Physiological and Inflammatory Responses to UK Fire Service Instructors' Working Practices

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A thesis submitted in partial fulfilment of the requirements of the University of Brighton for the degree of Doctor of Philosophy

September 2018

ABSTRACT

This thesis aimed to provide evidence informed recommendations for UK Fire Service Instructor (FSI) working practices to reduce the risk of symptoms of ill health and cardiovascular events. Study 1 provides a detailed insight into the working practices of FSI, collected via a survey. Data indicated that FSI had little guidance on hydration, cooling methods, or fire exposure (wear) limits. In addition, 41% of FSI reported symptoms of ill health including: broken sleep, fatigue, headaches, mood swings, and coughing/breathing problems. To understand the acute responses to working practices, FSI were monitored during a wearing day for Study 2. A multi-compartment exposure, and the condition setter role within it, were noted to cause the greatest rise in core temperature. Training days that included a multi-compartment exposure also resulted in an increased inflammatory response. Pre-cooling methods, to minimise the rise in core temperature and inflammation, were assessed in a laboratory simulated wear in Study 3. A single 500 ml bolus of ice slurry was the only practical pre-cooling method to significantly reduce core temperature. To assess the chronic impact that repeated wears had on FSI, a heat occupational tolerance test (HOTT) was designed and assessed as valid and reliable in Study 4. Using the HOTT the tolerance status of FSI was compared to non-wearing controls in Study 5. FSI displayed an increased heat tolerance, suggestive of an acclimatised state. Improved tolerance was associated with an increased prevalence of ill health. Combined with elevated cytokine levels, this indicated FSI may exhibit a maladaptive response to frequent wears. Study 6 subsequently involved the collection of resting blood samples from 110 fire service personnel and demonstrated that FSI expressed signs of chronic systemic inflammation, which was linked to the number of wears that had been completed and symptoms of ill health similar to overtraining. FSI also exhibited elevated markers associated with cardiovascular risk. The informally suggested limit of 9 wears a month was found to be appropriate guidance, with those above the limit being 6 - 12 times more likely to express “at risk” levels for predictors of cardiovascular events and 16 times more likely to experience symptoms of ill health.

This thesis establishes that FSI are a unique population with a common collection of symptoms of ill health, and are at a chronic risk of elevated predictors of cardiovascular events and an overtraining like syndrome. To reduce the acute levels of inflammation and core temperature rises to fire exposures it is recommended that FSI roles are rotated, that exposure type is considered in course planning and that ice slurry pre-cooling is utilised. To reduce the prevalence of chronic systemic inflammation, symptoms of ill health, and cardiovascular risk, whilst maintaining an acclimatised state, a 9 wear per month limit is recommended.

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ABBREVIATIONS

ANOVA	Analysis of variance
BA	Breathing apparatus
BASO	Basophil
BF	Body fat
BSA	Body surface area
CFBT	Compartment fire behaviour training
CRP	C-reactive protein
cTNT	Cardiac troponin T
CV	Coefficient of variation
EHI	Exertional heat illness
ELISA	Enzyme-linked immunosorbent assay
EO	Eosinophil
FF	Firefighters
FSI	Fire service instructors
HCT	Haematocrit
HGB	Haemoglobin
HISI	Heat illness symptoms index
$\dot{H}_{prod}$	Heat production
HR	Heart rate
HTT	Heat tolerance test
HOTT	Heat occupational tolerance test
ICC	Intra-class correlation coefficient
IFNγ	Interferon gamma
IgG	Immunoglobulin G
IL-1β	Interleukin-1 beta
IL-6	Interleukin-6
LOA	Limits of agreement
LYMPH	Lymphocyte
MONO	Monocyte
MPV	Mean platelet volume
NEUT	Neutrophil
NLR	Neutrophil/lymphocyte ratio
PCV	Phase change vest
PLT	Platelets
PV	Plasma volume
PPE	Personal protective clothing
PSI	Physiological strain index
OR	Odds ratio
RBC	Red blood cells

RPE	Rating of perceived exertion
SR	Sweat rate
T_c	Core temperature
TEM	Typical error of the measurement
Th	T helper cell
TNFα	Tumour necrosis factor-alpha
T_{re}	Rectal temperature
TS	Thermal sensation
T_{skin}	Mean skin temperature
URTI	Upper respiratory tract infection
$\dot{V}O_2$	Volume of oxygen uptake
WBC	White blood cells

ACKNOWLEDGEMENTS

Firstly, to my supervisors, Alan, Mark and Peter, for your advice and guidance which has been invaluable throughout my time at the University of Brighton, I shall be forever grateful.

To Alan for giving me the opportunity to follow this amazing path in life that I never could have imagined. You have helped me to grow as both a researcher and a person.

To Mark, for all your thoughtful insights and words of encouragement.

To Peter, for every cheery morning greeting and for always having the patience and time to explain things, no matter the simplicity or complexity of the question.

To my participants and research assistants, without your hard work, time, and dedication this research would not exist.

To those in the Fire and Rescue Service that have been involved in this project, thank you for giving me the most memorable 4 years. Working with you provided me a constant reminder of why research is important.

To my fellow PhD students for sharing in this journey with me, reminding me that I was not alone, and joining me for both celebratory and motivational cake breaks.

To those friends who have continually encouraged and supported me, with unwavering enthusiasm and interest despite still not really knowing what it is that I do.

To my parents, for instilling in me a love of learning, and teaching me that what mattered most was being happy. Your unconditional support has enabled this thesis to be possible.

To Stephen, for standing by my side throughout this journey and for believing in me even when I did not believe in myself.

Thank you

DECLARATION

I declare that the research contained in this thesis, unless otherwise formally indicated within the text, is the original work of the author. The thesis has not been previously submitted to this or any other university for a degree and does not incorporate any material already submitted for a degree.

Signed:  Date: 26th September 2018

ACADEMIC PUBLICATIONS

The following published articles have been due to work contained within this thesis:

Chapter 4

Watkins, E. R., Hayes, M., Watt, P., & Richardson, A. J. (2018). Fire Service Instructors' Working Practices: A UK Survey. *Archives of Environmental & Occupational Health*, 1-9.

Chapter 5

Watkins, E.R., Hayes, M., Watt, P., Richardson, A.J. (2018). The acute effect of training fire exercises on fire service instructors. *Journal of Occupational and Environmental Hygiene*, 1-37.

Chapter 6

Watkins, E. R., Hayes, M., Watt, P and Richardson, A. (2018). Practical pre-cooling methods for occupational heat exposure. *Applied Ergonomics*, 70, 26-33.

Chapter 7

Watkins, E.R., Gibbons, J., Dellas, Y., Hayes, M., Watt, P. and Richardson, A., (2018). A New Occupational Heat Tolerance Test: A Feasibility Study. *Journal of Thermal Biology*, 78, 42-50.

1 INTRODUCTION

Occupational health is of great importance to minimise sick days taken by employees and to maximise individuals' wellness and ability to complete tasks (Sparks et al. 2001). Maintenance of workers health is of paramount significance when the occupation involves dangerous and extreme environments, due to the increased level of risk experienced by each employee (McDonough et al. 2015). Fire service instructors (FSI) are frequently exposed to high temperatures and hazardous situations when teaching trainee firefighters (FF) (Eglin et al. 2004). These live fire situations put them at risk of exertional heat illnesses (EHI) and cardiovascular events (Casa et al. 2015; Fahy et al. 2015). Anecdotally, instructors also report exhibiting increased cold and flu symptoms and reporting perfuse sweating that interrupts their sleeping patterns. There is little current research into the acute effects of a single fire exposure, also known as a "wear", on FSI (Eglin et al. 2004) and whilst the short term immune responses have briefly been reported for FF (Smith et al. 2004), the long term immunological changes have not yet been assessed. The high levels of physiological and perceptual strain experienced during a fire exposure (Petruzzello et al. 2009) suggests that a high frequency of wears may cause a response similar to that of over-trained athletes, where individuals experience an increased number of upper respiratory tract infections (URTI), persistent fatigue, altered mood states and decreased task performance (Mackinnon 2000). Although this concept is much discussed in the sporting world, there remains little empirical data to confirm its existence and to link the symptoms to altered immune function (Halson & Jeukendrup 2004). The opportunity to study a continually stressed population is often limited and therefore little is known about the long term changes in immune function that may occur with frequent exposure to high strain situations.

To be able to improve FSI health, the changes in immune function, physiological and perceptual strain and heat tolerance that occur over numerous wear exposures needs to be assessed. This knowledge will then allow for guidelines to be formed advising instructors on the ideal wear rotas to minimise decrements to their health. Practical interventions may also be suggested to aid instructors to reduce the thermal strain they experience.

Consequently, this thesis aims to gain an understanding of FSI current working practices in the UK and identify both the acute and chronic consequences of these practices. This information will then be used to form evidence based recommendations to improve FSI health. The experimental studies in this thesis involved both laboratory and field work, with an aim of maximizing the ecological validity of research designs when possible. Due to the nature of working directly with the UK Fire and Rescue Service, not all experimental studies were collected sequentially, with some overlap occurring between studies to enable data from adequate sample sizes to be collected. This thesis is presented in the following chapters:

Chapter 2 reviews the current literature pertaining to the acute and chronic physiological consequences of repeated fire exposures. Focus is given to the subsequent risk of heat illnesses, cardiovascular events and an overtraining like syndrome.

Chapter 3 details common methodology used throughout experimental chapters.

Chapter 4 (Study 1) outlines current working practices of UK FSI, in comparison to FF, from survey data collection. The association between practices and ill health is also investigated.

Chapter 5 (Study 2) investigates the acute physiological, perceptual and immunological responses to training fire exposures conducted by FSI. Differences in responses between the types of fire exposures, the roles performed by FSI and exposure patterns throughout a day are assessed.

Chapter 6 (Study 3) examines the effect of pre-cooling methods, which were identified in Chapter 4, on the acute physiological, perceptual and immunological strain from a simulated fire exposure.

Chapter 7 (Study 4) introduces the role of heat tolerance tests to assess FSI chronic responses to fire exposures. A new heat occupational tolerance test is evaluated for its validity and reliability.

Chapter 8 (Study 5) follows on from Study 4, using the heat occupational tolerance test to identify if FSI exhibit an acclimatised status in comparison to non-wearing individuals. This chapter also examines the presence of symptoms of ill health (as identified in Chapter 1) and subsequent associations with heat tolerance levels.

Chapter 9 (Study 6) leads on from Chapter 5 and investigates the chronic immunological consequences of repeated fire exposure in a larger sample size. Working history and presence of ill health amongst FSI and FF are assessed in relation to resting markers of immune function. Specific focus is given to the effect the number of fire exposures completed may have on levels of chronic inflammation, symptoms of ill health and predictors of cardiovascular event risk.

Chapter 10 highlights the principal findings from all experimental chapters, discusses the acute and chronic consequences of live fire exposures in FSI and consequently makes recommendations for FSI working practices.

2 LITERATURE REVIEW

2.1 Introduction

This literature review aims to provide an overview of the FSI role and identify the physiological consequences of acute fire exposures. The occurrence of EHI is then investigated, with discussion of EHI risk factors. An evaluation of different acute interventions that may reduce physiological strain then follows, with reference to recommendations currently provided within the Fire and Rescue Service.

Current literature regarding chronic consequences of frequent live fire exposures will then be reviewed. Focus will be given first to the risk of cardiovascular events, with an overview of potential mechanistic causes and then to overtraining, with a review of the cytokine hypothesis and possible links to occupational work.

Finally this review will investigate changes in immunological markers with heat exposure and exercise, with reference to how markers may be involved in systemic chronic inflammation, cardiovascular events, or overtraining.

2.2 Job Description

FSI train newly recruited and current FF across a wide range of skills, such as road traffic collisions, self-contained breathing apparatus (BA) and fire behaviour. Within the UK there are ~300 FSI, compared to approximately 34,400 FF (Fire Statistics Team & Home Office 2016). FSI themselves are specially trained to be able to teach trainees and must gain specific qualifications such as a Skills for Justice Level 3 Award in Breathing Apparatus Instructions or Compartment Fire Behaviour Training. FSI undergo the same physical employment tests as FF, which are currently recommended to include seven job specific tests (rural fire task, domestic fire task, ladder lift, ladder extension, ladder climb, pump assembly and enclosed space crawls) that are conducted at both selection and in a more developed form during service (criterion tests) (Blacker et al. 2016). FSI are also required to maintain a minimum of 42.3 $mL.kg.^{-1}min^{-1}$ $\dot{V}O_2$ max, which is the required standard of all FF (Siddall et al. 2016). FSI can work on secondment from their station or service, or work permanently at a training centre.

During sessions, FSI are responsible for the safety and well-being of trainees. Consequently, it is important that FSI are both physically fit and capable to complete rescues during training sessions, but also mentally alert, with the ability to make quick effective decisions. Previously, 3 out of 12 FSI have indicated they are unsure of their capabilities to conduct a rescue at the end of a live fire exposure (Eglin et al. 2004). An assessment of FSI rescue capabilities following a fire exposure suggests that whilst FSI are capable of performing an immediate 30m rescue drag of an 85 kg dummy, the scenario elicits near maximal heart rate (HR) (96 ± 5% of HR maximum) responses (Eglin & Tipton 2005). It is proposed that with individuals of lower aerobic capacity, in situations of greater environmental temperatures, increased casualty weight, or greater elevations in core temperature (T_c) a rescue may not be possible (Eglin & Tipton 2005).

Due to the nature of the occupation, FSI are required to complete wears on a frequent basis, meaning they are regularly exposed to high temperatures. Within purpose built fire training compartments temperatures range from 67 - 190ºC, with average temperatures experienced by FSI between 0.3m and 1.8m being 74 ± 42^{0}C (Eglin et al. 2004; Eglin 2007). However ambient temperatures measured from instructors' outside their protective clothing range from 55 ± 14°C at the shoulder and 42 ± 12°C at the hip (Eglin et al. 2004). The variation in temperatures is common within the fire house, with maximum temperatures noted at ceiling height and FSI staying low and sheltering where possible (Eglin 2007; Barr et al. 2010).

During a wear FSI are required to wear personal protective equipment (PPE) which includes: boots, tunic, trousers, fire hood, helmet and gloves. Typically this clothing consists of a flame resistant outer shell and an insulating thermal liner consisting of a moisture barrier and a thermal barrier. A BA must also been worn, which includes pressurized bottles of air that are supported by a metal back plate and adjustable straps at the shoulder, chest and waist (Bakri et al. 2012). The combined weight of the encapsulating PPE and BA is 16.9 - 27.4 kg (Barr et al. 2010). The weight varies depending on the brand of equipment used and the size of the gas cylinder carried.

2.3 The Effect of Heat on Fire Service Instructors

High temperatures experienced by FSI can disturb the thermoregulation of the body. Thermoregulation maintains the homeostasis of T_c at ~37°C, varying by ±0.8°C throughout the day, due to the circadian rhythm (Havenith, 2002). T_c naturally fluctuates during the day, reaching a peak plateau from 14:00 to 20:00 hrs and a minimum just after the mid-point of sleep, commonly around 05:00 hrs (Waterhouse et al. 2005). Increased ambient temperature can affect homeostasis, with changes superimposed upon circadian alterations (Waterhouse et al. 2005). Central thermoreceptors, in the preoptic anterior hypothalamus and peripheral thermoreceptors, in the skin, viscera and spinal cord, detect changes in temperature and, via afferent neurons, provide information to the hypothalamus (Illigens & Gibbons 2009). Consequently, heat loss mechanisms are instigated, including vasodilation of peripheral blood vessels and an increased sweat response (Kurz, 2008). Cutaneous vasodilation increases skin blood flow, consequently increasing convective heat transfer from the core to the periphery (Charkoudian 2016). Sweating is a sympathetic cholinergic response, resulting in an evaporative heat loss from the skin, consequently cooling the skin and increasing heat transfer from the surface of the skin to the environment, therefore dissipating large amounts of heat (Wendt et al., 2007). Figure 2.1 gives a schematic overview of the thermoregulation process.

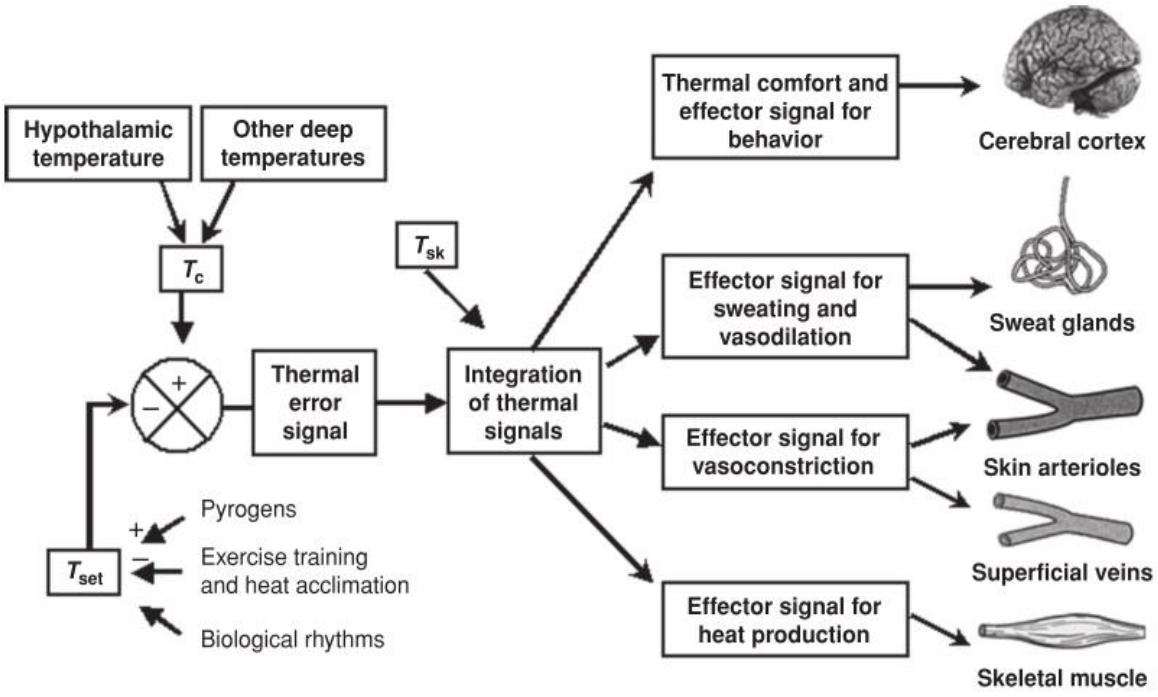

Figure 2.1 *Schematic of thermoregulatory control system, from Sawka & Young (2006). T_c = core temperature, T_{sk} = skin temperature, T_{set} = temperature set point.*

In addition to exogenous heat gain, heat is also produced during the conversion of metabolic energy into mechanical and thermal energy in all body cells. Due to the inefficiency of this process energy released by muscle contractions is 30 - 70% thermal (Krustrup et al. 2001; Bangsbo et al. 2001; González-Alonso 2012). During low intensity, steady-state exercise, like that used by FSI, heat production increases markedly at the onset, before plateauing. This pattern of heat production reflects the initial utilization of adenosine triphosphate (ATP) from primarily phosphorylcreatine catabolism, causing a heat production of 35 kJ.molATP^{-1} (Woledge & Reilly 1988), which then switches to primarily oxidative phosphorylation, with a heat production of 75 kJ.molATP^{-1}, once a steady state is established heat production then plateaus (Curtin & Woledge 1978; González-Alonso 2012). Metabolic heat production ($\dot{H}_{prod}$) therefore also stimulates heat loss mechanisms (Brotherhood, 2008).

To maintain T_c during exogenous and endogenous thermal gain the body must be able to lose sufficient heat. This balance to prevent heat storage can be represented by the heat exchange equation:

$$\pm S = M - (\pm W) - E \pm K \pm C \pm R \left(\frac{W}{m^2}\right)$$

where: S is heat storage, M is metabolism, W is work, E is evaporation, K is conduction, C is convection and R is radiation (Gavin 2003).

To prevent heat storage the body mainly relies on sweat loss to cool the body via evaporation. The ability to lose heat via radiation, convention and conduction is dependent on the surrounding environment. In a hot environment heat loss is impaired, due to a decrease in the thermal and water pressure gradients between

the body and the environment (Cheung et al. 2000). For FSI the radiation of electromagnetic waves causes heat gain during a wear, as the ambient temperature is greater than that of the outer clothing layer (Havenith 2002). In addition, conduction of heat via contact points with surrounding surfaces will cause heat gain for FSI, due to the surrounding surface temperatures. Finally, the movement of hot air past instructors will also add to the exogenous thermal load (Cheuvront et al. 2004; Morel et al. 2014).

In an attempt to dissipate heat, blood flow is redistributed to cutaneous circulation, competing with skeletal muscle blood flow requirements, resulting in a reduction in stroke volume and arterial pressure (Sawka et al. 2011; Périard et al. 2016). Consequently increased cardiovascular strain is experienced, with HR increasing to maintain cardiac output (Eglin 2007; Sawka et al. 2011; Périard et al. 2016). High sweat rates combined with minimal fluid replacement due to the live fire environment lead to reductions in plasma volume and consequently stroke volume (Charkoudian 2016). The combination of a 1°C rise in T_c with 4% dehydration can lead to a 20 ± 1% reduction in stroke volume and consequently a 13 ± 2% reduction in cardiac output (González-Alonso et al. 1997). Therefore, the cardiovascular system is put under increasing strain to maintain physical exercise whilst dissipating heat (González-Alonso et al. 2008).

Moreover, wearing PPE decreases an individual's ability to dissipate heat, as there is limited water vapour permeability across the clothing layers (Cheung et al. 2000). This is evident in the greater water vapour resistance of PPE (28.85 m^2 Pa/W) (Atalay et al. 2015) compared to a 100% cotton t-shirt (3.1 m^2 Pa/W) (Raccuglia et al. 2018). In addition, the further the distance away from the skin that moisture evaporation occurs the greater the reduction in the cooling efficiency of evaporation (Havenith et al. 2013). The weight of the PPE and BA also increases the work load experienced by instructors, therefore increasing heat production and cardiovascular strain (Huck 1988; Holmér et al. 2006). During stepping, walking and obstacle exercises in ambient temperatures metabolic rate increased by an average of 14.7% when wearing 6.66 kg of PPE compared to wearing tracksuit bottoms and a t-shirt (Dorman & Havenith 2009). Carrying a BA also increases O_2 consumption, with an 11 kg BA resulting in a $\dot{V}O_2$ of 28.1 ± 3.3 $mL.kg^{-1}.min^{-1}$ compared to 21.6 ± 2.5 $mL.kg^{-1}.min^{-1}$ without a BA at the end of exhaustive exercise (Bakri et al. 2012). A theoretical model predicted that metabolic rate would increase by 1% per kg of additional weight (Givoni & Goldman 1971). However based on the linear regression line formed from an assessment of a variety of PPE types, it has been more recently suggested that for each kg of additional weight metabolic rate increases by 2.7% (Dorman & Havenith 2009). Despite this, weight alone is not the only cause of increased metabolic rate, with differences in material, number of layers and BA harness design also having an impact (Dorman & Havenith 2009; Bakri et al. 2012). This is evidenced by Taylor et al. (2012) who noted that whilst the heaviest item worn by FF is their BA, during walking both the BA (11.30 kg) and protective clothing (without boots and helmet) (4.72 kg) contributed 9% to the metabolic rate increase.

Overall, in a wear situation the evaporative heat loss required to maintain a thermal steady state cannot be achieved, as a consequence of the PPE, BA, physical activity and environmental conditions (Montain et al. 1994; Cheung et al. 2000). This is referred to as an uncompensable heat stress situation and results in the body continually storing heat, causing a rise in T_c (Cheung et al. 2000). Increases in T_c following live fire exposures range from 0.6 - 3.2°C, with average maximum temperatures reaching 40.1°C, as reported in

Table 2.1. Previous monitoring of FF in a high rise fire scenario has resulted in 37.5% of exercises being terminated due to T_c exceeding the safety limit of 39.5ºC, with a further 40% terminated by FF or safety officers due to concern for safety (Optimal Performance Limited 2004). Instructors monitored during two live house fire exercises, have displayed an average HR of 131 ± 18 b.min^{-1} and 121 ± 20 b.min^{-1}, with maximum HR obtained equating to 87 ± 5% and 77 ± 11% of predicted HR maximum (Eglin & Tipton 2005). This suggests that although FSI are not usually operating in emergency situations, fire exposures result in high levels of physiological strain, which FSI experience on a regular basis.

2.3.1 Activity Termination in the Heat

Activity may be terminated early by an individual if they reach a critical T_c (González-Alonso et al. 1999; Walters et al. 2000). González-Alonso et al (1999) stated that regardless of initial T_c (35.9 ± 0.2ºC, 37.4 ± 0.1ºC or 38.2 ± 0.1ºC) all cycling exercises in 40°C, 19% RH were terminated at 40.1 – 40.2ºC, with those with a higher rate of heat storage (0.10ºC.min^{-1}) terminating exercise 25 min before those at a lower rate (0.05ºC.min^{-1}). It is suggested that the critical T_c may decrease cerebral blood flow and also reduce higher frequency brain waves, representing a reduced cortical activation and a reduction in the central drive to exercising muscles (Nielsen & Nybo 2003; Cheuvront et al. 2010) Individuals with only a moderate level of fitness ($\dot{V}O_2$ maximum 40 – 50 mL.kg^{-1}.min^{-1} or 53 mL.kg lean body mass^{-1}.min^{-1}) may have a lower critical temperature (~39 ºC) (Cheung & McLellan 1998; Selkirk & Mclellan 2001) due to a reduction in thermal tolerance and differences in the efficiency of blood flow distribution (Cheung & Sleivert 2004). As the minimum acceptable $\dot{V}O_2$ max for operational duties is set at 42.3 ml.kg^{-1}.min^{-1} (Siddall et al. 2016), FSI should fit within the moderate fitness range. However, distance runners have been observed to experience $T_c \geq 40$ ºC for extended time periods (Byrne et al. 2006). This suggests that during self-paced exercise individuals may vary their intensity to prevent a $T_c > 40$ ºC until exercise finish is anticipated, whereby intensity and consequently T_c, is increased (Byrne et al. 2006). Those using only set intensity activities to identify the limiting factor to exercise in the heat, may have missed this anticipatory response (Marino 2004).

The anticipatory response may be controlled via the central nervous system, with a central fatigue governor in the brain controlling exercise pacing and cessation in the heat (Noakes 2007). During exercise the subconscious brain uses feed forward and feedback control mechanisms to modulate the number of active motor units. Temperature and heat accumulation form part of the afferent sensory feedback which is processed by the central nervous system (Noakes 2011), although numerous factors can act on the central nervous system that can be modified to alter performance, as displayed in Figure 2.2. Based on the information the central governor receives power output is regulated to maintain homeostasis and protect the body, preventing a catastrophic physiological failure (Noakes et al. 2005; Swart et al. 2009; Noakes 2011). Consequently, individuals decrease their power output to enable exercise to be paced to maintain a T_c below dangerous hyperthermia levels for as long as possible (Marino 2004; Tucker 2009; Tucker & Noakes 2009). FSI typically complete self-paced low intensity tasks, such as setting fires and ensuring the safety of trainee FF (Eglin et al. 2004), and consequently it is likely that an anticipatory response occurs.

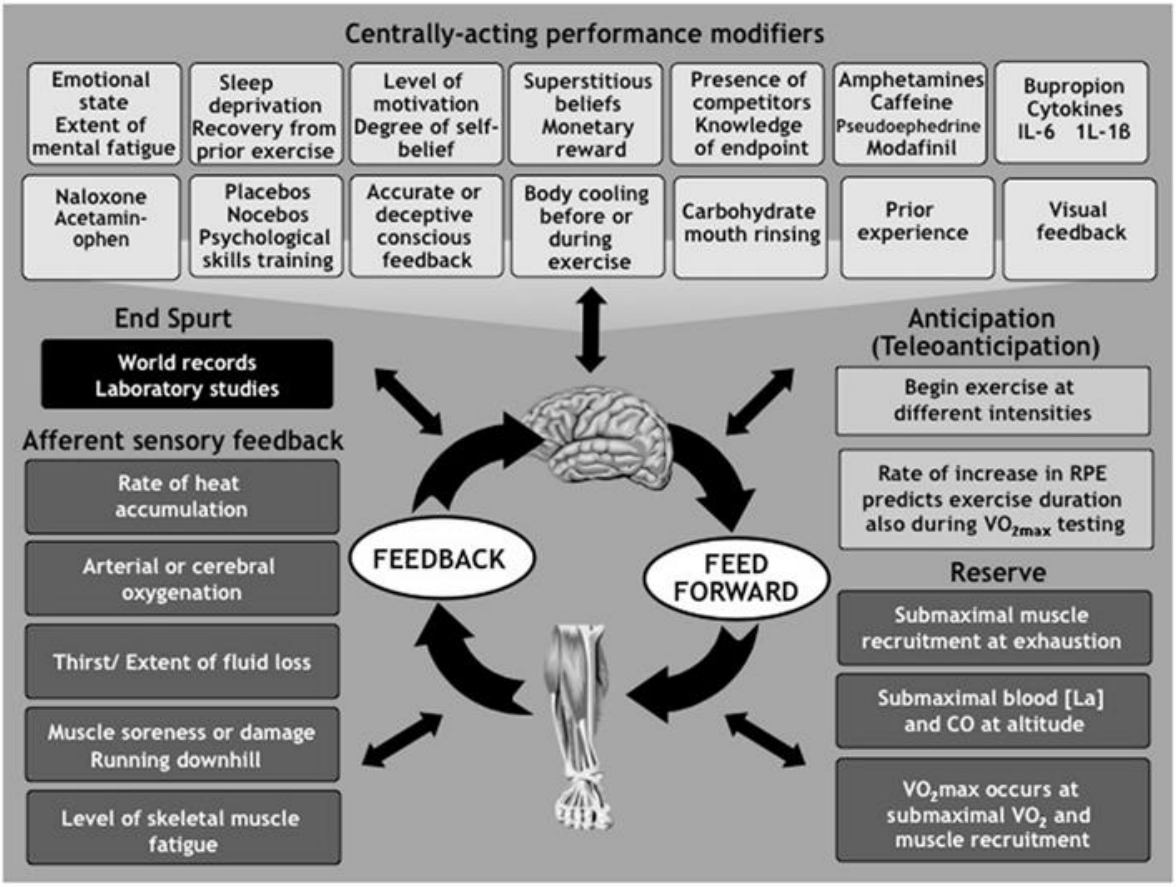

Figure 2.2 *Factors that play a part within the central governor model, including those that act on the central nervous system that can be modified to alter performance (Noakes, 2011a).*

However, no study has yet been able to demonstrate a critical T_c effect independent of other contributor factors such as skin temperature (T_{skin}) and cardiovascular strain (Cheuvront et al. 2010). Ely et al. (2009) note that when a critical T_c of 40ºC is reached there is no degradation in 8 km running velocity if T_{skin} is modest (30ºC), whilst a 6.7% decrement in speed occurs when skin temperature is 34ºC. A narrower T_{skin} to T_c gradient results in greater skin blood flow requirements, consequently elevating cardiovascular strain and potentially causing perfusion issues as skin, muscles and brain compete for blood flow (Cheuvront et al. 2010; Sawka et al. 2012). The displacement of blood to the periphery and consequential reduction in cardiac output can also result in reductions in $\dot{V}O_2$ max and therefore relative exercise intensity increases (Cheuvront et al. 2010). Overall, it is likely that elevated T_c alone is not the cause of exercise termination, but instead a multidimensional model including T_{skin}, blood flow, cardiovascular strain and the central nervous system should be considered (Nybo et al. 2014).

During an uncompensable heat stress situation, caused by wearing PPE, FF time to exercise termination, whilst walking on a treadmill, was significantly reduced in 35^0C compared to 25^0C, at different exercise intensities: very light 86.8 ± 5.1 min vs. 196.1 ± 12.9 min, light 67.3 ± 3.0 min vs. 134.0 ± 9.3 min, respectively, $p < 0.05$ (Selkirk & McLellan 2004). Of 111 trials, in 63% of terminations a T_c of 39.0 ºC was the leading factor, similar to the critical temperature for moderately trained individuals (FF $\dot{V}O_2$max: 51.3 ± 1.0 mL.kg^{-1}.min^{-1}). The remaining terminations were due to HR exceeding 95% of $HR_{peak,}$ the participant

feeling nauseous or reaching the 4 hr time limit. It is likely that due to the environmental temperatures and PPE that the T_{skin} to T_c gradient was also small and therefore may have contributed to the high cardiovascular strain, however Selkirk & McLellan (2004) do not provide T_{skin} values. The maximum environmental temperature used was lower than that experienced by FSI and therefore may underestimate the effect that heat has on instructors' T_c and task completion. Despite this, the rise in T_c and cardiovascular strain generated by exercising in an uncompensable heat stress situation, lead to exercise termination amongst FF.

Table 2.1 *Summary of studies reporting mean T_c and HR during fire-fighting tasks. * denotes ambient temperature is estimated from (Smith et al. 2001a) and Smith & Petruzzello (1998). Table adapted from Eglin (2007).*

Task	Participants	Duration (min x rep)	Ambient Temperature (°C)	Start T_c (°C)	Max/end T_c (°C)	Change in T_c (°C)	Measurement	HR Max ($b.min^{-1}$)	Reference
Fire-fighting activities	8 M ff	32	___	37.7	38.3	0.6	T_{re}	143	Romet & Frim (1987)
Fire-fighting activities	9 M ff	25 - 41	40 - 125	37.3	39.2	1.9	T_{re}	186	Bennett et al. (1995)
Ceiling haul	16 M ff	16	90	36.7	39.8	3.1	IR Tymp	176	Smith et al. (1997)
Fire-fighting activities	15 M ff	16	77 - 93	36.9	40.1	3.2	IR Tymp	139	Smith et al. (1996)
Fire-fighting activities	10 M ff	3 x 6	54 - 79	35.8	37.3	1.5	IR Tymp	194	Smith & Petruzzello (1998)
Fire-fighting activities	75 M fft	94	5 - 45	37.6	39	1.4	T_{re}	150-185	Ilmarinen & Koivistoinen (1999)
Fire-fighting activities	7 M tff	3 x 7	47 - 61	36.1	36.9	0.8	IR Tymp	189	Smith et al. (2001a)
Fire-fighting activities	11 M ff	3 x 5.5 - 6	47 - 79*	36.7	38.6	1.9	T_{re}	187	Smith et al. (2001b)
Wildland fire-fighting	179 M ff	36 - 217	19 - 35	___	38.7	___	T_{re}	152	Budd (2001)
Fire-fighting training	17 M ff	60	100 - 190	___	___	0.6 - 1.0	GI Pill	___	Rossi (2003)
Fire-fighting activities	12 ff	15	11 - 25	37.5	38.4	0.9	T_{re}	160	Griefahn et al. (2003)
Instructing activities	13 M i	33	74	37.5	38.5	1.0	T_{re} &/or GI pill	138	Eglin et al. (2004)
Instructing activities	10M i	12 - 92	48	37.6	38.0	0.4	Tre	131	Eglin & Tipton (2005)
Instructing activities	6M i	23 - 65	39	37.6	38.1	0.5	T_{re}	121	Eglin & Tipton (2005)

Task	Participants	Duration (min x rep)	Ambient Temperature (°C)	Start T_c (°C)	Max/end T_c (°C)	Change in T_c (°C)	Measurement	HR Max ($b.min^{-1}$)	Reference
Fire-fighting activities	16 (15 M, 1 F) ff	21 - 32	27	37.2 - 37.5	38.6 - 39.1	1.4 - 1.6	GI Pill	___	Richmond et al. (2008)
Simulated fire-fighting	114 M ff	18	71 - 82	___	___	0.72	GI Pill	167	Smith et al. (2011)
Fire-fighting training	40 M ff	180 (15 - 25 x 4 - 5)	___	37.1	38.9	1.8	GI Pill	159 - 213	Fernhall et al (2012)
Fire-fighting training	25 (23 M, 2 F) ff	20	___	37.5	38.2	0.84	GI Pill	175	Colburn et al. (2011)
Fire-fighting activities	94 (88 M, 6 F) ff	12	178 - 309	37.3	38.0	0.7	T_{re}	152	Burgess et al (2012)
Fire-fighting training	9 (6 M, 3 F) ff	180 (15 - 30 x 4)	___	36.9	38.7	1.8	GI Pill	188	Horn et al. (2013)
Simulated Fire-fighting	74 M ff	20 x 2	105	37.5	38.9	1.4	GI Pill	___	Walker et al. (2014)
Simulated Fire-fighting	42 M ff	20 x 2	100	37.3	38.7	1.4	GI Pill	91% HRmax	Walker et al. (2015)
Simulated Fire-fighting	24 M ff	14	85 - 135	37.4	38.7	1.4	GI Pill	188.0	Horn et al. (2015)
Instructing activities	6 M i	37	174	37.3	38.1	0.8	T_{re}	120	Watt et al. (2016)
Fire-fighting training	40 (36 M, 4 F) ff	11 - 28	54 - 744	37.0	37.91 - 38.88	0.93 – 1.77	GI Pill	178 - 188	Horn et al (2017)

The type of tasks, participant population (M = male, F = female, ff = fire fighter, tff = trainee firefighter, I = instructor), task duration (min x repetition), ambient temperature (°C), start, end and change in core temperature (T_c), the method of core temperature measurement (T_{re} = rectal temperature, IR tymp = infrared tympanic membrane temperature, GI pill = gastrointestinal pill) and HR max are given.

2.4 Exertional Heat Illness

Whilst exercising in an uncompensable heat stress situation FSI are at risk of developing an EHI. There are numerous types of heat illness that range in severity, see Table 2.2 Characteristics of heat related illnesses, from Lipman et al. (2013). for an overview.

Table 2.2 *Characteristics of heat related illnesses, from Lipman et al. (2013).*

Condition	Definition
Hyperthermia	A rise in body temperature above the hypothalamic set point when heat-dissipating mechanisms are impaired (by clothing or insulation, drugs, or disease) or overwhelmed by external (environmental) or internal (metabolic) heat production.
Heat edema	Dependent extremity swelling caused by interstitial fluid pooling.
Heat cramps	Exercise-associated painful involuntary muscle contractions during or immediately after exercise.
Heat syncope	Transient loss of consciousness with spontaneous return to normal mentation.
Heat exhaustion	Mild-to-moderate heat-related illness caused by exposure to high environmental heat or strenuous physical exercise; signs and symptoms include intense thirst, weakness, discomfort, anxiety, dizziness, syncope; core temperature may be normal or slightly elevated to > 37°C < 40°C.
Heat stroke	Severe heat-related illness characterized by a core temperature > 40°C and central nervous system abnormalities such as altered mental status (encephalopathy), seizure, or coma resulting from passive exposure to environmental heat (classic heat stroke) or strenuous exercise (exertional heat stroke).

Between a T_c of 37ºC and 40ºC heat exhaustion can occur, with symptoms including fatigue, dizziness, heavy sweating, nausea, vomiting, a headache, fainting and clammy skin, although, crucially cognitive status is maintained (Howe & Boden 2007). During a live fire situation a FSI presenting with heat exhaustion would need to be immediately removed from the fire, as although not life threatening in themselves, symptoms such as fainting and dizziness could result in injury during a wear.

When heat exhaustion is unnoticed or untreated it can develop into heat stroke, which usually occurs when $T_c \geq 40^o$ C and is accompanied by disturbance of the central nervous system, in the form of confusion, convulsions, or coma (Bouchama & Knochel 2002). Heat stroke can progress into multi-organ system failure, with the risk of mortality increasing the longer an individual's T_c is elevated above 40.5 ºC (Casa et al. 2015). Electrical conduction abnormalities within the cardiovascular system may occur, alongside myocardial ischemia (Akhtar et al. 1993; Chen et al. 2012). Musculoskeletal damage can also occur, with

rhabodomyolysis, the destruction of muscle cells, developing (Bouchama & Knochel 2002; Bagley et al. 2007). This causes proteins and myoglobin to leak into the circulation, resulting in blocked renal tubules within the kidney and ultimately acute renal failure if not treated (Bagley et al. 2007). Liver dysfunction is also common following heat stroke and although rare, may develop into hepatic failure, which carries a poor prognosis (Trujillo et al. 2009).

The risk of mortality can be reduced with immediate medical attention and cold water immersion, reducing T_c to < 40 ºC within the first 30 min of symptom presentation is recommended (Casa et al. 2006; Adams et al. 2015). Effective cold water immersion could result in up to 100% survival rate from heat stroke, as documented following an analysis of the treatment of 274 heat stroke patients following a 7 mile performance run (De Martini et al. 2015). However, individuals may take time to recover, with weeks to months needed for a full recovery (O'Connor et al. 2010). The earlier cooling and treatment occurs, the better the prognosis in terms of recovery time (McDermott et al. 2007).

There is no data to indicate the prevalence of heat illnesses that occur at training centers, as internal reports are only produced when hospitalization or fatalities occur. There is also no record of heat illness prevalence amongst FF in the UK. In the US heat illness is combined with other causes of fatalities into an overexertion/stress category, which is the leading cause of death amongst FF each year (Fahy et al. 2015; Fahy et al. 2017). However, this category also includes fatalities caused by cardiac events and therefore it is unclear what contribution is based on heat illnesses. A recent survey conducted in the US indicated that 22 out of 34 fire departments had at least one case of heat illness in the previous year, with 7 reporting the need for patient hospitalization and 1 department reporting a fatality (Bach et al. 2018). Within the US military 5,246 soldiers were hospitalized from heat illness between 1980 and 2002, with 37 deaths, equivalent to 0.3 per 100,000 soldiers per year. Although hospitalization rates over the period studied reduced, the number of heat stroke incidents increased eightfold (Carter et al. 2005). This indicates that those exercising in protective clothing are at risk of heat illness. Reducing the T_c achieved during live fire exposures could minimise this risk.

2.4.1 Risk Factors

An individual's risk of experiencing a heat related illness can be dependent on numerous personal characteristics. These characteristics may be either modifiable or non-modifiable risk factors. It is well established that aerobic fitness (Aoyagi et al. 1997), body composition (Selkirk & Mclellan 2001), age (Kenny et al. 2010), sex (Marsh & Jenkins 2002; Gagnon et al. 2013), burnt/tattooed skin (Ganio et al. 2015; Luetkemeier et al. 2017), medication (Hajat et al. 2010), geographical residence (Sawka et al. 2011), heat acclimation status (Horowitz 2014) and hydration status (Sawka et al. 2001; Casa et al. 2010) can all impact the occurrence of EHI. Table 2.3 provides an outline of risk factors.

Table 2.3 *Risk factors for exertional heat illnesses, adapted from (Coris et al. 2004; Moore et al. 2016).*

RISK FACTORS	
Non-modifiable	Modifiable
Infection	Low level of aerobic fitness
Chronic disease states	Obesity/high BMI
Older age (> 40 yrs)	Sleep deprivation
Medications (antihistamines, β-blockers)	Dehydration
Sex	Inadequate heat acclimatisation
Burnt/tattooed skin	Alcohol consumption
Sweat gland dysfunction	
Prior heat illness	

To reduce the risk of EHI modifiable risk factors should be improved. Currently, Fire and Rescue Service personnel are required to have a high level of aerobic fitness (> 42 $mL.kg^{-1}.min^{-1}$) and therefore policy and regular fitness testing is in place to mitigate this risk factor. Having a greater level of aerobic fitness results in presentation of a reduced resting T_c and an elevated sweat rate (SR), both of which are advantageous to limit the occurrence of high T_c (Cheung et al. 2000; Lisman et al. 2014). Consideration should also be given to body composition, as adipose tissue has a lower heat capacity (2.51 $J.g^{-1}.^oC^{-1}$) than lean tissue (3.65 $J.g^{-1}.^oC^{-1}$) and consequently those with a greater fat mass will experience a faster rate of increase in core temperature (Selkirk & Mclellan 2001).

2.4.1.1 Heat Tolerance

Heat tolerance is also a modifiable risk factor. Frequent exposure to increased temperatures can lead to heat acclimation, whereby individuals have a lower HR, reduced rise in T_c, increased SR and improved perceptual comfort when exposed to similar hot environments (Jay & Kenny 2010; Burk et al. 2012; Chalmers et al. 2014). The physiological adaptions caused by heat acclimation increase an individuals' heat tolerance and reduce the risk of EHI occurring (McDermott et al. 2007; Cleary 2007; Lipman et al. 2013). Although it should be noted that heat tolerance does not protect individuals in all circumstances, for example during prolonged high intensity exercise in extreme heat when hypohydrated (Druyan et al. 2013). In addition, the increased SR may result in greater dehydration, therefore compromising achievement of a euhydrated state (Eglin 2007). Heat tolerance is not currently assessed at any stage in a FF or FSI career (Optimal Performance Limited 2004). It is plausible that FSI who wear frequently may be better adapted to hot environments and therefore experience lower levels of physiological and perceptual strain.

However, Wright et al. (2013) reported no difference in change in T_c or HR between experienced FF and non-FF during a 4x15 min cycle at 45% of $\dot{V}O_{2peak}$ in 20% or 60% relative humidity, with a temperature of 35°C, $p > 0.05$. This similar tolerance to heat exhibited across the groups may be because FF exposure to heat stress is not frequent enough for heat acclimation to occur. Wright et al. (2013) reported that the FF responded to 158 ± 16 emergency calls per year, although this would have included a variety of emergency situations, not just fires. Ashley et al. (2015) suggests that individuals working in hot environments (50ºC,

20% RH) may take 6.1 ± 1.4 days to acclimate and that the longer time spent away from heat the more HR and T_c increased in response to tolerance tests. Following two weeks of no heat exposure, it was recommended that individuals complete 4 days of heat acclimation before beginning any work specific tasks. FSI are limited in the amount of time they have before a course begins and therefore an allocated re-acclimation time is likely to be unachievable. Furthermore, it is unclear how much time FSI receive away from wears, as the time varies between brigades. The decay of heat acclimation may also be affected by an individual's cardiovascular fitness (Pandolf 1998; Garrett et al. 2011). Therefore FSI with greater levels of cardiovascular fitness may retain adaptations for a longer period of time (Pandolf 1998). Overall, heat acclimation may be beneficial to FSI, as it may decrease the amount of physiological and perceptual strain they experience and may reduce the risk of EHI. Whether FSI display an acclimated state to heat has not been previously documented.

The measurement of heat tolerance itself is also an issue of contention. Current gold standard practice within military populations is to use the Israeli Defence Force test, which includes a 120 min walk at 5 $km.hr^{-1}$ with a 2% gradient in 40ºC and 40%RH whilst wearing shorts and t-shirt (Moran et al. 2004; Moran et al. 2007). However, recent research indicates the importance of controlling for H_{prod} to allow for unbiased comparisons of individuals with differing aerobic fitness levels and body composition (Jay et al. 2011; Cramer & Jay 2014; Cramer et al. 2015). Furthermore, this protocol has clear differences in clothing worn and environmental temperatures experienced when compared to wears completed by FSI. Consequently, the development of an ecologically valid heat tolerance test that controls for H_{prod} is first needed before assessment of FSI acclimation status can be conducted.

2.4.2 Acute Interventions

There are numerous acute interventions that might be utilized to reduce the physiological strain experienced with live fire exposure and minimise the risk of EHI. These include maintaining a euhydrated state, clothing choice beneath PPE, pre-cooling and post-cooling (McLellan & Selkirk 2006; Eglin 2007; Cheung et al. 2010). Maintaining a euhydrated state is critical to maintain SR and blood volume to enable heat dissipation. A hypohydrated state can lead to increases in physiological strain, with 0.12 - 0.25ºC increase in T_c and 3 - 5 $b.min^{-1}$ increase in HR for every 1% of body mass lost through sweating (Sawka et al. 2001; Casa et al. 2010). The reduction in HR is a consequence of reduced blood volume causing a decrease in stroke volume and ultimately cardiac output (González-Alonso et al. 2008). Dehydration can also cause decrements in cognitive performance, with a recent meta-analysis indicting tasks requiring attention, executive function and motor coordination more likely to degrade than lower-level cognitive tasks, such as reaction time or memory (Wittbrodt & Millard-Stafford 2018). From a firefighting perspective dehydration may therefore effect an individual's ability to correctly evaluate a situation and decide upon the most appropriate response. Furthermore, the level of cognitive impairment is related to the magnitude of dehydration, with those exhibiting > 2% body mass loss having larger decrements in cognitive performance (Wittbrodt & Millard-Stafford 2018). Rehydration following a wear is also of particular importance to FSI, so that they do not begin subsequent exposures in a hypohydrated state. FSI have previously been noted to have a SR of 0.86 - 1.59 $L.hr^{-1}$ during a wear (Eglin et al. 2004; Watt et al. 2016). Whilst this commonly equates to < 1% loss

in body mass, FSI have on occasion be noted to exhibit 3.1% loss in body mass, coinciding with dizziness and nausea (Eglin et al. 2004). Guidance states that individuals should consume 1 - 1.5 times the amount of sweat lost (Coyle 2004; Sawka et al. 2007; Chief Fire Officers' Association 2015). Replacement of two-thirds or total amount of fluid loss has been reported to enable exposure times on a treadmill walk in PPE to increase by 20% before a T_{re} of 39.5ºC is reached (McLellan & Selkirk 2006). It is also suggested that electrolytes be added to the fluid consumed to replace those lost through sweating and aid fluid retention, with 20 - 40mmol.L^{-1} of sodium recommended (Coyle 2004; Sawka et al. 2007). The use of a colour chart to monitor hydration status is also suggested within the Fire and Rescue Service (Chief Fire Officers' Association 2015). Informative guidance on hydration therefore currently exists for FF and FSI.

The use of shorts and a T-shirt beneath PPE instead of long trousers and a shirt has been suggested to help reduce heat stress (McLellan & Selkirk 2006). The switch from trousers to shorts within the New York City Fire Department resulted in no change in the incidence of burn injuries and a reduction of medical leave from heat exhaustion (Prezant et al. 2000). In exposure times of > 1hr, T_{re} is reduced when wearing shorts and during light activities (4.5 km.hr^{-1} walk) exposure time was extended by 10 - 15%. Watkins & Richardson (2017) further support this work, indicating that wearing a wicking base layer or shorts reduces the inflammatory response to a 45 min simulated fire exposure, with T_{re} also minimized by 0.27 - 0.31ºC compared to wearing a full length boiler suit. It is therefore clear that clothing beneath the PPE needs careful consideration, with alternative wicking layers or shorts & t-shirt recommended options instead of long trousers & shirts. Although it is important that changes to undergarments do not compromise the safety of individuals and therefore it is essential that PPE is in good condition.

Cooling interventions used pre, during (per cooling), or post exercise in the heat are common within sporting populations to reduce physiological strain and improve performance, with many reviews providing details of effective methods (Ross et al. 2013; Tyler et al. 2013; Bongers et al. 2014; Jones et al. 2012). Methods commonly fall into one of two categories, external, the application of cooling modalities to the skin, or internal, the ingestion of a cooling substance, although some methods use a combination of the two (Ross et al. 2013). Pre-cooling aims to lower initial T_c and increase the body's heat storage capacity, thus increasing the time taken to reach a critical temperature and prolonging exercise duration (Ross et al. 2013). Percooling is often used within breaks in play or during continuous exercise to limit the rise in T_c. A meta-analysis of nine studies identified a 10% improvement in cycling and running performance with percooling in ambient temperatures > 30ºC (Bongers et al. 2014). Both pre and per cooling therefore aim to reduce the incidence of EHI. Post cooling is used to reduce elevated T_c after exercise and is especially important in the presence of heat illness (McDermott et al. 2009).

From an occupational perspective percooling is not feasible during a wear, due to the nature of the tasks being completed and the PPE and BA preventing application of either external or internal methods. Post cooling has been widely studied within military and fire service populations. Cold water immersion (15ºC to the umbilicus) and ice slurry (7 g.kgBM^{-1}) ingestion have been recommended as the most effective cooling modalities, having been noted to reduce T_c by 0.093ºC.min^{-1} and 0.092ºC.min^{-1}, respectively, following a simulated 2x20 min fire search and rescue task (Walker et al. 2014). Walker et al. (2014) also

reported that both methods successfully reduced T_c to baseline measurements within 15 min. Due to the logistical difficulties of cold water immersion, ice slurry use is recommended as a post cooling intervention following a wear (Walker et al. 2014). Forearm post cooling has also previously been advocated within fire service communities (Chief Fire Officers' Association 2015), however a recent review of forearm immersion studies suggests it demonstrates inadequate cooling rates, with all studies reporting cooling of < 0.078ºC indicating it would take > 40 min to cool an individual with hyperthermia (42.2ºC) to a safer level (38.9ºC) (Brearley & Walker 2015). There is little research into the use of pre-cooling in an occupational situation. A review by Brearley (2012) suggests that ice slurry consumption prior to exposure may be beneficial for military and fire service personnel, as the potential heat storage conferred from crushed ice (489 $kJ.L^{-1}$) is greater than that of cold water (155 $kJ.L^{-1}$), whilst being easy to administer due to the minimal equipment, labour and cost involved. A study by Pryor et al. (2015) is the only investigation to assess the use of ice slurry in an occupational setting, identifying that pre-cooling with ice slurry reduced T_{re} by ~0.6ºC prior to completing a treadmill walk in 38.8 ± 1.2ºC in wildland firefighting equipment, with T_{re} remaining reduced for 30 min. However, the dosage of 7.5 $g.kgBM^{-1}$ implemented by Pryor et al. (2015) is unlikely to be logistically possible for FSI due to the time consuming nature of weighing both individuals and ice. In addition, precise ice measurements would require facilities in clean areas away from possible contamination from the training ground. Consequently, the use of an alternative, easy to administer, dosage requires investigation. The usefulness of other cooling modalities for pre-cooling Fire Service personnel has not been assessed and despite pre-cooling being suggested by the Chief Fire Officers' Association (2015) no clear guidance is given on method, timing, or dosage.

2.5 Cardiovascular Events

FF are 12 to 136 times more likely to die of coronary heart disease during a live fire suppression than during any other duty (Kales et al. 2007). Cardiac death accounted for 42% of FF deaths in America in 2012 and rose to 56% of deaths in 2014, it is the main cause of fatalities in any given year (Fahy et al. 2013; Fahy et al. 2015). However, Fahy et al (2013) noted that age and obesity may be contributory factors, with cardiac death rarely occurring in FF under the age of 35 yrs, unless they had underlying medical conditions. Despite this, the high incidence of myocardial infarction makes cardiac strain experienced by FF a major concern (Cheung et al. 2010; Yang et al. 2013). In addition, it is likely FSI are over the age of 35 yrs, due to the career experience needed to become an instructor. Individual characteristics such as body composition, age, cholesterol levels and blood pressure can interplay to predispose an individual to be at a greater risk of a cardiovascular events (Smith et al. 2016). However, live fire exposures themselves may trigger cardiovascular events, see Figure 2.3 for the association between fire exposure, cardiovascular response and sudden cardiac events, which will be discussed in the following section.

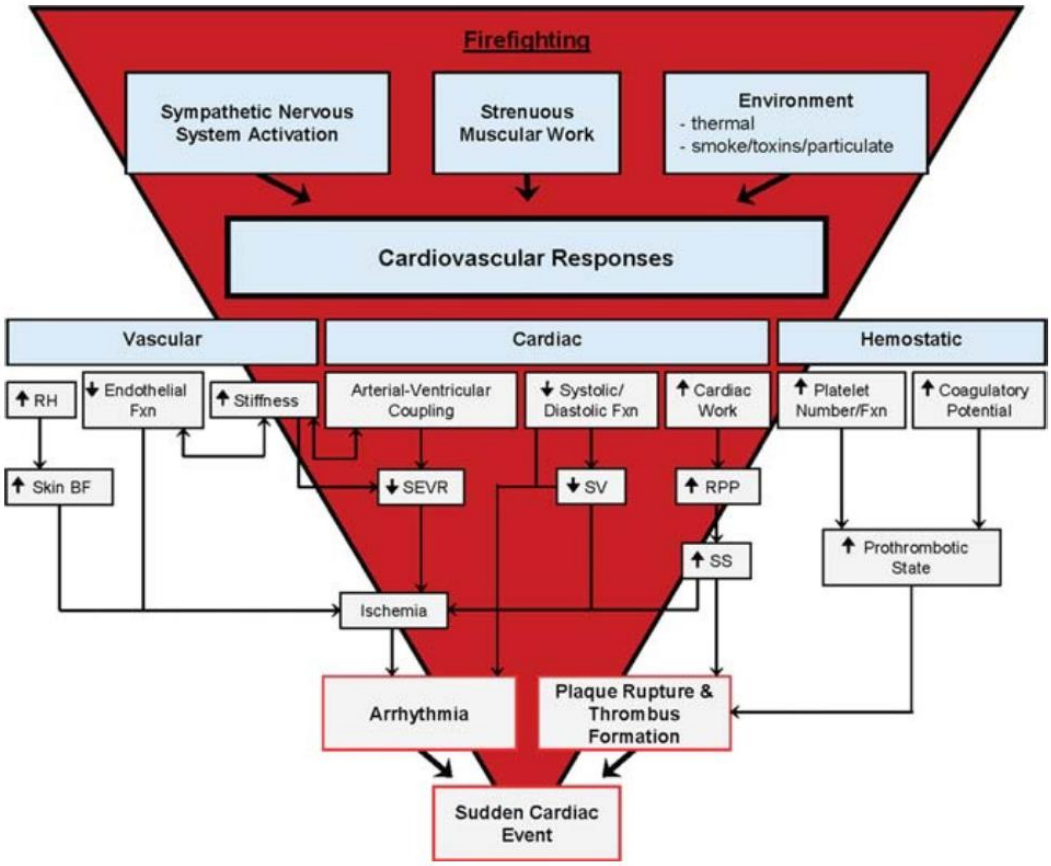

Figure 2.3 *Theoretical interplay between cardiac, vascular and hemostatic responses to firefighting in association with cardiac events, from Smith et al (2016). RH = reactive hyperemia, BF = blood flow, Fxn = function, SEVR = subendocardial viability ration, SV = stroke volume, RPP = rate pressure product, SS = shear stress.*

2.5.1 Myocardial Function

There are many possible causes of cardiovascular events as a consequence of firefighting. The prolonged heat exposure combined with physical activity may reduce myocardial function transiently, with both reduced systolic and diastolic function, which may arise from mitochondrial damage and reduced contractile function (Fernhall et al. 2012). Three hours of live fire training exercise, including four to five fire exposures lasting 15 - 25 min, can result in a 13% reduction in stroke volume and alterations in left ventricular function, with a reduced left ventricular shortening fraction from 33.0% to 28.6%. (Fernhall et al. 2012). Fractional shortening represents the change in left ventricular end diastolic, in relation to systolic, dimensions, with a value less than 29% indicating a mild decrease in function (Oechslin et al. 2000). This duration of firefighting has also been reported to induce central artery stiffness, with an increase in aortic pulse wave velocity from 6.3 ± 0.1 $m.s^{-1}$ to 6.5 ± 0.1 $m.s^{-1}$, p = 0.038 (Fahs et al. 2011). Aortic pulse wave velocity is the velocity at which pressure waves generated by a heart contraction propagates through the circulatory system, with a higher velocity indicating lower vessel distensibility and greater rigidity of the vascular wall (Pereira et al. 2015). Aortic stiffness has been suggested to be a predictor of cardiovascular events, with an increase in pulse wave velocity of 3.4 $m.s^{-1}$ resulting in a 16 - 20% increase risk of an event

(Hansen et al. 2006). However, although the increase noted by Fahs et al. (2015) was statistically significant, it is unclear what the clinical significance of an acute rise of 0.2 $m.s^{-1}$ is. Furthermore, the post firefighting pulse wave velocity (6.5 ± 0.1 $m.s^{-1}$) is within the lowest risk classification of < 8.9 $m.s^{-1}$, at which the hazard ratio is < 1.0 (Hansen et al. 2006).

