

Firefighting and the Heart

Implications for Prevention

Article, see p XXX

Stefanos N. Kales, MD,
MPH
Denise L. Smith, PhD

AQ2

EDITORIAL

Firefighting is widely recognized as a hazardous occupation. In particular, fire scenes are unpredictable and dangerous environments characterized by loud noise, high temperatures, flames, smoke with gaseous and particulate toxicants, and potential structural instability of affected buildings, among other hazards. Accordingly, during fire suppression, firefighters are at risk for various injuries, including burns, trauma, and smoke inhalation. Although less intuitive, the strenuous physical activity, emotional stress, and environmental pollutants encountered while fighting a fire place considerable strain on the cardiovascular system, and each of these exposures can act alone or in concert to increase the risk of cardiovascular disease (CVD) events among susceptible individuals.^{1,2} The investigation of Hunter et al³ solidifies and expands a growing body of science elucidating the pathophysiologic mechanisms^{1,2,4} through which fire-suppression activities markedly increase the risk of CVD events among firefighters: 10- to >100-fold greater risk compared with nonemergency fire department duties.^{2,5}

CARDIOVASCULAR STRAIN OF FIRE-SUPPRESSION ACTIVITIES

We use the term “fire-suppression activities” broadly here to refer to firefighting with the use of hoses and application of water, as well as to refer to forcible entry, building ventilation, and search and rescue operations at a fire scene, while distinguishing this class of duty from additional jobs performed by firefighters, such as medical and other calls. Structural fire-suppression activities are usually preceded by an alarm response, which produces an adrenergic “fight-or-flight” reaction with prominent sympathetic arousal.^{1,2,4} The increases in heart rate and blood pressure continue until scene arrival, where suppressing a fire requires significant aerobic effort (stair and ladder climbing), anaerobic power (forcible entry, search and rescue operations), and static exertion (heavy materials handling, cutting and chopping to ventilate the building, and advancing charged hoselines). The use of heavy (>25 kg), encapsulating personal protective equipment and the extreme heat of the fire add further to cardiometabolic demands and can also lead to hyperthermia and dehydration.^{1,2,4} As confirmed by the present and previous studies, all of these factors can produce further increases in heart rate and blood pressure, which are accompanied by alterations in blood flow, vascular shear stress, and electrolytes; decreased plasma volume; increased blood viscosity; a procoagulatory state^{3,4}; and, as documented in the present study, low-level myocardial injury and ischemia.³ While Hunter et al³ emphasize that this state promotes myocardial infarction, it is crucial to recognize that the same constellation of cardiovascular stressors is also arrhythmogenic (Figure). In fact, the autopsies of firefighters succumbing to on-duty

The opinions in this article are not necessarily those of the editors or of the American Heart Association.

Correspondence to: Stefanos N. Kales, MD, MPH, The Cambridge Health Alliance—Occupational Medicine, 1493 Cambridge St, Macht Bldg, Ste 427, Cambridge, MA 02139. E-mail skales@hsph.harvard.edu

Sources of Funding, see page XXX

Key Words: Editorials
■ cardiomegaly ■ cardiovascular disease ■ firefighting ■ sudden cardiac death

© 2017 American Heart Association, Inc.

