Title:

Heat Tolerance of Fire Service Instructors

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ABSTRACT

OBJECTIVES: Fire Service Instructors (FSI) experience repeated 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 aimed to establish whether FSI exhibit an increased heat tolerance and altered immunological response to heat exposures, compared to non-exposed individuals. The study also aimed 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 heat occupational tolerance test (HOTT) exercising at 6 $W.kg^{-1}$ (50.0 ± 1.0ºC, 12.3 ± 3.3% relative humidity) 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 rectal temperature (T_{re}) (-0.42ºC), change in T_{re} (-0.33ºC), and thermal sensation (-1.0) and increased sweat rate (+ 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 interleukin-6, interleukin-1β, and immunoglobulin G 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 symptoms.

Abbreviations:

BF	Body fat
BSA	Body surface area
CON	Control
CV	Coefficient of variation
FSI	Fire Service Instructor
HOTT	Heat occupational tolerance test
SR	Sweat rate

1. INTRODUCTION

Fire Service Instructors (FSI) are specially trained Fire Service personnel who deliver training courses to newly recruited and operational firefighters, to teach them key skills regarding fire behaviour, fire attack and use of breathing apparatus. Each training course typically involves multiple fire exposures in which FSI are responsible for both the learning and safety of their trainees. The tasks conducted by FSI can include leading or following a team inside a compartment/building through a training exercise or controlling the conditions of the exercise itself, which can involve being close to the fire to move ventilation doors and fuel sources. In the United Kingdom (UK) individuals can work permanently as a FSI, be seconded to a training centre for a specified number of years, or in some cases work as a part time FSI alongside their firefighting duties. Due to their occupation FSI experience fire exposures ~13 times a month in comparison to ~1 fire visited by firefighters (Watkins et al. 2018c). A combination of the environmental conditions, the heavy encapsulating protective clothing worn and the physical activity conducted during exposures, results in an uncompensable heat strain environment (Cheung et al. 2000). In these situations individuals experience elevation in core temperature (T_c) as their ability to dissipate heat is impaired (Cheung et al. 2000). Consequently, FSI frequently experience levels of high physiological strain and may also be at an increased risk of heat related illnesses (Eglin 2007; Petruzzello et al. 2009). An individual's ability to defend their T_c during physical activity in hot environments is referred to as their heat tolerance, with those who are more tolerant being at a reduced risk of heat illness (Kazman et al. 2013; Lisman et al. 2014). Previous research on firefighters indicates that there is no difference in their heat tolerance compared to the general population (Wright et al. 2013). However, modern fire services respond to a large variety of emergency calls with only a small proportion being fire incidents, in England 31% of all incidents attended in 2017/2018 were fires with 8% of all incidents being dwelling or building fires (Home Office, 2019). Consequently, a firefighter's role may not have a sufficient number of fire exposures to alter heat tolerance. In comparison, due to the elevated exposure levels experienced by FSI (Watkins et al. 2018c), the authors postulate that FSI may have an altered tolerance level.

Repeated heat exposures with sufficient thermal impulses can cause physiological adaptations that act to improve an individual's ability to cope and perform work or exercise 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 occurring through interaction with the environment (Sawka et al. 2011). Important markers of such adaptations during exercise are: reduced heart rate (HR) (-16 $b.min^{-1}$) and T_c (-0.2 - 0.34ºC), an increased sweat rate (SR) (+0.2 - 0.36 $L.hr^{-1}$) and improvement in performance (Sawka et al. 2011; Tyler et al. 2016). Acclimation protocols vary 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). Acclimation frequency can vary from one exposure per day, to twice daily, or intermittent exposure, although intervals between exposure of one week are too long to induce adaptations (Gill & Sleivert 2001; Willmott et al. 2016). To optimise adaptations 8 - 14 days of regular controlled hyperthermic heat exposures are recommended. Within 4 - 7 days of exposure up to 75% of adaptive changes can be measured (Pandolf 1998). Live fire exposures have been noted to increase core temp to 38.0 - 40.1°C (Smith et al. 1996; Eglin

& Tipton 2005), indicating individuals could experience close to or above the 38.5°C recommended for controlled hyperthermic acclimation.