Myocardial dysfunction may also be a consequence of myocardial oxygen demand exceeding oxygen supply (Smith et al. 2016). Elevated heart rate, more frequent ventricular contractions and decrease in time spent in diastole, reduces the time available for coronary perfusion (Lefferts et al. 2015). Moreover, the additional demand placed on the cardiovascular system due to the environmental temperatures and wearing of PPE increases the myocardial oxygen demand (Lefferts et al. 2015). The ratio between the systolic and diastolic portions of aortic pressure waveforms gives an indication of myocardial work/perfusion matching and is referred to as subendocardial viability ratio (SEVR) (Namasivayam et al. 2011; Lefferts et al. 2015). Following exercise in PPE in the heat SEVR has been noted to reduce by 27% (Lefferts et al. 2015), whilst 18 min fire exposure completed by FF has been reported to reduce SEVR by 30 - 35%. SEVR returned to baseline levels following 80 min of recovery (Horn et al. 2011) . Fire exposures may therefore lead to ischemia which can continue post initial exposure cessation. Ischemia can lead to arrhythmias and contribute to cardiovascular events (Smith et al 2016).

Myocardial function may also be reduced due to damage to the myocardium. This is evident through an increase in cardiac troponin I (1.5 $ng.L^{-1}$ to 3.0 $ng.L^{-1}$) post live fire exposure (Hunter et al. 2017). Cardiac troponins act as markers of myocardial damage and are structural regulatory proteins that control the interaction of actin and myosin within the myocardium (Sharma et al. 2004; Reichlin et al. 2009). The troponin complex in a myocardial sarcomeric unit consists of three troponin subunits, cardiac troponin T (cTnT) which connects the complex to tropomyosin of the thin filament, troponin C which binds calcium ions and troponin I which inhibits the hydrolysis of adenosine triphosphate (Shave et al. 2010). More than 90% of cardiac troponin is bound to tropomyosin, with the remaining amount unbound in the cytosol (Shave et al. 2010). An increase in cardiac troponin (≥ 14 $ng.L^{-1}$), alongside symptoms of ischemia, electrocardiogram changes indicative of ischemia, or imaging evidence of myocardium or heart wall motion abnormalities, are used to diagnose individuals with acute myocardial infarction (Thygesen et al. 2007; Thygesen et al. 2012). Although it has been suggested that an increase of cTnT of 7 $ng.L^{-1}$ or 8 $ng.L^{-1}$ from first presentation of symptoms to 2 hr post should also be used for diagnosis (Reichlin et al. 2011; Scharnhorst et al. 2012). Cardiac troponin prevalence in the general population is usually low and often below the detectable limits, with detectable levels associated with structural heart disease and cardiovascular events. Exhibiting an increased cTnT (≥ 14 $ng.L^{-1}$) at rest is associated with an increase in all-cause mortality (hazard ratio = 2.8) and cardiovascular mortality (hazard ratio = 1.7) (de Lemos et al. 2010).

Increased cTnT has been reported following endurance exercise, with higher intensities of shorter durations typically eliciting greatest increases (Shave et al. 2010). Following the Boston marathon, 68% of 482 runners had a rise in troponin, with 55 participants exhibting large increases of cardiac troponin, with 54 having increased cTnT (≥ 7.5 $ng.L^{-1}$) and 18 increased troponin I (≥ 5 $ng.L^{-1}$) (Fortescue et al. 2007). In

addition, a recent study by Richardson et al. (2018) also noted a 68.92 ± 30.16 ng.L^{-1} increase in cTnT with marathon performance, with all runners having post marathon values > 14 ng.L^{-1}; increases in cTnT were also positively correlated with relative exercise intensity. Elevated cTnT post marathon have been linked to reductions in right ventricular function, demonstrated by reduced endocardial velocities ($r = -0.70$, $p < 0.001$) (Neilan et al. 2006). However, no myocardium necrosis was found post a marathon in 14 participants, despite increases in cTnT (Mousavi et al. 2009). Opposing evidence has also been presented by George et al. (2009) who noted no correlation between troponin increase post exercise and ventricular dysfunction. The lack of relationship between acute troponin increases and ventricular dysfunction may indicate that the release of cTnT following exercise is a benign process, with cTnT originating from the cytosol being released with no release from the myocardium (Shave et al. 2010). Alternatively, it has been hypothesized that repeated exposure to endurance exercise may result in a cumulative effect, with frequent strain on the ventricles leading to myocardial necrosis, causing ventricular dysfunction and arrhythmias, which could be fatal (Harper 2010). Hunter et al (2007) have been the only group to study cardiac troponin in FF. To the authors knowledge there has been no assessment of FSI cTnT, either at rest or following a wear and it is therefore unknown if FSI experience an elevated cTnT and if this persists at baseline on subsequent days.

2.5.2 Atherosclerosis

Firefighting may also increase the risk of cardiovascular events via atherosclerosis, which is the formation of plaques that result in luminal narrowing or precipitating thrombi that obstruct blood flow (Bentzon et al. 2014). Plaques form from lipoproteins being engulfed by macrophages beneath the endothelium of the blood vessel and the eventual growth of smooth muscle cells covering them (Hansson 2005). Endothelium dysfunction and increased cell adhesion molecules also contribute to plaque formation (Falk 2006). A gap in the fibrous cap that separates the atheromatous lipid core from the blood flow instigates a plaque rupture, exposing the core of the plaque and inducing a thrombus (Hansson 2005; Falk 2006). Consequently, a thrombus can prevent blood flow and result in a myocardial infarction (Hansson 2005).

The occurrence of atherosclerosis may be related to platelet concentration. Platelets are small blood cell fragments that are involved in the clotting process (Huo & Ley 2004). A normal platelet range is defined as between 150 x10^9.L^{-1} to 400 x10^9.L^{-1} (Sloan et al. 2015). Mortality from a cardiovascular event has been found to be 2.5 times higher in those in an upper quartile for platelet count (mean: 342 x10^9.L^{-1}, range: 264 - 705 x10^9.L^{-1}, n: 121) when monitoring 487 healthy middle aged men (Thaulow et al. 1991). In addition, baseline platelet counts amongst patients presenting with myocardial infarction positively correlate with cardiac mortality at 1 yr follow up, with a high platelet count (≥ 234 x10^9.L^{-1}) predicting 1 yr mortality or repeated infarction (hazard ratio [HR] = 1.04) (Nikolsky et al. 2007). A live fire training exercise of 20 min has been noted to increase ex vivo thrombus formation by 73% in the low shear chamber and 66% in the high shear chamber (Hunter et al. 2017). This is likely due to systemic inflammation, sympathetic activation and dehydration leading to platelet stimulation and activation of coagulation pathways. In addition, hypovolemia causes increased haematocrit, increasing blood viscosity (Smith et al. 2016). This is evident in the 7% increase in platelet-monocyte aggregation and increase in platelet number, 234 ± 10 to 305 ± 15

$x10^9.L^{-1}$ (Hunter et al. 2017). These findings are supported by Smith et al. (2011) and Walker et al. (2015) who also report increased platelet counts (+ 64.6 $x10^9.L^{-1}$ and + 11.7 ± 19.7 $x10^9.L^{-1}$, respectively) post fire exposure. This increase has been documented to remain present at 24 hrs post wear (+ 15.9 ± 19.7 $x10^9.L^{-1}$) (Walker et al., 2015). These values post wear were all collected from FF, who conduct a different role during exposure than FSI. It is therefore unknown if FSI have the same response and if the frequency of their exposures impacts platelet count.

A recent review however postulates that the role of platelets in the pathogenesis of coronary heart disease is due mainly to their function, rather than their absolute number (Madjid & Fatemi 2013). Platelet size, estimated in individuals by mean platelet volume (MPV), may give a better indication of function and has been linked to an increased risk of cardiovascular events, with positive associations having been noted between MPV and clotting, arterial stiffness and cardiovascular disease (Sloan et al. 2015). A meta-analysis of 16 studies indicates that individuals who have experienced an acute myocardial infarction have a greater MPV (9.24 ± 0.84 fL) than those without myocardial infarction (8.48 ± 0.71 fL) (Chu et al. 2010). Patients with acute myocardial infarction who have an increased MPV are at a higher risk of death, with an odds ratio (OR) of 1.65. This OR rises to 2.01 if a high MPV is classified as ≥ 10.3 fL (Chu et al. 2010). MPV ≥ 11.01 fL also increases the risk of ischemic heart disease by a hazard ratio of 1.80 (Slavka et al. 2011). There have been no previous measures of MPV in FSI either at rest or post wear and therefore it is unknown if this key indicator of cardiovascular risk is impacted by their occupation.

In summary, FF have been reported to be at an increased risk of a cardiovascular event, with live fire exposure potentially triggering an event. The high level of cardiovascular strain from the physically demanding tasks and environmental conditions experienced, combined with psychologically stressful situations, could result in atherosclerosis developing into plaque ruptures. An elevated myocardial demand for oxygen that exceeds myocardial supply could also contribute, causing ischemia and consequently myocardial damage and arrhythmias. It is unknown what impact the FSI occupation may have on the risk of a cardiovascular event occurring. The number of fires experienced by instructors compared in FF is much greater and potentially puts instructors at a greater risk of a cardiac event, however this has not been previously assessed. In addition, whilst the risk of an event immediately prior to a heat exposure has been identified in FF, it has yet to be established if there is a long term increased risk of a cardiac event and if this may be linked to the number of fires experienced.

2.6 Overtraining

Chronic exposure to live fire situations may also result in a response similar to overtraining, with FSI anecdotally reporting feeling fatigued, suffering from colds and mood swings. Overtraining is defined as:

> "an accumulation of training and/or non-training stress resulting in long-term decrement in performance capacity with or without related physiological and psychological signs and symptoms of overtraining in which restoration of performance capacity may take several weeks of months". (Kreider et al. 1998)

It is suggested that with continual repeated exposures to the same stressors exhaustion can occur, with resistance to the stressor dropping below normal (Halson & Jeukendrup 2004). The pathophysiology behind overtraining is still debated, however it is commonly regarded to be caused by a high volume and/or intensity of physical activity with insufficient recovery time (Smith 2003). Key symptoms of overtraining include fatigue, insomnia, frequent illnesses and mood changes, however there are a large array of symptoms, see Table 2.3, that can present in a multitude of patterns in terms of presence and severity (Smith 2003).

Table 2.4 *Symptoms of overtraining, adapted from Fry et al. (1991).*

Type	Symptom
Physical Performance	Decreased performance
	Recovery prolonged
	Decreased maximum work capacity
	Loss of coordination
	Reappearance of mistakes already corrected
	Chronic fatigue
	Insomnia with and without night sweats
	Muscle soreness or tenderness
	Hypertension
	Weight loss
	Headaches
	Nausea
	Loss of appetite
Psychological	Feelings of depression
	General apathy
	Emotional instability
	Difficult concentrating
	Changes in personality
	Fear of competition
Immunological	Increased susceptibility and severity of illness colds and allergies
	Flu like illness
	Minor scratches heal slowly
	Bacterial infections
	Decreased lymphocyte counts
	Decreased functional neutrophils
	Increased eosinophil count
Biochemical	Depressed muscle glycogen
	Elevated cortisol
	Low free testosterone
	Elevated C-reactive protein

An increased prevalence and severity of upper respiratory tract infections (URTI) have also been linked with overtraining, with the relationship between exercise and URTI suggested to follow an "S" shaped curve (Malm 2006). Moderate exercise has been reported to improve an individual's ability to resist infections by 20 - 30%, whilst chronic high load exercise reduces the body's ability to protect again infections (Matthews et al. 2002; Moreira et al. 2009). To become an international athlete however, a greater ability to fight off infection is required, so as to maintain high training loads and consequently these individuals may be at a reduced risk of URTI (Malm 2006; Moreira et al. 2009; Hellard et al. 2015). It can also be suggested that elite athletes have improved lifestyle behaviour's to alter infection risk and experience less socio-economic stress as they may receive funding support (Walsh & Oliver 2016). This "S" curve therefore indicates that those with high volumes of exercise, as with overtraining, but not competing at an international level, may be at an increased risk of an URTI. An increased odds (OR = 5.9) of experiencing an URTI post marathon has been reported compared to non-marathon runners (Nieman et al. 1990). In addition, a significant positive relationship between training load and URTI presence (r = 0.72) has been recorded in well trained cyclists monitored over a 29 week training period (Ferrari et al. 2013). Conversely, further marathon research indicated that the incidence of URTI for marathon runners has no relationship with training volume for the 6 months prior to the race (Ekblom et al. 2006). This finding is supported by Fricker et al. (2005) who also found no differences in training load and running mileage between individuals who suffered from a URTI and those who were healthy over a four month period. The occurrence of URTI amongst overtrained individuals is hard to estimate, as research within the desired population is rare due to the ethical issues of overtraining a group of athletes (Armstrong & VanHeest 2002). Of 24 swimmers, with a 36.5% increase in swim volume over a 4 week period, 10 (42%) exhibited URTI. However, only 1 of those individuals was diagnosed as overtrained based on a decrease in performance or feelings of fatigue (Mackinnon & Hooper 1996). Currently there is no specific tool recommended for diagnosing overtraining and therefore the method used varies greatly between studies and, consequently, the validity of overtraining diagnoses is unclear (Halson & Jeukendrup 2004).

The prevalence of URTI in those with high load training has been linked to a proposed period of immunosuppression following exercise, whereby the risk of an infection increases. This is referred to as the "open window", which can last 3 to 72 hrs post exercise (Pedersen & Bruunsgaard 1995; Nieman 2000). Without sufficient rest, this may develop into chronic immunosuppression as the body attempts to cope with repeated pro-inflammatory events (Smith 2000). Natural killer cells, which are a subset of lymphocytes, have been reported to be depressed in recruits following a period of training (3 weeks) and a combat course (5 days), with this reduction correlating to URTI symptoms (r = -0.67) (Gomez-Merino et al. 2005) Reduced leukocyte and lymphocyte counts have also been noted following 2 - 3 weeks of Parachute Regiment training and coincided with a peak in UTRI (Whitham et al. 2006). However the authors concluded that the altered immunological markers were more likely to be a product of the URTI than the cause. Gleeson et al. (2000) reported no association between URTI and reduced natural killer cells or increased immunoglobulins after a 12 week intensive swimming programme, which resulted in 10 of 12 participants with URTI. The association between URTI, the "open window" and overtraining is consequently unclear.

Currently, overtraining is diagnosed by first excluding other etiological possibilities, such as an eating disorder, hypothyroidism, anemia, or diabetes. Following this an individual should present with a decrease in performance despite rest, mood changes and a lack of another cause of underperformance (Halson & Jeukendrup 2004). There is no definitive biomarker that can be used to identify overtraining, although measurements of cortisol, adrenocorticotropic hormone, growth hormone, cortisol to testosterone ratio, immunoglobulins and leukocyte numbers have all been suggested as possible means to detect overtraining (Halson & Jeukendrup 2004). The difficulty in identifying a biomarker for diagnosis is partly due to the poor understanding of the pathophysiology behind the syndrome. Currently, there are numerous hypotheses as to the mechanism of overtraining, with Kreher & Schwartz (2012) and Carfagno & Hendrix (2014) providing comprehensive reviews of the topic. The only hypothesis that currently attempts to encompass the possible cause of the multitude of symptoms related to overtraining is the cytokine hypothesis.

Cytokines are soluble hormone-like proteins that are the chemical messengers between cells, they can be activated by a variety of stimuli and are important in mediating inflammatory and immune responses (Elenkov et al. 2005). The cytokine hypothesis suggests that acute tissue trauma caused by repeated movements and muscle contractions leads to an inflammatory response, that without sufficient recovery develops into systemic chronic inflammation (Smith 2000; Smith 2003). The inflammatory response due to acute trauma causes leukocytes to move towards the damaged area, with neutrophils specifically involved in the initial acute inflammation phase, lasting no more than 24hours. With sufficient recovery, this mild inflammation is resolved by anti-inflammatory cytokines and can result in adaptive changes to muscle, bone and connective tissue (Smith 2000; Hanada & Yoshimura 2002). Without recovery local chronic inflammation can develop, with persistent accumulation of leukocytes and pro-inflammatory cytokines, ultimately resulting in a systemic immunological response (Smith 2000; Hanada & Yoshimura 2002). Persistent muscle and joint soreness has been reported in overtrained athletes (Kentta et al. 2001; Margonis et al. 2007), with supporting muscle biopsies reporting muscle fibre size variations and z disc streaming, whereby the actin anchoring points at the end of the sarcomeres are disrupted, indicating muscle damage is present (Grobler et al. 2004).

Interleukin-6 (IL-6), interleukin-1beta (IL-1β) and tumour necrosis factor alpha (TNFα) are the key cytokines suggested to cause overtraining symptoms (Smith 2000). IL-1β, TNFα and IL-6 enable the peripheral immune system to communicate with the central nervous system via directly crossing the blood brain barrier or activation of afferent neurons (Banks 2005). They can stimulate "recuperation" or "sickness" behaviours, which are a group of responses including weight loss, depression, fatigue and sleep disturbances (Dantzer et al. 2008). Cytokines can signal the brain via numerous pathways, they are able to cross the blood brain barrier via cytokine receptors, stimulate afferent nerves such as the vagal nerve and enter the brain via volume diffuse at circumventricular organs (Johnson 2002; Dantzer et al. 2008). Within the brain there are cytokine receptors located in the hypothalamus and consequently cytokine binding in this area can activate the autonomic nervous system and hypothalamic-pituitary-adrenal axis (Turnbull & Rivier 1999). Subsequently, the secretion of hormones such as corticotrophin-releasing factor and adrenocorticotropic are elevated (Turnbull & Rivier 1999; Carfagno & Hendrix 2014; Dantzer 2018; Goshen et al. 2008). The hippocampus in the brain is also effected by IL-1β, with the cytokine impairing

neurogenesis (Goshen et al. 2008; Goshen & Yirmiya 2009). As a consequence of hormone stimulation and decreased neurogenesis, sickness behaviours have been noted (Goshen & Yirmiya 2009). Endotoxin induced IL-6 and TNFα elevations have been reported to positively correlate with increased anxiety, depressed mood and decreased memory performance (r=0.40 to r = 0.75) (Reichenberg et al. 2001). Moreover, in mice injected with IL-1, those with genes coding for IL-6 were 80% less active than IL-6 deficient mice, indicating that IL-6 is involved in the development of sickness behaviours (Bluthé et al. 2000). The occurrence of self-reported symptoms of overtraining, such as sleep disturbances, depressed mood and fatigue, following 8 weeks of intense rowing training have also been reported to be associated with elevated levels of IL-1β, TNFα and IL-6 (Main et al. 2010). The relationship between overtraining and cytokine levels has yet to be clearly established, however the suggested increase in cytokines with overtraining, and the similarity of sickness behaviours to overtraining symptoms, provides a theoretical basis for the cytokine theory.

The main limitation the cytokine hypothesis is the lack of studies investigating overtrained populations. Studies are rare as coaches and researchers are often unwilling to cause a state of overtraining in a group of athletes, consequently research focuses on high training loads, overreached groups, or small numbers of individuals later diagnosed with the syndrome. Overreaching is different to overtraining, as symptoms are only short-term, with performance capacity returned in only a few days or weeks, instead of several weeks or months (Halson & Jeukendrup 2004). Therefore there is little evidence to demonstrate elevated cytokines in overtrained individuals. The current studies on overtraining focus on relatively short periods of time, with 7 months being the longest reported for a group with a high volume of training (Gleeson et al. 1995). In comparison, FSI experience high levels of physiological strain repeatedly for numerous years at a time and therefore may exhibit more pronounced immunological changes than previously reported.

Military populations have previously been considered to experience overtraining, with research focusing on recruit training. Participants completing a 45 day Australian Army Common Recruit Training course have been reported to experience symptoms of overtraining including sleep disturbances, fatigue and confusion, combined with increases in inflammatory markers TNF-a and CRP, which supports the cytokine hypothesis (Booth et al. 2006). Gomez-Merino et al. (2003) also suggest soldiers undergoing a 3 week training course ending in a 5 day combat course exhibit an overtraining syndrome with increased IL-6 (+34%), sleep deprivation and fatigue. In Spanish military recruits undergoing an 8 week recruitment programme, 24% (10/42) were classified as overtrained as diagnosed by testosterone/cortisol levels (Chicharro et al. 1998).

FSI have been reported to have elevated IL-6 before a no heat exposure wash out period (17.0 ± 5.7 $pg.mL^{-1}$) and post a 4 week instruction course (11.4 ± 1.0 $pg.mL^{-1}$), in comparison to a non-wearing control group (5.1 ± 2.0 $pg.mL^{-1}$ and 5.3 ± 2.3 $pg.mL^{-1}$, respectively). A reduction in lung function was also reported, with decreased forced vital capacity and forced expiratory volume in 1 second. A reduction of 7% was also noted $\dot{V}O_{2max}$, although this was not statistically significant (Watt et al. 2016). The combination of these findings, along with anecdotal reports of ill health, indicate that FSI may be suffering from an overtraining like syndrome. However, the study by Watt et al. (2016) involved a small sample of FSI (n = 6) from one

training center, therefore the prevalence of an overtraining like syndrome amongst FSI across the UK is unknown. If symptoms of ill health are wide spread, then developing an understanding of their cause may enable policy changes to be made to better protect the welfare of FSI. It is postulated that the number of live fire exposures may be related to overtraining symptoms.

2.7 Impact of heat exposure and exercise on markers of Immune Function

Immune function may play a role in FSI health, with each wear experienced by a FSI leading to acute changes in their immune response, which may be related to an overtraining like syndrome. This may occur due to the combination of both exercise (Gabriel et al. 1992; Mitchell et al. 2002) and extreme heat (Brenner et al 1995; Cross et al. 1996), resulting in an additive effect on immune function (Brenner et al. 1998; Cross et al. 1996; Mitchell et al. 2002). The immune system is split into two parts: innate responses that provide immediate host defence via barriers, phagocytic cells (neutrophils, monocytes, macrophages), the complement system, cytokines and acute phase proteins, and adaptive responses which consist of antigen specific reactions involving T and B lymphocytes (Parkin & Cohen 2001). Neutrophils and monocytes are granulocytic phagocytic leukocytes which are attracted to sites of injury or infection by the release of cytokines to destroy foreign pathogens (Amulic et al. 2012). Basophils and eosinophils are also leukocytes associated with innate immunity; they have specific involvement in allergic responses (Stone et al. 2010). B lymphocytes primarily play a key role in humoral immunity, with the production of antibodies (Lebien & Tedder 2008), whilst T lymphocytes are involved in cell-mediated immune responses, recognizing specific antigens presented to them and coordinating the death of intracellular infections via T helper (Th) and T cytotoxic cells (Parkin & Cohen 2001). There are a large array of proteins and cells that are involved in the inflammatory and immunological response to exercise (Parkin & Cohen 2001), to reflect on all of them is outside of the scope of this literature review. The key markers that have previously been mentioned will be discussed with focus on their response to exercise and heat exposure and association with cardiovascular risk and overtraining.

2.7.1 White Blood Cells

White blood cell (WBC) counts detail the number of leukocytes found in blood, including: neutrophils, monocytes, lymphocytes, basophils and eosinophils. WBC counts may be affected by an acute wear, with a 19% increase being documented post exposure (Watt et al. 2016). This increase may have been caused by increased T_c and physiological strain stimulating an increase in adrenaline, released via the sympathodrenal system, and a rise in adrenocortical hormones, such as cortisol, from adrenal medulla due to stimulation from the hypothalamic pituitary-adrenal axis (Smith et al. 2004; Smith & Vale 2006). An increase in adrenaline alters leukocyte surface expression of adhesion molecules, which leads to the demargination of leukocytes from vessel walls into circulation (Davis et al. 1991; Dimitrov et al. 2010). Exercise may also stimulate the demargination of neutrophils migration from bone marrow into the circulation, therefore causing leukocytosis (Pyne 1994; Franchimont 2004; Fehrenbach & Schneider 2006). Smith et al., (2004) also reported increases in WBC of between 45% and 88% from morning to evening

measures taken on four days involving live fire training sessions. Furthermore, Smith et al. (2011) studied the effect of an 18 min live fire activity on 114 male FF and noted an increase from 7.38 ± 2.02 $x10^9.L^{-1}$ pre wear to 10.15 ± 2.65 $x10^9.L^{-1}$ post, $p < 0.05$, and a rise in neutrophils of 30.4%. Leukocyte increases have also been documented following two 20 min simulated search and rescue tasks (+1.2 ± 1.2 $x10^9.L^{-1}$), with increases in neutrophils also noted (+1.4 ± 1.2 $x10^9.L^{-1}$) (Walker et al. 2015). By 24hr post heat exposure leukocyte and neutrophil values had returned to baseline levels (Walker et al. 2015). Current evidence therefore suggests that a single wear increases WBC counts and circulating neutrophils.

Repeated migration of neutrophils into circulation may deplete bone marrow neutrophil stores (Gleeson 2002). This may result in neutrophils present in circulation may be less mature and have a reduced phagocytic and oxidative burst activity, as they are forced to leave the bone marrow before they are fully developed (Keen et al. 1995; Robson-Ansley et al. 2007). This may result in a decrease in the ability of the innate immune system to combat infection. Whilst an increase in circulating neutrophils has been noted following acute wears and a week of firefighting, it is unknown how neutrophil levels are affected by long term exposure to extreme heat and exercise.

Watt et al. (2016) also assessed the immune response of FSI over a 7 week rest period and a 4 week wearing period. Following the 7 week break WBC numbers decreased from a high clinical level of 7.1 ± 0.6 $x10^9.L^{-1}$ to a more acceptable clinical level of 5.8 ± 1.5 $x10^9.L^{-1}$ (Gleeson, 2006). After the 4 week heat exposure period, instructors' WBC count remained similar to post 7 week values. It is unclear what the long term effect of repetitive wears has on FSI, as 4 weeks was not a long enough duration of time to see substantial increases in WBC. Furthermore, only 6 instructors took part in the study and therefore the results cannot be generalised to a wider population and may explain why the study found insignificant changes. Studies investigating the impact of intense training have reported no significant differences in leukocyte numbers after 10 weeks of cycling (Dressendorfer et al. 2002) and 9 weeks of aerobic exercise (Bresciani et al. 2011). However these studies did not combine exercise with heat, which may further alter immune function (Niess et al. 2003). Therefore, further research is required to determine how repeated physical activity in a hot environment impacts on leukocyte numbers.

2.7.2 Interleukin-6

IL-6 is involved in the acute phase response to injury, it is mostly considered to be a pro-inflammatory cytokine, although it is also involved in some anti-inflammatory activities (Scheller et al. 2011). It initiates signaling via a transmembrane glycoprotein, g130, which is a signal transducer, and an IL-6 receptor (Heinrich et al. 2003). IL-6 can signal macrophages, neutrophils, B-cells, some T-cells and hepatocytes (Scheller et al. 2011). As part of the acute-phase response IL-6 is released from endothelial cells, where it attracts neutrophils to tissue damage (Scheller et al. 2011). When signaling hepatocytes, a release of proteins, such as C-reactive protein (CRP), are stimulated, adding to the acute-phase reaction (Bluethmann et al., 1994; Petersen & Pedersen, 2005). At the site of damage IL-6 is then subsequently involved in attracting monocytes and inducing apoptosis of neutrophils to prevent further tissue damage from neutrophil secreted proteases (Scheller et al. 2011; Wilgus et al. 2013). Leukocyte transmigration is also

increased by IL-6 as it upregulates cell adhesion molecules on endothelial cells and lymphocytes (Chen et al. 2006).

Exercise can lead to increased IL-6 release from the muscle, which may induce lipolysis and fat oxidation in adipocytes and also stimulates the release of anti-inflammatory cytokines (Petersen & Pedersen 2005). This anti-inflammatory effect of exercise may consequently reduce chronic low grade inflammation, however as suggested by the cytokine theory of overtraining, with extreme exercise this could lead to progressive systemic inflammation. Endurance exercise has been noted to increased IL-6 from 1.0 ± 0.5 $pg.mL^{-1}$ to 9.6 ± 5.6 $pg.mL^{-1}$ 1hr post a 4hr cycling (Scharhag et al. 2005), from 1.1 ± 0.2 $pg.mL^{-1}$ to 36.0 ± 6 $pg.mL^{-1}$ post a half-marathon race (Nielsen et al. 2016) and from 1.27 ± 1.19 $pg.mL^{-1}$ to 101.40 ± 50.34 $pg.mL^{-1}$ following a marathon (Suzuki et al. 2003). The large variation in magnitude of the response may be due to the differences in exercise duration, type and intensity, with the greatest increase noted during full body, weight bearing exercise over a 2:33 – 2:41 hr marathon. Heat exposure may also increase IL-6, with mice studies demonstrating that achieving a T_c of 42.4°C without infection or exercise causes a ~8.5 fold increase in IL-6, which may be caused by release from the muscle, with mRNA IL-6 expression increased 5.4 fold within the soleus muscle (Welc et al. 2012). Firefighting search and rescue tasks have been reported to cause increases in IL-6 (Walker et al. 2015), with a 45 min wear conducted by FSI noted to elicit increases in IL-6 from 7.4 ± 1.5 $pg.mL^{-1}$ to 9.9 ± 4.4 $pg.mL^{-1}$ (Watt et al. 2016), indicating that low to moderate intensity physical activity combined with heat exposure can impact IL-6 levels.

A resting IL-6 level of 7.4 ± 1.5 $pg.mL^{-1}$ following a 7 week break from wearing reported by Watt et al. (2016) is much greater than the median resting IL-6 level of 1.46 $pg.ml^{-1}$ recorded in 202 healthy adult males (Ridker et al. 2000) and baseline levels in other IL-6 exercise studies (Suzuki et al. 2003; Scharhag et al. 2005; Nielsen et al. 2016). After a 4 week period of instruction courses, FSI IL-6 levels increased further to 11.4 ± 1.0 $pg.mL^{-1}$ (Watt et al. 2016). These findings are similar, but more pronounced, to the increased resting IL-6 noted following an eight day military training course that involved physical exercise combined with sleep deprivation and food rationing (2.2 ± 0.3 $pg.mL^{-1}$ to 4.5 ± 1.02 $pg.mL^{-1}$) (Nielsen et al. 2016). This may demonstrate low grade chronic inflammation following a period of repeated stressors. Elevated resting levels of IL-6 may also be a predictor of cardiovascular illnesses (Rauchhaus et al. 2000; Ridker et al. 2000; Spoto et al. 2014). The increase in cell adhesion molecules and CRP, combined with an increase in platelet production and reactivity induced by IL-6, may be involved in the formation of atherosclerosis (Lindmark et al. 2001). The involvement of IL-6 in the immune response, overtraining and atherosclerosis highlights the importance of further investigation into the presence of this cytokine within FSI.

2.7.3 C Reactive Protein

CRP is an acute phase protein which can activate the complement pathway, stimulate phagocytosis and bind to immunoglobulin receptors (Pepys & Hirschfield 2003; Black et al. 2004). Following an acute inflammatory stimulus, CRP can increase up to 1000 fold due to an increased synthesis from hepatocytes (Black et al. 2004). IL-6, when combined with transcription factors, stimulates the transcription of CRP, a

process which can be enhanced by the presence of IL-1β (Black et al. 2004; Ridker 2016). CRP can also be synthesized by neurons, monocytes, lymphocytes and atherosclerotic plaques, although CRP from these sources does not substantially influence plasma CRP levels (Jialal et al. 2004). Slightly elevated circulating levels of CRP, between 3 and 10 $mg.L^{-1}$, have been linked with an increased risk of cardiovascular disease, metabolic syndromes and colon cancer, due to the association of these conditions with chronic inflammation (Pepys & Hirschfield 2003). Independent of other risk factors, CRP levels predict the occurrence of myocardial infarction, coronary artery disease death, stroke, peripheral arterial disease and sudden death (Ridker 2003). There is also evidence to suggest that CRP itself is involved in the development of atherosclerosis, with CRP having been found bound to low density lipoproteins, upregulating endothelial adhesion molecules and increasing the uptake of low density lipoproteins by macrophages (Jialal et al. 2004). However, the role of CRP in the pathogenesis of atherosclerosis is yet to be firmly established. (Black et al. 2004; Koenig 2013). The American Heart Association and Centers for Disease Control and Prevention classify < 1 $mg.L^{-1}$ as low risk, 1 - 3 $mg.L^{-1}$ as average risk and > 3 $mg.L^{-1}$ as high risk for cardiovascular disease with a Framingham risk score of 10 - 20% (Pearson et al. 2003). A single measurement of CRP is sufficient in the majority of cases, although levels > 10 $mg.L^{-1}$ should be retested as they indicate the presence of major infections and trauma (Ridker 2003) . Weight loss, diet and exercise have all been noted to reduce both CRP and cardiovascular risk (Ridker 2003).

The acute CRP response to exercise is unclear as whilst theoretically levels should increase due to stimulation by elevated IL-6, as has been noted post marathon performance (Weight et al. 1991; Siegel et al. 2001; Kasapis & Thompson 2005), a heat stress test involving a 60 min cycle performed at 35% of peak power output by trained cyclists did not elicit a change in CRP (Costello et al. 2018), nor did two consecutive 20 min simulated fire exposures (Walker et al. 2015). A 10% decrease in CRP has also been noted post a wear completed by FSI (Watt et al. 2016). It is proposed this may be a consequence of increased CRP breakdown or removal rather than a reduction in synthesis (Watt et al. 2016).

Short term training has also been suggested to have no effect on CRP, with an 11 consecutive day heat acclimation protocol (Costello et al 2018) and 11 sessions, across 6 days, of high intensity interval training (Wiewelhove et al. 2015) having no effect on CRP. However, regular training (≥ 4 times a week over a 2 year period) has been reported to reduce resting CRP, with male swimmers and rowers having a reduced CRP (80% and 48%, respectively) compared to non-trained individuals (Kasapis & Thompson 2005). Joggers and aerobic runners have also been identified to be significantly less likely to exhibit elevated CRP (OR = 0.33 and OR = 0.31, respectively) compared to cyclists (OR = 1.30), swimmers (OR = 0.62) and weightlifters (OR = 0.83), suggesting that the exercise training type itself may also be a factor in CRP levels, although this could be due to confounding variations in intensity and duration of exercise (King et al. 2003). Nine months of marathon training has been reported to reduce CRP by 31% compared to a non-training control group (Mattusch et al. 1999), whilst exercise training completed by sedentary individuals over 20 weeks (3 sessions per week) can reduce CRP for those in the high risk catergory by 1.34 $mg.L^{-1}$ (-24%) (Lakka et al. 2005). A meta-analysis concluded that from 83 studies CRP had a small (effect size = 0.26) but significant decrease with training lasting > 2 weeks (Fedewa et al. 2016). These associations with

physical activity may be a consequence of reduced obesity levels and decreases in IL-6 response that can occur with training (Kasapis & Thompson 2005; Fedewa et al. 2016).

Alternatively, due to the association of CRP with inflammation it has been suggested as an indicator of overtraining (Smith 2000; Smith 2004; Kreher & Schwartz 2012; Meeusen et al. 2013). Following 1 month of high intensity Judo training combined with fasting for Ramadan CRP has been noted to increase from 2.93 ± 0.26 mg.L^{-1} to 4.60 ± 0.51 mg.L^{-1}. From a sample of 6 FSI displaying signs of overtraining, resting CRP was greater than that exhibited by a control group (7.4 mg.L^{-1} vs. 3.4 mg.L^{-1}) (Watt et al., 2016). However, there is a dearth of research into the impact of overtraining/high intensity training on CRP itself. Overall, as a consequence of the established association of elevated CRP with cardiovascular risk and the elevated risk of cardiovascular events noted within the fire service, it is of particular interest to identify CRP levels in FSI. However, CRP and IL-6 alone cannot distinguish a chronic inflammatory status and other key regulators of inflammation, such as IL-1β and TNFα, should also be considered (Del Giudice & Gangestad 2018).

2.7.4 Interleukin-1 beta

IL-1β is one of seven agonistic cytokines within the IL-1 family, which are cytokines involved in the mediation of autoinflammatory, autoimmune, infectious and degenerative diseases (Garlanda et al. 2013). IL-1β is produced by monocytes, macrophages, skin dendritic cells and brain microglia in response to other cytokines, pathogen-associated molecular patterns (PAMPs) and the complement system (Barksby et al. 2007). The precursor to IL-1β must be cleaved intracellularly by caspase-1 to enable the active IL-1β cytokine to be released; increased caspase-1 activity causing elevated IL-1β is linked to inflammation and autoinflammatory diseases (Barksby et al. 2007; Garlanda et al. 2013). IL-1β precursor can also be cleaved by extracellular neutrophil enzymes to produce the active IL-1β (Garlanda et al. 2013). IL-1β is pyrogenic and proinflammatory, increasing the expression of adhesion moleculares in endothelial cells and promoting diapedesis and the acute phase response (Barksby et al. 2007). Consequently, it is involved in numerous acute and chronic inflammatory conditions, such as rheumatoid arthritis, type 2 diabetes, myeloma and sepsis. IL-1β is present in low levels in healthy individuals, often being difficult to detect with a standard enzyme-linked immunosorbent assay (ELISA) analysis (Dinarello 2011). Daily production is suggested to be 6 ng which can increased 5 - 10 fold in individuals with autoinflammatory diseases (Dinarello 2011).

Increased IL-1β is also involved in atherothrombosis, due to its ability to stimulate procoagulant activity and leukocyte adhesion to vascular endothelial cells (Ridker et al. 2011). Furthermore, IL-1β is found within atherosclerotic lesions, with atherosclerotic arteries containing increased IL-1β levels, with the increase directly proportional to the severity of the atherosclerosis (Galea et al. 1996). IL-1β inhibitors are being trialed to reduce the rates of recurrent myocardial infarction, stroke and cardiovascular death in patients with stable coronary artery disease (Ridker et al. 2011). This trial indicates that treatment with an antibody targeting IL-1β can cause a 15% reduction in major adverse cardiovascular events (Ridker 2018).

Evidence suggests that IL-1β levels are not elevated following acute exercise, such as an ultra-marathon (100 km) performance (Krzemiński et al. 2016), marathon performance (Suzuki et al. 2000) and 1hr cycling at 75% of VO_2 max (Ullum et al. 1994). IL-1β levels are also not effected in middle aged individuals with osteoarthritis, by either moderate intensity swimming or cycling 3 days per week for 12 weeks (Alkatan et al. 2016), in healthy young adults across a 6 week high intensity interval training programme or with moderate intensity continuous training (Paolucci et al. 2018), or in elderly and young individuals following a 12 week regular moderate intensity exercise programme (Stewart et al. 2007). However, overtraining with a 10 week running protocol has been noted to induce 60 - 126% increases in IL-1β compared to a control group, although this was noted in mice and therefore may not be generalizable to a human population (Pereira et al. 2015). Consequently, although it is clear IL-1β is involved in inflammation and atherosclerosis and has been suggested to be mechanistically involved in overtraining, it remains unknown how it is effected by repeated stressor exposure and if it is involved in the prevalence of symptoms of ill health in FSI.

2.7.5 Tumour Necrosis Factor-alpha

The pro-inflammatory cytokine TNFα release from leukocytes and smooth muscle cells can be induced by lipopolysaccharides, antigens, IL-1 and TNFα itself (Vassalli 1992; Feldman et al. 2000). TNFα has two receptors, a high affinity receptor (TNFR-1) and a low affinity receptor (TNFR-2) which allow for signaling to cells (Chen & Goeddel 2002). It is involved in the regulation of other cytokines, such as IL-1 and IL-6, hormones, such as adrenaline, and leukocyte and endothelial cells (Vassalli 1992). Release of TNFα increases adhesion molecules at sites of inflammation, so promotes leukocytosis (Feldman et al. 2000). TNFα also has cytotoxic effects, playing an important role in cell apoptosis (Feldman et al. 2000). Extensive activation of TNFα can lead to tissue necrosis, with TNFα having a causative role in numerous diseases, such as sepsis, osteoporosis and autoimmune diseases (Chen & Goeddel 2002). TNFα may play a role in the development of atherosclerosis, with those with atherosclerosis more likely to exhibit greater levels of TNFα ($\chi^2 = 8.42$, $p = 0.004$) (Bruunsgaard et al. 2000). It is proposed that TNFα may be involved in the pathophysiology of atherosclerosis due to its stimulation of cellular adhesion molecules and IL-6 (Skoog et al. 2002).

It is unclear if TNFα increases following acute exercise. Wright-Beatty et al. (2014) reported no differences in TNFα following 4 x 15 min cycling in 35 ± 0.1°C conducted by FF, whereas moderate exercise has also been associated with an inhibition of TNFα expression, a 28 ± 10% reduction in TNFα mRNA expression at the end of 60 min exercise whilst wearing protective clothing (Jiminez et al. 2008). In the presence of infection pro-inflammatory cytokines TNFα and IL-1β are the first to be released and stimulate IL-6 production, however it is suggested that exercise alone does not elicit the same response, with TNFα not responding (Petersen & Pedersen 2005). However, Walker et al. (2015) documented increases of ~14% following simulated search and rescue in 100 ± 5°C. It is suggested that an increase in lipopolysaccharides from the gut with high T_c may result in an increase in TNFα, indicating why exercise alone, or with only moderate increases in T_c, does not elicit increased TNFα (Walsh & Whitham 2006; Peake et al. 2008;

Cooper et al. 2010). Wright-Beatty et al. (2014) recorded modest increases in T_c of 0.3 - 0.5°C, compared to Walker et al. (2015) who reported increases of 1.4 ± 0.5 °C. Increased TNFα has been reported 2hr post exercise in the heat compared to no increase following the same cycling exercise in a cool environment (2.9 ± 0.6 pg.mL^{-1} vs. 2.0 ± 0.3 pg.mL^{-1}, respectively) (Cooper et al. 2010). In contrast, Jiminez et al (2008) reported end T_{re} of 39.2 ± 0.2°C with inhibited monocyte TNFα expression, although this may indicate that the TNFα increase does not originate from monocytes, but possibly from another source. Increased TNFα post 4.06 ± 0.44 hr marathon (9.0 pg.mL^{-1} vs. 10.3 pg.mL^{-1}) occurred despite no changes in blood mononuclear cell mRNA TNFα expression, with exercising tissues instead proposed as the source (Bernecker et al. 2013).

Regular moderate exercise has been reported to reduce resting levels of TNFα in clinical populations, for example an average reduction of 8.5% was reported from four training centers implementing exercise programmes for chronic heart failure patients (Smart et al. 2011) and a 1.4 pg.mL^{-1} reduction in TNFα following a 12 week cycling intervention completed by impaired glucose tolerance obese women (Straczkowski et al. 2001). In comparison, high volume or intensity training may increase TNFα, with TNFα increases reported in elite junior rowers over a 6 week training period (1.3 ± 1.9 pg.mL^{-1} vs 2.4 ± 1.4) associated with increases in training distance (Main et al. 2009). Although no difference in TNFα was present at rest between elite rowers (21.4 ± 3.7 pg.mL^{-1}) and non-athletes (28.6 ± 8.8 pg.mL^{-1}), p = 0.455 (Nieman et al. 2000), or following 4 weeks of intensified cycle training (7.1 ± 1.4 pg.mL^{-1} vs. 7.4 ± 1.8 pg.mL^{-1}) (Halson et al. 2003). The variation in training time and intensity may be the cause of the mixed findings reported. There has been no previous evaluation of TNFα in FSI, whether at rest, following live fire exposure, or after a period of instruction courses and therefore it is unknown if their TNFα baseline levels or response to exercise is similar to the general population. The involvement of elevated resting TNFα with systemic low-grade inflammation and numerous inflammatory diseases, including an association with atherosclerosis, indicates that evaluation of TNFα in FSI is warranted.

2.7.6 Immunoglobulin-G

Chronic inflammation can also be identified with elevated levels of Immunoglobulin-G (IgG), which can provides information on the humoral immune status (Gonzalez-Quintela et al. 2008), with increased levels being an indicator of connective tissue diseases, hepatic diseases, chronic inflammation, or acute and chronic infections (Dispenzieri et al. 2002). IgG is an antibody created and released by plasma B cells, usually accounting for 75 - 80% of immunoglobulins in circulation and 10 - 20% of total plasma protein (Vidarsson et al. 2014). IgG works to recognise, neutralise and eliminate pathogens and toxic antigens (Kaneko et al., 2006; Brolinson & Elliot, 2007). Elevated IgG is also an indicator of pulmonary infections (Twigg 2005; Van De Weert-Van Leeuwen et al. 2014); IgG is the dominant antibody that protects the lung from influenza infection and is the second line of defence after IgA to protect the upper respiratory tract (Renegar et al. 2004; Bahadoran et al. 2016). A reduction in IgG may increase the occurrence and severity of mucosal infections, particularly those caused by microbes in the respiratory tract (Spiekermann et al. 2002)

An acute ~45 min wear has been documented to cause a reduction of IgG from (15.9 ± 4.8 $pg.ml^{-1}$) to (10.6 ± 2.9 $pg.ml^{-1}$) (Watt et al. 2016). However, an ultra-marathon (90 km) completed in 9.45 hrs has been noted to increase IgG by 12%, with values returning to baseline at 24 hrs post exercise (McKune et al. 2005). Regular exercise has been suggested to decrease IgG, with a 20 day rugby training camp, training 6$hr.day^{-1}$, decreasing IgG by 8% (Mashiko 2004). A long term study of swimmers suggests that mucosal IgG may be significantly lower for individuals throughout an intense 7 month training program, compared to a moderate exercise group (Gleeson et al. 1995). However serum IgG showed no differences between swimmers and a control group. These findings were repeated by Gleeson et al. (2000) following a 12 week swim training programme. This suggests that high levels of stress may lower the mucosal defence over time, but humoural immunity beyond the initial host defence may not be effected. Another study focusing on changes in IgG, noted a 14% decrease following an 8 week resistance training program working at 80% of 1RM, with a decrease also being present at 4 weeks (Mohebbi et al. 2012). This suggests that with high intensity exercise only 4 - 8 weeks may be needed for continuous IgG suppression to occur. However, regular training of athletes over a 4 month season has also been reported to have no impact on IgG levels (Córdova et al. 2010), whilst a high-training load during Ramadan elevated IgG levels by 15%, with levels remaining elevated 3 weeks after fasting was completed (Chaouachi et al. 2009). Overall, it is yet to be confirmed what effect exercise and training has on IgG, with a variety of differing reports published. It is likely that the differences in exercise type, duration and intensity play a role in the variability of findings, with the IgG response perhaps being stressor specific. Assessing the IgG response of FSI may enable a better understanding of the relationship between anecdotally reported illnesses and working practices.

2.8 Literature Summary

FSI regularly experience high levels of physiological and perceptual strain, due to their frequent exposure to extreme temperatures whilst wearing encapsulating clothing. Consequently, they may be at an increased risk of heat or cardiovascular illness. There are numerous acute interventions that may reduce this strain, with hydration and post-cooling techniques well documented. Alternatively, guidance on pre-cooling use remains vague. Heat tolerance may offer chronic protection against heat illnesses, however tolerance levels have not previously been ascertained in FSI. FSI have anecdotally reported frequent colds, severe fatigue and night sweats. Frequently completing wears may cause acute immunological changes, including the development of chronic systemic inflammation, which has been briefly documented with a small group of instructors. It is proposed that these responses may be similar to that of an overtraining syndrome, with elevated inflammation also associated with the increased risk of a cardiovascular event. However, there is no literature on the long term impact that regular exposure to highly stressful environments has on immune function, possibly due to the ethical implications of putting workers or athletes under such conditions for experimental purposes. Further investigation is therefore warranted into the long term immunological response of FSI to enable working patterns to be organized to minimise the risk of illness.

2.9 Research Questions and Hypotheses

The following research questions and associated hypothesis are proposed for this thesis.

Experimental Study 1: Fire Service Instructors' Working Practices: A UK Survey

- Do FSI use preparation and recovery techniques?
- Do FSI differ from FF in the symptoms of ill health they experience?
- Is there an association between FSI experiencing symptoms of ill health and working practices?

Based on the evidence in the literature it was hypothesized that (1) the majority of FSI would not use pre or post cooling techniques, but would use hydration guidelines, (2) a greater number of FSI would report symptoms of ill health than FF and (3) presence of ill health would be associated with exposure numbers.

Experimental Study 2: The Acute Effect of Training Fire Exercises on Fire Service Instructors

- Do different training exercises elicit different physiological responses?
- What effect does FSI role have on physiological responses?
- Does a day of fire exposures alter inflammatory and cardiac risk markers?
- What effect does exercise patterns have on inflammatory markers?

It was hypothesized that (1) there will be a significant difference in T_{re} responses to different exercise, (2) there will be a significant difference in T_{re} responses to roles, (3) a day of exercises will result in an increase in inflammatory and cardiac risk markers and (4) there will be a significant difference in inflammatory markers between exercise patterns.

Experimental Study 3: Practical pre-cooling methods for Fire Service Instructors

- What effect do practical pre-cooling methods have on physiological and perceptual responses to a simulated fire exposure?
- What effect do practical pre-cooling methods have on the inflammatory response to a simulated fire exposure?

It was hypothesized that (1) phase change vest, forearm cooling and ice slurry consumption would result in reduced physiological and perceptual strain compared to a control trial, (2) ice slurry consumption would generate the greatest reductions in strain compared to control and (3) all methods would result in a reduced inflammatory response, recorded as a reduction in IL-6, compared to control.

Experimental Study 4: The Validity and Reliability of a New Occupational Heat Tolerance Test

- Is the new heat occupational tolerance test (HOTT) valid in comparison to the standard heat tolerance test?
- Is the new HOTT reliable?

It was hypothesized that (1) T_c responses would be similar between the new and standard tests and (2) that physiological and perceptual responses would be similar at the end of both HOTT trials

Experimental Study 5: Heat Tolerance of Fire Service Instructors

- Do FSI develop a greater tolerance to heat than non-wearing individuals?
- What effect does heat tolerance have on FSI inflammatory response to heat exposures?
- Is FSI tolerance status maintained with normal working practices?

It was hypothesized that (1) FSI will have an increased tolerance compared to a control group, as evident by a reduced T_{re} at the end of the HOTT, (2) FSI will have a decreased inflammatory response to the HOTT compared to a control group, as evident by decreased inflammatory markers such as IL-6 and (3) FSI and the control group will maintain their physiological, perceptual and inflammatory responses following a 2 month period of normal working practices.

Experimental Study 6: Baseline Haematological and Immunological Measures in Fire Service Instructors and Firefighters

- Do FSI exhibit a different haematological and immunological profile to FF?
- What effect does the number of fire exposures have on cytokine levels?
- Is the presence of ill health amongst FSI related to cytokine levels?
- Is the current informally suggested guidance of a 9 wear a month limit appropriate?

It was hypothesized that (1) FSI will exhibit greater IL-6, CRP, cTnT and platelet volume and counts than FF, (2) a greater number of fire exposures will be related with elevated levels of cytokines, (3) there will be an association between those above the 9 wear limit and those exhibiting cytokine levels above the reference limits and (4) there will be an association between immunological markers and reports of ill health.

3 GENERAL METHODS

3.1 Ethics, health and safety

All experiments were approved by the University of Brighton Ethics Committee. All studies were conducted in line with university operating procedures and risk assessments. All participants completed medical questionnaires and consent forms before participating. Participants were free to withdraw at any time and any data collected was anonymous and confidential. Participants were also clearly informed that their discussions regarding participation or withdrawal would not be communicated to their employer.

3.1.1 Experimenters

Two experimenters were present at all times. When using the heat chamber, one experimenter remained outside, whilst the other was inside the chamber to ensure the safety of participants. During testing at fire service training grounds experimenters remained in a safe zone away from the fire and wore appropriate protective clothing, as directed by FSI on site. A trained first aider was available at all times during testing. Blood samples were collected by a trained phlebotomist.

3.1.2 Equipment cleaning procedure

To avoid contamination, face masks were soaked for 10 min in 1% Virkon disinfectant (Antec International UK) and then kept in a sterile environment before use. Skin thermistors and iButtons were cleaned with alcohol wipes and the treadmill was wiped down with disinfectant (Bioguard).

3.1.3 Waste disposal

Single use rectal thermistors were placed in biohazard bags then incinerated after use (PHS Group, Caerphilly, UK). Sharps (needles) were placed in a sharps bin. All biological material and waste was handled and disposed of in line with relevant guidelines.

3.1.4 Criteria for termination of experiments

Testing that occurred in the heat chamber in the Welkin Laboratories was terminated if T_{re} exceeded 39.7ºC, or if volitional exhaustion, heat illness, syncope, nausea, vomiting, disorientation, or exhaustion presented. Cooling techniques were then applied when removed from the heat, including: water ingestion, cold water immersion of hands and feet and use of a fan. Participants could only leave the laboratory when their T_{re} was within 0.5ºC of their baseline $T_{re.}$ Testing conducted at fire training grounds were terminated in accordance with the Fire and Rescue Service's rules, with the decision to terminate made by FSI.

3.2 Participants

Participants were recruited via email, twitter, posters and face to face meetings. After an expression of interest, information packs were provided with the full details of the study. Participants were then given time to consider the information. Experimenters then discussed study details with participants to ensure all details had been understood and to give participants the opportunity to ask any questions before agreeing to take part.

3.2.1 Medical Criteria

To be eligible to take part participants had to be active, with no known injuries. They also had to have no previous history of anaphylactic shock to needles, no rectal bleeding, anal fissures, or haemorrhoids. Participants also could not be involved in other research experimentation at the same time. FSI had to be declared fit to work by their Occupational Health.

3.3 Facilities

The majority of testing occurred at the Welkin Laboratories, which are laboratories accredited by the British Association of Sport and Exercise Sciences within the University of Brighton, based on the Eastbourne campus. These sessions involved participants entering an Environmental Chamber, 4.5 x 3.5 x 3m (WatFlow control system; TISS, Hampshire, UK).

Field testing locations included training grounds at Surrey Fire and Rescue Service Headquarter, Croydon Road, Reigate, RH2 0EJ; Gatwick Airport, London, RH6 0NP; East Sussex Fire and Rescue Service Training Centre, Batts Bridge Road, Maresfield, Uckfield, TN22 2HN; and The Fire Service College, London Road, Moreton-in-Marsh, GL56 0RH. Other fire stations were also visited for collection of blood samples, including Preston Circus Community Fire Station, Brighton; Bohemia Road Community Fire Station, Hastings; Eastbourne Community Fire Station, Eastbourne and Ashford Fire Station, Ashford.

3.4 Environmental conditions

3.4.1 Ambient laboratory temperature control

Seated resting ambient conditions were maintained using air conditioning. Air temperature, humidity and barometric pressure were recorded for each session using a portable weather station.

3.4.2 Experiment temperature and humidity

An environmental chamber with an available range of -20°C to +50°C and 0% to 95% relative humidity (RH) was used to conduct laboratory heat testing. Unless otherwise stated experimental trials were

conducted in 50ºC and 10% RH. Manual recording of chamber conditions was performed every 5 min during exercise trials to accurately describe the environment.

During live fire exposures temperatures varied throughout, with temperatures ranging from 70°C at floor level to 700°C at ceiling height, as recorded by thermocouples placed inside the building structures.

3.5 Treadmill Exercise Trials

All laboratory exercise was conducted in the environmental chamber on a Woodway treadmill (WoodwayPRO, Woodway GmbH, Weil am Rhein, Germany). Exercise intensity never exceeded a walking pace, but was altered slightly based on the needs of the study. Gradient remained at 1% during exercise tests (Jones & Doust 1996). Power produced whilst walking on the treadmill was calculated by:

$$\text{Vertical distance} = \text{speed } (m.min^{-1}) \times \text{incline} \times \text{time (min)}$$

$$\text{Power (W)} = (\text{body mass (kg)} \times \text{vertical distance}) / \text{time (min)}$$

3.6 Pre-trial diet and exercise standardisation

Experimental sessions were conducted at the same time of day, to account for changes due to circadian rhythms. Student and control participants had not been heat acclimated or experienced > 25ºC greater than 3 times a week within the 6 months prior to testing. FSI had conducted their normal working heat exposure routine. Prior to testing all participants were required to abstain from alcohol and exhaustive exercise for 24 hours, and from caffeine for 12 hours. They were also requested to consume similar diets in the 48 hours before each session and to arrive at the session in a euhydrated state.

3.6.1 Hydration assessment

Participants were required to have an equal and adequate level of hydration at the beginning to each testing session. Adequate hydration was assessed by achieving two of three criteria: urine osmolality < 700 $mOsm.kgH_2O^{-1}$, urine specific gravity < 1.020, or urine colour ≤ 3 (Sawka et al. 2007). At laboratory visits if participants were not euhydrated they consumed an additional 500 mL of water and were reassessed following a 30 min rest period.

3.6.1.1 Urine osmolality

Osmolality was measured using a handheld Osmocheck (Vitech Scientific Ltd, West Sussex, UK). It was calibrated prior to use with distilled water. Approximately 1ml of urine was pipetted on to the lens whereby the sample was measured using refractometry.

