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Tee L. Guidotti *Editor*

# Health Risks and Fair Compensation in the Fire Service

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Editor

# Health Risks and Fair Compensation in the Fire Service

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# Foreword

Over three hundred firefighters lost their lives on September 11, 2001 when they climbed the stairs of the twin towers in a brave attempt to save the lives of the thousands of people trying desperately to escape. This profound act of bravery and professional commitment to public safety was probably the greatest example of the selfless dedication that personifies firefighters across North America.

Thankfully the scale of human loss that occurred on 9/11 does not happen often. Nevertheless, firefighters risk their lives every time they enter a burning building. Not only that, but these brave women and men often have to deal with the very real and serious occupational health consequences of their work. A fact that policy makers all too often forget.

This book expertly documents the tenacious struggle for recognition by local and international firefighting unions for increased awareness of these health risks. It outlines the empirical evidence used by advocates to sway public officials and policy makers towards a greater understanding of the dangers of their profession. It brings overdue recognition and attention to the sheer numbers of individuals affected by various toxic chemicals and carcinogens by simply doing their job.

On a personal note, I am proud of the opportunity I had to work with Alex Forrest and local firefighters in Winnipeg in dealing with the lack of support for the disabled and injured firefighters in Manitoba. The efforts of Alex were critical in significantly broadening workplace safety and insurance benefits supplied to firefighters suffering from work-related injuries. Equally, I know Harold Schaitberger, General President of the International Association of Fire Fighters, who has also worked tirelessly to use science-based research to challenge the status-quo approach to injured firefighters. In doing so, he has made significant changes in modernizing the policy framework around injured firefighters across a variety of jurisdictions. I am also grateful to Tee Guidotti for doing the research and documentation that supported the drafting of legislation that led to Manitoba being the first province or state to adopt modern presumption legislation for firefighters. I also admire the scholarship that has gone into the chapters contributed by researchers at Laurentian University and other centers of excellence. We are fortunate indeed that these fine scientists have focused their attention and considerable talents on this issue.

This book is a compelling read for anyone interested in the various struggles and successes of the advocates who have worked towards effective change in workplace safety legislation. This book by Alex and his academic colleagues reminds us that we not only have a moral obligation to our firefighters, but we must have a robust legal framework, based on empirical evidence, to ensure that the brave firefighters diagnosed with occupational disease receive proper justice.

*Mr. Doer is the former Premiere of Manitoba and the current Ambassador of Canada to the United States.*

Gary Doer

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# Introduction

*"While everyone else is running out of a burning building, firefighters are rushing in."*

This old saying reflects the culture of courage and commitment that makes firefighting a special occupation. Firefighters are well aware of the many hazards they face, of course, but they cannot allow the risk to impede their vital work in protecting the public. They must cope with the risks. In the past, the coping mechanism was largely by attitude. Firefighters took pride in being "smoke-eaters," too tough to stop even when the going got rough. That does not mean that they took unnecessary risks, but that they often had to overcome deficiencies in technology and strategy by sheer will. Fire technology changed, as it should have, and fire science became a more sophisticated field, with its own body of specialized knowledge. Through incremental improvements in existing technology, rather than any great technological breakthrough, firefighters were given the tools to protect themselves, in the form of better turn-out gear and more practical self-contained breathing apparatus. The new attitude may be summarized on another old saying "There are old firefighters and there are bold firefighters, but there are no old, bold firefighters."

Firefighters are careful to protect themselves while protecting the public they serve. This is both a matter of workers' rights and a matter of operational effectiveness. After all, avoidable injury creates more casualties and failures that can cost lives. The modern fire service depends now on effective technology and training to protect themselves even while they protect the public. The operative firefighter's saying for the current generation may be "Let no man's ghost return to say his training let him down."

This book is about the chronic health consequences of firefighting: how to prevent them, identify them when they are not obvious, compensate for them after they occurred, and recognize them as having occurred in the line of duty. Acute injuries are usually easy to identify, and this book will not dwell on them, except to make a few observations on disability that may result.

Health consequences that develop over time are much more difficult. Chronic impairment and disability among firefighters has been an active topic of investigation for many years. A considerable amount of research has been done on cardiovascular disease, lung diseases, cancer, behavioral disorders, and a few other health problems, with large gaps in between. These issues are part of a broader discussion involving health risk, protection of public safety professionals, and equitable compensation for firefighters.

## How to Use This Book

This book was not written as a strictly scholarly exercise. The primary purpose is to contribute to recognition of firefighter health issues and their resolution. To do so, this book brings together in one source, and interprets, the world literature on physical health risks associated with firefighting, except for traumatic injury and personal wellness, to approximately January 2015. This book summarizes a large and complicated scientific literature that has applications well beyond the considerable scientific interest of the topic. These uses include:

- Support of research and education on the health issues associated with firefighting
- Prevention of health risks among firefighters by fire departments and unions
- Anticipation of common health problems by healthcare professionals who take care of firefighters
- Adjudication of claims by firefighters for workers' compensation and other compensation programs, identifying types of evidence that may support such claims and selected grounds for rebuttal (which cannot be as inclusive)
- Guidance for the legislation and implementation of presumption policies
- Promotion of further research in this fascinating field: there are many unanswered questions of general interest and much yet to be learned from the experience of firefighters
- Satisfaction and emotional closure for family and friends through recognition of deaths that occurred in the line of duty but that evolved in slow motion, through chronic diseases such as cancer.

It is anticipated that this book will often be used to support claims for compensation for various health problems. The best use of this reference for that purpose may be as follows, in this order for most claims for compensation or cases that are litigated:

1. As guidance to the initial merit review of a case, when it is determined whether or not the claim is plausible
2. As guidance to *general causation*, as an introduction to the literature that may or may not support a particular claim, as a springboard for further review and literature search

3. As guidance to *specific causation*, the evaluation of the particulars of the individual case to determine if the features of the case are consistent with the known risk profile
4. As guidance to possible grounds for rebuttal or challenge of a particular case.

New publications on disease risk and firefighting appear almost weekly. When using the book for the purpose of supporting a compensation claim or litigation, it is essential that the current literature be searched, in addition, in order to present current evidence and ensure that an important new reference has not been overlooked.

The new expert or new legal counselor should not skip over the chapters on epidemiology, interpretation, and presumption in order to jump to the chapter on a particular disease risk. These chapters provide vital background that will help prevent mistakes in preparing a case and are guides to reading the highly technical evidence derived from epidemiology and toxicology.

In this book, risk estimates will normally be presented as they were reported in the original paper. SMRs are given to three places, without decimals in the text when they are expressed as percentages (SMR%) but converted to decimals in the table in the Appendix for easier comparison. SMRs may be considered to be equivalent to relative risks. Relative risks are given as decimals, with no qualification. Odds ratios are given as decimals and identified as such. 95 % confidence intervals follow the point estimate, in the usual format (point estimate; 95 % confidence interval lower bound, upper bound), as in (RR 1.05; 0.45–2.08). The Appendix presents risk estimates for multiple-outcome population studies relevant to cancer.

www.teeguidotti.com Tee L. Guidotti

# Chapter 1
# Orientation

**Tee L. Guidotti, Alex Forrest, Michel Lariviére, Zsuzsanna Kerekes, and Danielle Valcheff**

Issues of health risk and fair compensation in the fire service are issues of occupational fairness, respect, and justice. These issues bring up ethical, legal, occupational health, and public safety issues that are concrete, urgent, and almost universal in the fire service. They are also operationally important, because they affect response at the fire scene, preparedness, disability and therefore availability of firefighters for response, income security, and costs. Protecting those who protect the public is not only about the firefighter: it is also about ensuring that the firefighter will be there and fit to protect the public.

This book brings together previously scattered information on the health experience of firefighters. It is intended to be used primarily as a resource for prevention, for research, and for documentation in establishing "general causation", the evaluation of probable cause and effect in legal matters. For these purposes, the book is designed as a point of departure, not an encyclopedia covering every topic. It is incumbent on the user to check the recent literature to determine if there have been new developments and to investigate alternative explanations and risk factors. This book has also omitted a detailed description of firefighting technology and procedures, because this information is readily available elsewhere.

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It is expected that this book will be heavily used as a reference by scientists studying firefighters, by fire service managers with responsibility for occupational health and prevention, by lawyers and workers' compensation case managers to determine the merit in compensation claims and for litigation, by experts who are retained to render opinions on the merits of a case or claim, and sometimes by physicians assessing the plausibility of a relationship to work in a firefighter patient. It is difficult for any book to meet all these expectations but there is value to bringing the material together in one place to be shared, examined, and argued over from different points of view.

By prior agreement among themselves, the authors of this book take responsibility only for the content of their own chapters. Any one author may or may not agree with something specific that another author has written in another chapter, but all agree on the importance of the subject. There may be instances in which two authors or sets of authors see an issue differently, and that is part of the process of gathering the evidence and searching for the truth.

## How to Use This Book

This book has been composed to present the evidence for health risks among firefighters. It has also been written to provide assistance for non-scientists in interpreting the evidence. It is intended for guidance, only, and should always be supplemented by up-to-date information.

This Chapter provides an orientation to issues in the health of firefighters. Readers should not skip this chapter, even if they are already familiar with firefighter health issues and have some understanding of the technical issues.

Chapter 2 orients the reader to what it is like to be a firefighter, to be on the receiving end of serious risks to life and health. It provides a sense of the depth of concern and frustration in the firefighting community, and an insight into the motivation for their advocacy for recognition of health risks and fair compensation. Chapter 13, the last chapter, returns to this spirit with a personal history of presumptive legislation in the Canadian province of Manitoba, where the current movement to achieve legislated presumption began.

Chapter 3 is a primer on epidemiological research. Readers who are unfamiliar with statistics, epidemiology or social sciences research should read this chapter first, before attempting any of the more evidence-based, technical chapters dealing with population studies (Chaps. 4, 6–11). All epidemiological risk estimates are just that—estimates—and represent the experience of the populations being studied. In this paper, the risk estimates will normally be presented as they were reported in the original paper. SMRs are given to three places, without decimals in the text when they are expressed as percentages (SMR%) but converted to decimals in the table in the Appendix for easier comparison. SMRs may be interpreted similarly to relative risks. Relative risks are given as decimals, with no qualification. Odds ratios are given as decimals and identified as such. 95 % confidence intervals follow the point estimate, in the usual format (point estimate; 95 % confidence interval lower bound,

upper bound), as in (RR 1.05; 0.45–2.08). The Appendix to this report presents risk estimates for multiple-outcome population studies, mostly relevant to cancer.

Chapter 4 is an elaboration on Chap. 3 and is intended primarily for the expert witness. It provides a framework for interpreting the evidence, and bringing the application of epidemiology from its traditional role of confirming (or contradicting) general causation to a source for informing special causation. The approach outlined in Chap. 4 has been used chiefly in Chap. 6, on cancer, where it best applies.

Chapter 5 is an overview of toxic hazards in firefighting. Toxicology is a complicated life science, with many complexities and variations. The presentation of chemical hazards in this chapter does not reflect this complexity because it must emphasize disease risk and outcome. The chapter contains an Appendix written for the reader who is unfamiliar with the principles of toxicology. It serves as a primer, an introduction to the field for those who need to read further but have little or no previous background in the field.

Chapters 6 through 9 deal with classes of health outcomes and their known hazards in firefighting. These chapters lack detailed discussion of non-occupational hazards and health risks, however. When they are used in preparation for evaluating an individual case or to justify a policy of rebuttable presumption, therefore, they must be supplemented by a comparably thorough analysis of other risk factors: lifestyle (including but not limited to smoking and obesity), family history (which is only an imperfect indication of hereditary predisposition), other occupational or avocational exposures (such as moonlighting jobs, hobbies, and military service), and personal medical history (such as asthma). The wise expert will use these chapters to understand the issue and then search the literature for new information (since 2014) and to assess the potential contribution of other causes.

Chapter 12, and one section in Chapter 4, deal with presumption. Chapter 12 deals with it as a concept in law; the section in Chap. 4 discusses the epidemiological rationale behind establishing a presumption. Neither is intended to substitute for expert medical or legal advice. Chapter 13 concludes the book with the inside story of how Manitoba led the world in developing presumptive legislation and provides a case study and model for advocacy on behalf of firefighters.

## Varieties of Firefighter

All firefighters are not the same. Firefighting is not a single occupation, although many firefighters are cross-trained. For example, municipal firefighters are cross-trained in fighting brush fires and often aviation fires, as well as emergency medical services.

There are three major categories of firefighters with respect to exposure assessment: municipal firefighters (professional or volunteer), industrial firefighters (who provide fire and rescue services in facilities such as mines, refineries, and chemical plants), and wildland firefighters. In addition, World Trade Center first responder members of the Fire Department of New York constitute a subgroup of municipal firefighters that experienced a much different exposure regime from other firefighters [1].

## *Municipal Firefighters*

Municipal firefighters are the principal topic of this book. They are firefighters who respond primary to structural fires in settled areas, such as cities and towns, in organized fire departments. Their exposure is defined primarily by the chemical and combustion characteristics of the structures and their contents.

There are two subclasses of municipal firefighters, professional and volunteer.

Professional firefighters are extensively trained, paid a salary, and are considered municipal employees. They are usually unionized and are on "first call" for fire alarms. This book primarily reflects their experience. In recent years, an increasing number of municipalities have required firefighters to assume additional responsibilities as emergency medical technicians, with cross-training in paramedical services. This is in part a response to the decreasing number of serious structural fires in major cities. Professional firefighters usually enjoy prestige and visibility in their communities.

Volunteer firefighters are also trained but not drilled as often. They are paid by the hour or by the call and do this service on their own time or time away from their regular jobs. In larger communities, such as cities, volunteer firefighters are often second-call, reinforcing the professional firefighters if needed, but primarily staff the fire hall when the professional firefighters are out on an alarm, so that the community is covered in their absence. Volunteer firefighters are motivated by a desire to serve and interest in the occupation, and they also enjoy recognition and prestige in their communities. Often, service as a volunteer firefighter is a point of entry into the fire service that leads to a career as a professional firefighter.

In some cities, smaller communities and rural areas, the volunteer fire department is expected to take all calls. Volunteer firefighters may have backup from county fire services but these are often distant, may have less capacity, and the local volunteer fire department would be expected to control the fire situation until they arrive, which could take hours. In remote communities, the volunteer fire department may be all there is. Equipment in rural volunteer fire departments may be adequate but is often less modern or carefully maintained than in the professional fire service. It is not unusual for equipment to be purchased by local fundraising. Volunteer firefighters in small communities also enjoy visibility and prestige, since they are recognized as citizens who step forward in time of danger and community need.

There are important differences between the experience of professional and volunteer firefighters. In general volunteer firefighters do not experience the number of calls, time at the fire scene, or the intensity of fire exposure that professional firefighters experience. They do not have the same level of health insurance and benefits as professional firefighters, and in some jurisdictions may not even qualify for workers' compensation insurance coverage. The time spent as a volunteer firefighter is relatively small compared to their regular job. Professional firefighters often "moonlight" at other jobs on their days off, but their main job is as a firefighter.

Although professional firefighters have been well studied, volunteer firefighters have not, although what evidence there is suggests that risk levels for outcomes of interest are not as elevated as for professional firefighters [2] and probably more closely resemble the profile of their regular jobs.

## *World Trade Center First Responders (New York City, 11 September 2001)*

WTC firefighter responders are those firefighters who were in New York City on 11 September 2001 ("9–11") and who responded to the disaster at the World Trade Center (WTC) site. They are a relatively small group, about 2000, but have attracted great interest and concern.

Since the events of 9–11, there have been numerous studies of the New York City Fire Department (FDNY) members who responded to the WTC tragedy [2]. Anomalous types of airways disease have been reported, in particular, forms of bronchiolitis previously under-appreciated, and studies have suggested a higher than expected rate of sarcoidosis (a parenchymal disorder with only secondary airways involvement) among both WTC responders and other firefighters. However, it should be noted that the experience of WTC responders was quite different from that of other firefighters. Studies of WTC first responders cannot be used uncritically to draw conclusions about municipal firefighters in general.

The exact exposure mix experienced at street level and in buildings on the first day of the disaster is not known and never will be. The immediate consequence of the conflagrations that ultimately destroyed the twin towers of the WTC was to create a powerful updraft as heated air from the buildings rose over Manhattan. This carried gaseous components, including volatile organic compounds, and fine dust up and away from the area for several hours, reducing exposure at street level. The collapse of the buildings then contributed a different mix, quite unlike most exposures experienced by municipal firefighters. This was mostly pulverized calcined calcium silicate derived from concrete, which was, as best can be reconstructed, relatively coarse (>10 μm aerodynamic diameter) dust yielding a highly alkaline pH (>8) in aqueous solution, together with an unknown quantity (because it was not measured) of ultrafine particulate matter which would have quickly dispersed. (Almost all dusts of practical toxicological significance, in general occupational medicine and in firefighting, are acid-forming, not alkaline.) Silica and glass fibres were present, but relatively little asbestos. The dust carried other toxicologically relevant materials, such as metals, including iron (which catalyzes oxidation reactions at the cellular level), chromium (a familiar and allergenic contaminant of Portland cement), and, in certain samples, lead. Polycyclic aromatic hydrocarbons would have been generated in abundance but with a different distribution than usual (because of the intense heat of the fire) and the volatile components (including benzene) would probably have dissipated early. The dust was accompanied at street level by a gaseous cloud of unknown composition which rapidly dissipated and which was replaced with focal sources of combustion products from fires at ground level, among them products of burning jet fuel, which have characteristics similar to ultrafine particulate air pollution derived from diesel fuel. Adsorption of volatile agents onto the dust particles is not known but certain to have occurred and to be toxicologically significant because respirable dust would carry volatile agents into the deep lung with high efficiency.

There is no counterpart in conventional municipal firefighting to this unusual profile of exposure, although some individual components, such as burning jet fuel, may be present in industrial, aviation, and military firefighting.

The intensity of exposure was also exceptional, since surviving NYPD firefighters entered the burning structures or were trapped within the plume at its worst, always without respiratory protection (because SCBA could not last long enough for rescue efforts), and did not have adequate respiratory protection available during the extended overhaul phase for, in most cases, weeks. Whether or not the profile of exposure is responsible for the apparent acceleration in decline in lung function and increase in symptoms (most famously but inaccurately "WTC cough") is not clear but probable. Thus, generalization from WTC responders to municipal firefighters should not be attempted at this time. Examination of the WTC responders' experience may, however, lead to hypotheses which can be tested on municipal firefighter cohorts in order to test whether generalization can be supported.

The majority of WTC-exposed fire department rescue workers experienced a substantial decline in airflow over the first 12 months post-9/11, in addition to the normal age-related decline that affected all responders, followed by a persistent plateau in pulmonary function in the 6 years thereafter. The spectrum of the resulting pulmonary diseases consists of chronic inflammation, characterized by airflow obstruction, and expressing itself in different ways in large and small airways. These conditions include irritant-induced asthma, nonspecific chronic bronchitis, aggravated pre-existing obstructive lung disease (asthma or COPD), and bronchiolitis. Conditions concomitant with airways obstruction, particularly chronic rhinosinusitis and upper airway disease, and gastroesophageal reflux, have been prominent in this population. Less common have been reports of sarcoidosis or interstitial pulmonary fibrosis. Pulmonary fibrosis and bronchiolitis are generally characterized by long latency, relatively slow progression, and a silent period with respect to pulmonary function during its evolution. For these reasons, the incidence of these outcomes may be underestimated and may increase over time. The spectrum of chronic obstructive airways disease is broad in this population and may importantly include involvement at the bronchiolar level, manifested as small airways disease. Evaluations that go beyond conventional screening pulmonary function testing and imaging may be necessary to identify these diseases in order to understand the underlying pathologic processes so that treatment can be most effective [1].

The experience of the FDNY members involved in the WTC response, and of WTC responders generally, was unique. Their health experience must be considered qualitatively different from other firefighter populations, for these reasons [2]:

- The firefighters involved in the WTC response had all the exposures common to other municipal firefighters with the addition of a complex exposure regime unique to the WTC event.
- FDNY members are recruited from a very large applicant pool and have a rigorous preplacement qualifying program; as well, being a firefighter in New York carries high prestige in the occupation. These factors introduce a potentially strong selection bias at the time of hire, resulting in a potential healthy worker

effect that is most likely to be observed in the cardiopulmonary fitness of applicants.
- The FDNY introduced health promotion, fitness, and cardiovascular wellness programs earlier than most other fire departments, which introduces a potentially strong retention bias related to cardiopulmonary status [3].
- The FDNY and two academic-based programs each maintain a comprehensive and elaborate monitoring program for WTC responders, introducing a potentially strong screening bias when compared to other municipal fire departments.
- There is strong evidence that FDNY members are indeed a separate and distinct cohort in that a very large excess "total cancer" incidence has recently been reported for FDNY WTC responders, notwithstanding that sufficient latency for solid tumours has not elapsed since "9–11". At least part of this excess may reflect the screening bias mentioned above [3].

Lessons can be learned from the WTC responder experience, but these lessons must be interpreted cautiously. They cannot be considered representative of the experience of all firefighters. Unless otherwise indicated in the text, WTC responders will not be considered in the rest of this book.

## *Wildland Firefighters*

Wildland firefighters are specialized firefighters who suppress forest and bush fires. They represent a hugely important subset of the firefighting profession, and provide an essential line of protection for civilians in rural communities. Wildland firefighters are engaged in seasonal work fighting brush- and forest-fires, with a somewhat simpler (although still chemically complicated) exposure regime and probably differ from municipal firefighters in their risk.

Their exposure regime of wildland firefighters is not closely comparable to that of municipal firefighters or of industrial firefighters although it features some of the same combustion products. Exposure to burning wood (and brush) is chemically simpler and toxicologically likely to be less carcinogenic than burning structures with synthetic materials. On the other hand, wildland firefighters are deployed for days at a time, rather than hours, often must camp in areas where smoke is present, and for reasons of practicality are not required to use self-contained breathing apparatus (SCBA), but may do so on a voluntary basis if they are exposed to smoke from potential allergens or toxic substances such as poison ivy, poison oak, or poison sumac (also known as "thunderwood"). Therefore although the smoke they inhale is likely to contain a lower (but not negligible amounts) concentration of carcinogens, their exposure duration is much longer than would be typical for a municipal firefighter. However, they are also outdoors, which reduces exposure through ventilation and when upwind of the source.

In addition to their fire suppression duties, wildland firefighters also participate in fire prevention and tactical controlled burns, to reduce fire hazard. Controlled

burns and backfires set to consume fuel in advance of a forest or brush fire both expose the wildland firefighter to more smoke than would fire suppression activities alone. Reduction of fuel mass by application of herbicides, a common practice, exposes the wildland firefighter to some chemicals associated with increased cancer risk, particularly the phenoxyacetate herbicides, which are associated with elevated risk for non-Hodgkin lymphoma.

Health outcomes for wildland firefighters have not been studied as extensively or using the same analytical methods as for municipal workers and requires further research. However, many presumptive legislation acts include them as covered firefighters.

### *Industrial Firefighters*

Industrial firefighters include firefighting battalions at industrial plants, mine fire and rescue teams, oilfields, and firefighters specialized for aviation and shipboard fires. Industrial firefighters vary considerably in exposure opportunity. The literature on this diverse group is significant but not large.

Industrial firefighting often involves unusual or unusually severe hazards related to chemical hazards on site. Industrial firefighting carries obvious risks in the chemical, oil refining, and upstream oil and gas industries. Firefighters are often deployed from their regular jobs in a fire emergency but a large plant may have its own response team. Most plants rely on the local municipal fire department.

Mine fire and rescue is extremely challenging because of the need to carry out operations in confined spaces underground. Extraordinary strength and endurance are required. Because of these physical demands, the assignment is prestigious and attracts volunteers from the regular workforce who are exceptionally fit and motivated.

Except for full-time airport firefighting units (most of which are stations of the municipal fire department), industrial firefighters are generally not public employees and are not considered to be covered under presumptive legislation. They are covered by their employer's workers' compensation coverage.

## Firefighting and Health Risk: An Orientation

Firefighting is a lifestyle as well as an occupation. A firefighter must be very physically fit to perform firefighting duties safely and effectively. In major cities, they are usually required to pass stringent medical preplacement screening and functional capacity evaluations. They are then encouraged, and in many fire departments required, to stay fit by working out and through frequent training exercises. As a result, firefighters are an unusually strong, fit, and resilient population physically.

Fire departments today are much more racially and ethnically diverse than in the past. However, although barriers to the entry of women into the occupation have fallen, firefighting remains overwhelmingly male-dominated. One reason for this is that the job performance requirements are extreme and fewer women than men in the applicant pool have the required upper body strength and endurance. Because there are so few women in the fire service, equipment is often designed for men (androcentric) and thus poorly adapted to female height, weight and strength [4].

Firefighting is a skilled occupation, in addition to requiring strength and endurance. Beyond responding to emergencies at heights, in confined spaces, and/or in darkness, firefighters use and maintain specialized equipment, promote fire safety, investigate incidents, enforce safety standards, work with allied professionals (e.g., police, EMS), and the general public. Firefighters also need to remain current about new technologies, maintain adequate fitness levels, show good judgment, and tolerate uncertainty. Teamwork and leadership skills have a life-or-death urgency that is rarely seen outside of military combat situations.

This skill level and expertise does not necessarily translate to job opportunities after retirement or disability, however. Because of the physical demands, firefighting careers tend to be short but intense and sometimes end in permanent disability rather than scheduled retirement. Smart, and especially older, firefighters are always looking ahead to plan a life after retirement and what they could do after their firefighting career ends, especially if it were to end abruptly. Other than leadership and teamwork, firefighting requires skills that have little counterpart in any other civilian occupation. Unlike police careers, which lead naturally to security-related work after retirement, there are only a limited number of civilian jobs in fire-related industries. Therefore, most firefighters are very conscious of the need to prepare themselves to do some other job after their firefighting career ends. They often take second jobs (called "moonlighting") or start small businesses or educate themselves in another field (even going to law school) knowing that someday they may need to fall back on a second career option. Thus, firefighters, almost uniquely among blue-collar workers, as a group are heavily invested in lifelong education and open to learning new skills.

Work organization in the fire service features complicated work schedules designed to keep firefighters on standby at the fire hall for as long as possible to provide coverage without incurring expensive overtime but allowing time for training. The most popular schedule is called the Kelly shift system. If consists of three firefighter teams (which frequently change) working three shifts in a 9-day recurring pattern: first day on duty for 24 h, second day off, third day on duty, fourth day off, fifth day on duty, followed by four consecutive days off duty, for a total of 56 h per week. This system preserves the same scheduled days on or off duty for individual firefighters until there is a scheduled transition and can be adjusted by adding an additional "Kelly day" as needed. Firefighters like this schedule in part because it minimizes commuting time and provides long periods of uninterrupted time off, during which they can pursue other interests. (It is common for firefighters to have second, less demanding jobs.)

Alarms are not equivalent to fires fought. Only about a third of alarms represent fires; the others are false alarms or non-fire emergencies, especially in fire depart-

ments where firefighters also provide emergency medical technician (EMT) services. Alarms per month or year vary considerably among fire halls but firefighters rotate among different stations during their careers so as a practical matter the differences tend to average out, at least within a given fire department.

Firefighting has been characterized as long stretches of boredom interrupted by moments of sheer terror. This is accurate. Most of a firefighter's time is spent on equipment maintenance, training, and domestic duties in the fire hall, such as cooking in rotation for the rest of the team on a shift. (Meals at a fire hall are famously hearty and preparing a good meal is a point of pride; researchers have given much attention to nutrition and metabolism among firefighters, as described in Chap. 8.) However, when the alarm goes off, surges in epinephrine (the "flight or fight" hormone) and heart rate result in a strong physiological response to stress in preparation for deployment. (This is sometimes a precipitating factor for heart attacks, as noted also in Chap. 8.)

Firefighting includes hazardous [5] and demanding work in physically dangerous conditions. The risks of firefighting interact, particularly expressed in elevated injury risk in the presence of other health conditions.

Firefighters are exposed to dangerous environments that include explosions, smoke, dust, toxic chemicals, darkness, heat, confined spaces, and at heights, as well as unpredictable conditions that result from natural disasters (e.g., earthquakes, floods, storms) or man-made disasters (e.g., arson, motor vehicle accidents, industrial accidents). These environmental risks and their physical effects are described in considerable detail elsewhere in this book (Chaps. 2, 4–6). Some of the problem with injury risk arises from poor visibility, both due to smoke and to a small degree to the constraints of the face masks required for respiratory protection.

The physical demands of firefighting often impose a severe physical and metabolic burden during fire suppression and especially rescue. Firefighters, like soldiers in combat, must sometimes operate at the extremes of human tolerance with respect to heat, physical exertion, and agility.

Firefighting would not be possible without turnout or "bunker", gear: helmet, coat, pants, hood, gloves, boots, and self-contained breathing apparatus. (The total cost of a set of this gear is about $6000, of which SCBA accounts for more than half.) Current recommendations are that every firefighter should have at least two "suits", and that they be cleaned professionally; until recently, however, most firefighters have had only one suit, which they cleaned themselves at the fire hall. The protective clothing is designed to protect against heat, not chemical exposure (Fig. 1.1). Without highly effective heat protection, the extreme environment would lead quickly to heat stress, which in addition to the risk of heat exhaustion and heat stress predisposes to injury, exhaustion, stress, and greater exposure to inhaled hazards, since an increased rate and depth of breathing associated with exertion and heat increases exposure. Heat also increases blood flow to the skin and increases absorption of toxic chemicals from the skin, much of which comes from contaminated turnout gear.

Firefighters are exposed to a number of hazardous chemicals associated with combustion that are known to be acutely toxic, such as carbon monoxide and cya-

**Fig. 1.1** Firefighter in full turnout, or "bunker" gear: helmet with face shield, hood (protects neck and face), jacket, body harness, bunker pants and suspenders, gloves, boots, carrying a fire axe. (Photograph © Dennis Swayze, used by permission.)

nide. Chief among these are carbon monoxide and cyanide, and, for chronic effects, the polycyclic aromatic hydrocarbons and benzene, and fine particulate matter from fire smoke (which is distinct form fine particulate matter in ambient air pollution). The analysis must therefore go beyond superficial averages and probe more deeply into the evidence. Chemicals encountered in firefighting are particularly significant as toxic agents for cardiovascular and respiratory effects. The most significant for cancer risk are polycyclic aromatic hydrocarbons (PAHs), asbestos, benzene, 1,3-butadiene, trichloroethylene, dioxins and furans, and vinyl chloride; formaldehyde may also be significant. In addition, exposure to exhaust from diesel engines, primarily in the enclosed space of the fire station, adds additional exposures, including nitroarenes. A major change in risk level occurred following the introduction in the 1950s of combustible plastic furnishing and building materials known to generate toxic combustion products which may be carcinogenic. More recently, the introduction of flame retardants, which are questionably effective at best, has introduced new and poorly defined toxic hazards. Individual fires may contribute substantial additional exposure, however, such as polychlorinated biphenyl compounds (PCBs). These toxic exposures are associated with increased cancer risk and potentially chronic respiratory disease (which has presented a confusing picture over the years).

Self-contained breathing apparatus (SCBA) is effective in reducing chemical exposure by the airborne route to the extent that it is worn and essential to protect against smoke and toxic gases (Fig. 1.2). However, SCBA is uncomfortable to wear for prolonged periods, especially under hot and humid conditions. As a result, firefighters have been reluctant to put it on until they smelled smoke strongly, and still typically remove their SCBA as soon as the visible fire is suppressed. Unfortunately, more chemicals with carcinogenic potential are produced or released during the phase when the fire is cooler and embers are smoldering, so that firefighters engaged

**Fig. 1.2** Firefighters donning self-contained breathing apparatus (SCBA) in preparation for entering a burning building. (Photograph © Dennis Swayze, used by permission.)

in making sure that the fire is out (called "overhaul") are disproportionately exposed to carcinogens in fire smoke. Fire departments have been lax in the past about requiring and enforcing firefighters to don SCBA early and to keep it on during overhaul, because it is a considerable burden on the firefighter.

Firefighters' knowledge of these risks creates anxiety; that is, such knowledge is anxiogenic; provoking the fight-or-flight response and the general adaptation syndrome described later in Chap. 11. One way of coping with the potential anxiety is denial, simply pretending that this is just another job. More often, firefighters cultivate resilience through group solidarity and a culture of tightly-knit comaraderie. Dark humor is also an important coping mechanism (See Chap. 2). A few, however, experience dysfunctional responses or precipitation of acute events expressing intercurrent mental illness or preexisting susceptibility.

Exposure to psychosocial hazards in firefighting can result in stress, burnout, mental illness (and its associated stigmatization), and chronic pain. Alcohol abuse may reflect an ineffective coping style and firefighters have been shown to have higher rates of hazardous drinking and binge drinking [6–8], than the general population. Sleep disorders (i.e., circadian rhythm sleep-wake disorders, insomnia), are likely experienced more frequently by firefighters [6, 9, 10].

Much of the contemporary culture of firefighting still strongly reflects Irish culture and Celtic traditions (such as playing the bagpipes at funerals), because of the Irish ethnic presence in big city fire departments in the US, Canada, and the UK in the nineteenth and twentieth centuries. This cultural continuity, together with the shared work experience, allows firefighters, of any ethnicity, from almost any community in the English-speaking world, to fit in immediately in any other fire hall with the same tradition.

## *Research on Health Risks of Firefighters*

Serious research on the health issues of firefighters has been conducted mostly since the post-World War II era, with the first major epidemiological cohort studies, conducted by Ernest Mastromatteo in Toronto, appearing in 1959 [11, 12]. If one counts studies on carbon monoxide, burns, and other hazards characteristic of firefighting, however, the history of health research relevant to firefighters is much older. By now, a large number of studies have been undertaken in various fire services, most of them similar but many of them qualitatively different. Today the literature on firefighters is large and firefighters are among the best studied of occupations, comparable to asbestos workers. However, that so much work has been done does not mean that the epidemiological and toxicological basis for assessing the health of firefighters is complete. There are still substantial unanswered questions; indeed, it can be said that only now are the most important and deeper questions becoming clear. It means that because firefighters are better understood than other occupations, it is much clearer what the unanswered questions are.

Table 1.1 presents an analysis of research publications before and after 11 September 2001 ("9–11"), the day of the terrorist assault on the World Trade Center (WTC) in New York that led to the entrapment and death of 343 firefighters, as well as 60 police and 8 emergency medical technicians, and destroyed the city's central coordinating center for emergency response. There has been a significant increase in the number of published articles on firefighters since then. This was observable in a variety of databases consulted for this chapter (Table 1.1). About 80 of these publications are specific for WTC responders. The rest of the increase represents to closely interrelated and overlapping trends: one is an increasing awareness of the issues and growth in research support for studies of firefighters, and the other is increasing awareness that firefighters are a model population for many health issues that are not unique to firefighting but that are characteristic of the occupation. Thus, since 9–11 there has not only been a number of useful studies on hazards but an even greater outpouring of useful and often imaginative studies on cardiovascular disease, psychosocial risk factors, resilience, nutrition and lifestyle, and fitness, all conducted on populations of firefighters. With grace and generosity, firefighters have cooperated in these studies for the greater good, however intrusive they may seem.

Most large studies on firefighters are similar in design and face similar limitations on power for rare outcomes; this characteristic has led to the popularity of meta-analysis as a way to discern trends and certainties. However, the core original studies also have their own characteristic strengths, weaknesses, firefighter populations, communities from which they are drawn, timeframes, local patterns of occupational hazard such as housing stock, and methodological nuances, sometimes subtle, that make them different. These differences are valuable because they can be used to drill down to investigate particular issues by examining subgroups, exposure-response--> relationships, anomalies, and confounding by smoking. The incremental addition of increasingly well-designed, larger, and well-conducted studies on firefighter health has been welcome, even though they do not always provide the same level of detail in analysis as earlier studies.

Meta-analysis has been performed in an effort to overcome some of these limitations [13–15], with limited success. The experience applying meta-analysis to studies of firefighters has not been satisfactory overall, in our opinion, and this approach does not provide sufficient guidance for individual cases [16]. In addition to combining data from many studies, meta-analysis also combines their errors and biases. It is suggested, and argued in Chap. 4, that these issues represent a class of problem in occupational epidemiology that is best approached rigorously by examining the structure of the problem outcome by outcome.

One of the most important issues in research on the health effects of firefighters is quantifying exposure. As noted, alarms are not necessarily fires and some firefighters may be involved in unusually intense or qualitatively different fires (for example, involving a chemical plant or chemical warehouse) that are not separately recorded or identified in the record. Duration of employment as a firefighter is usually all that is available to quantify exposure, but this measure is confounded by age, seniority (affecting rotation and job assignment), and era, by which is meant major changes in the technology of firefighting (steadily improving), quality of personal protection (general compliance with SCBA protection being fairly recent and still incomplete during overhaul), and constituents of fire smoke (synthetic polymers becoming widespread and abundant in the 1950s and 1960s. Fire departments also have many formal and informal means of protecting unfit or disabled firefighters, in order to protect seniority, years counting to retirement eligibility, and family income security. Firefighters who cannot perform all the duties of their job assignment have, variously over the years, been informally protected by their mates and kept away from the fire scene, assigned to "light-duty" jobs beyond the norm and sometimes for many years (light duty is usually reserved for temporarily disabled firefighters during their recovery and rehabilitation), or to special employee units for the partially disabled created in negotiated contracts. For all these reasons, years of service is a highly imperfect guide to exposure among firefighters, either individually or in groups.

**Table 1.1** Research on firefighters before and after 11 September 2001

| Database | First publication | Number of articles *prior* to "9–11" | Number of articles *after* "9–11" |
|---|---|---|---|
| PsychINFO | 1942 | 213 | 513 |
| PsycCRITIQUES | 1992 | 8 | 18 |
| PubMed Central CANADA | 1943 | 40 | 122 |
| Web of Science | 1955 | 556 | 1602 |
| PsycTESTS | 1977 | 3 | 5 |
| Academic One File | 1977 | 584 | 1989 |
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| Science Direct | 1922 | 2411 | 4794 |
| Total | – | 6490 | 18,236 |

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# Chapter 2
# On the Receiving End: Being a Firefighter

**Alex Forrest**

There is no doubt that the issue of occupational cancer and firefighting has gained attention around the world and it is not going away. Fire fighters everywhere are fighting fires that contain carcinogenic chemicals in the smoke and they are dying of cancer because of these exposures. There is no way we can eliminate this danger from firefighting and as our society becomes more and more reliant on plastics and chemicals this danger will be increasing.

There are many examples of an all-encompassing single incident that can be so intense and produce so many toxins that it can be shown to have a connection to occupational health ailments. One glaring example of this is the most traumatic and one of the greatest tragedies for our union and the fire service, that being 9/11. On that day on September 11, 2001, we lost 343 fire fighters at the scene and later hundreds of fire fighters and other workers were diagnosed with and some have died as a result of occupational health issues. These were related to the work they did on the pile for weeks and months in the aftermath, in the cleanup and recovery operations related to that disaster. It is such a cruel fact that the tragedy of occupational disease is still taking good people away from us.

We must always remember that when a fire crew pulls up to a fire and is about to go in to attack that fire, they do not know what is in the building they are going to enter. The fire fighters do not know what fluids, chemicals or other hazardous materials are being stored in that building. Firefighters know that cancer causing agents are the by-products of normal house fires but there may be other materials involved that firefighters do not know about. Many times firefighters will never know what chemicals they were exposed to and as such the dangers firefighters face from carcinogens are likely underestimated. Firefighters never know the level of exposure that we have put ourselves through in the duration of our careers as fire fighters.

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As a side note, in almost every jurisdiction in North America, every profession except firefighting has some provision for workers so that they do not have to accept unsafe work, called “the right to refuse”. This is true for almost all professions and although firefighters may have this right on paper, in actual practice firefighters do not have this right. If fire fighters refused unsafe work, we would never go into a fire. All fires are dangerous. If firefighters do not respond to fires, emergencies, or disasters, who would?

Firefightinghas become more dangerous and firefighters still do not have gear that can protect firefighters from all of the hazards of the job.

## Toxic Hazards on the Job

There is no such thing as a harmless, minor fire. At every fire firefighters attend to firefighters come into contact with deadly carcinogens and every fire gives off harmful chemicals such as benzene, soot (PAH’s), formaldehyde, diesel particulates and an assortment of other deadly gases. Over time, this contact with carcinogens causes occupational cancer in fire fighters. It is not one fire that kills us, it is the hundreds of fires that firefighters attend to over the course of our careers.

Even though firefighting gear has become more advanced and has improved our safety two fold in the last few years, the toxicity of fires has arguably increased five times or more when compared to what it was 10 years ago. We believe that this is due to the increased use of plastics and fire retardants.

It is an undeniable fact in today’s society that dozens of new plastics are coming onto the market every year. Building construction throughout the world has gone from wood based to composite construction, which uses more plastics and glues, in order to combat the rising cost and scarcity of wood products.

When firefighters attend a house fire firefighters know that the house is filled with things that are largely made of plastic, glue, resin, and other combustible materials and when these materials burn they release a toxic cocktail of cancer causing agents. This is largely the result of the “plastic society” in which we all now live. The houses and buildings firefighters go into are composed of large amounts of plastic, glue and resin that are being used as alternatives to wood. Electronics are also a part of our society and when these electronics burn the wiring, the components and plastic housings release deadly toxic smoke.

One aspect of the job that is so dangerous to firefighters is likely the most ironic. For years firefighting organizations and fire departments promoted the use of fire retardants as a way to minimize the impact of residential fires. What is ironic is that these fire retardants actually have little impact in delaying fire but are one of the most dangerous parts of modern day fires. Fire retardants have been promoted by chemical companies as a way to minimize the effect of fires but firefighters are just now learning that there is no scientific basis for the effectiveness of flame retardants. However, there is a scientific basis to indicate that these chemicals are making fires more toxic and are likely responsible for many of the high rates of cancer in fire

fighters. Further to the ironic nature of this issue, fire fighter organizations were convinced by the chemical companies to assist in the promotion of these useless but extremely toxic chemicals. The entire sad story has been documented in a three-part expose in the *Chicago Tribune*, with additional investigative reporting in the same newspaper [1–4].

The first thing to understand about the cancer risk to fire fighters is how firefighters come into contact with these cancer causing elements. Firefighters do not just come into contact with cancer causing smoke and soot while they are fighting the fire. They encounter it when they are entering and leaving the building and even after the fire is extinguished, the danger is still there.

There are three main ways in which the chemicals that are released during a fire go into the fire fighter's body and these are ingestion, inhalation, and skinabsorption. Ingestion is the minor part, although some chemicals might be swallowed, especially if a firefighter does not wash hands before eating. Much more important are inhalation of airborne particles and gases and absorption through the skin. Inhalation has been recognized for as long as people have been worried about the health of firefighters, but skin absorption has only recently come to be appreciated as important.

It is generally accepted that there will be a myriad of chemicals in a house fire and firefighters must always remember the synergetic effect of chemical mixtures and toxicity. One chemical plus another chemical is not two chemicals in regards to the hazard level. Rather one chemical plus one chemical might result in three or five times the level of carcinogenic exposure, depending on the chemicals involved and how they interact with each other. The creation of a whole that is greater than the sum of its parts is called the synergy effect and with every chemical that is added to the mix it gets more dangerous. When firefighters see the different colors in the smoke firefighters just look at each other and say, "There is some bad crap in there".

## Turnout Gear Gives Limited Protection

The world's fire services have not invented firefighting protective equipment or clothing that will completely prevent exposure to cancer-causing agents.

Firefighters have very strong guidelines for structural firefighting protective clothing as detailed in The National Fire Protection Associations (NFPA) guidelines and code 1851 (NFPA 1851 [5] Standard, 1971 [6] and 2014). Firefighters have some of the best protective gear in the world and firefighters have some of the highest standards possible for the care and maintenance of our clothing, but turnout gear is designed to protect firefighters from ambient heat. With turnout gear firefighters are able to withstand upwards of 1000 degrees Celsius and even higher temperatures for short periods of time.

Turnout gear does not protect firefighters from chemical exposure to occupational carcinogens. Our firefighting gear has to be able to breathe when firefighters fight fires so that the heat buildup in the clothing is able to dissipate. If this buildup

of heat inside our gear was not able to escape then heat stress would kill us. In order for the gear to protect us from ambient heat it must be able to breathe; it has to release the heat out of our gear or firefighters would have catastrophic heat-related injuries and deaths from heat stroke. If the material can breathe and let heat out then it will also let air in. It allows the atmosphere from the fire passes through the gear to our skin; air that contains cancer-causing toxins.

If our gear breathes then it allows deadly carcinogens to come through our gear and they ultimately end up on our skin and are then absorbed into the body. If our gear and our clothing has the ability to breathe it allows carcinogens from the fire environment to pass through the material to our skin.

Every fire fighter who has ever fought a fire has taken a shower 3 days later and noticed that the water is still black as it runs down the drain. This is after multiple showers both in the time immediately after the fire and in the following days. This deposited soot contains and is representative of the deposition of chemical carcinogens on the skin, where they can be absorbed into the body.

These deadly chemicals are then absorbed through the largest organ of our body, our skin, just as airborne chemicals and particles are absorbed through the lung. The body then attempts to process these deadly chemicals and remove them from the blood, urine and vital organs. It is no coincidence that some of our strongest connections to cancer are through these filters of the body such as the bladder, kidney and the blood itself, as well as the brain and testes, that have some of the highest proportionate blood supply in the body.

The rate of skinabsorption in a fire fighter's body increases as skin temperature increases. Our skin temperature begins to increase almost as soon as firefighters put on the gear and the psychological and physical demands of firefighting further increase the skin temperature and therefore the rate of absorption. The areas where firefighters have the highest rates of absorption are found around the jaw, the neck and the crotch. These are all areas where our gear is most susceptible to carcinogenic penetration, due to openings.

Fire fighters have very good respiratory protection in the form of SCBA (Self Contained Breathing Apparatus), which allow us to function in a hostile environment with heat in excess of hundreds of degrees Celsius. Nevertheless, firefighters breathe in toxic smoke as firefighters put our gear on and then again when firefighters take it off as contaminated gear. After a fire, firefighters are filthy and our gear is covered with soot containing toxic chemicals. This is unavoidable because as firefighters take the gear off it is off-gassing the carcinogens that are in the soot which is now embedded in our clothing. Firefighters breathe that in as firefighters touch our gear and our faces and inevitably ingest this material as well. On a positive note both ingestion and inhalation can be minimized by proper SCBA procedures and adherence to the rules of using breathing apparatus not only during the knockdown phase of the fire but also during the other phases such as salvage, overhaul and fire investigation.

The bottom line is that firefighters have little protection from contact with the deadly carcinogens created in a fire and this contact, over time, leads to exposure sufficient to cause occupational cancers.

Fire fighters know the flaws of our firefighting clothing and, around the world, firefighters are working with manufacturers to try to invent clothing and materials that can protect firefighters from both ambient heat and toxins. The clothing needs to have the ability to breathe and yet create a barrier to the harmful by-products of fire.

Until the time comes that firefighters have such gear it must be clearly stated that fire fighters are going into fires around the world every day and every time they step into that fire environment they are one step closer to contracting occupational cancer.

## Diesel Exhaust

The International Agency for Research on Cancer (IARC) is the world authority on what causes cancer. Diesel particulates are an IARC Group 1 carcinogen, meaning that it is known to cause cancer in human beings—that means us [7]. An important way to minimize carcinogen exposure is proper diesel extraction systems in all fire halls. However, for years Canadian fire halls had no diesel particulate removal systems. I remember from early in my career in Winnipeg, every spring firefighters would do a major cleaning of the fire halls and everything was coated with thick, black soot from the diesel particulates. It was on our apparatus floor, in the alarm room and in our living quarters. Firefighters were literally being exposed to carcinogens both at fires and in the fire halls.

I will never forget what happened one day when the television set in the fire hall stopped working. A person came in to repair it, took the back cover off and gasped to see all of the components covered with a black tar-like substance. That was diesel particulates. I always said that if diesel particulates do that to the inside of a television what are they doing to the insides of a fire fighter?

## The Family Bond

Firefightingis known to be one of the most closely-knit professions in the world. We truly are a family! We watch over each other at work, we watch over each other away from the job and above all when one of us is in times of trouble or is killed due to the work we do, we always look after the family. This tradition goes back hundreds of years. I believe this bond was the result of the horrific consequences of our job of fighting fire. The sheer depth of destruction and terror has a uniting effect upon all of us, brothers and now sisters in arms.

Firefighters in North America and Australia successfully deal with the need for proper financial support and coverage for firefighters stricken with occupational cancer through presumptive legislation [8].

We have been able to have Acts of presumptive legislation passed in the majority of jurisdictions in North America through politics and science, but the people who put a personal face to our work have been the firefighters and their family members who have had to deal with this issue personally.

I could literally write a book about the many spokespersons who have assisted us in convincing the politicians to pass proper protective laws for firefighters who have been diagnosed with occupational disease. From Ross Lindley in Australia to Joe Adamkowski in Thunder Bay, Ontario to George Hemming in Alberta, to the many spokespersons in Manitoba including an army of widows who would not take no for an answer such as Angel Stoyko, Lynn Davidson, Janet Sabourin, Nancy Klassen, Debbie Woodman and Gerry Schedler as well as Janet Reed from Western Australia. Janet bravely told her story to the Australian Senate Inquiry that led to presumptive legislation in Australia [8]. One of my great friends in the union movement, Robert Hall from British Columbia, was not only a spokesperson but also a labour leader who assisted in having his province pass presumptive legislation. However, there is one person whose story best explains why we owe these spokespersons so much and that is Rick Stoyko. Firefighters from all over the world have him to thank for their presumptive legislation. I think it is important for me to tell his story as it best exemplifies the firefighters who put themselves out there in public, not for their own benefit but for the benefit of their fellow firefighters and their families.

## We Laugh Not to Cry

One aspect of life as a firefighter that must be understood in order to understand us is the importance of humour in a firefighter's life. As you read this you will see that there is sometimes a type of dark humor. There is a reason for this! Humour can be one of the most effective ways in which to deal with stress and shock. It is a way to share feelings with fellow brothers and sisters.

I remember watching an episode of "Rescue Me" when the firefighters were around the table and talking about "ass cancer". It was as tragic as it was funny, but it was reality and that conversation could have taken place in any fire hall in the country. In my opinion, if we didn't have humour as a coping mechanism our rates of post traumatic stress disorder (PTSD) and other issues would be much higher.

Firefighters deal with death almost daily; we deal with death in ways that members of the public never experience, death not only as a result of fires but also from heart attacks, strokes, MVAs, murders, suicides and the list goes on. These deaths are not confined to any age, as we deal with the deaths of the very young to the very old. We also deal with death as a profession as dozens of firefighters die every year in North America. There are large multiple line-of-duty deaths such as our "fallen 19" from Prescott, Arizona, and the recent incidents of firefighter deaths in Texas, but we also have single firefighter line-of-duty deaths almost daily. Throughout North America firefighters die of occupational cancer at an alarming rate. I have been to too many firefighter line of duty death funerals and one thing that I have said

to my loved ones is, at my funeral do not play the bagpipe rendition of Amazing Grace. I have come to hate that song!

What comes out of all this death? The humor of the job. We joke a lot about things that are not funny because if we cried we could not do our job.

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# Chapter 3
# Epidemiology

**Nancy E. Lightfoot**

The principal discipline and methodology to assess risks to the health of firefighters has been epidemiology, a discipline that focuses on the distribution and risk factors for various health outcomes in a population. A population can be a community or members of an occupation, in this case the population of firefighters. This chapter is intended to orient the non-technical expert to basic epidemiological terms, particular study design, their advantages and disadvantages; and their value in relation to identifying factors that cause health events in the specific population of firefighters; the processes used to interpret individual studies, and the overall strategy to assess the literature as a whole (considered in greater detail in Chap. 4).

## A Science of Populations

Epidemiologyis the discipline that focuses on the distribution and potential causes, or potential risk factors, of various health events or health outcomes of interest (for example, diseases, injuries, and conditions), and the size, or magnitude, of risk associated with potential causes of those events in *populations*, such as firefighters. Epidemiology relies entirely on statistics and sometimes measurement of exposure in real life. It is used to study groups of people, or *human populations*, never individual cases and for all practical purposes cannot be used to conduct controlled experiments that apply to causation arising from occupation. Although toxicology, clinical medicine, and exposure assessment each play a role and provide insights, epidemiology is the main approach used by those who study health risk in general and specific health risks among firefighters and the only approach that

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defines risk in occupational groups. Epidemiology derives estimates of the frequency of outcomes in the past and these can be used to predict risk in the future.

Epidemiology makes generalizations about risk based on population-based, or group, data and hence has limited ability to be applied at the individual case level for adjudications where results for groups can only be regarded as rough estimates for individuals. Epidemiology therefore plays a role primarily in general causation, to show that a connection with health is not only possible but likely both in general and for the individual case that matches characteristics of the population.

Epidemiological investigations to determine potential causes and the size of risk can take several years to complete and there is no perfect study, ever. The trick is to do studies that compensate for the inevitable flaws and minimize error. It is generally desirable to make decisions about risk based on the results from several studies. Compensation for specific health events may be awarded to individual workers based on eligibility criteria documented by epidemiology, and utilized by workers' compensation carriers and by law. Epidemiological data are also used to estimate safe levels of exposure to a hazard and to set occupational exposure levels (standards) for allowable exposure. Occupational health surveillance, which is monitoring populations to look for the appearance of disease, is undertaken to provide information on current risk levels and gives an indication of how effectively protective measures are working and whether new measures are needed. Surveillance is therefore of interest to governments, employers, unions, and workers to identify and describe patterns of the types of workers, in which locations, and how many of them are experiencing the health event(s) of interest. Surveillance is often conducted before planning focused studies that attempt to identify potential causes of disease, in order to better understand disease risk patterns and how they vary.

In epidemiology, something to which a firefighter is exposed is generally considered a potential cause of a health event if, given the potential for error, there are more cases of the health event among firefighters than in a relevant comparison population, and especially if one is 95 % or more certain that it results in the health event or if it can be shown to double the risk (explained in Chap. 4).

Occupational epidemiology case-control, cohort, and meta-analyses studies form a part of the information used by policy makers, legislators, and adjudicators. Occupational epidemiologists can contribute studies, analysis, and additional data to assist others evaluating the weight of the evidence about causes of health events for firefighters [1]. The world of policy makers, legislators, and adjudicators tends to move at a faster pace than for occupational epidemiology and can be influenced by the efforts of political and special interest groups [2]. While occupational epidemiological teams may engage such groups, their focus is to look beyond agendas and to conduct and interpret high quality scientific studies. While it can be time consuming, it is also helpful for occupational epidemiologists to be involved on interdisciplinary teams in the creation of firefighter compensation-related policies to ensure correct use, critique, and interpretation of available study information and evaluation of generated policies.

The high-level, contentious, and often adversarial world of policy makers, legislators, and adjudicators tends to move at a faster pace than occupational epidemiology but has expectations of what those answers will be and looks for findings favorable to their position, whether they are municipal government associations, fire chiefs, firefighter unions, or other stakeholders [2]. The function of research teams in epidemiology is not to confirm or validate the presence or absence of a health risk. It is to see if one exists. The epidemiologist needs to look beyond agendas and to conduct and interpret high quality scientific studies. The interpretation of the epidemiologic studies for application to compensation, prevention is the next step beyond epidemiological research and is discussed in Chap. 4.

## *Health Event Causation*

A *health event*or *outcome* can be death, cancer, an acute illness, a chronic disease, or any other event or condition that affects health. In studies of firefighters, information is most accurate for mortality (through death certificates) or cancer (through cancer registries, which cover most of the population in North America and Australia). Information on injuries comes largely from workers' compensation claims. Other outcomes are much less accurately recorded, usually not separately tracked by surveillance programs, and are therefore much harder to study.

Two important mathematical concepts in epidemiology are health event *incidence* and *prevalence*. Incidence is the number of new cases of a health event, divided by the population considered to be at risk, over some time period, usually but not always a year. Prevalence is the number of existing cases of a health event, divided by the population considered to be at risk, at some point in time or over a defined period, such as a year. Incident health events are newly diagnosed health events, but prevalent ones can be either newly or previously diagnosed. For the purpose of determining health event causation, the study of incident health events is preferred, because incident events are more recent in time, are not as subject to errors in recall, and are not affected by either treatment over time or death rates from unrelated causes.

A *risk factor* is an environmental condition or exposure, either occupational such as exposure to fire smoke and diesel exhaust, or nonoccupational, such as smoking habit or age. It is often a *potential cause* of the health outcome or it may relate in some other way to the potential cause, such as being in the same place at the same time. For example, if one kept track of firefighters' exposure to water, cumulative liters or gallons of exposure to water would be correlated to the risk of health outcomes in firefighters but not because exposure to water causes cancer, heart disease, or anything else. The job requires holding a hose and the amount of water a firefighter is exposed to just reflects how often and for how long they have fought fires and therefore how long they were exposed to fire smoke. Thus, water

can be counted as an uninformative risk factor because it would be statistically associated with the outcome but is not a potential cause of health events (except for scald burns).

The statistical relationship between a risk factor and a health event or outcome is called an *association* if there seems to be a connection reflected in correlation. Associations occur when there is a statistical correlation between the risk factor and the frequency of the event or outcome, for whatever reason. A potential cause (a risk factor) may be *causal*, that is, it may truly lead to the health event, a confounder (something that is linked to both the true cause and the outcome, like water, but is not the risk factor of interest), or it may be not cause the event and the association may have occurred either by chance (confusingly called *error* in statistics) or by a flaw in the study design or execution.

Sir Austin Bradford Hill, an eminent statistician, recommended that nine criteria be utilized as guidelines to assess whether an epidemiological association between an exposure and health event is likely to be causal [3]: strength, consistency, specificity, temporality, biological gradient, plausibility, coherence, experiment, and analogy [3]. Strong associations, where an exposure that doubles the risk or increases risk at least 1.5 times or more, provide stronger support for a causal relationship, although Hill also advised caution because causal associations may still be possible even though the association is small. A consistent association between an exposure and health event that has been observed several times by different people under different conditions also provides strong support for a causal relationship. (There is an on-going argument in epidemiology over how much consistency should be expected.) If a specific exposure results in a particular or a small number of health events, it is more likely to be causal. Temporality is the one essential criterion because the suspected cause absolutely must occur before the health event occurs in time. A biological gradient the demonstrates increased frequency of the health event, and often a more severe health event with increasing amount of exposure, is an important, but not required, causal criterion. If the association between exposure and health event is biologically plausible, that is consistent with previous literature, it is more likely to be causal, but its absence does not preclude causation. In relation to the strong criterion of coherence, the cause and effect relationship should make sense given the natural history and biology of the health event. Experimental validation of cause and effect is a very strong, but not required, criterion, when possible. If a similar association between a similar exposure and similar health event has been observed previously and proved to be causal, then by analogy, a cause and effect relationship is of greater likelihood, but this is considered a weaker criterion that is always trumped by direct evidence. Associations between an exposure and event can be causal or non-causal, and in general, the more Hill criteria satisfied, the more support for a causal association. However, it is critical always to consider the possibility of chance, versus causal associations, thus determining epidemiological causation is rather challenging [1, 3]. This is why epidemiology goes beyond *descriptive statistics*, such as how many cases there have been and what the rates are, and is heavy with *inferential statistics*, using calculations to take into account how likely it is that the findings arose by chance.

## Studies of Causation

Epidemiology demonstrates statistical associations, or the lack of them. By itself it does not prove causation but it can be used to rule out improbable causes and to point in the direction of what are probably true causal relationships.

Three types of epidemiological research health study designs are typically used to determine health event causation: cohort studies (usually the strongest), case-control (strong but limited to a single outcome at a time), and cross-sectional (intrinsically weak but useful for some specific purposes). Meta-analysis is a systematic way of combining studies into a larger and more comprehensive study, using well-defined rules and procedures. A checklist of criteria is provided in Table 3.1 to appraise and critically evaluate case-control and cohort health studies in the literature. Meta-analysis is a method by which results from case-control and cohort studies are combined to derive an overall estimate of risk for a particular outcome associated with a particular risk factor(s); it also has advantages and disadvantages. Examples given show how to determine if a particular health event may be caused by a risk factor common to firefighters.

There are a limited number of possible designs that can be used in epidemiology. They are distinguished by time (looking forward from when the exposure took place or in the present or looking backward from when the diagnosis was made), and by the use of a comparison group or *controls*.

There is an obvious omission in the list of epidemiological study designs used to investigate occupational risk among firefighters. The strongest epidemiological study design of all is called a *clinical trial*. Clinical trials are usually used to determine whether a certain intervention or treatment works, such as the efficacy (effectiveness under ideal conditions) of a new drug compared to existing drugs) but it is only rarely used to study causation except for documenting side effects of drugs and other treatments. It is almost never used in occupational epidemiology because it involves randomly assigning some subjects to an exposure (such as taking a drug under supervision) and others to a group that does not receive the intervention or treatment. Obviously this cannot be done for exposures that occur as part of an occupation and in which recruitment and job assignment is never random.

Cross sectional, or the "right now" survey or surveillance type of study that obtains information about exposure(s) and a health event(s) at a given point time, almost always the present. (It is possible to do a cross-sectional study for the past, from historical records, but this is not usually very useful.) Even when there is a suitable comparison group, a cross-sectional study cannot be used to identify risk factors because one cannot be sure whether the exposure or the health event came first. Cross sectional studies are mainly useful to obtain information on risk factors (such as smokingprevalence rates at a given time) or in the early stages of designing a more powerful study, to show where an epidemiologist might want to look harder. They will not be discussed further.

When the interest is in determining causation, or etiology of a health event, two types of epidemiological study designs can be employed, a case-control study or a cohort study.

**Table 3.1** Checklist for critical appraisal: occupational case-control and cohort studies

| | |
|---|---|
| 1. | How informative is the title of the paper? |
| 2. | Previous author experience in area of research and interdisciplinary research team? |
| 3. | Does the abstract flow well, list the study research question(s), methods, provide a good summary of the results and their implications, and not require re-reading? |
| 4. | Does the introduction provide a balanced summary of the relevant literature? Did they miss any important information? |
| 5. | Is/are the research question(s) provided clear and do they specify the geographic location of the study? Is a conceptual model to guide the research process provided and if not, what might have been used? |
| 6. | Was/were the appropriate study design(s) used for the research question(s)? |
| 7. | In the methods section, detail about the study process should be provided and be clear and specific. |
| 7.1. | Were inclusion and exclusion criteria provided for study participants? |
| 7.2. | Was the sample size calculation provided? |
| 7.3. | Was the selection of the sample participants described? |
| 7.4. | Was data collection done under similar conditions for cases and controls, or cohort members? |
| 7.5. | Was it clear what questions were asked of participants and whether any previously validated scales that would be helpful included? |
| 7.6. | Was it clear what and how exposure data were collected, under what conditions, and were they accurate and reliable? |
| 7.7. | Was the questionnaire piloted tested with a similar group as the participants prior to use in the study? |
| 7.8. | Where did the study undergo ethics review? |
| 7.9. | Were the appropriate statistical tests utilized? |
| 8. | In the results section, are table numbers provided and do they add up, apart from rounding errors? Is/are the study response rate(s) provided? Did they miss something that should be provided? |
| 9. | Does the discussion section summarize the findings and discuss them in light of other relevant literature, address the study response rate, study strengths and limitations, including the types of any potential biases and errors (for example, recall bias, selection bias, misclassification error, healthy worker effect bias, confounding, effect modification), and their effects, whether any conflicts of interest might have occurred, and are suggestions for further study identified? |
| 10. | Are there any implications from the study results and conclusions that might affect policies related to exposures and their risk for firefighters? |
| 11. | How well written is the paper? Is it free from spelling and grammar errors? |
| 12. | Who funded the study and could this influence the results and their interpretation? |

## *Cohort Study Design*

Most studies of firefighters use the cohort study design.

A cohort study starts with a group of similar people who do not have the health event(s) of interest and either follows them forward in time to determine if the health event(s) occur(s) or can start back in time and follow them over time to determine if the health event(s) occur. In contrast to a case-control study, cohort studies

can, if desired, study risk factors for more than one health event. This type of study can be referred to as the "thinking ahead or let's follow what happens" study design.

The main study question is whether exposure is associated with increased incidence of disease [4]. There can be various starting points in cohort studies. In a prospective cohort study, participants are classified according to an exposure(s) and followed, prospectively, into the future, over time to determine the occurrence of new or incidence cases of a health event(s) [5]. The key in a cohort study is to determine exposure or classification (by occupation) at the beginning of the time period. This can also be done by starting to follow subjects in the present but it is usually done by documenting their exposure in the past through records.

Most cohort studies of firefighters are of this type, called a *historical prospective study*. They incorporate both forward- and backward-looking approaches [2]. For firefighters, this is usually done by reconstructing duration of service, job title, and station assignment. Many occupational cohort studies rely on payroll data to establish the cohort. It is important to evaluate the quality of the information in a historical cohort, because the information was not collected for epidemiological study purposes.

The cohort study follows participants over time to calculate the rate at which a new health event occurs and identifies risk factors for the health even [4]. Cohort studies are considered superior to case-control studies because they can provide information about more than one health event and provide incidence rates of health events, thus yielding direct assessment of risk [2].

*Relative risk* is the ratio of the incidence rate of a health event in an exposed group to the incidence rate of the health event in an unexposed group. The relative risk of the health event is the measure of association used in many cohort studies. It compares the probability, or risk, of the health event associated with an exposure as compared with the risk of the health event in the unexposed comparison group, which is often the general population. Relative risk can be interpreted similarly to odds ratio interpretation in terms of statistical significance and confidence intervals, but is considered superior to the odds ratio as it is a direct estimate of risk based on incidence rates, rather than solely being based on the odds of exposure. A relative risk of 1, by definition, means that the risk or rate of the health event among the exposed is not different from the risk of the health event in the unexposed. A relative risk of 2 or more, means that that the risk is twice as high or more among the exposed versus the unexposed. A statistically significant positive association between the exposure and health event has occurred if the confidence interval surrounding the relative risk does not include 1.0, using the decimalized number to indicate actual measurement. If the relative risk is under 1.0, and the confidence interval surrounding the relative risk is under 1.0, the risk is statistically significantly lower in the exposed versus unexposed group and the exposure is considered protective from the health event [2].

*Attributable risk* is used in cohort studies to identify the difference between the incidence rate of the health event in the exposed group and the incidence rate in the unexposed group. Population risk difference (also called attributable risk fraction) is the difference between the rate of the health event in the unexposed and the

overall rate in the population under study and gives an indication of the benefit to the study population by modifying, or getting rid of, an exposure [2]. It is important (especially for the expert) to recognize that *attribution*, in epidemiology, means determining the fraction of cases in a group or population that are associated with a given risk factor. It does not apply to the individual caase. The estimation of the degree or proportion to which a given cause contributed to the risk of disease in an individual is called *apportionment*, which is a term from workers' compensation. The two are not the same.

Another measure of association used in some cohort studies, often those that are occupationally based such as studies of firefighters, are *standardized incidence and standardized mortality ratios*. They are called "standardized" because they take into account the age structure of the population. The standardized incidence ratio (SIR) is the ratio of a newly diagnosed observed health event in the population under study to the expected number based on applying information about the health event from a standard population. Similarly, the standardized mortality ratio (SMR) incorporates the number of deaths, versus newly diagnosed health events. Interpretation of these measures of association is similar to odds ratios and relative risks, where the SIRs of SMRs and their confidence intervals, most frequently 95 % CIs, are considered, but they are often multiplied by 100 and presented as percentages.

Advantages of cohort studies are: direct observation of risk (making them superior to case-control studies to determine risk of exposures), well defined exposure, ability to study uncommon exposures, and that exposure definitely occurs before the health event. Disadvantages include: they usually take much time, often many years if prospective starting in the present, and are costly and labour intensive, complicated and difficult to do. Participants can be lost to follow-up over time, especially if they die or move away, and exposures can be classified incorrectly [2].

## *Case-Control Study Design*

Case control studies are an important class of study design in occupational epidemiology but there have been relatively few performed on firefighters. This is because investigators are are primarily concerned with outcomes after exposure and cohort studies are a more efficient way of studying these. There are relatively few firefighters in the general population (compared, for example, to police) and so the case-control study design, which begins with a particular outcome, is less efficient for studies of disease causation among firefighters.

A case-control study starts by defining and selecting a sufficiently large number of people who have experienced the outcome or health event of interest (the cases), and then carefully selecting those who have not experienced the event (the controls) but who are otherwise reasonably similar to cases. This study design is usually used when the health event is fairly uncommon but one can be identified through a reliable source, such as hospital records or a cancer registry. The investigator then gathers information about both individual cases and controls, usually by

a questionnaire, on exposures of interest in the past, sometimes from many decades ago. The questionnaire may be administered to study participants in various ways (for example, in person, by telephone, by mail, or online, or next of kin if deceased). This type of study can be referred to as "the can you remember study," given that study participants must recall information about past exposures over time. The questionnaire used is generally not short and the number of items asked often presents a practical problem. The questionnaire may obtain information on medical history, residence history, occupational history, physical activity history, dietary history, and whatever else is required and cannot be obtained from records. This type of study compares the exposure histories of cases and controls to identify likely exposures, or potential risk factors, for the health event [5].

Accuracy is based on their recollections but it is much more important that the information be gathered in exactly the same way for cases and control than that every response be perfectly accurate. The reason is that if the study is administered in exactly the same way to both groups, small inaccuracies will cancel out when cases are compared to controls, but if the study is conducted with a systematic, regular difference that causes one group to report a risk factor more or less than the other, the comparison between the two will be thrown off and could affect the estimated risk much more.

In this type of study, cases may be matched, individually or as a group, to controls on certain variables, such as age, gender, and socioeconomic status, etc. to make cases and controls similar and exclude the influence of those variables on the health event. Alternatively, matching of cases may not be employed and these factors can be studied for their separate influence and, particularly, for interaction with the other risk factors. Whether or not matching is incorporated influences the type of statistical analysis needed in the study [5].

The *odds ratio* is a measure of association between the frequency of exposure and the frequency of health event that is calculated in case-control studies. In odds ratios, the odds (not the rate) of exposure reported by cases of a health event are estimated and compared to the odds of exposure in in controls, who have not experienced the health event. If the odds of exposure is the same for cases and controls, the odds ratio (OR) = 1. If the OR exceeds 1, then cases have higher odds of exposure than controls so the exposure is considered to be a risk factor. If the OR is under 1, then cases have lower odds of exposure than controls, so the exposure is considered protective. However, one must not only consider the odds ratio, but the confidence interval or range in which the odds ratio may lie when determining risk of an exposure. The odds ratio is of statistical importance, or is statistically significant if the 95 % (as this is often the level selected for statistical importance) confidence interval (CI) does not include 1. If the 95 % confidence interval is less than one, then the exposure is considered statistically significantly protective, but if greater than one, then the exposure is considered to statistically significantly increase risk. One must consider odds ratios with their confidence intervals, rather than simply trying to compare the size of odds ratios [2, 5].

Case-control studies are often fairly quick and easy to do compared to cohort studies, tend to be less expensive, and include fewer people than cohort studies.

The major criticism of case-control studies is that they usually rely upon the memory of study participants and may suffer from *recall bias*, because people who have experienced the health event (cases) are likely to be more concerned about their past exposures and may remember them more clearly than controls. If this happens, cases and control have systematically different memories about the past exposure and bias is introduced, as previously described [2, 5].

### *Meta-Analysis*

One type of study, called a meta-analysis, combines the results of several studies to determine the risk associated with an exposure(s). This can be done for exposures of interest in case-control and cohort studies of firefighters. However, errors in any study contribute to error in the overall interpretation. Similar studies can be combined into a summary statistic, but caution needs to be exercised if the studies are dissimilar and there are statistical tests available to measure the amount of variability among studies [6]. Considerable detail, such as exposure-response relationships and comparisons among job assignments, are also lost in meta-analysis because the data used from each study must be comparable. It is desirable to avoid including studies of poor quality in meta-analyses and to also consider the effects of publication bias, or the preferential publication of studies that identify a statistically significant outcome for the exposure(s) of interest [7]. Studies utilized in a meta-analysis can also be subjected the critical appraisal criteria presented in Table 3.1. Chapter 4 presents a more detailed critique of meta-analysis.

## Critical Appraisal

Just because a paper from a research study is published, this does not guarantee the quality of the study, results, and interpretation. Thus, an important skill for an investigator or expert to develop is critical appraisal of epidemiological papers. General critical appraisal skills are the foundation for determining what the literature actually says and how strong is the evidence. A checklist or steps that can guide critical appraisal for occupational case-control and cohort studies appear in Table 3.1.

From time to time, someone will read the literature and prepare a *review article* that summarizes what original research articles have shown. There are principles for evaluating review articles as well as original research articles. A *narrative review* summarizes the information based on literature selected by the author of the review while a *systematic reviews* identifies all available relevant literature found through search terms and covers the topic in a structured way. A meta-analysis is a systematic review that also combines the measures of association for the health event(s) into one summary measure based on risk estimates from several articles of high quality [8].

Review articles are always written from a point of view and with a purpose in mind. Thus, they are often of questionable value as evidence and often omit issues that were not of interest to the author. Anyone seriously interested in risk factor for a health event(s) must usually go to the original sources eventually and not confine their efforts to examining review papers alone.

General critical appraisal involves a critique of all relevant aspects of an original published research paper: abstract; introduction; the methods; the results; the discussion and conclusion; the references and sources of funding. Above all, the entire paper should be well written, easy to read and not require re-reading for understanding, and lack spelling and grammar errors. Evaluating a paper for the strength of evidence is essentially the same as doing so for general quality (Table 3.1).