Acclimation status typically decays in 2 - 4 weeks, although physical activity can help maintain adaptations for longer (Garrett et al. 2009; Périard et al. 2015). A recent review states that following heat acclimation lasting ≥ 5 days 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). Therefore whilst firefighters may not be able to maintain an acclimatised status based on their occupational exposures, maintenance may be feasible for FSI due to their greater number of fire exposures. There has not been any assessment of FSI heat tolerance to date, however heat acclimatisation has previously been noted in seasonal wildland firefighters, 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).

The immunological consequences of heat acclimation also need to be further understood, as increases in numerous parameters of inflammation, such as interleukin-6 (IL-6), white blood cell counts (WBC) and neutrophils (NEUT), are noted following acute fire exposures (Smith et al. 2005; Walker et al. 2015; Watkins et al. 2018b). Recent research indicates that heat acclimation has no impact on immune function, with no change in WBC, IL-6 or C-reactive protein (CRP) following 4 – 11 sessions of acclimation (Guy et al. 2016a; Willmott et al. 2016a; Costello et al. 2018). However, it has been suggested that improvements in heat tolerance may reduce inflammatory cytokine production during thermal stress (Kuennen et al. 2011). Currently, it is unknown what impact long term acclimation may have on an individual's immune function; no heat acclimation study longer than 11 exposure days has measured immunological markers. Repeated heat exposure, with minimal recovery, have been postulated to compromise individuals' immune systems and therefore be related to the occurrence of illnesses (Walsh et al. 2011). Supporting evidence for this concept is sparse currently (Walsh and Oliver 2016). Consequently, it is also of interest to investigate whether heat acclimatisation, gained through FSI occupational exposures, could be related to symptoms of ill health that have previously been noted in this population (Watkins et al. 2018c).

Markers of inflammation, such as IL-6, CRP and interleukin-1beta (IL-1β), are also predictors of cardiovascular events (Ridker et al. 2000; Ridker 2003; Ridker 2016). The main cause of death amongst firefighters is a cardiovascular event, accounting for 38-42% of fatalities in the USA over the past 10 years (Fahy et al. 2017). However, this data should be interpreted with caution when considering the UK Fire and Rescue Service, given the differences between countries in cardiovascular risk factors, such as the increased prevalence of obesity noted in USA firefighters compared to those in the UK (Munir et al. 2012). Despite this, firefighters have been reported to be 12 – 136 times more likely to experience a cardiovascular event following a fire than during nonemergency duties (Kales et al. 2007). Elevated cardiac troponin T (cTnT), a marker of myocardial ischemia, and increased platelet activity, mean platelet volume and platelet thrombus formation have all been noted after fire exposures (Hunter et al. 2017; Watkins et al. 2018b). However the persistent impact repeated fire exposure has on myocardial damage and predictors of cardiovascular events has yet to be investigated.

This study aimed to identify whether FSI develop a greater level of heat tolerance than individuals not exposed to repeated heat exposures and if so does chronic heat acclimatisation impact immune function and measures of myocardial damage. The study also aimed to establish whether heat tolerance markers are maintained by FSI over a 2 month period of their normal occupational schedule.

2. METHODS

2.1 Participants

Twenty-two participants were recruited for this study: 11 FSI and 11 non-exposed individuals as a control group (CON) (Table 1). CON were University lecturers 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). Of the FSI, 9 did not complete additional fire station duties at the time of the study, those who did were asked to report all fire exposures experienced regardless of role performed. Participants gave informed written consent and completed a medical questionnaire detailing any symptoms of ill health before each session. Participants were required to avoid caffeine and exhaustive exercise 12hr before each session, and alcohol 24h before. The medical questionnaire also checked that they were not taking any medications that may have altered their thermoregulatory response. The study was approved by the University of Brighton ethics committee.

