat produced inside the body is fundamental to the assessment of the human thermal environment. Heat is a primary component of the chemical reactions in human cells used to provide energy for all human functions. The amount of heat produced is calculated by subtracting the external work performed to complete a given function (W) from the rate of metabolic energy expended (M). Since metabolic energy is derived primarily through oxidative pathways at rest and during exercise, metabolic rate is generally proportional to the rate of oxygen consumption ($\dot{V}O_2$) and estimates are typically performed using indirect calorimetry based on steady-state $\dot{V}O_2$, accounting for the macronutrient energy equivalents of carbohydrates (21.13 kJ per litre of oxygen consumed) and fats (19.62 kJ per litre of oxygen consumed) [4, 5]. Thus, M can be estimated with

$$M = \dot{V}\mathrm{O}_2 \cdot \frac{\left\{\left[\left(\frac{RER - 0.7}{0.3}\right) \cdot 21.13\right] + \left[\left(\frac{1.0 - RER}{0.3}\right) \cdot 19.62\right]\right\}}{60} \cdot 1000\ (W)$$

(2)

where: $\dot{V}O_2$ is the rate of oxygen consumption in litres per minute (L/min) and RER is respiratory exchange ratio (volume of carbon dioxide produced ($\dot{V}CO_2$) divided by the volume of oxygen consumed ($\dot{V}O_2$)).

When a person performs an activity, some of the heat produced will be used to perform external work. Humans are largely inefficient at performing external work so typically, mechanical efficiency (i.e., M divided by W) is < 20% meaning that most of the energy produced inside the body is released as heat rather than effectively used to perform an activity. During outdoor cycling, external work is calculated by accounting for factors like wind resistance and the incline of the terrain, while on a stationary cycle ergometer, mechanical work is equal to the multiplication of the force generated to overcome the resistance of the wheel by the speed of the ergometer belt. For running, the energy converted into work is lower and if running on a flat surface, requires negligible external work with all metabolic energy converted to heat [6].

Dry Heat Transfer

Dry or "sensible" heat transfer is the sum of the convective (C), conductive (K), and radiative (R) heat transfer pathways. All three pathways follow the second law of thermodynamics and flow in the direction of high to low temperatures between the skin and the surrounding environment.

Convective heat transfer is primarily driven by a temperature gradient between the skin surface (T_{sk}) and ambient air (T_a), along with the rate of air flow across the skin. When T_a is lower than $T_{sk,}$ heat is transferred away from the body resulting in a net heat loss. Conversely, if T_a is greater than T_{sk}, the direction of transfer is reversed resulting in net heat

gain. As air speed increases, convective heat transfer is increased in the direction of the established gradient between the skin and the air. Additionally, as the drive for convective heat transfer depends of the proportion of skin that is exposed to the natural environment, the amount and type of clothing worn also influences convection by creating a microclimate between the boundary layer of the clothing and the skin surface, insulating areas of the body and limiting the capacity for heat transfer [7].

Heat transfer via radiation is determined by; the temperature difference between the skin and all physical objects that are emitting or absorbing radiation within the surrounding environment, the emissivity of the object; and the effective radiative area of the body exposed to the radiant source [8]. Mean radiant temperature (T_r) indicates the net influence of these emissions from the environment, including the sun [8]. When indoors, T_r is assumed to equal ambient temperature without any significant sources of radiation present. However, when outside, T_r is estimated using black globe temperature (T_g) and air speed (v_{air}) using [9]

$$T_r = \left[\left(T_g + 273\right)^4 + \frac{1.1 \cdot 10^8 \cdot v_{air}^{0.6}}{0.44} \cdot \left(T_g - T_a\right) \right]^{0.25} \text{(°C)}$$

(3)

Convective and radiative heat transfer from the skin can be calculated together with the average of the mean radiant and ambient air temperature, weighted by each respective heat transfer coefficient, known as operative temperature (T_o). When indoors, there is no additional source of radiation so T_o can be replaced with T_a. Following on, heat loss from the skin via convection and radiation can be estimated with

$$C + R = \frac{(T_{sk} - T_o)}{(R_{cl} + \frac{1}{h \cdot f_{cl}})} \cdot A_D \ (W)$$

(4)

where T_{sk} is mean skin temperature (°C); T_o is operative temperature (°C); R_{cl} is the dry heat transfer resistance of clothing ($m^2 \cdot °C \cdot W^{-1}$); h is the combined connective heat transfer

coefficient (in $W \cdot m^{-2} \cdot K^{-1}$); f_{cl} is the area factor of clothing that estimates the surface area for heat exchange is increased because of the clothing worn.

The dry heat transfer resistance of clothing (R_{cl}) can either be derived from "clo" units in tables provided by the International Organization for Standardization [10] or by using a thermal manikin. The clothing area factor (f_{cl}) is estimated by assuming a correlation between the increase in area factor and R_{cl} of a clothing ensemble [11]. The combined heat transfer coefficient (h) is calculated as the sum of the convective (h_c) and radiative (h_r) coefficients. h_c depends on the air velocity, nature of airflow, movement velocity, posture, barometric pressure, and clothing properties [12]. h_r indoors is assumed to be 4.7 $W \cdot m^{-2} \cdot K^{-4}$, however, can be estimated more precisely using

$$h_r = 4\varepsilon\sigma \cdot \frac{A_r}{A_D} \cdot \left[273.2 + \frac{t_{sk} + t_r}{2}\right]^3 (W \cdot m^{-2} \cdot K)$$

(5)

Where ε is the emissivity of the body surface; σ is the Stefan-Boltzmann constant (5.67 x 10^{-8} $W \cdot m^{-2} \cdot K^{-4}$); and A_r/A_D is the fraction of the body surface that is involved in radiative heat transfer and is determined by posture.

Conduction is the transfer of heat through direct contact between the skin and a solid object and is determined by the conductivity of the object, the total surface area in contact, the time spent in contact and the temperature gradient between the object and the skin surface [8]. Typically, conduction is negligible during human heat stress conditions, however, has the potential to be a significant source of heat gain or loss, provided there is a large gradient between the temperature of the object and the skin, augmented by the thermal conductivity of the object and the surface area of the body the conductive material is applied to. For example, the use of warming blankets [13], circulating water via a water perfused suit or ice put in direct contact with the skin [14] are all used to maximize heat transfer via conduction and the magnitude of the associated heat transfer is calculated with

$$K = k \cdot A \cdot \frac{(T_o - T_{sk})}{L}$$

(6)

where k is the thermal conductivity of the object in contact with the skin; A is the skin surface area in contact with the object (m^2); T_o is the temperature of the object (°C); T_{sk} is the temperature of the skin (°C), and L is the thickness of the object in contact with the skin.

Respiratory Heat Loss

Respiratory heat loss occurs via convection and evaporation. As air is inhaled, it is warmed to body temperature and becomes saturated with water vapour, so the amount of heat loss is determined by the ambient temperature and humidity of inspired air, along with the rate of ventilation per minute (volume of air moved in and out) and the air density [15]. Accordingly, respiratory heat loss via convection and evaporation can be estimated with [16]

$$H_{res} = \begin{bmatrix} \left(0.001516 \cdot M(28.56 + 0.641 \cdot P_a - 0.885 \cdot T_a)\right) \\ +(0.00127 \cdot M(59.34 + 0.53 \cdot t_a - 11.63 \cdot P_a) \end{bmatrix} (W)$$

(7)

where H_{res} is respiratory heat loss; M is metabolic energy expenditure (W); T_a is inspired air temperature (°C); and P_a is the vapour pressure of inspired air (kPa).

Evaporative Heat Loss

Evaporative or “insensible” heat loss from the skin is the largest modifiable avenue of heat loss from the body. When heat production exceeds heat loss via dry heat loss pathways, humans will attempt to maintain heat balance by producing sweat and secreting it to the skin surface, predominantly from eccrine sweat glands [15]. This secreted sweat can liberate 2426 J of heat energy for every gram that is evaporated from the skin surface. In scenarios where air temperature equals or exceeds skin temperature and dry heat loss is minimized, evaporative heat loss becomes the only feasible pathway to dissipate heat without an external intervention [8]. Evaporation is driven by the vapour pressure gradient between the skin

surface and the surrounding environment and the air flow across the skin surface. To estimate evaporative heat loss requires accurate measures of whole body sweat loss and the fraction of secreted sweat that evaporates (i.e., sweating efficiency) with

$$E_{sk} = (m_b \cdot \lambda \div 60) \cdot \frac{S_{eff}}{100} (W)$$

(8)

where m_b is the rate of body mass change ($g \cdot min^{-1}$); λ is the latent heat vapourisation of water (2426 $J \cdot g^{-1}$); and S_{eff} is the proportion of secreted sweat that evaporates from the skin surface (%).

When exercise is performed in a humid and/or still environment, sweating efficiency declines meaning that a larger proportion of sweat that is secreted to the skin surface does not evaporate and either remains on the skin, drips off the body, or gets trapped in clothing. Sweating efficiency is determined by the "skin wettedness" required (ω_{req}) which is the proportion of skin surface that needs to be saturated in sweat to achieve thermal balance, and is estimated by dividing the evaporation required to achieve heat balance (E_{req}) by the maximal evaporative rate possible in the ambient environment (E_{max}) with

$$\omega_{req} = \frac{E_{req}}{E_{max}} (ND)$$

(9)

where E_{req} is defined by rearranging the fundamental heat balance equation and assuming a rate of body heat storage of zero:

$$E_{req} = (M - W) - (\pm C \pm K \pm R) - H_{res} (W)$$

(10)

E_{max} is determined by the water vapour pressure gradient between the skin and the air, as well as air speed, clothing properties, and the maximum proportion of the skin that can be physiologically saturated with sweat (ω_{max})

$$E_{max} = \omega_{max} \cdot \frac{\left(P_{sk,sat} - P_a\right)}{\left(R_{e,cl} + \frac{1}{h_e \cdot f_{cl}}\right)} \cdot A_D \ \ (W)$$

(11)

where ω_{max} is maximum skin wettedness; $P_{sk,sat}$ is the water vapour pressure when the skin surface is completely saturated (kPa), calculated using Antoine's equation [17]; P_a is the water vapour pressure measured in the ambient air (kPa); $R_{e,cl}$ is the evaporative heat transfer resistance of clothing ($m^2 \cdot kPa \cdot W^{-1}$); f_{cl} is the clothing area factor; h_e is the evaporative heat transfer coefficient ($W \cdot m^{-2} \cdot kPa^{-1}$) calculated by multiplying h_c by the Lewis relation coefficient (16.5 K/kPa), accounting for barometric pressure [12]; and A_D is body surface area (m^2).

Expressing skin wettedness as ω_{req} defines the physiological compensability of the environment. When ω_{req} is less than ω_{max}, conditions are considered compensable and an individual can lose sufficient heat through evaporation to compensate for net heat gain, while if ω_{req} exceeds ω_{max}, conditions are considered 'uncompensable', and insufficient heat will be lost through evaporation, and thus heat will continue to accumulate within the individual. This concept is shown in Figure 1 where a given exercise prescription of 60-min of cycling at a fixed intensity can lead to large amounts of heat stored inside the body when the conditions are considered 'uncompensable' due to the thermal stress exceeding the upper physiological limit for sweat rate and an inability to maximally wet the skin [1] (Figure 1A, 1B).

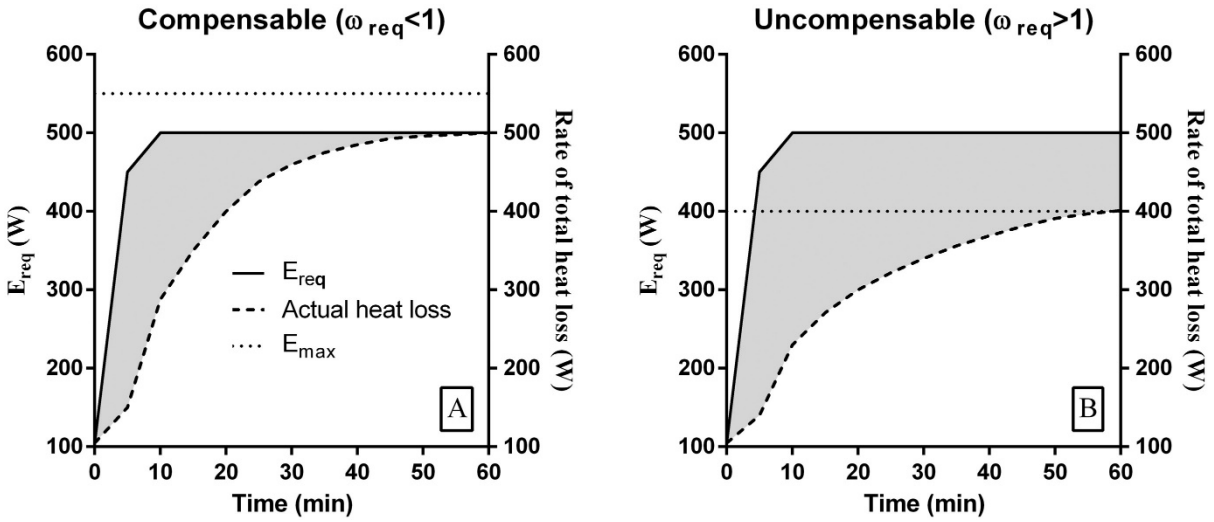

Figure 2.1 – *Figure reprinted with permission from Cramer et al. 2016 (Appendix A) [1]. Representative pattern of change in the evaporation required for heat balance (E_{req}) and the rate of total heat loss during 60 min of exercise at a fixed intensity in compensable (A) and uncompensable (B) conditions.*

Heat Storage

When an imbalance exists between the rate of heat production and the rate of heat loss, body heat storage occurs (S). At rest in a thermoneutral environment, typically S is zero as the body can achieve heat balance through dry heat loss pathways without the need for any evaporative heat loss. Following on, if the combination of environment, clothing and activity create a scenario where total heat gain exceeds the rate of total heat loss ($S > 0$), heat storage and internal temperature will increase. Conversely, if a similar scenario leads to total heat loss outweighing the rate of heat gain ($S < 0$), heat storage and internal temperature will fall. The change in body heat content (kJ) is the product of the time and the net difference between the rate of heat gain and heat loss.

Human Heat Stress

When the combination of environmental (air temperature, humidity, radiant temperature, air velocity) and personal (clothing, activity) factors lead to heat storage, the thermoregulatory system responds to increase heat loss. Activating heat loss pathways are an attempt to defend core temperature from reaching critical values that can result in compromised organ function, heat illness and death [18, 19]. With increases in the duration and frequency of high ambient temperatures (i.e., heatwaves) linked to excess human mortality [20, 21], and that one third of the population are exposed to > 20 days of deadly environmental heat each year [22], understanding the mechanisms that underpin these physiological responses would help develop heat-health management systems for key demographics (i.e. occupational workers) to minimise the potential negative health effects of excessive heat stress.

Physiological Responses to Heat

During heat stress, continued heat storage predicated on sufficient heat stress causes vasodilation that increases skin temperature, followed by a sweating response that has a physiological limit [23]. Concurrently, a gene expression response (heat stress response) is activated to assist in maintaining core body temperature and thermal balance. Once this limit is reached, the rate of heat storage becomes more rapid as the amount of required evaporative heat dissipation exceeds the maximum evaporative capacity for the environment. If unattenuated, as the rate of heat storage increases, clinical presentations of heat stroke can occur that result from a compromised viability of organ tissues, allowing endotoxins to enter the cell wall, leading to cell injury and dysfunction [24].

Increases in skin and/or internal body temperature are sensed by thermoreceptors [25]. Included with these receptors are transient receptor potential (TRP) channels that serve to discriminate between the range of temperatures sensed by mammals [26, 27]. Information

detected by thermoreceptors is propagated to the central nervous system to generate thermoregulatory responses. Within the central nervous system, the primary somatosensory cortex in the parietal lobe can perceive skin or internal temperature changes and is responsible for adjustments in behaviour like seeking shelter when exposed to the radiant heat from the sun or removing layers of clothing when performing vigorous activity [28]. Conversely, the preoptic area of the hypothalamus responds primarily to core body temperature and elicits autonomic responses that include sympathetic nerve activation to increase blood flow to the periphery via vasodilation, and cholinergic pathway activation to secrete sweat to the skin surface [26].

When body temperature increases, blood is taken from the core to the periphery via dilation of the cutaneous vascular bed to increase blood flow and help improve heat transfer between the muscles, skin, and environment. Because the oxygen demand in metabolically active tissue is unaltered, the amount of blood flow must be split between the active tissues and the vascular beds. This splitting of resources increases cardiac output and reduces splanchnic and kidney blood flow to help maintain arterial blood pressure [29] and consequently, the heart must work harder to support the additional cardiac demand [30]. This increased demand is the primary health concern during heat stress, particularly in population groups with pre-existing conditions that can result in a compromised ability to deliver oxygen effectively to the heart [19].

Body water lost through excessive sweating must also be replaced otherwise the associated dehydration can further exacerbate thermal, cardiovascular and renal strain [31]. The magnitude of strain depends on the level of hypohydration and the type of fluid loss (iso-osmotic or hypo-osmotic). Dehydration has been proposed to impact thermosensitive neurons in the hypothalamus, causing a delayed onset of sweating [32] and a blunted thermosensitivity [33], resulting in a greater rise in core temperature to achieve steady state

sweating. For the cardiovascular system, reductions in blood volume stemming from dehydration decrease venous return to the heart and cause subsequent reductions in pre-load and stroke volume [34]. Further, renally, although poorly understood in humans, heat-related reductions in renal blood flow have been shown to produce local ischemia in rats [35] and consequently a localized reduction in ATP. A depletion of ATP can theoretically increase the risk of acute kidney injury (AKI) which, even though reversible, can be fatal if untreated and is linked to chronic kidney disease (CKD) which can result in the need for renal replacement and/or dialysis [36].

At a cellular level, heat stress and the associated elevations in temperature can cause proteins to unfold or misfold, leading to cell injury and death [37]. Our bodies have a proposed heat stress response [37] that mirrors model organisms, such as, flies, worms, and yeasts [38] and is activated when an influx of sub-optimal proteins are detected in our system. The role of the heat stress response is to ensure the integrity of our system by identifying misfolded proteins and assisting in refolding them to preserve cellular structures and functions [39, 40]. The proteins that mediate this response are heat shock proteins (HSP) whose expression rapidly increase in anticipation of, during and after acute heat stress [41]. From a public health perspective, conditions that limit the effectiveness of HSP expression and the concurrent heat stress response like aging or lack of adaptation to the heat may act to predispose an individual to a heat-related adverse event.

Occupational Heat Stress

Heat stress poses a significant occupational hazard in the workplace [42, 43]. Stemming from hot ambient temperatures, some of the negative impacts are higher work accident frequency due to concentration lapses [44], higher levels of fatigue, poorer decision making and decreased cognitive performance [45]. By the end of the century, half the global

population is predicted to experience harmful climate conditions and this number will be disproportionately skewed for workers in climate-vulnerable industries, including agriculture and construction [22]. These sectors currently employ approximately half the global labour force (agriculture ~32%; construction ~10%) and the associated heat-induced labour loss is expected to be $2400 billion USD by 2030 [46-48].

An increased heat stress risk during occupational work typically refers to a combination of high metabolic heat production and long exposure times, providing a platform to generate and store excess heat in the body [8]. Fundamentally, the characteristics that define the thermal environment are air temperature, radiant temperature, humidity and air velocity with the interaction of each parameter with an individual's activity level and clothing worn vital to predict how workers may behaviourally or physiologically compensate to a given stress [8]. As ambient temperature increases the gradient between air temperature and skin temperature decreases, limiting convective heat loss. If outdoors, solar radiation from the sun can add additional heat to the body via radiation. In humid environments, relative increases in the water vapour in the air limit our evaporative capacity by decreasing the vapour pressure gradient between our skin and the environment [8]. Air flow influences heat transfer to and from the body, with increased flow permitting a greater potential for cooling via evaporation provided there is enough sweat pooled on the skin surface [8]. For personal parameters, as the physical demands of a task increase, body temperature will increase in response to the greater engagement of skeletal muscles to perform work, releasing a substantial amount of heat [15] while the ability to successfully dissipate heat from the body to the surrounding environment is mitigated as the dry insulative and evaporative resistance of the clothing worn increases [8, 11].

Consequently, in conditions where the combination of environmental and personal factors constitute sufficient challenges to the thermoregulatory system, workers typically

reduce their work output relative to normal conditions by taking more rest breaks to facilitate a greater fluid consumption to offset fluid losses, or slowing down their pacing throughout the workday [49]. These strategies while potentially unappealing for employers with the assumption that more breaks would facilitate less absolute output, are advantageous for overall productivity with fewer rates of accidents and sick leave culminating in reduced absenteeism [50, 51].

Since the combination of environment, clothing and heat production can elicit significant strain for occupational groups, finding appropriate solutions to augment pacing strategies to help mitigate thermal strain have utility for workers and businesses [43]. Independent of the pacing and hydration considerations above, an umbrella review by Morris et al [52] grouped strategies to mitigate occupational heat strain as either environmental cooling (e.g. air conditioning), clothing optimization (e.g. air cooled garments), innate conditioning (e.g. heat acclimation) or personal cooling (e.g. skin wetting, cold water immersion). The authors concluded that the order of effectiveness to help mitigate occupational strain for businesses was to attempt to; completely cool the local environment with air-conditioning units or accelerated air flow with a fan; have workers wear light, brightly coloured or reflective clothing that is breathable and loose to protect from solar radiation; and improve workers physiological state through physical fitness or level of adaptation to the heat [52].

Predicting Heat Strain

Indices that integrate the effects of the thermal environment can predict thermal strain for occupational groups or individuals and are used to design suitable interventions or establish safe working limits [8]. Heat stress indices are considered rational, empirical, or direct with rational indices based on calculations involving the fundamental human heat

balance equation; empirical indices based on equations derived from physiological responses of human subjects; and direct indices using measurement of instruments (usually temperature) to 'simulate' the human body response [8].

For empirical or direct indices, Wet Bulb Globe Temperature (WBGT) is the most widespread for use in heat-stress management. The index was developed as a control measure for heat stress in American military training camps in the 1950's and when including solar radiation, incorporates a black copper sphere that covers a temperature sensor (black globe temperature), a thermometer covered with a wet cotton wick that is suspended and exposed to the ambient environment (natural wet bulb temperature), and a regular dry bulb thermometer that is shielded from radiation (dry bulb temperature) [53]. Black globe is equal to dry bulb temperature unless solar radiation causes the copper sphere to heat up. Only when the sphere is being warmed up from solar radiation is the measure sensitive to the effect of the wind [53]. The natural wet bulb accounts for humidity and air speed by measuring the amount of evaporation from the wick and the dry bulb temperature is only affected by ambient air temperature. The index outputs a single value (in °C) that is used to indicate when an environment is too risky to work, train or compete. While the WBGT is widely used, the three components of WBGT (natural wet bulb temperature, black globe temperature, and aspirated air temperature) are often not measured. Indeed 70% of a given WBGT value is derived from natural wet bulb temperature, and this value is rarely directly measured. When measured, The output WBGT is sensitive to changes in the potential for convection, conduction, radiation, and evaporation, the index does not account for wind when there is no solar radiation present and it does not perform well in very hot and dry environments because it does not directly account for limitation to sweat output of humans [54].

The most established rational index is the predicted heat strain method (PHS) that is also the internationally recognized standard for the analytical determination and interpretation

of heat stress [16, 55]. This model was first established as the required sweat rate model then further developed with a large database of responses of people to hot conditions that were divided into two parts, one half used to develop the model and the other to evaluate it [16]. By accounting for factors like the distribution of heat stored in the body, the rate of increase in skin temperature and sweat rate through exponential averaging, the evaporative efficiency of sweating, and the influence of ventilation on clothing insulation, the PHS is considered the most complete rational model to predict core body temperature (rectal temperature) and sweat loss responses in a thermal environment. Additionally, by setting limit values for maximal skin wettedness, sweat rate, water loss and rectal temperature, the model outputs a maximum allowable exposure time that flags when either rectal temperature or accumulated water loss reaches corresponding limits. Even with a comprehensive assessment of the thermal environment and accompanying thermal strain, the PHS still assumes that the maximum sweat rate for acclimatized individuals is on average 25% higher than non-acclimatized. Additionally, with recent evidence to support a lower maximal skin wettedness value for unacclimatized (0.7) compared to partially (0.85) and completely (0.97) acclimatized individuals [56], the binary input within the model (0.85 “unacclimatized” and 1.0 “acclimatized”) that ultimately determines the maximum sweat rate may lead to an overestimation of the compensable limits for unacclimatized workers, increasing risk of excessive heat strain.

In addition to heat stress indices (e.g. WBGT) and heat strain indices (e.g. PHS), work stemming from the US Army Research Institute for Environmental Medicine [57] led to another approach that uses data collected during experimental studies to create prediction equations for the development of heart rate [58], rectal temperature [59], sweat rate [60] and water loss [61] for exercise in warm and hot environments. The main application of the equations is in military logistics (e.g. calculation of water provision requirements) and they

are not suitable for induvial control of hydration [53]. Over the last 70 years, 100 heat stress indices have been developed to varying levels of success and application [53]. For the current thesis, the predicted heat strain model was selected for use in a modelling study presented in chapter 3. This index was selected because it estimates the key human thermo-physiological responses that can be used as a forcing function for predicting heat acclimatization (i.e., rectal temperature and sweat rate), and it has been widely validated with human physiological data in the same context as which we were applying the model (i.e., occupational heat stress settings) [53].

Human Heat Adaptation

As humans progressively and repeatedly expose themselves to ambient environments that stress the thermoregulatory system to elevate core temperature, a series of physiological adaptations occur to minimize the strain placed on the body. These changes serve as a protective measure for individuals by reducing the risk of heat stroke [24], with 75% of occupational heat related deaths linked to a sub-optimal state of heat adaptation [62], and help restore decrements in exercise performance [63]. The physiological adaptation to the heat has been well documented evidenced by a lower resting core temperature to allow a greater heat storage to occur before reaching a critical values, a higher maximum sweat rate as a result of an increase in the amount of sweat glands activated, and greater cardiovascular stability supported by an expanded plasma volume [3, 64-66]. Even though the impact of adaptation to the heat can be shown with a combination of thermoregulatory, cardiovascular and perceptual measures, ultimately, these changes are driven by a modification of the boundary between compensable and uncompensable heat stress, secondary to an alteration in an individual's maximum evaporative capacity [8].

For sweating specifically, heat adaptation increases maximal skin wettedness, with 10 days of passive acclimation in a hot-humid environment being shown to increase ω_{max} to 1.0 [67, 68] while 8 days of cycling in similar conditions led to an average ω_{max} value of 0.97 [56]. Regular heat exposure also increases maximal sweat rate and sensitivity through increases in the amount of eccrine glands secreting sweat per unit area [69], while facilitating a conservation of sodium loss to maintain fluid balance by maintaining the osmotic pressure gradient between intracellular and extracellular fluid [70]. The volume of plasma in the blood is expanded [71] with the proposed mechanism being either an increase in extracellular fluid through the retention of sodium chloride or the oncotic effect of intra-vascular protein [71].

Improvements in cardiovascular stability are typified by a lowering of both resting and exercising heart rate for a given work rate and these reductions are proposed to be partly because of an improved redistribution of blood volume but primarily mediated by plasma volume expansion [72].

Improvements in thermal tolerance are inextricably linked to heat adaptation in that adaptation reduces heat strain while tolerance increases survivability to a given heat strain [3]. Thermal tolerance is governed by the heat shock response (see "Human Heat Stress"), where heat shock proteins (HSP) of differing molecular masses either maintain structural proteins (intracellular) or act as a signal to trigger the immune-stimulatory response (extracellular) [73]. In humans, the expression of HSP's as a family during acclimation remains unclear, however, a positive effect on the expression of one specific protein (HSP70) has been shown in humans, with the frequency (number of days) the only factor moderating the response [74].

Broadly, there are two classes of thermal adaptation to the heat, either with exposure to natural (heat acclimatization) or artificial/experimentally developed (heat acclimation) environments [65], with both eliciting similar physiological adaptations [75, 76] despite heat acclimatization proposed to provide more specific responses due to exposures in the exact conditions that the work task or competition will occur [3].

Heat Acclimatization

Seasonal temperature changes theoretically have the capacity to induce adaptation with ambient temperatures increasing during summer months. Given the forcing function for heat adaptation is a sufficient stimulus to elevate body temperature to critical values, it follows that if the interaction between environmental conditions, heat production and clothing is potent enough, a significant adaptive response will occur. However, unlike artificial

exposures where the magnitude of stimulus can be regulated with a climate chamber, a reliance on the prevailing climate is necessary to induce adaptation [77]. This limitation makes it challenging to design experiments that can effectively induce and subsequently evaluate heat acclimatization in a population group given the inherent variability in the climate and subsequent heat exposure between individuals.

The evidence to support seasonal acclimatization is mixed. During summer months compared to winter, students and athletes in Japan had a lower end-exposure core temperature and greater sweat rate after a 60-min lower leg hot water immersion trial [78, 79], while wildland firefighters showed a lower rectal temperature and rating of perceived exertion during a 60-min heat stress trial (walking at 50% $\dot{V}O_{2\ max}$) after a 4-month wildland fire season [80]. For Australia specifically, in a Mediterranean climate (Perth, Australia) sweat losses during 35 minutes of cycling at 40% $\dot{V}O_{2max}$ during summer (0.47±0.14 $L \cdot h^{-1}$) was significantly higher than during winter (0.41±0.17 $L \cdot h^{-1}$) for 29 male manual outdoor workers of different trades [81]. Conversely, research on high-level distance runners in the US and healthy adults in Canada did not demonstrate any differences in makers of heat acclimatization during an incremental running test or 90-min of cycling (60% $\dot{V}O_{2max}$) respectively. A recent review by Brown et al [77] further supports the concept of seasonal heat adaptations reporting that a mean summer WBGT of 25.2 ± 2.8℃ will induce an adaptive response in one or more physiological responses [77], however, included in the same review, the authors highlight the difficulty in standardizing exposures when trying to determine the independent influence of season on individual level of adaptation. Of the 29 studies included in the review, 69% did not provide information about environmental exposure leading into testing periods with testing protocols ranging from 20-min of recumbent cycling to 90-min of lower body hot water immersion at varying intensities [77].

Taken together, these data suggest that the propensity to adapt naturally to the heat is possible, but dependent on two key conditions being met. Firstly, that the ambient conditions of the geographic location must be conducive to facilitate adaptation and second, that the individual exposure to that environment must be sufficient. Additionally, through the manipulation of both metabolic activity and clothing worn, an individual can further augment their adaptation potential for a given ambient environment and individual exposure.

Heat Acclimation

Our understanding of heat acclimation originates from the mining industry [82-85]. Due to the prevalence of heat stroke in miners, screening tests were developed to assess the tolerance of each worker, then assign an appropriate protocol to acclimate them in preparation for the conditions expected [86]. Screening sessions were conducted in converted environmental chambers that simulated the hottest underground mining locations; 35°C wet bulb, 35°C dry bulb, with minimal airflow and a simulated work task (shovelling rock) for 60-min [82, 83]. Based on the workers final core temperature they were deemed heat tolerant (< 38.1°C) or intolerant (> 38.9°C) and assigned 4 or 14 days of acclimation respectively. Individuals with final core temperatures between 38.1°C and 38.9°C were assigned 7 heat acclimation sessions. This system was further developed by Wyndham, who more precisely controlled work rate with a block stepping task so that the tolerance testing and subsequent acclimation protocols could be adjusted for different body morphology, training status, and gender [84, 86]. These sessions were designed to match the working level more closely in the mines (1.0 L/min) and extended to 4 hours with sweat rate and heart rate incorporated into the models to predict tolerance [84, 85].

More recently, repeated heat exposures to improve thermal tolerance are primarily used for military, sporting, or occupational tasks [87-90]. Within this framework the

effectiveness of each heat acclimation regimen is dependent of the magnitude and rate of physiological change, however, there is little consensus on the optimal approach [64]. Although passive exposures have been shown to clearly induce adaptation [91, 92], and are viewed as potentially a more practical means of implementing heat acclimation in high-level athletes and clinical population groups [93-95], the addition of exercise with heat exposure provides additional strain that generally elicits more profound adaptations, more rapidly [3, 75]. Heat acclimation is generally achieved through four broad pathways: constant work rate exercise [96, 97]; self-paced exercise [98]; controlled hyperthermia [99, 100]; and controlled heart rate [56, 101] (Figure 3). Constant work rate exercise allows multiple individuals to acclimate concurrently, however, implements a constant forcing function which, as individuals progressively adapt, results in less physiological strain and a reduced adaptation [102]. By contrast, using a controlled hyperthermia approach that was first proposed by Fox [99] provides a heat stimulus that maintains a given core temperature (38.5℃) to progressively overload physiological adaptation as resting core temperature is reduced during acclimation [65]. Owing to this manipulation of the endogenous and/or exogenous thermal stress, the controlled hyperthermia method is often preferred as the method to most effectively induce significant adaptations to the heat [3]. In taking the principle of progressive overload and applying it to cardiovascular strain, a recent controlled intensity method that aims to maintain a given heart rate corresponding to a relative intensity (i.e., increase power output as adaptation occurs) has been proposed as an alternative that would provide a constant cardiovascular stimulus, based on the level of strain targeted [3, 101].

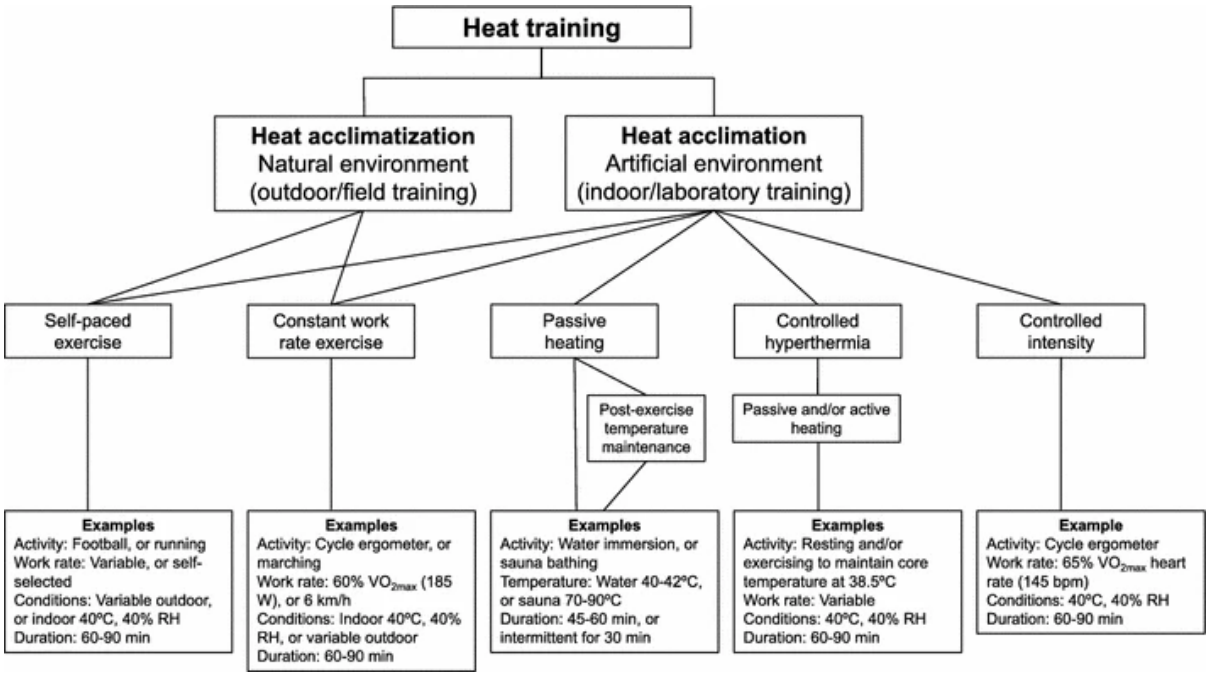

Figure 2.2 - *Figure reprinted with permission from Daanen et al. 2018 (Appendix C) [2]. Schematic overview of methods for heat acclimation and heat acclimatization, with examples. Various combinations of temperature and humidity are possible, as well as the use of portable heaters and wearing additional clothing.*

Time Course of Induction and Decay of Heat Adaptation

The time course of human heat acclimatization remains unclear given the challenges in quantifying the level and progressive effect of each exposure. Accordingly, this section will discuss the existing literature specifically pertaining to heat acclimation where the ability to control the environment allows appropriate comparisons between groups. The induction of heat acclimation begins after the first exposure, and it is generally accepted that complete adaptation will occur between 7 and 14 days of repeated exposures providing a sufficient stimulus that increases whole body temperature and sweating for a sustained period [3, 64, 103]. During this time, 75-80% of all adaptations are proposed to occur during the first 4-7 days [3, 87] (Figure 4). For the optimal duration of exposure, seminal work by Lind and Bass

demonstrated that heat exposures of 100 min/day were more effective at inducing changes in core body temperature and heart rate than two 50-min daily exposures, and that two 100-min daily exposures were no more effective than one [104]. Following on from this work, a timeframe of 100-min per day was proposed as the most effective to induce adaptation, however, several studies have shown significant improvements with shorter timeframes [105, 106]. Intermittent heat acclimation protocols have been proposed as a viable alternative in situations where fatigue and dehydration should be avoided [103], however, have been shown to elicit suboptimal adaptations when compared to daily exposures [107], with no adaptation in HR, core temperature or sweat rate if sessions are separated by 1-week [108]. It should also be noted that despite consistent reporting of the acclimation response at a cohort level, there is significant individual variability in the magnitude and time course of adaptation. These differences have previously been identified as high- and low responders [109, 110], however there is limited work showing what factors predispose individuals to either group. While body dimensions or $\dot{V}O_{2\ peak}$ did not explain differences in heat adaptation level, Alkemade et al [111] recently reported that larger individuals (high body mass, low surface area-to-mass ratio) were more likely to be high sudomotor responders (i.e. increased whole body sweat rate adaptation) while smaller individuals (low body mass, high surface area-to-mass ratio) were more likely to be high cardiovascular responders (i.e. increased heart rate adaptation).

