

Ultrafine Particle Exposure During Fire Suppression—Is It an Important Contributory Factor for Coronary Heart Disease in Firefighters?

C. Stuart Baxter, PhD, Clara Sue Ross, MD, JD, Thomas Fabian, PhD, Jacob L. Borgerson, PhD, Jamila Shawon, MS, Pravinray D. Gandhi, PhD, James M. Dalton, MArch, and James E. Lockey, MD, MS

Objective: Coronary heart disease (CHD) is the primary cause of death among US firefighters during fire suppression. In other populations, exposure to respirable particles, including ultrafine particles, has been widely implicated as a risk factor for CHD. This study is the first to report detailed characterization of respirable particles released by combustion of an automobile and model residential structures under firefighter exposure conditions. **Methods:** Characterization was performed when feasible during knockdown and routinely during overhaul. **Results:** Ultrafines accounted for >70% of particles in all fire suppression stages, occurring in concentrations exceeding background by factors between 2 (automobile) and 400 (bedroom), consistent among all structures. **Conclusions:** Exposure to ultrafine particles during fire suppression should be considered a potential contributing factor for CHD in firefighters. Of major significance is their predominance during overhaul, where firefighters frequently remove respiratory protection.

Fatal coronary heart events such as sudden death, myocardial infarction, and fatal arrhythmia are responsible for 45% of ~100 annual US firefighter deaths while on duty.^{1,2} During fire suppression, which includes knockdown to extinguish the fire or limit its growth, followed by overhaul to prevent reignition of partially burned material, US firefighter deaths from acute cardiovascular events occur at a rate of 10 to 100 times higher than during nonemergency duties.³ US firefighters have also been reported to experience an increased incidence of unspecified nonfatal CHD events such as chest pain at increased rates during alarm response and fire suppression compared with nonemergency activities.^{4,5}

Risk factors for CHD in firefighters may be personal and work related or both. Personal risk factors include hypertension, obesity, increased serum cholesterol and triglyceride levels, and smoking.^{6–10} Potential work-related risk factors include excessive noise levels; physical, heat, and psychological stress; dehydration; extended work shifts; and exposure to chemical asphyxiants, such

From the Department of Environmental Health (Dr Baxter, Dr Ross, Dr Lockey) University of Cincinnati Academic Medical Center, Cincinnati, Ohio; Underwriters Laboratories Inc (Dr Fabian, Dr Borgerson, Dr Gandhi, Ms Shawon), Northbrook, Ill; and Chicago Fire Department (Mr Dalton), Chicago, Ill.

Dr Lockey reports serving as a paid expert witness or independent medical examiner or both in workers' compensation and disability cases, including cases involving firefighters.

The JOEM Editorial Board and planners have no financial interest related to this research.

Address correspondence to: C. Stuart Baxter, PhD, Department of Environmental Health, Kettering Laboratory, University of Cincinnati Academic Medical Center, 3223 Eden Avenue, PO Box 670056, Cincinnati, OH 45267-0056; E-mail: c.stuart.baxter@uc.edu.

The contents are the responsibility of the authors and do not necessarily reflect the views of the Department of Homeland Security.

Copyright © 2010 by American College of Occupational and Environmental Medicine

DOI: 10.1097/JOM.0b013e3181ed2c6e

Learning Objectives

- Discuss the contribution of coronary heart disease to on-duty deaths in firefighters, including the personal and work-related contributors to this risk.
- Outline the methods used in this laboratory study to assess potential exposure to ultrafine and other particles during fire suppression activities.
- Summarize the authors' conclusions as to whether ultrafine particles can be considered "an important contributory factor" to the risk of coronary events in firefighters.

as carbon monoxide, hydrogen cyanide, and hydrogen sulfide. Any of these factors could precipitate an acute cardiovascular event, particularly in individuals with underlying cardiovascular disease.¹¹ A role for workplace factors was suggested by an altered circadian distribution of on-duty deaths from CHD compared with the general population, with the highest odds ratio occurring during fire suppression activities.³

