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Edited by
R.P. Thornton & L.J. Wright

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Welcome from Editors

It is our pleasure to bring to you the compiled papers from the Research Forum of the AFAC and Bushfire CRC Annual Conference, held in the Perth Exhibition and Convention Centre on the 28th of August 2012.

These papers were anonymously referred. We would like to express our gratitude to all the referees who agreed to take on this task diligently. We would also like to extend our gratitude to all those involved in the organising, and conducting of the Research Forum.

The range of papers spans many different disciplines, and really reflects the breadth of the work being undertaken, The Research Forum focuses on the delivery of research findings for emergency management personnel who need to use this knowledge for their daily work.

Not all papers presented are included in these proceedings as some authors opted to not supply full papers. However these proceedings cover the broad spectrum of work shared during this important event.

The full presentations from the Research Forum and the posters from the Bushfire CRC are available on the Bushfire CRC website www.bushfirecrc.com.

Richard Thornton and Lyndsey Wright

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The content of the papers are entirely the views of the authors and do not necessarily reflect the views of the Bushfire CRC or AFAC, their Boards or partners.

The Stress of Firefighting: Implications for Long-term Health Outcomes

*Luana Main, Jenni Raines, Paul Della Gatta, Alex Wolkow, Rod Snow, & Brad Aisbett
School of Exercise and Nutrition Sciences, Deakin University*

Abstract

Fire and rescue staff routinely endure significant psychological and environmental stress exposure on the job. While much has been done to improve understanding of the physiological effects of exposure to these conditions, little has been done to quantify the inflammatory stress response that firefighters are exposed to during wildfire suppression. Therefore the aim of the present study was to explore whether firefighters experienced a change in inflammatory markers following one day, and across two days of wildfire suppression tasks. Twelve male fire-fighters participated in two consecutive days of live-fire prescribed burn operations in Ngarkat National Park, South Australia. Typical work tasks included lighting burns, patrolling containment lines, suppressing spot fires, and operating vehicles. A number of the inflammatory markers changed significantly across the course of a shift and several presented with an attenuated response across the second day. This finding implies that there was a compounding effect of repeated exposure to these stressors which could have considerable implications for managing fire-fighters health and wellbeing over a multi-day campaign. Further research is required to see which fire ground stressor, or combination of stressors is causing these changes in the inflammatory markers across consecutive work shifts.

Introduction

Each year thousands of volunteer and career staff from fire and rescue emergency services routinely endure significant psychological and environmental stress exposure on the job to ensure public safety. While much has been done to improve our understanding of the physiological effects of exposure to these conditions (e.g. Heart rate, core temperature: Aisbett *et al.* 2012), little is known about the implications of repeated exposure to these stressors on firefighter health. Across each shift, firefighters are required to perform intermittent, intense physical labour (Cuddly *et al.* 2007; Aisbett *et al.* 2007), often in hot (Black 1987; Hancock *et al.* 2007), and smoky (Reisen and Brown 2009) conditions. Other environmental stressors may also include exposure to dust and pollutants (Carlisle and Sharp 2001), toxic chemicals (Miranda *et al.* 2010), and excessive noise (Pepe *et al.* 1985). In addition, firefighters may also work for extended periods of time, across both day and night shifts, and often with little rest between consecutive shifts (Cater *et al.* 2007). In isolation, these factors such as heat, smoke (or its constituent elements), and sleep disruption may have a detrimental impact on cognitive and physical work capacity (Carlisle and Sharp 2001; Hancock *et al.* 2007; Lim and Dinges 2010; Nybo 2008). However the impact of these stressors in combination, and effect of repeated exposure to these stressors on the (long-term) health of personnel has yet to be determined.

Just as firefighters work to protect people and property, fire agencies strive to keep their personnel safe and healthy. For example the use of personal protective clothing (PPC) and personal protective equipment (PPE) serve to physically protect personnel from some of the hazards of wildfire suppression. Similarly the implementation of work to rest guidelines have been designed to protect firefighters against injury and even death resulting from fatigue related errors. However while many of the workplace stressors these personnel face have been documented; far less evidence is available on firefighter's inflammatory response to these stressors. Among the scientific community there is emerging evidence supporting the principle that psychologically (and physiologically) stressful events lasting anywhere from minutes to years are associated with changes in the immune system (Sergeyev & Miller, 2004), for which inflammatory cytokines such as Interleukin (IL)-1 β , IL-6, and Tumor Necrosis Factor (TNF)- α , appear to be the major messenger molecules in this network. Activation of these immune cells produces an array of physiological, behavioural, affective, and cognitive changes in response to stress exposure. For example, a decrease in mood state, influencing the desire to withdraw from social engagement, and increased perceptions of fatigue, which it is reasoned have evolved to promote recovery following exposure to a stressor (Maier & Watkins, 1998).