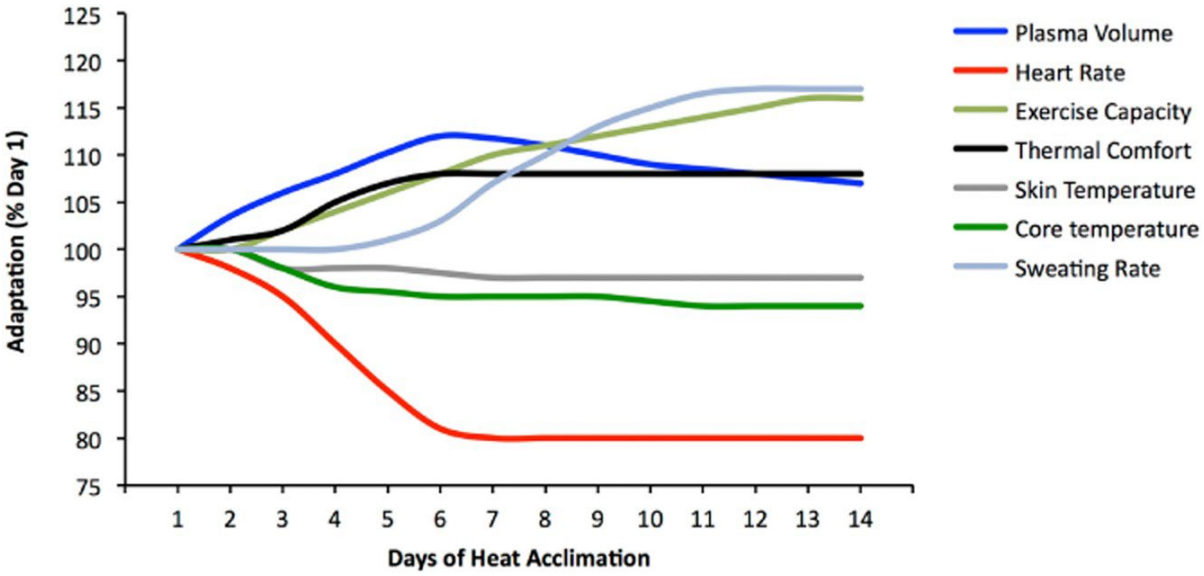

Figure 2.3 - *Figure reprinted with permission from Periard et al. 2015 (Appendix D)* [3].

Time course of induction in human adaptations to heat stress.

Our understanding on how humans progressively lose accumulated adaptation is more limited, with confounding variables during induction like the duration and type of acclimation, along with the ambient conditions during decay, rate of decay varies considerably between studies. Most of the available literature uses measures of core temperature, heart rate and sweat rate to document decay, with a stepwise multiple regression of 12 studies suggesting a decay of 2.5% in heart rate and core temperature adaptation for each day that was not deemed an acclimation day, provided that at least 5 acclimation days were completed prior to evoke stable adaptations [2]. The authors concluded that the decay rate was "strongly dependent on the number of acclimation days" and that changes in sweat rate were more sustainable with longer heat acclimation protocols [2]. Similar to inducing heat acclimation, high variability exists in the individual response to adaptation decay.

Conclusion

An understanding of how humans adapt to the heat provides us with key information around human health and performance. Identifying when and to what extent humans adapt naturally to their environment will help protect vulnerable groups from the increasing frequency and severity of heatwaves. Estimating key indicators of adaptation used to predict heat strain will help inform safe work policies and finding non-invasive, feasible strategies to assess adaptation will improve the accessibility of heat stress testing and allow findings from laboratories to be implemented in a broader population. Accordingly, the work presented in this thesis will focus on the following.

1. Model the magnitude and time-course of natural heat acclimatization for different occupational groups of varying exposure throughout the year across the eight capital cities of Australia.
2. Quantify the time-course of changes in a key marker of human adaptation to the heat (maximum skin wettedness or ω_{max}) throughout 7 consecutive days of heat acclimation.
3. Develop a non-invasive heat stress test capable of detecting changes in physiological adaptation status throughout 7 days of heat acclimation that could feasibly be implemented in an at-home setting.

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CHAPTER 3: EXPERIMENTAL STUDY ONE

Using the Predicted Heat Strain Model to Estimate the Magnitude and Time Course of Natural Heat Acclimatisation Throughout a Typical Summer in Australia

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Abstract

Purpose: To predict the magnitude and time-course of natural heat acclimatisation for different occupational groups of varying exposure throughout the year in Australia.

Methods: We retrieved hourly weather data for a representative year across the eight capital cities of Australia, then used the *Predicted Heat Strain (PHS)* Model that resides within the international standard for the assessment of occupational heat strain (ISO 7933) to predict maximum daily rectal temperature (T_{re}) for: 1) office workers in air-conditioning (IND-AC) buildings; or 2) office workers in naturally ventilated buildings (IND-NV); 3) outdoor labourers in the shade (OUT-SHA), 4) outdoor labourers in the sun (OUT-SUN); 5) outdoor labourers in the sun with elevated levels of physical activity (OUT-SUN-ACT); 6) outdoor labourers outdoors in the sun with elevated levels of physical activity wearing protective clothing (OUT-SUN-ACT-CLO). Using maximum daily T_{re}, we designed a simple scoring system to estimate a "natural heat acclimatisation score (NAS)" between 0 and 5 to show if groups were "not acclimatised" 0-2.9 (0%), "partially acclimatised" 3.0-3.9 (1-49%), "mostly acclimatised" 4.0-4.9 (50-99%) or "completely acclimatised" 5.0 (100%) each month throughout the year.

Results: IND-AC, IND-NV, and OUT-SHA are estimated to remain "unacclimatised" at all time points throughout the year irrespective of climate and geographical location. For OUT-SUN, OUT-SUN-ACT, and OUT-SUN-ACT-CLO, four of the eight capital cities were estimated to reach peak heat acclimatisation in February (Sydney, Melbourne, Perth, Brisbane), three cities in January (Adelaide, Canberra, Hobart), and one city in April (Darwin). The highest heat acclimatisation scores were always observed in OUT-SUN-ACT-CLO, with all locations (except Hobart) "mostly acclimatised" by the end of summer

(February) and two cities spending ≥ 6 months “mostly acclimatised” throughout the year (Darwin, Brisbane).

Conclusion: In Australia, office workers irrespective of climate do not experience natural heat acclimatisation at any point during the year with minimal exposure to the ambient environment. For outdoor labourers that work in the sun, most locations in Australia show a progressive increase in acclimatisation during summer months with a peak adaptation status typically reached by February. The extent of this estimated heat acclimatisation is limited unless workers elevate their physical activity and can be further augmented by wearing additional protective clothing.

Introduction

The importance of developing heat-health management systems to minimise the potential negative health effects of hot summer weather on occupational workers has become increasingly evident [1]. This is especially so in Australia, which mostly experiences long, hot summers, and whose economy has been estimated to annually suffer more than $6B in lost productivity due to heat stress [2].

A key component of managing heat stress risk, is characterising the level of naturally acquired physiological protection to the heat a worker may have at different times of the year through repeated recent heat stress exposures, i.e., heat acclimatisation [3]. Under the right conditions, the ability of the human body to physiologically adapt to the heat can be profound. Maximum sweat rate can be elevated substantially, while a lower core temperature at rest allows a greater level of body heating to occur before critical absolute body temperatures are reached. An expanded blood plasma volume also lowers heart rate during a given heat exposure and improves cardiovascular stability. Collectively these adaptations improve heat resilience and lower heat stress risk [1].

It is a widely held conviction by many epidemiologists that people naturally acclimatise to the heat throughout a given Australian summer [4-6]. However, it is unclear whether the stimulus for a physiological heat acclimatisation, i.e., combination of exposure to the natural climate, physical activity levels, and clothing worn, is sufficient to generate a complete, or even partial, adaptation to the heat. Even if heat acclimatisation does occur, the timeline for its full development, and therefore the acquisition of its protective benefits, is not clear. It is also unknown how these characteristics are altered by the large differences in prevailing climate across the various regions of Australia.

It is known from experiments conducted in controlled climate chambers (heat acclimation) [7-9], that at least 7 consecutive days of exposure to high temperatures and often humidity for at least 1-2 hours while performing moderate to vigorous exercise are required to induce a partial heat acclimation. Full heat acclimation usually requires 10 to 14 days of consecutive exposure [10], and these adaptations disappear at 1- to 2-times rate at which they are acquired [11]. Apart from people living in hot/humid regions of Australia performing physically active jobs, it therefore seems unlikely that a fully heat acclimatised state will be ever reached. In more temperate Australian regions, any partial heat acclimatisation may not be acquired until the middle of the summer, if at all.

There is mixed experimental work investigating the magnitude of natural acclimatisation directly, with marked physiological changes observed between summer and winter time points in Japanese men [12] and American soldiers [13], but not in Canadian office workers [14] or North-American distance runners [15]. For Australia specifically, in a Mediterranean climate (Perth, Australia) sweat losses during summer (0.47±0.14 $L \cdot h^{-1}$) were significantly higher than during winter (0.41±0.17 $L \cdot h^{-1}$) during 35 minutes of cycling (40% $\dot{V}O_{2max}$) for 29 male manual outdoor workers of different trades [16]. More recently, a review investigating the effect of seasonal heat acclimatisation on young, healthy adults concluded that, the magnitude of adaptation is highly dependent on a combination of environmental and physical activity characteristics including the duration and intensity of outdoor physical activity along with the timing of exposures [17]. From a physiological perspective, the resultant effects of heat adaptation via natural (acclimatisation) or artificial (acclimation) pathways ultimately serve to increase our ability to dissipate heat from our body. Consequently, if a heat exposure results in a similar physiological response (i.e. increase in core body temperature), the magnitude of adaptation, regardless of induction method, should also be similar.

Fundamentally, the characteristics that define human responses to a thermal environment are either environmental (air temperature, radiant temperature, humidity, air velocity) or personal (clothing, heat production) with the interaction of each parameter vital to predict how humans may behaviourally or physiologically compensate to a given stress [18]. For the ambient environment, as air temperature increases, the gradient between air and skin temperature decreases, limiting convective heat loss. If outdoors, the solar radiation from the sun can add additional heat to the body via radiation. In tropical climate zones (e.g. Northern Territory, Australia) the relative increase in water vapour in the air limits the our ability to liberate secreted sweat from the skin surface, preventing our bodies from effectively cooling through evaporation [18]. Air flow influences heat flow to and from the body and hence body temperature, with increased air flow facilitating a greater cooling capacity through increased evaporation provided there is sufficient sweat pooled on the skin surface [19]. For personal parameters, the two clothing characteristics that influence heat transfer from the skin to the environment are the dry insulation of clothing (i.e. the resistance to heat transfer between the skin and the clothing surface) and the evaporative resistance of clothing (i.e. the resistance for sweat to transport through the clothing to the external environment) where the ability to successfully dissipate heat from the body to the surrounding environment is mitigated as the dry insulative and evaporative resistance of the clothing worn increases [18, 20]. Heat production is the rate of energy production (energy expenditure) minus the external work performed to complete an activity or task. As the physical demands of a task increase, body temperature will increase in response to the greater engagement of skeletal muscles to perform work, releasing a substantial amount of heat [21]. If the cumulative effect of each parameter creates an environment that challenges the thermoregulatory system sufficiently and consistently, natural adaptation to the heat is more likely to occur.

Globally, air-conditioning is widely considered the most widespread and effective strategy to mitigate against adverse events during heatwaves [22], with the purchase of personal and industry units rapidly rising as living standards continue to improve. This improvement has led to a predicted 700 million new units available by 2030 [23] and while effective at lowering the local temperature of the indoor environment, has a substantial economic cost [2] and limits exposure to the ambient environment, with large groups of the population engaging in behavioural responses to hot weather by employing air-conditioning in their offices, cars and homes. Equally, as Australia and the world increasingly shift to a more sedentary lifestyle [24], limiting time spent engaging in activities that increase metabolic rate and the concurrent sweating response could also minimize the potential for natural heat acclimatisation.

In this chapter of my thesis, we sought to model the independent influence of different environmental and personal parameters on predicted heat acclimatisation status throughout a representative year in Australia. We developed six profiles to characterize indoor and outdoor workers at varying levels of natural exposure to the ambient environment. By retrieving hourly weather data over a 12-month period for the eight capital cities of Australia and inputting the prevailing conditions into an adapted Predicted Heat Strain model (ISO 7933), we estimated daily thermal strain and an associated "acclimatisation score", reported as a 7-day rolling average to estimate the natural adaptation for different occupational groups throughout a calendar year.

Methods

Individual Profiles

We created six unique worker profiles to assess the potential development of natural heat acclimatisation throughout a representative year in the eight state and territorial capital cities of Australia.

These profiles were:

1) Office workers in an air-conditioned space during their working hours (IND-AC)
2) Office workers that work in a naturally ventilated building (IND-NV)
3) Outdoor labourers that work in the shade (OUT-SHA)
4) Outdoor labourers that work in the sun (OUT-SUN)
5) Outdoor labourers that work in the sun and engage in regular physical activity outside of working hours (OUT-SUN-ACT)
6) Outdoor labourers that work in the sun with more protective that engage in regular physical activity outside of working hours (OUT-SUN-ACT-CLO).

Each profile estimated responses for an average size Australian male (weight: 88.3 kg; height: 1.79 m; BSA: 2.07 m^2) [25] and for "awake" hours during a standardized week (06:00 – 22:00).

Indoor workers (IND-AC, IND-NV) were office-based occupations that, on average, completed minimal activity during the work-day (seated desk work: 1.7 METs) [26]. Outdoor workers (OUT-SHA, OUT-SUN, OUT-SUN-ACT, OUT-SUN-ACT-CLO) were given an hourly heat production (3.9 METs) during the work-day to align with the average energy cost of a job based in the agriculture industry including work tasks like harvesting, planting and striking/shovelling the ground [27]. OUT-SUN-ACT and OUT-SUN-ACT-CLO both

completed 1 hour of moderate intensity exercise (4.4 METs), equivalent to a brisk walk each day (approximately 6 $km \cdot h^{-1}$) to align with Australian minimum physical activity guidelines [28]. All other groups did not engage in additional physical activity outside of their work task requirements. Designated working hours for indoor workers were 09:00-17:00 and 07:00-15:00 for outdoor workers. Standardized clothing was assigned during working hours for OUT-SHA, OUT-SUN, and OUT-SUN-ACT (Kansas State Uniform, Icl: 0.57 clo (sitting), 0.68 clo (standing),) with OUT-SUN-ACT-CLO assigned the ASTM standard reference uniform (shirt not tucked into (over) pants, shirt top button open; Icl: 0.93 clo (sitting), 0.90 clo (standing)) [29, 30]. All other conditions had the amount of clothing worn predicted based on the ambient temperature at the start of the day (6AM) [31]. Outside of designated work or exercise, no other exposure either physical or outdoors was assigned.

Weather Data

To contextualise the conditions for each occupational group, hourly environmental data from airport weather stations of each capital city in Australia (8 locations: Adelaide, South Australia; Brisbane, Queensland; Canberra, Australian Capital Territory; Darwin, Northern Territory; Hobart, Tasmania; Melbourne, Victoria; Perth, Western Australia; Sydney, New South Wales) were obtained through a freely available web-based application built to support climate scientists by providing Energy Plus Weather files for 27,500 locations worldwide [32]. Ambient temperature, solar radiation, relative humidity, and air velocity were extracted for each hour of a Typical Meteorological Year (TMY) for each regional location, where hourly data that best represents median weather conditions over a multiyear period are presented as the most usual conditions. For example, a dataset that is available from 2010-2020 might use data from 2012 for January, 2018 for February, and so on. Given air velocity values are reported from 10 m above ground level, a correction factor for various terrain characteristics was applied to approximate values experienced while working at

approximately ground level (2 m) [33]. Mean radiant temperature was estimated using the ISO 7726 standard [34]. A climate summary of each location is presented in Table 1.

Predicted Heat Strain

Hourly climate data for each location were combined with the individual characteristics of each profile and input into a model to predict rectal temperature (T_{re}) throughout the day [35]. Each simulated day started with a T_{re} of 36.8℃ and recorded changes every hour with the highest absolute hourly T_{re} achieved during each day (1dp) extracted and assigned a daily heat exposure score (DS) from 0-5. The DS associated with increasing T_{re} were: DS = 0 (36.8℃), DS = 1 (37.1℃), DS = 2 (37.2℃), DS = 3 (37.6℃), DS = 4 (38.0℃), and DS = 5 (38.5℃) [36-38]. Between each DS score our model assumed a linear relationship and that the time course of acclimatisation followed existing acclimation data in that "complete adaptation" could occur with an elevation in T_{re} to 38.5℃ for 7 consecutive days [10, 37, 39] and that anything less than a 0.8℃ increase in T_{re} (absolute T_{re} of 37.6℃) would not be sufficient to induce sustained physiological adaptations [37, 38]. To determine natural acclimatisation score (NAS) a rolling average of each groups previous seven DS was calculated to account for the accumulated effect of repeated exposures of a significant magnitude on the onset and decay of adaptation to the heat [10, 39]. If NAS was < 3, the group was considered "not acclimatised", an NAS between 3 and 3.9 was "partially acclimatised", an NAS between 4 and 4.9 was "mostly acclimatised" and an NAS = 5 was "completely acclimatised".

The primary outcome of interest was average monthly NAS for each location (as a percentage between NAS = 3 (0%), NAS = 4 (50%), and NAS = 5 (100%)) to observe both the magnitude and time-course of natural acclimatisation. Secondary outcomes of interest

were the number of days spent partially, mostly, or completely acclimatised throughout the year and the peak NAS for each regional location.

Results

Office Workers

Our model estimates that for both groups of office workers (IND-AC, IND-NV) there was not a sufficient thermal stimulus to naturally adapt, meaning that both groups remained "unacclimatised" (NAS < 3) at all time points throughout a typical meteorological year, irrespective of climate or geographical location.

Outdoor Labourers

National

Pooling the data from all eight locations to create a national, unweighted average shows that outdoor labourers working in the shade, similarly to office workers remain "unacclimatised" at all points throughout the year. This average also shows that peak natural acclimatisation for all other groups (OUT-SUN, OUT-SUN-ACT, OUT-SUN-ACT-CLO) occurs at the end of summer (February) with OUT-SUN "partially acclimatised" at 6% (NAS = 3.12), OUT-SUN-ACT "mostly acclimatised" at 56% (NAS = 4.11), and OUT-SUN-ACT-CLO "mostly acclimatised" at 63% (NAS = 4.26) (Figure 1).

Regional

Regional differences in the time course and magnitude of natural acclimatisation are shown in Figure 2. Peak acclimatisation for all locations was in the OUT-SUN-ACT-CLO group and occurred at the end of summer (February) for Sydney at 65% (NAS = 4.3), Melbourne at 55% (NAS = 4.1), Perth at 81% (NAS = 4.6), and Brisbane at 67% (NAS = 4.3) while Adelaide at 67% (NAS = 4.3), Hobart at 29% (NAS = 3.6), and Canberra at 63% (NAS = 4.3) peaked during January. Darwin peaked at 97% (NAS = 4.9) during April.

For OUT-SUN, none of the cities predicted the group would be “mostly acclimatised” at any point throughout the year. Locations that estimated “partial acclimatisation” for consecutive months were Canberra (December – February), Adelaide (December – March), Perth (December – March), and Darwin (July – June) with the maximum percentage attained at each location 17%, 20%, 22%, and 42% respectively.

For OUT-SUN-ACT, our model predicted that two of the eight locations (Melbourne and Hobart) would not be able to “mostly acclimatise” the group at any point throughout the year with an estimated maximum of 23% in Hobart and 43% in Melbourne. For the other cities, the group was estimated to be “mostly acclimatised” in Sydney during February (53%), Canberra from December to February (min: 55%; max: 59%), Adelaide from January to March (min: 51%, max: 59%), Perth between January to March (min: 65%; max: 73%), Darwin for all 12 months of the year (min: 74%; max: 97%), and Brisbane from November till March (min: 50%; max: 58%).

There were small differences between OUT-SUN-ACT and OUT-SUN-ACT-CLO where in Sydney, Melbourne, and Perth the group was predicted to be “mostly” not “partially” acclimatised in December (Brisbane), January (Sydney), February (Melbourne) and April (Sydney, Brisbane).

Discussion

In this study, we developed profiles of representative occupational groups to model natural heat acclimatisation status throughout a representative year in Australia. We used a well-validated model of assessing heat strain used by the International Standardisation organisation (ISO) in occupational settings to predict core temperature responses each day during a 12-month period. The maximum core temperature achieved each day was attributed with a heat exposure score ranging from 0 to 5 that was averaged over 7 consecutive days as a natural acclimatisation score, to capture the acquisition and waning of heat acclimatisation with varying heat exposure. Collectively, this approach showcases the potential effect varying levels of natural exposure throughout a typical year in Australia have on heat adaptation throughout the year, particularly during summer months. Based on our analysis, natural heat acclimatisation does not occur if the occupation takes place predominantly indoors or outdoors in the shade. In contrast, our findings suggest that the additional radiant load from the sun can elicit sufficient heat strain to naturally acclimatise depending on the prevailing climate. This response is estimated to be augmented, irrespective of climate, by performing additional physical activity outside of working hours, and further augmented by wearing more protective work clothing. Taking an unweighted average of the eight cities tested, our model predicted that an increase in natural acclimatisation typically began at the start of spring (September), reaching a peak at the end of summer (February) and, depending on location, maintained until the end of Autumn (May).

Regionally, Australia experiences a variety of climatic conditions, from the hot humid tropics in the northern parts of Queensland and Western Australia as well as the Top End of the Northern Territory, through the hot and arid/semi-arid central deserts of the Outback, to the temperate coastal fringes of Western Australia, South Australia, Victoria, New South

Wales and Tasmania [40]. According to the Köppen classification the eight capital cities can be classified as either; tropical savanna (Darwin), Mediterranean (Perth, Adelaide), humid sub-tropical (Sydney, Brisbane), oceanic (Canberra), and temperate oceanic (Melbourne, Hobart) [41]. Accordingly, our model shows that each city tested has a unique profile for the onset, maintenance and decay of acclimatisation that fits the climate characteristics for each city. Darwin, located in the far north of central Australia, has two distinct seasons where conditions are either hot-humid (November – April) or hot-dry (May – October) and monthly mean afternoon (3PM) temperatures of between 30-32°C with high humidity [42]. These conditions mean that the potential to induce and maintain acclimatisation is very high and our model highlights this with both OUT-SUN-ACT and OUT-SUN-ACT-CLO remaining "mostly acclimatised" for all 12 months of the year. Perth, in the south-west of Australia, however, is a unique location in that it is acutely at risk of the negative impacts of climate change because the city experiences easterly winds brought about by high-pressure systems that travel over the hot-dry desert landscape [43]. Consequently, its acclimatisation profile has a sharp increase in natural acclimatisation during the summer months that peaks in February at 22%, 73%, and 81% for OUT-SUN, OUT-SUN-ACT, and OUT-SUN-ACT-CLO respectively. According to our model, Sydney, Canberra, Melbourne, and Adelaide despite having different weather profiles, largely generate a similar magnitude of natural adaptation. In Sydney and Melbourne, the increase in acclimatisation status is more gradual from October through until February whereas in Canberra and Adelaide the increase is more pronounced, with Canberra increasing from 22% to 59% between November and December, and Adelaide from 42% to 59% between December and January. Brisbane is characterized by very sunny, humid summers combined with mild, dry winters [44] and according to our model, these characteristics make it somewhat like Darwin in that for extended periods of the year (October – April) the environmental conditions are sufficiently stressful to facilitate

acclimatisation provided there is enough exposure. Hobart is classified as a temperate climate and was the only location that our model predicted would not be viable to "mostly acclimatise" at least one occupational group throughout the year. As the average monthly maximum temperatures in Hobart range from 12-21°C it appears that the conditions are not sufficient to drive adaptation.

Reductions in the perceived risk of heat-related illness during extreme heat and the development of exertional heat illness from occupational and sporting tasks throughout a given year are often ascribed to individuals naturally adapting as the weather becomes progressively hotter through the summer [45-48]. While there is growing evidence to support natural heat acclimatisation in young, healthy adults, the magnitude of adaptation is shown to be highly dependent on environmental and physical activity characteristics, including the duration and intensity of activity and the timing of exposures [17]. Our model estimates that progressive acclimatisation can occur during summer months, however, this adaptation would only be evident with a large heat stimulus that far exceeds the level of natural exposure for most working Australians. The stimulus modelled in the current study combines a physically-demanding job in the Agriculture industry [27], cited as one of the industries most affected by heat stress [49], with regular bouts of additional physical activity, to demonstrate the likely necessary exposure to acquire significant adaptation. Both OUT-SUN-ACT and OUT-SUN-ACT-CLO profiles had 35 working hours and 7 exercising hours per week exposed to the natural environment (2520 min/week). Our model estimates that daily lifestyle choices that allow modern behavioural adaptations (e.g. air-conditioning use) have a substantial impact on the potential for adaptation to the heat, a notion previously proposed in military and general population settings [13, 14].

The time-course of the physiological responses associated with heat adaptation have been well-established [7, 10, 39], with the potential to achieve approximately complete heat

acclimation between 7-14 days of consecutive exposure [50, 51]. Improvement in tolerance from repeated exposures to the heat require a sustained elevation in core temperature that predominantly increases maximum capacity to sweat, altering the boundary between compensable and uncompensable heat stress [52]. As the forcing function to improve sweating capacity theoretically does not change irrespective of induction method, physiological acclimation and acclimatisation were treated as synonymous for this study. Accordingly, our model used the minimum forcing function established in the literature to quantify exposure and theoretically achieve complete adaptation with a core temperature increase to 38.5℃ for seven consecutive days [7, 10]. To date, there is no experimental work to the best of our knowledge that compares the adaptive response between acclimation and acclimatisation given the inherent practical issues with standardizing exposure. Additionally, there is no consensus on the optimal method to accurately assess adaptation status, particularly in field settings. Verification of these findings in field-based trials that involve regular assessment of individual adaptation to the heat is required.

Although the models used to predict core temperature responses and subsequent thermal strain are well validated, these data should be interpreted with caution without accompanying experimental data. The findings from our model are consistent with more recent observations [17] that seasonal acclimatisation can occur provided sufficient exposure to heat, however, our model was fixed for the average energy cost of a pooled analysis of 14 studies that measured 137 agriculture work tasks with indirect calorimetry [27]. Differences in the adaptation response will vary based on individual activity level, including the specific work task being performed, and the properties of the clothing worn and therefore, should not be extrapolated to other occupations in different geographic locations. All climate data in this study were collected from airport weather stations in each city, and thus differences between airport, inner city and more regional locations will exist. Data collected from airport

meteorological stations allow national and international comparisons due to standardized instrumentation ensuring homogeneity of data. However, airports are often located outside of city centres where they are not subjected to densely populated urban areas and the accompanying urban heat island effect [53]. Given the work task for our model was a job in the agriculture industry, airport stations presented the most available data that closely matched the environmental conditions expected [33].

Conclusion

In Australia, both office workers and outdoor labourers that work in the shade, irrespective of climate are not predicted to naturally heat acclimatise at any point during the year with minimal exposure to the ambient environment. Outdoor labourers that work in the sun (e.g., farmers in the agriculture sector) can progressively acclimatise in most locations in Australia with peak acclimatisation typically occurring at the end of summer in February. The extent of this heat acclimatisation is limited unless workers elevate their physical activity and can be further augmented by wearing additional protective clothing including long-sleeves, coveralls etc.

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Table 3.1 – *Climate summary of a typical meteorological year for cities used to estimate natural acclimatisation status.*

Location	**Coordinates**		**Climate Classification**	**Temperature**			
City	Longitude	Latitude	Köppen-Geiger Climate Zone	Average (°C)	99th Percentile (°C)	1st Percentile (°C)	Maximum (°C)
Sydney	151.173	-33.946	Humid subtropical, no dry season	17.8	30.5	7.0	40.6
Canberra	149.195	-35.307	Marine west coast, warm summer	13.5	32.7	-2.0	38.4
Melbourne	144.832	-37.666	Marine west coast, warm summer	14.2	32.0	4.0	37.0
Adelaide	138.520	-34.953	Mediterranean, warm summer	16.6	32.7	5.5	37.8
Perth	115.967	-31.940	Mediterranean, hot summer	18.5	35.4	5.9	40.6
Darwin	130.893	-12.424	Tropical wet and dry or savanna	27.3	33.2	19.0	36.0
Brisbane	153.116	-27.407	Humid subtropical, no dry season	20.5	29.1	8.0	32.7
Hobart	147.510	-42.836	Marine west coast, warm summer	13.0	26.4	3.0	36.1

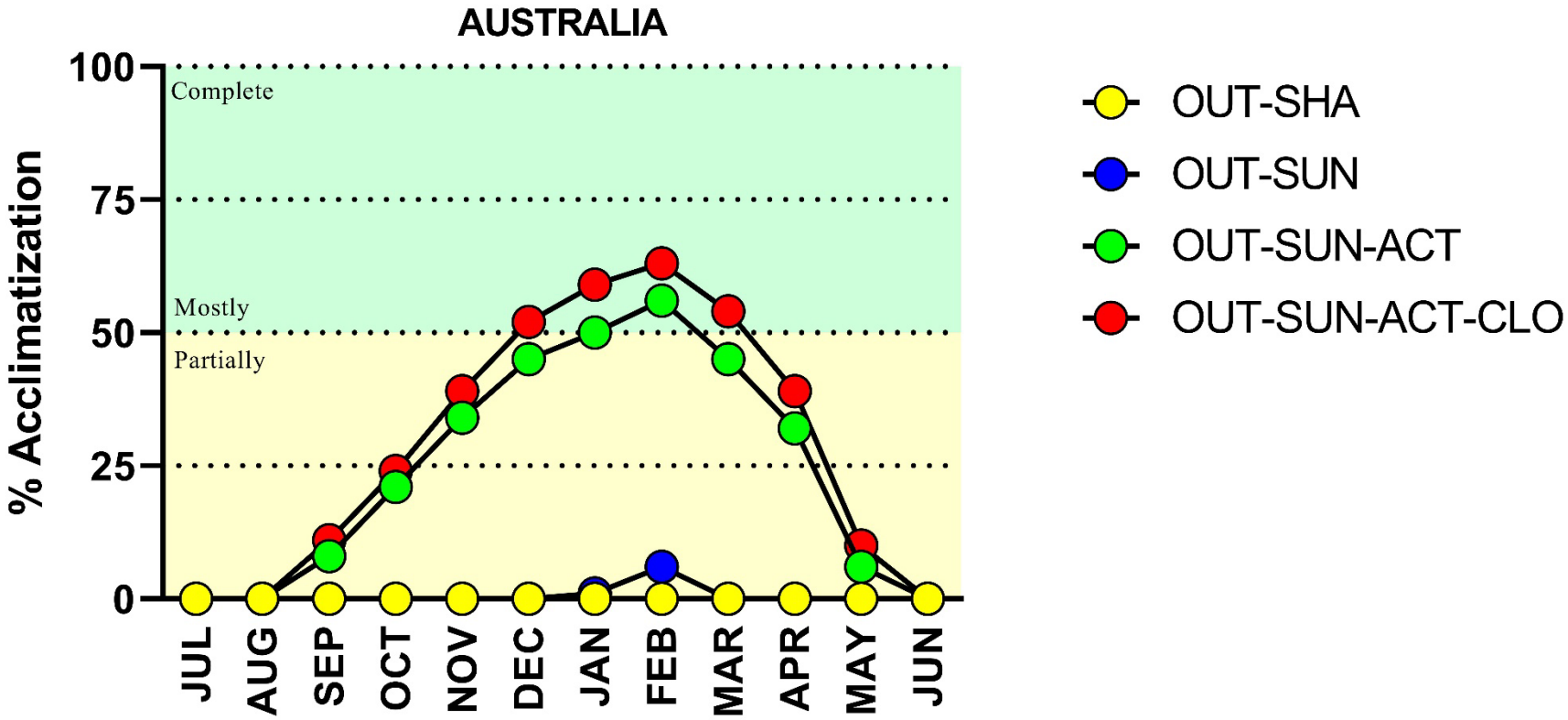

Figure 3.1 – *Average percentage of natural acclimatization reached over a representative year in Australia when working; outdoors in the shade (OUT-SHA), outdoors in the sun (OUT-SUN), outdoors in the sun with additional exercise (OUT-SUN-ACT), outdoors in the sun with additional exercise and heavier clothing (OUT-SUN-ACT-CLO). Partially acclimatized (Partially - shaded yellow), mostly acclimatized (Mostly - shaded green) and completely acclimatized (100%).*

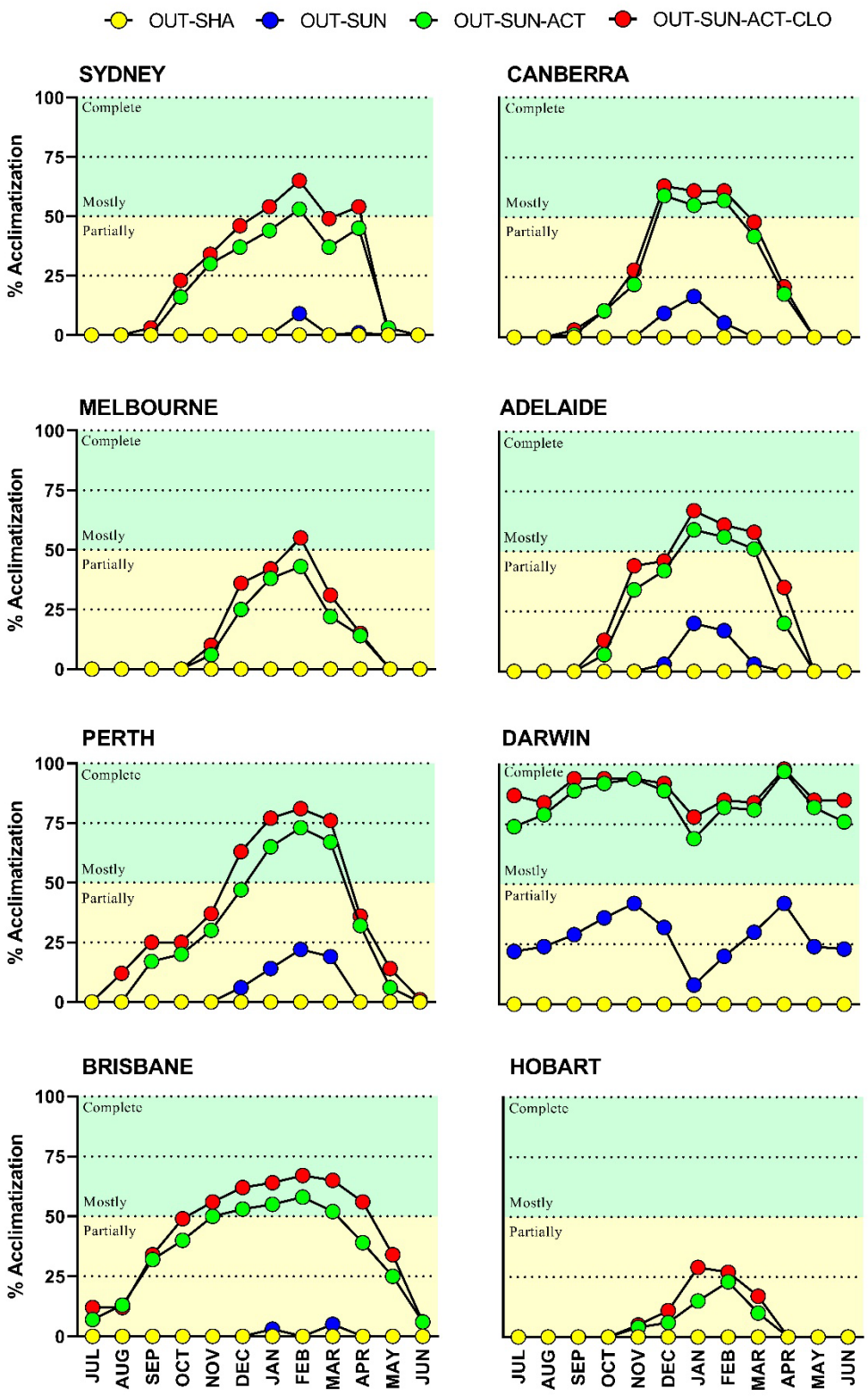

Figure 3.2 – *Regional variation in natural acclimatization during a representative year when working; outdoors in the shade (OUT-SHA), outdoors in the sun (OUT-SUN), outdoors in the sun with additional exercise (OUT-SUN-ACT), outdoors in the sun with additional exercise and heavier clothing (OUT-SUN-ACT-CLO). Partially acclimatized (Partially - shaded yellow), mostly acclimatized (Mostly - shaded green) and completely acclimatized (100%).*

CHAPTER 4: EXPERIMENTAL STUDY TWO

Quantifying the Time-course of Changes in Maximal Skin Wettedness with 7 days of Heat Acclimation

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Abstract

Purpose: To quantify the time-course of changes in maximum skin wettedness (ω_{max}) – i.e., the proportion of skin surface area covered in sweat at the point of uncompensable heat stress (0.06 = completely dry skin, 1.00 = the entire skin surface completely saturated with sweat; no units), throughout 7 consecutive days of heat acclimation.

Methods: Eight healthy adults (5M, 3F) completed a humidity-ramp protocol (RAMP) on days 1, 3, 5 and 7 of seven consecutive days of heat acclimation. In each RAMP trial, participants cycled continuously at 275 $W \cdot m^{-2}$ for 120 min at 37˚C: 60-min at a vapour pressure of 2.05 kPa (35% RH) followed by 60-min with vapour pressure increased by 0.045 $kPa \cdot min^{-1}$. An upward inflection in esophageal temperature (T_{eso}) signalled a transition from compensable to uncompensable heat stress with the critical vapour pressure at this point used to calculate ω_{max}. In the days between RAMP assessments participants cycled for 90-min at 75% age-predicted HR_{max} at 37˚C, 60% RH. T_{eso}, rectal temperature (T_{rec}), heart rate (HR), whole-body sweat rate (WBSR), activated sweat gland density ($AGSD_{back}$), skin temperature (T_{sk}), thermal sensation (TS) and rating of perceived exertion (RPE) were measured throughout.

