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Post exercise Sauna bathing is an effective method of heat acclimating middle distance and endurance runners

by

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Abstract

This study investigated whether post-exercise sauna bathing can effectively heat acclimate middle distance and endurance runners. 16 runners (age 20 ± 2 years, BMI 21.3kg/m² ± 1.2kg/m², 11 females: 5 males) performed a heat tolerance test (HTT, 30 minutes running at 9kph, 2% incline; 40°C, 40%RH) pre, 3 weeks and 7 weeks after an heat acclimation (HA; 30-minutes post-exercise sauna bathing (105-108°C) 3±1 times per week) or control. Measures included rectal temperature, skin temperature, sweat loss, heart rate, and forearm blood flow. Perceptual responses (perceived exertion, thermal comfort and thermal sensation) were also recorded.

3 week cohort: (n = 16, 10 Sauna, 6 Control). Following HA, sauna participants showed significantly reduced core temperature (p = .023), skin temperature (p = .020), HR (p = .020), increased sweat sensitivity (p = .032) and improved perceptual responses (all p = <.01) during the HTT. Controls saw no significant changes in any measure.

7 week cohort: (n = 7, 5 Sauna, 2 Control). Core temperature (p = <.01), skin temperature (p = .03), exercising heart rate (p = .017) and perceptual measures (all p= <.01) during the HTT were further reduced in sauna participants. Additionally post-exercise forearm blood flow and forearm blood flow sensitivity decreased from pre-intervention to 7 weeks (p = .015 and p = .008 respectively). Controls saw no significant changes in any measure.

This study found 3 weeks of post-exercise sauna bathing to be an effective heat acclimation strategy. Data from the 7-week cohort shows some evidence that continuing HA from 3 weeks to 7 weeks results in further adaptation.

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3W	3 Week
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ANOVA	Analysis of variance
FBF	Forearm Blood Flow
HA	Heat Acclimation
HR	Heart Rate
HTT	Heat Tolerance Test
HWI	Hot Water Immersion
LDF	Laser Doppler Flow
LPT	Lactate Profile Test
LTHA	Long term Heat Acclimation
MTHA	Medium Term Heat Acclimation
PBF	Peripheral Blood Flow
PV	Plasma Volume
RH	Relative humidity
RPE	Rate of Perceived Exertion
SD	Standard Deviation
SKBF	Skin Blood Flow
STHA	Short Term Heat Acclimation
SV	Stroke Volume
T-rec	Rectal temperature
T-sk	Skin Temperature
VO2 Max	Maximal Oxygen Consumption
VOP	Venous Occlusion Plethysmography

1. Introduction

Endurance sports such as running, cycling, triathlon and pitch-based sports are primarily conducted in outdoor environments. For many, being exposed to the elements is a part of the appeal of their sport, however this exposure can pose challenges. In the world of 'ultra-events' challenging environmental conditions can even be a defining feature of the event itself, for example the 'Marathon des Sables' which is conducted in the heat of the Sahara Desert. Whilst recreational exercisers have autonomy in their exercise regime, competitive athletes do not. Governing bodies are responsible for sanctioning competitive events, with athletes being obliged to compete in order to maintain their elite status. The 2019 IAAF World championships held in Qatar received criticism from athletes due to the problems created by extreme heat. 28 of the 68 starters of the women's marathon retired, with many of the competitors requiring medical attention for heat related illness such as cramping and collapse. In severe cases heat illness symptoms can progress to exertional heatstroke, potentially resulting in organ failure and central nervous system dysfunction that can prove fatal (Armstrong et al., 2007). In the 2019 World Championships even athletes who did not suffer from heat related illness recorded finishing times for the marathon and other endurance events that were notably slower than previous championships, with the women's marathon time being the slowest ever recorded at a world championships (World Athletics | Marathon Result | IAAF World Athletics Championships, Doha 2019, 2019).

The 2019 IAAF championships were not a one off as previous large-scale global events have also been affected by heat, including the 2004 Athens Olympics, 2008 Beijing Olympics and 2016 Rio Olympics. Looking forward to 2020 there is concern that environmental conditions in Tokyo may become dangerous for athletes to compete in. Concerns are so great that the

marathon has been relocated to Sapporo, a city 500 miles north of Tokyo with a cooler climate.

Some sports operate as global tours such as the ITU world triathlon series and cycling's UCI World Tour. Trans-meridian travel for such events can result in 'jet lag' whereby the circadian rhythms of athletes are disrupted, resulting in poor sleep and potentially impaired athletic performance (Lee & Galvez, 2012). In addition to jet lag sudden travel to a region of a drastically hotter climate could prove problematic for athletes; especially those who live and train in cooler climates. The negative impacts of travel on athletes becomes even greater with cross-hemisphere travel where the seasons are opposing, and competitors of the hosting nation may benefit from natural acclimatisation (Sawka & Coyle, 1999), putting visiting athletes at a disadvantage. Cycling's 'Tour Down Under' is held in the heat of the Australian summer where temperatures regularly exceed 40°C. Riders, travelling to compete from a European winter, typically struggle to perform well in the race and this is reflected by the event having only 2 northern hemisphere winners in the last 10 years, despite European dominance in the sport overall (Santos Tour Down Under 2019 Results, 2020). Whilst other factors will be of influence it is likely that heat adaptation is a major contributor to performance in this race.

The significance of heat on sporting competition is becoming ever greater as climate change is producing more cases of extreme weather, with increasing frequency, intensity and duration of observed global heatwaves and warm spells (Perkins et al., 2012). The implication being that competition in typically hot regions may become hotter still, and even more physiologically challenging. Another implication is that heat related performance impairment and illness may become a risk in countries that have historically been cooler.

Actions can be taken to address the challenge created by heat. Organisers can shift start times to allow events be held at the coolest time of day, and in the case of the 2022 Football World Cup in Qatar the event itself has been moved from July to November when the conditions are significantly cooler. There are also technological solutions to overcome heat such as stadium air-conditioning, and in the case of the Tokyo 2020 marathon a heat reflective running surface. Though these solutions provide some relief under some circumstances it is unavoidable that athletes will still have to compete under challenging, hot conditions. Heat acclimation is a process of an individual exposing themselves to an artificial heat stimulus using a structured approach, resulting in adaptations that improves their capacity to perform sustained physical work in a hot environment (Periard et al., 2015). Heat acclimation is therefore an important strategy to employ if athletes want to minimise the debilitative effects of heat on their athletic performance (Racinais et al., 2015).

There are numerous protocols to choose from when deciding to heat acclimate, however many of these protocols rely on the use of a dedicated environmental chamber. These chambers are expensive to operate and not widely available. Many athletes will not be able to access an environmental chamber owing to the cost and limited availability. Another option is for athletes to arrive at the competition environment days or weeks prior to competition. This allows athletes to recover from any potential jet lag, but crucially this time would allow for acclimatisation to the environment. Meaningful physiological adaptation has been shown to occur with short term HA ($\leq$ 7 days) (Tyler et al., 2016), so for time limited athletes/teams this would be a good option. Casadio et al. (2016) recommend 1 to 2 weeks of consecutive day HA in the environmental conditions of competition as best practice, but acknowledge under real world conditions this practice is unlikely due to the added stress to athlete training load, cost of travel, logistics and potential need to attend other competitions. A

recommendation of Casadio et al. (2016) is to use post-exercise sauna as a means to overcome these limitations, however research in this area is lacking; making the current study highly relevant.

In recent years passive heat acclimation strategies have been developed (Heathcote et al., 2018), with post-exercise sauna bathing being one strategy. Sauna based acclimation could serve as a more practical mode of acclimation than chamber acclimation (Casadio et al., 2016) as saunas are commonly found at gyms, leisure centres and swimming pools; making them publicly available at a reasonable expense. At present post-exercise sauna bathing has only been investigated by two studies. Scoon et al., (2007) investigated the potential impact of a post-exercise sauna bathing protocol on running performance in cool conditions, but did not investigate the impact of sauna bathing on thermoregulation during exercise in the heat. Stanley et al. (2015) also conducted a post-exercise sauna bathing study, investigating whether heart rate variability could be predictive of plasma volume changes with HA. As with Scoon et al. (2007) thermoregulation during exercise in the heat was not investigated. Both studies report increases in plasma volume with HA, and Stanley reports some limited exercising heart rate data; however it is still unclear how post-exercise sauna bathing impacts thermoregulation in the heat. There is no published data from post-exercise sauna studies regarding key acclimation markers such as changes in core temperature, skin temperature, sudorific function, peripheral blood flow and perceptual responses. The current study aimed to address this gap in the literature by assessing whether post-exercise sauna bathing can effectively heat acclimate endurance athletes. The current study also attempted to establish what physiological adaptations are responsible for achieving any acclimation that occurs.

2. Literature review

2.1: What is heat acclimation, and what is its purpose?

Heat acclimation (HA) is a process whereby an individual repeatedly exposes themselves to an artificially produced hot environment in order to confer physiological adaptations that will serve beneficially in the heat. Effective HA enables an athlete to reduce the severity of performance impairment that is experienced with prolonged exercise under hot conditions (Periard et al., 2015). Heat acclimation differs to heat acclimatisation in that acclimation is induced through exposure to an artificially generated heat stimulus (environmental chamber, sauna, steam room, hot bath), whereas acclimatisation occurs through prolonged exposure to a naturally hot environment. Acclimation and acclimatisation can be grouped together using the more general term 'heat adaptation'. The focus of this study will be on acclimation and therefore the abbreviation 'HA' will refer to heat acclimation only. HA can be conducted either actively or passively with protocols typically lasting in the range of 7 to 10 consecutive day's (Tyler et al., 2016), although numerous variations of protocol exist. By the end of the acclimation period an individual should have acquired improved thermal tolerance, resulting in an improved capacity to perform physical work in a hot environment. In an occupational setting the focus of HA may be to reduce the risk of heat related illness in workers and to maximise productivity. Though health is undoubtedly important for athletes too, an athlete's priority may be to use HA as a means to aid their aerobic exercise performance. Athletes who are unaccustomed to hot environments are vulnerable to significant performance reductions when faced with competition in a hot environment, particularly in longer duration events (Nybo et al., 2014; Guy et al., 2015). Heat acclimation is important for athletes competing in

hot environments as it can reduce the performance decrement induced by the heat, aiding sporting performance (Casadio et al., 2016).

2.2: History and Background of HA

The concept of heat adaptation is not new, the first known report regarding human heat acclimatisation was written by James Lind back in 1786 (Lind, 1786, as cited in Periard et al., 2015). Research into HA in relation to athletic performance is however a fairly recent development; with little athlete-focused research conducted before the new millennium. Most HA protocols were developed for occupational and military purposes and have later been adapted for application under athletic contexts (Periard et al., 2015), though such protocols may not be appropriate for athletes to follow due to their lack of specificity. Early HA protocols were targeted for modest workloads in populations less fit than athletes. Furthermore the demands of the military and other occupations differ to those of athletes. In recent years, more athlete focused research has been conducted. The purpose of such research is to find HA protocols that elicits the greatest performance benefit to athletes.

At present there is an increased drive to generate and validate protocols that are not only effective from a physiological perspective but also make pragmatic sense and are specific to the needs of the athlete. Passive HA protocols attempting to fulfil these demands are currently emerging (Racinais et al., 2017; Pallubinsky et al., 2017; Zurawlew et al., 2016; Zurawlew, et al., 2018; Heathcote et al., 2018). Though meta-analyses and systematic reviews on HA do exist there is still no clear consensus on the most effective protocols due to the large methodological differences between studies (Tyler et al., 2016). The most appropriate form of

HA is likely the method that best meets the specific needs of the athlete, such as their available time, resources and competition environment (Pryor et al., 2019).

2.3 Physiology of Heat Acclimation

2.3.1: Summary of overall physiological responses

Effective HA entails a multitude of physiological adaptations, with each adaptation contributing to what can be described as a 'cocktail effect'. Each adaptation may be of relatively modest individual effect but when integrated with other adaptations the improvement in thermoregulation can become highly significant. The combined effect of individual adaptations to a HA stimulus can be seen by a reduction in the core temperature of a person when exposed to heat stress (Sawka et al., 2001). Reductions in core temperature are therefore the easiest and most reliable means to compare the efficacy of HA protocols. Currently no study has assessed the efficacy of post-exercise sauna bathing on thermoregulation, however some studies have investigated the efficacy of other passive based HA protocols.

Though Scoon et al. (2007) and Stanley et al. (2015) applied post exercise sauna bathing protocols, neither of these studies measured changes in body temperature change. One previous study (Leppaluoto et al., 1986) has examined changes in rectal temperature with HA following a passive sauna exposure. Participants underwent 4 x 30 minute of sauna exposures for 7 days and showed a significant reduction in rectal temperature (-0.5°C) by the end of the seventh day (Table 1). Shido et al. (1999) and Pallubinsky et al. (2017) also applied passive hot air exposure protocols, but used lower temperatures over longer durations (Table 1). Despite the greater accrued heat exposure time in these studies, post-HA rectal temperature reductions were notably less than those reported by Leppaluoto et al. (1986). Zurawlew et al.

(2016; 2018) applied the method of Hot Water Immersion (HWI) for a HA intervention (Table 1). As with the current study, exercise was performed immediately before HWI, resulting in a pre-elevation of core temperature prior to starting HWI. Following 6 days of HA intervention, rectal temperature reductions were greater than those reported by Shido et al. (1999) and Pallubinsky et al. (2017), but not as great as Leppaluoto et al. (1986). Brazaitis & Skurvydas (2010) also conducted a HWI immersion HA study that caused significant rectal temperature reductions post-HA (Table 1). Unlike the studies of Zurawlew et al. (2016; 2018) the study did not use pre-HWI exercise, thus core temperature was not elevated upon commencement of HWI. It could be postulated that the lack of prior exercise resulted in a lower thermal load than Zurawlew's protocol, resulting in a smaller reduction in post-HA rectal temperature. A summary of the described studies is presented below in Table 1.

Table 1. Summary of previous passive HA studies that have assessed the difference in rectal temperature pre and post passive HA using a form of heat tolerance test (HTT).

Study	**HA Protocol**	**HTT details**	**Mean Rectal Δ°C ± SD or CI**
Pallubinsky et al., 2017	Passive HA (air) 6h/day, 7 days	Resting	-0.14°C ± 0.15
Leppäluoto et al., 1986	4 x 30 mins a day sauna (80°C), 7 days	Resting	-0.5°C ± 0.2
Shido et al., 1999	Passive HA (air, 46°C) 4h/day, 10 days	Resting	-0.19 to -0.24°C ± N/A
Zurawlew et al., 2016	Post-exercise HWI (40°C) for 40 mins per session over 6 days	Active, 45 min run at 65% VO2 Max	-0.36°C (CI -.24 to -.49)
Zurawlew et al., 2018	Post-exercise HWI (40°C) for 40 mins per session over 6 days	Active, 45 min run at 65% VO2 Max	-0.43°C to -0.47°C ± N/A
Brazaitis & Skurvydas, 2010	HWI 45min, 7x in 14 days	Resting, 45 min HWI at 44°C	-0.3°C ± N/A
			Mean = - 0.33°C

Included studies investigated passive HA, assessing rectal temperature throughout a fixed condition HTT.

Due to the inherently integrated nature of the body's autonomic responses it can be difficult and potentially inappropriate to simply analyse each physiological adaptation as an independent entity, in fact such separation of responses would result in an oversimplification and potential misunderstanding of the matter. This section will therefore discuss the physiological underpinnings of heat acclimation, discussing each adaptation whilst also evaluating adaption as a more holistic, integrated response. Evidence is discussed from a spectrum of studies, including active and passive protocols; however greater attention is given to passive studies given their greater relevance to the current study.

2.3.2: Principles of thermoregulation and adaptation

The thermoregulatory system is a homeostatic control system whereby the body's effector mechanisms act to maintain the 'set point', a temperature of approximately 37°C in humans. To achieve this, the level of heat input into and heat output from the body must be in equilibrium. There are four pathways through which heat can be either gained or lost from the body, and these are conduction, convection, radiation and evaporation. Under typical exercise conditions evaporative sweat losses account for approximately 80% of total heat loss, with most of the remaining heat loss coming from skin to air convective losses, though this is subject to environmental conditions and exercise intensity (Widmaier et al., 2008). Under conditions of exercising in heat, the magnitude of heat gain can exceed that of heat loss resulting in net heat storage within the body and elevation of core temperature. Should this heat imbalance persist then core temperature will elevate to a dangerously high level. At core body temperatures of 41°C serious heat illness can occur, with 43°C considered the survival limit (Widmaier et al., 2008). Heat acclimation is designed to improve the effectiveness of the body's heat loss effectors, attenuating the rise of core temperature under heat stress, thus reducing the risk of exertional heat illness (Widmaier, et al., 2008). In regard to athletic

performance HA enables greater heat loss capacity for a given workload, and at high or maximal intensities the work rate can be sustained for longer before debilitating effects of heat occur (Kenney et al., 2012).

As previously discussed HA can be conducted in numerous ways, with application of different environmental conditions, activity modalities and time courses. One factor that must be consistent is that the HA protocol must be strenuous enough to create a large enough homeostatic disturbance to result in physiological overload (Taylor, 2014). Furthermore, repeated HA sessions will be required to achieve a summative effect that will ultimately translate to meaningful acclimation (Taylor, 2014).

2.3.3: Circulatory modifications

Exercise alone generates an increased requirement for blood for the exercising muscle mass, and exposure to heat alone generates an increased requirement for blood in the peripheral tissues in order to dissipate heat. When exercise and heat are combined the demand for increased perfusion of both the active muscle and peripheral tissues becomes great, and results in elevated cardiovascular strain (Kenney et al., 2012). This strain can become so great that the distribution of the cardiac output becomes compromised, ultimately resulting in a trade-off between maintaining exercise intensity at the expense of hyperthermia or maintaining a stable core temperature at the expense of a reduced exercise intensity (González-Alonso et al., 2008).

Peripheral circulatory adaptations have been investigated for decades and are relatively well understood. The response of the peripheral vasculature is dependent on the conditions under which testing is conducted, with peripheral blood flow varying based on environmental

conditions and importantly whether testing is conducted using fixed or relative work methods (Roberts et al., 1977).

Eichna et al., (1950) heat acclimated three young men and conducted fixed work heat tolerance test (HTT) testing pre and post acclimation using a treadmill walking protocol. The fixed work HTT protocol involved 5x 10 minutes of walking at 2.5mph at a 2.5% gradient, with 2-minute intervals between. Environmental conditions were 50.6°C and 15%RH. Results showed that the reduced rectal temperature (T-rec) with HA was mainly the result of improved heat elimination through sweating. An increase of 11% in sweat rate accounted for >75% of the heat reductions, with the rest of the reductions attributed to reductions in metabolism. Improved sweating function resulted in cooler skin temperatures with HA, which widened the internal temperature gradient from core to skin, increasing heat transfer through the bloodstream; improving the removal of excess deep body heat. This improved heat transfer permitted a lesser volume of blood to carry the same amount of heat, thus reducing the requirement for vasodilation of the periphery, which was 70% of the Pre-HA value. These findings are of value, however peripheral blood flow (PBF) was not measured directly, but was estimated using the Hardy and Soderstrom formula. The study acknowledged that results may not be quantitatively accurate.

A marginally later study by Wyndham (1951) used the direct method of venous occlusion plethysmography (VOP) of the forearm as an indicator of PBF. Plethysmography served as a better indicator than a formula based estimation but the plethysmograph used was a water based system, not the more accurate mercury based strain gauge system later introduced by Whitney in 1953 (Whitney, 1953). As the same equipment was used throughout the study however, any patterns in Wyndham's results should be valid. Wyndham's results show reductions in forearm blood flow (FBF) following HA, and these were associated with

increased sweat output as well as reduced skin and rectal temperatures when working at the same absolute workload and conditions. Wyndham's results show strong support for the results of Eichna et al. (1950), with Wyndham agreeing with Eichna that the reduction in FBF was largely the result of improved sweating cooling the skin, widening the internal thermal gradient, thus improving heat dissipation from core to periphery. Wyndham also tested participants in a variety of hot conditions. When tested at the same fixed exercise intensity but under higher severities of external heat, FBF was always higher than in cooler conditions. However, acclimation still reduced the FBF value at each heat condition relative to the pre-acclimation state.

Whitney (1954) conducted a study seeking to better establish the peripheral blood flow response to heat and acclimation under fixed work conditions. Whitney acknowledged the findings of Eichna and Wyndham's experiments but felt compelled to conduct further study, especially given the contradictory results obtained by Scott et al., (1940). Scott's results were likely due to the measurement of finger blood flow, which varies in response to that of the arm. Modern venous occlusion plethysmography practices exclude hand blood flow being included in FBF measurements (Wythe et al., 2015). Whitney employed a mercury in rubber strain gauge of his own creation, this form of strain gauge is used in the modern day (and the current study) and is sometimes to referred to as a 'Whitney strain gauge'. Forearm Blood flow (FBF) readings from this venous occlusion plethysmography method show changes in volume related to the whole forearm. The VOP procedure is designed so that FBF readings are taken from an immobilised arm, which ensures forearm muscles remain at rest and therefore muscle blood flow remains constant throughout the measurement. This method means that any changes in forearm volume detected by VOP are the direct result of a

modified blood flow to the peripheral vasculature within the forearm and not changes in muscle blood flow (Caldwell et al., 2016; Charkoudian et al., 2003).

HA was active, involving ergometer cycling at 20 minutes exercising followed by 20 minutes of recovery, which was repeated a further 2 times. Conditions were 45°c and 30%RH. VOP recordings were obtained 15s post-exercise after each bout. Results were obtained from 4 healthy men of the Royal Navy. Whitney reported that all 4 participants showed reduced FBF with an increased number of exposures to heat. Plethysmography data is inconsistent however, with sometimes severe intra and interpersonal changes. Whitney chose to compartmentalise the arm and hand in only one of four participants and provides minimal discussion for the 'forearm only' data. The irregularities in the data are likely to stem from the influence of the hand circulation and its arteriovenous shunts (Walløe, 2015; Wythe et al., 2015). Whitney applied an assumption of equal volume elasticities of the vessels of the forearm and hand, and acknowledges this as a potential source of error. Interestingly Whitney also recorded skin and rectal temperatures, sweat rate and metabolic rate; but found no change in these variables. This suggests the protocol was not sufficient for inducing heat acclimation or there were fundamental methodological issues resulting in type 2 error. It is surprising that reductions in FBF were reported in all participants despite the apparent lack of adaption in other physiological variables; especially since Eichna et al. (1950) and Wyndham (1951) reported clear associations between reduced FBF and increased sweat outputs and reduced skin and rectal temperatures in their studies. The lack of acclimation markers in Whitney's study calls into question the validity of the FBF data.

