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ACUTE CARDIOVASCULAR EFFECTS OF FIREFIGHTING AND ACTIVE COOLING DURING REHABILITATION

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Abstract

Objectives—To determine the cardiovascular and hemostatic effects of fire suppression and post-exposure active cooling.

Methods—Forty-four firefighters were evaluated prior to and after a 12 minute live-fire drill. Next, 50 firefighters undergoing the same drill were randomized to post-fire forearm immersion in 10°C water or standard rehabilitation.

Results—In the first study, heart rate and core body temperature increased and serum C-reactive protein decreased but there were no significant changes in fibrinogen, sE-selectin or sL-selectin. The second study demonstrated an increase in blood coagulability, leukocyte count, factors VIII and X, cortisol and glucose, and a decrease in plasminogen and sP-selectin. Active cooling reduced mean core temperature, heart rate and leukocyte count.

Conclusions—Live-fire exposure increased core temperature, heart rate, coagulability and leukocyte count; all except coagulability were reduced by active cooling.

Keywords

firefighting; coagulation; active cooling

INTRODUCTION

Sudden cardiac incidents are the leading cause of firefighter line-of-duty deaths.[1] Fire suppression, which involves high levels of exertion and exposure to heat and potentially smoke, has the highest risk for cardiovascular death.[2] These exposures and resultant cardiovascular changes may be tolerated by healthy firefighters, but can have severe consequences in those with heart disease. In a study of firefighters dying from cardiac incidents while on the job the majority had underlying cardiovascular disease, although only one quarter had a prior diagnosis of coronary heart disease or other evidence of arterial-occlusive disease.[3]

Although the linkage between elevation of core body temperature and myocardial infarction remains to be firmly established, rise of core body temperature during exercise increases

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cardiac stress, including tachycardia.[4] In addition, live-fire exposure involving strenuous activity and increase in core body temperature increases platelet number and platelet aggregation as well as activation of coagulation factors in firefighters.[5] Forearm immersion in cold water more rapidly cools firefighters during rehabilitation than other cooling mechanisms tested,[6] although we are not aware of any studies evaluating the effectiveness of active cooling on blood markers of inflammation, endothelial and leukocyte activation or coagulability.

The purpose of this study was to evaluate the cardiovascular and hemostatic consequences of fire suppression and determine if active cooling during the firefighter rehabilitation period could reverse any identified adverse effects. We hypothesized that the combined exposure of strenuous exercise and increase in core temperature would cause a systemic inflammatory response, vascular cell dysfunction and an increase in blood coagulability, all known pathways leading to myocardial infarction. Specific biomarkers evaluated included C-reactive protein (CRP) and sP-selectin (inflammation), sE-selectin (endothelial activation/dysfunction), sL-selectin (leukocyte activation), troponin-I (myocardial injury), leukocytosis, neutrophil, platelet and monocyte activation and fibrinogen, Coagulation Index using thromboelastography (TEG) and factor VIII (hypercoagulable state).[7–17] We also hypothesized that active cooling would reduce core body temperature and mitigate any fire suppression-associated changes in these biomarkers.

METHODS

This research was approved by the University of Arizona Institutional Review Board. All subjects provided written consent prior to participation and completed a health, exercise and workplace history questionnaire. Firefighters had at least 48 hours with no fire smoke exposure prior to entering in the study. The live-fire drills were designed by the safety officers of the Tucson Fire Department (TFD) to approximate a fire-suppression response with interior entry and took place in the burn facility at the Tucson Public Safety Academy. All subjects wore full turn-out gear including self-contained breathing apparatus (SCBA) and were accompanied by a safety officer. Materials that approximated contents commonly found during the suppression of residential or commercial structural fires were used to create the fires within the burn facility, and additional heat was generated by combustion of wooden pallets and natural gas.

In the first part of the study, a self-matched observational study design was used to evaluate the acute effects of fire suppression in 44 firefighters. For the second part of the study, we conducted a parallel-group randomized trial of 50 firefighters to evaluate the effectiveness of aggressive cardiac rehabilitation using forearm cooling at the fire scene. Post-fire suppression blood collection time was reduced from 90 minutes in the first part of the study to 30 minutes in the second part of the study based on the main laboratory endpoints selected for analysis.

