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 sympathethic nervous system (2), increased cardiovascular and thermal strain (11,17), and dehydration (16). Catecholamines stimulate platelets and several coagulatory factors, suggesting...