An Examination of the Influence of Age on Behavioural Thermoregulation Responses During Heat Exposure

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1 Abstract

Heat wave events are increasing in frequency and severity, resulting in many heat-related deaths in older adults. This thesis aimed to develop an understanding of how increasing age impacts thermoregulatory behaviour while exercising during heat exposure. The thesis also looked to identify what, if any, factors influence this relationship.

The first study examined the relationship between age and behavioural thermoregulation during walking exercise in 35°C. Thermoregulatory behaviour was assessed by having participants walk at a self-selected speed that elicited an RPE of 13 in 22°C and 35°C conditions. The multiple regression model did not significantly predict thermoregulatory behaviour (p = 0.406, R^2 = 0.156). None of the variables added statistical significance to the prediction (p > 0.05). For the male participants, the multiple regression model did significantly predict thermoregulatory behaviour (p = 0.038, R^2 = 0.469). Age added statistical significance to the prediction (p = 0.005). Men age ≥60 years were less able to implement thermoregulatory behaviours than men aged <60 years (p <0.05). For the female participants, the multiple regression model did not significantly predict thermoregulatory behaviour (p = 0.906, R^2 = 0.184). None of the variables added statistical significance to the prediction, p > 0.05.

The second study identified factors that influence the diminished ability to reduce exercise intensity in 35°C conditions in older men. No significant differences were evident between men aged 18-35 and ≥60 years in their anthropometry, physical activity and fitness, whole body sweat rate, cardiovascular, or thermal perceptual responses. The older men did not reduce walking speed when exposed to 35°C temperatures, but the young men did. Factors influencing thermoregulatory behaviour in men aged ≥60 years need to be identified as they offer potential intervention strategies to reduce heat stress and heat-related illness risk.

By using pre-warming and radiant heat lamps, the final study investigated the influence of skin and rectal temperature thermoregulatory behaviour in exercising men aged >65 years. Young and older men cycle at a fixed RPE of 13 for 30 min. Trials included a stable ambient control (22°C), changing ambient temperature (22 - 35°C), a pre-warmed stable ambient (rectal elevated 1°C, 22°C ambient), and a pre-warmed changing ambient (rectal elevated by 1°C, 22 - 35°C ambient). Young men implemented thermoregulatory behaviour in response to changing ambient conditions, whereas older men did not (thermoregulatory behaviour scores: Young CHC 1.09 ± 0.12, W+CHC 1.08 ± 0.16. Older CHC 0.94 ± 0.09, W+CHC 0.97 ± 0.07). This was despite similar skin and rectal temperature, and thermal perception responses between the two age groups. Older men are unable to adjust exercise intensity in response to changing ambient conditions, thus skin temperature is a less effective driver of thermoregulatory behaviours in older men than young men.

The primary outcome of this thesis is that older (≥60 years) men do not voluntarily implement thermoregulatory behaviours while exercising in 35°C conditions, whereas younger men do. No physiological or perceptual factors could be identified to explain this inability to implement thermoregulatory behaviours. This has public health implications, as older adults, particularly men, are unable, or unwilling to voluntarily reduce their heat exposure during heat wave events. This will increase their risk of heat-related illness putting strain on health and emergency services. Public health messaging should move to educate adults aged ≥60 years of their insensitivity to heat exposure, the reduced likelihood of them implementing necessary behavioural thermoregulation strategies, and the implications if they do not voluntarily adopt cooling strategies.

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4 Declaration of authorship

I, Alison Millyard declare that the thesis entitled:

An Examination of the Influence of Age on Behavioural Thermoregulation Responses During Heat Exposure,

and the work presented in the thesis are both my own, and have been generated by me as the result of my own original research. I confirm that:

- This work was done wholly or mainly while in candidature for a research degree at the University of St. Mark & St. John;
- Where I have consulted the published work of others, this is always clearly attributed;
- Where I have quoted from the work of others, the source is always given. With the exception of such quotations, this thesis is entirely my own work;
- I have acknowledged all main sources of help;
- Where the thesis is based on work done by myself jointly with others, I have made clear exactly what was done by others and what I have contributed myself;
- Parts of this work have been published as:
  Millyard, A., Layden, J. D., Pyne, D. B., Edwards, A. M., & Bloxham, S. R. (2020). Impairments to Thermoregulation in the Elderly During Heat Exposure Events. *Gerontology and Geriatric Medicine*, *6*, 233372142093243. https://doi.org/10.1177/2333721420932432

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Date:

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5 Publications

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6 Conference presentations

International Conference on Environmental Ergonomics – 2019 – Amsterdam

Influence of age on perceptual responses to thermal stress at rest and following exercise.

Alison Millyard, Joe Layden, Andrew M. Edwards, David B. Pyne, Saul Bloxham.

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8 List of Abbreviations

%	Percentage
~	Approximately
∑	Sum of
°C	Degrees Celsius
6MWD	Six minute walk distance
6MWT	Six minute walk test
ANOVA	Analysis of variance
ASHRAE	The American Society of Heating, Refrigerating and Air-Conditioning Engineers
BBC	British Broadcasting Corporation
BM	Body mass
BMI	Body mass index
bpm	Beats per minute
BSA	Body surface area
CA	California
CCC	Control trial with no pre-warming or heat lamps
CHC	Trial with no pre-warming and heat lamps activated
CI	Confidence interval
COVID-19	Coronavirus disease 2019
CRP	C-reactive protein
CVC	Cutaneous vascular conductance
d	Cohen's d effect size
DNA	Deoxyribonucleic acid
DP	Diastolic pressure
EEG	Electroencephalogram
EMG	Electromyogram
EP	Ethics proposal
EP050	Ethics proposal 50
EP103	Ethics proposal 103
EU	European Union
FL	Florida
$h \cdot d^{-1}$	Hours per day
HEPRU	Human and Environmental Physiology Research Unit
Hz	Hertz

IPCC	Intergovernmental Panel on Climate Change
ISAK	The International Society for the Advancement of Kinanthropometry
IUPS	International Union of Physiological Sciences
kg	Kilogram
$kg \cdot m^{-2}$	Kilogram per meter squared
KJ	Kilojoule
km	Kilometre
$L \cdot min^{-1}$	Litres per min
LBM	Lean body mass
LDF	Laser Doppler flow
L-NAME	N^G-nitro-L-arginine methyl ester
m	Metres
MAP	Mean arterial pressure
MET Office	Meteorological Office
METs	Metabolic equivalents
min	Minute
mL	Millilitre
$mL \cdot kg^{-1} \cdot min^{-1}$	Millilitres per kilogram per minute
$mL \cdot kgLBM^{-1} \cdot min^{-1}$	Millilitres per kilogram of lean body mass per minute
mm	Millimetre
mM	Millimolar
mmHg	Millimetres of mercury
$nl \cdot min^{-1}$	Nanolitres per min
NO	Nitric oxide
O	Older participants
O_2	Oxygen
OTS	One Touch Stockings of Cambridge
PASE	Physical Activity Scale for the Elderly
PU	Perfusion unit
RH	Relative humidity
RPE	Rating of perceived exertion
SD	Standard deviation
TRP	Transient receptor potential
TRPV	Transient receptor potential vanilloid

UAE	United Arab Emirates
UK	United Kingdom
US	United States
$\dot{V}O_2max$	Maximal oxygen uptake
$\dot{V}O_{2peak}$	Peak oxygen uptake
W	Watts
W+CCC	Trial with pre-warming and no heat lamps
W+CHC	Trial with pre-warming and heat lamps activated
$W \cdot kg^{-1}$	Watts per kilogram
$W \cdot m^{-2}$	Watts per meter squared
WBSR	Whole body sweat rate
WMO	World meteorological organisation
Δ	Change

9 Introduction

Extreme weather events, including heat waves, are becoming more frequent and this trend is set to continue (American Meteorological Society, 2012; Met Office, 2018). Tens of thousands of deaths have been caused by heat waves across Europe since 2000 (European Environment Agency, 2016). There are an estimated 1,500 heat-related deaths annually in the US (Epstein et al., 2005). A health centre in Paris recorded 2,814 deaths during the 2003 heatwave, 81% of these were in people aged >75 years (Epstein et al., 2005). In 2021, Canada experienced a heatwave that caused at least 130 deaths, many of those being from vulnerable populations including older adults (BBC, 2021). Weather-related heat deaths demonstrate that discrete groups of the population cannot cope effectively with extreme temperatures. Adults aged ≥65 years, in particular, have been identified as a sub-population at risk during extreme heat weather events (Åström et al., 2011). Fouillet et al., (2006) reported that during August 2003 in France, higher age was associated with an increased mortality ratio. A hospital in Lyon, France investigated their excess deaths and reported that 80% of their heat-related deaths were patients aged over 75 years (Vanhems et al., 2003). Across the US between 1999 and 2010, 8,081 heat-related deaths were recorded, approximately a third of these (2,901) were in adults aged ≥65 years (Centers for Disease Control and Prevention, 2016). This age group is increasing in size globally. In 2019 18.5% of the UK population was aged >64 years, and this proportion is expected to increase to 23.9% by 2039 (Office of National Statistics, 2021). Worldwide, the number of people aged >64 years is predicted to increase from 703 million in 2019, to 1.5 billion by 2050, making up 16% of the world's population (United Nations Department of Economic and Social Affairs Population Division, 2019). This increase in the number of older adults puts more people at risk during an increasing number of heat wave events. Overall, it is likely that many more people will continue to suffer illness and death during heat waves unless the understanding around the causes of heat death in older adults improves.

Current National Health Service (NHS) guidelines in the UK emphasise that people aged ≥75 years are at risk during heat wave events (NHS, 2019) despite strong evidence that those aged ≥65 years are at an increased risk of illness and/or death during heat waves (Åström et al., 2011; Centers for Disease Control and Prevention, 2016; Public Health England, 2019). Public Health England (PHE) had guidelines for how to address public health issues in England. Whilst PHE's heat wave advice advocated for modifying behaviour during heat waves (Public Health England, 2020), it did not emphasise who is most at risk. Neither the NHS nor PHE mention that individuals may underestimate their personal risk level during heat wave events. When surveyed, 35% of 908 people living in North America (Dayton, US, Philadelphia, US, Phoenix, US, and Toronto, Canada) aged ≥65 years stated that

the heat posed no danger to them personally (Sheridan, 2007). This lack of acceptance of personal risk will likely result in a reluctance to undertake any mitigating strategies during heat wave events. This was evidenced in the same survey where between 35% and 57% of respondents stated that they did not alter their behaviour when a heat warning was issued (Sheridan, 2007). During the 2003 French heat wave adults aged ~82 years old who adopted behavioural strategies reduced their risk of dying (Vandentorren et al., 2006). For example, those who visited cool places had an odds ratio of dying of 0.54, whereas those who stayed home had a 3.90 odds ratio of death. Drinking >1 L of water a day had an odds ratio of 1.00 versus 2.64 and 16.84 for drinking 0.5-1 L and <0.5 L of water a day, respectively (Vandentorren et al., 2006). Older adults who are willing and able to implement behavioural cooling strategies are clearly at an advantage during heat waves, however, there is a significant number who do not identify as at-risk despite being >65 years old, and thus reluctant to employ these simple but effective strategies. This indifference could result in mortality rates during heat waves remaining high within the ≥65 years age group.

Exposure to hotter than usual temperatures pose a thermoregulatory challenge to the human body. This is particularly challenging when temperature increases occur suddenly, as with a heat wave event in the spring, as this impedes opportunities for acclimatisation. Heat wave health issues are not isolated to hotter climates, as individuals in moderate climates are also susceptible to increased hospital admissions and deaths (Johnson et al., 2005). The 10 warmest years on record (since 1884) in the UK have occurred since 2002 (2002-2007, 2011, 2014, 2017, 2018), and the 10 coldest years all occurred before 1964 (Met Office, 2019a). Individuals in more moderate climates are at a disadvantage during a heatwave event as they have had little to no time to acclimatise to the heat, whereas those residing in hotter climates typically have had ample time to partially or fully acclimatise (Knowlton et al., 2009). This effect can be seen with the variation in heat wave definitions seen around the world.

The World Meteorological Office (WMO) classifies a heat wave as five or more consecutive days with a maximum temperature 5°C above the normal daily maximum temperature, with the reference period deemed the years between 1961 – 1990 (Frich et al., 2002). However, many countries have adopted their own definition of a heat wave. The threshold temperature for a heatwave in the UK varies slightly across the country from 25-28°C for three consecutive days (Met Office, 2019b). However, in Adelaide, Australia the criteria for a heat wave is warmer than the UK with a definition of

5 consecutive days with temperatures of ≥35°C, or three consecutive days with temperatures of ≥40°C (Australian Government: Bureau of Meteorology, 2010).

These variations in heat wave definitions demonstrate how humans can adapt to different climates given time. Humans living in different regions adapt to their local ambient conditions. Residents of cold regions (Russia) have fewer active sweat glands than residents of temperate (Japan) and tropical areas (Philippines; Hori, 1995). Thus, the degree of strain and health risk associated with heat can be relative to the natural climate an individual lives in. An Australian resident could cope with a moderate northern European heat wave relatively easily. The issue is how acclimatised are people to hot conditions and implementing appropriate behavioural thermoregulation strategies. Consequently, during a heat wave event with a sudden increase in ambient temperature, hindering full acclimatisation of autonomic thermoregulatory mechanisms, many people succumb to heat-related illness and death. These deaths are preventable through behavioural thermoregulatory strategies, both during exertional and passive heat exposure, as shown with modelling and laboratory-based research. Voluntarily reducing exercise intensity prevents dangerous increases in core temperature (Flouris & Schlader, 2015). At rest, using an electric fan in temperatures <42°C is effective in reducing heat strain (Jay et al., 2015; Ravanelli et al., 2015). Wearing a wet t-shirt is also a useful intervention to reduce heat strain, however, coupling its use with an electric fan is not as effective at ameliorating the rise in core temperature than no intervention (Cramer et al., 2020). These interventions are relatively cheap and effective in helping to reduce heat-illness risk. Behavioural strategies rely on effective afferent signalling relaying temperature information from the skin and core so that physiological responses can be implemented via efferent neuron signals. Afferent and efferent conduction speeds decline with age (Navaratnarajah & Jackson, 2017). Weather-related heat deaths demonstrate that discrete groups of the population cannot cope effectively with sudden increases in environmental temperatures. If these vulnerable populations are able to access behavioural thermoregulation interventions they offer methods of reducing heat strain (physiological response to environmental heat stress (Huang et al., 2020), however, they are only effective if they are implemented.

Ageing impacts thermoregulation in several ways (Figure 9-1***Figure 9-1.*** *Factors contributing to increased risk of heat illness and death in ageing.*). In laboratory-based, cross-sectional research, older adults (≥50 years) store 1.3 - 1.8 times more body heat when exposed to the same heat load than younger adults (19-30 years) during both exercising and passive heat exposure in both humid and dry conditions (35-44°C, 15-30% relative humidity (RH); Kenny et al., 2017a; Larose et al., 2014; Stapleton et al., 2015a). The higher heat storage in the older individuals is due to a reduction in heat loss (Larose

et al., 2014) caused by an attenuated sweat response (Kenny et al., 2017a; Stapleton et al., 2015a) and increased dry heat gain (Kenny et al., 2017a). These studies clearly show a reduced thermoregulatory function with ageing.

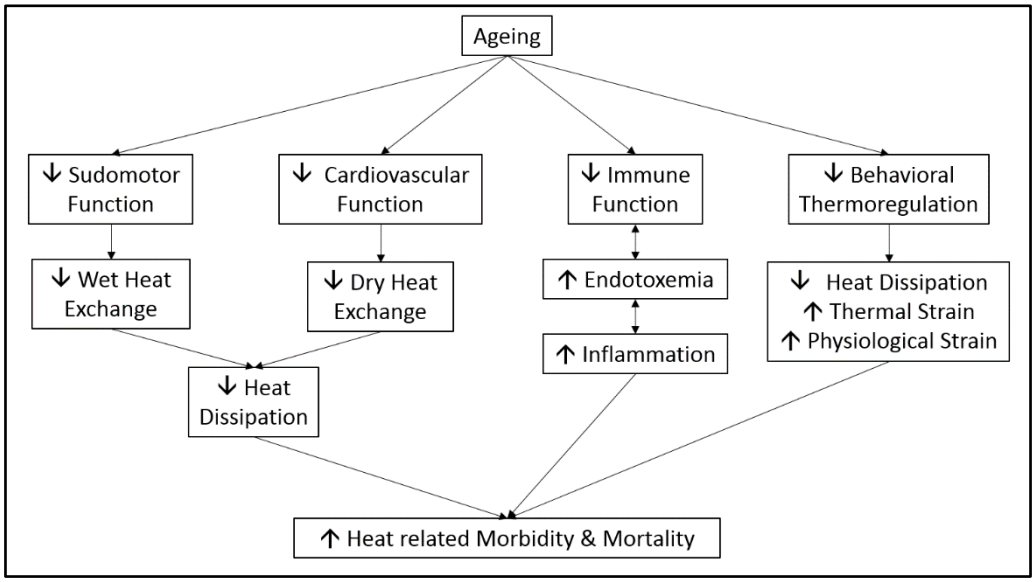

Figure 9-1. *Factors contributing to increased risk of heat illness and death in ageing. Reproduced from Millyard et al., (2020).*

Sweating is a critical mechanism for heat loss in humans, particularly when ambient temperature is above skin temperature, as dry heat exchange results in heat gain in these situations. Sweating function declines with age at differing rates. Sudomotor function declines first in the legs, followed by progressive decrements in the upper body (Inoue et al. 2004). Reduced sweating capacity has been observed by age 40 years (Dufour & Candas, 2007; Larose et al. 2013). Loss of sweating capacity originates from the reduced function of each sweat gland rather than a reduction in the number of sweat glands (Inoue et al. 2004) and is thought to be caused by local rather than central factors (Dufour & Candas, 2007). Older adults have a higher core temperature threshold for the onset of sweating when exposed to 40°C and 40% RH. Men aged >60 years at rest started sweating at a core temperature of 37.0°C whereas men aged <40 years started sweating at 36.7°C (Sagawa et al. 1988). This difference in onset of the sweating response was also observed in females, with women aged 58 years sweating at core temperatures of 37.3°C and 37.5°C compared with 37.1°C and 37.2°C for women aged 23 years while exercising (3 x 30 min cycling at 250 W, 325 W, and 400 W) in the heat (40°C, 15% RH; Stapleton et al. 2015). The delayed onset of sweating coupled with the inability to increase and maintain a high sweat rate will impair the effect of cooling from sweat reducing its effectiveness, resulting in a higher core temperature and greater heat strain in adults aged ≥65 years. Individual sweating rates vary greatly. Sato & Sato, (1983) reported sweat rates ranging from 0.8 to 10.1 nl·min^{-1} per gland. Therefore, the decline in sweat response is likely to be highly individualized.

Nitric oxide (NO) stimulates sweat production in young adults (Amano et al. 2017; Stapleton et al. 2014). However, NO inhibition has little effect on sweat rate in men aged >60 years (McGarr et al. 2019; Stapleton et al. 2014), thus they rely on non NO mediated mechanisms to induce sweating.

A high level of aerobic fitness increases sweating capacity in young and older adults (Fritzsche & Coyle, 2000; Tankersley et al. 1991). Young men (25 ± 2 years) who regularly undertake endurance exercise ($\dot{V}O_2$peak 62.4 ± 1.7 mL·kg^{-1}·min^{-1}), have a higher whole body sweat rate when cycling than young men (27 ± 1 years) who do not undertake regular endurance training ($\dot{V}O_2$peak 44.2 ± 1.8 mL·kg^{-1}·min^{-1}) (Fritzsche & Coyle, 2000). After two hours of intermittent cycling (20 min bouts at 50, 70, and 90% of $\dot{V}O_2$peak, interspersed with 30 min rest) in ~23-24 °C, 50 ± 5% relative humidity, whole body sweat rate was significantly higher in the trained men than the untrained men (15.7 ± 1.9 v 9.1 1.6 g/kg, p <0.05). Mean age, body mass, surface area, and maximal heart rate were all similar between the groups, indicating that the aerobic fitness of the men was the factor affecting the whole body sweat rate. Tankersley et al. (1991) examined how ageing influences this relationship by recruiting three groups; a young group (29 ± 1 years; $\dot{V}O_2$max 44.0 ± 2.7 mL·kg^{-1}·min^{-1}), an older group matched for fitness with the young group (64 years; $\dot{V}O_2$max 46.4 ± 2.1 mL·kg^{-1}·min^{-1}), and an older unfit group (66 ± 1 years; $\dot{V}O_2$max 32.9 ± 2.1 mL·kg^{-1}·min^{-1}). When these groups cycled in the heat (30°C, 50-60% RH) for 20 min at 67.5% $\dot{V}O_2$max, the young group had the highest chest sweat rate and the older unfit had the lowest (significantly lower than young at 10 min), but the older fit group's chest sweat rate was between the young and older unfit groups. This study demonstrates that ageing does reduce local chest sweat rate in men exercising in the heat, but that maintaining a high level of aerobic fitness will ameliorate this decline, offering a greater level of evaporative heat dissipation than unfit older men. As maintaining a high level of aerobic fitness into older age requires a high volume of training, which is unattainable for many, work should focus on how ageing and physical activity levels, rather than aerobic fitness level, affect this relationship.

With ageing, the cardiovascular system experiences functional and structural changes (Edwards & Hettinga, 2018). Total blood volume decreases (Davy & Seals, 1994), reactive oxygen species increase and NO availability reduces, yielding a decrease in endothelial-dependent dilation and a reduced blood flow (Donato et al., 2015). These alterations put added stress upon the cardiovascular system. Dry heat transfer relies on the cardiovascular system redistributing blood towards the skin, a mechanism that will be impaired in those with the additional strain of cardiovascular disease. The risk of death during a heat wave is increased in cardiovascular disease patients, with an odds ratio of 2.3-7.2 (Naughton et al. 2002; Semenza et al. 1996), and a relative risk of 2.0-2.4 (Kaiser et al. 2007; Wainwright et al. 1999). Recent work has demonstrated that in healthy active older adults free from cardiovascular disease, calf blood flow is attenuated during passive heat exposure (Kenny et al. 2017).

Older adults increase their skin blood flow ~2-3 fold less than their younger counterparts during passive (supine rest in water perfused suit at 50°C until thermal tolerance reached) and active (seated rest for 50 min, cycling for 20 min at 35% $\dot{V}O_2$max, 30 min at 60% $\dot{V}O_2$max in 36°C, 20% RH) heat exposure (Ho et al. 1997; Minson et al. 1998). This reduction in skin blood flow is related to a reduced stroke volume attenuating the increase in cardiac output (Minson et al. 1998). These cardiovascular differences were observed in healthy older adults during passive heat exposure, without the additional demand of supplying exercising muscle with adequate blood flow, or the impairment of cardiovascular disease. Attenuated skin blood flow will reduce dry heat loss, and therefore increase heat strain on the body. Healthy ≥65 year-olds will struggle to dissipate heat effectively compared with their younger counterparts, resulting in increased thermal and physiological strain. Those with the additional strain of cardiovascular disease will incur a greater physiological and thermal strain.

As low levels of fitness is associated with an increased risk of heat illness during heat exposure (Westwood et al., 2020), work has looked at how maintaining a high level of aerobic fitness affects the skin blood flow response seen with ageing. Men aged 66 ± 1 years with a maximal oxygen uptake of 32.9 $mL \cdot kg^{-1} \cdot min^{-1}$ had half the forearm blood flow of men aged 29 ± 1 years with a maximal oxygen uptake of 44.0 $mL \cdot kg^{-1} \cdot min^{-1}$ when cycling at 65% of maximal oxygen uptake for 20 min in the heat (30°C; 40-50% RH; Tankersley, Smolander, Kenney, & Fortney, 1991). These older men were considered fit for their age as their aerobic capacity was within one standard deviation of their age-adjusted predicted value. A highly fit older group (64 ± 2 years) with a similar maximal oxygen uptake (46.4 $mL \cdot kg^{-1} \cdot min^{-1}$) to the young men were able to maintain a similar forearm blood flow to the young men. The volume of exercise training required to maintain such a high maximal oxygen uptake into older age is unfeasible for most. As such ageing will result in a decline in skin blood flow for the general population.

Adapting behaviour to the surrounding environment is an important aspect of thermoregulation (Flouris & Schlader, 2015). Behaviour changes such as seeking shade or air-conditioned buildings, increasing fluid intake, removing clothing layers, or taking a cooling shower can help prevent the onset of heat illness (Harduar-Morano et al. 2016). Behavioural changes to environmental temperatures appear to be driven by thermal discomfort (Gagge et al. 1967). Waldock et al. (2018) explored the perceptual responses of adults aged ≥65 years during exercise in a hot environment and reported that despite increases in ambient temperature from 25°C to 35°C, and concomitant increases in skin and core temperature. Participants did not perceive thermal comfort to be more uncomfortable when cycling at 6 metabolic equivalent of task (METs) for 30 min. If ≥65 year-olds have a diminished perception of discomfort despite being physiologically challenged, they may be reluctant to adapt

their behaviour(s) to reduce the thermal challenge. Studies are required to examine the role of perception of heat and physiological strain in older adults to determine causal factors.

With an ageing population and increases in the severity and frequency of extreme weather events, heat illness will become a major public health issue with a local, national, and global impact. A key issue is the apparent lack of perception of thermal and physical strain during heat exposure in ≥65 year-olds. Adapting behaviour during heat exposure has the potential to minimise the risk of heat illness, and research is vital in establishing how ≥65 year-olds behave during heat exposure. Evidence-based research is needed to inform heat-related community-level guidelines, practices and policies for older adults.

10 Literature Review

10.1 Introduction

Heat-related morbidity and mortality in adults aged ≥65 years is a public health issue that requires investigation. Understanding the factors that increase the risk of heat illness in older adults is important to understand what type of intervention is required to improve the health of older adults during heat wave events. This literature review will examine the current knowledge of human thermoregulation with a particular focus on adults aged ≥65 years. Heat illness, and behavioural and autonomic thermoregulation will be discussed, leading to the identification of areas that require more research.

10.1.1 Heat Illness

Heat illnesses vary in severity from minor ailments, such as heat rash, that can be treated at home, to severe, life-threatening heat stroke (Centers for Disease Control and Prevention, 2017). Heat stroke can be either exertional or passive in origin. Exertional heat stroke occurs when metabolic heat production is high, often through exercise, athletic training or occupational activities, and when motivation to complete a task overrides behavioural responses (Corbett et al., 2018; Coris et al., 2004; Lucas et al., 2014; Shafie et al., 2007). Passive heat stroke occurs because of passive heat exposure without a high metabolic heat production. Passive heat stroke often affects unhealthy and vulnerable populations, such as adults aged ≥65 years, who do not have the physiological or behavioural capacity to regulate core temperature at a safe level (Dematte et al., 1998). There are variations within the literature on the core temperature threshold that should be used to diagnose heat stroke. However most experts agree on either >40.0°C (Armstrong et al., 2007; Casa et al., 2012) or >40.6°C (Deng et al., 2018; Shahid et al., 1999). Research on passive heat stroke often utilises the 40.6°C definition (Deng et al., 2018; Shahid et al., 1999), whereas exertional studies have primarily used 40.0°C (Armstrong et al., 2007; Casa et al., 2012). Differences in core temperature definition could relate to variation in core temperature measuring techniques. Rectal and oesophageal temperature differ by ~0.1°C after 10 min of rest in the heat (30°C), and after 10 min of cycling at ~150 b·min^{-1} rectal temperature was ~0.4°C cooler that oesophageal (Teunissen et al., 2012). An elevated core temperature is coupled with nervous system disturbances and multiple organ failure in both exertional and passive heat stroke (Armstrong et al., 2007; Casa et al., 2012; Deng et al., 2018; Shahid et al., 1999).

10.2 Heat Exchange in Humans

Maintaining a stable core temperature is vital for homeostasis and proper physiological functioning. At rest, homeostasis typically regulates the core temperature to ~37.0 ± 1.0°C (McArdle et al., 2015). A fall in core temperature results in hypothermia, often defined as a core temperature <35.0°C (Gentilello, 1995), while increases in core temperature result in hyperthermia, although there is no universally accepted temperature definition (Axelrod & Diringer, 2008). The hypothalamus controls thermoregulatory responses in the body. When heat production and heat loss are balanced core temperature will remain stable. Where heat production is greater than heat loss core temperature will increase, and vice versa. Temperature regulation can be formulated as follows (Bligh & Johnson, 1973):

Equation 10-1. *Heat balance equation* (Bligh & Johnson, 1973)

$$S = M \pm E - (\pm W) \pm R \pm C \pm K \quad (\text{W or W}\cdot\text{m}^{-2})$$

Where:

S = Rate of storage of body heat
M = Metabolic heat production
E = Evaporative heat transfer
W = Mechanical work
R = Radiant heat exchange
C = Convective heat transfer
K = Conductive heat transfer

Temperature sensitive receptors (thermoreceptors) detect temperature change and are located centrally in the brain stem, spinal cord, and preoptic anterior hypothalamus, and peripherally in the skin (Romanovsky, 2007). Transient receptor potential (TRP) channels transmit information on temperature sensation from around the body, including the skin, to the hypothalamus (Caterina, 2007). TRP vanilloid (TRPV) channels are of particular interest in warmth sensation; TRPV 3 is activated at temperatures >33°C, and TRPV 4 activated at ~27-42°C (Patapoutian et al., 2003). Afferent signals are co-ordinated by the hypothalamus. If core temperature deviations are detected, the hypothalamus initiates mechanisms designed to return core temperature to a resting level.

To understand the issues affecting thermoregulation and ageing it is important to identify how thermoregulation functions in a healthy young adult. The human musculo-skeletal system is ~20% efficient at generating movement energy when cycling (Smith et al., 2005), the rest of the energy is

released as heat. This relationship is important for behavioural thermoregulation as adjusting exercise intensity alters how much metabolic heat is produced, and in turn, how much heat must be dissipated. The body has several mechanisms to lose heat. Heat can be dissipated through dry (convection, conduction, radiation), and wet (evaporation) heat exchange (Armstrong, 2000; McArdle et al., 2015). Unlike many homeostatic response systems, temperature regulation through skin blood flow and sweating is a proportional response, and greater increases in core temperature result in greater increases in sweat rate (Benzinger, 1969), until a plateau is reached (Choi et al., 2019).

Conduction is the direct transfer of heat energy between two solid objects. Heat is transferred from the warmer to the cooler object and can be lost or gained by the body through conduction. The degree of heat transferred depends on the temperature difference between the skin and surface in contact with the skin, and the thermal properties of the surfaces (McArdle et al., 2015). Conduction typically plays a trivial role in heat exchange in most activities as the body is only in direct contact with a small area - for example feet on the floor when walking (Kenny & Jay, 2013). However, conduction plays a more important role in those individuals who are bed-bound or seated for much of the day. The large area of skin in direct contact with the bed or chair will prevent convective cooling as air cannot circulate around the body. This restriction will also prevent sweat evaporation, thus hindering evaporative cooling. However, if the bed is cooler than skin temperature conductive cooling occurs, thus water-perfused cooling systems offer an effective strategy for reducing heat strain in bed-bound patients.

Convection is the transfer of heat energy from a solid (e.g. skin) to a fluid (e.g. air or water) and relies on the movement of the fluid (Armstrong, 2000). If air movement around the skin is slow then an individual will have a layer of insulation, and heat energy will transfer slowly. If air movement around the skin is faster, cooler air is replacing the insulation layer and heat will be continually transferred away from the body. The rate of heat loss through convection also depends on skin blood flow (Charkoudian, 2016). Heat from the core is transferred to the skin via convection within the blood. Vasomotor control determines the level of dilation and constriction of blood vessels in the skin, thus allowing more, or less blood flow and consequently heat, respectively to reach the skin and dissipate into the ambient environment. If this system is impaired, as is seen with ageing (Brandes et al., 2005; Lakatta & Levy, 2003), then convection is less effective as the volume of blood redistributed to the skin is reduced. In healthy ageing (aged >55 years) skin blood flow is reduced by 25-50% during whole body heating (Holowatz et al., 2010). While healthy aging results in reduced skin blood flow, older men (62 ± 7 years) with heart failure have a 34% lower cutaneous vascular conductance during cycling exercise (4 $W \cdot kg^{-1}$ of heat production) in the heat (~30°C, ~25% RH) than healthy age matched (61 ±

7 years) peers (Balmain et al., 2018). This effect results in an ~40% greater increase in rectal temperature for the heart failure participants compared with the healthy men (Balmain et al., 2018). Thus, convection and skin blood flow are important mechanisms for heat dissipation with both these elements impaired by ageing and disease. If the fluid around the body is warmer than the skin, then heat can be gained by the body. Thus, a fan in a hot room often does not provide much relief as the air moving around the body is warm so the temperature gradient is insufficient to provide a cooling effect. The cooling effect of water is 32 times greater than that of air causing convective heat transfer to occur much more quickly in water than air (Keim et al., 2002). This effect is why 28°C water feels much cooler than 28°C air.

Thermal radiation occurs when objects, including humans, emit electromagnetic waves transferring heat from an area of higher heat to an area of lower heat. When the surrounding objects are warmer than the body heat is gained and vice versa (Sawka et al., 2011). Unlike conduction and convection, radiation does not require direct contact between molecules. The process of radiation is how the sun warms the Earth. In the desert, a partially clothed human can expect to experience 233 W of radiant heating from the sun, this is approximately halved to 116 W when light coloured clothing is worn (Adolph et al., 1947). Minimising the effect of this radiant heat by avoiding direct sunlight offers another way to reduce heat strain during a heat wave event by adapting human behaviour.

Heat loss through evaporation is achieved via sweat and water lost in the respiratory passages due to breathing. Evaporation is the only heat transfer mechanism that can lose heat when ambient temperature exceeds skin temperature (Kondo et al., 1999). As the sweat evaporates from the skin, surface heat is lost to the environment. Around 580 kcal of energy is dissipated with every litre of sweat that evaporates (Keim et al., 2002). If sweat does not evaporate from the skin and drips off the body instead, it provides no cooling power. This situation can become problematic in conditions where sweat does not evaporate, such as when humidity is high, or if heavy protective clothing or uniforms are worn, or those who are bedridden.

10.3 Behavioural Thermoregulation

Unlike autonomic thermoregulation, behavioural adaptations to the ambient conditions have an almost unlimited capacity to regulate body temperature. Behavioural thermoregulation during heat exposure can manifest itself in a variety of ways including seeking shade or air conditioned buildings, increasing fluid intake, removing clothing layers, or taking a cooling shower (Morano et al. 2016), and are undertaken when thermal discomfort is experienced until a state of thermal comfort is achieved (Gagge et al., 1969). Thermal comfort is a “state of mind which expresses satisfaction with the thermal

environment" (The American Society of Heating Refrigerating and Air-Conditioning Engineers, 2013). During exercise in the heat, reducing workload is an effective way to reduce metabolic heat gain and thus, reduce heat stress. Similar to pacing during sporting performance. Investigating the factors influencing thermoregulatory behaviours is important for understanding how interventions can be effective in improving behavioural responses to the ambient environment. Thermal inputs activate several areas of the brain, including those associated with the sensory-discriminatory aspect of pain (temporal and spatial features), the affective-motivation aspect of pain (unpleasantness of the pain), and motor control (Becerra et al., 1999; Flouris, 2011). Skin and core temperature provide thermal input to the thermoregulatory system (Adair, 1974; Tan & Knight, 2018). Skin temperature, rather than core temperature, drives thermoregulatory behaviours (Schlader et al., 2009; Simon et al., 1986; Tucker et al., 2004, 2006).

10.3.1 Behavioural Thermoregulation and Heat Wave Events

Drinking >1 L of water a day during a heatwave significantly reduced the risk of heat related illness and death during the France 2003 heat wave (Vandentorren et al., 2006). *Ad libitum* fluid intake was sufficient to reduce rectal temperature in young adults (25 ± 4 years; 6 male, 6 female) intermittently walking at 3 METs for 3 hours in ~40°C ambient temperature compared to no fluid replacement (Graham et al. 2020). After 120 min of exercise, rectal temperature was significantly cooler than when drinking no water when drinking *ad libitum* and was no different to full fluid replacement. The volume of water consumed (1.30 ± 0.4 L) was also similar to the full fluid replacement trial where participants were prescribed full replacement of fluid lost through sweating (1.41 ± 0.32 L). Thus, young adults are capable of voluntarily drinking enough water to prevent increased rectal temperatures during prolonged walking exercise. Preventing dehydration is an important intervention to reduce heat strain during heat wave events. However, many older adults have a low intake of water and fluids in their daily lives, particularly those in residential care (Godfrey et al., 2012; Keller, 2006). Thus, research is required that examines how effective healthy older adults are at preventing dehydration through *ad libitum* fluid consumption when exposed to hot ambient conditions.

10.3.2 Mediators of Behavioural Thermoregulation

Skin temperature influences thermal comfort, which, in turn, influences behavioural thermoregulation. When the skin temperature of men aged 24 ± 2 years was clamped at 30, 34, or 36°C and core cooling was initiated with intravenous fluids, regression analysis revealed that skin and core temperature contribute equally to thermal comfort. However, core temperature contributes ~2-4 times as much as skin temperature to autonomic responses (vasoconstriction, metabolic heat production, norepinephrine, and epinephrine; Bulcao et al., 2000). Having skin temperature

contribute to thermal comfort allows the body to pre-empt changes in core temperature and initiate thermoregulatory behaviours before core temperature is affected and maintain homeostasis. Skin temperature drives behavioural thermoregulation by utilising both feedforward and feedback mechanisms: non-hairy, glabrous skin provides feedforward information for thermoregulatory behaviour (e.g. testing water temperature with your hand), while hairy, non-glabrous skin that is often insulated with clothing provides feedback information on the effectiveness of behavioural interventions (Romanovsky, 2014). Understanding how age impacts on the relationship between skin temperature and behavioural thermoregulation could offer important areas for interventions during heat wave events in older adults.

10.3.3 Behavioural thermoregulation in Young Adults

With behavioural strategies, young adults can typically maintain core temperature during a variety of challenges. Schlader et al. (2009, 2013) investigated thermoregulatory behaviour at rest in young (~25 years) healthy men by utilising two environmental chambers, one hot (~45°C) and the other cold (~9°C), with an automated chair that travelled between the chambers when participants pushed a button. Participants were free to move between the two chambers as they wished, by pushing the button to represent their thermoregulatory behaviour. Using this strategy young men maintained a stable core temperature (~37.0°C), despite skin temperature fluctuating between ~29°C and ~34°C (Schlader et al., 2009, 2013). Participants initiated movement between chambers at a similar level of whole body thermal discomfort when moving from hot to cold and vice versa (Schlader et al., 2013). Thus, skin temperature and thermal discomfort are important drivers for the initiation of thermoregulatory behaviour. Implementing effective thermoregulatory behaviours can maintain core temperature homeostasis. These studies only used young men, so the results and their implications may differ in women and older adults aged ≥65 years.

When exercising, metabolic heat load increases. Correctly pacing physical exercise in hot ambient conditions can largely prevent a dangerous rise in thermal and physiological strain on the body. While skin temperature influences behavioural thermoregulation throughout passive heat exposure, it was only at the onset of exercise that skin temperature, thermal sensation, and thermal comfort affected behavioural thermoregulation (i.e. exercise intensity) during a 60 min self-paced cycling time trial in young (30 ± 9 years) men (Schlader et al., 2011b). This study used water-perfused clothing to control skin temperature, with the water increasing from cold (-6.3°C) to hot (61.1°C) in one trial and decreasing from hot (61.3°C) to cold (-4.5°C) in another. The thermal sensation followed the pattern of the water temperature, thermal comfort increased for both trials for the first 20 min, before continuing to increase for the cold-to-hot trial and decreasing for the hot-to-cold trial, and RPE

increased as time increased. The total work completed was ~2% higher in the cold to hot trial than the hot to cold trial (924 v 902 KJ), a difference attributed to a greater power output at the beginning of the exercise in the cold to hot trial (Schlader et al., 2011b). Core temperature was similar between the trials, thus did not influence perceived effort or thermal comfort. Skin temperature did not appear to influence perceived effort as there was no difference in perceived effort between the trials (Schlader et al., 2011b). Thus, thermoregulatory behaviour (i.e. exercise intensity) is selected at the onset of exercise rather than adjusted during exercise. When exercising during the cooler morning hours it is important to be mindful of increasing ambient temperatures later in the day as participants may not realise the need to reduce metabolic heat load and the risk of exertional heat illness. There are also implications for exercising in changing conditions with intermittent shade. If individuals are unable to respond to an increase in sun exposure then metabolic heat gains will increase.

Similar findings have been observed when using a fixed RPE model, where workload is reduced before core temperature reaches a dangerous level. When young men (23 ± 4 years) were asked to cycle at an intensity equivalent to 16 on Borg's RPE scale, power output dropped at a faster rate in 35°C than 25°C or 15°C. Participants dropped to 70% of their initial power output after 34 min in 35°C, 49 min in 25°C, and 50 min in 15°C (Tucker et al., 2006). Rate of heat storage was higher in 35°C only at 1%, 90% and 100% of trial completion, for most of the trial (10-80% completion) rate of heat storage did not differ between the trial conditions (~10-20 $kJ \cdot min^{-1}$). This similar rate of heat production resulted in a rectal temperature that remained comparable between conditions until 90% of the trial was completed, after this the 35°C trial rectal temperature was ~39.0°C while the 15°C and 15°C trials were ~38.5°C (Tucker et al., 2006). Mean skin temperature was ~2°C and ~6°C higher in 35°C than 25°C and 15°C trials respectively, and participants felt warmer in 35°C than in 25°C and 15°C from the start of the exercise (Tucker et al., 2006). It appears the participants implemented thermoregulatory behaviours in an anticipatory fashion, driven by skin temperature and feelings of warmth, rather than core temperature, to reduce heat strain during exercise in the heat.

Another study used a similar protocol to investigate skin temperature and thermal perception, greater perceptions of warmth influence cycling power output regardless of skin temperature. Young (23 ± 1 years) men cycled at a fixed RPE of 16 until power output dropped by 30%; increasing perception of warmth by heating participant's faces via a heater or by capsaicin application resulted in a ~7-10 min quicker decline in power output than when the face was cooled by a fan or menthol application (Schlader et al., 2011a). The capsaicin and menthol had no influence on skin temperature but successfully manipulated thermal comfort in a similar way to the heater and cooling fan, respectively. As with Tucker et al. (2006), core temperature did not differ between trials with Schlader et al. (2011a). It seems that thermal sensation and discomfort influence thermal behaviours regardless of skin

temperature. Schlader et al. (2011a) only recruited young men to their study; men aged 67 years appear to have a different perception of thermal comfort than young adults (Taylor et al., 1995) so further research is required to understand how thermal perceptions and skin temperature affect thermoregulatory behaviours in older adults.

Behavioural thermoregulation during exercise is likely implemented in an anticipatory manner by central control, thus reducing muscular work before hyperthermia is reached. Tucker et al. (2006) measured the electrical stimulations in the *vastus lateralis* using electromyography (EMG) before and after a fixed RPE cycling protocol. Despite reduced EMG activity in the hot (35°C) trial compared to the cool (15°C) trial at 60%, 90% and 100% of trial completion, there was no difference in activity during the final 30 second sprint at the end of the trial (Tucker et al., 2006). It appears that muscular fatigue is not the cause of the reduced workload during self-paced exercise in the heat, as central control reduces muscular activation. Recent work has examined brain activity during heat exposure to determine how heat stress could influence central control of thermoregulation. Young (35 ± 3 years) men experience electroencephalographic (EEG) alterations during passive hyperthermia (gastrointestinal temperature 39.1°C) in hot (50°C, 50% RH) conditions, with elevations in theta wave power increasing cognitive load and impairing ability to complete complex cognitive tasks compared with euthermic core temperature (36.9 °C) in thermoneutral (25°C, 50% RH) conditions (Gaoua et al., 2018). Time to elicit the correct answer during the One Touch Stocking of Cambridge-6 (OTS-6) test took 13 seconds longer under hyperthermic conditions (Gaoua et al., 2018). During passive hyperthermia, the brain is functioning closer to its total capacity, thus cognitive tasks are hindered.

EEG activity has also been monitored during a self-paced 750 kJ cycling time trial in the heat (35°C, 60% RH) in young (34 ± 4 years) men (Périard et al., 2018). The time trial took substantially longer in the heat than in the cool (18°C, 40% RH; [hot v cool] 55 v 48 min) as power output was significantly lower in the heat from 50% of trial completion onwards. Despite reduced power output, rectal temperature was ≥0.5°C higher in the hot trial from 70% completion onwards, and RPE was similar throughout the trials. Alpha and beta brain waves were measured throughout the exercise; in the central region alpha activity was lower in the heat than the cool at 30%, 70%, 90%, and 100% completion, while beta activity was lower from 30% completion onwards. In the frontal region alpha activity tended to be lower, and beta activity was lower from 50% completion onwards in the heat compared with the cool condition (Périard et al., 2018). High alpha activity is associated with focus and ignoring irrelevant stimuli, while beta activity is required for mental readiness, thus heat stress during exercise appears to hinder cognitive attention and readiness. These studies demonstrate altered brain wave activity in young healthy males during heat exposure. Further work is necessary to

establish if this altered activity impacts upon the central control of behavioural thermoregulation, hindering the implementation of cooling strategies during heat exposure.

Reduced cognitive functioning has been recorded in a real world setting as well as in the laboratory. Students aged 20 ± 2 years old living in accommodation with and without air conditioning had their cognitive function measured prior to, and throughout a heat wave (Cedeño Laurent et al., 2018). The air-conditioned accommodation had a mean indoor temperature of 21.4 ± 1.9°C, the non-air-conditioned accommodation had a significantly higher mean indoor temperature of 26.3 ± 2.5°C. Those without air conditioning performed worse on the Stroop colour word test (assessing cognitive speed and inhibitory control) and an addition subtraction test (assessing cognitive speed and working memory) than those with air conditioning, even when results were corrected for the learning effect by using a difference-in-difference analysis (Cedeño Laurent et al., 2018). This study demonstrates the negative impact heat waves have on cognitive function if indoor conditions are not cooled sufficiently. A reduced cognitive functioning during heat waves could reduce the ability to make decisions readily and easily about implementing cooling behaviours.

During heat waves people are exposed to high ambient heat for longer periods than most laboratory-based studies. However, research investigating how workers cope in high ambient conditions has evaluated the effect of much longer exposure times. Acclimatised male mine workers (aged 35 ± 8 years) exposed to hot conditions (wet bulb globe temperature 31°C) for 7.5 to 9 hours had a mean high core temperature of 38.3°C, and the mean increase in core temperature over a shift was 1.4°C (Brake & Bates, 2002). A control group conducted office-based work in an air-conditioned building (24°C, 50% RH) had a mean high core temperature of 37.6°C, and the mean increase in core temperature over the office workers shift was 0.9°C (Brake & Bates, 2002). A large portion of the increase in core temperature for the mine workers over a shift was related to the natural circadian rhythm. The mine workers were able to self-pace their work to prevent a catastrophic rise in core temperature. The participants in this study were all acclimatised to their working conditions and the physically demanding nature of the work, and well-educated on the risks of working in the heat. Workers who are unfamiliar with their work and relatively uneducated about the risks of working in the heat are also able to regulate their work load and prevent dangerous increases in core temperature (Miller et al., 2011). Construction workers (aged 20 to 60 years) working in the UAE (summer temperatures ~32-42 °C, highs of up to 50 °C (World Bank Group, 2021c)) originally from South Asia (India, Pakistan, and Bangladesh (summer temperatures ~25-35 °C (World Bank Group, 2021a, 2021b)) were able to maintain a mean working heart rate <100 $b \cdot min^{-1}$ when exposed to a thermal work limit of 140-200 $W \cdot m^{-2}$ (Miller et al., 2011). Aural temperature at the start of the work shifts was ~36.9°C, at the end of the shift it was ~37.1°C (Miller et al., 2011). The construction workers

were able to self-pace their workload to prevent a dangerous rise in aural temperature and heart rate. Thermoregulatory behaviour during exercise appears to be an innate trait that can be implemented in young healthy adults without any training on working in the heat. Although this study includes a wide range of ages, it did not specifically investigate how age effects thermoregulatory behaviour, thus further research is required to elucidate how age impacts thermoregulatory behaviours during heat exposure.

10.3.4 Behavioural Thermoregulation in Older Adults

Individuals need to be able to perceive changes in skin temperature and thermal comfort as thermoregulatory behaviours are only implemented when thermal discomfort is experienced. Thermal discomfort will alter as skin temperature increases or decreases. Work in men aged 67 ± 4 years shows that they are less able to detect ambient temperature changes than young men (Taylor et al., 1995). When given a dual position switch that either warmed or cooled a room and instructed to maintain a comfortable temperature, older men (67 ± 4 years) let the ambient temperature fluctuate more than young (23 ± 3 years) men ([older v young] 32.7°C to 15.9 v 32.7°C to 16.9°C), although the mean temperature was similar between the groups ([older v young] 24.5 v 24.9°C; Taylor et al., 1995). The older men felt more thermally comfortable when they initiated warming than the young men, despite doing so at a cooler temperature ([older v young] 15.9 v 16.9°C; Taylor et al., 1995). Thus, men aged ~67 years require a greater thermal stimulus to feel uncomfortable and have a reduced ability to identify when thermoregulatory behaviours should be implemented. However, the older group had a ~2°C lower mean skin temperature than the younger group throughout the trial. Yet, both age groups implemented cooling at the same ambient temperature (32.7°C), this suggests that older men will implement cooling behaviours at lower skin temperatures than young men thus reducing their heat illness risk, challenging our hypothesis that older adults require a warmer skin temperature to implement cooling behaviours. Further investigation is required to understand why the older men are less able to maintain a stable ambient temperature while experiencing a cooler skin temperature.

Proactive thermoregulatory interventions prior to a marked increase in core temperature would be of great benefit to prevent an increase in heat load before it becomes problematic. Pacing studies show that during exercise performance there is an anticipatory reduction in workload before core temperature increases (Schlader et al., 2011a; Tucker et al., 2004, 2006). Young (25 ± 5 years) men performing a 20 km cycling time trial in the heat (35°C, 60% RH) took 2.7% longer than in the cool (15°C, 60% RH) with power output being significantly lower in the hot trial for the final 20% of the trial (Tucker et al., 2004). Rectal temperature only differed at the end of the time trial, whereas skin

temperature was warmer throughout the hot trial (Tucker et al., 2004). This indicates that during time trial exercise, young men are able to initially voluntarily ignore information from thermoreceptors in the skin when motivated to complete exercise as quickly as possible. Nevertheless, eventually exercise intensity will reduce before core temperature increases to reduce the level of heat strain experienced. Despite the reduction in power output and increased time to complete the trial, rating of perceived exertion (RPE) did not differ at any time between the conditions (Tucker et al., 2004). These results demonstrate the potential for a central anticipatory control of behavioural thermoregulation. Further work is needed to identify how ageing impacts this anticipatory control. This work used stable conditions, the effect of changing ambient conditions on thermoregulatory behaviours cannot be elucidated from this study.

Heat exposure has a detrimental effect on cognitive function in young adults. With healthy ageing there is a decline in cognition and perception (Roberts & Allen, 2016), and overall EEG power, particularly in the alpha wave range (Vysata et al. 2012). However, work has demonstrated that older adults (73 ± 7 years) perform equally well in selected tests from the Cambridge neurophsychological test automated battery in 24°C and 32°C (Trezza et al., 2015). While overall there was no significant difference in congnitive function between the two environments, Trezza et al. (2015) identified discrete risk factors for reduced cognitive function in the heat. Participants who performed at least four bouts of exercise per week performed better than those who did not, and increased relative humidity resulted in reduced cognitive performance in the heat. This study used a relatively mild heat stress of 32°C to replicate a usual urban summer day, whereas others have used additional metabolic heat stress (Périard et al., 2018) or very high ambient temperatures of 50°C (Gaoua et al., 2018), thus greater levels of heat strain may result in greater cognitive deficits. As cognition is hindered with ageing, it is important to understand how hyperthermia and heat stress impacts cognitive function in older adults, and their ability to implement thermoregulatory behaviours.

As thermal comfort appears to drive the self-selection of exercise intensity (Schlader et al., 2011a), studies have investigated how older adults (≥65 years) perceive their thermal comfort during exercise in the heat (Tsuzuki & Iwata, 2002; Waldock et al., 2018). During fixed intensity stepping exercise (15 cm step, 16 steps a min) in 23°C and 27°C, the older group (69 ± 4 years) perceived the 27°C trial to be warmer than the 23°C trial (Tsuzuki & Iwata, 2002). This outcome indicates that thermal perception still functions adequately in adults aged 69 years, however, there was no young control group to compare against. We do not know how young adults would have responded to these conditions. More recently, using two independent groups each with 9 participants, Waldock et al. (2018) demonstrated that when cycling at 6 METs for 30 minutes, older adults (men and women, ~70 years) did not report 35°C to be more thermally uncomfortable than 25°C, despite concomitant rises in skin (~3.1°C higher

in 35°C trial) and core (~0.1°C higher in 35°C trial) temperature. Six METs is the lower boundary for vigorous activity (U.S. Department of Health and Human Services, 2008). If the ≥65-year-olds have a diminished perception of discomfort during their daily lives, they may be reluctant to adapt their behaviour(s) to reduce the thermal challenge independent of, and in addition to, impaired autonomic thermoregulation. As these studies ((Tsuzuki & Iwata, 2002; Waldock et al., 2018) were assessing thermal perceptions they used a fixed-intensity approach, this allowed for thermal perceptions to alter but not exercise intensity. Using a fixed RPE approach would allow participants to implement thermoregulatory behaviours and elucidate if the lack of sensitivity to high ambient temperatures prevents them from initiating thermoregulatory behaviours. As behavioural thermoregulation can prevent the onset of heat illness, understanding the thermal perception and behavioural response of the over 65s is important as they have been identified as an at-risk population during heat wave events. Allowing older participants the opportunity to adapt their behaviour during heat exposure will help uncover how ageing impacts thermoregulatory behaviours.

Behavioural thermoregulation offers substantial opportunity to reduce heat strain. However, behavioural thermoregulatory strategies are only implemented if individuals can detect and react to thermal discomfort. In young adults, at rest and during exercise, this process is robust, however in adults aged ≥65 years, there appears to be a reduced ability to detect thermal stress and discomfort. The current understanding is summarised in Figure 10-1. Flow diagram of behavioural thermoregulation pathway. Currently, there is little research investigating how age impacts thermoregulatory behaviours when participants are free to initiate thermoregulatory behaviours. Thus, research is required to understand how older adults, aged 65 years and over, behave during

heat exposures given that modifying thermoregulatory behaviour offers a simple and effective means of reducing heat strain, and potentially heat-related morbidity and mortality.

