FIREFIGHTING

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1. Exposure Data

1.1 Activities and tasks of firefighters

The terms 'firefighting' and 'firefighters' are broad and encompass several types of fire scenarios such as municipal, wildland, industrial, aviation, military, and oil wells. Some municipal firefighters may be permanently assigned to tasks other than fighting fires, including fire scene investigation (i.e. the investigation of suspected criminal fires started by arsonists), hazardous material response, building safety inspections, or technical and administrative support. These individuals may or may not have experience fighting fires, and may or may not be working for municipal fire departments. In addition, municipal firefighters are increasingly being called upon for emergency medical response. Finally, the term "firemen" may refer either to firefighters or to individuals who operate and maintain equipment for power generation (e.g. steam boilers), heating, ventilation, humidity control, refrigeration, and air conditioning. Workers in this latter category are also referred to as "stationary engineers" or "stationary firemen" (Decoufle *et al*., 1977), and are not considered in this monograph.

There are two more or less distinct phases in municipal structural firefighting: knockdown and overhaul. During knockdown, firefighters control and extinguish the fire. Approximately 90% of municipal structural fires are either extinguished within 5–10 minutes, or abandoned and fought from the outside. This results in an average duration of heavy physical activity at fires of approximately 10 minutes (Gempel & Burgess, 1977; Gilman & Davis, 1993). Knockdown of large fires may last much longer. During overhaul, any remaining small fires are extinguished. The environment during overhaul is not as hot or as smoky as during knockdown, but it still contains products of combustion from small fires or smouldering material. Exposure can differ widely between the two phases of firefighting. The determination of when overhaul begins varies from one fire department to another, and is often left to the judgement of individual firefighters or group leaders (Jankovic *et al*., 1991; Austin *et al.*, 2001a). Municipal structural fires may

be fought in aggressive attack mode during knockdown, or defensively from the outside. In the past, firefighters may have more often attempted to enter a burning structure to fight the fire. For safety reasons, however, modern fire departments are increasingly adopting a defensive approach, unless there are human victims inside the building.

A municipal fire department is composed of 1st line firefighters (pump, ladder, and rescue crews, and operations chiefs) and 2nd line firefighters (drivers and division chiefs). Combat firefighters assigned to pump trucks, ladder trucks, or rescue trucks perform tasks specific to each of those crews. In some municipalities, there is movement of firefighters between different firehalls, while in others, a firefighter is assigned to the same crew at the same firehall for most of his or her career. It is conceivable that there would be differences in exposures between pump truck and ladder truck crews, although no such difference was observed in one older study (Gold *et al*., 1978).

In addition to fighting accidental fires and criminal fires, firefighters and firefighter recruits may be involved in training fires staged in buildings or simulators. Hill *et al*. (1972) describe a permanent structure used for training purposes where approximately 5500 litres of diesel fuel was burned in the lower portion of the building.

Analogous to knockdown and overhaul, wildland firefighting also comprises two phases, referred to as "attack" and "mop-up." Attack at a wildland fire generally extends over a long period of time, one fire lasting hours, days or weeks. The frequency of aggressive strategies and tactics by firefighters may increase where there is an attempt to save residential developments. Municipal firefighters may also be called upon to fight wildland fires within or adjacent to municipal limits.

Both municipal firefighters and wildland firefighters engage in heavy work activity at fires. In particular, wildland firefighters who use hand tools and carry a considerable amount of equipment with them engage in heavy work activity levels while fighting forest fires (Budd *et al*., 1997; Ruby *et al*., 2002). Typical tasks include hiking, fire-line construction, chainsaw work, and brush removal. As a result, the amount of chemicals inhaled is greater for a firefighter at heavy work levels without respiratory protection than for a worker engaged in regular levels of work (Reh & Deitchman, 1992; Reh *et al.*, 1994). This needs to be taken into consideration when comparing exposure levels to occupational exposure limits that were developed assuming regular work levels.

Also, studies relating to municipal firefighters usually do not distinguish between the different categories of exposed and unexposed firefighters or between the different task assignments.

1.2 Composition of fire smoke

1.2.1 *Fire chemistry*

Smoke from fires comprises suspended liquid and solid particulate matter, gases and vapours that result from the combustion or pyrolysis of material. There is a very large number of toxic components in smoke (for reviews, see Tuve, 1985; Meyer, 1989; DiNenno *et al*., 2002; Côté, 2003). The basic form of the overall combustion reaction of organic (carbon-containing) compounds is illustrated by the burning of methane:

$$CH_4 + 2O_2 + \text{Energy of activation} \rightarrow CO_2 + 2H_2O + \text{Heat of combustion} + \text{Light}$$

Given the appropriate ratio of fuel (wood, solvent, plastic, rubber), oxygen, and combustion temperature, the products of combustion should be only water and carbon dioxide (CO_2).

Complete combustion is approached only under carefully controlled conditions. Uncontrolled or unintentional combustion tends to be "fuel rich" and therefore incomplete. The combustion of methane (CH_4) illustrates the formation of free radicals in an 11-step chain reaction, the first two of which are:

$CH_4 \rightarrow CH_3^{\bullet} + H^{\bullet}$

$H^{\bullet} + O_2 \rightarrow OH^{\bullet} + O^{\bullet}$

The free radicals formed during combustion are very reactive and side reactions are propagated to yield hundreds of chemical products, and smoke.

Most polymers found in buildings will burn or thermally degrade to simpler monomers. Thermal degradation products include methane, ethane, ethylene, benzene, toluene, and ethylbenzene in addition to the following monomers: ethylene, vinyl chloride, acrylonitrile, tetrafluoroethylene, styrene, methyl methacrylate, ethylene glycol, terephthalic acid, phenol, formaldehyde, hexamethylenediamine, adipic acid, propene, vinyl chloride, vinyl acetate, vinylidene chloride, chloroprene, 1,3-butadiene, ethyl acrylate, ethylene oxide, methylacrylate, urea, phenol, and isoprene.

The burning of plastics typically produces voluminous amounts of soot, together with higher levels of hydrogen cyanide (HCN), hydrochloric acid (HCl) and acrolein (CH_2=CHCHO) than the burning of materials such as wood, and fossil fuels. More smoke evolves from fires involving aromatic polymers, such as polystyrene, compared to aliphatic polymers, such as polyethylene.

In addition to the chemical agents described above, particulate matter is produced under conditions of incomplete combustion. The particulate matter is an aerosol consisting of condensed phase components of the products of combustion and finely divided carbon particulates that have not undergone combustion but remain suspended in the air. Although the particles themselves are microscopic in size (0.3–1.6 μm), they

rapidly coalesce and thereby become visible. These particles are also adsorbents (similar to activated charcoal) and are an additional vehicle for the transport and inhalation of toxic combustion products. Smouldering yields a substantially higher conversion of fuel to toxic compounds than does flaming, although it occurs more slowly (Ohlemiller, 2002).

1.2.2 *Modern versus pre-modern fires*

All types of fire release toxic and carcinogenic substances, including benzene, 1,3-butadiene, and formaldehyde. The focus has generally been on substances having short-term acute effects: carbon monoxide (CO), carbon dioxide, hydrogen cyanide, nitrogen oxides (NO_x), sulfur dioxide (SO_2) and hydrogen chloride. With the increasing use of polymers in building construction and furnishings, there is concern that the burning of these new materials might release large quantities of other highly toxic substances (Austin *et al*., 2001b).

Combustion and pyrolysis products from newer building materials and furnishings were believed to be more toxic than smoke from fires in buildings built before these materials became commonplace, and more toxic than smoke from wildland fires (Betol *et al*., 1983; Alarie, 1985). However, many of the carcinogenic products of combustion identified are volatile organic compounds and are common to most burning materials. In a more recent study, no new or unusual non-polar volatile organic compounds (VOCs) were observed in current structural fires compared to the combustion of wood (Austin *et al*., 2001b, 2001c). Adding polyvinyl chloride (PVC) to the fire load at simulated apartment fires was observed to significantly increase levels of polychlorinated phenols (IARC Group 2B), while polycyclic aromatic hydrocarbon (PAH) levels remained essentially unchanged (Ruokojärvi *et al*., 2000). The increases in levels of polychlorinated biphenyls (PCBs, 0.021 to 0.031 mg/m^3), polychlorinated benzenes (0.002 to 0.010 mg/m^3) and I-TEQs [or PCDD/F] (3.5 to 5.4 ng/m^3) as products of combustion were not significant [possibly due to the small sample size]. In another study, proportionately higher levels of ethyl benzene (IARC Group 2B) were found at an electronics factory fire when compared to levels at residential and mixed occupancy fires (Austin *et al*., 2001b).

The emission of combustion products (in mg per kg of material burned) for the same material varies greatly depending on combustion conditions such as ventilation (oxygen supply), temperature, and heating rate. Nonetheless, the relative amounts of the various non-polar VOCs found in smoke at municipal structural fires have been found to be remarkably similar from fire to fire, namely with the same 14 of 144 target compounds, dominated by benzene (IARC Group 1), toluene and naphthalene (IARC Group 2B) (Austin *et al*., 2001b, 2001c).

1.2.3 *Carcinogens found in smoke at fires*

Table 1.1 lists the agents in Groups 1, 2A, and 2B that have been detected at fires in one or more studies, together with corresponding IARC evaluations, human and animal evidence of carcinogenicity, and for the agents in Group 1, the cancer sites in humans.

Table 1.1. IARC evaluations and cancer sites in humans of chemicals measured at fires

Chemicals measured at fires	Overall evaluation	Human evidence	Animal evidence	Volume	Cancer sites in humans (For Group 1 agents only)
Acetaldehyde	2B	Inadequate	Sufficient	36, Suppl. 7, 71	
Arsenic	1	Sufficient	Limited	23, Suppl. 7	Skin, lung, liver (angiosarcoma)
Asbestos	1	Sufficient	Sufficient	14, Suppl. 7	Lung, mesothelioma, larynx, gastrointestinal tract
Benz[*a*]anthracene	2B	Inadequate	Sufficient	32, Suppl. 7, 92	
Benzene	1	Sufficient	Limited	29, Suppl. 7	Leukaemia
Benzo[*b*]fluoranthene	2B	No data	Sufficient	32, Suppl. 7, 92	
Benzo[*k*]fluoranthene	2B	No data	Sufficient	32, Suppl. 7, 92	
Benzofuran (coumarone)	2B	No data	Sufficient	63	
Benzo[*a*]pyrene	1	No data	Sufficient	32, Suppl. 7, 92	Lung, bladder, skin
1,3-Butadiene	1	Sufficient	Sufficient	71, 97	Lymphohaematopoietic system
Cadmium	1	Sufficient	Sufficient	58	Lung
Carbon black (total)	2B	Inadequate	Sufficient	65, 93	
Chrysene	2B	Inadequate	Sufficient	3, 32, Suppl. 7, 92	
Dibenz[*a*,*h*]anthracene	2A	Inadequate	Sufficient	32, Suppl. 7, 92	
Dichloromethane (methylene chloride)	2B	Indadequate	Sufficient	71	
Ethylbenzene	2B	Inadequate	Sufficient	77	
Formaldehyde	1	Sufficient	Sufficient	88	Nasopharynx; (nasal sinuses and leukaemia, suggested)
Furan	2B	Inadequate	Sufficient	63	

Table 1.1 (contd)

Chemicals measured at fires	Overall evaluation	Human evidence	Animal evidence	Volume	Cancer sites in humans (For Group 1 agents only)
Indeno-1,2,3-[*cd*]pyrene	2B	Inadequate	Sufficient	32, Suppl. 7, 92	
Isoprene	2B	Not available	Sufficient	60, 71	
Lead				23, Suppl. 7, 87	
Lead compounds, organic	3	Inadequate	Inadequate	23, Suppl. 7, 87	
Lead compounds, inorganic	2A	Limited	Sufficient	23, Suppl. 7, 87	
Naphthalene	2B	Inadequate	Sufficient	82	
2-Nitroamisole	2B	Inadequate	Sufficient	65	
Polychlorophenols	2B	Limited		41, Suppl. 7, 53, 71,	
Pentachlorophenol			Sufficient		
2,4,6-Trichlorophenol			Limited		
Polychlorinated biphenyls (aroclor; 54%) (chlorodiphenyl)	2A	Limited	Sufficient	18, Suppl. 7	
Polychlorinated dibenzodioxins[a]: see TCDD					
Radioactivity (γ activity)	1	Sufficient	Sufficient	78	All sites combined
Radionuclides (α-particle-emitting)	1	Sufficient	Sufficient	78	All sites combined
Radionuclides (β-particle-emitting)	1	Sufficient	Sufficient	78	All sites combined
Silica (crystalline)	1	Sufficient	-	68	Lung
Silica (amorphous)	3	Inadequate	Inadequate	68	

Table 1.1 (contd)

Chemicals measured at fires	Overall evaluation	Human evidence	Animal evidence	Volume	Cancer sites in humans (For Group 1 agents only)
Styrene	2B	Limited	Limited	60, 82	
Sulfuric acid[b]	1	Sufficient	No data	54	
2,3,7,8-tetrachloro dibenzo-*para*-dioxin	1	Limited	Sufficient	69	All sites combined, lung, non-Hodgkin lymphoma, sarcoma
Tetrachloroethylene (perchloroethylene)	2A	Limited	Sufficient	63	Cervix, oesophagus, non-Hodgkin lymphoma
Toluene diisocyanates	2B	Inadequate	Sufficient	39, Suppl. 7, 71	
Trichloroethylene	2A	Limited	Sufficient	63	Liver and biliary tract, non-Hodgkin lymphoma, renal cell
Trichloromethane (chloroform)	2B	Inadequate	Sufficient	73	
Triphenylene	3	Inadequate	Inadequate	32, Suppl. 7, 92	

[a] Polychlorinated dibenzo-*para*-dioxins as a group are classified in Group 3
[b] Evaluation of occupational exposures to strong inorganic acid mists containing sulfuric acid

1.3 Exposure

1.3.1 *Characterization of firefighter exposures*

The characterization of exposures to fire gases and smoke is challenging due to several factors: work schedules of 10- to 24-hour shifts for 188 days in a year; wide variations between firefighters' time spent at fires; intermittent exposures; exposure to a complex mixture of gases, vapours and particulate matter; unknown effect of heat; gases and free radicals may also be adsorbed onto particulate matter; some semivolatile organic compound (SVOC) vapours measured in the air may be distributed between the solid and vapour phase, this equilibrium shifting in either direction depending on the temperature and on the density of the smoke; and, the difficulty in collecting samples at unpredictable locations in a dangerous and rapidly changing environment.

Given the multitude of chemicals in smoke, some substances may produce metabolites that alone or in combination with other substances or metabolites may become hazardous.

1.3.2 *Time spent at fires*

The number of runs and the time spent at fires varies tremendously between firehalls, depending on the geographic location, the social and economic environment, staffing, and the types of call (number of fires, types of fire, medical calls, hazardous materials [HAZMAT]).

Probably as a result of improved building codes compared to past decades, municipal firefighters today spend surprisingly little time at fires. In a study in Montreal, the time spent at fires was calculated based on an extensive database compiled over a period of 12 months (Austin *et al*., 2001a). Firefighters from the least busy firehalls responded to approximately eight structural fires per year or 19 fires of all kinds, spending 15.1 hour/yr per firefighter at fires. Firefighters from the busiest firehalls responded to 3.13 times as many structural fires per year (25 structural fires, or 62 fires of all kinds), and spent 3.3 to 3.6 times as long at fires (54 hours). This study did not distinguish between 1st line and 2nd line firefighters. However, based on discussions with the fire department, it was estimated that 2nd line combat firefighter exposures were less than 50% those of 1st line combat firefighters. Overall, firefighters responding to fires spent between 0.75% and 2.7% of their time at fires over the course of a year. More recently, Kales *et al*. (2007) used a similar method to estimate time spent at fires for a municipal fire department in the USA, and national data supplied by the National Fire Protection Association (NFPA) and the International Association of Fire Fighters (IAFF) to produce estimates for smaller fire departments and large metropolitan fire departments, respectively. Firefighters spent 1%, 2%, and 5% of their time at fires in small, municipal, and metropolitan fire departments, respectively. This would represent 20–100 hours per year. Kales *et al*. (2007) estimated

that firefighters responded to an average of 1.7 (Standard Deviation (SD), 0.1) to 7.0 (SD, 6.3) fire incidents per year.

Burgess *et al*. (2003) estimated the time spent inside structural fires broken down by tasks for two fire departments in Arizona, USA. The results were: entry/ventilation 5.7 ± 11.7 hour/yr (Phoenix), and 3.5 ± 3.7 hour/yr (Tucson); rescue 5.0 ± 8.0 hour/yr (Phoenix), and 2.1 ± 2.7 hour/yr (Tucson); knockdown (extinction) 5.6 ± 8.9 hour/yr (Phoenix), and 4.5 ± 4.4 hour/yr (Tucson); overhaul 15.0 ± 3.7 hour/yr (Phoenix), and 20.8 ± 76.8 hour/yr (Tucson); and, support/standby 16.3 ± 28.6 hour/yr (Phoenix), and 19.1 ± 76.7 hour/yr (Tucson). Total firefighter activity at fires in Phoenix and Tucson was a mean of 47.6 hour/yr and 50.0 hour/yr, respectively.

In a study among firefighters in Washington, DC, ($n = 43$), at the time of the survey, an average of 9.2 days had elapsed since the last fire. Also, 0.33 fires had been fought in the previous 24 hours, 1.33 in the previous week, 5.91 in the previous month, and 57.1 fires in the previous year (Liou *et al*., 1989).

Little information is available concerning the time that firefighters outside of North America spend at fires. The organization and practices of fire departments might differ, and a greater number of fires may occur at other locations. In one study in Inchon, Republic of Korea, firefighters were questioned about their firefighting activity during the previous 5 days; among these, 33% (24 of 73) had had no fire exposure, 49% (36 of 73) had had less than 8 hours' fire exposure, and 18% (13 of 73) had had more than 8 hours' exposure to fire (Hong *et al*., 2000). Four of 13 volunteer firefighters in Sweden reported that they had not fought any fires within the previous 3 months, while the other nine reported having fought one fire each (Bergström *et al*., 1997). All 13 firefighters had been working as active firefighters for at least 3 years.

Wildland firefighters go to fires more frequently and spend more time at fires during a season than do municipal firefighters during an entire year, and all of their exposure occurs during the wildfire season. A total of 47 California wildland firefighters were surveyed to determine the extent of their firefighting activity (Rothman *et al*., 1993). Early in the wildland fire season, firefighters reported that they had spent a mean of 0.11 hours (Standard Error (SE), 0.89) fighting fires during the previous week, 12.06 hours (SE, 2.77) during the previous 2 weeks, and 16.74 hours (SE, 3.15) during the previous 4 weeks. Firefighting activity increased significantly during the late season, when wildland firefighters reported they had spent a mean of 22.36 hours (SE, 5.03) fighting fires during the previous week, 54.81 hours (SE, 9.29) during the previous 2 weeks, and 97.38 hours (SE, 15.26) fighting fires during the previous 4 weeks (Rothman *et al*., 1993). In the USA, Hot Shot crews [highly-skilled wildland firefighters specially trained in wildland fire suppression tactics] have been estimated to spend 64 days at wildfires and 5 days at prescribed burns, on average, per year, (Booze *et al*., 2004). In Quebec, in 2005, the agency responsible for wildland firefighting reported that wildland firefighters had spent a total of 145 689 hours at fires, or 755 hours per firefighter, on average for that year (Austin, 2008).

1.3.3 *Surrogates of exposure*

As a matter of practicality, epidemiologists have generally used years of employment or, in one case, years of active duty fighting fires (Demers *et al*., 1994), as a surrogate for exposure to smoke. This does not take into account the reduction in exposures when respiratory protection was used, differences between exposure groups, the intermittent nature of exposures, differences in tasks, or the fact that not all firefighters actually combat fires. In a Montreal study, only 66% of fire department personnel were 1st line firefighters (Austin *et al*., 2001a). Years of employment has not been found to correlate with exposure to combustion products or related adverse health effects (decline in pulmonary function or airway responsiveness) (Musk *et al*., 1977; Takehito & Maeda, 1981; Sparrow *et al*., 1982; Sherman *et al*., 1989). The number of fires fought has, however, been correlated with the mean annual reduction in pulmonary function (Peters *et al*., 1974). Among firefighters at the same fire, statistically significant differences in exposure to combustion products have been found between front-line firefighters and both squad leaders and ordinary firefighters (Takehito & Maeda, 1981). The same study found no significant difference between ordinary firefighters and the officers who accompanied them.

Two epidemiological studies used estimated cumulative runs as a surrogate for exposure (Austin *et al*., 2001a; Baris *et al*., 2001). In one study (Austin *et al*., 2001a), a good correlation between the number of runs per firehall and time spent at fires was observed ($r = 0.88$). However, different crews could have similar numbers of runs yet spend significantly different lengths of time at fires. The study by Austin *et al*. (2001a) identified distinct firefighter exposure groups based on job title, fire hall assignment, and time spent at fires.

1.3.4 *Exposure to carcinogens found in smoke at fires*

Table 1.2 presents the results of the studies that have measured the substances listed in Table 1.1, and particulate matter (total, respirable, PM_{10}). Unless otherwise indicated, reported levels do not take into consideration the use of respiratory protection. Table 1.3 provides a summary of the results from Table 1.2 for each substance, according to the type of fire or exposure (i.e. wildland, municipal, training fire, or municipal fire scene (arson) investigation).

The carcinogens found in one or more studies include nine known human carcinogens (Group 1), four probable human carcinogens (Group 2A), and 21 possible human carcinogens (Group 2B) (for a review, see Bendix, 1979; Lees, 1995).

Many of the wildland and municipal firefighter studies result from opportunistic sampling with sometimes wide margins of error, and may not be representative of firefighter exposures.

Two studies reported extremely high levels of benzene, up to 165 and 250 ppm (Burgess *et al*., 1979; Brandt-Rauf *et al*., 1988, respectively) [the former study used an accurate and precise sampling and analytical methodology]. Benzene levels in the remaining studies ranged from not detected to 23 ppm.

