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Thermoregulatory adaptations following an intermittent post-exercise sauna bathing intervention are partially sustained after 2 weeks decay in trained middle-distance runners.

by

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Abstract

This study investigated the decay of heat acclimation (HA) adaptations across a two-week period of continued training with no HA stimulus, following three-weeks post-exercise sauna bathing. The subsidiary aim was to investigate whether we could repeat findings our laboratory has previously published (Kirby et al, 2020; 2012), and confirm that post-exercise sauna bathing across three-weeks can effectively heat acclimate endurance athletes. Nineteen trained endurance athletes (mean ± SD, age 20 ± 1 years, $\dot{V}O_{2max}$ (60.0 ± 6.0 mL·kg−1·min−1) performed a running heat tolerance test (30 min, 9 km·h−1/2% gradient, 40 °C/40%RH; HTT) pre, post 3-weeks of normal exercise training plus 30-min post-exercise sauna bathing (85–94 °C) 3 ± 1 times per week, and post 5-weeks after normal exercise only. Following post-exercise sauna bathing resting core body temperature (0.23 ± 0.06°C, $p <$ 0.01), peak core body temperature (0.26 ± 0.07°C, $p = 0.004$), end mean skin temperature (0.90 ± 0.13°C, $p < 0.001$), resting heart rate (HR) (7 ± 2 bpm, $p = 0.42$) and peak HR (6 ± 2 bpm, $p = 0.002$) decreased before and during the HTT. Perceptual measures including RPE, thermal sensation and thermal comfort, and sweat losses showed no significant changes ($p >$ 0.05), however. Peak core body temperature (0.17 ± 0.06°C, $p = 0.035$) was partially retained 2-weeks post HA, along with a partial retainment of end skin temperature and HR (although not significant ($p > 0.05$)). Resting and peak core body temperature and peak skin temperature had a lower decay rate than resting and peak HR (20%, 30%, 24% and 44% and 50% decay rate, respectively). Three-weeks post-exercise sauna bathing is an effective and pragmatic method of HA that sustains some HA adaptations for two-weeks.

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To the participants who volunteered to be part of this study and showed up to each sauna after tiring training, and lasted through those long hard 30 minutes in the heat. Without you all this research would not have been possible.

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List of Abbreviations

ANOVA	Analysis of variance
ATP	Adenosine triphosphate
CNS	Central nervous system
HA	Heat acclimation
HR	Heart rate
HRA	Heat re-acclimation
HTT	Heat tolerance test
HWI	Hot water immersion
ICC	Intra-class correlation coefficient
MAP	Mean arterial blood pressure
RPE	Rating of perceived exertion
SD	Standard deviation
SV	Stroke volume
TEM	Typical error of measure
T_{sk}	Skin temperature
$T_{recRISE}$	Change in rectal temperature
TTE	Time trail to exhaustion
VO2max	Maximum oxygen consumption
WBGT	Wet bulb globe temperature

1. Introduction

Heat acclimation (HA) is a method of training and preparation that is gaining popularity amongst athletes to optimise their performance in the heat. HA can be achieved via active strategies (i.e. exercising in hot environments, either outdoors or often in a lab-controlled setting e.g. heat chamber), or passive strategies (not requiring exercising in the heat, instead includes using modalities like hot baths and sauna (Heathcote et al., 2018)). Athletes are increasingly required to compete in hot environmental conditions, such as the 2023 World Athletic Championships in Budapest. Therefore, they need to be exposed to similar or even slightly hotter environmental conditions in order to prepare and adapt their body for the additional physiological strain created by exercise performance in the heat (Sawka et al., 2016). Such physiological adaptations can be gained through HA (Périard et al., 2015). HA has its limitations, however, with only 15% of athlete's surveyed in a study by Périard et al. (2017) using HA to prepare for competition in the heat.

HA can be affected by an athlete's training and travel schedules as a result of limited time and available resources (Pryor et al., 2019). It is also essential to consider the timing in which HA is carried out. Although it is recommended to acclimate for 2-weeks before a major competition, athletes also need to taper prior to competition. Tapering is important after a period of strenuous training, to help reduce fatigue, repair muscle damage and replenish muscle glycogen stores (Le Meur, Hausswirth & Mujika, 2012). Thus tapering gives the body and mind time to recover and prepare for race day (Grivas, 2018). To help avoid disruption to an athlete's training programme, post-exercise sauna bathing has been shown as a practical and accessible HA alternative to active HA (Scoon et al., 2007; Kirby et al., 2021). However, it is unknown when post-exercise sauna bathing should be implemented or halted prior to competition.

Heat acclimation decay/HA decay rate relates to the rate per day at which adaptations are lost over time after an individual has stopped being exposed to heat stress (Pandolf, 1998). The rate and process of HA decay is still debated within the literature, due to the relatively limited number of studies and various protocols used to examine HA decay. For athletes, HA or heat re-acclimation (HRA) sessions should be performed in advance of competition, avoid interfering with the taper period whilst also ensuring HA decay is limited. Furthermore, the literature suggests the rate of decay is rapid (e.g. 2.5% loss in adaptations per day; Daanen et al, 2018) and therefore, exploring methods that allow us to sustain HA adaptations for ~2–4-weeks would also be valuable (Daanen et al., 2018 and Pryor et al., 2019). Therefore, it needs to be established if an alternative HA protocol, such as post-exercise sauna bathing, has the same decay profile as more traditional active HA protocols. It also needs to be understood how long athletes have until they HA adaptations are lost after stopping a post-exercise sauna bathing intervention.

2. Literature Review

2.1. Heat balance

Humans are homeotherms with their resting core body temperature tightly regulated at a mean value of ~37°C via behavioural and physiological mechanisms (Tansey & Johnson, 2015). Humans are also endothermic as heat is generated during metabolism. Specifically due to the low metabolic efficiency of generating adenosine triphosphate (ATP), at least 75-80% of energy released during physical activity is lost as heat and only 20-25% is external work (Sureda et al., 2015). Thus when exercising the working muscle produces a large amount of heat. This metabolic heat production needs to be balanced with the rate of heat exchange to the surrounding environment otherwise humans become hyperthermic (Périard, Eijsvogels & Daanen, 2021).

Biophysical properties control heat exchange between the body and environment and there are four main pathways that the body uses to gain and dissipate heat. These include radiation, conduction, convection and evaporation (Cheung, 2010). Conduction transfers heat by having physical contact with an object that draws out or absorbs the heat. Convection is heat transfer via the movement of a fluid across our body, such as air or water. A body radiates heat if the environments cooler or it can gain heat through radiation from the sun. Finally, evaporation transfers heat through the main process of sweating. Overall, Heat Exchange = Radiation + Conduction + Convection + Evaporation (Cheung, 2010). Biophysical properties are dictated by the surrounding temperature, humidity and air motion, sky and ground radiation & clothing. Thermoregulation is the balance between generating and losing heat to maintain core body temperature (Romanovsky et al., 2018). When core body temperature increases, as occurs during exercise, heat produced by working muscles is transferred to the skin and on to the environment through cutaneous vasodilation. Thus, skin

temperature increases proportionally with metabolic work (Galloway & Maughan, 1997; Hargreaves, 2008). Notably, the body dissipates heat from the skin largely via evaporation of sweat, which facilitates heat loss and cools the skin (Hardy, DuBois, & Soderstrom, 1938). This helps maintains a core-to-skin temperature gradient that helps maintains thermal balance. When exercising in hot environments this core-skin temperature gradient is narrower, which determines that there is less capacity for the core to lose heat (via the skin) to the environment (Rowell, 1993; Sawka and Wenger, 1988). Consequently, core body temperature increases rapidly. Heat loss is further impaired under conditions of heat and high humidity due to our reliance on evaporative heat loss (Cramer et al., 2022). Thus, exercise in a hot environment, particularly a hot humid environment, challenges thermoregulatory homeostasis and results in a state where core body temperature is elevated (i.e. hyperthermia). This has significant consequences for an individual's physical performance and safety.

2.2. Exercising in hot environments

Heat stress is a result of environmental conditions, including temperature and humidity, physical work rate, and other factors that impede heat loss, for example the wearing of heavy clothing (Sawka et al., 1996). The interaction of environmental heat stress and exercise significantly increases physiological strain (Nadel, 1977). This is because exercising heat stress creates conflict between thermoregulatory and other homeostatic requirements, which together can lead to significant health risks and decreases in aerobic performance (Sawka et al., 1996). Increases in whole body temperature due to exercise heat stress result in a spectrum of health conditions referred to as exertional heat illnesses (Armstrong et al., 2007). These illnesses range from mild conditions such as heat cramps (which stops a person from being able to exercise for a short time) and heat syncope (which

causes loss of consciousness), to more severe conditions such as heat exhaustion (which can lead to hypotension, cardiovascular insufficiency, and heart failure), and heat stroke (which is associated with central nervous system (CNS) dysfunction, and organ failure (Périard, Eijsvogels & Daanen, 2021)). Performance decrements also arise from thermoregulatory strain. These include reductions in sustainable and maximal power output, and peak oxygen uptake during extended periods of high intensity, self-paced exercise in the heat (Périard et al., 2011). In particular, submaximal (marathon or triathlon) and high intensity sports are effected by heat stress. For example, Ely et al. (2007) demonstrated the impact of heat stress on marathon running performance and finishing times. Specifically, as wet bulb globe temperature (WBGT) increased from 10 to 25°C (dry bulb temperature 8-22°C), elite performers slowed by ~2% (2–3-min), whilst 3-hour competitors slowed by almost 10% (18-min). Similar to Ely et al. (2007), Périard et al. (2011) also measured the consequences of heat stress on endurance athletes. Measuring time trial performance across 40km of cycling in both hot (35°C) and thermoneutral (20°C) conditions, they showed that in the hot condition participants took longer to complete the time trial with a duration of 64.3 ± 2.8 min compared to 59.8 ± 2.6 min in the thermoneutral condition. The hot condition also had an overall lower power output of 242.1 W compared to the thermoneutral condition with a power output of 279.4 W and power output in the heat was impaired after 20-mins. Further to this, core body temperature consistently increased across the hot time trial and alongside a progressive increase in cardiovascular strain over ~60-min, this was believed to have caused these results (Periard et al., 2011). Both these studies demonstrate athletes underperform in the heat compared to their performance in cooler environments, with this underperformance largely caused by the increased strain on the thermoregulatory and cardiovascular systems.

The major contributors to impaired performance in the heat include impaired cellular and CNS function, quicker muscle and peripheral fatigue, and impaired cardiovascular and

thermoregulatory function (Périard, Eijsvogels & Daanen, 2021). As mentioned previously, during submaximal aerobic exercise under heat stress, there is an increase in whole body (core, skin and muscle) temperature. Increased whole body temperature results in a lessening core-to-skin temperature gradient and a reflex increase in skin blood flow for the purpose of thermoregulatory needs and heat dissipation (Périard, Eijsvogels & Daanen, 2021). The cardiovascular system also has to maintain perfusion pressure and oxygen delivery to exercising muscles for metabolic demands. This increases cardiovascular strain including increases in heart rate (HR) and decreased stoke volume (SV) which compromises cardiac output and mean arterial blood pressure (MAP). Alongside this, with exercise under heat stress, $\dot{V}O_{2max}$ decreases while perceived exertion increases (Robertson, 1982; Pandolf, 1982). All of these together with the adjustments in blood flow to the skin, active muscles, and the brain, have been suggested to mediate fatigue and lead to a reduction in work rate and volitional exhaustion. This is all supported by findings from Ely et al. (2010). Participants performed a 15-min self-paced cycling bout in temperate (20°C) and hot (40°C) conditions and it was found ~17% less work was performed in the hot condition compared to the cooler condition (Ely et al., 2010). They demonstrated this impairment was as a result of ~5°C increases in skin temperature and ~10 beats/min increases in HR during the time trial in the hot conditions. Voluntary activation of the exercising muscles is also suppressed in the heat by a reduction in the neural drive for motor unit recruitment. Increases in muscle temperature, increase metabolic and mechanical disruption which stimulates group III/IV muscle afferents and provides inhibitory feedback to the CNS (Périard, Eijsvogels & Daanen, 2021). Muscle fatigue with exercise in hot environments is also caused when muscle glycogen breakdown is accelerated, muscle lactate production is increased (decreasing pH), and free radical production in skeletal muscles in increased (Maughan, 2010).

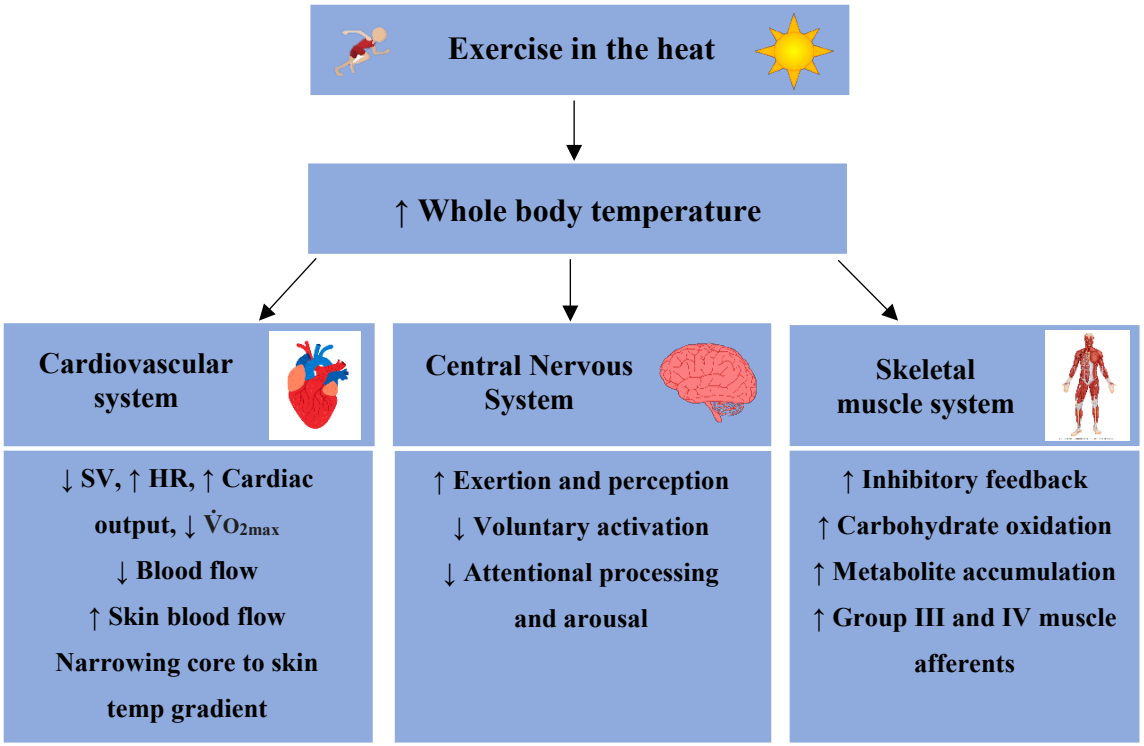


Fig 1. Schematic representing the effect of an increase in whole body temperature (core, skin and muscle) on the cardiovascular, central nervous system (CNS) and skeletal muscle function when exercising in the heat (adapted from Periard et al., 2015).

Ultimately heat tolerance is defined by an individual's capacity to physiologically adjust to this heat stress and defend against a rapid rise or reaching a critical core temperature (Mitchell et al., 2019). With extended exposure to heat, humans are exceptional at adapting and tolerating to climatic heat stress (Periard et al., 2015). Some well-trained athletes can reach a core body temperature of 41.5°C without any short or long term damaging effects (Périard, Eijsvogels & Daanen, 2021). Heat tolerance and the effectiveness of the thermoregulatory system can be influenced by personal factors such as acclimatisation state (Wenger, 1988) and aerobic fitness (Armstrong and Pandolf, 1988), alongside hydration level (Sawka and Pandolf, 1990). Trained athletes who have acclimatised to the heat and stay fully hydrated stop the effects of heat stress mentioned above, and can perform optimally in hot

environments (Sawka et al. 1993). Therefore, gaining this heat tolerance is particularly important for athletes expecting to compete in hot conditions.

2.3. Heat acclimation

Heat acclimation is the process of physiological adaptation that improves a person's heat tolerance and enables an individual to exercise for long periods in the heat with limited performance impairment (Gibson et al., 2015). HA specifically occurs in an artificial environment (e.g. laboratory or environmental heat chamber), with an individual periodically exposed to exercise in the heat over several days. Whereas, heat acclimatisation is where an individual is simply exposed to natural hot climates for a long period of time. Overall, both HA and heat acclimatisation aim to improve an individual's capacity to lose heat from their body. This study looked at HA as opposed to heat acclimatisation, therefore HA is the focus of this literature review.

2.3.1. Background of Heat Acclimation:

Human heat adaptation has been a known concept for centuries. In 1786, the first report on human heat acclimatisation was written by James Lind (Lind, 1786, as cited in Periard et al., 2015). It is unequivocal that HA improves an individual's heat tolerance. Research into HA in relation to athlete's performance is somewhat limited and has only been focused on in the last 20-years. Originally, HA protocols were targeted for moderate workloads in less fit populations than athletes, e.g. occupational and military purposes (Periard et al., 2015). These HA studies were later adapted for athletic contexts but due to their lack of specificity and the fact that the requirements of military and other occupations differ to those of athletes, these protocols are considered less appropriate for athletes to follow. Therefore, the gold standard traditional HA protocol involves maintaining an elevated

core body temperature by 1-2 degrees Celsius for ~60 minutes, for a minimum of 5-consecutive days, or over a 2-week period allowing for a few rest days (Deshayes et al., 2023).

2.3.2. Purpose of Heat Acclimation:

The interaction between hot environments and exercise not only brings about physiological adaptations necessary to optimise performance but helps athletes understand how they can prepare for competition (Bergeron et al, 2012 and Racinais et al, 2015). HA training is most beneficial when it simulates the expected competitive environment and fits around the athletes available time and resources. Optimal results occur when HA is specific to the climate the athletes will be exposed to and the physical exercise intensity they will perform (Pryor et al., 2019). It is especially important for athletes who take part in longer duration/distance events (Nybo et al., 2014; Guy et al., 2015). HA occurs relatively rapidly (Gibson et al., 2015). Most HA benefits occur in only a week of heat exposure (Pandolf, 1998 and Shapiro, Moran, and Epstein, 1998) and thermoregulatory benefits are generally optimised after 10-14-days (Périard, Racinais, and Sawka, 2015).

Common HA physiological adaptations include reduced core and skin temperatures, decreased HR, increases in skin blood flow and sweating plus improved fluid balance and cardiovascular stability (the heart, lungs and blood transport oxygen more effectively during sustained periods of exercise) (Taylor, 2014; Periard et al., 2015). HA increases the onset threshold, sudomotor sensitivity and max capacity of sweating and skin blood flow responses (Periard et al., 2015). HA also results in decreased electrolyte loss during exercise, aiding retention of total body water (Periard et al., 2015). Increasing total body water means there is more body water available for sweating and evaporative heat loss. This then decreases skin blood flow demand, which in turn, shifts blood volume to the central circulation and activates

the sweat response sooner (Periard et al., 2015). Therefore, after HA, for a smaller increase in core body temperature, there is the ability to sweat and dilate the vessels more (Frye and Kamon, 1981). Improvements in evaporative cooling are beneficial as it minimises heat storage during long periods of exercise. Heat is lost quicker to the environment, leading to cooler skin and core body temperatures during exercise (Periard et al., 2015). This then reduces cardiovascular strain, decreasing HR and increasing SV.

Therefore, overall HA can be used to augment physical training by mitigating heat-related changes in cardiovascular, CNS and skeletal muscle function that can cause negative effects on performance. It can alter substrate metabolism via glycogen sparing, helping prevent/avoid fatigue; increasing lactate threshold so individuals can exercise at a higher intensity; reducing lactate concentrations during exercise, and improving economy (Périard et al., 2015).

2.3.3. Aerobic performance benefits:

HA benefits on aerobic exercise performance in the heat have been well researched and accepted. Submaximal exercise performance and enhanced endurance exercise capacity has been shown to improve by ~23% following HA (Tyler et al., 2016). These HA-related aerobic performance improvements are accredited to the numerous physiological adaptations gained through HA (as outlined in the previous section). The key HA adaptations improving performance specifically during submaximal exercise in the heat include increased $\dot{V}O_{2max}$ (from 4% up to 10%) (Lorenzo et al., 2010 and Keiser et al., 2015), a reduction in oxygen uptake (Sawka, Wenger & Pandolf, 2010; Sawka et al., 1983) and blood and muscle lactate accumulation (Febbraio et al., 1994), as well as an increase in lactate threshold (Lorenzo et al., 2010; Neal et al., 2016) and reduced muscle glycogen utilisation (King et al., 1985).