An additional occupational cardiovascular risk factor that is receiving increasing attention is exposure to respirable particles, including those in the ultrafine range (diameters <0.1 μm), which have been suggested to be capable of inducing remote cardiovascular events after respiratory deposition by several mechanisms.¹² Ultrafine particles seem to be the most physiologically and toxicologically active among these particles¹³ and have been detected in increased number densities from several industrial processes.^{14–17} Combustion of fossil fuels also results in increased number densities of these particles in urban air and has led to several attempts to correlate these environmental levels with cardiovascular disease.^{18–20} In recent clinical studies, these correlations have been supported by findings of a variety of changes in cardiovascular parameters in healthy volunteers.^{20,21} A recent CHD study examining the effect of ultrafine diesel exhaust particle exposure in construction workers showed analogous adverse effects.²²

In this study, we characterized the number densities and size distributions of particles, including those in the ultrafine range, produced during the combustion of various model residential rooms and structures in a large laboratory setting, to determine the potential for firefighter exposure to these materials.

METHODS

Scenarios

Seven fire tests were conducted in the large-scale fire test laboratory at Underwriters Laboratories. The seven tests represented typical residential building and automobile fire scenarios to which firefighters routinely respond.²³ The six residential building scenarios included a living room, bedroom, kitchen, attic structure, and composite and traditional wood deck structures. The automobile scenario involved a passenger compartment fire. Smoke parti-

cles were characterized during fire service response activities as summarized in Table 1.

The smoke particle sampling probe was positioned at a standing face-level of 1.68 m (5 ft 6 in) in all scenarios (Fig. 1). For the room scenarios (ie, attic, bedroom, kitchen, and living room), the probe was placed in the interior of the room, centered on the

right wall. For the passenger compartment fire scenario, the probe was centered above the driver's window.

The attic test structure consisted of a pitched roof attic over a room measuring 3.7 × 4.3 m (12 × 14 ft) in size and 2.4 m (8 ft) in height. The structure had a US standard 32-inch wide interior doorway (0.8 × 2 m) and a 0.9 × 0.6 m (36 × 24 in) size sliding window. The attic was filled with an assortment of typical items such as toys, an artificial Christmas tree, books, clothing, coffee maker, drapes, cable, PVC pipe, infant mattress, newspaper, cardboard boxes, books, and stuffed animals, reflecting its common storage function.

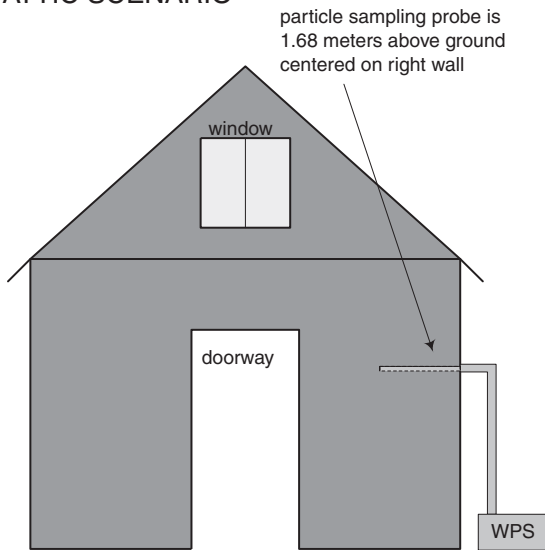
TABLE 1. Particle Sampling Methodology for Fire Test Scenarios

Scenario	Particle Sampling Period
Attic	Ignition through overhaul
Bedroom	Overhaul
Kitchen	Overhaul
Living room	Overhaul
Composite deck	Overhaul
Wood deck	Overhaul
Passenger compartment	Overhaul

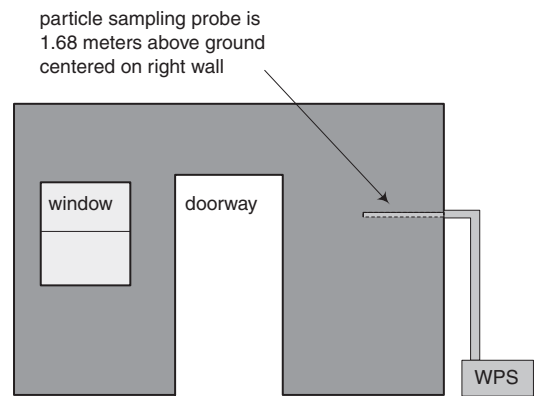
Smoke Particle Measurements

Number density distributions for smoke particles ranging from 0.01 to 10 μm in diameter were determined using a Wide Range Particle Spectrometer M1000 × P (MSP Corporation, Shoreview, MN). Potential compromise of instrument function due to clogging of the sampling port or line by water applied to fires during the knockdown phase restricted smoke particle measurements to the overhaul phase for all building scenarios except the