3.6.1.2 Urine specific gravity

To measure urine specific gravity a handheld refractometer was used (Index Instruments Ltd, Cambridgeshire, UK). It was calibrated with distilled water prior to use. Approximately 2ml of urine was pipetted onto the glass lens and the refractometer then held to the light and, by looking through the eye lens, values were recorded from the scale within.

3.6.1.3 Reliability of urinary analysis

Urine samples were collected from 10 individuals and analysed for osmolality and urine specific gravity. Each sample was measured in duplicate. Absolute and relative technical error of the measurement (TEM) was calculated, along with intra class correlation coefficient with 95% confidence intervals, see Table 3.1.

Table 3.1 *Mean ± standard deviation (SD), typical error of the measurement (TEM) and TEM as a coefficient of variation (TEM (CV %)) for repeated measures of osmolality and specific gravity.*

	Osmolality (mOsm.kgH_2O^{-1})		**Specific Gravity**	
	1	**2**	**1**	**2**
Mean ± SD	440 ± 257	443 ± 257	1.015 ± 0.006	1.015 ± 0.006
TEM	3.24		0	
TEM (CV %)	1.26		0	

3.7 Anthropometric assessment

3.7.1 Height

A fixed stadiometer (SECA 220 Stadiometer, Germany) was used to measure height, with the participant asked to stand vertically in the anatomical position, facing away from the meter. The stadiometer arm was then lowered to horizontal. The scale was read to the nearest 0.5 cm.

3.7.2 Body mass

Nude body mass (BM) was measured using Adam GFK 150 digital body scales (Adam Equipment Inc., Connecticut, USA) and recorded to 0.01 kg. This procedure was carried out in a private room, where participants self-reported their BM to the experimenter. The scales were calibrated prior to use, using a 20 kg weight.

3.7.3 Body Composition

Body composition was assessed using an air displacement plethysmograph (BOD POD, COSMED, Italy) which uses whole body densitometry. Participants were asked to present themselves in a euhydrated stated, which was determined as previously described. They were all requested to wear minimal clothing, e.g. tight shorts or swimming costume, a swimming hat, to remove any jewellery and to void their bladders prior to assessment. Body mass was then determined using integrated BOD POD digital scales. Small volume changes within the chamber, caused by a computer controlled diaphragm, allow for the resulting pressure changes to be measured and consequently the determination of body volume. The volume of the empty BOD POD chamber was first determined using a 50L calibration cylinder. Participants then sat in the BOD POD and the door was sealed, the resulting volume was subtracted from the initial empty chamber volume. Thoracic lung volume was then estimated and taken into account to give a final body fat (BF) percentage.

3.7.3.1 Bod Pod Validity

To determine the construct validity of the BOD POD, 8 participants (male: 4, female: 4, age: 24 ± 3 yrs, mass: 65.37 ± 13 kg) completed a body composition session involving calculation of BF percentage via the gold standard assessment of hydrostatic weighing, 7 site skin fold assessment and use of the BOD POD.

Skin folds were measured at 7 locations: subscapular, triceps, midaxillary, pectoral, abdominal, suprailliac and mid thigh, using skin fold calipers. An average of three measurements within 2 mm of each other was taken. BF percentage was then calculated by the following equations (Siri 1956; Jackson & Pollock 1978; Jackson et al. 1980):

Body density:

Male

$$= 1.112 - (0.00043499 \times \sum 7) + [0.00000055 \times (\sum 7)^2] - (0.00028826 \times \text{age})$$

Female

$$= 1.097 - (0.00046971 \times \sum 7) + [0.00000056 \times (\sum 7)^2] - (0.00012828 \times \text{age})$$

BF percentage:

$$= [(4.95/\text{body density}) - 4.5] \times 100$$

Hydrostatic weighing was conducted in ~30ºC water in a plunge tank. Participants were asked to sit in the sling of the weighing scales, exhale maximally and slowly fully submerge themselves. A recording of the mass was then taken before the participant re-surfaced. An average of 10 successful measures was taken. BF percentage was calculated using the following equations:

Residual lung volume (RV) (Boren et al. 1966):

Male

= [Age (years) x 0.0115] + [Height (cm) x 0.019] – 2.24

Female

= [Age (years) x 0.0210] + [Height (cm) x 0.023] – 2.978

Body volume (V_B):

= [(BM_D) – (BM_{UW})] / density of water

where BM_D is mass in air and BM_{UW} is mass in water.

Body density ($D_{B)}$:

= BM_D / (V_B – RV – GIG)

where GIG is gastrointestinal gases estimated at 100 mL.

BF percentage:

= [(4.95/body density) – 4.5] × 100

Intra-class Correlation Coefficients (ICC) with 95% confidence intervals (95% CI) were calculated between each method (Table 3.2). Bland-Altman plots were also generated with limits of agreement (LOA), with the individual participant differences between the two methods plotted against the respective individual means (Figure 3.1).

Table 3.2 *Mean ± standard deviation (SD), typical error of the measurement (TEM), TEM as a coefficient of variation (TEM (CV %)) and intra-class correlation coefficient (ICC) with 95% confidence intervals (CI) for body fat (%) measured using the BOD POD and compared to skinfolds and hydrostatic weighing.*

	Body Fat %		
	BOD POD	**Skinfolds**	**Hydrostatic Weighing**
Mean ± SD	16.17 ± 10.49	13.57 ± 8.36	15.52 ± 11.51
ICC (95% CI)		0.928 (0.675 - 0.985), p < 0.001	0.974 (0.874 - 0.995), p < 0.001
Mean bias (LOA)		-1.9 (-12.31, 8.42)	0.65(-6.56, 7.86)

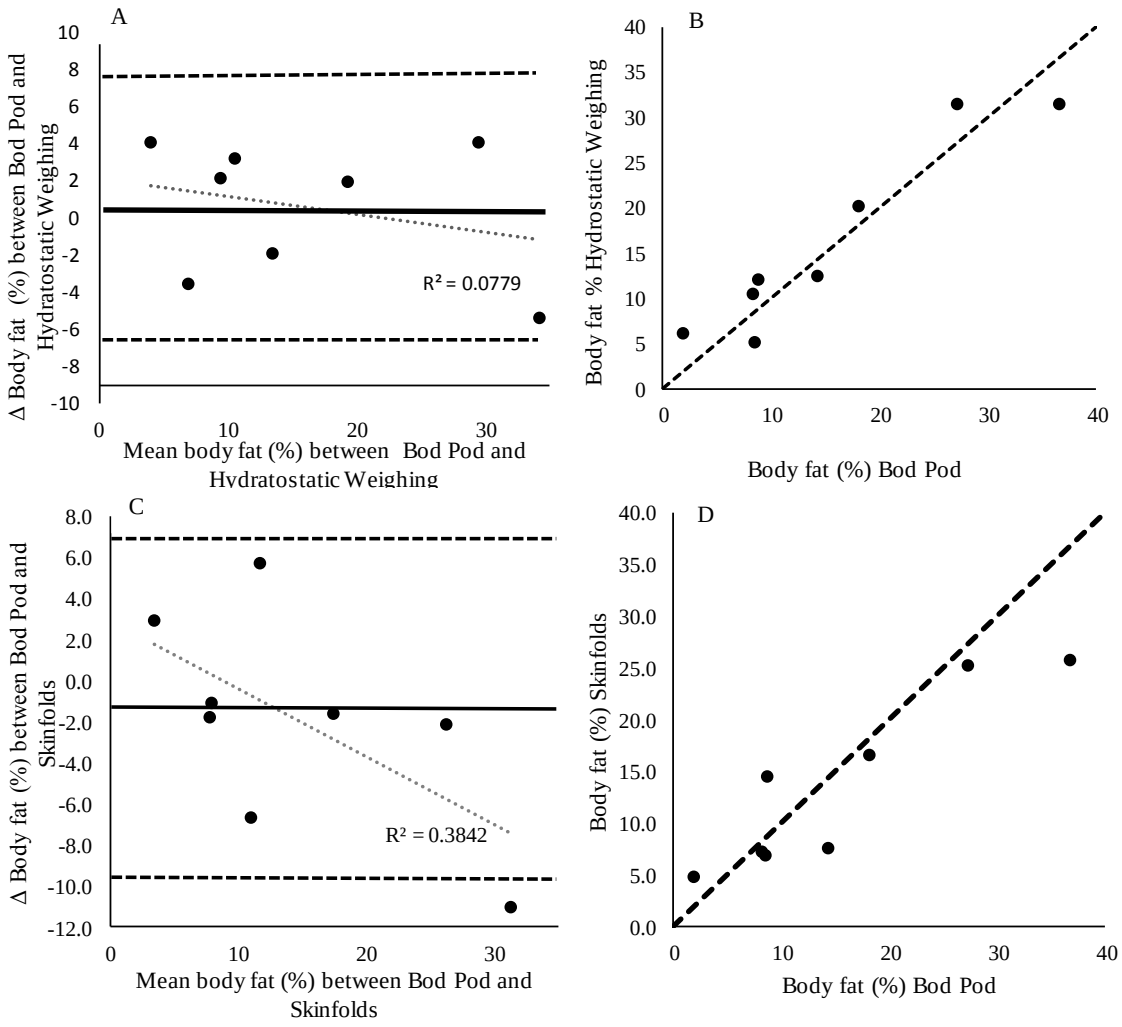

Figure 3.1 *Bland-Altman plots presented with mean bias and LOA and line of equality graphs for mean body fat (%) between Bod Pod and Hydrostatic Weighing (A, B, respectively) and Bod Pod and skinfold (C, D, respectively).*

3.7.3.2 Bod Pod Reliability

To assess the reliability of the BOD POD, 9 participants (male: 4, female: 5, age: 24 ± 3 yrs) were measured for BF percentage on two consecutive days. Testing occurred at the same time of day, prior to breakfast and whilst participants were in a euhydrated state. Intra-class correlation coefficients with 95% confidence intervals were calculated, along with absolute and relative typical error of measurements (TEM). TEM was calculated from the standard deviation of the mean difference for each pair of trials using the formula TEM = $SD_{(diff)} / \sqrt{2}$, see Table 3.3.

Table 3.3 *Mean ± standard deviation (SD), typical error of the measurement (TEM) and TEM as a coefficient of variation (TEM (CV %)) recorded via the BOD POD on two occasions.*

	BODY FAT %	
	1	2
Mean ± SD	15.46 ± 10.20	16.33 ± 10.53
TEM	0.74	
TEM (CV%)	4.67	

3.8 Physiological measures

3.8.1 Rectal Temperature

T_c was measured via a Henley single use rectal temperature probe (449H, Henleys Medical, Hertfordshire, UK) passed 10 cm past the anal sphincter, to provide rectal temperature T_{re}. Temperature was then displayed on logging monitors (YSI, 4600 series, YSI, Hampshire, UK).

3.8.1.1 Reliability of rectal thermistor

Reliability of the rectal thermistor was determined during a resting state in temperature laboratory conditions (23°C, 34% RH). One participant rested for 10 min to allow for T_{re} to stabilise, measurements were then recorded every minute for two 10 min periods. Reliability tests were there conducted on the duplicate measures (Table 3.4.).

Table 3.4 *Mean ± standard deviation (SD), typical error of the measurement (TEM) and TEM as a coefficient of variation (TEM (CV %)) for repeated rectal temperature (T_{re}) measurements.*

	Resting T_{re} (°C)	
	1	2
MEAN ± SD	37.47 ± 0.01	37.49 ± 0.01
TEM	0.01	
TEM (CV%)	0.02	

3.8.2 Skin temperature

Skin temperature during laboratory sessions was measured via contact skin thermistors placed at four locations, according to Ramanathan, (1964): pectoral, triceps, quadriceps and gastrocnemius and recorded via a 1000 series Squirrel Data Logger (Grant Instruments, Cambridgeshire, UK).

During live fire exposure wireless iButtons were used to record skin temperature at 1 min intervals; they were positioned identical to skin thermistor locations. Data were downloaded from the iButtons post exposure.

Mean skin temperature (T_{skin}) was then determined using measurements taken from the four skin sites:

$$T_{skin}\ (^0C) = 0.3(T_{chest} + T_{upper\ arm}) + 0.2(T_{upper\ leg} + T_{lower\ leg})$$ (Ramanathan, 1964).

3.8.2.1 Reliability of skin thermistors

The reliability of the skin thermistors and iButtons were determined following a 10 min stabilisation period in ambient conditions (26°C, 40% RH). Measures were then recorded every minute for two 10 min periods, with reliability tests conducted on the duplicate measures, see Table 3.5 and 3.6.

Table 3.5 *Mean ± standard deviation (SD), typical error of the measurement (TEM) and TEM as a coefficient of variation (TEM (CV %)) for repeated skin thermistor measurements.*

	SKIN THERMISTOR (°C)	
	1	**2**
MEAN ± SD	25.97 ± 0.04	26.00 ± 0.05
TEM	0.05	
TEM (CV%)	0.20	

Table 3.6 *Mean ± standard deviation (SD), typical error of the measurement (TEM and TEM as a coefficient of variation (TEM (CV %)) for repeated iButton measurements.*

	IBUTTON (°C)	
	1	**2**
MEAN ± SD	26.16 ± 0.16	26.75 ± 0.41
TEM	0.32	
TEM (CV%)	1.23	

3.8.2.2 Validity of iButtons

The construct validity of the iButtons was assessed across a range of skin temperature by comparing values to those recorded from standard use skin thermistors. On three occasions the iButtons and skin thermistors were worn underneath PPE during a 40 min walk in the heat chamber in 50 ºC and 10% RH.

In total 120 temperature measurements were taken from each type of equipment. Intra-class correlations (ICC) with 95% confidence intervals (95% CI) were calculated between each method (Table 3.7.). Bland-Altman plots were also generated with LOA, with the differences between the two methods plotted against the respective temperature means. A line of equality graph was also produced (Figure 3.2.).

Table 3.7 *Mean ± standard deviation (SD), typical error of the measurement (TEM), TEM as a coefficient of variation (TEM (CV %)) and intra-class correlation coefficient (ICC) with 95% confidence intervals (CI) between skin temperature measured from thermistors compared to iButtons.*

	TEMPERATURE (°C)	
	THERMISTOR	**IBUTTON**
MEAN ± SD	36.61 ± 2.02	36.77 ± 2.11
ICC (95% CI)	0.987 (0.979 - 0.992), p < 0.001	
MEAN BIAS (LOA)	-0.16 (0.70, -1.01)	

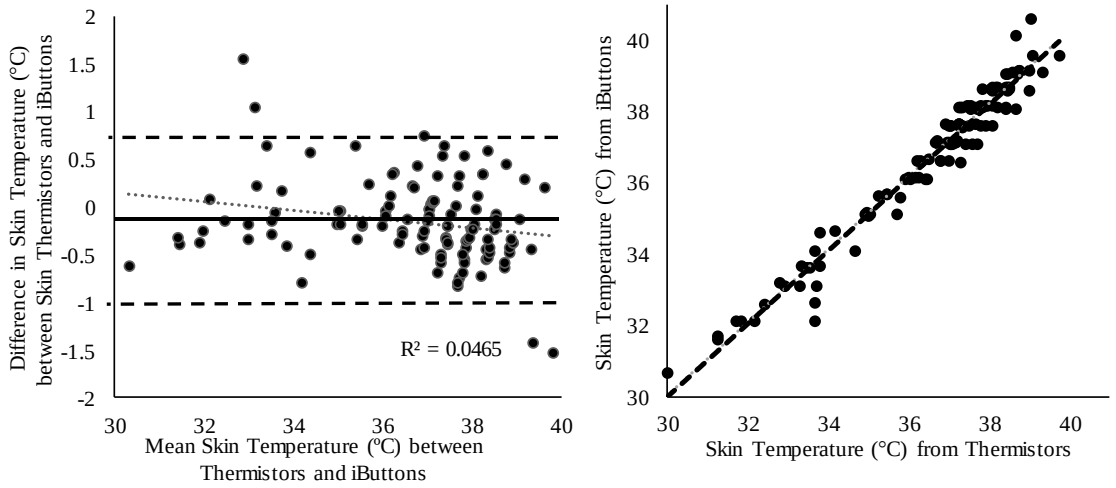

Figure 3.2 *Bland-Altman plot presented with mean bias and LOA (A) and a line of equality graph (B) for mean skin temperature (°C) between thermistors and iButtons.*

3.8.3 Metabolic gas analysis

Expired metabolic gas was measured via either manual Douglas bag collection when few samples were required, or by breath by breath online analysis (Metalyzer Sport, Metasoft Studio, Cortex, Biophysik GmbH, Leipzig, Germany).

Douglas bags (120L Douglas Bags, Havard Apparatus Ltd, Kent, UK) were fully evacuated prior to use. Participants wore a nose-clip and inserted a mouth piece connected to a 2 way valve (in house with diaphragms from MSA, Britain Ltd., Coatbridge, UK) and Douglas bag via a falconia tube (Baxter Woodhouse & Taylor Ltd., Cheshire, UK). Expired gas was collected for ~45s timed on a stop clock. Gas samples collected were analysed using a gas analyser (Servonex Xentra 4100, Servonex International Ltd, Crowborough, UK and Buhler Gas Sample Dryer, Type PKE4, Buhler Technologies GmbH, Ratingen, Germany). The gas analyser was calibrated using Nitrogen (N) and a mixture of known gases of oxygen (O_2) and carbon dioxide (CO_2) quantities (British Oxygen Company, UK). Samples were zeroed with 100% N, then set with 80% N, 15% O_2 and 5% CO_2, followed by 80% N 18% O_2 and 2% CO_2. Gas temperature and volume were then sampled using a vacuum pump and dry gas meter (Havard Apparatus Ltd, Kent, UK). Barometric pressure was also recorded. Resulting $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}e$ were calculated using the Haldane Transformation.

The breath by breath online gas analyser was calibrated prior to use following manufacturer's instructions. Calibration included adjustments for pressure, volume and gas sensors. Barometric pressure, from a portable weather station, was entered into the calibration software. Volume was calibrated via a manual syringe, with 5 acceptable cycles of a flow rate of 2 - 4 $L.s^{-1}$ required. According to the manufacturer's instructions, the flow sensor measured values to the accuracy of ± 2% with a range of 0.05 - 20 $L.s^{-1}$. Two different gases (20.93% O_2 with 0.03% CO_2 and 15.00% O_2 with 5.05% CO_2) were used to calibrate the O_2 sensor and CO_2 sensors. According to the manufacturer's instructions, the O_2 sensor and CO_2 sensors measured values to the accuracy of 0.1 Vol% with a range of 0 to 60% and 0 to 13% respectively.

Reliability of the Douglas bag and the Metalyzer $\dot{V}O_2$ and RER measures were calculated from the collection of 10 gas samples taken on two occasions from one individual at both rest and exercise (Table 3.8 and Table 3.9). Exercise performed involved 2 x 10 min walk on a treadmill in 50ºC 15% RH. Mean bias and LOA between $\dot{V}O_2$ and RER from the two methods during exercise are also provided in Table 3.10.

Table 3.8 *Mean ± standard deviation (SD), typical error of the measurement (TEM) and TEM as a coefficient of variation (TEM (CV %)) for repeated measures of VO_2 ($L.min^{-1}$) recorded via Douglas bags.*

	REST				**EXERCISE**			
	VO_2 ($L.min^{-1}$)		RER		VO_2 ($L.min^{-1}$)		RER	
	1	2	1	2	1	2	1	2
MEAN ± SD	0.20 ± 0.02	0.21 ± 0.02	0.81 ± 0.02	0.82 ± 0.02	0.66 ± 0.04	0.64 ± 0.04	0.77 ± 0.03	0.78 ± 0.02
TEM	0.01		0.02		0.03		0.02	
TEM (CV%)	4.14		2.42		4.75		2.39	

Table 3.9 *Mean ± standard deviation (SD), typical error of the measurement (TEM) and TEM as a coefficient of variation (TEM (CV %)) for repeated measures of volume of oxygen uptake ($\dot{V}O_2$) ($L.min^{-1}$) and respiratory exchange ratio (RER) recorded via the Metalyser.*

	REST				**EXERCISE**			
	VO_2 ($L.min^{-1}$)		RER		VO_2 ($L.min^{-1}$)		RER	
	1	2	1	2	1	2	1	2
MEAN ± SD	0.26 ± 0.04	0.24 ± 0.04	0.89 ± 0.03	0.91 ± 0.03	0.82 ± 0.03	0.76 ± 0.03	0.88 ± 0.01	0.91 ± 0.01
TEM	0.01		0.03		0.01		0.01	
TEM (CV%)	3.66		3.6		1.56		0.86	

Table 3.10 *Mean ± standard deviation (SD) and Bland-Altman mean bias with 95% LOA for volume of oxygen uptake ($\dot{V}O_2$) (L.min^{-1}) and respiratory exchange ratio (RER) collected via the douglas bag and metalyser methods..*

	$\dot{V}O_2$ (L.min^{-1})		**RER**	
	Douglas Bag	Metalyser	Douglas Bag	Metalyser
MEAN ± SD	0.65 ± 0.04	0.79 ± 0.04	0.78 ± 0.02	0.89 ± 0.02
MEAN BIAS (LOA)	0.14 (0.03 – 0.24)		0.12 (0.07 – 0.16)	

3.8.3.1 Metabolic Heat Production

Metabolic heat production (H_{prod}) was calculated as the difference between metabolic energy expenditure (M) and the external work rate (W) (as per section 3.5). H_{prod} is expressed relative to body mass in W.kg^{-1} (Cramer & Jay 2014)

M and H_{prod} was calculated using the equation below (Cramer & Jay 2014):

$$M(Watts) = \dot{V}O_2 \frac{\left(\frac{RER-0.7}{0.3} e_c\right) + \left(\frac{1-RER}{0.3} e_f\right)}{60} \; x\; 1000$$

$$H_{prod}\,(W.kg^{-1}) = (\dot{M} - W)/\, Body\ Mass$$

where e_c is the caloric equivalent per litre of oxygen for the oxidation of carbohydrates (21.13kJ) and e_f is the caloric equivalent per litre of oxygen for the oxidation of fat (19.62kJ).

3.8.4 Heart Rate

During laboratory experimentation HR was recorded using a Polar FT1 HR monitor affixed to the participant via a chest strap (Polar electro, Kempele, Finland), accurate during steady state conditions to ± 1% or ± 1 b.min^{-1}, whichever is larger, according the manufacturer's instructions.

In live fire scenarios HR was recorded continuously via downloadable HR monitors (Polar RS800 and Polar Team^2 System [Polar Electro, Oy, Kempele, Finald]), accurate to ± 1% or ± 1 b.min^{-1}. HR monitors were affixed to the participant via a chest strap. The RS800 watches remained in an inside tunic pocket to prevent malfunctions due to the heat. Data were downloaded via the Polar ProTrainer 5 or Team^2 software post exposure.

3.8.5 Physiological Strain Index

Physiological strain index (PSI) was calculated based on changes in HR and T_{re}, using an equation by Moran et al. (1998):

$$PSI = 5(T_{re}t - T_{re0}) \times (39.5 - T_{re0})^{-1} + 5(HRt - HR_0) \times (180 - HR_0)^{-1}$$

where $T_{re}t$ and HR*t* were simultaneous measurements taken every 5 min during the exposure and T_{re0} and HR_0 represent baseline states.

3.8.6 Sweat Rate

Towel dried nude BM was recorded to the nearest 0.01 kg using Adam GFK 150 digital body scales (Adam Equipment Inc., Connecticut, USA) before and after heat exposure. Fluid intake was restricted throughout and values were corrected for urine output. During live fire scenarios participants were allowed to consume water, in accordance with Fire and Rescue Service safety guidelines, however water consumption was measured and BM values were correct accordingly. SR was calculated by:

$$\text{Sweat rate } (L.hr^{-1}) = (\text{Body mass pre (kg)} - \text{Body mass post (kg)}) / \text{Time (minutes)} * 60$$

3.9 Haematological measures

Venous bloods samples were drawn from the antecubital fossa prior to and post experimental sessions. A 10ml whole blood sample was collected using a 21G hypodermic needle (BD, Microlance 3, BD Infusion Therapy AB, Helingborn, Sweden) and a 10ml syringe (BD Plastipak syringes, Becton & Dickinson, UK). Samples were then divided equally into 5ml tubes containing EDTA anticoagulant (Starstedt Ltd, Leicester, UK). All whole blood samples were analysed within 2 hours and then centrifuged (Eppendorf Refrigerated Centrifuge Model 5804R, Stevenage, UK) at 4,500 rpm for a period of 10 min at 4°C to separate plasma. Plasma was pipetted (3ml Pasteur Pipettes, Western Laboratory Service, Hampshire, UK) into 1.5ml microtubes and stored at -86ºC (Sanyo Ultra Low MDF-U50V, VIP Series, Sanyo UK, Watford, UK) until analysis.

3.9.1 Whole Blood Analysis

Whole blood samples were assessed using an automated haematology analyser (XT2000i, Sysmex, UK). The analyser uses fluorescent flow cytometry and hydrodynamic focusing technologies, to identify leukocyte counts and leukocyte content, including differentiation between neutrophils, eosinophils, basophils, lymphocytes and monocytes. Leukocytes are labelled with fluorescent polymethine dyes, which revealed the nucleus-plasma ratio of each stained cell, allowing for differentiation. Cells were classified based on the combination of side scatter, based on the inner complexity of the cell, forward scatter,

determined by cell volume and fluorescence of nucleic acid material. Erythrocyte number, platelet number and volume are also calculated. Erythrocytes and platelets were counted using direct current detection with hydrodynamic focusing to minimise recirculation. The haematocrit was then directly determined based on erythrocyte detection. Prior to each testing session a quality control check was conducted, using e-CHECK Level 2 control blood (Sysmex, UK), with analysis compared to the expected reference ranges. All values were corrected for changes in plasma volume (ΔPV%), which were estimated from haemoglobin and haematocrit (Dill & Costill 1974):

$$BV_A = BV_B\ (Hb_B/Hb_A)$$
$$CV_A = BV_A\ (Hct_A)$$
$$PV_A = BV_A - CV_A$$
$$\Delta\ BV\ \% = 100\ (BV_A - BV_B) / BV_B$$
$$\Delta\ CV\ \% = 100\ (CV_A - CV_B) / CV_B$$
$$\Delta\ PV\ \% = 100\ (PV_A - PV_B) / PV_B$$

where Hct is haematocrit, Hb is haemoglobin, BV is blood volume, CV is red cell volume and PV is plasma volume, with subscript B and A referring to before and after dehydration.

3.9.1.1 Whole Blood Analysis Reliability

Reliability of Sysmex complete blood count was assessed by duplicate analysis of the same blood sample collected from 11 male participants (age: 34 ± 8 yrs, height: 180.9 ± 4.5 cm body mass: 83.8 ± 16.0 kg). TEM and TEM (CV%) were calculated for each variable, see Table 3.9.

3.9.2 Enzyme-linked Immunosorbent Assay

Total plasma concentrations of IL-6, IL-1β, TNFα, CRP and IgG were determined using quantitative sandwich ELISA kits provided by R & D Systems (Minneapolis, MN). All samples and provided standards were analysed in duplicate. A standard curve was constructed using standards provided in the kits and the cytokine concentrations were determined from the standard curves using linear regression analysis. Microtiter 96 well plates were coated with the capture antibody and left to incubate overnight. The wells were then washed and a blocking buffer applied, before another wash commenced. Samples and standards were then added to the plate and incubated. Following incubation the wells were washed, a detection antibody was added and then left for another incubation period. Plates were then washed and horseradish peroxidase added, then incubated. A further wash followed, then a substrate solution was pipetted into the wells and a final incubation occurred. After incubation a stop solution was added to the wells. The plates were then read at the appropriate wavelength on a plate reader (BioTek Universal Microplate Reader ELX 800, BioTek UK, Bedfordshire, UK). Intra and inter assay coefficient of variation for each plasma variable is presented in the relevant chapters.

Table 3.11 *Mean ± standard deviation for sample 1 and 2, typical error of the measurement (TEM) and TEM as a coefficient of variation (TEM (CV %)) for repeated measures of white blood cells (WBC), red blood cells (RBD), platelets (PLT), neutrophils (NEUT), eosinophils (EO), basophils (BASO), lymphocytes (LYMPH), monocytes (MONO), haemoglobin (HGB), haematocrity (HCT).*

	WBC $x10^9.L^{-1}$	**RBC** $x10^{12}.L^{-1}$	**PLT** $x10^9.L^{-1}$	**NEUT** $x10^9.L^{-1}$	**EO** $x10^9.L^{-1}$	**BASO** $x10^9.L^{-1}$	**LYMPH** $x10^9.L^{-1}$	**MONO** $x10^9.L^{-1}$	**HGB** $g.dL^{-1}$	**HCT** %
SAMPLE 1	6.19 ± 1.12	5.07 ± 0.42	227.9 ± 40.0	3.39 ± 0.76	0.28 ± 0.65	0.18 ± 0.19	1.87 ± 0.80	0.48 ± 0.21	15.57 ± 1.34	44.24 ± 2.87
SAMPLE 2	6.20 ± 1.08	5.11 ± 0.36	230.3 ± 37.5	3.38 ± 0.70	0.28 ± 0.64	0.19 ± 0.21	1.86 ± 0.79	0.49 ± 0.21	15.77 ± 0.99	44.66 ± 2.19
TEM	0.12	0.08	6.2	0.12	0.01	0.02	0.07	0.05	0.55	0.78
TEM (CV%)	1.98	1.66	2.7	3.6	4.54	9.92	3.73	10.08	3.49	1.75

3.10 Perceptual Scales

Rating of perceived exertion (RPE) (Borg 1982) was recorded at rest, every 10 min during laboratory testing and prior to and immediately post live fire exposures. Thermal sensation (TS), a scale from 0 "unbearably cold" to 8 "unbearably hot" with 0.5 integers (Gagge et al. 1969), was recorded immediately following RPE. Verbal anchoring was given for top, middle and bottom ratings. A Heat Illness Symptoms Index (HISI) was also asked prior to and post heat exposure during studies with direct FSI involvement. Participants were asked to score a range of symptoms from 1 " no symptoms" to 10 "had to stop due to symptoms" (Coris et al. 2006) (Figure 3.3). In total 13 symptoms were asked, giving a maximum possible score of 130, symptoms are given in Appendix 1.

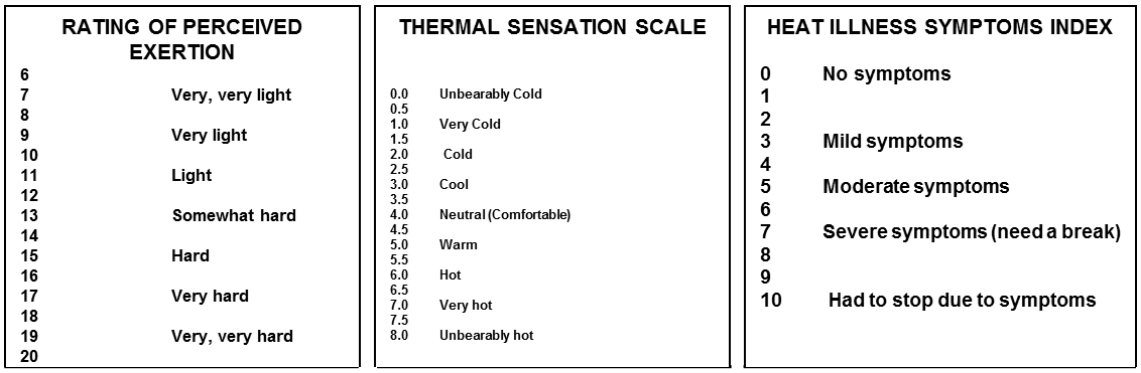

Figure 3.3 *Perceptual scales of rating of perceived exertion, thermal sensation and heat illness symptoms index.*

3.11 Statistical analysis

Data were analysed using a statistical package (IBM SPSS Statistics version 22) and reported as mean ± standard deviation (SD) unless otherwise stated.

3.11.1 Central Tendency

Unless otherwise stated in the study chapter, central tendency of a data set is expressed as the mean. This is calculated from the sum of the data, divided by the number of data points and expressed in unit of the measure.

$$\text{Mean} = (\Sigma\, xi) / n$$

where xi is the data and n the number of data points within the analysis.

3.11.2 Variation of Data Sets

Standard deviation (SD) is used to describe the variation within a data set. The variation of the data describes the distribution of data around the mean, with a small variance indicating little spread of data. One SD is provided alongside the mean to describe 68.2% of the normal distribution of the data set. SD is calculated from the square root of variance.

$$SD = \sqrt{[\Sigma (xi - X)^2 / (n - 1)]}$$

where xi is each individual data point, x is the mean of the data set and n is the number of data points.

3.11.3 Reliability Statistics

3.11.3.1 Typical Error of the Measurement

TEM was used to calculate the absolute reliability of measurements. TEM was determined from the SD of the difference score between repeated measures divided by the square root of two.

$$TEM = SD_{diff} / \sqrt{2}$$

where SD_{diff} is the standard deviation of the difference between two repeated measures.

TEM is also presented in relative terms as a coefficient of variation, reflecting the TEM as a percentage of its respective mean.

$$TEM\ (CV\%) = TEM/((X_1 + X_2)/2)*100$$

where X_1 is the first mean and X_2 is the second mean.

3.11.3.2 Intra-class Correlation Coefficient

ICC with 95% confidence intervals were conducted to assess the absolute agreement between two measures. An ICC of > 0.90 was classified as a high correlation, 0.70 - 0.80 as moderate and below 0.70 as low (Vincent & Weir 1995).

3.11.4 Normality of Data

Data was checked for normal distribution using the Shapiro-Wilk test, with a non-significant findings (p > 0.05) indicating that the distribution of data was not significantly different a normal distribution.

3.11.5 Sphericity of Data

Mauchly's test was used to assess sphericity. If the findings of the test were not significant ($p > 0.05$), it was concluded that there were no significant differences between the variances of the differences between conditions. In the case of a significant test with a sphericity estimate of ≤ 0.75 the Greenhouse-Geisser correction was used for interpretation of the ANOVA, whilst a sphericity estimate of > 0.75 warranted used of the Huynh-Feldt correction. These correction factors were applied to the degrees of freedom used to assess the observed F-ratio (Field 2013).

3.11.6 Statistical significance level

Statistical significance was set at an alpha level of 0.05, therefore p values below this value were classified as significant. With this alpha level the confidence interval is 95%, suggesting that 5% of the time a true null hypothesis may be incorrectly rejected, a Type I error, a false positive. The alpha level was set at 0.05 to control the Type I error rate.

3.11.7 Parametric Data

3.11.7.1 Correlations

Bivariate correlations to establish Pearson's correlation coefficients were performed to give a measure of linear correlations between two variables. The correlation coefficient (r) provides detail on how strong a relationship between two variables is. A weak relationship is represented by $r < 0.03$, a moderate relationship by $0.3 \leq r < 0.5$ and a strong relationship by $r > 0.5$ (Cohen 1988).

3.11.7.2 T-tests

Differences between two sets of normally distributed data were analysed using independent or paired samples T tests. The T test determines if the mean of a group is significantly different from the mean of another group. The T-ratio produced offers the relationship between the differences in means to the variability in the data and the sample size.

3.11.7.3 Analysis of Variance

To analyse normally distributed data with multiple experimental groups and analysis of variance (ANOVA) was conducted. The F statistic is a ratio of the variability of two sets of data, it takes into account the variability of each group mean from the overall mean and the variance within each group from the group mean. A significant effect or interaction within an ANOVA was followed up using Bonferroni correction to manipulate the level of significance for individual tests. This was conducted to combat the familywise error rate that can occur with multiple comparisons. Consequently, the type I error rate during follow up tests remained at 5%.

3.11.7.4 Effect Sizes

To identify the size of the difference between means, effect sizes are used. For comparison between two groups of data Cohen's *d* is provided, with d_s used for independent groups and d_z used for repeated measures designs as per Lakens (2013). Cohen's d considers the difference between means and the pooled standard deviation of the means (Lakens 2013). Cohen's *d* can be interpreted as small (*d* = 0.2), medium (*d* = 0.5), or large (*d* = 0.8) (Cohen 1988).

$$d = (X_1 - X_2) / SD$$

Comparison for data involving multiple independent variables used the effect size of partial eta squared (η_p^2), which indicates the degree of association between the variance in the dependent variable and the independent variable in question. This effect size represents the sum of the square of the effect in relation to the sum of the square of the effect and the sum of the square of the error. The sum of the squares represents the sum of the squared differences of each observation from the mean. SPPS 22 was used to conduct this calculation.

3.11.8 Non- Parametric Data

Data of a categorical nature, or data that were not normal distributed, were analysed using non-parametric statistics. To identify differences between two independent groups a Mann-Whitney U test was conducted. This test assigns rank order to data to determine if two samples are likely to have come from the same population. To identify differences between paired samples a Wilcoxon signed-rank test was conducted, whereby observed differences were ranked in size order and the sum of positive and negative ranks assessed for deviation from a median of zero.

Relationships between variables of a non-parametric nature were assessed via Spearman correlation coefficients or point biserial correlations. The Spearman correlation coefficient measures the strength between two ranked variables. Point biserial correlations were used to identify relationships between two variables, when one of the variables was categorically dichotomized.

Pearson's chi square analysis was conducted to identify if observations based on two or more variables were independent of each other. If > 20% of expected frequencies within each category were less than 5 then a Fisher's exact test was selected instead. Odds ratios with 95% confidence intervals were subsequently conducted to identify the association between a variable and an outcome.

4 STUDY 1: FIRE SERVICE INSTRUCTORS' WORKING PRACTICES: A UK SURVEY

4.1 ABSTRACT

OBJECTIVES: Due to frequent heat exposure FSI are at risk of EHI, cardiovascular events and physiological and immunological stress similar to overtraining. Analysis of the symptoms experienced and working practice of FSI will inform interventions aimed at minimising health risks. The aim of this study was to collect data on FSI symptoms and practice compared to operational FF.

METHODS: Online surveys were distributed via email to UK FSI and FF. Categorical data responses reported as frequencies and percentages and Pearson's chi square analysis conducted to measure the associations between variables, with significance set at $p < 0.05$.

RESULTS: One hundred and thirty UK FSI (age: 43 ± 7 yrs, time in job: 5 ± 5 yrs) and 232 FF (age: 41 ± 8 yrs, time in job: 16 ± 8 yrs) responded to the surveys. BA training was the most common heat exposure experienced, 39 (35%) FSI completed 6 - 10 exposures in the previous month. Most FF (92%) completed ≤ 5 fire exposures in the previous month. The number of exposures FSI experienced ranged from 2 - 10 per week. Fifty (45%) FSI had no limit to numbers of exposures. Few FSI followed hydration guidelines, or pre or post cooling methods. New symptoms of ill health were reported by 45 (41%) FSI: fatigue, headaches, broken sleep and heart palpitations, and 48 (21%) FF: back pain and post-traumatic stress disorder. FSI with ≥ 11 BA wears per month were 4.5 times, $p = 0.041$, more likely to experience new symptoms, with those completing > 9 wears altogether 9 times more likely, $p < 0.001$.

CONCLUSION: A large proportion of FSI are experiencing new symptoms of illness after starting their career and whilst guidelines on exposure and hydration may exist, they are not universally in place to reduce the risk of future health problems.

4.2 INTRODUCTION

FSI are responsible for training newly recruited and operational FF (section 2.2). Instructors who teach on BA and CFBT courses experience wears on a regular basis. Acute wear exposure can cause high levels of physiological and perceptual strain, due to the extreme environment, heavy protective clothing worn and physical activity completed (Petruzzello et al. 2009; Watt et al. 2016) (section 2.3).

Frequent fire exposure may put FSI at risk of cardiac-related events, which are the highest cause of death amongst FF, accounting for 56% of US FF deaths in 2014 (Fahy et al. 2015) (section 2.5). Numerous inflammatory markers, such as IL-6, platelet number and CRP, have been documented to increase following fire exposure (Walker et al. 2015). Repeated wears may therefore have a chronic effect on these markers (Watt et al. 2016), potentially increasing an instructor's risk of a cardiac event. During an acute wear FSI are also at risk of suffering from an EHI (Eglin et al. 2004) (see section 2.4). In severe cases EHI can lead to organ failure and mortality (Casa et al. 2012).

With appropriate preparation and recovery from each wear the risk of cardiac events and EHI could be reduced (section 2.4.2). The latest "Health Management of Training Centre Instructor's" report suggests that individuals drink 1.5 times the amount of fluid lost during heat exposure, with case study examples of ~750 – 800 mL prior to exposure, ~400 – 1000 mL taken on during the activity and 1000 mL post exposure given (Chief Fire Officers' Association 2015). The report also suggests a minimum of 2 hr between exposures, with cooling methods such as ice slurry, forearm cooling, dress down procedures and wearing ice vests all recommended (Chief Fire Officers' Association 2015). There is no clear guidance on wear limits, only that it is important to instigate a method to safeguard instructors' health (Chief Fire Officers' Association 2015). However, it is unknown if instructors in the UK have been directly provided with and are following this advice.

As a result of repeated wear exposures recent research suggests FSI may be at risk of an overtraining like syndrome (Watt et al. 2016) (see section 2.6). Overtraining symptoms have previously been documented in Army recruits during training courses (Booth et al. 2006), with prevalence amongst special military unit training suggested to be 24% of participants (Chicharro et al. 1998). The prevalence of overtraining varies greatly amongst athletes, from 10 - 64%, with numbers altering based on the level of physical demand required for the sport and the duration of participation (Raglin et al. 2000; Kentta et al. 2001; Gustafsson et al. 2007; Matos et al. 2011; Birrer et al. 2013). However, it is important to note that there is no single universally used diagnostic tool for overtraining, which may influence the prevalence rates reported (Halson & Jeukendrup 2004). Amongst FSI, symptoms of overtraining have only been anecdotally reported. For FSI an overtraining like syndrome may not only be detrimental to their health, but also to their occupational functionality, with loss of coordination, difficulty concentrating and a reduced maximum work capacity. It is therefore important to identify the type and frequency of symptoms and illnesses experienced by FSI to form the basis for further investigation into the proposed overtraining response that repeated wear exposures may cause.

Therefore, the purpose of the present study was to document the working practices of FSI in comparison to FF, from across the UK, with specific reference to details of: wear types completed, preparation and recovery methods used and illnesses and symptoms experienced. A secondary objective was to identify associations between new symptoms reported by FSI and the number of wears they completed, if they follow hydration guidelines and their perception of recovery. It was hypothesized that (1) the majority of FSI would not use pre or post cooling techniques, but would use hydration guidelines, (2) a greater number of FSI would report symptoms of ill health and (3) presence of ill health would be associated with exposure numbers.

4.3 METHOD

To establish the common demographics and working practices of instructors within the UK, in comparison to FF, both groups were invited to take part in an online survey. The survey was advertised across the UK via direct emails and social media. Those who responded to the survey and left their email address were contacted to ask them to further distribute the survey to their colleagues. The survey was also distributed upon visits to UK Training Centres and Fire Stations. All participants were informed that individual responses would be kept confidential and therefore would not be provided to their superior colleagues. The study was approved by the University of Brighton ethics committee.

Prior to the survey a focus group was held with FSI from the "Health Management Research Project for Live Fire Instructors" to shape the direction of the questions and ensure the content was appropriate for the desired population. The focus group also identified the types of wears instructors complete, these were listed as: BA, CFBT and BA cold/no smoke exposures. The focus of BA cold/no smoke and BA wears are for the students to be familiar with and learn how to use the equipment, whilst CFBT focuses on understanding how fire behaves and how to extinguish it. BA cold/no smoke wears involve learning how to use BA equipment in a temperate environment, however it should be noted PPE is worn and therefore elevated T_c may still occur. BA wears involve exposures to high environmental temperatures with instructors responsible for following teams of students to coach them when required. CFBT uses controlled live fires, where instructors coach students or remain outside the fire unit to control the environment with ventilation. Full details of the aim of exposures can be found in the SFJ Awards Qualification Handbook (SFJ Awards 2017). Preparation and recovery methods used at training centres were also suggested.

4.3.1 The Survey

Following the focus group a survey was generated using an online survey tool (surveymonkey.com, California, Palo Alto, USA). The survey consisted of 6 sections, covering: demographics, wear types, preparation, recovery, illnesses and an option to give any additional comments. The survey was then piloted to four members of the Health Management Research Project who were asked to provide feedback. Feedback consisted of extending the time options for the hot wear durations, altering rank orderings to

match Fire Service Questionnaires (3 being the hardest, 1 being the easiest) and ensuring the correct jargon was used throughout.

After corrections the survey consisted of 37 questions (see Appendix 1 for survey questions). Details of respondents' age, gender and time as an instructor were gathered, alongside how often they completed each type of wear and which wear they found the most physically challenging. Questions orientated around preparation and recovery included: whether they had a routine, what methods they used and how long they prepared/recovered for. Instructors were also asked how often they were ill, what type of symptoms they experienced and if they felt comfortable requesting sick days. Language used throughout the survey was non-biased, with neither positive nor negative phrasing of the introduction and questions.

A shorter version of the survey, containing only 10 questions, was designed for FF. The questions were taken from the demographic, wear frequency and illness sections of the instructor survey. The surveys were live for 12 months.

4.3.2 Statistical Analysis

Demographic quantitative data is displayed as mean ± standard deviation (SD). A Mann-Whitney U test was conducted to analyse differences in interval data between FF and FSI when normal distribution assumptions were violated, as determined by a Shapiro-Wilk test (section 3.11). Categorical data were analysed as described in section 3.11.8. Prevalence of categorical data responses are reported in frequencies and percentages, with Pearson's chi square analysis performed to assess if the distribution of categorical variables differ from one another in relation to those who reported new illnesses and symptoms and the number of wears they completed, whether they followed hydration guidelines and their perception of recovery post wear. Subsequent odds ratios and 95% CI were calculated to analyse associations. Significance level was set at $p < 0.05$ (section 3.11.6). Qualitative responses to open questions were categorised into key themes, with a minimum of three statements per theme.

4.4 RESULTS

The survey was completed by 130 FSI and 232 FF; response numbers for individual questions vary, as questions that did not apply could be skipped. Table 4.1 details the demographic of responders. All participants completed the survey within two days of initial engagement with the questions. Fisher's exact tests revealed that FSI responses did not differ by time of year of survey completion for reported wear numbers (BA: $p = 0.81$, CFBT: $p = 0.159$, BA cold/no smoke: $p = 0.464$) or new symptoms of ill health ($p = 0.296$).

Table 4.1 *Demographic details of both Fire Service Instructor and Firefighter survey responders.*

	Fire Service Instructor	Firefighter
Age Mean (yrs)	43	41
Age Standard Deviation (yrs)	7	8
Time as FSI Mean (yrs)	5	N/A
Time as FSI Standard Deviation (yrs)	5	N/A
Number of Male Participants	124	215
Percentage of Male Participants (%)	95%	93%
Number of Female Participants	6	17
Percentage of Female Participants (%)	5%	7%

FSI = fire service instructor

4.4.1 Wear Types

The frequency of wear types completed by FSI in the previous month is presented in Figure 4.1. Overall the median number of wears completed by FSI (± interquartile range) was 13 ± 8, with the minimum number experienced being 0 and the maximum being 40. When broken down into wear types, FSI completed 5 ± 6 BA wears, 3 ± 5 cold/no smoke wears and 3 ± 4 CFBT wears. The mode duration of a BA wear was 26 - 30 min reported by 24 (23%) FSI, 36 - 40 min for CFBT reported by 21 (20%) FSI and 26 - 30 min for BA cold/no smoke reported by 22 (23%) FSI.

The median number of live fire wears experienced by FF was 1 ± 3, with 214 (92%) FF having completed ≤ 5 wears in the previous month. Only 17 (13%) FSI completed ≤ 5 wears in a month.

When asked to order the wear types by how physically straining they found them, 60 (59%) reported that CFBT generated the greatest strain and 78 (76%) FSI selected BA cold/no smoke as the least straining, see Table 4.2.

Table 4.2 *Ranking of wears in order of perceived physiological challenge, 1 being the easiest to complete and 3 being the hardest. N/A for those that did not complete that wear type.*

Type of Wear	**Perception of Physiological Challenge**			
	1	2	3	N/A
BA live fire	9	66	24	1
BA cold/no smoke	78	10	17	3
CFBT	16	32	60	2

BA = breathing apparatus, CFBT = compartment fire behaviour training

Of the respondents 39 (35%) FSI thought that they completed too many wears in a week and 50 (45%) reported that they were unaware of any wear limit set by management. Of those who did have a limit (62, 55%), it ranged from 2 - 10 per week. The only commonly cited limit was 3 a week, 9 a month.

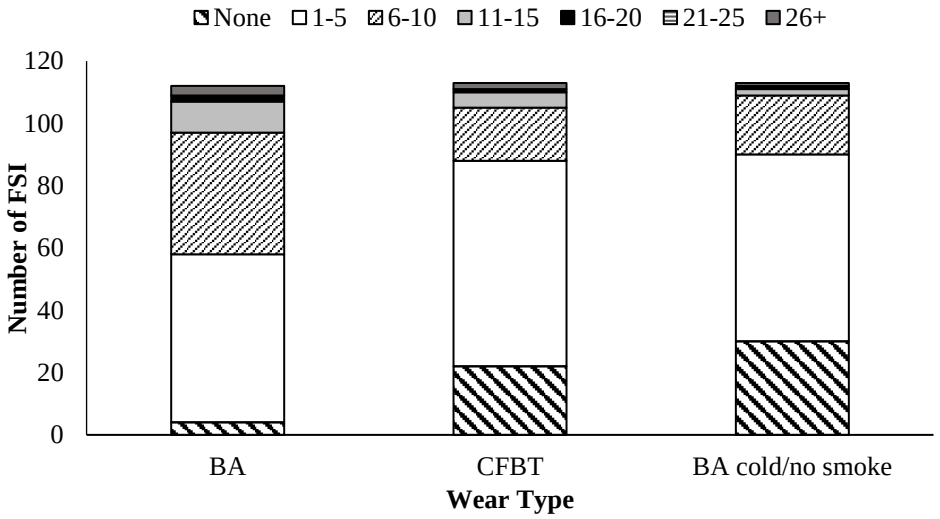

Figure 4.1 *The number of FSI who completed each frequency of wear types during a one month period. FSI = fire service instructor, BA = breathing apparatus, CFBT = compartment fire behaviour training.*

4.4.2 Preparation for a Wear

The majority of FSI, 79 (73%), were not allocated a specific time period to prepare for a wear. FSI reported that prior to a BA or CFBT wear 69 (68%) checked their hydration status, 10 (10%) drank ice slurries, 2 (2%) wore an ice vest, 6 (6%) wore a phase change vest, 1(1%) placed their forearms in cold water buckets and 36 (35%) had no preparation method, see Figure 4.2. The alternative methods reported by 20 (20%) FSI were grouped into themes and included: drinking water, checking tympanic temperatures and using rehydration sachets. Some FSI (15, 15%) used multiple methods of preparation, with the most common combinations being hydration checks with wearing a phase change vest (FSI: 6, 6%) and hydration checks with ice slurry consumption (FSI: 5, 5%). Having no hydration guidelines was reported by 44 (41%) FSI. Those with guidelines reported using visual charts to check urine colour and specific advice from within their service on water consumption.

4.4.3 Recovery from a Wear

Seventy-six (70%) FSI also stated that they do not have a set allocated recovery time; FSI from organisations with a specified recovery time had a minimum of 2 hrs between wears allocated. However, 18 (55%) of those with a set recovery time reported having an actual average recovery period of less than 2 hours. Average recovery periods ranged from 5 min to 2 hrs, with 31 (32%) FSI having ≤ 15 min and 44 (45%) having > 30 min. Those that had > 30 min reported having additional tasks to do in that time, including debriefing students, paperwork and eating lunch. When asked how they recovered from a hot wear, FSI responses were: 6 (6%) drank ice slurries, 79 (78%) drank water, 20 (20%) placed their forearms

in cold water buckets and 9 (9%) had no recovery routine, Figure 4.2. Themes of alternative methods included using rehydration sachets and dress down procedures. Some FSI (24, 24%) used multiple methods of recovery, with the most common combinations being drinking water with rehydration sachets (FSI: 8, 8%) and drinking water with forearm cooling (FSI: 9, 9%).

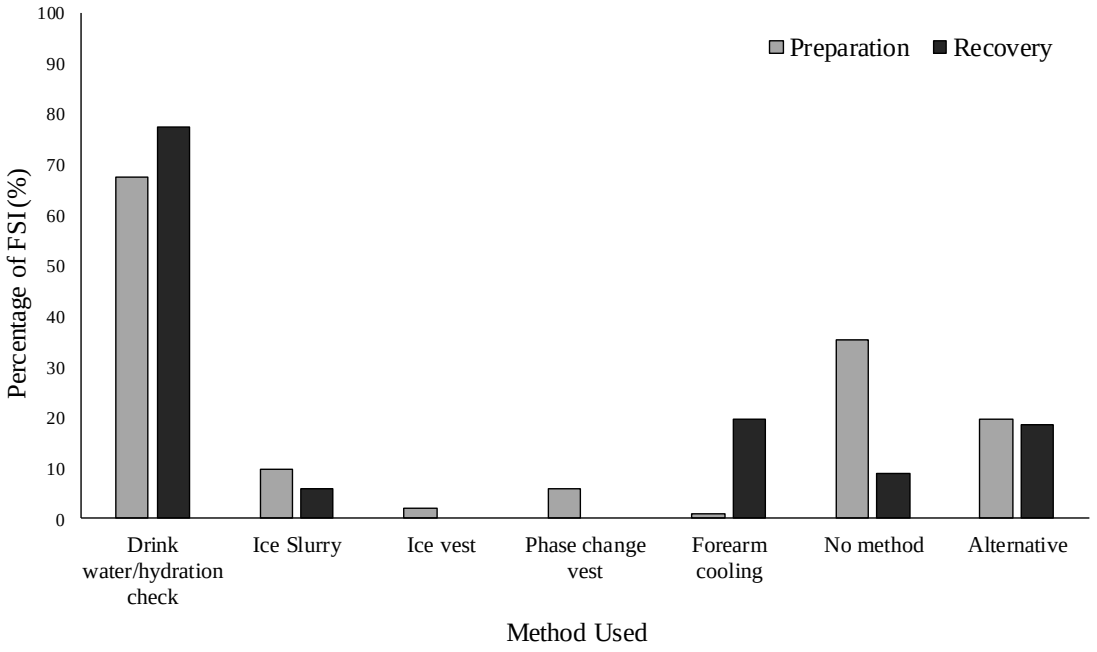

Figure 4.2 *The percentage of FSI using different methods during the preparation and recovery periods prior to and following a wear. The preparation graph represents 102 responders with 144 method responses; the recovery graph represents 102 responders with 133 method responses. FSI = fire service instructor.*

Only 3 (3%) FSI reported that they felt fully recovered after their normal recovery period post wear, with the majority, 71 (65%), stating they were only just recovered, feeling worn out and warm. A further 9 (8%) FSI were exhausted and hot after a wear and did not feel recovered.

4.4.4 Illness

Over the previous year FF reported taking a greater number of sick days compared to FSI (3 ± 7 vs. 1 ± 3, respectively), $U = 18446$, $p < 0.001$. However, 49 (45%) FSI also reported that they did not feel comfortable reporting illness at work and taking sick leave. Participants were asked to recall periods of cold or flu over the last year, with 27 (25%) FSI having not suffered from a cold or flu, 60 (55%) had one or two occurrences, 20 (18%) had suffered 3 - 6 times, 1 (1%) had symptoms once a month and 2 (2%) suffering 2-4 times a month. In comparison, 51 (22%) FF reported no cold or flu occurrences, 134 (59%) had symptoms once or twice in the year, 40 (18%) suffered 3 - 6 times and 4 (2%) had a cold or flu once a month.

When FSI were asked if they frequently suffer from any other illnesses or symptoms that they did not experience prior to becoming an instructor, 45 (41%) said Yes. Some (48 (21%)) FF also reported suffering from a new illness or symptoms that they did not experience before becoming a FF.

There was an association between the number of BA wears completed (≤ 5, 6 - 10, or ≥ 11 a month) and presence of new symptoms, χ^2 (2) = 6.37, p = 0.041. The odds of suffering from a new illness or symptom was 1.6 (95% CI 0.66 - 3.66) times higher for FSI who conduct 6 - 10 BA wears a month and 4.5 (95% CI 1.33 - 15.09) times more likely when completing ≥ 11 BA wears, compared to those who completed ≤ 5. There was also an association between the total number of wears completed (≤ 9 or > 9 a month) and presence of new symptoms χ^2 (1) = 20.30, p < 0.001, in FSI. The odds of suffering from a new illness or symptoms was 9 (95% CI 3 - 26) times higher in those completing > 9 wears a month, than those completing ≤ 9. There was no association between those suffering from new symptoms and those who had hydration guidelines, χ^2 (1) = 0.60, p = 0.691. How recovered FSI felt after their wear recovery (fully recovered, fine, worn out and warm, and exhausted) was associated with the presence of new symptoms, (p = 0.004, Fisher's Exact Test). Those who felt exhausted after a wear were 16.8 (95% CI 2.69 - 104.82) times more likely to suffer a new symptom than those who felt fine afterwards.

New symptoms reported by FSI were: fatigue (16), headaches (12), broken sleep (8), musculoskeletal pain (8), heavy sweating (6), problems thermoregulating (5), heart palpitations (4), blood shot eyes (3), mood swings (3) and coughing and breathing problems (4). The common themes from FF were: back pain (11), post-traumatic stress disorder (PTSD) or depression (7), coughing or breathing problems (10) and headaches (5). FSI and FF were also asked how they felt both physically and mentally at the end of a working week, results shown in Table 4.3.

Table 4.3 *Fire Service Instructor and Firefighter perception of how they feel, both physically and mentally, at the end of an average wearing week.*

	Nature of Illness	**Frequency of Feelings Post Wearing Week**					
		Ill	Exhausted	Tired	OK	Good	Great
Fire Service Instructor	Physical	2 (2%)	28 (26%)	65 (59%)	9 (8%)	6 (6%)	0 (0%)
	Mental	1 (1%)	22 (21%)	58 (54%)	17 (16%)	8 (8%)	1 (1%)
Firefighter	Physical	0 (0%)	28 (12%)	102 (20%)	47 (20%)	35 (15%)	18 (8%)
	Mental	3 (1%)	28 (12%)	96 (42%)	45 (20%)	37 (16%)	19 (8%)

4.4.5 Additional Comments

From the 39 (30%) FSI who left additional comments, the key themes were: frequently feeling fatigued (4), being worried about their health (4), concern over the number of wears (14), lack of fresh protective clothing (5), worry about breathing in contaminants (4) and support for hydration and cooling methods to reduce the acute effects of a wear (4).