2.2 Experimental Design

Participants completed two trials separated by a 2 month period, to capture the maintenance of any heat adaptations. Each trial consisted of completion of the International Physical Activity Questionnaire (IPAQ) (Craig et al. 2003), assessment of body composition, blood sample collection, and a heat occupational tolerance test (HOTT) (Watkins et al. 2018a). Both sessions were completed at the same time of day to control for circadian rhythms. Participants were required to attend the laboratories in a euhydrated state, as confirmed by a urine colour (U_{col}) of ≤ 3, osmolarity (U_{osm}) of < 700 mOsm.kgH_2O^{-1} (Pocket Pal-Osmo, Witech Scientific) and specific gravity (U_{spg}) of < 1.020 (hand refractometer, Atago Co., Japan) (Sawka et al. 2007).

2.2.1 Testing Procedures

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. Body composition was assessed via a Bod Pod (Cosmed, Italy) using air displacement plethysmograph. Measurements occurred post hydration assessment and prior to the 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 a heat chamber (50 ± 1.0ºC, 12.3 ± 3.3% RH) and performed a 40min walk test at 6 $W.kg^{-1}$ metabolic heat production whilst wea, as described by Watkins et al. (2018a).

2.2.2 Measures

Nude body mass was recorded pre and post heat exposure (Adam GFK 150 Body Scales, Connecticut, USA) for calculation of SR as per Equation 1. Clothed mass was also measured immediately pre HOTT to enable metabolic heat production to be calculated. A single use rectal temperature probe (449H, Henleys Medical, Hertfordshire, UK) was passed 10cm past the anal sphincter and displayed on logging monitors (YSI, 4600 series, Hampshire, UL) to measure rectal temperature (T_{re}). Contact skin thermistors were placed on the mid-belly of the pectoralis major, biceps brachii, rectus femoris and gastrocnemius and recorded via a 1000 series Squirrel Data Logger (Grant Instruments, Cambridgeshire, UK) to measure mean skin temperature (T_{skin}) as per Equation 2. A Polar FT1 HR monitor (Polar electro, Kempele, Finland) was positioned around the chest. HR, T_{re} and T_{skin} were recorded at the end of the rest period and every 5 min throughout the HOTT. Thermal sensation (TS), on a scale from 0 "unbearable cold" to 8 "unbearably hot" (Young et al. 1987) and rating of perceived exertion (RPE), on a scale of 6-20 (Borg 1982), were collected after resting and then every 10 min during exercise. The heat illness symptoms index (HISI) was also recorded pre and post heat exposure (Coris et al. 2006).

Equation 1.

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

Equation 2.

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

(Ramanathan, 1964).

2.2.3 Venous Blood Collection

Prior to and immediately post the HOTT venous blood samples (10 mL) were collected from the anti-cubital fossa. Samples were analysed for complete blood counts using an automated haematology analyser (XT2000i, Sysmex, UK) and then centrifuged at 4,500 rpm for 10min at 4°C to separate plasma, which was frozen at -86°C for subsequent analysis. ELISA analysis was conducted for IL-6, TNF-a, IL1-β, CRP and IgG (R&D Systems, Minneapolis, USA). Plasma samples were also analysed for cTnT using an electrochemiluminescence assay (Roche Modular E170 (fifth generation); Basel, Switzerland). This had a limit of blank of 3 $ng.L^{-1}$ and CV at the upper reference limit of 14 $ng.L^{-1}$ of <8% (Westermann et al. 2017).

2.3 Statistical Analysis

Data were analysed using IBM SPSS Statistics 22. Data were assessed for normality and sphericity. Due to previously reported differences at baseline in FSI compared to a control group (Watt et al. 2016) haematological

markers were analysed for baseline differences in Trial 1 by independent samples t-tests or Mann Whitney U tests.