ATTIC SCENARIO

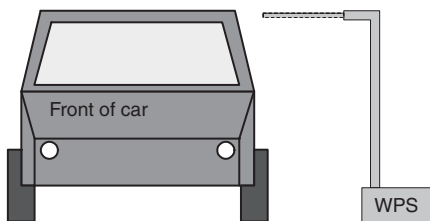


ROOM SCENARIO



AUTOMOBILE SCENARIO

particle sampling probe is 1.68 meters above ground centered on front window for the passenger compartment fire



DECK SCENARIO

particle sampling probe is 1.68 meters above deck on front side of wall

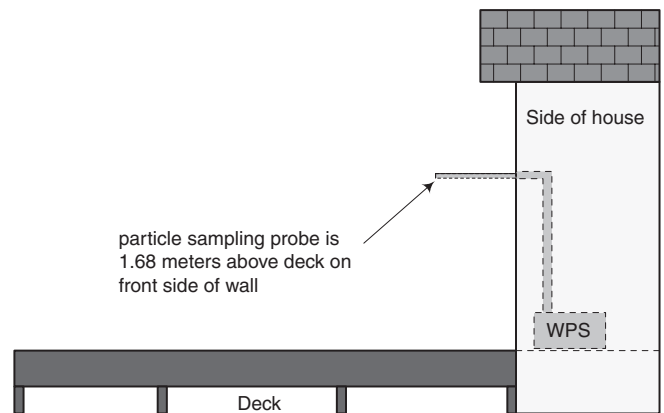


FIGURE 1. Wide range particle spectrometer probe placement for fire test scenarios.

TABLE 2. Particle Number Density and Size Distribution During Overhaul

Scenario	Average Number Density (Particles/cm ³)*	Relative Number Density†	Fraction of Particles Within Size Ranges μm*‡			
			0.01–0.11	0.11–1.0	1.0–2.5	2.5–10
Attic	1.20 ± 1.01 × 10 ⁵	83.37	0.76 ± 0.10	0.24 ± 10	0.00 ± 0.00	0.00 ± 0.00
Bedroom	2.11 ± 3.26 × 10 ⁶	372.92	0.84 ± 0.29	0.16 ± 0.29	0.00 ± 0.00	0.00 ± 0.00
Kitchen	6.34 ± 8.26 × 10 ⁴	200.49	0.79 ± 0.11	0.21 ± 0.11	0.00 ± 0.00	0.00 ± 0.00
Living room	5.92 ± 2.82 × 10 ⁵	70.64	0.83 ± 0.07	0.16 ± 0.07	0.00 ± 0.00	0.00 ± 0.00
Composite deck	4.55 ± 5.98 × 10 ⁴	10.20	0.70 ± 0.19	0.30 ± 0.19	0.00 ± 0.00	0.00 ± 0.00
Wood deck	8.55 ± 1.53 × 10 ⁴	10.04	0.76 ± 0.10	0.24 ± 0.09	0.00 ± 0.00	0.00 ± 0.00
Passenger compartment	1.96 ± 0.62 × 10 ⁴	2.28	0.91 ± 0.15	0.09 ± 0.15	0.00 ± 0.00	0.00 ± 0.00
Background average	5.36 ± 3.4 × 10 ³	—	0.91 ± 0.04	0.09 ± 0.04	0.00 ± 0.00	0.00 ± 0.00

*Expressed as mean ± SD.

†Expressed as ratio of means of Average Number Densities during overhaul and Average background values for the same scenario.

‡Upper values in size ranges are less than those actually stated but have been adjusted to the nearest 0.01 μm.