Table 1.2. Studies of exposures of firefighters to selected chemicals and agents

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Acetaldehyde									
Jankovic *et al*. (1991), USA	Municipal		22 22 22	21 5 4	ppm ppm ppm		ND ND ND	8.1 1.6 0.9	Knockdown Overhaul Inside face mask
Kelly (1991), USA	Wildland	Shift	1	20	ppm		ND	0.1	Mop-up
NIOSH (1992), USA	Wildland	Shift	1	20	ppm		ND	ND	Mop-up
Reh *et al*. (1994), USA	Wildland		1 1	3 2	ppm ppm		0.01 0.03	0.02 0.04	Low smoke levels Medium smoke levels
Kinnes & Hine (1998), USA	Municipal	TWA	5	8	ppm		ND	0.13	Arson investigation
Bolstad-Johnson *et al*. (2000), USA	Municipal	> 20 min	25	96	ppm	0.34 ± 0.41	0.041	1.75	Overhaul lasting min 20 min
Andreae & Merlet (2001), Germany	Wildland	Multiple	Multiple data sources	–	mg/kg[a]	[607 ± 345]			Means of reported mean emissions factors for savanna and grassland, tropical forest, extratropical forest, biofuel burning, charcoal making, and agricultural fires

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Burgess *et al*. (2001), USA	Municipal (testing)	Overhaul > 25 min Overhaul < 25 min	7 9	22 19	ppm ppm	0.158 ± 0.037 0.383 ± 0.494			No respiratory protection SCBA used
Reisen *et al*. (2006), Australia	Wildland		6	25	ppm	< 0.08	ND	0.26	4 prescribed and 2 exceptional burns
Arsenic									
Turkington (1984), USA	Municipal	10–15 min	1	1	mg/m^3	0.14			–
Asbestos (chrysotile)									
Bridgman (2001), U.K.	Municipal			2	f/cm^2	[0.0029]	[0.001]	[0.0043]	Factory fire: chrysotile fibres in the weave of the outer fabric of firefighters' tunics

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Benzene									
Hill *et al*. (1972), U.K.	Training	Grab sample	NR	1	ppm	1.17			Pool fire
Burgess *et al*. (1979); Treitman *et al* (1980), USA	Municipal		NR	181/197	ppm		ND	165	Inside burning structures during latter stages of structural fires
Turkington (1984), USA	Municipal	10–15 min	1	1	ppm	1.00			
Lowry *et al*. (1985a), USA	Municipal	At fire	75	NR	ppm	Detected in most fires			Mixed type of exposure
Brandt-Rauf *et al*. (1988), USA	Municipal	30 min	6	11	ppm	[59.18 ± 83.86]	ND	250	Low smoke levels; Mostly wood structures
		30 min	2	7	ppm	[26.17 ± 30.59]	ND	83.3	Moderate smoke levels; burning mostly
		30 min	5	6	ppm	[94.87 ± 92.73]	ND	225	High/Intolerable smoke levels; building and contents
		30 min	2	2	ppm		23	34	Automobile

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Jankovic *et al*. (1991), USA	Municipal		22 22 22	15 2 4	ppm ppm ppm		ND ND ND	22 0.3 21	Knockdown Overhaul Inside mask
Kinnes & Hine (1998), USA	Municipal	TWA	5	4	ppm	trace			Arson investigation; benzene concentration between LOD and LOQ (0.04–0.12 ppm)
Bolstad-Johnson *et al*. (2000), USA	Municipal	> 20 min	25	95	ppm	0.383 ± 0.425	0.07	1.99	Overhaul
Reinhardt *et al*. (2000), USA	Wildland	Shift TWA Fireline TWA			ppm ppm	0.016* 0.028*		0.058 0.088	Prescribed burns (1991–1994)

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Reinhardt & Ottmar (2000), USA	Wildland	Shift TWA Fireline TWA Shift TWA Fireline TWA			ppm ppm ppm ppm	0.004* ± 3.6* 0.006* ± 3.6* 0.02* ± 0.003* 0.04* ± 0.14*		0.25 0.38 0.02 0.04	Project fires (1992–1995) Initial attack (1992–1995)
Andreae & Merlet (2001), Germany	Multiple	–	Multiple data sources	NR	mg/kg[a]	[693 ± 663]			Means of reported mean emissions factors for savanna and grassland, tropical forest, extratropical forest, biofuel burning, charcoal making, and agricultural fires
Austin *et al*. (2001b), Canada	Municipal	15 min	9	9	ppm	3.38 ± 3.45	0.12	10.76	7 mixed occupancy buildings, one electronics industry, one 9-day smouldering fire
Austin *et al*. (2001c), Canada	Municipal (simulated)	Grab sample	15	60	ppm	detected		0.1	In separate burns: wood (spruce), bed mattress, sofa foam, cardboard, plywood, gasoline, varsol, white foam insulation
Burgess *et al*. (2001), USA	Municipal	Overhaul >25 min Overhaul >25 min	7 9	23 20	ppm	ND 0.557 ± 0.465			No respiratory protection SCBA used
Reisen *et al*. (2006), Australia	Wildland		6	8	mg/m^3	0.12	0.002	0.26	4 prescribed and 2 exceptional burns

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Benzofuran (coumarone)									
Andreae & Merlet (2001), Germany	Wildland emissions	Multiple	Multiple data sources	–	mg/kg[a]	[19 ± 12]			Means of reported mean emissions factors for savanna and grassland, tropical forest, extratropical forest, biofuel burning, charcoal making, and agricultural fires
Austin *et al*. (2001b), Canada	Municipal	15 min	9	9	ppm		0.2	2	7 mixed occupancy buildings, one electronics industry, one 9-day smouldering fire
1,3-Butadiene									
Andreae & Merlet (2001), Germany	Wildland	Multiple	Multiple data sources	NR	mg/kg[a]	[87 ± 79]			Means of reported mean emissions factors for savanna and grassland, tropical forest, extratropical forest, biofuel burning, charcoal making, and agricultural fires
Austin *et al*. (2001b), Canada	Municipal	15 min	9	9	ppm	1.03 ± 1.49	0.03	4.84	7 mixed occupancy buildings, one electronics industry, one 9-day smouldering fire

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Austin *et al*. (2001c), Canada	Municipal (simulated)	Grab sample	15	60	ppm	detected			In separate burns: wood (spruce), bed mattress, sofa foam, cardboard, plywood, gasoline, varsol, white foam insulation
Cadmium									
Bolstad-Johnson *et al.* (2000), USA	Municipal	>20 min	25	46	–	ND			Overhaul
Carbon black (Total)									
Andreae & Merlet (2001), Germany	Wildland	Multiple	Multiple data sources	–	mg/kg[a]	[747 ± 376]			Means of reported mean emissions factors for savanna and grassland, tropical forest, extratropical forest, biofuel burning, charcoal making, and agricultural fires

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Dichloromethane (methylene chloride)									
Lowry *et al*. (1985a), USA	Municipal	At fire	75	–	ppm	detected			Mixed types of exposure
Brandt-Rauf *et al*. (1988), USA	Municipal	30min	1	1	ppm	0.280			Mostly wood structures burning mostly building and contents
Ethylbenzene									
Hill *et al*. (1972), U.K.	Training	Grab sample	NR	1	ppm	0.382			Pool fire
Lowry *et al*. (1985a), USA	Municipal	At fire	75	NR	ppm	detected			Mixed type of exposure
Andreae & Merlet (2001), Germany	Wildland	Multiple	Multiple data sources	NR	mg/kg[a]	[54 ± 58]			Means of reported mean emissions factors for savanna and grassland, tropical forest, extratropical forest, biofuel burning, charcoal making, and agricultural fires
Austin *et al*. (2001b), Canada	Municipal	15 min	9	9	ppm	0.86 ± 1.94	0.01	5.97	7 mixed occupancy buildings, one electronics industry, one 9-day smouldering fire

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Austin *et al*. (2001c), Canada	Municipal (simulated)	Grab sample	15	60	ppm	measured			In separate burns: wood (spruce), bed mattress, sofa foam, cardboard, plywood, gasoline, varsol, white foam insulation
Formaldehyde									
Turkington (1984), USA	Municipal	10–15 min	1	1	ppm	0.71			
Lowry *et al*. (1985a), USA	Municipal	At fire	75	–	ppm	5.0	1	15	Mixed types of exposure
Brandt-Rauf *et al*. (1988), USA	Municipal	30min	6	11	ppm	0.12 ± 0.27	ND	0.8	Mostly wood structures burning with low smoke levels
		30 min	2	7	ppm	0.49 ± 1.24	ND	3.3	Mostly wood structures with moderate smoke levels
		30 min	5	6	ppm	1.74 ± 3.67	ND	8.3	Mostly wood structures with high/intolerable smoke levels
		30 min	2	2		ND			Automobile
Jankovic *et al*. (1991), USA	Municipal	NR	22	16	ppm		ND	8	Knockdown
			22	5	ppm		ND	0.4	Overhaul
			22	5	ppm		ND	0.3	Inside mask

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Kelly (1991), USA	Wildland	Shift	1	20	ppm		ND	0.1	Mop-up
NIOSH (1992), USA	Wildland	Shift	1	20	ppm			0.07	Mop-up
Materna *et al*. (1992), USA	Wildland	Fireline TWA	4 fire seasons	30	ppm	0.16	0.048	0.42	Project fires (1987–1989); mop-up
Reh & Deitchman (1992), USA	Wildland	NR	3 1 1	NR 3 2	ppm ppm ppm		ND 0.01 0.06	0.03 0.02 0.07	Low smoke levels Low smoke levels Medium smoke levels
Kinnes & Hine (1998), USA	Municipal	TWA	5	3	ppm		0.06	0.18	Arson investigation
Bolstad-Johnson *et al.* (2000), USA	Municipal	> 20 min	25	96	ppm	0.25 ± 0.252	0.016	1.18	Overhaul
Reinhardt & Ottmar (2000), USA	Wildland	Fireline TWA	NR		ppm ppm	0.018* ± 2.3* 0.028* ± 3*		0.093 0.092	Project fires (1992–1995) Initial attack (1992–1995)
		Shift TWA			ppm ppm	0.013* ± 2.4* 0.006* ± 3.1*		0.084 0.058	Project fires (1992–1995) Initial attack (1992–1995)

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Reinhardt *et al*. (2000); Slaughter *et al*. (2004), USA	Wildland	Fireline TWA Shift TWA.	NR		ppm ppm	0.075* 0.047*		0.6 0.39	Prescribed burns (1991–1994) Prescribed burns (1991–1994)
Andreae & Merlet (2001), Germany	Wildland	Multiple	Multiple data sources	NR	mg/kg[a]	[1347 ± 978]			Means of reported mean emissions factors for savanna and grassland, tropical forest, extratropical forest, biofuel burning, charcoal making, and agricultural fires
Burgess *et al*. (2001), USA	Municipal	Overhaul > 25 min	7	22	ppm	0.190 ± 0.182			No respiratory protection
		Overhaul > 25 min	9	19	ppm	0.257 ± 0.249			SCBA used
Reisen *et al*. (2006), Australia	Wildland		6	25	ppm	0.230	0.04	0.79	4 prescribed and 2 exceptional burns
Free radicals (short-lived)									
Jankovic *et al*. (1993), USA	Municipal	At fire At fire	7 10	7 10	counts/min counts/min		ND ND	127 920	Knockdown Overhaul

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Free radicals (long-lived)									
Lowry *et al*. (1985b), USA	Municipal (simulated)	1 h; 2 room changes	6	6	ppm			1200	"Low energy fire" producing minimal radiant heat; burning 2 kg of paper, cotton and polyester clothing, plastics (including PVC), and wood
		1 h	6	–	ppm			1000	
Jankovic *et al*. (1993), USA	Municipal	At fire	–	–	detected by ESR	detected	–	–	Knockdown
		At fire	–	–	–	detected	–	–	Overhaul
Leonard *et al*. (2000), USA	Wildland	3.5 h	1	6	–	detected			Experimental fire
Leonard *et al*. (2007), USA	Wildland	3.5 h	1	6	–	detected			Mop-up and back-burn operations
Furan									
Lowry *et al*. (1985a), USA	Municipal	At fire	75	–	ppm	detected			Mixed types of exposure

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Andreae & Merlet (2001), Germany	Wildland	Multiple	Multiple data sources	–	mg/kg[a]	[508 ± 265]			Means of reported mean emissions factors for savanna and grassland, tropical forest, extratropical forest, biofuel burning, charcoal making, and agricultural fires
Austin *et al*. (2001b), Canada	Municipal	15 min	9	9	ppm		0.2	2	7 mixed occupancy buildings, one electronics industry, one 9-day smouldering fire
Isoprene									
Hill *et al*. (1972), UK	Training	Grab sample	–	1	ppm	0.167			Pool fire
Andreae & Merlet (2001), Germany	Wildland	Multiple	Multiple data sources	–	mg/kg[a]	[34 ± 36]			Means of published emissions factors for savanna and grassland, tropical forest, extratropical forest, biofuel burning, charcoal making, and agricultural fires
Lead									
Turkington (1984), USA	Municipal	10–15 min	1	1	mg/m^3	1.4			

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Bolstad-Johnson *et al*. (2000), USA	Municipal	> 20 min	25	46	mg/m^3	0.03	0.03	0.03	Overhaul lasting minimum 20 min
Naphthalene									
Hill *et al*. (1972), U.K.	Training	Grab sample	–	1	ppm	0.418			Pool fire
Kinnes & Hine (1998), USA	Municipal	TWA	5	5	$\mu g/m^3$		200	0.038	Arson investigation
Bolstad-Johnson *et al.* (2000), USA	Municipal	> 20 min	25	88	ppm	0.043 ± 0.019	0.014	0.103	Overhaul lasting minimum 20 min
Austin *et al*. (2001b), Canada	Municipal	15 min	9	9	ppm	0.62 ± 0.68	0.01	2.14	7 mixed occupancy buildings, one electronics industry, one 9-day smouldering fire
Austin *et al*. (2001c), Canada	Municipal (simulated)	Grab sample	15	60	ppm			3	In separate burns: wood (spruce), bed mattress, sofa foam, cardboard, plywood, gasoline, varsol, white foam insulation

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Particulate matter, PM_{10}									
Miranda *et al*. (2005), Portugal	Wildland	15 min average	1	–	mg/m^3	–	–	3.0	Near the fire
Particulate matter, respirable									
Kelly (1991), USA	Wildland	Shift	1	26	mg/m^3		0.040	4.3	Mop-up
NIOSH (1992), USA	Wildland	Shift	1	20	mg/m^3	0.49			Mop-up
Materna *et al*. (1992), USA	Wildland	Fireline TWA	5 fire seasons	22	mg/m^3	1.75	0.327	5.14	Project fires (1987–1989); mop-up
					mg/m^3		0.235	2.71	Prescribed burns
McMahon & Bush (1992), USA	Wildland	2.8 h	14		mg/m^3	1.3**	0.2	3.7	Prescribed burn
Reh & Deitchman (1992), USA	Wildland		1	3	mg/m^3		1.3	1.7	Medium smoke levels
Reh *et al*. (1994), USA	Wildland		1	3	mg/m^3		0.6	1.1	Low smoke levels

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Kinnes & Hines (1998), USA	Municipal	TWA	5	5	mg/m^3		ND	1.2	Arson investigation
Bolstad-Johnson *et al.* (2000), USA	Municipal	> 20 min	25	93	mg/m^3	8.01 ± 8.02	0.71	25.7	Overhaul lasting a minimum of 20 minutes
Reinhardt & Ottmar (2000), USA	Wildland	Shift TWA Fireline TWA Shift TWA Fireline TWA	NR	NR	mg/m^3 mg/m^3 mg/m^3 mg/m^3	0.5* ± 2* 0.7* ± 1.9* 0.022* ± 2.5* 1.11* ± 1.6*		2.3 2.9 1.6 2.5	Project fires (1992–1995) Initial attack (1992–1995)
Reinhardt *et al.* (2000); Slaughter *et al.* (2004), USA	Wildland	Shift TWA Fireline TWA			mg/m^3 mg/m^3	0.6* 1*		6.9 10.5	Prescribed burns (1991–1994)
Andreae & Merlet (2001), Germany	Wildland	NR	Multiple data sources	–	mg/kg^a	[7933 ± 3206]			Means of reported mean emissions factors for savanna and grassland, tropical forest, extratropical forest, biofuel burning, charcoal making, and agricultural fires

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Burgess *et al*. (2001), USA	Municipal	Overhaul > 25 min Overhaul > 25 min	7 9	24 19	mg/m^3	ND 6.180 ± 7.800			No respiratory protection SCBA used
Miranda *et al*. (2005), Portugal	Wildland	15 min average	1	NR	mg/m^3			3.0	Near the fire
Particulate matter, total									
Hill *et al*. (1972), U.K.	Training	Grab sample	NR	NR		–			Pool fire; 80% of particle with diameter < 1 μm
Gold *et al*. (1978), USA	Municipal	~10 min	–	90	mg/m^3	21.5* ± 4.7*	4	650	Knockdown and overhaul
Burgess *et al*. (1979); Treitman *et al*. (1980), USA	Municipal		–	66	mg/m^3		ND	20000	Inside burning structures during latter stages of structural fires (knockdown)
Turkington (1984), USA	Municipal	10-15 min	1	1	mg/m^3	36			–

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Atlas *et al*. (1985), USA	Training	During fire smoke	6	11	mg/m^3	47*	–		200 L of diesel oil floating on a pool of water
								300	Heavy smoke levels
			6	11	mg/m^3		0.15	0.5	
Froines *et al*. (1987), USA	Municipal	Shift	0	7	mg/m^3		0.035	0.48	Diesel emissions in firehalls (4 New York, 2 Boston, 4 Los Angeles)
		Shift	0	9	mg/m^3	0.748			
Brandt-Rauf *et al*. (1988), USA	Municipal	30 min	24	5	mg/m^3	83 ± 131	10.1	344	Mostly wood structures burning mostly building and contents
Duclos *et al*. (1990), USA	Wildland	12 days	1	NR	mg/m^3		0.578	4.158	
Jankovic *et al*. (1991), USA	Municipal		22	4	mg/m^3		ND	560	Knockdown
			22	25	mg/m^3		ND	45	Overhaul
Materna *et al*. (1992), USA	Wildland	Fireline TWA	6 fire seasons	22	mg/m^3	9.46	2.7	37.4	Project fires (1987–1989); mop-up
McMahon & Bush (1992), USA	Wildland	0.3–1.6 h	14		mg/m^3	6.3**	2	44.9	Prescribed burns

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Reh & Deitchman (1992), USA	Training		3	NR	mg/m^3		0.1	47.7	
Kinnes & Hine (1998), USA	Municipal	Peak TWA	5 5	5 5	mg/m^3 mg/m^3		3.5 0.2	31.6 5.3	Arson investigation
Bolstad-Johnson *et al*. (2000), USA	Municipal	> 20 min	25	46	mg/m^3	1.82 ± 8.73	0.364	30.79	Overhaul lasting a minimum of 20 min
Reinhardt *et al*. (2000), USA	Wildland	Shift TWA Fireline TWA Shift TWA Fireline TWA			mg/m^3 mg/m^3 mg/m^3 mg/m^3	1.5* ± 1.7* 1.7* ± 1.8* 1.39* ± 1.2* 5.32* ± 1.4*		4.2 4.4 1.81 8.64	Project fires (1992–1995) Initial attack (1992–1995)
Andreae & Merlet (2001), Germany	Wildland	Multiple	Multiple data sources	NR	mg/kg^a	[10114 ± 4512]			Means of reported mean emissions factors for savanna and grassland, tropical forest, extratropical forest, biofuel burning, charcoal making, and agricultural fires

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Reisen *et al*. (2006), Australia	Wildland		6	21	mg/m^3	0.2– > 9		8–20	4 prescribed and 2 exceptional burns; p gravimetric: 2.6–5 mg/m^3 (n = 2)
Leonard *et al*. (2007), USA	Wildland	3.5 h	1	6					Mop-up and back burn operations; 20.2% ultrafine particles (0.042–0.24 μm mean diameter); 43.8% fine particles (0.42–2.4 μm mean diameter)
Pentachlorophenol									
Ruokojärvi *et al*. (2000), Finland	Municipal (simulated)	During fire During fire	5 2	5 2	$\mu g/m^3$ $\mu g/m^3$	53 ± 45 230 ± 99	14 160	104 300	Apartment without PVCs Apartment with PVCs
Polychlorinated biphenyls (Aroclor; 54%)									
Ruokojärvi *et al*. (2000), Finland	Municipal (simulated)	During fire During fire	5 2	5 2	$\mu g/m^3$ $\mu g/m^3$	21 ± 16 31 ± 35	2.8 6.1	36 56	Apartment without PVCs Apartment with PVCs
Polychlorinated dibenzodioxins									
PCDD									
Ruokojärvi *et al*. (2000), Finland	Municipal (simulated)	During fire During fire	5 2	5 2	ng/m^3 ng/m^3	43 ± 49 69 ± 5.7	12 75	130 83	Apartment without PVCs Apartment with PVCs

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
PCDD/F as I-TEQ									
Ruokojärvi *et al*. (2000), Finland	Municipal (simulated)	During fire During fire	5 2	5 2	ng/m^3 ng/m^3	3.5 ± 2.5 5.4 ± 0.71	1 4.9	7.2 5.9	Apartment without PVCs Apartment with PVCs
PCDF									
Ruokojärvi *et al*. (2000), Finland	Municipal (simulated)	During fire During fire	5 2	5 2	ng/m^3 ng/m^3	96 ± 56 131 ± 24	21 114	160 148	Apartment without PVCs Apartment with PVCs
Polycyclic Aromatic Hydrocarbons									
Feunekes *et al*. (1997), Netherlands	Training	0.5–1.5 h	≥ 1	10	mg/m^3	10.68			Intense firefighting, black smoke
Ruokojärvi *et al*. (2000), Finland	Municipal (simulated)	During the fire During the fire	5 2	5 2	mg/m^3 mg/m^3	121 ± 199 117 ± 33	6.4 94	470 140	Apartment without PVCs Apartment with PVCs
Andreae & Merlet (2001), Germany	Wildland emissions	–	Multiple data sources	–	mg/kg[a]	[21 ± 9]			Means of reported mean emissions factors for savanna and grassland, tropical forest, extratropical forest, biofuel burning, charcoal making, and agricultural fires

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Benz[*a*]anthracene									
Jankovic *et al*. (1991), USA	Municipal		3	3	mg/m^3	0.015		0.03	Knockdown
			3	3	mg/m^3	0.001		0.003	Overhaul
Kinnes & Hine (1998), USA	Municipal	TWA	5	5	mg/m^3		ND	0.00029	Arson investigation
Bolstad-Johnson *et al*. (2000), USA	Municipal	> 20 min	25	88	mg/m^3	0.0249 ± 0.0049	0.019	0.028	Overhaul lasting a minimum of 20 min
Benzofluoranthenes, unspecified									
Atlas *et al*. (1985), USA	Training	During fire	1	1	mg/m^3	0.0124			200 L of diesel oil floating on a pool of water
									Heavy smoke levels
			1	1	mg/m^3	0.00014			Very light smoke levels
Benzo[*b*]fluoranthene									
Jankovic *et al*. (1991), USA	Municipal		3	3	mg/m^3	0.006		0.012	Knockdown
			3	3		ND			Overhaul