Normally distributed data were analysed by a two way mixed method ANOVA 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 (Trial 1 vs. Trial 2) 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. Mann Whitney U tests were conducted to identify differences in absolute change between groups (FSI vs CON). A Wilcoxon signed rank test was then conducted to establish if change altered between trials (Trial 1 vs Trial 2). Significance was set at $p < 0.05$. Effect sizes are presented as partial eta squared for ANOVA effects and Cohen's d_s or d_z for differences between two groups of data, conducted and interpreted in accordance with Cohen (1988) and Lakens (2013).

Pearson or Spearman correlation analysis were conducted between end HOTT T_{re} and possible related factors such as: body fat (BF) %, exposure number and age. Correlation analysis was also conducted between haematological variables and end HOTT T_{re}. Point biserial or rank biserial correlations were conducted between FSI reported presence of ill health and end HOTT T_{re} and haematological variables.

3. RESULTS

3.1 Trial 1 Baseline Characteristics

All participants met the euhydration criteria, with no differences in U_{col}, U_{osm}, or U_{spg} between FSI and CON in Trial 1 ($p > 0.05$). All participants were classified with a high level of physical activity based on their IPAQ scores. However, FSI reported a greater amount of METmins.week^{-1} than CON ($p = 0.019$) (Table 1), with FSI completing a greater number of METmins.week^{-1} from their work compared to CON (5699 ± 3570 METmins.week^{-1} vs 792 ± 1862 METmins.week^{-1}) ($p = 0.001$). There was no difference in BF%, body surface area (BSA), or BSA/mass ratio between groups ($p > 0.05$) (Table 1).

At Trial 1 symptoms of ill health were reported by 6/11 FSI participants and 1/11 CON participants. All six FSI reported cold symptoms, including blocked noses and coughs, two of the six participants reported night sweats, and one 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.

Table 1. *Demographic details for FSI and CON at their initial trial and 2 months later at 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 ($p > 0.05$).*

	Trial 1		**Trial 2**	
	FSI	CON	FSI	CON
Number of Males	9	9		
Number of Females	2	2		
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 ($MET.min.week^{-1}$)	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.

3.2 Trial 1 Heat Tolerance

Table 2 displays the resting and end of HOTT values for all physiological and perceptual variables. Two way ANOVA analysis revealed a difference in T_{re} between the FSI and CON across the HOTT test ($p = 0.013$, $\eta_p^2 = 0.216$). Follow up tests revealed no difference in T_{re} at rest or 20 min ($p > 0.05$), but a difference was present at 40 min ($p = 0.013$, $d_s = 1.16$) with T_{re} being greater in the CON group compared to the FSI group. Change in T_{re} (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 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 1 for T_{re} at 40 min for all participants along a continuum, as recommended by Watkins et al. (2018a). 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.

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 $L.hr^{-1}$) ($p = 0.015$, $d_s = 1.40$). Both HR and ΔHR increased throughout the HOTT ($p < 0.001$, $\eta_p^2 = 0.960$ and $p < 0.001$, $\eta_p^2 = 0.951$, respectively) but increases were not affected by group ($p > 0.05$). PSI and T_{skin} also both did not differ at any time between the groups ($p > 0.05$).

3.2.1 Perceptual Responses

Both RPE and HISI increased with the HOTT ($p < 0.001$, $\eta_p^2 = 0.893$ and $p < 0.001$, $\eta_p^2 = 0.711$, respectively),

but this increase was similar between groups ($p > 0.05$). However, the increase in TS that occurred throughout the HOTT ($p < 0.001$, $\eta_p^2 = 0.906$) was different between the groups ($p = 0.048$, $\eta_p^2 = 0.111$), with TS at 40 min being greater in CON than FSI ($p = 0.010$, $d_s = 2.00$).

3.2.2 Haematological Responses

Table 3 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 or post time points ($p > 0.05$).