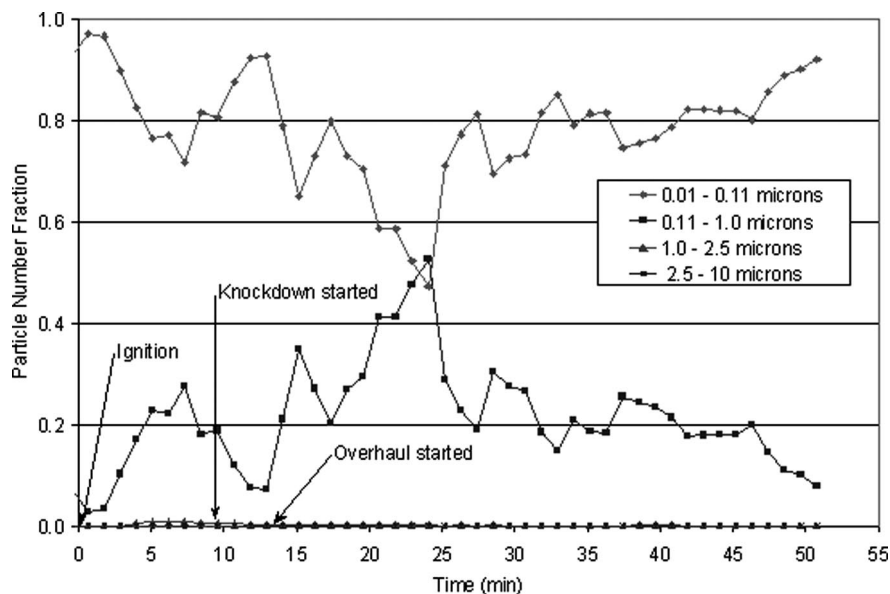


FIGURE 2. Particulate size distribution after ignition of the attic structure.

attic test structure. The overhaul phase is the most critical in consideration of firefighter particle exposure, however, because respiratory protection is commonly not worn during this period. Particle size and number density distributions were measured every 67 seconds for ~30 minutes. Background measurements were completed 90 minutes before conducting the test fires.

RESULTS

The average particle number density, measured in particles per cubic centimeter, and size distribution, measured as the fractions of particles within specific size ranges, is presented in Table 2, for the overhaul stage of each fire scenario. Particle densities measured during overhaul exceeded the average fire test laboratory background concentration at Underwriters Laboratories by a factor of ~2 in the lowest number density scenario (automobile passenger compartment) and 400 in the highest number density scenario (bedroom). Higher particle number densities were observed for the room scenarios (attic, bedroom, kitchen, and living room) than the external decks and automobile, presumably due to containment within an enclosed space. The number density of particles between scenarios would also be expected to be different between scenarios, given the differences in materials involved. Despite differences in

furnishings and other items (and their chemical compositions) involved in the fires, the smoke particle size distributions were found to be similar for all seven scenarios with particles <1 μm in diameter comprising >99% of those measured.

In the attic fire scenario, particles with diameters in the ultrafine range (0.1 μm or less) were prevalent throughout the fire growth, knockdown (beginning 9.12 minutes after ignition), and overhaul stage (beginning 13.43 minutes after ignition and continuing until the end of data collection; Figs. 2, 3).

DISCUSSION

These findings demonstrate that ultrafine particles are generated at high number densities increased relative to background values during the knockdown and overhaul stages of fire suppression. Number densities seemed to differ markedly between our study scenarios, which was to be expected on the basis of distinct types of residential materials and appliances being combusted in different situations (eg, toys and holiday decorations in the attic and couch and television in the living room). However independent of study scenarios, ultrafine particles were the most prevalent type of particulate matter generated.

