INTRODUCTION

Firefighting is a physically strenuous job. Firefighters routinely perform heavy work in hot and hostile environments. The personal protective equipment that firefighters wear to protect themselves from these extreme environments also hinders heat dissipation. The physiological strain associated with firefighting is the result of a combination of strenuous physical exertion, heat stress, and activation of the sympathetic nervous system due to the dangerous and unpredictable nature of the work. Because of this combination of stressors, firefighting results in substantial physiological disruption. Research has documented that firefighting leads to elevated heart rate and core temperature, a decrease in plasma volume and stroke volume, and alterations in hormonal and immune function (Barnard and Duncan, 1975; Romet and Frim, 1987; Manning and Griggs, 1983; Smith et al, 2001a; Smith et al, 2001b; Smith et al, 2005).

Sudden cardiac events are the leading cause of line of duty deaths among firefighters in the United States. In fact, the United States Fire Administration has reported that for an 11-year period (1990-2000), 43.9% of all line of duty deaths among firefighters were due to cardiac events, nearly double the second leading cause of death (trauma) (USFA, 2002). Recently, Kales and colleagues (2007) documented the relative risk of sudden cardiac death associated with different firefighting activities. The authors reviewed records of firefighting line of duty deaths between 1994 and 2004 (excepting the deaths associated with September 11, 2001). Odds ratios were calculated based on the amount of time that firefighters spend in various duties and the number of fatalities that occurred during each type of duty. Despite the fact that firefighters report spending only 1 to 5% of their time in fire suppression activities, 32% of line of duty deaths occurred during these activities. The statistical analysis revealed that firefighters had a 12 to 136-fold increased risk of suffering a fatal cardiac event following fire suppression activities than during nonemergency duties (Kales et al, 2007).

Thrombus formation is the central process in many sudden cardiac events and activation of platelets has been strongly linked to sudden cardiac events following strenuous exercise (Mittleman et al, 1993; Willich et al, 1993). However, the effect of firefighting - with the combined stress of heavy physical exertion, sympathetic nervous activation and heat stress - on platelet number and activity has not been described. Therefore, the purpose of this study was to examine the effect of live-fire firefighting training on platelet number and aggregability.

METHODS

Participants:

Ten apparently healthy professional firefighters, who had recently been hired by full-time career departments and were taking part in a six-week academy course, were recruited for this study. Prior to testing, written informed consent was obtained from all participants, indicating that they understood all the procedures, risks, and benefits of this study and further indicating
that participation in the study was voluntary. Testing protocols were approved by the Human Participants Institutional Review Board. Potential participants were excluded from the study if they reported taking acetylsalicylic acid (ASA) or other medications known to affect platelet function within two weeks of testing.

Participant’s age, height, weight, aerobic fitness capacity (maximal oxygen consumption) and percent body fat were obtained prior to testing. Percent body fat was estimated via 7-site skin fold measurements according to the equations of Jackson and Pollock (1985). Maximal oxygen consumption was estimated based on the equation of Kline et al. (1987).

Procedures:
Participants reported for testing in a fasted state at 0800 hours and were fed a standardized meal (240 ml cereal, 120 ml of skim milk and decaffeinated coffee). After preliminary measures and a blood draw, participants performed firefighting drills in a training structure that contained live fires on the second and fourth floors. The firefighting drills consisting of four individual firefighting tasks: carrying a “high-rise” pack up stairs, performing a dummy drag, performing a primary search, and discharging a pump can. Specifically, participants ascended 3 flights of stairs and then descended 2 flights while carrying a 16.2 kg “high rise” pack. On the second floor, they dragged a dummy around the outside of the room (dimensions of the room: 10.3 x 12.1 m). Participants then carried the “high rise” pack to the third floor where they performed a primary search around the perimeter of a room. Finally participants moved to the fourth floor with the “high rise” pack where they discharged 15 pumps of water from a 5 gallon pump-can. Immediately following the completion of the fourth task, participants returned to the first floor and were instructed to complete the same series of firefighting drills for a second time. Participants were instructed to complete the tasks in as little time as possible while not compromising the quality of the task or their own safety. Participants wore personal protective equipment that met current National Fire Protection Agency standards (NFPA 1500) and a self-contained breathing apparatus (SCBA). Data collection occurred on the first floor of the fire-training structure before and after the drills.

Measures:
Venous blood samples were obtained via a venipuncture in an anticubital vein prior to and after the firefighting drills. A 21 gauge needle was used to draw blood directly into a 5mL tube containing K3 EDTA for CBC analysis and a 4.5mL tube containing 0.105M sodium citrate for platelet closure time. Blood samples were kept at room temperature (~21ºC) until they were analysed (within two hours). Blood samples were analysed for closure time (an index of platelet aggregation) using a Platelet Function Analyzer (PFA 100; Dade Bearing Inc., Florida). The PFA-100 test uses a small sample (800 µL) of whole citrated blood which is aspirated through a small tube under high shear force to activate the platelets. The blood then passes through an aperture (150 µm in diameter) in a membrane that is coated with collagen and ADP (Col/ADP) or collagen and epinephrine (Col/Epi). As the platelets adhere to the membrane and begin to aggregate they intrude into the lumen of the aperture, eventually fully occluding the aperture and arresting blood flow through the membrane. The PFA-100 measures closure time - the time (in sec) it takes to form a platelet aggregate and occlude blood flow (Francis, 2007). Thus, a shorter closure time reflects increased aggregability.