4.5 DISCUSSION

The study aimed to document the working practices of FSI across the UK, to enable a better understanding of the frequency of heat exposures, the types of preparation and recovery methods used and the prevalence and type of illnesses experienced. FF were used as a comparative group, to identify if the symptoms or illnesses experienced were specific to FSI alone. The study also aimed to establish whether there was an effect of wear number, hydration advice, or perception of recovery on the presence of overtraining symptoms or illness. The number and duration of wears completed varied, however a larger proportion of FSI completed 6 - 10 BA wears a week than CFBT, with more FSI conducting no CFBT. The mode time for wear duration for BA and BA cold no/smoke was 26 - 30 min, whilst CFBT lasted slightly longer, 36 - 40 min. As expected due to their occupation, FSI completed a greater number of fire exposures than FF. The findings from this survey also suggest that not all FSI are provided with, or follow, the advice on hydration and recovery methods suggested in the research literature, consequently alternate hypothesis (1) can be accepted. Furthermore, the prevalence of new symptoms and illnesses since starting the occupation is high (41%), with prevalence potentially linked to high wearing numbers and poor recovery. Some FF also experience new symptoms (21%), although of a different nature to FSI. Both alternate hypotheses (2) and (3) can therefore also be accepted.

4.5.1 Wear Types

The variety of wear frequency and duration reported was as expected based on previous findings (Eglin et al. 2004; Watt et al. 2016). It is clear that whilst the majority of FSI have a limit to the number of wears they can complete, as recommended by the Chief Fire Officers Association guidance (2015), there is not a universal practice. A suggested example of good practice referred to by the Chief Fire Officers Association (2015), of 3 a week and 9 a month exposure limit, seems to be used by some services. There is large variation in the number of permitted wears set by each service, however there is currently no evidence based literature to support either the conservative or liberal prescription. Additionally, some FSI (4) report a very high limit (≥ 8 per week) of heat exposure frequency. This study also showed data that suggests FSI who complete > 9 wears a month are 9 times more likely to experience new symptoms or illnesses.

Repeated heat exposures may cause chronic inflammation in FSI, with elevated resting CRP (7378 ± 3770 $ng.mL^{-1}$) and IL-6 levels (11.4 ± 1.0 $pg.mL^{-1}$) previously documented (Watt et al. 2016). Inflammation plays a role in both the initiation and progression of atherosclerosis (Koenig 2013). Elevated levels of

inflammation, exacerbated by reduced recovery time between wears (Walker et al. 2015), suggests that FSI may be at an increased risk of a cardiovascular event as the frequency of heat exposures rises.

4.5.2 Preparation and Recovery

This study also highlights the need for additional hydration education, as 44 (41%) FSI were unaware of any hydration guidelines. This is despite guidelines indicating that 1.5 times the amount of fluid lost during exposure should be consumed (Chief Fire Officers' Association 2015). During a wear FSI have been reported to lose 1.28 $L.hr^{-1}$ of sweat, equivalent to 1% of their body mass (Watt et al. 2016) as the body attempts to dissipate heat gained from the environment and metabolic activity via sweating. However, some FSI carry out wears multiple times a day and consequently may become progressively hypohydrated (Hillyer et al. 2015). A reduction of body mass post exercise of $\geq$ 2% can cause decrements in both psychological and physiological performance, for example increasing decision making time and reducing fine motor skill accuracy (Hillyer et al. 2015; Masento et al. 2014). Consequently, this could lead to unsafe behaviour in the workplace for instructors and their trainees. However, no association was present between those who used hydration guidelines and FSI who reported new illnesses and symptoms. It may therefore be postulated that hydration status alone is not the reason for reporting of FSI ill health. Whilst hydration guidelines should be put in place and further education about guidelines is needed amongst FSI, this may not prevent the occurrence of new symptoms amongst FSI.

Pre-cooling may be a practical intervention available to FSI to reduce the physiological strain experienced during a wear and is recommended by national FSI guidelines (Chief Fire Officers' Association 2015). As FSI know in advance when fire exposure will occur they could utilise pre-cooling techniques in a systematic way that may not be possible for FF. The reduction of T_c prior to exercise in the heat via cooling methods is a well-established technique amongst athletes, as it can increase the body's heat storage capacity, enabling individuals to maintain their work intensity for longer, or reduce their end T_c (Ross et al. 2013). There are various pre-cooling methods available, including cold water immersion (Booth et al. 1997), ice jackets (Arngrïmsson et al. 2004) and ice slurry consumption (Siegel et al. 2012), which have been reported to reduce T_c prior to exercise by 0.3 - 0.7ºC (Ross et al. 2013). For each method there is a variation of T_c recorded in the literature due to the differences in doses and durations that cooling has been applied for. It is unclear which method of cooling is most beneficial for FSI, with specific advice on duration and dosage also currently not provided (Chief Fire Officers' Association 2015). Consequently, further research is needed into the use of practical pre-cooling methods for FSI, with the need for this research to be fed into FSI guidelines.

Additional information on the use of post cooling techniques should also be provided to FSI, as despite scientific literature supporting the use of various methods, little is being practically applied in the UK. Cheung et al., (2010) offers a comprehensive review of the use of recovery forearm immersion cooling for FF, highlighting that it is a promising practical countermeasure to the physiological strain experienced. A 20 min period of forearm cooling in 17ºC water may elicit a reduction in T_c of 0.5 ± 0.1 ºC (Selkirk et al. 2004). The use of 17ºC water enhances the practicality of the technique, as water from local supplies is

around this temperature and therefore no additional ice or chillers are required. However, a more recent review by Brearley & Walker (2015) reported that hand and forearm cooling results in an unacceptable cooling rate of < 0.07°C.min^{-1}. The authors suggested that multi segment immersion offers a more effective alternative and can elicit cooling rates of 0.10°C.min^{-1}, although this method may be logistically difficult for FSI. Ice slurries may offer a practical alternative to multi segment immersion, with cooling rates of 0.09°C.min^{-1} having been reported post live fire exposure, following consumption of 7 g.kgBM^{-1} of ice slurry in 15 min (Walker et al. 2014).

The responses to questions about recovery time in this survey highlight that few FSI have a period to rest and cool down, without other demands made of them. To discover what FSI view as a recovery period the questions in this survey were kept broad, asking only if they were allocated specific recovery time, the average recovery time experienced and an option to provide details. The questions did not specify what should occur in the recovery time. Responses indicate that the recommended 2hr minimum between heat exposures (Chief Fire Officers' Association 2015) is thought of by some FSI as their recovery period and can involve other tasks and often covers the lunch break period. However, FSI are often not receiving a full 2hr period between wears. Guidance for providing adequate time to rest immediately post a wear, when rehydration and cooling can occur, should be considered.

4.5.3 Illness

Prevalence of cold and flu symptoms amongst FF and FSI were similar, with 22% vs 25% not experiencing symptoms, 59% vs 55% having one or two occurrences and 19% vs 21% suffering more than 3 times a year. This is similar to the prevalence of cold and flu amongst a normal European population group have previously been reported with 25% hardly ever catching a cold, 50% experiencing symptoms once or twice a year and 21% suffering 3 - 4 times a year (Hull et al. 2013). This suggests that FF and FSI do not sufferer from a greater number of URTI.

A greater proportion of FSI reported experiencing new symptoms (41%) than FF (21%). Two symptoms crossed over both groups: headaches and coughing and breathing problems. FF have previously been reported to have a high prevalence of dyspnea, coughs, sinusitis and headaches (Mustajbegovic et al. 2001; Miedinger et al. 2007), alongside lower forced vital capacities (5.09 ± 0.68 L) and forced expiratory volume in one second (4.06 ± 0.56 L) compared to predicted values (5.26 ± 0.61 and 4.38 ± 0.54, respectively) $p <$ 0.05 (Mustajbegovic et al. 2001). FF have also been reported to have an increased risk of developing adult-onset asthma compared to police officers (OR = 1.23) (Ribeiro et al. 2009). Exposure to air pollutants, from combustion products, is the probable cause of these symptoms, as they have been documented to exacerbate respiratory symptoms and increase bronchial reactivity (Miedinger et al. 2007; Greven et al. 2011). Minimising exposure to "dirty" PPE and the use of effective decontamination processes, alongside good occupational hygiene practices, such as wearing BA when near smoke on the training ground and during overhaul situations, may reduce individual's exposure to toxic air contaminants (Fent et al. 2017). Future investigation into the practices used within the UK fire service, with reference to the effectiveness of different processes, could be warranted.

FF also reported experiencing back pain that had not existed prior to their occupation. Previous studies have documented that up to 44% of FF may suffer from back pain, with this percentage rising with time in service (Lusa et al. 2015). The additional weight carried by FF due to PPE and BA, may result is additional compressive forces on the spine during landing movements (Vu et al. 2017). Reduced ankle flexibility caused by restrictive firefighting boots may also be linked to back pain, with increased ground reaction forces reported and greater compressive forces in the lumbar region (Vu et al. 2017). Frequent heavy lifting and wearing PPE have also been reported to increase the odds (OR = 1.3) of suffering from lower back pain in the military (Roy & Lopez 2013). FF wear PPE to respond to all emergency calls in the UK and therefore may find themselves standing in full PPE for numerous hours at a time, for instance when attending a road traffic collision. Consequently, these prolonged periods of PPE wearing combined with manual tasks may explain why this symptom was prevalent amongst FF but not FSI.

The final symptoms reported only by FF were PTSD and depression. Operational FF are likely to see traumatic events, being the first responders to a larger variety of situations. Prevalence of PTSD amongst first responders ranges from 10 - 32% (Fullerton et al. 2004; Berger et al. 2012). Assisting survivors and exposure to fatally injured persons increases the likelihood of PTSD (OR = 2.98 and OR = 3.40, respectively) (Fullerton et al. 2004). Walker et al., (2016) suggest that FF may be predisposed to PTSD, due to chronic low grade inflammation they may suffer as a consequence of altered sleep patterns, high physical workloads, injuries and heat and smoke exposure. Whilst this study suggests that FF in the UK are not frequently exposed to fire situations, shift patterns and manual tasks could still be involved in predisposing FF to PTSD.

Amongst the new symptoms reported by FSI are those that have also been associated with overtraining. Insomnia (with and without night sweats), fatigue, mood swings and muscle pain as reported by the FSIs in this survey are all signs of an overtraining syndrome (Smith 2003). The combination of physical activity and cognitive task performance in extreme temperatures, can generate a highly stressful environment for FSI (Smith et al. 1997; Petruzzello et al. 2016), which is a key contributory factor to overtraining (Meeusen et al. 2013). Frequent high stress experiences may also lead to chronic inflammation, evident by increased IL-6 and have been linked to immune function dysregulation (Padgett & Glaser 2003). Suppression of cell mediated immune function has been hypothesised to be a leading factor in the development of overtraining syndrome (Smith 2003). It can therefore be postulated that repeated exposure, of up to 10 times a week as documented in this study, could result in FSI developing a syndrome similar to that of the overtraining syndrome.

The presence of some of the new symptoms reported by FSI could also impact on their safety whilst conducting wears. Sleep deprivation is a risk factor for EHI (Coris et al. 2004), with partial sleep disruption increasing heat straining during exercise in a hot environment when performed in the afternoon (Tokizawa et al. 2015). Sleep deprivation can also increase the risk of inflammatory disease, with markers of inflammation, such as IL-6, exhibiting a greater increase during exercise when individuals are sleep deprived (Abedelmalek et al. 2013). In addition, a 10-fold increase in IL-6 levels has been noted following

seven consecutive days of sleep deprivation (Gundersen et al. 2006). With IL-6 already elevated in FSI both at rest and post wear (Watt et al. 2016), sleep deprivation may be further exacerbating the rise.

FSI also reported experiencing heart palpitations, which although usually benign, can be caused by arrhythmias, which include supraventricular tachycardia, ventricular extrasystoles, or atrial fibrillation (Wannamethee et al. 1995; Abbott 2005). Suffering from an arrhythmia can increase the risk of sudden cardiac death (relative risk 3.2, CI 95% 2.0 - 5.3) (Wannamethee et al. 1995). Atrial fibrillation is also a predictor of cardiovascular events (rate ratio 1.8, CI 95% 1.3 - 2.5), with 66% of men with atrial fibrillation experiencing an event over a 20 year period, compared to 45% of asymptomatic men (Stewart et al. 2002). In addition, those with the condition have a 4 - 5 fold increased risk of embolic stroke (Kannel & Benjamin 2008). The possibly life threatening consequences of sleep deprivation and heart palpitations reported by FSI suggest that further investigation into the health of FSI is warranted, with determining methods and guidelines to reduce the incidence of these new symptoms of paramount importance.

4.5.4 Limitations

Whilst the study reports working practices of FSI compared to FF, it can only offer a snapshot of the UK, as not all UK Fire and Rescue Services responded to the survey. However, 33 training centres and 46 of 52 UK Services were represented in the survey. The total number of BA wears completed by FSI may be underestimated, as specific information regarding the completion of wears outside of instructing duties was not gathered. In addition, reports of illnesses were subjective, as verification by a medical professional was not obtained. Due to the subjective nature of these findings cause and effect cannot be confirmed between variables. Furthermore, participants were asked to give retrospective accounts of working practices and illnesses, which may have resulted in recall bias. The distribution of the survey via word of mouth may have caused some response bias toward those suffering ill health, as they may have been more inclined to make their voice heard. However to try and mitigate against this, wording of the title and introduction referred only to working practices, not health, and included non-biased language.

4.6 CONCLUSION

This survey is the first to document FSI working practices from different training centres in the UK. FF experienced few live fire situations in comparison to FSI. FSI who complete a greater number of BA wears may be at an increased risk of suffering from a new symptom or illness. Some services employ a wearing limit, which would appear to be justified good practice based on the findings of this study. Further research into how many wears can be conducted before negative health consequences develop is needed. In addition, further education on the importance of and how to remain rehydrated is required, although this may not be the cause of FSI illnesses. Research into the benefit of pre-cooling methods for FSI is also required, with the need for this information, and that of effective post cooling methods, to be better fed into FSI guidelines. Some FF (21%) are reporting new health issues, although of a different nature to FSI, with key concerns being back pain and PTSD. Overall, 41% of FSI are experiencing new health problems after becoming

instructors, with some of the symptoms reported increasing the risk of EHI and cardiovascular events. Consequently, this survey highlights the need for quantitative laboratory and field investigations into the health of FSI.

5 STUDY 2: THE ACUTE EFFECT OF LIVE FIRE EXPOSURES ON FSI

5.1 ABSTRACT

OBJECTIVES: The strain experienced by FSI from different types of wears is not well understood. This study aims to identify the physiological and perceptual response of FSI to three wear types: DEMO, ATTACK, COMPARTMENT, and the different roles performed: SETTER, INSTRUCTOR. The study also aims to assess the effect that different wear patterns over a day (BOX, MULTI, COMBINATION) have on immunological responses.

METHODS: Sixteen FSI (age: 41 ± 8 yrs, body mass: 83.7 ± 6.7 kg, height: 177.0 ± 6.7 cm) were recruited, with 10 FSI completing the three exercises. Physiological and perceptual measures were collected prior to and immediately post each exercise. Venous blood samples were collected at the beginning and end of each day. One way ANOVAs were conducted to assess differences in physiological variables between exercise types, independent samples t-tests were conducted between roles. Day changes in haematological variables were assessed by paired sample t-tests and analysed by one way ANOVAs to identify differences between exercise patterns.

RESULTS: The COMPARTMENT exercise resulted in a greater change in rectal temperature (ΔT_{re}) (0.49 ± 0.28°C) than both the DEMO (0.23 ± 0.19°C, p = 0.045) and ATTACK (0.27 ± 0.22°C, p = 0.016). Within the COMPARTMENT exercise, the SETTER resulted in a greater ΔT_{re} and rating of perceived exertion than the INSTRUCTOR (0.67 ± 0.29°C vs. 0.43 ± 0.18°C, p = 0.027 and 14 ± 2 vs. 11 ± 2, p = 0.001, respectively). Following a day of fire exercises WBC, NEUT, LYMPH, MONO, PLT, MPV, IL-6 and cTnT all increased ($p < 0.05$). Exercise patterns containing a COMPARTMENT exercise resulted in greater PLT, MPV and IL-6. Total daily variation in ΔT_{re} was correlated with post exercise WBC, MONO and LYMPH.

CONCLUSION: COMPARTMENT exercises produce the greatest physiological strain, with the SETTER role within this exercise causing the greatest $\Delta T_{re.}$ Consequently, the SETTER may be at an increased risk of heat illness, although predominately physiological responses remain within safe limits. Exercise patterns that include a COMPARTMENT exercise also generate a greater inflammatory response.

5.2 INTRODUCTION

The physiological impact of a live fire has been well documented in FF, with numerous detailed investigations conducted over the previous 30 yrs (see Table 2.1) (Romet & Frim 1987; Smith et al. 1996; Eglin 2007; Petruzzello et al. 2009; Cheung et al. 2010; Horn et al. 2013). It is widely established that the combination of physical exercise, PPE, BA, and extreme temperatures result in an uncompensable environment (section 2.3) (Cheung & McLellan 1998; Cheung et al. 2000). Consequently, skin temperature, HR, and T_c increase, in combination with perception of effort and thermal sensation. Ultimately this would result in heat stroke if an individual is not removed from the heat source (Cheung et al. 2010). During training activities a T_c upper safety limit of 39ºC is recommended by the Fire and Rescue Service (Chief Fire Officers' Association 2015; Her Majesty's Fire Service Inspectorate 2004). However, there is a paucity in research into the actual response exhibited by FSI, with only Eglin et al. (2004, 2005) and Watt et al. (2016) having used FSI as participants. Whilst the environment experienced may be similar, FSI perform different tasks to FF. FSI are responsible for the safety of FF, for instructing and training them and also for setting the conditions in which the training occurs. Consequently, FSI tasks include setting fires, moving pallets or boarding, opening vents, positioning dummies or teaching FF. Instructing often entails resting in cooler parts of the building to observe the FF (Eglin et al. 2004). Following a training exercise FSI have been reported to have an increase in T_c of 0.27 - 1.0°C with a maximum HR of 134 - 162 $b.min^{-1}$ (Eglin et al. 2004; Eglin & Tipton 2005; Watt et al. 2016), in comparison to increases of 1.4 - 3.2°C in T_c and maximum HR of 134 - 194 $b.min^{-1}$ recorded from FF (Smith et al. 1996; Smith & Petruzzello 1998; Walker et al. 2015). This suggests that FSI may be working at a lower intensity of physical activity than FF, perhaps due to the type of occupational tasks being performed. Nevertheless, as documented in Chapter 4 FSI experience fire conditions more frequently than FF, with FSI completing 13 ± 8 exercises a month compared to 1 ± 3 fires experienced by FF.

The work conducted by FSI is also multi-faceted, with different exercises used to teach different scenarios to FF. Chapter 4 identified that the majority of FSI (59%) felt that CFBT were the most physiologically challenging exposures. A CFBT course may include two types of fire exercises, a single box exercise and a multiple compartment fire search scenario. There are two types of single box exercises, a demonstration where trainees learn about fire behaviour (DEMO) and an exercise where trainees practice fire suppression (ATTACK). Single box exercises focus on training FF about fire behaviour and the best approaches to dealing with the situation (SFJ Awards 2017). A compartment fire search scenario exercise is designed to use the skills gained in the box exercises in a multi-compartment scenario, where FF may be unaware of the exact location of the fire and are asked to find and recover a dummy (COMPARTMENT). Eglin et al. (2004) alludes to the differences between these exercises, although few single boxes were conducted and therefore no direct comparisons between exercise types were made. The pattern in which these exercises are conducted also varies across training centres, with some conducting a DEMO and an ATTACK in a day (BOXES) and two COMPARTMENT exercises in a day (MULTI), and others performing a DEMO and ATTACK in the morning and COMPARTMENT in the afternoon (COMBINATION). During each type of exercise there are two active roles that FSI may conduct: a condition setter who sets and controls the fire environment (SETTER), or an instructor who is responsible for taking trainees through the exercise and

teaching (INSTRUCTOR). It is currently unknown if these exercise types or roles result in different levels of physiological strain, or what impact exercise patterns may have.

Live fires experienced by FF have been noted to stimulate an inflammatory response, with increases in IL-6, platelet numbers and significant leukocytosis occurring (Smith et al. 2004; Walker et al. 2015). These biomarkers have been suggested to be involved with an increased risk of cardiac events (see sections 2.5 and 2.7) (Ridker et al. 2000; Chu et al. 2010). Markers of cardiac muscle damage, cTnT and cTnI, have previously been noted to increase in FF following training fire exercises (Hunter et al. 2017). Immune disturbances following physical activity have also been suggested as the possible mechanism for the overtraining response (see section 2.6) (Smith 2000; Kreher & Schwartz 2012), which has been proposed to be present in some FSI (Watt et al. 2016). The high prevalence of new symptoms of ill health in FSI (41%) and the association of ill health with perception of recovery and wear numbers, as noted in Chapter 4, offers support to the overtraining findings of Watt et al (2016). However, the inflammatory findings of Watt et al. (2016) are yet to be repeated in a larger population size and the impact that exercise patterns may have on inflammatory responses is unknown. Being able to plan courses to minimise the physiological strain and inflammatory response experienced by FSI could in the long term reduce the risk of cardiac events and an overtraining like syndrome.

This study aims to identify the physiological and perceptual response of FSI to three different training exercises and the different roles performed within each exercise. The study also aims to assess the effect that different exercise patterns over a day may have on immunological responses and measures of myocardial damage. It was hypothesised that: (1) there will be a significant difference in T_{re} responses to different exercise, (2) there will be a significant difference in T_{re} responses to roles, (3) a day of exercises will result in an increase in inflammatory and cardiac risk markers, and (4) there will be a significant difference in inflammatory markers between exercise patterns.

5.3 METHOD

Sixteen FSI (age: 41 ± 8, body mass: 83.7 ± 6.7 kg, height: 177.0 ± 6.7 cm) were recruited from three training centres: East Sussex Fire and Rescue Service, Surrey Fire and Rescue Service, and the Fire Service College at Moreton-in-Marsh. Fourteen FSI were male, two were female. Participants gave informed written consent and completed a medical questionnaire (section 3.1).

Participants were requested to avoid caffeine 12 hours before each session, and alcohol and exhaustive exercise 24 hours before. Participant adherence was checked with a questionnaire completed before each session (section 3.1).

5.3.1 Experimental Design

Testing occurred for FSI at their respective training centres (section 3.3). A medical room or portable cabin was used as a base field laboratory to instrument participants and collect baseline measures. Participants completed heat exposures as per their normal job, with all exposures forming part of a taught course. Female

participants were monitored during exercises when they were in the early follicular stage of their menstrual cycle as ascertained via a self-reported menstrual cycle questionnaire (Pivarnik et al. 1992).

At the beginning of the day, prior to the first heat exposure participants were requested to be euhydrated, hydration status was checked as described in section 3.6.1. Hydration status was also reassessed after their break, prior to their second exposure. See Figure 5.1 for a schematic of the experimental monitoring.

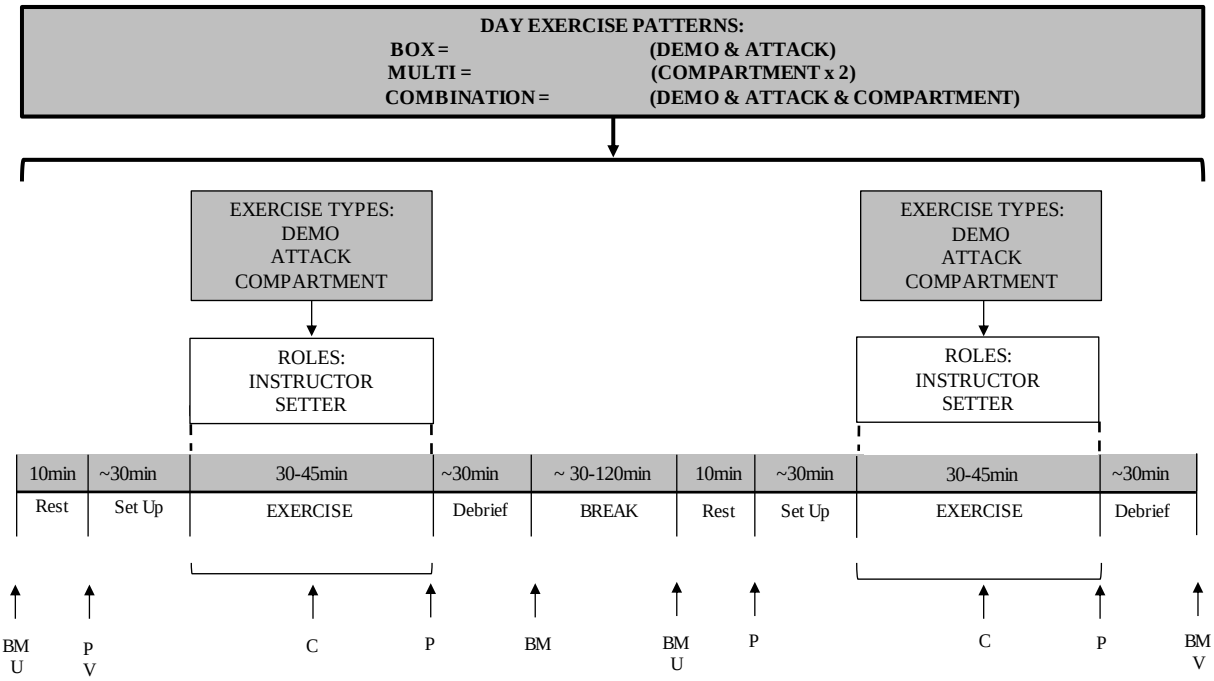

Figure 5.1 *Schematic of experimental design with data collection time points. The "Debrief" period included brief communication with students, any immediate fire ground tasks, and doffing of personal protective equipment. Abbreviations of measurement collections are: BM = nude body mass; U = urine hydration check; P = physiological and perceptual measurements of* T_{re}, T_{skin}, *HR, RPE, TS and HISI; V = venous blood sample; C = continuous HR and* T_{skin}.

5.3.2 Fire Exposures

Training exercises were assessed as individual exercises, with blood samples assessed over a day involving multiple fire exercises. There were 3 types of exercises: DEMO, ATTACK and COMPARTMENT. Within each exercise FSI performed one of two roles, a SETTER or an INSTRUCTOR. In total 19 fire exercises were assessed. There were 12 total days monitored, made up of 3 different exercise patterns: BOX, consisting of 2 single box fire exercises, a DEMO and an ATTACK; MULTI: 2 COMPARTMENT exercises; COMBINATION, 2 single box exercises, a DEMO and an ATTACK, followed by a COMPARTMENT in the afternoon. Within each exercise up to 5 participants were monitored and some participants completed the same exercise or pattern on more than 1 occasion. Overall 74 sets of individual exercise data and 36 sets of day blood data were recorded. Table 5.1 details the number of days assessed with participant anthropometric data, whilst Table 5.2 provides participant details for exercises and roles. Instructors were at times responsible for the outside safety of an exercise and consequently did not all complete every type of exercise.

Table 5.1 *The number of days collected with participant numbers and anthropometric data.*

DAY Exercise Patterns	Number of Days	Number of Participants	Participant Age (yrs)	Participant Height (cm)	Participant Body Mass (kg)
BOX	18	10	42 ± 8	164.9 ± 29.9	94.5 ± 34.0
MULTI	10	6	41 ± 8	170.0 ± 9.2	80.6 ± 18.6
COMBINATION	8	6	46 ± 9	178.7 ± 4.0	84.3 ± 10.3

Table 5.2 *The total number of exercises monitored and roles within each exercise collected, with participant numbers and anthropometric data.*

EXERCISES Types	Role	Number of Exercises	Number of Participants	Participant Age (yrs)	Participant Height (cm)	Participant Body Mass (kg)
DEMO	SETTER	10	9	44 ±9	177.2 ± 6.8	89.1 ± 12.0
	INSTRUCTOR	13	12	42 ± 8	175.8 ± 7.9	86.5 ± 13.4
	TOTAL	23	14	42 ± 7	175.4 ± 8.1	84.8 ± 14.1
ATTACK	SETTER	11	9	44 ± 8	174.7 ± 5.5	85.7 ± 13.8
	INSTRUCTOR	12	10	41 ± 8	175.4 ± 9.7	81.8 ± 14.9
	TOTAL	23	14	42 ± 7	175.4 ± 8.1	84.8 ± 14.1
COMPARTMENT	SETTER	10	4	45 ± 9	174.6 ± 8.5	80.9 ± 14.0
	INSTRUCTOR	18	11	43 ± 7	172.8 ± 7.8	78.0 ± 16.5
	TOTAL	28	12	44 ± 8	174.3 ± 8.2	82.4 ± 14.5

5.3.3 Physiological Measures

Nude body mass was recorded prior to and post each wear to calculate SR (section 3.8.6). Due to the dangerous nature of their job, and the frequency of wears completed as part of a course, FSI were allowed to consume water ad libitum during and after exposures. Participants were requested to record the volume of water consumed, which was later taken into account for SR calculations.

Participants were fitted with a downloadable HR monitor (section 3.8.4), iButtons for T_{skin} (section 3.8.2), and asked to insert a rectal probe for T_{re} measurement (section 3.8.1). Data was recorded following a 10 min rest period in the base laboratory prior to PPE being worn. Live fire exposure began 30 - 60 min post resting period, as time was needed by FSI to brief FF, don PPE, and set up. HR and T_{skin} was recorded continuously during the wear, whilst T_{re} was measured immediately on exit.

5.3.4 Perceptual Measures

RPE, TS and HISI (section 3.10) were recorded at the end of the rest period. Measures were repeated immediately post wear, whilst T_{re} was recorded.

5.3.5 *Venous Blood Collection*

A venous blood sample (10mL) was taken from the anti-cubital fossa by a trained phlebotomist (section 3.9) during the rest period and within 30 min of the last wear of the day. The delay in sampling post exposure was due to ensuring the safety of FF and the removal of PPE. Samples collected at the East Sussex and Surrey training centres were analysed for CBC using the Sysmex at the Welkin Laboratories (section 3.9.1). Samples from the Fire Service College were analysed at Coventry University. This was to ensure all samples were analysed within 2 hrs of collection. Samples were subsequently centrifuged and frozen for later IL-6, CRP and cTnT analysis (section 3.9.2). IL-6 intra assay CV% was 8.6% and inter assay CV was 11.1%. CRP intra/inter assay CV were 5.8% and 9.1% respectively. The upper reference limiti for cTnT, based on the 99th percentile, was 14 ng.L^{-1}. Post day haematology variables were corrected for changes in plasma volume (section 3.9).

5.3.6 *Statistical Analysis*

All physiological, perceptual, and haematological variables data were analysed using IBM SPSS Statistics version 22, reported as mean ± SD, with significant set at $p < 0.05$ (section 3.11). Data were tested for normality using the Shapiro-Wilk test (3.11.4). Effect sizes are presented as partial eta squared (η_p^2) for ANOVA results and Cohen's *d* for comparison between two groups of data (section 3.11.7.4).

5.3.6.1 Exercise Analysis

Due to the nature of the courses run by FSI time of data collection could not be altered, consequently change in physiological variables (ΔT_{re}, ΔT_{skin}, and ΔHR) were analysed via one way repeated measures ANOVA, to control for baseline variations due to circadian rhythms. SR and average HR were also analysed by one way repeated measures ANOVA. Two way repeated measures ANOVA were conducted on all other dependent variables (RPE, TS, HISI) between pre and post measurements to identify if there is a significant main effect for time (PRE vs POST) and exercise (ATTACK, DEMO, COMPARTMENT) and a significant interaction between time and exercise (section 3.11.7.3). Sphericity was assessed as per section 3.11.5. Bonferroni corrected follow up tests were conducted where significant interactions were identified.

Assessment of differences in physiological variables (ΔT_{re}, ΔT_{skin}, ΔHR, average HR, and SR) between roles (SETTER vs INSTRUCTOR) in each exercise were performed via an independent samples t-test (section 3.11.7.2). Perceptual variables (RPE, TS, and HISI) were analysed via a two way between measures ANOVA to identify if changes over time (PRE vs POST) were different between role types (SETTER vs INSTRUCTOR) within each exercise.

5.3.6.2 Day Analysis

Paired samples t-tests were conducted to establish if there was a difference between pre and post levels of haematological variables to assess the effect of a day of exercises on FSI CBC, IL-6, CRP, and cTnT

(section 3.11.7.2). Mann Whitney U tests were used where data violated normality assumptions. These variables were also analysed using a one way between subjects ANOVA to identify if there was a difference in change of each blood marker for the exercise patterns (BOX, MULTI, or COMBINATION). If homogeneity of variance assumption was violated, Brown-Forsythe correction was applied. Data that violated normality assumptions was analysed by a Kruskal-Wallis test. Bonferroni corrected follow up tests were used to identify where significant differences occurred. Pearson's or Spearman's correlation coefficients were calculated for blood variables and total day variation in ΔT_{re}. Correlation coefficients were interpreted as described in section 3.11.7.1

5.4 RESULTS

5.4.1 Hydration status

Hydration status in the morning was: U_{col} 3 ± 2, U_{osm} 458 ± 266 mOsm.kgH_2O^{-1}, and U_{spg} 1.014 ± 0.008. In the afternoon U_{col} was 3 ± 2, U_{osm} 566 ± 298 mOsm.kgH_2O^{-1}, and U_{spg} 1.017 ± 0.010. There was a significant increase in osmolality (p = 0.020) and specific gravity (p = 0.040) values from the morning to the afternoon. In total 69% of FSI started their morning exercise euhydrated, compared to 44% of FSI who began the afternoon exercise euhydrated.

5.4.2 Exercises

A complete DEMO, ATTACK and COMPARTMENT exercise was performed by 10 participants (age: 44 ± 8 yrs, height: 163.4 ± 29.1 cm, body mass: 92.0 ± 35.0 kg). The mean duration of each exercise type was: DEMO 33.7 ± 7.0 min, ATTACK 38.5 ± 8.7 min, and COMPARTMENT 38.5 ± 10.1 min. Live fire temperatures ranged from 70°C at floor level to 700°C at ceiling height depending on position in the structure.

5.4.2.1 Physiological Response to Exercises

See Figure 5.2 for ΔT_{re}, ΔHR, average HR, and ΔT_{skin} from the 10 participants who completed all exercise types. One way ANOVA of these participants' responses revealed significant differences in ΔT_{re} between the exercises (p = 0.004, η_p^2 = 0.465), with COMPARTMENT (0.49 ± 0.28°C) resulting in a greater ΔT_{re} than DEMO (0.23 ± 0.19°C) (p = 0.045, d_z = 1.00) and ATTACK (0.27 ± 0.22°C (p = 0.016, d_z = 1.22). Absolute T_{re} post exercise was 37.79 ± 0.16 for DEMO, 37.92 ± 0.30 for ATTACK, and 37.92 ± 0.35 for COMPARTMENT, with T_{re} only exceeding 39.0°C on two occasions in the COMPARTMENT exercise. ΔHR and average HR were both not different between exercises (p = 0.225, η_p^2 = 0.153 and p = 0.833, η_p^2 = 0.020, respectively). When assessed as a percentage of exercise time, ΔHR was also not different at any % of time between exercise types (p = 0.772, η_p^2 = 0.075).

ΔT_{skin} was different between exercise types (p = 0.011, η_p^2 = 0.396), with a greater ΔT_{skin} occurring in DEMO (7.52 ± 1.60°C) compared to ATTACK (5.19 ± 1.70°C) (p = 0.030, d_z = 1.08). There was no difference between COMPARTMENT (7.41 ± 2.12°C) and either DEMO (p = 1.00) or ATTACK (p =

0.156). When assessed as a percentage of exercise time, ΔT_{skin} was not different at any % of time between exercise types (p = 0.107, η_p^2= 0.140). SR was not different between the exercise types (p = 0.762, η_p^2 = 0.116).

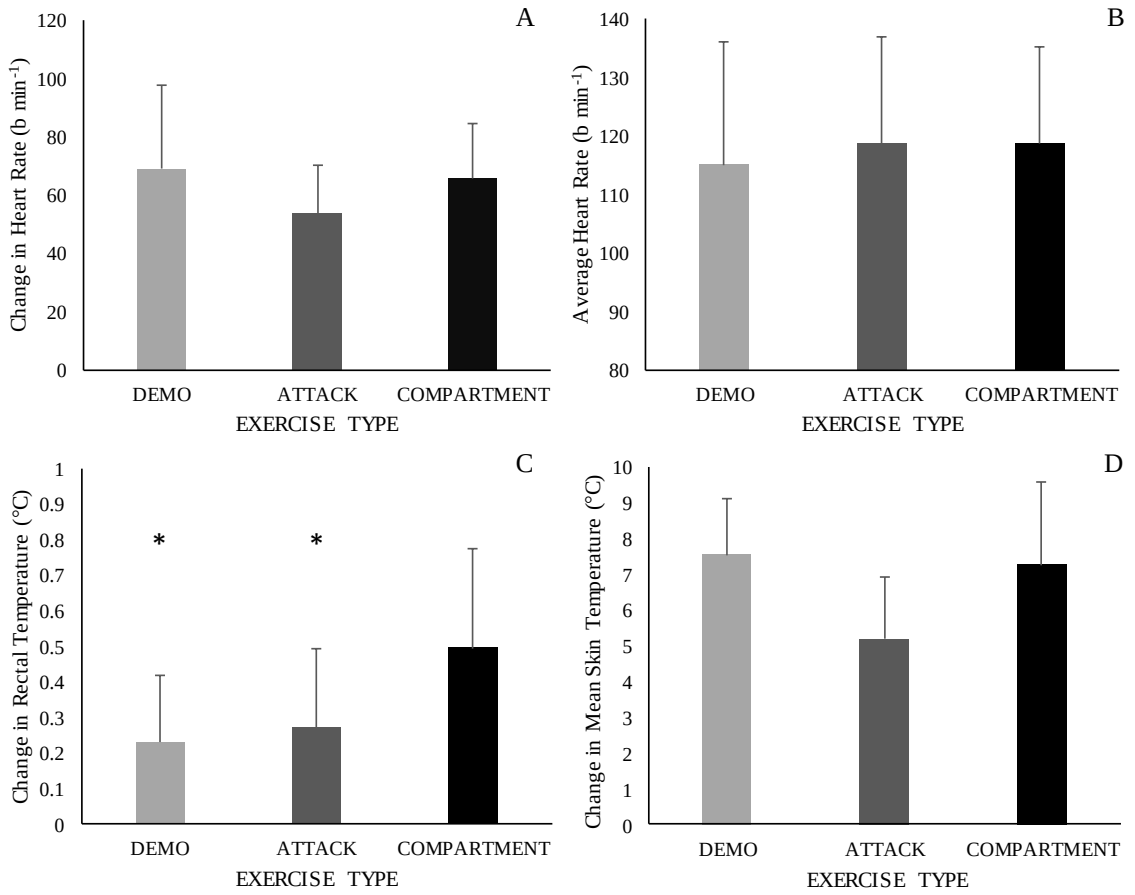

Figure 5.2 *Physiological variables (mean ± SD) for each exercise from the 10 complete participants, graph A displays change in heart rate data, B average heart rate, C change in rectal temperature, and D change in mean skin temperature. * denotes a significant difference ($p < 0.05$) from the COMPARTMENT exercise.*

5.4.2.2 Perceptual Response to Exercises

Analysis of the 10 participants who completed all exercise types revealed RPE increased post exercises (p < 0.001, η_p^2= 0.965), however this increase was not different between exercise types (p = 0.354, η_p^2 = 0.109). Change in RPE was 5 ± 3 in DEMO, 6 ± 2 in ATTACK, and 5 ± 2 in COMPARTMENT. TS followed a similar pattern, increasing post exercise (p < 0.001, η_p^2 = 0.848), but with no difference in increase between the exercise types (p = 0.598, η_p^2 = 0.056). Change in TS was 2 ± 1 in DEMO, 1.5 ± 1 in ATTACK and 1.5 ± 1 in COMPARTMENT. An increase of 1 is the difference from "neutral" to "warm", and increase of 2 from "neutral" to "hot". HISI also increased over time (p = 0.002, η_p^2 = 0.66) and was not effected by exercise type (p = 0.501, η_p^2 = 0.74). Increases in HISI were 10 ± 9 in DEMO, 7 ± 6 in ATTACK, and 10 ± 11 in COMPARTMENT.

5.4.3 Roles

Data collected from all 16 participants were analysed for assessment of differences between role types.

5.4.3.1 Physiological Response to Roles

There was no difference in ΔT_{re} between SETTERS and INSTRUCTORS for the DEMO (p = 0.875) or ATTACK exercises (p = 0.864). However, for the COMPARTMENT exercise SETTERS exhibited a greater ΔT_{re} than INSTRUCTORS (0.67 ± 0.29°C vs. 0.43 ± 0.18°C, (p = 0.027, d_s = 0.99). ΔHR was similar between SETTERS and INSTRUCTORS in the DEMO (p = 0.668), ATTACK (p = 0.648), and COMPARTMENT (p = 0.488). Average HR was also similar between the roles for all exercise types (DEMO: p = 0.251; ATTACK: p = 0.584; COMPARTMENT: p = 0.751). Furthermore ΔT_{skin} and SR were similar between roles in the DEMO (p = 0.537, p = 0.870), ATTACK (p = 0.487, p = 0.066), and COMPARTMENT (p = 0.339, p = 0.385). See Figure 5.3 for ΔT_{re}, ΔHR, average HR, and ΔT_{skin} for the two roles in each exercise type.

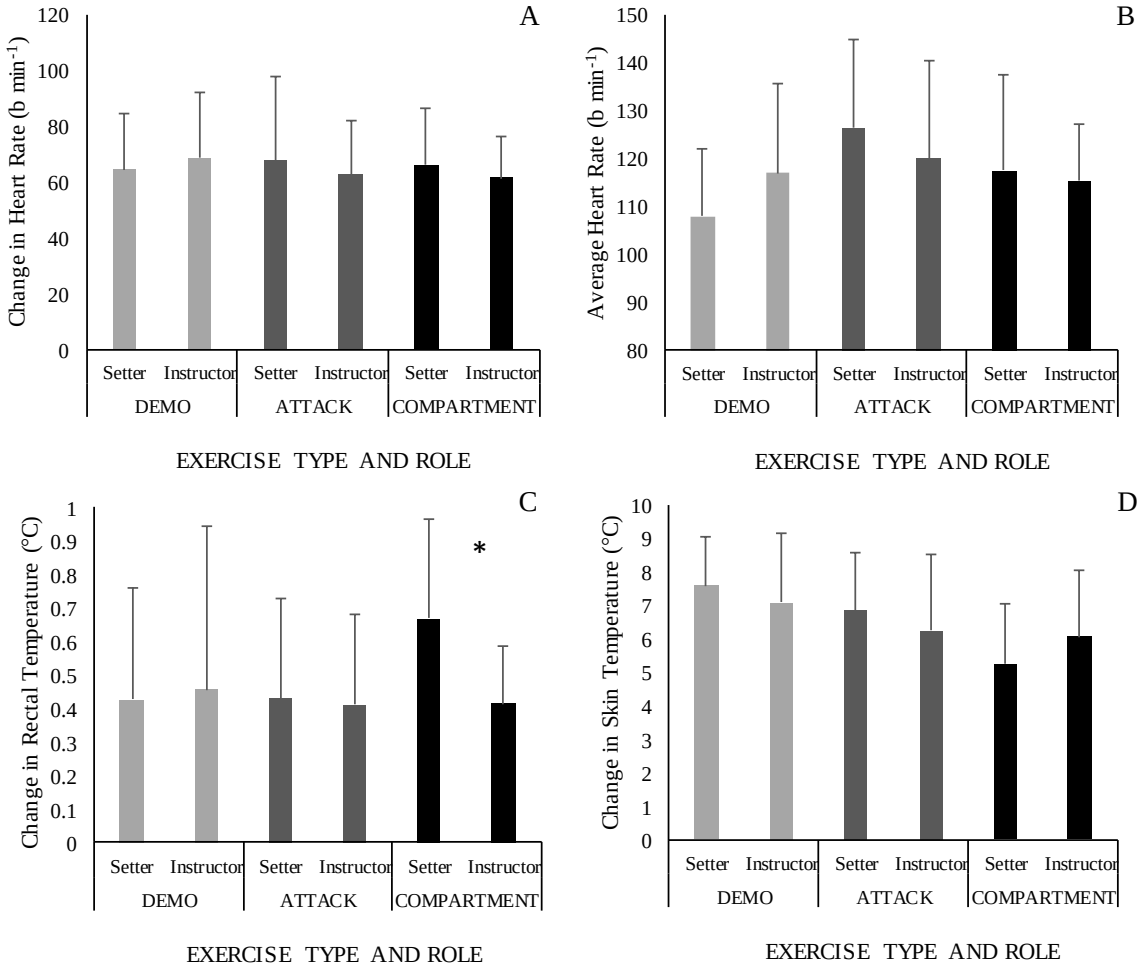

Figure 5.3 *Physiological variables for both roles (SETTER and INSTRUCTOR) within each exercise type. Graph A displays change in heart rate data, B average heart rate, C change in rectal temperature, and D change in mean skin temperature. * denotes a significant difference between the roles.*

5.4.3.2 Perceptual Response to Roles

TS increased in each exercise ($p < 0.001$), but this increase was not effected by FSI role in the DEMO ($p = 0.40$, $\eta_p^2 = 0.032$), ATTACK ($p = 0.360$, $\eta_p^2 = 0.040$), and COMPARTMENT ($p = 0.064$, $\eta_p^2 = 0.126$). RPE increased in all exercises ($p < 0.001$), but the increase was also not effected by FSI role in the DEMO ($p = 0.220$, $\eta_p^2 = 0.068$) and ATTACK ($p = 0.536$, $\eta_p^2 = 0.019$). Alternatively, the increase in RPE following the COMPARTMENT exercise was different between roles ($p = 0.001$, $\eta_p^2 = 0.328$), with post RPE being greater in SETTERS (14 ± 2) than INSTRUCTORS (11 ± 2) ($p = 0.002$, $d_s = 1.35$). HISI increased in all exercises ($p < 0.001$), but this increase was not effected by FSI role in the DEMO ($p = 0.958$, $\eta_p^2 = 0.000$) and ATTACK ($p = 0.537$, $\eta_p^2 = 0.018$). In the COMPARTMENT exercise ANOVA analysis suggests HISI was different between the roles over time ($p = 0.042$, $\eta_p^2 = 0.150$), although follow up tests did not identify differences pre or post exercise ($p = 0.316$ and $p = 0.173$, respectively).

5.4.4 Days

Haematological data collected from all 16 participants was analysed for assessment of differences pre to post a day containing fire exercises and the impact of exercise patterns on variables.

5.4.4.1 Haematological Response

At the end of a working day containing fire exercises, analysis of CBC (n = 36) revealed a significant increase in WBC, PLT, MPV, NEUT, LYMPH, and MONO ($p < 0.01$). IL-6, CRP, and cTnT levels were not normally distributed, as determined by the Shapiro-Wilks method. A Wilcoxon signed rank test revealed an increase in IL-6 ($p < 0.001$) and cTnT ($p < 0.001$), however there was a decrease in CRP ($p = 0.048$). See Table 5.3 for mean ± SD for all hematology markers data.

Table 5.3 *CBC, IL-6, CRP and cTnT pre and post a FSI working day consisting of multiple fire exercises (n = 36). * denotes a significant difference between pre and post a working day.*

Haematological Variable	Pre day (mean ± SD)	Post day (mean ± SD)	Effect size (d_z)
WBC ($x10^9.L^{-1}$)	5.74 ± 1.01	8.15 ± 1.67*	1.69
PLT ($x10^9.L^{-1}$)	209 ± 43	262 ± 59*	1.45
MPV (fL)	10.33 ± 1.04	11.16 ± 2.15*	0.54
NEUT ($x10^9.L^{-1}$)	3.15 ± 0.91	4.76 ± 1.41*	1.25
LYMPH ($x10^9.L^{-1}$)	1.78 ± 0.44	2.32 ± 0.57*	1.13
MONO ($x10^9.L^{-1}$)	0.61 ± 0.18	0.87 ± 0.30*	1.05
EO ($x10^9.L^{-1}$)	0.15 ± 0.10	0.15 ± 0.10	0.10
BASO ($x10^9.L^{-1}$)	0.08 ± 0.19	0.05 ± 0.04	0.19
IL-6 ($pg.mL^{-1}$)	4.09 ± 3.67	5.22 ± 4.20*	1.14
CRP ($mg.L^{-1}$)	1.46 ± 1.42	1.36 ± 1.34*	0.35
cTnT ($ng.L^{-1}$)	3.99 ± 1.38	5.44 ± 1.94*	1.08

WBC = white blood cell count, PLT = platelet count, MPV = mean platelet volume, NEUT = neutrophil count, LYMPH = lymphocyte count, MONO = monocyte count, EO = eosinophil count, BASO = basophil count, IL-6 = interleukin-6, CRP = C reactive protein, cTnT = cardiac troponin T.

5.4.4.2 Haematological Response to Exercise Patterns

Differences between exercise patterns were only identified for ΔMPV (p = 0.024), ΔPLT (p = 0.035), and ΔIL-6 (p = 0.013). ΔMPV was greater following the MULTI (1.88 ± 1.29 fL) compared to BOX (0.28 ± 1.59 fL), (p = 0.020, d_s = 1.07). MULTI also resulted in a greater ΔPLT (81 ± 52 x10^9.L^{-1}) than BOX (43 ± 25 x10^9.L^{-1}) (p = 0.020, d_s = 1.03). ΔIL-6 was greater in the COMBINATION order (2.35 ± 1.07 pg.mL^{-1}) in comparison to both MULTI (0.91 ± 0.70 pg.mL^{-1}, p = 0.011, d_s = 1.63) and BOX (0.84 ± 0.82 pg.mL^{-1}, p = 0.003, d_s = 1.68). See Figure 5.4 for differences in ΔWBC, ΔPLT, ΔMPV and ΔIL-6 between the exercise patterns.

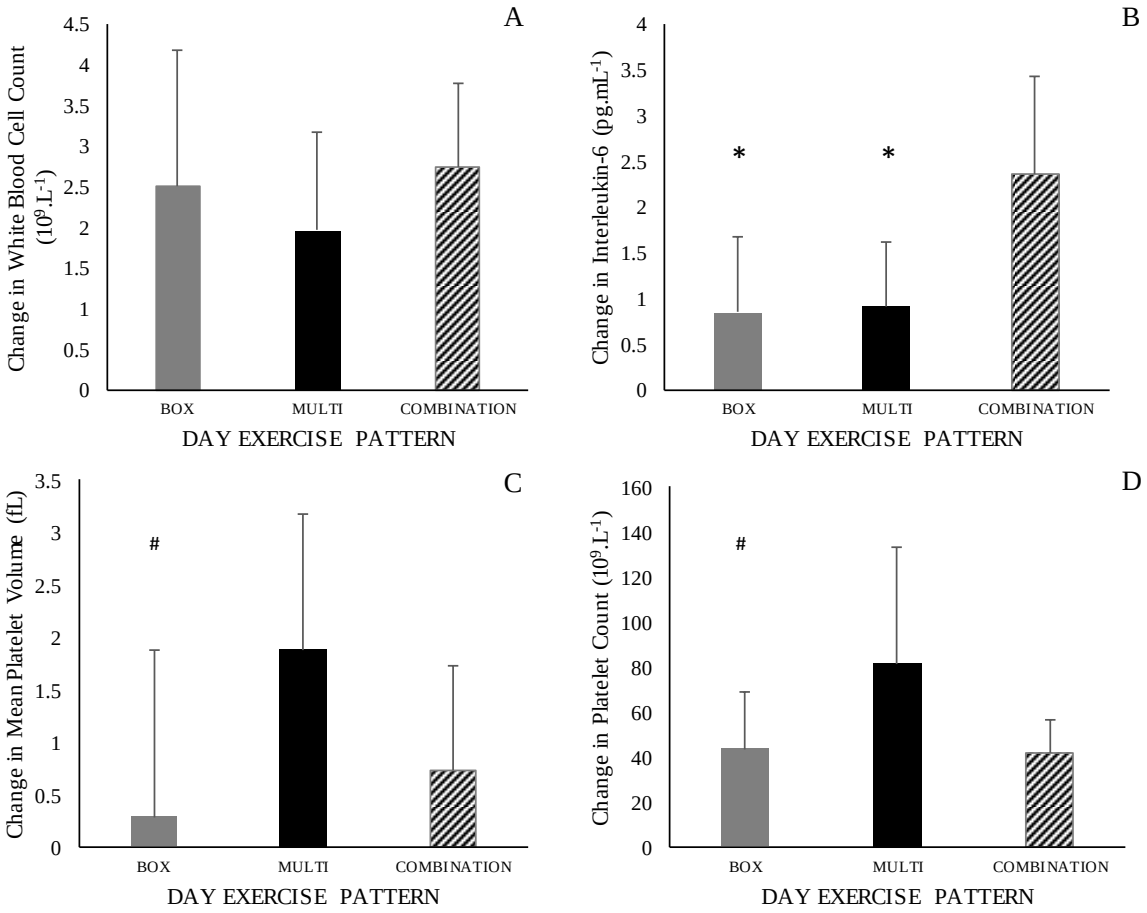

Figure 5.4 *Haematological variables (mean ± SD) following different exercise patterns (BOX, MULTI, COMBINATION). Graph A displays change in white blood cell count, B change in interleukin-6, C change in mean platelet volume, and D change in platelet count. * denotes a significant difference from COMBINATION and # denotes a significant difference from MULTI ($p < 0.05$).*

5.4.4.3 Correlations

Total variation in ΔT_{re} across a day were significantly correlated with ΔWBC (r = 0.437, p = 0.010), ΔMONO (r = 0.588, p < 0.001), post WBC (r = 0.542, p = 0.001), post MONO (r = 0.729, p < 0.001), and post LYMPH (r = 0.513, p = 0.002). Total ΔT_{re} was not correlated with any other haematological marker.

5.5 DISCUSSION

This study aimed to assess the physiological, perceptual, and immunological consequences of current UK FSI working practices, with specific focus on the impact of different CFBT exercise types, different roles performed, and different daily exercise patterns. The COMPARTMENT exercise type resulted in a greater ΔT_{re} than DEMO or ATTACK. In addition, in the COMPARTMENT exercise the SETTER had a greater ΔT_{re} and RPE than the INSTRUCTOR. Markers of inflammation and cardiac muscle damage were increased following a day of fire exercises, with MULTI resulting in a greater ΔPLT and ΔMPV and COMBINATION causing the greatest ΔIL-6. All alternate hypothesis can consequently be accepted.

5.5.1 Exercise Physiological and Perceptual Responses

Only two previous studies have investigated the physiological and perceptual responses of instructors to training fire exercises. Eglin et al. (2004) documented the responses from FSI at the UK Fire Service College, noting increased T_{re} of 0.27 ± 0.02°C (n = 2) and 0.70 ± 0.46°C (n = 4) from DEMO and ATTACK exercises, respectively. When all exercise types were grouped together, a mean T_{re} of 38.5 ± 0.9°C was reported (n = 32). The authors concluded that the T_{re} remained within reasonable limits, < 41°C, but that some FSI did present with a T_{re} of concern ≥ 39°C. Eglin et al. (2004) also reported maximum HR of 138 ± 26 b.min^{-1} and average HR of 109 ± 22 b.min^{-1} (n = 34). Watt et al. (2016) reported similar T_{re} responses of 38.06 ± 0.34°C and 38.08 ± 0.26°C post an exercise (n = 6) and average HR of 101 ± 17 b.min^{-1} and 97 ± 10 b.min^{-1}. The findings of this study are in accordance with both Eglin et al. (2004) and Watt et al. (2016), with overall post T_{re} from all exercise types and participants remaining within safe limits (37.92 ± 0.31°C) (n = 74), although on two occasions FSI did display post T_{re} of ≥ 39°C. Overall peak HR (147 ± 20 b.min^{-1}) and average HR (117 ± 5 b.min^{-1}) were also similar to the findings of Eglin et al. (2004) and Watt et al. (2016).

Due to the larger number of fire exercises monitored and the use of multiple fire training centres, this study is the first to be able to offer a statistical comparison of the different types of exercises conducted by FSI. The COMPARTMENT exercise resulted in a ΔT_{re} 0.26°C and 0.22°C greater than DEMO and ATTACK, respectively. It is currently unclear what the long term implications of repeatedly experiencing increased T_c are, however, Watt et al. (2016) suggest that it may result in an overtraining effect, with possible health implications such as an altered immune function. In addition, FSI exhibited no differences in RPE or TS, suggesting that they are unable to subjectively detect differences in physiological strain between exercise types. Consequently, it can be suggested that repeated COMPARTMENT exercises in a day, performed on consecutive days should be avoided, to reduce the time spent at a high T_c.

5.5.2 Exercise Roles

Within the COMPARTMENT exercise there were significant differences in ΔT_{re} and RPE between the roles performed by FSI. When working as an INSTRUCTOR, teaching and monitoring trainees, FSI had a lower ΔT_{re} and RPE than those working as a SETTER. Although SETTERS are predominately outside the structure, they still wear full PPE and BA whilst performing tasks such as carrying pallets and moving ventilation doors, which are often close to the fire. It is likely that the increased perception of effort and ΔT_{re} is caused by the proximity to the source of the fire, alongside the more physically demanding tasks (Horn et al. 2017). Horn et al. (2017) have recently provided comprehensive analysis of FF job roles during realistic firefighting scenarios created in purpose built multiple room structures. The authors found an increased ΔT_c during the outside ventilation role (1.84 ± 0.49 °C) compared to inside roles (0.93 ± 0.27°C). Although the ventilation aspect and location of the roles are similar, it is important to note that the exact tasks studied differ between this study and that of Horn et al. (2017). For instance, the focus on a realistic fire scenario, rather than training, combined with the more demanding tasks required of FF in comparison to FSI, likely resulted in a greater level of physical activity. These differences therefore explain the greater ΔT_c reported by Horn et al. (2017) than documented in this study. From an applied position, the staffing of courses should be considered, as it is postulated that role rotation, to minimise frequent conductance of the SETTER role, may reduce the cumulative physiological strain experienced.