Up to now the studies presented have involved active acclimation, however Fox et al. (1962) assessed changes in PBF following a passive HA protocol; specifically hot water bathing. As the current study is investigating Sauna based acclimation Fox's passive water bathing study

is of greater relevance than active acclimation based studies. The heat tolerance test Fox applied was however not a fixed workload protocol like the previously mentioned studies, consequently the results are not directly comparable to fixed work studies. The HTT involved hot water immersion, with bath temperatures being increased over time until a core temperature of 38.5°c was reached, at which point the experiment was terminated. Areas of the body exposed to air were insulated using a vapour suit. The purpose of this was to prevent sweat evaporation in order to negate sweat adaptations and to allow similar rates of heat gain between acclimated and non-acclimated subjects. Removing the influence of sweat isolates the effect of HA on FBF alone, however this condition is un-natural and does not represent real world thermoregulatory responses. Nonetheless, following acclimation Fox reports increases in FBF of 30% during the HTT when body temperatures are matched for the acclimated vs non-acclimated condition. Acclimated subjects were able to tolerate higher bathing temperatures before reaching the 38.5°c cut-off, and this was likely associated with lower initial body temperature and threshold shifts with HA. The findings of Fox et al. (1962) support that of Wyndham et al. (1951) in that when core temperature is matched between acclimated and un-acclimated states FBF is higher in acclimated individuals to maximise heat elimination. Fox's findings do not directly reveal what would happen to FBF with HA under fixed work and fixed environment conditions, however Wyndham (1951) found a reduction in FBF under such conditions, and furthermore 'Since arm blood flow is higher at any given internal temperature after acclimation, the lower blood flow which is reported to accompany heat acclimation must result from the lower body temperatures' (Roberts et al., 1977).

There is limited available data for the measurement of forearm blood flow using the alternative method of laser doppler flow (LDF), especially in the context of repeat measure studies applying fixed work heat tolerance tests pre and post acclimation. A potential

explanation for this is that the LDF method has the limitation of calculating flow using arbitrary values and cannot calculate absolute flow (Charkoudian, 2003). LDF values can be normalised by calculating maximal skin vasodilation, however this would require an external stimulus to provoke the maximal response, which extends and complicates a study protocol further.

Neal et al. (2016) utilised LDF in a study applying short-term HA, involving 5 days of exercise for 90 minutes per day in conditions of 40°C and 50%RH. A fixed work heat stress test was conducted pre and post-acclimation and revealed no significant change in skin blood flow with HA. Lorenzo & Minson (2010) also investigated the effect of active-HA on skin blood flow responses using the LDF method. They showed that active acclimation increased the sensitivity of forearm blood flow to a given level of stimulation, and that maximal forearm blood flow was not modified by heat acclimation. More recently, a study by Pallubinsky et al. (2017) investigated the influence of HA on FBF responses using the LDF method. Pallubinsky used a passive HA protocol, subjecting participants to mild passive heat acclimation for 7 days (Table 1). Acclimation resulted in a 28% reduction in forearm blood flow, a finding that did not agree with the study's hypothesis. Core temperature showed only a small albeit significant reduction post-HA and skin temperature was elevated in the post-HA condition relative to pre-HA. As reductions in core and skin temperature are key drivers of reducing skin blood flow it was surprising to the researchers that a reduction in FBF was observed without a reduction in skin temperature or an appreciable attenuation of core temp rise. Importantly the authors acknowledge that the limitation of the LDF technique is that the values generated are arbitrary units as they are based on an established baseline LDF value at the start of each testing session, and are not an absolute value. Despite this potential source of error from the LDF method, the finding of a reduction in FBF under the conditions of a

matched condition HTT is in agreement with the findings reported from the previously mentioned VOP studies in this section.

2.3.4: Sweating modifications:

When the body experiences warming the initial heat loss mechanism that is recruited is peripheral vasodilation, which enhances dry heat losses through radiation and convection (Sawka et al., 2011). Sweating is a secondary thermoregulatory response that is employed when the level of heat gain exceeds the capacity of dry heat loss mechanisms alone (Kenney et al., 2012). When exercising in hot conditions the level of heat gain is so great that sweating accounts for the vast majority of heat loss, and when the environmental temperature exceeds skin temperature heat can be removed only by evaporation (Kenney et al., 2012).

HA produces a threshold shift that results in the earlier onset of sweating at a lower body temperature relative to the pre-HA state (Patterson et al., 2004). The reduced threshold temperature relates to a reduction in resting body temperature with HA, the absolute amount of temperature change required to initiate sweating is unaltered (Patterson et al., 2004). Heathcote et al. (2018) reported a core temperature sweat threshold change of -0.31°C in passive HA studies as well as increases in sweat sensitivity once sweating is initiated. The earlier onset of sweating and accompanying sweat sensitivity improves heat loss in the early stages of exercise, attenuating the rise in core temperature, reducing the negative thermoregulatory implications. Improved heat loss from sweating results in lower skin temperatures, which would ultimately serve to reduce peripheral blood flow as discussed by Eichna et al. (1950) and Wyndham (1951).

Increased sweat loss is another known adaptation to HA. Crucially, increases in sweat output are only beneficial if there is a capacity for the additional sweat to be removed by

evaporation. Unevaporated sweat that remains on the skin significantly limits heat loss, but can have a further negative consequence as excess sweat secretion leads to faster dehydration, which would impair performance and risk health (Kenney et al., 2004). As there is no sweating data from any previous post-exercise sauna studies one of the closest studies with sweat data is that of Zurawlew et al. (2016) which employed post-exercise hot water immersion. The study used consecutive day acclimation and by the 6th day of HA sweat rate was 16% higher than day 1 (p = <.01). Other passive HA studies that have used intermittent day acclimation have also shown increases in sweat output (Bonner et al., 1976; Brazaitis & Skurvydas, 2010; Shin et al., 2013), however unlike the current study none of these studies utilised exercise to pre-elevate core temperature before acclimation sessions began. The additional heat stress resulting from entering HA sessions with an already elevated core temperature may potentially result in a differing magnitude of sweat adaptation response.

The distribution of sweat output across the surface area of the body can also be modified with HA. Activation of more eccrine sweat glands can increase the degree of sweat coverage over the skin, creating a greater potential for sweat evaporation (Ravenelli et al., 2018). Furthermore there is some evidence to suggest that sweat may be preferentially redistributed with HA (Patterson et al., 2004). Patterson found HA induced the greatest increase in local sweat rates at the arms. The arms and hands have a high surface area to volume ratio, which would result in efficient sweat evaporation and therefore greater heat elimination; making the arms a logical region to experience a greater increase in sweat rate. Once again this provides a basis to say that increased sweating function will ultimately reduce peripheral blood flow, and in particular forearm blood flow. Additionally Lorenzo & Minson (2010) reported that ‘increased sweat rates will decrease skin temperature, which may limit the rise in core

temperature, decreasing SkBF requirements, and allow a greater fraction of the available cardiac output to be directed to active muscles' (Lorenzo & Minson., 2010. p. 1742).

Changes in sweat composition are reported with heat acclimation, with acclimation causing a greater reabsorption of electrolytes within the sweat gland, producing more dilute sweat. This adaptation has been consistently reported with active HA methods (Chinevere et al., 2008; Buono et al., 2007; Houmard et al., 1990; Peterson et al., 2010; Nielson et al., 1997; Kirby & Convertino, 1986; Racinais et al., 2012; Racinais et al., 2014) as well as passive methods (Dill et al., 1938; Allan & Wilson, 1971; Ogawa et al., 1982). The more dilute sweat, widens the water vapour pressure gradient, ultimately increasing the level of evaporation for a given set of skin and ambient air temperatures (Taylor, 2014; Periard et al., 2015). This improved evaporative capacity would facilitate heat loss by ensuring maximal sweat removal, meaning any increases in sweat rate are utilised by the additional sweat evaporating and removing additional heat from the skin. This increased heat removal provides further reasoning as to why FBF may decrease as a result of HA.

2.3.5: Cardiovascular and haematological changes

A common adaptation reported to occur with HA is plasma volume expansion, with adaptation evident after just a couple of HA exposures (Periard et al., 2015). Exercise in a hot environment has been shown to increase the secretion of the hormone aldosterone (Francesconi et al., 1983; Kosunenkj, 1976). Elevations of aldosterone cause an increased sodium reabsorption during the sweating process, explaining the previously mentioned sweat dilution. The retention of sodium modifies the osmotic gradients in and around the vasculature resulting in a net movement of water into the blood, thus expanding plasma volume and overall blood volume (Sawka et al., 1996). Prolonged exercise and heat will result

in profuse sweating and therefore dehydration, causing haemoconcentration. Albumin concentration in the vascular space increases with dehydration, increasing colloid osmotic pressure which ultimately draws water into the vascular space; further contributing to plasma volume expansion (Kirby & Convertino, 1986; Bonner et al., 1976; Kissling et al., 2019).

Another key marker of heat acclimation is a reduction in heart rate at rest and particularly during exercise (Tyler et al., 2016). This occurs because of the plasma volume expansion causing an increase venous return, thus greater stroke volume (Wendt et al., 2007; Periard et al., 2016). As cardiac output is the product of stroke volume (SV) and HR, HR decreases as a consequence of an elevated SV for a given cardiac output.

Plasma volume (PV) expansion has been artificially induced in humans through albumin infusion and this expanded PV alone, without HA, is capable of lowering HR during exercise in the heat (Sawka et al., 1983). Data from Rowell 1967; Wyndham et al., 1968 and Nielson et al., 1993 also support the concept of PV expansion driving increased SV and reductions in HR. A separate study by Nielson et al., (1997) found no change in SV despite an increase in PV, a finding in contrast to the other studies. Crucially though, Nielson's 1997 study was conducted in humid conditions whereas the other studies were in hot, dry conditions. This suggests that PV expansion may be responsible for reducing exercising HR under hot, dry conditions; but not humid conditions. This difference in response may be due to the differing circulatory demands and greater sweat losses that occur with exercise in humid environments.

Though there are only two existing studies regarding post-exercise sauna bathing, both of which (Scoon et al., 2007; Stanley et al., 2015) measured changes in plasma volume with HA. Both studies reported increases in plasma volume with HA. Scoon et al., (2007) reported a plasma volume expansion of +7.1% and a resulting increase in total blood volume (+5.6%).

Scoon also found a smaller increase in red cell volume (+3.5%), however given the greater proportion of plasma in the blood and the greater percentage increase in PV the overall effect of the HA was a haemodilution, as shown by a 2% reduction in haematocrit. Stanley et al., (2015) also tracked changes in plasma volume with a post-exercise sauna bathing protocol. Stanley measured PV daily, observing a peak PV increase of +17.85% after 4 days of sauna intervention. Despite such a large early expansion, PV expansion reduced towards pre-HA levels after 4 sauna exposures, and was not significantly different to pre-HA levels at exposures 7,8 and 9. Reductions in PV expansion following an initial rise are believed to be the result of the constant workload model, where a forcing function is not sustained over time (Wyndham et al., 1968).

The results of Scoon and Stanley's studies leaves little doubt that post-exercise sauna bathing can induce increases in plasma volume; although, other key phenotypic thermoregulatory HA adaptions were not assessed in these studies. A key benefit of expanded plasma volume is improved cardiovascular stability during exercise in heat. This is perhaps best demonstrated by reductions in syncope rates as HA progresses (Bean & Eichna, 1943). Improved cardiovascular stability therefore allows HA individuals to better maintain exercise and control thermoregulatory responses. In the context of thermoregulation alone an expanded PV may be of benefit by raising the specific heat capacity of the blood, improving heat transfer from muscle to skin, facilitating dry heat loss from the periphery without the need for increases in PBF (Sawka et al., 2011). This is with the assumption that environmental conditions allow for dry heat loss.

2.3.6 Perceptual measures

Perceived exertion, thermal comfort and thermal sensation can be used as perceptual markers of exercise and thermoregulatory stress. Effective HA would result in adaptations that produce a reduction in RPE and improvements in thermal sensation and comfort (Periard et al., 2015; Heathcote et al., 2018). There is limited data regarding perceptual changes with HA, with data becoming even more limited when analysing studies that have assessed perceptual responses under fixed-work, sub-maximal testing conditions.

Rating of perceived exertion (RPE) is typically expressed using the Borg scale (Borg, 1980), where exertion is reported on a scale of 6 to 20, where at a score of 6 exertion is lowest and at a score of 20 exertion is maximal. Predominantly, HA causes a reduction in RPE when exercising in the heat. Armstrong et al. (2005) reported RPE to be significantly reduced post-HA, however the HA intervention was conducted with physical training in inactive females therefore it is likely improvements in participant fitness from this training modified RPE. Neal et al. (2015) and Kelly et al. (2016) both conducted 5-day active HA interventions in participants who were already trained and maintained normal training throughout the short study period, meaning non-HA related fitness changes were unlikely. Both these studies reported RPE to significantly decrease following HA when assessed using a fixed work sub-maximal HTT pre and post-HA. Cotter et al. (1997) also conducted a 5-day active HA intervention, however in contrast to Neal et al. (2015) and Kelly et al. (2016) there was no reported change in RPE or any other perceptual measure. The lack of change in RPE can likely be explained by the study design, as unlike Neal and Kelly's studies Cotter et al. (1997) did not apply a standardised fixed-work HTT pre and post HA. Instead physiological and psychological markers of HA were measured during the first and last days of an isothermic HA session where exercise load was manipulated to maintain a 1.4°C tympanic temperature

elevation. Despite the lack of work standardisation the amount of work completed was not significantly different post-HA, suggesting the HA protocol was not highly effective; potentially further explaining the lack of change in RPE and other perceptual measures.

Undoubtedly the most relevant studies to have assessed the effect of HA on RPE are those of Zurawlew et al. (2016) and Zurawlew et al. (2018), as both these studies employed a post-exercise passive HA protocol. Furthermore HA was assessed using a fixed work HTT pre and post-HA. The 2016 study reported significant reductions in RPE post-HA, a finding which was replicated in the 2018 study which employed an identical HA protocol. As there is no data regarding the effect of HA on RPE with post-exercise sauna bathing these findings from Zurawlew et al. (2016, 2018) serve as the most relevant literature from which a hypothesis can be established.

Often thermal comfort and thermal sensation are measured in conjunction with RPE. The aforementioned studies from Neal et al. (2015) and Kelly et al. (2016) reported improvements in thermal comfort with HA. Conversely, Cotter et al. (1997) reported no change in either thermal comfort or thermal sensation; however as mentioned previously this is likely a product of the study design and efficacy of the HA protocol. The studies from Zurawlew et al. (2016, 2018) which reported reductions in RPE with HA also reported significant reductions in thermal sensation, though thermal comfort was not assessed. Peterson et al. (2010) conducted a HA study applying 4 consecutive days of high intensity interval exercise on cycle ergometers in 30°C and 60%RH conditions, with HA assessed using the fixed-work, sub-maximal HTT method. Post-HA thermal sensation, which was assessed using the same 1 to 13 scale as the current study showed improvement, reducing significantly from 10.5 (± 0.60) down to 9.2 (±1.0). Thermal comfort which was assessed using a different scale to the current study also showed significant improvement. Racinais et al, (2017) found no significant

changes in thermal comfort or thermal sensation following an 11-day passive HA protocol (1h per day exposure to 50°C and 50%RH for 11 days). In this study as in Cotter et al. (1997) study, perceptual measures were not measured under fixed work submaximal conditions. Instead, thermal comfort and sensation were measured during a 2-minute maximal voluntary contraction task which is not comparable to the sub-maximal aerobic exercise conditions of the current study.

2.3.7 Metabolism

Multiple studies have found heat acclimation to lower the metabolic cost of sub-maximal exercise, resulting in improved exercise efficiency (Tyler et al., 2016). As neither Scoon et al. (2007) or Stanley et al. (2015) investigated metabolism it is unknown whether metabolic changes occur with post-exercise sauna bathing specifically. Leppaluoto et al. (1986) did report reductions in metabolic rate with a sauna bathing HA protocol, however metabolism was assessed at rest and therefore did not reveal any information about metabolic function during exercise, or exercise efficiency. The current study did not assess metabolism. It appears possible that if metabolic adaptations occur were to occur with HA in the current study this may be indirectly expressed by participants reporting a reduced perception of effort (lower RPE score) during the post-HA HTT. This is because a reduction in metabolism means a reduced oxygen cost for a given exercise, increasing exercise efficiency.

2.3.8 Performance

The purpose of HA in the context of exercise and sport is to improve athletic performance. Performance changes with HA can however be measured from different perspectives. If defining performance as the maximal amount of work completed (aerobic fitness) then there is evidence to show that heat acclimation improves this form of performance in both hot and

cool conditions. Lorenzo et al. (2010) heat acclimated 10 cyclists using a 10 consecutive day active HA protocol. Post-HA participants demonstrated significant increases in VO_2 max, lactate threshold and improved time-trial performance in both hot (38°C) and cool testing conditions (13°C). A later study by Zurawlew et al. (2016) used hot-water immersion to acclimate participants. As with Lorenzo et al. (2010) performance improvements were found post-HA in the heat, however in contrast to Lorenzo et al. (2010) there was no reported performance change in cool conditions. Most relevant to the current study are the findings of Scoon et al. (2007) who applied a post-exercise sauna protocol. The study reported a 32% increase in run to exhaustion time, equating to a 1.9% reduction in 5km run time. The improvement in running performance was attributed primarily to the significantly increased plasma and whole blood volume following acclimation.

Performance in regards to HA can also be seen from the perspective of thermoregulation alone. An improved thermoregulatory performance under fixed-work, sub-maximal exercise conditions would be best indicated by reductions in core temperature (Sawka et al., 2011). Such performance changes in thermoregulation with HA under passive conditions are described in Table 1.

2.4 HA Protocols

2.4.1: Rationale for protocol selection

Coaches or athletes looking to use HA should always consider the pragmatic value of such an addition to their training regime. Protocols that are too time consuming, complicated or excessively strenuous may impede the athletes training and have debilitative rather than facilitative effects on the athlete (Pryor et al., 2019). Factors that require consideration include: acclimation modality, frequency and duration; each of which will be discussed.

2.4.2: Modality - Active versus Passive HA

Active HA requires the acclimating individual to be exercising during the heat exposure period, although there is no definition for what level of physical activity classes as 'active'. The most common modalities of exercise are ergometer cycling and treadmill running/walking, although some studies have employed 'bench stepping' and 'circuit training' protocols (Tyler et al., 2016). Due to the additional stress generated by the heat stimulus, exercise intensity is typically low to moderate with active protocols. High environmental temperatures combined with high intensity exercise would result in the fast onset of hyperthermia, and is typically avoided.

The majority of the literature on HA is focused on research into active HA, as opposed to passive HA (Tyler et al., 2016; Heathcote et al., 2018). Before evaluating the efficacy of active HA it is worth considering that though active HA is more highly researched it is a form of acclimation that is more difficult to practice. This is due to the requirement of an environmental chamber capable of being heated and humidified to the required level, as well as holding exercise equipment. As a result active HA is less likely to be a viable option for amateur athletes who lack resources or the sufficient financial support. An additional consideration is how to integrate active HA sessions into an athletes' training schedule. The intensity of exercise during active HA sessions is typically lower than normal training intensity due to the need to maintain a safe core temperature. As most protocols require acclimation on consecutive days this will result in a significant accumulation of time training at low intensity, and this may interfere with the larger training plan for an athlete (Casadio et al., 2016). Due to decay, HA is most effective when conducted almost immediately prior to

competition (Daanen et al., 2018), however this same period can coincide with the tapering of an athletes training. There may be a clash between the required exercise intensities of HA and that of the tapering period for the athlete, and this may result in compromised training quality (Casadio et al., 2016; Pryor et al., 2019). A potential solution is to conduct active HA two to three weeks before competition, with subsequent 'top-up' sessions to maintain adaptations during the taper to competition (Casadio et al., 2016; Pryor et al., 2019). Such complexities at such a crucial stage of competition preparation may however be avoided through passive HA, as will be elaborated upon later.

There are suggestions within the literature that active HA is more effective than passive HA (Taylor, 2014; Daanen et al., 2018), and this is somewhat reflected by the increased number of studies investigating active HA. The primary reason for this belief is due to the specificity that active HA entails (Tyler et al., 2016). Exercising in environmental conditions similar to those anticipated for competition will theoretically allow for the most relevant adaptations for the individual athlete. Exercise results in a large increase in metabolic heat production, and when conducted in a hot environment the combination of endogenous and exogenous heat acts to produce a large thermal impulse, and it is this thermal impulse that stimulates adaptation when applied in a repetitive manner; ultimately accumulating to produce adaptation (Taylor, 2014). Passive HA protocols involve an athlete being exposed to high exogenous heat under resting conditions. As endogenous heat production is much lower at rest than in exercise the environmental temperature is higher in passive HA than in active HA in order to produce the required increases in core body temperature that stimulates strong thermoregulatory responses and ultimately adaptation over time. Effective acclimation may not be as simple as just raising core temperature however, as the physiological demands created by active and passive HA vary, and this may induce differing acclimation responses.

For example under matched heat stress conditions peripheral blood flow is higher in hot passive conditions than hot active conditions (Gonzales Alonso, 2008). This is due to the increased blood supply to active muscles during active HA limiting the availability of blood to supply the peripheral vasculature (Sawka et al., 2011).

2.4.3: Duration and frequency of HA protocols

HA protocols can be generally classified as short term (STHA) (≤7 days), medium term (MTHA) (8-14 days) or long term (LTHA) (≥15 days) (Chalmers et al., 2014), although there is slight variability in these definitions (Garrett et al., 2011). A simpler interpretation is short term (1 week or less), medium term, 1 to 2 weeks, and long term being over 2 weeks. A limitation of this classification is that it appears to make the assumption of consecutive days. To account for protocols that apply HA intermittently it would be more appropriate to use the term 'exposures' instead of 'days'.

Tyler et al. (2016) sought to quantify the effect of different duration HA protocols. The literature search in itself produced an interesting finding in that of the 96 studies that met the inclusion criteria only 9 studies employed long term HA, with 61 studies being medium term, and 30 being short term. The study concluded that both short and medium term HA are effective at inducing HA, however maximum acclimation is achieved through use of long term HA. Most of the thermoregulatory adaptations that occur with HA occur in the earliest stages of HA, with plasma volume expansion and reductions in HR evident following 5 to 7 exposures (Periard et al., 2015). These early adaptations can result in significantly improved thermoregulation in the heat, however a margin for further improvement still exists with LTHA as this enables more complete sweat response adaptations (Tyler et al., 2016; Daanen,

et al., 2018). Though STHA and MTHA have been shown to increase sweat rates, greater elevations in sweat rates are found with LTHA. These higher sweat rates reported with LTHA are likely due to adaptation of the sweat glands to increase their sensitivity, resulting in greater sweat output for a given temperature increase. A threshold shift that initiates earlier sweating may also contribute increased sweat losses (Taylor, 1986). It could be argued that the increased sweat losses produced from LTHA result in faster dehydration, offsetting the additional benefits of LTHA. This is debatable as increased sweat rates will allow for better thermoregulatory control, so as long as the athlete consumes fluids sufficiently this would be an optimal strategy. Furthermore there is evidence to show that HA results in an improved ability for acclimated individuals to better manage their hydration status (Periard et al., 2016).