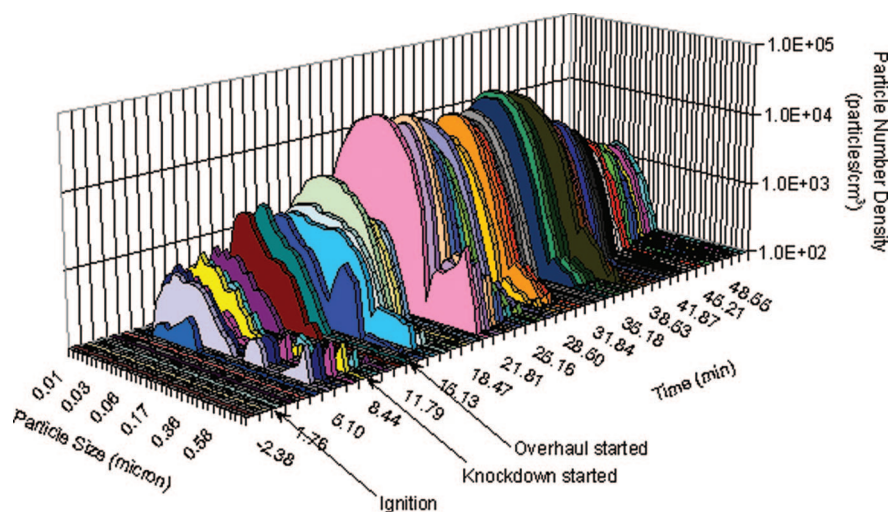


FIGURE 3. Particle number density and size distribution after ignition of the attic structure.

Firefighters are exposed to high levels of ultrafine particles throughout fire suppression, but during the knockdown phase, they are usually protected through use of self-contained breathing apparatus (SCBA). The resultant potential ultrafine exposure during overhaul may account in part for the previously demonstrated increased cardiovascular morbidity and mortality associated with fire suppression activities relative to non-emergency duties.³ In general population studies, a positive correlation has been established between increased exposure to urban air pollution-related particulate matter (PM₁₀ and PM_{2.5}) and increased cardiovascular morbidity and mortality.^{24–31} The contribution attributable solely to ultrafine particles, however, is not well characterized.^{24–27} The density of ultrafine particles (diameter 0.1 μm or less) in urban air ranges from 5×10^3 to $>3 \times 10^5$ particles per cubic centimeter;¹⁸ therefore, the values in the upper part of this range are comparable with those found during fire suppression in this study. In urban situations, ultrafine particles are generated primarily from fossil fuel combustion sources such as coal powered utilities and diesel engines. Their number densities vary with time of day but decrease with distance from the source of generation as agglomeration increases particle diameters.¹⁹ Short-term exposure to ultrafine particles at average levels of 1.2 to 1.5×10^5 particles per cubic centimeter induced a variety of changes in cardiovascular parameters in healthy volunteers, in recent clinical studies.^{20,21} A recent mortality study examining the effect of occupational particulate exposure, including diesel exhaust in construction workers, showed an increased rate of ischemic heart disease (IHD).²²

The exposure findings in this study are consistent with other studies in terms of the relationship between background and workplace ultrafine particle exposure levels.^{14–17} Overall, previous results suggest a possible association between occupational particulate exposures and IHD mortality as well as non-fatal myocardial infarction, and stronger evidence of associations with heart rate variability and systemic inflammation, which are potential intermediates between occupational PM exposure and IHD.¹⁴ In an industrial setting involving welding, smelting, molding, laser cutting, and fettling (removing excess materials from aluminum molds), ultrafine particle area concentrations ranged from 1.2×10^4 to 1.3×10^5 particles per cubic centimeter.¹⁵ During the use of electrocautery, lasers and ultrasonic scalpels in health care surgical settings health care worker experienced brief exposure to ultrafine particles in excess of 1.0×10^5 particles per cubic centimeter.¹⁷ In our study of

combustion of home structural materials, number densities similar to or in excess of these values were generally observed (4.6×10^4 to 2.1×10^6 per cubic centimeter). Our sampling strategy may not be completely representative of particle concentration over a range of standing heights, distances from source locations, or burn durations, however, since the instrument probe was fixed in the center of structure walls at an elevation that represented firefighter standing height.

