Effect of Live-Fire Training Drills on Firefighters' Platelet Number and Function

Denise L. Smith, PhD, Steven J. Petruzzello, PhD, Eric Goldstein, MPP, Uzma Ahmad, MD, Krishnarao Tangella, MD, Gregory G. Freund, MD, Gavin P. Horn, PhD

Abstract

Background. The leading cause of line-of-duty death among firefighters is sudden cardiac events. Platelets play a critical role in the formation of an occlusive thrombus during an ischemic event. **Objective.** The purpose of this study was to examine the acute effect of firefighting on platelet number and aggregability. **Methods.** Apparently healthy male firefighters ($N = 114$; age 29.4 ± 7.8 years) participated in 18 minutes of simulated firefighting activity in a training structure that contained live fires. Blood samples were obtained before and after simulated firefighting activity and analyzed for complete blood cell count (CBC), chemistry, and platelet number and function. Platelet function was measured using a PFA-100 analyzer to assess platelet aggregability. **Results.** As expected, performing firefighting activity resulted in significant increases in heart rate (75 b·min^{-1}) and core temperature (0.7°C), and significant changes in blood chemistry values. The most important finding in this study is that 18 minutes of simulated firefighting caused a 24% increase in platelet number and a significant increase in platelet aggregability. **Conclusions.** Firefighting resulted in a significant increase in platelet number and aggregability, indicating that even short bouts of firefighting can increase thrombotic potential in apparently healthy firefighters. **Key words:** thrombosis; platelet count; platelet function test; platelet aggregation; firefighter

Introduction

While operating at a structure fire, firefighters perform strenuous physical work while wearing heavy personal protective equipment (PPE), often in hot environments and under physiologically stressful conditions. Firefighting results in significant cardiovascular and thermal strain as a result of this combination of strenuous work, heavy and insulating PPE, psychological stress, and environmental extremes. Of particular concern is the high rate of line-of-duty deaths attributable to sudden cardiac events: approximately 45% of line-of-duty deaths in the fire service.[1] Furthermore, a 2007 study by Kales and coworkers clearly demonstrated that the relative risk of suffering from a fatal cardiac event was 10–100 times greater following fire-suppression activities than during nonfire duties.[2] Cardiac emergencies have obvious consequences for the individual involved, but cardiac events on the fireground also have potential to significantly impede fireground operations, including fire suppression and victim rescue, thereby negatively impacting public safety. In light of the consequences of thrombus formation during emergency operations, and the evidence that cardiac events are much more likely to occur during or shortly after fire-suppression activities, it is important to describe the acute effects of firefighting on firefighters' platelet number and function.

Heavy physical exertion (such as fire-suppression operations) can serve as a trigger for sudden cardiac events.[3–5] Sudden cardiac events are typically the result of plaque disruption and subsequent thrombus formation; however, approximately one-third of acute coronary syndromes, particularly sudden cardiac death, occur without plaque disruption and require only superficial erosion of fibrotic plaque.[6] Platelets play a critical role in the formation of an occlusive thrombus, and an increase in platelet aggregation is associated with unstable angina and myocardial infarction.[3,4,7]

Despite the challenging conditions that firefighters routinely encounter in the line of duty and the high occupational rate of sudden cardiac events, there is a paucity of data on the effect of firefighting on blood parameters and thus minimal information on which to judge effective medical treatment options. Given that sudden cardiac events are the leading cause of line-of-duty death among firefighters, it is important to

TABLE 1. Descriptive Characteristics of the Participating Firefighters ($N = 114$)

Age, yr	29.4 (7.8)
Height, m	1.77 (0.07)
Weight, kg	88.1 (15.7)
BMI, kg/m^2	28.2 (4.5)

Data are expressed as mean ($\pm$ standard deviation).
BMI = body mass index.

describe the effect of firefighting activities on firefighters' platelets. Platelet count and function are related to cardiovascular death in apparently healthy men,[8] and platelet response to strenuous exercise is often implicated in the formation of an occlusive thrombosis.[3,6]

The purpose of this study, therefore, was to describe changes in firefighters' platelet number and aggregability in response to live-fire training in order to improve our understanding of the potential mechanisms of sudden cardiac events shortly after fire-suppression activities. This study also examined the effect of live-fire training on firefighters' blood chemistry and hematologic variables.