Given that long term HA is believed to yield the most effective HA (Tyler et al., 2016; Daanen et al., 2018) it is surprising to find a lack of research interest. One possible explanation is that from a research perspective this would create a lengthy, time consuming study. All of the 9 long term studies employed consecutive day acclimation with protocols ranging from 15 to 24 days. It is understandable from a coach or athlete perspective that such a long-term consecutive day protocol may seem daunting given the discomfort of acclimating as well as increased time taken from regular training. A potential solution would be to implement long term HA protocols using an intermittent exposure method. Studies have shown that 2 or 3 days between HA exposures still results in effective acclimation (Duvnjak-Zaknich et al., 2019; Fein et al., 1975). Intermittent exposure may also reduce any fatiguing effect of HA (Periard et al., 2015).

2.4.4: Decay

A 2018 review and meta-analysis (Daanen et al., 2018) investigated the nature of decay following a HA protocol. A limited number of studies exist in this area, consequently all modalities of HA were included. It was concluded that adaptations in heart rate and core temperature decay at the rate of approximately 2.5% per day. The time course of decay in sweating adaptation was less clear, but sweating adaptations were better preserved following longer-term HA protocols; a logical finding as sudorific adaptations take longer to develop than cardiovascular adaptations. The review did not have the scope to directly compare the decay of consecutive day versus intermittent day HA studies.

Fein et al. (1975) heat acclimated participants in two groups, one group using consecutive day HA and the other acclimating every third day, with results showing no significant effect on the rate of heat acclimation between the groups. More recently Duvnjak-Zaknich et al. (2019) conducted a similar study, assigning participants to either a 'Continuous' protocol (8 HA session over 8 days) or an 'Intermittent' protocol (8 HA sessions over 15 days). As with Fein et al. (1975) participants of both protocols showed evidence of acclimation, with no significant difference in end of HTT core temperature, skin temperature or heart rate between the groups by the end of the HA protocol. Follow up testing was also conducted 2 weeks after the completion of the HA protocols, which revealed no significant difference in the rate of decay of adaptations between Continuous and Intermittent participants. Barnett and Maughan (1993) applied intermittent HA to five subjects with intervals of 1 week between HA sessions, with 4 sessions in total. Given the low number of accrued sessions and large time periods between sessions it is unsurprising that no significant changes were found in core temperature, heart rate, sweat loss, skin temperature or VO2 max; resulting in the conclusion

that no heat acclimation occurred. This is likely due to adaptation decay occurring between sessions and the short number of accrued HA sessions.

It can be concluded that long term HA protocols that induce more complete adaptation result in a longer period of benefit before HA status is lost through decay. It appears that allowing intervals of 2 to 3 days between heat exposures does not result in decay between exposures and the HA stimulus can be summated to produce meaningful adaptation. It appears that the adaptation achieved with intermittent protocols can be as efficacious as continuous protocols, however one week intervals or more are too long and will not result in HA.

2.4.5: Temperature regulation models: Constant load vs Controlled hyperthermia model

The majority of heat acclimation protocols apply a constant load method (Tyler et al., 2016), where the exercise and environmental conditions remain unchanged throughout the entire acclimation period. This method is relatively simple to conduct and has proven to effectively acclimate athletes (Tyler et al., 2016), but may not maximise their full acclimation potential. With a constant load protocol the athlete will acclimate and improve their thermal tolerance throughout the acclimation period, however as HA progresses the athlete becomes relatively habituated and the physiological stress generated becomes progressively smaller over time. Due to this the stimulus driving adaptation, though still present, is reduced at the latter stages of acclimation compared to the initial stages; potentially resulting in sub-optimal acclimation (Taylor, 2014). Taylor argues that the constant load model is commonly used in athletic training contexts due to the influence of tradition, which stems from the needs of occupational settings where workload is fairly consistent. As constant load protocols are essentially adaptations of earlier occupational based protocols, it could be argued they are built on a foundation that is fundamentally flawed for athletes (Taylor, 2014).

The ‘controlled hyperthermia’ approach, sometimes referred to as ‘isothermic’ HA or ‘thermal clamping’ is an alternative to constant load HA. The regulated variable is core body temperature, which is aimed to be kept at a constant level of elevation throughout the entire acclimation process. As the subject acclimates and improves their thermoregulatory responses the target core temperature must be met by creating more challenging conditions such as increasing exercise workload or modifying environmental conditions. This practice can be regarded as a form of progressive overload, which ensures consistently high levels of physiological stress throughout the acclimation period, and the maintenance of a strong stimulus for adaptation.

Theoretically, the controlled hyperthermia model is an appealing concept, however its practical application under real world conditions does present challenges that may deter coaches and sports scientists from its use. In order to maintain a stable, elevated core temperature a reliable measure of core temperature is needed, requiring an oesophageal or rectal thermistor; or costly ingestible thermometer pills. The impracticality and potential discomfort of these methods is problematic under real world conditions. Furthermore whilst the theory is logical in its principles there is little evidence to support the claim that isothermic acclimation creates greater acclimation than the more traditional constant load method. Gibson et al (2015) found similar adaptations between constant work and controlled hyperthermia HA protocols over both short and longer term periods. This calls in to question whether the additional logistics of the controlled hyperthermia protocol are justified.

2.5 Aims and Hypothesis

The aim of this study was to assess whether post-exercise sauna bathing can effectively heat acclimate endurance athletes. This study also aimed to uncover which physiological mechanisms may be responsible for enabling any adaptation to the post-exercise sauna stimulus. Based on a review of the relevant literature the following hypotheses were established.

Heat acclimation will result in:

1) A reduced Core (rectal) temperature during exercise.
2) A reduced forearm blood flow when measured at the end of exercise.
3) Increased sweat losses and consequently lower skin temperatures during exercise.
4) Improved cardiovascular stability, indicated by reduced exercising heart rate.
5) Improved perceptual responses (RPE, thermal comfort, thermal sensation) during exercise.
6) Significant adaptation after 3 weeks, with further adaptation evident after 7 weeks.

3. Methods

3.1: Investigators

This study was principally designed and led by Nathalie Kirby as a part of her PhD studies at the University of Birmingham. It was my personal responsibility to design and implement the VOP protocol for this study, as well as working alongside Nathalie and undergraduate students to run HTT sessions, conduct performance testing and supervise sauna bathing sessions. Data analysis for this thesis was conducted primarily by myself, with some limited shared resources. All aspects of the design of the study were overseen by project supervisors Dr Rebekah Lucas and Dr Sam Lucas. This study was also conducted in collaboration with the staff of the University's high performance centre who are involved in the regular training and testing of the athletes participating in this study.

3.2: Participants

Male and female endurance runners from the University of Birmingham Athletics Club were recruited for this study, with tables 2a and 2b showing participant characteristics. Before participation, each volunteer had to provide written consent. All protocols were in accordance with the Helsinki agreement and approved by the University of Birmingham ethics committee (ERN_18-0958). 28 participants were recruited initially, however significant dropout occurred throughout the study, largely due to training related injury. Some participants were excluded for poor sauna adherence and missing data. As a result N=16 for the 3 Week cohort (10 Sauna, 6 control). N=7 for 7 week cohort (5 Sauna, 2 control).

Table 2a: Participant characteristics for the 3 Week cohort

Characteristic	Sauna (Mean ± SD)	Control (Mean ± SD)
N	**10**	**6**
Females: Males	**8:2**	**3:2**
Age (years)	**20 ± 1**	**20 ± 3**
Height (metres)	**1.69 ± 0.00**	**1.70 ± 0.00**
Body mass (kg)	**60.9 ± 6.6**	**61.3 ± 4.3**
BMI (kg/m²)	**21.3 ± 1.4**	**21.3 ± 0.8**

Unpaired T-tests revealed no significant differences between sauna and control participants for any of the assessed characteristics

Table 2b: Participant characteristics for the 7 Week cohort

Characteristic	Sauna (Mean ± SD)	Control (Mean ± SD)
N	**5**	**2**
Females: Males	**3:2**	**0:2**
Age (years)	**19 ± 1**	**22 ± 4**
Height (metres)	**1.70 ± 0.10**	**1.71 ± 0.00**
Weight (kg)	**63.0 ± 6.1**	**62.8 ± 0.9**
BMI (kg/m²)	**21.8 ± 1.2**	**21.8 ± 0.3**

Unpaired T-tests revealed no significant differences between sauna and control participants for any of the assessed characteristics.

3.3: Study Design

This study applied a non-randomised partial cross-over design. This means participants who completed the first round of testing could volunteer to participate in the second round in the other condition following the wash-out period. Accounting for participant dropout only one participant crossed over and completed both control and sauna conditions. The first round of testing was conducted in the months of October, November and December; with the second round being conducted in January, February and March. This period avoided the warmer summer season, ensuring natural heat acclimatisation would not interfere with the study. Due to University term dates the Christmas holiday period provided an ideal washout for participants before the cross-over in conditions. The 3W cohort and 7W cohort share some but not all participants, and therefore cannot be compared. The initial study design involved one cohort, where all participants completed 7 weeks of testing, however participant drop-out and technical issues prevented this design from being used. Participants of both the control and Sauna group were part of the same running club with similar training programmes.

For participants in the Sauna intervention heat acclimation was planned as 25 post-exercise sauna sessions spread across 7 weeks, equating to 3 to 4 sauna exposures per week. Each sauna exposure was planned as 30 minutes in duration (or as long as tolerable up to 30 minutes) and was conducted immediately after participants finished their regular training run. Participants were permitted to sauna bathe after any type of run, on any day of the week. Participants were advised to distribute their sauna exposures as evenly as reasonably possible throughout the intervention, however this could not be strictly enforced due to participants' personal and training schedules. The overall study protocol can be seen in figure 1. Sauna temperature was typically between (105°C and 108°C). Participants were permitted to drink water ad libitum in the sauna.

Testing was conducted in the week before the intervention (Pre-intervention), after the third week of intervention (W3), and in the week after the intervention end (W7). This time frame was selected so that testing would occur as close as possible to a 28 day interval in order to account for and exclude the influence of menstrual related variations in body temperature in female participants. All participants were given iron supplements at 65mg daily (Nature Made®, CA, USA) starting 2 weeks prior to the pre-intervention HTT, with supplementation continuing throughout the study. Supplementation was to prevent low iron status being detrimental to health and performance, a matter of even greater importance in active females (DellaValle, 2013). Throughout the study all participants kept written records of training, which were later used to quantify training load (figures 5a and 5b). Quantifying training load allows for any potential training effects to be accounted for.

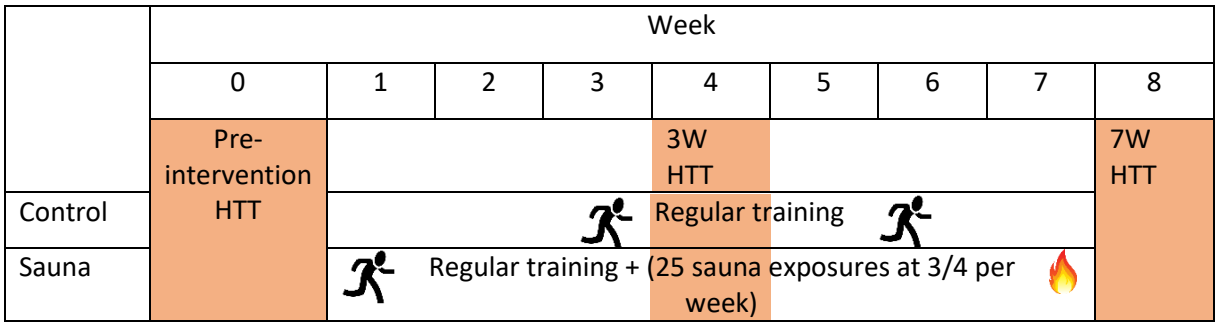


Figure 1: Schematic of study Protocol

3.4: Testing Protocol

On a testing week the performance testing and heat tolerance testing were performed on separate days, with a minimum of one day gap to allow for recovery. Scheduling logistics meant for some participants only one day of recovery was possible, however, for all participants the repeat tests at weeks 3 and 7 were conducted with the same recovery period as in the pre-intervention testing. This was to ensure recovery periods were standardised and therefore would not influence the test results. For all testing days upon arrival at the lab participants were first asked to provide a urine sample, which was used to establish urine osmolality (Osmocheck, Vitech Scientific Ltd., West Sussex, UK) and therefore hydration level. Participants with an osmolality >700mOsm/L were required to consume 300ml of water before exercise testing could begin. Participants were also fitted with a heart rate monitor (Polar Electro, Kempele, Finland) to record HR throughout testing. Pre and post HTT body mass was also recorded for each testing session.

3.5: Performance testing

This was a part of the larger study that also investigated the potentially ergogenic effects of post-exercise sauna HA on aerobic performance in cool conditions. Details of performance testing are described only relatively briefly as they were not the focus of this current thesis. Testing started with a 10 minute warm up and stretching period. This was followed by a lactate profile test (LPT). The LPT was conducted using treadmill running, and involved participants running 3-minute stages, at 1kph increments. Starting pace for the LPT was set as 5km run personal best pace minus 2kph and the test continued until the participant exceeded 4mmol/l of lactate, at which point exercise stopped. The 1kp/h speed increments were applied

to ensure the lactate threshold (4.0mmol/L) was met and exceeded without the test taking too long to conduct. Increments of 0.5kph for example would increase test time substantially which could cause fatigue and energy depletion for the participant. As the LPT was subsequently followed by a VO_2 max test it was important that the LPT did not excessively tire the participants as this could result in an underestimation of VO_2 max. Following a rest period, the VO2 max test was then conducted. The starting pace was 2kph slower than lactate threshold speed based on the results of the LPT immediately prior. Stages lasted 1 minute, with the first 2 stages being increases in speed (1kph each stage), and the subsequent stages being increases in gradient only (1% each stage). The test ended when participants reached exhaustion and could no longer sustain exercise. VO2 max was calculated as the 1 minute average at peak oxygen consumption.

3.6: Heat Tolerance Testing

The HTT protocol involved participants running on a treadmill inside an environmental chamber (Peak Performance Chamber, TISS) for 30 minutes at a constant workload of 9kph at 2% incline. Conditions were set at an ambient room temperature of 40°C and 40% relative humidity (RH). The exercise workload and environmental conditions were selected based on the work of Mee et al. (2015). The study found that conducting a running based HTT under these conditions resulted in very good reliability, shown through low within person variation of key HTT markers such as rectal temperature, skin temperature, heart rate and sweat loss; all of which were assessed in the current study. Furthermore the study population used by Mee et al. (2015) included male and female participants who were of a relatively similar age, body composition and fitness to participants of the current study, reinforcing the relevance of

the application of this HTT protocol for the current study. Cramer & Jay (2014) propose a metabolic heat production model approach for HTT design. This approach enables comparison between groups of differing body mass and body surface area, however this is not relevant to the participants of the current study. In the current study there is low variation in the physical characteristics of participants within groups, and no significant differences in physical characteristics between sauna and control participants (table 2a and table 2b).

For the safety of participants the HTT would be terminated if rectal temperature (T-rec) met or exceeded 39.5°C, however no participant reached this temperature. Participants were able to terminate the HTT at any point should they wish, however all participants completed all HTT tests fully.

Core temperature was measured using a rectal thermistor (Mon-a-Therm, Covidien, Mansfield, MA). Participants were instructed to insert the thermistor at a minimum of 10cm past the rectal sphincter. Skin temperature was measured using skin thermistors (Squirrel Thermal Couples, Grant Instruments, Cambridge, UK) placed on 4 sites: the upper arm (bicep), chest, calf and back. The equation ((0.3x chest) + (0.3x arm) + (0.2x thigh) + (0.2x calf)) was later used to calculate mean skin temperature (Ramanathan, 1964). The rectal thermistor and skin thermistors were connected to a data logger (Squirrel 2020 series, Eltek, Ltd., UK). Temperature readings were logged at 30 second intervals.

Heart rate was monitored throughout the HTT using a Polar chest strap heart rate monitor (Polar Electro, Kempele, Finland). Recordings of HR were taken at 10, 20 and 30 minutes into the HTT, with the recorded value being the exact value recorded at each ten minute interval, not an average of each ten-minute period.

Sweat loss was measured by calculating the change in body mass before and after the HTT. Measurements were taken after towel drying and in the nude state as clothing has the potential to retain moisture and affect the body mass reading. Sweat sensitivity was also measured, however the current study did not use sweat capsules to monitor sweat rate throughout the HTT. Instead sweat sensitivity for the current study is calculated by taking the total sweat loss (ml) for the HTT and dividing it by rectal temperature change (°C) over the course of the HTT. The output is a value showing sweat loss per degree Celsius of rectal temperature change, thereby normalising the data to exclude the influence of core temperature change on sweat loss. Data is presented as a bar graph, formatted in the same manner as the raw sweat loss data. This approach and presentation is adapted from that used by Wingo et al. (2008).

Participants were not permitted to drink during the HTT in order to avoid any cooling or heating effect of water consumption. Participants were permitted to consume liquids after the final measurement of the HTT had finished (post-exercise VOP). Any liquids consumed between the arrival weigh in and post-HTT weigh in was measured and factored into the calculation of body mass change. Body mass was measured using SECA 876 scales (SECA, Hamburg, Germany), accurate to 0.1kg.

Perceptual measures were recorded throughout the HTT, at 10, 20 and 30 minutes. Participants were asked to provide their perceived exertion using the Borg scale (RPE; Borg, 1982). Thermal comfort and thermal sensation were reported using scales modified from Toner et al. (1986). Thermal sensation used a scale of 1 to 13 where ‘1’ represents unbearably cold, ‘7’ represents ‘neutral’ and 13 represents ‘unbearably hot’. Thermal comfort used a scale of 1 to 10 where ‘1’ represented comfortable, ‘5’ represented uncomfortable and ‘10’ represented ‘extremely uncomfortable’.

Venous Occlusion Plethysmography was applied under 2 conditions. Firstly at rest under temperate environmental conditions (21°C-23°C) to establish resting forearm blood flow prior to the HTT. The second measurement was taken 6 minutes and 30 seconds after the end of the HTT, whilst remaining in the chamber under the same environmental conditions as the HTT.

For the resting (pre-HTT) measure the participant was required to sit still in a chair for 10 minutes to allow the normalisation of blood flow (Wythe et al., 2015). During this period the VOP equipment was set up. A strain gauge (Hokanson, Bellevue, WA) was applied to the upper forearm of the left arm. Strain gauge size selection was done by measuring the participants forearm at the widest point and subtracting 2cm. A pen was used on the forearm to mark the strain gauge position so an identical position could be established for the post-HTT measure. Strain gauge position was also measured and recorded from the elbow using a tape measure to ensure the position could be replicated on future lab visits. With the strain gauge in place an inflation cuff was then fitted to the wrist (TMC7, Hokanson) and upper arm of the participant (SC10D, Hokanson). During testing the wrist cuff was manually inflated to a pressure of 220mmHg. This supra systolic pressure occluded circulation to the hand ensuring no influence of arteriovenous shunts on the rate of forearm filling (Wythe et al., 2015). An automated inflator (Rapid cuff inflation system, Hokanson) inflated the upper arm cuff to a pressure of 40mmHg, preventing venous outflow from the arm without impairing arterial inflow (Wythe et al., 2015). The strain gauge was connected to a plethysmograph (EC6, Hokanson), which was connected to a laptop where data was recorded using a data logging programme (LabChart 7, ADinstruments, Dunedin, NZ). After 10 minutes of rest, the wrist cuff was inflated and after 60s of inflation the first recording was taken by inflating the upper arm cuff to 40mmHg. VOP recordings were 10s in duration followed by a 20s recovery period to ensure complete venous outflow and restoration of normal flow (see figure 2c).

Venous outflow was also facilitated by using a padded stand to elevate the left arm to the approximate level of the heart. Three recordings were taken, with values averaged for analysis. The post-HTT procedure was identical to that of the pre HTT, with the exception of recordings being conducted within the environmental Chamber and starting after 6 minutes and 30 seconds of seated rest, as opposed to 10 minutes with the pre-HTT measure.

VOP response can vary greatly due to factors such as: occlusion time, exercise type, exercise intensity, temperature and sampling site (Wythe et al., 2015). It is therefore most appropriate for the researcher to establish their own sampling period in order to achieve the most relevant, accurate and reliable results. In the case of this study recording periods for VOP were 10 seconds in duration. VOP slope analysis should be conducted when the slope demonstrates linearity, as this is when FBF is unimpeded (Wythe et al., 2015). The 4 to 8 second period promoted by Wythe et al., (2015) applied well to the data obtained from the resting pre-HTT measure, as shown by the highlighted section in figure 2a. In the post-HTT condition peripheral blood flow was substantially elevated. Consequently the arms capacitance was reached sooner than in the pre-HTT VOP measure, with the plateau observed well within the 10 second recording period. It was therefore deemed appropriate to select a sample period of 0.5 to 2.5s in the post-HTT condition (see figure 2b). Though this period is short, the reliability is substantially improved through averaging of the slopes of 3 recordings (see figure 2c).