Results: ω_{max} values were progressively and significantly greater from Day 1 (0.65±0.08) to Day 3 (0.73±0.09; P<0.01), to Day 5 (0.78±.10; P=0.01), to Day 7 (0.87±0.06; P=0.01) of heat acclimation. Compared to Day 1 (0.91±0.19 $L \cdot h^{-1}$), WBSR was not different on Day 3 (0.98±0.26 $L \cdot h^{-1}$; P=0.16), however, was higher on Day 5 (1.03±0.22 $L \cdot h^{-1}$; P=0.01) and Day 7 (1.07±0.15 $L \cdot h^{-1}$; P<0.01). Activated sweat gland density on the back ($AGSD_{back}$) increased from Day 1 (65±13 $glands \cdot cm^{-2}$) to Day 3 (72±19 $glands \cdot cm^{-2}$; P=0.03), Day 5 (78±16 $glands \cdot cm^{-2}$; P<0.01), and Day 7 (82±18 $glands \cdot cm^{-2}$; P<0.01). No differences were observed in sweat gland output (SGO_{back}) (P=0.22).

Conclusion: ω_{max} significantly increased throughout 7 days of heat acclimation. Progressive increases were predominantly mediated by an increase in the number of active sweat glands, not the output per gland. These data are the first to report dynamic changes in ω_{max} with progressive heat acclimation and provide new information about ω_{max} levels between the standard "unacclimated" and "acclimated" states in existing heat stress models.

Introduction

During heat stress, humans maintain thermal homeostasis by dissipating heat from the skin surface to the surrounding environment at the same rate at which heat is internally generated [1]. A prolonged disturbance in human heat balance results in an accumulation of internal heat storage, causing body temperatures to increase. Eventually, body temperatures may be reached that are sufficiently high to elicit damaging and potentially fatal conditions such as exertional heat stroke [2].

A common strategy to reduce the risk of exertional heat illnesses is to engage in repeated daily bouts of exercise heat stress exposures over an extended period to elicit a series of physiological heat adaptations (i.e., heat acclimation) [3]. Typically, these exposures require an individual to exercise for >60-90 minutes in hot ambient conditions for a minimum of 7 consecutive days. Physiological heat acclimation is evidenced by a lower (~0.3°C) resting core temperature, a higher maximum (~25%) sweat rate, and greater cardiovascular stability (~10 bpm lower heart rate) predominantly supported by an expanded (~4-15%) plasma volume [4]. A key biophysical adaptation with heat acclimation is the modification of the boundary between compensable and uncompensable heat stress, secondary to an alteration in an individual's maximum evaporative capacity (E_{max}). This alteration is mediated by a change in a person's maximal skin wettedness (ω_{max}) – i.e., the maximum proportion of skin surface area that can be covered in sweat (1.00 = the entire skin surface completely saturated with sweat).

Maximum skin wettedness values are important because they are used to model heat stress risk for active people in hot environments (e.g., occupational settings). Currently, international heat stress standards (e.g., ISO 7933) use two fixed values for ω_{max}. Values of 0.85 and 1.00 are assigned to "unacclimated" and "acclimated" individuals to the heat, respectively [5]. These values are supported by seminal work conducted in the 1970's [6], but

with a modest sample size (n=4), and with participants resting supine, which may have artificially increased ω_{max} for a given environmental stress because sweat can accumulate more completely without dripping when laying supine compared to an upright posture [7]. Using a binary input system in reference to heat "acclimation status" also does not account for changes in adaptation status as individuals are progressively exposed to the heat. Without an understanding of the time course of the individual transition from "unacclimated" to "acclimated", an accurate assessment of an individual's heat stress risk *during* the acclimation process is challenging.

The time-course for induction of most of the physiological characteristics of heat acclimation is well established, particularly in sporting populations [8-10]. Previous work has proposed that 75-80% of total human physiological adaptation to the heat occurs in the first week of repeated exposures [8, 11]. However, the time-course of changes in the biophysical characteristic of ω_{max} – and thus the boundary of uncompensability - is not well understood. Previous work from our laboratory demonstrated the independent influence of 8 weeks of aerobic training followed by 8 days of heat acclimation on ω_{max} with mean values of 0.72 (pre-training), 0.84 (post-training) and 0.95 (post-heat acclimation) [12]. These increases were predominantly mediated by a more even and complete coverage of sweat across the skin surface, with the amount of active sweat glands (ASGD) increasing from pre-training to post-training, to post-acclimation, without a concurrent increase in the amount of sweat produced by each gland.

The purpose of the present study was to quantify the time course of changes in ω_{max} throughout 7 consecutive days of heat acclimation. We compared the critical environmental limits (used to calculate ω_{max}) for eight healthy volunteers using a humidity ramp protocol on alternate days (1, 3, 5, and 7) and a 90-min fixed heart rate exercise heat stress protocol (days 2, 4, and 6) to facilitate consistent heat adaptation [12]. We hypothesized that ω_{max} would

progressively increase with heat acclimation until Day 7, and ii) this adaptation would be predominantly mediated by an increase in heat-activated sweat gland density (ASGD).

Methods

Participants

A threshold for a meaningful difference in ω_{max} within participant between testing days was defined as 0.05 (i.e.,5%). Based on comparable research [12], an effect size of 0.90 was determined. Using G*Power 3 software and with an alpha set to 0.05 and a beta value of 0.1, a power calculation determined a minimum sample size of 16 participants free from cardiovascular and metabolic health disorders.

Approval for the project was obtained from The University of Sydney Human Research Ethics Committee (HREC#:2021/233) and written informed consent was acquired from all participants before starting the study. At present, thirty-one participants have been recruited to participate in the study. Of the thirty-one, eighteen withdrew after an extended lab closure, owing to COVID-19, that mandated a cessation of research activity between June 2021 and February 2022. Three cited issues with the invasive measures taken and another three cited time commitment as the reason to discontinue the project. Accordingly, 8 participants (Male: 5, Female: 3) completed the study (age: 27 ± 5 years; height: 177 ± 8 cm; weight: 71.9 ± 8.0 kg; body surface area (BSA): 1.88 ± 0.14 m^2; body mass index (BMI): 23.0 ± 1.5 $kg \cdot m^{-2}$; HR_{max}: 194 ± 4 bpm; $\dot{V}O_{2peak}$: 44.6 ± 6.7 $ml \cdot kg^{-1} \cdot min^{-1}$; activity level: 2482 ± 1045 $METmin \cdot week^{-1}$).

Instrumentation

Esophageal (T_{eso}) and rectal (T_{rec}) temperature were measured using general purpose thermistor probes (400TM; Covidien, Mansfield, MA, USA) inserted through the nasal cavity into the esophagus, and 15 cm past the anal sphincter into the rectum, respectively.

Skin temperatures were measured with eight wireless temperature sensors (Buttons DS1921H-F5, Embedded Data Systems, USA) fixed to the forehead (centre), chest (left, upper), scapula (right), deltoid (right), triceps (left), hand (left), thigh (right), calf (left). Mean

skin temperature (T_{sk}) was calculated according to the international standard for the measurement of body temperature [13].

Heart rate was measured by telemetry with a watch and accompanying strap (Polar Ignite, Kempele, Finland).

Local sweat rate on the back (LSR_{back}) and forearm (LSR_{arm}) were measured using ventilated sweat capsules secured to the skin using surgical tape (Transpore, 3M). Instrument grade dry air was passed through each capsule (4 cm^2) and flow was maintained at a constant rate of 500 $ml \cdot min^{-1}$. Local sweat rate was calculated by multiplying the absolute humidity of effluent air and the flow rate, then dividing by the amount of skin surface covered by the capsule, expressed per minute ($mg \cdot cm^{-2} \cdot min^{-1}$).

Activated sweat gland density was taken on the back (ASGD) using a modified iodine-paper technique described previously Randall [14] and modified by Davis [15] and Gagnon [16]. 100% cotton paper (Moab Entrada; Legion Paper, New York, USA) was cut to a predetermined size (9 cm^2) and placed into a sealed container contained iodine in solid form. Each piece of paper was elevated so that it did not make direct contact with the iodine. The paper was soaked for 2 h to allow saturation before being placed on the skin (5 s) to obtain an impression of density every 15-min during exercise. Sweat gland output (SGO) was calculated by dividing LSR_{back} by ASGD.

Rating of perceived exertion (RPE) was asked using a 6-20 Borg scale [17] while thermal sensation (TS) was asked with a modified visual analogue scale (0-240 mm) with anchor points ranging from “slightly cool” to “extremely hot”. Both measures were taken every 15-min during exercise.

Experimental Design

Participants completed a preliminary testing session involving a sub-maximal and maximal aerobic capacity test to calculate external workload for future sessions and $\dot{V}O_{2peak}$

respectively. The preliminary session was followed by seven consecutive days of heat acclimation involving alternate days of either a humidity ramp test (HEAT-RAMP) or a fixed heart rate exercise session (HEAT-TRAIN) (Figure 1). All trials for each participant started at the same time of day (± 1 h) to minimize the influence of changes in body temperature during natural circadian rhythm [18]. Participants were asked to refrain from alcohol, caffeine, and strenuous physical exercise for 24 h prior and during the 7-day protocol, as well as consume a standardized breakfast each day. The current project did not control for stage of menstrual cycle for female participants. When entering the laboratory for each experimental session participants provided a mid-stream, first-pass urine sample to confirm euhydration (specific gravity <1.020; if >1.020, 300 ml of tepid water was consumed). Participants were then instructed to change into standardized clothing (shorts, socks, and shoes (males) or sports bra, shorts, socks, and shoes (females)), self-insert a rectal thermistor and attach a heart rate strap. Participants then completed a "baseline" assessment involving 5 minutes of seated rest where rectal temperature, heart rate and blood pressure were recorded. Next, participants had their nude body mass measured in triplicate (Mettler, Toledo) before instrumentation, and then transferred into the climate chamber for their pre-exercise resting measures and exercise session.

Protocol - Preliminary Session

During the preliminary session participants first provided a detailed account of their physical activity level using a standardised self-report questionnaire [19] that was used to calculate average activity level in the week prior to completing the study. After completing the questionnaire, participants had their nude body mass measured on a platform scale and height measured with a wall-mounted stadiometer. After confirming euhydration, participants first completed a sub-maximal oxygen consumption test on a cycle ergometer (Corival, Lode, Netherlands) involving three 4-min stages (60, 90 and 120 W) used to determine the external

workload required to elicit a fixed heat production (275 W/m^2) for subsequent RAMP trials [20]. Following a 10-min break, participants then completed a maximal aerobic capacity ($\dot{V}O_{2peak}$) test on the same cycle ergometer set to an external workload of 120 W and increased incrementally by 20 W every minute until volitional exhaustion [21]. Breath-by-breath oxygen consumption was measured using indirect calorimetry via a metabolic cart (Quark, CPET, Cosmed, Australia).

Protocol - Humidity Ramp Trial (RAMP)

After instrumentation, participants completed 10 minutes of seated rest on a cycle ergometer followed by continuous cycling at a fixed metabolic heat production (H_{prod}) of 275 W/m^2 for 120 minutes at 37℃: 60-min at a fixed vapour pressure of 2.05 kPa (35% RH) followed by 60-min with vapour pressure increased by 0.045 kPa·min^{-1}. After achieving a steady-state T_{eso} temperature during the first 60-min of exercise, defined as less than a 0.05℃ increase in esophageal temperature (T_{eso}) in the preceding 10-min, during the following 60-min an upward inflection in T_{eso} signalled a transition from compensable to uncompensable heat stress with the critical vapour pressure (P_{crit}) at this point used to calculate ω_{max} (Figure 2). P_{crit} was verified by visual inspection, previously shown to be an acceptable method of assessing critical environmental limits [22, 23]. Warm fluid (37℃) was administered as a 4 ml/kg bolus after 15, 30 and 45-min of exercise, with fluid restricted during the second hour of exercise while oxygen consumption was being continuously measured.

Protocol – Heat Training Session (TRAIN)

On days 2, 4 and 6 participants entered the climate chamber (37℃, 60% RH) where they completed 10-min of seated rest on a cycle ergometer before 90-min of continuous cycling at a fixed heart rate (75% HR_{max}). Clamping heart rate was chosen to facilitate a progressive increase in workload throughout the 7-days given the noted response in resting

and exercising heart rate during heat acclimation [24]. Water at 37°C was administered in a 4 ml/kg bolus at 20, 40, 60, and 80-min.

Calculations

Heat balance parameters were estimated using partitional calorimetry [20]. The fundamental human heat balance equation states that

$$M - Wk = \pm C \pm K \pm R \pm H_{res} + E\ (W) \quad (1)$$

Where M is metabolic rate; Wk is external work rate; C, K and R are the rates of dry heat transfer via convection, conduction, and radiation; H_{res} is heat transfer via respiration; and E is the rate of evaporative heat transfer.

The rate of internal metabolic heat production (H_{prod}) was calculated by subtracting external work (Wk) from metabolic energy expenditure (M).

$$H_{prod} = M - Wk\ (W) \quad (2)$$

Wk was measured as the external workload in watts on the cycle ergometer while M was estimated with the following equation,

$$M = \dot{V}O_2 \cdot \frac{\left\{\left[\left(\frac{RER - 0.7}{0.3}\right) \cdot 21.13\right] + \left[\left(\frac{1.0 - RER}{0.3}\right) \cdot 19.62\right]\right\}}{60} \cdot 1000\ (W) \quad (3)$$

where $\dot{V}O_2$ is the rate of oxygen consumption in litres per minute (L/min) and RER is respiratory exchange ratio (volume of carbon dioxide produced ($\dot{V}CO_2$) divided by the volume of oxygen consumed ($\dot{V}O_2$)).

Conductive heat transfer (K) was considered negligible given the minimal surface area of skin in contact with a solid surface.

Convective and radiative heat transfer from the skin were calculated together with

$$C + R = \frac{(T_{sk} - T_a)}{\left(R_{cl} + \frac{1}{h \cdot f_{cl}}\right)} \cdot A_D(W) \quad (4)$$

where T_{sk} is mean skin temperature, T_a is ambient temperature, R_{cl} is the dry heat transfer resistance of clothing, h is the combined heat transfer coefficient, f_{cl} is the area factor of clothing and A_D is body surface area.

R_{cl} was estimated from the amount of clothing worn [25] and f_{cl} was estimated by assuming a correlation between the increase in area factor and R_{cl} of the clothing worn [26].

The combined heat transfer coefficient (h) is calculated as the sum of the convective (h_c) and radiative (h_r) coefficients. For cycle ergometry [27], h_c was calculated as

$$h_c = 8.3 \cdot \left(0.07 + 0.0043 \cdot f_{ped} + v_{air}\right)^{0.86} (W \cdot m^{-2} \cdot K^{-1}) \quad (5)$$

where f_{ped} is the pedalling frequency (revolutions/min) and v_{air} is the air velocity (m/s).

h_r was calculated as

$$h_r = 4\varepsilon\sigma \frac{A_r}{A_D}\left(273.2 + \frac{t_{sk} + t_r}{2}\right)^3 (W \cdot m^{-2} \cdot K) \quad (6)$$

where ε is emissivity of the body surface (assumed to be 0.95) [28], σ is the Stefan-Boltzmann constant (5.67 x 10^{-8} $W \cdot m^{-2} \cdot K^{-4}$), and A_r/A_D is the fraction of body surface participating in radiative transfer and determined by posture (assumed to be 0.7 for sitting) [29].

Respiratory heat loss (H_{res}) is associated with the rate of metabolic energy expenditure [30], and is calculated as the sum of both evaporative (E_{res}) and convective (C_{res}) respiratory heat loss with the following equation:

$$H_{res} = 0.0173 \cdot \left(H_{prod}\right) \cdot (5.87 - P_a) + 0.0014 \cdot \left(H_{prod}\right) \cdot (34 - T_a) (W \cdot m^{-2}) \quad (7)$$

where T_a is inspired air temperature (°C); and P_a is the vapour pressure of inspired air (kPa).

The requirement for evaporative heat loss (E_{req}) can be calculated by rearranging the conceptual heat balance equation:

$$E_{req} = H_{prod} - (C + R + H_{res}) (W \cdot m^{-2}) \quad (8)$$

The maximum rate of evaporative heat loss for a given environment (E_{max}) can be calculated as follows:

$$E_{max} = \omega_{max} \cdot \frac{(P_{sk,s} - P_a)}{R_{e,cl} + \left(\frac{1}{h_e \cdot f_{cl}}\right)} \ (W \cdot m^{-2}) \quad (9)$$

where ω_{max} is the maximum skin wettedness [31]; $P_{sk,s}$ is the vapour pressure at the skin surface while saturated with sweat (kPa); P_a is the absolute ambient vapour pressure (kPa); $R_{e,cl}$ is the evaporative resistance of clothing (assumed to be 0.002 $kPa \cdot m^{-2} \cdot W^{-1}$) [25]; f_{cl} is the clothing area factor (considered negligible given minimal clothing worn); and h_e is the evaporative heat transfer coefficient ($W \cdot m^{-2} \cdot K^{-1}$). $P_{sk,s}$ can be calculated using Antoine's equation [32];

$$P_{sk,s} = \exp\left(18.956 - \frac{4030.18}{\frac{T_{sk} + 235}{10}}\right) (kPa) \quad (10)$$

P_a is derived by finding P_{crit}, identified during the RAMP trial. h_e is estimated with the following:

$$h_e = 16.5 \cdot h_c \ (W \cdot m^{-2} \cdot kPa^{-1}) \quad (11)$$

When an upwards inflection in T_{eso} is observed during a RAMP trial, the amount of evaporation required to maintain heat balance (E_{req}) is equal to the maximum evaporative capacity (E_{max}) for that environment. Consequently, by substituting E_{max} with E_{req} and rearranging equation 10, ω_{max} can be estimated with:

$$\omega_{max} = \frac{E_{req} \cdot \left(R_{e,cl} + \left(\frac{1}{h_e}\right)\right)}{P_{sk,s} - P_{crit}} \ (ND) \quad (12)$$

Statistical Analyses

All data are reported as mean and standard deviation (mean ± SD). A one-way repeated-measures analysis of variance was employed to determine the main effect of Day (4 levels: 1, 3, 5, 7) for T_{eso}, T_{rec} and HR at rest; ω_{max}, P_{crit}, T_{eso}, T_{rec}, T_{sk}, LSR_{back}, LSR_{arm}, $ASGD_{back}$ and

SGO_{back} at the identified critical environmental limit (P_{crit}); and HR, TS, RPE, and whole body sweat rate (WBSR) at the end of exercise. All outcome variables were first checked for normality and sphericity. Post hoc comparisons were performed using two-tailed paired-sample t-tests while maintaining a fixed probability (5%) of making a type 1 error with a Holm-Bonferroni correction. All analyses were performed using the statistical software package GraphPad (Version 9).

Results

Pre-exercise Resting measures

A main effect of day was observed for resting T_{rec} ($P = 0.02$; $f = 5.23$), T_{eso} ($P = 0.049$; $f = 3.66$) and HR ($P = 0.032$; $f = 4.41$) (Table 1). T_{eso} was lower on Day 5 ($P = 0.036$) and Day 7 ($P = 0.007$) compared to Day 1. T_{rec} was lower on Day 7 ($P = 0.010$) compared to Day 1. HR was lower on Day 7 compared to both Day 1 ($P = 0.005$) and Day 3 ($P = 0.022$).

Data at P_{crit} (T_{eso} inflection point during the RAMP trial)

Individual and mean ω_{max} values on Day 1, 3, 5, and 7 are presented in Figure 3. ω_{max} values were progressively and significantly greater from Day 1 (0.65 ± 0.08) to Day 3 (0.73 ± 0.09; $P<0.01$), to Day 5 (0.78 ± 0.10; $P=0.01$), to Day 7 (0.87 ± 0.06; $P=0.01$) of heat acclimation ($P < 0.01$; $f = 38.21$). Similarly, this increase was reflected in the vapour pressure that the inflection point occurred (P_{crit}), which was also greater from Day 1 (2.80 ± 0.40 kPa) to Day 3 (3.20 ± 0.30 kPa; $P < 0.01$), to Day 5 (3.45 ± 0.25 kPa; $P < 0.01$), to Day 7 (3.65 ± 0.15 kPa; $P = 0.05$).

Figure 4 shows the mean responses for activated sweat gland density and sweat gland output throughout heat acclimation. $ASGD_{back}$ increased from Day 1 (65 ± 13 glands·cm^{-2}) to Day 3 (72 ± 19 glands·cm^{-2}; $P = 0.03$), Day 5 (78 ± 16 glands·cm^{-2}; $P < 0.01$), and Day 7 (82 ± 18 glands·cm^{-2}; $P < 0.01$) of heat acclimation ($P < 0.01$; $f = 16.41$). No differences were observed in either T_{sk} ($P = 0.35$; $f = 1.08$) or SGO_{back} ($P = 0.22$; $f = 1.66$).

Data at End-Exercise

A main effect of day was observed for WBSR ($P < 0.01$; $f = 28.11$) with Day 5 (1.03 ± 0.22 L·h^{-1}) and Day 7 (1.07 ± 0.15 L/h) higher than Day 1 (0.91 ± 0.19 L/h; $P=0.01$, $P<0.01$). No differences were found for end-exercise TS ($P = 0.11$; $f = 2.41$) or RPE ($P = 0.07$; $f = 3.30$) (Table 1).

Discussion

The present study is the first to the best our knowledge to quantify dynamic changes in ω_{max} throughout a 7-day heat acclimation protocol. Changes in pre-exercise measures in body temperature and heart rate alongside increases in whole body sweating demonstrate our heat acclimation protocol was sufficient to induce partial physiological heat adaptation. Individuals had a significantly lower rectal temperature (0.15 ± 0.11℃), esophageal temperature (0.15 ± 0.15℃) and heart rate (5 ± 4 beats·min^{-1}) with an increased whole body sweat rate (0.17 ± 0.09 L·h^{-1}) by Day 7 of acclimation. Confirming our initial hypothesis, maximal skin wettedness increased with each subsequent RAMP trial (performed every other day) from an initial value of 0.65 ± 0.08 on Day 1 to 0.73 ± 0.09, 0.78 ± 0.10, and 0.87 ± 0.08 on Days 3, 5, and 7, respectively. The current data also support our previous work and secondary hypothesis that the observed improvement in ω_{max} was driven predominantly by an increase in the amount of active sweat glands on the skin, rather than the amount of sweat produced by each gland. The density of heat-activated sweat glands before acclimation of 65 glands·cm^{-2}, and increased to 72, 78 and 82 glands·cm^{-2} on Days 3, 5, and 7, respectively. Our other hypothesis of a plateau in ω_{max} by Day 7 of heat acclimation was not supported with an average increase of 0.09 ± 0.08 from Day 5 to Day 7.

The present data align with previous work [12] that untrained, unacclimated individuals can maximally saturate their skin surface with sweat ~10-20% less than is currently assumed by the ω_{max} employed (0.85) in international standards [5]. Given the work informing these values were conducted during supine rest and that the exercise modality for the current study was upright cycling, the difference in maximal coverage can be explained by a reduction in sweating efficiency during upright exercise. An individual that increases metabolic rate while upright has a greater potential to drip secreted sweat because gravity limits the opportunity for sweat to pool on the skin combined with excess movement of the

limbs [33]. By contrast, when lying down, sweat is much more likely to accumulate and pool on the skin surface, permitting a relatively greater coverage over the torso and back for a consistent heat stimulus, possibly artificially increasing ω_{max} in a given ambient environment [7]. The current data builds on existing work to suggest that a more representative ω_{max} value for an untrained unacclimated individual completing a task elevating metabolic rate in an upright position is closer to 0.65-0.75. This difference may currently lead in some cases to a substantial under-estimation of predicted heat stress risk for unacclimated individuals. A practical example of this difference can be seen using the current ISO 7933 standard to simulate representative working scenarios. An 8-hour shift for an "unacclimatized" (ω_{max} = 0.85) worker in road construction at a fixed relative humidity (35%), wearing standardized clothing (0.6 clo), and working at an average heat production of 150 $W \cdot m^{-2}$, estimates 41°C as the ambient temperature that would result in an increase in rectal temperature beyond a safe cut-off limit (38.0°C), and that limit would be reached 2 hours into the simulated shift. By contrast, changing ω_{max} from 0.85 to 0.65 in the same scenario lowers the acceptable temperature limit by 2°C to 39°C and the rectal temperature cut-off would occur 90 minutes into the simulated shift.

After 7 days of heat acclimation, ω_{max} was increased from 0.65 to 0.87, demonstrating that our heat stimulus protocol was not sufficient to elicit complete biophysical adaptation, i.e., ω_{max} = 0.95 to 1.00. These data confirm that the induction of ω_{max} throughout heat acclimation align with previous reports that propose most of the physiological adaptation to the heat can be achieved during the first week of repeated exposure and is approximately linear in nature [8]. The forcing function for adaptation during the current protocol was administered as a 90-min session at a fixed percentage of maximum heart rate between assessments of ω_{max} (HEAT-TRAIN) which, has been demonstrated as a viable method to provide a sufficient thermal impulse [9, 12], further evidenced by the reduction in resting and

end-exercise heart rate along with the increase in average power output and WBSR throughout the protocol. The significant increase in ω_{max} between Day 1 and Day 3 demonstrates that physiological improvements can be attained with as few as two consecutive heat exposures when combining heat and exercise for an unacclimated group. This response has been previously termed as a "physiological accommodation" that serves as a precursor to adaptation from repeated stress [34], however, this is the first study to date to quantify the net positive biophysical impact of consecutive acclimation exposures on elevating the boundary of compensability in a previously unacclimated group. Considering the 0.09 ± 0.08 improvement in ω_{max} from Day 5 to Day 7 and the final ω_{max} value of 0.87 ± 0.06 representing a "partially acclimated" individual, these data show that additional sessions are required to reach "complete adaptation" and that the time-course of ω_{max} between "partial" and "complete" acclimation remains unclear. A study design that implements the current approach to induce "complete acclimation" (i.e. 14 days) would provide additional value to this area.

Aerobic fitness has long been considered a predictor for individual ω_{max} [35]. In a recent analysis, an alternative notion was proposed that activity status, specifically aerobic training and not aerobic capacity is primarily responsible for individual variability in ω_{max} [36]. That is, while often linked together, a partial heat acclimation response (evidenced by an increase in ω_{max}) occurs due to the repeated thermal challenge of exercise rather than improvements in fitness (i.e., $\dot{V}O_{2max}$) *per se*. These data appear to support those findings with the cohort displaying similar measured aerobic capacities (44.6 ± 6.7 $ml \cdot kg^{-1} \cdot min^{-1}$) despite having large differences in individual activity levels measured as $METmin \cdot week^{-1}$. Of the eight participants completed to date, the two with the highest estimated ω_{max} on Day 1 (coloured circles grey and green, Figure 3) were the two highest self-reported activity levels in the group (3459 and 3024 $METmin \cdot week^{-1}$). By contrast, the lowest ω_{max} on Day 2

(coloured circle dark blue, Figure 3) had the lowest self-reported activity level in the group (951 METmin·week^{-1}). These preliminary findings support the notion that differences in training status (i.e., activity level) and not aerobic capacity may independently influence ω_{max}.

Significant differences were observed in local sweat rate on the forearm, however, no differences were observed in local sweat rate on the back. Without a complete sample size, we are unable to conclusively determine that LSR on the back is not different as a function of heat acclimation. While higher local sweat rates are fundamental physiological responses to heat acclimation [10], and local sweat rate on the back has been shown to mirror forearm and whole body responses after acclimation [12], there is evidence to support a relative redistribution of sweat between the torso/core and periphery [37, 38]. The proposed redistribution of sweat to the limbs results in a more uniform and thus improved distribution of skin wettedness, leading to a higher evaporative capacity. Collectively, these data support the notion that an increase in maximum evaporative heat loss capacity is facilitated by an increase in whole body sweat rate, mediated by an increase in the number of active sweat glands present on the skin, not the amount of sweat produced by each gland. Theoretically, an increase in density would permit a more complete coverage of the skin surface compared to an increase in output per gland that may lead to higher sweat losses without concurrent improvements in ω_{max}.

Perspectives

The present findings can potentially contribute to the improvement and sensitivity of existing guidelines for the prediction and management of heat stress risk in occupational groups. Firstly, the current data further indicates that the assignment of a ω_{max} value of 0.85 for an “unacclimated” individual likely overestimates their ability to reach thermal

compensability for a given heat stress. Consequently, making an appropriate decision on safe working limits in hot environments may be compromised. Secondly, these preliminary data provide increased sensitivity by populating intervening points between “unacclimated” and “acclimated” states that can be used for existing heat strain models. With the prevalence of heat exposure strategies to help mitigate against adverse events in the heat, these data would potentially allow a more nuanced assessment of the likely strain as occupational groups are progressively exposed to the heat.

Considerations

Owing to the ongoing COVID-19 pandemic, our laboratory was shut down for an extended period (9 months) during data collection for this study and consequently, this study is not yet adequately powered for the statistical analysis planned prior to study commencement. The current chapter therefore represents preliminary findings for this study and the conclusions may change once the intended number of participants have completed the study. Data collection is planned for completion in 2023.

An increased natural level of heat adaptation (acclimatization) would independently influence maximal evaporative capacity. Despite some trials being completed during warmer months, observed values for ω_{max} on Day 1 aligned with our previous work, indicating that values we estimated were reflective of a similar, unacclimated population group.

Conclusion

Significant increases in ω_{max} were observed as early as 3 days into a 7-day heat acclimation protocol. Progressive increases in ω_{max} – predominantly mediated by increases in the number sweat glands activated, and not the volume of sweat secreted by each gland – were observed throughout the 7 days of heat acclimation. To the best of our knowledge, these are the first data to report how maximum skin wettedness dynamically changes with progressive heat acclimation. These data may assist future predictive heat stress risk modelling for people progressively exposed to heat stress from an initial unacclimated state and provide new information about ω_{max} levels between the standard "unacclimated" and "acclimated" states in existing heat stress models.

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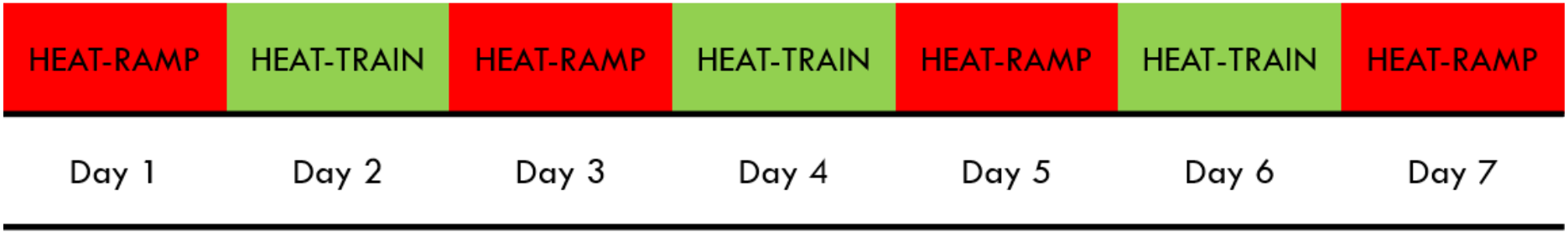

Figure 4.1 – *Illustration of the study timeline. HEAT-RAMP, humidity ramp protocol, continuous cycling at 275 W·m^{-2} for 120 minutes at 37°C; 60-min at a fixed vapour pressure of 2.05 kPa (35% RH) followed by 60-min with vapour pressure increased by 0.045 kPa·min^{-1}; HEAT-TRAIN, fixed HR exercise session, continuous cycling for 90 minutes at 75% age-predicted HR_{max} in a hot-humid environment (37°C, 60% RH).*

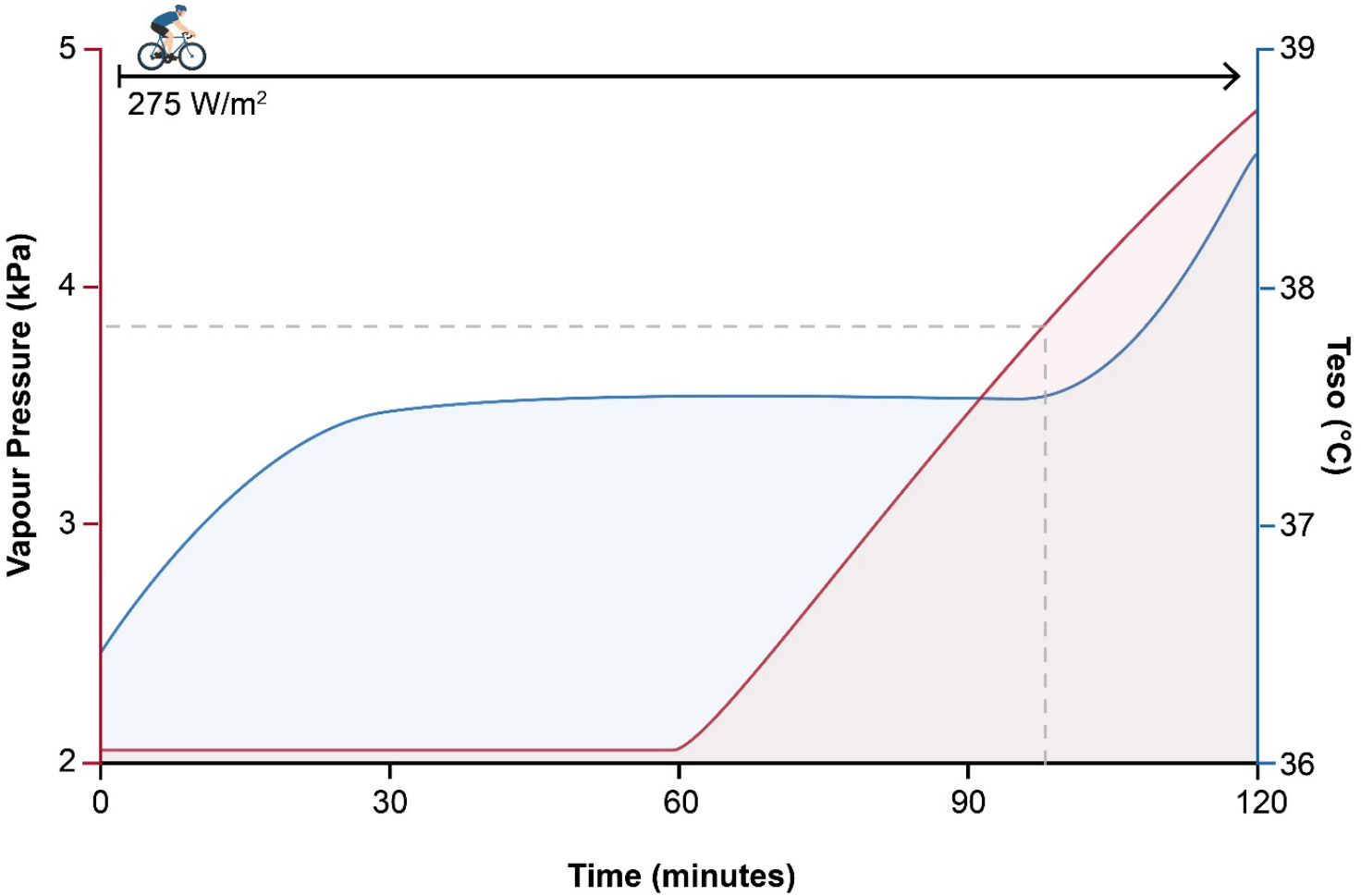

Figure 4.2 – *An illustration of the HEAT-RAMP protocol and how to identify critical environmental limits (P_{crit}). Vapour pressure (kPa) (Red) increases linearly from 60-120 minutes after achieving a steady-state T_{eso} (blue). When T_{eso} inflects, the corresponding vapour pressure (dotted line) can be used to estimate ω_{max}.*

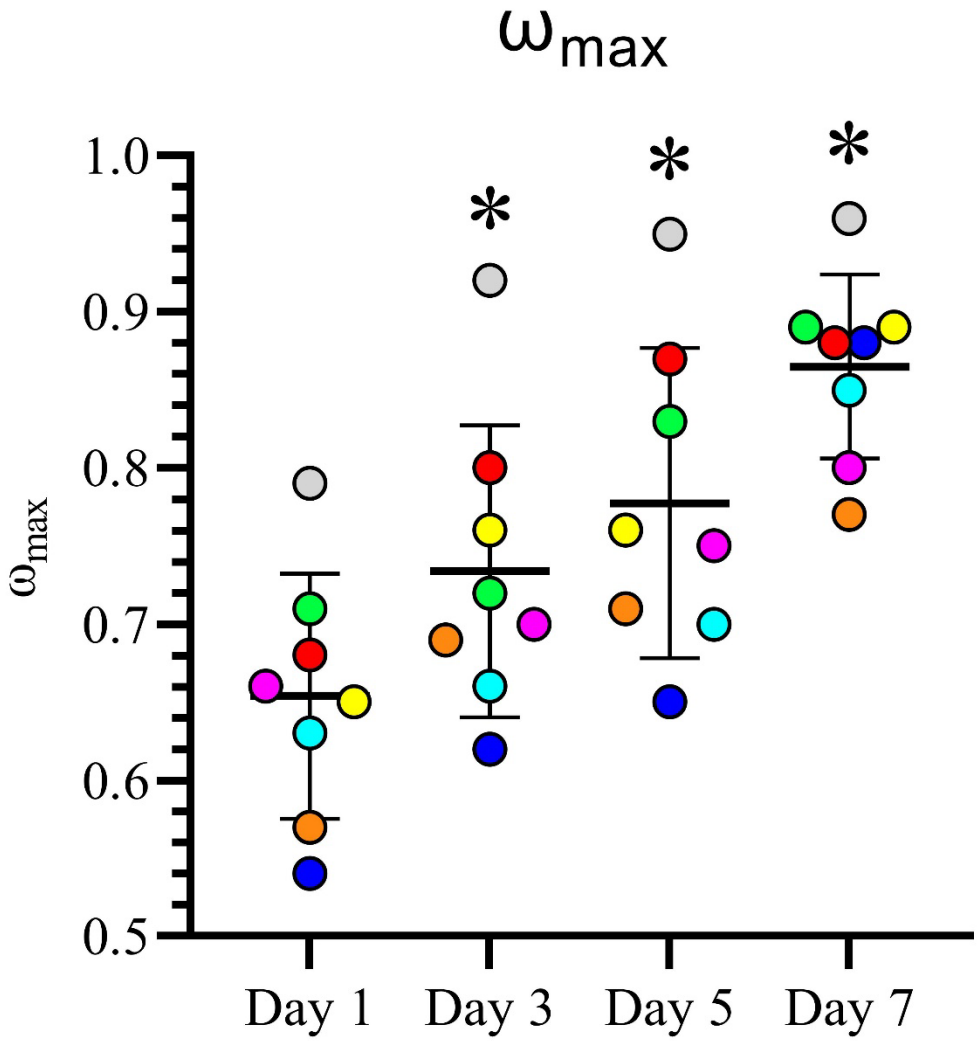

Figure 4.3 – *Individual and mean values of ω_{max} on days 1, 3, 5, and 7 of heat acclimation. Each colour represents an individual in the total dataset.* * *Significantly greater than the previous day ($P < 0.05$).*