The mechanisms underlying the observed increased cardiovascular morbidity and mortality in general population studies from exposure to particulate air pollution have not been fully delineated and are the subject of ongoing research.¹³ Unique properties of ultrafine particles that may be involved include their reactivity, large surface area to mass ratios, and ability to transport other toxicants to target organs.^{22,32} The large surface area to mass ratio enables the transport of large amounts of adsorbed toxic agents (such as those generated during a fire) to internal targets.^{32,33} Translocation from the airways into the circulation and lymphatics and the resulting potential for cardiovascular toxicity has also been suggested as a component for ultrafine particle toxicity, but this potential mechanism remains controversial.^{33,34} Proposed mechanisms by which inhaled ultrafine particles may induce adverse effects on the cardiovascular system include induction and release of mediators of systemic inflammation and oxidative stress, alteration of autonomic balance (including heart rate variability), and direct effects on the vasculature of particle constituents that have entered the systemic circulation.^{12,35} Physiologic responses reported to exposures containing ultrafine particles at unspecified levels include triggering of myocardial ischemia and infarctions as a result of acute arterial vasoconstriction,³⁶ endothelial dysfunction,³⁷ arrhythmias,³⁸ and procoagulant/thrombotic actions.^{39–41} Long-term intermittent exposure to ultrafine particles with diameter $<0.18 \mu\text{m}$ has also been shown to enhance the chronic genesis of atherosclerosis in mice at a number density of 5.6×10^5 per cubic centimeter, comparable with that found in our structural fire scenarios.⁴² Therefore, these previous studies in conjunction with our findings support the hypothesis that exposure to high levels of ultrafine particles may represent a potential additional risk for CHD events in firefighters during fire suppression duties.

A variety of recommendations for firefighters and fire departments have arisen from previous analyses of firefighter on-duty CHD morbidity and mortality. These recommendations have focused on medical screening for CHD risk factors, medical management of personnel with known risk factors for the development or exacerbation of CHD, implementation of wellness/fitness pro-

grams, and the undertaking of further research activities and risk reduction measures (including personal protective equipment),⁴³ “Heart presumption” workers’ compensation legislation, affording a presumption that CHD in a firefighter is work related, has also been enacted in many states.⁴⁴

Ultrafine particles are not observable by the human eye. This may create a false sense of safety that leads firefighters to remove their SCBA protective equipment during overhaul to ameliorate the physical burden and potential heat stress associated with continued usage of SCBA.⁴⁵ Recommendations for more consistent usage of respiratory protective equipment during overhaul stemmed from a previous firefighter study showing that levels of exposure to several volatile chemicals, but not respirable or total particles, exceeded published ceiling exposure guidelines during this activity.⁴⁵ Changes in spirometric measurements and lung permeability parameters have also been documented in firefighters not wearing respiratory protection during overhaul.⁴⁶ Further research is needed to identify appropriate methods of usage and type of respiratory protective equipment required to limit firefighters’ workplace exposures to ultrafine particles during overhaul activities, yet that do not create additional burdens such as heat and physical stress.^{46,47}

Our findings demonstrate that ultrafine particles are generated at high number densities, increased relative to background values, during the knockdown and overhaul stages of fire suppression and can represent the most prevalent type of particulate matter generated during combustion of common residential materials and products during these stages. The results further emphasize the need for additional research examining whether exposure to ultrafine particles is a coronary event risk factor in firefighters. As a precautionary principle, respiratory protection measures during overhaul should be considered to decrease potential ultrafine particulate exposure of this population.

ACKNOWLEDGMENT

This study was supported by research grant EMW-2007-FP-02093 from the Department of Homeland Security AFG Fire Prevention & Safety Grants program and conducted jointly by Underwriters Laboratories Inc, the Chicago Fire Department, and the University Of Cincinnati College Of Medicine.