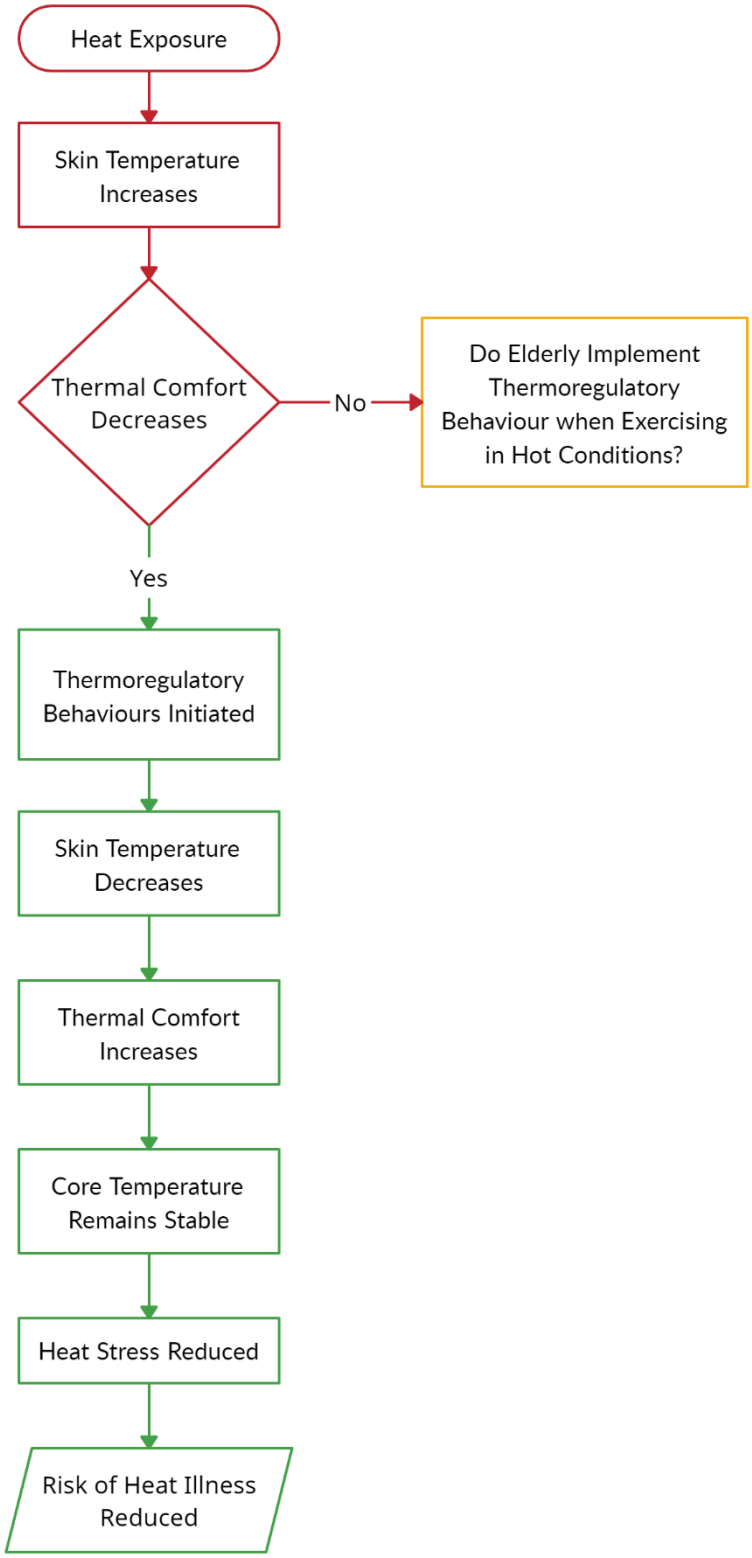

Figure 10-1. *Flow diagram of behavioural thermoregulation pathway*

10.4 Autonomic Thermoregulation

10.4.1 Skin blood flow

Dry heat exchange relies on increasing skin temperature via elevations in skin blood flow. In thermoneutral (ambient temperature ~22-24 °C) and mild heat stress conditions (ambient temperature ~30 °C), skin blood flow alterations alone can maintain a stable core temperature (Stapleton, 2015). Increasing blood flow to the skin allows heat to transfer from the core to the skin, and on to the external environment via convective heat exchange (Wong & Hollowed, 2017). This increase in skin blood flow relies upon the cardiovascular system, an increase in cardiac output, redistribution of blood towards the skin, and a reduction in blood flow to the splanchnic and renal systems (Charkoudian, 2003; Rowell, 1974).

Skin blood flow is reliant upon the cardiovascular system being able to adapt and increase cardiac output and redistributing blood from the internal organs to the skin during heat stress (Charkoudian, 2003; Johnson & Proppe, 1996). If the cardiovascular system is not capable of increasing cardiac output, or redirecting blood to the skin, then the amount of heat dissipated from the skin will be compromised. A healthy cardiovascular system is vital for maximising heat loss capabilities during heat stress.

10.4.1.1 Control of Skin Blood Flow

The cardiovascular responses to thermal stress are co-ordinated in the brain. Blood vessels in nonglabrous skin are controlled by two sets of sympathetic nerves: noradrenergic nerves for vasoconstriction and cholinergic nerves for vasodilation (Charkoudian, 2003). This process is controlled in the preoptic anterior hypothalamus (Charkoudian, 2003; Levy & Pappano, 2007). At rest in a thermoneutral environment, skin blood flow is around 30-40 $mL \cdot min^{-1} \cdot 100\ g^{-1}$of skin (Johnson, Minson, & Kellogg, 2014). In these conditions skin blood flow is controlled via alterations in vasoconstriction (McCord et al., 2006) which in turn is controlled by sympathetic noradrenergic nerves releasing noradrenaline to act upon α_1 and α_2-receptors (Johnson et al., 2014; McCord et al., 2006; Wong & Hollowed, 2017). A reduction in vasoconstriction via this system causes the initial rise in skin blood flow (Kellogg, 2006; Wong & Hollowed, 2017), and accounts for 5-15% of the increase in skin blood flow seen during heat stress (McCord et al., 2006). Vasomotor control is autonomically controlled to co-ordinate a thermoregulatory response to protect homeostasis – this process is vital for dry heat loss from the body.

If heat strain continues to increase after vasoconstriction has been removed, active vasodilation takes place to increase dry heat loss. Vasodilation initiated by sympathetic cholinergic vasodilator nerves

accounts for 85-95% of skin blood flow increases (McCord et al., 2006), resulting in a skin blood flow of up to 8 $L \cdot min^{-1}$ (Johnson et al., 2014; Johnson & Proppe, 1996) that facilitates ~1,500 $kcal \cdot h^{-1}$ of heat dissipation (Johnson, Brengelmann, Hales, Vanhoutte, & Wenger, 1986). The mechanisms controlling active vasodilation are unclear despite substantial research in the area (Wong & Hollowed, 2017). The sympathetic cholinergic nerves that control vasodilation appear to release acetylcholine alongside at least one other co-transmitter (McCord et al., 2006; Wong et al., 2004; Wong & Hollowed, 2017).

Much work has investigated the role of nitric oxide neurotransmitters in vasodilation (Fujii et al., 2014; McCord et al., 2006; Minson et al., 2001). Early work using the rabbit ear determined that nitric oxide and another unknown neurotransmitter are required for active vasodilation (Farrell & Bishop, 1995; Taylor & Bishop, 1993). Nitric oxide is thought to be a direct effector of vasodilation and contributes ~30-45% of active vasodilation (Kellogg, Zhao, Friel, & Roman, 2003; Wilkins, Holowatz, Wong, & Minson, 2003; Wong & Hollowed, 2017). With whole-body heating of young adults (~23 years), blockade of nitric oxide or cyclooxygenase inhibits cutaneous vascular conductance, simultaneous blockade of both nitric oxide and cyclooxygenase yields an even greater inhibition of cutaneous vascular conductance (McCord et al., 2006). This result provides evidence for the role of nitric oxide in vasodilation, and shows that the cyclooxygenase pathway is involved in active vasodilation during heat stress (McCord et al., 2006). It seems clear that nitric oxide and the cyclooxygenase pathway are involved in active vasodilation at rest. However, during recumbent cycling in a hot environment (35°C, 20% RH) at a moderate heat load of 400 W of metabolic heat production, cyclooxygenase inhibition had no effect on forearm skin blood flow in young (~24 years) men. In contrast, nitric oxide inhibition reduced skin blood flow during moderate heat loads of 400 W metabolic heat production (Fujii et al., 2014). When metabolic heat load was higher (700 W), inhibiting nitric oxide and/or cyclooxygenase had no impact on forearm skin blood flow (Fujii et al., 2014). The mechanisms controlling active vasodilation vary under different heat stress situations, and there is more than one pathway involved in active vasodilation. The active vasodilation response during a heat wave could have a varied response due to the length of exposure, more research is required to investigate how a prolonged multi-day heat exposure impacts skin blood flow. Given that the mechanisms that control active vasodilation are not fully understood, and accordingly the process of ageing on this system is also unclear. It may be that with ageing there is a diminished response in this pathway, either a reduction in nitric oxide and other neurotransmitters, or a reduced sensitivity to them.

10.4.1.2 Effect of Age on Nitric Oxide and Skin Blood Flow

Ageing reduces vasodilation during heat stress, with both the axon-mediated initial peak and nitric oxide-mediated plateau in cutaneous vascular conductance hindered with ageing (Minson et al.,

2002). This study included both a young (18-24 years) and an older (69-84 years) group allowing for direct comparison between the ages. The inhibition of skin blood flow was measured as a percentage of maximal, thus the reduced skin blood flow response in the older group was not simply due to a reduced absolute maximal skin blood flow. After infusion of N^G-nitro-L-arginine methyl ester (L-NAME; a nitric oxide synthase inhibitor) the percentage of maximal cutaneous vascular conductance was similar between the groups (Minson et al., 2002). The older group were less reliant on nitric oxide-dependent vasodilation. Nitric oxide-dependent vasodilation is inhibited with ageing.

A decline in cutaneous vascular conductance could relate to a reduced availability of nitric oxide; the availability of L-arginine, a nitric oxide precursor, and nitrite and nitrate, metabolites of nitric oxide, are all diminished with ageing (Reckelhoff et al., 1994). However, work from the same research group indicates the decline in skin blood flow with age is due to an unknown active vasodilator (Holowatz et al., 2003). Participants wore a liquid conditioning garment with 50°C water perfused around the suit until core temperature increased by 1°C. Inhibition of nitric oxide synthesis had little effect upon cutaneous vascular conductance in older (71 ± 6 years) adults, but young (23 ± 2 years) adults experienced a marked decrease in cutaneous vascular conductance (Holowatz et al., 2003). The older group had a substantially lower resting core temperature than the young group (oral temperature; 36.2 v 36.5°C); so, a 1.0°C increase from baseline resulted in different final core temperatures and evidence of a delayed vasodilation response in the older group. However, increases in skin blood flow began at similar absolute core temperature thresholds (control: older 36.6°C, young 36.5°C; nitric oxide inhibited: older 36.8°C, young 36.7°C; Holowatz et al., 2003). The relatively low core temperatures used in this protocol does not permit analysis of the physiological consequences when older adults are exposed to a heat challenge, their response could be similar to that of a young group with a greater thermal input from core temperature. Holowatz et al. (2003) used oral temperature as an indicator of core temperature, thus the core temperatures reported are probably slightly low as oral temperature is known to be ~0.4°C lower than intra-pulmonary arterial temperature (Ilsley et al., 1983). Adding 0.4°C to the temperatures reported by Holowatz et al. (2003) still only gives a core temperature of 36.9-37.2°C.

10.4.1.3 Skin Blood Flow During Passive Heat Exposure

During heat exposure, all adults experience an increase in strain on the cardiovascular system. Prior to three hours of passive heat exposure (43°C, 30% RH), young (24 ± 3 years) adults had a higher cardiac output than older (64 ± 6 years) adults (6.0 ± 1.1 v 3.8 ± 1.1 $L \cdot min^{-1}$) caused by a greater stroke volume in younger adults (82 ± 19 v 57 ± 18 mL). After the heat exposure stroke volume remained higher in the younger groups and dropped in both groups to 59 ± 12 mL and 39 ± 12 mL for the younger

and older groups, respectively. Heart rate did not differ between the groups. The reduced stroke volume in the older adults contributed to an impaired ability to dissipate heat and a 1.9-fold greater increase in heat storage for the older adults (193 ± 133 v 372 ± 191 kJ; Kenny et al., 2015). The reduced baseline capacity in the cardiovascular system of older adults reduces the reserves available for responding to a thermoregulatory challenge.

Young (23 ± 1 years) men increase their cardiac output during passive heating from ~6 $L \cdot m^{-1}$ to ~11 $L \cdot m^{-1}$ by increasing stroke volume and heart rate, however, older men (70 ± 3 years) increased their cardiac output from ~6 $L \cdot m^{-1}$ to only ~7 $L \cdot m^{-1}$ (Minson et al., 1998). This reduction in cardiac output response coincides with a reduction in maximal heart rate and an increase in arterial stiffness with age. Men aged 65 years have a maximal heart rate 29 $b \cdot min^{-1}$ lower than men aged 25 years due to a 25 $b \cdot min^{-1}$ reduction in intrinsic heart rate, and a 39% reduction in sensitivity to β-adrenergic stimulation (Christou & Seals, 2008). At rest, the men aged 65 ± 5 years are functioning approximately 30 $b \cdot min^{-1}$ closer to their heart rate maximum (Christou & Seals, 2008), thus have less reserve to utilise when experiencing physiological and thermal stress. Aortic pulse wave velocity (indicator of aortic stiffness) increases with age, particularly after 50 years, and is ~2-fold greater by 80 years than at 20 years (McEniery et al., 2005). Thus, the older adults (≥65 years) cannot increase cardiac output or redistribute blood flow as efficiently as young adults, resulting in a greater level of cardiovascular stress that contributes to the increased mortality and morbidity rate seen during a heat wave (Vandentorren et al., 2006). This reduced cardiovascular function makes reducing heat strain via thermoregulatory behaviours more important as it will reduce cardiovascular stress and reduce risk of a cardiac event requiring medical treatment.

In healthy older adults skin blood flow is reduced during whole body heating by 25-50% (Holowatz et al., 2010), this is partly due to reduced influence of the sympathetic nervous system on skin blood flow during heat stress. Men aged ≥65 years have a reduced sensitivity sympathetic nerve activity in the skin to heating (Grassi et al., 2003; Tew et al., 2011a; Tew et al., 2011b). During a 45-minute rise in ambient temperature from 23°C to 31°C, the skin sympathetic nerve activity response followed the same pattern in both older men (72 ± 2 years) and young men (24 ± 2 years). At rest, the magnitude of the response was 61% lower in the older group, despite there being no difference in skin temperature response between the groups (Grassi et al., 2003). However, when exposed to music for an arousal stimulus, there was no difference in skin sympathetic nerve activity response between the groups (Grassi et al., 2003). This outcome indicates that skin sympathetic nerve activity is impaired during thermal stimuli but unaffected by arousal stimuli in older men.

10.4.1.4 Skin Blood Flow During Active Heat Exposure

Maintaining a high level of aerobic fitness protects against the decline in sympathetic control of skin blood flow. Tew et al. (2011a) and Tew et al., (2011b) investigated the role of physical fitness in protecting skin sympathetic nerve functioning in ageing men. When local heating was applied to the skin, the initial rise in skin blood flow was reduced by 19-29% in older untrained men (64 ± 1 years, $\dot{V}O_2$max 28 ± 2 mL·kg^{-1}·min^{-1}) compared to young trained men (24 ± 1 years, $\dot{V}O_2$max 58 ± 3 mL·kg^{-1}·min^{-1}), young untrained men (25 ± 1 years, $\dot{V}O_2$max 40 ± 2 mL·kg^{-1}·min^{-1}), and older trained men (64 ± 1 years, $\dot{V}O_2$max 44 ± 2 mL·kg^{-1}·min^{-1}; Tew et al., 2011a). With the application of an anaesthetic cream to the skin, the initial peak in skin blood flow was reduced for every group and the difference with the older untrained group was negated. The reduced skin blood flow under control conditions for older untrained group is, at least in part, due to a reduced functioning of skin sensory nerves (Tew et al., 2011a). As the older trained group displayed a similar response to local heating as the young trained and young untrained groups, it appears that maintaining a high $\dot{V}O_2$max with increasing age protects skin sensory nerve function.

A similar study by Tew et al. (2011b), using the same participants as Tew et al. (2011a), found that ageing reduces the contribution of the noradrenergic sympathetic system to skin blood flow increases during local heating, and that maintaining a high $\dot{V}O_2$max reduced this decline. The older untrained group did not respond to sympathetic nerve block whereas this caused a reduced skin blood flow response in the older trained and both young groups (Tew et al., 2011b), maintaining aerobic fitness into older age sustains the sympathetic nervous response to local skin heating.

As cardiovascular functioning is important to dry heat exchange, the influence of fitness and ageing on thermoregulatory responses has been investigated. When cycling for 20 min at 65% of $\dot{V}O_2$max in the heat (30°C; 40-50% RH), young men (29 ± 1 years, $\dot{V}O_2$max 44.0 mL·kg^{-1}·min^{-1}) had ~2-fold greater forearm blood flow than older men (66 ± 1 years, $\dot{V}O_2$max 32.9 mL·kg^{-1}·min^{-1}; Tankersley, Smolander, Kenney, & Fortney, 1991). These two groups were considered of average fitness as their $\dot{V}O_2$max values were within one standard deviation of their age-adjusted predicted value. This outcome illustrates the decline in skin blood flow with ageing. Cardiac output, splanchnic and renal blood flows were not assessed in this study, so it is unclear whether the source of the reduction in skin blood flow is a reduced cardiac output, or a reduced ability to redistribute blood flow from the internal organs. An older highly fit group, approximately two standard deviations above age predicted $\dot{V}O_2$max, was also included in this study (64 ± 2 years, $\dot{V}O_2$max 46.4 mL·kg^{-1}·min^{-1}; Tankersley et al., 1991). This group had a similar forearm blood flow to the young group. Maintaining a high level of fitness into older age reduces the decline in skin blood flow and thus dry heat exchange.

However, this older fit group undertook several hours of exercise training per day and competed in senior running events. This level of activity does not represent the general population as only 4.1% of adults aged 40-79 years in the UK achieve ≥30 min·day^{-1} of movement activity ≥2,020 counts·min^{-1} in bouts of ≥10 min (Berkemeyer et al., 2016). It is important to understand how older people can maintain their cardiovascular and thermoregulatory functioning without undertaking a substantial level of physical activity. It may be that autonomic thermoregulation cannot be adjusted sufficiently without these high levels of activity. If this is the case, alternative interventions are needed to reduce the heat stress in older adults who are unable to maintain high levels of physical activity and fitness.

Research has studied age, training, and fitness levels to investigate how these factors influence skin blood flow in an older population. When cycling at 60% $\dot{V}O_2$max in a hot dry environment (36°C, 20% RH), the volume of blood redistributed from the splanchnic and renal systems was influenced by age and fitness. Physically fit ($\dot{V}O_2$max 41.8 mL·kg^{-1}·min^{-1}) older men (64 ± 2 years) could not redistribute blood flow to the skin as effectively as sedentary young men (26 ± 2 years) with a similar level of aerobic fitness ($\dot{V}O_2$max 42.7 mL·kg^{-1}·min^{-1}; Ho et al., 1997). Therefore, age, rather than aerobic capacity, has a greater influence over blood redistribution from the splanchnic and renal systems to the skin. Sedentary older men (65 ± 1 years, $\dot{V}O_2$max 28.0 mL·kg^{-1}·min^{-1}) can increase their forearm blood flow by increasing their cardiac output after a 4-week intense training programme (1 hour/day, 5 days a week), but this has no effect on splanchnic and renal vasoconstriction (Ho et al., 1997). With age, aerobic capacity declines, healthy men in their 20s have a $\dot{V}O_2$peak of 47.2 ± 7.9 mL·kg^{-1}·min^{-1}, whereas healthy men in their 60s have a reduced $\dot{V}O_2$peak at 35.7 ± 5.3 mL·kg^{-1}·min^{-1} (Sanada et al., 2007). The fit older men used by Ho et al. (1997) were aerobically fit for their age. Although maintaining a high level of aerobic fitness cannot fully negate the decline in skin blood flow function seen with increasing age, it does ameliorate the decline through improved cardiovascular functioning. These adaptations promote better blood flow redistribution and, where safe, increasing physical fitness and activity should be encouraged to reduce heat strain and illness during heat wave events. Ho et al. (1997) only recruited six participants per group, so these findings warrant further investigation. There is also a lack of evidence of the effect of fitness and blood flow redistribution in older women, so further work is needed to investigate if women have a different response to men.

A cross-sectional study confirmed that maintaining high fitness levels into the seventh decade prevents the decline in cardiovascular thermoregulatory functioning (Best et al., 2012). During an hour of cycling at 70% of $\dot{V}O_2$max in hot conditions (35°C, 40% RH) highly-trained older (56 ± 5 years, $\dot{V}O_2$max 59.3 mL·kg^{-1}·min^{-1}), moderately-trained younger (27 ± 5 years, $\dot{V}O_2$max 55.9 mL·kg^{-1}·min^{-1}), and highly-trained younger (26 years ± 4, $\dot{V}O_2$max 68.1 mL·kg^{-1}·min^{-1}) all had a similar cutaneous

vascular conductance response of ~1-2 units (Best et al., 2012). It appears that maintaining a high level of aerobic fitness prevents a decline in cutaneous vascular conductance into at least the sixth decade. The addition of older moderately or untrained group may show an influence of fitness on cutaneous vascular conductance with age. The highly trained groups undertook at least five 1-2 hour cycling training sessions a week for at least the last 2 years. The older group in this study were relatively young as the eldest was only 63 years, thus it is unclear if the protective mechanism of maintaining a high $\dot{V}O_2$max persists into older age above 63 years. This may explain the difference in findings between Best et al. (2012) and Ho et al (1997), the decline in blood flow redistribution and cutaneous vascular conductance may not occur until the eighth decade. The greater level of aerobic fitness in the Best et al. (2012) participants may also have provided an even greater level of protection from decline in functioning. As heat related morbidity and mortality increases in the over 65s (Centers for Disease Control and Prevention, 2016), further work is required to investigate the impact of aerobic fitness on skin blood flow in this age group.

Best et al. (2012) focussed on a highly-trained older group as it has been argued that from an evolutionary perspective, humans are meant to maintain a high level of activity and fitness (Booth et al., 2002). While this may be the case, rapid industrialisation has made life less physically demanding, thus increasing the prevalence of sedentary lifestyles and reducing overall activity levels (Erikssen, 2001). Focussing on highly trained older adults will not give a reliable indicator of ageing human physiology in the 21st century as maintaining a high level of fitness does not reflect 'normal' ageing (Chad et al., 2005; Ramires et al., 2017). In summary, it appears the findings from Best et al. (2012) may not be directly applicable to most older adults as this high level of aerobic fitness is not representative. Further work is needed to examine how increases in age and declines in physical fitness impact skin blood flow and blood redistribution during heat exposure.

Ageing reduces skin blood flow and therefore the capacity to dissipate heat via dry heat exchange (Best et al., 2012; Ho et al., 1997; Holowatz et al., 2003; Minson et al., 2002; Tankersley et al., 1991). While maintaining and improving fitness in older age can ameliorate the decline in cardiovascular functioning and dry heat exchange, the levels of exercise required to achieve these effects are impractical for many individuals due to age-related functional losses. Other more practical interventions are required to reduce the physiological and thermoregulatory strain experienced by many during heat wave events. If improving autonomic thermoregulatory functioning proves difficult and impractical, thermoregulatory behaviours and interventions are likely the most useful avenues to reducing heat-related morbidity and mortality.

10.4.2 Sweating

Sweating is a critical mechanism for heat loss in humans, particularly when the ambient temperature is above skin temperature as dry heat exchange results in heat gain in these situations (Gagnon & Crandall, 2018). Apocrine sweat glands do not play a role in thermoregulatory sweating (Gagnon & Crandall, 2018) and will not be discussed in this review. Eccrine sweat glands cover most of the skin surface and play a vital role in whole body cooling during heat stress (Machado-Moreira et al., 2008; Smith & Havenith, 2012) as one litre of sweat can dissipate ~580 kcal of heat (Keim et al., 2002).

Eccrine sweat glands release sweat on to the skin which then evaporates dissipating heat. The skin is cooled and consequently the blood at the surface of the skin is also cooled. The eccrine sweat glands are comprised of a secretory coil in the dermis layer of skin, where a straight section of re-absorptive duct travels out to the epidermis layer and becomes the intraepidermal sweat duct before reaching the surface of the skin at the sweat pore (Hu et al., 2018). Sweat is a hypotonic saline solution with around 0.2-0.4% NaCl content (McArdle et al., 2015). As with skin blood flow, thermoregulatory sweating is controlled centrally by the preoptic anterior hypothalamus, and regulated via sympathetic cholinergic nerves (Kimura et al., 2007). The neural pathway from the preoptic anterior hypothalamus to the sweat gland is not fully understood (Shibasaki & Crandall, 2010). ***Figure 10-2.*** *Proposed neural pathway from thermoreceptors to sweat glands (Sato et al., 1989; Shibaski & Crandall, 2010).* Figure 10-2 displays a pathway proposed by Sato et al., 1989 and Shibasaki & Crandall, 2010. Individual eccrine sweat glands are innervated by multiple nerves (Kennedy et al., 1994), both cholinergic and adrenergic (Uno, 1977; Wang et al., 2011). Acetylcholine (Ach) only plays a role in thermoregulatory sweating, whereas noradrenaline is involved with thermoregulatory and emotional sweating (Hu et al., 2018). Ach released by the cholinergic nerves activates both muscarinic and nicotinic receptors on eccrine sweat glands (Fujii, Louie, McNeely, Amano, et al., 2017; Fujii, Louie, McNeely, Zhang, et al., 2017), resulting in sweat production. Any impairments in this process will reduce the sweat rate and depress the capacity to dissipate undesirable heat loads.

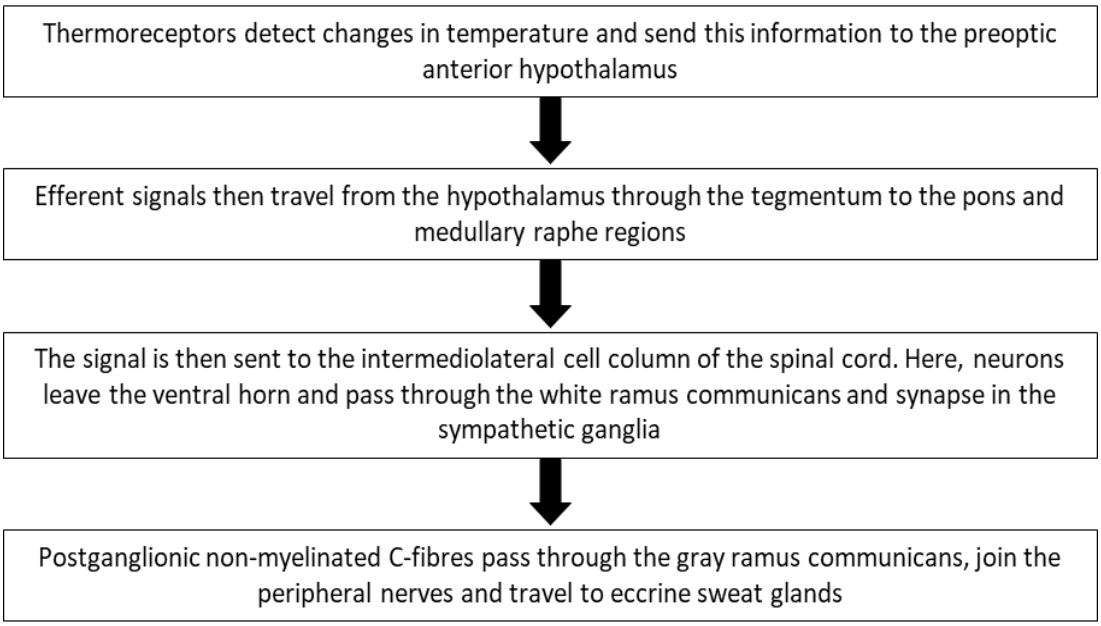

Figure 10-2. *Proposed neural pathway from thermoreceptors to sweat glands (Sato et al., 1989; Shibaski & Crandall, 2010).*

10.4.2.1 The Sweat Response During Passive Heat Exposure

Sweat rate is highly individualised and dependent upon various factors; men produce more sweat than women due to higher metabolic heat production, but the regional distribution of sweat is similar in both sexes (Smith & Havenith, 2012). Heat acclimation increases an individual's sweat rate, and increased aerobic fitness also increases sweat rate as a result of exercise training inducing modest heat acclimation from increasing core temperature during exercise (Sawka et al., 1996). However, with ageing the regional distribution of sweat becomes disrupted and the capacity to sweat reduces by ~17% from 65 to 70 years of age (Inoue, 1996). This effect is thought to be a result of a reduced function of each sweat gland rather than a reduction in the number of sweat glands (Inoue et al. 2004), and likely caused by local rather than central factors, such as a reduction in thermal sensitivity causing a delayed or weaker signal to the hypothalamus (Dufour & Candas, 2007). The size of a sweat gland accounts for ~80% of the variability in maximal sweat gland output as larger sweat glands are more sensitive to cholinergic activity (Sato & Sato, 1983). Consequently the reduction in output per sweat gland with ageing could be a sign of sweat gland atrophy (Inoue et al., 1999) or a reduced sensitivity to acetylcholine (Stapleton et al., 2015a). Work is needed to elucidate what influences the decline in sweat rate with ageing, and interventions that could be implemented to slow it down.

Older adults can have a different sweat response than younger adults. Older men (66 ± 2 years) have a higher onset of sweating threshold core temperature than young men (27 ± 2 years; oesophageal temperature; young 36.69 ± 0.06°C, older 36.97 ± 0.05°C) when at rest in a room warming at a rate of

1°C·min^{-1}, from 26°C to 40°C (Sagawa et al., 1988). At baseline both groups had a similar core temperature (young 36.79 ± 0.04°C, older 36.72 ± 0.09°C). This delayed sweating initiation resulted in a higher core temperature for the older group at the end of the heat exposure (125-130 min; young 37.36 ± 0.04°C, older 37.58 ± 0.06°C; Sagawa et al., 1988). It seems that older adults must experience more heat stress before sweating is initiated, which causes a greater level of heat strain, even at rest. Dufour & Candas (2007) showed similar outcomes during passive heat stress (40°C, 90 min). The older (68 ± 4 years) men in this study could only match the sweat rate of the younger (24 ± 3 years) men for the first 20 min of heat exposure. After this the younger group increased sweat rate significantly faster than the older group, but the older group were able to match the sweat response of a middle-aged (45 ± 3 years) group (Dufour & Candas, 2007). Sublingual (oral) temperature was used to assess core temperature, and all three age groups had a similar core temperature at the onset of the trial (~37.0°C). However, the older and middle-aged groups experienced a greater increase in core temperature than the young group by the end of the trial (young ~37.5°C, older & middle aged ~37.7; Dufour & Candas, 2007). These outcomes indicate that the decline in sudomotor function begins before old age, somewhere between 30-40 years.

A reduced sudomotor function will delay the effect of cooling that sweating creates therefore extending the period with increased core temperature that older adults experience compared with younger counterparts. These studies have some considerations: the study by Sagawa et al. (1988) had small sample groups, with only 6 older men, and 10 younger men. The participants were also all men, so similar results may not be seen in women. Dufour & Candas (2007) had a larger sample with 15 men in each group. However, the use of oral temperature as an indicator of core temperature is not ideal. Participants in the Dufour & Candas, (2007) study were matched for stature, body mass, and body surface area, but other variables were not reported such as body composition and maximal aerobic capacity. There is still limited research with a female population. It is important to understand when these sweating impairments begin, as these studies neglected to include adults aged 30-40 years, so it is not clear when these decrements occur.

10.4.2.2 The Sweat Response During Active Heat Exposure

During exercise, sweating is vital for dissipating heat gained from metabolism. If older adults cannot sweat as effectively as young adults during exercise, they will experience greater heat gain when exercising at the same intensity. Work by Larose et al. (2013) investigated the influence of age on sweat rate during intermittent exercise (4 x 15 min separated by 15 min rest, at a metabolic heat production of 400 W in 35°C, 20% RH). Eighty-five men were matched for $\dot{V}O_2$peak (21-30 years, $\dot{V}O_2$peak 53.1 mL·kg LBM^{-1}·min^{-1} n 18; 40-44 years, $\dot{V}O_2$peak 53.1 mL·kg LBM^{-1}·min^{-1}, n 15; 45-49 years,

$\dot{V}O_2$peak 55.4 mL·kg LBM^{-1}·min^{-1}, n 15; 50-55 years, $\dot{V}O_2$peak 49.8 mL·kg LBM^{-1}·min^{-1}, n 21; 56-70 years, $\dot{V}O_2$peak 47.9 mL·kg LBM^{-1}·min^{-1}, n 16). Compared to the 21-30 year-olds the 56-70 year-olds had a significantly lower sweat rate in all four exercise bouts, the 50-55 year-olds had a lower sweat rate in the second and third exercise bouts, and the 45-49 year-olds had a lower sweat rate in the final exercise bout. However, during the resting periods there was no difference in sweat rate between the groups (Larose et al., 2013). Overall body heat storage was significantly higher for all of the men aged ≥40 years than the 21-30 year olds (21-30 years ~150 KJ, 40-44 years ~250 KJ, 45-49 years ~275 KJ, 50-55 years ~250 KJ, 56-70 years ~250 KJ; Larose et al., 2013). Despite these differences in body heat storage, there was no difference between the age groups for rectal temperature throughout the trial. This outcome may be relate to the use of direct calorimetry providing a more precise measure of heat storage, while rectal temperature is much more affordable and convenient (Kenny et al., 2017b), it can only give an indication of heat storage in a specific region of the body (Larose et al., 2013). As the groups were matched for fitness, these differences in sweat rate and heat storage are age-related. With increasing age, the onset of sweating is delayed, particularly for mean aged >55 years, resulting greater levels of heat storage in older men. This impairment in thermosensitivity is evident by middle-age, putting even more people at risk of heat illness. This study did not include men aged 31-39 years, thus the need for work investigating what happens to thermoregulatory capacity during the 4th decade of life is still warranted.

Maintaining a high fitness level attenuates the natural decline in sweat rate with ageing (Best et al., 2012; Stapleton et al., 2015a, 2015b; Tankersley et al., 1991). Tankersley et al. (1991) recruited three groups, a young group (29 ± 1 years; $\dot{V}O_2$max 44.0 ± 2.7 mL·kg^{-1}·min^{-1}), an older group matched for fitness with the young group (64 years; $\dot{V}O_2$max 46.4 ± 2.1 mL·kg^{-1}·min^{-1}), and an older unfit group (66 ± 1 years; $\dot{V}O_2$max 32.9 ± 2.1 mL·kg^{-1}·min^{-1}). When these groups cycled in the heat (30°C, 50-60% RH) for 20 min at 67.5% $\dot{V}O_2$max, the young group had the highest chest sweat rate and the older unfit had the lowest (significantly lower than young at 10 min), but the older fit group's chest sweat rate was between the young and older unfit groups. All three groups began sweating at a similar core (oesophageal) temperature (young 36.82 ± 0.11°C, older fit 36.84 ± 0.09°C, older unfit 36.72 ± 0.10°C). Skin and oesophageal temperature was similar between the groups throughout the trial. This study demonstrates that ageing does reduce local chest sweat rate in men exercising in the heat, but that maintaining a high level of aerobic fitness will ameliorate this decline, offering a greater level of evaporative heat dissipation than unfit older men. Despite the reduced chest sweat rate, the older unfit men had a similar core temperature to the young and older fit groups throughout the trial. However, as the exercise was relative to their overall fitness level, the older unfit group were exercising at a lower metabolic heat production. Presumably, the older unfit group should have

experienced a lower core temperature than the other two groups if there was no decline in sweat rate.

Studies have differentiated age and fitness to clarify whether the reduction in sudomotor function with age is caused by the age-related decline in fitness, or by ageing itself. Best et al. (2012) recruited three groups of men, a young highly fit group (26 ± 4 years, 68.1 ± 4.8 mL·kg^{-1}·min^{-1}), a young moderately fit group (27 ± 5 years, 55.9 ± 3.4 mL·kg^{-1}·min^{-1}), and an older highly fit group (56 ± 5 years, 59.3 ± 6.7 mL·kg^{-1}·min^{-1}). Each group cycled for one hour at 70% of their $\dot{V}O_2$max in hot conditions (35°C, 40% RH). The mean whole body sweat loss (young highly fit 1.78 kg, young moderately fit 1.37 kg, older highly fit 1.39 kg), sweat loss relative to body surface area (young highly fit 0.93 kg·m^{-2}, young moderately fit 0.69 kg·m^{-2}, older highly fit 0.76 kg·m^{-2}), and sweat loss relative to body surface area and power output (young highly fit 3.69 g·m^{-2}·W^{-1}, young moderately fit 3.30 g·m^{-2}·W^{-1}, older highly fit 3.83 g·m^{-2}·W^{-1}) did not differ significantly between the groups. Core (rectal), and skin temperature were similar between the groups throughout trial. It appears that aerobic fitness did not influence sweat rate in young men exercising in the heat. It is well established that a higher aerobic capacity results in a higher sweat rate (Green et al., 2004). The normative value for $\dot{V}O_2$max of men aged 30 years is 48 mL·kg^{-1}·min^{-1} (Shvartz & Reibold, 1990). Thus, the young moderately fit group in this study would be considered fit and not representative of the general unfit population. Inclusion of a young unfit group should demonstrate lower whole body sweat rates in this group, thus demonstrating that aerobic fitness, as well as age, influences whole body sweat rate. The older men would also be considered very fit as the normative value for men aged 50 years is 35 mL·kg^{-1}·min^{-1} (Shvartz & Reibold, 1990). Best et al. (2012) used a relatively young group for their older age group at 56 ± 5 years. Nevertheless, declines in thermoregulatory functioning have been identified as early as the fifth decade (Dufour & Candas, 2007; Larose et al., 2013). Overall, the findings from this study indicate that a high aerobic fitness capacity will protect against declines in sweating function into the sixth decade, however, the level of fitness required is high. Future work is required to investigate if this protection carries on beyond the sixth decade of life, and whether lower levels of aerobic fitness offer as much protection.

Research has investigated the influence of age and fitness on sudomotor function and heat storage during exercise in the heat (Stapleton et al., 2015a). This study had 4 groups of men, young (21 ± 1 years, $\dot{V}O_2$peak 50.0 ± 3.7 mL·kg^{-1}·min^{-1}), middle-aged trained (49 ± 5 years, $\dot{V}O_2$peak 51.0 ± 6.8 mL·kg^{-1}·min^{-1}), middle-aged untrained (48 ± 5 years, $\dot{V}O_2$peak 37.3 ± 3.5 mL·kg^{-1}·min^{-1}), and older (65 ± 3 years, $\dot{V}O_2$peak 37.9 ± 7.8 mL·kg^{-1}·min^{-1}); the older and middle-aged untrained groups had a substantially lower $\dot{V}O_2$peak than the young and middle-aged trained groups. Participants cycled for

30 min bouts with 15 min rest between bouts at increasing exercise intensity (bout 1 300 W, bout 2 400 W, bout 3 500 W) in a direct calorimeter set to 40°C and 15% relative humidity. There was no difference between the groups in mean body temperature at the onset of sweating. The rate of evaporative heat loss was greater in the young group than the other groups during exercise bout 1 (young 1,575 ± 1,160 $W \cdot °C^{-1}$, middle-aged trained 1,022 ± 270 $W \cdot °C^{-1}$, middle-aged untrained 879 ± 415 $W \cdot °C^{-1}$, older 721 ± 318 $W \cdot °C^{-1}$), similar during exercise bout 2, while in exercise bout 3 the middle-aged untrained and older groups rate of heat loss was lower than the young and middle-aged trained groups (young 1,295 ± 629 $W \cdot °C^{-1}$, middle-aged trained 1,031 ± 352 $W \cdot °C^{-1}$, middle-aged untrained 513 ± 238 $W \cdot °C^{-1}$, older 411 ± 263 $W \cdot °C^{-1}$). These differences in rate of evaporative heat loss resulted in a greater increase in body heat content for the middle-aged untrained and older groups than the young and middle-aged trained groups (young ~325 KJ, middle-aged trained ~400 KJ, middle-aged untrained ~550 KJ, older ~500 KJ). Unlike Larose et al. (2013), Stapleton et al. (2015a) identified a higher core temperature (oesophageal) at the end of the third exercise bout in the older and middle-aged untrained compared with the young and middle-aged trained groups (young ~37.4°C, middle-aged trained ~37.3°C, middle-aged untrained ~31.9°C, older ~37.8°C) All groups began the trial at a similar core temperature (young 36.77 ± 0.4°C, middle-aged trained 36.72 ± 0.2°C, middle-aged untrained 36.83 ± 0.2°C, older 36.83 ± 0.3°C). Collectively these results demonstrate that maintaining fitness will help to prevent a decline in evaporative heat loss at least into middle age. The addition of an older trained group would help to identify whether the protection a higher level of aerobic capacity offers continues beyond middle age.

Another study, similar to Larose et al. (2013) and Stapleton et al. (2015a), investigated the effect of fitness on the age-related decline in heat loss for females (Stapleton et al., 2015b). This study used that same protocol as Stapleton et al. (2015a), however the exercise intensities were lower (bout 1 255 W, bout 2 325 W, bout 3 400 W). The young group was aged 23 ± 4 years, the older group 58 ± 5 years, and the groups were matched for aerobic fitness (young 39.7 ± 8 $mLO_2 \cdot kg^{-1} \cdot min^{-1}$, older 39.0 ± 7.7 $mLO_2 \cdot kg^{-1} \cdot min^{-1}$), body composition (young 24 ± 5% body fat, older 24 ± 5% body fat), and body surface area (young 1.7 ± 0.1 m^2, older 1.7 ± 0.1 m^2). The younger women increased their body heat storage by ~250 KJ, and the older women by ~400 KJ. During the rest periods there was no difference in body heat storage between the groups. The overall greater level of heat storage in the older women resulted in a higher core temperature at the end of the trial (young 37.56 ± 0.3°C, older 37.91 ± 0.4°C) after both groups started at a similar core temperature (young 37.14 ± 0.3°C, older 37.18 ± 0.2°C). Skin temperature did not differ between the groups at any point in the trial. The increase in body heat storage for the older women was caused by a delayed onset of sweating in the second and third exercise bouts. During bout 2 the older women started sweating when core temperature was 0.27°C

warmer than the younger women, and in bout 3 they were 0.30°C warmer at the onset of sweating. Evaporative heat loss was also lower for the older women during exercise, in exercise bout 1 at 5 and 10 min, for exercise bout 2 at 20, 25, and 30 min, and in exercise bout 3 from 10 min onwards evaporative heat loss was significantly lower in the older women than the young women. These results demonstrate that older women have a reduced level of evaporative heat loss when exercising in the heat, despite being matched for fitness, body composition, and body surface area. This outcome contradicts the findings in men (Stapleton et al., 2015a) that having the same level of aerobic fitness in older men and young protects against the decline in evaporative heat loss. Further work should investigate why maintaining aerobic fitness reduces the decline in evaporative heat loss in men but not in women. The inclusion of an older unfit group may reveal that a higher aerobic fitness protects against an even greater decline in evaporative heat loss in women. At 58 ± 5 years, the older group in this study (Stapleton et al., 2015b) were relatively young, thus it is clear that the decline in sweating function occurs by the sixth decade in women.

Maintaining a high level of aerobic fitness reduces the decline in sudomotor functioning in men. However, to maintain a $\dot{V}O_2$max that can be protective (>40 mL·kg^{-1}·min^{-1}; Best et al., 2012; Ho et al., 1997; Tankersley et al., 1991) requires a substantial amount of physical activity. After 9-12 months of training four times a week for 45 min at ~80% of maximal heart rate, men aged 65 ± 4 years increased their $\dot{V}O_2$max from 28 mL·kg^{-1}·min^{-1} to 35 mL·kg^{-1}·min^{-1}, and women aged 63 ± 3 years from 22 mL·kg^{-1}·min^{-1} to 27 mL·kg^{-1}·min^{-1} (Kohrt et al., 1991). Despite the prolonged exercise regime, these participants did not achieve the $\dot{V}O_2$max level known to ameliorate the decline in thermoregulatory functioning. These participants also benefitted from a structured training programme led by dedicated exercise professionals. Older women (78 ± 3 years) struggled to complete an hour of cycling at 50 W in 35°C, 40% RH environment (Daanen & Herweijer, 2015). Out of 24 sessions, over half had to reduce their workload to 30 W to ensure they completed the hour-long session. Only 4.1% of older adults (69 ± 7 years) in the UK meet the current activity guidelines of ≥30 min a day of a movement intensity ≥2,020 counts per min (Berkemeyer et al., 2016). Thus, increasing $\dot{V}O_2$max to a protective level requires a substantial commitment to exercise, training and/or physical activity that many older adults are incapable of achieving given age-related functional losses.

10.4.2.3 Possible Mechanisms Causing Age-Related Decline in Sweating

There is an age-associated reduction in sweat rate, and recent work has focussed on establishing causes of this decline. At rest, nicotinic-mediated sweating is not influenced by healthy ageing, however, muscarinic-mediated sweating declines with age (Fujii et al., 2019). Ageing effects sweat rate differently in men and women. Basal sweat rate in men was similar between the young (24 ± 3

years) and older groups (61 ± 8 years) with the older men tending to have a higher sweat rate (0.25 v 0.19 mg·min^{-1}·cm^{-2}). The older (60 ± 8 years) women had a lower resting sweat rate than young (22 ± 5 years) women (0.17 v 0.24 mg·min^{-1}·cm^{-2}; Fujii et al., 2019). This decline in sweat rate in women is seen at rest without the addition of a heat stress. Local methacholine (a muscarinic receptor agonist) administration was used to assess muscarinic-mediated sweating at a range of doses (0.0125, 0.25, 5, 100, and 2,000 mM). The methacholine increased muscarinic mediated sweating from baseline in young men at all doses, young women at doses ≥5 mM, and for older men and women at doses ≥0.25 mM. For both age groups, the women had a lower sweat rate than the men, for the young group at all doses of methacholine, and for the older group in doses ≥5 mM. Young men had a greater sweat rate than older men at 0.0125, 0.25, and 5 mM of methacholine, thus they needed higher doses to reach the same sweat rate as young men. With the women the difference in sweat rate occurred only at 2,000 mM, the highest dose. These results demonstrate that muscarinic-mediated sweating declines in older age for both men and women, however this occurs in a different way between the sexes with women being impaired at high doses and men at lower doses of methacholine (Fujii et al., 2019). With local nicotine (a nicotinic receptor agonist) administration there were no differences in sweating response between any of the groups (Fujii et al., 2019).

These results from Fuji et al. (2019), coupled with those from Stapleton et al. (2015a, 2015b), demonstrate that ageing affects sudomotor function in men and women differently, thus, careful consideration regarding sex is required when examining decrements in thermoregulatory function with age. Further work is needed to elucidate why sex influences the decline in thermoregulatory functioning. Any research in thermoregulation and ageing should consider how the sex differences will impact the results of the study even if investigating sex differences is not the aim of the research.

As well as being involved with skin blood flow, nitric oxide and the cyclooxygenase pathway are also involved with sudomotor function. During intermittent exercise (2 x 30 min cycling, separated by 20 min rest) in the heat (35.0°C, 20% RH), nitric oxide inhibition with L-NAME or cyclooxygenase inhibition with Ketorolac, attenuated sweat rate in young (24 ± 4 years) men (Fujii et al., 2014). However, this effect was only evident during moderate intensity (rate of metabolic heat production 400 W) cycling, and there was no difference in forearm sweat rate at higher intensity (rate of metabolic heat production 700 W) cycling. The effects did not persist into the post-exercise rest period. Combining L-NAME and Ketorolac did not decrease the sweat response further than L-NAME or Ketorolac alone (Fujii et al., 2014), implying they function as part of the same pathway or cooperatively. If they worked independently there would be an additive effect of L-NAME and Ketorolac administration.

Further work has examined nitric oxide mediated sweating in older men. The inhibition of nitric oxide with L-NAME has little impact of forearm sweat rate during intermittent cycling (3 x 15 min cycling at 300 Wm^{-2} separated by 15 min rest) in the heat (35°C, 20% RH) in men aged 64 ± 5 years (Stapleton et al., 2014). This study included a young active (23 ± 3 years, $\dot{V}O_2max$ 50.2 $mL \cdot kg^{-1} \cdot min^{-1}$) male group that exhibited a reduction in forearm sweating with infusion of L-NAME at the end of each exercise bout (Stapleton et al., 2014). Nitric oxide-dependent thermoregulatory sweating is diminished in older adults. The rise in core (oesophageal) temperature from baseline to post trial tended ($p = 0.067$) to be greater in the older men than the young men (1.12 v 0.84°C; Stapleton et al., 2014), while this was an insignificant difference, it tends to suggest that the reduced sweat rate leads to an increased level of heat storage. If this increased heat storage cannot be offset via thermoregulatory behaviours, then older men will be at an increased risk of heat-related illness during heat wave events.

With age, sweating function declines due to a reduced function of individual sweat glands, resulting in a reduced capacity to dissipate heat via wet heat exchange. Research has shown that having a high $\dot{V}O_2max$ in older age ameliorates this decline in sudomotor functioning. However, older adults face issues with adherence and tolerance to exercise programs required to maintain a high level of aerobic fitness. Behavioural thermoregulation may provide a more suitable avenue for thermoregulatory interventions that can be easily implemented to the population at large.

10.4.3 Immune response to heat stress

During heat stress, blood is redistributed towards the skin for dry heat dissipation, causing a reduction in blood flow to the internal organs, including the gut (Ho et al., 1997). This leads to a hypoxic environment (Hall et al., 1999), resulting in a leaky gut that allows endotoxin to escape from the gut and cause an inflammatory immune response in both passive (Bouchama et al., 1991) and exertional heat stress (Selkirk et al., 2008; Zuhl et al., 2014). This sequence of events pose a risk to adults aged 65 years and over as ageing causes an increase in the basal inflammatory state (Claesson et al., 2012) as the result of prolonged exposure to antigen stress (Müller-Werdan, 2007) and an increasing amount of extra-cellular DNA (Lan et al., 2019). Thus, older adults have a reduced capacity to initiate an immune response during heat stress.

Older adults (77 ± 5 years) have an increased level of serum zonulin (a marker of gut permeability) than young (23 ± 4 years) adults (~2.3 v ~1.6 $ng \cdot mL^{-1}$; Qi et al., 2017). These samples were taken at rest, without the additional stress of heat exposure or exercise, which is known to increase gut permeability (Zuhl et al., 2014). The groups were also matched for stature, body mass, and body mass index suggesting that this increase is age-related. Qi et al. (2017) also investigated the relationship between physical activity and gut integrity. A significant negative relationship ($r = -0.41$, $p = 0.016$)

was evident between the number of daily steps and the level of serum zonulin, with the more active adults having a greater gut integrity. These results provide evidence that ageing decreases gut integrity, which will allow greater levels of endotoxin to escape into the system circulation. Maintaining a higher level of physical activity can ameliorate this increase in zonulin. The physical activity relationship requires further work in adults ≥65 years as Qi et al. (2017) did not partition the age groups for this analysis, thus it is possible that those with the high number of steps could all have been from the younger group. Further work is needed to assess how heat exposure impacts the gut permeability in the over 65s.

The microbiota diversity in the gut plays an important role in heat induced endotoxemia. With age there are alterations in the gut microbiota increase gut permeability resulting in an increase in cytokine expression and a reduced ability to repair damaged DNA (Guedj et al., 2019; Thevaranjan et al., 2017). Increasing the diversity through a diet high in anti-inflammatory and antioxidant properties will help to maintain the integrity of the gut lining and reduce the amount of endotoxin that can leak into systemic circulation (Armstrong et al., 2018). With ageing there is a decrease in the diversity of gut microbiota (Biagi et al., 2010; Claesson et al., 2012). This mechanism could explain the increase in gut permeability seen in adults aged ≥70 years (Qi et al., 2017). Work needs to be carried out to reveal how gut permeability in older adults is altered with heat stress. As adults aged ≥70 years have an increased gut permeability at rest and a higher level of basal inflammation, the addition of heat stress may increase gut permeability further and put a strain on the aged immune system that is already functioning closer to its capacity, resulting in endotoxic shock earlier than in younger adults.

There appears to be an interaction between the immune system, gut permeability, and cardiovascular function. Patients with chronic heart failure have pathogenic gut flora overgrowth, increased gut permeability, and increased C-reactive protein (CRP) levels, this relationship correlated positively with severity of heart failure (intestinal permeability and right arterial pressure $r = 0.55$, $p < 0.0001$; CRP and intestinal permeability $r = 0.78$, $p < 0.0001$; CRP and right arterial pressure $r = 0.78$, $p < 0.0001$; Boschi et al. 2015). CRP is a strong predictor of cardiovascular disease and mortality (Proctor et al. 2015), and reduces nitric oxide availability (Shrivastava et al. 2015). Researchers should evaluate interventions for reducing inflammation or gut permeability to improve the immune response and reduce cardiovascular strain. Probiotics, prebiotics and phenols can increase the diversity of gut microbiota and reduce gut permeability (Boulangé et al. 2016; Marchesi et al. 2016). Therefore probiotics, prebiotics, and phenols offer a potential dietary intervention for reducing ageing related inflammation and increasing gut integrity, and thus reducing cardiovascular strain.

Endotoxin has emerged as playing a role in heat stress in aged animals (Hall et al., 2001) and young adult humans (Selkirk et al., 2008). Ageing increases the risk of endotoxic shock during heat stress due to an impaired immune system in those aged ≥60 years (Navaratnarajah & Jackson, 2017) and a decrease in gut wall integrity in adults aged ≥70 years (Qi et al., 2017). Research investigating the immune response to heat stress in older adults (≥60 years) is required to reveal if reduced immune functioning in older adults increases the risk of heat morbidity and/or mortality. Work is also required to identify if the ingestion of probiotics and prebiotics can ameliorate the impact of increased endotoxin circulation during heat stress.

10.5 Conclusion

There is substantial evidence supporting predictions on patterns of global temperature and climate change and how this will lead to an increase in heat wave events. Policy makers need to respond to both changes in climate and demographic shifts with an ageing population worldwide. The combination of these challenges will likely lead to an increase in heat-related morbidity and mortality across the world.

With ageing there is a decline in sudomotor functioning and skin blood flow responses, causing a reduction in the capacity to dissipate heat during heat exposures. A high level of aerobic fitness reduces the rate of these decline, allowing fit older adults to dissipate more heat than their unfit age-matched counterparts during heat stress. Sex influences this age-related decline in thermoregulatory functioning. More work is required with female participants to identify why their relationship between age and thermoregulation is different to men. Future work investigating ageing and thermoregulation also needs to acknowledge this influence of sex on thermoregulatory decline. It seems advisable that research studies should separate men and women to ensure appropriate interpretation of results, for evidence-based framing of outcomes and practical applications.

Thermoregulatory behaviours offer simple and effective strategies for reducing heat strain during heat wave events. However, many adults aged ≥65 years are still dying despite these simple strategies. Research is crucial to identify why older adults are not implementing behavioural changes to reduce their heat stress. Skin temperature and thermal comfort drive thermoregulatory behaviours in young men, this relationship is not well established in adults aged ≥60 years. A greater understanding of how ageing impacts thermoregulatory behaviours will allow for improved guidelines on how to cope during heat wave events.