METHODS

The participants were 114 male firefighters, from approximately 40 fire departments across the state of Illinois. Participants were career ($n = 53$) and volunteer ($n = 55$) firefighters (six individuals served on both career and volunteer fire departments). Table 1 displays the descriptive data for the study participants (mean $\pm$ standard deviation).

Participants signed an informed consent document indicating that they understood the risks and benefits of participation and that their participation was voluntary. This study was approved by the University of Illinois Institutional Review Board. Prior to participation in the testing, participants also completed a health history questionnaire. Firefighters who indicated that they had a diagnosed history of atherosclerotic cardiovascular disease or were taking medications for high blood pressure or cholesterol were excluded from participating in the study. Participants who had taken medications that affect blood platelets or blood clotting (such as aspirin, other pain relievers, or cold medications) were not allowed to participate until at least seven days had passed from their last ingestion of such medication. A total of 143 subjects were screened for this study.

The participants ingested a silicone-coated gastrointestinal (GI) core temperature capsule (Mini Mitter, VitalSense; Philips Respironics, Bend, OR) six to 12 hours prior to reporting for the study. Simulated firefighting activities were performed in a six-story training tower at the Illinois Fire Service Institute. The participants were instrumented with a heart rate (HR)

monitor (Polar Electro Oy, Kempele, Finland), and a venous blood sample was obtained before participants donned their PPE. Baseline physiologic data (HR and core temperature [T_{co}]) were then recorded and the participants were asked to answer several perceptual measures (rating of perceived exertion,[9] thermal sensation scale[10]). Participants then donned their mask and hood and entered the training course. Once the simulated firefighting activities were completed, the participants immediately returned to the first floor of the training building, completed the same measurements, and provided post–simulated firefighting activity blood samples.

The simulated firefighting activities took place on the second floor of the training building where temperatures at waist level (1.2 m) averaged between 71°C and 82°C. Throughout the study, participants wore National Fire Protection Association (NFPA) 1971[11]–compliant PPE that was purchased for the study. Participants wore a Scott 50i self-contained breathing apparatus (SCBA) with a 30-minute carbon-fiber air cylinder. The average weight of a full set of PPE with SCBA was 19.8 kg. The firefighters completed 18 minutes of simulated firefighting activity consisting of nine 2-minute periods of alternating rest/work cycles. Thus, the drills involved 8 minutes of activity and 10 minutes of rest in a building that contained live fires. The work cycles included stair climbing, simulated forcible entry using a Keiser Force Machine (Keiser Corporation, Fresno, CA), a simulated secondary search, and simulated hose advance.

The T_{co} and HR readings were collected after the participant donned his PPE (before firefighting) and immediately after firefighting activities were completed (post–simulated firefighting activity). Additionally, HR was recorded while the participants were engaged in the simulated firefighting tasks. Venous blood was drawn from the antecubital vein using a 21-gauge needle before firefighters donned their PPE and after firefighting activity. Blood samples were obtained with little or no stasis by a trained phlebotomist.

For CBC analysis, venous blood samples were collected in Vacutainer tubes (Becton, Dickinson and Company, Franklin Lakes, NJ) containing tripotassium ethylenediaminetetraacetic acid (K3 EDTA), maintained at room temperature, and analyzed within 2.5 hours after collection. The CBC analysis was performed using a Cell-Dyn 3200 automated analysis system (Abbott Laboratories, Abbott Park, IL) using flow-cytometry technology. A board-certified pathologist evaluated the morphologic aspects of the peripheral smears (before and after simulated firefighting activity).

For the blood chemistry analysis, venous blood samples were collected in serum separator tubes, inverted several times, allowed to clot at room temperature, and then centrifuged at 3,700 rpm for 9 minutes. The

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samples were stored refrigerated at 4°C if they were not tested immediately. Testing was performed on a Vitros 950 analyzer (Johnson & Johnson, Ortho Clinical Diagnostics, Kowloon, Hong Kong).