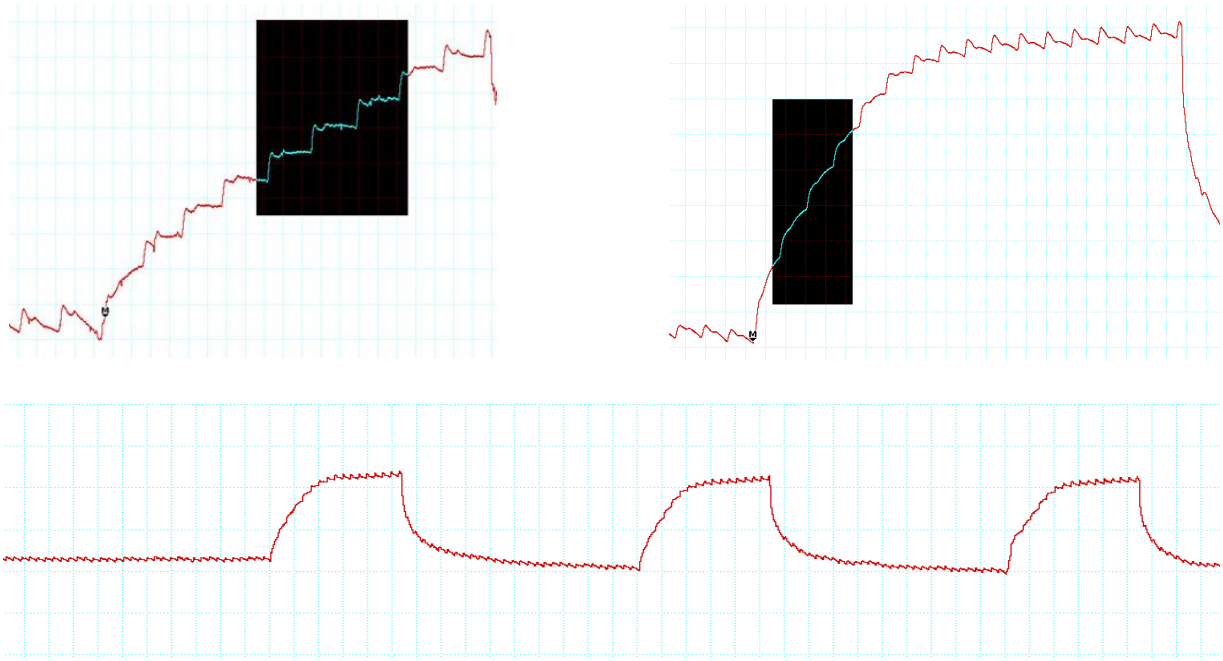


Figure 2a (top left): Resting (pre-HTT) VOP trace. Highlighted section of trace shows 4 to 8 seconds after upper arm cuff inflation, note linearity of the slope. **Figure 2b** (top right): post-HTT VOP trace. Highlighted section shows 0.5 to 2.5s after upper arm cuff inflation, note the clear plateau of trace after the highlighted section. **Figure 2c** (bottom): Typical Trace showing repeated VOP measures using the 10s inflation/20s rest method. The 3 inflation readings were subsequently averaged for analysis. Note: Scales for figures 2a, 2b and 2c differ.

Forearm blood flow sensitivity was measured using a similar method to that of sweat sensitivity. The change in FBF produced by the HTT was determined by calculating the difference between resting FBF and post-exercise FBF. This change in FBF was then divided by the change in core temperature over the course of the HTT, to produce a sensitivity value for FBF expressed as ml//100ml/min/°C.

3.7 – Participant training logs

All participants were asked to record all training activities throughout the course of the study to account for any potential training effects that may influence the results of the study. As all participants trained as a part of the same running club it was anticipated that there would be low variation between individuals and conditions. Regular training sessions were set out primarily by the participants' coach and were categorised as the following:

- Long run – exceeding 10km
- Easy run – any run at an RPE of 3 or less
- Tempo run – moderate to high intensity run around lactate threshold pace
- Interval – repeated effort exercises, including track-based sessions
- Races – competitive running events
- Cross-training – Non-running exercises as a part of training such as cycling, swimming or strength and conditioning work.

Table 3a shows training data for the 3W cohort, with table 3b showing training data for the 7W cohort.

Table 3a Training log and sauna adherence data for 3W cohort participants for the entire study period. Data is expressed as the group mean ± SD.

Total running distance (miles)	121 ± 44	136 ± 74
Long run sessions	2 ± 1	3 ± 1
Easy run sessions	7 ± 3	6 ± 3
Tempo run sessions	3 ± 2	2 ± 1
Interval/HIIT/Track sessions	4 ± 1	5 ± 1
Races	2 ± 1	2 ± 1
Cross-training sessions	2 ± 3	4 ± 7
Total sauna exposure time (minutes)	226 ± 66 (target = 300)	N/A
Sauna duration per session (minutes)	29 ± 1 (target = 30)	N/A

Sauna condition data N=9, one participant (10J) was excluded due to insufficient recordings of training. Control condition data N=6. Unpaired T-tests revealed no significant differences in any of the training variables between groups. Note: target exposure time of 300 minutes is based on full attendance of 10 sessions over the 3W period.

Table 3b Training log and sauna adherence data for 7W cohort participants for the entire study period. Data is expressed as the group mean ± SD.

Variable	**Experimental (Sauna)**	**Control**
Total running distance (miles)	117 ± 43	202 ± 51
Long run sessions	2 ± 1	4 ± 1
Easy run sessions	7 ± 3	8 ± 4
Tempo run sessions	3 ± 2	3 ± 0
Interval/HIIT/Track sessions	3 ± 1	6 ± 1
Races	2 ± 1	3 ± 1
Cross training	3* ± 3	0* ± 0
Total sauna exposure time (minutes)	386 ± 132 (target = 750)	N/A
Sauna duration per session (minutes)	29 ± 1 (target = 30)	N/A

Sauna condition data N= 5. Control condition data N=2. Unpaired T-tests revealed no significant differences in training variables except for significantly more cross-training sessions for the sauna group ($P < 0.01$). Note: target exposure time of 750 minutes is based on full attendance of all 25 sessions.

3.8: Data analysis

All statistical testing was conducted using SPSS (IBM, version 24.0, IBM, New York, NY). Analysis of single time point measures (Sweat loss and VOP) between pre-intervention and 3 weeks were conducted using a dependent sample T-test for each group (Sauna and Control). A one-way repeated measures ANOVA was used for Sweat loss and VOP data to compare Pre-intervention, 3 week and 7 week data for each group. For T-rec, T-sk, HR, RPE, Thermal comfort, and Thermal sensation a two way repeated measures ANOVA was applied to compare time points within each HTT (10, 20, 30 minutes) and compare between test points (Pre-intervention to 3 weeks) and (pre-intervention, 3 weeks, 7 weeks). The two-way repeated measures were conducted for each group. Bonferroni corrections were applied to the post-hoc analysis of all ANOVA tests, with post-hoc data reported only when a significant main effect for test point or test point by time interaction was found, with data presented as mean value ± SE. Significance level was set for all variables at $p<0.05$. This data analysis approach has been previously used by of Lorenzo et al., (2010) who applied a similar study design.

4. Results

Results are presented firstly for the 3-week cohort. This is followed by data for the 7-week cohort. The term 'test point' refers to the points at which testing was conducted (Pre-intervention, 3 weeks and 7 weeks). The term 'time point' refers to times within the HTT (10, 20 and 30 minutes). All figures except for perceptual data show the individual participant data. Below is a key for the individual data.

Sauna group	
Colour	**Participant ID**
	= 3O
	= 4O
	= 6O
	= 11O
	= 7J
	= 9J
	= 10J
	=11J
	= 13J
	= 14J

Control Group	
Colour	**Participant ID**
	= 2O
	= 8O
	= 9O
	= 21J
	= 22J
	= 24J

Key

= Mean reading for a group

'*' = significant difference ($p \leq .05$) versus pre-intervention

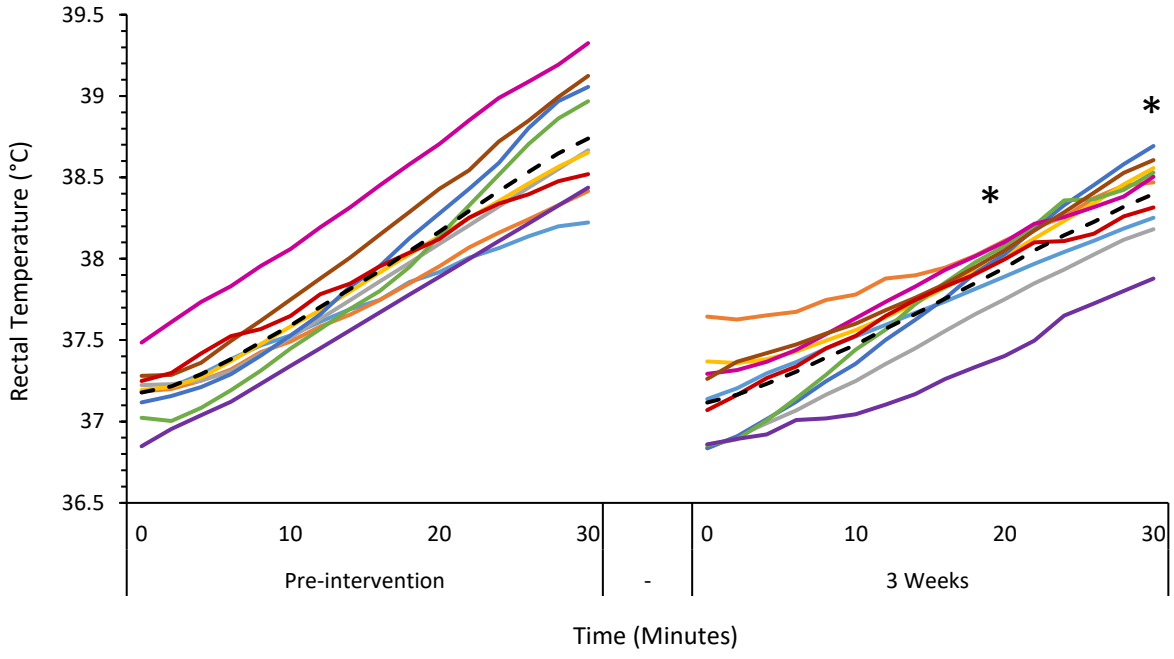
'†' = significant difference ($p \leq .05$) at W7 vs W3

* = Significant main effect

4.1: 3-Week cohort results

4.1.1: Rectal temperature

The sauna group showed a significant main effect for test point ($F(1, 9) = 7.49$, $p = .023$, ηp2 = .45), time ($F(1.1, 10.04) = 229.38$, $p < .001$, ηp2 = .96) and test point by time interaction ($F(1.59, 14.33) = 9.71$, $p = .003$, ηp2 = .52). Post-hoc analysis revealed rectal temperature was significantly lower at 3W vs pre-intervention at 20 minutes (-0.22 ± 0.07°C, $p = .014$) and at 30 minutes (-0.34 ± 0.09°C ($p = .004$); Figure 3a). For the control condition (figure 3b) there was no significant main effect for test point ($F(1, 3) = .55$, $p = .51$, ηp2 = .15). There was a significant main effect for time ($F(1.02, 3.06) = 28.86$, $p = .012$, ηp2 = .91). There was no significant test point by time interaction ($F(1.23, 3.69) = 1.608$, $p = .29$, ηp2 = .35).



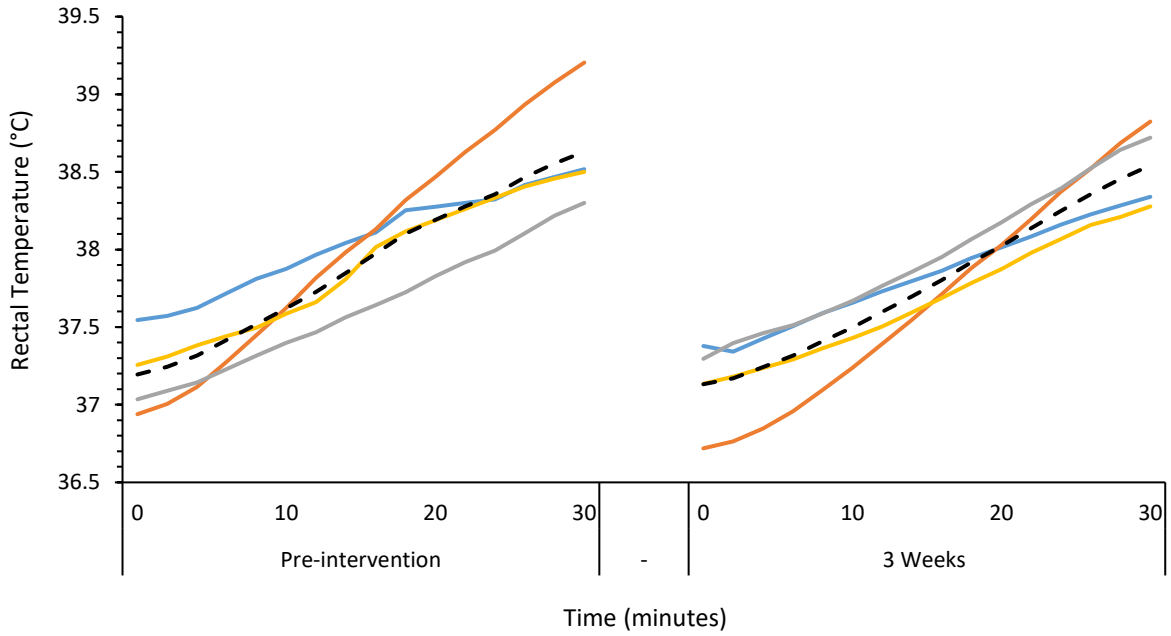


Figure 3a (top) and **Figure 3b** (bottom): Rectal temperatures of Sauna and control group participants respectively during the HTT at Pre-intervention and after 3 weeks of intervention.

4.1.2: Mean skin temperature

The Sauna group showed a significant main effect for test point ($F(1, 8) = 8.48$, $p = .020$, ηp2 = .52; figure 4a), time ($F(1.26, 10.05) = 16.92$, $p = .001$, ηp2 = .68), but no test point by time interaction ($F(1.37, 11) = .71$, $p = .46$, ηp2 = .08). Post-hoc analysis of test point showed a significant skin temperature reduction from Pre to 3W (-0.55 ± 0.19°C, $p = .020$). For the control condition there was no significant main effect for test point F(1, 4) = .025, $p = .88$, ηp2 = .006, figure 4b). There was a significant main effect for time ($F(3, 12) = 22.2$, p =.<.001, ηp2 = .85). There was no significant test point by time interaction F(3, 12) = 2.33, p = .13, ηp2 = .37.

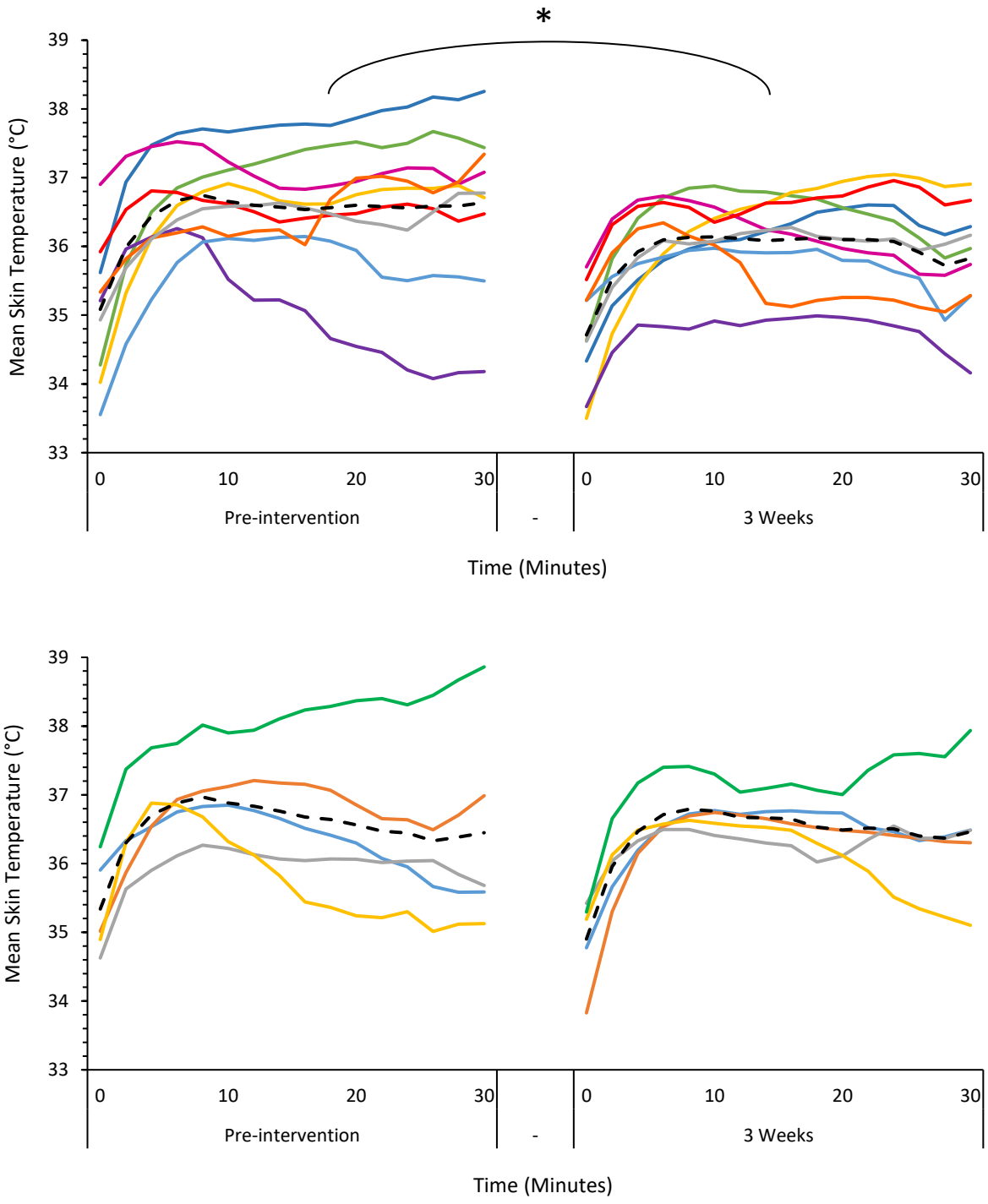


Figure 4a (top) and **Figure 4b** (bottom) Mean skin temperatures of Sauna and control group participants respectively during the HTT at pre-intervention and 3-weeks into intervention.

4.1.3: FBF

Rest: For the Sauna condition no significant change in FBF was observed between pre-intervention and 3W ($t(8) = 1.05$, $p = .32$, figure 5a). For the control condition no significant change in FBF was observed between pre-intervention and 3W ($t(4) = 1.045$, $p = .36$, figure 5b).

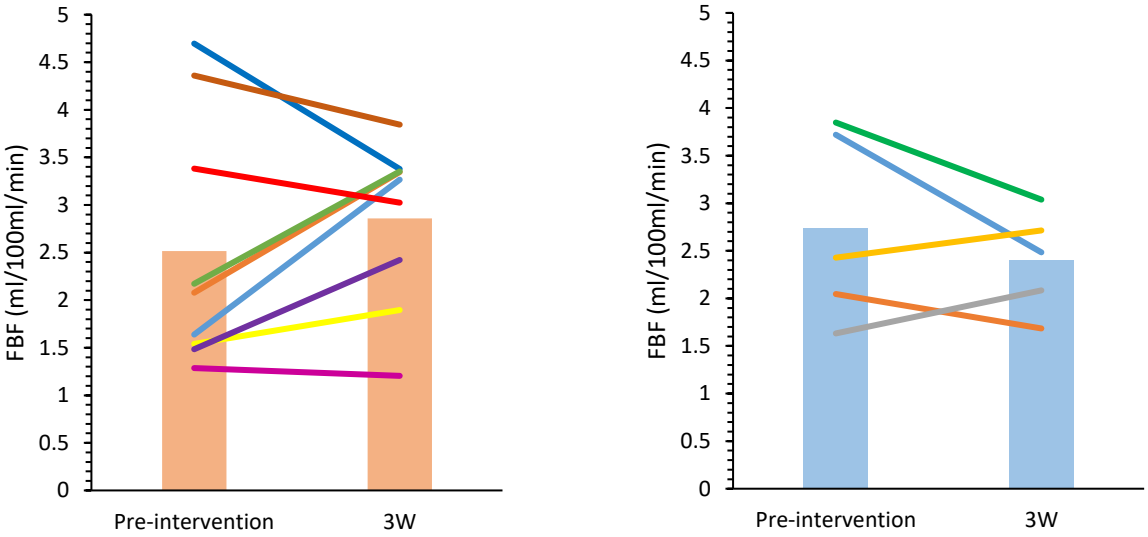


Figure 5a (above left) and **figure 5b** (above right): Forearm blood flow at rest prior to the HTT for sauna and control groups respectively. Data is shown for pre-intervention and 3-weeks into intervention.

Post-Exercise: For the Sauna condition no significant change in FBF was observed between pre-intervention and 3W ($t(9) = .71$, $p = .49$; figure 5c). For the control condition no significant change in FBF was observed between pre-intervention and 3W ($t(4) = 1.69$, $p = .17$; figure 5d).

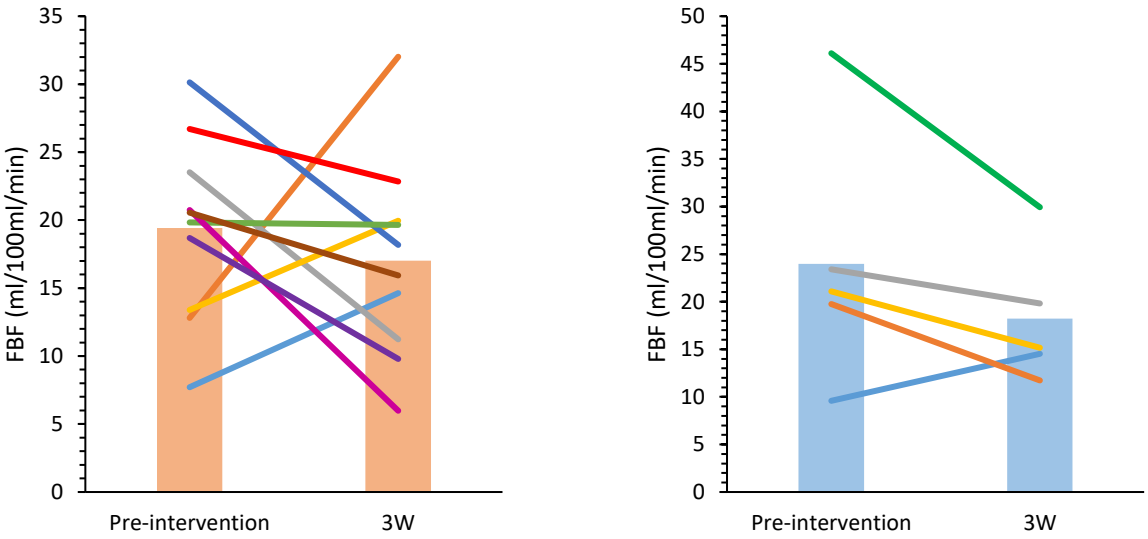


Figure 5c (above left) and **figure 5d** (above right): Forearm blood flow 6 minutes, 30 seconds post-HTT in 40°C and 40% RH conditions for sauna and control groups respectively. Data is shown for pre-intervention and 3-weeks into intervention. Note: Y-axis' vary in scale.

Post exercise FBF Sensitivity

Post- exercise: For the Sauna condition no significant change in FBF sensitivity was observed from pre-intervention to 3W ($t(9) = .57$, $p = .58$; figure 5e). For the control condition no significant change in FBF was observed between pre-intervention and 3W ($t(4) = 1.18$, $p = .31$); figure 5f).

