

Medical and biological factors affecting mortality in elderly residential fire victims: a narrative review of the literature

Erik Eggert¹ and Fredrik Huss^{1,2}

Abstract

For older people (aged over 65 years), the risk of dying in a residential fire is doubled compared to the general population. Obvious causes of death mainly include smoke inhalation and burn injuries. That older people are more fragile and have more concurrent diseases is inherent, but what is it that makes them more vulnerable? It is known that the number of elderly people is increasing globally and that the increased risk of death in fires can be explained, at least in part, by physical and/or cognitive disabilities as well as socioeconomic and behavioural factors. The possibility that medical illnesses and an aging organism/tissues might explain this increased risk has not been shown to the same extent. Therefore, this narrative literature review focuses on medical and biological explanations. An initial search using the terms ‘elderly’, ‘fatal’, ‘residential’ and ‘fire’ yielded some interesting articles. Using a broader snowball search also accepting grey literature, several additional risk factors could be identified. Cardiovascular diseases, in particular atherosclerotic heart disease, greatly increases the vulnerability to, for example, carbon monoxide and probably also other asphyxiating gases. Cardiovascular diseases and lack of physical fitness may also increase vulnerability to heat. Burned elderly patients are also at a higher risk of death than younger patients, but it is controversial whether it is age itself or the pre-existing illnesses that come with age that increase the risk. Immunosenescence, malnutrition and female gender are other risk factors for poorer outcome after burns, all of which are common among older people.

Keywords

Elderly, smoke, heat, vulnerability, fatal, residential fire

Lay summary

Elderly people have an increased risk of dying in house fires for several known reasons. This review explores possible medical/biological explanations and finds heart disease to be an important explanation.

Introduction

As a result of advances in, for example, social care and medicine, the global number of elderly people (aged over 65 years) is increasing rapidly and is projected to increase rapidly from 0.6 billion people today and reach 1.5 billion in 2051.¹ People aged over 65 years have twice the mortality risk from fires than the general population. This increases to three times the risk at age > 75 years

¹The Burn Center, Department of Plastic- and Maxillofacial Surgery, Uppsala University Hospital, Sweden

²Department of Surgical Sciences, Plastic Surgery, Uppsala University, Uppsala, Sweden

Corresponding author:

Erik Eggert, The Burn Center, Department of Plastic- and Maxillofacial Surgery, Uppsala University Hospital, Akademiska sjukhuset, SE-75185 Uppsala, Sweden.

Email: erik.eggert@akademiska.se



and four times the risk at > 85 years, compared with that of the general population.²

It has previously been shown that the risk of injury and death in a residential fire is increased in risk groups such as men, those impaired by alcohol or other drugs, smokers, physically disabled or cognitively impaired patients, and extreme old age.^{3–8} Behavioural factors, as well as cultural and sociodemographic differences, also aid in explaining this increased risk.^{5,9–13}

The aim of this literature review was to find other possible medical and/or biological explanations that make the elderly more vulnerable to fire, smoke or heat that could explain the increased morbidity and mortality in elderly involved in a residential fire.

Methods

In June 2015, a review of the available literature on the subject medical or biological vulnerability to fire death was conducted. No peer-reviewed studies covered the specific topic, as expected. The PubMed and Cochrane Library databases were searched for publications using the query terms ‘elderly’, ‘fatal’, ‘residential’ and ‘fire’. Limitations used were: date of publication – all, language – English, species – human. Snowballing generated further publications including papers from grey literature (which were also considered). Publications on immobilisation, substance abuse and other non-medical explanations to increased fire mortality in the elderly were excluded.

Results

Causes of death in residential fires

Smoke inhalation alone or in combination with cutaneous burns accounts for up to 75–80% of deaths after residential fires. Smoke inhalation rather than burns is the major cause of death, as only 26% are attributable to burns alone.^{14–16} Immediate causes of death include asphyxiation, heat stroke (core hyperthermia) and distributive shock as a result of burns. Less common causes include associated trauma or earlier deaths (from homicide, suicide or disease). When looking at causes of death, an important aspect is incapacitation by, for example, fire gases or heat retaining the individual within the danger zone causing further damage and ultimately death. This is not always evaluated in autopsies and forensic studies. Gases and heat are incapacitating at lower than lethal levels¹⁷ and human tolerance most likely declines with advanced age and pre-existing illnesses.