However, it still remains unclear what mechanisms are behind the performance improvement adaptations listed above.

Not only has it been shown that the physiological adaptations achieved by HA can lead to improvements in aerobic capacity/performance in hot conditions, they may also improve aerobic exercise performance and be used as an ergogenic aid in temperate-cool conditions. For example, Kirby et al. (2020), who used the same protocol and laboratory as this current study, found running speed at 4 mmol L−1 [La−] increased by ~4%, and time to exhaustion increased by ~12% after completing a HA protocol consisting of post-exercise sauna bathing (101–108°C, 5–10% relative humidity (RH)) 3 ± 1 times per week. Temperate (18°C) exercise tests were used to measure both running speed and time to exhaustion. These included a lactate profile test consisting of 3-min stages (1% gradient; speed increased by 1 km/h each stage) until lactate exceeded 4 mmol/L, and a ramp-style VO_{2max} test in which speed was increased each minute by1 km/h until the speed at which > 4 mmol/L [La^{-}] during the lactate profile test was reached and then the gradient was increased by 1% each minute until volitional exhaustion. Other studies, including a study by Lorenzo et al. (2010), have also shown HA improved performance. Similar to Kirby et al. (2020), Lorenzo et al. (2010) also found time to exhaustion in cool conditions to improve after HA but this time by 6%. Lorenzo et al. (2010) also found a 5% increase in $\dot{V}O_{2max}$. They used a different HA protocol to Kirby et al. (2020), however, that consisted of 10-consecutive days of two 45-min cycling bouts (10-min rest in between) at ~50% $\dot{V}O_{2max}$ in 40°C; 30% RH and tested maximal aerobic power ($\dot{V}O_{2max}$), time-trial performance, and lactate threshold, in cool conditions of 13°C, 30% RH. Other studies have shown no significant improvements in performance, however. For example, Keiser et al. (2015) found training in the heat beneficial for $\dot{V}O_{2max}$ and time trial exercise performance in the heat (38°C) but not in normal temperatures (18°C). They used a HA protocol consisting of 10-days of 90-min exercise training in 38°C and 30%

RH and tested time trial performance over 30-min in 18°C. Therefore, it appears heat training as an effective ergogenic method for performance in temperature environments is still up for debate and the HA protocol and testing conditions may influence temperature performance outcomes.

2.3.4. Heat Acclimation Conditions:

When looking at favourable conditions for HA, as mentioned previously, replicating similar or slightly more stressful competition conditions have been recommended (Périard et al, 2015). This includes using protocols with the same exercise mode, ambient temperatures and humidity as expected in competition. Conditions during HA protocols should aim to increase core body temperature to ~38.5°C (Regan et al., 1996; Taylor and Cotter, 2006) as exposing an individual to this level of heat strain is enough for physiological adaptations to occur (Gibson et al., 2015). Elevating core body temperature above 38.5°C has been shown to not induce any additional HA advantages (Gibson et al., 2015). Therefore, the HA approach used strongly influences the physiological adaptations gained in both magnitude and timeframe (Daanen et al. 2018).

There are different types of HA approaches. There are two main modalities of HA training including active and passive. Active HA combines environmental heat exposure and exercise, such as walking, running or cycling, often in lab-based controlled settings (Pallubinsky et al., 2017). Active HA approaches include constant work rate exercise, controlled hyperthermia and controlled HR and self-paced exercise (Périard, Eijsvogels & Daanen, 2021). The constant work rate approach is where the participant exercises at a constant predetermined workload (controlled exercise intensity, frequency and duration) in hot and/or humid conditions. Using a constant workload from a pre-acclimation baseline and exercising within a stable hot environment, eliminate the issues of having to consistently

monitor work: rest ratios in order to maintain specific core body temperatures, which can be difficult and usually requires alterations throughout each session. On the other hand, this type of approach does not always optimally potentiate the stimulus (e.g. increase core temperature high enough to bring about HA adaptations). Specifically, the thermal strain experienced at the beginning of a HA protocol diminishes as participants start to adapt (Taylor and Cotter, 2006, Taylor, 2014). The controlled hyperthermia technique (also known as isothermic method) is where during a HA session, the workload, work-rest ratio or heat exposure is altered to maintain a target core body temperature of ~38.5°C. Similarly, the controlled HR technique is where a target HR is maintained during a HA session (Taylor, 2006; Périard et al., 2015). Gradually increasing the intensity, duration or frequency of exercise sessions performed in a hot environment, stimulates a rise in core body temperature/HR until the HR target is reached. To facilitate HA, higher core body temperatures are provoked through hard interval training or continuously exercising at intensities higher than 50% VO_{2max} (Pandolf et al. 1977; Williams et al. 1967). The self-paced exercise approach takes into consideration fitness level, training and the hot ambient conditions and depending on these factors the individual will select their own work rate during exercise in the heat. It is particularly beneficial for team sports as individuals can self-regulate their effort according to fitness level, training parameters and ambient conditions, whilst exercising in large groups (Racinais et al., 2012). The potential disadvantage of this approach is how difficult it is to standardise individual stimuli around training sessions. Individuals select their own work rate based on their levels of perceived discomfort in the heat, therefore problems can arise with the different levels of thermal load (Tyler et al., 2016). Overall, isothermic HA methods appear to provide a more efficient means of at inducing heat adaptations than constant workload methods (Gibson et al., 2015).

The amount of daily exposures seem to be more important than the duration of the exposure itself (Shaw et al., 2022). This is based on previous studies which showed phenotypic HA adaptations (adaptations to specific environmental stresses; Taylor, 2006) following HA sessions of varying duration (90-min (Garrett 2012; 2019), 30-min to 120-min (Tyler et al., 2016), and 240-min (Racinais et al., 2012). Short-term (<7 days; Garrett et al., 2011), medium-term (8–14 days; Garrett et al., 2011) and long-term (> 15 days; Garrett et al., 2011) HA interventions can improve exercise capacity and performance (Tyler et al., 2016). However, medium-term and long-term HA protocols appear to provide more complete adaptations than short-term HA protocols, which induce only 80% of HA adaptations (Tyler et al., 2016; Moss et al., 2020). Although short-term HA protocols have been shown to induce core body temperature and HR HA adaptations in highly trained athletes in as little as 5-days (Stone et al., 2022), to achieve sweat response adaptations, HA protocols should aim to last at least 14-days (long-term HA) (Tyler et al., 2016). However, short-term HA protocols are arguably the most economical for athletic populations. For example, when integrating HA into a pre-competition taper (Gibson et al., 2015). Regardless of the length of HA protocol, it has been suggested intervening periods of 2-3 days of cool weather with heat exposure (Périard et al., 2015; Sawka et al., 2003) does not seem to interfere with HA or effect HA adaptations. Also, intermittent protocols seem to be as effective at achieving adaptations as consecutive protocols, as long as the interval between HA sessions is not more than around 3-days (Fein et al., 1975 and Duvnjak-Zaknich et al., 2019).

Overall, compared to passive HA, active HA strategies have been greatly researched in the literature (Tyler et al., 2016; Heathcote et al., 2018). However, active strategies have several practical and economic limitations (Heathcote et al., 2018). Active methods require subjects who live in cool/temperate conditions to travel to hotter environments or use methods such as a heat chamber. These methods are both expensive and can be logistically

difficult and impractical (Casadio et al., 2016). As well as this, active methods combine exercise and heat exposure, which typically replaces/interferes with normal training and specific taper periods. This can have a negative effect on an athlete's training outcome (Heathcote et al., 2018).

In contrast to active HA strategies, there has been comparatively little research on passive HA strategies (Tyler et al., 2016; Heathcote et al., 2018). Instead of using exercise in a hot environment to induce HA adaptations, passive HA involves regular exposure to a high exogenous heat load through methods such as a hot bath (e.g., ~40°C), environmental chamber (e.g., >45°C), or sauna (e.g., 70–90°C) (Périard, Eijsvogels & Daanen, 2021). Passive strategies often provide cost effective and more practical solutions (Heathcote et al., 2018). Based on the evidence in the literature, passive HA strategies bring about many physiological heat adaptations, specifically decreasing exercising and resting core body temperature and HR, inducing plasma volume expansion, and initiating an earlier onset of sweating as well as higher sweat output (Gibson et al., 2020). Such adaptations have been shown to improve aerobic performance as well as thermoregulatory, cardiovascular, and perceptual responses (Heathcote et al., 2018). A reduced core body temperature during rest and exercise is a key variable associated with successful HA (Tyler et al., 2016). A review by Heathcote et al. (2018) found passive HA strategies to reduce resting core body temperature by an average of ~0.26°C following 10–11-day passive HA protocols that used 1-hour, 2-hour, or 4-hour heat exposures per day. A more recent study by Barry et al. (2020) also found that resting core body temperature reduced by 0.29°C after a 7-day passive HA protocol. Comparatively, a meta-analysis by Tyler et al. (2016) found a ~0.2°C reduction in resting core body temperature following active HA. Therefore passive HA protocols can suffice as an effective alternative method to these active strategies. There are limitations to passive strategies, however. Although athletes can incorporate passive HA more easily into their

training programmes and schedules, as passive HA exposes a person to realistic heat challenges they could encounter in an everyday situation without requiring exercise in the heat (Zurawlew et al., 2018), passive heat exposures alone may not be as effective as active HA for sport-specific HA adaptation (Daanen et al., 2018). Further, it is unknown how long physiological adaptations from passive HA are maintained.

The final HA method used is post-exercise heat stress. In this method, exercise is performed under temperate conditions and immediately after exercise an additional period of heat stress is applied (Daanen et al, 2018). This post-exercise heat stress can be elicited via methods such as sauna or hot water immersion (HWI) with ambient/water temperatures of at least 80°C and 40°C, respectively (Heathcote et al., 2018). A review by Heathcote et al. (2018) has reported that ~6–7 post-exercise heat stress exposures are enough to induce advantageous physiological and performance effects (Brazaitis and Skurvydas, 2010; Zurawlew et al., 2016). However, medium term protocols (8-14 day duration) seem to have been most used in protocols (Scoon et al., 2007; Beaudin et al., 2009; Zurawlew et al., 2016). Post-exercise heat stress serves to maintain or further increase core and skin temperatures elicited from exercise (Heathcote et al., 2018) and therefore provoke a HA adaptation stimulus (Fox et al., 1963). Specifically, post-exercise HA and associated elevations in core body temperature (above what can be achieved with exercise in cooler conditions) has been reported to induce a greater heat shock protein synthesis, which can lead to larger thermoregulatory-adaptive responses (Casadio et al., 2016; Casa, 2018). The adaptations induced by this approach are similar to those of more traditional HA protocols (Périard et al., 2021). Therefore, research highlights such benefits could be replicated for athletes looking to use alternative strategies to active HA. For example, Ashworth et al. (2023) found post-exercise HA benefitted and provided HA adaptations to military units operating in the heat. Ashworth et al. (2023) conducted a 5-day post-exercise HA regime in

either the sauna (70 °C, 18% RH) or HWI (40 °C) for ≤40 min and showed that thermoregulatory adaptations were induced. Furthermore, all changes could be tracked daily which allowed on-going assessment of adaptations (Ashworth et al., 2023). Indeed, Kirby et al. (2020) showed that 30-mins post-exercise sauna bathing, 3 ± 1 times per week for 3-weeks induced thermoregulatory adaptations during a heat tolerance test (HTT). Additional to the physiological benefits of post-exercise heat stress, this method avoids the challenges associated with traditional active HA by providing little impact on an athletes training and offers more flexibility (Kirby et al, 2020). Therefore, overall it provides an alternative practical HA approach in individuals wanting to maintain training quality or who find the more traditions exercise-based interventions harder to adopt (Casadio et al., 2017; Saunders et al., 2019). It is currently unclear what is considered too long between exercise ending and the post-exercise heat stress commencing, however (Scoon et al., 2007).

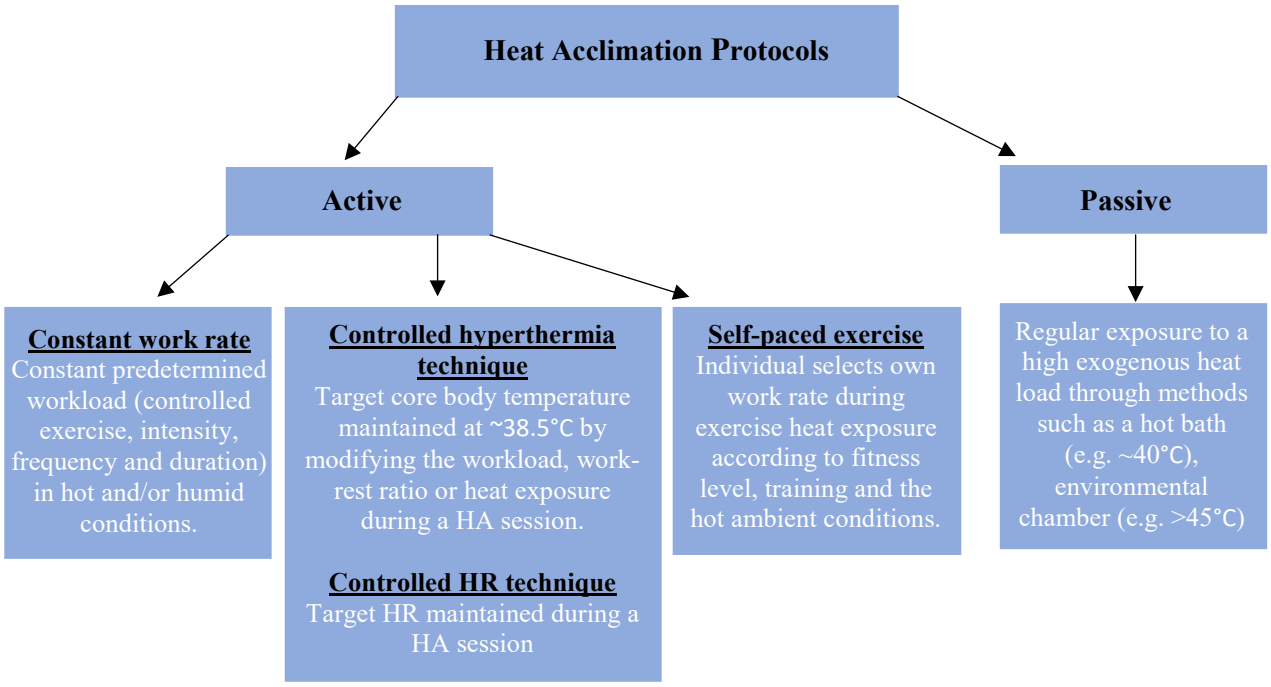


Fig. 2 Schematic representing the different types of heat acclimation (HA) protocols divided into active and passive methods.

2.3.5. Post-exercise Sauna Bathing:

Sauna (i.e., Finnish bath) protocols involve sitting in a wooden floored and panelled room exposed to dry heat and high temperatures (≥80°C with only 5-10% relative humidity) for 30-40 minutes) (Hannuksela and Ellahham, 2001). The sauna offers a hot-dry heat that allows for evaporative heat loss (Ashworth et al., 2023). Traditional HA strategies are not always the best method for athletes who live in cold temperate environments or do not have access to artificial methods such as an environmental chamber. Therefore, instead of training in natural or controlled laboratory-based hot environmental conditions, post-exercise sauna bathing has been suggested as an alternative method (Stanley et al., 2015; Scoon et al., 2007). Furthermore, when athletes exercise in the heat their training can become compromised as exercise has to be completed at a lower rate. This is because they struggle to cope with the heat load and the associated increased physiological strain. Sauna exposures after a training session have been suggested to remove some of this strain associated with exercising in the heat (Scoon et al., 2007).

The temperature of the sauna should be at least 80°C so that the target core body temperature of around 38.5°C can be reached (Scoon et al., 2007). Exposing participants to hot ambient temperatures of this magnitude provides a significant external heat load that allows HA to be attained due to raised skin temperatures (Regan et al. 1996; Shvartz et al. 1973). Based on studies by Scoon et al. (2007) and Stanley et al. (2015), participants should be exposed to a heat stimulus that has a minimum duration of 30-minutes per session. Both studies reported significant plasma volume expansion (7–17%) after using ~10–15 30-minute post-exercise sauna sessions. Furthermore, Scoon et al. (2007), found a 32% improvement in running time to exhaustion (at the runners' personal best time over 5 km) after 12 post-exercise (i.e. training bouts in cool environments) sauna bathing (90°C for 30-

min) sessions, interspersed across a 21-day protocol. Scoon et al. (2017) also found post-exercise sauna bathing to enhance endurance performance of sub-elite runners by 2%. Previously our laboratory has examined the effectiveness of intermittent post-exercise sauna bathing on heat tolerance and exercise performance in temperate conditions. Kirby et al. (2019) showed 3-weeks of intermittent post-exercise sauna bathing (10 sessions in total) induced HA adaptations (reduced HR, core body temperature and skin temperature, and increased sweat gland activity) and these changes were similar in females and males (Kirby et al., 2021). Post-exercise sauna bathing also improved markers of exercise performance in temperate conditions (i.e., $\dot{V}O_{2max}$, running speed at 4 mmol/L [La^-] and time to exhaustion (TTE)), more than endurance training alone (Kirby et al., 2019). On average post-exercise sauna bathing improved VO_{2max} by ~ 8%, running speed at 4 mmol/L [La^-] by ~ 4%, and time to exhaustion by ~ 12%. Conversely, 3-weeks of endurance training alone did not change markers of exercise performance in the control group. Peak core body temperature was the only physiological variable to show further decreases (− 0.1 °C) after 7-weeks of post-exercise sauna bathing (other physiological variables $p > 0.05$) and the drop-out rate during these 7-weeks highlights the difficulty in implementing a long-term HA protocol into a competitive endurance runners' usual training programme (Kirby et al., 2021).

The findings from previous studies supports recommendations to use post-exercise sauna bathing as opposed to active HA. Our laboratory has previously found this sauna protocol to be a practical and realistic method to bring about HA adaptations and improve the performance of high-level athletes. It brings about advantageous physiological and performance responses by inducing thermoregulatory benefits along with considerable heat protective effects, in a less time-consuming period (Casadio et al., 2016). Thus, this method appears to be an effective training and conditioning technique and ergogenic aid that can be added into an athlete's training program. Unlike more demanding methods like exercising in

hot climates or heat chambers, it can be easily integrated into daily routines, does not require specialised equipment or outdoor conditions, and provides consistent, controlled heat exposure (Periard, Racinais and Sawka, 2015). However, the decay rate following sauna-induced HA is unclear and therefore, requires more research immediately after or during a taper period.

2.3.6. Heat acclimation considerations:

The full effects of HA in athletic populations and the decay rate of HA remains unclear. Performance enhancements through HA can be influenced by a person's fitness, training status, age, and sex (Daanen et al. 2018). Therefore, it is important to consider how each athlete responds differently to HA, as well as adjusting the intensity depending on the individuals' training phase and time available (Taylor, 2006). For example, Benjamin et al. (2019) found highly trained individuals to have larger HA adaptations. It has also been suggested that some HA protocols can impact athletes preparations for major competitions, particularly disrupting final preparations (i.e., taper) (Mujika, 1998). This is because HA protocols have to be incorporated into the athletes usual training regime which causes large interferences (Saunders et al., 2019). Tapering is a period of time in which training load is progressively reduced. It occurs after a period of hard training and before a competition. The aim of a tapering period is to enhance performance by reducing the physiological and psychological stress experienced during daily training (Mujika & Padilla, 2003). Therefore, for HA to not hinder this process, it should either complement or be carried out before the taper period. Many athletes today are having to compete in different climates across the world, normally within a short time frame, therefore this knowledge is particularly important (Garrett et al., 2009).