AQ3

F1

AQ4

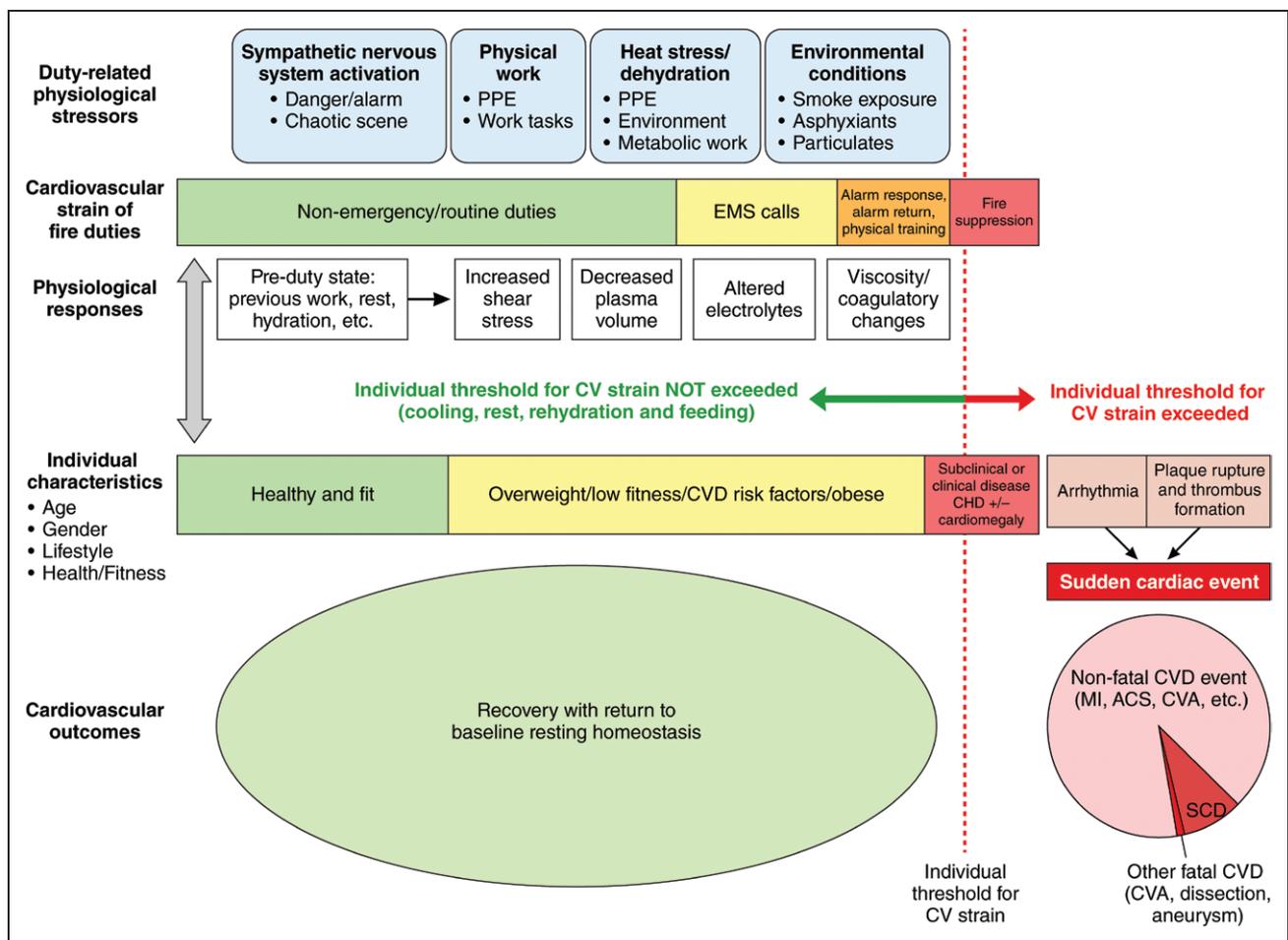


Figure. Both the average cardiovascular strain of fire duties and individual firefighter susceptibility increase.

In the vast majority of cases, even during fires where cardiovascular strain is quite high, firefighters with or without underlying health issues fully recover to their baseline states. Infrequently, however, the cardiovascular strain threshold of a vulnerable firefighter is exceeded, and through 1 or more of the mechanisms depicted, an acute CVD event is triggered. For every death caused by SCD, ≈17 to 25 nonfatal events occur. CV indicates cardiovascular; CVA, cerebrovascular accident; CVD, cardiovascular disease; EMS, emergency medical services; PPE, personal protective equipment; and SCD, sudden cardiac death.

sudden cardiac death (SCD) strongly suggest that most of these fatalities result from arrhythmias.^{6,7}