Table 1. Important fire gases.

Asphyxiants	Irritants
Carbon monoxide	Hydrogen chloride
Carbon dioxide	Hydrogen bromide
Oxygen deficiency	Hydrogen fluoride
Hydrogen cyanide	Sulphur dioxide
	Nitrogen dioxide
	Acrolein
	Formaldehyde

Important gases in fires as stated in International Standards Organization (ISO) document 13344.¹⁸

Smoke

Smoke is a complex mixture of many different gases and particles, and its composition depends on the material(s) burning. The composition also varies considerably with the time that has elapsed since ignition and with the supply of oxygen. We reviewed the main toxic gases in smoke (Table 1) to examine indications of increased toxicity with ageing or pre-existing illnesses. Gases in smoke can be divided into asphyxiants (that cause tissue hypoxia) and irritants (that irritates mucous membranes causing damage to the respiratory system and sight).¹⁸

Carbon monoxide

Carbon monoxide is the major toxic gas in smoke from burning wood and polymers¹⁴ and causes most deaths after smoke inhalation in residential fires.¹⁹ The mechanisms include formation of carboxyhaemoglobin, which disables oxygen transport and causes a leftward shift of the dissociation curve of unaffected haemoglobin, together with competitive inhibition of cytochrome oxidase that disrupts cellular respiration and binds to cardiac myoglobin, so blocking the oxygen reserves.²⁰

Several studies have postulated increased carbon monoxide toxicity in patients with cardiovascular disease, causing incapacity and death at sublethal levels,^{15,21–26} which at least partly explains why elderly people are more likely to die. Birky et al. investigated 541 deaths after fires and found that 60% of those who died did so of carbon monoxide poisoning alone (using more than 50% carboxyhaemoglobin as the lethal concentration), 20% of deaths were attributed to carbon monoxide poisoning together with

cardiovascular disease or other toxic compounds and only 11% died from burns alone.¹⁶

In a group being treated with hyperbaric oxygen for severe carbon monoxide poisoning, an older subgroup was found that had regional abnormalities of wall movement on echocardiography, which suggests that previously asymptomatic atherosclerotic disease gave rise to local ischaemia during the exposure to carbon monoxide.²⁷ This study also found that male gender and a history of hypertension were predictors of myocardial injury induced by carbon monoxide.

Several studies on people known to have exertional angina pectoris, and who were exposed to low concentrations of carbon monoxide, showed a dose-response relation with carboxyhaemoglobin concentrations and time to angina, as well as changes in the ST segment.^{28,29} While healthy adults can tolerate 10–15% carboxyhaemoglobin and remain unaffected, patients with coronary artery disease may develop angina quickly during exercise with concentrations as low as 2%. Exposure to 15% carboxyhaemoglobin has been reported to cause myocardial infarction.³⁰ Purser investigated a fire, at a care home for elderly residents with various illnesses, that resulted in the death of 14/19 elderly.³¹ A trend towards increased susceptibility to carbon monoxide poisoning in the residents with cardiovascular disease was found. For heart disease alone, the increase was statistically significant.

Carbon dioxide

Inhaled carbon dioxide is well tolerated in low concentrations, when it acts as a simple asphyxiant by displacing oxygen. With increasing hypercapnia tachypnoea, tachycardia, cardiac arrhythmias, altered consciousness and cardiac stress increase.³² Hypercapnia is thus probably more dangerous for people with cardiovascular diseases. Ambient carbon dioxide concentration of more than 10% is potentially lethal.³³ In a residential fire, the concentrations of carbon dioxide are unlikely to rise fast enough to kill someone before other, more fast-acting, gases reach lethal concentrations. The main effects of carbon dioxide are increased respiratory rate that increases the uptake of other toxic agents and synergise with them, as a result of displacement of oxygen.¹⁵ Elderly people have a reduced ventilatory response to hypoxia and hypercapnia,³⁴ which may make them more vulnerable in a fire incident.