11 General Methods

11.1 Experimental Design

This thesis has used laboratory based research, this allowed for the environmental conditions to be tightly controlled ensuring that each participant was exposed to similar conditions. This permitted thermoregulatory behaviours to be assessed through changes in exercise intensity while preventing other thermoregulatory behaviours. The laboratory based approach also meant that more invasive physiological measures could be employed than would be practical in a field based setting (e.g. rectal temperature instead of aural).

11.2 Ethics

All experiments within this thesis were approved by Plymouth Marjon University Ethics Committee and conducted within the guidelines of the Declaration of Helsinki, 2013. All standard operating procedures and risk assessments for Plymouth Marjon University were followed during experimentation.

11.3 Equipment Cleaning

Standard laboratory cleaning procedures were used to prevent contamination. Equipment was cleaned after each use. Heart rate monitors and straps were soaked in sterilising fluid for 30 min, rinsed and dried. Skin thermistors and the laser Doppler probe were cleaned using alcohol wipes. All other equipment was wiped down using a disinfectant surface spray.

11.4 Waste Disposal

All biological waste was handled and disposed of in line with relevant guidelines. All single-use equipment was disposed by incineration. All sharps were disposed in designated sharps bins.

11.5 Participants

Participants in this thesis were all adults (≥18 years) and were excluded if they had a history of cardiovascular disease, diabetes mellitus, or heat stroke, if they were a smoker, if they had been exposed to heat strain in the previous 12 weeks (saunas, steam rooms, sun bed, foreign travel to hot climates). All women were postmenopausal and not taking hormone replacement therapy. Once participants were familiarised with the laboratory procedures written informed consent was given for each study.

11.6 Termination of Experiment Criteria

All participants were informed of their right to withdraw from the studies at any time without the requirement to provide a reason. Exercise and heat exposure was terminated if:

- T_{re} ≥39.0 for the treadmill experimental protocols, and ≥39.5°C for the cycling experimental protocols in line with the ethical approvals given by the university
- The participant withdrew owing to volitional exhaustion
- The participant began to show signs of heat illness

Once removed from the environmental chamber, cooling strategies were implemented. Participants were seated in a thermoneutral room with a cooling fan and an external door open. They were also given cool water to drink *ad libitum*.

11.7 Pre-Trial Controls

Participants were all asked to follow the same pre-trial controls to standardise each trial: 48 h before each trial participants were asked to avoid consuming; alcohol, caffeine, supplements, pain killers, and strenuous exercise. The evening prior and 2 h before each trial participants were asked to consume 500 mL of water, as recommended by the American College of Sports Medicine (Convertino et al., 1996; Sawka et al., 2007). Participants were asked to confirm that they had followed these guidelines upon their arrival at the laboratory.

11.8 Anthropometric Assessment

All anthropometric measures were taken by an ISAK (International Society for the Advancement of Kinanthropometry) Level 1 Accredited experimenter.

11.8.1 Stature

Stature was assessed to the nearest mm using a stadiometer (217, Seca, Hamburg, Germany). Participants removed shoes and stood with their back to the stadiometer, feet together, heels, buttocks, and upper back touching the stadiometer. The head was then placed in the Frankfort plane, and the stadiometer arm lowered until it rested on the most superior aspect of the head.

11.8.2 Body Mass

Body mass was assessed using a digital scale (MC-180MA, Tanita, Tokyo, Japan) and recorded to the nearest 0.05 kg. Any nude body mass assessments were carried out by the participant alone in private, and participants self-reported their body mass to the experimenter.

11.8.3 Skinfolds

Skinfold measurements were taken in accordance with ISAK guidelines, at 4 sites (triceps, biceps, subscapular, and supra-iliac) on the right-hand side of each participant. Participants stood relaxed in the anatomical position. Measures were taken using skinfold callipers (Harpenden, West Sussex, UK). Skinfolds were recorded to the nearest 2 mm. Measures were taken in duplicate; a third measure was taken if the first two were not within 5% of each other. A mean was used if two measures were taken, and the median if three were taken.

Body density was calculated using Durnin & Womersley, (1974).

Table 11-1. *Body density formula taken from (Durnin & Womersley, 1974). D = body density. L = log of ∑skinfolds.*

Age (years)	Male	Female
17-19	D = 1.1620 - (0.0630 x L)	D = 1.1549 - (0.0678 x L)
20-29	D = 1.1631 - (0.0632 x L)	D = 1.1599 - (0.0717 x L)
30-39	D = 1.1422 - (0.0544 x L)	D = 1.1423 - (0.0632 x L)
40-49	D = 1.1620 - (0.0700 x L)	D = 1.1333 - (0.0612 x L)
≥50	D = 1.1715 - (0.0779 x L)	D = 1.1339 - (0.0645 x L)

Body composition was then calculated using the Siri equation:

Equation 11-1. *Siri equation.*

$$Body\ fat\ \% = \left(\frac{495}{Body\ Density}\right) - 450$$

11.9 Temperature

All temperature data was recorded by a data logger (SQ2040, Grant Instruments, Cambridge, UK) at 15 sec intervals throughout experimental trials. The final four recordings of each five min period were averaged to give a 1 min mean every 5 min.

11.9.1 Core Temperature

Thermoregulation research requires measurement of heat gain/loss. Direct calorimetry provides a direct measure of this. However, direct calorimeters big enough to house a human are rare and expensive, there is only one direct human calorimeter in use today, located at the University of Ottawa (HEPRU, 2021; Kenny, Notley, et al., 2017). Therefore, researchers use core temperature measurement to assess heat transfer. Non-invasive measurements of core temperature offer the benefit of ease and a lack of discomfort for participants. However, non-invasive measurements can be easily influenced by ambient temperature or other activities such as consuming hot or cold liquids

(Moran & Mendal, 2002). Thus oral, axilla, and tympanic temperature were not considered for this thesis as participants would be free to consume water and the ambient conditions would differ between trials.

Invasive measures offer more accuracy than the non-invasive techniques, but with downside of being more uncomfortable for the participant. The most accurate measure is pulmonary artery as blood is brought to the measurement site from the core and the rest of the body (Brengelmann, 1987). However, this method requires a catheter to be fed into the pulmonary artery by a trained professional. This presents a level of expertise and invasion that was deemed unnecessarily high for this thesis.

Telemetric pills provide a relatively easy method of assessing core temperature for participants. The pills are ingested several hours prior to data collection and transmits the temperature of the digestive tract. Transit time of the pill can vary widely from ~12 to ~136 h (McKenzie & Osgood, 2004a). It is impossible to detect the exact location of the pill during its transit through the body, but it has been shown to correlate well with rectal probe data (r^2 = 0.80; McKenzie & Osgood, 2004). These pills present a greater financial burden at ~£50 each, whereas the thermistors required for oesophageal and rectal temperature are ~£5 each.

Rectal and oesophageal temperature have been used throughout physiology research in humans. Both provide a greater level of accuracy than the non-invasive measures, and a greater level of practicality than pulmonary artery measures. Despite being slower to react to core temperature changes than oesophageal temperature, rectal temperature is often considered to be the most practical technique (Moran & Mendal, 2002). It is well tolerated by participants, unlike oesophageal temperature, which causes irritation to the nasal passages (Stuart et al., 2000). Rectal temperature was used to evaluate deep body temperature and indicate core temperature throughout the studies presented here. A single-use rectal thermistor (Philips, Amsterdam, The Netherlands) was self-inserted by participants ~12 cm past the anal sphincter. Rectal temperature was used as it is an effective, cost-effective, and well-tolerated method of assessing core temperature.

11.9.2 Skin Temperature

Skin temperature was measured using surface temperature thermistors (EU-U-VL5-0, Grant Instruments, Cambridge, UK) at 4 sites (chest, biceps, thigh, and calf) on the left side of the body. The thermistors were taped to the skin using a single layer of porous kinesiology tape (Rock Tape, Essex, UK). Mean skin temperature was calculated using Ramanathan (1964).

Equation 11-2. *Weighted mean skin temperature* (Ramanathan, 1964).

$$Skin\ temperature = 0.3\left(T_{chest} + T_{biceps}\right) + 0.2\left(T_{thigh} + T_{calf}\right)$$

11.9.3 Ambient Temperature

A temperature thermistor (EU-U-VL5-0, Grant Instruments, Cambridge, UK) hung from the ceiling ~30 cm above the head of the participant measured ambient temperature.

11.10 Heart Rate

Heart rate was measured using a short-range telemetry system (FT1, Polar Electro, Kempele, Finland). Values were recorded at 5 min intervals.

11.11 Whole-Body Sweat Rate

Whole-body sweat rate (WBSR) was assessed using the change in body mass calculation. Dry nude body mass was measured before and after each trial. Urine output, fluid consumption and trial duration were accounted for in the calculation:

Equation 11-3. *Whole body sweat rate equation.*

$$WBSR = \frac{((BM_{pre} - BM_{post}) + Fluid\ Consumed - Urine\ Output)}{Trial\ Duration}$$

Where:

WBSR is measure in $L{\cdot}h^{-1}$

BM = Body mass in kg

Fluid consumed and urine output is measured in kg

Trial duration is measured in h

11.12 Blood Pressure

Blood pressure was measured at the same time as skin blood flow measures. Blood pressure was measured using an automatic blood pressure monitor (HEM-907, Omron Healthcare Inc., Japan), from the right arm, the opposite arm to the skin blood flow measurement. From blood pressure, mean arterial pressure was calculated.

Equation 11-4. *Mean arterial pressure equation.*

$$MAP = \frac{(2\ \times DP) + SP}{3}$$

Where:

MAP = Mean arterial pressure

DP = Diastolic pressure

SP = Systolic pressure

Mean arterial pressure was used to calculate cutaneous vascular conductance.

Equation 11-5. *Cutaneous vascular conductance equation.*

$$CVC = \frac{LDF}{MAP}$$

Where:

CVC = Cutaneous vascular conductance

LDF = Laser Doppler flow

MAP = Mean arterial pressure

11.13 Perceptual Measures

11.13.1 Rating of Perceived Exertion

Preliminary data collection sessions included a familiarisation with the rating of perceived exertion (RPE) scale (Borg, 1982). The RPE scale was shown to participants and a verbal explanation given of what the scale represented. Participants were asked to walk (Experimental Chapter One, Experimental Chapter Two) or cycle (Experimental Chapter Three) at what felt like an RPE of 13 ('somewhat hard'). They were blinded to their speed/power output and told to adjust the treadmill speed/cycle ergometer resistance as needed to achieve an RPE of 13. Participants indicated when they reached a treadmill speed/cycle ergometer resistance felt 'somewhat hard' (RPE 13). After 3 min participants confirmed that the treadmill speed/cycle ergometer power output represented an RPE of 13 and this value was recorded (and not disclosed to the participant). Participants then sat at rest for 5 min. Participants repeated this procedure until they were able to replicate the speed/power output that elicited an RPE of 13. On average this took 4 ± 1 attempts.

During exercise RPE was assessed every 5 min. A laminated paper copy of the Borg scale, ranging from 6 to 20 was used. The scale had written descriptions of various levels of exertion; "very, very light" (6), "very light" (9), "fairly light" (11), "somewhat hard" (13), "hard" (15), "very hard" (17), and "very, very hard" (19) (Borg, 1982).

11.13.2 Thermal Sensation

The American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) Scale for thermal sensation was used. The scale went from -3 to +3, with wording to describe each number; "cold" (-3), "cool" (-2), "slightly cool" (-1), "neutral" (0), "slightly warm" (+1), "warm" (+2), "hot" (+3).

Participants were asked to describe their level of thermal sensation every 5 min throughout experimental conditions.

11.13.3 Thermal Comfort

ASHRAE define thermal comfort as the "condition of mind that expresses satisfaction with the thermal environment" (The American Society of Heating Refrigerating and Air-Conditioning Engineers, 2013). The Bedford scale of thermal comfort (Bedford, 1936) was used to measure the comfort of participants every 5 min throughout experimental conditions. This is a 7-point scale from -3 to +3, with words to describe each number; "much too cool" (-3), "too cool" (-2), "comfortably cool" (-1), "comfortable" (0), "comfortably warm" (1), "too warm" (2), "much too warm" (3).

Perception of thermal comfort differs slightly from thermal sensation. Comfort may be influenced by the expectation of the environment and adaptation to the environment (Zhang, 2003). An individual may sense that they are warm (+2 on the ASHRAE scale) but feel comfortable (0 on the Bedford scale) at this level of thermal sensation, hence the use of both thermal comfort and sensation measurements.

11.14 Physical Fitness and Physical Activity

11.14.1 Six-minute Walk Test

The 6-minute walk test (6MWT) was conducted during preliminary visits in a sports hall following guidelines from the American Thoracic Society (Crapo et al., 2002). This test was selected over maximal oxygen uptake testing as it was deemed a more accessible exercise test and reflects daily living exercise (Burr et al., 2011). A 30 m straight line distance was measured out with a tape measure and marked with cones every 5 m. Participants were verbally instructed to walk as far as they could in 6 min without breaking into a run. They were also informed that at the end of the test they should stop and remain stationary until a researcher had noted their location along the tape measure. They walked around the line of cones in a clockwise fashion. They were informed every 2 min of how much time had passed and how much time remained. Heart rate and RPE measures were taken every 2 min. A researcher noted every lap the participants completed on a tally chart. A chair was located close to the course in case participants needed to rest. No encouragement was provided to participants.

11.14.2 Physical Activity Scale for the Elderly

The physical activity scale for the elderly (PASE) was administered during preliminary trials. Participants were asked to consider their activity levels for the previous 7 days when responding to the questionnaire. The PASE was chosen as it was specifically developed for older adults (≥65 years)

to capture daily living activities of this population (Washburn et al., 1993). Items of the PASE were weighted (

Table **11-2**). Half the items are calculated as a mean number of hours participated in per day multiplied by the PASE weight; the other half are scored at as engaged in or not engaged in (see

Table **11-2** for an example).

Table 11-2. *PASE item weights. Data taken from* (Washburn et al., 1993)*. * denotes activities that are scored as engaged in (1) or not engaged in (0).*

PASE Activity	PASE Weight	Example	
		Mean $h \cdot d^{-1}$	Item Score
Muscle strength/endurance	30	0.3	9.0
Strenuous sport	23	0.2	4.6
Moderate sport	23	0.6	13.8
Light sport	21	0.0	0.0
Job involving standing or walking	21	5.0	105.0
Walking	20	0.5	10.0
Lawn work or yard care*	36	0.0	0.0
Caring for another person*	35	0.0	0.0
Home repairs*	30	0.0	0.0
Heavy housework*	25	1.0	25.0
Light housework*	25	1.0	25.0
Outdoor gardening*	20	0.0	0.0
		Total PASE score	192.4

11.14.3 Accelerometry

An accelerometer (GT3X, Actigraph LLC, Pensacola, FL) was provided to each participant to wear for 7 days between trial visits to assess physical activity levels. The accelerometer was programmed to record biaxial data, begin recording at the end of the participant visit, and finish at the start of their next visit. A researcher demonstrated to participants how to wear the accelerometer around their waist, and were instructed to wear it at all times, except when in water (e.g. swimming or showering) and when in bed. Activity count was calculated using Actilife software (version 6.13.3, Actigraph LLC, Pensacola, FL). The total number of axis counts was divided by total epochs to give a mean daily activity count. If a participant forgot to wear the accelerometer for a day, this day was deleted from the calculation.

12 Experimental Chapter One: Relationship between age and ability to implement thermoregulatory behaviours in thermoneutral and hot ambient conditions

12.1 Introduction

As the increase in heatwave frequency and severity is set to continue (American Meteorological Society, 2012; Met Office, 2018). Couple this with a rise in the number of older adults (Office of National Statistics, 2018), the heat-related mortality and morbidity in older adults is growing and requires attention. Older adults are less able to regulate their body temperature via autonomic thermoregulation than young adults (≤31 years); skin blood flow is diminished by ~40% in adults aged ≥55 years (Kenney, 1988; Kenney et al., 1997) and sweat rate is reduced by ~10-25% in men aged ≥56 years (Inoue et al., 1998; Larose et al., 2013).

These decrements collectively result in a ~60% increase in body heat storage in men and women and are seen as early as the age of 40 years (Larose et al., 2013, 2014; Stapleton et al., 2015b). Men aged ≥40 years had a ~60% greater body heat storage after intermittent cycling in the heat (35°C, ~20% RH) than men aged 20-31 years (Larose et al., 2013). However, in more humid conditions (35°C, 60% RH), the difference in heat storage does not occur until the age of 45 years, and was reduced at ~36% greater heat storage then men aged 20-30 years (Larose et al., 2014). The decrement in heat dissipation occurs at a younger age in dry heat than humid heat. This suggests that sudomotor function declines prior to skin blood flow as humid heat relies less on sweat evaporation for heat dissipation than dry heat. Similar results were found in women after intermittent cycling in hot conditions (40°C, 15% RH), women aged 58 ± 5 years had a ~60% higher level of heat storage than women aged 23 ± 4 years (Stapleton et al., 2015b). These groups were matched for body mass, stature, body surface area, body fat percentage, and $\dot{V}O_2$peak, indicating that this as an age-related decline in heat dissipation rather than a fitness or anthropometric related difference. Autonomic thermoregulatory decrements result in impaired ability to dissipate metabolic heat by middle age in both men and women.

These studies (Larose et al., 2013, 2014; Stapleton et al., 2015b) provide evidence that autonomic thermoregulation is impaired in 40-year-olds compared with those aged 18-31 years. The data from Larose et al. (2013, 2014) appears to show a threshold age for decline in heat storage as all the men aged ≥40 years had a similar level of heat storage. Including men aged 32-39 would allow identification of a threshold age or of a more linear decline in heat dissipation capacity.

Research utilising a regression analysis is required to clarify the relationship between age and thermoregulatory ability. The body heat storage data from Larose et al. (2013) and Larose et al. (2014) suggests that once adults reach 40-45 years there is little further decrement in heat loss capacity as

the 40-70 year-olds had similar levels of heat storage. This is not reflected in the mortality risk during heat waves. A cross-sectional study has shown that the odds ratio of heat-related death increases from age 35 years upwards (Figure 12-1; Berko et al., 2014). This data shows a steady increase in heat mortality risk until the age of 75 years when risk increases faster beyond 75 years (***Figure 12-1.*** *Odds ratio of heat-related mortality in the United States 2006-2010. Data from (Berko et al., 2014).*Figure 12-1). While Berko et al. (2014) established a link between age and heat-related mortality, the underpinning cause is yet to be established. Behavioural thermoregulation may be providing adults aged 35-75 years with protection from heat-related mortality. Investigations into how ageing impacts behavioural thermoregulation will offer insight into how adults voluntarily reduce their heat-related morbidity/mortality risk.

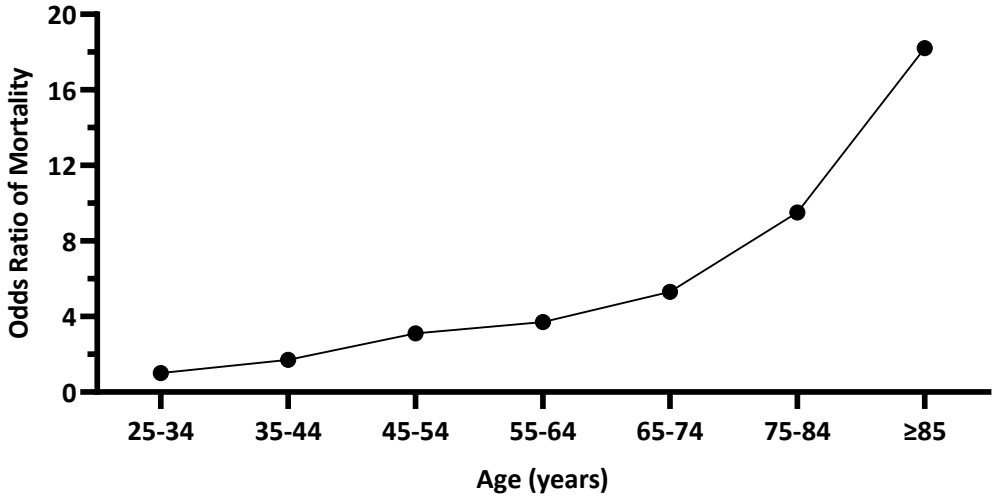

Figure 12-1. *Odds ratio of heat-related mortality in the United States 2006-2010. Data from (Berko et al., 2014).*

Thermoregulatory behaviours, such as reducing exercise intensity, removing clothing layers, or taking cooling showers during heat exposure are important strategies for reducing heat strain and maintaining homeostasis independently of autonomic thermoregulation (Harduar Morano et al., 2016). Under uncompensable heat stress, behavioural alterations are required to regulate body temperature as autonomic thermoregulation (i.e., sweating and skin blood flow) has a limited capacity to regulate body temperature. During exercise, thermoregulatory behaviour can manifest as a voluntary reduction in exercise intensity, lowering heat stress by decreasing metabolic heat production. Young men (23 ± 3 years) complete ~28% less work during a self-paced 30 min cycling time trial in uncompensable conditions (~40°C, ~19% RH) than in compensable conditions (~20°C, ~22% RH; Schlader et al., 2011c). Thermoregulatory behaviours in resting young men are driven by

skin temperature changes (Schlader et al., 2009, 2013; Schlader et al., 2016a; Schlader et al., 2016b) and thermal comfort (Schlader et al., 2011a). Schlader et al. (2009, 2013), and Schlader et al. (2016b) used rectal temperature to measure core temperature and acknowledged its limitation of being slow to react to changing core temperature. However, Schlader et al. (2016a) used telemetric pills to assess core temperature, confirming that young adults can maintain a stable core temperature (~37.0°C) despite fluctuating skin (~32°C to ~34°C) and ambient (~17°C to ~40°C). Behavioural thermoregulation work has focussed on young healthy adults, with little work investigating the impact of ageing on thermoregulatory behaviours in adults aged ≥60 years.

Evidence suggests that thermoregulatory behaviours in those aged >60 years are compromised (Collins et al., 1981; Taylor et al., 1995). Men aged >60 years given a dual position switch that warms or cools a room allow the ambient temperature to fluctuate more than younger (<40 years) men, despite feeling more thermally comfortable throughout the 2 – 2.5 h trial (Collins et al., 1981; Taylor et al., 1995). Older adults appear to be less sensitive to their thermal environment than their younger counterparts when at rest. More recently, work has investigated thermal comfort in adults aged 72 ± 5 years during exercise in a hot environment. Waldock et al. (2018) recruited older (72 ± 5 years old) participants to recumbent cycle at fixed intensity of 6 metabolic equivalents for 30 min in 25°C and 35°C on two separate occasions. Peak rectal temperature was similar between the conditions ([25 v 35°C] 37.6°C v 37.7°C). Peak skin temperature was 3.17°C warmer in the 35°C trial than the 25°C trial (32.9°C v 36.1°C). Despite the elevated skin temperature in the 35°C condition, the participants felt similar levels of thermal comfort (5-point scale) in both trials. This study indicates that older adults reduced sensitivity to the thermal environment is present during exercise as well as at rest. As thermal comfort modifies thermoregulatory behaviour, these results suggest that older adults would be less able to implement thermoregulatory behaviours during heat stress. The use of fixed-intensity exercise allowed for the examination of thermal comfort in different ambient condition. Using a fixed rating of perceived exertion protocol would allow participants to express their self-selected thermoregulatory behaviours. It will also enable the evaluation of how thermal comfort insensitivities identified by Waldock et al. (2018) impacts on thermoregulatory behaviour in older adults.

With ageing there is a decline in autonomic thermoregulatory capacity that causes an increase in heat storage while exercising in hot conditions, however, it is unclear at what age this decline is significant. Thermoregulatory behaviours offer an effective solution to reducing heat strain. Reducing exercise intensity during heat exposure would be a thermoregulatory behaviour that reduces metabolic heat gain and, in turn, heat stress. Young adults can implement these behaviours by responding to changes in thermal comfort and skin temperature. There is evidence that thermal comfort is less sensitive to ambient conditions in older adults. It remains unclear if this insensitivity results in a reduced ability

for older adults to reduce exercise intensity during heat exposure. This study aims to quantify the relationship between age and thermoregulatory behaviours during walking exercise in temperate and hot conditions. We expect that age and thermoregulatory behaviour are negatively related, increased age leads to reduced ability to implement thermoregulatory behaviours. We also predict that this relationship is similar between men and women.

12.2 Methods

12.2.1 Participants

Fifty-nine healthy adult participants aged 19 – 86 years (23 females, 36 males) volunteered to participate in the study. Participants were non-smokers, free from any known respiratory, cardiovascular, or metabolic disease and not taking any medications known to affect thermoregulation (e.g. beta blockers, diuretics, calcium channel blockers; Becker & Stewart, 2011). To eliminate the influence of the menstrual cycle on core temperature only post-menopausal females were included, none where on hormone replacement therapy. Participant anthropometric data is presented in **Error! Reference source not found.**. The experimental protocol was approved by the Plymouth Marjon University Ethics Panel (EP050) in accordance with the Declaration of Helsinki. Volunteers provided written informed consent after explanation of the purpose, benefits, risks, experimental procedures of the study, and were reminded of their right to withdraw without providing an explanation, before participating in the study.

12.2.2 Experimental overview

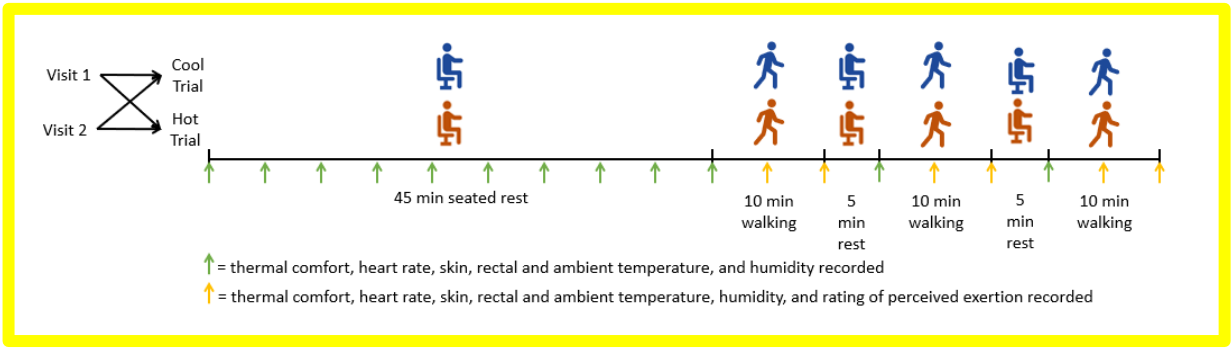

Figure 12-2. *Schematic of protocol for the experimental trials.*

This study utilised a single group comparison of performance, physiological and perceptual responses to exercise between hot and cool conditions. A self-paced, fixed RPE protocol (similar to Tucker et al., 2006) was employed where participants initially completed a familiarisation session to get comfortable with the RPE scale and the motorised treadmill controls. Participants were able to replicate their self-selected speed 4 ± 1 attempts. Instructions were presented orally to participants prior to each session. Participants were instructed to self-select a walking speed that elicited an RPE

of 13 on a 6 -20 point Borg RPE scale (Borg, 1982). They were advised they were free to adjust this walking speed freely to maintain an RPE of 13. Participants were blinded to the speed they selected and received no feedback on how far they had walked, heart rate, rectal temperature, or skin temperature. No verbal encouragement was given to the participants. Anthropometric measures (stature, body mass, and body composition) were also taken during this familiarisation session.

Participants completed two experimental trials (conducted in a randomised counterbalanced order, with at least 48 h between trials), one in 22°C (Control; 21.9 ± 0.6°C, 40 ± 4% relative humidity (RH)), and the other in 35°C (Hot; 34.9 ± 0.3°C, 40 ± 4% RH) to assess how ambient temperature impacts self-paced exercise, and the effect of age on this relationship. Participants conducted their trials at the same time of day to minimise the effects of circadian fluctuations in core temperature. Participants were also asked to abstain from alcohol, caffeine, non-steroidal anti-inflammatory drugs, and strenuous exercise in the 24 h before trials, and consume 500 mL of water the evening before and 2 h before arriving at the laboratory. Clothing consisted of a t-shirt, shorts, and walking/running shoes. Experimental trials were identical apart from the ambient conditions.

During the experimental trials, participants sat at rest in the environmental chamber for 45 minutes. Participants then completed three bouts of 10 min walking (separated by 5 min of seated rest) on a motorised treadmill at a self-selected (but blinded) speed that elicited an RPE of 13. Participants were reminded at the onset of walking, and after 5 min of walking, to self-select a speed that elicited an RPE of 13. Water was available for *ad libitum* consumption throughout the trials.

12.2.3 Experimental procedures

In a preliminary visit, anthropometric measurements (stature, body mass, skinfolds) and physical fitness and activity measurements (6-minute walk test, physical activity scale for the elderly [PASE] questionnaire, accelerometers) were taken as described in the General Methods (Chapter 11.8. Anthropometric Assessment; Chapter 11.14. Physical Fitness and Physical Activity). Participants were then familiarised with the RPE scale. Participants self-selected a treadmill speed that felt 'somewhat hard' (RPE of 13) without being able to see what that speed was. They indicated when they had reached a speed that felt 'somewhat hard' and walked for 3 min. After 3 min, participants confirmed that the speed represented a feeling of 'somewhat hard' and this value was recorded (not revealed to the participant) before they sat at rest for 5 min. Participants repeated this procedure until they were able to replicate the speed that elicited an RPE of 13.

During the experimental trials rectal, skin, and ambient temperature were assessed as described in the General Methods (Chapter 11.9. Temperature). Heart rate and thermal comfort was assessed every 5 min throughout the trials. RPE was assessed every 5 min during the walking exercise.

The distance walked in each of the three exercise bouts was recorded, and then summed to give total distance walked in each trial. A marker of thermoregulatory behaviour was calculated as:

Equation 12-1. *Thermoregulatory behaviour established as a change in distance walked as a function of ambient temperature.*

$$Thermoregulatory\ Behaviour = \frac{Total\ distance\ walked_{Control}}{Total\ distance\ walked_{Hot}}$$

This equation gave a ratio of distance walking in the control trial compared with the hot trial and thus a measure of thermoregulatory behaviour. A result >1 indicated that the participant walked further in the control trial, <1 they walked further in the hot trial, and 1 they walked the same distance in both trials.

12.2.4 Statistical analysis

A multiple regression was run to predict thermoregulatory behaviour from age, stature, body mass, body composition, mean skin and rectal temperature, and mean heart rate and thermal comfort. The amount of variance of thermoregulatory behaviour due to age is presented as R^2. An $R^2 = 1$ demonstrates that 100% of the variance is due to age, while $R^2 = 0$ is interpreted that none of the variance is related to age. Regression data are reported as mean ± 95% confidence interval (CI). Change in rectal temperature from the onset of the trial to the end of the trial was assessed using a paired samples t-test. Mean skin temperatures for all participants were compared between trials using a two-way repeated measures ANOVA. One-way ANOVA was used to compare thermoregulatory behaviour between male participants grouped by age. All descriptive data are presented as mean ± standard deviation (SD). The level of significance for all analyses was set at $p \leq 0.05$. Effect size was defined as small $d = 0.20$, partial $\eta^2 = 0.01$; medium $d = 0.50$, partial $\eta^2 = 0.06$; and large $d = 0.80$, partial $\eta^2 = 0.14$ (Cohen, 1988; Cohen 1969). All statistical analyses were conducted using Statistics Package for the Social Sciences (SPSS v28, IBM Corp, Armonk, NY).

12.3 Results

12.3.1 Participant data and temperature responses

Participants' age and anthropometric data are presented in **Table 12-1**.

Table 12-1*. Age and anthropometric data of all participants. Data presented as mean ± SD.*

Sex	Age (years)	Stature (cm)	Body Mass (kg)	Body Mass Index	ΣSkinfolds (mm)
Male	46 ± 20	177 ± 7	76.4 ± 10.9	24.3 ± 2.9	33.7 ± 10.5
Female	60 ± 8	163 ± 4	67.7 ± 11.4	25.5 ± 4.4	60.6 ± 27.5

A paired samples t-test was run to determine the difference between the control and hot trial in change in rectal temperature from baseline to the end of the trial. There were no outliers, as assessed by inspection of a boxplot for values greater than 3 box lengths from the edge of the box. The difference scores for the control and hot trial were normally distributed, as assessed by Kolmogorov-Smirnov's test (p = 0.200). The hot trial resulted in a 0.45 (95% CI, 0.33 to 0.57) °C greater rise in rectal temperature compared to the control trial, a statistically significant difference, $t(57) = 7.462$, $p < 0.001$, d = 0.980. Data presented in **Figure 12-3**.

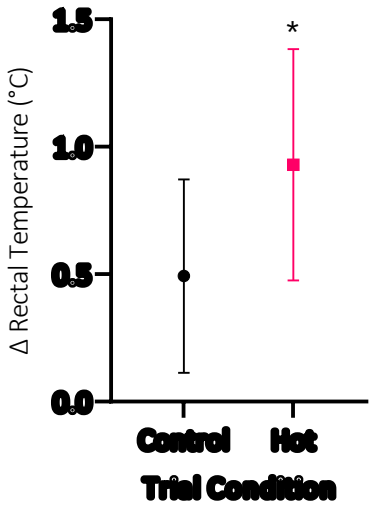

Figure 12-3*. Change in rectal temperature from baseline to end of trial. Data presented as mean ± SD. * denotes significantly different to the control trial, p <0.001.*

Pre-trial, at the onset of exercise and post-trial skin temperature for the control and hot trials are presented in **Figure 12-4**. A two-way repeated measure ANOVA was run to determine the effect of different ambient temperatures over time on skin temperature. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. Skin temperature was normally distributed (p >0.05) except for at the end of the hot trial (p = 0.015), as assessed by Kolmogorov-Smirnov test of normality on the studentised residuals. Skin temperature violated Mauchly's test of Sphericity, $\chi^2(2) = 20.658$, $p < 0.05$, therefore the Greenhouse-Geisser correction was applied. Data are mean ± standard deviation, unless otherwise stated. There was a statistically significant two-way interaction between trial condition and time, $F(1.5, 78.016) = 24.814$, $p < 0.001$, partial $\eta^2 = 0.323$, $\varepsilon = 0.750$. Therefore, simple main effects were examined.

Skin temperature was 4.04 (95% CI, 3.81 to 4.27) °C higher at the start of the hot trial than the control trial, a difference that was statistically significantly, $F(1, 55) = 1208.890$, $p < 0.001$. Skin temperature was 5.41 (95% CI, 5.14 to 5.69) °C higher at the onset of exercise in the hot trial than the control trial,

a difference that was statistically significant, $F(1, 55) = 1580.373$, $p < 0.001$. Skin temperature was 4.21 (95% CI, 3.81 to 4.62) °C higher at the end of the hot trial than the control trial, a difference that was statistically significant, $F(1, 55) = 437.362$, $p < 0.001$.

There was a statistically significant effect of time on skin temperature in the control trial, $F(2, 104) = 27.714$, $p < 0.001$. Skin temperature at the baseline of the control trial and at the onset of exercise time points differed by 0.30 (95% CI, -0.18 to 0.78) °C, a not statistically significant difference, $p = 0.376$. At the end of the control trial skin temperature was higher than the start of the control trial by 1.11 (95% CI, 0.77 to 1.44) °C, a statistically significant difference, $p < 0.001$. At the end of the control trial skin temperature was higher than the onset of exercise in the control trial by 1.40 (95% CI, 0.78 to 2.03) °C, a statistically significant difference, $p < 0.001$.

There was a statistically significant effect of time on skin temperature in the hot trial, $F(2, 112) = 46.452$, $p < 0.001$. Skin temperature was warmer at the onset of exercise than pre-trial in the hot trial by 1.11 (95% CI, 0.80 to 1.42) °C, a statistically significant difference, $p < 0.001$. At the end of the hot trial skin temperature was higher than the start of the hot trial by 1.24 (95% CI, 0.87 to 1.61) °C, a statistically significant difference, $p < 0.001$. Skin temperature at the end of the hot trial compared to at the onset of exercise in the hot trial differed by 0.13 (95% CI, -0.24 to 0.50) °C, a not statistically significant difference, $p = 0.999$.

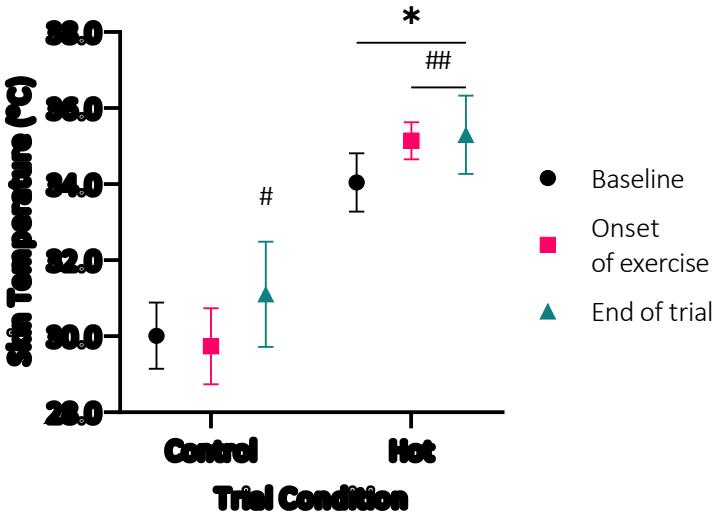

***Figure 12-4**. Skin temperature at baseline, onset of exercise, and end of the trials. Data presented as mean ± SD. 8 denotes significantly warmer than the control trial. # denotes significantly warmer than at baseline and onset of exercise in the control trial. ## denotes significantly warmer than at baseline in the hot trial. p <0.05.*

12.3.2 Influence of age on thermoregulatory behaviour

A multiple regression was run to predict thermoregulatory behaviour from age, stature, body mass, body composition, mean skin and rectal temperature, and mean heart rate and thermal comfort for all participants (men and women). There was linearity as assessed by partial regression plots and a plot of studentised residuals against predicted values. There was independence of residuals, as assessed by a Durbin-Watson statistic of 1.726. There was homoscedasticity, as assessed by visual inspection of a plot of studentised residuals versus unstandardised predicted values. There was no evidence of multicollinearity, as assessed by tolerance values greater than 0.1. There were no studentised deleted residuals greater than ± 3 standard deviations, and no values for Cook's distance above 1. The assumption for normality was met, as assessed by a P-P Plot. The multiple regression model did not significantly predict thermoregulatory behaviour, $F(8, 46) = 1.062$, $p = 0.406$, $R^2 = 0.156$, adjusted $R^2 = 0.009$ (**Figure 12-5**). None of the variables added statistically significantly to the prediction, $p > 0.05$. Regression coefficients and standard errors can be found in

Table 12-2.

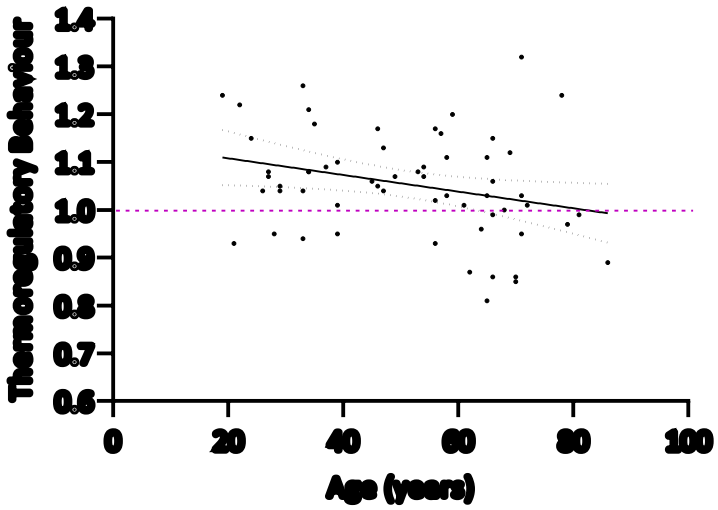

Figure 12-5. *Linear regression of age and thermoregulatory behaviour for all participants. Dotted lines are the 95% CI. Points above the purple line implemented thermoregulatory behaviours by walking further in the control than the hot trial, below the purple line represents walking further in the hot trial than the control trial.*

***Table 12-2**. Regression coefficients of multiple linear regression for all participants. Dependent variable is thermoregulatory behaviour. Model = "Enter" method in SPSS Statistics;* B *= unstandardised regression coefficient; CI = confidence interval; LL = lower limit; UL = upper limit;* SE B *= standard error of coefficient; β = standardised coefficient;* R^2 *= coefficient of determination; Adj.* R^2 *= adjusted* R^2*. Significance set at* $p \leq 0.05$*. n = 55.*

Thermoregulatory behaviour all	*B*	95% CI for *B*		SE *B*	β	*p*	R^2	Adj. R^2
		LL	UL					
Model							0.156	0.009
Constant	2.624	-2.972	8.220	2.780		0.350		
Age	-0.002	-0.004	0.000	0.001	-0.271	0.087		
Stature	0.001	-0.006	0.007	0.003	0.053	0.838		
Body Mass	-0.001	-0.004	0.003	0.002	-0.055	0.788		
Body Composition	0.000	-0.006	0.005	0.003	-0.015	0.948		
Mean Skin Temperature	-0.016	-0.073	0.042	0.029	-0.079	0.590		
Mean Rectal Temperature	-0.028	-0.176	0.120	0.074	-0.059	0.703		
Mean Heart Rate	0.000	-0.003	0.002	0.001	-0.028	0.853		
Mean Thermal Comfort	0.050	-0.003	0.103	0.026	0.269	0.062		

12.3.3 Influence of age on thermoregulatory behaviour: sex differences

A multiple regression was run to predict thermoregulatory behaviour from age, stature, body mass, body composition, mean skin and rectal temperature, and mean heart rate and thermal comfort for the female participants. There was linearity as assessed by partial regression plots and a plot of studentised residuals against predicted values. There was independence of residuals, as assessed by a Durbin-Watson statistic of 1.666. There was homoscedasticity, as assessed by visual inspection of a plot of studentised residuals versus unstandardised predicted values. There was no evidence of multicollinearity, as assessed by tolerance values greater than 0.1. There were no studentised deleted residuals greater than ± 3 standard deviations, and no values for Cook's distance above 1. The assumption for normality was met, as assessed by a P-P Plot. The multiple regression model did not significantly predict thermoregulatory behaviour, $F(8, 14) = 0.394$, $p = 0.906$, $R^2 = 0.184$, adjusted $R^2 = -0.283$ (**Figure 12-6**). None of the variables added statistically significantly to the prediction, $p > 0.05$. Regression coefficients and standard errors can be found in

Table 12-3.

***Table 12-3**. Regression coefficients of multiple linear regression for female participants. Dependent variable is thermoregulatory behaviour. Model = "Enter" method in SPSS Statistics; B = unstandardised regression coefficient; CI = confidence interval; LL = lower limit; UL = upper limit; SE B = standard error of coefficient; β = standardised coefficient; R^2 = coefficient of determination; Adj. R^2 = adjusted R^2. Significance set at $p \leq 0.05$. n = 23.*

Thermoregulatory behaviour women	*B*	95% CI for *B*		SE *B*	β	*p*	R^2	Adj. R^2
		LL	UL					
Model							0.184	-0.283
Constant	2881	-7.540	13.302	4.859		0.563		
Age	0.002	-0.006	0.010	0.004	0.119	0.674		
Stature	-0.003	-0.015	0.009	0.006	-0.228	0.564		
Body Mass	0.003	-0.005	0.010	0.004	0.251	0.512		
Body Composition	-0.008	-0.020	0.005	0.006	-0.515	0.214		
Mean Skin Temperature	-0.030	-0.182	0.121	0.070	-0.115	0.672		
Mean Rectal Temperature	-0.007	-0.288	0.274	0.131	-0.013	0.960		
Mean Heart Rate	0.000	-0.006	0.005	0.003	-0.023	0.934		
Mean Thermal Comfort	0.035	-0.069	0.138	0.048	0.178	0.485		

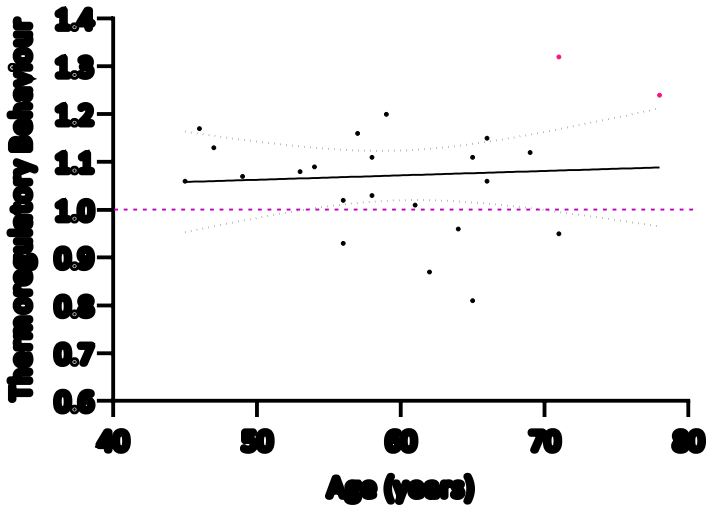

***Figure 12-6**. Linear regression of age and thermoregulatory behaviour for the female participants. Dotted lines are the 95% CI. Points above the purple line implemented thermoregulatory behaviours by walking further in the control than the hot trial, below the purple line represents walking further in the hot trial than the control trial. Pink data points show two anomalous participants (see: 12.4.1 Female participants).*

A multiple regression was run to predict thermoregulatory behaviour from age, stature, body mass, body composition, mean skin and rectal temperature, and mean heart rate and thermal comfort for the male participants. There was linearity as assessed by partial regression plots and a plot of studentised residuals against predicted values. There was independence of residuals, as assessed by a Durbin-Watson statistic of 2.217. There was homoscedasticity, as assessed by visual inspection of a

plot of studentised residuals versus unstandardised predicted values. There was no evidence of multicollinearity, as assessed by tolerance values greater than 0.1. There were no studentised deleted residuals greater than ± 3 standard deviations, and no values for Cook's distance above 1. The assumption for normality was met, as assessed by a P-P Plot. The multiple regression model did significantly predict thermoregulatory behaviour, $F(8, 23) = 2.540$, $p = 0.038$, $R^2 = 0.469$, adjusted $R^2 = 0.284$ (**Figure 12-7**). Only age added statistically significantly to the prediction, $p = 0.005$. Regression coefficients and standard errors can be found in **Table 12-4**

***Table 12-4**. Regression coefficients of multiple linear regression for male participants. Dependent variable is thermoregulatory behaviour. Model = "Enter" method in SPSS Statistics; B = unstandardised regression coefficient; CI = confidence interval; LL = lower limit; UL = upper limit; SE B = standard error of coefficient; β = standardised coefficient; R^2 = coefficient of determination; Adj. R^2 = adjusted R^2. Significance set at $p \leq 0.05$. n = 32.*

Thermoregulatory behaviour men	*B*	95% CI for *B*		SE *B*	β	*p*	R^2	Adj. R^2
		LL	UL					
Model							0.469	0.284
Constant	3.939	-2800	10.678	3.258		0.239		
Age	-0.003	-0.005	-0.001	0.001	-0.528	0.005		
Stature	0.002	-0.006	0.010	0.004	0.133	0.611		
Body Mass	-0.001	-0.006	0.005	0.003	-0.076	0.792		
Body Composition	-0.005	-0.014	0.005	0.005	-0.219	0.328		
Mean Skin Temperature	-0.029	-0.089	0.031	0.029	-0.173	0.330		
Mean Rectal Temperature	-0.049	-0.224	0.126	0.085	-0.103	0.568		
Mean Heart Rate	-0.002	-0.004	0.001	0.001	-0.224	0.257		
Mean Thermal Comfort	0.037	-0.029	0.102	0.032	0.196	0.258		

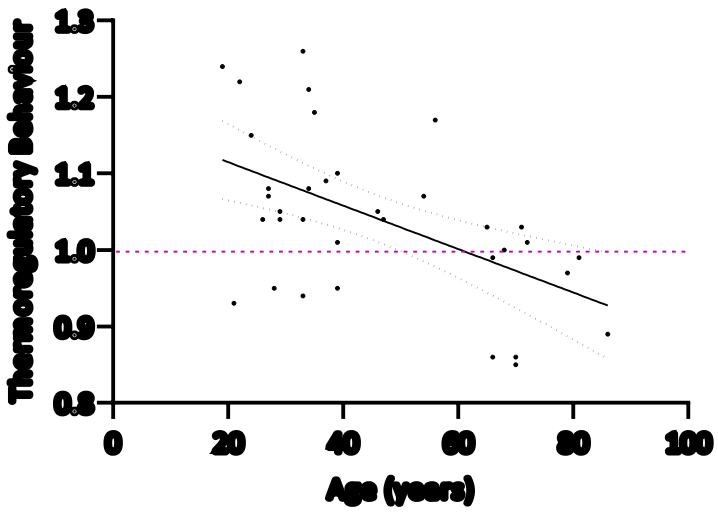

***Figure 12-7**. Linear regression of age and thermoregulatory behaviour for the male participants. Dotted lines are the 95% CI. Points above the purple line implemented thermoregulatory behaviours by walking further in the control than the hot trial, below the purple line represents walking further in the hot trial than the control trial.*

An independent samples t-test was run to determine if mean skin temperature throughout the hot trial differed between the male and female participants. Data are mean ± standard deviation, unless otherwise stated. There were 35 male and 23 female participants. There were no outliers in the data, as assessed by inspection of a boxplot. Thermoregulatory behaviour scores were normally distributed for the women, as assessed by Shapiro-Wilk's test (p >0.05). Thermoregulatory behaviour scores for the men were not normally distributed (p = 0.014). As the independent t-test is robust, analysis continued. There was homogeneity of variance, as assessed by Levene's test for equality of variances (p = 0.398). There was no statistically significant difference between the male and female participants, $t(56) = 0.180$, $p = 0.858$, $d = 0.05$.

A one-way ANOVA was conducted to determine if the ability to implement thermoregulatory behaviours was different for different age groups of men. Participants were classified into three groups: young (18-30 years old, n = 10), middle (31-60 years old, n = 15), and older (>60 years old, n = 11). There were no outliers, as assessed by boxplot; data was normally distributed for the young and middle groups as assessed by Shapiro-Wilk test (p >0.05), older group was not normally distributed (Shapiro-Wilk p = 0.024); and there was homogeneity of variances, as assessed by Levene's test of homogeneity of variances (p = 0.846). Data is presented as mean ± standard deviation. Thermoregulatory behaviour score was significantly difference between age groups, $F(2, 33) = 8.105$, $p = 0.001$, partial $\eta^2 = 0.480$ (**Figure 12-8**). The older group had the lowest thermoregulatory behaviour score (0.95 ± 0.07), the young (1.08 ± 0.10) and middle (1.08 ± 0.09) groups had similar thermoregulatory behaviour scores. Tukey post hoc analysis revealed that the older group's

thermoregulatory behaviour score was statistically significantly lower than the young (mean difference -0.124, 95% CI (-0.219 to -0.046) p = 0.008) and middle groups (mean difference -0.132, 95% CI (-0.218 to -0.046) p = 0.002). There was no statistically significant group difference between the young and middle groups.

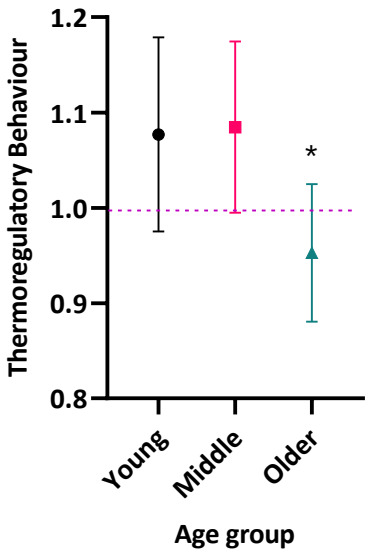

Figure 12-8. *Thermoregulatory behaviour of men by age group. Data presented as mean ± SD. * denotes statistically significantly different from the young and middle groups, p <0.05. Points above the purple line implemented thermoregulatory behaviours by walking further in the control than the hot trial, below the purple line represents walking further in the hot trial than the control trial.*

12.4 Discussion

When examining all participants, the linear regression model did not identify a significant relationship between age and thermoregulatory behaviour (via pacing strategy) (adjusted R^2 = 0.009, p = 0.406). Notably, when the participants were separated by sex the linear model identified a significant inverse relationship between age and thermoregulatory behaviour for the men (adjusted R^2 = 0.284, p = 0.038), with age being a significant factor (p = 0.005), but not for the women (adjusted R^2 = -0.283, p = 0.906). While the men had a near-linear decline in thermoregulatory behaviour with increasing age, the women's thermoregulatory behaviour declined between the ages of 45 and 60 years before beginning to improve again from 60 to 78 years. Men aged ≥61 years did not implement thermoregulatory behaviours as effectively as men aged 18-30 years when exercising in the heat (**Figure 12-8**). This is the first study to evaluate a wide range of ages when examining thermoregulatory behaviour during exercise in hot conditions.

The use of a fixed RPE approach with older participants aged >60 years was novel. This allowed for the examination of the relationship between age and thermoregulatory behaviour while exercising in the heat. This study demonstrates that older men are less inclined to self-regulate behaviour sufficiently to reduce exercise intensity when undertaking 30 min of walking exercise in the heat (**Figure 12-7**). The increase in rectal temperature from baseline to the end of the trials in the present study demonstrates that participants did experience heat strain. Rectal temperature increased significantly during the control trial by 0.5°C and by 0.9°C in the hot trial. Rectal temperature at the end of the trials was significantly warmer in the hot trial than the control trial. Despite this rise in rectal temperature, older adults were less likely than younger adults to reduce their walking speed in the heat.

The influence of skin temperature on thermoregulatory behaviour has been demonstrated both at rest (Schlader et al., 2009) and during exercise (Schlader et al., 2011b). A skin temperature increase of ~5.8°C results in 2.4% decrease in cycling power output in young healthy men (Schlader et al., 2011b). When resting for 60 min in 32°C young (~22 years) adults pushed a button that initiates neck cooling ~100 times, in 42°C conditions this increased to ~1,000 times when mean skin temperature was ~1.8°C warmer than in the 32°C (Vargas et al., 2018b). Otani et al. (2019) saw an ~8% decline in power output with a skin temperature increase of ~0.5°C during fixed intensity cycling. The ~5°C warmer skin temperature at the onset of exercise in the hot compared to the control trial in the present study elicited a thermoregulatory behavioural response in young healthy adults, however, the older men (18-30 v ≥61 years, p = 0.008. 31-60 v ≥61 years, p = 0.002) were less likely to voluntarily reduce their exercise intensity in the heat. This suggests that the older men were less sensitive to the warmer skin temperature and decreasing the inclination to reduce walking speed in the hot trial.

Previous work has demonstrated that sensitivity to thermal stimulus applied to the skin declines with age, particularly for hot stimuli (Guergova & Dufour, 2011). When a thermode applied to the skin is heated, adults aged >60 years detect the increase later than adults aged ≤50 years (Dufour & Candas, 2007). This reduced sensitivity to thermal stimuli in the skin suggests older adults may require greater increases in skin temperature than younger adults before they implement thermoregulatory behaviours during exercise in the heat. The present study suggests that the reduced sensitivity to thermal stimulus results in a reduced ability to implement thermoregulatory behaviours during heat exposure, so older men may require a greater increase in skin temperature to implement cooling behaviours. Impaired implementation of thermoregulatory behaviours during heat wave events increases the likelihood of heat-related illness and death. For example, during the 2003 European heat wave those who did not leave home had a ~7 times greater risk of death than those who visited cool places (Vandentorren et al., 2006). If older adults delay using cooling behaviours during heat exposure events it poses a risk to their health and the resources of the healthcare system. The relationship

between skin temperature and thermoregulatory behaviour during exercise in older adults warrants further investigation.