Exposures were 12 minutes in duration at temperatures measured 2.5 meters above floor level ranging from 178 to 297°C for the first study and 190 to 309°C for the second study. The firefighters carried out strenuous activities included in a circuit of work: 75 kg manikin drag, carrying two sections of 2.5 inch hose up and down stairs and maze room crawl, repeated as needed. The protocol of 12 minutes duration at the set temperatures was chosen as the maximum exposure that was considered safe by the firefighter safety officers, based on use of one bottle of compressed air. Following cessation of live-fire exposure, the firefighters went through the TFD standard rehabilitation process, involving sitting in the shade with turn-out jackets removed and turn-out pants either removed or pulled down and turned over the boots, which continued to be worn. They were provided cold water or sports

drinks, and blood pressure and heart rate were monitored. Following rehabilitation they stowed their gear and waited in the equipment bay of an adjacent fire station until their second blood draw, during which time they were provided additional liquids and food items.

Core body temperature was measured using an internal probe and heart rate using a chest belt unit (HQ, Inc. Palmetto, FL). For the first part of the study, the probe was swallowed early in the morning of the live-fire drill. For the second part of the study, the subjects swallowed their temperature probes prior to going to bed the night before the live-fire drill. In cases where the probe was no longer in their gastrointestinal tract when arriving at the study site, subjects swallowed a second probe approximately 30–60 minutes prior to starting the study. In both parts of the study, the heart rate monitoring unit was attached at the scene and core temperature and heart rate were recorded prior to entry, throughout the live-fire drill and for at least one hour following cessation of exposure.

In the first part of the study, blood was collected immediately prior to and 90 minutes after cessation of fire suppression using an 8.5 ml serum separator tube (SST), a 4.5 ml sodium citrate tube, a 4.0 ml EDTA tube and a 6.0 ml heparin tube by a qualified phlebotomist. The SST tube was allowed to clot for 30 minutes before centrifugation for 15 minutes at 1000 times gravity. The sodium citrate, EDTA and heparin anticoagulant tubes were centrifuged within 30 minutes of collection for 15 minutes at 1000 times gravity. The sodium citrate and heparin tubes were centrifuged for an additional 10 minutes at 10,000 times gravity. All serum/plasmas were aliquotted and stored at -20°C . Samples were transferred to the laboratory the same day and stored at -80°C until assayed.

Serum was analyzed by enzyme-linked immunosorbent assay (ELISA) for the concentration of sE-selectin, sL-selectin, and CRP (R&D Systems, Minneapolis, MN) and troponin I (ALPCO, Salem, NH). Citrated plasma was analyzed for fibrinogen (ALPCO, Salem, NH). Assay results in duplicate including standards and controls were obtained using an automated microplate reader, Model ELx808 (BioTek, Instruments, Inc., Winooski, VT). Concentration of samples was determined from standard curves using a four parameter algorithm for best fit (KC4, BioTEK, Winooski, VT).

For the second part of the study, TFD and Northwest Fire Department subjects were consented in firefighter teams of four, whenever possible. Fires were prepared and core temperature and heart rate were monitored and recorded as described above. Immediately following the live-fire drill, they were randomized within blocks of two or four to experimental (active cooling plus standard rehabilitation treatment) or control (standard rehabilitation treatment only) groups for the 15 minute cardiac rehabilitation period, so that for each four man team, there were two experimental and two control subjects. Active cooling employed a cooling chair (Kore Kooler® Rehab Chair, Morning Pride Manufacturing, Dayton, OH) with water filled wells into which the firefighters placed their forearms, and, based on their individual preferences, wrists and hands. The water temperature was kept as close to 10°C as possible by adding ice. Standard rehabilitation included resting in the shade and consuming cool beverages (water or a sports drink) ad libitum. Blood pressure was monitored twice during rehabilitation, once during the initial five minute period and once during the final five minute period. Blood collection was performed immediately before the live-fire drill and 15 minutes following rehabilitation (30 minutes following the end of fire suppression). Blood samples were processed immediately following collection, transported to the University of Arizona within two hours and frozen at -80°C until later analysis. For TEG and flow cytometric analyses, an additional sodium citrate anticoagulant tube was drawn for processing and transported to the Sarver Heart Center Applied Cardiovascular Research Laboratory for analysis within two hours of collection.