Oxygen depletion

An oxygen-deficient atmosphere in a residential fire is a result of consumption of oxygen by the

process of combustion, and the combined action of other simple asphyxiants that displace oxygen from the ambient air. Oxygen deprivation alone can lead to incapacity at around 10% and be lethal below 7%. However, such low concentrations are unlikely without extreme heat, which in itself would be lethal (as in a flashover, when the room temperature reaches a point where most flammable materials autoignite and engulf the room with flames).³⁵

Hydrogen cyanide

Hydrogen cyanide is a highly toxic product of combustions that involve modern materials such as synthetic polymers and organic materials with high nitrogen content. There is still some controversy about its role in deaths from inhalation of smoke, but hydrogen cyanide is increasingly recognised as an important asphyxiant that requires immediate treatment. Its main toxic effect results from its molecular similarity to oxygen. It reversibly binds to cytochrome a₃ and disrupts the oxidative metabolism at the cellular level. Affected cells must then use inefficient anaerobic metabolism that produces lactate, which leads to acidosis. This lactic acidosis can be used clinically to diagnose hydrogen cyanide exposure indirectly and prompts the immediate use of an antidote such as hydroxocobalamin. Hydrogen cyanide is difficult to detect both clinically and forensically because it both forms and degrades in dead tissue.³⁶ High concentrations of hydrogen cyanide are often found together with high concentrations of carbon monoxide during autopsies.¹⁶

Although no studies were found that directly indicated increased vulnerability to hydrogen cyanide in any risk group,³⁷ there is probably enough synergy with other asphyxiants and people with cardiovascular disease may well be more vulnerable. The synergy between carbon monoxide and hydrogen cyanide toxicity has been shown in animals.^{38,39}

Nitrogen dioxide

Nitrogen dioxide is an irritant gas with asphyxiating potential through the formation of methaemoglobin which blocks oxygen transport in a similar way to carbon monoxide and carboxyhaemoglobin. Clinically relevant methaemoglobin concentrations above 10% have been sampled from people who died in fire incidents.⁴⁰

Asphyxiation synergy

All asphyxiants in smoke from a residential fire, both simple (gases that displace oxygen from

ambient air and thereby lower the oxygen concentration) and systemic, ultimately lower tissue oxygen concentrations. Carbon monoxide has been extensively studied and cardiovascular diseases seem to increase a person's vulnerability to it through cardiac ischaemia. Other asphyxiants may produce similar effects and act in synergy, but we found no reports that directly supported this in humans, though animal models do indicate synergy.^{38,39}

Irritants

Irritant gases include hydrogen chloride, nitrogen dioxide, sulphur dioxide, hydrogen bromide, acrolein and formaldehyde, all of which cause lung damage and have important and lethal roles in lung injury. People with pre-existing conditions such as chronic obstructive pulmonary disease have fewer physiological reserves and any inhalation injury may be dangerous. Irritant gases can also cause bronchospasm, hypersecretion of mucus membranes and visual impairment, which render the person unable to escape the danger zone.⁴¹ The incapacitating effects of these irritant gases play an important part in simulations of fires and when new materials are being fire-tested.¹⁸

The effects of the principal irritant gases are similar and hydrogen chloride is the most studied irritant in smoke, although few data about its action in humans are available.⁴² Together with other irritants it can play an important part in incapacitating somebody in a fire incident, but even in a fire with appreciable chlorine pyrolysis, carbon monoxide will reach incapacitating concentrations sooner.⁴³

People with hyper-reactive airways, e.g. asthmatics, may be more sensitive to low concentrations of hydrogen chloride, but at lethal or incapacitating concentrations the intraspecies variation is likely to be small.⁴² No studies that specifically included older people were found.

Heat stroke

Classic heat stroke is caused by high ambient temperatures, and older people seem to have an increased risk. Several studies have concluded that age itself does not increase the risk of heat stroke or heat (in)tolerance in general. Instead, it is pre-existing illnesses and a sedentary lifestyle with low aerobic capacity that cause the vulnerability.^{44,45} The increase in temperature in a residential fire is often so fast that classic heat stroke

probably does not have time to develop before other mechanisms of incapacitation and death occur, but there may be decreased tolerance to heat in people with cardiovascular disease. Ambient air temperatures of 90°C can be tolerated for roughly 46 min without resulting in cutaneous burns, but only slightly higher temperatures (108°C) can cause burns in only 31 s and be incapacitating in minutes.^{46,47} Heat ($\geq 31^\circ\text{C}$) together with exposure to carbon monoxide has been studied in healthy adults and the results indicate some effect, with greater reporting of exertion than under thermoneutral conditions.⁴⁸ The heat stress may act rapidly, in synergy with smoke intoxication, to incapacitate a person.