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Kinnes & Hines (1998), USA	Municipal	TWA	5	5	mg/m^3		ND	0.00021	Arson investigation
Bolstad-Johnson *et al.* (2000), USA	Municipal	> 20 min	25	88	mg/m^3	0.0223 ± 0.0106	0.01	0.034	Overhaul
Benzo[*k*]fluoranthene									
Jankovic *et al*. (1991), USA	Municipal		3 3	3 3	mg/m^3 mg/m^3	0.003 0.001		0.006 0.004	Knockdown Overhaul
Kinnes & Hine (1998), USA	Municipal	TWA	5	5	mg/m^3		ND	0.00012	Arson investigation
Bolstad-Johnson *et al*. (2000), USA	Municipal	> 20 min	25	88	mg/m^3	0.0238 ± 0.0017	0.023	0.025	Overhaul
Benzo[*a*]pyrene									
Turkington (1984), USA	Municipal	10–15 min	1	1	mg/m^3	0.007			–

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Atlas *et al*. (1985), USA	Training	During fire	1	1	mg/m^3	0.00855	–		200 L of diesel oil floating on a pool of water.
								–	Very heavy smoke levels
		During fire	1	1	mg/m^3	4.5×10^{-5}			Very light smoke levels
Jankovic *et al*. (1991), USA	Municipal		3	3	mg/m^3	0.01		0.02	Knockdown
			3	3		ND			Overhaul
Feunekes *et al*. (1997), Netherlands	Training	0.5–1.5 h	≥ 1	10	mg/m^3	0.47			Intense firefighting; black smoke
Kinnes & Hine (1998), USA	Municipal	TWA	5	5	mg/m^3		ND	0.00039	Arson investigation
Bolstad-Johnson *et al.* (2000), USA	Municipal	> 20 min	25	88	mg/m^3	0.0332 ± 0.0136	0.019	0.05	Overhaul

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Chrysene									
Jankovic *et al*. (1991), USA	Municipal		3 3	3 3	mg/m^3 mg/m^3	0.01 0.001		0.02 0.003	Knockdown Overhaul
Bolstad-Johnson *et al.* (2000), USA	Municipal	> 20 min	25	88	mg/m^3	0.0129			Overhaul
Chrysene/triphenylene									
Atlas *et al*. (1985), USA	Training	During fire	1	1	mg/m^3	0.0181			200 L of diesel oil floating on a pool of water Very heavy smoke levels
		During fire	1	1	mg/m^3	0.00014			Very light smoke levels
Dibenzo[*a*,*h*]anthracene									
Jankovic *et al*. (1991), USA	Municipal		3 3	3 3	mg/m^3	0.003 ND		0.005	Knockdown Overhaul
Bolstad-Johnson *et al.* (2000), USA	Municipal	> 20 min	25	88	mg/m^3	0.0455 ± 0.0316	0.023	0.068	Overhaul

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Indeno-1,2,3-[*cd*]pyrene									
Jankovic *et al*. (1991), USA	Municipal		3	3	mg/m^3	0.01		0.02	Knockdown
Kinnes & Hine (1998), USA	Municipal	TWA	5	5	µg/m^3		ND	0.44–1.4	Arson investigation
Bolstad-Johnson *et al.* (2000), USA	Municipal	> 20 min	25	88	mg/m^3	0.0195 ± 0.0084	0.014	0.029	Overhaul
Radioactivity									
Volkerding (2003), USA	Wildland	2 days	1	4	Bq/m^3	–	2 x 10^{-4}	9 x 10^{-4}	α Emitters
			1	4	Bq/m^3	–	8 x 10^{-4}	0.004	β Emitters
			1	4	Bq/filter	–	ND	45.5	Bismuth-212
			1	4	Bq/filter	–	2.4	46.3	Lead-212
			1	4	Bq/filter	–	ND	17	Thallium-208
			1	4	Bq/m^3	–	ND	9.4	Uranium-234
			1	1	Bq/filter	–	–	0.002	Uranium-234

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Silica									
NIOSH (1992), USA	Wildland	Shift	1	10	mg/m^3		0.04	0.35	Mop-up
Styrene									
Hill *et al*. (1972), U.K.	Training	Grab sample	–	1	ppm	0.535			Pool fire
Andreae & Merlet (2001), Germany	Wildland emissions	Multiple	Multiple data sources	–	mg/kg[a]	[102 ± 96]			Means of reported mean emissions factors for savanna and grassland, tropical forest, extratropical forest, biofuel burning, charcoal making, and agricultural fires
Austin *et al*. (2001b), Canada	Municipal	15 min	9	9	ppm	0.5 ± 0.68	0.003	2.01	7 mixed occupancy buildings, one electronics industry, one 9-day smouldering fire
Austin *et al*. (2001c), Canada	Municipal (simulated)	Grab sample	15	60	ppm	detected		0.4	In separate burns: wood (spruce), bed mattress, sofa foam, cardboard, plywood, gasoline, varsol, white foam insulation

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Sulfuric acid									
Turkington (1984), 1984	Municipal	10–15 min	1	1	mg/m^3	28.5			–
Jankovic *et al*. (1991), USA	Municipal		22 22		mg/m^3 mg/m^3		ND ND	8.5 0.9	Knockdown Overhaul
Kinnes & Hine (1998), USA	Municipal	TWA	5	8	mg/m^3		0.08–0.27	0.29	Arson investigation
Burgess *et al*. (2001), USA	Municipal	Overhaul > 25 min Overhaul > 25 min	7 9	23 19	mg/m^3 mg/m^3	4.9 ± 8.5 13.6 ± 14.6			No respiratory protection SCBA used
Tetrachloroethylene (perchloroethylene)									
Brandt-Rauf *et al*. (1988), USA	Municipal	30 min	2	3	ppm	0.092 ± 0.04	0.064	0.138	Mostly wood structures burning (building and contents)
Trichloroethylene									
Brandt-Rauf *et al*. (1988), USA	Municipal	30 min	2	2	ppm	0.15	0.112	0.181	Mostly wood structures burning (building and contents)

Table 1.2 (contd)

Reference, location	Type of fire	Sampling period and duration	No. of fires	No. of samples	Units	Mean ± SD (*geometric mean, **median)	Min.	Max.	Comments
Trichloromethane (chloroform)									
Lowry *et al*. (1985a), USA	Municipal	At fire	75	–	–	detected			Mixed types of exposure
Brandt-Rauf *et al*. (1988), USA	Municipal	30 min	2	2	ppm	1.44	0.96	1.92	Mostly wood structures burning (building and contents)
Austin *et al*. (2001b), Canada	Municipal	15 min	9	9	ppm	detected			7 mixed occupancy buildings, one electronics industry, one 9-day smouldering fire
Austin *et al*. (2001c), Canada	Municipal (simulated)	Grab sample	15	60	ppm		25	465	In separate burns: wood (spruce), bed mattress, sofa foam, cardboard, plywood, gasoline, varsol, white foam insulation
Trichlorophenol									
Brandt-Rauf *et al*. (1988), USA	Municipal	30 min	1	1	ppm	0.1	0.1	0.1	Mostly wood structures burning (building and contents)

[a] emission factors
ESR, electron spin resonnance; LOD, limit of detection; LOQ, limit of quantitation; ND, Not Detected; SCBA, self-contained breathing apparatus

Thus, firefighters may be exposed to benzene levels exceeding the American Conference of Governmental Industrial Hygienists (ACGIH) 15-minute time-weighted average (15-min TWA) short-term exposure limit (STEL) of 2.5 ppm.

Two studies assessed 1,3-butadiene in smoke at structural and experimental fires (Austin *et al*., 2001b,c). Levels as high as 4.84 ppm were found at moments when at least some firefighters might remove their masks.

Formaldehyde levels measured in smoke at fires ranged from not detected to 15 ppm across studies (see Table 1.2).

One study measured pentachlorophenol at fires in simulated apartments with and without polyvinyl chloride (PVC) (Ruokojärvi *et al*., 2000). Levels of pentachlorophenol ranged from 14–160 μg/m^3 in those without PVC, and from 160–300 μg/m^3 in those with PVC.

Measurement of single specific PAHs at fires ranged from not detected to a maximum of 0.068 mg/m^3 for dibenzo[*a*,*h*]anthracene. In two studies, one of apartment fire simulations and one of training fires, measured total PAHs concentrations ranged from 6.4–470 mg/m^3 (Feunekes *et al*., 1997; Ruokojärvi *et al*., 2000).

Firefighter exposures to respirable particulate matter during overhaul rise to approximately 25 mg/m^3; levels of coarser particles range up to 20 000 mg/m^3 or higher (see Table 1.2). [In the case of wildland firefighters, reported results probably underestimated the actual exposures as these would have been collected during periods of low smoke levels.]

Exposures to VOCs are generally in the low ppm range for all categories of firefighters. [The results probably underestimated the exposures because they did not include the fraction adsorbed onto respirable smoke particles.] Austin *et al*. (2001b,c) found that although levels of total VOCs increased with time in fires burning solids, they decreased in time for fires burning liquids even though the levels of particulate matter increased. This suggests that a significant fraction of VOCs is adsorbed by the particulate matter and escapes detection when only the vapour phase is measured.

Overall, exposures of wildland firefighters to "low" levels of smoke appear to be comparable to those experienced by municipal firefighters during overhaul.

1.3.5 *Exposures to other agents*

(a) Asbestos

Asbestos used in constructions will be released during a fire in the form of fibres; asbestos sheets crack, sometimes disintegrating explosively, and more likely so if the sheet is worn or impregnated with resin (Hoskins & Brown, 1994). Chrysotile breaks down at 450–800 °C, and the amphiboles at 400–600 °C (Hoskins & Brown, 1994; Jeyaratnam & West, 1994). Thus, the denaturing of asbestos during fires may reduce exposure to asbestos fibres.

Table 1.3. Summary of reported concentrations of chemicals during firefighting operations (ranges or means)

Chemical	Units	Wildland	Municipal	Training	Arson investigation
Acetaldehyde	ppm	ND–0.26	ND–8.1	–	0.13[b]
Asbestos	f/cm^2		2.7[a]	0–2.3[d]	
Arsenic	mg/m^3	–	0.14[a]	–	–
Benzene	ppm	0.004[a]–0.38	0.07–250	1.17[a]	< 0.12[b]
Benzofuran	ppm	–	0.2–2	–	–
1,3-Butadiene	ppm	–	0.03–4.84	–	–
Cadmium		–	ND	–	–
Polychlorinated dibenzodioxins	ng/m^3	–	12–148	–	–
I-TEQs	ng/m^3	–	1–7.2	–	–
Dichloromethane	ppm	–	0.28[a]	–	–
Ethyl benzene	ppm	–	0.01–5.97	0.38[a]	–
Formaldehyde	ppm	0.01–0.79	ND–15	–	0.06–0.18
Free radicals					
Furan	ppm	–	0.2–2	–	–
Isoprene	ppm	–	–	0.167[a]	–
Lead	mg/m^3	–	0.03[a]	–	–
Naphthalene	ppm	–	0.01–2.14	0.418[a]	30–200mg/m^3
PM_{10}	mg/m^3	3.0[b]	–	–	–
PM respirable	mg/m^3	0.02[c]–10.5	ND–25.7	–	ND–1.2
PM total Knockdown only Overhaul only	mg/m^3	0.2[a]–44.9	ND–650 ND–20 000 ND–45	0.1–300	0.2–31.6
Pentachlorophenol	µg/m^3	–	14–300	–	–
Polycyclic aromatic hydrocarbons	mg/m^3	–	6.4–470	10.68[a]	–
Polychlorinated biphenyls	µg/m^3	–	2.8–56	–	–
Silica	mg/m^3		0.04–0.35		
Styrene		–	0.003–2.01	0.535[a]	–
Sulfuric acid		–	ND–28.5	–	0.29[b]
Tetrachloroethylene		–	0.064–0.138	–	–
Trichloroethylene		–	0.112–0.181	–	–
Trichloromethane		–	0.96–465	–	–
Trichlorophenol		–	0.1[a]	–	–

[a] mean; [b] maximum; [c] geometric mean; [d] from helmets and fumes of firefighters; ND, not detected

During a leather factory fire in Merseyside, United Kingdom, in 1994, most of the fallout arose from asbestos bitumen roof paper containing roughly 50% chrysotile (Bridgman, 2001). A low number of asbestos fibres were found on firefighter tunics (0.0029 f/cm^3; range 0.0011–0.0043 f/cm^3), and none was found on the firefighters' raincoats or policemen's uniforms. [A fire hose spray may have washed out airborne asbestos.]

Thermal protective clothing, gloves and helmets that contain asbestos usually contain chrysotile asbestos. In the United Kingdom the helmet covers for navy firefighters, which completely enclose their head and shoulders, used to be made of chrysotile asbestos (Lumley, 1971). Breathing zone samples from users of both new and old helmets with unlined asbestos cloth covers were analysed and had fibre concentrations of 2.30 f/cm^3 and 1.38 f/cm^3, respectively (Lumley, 1971).

(b) Polychlorinated biphenyls, polychlorinated dibenzofurans and polychlorinated dibenzodioxins

Synthetic dielectric (non-conducting) fluids are known as askarels. Firefighters may be exposed to PCBs at fires involving PCB-askarel filled transformers and capacitors (Hutzinger *et al*., 1985). When askarels burn, copious quantities of oily black soot are produced with very little fire. Where only PCBs are involved, polychlorinated dibenzofurans (PCDFs) are produced as combustion products. In transformers containing a mixture of PCB-askarel and polychlorobenzenes (PCBz), in addition to PCDFs, polychlorinated dibenzodioxins (PCDDs) combustion products are produced from the PCBz (Buser, 1985). PCDFs and PCDDs might also arise from *de novo* synthesis under certain conditions. PCDFs and PCDDs were reported to have been released from a house fire where a 50 lb [23 kg] container of hypochlorite and two gallons [7.6 L] of hydrochloric acid were stored for swimming pool maintenance along with paint thinners and solvents (Rao & Brown, 1990). Other sources of PCBs at fires may include fluorescent light ballasts, PCB-containing mastic, adhesives, duct liners, and fibreglass insulation wrap used in previous decades (Kominsky, 2000). Total 2,3,7,8-tetrachlorodibenzodioxin (TCDD) equivalent (TEQ) levels were 0.24 ppb in a basement soot sample and 0.39–0.75 ng/m^2 in two wipe samples.

PCB concentrations in wipe samples following a fire in a high rise office building were reported to be 7.1–151 µg/m^2 (range, < 1–87610 µg/m^2) (Kominsky, 2000). Debris from a chemical storage vault fire contained 100–750 ppm PCBs, and 2000 ppm PCBs was found in the lubricating grease from the air-handling units (CDC-MMWR, 1987). No PCBs were stored in the storage vault. The source of the PCBs was the paint that coated the surface of the ceiling tiles (15 300–51 000 ppm PCBs).

The use of PCBs in electrical equipment and its effect on some of the numerous fire-related incidents that have occurred in the USA and in Sweden have been reviewed by NIOSH (1986) and Rappe *et al*. (1985a), respectively. A PCB/PCBz-filled transformer fire occurred at the Binghamton State Office Building, New York, USA, in 1981 (O'Keefe *et al*., 1985). Levels of 2,3,7,8-TCDF and 2,3,7,8-TCDD in a

soot sample collected following the fire were 12 ppm and 0.6 ppm, respectively (Buser, 1985). The analysis of soot collected following a capacitor fire at a power station in Finland revealed 3 ppm of 2,3,7,8-TCDF (Buser, 1985). In an earlier study, the replicate concentrations of 2,3,7,8-TCDF and 2,3,7,8-TCDD in a composite soot were 273 and 124 ppm, and 2.8 and 2.9 ppm, respectively (Smith *et al*., 1982).

Firefighter thermal protective clothing can be contaminated with PCBs following fires involving PCBs. In one report, tests revealed 2.7–72 μg PCB/g of clothing following a fire (Kominsky & Melius, 1983). Following the Staten Island fire in the USA, gloves, outer coat sleeves, and outer pants contained peak PCB concentrations of 4 050 000 pg/100 cm^2, 56100 pg/100 cm^2, and 116 000 pg/100 cm^2, respectively (Kelly *et al*., 2002). Overalls and underwear used following the Surahammar fire in Sweden were washed every day (Rappe *et al*., 1985b). After 14 days of use, overalls were analysed and contained 28 ng/m^2 of 2,3,7,8-TCDF, and approximately 100 ng/m^2 all TCDFs combined. Similar levels were found after 1 month of use.

(c) Diesel and gasoline exhaust

Firefighters may be exposed to diesel/gasoline exhaust when vehicles exit and return to the firehall. In a study of diesel emissions in firehalls, shift mean personal measurements of total particulate matter were 0.035–0.48 mg/m^3 (worst-case scenario 0.748 mg/m^3) (Froines *et al*. 1987). [The sampling equipment was removed during the period of highest concentration of diesel exhaust, thereby underestimating actual exposures]. Background ambient particulate matter from ambient aerosol, smoking, and cooking was approximately 0.040 mg/m^3.

Mechanical systems have been available to fire departments since the early 1990s to divert the engine exhaust to the outside of the building (Peters, 1992).

Firefighters are also exposed to diesel emissions from response vehicles that remain running at the fire scene. Firefighters may be positioned near these vehicles during command operations, operation of pumps, when working in defence mode, and during rest breaks.

Firefighters may be exposed to diesel/gasoline exhaust during the operation of vehicles and gasoline-powered hand tools at both structural and wildland fires. No particular methods are used to reduce these exposures. In addition, wildland firefighters are exposed to vapours and combustion products from drip torches used when setting back fires.

(d) Shiftwork

Depending on the jurisdiction, firefighters may work 10-hour dayshifts and 14-hour night shifts, 24-hour or 48-hour shifts. However, given the low frequency of fires over a year, at least in North America, firefighters are often able to sleep at the firehall during the entire night.

(e) Others

Firefighters may be exposed to agents stored, manufactured, or otherwise present at the scene of fire, particularly in factories. Examples are exposure to 2-nitroanisole (Hengstler *et al*., 1995), and to toluene diisocyanate (Axford *et al*., 1976) (see Table 1.1).

In many cases, firefighters hold down a second job where they may also be exposed to other agents.

1.4 Biomarkers of exposure

New York City firefighters responded to a Staten Island transformer fire in 1998 in the USA. Exposed firefighters exhibited mean fasting blood serum PCB levels of 2.92 ± 1.96 ppb (range 1.9–11.0 ppb; $n = 58$) 2–3 weeks following exposure (Kelly *et al*., 2002). Mean levels of serum 2,3,7,8-TCDF, 2,3,7,8-TCDD, and TEQ were 0.20 pg/g (SD 0.69; range ND–2.78; $n = 60$), 3.77 (SD 4.16; range ND–13.4; $n = 60$), and 39.0 pg/g (SD 21.53; range 8.77–120.63; $n = 60$).

In a study of firefighters in Toronto, Canada, firefighters were exposed to low levels of smoke. Self-contained breathing apparatus was consistently used during knockdown, less consistently during overhaul, and intermittently during external firefighting activities. All urine produced during the 20 hours following the end of exposure was collected. Only two of 43 subjects were smokers. Ranges of urinary *trans*,*trans*-muconic acid levels in firefighters who were present at fires during knockdown only, during overhaul only, and during both knockdown and overhaul ranged from not detected to 2.82 mmol/mol creatinine ($n = 5$), to 1.12 mmol/mol creatinine ($n = 8$), and to 1.06 mmol/mol creatinine ($n = 24$), respectively (Caux *et al*., 2002). The only two firefighters who wore their masks at all times had no measurable urinary *trans*,*trans*-muconic. Levels of urinary 1-hydroxypyrene were 0.12 μmol/mol creatinine (range 0.05–0.19; $n = 5$), 0.23 μmol/mol creatinine (range 0.11–0.34; $n = 8$), and 0.38 and μmol/mol creatinine (range 0.08–3.63; $n = 24$), for the three groups respectively. There was no relationship between measured levels of urinary *trans*,*trans*-muconic acid and 1-hydroxypyrene.

One study was conducted following the September 11th, 2001, attack of the World Trade Center in New York City, USA (Edelman *et al*., 2003). Blood and urine samples were collected from firefighters 20 days following the attack. Table 1.4 presents adjusted geometric means of concentrations of the chemicals detected in blood and/or urine. The maximum levels of blood mercury found in firefighters following the attack were < 1.7 μg/L blood. Elevated total mercury levels > 20 μg/L blood in one control and 3 exposed firefighters represented organic mercury contributions from dietary sources (e.g. fish).

Table 1.4. Blood and urinary levels in firefighters 20 days following the World Trade Center attack in 2001

Agent	Matrix	Unit	Controls (n = 318)	Special command (n = 95)	Other (n = 195)
1,4-Dichlorobenzene	Blood	µg/L	0.165	0.343*	0.231
m-/*p*-Xylene	Blood	µg/L	0.051	0.081*	0.057
Cadmium	Urine	µg/L	0.377	0.351	0.303
Lead	Blood	µg/L	1.93	3.77*	2.43*
	Urine	µg/L	1.01	1.77*	0.96
Uranium	Urine	µg/L	0.00752	0.00610	0.00607
HCDBD	Lipid	pg/g	19.2	30.6*	25.9*
1-Hydroxypyrene	Urine	ng/L	62.5	159*	77.9

*Significantly elevated compared to the controls, $P < 0.01$
HCDBD, heptachlorodibenzodioxin

1.5 Respiratory protection

1.5.1 *Evolution of respiratory protection and protection factors*

In prior decades, firefighters were known as "smoke eaters." However, respiratory protection devices for firefighters have existed for over a century. Early US patented devices included air purifying respirators using charcoal to remove toxic gases and vapours (e.g. Guillemard, 1920), and carbon monoxide (e.g. Loeb, 1893), and self-contained breathing apparatus that supplied air to the user (e.g. Hurd, 1889). Air purifying respirators and self-contained breathing apparatus in use today have improved designs, but the basic principles are the same. Modern positive-pressure type self-contained breathing apparatus with a protection factor of 50 to more than 100 came into more widespread use during the 1960s and 1970s (Hyatt, 1976). These were replaced shortly thereafter with pressure-demand type self-contained breathing apparatus with a protection factor of 10 000 (Hyatt, 1976). Pressure-demand self-contained breathing apparatus are commonly used today by municipal firefighters.

1.5.2 *Efficacy of respiratory protection*

Pressure-demand self-contained breathing apparatus have been determined to be adequate in a firefighter risk assessment given the levels of fire atmosphere contaminants reported in the literature (Burgess & Crutchfield, 1995a,b). In these studies, 50 of

51 firefighters (98%) achieved a protection factor exceeding 10 000; estimates of worst case scenarios yielded a protection factor of 4600.

In 1978, firefighters in Scotland, United Kingdom, reportedly used self-contained breathing apparatus routinely at residential fires (Symington *et al*., 1978). There were no significant differences found in cyanide or thiocyanate levels between these firefighters ($n = 94$) and controls, suggesting that the breathing apparatus used offered adequate protection against hydrogen cyanide.