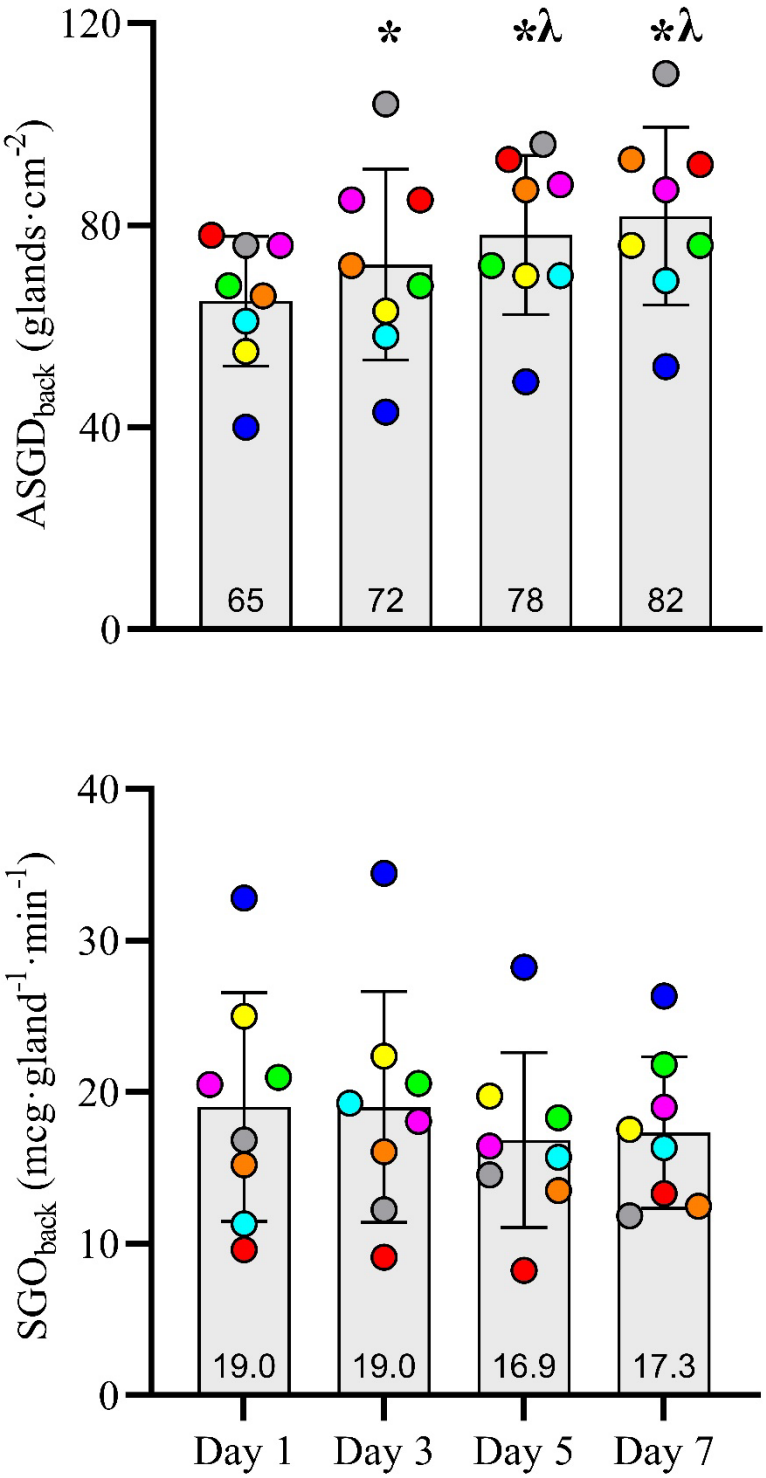

Figure 4.4 –*ASGD and SGO* individual and mean values for days 1, 3, 5, and 7 of heat acclimation. ***** *Significantly different to Day 1 ($P < 0.05$).* **λ** *Significantly different to Day 3 ($P < 0.05$).*

	Day 1	Day 3	Day 5	Day 7
Resting				
T_{rec} (°C)	37.04 ± 0.29	36.98 ± 0.25	36.92 ± 0.25	36.90 ± 0.27*
T_{eso} (°C)	36.89 ± 0.34	36.84 ± 0.25	36.78 ± 0.26*	36.74 ± 0.30*
HR (bpm)	74 ± 13	73 ± 13	71 ± 10	69 ± 12*$^{\lambda}$
Inflection Point				
ω_{max}	0.65 ± 0.08	0.73 ± 0.09*	0.78 ± 0.10*$^{\lambda}$	0.87 ± 0.06*$^{\lambda\beta}$
P_{crit} (kPa)	2.80 ± 0.40	3.20 ± 0.30*	3.45 ± 0.25*$^{\lambda}$	3.65 ± 0.15*$^{\lambda\beta}$
T_{sk} (°C)	36.23 ± 0.45	36.22 ± 0.32	36.15 ± 0.30	36.08 ± 0.26
LSR_{arm}	0.98 ± 0.20	1.18 ± 0.17*	1.20 ± 0.24*	1.31 ± 0.29*$^{\lambda\beta}$
LSR_{back}	1.18 ± 0.32	1.27 ± 0.25	1.25 ± 0.23	1.35 ± 0.20
ASGD (glands·cm^{-2})	65 ± 13	72 ± 19*	78 ± 16*$^{\lambda}$	82 ± 18*$^{\lambda}$
SGO (mcg·gland^{-1}·min^{-1})	19.0 ± 7.6	19.0 + 7.6	16.9 ± 5.8	17.3 ± 5.0
End-Exercise				
WBSR (L/h)	0.91 ± 0.19	0.98 ± 0.26	1.03 ± 0.22*	1.07 ± 0.15*
HR	169 ± 20	164 ± 16	159 ± 16	160 ± 18*$^{\lambda}$
TS	184 ± 43	176 ± 41	178 ± 35	157 ± 32
RPE	15 ± 5	15 ± 3	14 ± 4	13 ± 3

Table 4.1 – *Resting, inflection point and end-exercise variables representative of adaptation on Days 1, 3, 5, and 7 of heat acclimation.* ***** *Significantly different to Day 1 ($P < 0.05$).* $\boldsymbol{\lambda}$ *Significantly different to Day 3 ($P < 0.05$).* $\boldsymbol{\beta}$ *Significantly different to Day 5 ($P < 0.05$).*

CHAPTER 5: EXPERIMENTAL STUDY THREE

Developing a novel, non-invasive method to track changes in physiological heat acclimation status in an at-home setting

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Abstract

Purpose: To preliminarily assess a novel, non-invasive method to track changes in physiological heat acclimation status in an at-home setting.

Methods: Eight adults (5M, 3F) completed 6 consecutive days of heat acclimation (HA) consisting of, on days 1, 3 and 5, cycling for 120 minutes at a fixed heat production of 275 $W \cdot m^{-2}$ at 37˚C (60-min at 35% RH followed by 60-min increasing by 1.5% RH every 2 minutes); and on days 2, 4 and 6, cycling for 90-min at 75% age-predicted HR_{max} at 37˚C, 60% RH. Prior to the 1st (BL), and the morning of the 2nd, 4th, and 6th day of HA each participant completed a simple 45-min hot-water immersion test (HWT). The HWT consisted of lower leg submersion in 42˚C water with concurrent measurements of sublingual temperature (T_{sub}), heart rate (HR), forehead sweating onset time using a starch-iodine technique (OS), cumulative sweat loss on the forehead with an absorbent patch (SL) and thermal comfort (TC). True acclimation status was also verified via changes in rectal temperature (T_{rec}), HR, and whole-body sweat loss (WBSL) throughout the 6-day exercise HA protocol.

Results: After 6 days of HA, a true physiological heat adaptation was confirmed via 0.15 ± 0.11˚C lower pre-exercise T_{rec} ($P = 0.01$), 5 ± 4 $beats \cdot min^{-1}$ lower HR ($P < 0.01$), and 0.17 ± 0.09 $L \cdot h^{-1}$ greater WBSL ($P < 0.01$), relative to Day 1. Of the measures assessed during the HWT, OS was sensitive to changes in actual HA status. Compared to BL (23 ± 9 min), OS was shorter on Day 2 (17 ± 6 min; $P = 0.02$), Day 4 (16 ± 8 min; $P < 0.01$), and Day 6 (16 ± 8 min; $P < 0.01$). ΔTC was also sensitive to changes in actual HA status. Compared to BL (58 ± 15%), ΔTC was lower on Day 6 (29 ± 28%; $P = 0.02$). Day 6 was also lower than Day 2 (58 ± 30%; $P = 0.02$) and Day 4 (46 ± 31%; $P = 0.03$). With the sample size available,

no differences were observed for ΔT_{sub} ($P = 0.08$), ΔHR ($P = 0.40$), ΔTS ($P = 0.11$) or SL ($P = 0.35$) as participants became progressive heat-acclimated.

Conclusion: Our preliminary data in a limited sample size indicate that measurements of sweating onset time and thermal comfort during a 45-min hot-water immersion test may serve as potential methods for detecting changes in true HA status in a non-laboratory setting.

Introduction

Exposure to high heat stress environments is associated with excess mortality in vulnerable populations [1] and increased rates of exertional heat stroke during vigorous exercise in athletes, military personnel or occupational workers [2]. Most cases of heat-related illness are largely preventable with improved preparedness for upcoming instances of hot weather. A physiological heat adaptation through both natural (acclimatization) and artificial (acclimation) means is an effective strategy to help protect against negative health outcomes during extreme heat exposure [3]. Adapting to the heat requires repeated exposures that challenge the thermoregulatory system to improve heat dissipation pathways and heat tolerance, prolonging heat resilience [4]. Typically, the induction of heat acclimation is defined by a greater maximum sweat rate, a lower absolute core temperature and heart rate both at rest and throughout a given heat exposure, and a cooler thermal sensation [5]. A large proportion of the magnitude of these adaptations develop within 4-7 days, but is dependent on the intensity of the heat strain induced by the exposure [6]. Although there is ample work using standardised heat stress tests to assess changes in adaptation status [7-9], they typically involve expensive equipment and invasive measures in a laboratory environment. Tests that are easily employable for assessing acclimation/acclimatization status in an at-home setting would serve as a valuable tool to track heat adaptation in people in the community and potentially predict individual vulnerability to extreme heat at scale.

One challenge when designing a test sensitive to changes in heat acclimation/acclimatization status in an at-home setting is the induction of sufficient heat stress to assess markers of a physiological heat adaptation. Typically, heat stress is induced using exercise in a hot environment generated in a climate chamber [10], and heat adaptation status characterised by using relative changes in key measures from an established baseline

[8, 11]. Singular tests that evaluate heat acclimation are often termed “heat tolerance tests”, however, these tests were designed to provide a binary assessment on the suitability of an individual to return to work or competition following exertional heat stroke, not provide insight into their level of adaptation [12]. Currently, all of these practices require maintaining high levels of heat production and need individuals to come into a facility with highly specialised equipment (e.g. climate chamber) that is inaccessible to most members of the public. Tests that utilise a passive heat exposure e.g., use of saunas and hot water immersion may offer a more practical option for tracking potential changes in heat acclimation status in an at-home setting than exercise focused protocols [13, 14]. Of the two, hot water immersion is advantageous because of ubiquitous nature of water in most settings and that the conductivity of water is approximately 24 times that of air, meaning a lower water temperature can be used to elicit similar increases in body temperature compared to a sauna [15] to assess heat acclimation status. However, the associated strain that accompanies such a rapid increase in body temperature (~1.0℃ over 15-min in 41℃ water immersed to the shoulders [16]) may result in unnecessary risk when a more modest increase would allow adequate comparisons in acclimation status.

Another challenge in developing a method to quantify acquired physiological heat adaptation status is the suitability of the measures used. As examples, to measure body temperature accurately usually requires thermistors inserted in either the rectum or esophagus, both representing invasive measurement techniques. Ingestible telemetric pills are used frequently but are financially prohibitive. Sublingual temperature has been reported as a valid surrogate of deep-body temperature [17] but requires minimal influence from the surrounding environment (i.e. nasal only breathing) and an extended period to ensure a stable baseline temperature [18]. Measuring cardiovascular adaptations requires an accurate form of telemetry that can be monitored continuously, while local or regional measures of sweating

typically involve body sweat mapping [19] or ventilated capsules [20]. Identifying an onset threshold for sweating requires a highly responsive measure of core body temperature (e.g. esophageal temperature) and the continuous measurement of the amount of moisture present on the skin (e.g. ventilated sweat capsule).

The purpose of this study was to develop a novel, non-invasive method to assess potential changes in physiological heat acclimation status that can be ultimately used in large community-based studies assessing the time-course of heat acclimatization at the population level – as such, this test must be applicable to be utilised with a relatively low amount of resources in people's homes. To this end, we measured a variety of simple physiological and perceptual responses using a simple 45-min lower leg hot-water immersion test (HWT) in people who were becoming truly heat acclimated throughout the course of a progressive 7-day heat acclimation protocol in a climate chamber. We hypothesised that as true heat acclimation status evolved from daily exercise-heat stress exposure to a hot-humid environment in a climate chamber, measurable change in markers such as T_{sub}, HR, TS, TC, sweating rate (SL) and sweating onset time (OS) would be identifiable during a short pre-exposure lower leg hot-water immersion test.

Methods

Owing to the ongoing COVID-19 pandemic, data collection for this study is currently incomplete and consequently, we are not yet adequately powered for the statistical analysis planned prior to study commencement. The current chapter therefore represents preliminary findings for this study and the conclusions may change once the intended number of participants have completed the study. Data collection is planned for completion in 2023.

Participants

The University of Sydney Human Research Ethics Committee (HREC#:2021/233) approved this project and written informed consent was obtained from all participants before commencing. Eight healthy participants (Male: 5, Female: 3) completed the study (age: 27 ± 5 years; height: 176.6 ± 7.8 cm; weight: 71.9 ± 8.0 kg; body surface area (BSA): 1.88 ± 0.14 m^2). Based on previously reported differences in average change in resting heart rate data resulting from heat acclimation [10], the number of participants (n = 16) was determined with a power calculation (G*Power 3 software) assuming an α of 0.05, a β of 0.05 and an effect size of 1.0.

Experimental Design

Participants completed a hot water immersion test (HWT) as a baseline measure of heat acclimation status before (BL) completing six consecutive days of heat acclimation (HA), involving either 120-min of continuous cycling at a fixed metabolic heat production (Days 1, 3 and 5) or 90-min of continuous cycling at a fixed percentage of age-predicted maximum heart rate (75% HR_{max}). In addition to the BL measure, a HWT was also completed before exercise-heat exposure on Days 2, 4 and 6 to attempt to detect any changes in heat acclimation status (Figure 1). All trials within participant started at the same time of day (± 1 h) to minimize the influence of changes in body temperature during natural circadian

rhythm [21], with a 30-min washout period after completing the HWT before starting the heat acclimation protocol on days 2, 4, and 6. Participants refrained from alcohol, caffeine, and physical exercise for 24 h prior and during the 7-day protocol and consumed a standardized breakfast each day. Before each session participants would provide a mid-stream, first pass urine sample to confirm euhydration (specific gravity <1.020; if >1.020, 300 ml of tepid water was consumed).

A novel heat stress test to detect changes in heat acclimation status

Hot Water Immersion Test (HWT)

During BL and on days 2, 4, and 6, participants completed a 45-min lower leg hot water (42°C) immersion test (HWT), designed to try and detect changes in physiological markers of heat acclimation status that were elicited by an on-going lab-based exercise heat acclimation protocol (as described in Chapter 3). Upon arrival, participants were given standardized clothing to wear (Long Sleeve shirt, HEATTECH, 39% Polyester, 32% Acrylic, 21% Viscose, 8% Elastane, Uniqlo, Japan; Long Johns, HEATTECH, 38% Rayon, 31% Acrylic, 24% Polyester, 7% Spandex, Uniqlo, Japan). Once changed, participants remain seated in a standard room (22 ± 1˚C) for 15 minutes to allow a suitable time for T_{sub} to equilibrate. During this time, baseline measures were also taken. Approximately 2-min prior to the start of the hot-water exposure (see below) additional insulative layers were added in the form of a wearable blanket with the hood down (Huggle, 100% Polyester, USA) and a towel (100% cotton) draped over the upper legs. After 15-min baseline, participants immersed their feet and lower legs (~5 cm below the patella) into the bucket (Storage tub with rope handles, 65 L, Bunnings, Australia) filled with warm water. The bucket was filled with water during the first 5-min of baseline with 3 parts from the available hot tap and 2 parts from the available cold tap (10 L each part) (Round basin, 16 L, Bunnings, Australia). Hot water was added (1 L) every 5-min during the exposure to keep the temperature of the

water consistent (42 ± 1℃), with participants asked to move their feet in a circular motion for 10 seconds after each pour to ensure a consistent mixing of the water.

Measures

Water temperature (T_{water}) was recorded every 10 seconds using a general-purpose thermistor probe (400TM; Covidien, Mansfield, MA, USA) connected to a monitoring device (Data Logging Thermistor Meter, Digi-Sense, Cole-Palmer, Illinois, USA). Body temperature was measured with sublingual temperature (T_{sub}) using another general-purpose thermistor probe placed under the tongue in the posterior sublingual pocket, with participants instructed to keep their mouths closed during the baseline and exposure period. The probe was connected to a second identical monitoring device. Cardiovascular strain was measured with heart rate (HR) via telemetry (Polar Ignite, Kempele, Finland). Cumulative sweat loss was measured on the forehead (SL) with a 16 cm^2 technical absorbent patch (#2724 laminated Airlaid; Technical Absorbents, Northeast Lincolnshire, UK). Each patch had a 1.0-cm-wide border that was separated from the main patch to prevent sweat migration into the test area from adjacent areas [22]. Prior to experimental sessions, each patch was weighed without the border in a small plastic bag (Ziploc) to the nearest 0.1 mg using a precision scale (PS 510.R1, RADWAG, Poland). The patch and borders were both removed from their respective bags 90 seconds before application using gloves to prevent water and oils from hands being absorbed into the patch. The patch was held in place on the skin with a compression headband (Dri-Fit Headband, 92% Polyester, 8% Spandex, Nike, USA) to keep even pressure across the patch. The patch was left on for the entire duration of the exposure (45-min). After the exposure, the border was discarded, and the patch was reweighed in its original plastic bag. SL values were calculated as the difference between pre- and post-application mass in grams. The time that the onset of sweating occurred (OS) was measured with a starch-iodine technique (minor test), that has previously been used to assess sweating with a visual

intensity scale [23]. During the first minute after lower leg immersion, the left lateral neck was swabbed with tincture iodine (Iodine Tincture, Gold Cross, Australia), then dusted with corn starch (Essentials Cornflour, Coles, Australia). The site was inspected every minute for a change in colour from white to blue/purple. Once any change in colour was observed, the time (to the nearest minute) was recorded. Thermal sensation (TS) was rated with a modified visual analogue scale (0-240 mm) with anchor points ranging from "slightly cool" to "extremely hot". Thermal comfort (TC) was rated with a modified visual analogue scale (0-120 mm) ranging from "Not Uncomfortable" to "Very Uncomfortable". Both measures were taken every 5-min during the exposure and are reported as percentage change relative to maximum.

Heat Acclimation Sessions (HA)

As reported in Chapter 3, HA was induced throughout the course of 6 days using an exercise/climate chamber protocol, with participants completing either 90-min of continuous cycling at a fixed heart rate (75% HR_{max}) in a hot-humid environment (37℃, 60% RH) where body temperature fluid (37℃) was administered in a 4 $ml \cdot kg^{-1}$ bolus at 20, 40, 60, and 80-min (days 2, 4 and 6), or continuous cycling at a fixed metabolic heat production (H_{prod}) of 275 $W \cdot m^{-2}$ for 120 minutes at 37℃: 60-min at a fixed vapour pressure of 2.05 kPa (35% RH) followed by 60-min with vapour pressure increased by 0.045 $kPa \cdot min^{-1}$ where warm fluid (37℃) was administered as a 4 $ml \cdot kg^{-1}$ bolus at 15, 30 and 45-min (days 1, 3 and 5). Standardized clothing was worn for both sessions (shorts, socks, and shoes (males) or sports bra, shorts, socks, and shoes (females)).

To verify that each participant was heat acclimated, throughout the HA period, changes in rectal temperature (T_{rec}) were measured using a general-purpose thermistor probe (400TM; Covidien, Mansfield, MA, USA) inserted 15 cm past the anal sphincter into the rectum. Changes in heart rate were measured by telemetry with a watch and accompanying

strap (Polar Ignite, Kempele, Finland), and whole body sweat loss (WBSL) was estimated from pre- and post-trial body mass measurements (±2 g) using a platform scale (Mettler, Germany), corrected for weight gain through fluid ingestion [24].

Data Analysis

All data are expressed as means ± SD unless otherwise stated. A one-way repeated measures ANOVA with the repeated factors of "Day" (four levels: BL, 2, 4, 6) was used to assess start-HWT T_{sub} and HR. The same one-way ANOVA was used to analyze ΔT_{sub}, ΔHR, SL, OS, ΔTS, and ΔTC. All outcome variables were first checked for normality and sphericity. Post-hoc comparisons were performed using two-tailed paired-sample t-tests while maintaining a fixed probability (5%) of making a type 1 error with a Holm-Bonferroni correction. All analyses were performed using the statistical software package GraphPad (Version 9).

Results

True Acclimation Status

Each HWT was performed in parallel to an existing HA protocol. The purpose of the HWT was to attempt to track changes in true heat acclimation status induced with a combination of a progressive humidity ramp protocol and a top-up exercise-heat stress protocol on alternate days. As reported in Chapter 4, after 6 days of HA, a true physiological heat adaptation could be confirmed with a 0.21 ± 0.06AU higher ωmax ($P < 0.01$) increasing from 0.65 ± 0.08 (Day 1) to 0.87 ± 0.06 (Day 7). A 0.15 ± 0.11˚C lower pre-exercise T_{rec} ($P = 0.01$), a 5 ± 4 beats·min^{-1} lower pre-exercise HR ($P < 0.01$), and a 0.17 ± 0.09 L·h^{-1} greater WBSL ($P < 0.01$), relative to Day 1.

The novel HWT for tracking changes in HA status

Figure 2 shows the change in sublingual temperature (ΔT_{sub}) during each progressive test throughout acclimation. No main effect was observed ($P = 0.08$; $f = 3.30$) with values at BL (0.53 ± 0.17℃), Day 2 (0.42 ± 0.14℃), Day 4 (0.42 ± 0.23℃), and Day 6 (0.55 ± 0.19℃) not significantly different with the sample size acquired. Start-HWT T_{sub} at BL (36.56 ± 0.28℃), and after Day 2 (36.60 ± 0.26℃), Day 4 (36.62 ± 0.26℃) and Day 6 (36.47 ± 0.19℃) of HA were also not different ($P = 0.12$; $f = 2.51$).

Change in heart rate (ΔHR) during the HWT were not different ($P = 0.08$; $f = 2.92$) as true acclimation status changed, with reported values at BL (18 ± 12 beats·min^{-1}), Day 2 (12 ± 4 beats·min^{-1}), Day 4 (14 ± 8 beats·min^{-1}), and Day 6 (18 ± 10 beats·min^{-1}) similar (Figure 2). Similarly, no differences were observed ($P = 0.42$; $f = 0.94$) for start-HWT HR during BL (62 ± 13 beats·min^{-1}), Day 2 (66 ± 13 beats·min^{-1}), Day 4 (65 ± 11 beats·min^{-1}), and Day 6 (63 ± 9 beats·min^{-1}).

Figure 3 shows cumulated sweat values from the forehead (SL), and the onset time (in minutes) that sweating was observed on the left lateral neck (OS) during the HWT. SL values at BL (0.23 ± 0.14 g), Day 2 (0.23 ± 0.12 g), Day 4 (0.23 ± 0.13 g) or Day 6 (0.28 ± 0.16 g) were not different ($P = 0.35$; $f = 1.13$). However, a main effect of day was observed for OS ($P < 0.01$; $f = 9.25$), with OS times significantly shorter on Day 2 (17 ± 6 minutes; $P = 0.02$), Day 4 (16 ± 8 minutes; $P < 0.01$) and Day 6 (16 ± 8 minutes; $P < 0.01$) compared to BL (23 ± 9 minutes).

Alterations in perception during the HWT as true heat acclimation progressed are shown in Figure 4. Changes in thermal comfort (ΔTC) during the HWT was influenced by acclimation status ($P = 0.03$; $f = 4.20$) with the percentage of maximum discomfort on Day 6 (29 ± 28%) significantly lower than Day 4 (46 ± 31%; $P = 0.03$), Day 2 (58 ± 30%; $P = 0.02$), and BL (58 ± 15%; $P = 0.02$). Change in thermal sensation (ΔTS) during the HWT were not different throughout ($P = 0.11$; $f = 2.69$).

Discussion

Our battery of non-invasive measures for observing potential changes in heat acclimation status designed to be an easily employable protocol that could be implemented in an at-home setting was, with a limited sample size, somewhat effective at tracking true heat acclimation status. While there were no changes observed in sublingual temperature, heart rate, accumulated sweat produced on the forehead or whole-body thermal sensation during the HWT – despite true changes in in HA status, both the time that sweating starts on the neck and the magnitude of thermal discomfort experienced throughout our simple 45-min lower leg hot water immersion test did show consistent changes. Results from the previous chapter (see Chapter 4 "*Quantifying the Time-course of Changes in Maximal Skin Wettedness with 7 days of Heat Acclimation*") confirmed that the group did experience a true effect of heat acclimation, evidenced by a lower pre-exercise T_{rec} ($0.15 \pm 0.11^{\circ}C$), lower pre-exercise HR (5 ± 4 beats$\cdot$min^{-1}), and a greater WBSR (0.17 ± 0.09 L$\cdot$h^{-1}), relative to Day 1 during the same time period. Collectively, given that we know the group did acclimate throughout the six days, these results suggest that either; i) some of the measures taken were not appropriate or sensitive to changes in adaptation status or ii) the heat stimulus generated by the HWT was not sufficient to elicit enough heat stress to enable changes in physiological heat adaptation variables to be observed between exposures.

Significant differences were observed in the time that the onset of sweating occurred, however, no differences were observed in cumulative sweat loss locally on the forehead. Given a primary response to heat acclimation is an increase in maximum sweat rate [8, 9, 25], primarily mediated by an increase in the amount of sweat glands recruited rather than the amount of sweat produced per gland [9], a significant difference in onset threshold without a concurrent increase in the amount of sweat accumulated suggests that the measurement

technique (technical absorbent patch) was not sensitive to detect the changes in heat acclimation status. This sensitivity could have been caused by the sweating rate during the test approaching the absorbent capacity of the patch, causing some sweat to be left on the skin, however, the maximum sweat observed during our entire data collection so far (600 mg over 45 minutes) equates to 1.2% of the absorbent capacity of the patch. The saturation point of the absorbent material was therefore not close to being approached during any of our measurements, meaning that the possibility of alterations in patch mass resulting from excess sweat being left on the skin seems highly unlikely. Additionally, differences in sweat rate at different locations on the body [19, 25] makes comparisons between measurement sites (neck vs forehead) another potential source of variability in the limited dataset. Previous work has also proposed that sample times of longer than 5 minutes are not recommended for absorbent patches because of the impact on local heat loss and skin temperature [19], however, given changes in skin temperature due to the application of a technical absorbent patch have been shown to be minimal [26], the variability for cumulative sweat loss seems to be related to site location. A reduction in the rise of perceptual strain as participants became acclimated was observed in terms of thermal comfort. This response has been previously shown in an athletic population [27], however, the mechanism underpinning the graded response remains unclear. Seminal work from the 1970's propose that to maintain whole-body thermal comfort, three conditions need to be met; the body is in heat balance, sweat rate is within comfort limits and mean skin temperature is within comfort limits [28]. Of the three conditions, a lower local and mean skin temperature as participants progressively acclimate could be a possible reason for an improved thermal comfort, however, neither were measured during the HWT in the current study. No other measures taken during each HWT successfully tracked changes in adaptation status.

The measures taken to detect changes in heat acclimation status were chosen as non-invasive methods that could be feasibly implemented in an at-home or field setting. Core temperature, measured with sublingual temperature, while not suitable for active protocols that require heavy breathing stemming from an increased work rate, in passive exposures with sufficient time to equilibrate and minimal influence from the external environment, is considered a valid and responsive measure of core temperature [29]. While the importance of nasal-only breathing was highlighted before and during each trial, the evaporative heat loss associated with any breathing during the extended period (60 minutes in total with the 15-min baseline followed by the 45-min test) introduces a potential source of variability in the current dataset. Heart rate measured via telemetry is a highly accurate method to demonstrate cardiovascular strain and is used extensively in both laboratory and field settings. As reductions in heart rate were observed in the same cohort (5 ± 4 beats·min^{-1}) during the HA protocol described in Chapter 4 after 7 days of acclimation, the variability in the HWT dataset seems to be a function of the combination of an additional day of acclimation and a limited sample size, particularly to detect the influence of acclimation on change in heart rate throughout the 45 minute test. Even though the forehead produces a relatively large amount of sweat compared to other locations on the body in male athletes during exercise [19], the effect of the HWT to drive a sweating response was limited (~0.2 g detected) meaning that the measure was likely not sensitive enough to detect the changes in sweat loss throughout acclimation. The iodine-starch test (i.e. minor test), is used clinically to evaluate hyperhidrosis by showing the location of hyperactive sweat glands [23]. In assessing hyperactive sweat glands, a sweating intensity visual scale was created that allows an accurate assessment of when sweating occurs at a specific site. Although not able to quantify the amount of sweat secreted, using "discolouration" as a marker for the onset of sweating in this context is an effective method to track heat acclimation status.

Typically, progressive heat acclimation is designed with a protocol that both induces and demonstrates the magnitude of adaptation in thermoregulatory, cardiovascular, and perceptual markers. The current HWT was an attempt to assess, not induce adaptation and therefore required a level of heat stress that was sufficient to identify the independent influence of acclimation day without eliciting further adaptive benefits. Accordingly, a contributing factor to the inability for some markers of acclimation to be detected in the current protocol are the relatively modest increases in core temperature (~0.5°C) during the HWT. Ultimately, the potency of the current heat stimulus may not have been sufficient to enable differences in thermal and cardiovascular strain to be observed, particularly with a limited sample size. Previous work from our laboratory has demonstrated the condition dependent nature of the acquisition of heat acclimation, comparing core temperature and sweating responses during exercise in both compensable and uncompensable heat stress [30]. Consequently, showing improvements in heat dissipation during acclimation are best observed when the upper physiological limits of compensability are challenged. Following on, the current protocol may not have been thermally stressful enough to challenge the participants limit of compensability, potentially explaining why differences in perception and onset thresholds for sweating were observed without concurrent adaptations to cardiovascular or body temperature measures. Collectively, these data are encouraging that with a complete dataset to adequately power the study design, the current protocol may be an effective method to assess heat acclimation status.

Considerations

Owing to the ongoing COVID-19 pandemic, data collection for this study is currently incomplete and consequently, this study is not yet adequately powered for the statistical analysis planned prior to study commencement. The current chapter therefore represents

preliminary findings for this study and the conclusions may change once the intended number of participants have completed the study. Data collection is planned for completion in 2023.

In the time between receiving ethical approval to complete this study (10th May 2021) and the submission of this thesis (24st December 2022) our ability to collect data has been disrupted by government mandated lockdowns (June 25th – October 11th 2021), The University of Sydney suspension of research owing to concerns around mitigating the spread of coronavirus upon return to the university (October 11th – January 10th 2021) and a major chamber malfunction causing an extended block without use (27th September – November 14th 2022).

Conclusion

Of the battery of measures tested, the time that sweating onset occurred and the level of thermal discomfort during a 45-min hot water immersion test were both effective at tracking true heat acclimation status during a 6-day heat acclimation protocol. These preliminary data suggest that the novel protocol designed to be a practical, non-invasive method to track acclimation status could be a viable assessment of an individual's level of adaptation to the heat.