Data Analysis:
Data are presented as means and standard deviations. Paired t-tests (two-tailed) were performed on all variables to determine if there were any significant differences between pre and post firefighting values. The significance level was set at \( p < 0.05 \).

RESULTS

Participants were adult males with an average age of 26.9 ± 3.5 years. They had an average estimated aerobic capacity of 48.5 ± 3.5 ml·kg\(^{-1}\)·min\(^{-1}\) and had an average estimated body fat of 24.2 ± 10.9%. The total time to complete the firefighting drills averaged 9:32 minutes. Mean tympanic temperature increased significantly (1.4 ± 0.7°C) and plasma volume decreased significantly (10.5 ± 2.7%) from pre- to post-firefighting drills.

Table 1 presents changes in hematological variables as a result of firefighting activity. Post firefighting values are reported in uncorrected terms and corrected for changes in plasma volume according to the equation of Greenleaf et al, 1979. There was a significant leukocytosis that was evident even when values were corrected for changes in plasma volume. Platelet count increased significantly from pre to post firefighting (34%). When corrected for changes in plasma volume, post firefighting platelet counts were still 19% higher than pre firefighting values.

<table>
<thead>
<tr>
<th></th>
<th>Pre-Firefighting</th>
<th>Post-Firefighting Uncorrected *</th>
<th>Post-Firefighting Corrected b</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leukocyte count (x10(^3)/μL)</td>
<td>6.8 ± 1.1</td>
<td>12.1 ± 3.4*</td>
<td>10.8 ± 3.0*</td>
</tr>
<tr>
<td>Erythrocyte count (x10(^6)/μL)</td>
<td>4.9 ± 0.3</td>
<td>5.14 ± 0.3*</td>
<td>4.6 ± 0.3*</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>14.5 ± 0.7</td>
<td>15.4 ± 0.8*</td>
<td>13.8 ± 0.8*</td>
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<tr>
<td>Hematocrit (%)</td>
<td>43.1 ± 1.7</td>
<td>46.2 ± 1.7*</td>
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<tr>
<td>Platelet Count (x10(^3)/μL)</td>
<td>241.4 ± 47.5</td>
<td>322.8 ± 58.0*</td>
<td>288.8 ± 54.4*</td>
</tr>
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*p < 0.05 vs pre firefighting
*uncorrected for changes in plasma volume
*corrected for changes in plasma volume

As seen in Figure 1, mean closure time decreased significantly (p < 0.05) from pre- to post-firefighting activity (82.7 to 72.8 sec) when whole blood was exposed to collagen and ADP. When blood was exposed to collagen and epinephrine (not shown), there was no statistically significant difference detected in closure time. Figure 1 also reports individual changes in closure time for each participant. Seven of the 10 participants had a slight decrease in closure time whereas three of the participants had an increased closure time.
CONCLUSIONS

Consistent with earlier studies (Smith et al, 2001; Smith et al, 2005), we have found that firefighting activity results in a significant decrease in plasma volume and an increase in circulating leukocytes. The novel finding of this study, however, is that even short bouts of live-fire firefighting drills result in a significant increase in platelet count and a significant decrease in closure time, indicating that the blood may be hypercoaguable following firefighting activity.

Previous studies have generally found that strenuous exercise results in an increase in platelet number (El-Sayed et al., 1996; Ikaguri et al, 2003; Wang et al, 1994). For example, Ikaguri et al. (2003) investigated the platelet response to 30 minutes of cycle exercise at two different intensities in 7 healthy men aged 20-29 years. The authors reported no significant increase in platelet count when exercise was performed at 55% of VO\textsubscript{2}max, but they did report a 21% increase in platelet count when exercise was performed at 80% of VO\textsubscript{2}max. Because the firefighting activity in this study was only performed for less than 10 min, it is surprising to see such a large (34%) increase in platelet count. The elevated platelet count following firefighting is partially explained by shifts in body fluid. However, even after correcting platelet count for changes in plasma volume, there was a 19% increase in platelet count. Our results may be explained by sympathetic nervous stimulation resulting from participating in live-fire training or the heat stress encountered by our participants.

Research findings on the effect of exercise on platelet activity are conflicting. The preponderance of evidence suggests that platelet aggregation is increased with exercise in patients with coronary artery disease, but the effect of exercise on healthy individuals is less clear. Studies using whole blood flow cytometry have found in vivo activation and hyperactivity of platelets following exercise in sedentary subjects but not in physically trained subjects (El-Sayed et al, 2005). Ikarugi et al. (2001) reported that 40 minutes of cycling at 50-55% VO\textsubscript{2}max in premenopausal middle-aged women resulted in a significant increase (6.7%) in platelet number but no significant change in platelet occlusion time.

Figure 1. Platelet Aggregation Time Changes with Firefighting Activity
In conclusion, this study found that a short bout (~9:30 min) of strenuous live-fire firefighting causes an increase in platelet count and platelet aggregation as evidenced by a decrease in closure time. These results suggest that firefighting may result in a hypercoaguable state that could increase the risk of sudden cardiac events. Our results are particularly noteworthy as the firefighting activities used in this study were of short duration and the participants in this study were young, apparently healthy, and physically fit. It is likely that older individuals or less fit firefighters would have a more pronounced platelet response to firefighting activity. Further research is needed to elucidate the effects of prolonged firefighting on platelet count and function, to investigate the relationship between firefighter health status and platelet response, and to better describe how the hemostatic balance is altered by firefighting activity.

REFERENCES