Burns and burn care in elderly patients

Later causes of death include failure of treatment of shock after burns, multiple organ dysfunction syndrome (MODS) based on sepsis and septic shock and the active decision to choose palliative care in patients with a poor prognosis. Many studies on the outcome of burns in elderly people come from burn centres where there is an obvious risk of selection bias, as (some) patients with a poor prognosis will probably not be referred and instead end their lives at the local hospital. Some studies have investigated the age groups 46+ or 56+, but found no correlation with mortality or the presence of pre-morbid diseases,^{49,50} while another study found that mortality increased with age and that pre-existing illnesses did not affect outcome in patients aged over 65 years.⁵¹ However, in the youngest subgroup, in this study (56–65 years) pre-existing illnesses increased mortality.

Other studies have indicated that there is no clear correlation with age and mortality, instead it is pointed out that pre-existing illnesses affect mortality.^{52,53} Interestingly, one study found that older age correlated with overall mortality from burns but not when patients being given palliative care were excluded.⁵⁴ Some of the in-hospital mortality in elderly patients with burns is probably explained by this kind of decision.

Thombs et al. specifically looked at pre-existing illnesses and mortality from burns in a large group ($n = 32,449$), and found increased in-hospital mortality for patients with HIV, malignancies, obesity, alcoholism, pulmonary circulatory disorders, peripheral vascular disorders, and renal, hepatic and cardiac diseases. Naturally the amount of pre-existing illnesses/patient increased with age.⁵⁵

Malnutrition is common among elderly people and this may influence the healing process adversely.⁵⁶ Pre-existing protein and energy malnutrition increases mortality after burns in elderly patients.⁵⁷ Immunosenescence is another adverse factor. With increasing age the adaptive immune system deteriorates, which leaves the patient more prone to infections and subsequent mortality and morbidity.⁵⁸

Differences in burn care between the sexes

Interestingly, two studies showed significant differences between men and women in both the incidence and outcome of burns. Male predisposition to burns seems to disappear in older people and perhaps may be explained by fewer work-related burns and known female longevity.^{51,59} Some studies have shown increased mortality from burns in women aged under 61–70 years^{60–62} but not among older women, while some have shown generally higher mortality for women, in particular when they get older.⁵⁶ Chang et al. showed similar mortality for men and women among geriatric patients, but the women had significantly smaller overall burned %TBSA and full-thickness burned %TBSA.⁶³

The sex difference may in part be explained by differences in how the burn was sustained. Female sex hormones may play a part in the immune response to a burn, or may exert other unknown effects, as the increased mortality among women in different age groups correlated well with increased hormonal concentrations (15–61 years).⁶¹ One study specifically explored sex differences in healthcare and showed that less ancillary services during inpatient care were used for female patients, even after adjusting for confounders such as age, diagnosis-related group and length of stay. This means, for example, fewer radiographs and fewer laboratory analyses, which may indirectly affect outcome.⁶⁴

Discussion

Elderly people are at increased risk of dying in residential fires as a result of various interacting risks. Social and behavioural factors include living alone with no chance for immediate help if there is a fire, old houses that may have faulty heating or wiring, the use of extra heat sources such as electric space heaters or electric blankets, together with cooking. These, combined with cognitive impairment, can result in an increased

risk of a fire starting. Physical disabilities and impaired senses are more common with increasing age and also affect the person's ability to escape alive and uninjured. How much these risk factors contribute to the overall risk increase, however, is not clear.

People known to have coronary artery disease are clearly susceptible to carbon monoxide poisoning and are probably equally as sensitive to most other asphyxiants. With increasing age, occult cardiovascular disease is common and may explain some of the increased risk of dying at the scene of a fire. Cardiovascular diseases and cardiopulmonary instability can render elderly people susceptible to heat stroke, which in turn may act in synergy with carbon monoxide poisoning and further increase the risk.

In studies from burn centres, the results are conflicting regarding whether age alone increases mortality or if the increased mortality is the result of pre-existing illnesses that are more common with increasing age. Some of the increased mortality in elderly patients with burns is the result of limitations placed on treatment because of poor prognosis in patients who perhaps would be treated more aggressively had they been younger. Pre-existing malnutrition is common in elderly patients, and combined with immunosenescence results in poor healing and decreased resistance to infection, which in turn affects mortality. The male predisposition to burns seems to diminish with age and women with burns seem to do worse, for reasons that are not clear. With ageing populations and higher female life expectancy, this may add to mortality among elderly people.