2.3.7. *Measuring successful HA and HAD – HTT:*

A heat tolerance test (HTT) involves a controlled exposure to an exercise-heat stress and is used as a method to identify individuals' tolerance/intolerance to heat by comparing a temporary and permanent state of heat susceptibility (Mee et al., 2015). Typically HTTs involve exercising at a fixed work rate. Throughout the test, rectal temperature and HR are typically monitored, and sweat rate is calculated (Moran et al., 2007). To make sure no HA effect is elicited during testing, HTTs should not be repeated within 1-week (Barnett and Maughan, 1993). HTT can vary in duration, modality and intensity. Previous studies have used durations ranging from 30-120-minutes, different exercise modalities (including walking, running and cycling) and ambient conditions of 40 °C and 40% RH (Moran et al., 2004; 2007; Mee et al., 2015; Epstein et al., 1983). For example, a HTT involving 120-min walking on a treadmill at a pace of 5 km/h and a 2% gradient in ambient conditions of 40 °C and 40% RH, was developed to evaluate whether it was safe for military personnels to return to duty if they experienced exertional heat illnesses (Moran et al., 2004). Mee et al. (2015) examined the repeatability of a HTT protocol that involved participants running on a treadmill inside an environmental chamber for 30-minutes at a constant workload of 9km/h at 2% incline. Conditions for the HTT were set at an ambient room temperature of 40°C and 40% RH. Furthermore, the study population included both male (n=8) and female (n=8) participants who were of a relatively similar age, body composition and fitness. It was found that there were small differences between repeat HTTs, and low within person variation of key HTT markers such as peak rectal temperature (Intra-class correlation coefficient (ICC)=0.93, typical error of measure (TEM)=0.13 °C), peak skin temperature (ICC=0.79, TEM=0.30 °C), peak HR (ICC=0.99, TEM=2 beats min^{-1}), and sweat rate (ICC=0.95, TEM=162 g h^{-1}). Thus, the authors concluded that this type of HTT had a strong reliability when measuring physiological strain in the heat (Mee et al., 2015). Standardised

HTT procedures before Mee et al. (2015) did not use running as their mode of exercise. For some athletic populations (e.g. runners, team-sport), a 30-min running HTT is more applicable and easier to execute than longer walking HTTs or cycling HTTs.

2.4. Decay

HA is transient and the adaptations and benefits gained from HA gradually disappear if that exposure to the hot environment is removed and the heat stimulus is not maintained. Therefore, HA decay is the change in physiological adaptations after the cessation of HA (Daanen et al., 2018). Although there is a large amount of research on HA, comparatively little is known about HA decay. The time necessary to reach full decay and the characteristics defining this are still unclear.

HA decay has been shown to not be as rapid as its induction. Specifically, although you can induce adaptations within 5-10-days of HA, some adaptations (e.g. HR and core body temperature reductions) have been observed to be partially retained even up to 5-to-7-weeks after stopping the HA stimulus (Malgoyre et al., 2020). However, previous studies show different HA decay rates. For example, Pandolf (1998) showed when HA was not maintained via continued repeated exercise-heat exposures, HA benefits were retained for ~1-week before there was a gradual decay, with approximately 75% of adaptation gains lost by ~3-weeks. Garret et al. (2011) however, found 3-weeks following cessation of heat exposure characteristic HA adaptations had returned to pre-HA values. Weller et al. (2007), looked at a longer-term HA protocol and found the rate of decay for most physiological adaptations was approximately 26 days. More recent literature suggests that ~2.5% of adaptations are lost for every day an individual is not exposed to the heat (Daanen et al., 2018). When looking at the HA physiological responses, cardiovascular adaptations appear to be the first heat adaptations to occur and also seem to be the first to decay (Garrett et al., 2011). The first signs of decay

in these cardiovascular adaptations (e.g. reduction in HR) can be seen ~1-week post HA (Garret et al., 2011). However, physiological adaptations such as core body temperature, sudomotor habituation and sweat efficiency have a slower decay rate and appear to start decaying at approximately 14-days from the final heat exposure (Garrett et al., 2009; Weller et al., 2007; Daanen et al., 2018). This delayed decay rate for these HA adaptations is thought to be because they take longer to develop (Daanen et al., 2018).

Reasons for the limited knowledge on HA decay can be explained by large inter-study variability, likely due to the diversity of HA protocols and methods previous studies have used. For example, differences in the type of heat exposure used for HA, the training status of participants, as well as the number and duration of acclimation sessions may all affect HA decay (Garrett et al., 2011; Daanen et al., 2018). In particular, there appears to be a difference in decay rates following short-term vs long-term HA protocols (Daanen et al., 2018). Previous studies have shown that HA adaptations following short-term, active HA adaptations decay quickly and disappear within 2-weeks (Garrett et al., 2009; Williams et al., 1967; Neal et al., 2016). For example, Garrett et al., 2009 found a 5-consecutive day controlled hyperthermia HA protocol (T_a = 39.5°C, 60% RH) induced HA adaptations that persisted for 1-week but were not retained 2-weeks following HA. Consequently, in order to reduce the risk of an athlete compromising their pre-competition training, it has been recommended that the athlete should not complete a short-term heat acclimation more than ~1-week before moving to a heat stressful environment (Garret et al., 2009). Interestingly, Zurawlew et al. (2019) found HA adaptations following 6-days of post-exercise HWI, appear to be retained for at least 2-weeks after stopping HA, however. This 2-week decay period appears to align more closely with the decay period following medium-term protocols (Moss et al., 2022) and could appear favourable compared to other short-term active HA if it induces adaptations similar to those of active HA protocols (Daanen et al., 2018). Despite

this, long-term HA protocols have been widely known to cause more complete adaptations, and subsequently HA adaptations are maintained for longer before HA decay occurs (Daanen et al., 2018; Garret et al., 2009).

Another reason for the conflicting evidence in the literature is that it is difficult to standardise physical activity and/or environmental conditions during a decay period, which further challenges identifying a universal decay rate for HA adaptations following HA. The interval between HTT is another experimental design issue that can confound decay outcomes. This is because if there is not a large enough gap between HTTs, participants benefit from this extra heat stimulus that helps maintain HA adaptations (Barnett and Maughan, 1993). Both Saat et al. (2005) and Pandolf et al. (1977) had this issue as during the decay period they performed heat-stress tests 4-and 3-days, after HA, respectively. Therefore, it is recommended that HTTs should be performed at least 1-week apart (Barnett and Maughan, 1993). Furthermore, some previous studies are also limited by small sample sizes, incomplete HA and use of measurements with low reliability (Pandolf, 1998). Lastly, very few studies have assessed decay after 25 days (Malgoyre et al., 2020) and measure heat re-acclimation too early meaning heat adaptations are retained before complete decay (Daanen et al, 2018).

Due to the different findings and limited knowledge on how long HA adaptations are retained it is difficult to know how far in advance HA should be completed before athletic competitions. Tyler et al. (2016) suggested the believed optimal time to acclimate in the heat before a major competition is 2-weeks. To attenuate HA decay, exercise training in temperate environmental conditions during the decay period appears to help partially maintain HA adaptations, with further heat stress stimuli (such as hot baths) during decay also supporting the maintenance of HA adaptations (Daanen et al. 2018). However, more knowledge on the HA decay time course is required to give further answers to the length of time individuals

retain HA adaptations before needing to re-acclimate. Discovering this information could provide answers around avoiding HA decay before competition, ultimately improving athletes' performance and ensuring they retain their heat tolerance (Daanen et al, 2018).

2.5. HA and HAD effects on Core body temperature

As mentioned previously, an overall reduced resting and exercising core body temperature is one physiological HA adaptation (Periard et al., 2015). The majority of core body temperature adaptations appear to occur within 7-days of HA (Tyler et al., 2016). Previous studies have reported averages of 0.3°C reductions in resting core body temperature and these differences can be influenced by different HA protocols and climatic conditions (Höfler et al., 1969; Cotter et al., 1997; Patterson et al., 2004). For example, Patterson et al. (2004), found 3-weeks of HA using the controlled-hyperthermia technique, in which participants cycled for 90-mins per day, 6 days per week (40°C, 60% RH) reduced resting core body temperature by 0.32°C. Similarly, Buono, Heaney & Canine (1998) found a 0.3°C decrease in resting core body temperature following a 7-day HA protocol in which participants exercised for 2-hours per day in a hot and humid environment (35°C, 75% RH). With a decreased resting core body temperature, individuals have a great capacity to store heat as their 'heat sink' is larger (Sawka et al., 2011). HA-related reductions in resting core body temperature are also partially responsible for reducing core body temperature during and at the end of exercise (Tyler et al., 2016). For example, Buono, Heaney & Canine (1998) found core body temperature decreased by 0.6°C from 38.9 to 38.3°C post 7-days of HA at the end of 2-hours of exercise heat stress. Similarly, Shvartz et al. (1979) reported a 0.6°C decrease in core body temperature at the end of 2-hours of bicycle ergometer exercise heat stress (at 50% VO_{2max}) following 8 days of HA. Whereas Kirby et al. (2020) found 30-mins post-exercise sauna bathing reduced peak rectal temperature by 0.2°C during a 30-min HTTs.

A lower resting core body temperatures combined with a slower rate of rise, also means individuals can exercise for longer as it takes longer for them to reach an uncomfortable core body temperature (Sawka et al., 2011). This means HA-induced core body temperature changes are a potent adaptation that allows individuals to better manage heat stress.

Overall, exercise performance and capacity can be improved by reductions in core body temperature (Tyler, Sunderland, & Cheung, 2015). Therefore, maintaining core body temperature HA adaptations is important. The number/length of the heat exposure (short term vs long term HA) does not appear to effect the rate of core body temperature decay (short term vs long term HA) (Daanen et al., 2018). However, greater heat exposure duration seems to decrease core body temperature decay adaptations (Daanen et al., 2018). For example, Daanen et al. (2018) found 90-mins of daily exposure compared to 60-mins at a WBGT of 32 °C reduced core body temperature HA decay from 45 to 9%. Core body temperature has been reported to have a lower decay rate than other HA adaptations such as HR and mean sweat rate (Armstrong and Maresh 1991). Many studies show core body temperature HA-adaptations start to decay around 1-week after individuals are no longer exposed to heat but can still be retained for at least 2-weeks (Poirier et al., 2015; Zurawlew et al., 2019; Flouris et al., 2014). For example, mean percentage losses of core body temperature ranged from 13% to 25% after 6/7 days of decay and 18%–35% after 12–14 days of decay (Pandolf 1977, Saat et al., 2005 and Williams et al., 1967). Furthermore, a meta-analysis by Daanen et al. (2018) found core body temperature HA-adaptations had a decay rate of approximately 2.6% per day. Poirier et al. (2015) found core body temperature decayed ~33% after 14-days of no heat exposure following 90-min cycling (40°C, 20% RH) HA, with 75% decay after 28 days. Furthermore, Flouris et al. (2014) found reductions in core body temperature persisted for at least 2-weeks after completing a 14-day HA protocol comprising of 90-min cycling (40 °C; ~20 % RH), with only a 26% loss across this period. Lastly, Zurawlew et al. (2019) found

post-exercise HWI could retain core body temperature adaptations for at least 2-weeks. Core body temperature reductions from pre to 2-week decay were -0.36 ± 0.25°C at rest, -0.26 ± 0.24°C at sweating onset, and -0.36 ± 0.37°C at end-exercise. This is an important finding for the purpose of this study as it provides evidence that a post-exercise passive heat strategy, such as post-exercise sauna bathing is able to retain core body temperature adaptations for at least 2-weeks.

2.6. HA and HAD effects on Heart Rate

Another physiological marker of HA is a lower HR during exposure to the heat (Periard et al., 2015). As with core body temperature adaptations, the majority of HR adaptations appear to occur within 7-days of HA (Tyler et al., 2016). HR reduction is the first adaptation to occur from HA and develops in only 4 to 5 days (Sawka et al., 2011). Lorenzo et al. (2010) found peak HR during exercise to significantly reduce by 15 beats/min after a 10-consecutive day active HA protocol. Whilst, Weller et al. (2007) found HR to reduce by ~24 beats/min after a 10-consecutive day, active, controlled hyperthermia HA protocol. Pandolf et al. (1977) found HR to reduce by ~35 beats/min after a 9-consecutive day active HA protocol and finally, Kirby et al., 2020 found HR to reduce by 11 beats/min after 3-weeks of intermittent 30-mins post-exercise sauna bathing sessions. Some studies have proposed that exercising HR reductions following HA is either caused by increased parasympathetic activity, an increase in sympathetic tone following HA or alterations between sympathetic and parasympathetic dominance at different phases of HA (Horowitz and Meiri 1993; Epstein et al. 2010; Frank et al. 2001). However, the mechanism behind this reduced exercising HR is still unclear. Other studies have suggested HA-mediated plasma volume expansion is a large component of the effects that HA has on HR response and therefore enhances cardiac efficiency (Horowitz, 2003; Senay et al., 1976). However, not all individuals experience an

expansion in plasma volume following Heat acclimation (Periard et al., 2015). A-mediated plasma volume expansion is mainly attributed to the buildup of a greater plasma protein mass (Horowitz, 2003). This increase in plasma volume has two key benefits. Firstly it increases venous return and ventricular preload, reducing cardiac stain. Secondly, an increase total blood volume supports thermoregulatory heat loss responses (Sawka et al., 2011).

As mentioned previously, maintenance of this decrease in HR is important as it decreases physiological strain, allows an athlete to cope better when exercising in a hot environment, and minimises the occurrence of some forms of heat illness (Armstrong & Maresh, 1991; Périard et al., 2016). Studies in the literature show HR adaptations are retained for 1-2 weeks post HA. Garret et al. (2009) found after 90-min exercise (146 ± 21 beats/min) HR was reduced by 13 beats/min following 5-consecutive days of active HA, with this HA adaptation maintained after 1-week of decay but not after two (reduced by 9 beats/min) or three (reduced 6 beats/min) weeks of decay. Weller et al. (2007) showed no significant decay in exercising HR 12 and 26 days post HA. In a meta-analysis, Daanen et al. (2018) showed 2-weeks post HA, there was ~35% decay in exercising HR. It has been proposed that HR adaptations develop most rapidly during HA and decay the quickest (Pandolf et al., 1977; Williams et al., 1967). Saat et al. (2005) reported greater losses in HR HA adaptations than core body temperature adaptations, with 29% and 24% decay rates (on day 21), respectively. Pandolf (1998) also reported that within 2-weeks of decay up to 85% of HR adaptations are lost, compared to only 35% of core body temperature HA adaptations. When again looking at the Zurawlew et al. (2019) study which used a post-exercise HWI HA method, end-exercise HR adaptations were retained for at least 2 weeks during decay (−14 ± 10 beats/min reductions from Pre to 2-weeks decay). Thus, with HR adaptations appearing to decay the quickest after these traditional HA protocols, potentially causing significant consequences for an individual's physical performance, it is important to

investigate the effect of post-exercise sauna bathing on decay rate and whether HR adaptations can be retained for longer.

2.7. HA and HAD effects on sweat loss

During exercise in hot environments, heat loss responses such as sweat loss are essential in the regulation of core body and skin temperatures. Many previous studies have found HA increases the core body temperature onset threshold and max capacity of sweat responses (Periard et al., 2015). For example, Nielsen et al. (1997) found a 26% increase in whole body sweat rate after repeated exposure to exercise in the heat (35°C and 87% RH) for 8–13 consecutive days. Whilst Klous et al. (2020) found 10 consecutive days of active controlled hyperthermia HA (33 °C and 65% RH) increased local sweat rate from the eccrine sweat glands (arm: +58%, $P < 0.001$; back: +36%, $P < 0.05$) as shown by the significant decreases in sweat sodium, chloride, and lactate concentrations. Finally, Weller et al. (2007) found a HA-induced increase in evaporative cooling was supported by the ~1.2°C decrease in skin temperature across HA. However, HA has not always been found to increase sweat rate. For example, no significant changes were found by Zurawlew et al. (2019) in the drive for sweating onset, sweating sensitivity and whole body sweat rate after 6-days of post-exercise HWI. Furthermore, Pandolf et al. (1977) observed a decrease of ~0.8°C in core body temperature, but no change in body mass loss following HA. Thus, the same total sweat loss was achieved at a lower body core temperature in this study following HA. Changes in body mass are the primary method for measuring sweat loss (Gagnon & Crandall, 2018) but other sweat adaptations, including reductions in sweating threshold and a more even distribution of sweat over the skin surface, can go unnoticed through this method (Fox et al. 1964). Therefore, this could be a reason for the varied results in sweat rate HA adaptations. Variations in sweat rate HA adaptations could also be due to either core body temperature

reductions during HA or the fact that longer HA periods are associated with more stable sweat rate adaptations (Daanen et al., 2018).

When then looking at the maintenance of these physiological adaptations, it has been shown that improvements in heat dissipation decay gradually. Sweat rate decay appears to be effected by the amount of HA days, with slower decay occurring after longer HA protocols (Daanen et al., 2018). HA protocols that use hotter and dryer air conditions for their heat exposures also appear to better maintain sweat rate adaptations during decay (Poirer et al., 2015). However, the decay rate of sweat loss and sweat rate improvements are slower than the rate at which these adaptations are gained (Armstrong & Maresh 1991). Using direct calorimetry, Poirer et al. (2015) found the average percentage loss for evaporative heat loss was ~31% after 7 days and ~33% after 14 days of decay, therefore, across a 14-day decay period there is only ~4% reduction in evaporative heat loss rate. Concurrently, the cumulative change in body heat in this study was still 12% lower than the change measured before HA occurred. Poirier et al. (2015) found that the gradual decrease in evaporative heat loss and the gradual decrease in the thermosensitivity of sweating caused this progressively greater heat storage (day 21, +11%; day 28, +20%) during the decay period. Consequently, it appears from this particular study 7-days of HA were maintained after a week decay, suggesting that only 1-day of acclimation is lost for 1-day spent away from the heat. Comparatively, previous studies have reported that 6 to 14 days after HA mean percentage losses for local and whole-body sweat rates ranged from 54% to 79% (Garrett et al., 2009 and Williams et al., 1967). Added to this, Garret et al. (2009) found the time taken for more dilute sweat concentration and sweating onset to return to pre-HA values was around 3-weeks post HA exposure. Conversely, Duvnjak-Zaknich et al. (2018) found no differences in sweating measures (whole body sweat loss and sweat rate) following a 2-week decay period post HA. Overall, the decay

rate of sweat loss seems to vary substantially between studies and further research on particular HA methods needs to be conducted to clarify decay rates fully.

2.8. HA and HAD effects on thermal sensation, thermal comfort and ratings of perceived exertion

An individual's rating of thermal sensation, thermal comfort and perceived exertion measure an individual's perception of exercise and thermoregulatory stress. The Borg scale (Borg, 1980) is used to measure a person's rating of perceived exertion (RPE), where the exertion is reported on a scale of 6 to 20 (a score of 6 being the lowest and a score of 20 being maximal exertion). Thermal sensation and thermal comfort are most commonly measured using modified 13-point and 10-point scales, respectively (Gagge et al. 1967). Thermal comfort and thermal sensation are often measured with RPE (Periard et al., 2015). Thermoregulatory effector mechanisms such as sweating and skin blood flow mainly govern the discomfort felt in the heat which is affected either by air temperature, metabolism and skin and rectal temperatures (Gagge, Stolwijk & Hardy 1967). RPE is very tightly correlated to HR, core body temperature and oxygen consumption (Baker et al., 2000). This is because increases in core body temperature and cardiovascular drift during exercise hyperthermia and passive heating appear to correlate with RPE (Galloway and Maughan, 1997**;** Gonzalez-Alonso et al. 1998). Therefore, RPE should also correlate with HA-related decreases in sweating, skin blood flow, HR and core body temperature as well as attenuated reductions in $\dot{V}O_{2max}$ following HA (Periard et al., 2015).

Indeed, HA has been shown to reduce thermal sensation, thermal discomfort and RPE, although data is limited (Tyler et al., 2016). Specifically, thermal comfort in the heat has been shown to improve with repeat exposure to hot environments (Petersen et al., 2010; Gonzalez & Gagge, 1976; De Dear & Schiller Brager, 2001). For example, Petersen et al.

(2010) found participants to perceive exercise in the heat more comfortable after four high intensity (30–45 min) HA sessions on consecutive days at ~30°C and 60% RH using a cycle ergometer. HA has also been shown to have a moderate effect on RPE during exercise, reducing values by ~1 (Neal et al., 2015; Armstrong et al., 2005). Lastly, HA seems to have no effect on resting thermal sensation but can have a small effect on thermal sensation during exercise, reducing thermal sensation by ~0.9 (Neal et al., 2015; Racinais et al., 2014).