CVD EPIDEMIOLOGY IN FIREFIGHTERS

One must look beyond fighting fires to fully understand why SCD is the leading cause of duty-related fatalities among US firefighters, causing ≈45% of all job-related deaths.^{2,5} Although ≈33% of SCD events occur during fire suppression and the relative risk is highest during fires, >60% of firefighter SCD occurs across a variety of other duties. For example, CVD event risk is increased 5- to 7-fold during the sympathetic arousal of the alarm response.^{1,2,5} Additionally, the odds of SCD are also increased during physical training activities (including simulated fires, but also simple exercise and other non-fire training drills). Further proof that heavy protective equipment, heat stress, dehydration, and air pollution are not required to trigger CVD events in public safety

workers comes from law enforcement, where pursuits of and altercations with suspects increase the relative risk of SCD 30- to 70-fold compared with routine duties.⁸ Moreover, despite the marked alterations in physiological function that firefighters experience during fire suppression, it is highly unusual to observe a CVD event in a healthy and fit firefighter.^{2,4} The common denominator of occupational CVD events, including SCD across occupations and different types of duties, is an interaction between individual vulnerability and cardiovascular strain. When a public safety worker with underlying CVD (structural or coronary heart disease) is unable to tolerate a given load of cardiovascular stressors or threshold of cardiovascular strain, pathophysiologic changes precipitate or trigger a CVD event (Figure).

Consistent with the previous theoretical framework, case-control studies among firefighters demonstrate that SCD fatalities and CVD retirements have statistically higher burdens of classic CVD risk factors (eg, smok-

ing, hypertension, and obesity) than healthy controls.^{6,7,9} Furthermore, on-duty CVD events occur almost exclusively in firefighters with previously diagnosed CVD (20% to 30% of all events), firefighters with underlying (often subclinical) structural heart disease, firefighters with a clustering of traditional CVD risk factors and subclinical coronary heart disease, or persons belonging to >1 of the 3 previous categories.^{2,7,9} Autopsies of firefighter SCD victims typically show varying degrees of coronary atherosclerosis usually accompanied by left ventricular hypertrophy/cardiomegaly.^{6,7} The major role of cardiac enlargement in fire service SCD is highlighted by numerous cases where coronary heart disease is not present or the degree of coronary occlusion is ≤40% to 50% and no evidence of coronary thrombus or plaque rupture is present. Compared with firefighters dying of traumatic causes, the hearts of firefighters dying of SCD are significantly heavier, and cardiomegaly (heart weight >450 g) conveys a 5-fold increase in the risk of SCD.⁷ Older age also increases the risk of SCD and CVD retirement among public safety workers in a dose-response fashion, and this risk increases sharply beyond 60 years of age.^{5,8,9} Depending on the duty performed, the risks of job-related SCD among firefighters ≥60 years of age range from ≈4- to 18-fold greater than that of colleagues 40 to 49 years of age.⁵

Clinical research is also informative. Obesity among firefighters is associated with lower aerobic fitness, the clustering of various CVD risk factors, and a higher risk of cardiac enlargement.^{2,10,11} Firefighters with low physical fitness (≤10 METS) have a significantly and several-fold higher risk of metabolic syndrome, as well as EKG abnormalities and clinically abnormal heart rate recovery associated with maximal treadmill tests, compared with firefighters with excellent fitness (>14 METS).^{12,13} In the study of Hunter et al,³ 20 minutes of fire simulation training resulted in significant increases in cardiac troponin and more EKG-detected ischemia in carefully selected healthy firefighters. Repeating their experiments in firefighters who smoke and have health problems, low fitness, or even previously diagnosed heart disease would raise safety concerns. Yet it is known that many firefighters who respond on a regular basis to real fires and other stressful emergencies lack recommended fitness levels and have a high prevalence of **risk CVD factors**.²

IMPLICATIONS OF RESEARCH FOR REDUCING CVD IN THE FIRE SERVICE

Based on their study, Hunter et al³ suggest measures to decrease the cardiovascular risks of fire simulation training, including limiting the duration of exposure, active cooling, and effective rehydration. Given the confirmation that fire-suppression duties result in prothrombotic changes, more widespread use of low-dose aspirin

among middle-age firefighters should also be considered.¹⁴ However, such approaches, although reasonable, would do little to reduce the overall burden of CVD in the fire service. Based on all of the established data from physiological, epidemiologic, and clinical studies of representative fire service populations as summarized earlier, we reiterate a series of appropriate preventive measures,¹⁵ some of which are gradually being adopted by major US fire organizations. These measures include banning smoking and tobacco products in fire services, commonsense fitness and obesity standards, wellness programs that promote exercise and healthy diets, annual medical evaluations for all firefighters, and considering mandatory retirement from active firefighting (fire-suppression and other strenuous duties) at 60 years of age.

