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Stress hormones and vascular function in firefighters during concurrent challenges

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ABSTRACT

The purpose of this study was to examine the effects of concurrent physical and mental challenge on stress hormones and indicators of vascular function in firefighters. Twelve professional firefighters exercised at 60% VO_{2max} while participating in a computerized Fire Strategies and Tactics Drill (FSTD—fire strategies condition [FSC]), and again at the same intensity without the mental challenge (EAC). No differences in the amount of work performed between conditions existed, although the FSC resulted in greater perceptions of overall workload. Epinephrine and norepinephrine demonstrated significant interaction effects with elevated levels during the FSC. Cortisol responses were significantly elevated across time and for the FSC. Positive correlations were found between cortisol and interleukin-6, endothelin-1, and thromboxane-B_2, and a negative correlation between interleukin-6 and thromboxane-B_2. These results suggest that concurrent challenges results in exacerbated responses of stress hormones and suggests mechanisms that could contribute to the prevalence of cardiovascular events among firefighters.

1. Introduction

Firefighters have one of the highest rates of on-the-job heart attack deaths among all occupations (Kales et al., 2007) and heart disease is the primary cause of line-of-duty deaths (LODD) in firefighters (Kales et al., 2007; Soteriades et al., 2002). Multiple studies have shown that firefighters have increased mortality ratios of ischemic heart disease compared to other cohort populations (Baris et al., 2001; Calvert et al., 1999) and statistics obtained from the United States Fire Administration demonstrate that for the previous 20 years, approximately 50% of these LODD are the result of "stress/exertion" (e.g., myocardial infarctions, cerebrovascular accidents). Further, the National Fire Protection Association (NFPA) reports an average of over 300 cardiovascular related injuries per year among firefighters (Karter and Molis, 2009).

Although the increased risk of cardiovascular disease (CVD) among firefighters is appreciable (Choi, 2000; Kales et al., 2007), an explanation for the elevated LODD and non-fatal CVD events is absent. One possible occupational "hazard" that may help to explain the increased risk of CVD in firefighters is the amount of stress associated with fighting fires (Kales et al., 2007). Firefighters are often subjected to combinations of physical and psychological challenges (i.e., coping with physical dangers, mental stress, physiological exertion, heat, etc.) in the course of fulfilling occupational responsibilities (Beaton et al., 1998, 1999). Kales et al. (2007, 2003) compared morbidities resulting from cardiac arrests in fire suppression tasks, alarm responses, and non-emergency duties, and found a significantly higher relative risk of CVD death for the fire suppression tasks and alarm responses. Numerous unique environmental factors associated with fighting fires likely contribute to these findings (Kales et al., 2007).

Brief periods of laboratory induced mental and physical stress independently elicit the release of various hormones from the sympathoadrenal (SA) and hypothalamic–pituitary–adrenal (HPA) axes as well as inflammatory cytokines, endothelial adhesion molecules, and indicators of platelet activity. These acute stressors have also been shown to trigger CVD events (Chowdhury et al., 2003; Kales et al., 2007; Wilbert-Lampen et al., 2008). Further, each of these physiological factors is related to the development of atherosclerosis and acute coronary syndromes (Charmandari et al., 2005; Gidron et al., 2002).

Specifically, both physical and mental stress have been shown to elicit the release of epinephrine (EPI) and norepinephrine (NE) from the SA axis (Gerra et al., 2001; Schoder et al., 2000), and initiate the

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release of corticotrophic hormone, adrenocorticotrophic hormone (ACTH), and cortisol (CORT) from the HPA axis (Gerra et al., 2001). Furthermore, investigators have provided evidence that physical or mental stress can cause the release of interleukin (IL)-6 from a wide variety of cells, including skeletal muscle, adipose, and cytotoxic t-cells (Song et al., 1999). Additionally, increased levels of endothelin-1 (ET-1) and thromboxane-B_2 (TXB_2) have been observed as a result of mental or physical challenges (Camsarl et al., 2003; Song et al., 1999).

