Clotting and Fibrinolytic Changes after Firefighting Activities

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ABSTRACT

SMITH, D. L., G. P. HORN, S. J. PETRUZZELLO, G. FAHEY, J. WOODS, and B. FERNHALL. Clotting and Fibrinolytic Changes after Firefighting Activities. *Med. Sci. Sports Exerc.*, Vol. 46, No. 3, pp. 448–454, 2014. Approximately 45%–50% of all duty-related deaths among firefighters are due to sudden cardiovascular events, and a disproportionate number of these fatalities occur after strenuous fire suppression activities. **Purpose**: The objective of this study is to evaluate the effect of strenuous firefighting activities on platelets, coagulation, and fibrinolytic activity and to document the extent to which these variables recovered 2 h after completion of the firefighting activity. **Methods**: Firefighters performed 18 min of simulated firefighting activities in a training structure that contained live fires. After firefighting activities, firefighters were provided with fluid and allowed to cool down and then recovered for 2 h in an adjacent room. Blood samples were obtained prefirefighting, postfirefighting, and 2 h postfirefighting. **Results**: Platelet number, platelet activity, and coagulatory potential increased immediately postfirefighting and many variables (platelet function, partial thromboplastin time, and factor VIII) reflected a procoagulatory state even after 2 h of recovery. Fibrinolysis, as reflected by tissue plasminogen activator, also was enhanced immediately postfirefighting but returned to baseline values by 2 h postfirefighting. In contrast, inhibition of fibrinolysis, as evidenced by a reduction in plasminogen activator inhibitor-1, was depressed at 2 h postfirefighting. **Conclusions**: Firefighting resulted in elevated coagulatory and fibrinolytic activity. However, 2 h postfirefighting, tissue plasminogen activator returned to baseline and coagulatory potential remained elevated. The procoagulatory state that exists after firefighting may provide a mechanistic link to the reports of sudden cardiac events after strenuous fire suppression activities. **Key Words:** HEMOSTASIS, COAGULATION, FIBRINOLYSIS, PLATELET ACTIVITY

Large multicenter studies have reported a relative risk of 2.1 to 5.9 for experiencing an acute myocardial infarction within 1 h of heavy physical exertion (6 METs or higher) compared with light activity or no exertion (25,39). Coronary artery thrombus formation plays a critical role in exercise-induced myocardial infarctions (36). Changes in blood clotting and fibrinolytic activity influence thrombus formation, and an imbalance in these systems alters the risk of a thrombus formation.

Over the past 10 yr, approximately 40%–50% of duty-related deaths among US firefighters have been attributed to sudden cardiac events (10). In addition, a review of the number of cardiac fatalities and the number of cardiac injuries reported by the National Fire Protection Association over the past 5 yr suggests that there are approximately 25 nonfatal line-of-duty cardiac events for every fatal cardiac event. Although firefighters spend a small percentage of their time engaged in firefighting activity, a large portion of their cardiac fatalities occur during or shortly after firefighting activity. Retrospective studies indicate that firefighters are 53 to 64 times more likely to experience a fatal cardiac event during or shortly after fire suppression activity than during station duty (19,20). Importantly, these studies also report that firefighters have a relative risk of 5.2 to 7.6 of experiencing a fatal sudden cardiac event after physical fitness training compared with firehouse duties—a relative risk very similar to the risk of acute myocardial infarction in the general population (25,39). The much greater relative risk of a firefighter experiencing a cardiac event after fire suppression activities than during physical activity (PA) alone suggests that multiple stressors (i.e., exercise, heat, and psychological stress) may cause an exaggerated hemostatic response.

Firefighting involves heavy strenuous work while operating in a hostile environment and leads to activation of the sympathetic nervous system (2), increased cardiovascular and thermal strain (11,17), and dehydration (16). Catecholamines stimulate platelets and several coagulatory factors, suggesting

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that sympathetic stimulation may lead to a hypercoagulable state that increases the risk of thrombus formation (37,39). Firefighting has been shown to increase platelet number and activity (32).