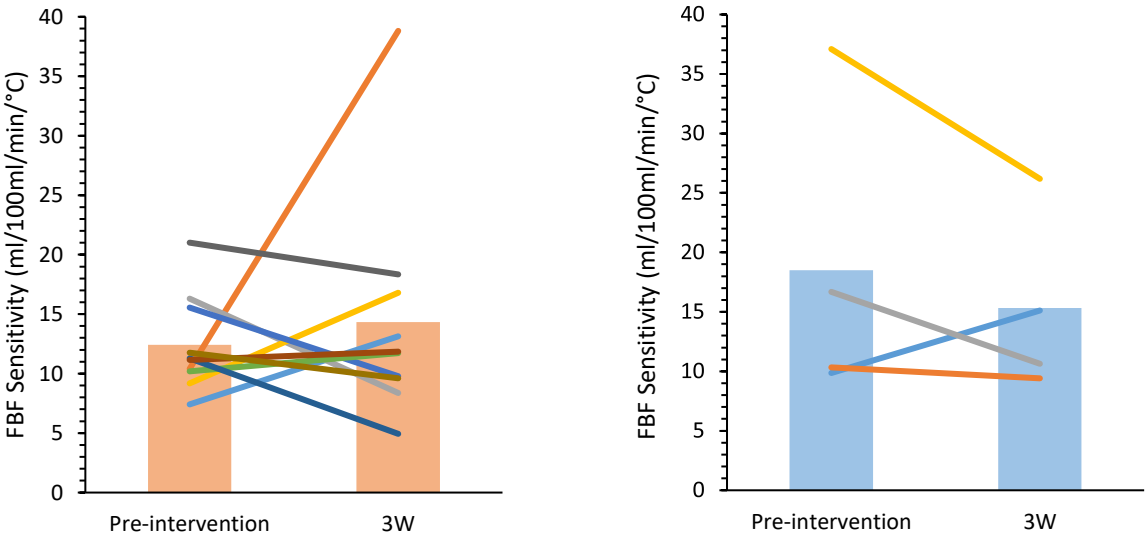
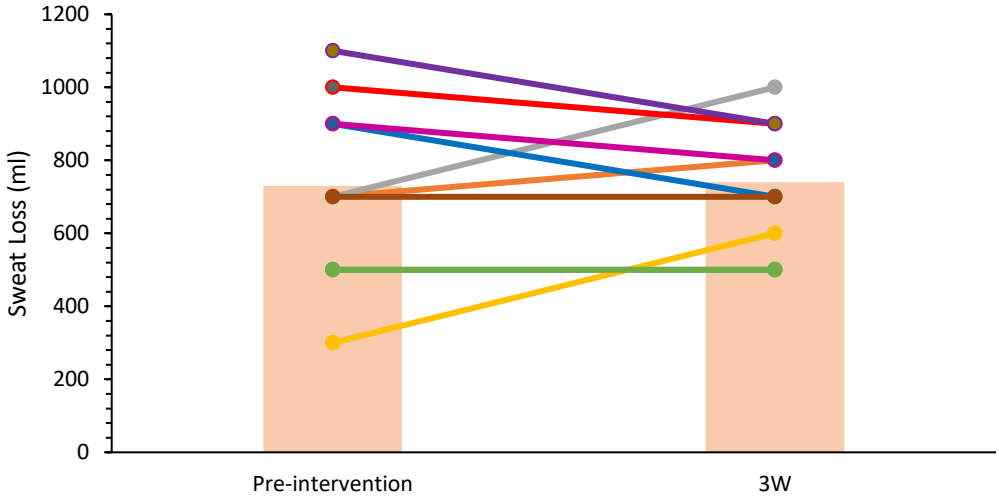


Figure 5e (above left) and **figure 5f** (above right): FBF Sensitivity calculated by FBF change for the HTT divided by temperature change for the HTT for sauna and control groups respectively. Data is shown for pre-intervention and 3-weeks into intervention.

4.1.4: Sweat Loss

For the Sauna condition no significant difference in sweat loss was observed between pre-intervention and after 3W of intervention ($t(9) = .176$, $p = .86$; figure 6a). For the control condition no significant difference in sweat loss was observed between pre-intervention and 3W of intervention ($t(5) = 1.185$, $p = .29$, figure 6b).



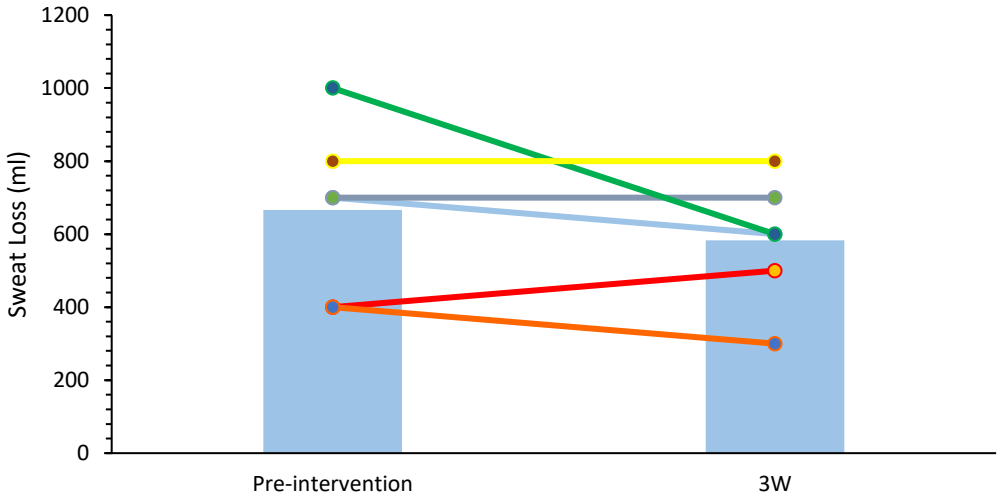


Figure 6a (above, top) and **Figure 6b** (above, bottom): Sweat loss data calculated through body mass change for sauna and control groups respectively for pre-intervention and after 3-weeks of intervention.

Sweat Sensitivity

For the Sauna condition a significant increase in sweat sensitivity was observed from pre-intervention to 3W ($t(9) = 2.54$, $p = .032$; figure 6c). For the control condition no significant change in sweat sensitivity was observed between pre-intervention and 3W ($t(3) = 1.25$, $p = .30$); figure 6d).

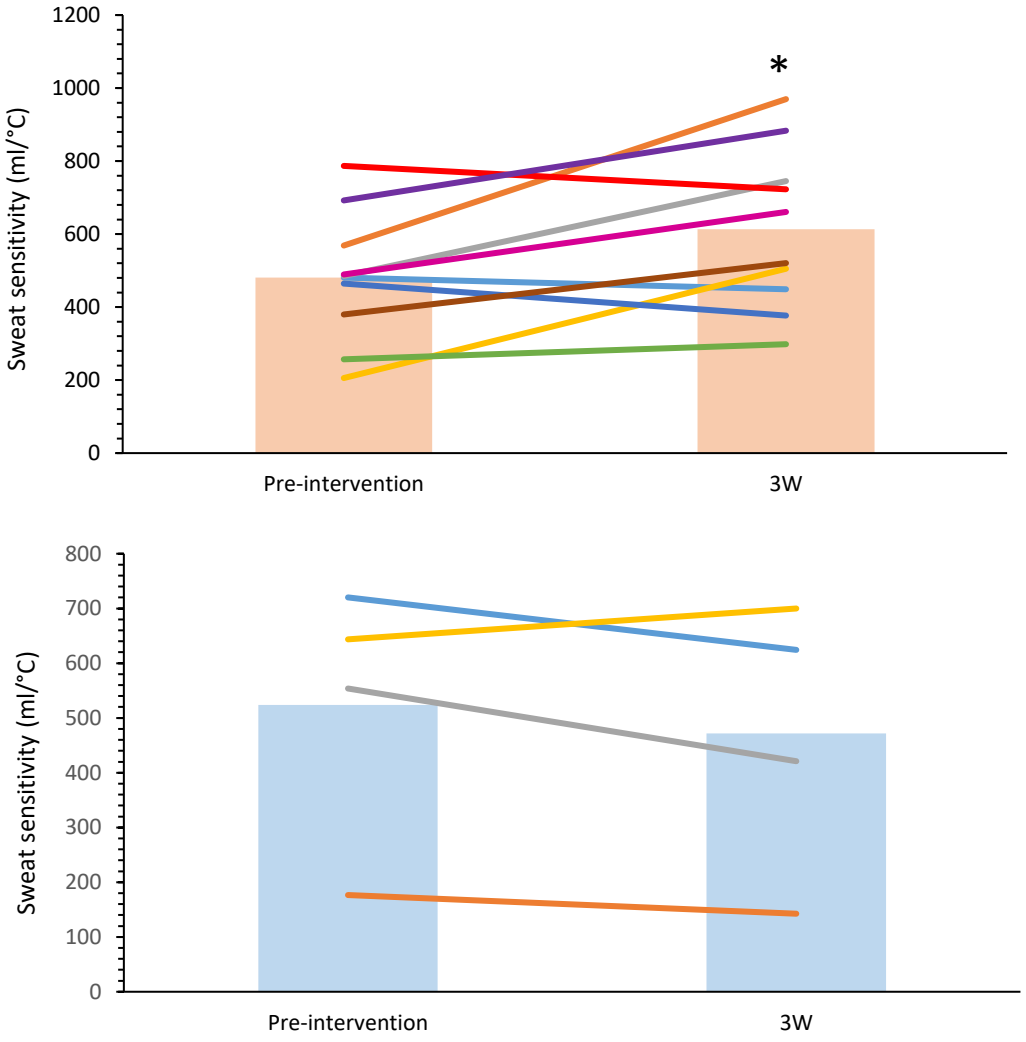


Figure 6c (above, top) and **figure 6d** (above, bottom): Sweat Sensitivity calculated by HTT sweat loss divided by HTT temperature change for sauna and control groups respectively. Data is shown for pre-intervention and 3-weeks into intervention.

4.1.5: Resting Heart Rate

For the Sauna condition a significant reduction in resting HR was observed at 3W compared to pre-intervention ($t(9) = 3.159$, $p = .012$; figure 7a). For the control condition no significant change in resting HR was observed between pre-intervention and 3W ($t(5) = 1.562$, $p = .18$, figure 7b).

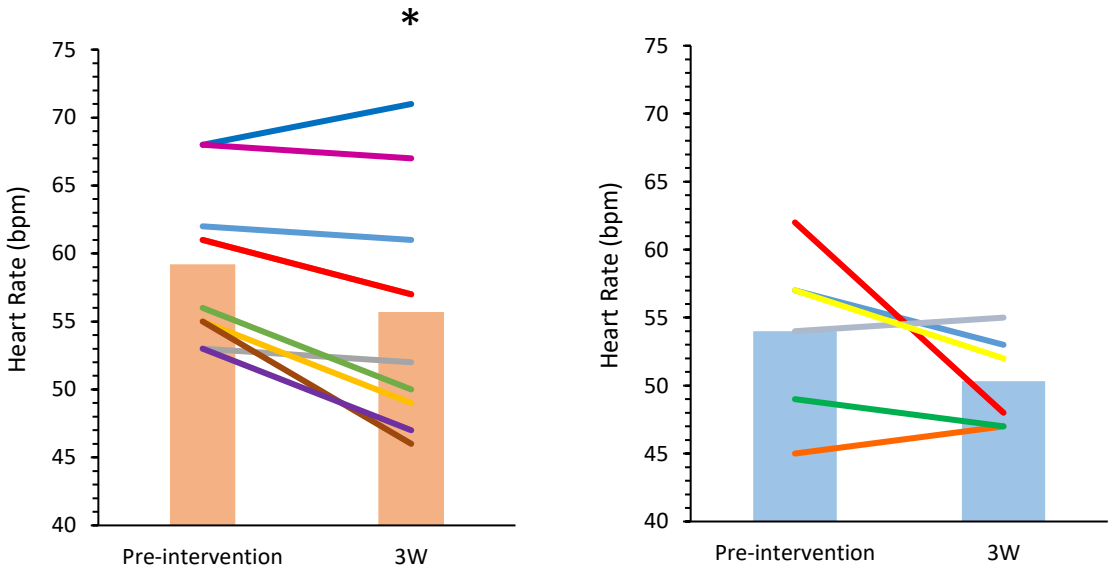


Figure 7a (above left) and **Figure 7b** (above right) Resting HR for sauna and control groups respectively at pre-intervention and after 3 weeks of intervention.

4.1.6: Exercising Heart rate

For the Sauna condition there was a significant main effect for test point ($F(1, 8) = 9.3$, $p = .016$, η p2 = .54, figure 8a) and time ($F(1.2, 16) = 4.54$, $p < .001$, ηp2 = .94), but no test point by time interaction (F(2, 16) = 2.296, $p = .13$, ηp2 = .22). Post-hoc analysis of test point data revealed a significant reduction in exercising HR from Pre to 3W (-10 ± 3bpm, $p = .016$). For the control condition there was no significant main effect for test point (F(1, 4) = .073, $p = .80$, ηp2 = .02, figure 8b). There was a significant main effect for time ($F(2, 8) = 32.1$, $p < .01$, ηp2 = .89). There was no significant test point by time interaction (F(2, 8) = 1.373, $p = .31$, ηp2 = .26).

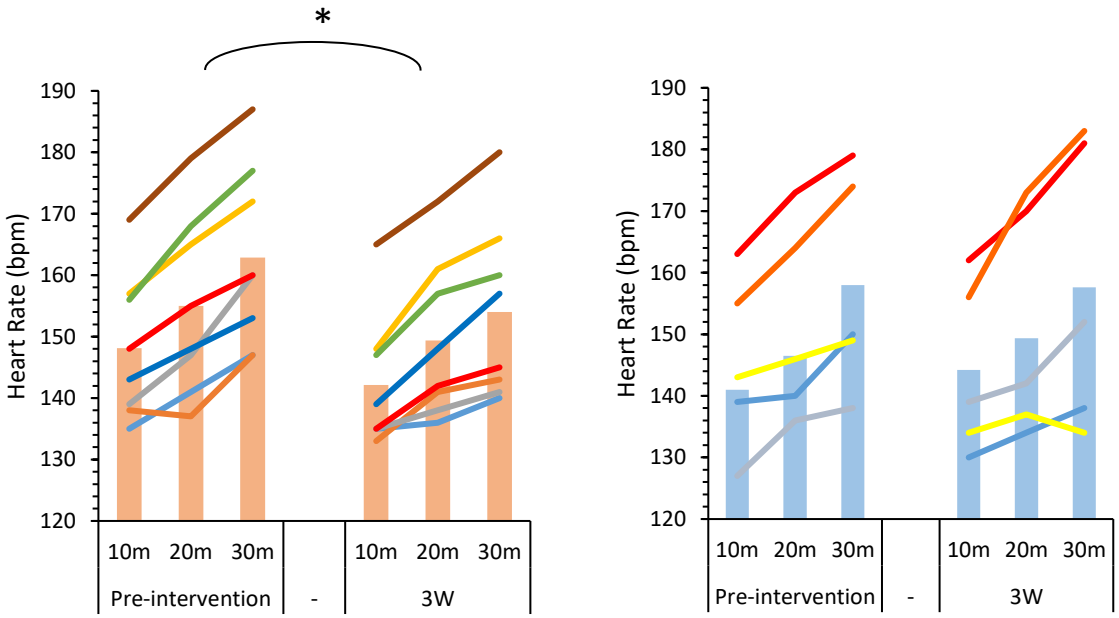


Figure 8a (above left) and **Figure 8b** (above right) Exercising HR for Sauna and Control groups respectively at pre-intervention and after 3 weeks of intervention. Note: Data for participant 10J was excluded due to suspected error in HR measurement.

4.1.7: Perceptual Scales

RPE

For the Sauna condition there was a significant main effect for test point ($F(1, 9) = 12.31$, $p = <.01$, $\eta p2 = .58$), time ($F(1.18, 10.64) = 23.1$, $p < .001$, $\eta p2 = .72$) and test point by time interaction ($F(2, 18) = 5.55$, $p = .013$, $\eta\ p2 = .38$) (table 4). Post-hoc analysis revealed a near significant reduction in RPE relative to pre-intervention at 20 minutes ($p = .051$), and by 30 minutes a significant reduction in RPE was observed ($p = <.01$, table 4). For the control condition there was no significant main effect for test point ($F(1, 5) = .493$, $p = .51$, $\eta p2 = .09$). A significant main effect for time was found ($F(2, 10) = 4.74$, $p = .036$, $\eta p2 = .49$). There was no significant test point by time interaction $F(2, 10) = .27$, $p = .77$, $\eta p2 = .05$).

Thermal Comfort

For the Sauna condition there was a significant main effect for test point ($F(1, 9) = 17.65$, $p = <.01$, $\eta p2 = .66$, and time ($F(2, 18) = 31.6$, $p < .01$, $\eta p2 = .78$), but no significant test point by time interaction ($F(1.41, 12.7) = 3.023$, $p = 0.098$, $\eta p2 = .25$) (table 4). Post-hoc analysis for test point revealed a significant reduction in RPE from Pre-intervention to 3W (-2 ± 0, $p = .002$). For the control condition no significant main effect was observed for test point ($F(1, 5) = 1.788$, $p = .24$, $\eta p2 = .26$), a significant main effect for time was found ($F(2, 10) = 9.23$, $p = .005$, $\eta p2 = .65$), and there was no significant test point by time interaction. ($F(2,10) = 2.009$, $p = .19$, $\eta p2 = .29$).

Thermal Sensation

For the Sauna condition there was a significant main effect for test point ($F(1, 9) = 12.94$, $p = <.01$, η p2 = .59), time ($F(1.24, 11.17) = 12.32$, $p < .003$, $\eta p2 = .58$) and also a significant test point by time interaction ($F(2, 18) = 3.86$, $p = .040$, η p2 = .30) (Table 4). Post-hoc analysis revealed that 3W thermal sensation was significantly reduced compared to pre-intervention at 10 minutes ($p = .045$), 20 minutes ($p = .019$) and 30 minutes ($p = <.01$). For the control condition there was no significant main effect for test point ($F(1, 5) = .056$, $p = .82$, $\eta p2 = .01$). There was a significant effect for time ($F(2, 10) = 4.36$, $p = .043$, $\eta p2 = .47$), and there was no significant test point by time interaction ($F(2, 10) = .745$, $p = .50$, $\eta p2 = .13$ (Table 4).

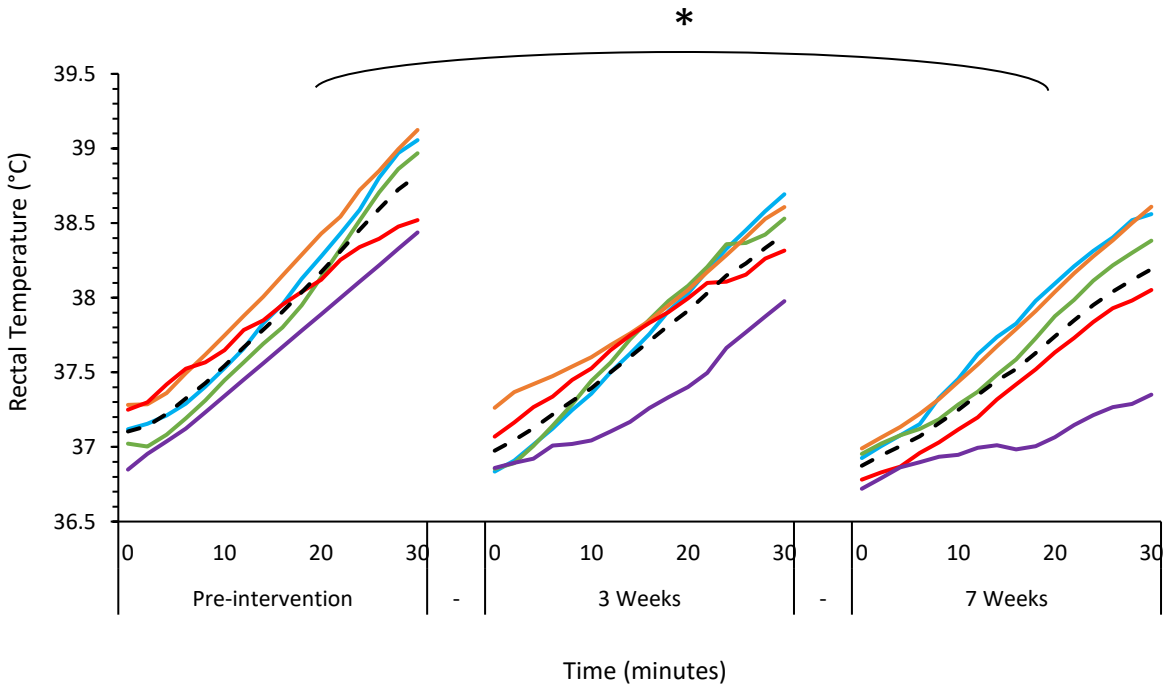
Table 4: Perceptual scale data for 10, 20 and 30 minute time points from the HTT at pre-intervention and after 3 weeks of HA. Data is shown as the group mean and Standard deviation.

		Pre-intervention			3W		
		10	**20**	**30**	**10**	**20**	**30**
RPE (6 to 20)	Sauna	9 ± 1	11 ± 1	12 ± 2	9 ± 2	10 ± 2	10* ± 2
	Control	10 ± 2	11 ± 2	11 ± 2	10 ± 2	10 ± 2	11. ± 2
Thermal Comfort (1 to 10)	Sauna	3 ± 1	5 ± 2	6 ± 2	2* ± 1	3* ± 1	4* ± 2
	Control	2 ± 1	3 ± 2	3 ± 2	2 ± 1	3 ± 2	4 ± 2
Thermal Sensation (1 to 13)	Sauna	9 ± 1	10 ± 1	10 ± 1	9* ± 1	9* ± 1	9* ± 1
	Control	9 ± 1	9 ± 1	9 ± 1	9 ± 1	9. ± 1	9 ± 1

4.2: Pre-intervention, 3 weeks, 7 weeks

4.2.1: Rectal temperature

For the Sauna condition a significant main effect was observed for test point ($F(2, 8) = 23.2$, $p = <.01$, $\eta p2 = .85$), time ($F(1.07, 4.28) = 116.45$, $p <.001$, $\eta p2 = .97$), but not test point by time interaction ($F(2.15, 8.62) = 3.414$, $p = .079$, $\eta p2 = .46$. Post-hoc analysis of test point data revealed 3W T-rec was significantly lower than at pre-intervention (-0.23 ± 0.03°C, $p = .004$), and 7W T-rec was significantly lower than at pre-intervention (-0.40 ± 0.07°C, $p = 0.014$). The reduction in T-rec from 3W to 7W was not significant (-0.16 ± 0.07°C, $p = .22$). For the control condition due to complete data being available from only 1 participant (21J) statistical testing could not be performed (figure 9b).