Hopefully, the impressive results of Hunter et al³ will persuade clinicians that firefighting is uniquely stressful on the cardiovascular system and encourage practitioners to aggressively evaluate and treat CVD risk factors in these invaluable public servants and, when indicated, perform additional studies (such as exercise stress testing, coronary artery calcium scans, or echocardiography) to detect subclinical atherosclerosis or cardiac enlargement. Finally, given the markedly higher risk (≤15-fold after covariate adjustment) of SCD among firefighters with established coronary heart disease or other structural heart disease, clinicians should consider recommending that patients with such a profile refrain from participating in strenuous emergency duties such as fire suppression and fire simulation.²

SOURCES OF FUNDING

This work was supported in part by US Department of Homeland Security, Federal Emergency Management Agency Assistance to Firefighters grant program awards EMW-2014-FP-00612 and EMW-2013-FP-00749.

DISCLOSURES

Dr Kales reports serving as a paid expert witness, an independent medical examiner, or both in workers' compensation and disability cases, including cases involving firefighters. Dr Smith reports serving as a consultant in cases involving medical evaluations and firefighter fatalities.

AFFILIATIONS

From Environmental & Occupational Medicine & Epidemiology, **Harvard School** of Public Health, Boston, MA (S.N.K.); Occupational Medicine, The Cambridge Health Alliance/Harvard Medical School, Cambridge, MA (S.N.K.); Health and Exercise Sciences, Skidmore College, Saratoga Springs, NY (D.L.S.); and University of Illinois Fire Service Institute, Champaign (D.L.S.).

FOOTNOTES

Circulation is available at <http://circ.ahajournals.org>.

REFERENCES

- Smith DL, Barr DA, Kales SN. Extreme sacrifice: sudden cardiac death in the US Fire Service. *Extrem Physiol Med*. 2013;2:6. doi: 10.1186/2046-7648-2-6.
- Soteriades ES, Smith DL, Tsismenakis AJ, Baur DM, Kales SN. Cardiovascular disease in US firefighters: a systematic review. *Cardiol Rev*. 2011;19:202–215. doi: 10.1097/CRD.0b013e318215c105.
- Hunter AL, Shah AVS, Langrish JP, Ratis JB, Lucking AJ, Brittan M, Venkatasubramanian S, Stables CL, Stelzle D, Marshall J, Graveling R, Flapan AD, Newby DE, Mills NL. Fire simulation and cardiovascular health in firefighters. *Circulation*. 2017;135:xx–xx.
- Smith DL, DeBlois JP, Kales SN, Horn GP. Cardiovascular strain of firefighting and the risk of sudden cardiac events. *Exerc Sport Sci Rev*. 2016;44:90–97. doi: 10.1249/JES.0000000000000081.
- Kales SN, Soteriades ES, Christophi CA, Christiani DC. Emergency duties and deaths from heart disease among firefighters in the United States. *N Engl J Med*. 2007;356:1207–1215. doi: 10.1056/NEJMoa060357.
- Geibe JR, Holder J, Peeples L, Kinney AM, Burress JW, Kales SN. Predictors of on-duty coronary events in male firefighters in the United States. *Am J Cardiol*. 2008;101:585–589. doi: 10.1016/j.amjcard.2007.10.017.
- Yang J, Teehan D, Farioli A, Baur DM, Smith D, Kales SN. Sudden cardiac death among firefighters ≤45 years of age in the United States. *Am J Cardiol*. 2013;112:1962–1967. doi: 10.1016/j.amjcard.2013.08.029.
- Varvarigou V, Farioli A, Korre M, Sato S, Dahabreh IJ, Kales SN. Law enforcement duties and sudden cardiac death among police officers in United States: case distribution study. *BMJ*. 2014;349:g6534.
- Holder JD, Stallings LA, Peeples L, Burress JW, Kales SN. Firefighter heart presumption retirements in Massachusetts 1997-2004. *J Occup Environ Med*. 2006;48:1047–1053. doi: 10.1097/O1.jom.0000235909.31632.46.
- Korre M, Sampani K, Porto LGG, Farioli A, Yang J, Christiani DC, Christophi CA, Lombardi DA, Kovacs RJ, Mastouri R, Abbasi S, Steigner M, Moffatt S, Smith DL, Kales SN. Cardiac enlargement in US firefighters: prevalence estimates by echocardiography, cardiac magnetic resonance and autopsies. *J Clin Exp Cardiol*. 2016;7:459.
- Korre M, Porto LG, Farioli A, Yang J, Christiani DC, Christophi CA, Lombardi DA, Kovacs RJ, Mastouri R, Abbasi S, Steigner M, Moffatt S, Smith D, Kales SN. Effect of body mass index on left ventricular mass in career male firefighters. *Am J Cardiol*. 2016;118:1769–1773. doi: 10.1016/j.amjcard.2016.08.058.
- Baur DM, Christophi CA, Kales SN. Metabolic syndrome is inversely related to cardiorespiratory fitness in male career firefighters. *J Strength Cond Res*. 2012;26:2331–2337. doi: 10.1519/JSC.0b013e31823e9b19.
- Baur DM, Leiba A, Christophi CA, Kales SN. Low fitness is associated with exercise abnormalities among asymptomatic firefighters. *Occup Med (Lond)*. 2012;62:566–569. doi: 10.1093/occmed/kqs112.
- Smith DL, Horn GP, Woods J, Ploutz-Snyder R, Fernhall B. Effect of aspirin supplementation on hemostatic responses in firefighters aged 40 to 60 years. *Am J Cardiol*. 2016;118:275–280. doi: 10.1016/j.amjcard.2016.04.032.
- Kales SN, Smith DL. Sudden cardiac death in the fire service. *Occup Med (Lond)*. 2014;64:228–230. doi: 10.1093/occmed/kqu057.