An acute bout of strenuous exercise is associated with increased platelet number and activity, coagulatory factors, and fibrin formation, thus creating a procoagulatory state (1,8,22,27,38). However, this increase in coagulation is normally accompanied by a parallel increase in endogenous fibrinolysis, leading to balanced hemostatic state even during strenuous exercise in normal healthy adults (12,35,38). There is evidence, however, suggesting that coagulatory potential may remain elevated into recovery, whereas fibrinolysis quickly returns to baseline values after very strenuous physical work (14,23). The imbalance between coagulation and fibrinolysis may contribute to the increased susceptibility to sudden cardiac events in the period after strenuous activity. It is unknown how heat stress affects the restoration of hemostatic balance after exertion.

Although limited data suggest that heat stress can independently promote a procoagulatory state (3,21), very little work has been done to characterize the coagulatory and fibrinolytic responses to firefighting despite the very high rate of duty-related cardiovascular events associated with fire suppression activity (4,32). Burgess et al. (4) have recently reported an increase in coagulatory factors immediately after 12 min of firefighting but did not report changes in fibrinolytic factors.

Given (a) the importance of the hemostatic system in acute coronary syndromes, (b) the knowledge that sudden cardiac events are the leading cause of line-of-duty deaths among firefighters, (c) the evidence that among firefighters, fire suppression is associated with a greater relative risk of sudden cardiac event than physical fitness training, (d) the strong evidence of hemostatic disruption after strenuous PA, and (e) the recognition of multiple stressors faced by firefighters, several of which have been shown to alter hemostasis, it is important to examine how firefighting affects hemostasis and to investigate hemostatic changes during recovery from firefighting. Therefore, the purpose of this study was to evaluate the effect of strenuous firefighting activities on platelets and clotting and fibrinolytic activity and to document the extent to which these variables recovered 2 h after completion of the firefighting activity. We hypothesized that firefighting would lead to increased platelet number and function, increased coagulatory potential, and enhanced fibrinolysis. Furthermore, we hypothesized that platelet activity and coagulatory potential would remain elevated after 2 h of recovery while fibrinolytic potential would return to baseline levels.

METHODS

Subjects

Eighteen male firefighters (mean ± SD: age = 25.4 ± 4.8 yr; height = 1.82 ± 0.08 m; body mass = 85.8 ± 13.5 kg; body mass index (BMI) = 25.7 ± 2.9 $kg \cdot m^{-2}$) participated in testing. Participants were not specifically heat acclimated for our study, but all participants were firefighters who were familiar with performing drills (or live fire activity) in their protective equipment. Before testing, participants completed a health history questionnaire. Exclusion criteria included a history of atherosclerotic cardiovascular disease, medications for high blood pressure or cholesterol, or medications known to affect hemostasis (aspirin, acetaminophen, ibuprofen, cold, or asthma medication).

Study Design

Participants were fully informed of the purpose of the study and the requirements of participation, and they were given an opportunity to ask questions to the investigators. 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.

This study investigated alterations in platelet function, coagulatory variables, and fibrinolytic variables immediately after strenuous firefighting and after 2 h of recovery.

Participants performed prescribed live firefighting drills wearing full personal protective equipment, including self-contained breathing apparatus, in a training structure that contained live fires. The average weight of the personal protective equipment and self-contained breathing apparatus was approximately 20 kg. After the firefighting activity, firefighters participated in incident scene rehabilitation (approximately 20 min), where they removed their helmet, hood, gloves, and bunker coat and sat in a cool room (approximately 20°C). During rehabilitation, participants were provided with water *ad libitum* and cooled passively to reflect what is recommended at a fire scene.

After the rehabilitation period, participants changed into dry clothes and walked to an adjacent building (fire station) for a 2-h recovery period. During recovery, firefighters were engaged in classroom activities, reading, or viewing television to mimic what may occur at a fire station after a fire call.