For platelet function analysis, venous blood samples were collected in a Vacutainer tube containing 3.2% sodium citrate. Samples were maintained at room temperature and analyzed within two hours after collection. Epinephrine (EPI)-induced and adenosine-5′-diphosphate (ADP)-induced platelet aggregability was analyzed using a PFA-100 Platelet Function Analyzer (Siemens Healthcare Diagnostics, Deerfield, IL). The PFA-100 is a system that permits the in vitro measurement of platelet function in anticoagulated whole blood.[12] The test is performed under high-shear conditions, such as occurs in stenotic vessels. Whole blood (mixed with citrate in the collection tube) is loaded into a disposable test cartridge reservoir. Each test cartridge contains a collagen membrane that also includes either EPI (10 μg) or ADP (50 μg). The PFA-100 analyzer aspirates the blood sample through a capillary (200 μm) and into a cup where it comes in contact with the agonist, and then passes it through an aperture (150 μm). The blood is aspirated under high shear rates of 5,000–6,000 seconds^{-1}. In response to the collagen and EPI or ADP, and the high shear rate, platelets are stimulated. The stimulated platelets adhere to the membrane and aggregate, causing occlusion of the membrane aperture and cessation of blood flow. The time to fully occlude the aperture is defined as "closure time" and is indicative of platelet function.[12,13]

Descriptive statistics were calculated for each variable before and after simulated firefighting activity and are reported as mean ± standard deviation. A Student's paired t-test was used to test for significant differences between the pre and post measurements.

RESULTS

Overall, there was a significant difference between the pre– and post–simulated firefighting activity values for most of the variables we tested, indicating that the 18-minute simulated firefighting activities undertaken in this trial caused significant physiologic changes. The most important findings of this study are that 18 minutes of moderate-intensity firefighting caused a significant increase in platelet number and platelet aggregation. In addition, 18 minutes of firefighting activity caused a significant increase in body temperature and HR. Posttrial HRs increased significantly (75 b·min^{-1} p < 0.001), with an average peak HR of 167.4 ± 15.3 b·min^{-1}, approximately 88% of age-predicted maximal HR. The post–simulated firefighting activity T_{co} values were significantly (p < 0.001) increased by 0.72°C ± 0.31°C compared with prefirefighting values. The firefighting activities undertaken in the trial resulted in significant (p <

TABLE 2. Changes in Blood Chemistry Values As a Result of Firefighting Activities ($N = 113$)

	Pre		Post		%
	Mean	SD	Mean	SD	Change
Glucose, mg/dL	81.93	17.67	104.6*	19.83	27.6
BUN, mg/dL	15.29	3.58	14.82*	3.48	−3.05
Creatinine, mg/dL	1.12	0.14	1.32*	0.17	17.4
Na$^+$, mmol/dL	142.7	1.35	145.2*	1.56	1.70
K$^+$, mmol/dL	4.06	0.35	4.17*	0.27	2.81
Cl$^-$, mmol/dL	102.5	2.04	104.4*	2.34	1.86
Total bilirubin, mg/dL	0.52	0.32	0.61*	0.36	18.3
Ca^{++}, mg/dL	9.77	0.31	10.47*	0.37	7.16
TP, g/dL	7.47	0.37	8.26*	0.41	10.6
Albumin, g/dL	4.69	0.24	5.33*	0.27	13.6
AST, U/L	30.80	9.84	33.64*	11.10	9.22
ALT, U/L	35.47	12.17	34.28*	13.21	−3.36
AlkP, U/L	80.80	17.45	85.37*	18.62	5.66
CO$_2$, mmol/L	27.46	2.13	19.15*	2.58	−30.3

*All measures are significantly different from those under the prefirefighting condition at p < 0.001.
AlkP = alkaline phosphatase; ALT = alanine aminotransferase; AST = aspartate aminotransferase; BUN = blood urea nitrogen; Ca^{++} = calcium; Cl$^-$ = chloride; CO$_2$ = carbon dioxide; K$^+$ = potassium; Na$^+$ = sodium; SD = standard deviation; TP = total protein.

0.001) increases in self-reported thermal sensations of 1.6 ± 0.9 units to a mean of 5.9 ± 0.9 (representing a change from "comfortable" to "hot") and a perceived exertion level of 14.5 ± 2.1 ("hard" or "heavy").

There was a significant change in all blood chemistry values (Table 2). The reduction in plasma volume was calculated at 9% (Greenleaf method[14]). Glucose level increased significantly by 28%, reaching a mean value of 104.6 ± 19.8 mg/dL after simulated firefighting activity (p < 0.001). Creatinine levels increased by 17%, reaching 1.3 ± 0.2 mg/dL (p < 0.001), along with concomitant increases in total protein and albumin levels. Most of the other measured blood chemistry variables (urea nitrogen, sodium, potassium, chloride, calcium, aspartate aminotransferase, and alkaline phosphatase levels) demonstrated significant increases from pre– to post–simulated firefighting activity measurements.