Conclusion

Several co-existing medical, social, behavioural and biological factors have been described to explain the increased mortality among elderly people. Cardiovascular disease in general, and heart disease in particular, increase vulnerability to carbon monoxide, and probably to other asphyxiating fire gases, thereby increasing the risk of dying in a residential fire. For elderly people who sustain burns in fires, there are several possible explanations for increased mortality but whether it is age alone or pre-existing illnesses that influence mortality is still not clear.

Declaration of conflicting interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

References

- Poulose N and Raju R. Aging and injury: alterations in cellular energetics and organ function. *Aging Dis* 2014; 5: 101–108.
- Gamache S. Reaching those at highest risk to fires and burns: Young children, older adults, and people in low-income communities. *Inj Contr Saf Promot* 2001; 8(3): 199–201.
- Elder AT, Squires T and Busuttill A. Fire fatalities in elderly people. *Age Ageing* 1996; 25: 214–216.
- Marshall SW, Runyan CW, Bangdiwala SI, et al. Fatal residential fires: who dies and who survives? *JAMA* 1998; 279(20): 1633–1637.
- Runyan CW, Bangdiwala SI, Linzer MA, et al. Risk factors for fatal residential fires. *N Engl J Med* 1992; 327: 859–863.
- Leth P, Gregersen M and Sabroe S. Fatal residential fires in the municipality of Copenhagen, 1991–1996. *Prev Med* 1998; 27: 444–451.
- Istre GR, McCoy MA, Osborn L, et al. Deaths and injuries from house fires. *N Engl J Med* 2001; 344: 1911–1916.
- Ahrens M. Smoke alarms in US home fires [NFPA (National Fire Protection Association) Website]. September, 2015. Available at: <http://www.nfpa.org/news-and-research/fire-statistics-and-reports/fire-statistics/fire-safety-equipment/smoke-alarms-in-us-home-fires> (accessed 23 November 2015).
- Miller I. Human behavior contributing to unintentional residential fires 1997–2003 [New Zealand Fire Service website]. February 4, 2005. Available at: <http://www.fire.org.nz/Research/Published-Reports/Pages/Human-Behaviour-Contributing-to-Unintentional-Residential-Fire-Deaths-1997-2003.html> (accessed 23 November 2015).
- Dissanaike S and Rahimi M. Epidemiology of burn injuries: highlighting cultural and socio-demographic aspects. *Int Rev Psychiatry* 2009; 21(6): 505–511.
- Warda L, Tenenbein M and Moffatt M. House fire injury prevention update. Part I. A review of risk factors for fatal and non-fatal house fire injury. *Inj Prev* 1999; 5: 145–150.
- Mulvaney C, Kendrick D, Towner E, et al. Fatal and non-fatal fire injuries in England 1995–2004: time trends and inequalities by age, sex and area deprivation. *J Public Health* 2008; 31(1): 154–161.
- McGwin G, Chapman V, Curtis J, et al. Fire fatalities in older people. *J Am Geriatr Soc* 1999; 47: 1307–1311.
- Alarie Y. The toxicity of smoke from polymeric materials during thermal decomposition. *Ann Rev Pharmacol Toxicol* 1985; 25: 325–347.
- Shusterman DJ. Clinical smoke inhalation injury: Systemic effects. *Occup Med* 1993; 8(3): 469–503.
- Birky MM and Clarke FB. Inhalation of toxic products from fires. *Bull NY Acad Med* 1981; 57(10): 997–1013.
- Purser D. Behavioural impairment in smoke environments. *Toxicology* 1996; 115: 25–40.
- International Standard Organization (ISO). ISO 13344 :2004 Estimation of the lethal toxic potency of fire effluents [International Standards Organization website]. October 15, 2004. Available at: <https://www.iso.org/standard/37850.html> (accessed 23 November 2015).
- Varon J, Marik PE, Fromm RE, et al. Carbon monoxide poisoning: A review for clinicians. *J Emerg Med* 1999; 17(1): 87–93.