There is currently no respiratory protection standard for wildland firefighters. One bottle of compressed air used with a self-contained breathing apparatus lasts approximately 15–30 minutes, so self-contained breathing apparatus are not an option for wildland firefighters who work extended shifts at fires for consecutive days or weeks. The only other options are administrative controls to reduce exposure, or the use of air purifying respirators. Air purifying respirators have recently been evaluated for use by firefighters (De Vos *et al*., 2006; Anthony *et al*., 2007). In some jurisdictions, such as Australia, wildland firefighters use negative-pressure air purifying respirators (De Vos *et al*., 2006). In many others, such as in Canada and the USA, wildland firefighters generally do not use any form of respiratory protection (Austin & Goyer, 2007).

1.5.3 *Prevalence of use of self-contained breathing apparatus*

Firefighters tend to use their masks "when they see smoke." In the past, there was some avoidance of the use of respiratory protection (Guidotti, 1992); over the years, firefighters have become much more health and safety conscious.

There are several other reasons why firefighters might be reluctant to use respiratory protection. These include the added physiological demands and heat stress placed upon the user, the difficulty in communicating while wearing a mask, and the desire to conserve air. However, several studies have demonstrated that firefighters are not able to visually assess the level of contamination. A study in Boston, USA, found no clear patterns or trends that would allow firefighters to predict the levels of smoke contaminants to which they were exposed (Burgess *et al*., 1979). In one study, a firefighter who was working without a respirator because he believed that his exposure was insignificant was actually exposed to 27 000 ppm of carbon monoxide (Burgess *et al*., 1977), 680 times the current ACGIH Treshold Limit Value (TLV, 25 ppm). Results of other studies also suggest that structural firefighters cannot estimate levels of smoke contaminants (Brandt-Rauf *et al*., 1988, 1989).

A "Mandatory Mask Rule" was fully implemented in 1977 at the Boston fire department requiring all firefighters to wear respiratory protective equipment before entering a building for firefighting operations. The mask is not to be removed until after knockdown and after the building has been thoroughly ventilated (Paul, 1977).

Since the introduction of modern self-contained breathing apparatus in the fire service, the lack of standard operating procedures (SOPs) mandating the use of respiratory protection equipment or the failure to enforce existing SOPs have resulted

in them not being used appropriately. Even where masks are consistently used during knockdown, they are usually not used or used inconsistently during overhaul.

In a study in West-Haven, CT, USA, half of the firefighters (eight of 16) involved in structural fires did not use breathing apparatus (Loke *et al*., 1976). In Washington DC, USA, the frequency of wearing masks during knockdown was: always (36%); very often (36%); never or seldom (5%). During overhaul, 62% never or seldom wore masks (Liou *et al*., 1989). A NIOSH study of different fire departments in the USA (Pennsylvania Fire Training Academy, Pittsburgh Bureau of Fire, New York City, Phoenix, Boston, and Cincinnati) found that 70% of municipal firefighters wore their self-contained breathing apparatus masks less than 100% of the time, and one third used them less than 50% of the time during knockdown (Jankovic *et al*., 1991). In Sweden, only four of nine volunteer firefighters surveyed reported having used protective equipment while fighting fires within the previous 3 months (Bergström *et al*., 1997). In a 1993–1994 study of Montreal firefighters, the storage and distribution of all compressed breathing air was tracked and records kept of the time and place of all cylinders, including initial and final pressures along with records of firefighter assignments and alarms. The authors concluded that respiratory protection was used for approximately 50% of the time at structural fires, but for only 6% of the time at all types of fires combined (Austin *et al*., 2001a). In Toronto, Canada, firefighters "reported consistent usage of self-contained breathing apparatus during knockdown activities inside structures, less consistent usage throughout internal overhaul activities, and intermittent usage during external fire fighting activities" (Caux *et al*., 2002). In a study in Phoenix and Tucson, AZ, respiratory protection was used during entry/ventilation for 86–95.4% and 74–91.7% of the time, respectively. During overhaul, Phoenix and Tucson firefighters used respiratory protection for 38% and 46.2% of the time, respectively (Burgess *et al*., 2003).

1.6 Regulations and guidelines

Table 1.5 presents occupational exposure limits for selected chemicals to which firefighters are exposed. Occupational exposure limits have been developed for workers generally exposed to single substances and engaged in light levels of work. Firefighters are exposed to a complex mixture of toxic combustion and pyrolysis products while engaged in very high workloads. Also, given the intermittent nature of the exposures, determination of TWAs will result in calculated exposures far below the established TLVs for different substances. In addition, this does not take into account peak exposures and possible synergistic effects of multiple, potential toxicants. Biomonitoring overcomes some of these difficulties and takes into account the use of respiratory protection.

Table 1.5. Regulations and guidelines for the chemicals measured in smoke at fires presented in Table 1.2 (ACGIH, 2007)

Chemicals measured at fires	Units	BEI		TLV/TWA		STEL		Ceiling	Permitted excursion	Maximum excursion
		ACGIH	EU	ACGIH	EU	ACGIH	EU	ACGIH	ACGIH	ACGIH
Arsenic	mg/m^3	Yes		0.01		–		–	0.03	0.05
Asbestos	f/cm^3	–		0.1	0.1	–		–	0.3	0.5
Acetaldehyde	ppm	–		–		–		25	–	25
Benz[*a*]anthracene	mg/m^3			–		–		–		
Benzene	ppm	Yes	Yes	0.5	1	2.5		–	–	–
Benzo[*a*]pyrene	mg/m^3									
1,3-Butadiene	ppm	Yes		2		–		–	6	10
Cadmium	mg/m^3	Yes		0.01		–		–	0.03	0.05
Carbon black (total)	mg/m^3	–	Under discussion	3.5	Under discussion	–	Under discussion	–	10.5	17.5
Dichloromethane (methylene chloride)	ppm	Yes		50	Under discussion	–		–	150	250
Ethylbenzene	ppm	Yes		100	100	125	200	–	–	–
Formaldehyde	ppm	–			Under discussion	–		0.3	–	–
Furan/tetrahydrofuran	ppm	–		–	50	–	100	–	–	–
Isoprene	ppm	–		–		–		–	–	–
Lead	mg/m^3	Yes		0.15	0.15	–		–	0.45	0.75
Naphthalene	ppm	–		10	10	15		–	–	–
Particulate matter (respirable)	mg/m^3	–		3		–		–	9	15
Particulate matter (total)	mg/m^3	–		10		–		–	30	50
Pentachlorophenol	$\mu g/m^3$	Yes		0.5						
Polychlorinated biphenyls (Aroclor; 54%) (Chlorodiphenyl)	$\mu g/m^3$	–		0.5		–		–	1.5	2.5
Polycyclic Aromatic Hydrocarbons	mg/m^3	Yes		0.2		–		–	0.6	1.0

Table 1.5 (contd)

Chemicals measured at fires	Units	BEI		TLV/TWA		STEL		Ceiling	Permitted excursion	Maximum excursion
		ACGIH	EU	ACGIH	EU	ACGIH	EU	ACGIH	ACGIH	ACGIH
Styrene	ppm	Yes		20		40		–	–	–
Sulfuric acid	mg/m^3	–		0.2	Under discussion	–		–	–	–
Tetrachloroethylene (Perchloroethylene)	ppm	Yes		25	Under discussion	100.0			–	–
Trichloroethylene	ppm	Yes		10	Under discussion	25.0		–	–	–
Trichloromethane (chloroform)	ppm	–		10	2	–		–	30	50
Trichlorophenol	ppm	–		–		–		–	–	–

ACGIH, American Conference of Governmental Industrial Hygienists; BEI, Biological Exposure Index; EU, European Union; STEL, short-term exposure limit; TLV, Threshold Limit Value.

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2. Studies of Cancer in Humans

2.1 Cohort studies (Tables 2.1 and 2.2)

Among municipal firefighters, five studies evaluated the incidence of cancer and 15 assessed mortality – some with multiple reports. None of these was adjusted for smoking, or other potential confounders.

Mastromatteo (1959) conducted a cohort mortality study of all firefighters employed with the city fire department in Toronto, Ontario, Canada during 1921–1953. The cohort consisted of 1039 active and retired firefighters. A total of 325 firefighters (31%) were lost to follow-up. The followed cohort accrued 25 918 person–years of observation; 271 deaths were recorded. Causes of death were determined by the examination of death certificates. Comparison was made to age-specific death rates for the Province of Ontario. Because most of the firefighters were urban dwellers, the author calculated death rates for the City of Toronto. There was no excess mortality from cancer at all sites combined (34 observed, 30 expected), and no site-specific analysis of cancer mortality.

Musk *et al.* (1978) conducted a cohort mortality study in 5655 firefighters with three or more years of service in Boston, USA, during 1915–1975. Firefighters were identified from employment records. The observed cause of death as stated on the death certificates of 2470 deceased firefighters was compared with expected numbers based on rates for the male population of Massachusetts. Cancer risks were only presented by organ system, and no statistically significant increases were seen.

Eliopulos *et al.* (1984) conducted a cohort mortality study of all males employed as full-time firefighters by the Western Australian Fire Brigade during 1939–1978. The cohort consisted of 990 male firefighters and follow-up was 98.3% complete through examination of death certificates. The cohort accrued a total of 16 876 person–years of follow-up; 116 deaths were recorded. Mortality from all causes was less than expected (standardized mortality ratio [SMR], 0.80; 95% confidence interval [CI]: 0.67–0.96). For those firefighters already employed at the study start date, mortality was a little higher (SMR, 0.84) than for those who started later (SMR, 0.74). There was no tendency for rates to rise with increasing duration of employment. SMR for all malignancies was 1.09 (95% CI: 0.74–1.56). Standardized proportional mortality ratios (SPMRs) were calculated for cancers, primarily by organ system, and no statistically significant excesses were seen.

Vena and Fiedler (1987) examined all full-time employees of the City of Buffalo, USA, who worked at least 5 years during 1950–1979. A total of 1867 Caucasian male firefighters employed for at least one year as a firefighter were studied.

Table 2.1 Overview of cohort studies of firefighters

Reference	Location	Outcome, Design	Study Period	Number of Workers	Exposure Surrogates Used
Mastromatteo (1959)	Toronto, Canada	Mortality (SMR)	1921–1953	1039	None
Musk *et al.* (1978)	Boston, USA	Mortality (SMR)	1915–1975	5655	None
Eliopulos *et al.*, (1984)	Australia	Mortality (SMR, SPMR)	1939–1978	990	None
Vena & Fiedler (1987)	Buffalo, USA	Mortality (SMR)	1950–1979	1867	Duration of employment
Heyer *et al.* (1990)	Portland, USA Seattle, USA	Incidence (SIR) Mortality (SMR)	1974–1989 1945–1980	2447 2289	Duration of firefighting Duration of employment
Beaumont *et al.* (1991)	San Francisco, USA	Mortality (SMR)	1940–1970	3066	Duration of employment
Grimes *et al.* (1991)	Honolulu, USA	Proportionate Mortality	1969–1988	205	None
Demers *et al.* (1992a; 1992b; 1994)	Seattle, Tacoma, USA	Mortality (SMR) 1992a Mortality (SMR) 1992b Incidence (SIR) 1994	1944–1979 1944–1979 1974–1989	4546 4528 2447	
Giles *et al.* (1993)	Western Australia	Incidence (SIR)	1980–1989	2865	None
Guidotti (1993)	Calgary, Edmonton, Canada	Mortality (SMR)	1927–1987	3328	Duration of firefighting
Aronson *et al.* (1994)	Toronto, Canada	Mortality (SMR)	1950–1989	5995	Duration of employment
Tornling *et al.* (1994)	Stockholm, Sweden	Mortality (SMR) Incidence (SIR)	1931–1983	1153	Duration of employment Number of runs
Deschamps *et al.* (1995)	Paris, France	Mortality (SMR)	1977–1991	830	None
Baris *et al.* (2001)	Philadelphia, USA	Mortality (SMR)	1925–1986	7789	Duration of employment Number of runs Company type engine, ladder

Table 2.1 Overview of cohort studies of firefighters

Reference	Location	Outcome, Design	Study Period	Number of Workers	Exposure Surrogates Used
Bates *et al.* (2001)	New Zealand	Mortality (SMR) Incidence (SIR)	1977–1995	4221	Duration of employment
Ma *et al.* (2005)	Florida, USA	Mortality (SMR) Incidence (SIR)	1972–1999 1981–1999	36 813	None
Ma *et al.* (2006)	Florida, USA	Mortality (SMR) Incidence (SIR)	1972–1999 1981–1999	222 4528	None

Adapted from LeMasters *et al*. (2006)

Vital status was determined for 99% of the cohort, resulting in 470 observed deaths. Significantly elevated SMRs were found for benign neoplasms (SMR, 417), cancer of the colon (SMR, 183), and cancer of the bladder (SMR, 286). Cause-specific mortality was presented by the number of years employed, calendar year of death, year of hire, and latency. Cancer mortality was significantly higher in the long-term firefighters, and risk of mortality from all malignant neoplasms tended to increase with increasing latency. Statistically significant excesses of colon and bladder cancer were observed among firefighters employed for 40 or more years.

Beaumont *et al.* (1991) calculated mortality rates for 3066 firefighters employed during 1940–1970 at the San Francisco Fire Department, USA. Vital status was ascertained through to 1982, and observed and expected rates were computed using United States death rates. About 3% of the population was lost to follow-up. Mortality was examined by duration of employment as a firefighter. The total number deceased (1186) was less than expected (risk ratio [RR] = 0.90), and there were fewer cancer deaths than expected (RR = 0.95). However, there were significant excess numbers of deaths from oesophageal cancer (12 observed, six expected). A statistically significant excess of biliary and related cancer was observed among firefighters employed for 30 or more years.

Grimes *et al.* (1991) conducted a proportionate mortality study involving all male firefighters with at least one year of service in the fire department of the City of Honolulu, USA. The observed percentage of firefighter deaths from each cause from 1969–1988 was compared statistically to the expected numbers of deaths for all males aged over 20 years in Hawaii's general population. The proportionate risk ratio (PRR) for all malignant neoplasms was 1.19 (95% CI: 0.96–1.49). Significant increases in risk of death were found for brain cancer (PRR, 3.78), prostate cancer (RR, 2.61), and cirrhosis of the liver (PRR, 2.3). [The Working Group noted that it does not appear as though PRRs were standardized by age and calendar period as is standard practice for this type of analysis.]

Heyer *et al.* (1990) examined the mortality among 2289 firefighters from Seattle, Washington, USA employed during 1945–1980. Subsequently, Demers *et al.* (1992a) examined the mortality of 4546 firefighters who were employed by the cities of Seattle and Tacoma (Washington, USA), and Portland (Oregon, USA) for at least one year during 1944–1979. Demers *et al.* (1992b) also examined the cancer incidence in 4528 firefighters from Seattle and Tacoma during 1944–1979. Mortality in these firefighters was compared to United States national mortality rates and to mortality rates of police officers from the same cities. Mortality was examined by the duration of employment as a firefighter (i.e., actually controlling fires) rather than as an inspector or a support person. This mortality was then compared to a reference group of police from the same cities. Complete follow-up was achieved for 98% of the firefighters. During 1945–1989 (the cohort was the same as Demers *et al.* [1992a] but the follow-up lasted until 1989), 1169 deaths occurred in the study population, and 1162 death certificates (99%) were collected. There was no excess risk of overall

mortality from cancer. Excesses of brain tumours (SMR, 2.1; 95% CI: 1.2–3.3) and lymphatic and haematopoietic cancers (SMR, 1.3; 95% CI: 0.9–1.8) were found. Younger firefighters (< 40 years of age) showed an excess risk of cancer (SMR, 1.45; 95% CI: 0.8–2.39), primarily due to brain cancer (SMR, 3.75; 95% CI: 1.2–8.7). The risk of lymphatic and haematopoietic cancers was greatest for men with at least 30 years of exposed employment (SMR, 2.1; 95% CI: 1.1–3.6), especially for leukaemia (SMR, 2.6; 95% CI: 1.0–5.4).

Demers *et al.* (1994) further examined the incidence of cancer in a subcohort of 2447 male firefighters who were employed for at least one year during 1945–1979 in Seattle and Tacoma, who were still alive on January 1st 1974. Incident cancer cases were ascertained through the Cancer Surveillance System of the Fred Hutchinson Cancer Research Center, a population-based tumour registry. The follow-up period was from 1974 to 1989. Cancer incidence in firefighters was compared with local rates and with the incidence among 1878 policemen from the same cities. The overall risk of cancer among firefighters was found to be similar to that of both the police (SIR, 1.0; 95% CI: 0.8–1.3) and the general male population (SIR, 1.1; 95% CI: 0.9–1.2). No excesses were observed for the most common organ sites. An elevated risk of prostate cancer was observed relative to the general population (SIR, 1.4; 95% CI: 1.1–1.7), but was less elevated compared with rates in policemen (incidence density ratio [IDR], 1.1; 95% CI: 0.7–1.8), and was not related to duration of exposure. The risk of colon cancer, although only slightly elevated relative to that of the general population (SIR, 1.1; 95% CI: 0.7–1.6) and the police (IDR, 1.3; 95% CI: 0.6–3.0), appeared to increase with duration of employment.

Giles *et al.* (1993) conducted a cancer incidence study of 2855 male firefighters employed by the fire brigade in Melbourne, Australia, during 1917–1988. All were operational personnel, who would more than likely have been called to combat fires. The follow-up period was from 1980 to 1989, and was estimated to have been 95% complete. To determine cancer incidence during the follow-up period, fire brigade employment records were linked to the Victorian Cancer Registry. SIRs were calculated by the direct method using the population of the State of Victoria as the reference group. The cohort accrued a total of 20 853 person–years, and 50 firefighters developed cancer during the period of observation. The SIR for all cancer sites and all ages combined was 1.13 (95% CI: 0.84–1.48). For firefighters under the age of 65 years, the all-site SIR was 0.84 (95% CI: 0.56–1.20); for those above 65 years of age, the all-site SIR was 2.14 (95% CI: 1.32–2.37). The only site-specific cancer that was elevated in the age group of 65 and older was colorectal cancer, with an SIR of 3.65 (95% CI: 1.13–7.94). The SIR for all other cancers in the age group 65 and above after removing colorectal cancer remained elevated, with a residual SIR of 1.83 (95% CI: 1.03–3.02).

Guidotti (1993) examined the mortality by cause of death for two cohorts totaling 3328 firefighters active during 1927–1987 in Edmonton and Calgary, Alberta, Canada. Associations were examined by cohort (before and after the 1950s) and by

years of service weighted by exposure opportunity. The study attained 96% follow-up of vital status and over 64 983 person–years of observation; 370 deaths were recorded. Excesses were observed for all malignant neoplasms (SMR, 1.3; 95% CI: 1.0–1.6), and for cancers of the lung (SMR, 1.4; 95% CI: 0.9–2.1), bladder (SMR, 3.2; 95% CI: 0.9–8.1), kidney and ureter (SMR, 4.1; 95% CI: 1.7–8.5), colon and rectum (SMR, 1.6; 95% CI: 0.9–2.7), pancreas (SMR, 1.6; 95% CI: 0.5–3.6), and leukaemia, lymphoma and myeloma (SMR, 1.3; 95% CI: 0.6–2.3). The lung cancer excess was confined to Edmonton; there was no consistent association with duration of employment, exposure opportunity, or decade of entry into the cohort (before or after the 1950s) except that the highest risk was observed among Edmonton firefighters with over 35 weighted years of service. Urinary tract cancer excess was observed mostly among firefighters entering service after 1950, and appeared to increase with the length of service and exposure opportunity, and was observed in both cities.

Aronson *et al.* (1994) conducted a retrospective cohort mortality study of all male employees of the six fire departments within metropolitan Toronto, Ontario, Canada ($n = 5995$). The study population consisted of all male firefighters who had worked for at least 6 full months in metropolitan Toronto at any time during 1950–1989. Mortality was ascertained through computerized record linkage and compared to that of the male Ontario population specific to cause, age, and calendar period during 1950–1989. The cohort accrued 114 008 person–years and the average duration of follow-up was 21 years. Mortality was examined by duration of exposure. The SMR for all malignant neoplasms was 105 (95% CI: 91–120), for brain tumours, 201 (95% CI: 110–337), and for "other" malignant neoplasms, 238 (95% CI: 145–367). Non-significant increases in risk were observed for some other sites, in particular rectum (SMR, 171), larynx (SMR, 140), and testis (SMR, 252).

Tornling *et al.* (1994) conducted a cohort mortality study of all male fire fighters employed for at least 1 year in the City of Stockholm, Sweden during 1931–1983 ($n = 1116$). The population was identified from annual employment records. Follow-up for mortality was from 1951 until 1986, and for cancer incidence from 1958 to 1986. Except for four persons who had emigrated from Sweden, follow-up was 100% complete. To assess the occupational exposure as a firefighter, an index of participation in number of fires was calculated for each individual based on the number of reports on all fires in Stockholm that had been maintained since the beginning of the twentieth century. The all-site cancer mortality in 1958–1986 was equal to the expected (SMR, 100; 95% CI: 83–119). An excess of stomach cancer incidence (SIR, 192; 95% CI: 114–304; 18 observed versus 9.37 expected) was observed. There was also a tendency for higher incidence and mortality in stomach and brain cancers with increasing number of fires. Four brain cancer cases were observed compared to 0.8 expected (SIR, 496; 95% CI: 135–1270) in the highest exposure category.