This acceptance that stress can influence and manipulate the release of circulating inflammatory markers has led to the suggestion that chronic exposure to these stressors may actually have a long term negative impact on individual's health and well-being (Gouin, 2011; Maier & Watkins, 1998). For example, by initiating changes in the hypothalamic-pituitary-adrenal axis and the immune system, chronic stress has been cited as a trigger for depression, cardiovascular deregulation, and diseases such as cardiovascular disease (Grippio & Johnson, 2009; Leonard, 2010). Disturbances to inflammatory markers have been observed following a 90-minute live-fire simulation of structural firefighting (Smith, 2005) and

after 37-minute of cycling ergometer exercise whilst performing firefighting tactical decision making (Huang *et al.* 2010; Webb *et al.* 2011). Extrapolating the findings from these environments to the immunological changes observed after a 10-12 hour day of wildfire suppression is problematic due to the differences in work duration, work to rest profiles, and environmental conditions. Further, the available literature provides no insight into the possible cumulative effect of a second day on wildfire suppression on firefighters' immune response. This information is particularly useful given the prevalence of multi-day deployments for Australian rural firefighters. Therefore the aims of the current study were to: 1) investigate whether an inflammatory response was mounted following a day of wildfire suppression tasks; and 2) investigate the effect that a second day of wildfire suppression tasks has on the same inflammatory markers.

Methods

Participants

Twelve male fire-fighters (age 29 ± 11 yr) participated in two consecutive days of live-fire prescribed burn operations in Ngarkat National Park, South Australia. Typical work tasks included lighting burns, patrolling containment lines, suppressing spot fires, and operating vehicles. Standard, fire-retardant PPC designed to shield the firefighter from environmental hazards and injury was worn throughout the shift as per fire agency guidelines. Approval for the project was obtained from the University Ethics Committee for human research. All participants received written and verbal explanation of the study informing them of the risks and benefits associated with participation. Written informed consent then was obtained from the participants prior to commencement of the study.

Testing Procedures

Upon arrival to the prescribed burn site pre-shift (0 h), participants were asked to sit for two minutes prior to providing a 10 mL venous blood sample from an antecubital vein. Following data collection, the firefighters assembled for their daily briefing and then commenced firefighting work. The end-shift data (12 h) were obtained at the on-site location, while the two hours post-shift data were obtained upon their return to the staging area (14 h). During hours 12 to 14, firefighters drove from the fire-ground to the staging area, showered, ate and rehydrated freely. This testing procedure was replicated for both days.

Blood sampling and analysis

Blood samples were obtained from each firefighter at 0hr, 12 h and 14 h. Following each testing session, all samples were centrifuged at $10,000 \text{ rev}\cdot\text{min}^{-1}$ for four minutes at room temperature, and then the plasma was separated into aliquots. These samples were stored at -20°C for up to three days whilst out in the field before being transferred into a -80°C freezer. Upon completion of the study, a selection of cytokines (IL-1 β , IL-2, IL-4, IL-5, IL-6, IL-7, IL-8, IL-10, IL-12p70, IL-13, IFN γ , GM-CSF, & TNF α) were simultaneously quantified using a High Sensitivity Cytometric Bead Array Human Inflammation Assay kit (CBA Kit: #HSCYTO-60SK). These assay kits provide a mixture of 13 micro-bead populations with distinct fluorescent intensities and were pre-coated with capture antibodies specific for each cytokine. All had inter and intra-assay variability's of $<10\%$. Analysis of the cytokines were

done according to the instructions of the manufactures of the Bioplex 200 array reader (V.5.0, Bio-Rad Laboratories, Hercules, CA).

Statistical Analysis

Due to the relatively small sample size, a Linear Mixed Modelling (LMM) approach with the SPSS Mixed Procedure (IBM SPSS Statistics for Windows, Version 20.0. Armonk, NY: IBM Corp.) was used for the analysis. Specifically, multilevel models were used to measure the effect of 'time of shift' and 'day of shift' on both mood disturbance and each of the inflammatory cytokines measured. The method of estimation used was restricted maximum likelihood, as this method is less biased than the full maximum likelihood with small sample sizes (Peugh & Enders, 2005). To ensure homogeneity of the data, log transformations were performed where necessary prior to statistical analysis. All analyses were performed using SPSS and $p < 0.05$ was used to indicate statistical significance.

Results

Within-days: Results indicated that across the course of a shift, there were significant changes in IL-1 β , IL-5, IL-7, IL-10, and TNF α ($p < 0.01$). Both IL-1 β and IL-7 increased over the 12h of controlled burn tasks (average increase 44% and 21% respectively). Following the end of the shift there was a general return towards baseline (0 h) measures, however only the 53% drop in IL-1 β between 12 h and 14 h was significant.