## *Appraising Original Research Papers*

General critical appraisal can commence with consideration about the paper's authors. Quite often it is the title of the paper that first catches the reader's attention. It helps to be informative and interesting or catchy. Next, one may consider whether the authors had previous experience in undertaking and publishing research about the health event of interest. Previous experience is advantageous, but not required. How experienced is the research team and is it interdisciplinary? Previous research experience in undertaking studies, in general, can be valuable, particularly using similar study designs. Incorporation of an interdisciplinary research team is valuable in approaching occupational research questions and can promote the use of a wider perspective of research methods and interpretations. Moving onto the abstract, it should be easy to read, lack spelling and grammar errors, and not overly technical, flow well and not require re-reading, and provide information about the study objective(s), methods, results, and conclusion.

The "Introduction" of the paper should provide a balanced review of the existing literature. If it does not, that is an indication of potential bias. It should include discussion of studies that have already explored the influence of the potential risk factor of interest, including studies that show a "positive" finding and those that do not, and summarize the other potential risk factors that are important in explaining the health event of interest. The literature provided in the Introduction need not be complete or systematic, but should be of adequate quantity to be convincing to the reader and representative of the currently available literature.

Near the end of the Introduction, the research question(s) should appear. Research questions do not need to be stated in the form of a question, but should specify who was studied and geographically where. Although not always mentioned, the conceptual framework used to guide the research process, such as the biomedical model, should be stated as it is helpful to be aware of the process used to guide the work. The research question is important in assessing evidence for purposes of compensation because it is a clue to the limitations of a study. If the authors were not looking for something (for example, differences in risk associated

with job assignment), they probably did not find it and the study was probably not designed in a way that it could have.

The "Methods" section is where the author lays out the approach and procedures that were taken to conduct the study. Critical appraisal of the Methods section should include identifying the type of study design used and it is helpful to know why the design was selected. There should be consistent use of similar terms. A clear description of the people that were included in the study must be provided, that is, their gender, age, and criteria about the health events of interest (for example, prostate-cancer confirmed by a pathologist, first diagnosed in the last 3 years, in men aged 45–84). Similarly, a clear description of those excluded from the study (for example, people of certain age, inability to undertake the tasks required, those with a past medical history that could confound the results, etc.) must be provided, hopefully, along with the reasons for exclusion. For a case-control study, specific criteria for inclusion and exclusion must be provided for the cases and the controls and a cohort study should also such information for members of the cohort. Data collection from the cases and controls, or members of a cohort, should occur under similar circumstances [9]. The sample size of people required for the study should be calculated and it is helpful, and encouraging, if a biostatistician on the research team was involved in this task, as it can be complicated.

The Methods section should also identify the type of questions asked (for example, characteristics of the people involved, work history, residence history, dietary history, medical and medication history, smoking history, physical activity history, etc). and whether the questions are reliable over time, that is, have been shown to demonstrate similar results from the same people, under similar conditions, at various times, and in what populations the questions have been tested, answered, and gave correct answers. It does not negate the value of the study if the questions have not been tested over time and in other populations, but if they have been it inspires more confidence in the results. It is also helpful if the questions have been previously pilot tested before wider spread use. Furthermore, most information obtained in a research study from people, or human biological specimens, that will contribute knowledge for others, must undergo review by a research ethics board to ensure research benefits, either no physical, emotional, social, or financial harm, or harm that can be minimized.

When evaluating the methods used in a paper, one should identify the assumptions and reasons for choosing the statistical tests used, in order to determine that the tests have been used correctly, instead of under conditions that make the results unusable. For easy location, the name of the test and statistical assumptions can be typed into a search engine.

In the Results section of the paper, a summary of the results is provided, usually in Tables. These are then interpreted in the discussion section. When critiquing the Results section of a paper, it is helpful to determine if the number of people, or other units, add up in the tables, or if not, if there is an explanation provided for the discrepancy. If risk increases with greater exposure, it is more likely to suggest a causal association.

The Discussion section should discuss the results in relation to current literature, as well as identifying both strengths and limitations of the study, including the types

of any potential biases or systematic distortion of results from the truth [2]. Potential sources of error and bias should be identified and their potential impact on the study results discussed. There should also be some indication of suggestions for further study in the Discussion section. This is not just a plea for more funding from the authors: it is also a concise way of communicating where the gaps and uncertainties are in research.

The reference section should not contain errors so that readers can locate the sources used, if desired. There may also be an acknowledgments section to thank some that assisted with the work and clearly identify any study funders.

At the end of the paper, there is usually a statement about who provided the funding for the study. It can be more reassuring to readers if public funding from some government source supported the study, rather than funding that comes from a particular industry that might have a vested interest in the results.

## *Analysis*

The next step is to determine what the paper means.

There are two types of error that can occur in epidemiological studies and that can call the study results into question: (1) random errors and (2) systematic errors, or *bias* [11].

Random error refers to an incorrect result due to chance with sources of variation that are equally likely to distort study measurements up or down [10]. Random error affects precision of a measurement(s) and is directly related to the study sample and size: it decreases with increasing study size, is not consistent, and does not always occur in the same direction [11]. Random error is unavoidable, so the only option to prevent it is by ensuring that the study is large enough and that the classification of exposed and unexposed is as accurately as possible.

Bias, or systematic error, occurs when there is a problem with the study design or execution such that sources of variation distort the study results in one direction [10], affecting accuracy. Some degree of bias, usually small, is inevitable in epidemiological studies; it becomes a problem when it produces misleading results. Systematic error occurs consistently in a measurement(s), has a specific direction such that the measured value is either more or less than the true value, and hence decreases the believability of the study results and ability to generalize to other similar populations [11]. Bias can result in any stage of the study, from literature selection (which can mislead the investigator or influence study design), use of a particular conceptual framework, to participant selection, measurement processes, and even influencing what is published [11].

Sackett produced an extensive list of types of bias in studies concerned with health event causation [12]. A few common biases are:

- *Selection bias* occurs when subjects do not represent the population from which they are selected.

- The *healthy worker effect* occurs when disease or death rates for an employed populations are lower than for the comparison population, because people who can hold down a job (particularly a strenuous occupation such as firefighting) tend to be healthier than the general population from which they come [2]. The healthy worker effect introduces a bias in estimating risk because an elevation in risk from a risk factor can be offset or masked by a decreased risk among people who can tolerate the demands of work. For example, firefighters tend to be very fit compared to most other people, so their heart disease risk overall tends to be lower and this makes it harder to see the effects of occupational factors on heart disease when they are compared to the general population.
- Misclassification bias occurs when those with the health event or those without the health event are wrongly classified [5], or when exposures are incorrectly assigned for study participants as exposed and not exposed. If misclassification error is the same for both groups, either those with the health event (i.e., the diseased) or those without the health event (i.e., not diseased), or exposed and not exposed, it reduces the estimate of risk and may minimize a true risk. It is worse if it occurs in only one of the groups being compared (for example, if records are kept differently) and can either increase or decrease the observed estimate of risk [13].

Sometimes a variable other than the one believed to be the risk factor that leads to the health event, one that has not been considered, is actually responsible for concealing or distorting the putative relationship between the original risk factor and health event [6]. A confounder, described earlier, distorts the effect of an exposure or risk factor on a health event by another variable [14] and can make the association between the exposure of interest variable and health event variable appear more or less significant than it actually is and hence must be included in any model [6]. The classic example is cigarette smoking, which changes risk of lung-cancer and heart disease and can distort risk estimates if the population being studied smokes much more or much less than the comparison population. (Firefighters generally smoke less.)

An *effect modifier*, or interaction term, occurs when there is a different relationship between the exposure or risk factor and the health event depending on the level of another variable. Both confounders and effect modifiers can enhance or mask a causal association between the risk factor and the health event. The confounder(s) needs to be included in the model and for effect modifiers, results are usually presented separately for each level of the third effect modifier variable [14].

It is important to consider how accurate and reliable the measurement of exposure might have been, if actual measurements were made. The paper must also indicate how any exposures were measured (for example, in individuals, in a work area, or in both), using what methods, under what conditions, by whom, and how accurate the measurements were.

If the study is done well and the population that has been studied is broadly representative, it may be applicable, or *generalizable*, to other similar firefighter populations in similar situations. However, if it is unique to a particular situation or location, it may not be generalizable. For example, studies of the first responders in

the 11 September 2001 World Trade Center tragedy, while critically important in their own right, are not particularly informative for assessing the health risk of firefighters under more conventional circumstances. The exposures were just too different.

Another consideration in evaluation of epidemiological studies is to assess whether there is documentation of appropriate safeguards for protecting confidentiality and ensuring adherence to ethical guidelines. Ethics in epidemiological research cannot be taken for granted [15]. Studies that treat ethical issues as an afterthought or that are poorly documented may have underlying problems that affect research design, the rigour of data collection, or bias on the part of the investigator.

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# Chapter 4
# Interpreting the Literature

**Tee L. Guidotti**

Does working in a given occupational mean an increased risk of a certain disease? Did a particular disease in a particular person arise from a particular cause? Specifically, how does one demonstrate that a disease, even one recognized to be more common in a particular occupation, actually arose from exposures related to that occupation in a given individual? Or did not?

Conventional medical evaluation only rarely answers the question definitively for diseases. (It does so much more often for injuries, which are usually obvious.) The science of forensic toxicology has taken other directions, and has not given issues of causation analysis for compensation a high priority. Biomarkers have not been systematically developed for this purpose. It must therefore be accepted that with the technology and expertise available today, certainty cannot be achieved in many and usually the great majority of disease claims by medical means.

There is no "gold standard" for determining that a given claim for disease arose out of occupation, as a firefighter or any other occupation, as there is (ideally) for diagnoses in clinical medicine, and not all diagnoses are that clear, either. In the end, for all but a tiny fraction of health outcomes (such as mesothelioma in relation to asbestos exposure) the court or adjudicator cannot know with absolute certainty whether or not a disabling condition arose from exposure related to occupation. Therefore demonstration of a connection is necessary but the conclusion that the connection represents cause and effect is subject to interpretation. Since the absolute truth cannot be known, the question becomes what level of certainty is agreed upon and for what purpose. For the compensation system, as a broad generalization, society has decided through laws, regulations, policies, and common sense that that level of certainty is basically the weight of evidence, or "more likely than not", rather than scientific certainty. Some parts of the compensation system use the

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standard of "substantial contribution," which means a contribution to the total risk that, compared to other hazards, was not trivial.

In all compensation systems, the central question is whether the disease (or other adverse outcome, but disease is much more difficult than injury) would have occurred in the absence of, or "but for", the hazards associated with the occupation in question. That is the central question of the process known as "causation analysis" as it is undertaken by expert witnesses in these cases, who are primarily but not exclusively physicians.

Epidemiologists, especially, frequently contribute to the discussion on "general causation", as will be described. By identifying risks as elevated in particular subgroups, such as job assignments or by length of service, they also inform "specific causation" for the individual claimant. Of course, epidemiology is also essential for evaluating risks from occupations such as firefighting compared to other, nonoccupational risk factors.

## Causation Analysis

As a practical matter, causationanalysis rests, as it always has, on two elements: general causation and specific causation. The better informed and reasoned the causation analysis on both sides, the more explicit and defensible the judgment and the more likely it will be that the system will adjudicate "correctly", meaning that a claim will be correctly identified as meritorious or not. This is not exactly the same as demonstrating conclusively that the injury arose from the hazards or conditions of work. In the end, it is unknowable whether one person's disease was actually caused by one factor or another—common conditions have common causes, there is a spontaneous or background rate to almost every disease that can arise from occupation, and there is always an infinitesimal chance that a highly unlikely cause was responsible. So the idea of "meritorious" has to mean that it is more likely than not that the condition arose from occupation or that occupation contributed substantially, not that it can be conclusively established that it did. A conclusive level of certainty is unattainable, and even if forensic toxicology develops a new generation of tests, there will always be residual uncertainty.

*General causation* establishes that the disease outcome (or injury) does happen in a particular group or associated with a particular exposure, more often or more severely than in other people, and therefore was more likely to happen at the time of exposure in the individual case under scrutiny. General causation is established primarily by epidemiology, the science of disease and risk factors in populations. In the past, toxicology played a much bigger role but epidemiology now provides most of the evidence for general causation in these cases. Toxicology is classically the science of "poisons", and now is better understood as the science of adverse effects of chemicals and the body's response to them. Because it is much easier (and presents many fewer ethical problems) to study a response to chemical exposure in animals than in human beings, toxicology provides invaluable knowledge, but

knowledge that is often difficult to apply to human beings, especially at relatively low exposure levels. Today, epidemiology trumps toxicology in general causation because it provides information on human beings in large enough numbers (populations) to derive some statistical estimates with confidence. Still, if there is a question about the mechanism of response or there is no epidemiological study on a topic, toxicology often plays an important role.

*Specific causation* establishes that the circumstances of the individual case are consistent with the factors that are known to be associated with the disease in question and point to the problem arising from, say, exposures that the claimant is known or can reasonably be expected to have sustained. It asks the questions of whether the person actually had the disease it is assumed they had, why this particular person was affected on the job, whether there are other good reasons he or she might have gotten the disease, and whether he or she would have gotten the disease anyway, if the exposure due to occupation had not happened. Specific causation requires knowledge of any unusual susceptibility on the part of the claimant that might cause them to develop a problem where another person might not, and some idea of their risk as a person apart from their occupation for developing the disease. Specific causation therefore usually requires a detailed analysis of the claimant's work history, medical history and medications, sometimes family history (to identify susceptibilities that are presumed genetic), and medical evidence, such as laboratory tests (such as lung function tests for respiratory outcomes and blood tests for suspected toxicity by agents such as lead). The methods of specific causation are similar to those of clinical medicine but directed at answering different questions, and heavily reliant on toxicology.

One step in specific causation is to rule out or account for risk factors other than the one of primary concern in the case (usually the occupation or the occupation-associated risk factor). In the case of a legislated or policy-driven rebuttable presumption of eligibility for compensation, this may mean addressing the grounds for rebuttal. In many cases, the mere presence of a risk factor unrelated to occupation is enough to compromise the case for eligibility, for example lung cancer in a heavy smoker. However, the presence of a competing risk factor is not necessarily disqualifying. For example, a light smoker who is also exposed occupationally to a lung carcinogen may well have a meritorious case on the basis of interaction (for example with asbestos), may have a claim recognized in a system that only requires "substantial contribution" to risk of the occupational risk factor, or when it can be established that the "but for" argument applies (most smokers do not get lung cancer and so the question might be posed "but for the additional risk factor, would this claimant have developed the disease," a formulation that often applies when the causal association is less strong)

Even with the technical limitations, causation analysis could be much more "scientific" than it usually is. This is because, in technical terms, individual cases could conceivably be analyzed statistically by a method known as Bayesian statistics. Conventional, a priori, statistics, familiar to all scientists, predicts the probability that something will happen in the future because of the frequency that it happened in the past and uses this to ascertain how likely an event might have been when it occurred.

This is not completely logical in causation analysis because the outcome has already happened, so predictions in the past of future events are off point. However, such statistics are used all the time in causation arguments. Bayesian analysis takes a different approach. One is trying to work out the probability that a patient's particular condition represents a particular diagnosis (in general medicine) or, similarly, the probability that an injured worker's condition was caused by a hazard, looking backward. This can in theory be calculated for an individual case, but only if one has a lot of information about the association and about the frequency of the association between the hazard and the condition in the population in question as well as a priori risk (which could be estimated from general causation). This is complicated, uncertain because of the assumptions involved, and almost impossible to explain to a judge, jury, or adjudicator, unless they happen to be a statistician. That is why the Bayesian approach, although it would seem well suited to the problem, is never used in medicolegal arguments or policy. Instead, with the evidence available the expert and the system as a whole are striving for a reasonably robust standard of certainty. In most adjudication systems, this constitutes "the weight of evidence" or some variation or adaption of this rule.

## Standards of Certainty

In order to make a decision regarding whether a claim has merit, the evidence from general causation and specific causation have to be weighed and a determination made to a consistent standard of persuasion. The standard of is not the same for every legal or adjudication system. This standard of persuasion reflects just how convinced the adjudicator (judge, jury, hearing officer) is that evidence meets a standard of certainty. There are different standards of certainty in common use:

- *Scientific standard of certainty*. This standard says that experiments have been replicated, lines of evidence converge, anomalies have been explained, and that scientists are quite sure of what is the truth. (Until another study upsets everything with an anomalous finding.) It is far more stringent than 95 % certainty, the figure usually cited, because this is for one experiment and scientific certainty requires a whole body of evidence. It is therefore the most stringent standard of certainty and frankly unattainable in almost all legal actions or in adjudication. Although scientific proof regarding causal associations of firefighting would be ideal, it is unattainable for most health outcomes due to practical limitations.
- *Beyond reasonable doubt*. This standard implies that the evidence is mostly complete and overwhelming, and provides no room for doubt. It is applied in criminal cases but not in civil cases or adjudication because it is overly stringent and the evidence is almost never as complete as it would be in a criminal prosecution.
- *Presumption*. Presumption, as applied to compensation for occupational disease, is the policy that because the risk of a disease is elevated in a particular group, members of that group who develop the disease will normally be assumed to have

developed it as a result of the hazards or conditions of their occupation. Presumptions are usually *rebuttable*, which means that if there is good evidence in an individual case that the disease arose from another cause or for another reason, this can be used to argue against the presumption being applied to that case.

- *More likely than not*. This standard of certainty, with variations, is the usual standard for civil litigation and adjudication. It requires that the weight of evidence favors one side or the other. In the event that the evidence is in balance (i.e. seems to be equal in weight both for and against), there are rules for tipping the balance. In civil litigation, the "benefit of the doubt" is given to the defendant. In workers' compensation, the laws establishing most systems usually specify that the "benefit of the doubt" goes to the claimant, giving the injured worker a small advantage to make up for the practical difficulty in making the case, which falls most heavily on the claimant. However, in some systems the benefit of the doubt is not specified and so, under tort litigation rules, goes to the employer. Formally, if all other things are equal and the weight of evidence is perfectly balanced (as it never is), then a situation in which occupation was equally likely to cause a disease as nonoccupational factors (50 %, or even odds) would correspond to a doubling of the risk for the general population or reference.
- *Any elevation in risk*. As a practical matter, some systems accept evidence of any increase in risk as evidence for an association with occupation. This may seem arbitrary but in view of the difficulty in demonstrating an elevation that is indeed present, it is a way of expediting recognition of occupational diseases. A simple elevation in risk demonstrated for a certain occupation is behind many of the diseases entered in *schedules* for compensation, in which a list of diseases is recognized for presumption among members of the occupation at risk.
- *Substantial contribution* Various systems have various names for this but the idea is that the claim will be accepted if there is evidence that occupational played a significant role in causing the disease, even if it was only a contributing factor. The most important examples are systems designed to compensate veterans and the specific system designed for railroad employees in the US.

The more stringent the criterion, the more likely it is that meritorious claims will be unfairly denied but the validity of those that are accepted will be more certain. The less stringent the criteria, the better chance a meritorious case has of achieving recognition and compensation but at the expense to the system of accepting more (often many more) non-meritorious claims. Every compensation system faces this dilemma. Although adjudication is slowly adopting new approaches of analysis and what is basically forensics, causation analysis relies on informed medical judgment and expert estimates of most probable association and of apportionment.

It would be unrealistic and an unfair burden of proof to require 95 % confidence, as implied in the confidence interval, as a "scientific standard of certainty" for the individual case rather than the preponderance of evidence. No plaintiff, claimant, or applicant could produce so much evidence or afford the cost of documenting everything to a level of scientific certainty. Cases for adjudication of an uncommon or rare disease, which is often the case with firefighters, would never be recognized

because the evidence would never be strong enough—ever. Reasonable inferences need to be made. Therefore society has sought to lower the burden of proof on the applicant, particularly in situations in which causation is difficult or impossible to prove, the "cause" of the outcome (actually, in this case the contribution to total risk) is likely to be multifactorial (which is often the case in cancer), and in which there is a social interest in protecting a group from an assumed risk, such as warriors and public safety personnel.

A simple elevation of risk is a much less stringent but more attainable and realistic threshold. Simple elevations reduce to an argument over how much excess represents a substantial contribution to risk in the occupation. Simple elevation of risk has also been used to justify compensation guidelines, particular when jurisdictions develop a "schedule" of compensable diseases already known by other means to be related to a particular occupation or when a common disease (such as lung cancer) is known to be elevated in a particular occupation but is hard to separate from a background of community risk. It is also appropriate when diseases are multifactorial in origin and the exposure has made a "substantial contribution" to risk and when "but for" the exposure, the claimant would not have got the disease, regardless of other exposures.

In the US, at least, there are several systems (the system for railroad workers being one of the largest) in which the criterion is whether the occupational hazard made a "substantial contribution" to overall risk, even if the hazard was not the necessary, sufficient, or sole causal factor. Some systems accept the argument that "but for" the exposure, the claimant would not have developed the condition, even if the risk factor in isolation was a relatively small part of overall risk. (For example, asbestos exposure in a cigarette smoker may contribute less risk than cigarette smoking on the basis of straight apportionment, but population studies suggest an interaction and so for a given level of cigarette smoking the risk from cigarette smoking may be doubled or more due to asbestos exposure; the conclusion is that "but for" the asbestos exposure, the claimant, even though he or she smoked, probably would not have developed lung cancer.)

## *Weight of Evidence*

The "weight of evidence" is the operative guidance in making the recommendations in this report. The term "weight of evidence" has two distinct meanings in the application of epidemiological evidence to compensation policies, corresponding to general and specific causation. It may refer to the weight of evidence for an *association* of a particular finding in general causation, such as an elevated risk for a particular disorder among firefighters. It may also refer to the weight of evidence that a particular disorder has arisen from occupation (firefighting) in a particular applicant.

A scientific standard of certainty is used for reporting the findings of individual studies, although this is not always appropriate to the situation. The most likely (or "point") risk estimate (however derived) is presented, together with the confidence

interval around the estimate, to describe the limits of 95 % certainty. These summary estimates apply to individual studies but the rules of 95 % certainty are not absolute and the 95 % confidence interval provides guidance, not a fomulaic answer. For example, when a study is small (and therefore has low power, as will be discussed) or there is corollary evidence for an elevated risk, it is reasonable to conclude that there is an elevation in risk even if it does not attain 95 % probability by conventional inference testing.

## Interpreting the Evidence for Firefighters

The approach taken in this book is to assess the weight of evidence identifying risks for firefighters in the world literature, to make a preliminary informed judgment to determine how these risks might apply to the history and personal characteristics of individual firefighters, and to offer recommendations that reflect the limits as well as the certainties of identified associations.

Most large studies on firefighters are similar in design and face similar limitations on power for rare outcomes; this characteristic has led to the popularity of meta-analysis as a way to discern trends and certainties. However, the core studies also have their own characteristic strengths, weaknesses, firefighter populations, communities from which they are drawn, timeframes, local patterns of occupational hazard such as housing stock, and methodological nuances, sometimes subtle, that make them different. These differences are valuable because they can be used to drill down to investigate particular issues by examining subgroups, exposure-response relationships, anomalies, and confounding by smoking. The incremental addition of increasingly well-designed, larger, and well-conducted studies on firefighter health has been welcome, even though they do not always provide the same level of detail in analysis as some earlier studies.

Meta-analysis has been performed in an effort to overcome some of these limitations [1–3], with limited success. The experience applying meta-analysis to studies of firefighters has not been satisfactory overall, in our opinion, and this approach does not provide sufficient guidance for individual cases [4]. One reason is that meta-analysis, by requiring data for a groups at a comparable and rather high level of aggregation, misses a great deal of evidence that can be derived from looking at subgroups and details about exposure and job assignment. It is suggested that these issues represent a class of problem in occupational epidemiology that is best approached rigorously by examining the structure of the problem outcome by outcome [4].

### *An Approach to Causation Analysis*

There are two attractive but simplistic, approaches to interpreting etiological studies in epidemiology when they are applied to causationanalysis and qualification for compensation. They lie at opposite ends of an interpretive spectrum.

The first is a "one hit" model (author's terminology, a play on words for a cancer causation model), in which any study that demonstrates an association with sufficient strength is considered to be sufficient evidence to accept the disease outcome as occupation-related. The idea is that as soon as one study shows a positive finding, it is first past the post and the outcome must be accepted as compensable. The "one hit" model is too likely to result in a Type I error, in which an association is thought to be true, or causal, but it is not in reality.

At the other extreme is the "model of scientific certainty", in which rigid consistency, absent homogeneity within studies, and doctrinaire conformity to rules. Such rules include 95 % certainty, an overly rigid application of the Hill criteria [5], and rules used in some approaches to meta-analysis [2]. This model is inappropriate because it is too likely to result in a Type II error, in which something that is truly associated or causal is judged not to be. Occupational health outcomes occur in a real world of uncertainty, confounding factors, community health trends, different populations, changing technology, epidemiological conventions that obscure diagnostic distinctions, inaccurate measurement and misclassification, methodological bias, and limited access to study populations to achieve statistical power.

The "one hit model" places undue emphasis on findings that might be due to chance variation and therefore places an unsupportable burden on the system of compensation by accepting some claims that are not, in actual fact, meritorious (through no fault of the claimant). The "scientific certainty model" places an unsustainable hardship on the applicant because proof is almost impossible to achieve by an individual claimant. Documentation is also a problem, because the scientific literature is not geared to assessing individual causation and so often does not provide essential information to make the link. The appropriate methodology, it would seem, lies somewhere in the middle, and involves critical analysis of the problem.

Issues of causation in firefighting may represent a class of problem in occupational epidemiology that is best approached outcome by outcome using principles of logic rather than advanced statistical techniques. Key to the validity of the methods described in this paper is the essential criterion that it is the weight of evidence, not scientific certainty, that determines the outcome of the case or claim in a legal setting, such as tort litigation and adjudication for compensation benefits [6].

In order to provide a more realistic analysis for the specific purpose of compensation, not scientific inquiry, a series of methods have been developed and applied to health risks of firefighters, primarily to cancer [4]. These principles include the following:

- Where data exist, scrutiny of subgroups for evidence of an increasing effect with increasing levels of exposure.
- Where data exist, separation of exposure metrics from length of service, because years as a firefighter covaries with age, changing job assignments, changing technology, and latency for disease.
- Convergent evidence among studies to identify a more reliable signal for "true" risk among firefighters against a background of random variation and uninformative studies (because of insufficient power) or compelling reasons why an elevation of this magnitude may be obscured through bias or confounding.

- Test for confounding, by determining if there is evidence for a higher risk estimate with progressive refinement in exposure assessment or increased exposure to work-related hazards.

All epidemiological risk estimates are just that—estimates—and represent the experience of the populations being studied. Uncommon events, such as lung cancer, are subject to chance variation. This is precisely why one derives confidence intervals for the estimates. The power of a study is its ability to detect an elevated risk when there actually is one. One likes to have a power of at least 80 % but few studies of occupational can achieve even 50 % for lung cancer, even though it is one of the most common cancers and since 1987 the most common cause of cancer deaths for both men and women [7]. This means that a large fraction of studies, without question, miss the true association. This is not an idiosyncratic opinion or controversial: it is inherent in the definition of power, as well as easily observed in practical experience.

## *Sources of Uncertainty*

Uncertainties in studies on risk of firefighters come from several sources:

- *Data gaps*. For example, there are no studies of lung cancer risk among nonsmoking firefighters. Such gaps clearly represent questions that have not been addressed, for whatever reason, in studies of firefighters. It is well known and accepted that "absence of evidence is not evidence of absence", as a general proposition. However for other outcomes, "the absence of evidence" may actually be "evidence of absence". This is because there is a bright spotlight on the profession. Firefighters are a highly visible occupation that has attracted great interest from the public and from scientists, both because of the features of the occupation (toxic exposures, extreme ergonomic demands) and because of convenience and cooperation as research subjects. There has been concerted and intense research on the occupation for five decades. Firefighters are closely monitored, and there has been an atmosphere of strong incentives and even competition for investigators at institutions around the world to study firefighters, even more so since 2001. An uncommon outcome (such as parotid gland carcinoma) or an elevated risk of a relatively common medical condition (such as peripheral neuropathy) appearing in firefighters would attract attention and be recorded in the scientific literature. This is not to say that nothing could possibly be missed, but it would be unlikely that a major association or consistent and obvious finding would be overlooked in this occupation.
- *Exposure-response relationships*. The most common measure of exposure in the literature, by far, is duration of employment as a firefighter. However, this measure does not take into account job assignments and activity, which may vary considerably. Duration of employment is also confounded by everything else that is time-dependent: age, career changes, changes in the technology of firefighting

and personal protection, and changes in fire smoke (especially when synthetic materials changed the hazard of fire smoke in the 1950s and 1960s). Very few studies (e.g. Baris et al. [8] and Guidotti [9]) report exposure indicators other than length of service. The absence of exposure information has severely limited interpretation of the literature for important outcomes, such as respiratory disorders.

- *Disease rubrics*. Important distinctions in clinical diagnosis are lost when diseases are put in categories. For example, the leukemias are separate and distinct diseases and at least one of them (acute myelogenous leukemia) is highly associated with benzeneexposure. However, the leukemias are almost always lumped together indiscriminately for analytical purposes (the exception being Aronson/L'Abbe and Tomlinson [10] and reports based on that work). The reason for such indiscriminate aggregation, which was much more common in the past, was that statistical methods work better with larger numbers, but applying improved statistical methods to larger numbers based on illogical combinations can actually obscure important findings more than it illuminates risk.
- *Disease identity (case definition)*. Developing scientific knowledge, particularly about causation, makes many disease rubrics tentative at best. For example, the global consensus on classification of the non-Hodgkin lymphomas has changed fundamentally at least four times since the 1970s [11]. It is now clear that certain individual lymphomas are caused by different exposures [12–14]. However, there are few studies on individual lymphoma types and risk from firefighting.
- *Statistical error*. In statistics, the term "error" does not mean a mistake. Random error means that because of chance, the true value is obscured by random variation. This is a characteristic of every epidemiological study. For rare diseases (using an epidemiological definition) such as cancers, this translates into an inability to be sure whether there is an elevated risk or not. The theoretical argument over whether "positive" studies (which show an elevation) outweigh "negative" studies (which do not) is a major preoccupation of occupational epidemiology. As a practical matter, positive results do matter more than negative studies in the situation of rare diseases and causation, because it is much easier to miss a true association because of bias and power limitations than it is to find a marked but spurious elevation in association appearing in multiple studies, in the absence of a high degree of confounding. The reasons will be discussed in detail.
- *Bias.* In principle, bias (a systematic error, in which the results are affected by some problem in gathering data) can result in an over- or under-estimate of risk. In practice, in etiological epidemiology of rare diseases it almost always results in an underestimate, such that associations are obscured.
- *Confounding*. Many other risk factors affect disease outcomes, most obviously smoking. Almost no studies on firefighters have corrected for confounders (the exception being Beaumont et al. [15]). The most serious source of confounding, however, may be time, because length of employment, duration of exposure, latency (for most solid cancers), and age (and therefore susceptibility to most cancers) are all closely correlated but not the same and there is inevitably insufficient data to disentangle the covariance.

- *Paradigm blindness*. The prevailing thinking in epidemiology is that each study represents the experience of a sample population from a universe of firefighters exposed more or less uniformly (with random variation) to hazards. This paradigm can blind investigators to the differences in generations and eras of exposure, to changes in underlying or "baseline" risk from the comparison populations, and to the reality that populations are collections of individuals, not tangible entities with an independent existence.

## *Smoking as a Confounder*

A confounder, in epidemiology, is a risk factor that is linked to both the risk factor under study and the outcome, so that it interferes with the interpretation of the risk factor under study. The confounding factor can be fairly described as a true risk factor, but one in which the investigator is not interested and which therefore gets in the way.

Smoking is the major potential confounding factor in epidemiologic studies of firefighters, as it is in many and perhaps most studies in occupational and public health epidemiology. However, it may not have as strong an effect as is usually assumed and is probably less of a problem than in other occupations. Smoking is also disappearing as a widespread habit among firefighters.

More recent studies of the prevalence of the smoking habit among firefighters also suggest that firefighters smoke much less than in the past and much less than the general population. Estimates for occupations identified in the National Health Interview Survey in 1987–1994, which would be relevant to chronic disease presenting at the current time, placed the prevalence of cigarette smoking among firefighters in the United States then at about 27 %. By comparison, law enforcement officers were 32 % [16]. Smoking is also declining, apparently faster among firefighters than in the population as a whole. Prevalence of smoking in the current Fire Department of the City of New York (FDNY) is only 3.5 % [17]. This is much lower than in the past, when general population smoking prevalence rates were closer to 40 %, but reflects overall trends in the population. There has never been documented evidence that firefighters smoke more than the general population and what scanty data exist from the past suggest that they smoked less than other occupations, at least in modern times.

Smoking is linked to many outcomes of interest in firefighting because cigarette smoke is a combustion product and therefore contains many of the constituents, carcinogenic and otherwise toxic, as fire smoke. (The differences between the two, and with air pollution, are discussed in Chap. 5 on toxic hazards.) As a result, smoking is a particular problem in studying cancers of the lung, larynx, pancreas, and bladder, and in coronary artery disease, chronic obstructive pulmonary disease (COPD), exacerbation of asthma, and peripheral vascular disease.

Cigarette smoke is much richer chemically than lignocellulosic fire smoke (smoke derived primarily from wood and paper), consisting of over 5000 individual

compounds, and contains toxicologically active components such as nicotine not present in fire smoke [18, 19]. Some of these components, including nicotine, act as anti-inflammatory agents, apparently damping down the acute irritation and inflammatory response of exposure to cigarette smoke [20–27]. This may explain why fire smoke appears to be more acutely irritating and suggests that cigarette smoke may also modify the response to fire smoke. The anti-inflammatory effect of cigarette smoke also appears to be highly selective, modulating some immune and inflammatory reactions and not others.

Cigarette smoking is a common habit and therefore occurs often in combination with other exposures. Smoking interacts, often strongly, with other exposures, so that combined exposures may significantly enhance, or at least modify, the outcome associated with the exposure of interest. This is less likely to occur with exposures that are chemically or toxicologically similar and the effect is likely to be additive, such as fire smoke, and more likely to occur when the mechanism of carcinogenesis is different and they have the potential to interact, such as cigarette smoke and asbestos.

Smoking is inversely associated with socioeconomic class and is more prevalent in some occupations, particularly those that are, paradoxically, either boring (stimulation as a relief from the tedium) or that involve social mixing (partially due to the transactional nature of sharing smokes). Firefighting has both of these characteristics, interestingly, and is considered a working-class, or "blue-collar", occupation, which suggests greater cigarette consumption. However, this was more true in the past than today. Contemporary firefighters tend to be much more educated, more health conscious, and more concerned about smoke effects because of their vocation. Firefighters also enjoy uniquely high status in their communities and educational standards are higher, so that social class generalizations from the past do not necessarily apply today.

There is also a statistical reason why smoking is a major confounder, which particularly applies to regression studies. Smoking is one of the few risk factors in which it is easy to quantify exposure in the individual case, by number of cigarettes or pack-years smoked. The ability to quantify risk precisely by number of cigarettes smoked per day makes it much harder to determine the contribution of non-smoking risk factors. Because it is based on the accuracy of prediction of one variable by another, regression methodology strongly favors continuous measurement of independent variables, so something that can be measured precisely is weighted more heavily than something that can only be measured crudely. As a consequence, smoking almost always emerges as carrying the greatest weight in the regression.

When information is available on smoking habits of subjects in a study, it is possible to adjust the risk estimates in the analysis. For individuals and for populations, the usual scale of assessing risk is the smoking history, is quantified as "packs per day." Assuming 20 cigarettes per pack (the US and the United Kingdom standard but there are 25 cigarettes per pack in Canada and Australia) a smoking history equivalent to 20 years at one pack per day (or roughly 16 "Commonwealth" packs) is conventionally accepted as associated with an unequivocal risk of chronic health effects.

At the same time, other people in the population smoke, and the reference groups to which firefighters are compared (usually, the general population or sometimes police) have a prevalence of smoking among their subjects as well. The biggest problem in interpretation would come when there is a discrepancy in smoking rates between the study population and the reference population. In the relatively few studies that have been done, the rate of smoking among firefighters has never been shown to be higher (and is usually reported to be lower) than the general population. This means that a health outcome is unlikely to be due to a higher rate of smoking among firefighters, when compared to the general population or a reference group, such as police, which is unlikely to smoke less.

When data on smoking is not available, at least some generalizations can be made. The difference in prevalence would require that rates of smoking be implausibly high the population of interest (in this case, firefighters) in order to explain a substantial elevation of risk (on the order of 50–100 %) for a smoking-related disease. The section of this report on lung cancer demonstrates why differential rates of smoking alone are almost never sufficient to explain a large excess risk for an outcome that is at least doubled in risk in the study population. This principle was first demonstrated by Fletcher and Ades [28], in 1984, in a study of foundry workers.

## *Latency*

Latency is the time that expires between the action of the cause and the manifestation of the outcome. In cardiovascular disease, it might be the time expired between the first injury to the lining of the coronary arteries and the rupture of a plaque or onset of a thrombus that initiates a heart attack (myocardial ischemia). In cancer, it is the time elapsed between initial induction of carcinogenesis and diagnosis of the cancer. Because the onset of carcinogenesis is unknowable, latency in cancer epidemiology is operationally defined as the elapsed time between first exposure to a risk factor (carcinogen) and the clinical manifestation of the disease.

True latency is rarely knowable for cancer, because the action of the cause cannot usually be pinpointed. It reflects the time after the genetic constitution of the cell has been altered that the cell is dormant, then becomes cancerous and finally proliferates by dividing until a cancer appears that is visible, detectable on tests or interferes with function and is discovered. Latency also varies by exposure, with higher exposures tending to shorten the period of time that elapses before the cancer is detectable.

It is generally held as a rule of thumb that the latency period for solid tumours is on the order of 15–20 years, but this should be understood simply as the most likely or modal latency, the time elapsed before an excess is observed, and not the minimum time required for the tumour to become manifest. Such rules of thumb do not necessarily apply to individuals. Cancers associated with occupational exposures can and do appear well before an arbitrary latency period, although there is usually

a minimum imposed by the biology of the tumour and its rate of proliferation. Tissues of the blood-forming organs may have very brief latency periods, on the order of a few years (for radiation-induced leukemia) but usually take longer. Some latency periods are unusually variable. Mesothelioma, a devastating cancer of the pleura (inside lining of the chest wall) typically requires decades to develop, on the order of 30–40 years. However, there have been thousands of cases in asbestos-exposed men in their twenties and thirties and a few cases of mesothelioma the disease will appear within 10 years of exposure. (The background rate of mesothelioma in the general population, unexposed to asbestos, is close to zero.)

Latency is also dependent on exposure (dose) level and age at earliest exposure: during an era of high exposure to these chemical carcinogens for bladder cancer in the early twentieth century, workers exposed to aniline dyes in the chemical industry in Delaware could develop bladder cancer in less than 10 years. Such extreme latencies are thankfully rare today.

Duration of employment is difficult to separate from latency, which, of course, is also confounded by the aging process. Duration of employment is also entangled with age, changing job assignments, changing technology for respiratory protection (and adherence to the use of self-contained breathing apparatus), changing composition of fire smoke (especially for older studies, before and after synthetic materials came to dominate fire smoke), and and changes in the risk of reference populations used for comparison (for example, smoking rates). That is why simple duration of employment, alone, is not very satisfactory as an indicator of relative exposure.

## "Positive" and "Negative" Findings

*Positive findings* are those that suggest an effect, such as an elevation in risk. *Negative findings* are those that do not show an effect, although they may be substantively important in doing so. Colloquially, experts and epidemiologists often speak of "positive studies" when the study has positive findings, and "negative studies" when there are no noteworthy or surprising findings. This casual language conceals much ambiguity. If a finding does not achieve statistical significance at the conventional level for scientific certainty (95 % for an individual study), and yet the elevation is not small and is consistent with convergent evidence for an effect, can it truly be said to be "negative"? If a study calculates statistical significance for 100 different outcomes (which is quite common in cohort studies) and five of them show statistical significance at $p<0.05$ but not much more, then are these findings truly "positive" when the chance of achieving that level of significance for any five outcomes out of any hundred is arguably 25 %? The answer, of course, is that inferential statistics, while important, does not tell the entire story. Context is everything.

Studies of firefighters are generally large and relatively similar to one another compared to other occupational groups but they almost always still have low power for rare outcomes and are by no means identical. Most occupational cohort studies used to evaluate cancer risk look at multiple sites and are designed to have sufficient

power for a relatively common outcome, such as heart disease; they are almost never designed for a single cancer, and when there is interest in a particular type of cancer they do not usually have sufficient power for resolution. Case-referent studies do examine outcomes one at a time but tend to be low in power to detect an association with occupation. Population monitoring studies (such as cancer registries) often have a similar problem and greater problems with misclassification (missing firefighting as the usual occupation). The approach used in this chapter depends on a close reading of the individual studies and piecing together a picture on the weight of evidence.

Power is defined as the probability that the true risk will be determined in a study. Power for most studies to examine a rare outcome is very low, especially population studies not designed to identify the outcome as the main finding. The approach of meta-analysis is to overcome problems of low power in individual studies by aggregating the studies statistically, weighted by contribution of information, and determine the central tendency of the risk estimate for all together. This presents other problems, as will be seen.

An epidemiological approach based on a standard of the weight of evidence, or "more likely than not", must accept the preponderance of evidence for an association even when that evidence does not achieve a scientific standard of certainty. This forces a different way of looking at studies with low power for the outcome of interest.

The essence of this approach is that if one believes that power considerations and inherent bias make it more likely that an association will be missed than that one will be revealed, then one must place greater weight on positive studies. This uncertainty over power means that studies that do show an excess risk should probably carry more weight in adjudication than studies that have not demonstrated an excess risk. Studies that show no elevation in risk may simply have missed the excess and convey no information. Studies that show an excess risk, especially if they are consistent and show a dose–response relationship (one important criterion of a true association) are likely to be more useful is assessing the probable magnitude of the true excess risk. This is not conventional wisdom in epidemiology, but neither is it necessarily a minority opinion in practice.

Occupational and environmental epidemiology generally, and studies of individually "rare" diseases, such as cancer by site, in particular, share the common and frequent problem of inconsistent findings among studies. What to do about negative studies when there are strong positive studies addressing an association is highly controversial largely because of the tacit assumption that inconsistency and inhomogeneity suggests disorder and lack of clarity when in actuality these attributes are entirely to be expected when there is very low power in rare outcomes. This is an important practical problem. Decisions on cancer prevention, health promotion, workers' compensation, personal injury, and worker protection depend on interpretation of seemingly conflicting studies.

Studies that are similar in design and that study similar populations may still yield inconsistent results, with some showing an excess risk and others showing no statistical evidence of an elevated risk for the same group. Studies that show an

excess risk, the "positive" studies, are often viewed skeptically because they could demonstrate a Type I error (suggesting that some finding is present when it is not), because of bias or chance. Studies that do not show an excessive risk, in that they estimate the risk at close to or below unity, are usually, and often wrongly, taken at face value in practice. However, in the situation of low power for a rare outcome, they are probably more likely to demonstrate a Type II error, most often either because of limitations in power or because of misclassification bias, which almost always results in an underestimation of risk.

In most situations in occupational epidemiology, the number of studies available from which to draw conclusions is small. This is not true for firefighters. Very few occupations have been studied as extensively and repeatedly using essentially the same methods as firefighters. This occupations can therefore be examined profitably as a body of evidence, rather than as a collection of individual studies. Even so, these studies should never be assumed to be identical. Trends over time may obviate the relevance of earlier studies in calculating current risk, if only because of differences in exposure profile and the underlying populations [29].

As a scientific problem, such discrepancies are often considered, and are always described in the literature, as a challenge for further investigation. ("More research is needed" is the usual phrase.) However, assessment of claims for workers whose exposure occurred in the past must as a practical matter be performed with the knowledge that exists today. No workers' compensation claim is going to wait or be put on hold for the definitive study, which will never come, nor should it be.

All other things being equal (which they never are), positive studies outweigh negative studies in epistemological if not statistical significance. In individual circumstances, this generalization, like all generalizations, may not be true, but the logic of power dictates that when the assumptions are satisfied, the burden of demonstrating that it is not true falls on those who question the association, not on those who place higher value on "positive" studies.

The argument over giving "positive studies" disproportionate weight assumes the following about the set of studies under consideration:

- The individual studies are based on comparable but not necessarily identical populations, approximating a sample of all workers in that occupation, notwithstanding that firefighters are recruited out of community populations with somewhat different underlying health characteristics.
- The studies are conducted using similar methodology, primarily cohort studies, with near-complete ascertainment of outcomes for subjects.
- There are a sufficient number of methodologically similar studies to reflect statistical variation due to random error on the level of individual studies (in other words, a reasonable estimate of standard error or the coefficient of variation among studies would be possible).
- Bias in the studies, including and especially confounding, is not strong enough to obscure the statistical anomalies at the level of collections of studies.

Practical decisions, especially where matters of equity are concerned, should therefore not be made on the basis of the "preponderance of evidence" if this is

considered to be the tendency of the majority of studies. One cannot tally positive studies in one column and negative studies in another and see which list is longer. This approach will inevitably miss the correct interpretation in this situation. The totality of the evidence should be considered, including possible reasons for divergent results, differences in the populations studied, signs of confounding (such as an increasing relative risk when exposure assessment is strengthened), and consideration of a bimodal distribution of risk estimates.

On a more technical level, it can be argued based on a strict definition of power that meta-analysis systematically underestimate the true risk when studies of low power are aggregated. Studies that have missed the effect entirely and predictably are combined with many fewer studies that did observe the effect. Unfortunately, there is no easy way to know this.

## Epidemiological Rationale for Presumption

Presumptions make the most sense if they recognize a risk level that corresponds to more likely than not, which is another way of saying the "weight of evidence", which is the standard of persuasion for civil litigation and the alternative dispute resolution systems (such as workers' compensation) that arose from it. This is sometimes called the "50 % + 1" rule. In reality, most workers' compensation acts are written so that in cases of uncertainty or roughly equal probabilities, the benefit of the doubt should go to the claimant, so the standard usually should only be "50 %", not 50 % + 1. (Closely argued cases are common.)

In an individual case, the standard of persuasion applies to the individual (plaintiff or claimant). Policies for presumption, however, apply to the group as a whole, meaning that for any given member of the group with a potentially compensable condition, all other things being equal, the cancer or other condition more likely than not arose out of their work.

In the search for "bright lines" to guide policy in the evaluation of elevations in risk and to underpin presumption, only one stands out as completely objective, in theory: a *doubling* of risk among members of the group (firefighters) compared to the reference population (sex-specific members of the community or a similar defined group). This is because in statistical terms it corresponds to the weight of evidence.

A doubling of risk implies in theory, that, all other things being equal, a claimant who applies from a defined population (in this case firefighters) and who conforms to the characteristics of most members of that population, is more likely than not to have had their health condition arise from risks associated with their shared risk factor (in this case occupation) [4]. Mathematically, a risk ratio of two literally means that the risk arising from firefighting equals the risk arising from other risk factors in the population from which firefighters are drawn (the community), and so constitutes an exact balance of probabilities. Evidence for a true elevation of risk as high as a doubling, once evidence for confounding, the potential for bias, and dilution have been taken into account, would ideally be the rigorous policy standard is for presumption.

In practice, a doubling of risk, while more realistic than "scientific certainty", is also a high standard. It is necessary to identify the correct subpopulations to identify a trend and, to apply the evidence to specific causation or rebuttal, the situation that most closely resembles the applicant's situation. This is often difficult. Confounding factors, dilution of the risk estimate, bias, and numerous other factors make it easy to miss a true doubling of risk or to make it impossible to see or recognize in the subpopulation most affected. Because it is so hard to demonstrate a true doubling, in practice when this is proposed as the standard, it is usually enough to show that the preponderance of evidence shows that for the occupation in question, a worker is at least as likely to have their condition arise from work as from other causes overall or in a subgroup to which the worker belongs.

However, a doubling of risk is not always obvious because even the best studies are affected by low power (inability to identify an elevated risk through conventional statistical significance, dilution (especially when disease categories are too broad and contain unrelated conditions), low numbers for the outcome under study (inevitable for uncommon conditions, such as specific cancers), sources of bias (well-insured firefighters being compared to a general population of the US in which 19 % of citizens under 65 were uninsured in 2009), and confounding (when a second factor is related to both the occupation and the outcome and so cannot be distinguished from a firefighting effect). In such cases, it is imperative to look more deeply for evidence, for example in subgroups, for an exposure-response relationship, and testing for confounding (by seeing if the risk estimate increases with more refined or accurate exposure assessment). Then the risk of the relevant subgroup can be compared with particulars of a specific case, as it should be.

A policy of presumption is actually a relatively stringent criterion for compensation, requiring a data-intensive analysis and a strong association (a risk of 2 is moderately high in occupational epidemiology). It is best viewed as strong evidence in support of causation and therefore strongly favouring a presumption, not a minimum threshold for presumption.

A doubling of risk need not be the only basis to establish a presumption, particularly if there is wide uncertainty and the quality of evidence is weak. In addition to implicitly recognizing that occupation is the primary "driver" of the outcome, with other risk factors playing a lesser role. Presumption implies that a factor, such as occupation, plays an predominant role and if "but for" the risk factor an individual would not have developed the disorder but that same risk factor is not responsible for at least half of risk (of a particular health outcome) in the population. An elevation in risk that is less than a doubling, but which is plausible on mechanistic grounds and that is shared by subjects in certain situations of common exposure has been used, for example for veterans in the Viet Nam conflict who were exposed to Agent Orange (phenoxyacetate herbicides).

A doubling of risk is also not the only basis for justifying a presumption. Some legal systems only require a "substantial contribution" to make the case. (Pennsylvania requires this to activate the presumption for firefighters with cancer.) Others require identification of specific chemical or other hazards, which of course are abundant in firefighting, as in Québec.

Accepting a doubling of risk as the conventional standard for presumption has its drawbacks. It is often difficult to tell whether the risk is in fact doubled against a background of statistical noise and study bias. Most sources of error predictably result in an underestimate of risk for the group, and so the doubling figure should not be taken too literally. Taking doubling as an overly strict standard also has the undesirable effect of denying many meritorious claims, by failing to compensate many firefighters whose disease did in fact arise from occupation.

Any system in practice will have some degree of misclassification: the goal is to minimize it to the extent allowed by medicine and the forensics of compensation. In providing compensation benefits, the system can err on the side of accepting claims that a disease is occupational when it is not (what statisticians call a Type I error). The system can also err on the side of claiming the disease is not occupational when it is (Type II error). For compensation systems, the consequences of a Type I error are greater, because denial of benefits to injured workers and families who need support causes greater social harm and is less fair to the individual than providing benefits in some cases in which the disease had another origin, which cannot be known.

The aim of social policy is and should be to give fair compensation to those who took the risk and were harmed, not to ensure that no (or very few) people who were not harmed are excluded from benefits. After all, the diseases are the same, the need is the same, they all shared in taking the risk in the first place, and they cannot be separated, medically or by any forensic means now available, from those whose disease did in fact arise from their occupation. The best that can be done is to set criteria and policies that excludes claimants whose disease clearly or probably did not arise from occupation to the extent possible, which is the function of the rebuttal.

**Expert Interpretation**

The expert must always remain aware of the level of certainty that applies in a given legal or compensation system: the weight of evidence. For the expert attempting to draft a reasonable and appropriate opinion on cancercausation, one of three levels of persuasion will apply:

- "Rebuttable presumption" on an empirical basis, which is the recognition in legislation or policy that a person who belongs to a particular occupational group and who has developed a certain condition, more likely than not developed that condition as a result of workplace exposure. The expert for the plaintiff or claimant, just as much as for the defense or employer, must always consider rebuttal arguments as well as framing the case from the point of view of the client. The presumption can be rebutted in the individual case if it can be shown that there is another factor (such as smoking history or heredity) that is more likely to have caused the outcome. Rebuttable presumption is the standard used in states and provinces that have legislated presumption acts, It is also sometimes followed *de facto* in workers' compensation internal adjudication policy.
- "Weight of evidence", which is the standard in civil litigation. Weight of evidence requires that in the totality of evidence the "weight of evidence" demonstrates a causal association by a "balance of probabilities", sometimes expressed as "50 %+1". Many workers' compensation acts, but not all, require that if the

balance is roughly equal, the benefit of the doubt should be given to the claimant. This is the usual standard of persuasion in workers' compensation (which derived from civil litigation), followed by states and provinces that do not have presumptionlegislation. Plaintiff or defensecounsel sometimes overstates the certainty of the case. Defense or employer counsel usually implies that a scientific standard of certainty is required. They key phrase is "reasonable medical certainty", which implies that the weight of evidence is satisfied.

- "Substantial contribution", which is a concise term for those systems and policies in which claims are accepted if it can be demonstrated that the exposure made a significant contribution to the risk of cancer, such that a risk that may have been present already was increased by a more than trivial amount. In such systems, evidence that occupation as a firefighter carries a significant risk would be sufficient; demonstration of an elevation as high as a doubling would not be necessary. How much of a contribution would be deemed substantial is open to question, but few seem to argue with an apportionment of at least 15 % of risk. Systems that use substantial contribution include Pennsylvania and the Federal Employees Liability Act (which, despite the name, is primarily important in this context as an example because of its provision for a reduced burden of proof for railroad workers).
- Presumption on the basis of exposure experience. Many compensation systems have adopted presumptions based on a relaxed view of "substantial contribution," in order to free the applicant or claimant from the burden of having to demonstrate the weight of evidence or to provide extensive documentation. This is most often conferred in situations where society views the "victims" as exceptionally meritorious, vulnerable, or heroic. This standard is usually adopted in rare cases when public sentiment favors being ensuring that all persons who were affected should be compensated, regardless of their ability to prove the claim. This standard has been applied to Vietnam Veterans exposed to Agent Orange and to residents of New York at the time of the "9–11" attacks who applied for compensation through the World Trade Center Victims Compensation Fund (the Zadroga Act).

Obviously, those systems that have adopted standards of persuasion that require less documentation and feature fewer grounds for rebuttal are more likely to favour the plaintiff or claimant. However, the role of the expert is not diminished in such systems. It remains central, to explain the circumstances, to inform adjudication, and to explain the association, all in terms that are accessible and understandable but accurate.

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# Chapter 5
# Toxic Hazards

**Tee L. Guidotti**

Firefighting as an occupation involves exposure to many chemical hazards, often at intense exposure levels. The main text of this chapter discusses specific chemical hazards and assumes a working knowledge of toxicology. The Appendix to this chapter is a short introduction to the science of toxicology and its principles. It may not be necessary for all readers, which is why it is an Appendix, but for those without a background in either toxicology or occupational health it will explain the basics of how chemical substances are handled by the body and how they affect the body.

Most chemical hazards in firefighting enter through the respiratory tract as airborne hazards, some exposure to chemicals occurs by skin contact, and there are relatively few opportunities for contamination of food (or, in an earlier era, cigarettes). The principal hazards include fire smoke, products of incomplete combustion (such as vinyl chloride and styrene), structural components such as asbestos (predominantly chrysotile in North America), and diesel exhaust (nitroarenes). A great deal is known about each of these chemicals. Each is handled by the body in its own way and has characteristic effects on the body (called a "toxidrome"). However, in firefighting they are not coming into contact with the body one chemical at a time: they come as mixtures, on top of one another, and sometimes in unpredictable combinations.

Some inhaled hazards pass through the lung as a portal of entry without causing it direct injury, such as carbon monoxide and the solvent-like chemicals. Others exert their primary effect on the lung itself. These inhaled agents are toxic, to some degree, to virtually every structure in the respiratory tract, from the epithelium of the upper respiratory tract to the alveoli of the deep lung. It is noteworthy that among the agents specifically listed in this paragraph, even those that are not usually considered to be toxic to the respiratory tract apart from carcinogenicity, such as

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PCBs and PAHs, have been shown in toxicological studies to have the potential to affect tissues present in the respiratory tract. Exposure during firefighting has changed over decades, with the introduction of synthetic materials (particularly in the 1970's) bringing to the traditional hazards of structural firefighting (in which wood smoke, which is relatively simple toxicologically, has predominated) a wider variety of potential exposures (including cyanide from nitriles and hydrochloric acid, from polyvinyl chloride-containing materials). As well, whatever happens to be on site and combustible, released during the destruction, or volatile may add to the exposure.

Simple exposure models based on the assumption of inhalation as the only route of exposure may not adequately characterize exposures incurred during firefighting. Recent evidence suggests that skinabsorption plays a greater role than previously believed and that the route of exposure may change the kinetics and therefore the risk of excretion and metabolism of other chemicals [1]. This is best established for PAHs, which have been demonstrated to be absorbed through the skin sufficiently (about 9-fold) to change the calculated risk of cancer in models (not in experiments). This observation lends credence to the frequent observation that mixtures and combinations of exposure may change ultimate effects.

Table 5.1 is a list of exposures, including combustion products, known to be encountered in firefighting. Individual sections in this report discuss the principal chemical hazards associated with the relevant disease outcomes. Not every exposure listed in Table 5.1 is discussed. Similarly, there are other toxic chemical exposures, such as hydrogen sulfide and its combustion product sulfur dioxide, that may be encountered in unusual situations, in this case oilfield fires, but which are not part of the usual atmosphere of municipal firefighting. When these occur the usual practice is to involve hazardous materials ("hazmat") teams in managing the incident, but exposure to these chemicals may not be recorded in the firefighter's personnel record or recalled many years later.

## Characteristics of Smoke

The type of fire matters to the toxicity of fire smoke. In general, urban structural fires produce more complicated toxic exposures than wildfires [2, 3], but the duration of exposure may be longer in fighting wildfires. Fires in industrial facilities or elsewhere where special hazards exist can present unique and potent threats. For example, a fire in a pesticide storage facility containing organophosphate pesticides is particularly dangerous because of the conversion of these chemicals to the more toxic paraoxon form. Tire dump fires tend to burn hot, to be difficult to extinguish, and environmentally damaging, emitting large quantities of particulate matter to air, and depositing hazardous chemicals on the soil, particularly benzene, where they migrate downward and contaminate groundwater. Methamphetamine laboratories ("meth labs"), are illicit drug-manufacturing locations that have become a modern scourge of firefighting and law enforcement; they are exceptionally hazardous, with

**Table 5.1** Exposures encountered in firefighting [3, 25]

| Exposures not produced by combustion chemistry | Exposures primarily associated with combustion |
|---|---|
| Antimony (constituent of flame retardant on turn-out gear)<br>*Asbestos*<br>Cadmium<br>Lead<br>PFOA (perfluorooctanoic acid and its product polytetrafluoroethylene)<br>*Pesticides*<br>Polybrominated biphenyl compounds (mixed, low)<br>*Polychlorinated biphenyl compounds* (mixed)<br>*Silica dust* | Acetaldehyde<br>Acrolein<br>Aldehydes (mixed)<br>Alkanes, straight chain (inc. propane[a])<br>Alkenes, straight chain (inc. propene[a], 1-butene[a]/2-methylpropene)<br>*Benzene*[a]<br>Benzaldehyde<br>Brominated hydrocarbons (low)<br>*1,3-Butadiene*[a]<br>Carbon dioxide[a]<br>Carbon monoxide[a]<br>*Chlorinated alkanes* (low)<br>Chlorobenzenes (low)<br>Cycloalkanes<br>Cyclopentenes<br>*Dioxins and furans* (including 2,3,7,8-dibenzodioxin and –furan[a])<br>Dichlorofluoromethane<br>Ethylbenzene<br>*Formaldehyde*<br>Glutaraldehyde[a]<br>Hydrogen chloride<br>Hydrogen fluoride<br>Hydrogen cyanide<br>Hydrogen fluoride<br>Isopropylbenzene<br>Isovaleraldehyde<br>*Methylene chloride*<br>Naphthalene (a PAH)<br>Nitriles (mixed)<br>Nitroarenes (analogues of PAHs)<br>Nitrogen dioxide<br>Particulate matter (fine)<br>Phosgene<br>*Polycyclic aromatic hydrocarbons* (mixture, including naphthalene[a])<br>Sulfur dioxide<br>Styrene[a] (possibly carcinogenic)<br>*Tetrachloroethane*<br>Toluene[a]<br>*Tricholoroethylene*<br>*Vinyl chloride (pyrolysis product of PVC)*<br>Xylenes (including o-xylene[a]) |

*Italics* indicate carcinogenic potential at levels encountered
"Low" refers to very small detected levels
[a]Predominate in nonspecific urban structural fires

**Fig. 5.1** Fighting a fire in a meth lab, a wooden house that has been used to "cook" methamphetamine. These fires are exceptionally dangerous because of the concentration of explosive, flammable, and highly toxic chemicals and deep contamination of the sites where they have been located. (© United Fire Fighters of Winnipeg, used with permission.)

highly flammable solvents and highly toxic products that permeate the structure and make cleanup extremely difficult and hazardous (Fig. 5.1).

Even in a simple house fire, firefighters are exposed to multiple chemicals because of the presence of synthetic materials. These multiple exposures occur mostly by the respiratory route but somewhat by skin [1]. For specific health effects, the combination and the timing may be significant, but not much is known about these interactions in human beings.

Fire smoke varies with the source and composition of the material burned and the heat of the fire. In general, wood smoke is chemically simpler than smoke derived from structures containing synthetic materials (some of which also serve as chlorine donors in chemical reactions). Fire smoke is not identical to cigarette smoke. Although they share many of the same constituents, in particular the PAHs, there are important differences between fire smoke generally and cigarette smoke. Tobacco smoke and fire smoke purely from burning wood ("lignocellulosic" fuel) has only trace amounts of trichloroethylene, a significant solvent chemical associated with cancer risk, and low levels of other halogenated organic compounds compared to fire smoke; structural fire smoke is rich in these halogenated compounds. Fire smoke from wood appears to have low levels of 1,3-butadiene but this carcinogen is elevated in both structural fire smoke and in cigarette smoke.

**Fig. 5.2** Rollover of a tanker truck, with burning gasoline. Note the black smoke, consisting of particulate matter, which is more abundant where combustion is taking place at lower temperatures. Note also that tires are on fire and also generating smoke. (© United Fire Fighters of Winnipeg, used with permission.)

Products of combustion exist in two physical phases, particulate matter and gases. Smoke consists of solid particles suspended in air (aerosol) in an atmosphere that contains gases, Most of these gases are intermixed (in effect, "dissolved") in air, such as carbon dioxide and cyanide, but some, such as sulfur compounds and gaseous hydrocarbons, are bound to the surface of the particles, often tightly, potentially changing their characteristics and toxicity. Therefore, particles should be understood not as a distinct phase unrelated to gas but as a complex mixture consisting of a particle core onto which is adsorbed other substances, including gases and volatile organic compounds. For example, the polycyclic aromatic hydrocarbons may exist in air as gases (the most volatile PAHs), as part of the matrix of a carbonaceous particle, and adhering to the surface of a particle. The single most important characteristic that determines the behavior of a particle (how long it stays suspended, where it lands in the lung, whether it migrates in the body) is size. Particles vary in size from the visible (soot) to the practically unmeasurable (ultrafine) (Fig. 5.2).

Gaseous combustion products tend to dissipate rapidly, especially in open air and when hot, and may be thought of as consisting of four non-exclusive categories in terms of toxicological behavior: (1) common combustion products that are benign or effectively inert (carbon dioxide), (2) common combustion products that exert their primary effect on the respiratory tract (phosgene, oxides of nitrogen), (3) common combustion products that cause systemic toxicity when absorbed by the pulmonary route (carbon monoxide, cyanide), (4) toxic air contaminants unique to a

particular situation such as hazmat operations or a fire in a production or storage plant (pesticide paraoxons, isocyanates). Of these, the second and third category are of greatest concern in characterizing the characteristic risks of municipal firefighters, but the fourth is critically important in defining the health risk of firefighter responders in specific situations (such as a fire in a factory or warehouse), military firefighters, and industrial firefighters. In general, combustion products in the gas phase are primarily of concern acutely and at the scene, although they may have chronic sequelae (consequences). An important feature of inhaled gases is that because of contact with and absorption by the moist epithelium (lining) of the respiratory tract, their penetration to the lower lung, where pulmonary edema may occur, is governed almost completely by solubility in water. This is why sulfur dioxide, which is highly soluble, mostly provokes severe cough but nitrogen dioxide, which is not, can cause potentially lethal pulmonary edema.

The role of particles is critical. Particulate matter from fires is carbonaceous, derived almost entirely from burning organic matter. Carbonaceous particulate matter is both a primary combustion product with its own effects (as soot or fine particulate matter) and a carrier of solid, gaseous, and potentially liquid-phase contaminants, such as solvent chemicals (which may also be in vapor form at the fire scene), polycyclic aromatic hydrocarbons, and nitroarenes.

The most important characteristic that defines the behavior of particles is size. Larger particles tend to deposit in the upper airway and are removed before they penetrate into the deep lung. Smaller particles (“particulate matter”) are capable of making their way to the deep lung and the smallest particles can even penetrate lung tissue and pass into the circulation. Generation of particulates from fire smoke has known characteristics. Fires with visible flames tend to produce smaller-sized (or “fine”) particulate matter than smoldering or charring fires without flames, a reflection of the less efficient combustion process. The finest particles come from polyurethane foam as fuel, but this is an anomaly. Overall, synthetic materials generate larger particles than wood, although they burn at higher temperatures. Particles generated by both flaming and non-flaming fires may aggregate into larger particles with time [4].

As important as characterizing the fine particulate matter in fire smoke is understanding what it is not. Fire smoke is not the same as cigarette smoke (which is even more complicated and, as noted, contains chemicals, such as nicotine, that act to suppress acute inflammation). The fine particles in fire smoke are also quite different in terms of chemical composition from fine particulate air pollution, which is largely derived from aggregated sulfate, with some derived from nitrate, rather than carbonaceous.

## Common Exposures in Firefighting

There is a bewildering variety of chemicals among these hazards. Which are present in varying concentrations and mixtures depending on the composition of the materials burned and the characteristics of the fire. In the most basic terms, the hotter the

fire, the less visible particle formation and the less acutely toxic the gases. The less hot the fire, the more likely it is to form clouds of particulate matter and the more toxic the gas, both acutely and in the long term for outcomes such as cancer risk. The simpler the fire substrate, in the sense of being closer to burning wood, the less toxic the emissions. The more complicated and rich in synthetic materials, the more toxic and the more likely the emissions are to be associated with chronic effects such as cancer. These are oversimplified rules of thumb, of course, but they work well in estimating the risk associated with fire smoke.

The products of incomplete combustion (such as carbon monoxide) and of lower-temperature burning tend to be most hazardous. Irritant gases, such as phosgene and cyanide, both of which are better known for their acute toxicity, and the higher oxides of nitrogen occur with more intense heat. Lower temperature combustion fails to completely oxidize the organic compounds and results in high concentrations of benzene, 1,3-butadiene, and polycyclic aromatic hydrocarbons (PAHs). Where a chlorine source is abundant (such as polyvinyl chloride plastics) chlorinated compounds such as trichloroethylene, vinyl chloride, and phosgene are a hazard, potentially associated with chronic and acute effects, respectively. Incidental exposure to hazardous materials may occur in one fire and not another due to their being released from sources in the burning structure or the presence on site of unanticipated hazards. In addition to smokeparticulate matter, there are often various dusts such as asbestos (still prevalent in older structures) or volatilized organic compounds (innumerable hydrocarbons, including styrene, benzene, and other compounds more familiar as solvents).

## *Asbestos*

Asbestos seems to derive its toxicity from its fibrous shape, not its elemental content. The type of asbestos most commonly used in North America was chrysotile. All forms of asbestos, as well as some other fibrous silicates, are carcinogenic, particularly causing lung cancer and mesothelioma, and cause non-malignant changes in the lung and pleura (lining of the thoracic cavity).

Asbestos is the naturally occurring fibrous form of six very different silicates (silicon and oxygen compounds, with varying metal and water content). It should be noted that the definition of asbestos is not mineralogical: it is commercial, and somewhat artificial. The six forms of asbestos are only the commercially marketed fibrous silicates; there are several others, including erionite, an equally hazardous fibrous silicate that is also naturally occurring but has no commercial use.

Asbestos would be most commonly encountered incidental to fighting fires in older buildings with structural insulation using asbestos products. That asbestos exposure is a problem for firefighters is now accepted because of the demonstration of high rates of mesothelioma (SMR 2.00; 1.03–3.49, SIR 2.29; 1.60–3.19), a cancer of the thoracic cavity essentially exclusively caused by asbestos exposure [5]. Suffice to say that it is now accepted worldwide that chrysotile, while less potent

than amphibole asbestos, is causally associated with both lung cancer and mesothelioma and with a variety of non-malignant lung disorders. While contemporary firefighters are unlikely to be heavily or repeatedly exposed to asbestos anymore, they are clearly exposed on occasion to this known carcinogen in disintegrating or disturbed structures containing asbestos materials.

## *Benzene*

Benzene is a cyclic (but not polycyclic, because it only has one ring) aromatic (meaning that it has a shared electron structure in the ring) hydrocarbon (meaning that it consists of carbon and hydrogen). It is a known IARC Group 1 carcinogen, established as a cause of a form of leukemia known as acute myelogenous leukemia (AML) and is suspected of an association with other types of leukemia and with certain lymphomas. It is also a known cause of a form of bone marrow failure called aplastic anemia and is almost certainly a cause of a related but rare condition known as myelofibrosis, both of which are associated with leukemia. Benzene is produced efficiently in combustion of organic material, especially at lower temperatures, and is the leading suspected cause of elevations in risk for leukemia and the coding aggregations that include leukemia.

Benzene is considered a highly specific carcinogen, in that the only cancer that it has been proven to cause unequivocally is AML. Some studies suggest a role as a cause of chronic myelogenous leukemia (CML) but the evidence is not clear and this outcome would be surprising because CML is a disease very distinct from the other leukemias; it is not just a chronic form of AML.

Recent recalculations of cancer risk associated with benzene suggest that it is even more potent than previously realized. Latency can also be quite short for cancer and is variable, on the order of months, with intense exposure, to many years. This is explained by the peculiar biology of the bone marrow, which is constantly producing blood cells, and which easily forms clones of transformed cells when production is suppressed.

## *1,3-Butadiene*

1,3-Butadiene is an organic compound produced by combustion that is present in fire smoke, cigarette smoke, diesel exhaust, and air pollution. It is well known and extensively studied because it is also a feedstock, used in large quantities together with styrene, as a polymer, It is used to make synthetic rubber for the manufacture of tires and together with acrylonitrile for nitrile polymer materials. Although 1,3-butadiene is hazardous, the polymers are quite safe and the butadiene-styrene polymer is even used in chewing gum.

1,3-Butadiene has been repeated studied and re-evaluated, in part because human and animal studies have been difficult to reconcile. It has become increasingly difficult to study the risk of 1,3-butadiene for the good reason that better controls are in place but also because the decline of the tire industry has dramatically reduced employment in the sector and therefore has limited the population of human subjects available for study.

1,3-Butadiene is highly chemically reactive (because of the double bond) and has long been considered a known animal carcinogen, which led to studies that finally identified it as a probable human carcinogen. IARC classifies it as a Group 1 (sufficient evidence) human carcinogen for leukemia. It is also suspected of causing lungcancer and lymphoma (not identified down to type). 1,3-butadiene is also known to be genotoxic and causes reproductive effects in animals due to gonadal atrophy, but the relevance of this to humans at typical exposure levels is not clear. Exposure to butadiene was associated with accelerated atherosclerotic heart disease in the synthetic rubber industry in the past but not now, probably because exposure levels were much higher.

## *Carbon Monoxide*

Carbon monoxide (CO) is a colorless, odorless, and nonirritating gas that is heavier than air and generated wherever there is combustion with a rich fuel-to-air ratio and relative oxygen deprivation, such as a smoldering fire or a low flame. CO is a particular hazard in fires, as a product of incomplete combustion, and therefore represents hazard to firefighters and fire victims. It is heavier than air and is particularly dangerous in confined spaces, where it may accumulate to high concentrations. Firefighters sustain significant exposure from CO, the characteristic product of incomplete combustion. Depending on the circumstances of the fire, firefighters may experience significant inhalation of CO and if self-contained breathing apparatus is not used, this sometimes reaches toxic and even fatal levels.

CO is the most common cause of environmental poisoning worldwide, including developing countries. It presents a particular and well-recognized hazard for firefighters. It is also an important constituent of cigarette smoke and so the baseline blood CO levels of smokers is higher than nonsmokers and generally proportionate (on a log scale) to when they last smoked. (In such cases, the possibility of concomitant cyanide toxicity should always be considered, as well.) CO, once inhaled, passes efficiently across the alveolar-capillary barrier and binds to hemoglobin quickly and almost completely. A consequence of the high affinity of CO for hemoglobin is that, over time, the level of carboxyhemoglobin rises with continued exposure as it is accumulated at the expense of oxygenated hemoglobin. CO then both prevents oxygen from occupying the binding site and, by a different mechanism, interferes with the release of oxygen at the level of the tissue. This reduces the capacity of blood to deliver oxygen to tissues. The net effect is progressively

less oxygenation of tissues with increasing accumulation of CO in the form of carboxyhemoglobin.

Carbon monoxide is directly cardiotoxic because it interferes with oxygen delivery to the heart muscle (myocardium), which is the highest oxygen-consuming tissue in the body. Oxygen requirements of the heart muscle (myocardial oxygen demand) is particularly high during periods of exertion, accelerated heart rate, and depleted blood volume (as by dehydration), all conditions that are common during fire suppression. Under these conditions, the heart needs more oxygen but the blood is unable to deliver it at the rate required, because carbon monoxide is blocking key binding sites in the molecule that carries it, hemoglobin. CO exposure is known to precipitate heart attacks (myocardial infarction) by direct means and, less often, may do so as well by inducing coronary artery spasm.

The result may be cardiac ischemia in persons with preexisting coronary artery disease; these changes may occur due to CO alone above 30 % carboxyhemoglobin. Induction of angina and increased frequency and complexity of arrhythmias have been demonstrated at levels as low as 6 % in subjects with coronary artery disease. Thus, one of the most serious health effects of even low-level exposure to CO is the risk of angina, ventricular arrhythmia, and possibly myocardial infarction in workers who may have silent or diagnosed coronary artery disease.