HA decay studies have found that alongside the maintenance of key HA adaptations such as reduced core body temperature and HR, perceptual variables such as RPE and thermal sensation are retained. Casadio et al. (2016) found thermal sensation and RPE decreased alongside core body temperature and HR after 5-consecutive days of HA (60-min per day in 35°C, 60% RH). Further, these HA adaptations were retained 2-weeks after the HA period. Similarly, Pryor et al. (2019) found perceptual measures did not significantly decay after 25-days of decay and remained lower than Pre HA values. A more recent study by Duvnjak-Zaknich et al. (2019) found no significant differences in subjective measures, including RPE and thermal sensation, at 2- and 3-weeks post HA as compared to immediately after the HA period. Finally, Zurawlew et al. (2019) found HA (through the method of HWI) elicited adaptations in RPE and thermal sensation that were retained for at least 2-weeks. Overall, it appears traditional HA protocols can sustain reductions in thermal sensation, thermal discomfort and RPE for at least 2-weeks similar to other HA adaptations, indicating less perceived strain and discomfort following HA.

2.9. Plasma volume changes with HA and HAD

Plasma volume expansion is another physiological adaptation that occurs with HA (Lorenzo et al., 2010). One benefit of plasma volume expansion is that it increases the specific heat capacity of blood, improving heat transfer from the core to the skin through

improved efficiency of skin blood flow responses (Periard et al., 2016; Sawka et al., 2011). Plasma volume expansion also supports cardiovascular stability through increased cardiac and vascular filling during exercise in the heat (Senay et al., 1976; Sawka et al., 2011). Following HA, plasma volume expansion has been shown to occur within as little as 3-days of heat exposure (Sawka and Coyle, 1999), with 5-days of HA being optimal to bring about the greatest adaptations in individuals who are fully hydrated (Harrison, 1985). Furthermore, during acute HA, plasma volume expansion is generally 4–15%, but has been found to range from 3% to 27% (Périard et al., 2015). For example, Lorenzo et al. (200) found a 6.5 ± 1.5% increase in plasma volume after 10 consecutive days of active HA (~50% $\dot{V}O_{2max}$ in 40°C). However, Stanley et al. (2015) found a larger increase of 17.8% in peak plasma volume expansion after only four exposures of 30-mins post-exercise sauna bathing per day. The reasons for the variability in plasma volume expansion found amongst studies is based on the HA day, a persons' hydration status when measured, skin temperature and whether the individual rested or exercising (Sawka et al., 1983; Harrison, 1985; Kenefick et al., 2014). Fitness levels of athletes may also contribute as a factor as endurance-trained athletes appear to already display an expanded plasma volume.

However, plasma volume expansion has been shown to be transient. When looking at short-term aerobic exercise alone, within 2-to-3 days of stopping the exercise, plasma volume expansion appears to decrease to baseline levels (Lamb and Murray, 1988). When then looking at HA decay, after an 8-day HA protocol followed by little activity for 7-days, plasma volume expansion decreases to baseline levels within 1-week after cessation (Convertino et al., 1980). Garrett et al. (2009) concurred with the above findings, showing that after 5-consecutive days of active HA, plasma volume expansion returns to pre-acclimation baseline levels 1-week following HA. Therefore, as plasma volume expansion is important for improving heat transfer from the core to the skin, it could give some

explanations to why HA adaptations such as increased core body ad skin temperatures and sweat loss are not maintained for long periods after stopping HA.

2.10. Participant Choice: - Lack of HA data in females

To date, the majority of HA studies have gathered data mainly from male participants. This means our understanding of exercising in the heat and HA adaptations are largely based on male data and female populations are understudied (Wickham, Wallace, & Cheung, 2021). A recent meta-analysis looking at the physiological (Tyler et al., 2016) and performance adaptations (Benjamin et al., 2019) following HA included a total of only 7% (76/1056) and 3% (3/101) female participants, respectively. Over the years, female participation in sport has significantly increased, with females making up ~49% of athletes competing in the 2021 Tokyo Olympics (Xie, 2021). This means that women are similarly exposed to the hot challenging environments during training and competition and therefore, this signifies the importance of research focusing more on female data so that they are not at a disadvantage in these major competitions (Hollerbach et al. 2017; Corbett et al. 2020).

The sparsity of HA data from women is due to a number of reasons. Firstly, menstrual cycle and associated fluctuations in female sex hormones have been shown to influence thermoregulatory responses (Pivarnik et al., 1992; Kolka et al., 2000; Stephenson & Kolka, 1999; Tenaglia et al., 1999; Janse et al., 2012). During follicular phase, oestrogen levels increase and this has been correlated with improved mood, higher pain tolerance and increased endurance (Glowing, 2023). Whereas, during the luteal phase, progesterone levels increase and this results in symptoms such as bloating, mood swings and fatigue (Glowing, 2023). When experimental protocols are conducted across different phases of the menstrual cycle, this can have an impact on thermoregulatory results and for heat tolerance or performance tests, responses in women could be misrepresented due to menstrual cycle

influences on thermoregulatory process and heat storage (Giersch et al., 2020). Specifically, core body temperature seems to be significantly affected during these phases. Resting core body temperatures fluctuates 0.5-0.8°C, over the menstrual cycle course (Baker & Driver, 2007; Stephenson & Kolka, 1999). The threshold for sweating and blood flow is increased in the luteal phase compared to the follicular phase which causes the body to heat up before the effector responses are activated (Kuwahara et al., 2005). This means to reach core body temperatures of ~38.5 °C less metabolic heat production is necessary (Wickham, Wallace, & Cheung, 2021). A study by Giersch et al. (2020) showed significantly greater initial and post-exercise core body temperature when women participants were in the luteal phase compared to the follicular phase. Similarly studies by Wickham, Wallace, & Cheung (2021) and Kuwahara et al. (2005) showed that when women were in the mid luteal phase there is an increase of ~0.3 to 0.5°C in their core body temperature setpoint due to the rising circulating progesterone levels during the luteal phase. Therefore, it is important to control the phases of the menstrual cycle during an intervention. However, arranging experimental testing schedules can be difficult so most studies have tested women in different phases of their menstrual cycle (Burrows & Bird, 2000). This is due to the length and nature of traditional HA protocols, as the normal menstrual cycle can vary between 22 to 36 days. Additionally, some women have irregular cycles (or commonly for endurance athletes, they can be amenorrheic; Bennell et al., 1997, Dale et al., 1979). Despite this, both Kirby et al. (2019) and Mee et al. (2018) controlled menstrual cycle phases in their study. Mee et al. (2018) used two 5-day HA interventions (controlled hyperthermia $T_{re} \geq 38.5$ °C) both which females performed during the follicular phase of their cycle. Similarly, to make sure female participants were tested during the same menstrual cycle phase, Kirby et al. (2021) conducted experimental trials (following 3-and 7-weeks) ~28 days apart. Overall, due to the menstrual

cycle impact, research including women participants can show many inconsistent results on thermoregulation.

The use of hormonal contraception in female participants should also be considered as this again seems to influence thermoregulatory responses. Hormonal contraceptives are widely used with approximately half of elite level female athletes using them (Martin et al. 2018). Charkoudian & Stachenfeld, (2014) show that progestin-only contraception administration seems to increase resting core body temperature and delay the core body temperature threshold for thermoregulatory peripheral effector responses and the onset of sweating during exercise, similarly to what happens during the mid-luteal phase (Lei et al. 2017, 2019; Giersch et al. 2020). These changes are reduced with a combined ethinyl estradiol-progestin contraception treatment (Charkoudian & Stachenfeld, 2014). Overall, there are minimal studies on how hormonal contraceptives affects heat tolerance and performance in the heat, let alone HA. Thus, more research is needed to confirm these findings (Martin et al. 2018).

Future studies need to address the methodological challenges, exploring sex differences in the responses to HA. This would help develop sex-specific HA protocols to optimise female athlete's performance.

When therefore looking specifically at the difference between biological sex during HA decay, there could be the suggestion females potentially lose HA adaptations faster. Females appear to take longer to achieve HA adaptations and in order to achieve the same magnitude of physiological adaptations as males, females appear to require a larger thermal stress (Mee et al., 2015; Mee et al., 2016). Therefore, it could be suggested that in order for females to also sustain these adaptations for longer they will again need greater thermal stress for this reason too. However, there are a very limited amount of decay studies that have used female participants due to difficulty in controlling the menstrual cycle across the decay period

(especially if the decay period is around 1-2 weeks, as mentioned before typical cycles range from 22 to 36 days). Therefore, questions around female responses during decay remain unknown and future studies need to focus on this in particular.

2.11. Participant Choice: - Athletes vs General population

Athletes are more likely to be exposed to heat stress than the general population because they often are required to travel to train and compete in hot environments, which determines that athletes may be more susceptible to heat-related illnesses (Price, 2016). HA can reduce the risk of hyperthermia and the detrimental performance effects of a hot environments (Guy et al., 2015). Therefore, HA adaptation and an improved capacity to tolerate heat stress is particularly relevant to athletic populations and should be a fundamental preparatory step for athletes competing in a hot environment. Yet only 15% of athletes acclimated in 2015, before the Beijing athletics world championships (Périard et al, 2017). It has been suggested that until untrained individuals achieve an acceptable fitness level when training in cooler temperatures, they should avoid exercising in the heat (Adams et al., 1960). To prepare individuals who are untrained and unacclimated for the effects and conditions experienced in a hot environment, they should use high intensity hard interval workouts (Adams et al., 1960).

Previous research has indicated that individual training status (trained vs untrained) has an impact on the magnitude and timeframe of HA adaptations (Sawka et al., 2015; Daanen et al., 2018). However, it is up for debate as to which individuals experience greater HA benefits. Due to athletes having higher fitness levels and a higher aerobic capacity ($\dot{V}O_{2max} > 47$ mL/kg/min) than individuals with a lower $\dot{V}O_{2max}$ ($\dot{V}O_{2max} < 47$ mL/kg/min), they sweat more and dilate the skin faster for a smaller change in mean core body temperature (tolerating a higher core body temperature at the point of voluntary fatigue

during HA interventions) meaning they are more effective at losing heat to the environment and heat acclimate faster (Cramer & Jay 2014; Cheung & Sleivert, 2004; American College of Sports Medicine, 2013). Subsequently, they gain greater HA adaptations such as having a lower initial core body temperature (~0.2°C) coupled with a higher final core body temperature (~0.7°C) (Price, 2016; Cheung & Sleivert, 2004). Furthermore, athletes with a higher $\dot{V}O_{2max}$ appear to lose adaptations more slowly during decay (Armstrong, and Maresh, 1991; Mikkelsen et al., 2019). On the other hand, highly trained athletes participate in high quality and intense training in preparation for competitions. Due to their level of conditioning, highly trained athletes may appear to respond physiologically as if they already possess many HA adaptations (Taylor 2000). This means compared with untrained or moderately trained participants, highly trained athletes potentially have less HA adaptive potential. Interestingly, Corbett et al. (2018) found that a 10-day active controlled hyperthermia HA (40 °C, 50% RH) intervention (interspersed by three 60-min HTTs) did not result in inter-individual adaptive responses relating to factors such as VO_{2max} (inter-participant range of 3.49–5.05 L/min) that supposedly influences the adaptive response to heat. They found individuals who demonstrated a high, or low, adaptive response on one index of HA did not necessarily demonstrate a similar response across all HA indices. Therefore, further research is still required to fully answer the question as to whether trained individuals experience greater HA benefits compared to untrained individuals.

All studies included in Daanen et al. (2018) literature review and up until 2018, have studied healthy fit individuals and not looked at the general population and how they respond to the cessation of HA. Daanen et al. (2018) analysed several HA decay studies and all of these specific studies included participants with a $\dot{V}O_{2max}$ score of 39 mL/kg/min and above. The age range of participants taking part in all decay studies analysed by Daanen et al. (2018) was 20-29 years old and guidelines suggest fit good males of this age have a $\dot{V}O_{2max}$ score of

46.5-52.4 mL/kg/min and fit good females have an average score of 37.0-41.0 mL/kg/min (American College of Sports Medicine, 2013). Since this meta-analysis in 2018, there have been very limited studies on HA decay and none of which have studied HA decay on the general population. Therefore, in order for the general population to benefit from HA, more research is needed on the decay rate of this population. Further research will also allow this comparison between both populations, potentially establishing the individual differences and how these can be controlled to increase the maintenance of HA adaptations.

2.12. Aims and Hypothesis:

The main aim of this study was to then assess whether a decay period of 2-weeks, in which no HA stimulus was applied but training is maintained, had a detrimental effect on adaptations gained from 3-weeks of post-exercise sauna bathing. The subsidiary aim was to see if we could repeat findings our laboratory has previously published (Kirby et al, 2020; 2012), and examine if 3-weeks of post-exercise sauna bathing effectively heat acclimated endurance athletes.

Based on a review of the relevant literature the following hypothesis was established. It was hypothesised that 3-weeks post-exercise sauna bathing would induce HA adaptations including decreased core temperature during exercise, increased sweat losses and consequently lower skin temperatures during exercise, reduced resting and exercising HR and improved perceptual responses (RPE, thermal comfort, thermal sensation) during exercise. These HA adaptations would then be partially lost (decay at a rate of ~2.5% per day; Daanen et al., 2018), after two weeks of endurance training without post-exercise sauna bathing, with core body temperature showing a slower rate of decay than HR and sweat loss.

3. Methods

3.1. Investigator Contributions

Part of the data set (n=8) reported in the current Msci thesis were collected in 2019-2020 and reported in N. Kirby's PhD thesis. The candidate A. Rex coordinated and led the data collection for the remaining cohort (n=11) in 2022.

A.R. contributions: Recruitment; leadership and coordination of data collection (n=11); data analysis and interpretation; and thesis write-up.

N.K. contributions: Study conception and design; recruitment, leadership and coordination of data collection (n=8).

All aspects of the study were overseen by project supervisors Dr Rebekah Lucas and Dr Sam Lucas.

3.2. Participants

Following ethical approval from the University of Birmingham Research Ethics Committee (ERN_19-18-0958), twenty-eight trained middle-distance and cross-country runners were recruited from the University of Birmingham's athletics club to take part in this study. Due to injury only nineteen athletes completed the HTT at 3-Weeks and completed the full study. The study was carried out in accordance with the Declaration of Helsinki and all participants were informed of the experimental procedures and possible risks involved in the study before providing written consent. All participants were healthy as defined by the completed General Health Questionnaire and had an excellent level of cardiorespiratory fitness as defined by their $\dot{V}O_{2max}$ ($\dot{V}O_{2max}$ > 51mL/kg/min) (American College of Sports Medicine, 2018). Female participants were asked to complete a menstrual cycle questionnaire at the commence of the study to track the menstrual cycle phase they were in for each HTT.

Throughout the protocol, participants were asked to keep a training diary (including session type, frequency, running distance, exercise duration, and session perceived exertion), which were later used to quantify and compare training load, during the sauna intervention and decay period. Training session RPE was measured using a 1–10 point scale (RPE1–10; 'very light session' to 'max effort session'). Participants took daily iron tablets (65g Ferrous sulphate) from 2 weeks prior to the first experimental trial until the completion of the protocol. This was to stop detrimental effects to performance and health through low iron status, especially in the active female athletes (DellaValle, 2013). For participant characteristics, see table 1.

Table 1: Participant characteristics

Characteristic	(Mean ± SD)
N	19
Females: Males	8:11
Age (years)	20 ± 1
Height (centimetres)	173 ± 9
Body mass (kg)	64.4 ± 7.8
BMI (kg/m²)	21.5 ± 2.7
$\dot{V}O_{2max}$ (ml kg−1 min−1)	60.0 ± 6.0

3.3. Experimental Design

A series of experimental trials were completed within a 2-week timeframe at baseline (Pre), following a 3-week intervention (Post) that consisted of normal exercise training plus 30-min post-exercise sauna bathing (in ambient air temperatures (85–94°C) 3 ± 1 times per week, and following 5-weeks after a 2-week decay period where participants continued with

their normal exercise but did not sauna (decay). Experimental trials included a running heat tolerance test (HTT) and a temperate exercise test (consisting of a lactate profile test to determine running speed at 4 mmol/L [La−] and a $\dot{V}O_{2max}$ test). The temperate exercise test was conducted at Pre only. The period in which testing took place avoided the warmer summer season, taking place from the months October to December, ensuring natural heat acclimatisation would not interfere with the study. For experimental design, see Fig. 3.

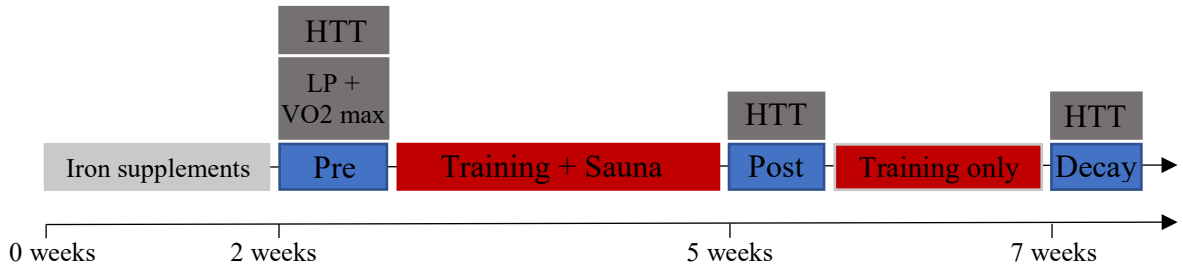


Fig. 3 Schematic of the experimental design. At baseline (Pre) participants completed temperate (18°C) exercise tests consisting of lactate profile (LP) and maximal aerobic capacity ($\dot{V}O_{2max}$) tests, and a running heat tolerance test (HTT; 30-min, 9km h−1/2% gradient, 40°C/40%RH). Participants then completed a 3-week post-exercise sauna bathing intervention. Additional HTT were completed post intervention at 3-Weeks (Post) and post-intervention 5-Weeks (following 2-weeks decay).

3.4. Post-Exercise Sauna Bathing

Heat acclimation was in the form of sauna bathing at the University of Birmingham Sport and Fitness Centre. Participants entered the sauna (85–94 °C, 5–10% RH at ~1m; iButton Hygrochron Logger, Maxim Integrated, California, USA) within ~5 min of stopping exercise. Sauna bathing typically followed low-intensity, continuous exercise training sessions (i.e., 'easy runs', 'long runs' or 'easy bikes') 3-4 times per week, aiming for an accumulated total of ~9 sessions over 3-weeks. Participants were advised to distribute their sauna exposures as evenly as possible across the 3-weeks, however, this was not followed by some participants

due to the participants' personal and training schedules. Participants were asked to remain in the sauna for 30-min, as per previous investigations (Scoon et al. 2007; Stanley et al. 2015). However, participants were permitted to exit the sauna or take a break if they were too uncomfortably hot at any stage of the intervention. Some participants did require approximately a 1-min break, which was added to the end time so that participants completed the full 30-minutes if possible. In the sauna, participants sat upright and were allowed to drink water as often or as necessarily desired. On the participants first and final sauna visit, body mass (kg) and fluids consumed (g) were measured pre and post sauna to calculate sweat loss and sweat rate during the first and last sauna visits. Heart rate (HR), ratings of thermal sensation and thermal comfort were taken every 10-mins during sauna. Thermal sensation and thermal comfort were measured using modified 13-point and 10-point scales, respectively (Gagge et al. 1967). During all other sauna visits, peak HR was recorded during the final minute of sauna exposure. Participants were given Polar watches (Polar Electro, Kempele, Finland) to wear in the sauna and HR was recorded on the Polar Beat application (Polar Beat, Kempele, Finland).