- Prien T and Traber DL. Toxic smoke compounds and inhalation injury. *Burns* 1985; 14(6): 451–460.
- Ernst A and Zibrak JD. Carbon monoxide poisoning. *New Engl J Med* 1998; 339(22): 1603–1608.
- Weaver LK. Carbon monoxide poisoning. *New Engl J Med* 2009; 360: 1217–1225.
- Stefanidou M, Athanaselis S and Spiliopoulou C. Health impacts of smoke inhalation. *Inhal Toxicol* 2009; 20: 761–766.
- Balraj EK. Atherosclerotic coronary artery disease and “low” levels of carboxyhemoglobin; Report of fatalities and discussion of pathophysiological mechanisms of death. *J Forensic Sci* 1984; 29(4): 1150–1159.
- Wright J. Chronic and occult carbon monoxide poisoning: we don't know what we're missing. *Emerg Med J* 2002; 19: 369–390.
- Balzan MV, Cacciottolo JM and Mifsud S. Unstable angina and exposure to carbon monoxide. *Postgrad Med J* 1994; 70: 699–702.
- Satran D, Henry CR, Adkinson C, et al. Cardiovascular manifestations of moderate to severe carbon monoxide poisoning. *J Am Coll Cardiol* 2005; 45(9): 1513–1516.
- Allred EN, Bleecker ER, Chaitman BR, et al. Effects of carbon monoxide on myocardial ischemia. *Environ Health Persp* 1991; 91: 89–132.
- Raub J. WHO Environmental Health Criterion 213: Carbon Monoxide (second edition) [World Health Organization website] November 30, 2004. Available at: http://www.who.int/ipcs/publications/ehc/ehc_213/en/ (accessed 24 November 2015).
- Griem P, Rodgers G and Camacho I. Carbon monoxide. In: *Acute Exposure Guideline Levels for Selected Airborne Chemicals: Volume 8*. Washington, DC: The National Academies Press, 2010: 49–143. Available at: <https://www.nap.edu/read/12770/chapter/7>
- Purser D. Effects of pre-fire age and health status on vulnerability to incapacitation and death from exposure to carbon monoxide and smoke irritants in Rosepark fire incident victims [Researchgate web site]. October 9, 2015. Available at: https://www.researchgate.net/publication/308942174_Effects_of_pre-fire_age_and_health_status_on_vulnerability_to_incapacitation_and_death_from_exposure_to_carbon_monoxide_and_smoke_irritants_in_Rosepark_fire_incident_victims (accessed 20 November 2015).
- Langford NJ. Carbon dioxide poisoning. *Toxicol Rev* 2005; 24(4): 229–235.
- Halpern P, Raskin Y, Sorkine P, et al. Exposure to extremely high concentrations of carbon dioxide: a clinical description of a mass casualty incident. *Ann Emerg Med* 2004; 43: 196–199.
- Sharma G and Goodwin J. Effect of aging on respiratory system physiology and immunology. *Clin Interv Aging* 2006; 1(3): 253–260.
- Alarie Y. Toxicity of fire smoke. *Cr Rev Toxicol* 2002; 32(4): 259–289.
- Huzar TF, George T and Cross JM. Carbon monoxide and cyanide toxicity: etiology, pathophysiology and treatment in inhalation injury. *Expert Rev Respir Med* 2013; 7(2): 159–170.
- Talmage S and Rodgers G. Hydrogen Cyanide. In: *Acute Exposure Guideline Levels for Selected Airborne Chemicals: Volume 2*. Washington, DC: The National Academies Press, 2002: 211–265.
- Levin BC, Paabo M, Gurman JL, et al. Effects of exposure to single or multiple combinations of the predominant toxic gases and low oxygen atmospheres produced in fires. *Fundam Appl Toxicol* 1987; 9(2): 236–250.
- Norris JC, Moore SJ and Hume AS. Synergistic lethality induced by the combination of carbon monoxide and cyanide. *Toxicology* 1986; 40(2): 121–129.
- Hoffman RS and Sauter D. Methemoglobinemia resulting from smoke inhalation. *Vet Hum Toxicol* 1989; 31(2): 168–170.
- Purser D. Physiological effects of combustion products and fire hazard assessment [Europacable web site]. May, 2009. Available at: <http://www.safety-during-fire.com/scientific/hazard.html> (accessed 23 November 2015).