At high levels or for prolonged periods, CO may deprives the brain of oxygen; the higher the exposure and the longer the duration, the worse the injury but there is much individual variation. Higher levels of CO may result in vision changes, seizures, and ultimately coma, permanent neurological injury if the victim survives the comatose state, or death. CO exposure also may result in either focal, stroke-like injury of particular brain structures or diffuse damage that resembles degenerative disease. Sudden exposure to very high levels may be fatal in minutes with no warning, due to chemical asphyxiation.

This was the conventional view of CO toxicity for many years. Recently, however, it has come to be appreciated that CO plays a role, still not completely clear, as a gaseous neurotransmitter in modulating activity in the central nervous system. This mechanism may explain discrepancies between what is observed in survivors of CO poisoning and what would be predicted due to oxygen deprivation alone. These complexities are beyond the scope of this chapter.

Non-smoking adults normally have carboxyhemoglobin levels at about 1 % and develop symptoms such as headache when their levels rise, variably, above approximately 5 %. Heavy smokers may not feel symptoms and may perform normally with levels of 5–10 %, at which non-smokers would demonstrate cognitive impairment on neurobehavioral testing. Tolerance to higher carboxyhemoglobin levels renders smokers less susceptible to the effects of CO, at least at lower concentrations.

Duration of exposure is as important as the level of exposure to CO because carboxyhemoglobin accumulates over time in a steady rate. Ventilation patterns also play a role in the exposure; higher minute ventilation results in increased accumulation. Significant elimination of CO occurs only when the atmospheric levels are low. Inhaled CO follows a strict mass effect: the amount of CO in the body is

determined, when the atmospheric concentration is elevated, by the product of concentration in the air, ventilatory volume (not rate) over time, and duration of exposure. Nothing else affects the determination.

## *Cyanide*

Cyanide (CN) is a colorless gas that is lighter than air and is perceived by those with the genetic capability to smell it as having an almond-like odor. In fires, it exists as the gas hydrogen cyanide. Hydrogen cyanide is released as a product during the combustion of plastics (particularly nitriles) and natural polymers, including silks, wool, and cotton. Hydrogen cyanide enters the body by inhalation and from the lungs passes into the bloodstream quickly. It is distributed rapidly throughout the body.

Unlike carbon monoxide, cyanide gas is irritating to the airways of the lung and so can cause cough and shortness of breath at relatively low concentrations and an irritative bronchitis in survivors.

The primary and lethal effect of cyanide is to prevent cells in critical organs of the body from taking up and utilizing oxygen. Like carbon monoxide, the organs affected first are those that require a high oxygen uptake, including the heart and lung. Symptoms of acute cyanide poisoning include loss of consciousness in seconds, seizures, coma, respiratory arrest, and cardiac arrest, which can occur within minutes after exposure to moderate to high concentrations of cyanide.

CN causes toxicity by inactivating mitochondrial cytochrome oxidase, which is critical for cells to derive the energy needed to stay alive. Cell death occurs because cells are unable to utilize oxygen in tissue in energy metabolism. CN is also highly irritating to mucous membranes and causes eye and throat irritation. It is quite possible that cyanide has other effects on the body but these have been less well studied.

## *Diesel Exhaust*

Combustion of diesel fuel in a diesel engine takes place at high temperatures and under high pressure, which favors complete oxidation. Emissions are therefore low in carbon monoxide, high in oxides of nitrogen, favoring formation of relatively simple hydrocarbons and particulate matter. Emissions are variable, depending on the fuel, in producing sulfur oxides which are subsequently transformed in the atmosphere to sulfate. Therefore, fresh diesel exhaust is a complex mixture coming out of the engine. Diesel exhaust becomes even more complex after it has undergone atmospheric transformations, because once released into the atmosphere diesel exhausts “age” and undergo various transformations due to photochemical reactions and aggregation of sulfate particles into fine particulate air pollution.

The distinction between fresh diesel exhaust and the aged diesel contribution to air pollution is critical.

Emissions from diesel exhaust present three sets of problems for firefighters. The primary problem for firefighters in general is with emissions of fresh diesel exhaust when engines are started up in response to an alarm. In the enclosed space of a fire station, firefighters can be exposed to an acute dose of exhaust. The secondary problem, for the community, is that airborne diesel exhaust contributes to community air pollution. (This is a broader public health issue, not an occupational problem unique to firefighters.)

Diesel exhaust, both fresh and aged, is recognized to be carcinogenic and causally associated with human cancer, specifically of the lung, in populations and occupations exposed to diesel exhaust. In June 2012, the International Agency for Research on Cancer (IARC) reclassified diesel-engine exhaust as a Group 1 carcinogen, meaning that there is sufficient evidence to conclude that diesel exhaust causes cancer in humans, drawn from both epidemiology studying exposed populations and toxicology studies using animal studies. However, this finding was not a surprise. In 1988, concluded that diesel exhaust was *probably* carcinogenic to human beings but the evidence was not completely conclusive [6]. IARC is a body of the World Health Organization that has as a primary purpose the evaluation of world knowledge to determine cancer risk from exposures to various agents. IARC is essentially universally considered authoritative in the field of cancer research.

The case is strongest for lungcancer. Several developments since 1988 persuaded IARC that the case for the carcinogenicity of diesel fuels had been fully made and was no longer speculative. The most important was the availability of studies on railroad workers, truckers, and underground miners who use diesel-powered equipment. The most important single study was on railroad workers, and showed an excess risk on the order of 1.40. because of the putative exposures involved, the risk of other cancers are likely to be raised as well, specifically upper airway, kidney, and bladder, which share many risk factors with lung [7].

It is well established that specific chemicals present in diesel exhaust cause cancer. In addition to many compounds already known to cause cancer, including and especially PAHs and 1,3-butadiene. Nitroarenes (see above) are nitrogenated versions of complex organic compounds called polycyclic aromatic hydrocarbons (PAHs) which are formed by combustion and comprise a mix of organic chemicals, several of them potently carcinogenic (See above).

Despite interest in cancer, few studies are available for human beings on acute respiratory and cardiovascular responses to fresh diesel-engine exhaust, because this has not been seen as a pressing problem. However, it is clear that fresh diesel-engine exhaust has potentially significant acute effects and that small particles have effects distant from the lung and into kidney tissue.

The gas phase of fresh diesel exhaust does not contain the many secondary pollutants that are important in urban air pollution but, depending on running conditions, may be rich in formaldehyde (a potent respiratory and mucosal irritant and upper airway carcinogen) and acetaldehyde. The particle phase of diesel exhaust also has irritant potential and may induce inflammation. Recent subchronic and

acute animal studies suggest that fresh (non-aged) diesel-engine exhaust results in relatively mild inflammatory effects.

The particulate phase of urban air pollution is derived in part, and until recent changes in diesel technology, largely from diesel-engine exhaust emissions. Fresh diesel-engine exhaust produces coarse and fine particulate matter, nitric oxide (nitrogen dioxide is a secondary product not present in diesel exhaust), carbon dioxide, some carbon monoxide (much less than gasoline engines), and oxidized sulfur compounds (sulfur dioxide and sulfates), variable depending on the sulfur content of fuels.

## *Fire "Retardants"*

Fire retardant chemicals are added to finished combustible products to prevent, delay, or slow down combustion and make the material more resistant to fire. Their principal, but not exclusive, use is to retard combustion of synthetic and natural textiles and polyurethane foam. They are heavily used in developed and developing countries, especially in Asia.

Because of the extensive use of fire retardants worldwide and export of treated products, people living in developed countries such as the US, the UK, and Ireland (the latter two countries having particularly high levels of use due to fire prevention-legislation) have detectable tissue and blood levels of these compounds (principally the polybrominated diphenyl ethers, PBDEs).

Specific important fire retardants are discussed under "polyhalogenated organic compounds". The most common are polybrominated diphenyl ethers, which are analogues of polychlorinated biphenyl compounds, (PCBs), discussed below; fire retardants also include many inorganic compounds (generally not of toxicological concern), organophosphates, and combined halogenated organophosphates. Fire retardants in common use today, besides antimony used on firefighting turnout gear, are usually halogenated with bromine, the chemistry of which resembles chlorine. The PBDEs chemically resemble the active configuration of thyroid hormone and other hormones. These chemicals are suspected to be human carcinogens, on the basis of laboratory studies, and cause reproductive and neurological abnormalities in laboratory animals. Some have properties of other chemicals of known toxicity and carcinogenicity, such as the PCBs, which were once themselves used as fire retardants.

The effectiveness of brominated fire retardant chemicals, principally the PBDEs, is vigorously disputed. The evidence submitted by the fire retardant industry for the safety and efficacy of the compounds is weak, based primarily on a single study with equivocal results that used much higher applications of PBDE than are currently used commercially.

Firefighter advocates assert strongly (see Chap. 2) that the current generation of fire retardants is ineffective for the purpose of fire prevention and places their health at risk. This is among the most controversial issues in the fire service today.

## *Formaldehyde*

Formaldehyde may or may not be a significant risk in firefighting. Formaldehyde is a Group 1 IARC carcinogen. Formaldehyde is a known nasal carcinogen in rodents and a suspected lung carcinogen in human beings. It is present at high concentrations in cigarette smoke and other combustion products. Formaldehyde is highly reactive and interacts immediately with tissues with which it comes into contact. If it plays a role, it most likely contributes to risk in lung cancer risk and possibly the lymphomas, by modifying proteins and causing antigenic stimulation, but this is speculative.

## *Nitroarenes*

The polycyclic aromatic hydrocarbons (PAHs) are well characterized toxic chemicals om their own right but they also have nitrogen-substituted derivatives that have similar effects and potency as carcinogens. They are a set of nitrogenated products generated most efficiently in diesel exhaust.

Chemically, a nitroarene can be described as a PAH with one or two nitro- groups (NO−) added on the outside. These nitroarenes are otherwise identical to the PAHs in basic structure. For every PAH, therefore, there is at least one and potentially several homologous nitroarenes. Several nitroarenes have recently been determined by IARC to have demonstrated sufficient evidence for carcinogenicity in experimental avenues and therefore possibly carcinogenic to humans (Group 2B): 3,7-dinitrofluoranthene, 3,9-dinitrofluoranthene, 1,3-dinitropyrene, 1,6-dinitropyrene, 1,8-dinitropyrene, 6-nitrochrysene, 2-nitrofluorene, 1-nitropyrne, 4-nitropyrene; a tenth, 3-nitrobenzanthrone, is considered to have limited evidence [7].

It has long been known that diesel exhaust was rich in PAHs and their corresponding nitro-arenes, several of which are potent carcinogens known or strongly suspected to cause human cancers: lung, skin, bladder, kidney and upper airway (including nasopharyngeal). Mechanistic evidence is also strong or moderate for the carcinogenicity of several of the nitroarenes, providing a more than plausible chain of causation. The same issues of mixtures and interactions described for the PAHs also apply to the nitroarenes.

The nitroarenes have been recognized as lung carcinogens (Group 1) by the International Agency for Research on Cancer (IARC). The IARC report, which is volume 105 in the IARC monograph series, has just appeared on-line (in 2013) and will be available in hard copy in 2014.

## *Oxidant Gases*

Oxidant gases consist of gas-phase airborne chemicals that have an oxidizing effect chemically in solution (and in tissue) or photochemically. They include nitrogen dioxide (requires high temperatures), nitric oxide (precursor to nitrogen dioxide and

produced in vehicular exhaust), phosgene (requires a chlorine source), ozone (rare in fire situations), and a large number of organic and nitrogenous compounds (many related to nitrogen dioxide) that are important in photochemical air pollution but probably not at fire scenes. Sulfur dioxide and the sulphates (which are formed from the oxidation of sulphur dioxide), by comparison, are toxic through irritation but are not oxidant gases.

The oxidant gases are potent lung irritants and can be very dangerous in special situations, such as confined spaces. Nitrogen dioxide, in particular, is formed at high temperature with increased yield from high temperatures and so is a potential hazard from uncontrolled diesel exhaust, but in practice serious and potentially lethal exposure occurs mostly in hazardous materials situations involving nitric acid. Phosgene is an even more potent oxidant and is implicated in both lung injury and potentially kidney effects. However, concentrations of both these gases do not reach high enough levels at a typical fire scene to be major hazards.

## *Particulate Matter (Fine and Ultrafine)*

Fine particulate community air pollution has been extensively studied because it is known to be associated with mortality and illness in populations, although the effects on individual is not predictable. Cardiovascular effects of air pollution are associated primarily with fine particulate levels, as a risk factor for cardiovascular mortality. These effects may occur in normal individuals without unusual susceptibility. Respiratory effects of air pollution, particularly complicating chronic bronchitis, may place an additional strain on cardiac function. In air pollution studies, the lag time for mortality form cardiovascular events associated with fine particulate air pollution persists beyond 24 h, which is the current criterion for recognizing "heart attacks" as occupational when they occur after a fire. This suggests that the allowable period for accepting cardiac events as related to fire smokeexposure needs to be longer than the customary 24 h, perhaps about 36 h.

Combustion in general generates clouds of small particles of varying sizes. The particulate matter of greatest concern is in the "fine" size range, which starts with 2.5 μm (micrometers, or "microns") and gets smaller. ("Ultrafine" starts at 0.1 μm.) Size is important for several reasons. The smaller the particle, the smaller the mass (which is the traditional measure of exposure, or dose) but the count of particles is much more numerous for a given mass and the combined surface area of the particles is much greater. This means that fine particulate matter that is biologically reactive is much more toxic than an equivalent (and, usually, much larger) mass of "coarse" particles, such as soot. In that size range, particles can also easily penetrate the tissues of the lung and enter the bloodstream. As a result, fine particulate matter exerts a toxic effect, particularly on the heart, much greater than its small mass would suggest.

Urban air pollution has a number of similarities with fire emissions, specifically the health risk of particulate matter in ambient air pollution from the combustion of fossil fuels, especially diesel emissions. However, there are also important differences.

The mix of sources of combustion products are not the same and the "aging" effect in the atmosphere chemically modifies the airborne chemicals and results in new particle formation.

Urban air pollution involves predominantly pollutants that have "aged" in the atmosphere for a period, usually for hours. The "aging" process in air pollution is important in the particulate phase for agglomeration of larger particles from fine particle nuclei and for increasing adsorption of volatile and aerosolized contaminants and in the gas phase for photochemical processes that lead to secondary pollutants, such as ozone, nitrogen dioxide, and aldehydes. In air pollution, fine particulate matter is also formed from the aggregation of sulfate (formed from the oxidation of sulfur, mostly in diesel fuels) into fine particles. It is unlikely that diesel exhaust in a fire station or from a fire pit would generate sulfate-derived particulate matter because this process takes time.

Emissions from fire smoke are formed at lower temperatures than occur in diesel engines and the usual sources contributing to community air pollution. Fire smoke involves fresh emission of combustion products and may or may not include sources of metals and chlorine that modify the characteristics of carbon particles. Fine particulate matter in fire smoke consists primarily of carbonaceous particles but they are not carbon alone. These particles carry volatile chemicals adsorbed on their surface and so have toxicity beyond that of the carbon alone. The presence of metals also adds to the toxicity, apparently through the catalysis of reactions that promote inflammation. The characteristics of fine particulate matter generated at a fire scene may differ from that found in ambient air pollution. Studies on this have only begun.

The size distribution of particles is somewhat more complicated than initially described. Ambient air pollution consists of particulate matter in three somewhat overlapping distributions characterized as cut points but best understood as distinct particle populations: coarse ($\leq$10 μm aerodynamic diameter, containing the bulk of the particulate mass), fine ($\leq$2.5 μm), and ultrafine ($\leq$0.1 μm, representing the largest number of individual particles), each of which represents a particular mode or population of particulate matter differentiated by composition as well as size. In other words, size is not only important in and of itself, but as a marker for different species of particles.

The smaller the particle size, the larger the surface area. Because the surface of these particles has a high affinity many biologically active chemicals, surface adsorption is critical to the biological effects of particulate matter. Fine and ultrafine particulate matter have many orders of magnitude greater capacity for binding volatile organic compounds in their surface and delivering them to deeper structures.

Particles in the coarse mode penetrate efficiently to the lower respiratory tract and are efficiently retained in the alveoli. However, they are also large enough to be deposited efficiently on the epithelial surface of bronchi and small airways and are thus likely to have airways effects as well as alveolar effects, mediated in part by macrophage uptake, and systemic effects. Particles in the fine range penetrate to the alveoli efficiently but are less likely to deposit in airways and more likely to migrate from the deep lung into the circulation and adjacent structures through intracellular junctions and through cells.

Ultrafine particles consist largely of aggregated or agglomerated structures of sulfate or nitrate, some with carbonaceous nuclei. These agglomerated particles tend to stick together when they touch, forming larger agglomerates over time. Fine particular matter consists of both carbon-derived particles, on which are adsorbed volatile and organic materials, and agglomerated sulfate and nitrate ultrafine particles, which build by accretion into the fine size range. Ultrafine particles behave more like gases than particles in their flow behavior and penetration to the deep lung, and migrate relatively freely, with the potential for systemic effects, however, the evidence for significant health effects is weaker than for fine particulate matter.

At the other extreme, coarse particulate matter in community air pollution predominantly consists of dust, particles of crustal origin (basically, very small dirt particles), bioaerosols, and, of interest in this context, carbonaceous particles formed by combustion on which are adsorbed a variety of volatile and organic materials.

The adsorbed chemical species on both coarse and fine particles are biologically significant. The particle forms a carrier with a large surface area onto which are adsorbed many constituents, particularly: volatile organic compounds, polycyclic aromatic hydrocarbons and nitroarenes, metals (particularly transitional metals and iron, which may be proinflammatory), sulfate, and oxides of nitrogen.

Particulate matter in modern urban air pollution is closely associated on a population basis with mortality risk, the risk of cardiovascular and respiratory disease, pneumonia (indicating an effect on susceptibility), emergency room admissions for asthma, and lungcancer risk. These effects, including and especially mortality, are linearly related to the concentration in air of fine particulate matter. (The relationship is not so clear for coarse or ultrafine particles.) Mortality is most apparent in the aged and chronically ill but are also visible in healthy younger populations, which has led to various theories of mechanism. One explanatory theory is that the timing of exposure is critical because people pass into and out of previously unrecognized stages of susceptibility for many factors, including and especially blood coagulability and thresholds for inflammation.

## *Polycyclic Aromatic Hydrocarbons*

The polycyclic aromatic hydrocarbons (PAHs) are a large family of organic compounds, built on multiple (two or more) aromatic rings, hence the equivalent name "arene". Several of them are known carcinogens (x is used here to indicate various isomers):

- Benz(a)pyrene x = a,e [isomers of benzpyrene; a is much more common]
- Dibenz(x)pyrene x = a,e; a,h; a,l [isomers of dibenzpyrene]
- Indeno(1,2,3—c,d)pyrene
- Benz(a)anthracene
- Benz(x)fluoranthracene x = b,j,k [isomers of benzfluoranthracene]

- Dibenzanthracene
- 7-H-dibenzocarbazole
- 5-methyl-chrysene
- Acridine(s)

In addition to recognizing individual PAHs as causing cancer in human beings, IARC has long recognized mixtures of PAHs, such as appear in fire smoke, to be carcinogenic, in Group 1 as a mixture. The significance of recognizing mixtures, apart from individual compounds, is that the PAHs always appear naturally as mixtures, usually with very similar composition and predictable concentrations relative to the most commonly used indicator of exposure, benz(a)pyrene. Some constituent PAHs have not yet been characterized for carcinogenicity. Combined exposure involving some PAHs which have characteristics of cancer promoting agents, with or without cancer initiation activity, may also produce a positive interaction with PAHs that are direct carcinogens, increasing the carcinogenic potency of the mixture.

The PAHs are also important in combination with other exposures characteristic of firefighting. They are products of incomplete combustion and are responsible for carcinogenesis in many settings, including as constituents of cigarette smoke. They are known constituents of fine and ultrafine particulate matter and of diesel exhaust. An analogous series of chemical compounds are heterocyclic with nitrogen and are also known to be carcinogenic, but these have not been as well characterized. PAHs are the leading exposures imputed in causing the many cancers that are elevated in firefighting.

PAHs have also been associated with accelerated atherosclerosis. They have been implicated in experimental studies to promote vascular disease and the development of coronary artery disease in animal studies.

The PAHs are also known constituents of fine and ultrafine particulate matter, described above, but their primary role as significant toxic agents for human beings is as carcinogens and in inducing chronic disease. This is a large family of organic compounds, several of which are known carcinogens. They are products of incomplete combustion and are responsible for carcinogenesis in many settings, including as constituents of cigarette smoke. An analogous series of chemical compounds are heterocyclic with nitrogen and are also known to be carcinogenic, but these have not been as well characterized.

## *Polyhalogenated Organic Compounds*

Polyhalogenated organic substances are organic compounds substituted with chlorine, bromine, or fluorine, which may be formed or released during a fire. Public concern currently revolves around brominated compounds used as “fire retardants”. However, the polyhalogenated hydrocarbons, particularly the dioxins and furans and the polychlorinated biphenyls (PCBs) are more widespread and have been of

concern for much longer for their general population risk. Like the PAHs, they always occur in mixtures.

Polyhalogenated (polychlorinated and polybrominated) hydrocarbons have characteristics in common, among them a strong tendency to concentrate in lipid-containing tissues of the body due to their preferential partition into and retention by lipid and the relative protection of some compounds to metabolism and breakdown. These same compounds are known as "persistent organic pollutants" (POPs) when they occur in the environment and they are also persistent in the human body. However, halogenated hydrocarbons are not necessarily elevated in all firefighters and appear to be associated primarily with specific fire events, not cumulative exposure to fires in general, and not all "POPs" are involved [8]. Each class has different "congeners", structural forms that the various organic compounds and numbers of halogen atoms attached to them can assume. Although some classes of polyhalogenated hydrocarbons and certain congeners within each class are highly refractory to biotransformation, others are not and over time blood and lipid levels slowly decline, all other things being equal. The congeners that remain are not necessarily those that played a role in carcinogenesis at the beginning of the process. One congener of PCBs in particular, 1,2,3,4,6,7,8-HpCDD, appears to be characteristic of exposure in fires because it stands out among moderate elevations of other POPs [8]. However, testing for this congener, or PCBs in general, or any of the other POPs is expensive and cannot be readily interpreted in the individual case, and so is not recommended. A history of exposure to a fire where they are likely to have been present in the fire smoke should suffice as evidence of exposure.

Brominated compounds have been used extensively in the past as fire retardants. Polybrominated fire retardants are heavily used in consumer products to reduce flammability, although their effectiveness is controversial. Although some of the polybrominated biphenyl compounds may be quite toxic, exposure to brominated compounds has not been considered to be an appreciable risk associated with firefighting. This may reflect lack of investigation, since these compounds are difficult to study and work with. They are suspected of presenting a health risk in children, however.

Dioxins and furans (more accurately, polychlorinated dibenzodioxins and—furans, but colloquially called "dioxins") are potent organochlorine compounds that are formed most efficiently during combustion in the presence of a carbon source at temperatures between 200° and 400°. Below this temperature window, they do not form efficiently and above the window they break up. Dioxins and furans also bioaccumulate because they are metabolized slowly and stored efficiently in lipids. Most of the concern for dioxins and furans comes from their high potency (they are among the most potent toxic agents known) which can cause health effects at very low exposure levels. The primary health effect of concern is carcinogenicity. Whether dioxins and furans initiate in addition to promoting cancer has been sharply debated but the consensus that appears to be emerging is that it can and there is no doubt that these compounds are among the most powerful cancer promoting agents known. They have an equally potent effect in inducing metabolism of other so-called "incomplete" carcinogens, enhancing their initiation of cancer by stimulating

expression of enzymes that convert them to a more active form. Thus, for adult exposures, cancer risk drives control and standards setting, even though the cardiovascular system is also emerging as another important target organ. Presumably due to induction of liver cholesterol-forming activity and local effects on the blood vessels favoring atherosclerosis, dioxins are now known to induce and accelerate atherosclerosis and therefore the risk of coronary artery disease in animal studies. In human studies, exposure to dioxins has been associated consistently with increased mortality from cardiovascular disease and especially ischemic heart disease (mostly myocardial infarction, the familiar "heart attack"), although there are many limitations and potential confounding factors in these studies [9]. Whether this is an important effect in human beings is not clear but the potential exists for dioxins to increase the risk of coronary artery disease among firefighters. Dioxins and PCBs also interfere with some hormones, including thyroid hormone, but this is more of a threat to children and theoretically to the fetus than to adult firefighters. Because dioxins and furans are formed de novo from combustion products, they are a permanent feature of fire smoke and an continuing management problem.

Polychlorinated biphenyl compounds (PCBs, of which there are 209 congeners) are not formed in settings of combustion but may be released from sources present at the fire scene, particularly in fires involving electrical transmission facilities and old transformers. Some of the PCB congeners act much like the dioxin congeners described above. Many of the PCBs have dioxin-like properties but generally at much lower potency. The major issue with the PCBs is that most of them are very slow to be metabolized, either by human beings or in the ecosystem, and stored efficiently in lipids. At ambient temperature they persist and are bioconcentrated, with amplification up the food chain. Thus, the more stable PCBs have become difficult management problems in ecotoxicology, very slow to degrade and persisting for years or decades. Many of the congeners (210 each) are toxicologically irrelevant. A few are highly toxic and have effects on the immune system and interfere with hormone activity, particularly thyroid hormone, which has best been described for children in other contexts of exposure. Of greatest concern for adult firefighters, the PCBs are also promoters, if not initiators, of cancer at multiple sites. Most concern for the PCBs and efforts to set protective standards have therefore centered on the potential for carcinogenicity. Because the PCBs are no longer being produced commercially (with very few exceptions), this problem should be diminishing.

Perfluoroalkyl acids (PFAAs), of which the most commonly encountered are perfluoroctane (PFOA, also called C8), perfluoroctane solfanate (PFOS, the active ingredient in Scotchguard®), and perfluorohexane sulfonate (PFHxS, a carpet treatment), are used as fire suppression chemicals and surfactants. They are found in fire extinguishing foam and surfactant (stain-resisting) surface coatings. Experimental studies have shown possible carcinogenic effects (on bladder) and heart disease. Firefighters have been shown to have elevated levels of PFOS and, significantly, PFHxA but not PFOA in serum; the levels were low and the differences were not great in absolute terms compared to other employment categories, but the coefficient of variation for the greatest difference was 33 % (for a mean of 39.28 ng/ml for PFOA). This suggests that firefighters may be at elevated risk of effects from

exposure to the PFAAs, depending on the potency and nature of the health effects, if established in human populations [10]. To date, however, the empirical evidence for PFAAs playing a causal role in firefighting is inadequate to form a judgment.

### *Trichloroethylene*

Trichloroethylene is a solvent used primarily as a degreasing agent. Trichloroethylene is properly called trichloroethene and often TCE or "trike"; it is not to be confused with trichloroethane, nor with vinyl chloride, which is sometimes called chloroethene but has a very different toxicity profile. Trichloroethylene is generated in fire smoke because of the presence of abundant chlorine sources but relatively little is generated in cigarette smoke.

Trichloroethylene is known to be present in modern fire smoke but is absent from cigarette smoke because tobacco is deficient in chlorine donors. Trichloroethylene is classified by IARC as a human carcinogen (Group 1) for kidneycancer and as a probable carcinogen (Group 2A) for other cancers, primarily the non-Hodgkin lymphomas. The US National Cancer Institute now considers trichloroethylene as an established human carcinogen. The evidence suggests multiple target organs in addition to kidney, including liver and, especially, the lymphatic system as lymphomas [11–20].

## Appendix: A Primer on Toxicology

Toxicology is a sophisticated and complicated science, grounded in biochemistry and physiology. This section is a short introduction to toxicology, provided so that references on individual toxic chemicals may be easier to follow by anyone concerned, whether physician, lawyer, human resources staff, hearing officer, judge, or legislator. This section should be unnecessary for experts, however. Experts, of course, should go to the primary literature whenever possible and should know all or most of this material already.

The most authoritative and convenient references available for individual chemicals are the Toxicological Profiles developed for the Agency for Toxic Substances and Disease Control. These publications are available in hardcopy or without cost on-line (http://www.atsdr.cdc.gov/toxprofiles/index.asp). Each Toxicological Profile summarizes the world literature on a particular chemical exposure, objectively and comprehensively. If they have a fault, however, it is that they tend to be too complete and sometimes include studies that are not particularly helpful. They also do not always explain the significance of the information. Overall, however, they are exceptionally well written and produced. Reading them can be a chore for someone who does not have a background in toxicology. Fortunately each one comes with a summary in nontechnical language in front. To get the most out of them or any such resource, however, it helps to know the principles of toxicology.

Toxicology is the science of how the human body handles and responds to chemicals. The part that has to do with how the body is exposed to chemicals and how they are handled is called “toxicokinetics”. It can be thought of as “what the body does to the chemical”. The part about how the body responds to the chemical is called “toxicodynamics.” It can be thought of as “what the chemical does to the body.”

Toxicity occurs as a result of the effect of the chemical on the body and the body’s response to it. Toxicity can range from barely detectable to death. When toxicity results in a set of symptoms and signs, called a “toxidrome”, that is characteristic of a particular chemical exposure, it is called “poisoning”, but toxicologists do not use this word as freely as it is used in regular language. (They prefer “toxicity.”) Once a chemical substance encounters the body and enters that route of exposure, it may cause local problems at the site of entry, for example lunginjury due to smokeinhalation, or it may enter the body (absorption), be carried somewhere else (distribution), and may cause problems elsewhere in the body. This is called “systemic toxicity.”An important example of systemic toxicity for firefighters is carbon monoxide, because carbon monoxide does not injure the lung at all, but it enters the body through the lung and has effects on many other organs.

A toxic effect may be “acute”, meaning occurring quickly. In toxicology, “acute” does not mean intense, as it may in common language. Carbon monoxide causes acute effects. A toxic effect may be “chronic”, meaning that the outcome evolves over a longer time. Cancer is a chronic effect.

## *Toxicokinetics*

“Toxicokinetics” describes what happens when the chemical gets taken into the body. Toxicologists often refer to all chemical substances not normally present in the body as “xenobiotics.” These can be chemical pollutants, drugs, venoms and natural poisons, cosmetics and personal care products, and even nutrients. The acute toxic effect of a xenobiotic is proportional to the concentration and for most is determined by concentration in the blood. This in turn depends in part on the rate of absorption and entry into the blood.

How a chemical reaches the body is called the “pathway of exposure” and the means by which it enters the body is called the “route of entry.” There are only so many routes of entry into the human body: breathing it in, getting it on skin or mucous membranes, or swallowing it; everything else is artificial and applies more in the lab than in real-world exposures, such as getting an injection.

For a gas or a particle of smoke coming off of a fire, the “pathway” is being carried on air and the route of exposure is inhalation, because the gas or particle is breathed into the lung. For skin contact, the pathway might be deposition of soot onto a surface which is then touched and the route of exposure is skin contact.

There are four phases that describe what happens to a xenobiotic in the body: absorption (how a xenobiotic gets into the body), distribution (how it is delivered to various parts of the body by the bloodstream and other means), metabolism (the

chemical biotransformation of the xenobiotic), and excretion. Figure 5.3 illustrates these four steps. The latter two steps, metabolism and excretion, combined, constitute "elimination," because they describe the removal of the xenobiotic from the body.

### Absorption

Xenobiotics may enter the body through any of several routes of entry, of which the most important for firefighters is inhalation. Inhalation is most important for firefighters but skin contact plays a small secondary role as a pathway for volatile organic compounds. Ingestion is comparatively unimportant for firefighting-related exposures.

For firefighters, by far the most common and the most important "route of exposure" is inhalation, when the chemical is airborne and breathed into the lungs. There is hardly any barrier between the air in the lung and the bloodstream, so chemicals in the form of a gas that are inhaled enter the circulation very quickly and completely. Inhalation delivers a lot of the substance to the blood stream quickly, leading to a high but short peak concentration. An example relevant to firefighters is carbon monoxide, which enters the bloodstream from the lungs very quickly, attaches to haemoglobin in red cells in the blood almost instantaneously, and is carried throughout the body within seconds. After that, other processes take over to determine blood concentration. In the case of carbon monoxide, the molecule detaches itself from haemoglobin slowly, over hours.

The lung has some means to protect itself. There are many "host defense mechanisms" that serve to protect a person (or an animal) from the effects of chemical exposures and to reduce absorption. Particle clearance from the lung is especially important, with special particles (alveolar macrophages) that engulf and remove inhaled particles, and a special mechanisms called the "mucociliary escalator" that bring up particles from the deep lung through moving a thin mobile layer of mucus upward. A soluble particle may be broken down by the alveolar macrophage which may release its constituent chemicals into the bloodstream.

Exposure by inhalation results in relatively efficient absorption of gases and a quick peak concentration of blood if the gas can penetrate to the deep lung. Whether the gas will penetrate efficiently depends on its solubility in water, reflecting clearance rates in the bronchial tree. Particles, on the other hand, are subject to a number of host-defense mechanisms in the respiratory tract that limit the efficiency of penetration to the alveolar level. Once there, their size prevents them from passing directly into the bloodstream and they must dissolve or be digested by macrophages (defense cells that engulf bacteria and particles and that try to digest them) before their constituent chemical contents can be absorbed and enter the bloodstream. Particles may contribute to systemic toxicity if they are composed of a soluble material, such as lead or polycyclic aromatic hydrocarbons. Particles at nanoscale (on the order of 100 nm or so) often have very different characteristics than larger particles of the same composition and tend to be much more toxic.

Absorption into the bloodstream after skinexposure is relatively slow and incomplete. Turnout gear is largely protective, preventing most skin contact. Some

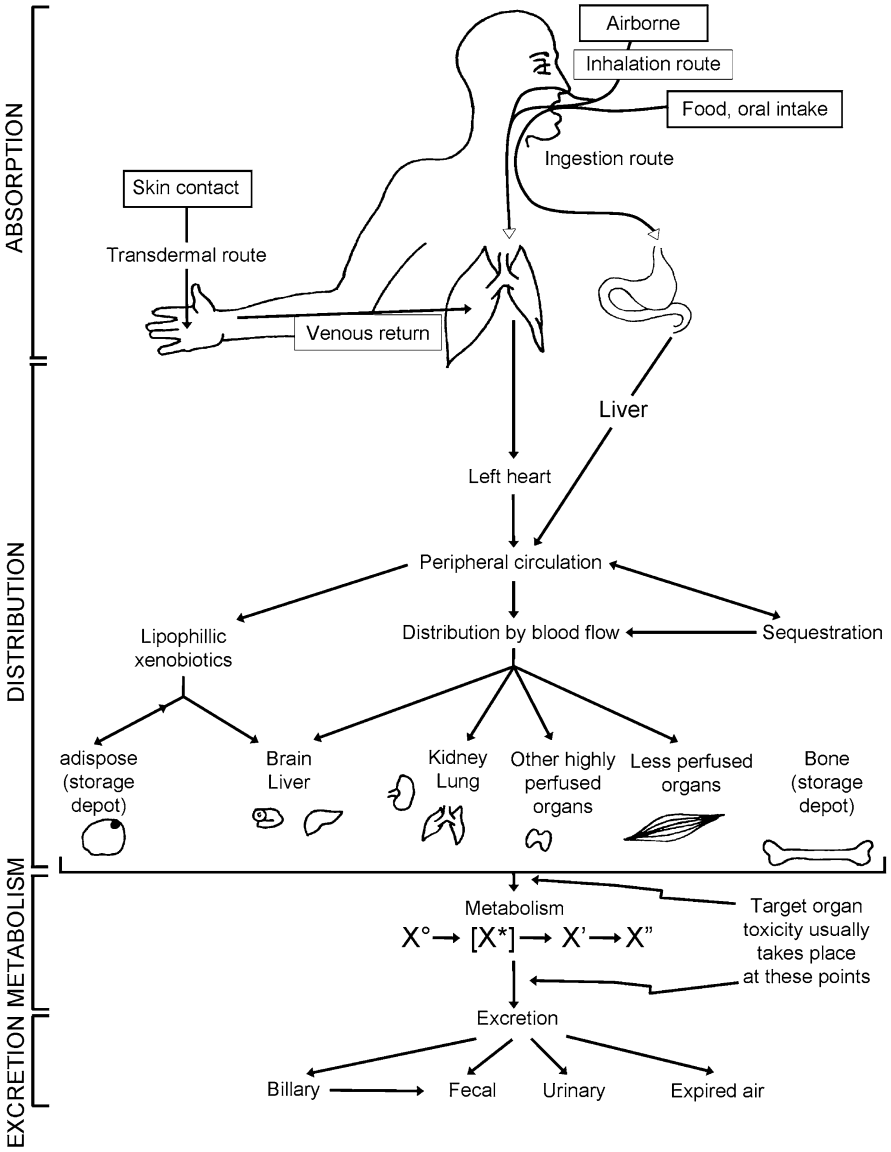


**Fig. 5.3** Principles of toxicokinetics: the passage of a chemical through the body. (Adapted from Guidotti TL. Principles of occupational toxicology (Chap. 7). In: Zenz C, Nickerson B, Horvath EP, eds. *Occupational Medicine: Principles and Practical Applications*, 3rd edn. Chicago, Mosby-Year Book, 1994, pp. 70–84.)

small concentrations of volatile xenobiotics such as solvent chemicals can be detected in the breath of firefighters who are fully protected by the inhalation route with SCBA and wearing turnout gear. This shows that despite some skin absorption does take place, even with protective clothing, but not much.. Skin is also the local

target organ for skin carcinogens, such as polycyclic aromatic hydrocarbons. Skin can be penetrated quickly by chemicals that dissolve easily in fats ("lipid-soluble") and this can facilitate the penetration of other chemicals as well. Within the context of firefighting, skin exposure is generally more likely to be important as a cause of local problems of the skin, including irritation and skin cancer, rather than as a route of exposure for systemic toxicity.

The third common route of exposure, ingestion, which is less important for firefighters than the other two. Ingestion is an important route of exposure for water and food and occasionally in special situations where food or a cigarette is contaminated by something on a person's hands.

### Distribution

Once the xenobiotic is absorbed and enters the bloodstream, it is transported to the capillary level in tissues of the body where it becomes available for uptake by the target organ. After one pass through the circulation, the xenobiotic is uniformly mixed in arterial blood, regardless of its route of entry. The peripheral tissues are therefore presented with an increasing concentration of xenobiotic in the blood which peaks and then declines, as the xenobiotic is distributed to tissues throughout the body, removed and stored in whatever tissue depots may accumulate the xenobiotic (sometimes this depot consists of proteins in the blood itself), and then eliminated by metabolism or excretion.

Uptake of a xenobiotic from the plasma by an organ depends on the blood flow to the organ and the affinity of the tissue for the material. In most cases, delivery of a xenobiotic depends on the blood supply to a tissue relative to its weight. The liver and kidneys each receive massive fractions of the cardiac output and are therefore presented with circulating xenobiotics in quantity.

Special transport mechanisms exist at the cellular level for some xenobiotics. As mentioned above in the context of absorption into the body, absorption of a xenobiotic from the bloodstream into the tissue depends importantly on the solubility of the xenobiotic in fat (how "lipophilic" it is). Lipophilic agents will accumulate in lipid-rich organs such as the nervous system or in liver. Organs with a lipid (fat) content accumulate much larger concentrations of highly lipophilic xenobiotics, such as organohalide pesticides or the PCBs, than occurs in plasma or in other organs. One important implication of storage in fatty tissues is that accumulation in breast tissue results in subsequent excretion into breast milk, which is the major route of exposure to a variety of xenobiotics for infants who breast feed. Where the physicochemical properties of the organ attract and bind metals, as in bone and kidney, a metal or semimetal xenobiotic will be sequestered and will accumulate over time.

Entry into some tissues is restricted by special barriers to passage, such as the blood-brain barrier and the placenta. These barriers generally keep out more toxic classes of xenobiotics and permit passage of nutrients and xenobiotics that resemble nutrients. The brain also receives a disproportionate fraction of the cardiac output but is partly protected by the blood-brain barrier; this barrier works well for most polar xenobiotics but is permeable to lipophilic compounds.

## Metabolism

Many xenobiotics are substrates for intracellular enzyme systems. There are many biochemical pathways and enzyme systems that metabolize xenobiotics, either as a primary function or as an incidental function to another role. From the standpoint of evolutionary biology, it is thought that most of these mechanisms of metabolism developed to detoxify and excrete harmful substances ingested in foods (especially natural toxins from plants and those in spoiled or putrefied foodstuffs) or to metabolize and therefore control levels of endogenous chemical compounds (such as steroid hormones).

These enzyme systems transform the xenobiotic in a series of steps from the original compound to a series of stable metabolites, often through unstable intermediate compounds. For many xenobiotics there are many pathways of metabolism, resulting in numerous metabolites. These transformations may have the effect of either "detoxifying" the xenobiotic by rendering the agent toxicologically inactive, or of "activating" the xenobiotic by converting the native agent into a metabolite that is more active in producing the same or another toxic effect. (By convention, a metabolite that is activated or unstable is often indicated with an asterisk.) An active xenobiotic may be transformed into an inactive metabolite, effectively removing the agent from the body in its toxicologically active form. The metabolism of xenobiotics ranges widely in scope, from highly specialized biochemical pathways, such as the biotransformation of cyanide, to complex and alternative pathways with several steps, such as occurs with benzene. For most important organic compounds, such s the polycyclic aromatic hydrocarbons (PAHs), however, there is a pattern to biotransformation.

In general, the enzyme systems available for the metabolism of organic xenobiotics such as the PAHs usually consist of two phases, especially in the very important "mixed function oxidase" (MFO) system. Phase I involves converting lipid-soluble, water-insoluble compounds into water-soluble products that are more easily excreted in urine or bile. This often results in activation, and results in a metabolite capable of interacting with macromolecules, such as DNA. Activation of a procarcinogen, for example, into a carcinogen may be the initial step in the early stages of carcinogenesis. Phase II involves the removal or conversion of chemical groups in such a way as to render the molecule more polar and therefore more easily excretable by the kidney (and less easily diffused back across the renal tubular epithelium after filtration). In the process, the activated xenobiotic metabolite from Phase I usually becomes inactivated. This process frequently involves "conjugation," the attachment of a chemical group (such as sulfonate or glucuronic acid) that makes the molecule much more hydrophilic (water soluble). This makes it much easier for the body to excrete the xenobiotic through the kidney or liver and eliminate it from the body.

Some enzyme pathways, such as those in the MFO system, have the effect of activating xenobiotics so that the metabolite is more toxic than the original chemical. Most, however, detoxify the chemical and clear it from the tissue and bloodstream and so contribute to its elimination.

The most complicated metabolic pathways are those for organic compounds. Metals may also be metabolized, however. The methylation of mercury and arsenic, especially, plays a major role in their toxicity. The methylation pathway of arsenic is species- specific and the reason why arsenic is a carcinogen in humans but not in animals.

### Excretion

The xenobiotic or its metabolites would remain and accumulate within the body if there were no mechanisms for excretion. Elimination is the term used for removal of the xenobiotic, especially from the bloodstream, by excretion or metabolism or sequestration (storage).

The kidney is the major route of excretion for most xenobiotics. The liver, besides being an important metabolizing organ, secretes some xenobiotics, including heavy metals such as lead and mercury, into bile, which passes into the small intestine, through the large intestine and out in feces. Sometimes, metabolites that are excreted in bile will be reabsorbed in the small intestine and will recirculate, a phenomenon called "enterohepatic circulation." Enterohepatic circulation causes many xenobiotics and metabolites, such as mercury and many organochlorines, to persist in the body much longer than they would otherwise.

Xenobiotics and their metabolites are also eliminated by various minor routes. Some gases leave the body by passing directly from the bloodstream into air in the lungs and are exhaled. Lipophilic xenobiotics, such as organochlorines, may also be excreted in breast milk, which is potentially a consideration for exposure of the child but is usually not a significant route of elimination from the mother. Water-soluble agents are filtered through sweat glands much as they are in the kidney, but this is not an important route of elimination. It is not true that a person can be "detoxified" from toxic substances by sweating in a sauna, steam bath, or sweat lodge.

## *Toxicodynamics*

There are as many potential mechanisms of toxic effects as there are reactions in biochemistry and functions in physiology, there are a few processes that have special characteristics that affect their behavior, such as inflammation (particularly important in lung disorders) and the causation of cancer (carcinogenesis).

Carcinogenesis is a complicated process. The body is constantly bombarded by carcinogenic chemicals and other influences. Carcinogenic chemicals that make it through the body's defences alter DNA and other molecules in the body continually but mostly nothing happens. Infrequently, at random, there is an event resulting in damage to DNA that does matter. This event causes a mutation or other defect that changes the cell's control mechanisms but the cell can still divide and grow. The process proceeds stepwise, with each step controlled by a more or less a random event. Each step and the total sequence takes time, which is called "latency." For environmental cancers, the latency of a cancer from the time of first exposure to detection of the tumor typically takes many years (generally 15 or more) or decades. (There are exceptions in which latency is shorter, for example, leukemia.) Unless stopped at a given step or the cell shuts down, this leads to a clone of abnormal cells that can form a tumor, can invade surrounding normal tissue, and can metastasize elsewhere in the body (often through the bloodstream).

Cancer is a disorder of genetically-determined control of cell division and growth which, once it begins, proceeds by its own biological determinants. While cancer can arise in any living tissue (although it is rare in many) and manifest itself in many tissue types, the actual number of genetic defects is limited, probably to only a few dozen. In theory, in the future it may be possible to assess causation of cancers much more accurately by looking at the genomics of the cancer of a particular tissue type rather than its site. Because the underlying cellular event occurs at random, cancer occurs at random in the susceptible population. One person may get it and the next person will not, purely by chance. However, increasing exposure to a carcinogen increases the probability that it will occur in an individual and increases the number of cancers that can be observed in the population. Epidemiologists use this association to identify probable causes of cancer.

The central principle in toxicodynamics is the relationship between exposure to the xenobiotic and the adverse response on the body. This relationship, called the "dose-response" or "exposure-response" relationship is the most fundamental idea in toxicology: the more of a toxic chemical one is exposed to, the greater the effect. Toxicologists often quote one of the great scientists of the late Middle Ages, Paracelsus, who was the first to recognize that "the dose makes the poison." Everything is toxic if the dose is high enough, including water. (Water intoxication, and the seizures it causes, is very rare and more often the result of an endocrine or psychiatric disturbance.) However, at a low enough level, everything is "safe," even the most potent poisons known. (Botulinum toxic and ricin remain lethal down to exceedingly low dosage levels but even they have levels below which there is no effect.)

There are three distinct varieties of the exposure-response relationship that need to be separated out. These are:

- The toxicological dose-response relationship, which refers to the principle that the response at a tissue or cellular level is proportionate to the amount of the agent delivered to the tissue; the other exposure-response relationships build on this one
- The clinical dose- or exposure-response relationship, which refers to the principle that in a given individual (human or animal), different symptoms and signs may appear as different effects predominate with increasing exposure; this is what we want to know for an individual
- The epidemiological exposure-response relationship, which refers to the principle that in a population of individuals, the cases of disease or toxicity become more frequent with increasing exposure; this is what we want to know if we are concerned about whether cancer is caused by a chemical.

In this chapter, and in most handbooks of hazardous substances, we are most concerned with the clinical exposure-response relationship. This type of exposure-response relationship describes what one would see in an individual with increasing exposure, which symptoms and signs would apper, and in the end what the toxidrome of severe toxicity (poisoning) would look like.

The epidemiological exposure response relationship relates exposure levels to the frequency of the response in a population, based on the number of people showing a sign characteristic of the outcome (such as a symptom) or the entire toxidrome

(which in epidemiology is called a "case definition"). This is the essential approach used in environmental epidemiology and yields what is usually called the "epidemiological" exposure-response relationship, with increasing count or rate associated with increasing exposure. In epidemiology, one is interested in how frequently a response is associated with a given level of exposure in a population. Recognized cases (based on the toxidrome, or some simplified "case definition", such as cancer type) are counted as cases, and if there is an association (and if it is causal) the frequency of cases should increase with increasing exposure. An increasing number of cases cross this threshold and are observed with increasing exposure, yielding the "epidemiologic" exposure-response relationship, which relates magnitude of exposure to frequency of disease, not severity. This relationship is particularly important for disorders that are "stochastic" (arising on a probabilistic basis) rather than showing gradations of severity as a result of exposure, such as cancer, immune-mediated disorders, and infectious disease.

Occupational toxicologists are very concerned that exposure to complex mixtures, such as fire smoke. Mixtures have the potential for numerous interactions and for producing unpredictable effects. Some xenobiotics are well known interact with others to produce disproportionate effects. For example, many chemicals in air hitch a ride onto particles (by adsorbing onto their surface) and penetrate more deeply into the lung than they wood otherwise. The classic example of a positive interaction (often called "synergism) is the combination of cigarette smoke (and possibly other forms of smoke) with asbestos: for cigarette smoke, the risk of lung cancer is a multiple of the risk from asbestos alone, not just the simple addition of the same risk as from the smoke alone. Other xenobiotics do not seem to interact and exert their actions in an additive way. Absence of an interaction is more likely when the pollutants are all members of the same class or have relevant chemical properties in common. For example, the combined effect of different PAHs that cause cancer is additional cancer, not a synergistic risk.

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# Chapter 6
# Cancer

**Tee L. Guidotti**

Firefighters were long suspected to have an excess risk of cancer but most investigators assumed that the site of greatest significance would be lung cancer. Early modern studies showed relatively low elevations, if any, in cancer risk overall and elevations in several sites other than lung, but only modest excesses in lung cancer, of a magnitude (roughly 150 % expected) common to many occupations, including many without exposure to fire smoke [1]. This excess was usually (but not convincingly) apportioned to cigarette smoking. As research filled in the gaps, however, this picture from the 1970s and 1980s turned out to be false and a new synthesis emerged: a real elevation due to occupation is obscured by lower individual risks among fire department members compared to the general population, which is the usual reference population for comparison. The pattern of their cancer risk should be well below the general population, given firefighters' profile of risk factors (including smoking). Instead, the absence of a strong healthy worker "lifestyle" effect overall, the elevations that become visible when internal reference groups are used (based on exposure), and the profile of cancers involved (mostly at sites plausibly associated with carcinogen exposure) all suggest that firefighters do indeed have an elevation in risk for cancers arising from their occupation but that this is offset because their smoking and other lifestyle factors should place them at lower risk than the general population. Furthermore, for some cancers the elevations are sufficiently strong and consistent among well-designed studies and relevant subgroups to have given rise to legislated presumptions. The cancers most often accepted as being associated with firefighting are kidney, bladder, testicular, leukemia, non-Hodgkin lymphoma (itself actually a family of cancers), brain, colon and rectum, and prostate.

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It is to be expected that firefighting confers a risk of cancer, given the exposure of firefighters to many known carcinogens. However, superficial analyses of the association of cancer with firefighting suggested that the excess for cancer overall is not particularly large compared to other occupations. This in part because the most obvious cancer association, lung, has been difficult to demonstrate. That cancer rates are in fact elevated for firefighters overall was often doubted in years past because it was not appreciated that firefighters have historically had low rates of smoking. High demands for fitness and stamina also tend to select against smokers, even at young ages; this aspect of the healthy worker effect has not been adequately investigated. However, when these risks are examined thoroughly, with due attention to subgroup analysis, exposure-response relationships, and collateral evidence, a different picture often emerges.

Certain relatively infrequent cancers, such as kidney, bladder, and testicular, demonstrate risk estimates that may be quite high in individual studies, but because the actual numbers of these particular cancers are low, statistical power is lacking and so there is great variation among studies which is often perceived as inconsistency. The demonstrable elevations in the most common cancers for men, such as lung, prostate, and colorectal, have not been extreme, and estimates tend to cluster around a relative risk of 1.5, which is often difficult to interpret (See Chap. 4). Risk for lung cancer, of course, has been confounded by cigarette smoking but probably not to the extent that has been assumed.

At least part of this paradox may be that in the past there were many more smoking firefighters and cigarette smoke is the single most important confounder for fire smoke-related cancers. In recent years, the prevalence of cigarette smoking among male firefighters has declined even more precipitously, and from a lower level, than for the general population of men. This may mean that the risk of combustion product-related common cancers among firefighters relative to reference populations may actually appear to decrease as a statistical calculation, although individual risk may still exceed the general population in fact. These and other interpretive problems are particularly acute for cancer, with its generally long latency period (except for leukemia) and would require longitudinal studies of representative firefighter cohorts to monitor [2]. Unfortunately funding constraints and limited interest among likely sponsors makes it unlikely that a high-quality longitudinal study could be maintained over the years that spans eras with fine-grained information to identify transitions.

## How to Use This Chapter

This chapter on firefighters cannot be used without interpretation as guidance for individual cases. Circumstances vary and each individual case must be evaluated on its merits.

This chapter provides an overview of the evidence for an association that is plausibly causal between different cancers and firefighting. That information, by itself,

is valuable in establishing the potential merit of a case or evaluating the evidence for a rebuttablepresumption on a group basis. However, it is not sufficient in itself to argue definitively for causation in an individual case. Experts, investigators, and other interested parties who use this book as a reference are strongly encouraged to seek and review thoroughly the most recent available information, not only for firefighting but for other causes, particularly those that could potentially rebut a presumption of causation. Whether making a case for or against causation or a policy of presumption, the experienced expert should spend as much time trying to poke holes in his or her own argument as in formulating it.

The literature search and database for this chapter is current as of 2014. The convention for expressing epidemiological risk estimates throughout the book is given in Chap. 1 (point estimate; upper bound–lower bound of the 95 % confidence interval). The basic principles of epidemiology are outlined in Chap. 3. Interpretive issues that apply to the firefighting literature are outlined in Chap. 4. In this chapter, rates refer to male firefighters, only, unless female gender is specified. This is because there are still relatively few female firefighters.

## Cancer Causation Issues

It is often written that there is insufficient data on firefighters to make a determination with respect to cancer risk. Actually, the data available on firefighters are among the most complete available for any occupation for risk of cancer. That is why firefighters have not only been studied for their own sake but have been used as a study population to examine broader issues in methodology and workers' compensation [3]. The problem is that most cancers are "rare" (in the epidemiological or biostatistical sense) and so any one study has low statistical power because there are relatively few cases available to support the analysis. In such situations, there will always be some studies that are "negative" (show no evidence of an elevation) and some that are "positive" (suggest an association). However, epidemiology is not a game played with a scorecard.

For the scientist (most likely an epidemiologist), the literature on firefighters presents a challenge. One needs to examine the individual studies carefully and to look for overall patterns to make sense of it all. It is not unusual for a study to report a finding of no elevation (i.e. "negative") for the group overall but for there to be associations and even exposure-response relationships within subgroups. Similarly, there are always a few studies that are indeterminate. The indeterminate studies usually feature an association that is strong in magnitude but does not achieve conventional statistical significance because of small numbers. These associations are often dismissed as "negative" but an alternative interpretation is that they are indicating an association but limited in showing it because of small numbers. The logic of analysis is detailed in Chap. 4.

The expert, no less than the adjudicator, must, to be fair to the client, separate his or her thinking from the intellectually lazy approach of applying scientific certainty

to the problem. Scientific certainty is unachievable for the individual case. It is almost never achievable for most problems of general causation in the real world, including firefighters' risk. The applicant or claimant cannot possibly achieve this standard. For an expert to adhere to a standard of scientific certainty is to impose an unacceptably high risk of a Type II error (rejecting a conclusion when it is in fact true) where it is not appropriate. To do so also contravenes the explicit legal instructions of both civil litigation and the workers' compensation acts that are based on it. These require the expert to offer an opinion on the basis of the weight of evidence, not scientific certainty.

## Interpretive Issues for Cancer Causation

Cancer has been at the center of the wave of presumptionlegislation that has changed compensation qualification for firefighters in North America and Australia. However, the evidence for an elevation in cancer risk on which it is based has often been controversial, usually inconsistent (as is to be expected with statistically rare outcomes), and often to be found only after looking carefully at subgroups and for evidence of an exposure-response relationship. The whole story is rarely told by a summary risk estimate.

The earliest studies of cancer frequency were based on mortality, then later on incidence. These studies suggested an almost naïve optimism that if an elevation in risk were present it would surely be found [4–6]. It soon became evident that there were daunting methodological and interpretive difficulties. Cancer in particular presented many difficult problems of interpretation, even beyond the issues discussed in Chap. 4.

Many interpretive problems are specific to cancer and not unique to firefighting, although they are especially severe for this occupation. These include:

- Latency, the duration of the period between first exposure and cancer detection, which is often short for leukemia and occasionally certain "solid" cancers following overwhelming exposure to radiation or specific chemical carcinogens (such as analine dyes or auramine for bladder cancer), but long for most other cancer and sometimes very long, as for mesothelioma and the non-Hodgkin lymphomas
- Exposure assessment, which appears superficially easy for firefighters from employment records but in reality can be complex, because years of service, alone, does not tell the whole story
- Era of entry, which is closely related to exposure assessment, because much has changed over the years: firefighting strategy and technology, personal protective equipment, and the material combusted have all changed
- Detection bias, since most professional firefighters, as municipal employees, have had better health care coverage in the US for longer than in other occupations, such that screening tends to identify more cancers and at an earlier stage

- Confounding by cigarette smoking, which is less of a problem for firefighters in the current era, but which may confound the older studies and require statistical manipulation to interpret
- Cancerresearch methodologies that obscure associations, such as the common practice of aggregating lymphomas to attain greater statistical power.
- Gender, because there is comparatively little empirical information on cancer risk for female firefighters because of low participation in the firefighting workforce; even with increasing recruitment, the number of women within the fire service is small.

## Occupational Risks for Cancer

Firefighting as an occupation involves exposure to many carcinogenic agents, which can be classified as follows:

- Carcinogenic chemicals arising from combustion, including polycyclic aromatic hydrocarbons (PAHs) and their nitrogen-containing analogues, and benzene
- Carcinogenic chemicals incidental to structural firefighting, including asbestos (predominantly chrysotile in North America) and polycyclic chlorinated biphenyl compounds (PCBs) and their corresponding furans
- Carcinogenic chemicals arising from work as a firefighter, including arenes from diesel exhaust
- Ultraviolet radiation, which may operate as a separate risk factors (for example, for wildland firefighters spending long periods outdoors without adequate skin coverage), by interaction with photosensitizing chemicals (such as many of the PAHs), or through interaction with initators or promoters of skin cancer (again, PAHs being the obvious examples)
- Electromagnetic fields, a somewhat speculative cancer risk factor, discussed below
- Shiftwork, which is a recognized cancer risk factor by IARC [7] and thought to operate through a neuroendocrine mechanism.

Specific chemical risks are described in Chap. 5. The most important route of exposure is inhalation, which places the lung at potential risk and is also a highly efficient pathway to the circulation. Sufficient absorption also occurs across the skin occurs that cancer risk may be enhanced or modified due to changes in the kinetics (excretion and metabolism) of carcinogens [8].

Recently, a paper appeared written by the respected former director of cancersurveillance and the cancer registry for Washington state. In it, he ventured the opinion, while providing no evidence, that increased cancer risk in firefighters may be caused by exposure to strong electromagnetic fields (EMF), a conclusion he inferred from the distribution of cancers by type, which is similar to that of cancers purportedly associated with EMF [9]. This is a weak argument and the parallel is highly unlikely to be significant. The spectrum of cancers he cites as a profile is actually highly nonspecific.

The association between EMF exposure and cancer risk is highly controversial and much less well grounded on evidence than that for fire smoke. There are many alternative explanations in chemical exposure, so that the need to invoke an unlikely cause such as EMF is not compelling. In short, EMF is not likely to be an etiologic factor for firefighter-associated cancers. The paper was published in a journal (*Medical Hypotheses*) that has as its reason for existence the airing of speculation to promote discussion, not presentation of evidence.

## Review Literature

There are many reviews of the cancer risk of firefighters, some of them excellent and many not so good, usually because they are not analytical. Each review soon becomes obsolete as new information accumulates with further data. This review literature will therefore not be discussed in detail here, in the interest of emphasizing primary sources.

Meta-analyses, as described in Chap. 4, are systematic studies that derive a statistical estimate of the risk, called the "summary risk estimate" from the findings of a set of studies. While subject to the same obsolescence as unstructured reviews, meta-analyses have the advantage of deeper analysis and yield a quantitative estimate of risk based on more than any one study. Meta-analysis is very useful in determining the trend of the literature for site-specific cancer rates overall. However, by its nature meta-analysis is uninformative about subgroups that could reveal important information useful for an appropriate method in causation analysis. Meta-analysis also cannot examine or meaningfully take into account methodological or classification differences among studies. In Chap. 4, the limitations of meta-analysis is discussed in detail and it is suggested that this estimate and the strengths of the method may conceal effects that are only visible when one looks at subgroups and for patterns.

In 2006, Lemasters et al. published a meta-analysis that summarized risk estimates for the world literature for most cancer sites [10]. The methodologically sophisticated meta-analysis by LeMasters [10] suggested an elevated risk for non-Hodgkin lymphoma, testes, multiple myeloma, and prostate, the last highly controversially.

Youakim conducted a more recent meta-analysis, limited to a few cancer sites in studies that considered duration of employment [11]. Youakim [11] demonstrated a statistically significant elevation among cohort mortality studies for cancers of kidney and brain and for non-Hodgkin lymphoma, and an elevation among subgroups for cancers of bladder and colon and for leukemia. This is consistent with the world literature and supported by subgroup analysis for several of the sites [3], as will be demonstrated further on in this chapter.

As will be noted, the two meta-analysis studies do not overlap at all in identifying candidate cancer types. Close examination suggests that the theory-based exclusionary criteria used in the algorithm for the study by LeMasters led to a "downgrade"

(the authors' terminology) of at least two cancer associations that would otherwise have met the criteria of most meta-analyses. This example demonstrates that meta-analysis, while providing a summary synthesis of the literature, is highly sensitive to study design and selection.

These meta-analyses, but not an earlier and now obsolete meta-analysis by Howe and Burch [12] from 1990, are referred in this report to where they are relevant. Howe and Burch (1990) should now be considered of historical interest only.

However, there is one review that matters above all else, and that is the meticulous evaluation undertaken by the International Agency for Research on Cancer (IARC), the authoritative UN body for cancerstatistics and prevention. In 2007, IARC recognized firefighting as associated with three cancers: testicular, prostate, and non-Hodgkin lymphoma. Firefighting, as an occupation, was therefore classified in Group 2B, "possibly carcinogenic to humans" on the basis of "limited evidence of carcinogenicity in humans" (See Chap. 5). The IARC criteria more nearly reflect scientific levels of certainty, rather than weight of evidence [7].

## Epidemiological Evidence for Cancers

The Appendix to this chapter summarizes the overall findings for most studies of cancer in firefighters that examined multiple outcomes. This table should not be used without qualification and elaboration, because the overall risk estimates can be as misleading as informative. Studies dating from before 1995 will be discussed in detail in the rest of this report only when there is a particular issue or point to be made, as they have been thoroughly discussed elsewhere. The reader is reminded that the convention for expressing the risk estimate and the 95 % confidence interval around it is (risk estimate; lower bound–upper bound). The reader is also reminded that when the risk estimate is greater than unity and the confidence interval is greater than unit risk (a relative risk of 1.0, or a standardized mortality ratio of 100 using the old percentage nomenclature), then the risk estimate is elevated with 95 % statistical certainty, meaning that the true risk that is being estimated would fall somewhere within the confidence interval 95 % of the time if the study were repeated many times. This convention is used more frequently in the following section, on site-specific cancer frequency.

Many early contributions to the literature on cancer mortality of firefighters were published before 1990 and used as their endpoint total cancer mortality or restricted their analysis to selected groups of cancer, usually aggregated, usually for relatively small firefighter populations. These studies have little probative value by today's standards, although they were valuable in initiating research in this important area. They are included in the summary table in the Appendix. This section summarizes the studies that meet contemporary standards.

Giles et al. [13] examined firefighters employed during the decade 1908–1989 by the Metropolitan Fire Brigade of Melbourne in the state of Victoria, Australia, with 95 % ascertainment and matched them to the state cancer registry to determine

standardized incidence, making it one of the earliest incidence studies. The numbers were relatively small and even the highest SIRs (standardized incidence ratios) showed wide confidence intervals and failed to achieve statistical significance. This paper was overlooked for many years because it was published in a limited-distribution Canadian government statistical publication that was not indexed in the usual sources. Despite the obvious effort put into the work by the authors, who were investigators at the Victorian Cancer Registry, there were no further reports from the group and there was no follow up in the world literature.

Burnett et al. [14] conducted a very large proportionate mortality study on firefighters in 27 American states from 1984 through 1990, using data from the National Occupational Mortality Surveillance (NOMS) system. Limitations of these data are partially overcome by the sheer size of the database, which, with 5744 deaths among white male firefighters, is beyond what could be achieved in any one cohort study.

Deschamp et al. [15] studied the recent experience of a relatively small number of fire fighters in Paris from 1977 to 1991, a period of 14 years. An elevated SMR was found for respiratory cancers (1.12), gastrointestinal cancers (1.14) and genito-urinary cancers (3.29) among other findings. However, the study is anomalous in several ways, uniquely demonstrating an elevated mortality from stroke (1.19) and a very low overall mortality (0.52), the lowest reported to date among firefighters. The significance of these anomalous findings cannot be interpreted with the available information.

Ma et al. [16] conducted a large study using the same database as Burnett et al. [14] to explore race-specific disparities in cancer mortality. The study was not intended to replicate or overlap with the Burnett et al., as its purpose was different, but it was much smaller and covered a mostly overlapping population, so it should not be considered to be a study independent of Burnett et al. For this study, the NOMS database was extended by 3 years to 1993 but lost data from three states that dropped out. As expected, the results were similar. Race as coded on the death certificates yielded 1817 deaths of white firefighters and 66 deaths of black firefighters. Of greater interest is the pattern of race-specific elevations. Ma et al. found an excess of cancer of the brain, specifically, in African-American but not white firefighters.

Bates et al. [132] reported a study on firefighters in New Zealand from 1977 to 1996, conducted to investigate the observation of a cluster of testicularcancer [17, 18]. This elevation was confirmed as finding independent of the cluster. This study is unusual in reporting both cancer incidence and mortality. It reports one of the lowest mortality ratios reported for firefighters (0.58), suggesting a strong healthy worker effect, unlike other studies. Bates et al. observed no significant elevation except for testicular cancer. The authors caution that matching to mortality data and cancer registration data may be incomplete prior to 1990 and suggest that they have greater confidence for findings after this date. The authors found a marked increase in testicular cancer and nonsignificant elevations in incidence in the 1977–1996 cohort of cancers of interest: lung (1.14; 0.7–1.8), which showed a modest increase with duration of service, bladder (1.14; 0.4–2.7), brain (1.27; 0.4–3.0), and "myeloleukemia" (1.81; 0.5–4.6), but not kidney (0.57; 0.1–2.1). Limiting the analysis to

the 1990–1996 subcohort, however, they found the increase in testicular cancer and a deficit in the same cancers, except for brain (1.59; 0.3–4.6), and no kidney or "myeloleukemia" cases. A strikingly different picture is observed in the pattern of deaths, however. Mortality among firefighters in the 1977–1996 cohort is elevated for bladder cancer (2.73; 0.3–9.8) but less than expected for lung (0.86; 0.4–1.6), brain (0.68; 0.1–2.4) and "hematopoietic cancer" (0.72; 0.2–1.8), and no deaths from testicular cancer. The discrepancy between incidence and mortality in cancers with a high case mortality, such as lung, is an anomaly. However, all numbers are small and the authors are candid in describing limitations of the database outside their control.

Baris et al. (2001) [19] conducted an exemplary cohort mortality study. This study should be accorded great weight because among recent studies it has exceptional power, spans most of the twentieth century, and has the most complete follow-up. The study therefore merits description in detail.

The cohort consisted of 7789 Philadelphia firefighters employed from 1925 to 1986 compared to US white male rates, comprising 204,821 person years of follow-up. The men were hired in their late 20s (on average) and worked for approximately 18 years, with an average of 26 years follow up. Baris et al. examined their cohort by age, duration of employment, job assignment and by number of runs to fight fires (enumeration of responses from the firehall) in three broad ordinal categories.

There were 2220 deaths among the members of the cohort. All causes of death and all cancers were approximately equal to the expected rates for all U.S. white males. The authors did observe statistically significant excesses for coloncancer (RR 1.51; 1.18–1.93). Nonsignificant excesses were reported for cancers of the buccal cavity and pharynx (1.36; 0.97, 2.14); for non-Hodgkin lymphoma (1.41; 0.91, 2.19); for multiple myeloma (1.68; 0.90–3.11) and for lung cancer (1.13; 0.97–1.32). With >20 years of firefighting, the following cancer sites showed elevated risks: colon cancer (1.68; 1.17–240); kidney cancer (2.20; 1.18–4.08); non-Hodgkin lymphoma (1.72; 0.90–3.31); multiple myeloma (2.31; 1.04–5.16); and benign neoplasms (2.54; 1.06–6.11).

Baris et al. developed a direct index of exposure by assessing risk by three categories of firefighting runs, with low exposure being less than 3322 runs; medium exposure being greater than or equal to 3323 and less than 5099 runs; and high exposure being greater than 5099 runs. Cancer of the pancreas showed a clear dose–response with rose from 1.02 for low to 1.17 for medium to 1.61 for high exposure. Although there were no other tumor sites with exposure-response gradient, when comparing low exposure (1.00) to high exposure, several cancer sites demonstrated increasing risk: stomach, 1.20; pancreas, 1.42; leukemia, 1.22; and benign neoplasms, 2.06. The authors also compared lifetime runs with diesel exposures, including a category of non-exposed.

Several cancer sites demonstrated increasing risks in the medium and high categories compared to unexposed: buccal cavity and pharynx, prostate, brain, multiple myeloma, and leukemia. There was also an apparent dose–response for assessment of low, medium and high exposure related to diesel exhaust for mortality from respiratory diseases (but not for any cancer). The risk rose from 1.00 (non-exposed) to

1.37 for low exposure to 1.45 for medium and finally to 1.49 among those in the high exposure group. Interestingly, there was no such exposure-response relationship for number of runs over the career of the firefighter (regardless of diesel exposure).

All of these excesses have relevance to toxicology and inhaled toxic hazards found in the firefighting profession, except the excesses for benign neoplasms. This is a "wastebasket", or residual category of diagnostic rubrics. Thus, it is not clear whether this represents a true elevation in some unusual class of tumor or (more likely) misclassification.

From the Baris et al. study, some tentative conclusions emerge. There were no significantly *reduced* SMRs for any of the a priori tumor sites plausibly linked with firefighting: brain, bladder, kidney, and lymphatic malignancies, as one might expect with simply random error. Further, the Baris study adds weight to observed associations between firefighting and cancers of lymphatic system and with kidney, and suggests associations with colon, pancreas and prostate cancers.

Ma's second paper [20, 21] was a cohort study of firefighters in the state of Florida yielding cancerincidence, not mortality. Studies of incidence are preferred to study cancers that are rarely or usually not fatal, such as thyroid cancer. The strength of this study was its very large population base and person-years of observation (over 413,000) and the accumulation of a very large number of female firefighters (2017), previously virtually unstudied. However women only entered the fire service in large numbers very recently and so there were only 52 deaths among the female firefighters in the Ma cohort. The risk of all cancers was significantly elevated for women (SIR expressed as a RR = 1.63; 1.22–2.09) but the pattern of cancers that showed an elevated risk among female firefighters suggested bias or confounding in this subgroup: cervical, thyroid, and Hodgkin disease. There were nonsignificant elevated risks among the women for kidney, stomach, colon and rectum, but not breast. Among male firefighters, the study confirmed elevated rates of cancers of bladder (1.29; 1.01–1.62) and testicular tissue (1.60; 1.20–2.09), and yielded an unanticipated finding, a significant elevation in cancer of the thyroid (3.97; 1.45–8.65). There was no elevation and actually a lower estimate of risk among men for brain, lung, and cancer of the lymphatic and hematopoietic systems, aggregated.

Bates [22], who also conducted the aforementioned study in New Zealand, conducted a registry-based case–control study of cancer among firefighters in California, comparing the odds of association with cancer types compared to all other registered cancer cases (Bates 2007). The advantage of this study was the enormous subject population. The Bates study is remarkable in confirming previously reported patterns of cancer risk (brain, testes, prostate), and in identifying elevations for esophagus and melanoma.

Kang et al. [23] was a registry-based cancer incidence study of 2125 white male Massachusetts firefighters during the years 1987 through 2003. The paper was a second installment of a study on Massachusetts firefighters, following up on an older study with the same study design [24]. It covers a relatively brief time period. Using standardized mortality odds ratios (SMOR), the authors compared firefighters

to police and to subjects in the registry for which occupations other than firefighting had been recorded, a highly artificial synthetic population intended to represent the employed population. For all cancers for which there was an elevation (colon, brain, bladder, kidney, and, unexpectedly, Hodgkin disease) the SMOR was higher when compared to police than to the general population. If one assumes that the frequency of disease is likely to be higher in the general population, this points to a differential in healthy worker effects, in which the healthy worker effect for firefighters is significantly less than that for police, another public safety occupation with similar selection characteristics. Indirectly, this is (weak) evidence for an occupational association for these outcomes, since one might expect the two public safety occupations to be similar except for fire-related risk factors.