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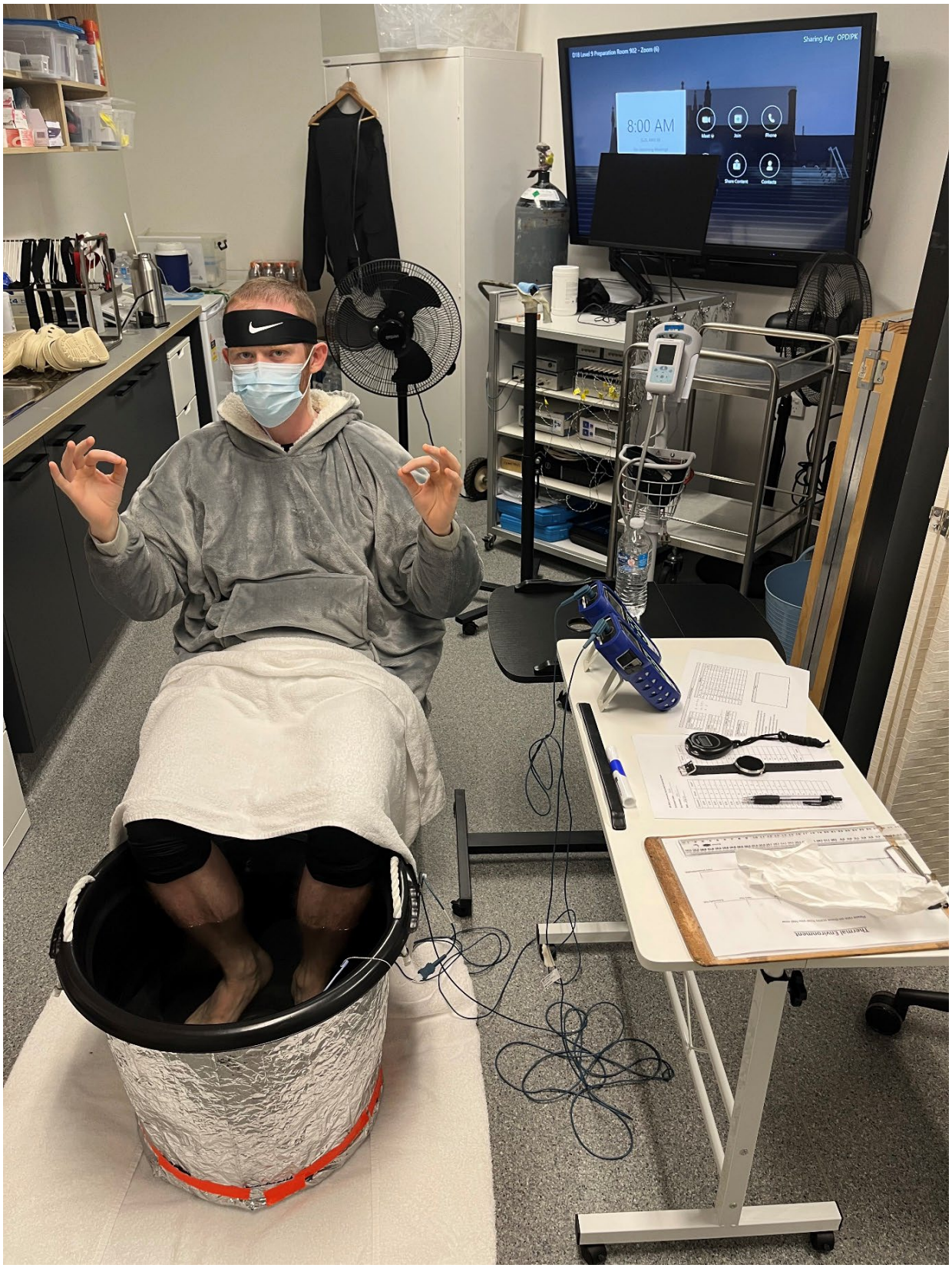

Figure 5.1 – *Picture of the setup for each hot water immersion test (HWT). Lower legs immersed in a bucket for 45 minutes while wearing thermals and a wearable blanket with a towel draped over the lap.*

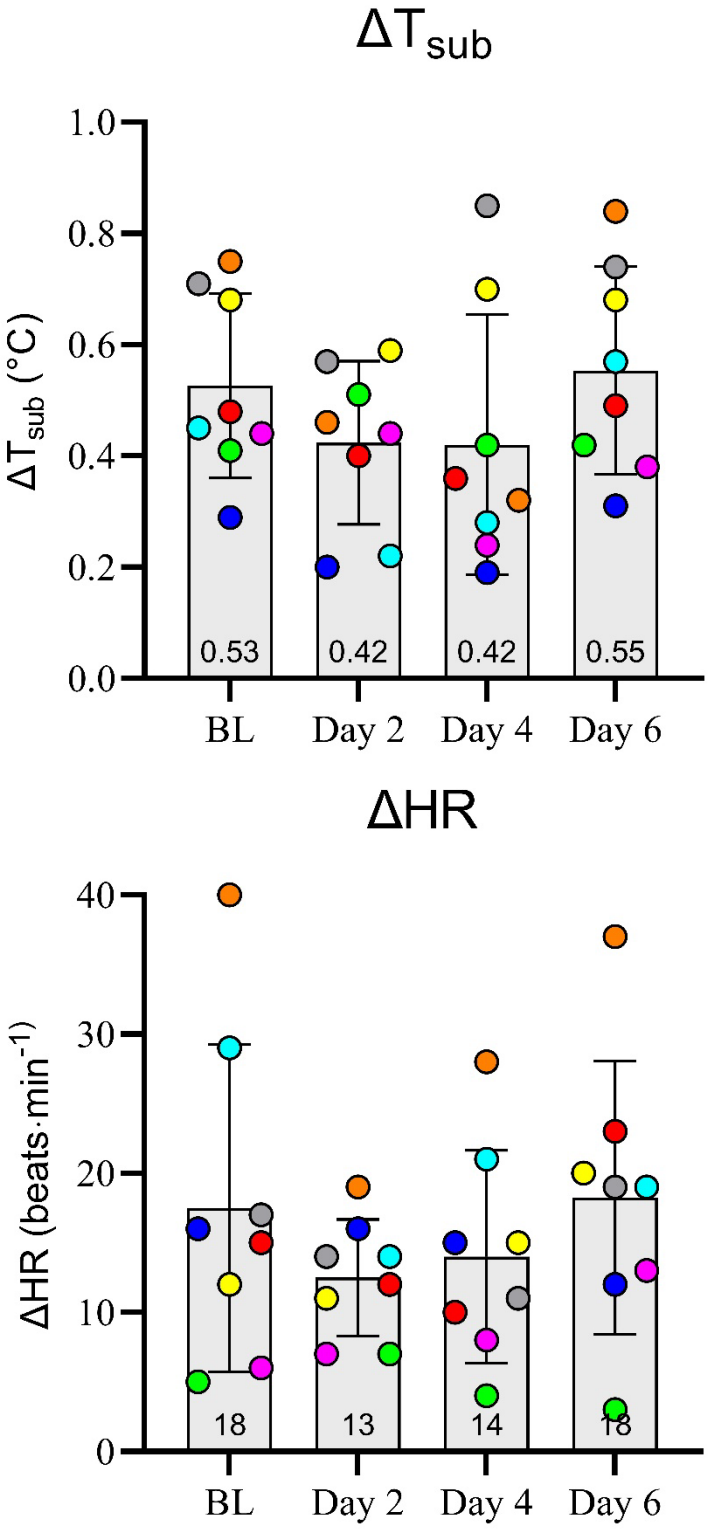

Figure 5.2 – *Mean and individual values for change in T_{sub} (A) and HR (B) during BL, day 2, day 4, and day 6 of heat acclimation.*

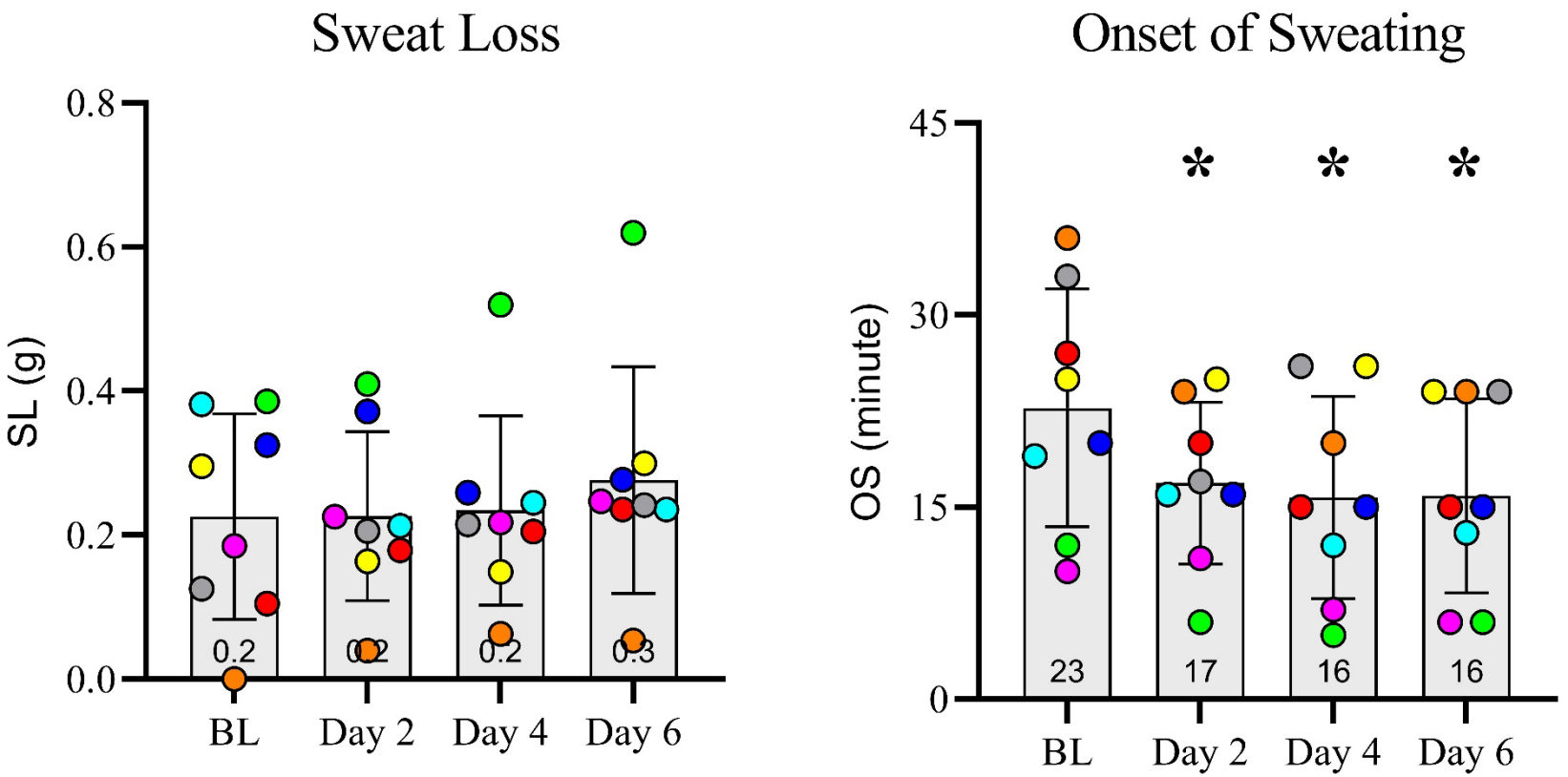

Figure 5.3 – *Individual and mean values for the cumulative sweat lost on the forehead during the exposure (SL) and the minute that colour change was noted on the left lateral neck in minutes (OS) throughout 7-days of heat acclimation. *Significantly different to BL (P < 0.05).*

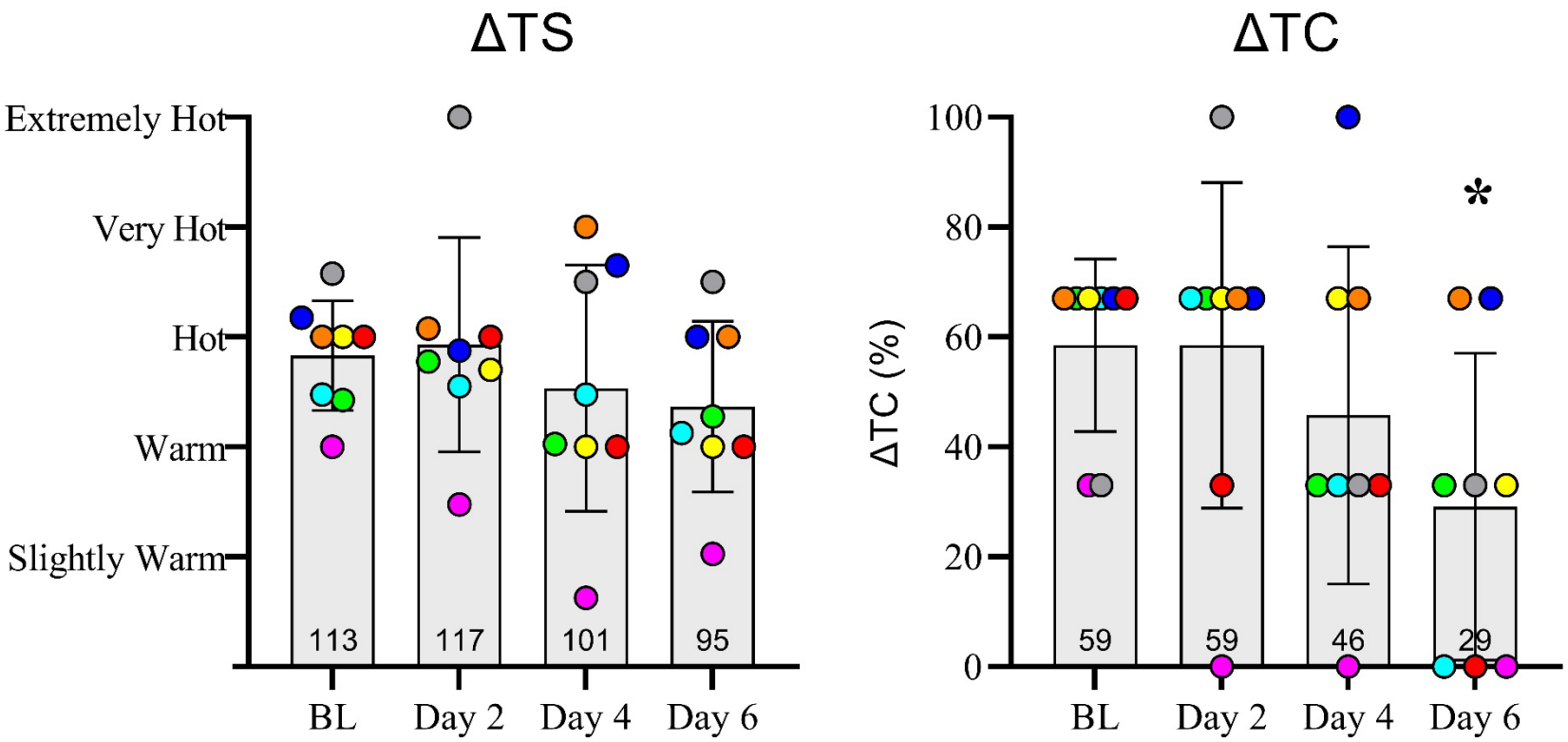

Figure 5.4 – *Mean and individual values for ΔTS and ΔTC during BL, day 2, day 4, and day 6 of heat acclimation. *Significantly different to all other days (P < 0.05).*

CHAPTER 6: GENERAL DISCUSSION AND CONCLUSIONS

Summary

To best combat the negative health outcomes associated with excessive heat stress exposure due to hot weather, developing heat-health management systems that can quantify inherent or developed heat adaptation is becoming increasingly important [1]. To our knowledge, there is limited research to show how the combination of ambient environment, physical activity and clothing worn modify the potential for natural heat acclimatization throughout a given summer, especially in an Australia context. Equally, the capacity to understand the factors that determine natural heat acclimatization among the general population is currently limited by the resource-intensive methods (i.e., climate chamber-based protocols) available to researchers wishing to measure acclimatization/acclimation status. Finally, quantifying the progression from an unacclimated to heat acclimated state through a key biophysical marker for predicting thermal strain – i.e., the maximum proportion of skin that can be saturated in sweat (ω_{max}), is poorly understood. Accordingly, the present thesis aimed to develop a broader understanding of the progression and magnitude of human physiological heat adaptation with the following aims:

1) Model the independent influence of different environmental and personal parameters on predicted acclimatization status throughout a representative year in Australia by estimating daily thermal strain and an associated "acclimatization score" for different occupational groups (Chapter 3)
2) Quantify the time course of changes in ω_{max} throughout 7 consecutive days of heat acclimation (Chapter 4).
3) Develop a novel, non-invasive method to assess changes in physiological heat acclimation status in an at-home setting (Chapter 5).

Key Findings

In chapter 3, our modelling approach considered two occupational roles (indoor office workers and outdoor agriculture worker) in different working situations. Our model estimated that due to the limited exposure of office workers to environmental heat stimuli, irrespective of location across Australia, the likelihood of attaining a protective benefit of heat acclimatization throughout a typical summer is minimal. In contrast, outdoor labourers that work under direct solar radiation were estimated to progressively acclimatise, depending on the prevailing climate (determined by geographical region), with the predicted extent of acclimatization being somewhat limited unless those same workers engaged in additional physical activity outside of working hours, augmented further by wearing protective clothing during working hours.

Chapter 4 showed that during 7 days of heat acclimation, the proportion of skin surface area that is saturated with sweat (i.e., maximum skin wettedness, ω_{max} [2]) increased linearly throughout without plateauing after 7 days of heat acclimation. Furthermore, aligning with previous work conducted in our laboratory and others, this experiment demonstrates that the progressive increases in ω_{max} were predominantly mediated by increases in the number, not individual output of active sweat glands.

In chapter 5, our preliminary data indicated that two of the six measures selected to track “true” acclimation status, i.e., sweating onset time and changes in thermal discomfort, were able to detect changes in day-to-day physiological heat adaptation during a simple 45-min hot-water immersion test (HWT).

Implications and Future Directions

Chapter 3 predicted physiological responses of occupational groups while systematically changing environmental parameters (i.e., different geographical locations, changes in radiant temperature with exposure to the sun) and personal factors (i.e., increased activity at or outside of work, more clothing) that ultimately determine thermal strain. The time course for physiological heat acclimation is well established [3-7], however, the evidence to support natural acclimatization directly is less developed [8-11], with the only study to our knowledge that demonstrates the independent influence of natural acclimatization in Australia is in a hot, dry climate (Perth, WA) [11]. From a physiological perspective, adapting to the heat via natural (acclimatization) or artificial (acclimation) exposures were considered analogous as both improve our heat tolerance by increasing our ability to dissipate heat from our body. Based on that notion, chapter 3 used the effects observed during an isothermic heat acclimation (i.e., increase in rectal temperature to 38.5°C for consecutive days) [3, 12, 13] to develop a working model to estimate day-to-day natural heat acclimatisation status throughout a calendar year.

The current data suggest that even if a person works outdoors for a living, wears extra protective clothing during each working day, and completes additional physical activity outside of working hours as per the World Health Organisation minimum physical activity guidelines [14], they will not reach a "complete acclimatization" status unless living in a tropical savanna environment (e.g., Darwin, Northern Territory). Further, for most locations in Australia, an individual's level of natural adaptation is estimated to be minimal unless they complete physical activity in the sun for extended periods both during and outside of working hours. These acclimatization estimations were modelled for an average Australian man working in the agriculture sector that is not necessarily representative of different societal

groups that may acclimatize at different rates. For example, from a thermoregulatory perspective, older adults are in general more susceptible to the ill-effects of heat exposure because of age-related decrements in sweating output and consequently heat loss via evaporation [15, 16]. Additionally, occupations in other large industries like construction and tourism have different activity profiles, job-specific demands and self-pacing strategies that will all influence the level of natural protection achievable [17]. Consequently, the current data should be developed to include various occupations and demographics that could serve as the foundation to develop location specific health advice relating to safe durations of "natural exposure" that will help acclimatize individuals of key at-risk groups when ambient conditions permit.

To mitigate the marked negative effect to human health caused by heat extremes and chronic hot weather, global, national, and regional organisations disseminate advice to help educate and protect the general public on safe practices [18, 19]. In Australia, public messaging relating to extreme heat and the associated health risks is distributed at the state level, so by providing evidence to support the timeline of acclimatization in different major cities, state governments can potentially create individualised advice based on their specific requirements. An example with the current data would be in Canberra, ACT, where knowing that an agricultural group is only "partially acclimatized" in November may warrant a more conservative approach in anticipation of a heatwave, with recommendations centred around taking more "planned breaks" or changing working hours [20] when compared to a similar heatwave in January where the group is likely to be "mostly acclimatized".

For chapter 4, one of the main findings was that our results support previous work [21] demonstrating that non-heat acclimated individuals can maximally saturate their skin surface with sweat ~10-20% less than the assumed maximum ω_{max} in current international standard (0.85) [22]. This finding builds on existing data to suggest that a more representative

ω_{max} value for untrained unacclimated individuals completing a task in an upright position that elevates metabolic rate is closer to 0.65-0.75 rather than 0.85. In addition, the main question answered in chapter 4 was how individuals progress from an unacclimated to heat acclimated state using ω_{max} to populate the intervening time points throughout a 7-day protocol. Our protocol was only able to "partially acclimate" the cohort showing that biophysically, 7 days of heat acclimation is insufficient to achieve a ω_{max} of ~1.00 [4].

A potential application for these findings is clearly presented in chapter 3 where we used the Predicted Heat Strain (PHS) model to estimate natural acclimatization in different cities across Australia. The difference between our observed results from chapter 4 (unacclimated ω_{max} ~0.7) and the default value used in the PHS model (unacclimated ω_{max} = 0.85) could result in a systematic underestimation of both estimated thermal strain and potential for adaptation. Equally, having additional values of ω_{max} to align with the progression from unacclimated to acclimated would permit a more nuanced assessment of the limit of compensability for a given environment. Ultimately, there were two reasons that the data collected in chapter 4 was not used for the modelling in chapter 3. Firstly, owing to interruptions from the COVID Pandemic outlined previously, the study presented in this thesis is presently underpowered based on our initial power calculations and definitive conclusions are difficult to make from the currently limited dataset. Indeed, for our current n of 8, the mean unacclimated ω_{max} value is 0.65, but these values range from 0.54 to 0.79. There is also a likely underlying influence of starting aerobic fitness, due to the partial heat acclimation induced by regular physical activity [23-26], and a potential alteration due to sex (see next paragraph below). Second, the PHS model is a well validated, widely accepted tool to analyze and interpret thermal strain during simulated heat stress [27-29] therefore the evidence needed for departing from the standard PHS procedures described in ISO 7933 far exceed those currently available from this thesis. A future direction for this work would be to

take the method executed in chapter 4 and extend the timeline to ensure complete adaptation (i.e. ω_{max} = 1.0). This design was the initial plan for chapter 4 but again, disruptions resulting from the COVID-19 Pandemic meant that the remaining time to complete the project required a shortening of the intervention, with a reduction in the number of days individuals could acclimate.

Another potential direction that was part of the initial design for the study described in Chapter 4 is sex-dependent comparisons in ω_{max} throughout heat acclimation. There is still debate as to whether the influence of reproductive hormones in women result in quantifiable differences between the sexes in their capacity to dissipate heat [30, 31]. Seminal work in the 1980's found that core temperature elevations during a fixed absolute exercise intensity (1.34 $m \cdot s^{-1}$ treadmill walking) was lower in women compared to men in a hot-humid climate, whereas the response was reversed in hot-dry environments [32], concluding that differences in surface area-to-mass ratio and a more efficient sweating suppression in humid conditions allowed women to tolerate humid conditions better than men, with a higher evaporative capacity in men permitting a more efficient response to dry heat [32]. Following on, when thermoregulatory responses were examined during exercise that was fixed for the amount of heat produced, expressed per unit of body surface area, men showed higher rates of evaporative heat loss compared to women, but only at higher intensities (300 $W \cdot m^{-2}$), suggesting that the maximum evaporative capacity of women may be lower than in men [33]. The proposed mechanism to explain this lower maximum capacity was a lower amount of sweat output per gland rather than a difference in the number of activated sweat glands [33].

A sub-analysis of the data presented in Chapter 4, that differentiates ω_{max} according to sex of our participants (5 males, 3 females) (Figure 1), potentially supports the notion that women could possibly have a lower maximum evaporative capacity, evidenced by an observable trend for a lower ω_{max} compared to their male counterparts. With the current

number of participants, the average increase in ω_{max} by the end of acclimation for males was 0.23 ± 0.07 compared to females 0.19 ± 0.04, with end acclimation ω_{max} of 0.90 ± 0.03 for males and 0.81 ± 0.04 for females. These data indicate that the theoretical limit for ω_{max} in both unacclimated and partially acclimated females may be lower compared to males. With a limited sample size an estimation of sex-dependent differences in ω_{max} is not currently possible, however, with a complete sample, we should be able to identify whether the upper limit for compensability is indeed different between women and men in both an unacclimated and partially acclimated state. If such a phenomenon does eventuate, another possible line of future enquiry may be to assess whether female participants may simply require a different heat acclimation protocol or stimulus in order to achieve complete biophysical heat acclimation (i.e., ω_{max} = 1.00).

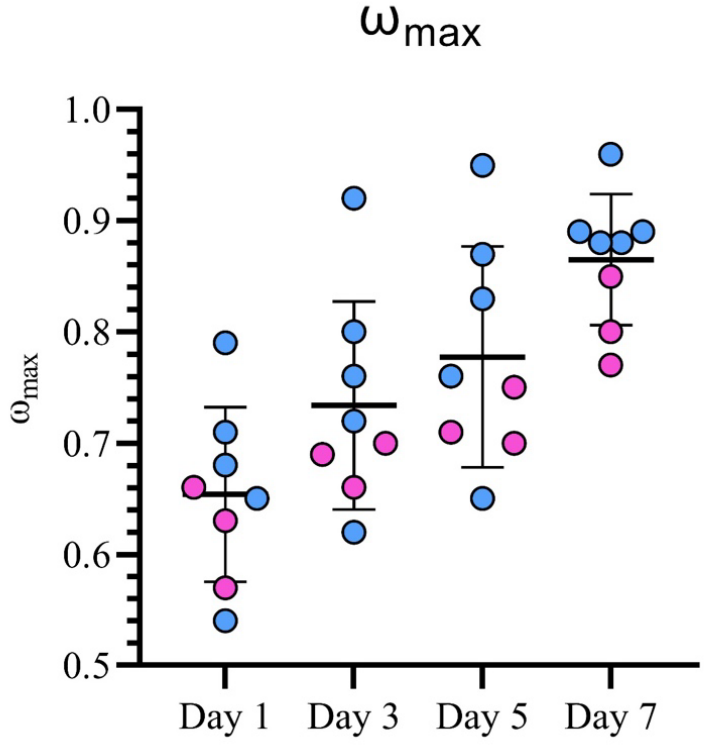

Figure 6.1 – *Individual and mean values of ω_{max} on days 1, 3, 5, and 7 of heat acclimation. Blue circles – Male participants, Pink circles – Female participants.*

Chapter 5 investigated the efficacy of a novel, non-invasive protocol involving a hot water immersion test (HWT) that could be administered in an at-home setting for assessing, not inducing, a physiological adaptation to the heat. This HWT was tested concurrently with the heat acclimation protocol outlined in Chapter 4 so that we could identify how effectively the test, and associated measures were able to track the "true" changes in physiological heat acclimation being observed. Even though by design, one potential contributing factor to the inability for some markers to be detected were the relatively modest increases in core temperature (~0.5°C) during the HWT, meaning that the heat stimulus may not have been sufficiently stressful to observe differences in thermal and cardiovascular strain with heat acclimation. Shown in Chapter 4 and in previous work from our laboratory and others [12, 13, 21, 34] the acquisition of heat acclimation appears to be inextricably linked to heat stress that challenges our upper physiological limits of compensability [21, 35]. At present, with a limited sample size it remains unclear if the HWT is thermally stressful enough to challenge thermal limits of compensability, however, the current data are encouraging with multiple measures demonstrating a difference throughout a short (6-day) acclimation protocol.

The broader implication for the overall method in chapter 5 is the potential to directly measure the physiological responses that were predicted in chapter 3. From a public health perspective, the importance of quantifying the level of physiological protection would help deliver appropriate, specific messaging and interventions to best mitigate the risk of heat-related illness during heatwaves [36, 37]. The results from chapter 3 highlight the potential for adaptation but to consolidate policy change would require that same information to be collected directly from different population groups. Despite having the ability to quantify individual level of adaptation to the heat with a singular value, outlined in chapter 4 and others [2, 21, 38], to implement a population level strategy requires a population level intervention. Issues around the cost and resources necessary to collect a large data set make

the current methods of assessing at-scale heat adaptation status not viable. Equally from a sporting perspective, having a simple test that can be taken to the individual/team and not compromise their training regime would allow a consistent tracking of adaptation to help quantify the effectiveness of specific programming or when selecting environments to train in regularly when preparing for competition in the heat [39].

Conclusions

This thesis builds upon existing work that aims to better understand how humans physiologically adapt to the heat. This concept was modelled for a representative indoor and outdoor worker in a natural environment (acclimatisation) and examined experimentally on eight healthy adults in an artificial environment (acclimation). Our aim was to use both possible induction pathways to show the potential to adapt with a large heat stimulus involving long duration continuous exercise in hot environments (acclimation), and if that magnitude of adaptation was likely or even possible when adding the variability of natural climatic conditions and lower intensities representative of occupational roles (acclimatisation). Additionally, this thesis aimed to improve accessibility to information that could be imminently useful for policy creation and public health guidance by developing a methodology that would provide insight into adaptation level without the need for invasive or expensive equipment. To achieve these aims this thesis used a validated model for predicting heat strain (ISO 7933), a seven consecutive day heat acclimation protocol, and a novel non-invasive hot water immersion test.

In chapter 3, our model predicted that throughout a representative year in Australia, office workers, irrespective of location, are unlikely to receive any protective benefit from the heat due to limited exposure. Further, natural protection for outdoor labourers is somewhat limited unless they engage in additional physical activity beyond the scope of their occupation. Collectively, these findings support that in an Australian context, natural protection from the heat is unlikely for most people given it is exceedingly rare to receive sufficient exposure to experience sustained physiological changes. In contrast, chapter 4 reports changes in maximum skin wettedness as early as 3 days into a 7-day acclimation protocol suggesting that when the heat stimulus is sufficient, intentional exposures can elicit

key adaptive responses in the short-term that can help mitigate heat stress risk. Finally, despite the equipment and procedures needed to identify improvements in key adaptive responses being invasive and complex, chapter 5 suggest that, even with a limited sample size, a novel, non-invasive protocol could be implemented to effectively track changes in true acclimation status. By measuring the time that sweating first occurs and the associated discomfort during a 45-min hot water immersion test, the protocol developed in this thesis could serve as a viable assessment of an individual's level of adaptation to the heat.

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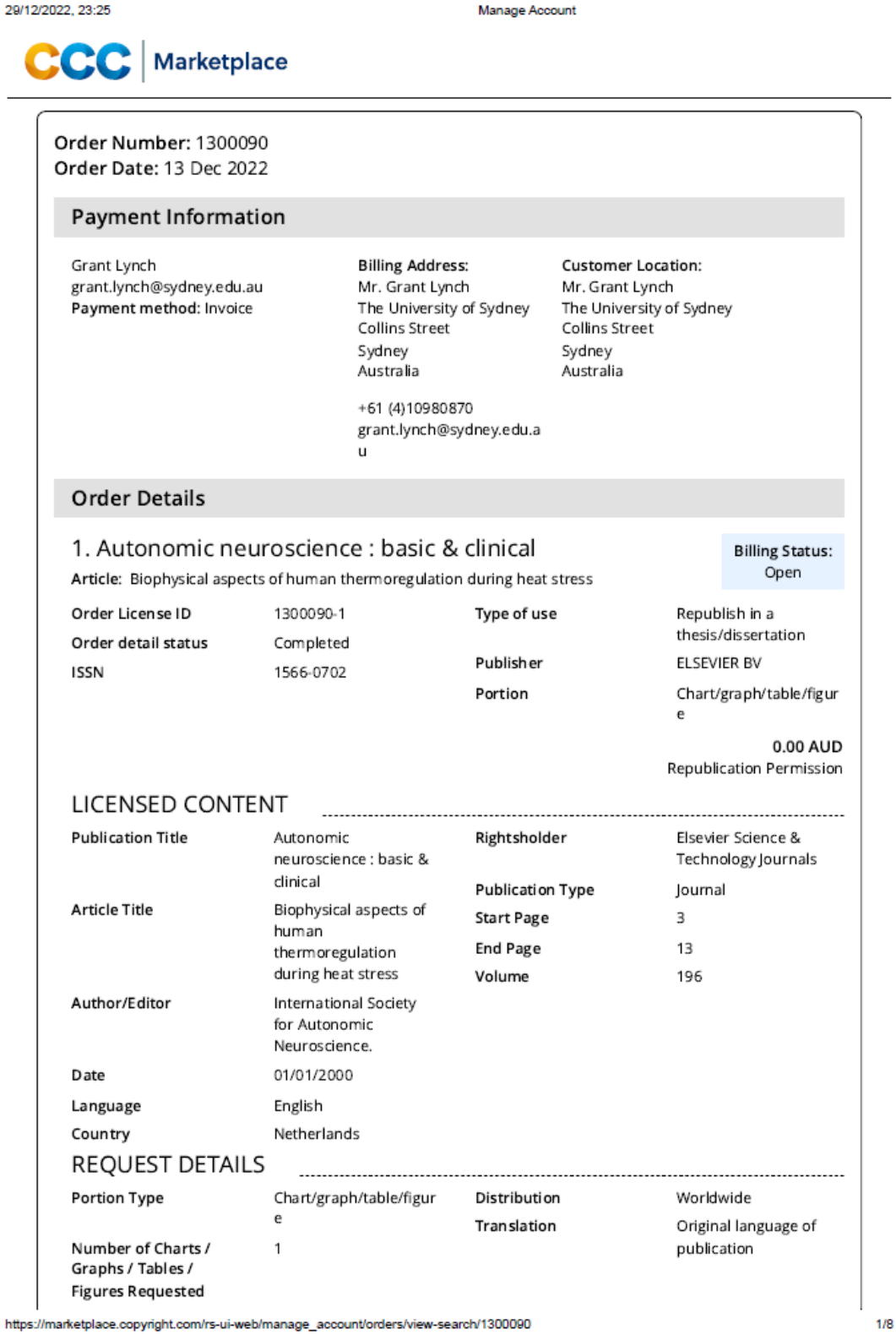
APPENDICES

Appendix A: Permission to use figure from Autonomic Neuroscience



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Appendix D: Ethical Approval for Experimental Studies

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Research Integrity & Ethics Administration
HUMAN RESEARCH ETHICS COMMITTEE

Monday, 10 May 2021

Prof Ollie Jay
Exercise Health and Performance; Faculty of Medicine and Health
Email: Ollie.jay@sydney.edu.au

Dear Ollie,

The University of Sydney Human Research Ethics Committee (HREC) has considered your application.

I am pleased to inform you that after consideration of your response, your project has been approved.

Details of the approval are as follows:

Project No.: **2021/233**
Project Title: **Mapping the time course and impact of human heat acclimation**
Authorised Personnel: **Jay Ollie; Lynch Grant;**
Approval Period: **10 May 2021 to 10 May 2025**
First Annual Report Due: **10 May 2022**

Documents Approved:

Date Uploaded	Version Number	Document Name
03/05/2021	Version 2	PIS (Clean)
18/03/2021	Version 1	Heat Safety Protocol
18/03/2021	Version 1	Flyer B
18/03/2021	Version 1	International Physical Activity Questionnaire
18/03/2021	Version 1	Physical Activity Readiness Questionnaire
18/03/2021	Version 1	Pre Participation Questionnaire
18/03/2021	Version 1	Project Timeline
18/03/2021	Version 1	Flyer A
18/03/2021	Version 1	Consent Form
18/03/2021	Version 1	Email Template

Condition/s of Approval

- Research must be conducted according to the approved proposal.
- An annual progress report must be submitted to the Ethics Office on or before the anniversary of approval and on completion of the project.
- You must report as soon as practicable anything that might warrant review of ethical approval of the project including:
  - Serious or unexpected adverse events (which should be reported within 72 hours).
  - Unforeseen events that might affect continued ethical acceptability of the project.
- Any changes to the proposal must be approved prior to their implementation (except where an amendment is undertaken to eliminate *immediate* risk to participants).
- Personnel working on this project must be sufficiently qualified by education, training and experience for their role, or adequately supervised. Changes to personnel must be reported and approved.
- Personnel must disclose any actual or potential conflicts of interest, including any financial or other interest or affiliation, as relevant to this project.

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- Data and primary materials must be retained and stored in accordance with the relevant legislation and University guidelines.
- Ethics approval is dependent upon ongoing compliance of the research with the *National Statement on Ethical Conduct in Human Research*, the *Australian Code for the Responsible Conduct of Research*, applicable legal requirements, and with University policies, procedures and governance requirements.
- The Ethics Office may conduct audits on approved projects.
- The Chief Investigator has ultimate responsibility for the conduct of the research and is responsible for ensuring all others involved will conduct the research in accordance with the above.

This letter constitutes ethical approval only.

Please contact the Ethics Office should you require further information or clarification.

Sincerely,

Associate Professor Helen Mitchell
Chair
Human Research Ethics Committee (HREC 1)

The University of Sydney of Sydney HRECs are constituted and operate in accordance with the National Health and Medical Research Council's (NHMRC) National Statement on Ethical Conduct in Human Research (2018) and the NHMRC's Australian Code for the Responsible Conduct of Research (2018)

Appendix E: Article Published in Medicine & Science in Sport & Exercise During PhD Candidature

In-Play Cooling Interventions for Simulated Match-Play Tennis in Hot/Humid Conditions

DANIELA SCHRANNER[1,2], LISA SCHERER[1,2], GRANT P. LYNCH[2], SVENJA KORDER[1,2], JOHN R. BROTHERHOOD[2], BABETTE M. PLUIM[3], JULIEN D. PÉRIARD[4], and OLLIE JAY[2]

[1]Technical University of Munich, Institute of Ergonomics, Garching, GERMANY; [2]Thermal Ergonomics Laboratory, Exercise and Sport Science, Faculty of Health Sciences, University of Sydney, NSW, AUSTRALIA; [3]Medical Department, Koninklijke Nederlandse Lawn Tennis, Amersfoort, THE NETHERLANDS; and [4]Athlete Health and Performance Research Centre, Aspetar Orthopaedic and Sports Medicine Hospital, Doha, QATAR

ABSTRACT

SCHRANNER, D., L. SCHERER, G. P. LYNCH, S. KORDER, J. R. BROTHERHOOD, B. M. PLUIM, J. D. PÉRIARD, and O. JAY. In-Play Cooling Interventions for Simulated Match-Play Tennis in Hot/Humid Conditions. *Med. Sci. Sports Exerc.*, Vol. 49, No. 5, pp. 991–998, 2017. **Purpose**: This study aimed to assess the efficacy of different in-play cooling strategies for mitigating heat strain during simulated tennis match-play activity in a hot/humid environment representing the most extreme conditions during the US Open (36°C, 50% relative humidity). **Methods**: On three occasions, nine males completed an intermittent treadmill protocol with an exercise intensity and activity profile simulating a four-set tennis match, with 90-s breaks between odd-numbered games and 120-s breaks between sets, according to International Tennis Federation rules. During breaks, 1) the currently used cooling strategy—an ice-filled damp towel around the neck and a cold-damp towel on the head and thighs (ICE); 2) wetting of arms, neck, face, and lower legs with a sponge in front of an electric fan (FAN_{wet}); or 3) no cooling (CON) were applied. Rectal (T_{re}) and mean skin (T_{sk}) temperature and HR were measured throughout. Thermal sensation and RPE were assessed during breaks. Trials were terminated upon reaching a $T_{re} \geq 39.5$°C or volitional exhaustion. **Results**: Seven, five, and one participant completed FAN_{wet}, ICE, and CON, respectively. By end set 1, ΔT_{re} was lower in FAN_{wet} (0.92°C ± 0.15°C) compared with CON (1.09°C ± 0.09°C, $P = 0.01$), and by end set 2, ΔT_{re} was lower ($P < 0.001$) in FAN_{wet} (1.55°C ± 0.23°C) and ICE (1.59°C ± 0.17°C) compared with CON (1.99°C ± 0.19°C). Mean RPE (FAN_{wet} = 13.9 ± 2.2, ICE = 13.6 ± 1.8, CON = 16.6 ± 1.8), HR (FAN_{wet} = 163 ± 21, ICE 164 ± 22, CON = 175 ± 19 bpm), T_{sk} (FAN_{wet} = 36.56°C ± 0.69°C, ICE 36.12°C ± 0.44°C, CON = 37.21°C ± 0.42°C), and thermal sensation were lower in FAN_{wet} and ICE ($P < 0.05$) compared with CON by end set 2. **Conclusions**: The currently recommended ICE strategy successfully mitigates thermal strain during simulated tennis match play in hot/humid conditions. The FAN_{wet} intervention is an equally effective alternative that may be more practical in limited resource settings. **Key Words:** HEAT STRESS, THERMOREGULATION, THERMAL STRAIN, SPORT

Among the tennis Grand Slam tournaments, the greatest number of heat-related health problems has occurred at the Australian and US Open tournaments. During the 2014 US Open alone, 12 male players had to abort play, at least four of them because of heat-related health issues. All incidents occurred at air temperatures ≥35°C with a relative humidity (RH) of ~50%. Under such conditions, the ability to dissipate heat to the surrounding environment is mostly dependent on evaporation, which is greatly limited. The resultant accumulation of heat inside the body can prove deleterious to performance and health if exercise is continued and countermeasures are not taken (6). Indeed, peak core temperatures of 38.9°C ± 0.3°C and 39.1°C ± 0.3°C have been reported during professional hard court tennis match play in males and females, respectively (4).

The principal in-play cooling intervention presently recommended by the International Tennis Federation (ITF) is the application of ice wrapped in a wet towel around the neck with damp, cold towels simultaneously placed on the head and thighs ("ice towels") (10). Despite their widespread use, the efficacy of this cooling strategy has not yet been assessed. Although some neck-cooling devices have been demonstrated to improve running performance in hot and humid conditions (29), there is no evidence that the currently recommended "ice towel" intervention is an effective cooling strategy for mitigating thermal strain when applied for only the short duration of in-play breaks (90–120 s) permitted by the standard structure of tennis play.

The principal heat loss avenue with the application of "ice towels" is conduction. Heat is transferred from the skin through direct contact with the colder surface of the towel. The heat loss capacity of this intervention is therefore determined by the total skin surface area in contact with the

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towels, the temperature difference between the skin, and the towel surface and the latent heat of fusion of ice (334 $J \cdot g^{-1}$) (22). However, the efficacy of this cooling strategy may be limited because of the small surface area covered (neck, head, and thighs), which is consequently available for heat exchange. Further, temperature differences between the towels and the skin will likely become progressively smaller as the surface temperatures of the towels and skin equilibrate during application, whereas the evaporation of sweat may also be simultaneously impeded from covered skin surfaces (22).

Relative to the melting of ice, the evaporation of water from the skin surface liberates approximately seven times more heat per gram (2427 $J \cdot g^{-1}$) (30). A cooling strategy promoting evaporative heat loss may therefore present a more efficacious alternative than the "ice towel" method. Public health heat wave guidance has previously suggested that fan use does not enable body cooling. However, recent studies have demonstrated that fan use can be beneficial at peak air temperatures experienced at the US Open (35°C–36°C) irrespective of ambient humidity (15). Moreover, maximizing skin moisture coverage by wetting the skin with a damp sponge should ensure the maximum potential for heat loss via evaporation is realized.