The firefighting drills lasted 18 min (requiring approximately one cylinder of air for most participants) and consisted of nine 2-min periods of alternating rest and work. The work cycles included stair climbing, simulated forcible entry, a simulated search, and simulated hose advance. The live fire drills were completed on the second story of a training building that contained live fires. Throughout the firefighting drills, trained personnel controlled the temperature in the training structure by monitoring thermocouple readings and adding small fuel packages to the fire sets sequentially and controlling the ventilation conditions in the room. The temperatures at 1.2 m above the floor were maintained at roughly 70°C–82°C, and the floor temperatures were maintained at approximately 35°C–41°C. The prescribed firefighting activities (other than stair climbing) required participants to work almost exclusively in the space within 1 m of the floor.

Descriptive characteristics were obtained before participating in firefighting drills. Height was measured (to the nearest 0.01 m) using a stadiometer, and body mass was measured (to the nearest 0.5 kg) using a digital beam balance platform. BMI was calculated as the body mass in kilograms divided by the height in meters squared. A cholesterol profile from a finger stick sample (Cholestech, Hayward, CA) was also assessed before the live-fire training. An activity questionnaire was administered to determine activity status.

HR and Core Temperature

Body temperature was measured continuously throughout the protocol using a monitor and a silicone-coated gastrointestinal core temperature capsule (Mini Mitter, VitalSense; Philips Respironics, Bend, OR). Participants swallowed a small disposable core temperature sensor capsule the night before the study was conducted. HR was measured using an HR monitor (Vantage XL; Polar Electro, Inc., Lake Success, NY).

Blood Sampling and Laboratory Methods

Venous samples were drawn from the antecubital vein using a 21-gauge needle by a trained phlebotomist. Samples were drawn before firefighting activity and again immediately postfirefighting and 2 h postfirefighting. Hemoglobin and hematocrit were assessed via complete blood count, and plasma volume changes were calculated on the basis of the Greenleaf method (6).

Platelet number and function. Platelet count was assessed at a local clinic from venous whole blood as part of a complete blood count analysis using the electrical impedance method (with an instrument such as the COULTER® LH 700 Series; Beckman Coulter, Inc., Fullerton, CA).

Platelet function was assessed by epinephrine (EPI)-induced and adenosine 5′-diphosphate-induced platelet aggregability using a platelet function analyzer (PFA-100; Dade Behring, Deerfield, IL). Venous blood samples were collected in a Vacutainer containing 3.2% sodium citrate, maintained at room temperature, and analyzed within 2 h of collection. Blood was pipetted (800 μL) into the disposable cartridges and then aspirated under high shear rates (5000–6000 s^{-1}) through an aperture cut into the membrane coated with collagen and adenosine 5-diphosphate (ADP) and a membrane coated with collagen and EPI. Time to occlusion was reported.

Coagulatory and fibrinolytic variables. Blood samples were drawn into tubes containing 3.2% sodium citrate for measurements of all coagulation and fibrinolytic factors, except for the assessment of tissue plasminogen activator (t-PA) activity, in which those samples were drawn into Stabilyte tubes (Biopool, Wicklow, Ireland). All samples were centrifuged at 2300 rpm for 25 min at 4°C with the plasma removed and placed into aliquots, and stored at −70°C for later analysis. Plasminogen activator inhibitor (PAI-1) antigen, t-PA antigen, and tissue factor (TF) were analyzed in duplicate using an enzyme-linked immunosorbent assay kit (American Diagnostica, Stamford, CT). Antithrombin III (AT-III) was analyzed in duplicate using an Actichrome chromogenic activity kit (American Diagnostica). t-PA and PAI-1 activity were analyzed in duplicate using commercially available chromogenic substrate kits (DiaPharma Group, Inc., West Chester, OH). Factor VIII (FVIII) activity was analyzed in duplicate using a chromogenic assay kit (Chromogenix/DiaPharma Group, Inc., West Chester, OH). Activated partial thromboplastin time (aPTT) was analyzed using STA-PTT Automate 5 (Diagnostica Stago, Inc., Parsippany, NJ)