For each subject before and after the fire-fighting drill, whole blood samples were collected in sodium citrate and quickly processed for Thromboelastography (TEG 5000, Haemonetics Corp., Braintree, MA) analysis as described by us previously.[18] Briefly, quality control samples were run each day prior to running subject samples. For each assay, 20 µl of 0.2M calcium chloride was added to each TEG well prior to adding 340 µl of whole blood sample. The assay was initiated immediately and the pin torque v. time curves were recorded. All standard TEG parameters were evaluated and both native blood samples (citrate) as well as kaolin-activated blood samples were tested. The Coagulation Index (CI), a measure of whole blood coagulability, was calculated as recommended by the manufacturer.[18]

One milliliter of whole blood, collected in EDTA, was processed within one hour for complete blood count (CBC), flow cytometry and blood glucose testing. The CBC was determined using a haematology analyzer (Beckman Coulter, Ac.T 5diff, Brea CA). Flow cytometry (BD FACSCalibur, Becton Dickinson Biosciences, Franklin Lakes, NJ) was used to determine neutrophil activation by detection of the cell surface expression of neutrophil CD11b,[19] platelet activation as measured by CD62P expression[20] and platelet-monocyte conjugates as measured by co-expression of CD42b and CD45 in the monocyte-gated window.[21] Blood glucose was measured using a glucose analyzer (Accu-Check, Roche USA, Nutley, NJ).

In addition, six (3 pre-fire and 3 post-fire) 500 µl samples collected using an EDTA plasma tube were sent to the University of Arizona Proteomics Core Facility for proteomics analysis. Following depletion of high abundance proteins and isoelectric focusing separation into 10 fractions, each fraction was subjected to comparative analysis by matrix-assisted laser desorption ionization (MALDI) mass spectrometry. Multidimensional protein identification technology (MudPIT) and Scaffold software analysis (Proteome Software, Portland, OR), run in triplicate, were used to identify additional candidate biomarkers for validation using ELISA, focusing on proteins associated with inflammation and haemostasis. Based on these results, alpha-2-macroglobulin (A2M), factor IX, factor X and plasminogen were analyzed for changes in the second study in addition to the a priori selection of factor VIII, cortisol and sP-selectin.

Plasma (heparin) was analyzed for sP-selectin (R&D Systems, Minneapolis, MN). Plasma (citrate) was analyzed for factor VIII (DiaPharma, Lexington, MA), factors IX and X (Aniara, Mason, OH), heparin cofactor II (USCNK, Wuhan, P.R. China) and plasminogen (ALPCO, Salem, NH). Serum was analyzed for A2M and cortisol (ALPCO, Salem, NH). Assays in duplicate for standards, controls, and samples, measurement and calculations were conducted as described above.

Selected biomarkers were evaluated for normality, and appropriate transformations were employed to obtain approximate normality. For comparison of any continuous or near continuous variable between two time points on the same subject, generalized estimating equations (GEE) were used to account for potential grouping effect in the experiment. GEE was also used to correlate potential covariates with certain parameters of interest. Changes in the biomarkers constituted the continuous dependent variables for multiple regression modeling. To build the regression model, all potential covariates were first correlated with the response variable in univariate models. Then all covariates significant at the 0.10 level in the univariate models were entered into a backward elimination process, with the significance level set at 0.05. In both the comparison tests and the regression analysis appropriate transformation were performed for continuous variables heavily skewed.

For the active cooling study, Fisher's exact test was used to compare baseline categorical variables between the two study arms (cooling versus standard), and Wilcoxon rank-sum test

was used to compare baseline continuous variables. All tests were double-sided and at 0.05 level. GEE was used to study the effect of cooling and the effect of fire exposure, and to correlate potential covariates with certain parameters of interest.

RESULTS

For both studies, characteristics of the participating firefighters are listed in Table 1. The active cooling subjects did not differ significantly from the control subjects on any of the variables except hours of exercise per week. For the first study, the average temperature within the live-burn facility was $230.4 \pm 10.8^{\circ}\text{C}$. Ambient temperatures in the shade immediately following the live-fire drill averaged $28.2 \pm 2.5^{\circ}\text{C}$ with relative humidity $17.8 \pm 4.3\%$. For the second study, the average temperature within the live-burn facility was $233.1 \pm 16.8^{\circ}\text{C}$. Ambient temperatures in the shade during the rehabilitation period averaged $32.5 \pm 3.6^{\circ}\text{C}$ with relative humidity $32.6 \pm 18.4\%$.