The aim of the present study was to assess the efficacy of 1) the currently recommended in-play cooling strategy for professional tennis ("ice towels" [ICE]) and 2) a new cooling strategy that optimizes evaporative heat loss (fan use with skin wetting: FAN_{wet}), for mitigating physiological and perceptual heat strain during a simulated tennis match play in an environment representing the peak heat stress conditions experienced at the US Open (36°C, 50% RH). It was hypothesized that relative to a control condition without active cooling, the FAN_{wet} cooling strategy during mandated rest breaks throughout a simulated tennis match activity would best mitigate elevations in thermal and perceptual strain in a hot and humid environment representative of the peak conditions historically experienced at the US Open.

METHODS

Participants

Nine healthy trained males, nonacclimatized to heat, were recruited to participate in the study. Their mean age, height, body mass, body fat, and maximum oxygen uptake ($\dot{V}O_{2max}$) were 25 ± 4 yr, 179.2 ± 5.7 cm, 77.3 ± 6.7 kg, 12.7% ± 6.1%, and 50.6 ± 6.3 $mL \cdot kg^{-1} \cdot min^{-1}$, respectively. Before any testing, participants gave written informed consent after they were informed about the risks of the study. All participants took part in one preliminary session and three experimental sessions on different days, separated by at least 48 h. The time of testing was kept constant within each subject. Participants refrained from strenuous physical activity and alcohol ingestion for 24 h before testing and from caffeine ingestion on testing days. All experimental procedures were first approved by the University of Sydney Human Research Ethics Committee.

Instrumentation

Rectal temperature (T_{re}) was measured in 8 of the 9 participants at 5-s intervals using a Mon-a-therm general-purpose thermistor probe 400TM (Covidien, Mansfield, MA) that was self-inserted 15 cm past the anal sphincter. Skin temperature (T_{sk}) was measured every minute using wireless iButtons (DS1921H; Maxim Integrated Products Inc., San Jose, CA) taped to the chest, shoulder, thigh, and calf, and a four-point weighted mean value was determined according to the weighting of Ramanathan (24). Heart rate (HR) was monitored every 5 s by using a Polar RS 400 watch (Polar Electro Oy, Kempele, Finland).

For subjective measures, participants were asked to rate their thermal sensation (TS) using a bipolar 200-mm visual analog scale ranging from "very cold" (0 mm), to "neutral" (100 mm), to "very hot" (200 mm). Further, the effects on potential performance were assessed using the 6- to 20-point Borg scale RPE but with a modified question: "How easy it is to continue the exercise?"

Protocol

Preliminary session. Upon arrival, the participant's physical characteristics were measured. Height and weight were measured using a wall-mounted stadiometer (HR-200; Tanita, Arlington Heights, IL) and digital scale (BWB-800, Tanita), respectively. Body composition was measured by dual-energy x-ray absorptiometry (GE-LUNAR Prodigy module; GE Medical Systems, Madison, WI). Then after a light warm-up, $\dot{V}O_{2max}$ was assessed on a treadmill according to the procedures set forth by the Canadian Society for Exercise Physiology (9). All respiratory variables were measured throughout, using a stationary pulmonary gas analyzer system (Quark CPET; Cosmed, Rome, Italy). Participants also completed five games (~16 min) of simulated tennis match-play activity (cf. Experimental session section) to familiarize themselves with the experimental protocol and to ensure that the average $\dot{V}O_2$ during the exercise protocol was comparable with the $\dot{V}O_2$ values (and therefore metabolic heat production) previously reported (23–29 $mL \cdot kg^{-1} \cdot min^{-1}$) during live professional tennis match play (11,27). Accordingly, a mean $\dot{V}O_2$ of 26.3 ± 1.2 $mL \cdot kg^{-1} \cdot min^{-1}$ was measured.

Experimental session. Participants performed three different trials in a counterbalanced order. All trials were completed in a climate-controlled chamber in the Thermal Ergonomics Laboratory at the University of Sydney. Upon arrival before each session, urine specific gravity (USG) was determined with a refractometer UG-α (Atago Co., Ltd., Tokyo, Japan). A cutoff USG of >1.020 was used to ensure that the participants were adequately hydrated (1) before starting the experiment. None of our participants exceeded this USG cutoff value on any occasion. Participants then inserted their T_{re} probe and were weighed. After the remaining instrumentation was completed, they entered the climate chamber regulated at an air temperature of 36.5°C ± 0.4°C and an RH of 51.0% ± 1.8%; these conditions were selected to simulate the

most extreme weather reported at the US Open in recent years. The participant conducted a 5-min warm-up on the treadmill at a self-chosen intensity, which was the same between all trials. Then the exercise protocol—designed to simulate the metabolic load of a tennis match—began with the preprogramed exercise profile controlled via hp-paragraphics (H/P/Cosmos Sports & Medical GmbH, Nussdorf-Traunstein, Germany). To simulate one "point," the participant ran at 16 $km \cdot h^{-1}$ for a variable period of 3–7 s (note: this time excludes 3 s of acceleration time), randomly selected for every "point" but with a mean "point" duration of 6 s within each "game," to align with the reported average point length for male elite tennis players at the US Open (16,20). Although $\dot{V}O_2$ was not measured during the experimental trials because of concerns that the addition of a facemask would disproportionately affect perceptual, and possibly physiological, thermal strain, the maintenance of the same average point length within each "game" ensured a similar cumulative $\dot{V}O_2$ throughout. Running at top speed was followed by a 3-s deceleration phase, a 3-s walk at 2 $km \cdot h^{-1}$, and a standing phase of 5 s. A total of six "points" constituted one "game," and eight "games" constituted one "set." In total, four "sets" were completed in each trial. According to the current regulations of the ITF, after every odd-numbered game (i.e., game 1, 3, 5, etc.), a rest break of 90 s was enforced, and after every "set," a rest break of 120 s was enforced, during both of which one of the three cooling strategies (see next section) were applied. TS was assessed three times per break: 1) directly before applying the cooling intervention, 2) immediately after removal of the cooling intervention, and 3) after they completed the next "game" of the protocol. In total, the simulated four-set match lasted a maximum of 108 min and 12 s for each trial. Trials were terminated early if T_{re} exceeded 39.5°C or volitional exhaustion occurred.

Cooling Strategies

During each of the experimental trials, one of the following three cooling strategies was applied in each of the rest breaks.

Ice towel (ICE). For one trial, two sets of three towels (70 × 140 cm, 100% cotton) were soaked with cold water and wrung. Per towel set, one damp towel, for application on the neck, was filled with 3 kg of ice and separated into three equal sections with a shoelace, and two damp chilled towels without ice were placed on the thighs and over the head. This strategy was performed according to current practice in elite tennis (E. Brady, personal communication, WTA, ITF Medical Commission, 2015). The towel set used in sets 1 and 2 was replaced at the beginning of set 3 and used for the remainder of the trial.

Fan with skin wetting (FAN_{wet}). Participants moistened the surface of their uncovered neck, face, arms, and thighs (for ~5 s) with a damp sponge that was soaked in water (initially at 15°C) and wrung out with both hands for ~2 s over a bucket before application, so no excess water dripped. Simultaneously, a commercially available 45-cm diameter fan (MFE745S2DQ, Moretti) placed 1.5 m in front of the participant, set at its highest speed, generated an air velocity of 6.4 $m \cdot s^{-1}$. The water was not replaced throughout the trial and consequently warmed by ~8°C–10°C by the end of the trial.

Control (CON). Participants sat quietly and applied no cooling intervention for the duration of each break, but they were allowed to ingest cold water.

Throughout each participant's first experimental trial (which was randomized between participants), they were allowed to drink cold water (~7°C) *ad libitum* during each mandated rest break. The amount of water ingested was measured and subsequently fixed for the remaining two trials to prevent differences in internal heat loss and any differences in sweat capacity because of the stimulation of abdominal thermoreceptors (18).

Statistical Analyses

Rectal temperature, HR, and RPE were averaged over the last 15 s of every odd game (i.e., games 1, 3, 5, and 7), and the last game, in each "set." Values for T_{re}, HR, and RPE were analyzed using a three-way repeated-measures ANOVA using the independent variables of "cooling intervention" (three levels: CON, ICE, and FAN_{wet}), "set" (four levels: sets 1, 2, 3, and 4), and "game" (five levels: games 1, 3, 5, 7, and the end of the set [game 8]). Note that the "cooling intervention" independent variable was reduced to two levels (ICE and FAN_{wet}) for analysis of data in sets 3 and 4 as a high dropout rate because of exhaustion, or a high core temperature was observed in the CON trial. TS and skin temperature from each "set" were separately assessed using a similar three-way repeated-measures ANOVA with the same independent variables of "cooling intervention" and "game," but with the additional independent variable of "time" (three levels: start of each break ["precool"], end of each break ["postcool"], and end of the following game ["next game"]). If the Mauchly's test indicated a violation of sphericity, a Greenhouse–Geisser correction was used. The alpha error level was set at $P < 0.05$ to establish statistical significance and maintained at that level during pairwise comparisons using a Holm–Bonferroni correction. The data are presented as mean ± SD, unless otherwise indicated. All statistical analyzes were performed using SPSS (Version 22.0; IBM SPSS Inc., Chicago, IL).

RESULTS

Duration/dropout rate. Of the nine participants, seven completed the FAN_{wet} trial, five completed the ICE trial, and only one completed the CON trial. Reasons for withdrawal are illustrated in Figure 1. Compared with the CON trial (79.8 ± 22.4 min), mean exercise time was longer (P = 0.021) in the FAN_{wet} trial (106.7 ± 4.4 min) and tended to be longer (P = 0.057) in the ICE trial (96.2 ± 23.9 min).

Rectal temperature (T_{re}). Mean baseline absolute T_{re} was similar (P = 0.586) between ICE (36.9°C ± 0.3°C),

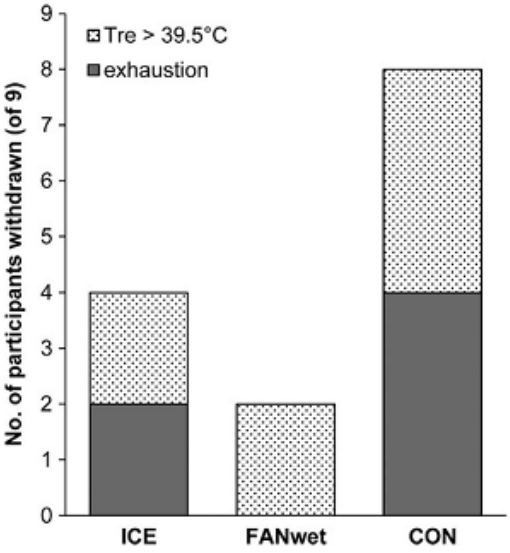

FIGURE 1—Number of participant withdrawals because of exhaustion or reaching the termination core temperature criterion (T_{re} > 39.5°C) in each trial.

FAN_{wet} (36.8°C ± 0.4°C), and CON (36.8°C ± 0.2°C). In the initial phase of set 1, the rise in T_{re} was similar between all trials. However, by game 7 of set 1, ΔT_{re} in the FAN_{wet} trial was lower relative to the CON trial (Fig. 2). Moreover, by game 1 of set 2, ΔT_{re} values in the FAN_{wet} and ICE trials were lower compared with CON (Fig. 2). These differences in ΔT_{re} persisted throughout the remainder of the "match" ($P < 0.001$) with a very similar ΔT_{re} evident between ICE and FAN_{wet}.

Skin temperature (T_{sk}). Mean baseline T_{sk} was similar ($P = 0.582$) between the ICE (35.6°C ± 0.8°C), the FAN_{wet} (35.8°C ± 0.6°C), and the CON (35.8°C ± 0.5°C) trials. In set 1, T_{sk} was lower in the ICE trial compared with CON before cooling ($P = 0.002$); however, immediately after cooling, T_{sk} was lower in both ICE ($P = 0.001$) and tended to be lower in FAN_{wet} ($P = 0.076$) relative to CON. By the end of the following game after each break, T_{sk} was lower in FAN_{wet} compared with CON ($P = 0.009$), but T_{sk} was lower in ICE compared with FAN_{wet} ($P = 0.013$; Fig. 3). In set 2, T_{sk} was lower in the ICE and FAN_{wet} trials compared with CON, both precooling and postcooling. These lower skin temperatures compared with CON remained until at least the end of the next game (Fig. 3). The same response as set 2 was evident in set 3, but T_{sk} became progressively higher in CON, yet T_{sk} was at similar levels to set 2 in ICE and FAN_{wet} and was not different between these two cooling trials (Fig. 3). Similarly, T_{sk} was highest in CON in set 4, and T_{sk} remained similarly low in the FAN_{wet} and ICE trials (Fig. 3).

TS. In set 1, TS for FAN_{wet} and ICE was cooler immediately after cooling compared with precooling. Moreover, TS was cooler postcooling compared with CON (Fig. 3). However, TS had returned to similar levels to CON in both the FAN_{wet} and the ICE trials by the end of the next game. In sets 2 and 3, TS was already lower at the start of the break in ICE and FAN_{wet} compared with CON. This difference became greater postcooling, and although TS returned to precooling levels by the end of the next game, it remained lower compared with CON in ICE and FAN_{wet} (Fig. 3). In set 4, TS

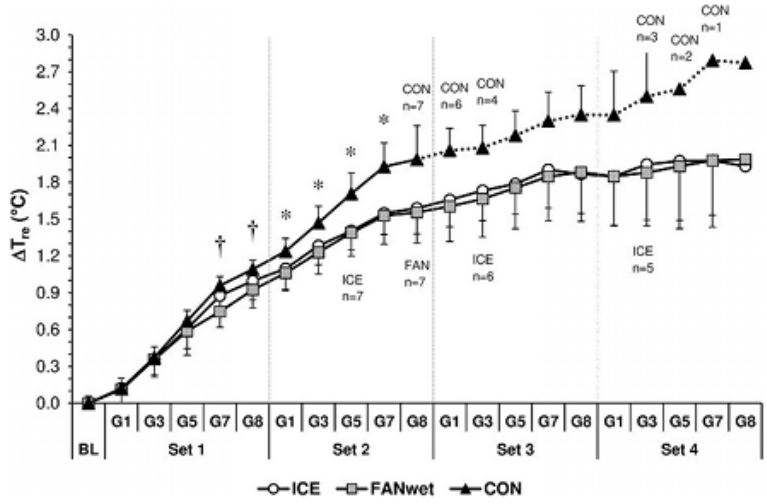

FIGURE 2—Changes in rectal temperature (ΔT_{re}) over the entire simulated tennis match. Vertical lines denote the end of each "set." Remaining participants (n) out of eight are indicated for each particular trial. Data are presented as means ± SEM. †Difference between control and FAN_{wet} trial ($P < 0.05$). *Difference between control and both ICE and FAN_{wet} trials ($P < 0.05$).

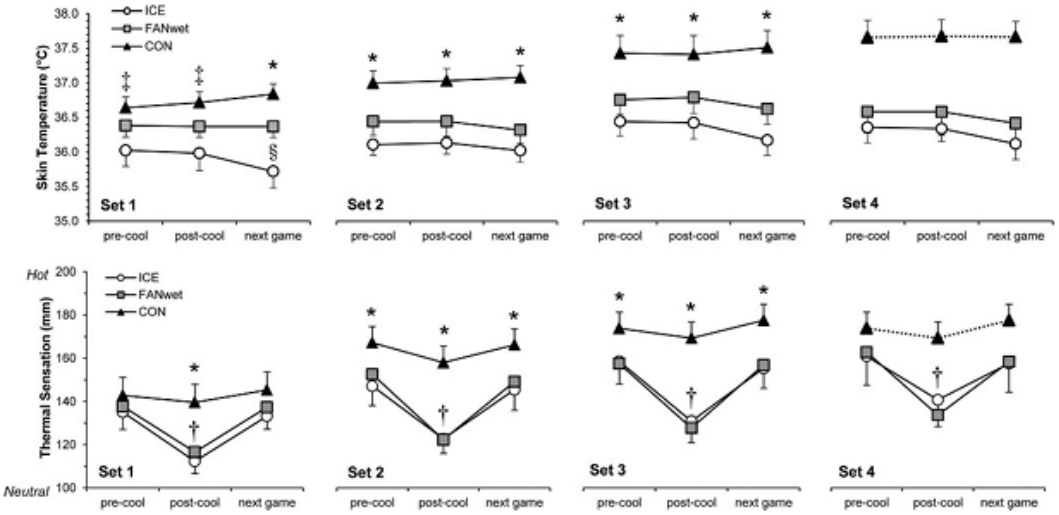

FIGURE 3—Mean T_{sk} values (top row) and mean TS votes (bottom row) within each "set" directly before the application of cooling (precooling), immediately after cooling (postcooling), and at the end of the next "game" that followed (next game). Remaining participants (*n*) are indicated for each particular trial. For set 4, data are limited to ICE and FAN_{wet} because of high drop out during CON. As there was no significant cooling intervention–game–time interaction, data within each set (i.e., from games 1, 3, 5, 7, and 8) were collapsed. Data are presented as means ± SEM. *Difference between control and both ICE and FAN_{wet} trials ($P < 0.05$). ‡Difference between ICE and CON only ($P < 0.05$). §Difference between ICE and FAN_{wet} ($P < 0.05$).

postcooling remained cooler relative to precooling in ICE and FAN_{wet}.

Exercise perception (with RPE scale). Exercise perception throughout simulated match-play activity is illustrated in Figure 4A. RPE tended to be lower in the ICE trial (8.3 ± 2.2, $P = 0.051$) but not the FAN_{wet} trial (9.0 ± 2.5, $P = 0.58$) compared with CON (9.4 ± 2.3) after the first game of set 1. At the end of set 1, RPE was lower (11.4 ± 2.3, $P = 0.023$) in the ICE trial and tended to be lower in the FAN_{wet} trial (11.9 ± 2.3, $P = 0.063$) compared with CON (12.9 ± 2.0). At the end of set 2, RPE was lower in the ICE trial (13.6 ± 1.8, $P = 0.004$) and the FAN_{wet} trial (13.9 ± 2.2, $P = 0.008$) compared with CON (16.6 ± 1.8). RPE was similar between ICE and FAN_{wet} at the end of set 3 (15.4 ± 3.3 vs 15.3 ± 2.4, $P = 0.23$) and set 4 (15.7 ± 3.2 vs 15.8 ± 2.4, $P = 0.19$).

HR. Mean HR throughout the simulated matches are given in Figure 4B. HR tended to be higher in CON compared with both FAN_{wet} ($P = 0.09$) and ICE ($P = 0.10$) by the end of set 1. HR in CON was higher compared with the ICE ($P = 0.016$) and FAN_{wet} ($P = 0.020$) trials by the end of set 2, but similar between ICE and FAN_{wet} ($P = 0.81$). HR was similar between ICE and FAN_{wet} at the end of set 3 (162 ± 19 vs 163 ± 20 bpm, $P = 0.57$) and set 4 (159 ± 15 vs 162 ± 17 bpm, $P = 0.27$).

DISCUSSION

The present study is the first to our knowledge to directly assess the efficacy of different in-play cooling interventions applied during mandated breaks in simulated tennis match-play activity for mitigating physiological and perceptual heat strain in hot/humid conditions. Compared with a control (CON) condition during which only cold water ingestion was permitted, the currently recommended (and used) "ice towel" cooling strategy that augments conductive heat loss by wrapping an ice-filled damp towel around the neck and placing two cold, damp towels on the head and thighs (ICE) led to a ~0.5°C smaller elevation in core temperature, an approximately two- to three-point lower RPE, a ~15-bpm lower HR, and a cooler whole-body TS. Promoting evaporative heat loss using the different cooling strategy of wetting the skin of the arms, neck, face, and lower legs with a damp sponge in front of an electric fan (FAN_{wet}) diminished physiological and perceptual heat strain to similar levels as the ICE trial compared with CON.

Under the very hot/humid ambient conditions assessed in the present study, four of the nine participants could not complete the four-set simulated tennis match because of exhaustion without an active cooling strategy (cold fluid ingestion only); and even with the ICE strategy, two participants stopped before the end of the trial because of exhaustion (Fig. 1). Given that the rate of heat-related incidents in tournaments such as the US Open is relatively low, this may indicate that the combination of climate and exercise in the present study represents the very worst-case scenario. The exercise intensities were selected to replicate rates of metabolic heat production and activity profiles previously measured in elite tennis players (11,27); however, the environmental conditions (36°C, 50% RH) have only been reached on one occasion (in 2014), with peak air temperatures for the US Open in most years typically

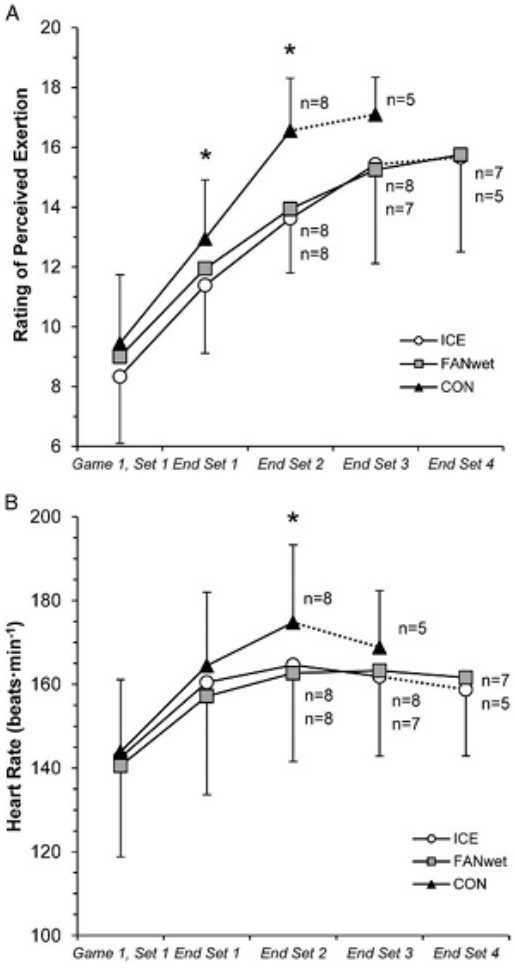

FIGURE 4—HR (A) and RPE (B) after the first "game" of the first "set" and then at the end of each "set." For set 4, data are limited to ICE and FAN_{wet} because of high drop out during CON. Data are presented as means ± SEM. *Significant main effects ($P < 0.05$).

closer to ~30°C–32°C. Moreover, the exercise protocol, while separating bouts of short duration fast running with intermediate periods of walking and standing (based on the activity profiles of elite tennis match play), required participants to run at the same speed (and generate the same $\dot{V}O_2$) in all sets, which was necessary to isolate the independent influence of cooling strategy on thermal strain. In a real-world scenario, however, an athlete would be free to behaviorally adapt as RPE and HR increases in later sets (Fig. 4A and B) before reaching exhaustion. Recent research has demonstrated that such behavioral thermoregulation occurs in tennis players competing in hot environments in the form of longer pauses in play between points (23) as opposed to a downregulation in exercise intensity as indicated by running speed (13) and point duration (23). Nevertheless, both the ICE and the FAN_{wet} strategies successfully dampened the progressive rise in RPE, which would have presumably reduced any heat-related decrements in performance in a competition setting (14). The ability to subjectively tolerate the heat during exercise is also directly influenced by aerobic fitness (7). The mean $\dot{V}O_{2max}$ of our participants of ~51 $mL \cdot kg^{-1} \cdot min^{-1}$ is slightly lower than the $\dot{V}O_{2max}$ values reported in elite male tennis players (~58 $mL \cdot kg^{-1} \cdot min^{-1}$ [5]), but no association was observed within our data between $\dot{V}O_{2max}$ and exercise duration. Moreover, there seems to be no reason to believe that the efficacy of any one cooling intervention would be altered by differences in $\dot{V}O_{2max}$.

Although the present study was designed to assess the efficacy of different cooling strategies for elite tennis players in a worst-case US Open setting, the findings are also relevant to subelite athletes, as well as elite tennis tournaments scheduled annually in various geographical regions with hot/humid conditions; for example, Australia (Sydney and Brisbane: January), Brazil (Rio de Janeiro and Sao Paulo: February), UAE (Dubai: February), Mexico (Acapulco: February), USA (Miami: March; Washington DC, Atlanta, Cincinnati, and Winston-Salem: August), Morocco (Marrakech: April), and China (Shenzen: September). The findings associated with the FAN_{wet} trial in particular are potentially most applicable to lower resource settings (e.g., amateur, club, and junior tennis matches) that would most likely not have the infrastructure necessary to prepare and replenish "ice towels." Indeed, the FAN_{wet} strategy presents a lower logistical burden requiring only a bucket of water, a sponge, and an electric fan, which could be battery powered. One potential practical disadvantage of the FAN_{wet} strategy could be increased risk of excess water soaking the athlete's clothing/shoes, which would probably be undesirable. During pilot testing, we found that the optimal method for avoiding this scenario was to wring out the sponge for 2 s over the bucket before applying to the skin. The water temperature in the FAN_{wet} strategy was ~15°C (typical cold tap water temperature), and the benefits were observed without the need to maintain a specific water temperature, as the water was not replaced throughout the trial. Given that this method primarily liberates heat via evaporation, one would not expect large differences in net heat dissipation with warmer or cooler water. However, the application of cooler water directly to skin may elicit a cooler TS because of a greater activation of cold-sensitive cutaneous thermoreceptors, which are densely populated on the neck and face (21,26).

Although TS was not different between the ICE/FAN_{wet} and the CON trials, both before each cooling period (precool) and by the end of the game after each cooling period (next game) throughout set 1 (Fig. 3), differences in T_{re} progressively developed across the same timeframe (Fig. 2). This observation demonstrates the potential benefit of beginning to apply either ICE or FAN_{wet} cooling strategies from the beginning of a tennis match, even before an athlete starts to feel the effects of the hot environment. Moreover, there was a

tendency for a lower RPE even after the application of the ICE intervention after the very first game of set 1. Weighted mean skin temperature (T_{sk}) was lowest with the ICE strategy (Fig. 3). Thirty percent of T_{sk} is determined by a sensor placed on the chest, which would have been covered and therefore directly cooled by the ice-filled towel wrapped around the neck. However, because 30% of the total body surface area was not cooled by the "ice towel," the reductions in whole-body T_{sk} in the ICE trial are probably slightly overestimated. Nevertheless, reanalysis of whole-body T_{sk} estimated without the chest still yielded a lower T_{sk} in the ICE trial relative to CON in all sets. Both the ICE and the FAN_{wet} cooling strategies had a more acute influence on TS compared with the changes observed in T_{sk} (Fig. 3), which can be explained by the strong influence of local skin temperature of the face/neck and hands on whole-body TS despite high skin temperatures elsewhere (2,3).

It was initially hypothesized that the greater conductive cooling with the ICE strategy would elicit skin cooling and a cooler TS but not necessarily lead to sufficiently large differences in overall heat dissipation to lower T_{re} because of the short application time (90-s breaks in play) and the greater barrier to evaporation from towels covering a large portion of skin surface. On the contrary, T_{re} was lower by the beginning of set 2 compared with the CON trial, indicating that the volume of conductive heat loss possible during the short breaks exceeded any reductions in latent heat loss that may have been minimized by evaporation from the towels themselves as they were moistened before applying. Cooling of the neck and thus blood ascending along the carotid artery has been postulated to lower brain temperature (28), which may have also occurred in the ICE trial. However, this would mostly likely not have been reflected by our core temperature measurements as they were measured in the rectum. Even if any cooling of the brain did occur, it did not lead to a cooler TS in the ICE trial compared with the cooling trial that did not involve local cooling of the neck (FAN_{wet} trial), and heat tolerance, which is postulated to be higher with brain cooling (19), was actually slightly lower in the ICE trial compared with FAN_{wet} (Fig. 1). Nevertheless, considering that the primary purpose of these cooling strategies is to prevent heat-related illnesses, the temperature elevation of the deep viscera, which is well characterized by T_{re} (12,25), was arguably the most important indicator of thermal status to assess because these are the temperature-sensitive tissues that are directly implicated in the development of exertional heat illness (17).

An experimental condition using fan use only without supplemental skin wetting was not assessed in the present study, so we therefore cannot dissociate the relative benefit of applying additional moisture to the skin surface from additional convection in the FAN_{wet} trial. Indeed, during maximal sweating, full skin area sweat coverage would be expected in a fully heat-acclimated individual (8), but such coverage would not be attained until steady-state sweating was achieved, which would be unlikely at the start of a match. The benefits of skin wetting were therefore probably most pronounced during the early stages of the simulated match. Augmented airflow could not have had any detrimental effects from a heat transfer perspective in the present environmental conditions because dry heat gain would not have occurred as air temperature was not greater than T_{sk}, and greater evaporation because of a much higher evaporative heat transfer coefficient with additional airflow would have certainly occurred—as evidenced by the smaller elevations in T_{re}. As the ICE and FAN_{wet} interventions were found to be equally effective at mitigating thermal strain, tennis players should be encouraged to adopt the strategy that suits their own personal preference and/or that which is available at specific events. Considerations include the time required to prepare "ice towels" and the resources needed (e.g., freezer for >3 kg of ice; power supply for fan [unless battery powered]). Players may also wish to avoid wet hands from the sponge in the FAN_{wet} trial; however, the nondominant hand can be used, the application time is relatively short (~5–10 s), water only needs to be applied once, and drying of the wet hand(s) will be accelerated by airflow from the fan for the remainder of the break (>60 s). The findings of the present study are only applicable to hot/humid environmental conditions and not necessarily hot/dry conditions characteristic of tournaments such as the Australian Open (e.g., ~44°C, <10% RH) because amplified airflow would (a) probably increase dry heat gain via convection because of a negative temperature gradient between the skin and the air and (b) potentially not increase evaporation (particularly without supplemental skin wetting) as sweat readily evaporates in dry climates. It also worth considering that the present environmental conditions do not account for any additional heat load that may arise from direct or reflected solar radiation. Finally, although almost identical, physiological, and perceptual responses were observed between ICE and FAN_{wet} trials, two participants withdrew because of exhaustion in the ICE trial, but none withdrew for this reason in the FAN_{wet} trial. The underlying reason for this difference is unclear, and it is possible that factors contributing to exhaustion other than those measured in the present study may have been altered by the cooling intervention used.

CONCLUSION

Relative to a control condition, during which only cold water ingestion was permitted during mandated breaks in play, the application of the currently recommended ICE cooling strategy successfully mitigated the development of physiological and perceptual heat strain during a simulated four-set tennis match in hot/humid environmental conditions representative of the peak heat stress historically recorded at the US Open. The FAN_{wet} cooling strategy during breaks in play was equally effective as the ICE strategy for reducing thermal strain and may present a more practical alternative in lower resource settings.

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The authors declare no conflict of interest. The results of the present study do not constitute endorsement by the American College of Sports Medicine. The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

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Appendix F: First Article Published in Journal of Science and Medicine in Sport During PhD Candidature

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Original research

Optimal cooling strategies for players in Australian Tennis Open conditions

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ABSTRACT

Objectives: We compared the utility of four cooling interventions for reducing heat strain during simulated tennis match-play in an environment representative of the peak conditions possible at the Australian Open (45 °C, <10% RH, 475 W/m^2 solar radiation).
Design: Nine trained males undertook four trials in a climate chamber, each time completing 4 sets of simulated match-play.
Methods: During ITF-mandated breaks (90-s between odd-numbered games; 120-s between sets), either iced towels (ICE), an electric fan (FAN_{dry}), a fan with moisture applied to the skin (FAN_{wet}), or ad libitum 10 °C water ingestion only (CON) was administered. Rectal temperature (T_{re}), mean skin temperature (T_{sk}), heart rate (HR), thermal sensation (TS), perceived exertion (RPE) and whole body sweating (WBSR) were measured.
Results: After set 3, T_{re} was lower in ICE (38.2 ± 0.3 °C) compared to FAN_{dry} (38.7 ± 0.5 °C; $p = 0.02$) and CON (38.5 ± 0.5 °C; $p = 0.05$), while T_{re} in FAN_{wet} (38.2 ± 0.3 °C) was lower than FAN_{dry} ($p = 0.05$). End-exercise T_{re} was lower in ICE (38.1 ± 0.3 °C) and FAN_{wet} (38.2 ± 0.4 °C) than FAN_{dry} (38.9 ± 0.7 °C; $p < 0.04$) and CON (38.8 ± 0.5 °C; $p < 0.04$). T_{sk} for ICE (35.3 ± 0.8 °C) was lower than all conditions, and T_{sk} for FAN_{wet} (36.6 ± 1.1 °C) was lower than FAN_{dry} (38.1 ± 1.3 °C; $p < 0.05$). TS for ICE and FAN_{wet} were lower than CON and FAN_{dry} ($p < 0.05$). HR was suppressed in ICE and FAN_{wet} relative to CON and FAN_{dry} ($p < 0.05$). WBSR was greater in FAN_{dry} compared to FAN_{wet} ($p < 0.01$) and ICE ($p < 0.001$).
Conclusions: Fan use must be used with skin wetting to be effective in hot/dry conditions. This strategy and the currently recommended ICE intervention both reduced T_{re} by ~0.5–0.6 °C and T_{sk} by ~1.0–1.5 °C while mitigating rises in HR and TS.

1. Introduction

Tennis is an intermittently high intensity sport often played in hot climates, with a significant risk of heat-related illness.[1] This problem was most evident during the 2014 Australian Tennis Open when ambient temperatures peaked at 44 °C, leading to the implementation of their extreme heat policy. A key aspect of this policy was the widespread use of various cooling interventions to mitigate thermal strain during mandated rest periods in match-play between odd-numbered games and between sets.[2] Specifically, ice wrapped in damp towels (ice towels) placed over the head, lap and around the neck are the currently recommended strategy for professional players (Personal communication: Elaine Brady, WTA; May 2015).

The application of ice towels may cool the skin, potentially lowering thermal sensation and improving perceived exertion.[5] However, towels present a barrier to sweat evaporation and skin cooling may cause local vasoconstriction,[6] despite elevations in core temperatures. On extremely hot days, when air temperature (T_a) far exceeds skin temperature (e.g. T_a: 45 °C), supplemental air flow across the skin with devices such as electric fans may increase the amount of heat added to the body via dry heat transfer (convection). But, the evaporation of sweat can be promoted to a far greater degree with the same increase in air flow.[7] Yet, due to the low ambient humidity (<10% RH) typically present at the Australian Open, sweat readily evaporates anyway, meaning greater air flow across the skin may provide no additional evaporation and potentially lead to a greater rise in core temperature due to the additional

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heat gain via convection—unless extra moisture is applied to the skin (e.g. with a damp sponge).[8]

While core temperatures of >39.0 °C have been reported during live professional tennis match-play,[3] to our knowledge only one study has assessed the impact of these different cooling interventions on physiological and perceptual heat strain in a tennis-related context. Specifically, either ice towels or fan use with additional moisture applied to the skin effectively mitigate elevations in thermal and cardiovascular strain in a warm/humid (36 °C, 50% RH) environment.[4] However, due to the aforementioned modifying effect of environmental conditions on evaporative and convective heat transfer, these findings cannot be directly translated to a very hot/dry setting.

Therefore, we compared the efficacy of (i) ice towels (ICE), (ii) electric fan use (FAN_{dry}), (iii) electric fan use with supplemental moisture applied to the skin (FAN_{wet}), and (iv) ad libitum 10 °C water ingestion only (CON), for reducing physiological and perceptual thermal strain during simulated tennis match-play in hot/dry conditions (45 °C, <10% RH). It was hypothesized that thermal and perceptual strain would be lower with FAN_{wet} and ICE compared to CON and FAN_{dry}, but athletes would be (and feel) coolest with FAN_{wet} and hottest with FAN_{dry}.

2. Methods

Nine male recreational athletes (22 ± 3 years; 179 ± 5 cm; 73.9 ± 5 kg; peak oxygen consumption (VO_{2peak}): 51.7 ± 4.6 ml/kg/min; body fat: 12.0 ± 4.7%) free from cardiovascular and metabolic health disorders were recruited for the study. VO_{2peak} inclusion criteria aligned with the existing literature on elite tennis players.[9] Participants did not consume caffeine or alcohol nor partake in strenuous exercise 24-h prior to testing. Approval was obtained from The University of Sydney Human Research Ethics Committee (HREC#:2015/420) and written informed consent was acquired from all participants.

Rectal temperature (T_{re}) was measured with a general-purpose paediatric thermistor probe (TM400, Covidien, Mansfield, MA, USA) inserted to a depth of 15 cm past the anal sphincter. Mean skin temperature (T_{sk}) was estimated using a weighted mean of chest, shoulder, thigh, and calf skin temperatures[10] with wireless temperature sensors (iButtonDS1921H-F5, Embedded Data Systems, USA) secured to the skin surface using surgical tape (e.g. Transpore, 3M, Canada). Heart rate (HR) was measured with a monitor placed around the chest (Polar RS400, Kempele, Finland). Whole body sweat rate (WBSR) was estimated from pre- and post-trial body mass measurements (±2 g) using a platform scale (Mettler, Germany), corrected for weight gain through fluid ingestion.

Thermal sensation (TS) was measured with a modified ASHRAE visual analogue scale, previously validated for use in physiological applications.[11] Each participant marked the scale ranging from "very cold" to "very hot" (0–200 mm) immediately before and after each break when interventions were applied, and again at the end of the following simulated game. The rating of perceived exertion (RPE) was measured using a modified Borg scale[12] with standard increments of 6 to 20, but with participants instructed to answer the specific question: "How easy is it to continue this exercise?" Values were measured before and after each break.