Ahn et al. [25] was a very large cohort study of Korean emergency response personnel, who perform firefighting and rescue duties. Subjects were active from 1980 to 2007 and were alive in 1995. Cancer was identified through registration in the national cancer registry, after a lag of 16 years. This study design may be expected to miss some cancers that developed earlier than 1996, including those that were fatal before 1995 and incident cancers resulting from earlier exposures in which the subject died before 1995 of any cause. The study was therefore biased toward an underestimate of risk. Even so, several significant elevations were found (colorectal, kidney, bladder, and non-Hodgkin lymphoma, compared to all Korean men. (A smaller subgroup of responders who did not perform firefighting had unusual characteristics, showing marked elevations in colorectal cancer and in cancers of bone and cartilage.) This first Ahn paper does not make the demographics of the population obvious. However, the Ahn cohort, which is more fully described in the second paper, is still relatively young, most of the emergency responders having joined the service in the 1990s at an age less than 30, and there were only 48 cancers in total. Ahn followed up with a second study of emergency responders in Korea that also reported quite low relative risks for all cancers other than leukemia, which was more than doubled [26]. It should be noted that leukemia is likely to have a much shorter latency period than other occupational cancers, so this may be the only observation on cancer risk in the two papers that is generalizable to other populations.

Daniels et al. [27], preliminary findings for which became available in 2013, is a massive study of cancer mortality and incidence among male firefighters in three cities conducted by the National Institute for Occupational Health and Safety (NIOSH). It is hereafter normally referred to as the "NIOSH Study", as it is regularly among interested colleagues, even though other studies referred to in this report (including Baris et al. [27] and Burnett [14]) have been conducted or supported by NIOSH. The methodology used in the NIOSH Study was more limited and more descriptive than other recent studies, favoring breadth, large numbers (29,993 firefighters), and evidence for or against replication among the three cities (Chicago, Philadelphia, and San Francisco) rather than drilling down for evidence of exposure-response relationships beyond length of service. The Study covered 1950–2009 and average date of entry into the fire service was 1968, which actually covers many older firefighters. Because the NIOSH Study overlapped earlier studies

of firefighters in Philadelphia (which had also been conducted by NIOSH) and San Francisco (which had been conducted by one of the authors on the team), the studies in those cities are not truly independent but the city-specific findings cannot be directly compared with their antecedents. Although the main report is almost entirely limited to cancer outcomes, chronic obstructive pulmonary disease was included in the main report, apparently as an epidemiological indicator for smoking-related health effects. Supplemental data published on-line together with the main report provide information on other non-cancer outcomes.

An important study by Pukkala et al. [28] of over 16,000 male firefighters was published in 2014, yielding more than 2500 incident cancer cases drawn from a defined population of 15 million people participating in a computerized census in each of five Nordic countries, which makes this study smaller but more diverse than the NIOSH Study [28, 29]. The study by Pukkala et al. is a population-based study, in which firefighters are presumed to have self-identified within the population, not a cohort study in which a group of subjects known and documented to be firefighters was followed over time. The design of Pukkala et al. is closer to studies such as Firth [12, 30] and Howe [12] rather than occupational cohort studies such as the NIOSH Study. (The implications of this will be discussed below.) Ascertainment of occupation was based on response to the census questionnaire, most often in the 1970s but without confirmation of continued employment or status. Firefighters who worked a majority of hours in a year were presumed to be "professional" firefighters, although whether this covered paid volunteer firefighters, a crucial point with respect to validity, was not made clear in the paper. (By the criteria reported, a "professional" firefighter status could have meant a person working in that capacity on a volunteer basis half time for a year as easily as a firefighter working full time for half the year or more.) Data on cancer incidence outcomes were derived from each country's cancer registry. Rates were presented for age group and years of service, with some outcomes commented upon by era. No data were available on job assignment, fire service activity, or urban status. Because volunteer firefighters, while poorly studied, can be assumed to have risks closer to the general population, the Nordic Study may (or may not—the methodology does not allow determination or bias) underestimate occupational risk.

Despite methodological issues, Pukkala et al. does describe important findings, such as an elevation in mesothelioma rates and adenocarcinoma of the lung. The study showed statistically significant elevations from all cancer (excepting non-melanoma skin cancer) and in adenocarcinoma of lung (but not squamous cell carcinoma), skin melanoma (mostly in older firefighters), non-melanoma skin cancer (significant in firefighters more than 70 years old), and prostate (mostly in younger firefighters), with elevated risk estimates not achieving conventional statistical significance for mesothelioma and for malignancies of the gall bladder and thyroid. There was substantial consistency by country for both mesothelioma and adenocarcinoma of lung, excepting Iceland. Norwegian firefighters showed a high and significant elevation of mesothelioma (the other Nordic countries showed elevations but wide confidence intervals). Adenocarcinoma of lung showed a similar pattern with the prominent elevation being in Denmark. Prostate cancer was significantly

elevated in Finland and Sweden; Norway also contributed an excess but did not achieve a level of statistical significance. Non-Hodgkin lymphoma was elevated in all countries but Finland, but did not achieve significance. Some elevations were specific to eras: penile cancer (1961–1975) and bladder (1976–1990). Among the cardinal cancers of concern, the results of the Nordic Study, in conclusion, are similar to the NIOSH Study for cancer overall, probably similar for skin melanoma, show a higher incidence of prostate, do not show elevation in buccal and pharyngeal cancer as did the NIOSH Study, and are similarly "negative" for several cancers, such as testes, brain, and non-Hodgkin lymphoma that were elevated in other studies and in the meta-analysis of LeMaster [10].

A prospective mortality and incidence study by Ide [31] in 2014 of cancer among 2200 full-time professional municipal firefighters was conducted in Strathclyde, a mixed residential, industrial, and rural district that includes Glasgow and other urban centers. 19 were female but were excluded from the analysis because of insufficient numbers to conduct a gender-specific analysis. The intent of this study was to focus on a recent 20-year interval, 1984–2005, for the purpose of evaluating contemporary risk, unmixed with historical risk of earlier cohorts. The statistical analysis of the Strathclyde study was not as sophisticated as in other studies but sufficiently robust for the purpose. The Strathclyde study should be considered as a surveillance study conducted on an existing administrative data set and therefore may underestimate risk. It is however representative of what a fire department can achieve with existing data.

In the Strathclyde study, cancer mortality and incidence were compared to the Strathclyde-area general population in the age range 20–54 years of age, however without age-standardization. The Strathclyde region is known in the UK for high prevalence of risk factors for cardiovascular disease [32], which tends to correlate with cancer risk factors related to smoking and diet [33]. The age distribution of firefighters was not precisely known for much of the period and was somewhat skewed by hiring for a force expansion in the mid 1970s. Unusually, the study had smoking information on its population: 33 % of the cohort smoked and 50 % never smoked. Crude cancer rates (unadjusted but compared to the same age group in the reference population), were reported as mortality and incidence; rates for firefighters were below those of the general male population in the same age group for the west of Scotland and Scotland nationally; only 15 cancer types were identified; only those are reported in this chapter. In this chapter, crude rates have been converted to relative risk in comparison with the West of Scotland for comparability with other studies; confidence intervals are not available but $p$ values were provided in the report. There were also gaps in data for 2 years. Notwithstanding these drawbacks, this was a targeted study of generally young firefighters from a particular, well-defined time period, and reports information on age and length of service at the time of diagnosis.

The Australian Firefighters' Health Study was also reported in 2014, although at the time of this writing had not been published. It is included in this book because publication in a peer-reviewed journal is imminent and it has already been extensively peer reviewed prior to release of the final report. This is a national Australian

study of firefighters conducted at Monash University, in Melbourne, under the sponsorship of the Australian Fire and Emergency Services Council, covering six Australian states and territories (the exceptions were South Australia and Tasmania). Uniquely, the study separately identified professional, part-time professional (uncommon elsewhere), and volunteer firefighters. It also straddled 1985 and so was able to address issues of era of employment as well as duration and contained some information on job classification (i.e. ether the firefighter was a trainer). There insufficient numbers to report a geographical breakdown of risk, so the data reported were exclusively for the national cohort, but state-specific death and incidence rates were used for reference. The study yielded 5713 deaths and 8750 incident (individually recognized) cancers among male firefighters; 536 deaths and 1055 incident firefighters were observed among female firefighters, although the numbers were still too small for robust analysis of gender differences. The Australian Firefighters' Health Study is the largest firefighter study conducted to date.

The Australian Firefighter Health Study demonstrated statistically significant elevations for cancer overall among full- and part-time professional firefighters but no site-specific elevations for professional firefighters except for prostate (which has many sources of bias), an elevation in brain among female firefighters, and melanoma (which is more common in Australia). Among volunteer firefighters, there were marked deficits in many causes of death. The explanation for this is almost certainly that Australian firefighters have had a much lower prevalence of lifestyle risk factors for cancer and nonmalignant disease than the Australian general population, particularly with respect to smoking (See Chap. 9). Therefore, there is probably an unusually strong healthy worker effect for cancer (the healthy worker effect being more commonly an explanation for cardiovascular disease and overall mortality). For professional firefighters, the explanation could be (and probably is) that a truly elevated occupational risk is superimposed on a reduced risk from lifestyle compared to the reference group, offsetting the risk estimate and underestimating the occupational risk of firefighting. Support for this interpretation is found in the much lower risk of volunteer firefighters, in which many statistically significant deficits in risk (observed below expected) are reported; volunteers have much less occupational risk than professional firefighters and are therefore an internal comparison. Further support for this interpretation is the very low mortality from cardiovascular disease reported for Australian firefighters of all types, which strongly suggests a robust healthy worker effect related to lifestyle with implications as well for cancer risk, especially related to smoking [33]. Therefore, it is not reasonable to interpret the absence of an elevation in risk in Australian firefighters as evidence of a low occupational risk in absolute terms or adequate personal protection. It is much more likely that an elevation of risk for cancer is simply offset statistically by an unusually strong healthy worker effect. Because of this feature, the Australian Firefighter Health Study will not be cited routinely in the site-specific profiles to follow, unless there is a particular reason.

Brice et al. [34] examined the mortality experience of French professional firefighters who were employed in that country in 1979, with some exclusions related to data access. The study is described as preliminary, with further reports to follow.

Unfortunately, subgroup analysis was lacking in the initial report, which was published in 2015: job assignment (including wildland or municipal firefighting), duration of employment, and location were not separately examined. Overall mortality and mortality from major causes of death (such as cardiovascular disease) were lower than that of the general French population, confirming the presence of a uniquely strong healthy worker effect. Although elevations were observed for some sites (colorectal, pancreas, oral, stomach, liver, and larynx), none were statistically significant. Because of the preliminary nature of the report, it will not be discussed further in this chapter.

Despite the underlying similarity of most studies in design within their class, individual studies vary considerably in evident bias and execution. Knowing the differences among studies helps interpretation greatly. For example, Beaumont et al. [35], while a fine and competently-executed study, is consistently lower in its risk estimates for disease outcomes compared to other studies of firefighters and so may distort attempts to pool data. This is also evident in comparing Beaumont et al. against other studies for colon (but not rectal) cancer. For this particular cause of death, the risk estimate derived from Beaumont et al. is close to 1. If this is an underestimate, in the context of generally lower risks in Beaumont than in other studies, it would have a tendency to dilute the summary risk. Beaumont et al. has a rather high weight among the studies in Lemasters' meta-analysis and the lower bound of the confidence interval for the summary estimate (0.99) is very close to 1. Thus, a study that generally seems to to report low estimates of risk across the board may, by itself, have resulted in the summary risk estimate failing to achieve statistical significance.

Several studies examine a population sample and attempt to determine which occupations are associated with which cancers, usually by matching with death certificates [12] or a disease registry [36]. We have observed that the risk estimates for these studies are usually much below those of occupation-specific studies. An exception to this general trend is Firth 1996 [30], which reports a more than ten-fold elevated risk for cancer of the larynx among firefighters in New Zealand, a finding not replicated in any other study. These studies are prone to misclassification bias, although the best of them, such as Firth, examine both usual and current occupation and registry studies are known to be subject to deficiencies in case ascertainment [23].

Studies of the proportion of occupations represented in registries or death certificates for a given disease are frequently useful. For example, Figgs in 1995 identified firefighters as demonstrating a pattern of cancer risk consistent with other occupations involving exposure to solvent chemicals, a suspected cause of lymphomas [37].

Papers often contain errors and it is instructive to know why. One paper using the proportionate method for occupation has on occasion been used to rebut claims for testicular cancer, Golka et al. [38], contains a significant error. On p. 388, it states that “No relevant studies on occupationally related testicular cancer have been published.” This flat statement ignores the important paper of Bates et al. (2001) [132], published three years before Golka et al., in a widely-available journal. The probable

reason for this omission is that the authors of Golka et al. [38] appear to have had a bias in their search strategy toward retrospective and surveillance studies based on monitoring data and that examine associations with many occupations, rather than studies that begin with the occupation and look for associations, whether prospective or retrospective. Search terms using disease outcome as the primary rubric are, based on experience, much more likely to miss findings relevant to specific occupations than searching on specific occupations. The methodology of population monitoring, with all its opportunities for misclassification and missed cases, is also, based on experience, more likely to yield systematically lower risk estimates than a well-constructed prospective study of a single occupation. Lipworth [39] presents a similar problem.

In theory, both registry and death certificate studies are likely to be biased by the potential for misclassification of occupation, since information on work is obtained only once and may differ from the subject's usual occupation. There is good evidence that the stated current occupation at the time of data collection corresponds well with usual occupation for firefighters, as for other occupations (κ statistic 74.6; 59.6–89.5) in interview studies [40]. Presumably this is also true for cancer registry studies. However, over the longer period of a lifetime current occupation is more likely to change. For death certificate studies there is a possibility, as yet unproven but likely, that misclassification of usual against last occupation could be greater for firefighters than for other occupation because firefighters often retire into second careers. Complicating matters, firefighters are likely to retain their subjective self-image and community identification as a firefighter beyond retirement, as a point of pride.

## Cancer by Site

In time, cancercausation will no longer be assessed on the basis of anatomical location and by trying to make an association on the basis of where the cancer happens to arise and what tissue appears to be its origin. Eventually, causation will be reported based on genomic evidence and associated with a much smaller number of indicators that reflect specific tumor biology, such as oncogene activation, p53 gene mutation, epigenetic profile and other characteristics. It will take more time to work out the essential genomic characteristics and biomarkers for forensic purposes, and then undoubtedly further time will elapse before a precedent set in the adjudication of some precedent-setting case forces a rethinking of cancer classification for purposes of compensation. Until that happens, the state of the art will continue to be to assess causation of cancers of a certain tissue type at a certain location in the body based on the known risk factors for that cancer type at that location.

There is little empirical information on cancer risk for female firefighters because of low participation in the firefighting workforce. Therefore unless specified otherwise, the observations noted are for male firefighters only.

## *Genitourinary Cancers*

The kidney and structures of the urinary tract are vulnerable to carcinogens that are filtered or excreted into urine.

Genitourinary cancers represent the conventional situation, in which individual diseases are more or less satisfactorily classified and risk estimates probably do reflect the experience of the group for the individual cancers. The various tissue types (urothelial in bladder and kidney pelvis, clear cell and chromophobe in kidney, the latter two sometimes found in the same tumor) are known to be induced by cigarette smoke and so an influence of fire smoke is highly plausible. Perhaps for this reason there seems to have been more widespread acceptance of claims by firefighters in this class than for other outcomes. The data are also relatively easy to interpret by tumor site [3].

### Bladder Cancer

Bladder cancer is a urothelial malignancy, meaning that it arises from the lining of the organ, called the transitional epithelium. There is evidence that this tissue is unusually prone to malignant transformation from a variety of chemicals, some of them now withdrawn from commerce because of the risk, and it is also elevated in the chemical industry (historically), after cancer chemotherapy, with radiation, after exposure to arsenic, among cigarette smokers, and in other occupations in which exposure to PAHs occurs. Even so, it is an uncommon cancer, ranking sixth in incidence among men in North America.

The overall pattern for bladdercancer is that individual studies may not show an excess but the literature as a whole shows evidence for an elevation. The meta-analysis from LeMasters (2006) [10] shows a summary risk estimate of 1.20 (0.97–1.46); in the LeMasters' evaluation, this suggestive finding was downgraded due to heterogeneity but internal evidence in the studies was not analyzed for evidence of an exposure-response relationship.

The evaluation of LeMasters was influenced by a number of studies that did not show an elevation overall, but several of these showed evidence for an elevation in subgroups or on replication. Burnett et al. [14] found no elevation in mortality for bladder cancer. The PMR was 101 for firefighters dying under the age of 65 and 99 for those dying at or over the age of 65. With 9 and 37 deaths, respectively, this is a large collection of deaths by bladder cancer. Using the same database, Ma et al. [16] reported that a not-quite statistically significant elevation of 1.2 was observed for bladder cancer among white firefighters and an elevation (but based on a single case) for African-American firefighters.

Improving the exposure assessment and examining subgroups experiencing higher exposure increases the mortality risk estimate in Baris et al. [19], which reported a slightly elevated SMR of 1.25 for bladdercancer, with greatest risk being among those hired before 1935 (RR 1.71; 0.94, 3.08), and among those with greater

number of runs during their first 5 years employed (RR 2.59; 0.64–9.84). This strongly suggests an exposure-response relationship or the reduced effect of confounding factors and should be considered evidence supporting a presumption on the basis of trend, whether or not individual comparisons achieve statistical significance.

Gaertner et al. [41] conducted a case-referent study of occupational risk factors for bladder cancer in Canada for the Medical and Occupational Disease Policy Branch of the Workplace Safety and Insurance Board of Ontario. Cases (887, a large number) in seven provinces were newly identified from 1994 to 1997 and referents were surveyed in 1996. Firefighters were identified as an occupation with an elevated risk estimate but the elevation was not statistically significant. It should be noted that population-based cancer risk studies are usually ineffective in identifying known elevated risks for individual occupations, unless they are very common and the association is very strong. That this association was identified in a study of this design outweighs the statistical inference test with respect to weight of evidence.

Ma et al. [21, 42, 43] demonstrated a significantly increased risk for bladder cancer among both male and female firefighters in Florida (male: SIR 1.29; 1.01, 1.62; female: 10.00, 0.13–55.60, based on a single case). Kang [23], with much smaller numbers, demonstrated a similar but not significantly elevated risk when firefighters were compared to police (SMOR 1.22; 0.89–1.69) and to a referent population (SMOR 0.93; 0.93–1.52). The contribution of the Kang data is to show consistency, in that even studies that do not show significantly elevated risk and that are likely to be underpowered for this outcome consistently demonstrate some elevation.

Ahn et al. [25] demonstrated an overall elevated risk of incidence for cancers of the urinary tract among Korean emergency responders, who serve multiple roles but are engaged in active firefighting. This large study based on the national cancer registry, which as noted seems likely to be biased toward an underestimate, demonstrated an elevated standardized incidence ratio for bladder cancer (SIR 1.77; 1.08–2.73).

The NIOSH Study [27] demonstrated no elevation in mortality for bladder overall (0.99; 0.79–1.22) or in any of the cities, but a borderline significant elevation for incidence overall (SIR 1.12; 1.00–1.25), a higher elevation for first cancer (1.1.8; 1.05–1.33 although the isolated but highly relevant finding of incidence among Caucasian men came close to significance (SIR 1.11; 0.99–1.24). However, no relationship to duration of employment was observed in the NIOSH Study.

Bladder cancer risk was only slightly and not significantly increased in the Nordic Study of Pukkala et al. [28] et al. overall (1.11; 0.96–1.28); no subgroup analysis was provided.

The study of Strathclyde firefighters showed no elevation in incidence (RR 0.92, based on two cases) compared to residents of the West of Scotland and Scotland [31]. The mean age at time of diagnosis for firefighters was 52 and the length of service was 27 years.

Most population monitoring studies of cancer and occupation tend to underestimate the risk relative to occupation-specific studies, which probably reflects misclassification and incomplete case ascertainment. Thus, it is of interest that one

Canadian study [41] of incident cancer shows an elevation in risk for firefighters (SIR 1.51; 0.59–3.84) although it failed to achieve statistical significance, as [44] did smoking, exposure to hair dyes, and consumption of fried foods, a novel observation for this cancer site. McGregor, reviewing the data for the occupational health institute of Québec (Institut de recherche Robert-Sauvé en santé et en sécurité du travail, called IRSST) in 2005, concluded that an increased risk of bladder cancer among firefighters is plausible, that the direction of the evidence is consistent, and that individual factors must be taken into consideration [45]. However, given the presence of exposures known to cause bladder cancer and strong evidence of an elevation in the literature that approaches the criterion for a presumption, the preponderance of evidence favors causation and sufficient weight to derive a presumption.

For bladder cancers, latencies are usually measured in decades but under conditions of intense exposure to potent carcinogens tend to be shorter and more variable than for other solid tumors. Aniline dye workers in the 1940s and 1950s showed a latency as short as 7 years, presumably due to high, constant exposure which may have compressed the latency period to its absolute minimum. This is not plausible for firefighters. The exposure of firefighters to potential bladder carcinogens is much less than for chemical workers in the 1920s. In Alberta bladder cancer did not appear before age 60 or before 20 years of service and showed a very long peak latency of 40 years [44].

On the basis of the totality of evidence, not any one study, and bearing in mind that studies are much more likely to be falsely negative than positive, it seems clear that the risk of cancer of the kidney is elevated, satisfying the criteria of weight of evidence, to a level that would merit presumption based on empirical evidence by the methodology and reasoning already outlined in Chap. 4 and elsewhere [3].

It would be difficult to accept latency under 10 years for bladdercancer in a firefighter but the literature from other occupations does not rule out latencies under 20 years. The extremely short latencies seen historically in the analine dye industry do not apply to the level of exposure likely in firefighting but demonstrate that bladder cancer can show higher variability in latency than other solid cancers. One might expect that the duration of service associated with risk among firefighters to be on the order of 15 years. Youakim determined in his meta-analysis that firefighters with more than 40 years of service had the highest observed risk [11].

### Kidney Cancer

Cancer of the kidney has become widely accepted as associated with firefighting by conventional criteria [11].

Cancer of the kidney is predominantly of the form known as renal cell or clear cell carcinoma, which may or may not include sarcomatous elements which confer a worse prognosis. However, 7 % of cancers of the kidney are transitional cell “urothelial” carcinomas, which arise from the renal pelvis (the funnel-like collecting

system) and are similar to most cancers of the ureter and bladder (collectively often called urothelial cancers).

Other forms of cancer arising in the kidney are rare as the predominant cell type but are often found embedded in a clear cell carcinoma as part of its variability. The reason this line of argument is important is that on occasion it has been argued that because the ICD-9 code for kidney cancers (189) is not exclusive for renal cell cancers, a case in which the predominant cell type was urothelial should be treated differently and the epidemiological evidence may be biased by the inclusion of transitional cell. This small proportion is unlikely to bias epidemiological studies significantly, even if there were a difference in risk of urothelial cancers between firefighters and a comparison population. Since urothelial cancers are associated with similar risk factors to renal cell carcinomas with respect to cigarette smoking (and therefore the chemical constituents of cigarette smoke which resemble those of firefighting), one would expect transitional cell carcinomas to have an elevated risk similar if not identical to to bladder cancer and to be elevated together with renal cell cancers, although this would be hard to detect in most epidemiological studies. Given the evidence for an elevation in risk of bladder cancer, there would also be an increased risk of urothelial cancer. In summary, the inclusion of transitional cell cancers of the renal pelvis in the ICD code for kidney cancer is not a serious objection to the evaluation of risk.

Burnett et al. [14] found a marked elevation in mortality for cancer of the kidney. The PMR was 141 for firefighters dying under the age of 65 and 144 for those dying at or over the age of 65. With 24 and 53 deaths, respectively, this is a large collection of deaths by kidney cancer. Using the same database, Ma et al. [16, 21, 42] reported a borderline statistically significant elevation of 1.3 for cancer of the kidney among white firefighters.

An exceptionally strong case-referent study in New Zealand [46] examining occupational associations of renal cell cancer cases demonstrated a highly elevated and highly significant relative risk for firefighters (OR 4.89; 2.47–8.93).

In data from Alberta, a very high elevation in mortality risk for kidney cancer was visible (4.14; 166, 853) overall, in both of the two cities studies, and in association with duration of employment and with exposure-adjusted duration of employment [44].

Baris and co-workers reported a doubling of risk for mortality with an RR 2.20; 1.18, 4.08 among those employed for 20 or more years [19].

Ma et al. found no elevated risk of incidence for kidney cancer among male firefighters in Florida but a high elevation (although based on a single case) among female firefighters (SIR 4.17; 0.05, 23.18) [21, 43].

Kang, in a more limited sample from Massachusetts, also showed an elevation in incidence but much smaller and not statistically significant, and higher as compared to police (SMOR 1.34; 0.90–2.01) but not to a referent population (SMOR 1.01; 0.74–1.38) [23].

Ahn et al. demonstrated an overall elevated risk for incidence for kidney cancers among Korean emergency responders, who serve multiple roles but are engaged in active firefighting [25]. This large study based on the national cancer registry,

which as noted seems likely to be biased toward an underestimate, demonstrated an elevated standardized incidence ratio for kidney cancer (SIR 1.59; 1.00, 2.41).

The NIOSH Study showed a statistically significantly elevated risks for kidney cancer overall (SMR 1.29; 1.05–1.58, SIR 1.27; 1.09–1.48) and in incidence in Chicago (1.62; 1.23–2.11) and among Caucasian men (SMR 1.31; 1.05–1.60) [27]. The risk was concentrated in younger, working-age groups (17–64 years: 1.41; 1.12–1.76), falling off after retirement age (65–85+ years: 1.17; 0.94–1.44), when there are many competing causes of mortality. There is also a suggestive stepwise increase in risk across three decades of employment duration, ending before the last category (30+ years). Taken together, this is strong evidence for an elevation.

The Nordic Study [28], on the other hand, showed no elevation in incidence overall. No subgroup analysis was presented, however. The Australian Firefighters' Health Study showed no elevation in kidney cancer for full-time but a non-significant elevated risk for part-time professional firefighters (1.34; 0.81–2.10).

The Ide [31] study of Strathclyde firefighters was highly significantly elevated ($p < 0.001$, based on 4 cases) at relative risks of 3.42 for mortality and 2.07 for incidence compared to residents of the West of Scotland and similar elevations compared to Scotland as a whole.

The Australian Firefighters' Health Study [47] did not show an elevated risk for cancer of the kidney among full-time firefighters (0.97; 0.67–1.36); even so, on close examination the study is not strong evidence against an association with firefighting and the results may be considered indirectly supportive. In the subgroup analysis, there is also an unusual, although non-significant, elevation for part-time firefighters (1.34; 0.81–2.10). More persuasively, there is clear exposure-response trend within full-time firefighters: the risk of kidney cancer was statistically significantly elevated for firefighters with at least 20 years service compared to those with less than 10 years service (omitting those with 3 months employment or less) and the risk of firefighters with 10–20 years service was intermediate. Although the relative risk for none of the groups was elevated compared to the reference population, the exposure-response trend was statistically significant. In addition to chemical carcinogens derived from combustion, the risk of kidney cancer is also affected by a number of lifestyle factors (obesity, hypertension, diabetes, medications). This finding therefore supports the idea that firefighters have an elevated risk arising from occupation against a background of lower risk compared to the general population.

On the basis of the totality of evidence, not any one study, and bearing in mind that studies are much more likely to be falsely negative than positive, it seems clear that the risk of cancer of the kidney is elevated, satisfying the criteria of weight of evidence to a level that would merit presumption based on empirical evidence by the methodology and reasoning already outlined in Chap. 4 and elsewhere [3].

It is not clear that latency in kidneycancer follows the same pattern as bladder cancer. Latency has not been as intensively studied for kidney cancer. On the basis of current understanding and the literature on firefighters, it might be difficult to accept an expired time since first exposure of under 15 years, just on the basis of the time

required for a solid tumor to proliferate. In his meta-analysis, Youakim found that firefighters with more than 30 years of exposure had the highest risk of mortality [11].

Certain references on renal cell cancer that have been cited occasionally in the past as refuting an association require clarification. In particular, studies that examine occupation and cancer risk on a population basis using surveillance data usually underestimate the association between the two which is revealed by cohort and case-referent study designs.

Lipworth [39], in an extensive review of occupational associations of renal cell cancer, did not address firefighting. Contrary to its conclusion, which is that occupation in general is not strongly associated with renal cell cancer, other studies published about the same time (including Zhang [48]) identified an elevated risk of renal cell cancer for several occupations (firefighters were not studied), and concluded, emphatically, "…occupational exposures may increase the risk of renal cell carcinoma." Moyad [49] was clearer when he wrote "…numerous occupations, occupational exposures, reproductive and hormonal changes or manipulations, and a variety of other factors may impact risk, but *overall* their contribution seems small compared with other more consistent risk factors." [Italics added.] Moyad is saying quite clearly in context that obesity and hypertension are major risk factors driving rates of renal cell carcinoma overall in the American population but that for individuals, other factors are significant drivers, among them occupation.

Given the presence of exposures known to cause kidneycancer and strong evidence of an elevation in the literature that approaches the criterion for a presumption, the preponderance of evidence favors causation and sufficient weight to derive a presumption.

Youakim determined in his meta-analysis that firefighters with more than 40 years of service had the highest observed risk [11].

### Testicular Cancer

IARC already recognizes an association between testicular cancer and occupation as a firefighter [7]. The Lemasters metanalysis found a summary risk estimate of 2.02 (1.30–3.13), the highest found in the meta-analysis [10].

Aronson/L'Abbe (1992) found a high elevation in mortality from testicular cancer (SMR 246; CI not reported) but with only three cases [50]. Bates et al. [1] found an odds ratio of 3.0 (1.3–5.90) for testicular cancer among firefighters in the New Zealand capital city of Wellington [18].

Stang et al. reported very similar findings in a case-referent study from northern Germany, although their odds ratio of 4.3 (0.7–30.5) was not statistically significant [51]. Such high risks are unlikely to be confounded by differences in the prevalence of cryptorchism (the major known risk factor), smoking (not known to be associated with testicular carcinoma) or other plausible alternative risk factors. In their community-based study of testicular carcinoma, only four firefighters and three controls were firefighters out of 269 and 797, respectively, making the power of their study very limited. Stang et al. also reported on duration of employment. Of

the four cases, two had been employed as firefighters more than 20 years and two for less than 4. Bates [22] then demonstrated a statistically significant elevation for testicular cancer among California firefighters (OR 1.54; 1.18–2.02).

Neither the NIOSH Study nor the Nordic Study [27, 28] demonstrated an elevation for testicular cancer. Ide [31], however, reported a small non-significant elevation in incidence (RR 1.2, based on four cases) for Strathclyde firefighters; there were no deaths. The Australian Firefighters' Health Study showed a much larger elevation closely approaching statistical significance (1.44; 0.98–2.05) [47].

Testicular cancer had not been considered in earlier studies and so an excess may easily have been hidden in aggregate figures reported for genitourinary cancers. There are five basic tissue types of testicular cancer, the most common by far being seminoma (about 95 %). Bates et al. [132] does not specify the histology of the tumors. Stang et al. reported that of the four in their study, two were embryomas (a cell type also found in mixed germ cell types), an unusually high frequency which suggests, but does not prove, that this type may be uniquely associated with occupational risk. It is not possible to sort this out with only the data currently available.

Given the totality of the evidence and the high relative risks observed in the studied that found an association, it is reasonable to establish a presumption for testicular carcinoma on the basis of current evidence, and this conclusion is consistent with the 2010 finding of IARC that testicular cancer is associated with firefighting [7]. However, given the methodological limitations of Bates et al. [132] and the lack of available evidence on exposure, tissue type of the tumors and latency, no further guidance can be recommended.

## Prostate Cancer

The question of prostate cancer is exceptionally difficult and controversial. This subsection does not represent a view universally shared in the firefighting community. It represents an interpretation with which reasonable experts may differ.

In LeMasters et al., the summary risk estimate was 1.28 (1.15–1.43), which the authors considered a major finding of the study. None of the individual studies they entered into the meta-analysis showed a risk even approaching that magnitude, for reasons that are unclear because higher numbers among older men should be much less affected by power limitations than any other cancer.

### Evidentiary Base

In general, with the exception of Giles et al. [13] and Demers et al. [52], studies conducted of firefighters before 1990 show no apparent elevation in frequency of prostate cancer. On the other hand, the elevation in incidence seen in Giles is quite high, and occurred in Australia, which had a national health service by 1980, making this study the most significant anomaly in the literature for prostate [13].

The timing of the increase in prostate cancer reported among firefighters therefore seems to match the widespread introduction of wellness and health promotion programs for firefighters, which place emphasis on screening for the disease.

IARC (IARC Monograph No. 98, 2007) [53] recognizes two studies that show an association between prostate cancer and occupation as a firefighter, Krstev (2008) [54] at a relative risk higher than 3, which is the only study to observe a risk of that high a magnitude, and Bates, with a relative risk about 1.2, which is more consistent with the world literature [22]. Many studies reviewed by IARC showed no elevation. The IARC monograph did not recognize firefighting as an occupation associated with prostate cancer. It only summarized the evidence. Another section of the document, on shiftwork, did imply that a wide range of cancers was associated with changing work shifts, which is characteristic of firefighting, but the connection to firefighting or to prostate cancer was not explicitly recognized. The polycyclic aromatic hydrocarbons have long been recognized by IARC as a group as a Category 1 carcinogen, but not specifically as a chemical risk for prostate cancer.

Demers et al. found that the observed elevation in mortality (relative risk 1.4) in prostate cancer demonstrated in his population of Washington state-based urban firefighters (1994) was much reduced when compared to police officers, rather than the general population [52]. Police are a group of municipal employees with similar benefits and comparable physical requirements to firefighters but without the same intense exposure to the characteristic carcinogens associated with firefighting.

Krstev et al. (1998) showed remarkably high elevations for prostate cancer in both white and African-American firefighters (4.75, 2.64, respectively) in a synthetic, combined group of firefighters from Atlanta, Detroit, and New Jersey [54]. The study featured small numbers of cases despite the large population of firefighters, and this suggests, but does not prove, a bias in case ascertainment. It is the clear outlier because of the high magnitude of the risk estimate.

Ma et al. [43] found elevations in prostate cancer incidence in both white and African-American firefighters but the elevation was small (mortality odds ratio 1.2) [16]. The frequency of prostate cancer is elevated in African Americans in the general US population, as is mortality from the disease, a situation which persists [55]. The equalization of relative rates among firefighters implies, but does not prove, that access to healthcare and screening is responsible for at least some of the disparity.

In a large cohort study of working adults in the Netherlands who were monitored for prostatecancer, firefighters had a *lower* incidence rate than the reference population [56]. However, the study was obviously underpowered and because of its unusual design features, bias was difficult to interpret. Zeegers et al. then conducted a nested case-referent study within the cohort, using as cases the subjects who developed prostate cancer. Because of this study design, they used a 99 % confidence interval rather than the usual 95 %, making this study even more underpowered than usual for the detection of infrequent outcomes in individual occupations. They found moderate reductions, not elevations, in risk among firefighters in their cohort. Other occupations showed substantial elevations. Police showed a very high (4.00) and statistically significant elevation in risk, which was

a new finding which has not since been replicated in other studies of police [57]. Rubber workers have been known in the past to have elevated rates of prostate cancer in other studies. In this cohort, they demonstrated a very high RR 4.18 and yet it did not achieve statistical significance. This suggests that although the Zeegers et al. study was large, the power to detect an excess in any one occupation was low. This study, for all its complications, lends little or no support for an association with prostate cancer. However, it is also an example of how population-based studies generally yield lower risk estimates than cohort studies for the same occupation and outcome.

Bates et al. (2007) demonstrated a statistically significant elevation in incidence for prostate cancer among California firefighters (OR 1.22; 1.12–1.33) [22].

A persuasive study that illustrates the complexity of the issue is that by Ahn et al. on Korean fire-rescue personnel [25]. They showed an elevated overall relative risk in incidence (1.60) but when divided into personnel with firefighting duties and those without the predominant risk elevation was among the non-firefighters (1.32 and 6.01, respectively) who were mostly administrative personnel working the same shifts, all calculated relative to the general Korean male population. These findings are contrary to what would be expected if firefighting were the operative risk factor but consistent with an employment effect and access to screening services and were observed for many other cancers. It was demonstrated in the paper that Korean fire-rescue personnel in general have much better health status than the general population, which may imply better health care and access to better screening services for a longer period. (Korea instituted mandatory national health insurance in 1977 but it did not cover all of the general population until 1989. Government employees, including fire-rescue personnel, joined the program in 1979.) The findings of this study, on balance, do not support an increased risk of prostatecancer for firefighters despite the appearance of a somewhat elevated risk estimate.

The NIOSH Study found no elevations (risk estimates very close to 1) except among non-Caucasians for both mortality (SMR1.64; 0.95–2.63) and significantly for incidence (1.26; 1.02–1.54) [27]. This is consistent with known racial differences in mortality and might also reflect disparity in health care among minorities. There was no association with employment duration.

Pukkala et al. [28] found a modest excess in prostate cancer incidence overall (SIR 1.12; 1.05, 1.22) but a marked excess of prostate cancer in the age group 30–49 years (2.59; 1.34–4.52), much less and not statistically significant in older age groups, over age 50. The authors suggested that this might be due to a screening effect. However, they also suggested that a shiftwork effect might be responsible, if the elevation reflected a biological elevation in risk. The findings of Pukkala et al. are consistent with the possibility that an elevated risk of prostate cancer exists in the fire service for younger firefighters, before becoming overwhelmed by the risks associated with aging.

There are some inconsistencies in the authors' interpretation of their findings in the Nordic Study for prostate cancer. Pukkala et al. overstated the magnitude of the findings overall in Daniels et al., to which they were comparing their own findings. In an earlier abstract of a presentation on the Nordic Study [29] the authors concluded

with respect to the totality of their study that "The most common cancer among men in the present cohort was prostate cancer (339,973 cases). Despite the huge number of cases, we were unable to demonstrate any occupation-related risks. The observed small occupational variation could be easily explained by varying PSA test frequency." [29] The absence of any occupational association would, of course, include firefighting, so there must have been a reinterpretation or revision following the initial analysis.

Ide [31] did not report prostate cancer for Strathclyde firefighters, probably because his cohort was relatively young.

The Australian Firefighters' Health Study [47] concluded that elevation in the incidence of prostate cancer was a major finding of their study. However, mortality from prostate cancer was not elevated, which might be expected if the overall incidence were increased. Most prostate cancers are either curable or not sufficiently aggressive to be lethal within the life of the patient, but some are not and one might expect cancers induced by chemical carcinogenesis to be more likely to be invasive. On the other hand, the elevation in incidence of prostate cancer risk for professional, part-time, and, uniquely in the study, volunteer male firefighters, demonstrated remarkably similar and uniformly statistically significant elevated risk levels (full-time professional: 1.23; 1,10–137; part-time professional: 1.51; 1.28–1.77; volunteer: 1.12; 1.08–1.16). This degree of consistency appeared without a monotonic exposure-response relationship despite the obvious differences exposure levels of the three categories. This raises the inevitable question of whether it is status as a firefighter that is associated with identification of prostate cancer rather than occupational exposure.

Evidence for an association between occupational and environmental exposures and prostatecancer has been much searched for but elusive, except for a consistent association with farming. Although an association has been found or suspected in some studies of firefighters, usually with low risk estimates, the totality of evidence does not support a presumptionfor firefighting.

## Potential Screening Bias

Prostate cancer is virtually a normal disease of aging for men, with a progressive incidence with age so steep that the cancer burden would be nearly universal if men lived long enough. Although some cases can be devastating, and aggressive prostate cancer has touched many lives, many and probably most prostate cancers do not cause death or even inconvenience during life. This is because most of them are "indolent" or latent", meaning that they grow slowly and are not aggressive [58, 59].

Most men never know that they have prostate cancer and never would without screening, because only a small fraction of prostate cancers behaves aggressively enough to cause symptoms and threaten life. Even many invasive prostate cancers are missed entirely during life although they can be demonstrated at autopsy [60–62]. The rest are "indolent", meaning that they grow slowly and do not invade, and so would not otherwise be detected during a man's lifetime. The result is a "reservoir"

(the term used by some investigators) of previously undetected cases that can be found if one looks hard enough.

Prostate cancer is often found incidental to an examination or evaluation, and common forms of it are not fatal or highly progressive when its onset is late in life. For these reasons, it is heavily under-diagnosed. Indolent prostate cancer is a common and virtually inevitable disease of aging men, such that elevations in risk are not really indicative of lifetime incidence. In many, possibly most, cases, prostate cancers have no effect on longevity or symptoms and do not warrant investigation to detect it, in the absence of screening. If screening were not undertaken, the disease would never be detected at all. Autopsies demonstrate a much higher rate of prostate cancers, mostly of the indolent variety, than detection during life. Most aged men do not have autopsies. If they did, the reported rate of prostate cancer in the general male population would be much higher and more accurately reflect the true prevalence.

Participation in screening programs is now driving reported trends in prostate-cancer. The frequency of recognition of prostate cancer in the general population has increased in recent years but mortality from the disease has not. Most observers think that this is because of improved recognition and diagnosis rather than a true increase in incidence. In particular, improved and more intensive and in some cases mandatory screening programs which include tests for detection of prostate cancer may explain all of the increase. When members of a group adhere to (comply with) a screening program for prostate cancer, these previously undetected cancers are identified and counted as cases, although they were there in the population all along unnoticed. Firefighters today participate in intensive wellness and screening programs, often mandatory, and so this so-called "overdetection" of prostate cancer is to be expected.

Comparing a group such as firefighters that adheres closely to screening programs to a reference population that generally does not (such as the general male population) will predictably result in an elevation in reported risk that does not reflect the true incidence of prostate cancer. This is called "detection bias" in epidemiology. The group that adheres more closely to screening programs will have many more observed cases of indolent or latent cancer, but probably will experience the same rate (given statistical uncertainty) of the more significant aggressive cancers as the reference population. The result will be an apparent elevation that is driven by detection bias rather than a true difference in rates. Screening is known to result in a larger yield of cases of prostate cancer than would be expected from the general population. It identifies mostly indolent cases that would not otherwise cause death or disability [16].

The magnitude of this "overdetection" (detection of tumors but without public health benefit) is reported to be approximately 30 % or 40 %, similar to the reported increase observed among firefighters. The rapid and widespread adoption of screening for prostate cancer (especially using "prostate-specific antigen", PSA) after 1987 has been associated with a large increase in reported prevalence but no change in mortality. It later became obvious that elevated PSA could occur in many situations other than invasive cancer and that the test was more sensitive than specific for

prostate cancer and that most prostate cancers that were being detected were not invasive. The additional cancers that are detected by aggressive screening are mostly indolent (which is not to say that they are "benign"—they are true cancers but only growing much too slowly to cause problems clinically). The more screening that is performed, the more these indolent cancers are detected and counted in cancer incidence studies but they were there all along. The relative risk in the LeMasters study was 1.28, for an attributable risk of 22 %, which would be quite consistent with the magnitude of screening bias [10].

The apparent elevation in prostate cancer frequency among firefighters does appear to have predated the introduction of the PSA test in 1987. Studies conducted prior to the late 1990s may or may not pick up a screening effect because the screening method used prior to PSA, digital palpation in the rectum, is partially effective. Wellness and screening programs for firefighters were already common at that time, although by no means universal in fire departmental. Professional firefighters in the US operate under contracts with negotiated benefits that provide access to good quality health care, comprehensive insurance that supports testing, health facilities that encourage it, and, especially, high participation rates in programs that promote it or may even require it. For example, the Dallas Fire Rescue Department is one of many that have established a wellness program in which regular screening for prostate cancer is conducted [63]. Not surprisingly, more cases seem to be observed in such populations. Digital palpation was (and often still is) deferred in a routine examination because it is unpleasant and unacceptable to some subjects. Provisions of the service by clinicians are known to have varied greatly in that period [64, 65]. There was ample opportunity for differences in adherence to have arisen between a motivated occupational group and the general population and between committed providers following a fire department protocol and unimpressed community health providers in screening adherence, even before the PSA test became widespread.

There is another reason to suspect that the elevation in reported prostate cancer incidence may be due to detection bias. In studies conducted the United States, the difference in incidence and mortality from prostate cancer between firefighters and their reference populations tends to be greater than in other countries in studies from the 1980s and 1990s, although not greatly. The difference is much more pronounced, however, between white firefighters and black firefighters, compared to race-specific reference rates. This suggests that the insurance and healthcare benefits associated with firefighting in that era played a role, since firefighting provided health benefits and minority populations were disproportionately disadvantaged by lacking health insurance attached to good jobs. In Canada, where residents during that period already enjoy equitable access to healthcare and where there is less difference than in the US between benefit plans for firefighters and access to care by the general population, no excess of prostate cancer was found among firefighters in Northeastern Ontario compared to the general male population among residents older than 50 [66]. These observations suggest that prostate cancer detection in this population was biased by access to health insurance as well as adherence to screening.

Interpreting studies of an occupation that characteristically has excellent health coverage and benefits, such as firefighters, screeningbias becomes very important.

Prostate cancer screening is widely practiced in the benefits plans for firefighters and has been heavily promoted as part of routine health screening, as in the many programs available to firefighters. Therefore one would expect that a heavily screened population would show an apparent, but not real, excess when the general population is used as the reference population. This differential may change as health insurance in the US becomes more broadly available.

### Potential Confounding Exposures

For the most part, prostatecancer is not closely or consistently associated with known carcinogens, not even cigarette smoking. Toxicological investigations and studies of populations intensely exposed to certain agents to which firefighters are exposed (PAHs and diesel exhaust, specifically) do show an elevation in risk of prostate cancer. Various individual studies have suggested an association of prostate cancer with exposure to cadmium, cutting oils, diesel fuel and fumes, herbicides, polycyclic aromatic hydrocarbons (PAH), polychlorinated biphenyls, soot, tar, mineral oil, and solvents. The more focused of these studies have yielded inconsistent results with diesel emissions as the most plausible association observed to date, attaining a remarkably high risk estimate of 3.7 in one study from Germany [67]. Firefighters are exposed to diesel emissions, and so it is plausible that there may be a contribution to risk in individual cases (for example, a young firefighter with exceptionally intense exposure to diesel exhaust), but this is not sufficient evidence to conclude general causation.

If PAHs, which are the predominant combustion-related exposure associated with firefighting, is a major risk factor for prostate cancer as it is for other cancers, one would expect the risk to be closely associated with cigarette smoking, which is the major source of highly intense exposure to PAHs in smoking adults. However, the relationship of prostate cancer with cigarette smoking is weak [68].

The conclusion to be drawn from these studies is that it is possible to make an individual case but the evidence is not yet sufficient and not yet demonstrated to a balance of probabilities that, in cases of intense exposure to PAHs, the risk of prostate cancer may be elevated among individual firefighters. The risk of incident prostate cancer over a lifetime for men is already so high that it depends more on longevity than occupation as a firefighter.

### Weight of Evidence

Prostate cancer, despite a number of studies that appear to suggest an excess, is an example of a diagnosis that does not fit the logical framework required for a presumption. On the face of it, the evidence would seem to suggest a rather weak association with some toxicological plausibility. Detailed examination of the problem, however, suggests that the association is the result of screeningbias, which would be exceptionally strong in this case. On the other hand, the totality of

evidence suggests that there may be an elevated risk for younger firefighters but that the contribution of risk from firefighting is not visible against the much higher background risk of the disease in older age groups.

Still, there may be an association between intense exposure and risk of prostate-cancer in individual cases, although not necessarily in general causation or sufficient to support a presumption. Taken together, the literature on prostate cancer could be construed as suggesting an association but one that falls well below a balance of probabilities for firefighters as a whole. However, a young age at detection or intensity of exposure may be a marker for an individual case in which occupation may play a role. There may be an association between prostate cancer and exposure to PAHs, arenes, and possibly other products of combustion, that contribute risk in individual cases in which exposure is exceptionally high. It may therefore be reasonable to argue specific causation in an individual case with these characteristics.

Taken as a whole, the evidence suggests that if there is an association and the occupation of firefighting mediated by exposure to combustion products and possibly diesel exhaust, it would have to be demonstrated in the individual case. However, the characteristics of prostate cancer and the high incidence of the indolent form among aging men do not support a conclusion of general causation for firefighters as a group.

## *Brain*

Youakim [11] and LeMasters et al. [10] both concluded that there were elevations in risk for cancer of the brain and associated intracranial structures among firefighters, using conventional criteria for meta-analysis. In LeMasters et al., the summary risk estimate was 1.32 (1.12–1.54), but this was later downgraded as a major finding of the study because of the algorithm used in the study, which by design discounted associations when heterogeneity was demonstrated among studies.

Cancers arising from brain tissue are relatively rare and may include twenty or more individual types by tissue and site of origin. Each type may or may not be a different disease, with its own risk factors. Epidemiological studies do not distinguish among them because they are individually rare, subject to miscoding and are aggregated into a more general ICD code when they are reported.

The risk of "brain cancer" as an aggregated category is increased in many studies but this risk is probably diluted by inclusion of cancers that are not associated with environmental or occupational factors (and, in the past, meningioma, which although intracranial is not a cancer of brain tissue itself). This leads to an inherent bias to underestimate the risk for that subset of cancers that may have a true association with firefighting. Analysis by specific tumor type might identify which, if any, is associated with the risk but because these cancers are uncommon such a study would require large populations and is unlikely to be undertaken anytime soon [3].

The most common type of brain cancer is glioma (a malignant glioma at stage IV is called glioblastoma multiforme, and has a poor prognosis) but this type only

constitutes about half of the total. Although studies of their etiologic associations have been inconsistent (and much less consistent than for firefighters) [69–79], gliomas (astrocytomas) are much more likely to be associated with environmental and occupational exposures than other types of brain tumor, and appear to be most consistently associated with occupations involving solvent chemicals [80], many of which are also present as constituents in fire smoke.

McGregor noted that there "is a tendency for risk of brain cancer to be higher than expected in firemen across the majority (10) of the 16 publications considered" but stated there are many uncertainties and that biological plausibility was lacking [81]. However, McGregor was basing his conclusion on the standard of scientific certainty, is not the standard of adjudication which is weight of evidence. The basis for his conclusion regarding plausibility is not clear, because the astrocyte, the cell of origin of gliomas, is metabolically active, involved in transport, and lipid-rich, all characteristics that would seem to favor chemical carcinogenesis (See Chap. 5).

A different approach is required to determine occupational risk within this category of tumors, inferring risk for the predominant type from the combined risk for the group. One can expect that the magnitude of elevated risk for glioma will be diluted by aggregation with non-glioma brain tumors. Therefore any consistent elevation in the rubric as a whole is likely to be an indicator of elevated risk for glioma but the magnitude will be attenuated by dilution. Further complicating analysis, statistical power is usually limited even in large cohort studies because brain is an uncommon tumor site.

Firefighters in the US Pacific Northwest active in the 1980s showed a doubling of risk of mortality (SMR% 257) at less than ten years of employment, peaking at over a tripling (353) up to 19 years [82]. Heyer et al. also showed a near-doubling of mortality risk (184) at less than 15 years duration of exposure in the same population [83]. It is not clear what the minimum latency for a braincancer might be, especially for rapidly-growing astrocytoma. It would be reasonable to assume from these studies that for aggressive brain cancers, expired time since first exposure may be under 10 years in some cases.

Bates (2007) demonstrated a statistically significant elevation in mortality from braincancer among California firefighters (OR 1.35; 1.06–1.72) [22]. Krishnan (2003) examined the association between glioma incidence and occupation in California and found remarkably high odds ratios for firefighters, both as longest-serving occupation (OR 5.88; 0.70–43.01) and ever-employed (OR 2.85; 0.77–10.58); however, because the study design was intrinsically low-powered for any one occupation; neither finding achieved statistical significance [84]. Kang found a statistically significant elevation in risk among firefighters in Massachusetts compared to police (SMOR 1.90; 1.10–3.26), which remained elevated but lost significance when compared to a different referent population (SMOR 1.36; 0.87–2.12) [23]. Thus, within positive studies there appears to be consistency in the risk estimates for this aggregated cancer category.

Ma et al. reported that no elevation in incidence was observed for brain cancer among white firefighters [16]. In her study of Florida firefighters she found a deficit

(SIR 0.58) among men and no cases among women firefighters [21]. Burnett did not observe an elevation for cancer of the brain [14].

Baris et al. also observed a relative deficit in mortality of brain cancer, with an SMR of 0.61 (0.31–1.22) [19]. Risk did not appear to be concentrated in any subset of firefighters by assignment, number of runs, or duration, although the highest SMR (1.18) was observed among firefighters with more than 729 runs in the first 5 years of duty. This study therefore does not invalidate the findings of other studies that suggest an elevation in risk (upper limit of the 95 % CI was 1.22), but it does not support them either.

The NIOSH Study (2013) found no elevation in mortality or incidence, overall or in any subgroup [27]. Similarly, the Nordic Study found no elevation in incidence for brain in general or for glioma in particular [28]. Ide [31] found no elevation in incidence among Strathclyde firefighters (RR 0.92) and no deaths.

The weight of evidence to date, predominantly from earlier studies, suggests that the elevation in risk for brain cancer reflects a true risk, probably for gliomas, which may be concentrated in certain subgroups, as demonstrated for African-America firefighters, the estimates for which are diluted by inclusion into the rubric of tumor types that are not associated with occupation. This conclusion may support a substantial contribution and causation in the individual case or presumption given that elevations observed cannot rule out a doubling for the cancers most likely to be associated with chemical hazards and it cannot be known for certain which types of brain cancer are responsible for the elevation, although glioma is most likely. Youakim, in his meta-analysis, showed that firefighters with over 30 years of service were most at risk [11].

## *Leukemia, Lymphoma, Myeloma*

"Leukemia, Lymphoma, Myeloma" was once a common aggregation in epidemiological studies, heavily used in the past in order to achieve larger numbers for statistical analysis. However, the grouping of these three distinct cancer categories has always been recognized by epidemiologists and clinicians to be an illogical combination, which rendered the analysis spurious. Elevations in one disease or a deficit in another can easily distort the aggregate risk estimate for the rubric. The only legitimate purpose for doing so would be to make a provisional assessment, but even then the aggregation was as likely to mislead as to inform.

## *Lymphoma*

The lymphomas are a super-family of cancers of the immune system and lymphatic tissue. Lymphomas are uncommon cancers but about twice as common as leukemias. Although some lymphomas, notably Hodgkin disease, have a peak in younger

years, most lymphomas tend to manifest themselves at older ages, and appear to have long latency periods, in some cases very long. Their relationships to environmental factors are therefore more difficult to determine, even compared to other cancers. There are many recognized lymphomas, each of which is a distinct disease. Together, they tend to contribute a small number of deaths in most studies and are difficult for epidemiologists to assess as a group, let alone individually. Lymphomas, as a broad generalization, are thought to be caused variously by viruses, chemicals (solvents, pesticides, and chlorinated hydrocarbons as a class have been implicated), persistent antigenic stimulation, or immunosuppression [85]. They tend to appear following suppression of the immune system, for example in association with HIV/AIDS.

Both clinically and epidemiologically, the most important division of the lymphomas is between Hodgkin lymphoma (also called Hodgkin's disease), which, although it comes in different forms, is characterized by the presence of a particular cell type, and everything else, which is called non-Hodgkin lymphoma. Large-scale population-based epidemiological studies in the past generally did not separate out the various types of lymphoma, or did so only as Hodgkin disease and non-Hodgkin lymphoma. When they did attempt to categorize them more precisely, they divided non-Hodgkin lymphoma into the obsolete categories "lymphosarcomas" and "reticulum cell sarcomas", which are only slightly more informative than the aggregated rubric. The rubrics used by epidemiology have historically lagged identification of lymphoma types by a generation. Aggregation was justified on the basis of attaining sufficient numbers for statistical analysis.

Hodgkin disease is actually a class of closely related lymphomas that tend to peak in young adulthood and again at older age and have not been associated with occupational or environmental exposures or occupational risks. (There are two studies that suggest an excess among firefighters, but the weight of evidence is not supportive and the skimpy literature is not ready for critical evaluation.) Hodgkin disease is thought to be unlikely to be associated with external exposure.

The non-Hodgkin lymphomas (NHL) are a heterogeneous family of about 30 cancers of the immune system and lymphatic tissue, each individually uncommon and some quite rare, with strong evidence for an association with chemical exposure for some but not for others. NHL is often taken together as a single undifferentiated rubric of NHL, potentially repeating the same error as the aggregate rubric described above with the same rationale and resulting in the same confusion. Even so, it remains very common, even usual, for NHL to be discussed as if it were a disease rather than a disease category.

Certain non-Hodgkin lymphomas have long been known to be associated with many environmental exposures and occupations involving chemicals, particularly involving herbicide exposure, dioxins and furans, and, most recently, solvents, especially trichloroethylene.) Chronic lymphocytic leukemia, which is more accurately considered a lymphoma that appears in blood, has been identified as a risk of Vietnam veterans exposed to herbicides on this basis, although leukemias in general are not so recognized. It has been clear for decades that each lymphoma is a specific disease, with its own risk factors.

IARC already recognizes an association between non-Hodgkin lymphoma and occupation as a firefighter [53]. IARC [7] conducted a meta-analysis on the rubric as a whole that showed an association between firefighting and non-Hodgkin lymphoma, with a significantly elevated summary risk estimate (SIR 1.21; 1.08–1.36). Both Youakim [11] and LeMasters et al. [10] also recognized a highly significantly elevated risk by conventional scientific criteria in their meta-analyses (in Lemasters, 1.51; 1.31–1.73). McGregor, on the other hand, concluded that the evidence was insufficient to come to any recommendation although he could not, even using the highly conservative conventional standard of scientific certainty, rule it out [86].

Non-Hodgkin lymphomas are individually rare except for the most common two, diffuse large B-cell lymphoma and follicular lymphoma, which are still uncommon diseases. There are over 30 types of lymphoma recognized in the current classification system. New types will certainly be identified in the future as genomic methods become more sophisticated. There are two distinct divisions of the NHLs. T-cell lymphomas constitute about 20 % of NHL and are not known at this time to be associated with occupational exposure, although it is possible that some could arise from viral infection or that persistent antigen stimulation that could be associated with occupation. B-cell lymphomas are malignancies of the cell line that is associated with the form of immunity that relates most closely to antibody development and includes diffuse large B-cell lymphoma (DLBCL), follicular lymphoma, a form of lymphoma that overlaps with chronic lymphocytic leukemia (small cell lymphocytic lymphoma), and several even less common diseases, including Burkitt's lymphoma (which is known to be associated with the Epstein-Barr virus), and Waldenström's macroglobulinemia, which traditionally has been considered among the myelomas. Further complicating the picture is that within the two main divisions it is common for lymphomas to transform into other cell types, reflecting that the immune system is an integrated whole.

The broad group of large B-cell lymphomas, of which there are at least 13, itself appears to be a collection of diseases likely to have different causes [87]. Different types of lymphoma are known to be associated with different occupational risk factors: follicular cell lymphoma with the meatpacking industry and small cell lymphoma, follicular, and diffuse large B-cell lymphomas (DLBCL) with solvent exposure [70, 87–91]. The evidence for solvent exposure will be presented in more detail below.

One may therefore conclude that when a study shows an elevation in the category of "non-Hodgkin lymphoma", it really indicates that one of the most common lymphomas or some but not all of the 30 diseases that make up that category are elevated; it does not mean that risk for every lymphoma across the board is necessarily elevated, although at the present state of the art it is too uncertain to be specific. Likewise, when the elevation is modest or even absent, it does not mean that the risk of a particular lymphoma is *not* elevated within the group. The inevitable conclusion is that summary risk estimates for the lymphomas as a class do not describe the risk for specific diagnoses within the lymphomas for exposed workers, specifically firefighters.

Just because the overall risk for non-Hodgkin lymphomas as a group may be elevated to, say, 1.50, does not necessarily mean that any of the individual non-Hodgkin lymphomas are all elevated to the same degree, or even elevated at all. However, in the absence of data for specific lymphomas it is only an inference to rule out an elevation for any one type, but a reasonable inference for those types that are common and more likely to be associated with chemical exposure.

Given an elevation in the class as a whole, the most parsimonious explanation is that if there is an elevation in one type that is sufficient to elevate risk for the whole group, it is more likely to be in the more frequent types, which would be DLBCP or follicular. DLBCL is the most common, at approximately 25 % of all lymphomas and follicular lymphoma is second, at about 20 %. If the risk for DLBCL, alone, were to be 2.0, then this elevation by itself would elevate the risk for all lymphomas to only 1.17, which is in fact very close to what is observed in many studies. This example does not suggest that DLBCL, or follicular lymphoma for that matter, are in fact doubled among firefighters—they may or may not be but the overall evidence for NHL is consistent with an elevation of this magnitude.

Lymphatic cancers were separately addressed in Burnett et al., which revealed an elevation in mortality for non-Hodgkin lymphoma [14]. The PMR was 161 for firefighters dying under the age of 65 and 130 for those dying at or over the age of 65. With 35 and 66 deaths, respectively, this is a large collection of deaths by lymphoma. These cancers were also separately identified by Ma et al. [16] who found a statistically significant elevation in mortality of lymphatic cancer was observed among white firefighters, with a MOR of 1.4. Among Florida firefighters, Ma et al. found no elevation in incidence among men (SIR 1.09; 0.61–1.80) but a large elevation among women firefighters (SIR 33.30; 0.44–185.00) but based on a single case [21]. (Ma also found an elevation in risk for Hodgkin disease, SIR 6.25; 1.26–18.30, although this lymphoma is not generally considered a plausibly occupational disease and is rarely elevated in occupational studies.)

Baris et al. [19] observed a not-quite significant overall elevation in mortality fromr non-Hodgkin lymphoma, with an SMR of 1.41. While not achieving statistical significance, this rose to 1.72 for firefighters with 20 years or more experience and 2.65 for those assigned to ladder companies. The subset hired between 1935 and 1944 did show a statistically significant elevation of SMR 2.19 (1.18–4.07). A reverse dose–response relationship was observed by number of runs, with the group experiencing the lowest number showing a significant elevation, with an SMR of 2.36 (1.31–4.26), but no relationship was found with runs during the first 5 years. Baris et al. found that among those employed more than 20 years, the SMR was 2.20; 0.90, 3.31). This suggests the possibility that these are true elevations in these subgroups.

Ahn et al. [25] demonstrated an overall elevated risk of incidence of non-Hodgkin lymphoma among Korean emergency responders, who serve multiple roles but are engaged in active firefighting. This large study based on the national cancer registry, which as noted seems likely to be biased toward an underestimate, demonstrated an elevated standardized incidence ratio for non-Hodgkin lymphoma overall (SIR 1.81; 1.12, 2.76).

Kang did not show an elevation in incidence (SMOR 1.10; 0.58–2.09 against all other occupations, 0.77; 0.31–1.92 against police) [23], but its predecessor study (Sama 1990) did, achieving high statistical significance against police (1.59; 89–284 and 327; 1.19–8.89) [24].

The NIOSH study has demonstrated a non-significant elevation (very close to borderline significance overall and in Philadelphia) of NHL (and an increase in risk for San Francisco compared to the original Beaumont study) [27]. Of particular interest is that it showed a strong and significant association between risk and duration of employment, for mortality and an elevation after two decades of employment (only) for incidence,

The Nordic Study [28] did not show an elevation in incidence for non-Hodgkin lymphoma (SIR 1.04; 0.83–1.29) or for any subgroup studied.

Ide [31] reported no elevation for lymphoma (overall) in either mortality (RR 0.74) or incidence (RR 0.81) compared to the West of Scotland, and also to Scotland overall. Because of the age of the population, there may be Hodgkin disease among the cases.

The Australian Firefighter's Health Study is the only study to date that has examined individual NHL among firefighters and no significant elevation was observed. (There was a non-significant elevation for follicular lymphoma among full-time professional firefighters, only.)

Among population monitoring studies, Figgs et al. [37] found an extraordinarily high and highly significant risk of mortality from non-Hodgkin lymphoma in firefighters in 24 states (MOR 5.6; 2.5–12.3) and demonstrate a strong parallel in this pattern of elevation to other occupations involving exposureto solvent chemicals.

More compelling evidence for an association between firefighting and DLBCL and follicular, are derived from collateral evidence for a causal association with particular solvent-class chemicals, as will be detailed later in this section. Follicular lymphoma, for example, has shown a hugely, significantly elevated risk (OR 7.00; 1.45–33.70) among occupations involving exposure to benzene but other lymphoma types (including DLBCL) have not [92]. For any of the other types to elevate the risk for the entire rubric, the elevation would have to be much greater than a doubling and is much less likely to drive the variably elevated risk for the class as a whole. On the other hand, DLBCL seems likely to show an association with trichloroethylene, an industrial solvent also observed as a constituent of fire smoke.

Increasingly, one class among the many exposures in firefighting has been identified as likely to cause non-Hodgkin lymphoma: trichloroethylene and other light organic chemicals found at fire scenes that are more often identified as solvents [93–102]. or chemicals that track with it in concentration, may be associated with elevation in risk of non-Hodgkin lymphomas in other settings [94, 96, 97]. There is important and strong evidence from a relatively large (for gene-toxicant interactions) prospective study conducted in Connecticut using genetic markers that the effect of chlorinated solvents in inducing non-Hodgkin lymphoma is modified by the activity of a specific DNA-repair gene (*MGMT* rs12917, specifically for DLBCL and follicular lymphoma [103]. Another gene, *BRCA 2* rs144848 (more familiar in conferring increased risk for breast cancer) modified the association between

benzene and risk of NHL overall [103]. This is an important set of observations because they confirm that there is a mechanism involving genetic predisposition that links solvent exposure and NHL risk for the two most common lymphomas, which supports the etiological role of solvents while also explaining why, for rare diseases, there would be variation expected from study to study.

If, as seems plausible, different environmental exposures are associated with functional changes in different cell types of non-Hodgkin lymphoma, then the etiology of a particular lymphoma may be more or less specific. A truly elevated risk that arises, for example, from exposure to some constituent of combustion products, may be diluted by inclusion with all the other types of lymphoma that have no association with the exposure. Analysis by specific tumor type might identify which, if any, is associated with the risk but these cancers are uncommon and such a study is probably not feasible for a single occupation. In that regard it is of interest that the two most common lymphomas, follicular lymphoma has been significantly associated with solvent chemicals, both in general [87, 104] and in a specific association between DLBCL and solvent chemicals appear to be associated the chlorinated hydrocarbons such as trichloroethylene (a degreasing agent [94, 102]. Trichloroethylene is known to be present in modern fire smoke, is absent from cigarette smoke, and is classified by IARC as a probable carcinogen (Group 1). Although trichloroethylene is the primary chemical implicated in cancer risk, other chemicals with equal or greater carcinogenic potential may track with trichloroethylene in the same way that a variety of volatile organic compounds track with benzene. It is also possible that benzene itself is associated with increased risk of specific lymphomas. Studies of benzene-exposed populations vary widely in reported risk for outcomes for the lymphomas but some of the reasons for variation, including genetic factors, are becoming known [103].

Thus, the weight of evidence, although not scientific certainty, shows an elevation in risk and appears to favor acceptance of DLBCL and follicular lymphoma as lymphomas associated with firefighting by the weight of collateral evidence [94]. The situation for other lymphomas is not so clear. They are individually even more uncommon diseases and evidence is simply lacking. That does not mean that they are not associated with firefighting, but that direct evidence is lacking. Because it is not possible to exclude an elevation for the other lymphomas and the class as a whole shows an elevation, the evidence is balanced and most workers' compensation systems require the adjudication to give the benefit of the doubt to the claimant in such cases. Thus, a case can and has been made [3] that until such time as risk can be differentiated with some assurance, it is only fair to recognize claims for the entire class.

The latency period for non-Hodgkin lymphoma appears to be very long in most cases, with latencies as long as 35 years frequently cited. It is possible that the gradual decline in function of the immune system with age allows an existing quiescent or suppressed lymphoma to grow and present itself and that this would explain the peak presentation of these diseases in the eighth decade of life.

However, the problem of minimum latency requires interpretation and clarification. The key is latency from what point? It is possible, indeed likely, that in some

cases the onset of immune deficiency would allow a lymphoma that is previously quiescent or suppressed to grow and present itself and that this is why most cases occur at an older age. The minimum latency can be very short with immunosuppression, however, for example following HIV infection and the onset of AIDS. Cases of lymphoma following the sudden onset of immune deficiency are relatively obvious, clinically. In that case, the usually long latency would just be truncated. If a lymphoma is caused by the immunodeficient state, however, which seems to be the case with HIV/AIDS, the latency could be very short, perhaps 5 years or less.

## Leukemias

Hematopoietic cancers are a family of disparate diseases that affect the blood-forming organs, most particularly bone marrow. Most are known as leukemias, because they first appear with abnormal numbers of their characteristic cell type in blood; some, however, appear as deficiencies in cell types and a failure of blood cell production, the most severe examples being myelofibrosis and aplastic anemia. Leukemias of a certain or related cell type (such as myeloid and monocyte cell types may transform from one type to another or from chronic to acute The leukemias are uncommon diseases, about half as frequent as non-Hodgkin lymphomas, overall. There are about a dozen well-recognized forms of leukemia, classified by cell of origin and by acuity, of which five or six predominate. Individually, leukemias are relatively rare and for those other than acute myeloid leukemia (AML) occupational associations are not reliably known. One relatively common type, chronic lymphocytic leukemia, is more properly classified as a lymphoma appearing in the blood.

Different environmental exposures may be associated with different cell types. AML is known to be associated with benzeneexposure, irrefutably. An association with acute lymphocytic leukemia (ALL) at a lower level of risk has been strongly suggested in some studies; myelofibrosis is closely associated with AML. These are therefore the leukemias that are a priori most likely to be associated with firefighting, given that fire smoke contains benzene. However, other leukemias have not been studied sufficiently to rule out an association.

A truly elevated risk of AML for firefighters, which may arise from exposure to benzene in combustion gases, may well be diluted by inclusion with all the other types of leukemia, many of which may have no environmental association. Unless studies are conducted on specific leukemias among firefighters, this problem cannot be resolved and the risk within the class must be inferred from the available data. Such research would be difficult because of the need to accumulate sufficient numbers of cases but not as difficult as for the lymphomas, with their greater number of individual diseases.

McGregor, using a standard of scientific certainty, concluded that an association between benzene and acute myelogenous leukemia was biological plausible but that the epidemiological evidence was not supportive for other leukemias [105]. He recognized that a substantial obstacle was the paucity of studies that addressed hematopoietic cancers separately and individually. LeMasters et al., in their meta-analysis,

concluded, using their standard of scientific certainty, that leukemia as an outcome (without differentiating among them) was possibly associated with occupation as a firefighter [10].

Hematopoietic cancers were separately addressed in Feuer et al. (1986) which found a non-significant elevation in mortality of PMR 1.86 (confidence interval not reported) [106]. Burnett et al. reported a PMR of 171 for firefighters dying under the age of 65 and 119 for those dying at or over the age of 65 [14]. With 33 and 61 deaths, respectively, this is a large collection of deaths by leukemia. Ma et al. [16] observed no apparent elevation for hematopoietic cancers, with an MOR of 1.1 among white firefighters. Among Florida firefighters, she observed no elevation in male and no cases in female firefighters [21]. Elevations were also absent in the recent NIOSH study [27] and a recent study from Korea (Ahn) [25], although an odd classification category in the latter ("lymphohematopoietic cancer") raises the possibility of misclassification and there was a rather high risk (1.68; 0.22–13.06) when firefighters were compared to non-firefighting members of the same department (emergency medical technicians).

Baris et al. [19] found no overall elevation in mortality from the leukemias (SMR 0.83; 0.50–1.37), not specified as acute or chronic or by type. A statistically significant elevation in SMR of 275 (1.03–7.33) was observed for firefighters assigned to ladder companies only, but not to those assigned to both ladder and engine companies. A non-significant elevation was observed for those with a high level of runs in the first five years, with an SMR of 2.44 (0.70–8.54) and with medium (but not high) levels of runs over a lifetime, with SMR of 2.50 (0.56–11.10). These data are not compelling evidence for a true association in this population but do not rule it out. Because of power considerations, the study by Baris et al. does not really clarify this issue.

There is also an important anomaly in the older literature. Aronson/L'Abbé and Tomlinson, in a study of firefighters in Toronto, uniquely reported risk for types of leukemia [50]. They observed an excess in mortality from "lymphatic" [lymphocytic] leukemia at 190 (42–485). This finding was highly influential in the IDSP report [107], but is anomalous. Acute myeloid leukemia (AML) would be expected to be elevated in circumstances in which benzene is a hazard, not lymphocytic. These findings suggest that it is premature to limit the presumption to AML.

Although Ontario now recognizes lymphocytic leukemia, the evidence presented by Aronson/L'Abbé and Tomlinson cannot be used to rule out the possibility of an association with AML. The evidence suggests (again, at the level of "more likely than not") that it cannot, be convincingly argued that only one form of acute leukemia, either myeloid or lymphocytic, should be recognized. Lymphocytic leukemia is suggested by the empirical data in one population, AML by the known toxicological profile of exposures experienced by firefighters. Thus, it is not possible to recommend a selective criterion that only recognizes AML, lymphocytic or, for that matter, only acute and not chronic leukemias.

Ahn et al. [26] found an extremely highly significant elevation in leukemia (83.7; 2.21–3166.3) among firefighters with 20 years of service or more, and high but non-significant elevation among firefighters with between 10 and 20 years of

service (6.54; 0.50–85.12). The Ahn et al. cohort is still quite young and so is better suited for evaluation of acute outcomes or cancers with short latencies than for chronic diseases and cancers with long latency.

Thus, at least for AML, a presumption is well grounded. However, the evidence is not clear enough to exclude other types and all types of leukemia combined. Giving the benefit of the doubt to the claimant, as required, suggests that a rebuttable presumption for leukemias as a class is the most defensible policy on the evidence for compensation purposes.

Leukemias tend to have short latencies, on the order of 5 years or so. Short latencies and therefore duration of employment for leukemia are reasonable, on the order of 4 years to ensure that no errors of exclusion are likely.

### Myelomas

Myelomas are B-cell lymphomas and malignant plasma cell dyscrasias, classified differently from the lymphomas for historical reasons, because the abnormal proteins they express have certain characteristics, and because their clinical manifestations tend to be similar among the class and different from other cancers. McGregor concluded that there was no evidence supportive of an association, based on the standard of scientific certainty [108]. LeMasters et al. reported a relatively high overall elevation in the literature up to that time (summary risk estimate 1.53; 1.21–1.94), making it a major finding of her study [10]. This is where matters stood until about 2008.