In the present study, the relationship between age and thermoregulatory behaviour appears to differ between men and women. The men displayed a near linear decline in thermoregulatory behaviour as age increased, the women displayed a near U-shaped relationship where thermoregulatory behaviour declined with age before improving again at around age 60 years. Two women, aged 71 and 78 years, implemented thermoregulatory behaviour particularly well, this may have influenced the results (**Figure 12-6**). See below for further analysis and discussion [12.4.1 Female participants]). Analysis of mean skin temperature, a driver of thermoregulatory behaviour, during the hot trial revealed no significant difference between the sexes, thus could not explain the difference in thermoregulatory behaviour observed between the men and women. Further examination of thermoregulatory behaviour differences between men and women is warranted.

Skin temperature for the men was 5.2°C warmer in the hot trial than the control trial, and 6.1°C warmer for the women. Young adults reduce exercise intensity when exposed to a ~5.8°C increase in skin temperature (Schlader et al., 2011b), thus the skin temperature increase experienced during the present study should have elicited a behavioural response to the heat stress. While the women responded behaviourally to this increased skin temperature, the men did not reduce their exercise intensity. This outcome indicates that women are more sensitive to increases in skin temperature, and men need a greater stimulus before they implement behavioural strategies during exercise in hot conditions.

It is well known that having a low level of physical fitness increases the risk of exertional heat illness (Westwood et al., 2020) and sustaining a high level of aerobic fitness attenuates the age-related decline in thermoregulatory ability (Notley et al., 2020). Lack of mobility also increases the risk of death during heat waves (Vandentorren et al., 2006). Thus, physical activity markers were assessed to see if they could explain any differences in thermoregulatory behaviours. There was no difference in the physical fitness or activity markers between the men and the women aged ≥45 years (**Error! Reference source not found.**) in the present study. It remains unclear why men are less able, or less inclined to implement thermoregulatory behaviours. These findings concur with recent work that showed women implement thermoregulatory behaviours more readily than men, despite having similar core temperature and mean skin temperature (Vargas et al., 2019b). The findings from Vargas et al. (2019b) and the present study suggest that older men are less sensitive to changes in skin temperature when implementing behavioural thermoregulation in the heat. Further work is required

to understand if men need a greater heat stimulus to elicit a behavioural response during heat exposure.

As the relationship between age and thermoregulatory behaviour differs between the men and the women, further analysis was conducted on the male response. To get a greater understanding of when the decline in thermoregulatory behaviour begins, the men were divided into age groups approximately by young, middle-aged, and older men. The older men did not implement thermoregulatory behaviours as effectively as the young men. The middle-aged men did not differ significantly from either the young or older men, but their median thermoregulatory behaviour was between the young and older men (young 1.08, middle-aged 1.06, and older 0.99). This result suggests that the decline in behavioural thermoregulation in men begins prior to age 60 but is not significant until over that. Future work investigating ageing and behavioural thermoregulation using group comparisons should consider men aged >60 years as an older age group.

Further analysis of the women could not be conducted as there were no women <40 years, and thus no control group of young women. Core temperature fluctuates throughout the menstrual cycle with resting core temperature being ~0.4°C warmer in the luteal phase than the follicular phase (Hessemer & Bruck, 1985; Kolka & Stephenson, 1989; Stephenson & Kolka, 1985). Thermoregulation also alters throughout the menstrual cycle, the core temperature threshold for the onset of sweating and skin blood flow while cycling in 35°C is ~0.3-0.5°C higher during the luteal phase than the follicular phase (Kolka & Stephenson, 1989; Stephenson & Kolka, 1985). So, the present study did not include pre-menopausal women to prevent the menstrual cycle fluctuations from influencing the results.

12.4.1 Female participants

As identified in **Figure 12-6**, two of the female participants implemented thermoregulatory behaviours particularly well despite being two of the oldest (71 and 78 years) female participants in the study. Further examination of the relationship between thermoregulatory behaviour and ageing in the female group was deemed necessary to establish if there was cause to remove these two participants from the analysis. An independent samples t-test was run to see if the anomalous participants had a different thermoregulatory behaviour score to the main group of female participants. Data are presented as mean ± standard deviation (**Figure 12-9**), unless otherwise stated. There were 2 anomalous participants and 21 female participants in the main group. There were no outliers in the data, as assessed by inspection of a boxplot. Thermoregulatory behaviour scores were normally distributed, as assessed by Shapiro-Wilk's test (p >0.05), and there was homogeneity of variance, as assessed by Levene's test for equality of variances (p = 0.375). The anomalous women (1.28 ± 0.06)

had a higher thermoregulatory behaviour score than the main group (1.05 ± 0.10), this was statistically significant, $t(21) = 3.089$, $p = 0.006$, $d = 0.100$.

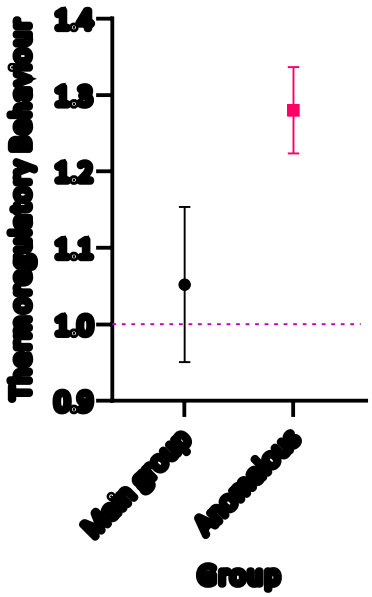

***Figure 12-9**. Thermoregulatory behaviour of main group of female participants and the two anomalous female participants. Data presented as mean ± SD. The purple dotted line represents walking the same distance in both trials. Points above the purple dotted line represent participants who walked further in the control trial than the hot trial, points below represent participants who walked further in the hot trial than the control trial.*

Anthropometric and physical fitness and activity data of the anomalous participants and the other female participants is presented in **Table 12-5**. The anomalous participants were older, had a higher body mass, BMI, sum of skinfolds, body fat %, and were less physically active than the rest of the participants. This result is unexpected as increased age (Centers for Disease Control and Prevention, 2016; Vanhems et al., 2003), high body mass and BMI are known risk factors for heat illness (Cleary, 2007; Donoghue & Bates, 2000).

Despite implementing better thermoregulatory behaviour, in the hot trial the anomalous group had a similar rectal temperature at the end of the trial (~38.0°C) indicating that the behaviour they implemented was insufficient to reduce heat strain significantly. Mean heart rate (~109 b·min^{-1}) was also all similar throughout the hot trial (Table 12-6). Skin temperature at the onset of exercise is a driver for implementing thermoregulatory behaviours (Schlader et al., 2011b), however, the skin temperature was similar between the anomalous pair and the rest of the female participants at ~35.2°C; (**Table 12-6**).

Table 12-5. *Anthropometric, fitness, and physical activity characteristics of the female participant's separated into the anomalous pair and the rest of the group. Data shown as mean ± SD. 6MWD = total distance covered in the 6 min walk test. PASE = Physical activity scale for the elderly*

Group	n	Age (years)	Height (cm)	Body Mass (kg)
Anomalous	2	75 ± 3.5	164 ± 0	83.4 ± 12.6
Rest of group	21	58 ± 7.4	163 ± 4	66.2 ± 10.0
	BMI ($kg \cdot m^2$)	∑Skinfolds (mm	Body Fat %	6MWD (m)
Anomalous	31 ± 4.6	96 ± 33	41 ± 5	469 ± 0
Rest of group	25 ± 4.0	57 ± 24	34 ± 5	614 ± 38

Table 12-6. *Physiological and perception data from the hot trial for the anomalous participants and the rest of the women. Data shown as mean ± SD.*

Group	n	Heart Rate ($b \cdot min^{-1}$)	Final Rectal Temperature (°C)	Skin Temperature Onset of Exercise (°C)	Mean Thermal Comfort
Anomalous	2	109 ± 10	38.06 ± 0.02	35.20 ± 0.09	1.8 ± 0.6
Rest of Group	21	109 ± 14	38.04 ± 0.37	35.19 ± 0.37	1.7 ± 0.6

Skin temperature at the onset of exercise was similar between the groups (~35.2°C), thus it is expected that thermal comfort would also be similar as skin temperature mediates thermal comfort (Schlader et al., 2011a; Schlader & Vargas, 2019). However, the anomalous pair felt slightly more thermally uncomfortable (1.8 v 1.7). This greater sensitivity to increased skin temperature resulting in a greater feeling of warmth might explain why the anomalous pair implemented thermoregulatory behaviours more effectively than expected.

Further details on the background of the participants (e.g., history of athletic training, living conditions) was not collected so cannot be investigated here. All participants for this study were recruited from the local population around Plymouth, UK. Anecdotally, many of the participants involved in this study were physically and socially active individuals with a keen interest in health and fitness. The results from this study may not represent a less physically active population.

As removing the anomalous women from the analysis did not remove the sex-related difference in the relationship between age and thermoregulatory behaviour, future work should separate the two sexes. Investigations should also focus on factors regulating the difference in this relationship. Only three of the female participants in this study were aged >70 years, two of them being the anomalous

pair. Therefore, there is an insufficient number of female participants aged >70 years to confidently conclude that thermoregulatory behaviour plateaus or increases above the age of 70 years. Including a sample group of women in their 70s and older may reveal that the decline in thermoregulatory behaviour is similar between the sexes. Recruitment of women was challenging for this study.

Moving forward this thesis will investigate the factors that are influencing thermoregulatory behaviours in men. This is the area that this thesis can have the greatest impact on understanding how to reduce heat-related morbidity and mortality. The data in the present study clearly demonstrate a decline in ability to implement thermoregulatory behaviours in men aged >60 years. This effect will put them at an increased risk of heat-related illness and death during heat wave events and poses a public health risk during these hot weather events.

12.5 Limitations

There are some limitations for the current study. Participants were all healthy, and had to fulfil inclusion criteria, including being free from cardiovascular disease and diabetes, as these are known heat illness risk factors (Åström et al., 2011; Semenza et al., 1996). Omitting participants with cardiovascular disease and diabetes will reduce the transferability of the results to these disease populations. Only post-menopausal women (not taking HRT) were included in the study to eliminate any hormonal influence on resting core temperature in women still experiencing their menstrual cycle. Future work should include pre-menopausal women to elucidate if these behavioural thermoregulatory responses are consistent across men and women of all ages. Participants adjusted their behaviour via the controls on a motorised treadmill, and for many participants using a treadmill was a novel or unusual experience. However, the preliminary visit was used to familiarise participants with the equipment and remove this novelty. Participants were able to repeat their walking speed within 4 ± 1 attempts during the familiarisation visit. Unlike previous work (Larose et al., 2014; Waldock et al., 2018), we used walking exercise instead of cycling, to increase the external validity of the study. Cycling exercise is novel to many, only 11% of adults in the UK make at least one cycling journey a week, however, 60% make at least one walking journey a week (Department for Transport, 2019).

12.6 Conclusion

Behavioural thermoregulation is somewhat impaired with age when undertaking walking exercise at a perceived exertion of 'somewhat hard' in hot ambient conditions (35°C, 40% RH). This confirms our hypothesis that age and thermoregulatory behaviour are negatively correlated. Our second hypothesis that this decline in thermoregulatory behaviour would be similar between men and women requires further investigation. The decline in thermoregulatory behaviours in women

appeared to plateau at ~65 years of age. A greater sample of women aged ≥70 years is required to elucidate this finding. The impairment in implementing behavioural changes when exercising in the heat is particularly evident in males aged >60 years. Older adults are less likely to initiate behaviours that reduce heat and physiological stress, such as reducing exercise intensity, during heat exposure. This scenario poses a public health risk during heatwave events as older adults are less able to reduce their heat exposure than younger adults. The next study in this thesis will look to further the understanding of the physiological and perceptual mechanisms underpinning the impairment in thermoregulatory behaviours in older men.

13 Experimental Chapter Two: Physiological and perceptual factors influencing behavioural thermoregulation in young and older men in thermoneutral and hot ambient conditions

13.1 Introduction

Behavioural adjustments offer an almost unlimited capacity to reduce heat stress (Benzinger, 1969). During exercise in the heat, adjusting behaviour to reduce exercise intensity, thus reducing metabolic heat load and heat stress is a thermoregulatory behaviour utilised by young healthy adults (Schlader et al., 2011c). However, the evidence from Experimental Chapter One in this thesis, indicates that older adults, particularly men, are less inclined to implement behavioural strategies during heat stress. Therefore, older men are at an increased risk of heat illness during heat wave events as they are unwilling to voluntarily reduce their exercise intensity in hot conditions. The present study will look to identify what factors may be causing this decline in thermoregulatory behaviours in older men.

As skin temperature is on the periphery it is more sensitive to changes in ambient temperature than core temperature. Transient receptor potential vanilloid channels are activated by heat (Caterina, 2007) causing thermoreceptors in the skin to signal the thermoregulatory centres in the hypothalamus to a change in the ambient conditions and peripheral body temperatures before core temperature is influenced. This makes skin temperature an effective driver of thermoregulatory behaviours (Schlader et al., 2009, 2013). Changes in skin temperature drive young men to voluntarily implement thermoregulatory behaviours at rest to protect core temperature (Schlader et al., 2009, 2013). In a hot environment, skin temperature rises immediately prior to thermoregulatory behaviour being implemented and is coupled with an increase in cutaneous vasodilation (Schlader et al., 2016a). Skin temperature also mediates thermoregulatory behaviours at the onset of self-paced exercise. In young, healthy males, total work completed in a 60 min cycling time trial is ~2.4% less when skin temperature is 35.2°C at the onset of exercise compared to when skin temperature is at 29.4°C (Schlader et al., 2011b). After 8 h of seated rest in a steady ambient temperature of 21.5°C or a transient temperature between 17 and 25°C, the mean skin temperature of older (67-73 years) men is ~0.5°C cooler than young (22-25 years) men (Schellen et al., 2010). This cooler skin temperature may negatively impact the behavioural response to heat stress in older adults as they are not receiving as great a stimulus from the skin as younger adults. Thus, investigations into how skin temperature influences thermoregulatory behaviour in older adults during heat stress are needed.

Thermal comfort occurs when there is an indifference to the thermal environment (IUPS Thermal Commission, 2001). While thermal comfort is experienced, thermoregulatory behaviours will not be initiated. Thermal comfort is influenced by skin temperature (Schellen et al., 2010) and so skin temperature drives thermoregulatory behaviours through thermal comfort (Schlader et al., 2011a;

Schlader & Vargas, 2019). Thermoregulatory behaviours are initiated by young (23 ± 1 year) men during self-paced cycling at a fixed intensity (RPE 16) when thermal discomfort is experienced, regardless of skin temperature (Schlader et al., 2011a). Young (22-25 years) men felt significantly more comfortable while resting in 21.5°C than older (67-73 years) men who preferred a warmer ambient temperature (Schellen et al., 2010). During cycling exercise at 6 METs adults aged ~70 years felt no difference in thermal comfort between 25°C and 35°C, despite skin temperature being ~3.2°C warmer in the 35°C condition (Waldock et al., 2018). These data suggest that older adults prefer warmer environmental conditions and are less sensitive to increases in skin temperature. This could result in a diminished ability to implement thermoregulatory behaviours during heat stress.

In Experimental Chapter One, adults could voluntarily implement thermoregulatory behaviours by controlling their exercise intensity. Men aged ≥61 years were less inclined to voluntarily reduce exercise intensity during heat stress as men aged 18-30 years. The older men in Experimental Chapter One were healthy, normotensive, free from diabetes and cardiovascular disease, and not taking any medications known to influence thermoregulation, yet they did not reduce walking speed as effectively in the heat as the young men. Work by Schellen et al. (2010), and Waldock et al. (2018) that identified an altered thermal comfort in older adults indicates that the reduced ability to implement thermoregulatory behaviours observed in Experimental Chapter One could be the result of a preference for warmer ambient temperatures in older men. Further investigations are needed to elucidate the impact of skin temperature and thermal comfort on behavioural thermoregulation in older men while exercising in the heat.

Vasodilation, behavioural changes, and sweating all contribute to defending core temperature during heat stress (Parsons, 2014). These are implemented in a graded response to conserve energy, changes in vasomotor tone precede thermoregulatory behaviours, behaviours are then initiated, minimising the sweat response (Schlader et al., 2013). This sequence of events allows the body to preserve energy while defending core temperature. For young (26 ± 4 years) adults resting in 40°C, in the 90 sec prior to the initiation of thermoregulatory behaviour forearm skin temperature rises by 0.2°C, and mean arterial pressure is 5 mmHg lower than thermoneutral baseline conditions as a result of changing cutaneous vasomotor tone (Schlader et al., 2016b). This work in young healthy adults demonstrates that the cardiovascular system must adjust during heat exposure, and these adjustments occur before thermoregulatory behaviours. Due to ageing, the cardiovascular system of older adults does not function as effectively as in young adults, with impairments including reduced maximal heart rate, reduced maximal cardiac output, and endothelial dysfunction (Karavidas et al., 2010). Increasing age also results in increased blood pressure (Hart et al., 2012), and reduced elasticity of the blood vessels (Lee & Park, 2013). Therefore, the reduction in mean arterial pressure that young adults experience

before implementing thermoregulatory behaviours may be blunted or delayed in older adults, resulting in a delay in initiating thermoregulatory behaviour. There is a need for research investigating the interaction of cardiovascular and behavioural responses to heat strain in older adults.

The aim of the study was to identify factors that influence this diminished ability to implement thermoregulatory behaviour during ambient heat stress that was identified in Experimental Chapter One. This will require the addition of cardiovascular and sudomotor function markers. We expect to find that men aged ≥60 years will have a lower skin temperature and will feel more comfortable during heat exposure than men aged <35 years. The findings from this study could help to guide recommendations for older men during heat stress. We expect that the older men will not implement thermoregulatory behaviours as effectively as the young men, older men will feel more thermally comfortable than the young men, and the older men will experience a greater level of heat strain than the younger men.

13.2 Methods

13.2.1 Participants

Twenty-two healthy male participants volunteered to participate in the study, 11 young (26 ± 4 years; mean ± SD) and 11 older (72 ± 7 years). Participants were non-smokers, free from any known respiratory, cardiovascular, or metabolic disease and not taking any medications known to affect thermoregulation (e.g. beta blockers, diuretics, calcium channel blockers; Becker & Stewart, 2011). Participant anthropometric data is presented in **Error! Reference source not found.**. The experimental protocol was approved by the Plymouth Marjon University Ethics Panel (EP050) in accordance with the Declaration of Helsinki. Volunteers provided written informed consent after explanation of the purpose, benefits, risks, experimental procedures of the study, and were reminded of their right to withdraw without providing an explanation, before participating in the study.

13.2.2 Experimental overview

This study utilised a group comparison of performance, physiological and perceptual responses to walking exercise in hot and thermoneutral conditions. A self-paced, fixed RPE protocol was employed where participants initially completed a familiarisation session to get comfortable with the RPE scale and the motorised treadmill controls. Anthropometric measures (stature, body mass, and body composition) were also taken during this familiarisation session.

Participants completed two experimental trials (conducted in a randomised counterbalanced order, with at least 48 h between trials), one in 22°C (Control; 22.1 ± 0.3°C, 41 ± 4% relative humidity (RH)) and the other in 35°C (Hot; 34.9 ± 0.2°C, 40 ± 4% RH). Participants sat at rest in the environmental

chamber for 45 min. Participants then walked on a motorised treadmill at a self-selected (but blinded) speed that elicited an RPE of 13 'somewhat hard: scale 6-20' for three bouts of 10 min separated by five min of seated rest (Figure 13-1). These assessed how ambient temperature impacts self-paced exercise, skin and rectal temperatures, cardiovascular and perceptual responses, and the effect of age on these responses. Trials were conducted at the same time of day to minimise the effects of circadian fluctuations in core temperature. Participants were also asked to abstain from alcohol, caffeine, non-steroidal anti-inflammatory drugs, and strenuous exercise in the 24 h before trials, and consume 500 mL of water the evening before and 2 h before arriving at the laboratory, as per guidelines from the American College of Sports Medicine (Convertino et al., 1996). Clothing consisted of a t-shirt, shorts, and walking/running shoes. Experimental trials were identical apart from the ambient conditions. Water was available for *ad libitum* consumption throughout the trials.

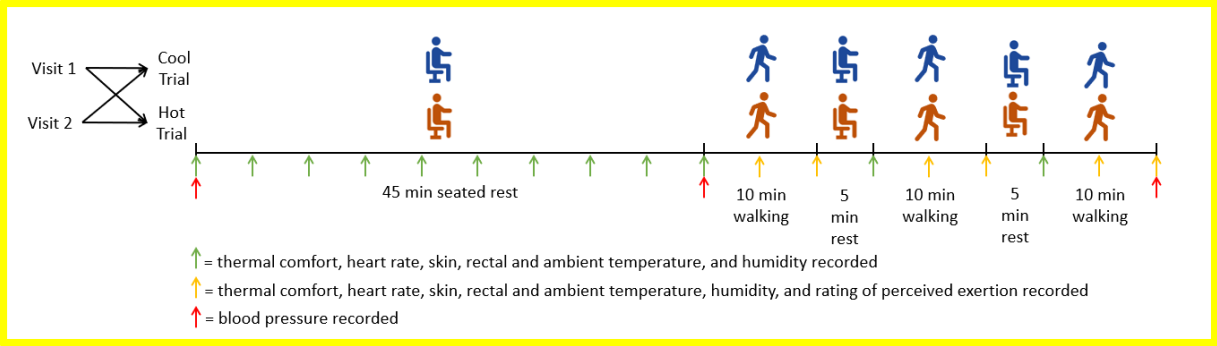

Figure 13-1. *Schematic of protocol for the experimental trials.*

13.2.3 Experimental procedures

In a preliminary visit, anthropometric measurements (stature, body mass, skinfolds) and physical fitness and activity measurements (6-min walk test and physical activity scale for the elderly [PASE] questionnaire) were taken as described in the General Methods (Chapter 11.8 Anthropometric Assessment. Chapter 11.14 Physical Fitness and Physical Activity). Participants were then familiarised with the RPE scale. Participants were asked to self-select a speed that elicited an RPE of 13, 'somewhat hard' while blinded to the speed. They then indicated when they had reached a speed that felt 'somewhat hard' and walked for 3 min. After 3 min, participants confirmed that the speed represented a feeling of 'somewhat hard' and the value was recorded (not revealed to the participant) before they sat at rest for 5 min. Participants repeated this procedure until they were able to replicate the speed that elicited an RPE of 13.

During the experimental trials rectal, skin and ambient temperature were assessed as described in the general methods. Heart rate and thermal comfort was assessed every five min throughout the trials. RPE was assessed every 5 min during the walking exercise. Blood pressure was assessed, as outlined

in the General Methods (Chapter 11.12 Blood Pressure), at the beginning of the trial, at the end of the rest period, and after the final exercise bout.

The distance walked in each exercise bout was recorded, these were summed to give total distance walked in each trial. A marker of thermoregulatory behaviour was calculated as:

Equation 13-1. *Thermoregulatory behaviour established as a change in distance walked as a function of ambient temperature.*

$$Thermoregulatory\ Behaviour = \frac{Total\ distance\ walked_{Control}}{Total\ distance\ walked_{Hot}}$$

This index gave a ratio of distance walking in the control trial compared with the hot trial and thus a measure of thermoregulatory behaviour. A result >1 indicated that the participant walked further in the control trial, <1 they walked further in the hot trial, and =1 they walked the same distance in both trials.

13.2.4 Statistical analysis

Data is presented as mean ± standard deviation, unless otherwise stated. The level of significance for all analyses was set at $p \leq 0.05$. Effect size was defined as small $d = 0.20$, partial $\eta^2 = 0.01$; medium $d = 0.50$, partial $\eta^2 = 0.06$; and large $d = 0.80$, partial $\eta^2 = 0.14$ (Cohen, 1988; Cohen 1969). All statistical analyses were conducted using Statistics Package for the Social Sciences (SPSS v28, IBM Corp, Armonk, NY).

13.3 Results

13.3.1 Participant characteristics and temperature responses

Participant anthropometric and physical activity data are presented in **Table 13-1**.

Table 13-1*. Participant anthropometric and physical activity data. Data presented as mean ± SD.* ***Bold*** *indicates significantly different to the young group, p < 0.001.*

Group	n	Age (years)	Stature (cm)	Body Mass (kg)	Body Mass Index (kg·m^2)	ΣSkinfolds (mm)	6MWT (m)	PASE Score
Young	11	26 ± 4	180 ± 7	74.5 ± 9.6	22.9 ± 1.7	30.1 ± 7.2	660 ± 89	259 ± 149
Older	11	**72 ± 7**	175 ± 8	72.5 ± 11.1	23.7 ± 3.1	34.7 ± 11.0	657 ± 91	199 ± 86

By design, the ambient temperature differed, and the relative humidity was similar between the trials. The ambient temperature during the control trial was 22.1 ± 0.3°C, and in the hot trial 34.9 ± 0.2°C

(mean ± SD. p <0.001). Relative humidity was similar between the trials at 41 ± 4% for the control trial, and 40 ± 4% for the hot trial (mean ± SD. p = 0.258).

A two-way ANOVA was conducted to examine the effects of trial condition and age on the change in rectal temperature from pre to post trial. Data are presented as mean ± standard deviation (**Figure 13-2**), unless otherwise stated. Residual analysis was performed to test for the assumptions of the two-way ANOVA. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. Normality was assessed using Shapiro-Wilk's normality test, residuals were normally distributed (p >0.05). The assumption of homogeneity of variance was violated, as assessed by Levene's test for equality of variances, p = 0.048. The interaction effect between age and trial condition on change in rectal temperature from pre to post trial was not statistically significant $F(1, 39) = 0.700$, $p = 0.408$, partial $\eta^2 = 0.018$. Therefore, analysis of main effects was conducted. There was a statistically significant main effect of age on change in rectal temperature, $F(1, 39) = 5.084$, $p = 0.030$, partial $\eta^2 = 0.115$. Being a young man was associated with a mean rectal temperature change 0.28 (95% CI, 0.03 to 0.53)°C larger than the older men, a statistically significant difference, $p = 0.030$. There was a significant main effect of trial condition on change in rectal temperature, $F(1, 39) = 13.948$, p <0.001, partial $\eta^2 = 0.263$. The control trial was associated with having a mean change in rectal temperature 0.46 (95% CI, 0.71 to 0.21) °C smaller than the hot trial, a statistically significant difference, p <0.001.

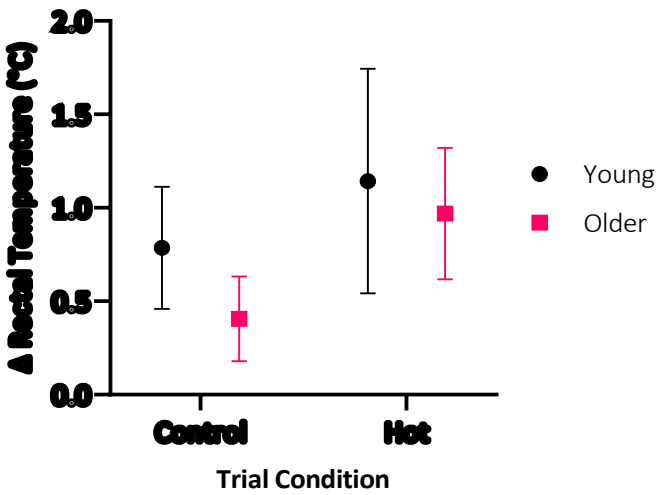

***Figure 13-2**. Change in rectal temperature in the control and hot trials for young and older participants. Data presented as mean ± SD.*

A three-way repeated measures ANOVA was conduction to determine the effects of age, trial condition, and time point on skin temperature. Data are mean ± standard deviation (**Figure 13-3**), unless otherwise stated. There was one outlier assessed as a value greater than 3 box lengths from

the edge of the box in a boxplot. Skin temperature was normally distributed (p >0.05), except for at the onset of exercise in the hot trial for the older men (p = 0.028) and at the end of the hot trial for the older men (p = 0.040), as assessed by Shapiro-Wilk's test of normality. Mauchly's test of sphericity indicated that the assumption of sphericity was met for the three-way interaction ($\chi^2(2) = 0.136$, p = 0.934). There was no statistically significant three-way interaction between age, trial condition, and time point, $F(2, 18) = 1.289$, p = 0.300. Mauchly's test of sphericity indicated that the assumption of sphericity was met for the two-way interaction of age and time ($\chi^2(2) = 1.583$, p = 0.453), but was violated for the two-way interaction of trial condition and time point ($\chi^2(2) = 7.342$, p = 0.025). Therefore, the Greenhouse-Geisser correction was applied for the two-way interaction of trial condition and time point. There was no statistically significant two-way interaction between age and trial condition, $F(1, 9) = 3.122$, p = 0.111. There was a statistically significant two-way interaction between age and time point, $F(2, 18) = 6.593$, p = 0.007. There was a statistically significant two-way interaction between trial condition and time point, $F(1.250, 11.246)$, p = 0.003, ε = 0.625.

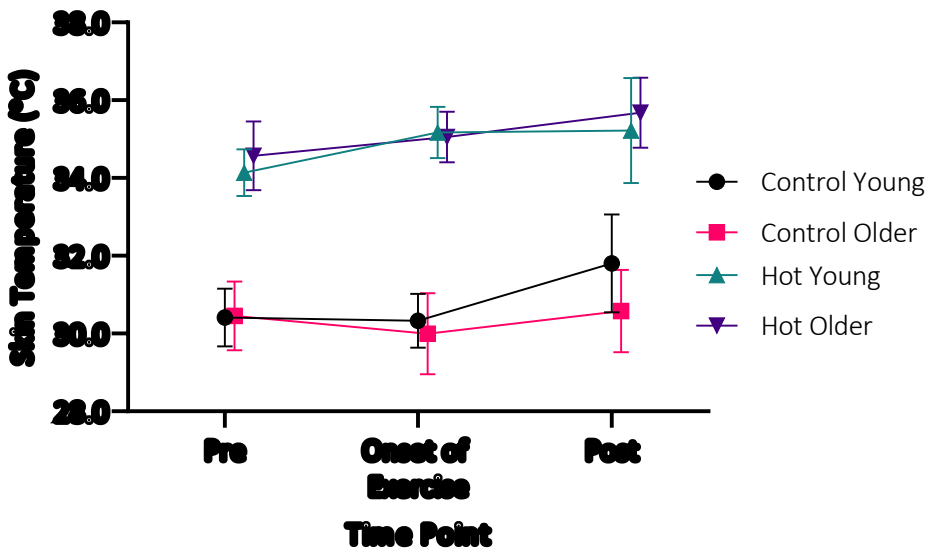

***Figure 13-3**. Skin temperature for young and older men during the control and hot trials. Data presented as mean ± SD.*

13.3.2 Thermoregulatory behaviour and perceptual responses

An independent samples t-test was run to examine the influence of age on thermoregulatory behaviour. Data are mean ± standard deviation, unless otherwise stated. There were 11 young and 11 older male participants. There were no outliers in the data, as assessed by inspection of a boxplot. Thermoregulatory behaviour scores were normally distributed for the young men, as assessed by Shapiro-Wilk's test (p >0.05). Thermoregulatory behaviour scores for the older men were not normally distributed (p = 0.024). As sample sizes were equal and the independent t-test is robust, analysis

continued. There was homogeneity of variance, as assessed by Levene's test for equality of variances (p = 0.464). The young men (1.06 ± 0.11) were better at implementing thermoregulatory behaviour than the older men (0.95 ± 0.07), a statistically significant difference of 0.11 (95% CI, 0.03 to 0.19), $t(20) = 2.906$, p = 0.009, d = 0.38, **Figure 13-4**.

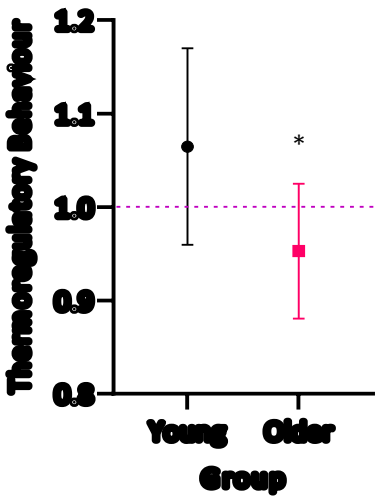

Figure 13-4. *Thermoregulatory behaviour score for the young and older men. Data presented as mean ± SD. * denotes older men significantly lower thermoregulatory behaviour score than the young men, p = 0.009. Points above the purple line implemented thermoregulatory behaviours by walking further in the control than the hot trial, below the purple line represents walking further in the hot trial than the control trial.*

A two-way ANOVA was conducted to examine the effects of age and trial condition on thermal comfort. Data are presented as mean ± standard deviation (**Figure 13-5**), unless otherwise stated. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. Data was normally distributed, as assessed by Shapiro-Wilk's test (p >0.05). The assumption of homogeneity of variance was violated, as assessed by Levene's test for equality of variances, p = 0.033. As sample sizes were equal and there was normality, the two-way ANOVA is robust, so analysis continued.

There was a statistically significant interaction between age and trial condition on thermal comfort, $F(1,40) = 4.644$, p = 0.037, partial η^2 = 0.104. Therefore, an analysis of simple main effects for age and trial condition was performed with statistical significance receiving a Bonferonni adjustment and being accepted at the p <0.025 level. The simple main effect of age on thermal comfort was not statistically significant in the control ($F(1, 40) = 1.505$, p = 0.227, partial η^2 = 0.036) or the hot trial ($F(1, 40) = 3.316$, p = 0.076, partial η^2 = 0.077). There was a statistically significant difference in mean thermal comfort score between the control and hot trials in young men ($F(1, 40) = 60.110$, p <0.001, partial η^2 = 0.600) and older men ($F(1, 40) = 116.653$, p <0.001, partial η^2 = 0.745). The mean difference in thermal

comfort was 1.471 (95% CI, 1.854 to 1.087, p <0.001) higher in the hot trial than the control trial for the young men, and 2.049 (95% CI, 2.433 to 1.666, p <0.001) for the older men.

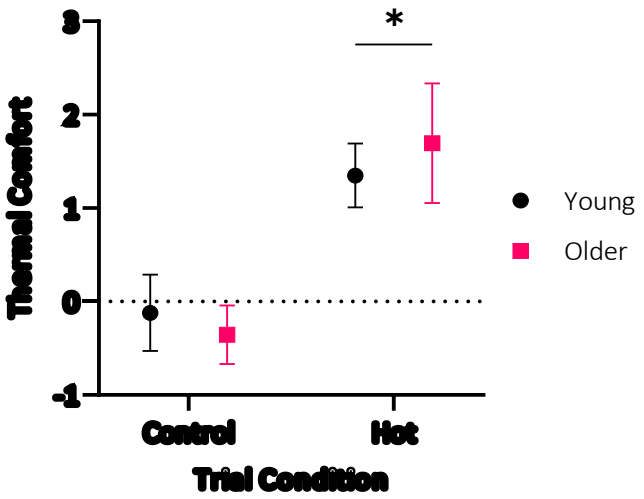

Figure 13-5. *Thermal comfort for young and older men in the control and hot trials. Data presented as mean ± SD. * denotes significantly higher thermal comfort score in the hot trial than the control trial, p <0.001.*

13.3.3 Physiological responses

A three-way mixed ANOVA was run to understand the effects of age, trial condition, and time point on heart rate (**Figure 13-6**). Heart rate was normally distributed, as assessed by Shapiro-Wilk's test (p >0.05). There were six outliers, as assessed by inspection of a boxplot. The outliers were kept in as they did not materially affect the results as assessed by comparison of the results with and without the outliers. Levene's test for equality of variance showed all data (p >0.05) except for the post control trial data (p = 0.025) had equal variance. The three-way ANOVA is somewhat robust to heterogeneity of variance. For the three-way interaction effect, Mauchly's test of sphericity indicated that the assumption on sphericity was met, $\chi^2(2) = 4.311$, $p = 0.116$. There was no statistically significant three-way interaction between trial condition, time point, and age $F(2, 40) = 0.580$, $p = 0.580$, partial $\eta^2 = 0.028$. There was no statistically significant two-way interaction between trial condition and age, $F(1, 20) = 0.011$, $p = 0.917$, partial $\eta^2 = 0.001$. There was no statistically significant two-way interaction between trial and time point, $F(2, 40) = 1.136$, $p = 0.331$, partial $\eta^2 = 0.054$. There was a statistically significant two-way interaction between time point and age, $F(2, 40) = 10.323$, p <0.001, partial $\eta^2 = 0.340$.

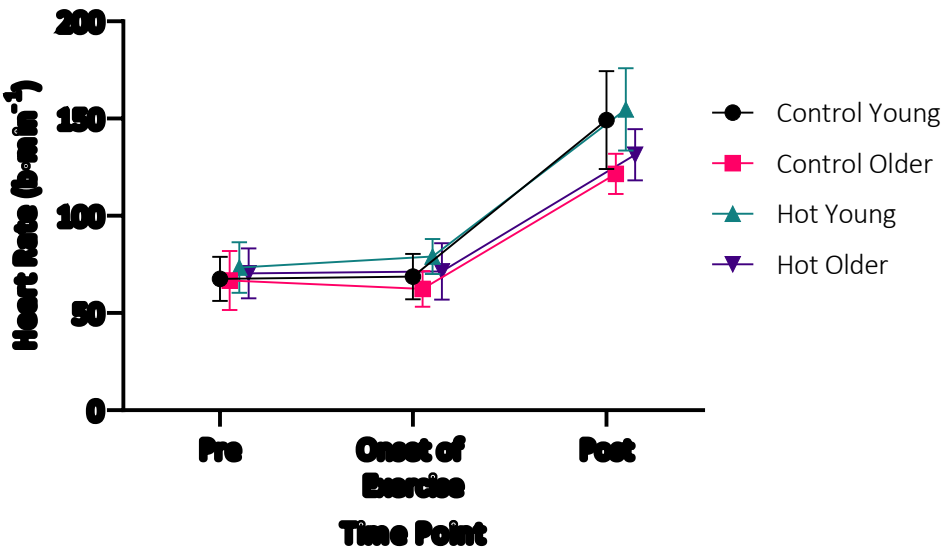

***Figure 13-6**. Heart rate during the control and hot trials for the young and older men. Data presented as mean ± SD.*

A three-way mixed ANOVA was run to understand the effects of age, trial condition, and time point on mean arterial pressure (**Figure 13-7**). Mean arterial pressure data was normally distributed, as assessed by Shipor-Wilk's test (p >0.05), except for the onset of exercise in the control trial for the young group (p = 0.019), and post-trial in the hot trial for the older group (p = 0.028). There was one outlier, as assessed by a boxplot. The outlier was kept in the analysis as it did not materially affect the results as assessed by a comparison of the results with and without the outlier. There was homogeneity of variances, as assessed by Levene's test for equality of variances (p >0.05). For the three-way interaction effect, Mauchly's test of sphericity was met, $\chi^2(2) = 0.995$, $p = 0.608$. There was no statistically significant three-way interaction between age, trial condition, and time point, $F(2, 34) = 2.220$, $p = 0.124$, partial $\eta^2 = 0.116$. There was no statistically significant two-way interaction between trial and age, $F(1, 17) = 0.53$, $p = 0.820$, partial $\eta^2 = 0.003$. There was no statically significant two-way interaction between time point and age, $F(2, 34) = 1.827$, $p = 0.176$, partial $\eta^2 = 0.097$. There was no statically significant two-way interaction between trial condition and time point, $F(2, 34) = 0.057$, $p = 0.945$, partial $\eta^2 = 0.003$.

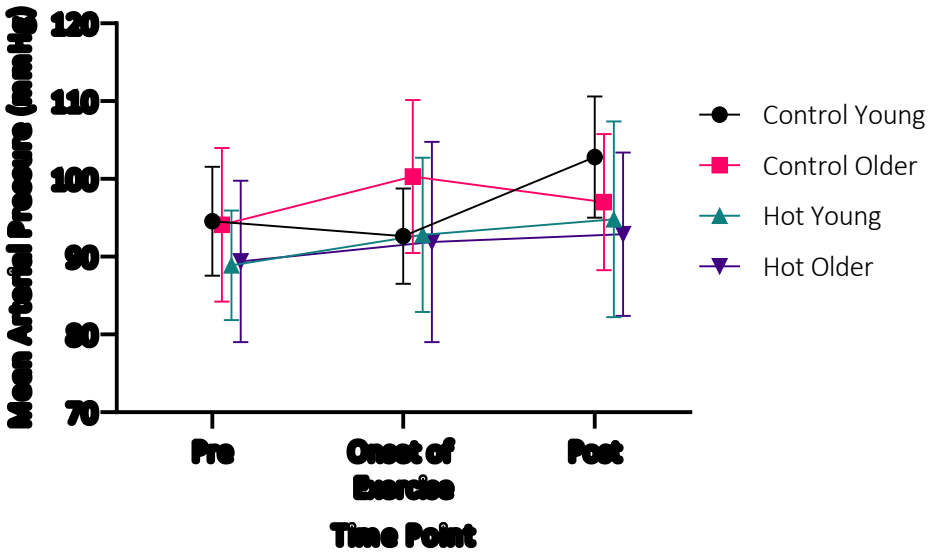

***Figure 13-7**. Mean arterial pressure during the control and hot trials for the young and older men. Data presented as mean ± SD.*

A two-way ANOVA was conducted to examine the effects of age and trial condition on whole body sweat rate. Data are mean ± standard deviation **Figure 13-8**, unless otherwise stated. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. Data was normally distributed (p >0.05), except for the young men in the hot trial (p = 0.011), as assessed by Shapiro-Wilk's normality test. Levene's test of homogeneity of variance indicated there was homogeneity of variances (p = 0.079).

There was a statistically significant interaction between age and trial condition on whole body sweat rate, $F(1, 33) = 4.220$, $p = 0.048$, partial $\eta^2 = 0.113$. Therefore, an analysis of simple main effects for age and trial condition was performed with statistical significance receiving a Bonferroni adjustment and being accepted at the p <0.025 level.

Whole body sweat rate was 0.126 (95% CI, 0.025 to 0.227) $L \cdot h^{-1}$ higher in the young men than the older men in the control trial, a statistically significant difference, $F(1, 33) = 6.462$, $p = 0.016$, partial $\eta^2 = 0.164$. In the hot trial, whole body sweat rate was not statistically significantly different between the young and older men, $F(1, 33) = 0.068$, $p = 0.795$, partial $\eta^2 = 0.002$.

In the hot trial whole body sweat rate was 0.178 (95% CI, 0.089 to 0.268) $L \cdot h^{-1}$ higher than the control trial for the young men, a statistically significant difference, $F(1, 33) = 16.519$, p <0.001, partial $\eta^2 = 0.334$. Whole body sweat rate was 0.316 (95% CI, 0.213 to 0.419) $L \cdot h^{-1}$ higher in the hot trial tan the control trial for the older men, a statistically significant difference, $F(1, 33) = 0.541$, p <0.001, partial $\eta^2 = 0.541$.

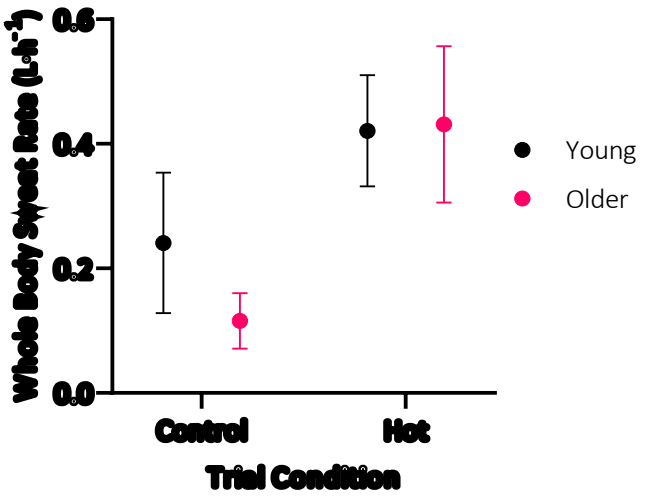

***Figure 13-8**. Whole body sweat rate during the control and hot trials for young and older men. Data presented as mean ± SD.*

13.4 Discussion

We have identified that men aged >65 years are less inclined to voluntarily initiate thermoregulatory behaviour through reducing exercise intensity while walking at an intensity perceived as 'somewhat hard' in hot ambient conditions (35°C, 40% RH), whereas men aged <35 years implement thermoregulatory behaviours. This lack of ability to reduce exercise intensity in older men was evident despite mediators of behavioural thermoregulation, skin temperature and thermal perceptions, having a similar response between young and older men. Older men are less sensitive than young men to skin temperature and thermal comfort, known drivers of thermoregulatory behaviour when exercising in the heat (Schlader et al., 2009, 2013; Schlader et al., 2011b).

Thermoregulatory behaviour is a vital mechanism for preventing heat illness during heat exposure. Young men have repeatedly demonstrated their ability to voluntarily implement thermoregulatory behaviours, both at rest (Schlader et al., 2013; Schlader et al., 2016a; Schlader et al., 2016b) and during exercise (Schlader et al., 2011b; Schlader et al., 2011c; Vargas et al., 2019a). Older adults perceive cycling at 6 METs in 25°C to be the same level of thermal comfort as cycling in 35°C (Waldock et al., 2018). As thermal comfort is a driver of thermoregulatory behaviours, Experimental Chapter One focussed on how age impacts thermoregulatory behaviours while walking in the heat. It was the first study to utilise a fixed RPE protocol with such a wide range of ages with 19 to 86 year-olds included. This work identified a decline in thermoregulatory behaviour with increasing age during walking exercise, particularly in men.

The aim of the present study was to identify if any physiological factors (heart rate, skin blood flow, mean arterial pressure, and whole body sweat rate) might be influencing this decline in thermoregulatory behaviour ability in men. We found no significant differences in these physiological factors between young (~26 years) and older (~72 years) men. The two groups also had similar anthropometry (stature, body mass, BMI, and sum of four skinfolds) and physical activity (PASE questionnaire) and physical fitness (6-minute walk test) measures. The older men in the present study did not implement thermoregulatory behaviours as effectively as the young men (thermoregulatory behaviour: young 1.06, older 0.95. $p = 0.009$). This study indicates that healthy older adults implement thermoregulatory behaviours less effectively than young adults.

Skin temperature mediates thermoregulatory behaviour. At the onset of exercise, a skin temperature of 35°C resulted in a 2.4% reduction in exercise intensity during a 60 min cycling time trial compared with a starting skin temperature of 29°C in young (~30 years) men (Schlader et al., 2011b). While sitting at rest in 42°C young (~22 years) adults expressed thermoregulatory behaviours by pressing a button that initiated neck cooling ~10x more often than when sitting in 32°C when skin temperature was 1.8°C cooler (Vargas et al., 2018b). Skin temperature increases of ~1.8°C are sufficient to drive thermoregulatory behaviours. Therefore, in the present study, both groups should have implemented thermoregulatory behaviours as they both experienced a ~5°C increase at the onset of exercise in the hot trial compared with the control trial. The young men responded as expected, the higher skin temperature in the hot trial helped drive a ~6% reduction in exercise intensity. The older men did not implement thermoregulatory behaviours as successfully as the young men, unexpectedly increasing their exercise intensity by ~5%. This outcome indicates that in older men there could be a disruption in the pathway between detecting skin temperature and initiating thermoregulatory behaviours, reducing the effectiveness of skin temperature as a mediator of thermoregulatory behaviours. It may be that men over 60 years of age require a greater rise in skin temperature before they implement thermoregulatory behaviours. Work manipulating skin temperature will help uncover how skin temperature mediates thermoregulatory behaviour in older men.

Skin temperature mediates thermoregulatory behaviours via its influence over thermal comfort (Bulcao et al., 2000). In the present study, both age groups perceived a similar level of thermal comfort and thermal sensation in both the control and hot trial. A study demonstrated that during 30 min recumbent cycling exercise at 6 METs, older (~72 years) adults did not perceive an increase in thermal discomfort when the ambient temperature was 35°C compared to 25°C (Waldock et al., 2018). The lack of increase in thermal discomfort was despite a ~3.2°C warmer skin temperature in the 35°C trial compared with the 25°C trial (Waldock et al., 2018). Participants in the present study experienced a similar level of heat strain as Waldock et al. (2018) with final rectal (~0.2°C difference) and skin (~0.4°C

difference) temperatures in 35°C trials being similar. Waldock et al.'s (2018) work demonstrated a lack of perception of thermal comfort in hot conditions, so it was anticipated that the older group would feel more comfortable in the heat than the young group and be less effective at implementing thermoregulatory behaviours. However, we observed that older men did perceive the hot trial to be warmer than the control trial, but they did not implement thermoregulatory behaviours. They also reported similar thermal perceptions as the young men.

As older adults have different thermal sensation preferences to younger adults (van Hoof et al., 2017), this outcome was unexpected. Differences in thermal sensation preference can often be explained by variations in metabolic heat production from differing activity levels between older and younger adults (Havenith, 2001), and environmental factors i.e. relative humidity, ambient temperature, and air velocity (Fanger, 1970; van Hoof et al., 2017). However, in the present study participants were exposed to the same environmental conditions, and carried out the same walking exercise, age was the only variable where the groups differed. It appears that thermal sensation in older men is less sensitive than in young men, thus a greater increase in ambient temperature may be required before they will voluntarily reduce their exercise intensity during heat exposure.

Despite the reduced ability to implement thermoregulatory behaviour, the older group did not exhibit a greater rectal (~0.2°C difference) or skin temperature (~0.5°C difference) than the young group at the end of the hot trial. Both groups experienced the same level of thermal strain, but the older men did not adjust their exercise intensity. The reduced ability to implement thermoregulatory behaviours may put the older group at an increased risk of heat-related illness during heat exposure. Previous work utilising whole body direct calorimetry demonstrates that marked differences in heat storage between age groups can occur with similar rectal temperatures (Larose et al., 2013). Whole-body direct calorimetry is more sensitive to changes in body heat storage than rectal temperature but requires specialised equipment (HEPRU, 2021); hence, the use of rectal temperature as an indicator of heat storage. Rectal temperature remained relatively modest, at the end of the hot trial the mean rectal temperature was 38.1°C for the young group and 37.9°C for the older group. While the rectal temperatures indicate that a similar level of heat storage was experienced by both age groups, use of a direct calorimeter might have revealed marked differences in heat storage.

In the present study, there was no difference in whole-body sweat rate between the two age groups during the hot trial. Previous research has demonstrated a reduction in sweating capacity in older adults. Men aged ~ 70 years have a 47% lower peripheral sweat rate than young (~27 years) men (Buono et al., 1991). During exercise, sweat rate in men aged ≥45 years was reduced by ~9.2%, contributing to a ~60% increase in body heat storage (Larose et al., 2013). The similarity in sweat rates

between the age groups in the present study could be due to the corresponding similarity in physical activity and fitness between the groups. Drinkwater et al. (1982) and Gonzalez et al. (1981) reported that $\dot{V}O_2max$ is related to sweating capacity, but age is not, for both men and women. Thus, whole-body sweat rate decline appears to be associated with the age-related decline in aerobic fitness rather than ageing itself.

Ambient conditions did not impact the mean arterial pressure for either age group at any time point. The older men had similar blood pressure responses to the young men throughout both trials. Unlike Schlader et al. (2016b), the present study did not identify a drop in mean arterial pressure before the young men implemented thermoregulatory behaviours. The differing methods employed by the present study and Schlader et al. (2016b) may explain this. Schlader et al. (2016b) used a resting protocol and measured beat-to-beat blood pressure continuously. The walking exercise may have prevented any drop in mean arterial pressure as exercise requires a higher blood pressure than seated rest. We were unable to replicate continuous blood pressure measurements, thus our intermittent readings may have missed the drop in blood pressure before thermoregulatory behaviours. This result leaves some uncertainty about the blood pressure response prior to exercise in a hot environment for both young and older men. However, we found similar responses at baseline, the onset of exercise, and post exercise, between the two age groups, suggesting that age does not influence the blood pressure response to heat exposure in healthy older men. Future work should employ continuous beat-to-beat blood pressure measurement to elucidate the relationship between blood pressure and thermoregulatory behaviour during exercise in the heat.

This study has some limitations, the first of which involves the use of a treadmill which forces participants to engage in a conscious decision to alter walking speed. This restriction prevents any behaviours that may be adopted without the awareness of the participant. Decision making on pacing during exercise is complex and not solely a conscious or subconscious process (Micklewright et al., 2017). Other investigators have demonstrated that pacing strategies can be implemented without conscious perception (Ansley et al., 2004). Utilising a method that allows participants to reduce exercise intensity without having to be aware of the decision to adjust exercise intensity would remove this limitation. However, walking was preferred in this study over cycling as only 11% of adults in the UK cycle weekly, whereas 60% make a walking journey weekly (Department for Transport, 2019). Also, the level of heat stress experienced by the participants was mild with the mean final rectal temperature of all participants reaching 38.0°C. It remains to be elucidated what level of heat strain is required for older men to voluntarily implement thermoregulatory behaviours.

13.5 Conclusion

Older men have similar physiological and thermal perception responses as young men when undertaking walking exercise in hot conditions. Rectal and skin temperature, heart rate, whole body sweat rate, skin blood flow, mean arterial pressure, and thermal comfort responses to 30 min of walking exercise in 22°C and 35°C are similar between men aged 26 ± 4 years and men aged 72 ± 7 years. Thus, our hypotheses that older men would experience a greater level of heat strain and would feel more thermally comfortable than young men can be rejected. Despite these similarities, the young men implemented thermoregulatory behaviours by reducing walking speed to a greater extent that the older men (1.06 v 0.95. $p = 0.009$). We can accept our first hypothesis that older men would implement thermoregulatory behaviours less effectively than young men. This puts older men at an increased risk of heat-related illness during heat wave events as they are unable to voluntarily reduce their heat strain. Thermoregulatory behaviours offer a nearly unlimited avenue for preventing heat stress. Public health heat wave advice should emphasise that older men are less able to perceive when thermoregulatory behaviours should be implemented.

14 Experimental Chapter Three: Thermoregulatory behaviour during fixed RPE cycling in changing ambient conditions in young and older men

14.1 Introduction

Experimental Chapter One: Relationship between age and ability to implement thermoregulatory behaviours in thermoneutral and hot ambient conditions identified that thermoregulatory behaviour is impaired in older adults, particularly men, and established that men aged ≥60 years do not reduce their walking speed when exposed to 35°C ambient conditions. The mechanisms responsible for this self-regulated behaviour remain unclear. Experimental Chapter Two: Physiological and perceptual factors influencing behavioural thermoregulation in young and older men in thermoneutral and hot ambient conditions could not identify a physiological or perceptual causative factor for the impairment in behavioural thermoregulation but did identify that older men are less able to implement thermoregulatory behaviours than young men. Understanding the contribution of skin and core temperature to thermoregulatory behaviours could elucidate possible mechanisms behind the decline in behavioural thermoregulation in older men.