5.5.3 Day Haematological Responses

Following a working day involving fire exercises, FSI experienced an increase in WBC (+24.7%), NEUT (+51.1%), LYMPH (+30.3%), and MONO (+42.6%). This replicates the findings of Watt et al. (2016) who also reported increases of WBC (+19%) and NEUT (32.3% and 26.7%), although differences in LYMPH and MONO were not noted. Smith et al. (2005) and Walker et al. (2015) also reported the occurrence of leukocytosis immediately following search and rescue tasks experienced by FF, although Walker et al. (2015) provided the heat stimulus via gas burners to 100 ± 5°C rather than live fire. Both studies reported increased WBC (+85% and +20%, respectively) and NEUT (+54% and +40%, respectively), with Smith et al. (2005) also noting increased LYMPH (+141%) and MONO (+134%). The greater increases noted by Smith et al. (2005) are likely the result of the live fire and fire suppression tasks which did not use a standardised work pace, providing a greater level of physiological strain.

Leukocytosis following physical activity and a hot environment is mediated by demargination of leukocytes from the vasculature, generated by sympathoadrenal activation increasing circulating catecholamine, which reduce the interaction between leukocytes and endothelial cells (Shephard 2003; Smith et al. 2005; Walsh & Whitham 2006). Repeated neutrophilia may deplete bone marrow neutrophil stores (Gleeson 2002) and consequently neutrophils present in circulation may be less mature and have a reduced phagocytic and oxidative burst activity, as they are forced to leave the bone marrow before they are fully developed (Keen et al. 1995). This may result in a decrease in the ability of the innate immune system to combat infection post activity. It is unknown what the long term implications of repeated increases in leukocyte counts are, however Watt et al. (2016) report increased resting WBC in FSI compared to a non-heat exposed control

group. Increased resting differential leucocyte counts are also a risk factor and prognostic indicator of cardiovascular outcomes (Madjid & Fatemi 2013).

PLT and MPV are also associated with risk of atherothrombosis and cardiovascular event, with MPV on average 0.92 fL greater in individuals who suffer an acute myocardial infarction (Chu et al. 2010). Increased PLT (+25.6%) and MPV (+8.0%) were exhibited by FSI following a day of fire exercises in this study. Larger platelets are metabolically and enzymatically more active, with a greater prothombotic potential, consequently MPV is associated with platelet aggregation, synthesis of thromboxane (a hormone released from platelets that stimulates aggregation), and expression of adhesion molecules (Sharma & Berger 2011).

It is well established that PLT increases following physical activity (El-Sayed et al. 2005). Live fire scenarios have also been demonstrated to increase PLT by 5 - 31% (Smith et al. 2011; Smith et al. 2014; Walker et al. 2015; Hunter et al. 2017). In addition, Smith et al. (2011, 2014) noted that platelet function, specifically aggregability, was enhanced, with a decrease in closure time when the blood was exposed to epinephrine and collagen (-14% and -20%, respectively). However, Smith et al. (2011) detected no significant change in MPV (Pre: 9.60 ± 1.81 fL vs Post: 9.56 ± 1.97 fL). To the author's knowledge, there have been no other assessments of MPV following live fires to compare this study's findings to. Furthermore, there is not currently a clear consensus on the impact that physical activity has on MPV. Previously it has been reported that MPV increases (+18%) with a treadmill stress test in patients with ischemic heart disease, although this finding was not repeated in healthy individuals (Yilmaz et al. 2004). Wright-Beatty et al. (2014) also reported no significant differences in MPV in FF following 4 x 15 min cycling in 35°C heat. Alternatively, Lippi et al. (2014) reported a small (+3%) but significant ($p < 0.001$) increase in MPV post a half marathon and Ahmadizad & El-Sayed (2003) have noted increased MPV with acute resistance activity in healthy participants. It is proposed that the combination of activity intensity, duration and the aerobic fitness of individuals are responsible for MPV changes and consequently some scenarios may not elicit a great enough stimuli to generate an increased MPV (El-Sayed et al. 2005). The multi-faceted cause of physiological strain in FSI may explain why increased MPV was noted, with the increased duration in comparison to Smith et al.(2011) the possible cause of the difference in findings between studies.

Additionally, increased IL-6 (+27.6%) was noted post a day of exercises in FSI, however a decrease in CRP (-7%) was seen. One of IL-6's many functions is to stimulate the release of CRP from hepatocytes (Pepys & Hirschfield 2003). Increased levels of IL-6 and CRP in the blood indicate that an inflammatory response has occurred. Watt et al. (2016) also noted a 33% increase in IL-6 in FSI, although baseline levels of IL-6 were greater than those found in this study (Pre: 7.4 ± 1.5 $pg.mL^{-1}$, Post: 9.9 ± 4.4 $pg.mL^{-1}$ vs. Pre: 4.09 ± 3.67 $pg.mL^{-1}$, Post: 5.22 ± 4.20 $pg.mL^{-1}$, respectively). Watt et al. (2016) also reported a 10% reduction in CRP post fire exercise, with postulation that this may be due to increased CRP turnover as a result of greater removal or breakdown. These findings were also repeated by Walker et al. (2016) with increases in IL-6, but not CRP post live fire. Overall, the increased IL-6, combined with the increase in other haematological variables previously discussed, indicates an FSI experience an inflammatory response to training fire exercises, which is in line with previous findings.

Repeated elevations of IL-6 could lead to chronic elevation, as noted by Watt et al. (2016). Resting increased IL-6 is associated with progression of atherosclerosis in individuals with vascular risk factors (Okazaki et al. 2014) and is also linked with an increased risk of myocardial infarction in healthy individuals (Ridker et al. 2000). In addition, it has previously been proposed that repeated acute stress with insufficient recovery time results in the development of systemic chronic inflammation, as noted by elevated resting cytokine levels, and consequently may be a cause of overtraining symptoms (Smith 2000; Smith 2003). IL-6 has been suggested as one of the key cytokines that may be involved in the cytokine hypothesis of overtraining, as it can stimulate responses such as fatigue and sleep disturbances (Smith 2000; Dantzer et al. 2008).

Completing a day of live fire exercises also resulted in increased cTnT. These findings are similar to that of Hunter et al. (2017) who reported increased cTnI from 1.4 $ng.L^{-1}$ to 3.0 $ng.L^{-1}$ following a 20 min training fire exercise completed by 17 FF. Often changes in cTnT and cTnI are reported following prolonged exercise such as marathon running and have been positively correlated with exercise intensity (Serrano-Ostáriz et al. 2011; Shave et al. 2007). A meta-analysis of cTnT responses to exercise revealed that running and football playing lasting $\geq$ 60 min can result in a rise of +26 $ng.L^{-1}$ (95% CI, 5.2 - 46.0) (Sedaghat-Hamedani et al. 2015). Although increases in cTnT have been noted from 30 min onwards when continuously running (Middleton et al. 2008). Considering this it is surprising that, given the low level of physical activity and duration of exercise that FSI experience, increased cTnT is exhibited. This indicates that heat exposure, and the physiological responses it generates, may contribute to a rise in cTnT, even without a high level of exercise intensity. Whilst the levels of cTnT reported in this study remain within the normal reference range, it can be suggested that the elevated levels noted post exercises indicates that minor myocardial injury may have occurred. Further research is required to identify what the long term implications of this may be for FSI.

When comparing exercise patterns, MULTI resulted in a greater ΔPLT and ΔMPV than BOX, whilst COMBINATION caused a greater ΔIL-6 than MULTI and BOX. These findings indicate that days containing a COMPARTMENT exercise may lead to an increased inflammatory response and could put FSI at an increased risk of cardiovascular events. However, cTnT was not different between patterns, signifying that exercise patterns may not affect the level of myocardial damage. Considering the ΔPLT, ΔMPV, ΔIL-6 and T_{re} responses together, it is suggested that where possible organisation of courses should avoid repeated COMPARTMENT exercises across numerous days. Future research should investigate the long term implication of different exercise schedules.

Day variation in ΔT_{re} displayed a significant positive correlation with ΔWBC, ΔMONO and post LYMPH. This suggests that ΔT_{re} may influence the inflammatory response in FSI and consequently by reducing the ΔT_{re} experienced across a day, FSI may be able to reduce both the strain and the inflammatory response that they experience. However, other haematological markers measured were not associated with ΔT_{re}, indicating that it may not be the only driving factor behind the increased inflammatory response and cardiovascular risk. It is important to note that total ΔT_{re} does not reflect the time that FSI spent at a high T_{re}, which unfortunately is unknown due to the absence of continuous measurements. Time spent at a higher

T_{re} could potentially provide a greater association to inflammatory changes in FSI, as it gives a better understanding of thermal load.

5.5.4 Limitations

The research design used in this study provided strong ecological validity to current UK FSI working practices and consequently findings of an extremely applied nature. However, as a result of this, numerous extraneous variables were not able to be controlled by the researchers, such as duration of exercises, exercise workload, and time of exercise completion. These issues have been taken into account where possible during data analysis, such as by assessing the change in physiological variables to account for changes in circadian rhythms and analysing continuous HR and T_{skin} data as the percentage of the individuals exercise time. Due to individual expertise of FSI, unfortunately all participants were not able to conduct all roles during the exercises, preventing a repeated measures analysis from occurring. In addition, data collection was limited to the exercises and exercise patterns currently being run by the training centres. Consequently, not all 16 participants were able to be included in the comparison of exercise types and repeated measures analysis of exercise patterns was not possible, as different training centres conducted different patterns. Blood sampling immediately post each exercise type was also not possible, due to the logistics of sample analysis within a suitable time frame and distance of training centres from laboratories. Consequently, only the impact that exercise patterns has on haematological variables was assessed.

5.6 CONCLUSION

In conclusion, FSI experience increased physiological and perceptual strain following a training fire exercises. These responses are exacerbated in the COMPARTMENT exercise and more so in the role of SETTER in comparison to INSTRUCTOR. FSI also experience an increased inflammatory response to a day of exercises, with increased ΔPLT, ΔMPV, and ΔIL-6 possibly indicating an increased risk of a cardiovascular event. Correspondingly, the increased cTnT post a day of exercises indicates that FSI may experience a minor level of myocardial damage. In addition, days containing a COMPARTMENT exercise further increase some markers of cardiovascular risk. Total day variation in ΔT_{re} was correlated with markers of inflammation, namely ΔWBC, ΔMONO and post exercise LYMPH, and consequently it can be postulated that minimizing the rise in T_{re} could decrease this inflammatory response. It is suggested that appropriate training practice scheduling, giving consideration to the frequency of COMPARTMENT exercises and conductance of the SETTER role, may reduce the cumulative physiological strain, and subsequent inflammatory response, experienced by FSI.

6 STUDY 3: PRACTICAL PRE-COOLING METHODS FOR FIRE SERVICE INSTRUCTORS

6.1 ABSTRACT

OBJECTIVES: FSI experience physiological and perceptual strain, alongside inflammation, during a fire exposure. This study aimed to identify a practical pre-cooling method to reduce the acute inflammatory response and physiological and perceptual strain experienced.

METHODS: Twelve male students from the University of Brighton (age 20 ± 1 yrs, body mass 75.0 ± 9.3 kg, height 177.3 ± 4.9 cm) completed 15 min pre-cooling (phase change vest [PCV], forearm cooling [ARM], ice slurry consumption [ICE], or a no cooling control [CON]) and a 45 min intermittent walk (4 $km.hr^{-1}$, 1% gradient) in 49.5 ± 0.6°C and 15.4 ± 1.0% RH, whilst wearing PPE. Variables were assessed for differences across time and between conditions via a repeated measures two way ANOVA.

RESULTS: ICE reduced T_{re} before heat exposure compared to CON (ΔT_{re}: 0.24 ± 0.09ºC, $p < 0.001$, d = 0.38) and during exercise compared to CON, ARM, and PCV ($p = 0.026$, $\eta_p^2 = 0.145$). Thermal sensation was reduced in ICE and ARM vs. CON ($p = 0.018$, $\eta_p^2 = 0.150$). IL-6 was not effected by pre-cooling (p = 0.648, $\eta_p^2 = 0.032$).

CONCLUSION: FSI should consume 500ml of ice slurry 15 min prior to a live fire exposure to reduce the physiological and perceptual strain experienced.

6.2 INTRODUCTION

Wearing PPE and performing physical work in the high environmental temperatures associated with fire exposures leads to an uncompensable heat stress environment, which causes high levels of physiological and perceptual strain (Cheung et al. 2000; Petruzzello et al. 2009). Consequently, during a wear FSI have been reported to exhibit elevated peak T_c (37.92 - 38.5ºC) and peak HR (134 - 162 b.min^{-1}) (Chapter 5; Eglin et al. 2004; Watt et al. 2016). As documented in Chapter 5, the rise in T_{re} is effected by the type of training exposure being completed and the FSI role within the exposure, with the multi-compartment exposure and the condition setter role within it causing the greatest increases in T_{re}. Whilst average peak T_c typically remain within a safe range, on occasion values ≥ 39.0ºC have been reported (Chapter 5; Eglin et al. 2004). These exposures therefore put FSI at an increased risk of suffering from a heat related illness, such as heat exhaustion and heat stroke (Lipman et al. 2013; Barwood et al. 2009). The acute symptoms of these illnesses, such as dizziness, syncope, and an altered mental state, are dangerous within an emergency response situation (Binkley et al. 2002). Heat strain can also lead to reductions in productivity, as seen with reduced work times recorded with increased heat strain whilst wearing FF equipment (Selkirk & McLellan 2004).

Heat exposure and exercise can also result in an inflammatory response, with inflammatory cytokines such as IL-6 having been recorded to increase post live fire exposure (Walker et al. 2015; Watt et al. 2016). In addition, Chapter 5 indicates that a day of wear completion can increase IL-6 by 28%. Frequent exposure to these scenarios may result in a raised resting level of IL-6 (17.0 ± 5.7 pg.mL^{-1}) (Watt et al. 2016). Elevated levels of IL-6 may lead to the development of atherosclerosis (Woods et al. 2000; Lindmark et al. 2001). Consequently, raised resting IL-6 levels increase the risk of suffering from a cardiovascular event, with resting levels of greater than 2.28 pg.mL^{-1} increasing the relative risk by 2.3 times compared to levels below 1.04 pg.mL^{-1} (Ridker et al. 2000). In 2014 56% of U.S. FF deaths were due to cardiac-related events, making it the leading cause of death amongst FF, although contributory factors were not considered in the study (Fahy et al. 2015). It is therefore desirable to reduce the inflammatory response that is experienced.

Pre-cooling is a frequently researched and applied method used by athletes to decrease physiological and perceptual strain in hot environments (Tyler et al. 2013). Pre-cooling aims to reduce T_c and subsequently increase the body's heat storage capacity (Ross et al. 2013). There is currently little research into the use of pre-cooling for occupations, possibly due to the logistical difficulties of implementing the methods suggested because of facilities and time constraints (Ross et al. 2013). Furthermore, it is unclear if pre-cooling methods have any effect on inflammation following exercise. From Chapter 4, it is clear that few FSI are currently using pre-cooling techniques, with only 10% using ice slurries, 6% a phase change vest (PCV), and 1% placing forearms in cold water buckets. The assessment of practical pre-cooling methods that can be easily applied by FSI is needed. With different pre-cooling methods available and used by FSI, a comparison of techniques is required to provide guidance to FSI on the most beneficial method.

Forearm and hand immersion pre-cooling has only previously been used in combination with other methods (iced towels, iced vest, or feet cooling) (James et al. 2015; Tokizawa et al. 2015). Hand and forearm cooling

in 9 - 18°C water for 20 - 30 min, combined with these other methods, may elicit a 0.2°C reduction in T_c immediately after pre-cooling, which may result in a 0.3 - 0.4°C reduction in change in T_c post exercise (Minett et al. 2012; James et al. 2015; Tokizawa et al. 2015). However, the application of a mixed method is unfeasible as it immobilises individuals and requires a large freezing storage capacity. Forearm and hand cooling alone could be beneficial, as there is a large potential for heat transfer, due to the high surface area to mass ratio, and the arteriovenous anastomoses (AVA) in the hands, together with superficial veins up to the elbow, which form a specialised heat exchange organ allowing for large variations in local blood flow (Vanggaard et al. 2012). The reduction of skin temperature noted with forearm cooling may also reduce thermal sensation from "hot" to "warm" (Minett et al. 2012; James et al. 2015). As a method currently reported to be used by FSI, it is important to establish if it is beneficial.

PCV may also be an effective cooling intervention, as it acts as a thermal storage medium, absorbing energy when the phase change material's state is altered from a solid to a liquid (Reinertsen et al. 2008). The vest is lined with numerous inserts of this phase change material. The latent heat fusion of phase change materials with a melting temperature of 28ºC has been reported between 126 - 159 $kJ.kg^{-1}$, although this can be influenced by the exact material used (Gao et al. 2010; Gao et al. 2011). PCV have been demonstrated to reduce end T_c by ~0.2°C, trunk temperature by 8°C and improve thermal sensation from "hot" to "warm" (Reinertsen et al. 2008; House et al. 2013), although there is minimal research into their use, especially in uncompensable environments.

Alternatively, ice slurries have been suggested as a practical method of reducing thermoregulatory strain in an occupational setting, due to the minimal preparation time and ease of use (Brearley 2012). Mechanistically, the slurry creates a large heat storing capacity due to the additional energy required to melt the ice (334 $kJ.kg^{-1}$) (Siegel et al. 2010). Slurry consumption has therefore been noted to elicit average T_c reductions of 0.32 - 0.66ºC prior to exercise as well as increased time to exhaustion and decreased HR at the end of an exercise period (Siegel et al. 2010; Siegel et al. 2012; Yeo et al. 2012; James et al. 2015). However, 7 - 7.5 $g.kgBM^{-1}$ slurry is currently recommended (Siegel et al. 2012; Brearley 2012), making preparation complex on a large scale for multiple individuals at a time.

This study therefore aimed to investigate the effect of forearm immersion, PCV and ice slurry consumption on the physiological and perceptual strain and inflammatory response generated whilst performing a simulated multi-compartment fire exposure. Practicality of the pre-cooling methods has been considered, with only a 15 min application time and ice slurry dosage maintained as a single bolus size for all participants, to ensure ease of use.

It was hypothesised that (1) PCV, forearm cooling (ARM) and ice slurry consumption (ICE) would result in reduced physiological and perceptual strain compared to a control trial (CON), (2) ICE would generate the greatest reductions in strain compared to CON, and (3) PCV, ARM and ICE would result in a reduced inflammatory response, recorded as a reduction in IL-6, compared to CON.

6.3 METHOD

6.3.1 Participants

Twelve physically active, > 3 times a week, non-heat acclimated males (age 20 ± 1 yrs, body mass 75.0 ± 9.3 kg, height 177.3 ± 4.9 cm) were recruited from University of Brighton. FSI were not recruited for this study due to logistical issues regarding geographical location and visit frequency. Participants gave informed written consent and completed a medical questionnaire before beginning the study (section 3.1). One participant dropped out in the CON trial due to nausea at 33 min and consequently was removed from data analysis. The remaining 11 participants (age 18 ± 2 yrs, body mass 70.0 ± 9.3 kg, height 162.3 ± 5.0 cm) completed all trials.

Participants were requested to avoid caffeine and exhaustive exercise 12 hours before each session and alcohol 24 hours before. Participant adherence was checked with a questionnaire completed before each session (section 3.6).

6.3.2 Experimental Design

Participants were required to complete four testing sessions: CON, PCV, ARM, ICE. Sessions began between 8:00am and 9:00am, to control for circadian rhythms and were performed in a randomised order. Upon arrival to the laboratory hydration levels were measured via a urine sample. Participants were required to be in a euhydrated state for testing to begin, which was checked as described in section 3.6.1.

Each session then consisted of a 10 min rest period and a 15 min pre-cooling period in ambient temperatures (24.7 ± 1.2ºC, 38.5 ± 8.0% relative humidity (RH)) whilst wearing a wicking base layer (Odlo), trousers (Ballyclare Special Products Ltd.) and boots (9005 GA, Jolly Scarpe, USA). During PCV cooling the vest (Dräger Comfort Vest CVP 5220, Drägerwerk AG & Co. Germany) was worn over the wicking base layer and fastened as tight as comfortably possible. The PCV contained 20 individual phase change material elements, made of a salt crystal mixuture, that were integrated into the lining of the vest. The product information for the vest states that the elements are designed to liquefy at a skin temperature of 27.78ºC. During the ARM trial the sleeves of the base layer were rolled up and both arms placed elbow deep in cold tap water (15.8 ± 1.1ºC). For ICE pre-cooling participants consumed 500ml of ice slurry (-1ºC) consisting of two thirds shaved ice produced using a snow cone maker (JM Posner, Watford, Tesco Stores Ltd, Cheshunt, UK) and one third diluted drinking cordial (Robinsons Orange, 0.6g per 100ml carbohydrate content). During CON, PCV and ARM trials participants were given 500ml of the same diluted cordial made with tap water (22ºC) to control for any hydration effect (Pryor et al., 2015).

After cooling, participants dressed in the remainder of the protective clothing: jacket (Ballyclare Special Products Ltd.), fire hood (MSA Gallet, Bellshill, UK), helmet (F1SF, MSA Gallet, Bellshill, UK), and gloves (Firemaster 3, Southcombe Brothers Ltd, Somerset, UK). They also donned a rucksack weighted at 9.52 kg to replicate a BA, making the total weight of the ensemble 17 kg. Participants then completed a simulated fire exposure, involving 45 min of intermittent exercise, alternating between 5 min walking (4

km.hr^{-1} and 1% gradient) and 5 min standing rest, on a treadmill (section 3.1.3.), in 49.6 ± 0.8°C and 15.4 ± 1.2% RH (see Figure 6.1 for schematic). This protocol was selected to elicit T_{re} and HR responses similar to that experienced by FSI during a multi-compartment exposure (Chapter 5; Watt et al. 2016; Watkins & Richardson 2017).

6.3.3 Measures

Nude body mass was recorded before and after each testing session (section 3.7.2). T_{re}, HR and T_{skin} (collected via skin thermistors) were recorded at the end of the resting period and every 5 min throughout pre-cooling and the heat exposure (section 3.8 and 3.8.4). Perceptual strain was assessed via RPE and TS at the end of the rest period and then every 5 min until the end of the heat exposure (section 3.10).

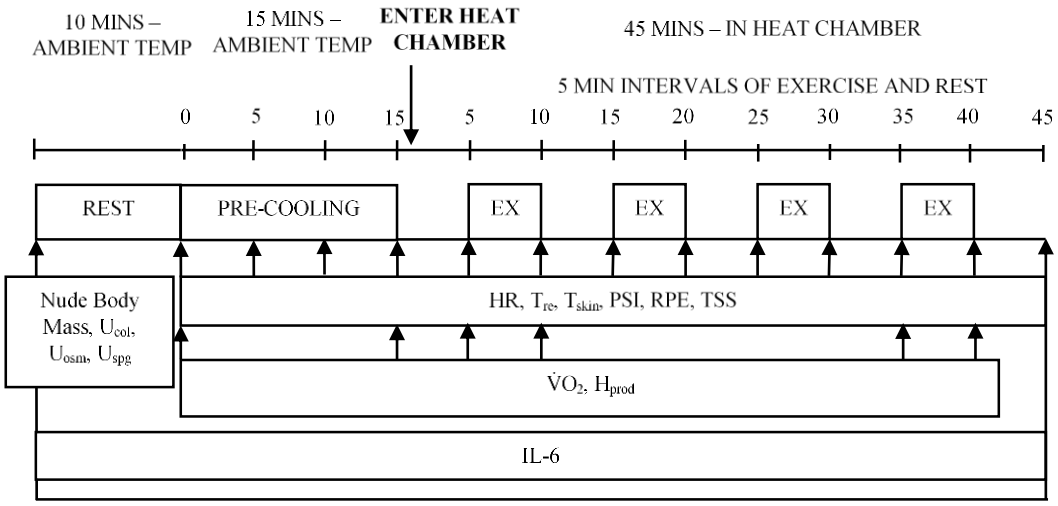

Figure 6.1 *Schematic of the testing protocol including 10 min rest period, 15 min pre-cooling period, and 45 min heat exposure during which participants alternated between 5 min walking (4 km.hr^{-1} and 1% gradient) and 5 min rest. Data collection time points are indicated. U_{col} = urine colour, U_{osm} = urine osmolality, U_{spg} = urine specific gravity, EX = exercise, HR = heart rate, T_{re} = rectal temperature, T_{skin} = skin temperature, PSI = physiological strain index, RPE = rating of perceived exertion, TS = thermal sensation, $\dot{V}O_2$ = volume of oxygen uptake, $\dot{H}_{prod}$ = metabolic heat production, IL-6 = interleukin-6.*

6.3.4 Gas Analysis

Expired gas collection occurred via Douglas bags after rest and pre-cooling, and at the beginning and end of the first and last exercise block. Gas samples were collected and analysed as per section 3.8.3.

6.3.5 Interleukin- 6

Venous blood (5ml) was collected from the ante-cubital fossa prior to the rest period and post each session (section 3.9). Whole blood was then centrifuged and stored at -86°C for later ELISA analyse for IL-6 (section 3.9.2) Intra/inter-assay coefficient CV was 8.0% and 5.3% respectively.

6.3.6 Derivative Calculations

T_{skin} was determined using measurements taken from the four skin sites (section 3.8.2) (Ramanathan 1964). PSI was calculated as per the equation described in section 3.8.5 (Moran et al. 1998) and H_{prod} was calculated as per the equations in section 3.5. SR was calculated at the end of heat exposure as per section 3.8.6.

6.3.7 Statistical Analysis

Data were analysed using IBM SPSS Statistics 22. Data were tested for normality and sphericity (section 3.11). Pearson's correlations (section 3.11.7.1) were run between ice slurry dosage ($g.kgBM^{-1}$) and the change in T_{re} (ΔT_{re}) at the end of pre-cooling and post heat exposure. Two way repeated measures ANOVAs (3.11.7.3) were conducted on all dependent variables to establish if there was a significant main effect for time and condition and a significant interaction between time and condition. Bonferroni corrected one way ANOVAs were used to establish where significant interactions occurred. Data is reported as the mean ± SD and significance was set to $p < 0.05$ (3.11.6). Effect sizes for main effects and interactions are reported as per section 3.11.7.4.

6.4 RESULTS

All participants met the urine hydration requirements before each session (see Table 6.1.). No gastrointestinal discomfort was reported with ice slurry consumption. There was no difference in SR between the conditions ($p = 0.228$, $\eta_p^2 = 0.132$) (see Table 6.1).

Table 6.1 *Mean ± SD for hydration values and sweat rate of each pre-cooling trial.*

	PCV	**ARM**	**ICE**	**CON**
U_{col}	2 ± 1	2 ± 1	2 ± 1	2 ± 1
U_{osm} ($mOsm.kgH_2O^{-1}$)	243 ± 230	209 ± 119	250 ± 177	276 ± 199
U_{spg}	1.007 ± 0.006	1.005 ± 0.003	1.006 ± 0.004	1.006 ± 0.006
SR ($L.hr^{-1}$)	1.62 ± 0.72	1.50 ± 0.87	1.52 ± 0.60	1.88 ± 0.51

U_{col} = urine colour, U_{osm} = urine osmolality, U_{spg} = urine specific gravity, SR = sweat rate

6.4.1 Core temperature

ΔT_{re} was statistically analysed due to differences present at rest in T_{re} between conditions ($p = 0.042$), Table 6.2 presents absolute values for physiological and perceptual strain variables. See Figure 6.2 for all ΔT_{re}

data. ΔT_{re} during pre-cooling was affected by the cooling method ($p < 0.001$, $\eta_p^2 = 0.735$), with ICE resulting in a reduced T_{re} compared to CON ($p < 0.001$, $d_z = 0.40$), ARM ($p < 0.001$, $d_z = 45$) and PCV ($p = 0.009$, $d_z = 0.32$) at the end of cooling. Throughout the heat exposure ΔT_{re} increased in all conditions ($p < 0.001$, $\eta_p^2 = 0.923$). ΔT_{re} was affected by the cooling condition ($p = 0.002$, $\eta_p^2 = 0.179$) with ΔT_{re} being reduced in ICE compared to CON up to 20 min ($p = 0.05$ $d_z = 0.26$). At 20 min ΔT_{re} was lower in ICE (-0.18 ± 0.14 ºC) compared to CON (-0.02 ± 0.08ºC, $p = 0.05$ $d_z = 0.26$), ARM (-0.02 ± 0.14ºC, $p = 0.043$ $d_z = 0.24$) and PCV (-0.02 ± 0.09ºC, $p = 0.029$, $d_z = 0.27$) (see Figure 2). At the end of the heat exposure there was no difference present between conditions ($p = 0.389$, $\eta_p^2 = 0.94$) with CON (0.59 ± 0.22ºC) and ICE (0.45 ± 0.29ºC) resulting in similar ΔT_{re} ($d_z = 0.15$). Dosage of ice slurry relative to the participants' body mass was 6.75 ± 0.84 g.kg^{-1}. There was no correlation between dose and ΔT_{re} at the end of pre-cooling ($r = 0.06$, $p = 0.850$) and post heat exposure ($r = 0.22$, $p = 0.505$).

Table 6.2 *Absolute values for rectal temperature, heart rate, skin temperature, thermal sensation, and rating of perceived exertion at rest, end of cooling and at 15 min, 30 min, and 45 min of heat exposure for each condition. * denotes a significant difference between CON and ICE and ‡ denotes a significant difference between CON and ARM, with significant set at p = 0.05.*

		Rest	**End Cooling**	**15 min**	**30 min**	**45 min**
Rectal Temperature (°C)	CON	37.13 ± 0.28	37.09 ± 0.28	37.05 ± 0.29	37.27 ± 0.35	37.72 ± 0.42
	ICE	37.05 ± 0.25	36.81 ± 0.25 *	36.76 ± 0.24 *	37.06 ± 0.27	37.51 ± 0.34
	ARM	36.92 ± 0.36	36.92 ± 0.35	36.85 ± 0.29	37.05 ± 0.31	37.48 ± 0.37
	PCV	37.03 ± 0.31	36.93 ± 0.33	36.96 ± 0.31	37.17 ± 0.34	37.57 ± 0.43
Heart rate (b.min^{-1})	CON	68 ± 11	71 ± 8	95 ± 10	130 ± 13	133 ± 10
	ICE	69 ± 11	70 ± 11	89 ± 13	124 ± 12	128 ± 15
	ARM	66 ± 13	67 ± 12	89 ± 13	122 ± 11	126 ± 12
	PCV	66 ± 11	65 ± 9	89 ± 12	122 ± 9	126 ± 11
Physiological Strain Index	CON		0.06 ± 0.32	1.04 ± 0.42	3.09 ± 0.70	4.2 ± 0.74
	ICE		-0.45 ± 0.40 *	0.32 ± 0.50 *	2.51 ± 0.66	3.60 ± 1.00
	ARM		0.03 ± 0.31	0.83 ± 0.44	2.69 ± 0.46	3.72 ± 0.61
	PCV		-0.27 ± 0.27	0.86 ± 0.38	2.73 0.62	3.72 ± 0.78
Skin Temperature (°C)	CON	31.97 ± 1.17	32.71 ± 1.22	36.36 ± 0.57	37.18 ± 0.41	37.81 ± 0.56
	ICE	32.08 ± 1.21	32.87 ± 1.16	36.38 ± 0.53	37.28 ± 0.43	37.45 ± 0.41
	ARM	32.26 ± 0.45	32.40 ± 0.59	36.17 ± 0.40	37.15 ± 0.39	37.36 ± 0.24
	PCV	31.86 ± 0.97	32.35 ± 0.94	35.75 ± 0.72	36.63 ± 0.64	36.88 ± 0.52
Thermal Sensation	CON	4.0 ± 0.0	4.0 ± 0.0	5.5 ± 0.5	6.5 ± 0.5	6.5 ± 0.5
	ICE	4.0 ± 0.0	3.0 ± 0.5 *	5.0 ± 0.0	5.5 ± 0.5 *	6.5 ± 0.5
	ARM	4.0 ± 0.0	3.0 ± 0.5 ‡	5.0 ± 0.5	6.0 ± 0.5	6.5 ± 0.5
	PCV	4.0 ± 0.0	4.0 ± 0.5	5.5 ± 0.5	6.0 ± 0.5	6.5 ± 0.5
Rating of Perceived Exertion	CON	6 ± 0	6 ± 0	7 ± 1	10 ± 2	9 ± 2
	ICE	6 ± 0	6 ± 0	7 ± 1	10 ± 2	8 ± 2
	ARM	6 ± 0	6 ± 0	7 ± 1	10 ± 2	8 ± 2
	PCV	6 ± 0	6 ± 0	7 ± 1	10 ± 2	9 ± 3

6.4.2 Heart Rate

There was no difference in change in HR (ΔHR) during the pre-cooling period between any of the cooling methods ($p = 0.154$, $\eta_p^2 = 0.140$). ΔHR rose during the heat exposure, ($p < 0.01$, $\eta_p^2 = 0.943$) but was not affected by cooling condition at any time ($p = 0.053$, $\eta_p^2 = 0.134$) (see Figure 6.2). At the end of the heat exposure ΔHR for CON was 66 ± 10 b.min^{-1}, 59 ± 10 b.min^{-1} for PCV, 60 ± 15 b.min^{-1} for ARM, and 59 ± 12 b.min^{-1} for ICE.

6.4.3 Physiological Strain Index

PSI data is displayed in Figure 6.2. PSI was reduced during the 15 min pre-cooling period, ($p = 0.007$, $\eta_p^2 = 0.390$) with this change being effected by pre-cooling condition ($p = 0.001$, $\eta_p^2 = 0.306$). At the end of pre-cooling PSI was reduced in the PCV and ICE trials compared to CON ($p = 0.019$, $d_z = 0.37$ and $p = 0.013$, $d_z = 0.50$, respectively). The heat exposure caused an increase in PSI in all conditions, ($p < 0.001$, $\eta_p^2 = 0.957$). This increase was effected by cooling method ($p = 0.05$, $\eta_p^2 = 0.135$), with ICE resulting in a lower PSI than CON at 10 min (0.83 ± 0.37 vs. 1.53 ± 0.47, respectively, $p = 0.006$, $d_z = 0.61$) and 15 min (0.32 ± 0.50 vs. 1.04 ± 0.42, $p = 0.007$, $d_z = 0.62$) (Figure 6.2). At 45 min there was no difference in PSI between the cooling methods ($p = 0.103$, $\eta_p^2 = 0.184$), with CON (4.21 ± 0.75) and ICE (3.60 ± 1.00) resulting in similar PSI ($d_z = 0.39$).

6.4.4 Skin Temperature

Change in T_{skin} (ΔT_{skin}) increased during the cooling period ($p < 0.001$, $\eta_p^2 = 0.693$), however, there was no difference between cooling methods at any specific time point (PCV: 0.49 ± 0.47ºC, ARM: 0.15 ± 0.47ºC ICE: 0.74 ± 0.28ºC, CON: 0.77 ± 0.23ºC, $p = 0.106$, $\eta_p^2 = 0.20$). Heat exposure caused an increase in ΔT_{skin} in all conditions ($p < 0.001$, $\eta_p^2 = 0.955$) until reaching a plateau at 30 min. The pre-cooling method had no effect on ΔT_{skin} throughout the heating phase of the protocol ($p = 0.969$, $\eta_p^2 = 0.050$), with end ΔT_{skin} being 5.02 ± 1.09ºC for PCV, 5.10 ± 0.72ºC for ARM, 5.26 ±0.70ºC for ICE and 5.89 ± 1.17ºC for CON. Conversely, cooling method effected ΔT_{chest} throughout the heat exposure ($p = 0.002$, $\eta_p^2 = 0.176$) with ΔT_{chest} being consistently lower in the PCV condition than the CON trial at every time point ($p < 0.05$). At 45 min ΔT_{chest} was 3.64 ± 1.71°C in the PCV condition compared to 5.16 ± 1.40°C in the CON condition ($p = 0.006$, $d_z = 0.69$).

6.4.5 Perceptual Strain

Cooling method altered TS during the pre-cooling period ($p < 0.001$, $\eta_p^2 = 0.556$), with TS at the end of cooling being greater in CON than ARM, ($p < 0.01$, $d_z = 1.13$) and ICE ($p < 0.001$, $d_z = 1.06$), but similar to PCV ($p = 0.142$, $d_z = 0.37$). The pre-cooling method continued to affect TS during the heat exposure at numerous time points ($p = 0.018$, $\eta_p^2 = 0.150$) with ARM resulting in a reduced TS compared to CON at 10 min ($p = 0.015$, $d_z = 0.53$) and 20 min ($p = 0.036$, $d_z = 0.45$) of heat exposure. At 30 min ICE generated a lower TS than CON ($p = 0.046$, $d_z = 0.61$). However, by the end of the heat exposure cooling method had

no effect on TS (p = 0.230, η_p^2 = 0.132), as displayed in Figure 2. RPE remained constant during pre-cooling for all conditions. Throughout the heat exposure RPE increased during walking periods (p < 0.001, η_p^2 = 0.626), however this was not affected by the pre-cooling method at any time (p = 0.676, η_p^2 = 0.078). Peak RPE occurred at the end of the last walk at 40 min: CON 11 ± 2, PCV 10 ± 3, ARM 11 ± 2, ICE 10 ± 3.

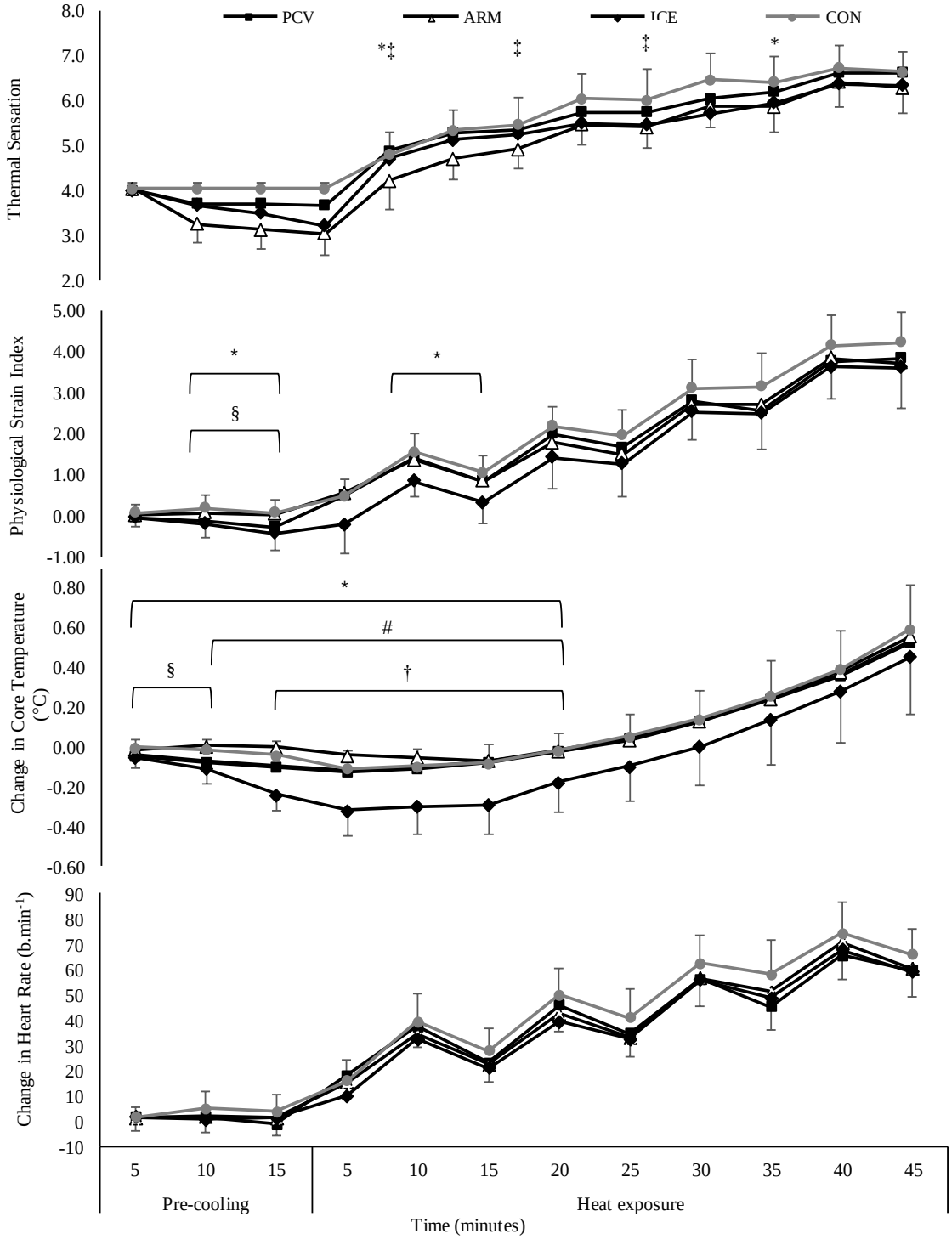

Figure 6.2 *Mean ± SD of physiological strain index, change in rectal temperature, change in heart rate and thermal sensation throughout the pre-cooling and 45 min heat exposure period. * denotes a significant difference between CON and ICE, # denotes a significant difference between ARM and ICE, † denotes a significant difference between PCV and ICE, § denotes a difference between PCV and CON, and ‡ denotes a significant difference between CON and ARM, with significance set at p < 0.05.*

6.4.6 Gas Analysis

$\dot{V}O_2$ increased during the cooling period ($p = 0.011$, $\eta_p^2 = 0.496$), but was not affected by the cooling method used ($p = 0.309$, $\eta_p^2 = 0.111$). During the heat exposure $\dot{V}O_2$ increased after the walking periods ($p < 0.001$, $\eta_p^2 = 0.971$), however this was similar in all cooling conditions ($p = 0.999$, $\eta_p^2 = 0.012$). In addition, H_{prod} was not different between cooling methods during the pre-cooling period ($p = 0.274$, $\eta_p^2 = 0.120$) and increases during exercise were similar across cooling conditions ($p = 0.997$, $\eta_p^2 = 0.016$). Changes in H_{prod} can be seen in Figure 6.3.

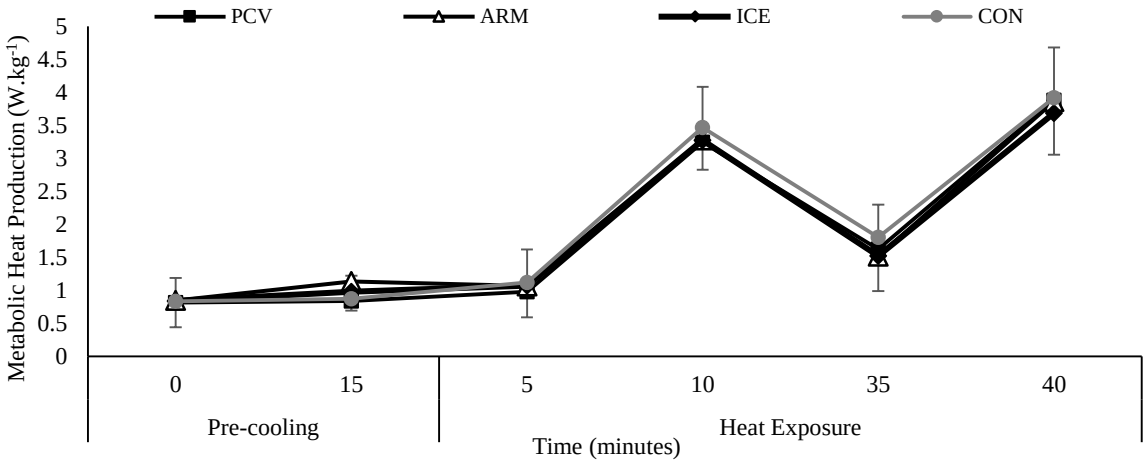

Figure 6.3 *Mean ± SD of metabolic heat production before and after the pre-cooling period, and prior to and post the first and last walking period during the heat exposure.*

6.4.7 Interleukin-6

IL-6 increased from rest to post heat exposure ($p = 0.002$, $\eta_p^2 = 0.621$). However, this increase was similar between the pre-cooling methods ($p = 0.649$, $\eta_p^2 = 0.033$). Pre heat exposure IL-6 was 1.66 ± 1.54 pg.mL^{-1} for PCV, 1.95 ± 1.63 pg.mL^{-1} for ARM, 2.05 ± 1.57 pg.mL^{-1} for ICE, and 2.55 ± 2.12 pg.mL^{-1} for CON. At the end of the heat exposure IL-6 was 2.84 ± 3.29 pg.mL^{-1} for PCV, 2.50 ± 1.66 pg.mL^{-1} for ARM, 3.12 ± 2.52 pg.mL^{-1} for ICE and 3.63 ± 3.14 pg.mL^{-1} for CON.

6.5 DISCUSSION

The aim of this study was to investigate practical pre-cooling methods which could reduce the physiological and perceptual strain experienced by FSI. It was hypothesised that (1) PCV, ARM and ICE would result in reduced physiological and perceptual strain compared to CON, (2) ICE would generate the greatest reductions in strain compared to CON, and (3) PCV, ARM and ICE would result in an attenuated rise in IL-6, compared to CON. The findings from this study suggest that hypothesis (1) can be rejected, whilst hypothesis (2) can be accepted, as the ICE condition was the only cooling intervention to decrease T_{re} in the 15 min pre-cooling period and resulted in a reduced ΔT_{re} during the first 20 min of the simulated fire exposure. PSI was also minimised in the ICE trial. Although ARM resulted in improved TS, this was without concomitant physiological change. PCV resulted in no changes in physiological or perceptual strain

compared to the CON condition, but did reduce T_{chest}. Hypothesis (3) can also be rejected as pre-cooling had no effect on IL-6.

6.5.1 Physiological Strain

The ICE method was the only strategy that successfully pre-cooled the participants in comparison to CON, reducing T_{re} by 0.24 ± 0.09ºC. ICE consumption has previously been reported to reduce T_{re} by 0.43 ± 0.14ºC and 0.66 ± 0.14ºC when ingesting 7.5 g.kgBM^{-1} in 30 min (Siegel et al. 2012), 0.50 ± 0.20ºC after consuming 8 g.kg^{-1} of slurry in 30 min (Yeo et al. 2012), and 0.32 ± 0.11ºC following consumption of 7.5 g.kg^{-1} in 20 min (James et al. 2015). In comparison to these studies, the ΔT_{re} recorded in this investigation was not as pronounced. However, the reduced consumption time period and altered dosage are probable causes of this. This study applied a 15 min consumption period with the dosage maintained at 500ml (equating to 6.75 ± 0.84 g.kg^{-1}), so as to allow speed and ease of use for FSI. However, there was no correlation between body mass specific dosage and $\Delta T_{re,}$ suggesting that a single sized bolus of slurry is adequate to use in this scenario. Although these protocol changes deviate from previously published research, they enable the pre-cooling method to be more practically applicable to a field environment, where time and space to weigh participants and ice may be limited.

The reduced T_{re} generated by the ICE pre-cooling continued into the exercise period, translating into a reduced PSI in the ICE trial. Pryor et al (2015) are the only group to have previously investigated the use of ice slurries as a pre-cooling intervention for individuals in an uncompensable heat stress environment exercising in PPE. The authors noted similar findings to this study, with ICE reducing T_c during the first 30 min of the test, with no differences in T_{skin} and HR. The extended impact of ICE on T_c noted by Pryor et al. (2015) may be due to the increased dosage and consumption time; 7.5 g.kg^{-1} over 30 min vs the 6.75 ± 0.84 g.kg^{-1} over 15 min used in this study. In addition, the exercise intensity experienced may also be a factor in the differing responses, with Pryor et al (2015) using a 45 min continuous walk test, starting at 6.4 km.hr^{-1}, with their participants required to complete 4.8 km in the time period. This higher intensity of exercise resulted in a greater rise in control trial T_c, leading to an effect of slurry consumption up to 30 min.

Alternatively, Morris et al. (2016) suggest that ice slurry consumption may reduce SR, increasing heat storage via a reduction in sweat evaporation. This response may be due to the visceral thermoreceptors instigating cold thermoeffector responses and inhibiting warm-sensitive neurons (Morris et al. 2017). The authors consequently advise caution for ice slurry prescription. However, the uncompensable environment, and specifically the PPE, used in this study minimise evaporative SR regardless of fluid temperature, with the beneficial impact of a heat sink therefore out weighing the negative reduction in SR. This is evidenced with the similar SR reported between conditions in this study. Consequently, it can be suggested that FSI completing fire exposures may benefit from ice slurry consumption, with also possible benefits for other occupational tasks involving 20 - 30 min physical activity conducted whilst wearing PPE, such as police firearm house entry (Blacker et al. 2013). In addition, work performed at a higher intensity, or application of a larger dose, may result in the greatest benefit. Furthermore, reducing the time spent at an increase T_c

may result in a decrease in the total thermal load experienced by FSI from regular fire exposures, which is suggested to be involved in the occurrence of overtraining symptoms (Watt et al. 2016).

Ice slurry consumption may also minimise the decrease in cognitive function experienced with dehydration. It is important that emergency responders are vigilant and make accurate decisions, therefore maintaining cognitive function whilst wearing PPE is vital (Barr et al. 2010). Individuals who are dehydrated may take longer to make decisions and have reduced visual vigilance (Cian et al. 2001; Ganio et al. 2011). Dehydration can also increase fatigue and the risk of suffering from an EHI (Casa et al. 2015). Ice slurries may therefore offer the combined benefits of helping individuals to begin a task in a euhydrated state, as well as reducing the physiological strain that they experience.

In contrast to ICE, ARM had no pre-cooling effect, with no reduction in T_{re} expressed. For effective heat loss via the AVA system a temperature gradient is needed. When a gradient is not present, such as when a thermoneutral forearm is immersed in water below 21.5ºC, AVA vasoconstriction occurs and blood flow is reduced (Walløe 2016). It is therefore likely that because pre-cooling was conducted whilst the body was in a thermoneutral state and with 15.8 ± 1.1ºC water, that a rapid sympathetically-mediated vasoconstrictive response occurred, reducing blood flow to the peripheries, and causing blood to pool in the torso and body core (Cheung 2015). Consequently, forearm immersion was unsuccessful at reducing T_{re} prior to exercise. In addition, cold water forearm immersion can reduce both fine and gross manual dexterity, with just 5 min of immersion decreasing fine dexterity by 18% (Cheung et al. 2003; Cheung & McLellan 1998). Forearm pre-cooling may therefore have a negative impact on individuals' abilities to effectively conduct tasks requiring good manual dexterity, such as grasping tools or using the radio to communicate (Lee et al. 2015).

The PCV also did not alter T_{re} at the end of the pre-cooling period (ΔT_{re} PCV: -0.10 ± 0.05 vs. CON -0.04 ± 0.07). Similar findings have been reported by Quod et al. (2008) who found no difference in T_{re} after a 40 min cooling period in a warm environment (33.7 ± 1.0ºC) whilst wearing a PCV. Using a frozen PCV during a 30 min pre-cooling period has been noted to decrease T_c by 0.2 ± 0.2ºC (Brade et al. 2014). However, this investigation used the Drager PCV from room temperature, as instructed by the manufactures product information. Using frozen vests may be impractical when donning PPE multiple times a day is required, as there may be only a limited time period for the vests to refreeze between uses. Purchasing multiple vests for each individual to use, to prevent the need for refreezing, could be costly, especially when ice slurry consumption offers a cheaper and more effective alternative.

Neither PCV nor ARM resulted in a reduced rise in T_{re} during the heat exposure. Creating a thermal gradient between the core and periphery has previously been suggested to enhance heat dissipation and reduce the rate of heat storage (Duffield et al. 2009). Cooling the skin has also been suggested to reduce vasodilation of the peripheral capillary beds, consequently lowering cardiovascular strain (James et al. 2015). This was not present in this investigation, as noted via the similar ΔT_{re} and ΔHR between ARM, PCV and CON. Skin cooling which induces vasoconstriction may mediate any changes in core to skin gradient due to reductions in skin blood flow and could explain why external pre-cooling was not effective.

However, T_{chest} was reduced by the PCV during the heat exposure, suggesting that the vest does produce a local cooling effect. Similar reductions have been reported when using PCV with 24ºC and 28ºC melting points (Gao et al. 2011). Alternatively, House et al (2013) found reduced T_{chest} matched with decreased HR and T_{re} at the end of a 45 min step test. These reductions were seen when using PCV with a melting point of 0ºC and 10ºC. Therefore it can be postulated that using a PCV with a lower melting point may cause a reduction in skin temperature that alleviates thermal strain, however the Drager PCV offers no benefits during a simulated 45 min exposure.

6.5.2 Perceptual Strain

A reduced perception of TS was present during the exercise phase of the protocol for the ARM and ICE condition. ICE decreased TS at 30 min, suggesting that individuals may not experience any reductions in perceptual strain during heat exposures of longer durations. Previous ice slurry studies have also reported reduced TS lasting up to 30 - 35 min during rest (Onitsuka et al. 2015) and running time to exhaustion trials (Siegel et al. 2010; Siegel et al. 2012). A reduction in TS with ice slurry consumption may be caused by the stimulation of visceral thermoreceptors in the stomach (Morris et al. 2017).

During the ARM condition, the reduced TS was not paired with a decrease in T_{re}. This may be dangerous, as individuals may not be aware of the level of thermoregulatory strain that they are experiencing. A reduced TS present without temperature alterations can lead to a 20.7% increase in work output during exercise (Schlader et al. 2011). Therefore, individuals may work harder, or for longer, if they inaccurately perceived a lower thermal strain.

6.5.3 Inflammatory Response

The pre-cooling methods resulted in similar increases in IL-6. Increased IL-6 release post exercise indicates that an acute inflammatory response occurred during the simulated fire exposure (Bluethmann et al. 1994; Fischer 2006). The similar responses in IL-6 reported in this study support the findings of Duffield et al. (2009) who also noted no difference in IL-6 levels following a mixed method pre-cooling strategy and a 40 min intermittent exercise period. Consequently, it can be suggested that despite ICE causing a reduction in the physiological and perceptual strain experienced, the inflammatory response is not affected. However, there are many other predictors of cardiovascular events which have not been measured in this study (Bodor 2016) and therefore it cannot conclusively be claimed that pre-cooling has no effect on the risk of cardiovascular events.

6.5.4 Application

This study is the first to suggest a single bolus of ice slurry, rather than a set $g.kgBM^{-1}$, can be used to reduce T_{re} prior to heat exposure. Using a single bolus allows a bottle of slurry to be easily administered with no prior weighing of either ice or individual, therefore making the pre-cooling method more practical as less time and equipment is needed. Whilst emergency incidents require rapid response with minimal time

prior to exposure, FSI have scheduled exposures and therefore could plan their pre-cooling. Time for ice slurry consumption could be easily allowed during briefing or set up and could be of particular benefit to FSI who complete multiple exposures in a day or week. Despite previous research on pre-cooling within an athlete population, currently pre-cooling is not often used within the UK Fire and Rescue Service, with only small numbers reporting using ice slurry, PCV or forearm cooling. This research offers those in the service a clear recommendation that ice slurry is the best pre-cooling method to choose, with a suggested volume and duration of consumption that is easy to administer. It also provides evidence that forearm cooling and PCV do not reduce T_{re} and could potentially mask physiological responses, consequently these methods are not recommended.

6.5.5 Limitations

This study was conducted in an environmental chamber to allow for control of environmental temperature and exercise intensity. This resulted in a reduced severity of exposure, due to the absence of radiant heat transfer and more extreme temperatures, although similar physiological responses were documented. It is recommended that further research should examine the effect of pre-cooling in a field environment. In addition, the participants involved were university students and consequently anthropometric characteristics may differ compared to FSI. Due to the multiple visits required to complete this study and the geographical spread of training centres, it was decided not to recruit FSI. As a consequence of the participant population and the cost of blood sample analysis, only IL-6 responses were assessed as part of this study.

The impact of dosage warrants further investigation, as the consumption of a greater volume of slurry, such as 750ml, over a longer duration, may have a greater impact on the physiological and perceptual strain experienced. However this may only be possible for some occupations due to time constraints.

6.6 CONCLUSION

Ice slurry ingestion is a practical method of pre-cooling that FSI can use to reduce their T_c, physiological strain, and TS for up to 30 min. Previously used methods of pre-cooling by FSI are not effective at reducing physiological strain, with ARM potentially being dangerous due to the perceptual masking of physiological responses. Overall, it is recommended that 500ml of ice slurry is consumed 15 min prior to a fire exposure to reduce the physiological and perceptual strain experienced, consequently lowering the risk of an EHI.

7 STUDY 4: THE VALIDITY AND RELIABILITY OF A NEW OCCUPATIONAL HEAT TOLERANCE TEST

7.1 ABSTRACT

OBJECTIVES: Heat tolerance tests (HTT) aim to identify those susceptible to heat illnesses and monitor heat adaptations. The standard test (HTT: 2 hr walk at 5 $km.hr^{-1}$ 1% gradient, 40°C 40% RH, in shorts and t-shirt) offers poor ecological validity to uncompensable heat strain. This study aimed to assess the validity and reliability of a new occupational heat tolerance test (HOTT: 40 min at 6 $W.kg^{-1}$ metabolic heat production, 50°C 10% RH, in protective clothing).

METHODS: Seventeen participants (age: 21 ± 3 yrs, mass: 81.7 ± 5.9 kg, height: 180.2 ± 6.6 cm) completed the HTT and HOTT to assess validity; 11 participants (age: 21 ± 2 yrs, mass: 80.48 ± 5.62 kg, height: 178.5 ± 6.3 cm) completed two HOTT trials for reliability assessment. Physiological measures (T_{re}, HR, T_{skin}) were recorded every 5 min, perceptual measures (TS, RPE) were recorded every 10 min. HISI was recorded at pre and post exercise.

RESULTS: Peak T_{re} displayed strong agreement (ICC = 0.86) and low typical error of the measurement (0.19°C) between HTT (38.67 ± 0.42°C) and HOTT (38.58 ± 0.36°C). HOTT and HTT identified the same 9 individuals as heat intolerant. Strong agreement was displayed between the two HOTT trials for peak T_{re} (38.53 ± 0.37°C vs. 38.54 ± 0.35°C, ICC = 0.98), peak HR (182 ± 20 $b.min^{-1}$ vs. 182 ± 21 $b.min^{-1}$, ICC = 0.99) and HISI (24 ± 15 vs. 26 ± 16, ICC = 0.95).

CONCLUSION: The HOTT is a valid and reliable test. It can consistently identify individuals' level of heat tolerance and should be used when assessing individuals wearing protective clothing in high temperatures, to identify and monitor those at greatest risk of heat illnesses.