Table 2.2. Cohort studies of cancer among firefighters

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Buccal cavity							
Beaumont *et al.* (1991), California, USA	3066 male firefighters employed 1940–70 Buccal cavity and pharynx	Fire department records	Overall	11	1.4 (0.7–2.6)		
Baris *et al.* (2001), Pennsylvania, USA	7789 firefighters employed 1925–86 Buccal cavity and pharynx	Employee service records	Overall	19	1.4 (0.9–2.1)		
			Duration of employment		**SMR**		
			≤9 yrs	4	1.2 (0.4–3.1)		
			10–19 yrs	9	1.8 (0.95–3.5)		
			≥20 yrs	6	1.1 (0.5–2.4)		
			Hiring period				
			Hired before 1935	10	2.1 (1.1–3.9)		
			1935–44	4	0.9 (0.3–2.3)		
			After 1944	5	1.1 (0.5–2.6)		
			Number of runs				
			Low (<3323)	7	1.7 (0.8–3.6)		
			Medium (3323–5099)	0	0		
			High (5099+)	2	0.8 (0.2–3.1)		
Ma *et al.* (2005), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records			**SMR**	Age, calendar year	
			Men	7	0.4 (0.2–0.9)		
			Women	0	–		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Ma *et al.* (2006), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records	Men Women	39 0	**SIR** 0.7 (0.5–0.9) 0	Age, calendar year	
Oral and pharyngeal cavity / UADT							
Demers *et al.* (1992a), Northwest, USA	4546 male firefighters employed 1944–79 in selected Northwest cities	Employment records	Overall	7	**SMR** 0.8 (0.3–1.7)		
Demers *et al.* (1992b), Northwest, USA	4528 firefighters and police officers employed by the cities of Seattle and Tacoma; oral and pharyngeal	Employment records		19 4	**SIR** 1.2 (0.7–1.9) **SMR** 1.0 (0.3–2.6)		Data for firefighters and police combined
Giles *et al.* (1993), Australia	2865 male firefighters employed 1917–89 Upper aerodigestive	Employment and union records, payrolls	Overall	6	**SIR** 1.5 (0.5–3.2)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Guidotti (1993), Alberta, Canada	3328 firefighters employed 1927–87 Oral	Personnel files	Overall	2	**SMR** 1.1 (0.1–4.1)		
Aronson *et al.* (1994), Ontario, Canada	5373 male firefighters employed 1950–89 Pharynx	Employment records	Overall	4	**SMR** 1.4 (0.4–3.6)		
Demers *et al.* (1994), Northwest, USA	2447 male firefighters employed 1974–89	Employment records	Overall <10 yrs 10–19 yrs 20–29 yrs ≥30 yrs	11 2 4 2 3	**SIR** 1.1 (0.6–2.0) 1.4 (0.2–5.1) 2.5 (0.7–6.4) 0.3 (0.0–1.2) 3.9 (0.8–11.0)		
Deschamps *et al.* (1995), France	830 male firefighters employed 1977–91 Pharynx	Employment records		2	**SMR** 0.8 (0.1–2.9)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Stomach							
Eliopulos *et al.* (1984), Australia	990 firefighters employed 1939–78	Western Australian Fire Brigade records	Overall	5	**SPMR** 2.0 (0.7–4.7)		
Vena & Fiedler (1987), New York State, USA	1867 male firefighters employed 1950–79	Death certificates	Overall	7	**SMR** 1.2 (0.5–2.5)		
Heyer *et al.* (1990), Washington, USA	2289 male firefighters employed at least 1 yr, 1945–80; follow-up until 1983	Employment records	Overall	6	**SMR** 1.1 (0.4–2.5)		
Beaumont *et al.* (1991), California, USA	3066 male firefighters employed 1940–70	Fire department records	Overall	22	**SMR** 1.3 (0.8–2.0)		
Grimes *et al.* (1991), Hawaii, USA	205 male firefighters	Death certificates	Overall	2	**PRR** 0.8 (0.3–2.1)		Not clear if standardized by age and calendar period

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Demers *et al.* (1992a), Northwest, USA	4546 male firefighters employed 1944–79 in selected Northwest cities	Employment records	Overall	16	**SMR** 1.1 (0.6–1.7)		
Guidotti (1993), Alberta, Canada	3328 firefighters employed 1927–87	Personnel files	Overall	6	**SMR** 0.8 (0.3–1.8)		
Aronson *et al.* (1994), Ontario, Canada	5973 male firefighters employed 1950–89	Employment records	Overall	7	**SMR** 0.5 (0.2–1.1)		
Demers *et al.* (1994), Northwest, USA	2447 male firefighters employed 1974–89	Employment records	Overall <10 yrs 10–19 yrs 20–29 yrs ≥30 yrs	8 2 1 4 1	**SIR** 1.4 (0.6–2.7) 3.0 (0.4–11.0) 1.2 (0.0–6.9) 1.1 (0.3–2.9) 1.4 (0.0–8.1)		
Tornling *et al.* (1994), Stockholm, Sweden	Men working as firefighters for at least 1 yr, 1931–83	Enrollment records	Overall	12	**SMR** 1.2 (0.6–2.1)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Baris *et al.* (2001), Pennsylvania, USA	7789 firefighters employed 1925–86	Employee service records	Overall	24	0.9 (0.6–1.4)		
			Duration of employment		**SMR**		
			≤9 yrs	4	0.6 (0.2–1.5)		
			10–19 yrs	14	1.4 (0.8–2.4)		
			≥20 yrs	6	0.7 (0.3–1.4)		
			Hiring period				
			Hired before 1935	17	1.2 (0.7–1.9)		
			1935–44	4	0.6 (0.2–1.6)		
			After 1944	3	0.5 (0.2–1.7)		
			Number of runs				
			Low (<3323)	4	0.7 (0.3–1.8)		
			Medium (3323–5099)	1	0.3 (0.1–2.2)		
			High (5099+)	2	0.7 (0.2–2.6)		
Bates *et al.* (2001), New Zealand	All firefighters employed at least 1 yr, 1977–95	Employment registry	Overall	3	**SIR** 0.8 (0.2–2.2)		Only results for men were presented
Ma *et al.* (2005), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records	Men	12	**SMR** 0.9 (0.5–1.4)	Age, calendar year	
			Women	0	–		
Ma *et al.* (2006), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records	Men	14	**SIR** 0.5 (0.3–0.9)	Age, calendar year	
			Women	0	–		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Colon/Colorectal							
Eliopulos *et al.* (1984), Australia	990 firefighters employed 1939–78 Intestinal cancer	Western Australian Fire Brigade records	Overall	4	**SPMR** 1.6 (0.4–4.1)		
Vena & Fiedler (1987), New York State, USA	1867 male firefighters employed 1950–79 Colon	Death certificates	Overall	16	**SMR** 1.8 (1.1–3.0)		
Beaumont *et al.* (1991), California, USA	3066 male firefighters employed 1940–70 Intestine except rectum	Fire department records	Overall	24	**SMR** 1.0 (0.6–1.5)		
Grimes *et al.* (1991), Hawaii, USA	205 male firefighters Colon	Death certificates	Overall	2	**PRR** 0.9 (0.4–2.2)		Not clear if standardized by age and calendar period

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Demers *et al.* (1992a), Northwest USA	4546 male firefighters employed 1944–79 in selected Northwest cities Colon	Employment records	Overall	24	**SMR** 0.9 (0.5–1.3)	Age, calendar year	
Giles *et al.* (1993), Australia	2865 male firefighters employed 1917–89 Colorectal	Employment and union records, payrolls	Overall	9	**SIR** 1.4 (0.6–2.6)		
Guidotti (1993), Alberta, Canada	3328 firefighters employed 1927–87 Colon and rectum	Personnel files	Overall	14	**SMR** 1.6 (0.9–2.7)		
Aronson *et al.* (1994), Ontario, Canada	5373 male firefighters employed 1950–89 Colon	Employment records	Overall	11	**SMR** 0.60 (0.3–1.1)	Age, calendar year	

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Demers *et al.* (1994), Northwest, USA	2447 male firefighters employed 1974–89 Colon	Employment records			**SIR**		
			Overall	23	1.1 (0.7–1.6)		
			<10 yrs	2	0.8 (0.1–2.9)		
			10–19 yrs	2	0.7 (0.1–2.6)		
			20–29 yrs	15	1.1 (0.6–1.9)		
			≥30 yrs	4	1.5 (0.4–3.9)		
Tornling *et al.* (1994), Stockholm, Sweden	Men working as firefighters for at least 1 yr, 1931–83 Colon	Enrollment records			**SMR**		
			Overall	6	0.9 (0.3–1.9)		
Baris *et al.* (2001), Pennsylvania, USA	7789 firefighters employed 1925–86 Colon	Employee service records	Overall	64	1.5 (1.2–1.9)		
			Duration of employment		**SMR**		
			≤9 yrs	18	1.8 (1.1–2.8)		
			10–19 yrs	16	1.1 (0.7–1.8)		
			≥20 yrs	30	1.7 (1.2–2.4)		
			Hiring period				
			Hired before 1935	16	1.0 (0.6–1.6)		
			1935–44	28	2.0 (1.4–2.9)		
			After 1944	20	1.6 (1.0–2.5)		
			Number of runs				
			Low (<3323)	23	1.9 (1.3–2.9)		
			Medium (3323–5099)	16	2.2 (1.4–3.6)		
			High (5099+)	9	1.2 (0.6–2.4)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Bates *et al.* (2001), New Zealand	All firefighters employed at least 1 yr, 1977–95	Employment registry	Overall	7	**SIR** 0.6 (0.2–1.2)		
	Colon		*Duration of employment*				
			0–10 yrs	1	0.4 (0.0–2.3)		
			11–20 yrs	1	0.5 (0.0–2.6)		
			>20 yrs	5	1.4 (0.4–3.2)		
Ma *et al.* (2005), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records	Men	38	**SMR** 1.1 (0.8–1.6)	Age, calendar year	
	Colon		Women	1	2.3 (0.0–12.7)		
Ma *et al.* (2006), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records	Men	78	**SIR** 1.2 (0.9–1.5)	Age, calendar year	
	Colon		Women	2	2.3 (0.3–8.2)		
Rectum							
Vena & Fiedler (1987), New York State, USA	1867 male firefighters employed 1950–79	Death certificates	Overall	7	**SMR** 2.1 (0.8–4.3)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Beaumont *et al.* (1991), California, USA	3066 male firefighters employed 1940–70	Fire department records	Overall	13	1.5 (0.8–2.5)		
Demers *et al.* (1992a), Northwest, USA	4546 male firefighters employed 1944–79 in selected Northwest cities	Employment records	Overall	8	**SMR** 1.0 (0.4–1.9)	Age, calendar year	
Aronson *et al.* (1994), Ontario, Canada	5973 male firefighters employed 1950–1989 Rectum and rectosigmoid junction	Employment records	Overall	13	**SMR** 1.7 (0.9–2.9)		
Demers *et al.* (1994), Northwest USA	2447 male firefighters employed 1974–89	Employment records	Overall <10 yrs 10–19 yrs 20–29 yrs ≥30 yrs	12 2 3 5 2	**SIR** 1.0 (0.5–1.8) 1.4 (0.2–4.9) 1.9 (0.4–5.4) 0.7 (0.2–1.6) 1.6 (0.2–5.6)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Tornling *et al.* (1994), Stockholm, Sweden	Men working as firefighters for at least 1 yr, 1931–83	Enrollment records			**SMR**		
			Overall	8	2.1 (0.9–4.1)		
Baris *et al.* (2001), Pennsylvania, USA	7789 firefighters employed 1925–86	Employee service records	Overall	14	1.0 (0.6–1.7)		
			Duration of employment		**SMR**		
			≤9 yrs	3	0.9 (0.3–2.7)		
			10–19 yrs	6	1.2 (0.5–2.6)		
			≥20 yrs	5	0.9 (0.4–2.2)		
			Hiring period				
			Hired before 1935	7	1.1 (0.5–2.2)		
			1935–44	3	0.7 (0.2–2.3)		
			After 1944	4	1.2 (0.5–3.2)		
			Number of runs				
			Low (<3323)	5	1.4 (0.5–3.3)		
			Medium (3323–5099)	1	0.5 (0.1–3.6)		
			High (5099+)	1	0.5 (0.1–3.9)		
Bates *et al.* (2001), New Zealand	All firefighters employed at least 1 yr, 1977–95	Employment registry			**SIR**		Only results for men were presented
			Overall	9	1.2 (0.5–2.2)		
			Duration of employment				
			0–10 yrs	2	1.2 (0.1–4.4)		
			11–20 yrs	2	1.4 (0.2–5.0)		
			>20 yrs	4	1.6 (0.4–4.1)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Ma *et al.* (2005), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records	Men Women	7 0	**SMR** 0.9 (0.4–1.9)	Age, calendar year	
Ma *et al.* (2006), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records	Men Women	23 1	**SIR** 0.9 (0.6–1.3) 5.3 (0.1–29.3)	Age, calendar year	
Skin							
Beaumont *et al.* (1991), California, USA	3066 male firefighters employed 1940–70	Fire department records	Compared to police Overall	6 7	**IDR** 1.1 (0.3–4.8) 1.7 (0.7–3.5)	Age, calendar year	White men only
Demers *et al.* (1992a), Northwest, USA	4546 male firefighters employed 1944–79 in selected Northwest cities	Employment records	Overall	6	**SMR** 1.0 (0.4–2.1)	Age, calendar year	
Guidotti (1993), Alberta, Canada	3328 firefighters employed 1927–87	Personnel files	Overall	0	**SMR** 0 (0–3.3)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Tornling *et al.* (1994), Stockholm, Sweden	Men working as firefighters for at least 1 yr, 1931–83; Non-melanoma skin cancer	Enrollment records	Overall	5	**SMR** 1.5 (0.5–3.5)		
Baris *et al.* (2001), Pennsylvania, USA	7789 firefighters employed 1925–86	Employee service records	Overall	10	1.2 (0.6–2.2)		
			Duration of employment		**SMR**		
			≤9 years	2	0.8 (0.2–3.0)		
			10–19 years	5	1.7 (0.7–4.1)		
			≥20 years	3	1.1 (0.3–3.3)		
			Hiring period				
			Hired before 1935	3	1.5 (0.5–4.5)		
			1935–44	1	0.4 (0.1–3.0)		
			After 1944	6	1.5 (0.7–3.3)		
			Number of runs				
			Low (<3323)	1	0.4 (0.1–2.5)		
			Medium (3323–5099)	5	3.1 (1.3–7.5)		
			High (5099+)	1	0.5 (0.1–3.8)		
Ma *et al.* (2005), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records			**SMR**	Age, calendar year	
			Men	17	0.9 (0.5–1.4)		
			Women	0	–		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Ma *et al.* (2006), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records	Men Women	99 5	**SIR** 1.2 (1.0–1.4) 3.0 (1.0–7.0)	Age, calendar year	
Melanoma							
Demers *et al.* (1992b), Northwest, USA	4528 male firefighters employed 1944–79	Employment records		15 5	**SIR** 1.2 (0.7–2.0) **SMR** 1.6 (0.5–3.8)		Data for firefighters and police combined
Giles *et al.* (1993), Australia	2865 male firefighters employed 1917–89	Employment and union records, payrolls	Overall	5	**SIR** 1.1 (0.4–2.5)		
Aronson *et al.* (1994), Ontario, Canada	5373 male firefighters employed 1950–1989	Employment records	Overall	2	**SMR** 0.7 (0.1–2.6)		
Demers *et al.* (1994), Northwest, USA	2447 male firefighters employed 1974–89	Employment records	Overall <10 years 10–19 years 20–29 years ≥30 years	9 0 4 4 1	**SIR** 1.2 (0.6–2.3) 0 (0.0–2.6) 2.3 (0.6–5.8) 1.1 (0.3–2.7) 2.4 (0.1–13.0)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Tornling *et al.* (1994), Stockholm, Sweden	Men working as firefighters for at least 1 yr, 1931–83 Malignant melanoma	Enrollment records	Overall	2	**SMR** 0.8 (0.09–2.9)		
Bates *et al.* (2001), New Zealand	All firefighters employed at least 1 yr, 1977–95	Employment registry	Overall 0–10 years 10–19 years >20 years	23 7 6 6	**SIR** 1.3 (0.8–1.9) 1.7 (0.7–3.5) 1.8 (0.6–3.8) 1.7 (0.6–3.6)		Only results for men were presented
Prostate							
Vena & Fiedler. (1987), New York State, USA	1867 male firefighters employed 1950–79	Death certificates	Overall	5	**SMR** 0.7 (0.2–1.7)	Age, calendar year	
Beaumont *et al.* (1991), California, USA	3066 male firefighters employed 1940–70	Fire department records	Overall	8	**SMR** 0.4 (0.2–0.8)	Age, calendar year	
Grimes *et al.* (1991), Hawaii, USA	205 male firefighters	Death certificates	Overall	4	**PRR** 2.6 (1.4–5.0)	Age, calendar year	Not clear if standardized by age and calendar period

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Demers *et al.* (1992a), Northwest, USA	4546 male firefighters employed 1944–79 in selected Northwest cities	Employment records			**SMR**	Age, calendar year	
			Overall	30	1.3 (0.9–1.9)		
			<10 years	3	2.4 (0.5–7.1)		
			10–19 years	2	1.1 (0.1–4.1)		
			20–29 years	14	1.2 (0.7–2.1)		
			≥30 years	11	1.4 (0.7–2.4)		
					IDR		
			Compared to police	30	1.4 (0.7–2.9)		
Giles *et al.* (1993), Australia	2865 male firefighters employed 1917–89	Employment and union records			**SIR**	Age, calendar year	
			Overall	5	2.1 (0.7–4.9)		
Guidotti (1993), Alberta, Canada	3328 firefighters employed 1927–87	Personnel files			**SMR**	Age, calendar year	
			Overall	8	1.5 (0.6–2.9)		
Aronson *et al.* (1994), Ontario, Canada	5373 male firefighters employed 1950–89	Employment records			**SMR**	Age, calendar year	
			Overall	16	1.3 (0.8–2.2)		
			Duration of employment				
			<15 years	1	1.6 (0.1–9.0)		
			15–29 years	5	2.4 (0.8–5.7)		
			30+ years	9	1.0 (0.4–1.8)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Demers *et al.* (1994), Seattle & Tacoma, USA	2447 male firefighters, employed 1974–89	Employment records			**SIR**	Age, calendar year	Subpopulation of Demers *et al.*, (1992)
			Overall	66	1.4 (1.1–1.7)		
			<10 years	7	1.4 (0.6–2.8)		
			10–19 years	6	1.2 (0.4–2.6)		
			20–29 years	47	1.5 (1.1–2.0)		
			≥30 years	6	0.9 (0.3–1.9)		
					RR		
			Compared to police	66	1.1 (0.7–1.8)		
Tornling *et al.* (1994), Stockholm, Sweden	Men working as firefighters for at least 1 yr, 1931–83	Enrollment records			**SMR**	Age, calendar year	
			Overall	14	1.21 (0.7–2.0)		
Baris *et al.* (2001), Pennsylvania, USA	7789 firefighters employed 1925–86	Employee service records			**SMR**	Age, calendar year	
			Overall	31	1.0 (0.7–1.4)		
			Duration of employment				
			≤9 years	15	2.4 (1.4–3.9)		
			10–19 years	5	0.5 (0.2–1.1)		
			≥20 years	11	0.7 (0.4–1.3)		
			Hired before 1935	12	0.8 (0.4–1.3)		
			1935–44	14	1.4 (0.8–2.3)		
			After 1944	5	0.8 (0.3–2.0)		
			Number of runs				
			Low (<3323)	10	1.3 (0.7–2.5)		
			Medium (3323–5099)	3	0.7 (0.2–2.0)		
			High (5099+)	6	1.4 (0.6–3.2)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Bates *et al.* (2001), New Zealand	All firefighters employed at least 1 yr, 1977–95	Employment registry	Overall	11	**SIR** 1.1 (0.5–1.9)	Age, calendar year	Only results for men were presented
			Duration of employment				
			0–10 years	3	1.5 (0.3–4.3)		
			11–20 years	1	0.6 (0.1–3.3)		
			>20 years	1	0.3 (0.1–1.6)		
Ma *et al.* (2005), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records	Overall	21	**SMR** 1.1 (0.7–1.7)	Age, calendar year	
Ma *et al.* (2006), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records	Overall	209	**SIR** 1.1 (1.0–1.4)	Age, calendar year	Same population as Ma *et al.*, (2005)
Testis							
Giles *et al.* (1993), Australia	2865 male firefighters employed 1917–89	Employment and union records, payrolls	Overall	2	**SIR** 1.2 (0.1–4.2)	Age, calendar year	

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Aronson *et al.* (1994), Ontario, Canada	5373 firefighters employed 1950–89	Employment records	Overall	3	**SMR** 2.5 (0.5–7.4)	Age, calendar year	
			Duration of employment				
			<15 years	3	3.7 (0.8–10.7)		
			15–29 years	0	0.0 (0.0–14.2)		
			30+ years	0	0.0 (0.0–36.9)		
Bates *et al.* (2001), New Zealand	All firefighters employed at least 1 yr, 1977–95	Employment registry	Overall	11	**SIR** 1.6 (0.8–2.8)	Age, calendar year	Only results for men were presented
	Testicular		*Duration of employment*				
			0–10 years	3	1.6 (0.3–4.5)		
			11–20 years	4	3.5 (1.0–9.0)		
			>20 years	2	4.1 (0.5–14.9)		
Ma *et al.* (2006), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records	Men	54	**SIR** 1.6 (1.2–2.1)	Age, calendar year	
Brain / CNS							
Musk *et al.* (1978), Massachusetts, USA	5655 male firefighters employed 1915–75	Death certificates		8	**SMR** 1.0		Confidence interval not provided, not calculated

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Vena & Fiedler (1987), New York State, USA	1867 male firefighters employed 1950–79 Brain	Death certificates	Overall	6	**SMR** 2.4 (0.9–5.1)		
Heyer *et al.* (1990), Washington, USA	2289 male firefighters employed at least 1 yr, 1945–80; follow-up until 1983	Employment records	Overall *Duration of employment* <15 years 15–29 years 30+ years	3 2 1 0	**SMR** 1.0 (0.2–2.8) 1.84 (0.22–6.49) 0.86 (0.10–3.11) 5.03 (1.04–14.70)		
Beaumont *et al.* (1991), California, USA	3066 male firefighters employed 1940–70	Fire department records	Overall	5	0.8 (0.3–1.9)		
Grimes *et al.* (1991), Hawaii, USA	205 male firefighters Brain	Death certificates	Overall	2	**PRR** 3.8 (1.2–11.7)		Not clear if standardized by age and calendar period
Demers *et al.* (1992a), Northwest, USA	4546 male firefighters employed 1944–79 in selected Northwest cities	Employment records	Overall	18	**SMR** 2.1 (1.2–3.3)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Guidotti (1993), Alberta, Canada	3328 firefighters employed 1927–87 Brain	Personnel files	Overall	3	**SMR** 1.5 (0.3–4.3)		
Aronson *et al.* (1994), Ontario, Canada	5373 male firefighters employed 1950–89	Employment records	Overall	14	**SMR** 2.0 (1.1–3.4)		
Demers *et al.* (1994), Northwest USA	2447 male firefighters employed 1974–89 Brain	Employment records	Overall <10 years 10–19 years 20–29 years ≥30 years	4 1 0 3 0	**SIR** 1.1 (0.3–2.9) 1.6 (0.0–8.8) 0 (0.0–4.6) 1.6 (0.3–4.6) 0 (0.0–16)		
Tornling *et al.* (1994), Stockholm, Sweden	Men working as firefighters for at least 1 yr, 1931–83 Brain	Enrollment records	Overall	5	**SMR** 2.8 (0.9–6.5)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Baris *et al.* (2001), Pennsylvania, USA	7789 firefighters employed 1925–86 Brain	Employee service records	Overall	8	0.6 (0.3–1.2)		
			Duration of employment		**SMR**		
			≤9 years	2	0.5 (0.1–1.9)		
			10–19 years	2	0.4 (0.1–1.8)		
			≥20 years	4	0.9 (0.4–2.5)		
			Hiring period				
			Hired before 1935	1	0.4 (0.1–2.6)		
			1935–1944	3	0.7 (0.2–2.2)		
			After 1944	4	0.7 (0.3–1.8)		
			Number of runs				
			Low (<3323)	3	0.6 (0.2–1.9)		
			Medium (3323–5099)	2	0.8 (0.2–3.1)		
			High (5099+)	2	0.7 (0.2–2.9)		
Bates *et al.* (2001), New Zealand	All firefighters employed at least 1 year, 1977–95 Brain	Employment registry	Overall	5	**SIR** 1.3 (0.4–3.0)		Only results for men were presented
Ma *et al.* (2005), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records			**SMR**	Age , calendar year	
			Men	13	0.7 (0.4–1.1)		
			Women	0	–		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Ma *et al.* (2006), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records	Men Women	14 0	**SIR** 0.6 (0.3–1.0)	Age, calendar year	
Non-Hodgkin lymphoma							
Giles *et al.* (1993), Australia	2865 male firefighters employed 1917–89	Employment and union records	Overall	4	**SIR** 1.9 (0.5–4.7)	Age, calendar period	
Aronson *et al.* (1994), Ontario, Canada	5373 male firefighters employed 1950–89 Lymphosarcoma	Employment records	Overall	3	**SMR** 2.0 (0.4–6.0)	Age, calendar year	
Demers *et al.* (1994), Seattle & Tacoma, USA	2447 male firefighters employed 1974–89	Employment records	Overall *Duration fire fighting* <10 years 10–19 years 20–29 years 30+ years	7 1 1 5 0	**SIR** 0.9 (0.4–1.9) 0.9 (0.4–4.9) 0.6 (0.1–4.5) 1.2 (0.4–2.7) 0.0 (0.0–5.8)	Age, calendar year	