For the first study, a typical core temperature graph collected from one of the firefighter subjects is illustrated in Figure 1. Live-fire exposure began an average of 39 ± 11 minutes from initiation of core temperature and heart rate monitoring, and, due to the donning of turn-out gear and warm ambient conditions, their core temperature began to rise before entering the burn facility. The core body temperature and heart rate rose during the live-fire exposure and peaked during the 10–15 minute period following cessation of exposure.

The changes in cardiovascular markers associated with live-fire drill exposure in the first study are listed in Table 2. The mean and maximum core body temperatures during the fire exposure period and the 15 minute period post-fire were significantly increased relative to the 15 minute period pre-fire. The mean heart rate during the fire exposure and the 15 minute period post-fire were significantly increased compared to the 15 minute period pre-fire. The second post-fire systolic blood pressure measurement was significantly decreased compared to the pre-fire value, while the first post-fire diastolic blood pressure measurement was significantly increased compared to the pre-fire value. CRP showed a significant post-fire decrease compared to the pre-fire value, while fibrinogen, sE-selectin and sL-selectin had no significant change. Pre- and post-exposure troponin I values were all below the limit of detection (data not shown).

Multiple regression analysis demonstrated that ever-smoking was associated with a reduction in mean (-0.20°C , $p=0.02$) and maximum (-0.26°C , $p=0.003$) core temperature during fire exposure as well as the maximum (-0.29°C , $p=0.01$) core temperature during the 15 minute period post-fire. Exercise was associated with an increase in these outcomes in multiple regression analysis; doubling the exercise hours per week was associated with an increase of 0.17 ($p=0.02$) and 0.29°C ($p=0.002$) in the mean and maximum temperature during fire and the maximum temperature during the 15 minute period post-fire, respectively. For the change in mean core temperature after fire, the only significant predictor was the mean heart rate during the 15 minute period pre-fire, which was inversely associated with temperature (data not shown).

In additional multiple regression analyses (data not shown), significant predictors for change in fibrinogen included triglycerides and pre-fire fibrinogen, both with negative association, and maximum heart rate during the 15 minute period before fire, which had a positive association. The only significant predictor for change in CRP was total cholesterol level, which had a positive association. Ever-smoking was positively associated with change in sL-selectin level, while VO_2 max was negatively associated. Both mean fire temperature and pre-fire sE-selectin were negatively associated with change in sE-selectin level.

For the second study, the effects of active cooling on core temperature and heart rate are illustrated in Figures 2a-d and listed in Table 3. The 95% confidence intervals for the treatment and control groups diverged approximately ten minutes after starting rehabilitation. The divergence increased during the last five minutes of active cooling and persisted for over 30 minutes following cessation of treatment. A similar pattern was observed with heart rate. Compared to standard rehabilitation, active cooling resulted in a 0.28 °C (95% CI 0.06-0.50) decrease in post-fire mean core temperature and a 19.4 (95% CI 12.0–26.8) beat per minute decrease in mean heart rate. Active cooling was associated with a reduced decline in the 2nd post-fire systolic diastolic blood pressures.

Regarding the TEG analysis (Table 3), the citrated native blood samples were significantly more coagulable post-exposure, with a decrease in clot firmness time (k) and an increase in maximum clot strength (MA) and the coagulation index (CI). The observed increase in whole blood coagulability was not mitigated by active cooling. For the TEG analysis of kaolin-activated blood, only k was significantly decreased following the live-fire drill (data not shown). For complete blood count variables, active cooling reduced the change in leukocyte (WBC) count. Although not affected by active cooling, neutrophil count increased following the live-fire drill while monocyte count and platelet count decreased following the live-fire drill. Live-fire drill exposure was associated with some but not all measures of neutrophil activation, which did not improve with cooling. There were no changes in platelet P-selectin expression or monocyte-platelet conjugate formation as measured by flow cytometry when comparing pre- and post-exposure values (data not shown). For other markers, plasminogen ($p = 0.013$) and sP-selectin ($p = 0.013$) decreased following the live-fire drill, while factors VIII and X, cortisol and glucose increased. Compared to controls, active cooling reduced A2M and factor IX.