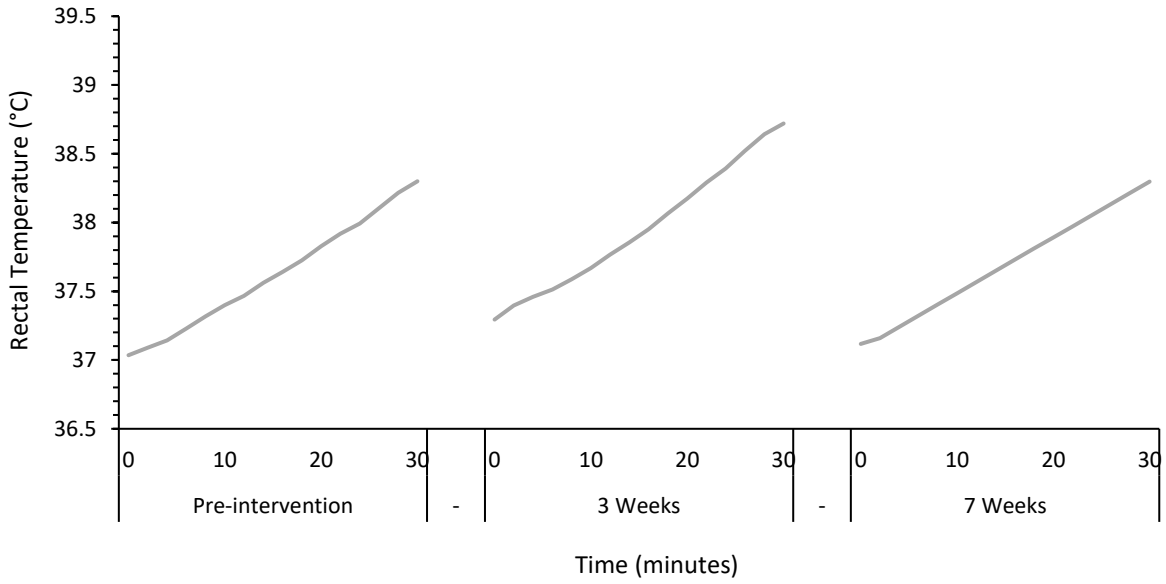


Figure 9a (top) and **Figure 9b** (bottom) Rectal temperatures of Sauna and control group participants during the HTT at pre-intervention, 3-weeks and 7-weeks into intervention.

4.2.2: Mean Skin Temperature

For the Sauna condition a significant main effect was observed for test point ($F(2, 6) = 6.66$, $p = .030$, $\eta p2 = .69$), time ($F(3, 9) = 8.25$, $p = <.01$, $\eta p2 = .73$), but not test point by time interaction ($F(1.72, 5.15) = .825$, $p = .47$, $\eta p2 = .22$ (figure 10a). Post-hoc analysis of test point data revealed T-sk was not significantly different between pre-intervention and 3W (-0.55 ± 0.34°C, $p = .62$), and not significantly different from 3W to 7W (-0.47 ± 0.26°C, $p = .50$). T-sk reduction was near significant from pre-intervention to 7W (-1.01 ± 0.23°C, $p = 0.062$). For the control condition there was no significant main effect for test point ($F(1, 1) = .095$, $p = .91$, $\eta p2 = .09$). There was a significant main effect for time ($F(1, 1) = 26.45$, $p = .012$, $\eta p2 = .96$. There was no significant test point by time interaction $F(1, 1) = .523$, $p = .60$, $\eta p2 = .34$ (figure 10b). Note: Statistical testing for control group was N = 2, therefore the value of these statistics is highly limited.

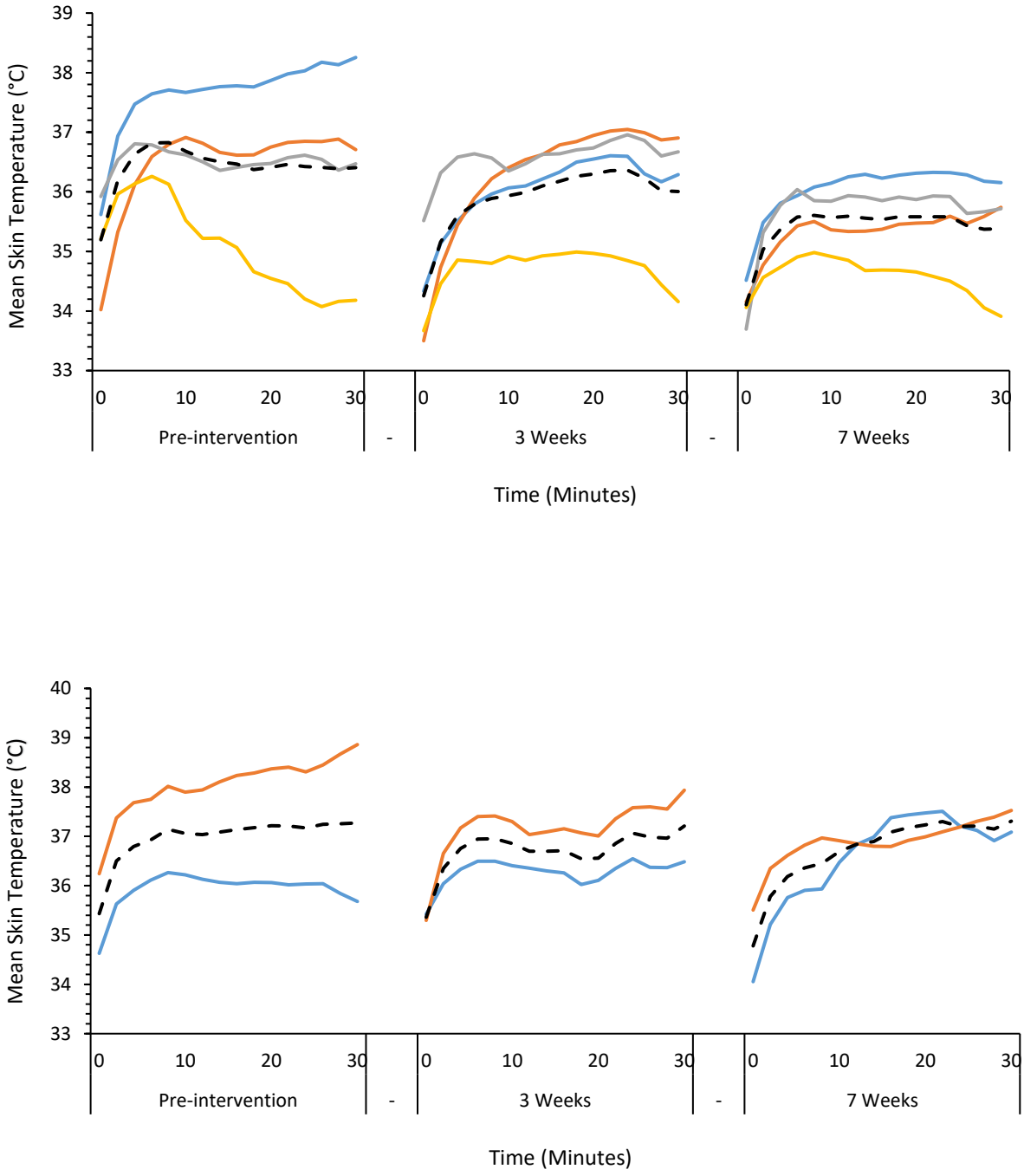
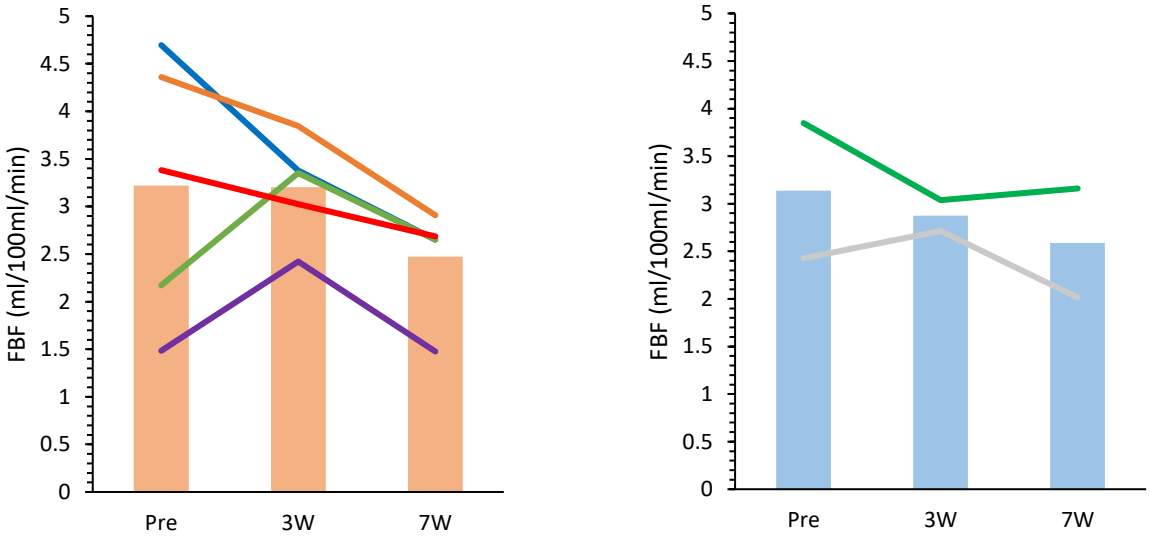


Figure 10a (top) and **Figure 10b** (bottom): Mean skin temperatures of Sauna and control groups respectively during the HTT at pre-intervention, 3-weeks and 7-weeks into intervention.

4.2.3: FBF

Rest: For the Sauna condition there was no significant main effect for test point F(2, 8) = 2.449, *p* = .15, ηp2 = .38 figure 11a). For the control condition there were no significant change in FBF for test point F(1, 1) = .929, *p* = .51, ηp2 = .48, figure 11b).



Figures 11a (above left) and **figure 11b** (above right): Forearm blood flow at rest prior to the HTT for sauna and control groups respectively. Data is shown for pre-intervention, 3 weeks and 7 weeks into intervention. Note: 'Pre' refers to 'Pre-intervention'.

Post Exercise: For the Sauna condition a significant reduction in FBF was observed over time F(2, 8) = 9.92, P = 0.007, ηp2 = 0.71). Post hoc analysis revealed no significant reduction in FBF from Pre to 3W (-3.30 ± 2.67 ml/100ml/min, *p* = .85), and no significant reduction from 3W to 7W (-8.82 ± 3.46 ml/100ml/min[1], *p* = .19). There was a significant reduction in FBF at W7 compared to pre-intervention (-12.11 ± 2.16 ml/100ml/min, *p* = .015; figure 11c). For the control condition there was no significant main effect for test point F(1, 1) = 1.626, *p* = .42, ηp2 = .62, figure 11d).

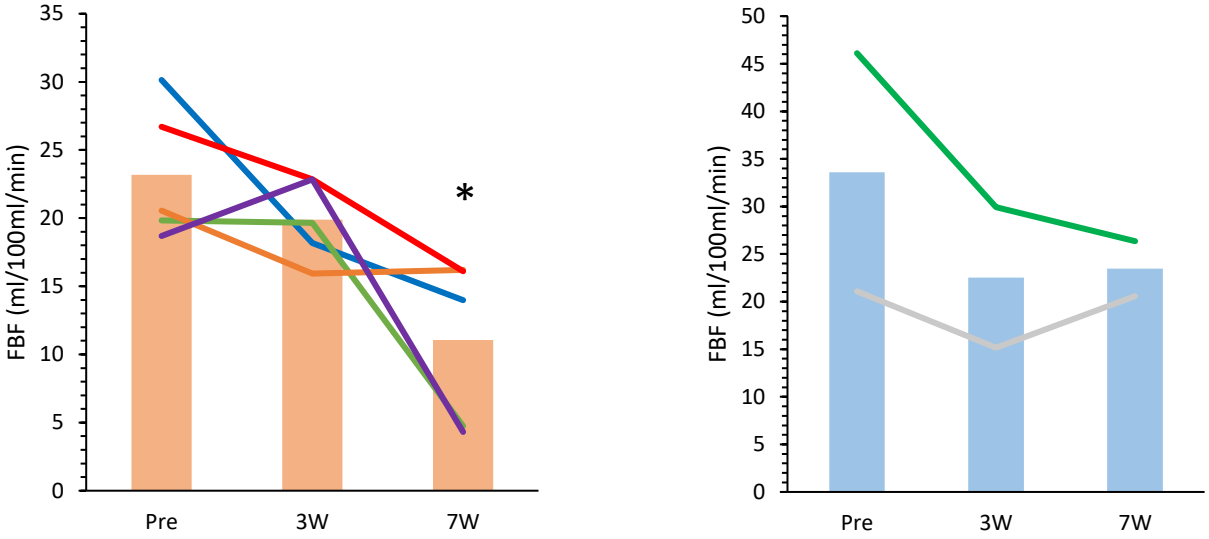


Figure 11c (above left) and **figure 11d** (above right): Forearm blood flow post-HTT for sauna and control groups respectively. Data is shown for pre-intervention, 3 weeks and 7 weeks into intervention. Note: Y-axis' vary in scale between figures 11c and 11d. Note: 'Pre' refers to 'Pre-intervention'.

Post-exercise FBF Sensitivity

For the sauna condition there was a significant change in FBF sensitivity over time $F(2, 10) = 8.35$, $p = 0.08$, $\eta p2 = 0.625$) (figure 11e). Post hoc analysis revealed no significant difference between pre-intervention and 3W (+0.49 ± 1.98 ml/100ml/min¹/°C, $p = 1$), or between 3W and 7W (-6.15 ± 1.86 ml/100ml/min¹/°C, $p = .063$). A significant difference was found between pre-intervention and 7W (-5.66 ± 1.02 ml/100ml/min¹/°C, $p = .008$). Statistical testing could not be performed for the control group as N = 1 (figure 11f).

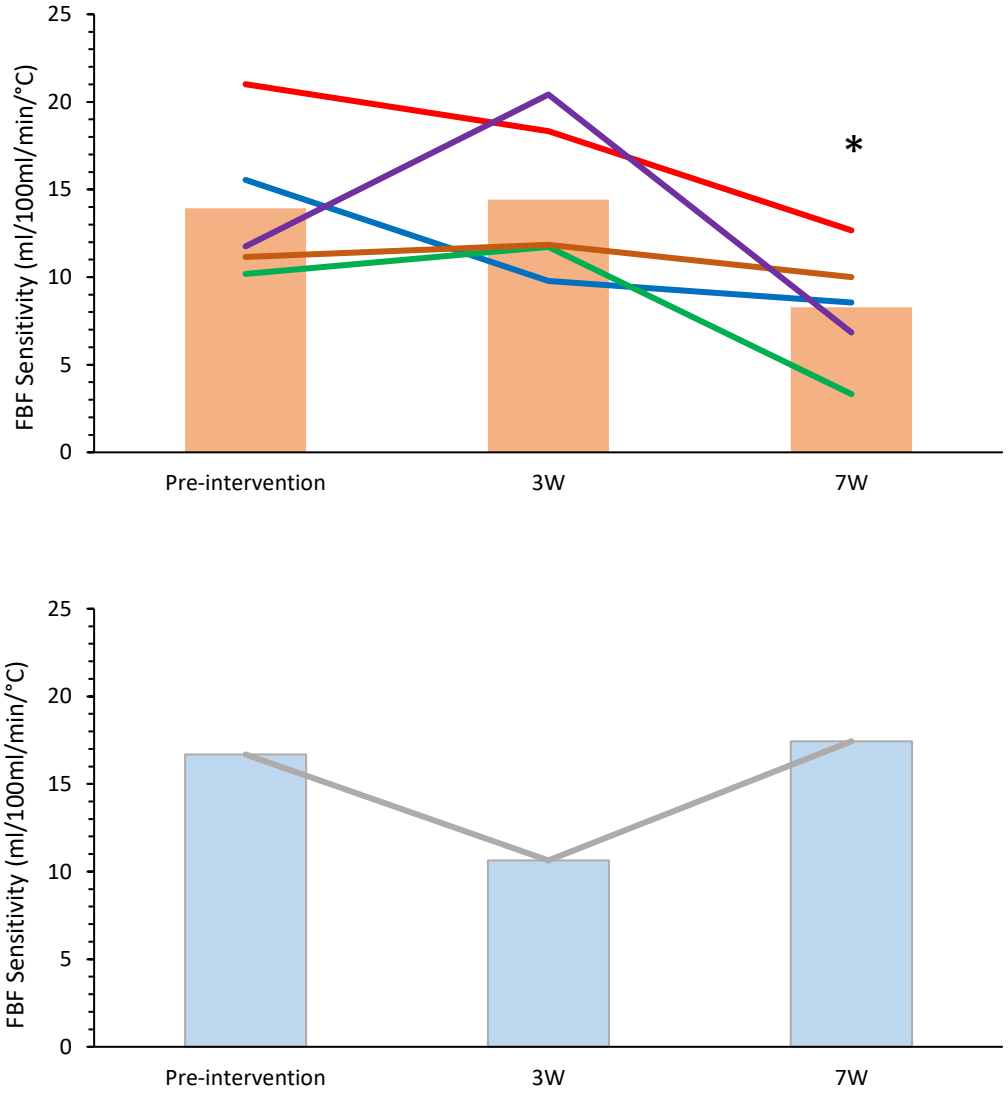


Figure 11e (above, top) and **Figure 11f** (above, bottom): FBF sensitivity was calculated by HTT FBF change divided by HTT temperature change. Data is shown for pre-intervention, 3-weeks into intervention and after 7 weeks of intervention.

4.2.4: Sweat Loss

For the Sauna condition there was no significant main effect for sweat loss (F(2, 8 = 1.849, *p* = .22, ηp2 = .32, figure 12a). For the control condition there was no significant main effect for sweat loss (F(1, 1) = 3, *p* = .33, ηp2 = .75 figure 12b).

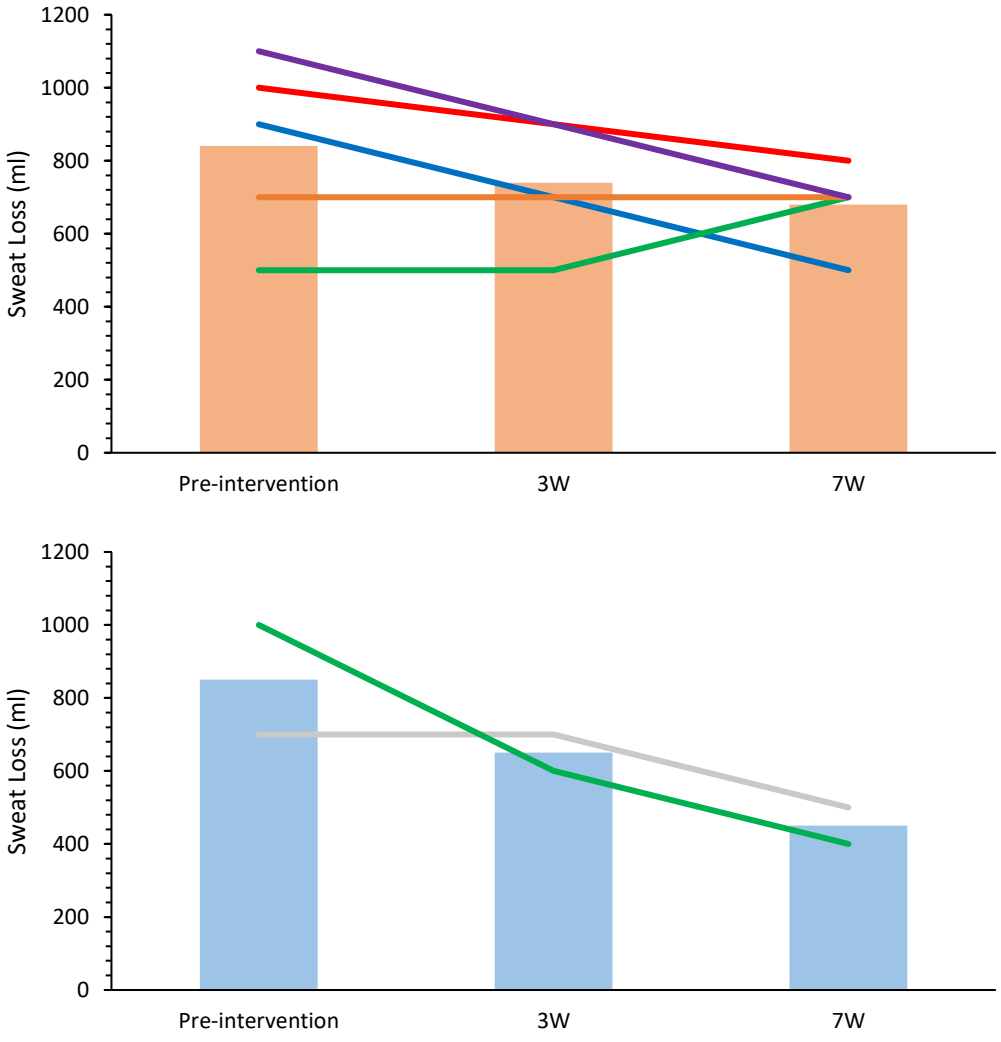


Figure 12a (top) and **figure 12b (bottom)**: HTT sweat loss data for sauna and control respectively, calculated through body mass change. Data is for pre-intervention, 3 weeks and 7-weeks of intervention.

Sweat Sensitivity

For the sauna condition there was no significant change in sweat sensitivity over time F(2, 8) = 0.43, p = 0.66, ηp2 = 0.096) (figure 12c). Statistical testing could not be performed for the control group as N = 1 (figure 12d).