Peak rectal temperature during the hot (35°C ambient) trials in Experimental Chapters One and Two reached ~38.0°C for all participants. While this was a ~1°C increase from the onset of the trials, core temperature of 38 – 40°C are not abnormal during exercise, particularly in hot conditions (Gleeson, 1998). Core temperature of young (~30 years) men performing 5,000 m of rowing at a fixed RPE of 15 ('hard') did not differ between ~35°C and 18°C ambient conditions with final core temperatures being ~38.5°C in both conditions (Lander et al., 2014). Thus, young men experience similar core temperature increases while exercising at a fixed RPE in temperate and hot conditions. Older men might require a higher rectal temperature before they will voluntarily initiate thermoregulatory behaviours. Hot water immersion at 40°C has been shown to induce a greater thermal challenge than exercising for the same amount of time at 50% of $\dot{V}O_2$max in 40°C (Greenfield et al., 2021). Experimental Chapter Three will employ hot water immersion to pre-warm participant's rectal temperature by 1°C before they undertake fixed RPE cycling for 30 min. This approach will permit examination of thermoregulatory behaviour when participants are experiencing heat strain prior to the onset of exercise, rather than using the exercise to induce heat strain.

Skin temperature mediates thermoregulatory behaviour (Schlader et al., 2009, 2013). However, the evidence from Experimental Chapter One: Relationship between age and ability to implement thermoregulatory behaviours in thermoneutral and hot ambient conditions Experimental Chapter Two: Physiological and perceptual factors influencing behavioural thermoregulation in young and older men in thermoneutral and hot ambient conditions indicates that skin temperature either has

little influence on thermoregulatory behaviour in older men exercising in the heat, or older men have a reduced sensitivity to increases in skin temperature. The young men experienced skin temperature during the hot trials of 35.24°C and 35.22°C in experimental chapters one and two respectively, while the older men experienced skin temperatures of 35.40°C and 35.68°C. This was an increase from the control trials of 3.45°C and 3.36°C in experimental chapters one and two respectively, for the young men, and 4.48°C and 4.60°C for the older men. Young (~30 years) men reduce rowing power output when skin temperature increases by 0.5°C under changing ambient conditions (Lander et al., 2014). During a 5,000 m rowing trial where RPE was fixed at 15 ('hard'), radiant heat lamps were used to increase the ambient temperature from 18 to 35°C from the 1,000 to 2,000 m and 3,000 to 4,000 m distances. Each 1,000 m took ~4 min to complete, so the heat lamps were activated for ~4 min during each warming period. Skin temperature rose ~0.5°C and power output declined by 6% during the first heat exposure (Lander et al., 2014). Young men were able to initiate thermoregulatory behaviours within 4 min of a change in ambient conditions. There is a dearth of research investigating thermoregulatory behaviours in exercising older adults during heat stress. Using heat lamps to alter the ambient temperature provides a way of rapidly inducing skin temperature changes in exercising individuals. It also allows for the examination of the effect of skin temperature, independently of core temperature, on thermoregulatory behaviours as skin temperature will change more rapidly than core temperature. We will employ this radiant heat lamp method in the present study to try and elucidate the influence of skin temperature on thermoregulatory behaviour in men aged ≥60 years.

There are contrasting models of how self-selected exercise intensity, or pacing, is controlled. The psychobiological model proposes that pacing is controlled consciously (Marcora, 2010), whereas the central governor theory suggests that motor units are controlled subconsciously (St Clair Gibson et al., 2006). These two theories are opposed, and it has been postulated that pacing is a multidimensional process requiring both conscious and subconscious, or sub-awareness input (Edwards & Polman, 2013; Micklewright et al., 2017). The treadmill protocol utilised in Experimental Chapters One and Two prevented any subconscious alterations in walking speed as participants had to consciously decide to push a button to alter the treadmill speed. Allowing participants to unknowingly adjust their exercise intensity may lead to the implementation of thermoregulatory behaviours sooner, and to a greater extent, than when they must consciously push a button on a treadmill. Cycling ergometer exercise will allow participants to unknowingly and knowingly adjust their exercise intensity by adjusting their pedal cadence. Therefore, the current study, will employ cycling exercise at a fixed RPE.

Pacing strategy during exercise in the heat is thought be anticipatory, implemented as soon as core temperature starts to increase, rather than once a threshold (core) temperature is reached (Tucker & Noakes, 2009). Within 4 min of starting a cycling time trial in 35°C at a fixed RPE of 16 (between 'hard'

and ‘very hard’), young (~23 years) men implemented pacing strategies that maintained a similar level of heat storage as a 15°C trial (Tucker et al., 2006). Thus, young men can voluntarily implement thermoregulatory behaviours while cycling at a fixed RPE by reducing their power output. Research investigating thermoregulatory behaviour in exercising adults has neglected to include older adults (≥60 years).

The use of pre-warming and changing environmental conditions with older participants will facilitate investigation of the independent and combined effect of rectal and skin temperature on behavioural thermoregulation during fixed RPE exercise. This novel approach should identify whether skin or rectal temperature has a greater influence on thermoregulatory behaviour in older men, or if an increase of both skin and core temperature are required for initiation of thermoregulatory behaviours. We hypothesise that: pre-warming will result in a reduction in total work done for both young and older men; power output will be reduced when the radiant heat lamps are turned on for both young and older men; older men will feel more thermally comfortable than young men; and the older men will experience a greater level of heat strain.

14.2 Methods

14.2.1 Participants

Nineteen healthy males, nine young (19-31 years) and ten older (66-87 years) volunteered to participate in the study. Participants were non-smokers, free from any known respiratory, cardiovascular, or metabolic disease, and not taking any medications known to affect thermoregulation. Participant anthropometric data is presented in **Error! Reference source not found.Error! Reference source not found.**. The experimental protocol was approved by the Plymouth Marjon University Ethics Panel (EP103) in accordance with the Declaration of Helsinki. Volunteers provided written informed consent after explanation of the purpose, benefits, risks, and experimental procedures of the study before participating in the study.

14.2.2 Experimental overview

This study used a group comparison of performance, temperature, and perceptual responses to cycling in 22°C or changing conditions (22-33°C), with and without passive heating before cycling exercise in younger and older men. A self-paced, fixed RPE protocol was used (similar to Lander et al., 2014), with participants completing a familiarisation session to get comfortable with the RPE scale and the cycle ergometer. Anthropometric measurements were also taken in the familiarisation session.

Participants completed four experimental trials consisting of 30 min of cycling at a fixed rating of perceived exertion (RPE) of 13, equivalent to ‘somewhat hard’ (Borg, 1982). Participants were blinded

to their speed, power output and heart rate data, and received no feedback on how far they had cycled during the trial. The control trial consisted of 30 min of cycling in 21.5 ± 0.5°C ambient conditions. Another trial was completed in changing ambient conditions, where radiant heat lamps were activated during 10 – 20 min of 30 min fixed RPE cycling. A third trial was conducted with pre-

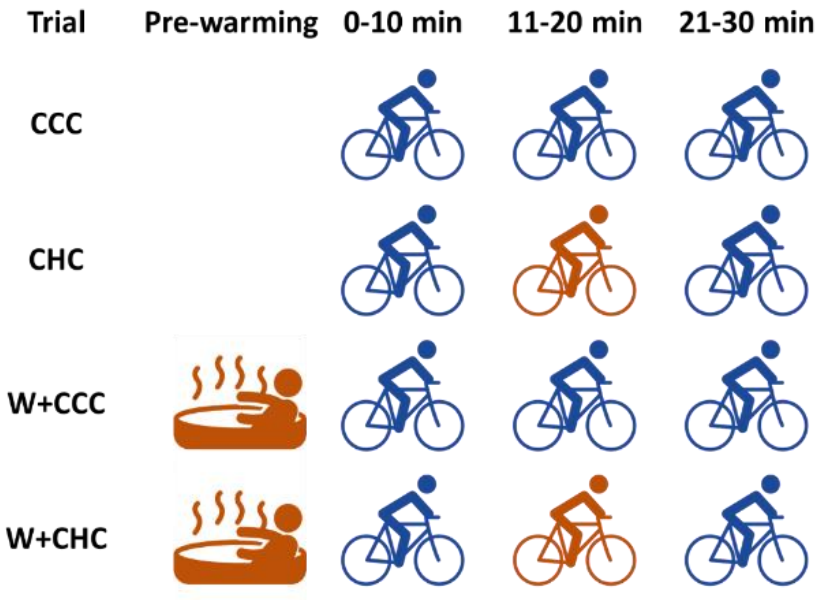

Figure 14-1. *Schematic diagram of the experimental design. Blue bicycle indicates radiant heat lamps turned off. Red bicycle indicates radiant heat lamps turned on. Red bath indicates pre-warming. CCC = control trial, no pre-warming, no heat lamps activated. CHC = trial with no pre-warming, heat lamps activated during middle 10 min of cycling. W+CCC = trial with pre-warming, no heat lamps activated. W+CHC = trial with pre-warming, and heat lamps activated for middle 10 min of cycling.*

warming, by immersion in 40 – 42°C water, of rectal temperature by 1°C prior to the 30 min of cycling in 21.5 ± 0.5°C with no heat lamp activation. The final trial condition included both pre-warming and heat lamp activation during 10 – 20 min of the 30 min fixed RPE cycling. Four radiant heat lamps were positioned around the participant, 1 m away from the centre of the cycle ergometer. Ten min into the trials using the lamps they were turned on for 10 min, resulting in an increase in ambient temperature to 33.3 ± 1.4°C.

Trials were conducted in a randomised order, participants arrived at the same time of day, and with at least 48 h between trials. Participants were also asked to abstain from alcohol, caffeine, non-steroidal anti-inflammatory drugs, and strenuous exercise in the 24 h before trials, and consume 500 mL of water the evening before and 2 h before arriving at the laboratory. Clothing consisted of a t-shirt, shorts, and walking/running shoes. Water was available for *ad libitum* consumption throughout the trials.

Mean power output was calculated in 10 min periods (0-10 min, 11-20 min, and 21-30 min). A marker of thermoregulatory behaviour within a trial was then calculated as:

Equation 14-1*. Thermoregulatory behaviour established as a change in power output as a function of time.*

$$Thermoregulatory\ Behaviour = \frac{Mean\ Power\ Output_{Trial\ A\ Time\ Period\ x}}{Mean\ Power\ Output_{Trial\ A\ Time\ Period\ y}}$$

Between trials thermoregulatory behaviour (CCC v CHC, CCC v W+CCC, CHC v W+CHC, W+CCC v W+CHC) was also compared at each time period and calculated as:

Equation 14-2. *Thermoregulatory behaviour established as a change in power output as a function of trial condition.*

$$Thermoregulatory\ Behaviour = \frac{Mean\ Power\ Output_{Trial\ A\ Time\ Period\ x}}{Mean\ Power\ Output_{Trial\ B\ Time\ Period\ x}}$$

This index gave a ratio of power output within a trial as duration increased, and between trials with different ambient and pre-warming conditions. A result >0 indicated that the participant reduced power output between conditions being compared, <0 they increased power output between conditions being compared, =0 there was no difference between conditions being compared.

14.2.3 Experimental procedures

In the preliminary visit, anthropometric measures (stature, body mass, skinfolds) were taken as described in the General Methods. Participants were then familiarised with the RPE scale. Participants were asked to adjust the cycle ergometer (Wattbike Pro/Trainer, Wattbike, Nottingham, UK) so that they were comfortable on the bike. The seat and handlebar positions were recorded for use in all subsequent trials. Next, participants self-selected a resistance on the cycle ergometer that elicited an RPE of 13, 'somewhat hard' while blinded to their power output and speed. After 3 min participants confirmed that the resistance settings felt 'somewhat hard', and their power output was recorded (but not revealed to the participant) before they sat at rest for 5 min. Participants repeated this procedure until they were able to replicate a power output that elicited an RPE of 13. The resistance settings were recorded for use in subsequent trials.

During hot water immersion both rectal and water temperature were monitored continuously. Heart rate was recorded every 5 min. Water was available to drink *ad libitum.* Once rectal temperature had increased by 1°C, participants were removed from the hot water bath and given privacy while they changed into dry clothing, the rectal thermistor remained in place. Participants then entered the environmental chamber where skin thermistors were attached as described in the General Methods (Chapter 11.9.2 Skin Temperature). An ambient temperature thermistor was hung from the ceiling

~30 cm above the participant's head. Temperature data was collected as described in the General Methods (Chapter 11.9 Temperature). Participants then mounted the cycle ergometer and began the 30 min cycling trial. Heart rate and power output were recorded at ~1 sec intervals by the Wattbike. These data were then averaged into 10 min blocks. RPE was assessed every 5 min.

14.2.4 Statistical analysis

For *a priori* power analysis, conventional α (0.05) and β (0.20) parameters and a large effect size (1.3), based on data from Experimental Chapter Two were employed. The estimations indicated that 9 subjects per group was appropriate to detect meaningful differences. Power analysis was performed using G*Power (G*Power, Düsseldorf, Germany).

Data is presented as mean ± standard deviation, unless otherwise stated. The level of significance for all analyses was set at $p \leq 0.05$. Effect size was defined as small $d = 0.20$, partial $\eta^2 = 0.01$; medium $d = 0.50$, partial $\eta^2 = 0.06$; and large $d = 0.80$, partial $\eta^2 = 0.14$ (Cohen, 1988; Cohen 1969). All statistical analyses were conducted using Statistics Package for the Social Sciences (SPSS v28, IBM Corp, Armonk, NY).

14.3 Results

14.3.1 Participant characteristics and temperature responses

Participant characteristics are presented in **Table 14-1**.

***Table 14-1**. Participant anthropometric data. Data presented as mean ± SD. Bold denotes significantly different from the young group.*

	n	Age (years)	Stature (cm)	Body Mass (kg)	Body Mass Index	Σskinfolds (mm)
Young	9	24 ± 4	181 ± 5	86.9 ± 19.6	26.6 ± 5.7	47.1 ± 29.2
Older	10	**70 ± 7**	176 ± 5	73.8 ± 9.0	23.8 ± 2.6	33.5 ± 15.3
p value	-	<0.001	0.067	0.077	0.188	0.234

A two-way repeated measures ANOVA was run to determine the effect of trial conditions and time point on ambient temperature (**Figure 14-2**). Analysis of studentised residual showed that there was normality, as assessed by the Shapiro-Wilk test of normality (p >0.05), and no outliers as assessed by no studentised residuals greater than ± 3 standard deviations. Mauchly's test of sphericity indicated that the assumption of sphericity had been violated for the two-way interaction, $\chi^2(2) = 89.831$, p <0.001, therefore a Greenhouse-Geisser correction was applied.

There was a statistically significant two-way interaction between treatment and time, $F(3.145, 56.609) = 246.068$, $p < 0.001$, $\varepsilon = 0.524$. Therefore, simple main effects were run.

Ambient temperature was not statistically significantly different between trials at during the first 10 min of cycling, $F(1.974, 35.535) = 0.726$, $p = 0.489$, $\varepsilon = 0.658$.

There was a statistically significant effect of trial condition on ambient temperature during the middle 10 min of cycling, $F(2.020, 36.368) = 935.734$, $p < 0.001$, $\varepsilon = 0.673$. Ambient temperature was warmer in the CHC (33.24 (95% CI) 32.53 to 33.95°C) and W+CHC (33.39 (95% CI) 32.76 to 34.02°C) trials than the CCC (21.48 (95% CI) 21.28 to 21.70°C) and W+CCC (21.46 (95% CI) 21.20 to 21.72°C) trials.

There was a statistically significant effect of trial condition on ambient temperature during the final 10 min of cycling, $F(1.995, 35.911) = 13.631$, $p < 0.001$, $\varepsilon = 0.665$. Ambient temperature was warmer in the CHC (23.53 (95% CI) 22.60 to 24.45°C) and W+CHC (23.92 (95% CI) 22.83 to 25.02°C) trials than the CCC (21.58 (95% CI) 21.36 to 21.80°C) and W+CCC (21.54 (95% CI) 21.28 to 21.80°C) trials.

There was a statistically significant effect of time on ambient temperature during the control trial (CCC), $F(2, 36) = 7.218$, $p = 0.002$. Ambient temperature was warmer during the first 10 min (21.80 (95% CI) 21.55 to 22.05°C) than the middle (21.48 (95% CI) 21.28 to 21.69°C, $p = 0.001$) and final 10 min of cycling (21.58 (95% CI) 21.36 to 21.80°C, $p = 0.050$).

There was a statistically significant effect of time on ambient temperature during the no pre-warming, heat lamp trial (CHC), $F(2, 36) = 336.332$, $p < 0.001$. Ambient temperature was warmer during the middle 10 min (33.24 (95% CI) 32.53 to 33.95°C) than the first (21.97 (95% CI) 21.68 to 22.27°C, $p < 0.001$) and final 10 min of cycling (23.53 (95% CI) 22.60 to 24.45°C, $p < 0.001$). The final 10 min of cycling were also warmer than the first 10 min of cycling, $p = 0.006$.

There was a statistically significant effect of time on ambient temperature during pre-warming, no heat lamps trial (W+CCC), $F(2, 36) = 40.230$, $p < 0.001$. Ambient temperature was warmer during the first 10 min (22.03 (95% CI) 21.79 to 22.28°C) than the middle (21.46 (95% CI) 21.20 to 21.71°C, $p < 0.001$) and final 10 min of cycling (21.54 (95% CI) 21.28 to 21.80°C, $p < 0.001$).

There was a statistically significant effect of time on ambient temperature during the pre-warming, and heat lamp trial (W+CHC), $F(2, 36) = 302.170$, $p < 0.001$. Ambient temperature was warmer during the middle 10 min (33.39 (95% CI) 32.76 to 34.02°C) than the first (21.74 (95% CI) 21.18 to 22.30°C, $p < 0.001$) and final 10 min of cycling (23.92 (95% CI) 22.83 to 25.02°C, $p < 0.001$). The final 10 min of cycling were also warmer than the first 10 min of cycling, $p = 0.002$.

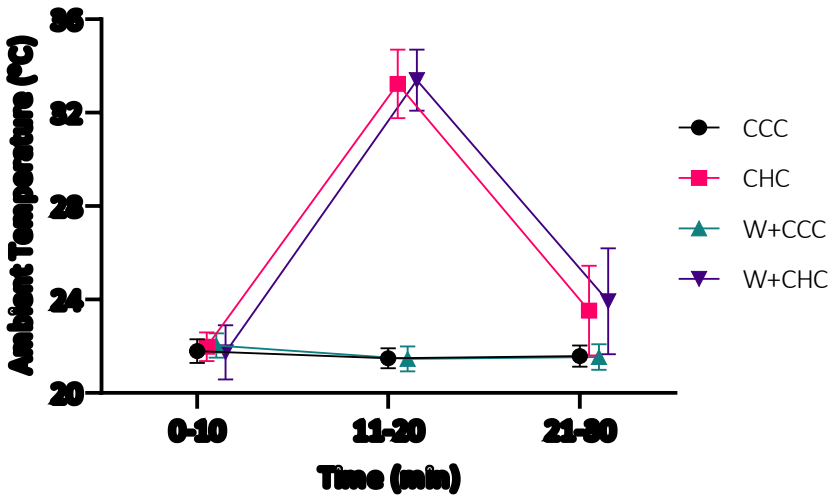

***Figure 14-2**. Ambient temperature for the first, middle, and final 10 min of cycling in each trial. Data presented as mean ± SD.*

A three-way mixed ANOVA was run to examine the effects of age, trial condition, and time point on mean skin temperature (**Figure 14-3**). There were three outliers, as assessed by inspection of a boxplot for values greater than 3 box-lengths from the edge of the box. These were kept in the analysis as they did not materially affect the result. Mean skin temperature data was normally distributed (p >0.05), except for the final 10 min of cycling in the control trial for the older men (p = 0.023), as assessed by Shapiro-Wilk's test. There was homogeneity of variances, as assessed by Levene's test for equality of variances (p >0.05). Mauchly's test of sphericity indicated that the assumption of sphericity was violated, $\chi^2(20) = 35.580$, p = 0.020. Therefore, the Greenhouse-Geisser correction was applied.

There was no statistically significant three-way interaction between age, trial condition, and time point on mean skin temperature, $F(3.792, 56.886) = 1.258$, p = 0.297, partial $\eta^2 = 0.077$, $\varepsilon = 0.632$. There was no statistically significant two-way interaction between trial condition and age on mean skin temperature, $F(1.980, 29.699) = 1.159$, p = 0.327, partial $\eta^2 = 0.072$. There was no statistically significant two-way interaction between time point and age on mean skin temperature, $F(1.331, 19.970) = 1.205$, p = 0.302, partial $\eta^2 = 0.074$. There was a statistically significant two-way interaction between trial condition and time point on mean skin temperature, $F(3.792, 563886) = 130.462$, p <0.001, partial $\eta^2 = 0.897$.

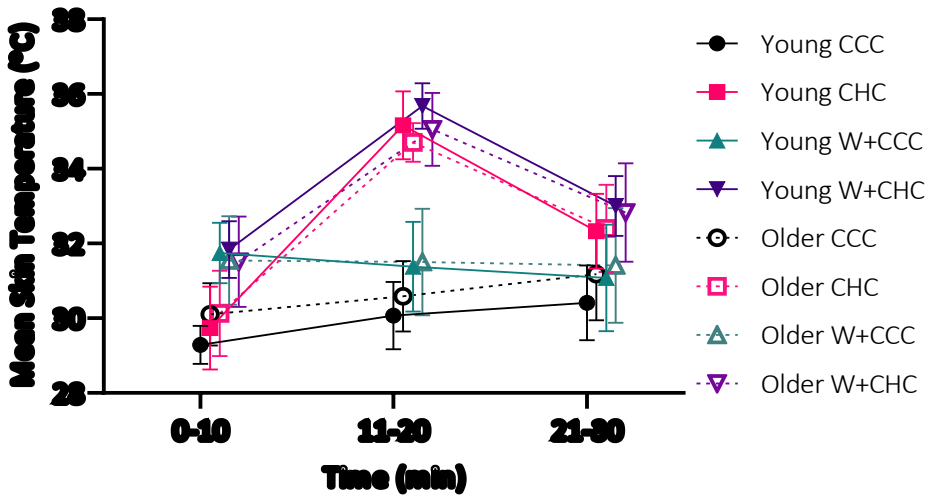

***Figure 14-3**. Mean skin temperature during the first, middle, and final 10 min of cycling for each trial in the young and older men. Data presented as mean ± SD.*

A three-way mixed ANOVA was run to examine the effects of age, trial condition, and time point on rectal temperature (**Figure 14-4**). There were no outliers, as assessed by inspection of a boxplot for values greater than 3 box-lengths from the edge of the box. Rectal temperature data was normally distributed, as assessed by Shapiro-Wilk's test (p >0.05). There was homogeneity of variances, as assessed by Levene's test for equality of variances (p >0.05). Mauchly's test of sphericity indicated that the assumption of sphericity had been violated for the two-way interaction, $\chi^2(2) = 86.545$, p <0.001, therefore a Greenhouse-Geisser correction was applied.

There was no statistically significant three-way interaction between age, trial condition, and time point on rectal temperature, $F(3.258, 58.650) = 2.692$, $p = 0.050$, partial $\eta^2 = 0.130$, $\varepsilon = 0.543$. There was a statistically significant two-way interaction between trial condition and age on rectal temperature, $F(2.831, 50.960) = 3.383$, $p = 0.027$, partial $\eta^2 = 0.158$. There was no statistically significant two-way interaction between time and age on rectal temperature, $F(1.012, 18.214) = 1.793$, $p = 0.197$, partial $\eta^2 = 0.091$. There was a statistically significant two-way interaction between trial condition and time point on rectal temperature, $F(3.258, 58.650) = 56.403$, p <0.001, partial $\eta^2 = 0.758$.

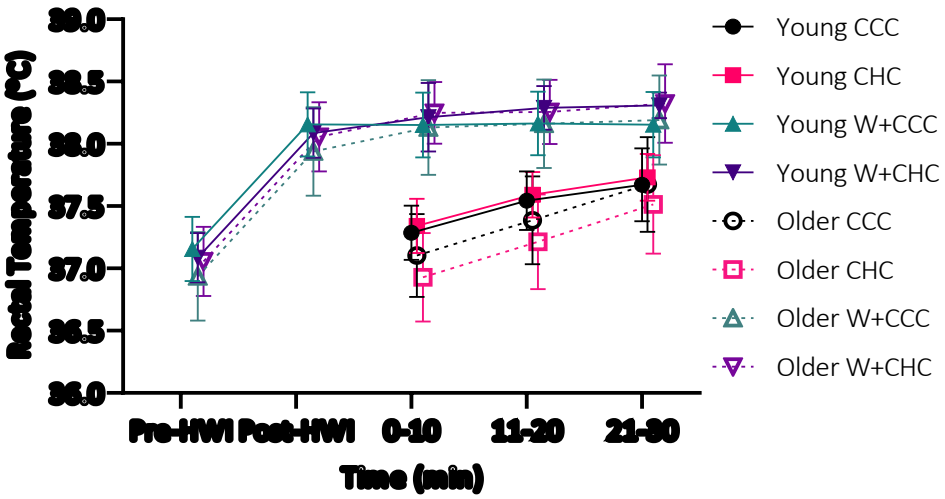

Figure 14-4*. Rectal temperature during the trials for the young and older men. HWI = hot water immersion. Data presented as mean ± SD.*

14.3.2 Thermoregulatory behaviour

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour when comparing the CCC and CHC trials (**Figure 14-5**). There was one outlier, which had a studentised residual value of 3.05. This outlier was kept in the analysis as it did not materially affect the results. Thermoregulatory behaviour data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variances, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's test of equality of covariance matrices (p = 0.078). Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi^2(2) = 8.115$, $p = 0.017$. Therefore, the Greenhouse-Geisser correction was applied.

There was no statistically significant two-way interaction between time point and age on thermoregulatory behaviour, $F(1.431, 24.324) = 1.496$, $p = 0.241$, partial $\eta^2 = 0.081$, $\varepsilon = 0.715$. The main effect of time point showed a statistically significant difference in thermoregulatory behaviour at different time points, $F(1.431, 24.324) = 5.098$, $p = 0.023$, partial $\eta^2 = 0.231$. The final 10 min of cycling (0.97 (95% CI) 0.90 to 1.03) had a higher thermoregulatory behaviour score than the first 10 min of cycling (1.05 (95% CI) 0.99 to 1.10) $p = 0.031$). Thermoregulatory behaviour score for middle 10 min of cycling (0.99 (95% CI) 0.91 to 1.07) did not statistically significantly differ from either the first ($p = 0.349$) or the final ($p = 0.289$) 10 min of cycling. The main effect of age showed no statistically significant difference between the young (1.01 (95% CI) 0.93 to 1.10) and older (0.99 (95% CI) 0.91 to 1.07) men in thermoregulatory behaviour score, $F(1, 17) = 0.107$, $p = 0.748$, partial $\eta^2 = 0.006$.

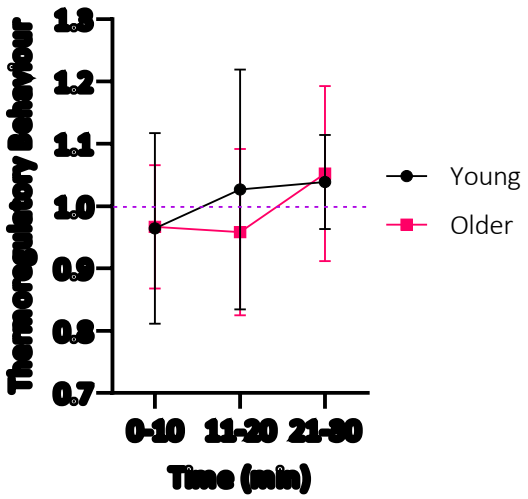

Figure 14-5. *Thermoregulatory behaviour of young and older men during the first, middle, and final 10 min of cycling when comparing the CHC trial to the CCC trial. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output in the CHC trial compared to the CCC trial.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour when comparing the CCC and W+CCC trials (**Figure 14-6**). Data is presented as mean ± standard deviation, unless otherwise stated. There was one outlier, which had a studentised residual value of 3.05. This outlier was kept in the analysis as it did not materially affect the results. Thermoregulatory behaviour data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variances, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's test of equality of covariance matrices (p = 0.092). Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi^2(2) = 11.272$, $p = 0.004$. Therefore, the Greenhouse-Geisser correction was applied.

There was a statistically significant interaction between age and time on thermoregulatory behaviour, $F(1.328, 22.582) = 4.404$, $p = 0.038$, partial $\eta^2 = 0.206$, $\varepsilon = 0.664$. Therefore, simple main effects were examined. There was no statistically significant difference in thermoregulatory behaviour between the age groups during the first 10 min of cycling ($p = 0.121$, partial $\eta^2 = 0.136$), the middle 10 min of cycling ($p = 0.271$, partial $\eta^2 = 0.071$), or the final 10 min of cycling (p = 0.836, partial $\eta^2 = 0.003$). There was no statistically significant effect of time on thermoregulatory behaviour for the young men (p = 0.282, partial $\eta^2 = 0.146$). There was a statistically significant effect of time on thermoregulatory behaviour for the older men, $F(2, 18) = 6.033$, $p = 0.010$, partial $\eta^2 = 0.401$.

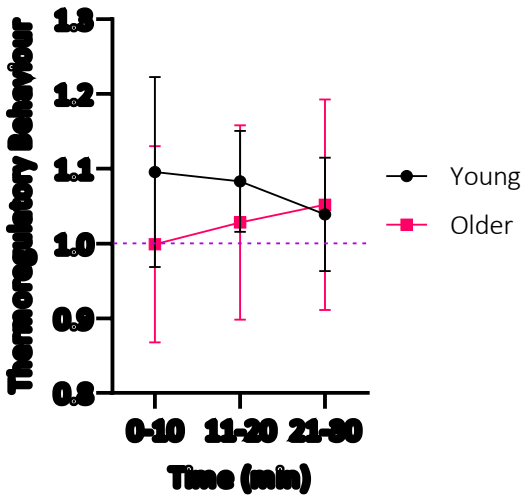

***Figure 14-6**. Thermoregulatory behaviour of young and older men during the first, middle, and final 10 min of cycling when comparing the W+CCC trial to the CCC trial. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output in the W+CCC trial compared to the CCC trial.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour when comparing the CCC and W+CHC trials (**Figure 14-7**). Data is presented as mean ± standard deviation, unless otherwise stated. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. All data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variance, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's equality of variance matrices (p = 0.03). Mauchly's test of sphericity indicated that the assumption of sphericity was met for the two-way interaction, $\chi^2(2) = 4.944$, $p = 0.084$.

There was no statistically significant two-way interaction between time point and age on thermoregulatory behaviour, $F(2, 34) = 0.949$, $p = 0.397$, partial $\eta^2 = 0.053$. Therefore, main effects were examined. The main effect of time point showed a statistically significant difference in thermoregulatory behaviour at the different time points, $F(2, 34) = 3.519$, $p = 0.041$, partial $\eta^2 = 0.171$. The main effect of age group showed that there was no statistically significant difference in thermoregulatory behaviour between the young and older men, $F(1, 17) = 0.713$, $p = 0.410$, partial $\eta^2 = 0.040$. The first 10 min of cycling (1.02 (95% CI) 0.956 to 1.084) did not significantly differ from the middle (1.06 (95% CI) 1.00 to 1.12, $p = 0.108$) or final 10 min of cycling (1.06 (95% CI) 1.00 to 1.12, $p = 0.207$). The middle 10 min of cycling was not significantly different to the final 10 min of cycling ($p = 0.999$).

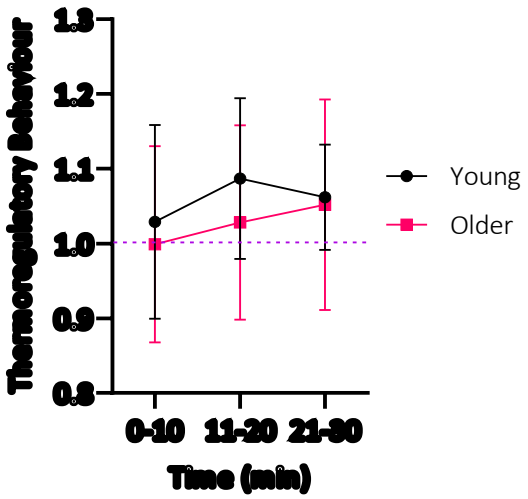

Figure 14-7. *Thermoregulatory behaviour of young and older men during the first, middle, and final 10 min of cycling when comparing the W+CHC trial to the CCC trial. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output in the W+CHC trial compared to the CCC trial.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour during the CCC trial (**Figure 14-8**). Data is presented as mean ± standard deviation, unless otherwise stated. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. All data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variance, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's equality of variance matrices (p = 0.02). Mauchly's test of sphericity indicated that the assumption of sphericity was met for the two-way interaction, $\chi^2(2) = 3.499$, $p = 0.174$.

There was a statistically significant interaction effect of age and time on thermoregulatory behaviour during the CCC trial, $F(2, 34) = 3.813$, $p = 0.032$, partial $\eta^2 = 0.183$. Therefore, simple main effects were run. There was no statistically significant difference in thermoregulatory behaviour between the age groups when comparing the middle 10 min of cycling to the first 10 min of cycling, $F(1, 17) = 4.114$, $p = 0.058$, partial $\eta^2 = 0.195$. The young men implemented statistically significantly better thermoregulatory behaviour (1.05 (95% CI) 1.01 to 1.08) in the final 10 min of cycling when compared to the middle 10 min of cycling than the older men (0.98 (95% CI) 0.94 to 1.01), $F(1, 17) = 7.835$, $p = 0.012$, partial $\eta^2 = 0.315$. The young men implemented statistically significantly better thermoregulatory behaviour (1.07 (95% CI) 0.99 to 1.15) in the final 10 min of cycling compared to the first 10 min of cycling than the older men (0.94 (95% CI) 0.86 to 1.01), $F(1, 17) = 6.658$, $p = 0.019$, partial $\eta^2 = 0.281$.

For the young men, thermoregulatory behaviour was not statistically significantly different between time points, $F(2, 16) = 1.801$, $p = 0.197$, partial $\eta^2 = 0.184$. For the older men, thermoregulatory behaviour was statistically significantly different between time points, $F(2, 18) = 4.120$, $p = 0.034$, partial $\eta^2 = 0.314$.

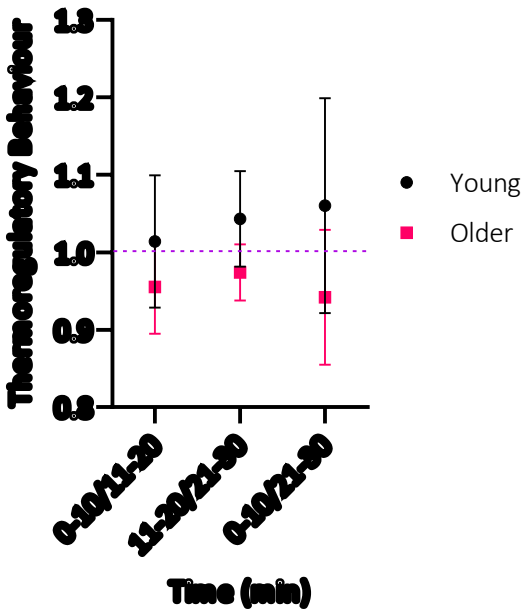

Figure 14-8. *Thermoregulatory behaviour of young and older men throughout the CCC trial by comparing the middle to the first 10 min of cycling, the middle to the final 10 min of cycling, and the final to the first 10 min of cycling. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output as time increased.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour during the CHC trial (**Figure 14-9**). Data is presented as mean ± standard deviation, unless otherwise stated. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. All data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variance, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's equality of variance matrices ($p = 0.03$). Mauchly's test of sphericity indicated that the assumption of sphericity was met for the two-way interaction, $\chi^2(2) = 3.486$, $p = 0.175$.

There was a statistically significant interaction effect of age and time on thermoregulatory behaviour during the CHC trial, $F(2, 34) = 9.085$, p <0.001, partial $\eta^2 = 0.348$. Therefore, simple main effects were run. The young men implemented statistically significantly better thermoregulatory behaviour (1.09 (95% CI) 1.03 to 1.15) in the middle 10 min of cycling compare to the first 10 min of cycling than the older men (0.94 (95% CI) 0.87 to 1.00), $F(1, 17) = 13.478$, $p = 0.002$, partial $\eta^2 = 0.442$. There was not statistically significant difference in thermoregulatory behaviour between the age groups when

comparing the final 10 min of cycling to the middle 10 min of cycling, $F(1, 17) = 1.503$, $p = 0.237$, partial $\eta^2 = 0.081$. The young men implemented statistically significantly better thermoregulatory behaviour (1.12 (95% CI) 1.03 to 1.20) in the final 10 min of cycling compared to the first 10 min of cycling than the older men (0.94 (95% CI) 0.86 to 1.02), $F(1, 17) = 9.743$, $p = 0.006$, partial $\eta^2 = 0.364$.

For the young men, thermoregulatory behaviour was statistically significantly different between time points, $F(2, 16) = 4.029$, $p = 0.038$, partial $\eta^2 = 0.335$ (first to middle 10 min, 1.09 (95% CI) 1.01 to 1.17. Middle to final 10 min, 1.03 (95% CI) 0.98 to 1.08. First to final 10 min, 1.12 (95% CI) 1.01 to 1.22). For the older men, thermoregulatory behaviour was statistically significantly different between time points, $F(2, 18) = 6.412$, $p = 0.008$, partial $\eta^2 = 0.416$ (first to middle 10 min, 0.94 (95% CI) 0.90 to 0.99. Middle to final 10 min, 0.99 (95% CI) 0.96 to 1.03. First to final 10 min, 0.94 (95% CI) 0.86 to 1.02).

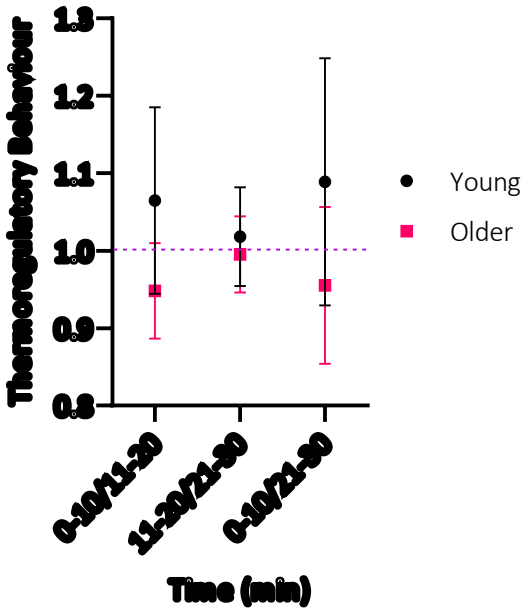

Figure 14-9. *Thermoregulatory behaviour of young and older men throughout the CHC trial by comparing the middle to the first 10 min of cycling, the middle to the final 10 min of cycling, and the final to the first 10 min of cycling. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output as time increased.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour during the W+CCC trial (**Figure 14-10**). Data is presented as mean ± standard deviation, unless otherwise stated. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. All data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variance, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's equality of variance matrices (p = 0.01).

Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi^2(2) = 8.373$, $p = 0.015$. Therefore, a Greenhouse-Geisser correction was applied.

There was no statistically significant interaction effect of age and time on thermoregulatory behaviour during the W+CCC trial, $F(1.421, 24.157) = 0.634$, $p = 0.487$, partial $\eta^2 = 0.036$. Therefore, main effects were examined. The main effect of time did not show a statistically significant difference in thermoregulatory behaviour at different time points of the W+CCC trial, $F(1.421, 24.157) = 0.012$, $p = 0.988$, partial $\eta^2 = 0.001$. The main effect of group showed no statistically significant effect of age on thermoregulatory behaviour in the W+CCC trial, $F(1, 17) = 0.601$, $p = 0.449$, partial $\eta^2 = 0.034$.

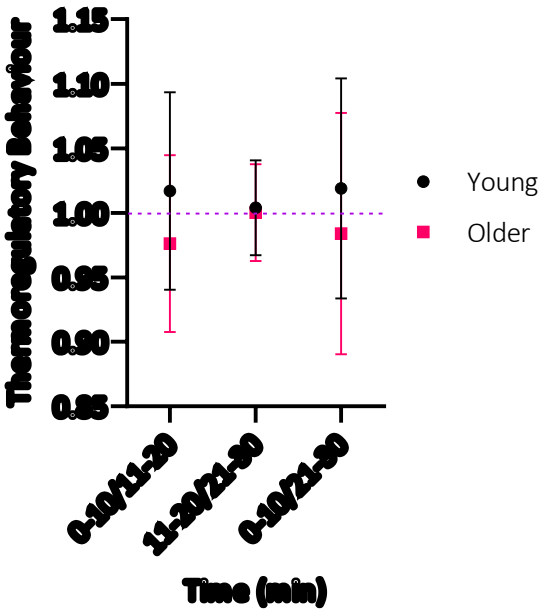

Figure 14-10. *Thermoregulatory behaviour of young and older men throughout the W+CCC trial by comparing the middle to the first 10 min of cycling, the middle to the final 10 min of cycling, and the final to the first 10 min of cycling. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output as time increased.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour during the W+CHC trial (**Figure 14-11**). Data is presented as mean ± standard deviation, unless otherwise stated. There was one outlier, which had a studentised residual value of -3.08. This outlier was kept in the analysis as it did not materially affect the results. Thermoregulatory behaviour data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variances, as assessed by Levene's test of homogeneity of variance (p >0.05) for the middle 10 min compared to the final 10 min, and the first 10 min compared to the final 10 min, but not for the first 10 min compared with the middle 10 min ($p = 0.014$). There was not homogeneity of covariances, as assessed by Box's test of equality of covariance matrices (p <0.001). The analysis was continued as the two-way

mixed ANOVA is robust to this violation. Mauchly's test of sphericity indicated that the assumption of sphericity was met, $\chi^2(2) = 1.265$, $p = 0.531$.

There was a statistically significant interaction between the time period and age group on thermoregulatory behaviour, $F(2, 34) = 1.225$, $p = 0.021$, partial $\eta^2 = 0.203$. Therefore, simple main effects were run.The young men implemented statistically significantly better thermoregulatory behaviour (1.09 (95% CI) 1.02 to 1.15) in the middle 10 min of cycling compare to the first 10 min of cycling than the older men (0.97 (95% CI) 0.91 to 1.03), $F(1, 17) = 7.685$, $p = 0.013$, partial $\eta^2 = 0.311$. There was not statistically significant difference in thermoregulatory behaviour between the age groups when comparing the final 10 min of cycling to the middle 10 min of cycling, $F(1, 17) = 0.958$, $p = 0.341$, partial $\eta^2 = 0.053$. The young men implemented statistically significantly better thermoregulatory behaviour (1.11 (95% CI) 1.01 to 1.21) in the final 10 min of cycling compared to the first 10 min of cycling than the older men (0.96 (95% CI) 0.86 to 1.05), $F(1, 17) = 5.662$, $p = 0.029$, partial $\eta^2 = 0.250$.

For the young men, thermoregulatory behaviour was not statistically significantly different between time points, $F(2, 16) = 3.496$, $p = 0.055$, partial $\eta^2 = 0.304$. For the older men, thermoregulatory behaviour was not statistically significantly different between time points, $F(2, 18) = 0.792$, $p = 0.468$, partial $\eta^2 = 0.081$.

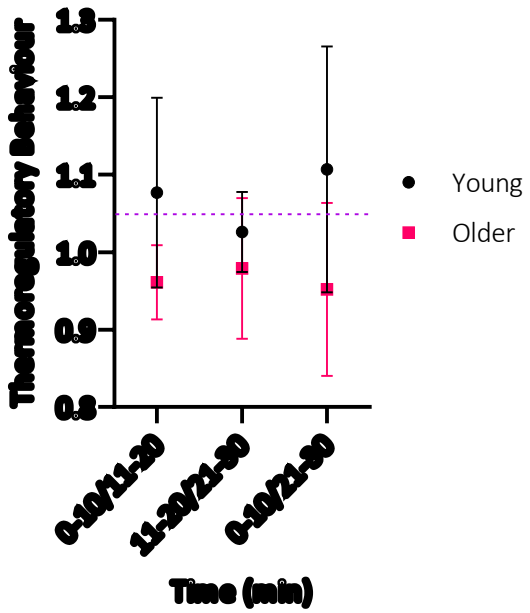

Figure 14-11. *Thermoregulatory behaviour of young and older men throughout the CHC trial by comparing the middle to the first 10 min of cycling, the middle to the final 10 min of cycling, and the final to the first 10 min of cycling. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output as time increased.*

14.3.3 Thermal Comfort

A three-way mixed ANOVA was run to understand the effects of trial condition, time, and age on thermal comfort (**Figure 14-12**). Thermal comfort scores were normally distributed, as assessed by Shapiro-Wilk's test (p >0.05). There were 17 outliers, as assessed by inspection of a boxplot. The outliers were kept in the analysis as they did not materially affect the results, as assessed by comparison of the results with and without the outliers. There was homogeneity of variances, as assessed by Levene's test for equality of variances (p >0.05). Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the three-way interaction effect ($\chi^2(20) = 33.674$, $p = 0.031$). Therefore, the Greenhouse-Geisser correction was applied.

There was a statistically significant three-way interaction effect of time point, age, and trial condition on thermal comfort, $F(3.244, 55.142) = 3.649$, $p = 0.016$, partial $\eta^2 = 0.177$, $\varepsilon = 0.541$. Therefore, simple two-way interactions were examined. Statistical significance of a simple two-way interaction was accepted at a Bonferroni-adjusted alpha level of 0.025. Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction effect in the young men ($\chi^2(20) = 37.329$, $p = 0.020$). Therefore, the Greenhouse-Geisser correction was applied.

The simple two-way interaction between trial condition and time for the young men was statistically significantly different, $F(1.946, 15.568) = 9.827$, $p = 0.002$, partial $\eta^2 = 0.551$, $\varepsilon = 0.324$. Mauchly's test of sphericity indicated that the assumption of sphericity was met for the two-way interaction effect in the older men ($\chi^2(20) = 22.204$, $p = 0.383$). The simple two-way interaction between trial condition and time for the young men was statistically significantly different, $F(6, 54) = 7.120$, p <0.001, partial $\eta^2 = 0.442$. Simple simple main effects were then run.

Statistical significance of a simple simple main effect was accepted at a Bonferroni-adjusted alpha level of 0.025. The simple simple main effect of trial for young men during the first 10 min of cycling on thermal comfort was statistically significant, $F(3, 24) = 5.418$, $p = 0.005$, partial $\eta^2 = 0.404$. During the CCC trial thermal comfort was 0.4 (95% CI, 0.1 to 0.8), this was statistically significantly lower than the W+CHC trial at 1.1 (95% CI, 0.7 to 1.6), $p = 0.042$.

The simple simple main effect of trial for older men during the first 10 min of cycling on thermal comfort was statistically significant, $F(3, 27) = 6.176$, $p = 0.002$, partial $\eta^2 = 0.407$.

The simple simple main effect of trial for young men during the middle 10 min of cycling on thermal comfort was statistically significant, $F(1.932, 15.459) = 18.700$, p <0.001, partial $\eta^2 = 0.700$. Thermal comfort in the CCC trial (1.0 (95% CI) 0.7 to 1.3) was statistically significantly lower than the CHC (2.2 (95% CI) 1.7 to 2.6, $p = 0.010$) and the W+CHC trial (2.2 (95% CI) 1.7 to 2.6, $p = 0.007$). The CHC trial

(2.2 (95% CI) 1.7 to 2.6) was also statistically significantly higher than the W+CCC trial (1.1 (95% CI) 0.7 to 1.5, $p = 0.022$). The W+CCC trial (1.1 (95% CI) 0.7 to 1.5) was statistically significantly lower than the W+CHC trial (2.2 (95% CI) 1.7 to 2.6, $p < 0.001$).

The simple simple main effect of trial for older men during the middle 10 min of cycling on thermal comfort was statistically significant, $F(3, 27) = 3.190$, $p = 0.040$, partial $\eta^2 = 0.262$.

The simple simple main effect of trial for young men during the final 10 min of cycling on thermal comfort was not statistically significant, $F(3, 24) = 1.508$. $p = 0.238$, partial $\eta^2 = 0.159$.

The simple simple main effect of trial for older men during the final 10 min of cycling on thermal comfort was statistically significant, $F(3, 27) = 4.457$. $p = 0.011$, partial $\eta^2 = 0.331$.

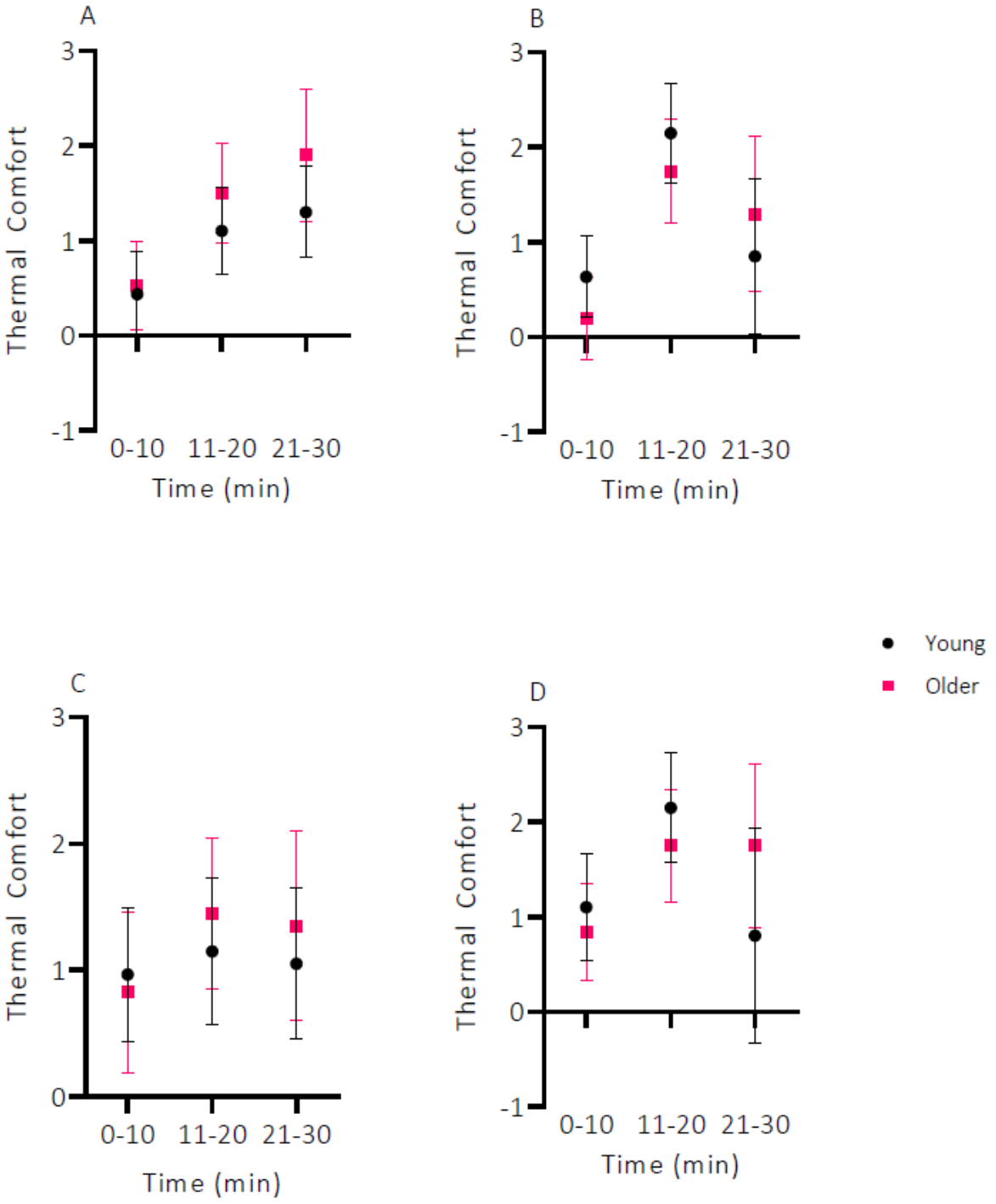

***Figure 14-12**. Thermal comfort of young and older men at the first, middle, and final 10 min of each trial. A = CCC trial, B = CHC trial, C = W+CCC trial, D = W+CHC trial. Data presented as mean ± SD.*

14.4 Discussion

The young men responded to pre-warming (CCC trial compared to W+CCC trial) by reducing their power output resulting in a high thermoregulatory behaviour score (1.10 ± 0.12) during the initial 10 min of cycling. whereas the older men maintained the same power output despite the 1.0°C warmer rectal temperature, resulting in a thermoregulatory behaviour score of 1.00 ± 0.13. The age groups displayed similar responses when comparing the CCC to the CHC trials and the CCC compared with the W+CHC. When examining thermoregulatory behaviour within each trial the age groups differ in the CCC, CHC, and W+CHC trials. The older men increased power output as time increased during the CCC trial while the young men decreased. The young men responded to the ambient temperature increase during the CHC and the W+CHC trials by reducing power output (thermoregulatory behaviour scores: CHC 1.09 ± 0.12, W+CHC 1.08 ± 0.16) while the older men increased power output (thermoregulatory behaviour scores: CHC 0.94 ± 0.09, W+CHC 0.97 ± 0.07).

This was the first study to examine thermoregulatory behaviours during exercise in changing ambient conditions with older men. We utilised radiant heat lamps during cycling exercise to identify thermoregulatory responses to increases in skin temperature. This step was undertaken to manipulate skin temperature while minimising their impact on rectal temperature. Radiant heat lamps have been used previously with young (~30 years) men rowing for 5,000 m at a fixed RPE of 15 in changing ambient conditions (radiant heat lamps turned on at 1,000 – 2,000 m and 3,000 – 4,000 m distance; Lander et al., 2014). Skin temperature increased by 0.51°C during the first warming phase, and power output dropped by 6% ($p = 0.03$). During the second warming phase skin temperature increased by 0.15°C, power output reduced by 4% ($p = 0.10$. Lander et al., 2014). In our study the heat lamps caused a ~5°C and a ~3°C increase in skin temperature for both age groups in the CHC and W+CHC trials, respectively, to ~35°C. This caused significantly different thermoregulatory behavioural responses in the young and older men in the CHC trial. The young men reduced their power output when the radiant heat lamps were activated. This was a similar response as was reported by Lander et al. (2014). The older men however, increased their power output when the radiant heat lamps were active and skin temperature increased. Their thermal comfort also increased so they felt warmer but did not reduce their metabolic heat load. This is an important finding as it appears that older men ignore or unable to respond to drivers of thermoregulatory behaviour.

This has public health implications, especially given that adults aged 65 years and over are vulnerable during heat wave events. The inability of older men to respond to changing skin temperature and thermal perception may prevent them from mitigating their heat risk via compensatory thermoregulatory behaviours. This effect may add to the strain on health services during heat wave

events, and in the aftermath, as those who suffer severe heat illness/stroke may lose physical independence as a result. Advice given during heat wave events should include guidance that during exercise in the heat older men are unable to voluntarily respond to increases in skin temperature, so should be mindful to reduce activity levels even if they do not perceive themselves to be at risk.