Multiple regression analyses were carried out for each of four response variables: changes in mean and maximum body temperatures from pre-fire to in-fire measurements, and the corresponding changes from pre-fire to post-fire measurements. The following covariates were considered: intervention status, age, gender, race/ethnicity (whites versus others), smoking status (ever smoker and current smoker), mean and maximum fire temperatures, interaction of the intervention status and mean fire temperature, pre-fire measurements of mean and maximum body temperature, mean and maximum heart rate, height, weight, BMI, waist circumference, waist/height ratio, VO₂, total cholesterol, HDL, LDL, triglyceride, total cholesterol/HDL ratio, systolic and diastolic blood pressures, and exercise hours per week. In these regression analyses (data not shown), pre-fire mean temperature was negatively associated with all four temperature changes. Other covariates significant for one or more outcomes include waist circumference, waist/height ratio, LDL, and pre-fire max heart rate. Each 1 cm increase in waist circumference was associated with 0.014 °C decrease in change of in-fire max temperature ($p=0.0006$), while each 0.1 unit increase in waist/height ratio was association with 0.25 °C decrease in the change of post-fire mean temperature ($p=0.0002$). Each 1 mg/dl increase in LDL was associated with 0.002 °C decrease in change of the post-fire mean temperature ($p=0.023$).

DISCUSSION

The current study demonstrated elevated core temperature and heart rate associated with fire suppression activities, consistent with other published studies.[22–24] Fire suppression requires the performance of sustained strenuous activities while under the conditions of high heat stress. This combination of stressors has not been studied as well as the effects of exercise alone. Sustained heart-rate levels between 180 beats per minute less age in years[25, 26] and 220 beats per minute less age for cardiovascular-conditioned persons[27] are associated with excessive heat strain. The firefighters in our study demonstrated heart

rates in this range during their live-fire drill, although the strenuous activities performed almost certainly contributed to the elevation in heart rate.

In contrast to our initial hypothesis, we did not observe significant inflammatory or cell activation, dysfunction or injury response in this study, at least as evidenced by a decrease in CRP[28] and no changes in sL-selectin, sE-selectin or troponin I.[7–12] CRP is a well studied inflammatory marker and risk factor for incident cardiovascular disease.[13] sL-selectin is a marker of endothelial cell dysfunction that is decreased with cardiovascular disease.[14] sE-selectin helps mediate leukocytes adhesion to endothelial cells and subsequent transmigration; in patients with cardiovascular disease, increased levels are associated with future death.[15] Troponin I is a direct measure of myocardial cell injury.[16]

We did observe an increase in neutrophil count, suggestive of a mild inflammatory response, following the drill. In addition, we observed a significant neutrophil response to acute fMLP stimulation following the drill. The increased CD11b expression on neutrophils in response to fMLP suggests that the fire drill experience ‘primed’ but did not frankly activate the PMNs. The observations that neither platelet P-selectin nor platelet-monocyte conjugates were increased following the drill also indicate that the intervention did not cause a significant inflammatory response in this group of firefighters. Acute or chronic increases in conjugate formation are associated with increased myocardial ischemic events.[21]

Consistent with our initial hypothesis, live-fire drill exposure resulted in an increase in blood coagulability measured through TEG and factors VIII and X, although the increase in coagulation index did not reach the pathophysiologic range. The consistent increase in blood glucose and serum cortisol after the drill in both groups suggests a modest stress response and the hyperglycemia may have contributed to the observed increase in the coagulability.[20] These results support previous findings that firefighting can increase the propensity of blood to clot[5] and help define physiologic mechanisms for the increased risk of cardiac death associated with fire suppression activities.[2, 29] In addition, observed increases in factors VIII and X indicated at least a transient increased risk of thrombosis,[30, 31] the latter assuming conversion to factor Xa occurred. However, there were no changes in fibrinogen, a coagulation protein that in prospective studies is positively associated with development of cardiovascular disease[17]

Sustained, strenuous exercise (even at normal ambient temperatures) is known to cause a systemic inflammatory response as indicated by leukocytosis, a PMN CD11b increase and an increase in platelet-leukocyte conjugates.[32] However, the relative lack of responses observed in this study, despite the fire drill protocol inducing a stress response, may be explained by the subjects being physically fit and thus able to ‘handle’ the stress. Our findings of a positive association of ever-smoking and the negative association of VO₂ max with change in sL-selectin level also suggest that improved fitness may help mitigate inflammatory responses to fire-fighting. It is known that exercise and conditioning can blunt the pro-inflammatory and pro-coagulant effects of exercise, even in older individuals.[33] We believe the overall state of fitness and exercise training of the firefighter subjects blunted the pro-inflammatory response and allowed only a modest, though statistically significant increase in blood coagulability. Less conditioned individuals may have demonstrated more significant pro-thrombotic and pro-inflammatory responses and, if so, a greater pro-coagulant effect. Future research would clearly be required to test this notion.