The hematologic responses to the firefighting trials are reported in Table 3. There was a significant increase of circulating leukocytes from before to after simulated firefighting activity, with the number of leukocytes increasing by 38% (p < 0.001). Significant (p < 0.001) increases in monocytes, lymphocytes, and neutrophils were also seen, along with general leukocytosis. There was also a demonstrated increase (p < 0.001) in red blood cell count, hemoglobin, and hematocrit of approximately 5% that is likely due to the hemoconcentration. The pathologist found that for each blood smear, the morphologic evaluation was correlated with the quantitative hematologic values. No significant hemolysis was detected. Red blood cell, white blood cell, and platelet morphologies were equivalent for pre

TABLE 3. Changes in Hematology Values As a Result of Firefighting Activities ($N = 113$)

	Pre		Post		%
	Mean	SD	Mean	SD	Change
WBCs, K/μL	7.38	2.02	10.15*	2.65	37.5
Neutrophils, K/μL	4.13	1.70	5.39*	1.95	30.4
Lymphocytes, K/μL	2.38	0.81	3.61*	1.19	51.6
Monocytes, K/μL	0.58	0.15	0.79*	0.23	35.7
Eosinophils, K/μL	0.19	0.15	0.21*	0.19	10.4
Basophils, K/μL	0.10	0.04	0.15*	0.06	50.7
RBCs, M/μL	5.08	0.53	5.38*	0.31	5.71
Hemoglobin, g/dL	15.7	1.4	16.5*	0.9	4.89
Hematocrit,%	46.2	2.6	48.6*	2.6	5.17
MCV, fL	90.3	3.1	90.5*	3.0	0.27
MCH, pg	30.8	1.3	30.6*	1.3	−0.49
MCHC, g/dL	34.1	0.9	33.9*	0.9	−0.76
RDW,%	11.6	1.1	11.8	0.6	1.47
Platelets, K/μL	264.4	53.8	329.0*	66.4	24.4
MPV, fL	9.60	1.81	9.56	1.97	−0.49

*Significantly different from those under the pre condition at $p < 0.001$.
K = 1,000; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red-cell distribution width; SD = standard deviation; WBC = white blood cell (leukocyte).

and post samples as evaluated by light microscopy. In addition, no circulating blasts were indentified.

There was a significant (~24%) increase in platelet number following firefighting activity ($p < 0.001$). Additionally, firefighting activities caused a decrease in closure time when the blood was exposed to collagen and EPI ($p < 0.001$; ~14%), indicating that platelet function was enhanced following firefighting activity (Fig. 1). Student's t-tests indicated that the approximately 3% decrease in closure time in the platelets exposed to collagen and ADP was not significant.

DISCUSSION

The most important finding of this study is that a relatively short bout of live-fire firefighting activity causes a significant increase in platelet number and ag-
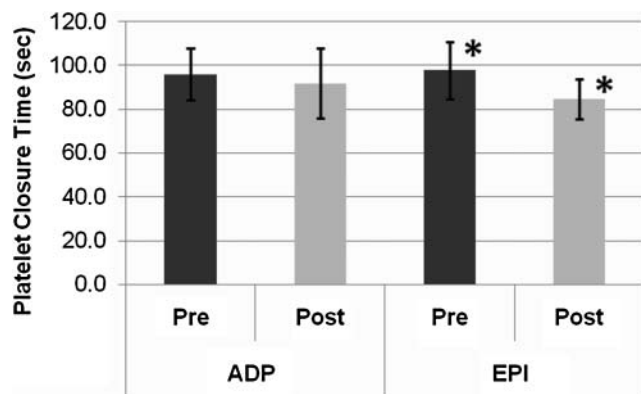

Figure 1. Platelet function response to firefighting activities. Error bar indicates one standard deviation ($N = 97$, *$p < 0.001$). ADP = adenosine-5´-diphosphate; EPI = epinephrine.

gregability in apparently healthy, young firefighters. These findings suggest that participating in live-fire firefighting activities may increase an individual's risk of thrombosis formation. Furthermore, the results of this study confirm the results of earlier studies, with smaller sample sizes, that have found that even short bouts of firefighting activity in a live-fire environment result in significant physiologic strain reflected by an increase in HR and T_{co}, alterations in blood chemistry, and leukocytosis.[15–19]