Previous work using a fixed RPE cycling protocol indicated young men (<35 years) drop to 70% of their starting power output in ~22 min (Flood et al., 2017), and ~33 min (Parton et al., 2021), albeit using an RPE of 16 (between 'hard' and 'very hard'). We expected that power output would decline during all the trials, but at differing rates. The younger men behaved as anticipated, their power output declined from the first to the last 10 min in the CCC trial (thermoregulatory behaviour 1.07 ± 0.15). Whereas the older men maintained their power output throughout the trials with little effect of time, pre-warming, or changing ambient conditions. In the CCC trial the older men increased power output from the first to the final 10 min (thermoregulatory behaviour 0.94 ± 0.10). It appears that older men do not voluntarily adjust their power output in response to an elevated rectal temperature, to rapid increases in ambient and skin temperatures, or to increases in thermal comfort and thermal perception. This inability to reduce metabolic heat load during heat exposure will put them at an elevated risk of heat illness during heat wave events that are becoming more severe and more common.

The pre-warming trials in the present study caused a ~2°C warmer mean skin temperature, for both age groups, at the onset of exercise than the non-pre-warmed trials (~32°C v ~30°C), alongside the 1°C increase in rectal temperature. Skin temperature at the onset of exercise is known to influence self-selected exercise intensity in young (30 ± 9 years) men during a 60 min cycling time trial (Schlader et al., 2011b). In the present study young men had a lower cycling power output at the start of the cycling exercise when skin and rectal temperature was elevated prior to exercise (thermoregulatory behaviour value: CCC/W+CCC = 1.10 ± 0.12; CHC/W+CHC = 1.12 ± 0.27). The older men did not respond to an increased skin and rectal temperature at the onset of the W+CCC and W+CHC trials as their cycling power output was not different from the CCC and CHC trials, respectively (thermoregulatory behaviour value: CCC/W+CCC = 1.00 ± 0.13; CHC/W+CHC = 1.04 ± 0.11). These findings differ to those of Schlader et al. (2011b) who reported that at the onset of exercise, skin temperatures of ~35°C resulted in a 2.4% reduction in exercise intensity compared with skin temperatures of ~29°C (Schlader et al., 2011b). A mean skin temperature of 36.3°C resulted in resting young (~22 years) adults implementing cooling behaviours to a greater extent than when skin temperature is 34.5°C (Vargas et al., 2018b). In contrast to Vargas et al. (2018b), thermoregulatory behaviours were not implemented by the older group in the present study, despite a greater increase in mean skin temperature (2.0°C v 1.8°C). This difference in findings may be due to the lower absolute mean skin temperatures induced

in our study. Vargas et al. (2018b) and Schlader et al. (2011b) were examining skin temperatures ~35°C, whereas we induced mean skin temperatures of ~32°C prior to the cycling exercise. However, the radiant heat lamps used in the CHC and W+CHC trials did cause mean skin temperature to rise to ~35°C during exercise for both the young and older men, yet the older men still did not implement thermoregulatory behaviours. While the young men implemented thermoregulatory behaviours in the W+CCC trial with a ~2°C increase in mean skin temperature, rectal temperature was also increased by 1°C. It is unclear if a ~2°C increase in mean skin temperature is sufficient to result in thermoregulatory behaviour implementation without the additional stimulus of an increased in core temperature.

The young and older men had similar rectal and mean skin temperature responses throughout the study. During the cycling exercise in the CCC and CHC trials rectal temperature increased for both groups. However, in the pre-warmed trials (W+CCC and W+CHC), rectal temperature remained stable once the participants exited the hot water (pre-exercise ~38.1°C, post-trial ~38.2°C), cycling cause no further increase in rectal temperature. Our data suggest that when men, regardless of age, exercise at a fixed RPE of 13 for 30 min, they do not produce enough metabolic heat to increase core temperature above ~38°C. An elevated rectal temperature at the onset of exercise did not cause thermoregulatory behaviours to be implemented in the older men. The young men had a mixed response, they reduced power output in the first 10 min of the W+CCC trial compared to the CCC trial, but did not adjust power output in the W+CHC trial compared to the CHC trial. Our data suggests that rectal temperature at the onset of exercise does not drive thermoregulatory behaviours in older men and has a limited influence in young men. During fixed intensity cycling (50% $\dot{V}O_2$peak), young men (24 ± 3 years) reported greater RPE scores when their oesophageal temperature was 37.5°C at the start of exercise than when it was 36.9°C (Tsuji et al., 2012). Thus, it was anticipated that an elevated rectal temperature prior to exercise would result in thermoregulatory behaviours being implemented via reduced power outputs.

It has consistently been shown that older men are less sensitive to changes in ambient temperature. When at rest, men aged 60 years and over feel more thermally comfortable in changing ambient temperatures than young men, and when given control of a dual position thermostat, allow greater fluctuations of ambient temperature (Collins et al., 1981; Natsume et al., 1992; Taylor et al., 1995). Older men (66-75 years) have also been shown to feel more comfortable than young men (22-27 years) after exiting a hot bath (42°C) and sitting at rest in 20°C conditions (Ono et al., 2017). Older men have a greater range for thermo-neutrality, and they need to experience a greater thermal input before they initiate thermoregulatory behaviours. This appears to be the case during exercise as well. The older men felt warmer than the young men in the CCC trial yet increased their cycling power

output throughout this trial. Both age groups experienced similar thermal perception at the onset of the trials and during the radiant heat lamp exposures. Yet during the CHC trial the older men increased cycling power output, while the young men reduced cycling power output when the radiant heat lamps were active. This study indicates that older men are less sensitive than young men to changes in thermal perceptions while exercising in stable and changing ambient conditions.

Aerobic fitness and body composition are mediators of heat illness risk, a greater BMI and proportion of body fat, and lower aerobic fitness increases risk of heat illness. (Westwood et al., 2020). In the present study, the young and older men had similar anthropometry, with stature, body mass and body composition being similar between the young and older men. However, the young men tended to have a greater body mass, BMI, and sum of skinfolds. This was due to two of the younger participants having a body mass of 110 kg and 121 kg, whereas the heaviest older man was 93 kg. As a higher body mass and BMI is associated with a greater risk of heat illness, the older men may have been at a slight advantage in the present study. Despite the anthropometric outliers, both age groups had similar fitness levels as the total power output in the CCC trial did not differ between the age groups (young = 510 W, older = 3171 W. $p = 0.477$). Age was the only baseline measurement that differed between the age groups. Thus, it seems that the impaired thermoregulatory behavioural response is an age-related decline rather than associated with body composition or fitness level.

Experimental Chapter Three was designed to try and increase the amount of heat stress experienced by the participants as Experimental Chapter One: Relationship between age and ability to implement thermoregulatory behaviours in thermoneutral and hot ambient conditions and Experimental Chapter Two: Physiological and perceptual factors influencing behavioural thermoregulation in young and older men in thermoneutral and hot ambient conditions had only caused rectal temperature to reach ~38°C at the end of the exercise in the hot trials. The implementation of pre-warming with hot water immersion caused rectal temperature to reach ~38°C prior to the exercise starting. This outcome was achieved despite differing transfer times from the bath to the cycle ergometer for individual participants. They were allowed to exit the bath and change clothes at their own pace to reduce the risk of fainting and injury after being immersed in hot (~42°C) water. However, rectal temperature did not increase further in either the W+CCC or W+CHC trials for either the young or older men. It appears that when men exercise at an RPE of 13, rectal temperature plateaus at ~38°C.

A limitation of the present study was that participants could not be blinded to the heat lamps being turned on or off. The rapid increase in ambient temperature allowed them to perceive this increase relatively soon after they were turned on. However, participants were not provided with any feedback on their power output, speed, cadence, or heart rate, and were unaware of the purpose of the study.

14.5 Conclusion

We demonstrated that when skin and rectal temperature are elevated prior (CCC compared with W+CCC) to exercise young men implement thermoregulatory behaviours by reducing power output, whereas older men do not implement these behavioural responses. However, this was a mixed response as the W+CHC trial did not result in thermoregulatory behaviours being implemented at the onset of exercise (CCC compared with W+CHC). During changing ambient conditions, young men are able to implement thermoregulatory behaviours in response to ambient temperature increases, whereas older men are not. These outcomes are evident despite younger and older men in this study having similar anthropometry (height, body mass, body composition) and physical fitness characteristics. There was also no difference in skin or rectal temperature response between the age groups, indicating that older men are less sensitive to changes in skin temperature. Public health advice should emphasise that being aged 60 years or above alone is a risk factor for heat-related illness, even in individuals with no pre-existing health issues.

15 General Discussion

15.1 Key Findings

No work has previously systematically investigated how adults aged >60 years implement pacing behaviours during fixed perception of exercise in the heat. Thermoregulatory behaviours provide effective and simple ways of reducing heat strain in different cohorts in a wide variety of community and sporting settings. Individuals in a free-living situation voluntarily implement thermoregulatory behaviours, consequently, if individuals are unable to perceive when they should be making these behavioural adjustments the benefits of thermoregulatory behaviours are not felt. This thesis is the first to investigate behavioural thermoregulation during exercise in the heat across the adult lifespan. We found men aged ≥60 years have a diminished ability to implement thermoregulatory behaviours during walking exercise in 35°C conditions. Our data indicates that increasing age also reduces thermoregulatory behavioural ability in women until the age of ~65 years; more work is required to identify if increasing age beyond 65 years further reduces thermoregulatory behaviour in women. We also identified that the decrement in behavioural thermoregulation occurs in older men despite similar rectal and skin temperature, perceptual, sweat, and cardiovascular responses between young and older men. Increases in rectal temperature drive a reduction in total work done for young men, but older men maintain total work done despite increased rectal temperature. The hypotheses and outcomes for each experimental chapter are presented in Table 15-1**Error! Reference source not found.**.

Experimental Chapter One: Relationship between age and ability to implement thermoregulatory behaviours in thermoneutral and hot ambient conditions employed a regression analysis to reveal the impact of ageing across the adult lifespan on thermoregulatory behaviour. This was the first study to include adults in the 3rd – 9th decade of life to identify the pattern of the relationship between age and thermoregulatory behaviour. The use of a fixed RPE protocol with older adults was novel, it allowed participants to voluntarily self-select their exercise intensity and display their thermoregulatory behaviours. We found that men experience a near linear decline in thermoregulatory behaviour from 30 years to 86 years. Men over the age of 60 years were less able to reduce their walking speed in 35°C conditions than men aged 18-30 years. Dividing the male participants into young (18-30 years), middle-aged (41-60 years), and older (>60 years) cohorts revealed that the decline in thermoregulatory behaviour is significant in older men, but not middle-aged when compared with young men. With this finding we used ≥60 years as the threshold for older men in our subsequent studies. Experimental Chapters Two and Three went on to investigate potential causes for this impaired behavioural thermoregulation. We recommend that future work examining

thermoregulatory behaviours and ageing should consider men aged ≥60 years as older age and likely having an impaired behavioural thermoregulation. There also appears to be a sex difference in the relationship between age and thermoregulatory behaviours, however, this relationship was skewed by two anomalous female participants. These two participants were two of the oldest (71 and 78 years) yet implemented behavioural thermoregulation particularly well. Our data revealed that thermoregulatory behaviour in the women declines similarly to the men until a plateau at around the age of 65 years. Further work with a greater number of female participants aged ≥70 years is required to clarify this finding. Men aged ≥60 years have an impaired ability to implement thermoregulatory behaviours during heat exposure. This will lead to a greater level of heat strain experienced by this population. Further work is needed to elucidate factors causing this impairment, and whether thermoregulatory behaviours can be implemented more effectively by older men.

Experimental Chapter Two: Physiological and perceptual factors influencing behavioural thermoregulation in young and older men in thermoneutral and hot ambient conditions focussed on male participants and factors causing the reduction in ability to use thermoregulatory behaviours. Both the younger and older men had similar stature, body mass, body mass index, body fat percentage, 6-minute walk test, and physical activity scale for the elderly scores. The skin and rectal temperature, heart rate, mean arterial pressure, and whole body sweat rate responses were also similar between the age groups. Skin blood flow only differed between the age groups at the end of the 22°C control trial with the younger men having a higher forearm skin blood flow than the older men. While the older men have similar physiological and perceptual responses to young men during walking at an RPE of 13 in 35°C, they were unable to voluntarily implement thermoregulatory behaviours as effectively as young men. The similarities in anatomy and fitness, physiological, and perceptual responses indicates that the reduction in thermoregulatory behaviours is an age-related decline. Rectal temperature at the end of the 35°C trial reached 38.1°C for the young group and 37.8°C for the older group. While this was a ~1.0°C increase from the start of the trial, this is still a modest final rectal temperature. A greater level of heat strain may be required for older men to initiate thermoregulatory behaviours.

Experimental Chapter Three: Thermoregulatory behaviour during fixed RPE cycling in changing ambient conditions in young and older men

15.2 Introduction

Experimental Chapter One: Relationship between age and ability to implement thermoregulatory behaviours in thermoneutral and hot ambient conditions identified that thermoregulatory behaviour is impaired in older adults, particularly men, and established that men aged ≥60 years do not reduce

their walking speed when exposed to 35°C ambient conditions. The mechanisms responsible for this self-regulated behaviour remain unclear. Experimental Chapter Two: Physiological and perceptual factors influencing behavioural thermoregulation in young and older men in thermoneutral and hot ambient conditions could not identify a physiological or perceptual causative factor for the impairment in behavioural thermoregulation but did identify that older men are less able to implement thermoregulatory behaviours than young men. Understanding the contribution of skin and core temperature to thermoregulatory behaviours could elucidate possible mechanisms behind the decline in behavioural thermoregulation in older men.

Peak rectal temperature during the hot (35°C ambient) trials in Experimental Chapters One and Two reached ~38.0°C for all participants. While this was a ~1°C increase from the onset of the trials, core temperature of 38 – 40°C are not abnormal during exercise, particularly in hot conditions (Gleeson, 1998). Core temperature of young (~30 years) men performing 5,000 m of rowing at a fixed RPE of 15 ('hard') did not differ between ~35°C and 18°C ambient conditions with final core temperatures being ~38.5°C in both conditions (Lander et al., 2014). Thus, young men experience similar core temperature increases while exercising at a fixed RPE in temperate and hot conditions. Older men might require a higher rectal temperature before they will voluntarily initiate thermoregulatory behaviours. Hot water immersion at 40°C has been shown to induce a greater thermal challenge than exercising for the same amount of time at 50% of $\dot{V}O_2$max in 40°C (Greenfield et al., 2021). Experimental Chapter Three will employ hot water immersion to pre-warm participant's rectal temperature by 1°C before they undertake fixed RPE cycling for 30 min. This approach will permit examination of thermoregulatory behaviour when participants are experiencing heat strain prior to the onset of exercise, rather than using the exercise to induce heat strain.

Skin temperature mediates thermoregulatory behaviour (Schlader et al., 2009, 2013). However, the evidence from Experimental Chapter One: Relationship between age and ability to implement thermoregulatory behaviours in thermoneutral and hot ambient conditions Experimental Chapter Two: Physiological and perceptual factors influencing behavioural thermoregulation in young and older men in thermoneutral and hot ambient conditions indicates that skin temperature either has little influence on thermoregulatory behaviour in older men exercising in the heat, or older men have a reduced sensitivity to increases in skin temperature. The young men experienced skin temperature during the hot trials of 35.24°C and 35.22°C in experimental chapters one and two respectively, while the older men experienced skin temperatures of 35.40°C and 35.68°C. This was an increase from the control trials of 3.45°C and 3.36°C in experimental chapters one and two respectively, for the young men, and 4.48°C and 4.60°C for the older men. Young (~30 years) men reduce rowing power output when skin temperature increases by 0.5°C under changing ambient conditions (Lander et al., 2014).

During a 5,000 m rowing trial where RPE was fixed at 15 ('hard'), radiant heat lamps were used to increase the ambient temperature from 18 to 35°C from the 1,000 to 2,000 m and 3,000 to 4,000 m distances. Each 1,000 m took ~4 min to complete, so the heat lamps were activated for ~4 min during each warming period. Skin temperature rose ~0.5°C and power output declined by 6% during the first heat exposure (Lander et al., 2014). Young men were able to initiate thermoregulatory behaviours within 4 min of a change in ambient conditions. There is a dearth of research investigating thermoregulatory behaviours in exercising older adults during heat stress. Using heat lamps to alter the ambient temperature provides a way of rapidly inducing skin temperature changes in exercising individuals. It also allows for the examination of the effect of skin temperature, independently of core temperature, on thermoregulatory behaviours as skin temperature will change more rapidly than core temperature. We will employ this radiant heat lamp method in the present study to try and elucidate the influence of skin temperature on thermoregulatory behaviour in men aged ≥60 years.

There are contrasting models of how self-selected exercise intensity, or pacing, is controlled. The psychobiological model proposes that pacing is controlled consciously (Marcora, 2010), whereas the central governor theory suggests that motor units are controlled subconsciously (St Clair Gibson et al., 2006). These two theories are opposed, and it has been postulated that pacing is a multidimensional process requiring both conscious and subconscious, or sub-awareness input (Edwards & Polman, 2013; Micklewright et al., 2017). The treadmill protocol utilised in Experimental Chapters One and Two prevented any subconscious alterations in walking speed as participants had to consciously decide to push a button to alter the treadmill speed. Allowing participants to unknowingly adjust their exercise intensity may lead to the implementation of thermoregulatory behaviours sooner, and to a greater extent, than when they must consciously push a button on a treadmill. Cycling ergometer exercise will allow participants to unknowingly and knowingly adjust their exercise intensity by adjusting their pedal cadence. Therefore, the current study, will employ cycling exercise at a fixed RPE.

Pacing strategy during exercise in the heat is thought be anticipatory, implemented as soon as core temperature starts to increase, rather than once a threshold (core) temperature is reached (Tucker & Noakes, 2009). Within 4 min of starting a cycling time trial in 35°C at a fixed RPE of 16 (between 'hard' and 'very hard'), young (~23 years) men implemented pacing strategies that maintained a similar level of heat storage as a 15°C trial (Tucker et al., 2006). Thus, young men can voluntarily implement thermoregulatory behaviours while cycling at a fixed RPE by reducing their power output. Research investigating thermoregulatory behaviour in exercising adults has neglected to include older adults (≥60 years).

The use of pre-warming and changing environmental conditions with older participants will facilitate investigation of the independent and combined effect of rectal and skin temperature on behavioural thermoregulation during fixed RPE exercise. This novel approach should identify whether skin or rectal temperature has a greater influence on thermoregulatory behaviour in older men, or if an increase of both skin and core temperature are required for initiation of thermoregulatory behaviours. We hypothesise that: pre-warming will result in a reduction in total work done for both young and older men; power output will be reduced when the radiant heat lamps are turned on for both young and older men; older men will feel more thermally comfortable than young men; and the older men will experience a greater level of heat strain.

15.3 Methods

15.3.1 Participants

Nineteen healthy males, nine young (19-31 years) and ten older (66-87 years) volunteered to participate in the study. Participants were non-smokers, free from any known respiratory, cardiovascular, or metabolic disease, and not taking any medications known to affect thermoregulation. Participant anthropometric data is presented in **Error! Reference source not found.Error! Reference source not found.**. The experimental protocol was approved by the Plymouth Marjon University Ethics Panel (EP103) in accordance with the Declaration of Helsinki. Volunteers provided written informed consent after explanation of the purpose, benefits, risks, and experimental procedures of the study before participating in the study.

15.3.2 Experimental overview

This study used a group comparison of performance, temperature, and perceptual responses to cycling in 22°C or changing conditions (22-33°C), with and without passive heating before cycling exercise in younger and older men. A self-paced, fixed RPE protocol was used (similar to Lander et al., 2014), with participants completing a familiarisation session to get comfortable with the RPE scale and the cycle ergometer. Anthropometric measurements were also taken in the familiarisation session.

Participants completed four experimental trials consisting of 30 min of cycling at a fixed rating of perceived exertion (RPE) of 13, equivalent to 'somewhat hard' (Borg, 1982). Participants were blinded to their speed, power output and heart rate data, and received no feedback on how far they had cycled during the trial. The control trial consisted of 30 min of cycling in 21.5 ± 0.5°C ambient conditions. Another trial was completed in changing ambient conditions, where radiant heat lamps were activated during 10 – 20 min of 30 min fixed RPE cycling. A third trial was conducted with pre-warming, by immersion in 40 – 42°C water, of rectal temperature by 1°C prior to the 30 min of cycling

in 21.5 ± 0.5°C with no heat lamp activation. The final trial condition included both pre-warming and heat lamp activation during 10 – 20 min of the 30 min fixed RPE cycling. Four radiant heat lamps were positioned around the participant, 1 m away from the centre of the cycle ergometer. Ten min into the trials using the lamps they were turned on for 10 min, resulting in an increase in ambient temperature to 33.3 ± 1.4°C.

Trials were conducted in a randomised order, participants arrived at the same time of day, and with at least 48 h between trials. Participants were also asked to abstain from alcohol, caffeine, non-steroidal anti-inflammatory drugs, and strenuous exercise in the 24 h before trials, and consume 500 mL of water the evening before and 2 h before arriving at the laboratory. Clothing consisted of a t-shirt, shorts, and walking/running shoes. Water was available for *ad libitum* consumption throughout the trials.

Mean power output was calculated in 10 min periods (0-10 min, 11-20 min, and 21-30 min). A marker of thermoregulatory behaviour within a trial was then calculated as:

Equation 14-1*. Thermoregulatory behaviour established as a change in power output as a function of time.*

$$Thermoregulatory\ Behaviour = \frac{Mean\ Power\ Output_{Trial\ A\ Time\ Period\ x}}{Mean\ Power\ Output_{Trial\ A\ Time\ Period\ y}}$$

Between trials thermoregulatory behaviour (CCC v CHC, CCC v W+CCC, CHC v W+CHC, W+CCC v W+CHC) was also compared at each time period and calculated as:

Equation 14-2. *Thermoregulatory behaviour established as a change in power output as a function of trial condition.*

$$Thermoregulatory\ Behaviour = \frac{Mean\ Power\ Output_{Trial\ A\ Time\ Period\ x}}{Mean\ Power\ Output_{Trial\ B\ Time\ Period\ x}}$$

This index gave a ratio of power output within a trial as duration increased, and between trials with different ambient and pre-warming conditions. A result >0 indicated that the participant reduced power output between conditions being compared, <0 they increased power output between conditions being compared, =0 there was no difference between conditions being compared.

15.3.3 Experimental procedures

In the preliminary visit, anthropometric measures (stature, body mass, skinfolds) were taken as described in the General Methods. Participants were then familiarised with the RPE scale. Participants were asked to adjust the cycle ergometer (Wattbike Pro/Trainer, Wattbike, Nottingham, UK) so that they were comfortable on the bike. The seat and handlebar positions were recorded for use in all

subsequent trials. Next, participants self-selected a resistance on the cycle ergometer that elicited an RPE of 13, 'somewhat hard' while blinded to their power output and speed. After 3 min participants confirmed that the resistance settings felt 'somewhat hard', and their power output was recorded (but not revealed to the participant) before they sat at rest for 5 min. Participants repeated this procedure until they were able to replicate a power output that elicited an RPE of 13. The resistance settings were recorded for use in subsequent trials.

During hot water immersion both rectal and water temperature were monitored continuously. Heart rate was recorded every 5 min. Water was available to drink *ad libitum.* Once rectal temperature had increased by 1°C, participants were removed from the hot water bath and given privacy while they changed into dry clothing, the rectal thermistor remained in place. Participants then entered the environmental chamber where skin thermistors were attached as described in the General Methods (Chapter 11.9.2 Skin Temperature). An ambient temperature thermistor was hung from the ceiling ~30 cm above the participant's head. Temperature data was collected as described in the General Methods (Chapter 11.9 Temperature). Participants then mounted the cycle ergometer and began the 30 min cycling trial. Heart rate and power output were recorded at ~1 sec intervals by the Wattbike. These data were then averaged into 10 min blocks. RPE was assessed every 5 min.

15.3.4 Statistical analysis

For *a priori* power analysis, conventional α (0.05) and β (0.20) parameters and a large effect size (1.3), based on data from Experimental Chapter Two were employed. The estimations indicated that 9 subjects per group was appropriate to detect meaningful differences. Power analysis was performed using G*Power (G*Power, Düsseldorf, Germany).

Data is presented as mean ± standard deviation, unless otherwise stated. The level of significance for all analyses was set at $p \leq 0.05$. Effect size was defined as small $d = 0.20$, partial $\eta^2 = 0.01$; medium $d = 0.50$, partial $\eta^2 = 0.06$; and large $d = 0.80$, partial $\eta^2 = 0.14$ (Cohen, 1988; Cohen 1969). All statistical analyses were conducted using Statistics Package for the Social Sciences (SPSS v28, IBM Corp, Armonk, NY).

15.4 Results

15.4.1 Participant characteristics and temperature responses

Participant characteristics are presented in **Table 14-1**.

***Table 14-1**. Participant anthropometric data. Data presented as mean ± SD. Bold denotes significantly different from the young group.*

	n	Age (years)	Stature (cm)	Body Mass (kg)	Body Mass Index	Σskinfolds (mm)
Young	9	24 ± 4	181 ± 5	86.9 ± 19.6	26.6 ± 5.7	47.1 ± 29.2
Older	10	**70 ± 7**	176 ± 5	73.8 ± 9.0	23.8 ± 2.6	33.5 ± 15.3
p value	-	<0.001	0.067	0.077	0.188	0.234

A two-way repeated measures ANOVA was run to determine the effect of trial conditions and time point on ambient temperature (**Figure 14-2**). Analysis of studentised residual showed that there was normality, as assessed by the Shapiro-Wilk test of normality (p >0.05), and no outliers as assessed by no studentised residuals greater than ± 3 standard deviations. Mauchly's test of sphericity indicated that the assumption of sphericity had been violated for the two-way interaction, $\chi^2(2) = 89.831$, p <0.001, therefore a Greenhouse-Geisser correction was applied.

There was a statistically significant two-way interaction between treatment and time, $F(3.145, 56.609) = 246.068$, p <0.001, $\varepsilon = 0.524$. Therefore, simple main effects were run.

Ambient temperature was not statistically significantly different between trials at during the first 10 min of cycling, $F(1.974, 35.535) = 0.726$, $p = 0.489$, $\varepsilon = 0.658$.

There was a statistically significant effect of trial condition on ambient temperature during the middle 10 min of cycling, $F(2.020, 36.368) = 935.734$, p <0.001, $\varepsilon = 0.673$. Ambient temperature was warmer in the CHC (33.24 (95% CI) 32.53 to 33.95°C) and W+CHC (33.39 (95% CI) 32.76 to 34.02°C) trials than the CCC (21.48 (95% CI) 21.28 to 21.70°C) and W+CCC (21.46 (95% CI) 21.20 to 21.72°C) trials.

There was a statistically significant effect of trial condition on ambient temperature during the final 10 min of cycling, $F(1.995, 35.911) = 13.631$, p <0.001, $\varepsilon = 0.665$. Ambient temperature was warmer in the CHC (23.53 (95% CI) 22.60 to 24.45°C) and W+CHC (23.92 (95% CI) 22.83 to 25.02°C) trials than the CCC (21.58 (95% CI) 21.36 to 21.80°C) and W+CCC (21.54 (95% CI) 21.28 to 21.80°C) trials.

There was a statistically significant effect of time on ambient temperature during the control trial (CCC), $F(2, 36) = 7.218$, $p = 0.002$. Ambient temperature was warmer during the first 10 min (21.80 (95% CI) 21.55 to 22.05°C) than the middle (21.48 (95% CI) 21.28 to 21.69°C, $p = 0.001$) and final 10 min of cycling (21.58 (95% CI) 21.36 to 21.80°C, $p = 0.050$).

There was a statistically significant effect of time on ambient temperature during the no pre-warming, heat lamp trial (CHC), $F(2, 36) = 336.332$, p <0.001. Ambient temperature was warmer during the middle 10 min (33.24 (95% CI) 32.53 to 33.95°C) than the first (21.97 (95% CI) 21.68 to 22.27°C, p

<0.001) and final 10 min of cycling (23.53 (95% CI) 22.60 to 24.45°C, p <0.001). The final 10 min of cycling were also warmer than the first 10 min of cycling, p = 0.006.

There was a statistically significant effect of time on ambient temperature during pre-warming, no heat lamps trial (W+CCC), $F(2, 36) = 40.230$, p <0.001. Ambient temperature was warmer during the first 10 min (22.03 (95% CI) 21.79 to 22.28°C) than the middle (21.46 (95% CI) 21.20 to 21.71°C, p <0.001) and final 10 min of cycling (21.54 (95% CI) 21.28 to 21.80°C, p <0.001).

There was a statistically significant effect of time on ambient temperature during the pre-warming, and heat lamp trial (W+CHC), $F(2, 36) = 302.170$, p <0.001. Ambient temperature was warmer during the middle 10 min (33.39 (95% CI) 32.76 to 34.02°C) than the first (21.74 (95% CI) 21.18 to 22.30°C, p <0.001) and final 10 min of cycling (23.92 (95% CI) 22.83 to 25.02°C, p <0.001). The final 10 min of cycling were also warmer than the first 10 min of cycling, p = 0.002.

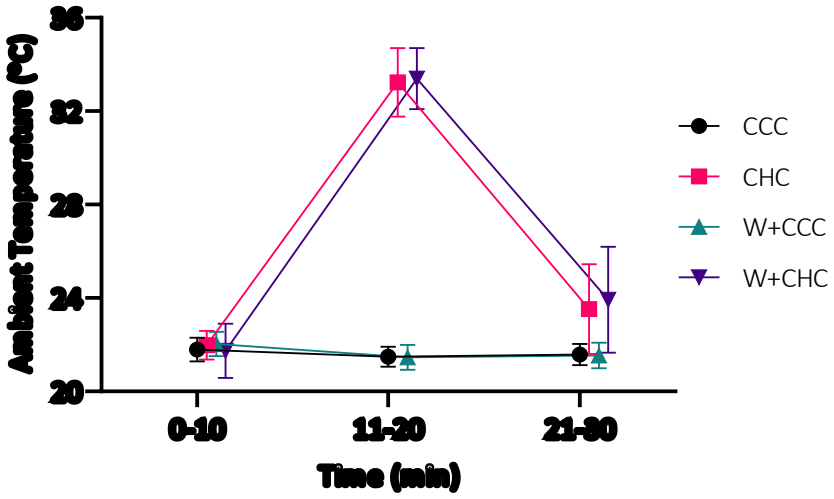

***Figure 14-2**. Ambient temperature for the first, middle, and final 10 min of cycling in each trial. Data presented as mean ± SD.*

A three-way mixed ANOVA was run to examine the effects of age, trial condition, and time point on mean skin temperature (**Figure 14-3**). There were three outliers, as assessed by inspection of a boxplot for values greater than 3 box-lengths from the edge of the box. These were kept in the analysis as they did not materially affect the result. Mean skin temperature data was normally distributed (p >0.05), except for the final 10 min of cycling in the control trial for the older men (p = 0.023), as assessed by Shapiro-Wilk's test. There was homogeneity of variances, as assessed by Levene's test for equality of variances (p >0.05). Mauchly's test of sphericity indicated that the assumption of sphericity was violated, $\chi^2(20) = 35.580$, p = 0.020. Therefore, the Greenhouse-Geisser correction was applied.

There was no statistically significant three-way interaction between age, trial condition, and time point on mean skin temperature, $F(3.792, 56.886) = 1.258$, $p = 0.297$, partial $\eta^2 = 0.077$, $\varepsilon = 0.632$. There was no statistically significant two-way interaction between trial condition and age on mean skin temperature, $F(1.980, 29.699) = 1.159$, $p = 0.327$, partial $\eta^2 = 0.072$. There was no statistically significant two-way interaction between time point and age on mean skin temperature, $F(1.331, 19.970) = 1.205$, $p = 0.302$, partial $\eta^2 = 0.074$. There was a statistically significant two-way interaction between trial condition and time point on mean skin temperature, $F(3.792, 563886) = 130.462$, $p < 0.001$, partial $\eta^2 = 0.897$.

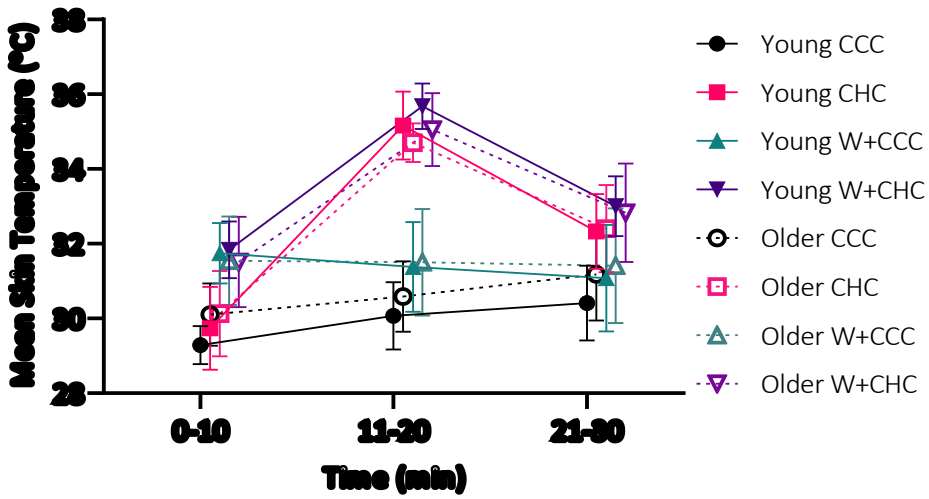

***Figure 14-3**. Mean skin temperature during the first, middle, and final 10 min of cycling for each trial in the young and older men. Data presented as mean ± SD.*

A three-way mixed ANOVA was run to examine the effects of age, trial condition, and time point on rectal temperature (**Figure 14-4**). There were no outliers, as assessed by inspection of a boxplot for values greater than 3 box-lengths from the edge of the box. Rectal temperature data was normally distributed, as assessed by Shapiro-Wilk's test ($p > 0.05$). There was homogeneity of variances, as assessed by Levene's test for equality of variances ($p > 0.05$). Mauchly's test of sphericity indicated that the assumption of sphericity had been violated for the two-way interaction, $\chi^2(2) = 86.545$, $p < 0.001$, therefore a Greenhouse-Geisser correction was applied.

There was no statistically significant three-way interaction between age, trial condition, and time point on rectal temperature, $F(3.258, 58.650) = 2.692$, $p = 0.050$, partial $\eta^2 = 0.130$, $\varepsilon = 0.543$. There was a statistically significant two-way interaction between trial condition and age on rectal temperature, $F(2.831, 50.960) = 3.383$, $p = 0.027$, partial $\eta^2 = 0.158$. There was no statistically significant two-way interaction between time and age on rectal temperature, $F(1.012, 18.214) = 1.793$,

p = 0.197, partial η^2 = 0.091. There was a statistically significant two-way interaction between trial condition and time point on rectal temperature, F(3.258, 58.650) = 56.403, p <0.001, partial η^2 = 0.758.

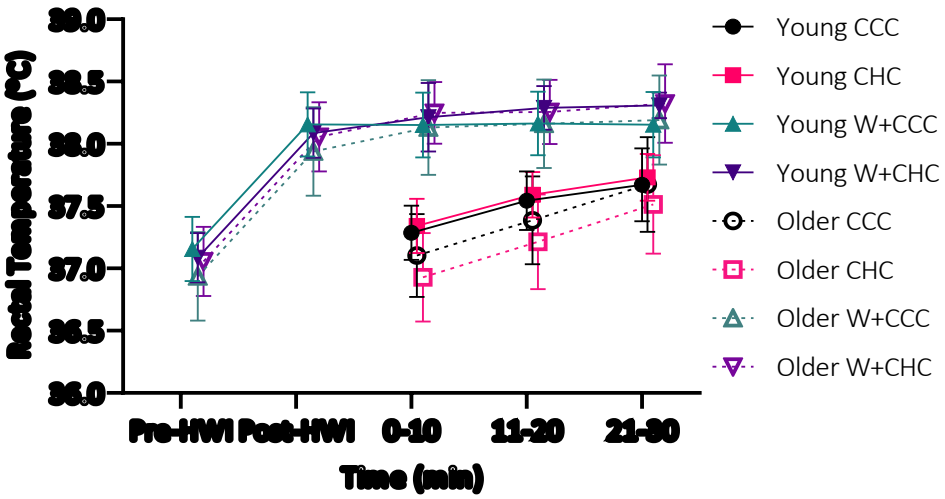

Figure 14-4*. Rectal temperature during the trials for the young and older men. HWI = hot water immersion. Data presented as mean ± SD.*

15.4.2 Thermoregulatory behaviour

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour when comparing the CCC and CHC trials (**Figure 14-5**). There was one outlier, which had a studentised residual value of 3.05. This outlier was kept in the analysis as it did not materially affect the results. Thermoregulatory behaviour data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variances, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's test of equality of covariance matrices (p = 0.078). Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi^2(2)$ = 8.115, p = 0.017. Therefore, the Greenhouse-Geisser correction was applied.

There was no statistically significant two-way interaction between time point and age on thermoregulatory behaviour, F(1.431, 24.324) = 1.496, p = 0.241, partial η^2 = 0.081, ε = 0.715. The main effect of time point showed a statistically significant difference in thermoregulatory behaviour at different time points, F(1.431, 24.324) = 5.098, p = 0.023, partial η^2 = 0.231. The final 10 min of cycling (0.97 (95% CI) 0.90 to 1.03) had a higher thermoregulatory behaviour score than the first 10 min of cycling (1.05 (95% CI) 0.99 to 1.10) p = 0.031). Thermoregulatory behaviour score for middle 10 min of cycling (0.99 (95% CI) 0.91 to 1.07) did not statistically significantly differ from either the first (p = 0.349) or the final (p = 0.289) 10 min of cycling. The main effect of age showed no statistically

significant difference between the young (1.01 (95% CI) 0.93 to 1.10) and older (0.99 (95% CI) 0.91 to 1.07) men in thermoregulatory behaviour score, $F(1, 17) = 0.107$, $p = 0.748$, partial $\eta^2 = 0.006$.

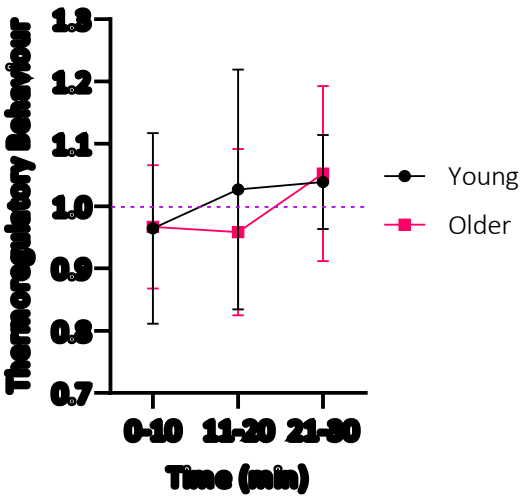

Figure 14-5. *Thermoregulatory behaviour of young and older men during the first, middle, and final 10 min of cycling when comparing the CHC trial to the CCC trial. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output in the CHC trial compared to the CCC trial.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour when comparing the CCC and W+CCC trials (**Figure 14-6**). Data is presented as mean ± standard deviation, unless otherwise stated. There was one outlier, which had a studentised residual value of 3.05. This outlier was kept in the analysis as it did not materially affect the results. Thermoregulatory behaviour data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variances, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's test of equality of covariance matrices (p = 0.092). Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi^2(2) = 11.272$, $p = 0.004$. Therefore, the Greenhouse-Geisser correction was applied.

There was a statistically significant interaction between age and time on thermoregulatory behaviour, $F(1.328, 22.582) = 4.404$, $p = 0.038$, partial $\eta^2 = 0.206$, $\varepsilon = 0.664$. Therefore, simple main effects were examined. There was no statistically significant difference in thermoregulatory behaviour between the age groups during the first 10 min of cycling ($p = 0.121$, partial $\eta^2 = 0.136$), the middle 10 min of cycling ($p = 0.271$, partial $\eta^2 = 0.071$), or the final 10 min of cycling (p = 0.836, partial $\eta^2 = 0.003$). There was no statistically significant effect of time on thermoregulatory behaviour for the young men (p =

0.282, partial η^2 = 0.146). There was a statistically significant effect of time on thermoregulatory behaviour for the older men, $F(2, 18) = 6.033$, $p = 0.010$, partial $\eta^2 = 0.401$.

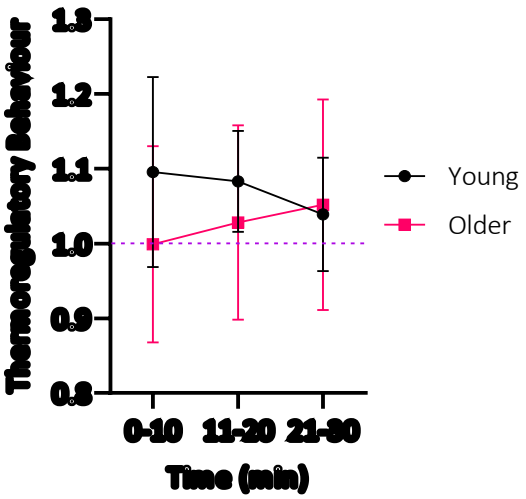

***Figure 14-6**. Thermoregulatory behaviour of young and older men during the first, middle, and final 10 min of cycling when comparing the W+CCC trial to the CCC trial. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output in the W+CCC trial compared to the CCC trial.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour when comparing the CCC and W+CHC trials (**Figure 14-7**). Data is presented as mean ± standard deviation, unless otherwise stated. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. All data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variance, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's equality of variance matrices (p = 0.03). Mauchly's test of sphericity indicated that the assumption of sphericity was met for the two-way interaction, $\chi^2(2) = 4.944$, $p = 0.084$.

There was no statistically significant two-way interaction between time point and age on thermoregulatory behaviour, $F(2, 34) = 0.949$, $p = 0.397$, partial $\eta^2 = 0.053$. Therefore, main effects were examined. The main effect of time point showed a statistically significant difference in thermoregulatory behaviour at the different time points, $F(2, 34) = 3.519$, $p = 0.041$, partial $\eta^2 = 0.171$. The main effect of age group showed that there was no statistically significant difference in thermoregulatory behaviour between the young and older men, $F(1, 17) = 0.713$, $p = 0.410$, partial η^2 = 0.040. The first 10 min of cycling (1.02 (95% CI) 0.956 to 1.084) did not significantly differ from the middle (1.06 (95% CI) 1.00 to 1.12, $p = 0.108$) or final 10 min of cycling (1.06 (95% CI) 1.00 to 1.12, p =

0.207). The middle 10 min of cycling was not significantly different to the final 10 min of cycling (p = 0.999).

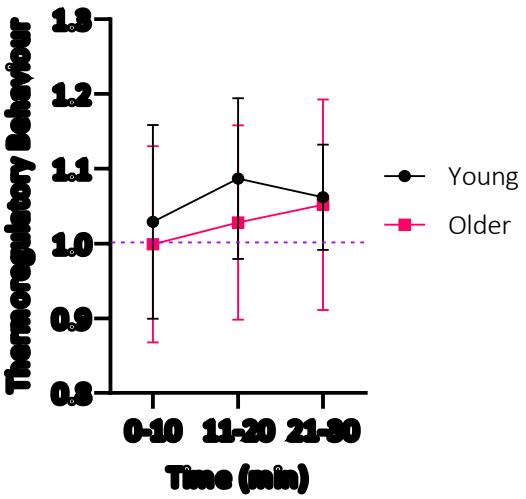

Figure 14-7. *Thermoregulatory behaviour of young and older men during the first, middle, and final 10 min of cycling when comparing the W+CHC trial to the CCC trial. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output in the W+CHC trial compared to the CCC trial.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour during the CCC trial (**Figure 14-8**). Data is presented as mean ± standard deviation, unless otherwise stated. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. All data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variance, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's equality of variance matrices (p = 0.02). Mauchly's test of sphericity indicated that the assumption of sphericity was met for the two-way interaction, $\chi^2(2) = 3.499$, $p = 0.174$.

There was a statistically significant interaction effect of age and time on thermoregulatory behaviour during the CCC trial, $F(2, 34) = 3.813$, $p = 0.032$, partial $\eta^2 = 0.183$. Therefore, simple main effects were run. There was no statistically significant difference in thermoregulatory behaviour between the age groups when comparing the middle 10 min of cycling to the first 10 min of cycling, $F(1, 17) = 4.114$, p = 0.058, partial η^2 = 0.195. The young men implemented statistically significantly better thermoregulatory behaviour (1.05 (95% CI) 1.01 to 1.08) in the final 10 min of cycling when compared to the middle 10 min of cycling than the older men (0.98 (95% CI) 0.94 to 1.01), $F(1, 17) = 7.835$, p = 0.012, partial η^2 = 0.315. The young men implemented statistically significantly better thermoregulatory behaviour (1.07 (95% CI) 0.99 to 1.15) in the final 10 min of cycling compared to the

first 10 min of cycling than the older men (0.94 (95% CI) 0.86 to 1.01), $F(1, 17) = 6.658$, $p = 0.019$, partial $\eta^2 = 0.281$.

For the young men, thermoregulatory behaviour was not statistically significantly different between time points, $F(2, 16) = 1.801$, $p = 0.197$, partial $\eta^2 = 0.184$. For the older men, thermoregulatory behaviour was statistically significantly different between time points, $F(2, 18) = 4.120$, $p = 0.034$, partial $\eta^2 = 0.314$.

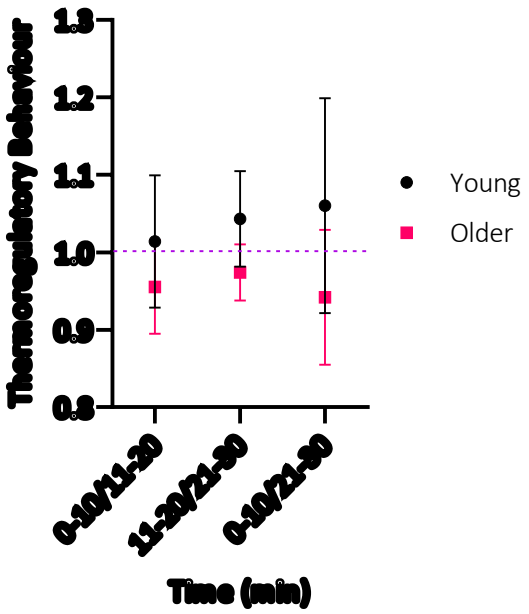

Figure 14-8. *Thermoregulatory behaviour of young and older men throughout the CCC trial by comparing the middle to the first 10 min of cycling, the middle to the final 10 min of cycling, and the final to the first 10 min of cycling. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output as time increased.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour during the CHC trial (**Figure 14-9**). Data is presented as mean ± standard deviation, unless otherwise stated. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. All data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variance, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's equality of variance matrices ($p = 0.03$). Mauchly's test of sphericity indicated that the assumption of sphericity was met for the two-way interaction, $\chi^2(2) = 3.486$, $p = 0.175$.

There was a statistically significant interaction effect of age and time on thermoregulatory behaviour during the CHC trial, $F(2, 34) = 9.085$, p <0.001, partial $\eta^2 = 0.348$. Therefore, simple main effects were run. The young men implemented statistically significantly better thermoregulatory behaviour (1.09

(95% CI) 1.03 to 1.15) in the middle 10 min of cycling compare to the first 10 min of cycling than the older men (0.94 (95% CI) 0.87 to 1.00), $F(1, 17) = 13.478$, $p = 0.002$, partial $\eta^2 = 0.442$. There was not statistically significant difference in thermoregulatory behaviour between the age groups when comparing the final 10 min of cycling to the middle 10 min of cycling, $F(1, 17) = 1.503$, $p = 0.237$, partial $\eta^2 = 0.081$. The young men implemented statistically significantly better thermoregulatory behaviour (1.12 (95% CI) 1.03 to 1.20) in the final 10 min of cycling compared to the first 10 min of cycling than the older men (0.94 (95% CI) 0.86 to 1.02), $F(1, 17) = 9.743$, $p = 0.006$, partial $\eta^2 = 0.364$.

For the young men, thermoregulatory behaviour was statistically significantly different between time points, $F(2, 16) = 4.029$, $p = 0.038$, partial $\eta^2 = 0.335$ (first to middle 10 min, 1.09 (95% CI) 1.01 to 1.17. Middle to final 10 min, 1.03 (95% CI) 0.98 to 1.08. First to final 10 min, 1.12 (95% CI) 1.01 to 1.22). For the older men, thermoregulatory behaviour was statistically significantly different between time points, $F(2, 18) = 6.412$, $p = 0.008$, partial $\eta^2 = 0.416$ (first to middle 10 min, 0.94 (95% CI) 0.90 to 0.99. Middle to final 10 min, 0.99 (95% CI) 0.96 to 1.03. First to final 10 min, 0.94 (95% CI) 0.86 to 1.02).

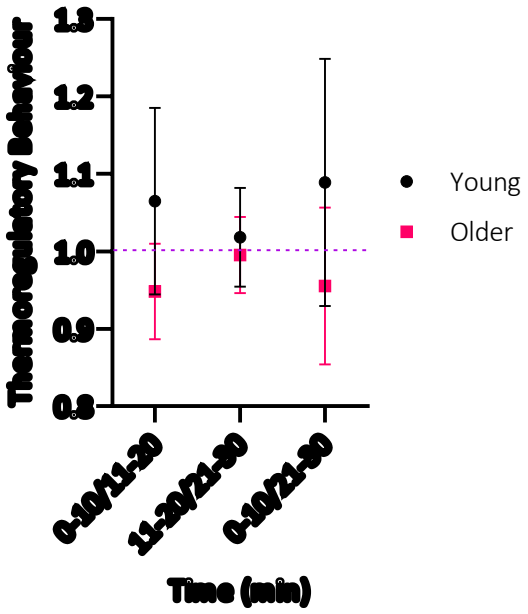

Figure 14-9. *Thermoregulatory behaviour of young and older men throughout the CHC trial by comparing the middle to the first 10 min of cycling, the middle to the final 10 min of cycling, and the final to the first 10 min of cycling. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output as time increased.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour during the W+CCC trial (**Figure 14-10**). Data is presented as mean ± standard deviation, unless otherwise stated. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. All data was normally distributed, as assessed by normal Q-Q plots. There

was homogeneity of variance, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's equality of variance matrices (p = 0.01). Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi^2(2) = 8.373$, $p = 0.015$. Therefore, a Greenhouse-Geisser correction was applied.

There was no statistically significant interaction effect of age and time on thermoregulatory behaviour during the W+CCC trial, $F(1.421, 24.157) = 0.634$, $p = 0.487$, partial $\eta^2 = 0.036$. Therefore, main effects were examined. The main effect of time did not show a statistically significant difference in thermoregulatory behaviour at different time points of the W+CCC trial, $F(1.421, 24.157) = 0.012$, $p = 0.988$, partial $\eta^2 = 0.001$. The main effect of group showed no statistically significant effect of age on thermoregulatory behaviour in the W+CCC trial, $F(1, 17) = 0.601$, $p = 0.449$, partial $\eta^2 = 0.034$.

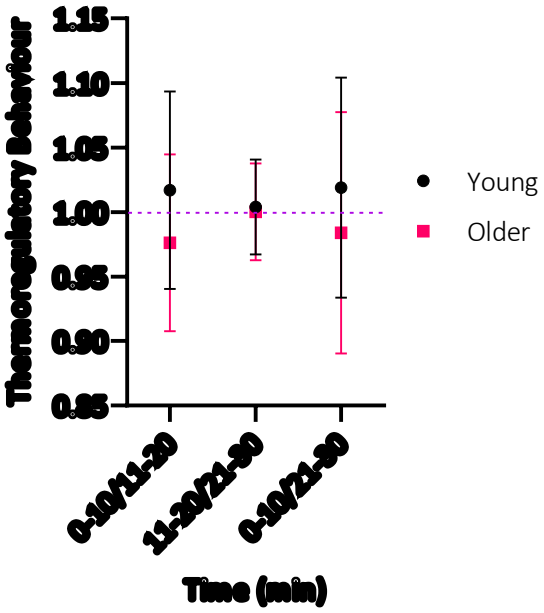

Figure 14-10. *Thermoregulatory behaviour of young and older men throughout the W+CCC trial by comparing the middle to the first 10 min of cycling, the middle to the final 10 min of cycling, and the final to the first 10 min of cycling. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output as time increased.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour during the W+CHC trial (**Figure 14-11**). Data is presented as mean ± standard deviation, unless otherwise stated. There was one outlier, which had a studentised residual value of -3.08. This outlier was kept in the analysis as it did not materially affect the results. Thermoregulatory behaviour data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variances, as assessed by Levene's test of homogeneity of variance (p >0.05) for the middle 10 min compared to the final 10 min, and the first 10 min compared to the final 10 min, but not for the first 10 min

compared with the middle 10 min (p = 0.014). There was not homogeneity of covariances, as assessed by Box's test of equality of covariance matrices (p <0.001). The analysis was continued as the two-way mixed ANOVA is robust to this violation. Mauchly's test of sphericity indicated that the assumption of sphericity was met, $\chi^2(2) = 1.265$, $p = 0.531$.

There was a statistically significant interaction between the time period and age group on thermoregulatory behaviour, $F(2, 34) = 1.225$, $p = 0.021$, partial $\eta^2 = 0.203$. Therefore, simple main effects were run.The young men implemented statistically significantly better thermoregulatory behaviour (1.09 (95% CI) 1.02 to 1.15) in the middle 10 min of cycling compare to the first 10 min of cycling than the older men (0.97 (95% CI) 0.91 to 1.03), $F(1, 17) = 7.685$, $p = 0.013$, partial $\eta^2 = 0.311$. There was not statistically significant difference in thermoregulatory behaviour between the age groups when comparing the final 10 min of cycling to the middle 10 min of cycling, $F(1, 17) = 0.958$, $p = 0.341$, partial $\eta^2 = 0.053$. The young men implemented statistically significantly better thermoregulatory behaviour (1.11 (95% CI) 1.01 to 1.21) in the final 10 min of cycling compared to the first 10 min of cycling than the older men (0.96 (95% CI) 0.86 to 1.05), $F(1, 17) = 5.662$, $p = 0.029$, partial $\eta^2 = 0.250$.

For the young men, thermoregulatory behaviour was not statistically significantly different between time points, $F(2, 16) = 3.496$, $p = 0.055$, partial $\eta^2 = 0.304$. For the older men, thermoregulatory behaviour was not statistically significantly different between time points, $F(2, 18) = 0.792$, $p = 0.468$, partial $\eta^2 = 0.081$.

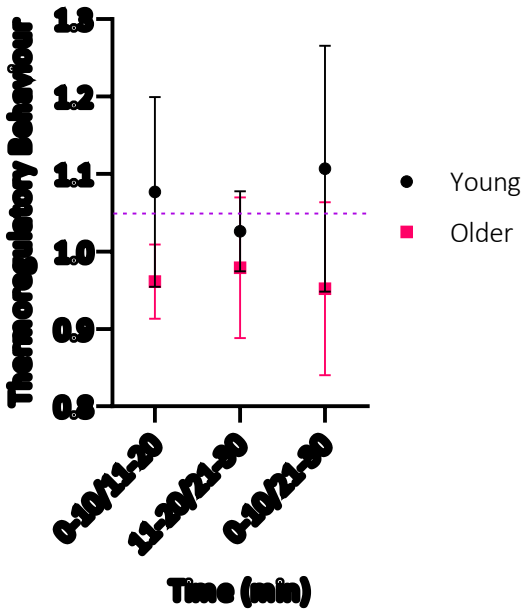

Figure 14-11. *Thermoregulatory behaviour of young and older men throughout the CHC trial by comparing the middle to the first 10 min of cycling, the middle to the final 10 min of cycling, and the final to the first 10 min of cycling. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output as time increased.*

15.4.3 Thermal Comfort

A three-way mixed ANOVA was run to understand the effects of trial condition, time, and age on thermal comfort (**Figure 14-12**). Thermal comfort scores were normally distributed, as assessed by Shapiro-Wilk's test (p >0.05). There were 17 outliers, as assessed by inspection of a boxplot. The outliers were kept in the analysis as they did not materially affect the results, as assessed by comparison of the results with and without the outliers. There was homogeneity of variances, as assessed by Levene's test for equality of variances (p >0.05). Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the three-way interaction effect ($\chi^2(20) = 33.674$, $p = 0.031$). Therefore, the Greenhouse-Geisser correction was applied.