42. Bast C and Hinz J. Hydrogen chloride. In: *Acute Exposure Guideline Levels for Selected Airborne Chemicals: Volume 4*. Washington, DC: The National Academies Press, 2004: 77–121.
43. Gann RG, Averill JD, Butler KM, et al. International Study of the Sublethal Effects of Fire Smoke on Survival and Health (SEFS): Phase I Final Report. Gaithersburg: National Institute of Standards (NIST); 2001. NIST Technical Note 1439. Available at: <https://www.nist.gov/publications/international-study-sublethal-effects-fire-smoke-survivability-and-health-sefs-phase-1> (accessed 23 November 2015).
44. Pandolf KB. Aging and human heat tolerance. *Exp Aging Res* 1997; 23(1): 69–105.
45. Kenney WL. Thermoregulation at rest and during exercise in healthy older adults. *Exerc Sport Rev* 1997; 25: 41–76.
46. Hill IR. Incapacitation and fires. *Am J Forensic Med Pathol* 1989; 10(1): 49–53.
47. Yeo TP. Heat stroke: a comprehensive review. *AACN Clin Issues* 2004; 15(2): 280–293.
48. Bunell DE and Horvath SM. Interactive effects of heat, physical work, and CO exposure on metabolism and cognitive task performance. *Aviat Space Environ Med* 1989; 60(5): 428–432.
49. Saffle JR, Larson CM, Sullivan J, et al. The continuing challenge of burn care in the elderly. *Surgery* 1990; 108(3): 534–543.
50. Lundgren RS, Kramer CB, Rivara FP, et al. Influence of comorbidities and age on outcome following burn injury in older adults. *J Burn Care Res* 2009; 30(2): 307–314.
51. Covington SD, Wainwright DJ and Parks DH. Prognostic indicators in the elderly patient with burns. *J Burn Care Rehabil* 1996; 17: 222–230.
52. Masud D, Norton S, Smailes S, et al. The use of a frailty scoring system for burns in the elderly. *Burns* 2013; 39: 30–36.
53. Rao K, Ali SN and Moiemens NS. Aetiology and outcome of burns in the elderly. *Burns* 2006; 32: 802–805.
54. Mahar P, Wasiak J, Bailey M, et al. Clinical factors affecting mortality in elderly burn patients admitted to a burns service. *Burns* 2008; 34: 629–636.
55. Thombs BD, Singh VA, Halonen J, et al. The effects of pre-existing medical comorbidities on mortality and length of hospital stay in acute burn injury. *Ann Surg* 2007; 245: 629–634.
56. Pereira CT, Barrow RE, Sterns AM, et al. Age-dependent differences in survival after severe burns: a unicentric review of 1674 patients and 179 autopsies over 15 years. *J Am Coll Surg* 2006; 202(3): 536–548.
57. Demling RH. The incidence and impact of pre-existing protein energy malnutrition on outcome in the elderly burn patient population. *J Burn Care Rehabil* 2005; 26: 94–100.
58. Franceschi C, Bonafè M and Valensin S. Human immunosenescence: the prevailing of innate immunity, the failing of clonotypic immunity, and the filling of immunological space. *Vaccine* 2000; 18: 1717–1720.
59. Pham TN, Kramer CB, Wang J, et al. Epidemiology and outcomes of older adults with burn injury: an analysis of the national burn repository. *J Burn Care Res* 2009; 30(1): 30–36.
60. O’Keefe GE, Hunt JL and Purdue GF. An evaluation of risk factors for mortality after burn trauma and the identification of gender-dependent differences in outcomes. *J Am Coll Surg* 2001; 192: 153–160.
61. McGwin G, George RL, Cross JM, et al. Gender differences in mortality following burn injury. *Shock* 2002; 18(4): 311–315.
62. Kerby JD, McGwin G, George RL, et al. Sex differences in mortality after burn injury: results of analysis of the national burn repository of the American burn association. *J Burn Care Res* 2006; 27: 452–456.
63. Chang EJ, Edelman LS, Morris SE, et al. Gender influences on burn outcomes in the elderly. *Burns* 2005; 31: 31–35.
64. Jha AK, Kuperman GJ, Rittenberg E, et al. Gender and utilization of ancillary services. *J Gen Intern Med* 1998; 13: 476–481.

How to cite this article

Eggert E and Huss F. Medical and biological factors affecting mortality in elderly residential fire victims: a narrative review of the literature. *Scars, Burns & Healing*, Volume 3, 2017. DOI: 10.1177/2059513117707686.