AUTHOR QUERIES

Authors please note: Authors are responsible for any page charges as outlined in the acceptance letter or as indicated on the Instructions for Authors (<http://circ.ahajournals.org/site/misc/ifora.xhtml>). Unless you have selected open access for your article, or it is otherwise noted on the acceptance letter, page charges are as follows: \$70 per black and white page (print articles only) or \$35 per page (online-only articles only). For each color page (print only), please add \$653/page. If there are any concerns regarding these charges, these should be addressed within 48 hours of receiving the s-proof. Author(s) will be invoiced for all color and page charges post publication. If you have selected open access for your article, please refer to details in the queries below.

AUTHOR PLEASE ANSWER ALL QUERIES

- AQ1—Please turn to pages 2 and 3 of your proof and review the author’s name and running head, which appear in the upper left-hand and right-hand margins. Please check the author’s name in the upper left-hand margin. The running head, in the upper-right margin must be 50 or fewer characters in length, including spaces and punctuation. If your original short title was longer than 50 characters, we may have shortened it. Please modify if necessary (but observe our length guidelines).
- AQ2—Please confirm that all authors are included in the correct order in the byline and that all names are spelled correctly, including special characters, accents, middle initials, and degrees, if applicable. For indexing purposes, confirm author names have been correctly identified as given names (blue) and surnames (red). Color in the byline will not appear on the final published version. Note that journal style discourages listing American honorary degrees in the byline; such degrees are deleted during editing.
- AQ3—Key words have been inserted and edited to match the US National Library of Medicine's Medical Subject Headings (<http://www.nlm.nih.gov/mesh/MBrowser.html>). If they need modification, please refer to these lists and limit the total number of key words to 7.
- AQ4—If you have color in your proof, please indicate whether you approve the color charge by returning the color agreement with your corrections. The color agreement can be found at “<http://links.lww.com/circ/A10>” . The rate is \$653 per 1 printed color page. If you prefer black and white figures, please indicate so on your proof corrections. Please note: There is no online-only color option for Circulation articles. If your article is black and white in print, the online version will also be black and white. If you have any questions, please contact “<mailto:circulationjournal@lww.com>”
- AQ5—Please carefully review any Acknowledgments, Sources of Funding, and/or Disclosures listed at the end of the manuscript (before the References), and confirm that they are accurate and complete for all authors.

AQ6—Please confirm that all authors' institutional affiliations (including city/state/country locations) are correct as shown in the affiliations footnote.