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.

3.3 Maintenance of Heat Tolerance

Hydration status was the same for pre and post 2 month trials, with no differences in U_{col}, U_{osm} or U_{spg} ($p > 0.05$) (Table 1). There was also no difference in IPAQ scores or BF% between the trials ($p > 0.05$). The number of exposures 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, perceptual and haematological variables exhibited the same response between trials, with no differences between trials at any time point ($p > 0.05$) (Table 2 & 3).

3.4 Correlations

Analysis of all participants' data revealed T_{re} at 40 min was correlated with the number of exposures completed in the previous 2 weeks ($r_s = -0.589$, $p = 0.004$), see Figure 2. Age, IPAQ score, SR, BSA, and BSA/mass ratio 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 exposures ($r_{rb} = 0.543$, $p = 0.009$). Of those reporting symptoms of ill health, 4/6 FSI were classified in the "green zone" and 2/6 were in the "yellow zone" based on the continuum interpretation of T_{re}. The single individual who reported adult onset asthma in the CON group was classified in the "yellow zone".

Table 2. *Physiological and perceptual variables at rest and end of exposure for Trial 1 and 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 ($p > 0.05$).*

	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 3. *Haematological variables at rest and post heat exposure 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 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 ($10^9.L^{-1}$)	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 ($10^9.L^{-1}$)	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 ($10^9.L^{-1}$)	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
LYMPH ($10^9.L^{-1}$)	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
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,, LYMPH = lymphocyte, 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

4. DISCUSSION

This study demonstrates that the 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. However, there was no difference in FSI and CON immunological responses to a heat exposure. Alternatively, FSI did exhibit greater baseline levels of IL-1β, IL-6, and IgG. Physiological, perceptual, and haematological responses were the same following a 2 month working period, indicating that FSI maintain their heat acclimatised status, even with fluctuations in heat exposure numbers.

4.1 Acclimation Status

A meta-analysis of heat acclimation studies show T_c changes of 0.18 ± 0.14ºC at rest and 0.34 ± 0.24ºC at comparable time points following 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. 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 in this study, which may be a consequence of the between subjects design of this investigation and large HR standard deviations 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 due to females' lower sudomotor thermosensitivity (Gagnon et al. 2013). However, analysis of male SR revealed that FSI had a 0.25 $L.hr^{-1}$ greater SR, similar to the 0.20 - 0.36 $L.hr^{-1}$ increase previously reported following acclimation (Patterson et al. 2004; Weller et al. 2007; Gibson et al. 2015; Willmott et al. 2016). 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 is impaired by PPE. Participants in the FSI group also exhibited a 1.0 improvement in TS, which is similar to the 0.2 – 0.8 reduction in TS previously noted with heat acclimation (Gibson et al. 2015; Tyler et al. 2016). TS may be an instigator of behavioural thermoregulation, with a reduced warmth sensation resulting in the attenuation of decreasing work rates that occur when exercising in uncompensable environments (Flouris and Schlader 2015). Overall, T_{re}, SR, and TS responses suggest that FSI exhibit an acclimatised profile in comparison to non-exposed individuals.

The acclimatised profile exhibited by FSI may have been a consequence of exposure number, with end T_{re} being negatively correlated with the number of fire exposures in the previous 2 weeks. This finding indicates that those with a greater number of exposures demonstrate a more acclimatised profile. However, not all FSI had a lower peak T_{re} than those in the CON group, with one FSI being classified as in the "red zone" on the peak T_{re} continuum. This individual had the highest BF% of all participants (32%), classifying them as overweight (> 25%) (Gallagher et al. 2000; De Lorenzo et al. 2003). Adipose tissue has a lower heat capacity in comparison to lean tissue (2.97 $J.g^{-1}.°C^{-1}$ vs 3.66 $J.g^{-1}.°C^{-1}$) and therefore those with a higher BF% have a reduced heat storage capacity, which can cause an increased rate of rise in T_c (Selkirk and Mclellan 2001; Dervis et al. 2015). To improve heat tolerance without additional heat exposures FSI should ensure they have a healthy body composition. It is possible that aerobic fitness is also correlated to