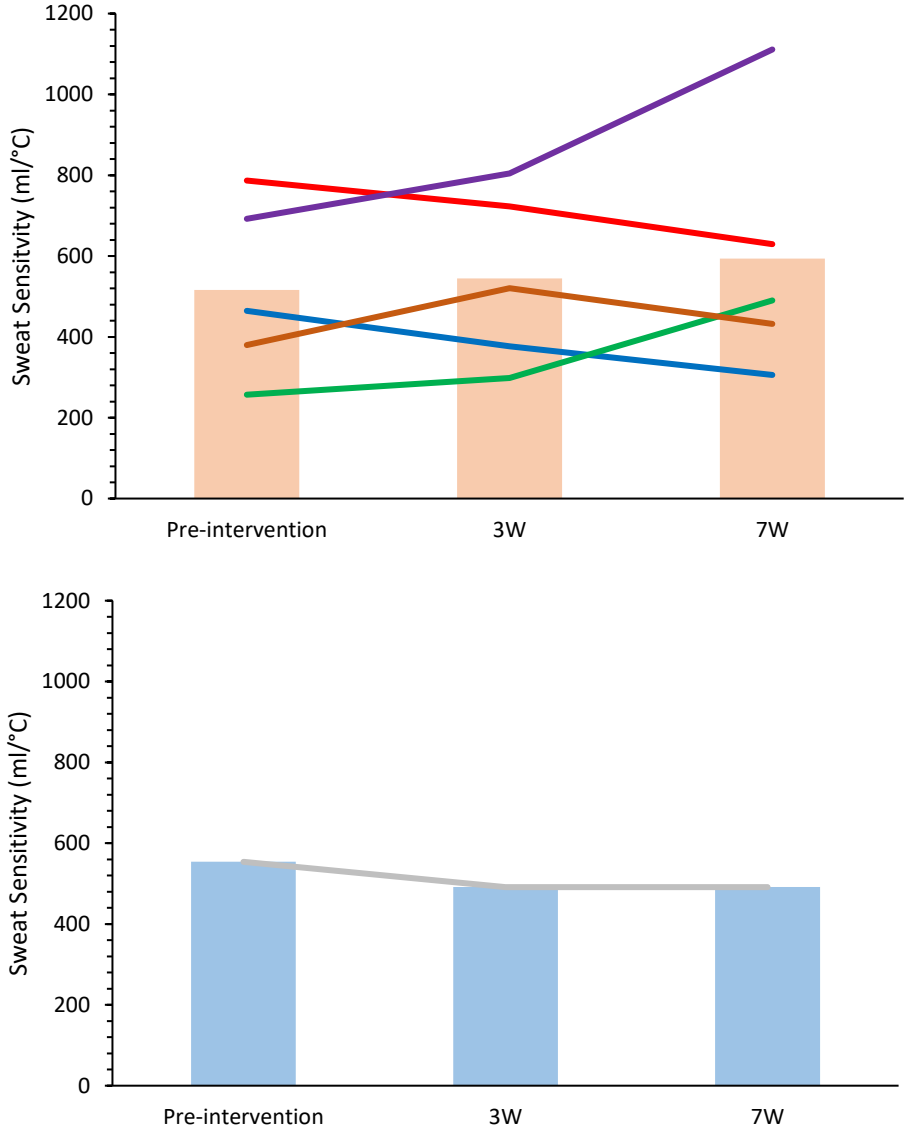


Figure 12c (above, top) **and figure 12d** (above, bottom): Sweat Sensitivity calculated by sweat loss divided by HTT temperature change for the sauna group. Data is shown for pre-intervention, 3-weeks into intervention and after 7 weeks of intervention.

4.2.5: Resting Heart Rate

For the Sauna condition there was no significant main effect for resting heart rate (F(2, 8) = 3.403, p = .085, ηp2 = .46 figure 13a). For the control condition there was no significant main effect for resting heart rate (F(1, 1) = .020, p = .91, ηp2, figure 13b).

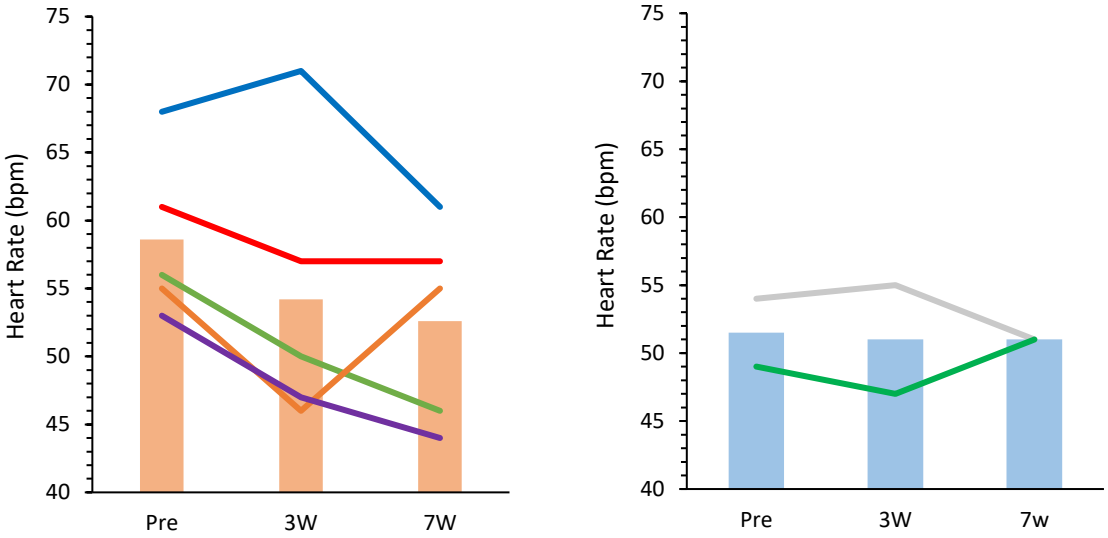


Figure 13a (above left) and **Figure 13b** (above right) Resting HR for sauna and control groups at pre-intervention, 3 weeks and 7 weeks respectively. Note: 'Pre' refers to 'Pre-intervention'.

4.2.6: Exercising Heart Rate

For the Sauna condition there was a significant main effect for test point (F(2, 6) = 8.58, p = .017, ηp2 = .74), time (F(2, 6) = 47, p <.001, ηp2 = .94), but no significant test point by time interaction (F(4, 12) = .299, p = .87, ηp2 = .09, figure 14a). Post-hoc analysis revealed no significant difference in exercising HR between any of the individual test points: Pre to 3W (-8 ± 3bpm, p = .25), 3W to 7W (-2 ± 1bpm, p = .21) and Pre to 7W (-10 ± 3bpm, p = .14). For the control condition complete data was available from only 1 participant so statistical testing

was not performed. For this participant pre-intervention HR for 10m, 20m and 30m was 126bpm, 136bpm and 138bpm respectively. 3W HR was 139bpm, 142bpm and 152bpm. 7W HR's were 136bpm, 144bpm and 150bpm.

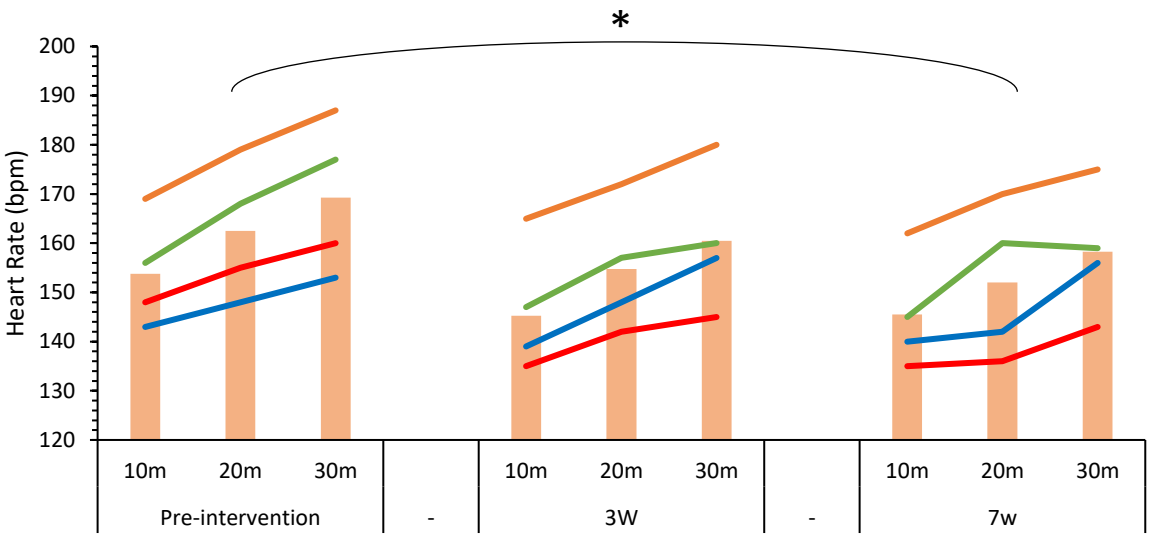


Figure 14a (above): Exercising HR for sauna group participants at pre-intervention, 3 weeks and 7 weeks.

4.2.7: Perceptual Scales

RPE

For the Sauna condition there was a significant main effect for test point ($F(2, 8) = 27.59$, $p = <.01$, $\eta p2 = .87$), time ($F(2, 8) = 18.15$, $p = .001$, $\eta p2 = .82$) and also a significant test point by time interaction ($F(4, 16) = 5.16$, $p = .007$, $\eta p2 = .56$). Post-hoc analysis revealed at 30 minutes a significant reduction in RPE was observed at 3W compared to pre-intervention ($p = .009$). 7W RPE was also significantly lower than pre-intervention at 30 minutes ($p = .004$), and furthermore 7W RPE was significantly lower than 3W at 30 minutes ($p = .012$) (table 5). For the control condition no significant main effect was observed for test point ($F(2, 2) = 2.579$, $p = .28$, $\eta p2 = .72$. There was no significant effect for time ($F(1, 1) = 18.143$, $p = .15$, $\eta p2 = .948$; and there was no significant effect for test point by time interaction $F(1, 1) = 1$, $p = .50$, $\eta p2 = .5$, table 5).

Thermal Comfort

For the Sauna condition there was a significant main effect for test point ($F(2, 8) = 16.13$, $p = .002$, $\eta p2 = .80$), significant effect for time ($F(2, 8) = 9.3$, $p = .008$, $\eta p2 = .70$), but no significant test point by time interaction ($F(4, 16) = 1.624$, $p = .22$, $\eta p2 = .29$, table 5). For the control condition there was no significant main effect for test point ($F(2, 2) = .259$, $p = .79$, $\eta p2 = .21$); no significant effect for time ($F(8.2, 16.4) = 2.654$, $p = .11$, $\eta p2 = .38$); and no significant interaction ($p = .59$; table 5).

Thermal Sensation

For the Sauna condition there was a significant main effect for test point ($F(2, 8) = 10.35$, $p = .006$, η p2 = .72), time ($F(2, 8) = 4.54$, $p = .048$, ηp2 = .53), but no significant test point by time interaction (F(1.57, 6.28) = .681, $p = .51$, ηp2 = .15) (table 5). For the control condition there was no significant main effect for test point (F(1, 1) = 13, $p = .17$, ηp2 = .93), time (F(1, 1) = 19, $p = .14$, ηp2 = .95), or test point by time interaction (F(4, 4) = .778, $p = .59$, ηp2 = .44, table 5).

Table 5: Perceptual scale data for 10 minute, 20 minute and 30 minute time points from the HTT. Data is presented for pre-intervention, 3 weeks and 7 weeks.

		Pre-intervention			3W			7W		
		10	**20**	**30**	**10**	**20**	**30**	**10**	**20**	**30**
RPE (6 to 20)	Sauna	9 ± 2	11 ± 2	13 ± 2	10 ± 1	10 ± 2	10* ± 2	8 ± 1	9 ± 2	9*† ± 2
	Control	12 ± 2	13 ± 1	14 ± 2	11 ± 1	11 ± 3	13 ± 4	10 ± 3	12 ± 4	13 ± 3
Thermal comfort (1 to 10)	Sauna	3 ± 0	5 ± 1	6 ± 2	2 ± 1	3 ± 1	4 + 2	2 ± 1	3 ± 1	3 ± 1
	Control	3 ± 0	4 ± 0	5 ± 1	3 ± 1	4 ± 1	5 ± 1	3 ± 2	5 ± 2	5 ± 1
Thermal Sensation (1 to 13)	Sauna	9 ± 1	10 ± 1	10 ± 1	8 ± 1	9 ± 1	9* ± 1	8 ± 1	9 ± 1	9 ± 1
	Control	9 ± 1	10 ± 0	10 ± 0	9 ± 0	9 ± 0	9 ± 1	9 ± 1	10 ± 1	10 ± 0

5. Discussion

5.1: Summary of results

The aim of this study was to establish whether or not post-exercise sauna bathing is an effective method of heat acclimating endurance athletes, and to establish potential mechanisms that produce acclimation. The main findings of the current study are that 3 weeks of post-exercise sauna bathing caused participants to heat acclimate and improve their exercising heat tolerance, with some indicators of further acclimation after 7 weeks of intervention. Changes in whole body sweat loss did not occur, although sweat sensitivity increased suggesting improved sudorific function. By 7 weeks there was evidence of reductions in peripheral blood flow.

3W cohort

A re-setting of resting core temperature has been observed as an adaptation in other heat acclimation studies, however the current study found no such adaptation. No change in resting core temperature was observed with HA, however during exercise the increase in core temperature was attenuated following 3W of HA, although this difference was only significant at 20 and 30 minutes, and not the earliest stages of exercise. Sweating is an essential mechanism for heat elimination, but this study found no change in sweat loss during the HTT following 3W of HA. However sweat sensitivity data indicates that improvements in sweating function occurred enabling a lower core temperature at the end of the HTT for the same total sweat loss. These improvements could include increased sweat distribution across the skin, dilution of sweat and earlier sweating onset; all of which would act to increase the effectiveness of evaporative heat losses. As the HTT environment (40°C) did not permit dry

heat loss, the reductions in core and skin temperature are almost certainly due to these other sweat based adaptations. VOP data showed no significant change in FBF values after 3W of HA, suggesting peripheral circulatory adaptations had not occurred. Exercising HR was significantly reduced, suggesting reduced cardiovascular strain with exercise in the heat. Perceptual measure data showed improvements indicative of acclimation with reductions in RPE as well as improvements in thermal comfort and sensation scores.

7W cohort

Resting core temperature showed a modest reduction at 3W but a more substantial reduction by 7W. Exercising T-rec was reduced across the entirety of the HTT after 3W of HA, and was further reduced after 7W of HA. No changes in sweat loss occurred at either 3W or 7W relative to pre-intervention, suggesting that the lack of change in sweat loss is a result of this HA method, and not a result of insufficient time for the adaptation to manifest. VOP data showed no significant change in FBF at 3W, however by 7W a significant reduction in exercising FBF was found compared to pre-intervention. A reduced FBF with HA aligns with findings from Eichna et al (1950) and Wyndham (1951). These studies concluded that the lack of dry heat loss would be compensated for by an increase in sweat loss; which is not true for the current study; however sudorific function is believed to have improved in other ways. Exercising heart rate was notably reduced after 3W (though not statistically significantly), but no further reductions in HR were observed after W7. This suggests cardiac adaptations were virtually complete after week 3, a finding in accordance with existing knowledge and the study hypothesis. Perceptual measure data showed improvements in all measures after W3, with further improvements after W7; although of these further improvements from W3 to W7 only RPE was statistically significant. The improvements in perceptual measures were logical

findings given the reductions in core and skin temperatures as well as heart rate that occurred with HA.

5.2: Adherence

Evidence for heat acclimation occurring is clear despite participants not reaching the intended total accumulated sauna exposure time. Adherence data (tables 3a, 3b) revealed that when participants did attend the sauna they were typically able to complete or nearly complete the full 30 minute exposure, thus the lower than intended sauna exposure time is the result of missed sessions, not finishing sessions early.

Adherence for the 3W cohort was relatively good at 226 minutes of the intended 300 (75% adherence). Adherence for the 7W cohort is also good after 3W of HA, at 252 minutes of the intended 300 (84% adherence). For the 3W to 7W period however adherence becomes substantially lower resulting in a 7 week total exposure of 386 minutes of the intended 750 being completed (51% adherence). This low adherence in the latter stages of HA may in part contribute to the diminished reduction in T-rec found from W3 to W7. An adaptation plateau is another likely influence too. The reasons why participants reduced their adherence from 3W to 7W was not investigated. Heat acclimation can be physically uncomfortable and perhaps participants' motivation to attend deteriorated with time. Another potential explanation is that this method of HA relies on prior training/exercise, thus if training is cancelled or the athlete cannot train due to injury then post-exercise sauna bathing may not be possible. Furthermore, the method of HA in this study employs exercise and then heat exposure, a format that is somewhat time intensive; which could clash with the participants' other commitments, resulting in sub-optimal sauna attendance.

Adherence data indicates that participants are largely able to sustain commitment to a 3W intermittent HA protocol, but struggle to maintain their attendance for a 7W protocol. Even with poor attendance in the 3 to 7 week period of acclimation participants still exhibited some clear markers of improved acclimation status relative to their 3W values.

5.3: Adaptations to post-exercise sauna bathing

5.3.1: Core Temperature

A change in core temperature, as measured by T-rec in this study is the clearest indication of adaptation to heat, as a change in T-rec is the product of all physiological adaptations that modify the thermoregulatory response to heat stress and exercise (Sawka et al., 2011).

It is commonly reported that with HA a person's resting core temperature becomes lower (Tyler et al., 2016), a process that is achieved through modification of the thermoregulatory set-point (Gisolfi & Wenger, 1984). One benefit of this is a larger temperature buffer for the body before a state of hyperthermia is reached. Another benefit is that heat loss mechanisms can be initiated at a lower body temperature than in the pre-HA state, thus attenuating heat gain in the early stages of exercise (Sawka et al., 2011).

The magnitude of the change in resting core temperature has been reported as -0.18°C when accounting for a variety of HA methods (Tyler et al., 2016), and more specifically -0.26°C for studies employing passive HA (Heathcote et al., 2018). In the current study the 3W cohort showed a negligible reduction of -.0.063°C in pre-exercise T-rec. Though core temperature was significantly reduced overall from pre-intervention to 3W it appears a re-setting of resting core temperature did not occur. In the 7W cohort however, mean resting T-rec was reduced by 0.13°C by 3W and by 0.23°C by 7W. Though not statistically significant (p =.092) the 7W value in the current study indicates that a re-setting of core temperature did occur with HA,

furthermore the reductions were observed in all five participants. It does also appear possible that HA can occur without the re-setting of resting core temperature, as Pallubinsky et al. (2017) passively acclimated participants and found reduced core temperatures during exercise but not at rest.

Despite the 3W cohort showing little change in resting T-rec, once exercising a difference in T-rec became evident with HA. At the end of the HTT, T-rec was reduced by 0.34°C after 3 weeks of HA, a value highly similar to the 0.33°C reduction found by other passive HA studies reported in table 1. This finding validates that 3 weeks of post exercise sauna bathing is as effective as other passive HA methods. It was also found that participants in the 7W cohort significantly reduced their end of HTT T-rec at 3 weeks (-0.40°C), with a further (non-significant) reduction to -0.63°C by 7 weeks.

As core temperature is the best single marker of HA status (Sawka et al., 2011) this data shows that whilst 3 weeks of post-exercise sauna bathing is effective, HA is not complete and athletes may benefit from a more prolonged HA protocol of 7 weeks. The additional (non-significant) reduction in T-rec seen from 3 to 7 weeks is notable, however the rate of improvement is diminished compared to the initial HA period up to 3W. This diminished improvement is unsurprising as most adaptation occurs within the early stages of HA, with 75% to 80% of adaptation occurring within 4 to 7 exposures (Pandolf, 1998; Shapiro et al., 1998); thus room for further acclimation is limited after this point. The limited improvements with additional time makes the value of long term HA questionable and should be an important consideration when trying to integrate post-exercise sauna bathing into an athletes training regime.

5.3.2: Forearm Blood Flow

Resting FBF showed no significant change in the 3W cohort, and in the 7W cohort resting FBF showed no change relative to pre-intervention at 3W or 7W. The lack of change in resting FBF with HA suggests any modification of FBF with HA occurs only as an acute response to exercise and heat stress, and is not a chronic adaptation that modifies FBF in the resting state. This conclusion is supported by evidence from Lorenzo & Minson (2010), who determined that HA did not modify the structure of the cutaneous vasculature, instead changes in SKBF that occurred with HA were driven by an increased sensitivity of response to the vasodilator acetylcholine (ACh). ACh is mediated by the response of prostanoids, nitric oxide, and possibly an endothelium derived hyperpolarizing factor (Lorenzo & Minson, 2010). In the current study resting FBF was assessed in a non-thermally challenging environment (room temperature 21-23°C). Consequently whilst HA may have increased participants sensitivity to factors that stimulate ACh, under these resting conditions where there was no exercise or elevation in internal temperature there was no stimulus for such factors to stimulate the action of ACh and therefore no modification of peripheral blood flow. If HA modified PBF through a central mechanism that altered a 'set-point' for skin blood flow then a difference in resting FBF following HA may be expected, however there is no evidence in the literature for such an adaptation. It has been proposed that there is some central adaptation relating to skin blood flow with HA, expressed as a reduced core temperature threshold for the onset of peripheral vasodilation without a change in flow sensitivity (Fox et al., 1963; Roberts et al., 1977; Yamazaki & Hamasaki, 2003). This mechanism would not however influence resting FBF under temperate conditions. Furthermore the findings of Lorenzo & Minson (2010) have shown this central mechanism to

be inaccurate due to their finding of HA increasing flow response sensitivity and not just creating a threshold shift (Periard et al., 2015).

In the 3W cohort no change in post-exercise FBF was found following HA. Likewise in the 7W cohort no change was found after 3 weeks of HA, however a significant reduction in post-HTT FBF was observed by 7 weeks (relative to pre-intervention). Individual data shows that FBF reductions occurred in all five participants from pre-intervention to W7. The individual data highlights that the significance of the reduction seen between pre-intervention and W7 is driven by particularly low 7W FBF values in participants 9J and 14J; with readings of just 4.74 and 4.31ml/100ml/min^{-1} respectively. These readings created concerns over their validity. The data files for these readings were re-analysed and showed good calibration and a consistent trace with individual pulses clearly visible. The three recordings from which the mean FBF value was calculated were also consistent. Furthermore other participants were tested in the hour before and after, and showed more 'typical' FBF values, such as 16.2ml/100ml/min^{-1} in subject 11J. The implication of this being that the plethysmography equipment was operating without fault on that day of testing. Such low post-HTT FBF values were only observed at 7W testing, and were not found in any participants at the pre-intervention and 3W test points (where participant numbers were higher), further supporting the idea that the readings were the result of adaptation and not error. It is unlikely that the low values could be the result of an inflation fault because the slope of the VOP trace showed a linear increase throughout the entire inflation period, indicating the cuff was causing the intended occlusion of the arm. With all these factors in mind it seems likely that the very low readings obtained from subjects 9J and 14J are valid and are due to a particularly strong adaptation response to the HA stimulus.

As nobody has yet investigated the thermoregulatory adaptations to post-exercise sauna bathing this is the first evidence to suggest this form of heat acclimation can stimulate adaptations to modify the control of the peripheral vasculature during heat stress. These data also reveal that 3 weeks of intermittent post-exercise sauna bathing may not be sufficient to produce a modification to FBF, but that 7 weeks may be. As the participant numbers for 7 weeks were relatively low (N=5) further testing with more participants would be required to truly validate this.