In a preliminary trial, participants completed a Physical Activity Readiness Questionnaire (PAR-Q) and an American Heart Association Screening Questionnaire. Their body mass, height, and VO_{2peak} were measured. Additionally, mean VO_2 over the course of five simulated games of tennis match-play [see *Experimental Protocol*] was assessed to ensure that VO_2 (26.7 ± 1.7 ml/kg/min), as well as relative intensity (~52% VO_{2peak}), during the experimental protocol were comparable to tennis match-play on the same surface as the Australian Open (VO_2: 27.5 ml/kg/min[13]; %VO_{2peak}: ~55%[14,15]). VO_{2peak} and mean VO_2 were measured using a metabolic cart (COSMED, Quark CPET, Italy) on a treadmill (h/p cosmos Saturn 300/125, Germany). VO_{2peak} was assessed following the protocol of the Canadian Society of Exercise Physiology.[16] In addition, body fat

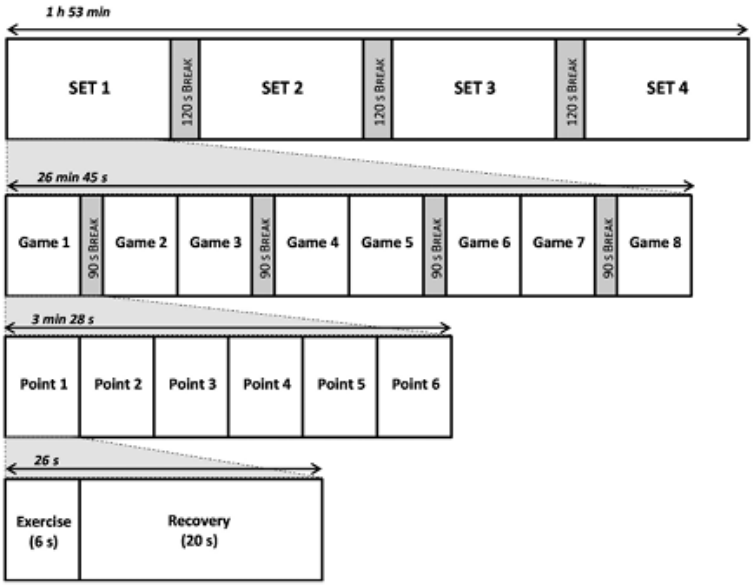

Fig. 1. Schematic illustrating the breakdown of the protocol to simulate four sets of tennis match-play. Each set consisted of eight games, with a 120-s break between sets and a 90-s break upon the completion of each odd-numbered game. Each game consisted of six identical points comprised of 6 s of high intensity exercise and 20 s of recovery.

percentage was measured with Dual X-ray Absorptiometry (DXA) on a separate visit to the laboratory (Prodigy, GE, Melbourne).

Participants also completed four experimental sessions in a counterbalanced order. In all sessions exercise was performed in a climate chamber regulated to simulate the peak environmental conditions reported at the 2014 Australian Tennis Open: 45 ± 1 °C, 9 ± 2% RH, 29.4 ± 0.6 °C Wet Bulb Globe Temperature (WBGT). In addition, two solar lamps (1000-W metal halide CSI lamps manufactured by GE Lighting), produced light with a spectrum similar to that of sunlight. These lamps, which have been used widely across several industries for this purpose,[17,18] were mounted side-by-side, at a 45° angle, 1.2 m from the participant, at a height of 2.3 m and a mean radiant load of 475 W/m^2.

Upon arrival, urine specific gravity (USG) was assessed using a refractometer (ATAGO, Japan) to ensure euhydration, i.e. USG <1.020.[19] Following instrumentation and a 30-min baseline period in thermoneutral (~21 °C, ~40% RH) conditions, HR, body mass, T_{re} and T_{sk} were measured. Participants then entered the climate chamber for 5-min of baseline rest. The exercise protocol, which was designed to simulate the temporal profile and volume of metabolic heat production of a professional tennis match, including the exact timing duration of rest breaks mandated by the Rules of Tennis, commenced (Fig. 1). The protocol consisted of a series of 16 km/h sprints lasting an average of 6 s (based on an exercise:recovery ratio that would elicit a mean VO_2 between 23–29 ml/kg/min[9]). This was followed by periods of recovery of 20 s.[9,14] One 26-s exercise:recovery cycle was recognized as 1 simulated "point". Each simulated "game" consisted of 6 "points"; and each simulated "set" consisted of 8 "games". Each session comprised of 4 simulated "sets" of a total time of 113 min (Fig. 1). Each mandated break of 90 s between odd games and 120 s between sets were implemented in accordance with ITF rules with participants allowed a further 15 s to prepare for the start of the next game. Throughout, three 46-cm diameter fans stacked vertically were placed 1.25 m in front of the participant producing an air velocity of 0.2 m/s. During each break, participants sat on a plastic chair (sheltered from radiant load), and applied one of four cooling interventions, repeated throughout each trial:

The Ice towel trial *(ICE)* was conducted in accordance with existing recommendations from the ITF Sport Science Medicine Commission (personal communication: Ms. Elaine Brady, WTA): Participants placed a wet towel (100% cotton) filled with crushed ice (2.5 kg) around their neck and placed damp towels on their lap and over their head for the duration of each break. All towels were kept in a freezer until application.

For the Fan-only trial *(FAN_{dry})*, a 45-cm diameter high-speed fan (Moretti, China) at 6.4 m/s was placed 1.0 m from the seated participant for the duration of each break.

The Fan with skin wetting trial *(FAN_{wet})* required participants, while sitting in front of the same fan, to wring out a damp sponge soaked in 15 °C water with both hands for 2 s over a bucket before applying to the skin surface of their head, neck, arms and thighs so no excess water dripped off the body.

For the Control trial *(CON)*, participants sat with no external cooling method applied, but were allowed to ingest 10 °C water (see below).

Throughout each break in their first trial, participants could consume cold water (10 °C) *ad libitum*. The total amount consumed within each subsequent trial was then fixed to minimise any confounding influence of differential cooling of abdominal thermoreceptors[20] and consequent modulations in sweat losses.

All data are expressed as means with standard deviation (±SD). A two-way repeated measures ANOVA employing the independent variables of intervention (4 levels: ICE, FAN_{dry}, FAN_{wet} and CON) and set (5 levels: rest, end of game 8 of set 1, 2, 3 and 4) was used to assess T_{re} and T_{sk}. Thermal sensation, HR and RPE were analysed using a three-way repeated measures ANOVA employing the independent variables of intervention, set, and time (3 [HR, TS] or 2 [RPE] levels: start break [pre], end break [post], and end of following game [next]). WBSR was analysed using a one-way repeated measures ANOVA with the independent variable of intervention. Post-hoc comparisons were performed using paired-sample t-tests while maintaining a fixed probability (5%) of making a type 1 error with a Holm–Bonferroni correction. All analyses were performed using SPSS 21.0 for Windows.

3. Results

Pre-exercise T_{re} in CON (36.7 ± 0.3 °C), ICE (36.7 ± 0.2 °C), FAN_{dry} (36.7 ± 0.3 °C) and FAN_{wet} (36.7 ± 0.3 °C) were similar ($p = 0.67$). The rise in T_{re} with exercise time was different depending on the cooling intervention used ($p < 0.001$) (Fig. 2a). By the end of set 3, T_{re} was lower in ICE (38.2 ± 0.3 °C) compared to both FAN_{dry} (38.7 ± 0.5 °C; $p = 0.02$) and CON (38.5 ± 0.5 °C; $p = 0.05$), while T_{re} in FAN_{wet} (38.2 ± 0.3 °C) was lower compared to FAN_{dry} ($p < 0.05$). By the end of set 4, T_{re} was lower ($p < 0.05$) in ICE (38.1 ± 0.3 °C) and FAN_{wet} (38.2 ± 0.4 °C) compared to both FAN_{dry} (38.9 ± 0.7 °C) and CON (38.8 ± 0.5 °C).

Values for T_{sk} differed between cooling interventions as simulated playing time progressed ($p < 0.001$) (Fig. 2b). By the end of set 1, T_{sk} in ICE was lower than all other trials ($p < 0.001$) and remained lower for the duration of exercise. Similarly, T_{sk} was lower in FAN_{wet} compared to FAN_{dry} (set 1, 2 and 4: $p < 0.05$; set 3: $p = 0.06$). By the end of set 4 T_{sk} values were lower in CON compared to FAN_{dry} ($p = 0.04$). By the end of set 4, T_{sk} was 35.3 ± 0.8 °C in ICE, 36.6 ± 1.1 °C in FAN_{wet}, 37.6 ± 1.0 °C in CON, and 38.1 ± 1.3 °C in FAN_{dry}.

Irrespective of set played, a significant interaction was observed between intervention and time ($p = 0.04$), with TS in ICE and FAN_{wet} being lower at the end of the break compared to before the break, and then returning to pre-break values by the end of the set that immediately followed the break (Fig. 3a). Nonetheless, TS remained cooler in both ICE and FAN_{wet} relative to CON and FAN_{dry} throughout ($p < 0.05$). No differences in TS at the start, end, or after the break were observed between the FAN_{wet} and ICE trials.

Within each break, HR decreased significantly with the application of each intervention ($p < 0.001$), but returned to pre-break values by the end of the following game (Fig. 3b). However, the decrease in HR was greater in ICE and FAN_{wet} relative to both CON and FAN_{dry} ($p < 0.05$). These differences in HR between interventions became more pronounced as the simulated match progressed ($p = 0.02$).

WBSR was 1.39 ± 0.15 L/h in CON, 1.29 ± 0.15 L/h in ICE, 1.30 ± 0.11 L/h in FAN_{wet}, and 1.45 ± 0.12 L/h in FAN_{dry}. WBSR was different depending on cooling intervention ($p = 0.01$) with a greater WBSR in FAN_{dry} compared to FAN_{wet} ($p = 0.01$) and ICE ($p < 0.001$).

Throughout each simulated match, RPE was different depending on the intervention used ($p < 0.05$) with the lowest ratings in ICE and FAN_{wet}, and highest in CON (Fig. 3c). Irrespective of the intervention, RPE progressively increased with each set ($p < 0.001$). Similarly, RPE was always lower at the end of the break compared to before each break ($p < 0.01$), but this decline in RPE was not altered by intervention ($p = 0.13$).

4. Discussion

This is the first study to assess the influence of different cooling interventions in a hot/dry environment in a tennis-specific setting analogous to the Australian Tennis Open, including a simulated solar heat load.[18] The FAN_{wet} intervention was more effective at

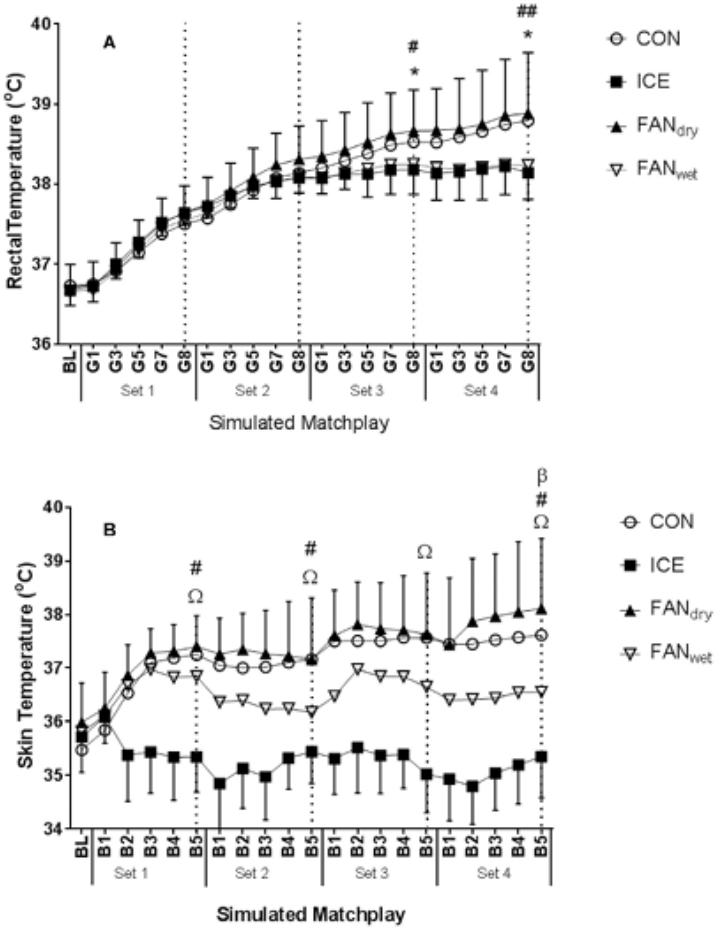

Fig. 2. Rectal temperature (A) and mean skin temperature (B) during simulated tennis match-play in the CON, ICE, FAN_{dry}, and FAN_{wet} trials. BL = baseline, G = game, B = break in play, * = ICE < CON and FAN_{dry}; # = FAN_{wet} < FAN_{dry}; ## = FAN_{wet} < FAN_{dry} and CON; Ω = ICE < CON, FAN_{dry} and FAN_{wet}; β = CON < FAN_{dry}. Statistical comparisons made at dotted lines.

mitigating thermal strain than CON and FAN_{dry}. By the end of set 4, T_{re} was ~0.5–0.6 °C lower (Fig. 2a), and T_{sk} was ~1.0–1.5 °C lower (Fig. 2b) in the FAN_{wet} trial, while TS was cooler throughout simulated match-play. Moreover, the ICE intervention was comparable to the FAN_{wet} trial with similar T_{re} and TS responses, but T_{sk} was ~1.3 °C lower than the FAN_{wet} trial. Collectively, these observations suggest that using either an ICE or FAN_{wet} intervention would help elicit a cooler TS and reduce thermal strain during tennis match-play in very hot/dry and sunny conditions.

The FAN_{dry} trial did not appear to reduce thermal strain or lead to a cooler TS. Instead, lower physiological and perceptual strain was only observed when additional moisture was applied to the skin at the start of the break. By increasing air movement across the skin surface, fan use can improve sweating efficiency, i.e. the proportion of sweat secreted onto the skin surface that evaporates and thus contributes to latent heat dissipation.[21] However, because the environmental conditions were dry, sweating efficiency was likely ~100% (i.e. all sweat evaporated) even without additional airflow. Indeed, anecdotally, no sweat dripping/pooling was observed in the CON trial. Nevertheless, because ambient temperature (45 °C) exceeded T_{sk} (~36–38 °C; Fig. 2b) a greater air velocity across the skin increased dry heat gain through convection without any concomitant elevation in evaporative efficiency; this notion is supported by the higher WBSR in the FAN_{dry} trial compared to FAN_{wet}. In contrast, when additional moisture was applied to the skin (FAN_{wet} trial), greater rates of evaporation would have been achieved with fan use as skin wettedness, at least temporarily, would have been greater than that possible with physiological sweating alone.[22]

The present findings indicate that in hot/dry conditions similar to the peak conditions reported at the Australian Open, utilizing either the FAN_{wet} or ICE cooling intervention is an effective way to reduce thermal strain and elicit a cooler TS in a tennis-specific setting. Based on fundamental heat balance laws, mechanisms that maximise potential evaporative heat loss through a greater skin wettedness (i.e. total body surface moisture coverage) and evaporative heat transfer coefficient, are as efficacious as the current recommendations for in-play cooling. A potential advantage of the

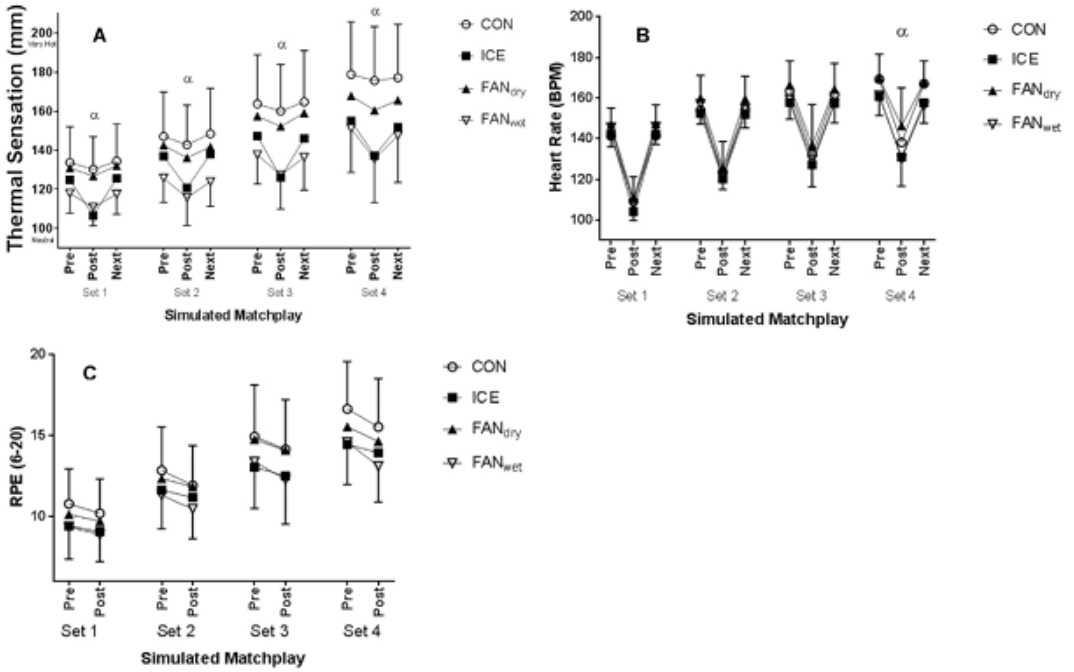

Fig. 3. Thermal sensation (A), heart rate (B), and rating of perceived exertion (C) during simulated tennis match-play in the CON, ICE, FAN_{dry}, and FAN_{wet} trials. Values were recorded before the cooling intervention was applied (Pre), immediately after the cooling intervention (Post), and at the end of the next game (Next). α = FAN_{wet} and ICE < FAN_{dry} and CON.

proposed FAN_{wet} intervention is the ease of preparation, which may be particularly relevant to sub-elite levels of tennis competition. As the FAN_{wet} trial does not require a freezer, it may present a more convenient option for athletes across levels without the required resources to prepare ice for on-court use. In addition, athletes must also consider the environmental conditions during competition. In warm and humid conditions, such as those experienced at the US Tennis Open, the dynamics of skin surface heat transfer will differ. Specifically, sweating efficiency is lower, meaning a smaller percentage of secreted sweat evaporates,[23] and as air temperature is also lower, dry heat gain would not be as great with fan use, thus supplemental skin wetting might not be necessary as maximal skin wettedness may have already been achieved physiologically.

A potential limitation in the present study is the use of treadmill running as opposed to tennis specific activities. However, as the intermittent activity profile of the sport was replicated and the metabolic heat production elicited by the exercise protocol was the same as previously reported during live tennis match-play,[13] the level of heat strain would have been comparable. In addition, conducting the study in a climate-controlled environment with simulated solar radiation, as opposed to outdoors, enabled us to precisely assess the independent influence of the tested cooling interventions. While TS was assessed during the present study, thermal comfort was not. However, as thermal comfort in hot environments typically improves with a cooler skin,[24] which was observed throughout exercise in both the FAN_{wet} and ICE trials, an improved comfort would have likely been observed compared to the CON and FAN_{dry} trials. Finally, the current study was not a performance trial as the primary aim was to assess the influence of four cooling interventions on thermal strain and sensation. As a result, it remains to be determined whether the FAN_{wet} and ICE interventions lead to an improved tennis performance. However, the benefit of skin cooling in the heat on the performance of differing intermittent running protocols have been shown in several previous studies.[25–28]

5. Conclusion

In a hot/dry environment, cooling interventions applied during the short-mandated breaks in tennis match-play activity can significantly reduce thermal strain. In comparison to 10 °C water ingestion only without any additional cooling (CON) or the use of a fan alone (FAN_{dry}), fan use with additional moisture applied to the skin (FAN_{wet}) or the application of iced towels (ICE), mitigate the development of heat strain.

Practical implications

- In hot, dry and sunny conditions tennis players can cool themselves effectively during mandated in-play breaks using either "ice towels" (an ice-filled damp towel around the neck and placing two cold-damp towels on head and thighs), or by dampening the thighs, neck and arms and sitting in front of a fan.
- Drinking cold water alone or sitting in front of a fan without supplemental skin wetting does not provide optimal cooling as low ambient humidity results in the immediate evaporation of sweat even without extra airflow across the skin.
- This study provides evidence that the currently recommended "ice towel" strategy should be advocated in Australian Open con-

ditions, and provides a practical alternative, which may be more convenient in lower resource environments.

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Appendix G: Second Article Published in Journal of Science and Medicine in Sport During PhD Candidature

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Original research

Brief in-play cooling breaks reduce thermal strain during football in hot conditions

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ABSTRACT

Objectives: The study examined if three feasible strategies involving additional in-play cooling periods attenuate the core (rectal) temperature rise during simulated football matches.
Design: Four counterbalanced experimental trials in an environmental chamber set to 35 °C ambient temperature, 55% relative humidity, and 30 °C WBGT.
Methods: Twelve healthy well-trained football players completed a regular simulated match (REG), regular simulated match with additional 3-min cooling periods at the 30-min mark of each half inclusive of chilled water consumption ($COOL_{water}$), regular simulated match with additional 3-min cooling periods at the 30-min mark of each half inclusive of chilled water consumption and the application of an ice towel around the neck ($COOL_{towel}$), regular simulated match with an extended (+5 min; total of 20-min) half-time break ($HT_{extended}$).
Results: The difference in rectal temperature change was significantly lower in the $COOL_{water}$ (−0.25 °C), $COOL_{towel}$ (−0.28 °C), and $HT_{extended}$ (−0.21 °C) trials in comparison to the REG (all $p < 0.05$). Exercising heart rate and session rating of perceived exertion was lower in the $COOL_{water}$ (−13 bpm; −1.4 au), $COOL_{towel}$ (−10 bpm; −1.3 au), and $HT_{extended}$ (−8 bpm; −0.9 au) trials in comparison to the REG trial (all $p < 0.05$). The cooling interventions did not significantly change skin temperature or thermal sensation in comparison to the REG (all $p > 0.05$).
Conclusions: All three cooling interventions attenuated core body thermal strain during simulated matches. The laboratory-based study supports the use of brief in-play cooling periods as a means to attenuate the rise in core temperature during matches in hot and humid conditions.

Practical implications

- The efficacy of currently advocated brief in-play cooling breaks are supported by this laboratory-based study.
- The $COOL_{water}$ and $HT_{extended}$ strategies appear more feasible for all levels of play than $COOL_{towel}$ whilst providing similar cooling benefits.
- The $HT_{extended}$ strategy may be preferred by organisations that do not desire breaks during each half of play and allows further time for additional and more extensive cooling strategies during half-time.

1. Introduction

Numerous worldwide football competitions (A-League [Australia] and Major League Soccer [United States]) and tournaments (2014 World Cup in Brazil) occur in stressful thermal conditions. Hot and humid environments influence the cardiovascular, cardiorespiratory, metabolic, perceptual, and fatigue response to exercise,[1] and therefore have a modulating effect upon football players physical output during matches.[2–4] Furthermore, there is an additional thermal burden onto the heat stress already induced by intense exercise, predominantly through

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the impediment of sweat evaporation, subsequently leading to lower skin surface heat dissipation and thus greater internal heat storage.[5] Significant rises in internal heat storage increase the risk of heat-related health problems,[6,7] which are preventable and encompass a range of conditions.[5,8] The potentially fatal nature of heat-related illness[9] warrants precaution and additional safety measures should mitigate the risk of exertional heat illness during competition in environmentally stressful conditions.

Core body temperature is the primary indicator of human thermal strain. Mean core temperature values above 39 °C have been observed during football matches in hot[6,10] and temperate[11,12] conditions. Notably, individual peak values over 40 °C have been reported,[6,10,13] which is associated with a substantial increase in the risk of heat stroke.[14] A feasible strategy for all levels of participation (amateur to professional) to potentially reduce the rise of core body temperature during matches is brief in-play cooling breaks.[15] Indeed, a review by Grantham et al.[5] recommended additional 'fluid' breaks if the wet-bulb globe temperature (WBGT) exceeds 30 °C during football competition. In 2014, the Fédération Internationale de Football Association (FIFA) sanctioned additional 3-min cooling periods for the World Cup competition at the 30-min mark of each half during matches that were played in conditions of >32 °C WBGT.[16] Brief cooling periods are also implemented in the A-League (Australia) and Major League Soccer (United States) competitions when environmental conditions exceed 26 °C and 28 °C WBGT, respectively. Additional breaks during play provide an opportunity to restrain the exercise-induced rise in core temperature and facilitate additional fluid intake. Additional cold water ingestion may be an effective strategy to facilitate cooling of an athlete during exercise in hot, humid, and still environments.[17] This relates to the high efficiency of internal heat loss in the face of restrictions that the prevailing conditions place upon heat loss via sweat efficiency.[17] Previous studies have examined the use of simple cooling strategies during simulated[18–20] and field[10] football matchplay, but were applied only during the pre-match and half time periods.[10,18,19] No evidence is available that examines the effectiveness of in-play (i.e. during each half of play) cooling periods, yet they are already currently advocated by FIFA. Therefore, the study will inform the extreme heat policy guidelines for football governing bodies by testing the efficacy of in-play cooling breaks within a controlled environment.

Since the regular cancellation of matches in hot and humid conditions is not feasible, cooling strategies need to be devised that are pragmatic for not only professionals, but community-based amateur competitions that are vital for public health.[15] The aim of the study was to examine if three feasible interventions (trials) involving additional cooling periods attenuate the core (rectal) temperature rise during simulated football matches in comparison to a regular match (REG trial). The three strategies were inclusive of; 1) two additional 3-min cooling periods with chilled water ingestion (as advocated by FIFA; $COOL_{water}$ trial), 2) two additional 3-min cooling periods with chilled water ingestion and an ice towel placed around the neck ($COOL_{towel}$ trial), 3) an additional 5-min cooling break at the end of half time ($HT_{extended}$ trial). We hypothesised that all three strategies would result in an attenuation of the core temperature rise in comparison to REG.

2. Methods

Twelve healthy well-trained football players were recruited, provided written informed consent to participate, and completed the study. The mean age, height, body mass, and maximal oxygen consumption was 24 ± 7 y, 179 ± 5 cm, 75 ± 6 kg, and 58 ± 3 mL/kg/min, respectively. Additionally, five other participants withdrew from the research due to illness and injuries sustained during football participation external to the study. The study had institutional ethics approval.

Participants completed one preliminary and four experimental sessions, separated by at least 48-h. Maximal oxygen consumption was assessed on a motorized treadmill[21] using a metabolic cart (Quark CPET, Cosmed, Italy) during the preliminary trial. All experimental trials were completed in an environmental chamber set to 35 °C ambient temperature, 55% relative humidity, and 30 °C WBGT (according to indoor formula for WBGT[22]). Participants refrained from strenuous exercise and alcohol 24-h prior to experimental trials. Caffeine was prohibited 8-h prior to trials and participants consumed a cold 600 mL sports drink (Gatorade, Australia) two hours prior to attending the laboratory. Participants completed the REG trial first and then were assigned using blocked randomization into the $COOL_{water}$, $COOL_{towel}$, and $HT_{extended}$ trials using a counterbalanced approach across possible trial order combinations.

The simulation protocol for the REG trial is outlined in Fig. 1 (total duration: 106-min). The REG trial was completed first to quantify *ad libitum* consumption of chilled water (10 °C) during the half-time break so that fluid intake could be subsequently matched during the half-time periods of the intervention trials. Participants remained seated in the chamber outside of the direct flow of the fan during the half-time period.

The $COOL_{water}$ and $COOL_{towel}$ trials included the same protocol as the REG but with the addition of 3-min cooling periods at the 30-min mark of each half (total duration: 112-min). The 3-min cooling periods during the $COOL_{water}$ trial involved the administering of an additional 3.2 mL/kg of body mass of chilled water (10 °C) to participants whilst they remained standing outside of the direct flow of the fan. The total volume of water and time requirement for consumption is similar to previous studies examining endurance performance in the heat.[23] The $COOL_{towel}$ trial involved participants consuming chilled water (10 °C; 3.2 mL/kg) and draping an ice towel around the neck and shoulders for 2.5-min during each 3-min cooling period. The ice towel (100% cotton) was soaked with cold water, wrung, and filled evenly with 2.5 kg of ice, consistent with similar studies in tennis players.[24,25]

The $HT_{extended}$ trial included the same simulation protocol as the REG trial but with an extended half time period (20-min instead of 15-min; total duration: 111-min). No extra fluid was provided during the half time break in addition to the matched quantity for all other half time periods.

The pre-programmed treadmill (Pulsar 3p, h/p/cosmos Sports & Medical gmbh, Germany) protocol followed similar principles to previous motorized treadmill simulations[26] but was adjusted in consideration of the environmental conditions and pilot testing (Fig. 1). Participants wore football shorts, shin guards, socks, running shoes, and a standardized jersey (100% polyester, Nike, Thailand) during match simulations. A Kestrel 4600 heat stress tracker monitored environmental conditions and was placed next to the treadmill, in line with both the position of the participant and airflow generated by an electric fan. Ambient temperature (n = 12), relative humidity (n = 12), and WBGT (n = 8 due to technical issues) were recorded at the start and end of each half of play. Urine specific gravity (USG) (Atago® refractometer, Japan) was measured at the beginning of all experimental trials to estimate hydration status. Change in body mass (±0.2 g; Mettler, Germany) over the duration of the simulation was used to calculate whole body sweat loss (g/h), corrected for weight gain through fluid ingestion. Rectal temperature (T_{rec}) was continuously monitored following the self-insertion of a temperature probe (Covidien™ Mon-a-Therm™ 400TM, USA) 15-cm past the anal sphincter. Exercising HR was continuously monitored (Polar RS400, Finland). Regional skin temperature was continuously measured wirelessly by sensors (iButton® DS1922L, Maxim Integrated, USA) placed on the chest, arm, mid-thigh, and calf to determine mean skin temperature.[27] Thermal sensation was

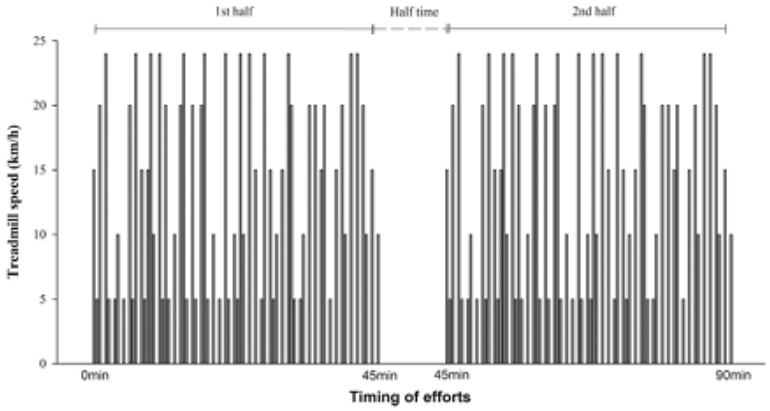

Fig. 1. A schematic of the treadmill protocol for the REG trial. All trials followed an identical exercise protocol, with the addition of either in-play cooling breaks (+3 min each) at the 30-min mark of each half ($COOL_{water}$ and $COOL_{towel}$) or an extended half time break (+5-min: $HT_{extended}$).
The total distance of the treadmill protocol was 10.8 km and the duration of each half for the REG trial was 45-min and 15 s, separated by a 15-min half-time break. The prescribed bout duration and percentage of total distance that participants remained at each designated speed was: 0 km/h (17 s), 5 km/h (40 s: 31.7%), 10 km/h (30 s: 27.6%), 15 km/h (22 s: 24.8%), 20 km/h (4 s: 8.0%), and 24 km/h (3 s: 7.8%). Treadmill acceleration (level 3 ["moderate"]: 0.34 m/s²) and deceleration (level 7 ["very fast"]: −0.68 m/s²) times were additional to the duration of the listed times. No warm-up was completed prior to the start of the simulation. The REG protocol elicited a mean exercising percentage of maximum heart rate (HR) of 73.7 ± 8.2% during the first half and 83.2 ± 6.1% during the second half of play.

measured every 5-min using a 200-mm modified ASHRAE visual analogue scale ranging from "very cold" (0 mm), through "neutral" (100 mm), to "very hot" (200 mm). An electric fan was placed four metres away from the exercising participant and operated to elicit a wind speed of approximately 1 m/s. Post-exercise session rating of perceived exertion (sRPE) was measured 15-min after the cessation of exercise.[28]

Data are expressed as means and 95% confidence intervals. One-way repeated measures ANOVA tests with a factor of 'session' (4 levels: REG, $COOL_{water}$, $COOL_{towel}$, and $HT_{extended}$) were used to analyse resting USG and sRPE. The change in exercising thermal (T_{rec} [end value] and skin temperature [mean of final 5-min]) and perceptual strain (thermal sensation [end value]) indicators were analysed at the end of each 15-min interval between the 30–90-min mark of each simulation. The first 30-min of each trial was identical and classified as a control period. Therefore, the 30-min mark was treated as a 'baseline' (i.e. '0' [no change]). Cardiovascular strain (HR) was analysed over the same time period (30–90-min), but using absolute values rather than change values and using the mean value of each 15-min interval. All variables displayed in figures are shown in absolute values across the entirety of the match to improve contextualization. Two-way repeated measures ANOVA with a factor of 'time' (5 levels: 45-min, end of half-time, 60-min, 75-min, and 90-min) and 'session' (4 levels: REG, $COOL_{water}$, $COOL_{towel}$, and $HT_{extended}$) were used to examine the dependent variables of T_{rec}, skin temperature, HR, and thermal sensation. In the event of a significant main effect, post-hoc analyses were performed using paired-samples t-tests. The probability of making a type I error in all tests was maintained at 5%. All analyses and figures were performed and created in SPSS version 24 (Chicago, USA), Sigmaplot 13 (San Jose, USA), and GraphPad Prism 7.0 (La Jolla, USA).

3. Results

Resting USG was similar among the REG (1.011 [1.007–1.015]), $COOL_{water}$ (1.013 [1.007–1.019]), $COOL_{towel}$ (1.014 [1.008–1.019]), and $HT_{extended}$ (1.009 [1.004–1.013]) trials ($p = 0.308$). The mean trial environmental conditions were 35.7 (35.6–35.8)°C ambient temperature, 52.5 (52–53.1)% relative humidity, and 30.1 (30.1–30.2)°C WBGT.

The mean difference in T_{rec} change between rest and the 30-min mark for each trial was similar ($p = 0.377$; Fig. 2). There was a main effect of session between the 30 and 90-min match period (per 15-min interval), with the difference in exercising T_{rec} change significantly lower in the $COOL_{water}$ (−0.25 [−0.40 to −0.10]°C; $p = 0.004$), $COOL_{towel}$ (−0.28 [−0.39 to −0.17]°C; $p < 0.001$), and $HT_{extended}$ (−0.21 [−0.28 to −0.14]°C; $p < 0.001$) in comparison to REG. The difference in exercising T_{rec} change was similar between all cooling intervention trials (all $p > 0.05$). There was a significant session by time interaction ($p = 0.002$), with Fig. 2 demonstrating that a smaller change in T_{rec} (i.e. remained cooler) was evident during all 15-min intervals between the 30–90-min period for the $COOL_{water}$ and $COOL_{towel}$ trials (all $p < 0.05$). The smaller change in T_{rec} was evident from half-time onwards during the $HT_{extended}$ session (all $p < 0.05$). Supplementary material 1 demonstrates the individual total change in T_{rec} during the 30–90-min period of each cooling intervention in comparison to REG.

Fig. 3A demonstrates the mean absolute HR during all trials. There was a main effect of session, with the $COOL_{water}$ (−13 [−19 to −8]bpm; $p < 0.001$), $COOL_{towel}$ (−10 [−17 to −3]bpm; $p = 0.010$), and $HT_{extended}$ (−8 [−13 to −2]bpm; $p = 0.011$) trials associated with a lower mean exercising HR in comparison to REG. Exercising HR was also lower during the $COOL_{water}$ (−5 [−10 to −1]bpm; $p = 0.016$) trial in comparison to $HT_{extended}$ (Fig. 3A). Fig. 3B illustrates the mean skin temperature during all trials. The difference in mean skin temperature change during the 30–90-min period was similar between all sessions (main effects $p = 0.357$). There was a main effect of session, but the difference in the change of thermal sensation during the 30–90-min period in comparison to the REG trial was similar during the $COOL_{water}$ (−9 [−20 to 3]mm [cooler]; $p = 0.128$), $COOL_{towel}$ (−2 [−11 to 6]mm [cooler]; $p = 0.582$), and $HT_{extended}$ (+3.2 [−6 to 13]mm [warmer]; $p = 0.466$) trials. However, the difference in the change of thermal sensation was lower (i.e. remained cooler) during the $COOL_{water}$ (−12 [−21 to −3]mm; $p = 0.011$) and $COOL_{towel}$ (−5 [−11 to 0]mm; $p = 0.042$) trials in comparison to $HT_{extended}$.

Whole body sweat rate during the simulated matches was similar between the REG (1200 [1085–1315]g/h), $COOL_{water}$ (1208

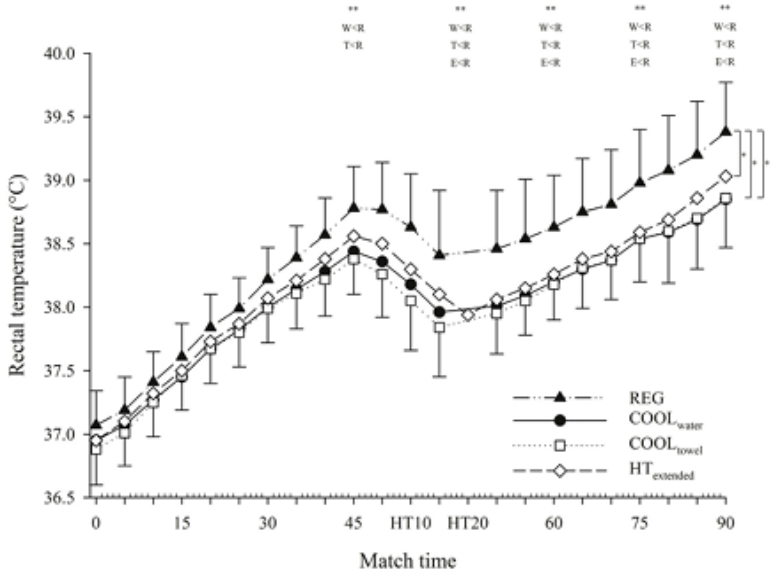

Fig. 2. Mean absolute rectal temperature throughout the four experimental trials. Data are presented as means and standard deviations (for the low and high trials).
*Significant ($p < 0.05$) session difference in comparison to REG for the mean rectal temperature change during the 30–90-min time period. **Significant ($p < 0.05$) difference in comparison to REG trial for specific 15-min time intervals; *R* regular game; *W* $COOL_{water}$ trial; *T* $COOL_{towel}$ trial; *E* $HT_{extended}$ trial.