Baris et al. found an overall non-significant excess of mortality (RR 1.7; 0.9–3.1) increasing with duration of employment, with 20+ years having a borderline statistically significant SMR of 2.31 (1.0–5.2), and a statistically significant SMR of 2.54 (1.2–5.7) for engine company employment only, with some suggestion of correlation with medium and high diesel exposures (latter based on small numbers of deaths) [19]. This pattern is suggests a association too strong to be dismissed as confounding. However, the NIOSH Study [27], which did not examine specific jobs, did not find an elevation overall or in any subgroup or city, including Philadelphia, which was the location of the Baris study, even though the two studies would have included many of the same subjects.

The weight of direct evidence suggests that myelomas and related cancers in this category (it is a large and complicated rubric) can arise from occupation as a firefighter, but it is entirely unclear which and it is possible, given the biology, that more than one or even all could be. Giving the benefit of the doubt to the worker, as required, the preponderance of the thin evidence favors causation and that the association reaches a level (doubling of risk) that favors a presumption. This is also consistent with the recommendation for non-Hodgkin lymphomas, some of which, particularly the more common B-cell lymphomas, overlap with myelomas biologically and therefore possibly in causation.

The latency period for myeloma appears to be very long in most case, but without a clear causal relationship from which to mark the beginning of exposure it is unclear how one could accurately measure it. The minimum latency is unclear.

### Interpretation

The weight of evidence for lymphatic cancer of the non-Hodgkin type and hematopoietic cancer suggests that the elevation in risk reflects a true risk in certain subgroups but these subgroups cannot be readily identified by usable criteria in adjudication. So far, the weight of evidence suggests that AML, DLBCL, and follicular lymphoma, as well as various myelomas, in a firefighter are more likely than not to arise out of their work. Likely candidates among fire smoke exposures have been identified for which the evidence is strong, including human studies. Because the other individual disease risks cannot be separated using current knowledge, the doctrine of giving the benefit of the doubt to the applicant suggests that they be taken as a group for purposes of presumption, at least until more information becomes available.

## *Lung Cancer*

Lung cancer has been the most difficult cancer site to evaluate in epidemiologic studies of firefighters. Despite the obvious exposure to carcinogens inhaled in smoke, it has been difficult to document an excess in mortality from and incidence of lung cancer of a magnitude and consistency compatible with occupational exposure.

The risk of lung cancer associated with any occupation is usually overwhelmed by the effect of cigarette smoking if there is a difference in smoking rates compared to the reference population. On the other hand, as an example will show, the confounding effects of smoking may be exaggerated. The evidence suggests that an association does exist but that it is heavily obscured by confounding factors and may not be as strong as might be supposed in the toxicological literature. Confounding among firefighters is also not straightforward, as it reflects changing patterns of tobacco use in the past (See Chap. 4.).

Occupational exposure assessment is also much more complicated than tallying person years of exposure. Municipal fighters entering the fire service in Alberta from 1927 to 1987 showed some evidence for an increase in risk of mortality overall and by duration of exposure, but the trend for the first two decades was not smooth or significant. The elevation falls off after 20 years, and then becomes marked and significant after 35 years. The data also suggest a high risk for new hires and firefighters who may not have completed their probationary period satisfactorily [44]. It is noteworthy, however, that smoking rates were low in the general population of

Alberta at the time, so that this study may come closer to an unconfounded comparison than most other studies of firefighters [109].

The trend may be heavily confounded by smoking and by era but it could also suggest that duration of firefighting is only part of the picture. Susceptibility to lungcancer (which in at least some phenotypes also confers susceptibility to chronic obstructive pulmonary disease) may play a role in both early incidence, survival, and later incidence. Respiratory protection has reduced some individual exposure levels to combustion products since the 1970s, to the extent of individual and group compliance, and this may be the reason that studies rich in recent person-years of observation, such as Baris et al. [19], do not observe such elevations. On the other hand, Ma et al. [43], who describes no elevation in risk for male firefighters in Florida, found the usual moderately elevated risk for lung cancer (SIR 1.40; 0.28–4.08) among females, who in general entered the fire service so recently relative to men that most or all the required latency has not yet expired. Smoking rates have fallen precipitously and the Australian Firefighters' Health Study even shows a striking deficit of lung cancer for all firefighting groups (0.81; 0.65–1.00 for full-time professional firefighters) but of course not as low as might be expected in a non-smoking population [47]. Ide [31] found a significant deficit among Strathclyde firefighters in the mortality rate (RR 0.27) and incidence rate of lung cancer (RR 0.3, $p<0.001$), but this is more likely to reflect historically high smoking prevalence in the general population of the West of Scotland which served as the reference group.

Firefighters appear to have smoked generally less than the general population for a very long time and less than other "blue collar" occupations in the 1990s [110, 111]. It is estimated from recent data in the central states of the US that approximately 13.6 % of professional firefighters smoke, less than the 21 % of the general adult population and much less than the 29 % prevalence of comparably highly-paid, highly-skilled blue-collar workers. Firefighters, at least in North America, appear to smoke less even than the 20 % prevalence of white collar workers, who are usually taken to define low-risk groups for lung cancer and other smoking-related disorders [110, 112]. Therefore, the proportion of the lung cancer burden attributable to occupation as a firefighter, rather than cigarette smoking, is likely to be higher than generally assumed in the literature.

A comparison that takes into account the prevalence of cigarette smoking is illuminating.

### Population Risk Attributable to Firefighting

Many studies have shown an excess of lung cancer on the order of 20–80 % (i.e. SMRs around 120 or 180), a magnitude not uncommon in studies of other blue collar occupations with less plausible exposure levels [113]. LeMasters, summarizing the literature to 2006, found no elevation [10].

However, the empirical findings on lung cancer from recent, well-designed epidemiological studies have been inconsistent [114]. One study from Denmark

reported a standardized mortality ratio of 317 for older fire fighters but the comparison population was unusual and difficult to interpret [115]. Studies on cohorts from San Francisco [35] and Buffalo [116] showed no excess and even suggested a deficit, as do most of the population monitoring studies (which systematically tend to underestimate risk, and so are not cited in this section).

On the other hand, the NIOSH Study, with its large number of subjects and power, showed a statistically significant overall elevation for both mortality and incidence (SMR 1.10; 1.04–1.17) a remarkably consistent across almost all subgroups, except for no elevation among non-Caucasians [27].

The only unequivocal increase in lung cancer mortality or incidence observed to date comes from the Nordic study [28], which provides evidence for a significant elevation of incidence for adenocarcinoma of the lung (SIR 1.29, 1.02–1.60), but not other tissue types. This presents an interpretive problem because adenocarcinoma is the characteristic tissue type for both non-smokers and in persons exposed to passive cigarette smoke, although the frequency is also elevated in smokers compared to non-smokers. This suggests that in this context fire smoke might be behaving more similarly to sidestream cigarette smoke than to mainstream cigarette smoke. However, this observation must remain speculative until more information is available and the finding is confirmed.

There are collateral reasons for thinking that the true risk has been underestimated in career firefighters. All but a few extant studies that are positive, relevant, close to the primary data, large and well done seem to cluster in a band from an excess of 30–68 %, as can be seen in the Appendix. The principal exceptions are Baris et al. [19], and Vena and Fiedler [116]. Baris et al., despite a low overall risk (1.13; 0.97–1.32) does report suggestive elevations in certain subgroups, notably fire fighters with less than 9 years of service (1.52; 1.16–2.01), those assigned to engine companies (1.18; 0.93–1.51), and those hired before 1935 (1.30; 0.97–1.73). Vena and Fiedler present one of the lower overall risks in the fire fighting literature (0.94; 0.62–1.36) but a close reading of their data show a possible exposure-response relationship with duration of employment (a near-monotonic increase of 0.14 relative risk for each of five decade of fire service, nonparametric $p<0.07$) and a statistically significant excess (at $p<0.01$) for fire fighters with more than 40 years of fire service (1.29). Vena and Fiedler compared their incident cases to the US general population, however, in that era Buffalo residents, presumably including firefighters who lived there, already had one of the highest mortality rates of cancer in the United States, so that elevation above background might have been constrained in magnitude [117]. Heyer et al. [83] reported an overall risk of only 97 (65–139) but observed an elevated risk among fire fighters aged 65 years or more, when the incidence of lung cancer tends to peak. Thus, even in so-called "negative" studies there are hints of a possible association.

Among those studies that appear to be unequivocally "negative", Beaumont et al. [35] reports the lowest risk (0.84; 0.64–1.08). This same study is unusual among the major studies because it also shows the largest healthy worker effect, which is atypical for firefighters [2]. It also shows the lowest overall mortality from all causes

(0.90), the lowest mortality rate from cancer (0.95), an atypical age distribution, and a high rate of cirrhosis.

At the other extreme is Hansen et al. [115] in which an overall risk (as SMR%) of 163 (75–310) was accompanied by a tripling of risk (SMR% 317) for firefighters aged 60–74. This was an imaginative Danish study that aggregated other occupational groups into a synthetic reference group. However, the artificiality of this construct makes the study difficult to interpret.

In a study of urban fire fighters in Alberta [44], trends appeared to suggest a true risk (originally reported as SMR% but converted here to decimal) on the order of 1.50 in that population. The overall SMR for lungcancer was 1.42 (95 % confidence interval (0.91, 2.11), statistically not significant, and statistically indistinguishable from 150. However, lung cancer was elevated to an SMR% of 167 among fire fighters entering the fire service in the 1960s, the most recent cohort at the time of the study for which the expected latency period had elapsed. This is not strong evidence, because it is based on only two cases, but the following cohort of firefighters entering in the 1970s showed an even greater risk, 261 (although based on a single case). The risk of lung cancer also showed an exposure-response relationship in our data, with groups of fire fighters who had higher exposure opportunities and duration showing elevations on the order of 2.00. By duration of employment, an initially high risk for those with less exposure declined with duration of employment but achieved a doubling for those working 40 or more years (although only two firefighters were in that group). More persuasively, when duration of employment was corrected for exposure opportunity in job classification, the exposure-response relationship changed to suggest, following an initially high risk among probationary fire fighters or those unfit for duty, a more or less consistent but low elevation for the middling exposed varying around 1.50 (range 0.32–2.58), and a significantly elevated risk (4.08, $p<0.05$) for those with more than 35 exposure opportunity-weighted years of employment.

It was observed above that the study of urban firefighters in Alberta probably comes closer to an unconfounded estimate of the risk of lungcancer as other studies of firefighting, because of low rates of smoking in the reference population. An important factor in the Alberta study, which was not appreciated at the time of initial publication, is that cigarette smoking was historically less of a confounding factor in Alberta than it has been in other populations [109]. Subsequent studies of smoking-related lung disease outcomes suggest that smoking rates have been historically low in the province compared to the rest of the country and this is reflected in lower mortality from chronic obstructive pulmonary disease. In recent years mortality rates for smoking-related disorders appear to have converged with the rest of Canada as smoking rates in the rest of the country have gone down and those in Alberta have changed less dramatically. This suggests, but does not prove, that the Alberta experience is less confounded by cigarette smoking than elsewhere.

However, an unexplained anomaly of the Alberta data is that the excess was seen in one city (Edmonton) and not another (Calgary). In Edmonton alone, the risk was 2.01, the highest overall risk for lungcancer reported [44]. The two cities represent an internal replication because the same study team collected data from both cities

following the same protocol, matched against death certificates concurrently and analyzed both data sets simultaneously. Other causes of death were consistent between the two populations.

Unfortunately, the data from other studies cannot be disaggregated on the same basis as the Alberta cohort. Even so, Baris et al. [19], although negative overall, appeared to show the same effect over the first 9 years.

### Smoking and Firefighting

It is well established to the point of being a convention to accept 20 American pack-years (equal to 16 Canadian pack-years) of smoking (pack-years represent the total number of packs per day multiplied by the number of years a person smokes at that level) as the point at which lung cancer risk begin to rise noticeably and exponentially. This is not a threshold for toxicity, as there is a measurable risk below that level; there is no safe level of cigarette smoking. It is simply the visible inflexion point at which the risk curve takes off for the population. It should be noted that, like duration of employment, smoking history is confounded by age, since years as a smoker are years lived, and after about age 50 (noticeably after 55) the risk of lung cancer among smokers rises as a power function. It should be noted in passing that notwithstanding the high risk of lung cancer associated with cigarette smoking, most cigarette smokers do not develop lung cancer. Whether they would if they all lived long enough is another question.

It should be noted that, paradoxically, cigarette smoking exposure is more potent per unit smoked at low intensities of smoking, meaning that the risk *changes* more dramatically at levels below one half-pack per day than at heavier smoking levels, but the overall excess risk is still higher with cumulative exposure, with longer duration and intensity. As a practical matter, this means that a cigarette smoking habit of about 25 years' duration probably roughly matches the risk from firefighting but that above that level cigarette smoking becomes much more of a driver of cancer risk and overwhelms the firefighting effect, rendering it negligible compared to smoking.

Whether the additional risk from firefighting contributes substantially to the overall risk in a cigarette smoker depends on what is deemed to constitute a substantial contribution. Certainly, one could make a case that at 20 (American) pack-years, firefighting is still contributing a significant amount of risk, which, if apportioned in a "typical" individual taken from this population, might be about half of the risk of lungcancer (recognizing great uncertainties and individual factors). In such a situation, perhaps half of firefighters who smoked at that level would not have developed cancer "but for" their work as a firefighter. Above that level of smoking, however, the risk from smoking dominates, making the relative contribution from firefighting small.

It might be argued that, like other carcinogens, cigarette smoking and other exposures arising from firefighting (fire smoke and diesel exhaust) are positively interactive (or "synergistic") and cause more lungcancer than would occur otherwise.

This is possible but unlikely, because fire smoke and cigarette smoke are products of combustion and mostly contain the same carcinogenic chemicals, particularly polycyclic aromatic hydrocarbons and nitrenes, and other carcinogens including vinyl chloride and benzene. However, interaction cannot be completely ruled out on toxicological grounds because the smokes are not exactly alike.

Fire smoke, although generally simpler as a mixture but still complex (see Chap. 5), contains a variety of compounds not found or found in less concentration than in cigarette smoke, including trichloroethylene and other chlorinated hydrocarbons. Fire smoke contains much higher levels of trichloroethylene (a carcinogenic solvent chemical) and cigarette smoke contains a radionuclide that is concentrated in tobacco ($^{210}$Po), among other differences. Cigarette smoke inhalation does not carry the risk of exposure to asbestos but firefighting clearly does. Some constituents of diesel exhaust (a more potent source of nitroarenes) are part of the exposure profile of firefighters but not cigarette smokers. Also, as noted in Chap. 5, cigarette smoke has some anti-inflammatory activity that fire smoke does not appear to demonstrate. These differences mean that fire smoke and cigarette smoke (and for that matter fine particulate air pollution) cannot be equated in their toxicity, and may show substantial differences.

For purposes of assessing cancer risk, however, it likely that the relative contribution to risk of cigarette smoke and smoke inhaled during firefighting are proportionate to exposure on a simple additive basis (considering exposure level) to a first approximation. Because the toxicological properties of the smoke are generally similar and mostly act by the same biological mechanisms, the contribution to risk should be additive, not interactive (synergistic). Smoking habits are variable but the average smoker almost certainly inhales smoke for a longer total time period during the day than the average firefighter does during the course of an average working day. Cigarette smoke is also inhaled directly from the source and so is less dilute than most (by no means all) opportunities to inhale fire smoke.

However, this argument applies only to lung cancer, not to deep cancers or to respiratory effects. Although similar, cigarette smoking is not identical to fire smoke. Cigarette smoke, as a complex mixture, also contains some poorly characterized anti-inflammatory agents that suppress the potential acute irritation, which may in part explain why people tolerate the irritation to airways. Cigarette smoke contains many constituents that act locally to induce cancer. In lung, smoke constituents are mostly pre-metabolized (locally, in the liver, or in the lung), which may activate procarcinogens, adding to the vulnerability of that organ.

Even so, the argument for a difference in the effect of the two smokes is more convincing for respiratory effects than for carcinogenicity. The weight of evidence suggests strongly that cigarette smoking is likely to be at least as potent as fire smoke and usually more concentrated when inhaled, and exposure clearly occurs more frequently among smokers. In that case, the effects are much more likely to be additive and proportionate to the cumulative carcinogenic exposure.

By a mathematical manipulation of the known data, it has been proposed that compared to nonsmokers as a group, nonsmoking firefighters are estimated to have

much more than a doubling of risk compared to other people who do not smoke [3]. The exact value of the estimated risk, about a tripling, is unimportant because of the compounded uncertainties; that it clearly exceeds a doubling is what matters most. This derivation suggests that the risk of lung cancer in a non-smoking firefighter is more than doubled (estimated to be about 3.3) compared to the general population of non-smokers, making allowances for great uncertainties. This suggests that exposure within the profession of firefighting is important and, making allowances for uncertainty, triples the risk of lung cancer in a person who would otherwise have a very low risk. An elevation of three-fold is a large risk for an occupational hazard.

## *Mesothelioma*

For many years firefighters have sought recognition of their exposure to asbestos as an important hazard but this has been difficult to substantiate because the characteristic signs of lung diseases due to asbestos are not frequent on chest films of fire fighters. (There is no proper study, but over the years numerous opportunities to have made the observation.) This has now been accomplished with the demonstration in two studies of an elevated risk of mesothelioma in firefighters.

Mesothelioma is a distinct cancer of the lining of the thoracic or abdominal cavities (the pleura and the peritoneum, respectively). It is caused by asbestosexposure, almost exclusively. Some cases are associated with fibrous (asbestos-like) naturally-occurring minerals called zeolites (mostly erionite). It is controversial whether mesothelioma ever occurs without exposure to an asbestos-like agent, but if it does this is exceedingly rare. Mesothelioma, unfortunately, has a poor prognosis and so mortality and incidence data are similarly informative.

Exposure to asbestos is likely to occur when firefighters engage in cutting into structures and in overhaul, when asbestos-containing materials are present. This is most likely to occur in fighting fires in older buildings, including houses, in recent years especially those that have not been renovated or remediated.

The NIOSH Study demonstrates a significantly elevated risk for overall mortality (2.00; 1.03–3.49) and incidence (2.29; 1.60–3.19), and in all three cities (Chicago and Philadelphia and elevated but not statistically significant in San Francisco) [27]. This is entirely consistent with asbestos-containing materials present in older housing and building stock.

The Nordic Study demonstrated an overall risk of mesothelioma was elevated (SIR 1.55; 0.90–2.48) and significantly elevated among firefighters over 70 years of age (SIR 1.56; 1.25–4.56) [28]. The latency period for mesothelioma is variable but tends to be very long; four or five decades is not unusual. Firefighters currently at or older than age 70, if they entered the fire service in their twenties or thirties, could have been exposed outside the fire service in the 1960s or 1970s when asbestos was still used in construction, in addition to exposure during their tenure in the fire service. However, the persistence of the elevation in firefighters younger than 70 strongly suggests that their relevant exposure occurred mostly or exclusively on the

job, because asbestos was removed almost completely and abruptly from commerce in developed countries in the 1970s, particularly in Scandinavia.

The Australian Firefighters' Health Study showed a broad general elevation in mesothelioma risk among firefighters but not to a level of statistical significance [47].

Obviously this presumption would be rebuttable if there were other, more significant exposures to asbestos, for example in moonlighting jobs and construction. However, the persistence of the elevation into more recent cohorts and younger firefighters, well after asbestos was removed from general commerce and construction in North America, argues that the risk is occupational to firefighting.

Therefore the weight of evidence strongly favors the conclusion that mesothelioma is an occupational disease of firefighters, at a level sufficiently strong to justify a presumption.

## *Colon and Rectal Cancer*

Colon and rectal cancer are two categories of cancer of the large bowel which are biologically virtually identical. Both are virtually always adenocarcinoma. Among firefighters, risk for colon and rectal cancer has focused more often on occupation-related lifestyle issues than on chemical exposure. Shared risk factors for both colon and rectal cancer include: lack of exercise and sedentary lifestyle; a diet low in fiber, fruit, and vegetables, inflammatory bowel disease (ulcerative colitis or Crohn's disease), dietary consumption of fatty foods; obesity and diabetes; heavy alcohol intake; and tobacco consumption. There are also several hereditary conditions that predispose to colon cancer, of which familial polyposis is the most common. Chronic or frequent constipation may be a symptom of colon cancer but does not appear to be a risk factor for its development.

Because colon and especially rectal carcinoma can be detected early by screening tests, such as visible blood in stool, tests for occult blood in stool, sigmoidoscopy (rectum only), colonoscopy, and imaging studies, these two cancers could be subject to similar screening biases as described earlier for prostatecancer. However colon and rectal cancers eventually declare themselves with symptoms and signs while indolent prostate cancer may go undetected until the end of life, so the comparison is not exact.

Lemasters et.al [10]. demonstrated a significantly elevated risk of approximately equal magnitude for both colon cancer (summary risk 1.21; 1.03–1.54) and rectal cancer (1.29; 1.10–1.51). Individual studies vary, with some (Demers 1992) [82] showing no elevation in either but most showing an elevation in either or both.

The literature generally supports the conclusion that there is an increased risk of coloncancer among firefighters, in general, but not that this increased risk equals or exceeds a doubling, which would correspond to the criterion of "more likely than not" in the individual case. Recent studies, including thorough and detailed work of high quality such as Baris et al., although showing variability common in such

occupational studies, have not refuted this conclusion and have strengthened the evidence for an association both by replication [19]. Overall, Baris et al. found an SMR of 1.51 (1.18; 1.93), based on 64 deaths; there was no consistent dose–response for duration of employment or for cumulative number of runs. However the risks were greater than 1.00 for all three levels, 1.93 for low; 2.22 for medium and 1.22 for high number of runs. Elevated colon cancer risk has been reported in many other studies, including Schwartz and Grady, who examined occupational associations of colon cancer in New Hampshire in the 1980s [118]. Vena and Fiedler in Buffalo [116] reported the highest risk, a significantly elevated SMR of 1.83, but used US national rates as a reference and at the time Buffalo had a highly elevated cancer rates compared to the US population as a whole.

Kang, using a methodology that tends to underestimate risk, observed a statistically significant excess in incidence when compared to police (SMOR 1.36; 1.04–1.79) but not another referent population (SMOR 1.15; 0.93–1.43) [23].

Ahn et al. [25] demonstrated an overall elevated risk of incidence for cancers of the colon and rectum among Korean emergency responders, who serve multiple roles but are engaged in active firefighting. This large study based on the national cancer registry, which as noted seems likely to be biased toward an underestimate, demonstrated an elevated standardized incidence ratio for colorectal cancer (SIR 1.35; 1.07.1.67).

The NIOSH Study reported a consistent and significant elevation in both mortality and incidence of colon cancer (SMR 1.31; 1.16–1.48, SIR 1.28; 1.09–1.43), consistent across racial groups and showing a small monotonic increase in the first three (out of four) decades of employment duration [27]. Similarly, the same overall pattern was observed for rectal cancer with fewer numbers, without a clear duration-response relationship and with some associations not achieving statistical significance, (SMR 1.45; 1.16–1.78, SIR 1.11; 0.95–1.30). Neither were compared by city.

The Nordic Study [28], showed a non-significant elevation in incidence for coloncancer (1.14; 0.99–1.31) but not rectal cancer (0.99; 0.82–1.10).

Ide [31] did not find an elevation in colon cancer (four cases) mortality (RR 0.75) or incidence (RR 0.65) compared to the West of Scotland or Scotland as a whole.

The Australian Firefighter's Health Study showed a non-significant elevation in incidence for both colon and rectal cancer for full-time professional firefighters (1.13; 0.91–1.31 and 1.18; 0.89–1.54, respectively), no elevation for colon or rectum among part-time professional firefighters (0.91; 0.60–1.33 and 1.15; 0.75–1.80), and a significant deficit for colon and similar deficit for rectum among volunteer firefighters (0.87; 0.80–0.98 and 0.90; 0.80–1.01). This pattern does not prove but is consistent with an exposure-response relationship in which there is an elevated risk arising from occupational exposure but a strong healthy worker effect demonstrated most clearly among volunteers, which would be reasonable for a cancer that reflects lifestyle factors.

A few studies (Kang [23], Burnett [14]) show a wide discrepancy between colon cancer, which is significantly elevated, and rectal cancer, which is less often elevated.

It is possible that non-occupational risk factors are driving the reported differences. However, a significant divergence in the two cancer sites would require a difference in distribution of risk factors among firefighters compared to the general or reference population.

Many authors emphasize the differences in the risk factors associated with colon and with rectal cancer and suggest that this is an obstacle in interpreting earlier studies (such as Guidotti [44]) that report combined colorectal cancer rates. Rectal cancer shares the same risk factors as colon cancer but also features additional known risk factors associated with lifestyle among certain subgroups defined by sexual preference and practices. To confound the risk estimate for firefighting as an occupation, these subgroups would have to be substantially more frequently represented among firefighters than in the general population. There is no evidence or reason to believe that this is the case and it is unlikely.

A prospective study attempted to detect rectal and sigmoidal polyps in less than 200 firefighters in Phoenix (Arizona) over 7 years from 1988 to 1995. On the basis of unremarkable findings and no carcinomas found, the author concluded that there was no elevation in risk. However, this study did not meet contemporary standards for cancersurveillance studies: there was no reference group, the study population was tiny, no poweranalysis was performed, and the method of detection, sigmoidoscopy, was inadequate in that it does not visualize the full colon. This study, which was only reported in abstract, therefore should carry little weight in rebuttal and should be disregarded [119].

Youakim [11] concluded on the basis of his meta-analysis that the risk for colon cancer among firefighters was significantly elevated, appeared after 30 years of service, and was highest after 40 years.

Overall, the weight of evidence suggests that colon cancer and rectal cancer incidence are elevated among firefighters and that there is a consistent association with occupation as a firefighter. This association may or may not have more to do with status as a firefighter and work organization (such as shiftwork, exercise habits, and diet while at the fire station) than with exposure to fire smoke and other chemical hazards. It should be noted that whether the underlying cause is chemical carcinogenesis or work organization, the condition would arise from work as a firefighter.

Although currently accepted in some states as part of a cancerpresumption schedule, the best evidence for elevation in colon and rectal cancer rates among firefighters falls short of a doubling of risk, so that on epidemiological grounds the evidence may be considered insufficient to support presumption. However, for individual firefighters who are low risk for colon or rectal cancer due to their personal risk profile it may be reasonable to apportion colon cancer to occupational risk. Notionally, for example, if a person who develops colon cancer has a low a priori or antecedent risk profile (such as a vegetarian who exercise and has no family history) but develops the disease anyway could in his (or her) personal risk match the magnitude of the attributable risk for the group associated with firefighting (1.50 elevation overall implies that one third of the total risk is attributable to firefighting). Unfortunately, this argument become convoluted and cannot take into account

random effects, so it is speculative at present. However, in presumption systems that rely on demosntrating "substantial" or "significant" contribution to risk, the magnitude of risk associated with firefighting would seem to meet the standard easily.

## *Thyroid Cancer*

Thyroid cancer is a relatively uncommon cancer, the most common forms of which are easily treated and seldom fatal. It is therefore not usually observed to be elevated in mortality studies. There is no obvious exposure in firefighting that would be associated with thyroid cancer. Elevations have only been noticed recently in studies of firefighters, but this delay is more likely due to the application of different methods than it is to reflect trends over time. Thyroid cancer might be affected by screening-bias favoring detection of cases in individuals with better health care, since it is possible to have asymptomatic thyroid cancer.

The study by Ma et al. [43] of Florida firefighters contained the striking observation that cancer of the thyroid is markedly and statistically significantly elevated in incidence for both male and female firefighters (SIR male 1.77; 1.08–2.73; females 3.97, 1.45–8.65) [21]. As yet, these findings have not been replicated. Thyroid cancer appears separately in this study and was omitted from mortality studies by the same author, because mortality is a poor indication of frequency for this cancer site. Kang et al., in the study of Massachusetts firefighters, did not observe an excess in incidence but that study had much less power [23]. The NIOSH Study, which reports thyroid cancer in a set of supplemental tables posted on-line rather than in the main report), showed no elevation for thyroid cancer overall or by city [27]. Cancer of the thyroid was not elevated in the Nordic Study [28] and was not significantly elevated in the Australian Firefighters' Health Study [47].

As yet, there is insufficient evidence to establish a weight of evidence to make a recommendation.

## *Head and Neck Cancers*

Head and neck cancers have often been overlooked or aggregated in firefighter studies. These cancers tend to be highly disfiguring and dreaded.

Head and neck cancers tend to be individually uncommon and are easily misclassified. Head and neck cancers, in general, are associated with many risk factors in common, involving sunlight (ultraviolet radiation exposure, especially for lip), comorbidity (previous treatment for cancer, radiation), lifestyle (smoking, smokeless tobacco usage), and occupational exposures (wood dust, agriculture). Individual head and neck cancers have particular risk factors as well.

Lemasters in her meta-analysis suggested an overall elevation in the literature to 2006 (summary risk 1.23; 0.96–1.55) [10]. This meta-analysis did not take individual

sites into consideration. However, this is a more useful aggregation than most because head and neck cancers share such a remarkable number of risk factors.

*Cancer of the lip* was noted to be highly elevated by Beaumont [35] but not significantly so due to low numbers (RR 6.17; 0.75–22.29, two cases) and significantly by Sama [24] (MOR 5.9; 1.9–18.3, three cases) although in both studies the number was very small. The Australian Firefighters' Health Study showed small, non-significant elevations in all firefighter categories. Exposure to sunlight is one plausible explanation for these observations, with or without promotion by chemical carcinogens.

*Oral and pharyngeal cancers* are mostly squamous cell cancers, with known associations with smoking, smokeless tobacco, alcohol abuse, cocaine abuse, nickel subsulfide exposure (not a hazard of firefighting), welding fumes (preliminary), radiation, betel nut chewing, regular consumption of Chinese salt-cured fish, lichen planus (a disease of the mouth), and infection with Epstein-Barr virus or HPV16 (a human papillomavirus that often populates the female genital tract). None of these are plausible hazards associated with firefighting. Of the few studies of firefighters that do break out sites in the oral cavity and pharynx, most have not shown an elevation, with some conspicuous and noteworthy exceptions: Aronson/L'Abbe 1992 showed a non-significant elevation in risk of pharyngeal cancer (SMR% 139) increasing with years since first exposure (which conflates latency with exposure duration) [120]. Demers showed a modest, non-significant deficit in oral and pharyngeal cancers (SMR 0.81; 0.33–1.66) [52, 82]. Baris found an elevated risk (SMR 1.36; 0.87–2.14) for the buccal cavity and pharynx [19]. Kang found no elevation in cancers of the buccal cavity and nasopharyns [23]. The NIOSH Study [27] showed a significant elevation for cancer mortality and incidence of the mouth and pharynx (respectively 1.40; 1.13–1.72, and 1.39; 1.19–1.62), and marked elevations in incidence that are consistent across locations (except some anomalies in Philadelphia) and for various oral and pharyngeal structures, most of which do not achieve statistical significance because of small numbers. The Nordic Study did not observe elevation in incidence for oral cavity, pharynx, tongue, or lip. The Australian Firefighters' Health Study showed small, non-significant deficits in all firefighter categories after the exclusion of lip cancer. The overall impression is that while there may well be an elevation in risk for cancers of the mouth and pharynx, there are also a number of confounders or exposures unrelated to the duties of firefighting, chief among them oral tobacco use.

*Cancers of the salivary glands*, including the parotid gland (in the cheek), were not separately examined in most studies (for example, they are not separately examined in the NIOSH Study [27]) but became an outcome of interest after Ahn demonstrated a high but not significantly elevated incident risk based on very small numbers for Korean firefighters (SIR 2.34; 0.47–6.83, three cases) with no association with employment duration, but not seen in non-firefighting emergency medical technicians in the same departments [25]. This group was also somewhat elevated for white firefighters in the study by Ma (1998) at MOR 1.3 with only three cases). The Nordic Study [28] reported an elevation but with wide confidence interval (1.89; 0.81–3.11) based on ten cases. Parotid gland tumors are known to be

associated with Sjögren's syndrome (dry mouth), radiation exposure, and exposures in the rubber industry (thought to be dominated by nitrosamines), none of which is likely to apply to firefighting. Why the parotid gland would be so susceptible in firefighters is not clear but the weight of evidence suggests that an association, although not sufficient to consider a presumption.

*Cancer of the nasal sinuses* would be expected to be associated with firefighting because smoking is a risk factor and "coarse" (relatively large) particulate matter such as wood dust tends to be retained in these locations. Surprisingly, given the likely exposure of firefighters to wood dust and coarse particulate matter carrying PAHs, cancer of the nasal sinuses has been separately investigated only once, by Demers (1994) in a three-city study, which found an elevation based on very small numbers (SIR 2.12; 0.1–12.4, two cases) [52]. This is insufficient evidence on which to make a recommendation.

*Laryngeal cancer* might be expected to be associated with firefighting since the vocal apparatus is in a vulnerable position for exposure to inhaled carcinogens and cancer of the larynx is strongly associated with cigarette smoking. Cancer of the larynx shares many risk factors with lung cancer, including asbestos, with the additional risk factor of alcohol abuse. LeMasters found a summary risk estimate of 1.22 (0.87–1.70). Several studies have shown a deficit for firefighters individually [16, 35, 52]. The NIOSH Study showed highly variable risk estimates, with mortality significantly elevated once, in Chicago only (1.55; 1.01–2.27) and incidence elevated several times in subgroups and overall (1.50; 1.19–1.85). The Nordic Study found no elevation in risk. Thus, the weight of evidence is unclear but the association should be considered in individual cases because in most jurisdictions the legislative instruction is to give the benefit of the doubt to the worker.

## *Breast*

There has been insufficient experience with women firefighters to study, let alone determine, whether there is a contribution to risk of female breastcancer. On the other hand, there has been considerable interest and concern over the risk of male breast cancer.

In the United States, breast cancer among men is exceedingly rare, at about 1 case per 100,000 men per year, a rate that may be increasing slowly. Male breast cancers comprise much less than 0.5 % of cancers in men overall in the US, although rates are much higher in some parts of Africa. The strongest risk factor for male breast cancer is age, and most cases occur in men over age 65. When it does occur, the tissue type for male breast cancer is "infiltrating ductal carcinoma", which should not be confused with the relatively slow-growing "intraductal" type that has been diagnosed increasingly among women with near-universal use of mammography. Male breast cancer is normally estrogen-sensitive and aggressive.

Meta-analyses have not been conducted for this site because few or no early studies looked specifically at male breast cancer.

Male breast cancer has attracted attention in recent years because of several high-profile cases among firefighters. The Supreme Court of Nevada (whose authority is limited to that state) has ruled that a woman firefighter was entitled to presumption for breast cancer as arising out of work as a firefighter, in January 2011, on the basis that firefighting exposed workers of both sexes to chemical carcinogens. The court unfortunately focused on benzene as the putatively responsible carcinogen, which is unlikely but could serve as a surrogate for exposure to the many and varied other fire-associated carcinogenic exposures. The Court is quoted as opining "despite the limitations of some of the studies, we conclude that a reasonable person could have found from the totality of the evidence presented at the hearings that the benzene (the firefighter) was exposed to was reasonably associated with breast cancer." The emphasis on totality of evidence and "a reasonable person" (implying weight of evidence) is similar to the framework described in the 1995 and the 2005 papers cited above.

Ma (2005), evaluating risk of death (mortality) by cancer among firefighters in Florida, found a highly and highly significantly elevated rate of male breast cancer (SMR 7.41; 1.99–18.96) [43]. There appears to be some early evidence of convergence in risk between male and female breast cancer among firefighters, but the data to date are preliminary. There was also roughly a doubling of risk for female firefighters, but based only on a single case, because of the small number of women firefighters.

Kang found four cases in Massachusetts which resulted in an elevation in incidence for male breast cancer of 1.28 (1.00 being equivalent to the reference population) compared to all occupations reported in the Massachusetts cancer registry, and 0.25 compared to police [23]. This is entirely explicable by the rarity of the cancer and the very low likelihood of any one study demonstrating the true risk. The same is true for other etiological studies of cancer in firefighters, few of which examine male breast cancer separately. (Kang, in 2008, followed Ma's 2005 study, and their team was alerted to look for male breast cancer cases because Ma had already reported an association.)

The NIOSH Study found a non-significant elevation in mortality and incidence for male breast cancer (SMR 1.39; 0.60–2.73 with eight cases, SIR 1.26; 0.82–1.85, with 26 cases), with no data on subgroups [27].

The Nordic Study did not report on male breast cancer risk [28].

The Australian Firefighters' Health Study reported a high but non-significant elevation for full-time professional firefighters (2.49; 0.81–5.82), only, and no elevation among female firefighters [47].

A large study of male breast cancer incidence in the United States that was published in 2004 [121], early in the current debate on the efficacy of screening, determined that there is increasing frequency of detection of the disease, although not to the same degree as for women; it is more often detected at older age, after having already developed metastases, and at a larger tumor burden compared to women. However, for a given stage of the disease, survival was similar between male and female breast cancer. Because it is detected at a later stage, male breast cancer

overall, without adjusting for stage, has a poor prognosis, which makes mortality an accurate surrogate for incidence. This is important for the validity of statistical studies because it means that mortality (rate of death) is a reasonable surrogate measure for incidence (rate of new or newly detected disease) for male breast cancer and that elevated mortality tracks along with elevations in incidence in men. (It does not in women.)

It is assumed that male breast cancer and female breast cancer share some risk factors but not others. Female breast cancer is associated with nulliparity (not giving birth) and the absence of lactation, obesity (thought to be possibly due to diet and metabolic changes associated with it, but probably not obesity as such), and older age at giving first birth. These factors probably do not apply to male breast cancer. Female breast cancer risk is known to be associated with estrogen stimulation and is strongly suspected to be associated with exposure to chemicals that mimic the effects of estrogen. Female breast cancer risk is also associated with exposure of the breast to radiation. The male hormonal risk factors associated with breast cancer risk are those associated with increased estrogenic activity, such as testicular failure, gynecomastia, infertility, and significant alcohol abuse (presumably mediated through reduced clearance of estrogen by the liver). Female breast cancer is not associated with cigarette smoking, so there is no a priori reason to suspect that this would be a factor in male breast cancer either.

There are four plausible explanations that apply to general causation in male breastcancer.

The single most likely explanation is that firefighters are exposed regularly to products of combustion, including potent carcinogens listed in Chap. 5, that are relatively non-selective. In other words, they may cause cancer in bladder, kidney, or lung depending on where the chemical interacts with DNA, acting randomly at the cellular level in tissues. Men probably no different in the tissue susceptibility to these carcinogens but because there is very little breast tissue in men, cancers from exposures to carcinogens in that tissue are much less frequent.

The second-most likely explanation may be that because male breast cancer is estrogen sensitive and estrogen-receptor positive, and because men produce relatively little endogenous estrogen to compete with estrogen activity-mimicking toxic agents, breast tissue in men responds to stimulation from estrogen-like chemicals. (This is demonstrated periodically in outbreaks of adult gynecomastia, a disorder in which men grow excess breast tissue, caused by occupational exposure, such as one particularly well-studied case among pharmaceutical workers packaging birth control pills at a factory in Puerto Rico.) Among the combustion products to which firefighters are exposed are numerous polycyclic aromatic hydrocarbons and chlorinated polycyclic hydrocarbons (among them dioxins) that resemble estrogen structurally and are known to have some degree of estrogenic activity, although it is much less than estrogen itself.

Another plausible explanation is an effect of shiftwork, which is known to be a risk factor for female breast cancer and for which there is a well-developed, plausible explanation involving disruption of circadian rhythms and hormone cycles

that are associated with breast cancer. The net effect is to make breast tissue more susceptible to cancer initiation in the individual. This explanation has been accepted for female breast cancer by IARC. It would logically apply as well to male breast cancer [53].

The last somewhat plausible explanation is that electromagnetic fields induce cancer in firefighters surrounded by radio transmission and electronic equipment, a theory that has not gained wide support [9].

The weight of evidence suggests that a risk exists for male breastcancer but the evidence is not sufficiently developed to suggest a presumption based on empirical evidence.

## *Skin Cancer (Including Melanoma)*

Skin cancers are divided into non-melanoma (which includes basal cell and squamous cell carcinomas) and melanoma (for which the various cell types carry little practical significance for management). Melanoma is an uncommon but usually highly malignant cancer and can occur elsewhere in the body but is more common where there is sun exposure.

Firefighters are obviously exposed to carcinogenic chemicals present in fire smoke that are active in causing skincancer, chief among them PAHs, but are unlikely to be exposed to arsenic or ionizing radiation. Ultraviolet radiation from exposure to sunlight is probably the driving factor in firefighter risk, however. The role of chemical exposure is difficult to sort out mainly because of confounding from ultraviolet radiation.

By far the most plausible exposure responsible for elevated melanoma or other skincancer rates would be exposure to ultraviolet radiation in the form of sunlight outdoors, in which case one might expect that the elevation would be higher among wildfire fighters, who work outdoors for longer periods than urban firefighters. That does not seem to be the case, as reflected in studies of wildfire fighters alone, but the issue has not been separately addressed [122]. One might also expect turn-out gear to be protective against ultraviolet exposure, since it covers most of the body.

### Non-Melanoma Skin Cancer

Most non-melanoma skin cancers are nuisances but only rarely dangerous. Basal cell carcinoma is so common as to be almost a normal disease of aging and spreads locally in the skin. Squamous cell carcinoma is slightly more aggressive but rarely invades or metastasizes and only then in advanced stages when seriously neglected. Both are associated with ultraviolet radiation and therefore sun exposure and occur much more often in exposed areas on the body, in places with abundant sunshine, and in populations where skin color and type are susceptible to sun injury (burns easily). Dark-skinned people do get these forms of skin cancer but at much lower

rates than white-skinned people. They also occur disproportionately in families and individuals who have certain inherited conditions involving deficiencies DNA repair, of which the most serious are disabling genetic diseases. Firefighters who spend a great deal of time outdoors, including wildland firefighters, are obviously at risk. These common skin cancers are not tracked by cancer registries and so there are few reports on incidence and prevalence among firefighters. Certain chemical carcinogens (such as the polycyclic aromatic hydrocarbons, discussed in Chap. 5, and arsenic) and ionizing radiation also cause non-melanoma skin cancer.

The Nordic Study [28] reported elevations in non-melanoma skin cancer (1.33; 1.10–1.59, omitting Denmark), driven primarily by a significant elevation in Sweden (1.43; 1.12–1.79) and in older age groups and later periods of follow-up, peaking in the 1991–2005 period (1.39; 1.11–1.71). As well, a multicentre case–control study of occupation and non-melanoma skin cancer among Spanish firefighters found a large but not quite statistically significant elevation in risk for basal cell carcinoma among firefighters (RR 4.55, 0.96–21.57) [123].

### Melanoma

Melanoma is a skin cancer that is usually highly malignant (often metastatic or invasive when discovered) and is frequently fatal despite treatment. Melanoma shares with no-melanoma skin cancers various environmental risk factors, including vinyl chloride (which is present in fire smoke), PCBs, possibly solvents and arsenic. However, the most important shared risk factors of skin cancer involve electromagnetic radiation, both ionizing and, predominantly, ultraviolet radiation. While the other skin cancers show an exposure–frequency relationship that seems to be cumulative (reflecting total skin damage), melanoma appears to correlate more closely with discreet events of sunburn at a young age, suggesting that acute damage followed by healing contributes to risk. Here the major confounding factor is sun exposure, including sun-tanning behavior, time spent outdoors, and sunburn at a young age. No information is readily available on sun-related behavior of municipal firefighters compared to the general population.

Melanoma was identified as consistently elevated in the literature in the meta-analysis by Lemasters covering studies up to 2006 (summary risk 1.32; 1.10–1.57) [10]. Feuer (1986) demonstrated a significantly elevated risk of 2.70 against US white males and a elevated risk of PMR 1.90 against local, New Jersey white males [106]. Bates demonstrated a statistically significant elevation for melanoma among California firefighters (OR 1.50; 1.33–1.70) [124]. The NIOSH Study does not suggest an elevation for skin cancers (mortality from which would be associated with melanoma) [27]. The Nordic Study [28] reported a significant elevation in incidence for skin melanoma (1.25; 1.03–1.51), which appears to have been driven primarily by a significant elevation in Norway (1.61; 1.10–2.28) and a more modest elevation in Sweden that did not achieve significance (1.14; 0.85–1.50). Ide [31] found a significant elevation in incidence of melanoma in Strathclyde firefighters (RR 1.77, $p < 0.001$, based on six cases). At 15 %, the proportion of cases of melanoma among

all cancer cases also appears to be high, but this was not commented on. The Australian Firefighters' Health Study demonstrated a highly significant elevation in risk (SIR 1.45; 1.26–1.66) among full-time professional firefighters and a similar elevation in part-time firefighters, no elevation in male volunteer firefighters, and a high but non-significant elevation among female paid firefighters (2.10; 0.68–4.90) and a smaller but significant elevation among female volunteer firefighters (1.25; 1.05–1.46).

The highest risk for melanoma appears to be from episodes of acute sun damage occurring early in life and in young adulthood, a causal model that does not fit well with firefighting, but could fit with lifestyle factors. Much and probably most exposure to ultraviolet radiation occurs in recreational and other non-occupational settings, for firefighters as well as others. It is not clear that firefighters, as a group, take their vacations in sunny places, work outdoors, or otherwise behave in a manner that increases sun exposure relative more often than others in the population but this is certainly possible.

Thus, an association of melanoma with firefighting based on ultraviolet exposure would appear unlikely for municipal firefighters but likely for wildfire fighting where deployment is for prolonged periods, sun protection is not provided by turnout gear, and associated with camp living and outdoor work without protection. On the other hand, melanoma risk is empirically elevated among municipal firefighters in general and there are plausible associations with other risk factors, so the weight of evidence is probably close to even.

There is insufficient evidence to consider whether melanoma in organs other than skin is associated with firefighting; this is rare.

## *Other Cancer Types*

Elevations in risk have been found in other cancer types but so far without confirmation or replication. Except where noted, few studies have reported information needed to apply the appropriate analytical methods to these cancers. Rather than cite individual studies prior to 2006, therefore, the summary risk estimates of Lemasters are used here as a guide to the general trend of evidence [10].

*Esophageal cancer* was not elevated in the meta-analysis of Lemasters (2006) (summary risk 1.16; 0.86–1.57). Bates demonstrated a statistically significant elevation for esophageal cancer among California firefighters (OR 1.48; 1.14–1.91) [22]. It is difficult to identify a plausible exposure that could be responsible, although nitrosamines (more familiar as dietary risk factors) are formed by combustion. The NIOSH Study [27] demonstrated an excess risk of both mortality and incidence (SMR 1.39; 1.14–1.67, SIR 1.62; 1.31–2.00) with a marked difference between racial groups (Caucasians 1.46; 1.20–1.75, non-Caucasians 0.51; 0.11–1.49) and no duration-response relationship [27]. The Nordic Study [28] did not show an elevation in incidence (0.98; 0.66–1.39). The known risk factors for esophageal cancer

include esophageal reflux disease (resulting in Barrett's esophagus, a premalignant condition of the esophageal mucosa), alcohol intake, smoking, obesity, consumption of pickled vegetables (in Asia), consumption of scaldingly hot tea, and exposure to silica (well documented) and asbestos, the latter rather speculative. It is unlikely that these risk factors would be so prevalent among firefighters, so as to lead to an elevation in risk for this cancer as a group as high as a doubling of risk. Stronger associations are found for socio-economic status, location, and lifestyle than for occupation, in general. At present, the weight of evidence appears to favor an association but whether this association arises out of occupation cannot be adequately evaluated for firefighters.

*Laryngeal cancer* was not identified as elevated by Lemasters in the totality of the literature up to 2006 (summary risk 1.22; 0.87–1.70), but as expected for a treatable cancer incidence studies showed higher risks than mortality studies, although with wide confidence intervals and without achieving significance [10]. Firth [30] found an astronomical elevation in risk for cancer of the larynx (SIR% 1348; 254–3991) after adjustment for socioeconomic status but no similarly extreme finding has been reported in any other study. The NIOSH Study [27] found a substantial and significant increase in incidence but did not find an elevation in mortality overall, again as expected for a treatable cancer. Mortality was elevated but not significant overall, (1.26; 0.91–1.69) but did show a significant increase in Chicago (1.55; 1.01–2.27). Incidence, on the other hand, was elevated overall (1.50; 1.19–1.85) and across the board in the participating cities except San Francisco. The Nordic Study [28] did not show an increased risk (1.06; 0.72–1.50). The weight of evidence suggests that the risk of cancer of the larynx is elevated among firefighters, although it is not clear that the risk arises primarily out of work as a firefighter.

*Stomach* cancer, which is declining in frequency in the developed world, was demonstrated by Lemasters (2006) to show a significant elevation in the literature up to 2006 (summary risk 1.22; 1.04–1.44) [10]. An association with stomach cancer is plausible because significant amounts of material are mobilized from the respiratory tract in sputum and swallowed. Elevations. An elevation was observed individually in Beaumont (1.31; 0.82–1.99), which generally had anomalously low risk estimates for cancer [35]. The NIOSH Study [27] does not show an elevation for stomach in either mortality or incidence, overall or by city, Similarly, the Nordic Study [28] does not show an elevation in incidence overall or in any subgroup. The Australian Firefighters' Health Study shows no evidence of an increased risk for stomach cancer in its firefighter categories [47].

*Pancreatic* cancer was not significantly elevated in the meta-analysis of Lemasters (2006) but some, particularly early did not separately address this cancer [10]. Elevations with sufficient numbers to consider included Ma (1998), who found a relatively small but significantly elevated risk for white firefighters (MOR 1.2; 1.0–1.5) and a doubling of risk for black firefighters (2.0; 0.9–4.6), which did not achieve statistical significance with small numbers [16]. The NIOSH Study [27] showed no elevation overall or in any subgroup reported. The Nordic Study [28] showed a small and non-significant elevation (1.17; 0.94–1.45) that was not analyzed further.

The Australian Firefighters' Health Study showed no evidence of an increased risk for stomach cancer in its firefighter categories [47]. The weight of evidence would seem to suggest that cancer of the pancreas is not elevated among firefighters, but the evidentiary base is not definitive.

Cancer of the *small intestine* is very rare, and when it occurs is almost always an adenocarcinoma. Interest was drawn to this site by the demonstration of a non-significant elevation among Korean firefighters by Ahn [25] (SIR 2.46; 0.76–5.75, five cases) with no cases among non-firefighters in the same departments. The NIOSH Study (Daniels 2013) demonstrated an elevated but non-significant overall risk for both mortality (1.66; 0.72–3.27, based on only 8 cases) and incidence (1.15; 0.67–1.85, based on only 17 cases). The Nordic Study showed a small, non-significant elevation (1.15; 0.61–1.97, based on 13 cases). In the absence of a persuasive signal from the literature, one must examine associations to see if they might be relevant to firefighting. Known risk factors for this type of cancer include genetic disorders (including celiac disease) and conditions of chronic inflammation of the gastrointestinal tract (such as Crohn's disease), diet (red meat and foods that predispose to nitrosamine formation), and possibly smoking (studies vary). This hazard profile does not suggest an elevated risk for firefighters. At present the weight of evidence is not clear.

*Other gastrointestinal or digestive tract* cancers have been aggregated in most studies and so organ-specific rates for sites of interest (principally liver and biliary tract) generally cannot be separated. Those studies that have separately identified hepatobiliary cancers (Ahn [25], Baris [19]) show no elevation, with the highest risk among them reported by the NIOSH Study (1.30; 1.06–1.57), driven primarily by an excesses in Chicago (1.51; 1.15–1.95), which contributed more than half the cases. The Nordic Study separated liver and gallbladder and showed no elevation for primary liver cancer but an elevation for cancer of the gallbladder (1.45; 0.86–2.29). The Australian Firefighters' Health Study shows deficits in incidence of liver cancer in its firefighter categories [47]. The known risk factors for these diseases do not suggest an elevated risk for firefighters. At present the weight of evidence is not clear.

## Appendix

Summary of Health Risks Associated with Occupation as a Firefighter Reported in Large Population Studies

**Selected Cancer Risk Estimates from Major Studies of Firefighters. (SMRs originally given as percentages have been converted to decimals.) See text for discussion**

| Authors, year | Population | Design | Risk estimate | Lung | Colon | Rectum | Bladder | Kidney | Brain | CVD | COPD |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Ahn, 2012 [25] | Seoul, Korea | Cohort | SIR | 0.78 | 1.27 | | 1.60 | 1.56 | 0.53 | | |
| | | | | 0.55–1.09 | 1.01–1.59 | | 1.01–2.56 | 1.01–2.41 | 0.14–1.36 | | |
| Baris et al., 2001 [19] | Philadelphia FD | Cohort | SMR | 1.13 | 1.51 | 0.99 | 1.25 | 1.07 | 0.61 | 1.01 | 0.64 |
| | | | | 0.97–1.32 | 1.18–1.93 | 0.59–1.68 | 0.77–2.00 | 0.81–1.88 | 0.31–1.22 | 0.96–1.07 | 0.40–1.02 |
| Bates, 2007 [22] | California | Cancer registry | OR | 0.92 | 0.84 | | 0.79 | 0.98 | 1.23 | n/a | n/a |
| | | | | 0.84–1.01 | 0.74–0.94 | | 0.68–0.92 | 0.81–1.20 | 0.97–1.56 | | |
| Beaumont et al., 1991 [35] | San Francisco FD | Cohort | SMR | 0.84 | 0.99 | 1.45 | 0.57 | 0.68 | 0.81 | 0.95 | 0.75 |
| | | | | 0.64–1.08 | 0.63–1.47 | 0.77–2.49 | 0.19–1.35 | 0.19–1.74 | 0.26–1.90 | 0.87–1.04 | 0.43–1.23 |
| Burnett et al., 1994 [14] | 27 U.S. States | Surveillance | PMR | 1.02 | n/a | 1.48 | 0.99 | 1.44 | 1.03 | 1.01 | 0.83 |
| | | | | 0.94–1.11 | | 1.05–2.05 | 0.70–1.37 | 1.08–1.89 | 0.73–1.41 | 0.97–1.05 | 0.73–0.94 |
| Daniels, et al. [NIOSH], 2013 [27] | 3 US Cities Chicago, Philadelphia, San Francisco pooled | Cohort | SMR | 1.10 | 1.31 | 1.45 | 0.99 | 1.29 | 1.01 | | 0.73 |
| | | | | 1.04–1.17 | 1.16–1.48 | 1.16–1.78 | 0.79–1.27 | 1.05–1.58 | 0.79–1.27 | | 0.65–0.81 |
| | | | SIR | 1.12 | 1.21 | 1.11 | 1.12 | 1.27 | 1.02 | | |
| | | | | 1.04–1.21 | 1.09–1.33 | 0.95–1.30 | 1.00–1.25 | 1.09–1.48 | 0.76–1.34 | | |
| Demers, Heyer, Rosenstock, 1992 [82] | Pacific Northwest Municipal FDs | Cohort | IDR v. Police: | 0.95 | 158 | 0.89 | 0.16 | n/a | 163 | 0.88 | 0.89 |
| | | | | 0.67–1.33 | 0.73–3.43 | 0.30–2.66 | 0.02–1.24 | | 0.70–3.79 | 0.74–1.04 | 0.47–1.69 |

(continued)

(continued)

| Authors, year | Population | Design | Risk estimate | Lung | Colon | Rectum | Bladder | Kidney | Brain | CVD | COPD |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Deschamp et al., 1995 [15] | Paris France | Cohort | SMR | 1.12 | | | | | | 0.74 | 1.83 |
| | | | | 0.45–2.30 | | | | | | 0.20–1.90 | 0.05–10.21 |
| Dubrow and Wegman, 1984 [125] | Massachusetts firefighters, 1971–73 | Surveillance | SMOR on mortality | 0.86 | 0.76 | 0.60 | 1.10 | 1.22 | 0.86 | n/a | n/a |
| Eliopulos et al., 1984 [122] | Western Australia. Wild fire fighters | Cohort | SMR | 1.04 | 1.59 | n/a | 1.08 | n/a | 0.84 | 0.65 | |
| | | | | 0.42–2.13 | 0.43–4.07 | | 0.29–2.76 | | 0.60–1.14 | 0.13–1.90 | |
| Feuer and Roseman, 1986 [106] | New Jersey | Cohort | PMR, NJ state comparison | 0.92 | n/a | n/a | n/a | n/a | n/a | 1.11 | n/a |
| Giles et al., 1993 [13] | Melbourne FD, Australia | Cohort | SIR | 0.77 | 1.36 | 1.02 | n/a | n/a | n/a | | |
| | | | | 0.28–1.68 | 0.62–2.59 | 0.28–1.68 | | | | | |
| Glass et al., [47] | Australian FF Health Study | Cohort | SIR, SMR: here SIR for cancer SMR for non-malignant disease | 0.81 | 1,13 | 1.18 | 0.85 | 0.97 | 0.76 | 0.64 | 0.61 |
| | | | | 0.65–1.00 | 0.91–1.38 | 0.89–1.54 | 0.54–1.27 | 0.67–1.36 | 0.44–1.24 | 0.55–0.73 | 0.39–0.93 |
| Guidotti, 1992 [44] | Municipal firefighters, Alberta | Cohort | SMR | 1.42 | 1.61 | 3.16 | 4.14 | 1.47 | 1.10 | 1.57 | |
| | | | | 0.91–2.11 | 0.88–2.71 | 0.86–8.08 | 1.66–8.53 | 0.30–4.29 | 0.92–1.31 | 0.79–2.81 | |
| Guralnick, 1963 [126] | U.S. census, 1950 | Surveillance | SMR to 65y | 0.83 | 0.93 | n/a | n/a | n/a | 0.72 | n/a | |
| Hansen, 1990 [115] | Danish census | Cohort from census sample | SMR, v. civil servants | 1.63 | | | | | | 1.15 | n/a |
| | | | | 0.75–3.10 | n/a | n/a | n/a | n/a | n/a | 0.74–1.71 | |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Heyer et al., 1990 [83] | Seattle FD | Cohort | SMR | | 0.97 | 0.79 | 0.65 | n/a | n/a | 0.95 | 0.75 | n/a |
| | | | | | 0.65–1.39 | 0.32–1.64 | 0.08–2.37 | | | 0.20–2.79 | 0.63–0.89 | |
| Kang et al., 2008 [23] | Massachusetts, cancer registry, follow-up to Sama, 1990 | Surveillance | SMOR | All other | 0.91 | 1.15 | 1.03 | 1.19 | 1.01 | 1.38 | | |
| | | | | | 0.76–1.10 | 0.93–1.43 | 0.77–1.38 | 0.93–1.52 | 0.74–1.38 | 0.87–2.12 | | |
| | | | | v. MA police | 1.02 | 1.36 | 0.86 | 1.22 | 1.34 | 1.90 | | |
| | | | | | 0.79–1.31 | 1.04–1.79 | 0.58–1.26 | 0.89–1.69 | 0.90–2.01 | 1.10–3.26 | | |
| Kizer et al., 1987 [127] | California, adjusted for smoking, alcohol, SES | Surveillance | SMR Adjusted | | 1.32 | 1.31 | n/a | 1.15 | n/a | 1.26 | 0.57 | n/a |
| | | | | | 0.89–1.90 | 0.48–2.84 | | 0.24–3.37 | | 0.99–1.58 | 0.07–2.06 | |
| Lindsay et al., 1993 [128] | Canadian labour force sample | Cohort from census sample | SMR v. employed | | 1.11 | 1.54 | n/a | n/a | n/a | 0 | n/a | n/a |
| | | | | | 0.41–2.42 | 0.19–5.56 | | | | 0.00–6.25 | | |
| Ma et al., 1998 [16] white | 24 US states | Surveillance | MOR | | 1.1 | 1.0 | 1.1 | 1.2 | 1.3 | 1.0 | n/a | n/a |
| | | | | | 1.0–1.2 | 0.9–0.2 | 0.8–1.6 | 0.9–1.6 | 1.0–1.7 | 0.8–1.4 | | |
| Ma et al., 1998 [16] African–American | 24 US states | Surveillance | MOR | | 0.8 | 2.1 | 0 | 1.2 | 1.3 | 1.0 | n/a | n/a |
| | | | | | 0.5–1.3 | 1.1–4.0 | | 0.9–1.6 | 1.0–1.7 | 0.8–1.4 | | |
| Ma et al., 2005 [43] | Florida certification registry | Cohort | SIR | | 0.65 | 1.16 | 0.88 | 1.29 | 0.78 | 0.58 | n/a | n/a |
| | | | | | 0.54–0.78 | 0.92–1.45 | 0.56–1.32 | 1.01–1.62 | 0.52–1.14 | 0.31–0.97 | | |
| Mastromatteo, 1959 [5] | Toronto FD | Cohort | SMR | | n/a | n/a | n/a | n/a | n/a | n/a | | n/a |

(continued)

(continued)

| Authors, year | Population | Design | Risk estimate | Lung | Colon | Rectum | Bladder | Kidney | Brain | CVD | COPD |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Massachusetts Dept. of Public Health, 1990 | Massachusetts resident firefighters | Surveillance incidence | SMOR State: | 1.22 | 1.20 | 1.45 | 1.59 | n/a | 0.86 | n/a | n/a |
| | | | | 0.87–169 | 0.80–182 | 0.84–219 | 1.02–2.50 | | 0.34–2.15 | | |
| Milham, 1983 | Washington state residents | Surveillance | PMR | 1.05 | 0.90 | 1.03 | 2.33 | 1.15 | 1.94 | 1.16 | 0.53 |
| Musk et al., 1978 [129] | Boston FD | Cohort | SMR | 0.88 | n/a | n/a | 0.92 | 1.03 | 0.86 | n/a | n/a |
| Petersen and Milham, 1977 [133] | California state residents | Surveillance | PMR | 1.68 | 1.13 | 0.48 | 0.82 | 1.75 | 0.90 | 1.08 | [70] |
| Pukkala et al., 2014 [28] | Nordic countries, identified in 1970 | Cohort | SIR | 0.97 | 1.14 | 0.99 | 1,11 | 0.94 | 0.86 | | |
| | | | | 0.87–1.09 | 0.99–1.31 | 0.82–1.19 | 0.96–1.28 | 0.75–1.17 | 0.66–1.10 | | |
| Rosenstock et al., 1990 [130] | Pacific Northwest | Cohort | SMR v. US: | 1.34 | 0.95 | 1.19 | 0.23 | 0.27 | 2.10 | 0.82 | 1.19 |
| | | | | 0.90–1.91 | 0.41–1.87 | 0.44–2.59 | 0.03–0.83 | 0.03–0.87 | 1.31–3.17 | 0.74–0.90 | 0.72–1.83 |
| Sama et al., 1990 [24, 131] | Massachusetts, cancer registry | Surveillance | SMOR v police | 1.30 | 1.04 | 0.97 | 2.11 | n/a | 1.52 | n/a | n/a |
| | | | | 0.84–2.03 | 0.59–1.82 | 0.50–1.88 | 1.07–4.14 | | 0.39–5.92 | | |
| Tornling et al., 1990 [132] | Stockholm FD | Cohort | SMR | 0.90 | 0.85 | 2.07 | 0.31 | 1.10 | 2.79 | 0.98 | 0.00 |
| | | | | 0.53–1.42 | 0.31–1.85 | 0.89–4.08 | 0.01–1.70 | 0.30–2.81 | 0.91–6.51 | 0.81–1.17 | 0.00–0.48 |
| Vena and Fiedler, 1987 [116] | Buffalo FC | Cohort | SMR | 0.94 | 1.83 | 2.08 | 2.86 | 1.30 | 236 | 0.92 | n/a |
| | | | | 0.62–1.36 | 1.05–2.97 | 0.83–4.28 | 130–5.40 | 0.26–3.80 | 0.86–5.13 | 0.81–1.04 | |

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# Chapter 7
# Systemic Disorders and Other Medical Conditions

**Tee L. Guidotti**

The literature on firefighters is less helpful, overall, for in describing risk for health outcomes beyond cancer, heart disease, and respiratory disease. Evidence is usually fragmentary, in part because these diseases are seldom tracked through registries. The major exception is sarcoidosis, which has been closely scrutinized but for which the connection with firefighting remains unclear at the time of this writing. Certain infectious diseases are reportable to public health agencies, but these are seldom reported by occupation. For example [1, 2], hepatitis C is an infection of interest and is reportable in 49 states but this information is not usually reported by occupation. Those diseases that are not reportable or tracked by a registry must therefore be evaluated through death certificates. Mortality is an imperfect way of tracking the few disorders mentioned in this chapter that are generally fatal and useless for those that are not. Even those few that are likely to lead to death often show a bias in ascertaining cases due to access to medical care or systematic misclassification, especially for diseases that may be related to alcohol or drug abuse.

Sources for nonmalignant, non registry-recorded health conditions include some of the same studies listed in the Appendix. The most recent studies to include extensive non-malignant health outcomes are the supplementary tables for the NIOSH Study (2013) [3] and the Australian firefighters study (2014), which showed no elevations for non-malignant outcomes [4]. Because few studies do show elevations and those that do not are subject to bias, as described above, and under-reporting, this chapter will not present "negative" studies, those that do not show an elevation.

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## Immune Disorders, Including Sarcoidosis

There is essentially no literature on immune outcomes among firefighters except for the disease sarcoidosis.

Sarcoidosis is a disorder of the immune system, of unknown cause but highly-specific pathology, characterized by the formation of structured aggregates of inflammatory cells, called granulomas, consisting of "giant cells", which are derived from the scavenger cells of the body. In the lung, these granulomas appear in the interstitium, which is the connective tissue between the air space and the blood space. Sarcoidosis can also appear in other organs and can cause liver disease, eye inflammation, and an arthritis, often associated with a characteristic skin rash of the legs.

Scientists have been trying to find out what causes sarcoidosis for about 140 years with essentially no success. The only external agent known to produce the sarcoid reaction is an extract of the lymph glands of a person with sarcoidosis (the so-called Kveim antigen), which is not a particularly helpful observation because it cannot rule out infection, immune response, or toxic accumulation and cannot possibly be a mode of transmission. The granulomatous reaction mimics the characteristic response to a persistent antigen in the lung and other organs, although no such antigen has been identified as the cause of the disease despite years of searching. In many ways, sarcoidosis resembles tuberculosis, but no mycobacteria (the class of bacteria that includes tuberculosis) or other infectious agent, for that matter, has ever been found, despite intensive search. The disease most closely resembling sarcoidosis is beryllium disease, caused by exposure to beryllium in the workplace. This has led to extensive research seeking hidden beryllium-exposure in the workplace or aberrant reactions to more common metals in the environment that could produce a disease like sarcoidosis. This search has proven fruitless after over a century, and many decades during which hundreds (possibly thousands) of investigators have applied the most advanced methods to the problem.

Sarcoidosis, or at least interstitial lung disease due to sarcoid-like granulomatous inflammation, has been reported to be in excess among firefighters. This presents an interpretive problem. Sarcoidosis is difficult to recognize and requires a biopsy for diagnosis. This means that it is most likely to be recognized and diagnosed in populations with good medical care who are under close scrutiny. People who are not in a surveillance program are likely to have their sarcoidosis missed or diagnosed late. This means that there is a bias in comparing such populations to the general population or to other groups when health care access is different between them. Unfortunately, this has been a common situation in the United States, where health care is tied so closely to employment and the quality of care is linked to occupation. It is not clear whether firefighters have an excess of sarcoidosis or whether their rate of the disease is the true baseline and it is under-diagnosed in "everyone else".

Sarcoidosis has been demonstrated to be more common than expected among firefighters, with a point prevalence of 222/100,000 among over 11,000 New York City firefighters compared to none observed among 3000 single-assignment emergency medical services personnel [5]. This systematic investigation was based on a registry of cases established after the identification of a cluster of sarcoidosis

cases among New York firefighters in 1979 [6]. Because sarcoidosis is not a reportable disease and is rarely fatal, comparison or baseline incidence figures from other populations were not available.

The event-specific exposures experienced by WTC responders were qualitatively different from but were in addition to the conventional exposures of professional firefighters. Given this, any discrepancy in the rates of sarcoidosis between the two groups could provide a clue to the etiology of this puzzling disease. Sarcoidosis has also been observed in a number of WTC responders. Sarcoidosis, affecting approximately one hundred individuals, has been consistently reported to be elevated in prevalence by all cohorts within the different programs that follow WTC-exposed individuals [7, 8].

In the first 5 years post- 9/11, pathologic evidence consistent with new-onset sarcoidosis (or a sarcoid-like granulomatous lung disease) was found in 26 NYFD rescue workers, all with intra-thoracic adenopathy and 6 (23 %) with additional disease outside the chest [9, 10]. Thirteen were identified during the first year post-WTC (yielding an incidence rate of 86/100,000) and 13 during the next 4 years (yielding an average annual incidence rate of 22/100,000; as compared to 15/100,000 for NYFD personnel during the 15 years pre-WTC and 5–7/100,000 for a male Caucasian population). On the other hand, only 3 of the 26 cases had evidence of restriction, decreased diffusion, or both, and these studies do not account adequately for reporting and detection biases inherent to the compensation and disability claims post-9/11. Similar findings were suggested by studies in 2 other cohorts, The WTC Registry and the New York/New Jersey (NY/NJ) WTC Responders [9, 11]. The three studies each have surveillance and detection biases that make comparisons among them, and to non-local referent populations, difficult but the consistency of their findings is impressive. The component(s) of WTC dust responsible for this granulomatous reaction remains unknown as WTC dust has not been shown to contain substances known to produce granulomatous or giant cell reactions, such as beryllium, zirconium or tungsten.

## Neurological and Sensory Disorders

Very broadly, neurological disorders can be distinguished as diseases intrinsic to the nervous system and those secondary to the nervous system that arise from vascular insufficiency, which, with one exception, are much more common. That exception is noise-induced hearing loss (NIHL), a sensorineural disorder that is among the most common occupational diseases.

Mortality data is the usual way to evaluate other neurological disorders in populations, and that means that almost all cohort studies are dominated by stroke, the risk for which happens to be reduced among firefighters. Unless developed for a specific study in the community, neurological disorders are not entered into registries and, with the exception of NIHL, are not tracked for surveillance purposes with incidence data.

Intrinsic neurological conditions, such as degenerative neurological diseases of the brain, spinal cord, autonomic nervous system, or neuromuscular junction are much less common than stroke, which is caused by disorders of the blood vessels and clotting mechanism. Most degenerative neurological diseases occur relatively early in life, except for Alzheimer's diseases and other forms of dementia, which mostly occur after retirement and are usually not direct causes of death.

The rigorous selection and retention bias for fitness among firefighters ensures a strong healthy worker effect, in that firefighters who show early signs of neurological or neuromuscular disease (such as loss of strength, loss of coordination, seizures, cognitive disorders) are unlikely to be hired and unlikely to stay on the job once the symptoms present themselves. It is therefore not surprising that neurological disease is an uncommon cause of death among firefighters and so rates are lower among firefighters than in the reference populations.

Among the extant occupational cohort studies of firefighters, most show very low mortality from neurological diseases. Most of the firefighter cohort studies, especially the older and smaller ones, do not even report neurological diseases as a separate category. Among those that do [12, 13], the risk ratio is at or close to about 0.75, although most of these studies have wide confidence intervals due to the small number of cases, ranging down to 0.56 (Tornling) and a statistically significant 0.47 (0.27–0.83) [14].

Strokes (cerebrovascular accidents) are neurological conditions, of course, but on a population basis they are primarily the result of vascular disease. The risk factors for stroke are the same as for other cardiovascular diseases, and so stroke is considered in Chap. 8.

## *Motor Neuron Disease*

Motor neuron disease is a general rubric for a collection of relatively rare disorders, of which the most common is amyotrophic lateral sclerosis (ALS), a progressive disease that occurs in adulthood, preserves cognitive function and awareness, and typically results in a relentlessly advancing weakness until the muscles of swallowing and respiration no longer function, at which point the disease is always fatal due to pneumonia or respiratory failure.

Using mortality data (which is only reliable for some neurological disorders) derived from the National Occupational Mortality Surveillance System from 1982 to 1991, NIOSH investigators found an excess of deaths (PMR% 318, no confidence interval reported, with six deaths; population of firefighters not reported) for firefighters from motor neuron disease, but not other neurodegenerative disorders. However, no further information is available and, oddly, firefighters are not listed in an accompanying table of highest-ranked occupations at risk of motor neuron disease by race and sex, although athletes, with similar characteristics and a lower risk, were entered. This study design is usually considered insensitive, demon-

strated an excess of death from motor neuron disease for firefighters, along with other occupations, but in this case the study overcame many of its intrinsic statistical limitations by compiling large numbers. There were many occupations with higher and more stable risk estimates, including veterinarians, several of which had astronomical risk estimates based on small numbers. The study suggested that neurodegenerative disorders, in general, might have a link to solvent exposure (some solvent chemicals also being present in fire smoke) but sedentary and knowledge workers also showed elevations [15].

The study was repeated by NIOSH approximately 10 years later on mortality data from 1992 to 1998 using the same general methods, by the same group. However, firefighters were inexplicably omitted, although the patterns for solvent-exposed workers and knowledge worker remained [16]. Thus, the later study cannot be taken as a replication, since it appears that, for whatever reason, public safety workers were not studied.

However, a contemporary study of occupational associations of AML conducted in Italy showed a doubling of risk for firefighters (OR 2.0; 1.2–3.2) [17]. The study was conducted not to identify a toxic exposure but to test the authors' hypothesis that tissue hypoxia is a risk factor for the disease. The authors hypothesized that ALS may occur in response to hypoxia in individuals with a genetic susceptibility.