3.5. Training logs

Participants were asked to complete a training log each week outlining their exercise activities, throughout the period of the study. Participants largely trained together and therefore undertook similar training sessions as they were part of the same athletics club *(*University of Birmingham Athletics Club*).*

Training sessions were categorised as:

- Cross-training – non-running exercises including cycling, swimming or strength and conditioning work.
- Easy run – any length run done at a very controlled and comfortable pace
- Long run – a run exceeding ~6 miles
- Tempo run – moderate to high intensity run around lactate threshold pace
- Interval – repeated high intensity efforts, including track-based and grass sessions

- Races – competitive running events

3.6. Running Heat Tolerance Test (HTT)

Participants performed HTTs on the same day of the week, at the same time of day (± 2h/± 1 day). Participants were asked to maintain an adequate level of hydration (≥ 700 mOsm kg^{-1}; Osmocheck, Vitech Scientific Ltd., West Sussex, United Kingdom; Sawka et al. 2007), as well as refrain from consumption of alcoholic beverages for 24-hours prior to each HTT, as well as having a full sleep the night. They were asked to recall the food they had eaten prior to HTT, and to repeat the same diet for each subsequent session. On arrival to the laboratory, participants emptied their bladder to provide a urine sample, which was analysed for osmolality. If urine osmolality was ≥ 700 mOsm/kg (Sawka et al. 2007), participants were required to drink before the HTT and fluid consumption was measured and factored into the calculation of body mass change. Participants then recorded their towel-dried, nude body mass (kg), using digital scales (Seca 877, Seca, Hamburg, Germany). A nude reading was necessary as clothing has the potential to retain moisture and affect body mass readings. This was done before and immediately after the HTT to estimate whole-body sweat loss. Participants were then instructed to insert a rectal thermistor (Mon-a-Therm, Covidien, Mansfield, MA, United States) at a minimum of 10cm past the rectal sphincter for the measurement of rectal temperature. Rectal thermistors were used for temperature measurement in this study as they have been widely used and are considered very accurate (Sawka et al., 2011). The disadvantages to this method however, are that there can sometimes be a lag as it is further away from the heart and the large muscle can also generate heat and effect the reading (Cheshire, 2016). Mean skin temperature was recorded using skin thermistors (Squirrel Thermal Couples, Grant Instruments, Cambridge, UK) attached to four sites on the same side of the body: chest (pectoralis major), arm (biceps brachii), thigh (rectus femoris), and lower leg (gastrocnemius lateral head). The equation ((0.3x chest) + (0.3x arm)

+ (0.2x thigh) + (0.2x calf)) was used to calculate mean skin temperature (Ramanathan, 1964). A heart rate monitor (Polar Electro, Kempele, Finland) was given to the participants to wear throughout the protocol to record HR. Once instrumented, participant sat and rested in a chair for 10 minutes to allow the normalisation of blood flow (Wythe et al., 2015), following this resting HR and resting rectal temperature were recorded. Participants wore socks, shoes, and either shorts (males) or shorts and a sports bra (females) during HTT. This was replicated for every HTT. The HTT was performed in an environmental chamber (TIS Services, Hampshire, United Kingdom) with a fan-generated airflow of ~4 ms^{-1}. Conditions in the chamber were set at an ambient room temperature of 40°C and 40% relative humidity (RH). Participants ran on a treadmill (H/P/Cosmos Quasar 4.0, H/P/Cosmos, Germany) at 9 kmh^{-1} and 2% gradient for 30-min. This protocol chosen in the current thesis was the same 30-minutes HTT protocol used by Mee et al. (2015). It was chosen due to the reliable findings from Mee et al. (2015) and the fact this study included all participants from the running club. Participants were permitted to listen to their choice of music whilst running in the chamber. During the HTT rectal temperature and skin temperature were logged continuously (Squirrel 2022 series, Eltek, Ltd., United Kingdom). Temperature readings were logged at 1-second intervals. HR was also recorded continuously, using the Polar Beat application. Perceptual measures, including ratings of perceived exertion (RPE), thermal comfort and thermal sensation, were also obtained every 10-mins of the HTT. Thermal sensation and thermal comfort were measured using the same scales used in the sauna sessions. RPE was measured using the 6–20 point Borg Scale (RPE6–20; Borg 1982). Drinking was not permitted during the HTT. This was to avoid any cooling or heating effect of water consumption. Participants were permitted to consume liquids and urinate after body mass was taken immediately after the HTT had finished. Internal heat stress was not only measured for applied research purposes but also to ensure the safety of participants and stop the risk of overheating. If rectal

temperature of the participant met or exceeded 39.5°C, for the safety of the participants the HTT would be terminated. However no participant reached this rectal temperature during a HTT. Participants were able to stop the HTT at any point if they wished, however all participants completed the full 30-minutes.

3.7. Lactate profile test:

The lactate profile test was performed in temperate conditions (air temperature ~18°C). Participants were again asked to maintain an adequate level of hydration, as well as refrain from consumption of alcoholic beverages for 24-hours prior to the test. Participants height and body mass were measured on entry to the laboratory. Participants completed a light 10-min warm-up plus drills before commencing the lactate profile test on the treadmill (H/P/Cosmos Quasar 4.0, H/P/Cosmos, Germany). Participants completed 4-min stages with 30-sec to 1-min stops between stages for the collection of a blood sample from an earlobe. The test began at 1% gradient (Jones and Doust, 1996) and a speed 4 km/h slower than participants' 5-km race pace personal best, and increasing by 1 km/h for each stage thereafter. 1 km/h speed increments were used as they were optimal at ensuring the lactate threshold (4.0mmol/L) was met and exceeded, without the test taking too long and fatigue and energy depletion having an effect on participant results (Jones et al., 2019). Participants were permitted to listen to their choice of music and were provided with fan-generated airflow to improve comfort during the test. The test was either terminated when [La−] exceeded 4 mmol/L, or when the participant refused to continue onto another stage, which occurred between stages 4–7. During the test, HR (H10, Polar) and respiratory gas exchange were continuously measured. Respiratory gas exchange was sampled breath-by-breath using open-circuit spirometry (Vyntus CPX, Jaeger, Wuerzberg, Germany). Measures of blood lactate concentration [La−] were immediately analysed from earlobe capillary blood samples using a

Biosen C-Line Lactate analyser (EKF Diagnostics, Penarth, UK), which was quality checked each day and calibrated every 60-min. Participants were permitted to listen to their choice of music and were provided with fan-generated airflow to improve comfort during the test. The test was either terminated when [La−] exceeded 4 mmol/L, or when the participant refused to continue onto another stage, which occurred between stages 4–7.

3.8. Maximal aerobic capacity ($\dot{V}O_{2max}$)

Approximately 10 min after completing the lactate profile test, participants performed a $\dot{V}O_{2max}$ test. The test started at 1% gradient and at the speed 2 km/h slower than the speed at which the participant had exceeded 4 mmol/L [La−]. Speed increased 1 km/h each minute, until participants reached the speed at which >4 mmol/L [La−] had been reached. Then the gradient was increased by 1% each minute until participants reached volitional exhaustion. Participants were able to listen to music and were given consistent and loud encouragement during the test. Data from the Vyntus was averaged by 1-min intervals at peak oxygen consumption. This was then used to calculate $\dot{V}O_{2max}$ by taking the highest data point.

3.9. Data analysis

Statistical analysis was performed using GraphPad analysis software (GraphPad Prism version 8.0.0 for Windows, GraphPad Software, San Diego, California USA [Computer Software]). Absolute data and within-subject changes are expressed as mean ± standard deviation (SD). For each analysis, $p < 0.05$ (*), $p < 0.01$ (**) and $p < 0.001$ (***) were considered to be statistically significant. For HTTs data, including core body temperature, skin temperature, HR, RPE, Thermal comfort, and Thermal sensation a two way repeated measures analysis of variance (ANOVA) was applied to compare time points within each HTT (rest, 10, 20, 30 minutes) and compare between intervention points (Pre to Post) and

(Pre, Post, Decay). If there was a significant main effect or interaction in the ANOVA, pairwise post-hoc comparisons were performed using Tukey's multiple comparisons test. Paired t-tests were used to compare physiological and perceptual responses, including peak HR, during the first and last sauna sessions. The Shapiro Wilk test was used to check for normality in all data at all time points. Data that failed the normality check (including sweat loss and sweat rate, thermal comfort and thermal sensation, during saunas and RPE during training sessions), were compared using nonparametric tests including a Mann–Whitney *U* test or a Friedman's test with post hoc analysis by Wilcoxon sign-rank tests. The Geisser-Greenhouse's correction was used if the assumption of sphericity was violated, otherwise no correction was assumed. HA decay was quantified using the formula developed by Williams et al. (1967) and modified by Pandolf et al. (1977) (Eq. 1):

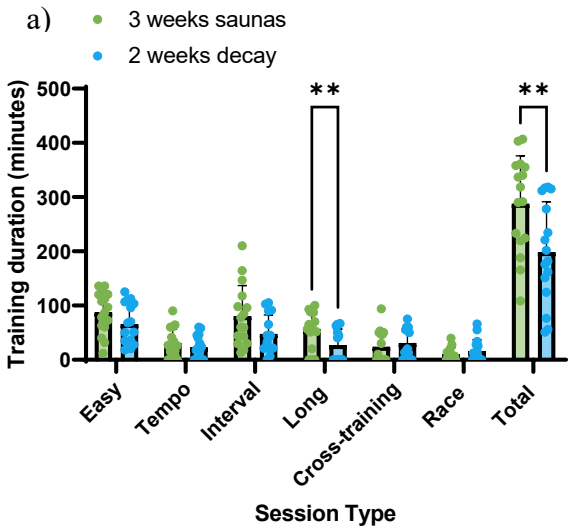
$$\boldsymbol{Decay = \frac{Acclimatized\ value - Value\ after\ decay}{Acclimatized\ value - Unacclimatized\ value}\ x\ 100}$$

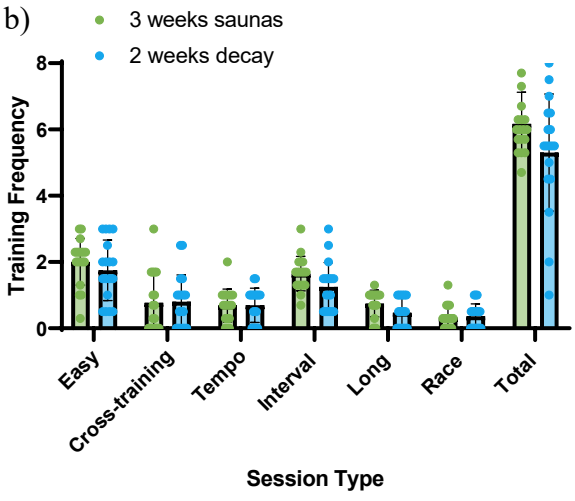
The linear relationship between training duration across the decay period/$\dot{V}O_{2max}$ max of participants and change in dependent variables from Post to Decay were assessed using Pearson correlation coefficient.

4. Results

4.1. Training

The number of training sessions per week across the 3-week sauna intervention and 2-week decay period, were not significantly different (6 ± 1 and 5 ± 2 respectively; $p = 0.38$). Average weekly training duration was higher during 3-week sauna intervention as compared to the decay period (288 ± 88 vs 199 ± 93 minutes respectively; $p < 0.01$). Training of participants was not different in type, frequency, or perceived exertion between the 3-week sauna intervention and 2-Week decay period (all $p > 0.05$; Fig. 4).





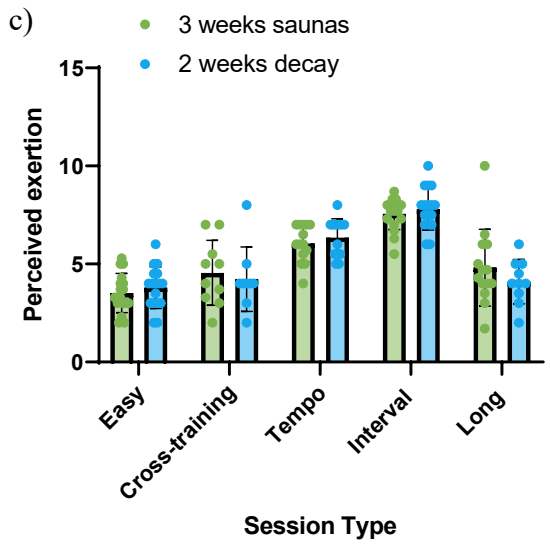


Fig 4. Participant's training volume (a) training duration, (b) training frequency, and c) perceived exertion during the 3week sauna intervention (light green) and the 2-week decay period with no sauna visits (light blue). Data presented as mean ± SD. Statistical differences between the two time periods are indicated by (**) $p<0.01$.

4.2. Menstrual Cycle

Six of the female participants reported normal menstrual cycles ranging between 24–37 days. Two female participants were using a form of hormonal contraception. For each female participant, the phase of their menstrual cycle during each HTT are displayed in table 2. Participant 12T was using a hormonal contraceptive coil and completed all tests in the active contractive-taking phase.

Table 2: Menstrual cycle phases of participants during all three HTT.

Participant	**Menstrual cycle phase**			**Oral contraceptives**
	Pre HTT	**Post HTT**	**Decay HTT**	
1D	Luteal phase	Ovulatory phase	Luteal phase	None

4D	Ovulatory phase	Follicular phase	Follicular phase	None
5D	Follicular phase	Follicular phase	Luteal phase	None
8D	Follicular phase	Follicular phase	Luteal phase	Hormonal coil
11D	Luteal phase	Luteal phase	Follicular phase	None
12T	N/A	N/A	N/A	Contraceptive coil
17T	Luteal phase	Luteal phase	Follicular phase	None
20T	Luteal phase	Luteal phase	Follicular phase	None

4.3. Post-Exercise Sauna Bathing Intervention

Participants attended sauna sessions 3 ± 1 times per week, completing a total of 9 ± 1 sauna sessions during the intervention and before the post HTT. Participants remained in the sauna for a total of 30 ± 0 min each session and participant's average total sauna duration across the 3 weeks totalled 274 ± 23 min. On average, across all sauna sessions, peak HR reached 126 ± 17 beats·min^{-1} whilst in the sauna.

Duration, average sweat loss and average sweat rate across all participants were not significantly different during the first sauna session compared to the last (9th) sauna session (all $p > 0.05$, Fig 5).

However, peak HR across all participants was 8 ± 14 beats/min higher during the first sauna session compared to the last session, $p < 0.05$. Average peak thermal comfort across all

participants was 2 ± 2 higher during the first sauna session compared to the last session, $p < 0.001$. Average peak thermal sensation across all participants was 1 ± 0 higher during the first sauna session compared to the last sauna session, $p < 0.01$.

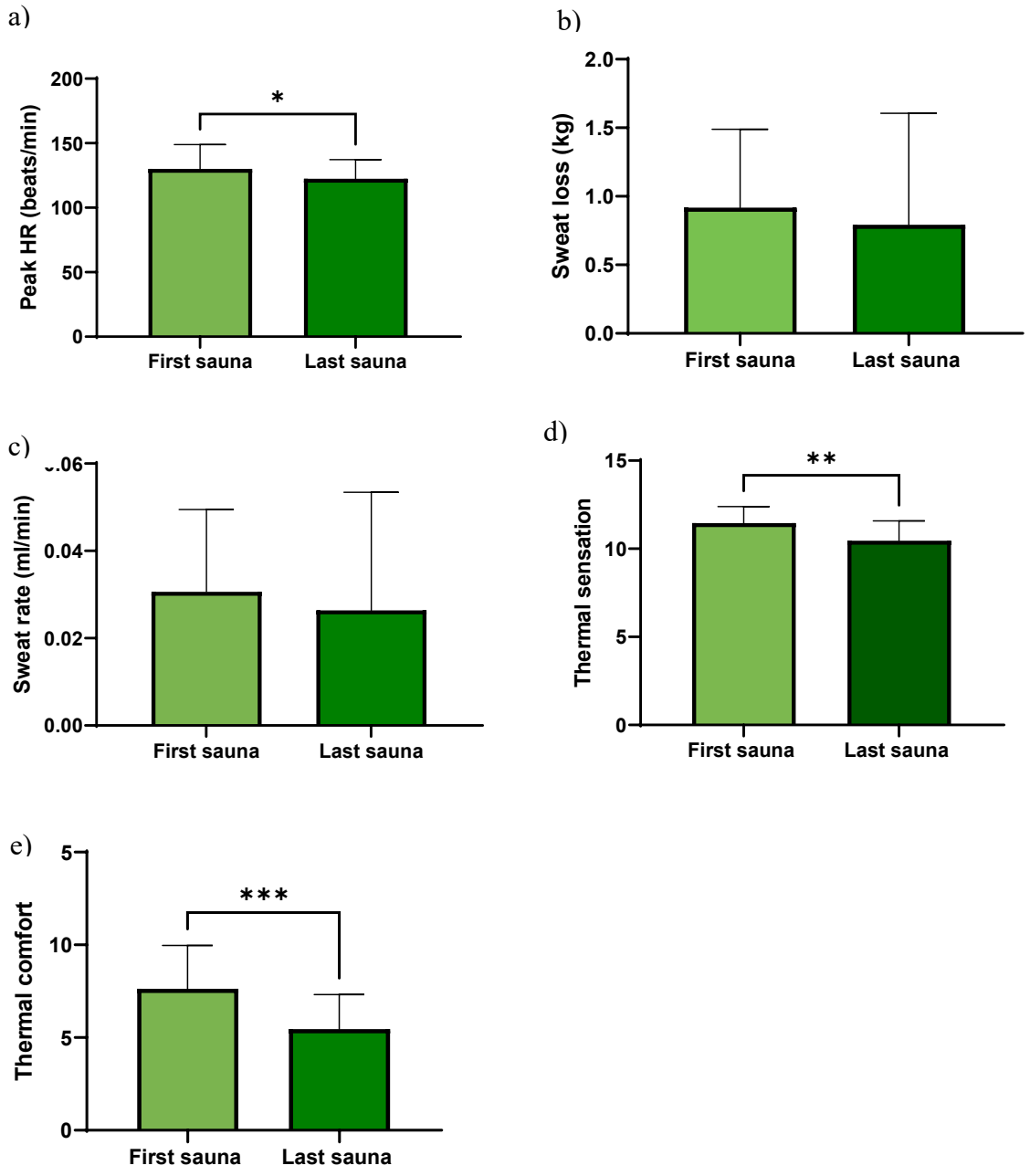


Fig 5. Physiological responses a) peak heart rate (HR) b) sweat loss and c) sweat rate and perceptual measures d) thermal sensation and e) thermal comfort of participants during their first (light great) and last (dark green) sauna visit. Data presented as mean ± SD. Statistical differences between the two visits are indicated by (*) $p<0.05$ (**) $p<0.01$ and (***) $p<0.001$.

4.4. Running Heat Tolerance Test

4.4.1. Core body temperature:

Core body temperature (rectal temperature) responses of participants during all three HTT are depicted in Figure 6a, b, c. For core body temperature there was a main effect between intervention HTTs [F (1.643, 26.28) = 13.72, $p < 0.001$] and a main effect of time [F (1.641, 26.26) = 246.9 $p < 0.001$] but no interaction effect [F (2.183, 34.93) = 0.5897, $p = 0.574$]. Post hoc analysis revealed significant decreases (all $p < 0.05$) in core body temperature between Pre and Post and Pre to Decay across all time points (5, 10, 15, 20, 25, 30 min). At rest core body temperature decreased by 0.23 ± 0.06°C Pre to Post ($p < 0.01$) and 0.19 ± 0.07°C from Pre to Decay ($p < 0.05$). Peak core temperature (30 mins) also decreased by 0.26 ± 0.07°C from Pre to Post ($p = 0.004$) and 0.17 ± 0.06°C from Pre to Decay ($p = 0.035$). No significant difference was found Post to Decay at any time point ($p > 0.05$), however.

Table 3: Physiological and perceptual responses during the running Heat Tolerance Test

	Pre	**Post**	**Decay**
$T_{recRISE}$ (°C)	1.20 ± 0.31	1.23 ± 0.3	1.25 ± 0.37
Peak T_{sk} (°C)	36.85 ± 0.54***	36.37 ± 0.44*	36.49 ± 0.64
Sweat loss (kg)	0.79 ± 0.46	0.75 ± 0.55	0.81 ± 0.46
Sweat rate (ml/min)	0.028 ± 0.017	0.025 ± 0.018	0.027 ± 0.014
Peak $RPE_{6–20}$	11 ± 2	11 ± 2	10 ± 2
Peak thermal sensation	10 ± 1	10 ± 1	10 ± 1
Peak thermal comfort	5 ± 2	4 ± 2	5 ± 2
Urine Osmolality (mOsm/kg)	402 ± 262	392 ± 254	426 ± 218

Data are presented as mean ± SD. Statistical differences between the timeframes are indicated by (*) $p<0.05$ (**) $p<0.01$ and (***) $p<0.001$.
T_{sk} skin temperature, $T_{recRISE}$ change in rectal temperature during exercise, $RPE_{6–20}$ rating of perceived exertion (6–20 point scale). Thermal sensation and thermal comfort were measured on 13- and 10-point scales, respectively.