The findings of the current study agree with the results of the much earlier investigations of Eichna et al. (1950) and Wyndham. (1951), which showed reduced FBF with HA under hot fixed workload conditions. Eichna found that post acclimation peripheral blood flow (PBF) post exercise reduced from 2.6L/m²/min to 1.5L/m²min, and to allow for comparison with the current study this translates to 57.7% of the pre-HA value. The current study found pre-intervention to 7W, FBF reduced to 47.8% of pre-HA levels, indicating that 7 weeks of post-ex sauna may be a stronger stimulus for circulatory adaptation than the 10 consecutive days of active HA employed by Eichna's study. It is important however to consider that Eichna measured whole body PBF. It should not be assumed that the FBF measured in this study can represent the entirety of the peripheral circulatory response, therefore the comparison to Eichna's finding is not without some limitation. Furthermore Eichna's data is for only 3 participants, so the reliability of the finding is likely low. A much more recent study by Pallubinsky et al. (2017) found similar results to the current study following a passive 'mild' heat acclimation. Participants rested for 6 hours per day in 33°C/22%RH conditions for 7 days, a protocol designed to represent vacation or heatwave conditions. Following acclimation participants exhibited a modestly reduced core temperature, an attenuated increase in forearm

blood flow (-28%) and no increase in sweat loss; findings highly similar to those of the current study.

Reductions in FBF have previously been accounted for by improvements in other heat loss mechanisms reducing the need for heat loss from the peripheral circulation (Eichna et al., 1950, Wyndham, 1951). In the case of the current study it is also believed improvements in the efficiency of sweat function are responsible for the observed FBF reduction at 7 weeks. The improved sweating function results in an attenuation of core temperature rise, and core temperature alone is a significant driver of the skin blood flow response (Caldwell et al., 2015). In the current study, core temperature rise was significantly attenuated over the course of HA (figure 9a), therefore explaining some of the reduction in FBF over time. Interestingly the FBF sensitivity data (Figure 11e), which controlled for changes in T-rec with HA, showed a significantly reduction from Pre-intervention to 7W. This indicates that reductions in FBF were not solely the product of a reduced core temperature rise with HA. Skin temperature is also known to influence forearm blood flow. Should FBF sensitivity have been calculated from mean body temperature then a different sensitivity outcome may have occurred. Core temperature was used for sensitivity calculations as most of the existing literature applies this method, which also reports that FBF sensitivity increases with active HA (Roberts et al., 1977; Lorenzo & Minson, 2010; Gisolfi & Wenger, 1984; Sawka et al., 2000, 2011; Tyler et al., 2016). Fox et al. (1963) however conducted a passive HA study using the HWI method for 12 to 24 days. In the results of the study figure 4 plots oral temperature against forearm blood flow, showing the increase in FBF response to heating is initiated earlier with HA (threshold shift), however the slope (sensitivity) of FBF increase shows no change with HA. This finding suggests passive HA may not elicit an increase in FBF sensitivity, as is reported to occur with active HA; better supporting the findings of the current study. It should also be

considered that these previous studies calculated sensitivity via methods that allowed for continuous measurement of blood flow rather than the two pre and post HTT VOP measures as used in the current study. Thus, the change in slope used to calculate FBF sensitivity is likely not to be as reliably as studies that used continuous measures of SKBF.

In summary the reduced post-exercise FBF and reduced FBF sensitivity appear to be adaptations that allow for greater perfusion of the core, thus reducing whole body circulatory demands and alleviating strain on the cardiovascular system (Gonzales-Alonso, 2008). A reduced PBF should also allow for the circulation to be better directed for the improved perfusion of exercising muscle, which may in part explain the improvement in RPE. Increased perfusion of the core would also better protect the function of vital organs and reduce the risk of syncope (Periard et al., 2015; Liang et al., 2013). The reductions in post-exercise FBF and FBF sensitivity are possible due to an increased efficiency of sweating at eliminating body heat.

5.3.3: Sweating

It has already been established that under the conditions of this study the dry heat loss mechanisms of conduction, convection and radiation are ineffective due to the higher environmental temperatures creating a thermal gradient towards the body. Therefore in the current study, it was hypothesised that HA would improve thermoregulation by improved evaporative cooling via increased sweat loss. Improved thermoregulation occurred as shown by reduced T-rec's, however there was no evidence for increases in sweat loss. This finding is in agreement with Pallubinsky et al., (2017), but not Eichna et al., (1950) who reported a 10% increase in sweat loss was responsible for 75% to 90% of their reported reduction in core temperature. Wyndham et al., (1951) did not measure sweat loss, and the findings of Whitney,

1954 show evidence of sweat loss increases in some participants but reductions in others. The effect of passive HA on sweat loss under fixed work conditions seems unresolved, but the findings of this study supports the evidence of HA not increasing sweat losses under such conditions.

There is physiological reasoning as to why no increase in sweat loss was observed. Firstly it is appropriate to look at what factors stimulate the sweat response. One factor is core temperature, whereby a higher temperature increases sweating and a lower temperature attenuates it (Wyndham et al.,1965; Nadel et al., 1971). In the current study T-rec was consistently lower than pre-intervention after 3W and even lower after 7 weeks. The lower core temperatures achieved by acclimation responses would therefore signal for an attenuated sweat rate. Resting T-rec was also reduced, although not significantly. A re-setting of resting core temperature can be accompanied by a threshold shift for sweating, resulting in earlier sweating onset (Nadel et al., 1974; Roberts et al., 1977.; Patterson, 2004). An earlier sweat onset results in cooler skin in the early stages of the HTT, and helps maintain cooler skin throughout the HTT. This is of particular relevance as skin temperature is also a stimulus for sweating, with higher skin temperatures increasing sweating, and lower temperatures attenuating it (Wyndham et al.,1965; Kondo et al., 1997). At both 3W and 7W mean skin temperature was consistently lower than pre-intervention throughout the HTT. This general trend for reduced skin temperatures would contribute to an attenuated sweating response, although the influence of skin temperature on sweating is much less severe than core temperature (Wyndham, 1965; Sawka et al., 1996).

It has also been found that skin blood flow is tightly linked to the sweat response. SKBF can independently modify sweat rate, with reductions in SKBF attenuating sweating (Wingo et al., 2010). In the case of the current study, there is some evidence to support this as figure 11c

shows a progressive decline in post-ex FBF from pre-intervention to 3W to 7W and this is mirrored by a progressive decline in sweat loss over the same time course in this cohort (figure 12a). It appears that the reduced core and skin temperatures as well as reduced SKBF with HA are all potential explanations as to why no increase in sweat loss was found with HA in the current study.

Though total sweat loss was unchanged with HA it is believed that other sweat based adaptations developed with HA, and were responsible for the measured improvements in thermoregulation.

One such adaptation is the modification of sweat composition. HA has been shown to dilute the sweat through increased electrolyte reabsorption, in particular sodium (Dill et al., 1938; Allan & Wilson, 1971; Ogawa et al.,1982; Chinevere et al., 2008). A more dilute sweat is more easily evaporated due to a widening of the water vapour gradient between the skin and surrounding air (Tyler et al., 2016). This increased efficiency of sweat evaporation, reduces the need for such high sweat rates to remove heat from the body, a logical adaptation to explain the findings of the current study.

Another adaptation known to occur with HA is a change in the distribution of sweat across the body. Though sweat glands are located across almost all regions of the body the degree of activation of these glands and their sweat rate is not universal (Patterson et al., 2004). HA has been shown to improve the distribution of sweat production across the body, improving total skin wettedness, thus increasing whole body evaporation and facilitating heat loss (Ravanelli et al., 2018). It has previously been believed that HA would shift sweat secretion more to the periphery due to its higher surface area to volume ratio compared to the core, enabling a more effective evaporation of sweat. Early studies (Hofler, 1968; Regan et al., 1996; Schvartz et al,

1979) showed evidence to support this, however there were methodological limitations. A more recent study (Patterson, 2004) found improved sweat gland activation across the body, but no clear evidence of any preferential redistribution of sweating to the periphery. Based on this evidence it seems possible that participants in this study may have increased their sweat gland activation across the body, but it is unlikely that increases in sweat gland activity were directed more towards the periphery. Improved sweat gland activation (even without a redistribution to the periphery) could aid evaporative heat dissipation across the body (Ravenelli et al., 2018); meaning increased sweat rates are not required to remove heat, which further helps to explain the current study's findings.

Though this study did not measure threshold shifts, it is well documented that HA can modify the threshold for the onset of sweating, with sweating occurring at a lower body temperature than in the pre acclimation state (Nadel et al., 1974; Roberts et al., 1977). The findings of Patterson (2004) suggest that sweat onset occurs following the same absolute change in mean body temperature. As HA induces lower resting body temperatures, the absolute temperature at which sweating in initiated is lower following HA. In the current study, the 3W cohort saw no significant change in resting T-rec, so an earlier onset of sweating is unlikely. Despite this, mean skin temperature in the early stages of the HTT (0-10 minutes) was lower following 3W sauna intervention (figure 4a), indicating improved evaporative heat loss. Participants in the 7W cohort also showed reduced mean skin temperature at 3W as well as 7W. As 7W resting T-rec was reduced by 0.23°C, this may have produced a threshold shift in sweating, enabling earlier heat elimination. This may explain the attenuation of skin temperature rise as HA progresses (see figure 10a). Henane and Valatx, 1973; Shido et al., 1999; Beaudin et al., 2009 have all previously shown that passive heat exposure is effective at inducing threshold shifts in sweating; and the current study shows indirect evidence in support of such findings.

As discussed, skin temperatures showed some reduction with HA, likely due to improved sudorific function. A reduced skin temperature at an elevated core temperature widens the thermal gradient from core to skin, facilitating heat dissipation to the periphery. As HA reduced core temperature in this study a reduction in skin temperature helps to maintain this thermal gradient and thus the control of core temperature. The dynamics of heat transfer are somewhat complicated though, with a reduced skin temperature also resulting in a widened thermal gradient between skin and the environment, thus increasing heat gain from the environment.

It is clear there are numerous physiological explanations as to why no increase in total sweat loss was observed with HA, including reduced stimulation for sweating from core temperature, skin temperature and peripheral blood flow. Improvements in the composition and distribution of sweat would also result in improved heat elimination per unit of sweat secreted, reducing the need for increases in total sweat output. Data from sweat sensitivity calculations provide more direct evidence to support the conclusion that sudorific adaptations did occur with HA. Sweat sensitivity significantly increased in the 3W cohort from pre-intervention to 3W, with (figure 6c) showing 8 of the 10 participants improved their sweat sensitivity. Sweat sensitivity did not change significantly in the 7W cohort, though mean sensitivity did increase with each test point. As the current study was not able to measure continuous sweat rates during the HTT it is unknown whether a shift in the onset of sweating occurred with HA. Nonetheless, T-rec rise was attenuated with HA despite no increase in sweat loss, indicating sweat based adaptations; which could be centrally induced such as a threshold shift, and/or peripherally based (increased sweat gland sensitivity). These finding along with analysis of the FBF data support the conclusion that the improvements in

thermoregulation with HA were the result of sweat based adaptations and not due to increased heat losses from the peripheral circulation.

To summarise, the current study did not observe increases in sweat loss with HA, a finding in discordance with the study hypothesis. Sweat sensitivity data does however indicate that sudorific adaptation occurred with HA. Improvements in sweat efficiency resulted in reduced core and skin temperatures, which likely explains much of the reduction in 7 week FBF.

5.3.4: Heart Rate

Resting HR data showed significant reductions in the 3W cohort, but no significant change between 3W and 7W in the 7W cohort. This lack of a significant reduction in the 7W cohort may be due to an underpowered statistical analysis. The observed reductions in resting heart rate are likely due to the effect of an expanded blood volume on the circulation, as PV expansion is proven to reduce HR (Sawka et al., 1983). As Scoon et al. (2007) and Stanley et al. (2015) both reported plasma volume expansion in their post-exercise sauna studies, it is possible that PV expansion occurred in the current study, and modified the resting HR.

Exercising HR data shows a much more distinct change following HA than resting HR data. In the 3W cohort, reductions in mean HR relative to pre-intervention are evident at 10, 20 and 30 minutes into the HTT. Data from the 7W cohort showed little difference at 7W compared to 3W data. The lack of further HR reduction from 3 to 7 weeks is unsurprising as HR reductions are an adaptation that develops rapidly after 4 to 5 consecutive days of HA, and is practically complete by 7 days (Periard, Racinais and Sawka, 2015). 7 sessions of consecutive HA would translate to 2-3 weeks of intermittent HA in the current study. This means by 3

week testing in the current study HR adaptations were likely to be complete or nearly complete, explaining the negligible change in HR from 3 to 7 weeks.

One explanation for the exercising HR reductions is the influence of an expanded PV, as discussed with resting heart rate, however with exercise there are additional stimuli that influence HR. HR during prolonged exercise in the heat is higher than matched intensity exercise in cool conditions, this phenomenon is known as cardiovascular drift (Wingo et al., 2012). Traditionally it has been believed this elevation in HR is due to increased cardiovascular strain created by the demand for high PBF during exercise, and this is referred to as the 'cutaneous circulation model' (Rowell, 1986). The model states that increased PBF in the heat causes pooling of blood in peripheral tissues, which reduces venous return, forcing HR to rise in order to maintain Q. Evidence to support this model is lacking however. Furthermore, data from the current study goes against this model. A significant reduction in FBF is found at W7, and according to Rowell's model this should improve central venous pressure, increase venous return, allow increased SV and consequently HR should reduce; but this does not occur. HR was not significantly different between W7 and W3 despite the difference in measured FBF over the same period. This suggests peripheral blood flow may not act as a significant influence on exercising HR.

A more contemporary theory to explain cardiovascular drift in the heat is that increases in body temperature cause increased stimulation of sympathetic pathways, which increases HR and forces reductions in SV due to reduced diastolic filling time (Coyle and González-Alonso, 2001). It has been shown that a clamped core temperature with exercise results in no decline in SV, whereas increases in core temperature reduced SV and increase HR (González Alonso et al., 1995, 1997). As the current study found significantly reduced core temperatures following HA this could largely explain the lower exercising heart rates following HA.

5.4.5: Perceptual responses

It was hypothesised that perceptual responses to exercising in the heat would improve following HA, meaning reduced RPE, and improved thermal comfort and thermal sensation scores. For all of these measures the hypothesis was confirmed.

It should be noted that the design of the HTT in this study did not allow for perceptual responses to influence behaviour, as this would have to be assessed either through a 'time to exhaustion' style test or a time trial where participants set their own pace. As this study's HTT was just 30 minutes and conducted at a fixed sub-maximal exercise intensity perceptual responses had no influence on exercise behaviour. However, any changes in perceptual response for the same absolute sub-maximal exercise intensity is ideal for assessing adaptation. In an applied context changes in perception could be responsible for altered pace selection (Heathcote et al., 2018) and change in exhaustion point (Stevens et al., 2018); both of which could have significant implications in regard to performance outcomes.

RPE

In the 3W cohort reductions in RPE relative to pre-intervention were seen 10, 20 and 30 minutes into the HTT. Core temperature is a known contributor to RPE (Travlos & Marisi, 1996), therefore, it is fitting that the attenuated rise in T-rec following 3W intervention corresponded to an attenuated rise in RPE during the HTT. In the 7W cohort a similar trend was observed to that of the 3W cohort. RPE reduced from pre-intervention to 3W to 7W, which was mirrored by reductions in T-rec over the same period, reinforcing the role of core temperature has in setting perceptual responses.

Whilst some of the reduction in RPE is due to improved thermoregulation it is also possible that improved fitness is a contributor. As mentioned in the methods, this study was a part of a

larger study that also assessed changes in aerobic fitness. Following HA participants showed significant increases in VO2 max and lactate threshold when running on a treadmill. As the HTT was conducted using a fixed workload, following HA this HTT would have been conducted at a lower percentage of the participants VO2 max and lactate threshold, meaning the relative effort applied to complete the running would have been reduced with HA. These reductions in relative effort result in reduced RPE (Cheuvront et al., 2010).

Thermal Comfort and Thermal Sensation

The measurement of thermal comfort relates to how an individual interprets their thermal state, whereas thermal sensation is simply recognising how hot or cold the body feels (Heathcote et al., 2018). Of these 2 measures it is argued that thermal comfort is of greater importance as it is the interpretation of body temperature that leads to behavioural thermoregulation (i.e., pacing, volitional exhaustion) (Heathcote et al., 2018). At 3 weeks significant reductions (improvements) were seen in both thermal comfort and thermal sensation scores, with further reductions found after the full 7 weeks. These perceptual changes align with the measured reductions in core and skin temperatures with HA. This is to be expected as afferent feedback of core and skin temperature are the primary dictators of the perception of thermal states (Nakamura & Morrison, 2007; Romanovsky, 2014).

6. Limitations

The primary limitation of the current study was low participant numbers as a result of dropout. Dropout was particularly high from 3W to 7W, resulting in low powered statistical analysis, especially for the control group where n = 2. The original intention of the study was for all participants to complete 7W of intervention, resulting in one cohort and one set of statistical analyses.

The post-exercise VOP held a limitation in its method. Ideally the VOP measure would be taken at the immediate cessation of exercise where the influence of exercise and environment on blood flow control will be most evident. In the current study VOP was conducted over 6 minutes post-exercise, meaning the full influence of exercise was missed owing to this recovery period. This 6 minute period was required due to the need to obtain other measures, move the participant and set-up equipment. Typically VOP is obtained immediately post-exercise by immobilising the arm with a sling, however whilst this works for ergometer cycling it is not feasible for the treadmill running used in the current study. Though the full influence of exercise could not be assessed in this study the post-exercise VOP data is still of value as the exercise period was a key driver for raising core temperature and thermoregulatory responses. As the post-exercise VOP was conducted in the heated chamber core temperature will have remained elevated 6 minutes post-exercise and thermoregulatory responses would still have been occurring.

It was surprising that sweat output showed no change following HA, however sweat sensitivity data helped justify this result. It would have been valuable to have measured sweat rate during the HTT, as the point of sweat onset could be established and any threshold shifts

determined; which would allow for more accurate sweat sensitivity calculations. Measures of sweat composition and sweat gland activation would also have been valuable to support the conclusions that were made.

A potential limitation of the perceptual measure data collection method is that the participant is capable of providing answers that they believe would be desirable for the outcomes of the study. Due to the nature of the study design participants could not be blinded to their condition and were largely aware that the sauna intervention was intended to facilitate exercise performance, which may have influenced their response.

7. Perspectives

These findings are the first to show that post-exercise sauna bathing is an effective method of heat acclimation. 3 sauna bathing sessions a week for 3 weeks proved sufficient at inducing significant improvements in exercising thermoregulation. 7 Weeks of heat acclimation resulted in further improvement in thermoregulation, however the rate of improvement was diminished and sauna adherence was lower. The reduced adherence suggests 7 weeks of intermittent HA proves challenging for athletes, and this should be considered for future research or practical application. Despite not reaching target sauna exposure time the participants of this study have demonstrated that post-exercise sauna bathing is an effective method of heat acclimation.

The observed reductions in core temperature after 3 weeks of acclimation are similar to those of other passive based HA protocols (figure 1), and data from 7 weeks of acclimation shows a greater core temperature reduction than any other passive HA study. These findings suggest post-exercise sauna bathing is one of the most effective strategies for preparing to compete in heat. Due to decay of adaptations, heat acclimation is best conducted as close to competition as possible (Daanen et al., 2018). It is therefore advised that athletes implement the protocol of the current study 3 weeks before competition, or if scheduling permits 7 weeks prior to competition in order to achieve the greatest level of adaptation. Whilst a 3 week or 7 week protocol is a large time investment for athletes the post-exercise sauna method is designed to not interfere with an athletes training, avoiding the disruption to training that occurs when using active HA protocols.

Though this method of HA induced clear adaptations it should be considered that the specificity of these adaptations may mean post-exercise sauna is better suited to hot, relatively

dry conditions. Adaptations from the current study are believed to be sweat based, a consequence of the high temperature and low humidity of the sauna acclimation stimulus. This form of HA may not prove as effective for athletes who need to acclimate for humid, more tropical conditions where sweating is less effective and there is a greater reliance on dry heat exchange mechanisms. Furthermore this study assessed thermoregulatory performance via a running based HTT, without further research it can not be guaranteed that this HA protocol is as effective at acclimating athletes who train and compete in other exercise modalities such as cycling.

8. Conclusions

This study is the first to assess whether post-exercise sauna bathing is an effective method of heat acclimation. Acclimation was evident as exercising core temperatures during a heat tolerance test were significantly reduced following 3W and 7W of intervention. After 3 weeks the reduction in end exercise core temperature was -0.34°C, similar to the -0.33°C of other passive HA protocols. This shows the current method is as effective as existing passive protocols, however at this point acclimation was not complete. By 7 weeks participants showed reductions in end exercise core temperature of -0.63°C, a reduction in excess of any other existing passive HA study. As other studies are much shorter in their acclimation duration it is unknown whether other passive methods such as hot water immersion have similar potential if conducted over a similarly long period of time to the current study.

The underlying physiological mechanisms that enabled core temperature reductions did not fully align with the hypothesis. FBF did show reductions by 7 weeks as hypothesised, however it was also anticipated that the lack of dry heat loss from the periphery would be compensated for by increased sweat loss; but this did not occur. With support from sweat sensitivity data it was concluded that other sudorific adaptations were occurring with HA, such as changes in sweat composition, improved sweat distribution and earlier sweating onset. Resting and exercising heart rate reduced with HA, as hypothesised. Such reductions were likely due to plasma volume expansion, which modified circulatory control. In exercise, the reduced core temperature produced by HA would have attenuated sympathetic stimulation of the heart, also contributing to the reduction in HR seen with HA.

Perceptual responses showed significant improvements following HA, with participants reporting significantly improved thermal comfort and thermal sensation. This was a logical

finding as the body was objectively cooler following HA. RPE also showed significant reductions following HA. Acclimated participants experienced increases in their VO2 max and lactate threshold, consequently following HA the fixed workload HTT would have been conducted at a lower relative intensity than pre-acclimation, explaining the reduction in RPE.

Though HA was evident, participants in the sauna group did not meet the intended exposure time, particularly between 3W and 7W. This was due to participants missing sessions, however when participants did attend sessions the 30 minute bathing time was met or very nearly met. 3 to 4 exposures per week for 7 weeks appeared to be too much of a commitment for the student athlete participants of this study, however as stated acclimation did occur even with a lower exposure than planned. As research on this specific form of HA is lacking the optimal time and frequency of sauna bathing is not known and warrants further research.

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