The highest core temperatures were seen during the first 15 minutes following exit from the fire, supporting a minimum duration of rehabilitation of 10–15 minutes for exposures similar to the one we simulated. In the second study, the active cooling using forearm immersion in

cold water had the greatest effect on core temperature during the final 5 minutes of the 15 minute treatment period, suggesting that our a priori decision to treat for 15 minutes was a reasonable approach. Forearm and hand cooling appears to be more effective than other active cooling methods such as use of a misting fan or cooling vest or passive cooling techniques.[6, 34] However, other effective cooling mechanisms may be available.[35] The cooling chairs used for this study are commonly available in the fire service, although to our knowledge they are not often used. Active cooling may not be as beneficial in more temperate weather conditions,[36] but in our study, conducted outside with temperatures in the 32.5 °C range, it proved useful in decreasing core temperature and heart rate.

In addition to reduction in core temperature, cooling chair treatment resulted in a marked reduction in mean heart rate, close to 20 beats per minute, during the rehabilitation period. As with the reductions in core temperature, the effect became greater with longer duration of treatment. Rate-pressure product, which is calculated by multiplying heart rate by systolic blood pressure, is associated with myocardial oxygen consumption.[37] Since cooling treatment, as compared with standard treatment, was not associated with a significant difference in systolic blood pressure during the rehabilitation period, the cooling-treatment associated reduction in heart rate and proportional decrease in rate-pressure product could be beneficial in firefighters at risk of cardiac events.

Although our study was not designed to evaluate firefighter comfort, it was our observation that firefighters undergoing active cooling were in general more comfortable than those in the control group. In particular, sweating appeared to stop earlier in the active cooling group. However, some firefighters found the cold water uncomfortable. Water at 20 °C is also effective when used for active cooling, although the reduction in core temperature is lesser in magnitude.[38] To accommodate firefighter comfort, we feel it would be acceptable to allow variation in water temperature from 10–20 °C. At 10 °C, some ice placed in the arm wells remained floating, so this could help guide addition of further ice if temperature probes were not available.

From our review of the active cooling literature, there may be additional benefits of treatment. For firefighters wearing full turnout gear and SCBA with repeated exposures to heat and exercise, separated by brief rehabilitation periods, forearm immersion extended by 60% the time to exhaustion or excessive rise in core body temperature, as compared with passive cooling.[6] It is also important to note that many of the pro-thrombotic effects measured with live-fire drill exposure were not reversed by active cooling, although the decrease in Factor IX associated with active cooling could potentially help reduce risk of thrombosis.[39] Interestingly, active cooling was associated with a reduction in A2M, a protein that helps to limit inflammation.[40]

A surprising finding was that exercising a greater number of hours per week prior to the exposure was associated with a greater increase in core body temperature, and increased waist circumference and LDL cholesterol had the opposite effect, contrary to our a priori expectations. While not previously reported in the peer-reviewed literature regarding firefighters, we believe the explanation for this finding is that firefighters in better cardiovascular condition exerted themselves more than less fit firefighters. Our findings are consistent with previous studies involving the use of chemically protective clothing where highly fit individuals demonstrated a greater increase in core body temperature than moderately fit individuals.[41]

A limitation of the study was the use of a live-fire drill rather than actual fireground incidents. At actual incidents of sufficient size, firefighters may reenter the structure following rehabilitation, or be exposed to conditions resulting in greater increases in core

temperature. However, although not an objective measurement, many of the firefighter study participants remarked that the amount of exertion and heat exposure was similar to that experienced in more involved house fires. Our study did not correct for decreases in plasma volume associated with fluid losses during the live-fire exposures, changes which have been described elsewhere.[24] Inhalation of smoke components can cause adverse effects even at relatively low concentrations,[42] although the use of SCBA in this study should have minimized exposure.

In conclusion, live-fire drill exposure significantly increased whole blood coagulability but not markers of vascular injury, and active cooling reduced core temperature, heart rate and leukocytosis. The continued rise in core temperature following cessation of live-fire exposure supports a minimum of 10–15 minutes of rehabilitation. It is our current opinion that active cooling during firefighter rehabilitation is useful when environmental temperatures are elevated.

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Figure 1.
Typical graph of body temperature over elapsed time before, during and after fire suppression, study 1.

Figure 2.