REFERENCES

- Fahy RF. *U.S. Firefighter Fatalities Due to Sudden Cardiac Death, 1995–2004*. Quincy, MA: National Fire Protection Association, June 2005. Available at: <http://www.nfpa.org/assets/files/PDF/OSCardiacDeath.pdf>. Accessed February 21, 2009.
- Guidotti TL. Occupational mortality among firefighters: assessing the association. *J Occup Environ Med*. 1995;37:1348–1359.
- 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*. 2005;356:1207–1215.
- 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.
- 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.
- Yoo HL, Franke WD. Prevalence of cardiovascular disease risk factors in volunteer firefighters. *J Occup Environ Med*. 2009;51:958–962.
- Glueck CJ, Kelley W, Wang P, Gartside PS, Black D, Tracy T. Risk factors for coronary heart disease among firefighters in Cincinnati. *Am J Ind Med*. 1996;30:331–340.
- Clark S, Rene A, Theurer WM, Marshall M. Association of body mass index and health status in firefighters. *J Occup Environ Med*. 2002;44:940–946.
- Soteriades ES, Hauser R, Kawachi I, Liarokapis D, Christiani DC, Kales SN. Obesity and cardiovascular disease risk factors in firefighters: a prospective cohort study. *Obes Res*. 2005;13:1756–1763.
- Ide CW. A longitudinal survey of the evolution of some cardiovascular risk factors during the careers of male firefighters retiring from Strathclyde Fire Brigade from 1985–1994. *Scott Med J*. 2000;45:79–83.
- Thompson PD, Franklin BA, Balady GJ, et al; American Heart Association Council on Nutrition, Physical Activity, and Metabolism; American Heart Association Council on Clinical Cardiology; American College of Sports Medicine. Exercise and acute cardiovascular events placing the risks into perspective: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism and the Council on Clinical Cardiology. *Circulation* 2007; 115:2358–2368.
- Brook RD. Cardiovascular effects of air pollution. *Clin Sci*. 2008;115:175–187.
- Araujo JA, Nel AE. Particulate matter and atherosclerosis: role of particle size, composition and oxidative stress. *Part Fibre Toxicol*. 2009;6:24.
- Fang SC, Cassidy A, Christiani DC. A systematic review of occupational exposure to particulate matter and cardiovascular disease. *Int J Environ Res Public Health*. 2010;7:1773–1806.
- Elihn K, Berg P. Ultrafine particle characteristics in seven industrial plants. *Ann Occup Hyg*. 2009;53:475–484.
- Evans DE, Heitbrink WA, Slavin TJ, Peters TM. Ultrafine and respirable particles in an automotive grey iron foundry. *Ann Occup Hyg*. 2008;52:9–21.
- Brüske-Hohfeld I, Preissler G, Jauch KW, et al. Surgical smoke and ultrafine particles. *J Occup Med Toxicol*. 2008;3:31–45.
- McMurry PH, Woo KS. Size Distributions of 3-100-nm Urban Atlanta aerosols: measurements and observations. *J Aerosol Med*. 2002;15:169–178.
- Zhu Y, William C, Hinds WC, Shen S, Sioutas C. Seasonal and spatial trends in fine particulate matter. Seasonal trends of concentration and size distribution of ultrafine particles near major highways in Los Angeles. *Aerosol Sci Technol*. 2004;38:5–13.
- Gong H Jr, Linn WS, Clark KW, et al. Exposures of healthy and asthmatic volunteers to concentrated ambient ultrafine particles in Los Angeles. *Inhal Toxicol*. 2008;20:533–545.
- Samet JM, Rappold A, Graff D, et al. Concentrated ambient ultrafine particle exposure induces cardiac changes in young healthy volunteers. *Am J Respir Crit Care Med*. 2009;79:1034–1042.
- Torén K, Bergdahl IA, Nilsson T, Järholm B. Occupational exposure to particulate air pollution and mortality due to ischaemic heart disease and cerebrovascular disease. *Occup Environ Med*. 2007;64:515–519.
- Federal Emergency Management Agency. Fire department fire run profile. *Topical Fire Report Series*. 2007;7:1–7.
- Laden F, Schwartz J, Speizer FE, Dockery DW. Reduction in fine particulate air pollution and mortality. Extended follow-up of the Harvard Six Cities study. *Am J Respir Crit Care Med*. 2006;173:667–672.
- Pope CA, Dockery DW. Health effects of fine particulate air pollution: lines that connect. *J Air Waste Manage Assoc*. 2006;56:709–742.
- Peters A, Dockery DW, Muller JE, Mittleman MA. Increased particulate air pollution and the triggering of myocardial infarction. *Circulation*. 2001;103:2810–2815.
- Gold DR, Litonjua AA, Zanobetti A, et al. Air pollution and ST-segment depression in elderly subjects. *Environ Health Perspect*. 2005;113:883–887.
- Ibald-Mulli A, Timonen KL, Peters A, et al. Effects of particulate air pollution on blood pressure and heart rate in subjects with cardiovascular disease: a multicenter approach. *Environ Health Perspect*. 2004;112:369–377.
- Tonne C, Melly S, Mittleman M, Coull B, Goldberg R, Schwartz J. A case-control analysis of exposure to traffic and acute myocardial infarction. *Environ Health Perspect*. 2007;115:53–57.
- Tonne C, Yanosky J, Gryparis A, et al. Traffic particles and occurrence of acute myocardial infarction: a case-control analysis. *Occup Environ Med*. 2009;66:797–804.
- Pope CA III, Muhlestein JB, May HT, Renlund DG, Anderson JL, Home BD. Ischemic heart disease events triggered by short-term exposure to fine particulate air pollution. *Circulation*. 2006;114:2443–2448.
- Wittmaack K. In search of the most relevant parameter for quantifying lung inflammatory response to nanoparticle exposure: particle number, surface area, or what? *Environ Health Perspect*. 2007;115:187–194.
- Wiebert P, Sanchez-Crespo A, Seitz J, et al. Negligible clearance of ultrafine particles retained in healthy and affected human lungs. *Eur Respir J*. 2006;28:286–290.
- Furuyama A, Kanno S, Kobayashi T, Hirano S. Extrapulmonary translocation of intratracheally instilled fine and ultrafine particles via direct and alveolar macrophage-associated routes. *Arch Toxicol*. 2009;83:429–437.
- Sjogren B. Occupational exposure to dust: inflammation and ischaemic heart disease. *Occup Environ Med*. 1997;54:466–469.