7.2 INTRODUCTION

In the previous chapters the working practices of FSI have been identified (Chapter 4) and the acute responses to these practices documented (Chapter 5). These studies have highlighted the possible increased risk of heat illness from certain training exposures and identified an acute intervention to reduce physiological and perceptual strain (Chapter 6). However, it is important to note that there are inter-individual differences in responses to heat exposure, as noted in Chapter 5 where on two occasions FSI displayed post T_{re} of ≥ 39°C, despite the average post exposure T_{re} being 37.92 ± 0.31°C. Some individuals are therefore at a greater risk of suffering from a heat illness than others, as a consequence of numerous modifiable and non-modifiable risk factors that can increase an individual's sensitivity to heat (Selkirk & Mclellan 2001; Kenny et al. 2010). Some of these factors can be acute, such as hydration status and recent medicinal intake, however others are chronic (Cheung et al. 2000; Moran et al. 2007).

Body composition, specifically the proportion of BF, and aerobic fitness are both chronic factors that have been suggested to affect heat tolerance. Individuals with a higher percentage of BF have a lower body heat capacity and consequently will experience a faster rate of increase in T_c at a set heat storage. This is due to the lower heat capacity of adipose tissue (2.51 $J.g^{-1}.{}^{o}C^{-1}$) compared to lean tissue (3.65 $J.g^{-1}.{}^{o}C^{-1}$) (Selkirk & Mclellan 2001). Individuals with a lower level of aerobic fitness, identified via maximal oxygen uptake ($\dot{V}O_2$ max), may also be less tolerant during exercise in the heat (Lisman et al. 2014). Aerobic training lowers the T_c threshold that sweating and vasodilation are initiated at, enhancing heat dissipation (Cheung et al. 2000). However, during an uncompensable heat stress situation this may not provide any additional benefit and could instead potentiate the rate of dehydration (Cheung et al. 2000).

Recent findings suggest that when exercise is set at a fixed percentage of $\dot{V}O_2$ max, metabolic heat production ($\dot{H}_{prod}$) has the greatest impact on heat tolerance, explaining ~50% of variation, with BF only having a small impact (2.3%) and $\dot{V}O_2$ max having no impact on change in T_c (Cramer & Jay 2015). When $\dot{H}_{prod}$ is controlled for, BF% has been noted to account for a 2.2ºC higher T_c in the high adiposity group, indicating that BF variations may influence an individual's heat tolerance (Dervis et al. 2015). When comparing individuals of different body mass, it is now recommended that the exercise intensity is set at a fixed metabolic heat production, in watts per kilogram, rather than at a fixed $\dot{V}O_2$ max, to prevent systematic variations in T_c and allow for an unbiased assessment of heat tolerance (Jay et al. 2011; Cramer & Jay 2014).

Due to the multi-faceted nature of heat tolerance, it is difficult to predict how individuals will respond from predisposing factors alone. A heat tolerance test (HTT) is therefore needed to successfully identify individuals who may be at a higher risk of a heat illness. A HTT can also be used to recommend when individuals can return to work post illness and establish if an individual has become acclimatised to heat exposure and is therefore better able to cope with the environment (Moran et al. 2007). HTT have been used within the military and with athletes for these reasons for many years.

The HTT most commonly used within occupational settings was developed for the Israeli Defence Force to evaluate military personnel's heat tolerance (Moran et al. 2004; Moran et al. 2007). The HTT involves a

2 hour walk in 40ºC and 40% RH at 5 km.hr^{-1} with a 2% gradient (Moran et al. 2007). According to the guidelines from Moran et al (2007), individuals with an end T_{re} exceeding 38.5ºC or a HR exceeding 145 b.min^{-1} should be classified as heat intolerant. Tolerant individuals are expected to have a T_{re} of 38.0 ±0.3ºC and HR of 120 ± 15 b.min^{-1}. They are also expected to display a plateau in T_c (Moran et al. 2004), with a difference of greater than 0.45°C in the last 60 min of the test suggested to distinguish heat intolerance in cases where T_{re} and HR provide borderline responses (Druyan et al. 2013). The further an individual deviates from the heat tolerant classification, the more pronounced their heat intolerance is. The HTT offers good construct validity, successfully distinguishing those who are heat tolerant from those who are not (Moran et al. 2007). Although, research does suggest that the use of a continuum to interpret heat tolerance, rather than dichotomous groupings, may better reflect the range and progression of individual responses (Taylor & Cotter 2006; Mee et al. 2015).

However, this HTT offers little ecological validity when wearing protective clothing. During the 2 hr test individuals exercise in shorts and t-shirts, allowing heat dissipation via evaporation to occur. This compensable heat stress environment is therefore different to that experienced with PPE. In addition, the use of a set exercise intensity, rather than a set metabolic heat production, may result in wide variations in T_c responses and prevent comparisons between individuals of differing biophysical characteristics from being made in an unbiased way. It is also highly unlikely that FSI will be exposed to high temperatures for 2 hr, as they are limited to the volume of gas available in their BA, and are often exposed for up to 40 min (Eglin 2007). To accurately assess heat tolerance in relation to occupational function, a new occupational HTT was designed to more closely replicate the type of heat exposure experienced.

This study aims to assess the validity of the new heat occupational tolerance test (HOTT) in comparison to the HTT and also evaluate the reliability of the HOTT. It was hypothesised that (1) T_{re} responses would be similar between the HTT and HOTT and (2) that physiological and perceptual responses would be similar at the end of both HOTT trials.

7.3 METHOD

7.3.1 Participants

Eighteen male participants were recruited (age: 21 ± 3 yrs, body mass: 81.3 ± 5.9 kg, height: 180.0 ± 6.5 cm) from the University of Brighton. Participants were given the option to select if they wished to be involved with the validity or reliability testing, with some individuals opting to complete both. Seventeen participants (age: 21 ± 3 yrs, body mass: 81.7 ± 5.9 kg, height: 180.2 ± 6.6 cm) completed the validity trials and eleven participants (age: 21 ± 2 yrs, body mass: 80.5 ± 5.6 kg, height: 178.5 ± 6.3 cm) completed the reliability trials. Participants were required to provide informed written consent and complete a medical questionnaire prior to taking part in the study (section 3.1). The study was approved by the University of Brighton Ethics Committee (section 3.1).

Participants were requested to avoid alcohol, caffeine, heat exposure > 25°C, and exhaustive exercise 24 hours prior to taking part, with adherence checked via a questionnaire (section 3.6).

7.3.2 Experimental Design

Testing sessions followed a randomised cross over design. Participants were asked to complete two HOTT for the reliability sessions and a HOTT and HTT for the validity sessions. Those involved in both the validity and reliability parts of this study were only required to complete 2 HOTT in total. Prior to their first session participants visited the laboratories to try on PPE, select sizes, and ensure they felt comfortable in the clothing. All trials were separated by a minimum of 5 days, to prevent heat adaptations, and were conducted in the morning beginning between 7:00am and 9:00am to control for circadian rhythms (Drust et al. 2005). Participants were also requested not to be exposed to heat > 25°C in the intervening days (Périard et al. 2015). Trials were completed during UK Autumn and Winter months (October – February).

7.3.3 HOTT

Each HOTT trial began with a 10 min rest period (23.3 ± 1.6°C, 34 ± 11% RH) whilst wearing fire protective clothing (boiler suit, trousers [Ballyclare Special Products Ltd.], jacket [Ballyclare Special Products Ltd.], boots [9005 GA, Jolly Scarpe, USA], fire hood [MSA Gallet, Bellshill, UK], helmet [F1SF, MSA Gallet, Bellshill, UK], and gloves [Firemaster 3, Southcombe Brothers Ltd, Somerset, UK]). Participants then entered the heat chamber (50.0 ± 1.1°C, 13 ± 2% RH) where they walked continuously for 40 min. During the first 15 min speed was altered every 5 min (starting from 4.5 $km.hr^{-1}$) to identify the correct speed needed for the participant to be working at a metabolic heat production of 6 $W.kg^{-1}$. Speed was then maintained throughout with only small adjustments of 0.2 $km.hr^{-1}$ made when necessary to maintain the desired heat production. This process of speed adjustment occurred in both HOTT sessions, although the maximum speed difference that occurred between trials was 0.2 $km.hr^{-1}$. A $\dot{H}_{prod}$ of 6 $W.kg^{-1}$ was selected as pilot testing identified that this workload was the greatest reached by individuals at the end of the HTT.

7.3.4 HTT

During the HTT trials participants rested for 10 min (22.2 ± 2.0°C, 31 ± 5 RH) whilst wearing their own shorts, cotton t-shirt and trainers. They then entered the heat chamber (40.2 ± 0.2°C, 40.7 ± 1% RH) and completed a 120 min walk at 5 $km.hr^{-1}$ on a 2% gradient, as specified by Moran et al., (2007).

7.3.5 Measures

Participants were asked to attend the laboratory in a euhydrated state, which was confirmed as described in section 3.6.1.

Nude body mass was recorded prior to and post each trial (section 3.7.2). Mass whilst clothed was also recorded after the resting period in both trials to allow for metabolic heat production calculation. T_{re}, HR,

and T_{skin} (recorded via skin thermistors) were measured at the end of the resting period and every 5 min throughout the exercise period (section 3.8 and 3.8.4).

Perceptual measures were recorded by RPE and TS at the end of the rest period and then every 10 min during exercise. HISI was also recorded prior to and post heat exposure (section 3.10)

7.3.6 Gas Analysis

Ventilatory gases were collected throughout the HOTT and during the first 40 min and last 20 min of the HTT. The face mask was removed between gas analysis periods. Gases were analysed using 30 sec averaging from a Metalyzer Sport analyzer (section 3.8.3). $\dot{V}O_2$ and RER each minute were used to calculate $\dot{H}_{prod}$.

7.3.7 Derivative Calculations

H_{prod} was calculated as described in section 3.5. T_{skin} was calculated using the measurements taken from the contact skin thermistors, as per section 3.8.2. Physiological strain index was calculated as per the equation in section 3.8.5. SR was calculated at the end of heat exposure (section 3.8.6).

7.3.8 Statistical Analysis

Data were analysed using IBM SPSS 22 and reported as mean ± standard deviation (3.11). Data were checked for normality using the Shapiro-Wilk method (section 3.11). Numerous reliability and validity statistics were conducted as described in section 3.11.3. ICC (95% CI) were calculated using an absolute agreement, two way mixed-effects model, as a measure of retest correlation between HOTT sessions and a measure of correlation between HOTT and HTT. An ICC of > 0.90 was classified as a high correlation, 0.70 - 0.80 as moderate, and below 0.70 as low (Vincent & Weir 1995). TEM is presented in both absolute values and values relative to the respective means as a coefficient of variation (CV). A CV% of < 10% was considered acceptable (Stokes 1985). Bland-Altman plots with 95% LOA were created with the individual participant differences between the trials plotted against the respective individual means. Acceptable mean bias levels and 95% LOA for bland-altman plots for reliability assessment were selected a priori based on the reliability mean bias and LOA from the running heat tolerance test (T_{re} -0.04ºC (-0.41ºC, 0.33ºC), T_{skin} 0.01ºC (-0.38ºC, 0.40ºC), HR 1 b.min^{-1} (-8 b.min^{-1},6 b.min^{-1}) PSI -0.1 (-0.93, 0.72), SR -0.13 L.hr^{-1}, (-0.49 L.hr^{-1}, 0.23 L.hr^{-1}), RPE 0 (-2,2), TS (0 (-1,1)) (Mee et al. 2015). Acceptable mean bias levels for bland-altman plots for validity comparisons were identified a priori based on the smallest detectable changes in heat tolerance noted from heat acclimation studies (T_{re} < 0.2ºC, HR < 5 b.min^{-1}, T_{skin} < 0.5ºC, PSI < 1, SR < 0.5 L.hr^{-1}, TS < 0.5, RPE < 1) (Mee et al. 2015; Gibson et al. 2015; Tyler et al. 2016). LOA criteria could not be identified a priori for validity comparisons as no previous validity comparisons to HTT have been performed. To identify differences between trials for all measures, paired sample T-tests were also conducted. Statistical significance was set at $p < 0.05$ (3.11.6).

To identify differences between tolerance groups, a two way mixed method ANOVA was performed (section 3.11.7.3). Follow up Bonferroni corrected T-tests were conducted to identify if differences were present at 20 min and 40 min, with the corrected alpha level of $p < 0.025$ used.

7.4 RESULTS

7.4.1 Validity of Variables

All participants were in a hydrated state at the beginning of the HTT (U_{col} 2 ± 1, U_{osm} 307 ± 203 mOsm.kgH_2O^{-1}, U_{spg} 1.009 ± 0.006) and HOTT (U_{col} 2 ± 1, U_{osm} 211 ± 136 mOsm.kgH_2O^{-1}, U_{spg} 1.006 ± 0.004) with no differences in U_{col} ($p = 0.79$), U_{osm} ($p = 0.072$), or U_{spg} ($p = 0.096$) between trials. The mean speed needed to achieve 6 W.kg^{-1} during the HOTT was 5.1 ± 0.4 km.hr^{-1}, ranging from 4 – 6 km.hr^{-1}. The mean H_{prod} achieved at the end of the HTT was 4.8 ± 1.0 W.kg^{-1}, ranging from 2.9 – 6.3 W.kg^{-1}.

7.4.1.1 Physiological Measures

Values of ICC, TEM and LOA for key physiological and perceptual measures are presented in Table 7.1. Moderate to weak correlations were present for peak T_{re} (ICC = 0.86), ΔT_{re} (ICC = 0.68), peak HR (ICC = 0.58), change in HR (ΔHR) (ICC = 0.55) and peak PSI (ICC = 0.64) between HTT and HOTT. T_{re} also demonstrated a low TEM and CV. No correlations were presented for peak T_{skin} (ICC = 0.09) and SR (ICC = 0.19). Figure 7.1 demonstrates acceptable mean bias and LOA of peak T_{re} and peak PSI between trials. Figure 7.1 also presents a large mean bias for peak HR.

In addition, paired samples T-tests revealed no differences between HTT and HOTT for peak T_{re} ($p = 0.182$), ΔT_{re} ($p = 0.098$), and ΔT_{skin} ($p = 0.288$). Differences were present for peak HR ($p < 0.001$), ΔHR ($p = 0.001$), peak PSI ($p = 0.023$), peak T_{skin} ($p < 0.001$), and SR ($p = 0.003$).

7.4.1.2 Perceptual Measures

Weak to moderate correlations were present for RPE (ICC = 0.58), and HISI (ICC = 0.87), with no correlation present for TS (ICC = 0.36). T-tests revealed no differences in HISI scores between trials, ($p = 0.481$). Differences were present for RPE ($p = 0.044$) and TS ($p = 0.030$) between HTT and HOTT.

Table 7.1 *Validity statistics between HTT and HOTT.*

	Peak T_{re} (°C)	**Peak HR (b.min^{-1})**	**Peak T_{skin} (°C)**	**Peak PSI**	**SR (L.hr^{-1})**	**Peak RPE**	**Peak TS**	**Peak HISI**
HTT	38.67 ± 0.42	157 ± 20	36.50 ± 0.79	7.17 ± 1.50	0.94 ± 0.17	14 ± 3	6 ± 1	23 ± 17
HOTT	38.58 ± 0.36	179 ± 18	38.35 ± 0.66	7.97 ± 1.20	1.30 ± 0.42	16 ± 2	7 ± 0.5	25 ± 13
TEM (CV%)	0.19 (0.49)	11 (6.56)	0.65 (1.74)	0.92 (12.20)	0.29 (26.22)	2 (14.23)	0.5 (8.88)	7 (30.47)
Mean bias (LOA)	0.09 (-0.43, 0.62)	-22 (-52, 9)	-1.85 (-3.66, -0.05)	-0.74 (-3.35, 1.76)	-0.36 (-1.17, 0.46)	-2 (-8, 4)	-0.50 (-2, 1)	-2 (-22, 19)
ICC (95% CI)	0.86 (0.63,0.95) $p < 0.001$	0.58 (-0.25,0.87) $p = 0.001$	0.09 (-0.13,0.41) $p = 0.217$	0.64 (0.06,0.87) $p = 0.011$	0.19 (-0.42,0.63) $p = 0.258$	0.58 (-0.04,0.84) $p = 0.027$	0.36 (-0.42,0.74) $p = 0.147$	0.87 (0.64,0.95) $p < 0.001$

T_{re} = rectal temperature, HR = heart rate, T_{skin}, = skin temperature, PSI = physiological strain index, SR = sweat rate, RPE = rating of perceived exertion, TS = thermal sensation, HISI = heat illness symptoms index.

Table 7.2. *Reliability statistics between HOTT1 and HOTT2.*

	Peak T_{re} (°C)	**Peak HR (b.min^{-1})**	**Peak T_{skin} (°C)**	**Peak PSI**	**SR (L.hr^{-1})**	**Peak RPE**	**Peak TS**	**Peak HISI**
HOTT1	38.54 ± 0.37	182 ± 20	38.54 ± 0.54	8.08 ± 1.24	1.21 ± 0.46	16 ± 3	7.5 ± 0.5	24 ± 15
HOTT2	38.55 ± 0.34	182 ± 21	38.51 ± 0.38	8.22 ± 1.23	1.27 ± 0.39	16 ± 3	7.5 ± 0.5	25 ± 16
TEM (CV%)	0.08 (0.20)	2 (1.26)	0.29 (0.75)	0.22 (2.70)	0.12 (9.90)	1 (5.01)	0.5 (4.32)	5 (20)
Mean bias (LOA)	0.02 (-0.20 0.23)	0 (-6, 7)	-0.03 (-0.82 -0.77)	-0.14 (-0.47, 0.75)	0.05 (-0.29, 0.39)	0 (-2, 2)	0 (-1, 1)	1 (-13, 15)
ICC (95% CI)	0.98 (0.92,0.99), $p < 0.001$	0.99 (0.98,1.00), $p < 0.001$	0.78 (0.14,0.94), $p = 0.016$	0.98 (0.93,0.99), $p < 0.001$	0.96 (0.85,0.99), $p < 0.001$	0.96 (0.86,0.99), $p < 0.001$	0.87 (0.51,0.97), $p = 0.002$	0.95 (0.80,0.99), $p < 0.001$

T_{re} = rectal temperature, HR = heart rate, T_{skin}, = skin temperature, PSI = physiological strain index, SR = sweat rate, RPE = rating of perceived exertion, TS = thermal sensation, HISI = heat illness symptoms index.

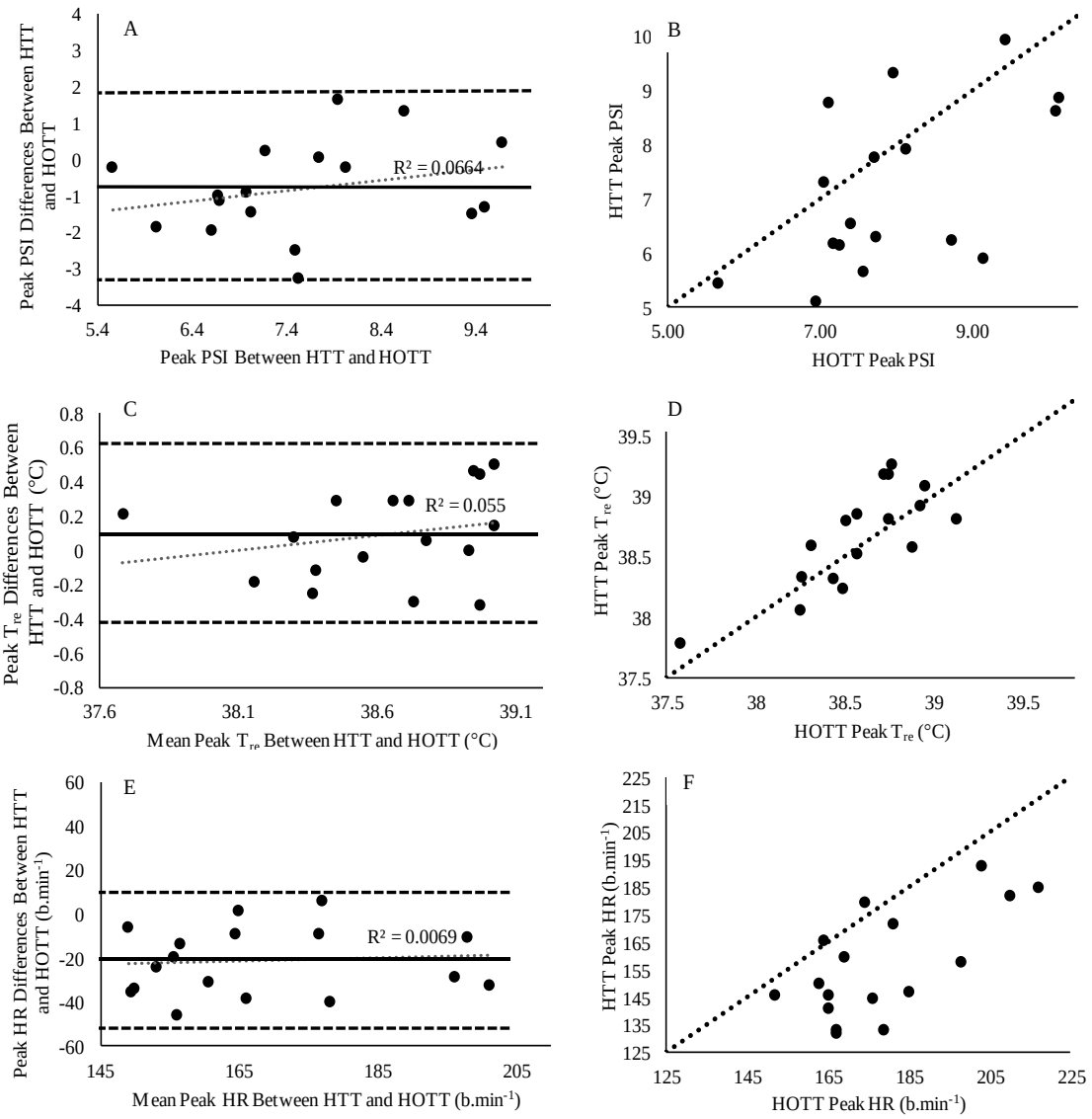

Figure 7.1 *Bland-Altman plots with 95% LOA and line of equality plots for peak physiological strain index (PSI) (A, B, respectively), peak rectal temperature (T_{re}) (C, D), and peak heart rate (HR) (E, F) between HOTT and HTT.*

7.4.2 Validity of Tolerance

Based on the T_{re} heat intolerance criteria set by Moran et al. (2007) (T_{re} > 38.5 ºC), 11 participants were classified as heat intolerant in both HTT and HOTT. All of the 11 also exhibited a ΔT_{re} of > 0.45°C in the final 60 min of the HTT, as suggested by Druyan et al. (2013). One participant was classed as heat intolerant in HTT but not in HOTT. Only one participant met the T_{re} criteria for heat tolerance ($T_{re} \leq 38.0$ ºC), with a T_{re} of 37.58°C in the HOTT and 37.79°C in the HTT. Of the remaining four participants who sat between 38.0ºC and 38.5ºC, two had a HR above the heat intolerance criteria of 145 b.min^{-1}. All participants displayed a peak HR above 145 b.min^{-1} in the HOTT.

When grouped by classification in the HOTT, heat intolerant participants had a greater peak T_{re} of 38.78 ± 0.19°C compared to 38.22 ± 0.33°C for those below the criteria set point ($p < 0.001$). A two way mixed

method ANOVA revealed a significant difference in ΔT_{re} between the groups (p = 0.038). The heat intolerant group exhibited changes at half way and the end of the walk test of 0.36 ± 12°C and 1.45 ± 0.22°C vs. 0.05 ± 0.12°C and 1.02 ± 0.27°C for those below the criteria point (p < 0.001, p = 0.003, respectively), as displayed in Figure 7.2.

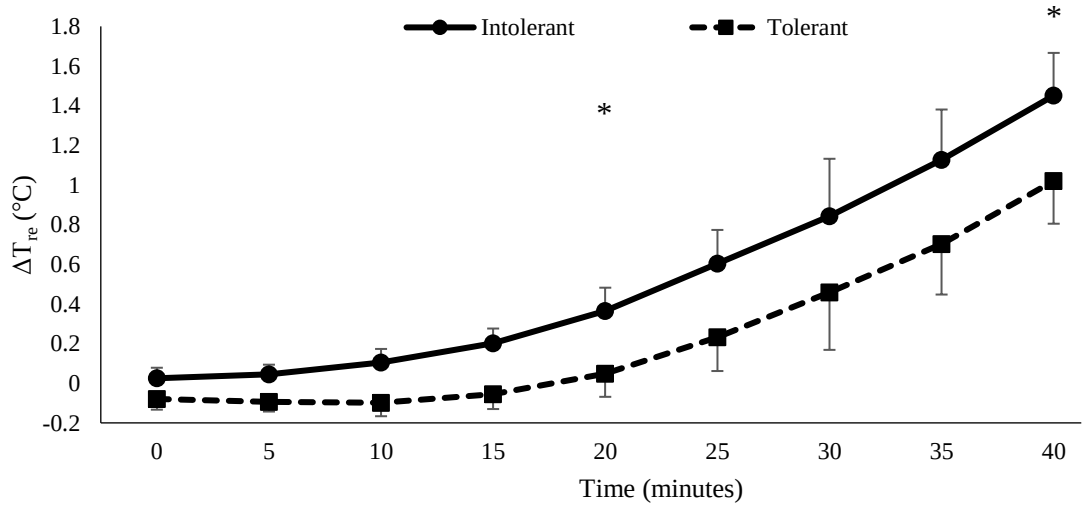

Figure 7.2 *Mean ± SD Change in rectal temperature (ΔT_{re}) for intolerant (n = 11) vs tolerant (n = 6) individuals in HOTT, classified by Moran (2004) 38.5°C criteria for intolerance. * denotes significant differences between the groups, p < 0.025.*

7.4.3 Reliability

Hydration requirements were met by all participants at the start of HOTT1 (U_{col} 2 ± 1, U_{osm} 211 ± 136 mOsm.kgH_2O^{-1}, U_{spg} 1.006 ± 0.004) and HOTT2 (U_{col} 2 ± 1, U_{osm} 299 ± 220 mOsm.kgH_2O^{-1}, U_{spg} 1.011 ± 0.012), with no differences between trials for U_{col} (p = 0.64), U_{osm} (p = 0.37), U_{spg} (p = 0.13). Table 7.2. presents values of ICC, TEM and LOA for key physiological and perceptual measures for the reliability between the two trials.

7.4.3.1 Physiological Measures

Moderate to strong correlations were observed between HOTT1 and HOTT2 for peak T_{re} (ICC = 0.98), ΔT_{re} (ICC = 0.77), peak HR (ICC = 0.99), ΔHR (ICC = 0.94), peak T_{skin} (ICC = 0.78), peak PSI (ICC = 0.98), and SR (ICC = 0.96). All physiological measures demonstrated acceptable TEM, CV and mean bias between trials. See Figure 7.3 for mean bias and LOA, and line of equality for peak T_{re}, peak HR, and peak PSI between HOTT trials.

T-tests indicated that there were no differences between HOTT1 and HOTT2 in peak T_{re} , ΔT_{re}, peak HR, ΔHR, peak T_{skin}, $\Delta T_{skin,}$ peak PSI, and SR (p > 0.05).

7.4.3.2 Perceptual Measures

Strong correlations were observed between HOTT1 and HOTT2 in peak RPE (ICC = 0.96), TS (ICC = 0.87), and HISI (ICC = 0.95). Acceptable CV and mean bias were present between trials in RPE and TS. There were no differences in peak RPE, TS, and HISI ($p > 0.05$).

7.4.3.3 Heat Tolerance

All HR exceeded the heat tolerance criteria set by Moran et al. (2007). When classified by peak T_{re} five participants were heat intolerant and all individuals were classified the same in both HOTT1 and HOTT2. One participant withdrew due to nausea at 30 min in both HOTT and was therefore also classified in the heat intolerant group, making six in total. No individual met the heat tolerant criteria of 38.0°C.

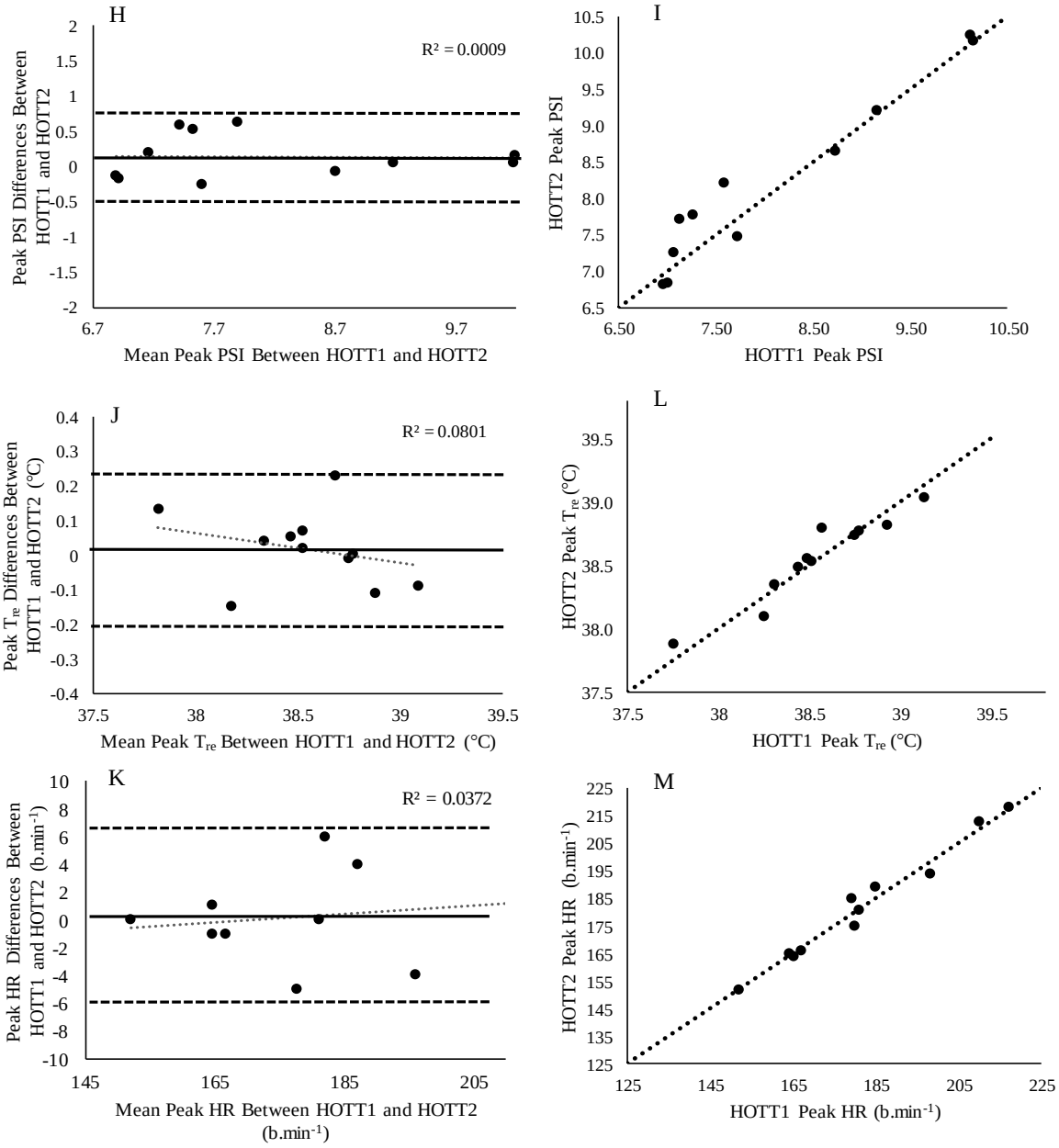

Figure 7.3. *Bland-Altman plots with 95% LOA and line of equality plots for peak physiological strain index (PSI) (H, I, respectively), peak rectal temperature (T_{re}) (J, K), and peak heart rate (HR) (L, M) between HOTT1 and HOTT2.*

7.5 DISCUSSION

The aim of the study was to assess the validity and reliability of the HOTT, a heat tolerance test purposefully designed to replicate the uncompensable heat strain experienced by FSI, and allow for inter-individual comparisons using a set $\dot{H}_{prod}$. The HOTT was able to replicate HTT T_{re} responses and results were replicated following a 5 day period. Consequently, hypothesis (1) and (2) can both be accepted.

7.5.1 Validity of Variables

Peak T_{re} displayed the greatest level of agreement between HTT and HOTT, with a TEM of 0.19°C (0.49%), a strong ICC (0.86), and a small mean bias 0.09°C. It is elevated T_{re}, combined with neuropsychiatric impairment, that is used to diagnose heat illnesses, with a T_{re} of 40.5°C indicating heat stroke (Casa et al. 2015) and therefore is one of the most important predictors of heat tolerance. Being able to predict if an individual is likely to suffer an EHI is a key reason why heat tolerance tests may be used. For FSI, this could indicate which individuals need to have closer monitoring, work on altering modifiable risk factors, or acclimation periods, to reduce the risk of heat illness.

HR showed a moderate level of agreement between HTT and HOTT, however HOTT had a bias of 22 $b.min^{-1}$ greater than HTT. The additional cardiovascular strain was caused by the environmental conditions, as HR increased to raise skin blood flow and facilitate evaporative heat loss (Stewart et al. 2014). However, the PPE reduces the ability for individuals to dissipate heat as sweat evaporation is prevented (Petruzzello et al. 2009). In addition, cardiovascular strain was also exacerbated by the minimal T_{skin} to T_{re} gradient in the HOTT (0.23ºC) which would have elevated skin blood flow above that of the HTT, which had a greater T_{skin} to T_{re} gradient (2.17ºC) (Cheuvront et al. 2010). Therefore HR continued to rise above that noted in the HTT in an attempt to regain thermal balance, but the uncompensable environment prevented this from occurring.

The strong agreement between HOTT and HTT for HISI, suggests that the HOTT induces a similar subjective symptoms response to HTT, alongside the similar T_{re} response. Wearing PPE and maintaining a $\dot{H}_{prod}$ of 6 $W.kg^{-1}$, instead of a continuous 5 $km.hr^{-1}$ walk, enabled a greater rate of rise in both physiological and perceptual measures in the HOTT, despite the test being 80 min shorter in duration. The rate of rise for T_{re} was 0.03 ± 0.01°C.min^{-1} in the HOTT vs. 0.01 ± 0.00°C in the HTT. Setting the test by $\dot{H}_{prod}$ rather than by speed allows comparisons to be made between individuals of a heterogenous group and also negates the effect of training status and small fat mass fluctuations during intra-individual comparisons (Cramer & Jay 2014).

7.5.2 Validity of Tolerance

Based on Moran's T_{re} criteria 16 of 17 participants were classified the same in both the HOTT and HTT. The HOTT was also successful at classifying individuals as heat intolerant or not, with statistical differences in peak T_{re} and ΔT_{re} present between the groups. Peak T_{re} was 0.56°C greater in the intolerant group, which is smaller than the 0.81°C difference in groups noted by Moran et al. (2004). A smaller difference between

groups is likely due to individual participant differences, as this study found little bias (0.09°C) between the two tolerance tests. A greater proportion of participants were classified as heat intolerant than previously noted (Moran et al. 2004), it is postulated that this may be due to the use of military individuals in previous studies, who are likely to have a superior level of aerobic fitness to the students who participated in this study. In addition, the participants in this study were not heat acclimated, with testing conducted during Autumn and Winter months, and consequently may be more likely to have an increased sensitivity to heat than participants of previous research conducted in hotter climates (Epstein 1990; Druyan et al. 2012).

Although Moran et al (2007) take a dichotomous approach to heat tolerance, in that individuals are either heat tolerant or heat intolerant, six participants displayed T_{re} responses that fitted into neither group. This indicates that heat tolerance should instead be viewed as a continuum, as previously suggested by Mee et al. (2015). Those individuals classified in neither group would therefore sit in the middle of the continuum. Individuals can move along the continuum by becoming more or less tolerant to the heat. This may be of particular use in an occupational setting as a way of identifying individuals who are at risk and monitoring their progress. Consequently, a continuum based approach is advised when interpreting individuals' responses.

7.5.3 Reliability

The HOTT demonstrates good reliability, with those who were heat intolerant distinguished as such in both HOTT1 and HOTT2. Furthermore, key measures of heat acclimation, such as peak T_{re}, peak HR, peak T_{skin} and SR (Moran et al. 2007; Sawka et al. 2011), all demonstrate strong correlations between the trials, with TEM and CV suggesting low intra-individual variability.

There has been no previous assessment of the reliability of the HTT and so comparison between the tests on this matter cannot be made. However the TEM and CV of peak T_{re}, (0.08°C, 0.2%), peak HR (2 $b.min^{-1}$, 1%), and SR (0.12 $L.hr^{-1}$, 9.9%) are similar to that reported by Mee et al., (2015) when assessing the reliability of a 9 $km.hr^{-1}$ 30 min running heat tolerance test (peak T_{re}: 0.13°C, 0.34%, peak HR: 2 $b.min^{-1}$, 1%, SR: 0.16 $L.hr^{-1}$, 9%). The findings are also in line with the T_{re} TEM (CV%) of 0.20°C (0.3%) (Hayden et al. 2004), 0.14 °C (0.4%) (Willmott et al. 2015), and aural temperature of 0.10°C (0.6%) (Brokenshire et al. 2009) previously reported from different cycling heat exposure tests of 30 - 60 min in duration. The HOTT is therefore a test that offers the same levels of reliability as other tolerance tests and consequently is a good alternative for use in an occupational setting where PPE is worn, as it better replicates the conditions experienced.

7.5.4 Application

The HOTT can be used to assess heat tolerance, with interpretation of responses along a continuum recommended. From an application perspective, a colour coded continuum could be proposed as it may be easily interpretable, with those exhibiting responses of < 38.0ºC sitting in a "green zone", individuals who are 38.0ºC to 38.5ºC in a "yellow zone", and > 38.5ºC in a "red zone". Those individuals who are just

beyond the “green zone” into the “yellow zone” would then be viewed as better able to cope in the heat than an individual at the far end of the “red zone”. Individuals who present further into the “red zone” can then be selected for acute and chronic interventions to improve their ability to cope in the heat. In addition, these individuals could then be monitored to see if they shift to the left to the early “red zone” or “yellow zone” with interventions, which using dichotomous heat tolerance and intolerance criteria would not reflect. For the Fire and Rescue Service it is often reaching a T_c of 39.0ºC which is a cut off point for exposure and therefore knowing a FSI or FF is able to tolerate an uncompensable environment, for the duration a BA lasts, without reaching this temperature is of key importance.

The HOTT also uses a set H_{prod} which controls for changes in fitness level that may occur, therefore offering a more valid measurement of heat tolerance alterations. From a research perspective this will also allow inter-individual comparisons to be made and consequently gives the HOTT a clear advantage over the HTT, which uses a fixed walking speed, for identifying and monitoring changes in the heat tolerance of individuals. The ability to compare individuals within a crew may enable resources, time or funding to be specifically targeted to those further towards the “red zone” and most at risk of a heat illness. Moreover, the HOTT lasts for 40 min instead of 120 min, making it more convenient to administer. In addition to use with FSI, the HOTT could also be employed to identify new FF recruits’ heat tolerance, as new recruits are at particular risk of heat illnesses during their first fire training sessions, having had no prior experience of the conditions. Currently, FSI are unable to predict which individuals within a cohort may be at greater risk. The HOTT may be used within the Fire and Rescue Service to monitor those identified as having a low tolerance to heat, with the aim to move them along the continuum. In any occupation involving physical activity in PPE, the HOTT could be used to safely monitor an individual’s return to work following an occurrence of EHI.

7.5.5 Limitations

No females were used as part of this study. It has previously been suggested that the criteria set points for tolerance using the HTT need to be re-evaluated when testing females, due to the possible thermoregulatory differences between sexes (Druyan et al. 2012). However, from an occupational perspective the heat load experienced during an exposure and the Fire and Rescue Service safety T_c is the same regardless of sex. Furthermore, as sex is one of a number of risk factors for EHI, the use of the same continuum for both males and females in a group will allow for identification of those most at risk.

In addition, it was not within the scope of this research to investigate the impact that heat acclimation or others factors that may impact heat tolerance has on the response of individuals to the HOTT. Future research should investigate how interventions may progress tolerance responses along the continuum. Individuals did not exercise at 6 $W.kg^{-1}$ from the beginning of the test, with the first 15 min varying for each individual as speed was adjusted to achieve the desired $\dot{H}_{prod}$. However, this enables individuals to only have to complete one visit to the laboratory for a HOTT to be conducted, therefore increasing the practicality of the protocol. Whilst the HOTT offers a more ecologically valid comparison to uncompensable heat stress environments, it does require specialist equipment, which could hinder its use

where research equipment is not accessible. Consequently, further research should be conducted to develop a simplified field version of the test that can be applied when resources are minimal.

7.6 CONCLUSION

This study demonstrates that the HOTT is a reliable alternative to the HTT. The test offers a closer representation of the type of heat strain experienced by FSI and others who wear PPE. It is also practically suitable, in that only one visit is required and the test is of a much shorter duration than the HTT. In addition, the use of a set H_{prod} to determine the speed of the test enables both intra and inter individual comparisons to be conducted. Therefore, it is suggested that the HOTT is used when assessing the heat tolerance of FSI and FF and could be used in other occupations where individuals wear protective clothing and work in an uncompensable heat stress environment.

8 STUDY 5: HEAT TOLERANCE OF FIRE SERVICE INSTRUCTORS

8.1 ABSTRACT

OBJECTIVES: FSI experience fire exposures a median of 13 ± 8 times a month; consequently they may develop an acclimatised state. However, the chronic immunological implications of heat acclimation are yet to be understood. This study aims to establish if FSI exhibit an increased heat tolerance and altered immunological response to heat exposures, compared to non-exposed individuals. The study also aims to identify if heat tolerance is related to symptoms of ill health.

METHODS: Twenty-two participants were recruited: 11 FSI (age: 41 ± 7 yrs, body mass: 77.4 ± 12.2 kg, height: 174.1 ± 8.2 cm) and 11 non-exposed controls (CON) (age: 41 ± 7 yrs, body mass: 75.9 ± 12.2 kg, height: 177.0 ± 8.1 cm). Participants completed a 40 min HOTT exercising at 6 $W.kg^{-1}$ (50.0 ± 1.0ºC, 12.3 ± 3.3% RH) on two occasions, separated by 2 months. Physiological and perceptual measures were collected throughout and venous blood samples were collected prior to and post exposure.

RESULTS: FSI displayed significantly reduced peak T_{re} (-0.42ºC), ΔT_{re} (-0.33ºC), and TS (-1.0) and increased SR (+0.25 $L.hr^{-1}$) at the end of the HOTT compared to CON ($p < 0.05$). FSI exhibited similar responses to the HOTT as CON for all haematological variables. However, resting IL-6, IL-1β, and IgG were significantly greater in FSI than CON. There was no difference in responses following the 2 month working period. FSI peak T_{re} was negatively correlated with symptoms of ill health ($r_{pb} = -0.473$, $p = 0.026$) and the number of fire exposures in the previous 2 weeks ($r_s = -0.589$, $p = 0.004$).

CONCLUSION: Despite increased heat tolerance compared to non-exposed individuals, FSI may develop a maladaptation to repeated fire exposures, with elevated resting cytokine levels and an increased prevalence of ill health.

8.2 INTRODUCTION

Individuals who are more tolerant to heat exposure, being better able to defend their T_c during physical activity in hot environments, are at a reduced risk of a heat illness (Kazman et al. 2013; Lisman et al. 2014). Mechanistically, this is related to the occurrence of thermotolerance, the cellular adaptations to heat stress by which the stimulation of heat shock proteins confers cytoprotection to subsequent thermal and non-thermal stresses (Kregel 2002; Tetievsky et al. 2008; Gibson et al. 2016). Heat shock proteins facilitate the maintenance of cellular integrity and homeostasis, acting as chaperones to aid intracellular transportation and folding of proteins, regulate apoptosis, and aid recovery at a cellular, organ and whole body level (Gibson et al. 2016; Henstridge et al. 2016). These cellular changes are associated with lower T_c and reduced inflammatory cytokine production during thermal stress (Kuennen et al. 2011), as well as an individual's overall ability to tolerate heat (Moran et al. 2006). As outlined in Chapter 7 and section 2.4.1 there are also numerous predisposing factors that can affect heat tolerance. These can either be permanent, such as sex, age, congenital diseases, and large areas of scarred skin, or transient, such as an individual's aerobic fitness, body composition, or hydration status (Moran et al. 2007; Lisman et al. 2014).

Repeated heat exposures that involve sufficient thermal impulses can also lead to both cellular and physiological adaptations, that can improve an individual's ability to cope and perform in a hot environment (Garrett et al. 2011; Périard et al. 2015). The process to acquire these adaptations is referred to as heat acclimation when conducted in a controlled/laboratory setting, or heat acclimatisation when they occur through natural interaction with the environment (Sawka et al. 2011). A reduced HR (-16 $b.min^{-1}$) and T_c (-0.2 - 0.34ºC), with an increased SR (+0.2 - 0.36 $L.hr^{-1}$) and improvement in performance, have been noted as the classic markers of heat acclimation (Sawka et al. 2011; Tyler et al. 2016). These markers coincide with improved thermal perception and comfort, reduced sodium loss, increased stroke volume, improved skin blood flow and increased blood volume (Sawka et al. 2011). The combination of these adaptations result in an increased thermoregulatory capacity, consequently improving the ability to preserve homeostasis and better defend against heat gain. Acclimation protocols are commonly implemented with athletes to improve their performance in endurance or team sports and to reduce the risk of EHI (Cheung et al. 2000; Lorenzo et al. 2010; Chalmers et al. 2014; James et al. 2017).

Acclimation protocols used with athletes to stimulate adaptations can vary in duration from 4 - 24 days and sessions may include exercising at a set work rate, a self-selected intensity, or a controlled hyperthermic level of 38.5°C (Armstrong et al. 2004; Gibson et al. 2015; Tyler et al. 2016). The set temperature for isothermic acclimation is also considered as the threshold T_c needed to promote the heat shock protein response and therefore stimulate thermotolerance adaptations (Gibson et al. 2016). Acclimation frequency can vary from one exposure per day, to twice daily, or intermittent exposure, although intervals of one week are too long to induce adaptations (Gill & Sleivert 2001; Willmott et al. 2016). To optimise adaptations 8 - 14 days of controlled hyperthermic heat exposures are recommended. However, up to 75% of adaptations can occur within 4 - 7 days of exposure (Pandolf 1998). Acclimation sessions are used by athletes prior to competition in hot environments and have been suggested as a way to prepare industrial workers and individuals in the military and the fire service for work in hot occupational settings (Jay & Kenny 2010; Ashley et al. 2015).

FF have previously been reported not to develop an increased tolerance to heat exposure (Wright et al. 2013). Due to the nature of the occupation, FF experience fewer live fires than FSI, as noted in Chapter 4, with the number dependent on the emergency calls they receive. Live fire exposures have been noted to increase core temp to 37.9 - 40.1°C (Chapter 5, Smith et al. 1996; Eglin & Tipton 2005), therefore individuals could experience close to or above the 38.5°C recommended for cellular adaptations and isothermic controlled T_c for acclimation. Long term heat acclimatisation has previously been noted in seasonal wildland FF, with 4 months of occupational heat exposure (61% of the time working at a fire) reducing T_c and physiological strain and improving rating of perceived exertion (Lui et al. 2014). It is postulated that the frequency of exposures, and elevation in T_c experienced, may lead to FSI developing an acclimatised state.

Acclimation status typically decays in 2 - 4 weeks, although physical activity can potentially help to maintain adaptations for longer (Garrett et al. 2009; Périard et al. 2015). A recent review states that after ≥ 5 days of heat acclimation every day without heat exposure results in ~2.5% reduction in T_c and HR adaptations (Daanen et al. 2018). It has been suggested that to sustain an acclimated state, one day of heat exposure is needed for every 5 days spent without it (Pandolf 1998; Taylor 2000). Re-acclimating following ≥ 2 weeks without heat exposure can also occur quicker than original heat acclimation, being achievable in 2 - 4 days instead of 5 - 10 days (Ashley et al. 2015; Daanen et al. 2018). In addition, longer heat re-acclimation (10 days) can cause adaptations greater than that noted with initial heat acclimation (Saat et al. 2005; Daanen et al. 2018). These re-acclimation responses may be a consequence of cellular memory for the acclimated state (Tetievsky et al. 2008). The amount of time between heat exposures varies across UK Fire and Rescue Services for FSI, however the median number of exposures completed a week by FSI is 3 (Chapter 4), which could result in 4 days a week without exposure. FSI therefore may develop an acclimatised state and be able to maintain it based on their occupational exposures. There has not previously been any assessment of FSI heat tolerance, as the Fire and Rescue Service do not currently use any form of heat tolerance testing (Optimal Performance Limited, 2004).

Chapter 7 introduced a new tolerance test, the HOTT, which has strong ecological validity as it is conducted whilst wearing PPE for a similar duration to a wear, whilst enabling inter and intra individual comparisons to be conducted due to its use of a set $\dot{H}_{prod}$. Furthermore, Chapter 7 details that the HOTT is both valid compared to the commonly used Israeli Defence for HTT, and reliable, with strong ICC and acceptable CV and mean bias for key measures of acclimation such as T_{re}, HR, and SR. Consequently, the HOTT should be used to identify the tolerance levels of FSI.

The immunological consequences of heat acclimation also need to be further understood. Recent research indicates that heat acclimation has no impact on immune function, with no change in WBC following a 4 day protocol (Willmott et al. 2016), no change in IL-6 or CRP with an 11 day protocol (Costello et al. 2018), and no change in IL-6 following 7 acclimation sessions over 18 days (Guy et al. 2016). Alternatively, a small study of FSI has previously noted altered baseline levels of numerous immunological markers compared to non-wearing individuals (Watt et al. 2016). Wright-Beatty et al. (2014) also report that FF

have a smaller increase in IL-6 following exercise in the heat (+0.67 ± 0.17 pg.mL^{-1}), than non-firefighting individuals (+1.10 ± 0.18 pg.mL^{-1}). The authors postulated that this may be due to adaptation to stressful situations or reduction of perceived exertion during the cycling exercise. However, no difference occurred in IL-6 responses between long serving FF (25 ± 2 years of service) and newer FF (3 ± 0 years of service). It is unknown what impact long term acclimation may have on an individual's immune function, with no heat acclimation study longer than 11 exposure days having collected immunological markers. Repeated heat exposure, with minimal recovery, have been postulated to compromise individuals' immune systems and therefore be related to the occurrence of minor illnesses (Walsh et al. 2011). Although the supporting evidence for this concept remains minimal (Walsh & Oliver 2016). Consequently, it is of interest to investigate if heat acclimatisation gained through FSI occupational exposures could be related to symptoms of ill health as described in Chapter 4.

The risk of FF experiencing a cardiovascular event also increases 12 to 136 times following a live fire exposure (Kales et al. 2007). Elevated cardiac troponin, a marker of myocardial ischemia, increased platelet activity and thrombus formation have also been noted in FF (Hunter et al. 2017). FSI also exhibit increased IL-6 (+28%), platelet counts (+26%), mean platelet volume (+8%), and cTnT (36%), following training exercises, indicating that these scenarios may increase the risk of cardiovascular events and cause minor myocardial damage (Chapter 5). However, the persistent impact repeated fire exposure has on cardiac damage has yet to be investigated.

Overall, this study aims to use the HOTT to identify if FSI develop a greater level of heat tolerance than non-wearing individuals and if so does chronic heat acclimatisation impact immune function and myocardial damage. The study also aims to establish if levels of heat tolerance are maintained by FSI over a 2 month period of their normal occupational schedule. Consequently, the study has 3 hypotheses: (1) FSI will have an increased tolerance to the HOTT, as evident by a reduced T_{re} compared to the CON group; (2) FSI will have a decreased inflammatory response to the HOTT, identified by decreased inflammatory markers such as WBC, NEUT, and IL-6 compared to the CON; and (3) FSI and CON will maintain their physiological, perceptual, and inflammatory responses following a 2 month period of normal scheduled wearing.

8.3 METHODS

8.3.1 Participants

In total 22 participants were recruited for this study. Eleven FSI (see Table 8.2) were recruited from three fire training centres, nine participants were male (body mass: 81.7 ± 8.6 kg, height: 177.4 ± 3.2 cm, age: 42 ± 8 yrs) and two participants were female (body mass: 58.3 ± 2.4 kg; height: 159.0 ± 5.7cm, age: 36 ± 3 yrs). FSI had been in their current role for 5 ± 5 yrs. Eleven non-wearing individuals were recruited as a control group (CON) (Table 8.2), with nine male participants (body mass: 80.4 ± 7.8 kg, height: 179.5 ± 5.3 cm, age: 40 ± 7 yrs) and two female participants (body mass: 55.8 ± 3.8 kg, height: 165.5 ± 10.6 cm, age: 45 ± 8 yrs). CON participants were selected to match FSI age, sex, and body composition. CON had not been involved in heat acclimation training or had > 3 consecutive days of heat exposure > 25°C in the

previous month (Périard et al. 2015). Participants gave informed written consent and completed a medical questionnaire prior to taking part (section 3.1). Participants were required to consume a similar diet and abide by dietary and exercise restrictions as described in section 3.6.

8.3.2 Experimental Design

Participants were required to complete two experimental trials separated by a 2 month period, to capture the maintenance of any heat adaptations rather than the immediate adaptation that may be present following 2 weeks of wearing, or a decay in tolerance due to days away from exposure after a training course. Each trial consisted of completion of the International Physical Activity Questionnaire (IPAQ), assessment of body composition, blood sample collection, and a HOTT. Both sessions were completed at the same time of day, with sessions commencing between 8:00am and 10:00am, to control for circadian rhythms. Participants were required to attend the laboratories in a euhydrated state (section 3.6.1).

8.3.3 International Physical Activity Questionnaire

The long form IPAQ was completed at the beginning of each trial. The questionnaire consisted of 27 questions on the type and duration of physical activity that the participant completed in a typical week in the previous 2 months (see Appendix 2). The questionnaire categorised the type of physical activity completed as walking, moderate or vigorous. Anchors were given to participants for moderate "a light jog can hold a conversation" and vigorous "working hard, increased breathing rate, can't hold a conversation" activity. The domain of activity was also reported as either part of their occupation, transport, chores, or leisure time. The IPAQ has been documented to be a valid and reliable way to assess an individual's physical activity level, with correlations between the IPAQ and activity log books, activity monitors, and aerobic fitness noted (Craig et al. 2003; Hagströmer et al. 2006). Data was collated in accordance with guidelines by Craig et al. (2003), resulting in a METmin.week^{-1} calculated to classify individuals as having low, moderate, or high physical activity level.

8.3.4 Body Composition

Body composition was assessed using an air displacement plethysmograph, as per section 3.7.3. Measurements commenced post hydration assessment and prior to the HOTT.

8.3.5 HOTT

The HOTT included a 10 min rest period (22.9 ± 1.2ºC, 31.2 ± 6.8% RH) whilst wearing fire protective clothing (boiler suit, trousers [Ballyclare Special Products Ltd.], jacket [Ballyclare Special Products Ltd.], boots [9005 GA, Jolly Scarpe, USA], fire hood [MSA Gallet, Bellshill, UK], helmet [F1SF, MSA Gallet, Bellshill, UK] and gloves [Firemaster 3, Southcombe Brothers Ltd, Somerset, UK]). Participants then entered the heat chamber (50 ± 1.0ºC, 12.3 ± 3.3% RH) and performed the HOTT as described in section 7.3.3.

8.3.6 Measures

HR, T_{re}, and T_{skin} were recorded at the end of the rest period and every 5 min throughout the HOTT (section 3.8 and 3.8.4). T_{skin} was recorded using wired skin thermistors. Nude body mass was recorded pre and post heat exposure for calculation of SR (section 3.8.6). TS and RPE were collected after resting and then every 10 min and HISI recorded pre and post heat exposure (section 3.10).

8.3.7 Venous Blood Collection

Prior to and immediately post the HOTT venous blood samples (10 mL) were collected from the anti-cubital fossa, as described in section 3.9. Samples were analysed for complete blood counts using the Sysmex (section 3.9.1) and then centrifuged and plasma stored for later ELISA analysis (section 3.9.2). ELISA's for IL-6, TNF-a, IL1-β, CRP and IgG were conducted, see Table 8.1 for CV%. Plasma samples were also sent to the Frontier Pathology laboratory, Royal Sussex County Hospital, Brighton for analysis of high sensitivity cTnT.

Table 8.1 *The inter and intra assay coefficient of variation (CV%) for each haematological variable analysed by ELISA.*

Haematological Variable	Inter Assay CV (%)	Intra Assay CV (%)
IL-6 ($pg.mL^{-1}$)	6.9	3.9
TNFα ($pg.mL^{-1}$)	7.3	3.2
IL-1β ($pg.mL^{-1}$)	14.9	4.4
CRP ($mg.L^{-1}$)	7.1	1.5
IgG ($mg.dL^{-1}$)	8.9	5.4

IL-6 = interleukin-6, TNFα = tumour necrosis factor alpha, IL-1β = interleukin-1 beta, CRP = C reactive protein, IgG = immunoglobulin G

8.3.8 Statistical Analysis

Data were analysed using IBM SPSS Statistics 22 (section 3.11). Haematological markers were analysed for baseline differences in Trial 1, due to previously reported differences at baseline in FSI compared to a control group (Watt et al. 2016). Independent samples t-tests were conducted to identify baseline differences (FSI vs. CON). A Mann Whitney U test was conducted for baseline comparisons (FSI vs. CON) when data violated normality assumptions.

Normally distributed data were analysed by a two way mixed method ANOVA (section 3.11.7.3) to identify differences between the time points of the HOTT and group (FSI vs. CON). A two way repeated measures ANOVA was then conducted to establish if responses changed between the trials (pre 2 months vs. post 2 months) across the time points of the HOTT. Bonferroni follow up tests were conducted where significant differences were identified. Data that violated normality assumptions were analysed by a Wilcoxon signed rank test to establish differences between pre and post trial 1 HOTT. Mann Whitney U tests were conducted to identify differences in absolute change pre 2 months between groups (FSI vs CON). A Wilcoxon signed

rank test was then conducted to establish if absolute change altered between trials (pre 2 months vs post 2 months). Significance was set at $p < 0.05$ (section 3.11.6). Effect sizes are presented as described in section 3.11.7.4. Correlations were conducted for all participants' between T_{re} at 40 min and possible related factors such as: BF%, exposure number, age (section 3.11.7.1). Pearson and Spearman correlation analysis was also conducted between haematological variables and 40 min $T_{re.}$ Point biserial correlations were conducted between FSI reported presence of ill health and 40 min T_{re} and haematological variables (section 3.11.8).