Effect of active cooling in study 2 on: a) median core temperature 0–15 minutes post-fire, b) median core temperature 0–50 minutes post-fire, c) median heart rate 0–15 minutes post-fire, and d) median heart rate 0–50 minutes post-fire. Solid lines are controls (thin solid lines are 95% CI) and dashed lines are active cooling (thin dashed lines are 95% CI).

TABLE 1

Firefighter Participant Baseline Characteristics.

Characteristic	First study (n = 44)	Second study control (n = 25)	Second study active cooling (n = 25)
Gender (n, %)			
female	4 (9.1)	1 (4.0)	1 (4.0)
Race/ethnicity (n, %)			
White, non-Hispanic	35 (79.5)	19 (76.0)	19 (76.0)
Hispanic	8 (18.2)	5 (20.0)	6 (24.0)
Black, non-Hispanic	1 (2.3)	1 (4.0)	0 (0.0)
Age (years)			
median (min, max)	38.6 (22.8, 59.3)	32.7 (23.9, 51.4)	36.5 (26.3, 47.5)
Height (cm)			
median (min, max)	175.3 (162.6, 190.5)	177.8 (162.6, 188.0)	177.8 (165.1, 190.5)
Weight (kg)			
median (min, max)	84.8 (61.2, 119.7)	83.5 (70.3, 116.6)	82.6 (65.3, 119.7)
BMI (kg/m ²)			
median (min, max)	27.3 (20.5, 36.0)	28.0 (20.9, 33.0)	25.6 (23.3, 35.8)
Waist circumference (cm)			
median (min, max)	88.9 (66.0, 111.8)	88.9 (76.2, 111.8)	88.9 (81.3, 106.7)
VO ₂ max (ml/kg/min.)			
median (min, max)	56 (42, 74)	56 (45, 63)	57 (42, 80)
Total cholesterol (mmol/L)			
median (min, max)	4.53 (2.67, 6.03)	4.61 (3.00, 6.37)	4.97 (3.91, 7.80)
HDL (mmol/L)			
median (min, max)	1.32 (0.73, 2.49)	1.30 (0.73, 1.92)	1.27 (0.91, 2.49)
Total cholesterol/HDL			
median (min, max)	3.2 (2.0, 6.2)	3.7 (2.6, 6.7)	4.0 (2.2, 6.3)
LDL (mmol/L)			
median (min, max)	2.75 (1.50, 3.76)	2.93 (1.63, 4.79)	3.29 (2.10, 6.09)
Triglycerides (mmol/L)			
median (min, max)	0.86 (0.36, 2.54)	1.01 (0.42, 3.60)	1.07 (0.47, 3.70)
Resting pulse (per min)			
median (min, max)	59 (40, 88)	61 (45, 82)	58 (45, 80)
Systolic pressure (mmHg):			
median (min, max)	120 (98, 146)	120 (106, 134)	118 (100, 146)
Diastolic pressure (mmHg):			
median (min, max)	77 (60, 86)	78 (62, 88)	80 (62, 90)
Exercise hrs/week *			
median (min, max)	10.7 (1.5, 84) *	3.3 (0.5, 9.6) ‡	2.1 (1.0, 6.0) ‡
Ever-smoking (n, %)	12 (27.3%)	6 (24.0%)	3 (12.0%)

* Data were missing for three participants.

[†]Significantly different between control and cooling group: $p = 0.046$

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TABLE 2

Change in cardiovascular markers from pre-fire values among firefighters in the first study (n=44).

Marker	Pre-fire (15 min interval) mean (sd)	During fire mean (sd)	Post-fire (15 min interval) mean (sd)
Mean core temp (°C)	37.3 (0.5)	37.7 (0.4) ***	38.0 (0.6) ***
Max core temp. (°C)	37.5 (0.4)	38.1 (0.4) ***	38.4 (0.5) ***
Mean heart rate (/min)	107.6 (10.4)	152.1 (12.1) ***	122.1 (17.5) ***
Max heart rate (/min)	199.8 (28.3)	202.8 (22.3)	209.2 (29.5)
	Baseline mean (sd)	0–5 min. post-fire mean (sd)	10–15 min. post-fire mean (sd)
Systolic BP (mmHg)	121.1 (10.4)	124.2 (13.7)	117.7 (11.2) *
Diastolic BP (mmHg)	76.2 (8.6)	79.4 (10.2) **	76.0 (6.9)
	Pre-exposure mean (sd)	Post-exposure mean (sd)	
CRP (nmol/L)	10.62 (10.90)	10.25 (10.75)	
Fibrinogen (g/L)	1.86 (0.36)	1.82 (0.28)	
sE-selectin (ng/ml)	40.5 (12.6)	40.4 (11.4)	
sL-selectin (ng/ml)	1053 (220)	1066 (231)	

Compared to pre-fire interval,

* p-value between 0.05 and 0.01 (inclusive);

** p-value between 0.01 and 0.001 (inclusive);

*** p-value < 0.001

TABLE 3

Effects of active cooling on cardiovascular markers among firefighters in the second study (n=50), mean value (standard deviation).