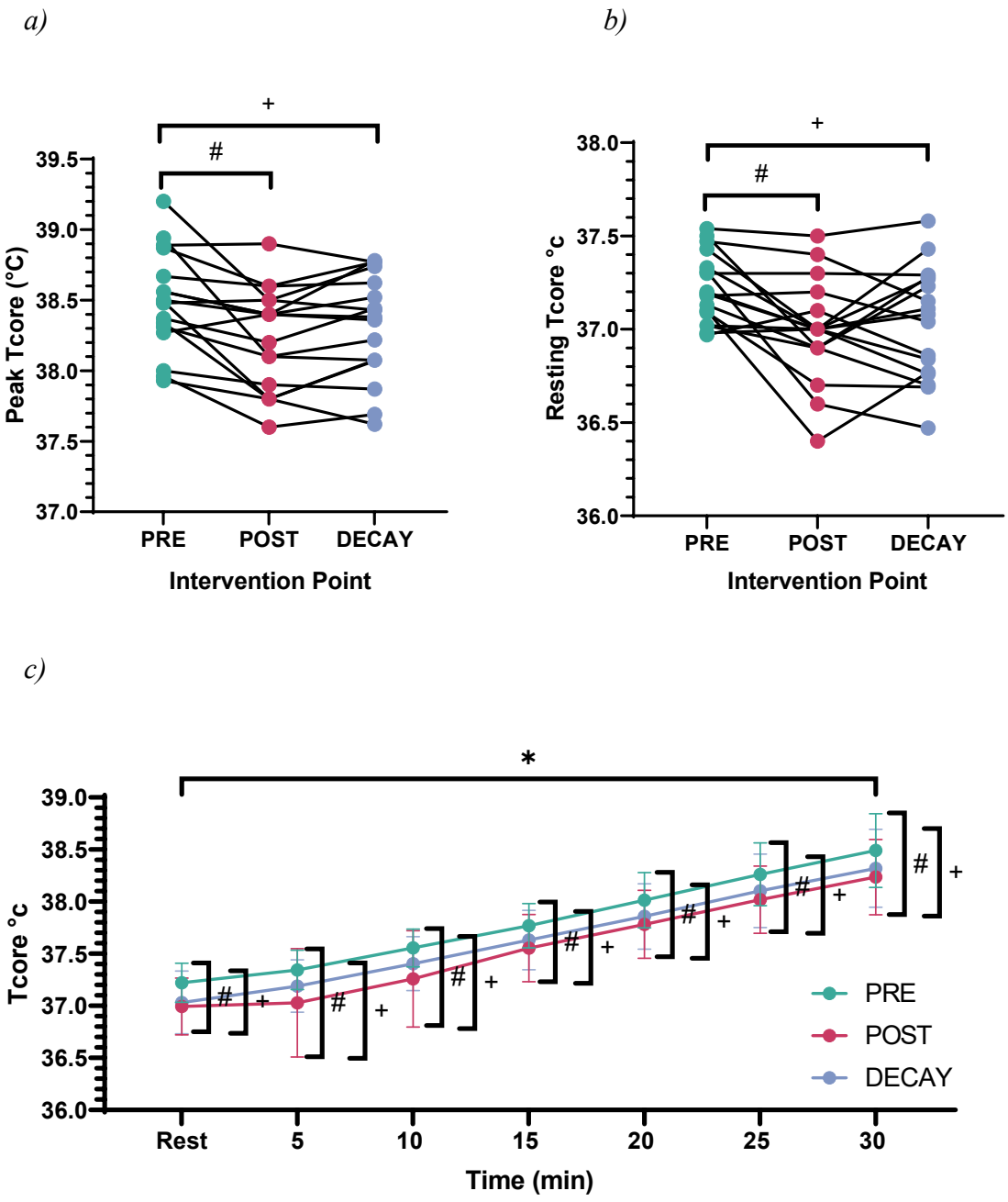


Fig 6. Physiological responses a) peak core body temperature b) resting core body temperature c) core body temperature during heat tolerance tests (HTTs). All participants completed 3 HTT at different time points: PRE (green), POST (pink) and DECAY (purple). Core body temperature during each HTT was recorded at time intervals: rest, 5 mins, 10 mins, 15 mins, 20 mins, 25 mins and 30 mins. Data presented as mean ± SD. Main effect of time significance relative to rest is indicated by (*) $p < 0.05$. Significant differences pre to post each time point are indicated by # $p < 0.05$; and significant differences pre to decay each time point are indicated by + $p < 0.05$.

4.4.2. Mean Skin temperature:

Mean skin temperature responses of participants during all three HTTs are depicted in Fig. 7. There was a main effect between intervention HTTs [F (1.833, 32.99) = 13.73, $p <$ 0.001]. There was also an interaction effect shown between intervention HTTs and time points [F (2.023, 36.41) = 5.378, $p = 0.009$]. However, there was no main effect of time (at 10-min, 20-min and 30min of HTTs) [F (1.179, 21.22) = 0.04329, $p = 0.874$]. Post hoc results revealed skin temperature decreased 0.31 ± 0.10°C from Pre to Post at 20 mins ($p = 0.014$) and 0.32 ± 0.12°C from Pre to Decay at 20 mins ($p = 0.047$). Post hoc results also revealed skin temperature decreased 0.90 ± 0.13°C from Pre to Post at 30 mins ($p < 0.001$) and increased 0.40 ± 0.15°C from Post to Decay at 30 mins ($p = 0.035$).

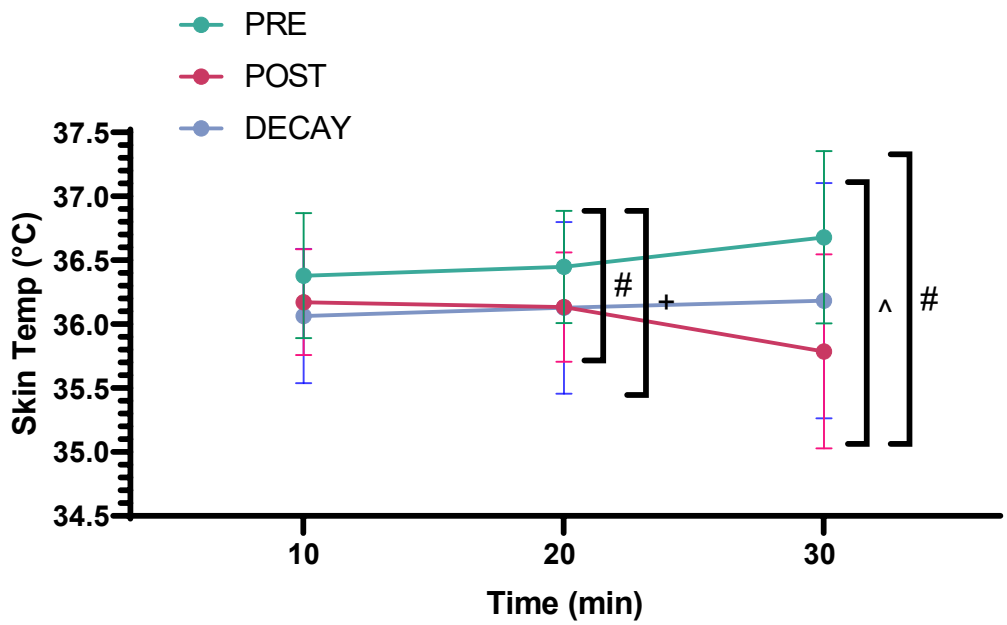
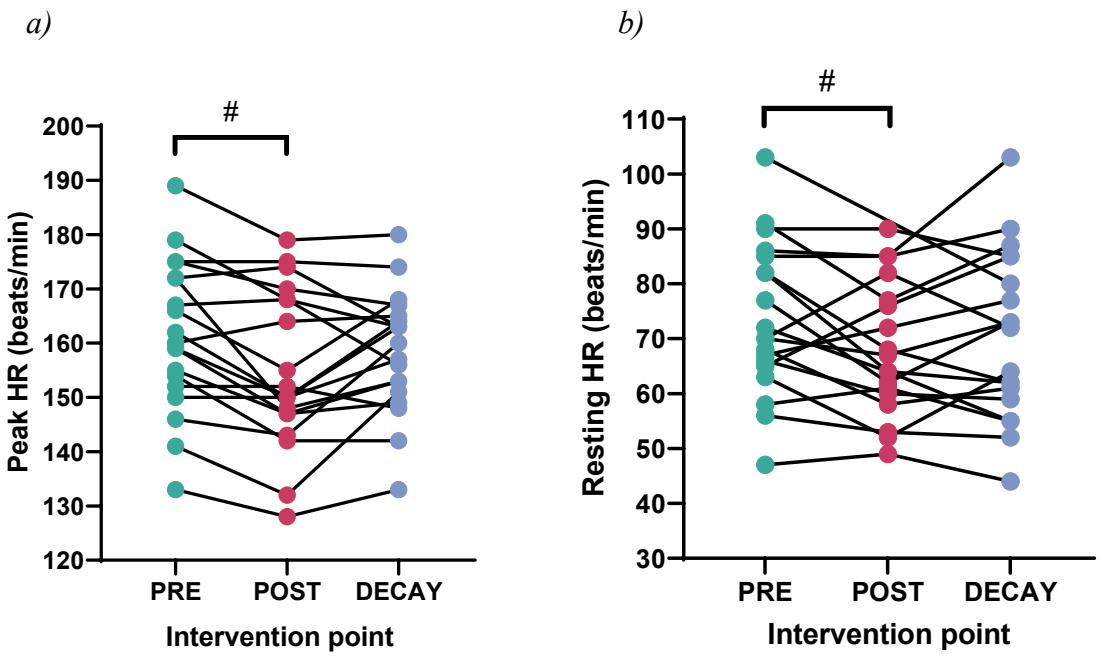


Fig 7. Mean skin temperature during heat tolerance tests (HTTs). All participants completed 3 HTT at different time points: PRE (green), POST (pink) and DECAY (purple). Data was recorded at time intervals: 10 mins, 20 mins and 30 mins. Data presented as mean ± SD. Significant differences pre to post each time point are indicated by # $p < 0.05$; significant differences pre to decay each time point are indicated by + $p < 0.05$; and significant differences post to decay each time point are indicated by ^ $p < 0.05$.

4.4.3. Heart Rate:

Average HR responses of participants during all three HTT are depicted in Fig. 8. Overall there was a main effect between intervention HTTs [F (1.964, 35.35) = 4.003, p = 0.028]. There was also a main effect between time points during the HTTs [F (1.219, 21.94) = 433.6, $p < 0.001$]. No interaction effect was shown, however [F (1.546, 27.58 = 0.5154, p = 0.557]. From Pre to Post, post hoc analysis revealed HR decreased at rest by 7 ± 2 bpm (p = 0.42), decreased at 20 mins by 4 ± 2 bpm (p = 0.34), and decreased at 30 mins (peak) by 6 ± 2 bpm from Pre to Post (p = 0.002). However, from Pre to Decay no significant difference was shown at any time point (all $p > 0.05$) and no significant difference was found Post to Decay at any time point (all $p > 0.05$).

Peak HR showed a significant main effect between intervention points (p = 0.012, table 3). Peak HR reduced by 6 ± 2 bpm from Pre to Post HTT (p = 0.002) but did not significantly decrease Pre to Decay or significantly increase Post to Decay ($p > 0.05$). Resting HR was not different across all three HTT tests ($p > 0.05$).



c)

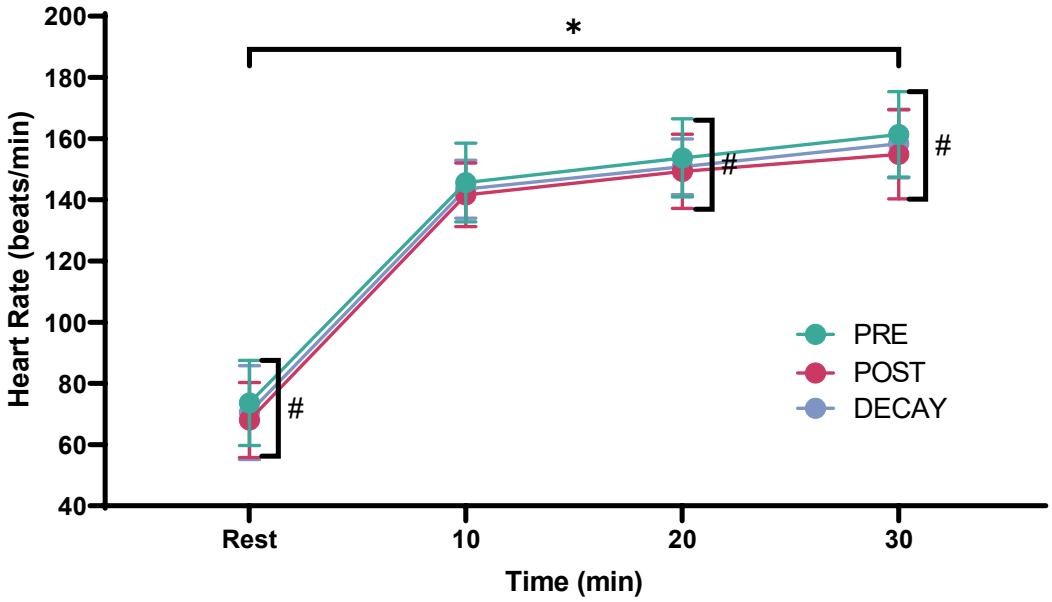


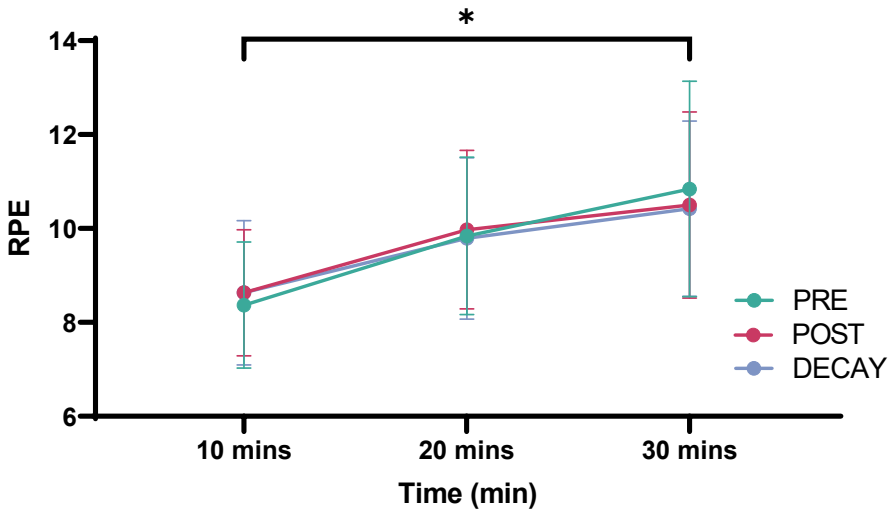
Fig 8. Cardiovascular responses a) peak heart rate (HR) b) resting heart rate (HR) c) average heart rate (HR) during heat tolerance tests (HTTs). All participants completed 3 HTT at different time points: PRE (green), POST (pink) and DECAY (purple). HR data during each HTT was recorded at time intervals: rest, 10 mins, 20 mins and 30 mins. Data presented as mean ± SD. Main effect of time significance relative to rest is indicated by (*) $p < 0.05$. Significant differences pre to post each time point are indicated by # $p < 0.05$.

4.4.4. Perceptual Measures:

Perceptual data at Pre, Post and Decay are detailed in Table 3 and depicted in Fig. 9a, b, c. RPE showed a main effect between time points during the HTTs [F (1.338, 24.08) = 41.56, $p < 0.001$]. No main effect between intervention HTTs [F (1.564, 28.16) = 0.05422, p > 0.05] or interaction effect [F (2.937, 52.87) = 1.117, p > 0.05] was shown, however. Thermal comfort also showed a main effect between time points during the HTTs [F (1.373, 24.72) = 39.93, $p < 0.001$]. No main effect between intervention HTTs [F (1.531, 27.56) = 1.189, p > 0.05] or interaction effect [F (2.420, 43.56) = 1.057, p > 0.05] was shown, however. Across all HTTs, post hoc analysis revealed thermal comfort at 10 mins increased by 1 ± 0 from Post to Decay ($p = 0.030$).

Thermal sensation showed a main effect between time points during the HTTs [F (1.851, 33.32) = 4.129, $p = 0.028$] and a main effect between intervention HTTs [F (1.435, 25.83) = 44.67, $p < 0.001$]. No interaction effect [F (3.133, 56.40) = 0.6036, $p > 0.05$] was shown, however. Post hoc analysis revealed thermal sensation at 10 mins decreased by 1 ± 0 from Pre to Post ($p = 0.019$).

a)



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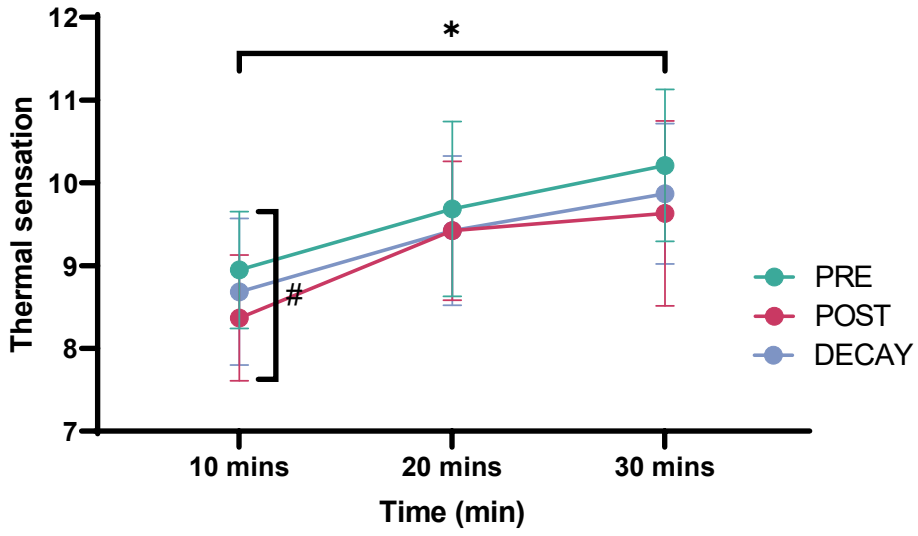

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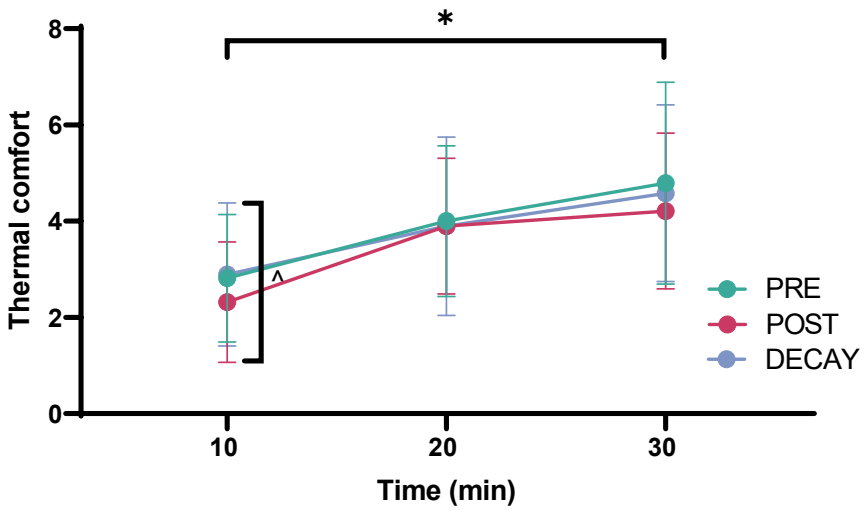

Fig 9. Perceptual measures a) thermal sensation b) thermal comfort and c) RPE during heat tolerance tests (HTTs). All participants completed 3 HTT at different time points: PRE (green), POST (pink) and DECAY (purple). Data was recorded at time intervals: 10 mins, 20 mins and 30 mins. Data presented as mean ± SD. Main effect of time significance relative to rest is indicated by (*) $p < 0.05$. Significant differences pre to post each time point are indicated by # $p < 0.05$; and significant differences post to decay each time point are indicated by ^ $p < 0.05$.