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Baris *et al.* (2001), Pennsylvania, USA	7789 firefighters employed 1925–86	Employee service records	Overall	10	1.4 (0.9–2.2)	Age, calendar year	
			Duration of employment		**SMR**		
			≤9 years	6	1.5 (0.7–3.3)		
			10–19 years	5	1.0 (0.4–2.5)		
			≥20 years	9	1.7 (0.9–3.3)		
			Hiring period				
			Hired before 1935	3	0.7 (0.2–2.2)		
			1935–1944	10	2.2 (1.2–4.1)		
			After 1944	7	1.3 (0.6–2.7)		
			Number of runs				
			Low (<3323)	11	2.4 (1.3–4.3)		
			Medium (3323–5099)	4	1.6 (0.6–4.1)		
			High (5099+)	2	0.7 (0.2–2.9)		
Ma *et al.* (2006), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records			**SIR**	Age, calendar year	
			Men	15	1.1 (0.6–1.8)		
			Women	1	33.3 (0.4–185)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Multiple myeloma							
Heyer *et al.* (1990), Seattle, USA	2289 male firefighters employed at least 1 yr, 1945–80; follow-up until 1983	Employment records	Overall *Duration of employment* <15 years 15–29 years 30+ years	3 0 1 2	**SMR** 2.25 (0.47–6.60) 0 (0–15.96) 1.11 (0.03–6.21) 9.89 (1.20–35.71)	Age, calendar year	
Demers *et al.* (1992a), Seattle, Portland & Tacoma, USA	4546 male firefighters employed at least 1 yr, 1944–79; follow-up until 1989	Employment records			**RR** 1.9 (0.4–8.4)	Age, calendar year	A police cohort used as a reference group. Reference rates for US white men were obtained from the National Institute for Occupational Safety and Health. Overlap with Heyer *et al.* (1990)
Aronson *et al.* (1994), Ontario, Canada	5373 male firefighters employed 1950–89	Employment records	Overall	1	**SMR** 0.4 (0.0–2.2)	Age, calendar year	

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Demers *et al.* (1994), Seattle & Tacoma, USA	2447 male firefighters employed 1974–89	Employment records		2	**SIR** 0.7 (0.1–2.6)	Age, calendar year	Overlap with Demers, 1992
Baris *et al.* (2001), Pennsylvania, USA	7789 male firefighters employed 1925–86	Employee service records	*Overall* *Duration of employment* ≤9 years 10–19 years ≥20 years *Hiring period* Hired before 1935 1935–1944 After 1944 *Number of runs* Low (<3323) Medium (3323–5099) High (5099+)	10 1 3 6 4 3 3 1 3 2	**SMR** 1.7 (0.9–3.1) 0.7 (0.1–5.2) 1.5 (0.5–4.7) 2.3 (1.0–5.2) 2.1 (0.8–5.5) 1.4 (0.5–4.4) 1.6 (0.5–4.8) 0.6 (0.9–4.1) 2.7 (0.9–8.4) 1.7 (0.4–6.9)	Age, calendar year	

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Leukaemia							
Musk *et al.* (1978), Massachusetts, USA	5655 male firefighters employed 1915–75 Lymphatic and haemapoietic	Death certificates	Overall	22	**SMR** 0.6		Confidence interval not provided, not calculated
Heyer *et al.* (1990), Washington, USA	2289 male firefighters employed at least 1 yr, 1945–80; follow-up until 1983 Leukaemia and aleukaemia	Employment records	Overall	7	**SMR** 1.7 (0.7–3.6)		
Beaumont *et al.* (1991), California, USA	3066 male firefighters employed 1940–70 Leukaemia and aleukaemia	Fire department records	Overall	6	0.6 (0.2–1.3)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Demers *et al.* (1992a), Northwest USA	4546 male firefighters employed 1944–79 in selected Northwest cities	Employment records		15	**SMR** 1.3 (0.7–2.1)		
Demers *et al.* (1992b), Northwest USA	4528 male firefighters employed 1944–79	Employment records		10 8	**SIR** 1.05 (0.5–1.9) **SMR** 1.3 (0.5–2.5)		Data for firefighters and police combined
Guidotti (1993), Alberta, Canada	3328 firefighters employed 1927–87 Leukaemia, lymphoma, myeloma	Personnel files	Overall	10	**SMR** 1.3 (0.6–2.3)		
Aronson *et al.* (1994), Ontario, Canada	5995 firefighters employed 1950–89 Lymphatic leukaemia	Employment records	Overall	4	**SMR** 1.9 (0.5–4.9)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Demers *et al.* (1994), Northwest USA	2447 male firefighters employed 1974–89	Employment records			**SIR**		
			Overall	6	1.0 (0.4–2.1)		
			<10 years	0	0 (0.0–4.4)		
			10–19 years	2	1.9 (0.2–6.8)		
			20–29 years	4	1.1 (0.3–2.8)		
			≥30 years	0	0 (0.0–5.4)		
Baris *et al.* (2001), Pennsylvania, USA	7789 firefighters employed 1925–86	Employee service records	Overall	15	0.8 (0.5–1.4)		
			Duration of employment		**SMR**		
			≤9 years	5	0.9 (0.4–2.3)		
			10–19 years	7	1.1 (0.5–2.4)		
			≥20 years	3	0.5 (0.2–1.4)		
			Hiring period				
			Hired before 1935	2	0.3 (0.1–1.3)		
			1935–1944	6	1.1 (0.5–2.4)		
			After 1944	7	1.1 (0.5–2.3)		
			Number of runs				
			Low (<3323)	5	0.8 (0.4–2.0)		
			Medium (3323–5099)	4	1.4 (0.5–3.6)		
			High (5099+)	4	1.3 (0.5–3.6)		

Table 2.2 (contd)

Reference, location, name of study	Cohort description	Exposure assessment	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Ma *et al.* (2005), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records	Men Women	14 0	**SMR** 0.8 (0.5–1.4) –	Age, calendar year	
Ma *et al.* (2006), Florida, USA	34 796 male and 2017 female professional firefighters	Employment records	Men Women	20 0	**SIR** 0.8 (0.5–1.2) –	Age, calendar year	

IDR, incidence density ratio; PRR, proportional risk ratio; SIR, standardized incidence ratio; SMR, standardized mortality ratio; SPMR, standardized proportional mortality ratio; yr, year

Deschamps *et al.* (1995) investigated all professional male members of the Brigade des Sapeurs-Pompiers de Paris ($n = 830$) who served for a minimum of 5 years as of January 1st, 1977. They were monitored for a 14-year period, with follow-up terminating on January 1st, 1991. Cause-specific mortality rates in these firefighters were compared with national mortality data provided by the Institut National de la Santé et de la Recherche Médicale. To assess the occupational exposure as a firefighter, data were collected on duration of employment as an active duty firefighter (as opposed to office work). These 830 firefighters accrued a total of 11 414 person–years of follow-up. Follow-up appears to have been 100% complete. There were 32 deaths in the cohort during the 14-year period of follow-up. When compared to the average French male, they were found to have a far lower overall mortality (SMR, 0.52 [95% CI: 0.35–0.75]). None of the cause-specific SMRs was significant. However, a greater number of deaths than expected was observed for genito-urinary cancer (SMR, 3.29) [based on one bladder cancer, and one testicular cancer], and digestive cancer (SMR, 1.14).

Baris *et al.* (2001) conducted a retrospective cohort mortality study among 7789 firefighters in Philadelphia, Pennsylvania, USA, on males employed during 1925–1986. Vital status was ascertained up until 1986. SMRs and 95% CI were calculated with expected numbers of deaths in the United States white male population, as the overwhelming majority of firefighters were white. Occupational exposure histories were abstracted from detailed records maintained by the Philadelphia Fire Department, and a job–exposure matrix was created for each firefighter. To estimate exposure–response relationships, the study used this matrix to compare mortality among groups of firefighters defined by the estimated number of career runs. There were 2220 deaths and a total of 6.2% of the cohort was lost to follow-up. In comparison with white males in the United States, firefighters had a similar mortality from all causes of death combined (SMR, 0.96), and all cancers (SMR, 1.10). Statistically significant excess risks were observed for colon cancer (SMR, 1.51). The risks of mortality from colon cancer (SMR, 1.68), kidney cancer (SMR, 2.20), non-Hodgkin lymphoma (SMR, 1.72), multiple myeloma (SMR, 2.31), and benign neoplasms (SMR, 2.54) were increased in firefighters with at least 20 years of service.

Bates *et al.* (2001) conducted a historical cohort study of mortality and cancer incidence in all remunerated New Zealand firefighters, who served during 1977–1995. Ascertainment of employment was through a registry maintained by the United Fire Brigades Association of New Zealand. The final cohort comprised 4221 male firefighters. To assess the occupational exposure as a firefighter, data were collected on duration of employment. The 4221 male firefighters in this cohort accrued a total of 58 709 person–years of follow-up. Follow-up was successful in tracing 93.5%. There were 117 deaths up until 1995. Cancer incidence was ascertained during 1977–1996. The SIR for all cancers was 0.95. For most sites, no excesses were observed. The only cancer for which this study provided evidence of an increased risk was

testicular cancer. Eleven testicular cancers were observed versus 7.1 expected (SIR, 1.55; 95% CI: 0.8–2.8). For the years 1990–1996, the SIR for testicular cancer was 3.0 (95% CI: 1.3–5.9).

Ma *et al.* (2005) examined age- and gender-adjusted mortality rates of 36 813 professional firefighters employed during 1972–1999 in Florida, USA, and compared those with that of the Florida general population. The study population consisted of 34 796 male and 2017 female professional firefighters. The racial/ethnic composition was caucasian (90.1%), hispanic (7%), and black (6.5%). Employment as a firefighter was ascertained from employment records in the Florida State Fire Marshall Office. Surrogate information on occupational exposures in firefighting was collected by examining the year of certification and duration of employment as a firefighter. No information was collected on smoking histories. A total of 1411 male and 38 female deaths with known causes were identified in this cohort. In male firefighters, a deficit of overall mortality from cancer was observed (SMR, 0.85). Excess risks were observed for male breast cancer (SMR, 7.41; 95% CI: 1.99–18.96), and thyroid cancer (SMR, 4.82; 95% CI: 1.30–12.34), each based on four cases. Mortality from bladder cancer was increased and approached statistical significance (SMR, 1.79; 95% CI: 0.98–3.00). Female firefighters had similar overall cancer mortality patterns to Florida women (SMR, 1.03), but the numbers were small for specific cancer sites.

In a further analysis of the same cohort, Ma *et al.* (2006) determined the relative cancer risk for firefighters in the State of Florida compared with the Florida general population. Employment as a firefighter was ascertained from employment records in the Florida State Fire Marshall Office. Cancer incidence was determined through linkage to the Florida Cancer Data System, a statewide cancer registry estimated to capture 98% of cancers in Florida residents. No pathological verification of cancer diagnoses was undertaken. A total of 970 male and 52 female cases of cancer were identified; 6.7% of the cohort were lost to follow-up. Male firefighters had significantly increased incidence rates of cancers of the bladder (SIR, 1.29; 95% CI: 1.01–1.62), testis (SIR, 1.60; 95% CI: 1.20–2.09), and of the thyroid (SIR, 1.77; 95% CI: 1.08–2.73). Female firefighters had significantly increased incidence rates of overall cancer (SIR, 1.63; 95% CI: 1.22–2.14), cervical (SIR, 5.24; 95% CI: 2.93–8.65) and thyroid cancers (SIR, 3.97; 95% CI: 1.45–8.65), and Hodgkin disease (SIR, 6.25; 95% CI: 1.26–18.26).

2.2 Case–control studies

Case–control studies have been used to examine the risk of firefighting and its association with various types of cancers. In all but one of these studies, ten or fewer firefighters were included in the case and/or control group. Several studies combined broad occupational categories with heterogeneous exposures such as firefighter and fireman, with the latter not necessarily working as a firefighter. These types of studies may result in exposure misclassification. Even within specific occupational groups such as firefighters, all would not have the same intensity or type of exposures. The

magnitude of this form of misclassification is unknown, but it is likely that the resulting misclassification will be non-differential with regard to cases and controls. Another limitation to case–control studies is that cases may be more likely than controls to remember jobs of shorter duration. Those jobs in the more distant past may be more likely recalled by cases than controls resulting in differential bias away from the null. Alternatively, in several of the reported studies, cases were more likely than the controls to provide proxy interviews by their survivors rather than by the cases themselves. Because of the relatively few studies available for individual organ sites, the studies were grouped into four categories including urogenital, brain and central nervous system, larynx and lung, and other.

2.2.1 *Cancers of the urogenital system*

Four cancers of the urogenital organs in relation to employment as a firefighter were considered (Tables 2.3 and 2.6).

Delahunt *et al.* (1995) examined pathologically confirmed incident renal cell carcinomas the New Zealand Cancer Registry during the period 1978–1986. The registry included 95% of those patients diagnosed and treated in both the public and private sector. At time of registration, the current or most recent occupation was recorded. Additional information collected included age, and smoking habits. Renal cell carcinomas with an ICD-9 code of 189.0 (malignant neoplasm of the kidney, excluding the renal pelvis) were evaluated. The control groups were a random sample of registrations drawn from all cases over 20 years of age, having primary tumours from sites other than the urinary tract registered during the same time period. There were a total of 710 male cases and 12 756 controls. There were 52 cases and 737 controls under the occupational classification of "Service" which included firefighters and five other occupational groups. The relative risk for firefighters was 4.7 (95% CI: 2.5–8.9).

Bates (2007) (see Table 2.6) conducted a registry-based case–control study using the California Cancer Registry. Anonymized records of all male cancers for the period 1988–2003 were collected. To identify firefighters, the occupation and industry fields were searched for titles including fire, firefighter, fire fighter, fireman, fire man, and fire chief. If the subjects indicated that they did not carry out firefighting activities, they were not considered. A total of 16 cancer organ sites were examined including kidney, bladder, prostate, and testis. For each analysis, all other cancers were used as controls except for those cancers shown in the initial analysis that had demonstrated a firefighting etiology; these included cancers of the lung, bronchus, bladder, prostate, colorectum, and skin melanomas. Analysis was limited to males aged 21–80 at time of diagnosis. There were 3659 firefighters and 800 448 controls in the analysis after exclusion of 13% of the files (n = 140 000) with no recorded occupation or industry. Logistic regression analyses were performed for each cancer type for which there had been more than 50 cancers recorded in firefighters. This was not done for for cancer of the thyroid (n = 32 cases) or multiple myeloma (n = 37 cases) as these two were based on prior hypotheses.

Table 2.3. Case–control studies of the urogenital system

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	No. of exposed cases	Odds Ratio (95% CI)	Adjustment for potential confounders	Comments
Delahunt *et al*. (1995) New Zealand, 1978–86	Renal cell (189.0)	Total number of renal cell carcinomas for all occupations –710 men (Cancer Registry); coverage of 95% incident cases including pathology coding. Occupational code for 86.2%; 5 categories of service workers including firefighters; in which, 52 cases with an unknown number of firefighters	Random sample drawn from all cancer cases except renal cell carcinoma aged over 20 years, having primary tumours from sites other than the urinary tract. 12 756 (all men, Cancer Registry); matched by age, and registration period. 737 controls for category of service workers	Occupation code used to identify employment	Firefighters unadjusted Firefighters age- and smoking-adjusted	NR NR	3.51 (2.09–5.92) 4.69 (2.47–8.93)	Age, smoking	Firefighters likely represented ~10 cases although exact numbers not reported

Table 2.3 (contd)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	No. of exposed cases	Odds Ratio (95% CI)	Adjustment for potential confounders	Comments
Krstev *et al*. (1998) Atlanta, Detroit and 10 counties in New Jersey, USA 1986–89	Prostate	Population-based incident cases from registry; 981 (479 blacks and 502 Caucasians) aged 40–79 years; cases selected by random sample to ensure broad distribution by age and race, a varying proportion of cases selected by random sampling. Histologically confirmed. Response rate not provided but 6 cases and 17 controls with no employment data	1315 (594 blacks and 721 Caucasians) population-based controls selected aged <65 years by random-digit dialling, and >65 selected by random sampling from computerized records of the Health Care Financing for each geographic area administration; matched by age, sex and race	In-person interviews by trained interviewers	Firefighting and prevention – All firefighting Duration of firefighting <5 years 5–19 years ≥20 years	 10 2 3 5	3.85 (1.34–11.10) 3.34 (1.13–9.91) – 1.66 (0.33–8.36) 3.94 (0.76–25.60) *P* for trend=0.07	Age, sex and race	Small number of cases and controls

Table 2.3 (contd)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	No. of exposed cases	Odds Ratio (95% CI)	Adjustment for potential confounders	Comments
Stang *et al*. (2003) Bremen, Essen, Hamburg, Saarbrücken, and Saarland, Germany, 1995–97	Testicular or extragonadal germ cell tumours	269 cases from an active reporting system of clinical and pathological departments; aged 15–69 years; 78% response rate; histologically confirmed. 4 cases (1.5%) were firefighters	797 controls selected randomly from mandatory registries of residence; 57% response rate; matched by age and region of residence 3 controls (0.4%) were fireghters	In-person and telephone interviews conducted by trained interviewers	Worked as a firefighter Ever ≥10 years of duration Work began ≥5 years before diagnosis	 4 2 3	 4.3 (0.7–30.5) 3.0 (0.2–45.5) 3.1 (0.4–24.4)	History of cryptorchidism	Number of firefighter case and controls too low for precise effect but trend is strong

Table 2.3 (contd)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	No. of exposed cases	Odds Ratio (95% CI)	Adjustment for potential confounders	Comments
Gaertner *et al*. (2004) Newfoundland, Prince Edward Island, Nova Scotia, Manitoba, Alberta, Saskatchewan and British Columbia, Canada, 1994–97	Bladder	Incident cases identified in Cancer Registries: 887 cases from Canadian provincial cancer registries; aged 20–74 years; 58 % male response rate, and 61% female response rate; 8 male firefighters	2847 population-based controls selected by random-digit dialling to recruit controls in Newfoundland and Alberta, and random sampling from the provincial health insurance plan database for other locations; 59% male response rate, and 65% female response rate; matched by age and sex 13 male controls	Mailed questionnaires and telephone follow-ups	Firefighter males only Duration of firefighting in years	8	1.51 (0.59–3.84)	Adjusted for age, province, race, smoking, ex-smoking, consumption of fruit, fried food and coffee	No females were included in the analysis
					>1–5	3	2.00 (0.43–9.49)		
					>5–15	1	0.86 (0.71–8.93)		
					>15	4	1.36 (0.36–5.16)		

NR, not reported

Logistic regression analyses adjusted for 5-year age categories, 4-year categories from date of diagnosis, five ethnic categories and five categories of an indicator of socioeconomic status. A total of 101 firefighters with a diagnosis of cancer of the kidney or renal pelvis were assessed, and the OR was 1.07 (95% CI: 0.87–1.31), adjusted for age, calendar period of diagnosis, race, and an indicator of socioeconomic status for the census block of residence.

Krstev *et al.* (1998) investigated incident prostate cancer cases in the USA using population-based cancer registries for Atlanta Georgia and Detroit Michigan, and for ten counties in the state of New Jersey during 1986–1989. Histologically confirmed cases were identified from pathology and outpatient records at hospitals included in these registries. Cases were selected by random sampling among the total number of cases identified in each age–race category. [Three additional cancer sites were investigated but not reported including oesophagus, pancreas, and multiple myeloma, and no published articles were located regarding these cancers.] Control subjects were proportional to the age, sex, and race distribution of the cases. Controls younger than 65 years of age were selected through random-digit dialling. Older controls were systematically selected by random sampling from computerized records of the Health Care Financing Administration stratified by three age groups, and race (african american or caucasian for each geographic area). Cases and controls were interviewed in person. There were 981 cases and 1315 controls analysed using unconditional logistic regression adjusted for age (< 60, 60–69, 70+), study site, and race. A total of ten cases and five controls were classified as firefighting (SOC 512.3). The overall adjusted OR for prostate cancer was 3.85 (95% CI: 1.34–11.10), for caucasians only (nine cases and three controls) 4.75 (1.26–18.00), and for african americans (three cases and two controls), 2.64 (0.43–16.20).

Bates (2007) evaluated 1144 firefighters diagnosed with cancer of the prostate (cohort described above for cancer of the kidney), and found an adjusted OR of 1.22 (95% CI: 1.12–1.33).

Stang *et al.* (2003) examined the risk of testicular cancer or extragonadal germ cell tumours during 1995–1997 in five German regions. Cases were reported through an active reporting system. A pathologist derived histological evaluations for 95% of the cases. Interviews were conducted with 269 of the 353 eligible cases, with a response rate of 78% including the two surrogate interviews. Controls were randomly selected from mandatory registries of residence. Approximately two controls were age- and region-matched for the cases between the ages of 15–34 years. Four controls were matched for those cases aged 35–69 to increase study power related to the fewer number of cases in this older age group. The response rate in the controls was 57%, with 918 interviewed (eight surrogate) of 1982 eligible subjects. Each job held for at least 6 months was recorded including job tasks and hours per week worked. These jobs were coded according to the International Standard Classification of Occupation. Conditional logistic regression models were calculated with matching factors including 5-year age groups, and geographic region. The adjusted ORs for ‘ever’

versus 'never' employed as a firefighter were 4.3 (95% CI: 0.7–30.5, four cases and three controls); for working as a firefighter ≥ 10 years, 3.0 (95% CI: 0.2–45.5, two cases and two controls); and for employment ≥ 5 years before the 'reference' date [date of diagnosis], 3.1 (95% CI: 0.4–24.4, three cases and three controls).