36. Urch B, Brook JR, Wasserstein D, et al. Relative contributions of PM_{2.5} chemical constituents to acute arterial vasoconstriction in humans. *Inhal Toxicol*. 2004;16:345–352.
37. Törnqvist H, Mills NL, Gonzalez M, et al. Persistent endothelial dysfunction in humans after diesel exhaust inhalation. *Am J Respir Crit Care Med*. 2007;176:395–400.
38. Dockery DW, Luttmann-Gibson H, Rich DQ. Association of air pollution with increased incidence of ventricular tachyarrhythmias recorded by implanted cardioverter defibrillators. *Environ Health Perspect*. 2005;113:670–674.
39. Nemmar A, Hoet PHM, Dinsdale D, Vermylen J, Hoylaerts MF, Nemery B. Diesel exhaust particles in lung acutely enhance experimental peripheral thrombosis. *Circulation*. 2003;107:1202–1208.
40. Mutlu GM, Green D, Bellmeyer A, et al. Ambient particulate matter accelerates coagulation via an IL-6-dependent pathway. *J Clin Invest*. 2007;117:2952–2961.
41. Baccarelli A, Zanobetti A, Martinelli I, et al. Effects of exposure to air pollution on blood coagulation. *J Thromb Haemostasis*. 2007;5:252–260.
42. Araujo JA, Barajas B, Kleinman M, et al. Ambient particulate pollutants in the ultrafine range promote early atherosclerosis and systemic oxidative stress. *Circ Res*. 2008;102:589–596.
43. NIOSH. *Alert: Preventing Firefighter Fatalities Due To Heart Attacks and Other Sudden Cardiovascular Events*. DHHS (NIOSH) Publication No. 2007–133. Atlanta, GA: NIOSH; 2007.
44. International Association of Fire Fighters. Available at: <http://www.iaff.org/hs/phi/disease/heartDisease.asp>. Accessed February 5, 2010.
45. Bolstad-Johnson DM, Burgess L, Crutchfield CD, Stormont S, Gerkin Wilson JR. Characterization of firefighter exposures during fire overhaul. *AIHAJ*. 2000;61:636–641.
46. Burgess JL, Nanson CJ, Bolstad-Johnson DM, et al. Adverse respiratory effects following overhaul in firefighters. *J Occup Environ Med*. 2001;43:467–473.
47. Anthony TR, Joggerst P, James L, Burgess JL, Leonard SS, Shogren ES. Method development study for APR cartridge evaluation in fire overhaul exposures. *Ann Occup Hyg*. 2007;51:703–716.