Given that the first study was relatively weak and showed a finding that could be explained by chance alone due to multiple comparisons, the epidemiological evidence for an association between firefighting and motor neuron disease may be considered unpersuasive at this time. On the other hand, two studies have found an association with firefighting at a risk estimate that is not only significant (in one and probably the other) but sufficiently high ($\geq 2$) to suggest a presumption in the individual case, which is a higher standard than association alone. This cannot be ignored and so in the absence of evidence to the contrary or a reason to believe that there was substantial confounding, the empirical result has to be accepted as the weight of evidence.

There have been numerous other attempts to identify occupational associations with motor neuron disease. Most have suggested an association with rural life and possibly farming, solvents (including hexane, although this may just be a surrogate exposure), pesticides, electrical shocks and exposure to strong electromagnetic fields. Recent studies have focused on cigarette smoking and formaldehyde exposure, which could be consistent with a risk for exposure to combustion products but is not strongly suggestive.

## *Neurosensory Disorders*

The common neurosensory disorder of firefighters, as with most occupations, is noise-induced hearing loss (NIHL).

## Vision

Firefighters are screened on hire for good vision. Visual impairment occurring due to occupational risk factors is not addressed in the literature but since this is monitored with regular physical examinations, the omission probably reflects lack of a problem rather than lack of vigilance in surveillance.

## Hearing

For many years, the literature did not reflect the true dimensions of the problem of NIHL among firefighters. NIHL is primary a disorder of cumulative or repetitive trauma to the auditory hair cells in the cochlea, the organ of hearing. Individual hair cells are "tuned" to particular frequencies. Loud noise damages the hair cells tuned to the corresponding frequency. Certain frequencies, particularly at and around 4000 Hz (a Hertz is a "cycle per second") are important in comprehending speech but are particularly vulnerable both biologically and physically because of the sound frequencies commonly encountered in the workplace. In some cases, NIHL is a mixed condition because it is also associated with atherosclerosis and smoking (because of vascular disease affecting the blood supply to the inner ear) and solvent exposure, which is related to direct neurotoxicity of the ciliated hearing cells. However, the necessary hazard that must be present for NIHL to occur is loud noise.

Noise exposure is a common and recognized hazard in firefighting, but the literature on noise-induced hearing loss is scanty, largely obsolete, and rudimentary. Contemporary equipment is much less noisy than in the past but few recent noise surveys have been published for the fire service. Major sources of noise include vehicles while in transit with the warning siren blaring and water exiting the hose at high pressure. Because of communication and warning requirements and the adverse conditions of hot and often steamy conditions, it is not generally feasible to wear hearingprotection while actively engaged in firefighting, although it may be possible while in transit.

Exposures to noise levels in excess of 100 dBA (a very uncomfortably high level of noise) for very short periods may occur while in transit with the siren on, yet these levels may still conform to both the "ceiling" (peak) and the the 8-h time-weighted average occupational exposure limits (the national occupational noise standard in Australia is 85 dBA 8-h averaged daily exposure with a 140 dBC permissible peak level) [18].

Sound levels within fire trucks, which may reach 110 dBA, have long been said to comply with American occupational health regulations and hearing thresholds for firefighters have been reported to be comparable to workers not exposed to noise within the cabin of fire trucks [19]. However, this is misleading. The US OSHA noise standard is 90 dBA 8-h TWA, 115 dB peak continuous noise and 140 impulse and compliance with the standard is achieved chiefly as a function of short duration. Peak noise levels may be an independent risk factor for NIHL and certainly is when it begins to approach impulse noise levels. The OSHA PEL for noise is also well known to be inadequate in providing protection, in that it is not fully protective against hearing loss, even for ears that do not have a biological susceptibility or preexisting disease.

It has also been suggested that firefighters are not at risk for NIHL and show no decrement consistent with occupational loss of hearing on serial audiometry [19, 20]. Further, studies that showed lower noise exposure, except during emergency response, still demonstrate that a substantial fraction of firefighters had NIHL [21]. This literature left the impression that NIHL among firefighters was not likely to be work-related. This conclusion no longer stands.

More definitive recent studies demonstrate clearly that currently a high percentage of municipal firefighters (40 % in San Francisco in the 1990s) do have NIHL, that the frequency and severity is associated with duration of service as a firefighter, occurs more frequently in the left ear, may occur early in the career of a firefighter, and that hearingprotection was effective but only used by 34 % of firefighters [22–24]. Loss of hearing follows a trend of accelerated loss over age that is particularly pronounced in cases in which there has already been significant loss. This means that damaged ears are more susceptible to further damage [25].

Hearing conservation programs for firefighters are not universal but when voluntary programs have been introduced compliance has appeared to be good with support and incentives [26–29]. On the other hand, outcome data are not available from current demonstration programs.

On the face of it, noise-induced hearing loss is an occupational risk of firefighting and one that can be mitigated with prevention of unnecessary exposure to noise. Noise-induced hearing loss is reported to be more frequent at earlier ages among firefighters than in the general population and to be more common in the left ear, which in North America is the ear facing the window on the driver's side [30].

## Genitourinary System

There is no suggestion in the literature for kidney or urinary tract conditions being associated with firefighting, except the cancers, as previously noted. The NIOSH Study did show a significantly elevated risk of death from acute glomerulonephritis (a kidney disease; SMR 1.56; 1.07–2.20), for which there is no obvious connection with firefighting, but not for individual cities and not for a long list of other genitourinary conditions [3]. This isolated finding probably represents an anomaly arising from multiple comparisons but deserves watching because no other extant study provides data down to this level of detail for kidney disease.

## Reproduction

There has been concern for some time on the potential reproductive hazards associated with fire smoke and inhaled contaminants, especially for women firefighters [31, 32]. There is contradictory and probably insufficient evidence to suggest congenital defects among offspring of firefighters.

Most attention has focused on chemical hazards for both male and female firefighters. As a practical matter, however, the primary focus has been on effects on the male side because there have been so few female firefighters until recently. For occupation to have an effect on the male side, it must be assumed that there is a genotoxic or epigenetic effect that is hereditable, rather than in utero effects. This considerably narrows the range of plausible congenital anomalies.

However, the hazard of greatest theoretical concern for reproduction in female firefighters has actually received scant attention: heat. Hyperthermia is known to be highly fetotoxic and is associated with severe congenital defects in experimental studies. Human beings have the capacity to control core temperature across a wide range of environmental temperatures, so the effects of external heat should be mitigated up to the level of physiological capacity. However, pregnancy reduces a woman's capacity to maintain stable core temperature, to an unknown and probably variable degree (experimental studies not being possible) and whatever risk there may be would be worse with dehydration. Although 2.1 % of fire scene injuries (in 1980) are reported to be due to heat exhaustion and some number of those are likely to represent incipient hyperthermia, the risk to the offspring of pregnant firefighters has still not been fully assessed [32].

An "exploratory" case–control study in British Columbia found a markedly and highly significantly elevated risk (up to OR 5.9; 1.60–21.83, for ventricular and also atrial septal defects compared to the general population) for two types of heart defects in children of firefighters, as compared against the general population and against police [33]. However, this dramatic finding has not been replicated. A case–control study performed on Toronto firefighters between 1979 and 1986 in order to confirm this finding demonstrated a much lower and non-significant odds ratio (1.22; 0.46–3.33) for the same categories of congenital anomalies [34]. Similarly, contemporaneous data from a birth outcomes registry in Sweden did not show an effect, either overall or for the specific heart defects reported in the original study [35].

The finding has carried over into the general literature on birth defects [36] without mention of the negative studies that followed. A comparison with other occupations involving exposureto solvent chemicals, such as painters, shows that the putative risk for the specific congenital defects were not shared, since solvent-exposed workers tended to show spina bifida and patent ductus arteriosus (which is a large vessel defect distinct from congenital cardiac defects) rather than the septal defects reported by Olshan et al. [33].

Likewise, a study based on the congenital defects registry of Atlanta found no excess risk for cardiac defects. On the other hand, firefighters were disproportionately and markedly represented among the fathers of children born with cleft lip and palate (OR 13.3; 4.0–44.4, but based on only four cases), heart anomalies (4.7; 1.2–17.8, based on three cases) other than ventricular septal defect (0.7; 0.1–5.3, based on a single case), clubfoot (2.9; 1.4–6.0, 13 cases), and hypospadias (2.6; 1.1–6.3, 8 cases), with non-significant elevation for hydrocephalus [37]. However, these

particular congenital anomalies do not go together logically and do not fit the timing of the common mechanisms of birth defects. The window of vulnerability for most of them occurs during the first trimester in utero, not before conception, and clubfoot and hypospadias are fetal, not an embryonic phenomenon. Thus, the plausible associations for firefighters will probably require much more study and more robust numbers before it can be assumed that the children of firefighters are at risk.

The absence of replication, combined with the absence of reports from congenital defects registries and other research centers that have a robust interest in associations with congenital heart disease, suggest that the findings for heart defects were at least not generalizable and cannot be considered definitive evidence for a risk for offspring of firefighters, but there are sufficient grounds for concern to monitor birth outcomes more closely and to conduct further studies.

## Skin Disorders (Nonmalignant)

Obviously firefighters may develop skin diseases like everyone else. Occupational exposures associated with skin cancer include ultraviolet radiation associated with work out of doors, and, potentially, PAHs and irritating chemicals in skin contact. These same exposures may cause other, nonmalignant skin disorders.

No references were found specific to dermatitis or skin conditions in firefighters. Except in the case of systemic diseases with cutaneous manifestations, including autoimmune disease with dermal presentations, skin diseases are rare as causes of death. Registries of dermatopathology are common but reports of nonmalignant skin diseases are rare and almost never studied for occupational associations.

Burn injuries are, not surprisingly, the occupational injury category characteristic of firefighters among public safety professions. In a series of 982 cases admitted to New York Presbyterian Hospital between 1992 and 2002, frequency of serious burns fell abruptly early in the decade, reflecting regional trends, and then continued more or less steady for the last 7 years, with some variation. Relative to serving as an officer (10 %), burn injuries were more often associated with search and rescue (16 %), and with nozzle and back-up positions (50 %), where holding the hose and being exposed to return spray may expose the firefighter to scalding hot water. It is perhaps reassuring with respect to professional competence that very few firefighters received burns from cooking at the fire hall (3 %). However, this database did not report injury rates, only proportions and only covers burns that were severe enough to require admission; it also did not evaluate fatalities separately (there were three in hospital). The most common anatomic parts burned were the lower extremities (37 %), especially among nozzlemen, and the head and neck (25 %) but not the feet (1 %), presumably because of protective clothing [38]. Reflecting a lower level of severity, burns treated in an outpatient clinic at the same institution

from 2000 through 2002 showed a similar pattern, except for fewer burns to the lower extremities (because "nozzleman burns", being scalds, tend to be severe and require usually hospital admission).

## Infectious Diseases

Infectious disease risk for firefighters has centered on blood-borne and respirable pathogens that can be transmitted from patient to first responder [39]. This literature will not be reviewed in detail but representative papers will be cited to support the points made. The diseases of chief concern are hepatitis B, hepatitis C, HIV/AIDS, tuberculosis, and MRSA.

Hepatitis B was long considered the single greatest hazard for emergency response personnel. Hepatitis B virus is easily transmitted by multiple routes and is one of the principal targets of universal precautions. There appears to be no evidence for a significant elevation in hepatitis B infection among firefighters up to the last decade [40]. This is not surprising, in that the disease is much dreaded and effective measures for self-protection are well established. Among civilian firefighters, persons who are positive for hepatitis B generally have at least one non-occupational risk factor, implying that infection is unlikely to arise from work. Occupational risk has probably dropped further since then due to increased adherence to universal precautions for hepatitis B infection than the general population, being young, screened, and to some extent supervised.

Hepatitis C infection, similarly, was not elevated among first responders overall but was correlated with needlestick risk, older age, and exposure to high-risk populations [1, 41], implying that some cases did arise from occupation but at a low rate. Hepatitis C is more often associated with intravenous drug use than even hepatitis B and is therefore less likely to be prevalent in an active-duty military population or a public safety occupation requiring peak performance and fitness.

The HIV/AIDS virus (human immunodeficiency virus, the pathogen responsible for AIDS) is less readily transmissible than hepatitis B and has similar characteristics. The military population and high-performance public safety occupations are have a low prevalence of HIV/AIDS infection. No papers were found on this particular infection but normally HIV/AIDS infection from occupational exposures (principally needlestick injuries) closely track hepatitis B rates.

Methicillin-resistant *Staphylococcus aureas* is any strain of the bacterium resistant to this essential antibiotic. MRSA is spread primarily by direct contact, which occurs more often in hospital settings but occurs as well in the community. MRSA infection is acute and it is unlikely that a compensable illness would result in large numbers or that an individual case would present an adjudication problem.

In summary, infectious diseases, while a potential hazard of firefighters in general, are unlikely in practice to result in compensable illness very often and when they do there are likely to be individual-specific circumstances.

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# Chapter 8
# Cardiovascular Risks of Firefighting

**Nancy Lightfoot, Elpidoforos S. Soteriades, and Stefanos N. Kales**

Cardiovascular disease (CVD) is the leading cause of on-duty death among firefighters (45 % of on-duty fatalities) and a major cause of morbidity. CVD in the fire service also has adverse public safety implications as well as significant cost impacts on government agencies. Recently, our understanding of CVD in the fire service has significantly improved and provides insight into the risks of firefighting and potential prevention strategies. The CVD risks of firefighting relate primarily to the interaction of physically and psychologically stressful duties with underlying CVD. These strenuous duties provoke a physiology of cardiovascular arousal in association with acute firefighting activities, which usually have no long-term consequences in healthy firefighters, but can trigger pathophysiologic changes and acute CVD events in firefighters with underlying heart disease. Accordingly, unique statistical approaches have documented that on-duty CVD events do not occur at random in the fire service. They are more frequent at certain times of day, certain periods of the year, and are overwhelmingly more frequent during strenuous duties compared to non-emergency situations. Moreover, as expected, on-duty CVD

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events occur almost exclusively among susceptible firefighters with underlying CVD. The most recent studies of on-duty deaths in the fire service indicate that both coronary heart disease (CHD) due to atherosclerosis of the coronary arteries and cardiac enlargement are key pathologic hallmarks of underlying CVD and often co-exist. Despite the strenuous nature of emergency duty, the prevalence of low fitness, obesity and other CVD risk factors in the fire service are high. Robust evidence for both highly prevalent risk factors and the interaction of strenuous duties with underlying CVD supports the aggressive application of preventive measures with proven benefits such as lifestyle modifications and medical treatment to mitigate CVD risk factors. Furthermore, all fire departments should have entry level medical evaluations, institute periodic medical and fitness evaluations and require rigorous return to work evaluations following any significant illness. Finally, based on overwhelming evidence supporting markedly higher relative risks of on-duty death and disability among firefighters with established CHD, with few exceptions, firefighters with known CHD, other atherosclerotic endpoints and marked cardiomegaly should be restricted from participating in emergency duties.

## Introduction

Cardiovascular disease (CVD) includes diseases that affect heart and/or blood vessel function. CVD, commonly called, heart or cardiac disease, is a major cause of death for both men and women in the United States and Canada, but its development can prevented, delayed and treated [1]. Clinical CVD, subclinical CVD, and the presence of particular CVD risk factors all elevate the risk of sudden cardiac death (SCD) [2]. SCD is defined as natural death resulting from cardiac causes, preceded by abrupt loss of consciousness within 1 h of the onset of the symptoms. Additionally, when unobserved, the definition of SCD is extended to deaths occurring in normally functioning individuals last seen alive and well within 24 h dying of cardiac causes. Improved awareness of traditional risk factors (smoking, hypertension, diabetes, dyslipidemia, obesity and sedentary lifestyle) and better detection, treatment, and risk factor modification, have resulted in decreased CVD mortality since 1980 in the U.S. and Canada [1]. Based on typical job duties, four major categories of firefighters may be identified: municipal firefighters (professional or volunteer who respond largely to structure fires in homes and businesses and usually also provide medical and rescue services); hazardous materials firefighters (often specially trained municipal firefighters who respond to chemical, biological or nuclear exposure scenarios such as accidents); industrial firefighters (private employees who form brigades to provide fire and rescue services within proprietary locations such as: mines, chemical plants, oil rigs and refineries), and wildland firefighters (who generally fight forest and bush fires). This discussion focuses primarily on municipal firefighters [3].

CVD is the most common cause of duty-related mortality among US firefighters, exceeding both burns and smoke inhalation [4], comprising almost 50 % of on-duty

deaths, while for each fatality, approximately 17 additional non-fatal line of duty cardiovascular events occur [5].

Structural firefighting is physically challenging and may involve: stair and ladder climbing while also handling heavy materials, forcible entry, victim search and rescue, cutting and chopping to provide ventilation, and advancing charged hose lines for fire attack and suppression [6, 7]. Physical demands and psychosocial responses to the extreme environmental conditions of firefighting are complex [7]. Subsequent to a fire alarm, a "fight-or-flight" reaction occurs among responding firefighters, along with a noticeable sympathetic arousal that results in heart rate elevation and blood pressure increase that persist during transport to the scene and emergency response [2, 8, 9]. Thereafter, required heavy strenuous work increases the already considerable cardiovascular strain [7]. Adverse environmental conditions and heavy personal protective equipment (PPE) can also lead to hyperthermia and dehydration that still further increase heart rate and blood pressure resulting in increased vascular shear stress, decreased plasma volume, changes in electrolytes, and increased blood viscosity, which lead to a pro-coagulatory state [7, 8, 10–12]. Thus, depending on the type and specifics of the emergency situation, firefighters may experience moderate to severe changes in physiological cardiac function, that do not normally place healthy fit firefighters at great risk. However, susceptible individuals with underlying structural or coronary heart disease (pathological changes that significantly increase the risk of thrombosis, coronary plaque rupture, and/or arrhythmia), may suffer a SCD or experience a non-fatal CVD event [2, 7–9, 12]. Cardiac events more commonly occur in those firefighters with underlying atherosclerosis and/or structural heart conditions [2]. In fact, on-duty CVD events occur almost exclusively among: (a) firefighters with previously diagnosed CVD, (b) firefighters with some type of underlying structural heart disease that often goes undetected, or 3) firefighters with a cluster of traditional CVD risk factors and/or subclinical CVD [7]. Some even suggest that most firefighters with clinically significant CVD should not be permitted to undertake strenuous emergency work [9].

As compared to the general population, clear evidence of an increased lifetime risk of CVD mortality or premature CVD mortality for firefighters has not been demonstrated, presumably due to the healthy worker effect [1]. As mentioned previously, on-duty CVD events tend to occur nearly exclusively in susceptible firefighters with underlying CVD—either previously diagnosed or subclinical CVD [9]. CVD events in firefighters occur more frequently at specific times of the day (the majority occurring between noon and midnight); during specific periods of the year (with a winter peak and a smaller summer peak); and are observed much more frequently during strenuous emergency activities [9, 13, 14]. Specific emergency firefighting duties are associated with higher coronary heart disease risk, with fire suppression associated with 10–100 times higher risk compared to non-emergency tasks [15].

The majority of SCD among firefighters is associated with underlying coronary heart disease (CHD) or atherosclerosis of the coronary arteries. However, increasing evidence points to left ventricular hypertrophy (LVH) and others types of cardiac enlargement (cardiomegaly) as important underlying pathologic conditions,

which are frequently found to co-exist when autopsies are conducted of firefighters succumbing to SCD [16, 17]. About 25–30 % of firefighting-related SCD cases occur in individuals with a previous diagnosis of CHD, a CHD clinical equivalent (e.g., peripheral artery disease, ischemic stroke, etc) or known structural heart disease (cardiomyopathies, congenital disease, etc) [2]. In summary, various occupational factors such as emergency firefighting duties, other stressful work, and smoke inhalation can trigger acute CVD events in firefighters, especially among those with underlying CVD [7].

When a firefighter is suddenly incapacitated as a result of a cardiovascular event, the firefighter, fellow firefighters, and the public may be at increased risk, particularly if this occurs while going to the fire, undertaking fire suppression, or during a rescue [5]. As a consequence, cardiovascular events and line-of-duty deaths in firefighters have legal consequences [5]. Death, disability, and retirement benefits for firefighters who have experienced line-of-duty cardiovascular events are funded by many U.S. states and several Canadian provinces [5]. Therefore, focused proactive interventions are needed to reduce the morbidity and mortality associated with cardiovascular events in firefighters [5].

Major modifiable risk factors for CHD include: tobacco use, high blood pressure, diabetes mellitus, dyslipidemia (elevated total cholesterol, low density lipoprotein cholesterol, or trigylcerides, and decreased levels of high density lipoprotein or a combination of the above); obesity, the metabolic syndrome; physical inactivity/lack of physical fitness; and poor dietary habits [1]. Non-modifiable risk factors include: age, gender and family history of CHD. Firefighters' hazardous exposures and other workplace factors that may increase the risk of CVD events include: smoke exposure and other environmental pollutants, noise, psychological stress (e.g., post-traumatic stress disorders and high job demand with low decisional control), shift work and partial sleep deprivation, fire-house dietary patterns, irregular physical exertion, unpredictable bursts of strenuous physical activity, excess heat or dehydration, physical training, and alarm response [1]. Therefore, firefighting is considered one of the most physiologically strenuous and psychologically demanding occupations (U.S. Fire Administration, 2012) [2, 18].

Young and older firefighters who suffer from sudden cardiac death or retire due to CVD display statistically significant excess burdens of smoking, hypertension, and obesity compared to age-matched fireghters [7, 9, 19–21]. As discussed previously, about 21–30 % of sudden cardiac firefighter fatalities are seen among those with a previously known diagnosis of CHD, major structural heart disease, or a clinical CHD equivalent, such as: peripheral artery disease, ischemic stroke, etc. [7].

Age is a strong predictor of both sudden cardiac death and CVD retirement for firefighters, where risks increase in a dose-response manner, with a larger upward spike in risk after age 60 [7, 9, 20]. Compared to colleagues 40–49 years old, duty-associated risks of firefighter SCD in those 60 years or older range from approximately 4–18-fold higher, depending on the particular duty. This chapter will focus in more in depth on risk factors, including: general risk factors, occupational exposures, and the impact of personal protective equipment.

## General Risk Factors

Risk factors for CVD in firefighters are several and include a combination of occupational and personal risk factors [5, 22]. Smoke exposure, containing a plethora of gases and particulates, represents a major risk factor present in essentially all fire environments [5]. Firefighters frequently take off their self-contained breathing apparatus during post-fire suppression (overhaul), which may lead to significant exposures to carbon monoxide and particulates. Carbon monoxide may precipitate myocardial hypoxia in susceptible persons even at relatively low levels [5, 22–24]. Moderate levels of carbon monoxide poisoning can lead to left ventricular regional wall motion abnormalities that may unmask CVD by creating mismatch between oxygen supply and demand [5, 25, 26].

Career firefighters tend to work either 24- or 48-h shifts, which can commonly produce sleep disruption and deprivation. Additionally, given the unpredictability of fire occurrence and variability in routine and shifts, they often have poor dietary habits secondary to irregular meal times [5]. Furthermore, firehouse culture frequently includes meals high in fat and refined carbohydrates [4, 9]. Sleep disturbance and deprivation are associated with insulin resistance, hypertension, CVD, and weight gain that may contribute to obesity [4, 5, 9, 27]. Firefighters also tend to display inadequate fitness for the intense physical exertion needed for fire suppression and few fire departments require regular physical exercise training [5]. Lack of regular activity may lead to obesity and metabolic syndrome that are risk factors for CVD and irregular episodic intense physical activity can trigger cardiovascular events [5, 28, 29]. Furthermore, should symptoms occur during fire suppression or intense physical work, firefighters in this workplace culture do not tend to withdraw from work even if they experience cardiovascular symptoms such as chest pain that may precede a fatal event [5]. Cultural factors such as shift work, crew cohesion, risk taking, and intrapersonal, interpersonal, and organizational influences may contribute to unhealthy and risky behaviours [5]. Exposure of firefighters to live fires increase core temperature, heart rate, coagulability, leukocyte count, all of which increase their risk of an adverse event.

Core temperature continues to increase after live-fire exposure ceases. This demonstrates the need for at least 10–15 min of rehabilitation [30]. Coagulability can also be decreased by active cooling in 10 °C water [30].

The evidence suggests the need for firefighter health promotion education, including the need for physical fitness and healthy dietary habits, and to work with fire departments to establish stricter policies for use of self-contained breathing apparatus in post-fire suppression work and to promote smoking cessation and no smoking policies [5].

## Specific Risk Factors

This section will address a variety of specific risk factors for CVD in firefighters in greater detail.

### *Poor Dietary Habits*

Given unpredictability of the work during work shifts, meal times can be unreliable with the possibility of fast-food choices and higher fat and carbohydrate consumption [9, 31]. A cross sectional study of 154 firefighters in the American Mid-West found that consumption of whole grains and alcohol were associated with reduced risk of CVD, whereas higher BMI (body mass index scores) and advancing age were associated with increased risk [32].

The Mediterranean diet consists of high consumption of olive oil, fruits, vegetables, non-refined breads and cereals, potatoes, legumes, and nuts, in addition to moderate consumption of fish and poultry, and a low intake of dairy products, red meat, processed meats, and sweets, and moderate wine intake. In a cross-sectional study in a cohort of 780 young active career male American firefighters, age 18 or over (mean age: 35.6, SD: 10.0), greater adherence to a Mediterranean-style diet resulted in significant inverse associations with metabolic syndrome, low-density-lipoprotein cholesterol, and reported weight gain as was significantly associated with higher high density lipoprotein-cholesterol [33].

### *Inadequate Physical Activity and Obesity*

In the American fire service, obesity ranges from 32 to 40 % and 77 to 90 % for both overweight and obese firefighters [2, 34–38]. In a cross sectional study of 332 American firefighters, the prevalence of obesity increased from 35 to 40 % over 5 years where weight gain occurred during the period of active duty [38]. Another cross-sectional study of 116 American male career firefighters, indicated that the prevalence of obesity was 51.7 % and there was no observed difference in the prevalence of traditional risk factors of CVD in obese versus non-obese men, but found significant differences in C-reactive protein (CRP), subendocardial viability ratio, and ejection duration index in those who were obese (Smith et al. 2012). Another cross-sectional secondary analysis study of 276 American firefighters reported that obese firefighters were non-significantly more likely to develop electrocardiographic (ECG)-associated left ventricular hypertrophy (LVH) (OR = 2.0, 95 % CI:0.6–6.6) and ECG abnormalities (OR = 1.5, 95 % CI: 0.6–3.9), after adjusting for age and systolic and diastolic blood pressure [38]. Regular physical activity of adequate duration promotes cardioprotection, however firefighters are not mandated to exercise, and long sedentary periods that promote atherosclerosis and increase the risk of acute events, along with infrequent and inadequate amounts of physical activity, predominate, leading to increased fat mass, metabolic syndrome, and weight gain [38]. On the contrary, physical activity and cardiorespiratory fitness (CRF) are inversely associated with development of coronary heart disease.

In a cross-sectional study, higher cardiorespiratory fitness (CRF) in 968 male career firefighters in three Midwestern American states, as measured by maximal exercise tests, was significantly associated with lower diastolic blood pressure,

body fat, trigylcerides, low-density lipoprotein cholesterol and total/high-density cholesterol ratio, after adjusting for age and BMI [39]. However, increasing BMI had strong independent unfavourable effects [39]. Another cross-sectional study of 1,149 American male career firefighters noted that CRF was inversely associated with the risk of ECG and autonomic exercise testing abnormalities, even after adjustment for age, BMI, and maximal metabolic equivalents [40]. In a cross-sectional study of 527 American career firefighters, increasing physical activity resulted in beneficial independent effects of CRF and physical activity frequency had similar beneficial effects on CVD risk profiles [41]. Additionally, physical activity was beneficial despite BMI category, but increasing BMI category had strong independent unfavourable results [41]. Obesity is another established CVD risk factor, with some observing that the prevalence among firefighters is increasing over time [27, 38, 42]. Clearly, physical activity needs to be strongly encouraged for all firefighters, with highest priority being devoted to enhancing physical activity frequency, followed by duration and intensity [41]. Prospective studies would assist in determining the value of exercise tolerance testing as predictors of future health and employment outcomes [39].

## *Shift Work*

There is mounting evidence for an association between shift work and increased risk of CVD [9, 43, 44, 45]. Second jobs and overtime work can add to chronic partial sleep deprivation or disturbance, associated with insulin resistance, weight gain, hypertension, and CVD. Sleep can also be affected by the psychological stress of public safety work [9].

## *Hazardous Environmental Conditions*

Firefighters are routinely exposed to fire smoke, which consists of toxic gases such as carbon monoxide and cyanide, as well as particulate matter. In recent decades, smoke exposure has been significantly diminished by mandatory use of self-contained breathing apparatus during fire attack and suppression, but firefighters do not often wear the apparatus or other respirators in "overhaul," the period immediately following fire suppression when inspection for remaining sources of potential re-ignition occurs, and when various noxious inhalants are still detected [9, 46, 47]. Smoke inhalation may result in tissue hypoxia due to carbon monoxide, cyanide, and direct hypoxemia, leading to myocardial ischemia in firefighters with underlying CVD and particulates have been associated with endothelial dysfunction, increased heart rate, decreased heart rate variability, increased blood pressure, increased blood coagulability, arrhythmia promotion, and accelerated progression of atherosclerosis [2, 9, 48–50].

### *Noise*

Noise is an intermittent exposure that increases blood pressure. Siren noises may elevate systolic blood pressure, but whether the increase in blood pressure is of short duration or results in chronically elevated pressures is unknown and requires further research [9, 51].

### *Psychological Stress*

Firefighters experience various psychological stressors and high occupational demands with low decisional latitude may increase stress levels that lead to elevated heart rate and blood pressure, as well as poor sleep quality [9]. Emotional stress, including excitement, frustration or anger, can trigger CVD events in those with known CHD [2]. Stress-susceptible firefighters exposed to more extreme stressors may also develop post-traumatic stress disorder that further impacts heart rate, blood pressure, and the metabolic syndrome [9, 27, 52–54].

### *Physical Workload, Heat, and Dehydration*

Structural firefighting may involve forcible entry, search and rescue, structure ventilation, and fire attack and suppression which require high levels of dynamic aerobic and static physical exertion, while wearing heavy, insulating personal protective equipment (PPE), and multiple layers of clothing that increase metabolic and thermal demands [2]. Heavy physical exertion or strenuous work can trigger acute CVD events, especially among sedentary individuals [2, 29, 55–57].

During their work, firefighters produce large amounts of metabolic heat and insulating PPE greatly reduces heat dissipation, and fire-related heat adds thermal strain [9]. Strenuous work in a hot location, while wearing protective clothing results in profuse sweating and dehydration [2]. Obesity, low levels of physical fitness, dehydration, lack of acclimatization, previous history of heat illness, and sleep deprivation predispose firefighters to heat stroke [9, 58, 59]. Other medical conditions that impair thermoregulation, such as diabetes, sweat gland dysfunction, viral illness, sunburn, cardiac disease, and medications that impair thermoregulation, including stimulants, anticholinergic and some cardiovascular drugs, also increase risk [9, 59, 60].

Firefighters also lose a lot of fluid through perspiration, due in part to wearing heavy turnout gear. This contributes to dehydration that decreases plasma volume and results in hemo-concentration, an increase in the concentration of several biochemical parameters, and an increase in prothrombotic tendencies that may be important for those with underlying vascular disease [9]. Firefighting activity leads to hyperthermia, dehydration, and substantial CV strain, therefore, in a susceptible firefighter CV strain may precipitate a sudden cardiac event [9].

### *Smoking*

There is no question that smoking is a major independent CVD risk factor and continues to be a problem in the fire service, with a current prevalence ranging from 10 to 18 % in general cohorts and 40–50 % among those dying from on-duty fatalities [9, 13, 19, 61]. There is further work to be done in the area of smoking cessation in firefighters.

### *Hypertension*

About 20–30 % of American firefighters have hypertension and this is anticipated to increase as a result of the increasing obesity epidemic [2, 38, 62, 63]. Hypertension is also a major risk factor for CVD morbidity and mortality, and higher blood pressure is associated with CVD risk factor clustering, older age, dyslipidemia, insulin resistance, and glucose intolerance [9, 36, 38, 51, 64–66]. In an American prospective study of 334 hazardous materials firefighters, followed on possible adverse outcomes (i.e., death, placement on "injured-on-duty" status, termination of duty, resignation, retirement, or incident cardiovascular disease), Cox proportional hazards regression models that included age, body mass index, smoking, cholesterol, and anti-hypertensive medication revealed a hazard ratio of 3.2 (95 % CI:1.50–7.04, $p=0.003$) for stage II hypertension, and 4.6 (95 % CI: 2.08–10.11, $p=0.0002$) if that stage of hypertension was untreated [64]. In fact, hypertension-associated risks are concentrated among those with uncontrolled hypertension [9]. The majority of incident CVD events in emergency responders occur in those in the pre-hypertensive or only mildly hypertensive range [64], therefore the above authors maintain that firefighters who have a systolic blood pressure of 160 mm Hg or greater should be evaluated further and demonstrate improved control of blood pressure before being determined as fit for duty [9].

### *Dyslipidemia*

Dyslipidemia is reported in over 20 % of firefighters, and in one prospective study, firefighters who developed coronary heart disease, had significantly higher total cholesterol, low density lipoprotein (LDL), and triglycerides [4, 51, 67].

### *Established CHD and CHD Equivalents*

Coronary heart disease (CHD) includes: peripheral artery disease, carotid stenosis, and history of thrombotic stroke or ischemic attack. The prevalence of established CHD is about 1 % in career firefighters [9]. Previously diagnosed CHD is a strong

independent risk factor for on-duty CHD events, on-duty CHD mortality, and CHD-related retirements [9, 13, 19, 20]. Previous evidence of myocardial damage due to past infarction is associated with an increased risk of death [9, 19]. When 87 male American firefighter fatalities due to CVD were compared to 113 male American firefighter survivors, who retired with disability pensions for heart disease subsequent to on-duty nonfatal events, previous CHD (OR = 4.09, 95 % CI:1.58–10.58 was a strong independent predictor of case-fatality among those that experienced on-duty CHD events [19].

## *Demographics of the Firefighter*

Advancing age is an independent risk factor for an adverse CVD outcome in firefighters even after multivariable adjustment for all other risk factors is applied, including an adjustment for types of duty performed, during or just before the onset of the CHD event [9, 15]. An Australian study of 73 firefighters in three age groups (i.e., 25–34, 35–44, and 45–54), who volunteered for physical testing, reported a significant decline in physical standards due to age, such that the older participants in the highest age group, demonstrated significantly poorer physical standards compared to the younger participants for cardiovascular fitness ($p < 0.05$), strength ($p = 0.001$) and simulated operational power testing tasks ($p < 0.001$). In addition, age-related body composition changes were noted independent of BMI [68]. It has been recommended that minimum recruitment standards and fitness programs consider age-related declines in physical capabilities and a minimum standard, regardless of age [68]. Currently, very few fire departments require periodic testing of veterans and if done, they do not require strict physical fitness requirements for continuing active duty [9].

In a cross-sectional sample or workers' health surveillance data from three fire departments in the Netherlands, women firefighters were more likely to display diminished physical requirements (OR = 28.5, 95 % CI:12.1–66.9) and less likely to have cardiovascular disease risk factors (OR = 0.3, 95 % CI:0.1–0.5), as compared to male firefighters [69]. Health requirements examined included: psychological, physical, sense-related, and cardiovascular components. As compared to volunteer firefighters, career firefighters were less likely to display diminished physical requirements (OR = 0.5, 95 % CI: 0.3–0.9), but had a higher prevalence of cardiovascular disease risk factors (OR = 1.9, 95 % CI:1.1–3.2) [69]. Compared to the youngest firefighters, the oldest were more likely to have CVD risk factors as compared to the youngest (OR = 4.4, 95 % CI: 1.7–11.1) and middle-aged (OR = 3.1, 95 % CI:1.2–7.9). Male, older age, and volunteer-status professional firefighters are therefore identified as high risk groups who are prone to at least one specific work-related diminished health requirement [69].

## Stroke (Cerebrovascular Disease)

Stroke is a manifestation of vascular disease in the blood vessels supplying the brain. The risk factors for the most common neurovascular condition, stroke, are generally the same as for heart disease. Stroke risk factors are also discussed under cardiovascular disorders. Thus, it is not surprising that rates of stroke as a cause of death closely parallel death from ischemic heart disease (mostly myocardial infarction, the principal form of heart attack).

Both stroke and ischemic heart disease mortality is decreasing, largely due to better treatment, but deaths from both are frequent and improvements in survival are observed across the population, not just in fit populations such as firefighters. Therefore, mortality compared to the general population remains a reasonable measure of stroke risk among firefighters and by that measure many studies of sufficient power show a significant and sometimes pronounced deficit of deaths from stroke among firefighters. For example, Baris et al. shows a relative risk of 0.83 (0.69–0.99) [70]. This deficit in stroke mortality has been present for a long time. Vena et al. showed a risk (PMR) of 0.76, compared to police [71]. However, this is a reflection of underlying cardiovascular risk factors, which tend to be favorable in modern firefighters.

## Compensation

Given that most firefighters are government employees who provide public services, CVD is important from both the health and policy perspectives, and has death, disability, and retirement benefits generally regulated by specific legislation. More than 35 states and seven Canadian province-level jurisdictions (i.e., Alberta, Manitoba, New Brunswick, Northwest Territories, Ontario, Saskatchewan, and the Yukon) [72] have presumptive legislation, that presumes their CVD to be work-related, despite the presence of standard CVD risk factors, thus entitling firefighters with heart disease to receive disability and/or death benefits [9]. CVD events that occur during or within a day of strenuous firefighting duties, including fighting a fire, responding to or returning from an alarm, or involving vigorous physical training of a strenuous rescue, are likely to be work-related [9, 73].

## Prevention

Of utmost importance is that preventive measures with proven benefits in the general population be much more aggressively offered to firefighters who work in public safety, have severe CV strain during emergency operations, and are exposed to increased risks of acute CVD adverse events during active duty including rescue

and fire suppression activities [9]. It is also recommended that all fire departments completely ban smoking and tobacco products, encourage wellness programs, balance anti-discrimination and employment law considerations with common sense obesity standards, have entry-level medical examinations, offer periodic medical surveillance and evaluations and specify return to work evaluation processes following major of significant illness that require occupational physician's authorization, and adopt mandatory retirement from active firefighting at age 60 [7, 9]. Aggressive action is urgently required to reduce line-of-duty deaths in firefighters and exercise training and risk reduction programs, with sufficient interaction between qualified fitness professionals and local fire departments is essential [74]. Thus, continuing, high quality, evaluated health promotion programs must be a priority for firefighters and their employers. Finally, most firefighters with known CHD or other clinically important atherosclerotic outcomes should not participate in strenuous emergency work and instead should be reassigned to appropriate modified duties [7].

Firefighting is a job that involves many hazardous tasks and environments that can place workers at higher risk of cardiovascular disease. Firefighters merit closer ongoing surveillance and active encouragement and participation in health promotion programs.

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# Chapter 9
# Respiratory Disorders

**Tee L. Guidotti**

Firefighting involves inhalation of products of combustion, toxic materials that happen to be on site, and particles generated by debris from disintegrating structures. Obviously the lung is the organ of first contact and plays a role both as the route of entry for systemically toxic agents, such as carbon monoxide and cyanide, and as target organ for these various insults, either acute or chronic. The first has been obvious and well accepted. Acute effects on the lung itself have now been well characterized. However, chronic effects on the lung itself have been difficult to prove until suitable longitudinal studies of pulmonary function became available. It is still remarkable how little non-cancer lung disease is associated with firefighting considering the extent and severity of the hazards. Nonetheless, chronic lung disease clearly does exist as a risk of firefighting,

One reason for the difficulty in demonstrating chronic effects was that early population studies focused on the question of whether mortality was elevated from chronic obstructive pulmonary disease (COPD) rather than using functional, measurable endpoints as outcomes. Another reason is that the extant literature for years did not appreciate the time course of acute effects. When functional endpoints were examined, the results were not always interpreted as they would be today. Even in retrospect, however, there was not much to suggest a relationship. One of the few studies of that era to use an index of exposure also did not show an exposure-response relationship, after an initial period likely to represent a subpopulation of recent hires who did not make it through probation [1].

Chronic respiratory disease other than lung cancer has not been prominent in population-based studies of firefighters and cohort mortality studies have generally not shown an effect. One apparent exception is a cohort followed in the US Pacific Northwest up to 1980 that was reanalyzed and found to show a healthy worker effect for overall mortality (SMR 82), which had been absent in most studies of that

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era, and no elevation in mortality from non-malignant respiratory disease (81; 71–89) when compared to the US general male population. On the other hand, firefighters were reported to show an excess risk of respiratory disease deaths (SMR 141; 86–294) when compared to police. However, in this study police had an unusually low mortality from non-malignant respiratory disease (SMR 48; 25–84), compared to the general US population [2]. Thus, it seems more likely that the study was actually uninformative, because of an anomaly in the police, rather than being the strong evidence for an effect among firefighters that the authors suggested.

A more nuanced way of thinking about lung effects was to consider acute and chronic on a continuum of effects [3]. Earlier investigators were not remiss in neglecting this obvious natural history. However, the evidence compiled to test this line of thinking was initially misleading.

Acute lung injury, as with other toxic effects, should be proportional to exposure, both in terms of peak concentration (which would be expected to correlate with provocation of bronchospasm, and cumulative exposure, or dose, which would be expected to correlate with inflammation and chronic effects. In one particular bad office building fire in Los Angeles burning polyvinyl chloride released clouds of thick black smoke (such fires also produce hydrochloric acid) and 19 firefighters demonstrated transient hypoxemia and two who were also involved in fire suppression did not. When they were retested a month later and compared to matched controls, they had returned to baseline lung function, which was within predicted limits for all but four who smoked. This study suggested that the acute lung injury of smoke inhalation was transient and did not lead to immediate de-compensation of baseline function. The authors concluded that acute smoke inhalation did not appear to predispose to the development of chronic respiratory symptoms or chronic functional respiratory impairment." [4] However, in retrospect this is an over-interpretation of the data in what is a small study, based on an exposure that is not representative of fire smoke in general. The study could not rule out a contribution to cumulative damage and risk of accelerated loss of function over time. Indeed, those are precisely the predominant concerns today.

The paucity of evidence for a benign (non-cancer) respiratory effect left the field in some turmoil. It seemed obvious that firefighters should be at risk for lung disease, both malignant and non-malignant. However the empirical evidence in the 1908s and 1990s was not supporting these conjectures. In the end, it was the wrong type of evidence.

## Acute Effects

The lung is a structurally simple but vulnerable organ, intimately linked physiologically as well as physically to the heart and circulation, and continuous with the upper respiratory tract, which is the site of many important host defenses that protect it, which shares many responses with the lower respiratory tract (such as airways reactivity) and the digestive system, to which it is related embryologically.

Because of its structure simplicity and functional limitations, the lung has only a limited number of possible responses to toxic injury and the immune or inflammatory reaction to that injury, which can be categorized in general terms as airways responses (reactivity and inflammation), alveolar and vascular responses (pulmonary edema and pneumonia-like inflammatory infiltrate), interstitial responses (most obviously pneumoconioses, of which asbestosis is most commonly cited by firefighters as a risk. (Although asbestosis itself is not observed, the risk of asbestos exposure relevant to cancer has now been indirectly confirmed by the demonstration of high rates of mesothelioma (an asbestos-associated disease) among firefighters [5].) Studies of the prevalence (usually) or incidence of lung disease, symptoms, and loss of pulmonary function among firefighters are relatively few and in the early years mostly cross-sectional rather than longitudinal. The latter is much more useful, both in determining both causation and disease risk and because decrement in lung function over time has high predictive value for individual prognosis as well as group risk.

Studies of lung disorders or of lung function are well-recognized to be subject to bias, most obviously confounding from smoking. Smoking rates appear to be less among firefighters than in the general population, so that there is a built-in overcorrection in mortality studies where the reference is the general population but when firefighters are studied alone or with a highly-selected comparison group (such as police), attribution becomes difficult. Survivor bias is a major problem in firefighters, because the well-known "healthy-worker effect" appears to be much less strong, historically, for firefighters than for other occupations of comparable socio-economic status (SES) but has also improved in recent years, creating a temporal discontinuity. (There is also a temporal variation in exposures due to changes in composition of housing and building materials.) Misclassification bias becomes a serious problem in studies of firefighters when attempting to make associations with particular disease categories, such as isolating risk among airways disorders (asthma, bronchitis and bronchiolitis, emphysema and their combinations, in the form of COPD).

Further, knowledge of the respiratory outcomes associated with firefighting has changed in recent years due to intensive studies of the New York Fire Department (FDNY) members who responded to the World Trade Center (WTC) tragedy in 2001, and for which anomalous types of airways disease have been reported (in particular, forms of bronchiolitis previously under-appreciated). These findings support the impression that WTC responders are experiencing different health care outcomes from other municipal firefighters without WTC-related exposures.

## *Acute Effects on Lung Function*

That firefighters may experience short-term drops in blood oxygen (hypoxemia) following smoke exposure has been known for many years and was quantified in the early studies of firefighters at a time when synthetic materials were already installed in residences and office buildings.

In the pioneering Boston studies, acute inhalation to fire smoke in a relatively unremarkable series of fires was noted to be associated with decreases in $FEV_1$% of 0.050 l on average, a reduction that is significant for a pre-/post-exposure change but not likely to be noticed by the firefighter, and 0.10 l in 30 % of subjects suggesting a subset with increased susceptibility (although only one subject in 39 gave a history of asthma). The loss of pulmonary function was transient but was proportional to intensity of exposure. Of interest is that second exposures within hours resulted in greater acute reductions in flow, proportional to the previous exposure. Cough and eye irritation were frequent but not severe [6]. Not commented on was the observation that in a small fraction of observations (roughly a third), flow not only did not decline but increased, greatly so in a few subjects, suggesting some unrecognized mechanism of bronchodilation.

The susceptibility of a subset of firefighters was further underlined with a small case series of prolonged reactive airways disease following exposure to fire smoke containing pyrolysis products of polyvinyl chloride (PVC), which consist principally of vinyl chloride and hydrochloric acid [7]. These cases, two of which would today be considered irritant-induced asthma and the other a severe organizing bronchitis modified by steroids, were used by the authors to highlight the dangers of PVC pyrolysis and combustion products. However, it can also be interpreted as highlighting the paradox that whether from exposure or susceptibility characteristics, relatively few firefighters show such dramatic changes.

Firefighters are not immune to the effects of cigarette smoking and evaluation of baseline function must therefore take smoking into account. Comparing smokers and non-smokers, most smoking firefighters had preserved the major ventilatory measures of pulmonary function (FVC, $FEV_1$) although they tended to have symptoms of productive cough, but a minority showed decreased $FEV_1$, $FEV_1$% ($\leq$70), or $dV_{max}$, while nonsmoking firefighters did not. (Such results are entirely to be expected among smoking populations.) More interesting, while as expected small airways disease (by the He dilution method) was present in firefighters in 35 % of smoking firefighters, without restriction by age, small airways disease was also present in 13 % of nonsmoking firefighters, but only among the nonsmoking firefighters with $\geq$25 years of fire service. The degree of small airways disease was not enough to cause respiratory impairment to be clinically significant. A small subset of these firefighters were engaged in one particular fire but did not show marked changes in their baseline lung function after the fire. The authors commented that their results were relatively benign in part because the fire was not especially severe, as indicated by relatively low carboxyhemoglobin measurements. Even so one of their cases, who had exhausted his SCBA air supply while in the basement of a building and had to breathe smoke on the way out, required hospitalization and had a profound chronic respiratory impairment and ultimately had to leave the fire service [8]. This relatively early study established that smoking played a role in respiratory impairment equal to or more likely greater than fire smoke inhalation under normal firefighting operations, but that under abnormal conditions acute and severe respiratory effects were possible, even in fires that did not involve exceptionally toxic inhalation (such as the combustion and pyrolysis products of polyvinyl chlorine mentioned above).

The issue of susceptibility naturally arises first in the context of airways reactivity and prior history of asthma. Therefore it was natural, in the subsequent groundbreaking study, to evaluate the acute response to fire smoke among firefighters in light of their baseline airways responsiveness to methacholine, the provocative test for airways reactivity. In a series of determinations following otherwise unremarkable fires, it was found that 24 % of firefighter subjects transiently lost more than 2 standard deviations in $FEV_1$, although as much as 10 % in only two (about 7 %) of cases, both of which showed increased methacholine responsiveness. Contrary to expectations, however, the degree of loss was not proportional to the initial degree of airways reactivity [9]. This led the authors to conclude that fire smoke acted by means other and in addition to simple airways irritation. However, three of the five subjects with greatest pre-/post-fire changes in flow had histories of childhood asthma. The study did not factor in smoking history, in part because the design of the study was grounded on physiological rather than toxicological principles and did not take into account possible tolerance effects. It was also impractical, given the study design requiring multiple measures, to recruit a reference population. Unfortunately, the study could not be repeated with a larger population and with a reference group.

Understanding of the acute effects of smoke inhalation has required studies conducted under controlled circumstances. The previous approach of studying firefighters following uncontrolled events proved to be misleading (see above). In practice, this has meant studying lung function, inflammatory responses, and physiological responses following controlled burns or in smoke chambers. In one such study using smoke chambers, in Singapore, an ethnically homogeneous group of firefighters (Malay) without airways reactivity (by histamine challenge) at baseline showed transient, acquired airways reactivity following smoke exposure, and a subset that had prolonged duration of reactivity showed persistence of flow reductions even after reactivity came back to normal after 24 h.

Similarly, a panel of Seattle firefighters, none of whom had documented asthma, showed exposure duration-related acutely decreased airflows ($FEV_1$, $FEF_{25–75\,\%}$) and airway responsiveness to methacholine from their baseline within hours after firefighting, with associated reduction in specific airway conductance. The findings were unrelated to smoking [10].

Less physiologically-grounded, more clinically-relevant studies also demonstrated that although most firefighters show a relatively small reduction in lung function, principally in $FEV_1$ and $FEV_{25–75\,\%}$ (indicative of small airways abnormality), a small subset showed more profound changes that could interfere with function [11]. These changes were independent of lung function.

Further studies during the overhaul phase of firefighting identified it as associated with acute decline in ventilator measures and increased measures of inflammation (CC16 and SP-A, described in the next subsection), and correlated with carboxyhemoglobin levels [12]. This strengthened the growing impression that overhaul involved significant exposure and could be as risky as knockdown. However, at the same time, an anomaly was identified in that similar changes were seen in firefighters who used cartridge (air-filtering) respirators, suggesting that the cartridges were not completely effective protection. This evidence argued strongly

for using SCBA during overhaul, rather than relying on air-filtering devices. However, practical considerations make this difficult on the ground.

Acute onset of respiratory symptoms, including shortness of breath, mucosal irritation and sinusitis were also documented, in addition to transient lung function changes [13].

At the same time, a parallel series of studies demonstrated that the same effect was observed among wildland firefighters. (Not reviewed here.) This was important in establishing that synthetic materials were not the only cause of acute lung function change, as wildfire smoke is predominantly of lignocellulose origin and less irrigating than smoke from burning synthetic materials. It was unclear which combustion products were responsible. However the wildland firefighter population also demonstrated an anomaly: not showing the expected response to increased concentrations of smoke-derived irritants [14]. Rather they were behaving as if exposure to wood fire smoke triggered a limited, maximum reaction.

## *Acute Inflammation*

Concomitant with acute changes in pulmonary function are changes in the expression and release of various acute response and inflammatory markers following exposure to fire smoke, at least in studies unconfounded by smoking. Not surprisingly, exposure to fire smoke provokes an acute inflammatory response in the lung, release of neutrophils and their accumulation in sputum, and release within a few hours of biomarkers such as IL-6, IL-8 and TNF-α, accompanied by a rapid decline in IL-10 (cytokines) [15, 16]. Similar effects were seen in wildland firefighters [17]. The lung response evolves to lymphocyte proliferation and elevation of fibronectin in lavage fluid [18]. (Novel biomarkers, including chitotriosidase, have been studied as a predictor of chronic effects in the World Trade Center population [19] but not as yet among firefighters in general.) None of this is surprising or out of keeping with what is known of the inflammatory response in the lung.

A more specific indicator that may be of value in structural firefighters is elevation in high-sensitivity C-reactive protein (CRP) levels. The elevation in CRP levels predicts reduced levels of airflow, although longitudinal data have not been conducted to assess causation [20]. In addition to suggesting a candidate for an inexpensive, readily available clinical marker, the finding is also consistent with the view that acute inflammation changes functional capacity and that effects might be cumulative.

Acute oxidant and irritant gas exposure may result in deep lung injury and capillary leak (also known, in the terminology of pathology, as “diffuse alveolar damage” and in the clinical literature as “toxic inhalation”), which progresses over time (usually hours) to first interstitial and then alveolar pulmonary edema, which carries a high mortality [21]. Fortunately, this outcome is rare in firefighters, despite the potential for it. The probable reason is that exposure to common fire smoke does not include the one combustion product most likely to do this: nitrogen dioxides.

The oxides of nitrogen can be formed from combustion but require high heat or pressure to produce in quantity. Rather toxic inhalation and pulmonary edema are more likely to occur in burning hazardous materials, or "hazmat" situations, where the combustion source and substrate produces highly reactive chemical products that are relatively water-insoluble and so penetrate to the deep lung, such as phosgene and paraoxons. These situations are not common, fortunately.

### *Transition from Acute to Chronic Effects*

Baseline circulating surfactant-associated protein and Clara cell protein (CC16) levels were lower for firefighters than police, in a cross-sectional study [22]. However, this should not be over-interpreted as suggesting that police have a higher level of response by inflammation in the lungs. More likely, inflammation in the lungs of firefighters may be low between incidents of exposure levels or conceivably tolerant or even downregulated in response to multiple acute inflammatory responses, whereas in police there may be more frequent low-level provocation of inflammation due to air pollution or vehicle exhaust.

It now seems clear that most of the acute effects of fire smoke resulting in lung responses are reversible and correct over a relatively short period, under normal circumstances. However, an individual firefighter may reach a tipping point in response to unusually severe exposure or because of personal susceptibility. In those exceptional cases, the acute effect results in subacute or chronic injury of such magnitude that it leads to impairment or prolonged recovery.

## Chronic Effects

Since 2001, the literature on chronic pulmonary health effects in firefighting has featured two areas of emphasis: the experience of undifferentiated municipal firefighters and that of respondents to the World Trade Center (WTC) tragedy. As noted earlier in Chap. 1, the exposure regime and the pattern of health outcomes are different for WTC responders and this is reflected in the compensation criteria for surviving FDNY responders, which is handled separately from claims from other firefighters and which has its own presumption criteria. While some lessons from the WTC responders are obviously generalizable, much is not. One example of a seemingly unique WTC-related phenomenon is the frequency of progression of what initially appears to be restrictive disease in a subset of WTC responders but which in fact represents air trapping [23], a finding that is suspected to be associated with the pathology of constrictive bronchiolitis [24]. This is not a feature of the literature on firefighters in general. Thus, this section will mention WTC responders only sparingly and where the issue is narrow and clearly relevant to all municipal firefighters.

## *Pulmonary Function*

One of the seminal studies on firefighters was significant not only because it proved an effect but because it provided the explanation as to why other studies did not. This early observation was then largely overlooked or forgotten by other investigators.

The pioneering studies on lung function among firefighters were conducted in Boston in the 1970s [6, 25–30]. Measurement of lung function by spirometry at baseline was repeated at one year and subsequently at an average interval of three and one-half years. The study revealed that lung function declined over time but not in a steady way and that the decline was not associated with the frequency of fires attended or, oddly, with smoking history. However, the study had many anomalies. Cigarette consumption was inversely proportional to the number of fires fought. (This is consistent with a firefighter having had enough of smoke of any kind, but also introduces a counter-trend that may have confounded the result.) Firefighters who had fought no fires had a higher rate of decline in ventilator measures, both FVC and $FEV_1$. (This suggests that there was a reason they were being kept away from fires, not that other duties affected their lung function.) Firefighters who had been involved in knockdowns or who had experienced "shellackings" or "pastings" (colloquial terms for being overwhelmed by smoke) did not show disproportionate decrements in pulmonary function. Excluding firefighters on the sick roll or with known illness did not affect the result. However, 21 % of subjects were lost to follow up, a very high number for a longitudinal study spanning only 3 years, and those who left the fire service had shown greater than average decrements in lung function on the first round of testing, after 1 year. Faced with these contradictions, the authors concluded that there were major confounding factors of selection that resulted in affected firefighters being excluded from service [28]. A second study, conducted on retirees, showed that selection factors within the fire service resulted in protection of firefighters in that era through transfers, administrative promotions, and especially retirement [29]. A third study at 6 years showed no accelerated loss of lung function, which was attributed to success in encouraging adherence to SCBA usage but may have been an artifact of the strong selection pressure that operated on firefighters who were symptomatic or even possibly subclinical but less robust in their performance. In the 6-month study, the authors recognized the earlier pattern of out-migration and internal accommodation within the fire service in their data [27].

The expected association between firefighting and accelerated decline in lung function was finally demonstrated unequivocally in a cohort of Boston firefighters studied by a different group. They determined that firefighters had a greater loss of pulmonary function than a non-firefighter male reference group followed in a normative aging study, together with larger variation (as measured by standard error, SE) and that the effect was not explained by age alone, initial function, or smoking, although smoking was associated with clinical symptoms (such as cough). For $FEV_1$, nonsmoking firefighters showed an average annual decline of $81 \pm 19$

(SE) ml/y compared to 64 ± 3.9 for nonsmoking subjects of the aging study. Initial function was higher for firefighters than for the reference population, which the authors credited to selection bias due to employment standards [31]. This study, by Sparrow et al., established that exposure to fire smoke is associated with accelerated decline in lung function in firefighters, as it is in other occupations with respiratory hazards.

However, this apparently clear demonstration of an association was then made not so clear, by a series of unrelated studies suggesting that cigarette smoking was a clear risk factor for decline in lung function but that firefighting, as a risk factor, showed primarily short-term effects and little evidence for chronic impairment in the long-term [32, 33, 8]. Even so, there were exceptions among these studies, in two of which firefighters demonstrated chronic changes that were associated with respiratory symptoms [11, 13] and one which suggested that cigarette smoking, in a population of high-prevalence (43 %) smoking Polish firefighters, played a minor role and that the effect of firefighting predominated.

Finally, an important study of professional firefighters in Seattle demonstrated that while ventilatory measures were indeed preserved in a stable population of volunteer subjects (implying the possibility of self-selection bias), the firefighters showed a decline in diffusing capacity ($D_{L\blacksquare CO}$) after adjustment for relevant factors such as age and smoking. The decline appeared to have two components: a general trend of decline associated with year and a much smaller decline associated with number of fires fought [12].

$D_{L\blacksquare CO}$ is routinely obtained in diagnostic pulmonary function testing but is not a screening test. $D_{L\blacksquare CO}$ correlates empirically with many disorders that affect the opportunity for gas transfer, but as a clinical study it is primarily useful for diseases that have less relevance for firefighting, such as interstitial lung disease. For other lung disorders, the $D_{L\blacksquare CO}$ has substantial drawbacks as a physiological measure, related as it is to blood volume, perfusion, and diffusing time, and so its use as a screening test for firefighters was not recommended.

Not surprisingly, among premorbid risk factors, firefighters with $\alpha_1$- antitrypsin deficiency lung function showed accelerated loss of lung function, even in phenotypically PiZ heterozygous firefighters who have a moderate serum level of circulating protease inhibitor [34]. Although empirically demonstrated for World Trade Center respondents, this particular finding of susceptibility is almost certainly generalizable to normal fire smoke and is observed in other situations of occupational exposure, and so is mentioned here. Homozygous PiZZ persons are unlikely to qualify or be retained as firefighters because their defect is likely to result in impairment that would disqualify them based on employment standards.

By far the most important susceptibility state across the range of pulmonary outcomes, however, is asthma or atopy, the hereditary predisposition to allergy characterized by asthma, sinusitis, childhood eczema, and allergic rhinitis and marked by an increase in serum immunoglobulin E and accompanied by airways hyperreactivity (the degree of which is quantified by responsiveness to inhaled methacholine). People with atopy are also variably predisposed to other lung conditions which are associated with decline in lung function. (The “Dutch hypothesis”, which links this

"allergic diathesis" with lung cancer and COPD remains to be proven definitively but is widely accepted.) For firefighting subjects with allergy or asthma, the primary problem is apportioning their decline in lung function among three drivers: atopic predisposition (hereditary), firefighting (acquired and occupational), and smoking (acquired and non-occupational). The problem is complicated in that, as described above, fire smoke can induce airways reactivity acutely. Dutch investigators (no relation) attempted to do so by examining firefighters who had not been exposed to fire smoke for at least 7 days. They found that increased airways responsiveness to methacholine was significantly associated with the number of fires fought in the previous 12 months, after adjustment for smoking (which had an independent effect) and for history of atopy, and that this effect was unaffected by age or gender. There was a strong interaction between atopy and the number of fires fought. The principal conclusion of the study was that firefighters, especially those with asthma or atopy, needed to adhere to respiratory protection [35]. However, the major contribution of this study to new knowledge was to elegantly unpack the relative contributions of the three drivers.

Bringing some confusion back into the order that had been emerging, a study from South Australia of longitudinal pulmonary function among municipal firefighters found multiple influences [36]. The reference population was a probabilistic sample of the adult (male) population of South Australia, monitored in a longitudinal health survey. The great majority of participants had either quit smoking or never smoked; depending on whether the first or second round of testing was used to define the "cohort", smoking prevalence rates were 5 % or 10 %. The methodology was somewhat unusual, in that an extreme case definition of accelerated decline in lung function was used to define the outcome: > 50 ml/y, reported to be the average annual decline for heavy smokers in Australia (the average for the general population is about 30). By comparison, most such studies are based on measured airflow or difference from previous measurement [37]. This study demonstrated that there was more than one trend playing out, which is another way of saying that there were probably multiple confounders obscuring the main trend of association between decline in lung function and fire smoke exposure. One trend was that younger generations of firefighters showed better pulmonary function at the time of entry into the fire service than their elders, so the population effect overall was clearly confounded by differences in the subcohorts. Another trend was that lung function did decline over time at an accelerated rate in older firefighters (>45 years), but stayed the same or even increased in younger firefighters (a highly improbable result, which will be discussed in detail below); the control population showed the expected slow longitudinal decline. The third trend was that preservation of lung function was associated with active use of respiratory protection. Firefighters gained more weight (although this finding was not statistically significant) than reference subjects. They did not report more asthma but did report a lower prevalence of chronic bronchitis and emphysema (6 %) compared to reference subjects (27 %), as would be expected among nonsmokers. Yet another trend, acknowledged by the authors, is the healthy worker effect [36].

This study has many issues, which the authors recognized. One is that the follow-up time was very short, less than 3 years on average, possibly too short to establish

a stable trend against a background of some variability. There were probably too few smokers to study interactions with smoking. Because of this, the proper comparison might have been between firefighters and nonsmokers in the reference group, which was not reported, although it should have been taken into account in the regression model.

On the face of it, the finding of a greater trend toward loss of lung function among firefighters who do not reliably use respiratory protection, which suggests that fire smoke has an effect, is inconsistent with no loss or an increase in lungfunction among firefighters compared to non-firefighters. Of course, an actual increase in lung capacity is not strictly possible but might appear to be the case with the reversal of reversible airways disease in a substantial number of subjects. The prevalence of asthma in a very large percentage of this population was not reported but was ruled out either, so this remains a possible explanation. It may also be that the case definition approach turned a continuous function (airflow) into a binary or step function and so distorted the regression analysis. There was a difference in the methodology of spirometry, with a stricter protocol for the firefighters, but a systematic error would not explain why firefighters' lung function appeared to increase. One possibility is that if there were significant error in the measurement, in which case there may have been a statistical regression to the mean after the initial measurement gave a skewed response. In short, this study is essentially uninterpretable on the basis of longitudinal trends, probably because of multiple confounding and dissimilarity in smoking prevalence, although it is suggestive that respiratory protection is a successful means of preserving lung function. As follow-up lengthens, the meaning of these trends may become clearer.

What is required, clearly, is a longitudinal study of firefighters without exceptional exposures large enough to have sufficient power to resolve trends due to occupation, smoking, atopic diathesis (including asthmahistory), and aging. Such as study is currently underway in the FDNY, where established protocols and identical equipment and technical staff ensured consistency. The FDNY team is following 940 new firefighter hires since the WTC tragedy, and using a much smaller number (97) of EMT personnel as a reference group; firefighters have more stringent employment standards. The prevalence of smoking overall was 3.5 %. Data from the first 5 years has now just become available, separated by overall rates and those for nonsmokers. The firefighters were significantly taller and had higher initial ventilator function; turnover was very low and the few firefighters who separated were individually documented not to have left for respiratory disability. Perhaps surprisingly, the study demonstrated an average loss of ventilatory function as $FEV_1$ of 45 ml/y for both groups, with essentially no difference (there was slightly greater decline in $FEV_1$%, which is a calculated rather than a primary indicator). There was no difference observed between nonsmoking and smoking firefighters, probably because the period of follow-up was too short for this to become apparent in the relatively young population of new firefighters; weight gain was the only factor observed to affect the trend in both occupational groups. The authors pointed out that in addition to being much larger than previous studies and having a much lower smoking prevalence to contend with, this was the first study to document longitudinal trends in a firefighter population with mandated and high levels of SCBA compliance [38].

Thus, the most defensible conclusion at present is that the current generation of firefighters is not demonstrating accelerated decline in ventilatory function, at least at this early time in their careers, probably because of enforced adherence to policies requiring use of respiratory protection. Older cohorts, however, may demonstrate some accelerated loss of airflow but the situation is complicated by cohort effects and is highly multifactorial, with ample room for confounding. Taking into account the important role of SCBA as effective protection, it can finally be concluded that fire smoke is indeed associated with accelerated decline in ventilatory function but that the contemporary firefighting profession is protected to the extent that firefighters adhere to appropriate respiratory protection.

## Clinical Outcomes

Pulmonary function reflects physiological changes. Clinical outcomes involve the appearance of distinct symptoms (which for lung disease emphasize cough, shortness of breath, and wheezing) or the diagnosis of specific diseases. The two main diagnoses of concern are asthma and obstructive airways disease, not to be confused with COPD (which implies the characteristic lung disorder due to smoking). Bronchitis, as has been shown, clearly occurs as an acute response and a chronic form of bronchial inflammation (as opposed to the characteristic lung disorder of chronic bronchitis) may contribute to asthma and obstructive airways disease in firefighting. Interstitial disease due to pneumoconiosis does not seem to occur in firefighters, although exposure to asbestos is confirmed and some mineral dust exposure may occur incidentally in the course of duties. (Mineral dust exposure is much more likely to be a factor in the WTC responders.) Sarcoidosis is discussed in Chapter 7, lung cancer in Chapter 6.

### *Asthma*

By definition, asthma is a disorder of reversible airways obstruction. Because it is defined by a nonspecific functional change and not by pathology or etiology, asthma is not really a single disease, although the respiratory disorder that children get and often outgrow probably is a coherent diagnosis. There are many other types of asthma but two are of most concern to firefighters. One is the importance of a history of asthma, either current or in childhood, as a marker for atopy and reactive airways, which may render a firefighter more susceptible to the effects of fire smoke. The other is irritant-induced asthma, which is a form of new-onset asthma in the adult that occurs when inflammation is induced in the airway by exposure to chemical irritants, as occur in abundance in fire smoke. It is suggested (see Chap. 5) but not proven that fire smoke is probably more irritating than cigarette smoke because the latter contains some agents that tend to damp down inflammation, including nicotine. This means that for a given exposure, fire smoke is likely to induce more inflammation

acutely and ultimately induce greater chronic effects than the same exposure to cigarette smoke. Of course, exposure to cigarette smoke is usually much higher because it is inhaled intentionally and repetitively into the respiratory tract as a nicotine-delivery device. Fire smoke is likely to cause chronic irritant-induced occupational asthma, whereas cigarette smoke induces its characteristic deep lung lesions resulting in emphysema in part because the smoke is more tolerable in the short term.

Given the acute changes in airflow and the known susceptibility of the airway to irritant-induced inflammation and bronchospasm, it is rational to expect that asthma rates would be elevated in firefighters. Surveillance data, which inevitably are biased by gross underreporting, suggest that firefighters had the second-highest reported rates of work-related asthma among all occupations in California, after janitors, in the mid-1990s [39]. Because of extreme and often systematic biases in reporting, which amplify the effects of small errors and distortions, reported rates are untrustworthy and the remainder of the list (which included bus drivers and "eligibility clerks" among high-risk occupations) is not so plausible.

Prevalence studies of asthma among firefighters, using a battery of diagnostic techniques with bronchoprovocation being the gold standard, confirms that asthma is under-diagnosed by community physicians among firefighters, at about 6 % for Swiss firefighters; a prevalence of 14 % was suggested as closer to the true value [40]. Brazilian municipal firefighters had a prevalence of symptoms leading to clinical diagnosis of asthma (without the gold standard) that was about 9.3 % and higher than police [41]. It would appear, then, that Swiss physicians are relatively conservative in making the diagnosis. However, these prevalence rates of asthma are still not far from reported asthma prevalence in most developed countries.

It should be noted that the default diagnosis of many uncritical clinicians for any variable lung disease is often "asthma", especially in a non-smoker. (In a smoker, it would be "COPD".) Some of the WTC responders have carried the diagnosis of asthma from their local physicians without confirmation or specialist evaluation. They are now being reevaluated through the efforts of monitoring programs, often receiving more nuanced diagnoses.

In short, given the combination or accelerated decline in ventilator function (see earlier discussion in this section) and induction or irritant airways inflammation, some individual firefighters may be pushed into respiratory impairment or insufficiency, particularly following poor recovery from unusually intense exposure situations. Thus, reversible airways obstruction in the form of irritant-induced asthma cannot be ruled out for firefighters but it must be very uncommon, especially with adequate respiratory protection. The picture is undoubtedly confused by inconsistency in the diagnosis of asthma.

### *Chronic Obstructive Airways Disease*

Chronic obstructive airways disease is used here as a descriptive term for acquired fixed airflow obstruction with or without airways reactivity, in order to emphasize the functional changes and to avoid the term "COPD". "COPD" is sometimes used

casually in medicine and epidemiology as a generic term for fixed airways obstruction, but in occupational and pulmonary medicine it has a well-understood and accepted definition as the name of a particular disease associated with smoking.

Chronic obstructive pulmonary disease (COPD) is a clinical entity, associated with a smoking habit, that involves individually variable contributions of three specific processes. (1) Emphysema is a general term for simplification (destruction) of the lung architecture. In smoking-related emphysema, it starts at the level of alveoli and shows a specific pathology (peribronchiolar alveolitis). As it progresses, it results in fixed airways obstruction and a rapid decline in ventilatory function over the long term. (2) Chronic bronchitis, an inflammation of the airway wall, can be a primary diagnosis but is most often minor or absent in COPD. 3) Variable degrees of reversible airways obstruction and hyperreactivity, which may or may not present clinically as asthma but is mostly responsible for short-term changes in lung function and is responsive to the same treatment. COPD may have all three elements or proportions of each, with fixed airways obstruction predominating but reversible airflow obstruction being the major target of treatment. There are other emphysemas, asthmas, and bronchitides besides those associated with smoking. They are not "COPD" in the true sense.

The population mortality studies discussed earlier (in great detail for cancer outcomes) are consistent in not showing elevated mortality from what would be recorded (and routinely misclassified) on a death certificate and compiled in vital statistics as "COPD", asthma, or total respiratory disease. Much as in asthma among firefighters, the search for the crime in the form of chronic obstructive airways disease associated with firefighting has not turned up either a victim or a smoking gun.

Firefighters can develop COPD if they smoke. However, fixed airways obstruction among firefighters, in the absence of smoking, would not be true COPD. It would be a form of chronic obstructive airways disease with its own features, characterized by accelerated decline in ventilatory function (see above). It would lack or modify the characteristic pathology of peribronchiolar inflammation and would probably have more regular features of bronchitis, with changes in the airway epithelium characteristic of chronic inflammation. To date, evidence for a novel type of chronic airways obstruction has been difficult to find. Even at the accelerated loss of function documented in older firefighters, they may escape respiratory impairment in their lifetime if their smoking habit is not extreme.

One reason for this paradox is that firefighters are under so much selection pressure. Individuals with a susceptibility to lung disease, either known or inapparent (see the reference above to the "Dutch hypothesis") may be self-selected to be more resistant to the irritating effects of fire smoke. This is speculation, because there is no biological marker for the effect other than rate of decline in lung function over time.

Individual cases of emphysema, respiratory disability, and respiratory failure are documented, such as the PVC-related cases noted above. Unfortunately, the few older case reports lack essential exposure information and clinico-pathological correlation and have been silent on degree of airway inflammation and presence or absence of obliterative bronchiolitis. These features would be considered essential to a contemporary case report. There have also been clear cases of misdiagnosis and misclassification in the literature, including asthma that was demonstrated without question to be advanced emphysema but was still misidentified as asthma in the title of the article [42].

In short, given the probability that accelerated decline in ventilator function (see earlier discussion in this section) could push some individuals into respiratory insufficiency, particularly following unusually intense exposure situations, fixed airways obstruction and chronic obstructive airways disease cannot be ruled out as a risk for nonsmoking firefighters but it must be rare.

## Conclusion

The weight of evidence at present supports the conclusion that individual firefighters may be at risk for disabling lung disease following specific, acute events associated with extreme exposure, which may interact with individual susceptibility. Individuals who have experienced these catastrophic events will have a compatible history.

The weight of evidence at present supports the conclusion that firefighters are at general risk for lung symptoms and decline in function in any form, probably highly variable. When it appears, this condition is typically diagnosed in the community as asthma. It may clinically resemble adult-onset, intrinsic asthma (which is actually often a form of chronic bronchitis) but in fact may consist of the inexorable accelerated decline of pulmonary function into clinical impairment, combined with a superimposed irritant-induced bronchitis. However, the condition is more complicated than conventional intrinsic asthma and is not well characterized.