Bates (2007) also evaluated 70 firefighters diagnosed with cancer of the testis (SEER code 28020, cohort described above for cancer of the kidney), and found an adjusted OR of 1.54 (95% CI: 1.18–2.02).

Gaertner *et al.* (2004) reported on incident cases of bladder cancer with a histological confirmation, identified through the National Enhanced Cancer Surveillance System programme in seven Canadian provinces. The cases were adults aged 20–74, identified during 1994–1997 and interviewed 2–5 months after diagnosis. Random selections of population controls were included in the programme by frequency-matching age and gender to all cancer cases. Random digit dialling was used during the 1996 calendar year to recruit controls living in Newfoundland and Alberta, while all other provinces used a random sample from the provincial health insurance database. Native Indians and subjects in the military were excluded from the study. Mailed questionnaires with telephone follow-up, as necessary, were used to gather data regarding sociodemographics, occupational history, smoking history, dietary habits, and specific agent exposures. The response rates for the male and female bladder cancer cases were 66% and 72%, respectively, and for the controls, 59% and 65%, respectively. The overall analysis included 887 cases and 2847 controls. In the analysis of firefighters, eight male cases and 13 male controls were considered. the Standardized Occupational Classification system was used to code occupations, with up to 12 occupations coded per person. Data analysis also included demographic information provided from the interviews. An unconditional logistic regression analysis was used adjusting for age, province, race, smoking, ex-smoking, and consumption of fruit, fried food, and coffee. For the analysis of 'ever' or 'never' worked as a firefighter for more than one year, an elevated OR of 1.51 (95% CI: 0.59–3.84) was found. When stratified by duration of employment as a firefighter, the ORs were: 2.0 (95% CI: 0.43–9.49) for > 1–5 years (three cases and four controls); 0.86 (95% CI: 0.708–8.93) for > 5–15 years (one case and three controls); and 1.36 (95% CI: 0.36–5.16) for > 15 years (four cases and six controls).

Bates (2007) assessed 174 firefighters diagnosed with cancer of the bladder (SEER code 29010, cohort described above for cancer of the kidney and Table 2.6), and found an adjusted OR of 0.85 (95% CI: 0.72–1.00).

2.2.2 *Cancer of the brain*

Four studies on brain cancer in relation to firefighting were considered, all from the USA (Tables 2.4 and 2.6).

Brownson *et al.* (1990) evaluated brain cancers using the Missouri Cancer registry. Cancer cases from public and private hospitals have been collected since 1972, and reporting has been mandated since 1984. The group of cases comprised Caucasian

males diagnosed with histologically confirmed brain and other central nervous system cancers (ICD codes 191 and 192). Four controls were randomly selected and frequency-matched from all Caucasian male patients diagnosed with cancers during the same time period. Control group cancers included cancers of the lip, oral cavity and pharynx, digestive organs and peritoneum, respiratory system, skin, bones and connective tissue, genitourinary system, and leukaemia, lymphoma, multiple myeloma, and other sites. Of the initially eligible cases, occupational information was lacking in 34% of the cases, and 38% of the controls. Analysis combined industries with Unites States census code related to justice, public order and safety which included firefighters, and for occupations combining police and fire protection services. Age- and smoking-adjusted ORs were elevated and reported as 2.1 (95% CI: 0.9–4.8, ten cases and 19 controls) for the industry of justice, public order and safety, and 2.2 (95% CI: 1.0–4.7, 12 cases and 22 controls) for police and fire protection workers. This excess risk among police and fire protection workers was confined to the astrocytic cell series (OR, 2.3; 95% CI: 1.0–5.1). The OR for firefighters examined separately was 2.0 (95% CI: 0.4–9.6), with an unknown number of cases and controls.

Carozza *et al.* (2000) conducted a population-based case–control study among adults in the San Francisco Bay area during 1991–1994. Lifetime job histories were available for this study. Using the Northern California Cancer Center population, 603 incident cases of gliomas among adults aged 20 years or older were identified with histological confirmation (ICD 9380–9481). Interviews were completed with 492 cases (82%), and 476 were analysed after additional exclusions. Using random-digit dialling, controls were frequency-matched by 5-year age groups as well as by gender and race/ethnicity. There were 754 potential controls identified with 22 removed because of their residence, insufficient level of English or some relationship to the cases. Of the 732 controls meeting the eligibility criteria, 462 (63%) interviews were completed. The job history data for cases and controls were provided by proxy for 45.6% and 0.9%, respectively. For each job reported, the following information was collected: name and location of the company, description of daily work activities, starting date and duration of job including hours worked per week. Jobs were coded using Standardized Occupational Classification 1980 and Standard Industrial Codes 1987 without knowledge of the case–control status. Duration of all jobs held for at least 6 months was analysed; the most recent 10 years were excluded to allow for a hypothesized 10-year latency period between the exposure and the clinical recognition of the disease. Subjects who were not employed in the occupational category being evaluated served as the 'unexposed' referent group. Multiple logistic analyses were used adjusting for age, gender, years of education and race (caucasian, non-caucasian). Astrocytic tumours were evaluated including glioblastoma, multiforme, and astrocytoma. The adjusted OR for 'ever' versus 'never' employed as a fireman was 2.7 (95% CI: 0.3–26.1), and for being diagnosed with having an astrocytic tumour, 3.6 (95% CI: 0.4–36, three cases, 1 control).

Table 2.4. Case–control studies of the brain

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	No. of exposed cases	Odds Ratios (OR) (95% CI)	Adjustment for potential confounders	Comments
Brownson *et al*. (1990) Missouri, USA 1984–88	Brain and other central nervous system cancers (191 and 192)	312 caucasian males; histologically confirmed brain and central nervous system cancers, identified through the Missouri Cancer Registry, maintained by the Missouri Department of Health	1248 frequency-matched (4:1) sample of controls chosen from all other caucasian male patients diagnosed with cancers in the same time period, including lip/oral cavity/pharynx, digestive organs and peritoneum, respiratory, skin, bones and connective tissue, genitourinary, leukaemia, lymphoma, and multiple myeloma, and other sites. Controls randomly selected within each of six age strata. 38% of controls excluded due to missing occupational information	Hospital medical records	*Brain Cancer by Industry* Justice/public order/safety *Brain Cancer by occupation* Police and fire protection services Astrocytic cell type only Firefighters only	10 12 NR NR	2.1 (0.9–4.8) 2.2 (1.0–4.7) 2.3 (1.0–5.1) 2.0 (0.4–9.6)	Adjusted for age and smoking	Limited to caucasian males due to small numbers of non-caucasians and lack of reported occupational diversity among females. 34 % of cases excluded because of missing occupational data. Analysis combined those in police and fire protection US census codes 413–427

Table 2.4 (contd)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	No. of exposed cases	Odds Ratios (OR) (95% CI)	Adjustment for potential confounders	Comments
Carozza *et al.* (2000) San Francisco Bay area including Alameda, Contra Costa, Marin, San Mateo, San Franscisco, and Santa Clara counties, USA 1991–94	Brain (Gliomas, 9380–9481)	603 cases of histologically confirmed incident cases of glioma. Age >20 years	462 controls matched by 5-year age groups, gender, and race/ethnicity, and identified by random-digit dialling	Interviews and Standard occupational and Industrial codes used	Ever employed as firefighter Astrocytic tumours	3 3	2.7 (0.3–26.1) 3.6 (0.4–36.1)	Matched on age, gender, education, and race	Only 3 cases and 1 control were firefighters. Duration of job calculated for every job held at least 6 months during subjects' lifetime also with the most recent 10 years excluded to allow for hypothesized 10-year latency period between exposure and clinical recognition of disease

Table 2.4 (contd)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	No. of exposed cases	Odds Ratios (OR) (95% CI)	Adjustment for potential confounders	Comments
Krishnan *et al*. (2003) Alameda, Contra Costa, Marin, San Mateo, San Francisco and Santa Clara counties, California, USA 1991–94 and 1997–99	Glioma (938.0, 948.1)	879 incident cases identified using Northern California Cancer Center's rapid ascertainment programme; 81% response rate	864 population-based controls selected by random-digit dialling; frequency- matched by age, race, and sex; 66% response rate	Interviews and Standard occupational and industrial codes used	Ever employed	9	2.85 (0.77–10.58)	Age, race and sex	40% of case participants reported by proxy
					Longest-held job as a firefighter	6	5.88 (0.70–49.01)		Referent group for analysis included those without the given occupational group as their longest-held job, including those who were never employed
					Longest-held job as a firefighter and astrocytic cases only	5	6.31 (0.73–54.40)		
					Longest held job as firefighter non-astrocytic cases	1	9.27 (0.55–155.27)		

Krishnan *et al.* (2003) conducted a follow-up study to the one designed by Carozza *et al.* (2000). This follow-up study examined incident glioma cases diagnosed during both 1991–1994 and 1997–1999. All adults newly diagnosed with glioma during these time periods were ascertained using the Northern California Cancer Center's rapid ascertainment programme. Controls were ascertained through random-digit dialling and matched to cases by age, race, and gender. There were 1129 eligible cases with 81% ($n = 896$) completing full interviews. In-person interviews were conducted for 98%, and there were 879 cases with complete information available for analysis. Of the eligible controls, 66% ($n = 864$) completed a full interview. In the analysis of 'ever' employed as a firefighter, the OR was 2.85 (95% CI: 0.77–10.58, nine cases and three controls). Analysis by the longest-held job resulted in an OR of 5.88 (95% CI: 0.70–49.01, six male cases and one male control). In the analysis of astrocytic cases, the OR was 6.31 (95% CI: 0.73–54.4, five cases and one control), and for the non-astrocytic cases, 9.27 (95% CI: 0.55–155.27, one case and one control). [These two studies are very similar with more cases and controls available in the Krishnan report. The Krishnan report, however, did not carry out analyses by 10-year latency period, and therefore both studies may be relevant.]

Bates (2007) also evaluated brain cancers (SEER code 31010) in firefighters as described above under kidney cancer and Table 2.6. There were 71 firefighters with brain cancer. The adjusted OR was 1.35 (95% CI: 1.06–1.72).

2.2.3 *Cancers of the larynx and lung*

One case–control study of cancer of the larynx and two studies of cancer of the lung were considered by the Working Group (Tables 2.5 and 2.6).

Muscat and Wynder (1995) conducted a case–control study of cancer of the larynx in New York City, USA, recorded during 1956–1965. Caucasian men from seven hospitals newly diagnosed with histologically confirmed cancer of the larynx were interviewed. Control subjects were also caucasian men frequency-matched to the cases by hospital of diagnosis, age (within 5 years), and year of interview. Eligibility as a control also required a hospital admission for a condition unrelated to an etiology associated with tobacco exposures including cancer of the prostate, lymphomas, benign prostatic hypertrophy, and various non-malignant conditions. All subjects were interviewed by personnel who were not blinded to the case–control status of subjects, with a 90% response rate. The questionnaire included information on smoking status (never, current or ex-smoker, number of cigarettes, pipe and cigars smoked, and alcohol intake). Data were collected on lifetime occupations and self-reported exposures to chemicals, metals, exhaust, asbestos, and other occupational substances. There were 235 cases and 205 controls. The cases compared to controls were most likely to be: current cigarette smokers, (66.4% and 24.4%, respectively), heavy cigarette smokers (> 31 cigarettes/day), (55.1% and 22.8%, respectively), and drink more than 7 ounces of alcohol per day (29.4% and 11.2%, respectively). Analyses were adjusted for current smoking status.

Table 2.5. Case–control studies of cancers of the larynx and lung

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	No. of exposed cases	Odds Ratios (OR) (95% CI)	Adjustment for potential confounders	Comments
Muscat & Wynder (1995) New York, USA 1956–65	Larynx	235 caucasian men from 7 hospitals with histologically confirmed laryngeal cancer	205 caucasian men, 90% response rate of "eligible patients"; frequency-matched by hospital, age (within 5 years) and year of interview. Controls selected for condition unrelated to tobacco-induced diseases and included prostrate cancer, lymphomas, benign prostatic hypertrophy, non-malignant conditions. Only 2 firefighters (1%)	Interview	Laryngeal Cancer Classified as working in diesel exhaust job Self-reporting exposure to diesel exhaust	2 36 13	0.96 (0.5–1.8) 1.47 (0.5–4.1)	Smoking	

Table 2.5 (contd)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	No. of exposed cases	Odds Ratios (OR) (95% CI)	Adjustment for potential confounders	Comments
Elci *et al*. (2003) Istanbul and Marmara region, Turkey 1979–84	Lung (ICD: 162.0 and 162.2 combined, and compared to 162.3, 162,4, 162.5 and 162.9)	1354 male cases of lung cancer; 442 cases without histological confirmation	1519 male cancer and non-cancer controls diagnosed with Hodgkin disease, soft tissue sarcoma, testis, bone, male breast and non-cancer benign pathologies	Standardized questionnair eJobs classified by Standard occupational and Industrial codes	*Lung Cancer by* Firefighter Squamous cell All bronchus and parenchyma	 10 4 9	 6.8 (1.3–37.4) 6.2 (0.8–46.2) 7.0 (1.3–39.1)	Age and smoking	22 women were excluded from analyses

Among the cases, two were employed as firefighters. Of those occupations which self-reported exposure to diesel exhaust, including truck drivers, firefighters, road workers, and mine workers (5.5 cases and 4.4 controls), the adjusted OR was 1.47 (95% CI: 0.5–4.1). For those occupations which self-reported exposure to diesel 'fumes', firefighter was not listed amongst them The authors noted that the self-reported exposure to diesel exhaust or diesel fumes may reflect uncontrolled confounding with cigarette smoking and alcohol consumption as almost all patients who reported diesel exposure were also heavy cigarette smokers, and consumed large amounts of alcohol.

Elci *et al.* (2003) examined the link between occupations and risk of lung cancer by histological types in Turkey. Cases were identified from an oncology treatment centre at one of the largest cancer hospitals, including treatment for workers, in Istanbul. After admission to hospital, all patients completed a standardized questionnaire administered by trained interviewers. There were 1354 male lung cancer cases with complete interview information identified during 1979–1984. An oncologist reviewed hospital records for diagnostic verification and classification of cancer types. When there were four or more cases per cancer type, histopathology and morphological type was examined. Patient controls "with the same sociodemographic background as the cases" were selected having the following diagnoses: cancers of the skin (non-melanoma), testis, bone, male breast, Hodgkin disease, soft-tissue sarcoma, and non-cancer patients. Of the 27 occupations, firefighting ($n = 10$ cases) had an excess risk of lung cancer, with an age- and smoking-adjusted OR of 6.8 (95% CI: 1.3–37.4). In firefighters, for squamous-cell carcinoma ($n = 4$), the age- and smoking-adjusted OR was 6.2 (95% CI: 0.8–46.2), and for peripheral tumours including bronchus and parenchyma ($n = 9$), the age- and smoking-adjusted OR was 7.0 (95% CI: 1.3–39.1).

Bates (2007) investigated cancers of the lung and bronchus in firefighters as described above under kidney cancer and in Table 2.6. There were 495 firefighters with these cancers. The adjusted OR was 0.98 (95% CI: 0.88–1.09).

2.2.4 *Cancers at other sites*

(*a*) *Multiple myeloma, non-Hodgkin lymphoma, and leukaemia*

Demers *et al.* (1993) identified cases of multiple myeloma through SEER tumour registries in four geographic locations including two counties in Washington State, two in Utah including Salt Lake City, five counties of metropolitan Atlanta, Georgia, and three metropolitan Detroit, Michigan, counties. All those potentially eligible included all incident cases diagnosed during 1977–1981. Controls were selected to be similar in age, gender, and region. In Washington State, 1683 population-based controls were selected by using two sampling units of four households. In other areas, a random-digit dialling method was used for selecting controls. Interviews were obtained from 692 (89%) of the cases or their survivors, and from 1683 (83%) of the controls.

Table 2.6. Case–control studies of other or multiple sites

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	No. of exposed cases	Odds Ratios (OR) (95% CI)	Adjustment for potential confounders	Comments
Demers *et al*. (1993) King and Pierce counties, Washington; Davis county, Salt Lake and Weber county, Utah; Atlanta, Georgia; Detroit, Michigan; USA 1977–81	Multiple myeloma	692 cases of multiple myeloma. Total incident cases from tumour registries in the USA, aged <80 years	1683 population-based controls in Washington state selected by two sampling units of four households, and in other areas random-digit dialling; response rate 83%; matched by age, sex, and region	Interview and proxy interviews	All firefighters Employed ≥10 years Self-responding	5 4 4	1.9 (0.5–9.4) 2.9 (0.4–21.6) 2.8 (0.5–14.3)	Sex, race, age, and study area.	Small number of cases and controls. Uncertain as to which jobs represent firefighting and prevention occupations

Table 2.6 (contd)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	No. of exposed cases	Odds Ratios (OR) (95% CI)	Adjustment for potential confounders	Comments
Bates (2007) California, USA 1988–2003	Oesophagus Colorectum Lung Melanoma Prostate Testis Bladder Brain Thyroid Leukaemias Non-Hodgkin lymphoma Multiple myeloma	3659 cases (all men) from the California Cancer Registry, aged 21–80 years; 94% histologically confirmed	All other males in registry that were not firefighters (*n*=800448) from California Cancer Registry except those diagnosed with cancers of the lung, bronchus, prostate, colorectum, and skin melanomas.	California Cancer Registry records	Oesophagus Stomach Colorectum Caecum Pancreas Lung & bronchus Melanoma Prostate Testis Bladder Kidney & renal pelvis cancer Brain Thyroid cancer Leukaemias Non-Hodgkin lymphoma Multiple myeloma	62 51 282 52 63 495 323 1144 70 174 101 71 32 100 159 37	1.48 (1.14–1.91) 0.80 (0.61–1.07) 0.90 (0.79–1.03) 1.09 (0.82–1.44) 0.90 (0.70–1.17) 0.98 (0.88–1.09) 1.50 (1.33–1.70) 1.22 (1.12–1.33) 1.54 (1.18–2.02) 0.85 (0.72–1.00) 1.07 (0.87–1.31) 1.35 (1.06–1.72) 1.17 (0.82–1.67) 1.22 (0.99–1.49) 1.07 (0.90–1.26) 1.03 (0.75–1.43)	SES quintile	Use of other cancer controls may have biased study toward null

SES, socioeconomic status

For the cases, 220 (32%) were interviewed by proxy. Analyses were adjusted for gender, race, 4-year age groups, and study area. The adjusted OR for employment in firefighting and prevention occupations was 1.9 (95% CI: 0.5–9.4, five cases and five controls), and for the self-reporting category, 2.8 (95% CI: 0.5–14.3, four cases). The OR for firefighters employed < 10 years was 0.9 (95% CI: 0.0–22.3, one case and two controls), while for those employed 10 or more years, the OR increased to 2.9 (95% CI: 0.4–21.6, four cases and three controls).

Bates (2007) also investigated multiple myeloma, non-Hodgkin lymphoma, and leukaemia in firefighters (for full study description see Section 2.2.1 and Table 2.6), for which the ORs were reported as 1.03 (95% CI: 0.75–1.43, 37 cases), 1.07 (95% CI: 0.90–1.26, 159 cases), and 1.22 (95% CI: 0.99–1.49, 100 cases), respectively.

(*b*) *Cancers of the gastrointestinal system and pancreas*

Bates (2007) conducted the only study investigating cancers of the gastrointestinal system in firefighters. The ORs for cancers of the stomach were 0.80 (95% CI: 0.61–1.07, 51 cases), of the colorectum 0.90 (95% CI: 0.79–1.03, 282 cases), of the caecum 1.09 (95% CI: 0.82–1.44, 52 cases), and of the pancreas 0.90 (95% CI: 0.70–1.17, 63 cases).

(*c*) *Thyroid cancer*

Bates (2007) assessed 32 firefighters with cancer of the thyroid, and found an OR of 1.17 (95% CI: 0.82–1.67).

(*d*) *Melanoma*

Bates (2007) investigated firefighters ($n = 323$) diagnosed with melanoma, and found a significant and elevated OR of 1.50 (95% CI: 1.33–1.70).

2.3 Descriptive studies

Several descriptive studies have provided results for firefighters. These have varied in their design including cohort studies based on record linkage, and studies based solely on death certificate or registry data. In some cases, these have been investigations specifically directed at firefighters. They are described in more detail below and in Tables 2.7 and 2.8.

2.3.1 *Cohort and linkage studies of firefighters*

Feuer & Rosenman (1986) conducted a study of deaths among active and retired firefighters from the state of New Jersey, USA, during 1974–1980. Firefighters were identified using pension records, and their duration of employment was also collected. Their mortality was compared to that of the police force, identified in the same manner, and of the general population. Proportionate mortality ratios (PMRs) were calculated based on 263 caucasian male firefighter deaths, and a significant excess of leukaemia was observed using to the police force as reference group.