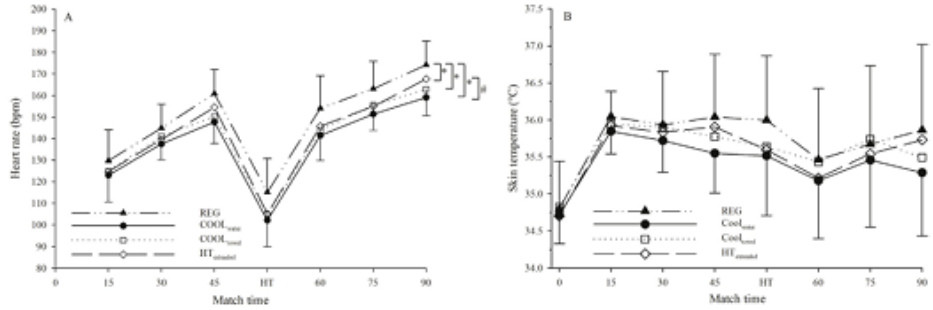

Fig. 3. The mean heart rate (A) and mean skin temperature (B) response to the four experimental trials. Data are presented as means and standard deviations (for the low and high trials).
*Significant ($p < 0.05$) session difference in comparison to REG for the mean heart rate during the 30–90-min time period. #Significant ($p < 0.05$) session difference in comparison to $HT_{extended}$ for the mean heart rate during the 30–90-min time period.

[1110–1305]g/h), $COOL_{towel}$ (1185 [1088–1282]g/h), and $HT_{extended}$ (1173 [1059–1287]g/h) ($p = 0.622$) trials. The mean sRPE for the four trials was; REG (6.4 [5.2–7.7]), $COOL_{water}$ (5.0 [3.8–6.3]), $COOL_{towel}$ (5.1 [4.0–6.2]), and $HT_{extended}$ (5.5 [4.4–6.6]). There was a main effect of session, with sRPE significantly lower in the $COOL_{water}$ (−1.4 [−2.3 to −0.5]; $p = 0.006$), $COOL_{towel}$ (−1.3 [−2.3 to −0.3]; $p = 0.013$), and $HT_{extended}$ (−0.9 [−1.8 to −0.1]; $p = 0.038$) trials in comparison to REG. Session RPE was also lower during the $COOL_{towel}$ (−0.4 [−0.7 to 0.0]; $p = 0.043$) trial in comparison to $HT_{extended}$.

4. Discussion

In support of our hypothesis, the $COOL_{water}$, $COOL_{towel}$, and $HT_{extended}$ trials attenuated thermal (T_{rec}) strain in comparison to a REG trial. Neither intervention resulted in a significantly greater cooling effect upon core body temperature than the others. The brief cooling interventions also reduced markers of exercise intensity (HR and sRPE) but did not significantly influence mean skin temperature or thermal sensation.

This is the first study to examine the effect of advocated brief in-play cooling periods during a simulated football match. The cooling interventions are considered feasible in a low-cost community setting, especially the $COOL_{water}$ and $HT_{extended}$ strategies. In a field setting, it may be necessary for players to seek shade during breaks to minimise potential heat gain from additional overall solar radiation exposure time (due to the prolonged total match time). A true control trial in comparison to the $COOL_{water}$ and $COOL_{towel}$ trials was not included. A true control trial would require players to complete a match with either no additional fluid ingestion during the 3-min breaks, or alternatively, players consume identical fluid intake at the same intervals during the 3-min break and

REG trials. However, these options are not practical or possible in a field setting, since most players will consume fluid during dedicated break periods (i.e. "drinks breaks"), and secondly, it is impossible to provide additional fluid to a team throughout a match without dedicated rest periods.

The T_{rec} response was attenuated following the first cooling period (30-min mark) of the first half for the $COOL_{water}$ (mean of −0.25 °C) and $COOL_{towel}$ (mean of −0.28 °C) trials. The implementation of cooling periods following 30-min of match-play is recommended by FIFA due to the finding that peak player core temperature in hot field matches occurs at the 30-min time point.[6] The T_{rec} response was attenuated at the end of half-time and throughout the remainder of the match for the $HT_{extended}$ trial (mean of −0.21 °C). A potential advantage of the $HT_{extended}$ trial is the additional time that the strategy affords for other extensive cooling practices (that might be logistically difficult during the 3-min breaks) during the major rest period that can reduce the change in core temperature during the second half of play.[18] However, not all half-time cooling strategies substantially influence core temperature during the second half of football,[19] therefore, potentially highlighting the importance of the 3-min cooling period during the second half of play for attenuating thermal strain in the latter stages of matches. Notably, the attenuation of absolute exercising core body temperature during the cooling intervention trials is similar to the magnitude commonly conferred through adaptations from a heat acclimation regime.[29] Intuitively, an aggregate effect between in-play cooling strategies and acclimation also likely exists.

Additional chilled water ingestion was provided during the $COOL_{water}$ and $COOL_{towel}$ trials (during the 3-min in-play breaks) whereas participants consumed the same quantity of fluid during the REG and $HT_{extended}$ trials. The lack of substantial difference in core body temperature cooling between all three cooling intervention trials may suggest that the additional chilled fluid intake during the $COOL_{water}$ and $COOL_{towel}$ trials had a trivial thermoregulatory effect. Cold fluid consumption during exercise may potentially lower evaporative drive due to the cooling effect from the internal heat transfer, with a subsequent potential to limit a net body cooling effect.[17] Overall, the observed attenuation of T_{rec} may relate more to the additional cessation in exercise and restraining of the exercise-induced rise in core temperature rather than the modest additional hydration per se, which on average mitigated trial percent total body mass losses by 0.5 ± 0.2% (2.2% vs 1.7%).

Cardiovascular (HR) and perceived physiological strain (sRPE) were substantially lower during all intervention trials in comparison to the REG trial. However, there was a lack of difference between all four experimental trials in the mean skin temperature change, and moreover, a lack of substantial change in thermal sensation in comparison to the REG trial. Additional rest periods during field matches may encourage players to upregulate their playing intensity (in comparison to the time period prior to the break) upon returning to play and the subsequent higher metabolic heat generation may offset any positive cooling benefits from the breaks.[30] However, it is uncertain if changes to activity pacing profiles will actually occur in an uncontrolled field setting based upon the inconsistent response of likely influencing contributing factors (cardiovascular strain, perceived physiological strain, and thermal sensation).

The primary limitation of the study is the controlled design of the treadmill protocol and subsequent applicability to field-based matches. The treadmill simulation isolated the effect of the cooling periods upon thermal strain, however, it does not represent the uncontrolled nature of field-based matches where player physical output is often paced. Further research should examine the translation of these laboratory-based results to uncontrolled field-based scenarios to examine the interaction between performance and thermal strain following brief in-play cooling strategies. Second, no exercise warm-up period was undertaken prior to each trial, with the resultant additional effect upon body temperature potentially influencing the response to subsequent cooling strategies.

5. Conclusion

The current laboratory-based controlled study supports the use of brief in-play cooling periods, as currently advocated by FIFA and other football organisations, as a means to attenuate the rise in core body temperature during matches in hot and humid conditions. The implementation of the cooling interventions are considered feasible in a low-cost community setting, especially the $COOL_{water}$ and $HT_{extended}$ strategies.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.jsams.2019.04.009.

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Appendix H: Third Article Published in Journal of Science and Medicine in Sport During PhD Candidature

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Original research

Optimal break structures and cooling strategies to mitigate heat stress during a Rugby League match simulation☆

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ABSTRACT

Objectives: To examine, 1) optimal structure of break periods to mitigate physiological heat strain during rugby league play (*Stage 1*); and ii) effectiveness of three different cooling strategies applied during breaks (*Stage 2*).
Design: Counter-balanced crossover design.
Methods: In 37 °C, 50% RH, 11 males completed six simulated 80-min (two 40-min halves) rugby league matches on a treadmill with different break structures: regular game (RG) (12-min halftime), 1-min or 3-min "quarter-time" breaks halfway through each half with a 12-min halftime break (R1C and R3C), a 20-min halftime break (EH), or 1-min or 3-min quarter-time breaks with a 20-min halftime break (E1C and E3C) [*Stage 1*]. Nine participants completed *Stage 2*, which assessed the application of either ice towels (ICE), an electric fan (FAN) or a misting fan (MST) during breaks in the E3C protocol which, in *Stage 1*, prevailed as the optimal break structure.
Results: Stage 1: Irrespective of quarter-time break duration, reductions in rectal temperature (-0.24 °C $\pm$ 0.24) and heart rate (-61 ± 10 bpm) during the halftime break were greater with a 20-min compared to a 12-min break (-0.08 ± 0.13 °C, $p = 0.005$; -55 ± -9 bpm, $p = 0.021$). *Stage 2*: End-game rises in rectal temperature were smaller ($p < 0.006$) in MST (1.41 $\pm$ 0.22 °C), FAN (1.55 $\pm$ 0.36 °C) and ICE (1.60 $\pm$ 0.21 °C) than in CON (1.80 $\pm$ 0.39 °C). The end-halftime heart rate was lower ($p < 0.001$) in ICE (89 $\pm$ 13 bpm), MST (90 $\pm$ 10 bpm) and FAN (92 $\pm$ 13 bpm) than in CON (99 $\pm$ 18 bpm).
Conclusions: Combining an extended halftime period and quarter-time breaks with MST application is the optimal cooling strategy for rugby league players in hot, humid conditions.

Practical implications

- Extending halftime breaks by 8 min (from 12 to 20 min) provides the most benefit in terms of mitigating thermal and cardiovascular strain in a simulated rugby league match in hot and humid conditions
- The addition of short quarter-time breaks of up to 3 min provides only marginal cooling benefit in the absence of cooling interventions. The most effective cooling intervention during breaks in play was the use of misting fans
- The findings of this study can be extended beyond rugby league to other high intensity intermittent sports with prolonged periods of play, e.g. association football, Australian football, rugby union

1. Introduction

Rugby league is a high-intensity sport consisting of two 40-min halves, which in many circumstances, such as the 2017 Rugby League World Cup and the preseason and start of the National Rugby League competition in Australia, take place in hot, humid conditions and thus presents a high risk of exertional heat illness.[2,3] Identifying strategies to mitigate heat strain in a rugby league match-play context is therefore essential for player safety. Chalmers et al.[7] demonstrated the effectiveness of brief cooling breaks combined with an active cooling intervention in mitigating thermal strain during simulated soccer match play. Previously, quarter-time breaks (1–3-min pauses halfway through each half during which players stay on the field) have been implemented in professional rugby league matches during hot weather, giving players an opportunity to hydrate. It is unclear though, if these brief breaks meaningfully off-set thermal strain as, without an aggressive cooling intervention, they may not be long enough to halt and reverse rises in core temperature. Alternatively, an extended halftime break (e.g. from 12 to 20 min), may allow greater reductions in core temperature.

☆ Given Professor Jay's role as Guest Editor, we would like to inform that he has had no involvement in the peer-review of this article and has no access to information regarding its peer-review. Full responsibility for the editorial process for this article was delegated to Associate Professor Jason Siegler.

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In other elite sports, interventions such as ice-towel application accelerate heat loss.[5,17] In hot, humid conditions, increasing airflow across the skin surface with an electric fan promotes sweat evaporation,[25,29] particularly with supplemental water applied to the skin.[21,23] This effect may be more simply achieved with a misting fan. However, such devices may unintentionally saturate the air surrounding the athlete with moisture, reducing or eradicating the water vapour pressure gradient between the skin surface and environment, thereby blunting evaporation.[25]

Combining in-play breaks with active cooling strategies may present the most effective approach to mitigate heat strain during rugby league play. Therefore, the aims of the present study were to i) determine the optimal break structure to reduce physiological heat strain during simulated rugby league match play (*Stage 1*) and ii) assess the efficacy of different practical cooling strategies [ice towels (ICE), electric fan use (FAN) and misting fan use (MST)] during the optimal break structure identified in *Stage 1* (*Stage 2*).

We hypothesized that i) an extended halftime break (from 12 to 20 min) and quarter time breaks (either 1 or 3 min) would reduce thermal strain, and combining both would augment this effect and ii) all three cooling interventions would reduce thermal strain relative to no intervention, with FAN being the most effective, and MST being the least effective.

2. Methods

Participants

The threshold for a meaningful mean difference in core temperature change between conditions was defined as 0.2 °C. Based on comparable published research[4] we determined an effect size of 1.0. Using G*Power 3 software and with *alpha* set to 0.05 and a *beta* value of 0.1, a power calculation determined a minimum sample size of 11.[11] Twelve unacclimated males were recruited, with one withdrawing before completing all six trials of *Stage* 1 and two more withdrawing after completing *Stage 1*. Accordingly, 11 participants completed *Stage 1* (age: 24 ± 4 y; height: 1.77 ± 0.07 m; body mass: 84.1 ± 5.8 kg; VO_2max: 4.5 ± 0.4 L/min) and 9 returned to complete *Stage 2* (age: 24 ± 4 y; height: 1.76 ± 0.07 m; body mass: 84.0 ± 6.4 kg; VO_2max: 4.4 ± 0.4 L/min).

Preliminary trial

Upon arrival, participants had their nude body mass measured on a platform scale (Mettler, Germany) and height measured with a wall-mounted stadiometer. Next, participants undertook a maximal oxygen consumption (VO_2max) exercise test on a treadmill,[30] during which oxygen consumption was monitored continuously via a metabolic cart (COSMED, Quark, CPET, Italy).

Instrumentation

Identical instrumentation was used for all experimental trials in *Stage 1* and *Stage 2*. Participants abstained from caffeine, exercise and alcohol in the 24 h leading up to all experimental trials and consumed ~500 mL of water and a light meal 2 h prior to the trial.

Upon arrival to the laboratory, participants provided a mid-stream urine sample to confirm euhydration (specific gravity <1.020; if >1.020, 250–300 mL of tepid water was consumed). Next, participants self-inserted a thermistor probe (TM400, Covidien, Mansfield, MA, USA) to ~15 cm beyond the anal sphincter, which was used to measure rectal temperature (T_{re}). Semi-nude (shorts only) body mass was then measured on a platform scale (Mettler, Toledo), before four wireless temperature sensors (iButtons DS1921H-F5, Embedded Data Systems, USA) were fixed via surgical tape to the chest (left, upper), shoulder (left, deltoid insertion), thigh (left, mid-anterior) and calf (left, mid-posterior). Mean skin temperature (T_{sk}) was calculated according to Ramanathan.[26] Heart rate (HR) was measured by telemetry (Polar RS400, Kempele, Finland) throughout the trial. Throughout, participants wore an authentic NRL match play jersey and shorts (ISC, Australia; 94% polyester, 6% spandex), as well as long sports socks (100% cotton) and running shoes (ensemble Icl = 0.4 Clo, Re,cl = 0.012 m^2/kPa/W).

Experimental trials

In both stages, the basic experimental protocol was identical, with differences only in break duration and distribution (*Stage 1)*, and cooling intervention (*Stage 2*). Participants commenced all trials at the same time of day (±1-h) with a comparable baseline core temperature (±0.3 °C). On each occasion, participants completed an intermittent treadmill protocol designed to replicate the duration and intensity of a professional rugby league match[15] (Supplementary Table 1). The protocol required participants to intermittently walk/run at various speeds matching those typical of a Rugby League match,[15] for two 40-min periods separated by a rested 'halftime' break (total 80 min active). The total distance covered was 7766 m (2183 m at 5 km/h, 2400 m at 10 km/h, 1583 m at 15 km/h, 933 m at 20 km/h, 667 m at 25 km/h; 860 s standing recovery). In *Stage 1*, participants completed six separate simulated matches on different days, with a different halftime break (12 or 20 min), and/or quarter-time break (1 or 3 min) (Fig. 1A). In *Stage 2*, participants completed three simulations, with timing and distribution of break periods remaining consistent (E3C protocol, i.e. 3-min quarter-time breaks, 20-min halftime; Fig. 2A), but three different cooling interventions implemented during the quarter-time and halftime breaks during which participants were seated. These interventions were:

(i) ICE: participants draped a wet towel (100% cotton) filled with crushed ice (~2.5 kg) around their neck, over both shoulders onto their chest;
(ii) FAN: an electric fan directed at the torso (Coolzone, Australia; 40 cm diameter) at 2.5 m/s; and
(iii) MST: the same high-speed fan with water spray (800 psi).

The E3C trial completed in *Stage 1* served as the control trial (CON) in *Stage 2*. A balanced, incomplete Latin square design determined trial order in *Stage 1* and for the three interventions in *Stage 2*.

Mean environmental conditions were 36.9 ± 0.4 °C, 51 ± 1% RH, reflecting a hot, humid day in the city of Cairns, Australia,[3] where part of the 2017 Rugby League World Cup was played. During the active stages of the protocol, three vertically stacked fans (0.5 m/s) were placed 1.25 m from the participant.

Rating of perceived exertion (RPE) (6–20[6]) and thermal sensation (TS) (0–200 mm[14]) were measured every 5 min throughout active stages (not including quarter-time breaks) and at the beginning, middle and end of the halftime period. During *Stage 1*, participants consumed ~20 °C water ad libitum during each break period. For *Stage 2*, participants consumed ~10 °C water. To standardize any reductions in sweat output,[22] participants drank ad libitum during break periods in their first *Stage 2* trial, and this volume was matched in subsequent trials. End-trial whole body sweat loss (WBSL) was calculated using pre and post semi-nude body masses and corrected for fluid consumption.

Statistical analysis

Stage 1: a two-way repeated measures ANOVA was employed to determine the main effects of quarter-time break length (3 levels: no break, 1-min, 3-min) and halftime break length (2 levels; 12-min or 20-min) on ΔT_{re}, change in HR (ΔHR), T_{sk}, TS and RPE across the whole-game simulation and within the 1st and 2nd half periods and during the halftime break. WBSL was compared between the six different break structures using a one-way ANOVA.

Stage 2: a two-way repeated measures ANOVA was employed to assess the main effect of intervention (4 levels: CON, FAN, ICE, MST) and time (3 levels: end-first half, end-halftime, end-game) on ΔT_{re}. A similar

analysis was employed for HR, T_{sk}, TS and RPE with the inclusion of a fourth time-point of baseline. WBSL was compared between conditions using a one-way ANOVA.

Trial-order effect was independently assessed for *Stage 1* and *Stage 2* using a one-way ANOVA.

When a significant main effect or interaction was found, post-hoc analyses were performed using two-tailed paired-sample *t*-tests while maintaining a fixed probability (5%) of making a type 1 error by employing a Bonferroni correction to adjust for multiple comparisons. The effect size of each t-test was calculated and reported as Cohen's d (d), and were interpreted as small ($d \geq 0.2 < 0.5$), medium ($d \geq 0.5 < 0.8$) or large ($d \geq 0.8$).[9] Data were assessed for normality by calculating skewness and kurtosis with values within ± 2.0 considered acceptable. Assumptions of sphericity were assessed via Mauchly's Test of Sphericity, and where violated, a Greenhouse-Geisser correction was applied. All analyses were performed using the statistical software packages SPSS (version 27.0) and GraphPad (version 8).

3. Results

All parameters were assumed normally distributed as indicated by skewness and kurtosis values within ± 2.0. Trial-order did not significantly impact results for any parameter ($p > 0.193$).

Stage 1 – Break timing and duration

Core and skin temperature

The change in T_{re} from baseline during the first half, the halftime break, and the second half was not influenced by quarter-time break existence/duration ($p > 0.081$). An extended 20-min halftime break led to a greater reduction in T_{re} (-0.24 ± 0.24 °C) during the halftime break ($p = 0.005$) than a 12-min halftime break (-0.08 ± 0.13 °C) (Fig. 1B).

Although quarter-time breaks did not impact T_{sk} ($p > 0.653$), a lower T_{sk} was observed at the end of halftime ($p = 0.011$) with an extended halftime break (Table 1).

Heart rate and sweat rate

The change in HR from baseline was not influenced by quarter-time break length/duration within the first half, halftime break, or second half ($p > 0.417$). An extended 20-min halftime break led to a greater reduction in HR (-61 ± 10 bpm) during the halftime break period ($p = 0.021$) than a 12-min halftime break (-55 ± 9 bpm) (Fig. 1C). Whole-body sweat loss was similar irrespective of break structure ($p = 0.192$) (Table 1).

Thermal sensation and rating of perceived exertion

The addition of quarter-time breaks, or extension of the halftime break did not influence RPE at any stage throughout the simulated match ($p > 0.071$). There was a main effect of quarter-time break duration on TS at the end of halftime ($p = 0.019$) with participants feeling cooler following an extended halftime break (Table 1). The duration of halftime break also had lasting effects, with lower TS values reported at the end of the second half ($p = 0.047$) (Table 1).

Stage 2 – Cooling strategies applied during breaks

Core and skin temperature

The change in T_{re} from baseline was significantly altered by cooling intervention ($p = 0.028$) with ΔT_{re} lower in MST ($p < 0.001$, $d = 1.40$) and FAN relative to CON ($p = 0.003$, $d = 1.02$), and lower in MST ($p = 0.013$, $d = 0.95$) than ICE at the end of halftime. End-trial ΔT_{re} was lower in MST ($d = 1.31$), ICE ($d = 0.74$) and FAN ($d = 0.73$) than in CON ($p < 0.006$), while ΔT_{re} in MST was lower than ICE ($p = 0.047$, $d = 0.85$) (Fig. 2B).

A difference in T_{sk} between different interventions was only observed at the end of halftime, when T_{sk} was higher in CON than in ICE ($p < 0.001$, $d = 2.79$), MST ($p < 0.001$, $d = 1.88$) and FAN ($p = 0.010$, $d = 1.59$) (Table 1).

Heart rate and sweat rate

A difference in HR was only present at the end of halftime, when HR was lower in ICE ($d = 0.65$), MST ($d = 0.62$) and FAN ($d = 0.49$) relative to CON ($p < 0.001$) (Fig. 2C). Whole-body sweat loss was lower ($p = 0.036$, $d = 0.97$) in MST than CON (Table 1).

Thermal sensation and rating of perceived exertion

A main effect of cooling intervention on TS was observed ($p = 0.036$). Across the trial duration participants felt cooler (lower score) in ICE ($d = 0.45$), MST ($d = 0.30$) and FAN ($d = 0.18$) than in CON.

Rating of perceived exertion progressively increased from the first half to the second half of the simulated match, but all changes in RPE were similar between interventions ($p = 0.475$) (Table 1).

4. Discussion

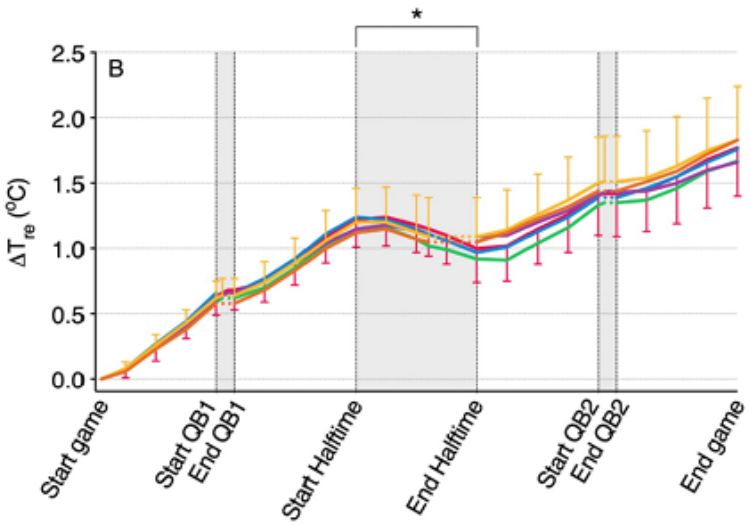
The present study demonstrates the utility of implementing extended break periods in combination with cooling strategies for rugby league in hot, humid conditions. *Stage 1* showed that extending the halftime break (from 12 to 20-min) facilitates greater core temperature and heart rate reductions, although quarter-time breaks in the absence of a cooling intervention provided only marginal additional benefits. The addition of cooling interventions in *Stage 2*, in particular a misting fan, further augmented core temperature and heart rate reductions which persisted for the remainder of play. We propose the utility of these findings extends beyond rugby league match-play and may be transferable to different high-intensity sports.

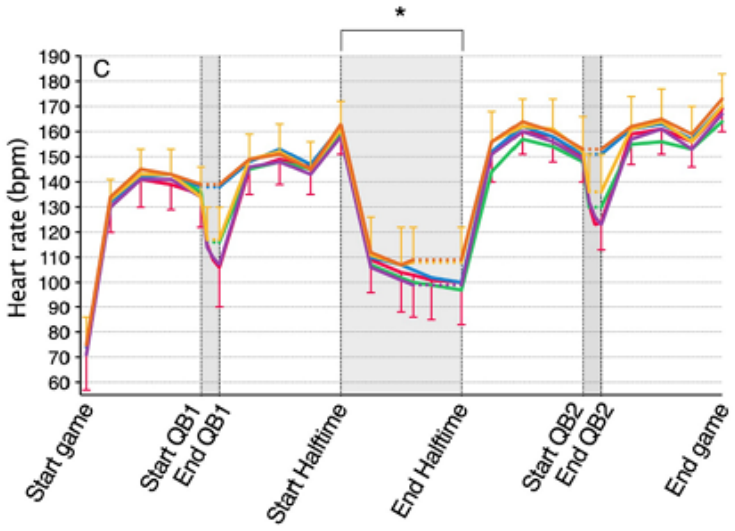
In *Stage 1*, 1-min or 3-min quarter-time breaks provided marginal, if any, cooling benefits. However, as these breaks also did not worsen strain, we included 3-min quarter-time breaks in *Stage 2* to provide additional time for the application of cooling interventions. The apparent effectiveness of an extended halftime break (from 12 to 20 min) but not quarter-time breaks in *Stage* 1 is most likely due to sustained elevations in post-exercise oxygen consumption (and therefore heat production) that are more than double pre-exercise rest after 5 min of passive recovery from prolonged exercise.[18] For a measurable level of cooling during shorter breaks, a cooling intervention is clearly essential.[7]

In contrast to our *Stage 2* hypothesis, misting fans (MST) were the most effective cooling strategy. We initially thought a humid microclimate would envelop our participants and impede sweat evaporation.[25] However, it appears that misting fans augmented evaporative heat loss by increasing skin wettedness,[23,28] with the evaporation of both secreted sweat and misted water likely accelerated by high airflow (2.5 m/s).[25] It is also possible that the misted water that remained in the air surrounding the participant absorbed latent heat from the air, creating a cooler microclimate and thus augmented convective heat loss secondary to a greater skin-to-air temperature gradient. Indeed, measurements of air temperature near the surface of a subset of participants during misting indicated air temperatures as low as 32–33 °C, despite an ambient temperature of 37 °C. Presumably, such an effect would be even more pronounced if ambient humidity was lower than the 50% relative humidity in the present study.

The electric fan (FAN) intervention provided a cooling effect under the tested conditions relative to the control condition. The utility of electric fans during hot, humid heatwaves has been demonstrated previously.[20,27] Although increasing airflow may cause a slight increase in convective heat gain (as skin temperature is lower than ambient


A
RG
40 min
12 min
40 min
R1C
20 min
1-min
20 min
12 min
20 min
1-min
20 min
R3C
20 min
3 min
20 min
12 min
20 min
3 min
20 min
EH
40 min
20 min
40 min
E1C
20 min
1-min
20 min
20 min
20 min
1-min
20 min
E3C
20 min
3 min
20 min
20 min
20 min
3 min
20 min


*
B
2.5
2.0
1.5
1.0
0.5
0.0
ΔTre (ºC)
Start game
Start QB1
End QB1
Start Halftime
End Halftime
Start QB2
End QB2
End game


*
C
190
180
170
160
150
140
130
120
110
100
90
80
70
60
Heart rate (bpm)
Start game
Start QB1
End QB1
Start Halftime
End Halftime
Start QB2
End QB2
End game

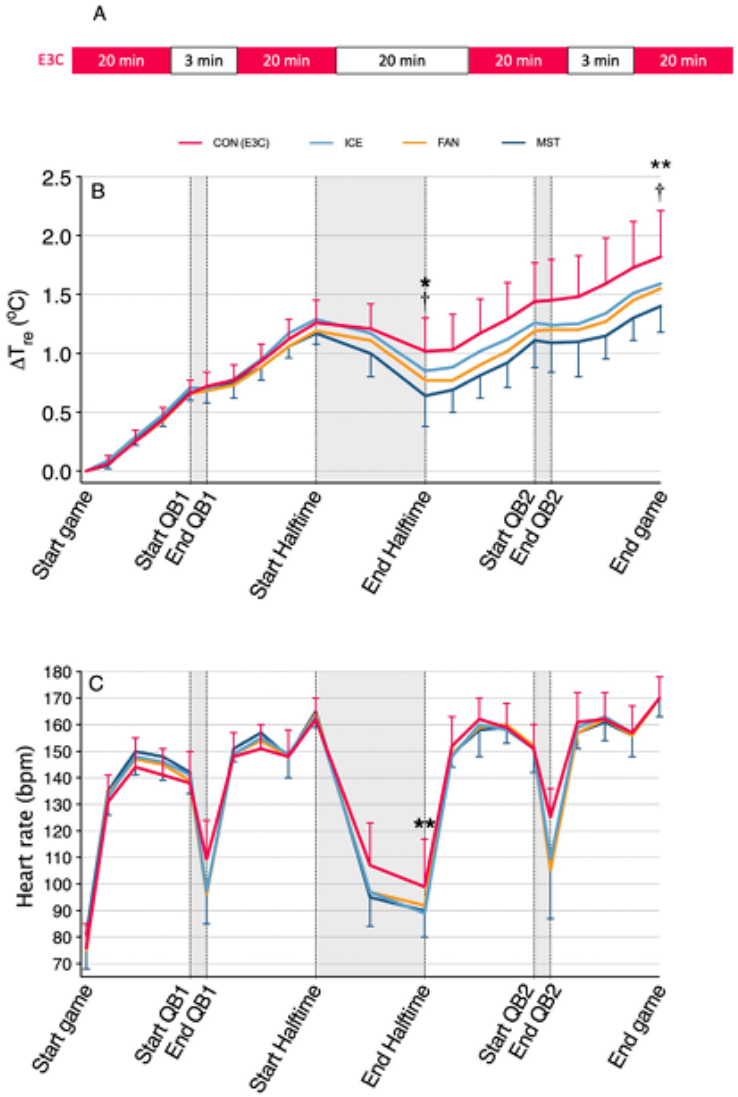


Fig. 2. Break protocol used throughout *Stage 2* (Panel A) and change in rectal temperature and heart rate during *Stage 2* (Panels B&C). ** denotes MST, FAN and ICE are all lower than CON ($p < 0.05$); * denotes MST and FAN are lower than CON ($p < 0.05$); † denotes MST is lower than ICE ($p < 0.05$).

temperature), the increase in evaporative efficiency is greater.[13,27] The independent benefit of increasing convective flow is once again demonstrated here in the FAN condition, however, the present study extends beyond the current knowledge base by demonstrating the additional benefit of augmenting convective flow with misting spray, as in the MST condition, core temperature rises were further mitigated relative to FAN, despite reduced sweating.

Ice towels have been demonstrated to be an effective cooling strategy during breaks in a tennis context both under very hot, dry[17] and hot, humid[25,29] conditions. In the present study, the ice towel intervention (ICE) did mitigate the development of thermal and cardiovascular strain relative to the control condition, but the benefit was greater in the MST condition. Ice towels accelerate local conductive heat loss from the skin on the neck and upper chest, but absolute heat loss is

Fig. 1. Overview of the six break period structures tested during *Stage 1* (Panel A). Shaded regions denote times when simulated match play was underway, unshaded sections show periods of rest. Note that the "E3C" design was used throughout *Stage 2*. Change in rectal temperature and heart rate during *Stage 1* (Panels B&C). Dotted lines bridge the timing gap caused by the varying lengths of each protocol (Panel A).* signifies a main effect of halftime length across the halftime period ($p < 0.05$).

Table 1
Summary data from Stage 1 ($n = 11$) and Stage 2 ($n = 9$). All data expressed as mean ± standard deviation.

		Stage 1						Stage 2			
		RG	R1C	R3C	EH	E1C	E3C	CON	MST	FAN	ICE
TS (mm)	Baseline	113 ± 8	111 ± 9	118 ± 15	116 ± 14	112 ± 8	112 ± 9	113 ± 10	109 ± 5	106 ± 4	106 ± 5
	End 1st Half	149 ± 25	144 ± 21	145 ± 23	140 ± 19	138 ± 18	141 ± 22	142 ± 25	141 ± 27	144 ± 27	135 ± 23
	End Halftime	136 ± 22	131 ± 23	124 ± 18	127 ± 17	122 ± 13	122 ± 17	123 ± 19	106 ± 13	110 ± 12	106 ± 8
	End Game	158 ± 28	155 ± 27	151 ± 27	146 ± 24	149 ± 28	154 ± 28	158 ± 30	147 ± 31	155 ± 35	142 ± 31
RPE (6–20)	Baseline	6 ± 0	6 ± 0	6 ± 0	6 ± 0	6 ± 0	6 ± 0	6 ± 0	6 ± 0	6 ± 0	6 ± 0
	End 1st Half	13 ± 3	12 ± 3	13 ± 3	13 ± 1	13 ± 2	13 ± 3	13 ± 3	12 ± 2	13 ± 2	12 ± 3
	End Halftime	8 ± 2	7 ± 1	7 ± 1	6 ± 1	7 ± 1	7 ± 1	7 ± 1	7 ± 2	6 ± 0	7 ± 2
	End Game	15 ± 3	14 ± 3	14 ± 3	14 ± 3	14 ± 2	15 ± 3	15 ± 3	14 ± 1	14 ± 2	14 ± 3
Tsk (°C)	Baseline	34.2 ± 0.8	33.8 ± 1.0	33.9 ± 1.2	33.9 ± 0.7	34.1 ± 0.8	33.8 ± 0.8	33.8 ± 0.9	33.9 ± 0.7	33.9 ± 0.6	34.2 ± 0.7
	End 1st Half	36.7 ± 0.3	36.7 ± 0.3	36.6 ± 0.5	36.6 ± 0.3	36.7 ± 0.4	36.6 ± 0.4	36.6 ± 0.5	36.8 ± 0.3	36.8 ± 0.5	36.7 ± 0.3
	End Halftime	37.1 ± 0.2	37.0 ± 0.2	36.9 ± 0.3	36.8 ± 0.2	36.8 ± 0.4	36.9 ± 0.4	36.9 ± 0.4	35.9 ± 0.6*	36.1 ± 0.6*	35.5 ± 0.6**
	End Game	36.5 ± 0.7	36.6 ± 0.5	36.4 ± 0.8	36.5 ± 0.5	36.5 ± 0.6	36.4 ± 0.6	36.4 ± 0.6	36.6 ± 0.4	36.9 ± 0.6	36.4 ± 0.5
WBSL (kg)	Whole Game	2.07 ± 0.41	2.02 ± 0.40	2.04 ± 0.34	2.12 ± 0.41	2.20 ± 0.46	2.11 ± 0.38	2.14 ± 0.40	1.83 ± 0.22*	2.09 ± 0.39	1.94 ± 0.32

* Less than CON ($p < 0.05$).
** Less than CON ($p < 0.0001$).

limited by the relatively smaller surface area of the skin participating in heat exchange, especially in comparison to the accelerated evaporation from a much greater skin area with the misting fan. Ice towels were the most effective strategy for eliciting a cooler thermal sensation. This finding is consistent with work demonstrating the marked impact rapid reductions in local skin temperature can have on thermal sensation,[1,31] especially on the neck and chest region where cold thermoreceptor density is relatively high.[19,24] While not assessed in the present study, the combination of ice towels and misting fans may provide additive perceptual and physiological cooling benefits. This notion requires verification with further research.

As previously observed, the addition of water to the skin surface caused a reduction in sweat output in the MST condition while still maintaining evaporative heat loss.[23] Typically, during prolonged intermittent sports such as rugby league, football and hockey, fluid replacement and the avoidance of dehydration are dictated by ad libitum fluid consumption. During prolonged (>1.5 h) exercise, when sweat rates are high, ad libitum fluid consumption is often insufficient[16]; the reduction in sweat output in the MST condition may therefore be an additional benefit, i.e. it reduces the potential for dehydration and its accompanying performance decrements and strain exacerbations.[8] However, the implementation of misting fans as a cooling intervention may not be practical in all sporting contexts; owing to the high water output, sports such as tennis or basketball in which the court surface may become slippery may be better suited to the use of an electric fan without the addition of misting but with the external application of water using a wet cloth or sponge.

Although we attempted to enrol participants of similar size and fitness to elite rugby league athletes, the participants in the present study were on average lighter than a typical player.[12] This discrepancy does not impact the findings related to cooling intervention effectiveness but does limit the comparability of some physiological data such as ΔT_{re} as heat production relative to body mass may be different.[10] Additionally, environmental conditions undoubtedly impact both the efficacy of various cooling interventions, and optimal break structures. In cooler conditions, wherein ambient temperature is lower than skin temperature, short cooling breaks may yield more tangible results without cooling interventions as convective heat loss be naturally greater. Additionally, in hotter, dryer environments accelerating airflow via an electric fan may be detrimental as evaporative efficiency is already high, yet the dry heat load via convection will be accelerated.[13,27] The effectiveness of the misting fan technique may be increased in a hot, dry environment wherein evaporation is limited primarily by sweat rate rather than a vapour pressure gradient.[25]

In conclusion, the present study demonstrates that extending the halftime break duration from 12 to 20 min during a simulated rugby league match in hot, humid conditions mitigates thermal and cardiovascular strain, however, the addition of short (1 to 3-min) quarter-time breaks provides limited additional benefit. The use of a high-speed misting fan during these breaks in play was more effective for mitigating rises in core temperature and heart rate than the use of an electric fan alone or the application of ice towels.

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Declaration of interest

None.

Confirmation of ethical compliance

Ethical approval was gained from the University of Sydney *Human Research Ethics Committee* (project number 2017/213).

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