4.4.5. Decay Rate:

Decay rate for the heat strain variables following 2 weeks of HA decay are detailed in Table 4. All physiological variables showed a degree of decay with resting core body temperature showing the smallest rate of decay and Peak HR showing the largest rate of decay.

Table 4: Percentage rate of HA decay (positive number) or gain (negative number) in the physiological variables following 2 weeks of HA decay.

	Average HAD rate (%)
Peak core temperature (°C)	30
Resting core temperature (°C)	20
Mean skin temperature (°C)	24
Peak HR (beats min−1)	50
Resting HR (beats min−1)	44

5. Discussion

5.1. Decay following Post-exercise Sauna Heat Acclimation:

The main findings of this study are that resting and peak core body temperature reductions were significantly sustained for 2-weeks post HA. However, mean skin temperature appeared to be only partially retained as there was a significant rise (0.40 ± 0.15°C) after the decay period as compared to Post the sauna intervention and mean skin temperature at Decay did not return to Pre-intervention levels (-0.50 ± 0.12°C decrease Pre to Decay), indicating only partial decay. Somewhat similar to mean skin temperature, HR also appeared to be only partially retained as although not significant, HR was still lower at Decay and did not return to Pre-intervention levels (-3 ± 2 beats/min), again indicating only partial decay post HA. Therefore, the current study's hypothesis was partially accepted and concludes 2-weeks after cessation of post-exercise sauna bathing, key HA adaptations do not return to baseline levels.

A decrease in exercising core body temperature and skin temperature in hot and temperate environments are primary indications of heat adaptation (Periard et al., 2015). This is because exercising core body temperature is the product of all physiological adaptations that modify the thermoregulatory response to heat stress and exercise (Sawka et al., 2011). Alongside reductions in core body temperature, HA-related reductions in skin temperature indicate decreased thermal strain. Furthermore, an attenuated rise in HR is another primary indication of heat adaptation because it signifies decreased cardiovascular strain in the heat and therefore improved cardiovascular stability (Sawka et al., 2011). When an individual is no longer exposed to the heat these HA adaptations are known to decay. Therefore, the timeframe of this decay is important to show when the individual is no longer adapted to the heat (Daanen et al., 2018). The current study's findings support other findings in the literature. For example, Zurawlew et al. (2019) found 6-consecutive days of post-exercise

HWI sustained core body temperature, skin temperature and HR adaptations for at least 2-weeks. They showed that resting core body temperature, end exercise core body temperature, mean skin temperature and peak HR all remained lower after 2-weeks Post HA (decay) compared to Pre, -0.36 ± 0.25°C, -0.36 ± 0.37°C, −0.77 ± 0.70°C and −14 ± 10 beats/min, respectively. Comparably, our study found smaller differences between Pre and Decay, with decreases of -0.19 ± 0.07°C, -0.17 ± 0.06°C, -0.50 ± 0.12°C and -3 ± 2 beats/min, respectively. Therefore, it appears that post-exercise HWI elicits larger changes in HA adaptations than post-exercise sauna bathing, however these adaptations were largely maintained following 2-weeks decay with both modalities. Tyler et al. (2016) has suggested consecutive HA causes larger HA adaptations than intermittent HA protocols. Therefore, the large HA changes in the Zurawlew et al. (2019) study could be due to the use of a consecutive HA protocol compared to the intermittent protocol this current study used. Garret et al. (2011) also used a short term HA protocol but a more traditional HA intervention involving 90-mins of controlled hyperthermia (targeted rectal temperature 38.5°C, T_a = 39.5°C, 60% RH) for 5-days consecutively. Garret et al. (2011) found HA-induced reductions in peak core body temperature (0.3°C) and peak HR (13 beats/min) did not significantly decay after a 1-week decay period but did decay after a 2-week decay period. Therefore, controlled hyperthermia HA interventions could result in faster decay of HA adaptations than post-exercise HWI. On the other hand, another traditional longer term HA intervention using controlled hyperthermia (targeted rectal temperature 38.5°C) by Weller et al. (2007) found similar results to this current study. Weller et al. (2007) used a HA protocol consisting of 60-mins of treadmill walking (1.53m/s; at an incline) in a heat chamber (dry-bulb temperature 46.1 ± 0.1°C; RH 17.9 ± 0.1%) followed by 10-min rest and 40 min further treadmill, for 10 days consecutively. Weller et al. (2007) found HA-induced reductions in end exercise core body temperature and HR did not significantly decay after a 12- and 26-day

decay period. However, it is possible that the maintenance of HA adaptations at 26 days were helped by the 100-min HTT (divided into 60- and 40-min, separated by a 10-min rest) at 12 days, and therefore may need to be interpreted with caution. Regardless, the maintenance of HA after 12 days found by Weller et al. (2007) is similar to this current study's 2-week decay findings. Overall, there is conflicting evidence for how long HA adaptations are maintained but these results may be influenced by the mode and duration of the HA intervention. The current study's findings provide useful evidence that intermittent protocols can be used instead of consecutive day interventions but it appears the number of sessions likely drives the overall rate of decay rather than the intervention duration (Fein, Haymes & Buskirk, 1975).

Other findings of this current study are that core body temperature and peak skin temperature had a lower decay rate than HR adaptations, as hypothesised. Resting and exercise core body temperatures and peak mean skin temperature showed decay rates of 20%, 30% and 24%, respectively, compared to a 50% decay rate in peak HR and a 44% decay rate in resting HR. Our research supports previous studies, where mean percentage losses of core body temperature have been found to range from 5% to 43% after 2-weeks of decay following various different HA methods (Daanen et al., 2018). For example, Weller et al. (2007) observed a mean decay rate in core body temperature of only 5% after 26 days of decay, following a 10 consecutive day HA protocol involving 60-mins of treadmill walking as mentioned previously. Conversely, Poirier et al. (2015), observed a mean decay rate of 43% after 2-weeks decay following a 14 consecutive day HA protocol comprising of 90-min cycling in the heat (40°C, 20% RH) at ~50% $\dot{V}O_{2max}$. Notably, Daanen et al. (2018) found the number of HA days appeared to not affect the decay rate. Instead, the speed of decay rate appeared to be caused by increases in heat exposure duration and reductions in heat exposure intensity. For example, HA decay in core body temperature was reduced from 45% to 9%

when daily exposure was extended from 60 to 90-min at a WBGT of 32 °C (Daanen et al., 2018). Therefore this current study could have found higher decay rates in resting and exercise core body temperature (20% and 30%, respectively) due to only 30-min post-exercise sauna bathing exposure. The decay rate of mean skin temperature seems to show conflicting results in the literature. Weller et al. (2007) found a slow decay rate of 8% in mean skin temperature after 12 days and 56% after 26 days decay following their 10-day HA protocol (60-min treadmill walking per day in dry-bulb temperature 46.1 ± 0.1°C). Whilst Stephens and Hoag (1981), who used a 10 consecutive day HA protocol (treadmill walking at 5.0 km/hr for 100 minutes per day in 33.5°C WBGT), observed a faster decay rate of 67% after 5 days decay. Daanen et al. (2011) used a 12 consecutive day HA protocol that involved participants cycling for 2-h per day for 9 days at 26°C WBGT and 3 days at 32°C WBGT and they also observed a faster decay of 57% after 6 days. More research is needed to draw firmer conclusions, however it could be assumed that the higher the ambient temperature used during the HA protocol, the slower the mean skin temperature decay rate.

Daanen et al. (2018) meta-analysis showed 2-weeks post HA there was ~35% decay in HR. Using a similar 14-day HA protocol (90-mins of cycling at ~50% peak oxygen uptake in 40 °C and ~20 % RH) after 2-weeks decay Flouris et al. (2014) and Poirier et al. (2015) found a mean decay rate of 60% and 53%, respectively. In both studies, HR showed the largest decay rate at 2-weeks. Notably an early decay study by Pandolf (1977) reported an 85% decay in HR adaptations (compared to a 35% decay in core body temperature) after 2-weeks of decay following, a 9 consecutive day HA protocol in which participants walked at 1.34 m/s for 110 min a day in 49°C, 20% RH. This supports the notion that cardiovascular adaptations, that are the first physiological adaptations to occur (4-5 days; Sawka et al., 2011), are also the first adaptations to be lost (Armstrong and Maresh, 1991; Williams et al., 1967; Pandolf et al., 1977; Armstrong and Maresh, 1991; Daanen et al., 2011; Flouris et al.,

2014). Overall, it appears post-exercise sauna bathing seems to maintain thermoregulatory and cardiovascular adaptations for the same period as other traditional HA methods, with the decay of adaptations occurring in the same order (i.e. HR first adaptation to dissipate and core body temperature slower). However, there is still a considerable variation in the decay rates reported across different HA interventions. This indicates that the study design likely has an overall effect on the results found across these studies (Daanen et al., 2018). Thus, the stimulus used in HA protocols appears to influence decay just like it does in acclimation (Heathcote et al., 2018). However, as there is not enough research on this topic, there is not enough evident from previous studies to confirm this supposition.

In contrast to our hypothesis, none of the three perceptual measures assessed, including RPE, thermal comfort and thermal sensation, significantly changed following 3-weeks of post-exercise sauna bathing. Without this HA adaptation in perceptual measures (i.e. no difference between Pre and Post), HA decay could not be assessed. These findings were similar to Kirby et al. (2020) who used the same HA protocol as the current study of 30-mins post-exercise sauna bathing. Kirby et al. (2020) found thermal sensation was not different between HTTs (10 ± 1 and 9 ± 1 at Pre and 3-Weeks, respectively). Likewise, thermal comfort was not different between HTTs (5 ± 2 and 4 ± 2 at Pre and 3-Weeks, respectively). Only peak RPE showed a reduction at 3-Weeks as compared to Pre ($p = 0.08$; 12 ± 3 and 11 ± 2, respectively). This indicates that post-exercise sauna bathing appears to cause limited perceptual adaptations, with participants still perceiving exercising in the heat challenging despite being physically acclimated. Similar to these perceptual measure findings, sweat loss showed no significant change following 3-weeks of post-exercise sauna bathing. Therefore, again, without this HA adaptation in sweat loss, HA decay cannot be assessed. Kirby et al. (2020) again found no increases in whole body sweat loss (0.9 ± 0.2 and 0.8 ± 0.1, Pre and Post respectively) similarly to our study. The absence of the increase in

sweat loss during both studies does not necessarily mean individuals did not improve their thermoregulatory response, however. As mentioned previously, reductions in core body temperature were seen and this reduction may have been the result of increased heat dissipation, but possibly through increased skin blood flow combined with greater peripheral sweat gland activation to improve evaporative heat loss efficiency (Smith & Havenith, 2019). In support of this, Kirby et al. (2020) found increases in sweat gland activation on the forearm (+ 54%) were exhibited following 3-weeks of intermittent post-exercise bathing. Athletes competing in hot-humid environments may find this increase in sweat gland activation to be a more efficient adaptation as it would improve evaporative capacity without exacerbating dehydration (Alber-Wallerström and Holmér, 1985). Unlike the current and Kirby et al. (2020) studies, Zurawlew et al. (2019) found after a 6-consecutive day HWI intervention end-exercise RPE and end-exercise thermal sensation were reduced (−1 ± 2 and −1 ± 1; $P < 0.05$, respectively). These reductions in both RPE and thermal sensation were still observed after 2-weeks following HA (−1 ± 2 and −1 ± 1; $P < 0.05$, respectively). Previous studies also show sweat loss increases to have been retained for 1-3-weeks Post HA, in which mean percentage local and whole body sweat rate losses ranged from 54% to 79% from periods of 6 to 14 days after HA (Duvnjak-Zaknich et al., 2018; Garrett et al., 2009; Williams et al., 1967). Therefore, post-exercise sauna bathing does not appear to elicit all the thermoregulatory and perceptual adaptations that other HA methods achieve. The methodological differences between studies (including the variations of exercise intensities, environmental parameters, heat loss measurements, etc. (Poirier et al., 2015)) could account for the differences observed. For example, sauna sessions took place in the University of Birmingham Sport and Fitness Centre and this meant sauna temperature could not be controlled and varied between sessions (85–94°C). Individual differences are also another factor to consider, for example, cardiovascular differences between athletes are common in

the onset and decay of HA. Athletes with high $\dot{V}O_{2max}$ usually reach a stable HA state faster (and lose adaptations slower) than those with a low $\dot{V}O_{2max}$ (Armstrong and Maresh, 2012). Therefore, the difference between participants having a higher $\dot{V}O_{2max}$ (60.0 ± 6.0) in the current study and participants having a lower $\dot{V}O_{2max}$ in other studies could be a contributing factor to the differences seen across studies.

In this study, average weekly training duration was higher during the 3-week sauna intervention as compared to the decay period. This lower training duration during the decay period could have accelerated the decay rate of HA adaptations such as such as peak HR and sweat loss, potentially explaining the partial decay seen during the 2-week decay period (Pryor et al., 2019). However, in the current study there appeared to be no relationship (as indicated by correlation corrections in the Appendix) between training duration and the ability to maintain HA adaptations after a 2-week decay period. Thus, the partial decay seen during the 2-week period could be as a result of another factor. Notably, although training duration appeared to be lower, the intensity of exercise performed during the 2-week decay period appeared to be higher, which may explain the weak relationship between training duration and HA adaptation maintenance. There also appeared to be no relationship between $\dot{V}O_{2max}$ and the ability to maintain HA adaptations after a 2-week decay period. As mentioned above, individual training status is believed to influence the magnitude and timeframe of HA (Sawka et al., 2015; Daanen et al., 2018). A reason for not seeing a relationship between $\dot{V}O_{2max}$ and HA decay could be due to the fact that the highly trained endurance athletes participating in the current study were already partially heat acclimated (Taylor et al., 2000) due to their training regime. Also, highly trained individuals, with higher fitness levels and a higher aerobic capacity than untrained individuals, appear to have a higher heat tolerance and therefore can withstand a larger heat stimulus (Armstrong, and Maresh, 1991; Mikkelsen et al., 2019). Therefore, it is possible that the highly trained

participants in the current study did not adapt as effectively to the 3-week post-exercise sauna (passive) intervention as a less trained cohort would have (Garret et al 2011). Overall, these data indicate that a reduction in training duration may not influence HA decay rate. However, further research is needed to examine this more fully. If a reduction in training load does not affect HA decay, this could have promising implications for tapering.

5.2. Adaptation following Post-exercise Sauna Heat Acclimation:

Further findings of this study are that 3-weeks of post-exercise sauna bathing resulted in a significant reduction in thermal and cardiovascular strain. It induced HA adaptations including significant reductions in resting and peak HR, peak skin temperature and peak and resting core body temperature. A decrease in both resting core body temperature and resting HR contributed to a lower core body temperature and HR sustained throughout the HTT (Periard et al., 2015). Therefore, peak core body temperature and HR were also significantly lower after the post-exercise sauna intervention (Périard, Eijsvogels & Daanen, 2021). However, as previously mentioned, other typical HA adaptations including sweat loss, and perceived exertion did not show significant changes in the current study. It is well documented that athletes can achieve decreased core body temperatures, skin temperatures and HR after post-exercise sauna bathing for 3-weeks (Scoon et al., 2007; Kirby et al., 2020; 2021). The HA adaptation improvements in our study resemble the results from the previous study by Kirby et al. (2020). They found sauna exposure immediately following training enhances the thermoregulatory-adaptive response by reducing peak core body temperature, skin temperature, and HR (-0.2°C, -0.8°C and -11 beats/min, respectively) more than exercise alone at 3-Weeks compared to Pre (all $p < 0.05$). Similar reductions were found in our study at 3-Weeks compared to Pre (0.26°C, -0.89°C and -7 beats/min, respectively). Similar HA physiological adaptations have been shown in other studies using different HA protocols. For

example, Lorenzo et al. (2010) found skin temperatures reduced from 34.8 ± 0.3°C to 33.9 ± 0.2°C after a 10-consecutive day HA protocol in which participants exercised on a cycle ergometer at ~50% $\dot{V}O_{2max}$ and environmental conditions of 40°C and 30% RH. Furthermore, Tyler et al., 2016 meta-analysis found a reduction of 0.57 ± 0.49°C in exercising mean skin temperature following HA. This meta-analysis also showed reductions in resting core body temperature of ~0.18 ± 0.14°C following a range of HA protocols. The slightly larger 0.23°C in resting core temperature shown in the current study compares favourably to this meta-analysis. However, post-exercise sauna bathing seemed to elicit smaller changes in other physiological adaptations compared to other HA protocols. For example, Buono et al. (1998) and Shvartz et al. (1979) both found larger decreases in peak core body temperature of ~0.6°C following 7 consecutive days of HA in which 2 hrs of exercise on a treadmill or bike was performed in a hot, humid environment (35°C, 75% RH) and 8 successive days of HA in which 2-hours of exercise on a bicycle ergometer was performed at 50% $\dot{V}O_{2max}$ (75W) in 39.8°C dry bulb, respectively. Other studies in the literature also show larger reductions in HR than both Kirby et al. (2020) and this current study. Lorenzo et al. (2010) found HR to significantly reduce by 15 beats/min after the 10-day HA protocol (~50% $\dot{V}O_{2max}$ in 40°C), whilst, Weller et al. (2007) found HR to reduce by ~24 beats/min after 100 mins cycling in a hot chamber (dry-bulb temperature 46.1 ± 0.1°C; RH 17.9 ± 0.1%) after 10-consecutive days. Added to this, Pandolf et al. (1977) found HR to reduce by ~35 beats/min after a 110 min walking HA protocol across 9 consecutive days in 49°C, 20% RH. From these data it can be suggested that post-exercise sauna bathing elicits a smaller stimulus than an active, constant work rate HA stimulus and this may lead to a smaller degree of HA adaptations. Further work directly comparing the post-exercise bathing and active HA is needed to confirm this assertion. Of note, reductions in cardiovascular strain are achieved by redistribution of blood volume and plasma volume expansion (Sawka et al., 1996; Wyndham et al., 1968). Although

these variables were not measured in the currently study, Scoon et al. (2007) found plasma volume increased following 12.7 ± 2.1 sessions of 31 ± 5 min of post-exercise sauna bathing. Thus it can be asserted that some degree of plasma expansion supported HR reductions in the current study.

HA adaptations (during the 3-week intervention) and maintenance (during 2-weeks decay) of resting and peak core body temperature in the current study is likely due to the disturbed thermal balance of the body during the sauna exposures this type of heat stimulus has been shown to elevate HR and core body and skin temperatures considerably (Pandolf et al., 1977; Zalewski et al., 2014; Sawicka, Brzostek & Kowalski, 2007). When humans are exposed to hot environments, their first reflex is to accelerate blood flow to the periphery to induce heat loss from the skin (Tansey & Johnson, 2015). This rise in mean skin temperature is linearly related to an increase in HR and a rise in core body temperature by 1°C increases HR by 7–9 beats/min (Gorman & Proppe, 1984). Average peak HR in the sauna in this current study reached 126 ± 17 beats/min which was similar to Kirby et al. (2020) who found Peak HR reached 127 ± 10 beats/min whilst in the sauna. Other studies have found increases in HR ranging between ~100-150 beats/min, depending on the individuals age, fitness and the ambient temperature of the sauna (Kauppinen & Vuori, 1986; Sohar et al., 1976; Leppäluoto et al., 1986). An increase in HR to approximately 120 beats/min establishes a beneficial HA adaptation, therefore providing evidence that the increase seen during the sauna exposures in our study likely led to changes in resting and peak core body temperature (Sawicka, Brzostek & Kowalski, 2007; Siquier-Coll et al., 2023). Although this study did not measure core body or skin temperature rises in the sauna, exposure to this large exogenous thermal stimulus with these very high temperatures (85–94°C) (compared to other HA methods), has previously caused increases in mean skin and core body temperatures up to 37.6-40ºC (Stanley et al., 2015; Zalewski et al., 2014; Sohar et al., 1976). Repeat increases in mean skin temperature,

as occurs during regular post-exercise sauna bathing, activates warm-sensitive neurons and induce hypothalamic neural network changes, which is believed to be the cause of reduced resting core body temperature (Tan et al., 2016). Overall, these findings provide evidence that the elevation in HR and skin and core body temperatures had an influence on the beneficial adaptations established during the Post HTT.