heat tolerance (Selkirk and Mclellan 2001) and although IPAQ classifications were similar between groups, FSI completed a greater amount of MET.min^{-1}.week^{-1}. However, the matching of body composition across groups and the implementation of a metabolic heat production controlled test may have attenuated this effect (Cramer & Jay 2014). There was also no correlation between IPAQ MET.min^{-1}.week^{-1} and peak T_{re}.

All participants maintained their level of heat tolerance over the 2 month period, as demonstrated by the similar responses in all physiological and perceptual variables. Previous research suggests that 1 – 2 months after adaptation to the heat is obtained only 2 - 4 days of exposure are needed to re-acclimate (Weller et al. 2007; Horowitz 2016). In addition, following heat acclimation a heat exposure every 5 days sustains adaptations and reduces the risk of exertional heat illness for up to a month after acclimation (Daanen et al. 2018; Pryor et al. 2018). Consequently, the long term career of experiencing repeated bouts of exposures may explain the acclimatised state demonstrated by FSI. However, it is important to note that extended periods away from exposures, such as when on annual leave, could reduce a FSI heat tolerance.

4.2 Haematological Response

Whilst physiological and perceptual variables indicate an acclimatised state in FSI, the change in haematological variables following the HOTT was similar between groups. Increases in WBC (+27.8%), NEUT (+34.2%), LYMPH (+30.9%), PLT (+15.8%) IL-6 (+55.0%), and cTnT (+20%) were present following the HOTT, alongside a reduction in CRP (-10.9%). This is in line with haematological responses previously reported following live fires (Smith et al. 2005; Walker et al. 2015; Watt et al. 2016; Watkins et al. 2018b). Furthermore, the similar haematological responses between FSI and CON replicates previous findings that 4 - 11 days of repeated heat exposure result in unaltered IL-6 (Kanikowska et al. 2012; Barberio et al. 2015; Guy et al. 2016b; Costello et al. 2018), WBC (Kanikowska et al. 2012; Willmott et al. 2016), and CRP (Costello et al. 2018). This study offers the first assessment of the inflammatory response to chronic heat exposures.

4.2.1 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), IL-1β was 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) and IL-6 (2.18 ± 2.39 pg.mL^{-1}) was in the upper quartiles of healthy individuals (≥ 1.47 pg.mL^{-1}) (Ridker et al. 2000). Consecutive heat exposure has been suggested to lead to systemic inflammation, with elevated resting IL-6 exhibited in FSI following a 4 week instruction course (11.4 ± 1.0 pg.mL^{-1}) (Watt et al. 2016). Combined stressors, such as sleep deprivation, high intensity physical exertion, and energy deficit, experienced with a 3 week military training and combat course, have also been noted to lead 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. In situations of chronic disease and acute tissue injury it can exacerbate damage (Dinarello 2011), 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 in mice, has been noted to increase IL-1β (Lira et al. 2010; Pereira et al. 2015). Symptoms of overtraining have also been reported to show distinct familiarity with symptoms of "sickness behaviour", such as weakness, malaise, fatigue and inability to concentrate, of which IL-1β plays a key role in instigating (Smith 2000; Dantzer 2001; Konsman et al. 2002). In addition, IL-1β is involved in the atherothrombotic process, as it stimulates adhesion molecules and procoagulant activity (Ridker et al. 2011). Consequently, a chronic elevation in IL-1β, indicating systemic inflammation, may suggest an increased risk of cardiovascular events and symptoms of an overtraining like response in FSI.