In comparison, IL-5, IL-10 and TNF α all significantly decreased over the 12 h of controlled burn tasks (average decreases of 23%, 40%, and 12% respectively). While there appeared to be some return towards 0 h levels all 14h measures for IL-5, IL-10 and TNF α were still significantly lower than 0 h levels ($p < 0.01$).

Between-days: There was a significant effect of performing repeated shifts on a number of the inflammatory cytokines. More specifically, IL-1 β ($p = 0.005$), IL-7 ($p = 0.004$), IL-4 ($p = 0.048$), IL-6 ($p = 0.036$), IL-8 ($p = 0.045$), and IL-13 ($p = 0.050$) all presented with an attenuated response across the course of the second day. The mean changes for the inflammatory cytokines within and between the two consecutive days of wildfire suppression are presented in Table 1.

	Day 1			Day 2		
pg/ μ l	0h	12h	14h	0h	12h	14h
IL-1 β	0.17 (0.06)	0.32 (0.07)	0.20 (0.06)	0.16 (0.07)	0.16 (0.07)	0.05 (0.04)
IL-4	2.98 (0.66)	3.27 (0.98)	2.70 (0.62)	2.39 (0.56)	2.78 (0.76)	2.26 (0.79)
IL-5	0.27 (0.11)	0.24 (0.09)	0.26 (0.09)	0.34 (0.11)	0.22 (0.09)	0.25 (0.12)
IL-6	2.84 (0.89)	5.57 (1.34)	3.86 (1.17)	2.67 (0.67)	2.28 (0.52)	3.70 (1.99)
IL-7	0.43 (0.06)	0.59 (0.06)	0.40 (0.08)	0.37 (0.06)	0.39 (0.09)	0.28 (0.07)
IL-8	2.88 (0.78)	2.83 (0.39)	2.66 (0.35)	2.19 (0.34)	1.97 (0.34)	2.61 (0.51)
IL-10	11.99 (3.77)	8.49 (1.91)	8.41 (2.24)	15.82 (6.06)	7.88 (2.07)	9.87 (2.64)
IL-13	0.88 (0.15)	1.03 (0.18)	0.96 (0.15)	0.91 (0.14)	0.75 (0.14)	0.69 (0.23)
TNF α	5.39 (0.71)	5.22 (0.55)	4.87 (0.55)	5.83 (0.68)	4.58 (0.81)	4.32 (0.81)

Table 1: Mean (\pm SE) values for those inflammatory cytokines which responded significantly to consecutive days of wildfire suppression.

Only one cytokine, IL-6, presented with a significant interaction effect for *time* and *day of shift* ($p=0.037$). On day 1 IL-6 increased over the 12h of controlled burn tasks (average increase of 96%), which was followed by a marked decrease between 12h and 14h (31%). However on the second day, IL-6 appeared to decrease (-15%) over the 12h of controlled burn tasks, before returning towards day one 14h levels at the end of day two (14h; Figure 1).

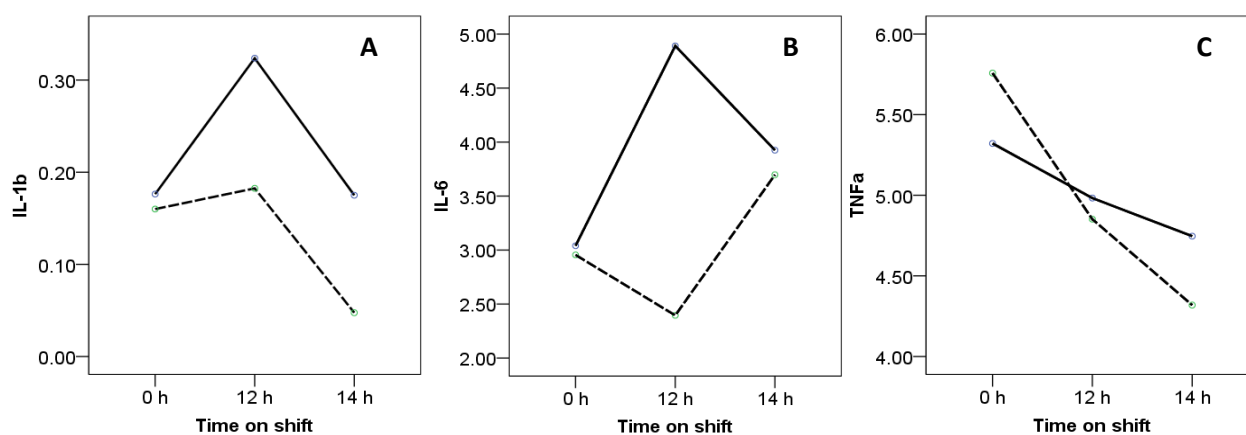


Figure 1: Mean changes in IL-1 β , IL-6 and TNF α over the course of each shift.