Data Analysis

Changes in plasma volume were calculated using the Greenleaf method (6). Blood variables that are expressed as a concentration were corrected for changes in plasma volume, and statistical analyses were performed for both the noncorrected and corrected values. Because the uncorrected values represent the relevant physiological state, these values are presented in the tables. However, when the analyses of corrected and uncorrected values produced different statistical results, these are noted in the text. Variables were checked for normal distribution, and those variables not normally distributed were log transformed (natural logarithm) before statistical analyses.

A one-way ANOVA with repeated measures was used to detect differences over time in hemostatic variables. After a significant *F*-ratio, *post hoc* tests were used to identify

TABLE 1. Classification by risk factors

Risk Factor	Frequency (*n* (%))
BMI[a] ($kg \cdot m^{-2}$)	
Underweight (<18.5)	0 (0%)
Normal weight (18.5−24.9)	7 (39%)
Overweight (25.0−29.9)	10 (56%)
Obesity (≥30)	1 (6%)
Total cholesterol[b] ($mg \cdot dL^{-1}$)	
Desirable (<200)	15 (82%)
Borderline high risk (200−239)	1 (6%)
High risk (≥240)	2 (12%)
LDL[b] ($mg \cdot dL^{-1}$)	
Optimal (<100)	9 (50%)
Near optimal/above optimal (100−129)	7 (39%)
Borderline high (130−159)	0 (0%)
High risk (160−189)	1 (6%)
Very high risk (≥190)	1 (6%)
HDL[b] ($mg \cdot dL^{-1}$)	
Low (<40)	6 (33%)
No category per reference (40−59)	9 (50%)
High (≥60)	3 (18%)
PA[c]	
Does not meet PA guidelines	13 (72%)
Meets/exceeds PA guidelines	5 (28%)
Smoker	
Yes	1 (6%)
No	17 (94%)
Family history of CVD	
Yes	3 (18%)
No	15 (82%)

[a]Classification based on established categories (29).
[b]Classification based on established criteria (28).
[c]PA guidelines: 30−60 $min \cdot d^{-1}$ (150 $min \cdot wk^{-1}$) of purposeful moderate exercise, or 20−60 $min \cdot d^{-1}$ (75 $min \cdot wk^{-1}$) of vigorous exercise, or a combination of moderate and vigorous exercise (13).

TABLE 2. Mean ± SD HR, core temperature, and change in plasma volume variables pre-, post-, and 2 h postfirefighting (N = 18)

Variables	Pre	Post	2 h Post
HR (beats per minute)	78 ± 11	162 ± 15[a]	67 ± 10[a,b]
Core temperature (°C)	37.14 ± 0.06	37.89 ± 0.09[a]	36.92 ± 0.05[a,b]
Change in plasma volume (%)	—	−3.99 ± 4.92	1.54 ± 4.92[b]

[a]Significantly different than Pre ($P < 0.05$).
[b]Significantly different than Post ($P < 0.05$).

specific time points that differed. Data were analyzed using SPSS version 18 (SPSS Inc., Chicago, IL). Descriptive data are expressed as mean ± SD. Statistical significance was set at $P < 0.05$ for all analyses.

RESULTS

Participants were relatively young, apparently healthy firefighters. The total mean cholesterol (174.8 ± 38.9 mg·dL^{-1}) was in the desirable range; the mean LDL cholesterol (107.4 ± 32.5 mg·dL^{-1}) was in the near optimal range; and mean HDL cholesterol (46.4 ± 12.6 mg·dL^{-1}) was in the average range. Table 1 presents data on the relative distribution of study participants in different categories based on cardiovascular disease risk factors. Seven firefighters were normal weight (18.5 ≤ BMI < 25), 10 firefighters were overweight (25 ≤ BMI < 30), and one firefighter was obese (BMI ≥30). Two of the participants had total cholesterol above 240 mg·dL^{-1}, one firefighter had LDL cholesterol above 190 mg·dL^{-1}, and six firefighters had HDL cholesterol less than 40 mg·dL^{-1}. Seventeen of the participants reported that they were nonsmokers, and one individual indicated that he was an "occasional" smoker. On the basis of activity recall data, 28% of participants met the current guidelines for PA. Three participants indicated that an immediate relative had died prematurely because of cardiovascular disease.