5.3. Post-exercise sauna protocol

Overall, average sweat loss and average sweat rate during the sauna sessions across all participants were not significantly different from the first sauna session as compared to the last sauna session. This was different to Ashworth, Cotter & Kilding (2023), who showed sweat rate significantly increased from 0.6 ± 0.6 L to 0.8 ± 0.8 L after a 5 day post-exercise sauna regime (70 °C, 18% RH). Sweat loss is greater after harder sessions compared to short easy runs (Arts & Kuipers, 1994). Although athletes were mostly exposed to the sauna after an easy run, this could vary between distance and pace and some sauna exposures happened after more intense exercise sessions. Furthermore, sauna temperatures did not remain constant and ranged from 85–94°C. Therefore, despite potentially acclimating to the sauna conditions, athletes could have performed a harder run before the first session or the conditions in the sauna could have been more extreme which could have caused increases in sweat loss, resulting in not significant differences between the first and last sauna. On the other hand, the current study found peak HR across all participants was higher during the first sauna session compared to the last session ($p < 0.05$). Therefore, despite no changes in sweat loss, lower peak HR during the last sauna session indicates successful HA in the participating individuals (Trachsel et al., 2020; Heathcote et al., 2018). Although Kirby et al. (2020), who used a 3 ± 1 times per week sauna regime (101–108 °C, 5–10% RH), found peak HR was not different across the 3-week intervention between the first and last sauna session (all $p > 0.05$),

Ashworth, Cotter & Kilding (2023) found similar findings in HR to this current study. HR significantly reduced from 110 ± 15 beats/min to 102 ± 15 beats/min compared to this current study's reduction from 130 ± 19 beats/min to 122 ± 15 beats/min.

However, average peak thermal comfort and thermal sensation were both higher during the first sauna session compared to the last session ($p < 0.05$). Therefore, participants appeared to adjust to the feeling and comfort of the sauna across the course of the sauna sessions. Opposite to the above findings, however, Ashworth, Cotter & Kilding (2023) found no significant decreases from the first to last sauna in thermal sensation (10.5 ± 1.2 to 10.3 ± 1.1, respectively) and thermal comfort (4.6 ± 2.1 to 4.4 ± 1.8, respectively). Overall, the differences found across all three studies mentioned are most likely explained by individual athlete responses to HA and therefore, these differences need to be considered (Casadio et al., 2017).

5.4. Menstrual cycle

Following Kirby et al., 2020, this study included a sample of female athletes that were not excluded based on their menstrual cycle patterns or hormonal contraceptive use. This was to be representative of the female athlete population and random sampling of the female cohort and their menstrual cycle phases is important for ecological validity (Kirby et al., 2021). During the study by Kirby et al. (2021), eleven out of the thirteen eumenhorreic female participants (regular menstrual cycles lasting 25–36 days) completed HTTs in the same phase of their menstrual cycle. In this current study, menstrual cycle was self-reported and most female participants reported being in the same phase for the first two HTT tests and then a different phase for the decay HTT. This was inevitable, as although the timeframe between Pre to Post was designed by Kirby et al. (2021) to fit the menstrual cycle, the 2-week decay period inevitably meant this HTT would be conducted in a different phase of the

menstrual cycle. The difference in phases during this current study could have caused variations in our results (Kirby et al., 2021). This is because hormonal fluctuations occur within the menstrual cycle and these are suggested to modify central regulatory mechanisms for thermoregulation (Inoue et al., 2005). Importantly in this study, three female participants completed the decay HTT during the Luteal phase, whereas they completed the Pre and Post HTTs in the Follicular phase of their menstrual cycle. The elevated progesterone concentrations during the Luteal phase would have caused an ~0.34°C increase in resting core body temperature and affected the onset threshold for sweating by 0.29°C (Mee et al., 2015; Yanovich et al., 2019). This could have affected Decay HTT results as increases in resting core body temperature for three participants were likely due to menstrual cycle effects as opposed to decay alone.

5.5. Training intervention

Structuring the protocol around the club's training schedule was important to ensure a similar exercise training stimulus between all participants. Furthermore, unlike most HA protocols, changes to the participants training were not required to accommodate any HA sessions. Most elite athletes have a complex and varied training programme, where stress is consistently applied to develop the physical and mental resiliency needed for optimal performance. The added stress applied through heat protocols has been shown to intensify internal training load further (Crowcroft et al., 2015), which then also potentially impairs an athlete's ability to recover for subsequent training sessions (Minet et al., 2015). Therefore, with active HA protocols, concurrent training has to be structured around HA, and questions arise to which sessions heat should be added to, and how subsequent routine training sessions should be adjusted based on prior heat stress. This passive HA method is therefore important as it allows athletes to complete regular training without heat stress causing disruption and

affecting training quality, whilst still allowing HA adaptations to be acquired (Casadio et al., 2017; Périard, Eijsvogels & Daanen, 2021).

5.6. Perspectives

The research around HA protocols for advantageous health and performance benefits is forever improving (Racinais et al., 2015). Kirby et al. (2020), already concluded that athletes can incorporate a post-exercise sauna regime (under controlled conditions) for 3-weeks (in which participants were exposed to only ten 30-min exposures) and achieve beneficial adaptations that help them adapt to the hot environments and prepare for competition.

This study's investigation of the maintenance of heat related adaptations after cessation of post-exercise sauna bathing is now another crucial addition to this ongoing topic of research. Although this study is not alone in looking at HA decay, no other study has previously measured the process of HA decay after a post-exercise sauna HA intervention. Specifically, the current study examined decay rates after 2-week as HA decay occurs within a 2-week timeframe and HRA is required (Poirier et al., 2015). Subsequently, this study showed that 2-weeks decay showed no significant reverse changes to core body temperature, and HR and skin temperature adaptations only partially decayed.

Athletes are continuously travelling abroad to compete in hot environments, with global warming increasing the temperatures of many countries further (Nowak et al., 2022). Therefore, athletes and coaches can now not only understand that passive heating interventions can induce favourable adaptations without overloading the body with heat stress and jeopardising the race, but in addition have the knowledge that these adaptations last for at least two weeks. The results found are important in helping find a way of implementing HA protocols into a competition setting. In the 2-weeks after HA, a fully heat-acclimated athlete

can recover and taper to achieve optimal form without experiencing significant decay in core body temperature or HR HA adaptations based on the data derived during this study. Moreover, post-exercise heat exposure could be conducted during the taper period to help maintain HA status, whilst still reducing their exercise/training load (Kirby et al., 2020). For example, participants could enter the sauna after their so called easy run during the taper and support HA maintenance.

5.7. Limitations and future research

This current study chose to not include a control group/condition. Kirby et el. (2020) previously concluded that intermittent post-exercise sauna bathing across three-weeks endurance training improves exercise heat tolerance and exercise performance markers in temperate conditions, compared to endurance training alone. Therefore, this evidence was the reasoning behind this study choosing not to have a control group. However, including a control group in this study could have demonstrated specificity in the effects that were observed and eliminated the possibility that the thermoregulatory changes were driven by their exercise training.

This study did not focus on the reasons behind individual differences. There were a few participants who appeared to respond better than others when looking at HA adaptations such as resting and peak core body temperature and HR. These results could be due to a number of different lifestyle or individual factors that potentially could have been more controlled. For example, participants who trained slightly less than other participants during the decay period could have accelerated the decay rate of HA adaptations (Benjamin et al., 2019). This is because exercise training in temperate environmental conditions during the decay period appears to help partially maintain HA adaptations (Daanen et al. 2018). As mentioned previously, this study did show how training duration was not correlated with HA

adaptations, however this was taken as an average not as individual responses. Although participants were at similar fitness levels it is difficult to know whether a slight difference in $\dot{V}O_{2max}$ may have caused some participants to sweat more and dilate the skin faster for a smaller change in mean core body temperature and lose adaptations more slowly during decay (Armstrong, and Maresh, 1991; Mikkelsen et al., 2019). Furthermore, at a population level, women tend to have lower aerobic/cardiorespiratory fitness and VO_{2max} when compared with men, potentially due to having a smaller body size, less muscle mass, and more fat mass (Gagnon & Kenny, 2012). Women also have reduced levels of sudomotor activity and sweat gland output at highest levels of required heat loss (Gagnon & Kenny, 2012; Ichinose-Kuwahara et al., 2010). When comparing males and females of similar age and health status, there is currently no evidence suggesting a disadvantage in thermoregulation when females exercise in the heat. Previous HA studies have found that the magnitude of physiological adaptations to HA are similar between both males and females, although there appears to be differences in the time course for the induction of HA adaptations (Wyndham et al. 1965; Shapiro et al. 1980; Mee et al. 2015). Therefore, the differences between the individuals training, $\dot{V}O_{2max}$ and sex are also up for debate and these could be factors contributing to the differences in responders.

Results during the HTT were also affected by lifestyle factors such as sleep. Some participants performed the tests under poor sleep which is considered a stressor on the body (McEwen 2006). Participants self-reported before all HTT and some reported less than 7-hours sleep the night before the trial which is considered as insufficient sleep (Mah et al., 2018). Acute sleep loss seems to have a negative effect on next-day exercise performance (Craven et al., 2022). This effect may be greater when the performance task is conducted in the PM after sleep deprivation the night before. Individuals can anticipate a ~ 0.4% decline in performance for every hour spent awake following acute sleep loss (Craven et al., 2022).

Sleep is very difficult to control but is a factor that needs to be looked into further to eliminate any detrimental effects. As well as this, on a couple of occasions participants were unwell when they performed the test. Although we tried to eliminate this issue by moving the test back a day or two, the participant was not guaranteed to be back to a healthy state by this time. Strenuous exercise during ongoing fever can be hazardous and should be avoided (Friman & Wesslén, 2000). When an individual has an illness/infection, the hypothalamus releases hormones that cause body temperature to increase (Friman & Wesslén, 2000). Therefore, as core body temperature increases during exercise, and then further increases during exercise in the heat, with this already high body temperature results can be inaccurate (Friman & Wesslén, 2000). Moving the test back, however, could have also had an effect on results as it would have meant it was not performed in the exact timeframe we allocated. Therefore, like sleep, illness is important to control, so is another factor that needs to be looked into further to eliminate any detrimental effects.

Although this study provided daily iron to our participants to help stop detrimental effects to performance and health through low iron status (DellaValle, 2013), we did not measure baseline iron status. Beard et al. (1990) observed how iron deficiency results in impaired thermoregulation and therefore may cause risks for individuals exposed to high temperatures. Therefore, it would have been beneficial to measure ferritin in the blood to confirm the participants had sufficient iron levels to minimise any risks associated and eliminate any outliers in the results.

Most participants completed 9 sauna sessions, however, a few participants appeared to complete 10 or 11 exposures. It is unclear how much of an impact this had on HA adaptations and decay rates, more sauna exposures could have meant increased HA adaptations and therefore a slower decay rate. Therefore, this could have been another factor

contributing to slightly different individual responses and controlling the amount of sauna sessions could have eliminated any possible skewed results.

This current study chose to focus on the key physiological HA adaptations but other adaptations including markers of exercise capacity have also been looked at (Kirby et al., 2020; Lee et al., 2022; Scoon et al, 2007). Lee et al. (2022) compared the effects of post-exercise sauna bathing to regular exercise, measuring cardiovascular adaptations including cardiorespiratory fitness, systolic blood pressure and total blood cholesterol. Kirby et al. (2020) also compared intermittent post-exercise sauna bathing across 3-weeks to endurance training alone and measured the improvement in exercise heat tolerance and exercise performance markers in temperate conditions, showing a 8% mean improvement in $\dot{V}O_{2max}$. Unlike Kirby et al. (2020) and Lee et al (2022), the current study focused on the thermoregulatory and cardiovascular decay rates only. Therefore, additional $\dot{V}O_{2max}$ tests (at Post and Decay) were not performed in the current study and subsequently, we cannot comment on how potential performance improvements resulting for this HA protocol may have decayed over 2-weeks. The decay rate of performance improvements following HA would be useful information for athletes using this protocol as part of their training or preparation for a major competition. Therefore, future research should incorporate fitness testing to find a way to implement this specific protocol into the competition setting.

Body core temperature is considered one of the most informative indicators of thermal stress (Montain et al, 1994; Sawka et al., 2001) and a key indicator in thermal comfort (Gagge et al., 1967). Therefore, given this importance of core body temperature, reliably monitoring, measuring, estimating, and predicting core temperature has become important within the research and clinical communities (Moran & Mendal, 2002). A notable limitation of this study was that core body temperature was measured using a rectal thermistor inserted 10cm past the anal sphincter. Although rectal thermistors are the most common method to

measure core body temperature, for reasons such as comfort and reliability the use of this method can be challenging (Poole & Stephenson, 1977). This method has been known to cause delay in the core body temperature readings as it is further away from the heart and the gluteus maximus and the surrounding muscles can also generate heat and effect the reading (Moran & Mendal, 2002). Furthermore, if the participant does not insert the rectal thermistor correctly there is a chance of the thermistor moving or falling out. Rectal measures in the current study were monitored to check whether the thermistor moved during testing, however, despite this monitoring there was some timepoints in some participants where rectal temperatures were inaccurate due to slipping. To address inaccurate (and non-physiological) readings, these data points were modelled. Other methods to estimate/approximate core body temperature have been developed which include the use of skin temperature, respiration rate and the most ideal method is using solely HR (Buller et al., 2013; Niedermann et al., 2014; Richmond et al., 2015). In addition, skin temperature was measured from temperature sensors (skin thermistors) in contact with the skin's surface. Due to sweating and movements whilst running, on occasions, the skin thermistors lost contact with the skin. Therefore, it is worth considering whether alternative core and body skin temperature measures could be used instead in future research.

Participants in this study were of a very similar age. Research suggests that athletes' peak physiological function occurs just before the age of 30 years and they can still continue to compete past this age (Gabbard, 2004). Therefore only having university athletes up to the age of 24 does not represent the entire athlete population. Although limited, when looking at highly trained individuals, data suggests that age does not affect HA benefits such as core body temperature and whole body sweat loss (Garrett et al., 2009; Best et al., 2014). However, no research to date has examined age-related differences in decay and re-acclimation. To date, decay studies have only used participants of ages 18 to 30- years

(Daanen et al., 2018). Therefore, future research should focus on studying mixed ages to ensure HA programmes suit the entire athlete population. Furthermore, this current study was unable to measure sex differences due to the sample size being too low. Despite this, this current study is unique as it is the only other study to measure decay in females other than Stephens and Hoag (1981).

6. Conclusion:

Overall, this study duplicated most of the findings of Kirby et al. (2020) and demonstrated that post-exercise sauna bathing induces some key thermoregulatory adaptations during exercise at an absolute workload. However, to the best of our knowledge, it is the first study to demonstrate that 2-weeks after cessation of post-exercise sauna bathing, HA adaptations, including resting and peak core body temperature, mean skin temperature and HR do not return to baseline levels. Therefore, we can now conclude for athletes who live in temperate conditions, post-exercise sauna bathing as part of a routine training plan over a period of 3-weeks is not only sufficient at generating HA adaptations in core temperature, skin temperature and HR but also sustains these key HA adaptations for 2-weeks.

This supports current recommendations to use post-exercise sauna bathing as an effective and pragmatic alternative method of HA, and an effective ergogenic aid (Stanley et al., 2015; Scoon et al., 2007). Post-exercise sauna bathing is believed to eliminate the impracticalities of other HA methods including increased physiological strain from exercising in the heat, compromised training and limited access to artificial methods such as an environmental chambers. The intermittent style is particularly favourable due to its flexibility and minimal impact on training unlike the challenges associated with traditional active HA protocols. Therefore, it can be advised to help athletes taper for competitions in hot environments.

7. List of References

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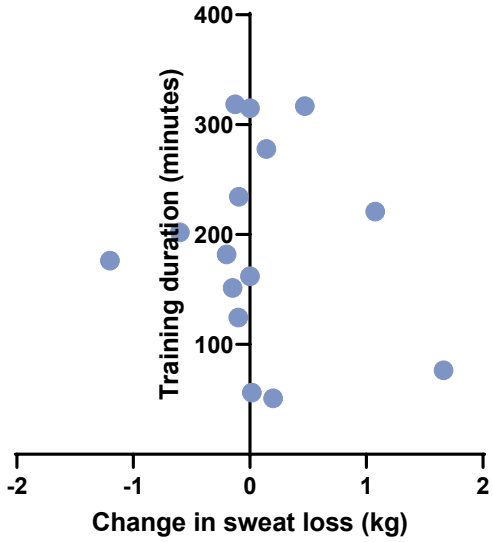
8. Appendix

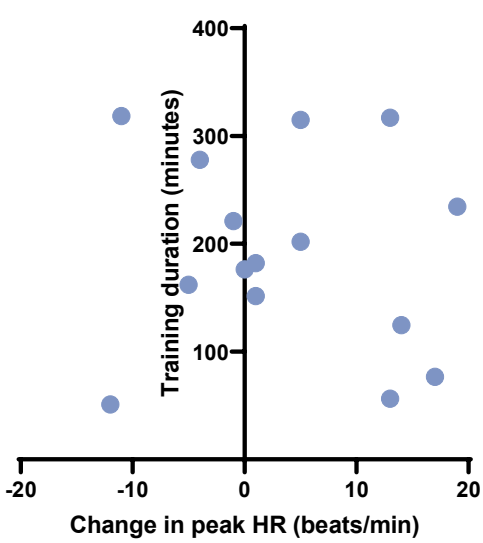
8.1.Correlations

Correlations between training duration across the decay period, participants' $\dot{V}O_{2max}$ and change in dependent variables from Post to Decay are depicted in Fig. 10a, b, c, d. There was a very weak negative correlation between minutes of training and sweat loss, r(13) = -0.13, p = 0.67. There was also a very weak negative correlation between minutes of training and peak HR, r(13) = -0.12, p = 0.67. Furthermore, there was a very weak negative correlation between Vo2 max score and sweat loss, r(14) = -0.03, p = 0.92. Lastly, there was a very weak positive correlation between Vo2 max score and peak HR, r(17) = 0.27, p = 0.27.

a)



b)



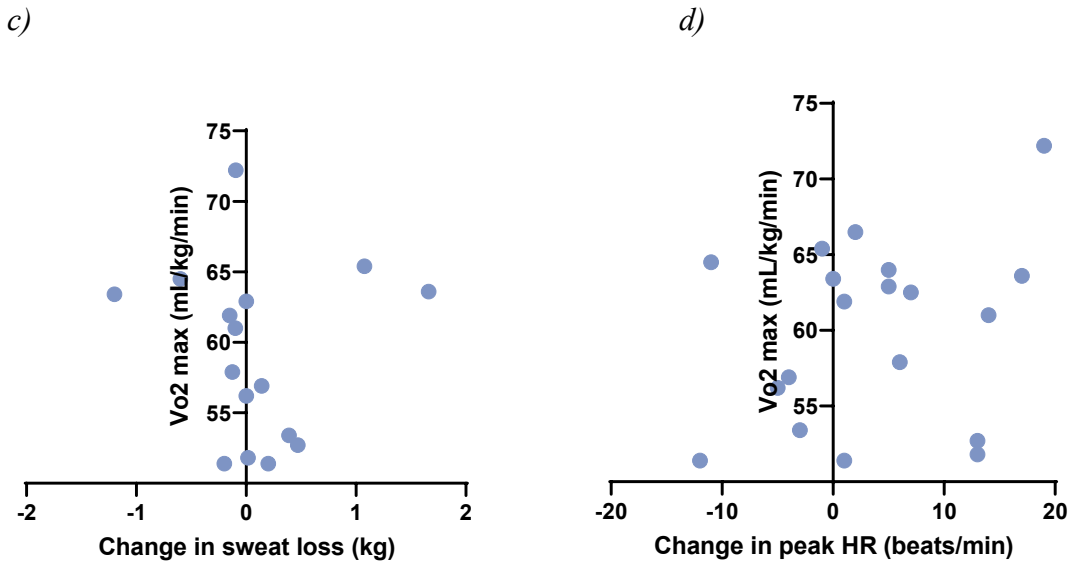


Fig 10. Correlations a) training duration and change in sweat loss b) training duration and change in peak HR c) Vo2 max and change in sweat loss and d) Vo2 max and change in peak HR. Training duration and change in variables are taken from post to decay. Data is presented as individual data points.