The clinical picture in firefighters is confused in part because of intense selection pressure frequently resulting in high or supranormal lung function on entry and preservation of lung function over many years despite inhalation of irritants that would normally be associated with accelerated decline. The picture that results appears to feature unusually stable lung function at baseline on which is superimposed multiple episodes of short-term, acute changes from which the lung recovers easily, until an exceptional exposure reaches a tipping point.

The weight of evidence at present does not support the conclusion that firefighters in general are at risk for chronic fixed obstructive airways disease as a direct result of firefighting in unexceptional situations. It is well known that unusually intense and toxic exposures (for example, to oxidant gases) may induce different types of obstructive airways disorders (such as bronchiolitis obliterans) but such cases are fortunately unusual and demonstrate compatible histories. Firefighters are of course not immune to smoking-related COPD if they smoke. Older firefighters who smoke and who have documented histories of participation in many knockdowns might experience an accelerated decline in lung function that "catches up with them" during their lifetime and presents as the onset of "COPD" after retirement, since clinicians would not be able to distinguish COPD from other forms of fixed airflow obstruction. Across the board, however, fixed airways obstruction does not appear to be a general or common problem among firefighters, contrary to expectation. This conclusion cannot be held too dogmatically, however, because all studies necessary to resolve the complicated issues have not been done and there is a strong healthy worker effect.

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# Chapter 10
# Injury, Musculoskeletal Disorders, and Ergonomics

**John M. Mayer and Sara A. Jahnke**

Not surprisingly, the occupational tasks of firefighting lead to an excessive number of injuries. In 2011, the National Fire Protection Association (NFPA) [1] estimated 70,090 line of duty injuries (LODI) among United States (US) firefighters. While numbers of injuries have been consistently dropping since an estimated 103,340 three decades ago despite the inherent dangers of the job, the rates of injury remain unacceptably high (See Fig. 10.1).

Firefighting is one of the most hazardous, physically demanding, and psychologically stressful occupations. Firefighters are required to perform at high levels of physical exertion for prolonged periods, with minimal rest, and in extreme work environments [2, 3]. These factors negatively impact the health and safety of firefighters, and their ability to carry out their mission to protect and serve the community [3].

Fire-related hazards are also changing over time. The tasks of fire suppression and rescue activities have expanded to include not only traditional firefighting and interior fire attacks, but also rescue activities, response to a variety of community threats, and patient care on medical calls. At the same time, characteristics of the fire scene have changed, with changing construction methods and materials.

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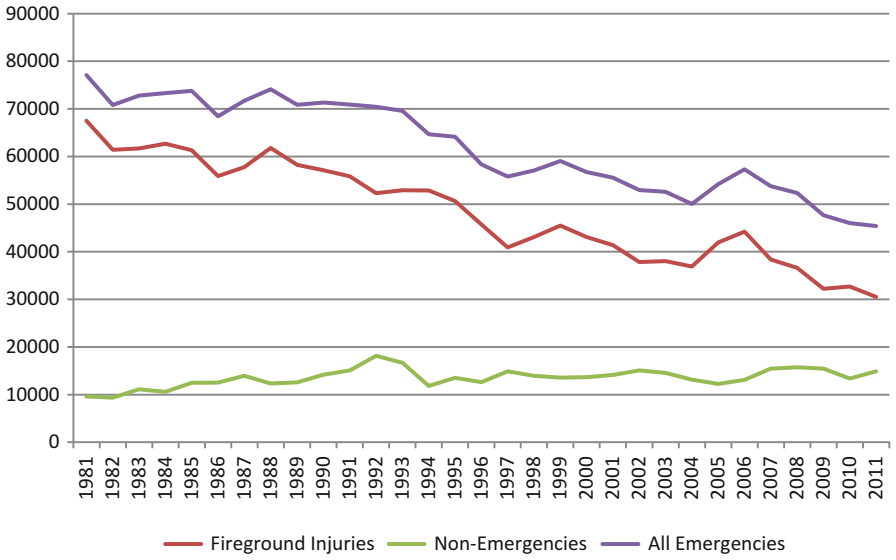


**Fig. 10.1** Number of injuries in the United States by year [1]

## Injury

Injury leading to compensable disability or death is all too common in the fire service. There is no available or speculative technology that would substantially remove or mitigate the inevitable hazards of the fire scene: structures are consumed and weakened, temperature extremes (mostly heat but also cold in wet winter conditions), uneven surfaces, poor visibility, cumbersome but necessary protective equipment, and work efforts close to the extremes of human endurance and capacity. It has been observed that firefighters accept a thin margin of operational safety protecting them from potentially fatal injury [4].

The predominant type of injury is minor trauma (over one-third), the most characteristic (almost unique to firefighters) is severe burns, and heat exposure [5, 6]. The least common injury type for firefighters among common injuries shared by public service occupations generally is vehicular accidents, a discrepancy that probably represents the sturdiness of engines and separation in the study from emergency medical technicians, who are at much higher risk [7].

For in-service injuries, the circumstances are generally documented. There are some overall trends reported in the literature that bear directly upon the fire service and should be noted. Although not separately studied in most studies, chronically disabling injuries are almost certain to show the same relationship as acute injuries with short-term disability.

As many as one-third of injuries in the fire service are the result of exercise, rather than occurring on the fire ground [6]. This reflects the decrease in the number of structural fires, the improvement in safety and equipment effectiveness, and the introduction of mandated and voluntary fitness programs in many fire departments. Since firefighters are often highly competitive and athletic, programs designed to keep them fit are often seen as invitations to engage in rigorous and competitive regimens, which may increase the risk of injury.

Firefighters who exercise have a much higher risk of on-duty injury than those who did not; however they had a much lower risk of non-exercise-related injury. In other words, mandated exercise programs increase the rate of injuries occurring on site from the exercise program but appear to protect against injury in the line of duty. The most significant risk factor for injury while exercising on duty was maximum pull weight as a fraction of body weight (4.03; 1.48–10.97), suggesting that weight training may be overdone by members who are already highly trained and may be exceeding their capacity [8]. Thus it may be concluded that mandatory or voluntary exercise may be strongly protective against injuries in the line of duty but incurs a cost in the form of more frequent, largely minor, exercise-related injuries that may result in temporary impairment.

## *Characteristics of Firefighter Injuries*

Studies of the nature of injuries incurred and types of duties being conducted at the time of injury vary in regard to methodology. The NFPA survey conducted periodically uses a purposive sampling of departments stratified by the size of community protected to extrapolate national injury rates [1]. Jahnke and colleagues [5] conducted a population based sample of 462 career firefighters from 11 fire departments in the Midwest US and assessed self-reported injury over the past 12 months. A study by Poplin et al. [6] examined data from administrative records of one large metropolitan department between 2004 and 2009. Despite differences in data collection methods and samples, a consistent pattern of injuries has emerged.

### Nature of Injuries

Nature of injuries, while not exactly mirrored in varying published reports, tend to follow a similar pattern between studies. Musculoskeletal injuries (e.g. sprain, strains) are consistently the leading type of injury among firefighters accounting for more than half [1] and up to three quarters (Jahnke et al. 2013) of reported injuries. Back injuries represent the majority of these musculoskeletal injuries [9]. Low back injury is the most common injury related to early retirement from the fire service [2, 3, 10]. Cuts and wounds are consistently the second most common

**Table 10.1** Nature of injuries among firefighters

| Karter et al. [1] | | Jahnke et al. 2013 [5] | | Poplin et al. [6] | |
|---|---|---|---|---|---|
| Nature of injury | % Injuries | Nature of injury | % Injuries | Nature of injury | % Injuries |
| Strain, sprain, muscular pain | 56.6 | Dislocation, strain, sprain | 76.3 | Sprain, strain | 67.1 |
| Wound, cut, bleeding, bruise | 14.6 | Superficial injury, wound | 13.0 | Contusion, laceration | 18.6 |
| Thermal stress (frostbite, heat) | 4.2 | Thermal stress/heat exhaustion | 0.0 | Inhalation | 0.8 |
| Burns (fire or chemical) | 3.4 | Fire/Chemical Burn, scald, frostbite | 5.2 | Burn | 2.9 |
| Smoke or gas inhalation | 2.5 | Respiratory injury | 0.0 | Medical | 2.7 |
| Dislocation, fracture | 2.7 | Fractures | 1.7 | Fracture, dislocation | 3.3 |
| Burns and smokeinhalation | 1.0 | Eye Injury | 1.7 | Eye injury | 1.9 |
| Heart attack or stroke | 1.2 | Heart attack, stroke | 0.0 | Puncture | 2.7 |
| Other respiratory distress | 1.5 | Concussion, internal injury | 5.2 | Electrical injury | 0.2 |
| Other injury | 12.4 | Other | 0.9 | | |

injuries with a prevalence range of 13.0–18.6 % (Jahnke et al. 2013; [6]). Less frequent injuries include burns, thermal stress, smokeinhalation, eye injuries, and electrical injuries (See Table 10.1).

## Type of Duty

Type of duty being conducted when injured varies somewhat by study completed. Both NFPA [1] and Jahnke (Jahnke et al. 2013) found fireground or fire/rescue activities to be the most frequent type of duty incurring injury (43.5 % and 27.9 % respectively). Only 10.2 % of injuries occurring in the Poplin [6] study occurred on the fire ground. It is unclear why the differences in rates exist, although the authors note their injury reports may be reflective of the departments' tendency to report all Occupational Safety and Health Administration (OSHA) and non-OSHA reportable injuries. Both Poplin [6] and Jahnke (Jahnke et al., 2013) found approximately a third of LODIs occurred during physical exercise (32.9 % and 27.0 % respectively). A category for physical training was not present in the NFPA report [1] which likely accounts for the difference. While concern exists about the risks of on duty

**Table 10.2** Firefighter injuries by type of duty

| Karter and Molis [1] | | Jahnke et al. 2013 [5] | | Poplin et al. [6] | |
|---|---|---|---|---|---|
| Duty | % Injuries | Duty | % Injuries | Duty | % Injuries |
| Fireground | 43.5 | Fire/Rescue activities | 27.9 | Fireground operations | 10.2 |
| Nonfire Emergency | 21.3 | On scene, non-fire call | 17.1 | Physical exercise | 32.9 |
| Training | 10.7 | Training | 6.3 | Training | 11.1 |
| Responding to/ returning from call | 5.5 | Physical exercise | 27.0 | Patient transport | 18.9 |
| Other on duty | 19.0 | Responding to/ returning from call | 8.1 | Other | 28.3 |
| | | Other on duty | 13.5 | | |

exercise, it should be noted that those who regularly engaged in on-duty exercise were more likely to incur an exercise injury, they also were half as likely to incur a non-exercise injury than their peers [5]. The least frequent duties to lead to injury were typically training (10.7 % [1], 6.3 % [5], and 11.1 % [6] and responding to or returning from a call (5.5 % [1], 8.1 % [5], and 18.9 % [6]. Findings from all studies highlight the dangers of firefighting extend beyond the traditional fire ground (See Table 10.2).

### Mechanism of Injury

In their review of department records, Poplin et al. [6] found that more than half of injuries of firefighters (53.1 %) were due to acute overexertion. The second leading mechanism of injury were cutting or piercing injuries (9.8 %) followed by being struck by or caught between objects (8.3 %). Falls accounted for 4.8 % of injuries. Thermal injury (2.6 %), transportation related injuries (2.2 %), and having a foreign object in an orifice (2.0 %) were experienced with similar frequency.

### Location of Injuries on Body

While injuries can occur to any part of the body, the most common injuries among firefighters occur in their lower extremities including the knees (37.3 %), upper extremities including elbows and wrists (23.2 %), and the back or spine (22.0 %) [6]. Nearly all lower extremity and back injuries are sprains and strains. Less frequently, injuries occur to the head, neck and face area (11.2 %), and the torso (4.0 %).

### *Cost of Injuries*

The National Institute of Standards and Technology [11] estimates the cost of firefighter injuries related to workers compensation and medical expenses, long term care, lost productivity and costs related to administration to be between $2.8 and $7.8 billion annually. Improving health and wellness among firefighters has been found to decrease injury related costs. For example, Kuehl and colleagues [12] found that workers compensation claims were significantly lower among Oregon fire departments that implemented the PHLAME program. Jahnke and colleagues [8] estimated a decrease in incident musculoskeletal injuries from 90/1000 firefighters to 54/1000 firefighters if all firefighters had a body mass index of 25 or less. While injuries are costly to the fire service and the communities they serve, appropriate intervention and prevention measures can decrease the costs.

## Musculoskeletal Injury Versus Disease

Musculoskeletal disorders (MSD) are classified as pain or functional disturbances that occur in the nerves, joints, tendons, cartilage or spinal disks. MSDs account for the largest proportion of work-related injuries in the United States [13]. According to data the from the Bureau of Labor Statistics in California, Seabury and McLaren [13] found that firefighters were 3.8 times more likely to experience a MSD than their private-sector peers and when MSDs are incurred, firefighters take twice as long to return to work.

### *Taxonomy of Injuries*

Within the category of "disorders", there are both "injuries" which are the result of an acute trauma or event and "diseases" which are the result of adverse health conditions that occur over time. There is, at times, overlap in the literature between the terms as injuries and disease not always discriminated between. Challenges also arise due to the overlap between injuries and diseases during daily occupational functioning. For instance, there are cumulative trauma disorders that are the result of repeated injuries over time. While injuries can be relatively easy to define and recognize, diseases can be more challenging.

#### Musculoskeletal Injuries

Most firefighters report experiencing an injury at some point during their career [14]. Injuries tend to be easier to identify as they are acute conditions that have clear symptoms such as bruising, pain or inflammation on the site of the injury.

These symptoms most commonly occur at or near the injury site so there is little difficulty in inferring causation. Traumatic musculoskeletal injuries are the result of a sudden release of kinetic (mechanical) injury such as trips, falls, being hit by or with an object, or sprains from stumbling. Resulting injuries often are fractures, contusions, acute strains, sprains, or dislocations. While there are some injuries that do not involve the musculoskeletal system (e.g. ruptured organ, penetrating injury), most injuries among firefighters do include some damage to bones and/or soft tissue [6, 13].

The majority of musculoskeletal injuries among firefighters are not fatal by themselves, but are nearly always part of major traumas experienced on the fire ground which can be fatal due to their fatal bleeding, sepsis, embolism, or other complications. However, most of these injuries are minor (96 %) and do not result in lost days of duty [6]. Compared to injuries incurred off the fire ground, injuries on the fire ground tend to be more moderate or severe.

### Musculoskeletal Disease

Diseases of the musculoskeletal system are the result of slower or repetitive release of energy that strains a relatively weak part of the body. While there are times that the onset of musculoskeletal disease can be discrete (e.g. low back pain), onset usually occurs over time with gradual decline until the inflammation results in discomfort. The causes of these diseases are most typically a conglomeration of several factors that are both occupational and non-occupational.

National monitoring of musculoskeletal diseases is limited as most diseases are non-fatal and therefore not measured in mortality data. Disability data is of some use as these diseases can lead to disability and retirement; however, most datasets do not differentiate between injury and disease which confound the findings. Often, these diseases are not registered or tracked in a systematic way. However, given the unpredictable and extreme work conditions firefighters face, it is not surprising that many firefighters report musculoskeletal concerns.

*Low Back Pain* is a common ailment among the general population with most people reporting experiencing it at some point in their lives. Given the strain and load emergency workers face while fighting fires and performing rescue activities, it is not surprising that this ailment also is common among firefighters. In general, chronic low back pain is considered a disease but there also are instances where the onset is attributable to a specific injury or incident or repeated injuries. Nuwayhid and colleagues [10] prospectively studied the work activities firefighters were engaged in when they experienced low back pain for the first time and found a number of occupational tasks to be associated with onset. In particular, high risk activities included interior operations with a charged hose (OR = 3.26), cutting structures (OR = 6.47), ascending or descending ladders (OR = 3.18), breaking windows (OR = 4.45), lifting heavy objects (OR = 3.07), and looking for hidden fires (OR = 4.32). Low risk activities included connecting hydrants (OR = 0.36), pulling boosters (OR = 0.19), participating in drills (OR = 0.09), or physical training

(OR = 0.16). Overall, there was an increased risk the further firefighters were from the fire station (OR = 0.10) and the closer they were to the fire ground (OR = 3.91). Findings support he intensity and risk associated with fire ground operations.

*Osteoarthritis* is often attributed to heavy physical workload. Firefighters are highly and significantly overrepresented in registries of osteoarthritis of both the hip (2.52, 1.38–4.64) and knee (2.93, 1.32–5.46) [15], suggesting a strong occupational association. As a test of the hypothesis that heavy physical load predisposes to osteoarthritis at both anatomical sites, higher frequencies of osteoarthritis have been generally observed among physically demanding non-firefighting jobs, such as mail carriers, with a particularly strong association noted with farming [15].

## Risk Factors for Musculoskeletal Injuries

Risk factors for musculoskeletal disorders, as for other health risks, can be divided into those that are inherent in the individual, such as age and sex, and those that can be modified.

### *Non-modifiable Risk Factors*

**Age** Aging is associated with a reduction in the frequency injuries among firefighters, not only with frequency but with re-injury rates and circumstances of injury. Older firefighters (40–44 years) tended to experience more falls from height, slips and falls, and incidence during rapid movement [16]. The authors suggest that the reactive behavior of older firefighters may modify their experience, being more cautious, possibly more aware of their limitations and of unsure footing, and using their accumulated experience to guide them in protecting themselves. This lends empirical support and a new twist of meaning to the old saying that "There are old firefighters, and there are bold firefighters, but there are no old, bold firefighters."

**Gender/Sex** Female firefighters experience a higher rate of injury than male firefighters, historically [17]. However, these studies are relatively old and may not reflect current selection, training, and recruitment practices. The conventional explanation for the finding is that women, on average, are smaller and have less upper-body strength than men, and are more likely to have diminished health capacity affecting performance with age [18], and so are therefore at a disadvantage when full strength is required or when exerting force in awkward situations. These data, while useful and valid for their time, are now 10 years old and may or may not be valid given the introduction of performance-based physical testing and other efforts to create gender-neutral preplacement screening for the fire service.

Current evidence suggests that women in the fire service currently are actually in better health and more fit than their male peers [19]. What is needed, if the issue requires further investigation, is a comparative study of injury rates for subjects at comparable levels of performance. The design of traditional turnout gear was often ergonomically mismatched to female firefighters and has required redesign in such features as size, fit to shape, and placement of pockets (Fig. 10.1).

**Past Health Habits and Medical History** Jahnke et al. demonstrated that a past history of smoking was a powerful risk factor for injury in firefighters (1.8; 1.31–2.99) [5]. A past history of smokeless tobacco had a slightly elevated risk, but did not achieve statistical significance (1.19; 0.70–2.04) [5]. In another study, a self-reported history of low back pain was been shown to be the strongest predictor of current low back pain among firefighters (Odds Ratio 44.90) [20].

## *Modifiable Risk Factors*

**Ergonomic Factors and Work Environment** The biomechanical factors that predispose to injury in firefighters are well known. For slips, trips, and falls, factors include personal factors such as body mass, fatigue, experience, and training, and occupational factors such as heavy and bulky personal protective equipment, impaired vision, heatstress, and slippery, uneven, or unstable surfaces [21]. The high rate of low back pain in firefighters can be partially attributed to high risk work activities that firefighters are expected to perform. Tasks associated with high risk of back injury include cutting into structures, axe work and cutting into walls during overhaul, breaking windows, holding hoses, rescue, and lifting objects greater than 18 kg. Tasks associated with low risk of back injury include connecting hoses, pulling hoses, and training activities. This pattern suggests that attributing low back pain primarily to lifting activities is an insufficient explanation, but is consistent with a contribution from other biomechanical and ergonomic factors (e.g. lower trunk torsion, such as when breaking a window with an axe) [10]. For comparison with the general population, Wai, Roffey, Dagenais, and colleagues recently conducted several comprehensive systematic reviews assessing the causal relationship of occupational factors and the risk of low back pain in the general population [22–29]. Surprisingly, very few occupational factors were causally related to low back pain. Evidence against, conflicting evidence, or insufficient evidence for a causal relationship with low back pain was found for awkward postures, bending or twisting, carrying, manual handling or assisting patients, pushing or pulling, sitting, and standing or walking. Moderate evidence for a causal relationship with low back pain was noted only for heavy lifting at the workplace. Whether similar relationships exist in firefighters is unknown.

**Obesity** Obesity is a major risk factor for injuries resulting in absence among firefighters, with a BMI-related increase in risk on the order of three to four depending on obesity category [30]. There are many obvious ergonomic reasons why this might be the case, among them lower back and core muscle endurance [31], increased effort requirement, physical bulk, and impaired heat transfer.

**Psychosocial Factors** An important risk factor that cuts across various types of injury and associated with fatalities is time pressure, which is associated with a higher ratio of fatalities per turn-out events except, when there is a human rescue involved. When human rescue is involved, the proportion of firefighter fatalities is low, implying that firefighters are acting carefully when protecting or rescuing people despite time pressure, but are not so cautious, and perhaps are even impatient, when they are working under time pressure to protect property [32]. Self-reported depression was a highly-significant risk factor for injury [5]. Personality types are associated with increased frequency and severity (mostly, duration) of injury, as is the case in the general population. Depressed, anxious, and asocial personality types are at greater risk, but since there are also behavioral correlates to these personality profiles (such as alcohol and substance abuse), and therefore a strong potential for confounding, personality type cannot be easily used for prediction and to do so in individual cases would raise serious issues of employment law and fairness.

**Physical Fitness** Numerous physical fitness and human functional performance measures have been associated with musculoskeletal injuries in the general population. Some of these measures have been studied in firefighters and are described below. Cady et al. demonstrated that physical fitness level as measured by five domains (upper and lower body isometric strength, total spine flexibility, heart rate recovery following stationary bike exercise, physical work capacity, and diastolic blood pressure during cardiovascular exercise) was inversely correlated with occurrence of back injuries in firefighters of Los Angeles County, CA [33]. That is, firefighters who were more physically fit experienced fewer low back injuries than their less fit counterparts. In a sample of 793 firefighters from San Diego Fire Rescue, CA, Mayer et al. noted that firefighters with a history of low back pain were physically deconditioned compared with those without history of low back pain [34]. For example, firefighters with a history of low back pain had significantly ($p<0.05$) lower isometric back muscular endurance, abdominal muscular endurance, upper and lower body muscular strength, upper body muscular endurance, and aerobic capacity; and higher body fat and percentage with hypertension/borderline hypertension. 83 % of firefighters had isometric back muscular endurance times below the recommended target time and 37 % were more than 1 standard deviation below the target time [35]. In the general population, deconditioned core trunk muscles have been shown to be strongly associated with low back pain. Individuals with low back pain exhibit a loss of strength and endurance in the trunk extensor muscles [36], atrophy of the lumbar multifidus muscles [37], fatty infiltration of the lumbar muscles [36], abnormal activity patterns of the core trunk muscles [38], and spinal instability [39]. An inverse relationship exists between isometric back extension endurance and the likelihood of future low back pain

episodes [40], and good dynamic trunk extensor endurance is predictive of a decreased incidence of work disability [41]. However, the implications of these relationships on clinically-relevant outcomes for low back injury prevention in firefighters have not been adequately studied. Recent evidence suggests that performance on the Functional Movement Screen is linked to injury risk in firefighters [42] and other workers, such as military [43]. The Functional Movement Screen reliability assesses functional movement quality through observer rating of seven tasks—in-line lunge, hurdle step, deep squat, quadruped rotary stability, active straight leg raise, shoulder mobility, and trunk stability push-up [44]. A score of $\leq 14$ on the Functional Movement Screen has been shown to be associated with a higher risk for musculoskeletal injuries in firefighters enrolled in a training academy [42].

## Screening for Musculoskeletal Injuries

The known risk factors for firefighter injuries that were discussed in the previous section can be assessed through various standardized screening tests, including self-reported health history, physical function, and psychosocial questionnaires, objective measurement of anthropometric characteristics and physical performance, and clinical examination. Given the wide variety of available screening tests, individual fire departments may customize a battery of tests to suit department-specific needs. Examples of a comprehensive screening framework for firefighters and functional capacity assessments are described in this section.

**Fire Service Joint Labor Management Wellness Fitness Initiative** Considering that firefighters are at high risk for many types of injuries and illnesses, the International Association of Firefighters (IAFF) and the International Association of Fire Chiefs (IAFC) developed the Fire Service Joint Labor Management Wellness Fitness Initiative (WFI) [2, 3]. The WFI provides guidance for fire departments to implement properly designed health, wellness, and fitness programs to assist firefighters in becoming more physically able to safely carry out their work duties and usual activities of daily living. The WFI consists of five major components [2, 3]: (1) medical evaluation, (2) fitness testing and exercise, (3) rehabilitation, (4) behavioral health promotion, and (5) data collection. Within the fitness testing component, several fitness domains are assessed through standardized screening procedures: Upper extremity strength (isometric grip and upper arm strength), lower extremity strength (isometric leg/back lift), lower extremity power (vertical jump), core muscular endurance (Plank Test), upper body muscular endurance (push-ups), lower extremity and trunk flexibility (Sit and Reach Test), cardiovascular endurance, and subcutaneous body fat. While isometric back extension muscular endurance has been shown to be a predictor of future incidence of low back pain and work disability following back injury in the general population and other workers [40, 41], the WFI does not provide guidance for assessment of this fitness domain.

**Functional Capacity Evaluations** Functional capacity evaluations (FCEs) are systematic, comprehensive, and standardized methods to assess a person's physical functional status in work-related tasks [45, 46]. Many FCE tests are commercially-available for assessment of a wide range of occupational demands, such as the EPIC Lift Capacity Test, Ergo-Kit FCE, ErgoScience Physical Work Performance Evaluation, Isernhagen Work Systems FCE (WorkWell FCE), and Progressive Isoinertial Lifting Evaluation (PILE). Generally, the validity of these tests for determining risk for injury or recovery from injury has not been established [45, 46]. Furthermore, to our knowledge, none of the major commercially-available FCEs mentioned above have been formally assessed in firefighters and published in the peer-reviewed literature. The Candidate Physical Ability Test (CPAT) is an FCE that has been developed specifically for firefighters [3]. The CPAT is a series of standardized tasks consisting of simulated line-of-duty firefighting activities. During the CPAT, the candidate wears a 50-lb vest while completing eight tasks: Stair Climb, Ladder Raise and Extension, Hose Drag, Equipment Carry, Forcible Entry, Search, Rescue Drag, and Ceiling Pull. While performance on CPAT is linked to cardiovascular and musculoskeletal physical fitness [47], the CPAT has not been validated to determine risk for injury or recovery form injury. Given the paucity of evidence regarding the validity of FCEs, particularly for firefighters, the task force of the Second National Fire Service Research Agenda Symposium recommended further research on this topic [48].

## *Interventions for Musculoskeletal Injury Prevention*

Very little high-quality evidence is available on interventions for prevention, reduction of risk, or reduction of adverse consequences to help guide decision-making related to musculoskeletal injuries in firefighters. We did not find any randomized controlled trials for the vast majority of possible interventions, and uncovered only one randomized controlled trial that evaluated the effectiveness (in terms of clinically-meaningful injury outcomes) of an exercise intervention for preventing musculoskeletal injuries in firefighters. The randomized controlled trial and preliminary studies are discussed in this section.

For comparison purposes, evidence from recent systematic reviews assessing intervention effectiveness in non-firefighters suggests that physical exercise is modestly effective for prevention of low back injury in the workplace and in working age individuals [49, 50]. Many other interventions, such as ergonomic interventions, low back support belts, and comprehensive back schools were deemed to be ineffective [49, 50]. We did not find any systematic reviews on interventions for prevention of other types of occupational musculoskeletal injuries in the general population.

**Exercise Interventions** In a randomized controlled trial, Hilyer et al. demonstrated that flexibility exercise training was effective in reducing the severity,

though not the frequency, of musculoskeletal joint injuries among firefighters [51]. In a non-controlled observational study, Cady et al demonstrated that implementing a general physical fitness program reduced low back injuries and costs in firefighters [52]. In a more recent non-controlled observational study, Peate et al. demonstrated that core stability exercise training was effective (compared with historical data) in reducing the frequency (by 44 %) and severity (62 % reduction in lost work time) of musculoskeletal injuries in firefighters [53]. Small but significant effects resulting in increased risk were observed for female gender, prior injury, and especially age [53]. Other exercise intervention studies without clinically-meaningful injury outcome measures that have been conducted in firefighters may also provide useful information for clinicians and future research. In a randomized controlled trial with firefighters, Mayer et al. observed that a 24-week worksite exercise program consisting of progressive resistance exercises for the back and core stability exercises was safe and effective for improving back and core muscular endurance [54]. Considering the link between poor back muscular endurance and increased risk of future back pain [40], exercise programs designed to improve back muscular endurance may be advantageous in reducing back injuries in firefighters, though this hypothesis has not been studied in randomized controlled trials. In a recent controlled study with firefighters, Beach et al. noted that 12-weeks of a general physical exercise intervention or an exercise intervention targeting specific movement qualities resulted in significant improvements in various physical fitness parameters compared with control [55]. Neither exercise group displayed improvements in lumbar spine biomechanical loading characteristics compared with control. The clinical relevance of these findings for firefighter injury prevention is unknown. The preliminary findings discussed above suggest that exercise interventions are potentially beneficial for musculoskeletal injury prevention in firefighters. Numerous exercise approaches are possible for implementation in firefighters ranging from general fitness programs to those focusing on specific areas of the body or specific areas of deficit found at screening. However, no evidence is available that indicates one type of exercise is superior to another in terms of clinical effectiveness and injury prevention. Therefore, decisions to implement on-duty exercise programs may be based on other factors, such as safety, individual or departmental preferences, and known barriers or facilitators. Some suggestions to improve safety and adherence on on-duty exercise programs in firefighters include: provide adequate supervision, schedule fitness activities during times when a lower call volume is expected if possible, perform exercise at submaximal levels but at intensity levels high enough to stimulate positive physiological adaptations, do not exercise to exhaustion, include aerobic activities to improve cardiovascular health, train with exercises that enhance mobility and stability, maintain proper hydration levels during exercise and replenish energy stores following exercise, train with a group or partner to enhance cohesiveness and encourage friendly competition, provide individual and group incentives for exercise, and emphasize the link between exercise, physical fitness, and job performance through appropriate education [54, 56].

**Fire Service Joint Labor Management Wellness Fitness Initiative** Preliminary analysis presented in the third edition of the WFI indicates that implementation of a WFI-based general wellness and fitness program in firefighters reduced the negative impact of musculoskeletal injuries over a 7-year period (1998–2004) [3]. Fire departments (Fairfax County, VA, Indianapolis, IN, Los Angeles County, CA, Phoenix, AZ) that implemented a WFI-based fitness program experienced a 28 % reduction in lost work days related to injury claims and a 23 % reduction in average cost per claim compared with the 7-year period before implementation. In contrast, fire departments (Austin, TX; Calgary, Alberta; Miami-Dade, FL; Seattle, WA) that did not implement a WFI-based fitness program experienced a 55 % increase in lost work days related to injury claims and a 35 % increase in average cost per claim compared with the 7-year period before implementation. While this analysis was not the result of a controlled prospective study and therefore is highly prone to bias, it provides useful information for planning of future clinical trials.

## *Recommendations*

Given the nature of fire and rescue activities, it is not surprising that injuries are a concern for first responders. Fitness and training have been found to reduce rates of non-exercise injuries which suggest fire departments should invest in health and wellness programs for their personnel. Given the high rate of injuries that occur during physical activity, it also is incumbent on departments to provide proper training and resources for firefighters so they can be adequately prepared to respond to the needs of their communities. Training for safely operating on the fire ground also is important for firefighters at all ranks to reduce risk.

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# Chapter 11
# Psychosocial Hazards and Risks

**Michel A.S. Larivière, Zsuzsanna Kerekes, and Danielle Valcheff**

## Introduction

Firefighters are regularly exposed to physical hazards that are uncommon in other occupations and only rarely part of normal human experience: natural and man-made disasters, flames and extreme heat, poor ventilation, structural collapses, and toxic chemicals [1, 2]. When asked to describe the dangers of this occupation, the general public is most likely to think of newscasts showing explosions, falls, people trapped in buildings, and rescues [1]. Based on data from the U.S. Bureau of Labor Statistics [3], Meyer et al. [4] identified a fatality rate in this occupation that was 4.5 times greater than the national average. Moreover, it was recently estimated that 45.2 % of American firefighters were injured while on the job in 2013 [5]. The longstanding recognition of firefighting as a dangerous occupation is certainly not surprising, but it is not limited to physical hazards.

The International Labour Organization [2, 6] and the World Health Organization [7] emphasize that work organization, management and design, working conditions, and workplace interpersonal relations are the main environmental, social and organizational sources of work-related distress. Within this broad context, psychosocial hazards are risk factors that can be harmful to physical and psychological health. While the effects of psychosocial hazards can be experienced organizationally [8], this chapter will focus on how psychosocial hazards affect individual workers in firefighting organizations.

With this framework in mind, we intend to identify and characterize the primary psychosocial hazards associated with firefighting. As will be seen in the following pages, these can lead to short-term distress and also chronic impairment. A secondary focus of this chapter is to identify the causal and mediating factors of

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these impairments with a view of possibly informing intervention efforts. In doing so, we endorse the view that service-related illnesses and injuries may not immediately appear "in the line of duty". However, the hazards in question can lead to delayed-onset outcomes that can range from a slightly diminished quality of life to mortality.

Although research on firefighting is a fairly recent undertaking, interest has grown rapidly. This is attributable in part to the tragic events of September 11, 2001. While research across most academic disciplines has increased with each decade [9], interest in psychosocial factors has been particularly augmented by increased sensitivity and recognition of mental health issues. Moreover, the stigma attached to mental illness is slowly dissipating [10, 11] and as a result, researchers have been able to offer more accurate estimates on the prevalence of specific illnesses. Therefore, health care resources are better deployed to better serve these needs [12, 13].

In the general population of the United States, the lifetime prevalence rate for any mental illness has been estimated at between 46.4 % [14] and 48.6 % [15]. Worldwide, it is estimated at 33.4 % [15].

Of course, rates fluctuate according to age, gender and culture [16, 17]. For instance, youth tend to experience more substance use disorders (11.9 %, [17]) and major depressive episodes (8.2 %, [17] to 9.1 %, [18]) than adults aged 25 or more. Women are more prone to mood and generalized anxiety disorder while men are more likely to suffer with substance-related disorders [17].

There is good reason to believe that prevalence rates for mental illness also vary according to one's occupation and the duties performed therein [19]. A good case in point is posttraumatic stress disorder (PTSD) [20], a condition we examine later in this chapter. As can be seen in Table 11.1, the lifetime prevalence rate for PTSD in the general population of North Americans is estimated between 6.8 % [14] and 9.2 % [23]. In Europe, the lifetime prevalence rate for PTSD is 1.3 % [24], which is similar to Asia at 1.2 % [25, 26] and Africa at 2.3 % [27].

There is limited information on the lifetime prevalence rates for PTSD among firefighters (see Table 11.2). However, one of the most often cited articles, published by Corneil et al. [34], estimated a *point* prevalence rate of 17 % in a Canadian sample of firefighters (n = 625) and 22 % in a sample of American firefighters (n = 203). However, this particular study utilized the Impact of Events Scale, which, despite good psychometric properties, is not a diagnostic instrument for PTSD. In any event, Corneil et al. [34] highlight a divergence between lifetime rates in the general population (e.g. 6.8–9.2 % in North America) and point prevalence rates among American firefighters at approximately 22 %.

Notable is the number of firefighters who, while not meeting diagnostic criteria for PTSD, still report suffering with some of its symptoms such as flashbacks, nightmares, hypervigilance/hyperarousal and avoidance [31, 35]. In a sample of German firefighters, Wagner et al. [35] estimated that 18.2 % of firefighters experience these symptoms. Firefighters also appear to be at greater risk for depression [36] and alcohol problems, especially binge drinking [36]. Higher rates of substance abuse may be an artifact of poor coping and often comorbid with other mental illnesses such as PTSD.

**Table 11.1** Prevalence rates for PTSD in the general population[a]

| Estimated[b] period prevalence rate[c] | Estimated lifetime prevalence rate[d] | Country (n) | Study references |
|---|---|---|---|
| 1.1 % (12-months) | | Europe (Spain, Italy, Germany, the Netherlands, Belgium, France) (21425) | Darves-Bornoz et al. [21] |
| 1.3 % (12-months) | | Australia (10641) | Creamer et al. [22] |
| 2.4 % (1 month) | 9.2 % | Canada (2991) | Van Ameringen et al. [23] |
| 3.5 % (12-months) | | USA (9282) | Kessler et al. [16] |
| | 6.8 % | USA (9282) | Kessler et al. [14] |
| 0.7 % | 1.3 % | Germany | Perkonigg et al. [24] |

[a]All the studies used in these tables used questionnaires, scales or interview methods, which permitted the identification of DSM-IV criteria for PTSD
[b]Estimated means at prevalence rates if one has information on samples of the population of interest
[c]Period prevalence rate means the proportion of the population that has the characteristic (as per the diagnostic schedule was delivered to the screened population in the mentioned studies) at any point during the mentioned, specific timeframe (usually 12 months) and used in conjunction with LTP
[d]Lifetime prevalance (LTP) means the proportion of a population who, at some point in their life up to the time of assessment, experienced the illness as per established diagnostic criteria

**Table 11.2** Period prevalence rates for PTSD in the firefighter population[a]

| Estimated period prevalence rate | Country (n) | Study references |
|---|---|---|
| 4.2 % (current) | USA (142) | Meyer et al. [4] |
| 4.4 % | The Netherlands (494) | Witteveen et al. [28] |
| 5.0 % | USA (131) | Del Ben et al. [29] |
| 5.4 % | Taiwan (410) | Chen et al. [30] |
| 6.5 % | UK (31) | Haslam and Mallon [31] |
| 7.3 % | Worldwide 14 countries (5680) | Berger et al. [20] |
| 12.0 % | Australia (52) | Bryant and Guthrie [32] |
| 14.3 % (1-month) | USA (3232) | Perrin et al. [33] |
| 17.0 % | Canada (625) | Corneil et al. [34] |
| 22.0 % | USA (203) | Corneil et al. [34] |

Definitions as per Table 11.1
[a]The other studies referenced in these tables used questionnaires, scales or interview methods, which permitted the identification of DSM-IV criteria for PTSD except Corneil et al.'s study [34]

## Psychosocial Characteristics

Researchers have demonstrated that firefighters may be at greater risk of developing certain mental health issues as a result of their work. This section will first discuss relevant contextual factors and then the impact of hazards that may contribute to undesirable psychological outcomes.

### *Personality Profile*

Given its unique occupational demands and risks, we might ask what "type" of person firefighting might attract. "…firefighting is high hazard work, but it is unique beyond this. In most high hazard work situations, the goal is hazard avoidance. In contrast, for firefighting, the principal work activity is hazard engagement…" ([37] p. 1171).

There are of course many reasons why individuals select one occupation or profession over another. Some of these influences may be simply based on circumstance. Others are social in nature and include political and economic factors such as public funding levels and labor supply/demand. However, the selection of one occupation or profession over another also stems from personal differences, which are rooted in a person's lived experiences and individual psychology. Helpful in understanding the latter is personality and vocational theory.

Researchers have proposed that personality is composed of stable traits or dimensions that cut across age, gender and culture [38]. These dimensions have been referred to as "factors" and based on considerable research [39], there is general consensus on a "five factor model" (FFM). Also known as the "Big Five", the FFM taxonomy includes: Extraversion, Agreeableness, Conscientiousness, Neuroticism, and Openness. Although widely accepted as an appropriate measure of normal personality, the Big Five did not emerge without criticism. Concerns were expressed about limiting an individual to five traits, and overlooking a variety of behavioral and contextual issues [40]. Despite this, the Five Factor Model has evolved into one of the most widely used frameworks for understanding personality in the twenty-first century [41]. Mindful that all traits fall on a continuum and that human behavior is also context dependent, we offer a brief description for each of the Big Five personality factors [42]:

- Extraversion: Focused on external stimuli, tend to be more social/gregarious, assertive, active, and more likely to seek excitement
- Agreeableness: Trusting of others, inclined toward open and honest communication, altruistic, cooperative/compliant, modest, and sympathetic toward others
- Conscientiousness: Methodical and attentive to detail, favor order and structure, dutiful, goal oriented, disciplined, and measured/considered
- Neuroticism: Prone to anxiety, depression, and anger/irritability. Tend to be more self-conscious/vulnerable, emotional, greater difficulty tolerating unpleasant events
- Openness: Tolerant of novelty, new ideas/values, appreciation of aesthetics, feelings, as well as complex experiences rather than standard routines

A few studies have attempted to identify a "firefighter profile". When compared to the general population, firefighters, including forest firefighters, have been found to score higher on scales measuring Extraversion [43, 44] and, in a sample of forest firefighters, scored lower on scales measuring Openness, Agreeableness and Conscientiousness [43]. There is also literature indicating that firefighters score higher on a measure of sensation-seeking [44, 45].

Traits related to a "firefighter profile", such as high Extraversion and low Openness, have been associated with "enthusiastic firefighting"; a term proposed in research by Fannin and Dabbs [46]. Here, participants were metropolitan firefighters whose duties included emergency medical services (EMS) work. Participants completed questionnaires and answered open-ended questions after which their performance was rated by senior firefighters ("expert judges") on domains including competence, enthusiasm, and unnecessary risk-taking. A high rating on "enthusiastic firefighting" indicated the participant was perceived as skilled and enthusiastic but at the highest levels, prone to unnecessary risk-taking. Lower levels of Openness and Agreeableness were related to a greater preference for firefighter vs. EMS duties [46].

### *Occupational Norms*

In the same way humans have personalities, some theorists have proposed that occupations also have "personalities". A well-known conceptualization of this idea is found in Holland's theory of vocational choice [47] where six dimensions are proposed to describe individuals and workplaces: Realistic, Investigative, Artistic, Social, Enterprising, and Conventional. Job satisfaction is predicted when there is compatibility between an individual's personality and an occupation's "personality" [48, 49]. Using Holland's theory, all occupations can be assigned to a category described by a three-letter code (e.g., ESA, CSR, SEC, etc.).

The code assigned to the occupation of firefighting is RES: Realistic, Enterprising, and Social, which helps understand the link between individual personality, vocational engagement, job duty preferences, as well as the working environment. Persons scoring high on the Realistic dimension in Holland's taxonomy are likely to be perceived as "doers". They tend to prefer using tools and equipment since this appeals to their practical side. This personality type is prone to being self-effacing, conforming, more materialistic, and genuine. For their part, Enterprising individuals enjoy work where they can lead, persuade, energize and influence others. They more often show self-assuredness, ambition, and confidence in their ability to handle critical situations. They value challenge, adventure and excitement. The Social type reflects a personality style that appreciates social interaction, teamwork, and helping others. They would be perceived more often as cooperative, empathic and friendly [48, 49]. These characteristics as a whole help explain the attraction to firefighting held by some. It also offers insight into how personality itself can be a protective or risk factor for work-related distress (e.g., how a highly artistic and investigative individual may not feel as well suited to firefighting duties). Meta-analytic work by Barrick et al. [48] identified that Extraversion is strongly related to Holland's Enterprising and Social typology. This finding speaks to the link between basic personality features and vocational interests and potentially, job satisfaction.

The psychosocial hazards associated with firefighting are better understood within a broader cultural framework. Due to the nature, respect [50], and visibility of their work, public perceptions and portrayals in popular culture have created a

firefighter image that has remained largely stable for over a century [51]. Cooper [51] conducted an analysis of how firefighters were represented in the nineteenth and early twentieth century (i.e., advertisements, photographs, prints, journals, movies, etc.). Emerging from her analysis was the portrayal of "ideal manhood", the quintessence of the masculine, hero-warrior [51]. Although much has changed in society since the early twentieth century, there is overlap between historical and contemporary representations of firefighting in popular culture. Research focusing on fictional representations of firefighters in film describes a workforce consisting of heterosexual, working class Caucasian males [52]. Furthermore, films and television series included in Willis' [52] analysis portrayed firefighters as protagonists, who were usually invulnerable to physical or psychological harm.

There are many commonalities between public perceptions of firefighter subculture and actual firefighter subculture, but there is limited research on the concepts of "brotherhood" and camaraderie [53]. Often captured in popular cultural references to firefighting is camaraderie, which encompasses mutual trust, cooperation, group acceptance, and collective goals [54]. Willis [52] also discusses hierarchy as an important part of firefighter subculture. The hierarchy can resemble a military-like chain of command that is perhaps most useful at the scene of an incident [52, 55]. Hierarchy can offer a sense of predictability and stability inside and outside of the firehouse. Minimizing doubt is a useful ally for firefighters when they are expected (by the public and themselves) to sacrifice their lives for others. To engage fully in a potentially self-sacrificing role, clear roles and commands, as well as the capacity to filter disturbing cues are necessary. The uniform itself assures visibility and confers a shared sense of identity.

The hierarchy is perhaps felt most strongly among new hires during their probationary period. There is often an initiation stage for probationary firefighters, who are commonly referred to as "probies" or "rookies". Probie initiation period is a rite of passage where they must prove their worth to fellow firefighters [56]. Rituals are used in this subculture not only for rookie induction but in the creation of a "home at the firehouse" among the "brothers". The firehouse is not simply a location where firefighters wait for an alarm but symbolizes a structured, united, and organized life that is designed for readiness. Other traditions or rituals that encourage group cohesion can be observed at training inductions, retirements, and funerals [52].

Young firefighters are not the only group that must work hard for acceptance and inclusion. Gender and sexual minorities also face a unique set of challenges. Women were late entrants into this occupation with the first applicant hired in 1974. By the 1980s, over 500 women were firefighters in the United States [57]. However, this still represents only 3 % of the workforce [50]. Similar to early-career firefighters, women report they expend extra effort to demonstrate they are capable and competent enough to earn the respect and acceptance of their co-workers [50]. In some reports, they are excluded from social and training opportunities [50], which potentially represent a risk factor during emergency situations.

A higher incidence of harassment and discrimination toward female firefighters has been reported. In research by Rosell et al. [57], 208 female firefighters from 37

departments were surveyed and 58.2 % reported sexual harassment. Predictably, these respondents experienced more job stress, sexual stereotyping, and acts of violence than non-harassed female firefighters. Consequently, they expressed more fear about attending the workplace and were more frequently absent from work. These researchers identified that one-half of these firefighters did not report incidents of harassment to their managers and among those who did; only about one-third received a formal response. Furthermore, Yoder and Aniakudo's [58] work described how African American female firefighters experienced a persistent pattern of social exclusion. Subjects reported little constructive feedback and instead, experienced "silent treatment" and stereotyping from their peers. The supervision from managers was perceived as excessive and punitive. They stated that white female firefighters coped better with gender stereotypes given the absence of race related issues. Sinden et al. [50] emphasized the importance of social inclusion in terms of decreasing anxiety and job strain while improving job satisfaction and creating a positive work environment for female firefighters. Despite reporting alienation, female firefighters have been reluctant to speak negatively about their male co-workers and report that attitudes toward female firefighters have been improving [50].

Sexual minorities represent another group whose trajectory toward acceptance in firefighter subculture may deviate from the prototypical heterosexual male firefighter. Firefighter subculture has been described as intolerant of male homosexuality where homophobic remarks are openly and frequently expressed [59]. Moreover, Fisher [60] expressed that homosexual individuals would be less inclined to disclose their sexual orientation or share details of their personal lives with their work colleagues if the work environment is not perceived as supportive. Interestingly, lesbian firefighters do not appear to be at greater risk of being peripheralized despite their double minority status (i.e., being female and homosexual). In fact, there is evidence suggesting it is easier for lesbian firefighters to assimilate into firefighter subculture than it is for gay men. Moreover, lesbians seem more readily accepted than heterosexual females [59]. Lesbians have reported they were better able to "fit in" the masculine dominated culture, were perceived by males as more capable, and were less sexualized than heterosexual females [59].

In summary, both social (e.g., culture) and psychological (e.g., personality) factors provide an important context for understanding not only the ethos of firefighting but the manner in which psychosocial hazards can be experienced. The literature suggests that public perceptions of firefighters are largely favorable and that the subcultural context and standards appear to play an important role in maintaining firefighter identity. However, this identity may risk the exclusion of minority groups.

## Psychosocial Hazards

Having offered epidemiological, psychological and cultural contexts to firefighting, we turn to specific psychosocial hazards in this occupation.

## *Taxonomy of Hazards*

A useful taxonomy has been offered by Leka and Cox [8] who listed a number of potential psychosocial hazards that would be applicable to any occupation. In doing so, Leka and Cox ([8] p. 124) were sensitive to "the significant changes (...) in the world of work that have resulted in emerging risks and new challenges in the field of occupational health and safety". Their efforts will help frame the discussions in this section of the chapter. Psychosocial hazards in the workplace can include the following ([8] p. 125):

- *Job content:* lack of variety or short work cycles, fragmented or meaningless work, under use of skills, high uncertainty, continuous exposure to people through work
- *Workload and work pace:* work overload, machine pacing, high levels of time pressure, continually subject to deadlines
- *Work schedule:*shiftwork, night shifts, inflexible work schedules, unpredictable hours, long or unsociable hours
- *Control:* low participation in decision-making, lack of control over workload, pacing, shiftwork, etc.
- *Environment and equipment:* inadequate availability, suitability or maintenance; poor environmental conditions such as lack of space, poor lighting, excessive noise
- *Organizational culture and function:* poor communication, low levels of support for problem solving and personal development, lack of definition, or agreement on, organizational objectives
- *Interpersonal relationships at work:* social or physical isolation, poor relationship with superiors, interpersonal conflict, lack of social support
- *Role in organization:* role ambiguity, role conflict, responsibility for people
- *Career development:* career stagnation and uncertainty, under promotion or over promotion, poor pay, job insecurity, low social value to work
- *Home-work interface:* conflicting demands of work and home, low support at home, dual career problems

## *Job Content*

Helpful in the understanding of job content is the Job Demands and Control model (JDC) conceptualized by Karasek [61], which focuses on two critical aspects of work. As the term suggests, job demands speak to issues such as workload (i.e., quantitative demands), work pace, time pressure, task complexity, dangerous work, and other factors that draw on a worker's personal resources for coping. Job demands are duties that require effort and can lead to negative physiological and psychological consequences. In that regard, some authors have underlined that modern workers face increasing mental and emotional demands and concomitantly fewer physical demands [62].

Sluiter [63] highlights the special demands of firefighting, referring to "driving with flashing lights and sirens, working under time pressure when human life is at stake, and making complex decisions in hazardous environments" (p. 434). During the course of their work, firefighters may need to assist highly vulnerable individuals with special medical needs such as those seen in a growing elderly population [64]. Bryant and Harvey [65] point out that firefighters witness violent deaths, provide or assist in rescue operations, support burn victims and provide medical attention to victims of accidents (which can include their colleagues). It is important to mention that failed attempts at helping or assisting victims brings its own set of challenges, not the least of which is coping with feelings of inadequacy, diminished self-esteem, and lower confidence [66].

Professional firefighting is changing and its practitioners are increasingly trained in emergency medical services [55]. This shift may decrease the likelihood of tedium (e.g., inactivity between and during calls), and afford new learning opportunities. On the other hand, one might speculate that integrated firefighting/EMS services predispose its workers to burnout or exhaustion. Research would be required to better understand the outcomes for this type of cross-training.

### *Workload and Pace*

In the line of duty, firefighters often work under extreme time pressures [37, 63] where life and property hang in the balance. The demands can have negative effects on cognitive abilities, especially on sound decision-making [67]. Volunteer firefighters [54] and wildland firefighters may be especially susceptible to these effects, since their firefighting duties may be required during their time off or after completing other work and home obligations. What is more, firefighters may be called to work long shifts and irregular schedules [68].

When responding to a call, Ponnelle (2003 cited by [69]) described that firefighters move through three distinct phases. The *pre-operational* (or anticipation) phase occurs between the alarm and the firefighter's arrival. During this time, firefighters will tend to report stress-related reactions but also heightened concentration. The *operational* phase begins with the worker's arrival on site and includes facing the fire/crisis. Here, firefighters report experiencing fear, distress but also motivation. They are also more likely to feel and/or show more aggression. The third and final phase is the *post-operational* phase, where firefighters are likely to express fatigue, excitement, relief, anger, or discouragement.

Unique stressors are inherent to each of these phases; however, it is worth recalling that an estimated 33 % of firefighter deaths are related to job stress [70]. This phenomenon certainly overlaps with cardiovascular problems [71], which are responsible for 45 % of on-duty deaths [72]. The pre-operational phase is where 13.4 % of on-duty deaths occur as a result of coronary events. A significantly higher proportion of deaths occur during the operational phase (32.1 %). The post-operational phase accounts for 17.4 % of on-duty deaths according to Kales et al. [72].

Other emergency service workers share similar stressors to firefighters (e.g., alarm reactions, disrupted sleeping patterns, and danger) but they are less likely to encounter toxic/noxious environments, heat stress, and extended physical exertion [72]. Moreover, some have pointed out that supervisors and the general public [71] have higher expectations for this occupational group. It may be the confluence of these factors that induce greater mental stress in firefighters and therefore results in more frequent heart-related problems than other emergency service workers [73].

### *Work Organization and Shiftwork*

Firefighting is a 24/7 enterprise that requires constant availability and hence, rotating work schedules. Shiftwork represents a major risk factor in all occupations and there is increased sensitivity to its health effects. From a physiological perspective, shiftwork is highly disruptive to natural circadian rhythms, which in turn affects the whole person. In the short-term, shift workers more frequently report insomnia, abdominal discomfort, fatigue, changes in behavior (e.g., aggression, sensitivity), decreased work performance, impaired cognitive abilities, and general discomfort than non-shift workers [36, 74]. In the longer term, shift workers experience higher rates of cardiovascular disease [75], diabetes, and obesity [76, 77], fatigue, and depression [78]. Lusa et al. [79] identified that sleeping problems occur after a 50-h workweek.

Individuals tolerate shiftwork differently based on a number of factors such as genetics, age, personality and actual shift schedules [74]. Regarding the latter, outcome studies appear to support the use of rapid forward rotating shifts, while discouraging permanent night work [80].

### *Job Control*

Job control or "decision latitude" is the second part of Karasek's [61] Job Demands and Control model and refers to perceived autonomy on the job. In essence, job control is a worker's sense of influence regarding their tasks [61, 81]. It includes decision authority (e.g., the flexibility to make job-related decisions) and skill discretion (e.g., the flexibility to use one's skills on the job). Karasek [61] hypothesized that a combination of high job demands and low job control would produce job strain. High strain employment has been identified as a risk factor for cardiovascular disease, depression, exhaustion, burnout, and job dissatisfaction [41, 61]. Perceived job control lowers stress levels and the risk of burnout [69].

Firefighting has been assessed as a high demand/low control occupation and therefore job strain is anticipated [30, 56, 69, 82, 83]. It follows that negative physical and psychological consequences would be anticipated according to JDC theory.

The perception of low control over their job [83] might be explained, at least partly, by a hierarchical and paramilitary work context that is governed by extensive rules and regulations [55]. As well, workloads, shift schedules, and shift durations are often unpredictable [55, 79, 84]. Not only may there be little decision-making latitude but decisions are often required in extraordinarily narrow timeframes and during acute stress conditions [37, 63, 85, 86]

Contrasting low job control is job autonomy, which is defined as "the degree to which the job provides substantial freedom, independence, and discretion to the employee in scheduling the work and in determining the procedures to be used in carrying it out" [87, p. 162]. Job autonomy has been found to play an important role in buffering against undesirable outcomes in a variety of occupations [83, 88, 89]. For example, one study found that firefighters who reported greater autonomy in their work schedule and time off reported lower job stress, work-leisure conflict, and work-family conflict [83]. They also perceived greater job support than those with less perceived autonomy. Although not a study of firefighters, Shirom et al. [88] concluded that perceived autonomy successfully buffers against the effects of burnout. In a sample of manufacturing employees, Parker et al. [89] found that job autonomy was positively related to employee ratings of supportive supervision, job security and organizational commitment. Additionally, Lambert et al. [89] studied firefighters' coping self-efficacy (FCSE) with a newly developed scale. The purpose of the study was to identify how different factors influence a worker's perceived competence in managing job-related distress and traumatic events. They concluded that firefighters who reported higher competence on managing distress at work (higher FFSCE) experienced greater levels of perceived autonomy and lower job distress.

Given the hierarchical culture of firefighting [52] and the unpredictable aspects of the work, finding opportunities where firefighters can increase job autonomy will likely result in favorable health outcomes. While the above is not an exhaustive review of the literature, the view that job autonomy mediates against negative outcomes is both well established and far-reaching across multiple domains in occupational research.

## *Environment and Equipment*

Firefighters are exposed to several treacherous environments that include explosions, smoke, dust, toxic chemicals, darkness, heat, confined spaces, and heights that result from natural disasters (e.g., earthquakes, floods, storms) or man-made disasters (e.g., arson, motor vehicle accidents, industrial accidents). These environmental risks and their physical effects are described in considerable detail elsewhere in this book and will not be repeated here. Suffice it to say in this section that firefighters' knowledge of these risks is anxiogenic; provoking the fight-or-flight response and the general adaptation syndrome described later in this chapter. These responses can be amplified by poorly maintained or malfunctioning equipment.

On this particular issue, some authors have mentioned that equipment design is often androcentric and thus maladapted to female height, weight and strength [50].

## *Organizational Culture*

Firefighter culture, as mentioned earlier, is the context for understanding psychosocial hazards. However, culture may itself represent a psychosocial hazard for members of this occupational group.

Healthy organizations and workplaces are primarily determined by the quality of the interactions that occur within them. When these are positive, group cohesion is more easily established, which then contributes to psychological wellbeing. Supervisors also play a key role in worker wellbeing. Sonnentag and Grant [66] emphasized that supervisory feedback has an important effect on a firefighter's perceived competence. If a rescue was unsuccessful, a supervisor's feedback is all the more important since they may need to communicate that the team "did everything possible". Varvel et al. [91] examined perceived social support and stress. They found that social integration and support from supervisors had the highest positive effect on reducing worker stress.

Organizational leadership and aspects of supervision can be cogent predictors of job satisfaction [92–95]. As supervisor-subordinate relationships are embedded in the social structure and hierarchical nature of firefighting [52], it is not unreasonable to infer that interactions across ranks may contribute to occupational adjustment. Riggio and Cole [94] highlight the importance of superiors modeling the behavior expected from subordinates, especially in a hierarchical and trust-based job such as firefighting. Thus, organizational leaders have an instrumental role to play in terms of employee job satisfaction, job performance, positive interpersonal relationships and also effective service delivery. Some aspects of leadership and supervisory support mitigate the influence of psychosocial hazards in firefighting. Riggio and Cole's [94] study also emphasizes the fact that trust is more than simply cooperation [96]. Trust is a psychological construct rooted in actions [96].

Thurnell-Read and Parker [97] identified commonly shared masculine values such as rationality, physical strength, technical competence, risk-taking, and responsibility. A possible offshoot of this hero-centered atmosphere is a reluctance to express feelings, and exposing emotional vulnerabilities. By limiting communication of this nature, opportunities to create or expand social support networks are compromised. Other negative effects are possible. Firefighters have higher rates of exposure to traumatic stressors [34] and higher rates of PTSD [32–34]. This paradox of greater emotional trauma and low communication helps explain the emotional avoidance or suppression in which firefighters engage [54] and sadly, their reluctance to seek psychological help when such is required [31, 98]. Reinforcing the importance of this issue is work by Gohm et al. [99] who investigated the emotional and cognitive functioning of 59 firefighters during in-vivo, emergency training situations. Personality features were also collected a priori.

Firefighters who were most aware of, or sensitive to, their emotional states, reported fewer difficulties with thinking clearly during high-pressure situations. As such, it appears that knowledge about feelings (e.g., identifying, labeling) may help firefighters avoid being overwhelmed by these feelings. Indeed, Gohm et al. [99] found fewer indications of "blanking out", forgetting, or overlooking incoming information among those with greater emotional awareness.

### *Interpersonal Relationships Within the Fire Service*

Similar to members of organizations such as the military and police, firefighters' health and wellbeing often depend on their co-workers. As such, significant trust, teamwork, and cohesion are required for the successful performance of their duties. In their absence, not only is physical health and safety jeopardized but the risk of depression and distress-related symptoms also increases [82, 86]. Thus, support from colleagues is essential to perform and survive in this occupation [83].

Savia [100] described a phenomenon referred to as the "loss of belongingness". This occurs when a firefighter leaves a cohesive group (e.g., as a result of illness, injury, retirement) and experiences a loss of shared identity. This causes loneliness and exposes the worker to mental health problems such as depression and substance abuse.

In a cross discipline review, Rousseau et al. ([96] p. 395) defined trust as "a psychological state comprising the intention to accept vulnerability based upon positive expectations of the intentions or behavior of another". While the benefits of trust may seem evident, researchers have indicated that when employees trust their colleagues, they are better positioned to "get the job done" than when employees do not [101]. Specifically, when trust is established, workers can focus their attention on task demands, whereas when trust is absent, workers focus their efforts on monitoring and on self-protective behaviors [56].

Researchers indicate that two conditions must be present for trust to arise: risk and interdependence. Risk, which "is the perceived probability of loss", and interdependence, defined as a context where parties' interests cannot be reached "without reliance upon one another", create the opportunity for trust to develop [96].

A study that investigated trust in a firefighter population identified that trust varied according to the task [56]. "Typical" tasks were defined as the day-to-day activities that were considered predictable and less dangerous whereas "high reliability" tasks were characterized by activities considered situationally unpredictable and potentially dangerous. Interestingly, ability did not predict trust in either typical or high-reliability contexts. Characteristics that successfully predicted trust for typical tasks were integrity, benevolence and identification, whereas integrity was the only predictor for trust in a high reliability context. In addition to understanding what predicts trust for firefighters, research also indicates that higher levels of typical and high-reliability trust predict against negative outcomes [56]. For instance, firefighters

who reported greater trust in their co-workers in high-reliability contexts reported fewer physical symptoms such as trouble sleeping, headaches, back pain, loss of appetite, and heartburn. In turn, higher levels of trust in typical contexts predicted lower levels of withdrawal. Firefighters with higher levels of withdrawal reported engaging in behaviors such as taking longer breaks, leaving work unnecessarily and using work time for personal matters [56]. Given that trust can improve work outcomes [101] and mediate against undesirable consequences [56], building trust within firefighter organizations may be an effective tool in fostering health and wellbeing.

An additional factor to interpersonal relations is connected to emotional labor, which results from suppressing one's true emotions in order to portray more "desirable" emotions in the workplace (Hochschild 1983 cited by [54, 102]). In doing so, a work environment becomes low in authenticity and perceived psychological safety; ingredients that are required for trust, acceptance, respect, comfort and confidence [103]. Tuckey and Hayward [54] identified that firefighters and other emergency service workers regulate their emotions in a similar manner during rescues. Rather than displaying their true emotions when they interact with victims, they most often portray self-confidence, trustworthiness, calmness, and interest. Problematic is that to suppress or modify their authentic feelings, firefighters may need to expend substantial energy [54], which in turn increases the likelihood of suffering burnout [102].

Besides the type of emotional inhibition required during emergencies, researchers have also studied chronic workplace stressors that firefighters may face such as autocratic management and inflexible bureaucracies [71]. These findings resonate with those offered by Natividade [104] who identified organizational hierarchies and the unacceptability of "making mistakes" as notable stressors.

### *Hierarchy and Role*

Given the paramilitary-like and hierarchical structure, there are few opportunities for role conflict or role ambiguity. Attendance at critical incidents is typically well-orchestrated efforts with clearly assigned duties among team members. However, there can exist intra-organizational strain between professional and volunteer firefighters wherein the former group expresses a greater sense of competence and more extensive training [105, 106]. For their part, volunteer firefighters opine they sacrifice more of themselves and are therefore more "heroic". They also believe they deserve more support and approval from the general public [107].

Career options may be somewhat limited for firefighters beyond promotion within the ranks [108] and perhaps lateral transfers within the organization. Firefighters report being insufficiently remunerated [109] and by most standards earn a relatively modest salary. Approximately one-third of firefighters in the United States hold a second job [109], which certainly would affect family life.

### *Work-Family Balance*

One of the expected psychosocial hazards of firefighting is a disruption to family life [83, 110], often as a result of shiftwork [111]. Firefighters may have little input on what shifts they work and might also be expected to extend their shifts as the needs of a particular emergency dictate. It follows that a recurring sentiment expressed by firefighters' partners is loneliness [111], which is made worse in families where the partner also holds a busy career. While shiftwork brings several challenges (e.g., missing special occasions) research participants have not overlooked its benefits such as having a partner at home with the children on some weekdays [111]. On the other hand, research participants have expressed that their partners often do not share their work-related stressors with them (e.g., critical incidents, injuries) choosing instead to repress or perhaps share them only with other firefighters [111]. Although about fifty percent of firefighters will turn to their family for support [112], their partners have reported wanting to "help more" [111]. Over time, respondents state they learn how to read their partners through their non-verbal communication such that they become increasingly aware of when they should engage in a supportive dialogue or simply "give them space" [111]. Still, some researchers have indicated that at times, partners feel peripheral to "the brotherhood", with all its shared experiences, language, and brand of humor. These dynamics might be expected to strain relationships to the point of dissolution. However, there is no clear evidence that firefighters divorce at higher rates than the general population ([110], Talbert 1996 cited by Marcucci 2001 [110]).

For their part, partners tend to rely on family systems and other firefighters' partners/families to help with stressors including those related to raising children or loneliness [110, 113]. Collectively, partners report being proud of their mate's occupation and ascribe positive values to firefighting. In some research, they have also expressed confidence in their partner's job skills and their concerns about workplace accidents appear to decrease over time [111]. On the other hand, partners worry increasingly over time about the long-term consequences of firefighting such as exposure to carcinogens [111]. These authors also found partners to be proud and supportive. The partners made note of the special demands of the job such as shiftwork, traumatic hazards that are transferred home, and the unique social atmosphere that exists among firefighters. There has also been mention in the literature of "ambiguous loss", which occurs when there is uncertainty about the extent to which a person is a participating member of the family [114]. For instance, there may be a significant difference between a firefighter's physical and psychological presence within the family unit [111]. Posttraumatic stress disorder and other mental health issues can certainly increase the psychological distance that is felt between a firefighter and his or her family members.

Despite the demands and risks of firefighting, Landen [82, 86] concluded that compared to other high-risk occupations (e.g., policemen, paramedics), firefighters report greater job satisfaction. This conclusion is consistent with findings from

Pendleton, et al. [115] who noted greater life satisfaction and more positive mood states (e.g., fewer indicators of anxiety and depression) among firefighters than workers in other high-risk occupations.

## Risks from Exposure to Psychosocial Hazards

Mental disorders and dysfunctional behavioral adaptations affect individuals regardless of their geography, culture or gender [116]. Firefighters are not immune, despite our assumptions about their strong resilience.

The extensive but by no means exhaustive list of psychosocial hazards described in the previous section will now be linked to a variety of outcomes, including stress, mental illness, and physical injuries. Perceptions, especially, have a strong influence on psychosocial hazards' degree of harm.

### *Stress*

During the early twentieth century, Walter Cannon [117] offered the first attempts at defining stress. Cannon [117] viewed stress as a 'fight-or-flight' response such that when a stressor (anything that potentially causes a stress reaction) confronts an organism, the response will either be to flee or to directly oppose the stressor. Hans Selye ([118] p. 137) defined stress as "the nonspecific response of the body to any demand made upon it". He then described a predictable pattern of responses when an organism is subjected to more chronic stressors, which he named the General Adaptation Syndrome (GAS) [119]. The GAS has three phases: Alarm Reaction, Resistance and Exhaustion. During the Alarm Reaction, homeostasis is disrupted, the endocrine glands (e.g., adrenals) are activated and energy is made more readily available. The Resistance phase results from continued exposure to a stressor. The characteristic signs of the Alarm Reaction disappear if adaptation is sufficient. Exhaustion occurs when the stressor is not removed and energy continues to be depleted. In this stage, signs from the Alarm Reaction reappear irreversibly and the organism dies.

Selye's [119] work would spawn a substantial body of knowledge including the work of Simeons [120] who argued that our species had not adapted effectively to modern stressors. In short, events that should not produce 'fight-or-flight' responses (e.g. a job interview, a speech) may actually cause harmful physiological changes. Subsequent researchers elucidated on the stress-illness connection: Wolf and Wolff [121] (digestion), Engel [122] (ulcerative colitis), and Friedman et al. [123] (coronary heart disease). Some have conceptualized stress differently by focusing on how an individual actually perceives an event. Kobasa et al. [124] argued that stressors that are seen as 'challenges' would be less damaging than if they are seen as 'threats'.

Because the personality features of the typical firefighter tend to be sensation seeking, self-assured, and extraverted as well as the subculture that supports heroism,

chivalry and risk-taking, stressors might be more often perceived as challenges than threats, compared to members of the general population. Alternatively, it might be that people who are prone to anxiety disorders or who are less resilient to stress are disinclined to enter firefighting [4]. If this is the case, it might help explain the lower prevalence rates for anxiety disorders among firefighters.

In situations where firefighters cope less successfully with distress and emotional self-regulation, they would be susceptible to cardiac events [30, 72], alcohol problems [4, 30, 36, 79, 100, 112, 125–128] and a greater sensitivity to anxiety [36, 126].

## *Mental Health*

A common reaction to stress is the experience of low self-esteem, irritability, worry, and excessive sadness. Typically, these are acute in nature and tend to self-resolve. They may be referred to as "mental health problems". However, mental illness entails both distress and impaired functioning.

According to the Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association [129], 5th ed. (DSM-5), a "mental disorder is a syndrome characterized by clinically significant disturbance in an individual's cognition, emotion regulation, or behavior that reflects a dysfunction in the psychological, biological, or developmental processes underlying mental functioning. Mental disorders are usually associated with significant distress or disability in social, occupational, or other important activities. An expectable or culturally approved response to a common stressor or loss, such as the death of a loved one, is not a mental disorder. Socially deviant behavior (e.g., political, religious, or sexual) and conflicts that are primarily between the individual and society are not mental disorders unless the deviance or conflict results from a dysfunction in the individual" ([126] p. 20).

Once identified as suffering with a mental disorder, workers may find themselves socially excluded and subjected to mistrust or rejection [98, 130]. This can have a devastating effect on an individual's quality of life and sense of self; particularly given the gender and subculture in question [131]. Consequently, firefighters may choose to conceal their distress and avoid seeking required help [31, 98]. Of course, this only increases suffering, affects work performance/productivity and often leads to presenteeism, absenteeism, and conflict with other employees.

## *Posttraumatic Stress Disorder*

In reviewing the mental health literature on firefighters, PTSD receives the most attention (see Table 11.2). As mentioned earlier, firefighters are exposed to traumatic occurrences such as death or threatened death, serious injury, violence,

disasters, and accidents. The exposure can be directly experienced or witnessed directly as it occurs to others [132, 133]. They might also learn that a violent/accidental event happened to a co-worker. New in the most recent edition of the Diagnostic and Statistical Manual of Mental Disorders, DSM-5, is a specific mention of first responders, given they may experience "repeated or extreme exposure to aversive details of the traumatic event"([129] p. 271).

Fullerton et al. [134], Lee et al. [135], and Meyer et al. [4] emphasize that being repeatedly exposed to traumatic events, especially natural disasters [20] is a major risk factor for developing PTSD among firefighters and rescue workers. Despite repeated exposures, it is important to note that most firefighters will not suffer with PTSD [28, 90, 136]. The reasons for this are not entirely clear; however, according to the general adaptation syndrome (GAS), the first response (i.e., the Alarm Phase) can facilitate a positive mental health outcome in the form of increased energy and focus. As Cannon [117] would have argued, it prepares for a successful "fight or flight". Among some individuals, a negative stress reaction can persist for up to 3 days and this is called an Acute Stress Reaction. Should the stress reaction persist beyond 3 days, a diagnosis of Acute Stress Disorder (ASD) may be conferred.

The effects of being exposed to traumatic events were measured in Fullerton et al.'s [134] study. Workers experiencing their first disaster suffered with ASD at a rate of 2.4 %. Approximately one-quarter (25.6 %) of those who had responded to multiple previous disasters suffered with ASD. Among those with multiple previous exposures, 16.7 % were diagnosed with PTSD 13 months after the disaster. In contrast, only 1.9 % of those not previously exposed to a disaster situation were diagnosed with PTSD. Risk factors for ASD included being young and single but PTSD had no such correlates. However, multiple prior exposures did predict the onset of PTSD and ASD was a risk factor. The broader literature on PTSD identifies a number of other risk factors and these have been categorized as pretraumatic, peritraumatic, and posttraumatic.

Pretraumatic factors are independent from the trauma and include childhood emotional problems by age 6 years, lower education and socio-economic status, lower intelligence, minority/ethnic status and familial psychiatric history, personality factors, gender, age, and genetic predispositions. Peritraumatic factors relate to the severity of the trauma and stress-regulating factors (e.g. biological changes). Posttraumatic factors include neurological reaction to stress, poor coping skills, adverse life events, loss, and insufficient social support [129, 137]

Among firefighters, the literature identifies history of mental illness [29, 30, 138], years of service/age [29, 30, 34, 35, 108, 139], rank [34, 139], financial problems [30], occupational stress [4, 34, 135], work-family conflict [30], personality (hostility [138]), low self-efficacy [108, 138, 139], helplessness [29, 31], self-blame [4], negative self-appraisal [32]; and poor physical health [30] as pretraumatic factors. In term of peritraumatic factors, the literature points to the intensity, frequency and duration of trauma exposure [4, 28, 34, 35, 135, 140]. Specifically, with increased exposure come greater biological vulnerabilities linked to such issues as hyperarousal [28]. Posttraumatic risk factors include poor coping skills [4, 139], avoidance [31, 105, 140–142], rumination [31], and insufficient or low social support [4, 34, 108, 111, 141].