Inflammatory cytokines, such as interferon (IFN)-α, IFN-γ, tumor necrosis factor (TNF)-α, IL-1, and IL-6 are activators of the HPA axis (Path et al., 2000), and can further stimulate the release of ET-1 from endothelial cells (Goraca, 2002). Thus, the combination of IL-6 and CORT has been suggested to influence the rate of specific protein release (such as C-reactive protein; CRP) from the liver, which in turn may influence inflammation and platelet aggregation (Bluethmann et al., 1994; Puddu et al., 2010). IL-6 has also been shown to elicit increases in the peptide ET-1, which is the strongest known vasoconstrictor peptide (Goraca, 2002).

Exacerbated and extended activation of stress hormones and inflammatory cytokines may facilitate an increase in vascular dysfunction, possibly resulting in increased platelet aggregation and enhance development of cardiovascular disease. Stress hormones and inflammatory cytokines have been shown to result in increased endothelial dysfunction and sequelae, including arteriosclerosis and atherosclerotic formation (Gidron et al., 2002; Spieker et al., 2002). It has been suggested that chronic systemic inflammation can result in elevation of the stress hormones and cytokines, thereby causing immune and metabolic disturbances, including CVD sequelae (Chrousos, 1995, 2000; Chrousos and Gold, 1992). Thus, this sequence of events may explain the increased prevalence of cardiovascular disease and the high incidence of on-duty cardiovascular related events and cerebrovascular incidences among firefighters (Baris et al., 2001; Calvert et al., 1999; Kales et al., 2003; Soteriades et al., 2002).

A limited number of previous studies have directly investigated dual mental and physical challenges on cardiorespiratory responses (Acevedo et al., 2006; Roth et al., 1990; Rousselle et al., 1995; Szabo et al., 1994; Webb et al., 2008), and few studies have specifically examined stress hormone responses (Huang et al., 2010; Webb et al., 2008). These studies have demonstrated exacerbated cardiorespiratory and stress hormone levels in response to dual-challenge, thus suggesting that the dual nature of stressors commonly experienced by firefighters may also result in exacerbations of other physiological functions. However, these studies have been limited in their scope by utilizing primarily college-aged individuals and psychological stressors such as Stroop Color Word and mental arithmetic tasks or the Trier Social Stress Test.

The effects of firefighting on cardiovascular reactivity and stress hormones has been examined through the use of pre- and post-testing during fire suppression training evolutions (Perroni et al., 2009; Smith et al., 2001, 2005, 1996; Smith and Petruzzello, 1998), and each of these studies has demonstrated increases in cardiovascular and stress hormones in response to the fire suppression drills. However, these studies were unable to control for or measure physiological workload that occurred during the actual fire suppression activities, hence it is difficult to tease out whether the increases reported were the result of physiological, psychological, or a combination of these stressors. By utilizing a controlled workload and a computerized Fire Strategies and Tactics Drill (FSTD), it may be possible to examine the cardiovascular and stress hormone responses of firefighters to concurrent stressors. Thus, the purpose of this study was to examine the effects of a combination of acute physical and mental challenge on stress hormones (ACTH, CORT, EPI, and NE) as well as indicators of vascular regulation (IL-6, ET-1, and TXB_2) in professional firefighters. It was expected that the combined physical and mental challenge would elicit greater elevations in stress hormones and vascular dysfunction during and following a dual stress condition compared to a physical stress alone. Further, it was anticipated that increases in the overall release (as measured by area-under-the-curve [AUC]) of the stress hormones would be related to increases in IL-6, and these increased levels of stress hormones would be related to the overall release (AUC) of ET-1 and TXB_2.