As seen in Table 2, firefighting activity resulted in an increased HR, reaching a mean peak HR of greater than 160 beats per minute by the end of the evolution. HR returned to below baseline values after 2 h of rest after firefighting activity, suggesting the prefirefighting values were not true resting values and represented an anticipatory response to activity. Core temperature increased by approximately 0.7°C after the 18 min of firefighting activity and returned to below baseline values after 2 h of recovery. Firefighting activity caused an average loss of plasma volume of approximately 4%. By 2 h postfirefighting, plasma volume was restored to baseline levels.

Platelet number and function. As seen in Table 3, platelet number increased significantly immediately postfirefighting ($P < 0.001$) and returned to baseline values 2 h postfirefighting. When the blood was exposed to collagen and EPI, platelet closure time decreased significantly immediately postfirefighting ($P < 0.05$) and then returned toward baseline, although it remained significantly lower than baseline at 2 h postfirefighting ($P < 0.05$). Platelet closure time when the blood was exposed to collagen and ADP decreased significantly after firefighting activity ($P = 0.018$) but was not different from baseline 2 h postfirefighting.

Coagulatory and fibrinolytic variables. Some blood samples were not analyzed because of hemolysis; thus, the number of samples is noted for each blood variable. aPTT decreased significantly immediately postfirefighting ($P = 0.025$) and remained significantly below baseline at 2 h postfirefighting ($P = 0.032$, Table 4). TF, uncorrected for changes in plasma volume, was significantly increased immediately postfirefighting compared with 2 h postfirefighting ($P = 0.018$). However, when corrected for changes in plasma volume, TF did not vary significantly with time, suggesting that hemoconcentration was primarily responsible for this change. FVIII increased significantly immediately postfirefighting ($P < 0.001$) and remained elevated at 2 h postfirefighting ($P = 0.002$). AT-III did not change significantly over time ($P = 0.055$), although there was a strong trend for AT-III to be higher after firefighting and in the recovery period.

Several fibrinolytic factors reflected a transient increase in fibrinolysis (Table 5). t-PA activity and antigen were elevated immediately postfirefighting ($P = 0.007$ and $P = 0.005$, respectively), but there were no significant differences between prevalues and 2 h postvalues ($P = 0.664$ and $P = 0.947$, respectively). There was also a significant effect of time on PAI-1 activity. *Post hoc* analysis revealed that PAI-1 activity was significantly lower both immediately postfirefighting and 2 h postfirefighting than prefirefighting ($P = 0.027$ and $P = 0.020$, respectively). PAI-1 antigen did not differ significantly between pre- and immediately postfirefighting but

TABLE 3. Mean ± SD platelet variables pre-, post-, and 2 h postfirefighting (N = 18)

Variables	Pre	Post	2 h Post
Platelet count	256.9 ± 61.7	300.8 ± 81.2[a]	254.4 ± 77.6[b]
EPI closure time (s)	117.1 ± 13.8	94.2 ± 20.5[a]	107.6 ± 18.6[a,b]
ADP closure time (s)	86.2 ± 16.2	73.9 ± 10.9[a]	79.7 ± 14.6

[a]Significantly different than Pre ($P < 0.05$).
[b]Significantly different than Post ($P < 0.05$).
ADP, adenosine 5-diphosphate induced.