These risk factor categories offer perspective on how they influence the emergence and possible entrenchment of symptoms. For instance, pretraumatic risk factors relate to how a firefighter perceives and is alarmed by distressing situations. Peritraumatic risk factors can come into play when negative outcomes occur (e.g., death, serious injury) despite the firefighter's effort. In these cases, it is not so much the objective experience that is of greatest influence but how the firefighter perceived their role during the incident [4, 32, 66, 108]. In the absence of supportive feedback, self-blame may result. The posttraumatic factors might entrench trauma-related symptoms, as there may be insufficient social support [4, 34, 108, 141] to counter anxious ruminations [31] or avoidant behaviors [31, 105].

As a result, some firefighters will experience the development of specific and often predictable symptoms. The symptoms of PTSD are often manifest in the 3 months after the event but their onset can be delayed several months or years in some cases. Symptoms must be present for at least one month for a diagnosis of PTSD. Characteristic among these are recurrent and involuntary recollections of the event such as distressing dreams and/or altered states where the individual relives the experience as though it were occurring at that moment (i.e., "flashbacks"). Significant psychological distress and/or physiological responses are precipitated by "triggering events" that resemble or symbolize an aspect of the traumatic event [129]. To meet criteria for posttraumatic stress disorder, the individual would make efforts to avoid things that are reminders of the event. Interestingly, despite the salience of the event, there may be a failure to recall important aspects of the event. Other cognitive effects of PTSD include increased negative expectations (e.g., "the world is a more dangerous place", "I will have poor judgment", "no one can be trusted"). Mood states are more likely to be negative and much like depression; the individual may no longer be interested in previously enjoyed activities. This may be associated with a feeling of estrangement from others and anhedonia. Anger and aggression may occur more frequently as could reckless behaviors such as dangerous driving, alcohol/drug abuse, and self-injurious behaviors. Perhaps paradoxically given higher-risk behaviors, those with PTSD are often hypervigilant (e.g., an excessive alertness to potential threats) and hyperresponsive (e.g., feeling "jumpy" and easily startled). Sleep and concentration are typically affected [129, 137].

Complete recovery can be expected within 3 months in one-half of cases. However, some individuals remain symptomatic for several years. Coping with PTSD is not easy but is helped made possible by perceived competence and social support. A key element is affective emotion regulation, which allows adaptive coping strategies. The restoration of trust [143], especially in their own ability [108], and competence [144] can be accomplished via supportive and objective exchanges with colleagues and supervisors [144, 145]. This permits an individual to draw meaning from the experience despite its traumatic nature. It also assists with establishing increased trust and cohesion within the group [91].

PTSD is associated with a higher risk of suicide. Since 2010, approximately 30 suicides per year occur among American firefighters. About 2 % of career firefighters and 4 % of volunteer firefighters admit to having attempted suicide and a significant proportion have experienced suicidal ideations (25.1 % of career firefighters

and 18.4 % of volunteer firefighters). Gender differences are noted in terms of suicidal ideations where 23.8 % of males admitting to such versus 38.5 % of females [146].

Cognitive-behavioral therapy is usually the treatment of choice for this condition. Here, the focus is three-fold: teaching coping/relaxation skills, imaginal and/or in-vivo exposure to unpleasant triggers/memories, and addressing negative thinking styles. Pharmacotherapy is often a useful adjunct to psychotherapy. Co-morbidity is common and sufferers are 80 % more likely than non-sufferers to meet criteria for another mental disorder such as major depressive disorder [129].

## *Mood and Anxiety Disorders*

There is general consensus that mood and anxiety disorders are caused by several factors. Some are biological in origin while others are psychological or sociocultural in nature [129]. In light of what has been discussed earlier in this chapter, the influence of occupational stressors should not be overlooked. Indeed, posttraumatic stress disorder and mood disorders are often comorbid. In this regard, major depressive disorder deserves particular attention.

### Major Depressive Disorder

The distinguishing feature of major depressive disorder is low mood or a loss of interest in previously enjoyed activities. Often, there is an incapacity to experience pleasure. In some individuals, the mood may be as irritable as it is sad. However, to qualify for a diagnosis, an individual would minimally also experience five of the following symptoms: changes in appetite or weight (increase or decrease), psychomotor activity and sleep (insufficient or excessive); difficulties with concentration, thinking or decision-making; recurrent thoughts of death or suicidal ideations/plans/attempts; decreased energy; decreased interest and pleasure; depressed mood or hopelessness, emptiness; and feelings of worthlessness or excessive guilt [129].

The one-year prevalence rate for major depressive disorder (see Table 11.3) in the United States is about 8.7 % and in Canada is 8.2 % [147]. Females are 1.5–3 times more likely to suffer depression from adolescence onward but these sex differences decline later in life. Again in the United States, the incidence of major depressive disorder peaks during the 20s though later life onset is not unusual. There are multiple risk factors for this illness: temperamental, environmental, and genetic/physiological. Neuroticism (described earlier as the tendency for negative affect) is a strong correlate of depression and individuals high in this trait are at greater risk during a stressful life event, which is in and of itself a predictor of the illness. Heritability accounts for about 40 % of major depressive disorder but it is important not to discount the contribution of an individual's personal circumstances. Divorce/

separation, family problems, loss, sleep deprivation, and chronic medical conditions (e.g., pain, diabetes, and cardiovascular disease) are notable risk factors [129].

In a study of firefighters by Regehr et al. [108], a significant positive linear relationship was found between years of service with levels of depression and traumatic stress. Further relationships were found between depression, low social support and low self-efficacy. In a small sample of firefighters, Monteiro et al. [128] found a significant positive correlation between depression and age. For their part, Carey et al. [36] found significant correlations between depression, hazardous drinking and disturbed sleep patterns. Chung and Park [148] found that injuries or near-miss accidents among firefighters led to higher rates of depressive symptoms (Table 11.4).

Burnout is a related construct to depression, which has been defined in a variety of ways but in most cases include the concepts of emotional and/or physical exhaustion, low levels of personal accomplishment, depersonalization or cynicism [150]. As can be seen, burnout has many overlapping features to depression (e.g., hopelessness, emotional emptiness, fatigue, somatic complaints) but is not a diagnosis. Rather, it is a potential outcome of workplace psychosocial hazards. Burnout has been positively related to other occupational stressors including emotional demands, traumatic stress symptoms, involvement in critical incidents and psychological distress. Importantly, firefighters who reported having more emotional resources and greater camaraderie had lower burnout scores [54].

**Table 11.3** Prevalence rates for major depressive disorder in the general population

| Estimated period prevalence rate | Estimated Lifetime prevalence | Country (N) | Study references |
|---|---|---|---|
| 6.7 % | | USA (9282) | Kessler et al. [16] |
| 8.2 % | | Canada (3505) | Vasiliadis et al. [147] |
| 8.7 % | | USA (5183) | Vasiliadis et al. [147] |
| | 16.6 % | USA (9282) | Kessler et al. [16] |

Definitions as per Table 11.1

**Table 11.4** Prevalence rates for major depressive disorder in the firefighter population

| Estimated period prevalence rate | Country (n) | Study references |
|---|---|---|
| 3.5 % | USA (142) | Meyer et al [4] |
| 7.4 % | Brazil (27) | Monteiro et al. [128] |
| 9.1 % | Korea (186) | Chung and Park [148] |
| 9.4 % | USA (106) | Liao [73] |
| 10.0 % | Brazil (303) | De Barros et al. [149] |
| 10.5 % | Taiwan (410) | Chen et al. [30] |
| 11.0 % | USA (112) | Carey et al. [36] |
| 12.5 % | USA (40) | Liao [73] |

Definitions as per Table 11.1

### Anxiety Disorders

Anxiety symptoms and disorders are less prevalent among firefighters [4, 115] than in the general population. Specifically, the estimated period prevalence rate for anxiety disorders is between 3.7 % [128] and 9.0 % [149] among firefighters. In contrast, anxiety disorders in the general population are estimated at 18.1 % [16]. However, we have previously discussed that firefighters are prone to emotional avoidance and therefore symptoms of this nature may be interpreted by these workers as strictly physiological. Indeed, there is considerable overlap between anxiety and cardiac-related symptoms (e. g., palpitations, pounding heart, sweating, shortness of breath, chest pain, etc.). As with many other occupations, firefighting would be more accepting of physical ailments than psychological distress such that symptoms would more likely be attributed to the former. We would propose that anxiety, though less frequently discussed and diagnosed in this population, may in fact be an important driver of higher cardiovascular morbidity rates [70, 72, 73] among firefighters.

## *Substance Use Disorders*

Substance use disorders are assigned when an individual continues to use a substance despite the significant problems it causes. The sufferer will experience impaired control regarding its use and may have been repeatedly unsuccessful in curbing the behavior. Also, the sufferer may spend excessive time obtaining, using and/or recovering from the effects of the substance such that daily activities increasingly revolve around the substance. Cravings, or intense desires to use, are common as are social impairments and an inability to fulfill obligations (e.g., at work or home). Additional criteria used in diagnosing substance use disorders are tolerance (i.e., needing increased dosages to produce similar effects) and withdrawal (i.e., a syndrome that results from decreased use). The number of symptoms that are experienced by an individual defines the severity of these disorders and range from mild to severe [129]. Table 11.5 offers prevalence rates for alcohol abuse in the general population.

The principal substance of concern in the fire service is alcohol. Hazardous drinking is defined as risky alcohol consumption that does not meet criteria for an alcohol use disorder [153]. The standards or limits for hazardous drinking are based on epidemiological research relating to health outcomes. In the USA, the recommended limits for hazardous drinking is 14 drinks per week for males and 7 drinks for females [153], whereby one drink is equivalent to 12 g of pure alcohol [154]. Carey et al. [36] also defined binge drinking as four or more drinks per day for men or three or more drinks per day for women.

Carey et al. [36] studied 112 firefighters and found that 58 % engaged in binge drinking behaviors and 14 % engaged in hazardous drinking behaviors (Table 11. 6). In this study, the authors pointed out that drinking negatively affects performance, reduces reaction time and disturbs natural circadian rhythms. Not only are these

**Table 11.5** Prevalence rates for alcohol abuse in the general population

| Estimated period prevalence rate | Estimated lifetime prevalence | Country (n) | Study references |
|---|---|---|---|
| 3.1 % | | USA (9282) | Kessler et al. [16] |
| | 13.2 % | USA (9282) | Kessler et al. [14] |
| 4.7 % | | USA (43093) | Grant et al. [151] |
| 4.7 % | 17.8 % | USA (43093) | Hasin et al. [152] |

Definitions as per Table 11.1

**Table 11.6** Prevalence rates for alcohol abuse in the firefighter population

| Estimated period prevalence rate | Estimated Lifetime prevalence | Country (n) | Study references |
|---|---|---|---|
| 3.7 % | | Brazil (27) | Monteiro et al. [128] |
| 10.6 % | 25.4 % | USA (142) | Meyer et al. [4] |
| 31.0 % | | Brazil (303) | De Barros et al. [149] |
| 41.8 % | | Australia (469) | McFarlane [127] |
| 53.7 % | | Taiwan (410) | Chen et al. [30] |

Definitions as per Table 11.1

findings relevant from a physical perspective, but alcohol is also known as a psychological depressant that increases the risk of suicide. Indeed, Savia [100] found that 15 % of firefighters who attempted suicide used alcohol regularly. For their part, Meyer et al. [4] reported in their sample of 142 firefighters that the rate of alcohol abuse in the previous year was 10.6 %. They also reported that problematic alcohol use in the previous year was 22.5 % of the sample. The estimated lifetime rate for alcohol abuse was 25.4 % and over the course of a lifetime, problematic alcohol use was reported by 40.1 % of respondents. Perceived social support, occupational stress, and a self-blaming coping style were predictors of alcohol problems in this sample.

Monteiro et al. [128] studied anxiety, depression, alcohol use, PTSD, and work environment factors in a sample of 27 firefighters in Southern Brazil. They found correlations between alcohol consumption and age ($r = .40$; $p = 0.05$), alcohol abuse and length of service ($r = 0.41$; $p = 0.05$), and between depression and age ($r = .39$; $p = 0.05$). Six firefighters met criteria for hazardous drinking and one person likely suffered with alcohol dependence. In a sample of 584 firefighters, Oh et al. [155] found that PTSD was more strongly associated with alcohol consumption than were other issues such as depression, anxiety, and other work stressors. Their findings parallel those of Bacharach et al. [125], who found that stressful and traumatic events were strongly associated with problematic drinking, which suggests a maladaptive coping strategy [86, 112, 127].

Monks [156] identified that firefighters are more likely to drink after work if they "beat a fire" or "needed to cope with loss". Drinking was perceived as a normal part of their subculture or a "social glue". As can be seen in the following tables, rates of problematic drinking are higher among firefighters.

## Sleep Disorders

Responding to alarm calls evokes the stress response and disrupts sleep patterns [109, 157]. It follows that firefighters report disrupted sleep, sleep deprivation, and poor sleep quality as frequent consequences of their work [36, 109]. Gaskill and Ruby [158] indicated that firefighters regularly sleep less than 7 h/night.

Sleep disorders are often co-morbid with PTSD, mood and anxiety problems as well as cognitive changes. Indeed, persistent sleep disturbances such as insomnia increase the likelihood of mental illnesses and substance abuse disorders [129].

### *Insomnia*

This disorder is defined by the dissatisfaction an individual experiences relative to sleep quantity or sleep quality. There would be concurrent problems with sleep initiation and/or, most commonly, sleep maintenance. The term 'non-restorative sleep' means that an individual does feel well rested after waking even though the duration of their sleep is sufficient. Insomnia disorder is typically accompanied by subjective impairments (e.g., poor work quality due to fatigue, poor focus/concentration) as well as sleep difficulties. Some behavioral and cognitive factors are known to exacerbate this disorder. For example, a striving or preoccupation with sleep might paradoxically make sleep worse. Poor sleep can also lead to problematic sleep habits such as frequent napping, excessive time in bed and the use of substances to initiate and/or maintain sleep (e.g., alcohol, over-the-counter medicines). Sufferers are more likely to report psychological symptoms and impaired cognitive functioning. Insomnia disorder is prevalent in about 6–10 % of the general population [129] and likely higher among firefighters based on what is known of the effects of shift work and alarm disrupted sleep.

### *Circadian Rhythm Sleep-Wake Disorder*

Circadian rhythm sleep-wake disorders are characterized by persistent or recurrent patterns of sleep disruptions that result from changes of the circadian system [129]. There are a number of variations of this disorder but in the context of firefighting, 'shift work type' is likely the most applicable. Here, the diagnosis is based on an individual's history of working outside typical hours on a regularly scheduled basis [129]. The worker would experience insomnia (e.g. at bedtime) and/or excessive sleepiness (e.g., during work hours). Usually, these symptoms resolve when the sufferer returns to a day-work schedule, which is often unlikely in the life of a firefighter.

### *Injuries and Chronic Pain After Injury*

Shiftwork is one of the major predictors of injuries [84, 159] and working more than 70-h has been shown to increase the likelihood of injury fourfold [79]. Glazner [84] reported that only 42 % of fire alarms occurred between 18:00-24:00 but 68 % of the injuries occurred at that time. The author concluded that disruptions in eating patterns and sleep-wake cycles increased the risk of work-related injuries. Regarding the connection between shiftwork and injuries, age appears to be an important mediator. Cloutier and Champoux [160] found that while older workers were less frequently injured, their injuries tended to be more severe and associated with longer workplace absences. These findings were similar to those found by Folkard [159].

A possible consequence of injury is chronic pain. In a study by Beaton et al. [109], 95 % of a mixed sample of American paramedics (n = 253) and firefighters (n = 1730) reported at least one work-related pain experience. Of these, about 48.2 % reported pain that was severe enough to affect their work. Higher pain ratings were recorded in older and more experienced workers but no gender differences were noted. In this same study, the association between pain and occupational stressors was also explored. The correlates included past critical incidents, sleep disturbance, labor-management conflict, coworker conflict and wage/benefit concerns. Non-work variables were family problems and poor health habits. Psychological factors such as depression, anxiety and anger were strongly associated with physical pain and were important mediators between work and non-work related stressors. The strong relationship that exists between chronic pain and major depressive disorder is such that physical injuries ought to be addressed as quickly and as effectively as possible. Once workers experience chronic pain, psychological services can be useful for non-pharmacological symptom management. Finally, physical and mental capacities may decline with age and recovery from injuries may be lengthier.

## Intervention

As it is well established that firefighters have higher levels of exposure to traumatic stress compared to the general population [20, 34], offering access to "psychological first aid" to affected employees seems both intuitively pleasing and ethically responsible. Group debriefing following exposure to potentially traumatic events, has been used by military [161], law enforcement [162] and emergency service personnel [163, 164].

A specific intervention is critical incident stress debriefing (CISD), which is also referred to as the Mitchell model [165]. CISD is a debriefing tool that is part of a larger critical incident stress management program (CISM), that can include

pre-incident preparation, individual crisis intervention, family support services, follow-up services, and when necessary, referrals to professional care [165, 166].

CISD is led by two trained debriefing facilitators and typically occurs between 2 and 7 days following exposure to a potentially traumatic event [165]. CISD is a seven-stage model developed for use with small, homogenous groups and the delivery of the debriefing protocol can range from 1 to 3 h. There are seven stages of CISD [166], including (1) the introduction phase, (2) facts phase, (3) thoughts phase, (4) reactions phase, (5) symptoms phase, (6) teaching phase, and (7) re-entry phase. During the introduction phase, the group facilitators will orient group members by describing the CISD process, outlining the guidelines, discussing issues such as confidentiality and voluntary participation, and motivate members to actively engage in the process. During the facts phase, each member is given the opportunity to provide an overview of the facts of the critical event. Members then discuss their thoughts (thoughts phase) and reactions (reaction phase) related to the incident, which is followed by an exploration of how the traumatic experience has affected their life. This is also the time when the emergence of distressing symptoms (symptoms phase) is discussed. During the teaching phase, facilitators will normalize participant reactions and provide psycho-education on symptoms and stress management strategies. The re-entry phase is the final stage that involves answering questions, making final statements, summarizing the discussion and providing members with information handouts.

In terms of the efficacy of CISD, research has produced mixed results. When trying to determine the effectiveness of any treatment intervention, the "gold standard" in research is to conduct a study with a randomized controlled trial (RCT) design. In three published RCT studies [161, 164, 167] and one meta-analysis [168], the investigators found that CISD did not differ from other conditions (i.e., no-treatment and stress management education) used for reducing symptoms of post-traumatic stress. Similarly, Lewis [169] found inconclusive results on the effectiveness of debriefing procedures following a traumatic event. It is important to note; however, that the findings from these three RCT studies indicated that CISD was not associated with harmful outcomes. In contrast, the meta-analysis by van Emmerik et al. [168] found that CISD may actually have detrimental effects. Although there is a lack of RTC data supporting the usefulness of CISD in reducing symptoms associated with trauma reactions, results suggest some beneficial outcomes. Specifically, individuals who received CISD appear to have lower alcohol consumption rates and higher quality of life ratings [164]. A qualitative study investigating the utility of CISM with both UK and Canadian firefighters found that although firefighters had mixed opinions of CISM programs, they were perceived as being culturally appropriate vehicles to promote social support, personal coping, and meaning-making following a traumatic incident [170]. Mandatory attendance was not perceived as helpful [170, 171]. Emerging consistently from comprehensive reviews of CISD is the need for more research [162, 165, 169, 172].

Evelry et al. [165] highlighted that research on the effectiveness of CISD is favourable as long as two conditions are met: (1) group facilitators have received

appropriate CISM training and (2) the program falls within acceptable CISM standards of practice (i.e., using homogenous groups rather than individual victims, using CISD with staff rather than primary victims). McEvoy [171] also emphasized that mandatory participation, mixed groups and reliving traumatic emotions are harmful. Blaney [170] proposed that CISD should not be viewed as a medical intervention but rather should be used as a health promotion framework. Doing so should lead to greater acceptance by firefighters and create a supportive environment.

Unlike CISD interventions that are used to buffer against the onset of PTSD symptoms and other forms of distress, psychotherapies are typically used following the onset of psychological symptoms. Of the three interventions identified by the Society of Clinical Psychology et al. [173] as having "strong research support", both prolonged exposure therapy and cognitive processing therapy fall under the umbrella of cognitive behavior therapy. Cognitive behavioral approaches to treating PTSD often include an assessment phase, socialization to treatment, anxiety management training, exposure exercises, cognitive restructuring [174], and skills training such as building social support systems, social and communication skills, and assertiveness training. Whereas prolonged exposure therapy involves gradually and systematically exposing the client to trauma related cues through imaginal and in-vivo exercises, cognitive processing therapy aims to challenge distorted thinking and beliefs related to the traumatic event [175, 176]. The final psychotherapy highlighted as having strong research support by the APA is present-centered therapy. Present-centered therapy is a problem-solving intervention and psycho-educative method that focuses on here and now issues rather than the trauma itself [177]. The role of the therapist is to guide clients to notice how the trauma has impacted their life and factoring such dynamics into the problem-solving process. Person-centered therapy is distinctly different from cognitive behavioral therapies, as it does not involve exposure tasks or cognitive restructuring [177].

Many of the psychosocial hazards identified in this chapter are inherent to the occupation and largely unavoidable (e.g., exposure to trauma). Given their predictability and the greater availability of sound research, managers and policy-makers are well positioned to support, supervise and care for firefighters. We anticipate that organizational leaders and policy makers will be increasingly tasked with promoting physical and psychological wellbeing and mitigating the effects psychosocial workplace hazards.

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# Chapter 12
# Presumption

**Alex Forrest**

A basic premise in today's firefighting is that the death or injury of a fire fighter as a result of occupational cancer, heart attack on duty, or acute respiratory disease is no different than a traumatic death or injury at the scene of a fire. It is not only the same in terms of who was responsible (and therefore where liability lies) but also in the valor and service of the fallen firefighter.

Cancer is the main issue in presumption for firefighters and the model for discussion of presumption legislation [1].This premise is accepted in today's fire service because it not one fire that kills us by cancer, it is the hundreds of fires we attend to in our firefighting careers that kill us through occupational cancer. It is the cumulative effect of exposure to the carcinogens at fires that creates an elevated risk for cancer in firefighters [2, 3] and this creates legal challenges when it comes to workers compensation and other insurance programs for firefighters.

The legal issues arise when we talk about the legal right of a firefighter to be compensated for an occupational disease such as cancer. Modern day workers compensation for fire fighters that is connected to cancer is called presumptive legislation, which in varying forms will reverse the normal legal onus in compensation claims. The first premise of this law is that the legislation recognizes the connection between firefighting and occupational diseases such as cancer and heart injury. Presumptive legislation in its most simple form reverses the burden of proof. The usual onus in legal claims is on the worker and in normal compensation claims the onus is also on the worker to prove that the injury is connected to his or her employment and to state when and where the injury occurred. However, under presumptive legislation the onus is reversed and is on the opposing organization (the employer or a workers' compensation carrier). The onus is on the organization that is challenging the worker's claim and that organization then has to show that the firefighter did not get their cancer or other condition as a result of his or her job history.

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## Presumptive Legislation

What is presumptive occupational legislation? A presumptive law links a particular occupation with a disease or condition that has been shown to be a hazard associated with that occupation. As a result of that linkage, if the individual employed in an occupation that is covered by the presumption contracts a disease or condition specified in the presumptive law, then that disease or condition is presumed to have arisen from, and is a direct result of, that person's occupation. Basically presumptive legislation allows you to gain coverage; no presumptive legislation means that you will not be covered.

In reality presumptive legislation is a legal term for switching of the burden of proof, it is a reverse onus clause, which is usually inserted in the particular jurisdiction's workers' compensation laws or statutes. It switches the burden of proof and evens the playing field so that the firefighter has the ability to show that they have been injured by their occupation. It reverses the onus onto the other side to prove that a specific firefighter's cancer is *not* an occupational disease. Prior to this legislation the onus was always on the employee to show at what particular fire he or she contracted cancer. This would be a very difficult thing to prove as it is not merely the tracking of one incident but hundreds of exposures throughout a person's firefighting career.

The legislation makes the presumption that if a firefighter who is diagnosed with a specific cancer has the qualifying length of service and meets other requirements then the cancer was contracted in the performance of duties as a firefighter.

The legislation usually outlines the specific requirements to qualify and these vary from jurisdiction to jurisdiction. In Canadian, Australian and many American jurisdictions the fire fighter must work a certain number of years, depending on the type of cancer. In the Manitoba legislation, which is replicated in legislation throughout most of Canada and Australia, a person must work 5 years as a firefighter to be able to qualify for coverage for leukemia, 10 years for brain cancer, 20 years for colon cancer and 10 years for breast cancer.

There is usually a requirement to be a specific type of firefighter, such as urban, professional, volunteer, wildland and so forth. Also, the cancer must be primary site, which means that the cancer must be the originating cancer and not a cancer that has metastasized from another area of the body. An example would be a firefighter who was covered for brain cancer but first had cancer of the colon. Since the brain cancer was not the primary or first site cancer the legislation would not be applicable to that firefighter, unless it is metastatic from a primary that is recognized.

In many jurisdictions there are restrictions on a retired fire fighter's coverage under presumptive legislation for occupational cancer, however, in the Manitoba model the injury can result from cancer that was diagnosed after the fire fighter retired (Manitoba 2002). The premise for this is that even if a firefighter is retired it does not mean that he or she will not be diagnosed with cancer as a result of his or her firefighting career. Again this goes to the deadly nature of occupational cancer,

which may surface after the person finishes his or her firefighting career. This again relates to the latency period between the exposure and the diagnosis of cancer. When the legislation applies to retired firefighters there is a formula for coverage.

In Canada we have had success in making the legislation retroactive, which allows the legislation to be backdated to a prior year. In Manitoba the legislation was initially passed in 2002 but the coverage goes back to fire fighters diagnosed with cancer after 1992. This aligned with the increase in the number of cancer studies that described a connection between fire fighting and occupational cancer. The provinces of Ontario, Saskatchewan and Alberta have also enacted retroactive presumptive legislation, although as a rule retroactivity is not part of the American or Australian presumptive legislation.

Almost all other legislation in Canada, the U.S.A. and Australia is proactive, which means it only applies to cancers diagnosed after the passing of the legislation but four Canadian provinces have some retroactivity to their legislation. This was done because of the premise that this legislation should have been passed years before it actually was and the retroactivity is what the jurisdiction did to correct the wrong.

The legislation is almost always rebuttable, which allows for some specific arguments that can make the presumptive legislation null and void. A specific one that comes to mind is that of smoking. Many jurisdictions state that if the firefighter is a smoker then the legislation does not apply to that firefighter. Sometimes it will apply only to one specific cancer as in Canada with lung cancer but in some American jurisdictions being a smoker nulls all legislation.

## History

Presumptive legislation for occupational diseases has a long history. It was intended to deal with problems that the workers' compensation system found difficult. Workers compensation itself was a response in 1883 to the terrible carnage of injuries during the Industrial Revolution of the late nineteenth Century. It worked reasonably well for obvious injuries but not for diseases. When the cause was not completely obvious, the cause and effect relationship could be argued, or the appearance of the disease was delayed, the workers' compensation system broke down. The worker could not sue for relief because workers' compensation was an "exclusive remedy", not allowing access to the courts unless it could be shown that the law itself had been misapplied. This left many workers sick, impoverished, and cut off from benefits because their injuries were internal and needed proof that the worker could rarely provide. The response by Parliaments and legislatures around the world was to define a list of recognized diseases that were characteristic of certain occupations and to mandate acceptance of claims for those diseases from persons working in those occupations.

However, we knew of the dangers of smoke and soot long before the first presumptive cancer appeared on the scene in North America. The relationship between

cancer and occupational exposure was recognized in the chimney sweeps of the early 18th century in London. Firefighters can be considered to be modern day chimney sweeps, and their risk of cancer comes from many of the same chemicals.

The issue of soot carrying chemicals having an impact on working people is not a modern idea as one of the first people to look at occupational cancer was Percival Pott, a London surgeon who lived in the 1700s and was the first to demonstrate that cancer can be influenced by the environment. Pott connected the high rates of scrotum cancer in young chimney sweeps to the soot that had direct contact with their skin together with a lack of hygiene because bathing was uncommon in those days and the soot accumulated in their regular clothes and on their skin. To understand the exposure of the chimney sweeps you have to understand the nature of a chimney sweep's job in eighteenth century England. The children were usually very young, as the smaller the child the easier it was for them to navigate London's chimneys. They wore very little if any clothes due to the dirty conditions and the cost of clothes. Pott made the connection without any of the modern scientific techniques or knowledge of carcinogenesis and that speaks to how evident the danger of cancer causing agents is in smoke and soot. We now know that soot carries with it cancer causing carcinogens including polycyclic aromatic hydrocarbons. The cancer that these poor children suffered from was squamous cell carcinoma of the testes and scrotum.

As a result of Pott's work the first form of presumptive legislation was enacted in England in 1788 when the Chimney Sweepers' Act was passed [4]. It recognized the link between chimney sweeping and cancer and said that no child could be a chimney sweep before they were 8 years old and he had to be a bound apprentice. Prior to that, many of the chimney sweeps were as young as 4 years old. The Chimney Sweepers' Act also stated that in order for a boy to become a chimney sweep he had to have the consent of his parents and suitable clothing had to be provided to the chimney sweeps so that they could not be made to work more or less naked. They also had to have suitable living quarters and cleaning conditions and be allowed to attend church every Sunday. It should also be noted that an amendment that could have funded proper licensing and monitoring of this law was defeated.

The first modern day presumption legislation for occupational disease in firefighters was enacted in California in 1982, with more recent amendments in 2010 and 2012. These are presented in Exhibit 1. The California legislation really is the gold standard for presumptive legislation within the United States. All fire fighters owe a great deal of gratitude to the International Association of Fire Fighters within California for the work that they did to get this presumptive legislation passed. The California Cancer Presumption Act of 2010, an amendment, created a disputable presumption that if a "firefighter", as defined, develops cancer, that cancer is considered an occupational injury for the purposes of the workers' compensation system. The compensation awarded for occupationally-related cancer must include full hospital, surgical, medical treatment, disability indemnity, and death benefits, as provided by workers compensation law. This presumption runs for 10 years, commencing on their last day of employment. It includes wildland firefighters and active firefighting members of a fire department that serves a United States

Department of Defense (DOD) installation or the National Aeronautics and Space Administration and who are certified by the DOD as meeting its standards for firefighters.

**Exhibit 1. Presumptive Disability Law in California**

California Labor Code §3212.1 (amended January 2012) [1]

This section applies to all of the following:

(1) Active firefighting members, whether volunteers, partly paid, or fully paid, of all of the following fire departments:
   (A) A fire department of a city, county, city and county, district, or other public or municipal corporation or political subdivision.
   (B) A fire department of the University of California and the California State University.
   (C) The Department of Forestry and Fire Protection.
   (D) A county forestry or firefighting department or unit.

(2) Active firefighting members of a fire department that serves a United States Department of Defense installation and who are certified by the Department of Defense as meeting its standards for firefighters.

(3) Active firefighting members of a fire department that serves a National Aeronautics and Space Administration installation and who adhere to training standards established in accordance with Article 4 (commencing with Section 13155) of Chapter 1 of Part 2 of Division 12 of the Health and Safety Code.

(4) Peace officers, as defined in Section 830.1, subdivision (a) of Section 830.2, and subdivisions (a) and (b) of Section 830.37, of the Penal Code, who are primarily engaged in active law enforcement activities.

(5) (A) Fire and rescue services coordinators who work for the Office of Emergency Services.
   (B) For purposes of this paragraph, "fire and rescue services coordinators" means coordinators with any of the following job classifications: coordinator, senior coordinator, or chief coordinator.
   (b) The term "injury," as used in this division, includes cancer, including leukemia, that develops or manifests itself during a period in which any member described in subdivision (a) is in the service of the department or unit, if the member demonstrates that he or she was exposed, while in the service of the department or unit, to a known carcinogen as defined by the International Agency for Research on Cancer, or as defined by the director.

(c) The compensation that is awarded for cancer shall include full hospital, surgical, medical treatment, disability indemnity, and death benefits, as provided by this division.
(d) The cancer so developing or manifesting itself in these cases shall be presumed to arise out of and in the course of the employment. This presumption is disputable and may be controverted by evidence that the primary site of the cancer has been established and that the carcinogen to which the member has demonstrated exposure is not reasonably linked to the disabling cancer. Unless so controverted, the appeals board is bound to find in accordance with the presumption. This presumption shall be extended to a member following termination of service for a period of three calendar months for each full year of the requisite service, but not to exceed 120 months in any circumstance, commencing with the last date actually worked in the specified capacity.
(e) The amendments to this section enacted during the 1999 portion of the 1999–2000 Regular Session shall be applied to claims for benefits filed or pending on or after January 1, 1997, including, but not limited to, claims for benefits filed on or after that date that have previously been denied, or that are being appealed following denial.
(f) This section shall be known, and may be cited, as the William Dallas Jones Cancer Presumption Act of 2010.

**Labor Code 3212.1.**

a. This section applies to active firefighting members, whether volunteers, partly paid, or fully paid, of all of the following fire departments:
   1. a fire department of a city, county, city and county, district, or other public or municipal corporation or political subdivision,
   2. a fire department of the University of California and the California State University,
   3. the Department of Forestry and Fire Protection, and
   4. a county forestry or firefighting department or unit. This section also applies to peace officers, as defined in Section 830.1, subdivision (a) of Section 830.2, and subdivisions (a) and (b) of Section 830.37, of the Penal Code, who are primarily engaged in active law enforcement activities.
b. The term "injury," as used in this division, includes cancer, including leukemia, that develops or manifests itself during a period in which any member described in subdivision (a) is in the service of the department or unit, if the member demonstrates that he or she was exposed, while in the service of the department or unit, to a known carcinogen as defined by the International Agency for Research on Cancer, or as defined by the director.
c. The compensation that is awarded for cancer shall include full hospital, surgical, medical treatment, disability indemnity, and death benefits, as provided by this division.

d. The cancer so developing or manifesting itself in these cases shall be presumed to arise out of and in the course of the employment. This presumption is disputable and may be controverted by evidence that the primary site of the cancer has been established and that the carcinogen to which the member has demonstrated exposure is not reasonably linked to the disabling cancer. Unless so controverted, the appeals board is bound to find in accordance with the presumption. This presumption shall be extended to a member following termination of service for a period of three calendar months for each full year of the requisite service, but not to exceed 60 months in any circumstance, commencing with the last date actually worked in the specified capacity.
e. The amendments to this section enacted during the 1999 portion of the 1999–2000 Regular Session shall be applied to claims for benefits filed or pending on or after January 1, 1997, including, but not limited to, claims for benefits filed on or after that date that have previously been denied, or that are being appealed following denial.

**3212.8.**

(a) In the case of members of a sheriff's office, of police or fire departments of cities, counties, cities and counties, districts, or other public or municipal corporations or political subdivisions, or individuals described in Chapter 4.5 (commencing with Section 830) of Title 3 of Part 2 of the Penal Code, whether those persons are volunteer, partly paid, or fully paid, and in the case of active firefighting members of the Department of Forestry and Fire Protection, or of any county forestry or firefighting department or unit, whether voluntary, fully paid, or partly paid, excepting those whose principal duties are clerical or otherwise do not clearly fall within the scope of active law enforcement service or active firefighting services, such as stenographers, telephone operators, and other office workers, the term "injury" as used in this division, includes a blood-borne infectious disease or methicillin-resistant *Staphylococcus aureus* skin infection when any part of the blood-borne infectious disease or methicillin-resistant *Staphylococcus aureus* skin infection develops or manifests itself during a period while that person is in the service of that office, staff, division, department, or unit. The compensation that is awarded for a blood-borne infectious disease or methicillin-resistant *Staphylococcus aureus* skin infection shall include, but not be limited to, full hospital, surgical, medical treatment, disability indemnity, and death benefits, as provided by the workers' compensation laws of this state.

**Government Code 31720.5.**

If a safety member, a fireman member, or a member in active law enforcement who has completed 5 years or more of service under a pension system established pursuant to Chapter 4 (commencing with Section 31900) or

under a pension system established pursuant to Chapter 5 (commencing with Section 32200) or both or under this retirement system or under the State Employees' Retirement System or under a retirement system established under this chapter in another county, and develops heart trouble, such heart trouble so developing or manifesting itself in such cases shall be presumed to arise out of and in the course of employment. Such heart trouble so developing or manifesting itself in such cases shall in no case be attributed to any disease existing prior to such development or manifestation.

As used in this section, "fireman member" includes a member engaged in active fire suppression who is not classified as a safety member.

As used in this section, "member in active law enforcement" includes a member engaged in active law enforcement who is not classified as a safety member.

**31720.6.**

a. If a safety member, a firefighter, or a member in active law enforcement who has completed 5 years or more of service under a pension system established pursuant to Chapter 4 (commencing with Section 31900) or under a pension system established pursuant to Chapter 5 (commencing with Section 32200) or both or under this retirement system or under the Public Employees' Retirement System or under a retirement system established under this chapter in another county, and develops cancer, the cancer so developing or manifesting itself in those cases shall be presumed to arise out of and in the course of employment. The cancer so developing or manifesting itself in those cases shall in no case be attributed to any disease existing prior to that development or manifestation.
b. Notwithstanding the existence of nonindustrial predisposing or contributing factors, any safety member, firefighter member, or member active in law enforcement described in subdivision (a) permanently incapacitated for the performance of duty as a result of cancer shall receive a service-connected disability retirement if the member demonstrates that he or she was exposed to a known carcinogen as a result of performance of job duties.

   "Known carcinogen" for purposes of this section means those carcinogenic agents recognized by the International Agency for Research on Cancer, or the Director of the Department of Industrial Relations.
c. The presumption is disputable and may be controverted by evidence, that the carcinogen to which the member has demonstrated exposure is not reasonably linked to the disabling cancer, provided that the primary site of the cancer has been established. Unless so controverted, the board is bound to find in accordance with the presumption. This presumption shall be extended to a member following termination of service for a period of

three calendar months for each full year of the requisite service, but not to exceed 60 months in any circumstance, commencing with the last date actually worked in the specified capacity.

d. "Firefighter," for purposes of this section, includes a member engaged in active fire suppression who is not classified as a safety member.
e. "Member in active law enforcement," for purposes of this section, includes a member engaged in active law enforcement who is not classified as a safety member.

**31720.7.**

a. If a safety member, a firefighter, a county probation officer, or a member in active law enforcement who has completed 5 years or more of service under a pension system established pursuant to Chapter 4 (commencing with Section 31900) or under a pension system established pursuant to Chapter 5 (commencing with Section 32200), or both, or under this retirement system, under the Public Employees' Retirement System, or under a retirement system established under this chapter in another county, develops a blood-borne infectious disease, the disease so developing or manifesting itself in those cases shall be presumed to arise out of, and in the course of, employment. The disease so developing or manifesting itself in those cases shall in no case be attributed to any disease existing prior to that development or manifestation.
b. Any safety member, firefighter, county probation officer, or member active in law enforcement described in subdivision (a) permanently incapacitated for the performance of duty as a result of a blood-borne infectious disease shall receive a service-connected disability retirement.
c. (1) The presumption described in subdivision (a) is rebuttable by other evidence. Unless so rebutted, the board is bound to find in accordance with the presumption. (2) The blood-borne infectious disease presumption shall be extended to a member following termination of service for a period of three calendar months for each full year of the requisite service, but not to exceed 60 months in any circumstance, commencing with the last date actually worked in the specified capacity. (3) Notwithstanding paragraph (2), the methicillin-resistant *Staphylococcus aureus* skin infection presumption shall be extended to a member following termination of service for a period of 90 days commencing with the last day actually worked in the specified capacity.
d. "Blood-borne infectious disease," for purposes of this section, means a disease caused by exposure to pathogenic microorganisms that are present in human blood that can cause disease in humans, including, but not limited to, those pathogenic microorganisms defined as blood-borne pathogens by the Department of Industrial Relations.

e. "Member in active law enforcement," for purposes of this section, means members employed by a sheriff's office, by a police or fire department of a city, county, city and county, district, or by another public or municipal corporation or political subdivision or who are described in Chapter 4.5 (commencing with Section 830) of Title 3 of Part 2 of the Penal Code or who are employed by any county forestry or firefighting department or unit, except any of those members whose principal duties are clerical or otherwise do not clearly fall within the scope of active law enforcement services or active firefighting services, such as stenographers, telephone operators, and other office workers, and includes a member engaged in active law enforcement who is not classified as a safety member.

**31720.9.**

a. If a peace officer member, as defined in Sections 830.1–830.5, inclusive, of the Penal Code, or firefighter member, with service under a pension system established pursuant to Chapter 4 (commencing with Section 31900) or under a pension system established pursuant to Chapter 5 (commencing with Section 32200), or both, or under this retirement system, under the Public Employees' Retirement System, or under a retirement system established under this chapter in another county, becomes ill or dies due to exposure to a biochemical substance, the illness that develops or manifests itself in those cases shall be presumed to arise out of, and in the course of, employment. The illness that develops or manifests itself in those cases shall in no case be attributed to any illness existing prior to that development or manifestation.
b. Any peace officer member or firefighter member, as described in subdivision (a), who becomes permanently incapacitated as a result of exposure to a biochemical substance shall receive a service-connected disability retirement.
c. The presumption described in subdivision (a) is rebuttable by other evidence. Unless rebutted, the board is bound to find in accordance with the presumption. This presumption shall be extended to a member following termination of service for a period of three calendar months for each full year of the requisite service, but not to exceed 60 months in any circumstance, commencing with the last date actually worked in the specified capacity.
d. For purposes of this section, a peace officer member or firefighter member, as described in subdivision (a), does not include a member whose principal duties are clerical or otherwise do not clearly fall within the scope of active law enforcement services or active firefighting services, such as stenographers, telephone operators, and other office workers.
e. "Biochemical substance" means any biological or chemical agent that may be used as a weapon of mass destruction, including, but not limited to, any chemical warfare agent, weaponized biological agent, or nuclear or radiological agent, as these terms are defined in Section 11417 of the Penal Code.

However, presumptive legislation for cancer in firefighters is a relatively new phenomenon. It was first passed in Manitoba in 2002. Exhibit 2 is the Presumptive Legislation of Manitoba, which began the modern movement and provided the template for presumptive legislation for cancer in firefighters in North America and now Australia (See Chap. 13).

**Exhibit 2. Presumptive Legislation of the Province of Manitoba**
C.C.S.M. c. W200 The Workers Compensation Act [5]

**Cause of occupational disease**

4(4) Where an injury consists of an occupational disease that is, in the opinion of the board, due in part to the employment of the worker and in part to a cause or causes other than the employment, the board may determine that the injury is the result of an accident arising out of and in the course of employment only where, in its opinion, the employment is the dominant cause of the occupational disease.

**Presumption**

4(5) Where the accident arises out of the employment, unless the contrary is proven, it shall be presumed that it occurred in the course of the employment; and, where the accident occurs in the course of the employment, unless the contrary is proven, it shall be presumed that it arose out of the employment.

**Definitions**

4(5.1) In this section,

**"full-time firefighter"** means a full-time member of a fire fighting department; (« pompier à temps plein »)

**"OFC personnel"** means personnel of the office of the fire commissioner, as provided for in *The Fires Prevention and Emergency Response Act*, whose duties include

(a) investigating the cause, origin and circumstances of fires,

(b) fire fighting, or

(c) delivering fire investigation or fire fighting training; (« membre du personnel du bureau du commissaire aux incendies »)

**"part-time firefighter"** means a casual, volunteer or part-time member of a municipal fire brigade. (« pompier à temps partiel »)

**Presumption re cancer: firefighters and OFC personnel**

4(5.2) If a worker who is or has been a full-time firefighter, a part-time firefighter or a member of OFC personnel suffers an injury that is

(a) a primary site brain cancer;
(b) a primary site bladder cancer;
(c) a primary site kidney cancer;
(d) a primary non-Hodgkin's lymphoma;
(e) a primary leukemia;
(f) a primary site colorectal cancer;
(g) a primary site ureter cancer;
(h) a primary site lung cancer;

(i) a primary site esophageal cancer;
(j) a primary site testicular cancer;
(k) multiple myeloma;
(l) a primary site prostate cancer;
(m) a primary site skin cancer; or
(n) a primary site breast cancer;

the injury must be presumed to be an occupational disease the dominant cause of which is the employment as a firefighter or as a member of OFC personnel, unless the contrary is proven.

**Application of presumption re cancer**

4(5.3) The presumption in subsection (5.2) applies to a worker

(a) who has been employed as a full-time firefighter, a part-time firefighter or a member of OFC personnel for a minimum period prescribed by the Lieutenant Governor in Council by regulation; and

(b) who has been regularly exposed to the hazards of a fire scene, other than a forest-fire scene, throughout that period of employment.

**Additional requirement re lung cancer**

4(5.4) In addition to the requirements of subsection (5.3), the presumption for a primary site lung cancer applies only to a worker who has been a non-smoker immediately before the day of the accident for a minimum period of time prescribed by the Lieutenant Governor in Council by regulation.

**Effective date of presumption re cancer**

4(5.5) The presumption in subsection (5.2) applies to accidents that happen to

(a) full-time firefighters on or after January 1, 1992; or
(b) part-time firefighters or OFC personnel on or after June 9, 2005.

**Presumption re heart injury: firefighters and OFC personnel**

4(5.6) If a worker who is a full-time firefighter, a part-time firefighter or a member of OFC personnel suffers an injury to the heart within 24 h after attendance at an emergency response, the injury must be presumed to be an accident arising out of and in the course of the employment, unless the contrary is proven.

**Regulations**

4(5.7) The Lieutenant Governor in Council may make regulations

(a) prescribing minimum periods of employment for the purpose of subsection (5.3), which may be

  (i) different for different diseases set out in subsection (5.2), and
  (ii) different for full-time firefighters, part-time firefighters and OFC personnel;

(b) prescribing the minimum period of time for which a worker must be a non-smoker for the purpose of subsection (5.4).

4(6) Repealed, S.M. 1989–90, c. 47, s. 4.

The Manitoba model of presumptive legislation really has taken off across Canada and Australia and there are a number of legal reasons why this is the case. The first is that it has to be justified morally in order to be supported by both political leaders and the public opinion. This legislation basically says that although we realize that not every cancer that fire fighters get is work related there are nevertheless scientific reasons to suggest that many cancers that fire fighters are diagnosed with are occupational in nature. The Manitoba model of presumptive legislation is a good trade off as it starts with the legal premise that for a firefighter to be covered under this legislation he or she must first meet some requirements. The firefighter must have worked a certain number of years as a firefighter and been exposure to toxins prior to being diagnosed with cancer. Not every cancer is occupational in nature and the cancers that are currently covered in Manitoba have strong scientific evidence to show a link between cancer causing agents and firefighting. Also, the cancer must be primary site and cannot have originated in a part of the body that is not covered under the legislation. The strength of this legislation is that as the science expands and becomes more sophisticated and sensitive to issues surrounding cancer in firefighters, so does the number of cancers that are covered under this legislation.

In 2011 a similar piece of legislation was passed in Australia. This all-encompassing piece of legislation applies to federal employees and jurisdictions and now fully covers all aspects of firefighting and occupational cancer. Again, it is based on solid, scientific evidence and is directly impacted by the work of IARC of the World Health Organization. Individual Australian states are debating and adopting similar legislation, one by one.

Presumptive legislation is now in place in three English-speaking countries. Canada, Australia and the United States all have jurisdictions with some type of presumptive legislation. In the United States at the time of this writing (2014) presumptive legislation is in place in 40 of 50 states. In Canada, it is in place in 11 of 13 provinces and territories. In Australia, it is in place for the Federal Government of Australia and in three of the six states (Western Australia, Tasmania, South Australia), and is currently being actively debated in the others. There are active movements in the jurisdictions in each of the three countries that lack presumptive legislation get it passed, advocated by firefighters' unions. For example, In Québec there has been repeated advocacy efforts for provincial legislation but a bill has never been introduced and so the issue has never gone to a floor vote; cases must be argued one at a time on the basis of the province's legislation on exposure to cancer-causing agents and "poisoning". In some states, such as Missouri, presumptive legislation is on the books but achieving recognition of occupational cancer is still difficult.

So far there is no legislation of this kind in the world outside of North America and Australia, although at the time of the writing of this book (2014) many European countries were very close to introducing some type of presumptive cancer legislation for firefighters. The legislation is gaining support in Sweden, Iceland, Finland, Denmark, Norway and Germany, among other countries.

## How Presumptive Legislation Works

The three pieces of presumptive legislation that are serving as models internationally, the Australian federal, the Manitoban and the Californian, allow for an easier legal road for fire fighters to have their cancers accepted as occupational in nature. Possibly the most important aspect of this legislation is that it allows a fire fighter easier access to proper workers' compensation so that the fire fighter can concentrate on battling the disease and not the entities that block the coverage.

Existing law provides that the presumptions listed above are disputable and may be controverted by evidence. However, unless controverted, the Workers' Compensation Appeals Board must find in accordance with the presumption. Controversion takes the form of a rebuttal, which can be on the grounds that the legislation does not apply, that the person was not a covered firefighter, that there was an insufficient period or employment or insufficient exposure, that insufficient time had elapsed for a disease with latency (such as cancer), that the disease had another cause more likely than firefighting (smoking and family history being the most common), or that the disease is actually something else than what is claimed.

Legislated presumptions come with grounds for *rebuttal*, or reasons to argue that occupation was not the causal factor. A "rebuttable presumption,+ applied to firefighters, simply states that firefighters with a particular disease and set of characteristics (starting with duration of service) will automatically be deemed eligible for compensation unless there is strong evidence that occupation did not cause the condition. Such evidence might include an intense smoking history, a hereditary predisposition to the disease (usually argued on the basis of family history), or another occupational exposure, such as prior exposure to asbestos.

Presumption shifts the burden of proof from the claimant to those who would challenge the relationship with work, usually employers or compensation insurance carriers. This relieves a substantial and onerous burden from the claimant, especially for cancer and other chronic disease cases, in which exposures and the sequence of likely events are difficult or even impossible to document, especially years later. It also relieves the system of having to listen repeatedly to arguments on causation, based on the same body of evidence for general causation, in each and every similar case.

Presumptions are usually but not always based on evidence that the association with occupation is causal and strong. The alternative to accepting a particular diagnosis as qualification for compensation is not to reject all cases. If a condition is not recognized as compensable, the alternative is to examine the particulars of the individual case to see if there is a reason to conclude that the condition arose out of work.

**Exhibit 3. Presumptive Legislation in Australia (Commonwealth of Australia, 2011)**

*Safety, Rehabilitation and Compensation Amendment (Fair Protection for Firefighters) Bill 2011 [6]*

**Outline**

The Safety, Rehabilitation and Compensation Amendment (Fair Protection for Firefighters) Bill 2011 (the Firefighters Bill) seeks to amend the *Safety, Rehabilitation and Compensation Act 1988* (the SRC Act) to simplify workers' compensation claims by firefighters who have contracted a range of prescribed cancers, and who have been employed for a certain period, by establishing a rebuttable presumption that the cancers are work-related.

Under this presumption, if a firefighter is diagnosed with one of the twelve cancers listed in the Bill, and has served as a firefighter for the relevant qualifying period, it will be presumed that the cancer is an occupational disease and is therefore compensable.

For each of the specified cancers, the Bill also includes a minimum length of service for which a firefighter must have been engaged in order to access workers' compensation under the presumption. The specific cancer types and the associated minimum qualifying service periods are listed below.

| Cancer Type | Qualifying Period of Service |
|---|---|
| Primary site brain cancer | 5 years |
| Primary site bladder cancer | 15 years |
| Primary site kidney cancer | 15 years |
| Primary non-Hodgkin's lymphoma | 15 years |
| Primary leukemia | 5 years |
| Primary site breast cancer | 10 years |
| Primary site testicular cancer | 10 years |
| Multiple myeloma | 15 years |
| Primary site prostate cancer | 15 years |
| Primary site ureter cancer | 15 years |
| Primary site colorectal cancer | 15 years |
| Primary site oesophageal cancer | 25 years |

The presumption proposed by the Bill would be accessible only by firefighters that are covered by the SRC Act.

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# Chapter 13
# How Presumptive Legislation Got Started in Manitoba: A Personal History

**Alex Forrest**

The tragic irony of our job is that a profession that we love so much is so hard on us!

At this time in Manitoba we are covered by presumption legislation for 14 cancers and heart injury that occurs within 24 h of an emergency response. Presumptive legislation is needed not only to provide justice and fair compensation for firefighters, but as an educational tool for Fire Departments. With this legislation goes great responsibility for all fire fighters to minimize their own risks of getting cancer. The educational tool is for the leadership of the fire service as it becomes part of their due diligence to make cancer prevention and awareness part of their overall health and safety program.

It is the responsibility of each and every fire fighter to ensure that he or she is educated as to the risks and knows how to minimize contact with carcinogens at fires, largely through the use of SCBA and cleaning procedures. Action must also be taken to minimize other cancer risks such as smoking, obesity, diet, health and lifestyle alongside proper and regular medical evaluation. However, firefighters can only do so much to reduce their risk of cancer and ill health. There has to be protection from exposure to hazards like cancer-causing chemicals and when protection fails, it is only right that there be fair compensation.

## One Firefighter's Journey

How did Alex Forrest get involved in advocating proper workers' compensation for firefighters stricken with occupational cancer? I am not a scientist but I can speak about firefighter health as a firefighter, a firefighter health and safety advocate and a lawyer.

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You have to look at someone's background in order to understand why that person does what he or she does. I grew up in a small town in Alberta, in a blue collar, lower income family. My grandfather on my mother's side died of black lung after working in a coal mine in Drumheller, Alberta and my father died of a heart attack on the job when I was 13.

At 17 I signed up for the Canadian Military and became a soldier. I always say the second-best thing I ever did in my occupational life was to join the army. The best thing I ever did was leave the army! I needed the discipline of the military but I grew to challenge the establishment and to question orders and directions. That last part does not play well in the military and as a result I was involved in many bouts of minor discipline. For some reason the military leadership does not appreciate criticism no matter how wrong the orders may be and so I ultimately became a firefighter, a lawyer and a union leader. It may seem like a very unlikely and unusual combination, but for me the three professions blend together perfectly.

On one hand you have the noble and well respected profession of firefighting. Firefighting is one of the most physical jobs a person can do, largely due to the fact that for many centuries the way we fight fires has remained a tremendously physical endeavor. Many times a successful rescue or a successful attack of a fire is directly related to the physical strength of firefighters.

I guess you could say that being a firefighter is one of the few strictly physical occupations left as it has not been dramatically changed by technology. On the other hand there is the maybe lesser respected but equally difficult profession of lawyer. With this profession it is strictly your intellect and your ability to reason that decides whether or not you win a case.

The last piece of the puzzle is union leader, which in my case is a union leader for the great profession of firefighting. This is one of the greatest honors that could be given to me. I love firefighting, I love my fellow firefighters and I love the profession itself. To be an advocate for such a great bunch of human beings is very humbling.

I am currently Union President for the United Firefighters of Winnipeg (UFFW), a position I have held since the late 1990's. I am also the Canadian Trustee for the International Association of Fire Fighters (IAFF), the only nationally-elected union position in the IAFF for a Canadian firefighter. I like the ability to question authority and in many cases give the authorities a good kick in the butt when they refuse to acknowledge mistakes, especially when they impact a firefighter's health and safety or the quality of the service provided to the public. Did I ever mention that the military should have some type of labour organization? But that's another book!

I was a firefighter for 6 years before I graduated law school and was called to the bar in the Province of Manitoba. I pursued my studies in law while I worked as a full time professional firefighter in Winnipeg's station number 5, which at the time was one of the busiest stations in Canada. I did this with the support of my fellow firefighters, who assisted me in switching all of my 10 h day shifts to 14 h night shifts for basically 4 years.

After law school I continued as a firefighter and also began a private law practice in Winnipeg. At that time the firefighters' union was going through a really difficult

time in almost all aspects of labour management and political relations. I began attending union meetings but I got frustrated with what was happening and with the policies of our union. One thing lead to another and in a very short time I ran for the presidency of the union and was elected president of the UFFW.

When I was elected I had no idea of the scope of the cancer risk for firefighters. That would soon change and my life would also be forever changed.

When I became president of UFFW we were in the middle of negotiations and I had a huge learning curve in regards to my job. I methodically went through all the relevant areas of the union such as members' welfare, grievances, negotiations and then workers' compensation. This was an area in which I believed I could be especially helpful, given my legal training and background.

One aspect of workers' compensation that I began to be involved with was that of occupational cancer and its connection to firefighting. Sometimes ignorance really is bliss but the more I read about cancer and its effect upon firefighters the more I found it hard to believe that there had never been one firefighter covered for occupational cancer in the history of the Province of Manitoba. In Winnipeg there were a large number of young firefighters who had been diagnosed with cancer and many had lost their battle with the disease. Attending so many funerals created the basis of my passion for what I was about to take on. It really is amazing how much time a person has to think while attending at a funeral!

Even more surprising to me was the fact that nowhere in Canada was there any legislation that allowed for a firefighter who had been diagnosed with cancer to be covered under workers' compensation. Firefighters were dying of cancer at an alarming rate and yet there was no acknowledgement that their deaths were connected to their job.

There were some jurisdictions in the US that had some type of coverage, but it was different than workers' compensation coverage would be in Canada. Many American jurisdictions dealt with the issue through medical care or pensionable instruments, but at least they recognized that there was a connection between firefighting and certain cancers. This was not the case in any province of Canada or in anywhere else in the world for that matter. Firefighters in Canada and Australia owe a great deal of gratitude to our American Brothers and Sisters in the IAFF, as they were the pioneers on this issue.

In dealing with this issue I started off very slowly but I kept reading various studies that suggested a connection between cancer and firefighting. I also began to look at the very nature of firefighting and to break it down so that I could better understand just how and why cancer is a part of a firefighter's career. The issue of cancer is made clear to every firefighter at every fire when we smell the smoke and know that we have come into contact with carcinogens. We know that these carcinogens not only get onto our skin but also into our pores, into our blood stream and into our major organs. We have the best protective firefighting clothing in the world, but, (and this is a very large "but) it cannot fully protect us. I now believe that there is no greater danger to today's firefighters than occupational diseases such as cancer.

For years, I worked hard to understand this issue on many levels. As a lawyer I understood that it is broken down into legal onus and balance of probabilities and

that led me to believe that the legal system within workers' compensation was ill equipped to deal with the nature of firefighter cancer. I worked to educate myself in the area of medicine and epidemiology, as I needed to understand exactly how and why human beings get cancer.

I also needed to understand the world of statistical significance and scientific analysis and I needed to understand the studies and the medical issues on the same level as the experts. One thing I have had to deal with is the attitude of many of the doctors and experts in that we are "merely firefighters". I will never forget the experience I had with one doctor when a young firefighter was excreting blood from his anus and his family doctor told him he was too young to worry about prostate or colon cancer. The doctor said that it was likely hemorrhoids, as he was only 39 years old, but if it were to get any worse then they would speed up the tests for cancer. I went to that doctor's office and explained to him that this person had been fighting fires for almost 20 years and he should therefore be placed at the top of the queue for cancer testing. The doctor was a little taken aback when I told him that we would take this firefighter to the US, pay for the testing and also go to the media. I told him that if it turned out that the firefighter had cancer then we would be back to "kick his ass" and after that he put the firefighter on the top of the list for testing. It turned out that the firefighter had the early stages of cancer. Today the battle still exists to have family doctors deal with firefighters in the same way as a person with a genetic predisposition to cancer.

I soon realized that as a union we were not as successful as we could be because we were not politically active. I learned very quickly that everything we do and every aspect of our job involves politics, whether it is a monetary issue or a legislative issue, all aspect of our job are affected by politics.

We do not have the luxury of saying that we are above the political world. We cannot have outsiders making decisions about how we fight fires and we cannot have politicians making decisions that affect our safety without our input. We also cannot have politicians making life and death decisions for us when they do not have a good understanding of the nature of our job and its dangers. We cannot leave politics alone because politics will not leave us alone!

Many firefighters believe that the people in politics who oversee our work genuinely support us and therefore give us proper legislation to ensure that we have the resources we need to fight fires and save lives. This is something that they do not question. These firefighters also believe that their political leaders will always ensure that the families of firefighters killed in the line of duty will be properly assisted by workers' compensation. This is naïve and just not true. Many politicians look at decisions involving our profession in purely political terms; they look at the cost, they look at their own personal agendas and above all they make decisions based on political considerations that are not solely in the best interest of either public safety or firefighters' health and safety.

My comments may lead you to believe that I have a great distrust and dislike of politicians, but that is far from the truth. There are some politicians who I find hard to respect but there are others who I have come to admire and I see them as great leaders.

Presumptive legislation could not have occurred without two things; science and political leadership. Many prominent politicians played a role in this story, including former Manitoba Premier Gary Doer, former Alberta MLA Richard Magnus, former Manitoba Emergency Measures Minister Steve Ashton, current Manitoba Premier Greg Selinger and of course former Winnipeg Mayor Sam Katz as well as our Australian political friends, Federal Senators Gavin Marshall and Penny Wright and Member of Parliament for Melbourne and Deputy Leader of the Australian Greens, Adam Brandt. These people have been nothing but supportive on these issues.

Needless to say, when I became president of UFFW I knew that success for us would mean that we would have to become political, and we were about to get political to a level that would make us one of the premier lobby groups in Manitoba and in Canada.

I was very fortunate to start my career in the union movement at the time when Harold Schaitberger had just became General President of the IAFF. Harold Schaitberger is one of the most dynamic union leaders in the world and his style of leadership and his belief in the importance of politics made him a mentor for me. Harold was a firefighter in Fairfax County in Virginia, a tough, right-to-work state that did not look kindly upon "Those union guys". He started in the union in Fairfax, then the Virginia state organization and he finally worked his way up the international ladder to the highest position in the largest firefighter union in the world.

Later in my work I met another great labour leader, Peter Marshal, General Secretary of the United Firefighters of Australia (Fig. 13.1). I met Peter at a Global Alliance meeting of the world's firefighter unions and I grew to admire him and learn from him. He has political savvy combined with a passion for representing his members and I admire what he has done and continues to do. The work that he has done for his members in Australia benefits firefighters all over the world.


**Fig. 13.1** Alex Forrest with Peter Marshall of UFFA and Manitoba Premier Greg Selinger

Everything was set; I had a mentor and a strategy in regards to our political involvement. I just needed to convince the UFFW Executive Council and membership of the validity of this new direction.

## Rick's Story

Rick was a firefighter's firefighter. Throughout his career he worked in the tough North End of Winnipeg. He hardly ever took a day off sick. He had no family history of cancer and he was 100 % fit and healthy.

As a child he lived in the North End and as he grew up he was a common sight around the local fire hall. When he turned 18 he applied for a position with the Winnipeg Fire Department but he was turned down. He kept applying and applying until he finally realized his dream and became a Winnipeg firefighter. Not only did he work in the North End but he truly realized his dream when he became Captain in the famous North End fire hall.

Unfortunately, this great firefighter started to have headaches for the first time in his life before he was even 50 years of age. When they became more intense he went to see his doctor and was diagnosed with advanced brain cancer, a type of cancer that his doctor told us was usually reserved for people who worked in the plastics industry. He was immediately put on a treatment regimen and then had surgery to remove the tumour. It was removed but quickly returned larger than before and Rick and his family were advised that the cancer was terminal and another operation would not be of any use. He was given weeks, maybe months to live.

Rick knew that we were fighting with workers' compensation at that time and that we had never had a single firefighter's cancer claim accepted as occupational in nature. Rick also knew that we were going to the politicians asking for legislation that would allow for firefighter occupational cancer claims to be accepted. We had asked for this in Manitoba in the 1990s under the Conservative government of Gary Filmon but each time we were turned down. We found out years later that the Conservative government had known that there was a link between firefighting and occupational cancer but they denied the legislation because they believed that it would be too expensive.

In 1999 we worked hard to get Gary Doer and the New Democratic Party (NDP) government elected in Manitoba and as a result we had a premier who was sympathetic to the tragedy of occupational cancer in firefighters. We also had a very sharp Minister of Labour by the name of Becky Barrett who believed in what the occupational cancer studies had to say. She worked to enact legislation that would allow for the recognition of cancer as an occupational disease related to firefighting. Becky Barrett actually met Rick Stoyko before he was diagnosed, as he was a resident of her riding. She met him while she was going house to house canvassing the electorate. It really is amazing how life works out sometimes!

After Rick Stoyko was diagnosed with brain cancer he came to me and explained his situation. He also said he wanted to help! He wanted to spend some of his precious

**Fig. 13.2** Captain Rick Stoyko, Manitoba Minister of Labour Becky Barrett and Alex Forrest at the media conference on the day that the first Canadian legislation was introduced in Manitoba

last days helping us to explain why firefighters needed this protection. He would always say "Firefighters are always there for the public 24/7 and we never let them down. Right now firefighters need the help of the public".

Rick Stoyko became the major spokesperson for the cause and he came with me to meetings with politicians, the media, other firefighters and even medical professionals. We knew that we were going to lose him soon but we did not want to lose his message and so we produced a video featuring Rick and his family. Rick became weaker and weaker but he kept on helping us until finally the day came when Manitoba was about to be the first province in Canada to enact presumptive WCB cancer legislation. Rick and I attended countless meetings and press conferences and we were also both in attendance at the Manitoba legislature on the day the legislation was introduced (Fig. 13.2.). Hundreds of firefighters filled the gallery of the legislature to witness the historic moment. Rick was with me at 5 am doing morning TV news shows and he was with me throughout the day doing countless TV, newspaper and radio interviews with media from across Canada. I believe our final media appearance was around 9 pm as we spoke on an evening news program.

Throughout that day, Rick was with his family and his firefighter friends, as he needed their support. You could see the energy leaving his body but he refused to stop. He told me he only had a few days left but this day was for his brothers and sisters in the fire service.

At one press conference after the legislation was introduced we gathered in the media room of the Manitoba legislature; Rick Stoyko, Becky Barrett and I sitting at the media table addressing the dozens of media and describing the historic significance of this legislation.

The room was packed with Rick's family and his firefighting family but you could see that Rick was exhausted as we answered question after question. I told Rick that any time he gave me the nod we would end the news briefing but he said no, as he knew the national media was in attendance and we had to get the message out, beyond the province of Manitoba to other provinces and jurisdictions.

Throughout all of this there was one defining moment when Rick was asked a question from the press gallery, "Mr. Stoyko, I have read all the studies and we have heard stories of how dangerous firefighting is. We are sorry about your terminal cancer but the question I have is, do you regret your decision to be a firefighter?"

Rick said, "All I ever wanted to be was a firefighter and when I became a firefighter my first Captain died of brain cancer in my rookie years. I have seen so many of my fellow firefighters die of cancer so early in life. Despite this I can tell you that I have talked about this with my family and I can tell you that if there is a thing such as reincarnation I want to come back as a firefighter".

I grew very close to Rick during this time and the funny thing is that he did not initially support me when I ran for Union President. That was something I never let him forget, but he would just laugh about it.

This is the tragic irony of our job: the profession of firefighting is dangerous and no one knows that better than firefighters, but this profession that is so hard on us and our families is a profession we love deeply. This is the dedication, sacrifice and honor of firefighting.

In February of 2003 Rick Stoyko died of occupational cancer. He became the first firefighter in the history of Canada to be covered under presumptive legislation for occupational cancer. On that day I cried for the first time since my father died and so did many of Rick's firefighter brothers and sisters.

Rick was given full departmental line-of-duty death funeral honors by the Winnipeg Fire Department and the city of Winnipeg. Hundreds of Winnipeg firefighters attended and marched alongside others from almost every major fire department in Canada. They were there to pay tribute to a great firefighter!

In almost every province of Canada and in jurisdictions throughout the United States, Australia and Europe I play the video that Rick recorded in 2002. I play it for firefighters, politicians and media as a way to educate them about this danger that is faced every day by firefighters around the world.

I also want to say that although it was the International Association of Firefighters (IAFF) and our Union that took the lead role on presumptive cancer legislation, we also had the support of many fire chiefs, including Fire Chief Robert Simonds who is now at the Hamilton Fire Department, Fire Chief Ken Thevenot, now in Okotoks Fire Department and Edmonton Fire Chief Ken Block who has become one of the leading advocates in the world for proper workers' compensation coverage for firefighters with occupational cancer. I know that the firefighters of Australia agree with me when I say that Chief Fire Officer Ken Block's testimony on the costs of presumptive legislation at the Australian Senate Inquiry was one of the main reasons why Australia now has presumptive legislation throughout their country (pp 30 Australian Senate Report 2011).

As a fire fighter trying to understand the studies and the risk of cancer due to exposure, I have come to the following conclusions:

1. Medical studies are not the result of an exact science and if you are expecting black and white answers to questions about risk factors and which cancers are most prevalent in firefighting then you will be disappointed. This is due to factors beyond the control of the study itself, such as the available sample size and other confounding issues that interfere with the conclusion.
2. Even though science is not able to give a comprehensive assessment of the cancer risk, it is clear that fire fighters will come into contact with cancer causing agents at fires and that they will not be fully protected from carcinogens.
3. Science agrees that our protective equipment does not completely protect us from cancer causing agents.
4. Study after study shows a connection between firefighting and cancer.
5. Science strongly suggests that the risk factors of cancer in fire fighters are likely underestimated, largely due to the "Healthy Worker Effect". Fire fighters are healthier than the general population and so our cancer rates should therefore be lower than in the general population.
6. Each study must be looked at as if it is one piece of the puzzle and no one study is definitive regarding the issue of firefighting and cancer.
7. Fire fighters all over the world are exposed to the same types of toxins. This is due to the global use of plastics. When I travel around the world this is the first myth that I must deal with. A fire in Canada has the same cancer causing carcinogens as in the U.S.A., Australia or Europe.

Every year the International Association of Firefighters holds a memorial to all our members who have died in the line-of-duty and we are now seeing that close to 60 % of those deaths are due to occupational cancer. I am writing this as a tribute to the many firefighters I have known who have fought the good fight against occupational cancer. We have buried too many brothers and sisters because of cancer we know to have been caused by their work saving others. What a tragic irony!

# About the Editor

Tee L. Guidotti is an international consultant in occupational and environmental health and medicine, currently practicing in Washington DC and at the time of this writing serving as a Fulbright Visiting Chair at the University of Ottawa in the Institute for Science, Society, and Policy.

Until recently he conducted his consulting practice through Medical Advisory Services of Rockville, Maryland, where he still holds the title of Senior Scientist, but he now conducts his consulting as a private individual, sometimes operating under the sole-proprietership Occupational + Environmental Health & Medicine.

Dr. Guidotti has had a long career in academia as a tenured full professor of occupational and environmental medicine. He retired in 2008 as Professor and Chair of the Department of Environmental and Occupational Health (GW) in the School of Public Health and Health Services, The George Washington University Medical Center, Washington DC, and Director of the Division of Occupational Medicine and Toxicology in the School of Medicine and Health Sciences. Prior to taking the positions at GW in 1999, he was for 15 years Professor of Occupational and Environmental Medicine and Director of the Occupational Health Program in the Department of Public Health Sciences at the University of Alberta in Edmonton, Canada, where in 1996 he was named a Killam Annual Professor.

Dr. Guidotti is perhaps best known in occupational medicine for his expertise on the more conventional occupational health problems of municipal firefighters and of oil and gas workers. His other interests include inhalation toxicology (especially products of combustion), air quality studies, risk science, and the evaluation of scientific evidence in law, workers' compensation and public policy. He combined these interests in a series of studies that ultimately laid the foundation for legislated presumptions for certain cancers among firefighters in Canada, led by the province of Manitoba, and the United States. His work has since been influential in the adoption of presumption for selected cancers in the US, Canada, and Australia. Most recently, he prepared an exhaustive review of the literature on firefighter's health for the Australian Department of Veteran's Affairs. He remains engaged in the issue

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and still participates in research, serving on the advisory committee of the Australian Firefighters' Study based at Monash University.

Dr. Guidotti was a local clinician-evaluator in Washington DC for World Trade Center (WTC) first responders and recovery personnel, and coordinated evaluations at the OM&TC for the WTC Medical Monitoring Program and the Red Cross Recovery Program. He subsequently served as an expert for the New York City firefighters in their lawsuit against the city for inadequate protection during the event, which was settled in 2010. He retained a strong interest in WTC issues and was concerned based on cases he had reviewed that an important health outcome (constrictive bronchiolitis) might be overlooked because it is difficult to diagnose. He worte up his findings and shared them with NIOSH in 2011. Eventually this led to a collaboration with many of the New York City-based investigators, including David Prezant and Rafael de la Hoz, and publication of a lengthy review in the *American Journal of Industrial Medicine* on the anniversary of the "9–11" event. He has served on other WTC-related bodies, including grant review committees for the National Institute of Occupational Health and Safety.

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Dr. Guidotti's academic interests have centered on occupational and environmental lung disease and cancer, air quality and airborne hazards, risk science, health and sustainability, and the evaluation of scientific evidence in law and public policy.

Dr. Guidotti is the author or coauthor of over 200 publications and has produced seven books, including *Global Occupational Health, Occupational Health Services: A Practical* Approach (in its second edition), *Science on the Witness Stand* (which argues for a true weight-of-evidence approach to adjudication in compensation and litigation), *Basic Environmental Health* (in its time the most popular graduate-level introductory textbook in environmental health), *The Canadian Guide to Health and the Environment* (a bestseller for the University of Alberta Press). Dr. Guidotti wrote the solo-authored three-volume *Praeger Handbook of Occupational and Environmental* Medicine, published in 2010, and *Health and Sustainability: An Introduction*, published in 2015.

Dr. Guidotti is the Editor in Chief of *Archives of Environmental and Occupational Health: An International Journal*. He is a Past President of both the American College of Occupational and Environmental Medicine and the Association of

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