The average HR reported immediately after returning to the first floor (i.e., 2 minutes after completing the hose advance portion of the live-fire drills) was 168 b·min^{-1}, which is consistent with the results of previous studies.[15,16] Following just 18 minutes of simulated firefighting activity, the T_{co} rose significantly by 0.72°C. This modest increase in firefighters' T_{co} would not be of particular concern to emergency medical services (EMS) personnel and poses no immediate risk of heat illness. However, the effect of this increase (and rate of rise) on physiologic function is unknown. Furthermore, this is a considerable rate of rise for only 18 minutes of work, which for most firefighters required slightly less than one cylinder of air. During actual firefighting operations, firefighters often consume multiple cylinders of air and continue to wear their PPE even after fire-suppression activities have ceased, which is likely to cause body temperature to continue to rise substantially during overhaul and cleanup activities. The increase in T_{co} reported here is higher than that found in an earlier study ($n = 8$) that reported a rate of rise of 0.032°C·min^{-1} during rescue and fire attack activities,[17] suggesting that there is variability in the T_{co} response depending on individual characteristics and the firefighting tasks that are undertaken.

Firefighting resulted in significant changes in blood chemistry; however, none of the changes were outside clinically normal ranges after this short-term bout. Elevated glucose levels, which increased more than 27% from pre– to post–simulated firefighting activity, are likely the result of a combination of hemoconcentration and increased sympathetic nervous activation.[18] A 17% increase in creatinine levels and concomitant increases in total protein and albumin levels are consistent with modest dehydration. Most other measured blood variables (urea nitrogen, sodium, potassium, chloride, calcium, aspartate aminotransferase, and alkaline phosphatase levels) demonstrated increases from pre– to post–simulated firefighting activity measurements; however, this was likely to be due primarily to the overall decrease in plasma volume. The changes that are reported here are consistent with earlier reports suggesting that short-term firefighting results in modest changes in blood chemistry in healthy, young firefighters.[18] The reduction in plasma volume (9%) that we documented in this study is less than has been reported in an early study. Smith et al. reported a plasma volume decrease of

approximately 15% when firefighters engaged in approximately 17 minutes of firefighting activity without rest periods.[18]

There was a significant increase of circulating leukocytes from before to after simulated firefighting activity, with the number of leukocytes increasing by 38%. The magnitude of this increase is considerably less than that reported following 17 minutes of firefighting activity.[19] The increase seen in this study is consistent with what has been reported during moderate-intensity exercise. The magnitude of exercise-induced leukocytosis is dependent on intensity of exercise, with increases of 20–50% being common for moderate-intensity exercise.[20] The increase in leukocytes is often attributed to the action of the sympathoadrenal-mediated stress hormones; however, there is also evidence that increased body temperature influences the magnitude of stress hormone release.[21]

There was a significant increase (24%) in platelet number following firefighting activity. Platelets play a critical role in the formation of a blood clot. The increase in platelet count normally seen with exercise (10–40%) is dependent on exercise intensity.[5] Ikarguri et al. reported a 21% increase in platelet count when young men performed cycling exercise for 30 minutes at 80% of maximum volume of oxygen consumption (VO_{2max}) (strenuous exercise), but found no change in platelet number when exercise was performed at 55% of VO_{2max} (moderate exercise).[5] The increase in platelet number reflects hemoconcentration and an increase in the catecholamines that cause platelets to be released from the spleen and lymphatic tissue.[22]

Platelet function plays a pivotal role in acute coronary artery disease, myocardial infarction, and arterial thrombosis.[23] Platelet activation and subsequent thrombus formation have long been linked to cardiovascular complications following strenuous exercise.[4,24] Enhanced blood flow, elevated HR, and high blood pressure can damage endothelial lining and even provoke plaque rupture, which can provide the surface for platelet adherence and thrombus formation. Firefighting leads to near-maximal HR and may produce a situation in which endothelial damage is more likely to occur. In this study, we found an increase in platelet activity, evidenced by a decrease in time required to form a thrombus and occlude the aperture (a decreased closure time) when the blood was exposed to collagen and EPI.

The effect of exercise on platelet function has led to conflicting results.[23,25] These are partly attributable to methodologic difficulties in the measurement of platelet function[25,26] in addition to changes in platelet function with variations in the intensity and duration of exercise, and the fitness level and health status of the exerciser.