TABLE 4. Mean ± SD coagulatory variables pre-, post-, and 2 h postfirefighting

Variables	Pre	Post	2 h Post
aPTT (s) (n = 13)	33.9 ± 4.93	31.9 ± 5.5[a]	31.8 ± 6.0[a]
TF (pg·mL^{-1}) (n = 17)	67.2 ± 36.7	72.4 ± 30.0	62.1 ± 32.9[b]
FVIII (IU·mL^{-1}) (n = 14)	90.1 ± 15.4	138.6 ± 36.0[a]	127.4 ± 43.2[a]
AT-III activity (%) (n = 17)	98.5 ± 8.7	102.7 ± 8.4	103.9 ± 8.0

[a]Significantly different than Pre ($P < 0.05$).
[b]Significantly different than Post ($P < 0.05$).

was significantly lower 2 h postfirefighting than prefirefighting ($P < 0.05$). When PAI-1 antigen levels were corrected for changes in plasma volume, there was no significant time effect.

DISCUSSION

We documented significant changes in platelet number and function, coagulation, and endogenous fibrinolysis immediately after live firefighting in a group of young, apparently healthy firefighters. The increase in HR and modest elevation in body temperature (0.7°C) in this study is consistent with results of other studies investigating short-term firefighting activity (17,32). These findings suggest that a short bout of live firefighting activities activates the coagulatory cascade and enhances fibrinolysis by 2 h postfirefighting activity, endogenous t-PA had returned to baseline although PAI-1 remained depressed, and platelet function and coagulation remained elevated. These data support the hypothesis that firefighters experience a hemostatic imbalance that is primarily prothrombotic during recovery from firefighting activity.

Epidemiological studies provide convincing evidence that strenuous physical exertion increases the risk of thrombotic events in individuals with underlying cardiovascular disease (25,36,39). Experimental evidence has shown that strenuous PA activates both coagulation and fibrinolysis. Acute exercise also leads to an increase in platelet number and platelet function that is dependent on the intensity of exercise (5,18,37). Wallén et al. (37) showed that maximal exercise (on a bicycle ergometer), mental stress, and EPI resulted in significant increases in platelet number and aggregation, which returned to baseline after 60 min of recovery. In our study, we found significant increase in platelet number and activation after a brief bout of strenuous, but intermittent, firefighting activity. The increase in platelet number was evident before corrections were made for changes in plasma volume and persisted after corrections were made, thus demonstrating that the increase in platelet number reflected more than just hemoconcentration. Catecholamine release as a result of sympathetic nervous system stimulation is likely involved in the release of platelets from the spleen and reticular tissue (18). The combination of heat stress and exercise results in increased sympathetic drive and catecholamine release compared with physical exertion alone (34). Although platelet number returned to baseline values by 2 h postfirefighting, platelet activation, evidenced by a shorter EPI closure time, remained significantly elevated 2 h postfirefighting. The discrepancy in the recovery data presented here compared with the data reported by Wallén et al. (37) raises the possibility that the combination of exercise and heat stress during live-fire fighting activities may lead to a more prolonged platelet activation than exercise alone.

Strenuous PA activates the coagulatory system in healthy subjects to a greater extent than moderate activity (5,14,38). Accompanying the increase in coagulatory variables, strenuous exercise also activates the fibrinolytic system as evidenced by elevated levels of t-PA activity and a decrease in PAI-1 activity, and these responses are dependent upon the intensity of the exercise (12,30,35,38,40). However, strenuous exercise may produce an imbalance in hemostasis. Strenuous exercise has been shown to increase thrombotic tendency, but this change is not evident after moderate exercise (5).