8.4 RESULTS

8.4.1 Trial 1 Baseline Characteristics

There was no difference in U_{col} ($p = 0.196$), U_{osm} ($p = 0.081$) or U_{spg} ($p = 0.069$) between FSI and CON in their initial trial. All participants met the euhydration criteria. All participants were also classified as having a high level of physical activity based on their IPAQ scores. However, FSI reported a greater amount of METmins.week^{-1} than the CON ($p = 0.019$) (Table 8.2). On comparison of the activity conducted, FSI completed a greater number of METmins.week^{-1} from their work than CON (5699 ± 3570 METmins.week^{-1} vs 792 ± 1862 METmins.week^{-1}) ($p = 0.001$). This accounted for 60 ± 20% of FSI METmin.week^{-1} in comparison to only 4 ± 5% of CON METmin.week^{-1}. Leisure activities accounted for 23 ± 16% of FSI METmin.week^{-1} compared to 58 ± 12% of CON METmin.week^{-1}. There was no difference in BF% ($p = 0.836$), BSA ($p = 0.934$), or BSA/mass ($p = 0.447$) between groups (Table 8.2).

At the initial trial symptoms of ill health were reported by 6/11 FSI participants and 1/11 CON participants. All 6 FSI reported cold symptoms, including blocked noses and coughs, 2 of the 6 participants reported night sweats, and 1 participant reported mood swings. The participant from the CON group reported frequent coughing, which was being investigated as possible adult onset asthma by a medical professional at the time of the study.

8.4.2 Trial 1 Heat Tolerance

Table 8.3 displays the resting and end of HOTT values for all physiological and perceptual variables. Two way ANOVA analysis revealed that there was a difference in T_{re} between the FSI and CON across the HOTT test ($p = 0.013$, $\eta_p^2 = 0.216$). Follow up Bonferroni corrected independent samples t-tests revealed no difference in T_{re} at rest ($p = 0.463$) or 20 min ($p = 0.361$), but a difference was present at 40 min ($p = 0.013$, $d_s = 1.16$) with T_{re} being greater in the CON group (38.56 ± 0.34°C) compared to the FSI group (38.14 ± 0.38°C). ΔT_{re} demonstrated a similar pattern, being different across the time points between FSI and CON ($p = 0.010$, $\eta_p^2 = 0.231$), with ΔT_{re} exhibiting no difference at 20 min ($p = 0.727$) but CON having a greater ΔT_{re} at 40 min (1.48 ± 0.34°C) than FSI (1.15 ± 0.28°C) ($p = 0.021$, $d_s = 1.06$). See Figure 8.1 for T_{re} at 40 min for all participants along a continuum, as described in Chapter 7. Based on interpretation of the continuum, FSI have an increased heat tolerance compared to CON, with only FSI sitting in the green zone, both CON and FSI in the yellow zone, and only one FSI in the red zone with six CON participants. When categorised as heat tolerant or intolerant based on the criteria determined in Chapter 7 one FSI was

classified as heat intolerant, whilst six participants from the CON group were heat intolerant.

There was no difference in SR between FSI and CON (p = 0.138). However, when female participants were removed from the data set, a greater SR was exhibited by FSI (1.35 ± 0.19 L.hr^{-1}) compared to CON (1.10 ± 0.18 L.hr^{-1}) (p = 0.015, d_s = 1.40). HR increased throughout the HOTT (p < 0.001, η_p^2 = 0.960) but was not different at any time point between the groups (p = 0.580, η_p^2 = 0.031). At 40 min HR was 166 ± 15 b.min^{-1} for FSI and 158 ± 14 b.min^{-1} for CON. ΔHR also displayed increases over time (p < 0.001, η_p^2 = 0.951) that were not affected by group (p = 0.632, η_p^2 = 0.037). PSI and T_{skin} also both did not differ at any time between the groups (p = 0.411, η_p^2 = 0.043 and p = 0.228, η_p^2 = 0.062, respectively).

Table 8.2 *Demographic details for FSI and CON at their initial trial and 2 months later at trial 2. * denotes significant difference at Trial 1 between FSI and CON (p<0.05).*

	Trial 1		**Trial 2**	
	FSI	CON	FSI	CON
Age (yrs)	41 ± 7	41 ± 7		
Height (cm)	174.1 ± 8.2	177.0 ± 8.1		
Body mass (kg)	77. 4 ± 12.2	75.9 ± 12.2	77.8 ± 12.8	75.0 ± 14.1
BF (%)	21.8 ± 5.9	21.3 ± 5.1	21.6 ± 6.3	20.2 ± 5.9
BSA (m^2)	1.92 ± 0.18	1.93 ± 0.19	1.92 ± 0.18	1.91 ± 0.20
BSA/mass ($cm^2.kg^{-1}$)	250.3 ± 17.3	256.4 ± 19.5	259.0 ± 23.5	249.7 ± 17.9
IPAQ (METmin.week^{-1})	9476 ± 5000*	5114 ± 2674	9377 ± 5168	4728 ± 1569
2 Week Fire Exposures	5 ± 3*	0 ± 0	5 ± 4	0 ± 0

FSI = fire service instructor, CON = control group, BF = body fat, BSA = body surface area, IPAQ = international physical activity questionnaire

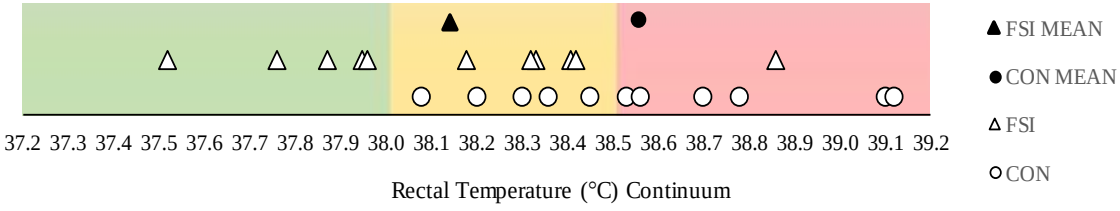

Figure 8.1 *A continuum of rectal temperatures (°C) exhibited at the end of the HOTT. The colour of the continuum used as suggested in Chapter 4. FSI = fire service instructor, CON = control group*

8.4.2.1 Perceptual Responses

RPE increased throughout the test ($p < 0.001$, $\eta_p{}^2 = 0.893$), but this increase was not different between groups ($p = 0.059$, $\eta_p^2 = 0.120$). RPE at 40 min was 15 ± 2 for FSI and 14 ± 2 for CON. HISI also increased following the HOTT ($p < 0.001$, $\eta_p^2 = 0.711$), but this increase was not effected by group ($p = 0.931$, $\eta_p^2 = 0.000$).

However, the increase in TS that occurred throughout the HOTT ($p < 0.001$, $\eta_p^2 = 0.906$) was different between the groups ($p = 0.048$, $\eta_p^2 = 0.111$). Follow up tests revealed no difference at rest ($p = 0.233$) or at 20 min ($p = 0.301$). However, at 40 min CON reported a greater TS (7.5 ± 0.5) than FSI (6.5 ± 0.5) ($p = 0.010$, $d_s = 2.00$).

8.4.2.2 Haematological Responses

Table 8.4 displays all haematological variables pre and post the HOTT for both trials. At baseline of Trial 1 FSI had greater levels of IL-6 ($p < 0.001$), IL-1β ($p < 0.001$), and IgG ($p = 0.001$) compared to CON. No other haematological variables differed at baseline ($p > 0.05$).

Both WBC and NEUT increased following the HOTT ($p < 0.001$, $\eta_p^2 = 0.666$ and $p < 0.001$, $\eta_p^2 = 0.549$, respectively) with the increase being different across groups ($p = 0.034$, $\eta_p^2 = 0.205$ and $p = 0.033$, $\eta_p^2 = 0.208$). However follow up tests did not identify any significant differences at pre ($p = 0.782$, $d_s = 0.12$ and $p = 0.940$, $d_s = 0.03$, respectively) or post ($p = 0.065$, $d_s = 0.83$ and $p = 0.073$, $d_s = 0.80$, respectively) time points.

PLT and LYMPH increased following the HOTT ($p < 0.001$, $\eta_p^2 = 0.620$ and $p < 0.001$, $\eta_p^2 = 0.641$, respectively) but the increase was not different between FSI and CON ($p = 0.371$, $\eta_p^2 = 0.040$ and $p = 0.663$, $\eta_p^2 = 0.010$, respectively). cTnT also increased following the HOTT ($p < 0.001$, $\eta_p^2 = 0.718$), although the pattern of increase was similar across the groups ($p = 0.952$, $\eta_p^2 = 0.000$). Alternatively, MPV decreased following the HOTT ($p = 0.002$, $\eta_p^2 = 0.380$), although this change was also not different between groups ($p = 0.583$, $\eta_p^2 = 0.015$). MONO, EO and BASO were unaltered by the HOTT ($p > 0.05$) regardless of group ($p > 0.05$).

Non-parametric statistical analysis revealed IL-1β and CRP decreased following the HOTT ($p = 0.013$, $p = 0.010$). However, TNFα and IgG were not changed post exposure ($p = 0.783$, $p = 0.592$). IL-6 increased following the HOTT ($p < 0.001$). There was no difference in absolute change between FSI and CON for CRP, TNFα, IgG and IL-6 ($p > 0.05$). Statistical analysis of differences between FSI and CON for change in IL-1β was not conducted as IL-1β was not detectable in 7 of 11 participants in the CON group.

Table 8.3 *Physiological and perceptual variables collected at rest and at the end of the 40 min HOTT during Trial 1 and 2 months later in Trial 2. * denotes a significant difference between FSI and CON in Trial 1, $p < 0.05$. No significant differences were detected between Trials 1 and 2.*

	Trial 1				**Trial 2**			
	FSI		CON		FSI		CON	
	REST	40 min	REST	40 min	REST	40 min	REST	40 min
T_{re} (ºC)	36.99 ± 0.32	38.14 ± 0.38 *	37.08 ± 0.23	38.56 ± 0.34	37.01 ± 0.22	38.23 ± 0.37	37.07 ± 0.23	38.56 ± 0.32
HR ($b.min^{-1}$)	67 ± 11	166 ± 14	61 ± 9	158 ± 13	67 ± 7	166 ± 12	63 ± 8	156 ± 16
PSI		6.71 ± 0.98		7.12 ± 0.96		6.83 ± 1.03		7.08 ± 0.83
T_{skin} (ºC)	31.90 ± 0.79	38.30 ± 0.54	32.17 ± 0.58	38.64 ± 0.38	32.17 ± 0.80	38.68 ± 0.40	32.25 ± 0.63	38.51 ± 0.41
SR ($L.hr^{-1}$)		1.22 ± 0.35		0.99 ± 0.29		1.20 ± 0.34		0.98 ± 0.27
RPE	6 ± 0	15 ± 2	6 ± 0	14 ± 2	6 ± 0	16 ± 2	6 ± 0	14 ± 3
TS	4.0 ± 0.5	6.5 ± 0.5 *	4.0 ± 0.5	7.5 ± 0.5	4.0 ± 0.5	6.5 ± 0.5	4.0 ± 0.5	7.0 ± 0.5
HISI	1 ± 1	27 ± 18	2 ± 2	26 ± 17	2 ± 2	25 ± 12	1 ± 1	30 ± 20

FSI = fire service instructor, CON = control group, T_{re} = rectal temperature, HR = heart rate, PSI = physiological strain index, T_{skin}= skin temperature, SR = sweat rate, RPE = rating of perceived exertion, TS = thermal sensation, HISI = heat illness symptoms index

Table 8.4 *Haematological variables at rest and post the HOTT for Trial 1 and Trail 2.* [$] *denotes a significant difference between FSI and CON at rest in Trial 1 and* [#] *denotes a significant difference between rest and 40 min in Trial 1 irrespective of group, $p < 0.05$. No significant differences were detected between Trials 1 and 2 ($p > 0.05$).*

	Trial 1				**Trial 2**			
	FSI		CON		FSI		CON	
	REST	40 min	REST	40 min	REST	40 min	REST	40 min
WBC ($x10^9.L^{-1}$)	5.49 ± 1.09	7.47 ± 1.76 [#]	5.38 ± 0.73	6.31 ± 0.88 [#]	5.41 ± 1.19	7.03 ± 1.52	5.67 ± 1.62	6.63 ± 1.65
PLT ($x10^9.L^{-1}$)	240 ± 50	271 ± 57 [#]	215 ± 39	256 ± 62 [#]	244 ± 51	285 ± 61	230 ± 36	268 ± 37
MPV (fL)	10.28 ± 0.90	9.80 ± 1.00 [#]	10.57 ± 0.75	9.91 ± 0.95 [#]	10.34 ± 1.02	10.04 ± 0.90	10.58 ± 0.78	9.76 ± 0.92
NEUT ($x10^9.L^{-1}$)	2.97 ± 0.67	4.39 ± 1.38 [#]	2.95 ± 0.72	3.47 ± 0.85 [#]	2.83 ± 0.74	3.70 ± 1.36	3.21 ± 1.15	3.77 ± 1.28
EOS ($x10^9.L^{-1}$)	0.18 ± 0.09	0.14 ± 0.08	0.17 ± 0.11	0.23 ± 0.34	0.17 ± 0.08	0.16 ± 0.08	0.15 ± 0.11	0.12 ± 0.09
BASO ($x10^9.L^{-1}$)	0.03 ± 0.01	0.03 ± 0.01	0.02 ± 0.01	0.03 ± 0.01	0.03 ± 0.02	0.03 ± 0.02	0.03 ± 0.01	0.03 ± 0.01
LYMPH ($x10^9.L^{-1}$)	1.83 ± 0.72	2.37 ± 0.94 [#]	1.66 ± 0.54	2.13 ± 0.51 [#]	1.86 ± 0.61	2.39 ± 0.60	1.63 ± 0.75	2.00 ± 0.77
MONO ($x10^9.L^{-1}$)	0.48 ± 0.14	0.51 ± 0.15	0.49 ± 0.12	0.49 ± 0.12	0.51 ± 0.14	0.56 ± 0.12	0.47 ± 0.24	0.48 ± 0.26
IL-6 ($pg.mL^{-1}$)	2.18 ± 2.39 [$]	2.82 ± 2.54 [#]	0.28 ± 0.37	0.98 ± 0.84 [#]	2.20 ± 2.50	3.28 ± 2.66	0.32 ± 0.56	0.78 ± 1.19
IL-1β ($pg.mL^{-1}$)	20.52 ± 18.19 [$]	18.40 ± 16.60 [#]	1.37 ± 2.06	1.25 ± 1.89 [#]	27.30 ± 29.03	25.91 ± 26.47	0.67 ± 1.20	0.60 ± 1.03
TNFα ($pg.mL^{-1}$)	9.16 ± 11.26	8.32 ± 10.85	2.16 ± 1.74	2.34 ± 1.64	10.73 ± 13.86	10.47 ± 12.78	1.78 ± 1.43	2.03 ± 1.39
CRP ($mg.L^{-1}$)	0.84 ± 0.55	0.79 ± 0.68 [#]	0.78 ± 0.47	0.66 ± 0.37 [#]	1.04 ± 1.00	0.98 ± 1.08	0.73 ± 0.58	0.66 ± 0.51
IgG ($mg.dL^{-1}$)	2453 ± 906 [$]	2450 ± 824	1286 ± 616	1185 ± 495	1897 ± 665	2292 ± 897	1271 ± 441	1429 ± 626
cTnT ($ng.L^{-1}$)	4.80 ± 1.07	5.70 ± 1.19 [#]	5.95 ± 1.85	6.84 ± 2.24 [#]	5.28 ± 1.75	6.03 ± 1.76	6.68 ± 2.79	7.83 ± 3.29

FSI = fire service instructor, CON = control group, WBC = white blood cell count, PLT = platelet count, MPV = mean platelet volume, NEUT = neutrophil count, EOS = eosinophil, BASO = basophil, LYMPH = lymphocyte, MONO = monocyte, IL-6 = interleukin-6, IL-1β = interleukin-1 beta, TNFα = tumour necrosis factor alpha, CRP = C reactive protein, IgG = immunoglobulin G, cTnT = cardiac troponin T

8.4.3 Maintenance of Heat Tolerance

Hydration status was the same for pre and post 2 month trials, with no differences in U_{col} (p = 1.00), U_{osm} (p = 0.918), and U_{spg} (p = 0.642) (Table 8.2). There was also no difference in IPAQ scores between the two trials (p = 0.774). BF% also remained the same between the trials (p = 0.179). The number of wears completed by FSI was also not different in the 2 weeks prior to trial 1 compared to trial 2 (p = 0.810), although when looking at individual data sets, two FSI had a difference of seven exposures between trials.

Similar to the initial visit, post 2 months 6/11 FSI participants and 1/11 CON participants presented with symptoms of ill health. Of the 6 FSI, 4 had reported symptoms at the initial visit. The CON participant was the same individual who presented at the initial trial and had been diagnosed with adult onset asthma in the 2 month period. All 6 FSI reported cold symptoms, including blocked noses and coughs, 3 of the 6 participants reported night sweats, 1 participant reported mood swings, and 1 participant reported extreme fatigue.

All physiological and perceptual variables exhibited the same response between trials, with no differences between trials at any time point (Table 8.3). All haematological variables were the same between trials and there were no differences between trials at any time point (Table 8.4).

8.4.4 Correlations

Analysis of all participants' data revealed T_{re} at 40 min was significantly correlated with the number of exposures completed in the previous 2 weeks (r_s = -0.589, p = 0.004), see Figure 8.2. Age, IPAQ score, SR, BSA, and BSA/mass were not correlated with 40 min T_{re} (p > 0.05). Of the haematological markers T_{re} at 40 min was only correlated with pre HOTT IL-1β (r_s = -0.448, p = 0.036).

Point biserial correlations revealed the presence of illness was negatively correlated with T_{re} at 40 min in the HOTT (r_{pb} = -0.473, p = 0.026) and positively correlated with the number of wears (r_{rb} = 0.543, p = 0.000). There were no significant correlations between presence of illness and any haematological variables.

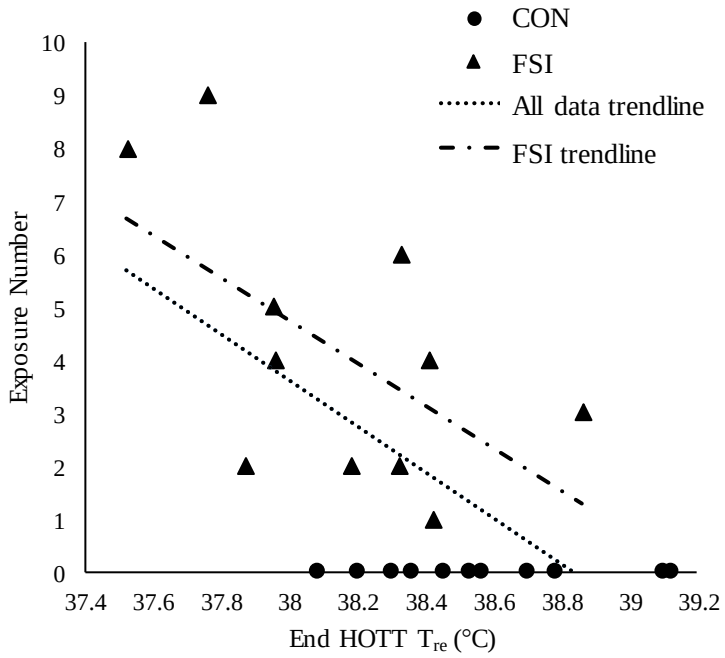

Figure 8.2 *End HOTT T_{re} plotted against exposure number. T_{re} = rectal temperature.*

8.5 DISCUSSION

This study aimed to identify if FSI develop a heat acclimatised state and if this state impacts their immune response to a heat exposure. The findings of this study demonstrate that the level of exposures experienced by FSI may be adequate to result in an acclimatised state. This is evidenced by reduced T_{re} and TS, and increased SR at the end of the HOTT in comparison to the CON group. Consequently, alternate hypothesis (1) can be accepted. This acclimatised state indicates that FSI are more thermotolerant than non-wearing individuals. In addition, acclimatisation of FSI may be linked to the number of wears completed. However, there was no difference in FSI and CON immunological responses to a heat exposure, suggesting that alternate hypothesis (2) can be rejected. Alternatively, FSI do exhibit greater baseline levels of IL-1β, IL-6, and IgG. In addition, the study aimed to assess if FSI maintained any adaptations over a normal working schedule. Physiological, perceptual, and haematological responses were the same following a 2 month working period, indicating that FSI maintain their heat acclimatised status, even with small fluctuations of exposure numbers, consequently alternate hypothesis (3) can be accepted.

8.5.1 Acclimation Status

A meta-analysis of 96 heat acclimation studies show T_c changes of 0.18 ± 0.14ºC at rest and 0.34 ± 0.24ºC at comparable time points following heat acclimation (Tyler et al. 2016). Although no differences in resting T_{re} were noted in this study, ΔT_{re} at the end of the HOTT was 0.33ºC lower in the FSI group. This is a smaller reduction than that noted following 10 days of intermittent heat acclimation (0.6 ± 0.7°C) or with 10 consecutive days of acclimation (1.0 ± 0.1°C) (Gill & Sleivert 2001). However, the decrease noted in this study is greater than that previously reported in acclimatised wildland FF (0.2°C) following 4 months

of seasonal deployment (Lui et al. 2014). Although the lower T_{re} noted in FSI sits within the range reported following heat acclimation protocols, the variation in actual reductions is likely the consequence of the duration, frequency, type, and intensity of the protocol that was implemented (Sawka et al. 2011). Acclimation has also been noted to reduce resting HR by 6 ± 5 $b.min^{-1}$ and by 16 ± 6 $b.min^{-1}$ during exercise (Tyler et al. 2016). However no differences in HR were noted between groups in this study. It is possible that this is a consequence of the between subjects design of this investigation and large HR standard deviations, which were close to or exceeded the reported changes in HR following acclimation, preventing the detection of small differences.

On initial analysis there was no difference in SR between groups, which is likely a consequence of the large standard deviation caused by the inclusion of the female data sets, as a result of females' lower sudomotor thermosensitivity (Gagnon et al. 2013), However, statistical analysis of just male SR revealed that FSI had a 0.25 $L.hr^{-1}$ greater SR. This is similar to the 0.20 - 0.36 $L.hr^{-1}$ reported following both short and long term heat acclimation (Patterson et al. 2004; Weller et al. 2007; Gibson et al. 2015; Willmott et al. 2016). An increase in SR is likely caused by a decrease in the sweating threshold temperature (Yamazaki & Hamasaki 2003) and also due to local sweat gland adaptations such as increased cholinergic sensitivity of the eccrine sweat gland or increased glandular hypertrophy (Lorenzo & Minson 2010; Buono et al. 2009). Whilst a greater SR is indicative of an acclimated status, it may be of little benefit in an uncompensable environment where an individual's evaporative capacity cannot increase. For FSI the PPE minimises their ability to evaporate sweat due to the impermeable nature of the clothing, consequently an increase SR is unlikely to assist with heat dissipation.

Participants in the FSI group also exhibited a 1.0 improvement in thermal sensation (from between "very hot" and "unbearably hot" to between "hot" and "very hot"), this is similar to the 0.8 reduction in thermal sensation reported by Tyler et al (2016) following a meta-analysis of heat acclimation studies**.** The reduced thermal sensations of FSI are also closer to that reported following long term 10 day heat acclimation (0.5) than has been noted to occur following short term 5 day acclimation (0.2) (Gibson et al. 2015). Thermal sensation may be an instigator of behavioural thermoregulation, with a reduced sensation of warmth resulting in the attenuation of the decreasing work rate that can occur when exercising in uncompensable environments (Flouris & Schlader 2015).

However, no differences in T_{skin} were detected between groups. T_{skin} reductions as a result of heat acclimation are a consequence of improved heat loss mechanisms, specifically an 11% increase in evaporative heat loss (Poirier et al. 2015). This increase has been noted to lead to a 26% reduction in body heat content (Poirier et al. 2015). However, evaporative heat loss cannot improve when wearing encapsulating PPE and may explain the similar T_{skin} responses recorded.

Overall, T_{re}, SR, and TS responses suggest that FSI exhibit an acclimatised profile in comparison to non-wearing individuals. However, the heat adaptations noted in FSI may not be as pronounced as those completing standard heat acclimation in a laboratory. This may be because similar bouts of heat exposure are conducted over time, consequently reducing the magnitude of the physiological strain in subsequent

exposures (Périard et al. 2016). To optimise adaptations, repeated exposures of the same level of thermal impulse, a controlled hyperthermia model, are required (Tyler et al. 2016).

The single FSI who was classified as heat intolerant had the highest BF% (32%), classifying the individual as overweight (> 25%) (Gallagher et al. 2000; De Lorenzo et al. 2003). To improve heat tolerance without additional heat exposures FSI should ensure they have a healthy body composition. End T_{re} was negatively correlated with the number of fire exposures in the previous 2 weeks, suggesting those with a greater number of exposures demonstrated a more acclimatised profile. It is possible that aerobic fitness is also correlated to heat tolerance (Selkirk & Mclellan 2001) and although IPAQ classifications were similar between groups, FSI completed a greater amount of physical activity, as evidenced by the increased $METmin^{-1}.week^{-1}$. However the matching of body composition across groups and the implementation of a test controlled by H_{prod} may have attenuated this effect (Cramer & Jay 2014). Measurement of $\dot{V}O_2$ max was not possible in this study due to time constraints; all participants had met the UK Fire Service $\dot{V}O_2$ max minimum standard of 42 $mL.kg.^{-1}min^{-1}$ at their occupational fitness test (Siddall et al. 2016), although it is likely this was indirectly measured via protocols such as the Chester Step test.

All participants maintained their level of heat tolerance over the 2 month period, as demonstrated by the similar responses in all physiological, perceptual, and haematological variables. This maintenance occurred even with a range of exposure numbers between the trial sessions, with two FSI having only 1 heat exposure in the 2 weeks prior to the second HOTT test. However, extended periods away from wearing, such as when on annual leave, could reduce a FSI heat tolerance. FSI may re-acclimatise quicker than is suggested with standard acclimation protocols, as their body may retain a molecular memory of acclimation state via heat shock protein genes (Tetievsky et al. 2008; Horowitz 2014; Daanen et al. 2018). Numerous genes associated with cytoprotection and thermotolerance, including genes coding for heat shock proteins, have been reported to remain activated, despite the physiological phenotype returning to a pre acclimatised state (Tetievsky et al. 2008). Moreover, molecular memory may also result in a greater acclimatisation response, with previous research suggesting that reacclimatisation can result in an 11% additional reduction in T_{re} than achieved with initial acclimation (Saat et al. 2005; Daanen et al. 2018). Consequently, while experiencing up to 8 exposures a month would not be enough to acclimate an average individual, the long term career of experiencing repeated bouts of these exposures may explain the acclimatised state demonstrated in this study by FSI. Previous research suggests that after adaptation to the heat is obtained, up to a month without heat exposure can be experienced with only 2 - 4 days of exposure needed to re-acclimate (Weller et al. 2007). More recent findings suggest that after a 2 month period of decline in acclimation state, only 2 days of exposure are required to regain acclimation (Horowitz 2016). A review of heat acclimation decay and re-acclimation also suggests that 5 - 10 days are adequate to induce initial stable adaptations, after which only 4 - 5 days are needed to fully re-acclimate following a 2 week break (Daanen et al. 2018). In addition, a recent study indicates that following heat acclimation a heat exposure every five days can sustain adaptations and may reduce the risk of EHI for up to a month after acclimation (Pryor et al. 2018; Daanen et al. 2018). Overall, this indicates that once acclimatised FSI may need 2 - 4 exposures a month to maintain their level of thermotolerance.

8.5.2 Haematological Response

Whilst physiological and perceptual variables displayed differences between the FSI and CON group indicative of an acclimatised state in the FSI, the change in haematological variables following the HOTT was similar between groups. Increases in WBC (+27.8%), NEUT (+34.2%), LYMPH (+30.9%), and PLT (+15.8%) were present following the HOTT. This is in line with haematological responses reported following live fire exposures in Chapter 5 (WBC +24.7%, NEUT +51.1%, LYMPH +30.3%, and PLT +25.6%). In addition, this supports the findings of other research studies into fire exposures, who detail similar increases in these haematological markers (Smith et al. 2005; Walker et al. 2015; Watt et al. 2016). However, no significant change in MONO was detected (+3.8%), and a decrease in MPV (-5.3%) was noted, opposing the findings of Chapter 5 which reports MONO and MPV increased (+42.6% and +8.0%, respectively). These differences could highlight altered responses to laboratory and live fire exposures and consequently may warrant further investigation to identify the cause of these MPV and MONO variations. Cytokine levels responded in a similar way to that detailed in Chapter 5, with increased IL-6 (+55.0%) and decreased CRP (-10.9%). The greater increase in IL-6 detailed in this study compared to Chapter 5 (+27.6%) may reflect the higher T_{re} achieved in the HOTT than that reported in Chapter 5 and the greater intensity of physical activity performed, as participants walked continuously during the HOTT in comparison to more intermittent movement performed whilst conducting FSI tasks.

This study also noted a decrease in IL-1β following the HOTT (-10.2%). It is well established that hyperthermia is related to elevated IL1-β (Fehrenbach & Schneider 2006; Heled et al. 2013), however investigations into IL-1β following exercise and heat exposure reveal contrasting findings. A decrease in IL1-β from 1.01 ± 1.51 $pg.mL^{-1}$ to 0.64 ± 0.14 $pg.mL^{-1}$ has been noted following a 164 km road cycling event in 35ºC (Luk et al. 2016). In contrast, two fold increases in IL-1β have been reported post marathon performance (Ostrowski et al. 1999) and following 2 hours of running in 35ºC (Snipe et al. 2018), whilst others report no change following a marathon (Suzuki et al. 2000), an iron man triathlon (Suzuki et al. 2006), and a heat stress test (Lim et al. 2009). The variety of responses reported may be due to different exercise intensities, durations, modalities, and environmental conditions. It has previously been suggested that TNFα and IL-1β may not respond following exercise, with IL-6 primarily being elevated rather than the classic pro-inflammatory cytokines (Petersen & Pedersen 2005). This is echoed in the unaltered TNFα response noted in this study (-5.8% $p > 0.05$). Although, small increases in TNFα have been reported following moderate and high intensity exercise with substantial increases (≥ 2ºC) in T_{re} (Lim et al. 2009; Snipe et al. 2018).

The HOTT also resulted in increased circulating cTnT (+20%); as a marker of cardiac muscle damage this indicates that minor myocardial injury may have occurred (Thygesen et al. 2010; Hunter et al. 2017). However, cTnT elevation was greater following live fire exposure (+36%), as reported in Chapter 5, than from the HOTT. This indicates that although maximum physiological strain experienced during the HOTT was greater than that of FSI training exposures, the fire exposures may generate greater cardiac muscle damage. Environmental temperatures have previously been noted to elevate cTnI levels without exercise, with increased cTnI having been reported in 52% of non-exertional heat illness cases during a heat wave

(Hausfater et al. 2010). This could indicate that the strain caused by the thermal environment may be a greater threat to cardiac damage than moderate exercise itself, although further evidence would need to be collected to support this theory. In addition, smoke/particulate exposure has been linked to an increased risk of myocardial infarction and therefore may also explain the greater increase in cTnT with live fire exposure (Brook et al. 2010; Koton et al. 2013). However, cTnT in direct relation to FF smoke exposure has not yet been investigated. Whilst cTnT was increased follow the HOTT, the response was similar between the FSI and CON group, suggesting that although FSI experience repeated heat exposures, it does not confer either a protective or detrimental impact to cardiac muscle.

These results indicate that FSI haematological responses to heat exposure are not altered compared to non-wearing controls, despite FSI physiological adaptations. This is in line with some previous research that indicates 4 - 11 days of repeated heat exposure result in similar IL-6 (Kanikowska et al. 2012; Barberio et al. 2015; Guy et al. 2016; Costello et al. 2018), WBC (Kanikowska et al. 2012; Willmott et al. 2016), and CRP (Costello et al. 2018) responses from day 1 to final exposure, and between heat acclimation and control groups. This is despite findings by Hailes et al. (2011) who report a blunted IL-6 response to heat exposure on day 5 (+1 $pg.mL^{-1}$) vs day 1 (+2.2 $pg.mL^{-1}$) of an acclimation protocol. Hailes et al (2011) also document this blunted response in other cytokines that were not measured in this study, including IL-10, IL1ra and TNF-rII. However no adjustment for changes in plasma volumes were made. There are currently no studies that assess the impact of heat acclimation of longer than 11 days on inflammatory markers, to compare the findings of this study to. Therefore, this study offers the first assessment of the inflammatory response to heat exposures with chronic acclimatization.

8.5.2.1 Comparison to Healthy Ranges

Baseline levels of IL-6, IL1-β, and IgG were greater in FSI than the CON group. Resting levels of IgG in FSI (2453 ± 906 $mg.dL^{-1}$) were outside of the standard reference values (650 - 1690 $mg.dL^{-1}$) (Ritchie et al. 1998), and IL-1β were also greater (20.52 ± 18.19 $pg.mL^{-1}$) than that commonly reported in healthy resting individuals (0.14 - 1.00 $pg.mL^{-1}$) (Di Iorio et al. 2003; La Fratta et al. 2018). In addition IL-6 at rest (2.18 ± 2.39 $pg.mL^{-1}$) was in the upper quartiles of apparently healthy individuals (≥ 1.47 $pg.mL^{-1}$). Consecutive heat exposure has been suggested to lead to an increased resting systemic inflammation, demonstrated by increased levels of pro (IL-12p40) and anti (ILra, IL-10) inflammatory cytokines following 5 days of heat acclimations (Hailes et al. 2011). This is also supported by findings of Watt et al. (2016) who report elevated resting IL-6 in FSI following a 4 week instruction course (11.4 ± 1.0 $pg.mL^{-1}$). Combined stressors, such as sleep deprivation, high intensity physical exertion, and energy deficit, experienced with a 3 week military training and 5 day combat course, led to increased resting IL-6 (pre: 1.59 ± 0.21 vs post: 2.68 ± 0.46 $pg.mL^{-1}$) although no differences in IL-1β occurred (Gomez-Merino et al. 2005).

IL-1β is a key mediatory of the inflammatory response and is essential for host defence against pathogens. However, in situations of chronic disease and acute tissue injury it can exacerbate damage (Dinarello 2011), by increasing expression of adhesion molecules in endothelial cells and promoting diapedseis and the acute

phase response (Barksby et al. 2007). Repeated exercise, designed to cause an overtraining response over 8 - 11 weeks by overloading intensity and reducing recovery periods, has been noted to result in increased IL-1β, alongside elevated lipopolysaccharides (LPS) levels (Lira et al. 2010; Pereira et al. 2015). LPS is involved in the release of IL-1β, due to its stimulatory effect on caspase-1 which cleaves the IL-1β precursor protein into mature IL-1β (Schumann et al. 1998). However, maximal expression of IL-1β following LPS exposure has been noted to occur 6 hours post exposure, so may not have been captured by the blood sampling conducted in this study (Chensue et al. 1991; Luk et al. 2016). Although these overtraining studies were conducted in rats, they may provide the mechanistic basis for the chronically increased IL-1β eluded to in this study. Symptoms of overtraining have been reported to show distinct familiarity with symptoms of "sickness behaviour", of which IL-1β plays a key role in instigating (Smith 2000; Konsman et al. 2002). The transfer of IL-1β, via volume transmission, from circumventricular organs to brain targets such as the amygdaloid complex, stimulates this sickness behaviour, with feelings of weakness, malaise, fatigue, and inability to concentrate (Dantzer 2001).

In addition, IL-1β also plays a role in the atherothrombotic process, due to the stimulation of adhesion molecules and procoagulant activity, with atherosclerotic lesions having been found to contain IL-1β (Ridker et al. 2011). Consequently, frequent increases in gut permeability, caused by exercising in an uncompensable environment, may stimulate a chronic increase in IL-1β, indicating systemic inflammation is present in FSI. This could be linked to the occurrence of cardiovascular events, due to the association of IL-1β and atherosclerosis.

Elevated IL-6 levels are also related to the occurrence of atherosclerotic events (Rauchhaus et al. 2000; Ridker et al. 2000; Spoto et al. 2014). In a large prospective study conducted by Ridker et al. (2000), healthy individuals in the upper quartile of IL-6 levels (> 2.28 $pg.mL^{-1}$) had a relative risk of a future myocardial infarction 2.3 (95% CI 1.3 - 4.3) times higher than those in the lower quartile (< 1.04 $pg.mL^{-1}$), with a 38% increase in risk of each quartile increase. IL-6 induces increased platelet production and reactivity, alongside increased cell adhesion molecules, and consequently may be involved in the formation of atherosclerosis (Lindmark et al. 2001). Furthermore, like IL-1β, reports of overtraining have been linked to IL-6, with increased IL-6 positively related to increased sleep disturbances, increased fatigue, and increased mood depression, consistent with sickness behaviour symptoms (Main et al. 2010). Repeated experience of excessive physiological and/or psychological stress, with minimal recovery time between exposures, has been proposed to lead to an elevation in IL-6 and IL-1β levels, inducing symptoms of a "sickness" behaviour and ultimately resulting in symptoms associated with an overtrained status (Smith 2000). Although this cytokine hypothesis for overtraining currently lacks substantial supporting research, the elevated IL-6 and IL1-β exhibited by FSI, indicating a low level chronic inflammatory status, alongside the increased occurrence of symptoms of ill health, suggests that further investigation may be warranted in a larger sample of the FSI population.

Increased levels of IgG are also an indicator of chronic inflammation or acute and chronic infections (Dispenzieri et al. 2002). IgG has been noted to increase in parallel to increases in IL-6 (Gonzalez-Quintela et al. 2008), but also in response to pulmonary infections as it acts to neutralize and eliminate pathogens

(Twigg 2005; Van De Weert-Van Leeuwen et al. 2014). Resting levels of IgG in elite athletes following a 4 month period of training have previously been reported to remain within the normal range (898 ± 218 $mg.dL^{-1}$) (Córdova et al. 2010), this is similar to the healthy range detected in elite swimmers following a 12 week training programme (1040 $mg.dL^{-1}$, 95% CI 936 – 1112 $mg.dL^{-1}$) (Gleeson et al. 2000). However values greater than the normal range have been detected when a month of Judo training was combined with the additional stressor of Ramadan (1742 ± 384 $mg.dL^{-1}$) (Chaouachi et al. 2009). Although in comparison, the IgG levels reported in this study in FSI are markedly greater than the previous training study presents, consequently a larger sample of the population should be assessed to identify if this occurrence is universal to the population. Elevation of IgG could indicate the presence of infection and inflammation in FSI.

8.5.3 Illness

FSI with a lower end T_{re} at 40 min are more likely to have symptoms of ill health and elevated IL-1β, as indicated by the negative correlations. Those that complete a greater number of wears are also more likely to experience symptoms of ill health. An acclimatised status reduces the risk of a high T_{re} and heat exhaustion from occurring and may improve an individuals' ability to conduct work in a hot environment. However, in FSI it may also result in a greater risk of symptoms of ill health. This possible maladaptation to heat exposure may relate to the number of heat exposures FSI are completing. Whilst this may suggest that heat acclimatisation of FSI poses a risk to FSI health, 4 out of the 10 tolerant FSI involved in this study did not report symptoms of ill health. These individuals had an average of 3 ± 2 wears in the previous 2 weeks, compared to those who reported symptoms who had 5 ± 3 wears. This further supports the suggestion that a minimum of 2 - 4 wears a month could maintain acclimatisation. Moreover, the establishment of an appropriate wear limit could enable FSI to maintain an acclimatised status, whilst reducing the risk of symptoms of ill health occurring. A greater data set is needed to be able to establish this limit, however the findings of this study indicate that this may fall between 4 - 5 wears every 2 weeks and therefore 8 - 10 wears a month.

8.5.4 Limitations

Participants $\dot{V}O_2max$ data was not collected, due to the logistical need for only one laboratory visit. Consequently, associations between tolerance and cardiovascular fitness cannot be made. Additionally, no comparison between female and male individuals could be made, as FSI were recruited from within a suitable travelling distance of the laboratory and as a result of the small and widely distributed FSI population, the number of individuals who met the inclusion criteria were limited. This is also the reason for the small number of FSI involved in the study. Knowledge of the actual thermal load experienced during wear exposures was also not collected and could be useful in future studies to inform how acclimatisation develops and provide clearer details on the relationship between wear numbers and markers of inflammation and ill health.

8.6 CONCLUSION

The findings of this study indicate that FSI develop an acclimatised status, with a reduced T_{re}, an improved TS, and an increased SR in response to a heat exposure. This status is maintained over a 2 month period, despite fluctuations in number of exposures, suggesting that the occupational exposure level experienced by FSI is adequate to both develop and maintain a chronic acclimatised status. It is indicated that a minimum of 2 - 4 exposures a month may be adequate to maintain thermotolerance. FSI display a similar immunological response to heat exposures as non-wearing individuals, although they do present with heightened levels of haematological markers, namely IL-6, IL-1β, and IgG. This suggests FSI experience low level chronic inflammation, which has previously been linked to risk of cardiovascular events, sickness behaviour, and overtraining. FSI exhibiting a greater tolerance to heat exposure, with lower end $T_{re,}$ are more commonly experiencing symptoms of ill health, signifying that maladaptation to heat exposures may occur. Future research should establish an appropriate recommendation for a wear number limit, to enable heat acclimatisation to be developed and maintained, whilst reducing the risk of ill health.

9 STUDY 6: HAEMATOLOGICAL AND IMMUNOLOGICAL MEASURES IN FIRE SERVICE INSTRUCTORS AND FIREFIGHTERS

9.1 ABSTRACT

OBJECTIVES: FSI may be at risk of cardiovascular events and an overtraining like syndrome, due to frequently experiencing fire exposures. This study aimed to establish if FSI are at an increased risk of a cardiovascular event in comparison to FF, identify if FSI experience an overtraining like syndrome linked to the number of fire exposures they complete, and evaluate the current informal exposure limit of 9 exposures per month.

METHODS: Venous blood samples were collected from 110 Fire Service personnel (age: 44 ± 7 yrs; height: 178.1 ± 7.1 cm; body mass: 84.3 ± 12.0 kg), including 53 FSI and 57 FF. Work and exposure history details were collected from all participants. Samples were analysed for complete blood counts, IL-6, IL-1β, TNFα, CRP, IgG, and cTnT. Participants above healthy reference ranges were classified as "at risk".

RESULTS: Neutrophil/lymphocyte ratio (NLR), PLT, cTnT, IL-6, IL-1β, IgG and CRP were significantly greater in FSI than FF ($p < 0.05$). Multiple regression analysis revealed 18.8% of IL6, 24.9% of IL-1β, 29.2% of CRP, and 10.9% of IgG variance was explained by month exposure number. Those above the 9 per month exposure limit were 6 - 12 times more likely to be classified as "at risk" and were 16 times more likely to experience symptoms of ill health.

CONCLUSION: Predictors of cardiovascular risk are elevated in FSI. Increased cytokine levels related to symptoms of ill health suggest FSI experience an overtraining like syndrome. Following an exposure limit could reduce the prevalence of these high risk factors and ill health; the current 9 exposures a month limit is reasonable guidance. FSI need to be targeted for interventions to help reduce the occurrence of chronic systemic inflammation.

9.2 INTRODUCTION

The combined expression of heat acclimatisation with markers of chronic inflammation in FSI, as indicated in Chapter 8, suggests that FSI may become "maladapted" to repeated heat exposures as a consequence of their occupation. Furthermore, Chapter 8 alluded to an association between chronic inflammation and the occurrence of symptoms of ill health with the number of wears FSI complete.

The prevalence of new symptoms of ill health amongst FSI was established in Chapter 4, with 41% of FSI experiencing symptoms they did not have prior to their career. These included symptoms such as: fatigue, broken sleep, mood swings and headaches. These are similar symptoms to those noted in individuals who suffer from overtraining, whereby repeated exposure to intense training with minimal recovery time, leads to exhaustion, ill health, and decrements in performance (Kreider et al. 1998; Smith 2003). The mechanistic cause of overtraining is debated, however the cytokine hypothesis offers an explanation for many of the syndromes symptoms and is the most comprehensive theory (Smith 2000; Carfagno & Hendrix 2014). Smith et al. (2000) hypothesised that repeated intense, high volume exercise without sufficient rest may result in an accumulation of inflammatory cytokines, resulting in chronic systemic inflammation. It is not conclusive however whether cytokines are the cause of overtraining, as there are limited possibilities to study individuals who experience high levels of strain over long periods of time (Carfagno & Hendrix 2014) and consequently many studies are performed on overreached athletes or following high intensity training. Therefore overtraining itself is often not investigated (Halson & Jeukendrup 2004). However, the presence of elevated cytokines associated with symptoms of ill health noted in FSI in Chapter 8 offers tentative evidence to support the cytokine hypothesis and occurrence of an overtraining like syndrome in FSI.

Inflammation is also closely related to the presence of atherosclerosis and cardiovascular events. Numerous haematological markers such as IL-6, CRP, and PLT are involved in the inflammatory process and are also predictors of cardiovascular events (Ridker et al. 2000; Pearson et al. 2003; Sharma & Berger 2011). FSI, along with FF, have an increased risk of a cardiovascular event following a live fire (Kales et al. 2007), with a cardiac event also being the leading cause of death within FF (Fahy et al. 2015). Currently, research has focused on the immediate impact that live fire has to the risk of cardiac events (Fahs et al. 2011; Fernhall et al. 2012; Hunter et al. 2017), with the long term effect not having been investigated. Chapter 5 and findings of Watt et al (2016) highlight that FSI exhibit increases in PLT, IL-6, and cTnT after a training fire exposure, whilst Chapter 8 suggests that predictors of cardiovascular events may be elevated in FSI on a daily basis. It can be theorised that if a live fire causes an increased risk, with minimal recovery time between fires, this risk may be elevated further by subsequent exposures. It is therefore of interest to establish if the number of fire exposures experienced is linked to an elevation in haematological markers associated with cardiovascular risk on a daily basis.

The possibility of an overtraining like syndrome and increased risk of cardiovascular events in FSI may both be linked with repeated high stress exposures combined with minimal recovery time. Consequently, the frequency of wear completion by FSI may be instrumental in the occurrence of these conditions. From Chapter 4, it was highlighted that there is no evidenced based guidance on the number of wears a FSI should be completing. Although 55% of FSI did have a limit set by management, they varied across services from

2 - 10 per week. The most commonly reported limit was 9 per month, which is not a formal policy, rather one proposed by the Chief Fire Officers' Association (2015). Concerns regarding the number of wears performed was also a key theme when FSI offered their opinions about their working practices, with 35% of FSI indicating they completed too many wears (Chapter 4). Being able to establish a suitable wear limit that potentially enables FSI to maintain their heat tolerance, whilst minimises the risk of a overtraining symptoms occurring and limits cardiac event risk factors, would enable guidelines for FSI across the UK to be put in place. An evidence based wearing limit could potentially reduce the symptoms of ill health experienced by FSI, reduce the risk of cardiac events, and prolong time spent as a FSI for those that wished to continue.

Consequently, this study aims to establish if FSI exhibit an altered baseline immunological level compared to FF. In addition, it aims to identify if altered immunological levels are related to the number of wears completed by FSI. Finally, the study aims to evaluate the informally suggested limit of 9 wears a month, to establish if completing wear numbers above or below the limit is associated with immunological markers above upper reference ranges and reports of ill health. There are therefore 4 hypotheses within this study: (1) FSI will exhibit greater IL-6, CRP, cTnT and platelet volume and counts than FF, (2) increased fire exposure numbers will be related with elevated levels of cytokines, (3) there will be an association between those above the 9 wear limit and those exhibiting cytokine levels above the reference limits, and (4) there will be an association between immunological markers and reports of ill health.

9.3 METHODS

One hundred and ten individuals (age: 44 ± 7 yrs; height: 178.1 ± 7.1 cm; body mass: 84.3 ± 12.0 kg) were recruited from Fire and Rescue Services across the UK to participate in the study. Of the 110 recruits, 53 were FSI (age: 45 ± 8 yrs; height: 176.6 ± 8.1 cm; body mass: 84.1 ± 8.1 kg) and 57 were FF (age: 44 ± 7 yrs; height: 179.5 ± 5.8 cm; body mass: 84.4 ± 10.1 kg). Of the FSI, 47 (89%) were male and 6 (11%) were female. Of the FF, 55 (96%) were male and 2 (4%) were female. Participants gave informed written consent and completed a medical questionnaire prior to taking part (section 3.1).

9.3.1 Experimental Design

Participants were required to provide a single venous blood sample and provide working history details on one occasion. This was collected at the Welkin Laboratories, or on location at Fire Stations, and Fire Training Centres. Samples were collected from all FSI between 8:00 - 10:00am. Samples were collected from FF between 8:00 - 10:00am or from 5:00 - 7:00pm, depending on their availability due to work requirements and rotas. All samples were collected prior to any heat exposure that day.

9.3.2 Venous Blood Collection

A 10mL venous blood sample was taken from the anti-cubital fossa by a trained phlebotomist at rest (section 3.9). All samples were analysed within 2hr of collection for CBC using the Sysmex (section 3.9.1) and then centrifuged with plasma being stored for later ELISA analyses (section 3.9.2). Samples collected

from East Sussex and Kent Fire and Rescue Services were analysed at the Welkin Laboratories. Samples collected at the Fire Service College were analysed for CBC at Coventry University at the Lab+, to enable analysis to be completed within the optimal 2hr window. ELISA's for IL-6, TNF-a, IL1-β, CRP and IgG were conducted as per section 3.9.2. The inter and intra assay coefficients of variation (CV%) for each ELISA is detailed in Table 9.1. Plasma samples were also analysed at the Frontier Physiology external laboratory for high sensitivity cTnT using an electrochemiluminescence assay, which had a blank of 3 $ng.L^{-1}$ and CV at the upper reference limit (14 $ng.L^{-1}$) of < 8% (Westermann et al. 2017). Upper reference limits, or limits at which there is an increased risk of a cardiovascular event, were identified for all haematological variables, with limits for variables measured via ELISA analysis increased by the corresponding inter assay CV. Limits are detailed in Table 9.3.

Table 9.1 *The inter and intra assay coefficient of variation (CV%) for each haematological variable analysed by ELISA.*

Haematological Variable	Inter Assay CV (%)	Intra Assay CV (%)
IL-6 ($pg.mL^{-1}$)	5.5	5.3
TNFα ($pg.mL^{-1}$)	8.7	3.6
IL-1β ($pg.mL^{-1}$)	11.5	4.3
CRP ($mg.L^{-1}$)	11.7	2.7
IgG ($mg.dL^{-1}$)	6.0	5.0

IL-6 = interleukin-6, TNFα = tumour necrosis factor alpha, IL-1β = interleukin-1 beta, CRP = C reactive protein, IgG = immunoglobulin G

9.3.3 Participant Details and Working History

Participants were required to complete a short questionnaire detailing their caffeine and alcohol intake and exercise participation in the previous 24 hrs. They were also asked if in the previous month they had suffered from symptoms of ill health identified in Chapter 4 (fatigue, broken sleep, heavy sweating, heart palpitations, mood swings, coughing, and breathing problems) or any other illnesses. Participants were also asked to provide a brief work history. Details of their time in the Fire and Rescue Service, if they were currently a FSI, and the time they had spent in their current role were collected. Participants also reported how many fire exposures (in either a training or operational capacity) they had completed in the previous week and in the last month.

9.3.4 Statistical Analysis

Data were analysed using IBM SPSS Statistics 22 and presented as mean ± SD (section 3.11). Data were tested for normality and sphericity (3.11). Differences in haematological variables between FSI and FF were assessed via independent samples t-tests, or Mann-Whitney U tests when data were not normally distributed (sections 3.11.7.2 and 3.11.8). Effect sizes are presented as described in section 3.11.7.4. Cohens effect sizes were interpreted as recommended by Cohen (1988). Pearson's chi-squared analysis was conducted to establish if there was an association between occupational group (FSI vs FF) and occurrence

of symptoms of ill health (YES vs NO)), with subsequent odds ratios conducted where significant associations occurred (section 3.11.8).

Multiple regression analysis was conducted to identify the relationship between work history and demographic variables (BMI, age, time in service, number of wears completed per week, number of wears completed per month) with haematological dependent variables. Where dependent variables did not meet normality assumptions bootstrap re-sampling was conducted. Bootstrapping involves the simulation of new data sets (n = 1000) from the original data and statistical analysis of all simulated data. This produces robust *p* values and standard errors of regressions coefficients. Where significant regression models were identified, regression coefficient statistical significance was interrogated for each predictor variable. Regression analysis was then rerun with only significant predictor variables to define the model.

Haematological markers identified as predicted from exposure numbers had data classified as either above ("at risk") or below ("healthy") upper reference range values. To evaluate the effectiveness of the current FSI suggested limit of 9 wears a month, Pearson's chi squared analysis was conducted to identify associations between those performing ≤ 9 or > 9 exposures a month and those in the "at risk" or "healthy" groups (3.11.8) . Pearson's chi squared analysis was also conducted to determine if reference range groups ("at risk" vs "healthy") were associated with ill health symptoms (YES vs NO), with follow up odds ratios conducted for significant associations (section 3.11.8). Significance level was set at $p < 0.05$ (3.11.6).

9.4 RESULTS

9.4.1 Differences between FSI and FF

FSI and FF had similar demographic details (Table 9.2), with no differences present between age, body mass, BMI, and time in the fire service ($p > 0.05$). FSI had completed a greater number of fire exposures in the previous week ($p < 0.001$) and month ($p < 0.001$) in comparison to FF (Table 9.2). The maximum number of exposures completed in a week and month by FSI were 8 and 20, respectively. In total 37 FSI were within the suggested limit of ≤ 9 wears per month, whilst 16 were not. No FF completed > 3 exposures per month.

Table 9.2 *Demographic and work history details for firefighters and fire service instructors. * denotes a difference between the two groups, p < 0.05.*

	Fire Service Instructors (mean ± SD)	**Firefighters (mean ± SD)**
Age (yrs)	45 ± 8	44 ± 7
Body mass (kg)	84.1± 13.8	84.4 ± 10.1
BMI (kg.m^2)	26.9 ± 3.3	26.2 ± 2.9
Time in Service (yrs)	19 ± 8	17 ± 7
Time in role (yrs)	6 ± 5*	12 ± 7
Weekly Exposures	2 ± 2*	0 ± 0
Monthly Exposures	6 ± 5*	1 ± 1

Reports of ill health were made by 3 (5%) FF and 16 (30%) FSI. FF who reported illness all reported colds. Of the FSI who reported illness, 10 (19%) had fatigue, 8 (15%) had a cold, 8 (15%) had broken sleep, 4 (8%) suffered from a cough, 4 (8%) had heavy sweating, 2 (4%) had mood swings, 1 (2%) suffered from heart palpitations and 1 (2%) had sinusitis. Pearson's chi squared analysis revealed a significant association between group and presence of illness (χ^2 (1) =11.941, p = 0.001), with FSI 7.78 (95% CI 2.12 - 28.62) times more likely to have a symptom of ill health than FF.

Analysis of CBC between FSI and FF revealed increased levels of NEUT (p = 0.029), PLT (p < 0.001), BASO (p = 0.003), and NLR (p = 0.044) in FSI compared to FF. FF had increased levels of MPV (p = 0.001) and EO (p = 0.032) than FSI. Analysis of other haematological variables revealed increases in cTnT (p < 0.001), IL-6 (p = 0.002), IL-1β (p = 0.006), CRP (p = 0.005), and IgG (p < 0.001) in comparison to FF. See Table 9.3 for details of haematological levels.

Table 9.3 *Resting levels of haematological variables in FSI and FF. * denotes a difference between groups, p < 0.05.† denotes value is greater than upper reference value. Upper limit for WBC, RVC, HGB, HCT, NEUT, LYMPH, MONO, EO and BASO from Bain et al. (2011), PLT and MPV from Briggs et al. (2007), NLR from Forget et al. (2017), cTnT from Zhelev et al. (2015), IL-6 from Ridker et al. (2000), TNFα from Todd et al. (2013), IL-1β from Di Iorio et al. (2003) and La Fratta et al. (2018), CRP from Pearson et al. (2003) and IgG from Fuggle (2017).*

	Fire Service Instructors (mean ± SD)	**Firefighters (mean ± SD)**	**Effect Size (d_s)**	**Upper Reference Limit**
WBC ($x10^9.L^{-1}$)	6.75 ± 1.53	6.27 ± 1.13	0.34	10.0
PLT ($x10^9.L^{-1}$)	240 ± 66*	185 ± 39	1.03	400
MPV (fL)	10.0 ± 0.9	10.3 ± 0.7	0.37	11.2
NEUT ($x10^9.L^{-1}$)	3.92 ± 1.16*	3.40 ± 0.81	0.52	7.0
LYMPH ($x10^9.L^{-1}$)	1.97 ± 0.52	1.97 ± 0.52	0	3.0
MONO ($x10^9.L^{-1}$)	0.63 ± 0.21	0.62 ± 0.17	0.05	1.0
EO ($x10^9.L^{-1}$)	0.15 ± 0.10*	0.19 ± 0.11	0.38	0.5
BASO ($x10^9.L^{-1}$)	0.08 ± 0.15*	0.04 ± 0.02	0.38	0.1
NLR	2.12 ± 0.84*	1.83 ± 0.63	0.39	3.53
cTNT ($ng.L^{-1}$)	4.79 ± 1.77*	3.62 ± 0.87	0.85	14
IL-6 ($pg.mL^{-1}$)	2.98 ± 3.56*†	1.12 ± 1.01	0.73	2.41
TNFα ($pg.mL^{-1}$)	4.29 ± 3.71†	2.81 ± 1.74	0.52	3.59
IL-1β ($pg.mL^{-1}$)	11.56 ± 19.48*†	2.18 ± 3.02†	0.69	1.00
CRP ($mg.L^{-1}$)	1.98 ± 1.74*	0.95 ± 0.81	0.77	3.35
IgG ($mg.dL^{-1}$)	1688 ± 836*†	917 ± 485	1.14	1696

WBC = white blood cell count, PLT = platelet count, MPV = mean platelet volume, NEUT = neutrophil count, LYMPH = lymphocyte, MONO = monocyte, EOS = eosinophil, BASO = basophil, NLR = neutrophil lymphocyte ratio, cTnT = cardiac troponin T, IL-6 = interleukin-6, TNFα = tumour necrosis factor alpha, IL-1β = interleukin-1 beta, CRP = C reactive protein, IgG = immunoglobulin G.