2. Methods

Twelve apparently healthy professional firefighters were recruited from a municipal fire department following approval from the Fire Chief to recruit participants. Prior to the study, the University Institutional Review Board approved the project and informed written consent was obtained from each participant. In addition, all participants were qualified to assume the duties of Incident Commander at a fire scene, and had participated in advanced fire training beyond the basic Firefighter I/II instruction required of professional firefighters in the State of Mississippi. This information was gathered on a self-report firefighting history and education questionnaire.

Participants in this study (a) were classified as low-risk individuals as categorized by ACSM (ACSM, 2010) risk stratification, (b) were free of cardiorespiratory and metabolic disorders, (c) were free of any known blood disorders (e.g., anemia, hemophilia), (d) were without hearing or vision problems, (e) were free of a history of psychological disorders and/or chronic illnesses, (f) had not used any prescription or nonprescription medication or tobacco products within the previous 8 h, (g) were consuming an average of less than ten alcoholic beverages per week, (h) had not experienced any major life events within 30 days of participation (e.g., wedding, death in family, divorce, etc.), and (i) had not engaged in actual fire suppression activities within the previous 72 h.

The participants included in this study were specifically recruited to avoid confounding variables including CVD risk factors (hypercholesterolemia, hypertension, impaired fasting glucose, obesity, and sedentary lifestyle) and other signs/symptoms suggestive of CVD. All participants in this study were free of the previously listed risk factors, and only two of the participants had a single CVD risk factor (one ex-smoker, smoke-free for 7 months, and one with family history).

2.1. Instrumentation

During all testing sessions, a ParvoMedics TrueOne 2400 integrated metabolic measurement system was used to assess cardiorespiratory variables during exercise. Exercise was performed on a CompuTrainer Pro Cycle ergometer with workload controlled by a CompuTrainer Coaching Software (Version 1.1) program specifically written for each participant. An Authorware program (Macromedia, Version 5.0) was specifically written to maintain consistency in data collection timing for the experimental protocols.

2.2. Testing protocol

All participants reported to the laboratory upon completion of their normal work shift (prior to 0730). A minimum of 48 h were allowed to elapse between the first and second session, and a minimum of one week and maximum of two weeks transpired between sessions 2 and 3; and the single and dual-task sessions were randomly assigned and counterbalanced between participants. Prior to each testing session, the participants were asked to abstain from food and caffeine consumption for at least 8 h, and alcohol consumption for 48 h. Participants were also instructed to maintain their normal physical activity levels throughout the duration of their involvement in the research experiment.

A total of three testing sessions comprised the data collection. Session 1 included completion of a medical history questionnaire, informed consent, a two-minute familiarization with the mental challenge, and an assessment of maximal oxygen consumption (VO_{2max}). Participants reached VO_{2max} when either the primary criterion of a plateau in oxygen consumption (VO_2) with an increase in workload was met or 2 of the 3 secondary criteria were achieved. The secondary criteria were (1) reaching predicted maximal heart rate, (2) achieving a respiratory exchange ratio (RER) of greater than 1.1, and (3) reporting a rating of perceived exertion (RPE; 15-point Borg Scale) of 19 or 20.

The second session (see Fig. 1) consisted of either the control condition (exercise alone condition; EAC) or the fire strategies condition (FSC), which served as the mental challenge condition (exercise while participating in a computerized Firefighter Strategies and Tactics Drill; FSTD). The EAC and FSC consisted of exercise at 60% VO_{2max} for a total of 37 min. The participant's workload in watts was predicted by equations developed by the American College of Sports Medicine (ACSM, 2010) and validated using direct measurement of VO_2. A workload of 60% VO_{2max} was selected for this protocol (1) because it was an intensity that would likely be below ventilatory and lactate threshold and (2) because this intensity was similar to the intensity experienced during fire suppression activities (Gledhill and Jamnik, 1992; Sothmann et al., 1992). Additionally, the 37 min duration was utilized to ensure methodological consistency. The initial 12 min of exercise was to ensure participants reached