There was a statistically significant three-way interaction effect of time point, age, and trial condition on thermal comfort, $F(3.244, 55.142) = 3.649$, $p = 0.016$, partial $\eta^2 = 0.177$, $\varepsilon = 0.541$. Therefore, simple two-way interactions were examined. Statistical significance of a simple two-way interaction was accepted at a Bonferroni-adjusted alpha level of 0.025. Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction effect in the young men ($\chi^2(20) = 37.329$, $p = 0.020$). Therefore, the Greenhouse-Geisser correction was applied.

The simple two-way interaction between trial condition and time for the young men was statistically significantly different, $F(1.946, 15.568) = 9.827$, $p = 0.002$, partial $\eta^2 = 0.551$, $\varepsilon = 0.324$. Mauchly's test

of sphericity indicated that the assumption of sphericity was met for the two-way interaction effect in the older men ($\chi^2(20) = 22.204$, $p = 0.383$). The simple two-way interaction between trial condition and time for the young men was statistically significantly different, $F(6, 54) = 7.120$, $p < 0.001$, partial $\eta^2 = 0.442$. Simple simple main effects were then run.

Statistical significance of a simple simple main effect was accepted at a Bonferroni-adjusted alpha level of 0.025. The simple simple main effect of trial for young men during the first 10 min of cycling on thermal comfort was statistically significant, $F(3, 24) = 5.418$, $p = 0.005$, partial $\eta^2 = 0.404$. During the CCC trial thermal comfort was 0.4 (95% CI, 0.1 to 0.8), this was statistically significantly lower than the W+CHC trial at 1.1 (95% CI, 0.7 to 1.6), $p = 0.042$.

The simple simple main effect of trial for older men during the first 10 min of cycling on thermal comfort was statistically significant, $F(3, 27) = 6.176$, $p = 0.002$, partial $\eta^2 = 0.407$.

The simple simple main effect of trial for young men during the middle 10 min of cycling on thermal comfort was statistically significant, $F(1.932, 15.459) = 18.700$, $p < 0.001$, partial $\eta^2 = 0.700$. Thermal comfort in the CCC trial (1.0 (95% CI) 0.7 to 1.3) was statistically significantly lower than the CHC (2.2 (95% CI) 1.7 to 2.6, $p = 0.010$) and the W+CHC trial (2.2 (95% CI) 1.7 to 2.6, $p = 0.007$). The CHC trial (2.2 (95% CI) 1.7 to 2.6) was also statistically significantly higher than the W+CCC trial (1.1 (95% CI) 0.7 to 1.5, $p = 0.022$). The W+CCC trial (1.1 (95% CI) 0.7 to 1.5) was statistically significantly lower than the W+CHC trial (2.2 (95% CI) 1.7 to 2.6, $p < 0.001$).

The simple simple main effect of trial for older men during the middle 10 min of cycling on thermal comfort was statistically significant, $F(3, 27) = 3.190$, $p = 0.040$, partial $\eta^2 = 0.262$.

The simple simple main effect of trial for young men during the final 10 min of cycling on thermal comfort was not statistically significant, $F(3, 24) = 1.508$. $p = 0.238$, partial $\eta^2 = 0.159$.

The simple simple main effect of trial for older men during the final 10 min of cycling on thermal comfort was statistically significant, $F(3, 27) = 4.457$. $p = 0.011$, partial $\eta^2 = 0.331$.

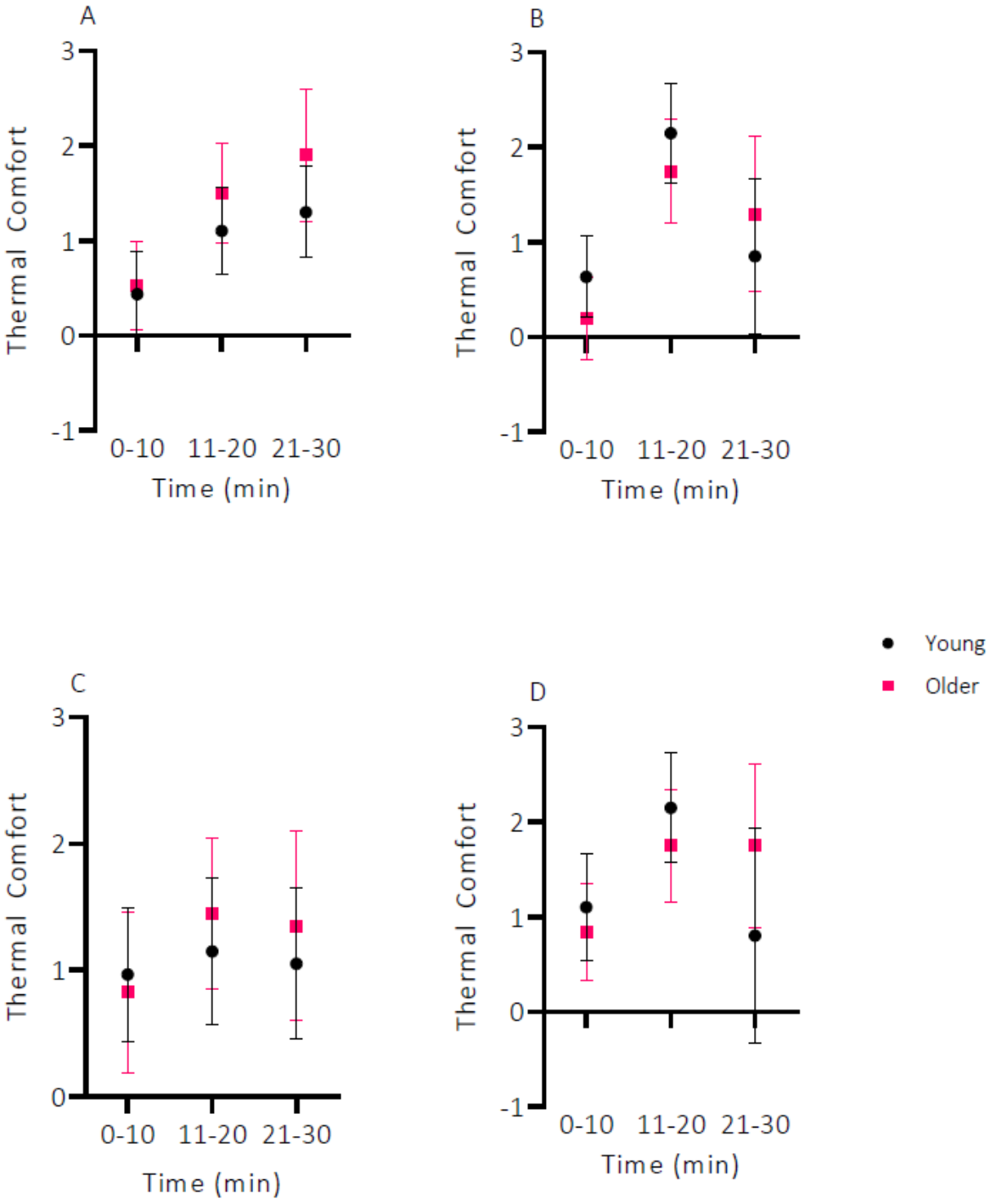

***Figure 14-12**. Thermal comfort of young and older men at the first, middle, and final 10 min of each trial. A = CCC trial, B = CHC trial, C = W+CCC trial, D = W+CHC trial. Data presented as mean ± SD.*

15.5 Discussion

The young men responded to pre-warming (CCC trial compared to W+CCC trial) by reducing their power output resulting in a high thermoregulatory behaviour score (1.10 ± 0.12) during the initial 10 min of cycling. whereas the older men maintained the same power output despite the 1.0°C warmer rectal temperature, resulting in a thermoregulatory behaviour score of 1.00 ± 0.13. The age groups displayed similar responses when comparing the CCC to the CHC trials and the CCC compared with the W+CHC. When examining thermoregulatory behaviour within each trial the age groups differ in the CCC, CHC, and W+CHC trials. The older men increased power output as time increased during the CCC trial while the young men decreased. The young men responded to the ambient temperature increase during the CHC and the W+CHC trials by reducing power output (thermoregulatory behaviour scores: CHC 1.09 ± 0.12, W+CHC 1.08 ± 0.16) while the older men increased power output (thermoregulatory behaviour scores: CHC 0.94 ± 0.09, W+CHC 0.97 ± 0.07).

This was the first study to examine thermoregulatory behaviours during exercise in changing ambient conditions with older men. We utilised radiant heat lamps during cycling exercise to identify thermoregulatory responses to increases in skin temperature. This step was undertaken to manipulate skin temperature while minimising their impact on rectal temperature. Radiant heat lamps have been used previously with young (~30 years) men rowing for 5,000 m at a fixed RPE of 15 in changing ambient conditions (radiant heat lamps turned on at 1,000 – 2,000 m and 3,000 – 4,000 m distance; Lander et al., 2014). Skin temperature increased by 0.51°C during the first warming phase, and power output dropped by 6% ($p = 0.03$). During the second warming phase skin temperature increased by 0.15°C, power output reduced by 4% ($p = 0.10$. Lander et al., 2014). In our study the heat lamps caused a ~5°C and a ~3°C increase in skin temperature for both age groups in the CHC and W+CHC trials, respectively, to ~35°C. This caused significantly different thermoregulatory behavioural responses in the young and older men in the CHC trial. The young men reduced their power output when the radiant heat lamps were activated. This was a similar response as was reported by Lander et al. (2014). The older men however, increased their power output when the radiant heat lamps were active and skin temperature increased. Their thermal comfort also increased so they felt warmer but did not reduce their metabolic heat load. This is an important finding as it appears that older men ignore or unable to respond to drivers of thermoregulatory behaviour.

This has public health implications, especially given that adults aged 65 years and over are vulnerable during heat wave events. The inability of older men to respond to changing skin temperature and thermal perception may prevent them from mitigating their heat risk via compensatory thermoregulatory behaviours. This effect may add to the strain on health services during heat wave

events, and in the aftermath, as those who suffer severe heat illness/stroke may lose physical independence as a result. Advice given during heat wave events should include guidance that during exercise in the heat older men are unable to voluntarily respond to increases in skin temperature, so should be mindful to reduce activity levels even if they do not perceive themselves to be at risk.

Previous work using a fixed RPE cycling protocol indicated young men (<35 years) drop to 70% of their starting power output in ~22 min (Flood et al., 2017), and ~33 min (Parton et al., 2021), albeit using an RPE of 16 (between 'hard' and 'very hard'). We expected that power output would decline during all the trials, but at differing rates. The younger men behaved as anticipated, their power output declined from the first to the last 10 min in the CCC trial (thermoregulatory behaviour 1.07 ± 0.15). Whereas the older men maintained their power output throughout the trials with little effect of time, pre-warming, or changing ambient conditions. In the CCC trial the older men increased power output from the first to the final 10 min (thermoregulatory behaviour 0.94 ± 0.10). It appears that older men do not voluntarily adjust their power output in response to an elevated rectal temperature, to rapid increases in ambient and skin temperatures, or to increases in thermal comfort and thermal perception. This inability to reduce metabolic heat load during heat exposure will put them at an elevated risk of heat illness during heat wave events that are becoming more severe and more common.

The pre-warming trials in the present study caused a ~2°C warmer mean skin temperature, for both age groups, at the onset of exercise than the non-pre-warmed trials (~32°C v ~30°C), alongside the 1°C increase in rectal temperature. Skin temperature at the onset of exercise is known to influence self-selected exercise intensity in young (30 ± 9 years) men during a 60 min cycling time trial (Schlader et al., 2011b). In the present study young men had a lower cycling power output at the start of the cycling exercise when skin and rectal temperature was elevated prior to exercise (thermoregulatory behaviour value: CCC/W+CCC = 1.10 ± 0.12; CHC/W+CHC = 1.12 ± 0.27). The older men did not respond to an increased skin and rectal temperature at the onset of the W+CCC and W+CHC trials as their cycling power output was not different from the CCC and CHC trials, respectively (thermoregulatory behaviour value: CCC/W+CCC = 1.00 ± 0.13; CHC/W+CHC = 1.04 ± 0.11). These findings differ to those of Schlader et al. (2011b) who reported that at the onset of exercise, skin temperatures of ~35°C resulted in a 2.4% reduction in exercise intensity compared with skin temperatures of ~29°C (Schlader et al., 2011b). A mean skin temperature of 36.3°C resulted in resting young (~22 years) adults implementing cooling behaviours to a greater extent than when skin temperature is 34.5°C (Vargas et al., 2018b). In contrast to Vargas et al. (2018b), thermoregulatory behaviours were not implemented by the older group in the present study, despite a greater increase in mean skin temperature (2.0°C v 1.8°C). This difference in findings may be due to the lower absolute mean skin temperatures induced

in our study. Vargas et al. (2018b) and Schlader et al. (2011b) were examining skin temperatures ~35°C, whereas we induced mean skin temperatures of ~32°C prior to the cycling exercise. However, the radiant heat lamps used in the CHC and W+CHC trials did cause mean skin temperature to rise to ~35°C during exercise for both the young and older men, yet the older men still did not implement thermoregulatory behaviours. While the young men implemented thermoregulatory behaviours in the W+CCC trial with a ~2°C increase in mean skin temperature, rectal temperature was also increased by 1°C. It is unclear if a ~2°C increase in mean skin temperature is sufficient to result in thermoregulatory behaviour implementation without the additional stimulus of an increased in core temperature.

The young and older men had similar rectal and mean skin temperature responses throughout the study. During the cycling exercise in the CCC and CHC trials rectal temperature increased for both groups. However, in the pre-warmed trials (W+CCC and W+CHC), rectal temperature remained stable once the participants exited the hot water (pre-exercise ~38.1°C, post-trial ~38.2°C), cycling cause no further increase in rectal temperature. Our data suggest that when men, regardless of age, exercise at a fixed RPE of 13 for 30 min, they do not produce enough metabolic heat to increase core temperature above ~38°C. An elevated rectal temperature at the onset of exercise did not cause thermoregulatory behaviours to be implemented in the older men. The young men had a mixed response, they reduced power output in the first 10 min of the W+CCC trial compared to the CCC trial, but did not adjust power output in the W+CHC trial compared to the CHC trial. Our data suggests that rectal temperature at the onset of exercise does not drive thermoregulatory behaviours in older men and has a limited influence in young men. During fixed intensity cycling (50% $\dot{V}O_2$peak), young men (24 ± 3 years) reported greater RPE scores when their oesophageal temperature was 37.5°C at the start of exercise than when it was 36.9°C (Tsuji et al., 2012). Thus, it was anticipated that an elevated rectal temperature prior to exercise would result in thermoregulatory behaviours being implemented via reduced power outputs.

It has consistently been shown that older men are less sensitive to changes in ambient temperature. When at rest, men aged 60 years and over feel more thermally comfortable in changing ambient temperatures than young men, and when given control of a dual position thermostat, allow greater fluctuations of ambient temperature (Collins et al., 1981; Natsume et al., 1992; Taylor et al., 1995). Older men (66-75 years) have also been shown to feel more comfortable than young men (22-27 years) after exiting a hot bath (42°C) and sitting at rest in 20°C conditions (Ono et al., 2017). Older men have a greater range for thermo-neutrality, and they need to experience a greater thermal input before they initiate thermoregulatory behaviours. This appears to be the case during exercise as well. The older men felt warmer than the young men in the CCC trial yet increased their cycling power

output throughout this trial. Both age groups experienced similar thermal perception at the onset of the trials and during the radiant heat lamp exposures. Yet during the CHC trial the older men increased cycling power output, while the young men reduced cycling power output when the radiant heat lamps were active. This study indicates that older men are less sensitive than young men to changes in thermal perceptions while exercising in stable and changing ambient conditions.

Aerobic fitness and body composition are mediators of heat illness risk, a greater BMI and proportion of body fat, and lower aerobic fitness increases risk of heat illness. (Westwood et al., 2020). In the present study, the young and older men had similar anthropometry, with stature, body mass and body composition being similar between the young and older men. However, the young men tended to have a greater body mass, BMI, and sum of skinfolds. This was due to two of the younger participants having a body mass of 110 kg and 121 kg, whereas the heaviest older man was 93 kg. As a higher body mass and BMI is associated with a greater risk of heat illness, the older men may have been at a slight advantage in the present study. Despite the anthropometric outliers, both age groups had similar fitness levels as the total power output in the CCC trial did not differ between the age groups (young = 510 W, older = 3171 W. $p = 0.477$). Age was the only baseline measurement that differed between the age groups. Thus, it seems that the impaired thermoregulatory behavioural response is an age-related decline rather than associated with body composition or fitness level.

Experimental Chapter Three was designed to try and increase the amount of heat stress experienced by the participants as Experimental Chapter One: Relationship between age and ability to implement thermoregulatory behaviours in thermoneutral and hot ambient conditions and Experimental Chapter Two: Physiological and perceptual factors influencing behavioural thermoregulation in young and older men in thermoneutral and hot ambient conditions had only caused rectal temperature to reach ~38°C at the end of the exercise in the hot trials. The implementation of pre-warming with hot water immersion caused rectal temperature to reach ~38°C prior to the exercise starting. This outcome was achieved despite differing transfer times from the bath to the cycle ergometer for individual participants. They were allowed to exit the bath and change clothes at their own pace to reduce the risk of fainting and injury after being immersed in hot (~42°C) water. However, rectal temperature did not increase further in either the W+CCC or W+CHC trials for either the young or older men. It appears that when men exercise at an RPE of 13, rectal temperature plateaus at ~38°C.

A limitation of the present study was that participants could not be blinded to the heat lamps being turned on or off. The rapid increase in ambient temperature allowed them to perceive this increase relatively soon after they were turned on. However, participants were not provided with any feedback on their power output, speed, cadence, or heart rate, and were unaware of the purpose of the study.

15.6 Conclusion

We demonstrated that when skin and rectal temperature are elevated prior (CCC compared with W+CCC) to exercise young men implement thermoregulatory behaviours by reducing power output, whereas older men do not implement these behavioural responses. However, this was a mixed response as the W+CHC trial did not result in thermoregulatory behaviours being implemented at the onset of exercise (CCC compared with W+CHC). During changing ambient conditions, young men are able to implement thermoregulatory behaviours in response to ambient temperature increases, whereas older men are not. These outcomes are evident despite younger and older men in this study having similar anthropometry (height, body mass, body composition) and physical fitness characteristics. There was also no difference in skin or rectal temperature response between the age groups, indicating that older men are less sensitive to changes in skin temperature. Public health advice should emphasise that being aged 60 years or above alone is a risk factor for heat-related illness, even in individuals with no pre-existing health issues.

was designed to increase the level of heat strain experienced by young and older men with passive pre-warming using hot water immersion. Ambient radiant heat lamps were used to manipulate skin temperature during exercise. This approach allowed the influence of skin and core temperature on thermoregulatory behaviour during 30 min of fixed RPE cycling to be separated. Skin and rectal temperature responses were similar between the age groups. Core and skin temperature at the onset of exercise dictate the implementation of thermoregulatory behaviours in young men. Older men do not implement thermoregulatory behaviours in response to an elevated rectal and skin temperature at the onset of exercise. Young men implement thermoregulatory behaviour in response to increasing ambient temperature by reducing power output. However, older men are reluctant to reduce their exercise intensity when ambient temperature rises.

Table 15-1. *Hypotheses for each experimental chapter, an indication of if they are rejected or accepted, and the reasoning behind the acceptance/rejection.*

Hypotheses	Accept/ Reject	Reason
Experimental chapter one: Relationship between age and ability to implement thermoregulatory behaviours in thermoneutral and hot ambient conditions		
There is a negative relationship between increasing age and ability to implement thermoregulatory behaviours	×	p = 0.087 for age and thermoregulatory behaviour for all participants. *p = 0.674 for age and thermoregulatory behaviour for women.* p = 0.005 for age and thermoregulatory behaviour for men
The relationship between age and ability to implement thermoregulatory behaviours is the same for men and women	×	Men had a nearly linear relationship, women had a similar as men relationship until ~65 years when two anomalous participants skewed the data to indicate a plateau in thermoregulatory behaviour decline
Experimental chapter two: Physiological and perceptual factors influencing behavioural thermoregulation in young and older men in thermoneutral and hot ambient conditions		
Older men will not implement thermoregulatory behaviours as effectively as young men	✓	Young men had a thermoregulatory behaviour score of 1.06, older men of 0.95, significantly different p = 0.009. (>1 = implemented thermoregulatory behaviour. ≤1 = did not implement thermoregulatory behaviour)
Older men will perceive the hot trial as more comfortable than the young men	×	No difference between age groups in thermal comfort
Older men will experience a greater level of heat strain	×	No difference between age groups in skin or rectal temperature during hot trial
Experimental Chapter three: Thermoregulatory behaviour during fixed RPE cycling in changing ambient conditions in young and older men		
Young men will implement thermoregulatory behaviours more effectively than older men	✓	Pre-warming resulted in thermoregulatory behaviours for the young men, but not the older men. Young men responded to heat lamps, whereas older men did not
Older men will feel more comfortable than young men when heat lamps are turned on	×	No difference in thermal comfort between age groups at any point.
Older men will experience a greater level of heat strain	×	No difference in skin or rectal temperatures between young and older men.

15.7 Implications

Our work has demonstrated that older adults, particularly men, are unable to voluntarily initiate thermoregulatory behaviours when experiencing heat strain, thus increasing their heat illness risk and vulnerability during heatwave events. This presents considerable public health implications as heat wave events are becoming more common (IPCC, 2013), and older adults are disproportionately

affected by heat wave events. Canada experienced record temperatures of 49.6°C in June 2021, causing at least 130 deaths, mostly in vulnerable populations including adults aged ≥65 years (BBC, 2021). It has become undeniable that heatwave events are set to continue and to increase in severity. This climate and weather scenario poses a major public health issue, especially if older adults are unable to voluntarily implement cooling behaviours efficiently.

Heat-related deaths in the over 65s continue to occur despite it being well understood that older adults have a diminished autonomic thermoregulatory capacity. Our findings suggest that men aged ≥60 years are less able to voluntarily reduce their heat stress as they do not implement thermoregulatory behaviours in situations that young men do. This highlights that men aged ≥60 years are at an even greater risk of heat illness than previously thought as they cannot compensate for their impaired autonomic thermoregulation via thermoregulatory behaviours. Unlike autonomic thermoregulation, behavioural thermoregulation (e.g., removing layers of clothing, seeking shade or air conditioning, taking cooling showers, reducing metabolic heat load) offers a near limitless ability to reduce heat strain. Young adults have repeatedly been shown to implement these behaviours, both at rest and during exercise (Schlader et al., 2009, 2013; Schlader et al., 2011c). The experimental outcomes of this thesis concur with these findings as the young men reduced walking speed in the 35°C trials in Experimental Chapter One: Relationship between age and ability to implement thermoregulatory behaviours in thermoneutral and hot ambient conditions and Experimental Chapter Two: Physiological and perceptual factors influencing behavioural thermoregulation in young and older men in thermoneutral and hot ambient conditionsand adjusted their cycling power output in response to increased skin and rectal temperature in Experimental Chapter Three: Thermoregulatory behaviour during fixed RPE cycling in changing ambient conditions in young and older men

15.8 Introduction

Experimental Chapter One: Relationship between age and ability to implement thermoregulatory behaviours in thermoneutral and hot ambient conditions identified that thermoregulatory behaviour is impaired in older adults, particularly men, and established that men aged ≥60 years do not reduce their walking speed when exposed to 35°C ambient conditions. The mechanisms responsible for this self-regulated behaviour remain unclear. Experimental Chapter Two: Physiological and perceptual factors influencing behavioural thermoregulation in young and older men in thermoneutral and hot ambient conditions could not identify a physiological or perceptual causative factor for the impairment in behavioural thermoregulation but did identify that older men are less able to implement thermoregulatory behaviours than young men. Understanding the contribution of skin and

core temperature to thermoregulatory behaviours could elucidate possible mechanisms behind the decline in behavioural thermoregulation in older men.

Peak rectal temperature during the hot (35°C ambient) trials in Experimental Chapters One and Two reached ~38.0°C for all participants. While this was a ~1°C increase from the onset of the trials, core temperature of 38 – 40°C are not abnormal during exercise, particularly in hot conditions (Gleeson, 1998). Core temperature of young (~30 years) men performing 5,000 m of rowing at a fixed RPE of 15 ('hard') did not differ between ~35°C and 18°C ambient conditions with final core temperatures being ~38.5°C in both conditions (Lander et al., 2014). Thus, young men experience similar core temperature increases while exercising at a fixed RPE in temperate and hot conditions. Older men might require a higher rectal temperature before they will voluntarily initiate thermoregulatory behaviours. Hot water immersion at 40°C has been shown to induce a greater thermal challenge than exercising for the same amount of time at 50% of $\dot{V}O_2$max in 40°C (Greenfield et al., 2021). Experimental Chapter Three will employ hot water immersion to pre-warm participant's rectal temperature by 1°C before they undertake fixed RPE cycling for 30 min. This approach will permit examination of thermoregulatory behaviour when participants are experiencing heat strain prior to the onset of exercise, rather than using the exercise to induce heat strain.

Skin temperature mediates thermoregulatory behaviour (Schlader et al., 2009, 2013). However, the evidence from Experimental Chapter One: Relationship between age and ability to implement thermoregulatory behaviours in thermoneutral and hot ambient conditions Experimental Chapter Two: Physiological and perceptual factors influencing behavioural thermoregulation in young and older men in thermoneutral and hot ambient conditions indicates that skin temperature either has little influence on thermoregulatory behaviour in older men exercising in the heat, or older men have a reduced sensitivity to increases in skin temperature. The young men experienced skin temperature during the hot trials of 35.24°C and 35.22°C in experimental chapters one and two respectively, while the older men experienced skin temperatures of 35.40°C and 35.68°C. This was an increase from the control trials of 3.45°C and 3.36°C in experimental chapters one and two respectively, for the young men, and 4.48°C and 4.60°C for the older men. Young (~30 years) men reduce rowing power output when skin temperature increases by 0.5°C under changing ambient conditions (Lander et al., 2014). During a 5,000 m rowing trial where RPE was fixed at 15 ('hard'), radiant heat lamps were used to increase the ambient temperature from 18 to 35°C from the 1,000 to 2,000 m and 3,000 to 4,000 m distances. Each 1,000 m took ~4 min to complete, so the heat lamps were activated for ~4 min during each warming period. Skin temperature rose ~0.5°C and power output declined by 6% during the first heat exposure (Lander et al., 2014). Young men were able to initiate thermoregulatory behaviours within 4 min of a change in ambient conditions. There is a dearth of research investigating

thermoregulatory behaviours in exercising older adults during heat stress. Using heat lamps to alter the ambient temperature provides a way of rapidly inducing skin temperature changes in exercising individuals. It also allows for the examination of the effect of skin temperature, independently of core temperature, on thermoregulatory behaviours as skin temperature will change more rapidly than core temperature. We will employ this radiant heat lamp method in the present study to try and elucidate the influence of skin temperature on thermoregulatory behaviour in men aged ≥60 years.

There are contrasting models of how self-selected exercise intensity, or pacing, is controlled. The psychobiological model proposes that pacing is controlled consciously (Marcora, 2010), whereas the central governor theory suggests that motor units are controlled subconsciously (St Clair Gibson et al., 2006). These two theories are opposed, and it has been postulated that pacing is a multidimensional process requiring both conscious and subconscious, or sub-awareness input (Edwards & Polman, 2013; Micklewright et al., 2017). The treadmill protocol utilised in Experimental Chapters One and Two prevented any subconscious alterations in walking speed as participants had to consciously decide to push a button to alter the treadmill speed. Allowing participants to unknowingly adjust their exercise intensity may lead to the implementation of thermoregulatory behaviours sooner, and to a greater extent, than when they must consciously push a button on a treadmill. Cycling ergometer exercise will allow participants to unknowingly and knowingly adjust their exercise intensity by adjusting their pedal cadence. Therefore, the current study, will employ cycling exercise at a fixed RPE.

Pacing strategy during exercise in the heat is thought be anticipatory, implemented as soon as core temperature starts to increase, rather than once a threshold (core) temperature is reached (Tucker & Noakes, 2009). Within 4 min of starting a cycling time trial in 35°C at a fixed RPE of 16 (between 'hard' and 'very hard'), young (~23 years) men implemented pacing strategies that maintained a similar level of heat storage as a 15°C trial (Tucker et al., 2006). Thus, young men can voluntarily implement thermoregulatory behaviours while cycling at a fixed RPE by reducing their power output. Research investigating thermoregulatory behaviour in exercising adults has neglected to include older adults (≥60 years).

The use of pre-warming and changing environmental conditions with older participants will facilitate investigation of the independent and combined effect of rectal and skin temperature on behavioural thermoregulation during fixed RPE exercise. This novel approach should identify whether skin or rectal temperature has a greater influence on thermoregulatory behaviour in older men, or if an increase of both skin and core temperature are required for initiation of thermoregulatory behaviours. We hypothesise that: pre-warming will result in a reduction in total work done for both young and older men; power output will be reduced when the radiant heat lamps are turned on for both young and

older men; older men will feel more thermally comfortable than young men; and the older men will experience a greater level of heat strain.

15.9 Methods

15.9.1 Participants

Nineteen healthy males, nine young (19-31 years) and ten older (66-87 years) volunteered to participate in the study. Participants were non-smokers, free from any known respiratory, cardiovascular, or metabolic disease, and not taking any medications known to affect thermoregulation. Participant anthropometric data is presented in **Error! Reference source not found.Error! Reference source not found.**. The experimental protocol was approved by the Plymouth Marjon University Ethics Panel (EP103) in accordance with the Declaration of Helsinki. Volunteers provided written informed consent after explanation of the purpose, benefits, risks, and experimental procedures of the study before participating in the study.

15.9.2 Experimental overview

This study used a group comparison of performance, temperature, and perceptual responses to cycling in 22°C or changing conditions (22-33°C), with and without passive heating before cycling exercise in younger and older men. A self-paced, fixed RPE protocol was used (similar to Lander et al., 2014), with participants completing a familiarisation session to get comfortable with the RPE scale and the cycle ergometer. Anthropometric measurements were also taken in the familiarisation session.

Participants completed four experimental trials consisting of 30 min of cycling at a fixed rating of perceived exertion (RPE) of 13, equivalent to 'somewhat hard' (Borg, 1982). Participants were blinded to their speed, power output and heart rate data, and received no feedback on how far they had cycled during the trial. The control trial consisted of 30 min of cycling in 21.5 ± 0.5°C ambient conditions. Another trial was completed in changing ambient conditions, where radiant heat lamps were activated during 10 – 20 min of 30 min fixed RPE cycling. A third trial was conducted with pre-warming, by immersion in 40 – 42°C water, of rectal temperature by 1°C prior to the 30 min of cycling in 21.5 ± 0.5°C with no heat lamp activation. The final trial condition included both pre-warming and heat lamp activation during 10 – 20 min of the 30 min fixed RPE cycling. Four radiant heat lamps were positioned around the participant, 1 m away from the centre of the cycle ergometer. Ten min into the trials using the lamps they were turned on for 10 min, resulting in an increase in ambient temperature to 33.3 ± 1.4°C.

Trials were conducted in a randomised order, participants arrived at the same time of day, and with at least 48 h between trials. Participants were also asked to abstain from alcohol, caffeine, non-

steroidal anti-inflammatory drugs, and strenuous exercise in the 24 h before trials, and consume 500 mL of water the evening before and 2 h before arriving at the laboratory. Clothing consisted of a t-shirt, shorts, and walking/running shoes. Water was available for *ad libitum* consumption throughout the trials.

Mean power output was calculated in 10 min periods (0-10 min, 11-20 min, and 21-30 min). A marker of thermoregulatory behaviour within a trial was then calculated as:

***Equation 14-1**. Thermoregulatory behaviour established as a change in power output as a function of time.*

$$Thermoregulatory\ Behaviour = \frac{Mean\ Power\ Output_{Trial\ A\ Time\ Period\ x}}{Mean\ Power\ Output_{Trial\ A\ Time\ Period\ y}}$$

Between trials thermoregulatory behaviour (CCC v CHC, CCC v W+CCC, CHC v W+CHC, W+CCC v W+CHC) was also compared at each time period and calculated as:

***Equation 14-2.** Thermoregulatory behaviour established as a change in power output as a function of trial condition.*

$$Thermoregulatory\ Behaviour = \frac{Mean\ Power\ Output_{Trial\ A\ Time\ Period\ x}}{Mean\ Power\ Output_{Trial\ B\ Time\ Period\ x}}$$

This index gave a ratio of power output within a trial as duration increased, and between trials with different ambient and pre-warming conditions. A result >0 indicated that the participant reduced power output between conditions being compared, <0 they increased power output between conditions being compared, =0 there was no difference between conditions being compared.

15.9.3 Experimental procedures

In the preliminary visit, anthropometric measures (stature, body mass, skinfolds) were taken as described in the General Methods. Participants were then familiarised with the RPE scale. Participants were asked to adjust the cycle ergometer (Wattbike Pro/Trainer, Wattbike, Nottingham, UK) so that they were comfortable on the bike. The seat and handlebar positions were recorded for use in all subsequent trials. Next, participants self-selected a resistance on the cycle ergometer that elicited an RPE of 13, 'somewhat hard' while blinded to their power output and speed. After 3 min participants confirmed that the resistance settings felt 'somewhat hard', and their power output was recorded (but not revealed to the participant) before they sat at rest for 5 min. Participants repeated this procedure until they were able to replicate a power output that elicited an RPE of 13. The resistance settings were recorded for use in subsequent trials.

During hot water immersion both rectal and water temperature were monitored continuously. Heart rate was recorded every 5 min. Water was available to drink *ad libitum.* Once rectal temperature had increased by 1°C, participants were removed from the hot water bath and given privacy while they changed into dry clothing, the rectal thermistor remained in place. Participants then entered the environmental chamber where skin thermistors were attached as described in the General Methods (Chapter 11.9.2 Skin Temperature). An ambient temperature thermistor was hung from the ceiling ~30 cm above the participant's head. Temperature data was collected as described in the General Methods (Chapter 11.9 Temperature). Participants then mounted the cycle ergometer and began the 30 min cycling trial. Heart rate and power output were recorded at ~1 sec intervals by the Wattbike. These data were then averaged into 10 min blocks. RPE was assessed every 5 min.

15.9.4 Statistical analysis

For *a priori* power analysis, conventional α (0.05) and β (0.20) parameters and a large effect size (1.3), based on data from Experimental Chapter Two were employed. The estimations indicated that 9 subjects per group was appropriate to detect meaningful differences. Power analysis was performed using G*Power (G*Power, Düsseldorf, Germany).

Data is presented as mean ± standard deviation, unless otherwise stated. The level of significance for all analyses was set at $p \leq 0.05$. Effect size was defined as small $d = 0.20$, partial $\eta^2 = 0.01$; medium $d = 0.50$, partial $\eta^2 = 0.06$; and large $d = 0.80$, partial $\eta^2 = 0.14$ (Cohen, 1988; Cohen 1969). All statistical analyses were conducted using Statistics Package for the Social Sciences (SPSS v28, IBM Corp, Armonk, NY).

15.10 Results

15.10.1 Participant characteristics and temperature responses

Participant characteristics are presented in **Table 14-1**.

Table 14-1*. Participant anthropometric data. Data presented as mean ± SD. Bold denotes significantly different from the young group.*

	n	Age (years)	Stature (cm)	Body Mass (kg)	Body Mass Index	Σskinfolds (mm)
Young	9	24 ± 4	181 ± 5	86.9 ± 19.6	26.6 ± 5.7	47.1 ± 29.2
Older	10	**70 ± 7**	176 ± 5	73.8 ± 9.0	23.8 ± 2.6	33.5 ± 15.3
p value	-	<0.001	0.067	0.077	0.188	0.234

A two-way repeated measures ANOVA was run to determine the effect of trial conditions and time point on ambient temperature (**Figure 14-2**). Analysis of studentised residual showed that there was

normality, as assessed by the Shapiro-Wilk test of normality (p >0.05), and no outliers as assessed by no studentised residuals greater than ± 3 standard deviations. Mauchly's test of sphericity indicated that the assumption of sphericity had been violated for the two-way interaction, $\chi^2(2) = 89.831$, p <0.001, therefore a Greenhouse-Geisser correction was applied.

There was a statistically significant two-way interaction between treatment and time, $F(3.145, 56.609) = 246.068$, p <0.001, $\varepsilon = 0.524$. Therefore, simple main effects were run.

Ambient temperature was not statistically significantly different between trials at during the first 10 min of cycling, $F(1.974, 35.535) = 0.726$, $p = 0.489$, $\varepsilon = 0.658$.

There was a statistically significant effect of trial condition on ambient temperature during the middle 10 min of cycling, $F(2.020, 36.368) = 935.734$, p <0.001, $\varepsilon = 0.673$. Ambient temperature was warmer in the CHC (33.24 (95% CI) 32.53 to 33.95°C) and W+CHC (33.39 (95% CI) 32.76 to 34.02°C) trials than the CCC (21.48 (95% CI) 21.28 to 21.70°C) and W+CCC (21.46 (95% CI) 21.20 to 21.72°C) trials.

There was a statistically significant effect of trial condition on ambient temperature during the final 10 min of cycling, $F(1.995, 35.911) = 13.631$, p <0.001, $\varepsilon = 0.665$. Ambient temperature was warmer in the CHC (23.53 (95% CI) 22.60 to 24.45°C) and W+CHC (23.92 (95% CI) 22.83 to 25.02°C) trials than the CCC (21.58 (95% CI) 21.36 to 21.80°C) and W+CCC (21.54 (95% CI) 21.28 to 21.80°C) trials.

There was a statistically significant effect of time on ambient temperature during the control trial (CCC), $F(2, 36) = 7.218$, $p = 0.002$. Ambient temperature was warmer during the first 10 min (21.80 (95% CI) 21.55 to 22.05°C) than the middle (21.48 (95% CI) 21.28 to 21.69°C, $p = 0.001$) and final 10 min of cycling (21.58 (95% CI) 21.36 to 21.80°C, $p = 0.050$).

There was a statistically significant effect of time on ambient temperature during the no pre-warming, heat lamp trial (CHC), $F(2, 36) = 336.332$, p <0.001. Ambient temperature was warmer during the middle 10 min (33.24 (95% CI) 32.53 to 33.95°C) than the first (21.97 (95% CI) 21.68 to 22.27°C, p <0.001) and final 10 min of cycling (23.53 (95% CI) 22.60 to 24.45°C, p <0.001). The final 10 min of cycling were also warmer than the first 10 min of cycling, $p = 0.006$.

There was a statistically significant effect of time on ambient temperature during pre-warming, no heat lamps trial (W+CCC), $F(2, 36) = 40.230$, p <0.001. Ambient temperature was warmer during the first 10 min (22.03 (95% CI) 21.79 to 22.28°C) than the middle (21.46 (95% CI) 21.20 to 21.71°C, p <0.001) and final 10 min of cycling (21.54 (95% CI) 21.28 to 21.80°C, p <0.001).

There was a statistically significant effect of time on ambient temperature during the pre-warming, and heat lamp trial (W+CHC), $F(2, 36) = 302.170$, p <0.001. Ambient temperature was warmer during

the middle 10 min (33.39 (95% CI) 32.76 to 34.02°C) than the first (21.74 (95% CI) 21.18 to 22.30°C, p <0.001) and final 10 min of cycling (23.92 (95% CI) 22.83 to 25.02°C, p <0.001). The final 10 min of cycling were also warmer than the first 10 min of cycling, p = 0.002.

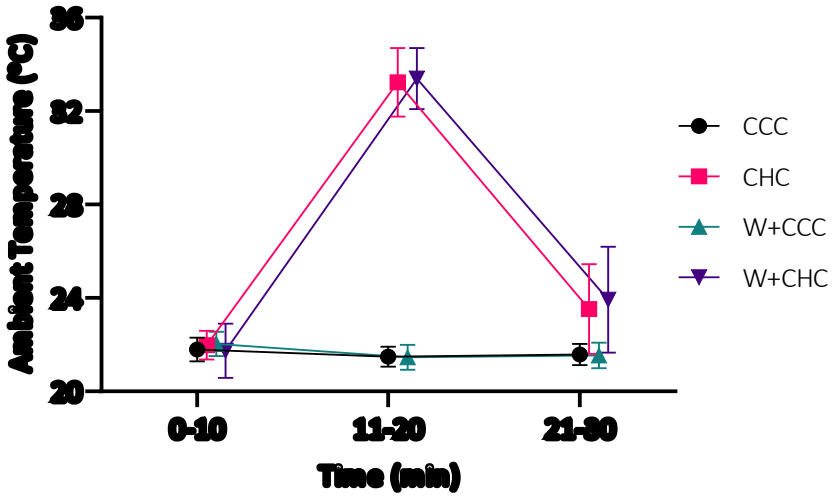

***Figure 14-2**. Ambient temperature for the first, middle, and final 10 min of cycling in each trial. Data presented as mean ± SD.*

A three-way mixed ANOVA was run to examine the effects of age, trial condition, and time point on mean skin temperature (**Figure 14-3**). There were three outliers, as assessed by inspection of a boxplot for values greater than 3 box-lengths from the edge of the box. These were kept in the analysis as they did not materially affect the result. Mean skin temperature data was normally distributed (p >0.05), except for the final 10 min of cycling in the control trial for the older men (p = 0.023), as assessed by Shapiro-Wilk's test. There was homogeneity of variances, as assessed by Levene's test for equality of variances (p >0.05). Mauchly's test of sphericity indicated that the assumption of sphericity was violated, $\chi^2(20) = 35.580$, p = 0.020. Therefore, the Greenhouse-Geisser correction was applied.

There was no statistically significant three-way interaction between age, trial condition, and time point on mean skin temperature, $F(3.792, 56.886) = 1.258$, p = 0.297, partial η^2 = 0.077, ε = 0.632. There was no statistically significant two-way interaction between trial condition and age on mean skin temperature, $F(1.980, 29.699) = 1.159$, p = 0.327, partial η^2 = 0.072. There was no statistically significant two-way interaction between time point and age on mean skin temperature, $F(1.331, 19.970) = 1.205$, p = 0.302, partial η^2 = 0.074. There was a statistically significant two-way interaction between trial condition and time point on mean skin temperature, $F(3.792, 563886) = 130.462$, p <0.001, partial η^2 = 0.897.

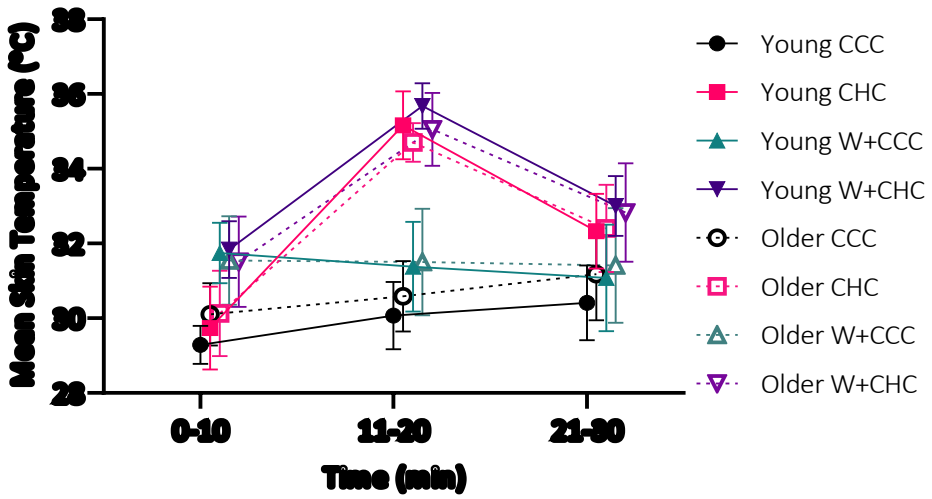

***Figure 14-3**. Mean skin temperature during the first, middle, and final 10 min of cycling for each trial in the young and older men. Data presented as mean ± SD.*

A three-way mixed ANOVA was run to examine the effects of age, trial condition, and time point on rectal temperature (**Figure 14-4**). There were no outliers, as assessed by inspection of a boxplot for values greater than 3 box-lengths from the edge of the box. Rectal temperature data was normally distributed, as assessed by Shapiro-Wilk's test (p >0.05). There was homogeneity of variances, as assessed by Levene's test for equality of variances (p >0.05). Mauchly's test of sphericity indicated that the assumption of sphericity had been violated for the two-way interaction, $\chi^2(2) = 86.545$, p <0.001, therefore a Greenhouse-Geisser correction was applied.

There was no statistically significant three-way interaction between age, trial condition, and time point on rectal temperature, $F(3.258, 58.650) = 2.692$, $p = 0.050$, partial $\eta^2 = 0.130$, $\varepsilon = 0.543$. There was a statistically significant two-way interaction between trial condition and age on rectal temperature, $F(2.831, 50.960) = 3.383$, $p = 0.027$, partial $\eta^2 = 0.158$. There was no statistically significant two-way interaction between time and age on rectal temperature, $F(1.012, 18.214) = 1.793$, $p = 0.197$, partial $\eta^2 = 0.091$. There was a statistically significant two-way interaction between trial condition and time point on rectal temperature, $F(3.258, 58.650) = 56.403$, p <0.001, partial $\eta^2 = 0.758$.

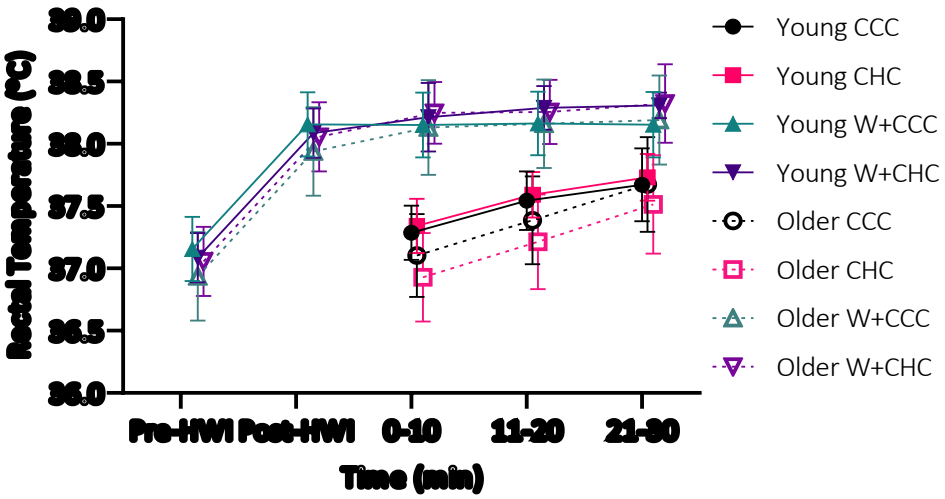

Figure 14-4*. Rectal temperature during the trials for the young and older men. HWI = hot water immersion. Data presented as mean ± SD.*

15.10.2Thermoregulatory behaviour

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour when comparing the CCC and CHC trials (**Figure 14-5**). There was one outlier, which had a studentised residual value of 3.05. This outlier was kept in the analysis as it did not materially affect the results. Thermoregulatory behaviour data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variances, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's test of equality of covariance matrices (p = 0.078). Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi^2(2)$ = 8.115, p = 0.017. Therefore, the Greenhouse-Geisser correction was applied.

There was no statistically significant two-way interaction between time point and age on thermoregulatory behaviour, $F(1.431, 24.324)$ = 1.496, p = 0.241, partial η^2 = 0.081, ε = 0.715. The main effect of time point showed a statistically significant difference in thermoregulatory behaviour at different time points, $F(1.431, 24.324)$ = 5.098, p = 0.023, partial η^2 = 0.231. The final 10 min of cycling (0.97 (95% CI) 0.90 to 1.03) had a higher thermoregulatory behaviour score than the first 10 min of cycling (1.05 (95% CI) 0.99 to 1.10) p = 0.031). Thermoregulatory behaviour score for middle 10 min of cycling (0.99 (95% CI) 0.91 to 1.07) did not statistically significantly differ from either the first (p = 0.349) or the final (p = 0.289) 10 min of cycling. The main effect of age showed no statistically significant difference between the young (1.01 (95% CI) 0.93 to 1.10) and older (0.99 (95% CI) 0.91 to 1.07) men in thermoregulatory behaviour score, $F(1, 17)$ = 0.107, p = 0.748, partial η^2 = 0.006.

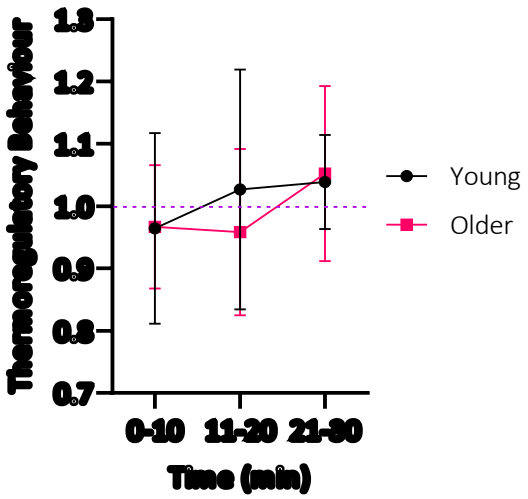

Figure 14-5. *Thermoregulatory behaviour of young and older men during the first, middle, and final 10 min of cycling when comparing the CHC trial to the CCC trial. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output in the CHC trial compared to the CCC trial.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour when comparing the CCC and W+CCC trials (**Figure 14-6**). Data is presented as mean ± standard deviation, unless otherwise stated. There was one outlier, which had a studentised residual value of 3.05. This outlier was kept in the analysis as it did not materially affect the results. Thermoregulatory behaviour data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variances, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's test of equality of covariance matrices (p = 0.092). Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi^2(2) = 11.272$, $p = 0.004$. Therefore, the Greenhouse-Geisser correction was applied.

There was a statistically significant interaction between age and time on thermoregulatory behaviour, $F(1.328, 22.582) = 4.404$, $p = 0.038$, partial $\eta^2 = 0.206$, $\varepsilon = 0.664$. Therefore, simple main effects were examined. There was no statistically significant difference in thermoregulatory behaviour between the age groups during the first 10 min of cycling ($p = 0.121$, partial $\eta^2 = 0.136$), the middle 10 min of cycling ($p = 0.271$, partial $\eta^2 = 0.071$), or the final 10 min of cycling (p = 0.836, partial $\eta^2 = 0.003$). There was no statistically significant effect of time on thermoregulatory behaviour for the young men (p = 0.282, partial $\eta^2 = 0.146$). There was a statistically significant effect of time on thermoregulatory behaviour for the older men, $F(2, 18) = 6.033$, $p = 0.010$, partial $\eta^2 = 0.401$.

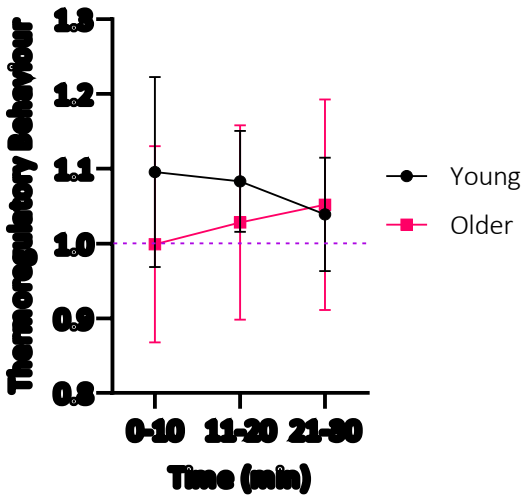

***Figure 14-6**. Thermoregulatory behaviour of young and older men during the first, middle, and final 10 min of cycling when comparing the W+CCC trial to the CCC trial. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output in the W+CCC trial compared to the CCC trial.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour when comparing the CCC and W+CHC trials (**Figure 14-7**). Data is presented as mean ± standard deviation, unless otherwise stated. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. All data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variance, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's equality of variance matrices (p = 0.03). Mauchly's test of sphericity indicated that the assumption of sphericity was met for the two-way interaction, $\chi^2(2) = 4.944$, $p = 0.084$.

There was no statistically significant two-way interaction between time point and age on thermoregulatory behaviour, $F(2, 34) = 0.949$, $p = 0.397$, partial $\eta^2 = 0.053$. Therefore, main effects were examined. The main effect of time point showed a statistically significant difference in thermoregulatory behaviour at the different time points, $F(2, 34) = 3.519$, $p = 0.041$, partial $\eta^2 = 0.171$. The main effect of age group showed that there was no statistically significant difference in thermoregulatory behaviour between the young and older men, $F(1, 17) = 0.713$, $p = 0.410$, partial $\eta^2 = 0.040$. The first 10 min of cycling (1.02 (95% CI) 0.956 to 1.084) did not significantly differ from the middle (1.06 (95% CI) 1.00 to 1.12, $p = 0.108$) or final 10 min of cycling (1.06 (95% CI) 1.00 to 1.12, $p = 0.207$). The middle 10 min of cycling was not significantly different to the final 10 min of cycling ($p = 0.999$).

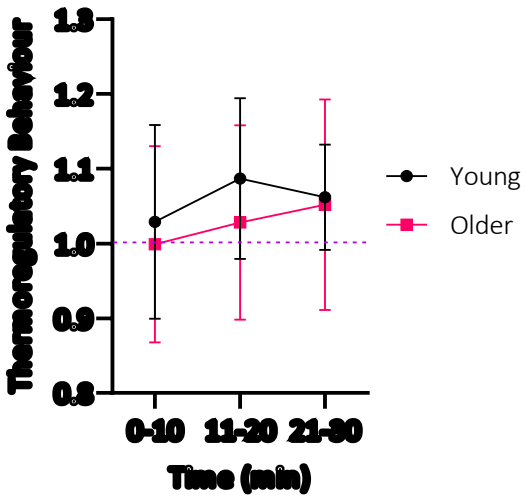

Figure 14-7. *Thermoregulatory behaviour of young and older men during the first, middle, and final 10 min of cycling when comparing the W+CHC trial to the CCC trial. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output in the W+CHC trial compared to the CCC trial.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour during the CCC trial (**Figure 14-8**). Data is presented as mean ± standard deviation, unless otherwise stated. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. All data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variance, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's equality of variance matrices (p = 0.02). Mauchly's test of sphericity indicated that the assumption of sphericity was met for the two-way interaction, $\chi^2(2) = 3.499$, $p = 0.174$.