Elevated IL-6 is also related to the occurrence of atherosclerotic events (Ridker et al. 2000; Rauchhaus et al. 2000; Spoto et al. 2014). Individuals in the upper quartile of IL-6 levels (> 2.28 $pg.mL^{-1}$) have 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 for each quartile increase (Ridker et al. 2000). 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, overtraining has been linked to IL-6, with increased IL-6 positively related to increased sleep disturbances, fatigue, and mood depression, consistent with sickness behaviour symptoms (Smith 2000; Main et al. 2010). The elevated IL-6 and IL1-β exhibited by FSI alongside the increased occurrence of symptoms of ill health, suggests that an overtraining like response may occur.

Increased levels of IgG are also an indicator of chronic inflammation or 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 3 - 4 months of training have previously been reported to remain within the normal range (898 - 1040 $mg.dL^{-1}$) (Gleeson et al. 2000; Córdova et al. 2010). However greater values 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). In comparison, the IgG levels exhibited by FSI are markedly greater than previous training studies indicate, consequently a larger sample of the population should be assessed to identify if this occurrence is universal to the population, as elevation of IgG could indicate the presence of infection and inflammation in FSI.

4.3 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 exposures 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 FSI in the "yellow and green zones" of the continuum did not report symptoms of ill health. These individuals had an average of 3 ± 2 exposures in the previous 2 weeks, compared to those who reported symptoms who had 5 ± 3 exposures. The establishment of an appropriate exposure limit could enable FSI to maintain an acclimatised status, whilst reducing the risk of symptoms of ill health occurring.

4.4 Limitations

Participants maximal volume of oxygen uptake 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. Symptoms of ill health were subjectively reported and not diagnosed by a medical professional. Knowledge of the actual thermal load experienced during 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 exposure numbers and markers of inflammation and ill health.

5. 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. FSI display a similar immunological response to heat exposures as non-exposed individuals, although they do present with heightened levels of haematological markers, namely IL-6, IL-1β, and IgG. This suggests FSI may experience chronic inflammation, which has previously been linked to risk of cardiovascular events, sickness behaviour and overtraining, although a greater data set is needed to support this. 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 an exposure number limit, to enable heat acclimatisation to be developed and maintained, whilst reducing the risk of ill health.

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LIST OF FIGURES

VITAE

Miss Emily Watkins completed her BSc (Hons) degree in Sport and Exercise Science in 2014 at the University of Brighton. She began her PhD at the University in October 2014, working with Fire Service Instructors to investigate their heat tolerance and immunological responses to frequent heat exposures. Emily is also a Technical Instructor for the Sport and Exercise Science degree.

Dr. Mark Hayes joined the University of Brighton in 2011 as a lecturer in sport and exercise science. Before this Mark taught in Further Education having studied Fine Chemicals, Pharmaceuticals and Microbiology in Ireland (1992–1995) followed by a BSc (Hons) in Sport and Exercise Science at the University of Brighton (1995-1998). On joining the University Mark completed his PhD, examining the efficacy of progressive heat acclimation for intermittent-sprint exercise in the heat. Mark teaches exercise and environmental physiology with consideration of performance, occupational and health aspects. In 2013 he was awarded a University of Brighton's Excellence in Facilitating and Empowering Learning Award.

Dr. Peter Watt has published over 100 peer reviewed articles, 100 abstracts and acts as a reviewer and editor for several international journals. Dr. Watt's research focuses on measuring metabolic and physiological changes occurring in humans during exercise, with application to health related problems, e.g. diabetes, obesity. Dr. Watt has supervised 16 Ph.D. and 1 M.Phil. project and is an Associate Professor.

Dr. Alan Richardson is a Principal Lecturer in Exercise and Environmental Physiology at the University of Brighton. His research focuses on human tolerance to hypoxia and severe heat exposure. Dr. Richardson worked as a research exercise physiologist at the Centre for Aviation, Space and Extreme Environment Medicine Xtreme Everest Project in 2007 and 2009. Since then Dr. Richardson has led research projects with the National Fire Service investigating the health and thermal loading of fire fighters and instructors.