9.4.2 Relationship between Deterministic Variables and Haematological Markers

Multiple regression analysis of haematological variables revealed significant models for IL-6, IL-1β, CRP and IgG only ($p < 0.05$). For these four haematological markers, age, time in service, and week exposure number were not predictor variables ($p > 0.05$). Following remodeling with only significant predictors, month exposure number explained 18.8% of IL-6 variance, 24.9% of IL-1β variance, 29.2% of CRP variance, and 10.9% of IgG variance. BMI also explained an additional 3% of CRP variance. Table 9.4 presents the summary statistics of the significant regression models.

Table 9.4 *The regression models for IL-6, IL1-β, CRP, and IgG, including the R^2 and p value for each model. The statistics for the predictor variables are also presented, including the observed β from the original data and the bias of β, standard error of β and p value from bootstrap analysis. * denotes $p < 0.05$.*

	R^2	Observed β	Bias	Standard Error	*p* value
IL-6	**0.188**				**< 0.001***
Monthly Exposure		0.245	-0.001	0.078	0.010*
IL1-β	**0.249**				**< 0.001***
Monthly Exposure		1.753	0.002	0.523	0.010*
CRP	**0.322**				**< 0.001***
Monthly Exposure		0.165	-0.001	0.037	0.002*
BMI		0.079	0.002	0.035	0.019*
IgG	**0.109**				**< 0.001***
Monthly Exposure		56.912	1.069	15.515	0.002*

IL-6 = interleukin-6, IL-1β = interleukin-1 beta, CRP = C reactive protein, IgG = immunoglobulin G

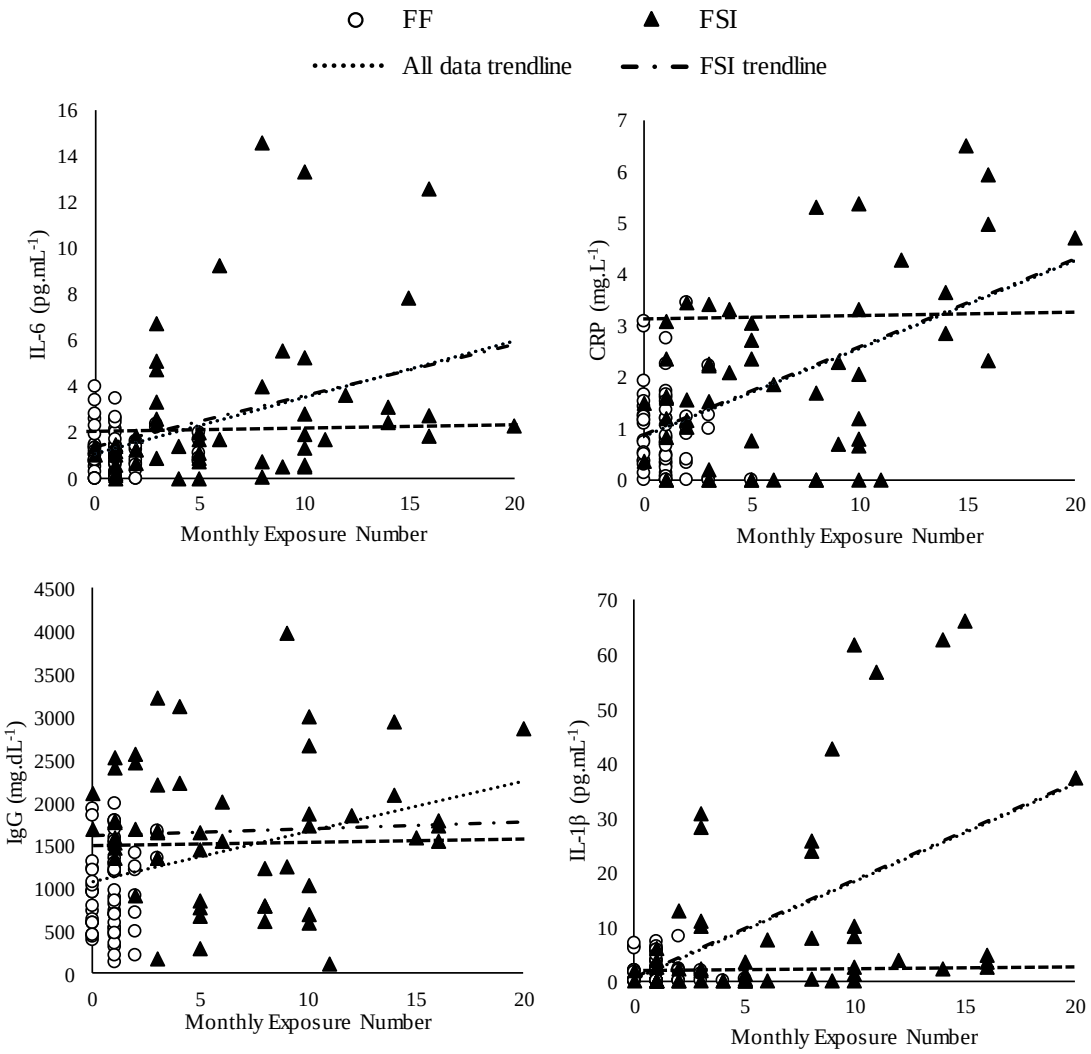

Figure 9.1 *IL-6, CRP, IL-1β, and IgG plotted against monthly wear number. Horizontal dashed line represents the upper limit for each variable, as identified in Table 9.3. IL-6 = interleukin-6, CRP = C reactive protein, IL-1β = interleukin-1 beta, IgG = immunoglobulin G.*

9.4.3 "At Risk" Reference Values Associated with Wear Limits and Ill Health

Haematological variables related to monthly wear numbers (IL-6, IL-1β, CRP and IgG) were grouped according to the upper reference range criteria. Those below the reference limit were classified as the "healthy" group, whilst those above were classified as the "at risk" group.

The current suggested monthly wear limit of 9 wears was associated with groupings for IL-6, IL-1β, CRP and IgG, see Table 9.5 for the association statistics and odds ratios. The 9 wear monthly limit was also associated with the presence of symptoms of ill health (ill health prevalence as referred to in section 9.4.1) (χ^2 (1) = 26.803, $p < 0.001$). Those above the wear limit were 15.74 times (95% CI 2.26 - 32.80) more likely to suffer symptoms of ill health than those below the limit.

Table 9.5. *Association between monthly wear limit (≤ or > 9 wears per month) and participants IL-6, IL-1β, CRP and IgG above or below upper reference levels. Odds ratios based on likelihood of those completing >9 wears per month exhibiting haematological levels above reference limits. * denotes significant association (p < 0.05).*

	Chi squared	*p* value	Odds Ratio (95% CI)
IL-6	11.981	0.002 *	6.27 (2.04 – 19.3)
IL-1β	7.784	0.004 *	7.00 (1.51 – 32.51)
CRP	21.023	0.001 *	12.43 (3.57 – 43.22)
IgG	14.563	0.001 *	7.55 (2.41 – 23.61)

IL-6 = interleukin-6, IL-1β = interleukin-1 beta, CRP = C reactive protein, IgG = immunoglobulin G

The reference limit groupings for IL-6, IL-1β, CRP and IgG were also associated with the presence of symptoms of ill health, see Table 9.6 for the association statistics and odds ratios.

Table 9.6 *Association between haematological group (above or below reference limits) with the presence of symptoms of ill health. Odds ratios based on the likelihood of those above reference limits exhibiting symptoms of ill health. * denotes significant association (p < 0.05).*

	Chi squared	*p* value	Odds Ratio (95% CI)
IL-6	11.695	0.002 *	5.63 (1.96 – 16.20)
IL-1β	5.131	0.040 *	3.67 (1.13 – 11.90)
CRP	10.507	0.004 *	6.05 (1.86 – 19.72
IgG	13.792	0.001 *	6.45 (2.24 – 18.58

IL-6 = interleukin-6, IL-1β = interleukin-1 beta, CRP = C reactive protein, IgG = immunoglobulin G

9.5 DISCUSSION

This study aimed to establish if FSI had a different expression of immunological markers than FF at rest. Furthermore, the study aimed to identify if wear numbers completed effected immunological markers. In addition, the study aimed to evaluate the current suggested guidance of a limit of 9 wears a month and identify if those in "at risk" groups were associated with the prevalence of ill health. Differences in numerous immunological markers were noted between FSI and FF, allowing alternate hypothesis (1) to be accepted. In addition, monthly wear numbers were related to IL-6, IL-1β, CRP and IgG levels, consequently the alternate hypothesis (2) can also be accepted. The current suggestion of a 9 wear limit is appropriate, with those above the limit more likely to be in "at risk" groups for IL-6, IL-1β, CRP and IgG. Moreover, those in "at risk" groups are more likely to experience symptoms of ill health. Alternate hypothesis (3) and (4) can therefore be accepted.

Of the FSI involved in this study, 30% reported symptoms of ill health, compared to 5% of FF. The occurrences reported are lower than the 41% of FSI and 21% of FF who reported ill health in Chapter 4. However, Chapter 4 referred to frequent symptoms since the beginning of their job role, whereas this study gathered information of symptoms experienced within the previous month. The type of symptoms reported

in this study are however similar to that of Chapter 4, reaffirming the prevalence of a unique set of symptoms in the FSI population.

Comparison of FSI and FF resting haematological variables revealed that NEUT, PLT, BASO, NLR, cTnT, IL-6, IL-1β, CRP, and IgG were all greater in FSI. Differences in CBC variables were all larger than their absolute TEM (section 3.9.1.1) and all other variables were greater than their respective ELISA inter assay CV%. However, mean levels fell below the upper reference limit for all variables other than IL-6, IL-1β, IgG and TNFα. This supports the elevated levels of IL-6, IL-1β and IgG reported at baseline in FSI in Chapter 8.

9.5.1 Cardiovascular Event Risk

An increased NLR represents neutrophilia, also indicated by the noted increase in NEUT, in combination with lymphocytopaenia, and is an indicator of systemic inflammation (Guthrie et al. 2013). NLR is of prognostic value for cardiovascular diseases, infections, inflammatory diseases, and numerous types of cancer (Guthrie et al. 2013; Martínez-Urbistondo et al. 2016). NLR is known to increase following acute exercise, remaining elevated but returning towards normal values 6 hrs post exercise (Nieman 1998; Nieman 2000). Neutrophils are involved in all stages of atherosclerosis, they increase the expression of adhesion molecules, limit vasodilation, and can lead to atherosclerotic plaque instability, making the plaque prone to rupture (Soehnlein 2012; Balta et al. 2016). In opposition, regulatory T cell lymphocytes are involved in the inhibition of atherosclerosis by regulating the inflammatory response and therefore low lymphocyte counts represent a poorly regulated immune response (Shah et al. 2014). Consequently, the combined ratio of these two leukocytes acts as an independent predictor of cardiac mortality in patients with coronary artery disease (Papa et al. 2008), those with acute decompensated heart failure (Uthamalingam et al. 2011), patients with myocardial infarctions (Bhat et al. 2013), and in a healthy general population (Shah et al. 2014). The elevated NLR identified in FSI in this study indicate that FSI experience systemic inflammation and may be at a greater risk of a cardiovascular event than FF.

Elevated cTnT in FSI indicates chronic low level myocardial damage. This is in contrast to the findings of Chapter 8, which found no difference between FSI and CON cTnT levels. It is likely that the larger population included in this study and a greater level of homogeneity within the FF group compared to the control group of Chapter 8, has enabled these differences to be identified. Baseline resting measurements of cTnT from healthy populations indicate typical levels of < 10 ng.L^{-1} and 5.60 ± 3.27 ng.L^{-1} in marathon runners (Neilan et al. 2006; Richardson et al. 2018) and 6.2 ng.L^{-1} in elite floorball players (Wedin & Henriksson 2015). This suggests that cTnT exhibited by FF and FSI are both below the healthy upper limit (< 14 ng.L^{-1}) and within the range of other healthy active population groups. Increased resting levels of cTnT in healthy individuals are related to left ventricular wall thickening and left ventricular systolic dysfunction, in addition to all cardiovascular mortality (de Lemos et al. 2010). With each tertile increase of cTnT from non-detectable to > 14 ng.L^{-1}, the hazard ratio of cardiovascular mortality increases. Those with cTnT between 3 - 4.4 ng.L^{-1} have a hazard ratio of 1.6 (95% CI 0.5 - 4.9), a cTnT of 4.4 - 6.6 ng.L^{-1} suggests a ratio of 2.4 (95% CI 0.9 - 6.1), and individuals with cTnT 6.6 - 14 ng.L^{-1} the hazard ratio of cardiovascular

mortality is 4.6 (95% CI 2.1 - 10.0) (de Lemos et al. 2010). FF consequently have a lower hazard ratio of cardiovascular mortality than FSI, with FF exhibiting 3.62 ± 0.87 ng.L^{-1} and therefore either falling in undetectable levels or within the first detectable tertile of 3 - 4.4 ng.L^{-1}, compared to FSI who exhibited 4.79 ± 1.77 ng.L^{-1} giving the average FSI a hazard ratio of 2.4.

MPV is also associated with incidence of myocardial infarction and coronary artery disease (Vizioli et al. 2009; Klovaite et al. 2011; Sansanayudh et al. 2014). However, MPV displayed no differences between FSI and FF and although FSI PLT (240 ± 66 x10^9.L^{-1}) was greater than FF PLT (185 ± 39 x10^9.L^{-1}), it remained below the upper reference range. This is similar to the PLT noted in previous FF studies of 264 ± 53 x10^9.L^{-1} (Smith et al. 2011), 257 ± 62 x10^9.L^{-1} (Smith et al. 2014) and 241 ± 11 x10^9.L^{-1} (Hunter et al. 2017), and resting PLT reported in Chapter 5 (209 ± 43 x10^9.L^{-1}) and Chapter 8 (240 ± 50 x10^9.L^{-1} and 244 ± 51 x10^9.L^{-1}) from FSI.

A key finding of this study is the elevated levels of inflammatory cytokines IL-6, IL1-β and acute phase protein CRP, in FSI. IL-6 is involved in the increase of cell adhesion molecules, platelet reactivity and CRP release, and therefore may be involved in atherosclerosis formation (Lindmark et al. 2001; Tzoulaki et al. 2005; Schuett et al. 2009). FSI exhibited a mean IL-6 of 2.98 ± 3.56 pg.mL^{-1}, in the upper quartile (> 2.28 pg.mL^{-1}) which has previously been related to a 2.3 times higher risk of myocardial infarction in apparently healthy men (Ridker et al. 2000). IL-6 levels in FSI reported in this study are not as great as those reported at rest by Watt et al (2016) (7.4 – 17.0 pg.mL^{-1}). However, this study included FSI with an average of 6 ± 5 exposures a month, whereas FSI who participated in the study conducted by Watt et al (2016) had completed 15 wears in a 4 week period. The participants therefore experienced a greater number of exposures than the average experienced by the FSI involved in this study.

IL1-β is also involved in increasing the expression of adhesion molecules, inducing procoagulant activity, and the stimulation of CRP synthesis and release (Jialal et al. 2004; Barksby et al. 2007; Ridker et al. 2011). Although IL1-β has not yet been established as a predictor of cardiovascular events with set classifications of risk, perhaps due to the variability in its measurement in plasma (Ridker et al. 2011). However, gene polymorphisms causing increased IL1-β are associated with risk of coronary artery disease and cardiovascular events (Tsimikas et al. 2014), and IL1-β is being targeted for atheroprotective interventions (Ridker 2016). This is the first study to identify elevated IL1-β levels in FSI.

CRP has also been detected in atherosclerotic lesions (Yasojima et al. 2001) and elevated levels have been described to increase adhesion molecules and reduce endothelial vasodilatory capacity (Fichtlscherer et al. 2000; Jialal et al. 2004). Whilst the mean CRP exhibited by FSI was 1.98 ± 1.74 mg.L^{-1}, the variation in levels was greater (as demonstrated by the large SD) and some individuals displayed CRP > 3.0 mg.L^{-1}, indicating a 2 fold increase in relative risk of cardiovascular disease compared to those with CRP < 1.0 mg.L^{-1} (Pearson et al. 2003). The elevated presence of IL-6, IL1-β, and CRP therefore demonstrates FSI experience chronic systemic inflammation and are at an increased risk of cardiovascular events.

9.5.2 Wear Limits

IL-6, IL-1β, CRP and IgG were related to the number of wears that had been completed in the previous month, with 11 - 29% of variance in these markers explained by wear numbers. These findings support the theory that performing a greater number of fire exposures elevates cytokine levels. The wear limit informally suggested and followed by some training centres (as indicated in Chapter 4) is 9 wears per month. The evaluation of this limit indicates that those above the limit are more likely to be in the "at risk" group for IL-6, IL-1β, CRP and IgG, with values above upper reference values. This indicates that those FSI conducting a greater number of wears may be at an increased risk of a future cardiovascular event. Moreover, individuals completing > 9 wears a month are 15.74 times more likely to suffer symptoms of ill health than those below the limit.

9.5.1 Overtraining Like Syndrome

The "at risk" groups for IL-6, IL-1β, CRP, and IgG were also associated with an increased likelihood of experiencing symptoms of ill health (IL-6 OR = 5.63, IL-1β OR = 3.67, CRP OR = 6.05, IgG OR = 6.45). These symptoms are similar to the symptoms of ill health reported in overtraining (fatigue, sleep disturbances, headaches, colds, and flu like illnesses) (Fry et al. 1991; Smith 2000). Consequently, this association between symptoms reported by FSI and elevated cytokine levels supports the cytokine hypothesis of overtraining (section 2.6).

In addition to IL-6, IL-1β and TNFα instigating "sickness" behaviours (see sections 2.6 and 8.5.3), ill health in overtraining may also be caused by a late immunosuppression that may occur following hyper-inflammation that follows exercise (Smith 2000). It is proposed that humoral immunity is up-regulated and consequently cellular immunity is suppressed during this period, increasing the risk of viral or bacterial infection (Buyukyazi et al. 2004; Lancaster et al. 2004; Smith 2004; Zhao et al. 2012; Kakanis et al. 2014). An increase in humoral immunity may also be related to an increase in allergies and immunoglobulins (Smith 2004). IgG was elevated in FSI (1688 ± 836 $mg.dL^{-1}$) in comparison to FF (917 ± 485 $mg.dL^{-1}$), with the mean level exhibited greater than the upper reference limit, possibly indicating increased humoral immunity and inflammation. However, the balance between cellular and humoral immunity is also carefully controlled by the regulation of specific cytokines, with IL-2, IL-12 and interferon gamma (IFNγ) being crucial in the development of Th1 cells that are instrumental in cellular immunity, and IL-4, IL-5, IL-10, IL-6, and TNFα being involved in the development of Th2 cells that are involved in humoural immunity (Glimcher & Murphy 2000; Smith 2003; Kidd 2003). Whilst the elevated IL-6 and TNFα in FSI support the possible increase in humoural immunity, a larger array of cytokines should be investigated in future studies to further understand the balance between humoral and cellular immunity in FSI. Overall, the association between reported symptoms of ill health, cytokine levels, and wear numbers indicates that FSI can experience an overtraining like syndrome.

9.5.2 Limitations

Unlike IL-6, IL-1β, CRP and IgG numerous haematological variables that were greater in FSI than FF were not associated with number of wears completed, or with any other demographic and work history detail. In addition, not all variation in IL-6, IL-1β, CRP, and IgG was explained by monthly exposure numbers. It is reasonable to suggest that there are many other factors that were outside of the scope of this specific study that may have influenced these variables, such as the overall thermal load experienced from the wears completed, the type of wears completed, level of smoke/particulate exposure, and FSI use of hydration guidance and pre/post cooling interventions for each wear performed. Future research should further explore the influence that other factors may have on systemic inflammation in FSI.

Participants who reported themselves as FSI were classified as such, regardless of wear exposure number, to ensure the data represented the FSI population. This resulted in two FSI with no wear completions in the previous month being included in the FSI group, despite them having a lower monthly wear number than the FF group mean. Due to the logistical difficulty of accessing this population and cost of blood sample analysis this study offers only a singular snapshot of FSI and FF immunological status. Presence of symptoms of ill health were also subjective and not verified by a medical professional. Furthermore, there are many risk factors of cardiovascular events, of which not all were collected in this study. Additional measures of overtraining and Th1/Th2 balance, such as changes in performance, changes in HR variability, testosterone/cortisol ratio, glucose depletion, IL-4, IL-12 and IFNγ, were also not included in this study and should be considered for future projects. The duration between wears was also not detailed as part of this study and should be included in future research. Overall, it is suggested that long term monitoring over multiple months is conducted to establish further extraneous variable information and identify changes in haematological variables.

9.6 CONCLUSION

In conclusion, FSI specifically need to be targeted for interventions to help reduce the occurrence of elevated haematological measures, as findings from this study suggest they are at an increased risk of a cardiovascular event and symptoms of ill health compared to FF. Furthermore, FSI exhibit elevated cytokine levels that are associated with ill health and may indicate FSI experience a syndrome similar to an overtraining response. Moreover, increased levels of IL-6, IL-1β, CRP, and IgG are related to the number of wears FSI complete in a month. The current suggestion of a 9 wear per month limit is reasonable guidance, as those above the limit are 15 times more likely to experience symptoms of ill health. Future research should investigate the impact that interventions designed to reduce the thermal load from frequent fire exposures have on the chronic systemic inflammation prevalent in FSI.

10 GENERAL DISCUSSION

This thesis aimed to gain an understanding of FSI current working practices in the UK and identify both the acute and chronic consequences of these practices. A secondary aim of this thesis was to provide evidence based recommendations for FSI to reduce the risk of heat exhaustion, symptoms of ill health, and cardiovascular events. This general discussion will summarise the principal findings from each experimental study chapter and then discuss collective findings with regards to acute and chronic responses. From this recommendations for FSI working practices will be outlined and future directions of research will be suggested.

10.1 Principal Findings

The first experimental chapter (Chapter 4) utilised a survey to gather information on current working practices of UK FSI. FF were also invited to complete a shorter survey to offer a comparative population group. The study enabled demographic details of FSI to be collected, with identification of the typical age of FSI (43 ± 7 yrs), proportion of male and females (95% and 5%, respectively), and average time they had been working as a FSI for (5 ± 5 yrs). The survey also documented that FSI experience fire exposures more frequently than FF (13 ± 8 vs 1 ± 3 exposures a month), and that currently there is no universal limit to the number of wears completed a month by FSI, although 9 wears was commonly reported. The study also highlighted that different wears may cause varying levels of physiological strain, with CFBT perceived as the most straining exposure. Findings also indicated that further research was needed into the use of pre-cooling methods, as they were rarely used by FSI and little guidance is available, either in the literature or within the Fire and Rescue Service, on methods, dosage, or duration of use. The key finding of this study was that 41% of FSI experience new symptoms of ill health since beginning their role, with a common set of symptoms presented, including: fatigue, mood swings, broken sleep, heavy sweating, and heart palpitations. These symptoms are similar to that of an overtraining like syndrome. FSI who felt exhausted after a wear were 16.8 times more likely to suffer new symptoms than those who felt fine, and FSI who completed > 9 wears a month were 9 times more likely to experience symptoms of ill health. The association between wear numbers and symptoms of ill health, combined with the lack of an evidence based wear limit, indicated that further investigation into the chronic consequences of wear exposures was needed.

Study 2 (Chapter 5) set out to assess the acute physiological, perceptual, and immunological responses to different types of training exposures, different roles performed, and exposure patterns completed over a day. Multi-compartment exposures were identified as generating the greatest increases in T_{re} (0.49 ± 0.28ºC), with the condition setter role also causing a greater rise in T_{re} than the instructing role (0.67 ± 0.29ºC). In the majority of cases FSI remained within safe ranges of T_{re}, however on two occasions a Tre of > 39ºC was exhibited. FSI also experienced increased inflammatory levels after a day of exposures, in addition to low levels of myocardial damage, as noted by elevated cTnT (+36%). Exposure patterns including a multi-compartment wear caused the greatest levels of inflammation.

To minimise the acute responses identified in study 2 (Chapter 5), practical pre-cooling methods, that were ascertained in study 1 (Chapter 4), were assessed. Study 3 (Chapter 6) found that with a 15 min pre-cooling period only ice slurry consumption was effective at reducing T_{re} prior to exposure (-0.24ºC) and minimising T_{re} and TS during a laboratory simulated fire exposure. Both the phase change vest and forearm immersion showed no beneficial consequences. The study also found that a single bolous of ice slurry (500ml) was effective, rather than specifying dosage by body mass as previously suggested in the literature. However, pre-cooling had no effect on inflammatory response, although only IL-6 was measured.

Study 4 (Chapter 7) set out to evaluate the validity and reliability of the HOTT, which was designed to identify heat tolerance using a H_{prod} controlled protocol. This protocol enables non-bias comparisons between individuals with different biophysical characteristics to be made and would allow the chronic consequences of regular fire exposures to be investigated. The study found the test was valid, being able to successfully identify individuals as heat tolerant and intolerant and generating similar T_{re} responses compared to the Israeli Defense Force HTT. The HOTT was also reliable, with strong ICC, and acceptable CV, mean bias and LOA for peak T_{re} (TEM (CV%) = 0.08°C (0.2%)), peak HR (TEM (CV%) = 2 $b.min^{-1}$ (1%)) and SR TEM (CV%) = 0.12 $L.hr^{-1}$, (9.9%)). A continuum based interpretation method of heat tolerance was recommended.

Study 5 (Chapter 8) subsequently used the HOTT to assess the heat tolerance of FSI compared to a non-wearing control group. FSI demonstrated an acclimatised status, having a reduced peak T_{re} (-0.42ºC) and TS (-1.0) and males having an increased SR (+0.25 $L.hr^{-1}$) compared to CON. The study also found that improved tolerance was associated with increased symptoms of ill health and an increased number of fire exposures having been completed. In addition, FSI displayed elevated baseline levels of IL-6, IL-1β, and IgG indicating that they may experience chronic systemic inflammation. FSI who completed 2 – 4 exposures a month were able to maintain their thermotolerance without the presence of ill health symptoms. Overall, this study indicates that FSI acclimatise to heat exposures, but this can lead to a maladaptation with increased symptoms of ill health.

To further investigate the prevalence of chronic systemic inflammation identified in study 5 (Chapter 8) and the prevalence of overtraining symptoms of ill health, study 6 (Chapter 9) analysed resting haematological and inflammatory markers in FSI and FF in relation to working and exposure history details. The study also evaluated the 9 wear per month exposure limit which was identified in study 1 (Chapter 4) and mentioned by the Chief Fire Officers' Association (2015). NLR, PLT, cTnT, IL-6, IL-1β, IgG and CRP were all greater in FSI than FF. In FSI IL-6, IL-1β, CRP and IgG were significantly related to monthly wear numbers, with wear numbers explaining 11 - 29% of variance in these markers. When these variables were grouped into "at risk" and "healthy" categories, according to literature's upper ranges and cardiovascular risk classifications, those above the 9 wear per month limit were 6 - 12 times more likely to be in "at risk" groups and were 16 times more likely to experience symptoms of ill health.

A summary of each experimental chapter's hypotheses can be found in Table 10.1.

Table 10.1 *Hypotheses for each experimental study within this thesis.*

HYPOTHESES	ACCEPT	REJECT
Study 1: Fire Service Instructors Working Practices: A UK Survey		
1) The majority of FSI would not use pre or post cooling techniques	✓	
2) A greater number of FSI would report symptoms of ill health than FF	✓	
3) Presence of ill health would be associated with exposure numbers.	✓	
Study 2: The Acute Effect of Training Fire Exercises on Fire Service Instructors		
1) T_{re} responses would be different between exposure exercises	✓	
2) T_{re} responses would be different between exposure roles	✓	
3) A day of exercises would increase inflammatory and cardiac risk markers	✓	
4) Inflammatory markers would be different between exercise patterns	✓	
Study 3: Practical Pre-cooling Methods for Fire Service Instructors		
1) Phase change vest, forearm cooling, and ice slurry would reduce physiological and perceptual strain compared to control		✓
2) Ice slurry would generate the greatest reductions in strain compared to control	✓	
3) All methods would result in a reduced inflammatory response		✓
Study 4: The Validity and Reliability of a New Occupational Heat Tolerance Test		
1) T_{re} responses would be similar between the new and standard tests	✓	
2) Physiological and perceptual responses would be similar at the end of both HOTT trials	✓	
Study 5: Heat Tolerance of Fire Service Instructors		
1) FSI would have an increased tolerance compared to a control group, as identified by a reduced T_{re} at the end of the HOTT	✓	
2) FSI would have a decreased inflammatory response to the HOTT compared to a control group, as identified by decreased inflammatory markers such as IL-6		✓
3) FSI and the control group would maintain their physiological, perceptual, and inflammatory responses following a 2 month period of normal working practices.	✓	
Study 6: Haematological and Immunological Measures in Fire Service Instructors and Firefighters		
1) FSI would exhibit greater IL-6, CRP, cTnT and platelet volume and counts than FF	✓	
2) Increased fire exposure numbers will be related with elevated levels of cytokines	✓	
3) There would be an association between those above the 9 wear limit and those exhibiting cytokine levels above the reference limits.	✓	
4) There would be an association between immunological markers and reports of ill health	✓	

10.2 Fire Service Instructors as a Population

Prior to the research presented within this thesis, there had been few investigations into the responses of FSI as a population, with just Eglin et al. (2004, 2005) and Watt et al. (2016) giving specific focus to FSI. However, the frequency of wears made by FSI, combined with the anecdotal symptoms of ill health, indicated that they may have a unique occupational role in comparison to standard FF, who are the focus of a plethora of investigations (Table 2.1). The work by Watt et al. (2016) provided the first assessment of the chronic health consequences of the FSI role, with indications of reduced physiological capabilities (-7% V̇O2max and -15% lung forced vital capacity) combined with elevated cytokine levels compared to a control group. This pilot work by Watt et al (2016) therefore highlighted the need for further investigation into FSI with reference to their working practices and health. Watt et al (2016) also suggested that FSI may experience an overtraining like syndrome, which this thesis sought to support or refute. Chapter 1 provided the first empirical evidence of the prevalence of ill health amongst FSI and confirmed differences in fire exposure completions between FSI and FF. Moreover, the study also provided a list of symptoms unique to FSI that were similar to those of an overtraining response.

10.3 Acute Fire Exposure

Eglin et al (2004) previously provided a detailed assessment of physiological responses to fire exposures completed by FSI. However, only Watt et al (2016) had previously assessed the immunological responses in FSI. Chapter 5 demonstrated elevated T_{re} (37.92 ± 0.31°C) and HR (147 ± 20 b.min^{-1}) post fire exposure, supporting the work of Eglin et al (2004) and Watt et al (2006). The majority of responses remained within safe zones, although on some occasions this was elevated above 39ºC, highlighting the risk of EHI in some instances. Inflammatory responses were also similar to that noted by Watt et al (2016). Chapter 5 also assessed the myocardial damage that occurred in FSI following a fire exposure, with findings indicating that acutely they may experience a similar low level of damage as previously reported in FF (Hunter et al. 2017). Furthermore, the identification of exposure types, patterns, and FSI roles which may elevate the risk of EHI can now inform working practices.

Acute interventions to reduce the risk of EHI have previously been suggested, with the maintenance of hydration status, and use of post and pre-cooling techniques highlighted. Hydration status and post cooling have been intensively researched with clear guidance surrounding fluid intake and cooling method use for the Fire and Rescue Service (McLellan & Selkirk 2006; Eglin 2007; Cheung et al. 2010). In comparison there is a dearth of pre-cooling advice available, both in literature and from the Chief Fire Officers' Association (2015) for FF. Consequently, experimental study 3 (Chapter 6) was conducted to provide specific guidance for FSI. T_{re} cooling with ice slurry consumption (-0.24 ± 0.09ºC) were close to that previously noted in athletic populations (0.32 – 0.66ºC), despite being the first to utilise a single bolus of slurry. Consequently, recommendations for preparation and recovery for live fire exposures can now be made in relation to the physiological consequences experienced. The integration of preparation and recovery methods with fire exposure and factors contributing to the risk of EHI are presented in Figure 10.2.

10.4 Chronic Health Consequences

The occurrence of symptoms of ill health established in Chapter 4 were investigated in both Chapter 8 and 9. Chapter 8 identified 6/11 participants exhibited symptoms of ill health (55%), whilst 16/53 (30%) FSI reported ill health in Chapter 9. These prevalence rates sit either side of that noted in Chapter 4 (41%). Consequently, the persistence of similar symptoms, exhibited only in FSI, throughout this thesis supports the suggested occurrence of an overtraining like syndrome in FSI. Previous work has indicated that overtraining symptoms may be caused by elevated cytokine levels as a consequence of high exercise loads with minimal recovery time (Smith 2000). Booth et al. (2006) support this hypothesis, having noted elevated IL-6 and CRP with the presence of overtraining in military recruits. Chapter 9 evidenced that ill health symptoms were related to elevated IL-6, IL-1β, CRP and IgG, whilst Chapter 8 also identified that improved heat tolerance was related to increased prevalence of symptoms.

In three experimental Chapters within this thesis (Chapter 5, 8, and 9) resting haematological measures have been collected. These have demonstrated elevated mean IL-6 (2.08 - 4.09 $pg.mL^{-1}$), CRP (1.46 - 1.98 $mg.L^{-1}$), IL1-β (11.56 - 20.52 $pg.mL^{-1}$), and IgG (1688 - 2462 $mg.dL^{-1}$) compared to healthy reference ranges or comparative population groups used within the thesis. These detected elevated cytokines indicate that FSI experience chronic systemic inflammation. Figure 10.1 displays the resting and post heat exposure levels of key haematological markers measured from FSI in comparison to healthy reference ranges. Inflammation is directly related to the risk of cardiovascular events, due to its involvement in the formation of atherosclerotic plaques (Koenig 2013). The risk of a cardiovascular event has previously been noted to be increased in FF following live fire exposures (Kales et al. 2007) and it was postulated that repeated exposures completed by FSI could cause them to be at an increased risk of events. Without an acute fire exposure stimulus, FSI exhibit increased predictors of cardiovascular events, namely IL-6, CRP, and IL-1β compared to FF, as demonstrated in Chapter 9.

The relationships between ill health, chronic inflammation, and heat tolerance indicated that the number of wears FSI complete may be a key causative factor. This would support the concept of an overtraining like syndrome in FSI, with high wear numbers equating to a high training load, as a consequence of the physiological strain experienced from each individual wear (as documented in Chapter 5). Findings from Chapter 9 demonstrate that FSI who follow the informal suggestion of a monthly 9 wear limit have lower cytokine levels and consequently are at a reduced risk of overtraining symptoms of ill health and of cardiovascular events. Figure 10.2 displays an integrative model of the chronic responses to heat exposures. Overall, this thesis establishes that there are chronic health risks associated with the FSI occupation, namely the risk of an overtraining like syndrome and cardiovascular events, both of which may be minimised with alterations to working practices.

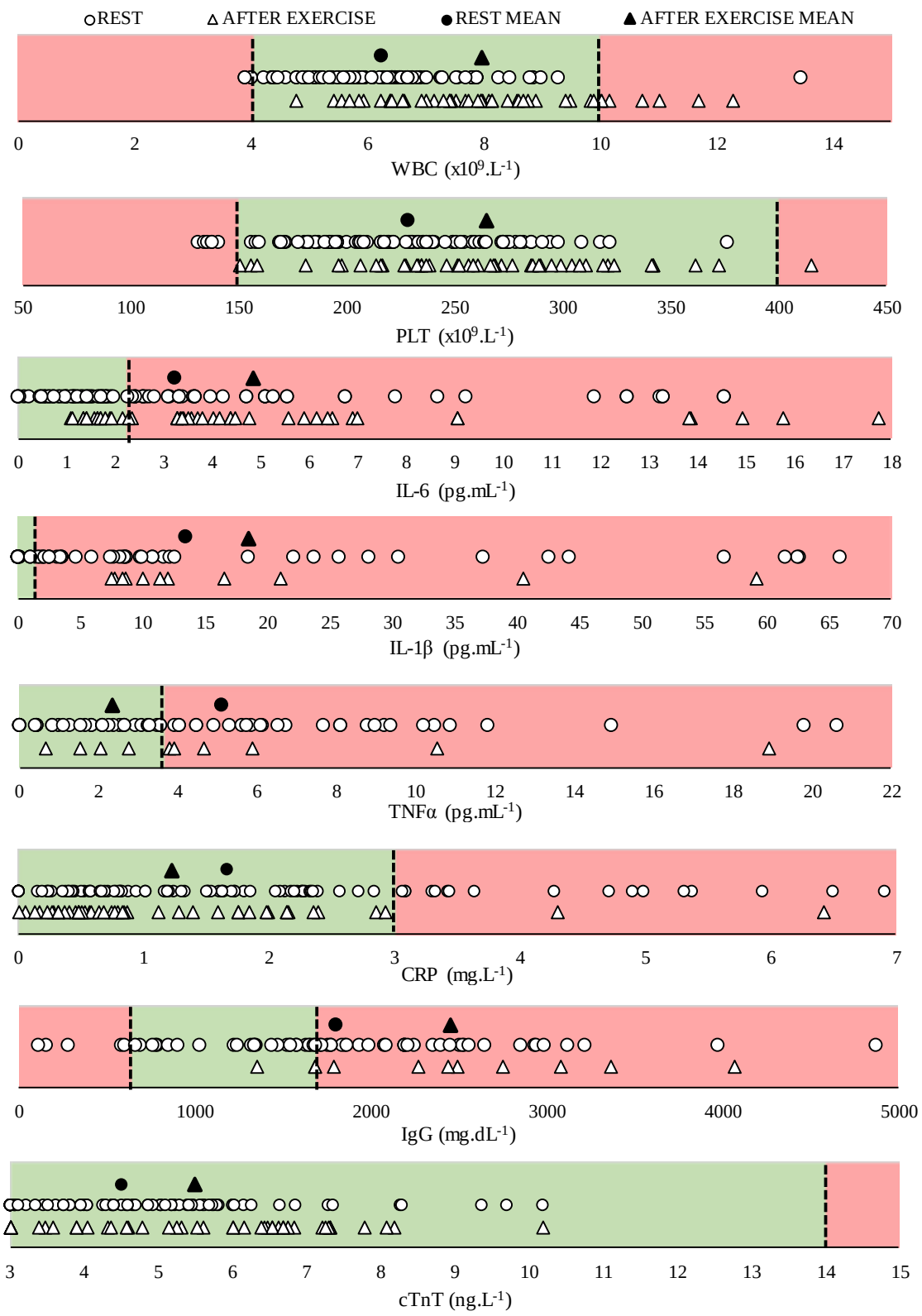

Figure 10.1 *Summary of biomarkers measured in Chapters 5, 8 and 9 from FSI at rest or following activity in a hot environment. The healthy reference range in the general population at rest is highlighted in green, with red shading denoting values above/below this range, as described in Chapter 9.*

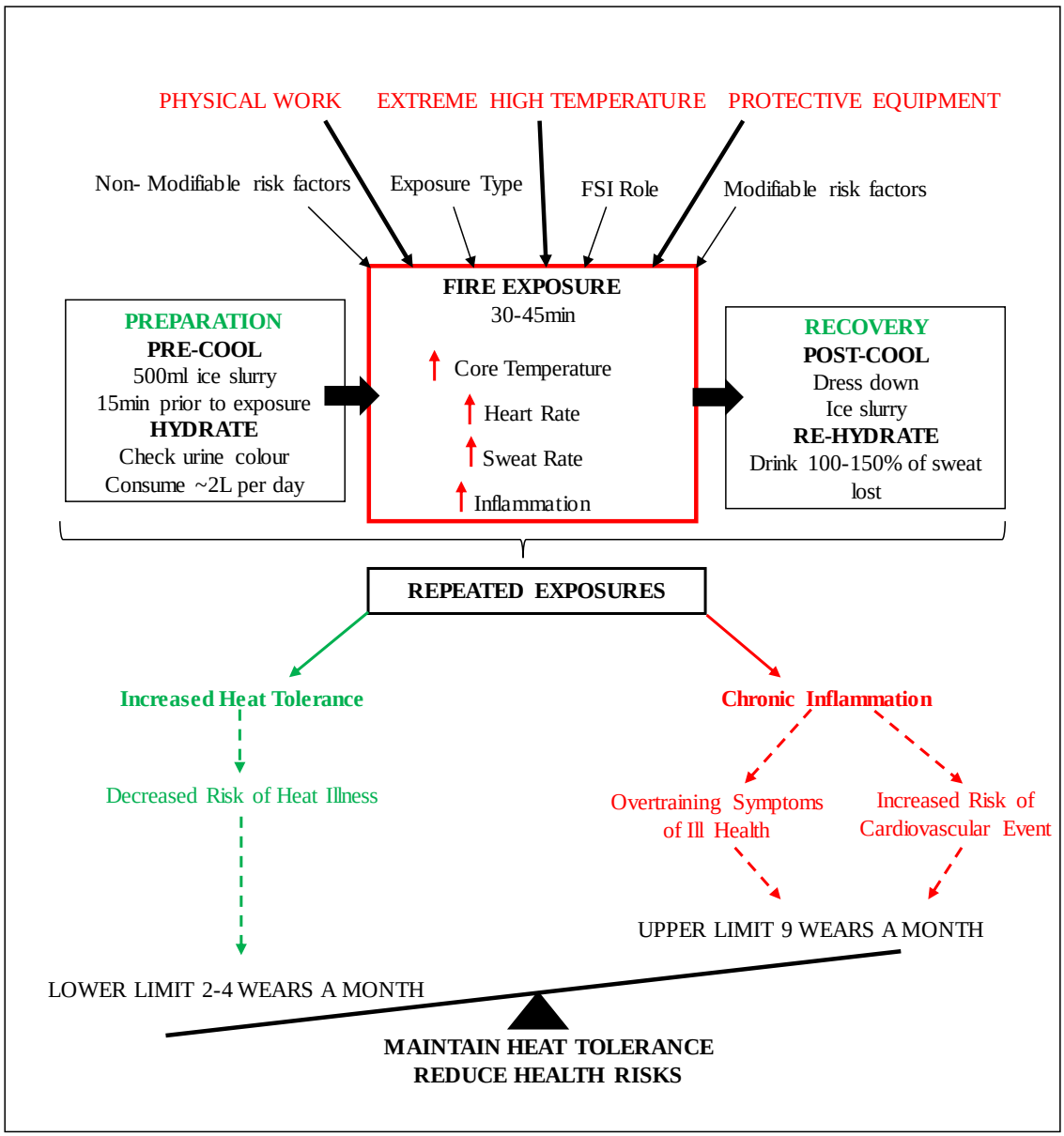

Figure 10.2 *Integrative model of the acute and chronic consequences of fire exposures, with contributory factors and suggested interventions.*

10.5 Recommendations for Fire Service Instructors' Working Practices

As a consequence of the findings of this thesis numerous recommendations can be made to FSI working practices to reduce the acute and chronic consequences of repeated fire exposures.

1) Further education and clearer guidance is needed on the importance of preparation and recovery methods including maintenance of a hydrated state and use of post cooling.

2) Courses should be planned to allow for rotation of FSI roles and to avoid repetition of multi-compartment exposures where possible.

3) Pre-cooling using a minimum of 500ml of ice slurry 15 min prior to a wear should be implemented to reduce the risk of EHI and overall thermal load experienced by FSI.

4) Training to become a FSI should include importance of controlling modifiable risk factors for heat tolerance, awareness that FSI will likely become more heat tolerant than recruits, and education that extended durations away from wearing may reduce FSI tolerance.

5) A 9 wear per month limit should be universally introduced to reduce the risk of chronic inflammation, overtraining symptoms and cardiovascular events.

6) Regular occupational health visits should check for elevated markers of chronic inflammation and overtraining symptoms of ill health.

These recommendations will be communicated to the National Fire Chiefs Council and the CFBT Instructor user group. They will also be shared with the National Operational Guidance team. An educational package commissioned by the Fire Brigades Union will also be created, providing information on consideration for preparation, recovery and heat illnesses in relation to fire exposures. Within the package the findings of this thesis will also directly inform a FSI welfare section. The learning resources created will then be available nationally to Fire and Rescue Services.

10.6 Future Directions

The findings of the experimental studies completed as part of this thesis have highlighted numerous potential areas for future research. Whilst the acute response to different CFBT wear exposures have been documented, there are still numerous other types of training exposures, such as the different types of BA wears, that have not specifically been assessed and could provide additional information on the total thermal load experienced by FSI. Furthermore, the use of the acute intervention of pre-cooling warrants additional investigation in a field study, although the ability to control the environmental temperatures experienced and workload of FSI would require careful consideration.

To expand upon the findings of this thesis, the chronic consequences of following the recommended wear limit should be assessed, with reference to the prevalence of overtraining symptoms and markers of cardiovascular risk. Inclusion of long term monitoring of the thermal load FSI experience would enable further exploration into the relationship between physiological strain, specifically elevated T_c, and levels of chronic systemic inflammation. This would provide additional insights into the chronic outcomes of FSI working practices.

In addition, primarily male individuals have participated in the experimental studies presented. This is likely a consequence of female individuals being a minority population within the Fire and Rescue Service and the logistical issues of testing due to geographical locations. However, with a recent increase in female FF recruits across the UK and the physiological and thermoregulatory differences between sexes, this may be an area that warrants future investigation. Moreover, through the process of data collection, anecdotal reports of issues specific to female health and welfare have been raised. Specifically, the HOTTs completed by two female FSI within study 5 (Chapter 7) were performed in the same menstrual cycle phases, with similar levels of heat tolerance detected, however it is unknown how menstrual phases may impact heat tolerance. Furthermore, with an aging population of female FF the effect that the menopause may have on HOTT responses is unclear. Consequently, future research should focus specifically on female individuals within the Fire and Rescue Service.

11 CONCLUSION

This thesis provides insight into the acute and chronic consequences of FSI working practices. FSI currently experience fire exposures a median of 13 ± 8 times a month. Acutely, live training fire exposures cause increased physiological and perceptual strain in addition to an inflammatory response and low levels of myocardial damage. Pre-cooling with ice slurry consumption is identified as a method to minimise the physiological and perceptual strain experienced. This thesis also evidences that frequent fire exposures can lead to the development of an acclimatised state, with increased heat tolerance. However, maladaptation can occur with high volumes of exposure, whereby systemic chronic inflammation develops, which is linked to overtraining symptoms of ill health and an increased risk of elevated predictors of cardiovascular events. Consequently, this thesis suggests that a balance should be sought between maintenance of heat tolerance and minimisation of maladaptation through the introduction of a monthly wearing limit.

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13 APPENDICES

13.1 Appendix 1

HEAT ILLNESS SYMPTOMS

PRE EXPOSURE		POST EXPOSURE	
SYMPTOMS	SCORE	SYMPTOMS	SCORE
Feeling Tired		Feeling Tired	
Swelling		Swelling	
Cramps		Cramps	
Nausea		Nausea	
Dizziness		Dizziness	
Thirst		Thirst	
Vomiting		Vomiting	
Confusion		Confusion	
Muscle weakness		Muscle weakness	
Heat sensations on head or neck		Heat sensations on head or neck	
Chills		Chills	
Stopping sweating		Stopping sweating	
Feeling lightheaded		Feeling lightheaded	
TOTAL		**TOTAL**	

13.2 Appendix 2

Fire Service Instructor Wear Survey

1) Welcome to the Survey

This survey is being conducted by the Centre for Sport, Exercise Science and Medicine at the University of Brighton. The survey is being conducted on behalf of the Fire Service for the purpose of understanding current Fire Service Instructor workloads and working practices in hot conditions.

The answers given in this survey will remain anonymous. Data from the survey will remain on a password protected computer at the University of Brighton. Raw data will only be viewed by the research staff of the University of Brighton. Individual responses will not be shared with the Fire Service. Any reports as a result of the survey will be sent to all those participating in the survey that have provided an email address and requested the information at the end of the survey.

Please complete as many questions as possible, however if you do not know the answer or wish not to respond then please leave it blank and move onto the next one.

* denotes similar questions were also asked of Firefighters.

2) Demographics

*1. What is your age?

*2. What is your sex?

*3. How long have you been a fire service instructor?

*4. What brigade are you a part of?

3) Working Practices

* 5. How many wears have you completed in the previous month?

BA Live Fire Wears

6. In the last month, how many BA live fire wears have you completed?

7. How long does an average BA live fire wear last?

8. What role do you usually have during a BA live fire wear?

BA Cold/No Smoke Wears

9. In the last month, how many BA cold/no smoke wears have you completed?

10. How long does an average BA cold/no smoke wear last?

11. What role do you usually have during a BA live fire wear?

Compartment Fire Behaviour Training (CFBT) Wears

12. In the last month, how many CFBT wears have you completed?

13. How long does an average CFBT wear last?

14. What role do you usually have during a CFBT wear?

15. Please rank the wears in the order that you find the most physically straining (eg. hardest to complete, greatest heart rate at the end, sweat the most). 1 being the easiest, 3 being the hardest.

BA live fire

BA cold/no smoke

CFBT

16. Do you think you do too many wears in a week?

- Yes
- No

17 . Do you have a limit to the number of wears you complete in a week?

- Yes
- No

If yes, what is the limit?

18. Please provide details of any other exposures/working practices that you regularly complete that you think may be relevant to this survey.

4) Preparation

19. Is there an allocated time period for you to prepare yourself for a hot wear?

- Yes
- No

If yes, what is the allocated period?

20. On average, how much time do you have to prepare yourself before a hot wear?

21. Which of these preparation methods do you use before a wear?

- checking hydration levels
- drinking ice slurry
- wearing ice vests
- wearing phase change vests
- placing forearms in buckets of cold water
- no preparation method

Other (please specify)

22. Do you have any guidelines or specific practices to ensure you are hydrated before a wear?

- Yes
- No

If yes please give details

23. On the scale below please rate how prepared you usually feel for a wear.

- Unprepared
- Only just prepared
- Prepared
- Fully prepared

5) Recovery

24. Is there an allocated time period for you to recover from a hot wear?

- Yes
- No

If yes, what is the allocated period?

25. On average, how much time do you have to recover from a hot wear?

26. What do you do in your recovery period?

- drink ice slurry
- drink water
- place forearms in buckets of cold water
- no recovery routine

Other (please specify)

27. On the scale below please rate how you feel following a recovery period after a wear.

Not recovered, exhausted and extremely hot.	Only just recovered, a little worn out and warm, but OK	Recovered, feeling fine	Fully recovered, feeling great

6) Health

* 28. Are you currently suffering from a cold or the flu?

- Yes
- No

* 29. In the last year, how often have you suffered from a cold or the flu?

* 30. Do you frequently suffer from any other illnesses or symptoms that you did not experience often before becoming an instructor?

- Yes
- No

If yes, please specify

* 31. How many sick days have you taken in the previous year?

* 32. On the scale below please rate how you feel at the end of an average wearing week.

	Ill	Exhausted	Tired	OK	Good	Great
Physically	Physically Ill	Physically Exhausted	Physically Tired	Physically OK	Physically Good	Physically Great
Mentally	Mentally Ill	Mentally Exhausted	Mentally Tired	Mentally OK	Mentally Good	Mentally Great

33. Do you feel comfortable reporting illness at work and taking sick leave?

- Yes
- No

7) Final comments

34. If you have any additional comments you would like to make, then please fill in the box below.

35. Please supply an Email address if you would like to be contacted about further research and findings

36. Do you wish to receive any reports generated from the findings of this survey?

- Yes
- No

Thank you for taking part in the survey.

13.3 Appendix 3

INTERNATIONAL PHYSICAL ACTIVITY QUESTIONNAIRE

This questionnaire originates in the paper by Craig et al. (2003)

The questions will ask you about the time you spent being physically active in an average week over the last 2 months. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Think about all the **vigorous** and **moderate** activities that you did. **Vigorous** physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. **Moderate** activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal.

JOB-RELATED PHYSICAL ACTIVITY

The first section is about your work. This includes paid jobs, farming, volunteer work, course work, and any other unpaid work that you did outside your home. Do not include unpaid work you might do around your home, like housework, yard work, general maintenance, and caring for your family. These are asked in Part 3.

1. Do you currently have a job or do any unpaid work outside your home?

☐ *.5.4.1 Yes*

☐ No → ***Skip to PART 2: TRANSPORTATION***

The next questions are about all the physical activity you did in the **last 7 days** as part of your paid or unpaid work. This does not include traveling to and from work.

2. During the **last 7 days**, on how many days did you do **vigorous** physical activities like heavy lifting, digging, heavy construction, or climbing up stairs **as part of your work**? Think about only those physical activities that you did for at least 10 minutes at a time.

_____ **days per week**

☐ No vigorous job-related physical activity → ***Skip to question 4***

3. How much time did you usually spend on one of those days doing **vigorous** physical activities as part of your work?

_____ **hours per day**
_____ **minutes per day**

4. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the **last 7 days**, on how many days did you do **moderate** physical activities like carrying light loads **as part of your work**? Please do not include walking.

_____ **days per week**

☐ No moderate job-related physical activity → ***Skip to question 6***

5. How much time did you usually spend on one of those days doing **moderate** physical activities as part of your work?

_____ **hours per day**
_____ **minutes per day**

6. During the **last 7 days**, on how many days did you **walk** for at least 10 minutes at a time **as part of your work**? Please do not count any walking you did to travel to or from work.

_____ **days per week**

☐ No job-related walking → ***Skip to PART 2: TRANSPORTATION***

7. How much time did you usually spend on one of those days **walking** as part of your work?

_____ **hours per day**
_____ **minutes per day**

PART 2: TRANSPORTATION PHYSICAL ACTIVITY

These questions are about how you traveled from place to place, including to places like work, stores, movies, and so on.

8. During the **last 7 days**, on how many days did you **travel in a motor vehicle** like a train, bus, car, or tram?

_____ **days per week**

☐ No traveling in a motor vehicle → ***Skip to question 10***

9. How much time did you usually spend on one of those days **traveling** in a train, bus, car, tram, or other kind of motor vehicle?

_____ **hours per day**
_____ **minutes per day**

Now think only about the **bicycling** and **walking** you might have done to travel to and from work, to do errands, or to go from place to place.

10. During the **last 7 days**, on how many days did you **bicycle** for at least 10 minutes at a time to go **from place to place**?

_____ **days per week**

☐ No bicycling from place to place → ***Skip to question 12***

11. How much time did you usually spend on one of those days to **bicycle** from place to place?

_____ **hours per day**
_____ **minutes per day**

12. During the **last 7 days**, on how many days did you **walk** for at least 10 minutes at a time to go **from place to place**?

_____ **days per week**

☐ No walking from place to place → ***Skip to PART 3: HOUSEWORK, HOUSE MAINTENANCE, AND CARING FOR FAMILY***

13. How much time did you usually spend on one of those days **walking** from place to place?

_____ **hours per day**
_____ **minutes per day**

PART 3: HOUSEWORK, HOUSE MAINTENANCE, AND CARING FOR FAMILY

This section is about some of the physical activities you might have done in the **last 7 days** in and around your home, like housework, gardening, yard work, general maintenance work, and caring for your family.

14. Think about only those physical activities that you did for at least 10 minutes at a time. During the **last 7 days**, on how many days did you do **vigorous** physical activities like heavy lifting, chopping wood, shoveling snow, or digging **in the garden or yard**?

_____ **days per week**

☐ No vigorous activity in garden or yard → ***Skip to question 16***

15. How much time did you usually spend on one of those days doing **vigorous** physical activities in the garden or yard?

_____ **hours per day**
_____ **minutes per day**

16. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the **last 7 days**, on how many days did you do **moderate** activities like carrying light loads, sweeping, washing windows, and raking **in the garden or yard**?

_____ **days per week**

☐ No moderate activity in garden or yard → ***Skip to question 18***

17. How much time did you usually spend on one of those days doing **moderate** physical activities in the garden or yard?

_____ **hours per day**
_____ **minutes per day**

18. Once again, think about only those physical activities that you did for at least 10 minutes at a time. During the **last 7 days**, on how many days did you do **moderate** activities like carrying light loads, washing windows, scrubbing floors and sweeping **inside your home**?

_____ **days per week**

☐ No moderate activity inside home → ***Skip to PART 4: RECREATION, SPORT AND LEISURE-TIME PHYSICAL ACTIVITY***

19. How much time did you usually spend on one of those days doing **moderate** physical activities inside your home?

_____ **hours per day**
_____ **minutes per day**

PART 4: RECREATION, SPORT, AND LEISURE-TIME PHYSICAL ACTIVITY

This section is about all the physical activities that you did in the **last 7 days** solely for recreation, sport, exercise or leisure. Please do not include any activities you have already mentioned.

20. Not counting any walking you have already mentioned, during the **last 7 days**, on how many days did you **walk** for at least 10 minutes at a time **in your leisure time**?

_____ **days per week**

☐ No walking in leisure time → ***Skip to question 22***

21. How much time did you usually spend on one of those days **walking** in your leisure time?

_____ **hours per day**
_____ **minutes per day**

22. Think about only those physical activities that you did for at least 10 minutes at a time. During the **last 7 days**, on how many days did you do **vigorous** physical activities like aerobics, running, fast bicycling, or fast swimming **in your leisure time**?

_____ **days per week**

☐ No vigorous activity in leisure time → ***Skip to question 24***

23. How much time did you usually spend on one of those days doing **vigorous** physical activities in your leisure time?

_____ **hours per day**

_____ **minutes per day**

24. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the **last 7 days**, on how many days did you do **moderate** physical activities like bicycling at a regular pace, swimming at a regular pace, and doubles tennis **in your leisure time**?

_____ **days per week**

☐ No moderate activity in leisure time → ***Skip to PART 5: TIME SPENT SITTING***

25. How much time did you usually spend on one of those days doing **moderate** physical activities in your leisure time?
_____ **hours per day**
_____ **minutes per day**

PART 5: TIME SPENT SITTING

The last questions are about the time you spend sitting while at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading or sitting or lying down to watch television. Do not include any time spent sitting in a motor vehicle that you have already told me about.

26. During the **last 7 days**, how much time did you usually spend **sitting** on a **weekday**?

_____ **hours per day**
_____ **minutes per day**

27. During the **last 7 days**, how much time did you usually spend **sitting** on a **weekend day**?

_____ **hours per day**
_____ **minutes per day**

This is the end of the questionnaire, thank you for participating.