Marker	Control pre	Control post	Cooling pre	Cooling post
Mean temp (°C) ###	37.5 (0.3)	38.5 (0.4)	37.4 (0.4)	38.2 (0.3)
Mean HR (bpm) ****##	112.2 (15.3)	124.1 (18.9)	108.4 (13.7)	104.7 (12.3)
Systolic blood pressure (mmHg)				
pre-fire	123.8 (8.8)		122.5 (8.5)	
0–5 min post-fire		126.8 (14.2)		123.9 (13.9)
10–15 min post-fire		112.5 (8.9)		115.6 (13.0)
Diastolic blood pressure (mmHg)				
pre-fire	76.8 (7.0)		76.2 (7.6)	
0–5 min post-fire		76.6 (10.5)		77.8 (8.3)
10–15 min post-fire		69.2 (11.1)		75.5 (9.1)
TEG Citrated				
R_min	8.8 (2.3)	8.2 (1.7)	8.6 (2.5)	8.5 (2.3)
k_min#	3.0 (1.3)	2.5 (0.5)	3.2 (1.4)	2.9 (1.2)
Angle	46.6 (14)	51.5 (9.9)	49.5 (10.9)	48.9 (14.4)
MA_mm#	58.3 (6.1)	60.6 (3.4)	56.8 (3.9)	58.2 (4.8)
CI#	1.4 (1.2)	1.8 (0.7)	1.1 (0.8)	1.4 (1)
CBC				
WBC count (10 ⁹ cells/L) *	6.06 (1.54)	6.30 (1.72)	5.93 (1.44)	5.84 (1.38)
Neutrophils (10 ⁹ cells/L) ##	2.79 (1.00)	3.12 (1.26)	2.59 (0.90)	2.74 (0.92)
Monocytes (10 ⁹ cells/L) #	0.59 (0.19)	0.54 (0.28)	0.59 (0.15)	0.54 (0.16)
Platelets (10 ⁹ cells/L) ##	0.25 (0.05)	0.26 (0.05)	0.23 (0.03)	0.24 (0.03)
Neutrophil activation				
TFI CD11b (Mean Channel × % Pos)	11.8 (8.9)	12 (9.5)	12.2 (9.8)	12.3 (10.8)
TFI CD11b (fMLP activated) #	26.3 (7.9)	30.0 (9.4)	26.5 (8.2)	29.1 (8.7)
Other				
Alpha-2-macroglobulin (mg/l) **	1863 (524)	1870 (577)	1880 (310)	1825 (319)
Plasminogen# (μmol/L)	1.39 (0.19)	1.34 (0.15)	1.34 (0.19)	1.28 (0.17)
sP-selectin (ng/ml) #	34.6 (14.7)	32.1 (13)	36.7 (11.7)	32.8 (11.3)
Cortisol (nmol/L) ###	315 (124)	519 (155)	348 (108)	502 (215)
Glucose (nmol/L) ###	4.72 (0.96)	5.71 (1.28)	4.54 (0.74)	5.53 (1.06)
Factor VIII (percent) ###	126.8 (90.1)	188 (138.7)	120 (46.2)	186.3 (135.9)
Factor IX (percent) **	79 (11.9)	78.6 (11.2)	80.2 (11.2)	78 (9.6)
Factor X (percent) ##	103.7 (14.4)	106 (13.8)	100.4 (11)	103.6 (12.9)

Comparison of change from baseline between controls and cooling subjects:

* p-value between 0.05 and 0.01 (inclusive);

**
p-value between 0.01 and 0.001 (inclusive);

p-value < 0.001

Comparison of post-fire with baseline (based on all subjects for markers not affected by cooling, and based on control subjects only for markers affected by cooling):

p-value between 0.05 and 0.01 (inclusive);

p-value between 0.01 and 0.001 (inclusive);

p-value < 0.001