Although many authors maintain that there is a balanced hemostatic response (increased coagulation coupled with increased fibrinolysis) to exercise in healthy individuals (22,24,38), several reports have shown that coagulatory variables remain elevated 1−2 h after strenuous exercise, whereas fibrinolytic variables quickly return to baseline levels (14,23). Hegde et al. (14) reported that 30 min of running at 70% of $\dot{V}O_{2max}$ resulted in an increase in FVIII activity that was sustained during a 1-h recovery period. In contrast, the t-PA activity was significantly elevated postrunning but gradually declined during the 1-h recovery period. Lin et al. (23) have reported that elevated coagulatory potential (increased FVIII) persisted at 2 h and 6 h of recovery, whereas fibrinolytic activity fell sharply. The discrepancy in coagulatory and fibrinolytic potential during recovery from strenuous activity may account for an increased vulnerability to myocardial infarction after strenuous activity. In the present study, we found that markers of enhanced coagulation (elevated TF and FVIII activity) persisted after 2 h of recovery. In contrast, t-PA, a maker of fibrinolysis, had returned to baseline by 2 h postfirefighting. Furthermore, the shortened aPTT time at 2 h post-FF provides evidence of a shift toward a procoagulatory state after 2 h of recovery.

Firefighters are a unique occupational group who perform strenuous work under stressful conditions. Strenuous firefighting activities can lead to attainment of maximal HR (7,31), elevated core temperature (17,32), dehydration (16), decreased stroke volume (11,31), increased arterial stiffness (9), and alterations in myocardial function (11). Burgess et al. (4) reported that short-term (12 min) firefighting leads to an increase in FVIII, but these authors did not report on fibrinolytic factors. Our current data provide evidence of a procoagulatory state

TABLE 5. Mean ± SD fibrinolytic variables pre-, post-, and 2 h postfirefighting

Variables	Pre	Post	2 h Post
t-PA activity (IU·mL^{-1}) (n = 8)	0.59 ± 0.38	2.37 ± 1.49[a]	0.65 ± 0.33[b]
t-PA antigen (ng·mL^{-1}) (n = 18)	6.34 ± 3.46	11.8 ± 9.3[a]	6.24 ± 2.85[b]
PAI-1 activity (AU·mL^{-1}) (n = 14)	3.75 ± 3.04	2.63 ± 2.65[a]	2.29 ± 2.28[a]
PAI-1 antigen (ng·mL^{-1}) (n = 17)	24.4 ± 16.9	26.1 ± 15.2	21.8 ± 14.8[b]

[a] Significantly different than Pre ($P < 0.05$).
[b] Significantly different than Post ($P < 0.05$).

after firefighting activities and persisting for 2 h postfirefighting, which may contribute to an increased likelihood of a thrombotic event. These findings support the theory that alterations in blood clotting and fibrinolysis may play a mechanistic role in the increased risk of sudden cardiac events during and after firefighting. However, the relative contribution of multiple stressors (strenuous physical work, heat stress, emotional stress) that are encountered during firefighting on the hemostatic response is unclear.

It is well documented that coagulatory and fibrinolytic responses to exercise differ between healthy individuals and those with underlying cardiovascular disease or risk factors for cardiovascular disease (26,40). In the current study, we tested a relatively young group of firefighters and excluded firefighters who had a known history of cardiovascular disease and those who used medications known to alter hemostatic variables. Our participants had lower prevalence of cardiovascular disease risk factors than what has been reported in the US Fire Service (15,33), and thus, our results may underestimate hemostatic disruption that is evident in older firefighters with more risk factors or those who possess clinical or subclinical cardiovascular disease. Furthermore, immediately after firefighting, our participants were provided with planned cooling and rehydration (incident rehabilitation) and placed into a relatively controlled, relaxed environment to recover. Hence, our results may not represent the full magnitude of changes that occur under actual firefighting conditions in which firefighters work for longer periods or operate in more extreme environments, thus increasing core temperature to a greater degree. Another limitation of our study is the relatively small sample size. However, the findings of this study demonstrate considerable hemostatic disruption after short-term firefighting activity and provide a potential link between the physiological stress of firefighting and high number of sudden cardiac events during and after fire suppression activities. Additional research including more severe firefighting activity, individuals with a range of cardiovascular risk profiles, and a larger population of firefighters is needed to better elucidate the hemostatic responses that occur during and after firefighting activity.

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The authors have no conflicts of interest to declare.

The results of the present study do not constitute endorsement by the American College of Sports Medicine.

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