There was a statistically significant interaction effect of age and time on thermoregulatory behaviour during the CCC trial, $F(2, 34) = 3.813$, $p = 0.032$, partial $\eta^2 = 0.183$. Therefore, simple main effects were run. There was no statistically significant difference in thermoregulatory behaviour between the age groups when comparing the middle 10 min of cycling to the first 10 min of cycling, $F(1, 17) = 4.114$, $p = 0.058$, partial $\eta^2 = 0.195$. The young men implemented statistically significantly better thermoregulatory behaviour (1.05 (95% CI) 1.01 to 1.08) in the final 10 min of cycling when compared to the middle 10 min of cycling than the older men (0.98 (95% CI) 0.94 to 1.01), $F(1, 17) = 7.835$, $p = 0.012$, partial $\eta^2 = 0.315$. The young men implemented statistically significantly better thermoregulatory behaviour (1.07 (95% CI) 0.99 to 1.15) in the final 10 min of cycling compared to the first 10 min of cycling than the older men (0.94 (95% CI) 0.86 to 1.01), $F(1, 17) = 6.658$, $p = 0.019$, partial $\eta^2 = 0.281$.

For the young men, thermoregulatory behaviour was not statistically significantly different between time points, $F(2, 16) = 1.801$, $p = 0.197$, partial $\eta^2 = 0.184$. For the older men, thermoregulatory behaviour was statistically significantly different between time points, $F(2, 18) = 4.120$, $p = 0.034$, partial $\eta^2 = 0.314$.

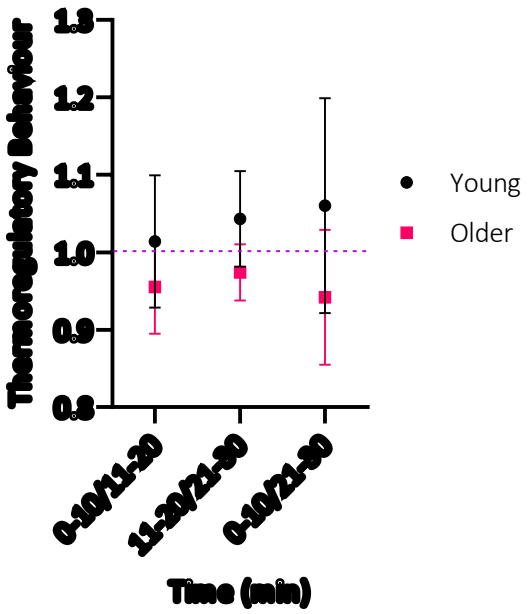

Figure 14-8. *Thermoregulatory behaviour of young and older men throughout the CCC trial by comparing the middle to the first 10 min of cycling, the middle to the final 10 min of cycling, and the final to the first 10 min of cycling. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output as time increased.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour during the CHC trial (**Figure 14-9**). Data is presented as mean ± standard deviation, unless otherwise stated. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. All data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variance, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's equality of variance matrices ($p = 0.03$). Mauchly's test of sphericity indicated that the assumption of sphericity was met for the two-way interaction, $\chi^2(2) = 3.486$, $p = 0.175$.

There was a statistically significant interaction effect of age and time on thermoregulatory behaviour during the CHC trial, $F(2, 34) = 9.085$, p <0.001, partial $\eta^2 = 0.348$. Therefore, simple main effects were run. The young men implemented statistically significantly better thermoregulatory behaviour (1.09 (95% CI) 1.03 to 1.15) in the middle 10 min of cycling compare to the first 10 min of cycling than the older men (0.94 (95% CI) 0.87 to 1.00), $F(1, 17) = 13.478$, $p = 0.002$, partial $\eta^2 = 0.442$. There was not statistically significant difference in thermoregulatory behaviour between the age groups when

comparing the final 10 min of cycling to the middle 10 min of cycling, $F(1, 17) = 1.503$, $p = 0.237$, partial $\eta^2 = 0.081$. The young men implemented statistically significantly better thermoregulatory behaviour (1.12 (95% CI) 1.03 to 1.20) in the final 10 min of cycling compared to the first 10 min of cycling than the older men (0.94 (95% CI) 0.86 to 1.02), $F(1, 17) = 9.743$, $p = 0.006$, partial $\eta^2 = 0.364$.

For the young men, thermoregulatory behaviour was statistically significantly different between time points, $F(2, 16) = 4.029$, $p = 0.038$, partial $\eta^2 = 0.335$ (first to middle 10 min, 1.09 (95% CI) 1.01 to 1.17. Middle to final 10 min, 1.03 (95% CI) 0.98 to 1.08. First to final 10 min, 1.12 (95% CI) 1.01 to 1.22). For the older men, thermoregulatory behaviour was statistically significantly different between time points, $F(2, 18) = 6.412$, $p = 0.008$, partial $\eta^2 = 0.416$ (first to middle 10 min, 0.94 (95% CI) 0.90 to 0.99. Middle to final 10 min, 0.99 (95% CI) 0.96 to 1.03. First to final 10 min, 0.94 (95% CI) 0.86 to 1.02).

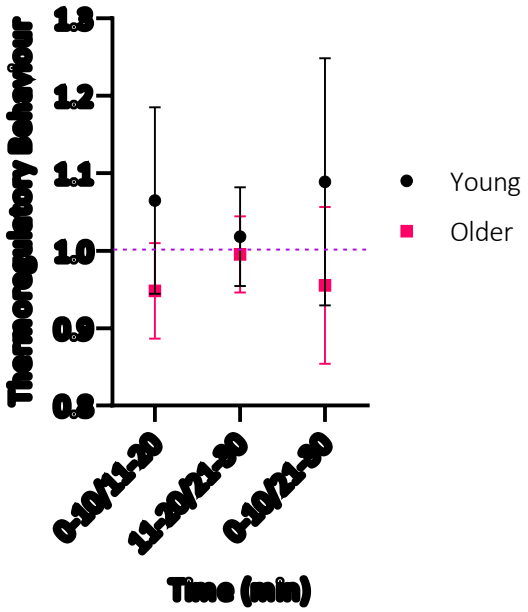

Figure 14-9. *Thermoregulatory behaviour of young and older men throughout the CHC trial by comparing the middle to the first 10 min of cycling, the middle to the final 10 min of cycling, and the final to the first 10 min of cycling. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output as time increased.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour during the W+CCC trial (**Figure 14-10**). Data is presented as mean ± standard deviation, unless otherwise stated. There were no outliers, as assessed by examination of studentised residuals for values greater than ± 3. All data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variance, as assessed by Levene's test of homogeneity of variance (p >0.05). There was homogeneity of covariances, as assessed by Box's equality of variance matrices (p = 0.01).

Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi^2(2) = 8.373$, $p = 0.015$. Therefore, a Greenhouse-Geisser correction was applied.

There was no statistically significant interaction effect of age and time on thermoregulatory behaviour during the W+CCC trial, $F(1.421, 24.157) = 0.634$, $p = 0.487$, partial $\eta^2 = 0.036$. Therefore, main effects were examined. The main effect of time did not show a statistically significant difference in thermoregulatory behaviour at different time points of the W+CCC trial, $F(1.421, 24.157) = 0.012$, $p = 0.988$, partial $\eta^2 = 0.001$. The main effect of group showed no statistically significant effect of age on thermoregulatory behaviour in the W+CCC trial, $F(1, 17) = 0.601$, $p = 0.449$, partial $\eta^2 = 0.034$.

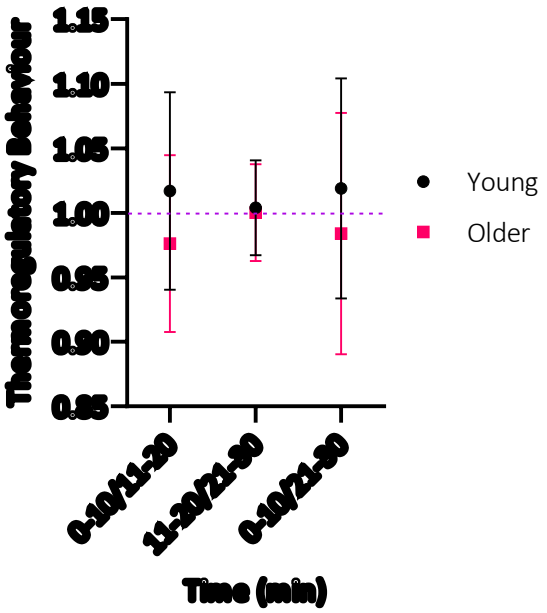

Figure 14-10. *Thermoregulatory behaviour of young and older men throughout the W+CCC trial by comparing the middle to the first 10 min of cycling, the middle to the final 10 min of cycling, and the final to the first 10 min of cycling. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output as time increased.*

A two-way mixed ANOVA was run to determine the effects of age and time point on thermoregulatory behaviour during the W+CHC trial (**Figure 14-11**). Data is presented as mean ± standard deviation, unless otherwise stated. There was one outlier, which had a studentised residual value of -3.08. This outlier was kept in the analysis as it did not materially affect the results. Thermoregulatory behaviour data was normally distributed, as assessed by normal Q-Q plots. There was homogeneity of variances, as assessed by Levene's test of homogeneity of variance (p >0.05) for the middle 10 min compared to the final 10 min, and the first 10 min compared to the final 10 min, but not for the first 10 min compared with the middle 10 min ($p = 0.014$). There was not homogeneity of covariances, as assessed by Box's test of equality of covariance matrices (p <0.001). The analysis was continued as the two-way

mixed ANOVA is robust to this violation. Mauchly's test of sphericity indicated that the assumption of sphericity was met, $\chi^2(2) = 1.265$, $p = 0.531$.

There was a statistically significant interaction between the time period and age group on thermoregulatory behaviour, $F(2, 34) = 1.225$, $p = 0.021$, partial $\eta^2 = 0.203$. Therefore, simple main effects were run.The young men implemented statistically significantly better thermoregulatory behaviour (1.09 (95% CI) 1.02 to 1.15) in the middle 10 min of cycling compare to the first 10 min of cycling than the older men (0.97 (95% CI) 0.91 to 1.03), $F(1, 17) = 7.685$, $p = 0.013$, partial $\eta^2 = 0.311$. There was not statistically significant difference in thermoregulatory behaviour between the age groups when comparing the final 10 min of cycling to the middle 10 min of cycling, $F(1, 17) = 0.958$, $p = 0.341$, partial $\eta^2 = 0.053$. The young men implemented statistically significantly better thermoregulatory behaviour (1.11 (95% CI) 1.01 to 1.21) in the final 10 min of cycling compared to the first 10 min of cycling than the older men (0.96 (95% CI) 0.86 to 1.05), $F(1, 17) = 5.662$, $p = 0.029$, partial $\eta^2 = 0.250$.

For the young men, thermoregulatory behaviour was not statistically significantly different between time points, $F(2, 16) = 3.496$, $p = 0.055$, partial $\eta^2 = 0.304$. For the older men, thermoregulatory behaviour was not statistically significantly different between time points, $F(2, 18) = 0.792$, $p = 0.468$, partial $\eta^2 = 0.081$.

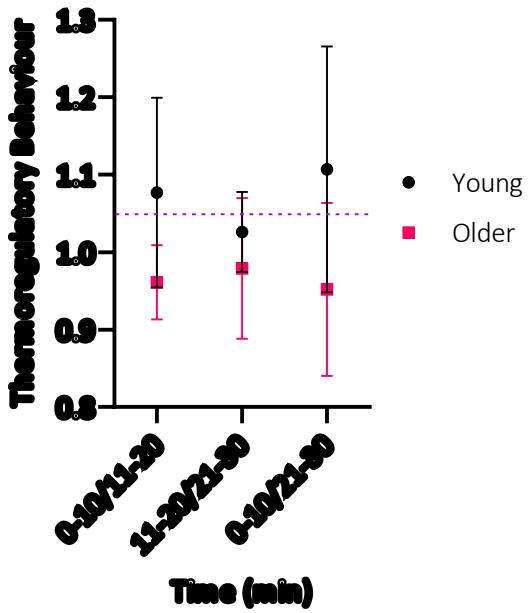

Figure 14-11. *Thermoregulatory behaviour of young and older men throughout the CHC trial by comparing the middle to the first 10 min of cycling, the middle to the final 10 min of cycling, and the final to the first 10 min of cycling. Data presented as mean ± SD. Points above the purple line indicate thermoregulatory behaviour was implemented by reducing cycling power output as time increased.*

15.10.3Thermal Comfort

A three-way mixed ANOVA was run to understand the effects of trial condition, time, and age on thermal comfort (**Figure 14-12**). Thermal comfort scores were normally distributed, as assessed by Shapiro-Wilk's test (p >0.05). There were 17 outliers, as assessed by inspection of a boxplot. The outliers were kept in the analysis as they did not materially affect the results, as assessed by comparison of the results with and without the outliers. There was homogeneity of variances, as assessed by Levene's test for equality of variances (p >0.05). Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the three-way interaction effect ($\chi^2(20) = 33.674$, $p = 0.031$). Therefore, the Greenhouse-Geisser correction was applied.

There was a statistically significant three-way interaction effect of time point, age, and trial condition on thermal comfort, $F(3.244, 55.142) = 3.649$, $p = 0.016$, partial $\eta^2 = 0.177$, $\varepsilon = 0.541$. Therefore, simple two-way interactions were examined. Statistical significance of a simple two-way interaction was accepted at a Bonferroni-adjusted alpha level of 0.025. Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction effect in the young men ($\chi^2(20) = 37.329$, $p = 0.020$). Therefore, the Greenhouse-Geisser correction was applied.

The simple two-way interaction between trial condition and time for the young men was statistically significantly different, $F(1.946, 15.568) = 9.827$, $p = 0.002$, partial $\eta^2 = 0.551$, $\varepsilon = 0.324$. Mauchly's test of sphericity indicated that the assumption of sphericity was met for the two-way interaction effect in the older men ($\chi^2(20) = 22.204$, $p = 0.383$). The simple two-way interaction between trial condition and time for the young men was statistically significantly different, $F(6, 54) = 7.120$, p <0.001, partial $\eta^2 = 0.442$. Simple simple main effects were then run.

Statistical significance of a simple simple main effect was accepted at a Bonferroni-adjusted alpha level of 0.025. The simple simple main effect of trial for young men during the first 10 min of cycling on thermal comfort was statistically significant, $F(3, 24) = 5.418$, $p = 0.005$, partial $\eta^2 = 0.404$. During the CCC trial thermal comfort was 0.4 (95% CI, 0.1 to 0.8), this was statistically significantly lower than the W+CHC trial at 1.1 (95% CI, 0.7 to 1.6), $p = 0.042$.

The simple simple main effect of trial for older men during the first 10 min of cycling on thermal comfort was statistically significant, $F(3, 27) = 6.176$, $p = 0.002$, partial $\eta^2 = 0.407$.

The simple simple main effect of trial for young men during the middle 10 min of cycling on thermal comfort was statistically significant, $F(1.932, 15.459) = 18.700$, p <0.001, partial $\eta^2 = 0.700$. Thermal comfort in the CCC trial (1.0 (95% CI) 0.7 to 1.3) was statistically significantly lower than the CHC (2.2 (95% CI) 1.7 to 2.6, $p = 0.010$) and the W+CHC trial (2.2 (95% CI) 1.7 to 2.6, $p = 0.007$). The CHC trial

(2.2 (95% CI) 1.7 to 2.6) was also statistically significantly higher than the W+CCC trial (1.1 (95% CI) 0.7 to 1.5, $p = 0.022$). The W+CCC trial (1.1 (95% CI) 0.7 to 1.5) was statistically significantly lower than the W+CHC trial (2.2 (95% CI) 1.7 to 2.6, $p < 0.001$).

The simple simple main effect of trial for older men during the middle 10 min of cycling on thermal comfort was statistically significant, $F(3, 27) = 3.190$, $p = 0.040$, partial $\eta^2 = 0.262$.

The simple simple main effect of trial for young men during the final 10 min of cycling on thermal comfort was not statistically significant, $F(3, 24) = 1.508$. $p = 0.238$, partial $\eta^2 = 0.159$.

The simple simple main effect of trial for older men during the final 10 min of cycling on thermal comfort was statistically significant, $F(3, 27) = 4.457$. $p = 0.011$, partial $\eta^2 = 0.331$.

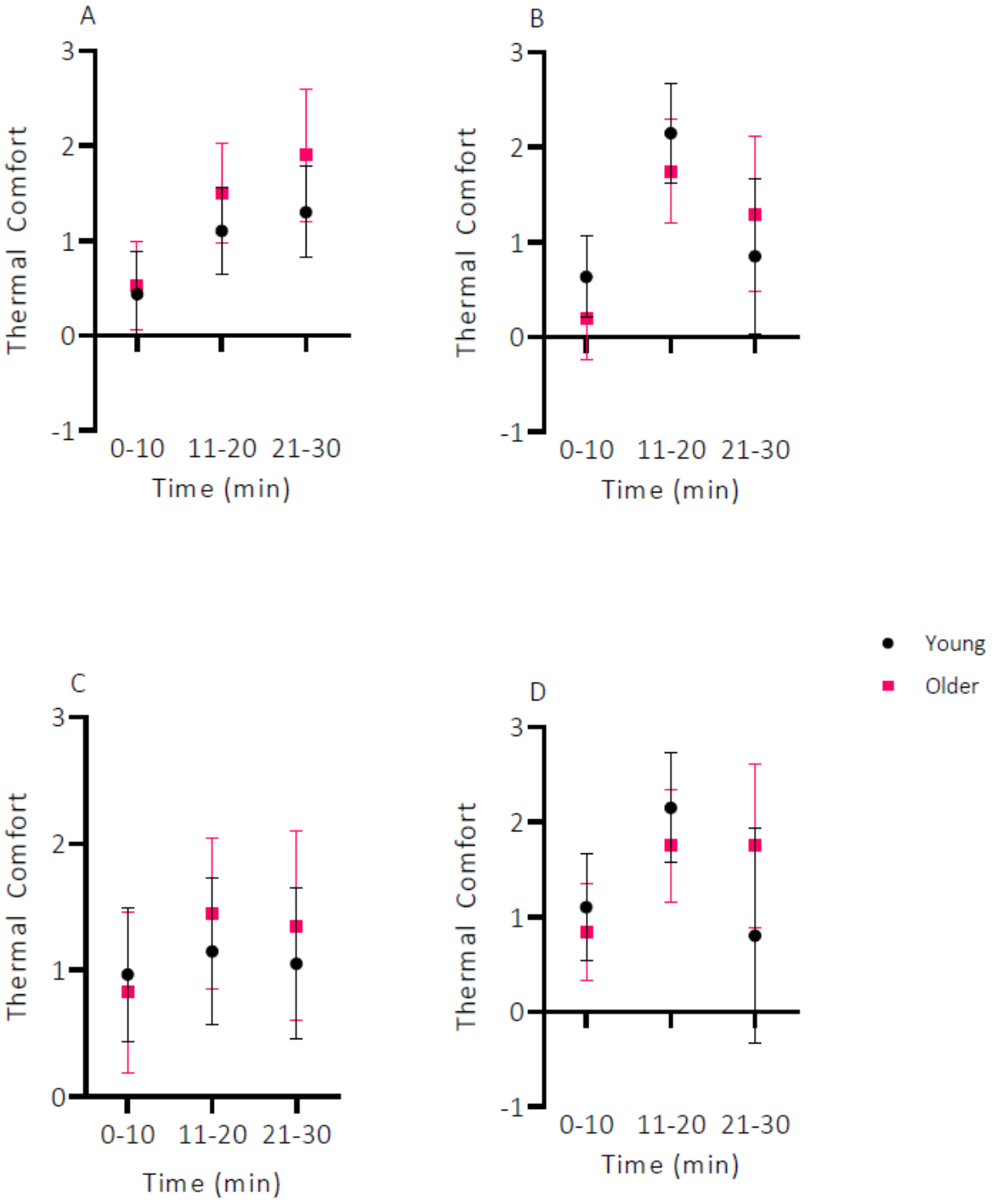

***Figure 14-12**. Thermal comfort of young and older men at the first, middle, and final 10 min of each trial. A = CCC trial, B = CHC trial, C = W+CCC trial, D = W+CHC trial. Data presented as mean ± SD.*

15.11 Discussion

The young men responded to pre-warming (CCC trial compared to W+CCC trial) by reducing their power output resulting in a high thermoregulatory behaviour score (1.10 ± 0.12) during the initial 10 min of cycling. whereas the older men maintained the same power output despite the 1.0°C warmer rectal temperature, resulting in a thermoregulatory behaviour score of 1.00 ± 0.13. The age groups displayed similar responses when comparing the CCC to the CHC trials and the CCC compared with the W+CHC. When examining thermoregulatory behaviour within each trial the age groups differ in the CCC, CHC, and W+CHC trials. The older men increased power output as time increased during the CCC trial while the young men decreased. The young men responded to the ambient temperature increase during the CHC and the W+CHC trials by reducing power output (thermoregulatory behaviour scores: CHC 1.09 ± 0.12, W+CHC 1.08 ± 0.16) while the older men increased power output (thermoregulatory behaviour scores: CHC 0.94 ± 0.09, W+CHC 0.97 ± 0.07).

This was the first study to examine thermoregulatory behaviours during exercise in changing ambient conditions with older men. We utilised radiant heat lamps during cycling exercise to identify thermoregulatory responses to increases in skin temperature. This step was undertaken to manipulate skin temperature while minimising their impact on rectal temperature. Radiant heat lamps have been used previously with young (~30 years) men rowing for 5,000 m at a fixed RPE of 15 in changing ambient conditions (radiant heat lamps turned on at 1,000 – 2,000 m and 3,000 – 4,000 m distance; Lander et al., 2014). Skin temperature increased by 0.51°C during the first warming phase, and power output dropped by 6% ($p = 0.03$). During the second warming phase skin temperature increased by 0.15°C, power output reduced by 4% ($p = 0.10$. Lander et al., 2014). In our study the heat lamps caused a ~5°C and a ~3°C increase in skin temperature for both age groups in the CHC and W+CHC trials, respectively, to ~35°C. This caused significantly different thermoregulatory behavioural responses in the young and older men in the CHC trial. The young men reduced their power output when the radiant heat lamps were activated. This was a similar response as was reported by Lander et al. (2014). The older men however, increased their power output when the radiant heat lamps were active and skin temperature increased. Their thermal comfort also increased so they felt warmer but did not reduce their metabolic heat load. This is an important finding as it appears that older men ignore or unable to respond to drivers of thermoregulatory behaviour.

This has public health implications, especially given that adults aged 65 years and over are vulnerable during heat wave events. The inability of older men to respond to changing skin temperature and thermal perception may prevent them from mitigating their heat risk via compensatory thermoregulatory behaviours. This effect may add to the strain on health services during heat wave

events, and in the aftermath, as those who suffer severe heat illness/stroke may lose physical independence as a result. Advice given during heat wave events should include guidance that during exercise in the heat older men are unable to voluntarily respond to increases in skin temperature, so should be mindful to reduce activity levels even if they do not perceive themselves to be at risk.

Previous work using a fixed RPE cycling protocol indicated young men (<35 years) drop to 70% of their starting power output in ~22 min (Flood et al., 2017), and ~33 min (Parton et al., 2021), albeit using an RPE of 16 (between 'hard' and 'very hard'). We expected that power output would decline during all the trials, but at differing rates. The younger men behaved as anticipated, their power output declined from the first to the last 10 min in the CCC trial (thermoregulatory behaviour 1.07 ± 0.15). Whereas the older men maintained their power output throughout the trials with little effect of time, pre-warming, or changing ambient conditions. In the CCC trial the older men increased power output from the first to the final 10 min (thermoregulatory behaviour 0.94 ± 0.10). It appears that older men do not voluntarily adjust their power output in response to an elevated rectal temperature, to rapid increases in ambient and skin temperatures, or to increases in thermal comfort and thermal perception. This inability to reduce metabolic heat load during heat exposure will put them at an elevated risk of heat illness during heat wave events that are becoming more severe and more common.

The pre-warming trials in the present study caused a ~2°C warmer mean skin temperature, for both age groups, at the onset of exercise than the non-pre-warmed trials (~32°C v ~30°C), alongside the 1°C increase in rectal temperature. Skin temperature at the onset of exercise is known to influence self-selected exercise intensity in young (30 ± 9 years) men during a 60 min cycling time trial (Schlader et al., 2011b). In the present study young men had a lower cycling power output at the start of the cycling exercise when skin and rectal temperature was elevated prior to exercise (thermoregulatory behaviour value: CCC/W+CCC = 1.10 ± 0.12; CHC/W+CHC = 1.12 ± 0.27). The older men did not respond to an increased skin and rectal temperature at the onset of the W+CCC and W+CHC trials as their cycling power output was not different from the CCC and CHC trials, respectively (thermoregulatory behaviour value: CCC/W+CCC = 1.00 ± 0.13; CHC/W+CHC = 1.04 ± 0.11). These findings differ to those of Schlader et al. (2011b) who reported that at the onset of exercise, skin temperatures of ~35°C resulted in a 2.4% reduction in exercise intensity compared with skin temperatures of ~29°C (Schlader et al., 2011b). A mean skin temperature of 36.3°C resulted in resting young (~22 years) adults implementing cooling behaviours to a greater extent than when skin temperature is 34.5°C (Vargas et al., 2018b). In contrast to Vargas et al. (2018b), thermoregulatory behaviours were not implemented by the older group in the present study, despite a greater increase in mean skin temperature (2.0°C v 1.8°C). This difference in findings may be due to the lower absolute mean skin temperatures induced

in our study. Vargas et al. (2018b) and Schlader et al. (2011b) were examining skin temperatures ~35°C, whereas we induced mean skin temperatures of ~32°C prior to the cycling exercise. However, the radiant heat lamps used in the CHC and W+CHC trials did cause mean skin temperature to rise to ~35°C during exercise for both the young and older men, yet the older men still did not implement thermoregulatory behaviours. While the young men implemented thermoregulatory behaviours in the W+CCC trial with a ~2°C increase in mean skin temperature, rectal temperature was also increased by 1°C. It is unclear if a ~2°C increase in mean skin temperature is sufficient to result in thermoregulatory behaviour implementation without the additional stimulus of an increased in core temperature.

The young and older men had similar rectal and mean skin temperature responses throughout the study. During the cycling exercise in the CCC and CHC trials rectal temperature increased for both groups. However, in the pre-warmed trials (W+CCC and W+CHC), rectal temperature remained stable once the participants exited the hot water (pre-exercise ~38.1°C, post-trial ~38.2°C), cycling cause no further increase in rectal temperature. Our data suggest that when men, regardless of age, exercise at a fixed RPE of 13 for 30 min, they do not produce enough metabolic heat to increase core temperature above ~38°C. An elevated rectal temperature at the onset of exercise did not cause thermoregulatory behaviours to be implemented in the older men. The young men had a mixed response, they reduced power output in the first 10 min of the W+CCC trial compared to the CCC trial, but did not adjust power output in the W+CHC trial compared to the CHC trial. Our data suggests that rectal temperature at the onset of exercise does not drive thermoregulatory behaviours in older men and has a limited influence in young men. During fixed intensity cycling (50% $\dot{V}O_2$peak), young men (24 ± 3 years) reported greater RPE scores when their oesophageal temperature was 37.5°C at the start of exercise than when it was 36.9°C (Tsuji et al., 2012). Thus, it was anticipated that an elevated rectal temperature prior to exercise would result in thermoregulatory behaviours being implemented via reduced power outputs.

It has consistently been shown that older men are less sensitive to changes in ambient temperature. When at rest, men aged 60 years and over feel more thermally comfortable in changing ambient temperatures than young men, and when given control of a dual position thermostat, allow greater fluctuations of ambient temperature (Collins et al., 1981; Natsume et al., 1992; Taylor et al., 1995). Older men (66-75 years) have also been shown to feel more comfortable than young men (22-27 years) after exiting a hot bath (42°C) and sitting at rest in 20°C conditions (Ono et al., 2017). Older men have a greater range for thermo-neutrality, and they need to experience a greater thermal input before they initiate thermoregulatory behaviours. This appears to be the case during exercise as well. The older men felt warmer than the young men in the CCC trial yet increased their cycling power

output throughout this trial. Both age groups experienced similar thermal perception at the onset of the trials and during the radiant heat lamp exposures. Yet during the CHC trial the older men increased cycling power output, while the young men reduced cycling power output when the radiant heat lamps were active. This study indicates that older men are less sensitive than young men to changes in thermal perceptions while exercising in stable and changing ambient conditions.

Aerobic fitness and body composition are mediators of heat illness risk, a greater BMI and proportion of body fat, and lower aerobic fitness increases risk of heat illness. (Westwood et al., 2020). In the present study, the young and older men had similar anthropometry, with stature, body mass and body composition being similar between the young and older men. However, the young men tended to have a greater body mass, BMI, and sum of skinfolds. This was due to two of the younger participants having a body mass of 110 kg and 121 kg, whereas the heaviest older man was 93 kg. As a higher body mass and BMI is associated with a greater risk of heat illness, the older men may have been at a slight advantage in the present study. Despite the anthropometric outliers, both age groups had similar fitness levels as the total power output in the CCC trial did not differ between the age groups (young = 510 W, older = 3171 W. $p = 0.477$). Age was the only baseline measurement that differed between the age groups. Thus, it seems that the impaired thermoregulatory behavioural response is an age-related decline rather than associated with body composition or fitness level.

Experimental Chapter Three was designed to try and increase the amount of heat stress experienced by the participants as Experimental Chapter One: Relationship between age and ability to implement thermoregulatory behaviours in thermoneutral and hot ambient conditions and Experimental Chapter Two: Physiological and perceptual factors influencing behavioural thermoregulation in young and older men in thermoneutral and hot ambient conditions had only caused rectal temperature to reach ~38°C at the end of the exercise in the hot trials. The implementation of pre-warming with hot water immersion caused rectal temperature to reach ~38°C prior to the exercise starting. This outcome was achieved despite differing transfer times from the bath to the cycle ergometer for individual participants. They were allowed to exit the bath and change clothes at their own pace to reduce the risk of fainting and injury after being immersed in hot (~42°C) water. However, rectal temperature did not increase further in either the W+CCC or W+CHC trials for either the young or older men. It appears that when men exercise at an RPE of 13, rectal temperature plateaus at ~38°C.

A limitation of the present study was that participants could not be blinded to the heat lamps being turned on or off. The rapid increase in ambient temperature allowed them to perceive this increase relatively soon after they were turned on. However, participants were not provided with any feedback on their power output, speed, cadence, or heart rate, and were unaware of the purpose of the study.

15.12 Conclusion

We demonstrated that when skin and rectal temperature are elevated prior (CCC compared with W+CCC) to exercise young men implement thermoregulatory behaviours by reducing power output, whereas older men do not implement these behavioural responses. However, this was a mixed response as the W+CHC trial did not result in thermoregulatory behaviours being implemented at the onset of exercise (CCC compared with W+CHC). During changing ambient conditions, young men are able to implement thermoregulatory behaviours in response to ambient temperature increases, whereas older men are not. These outcomes are evident despite younger and older men in this study having similar anthropometry (height, body mass, body composition) and physical fitness characteristics. There was also no difference in skin or rectal temperature response between the age groups, indicating that older men are less sensitive to changes in skin temperature. Public health advice should emphasise that being aged 60 years or above alone is a risk factor for heat-related illness, even in individuals with no pre-existing health issues.

. However, consistently throughout this thesis men aged ≥60 years did not implement thermoregulatory behaviours via reducing walking speed or cycling power output. Heat wave advice for older adults should emphasise that they may feel fine when in fact they need to initiate cooling strategies.

15.13 Possible Mechanisms of Reduced Thermoregulatory Behaviour in Older Men

Thermoregulatory behaviours are implemented when thermal discomfort is experienced. Older men in this thesis consistently reported similar levels of thermal comfort on the 7-point Bedford scale to young men who did implement thermoregulatory behaviours, yet older men did not implement thermoregulatory behaviours. Older adults have a different perception of thermal comfort than younger adults (Natsume et al., 1992; van Hoof & Hensen, 2006), and are less sensitive to changes in ambient temperature (Collins et al., 1981; Waldock et al., 2018). We anticipated that older participants would feel more thermally comfortable than the younger participants while exercising in the heat and this would drive any reductions in ability to implement thermoregulatory behaviour. While there was

no age-related difference in thermal comfort with both young and older men experiencing greater feelings of warmth in the heat, this only led to a decrease in exercise intensity for the young men. Older men did not reduce exercise intensity despite greater feelings of warmth in the hot conditions. Older adults prefer feeling warmer (Taylor et al., 1995) and require a greater level of thermal discomfort than their younger counterparts before initiating behavioural adaptations. This puts them at an increased risk of heat-related illness as they are unaware and unable to voluntarily reduce their thermal strain while exercising in conditions similar to those experienced during heatwave events experienced in the UK.

During Experimental Chapter Two: Physiological and perceptual factors influencing behavioural thermoregulation in young and older men in thermoneutral and hot ambient conditions whole body sweat rate was similar between the older and younger men, suggesting that skin wetness was also similar. Skin wetness accounts for ~50% of the drive to behaviourally thermoregulate, whereas mean skin temperature and core temperature contribute ~26% and ~22%, respectively (Vargas et al., 2018a). The inputs into thermal comfort (skin temperature and skin wetness) are similar between age groups in this thesis, resulting in comparable thermal comfort perceptions during the hot trial in Experimental Chapter Two. This comparable input resulted in similar thermal perceptions but resulted in differing thermoregulatory behavioural outputs. This implies that there is an impairment in signalling somewhere between the skin thermoreceptors and mechanoreceptors and the hypothalamus, or a failing of the hypothalamus to initiate a behavioural response to the information it receives.

Young men experienced skin blood flow and whole body sweat increases during the hot trial, and voluntarily implemented thermoregulatory behaviours in Experimental Chapter Two. The older men experienced an increase in skin blood flow and whole body sweat rate but did not implement thermoregulatory behaviours. Thermoregulatory strategies are implemented in a co-ordinated manner that ensures energy is not wasted. First, vasoconstrictor tone is removed to increase skin blood flow, then thermoregulatory behaviours are initiated, and finally sweating and vasodilation are implemented (Schlader et al., 2018; Schlader et al., 2016a). By increasing sweat rate prior to thermoregulatory behaviours, the older men made inefficient use of energy and water. Over a prolonged period, such as during a heat wave, this will result in a greater level of physiological strain through energy depletion and increased risk of dehydration if water is not adequately replenished. It could be that thermoregulatory behaviour declines before sudomotor function however, this thesis did not establish the maximal sweat rate of participant groups. Sudomotor function is known to be reduced in men aged >64 years compared with men aged 20-24 years (Inoue et al., 1999, 2004). The

heat stress used in this thesis may not have required maximal sweat rate, so the decline in sudomotor function may not be apparent under self-selected 'somewhat hard' exercise in 35°C.

The hot water immersion used in Experimental Chapter Three had the desired effect of elevating rectal temperature before the cycling exercise by ~1°C. The radiant heat lamps elevated skin from baseline by ~4-5°C and ambient temperature by ~11°C while they were turned on. The young and older men implemented thermoregulatory behaviours between trial conditions in a similar manner. However, within trials the older group did not implement thermoregulatory behaviours as effectively as the younger men towards the end of the CCC trial. The younger men reduced their power output at time increased, however the older men increased their power output. The younger men implemented thermoregulatory behaviours in response to ambient temperature increases, however the older men did not. This resulted in a significant difference in thermoregulatory behaviour during the CHC trial and tended to be different during the W+CHC trial. Thus, changing ambient conditions have little effect on power output in older men. As skin temperature was similar between the age groups, it seems that thermo-sensation signals from the skin to the hypothalamus may be hindered in older men. Fixed RPE rowing in changing ambient conditions has been used previously (Lander et al., 2014); this study saw similar results in their young male participants as the young men in Experimental Chapter Three. Young men reduced rowing power output by 4-6% when radiant heat lamps were activated for ~4 min (Lander et al., 2014). Young men can implement thermoregulatory behaviours by reducing exercise intensity within 10 min of a ~11°C increase in ambient temperature, while older men cannot. This response to changing ambient temperature was similar between the pre-warmed and non-pre-

warmed trials. Rectal temperature does not need to be elevated for young men to implement thermoregulatory behaviours in changing ambient conditions.

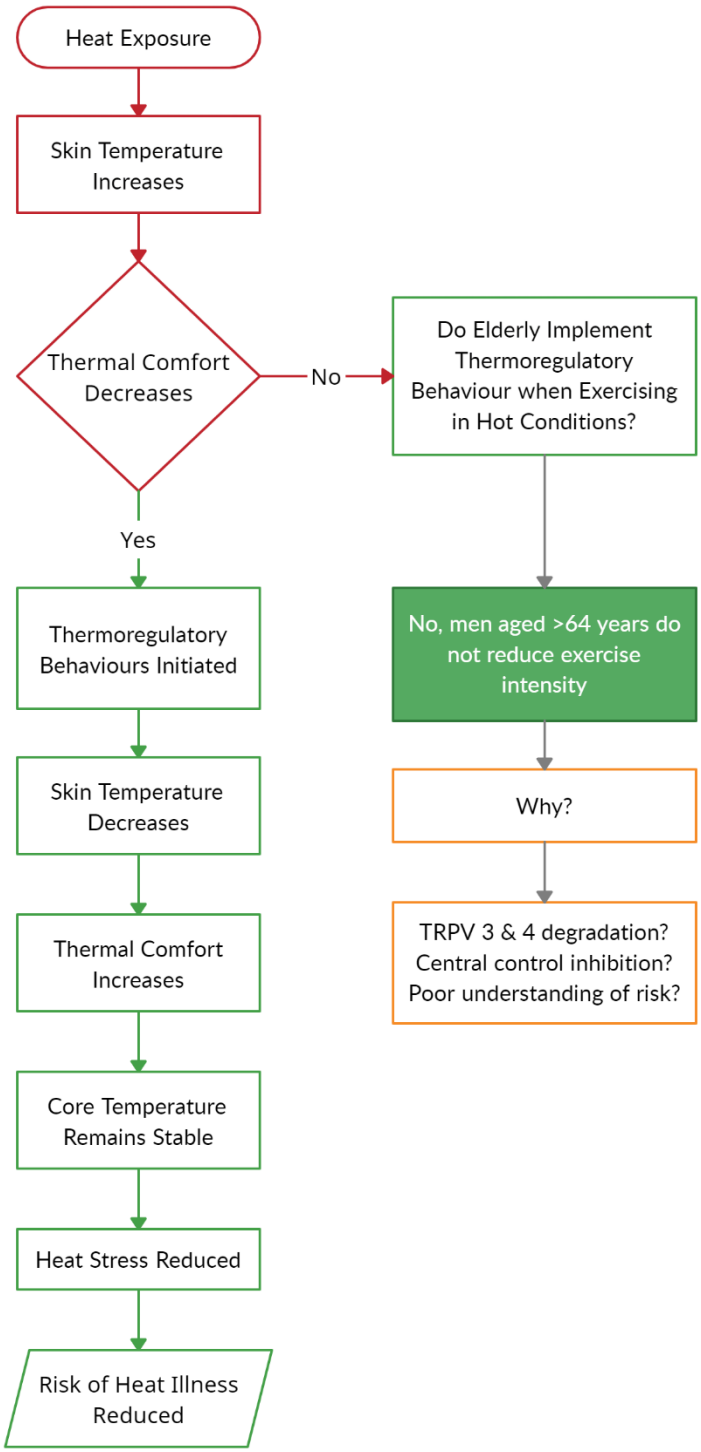

Figure 15-1. *Flow diagram of behavioural thermoregulation pathway (Figure 10-1) updated with findings from this thesis and future research questions.*

Total work done during Experimental Chapter Three was reduced in the young men after pre-warming compared to no pre-warming. The older men did not adjust their total work done after pre-warming. Rectal temperature at the onset of exercise drives thermoregulatory behaviours in young men. Previously, skin temperature at the onset of exercise has been demonstrated to mediate thermoregulatory behaviour during cycling in young men (Schlader, Simmons, et al., 2011b). During the pre-warmed trials, the hot water immersion also elevated skin temperature at the onset of exercise by ~2°C. This increased skin temperature at onset will have contributed to the drive to reduce total work done by the young group. Skin temperature alterations caused by the ambient heat lamps during the cycling did not markedly influence the total amount of work completed. It appears that skin temperature mediates thermoregulatory behaviours at the onset of exercise, but not during exercise.

The older men did not adjust their power output in any of the trials in Experimental Chapter Three. This was unexpected as we anticipated that power output would drop as participants became fatigued. During the control trial the young men dropped their power output by ~9% from start to finish, whereas the older men increased power output by ~6%. When young (~23 years) men cycle at a fixed RPE of 16 (between 'hard' and 'very hard'), power output declines linearly and this rate of decline is faster in 35°C conditions than in 15°C or 25°C (Tucker et al., 2006). During rowing at a fixed RPE of 15 ('hard') power output also declined by ~10% in young (~30 years) men while exposed to changing ambient temperature (Lander et al., 2014). The use of a lower RPE in Experimental Chapter Three than in Lander et al. (2014) and Tucker et al. (2006) may have resulted in insufficient fatigue being induced to cause a drop in power output.

Our findings suggest that older men may have impaired signalling from thermo-receptors in the skin. Skin thermo-receptors transmit information to the hypothalamus via transient receptor potential (TRP) channels (Caterina, 2007). TRP vanilloid (TRPV) 3 & 4 are involved with non-noxious warmth sensations in the skin; TRPV3 is activated at temperatures >33°C, and TRPV4 is activated at ~27-42°C (Patapoutian et al., 2003; Tominaga, 2007). In Experimental Chapter One and Two skin temperature during the cool trials was ~30°C, but in the hot trials it was ~35°C. TRPV4 channels would have been activated in both trials, but TRPV3 would only have been activated during the 35°C trials. Skin temperature at the onset of the trials in Experimental Chapter Three was ~29-32°C. TRPV4 would have been activated, whereas TRPV3 would not have been. When the radiant heat lamps were activated skin temperature rose to ~35°C, so TRPV3 channels should have become activated. TRP channels send thermal information the dorsal root ganglion to the spinal cord and then to the preoptic anterior hypothalamus (Romanovsky, 2007). If the TRPV3 channels are inhibited in older adults the preoptic anterior hypothalamus may not receive the information it needs to implement cooling strategies, including thermoregulatory behaviours.

Detection of temperature change at the skin is reduced in adults >60 years, particularly at the periphery (hand, foot, calf), when compared with middle-aged (40-50 years) and young (10-30 years) adults (Dufour & Candas, 2007). These adults did not have reduced perception to vibro-tactile stimulus (Dufour & Candas, 2007), indicating that the impairment in thermal stimulation is due to local impairment in the skin rather than central impairment. If thermal perception is inhibited in older adults, central control of thermoregulatory behaviour in the hypothalamus will not receive the stimulus to initiate behavioural interventions until a greater thermal threshold has been reached for older adults. Pain thresholds in the skin also increase with ageing, resulting in a greater incidence of injury and trauma to the skin (Bonté et al., 2019). Reduced sensitivity to stimuli at the skin surface has a detrimental effect on health in ageing. To help maintain thermoregulatory behaviours in older adults, interventions should focus on increasing the TRP channels sensitivity to temperature changes.

The trials used in this thesis were relatively short compared to the length of heat exposure experienced during heat wave events which, by definition, last for days. We have shown that older men are unable to implement thermoregulatory behaviours, even when rectal temperature is elevated prior to undertaking exercise. If older men have an elevated core temperature from exposure to a heatwave, they are unlikely to implement thermoregulatory behaviours in their day-to-day life.

15.14 Limitations

We recruited participants from the local population in Plymouth. The older participants who volunteered for this study had similar levels of fitness and physical activity as the young group. We did not specifically intend to match the participant groups for fitness and physical activity. As it was clear that participation involved exercise in hot conditions, less physically active older men may have been reluctant to volunteer. For the older men to maintain a similar level of fitness as the young men they would have to undertake aerobic training as maximal oxygen uptake declines at a rate of ~1% per year (Astrand et al., 1973). Anecdotally, many of the older participants reported being active members of running clubs and/or regularly completing organised running or triathlon events. Whereas the younger group reported little involvement with organised training and events. As training status rather than aerobic fitness is associated with exercise induced heat acclimation (Ravanelli et al., 2021), the structured training undertaken by the older men may have provided them with an exercise induced heat acclimation advantage over the younger participants.

Despite the deficit in thermoregulatory behaviour observed throughout this thesis, the older participants never experienced a warmer rectal temperature, or higher heart rate than the younger participants. This suggests that the level of heat strain was similar between all the participants in each experimental chapter. This could have been due to the absolute amount of exercise completed by

individuals. In Experimental Chapter One and Two the younger men walked ~2.7 km and older men walked ~2.6 km in the 35°C trials. While cycling in Experimental Chapter Three the average power output for the young men across the trials was ~110 W and for the older men ~105 W. There is a tendency for the older participants to use a lower absolute exercise intensity. This will result in a lower level of metabolic heat gain so rectal temperature will not rise as quickly in the older men as in the younger men.

The reproducibility of using a fixed RPE approach with an older adult population is somewhat unknown. A fixed RPE approach is not novel and has been used with a young adult population (Tucker et al. 2006). RPE is a widely used measure of exercise intensity, however, as it is a subjective measure it is not possible to objectively confirm if a participant is reporting the correct RPE (Williams, 2017). Each study in this thesis employed a familiarisation session allowing participants to acquaint themselves with the RPE scale and how an RPE of 13 (somewhat hard) feels to them. Participants were able to reproduce their walking speed or cycling power output within 4 ± 1 attempts.

15.15 Future work and recommendations

We demonstrated that with increased age there is a reduction in the ability to implement thermoregulatory behaviour during exercise in hot conditions, particularly in men aged ≥60 years. The thesis has been unable to identify what is causing this deficit as the drivers of thermoregulatory behaviours did not differ between the age groups studied; rectal and skin temperatures, thermal comfort, and whole body sweat rate were all similar between the age groups examined. The cause of the deficit remains to be elucidated. The ambient temperatures used in this thesis are representative of recent maximum temperatures experienced in the UK (Met Office, 2021). Our findings suggest that older men will be at greater risk of heat-related illness during heatwave events in the UK as they do not voluntarily implement thermoregulatory behaviours during exercise in 35°C ambient conditions.

The frequency and severity of heat waves is set to increase in the future (Met Office, 2018), and the population of adults aged ≥65 years is also on the rise (Office of National Statistics, 2021). The UK is less prepared now than it was 5 years ago for a heat wave (Climate Change Committee, 2021). Infrastructure and architecture have not been updated sufficiently to ensure that homes are kept cool in the summer. The onus to reduce heat-related health risk will therefore fall on the individual to adopt behavioural thermoregulation strategies during heat wave events. The lack of ability to behaviourally thermoregulate in adults aged ≥65 years is a public health concern as this age group are already at increased risk of heat illness through diminished autonomic thermoregulatory capacity (Inoue et al., 2004). Public Health England provides advice and recommendations to at risk populations during heat wave events, and uses heat-health alerts to inform the public of the current heat risk

(Public Health England, 2018). This guidance refers to the "elderly" as being a vulnerable population. Our work has demonstrated that adults, particularly men, have an impaired behavioural thermoregulation ability, and therefore increased risk of heat strain, by the age of 60 years. We suggest using age categories rather than subjective terms like "elderly" as this will be interpreted differently by individuals resulting in many underestimating their own heat-related illness risk. Public health guidelines should explicitly state that adults aged 60 years or over are at an increased risk of heat illness during heatwaves events. This will ensure the guidelines cannot be misinterpreted.

Despite Public Health England's guidelines, research has demonstrated that while the majority of survey respondents aged ≥70 years in the UK understand that increased age is a risk factor for heat-related illness, they do not see themselves as at risk, even some individuals with other co-morbidities (Abrahamson et al., 2008). The same relationship is seen in the US, individuals perceive health risks of heatwaves to be higher in other members of the community than their own personal risk (Howe et al., 2019). This dissociation demonstrates that the public health messaging is being understood, but that people are unaware of their individual risk. An individual's perceived heat-related health risk during a heatwave mediates the use of cooling behaviours (Ban et al., 2019); thus, as individuals are underestimating their own level of heat-related health risk they may not implement necessary thermoregulatory behaviours during heat exposure. Our findings also show that older men report the same level of thermal comfort as young men during heat exposure, while this results in the young men implementing thermoregulatory behaviours, the older men do not initiate thermoregulatory behaviour. We recommend educating the public on why heatwave guidance is issued, emphasising that individuals often underestimate their personal risk level. The focus should be that age alone is a risk factor for heat-related illness, regardless of underlying health issues, as many people with chronic health conditions subjectively categorise themselves as healthy when, objectively, they are at risk based on their Hierarchal Conditions Category (Tkatch et al., 2017). The guidelines should also state that while older adults may feel thermally comfortable during a heat wave event, they may be experiencing heat-stress and cooling behaviours should be implemented early, before older adults feel the need to.

Physical activity guidance from the UK government is for all adults to undertake at least 150 min of moderate, or 75 min of vigorous aerobic activity a week, or some combination of the two (Davies et al., 2019). Maintaining aerobic fitness and undertaking physical activity induces a small level of heat acclimation (Ravanelli et al., 2021). Thus, older adults who undertake regular exercise training may have protection during a heat wave event. The outcomes from this thesis indicate that advice on physical activity and exercise for ≥60-year-olds should be to maintain regular aerobic training throughout normal day to day life. However, when a heat wave occurs minimising heat stress takes

priority over exercise training and the need to meet activity guidelines. If older adults must undertake exercise training during a heat wave event, they should be advised to do so in air-conditioned gym facilities and to use cooling strategies (for example: cool shower, drinking ice slush) after their exercise session, even if they feel thermally comfortable. These precautions should help to reduce any core temperature increase during exercise and return core temperature to thermoneutral prior to leaving the air-conditioned environment.

There remain unanswered questions on thermoregulatory behaviour in ageing and heat exposure. First, the relationship between age and women requires further investigation. We could find no reason for the apparent increase in behavioural thermoregulation above the age of 70 years in the female cohort. This result appears to relate to two exceptional participants who stood out for their capacity to implement thermoregulatory behaviours, but not in any of the other physiological markers assessed. Inclusion of younger female participants, with considerations for menstrual cycle influences on thermoregulation, will help elucidate the relationship between behavioural thermoregulation and ageing in females.

Secondly, the mechanisms behind the impairment in behavioural thermoregulation needs to be elucidated. We demonstrated that the drivers of thermoregulatory behaviour (skin and rectal temperature, and thermal comfort) respond similarly in men aged ≥65 years as men aged ≤35 years, yet the older men were unable to implement thermoregulatory behaviours. Heat stress increases cognitive load and reduces cognitive performance (Cedeño Laurent et al., 2018; Gaoua et al., 2018). Students without access to air-conditioning during a Boston, US summer experience a ~4-13% decline in cognitive functioning compared to those with access to air-conditioning (Cedeño Laurent et al., 2018). The average housing temperature with air-conditioning was ~21°C, without air-conditioning ~26°C (Cedeño Laurent et al., 2018). Passive hyperthermia (core temperature ~39°C) causes a cognitive strain and electroencephalogram (EEG) alterations in men aged ~35 years (Gaoua et al., 2018). Simple cognitive tasks (One Touch Stockings of Cambridge, OTS-4) were not impaired, however, complex cognitive tasks (OTS-6) were impaired (Gaoua et al., 2018).

EEG activity alterations in men aged ~34 years are also apparent during a self-paced 750 kJ cycling time trial in 35°C conditions (Périard et al., 2018). Compared to 18°C conditions, alpha and beta activity in the frontal and central areas of the brain is decreased. Alpha activity is required for focus, mental readiness, higher levels of alpha activity increases the ability to block out unwanted stimuli (Thompson et al., 2008). Beta activity is associated with wakefulness, mental activity (Thompson et al., 2008). Thus, during self-paced exercise in the heat, the ability to focus and ignore irrelevant stimuli, wakefulness, and mental activity are all reduced. Périard et al. (2018) reported strong correlations (R

= 0.982) between alpha activity in the frontal region and RPE, core temperature, and thermal comfort, and beta activity is associated with thermal comfort (R = 0.951). RPE, core temperature, and thermal comfort responses were all similar between the age groups in this thesis, so they should have influenced brain activity to a similar extent. However, with ageing there is a known decline in overall EEG power of ~5% per decade (Vysata et al., 2012). Work is required to determine if older adult's attenuated ability to perceive exercise exertion is the result of a reduction in cognitive functioning.

15.16 Conclusion

We have demonstrated that older adults, particularly men, are unable to implement thermoregulatory behaviours when exercising in 35°C and changing (22-33°C) conditions. This is despite experiencing similar skin and rectal temperature, thermal perception, cardiovascular, and sweating responses to young men who were able to implement thermoregulatory behaviours in these conditions. Future work should focus on elucidating what is causing this decrement in thermoregulatory behaviours and clarifying the response in ageing women. Together with our work, this will help to inform the necessary interventions and policies required to reduce heat-related illness during future heat wave events. Public health guidelines should be updated to focus on objective age guidance rather than the term 'elderly' currently being used by PHE. Guidance should clearly state that anyone aged 60 years and over is at an elevated risk of heat-related illness, regardless of health status.

16 References

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17 Appendix

17.1 Covid-19 statement

We had to close our laboratory due to the coronavirus pandemic in March 2020. This prevented any further data collection for this thesis. As it was unclear when we would be able to re-open the laboratory, and *a priori* power analysis indicated that 9 participants per group was sufficient, we decided to move on to the data analysis and write of this study with 10 older and 9 younger participants in Experimental Chapter Three rather than evening up the group numbers.

17.2 Conference abstracts

17.2.1 International Conference on Environmental Ergonomics – 2019 - Amsterdam

Influence of age on perceptual responses to thermal stress at rest and following exercise

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Word limit: 300 words

Introduction: Studies have shown that older adults are less able to dissipate heat during exposure to high environmental temperatures. Recent work has indicated that older adults may also be less able than younger adults to implement protective behavioural changes during exercise in the heat, increasing their risk of heat illness.

Method: This study employed a randomised crossover design using Cool (22°C, 40% RH) and Hot (35°C, 40% RH) environmental conditions to compare thermoregulatory behaviour between young (18-35 yrs, n=10) and older (≥65 yrs, n=14) adults during 3 x 10 min of walking at a rating of perceived exertion (RPE) of 13. After a baseline visit including familiarisation with RPE, participants completed two experimental trials, (Cool and Hot). Participants sat at rest for 45 min upon entering the environmental chamber, after which they began walking on a motorised treadmill. Speed was blinded from participants and self-selected to elicit an RPE of 13. Distance (km) walked

during each bout was recorded. Rectal temperature ($T_{rec,}$ °C) and heart rate (HR bpm) were recorded every 5 min.

Results: Markers of physiological strain between the Cool (22.1 ± 0.29°C, 42 ± 5% RH) and Hot (35.1 ± 0.23°C, 41 ± 5% RH) trial increased for the older (HR 80 v 93 bpm, $p<0.01$; T_{rec} 37.03 v 37.85 °C, $p<0.01$) and younger group (HR 91 v 98 bpm, $p<0.05$; T_{rec} 37.57 v 37.99 °C, $p<0.05$). The older and younger group reduced their total distance walked by 1.5% and 8.0% respectively. Within the older group, a gender difference was evident with the women (n=5) walking 6.7% less distance in the heat, but the men (n=9) walked 1.5% further in the heat.

Conclusions: Older adults, particularly men, appear to be less able to adapt their behaviour during heat stress, resulting in an increased thermal and physiological strain.