Note: Day one is indicated by the solid line, and day two the dashed line for IL1 β (A), IL-6 (B) and TNF α (C) respectively.

Discussion

While much has been done to improve our understanding of the physiological responses to physical work and exposure environmental stressors (e.g. Huang *et al.* 2010; Smith, 2005; Webb *et al.*, 2011); the impact of these stressors in combination, and effect of repeated exposure to these stressors on circulating inflammatory factors has yet to be determined. Therefore the main objective of the current study was to investigate whether an inflammatory response was mounted following a day of wildfire suppression tasks; and investigate the effect of a repeated day of wildfire suppression tasks on the same inflammatory markers.

The three primary cytokines that have been associated with the acute-phase inflammatory response (to exercise) are IL-6, IL-1 β and TNF α (Smith 2004). In the current study, IL-6 increased over the course of a single day of wildfire suppression. Increases in IL-6 concentrations have been implicated with behavioural changes during periods of both physiological and psychological stress (Owen and Steptoe 2003; Pedersen and Febbraio 2005). More specifically, in both work and exercise settings (Lancaster, 2006; Nehlsen-Cannarella *et al.* 1997) elevations in IL-6 levels, as much as five times greater than baseline measures, have been observed (Brenner *et al.* 1999; Yang *et al.* 2011). Therefore while acknowledging the dearth of literature specifically in the emergency services context; the observed changes in IL-6 following a single 12h shift of wildfire suppression tasks in the current study are consistent with previous exercise based research, supporting the premise that wildfire suppression is physiologically stressful task.

Similarly, the observed elevations of IL- β and IL-7 following 12 h of wildfire suppression tasks in the current study provides further support for the hypothesised activation of the stress system to adjust homeostasis and increase chances for survival (Chrousos and Gold, 1992). IL-1B is an important mediator of the inflammatory response, while IL-7 has a key role in modulating immune responses (Fry and Mackall, 2002). Although TNF α was not significantly elevated, and in fact decreases were observed in the current study, it remains one of the more problematic cytokines to measure and as such further research is required to substantiate this finding.

Interleukin-5 and IL-10 were the other two cytokines which significantly decreased following a single 12 h shift of wildfire suppression tasks. While the effects of exercise on inhibitory cytokine IL-10 have not yet been widely reported, this anti-inflammatory cytokine is known to increase markedly in the circulation following exercise (Suzuki *et al.* 2000a). It has been hypothesized that its release could limit the release of the pro-inflammatory cytokines IL-1 β and TNF α after exercise (Suzuki *et al.* 2000b). In the current study IL-10 concentrations decreased on average 40% over the course of the 12-hour shift. However notably, there was a marked increase during the 2-hour recovery phase on day two which may be indicative of an up regulation of this cytokine to mediate the inflammatory response to the repeated day of wildfire suppression tasks. The exact role that IL-5 plays is unclear; however it has been identified as a key player in the coordination and orchestration of white-blood cells in inflammatory processes (e.g. Hirai *et al.* 1997).

Despite the intermittent and repetitive physical nature of emergency services work, no study to date has investigated the inflammatory or hormonal response to repeated days of physical work. Findings from the current study suggest that a number of inflammatory markers were significantly altered on day two of their suppression work. This suggests one of two things: One, it may imply that there was an adaptive or conditioning response to the work on the second day, such that it was less stressful. The alternative interpretation of the results suggests a compounding effect of repeated exposure to these stressors may have occurred, which could have considerable implications for managing fire-fighters health and wellbeing over a multi-day campaign. The required duration of rest between repeated bouts to prevent carryover effects from a first bout it is as yet unknown, although research by Degerstrøm and Østerud (2007) suggests that a 4 h rest period is insufficient.

Conclusion

Collectively the findings of the current study suggest that there was an inflammatory response to a 12 h shift of wildfire suppression; and that this response was altered following a second day of work. It must be acknowledged, that while a number of the inflammatory markers were significantly altered across the course of a shift, there was a high degree of variability between participants. With such a small sample size it is hard to account for this variance, however it is possible that some participants may have already had an attenuated resistance to the environmental stressors. Nevertheless, the findings of the present study are unique, and represent a novel contribution to our understanding of the impact of wildfire suppression on the acute-phase stress response and circulating inflammatory markers. Further research is required to see which fire ground stressor, or combination of stressors is causing the rise in firefighters' inflammation across consecutive work shifts. Similarly, we don't know how these inflammatory markers change in emergency situations. Moreover, given chronic exposure to these stressors may actually have a long-term negative impact on individual's health and well-being, we need to improve our understanding of how an individual's inflammatory response to a stressor is correlated with long term health outcomes.

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