In the study by Ikarguri et al., platelet reactivity (assessed by hemostatometry) was significantly increased following strenuous exercise but not after moderate exercise.[5] Kestin and coworkers (1993) investigated the effect of an incremental test to maximum on a motor-driven treadmill (strenuous exercise) on platelet activation and reactivity in sedentary and physically active participants.[26] These authors reported that strenuous exercise in sedentary volunteers, but not physically active volunteers, resulted in platelet activation and platelet hyperreactivity. Wang et al. (1994) reported that in both healthy sedentary volunteers and healthy physically trained volunteers, an incremental exercise test to volitional exhaustion on a bicycle ergometer (strenuous exercise) resulted in increased platelet adhesiveness and aggregation.[27] Moderate exercise (50–55% of VO_{2max} on a cycle ergometer for 30 min), however, resulted in a decrease in platelet adhesiveness and aggregation in the sedentary volunteers and a decrease in platelet adhesiveness in the trained individuals. Singh et al. (2006) found that activation was lower in a sedentary group of volunteers than in trained volunteers after one hour of cycling exercise at 70% of VO_{2max}.[28] Studies in individuals with cardiovascular disease strongly suggest that both moderate exercise and strenuous exercise enhance platelet function.[27,29]

In the current study, platelet function was assessed using a platelet function analyzer (PFA-100) that was designed to provide rapid screening of platelet dysfunction[12] and has since proven useful in assessing abnormalities in primary hemostasis.[30,31] This technique exposes citrated whole blood to hemodynamic conditions close to those encountered at the site of vascular injury or lesion. Test cartridges contain collagen and EPI or ADP. Collagen type-1 is used to simulate the subendothelial matrix on which platelet anchorage occurs. Epinephrine or ADP is added to the cartridge to act as a platelet agonist to accelerate the occlusion process.[12] Closure times from cartridges containing collagen and EPI and collagen and ADP vary, and changes in closure time of the cartridges differ depending on the perturbation.[12,30,31]

A rise in circulating platelets following exercise is generally attributed to the release of platelets from the vascular beds of the spleen, the bone marrow, and the intravascular pool of the pulmonary circulation.[22] The mechanisms responsible for platelet aggregation during strenuous exercise have not been fully elucidated. Hjemdahl et al. found that exercise-induced increases in catecholamine levels result in heightened platelet aggregation, indicating that the greater the concentration of catecholamines, the greater the platelet function.[32] However, studies that measured catecholamines or blocked alpha$_2$-adrenoreceptor sites demonstrated that platelet hyperaggregation during exercise cannot be fully explained by the adrenoreceptor pathway.[33] It is also possible that exercise stimulates circulatory activation of platelets, resulting in the

mobilization of newly formed and more metabolically active platelet.[34] Endothelial damage due to hemodynamic stress associated with exercise may expose collagen and enhance platelet activation and aggregation. Other potential mechanisms for exercise-induced platelet activation include increased lactic acid level, increased body temperature, and exercise-induced hemoconcentration.[23,25] Furthermore, evidence suggests that platelets can be activated directly by heat.[35]

Regardless of the mechanism, the increased activity of platelets reported in this study is of concern to EMS personnel and physicians treating firefighters who experience cardiovascular events during emergency operations. Considering the strong evidence that cardiac events are much more likely to occur during or shortly after fire-suppression activities than during other work-related duties, it is important that EMS personnel understand the acute physiologic effects of firefighting (especially on platelet number and function) so that they are better prepared to respond to medical emergencies involving firefighters.

LIMITATIONS AND FUTURE RESEARCH

Several limitations may have affected our results. First, this study used simulated firefighting activities in a training structure that contained live fires. This situation is less dangerous and thus likely less stressful than actual firefighting activities. As such, our results may underestimate the actual cardiovascular strain associated with firefighting activity. The firefighters in this study were relatively young and healthy, and thus may not represent the entire fire service population. It is probable that many of the changes we documented following firefighting activity would be exaggerated in older, less-fit individuals. This study included only measures of platelet number and function as indices of thrombotic potential. Future studies should assess a broader range of hemostatic variables to better understand the effect of firefighting on thrombotic potential.

CONCLUSIONS

Even a short bout of moderate firefighting activity (18 minutes) in young, apparently healthy male firefighters resulted in a large increase in circulating platelets and an increase in platelet aggregability (as measured by a decreased collagen and EPI closure time). These findings suggest that firefighting activity may lead to an increased risk of thrombus formation, which is a possible mechanism by which firefighting can trigger a sudden cardiac event in individuals with underlying cardiovascular disease. These results suggest the need for further research to describe hemostatic responses to firefighting and possible interventions and strategies to mitigate potentially dangerous alterations in hemostasis as a result of strenuous firefighting activity.

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