Table 2.7. Cohort and linkage studies of firefighters

Reference, location, name of study	Study population description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/ deaths	PMR/SMR/MOR (95% CI)	Adjustement for potential confounders	Comments
Feuer & Rosenman (1986), New Jersey, USA 1974–80	263 active and retired firefighters, identified using retirement system records	Pension records				**PMR**	Age	
			All cancers	Overall, compared to NJ	67	1.0 [n.s.]		
				≤20 years	15	0.9 [n.s.]		
				20–25 years	18	1.0 [n.s.]		
				>25 years	34	1.1 [n.s.]		
				Compared to Police	67	1.1 [n.s.]		
			Digestive	Overall, compared to NJ	20	1.1 [n.s.]		
				≤ 20 years	5	1.2 [n.s.]		
				20–25 years	5	1.0 [n.s.]		
				>25 years	10	1.2 [n.s.]		
				Compared to Police	20	0.9 [n.s.]		
			Respiratory	Overall, compared to NJ	23	0.9 [n.s.]		
				≤ 20 years	4	0.7 [n.s.]		
				20–25 years	7	1.0 [n.s.]		
				>25 years	12	1.0 [n.s.]		
				Compared to Police	23	1.0 [n.s.]		
			Skin	Overall, compared to NJ	4	1.9 [n.s]		
				≤ 20 years	0	0.0 [n.s.]		
				20–25 years	1	1.8 [n.s.]		
				>25 years	3	3.9 [n.s.]		
				Compared to Police	4	1.4 [n.s.]		
			Leukaemia	Compared to NJ	4	1.8 [n.s.]		
				Compared to Police	4	2.8 [P<0.05]		

Table 2.7 (contd)

Reference, location, name of study	Study population description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/ deaths	PMR/SMR/MOR (95% CI)	Adjustement for potential confounders	Comments
Hansen (1990), Denmark	Mortality among 886 men identified as firefighters in the 1970 census followed through 1980 compared to men in similar occupations	Occupation as reported in the Census		Males		**SMR**	Age, calendar period	
			All	Overall	21	1.2 (0.7–1.8)		
				Age 30–49	NR	4.4 (1.4–10.2)		
				Age 50–59	NR	1.0 (0.3–2.3)		
				Age 60–74	NR	1.9 (0.9–3.7)		
			Lung	Overall	9	1.6 (0.8–3.1)		
				Age 30–49	NR	0.0 (0.0–1.5)		
				Age 50–59	NR	1.4 (0.2–4.9)		
				Age 60–74	NR	3.2 (1.2–6.9)		

Table 2.7 (contd)

Reference, location, name of study	Study population description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/ deaths	PMR/SMR/MOR (95% CI)	Adjustement for potential confounders	Comments
Ma *et al.* (1998), 24 states, USA	Analysis of 1984–1993 death certificate data, 6607 firefighters identified	Usual occupation on death certificate		Overall, caucasian males		**MOR**	Age, year of death	
			All		1817	1.1 (1.1–1.2)		
			Lip		3	5.9 (1.9–18.3)		
			Colon		149	1.0 (0.9–1.2)		
			Rectum		27	1.1 (0.8–1.6)		
			Pancreas		88	1.2 (1.0–1.5)		
			Lung		633	1.1 (1.0–1.2)		
			Prostate		189	1.2 (1.0–1.3)		
			Bladder		48	1.2 (0.9–1.6)		
			Kidney & pelvis		49	1.3 (1.0–1.7)		
			Brain & CNS		41	1.0 (0.8–1.4)		
			non-Hodgkin lymphoma		76	1.4 (1.1–1.7)		
			Multiple Myeloma		28	1.1 (0.8–1.6)		
			Leukaemia		60	1.1 (0.8–1.4)		
			Soft tissue sarcoma		14	1.6 (1.0–2.7)		
				Overall, black males				
			All		66	1.2 (0.9–1.5)		
			Nasopharynx		1	7.6 (1.3–46.4)		
			Colon		9	2.1 (1.1–4.0)		
			Pancreas		5	2.0 (0.9–4.6)		
			Lung		15	0.8 (0.5–1.3)		
			Prostate		16	1.9 (1.2–3.2)		
			Brain & CNS		5	6.9 (3.0–16.0)		

* specify *P* value if no confidence interval indicated; MOR, mortality odds ratio; NJ, New Jersey; NR, not reported; n.s, not significant; PMR, proportionate mortality ratio; SMR, standardized mortality ratio

Hansen (1990) performed a study of Danish firefighters employed at the time of the 1970 national census. An analysis was then conducted of 57 deaths (21 from cancer) during 1970–1980 occurring among 886 males who had reported employment as firefighter. Men employed in similar occupations were used as the reference group, and an excess of lung cancer among firefighters over the age of 60 was reported, based on small numbers.

Ma *et al.* (1998) conducted a further analysis of a data set collected by Burnett *et al.* (1994) with additional years of follow-up using 1984–1993 death certificate data from 24 states in the USA. A total of 6607 deaths and 1883 cancer deaths among firefighters were identified based on the occupational titles on death certificates. Race-specific cancer mortality odds ratios (MORs) were calculated with all non-cancer deaths as the reference group. Analyses were adjusted for age and year of death. Among caucasian male firefighters, significant excesses were observed for cancers of the lip, pancreas, lung, prostate, kidney, and soft-tissue sarcoma and non-Hodgkin lymphoma. Among black male firefighters, significant excesses were observed for cancers of the nasopharynx, colon, prostate, and brain.

2.3.2 *Descriptive studies with firefighter results.*

There is a large body of descriptive epidemiology carried out for the purpose of occupational cancer and mortality surveillance. The results of these studies are summarized in Table 2.8.

Berg & Howell (1975) examined the risk of colorectal cancer by occupation using death certificate data from the USA and the United Kingdom and observed an excess among firefighters. [The Working Group noted that there was an overlap between the United Kingdom data included in this study and the meta-analysis by Dubrow & Wegman, 1983].

Williams *et al.* (1977) observed excesses of oral cancer, lung cancer, bladder cancer, and non-Hodgkin lymphoma based on the small number of cancers among firefighters that were included in the Third National Cancer Survey, USA. [The Working Group noted that Williams *et al.* (1977) was included in the meta-analysis conducted by Dubrow & Wegman (1983), but was unique in that occupation was ascertained by interview.]

Dubrow & Wegman (1983) summarized the results of ten early USA and United Kingdom studies and reported the results that appeared to be most consistent between the studies. Among those studies that reported results for firefighters, large intestine cancer and multiple myeloma were significantly elevated.

Morton & Marjanovic (1984) examined the incidence of leukaemia by occupation in the Portland–Vancouver metropolitan area in North-western USA, and excesses were observed among firefighters based on very small numbers.

Mortality among a cohort of 293 958 United States military veterans was examined by occupation and industry (Blair *et al.,* 1985). Usual occupation and industry as well as smoking information was determined from questionnaires

completed in 1954 and 1957, and 107 563 deaths were recorded during 1954–1970. Excesses of rectal, bladder, and brain cancers were observed based on very small numbers.

Gallagher *et al.* (1989) conducted a study of mortality by occupation and industry using death certificate data during 1950–1984 from the Canadian province of British Columbia. There were 1202 deaths among firefighters identified based on occupational titles on death certificates. PMRs were calculated with adjustment for 5-year age and calendar period. There were 197 cancer deaths, and a small excess of overall cancer as well as a significant excess of pancreatic cancer was observed.

In the USA, Sama *et al.* (1990) examined cancer incidence among firefighters using the Massachusetts Cancer Registry records for 1982–1986. Employment as a firefighter was based on the usual occupation reported to the Registry. The analysis was restricted to 315 Caucasian male firefighters. Case–control analyses were conducted for nine different cancer types and two 'unexposed' reference populations were used: policemen and statewide males. Standardized morbidity odds ratios (SMORs) were calculated and significant excesses of malignant melanoma and bladder cancer were observed compared to the general population. Excesses of bladder cancer and non-Hodgkin lymphoma were observed when compared to policemen.

An analysis of deaths in England and Wales (1979–1980 and 1982–1990) were examined by occupation (OPCS, 1995). A total of 2968 deaths among male firefighters and 16 deaths among their female counterparts were observed based on the last occupation listed on death certificates. Only statistically significant results were reported, and excesses of oesophageal, stomach, and gall bladder cancer mortality were observed among men.

A follow-up study was conducted in the Finnish working-age population identified in the 1970 census (Pukkala, 1995). A total of 1436 male firefighters were identified during the follow-up period during 1971–1985 through linkage with the Finnish tumour registry. No statistically significant excesses were observed. The largest excess reported was for non-localized prostate cancer.

In Canada, Finkelstein (1995) examined occupations associated with lung cancer using a case–control study based on death certificates in two Ontario cities, and observed an excess among firefighters based on small numbers.

Milham (1997) conducted a study of mortality by occupation and industry using death certificate data (1950–1989) from the state of Washington, USA. A total of 2266 deaths among firefighters were identified based on the occupational titles on death certificates. PMRs were calculated and adjusted by 5-year age group and calendar period. There were 197 cancer deaths and a small excess of overall cancer was observed as well as significant excesses of melanoma and lympho- and reticulosarcoma. [The Working Group noted that there was an overlap between this and the multistate studies conducted by NIOSH, but that this had the longest follow-up period and was the earliest study of its kind in North America.]

Table 2.8. Descriptive studies with results on firefighters

Reference, location, name of study	Study population description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/ deaths	RR/SIR/SMR (95% CI)*	Adjustement for potential confounders	Comments
Berg & Howell (1975), USA & UK	US males aged 20–44, who died in 1950, and UK male deaths, 1949–1953 and 1959–1963	Occupation on death certificate	Colorectum	Overall	39	**PMR** 1.72 **SMR** 2.79	Age, calendar period	Overlap with Dubrow & Wegman, (1983)
Williams *et al.* (1977), USA	34 male firefighters with incident cancer included in the Third National Cancer Survey, 1969–1971	Occupation from interview	Oral cavity	Overall, male	4	**OR** 2.44 [n.s.]	Age, race, education, smoking, alcohol	Overlap with Dubrow & Wegman, (1983)
			Colon		4	0.80 [n.s.]		
			Lung		8	1.78 [n.s.]		
			Prostate		5	0.90 [n.s.]		
			Bladder		4	2.72 [n.s.]		
			Lymphosarcoma		2	15.30 [n.s.]		
			Other, non-Hodgkin lymphoma		1	3.39 [n.s.]		

Table 2.8 (contd)

Reference, location, name of study	Study population description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/ deaths	RR/SIR/SMR (95% CI)*	Adjustement for potential confounders	Comments
Dubrow & Wegman (1983), USA & UK	Meta analysis of 10 surveillance studies	Varied	Large intestine (excl. rectum) Multiple myeloma	Overall, summary result from 5 studies reporting Overall, summary result from 3 studies reporting	 70 11	**Summary RR** 1.3 (P<0.05) 2.0 (P<0.05)	Age, at least	Studies included in the meta-analysis Milham (1976), Petersen & Milham (1980), OPCS (1978), Guralnick (1963), Williams *et al.* (1977), Decoufle *et al.* (1977), Gute (1981), OPCS (1971, 1972,1975), Dubrow & Wegman (1984)
Morton & Marjanovic (1984), Portland–Vancouver, USA	1678 leukaemia cases aged 16–67 from the records of 24 hospitals and death certificates, 1963–1977	Occupation abstracted from hospital records and death certificates	Leukaemia Lymphatic Non-lymphatic	Firefighters Overall leukaemia Lymphatic Non-lymphatic	 4 1 3	**SIR** 3.5 (P<0.01) 2.1 [n.s.] 4.5 (P<0.01)	Age	1970 Census data used for reference

Table 2.8 (contd)

Reference, location, name of study	Study population description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/ deaths	RR/SIR/SMR (95% CI)*	Adjustement for potential confounders	Comments
Blair *et al.*, (1985); Walrath *et al.* (1985); USA	Follow–up (1954–1970) of 902 USA Veterans reporting occupation as firefighter	Usual occupation from interview		Overall, Male		**SMR**	Age, calendar period, smoking	
			Intestine		8	1.4 [n.s.]		
			Lung, bronchus		13	1.1 [n.s.]		
Gallagher *et al.* (1989), British Columbia, Canada	Death certificate study 1950–1984. 1202 firefighter deaths	Usual occupation on death certificate				**PMR**	Age, calendar period	
			All		197	1.2 (1.0–1.3)		
			Colon		20	1.4 (0.8–2.1)		
			Rectum		10	1.2 (0.6–2.2)		
			Pancreas		19	1.7 (1.1–2.7)		
			Lung		50	1.0 (0.8–1.4)		
			Prostate		23	1.4 (0.9–2.1)		
			Bladder		9	1.5 (0.7–2.9)		
			Kidney		3	0.7 (0.1–2.1)		
			Brain		6	1.2 (0.4–2.7)		
			Non-Hodgkin lymphoma		7	1.5 (0.6–3.2)		
			Multiple myeloma		2	0.8 (0.1–2.9)		
			Leukaemia		8	1.3 (0.5–2.5)		

Table 2.8 (contd)

Reference, location, name of study	Study population description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/ deaths	RR/SIR/SMR (95% CI)*	Adjustement for potential confounders	Comments
Hrubec *et al.* (1992) USA	Follow-up 1954–80 of 902 USA veterans reporting occupation as a firefighter	Usual occupation on death certificate		Occupation as a firefighter		**RR (90% CI)**	Age, calendar period	
			All cancer		110	1.2 (1.1–1.4)		
			Rectum		7	2.2 (1.2–4.2)		
			Prostate		12	1.1 (0.7–1.7)		
			Bladder		8	2.1 (1.2–3.8)		
			Brain		5	2.3 (1.1–4.9)		
			Malignant lymphoma		2	0.4 (NR)		
			Leukaemia		3	0.7 (NR)		

Table 2.8 (contd)

Reference, location, name of study	Study population description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/ deaths	RR/SIR/SMR (95% CI)*	Adjustement for potential confounders	Comments
Sama *et al.* (1990) Massachusetts, USA	315 cases of cancer identified from the Massachusettes Tumor Registry between 1982–86 Two reference groups of unexposed cases; state population and police; aged 18 and older at the time of diagnosis	Usual occupation from tumour registry records		Caucasian males		**SMOR**	Age, smoking race	
			Colon	Overall, compared to state population	33	1.2 (0.8–1.8)		
				Overall, compared to Police	33	1.0 (0.6–1.8)		
			Rectum	Overall, compared to state population	22	1.4 (0.8–2.2)		
				Overall, compared to Police	22	1.0 (0.5–1.9)		
			Pancreas	Overall, compared to state population	6	1.0 (0.4–2.3)		
				Overall, compared to Police	6	3.2 (0.7–14.2)		
			Lung	Overall, compared to state population	71	1.2 (0.9–1.7)		
				Overall, compared to Police	71	1.3 (0.8–2.0)		
			Melanoma	Overall, compared to state population	26	2.9 (1.7–5.0)		
				Overall, compared to Police	18	1.4 (0.6–3.2)		
			Bladder	Overall, compared to state population	26	1.6 (1.0–2.5)		
				Overall, compared to Police	26	2.1 (1.1–4.1)		
			Brain & other nervous system	Overall, compared to state population	5	0.9 (0.3–2.2)		
				Overall, compared to Police	5	1.5 (0.4–5.9)		

Table 2.8 (contd)

Reference, location, name of study	Study population description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/ deaths	RR/SIR/SMR (95% CI)*	Adjustement for potential confounders	Comments
Sama *et al.* (1990) (contd)			Non-Hodgkin lymphoma	Overall, compared to state population	14	1.6 (0.9–2.8)		
				Overall, compared to Police	14	3.3 (1.2–9.0)		
			Leukaemia	Overall, compared to state population	6	1.1 (0.5–2.6)		
				Overall, compared to Police	6	2.7 (0.6–11.5)		
OPCS (1995), England and Wales UK	Death certificate study 1979–1980, 1982–1990. 2698 deaths among men and 16 deaths among women	Last occupation on death certificate				**PMR**	Age	Only statistically significant results reported
			All cancers	Men	432	1.16		
			Oesophagus		46	1.4 (1.0–1.8)		
			Stomach		91	1.5 (1.2–1.8)		
			Gallbladder		10	2.2 (1.1–4.1)		
			All cancers	Women	2	1.07		
Finkelstein (1995), Ontario, Canada	Population-based case–control study using death certificate data between 1979–1988 Males age 45–75 years	Usual occupation on death certificate	Lung	Male firefighters	6	**RR** 1.9 (0.6–6.3)	Age, year of death, city of residence	

Table 2.8 (contd)

Reference, location, name of study	Study population description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/ deaths	RR/SIR/SMR (95% CI)*	Adjustement for potential confounders	Comments
Milham (1997), Washington State, USA	Death certificate study 1950–1989. 2266 firefighter deaths	Usual occupation on death certificate				**PMR**	Age, Calendar period	
			All Cancers		476	1.1 (1.0–1.2)		
			Buccal cavity & pharynx		7	0.6 (0.3–1.3)		
			Oesophagus		11	1.1 (0.6–2.0)		
			Stomach		22	0.8 (0.5–1.2)		
			Colon		36	0.9 (0.6–1.2)		
			Rectum		15	1.1 (0.6–1.8)		
			Pancreas		28	1.1 (0.7–1.6)		
			Larynx		3	0.6 (0.1–1.8)		
			Lung		120	1.0 (0.8–1.2)		
			Prostate		56	1.1 (0.8–1.5)		
			Kidney		9	0.9 (0.4–1.6)		
			Bladder & urinary		23	1.4 (0.9–2.1)		
			Melanoma		9	2.1 (1.0–4.1)		
			Brain & nervous system		19	1.6 (0.9–2.4)		
			Lympho- & reticulosarcoma		13	1.8 (1.0–3.0)		
			Hodgkin lymphoma		7	1.8 (0.7–3.7)		
			Other lymphoma		3	0.5 (0.1–1.4)		
			Multiple myeloma		9	1.3 (0.6–2.4)		
			Leukaemia		27	1.4 (0.9–2.1)		

* specify *P*–value if no confidence interval indicated

NR, not reported; n.s, not significant

2.4 Case reports

Individual firefighters have applied for, and sometimes received, workers' compensation for cancer. An apparent cluster of cancer among firefighters was reported in an investigation of a chemical waste dump fire by NIOSH (Hrubec *et al.,* 1992). However, the authors concluded it was not likely to have been related to firefighting. [Given the limitations of these reports and the large number of descriptive, cohort, and case–control studies with data on firefighters, the Working Group did not believe that case reports would contribute to the evaluation.]

2.5 Meta-analyses

Two meta-analyses of studies of firefighters and cancer have been conducted (Howe & Burch, 1990; LeMasters *et al.,* 2006). The most recent meta-analysis included a great majority of the studies considered by the Working Group (LeMasters *et al.,* 2006). Cancer risk was significantly elevated for ten of the 21 cancer types analysed (stomach, colon, rectum, skin, prostate, testis, brain, non-Hodgkin lymphoma, multiple myeloma, and malignant melanoma). With the exception of testicular cancer (summary RR = 2.02), the summary relative risk estimates were moderate, ranging from 1.21 for colon to 1.53 for multiple myeloma. For four of these sites (prostate, testis, non-Hodgkin lymphoma, and multiple myeloma), findings were consistent across study designs and the types of study available. However, since that analysis, two additional large studies of cancer in firefighters had been published (Ma *et al.,* 2006; Bates, 2007). Therefore, another meta-analysis was performed by the Working Group to assess the impact of these recent studies.

Inclusion criteria for studies in this meta-analysis were reported estimates of relative risk with corresponding 95% confidence intervals or information that allowed their computation by the Working Group for 'ever' versus 'never' exposure to firefighting or employment as a firefighter. For those studies that did not report for this category, the relative risks and 95% confidence intervals were estimated by the Working Group from strata-specific relative risk and corresponding number of cases, assuming a normal distribution when possible. Studies that only reported point estimates without confidence intervals were not included. Proportionate mortality studies were also excluded. Statistical heterogeneity among studies was tested with the Q statistic. Summary relative risk estimates were obtained from random-effect models for prostate cancer ($Q = 32.816$, $P = 0.005$), and fixed-effect models for testicular cancer ($Q = 3.928$, $P = 0.560$), and non-Hodgkin lymphoma ($Q = 6.469$, $P = 0.486$). All statistical analyses were performed using STATA (version 9.0; StataCorp, College Station, TX).

Based on the Working Group's meta-analysis, three of the four sites remained statistically significant. Testicular cancer was evaluated based on six studies and

409 cases (Giles *et al.,* 1993; Aronson *et al.,* 1994; Bates *et al.,* 2001; Stang *et al.,* 2003; Ma *et al.,* 2006; and Bates, 2007). The results demonstrated an approximate 50% increased risk (1.47, 95% CI: 1.20–1.80, fixed effects). Prostate cancer was evaluated using 16 available studies and 1764 cases (Aronson *et al.,* 1994; Baris *et al.,* 2001; Bates *et al.,* 2001 ; Bates, 2007; Beaumont *et al.,* 1991; Demers *et al.,* 1994; Firth *et al.,* 1996; Giles *et al.,* 1993; Grimes *et al.,* 1991; Guidotti 1993; Krstev *et al.,* 1998; Ma *et al.,* 1998; Ma *et al.,* 2006; Pukkala, 1995; Tornling *et al.,* 1994; and Vena & Fiedler, 1987). The results showed a 30% elevated risk (1.30; 95% CI: 1.12–1.51, random effects). Non-Hodgkin lymphoma was evaluated based on seven studies and 312 cases, and had a 21% elevated risk estimate (1.21; 95% CI: 1.08–1.36, fixed effects) (Baris *et al.,* 2001; Bates, 2007; Giles *et al.,* 1993; Ma *et al.,* 1998, 2006; Pukkala, 1995; and Sama *et al.,* 1990).

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3. Studies of Cancer in Experimental Animals

No data were available to the Working Group.

4. Mechanistic and Other Relevant Data

4.1 Absorption, distribution, metabolism and excretion

Smoke is a complex mixture of chemicals in aerosol, gas, and vapour forms. The focus of this section of the monograph will be primarily on components of smoke from municipal and wildland fires. There is a paucity of information on the extent of exposure to firefighters from trash fires, vehicle fires, and non-wildfire vegetation fires, during which firefighters typically do not wear respiratory protection. Although not typical of exposures most firefighters encounter, there are published reports on the effects of firefighter exposure to specific incidents, including the World Trade Center fire and collapse, and specific industrial fires or clean-up operations. It should be kept in mind that the magnitude of these exposures are not representative of most fires.

Information on many of the specific chemicals found in smoke is available in previous IARC monographs. The data on absorption, distribution, metabolism, and excretion for select carcinogens contained in fire smoke are listed in Table 4.1. Only inhalation and dermal exposures were considered – the predominant occupational exposure routes in firefighters. One of the difficulties in evaluating the toxicokinetics and metabolism of combustion products in firefighters is the adsorption of chemical components onto particles (Fine *et al*., 2001). This will alter the absorption kinetics of these combustion products and may also cause a proportionally greater effect in the lungs compared to other tissues. Depending on their volatility, these chemicals may also exist at significant concentrations in the gas phase of smoke exposure as well. No chronic toxicity studies could be found on non-human exposure to combustion products from structural materials. Due to limited data, the toxicokinetics of chemical mixtures are not considered in this monograph, although they are likely to be of significant importance given the multiplicity of chemicals in smoke.

4.1.1 *Particles*

Particle deposition depends on the size and shape of the particle. Smoke from combustion of products such as wood tends to produce small particles that can easily reach the alveolar region of the lung, with a mode size distribution of 0.1–0.2 μm diameter (Kleeman *et al*., 1999). Particles not cleared by phagocytosis and transferred to the mucociliary escalator may be translocated to the interstitial tissue and to lung-associated lymph nodes (International Commission on Radiological Protection, 1994). This local

distribution of particles is consistent with the increased rate of lung cancer seen in rats exposed to carbon black (IARC, 1996). Chemicals adsorbed onto particles can be transported deep into the lung where depending on their solubility, they can either remain or slowly desorb into the lung-lining fluid.

Impaired particle clearance due to high loading of carbon black in experiments with rats results in increased accumulation of particles and chronic active inflammation. Increased collagen deposition from proliferating fibroblasts, increased epithelial cell proliferation, and metaplasia have been found at high lung burdens of carbon black. Most assays for mutagenicity are negative for carbon black. However, in rats exposed to carbon black by inhalation, *hprt* mutant frequency was elevated in type II cells following a 12-week exposure. Studies on DNA adducts are mixed with prolonged inhalation exposure not inducing a significant increase in DNA adducts in peripheral lung tissue of rats, but increasing DNA adduct levels in type II cells (IARC, 1996).

A specific exposure in firefighters consisting of mixed particulate and gas or vapour phase components is diesel exhaust, which shares many chemicals in common with wood smoke, including PAHs. Prolonged exposure of experimental animals to diesel engine exhaust leads to particle accumulation in macrophages, changes in the lung cell population, fibrotic effects, squamous metaplasia, and pathological changes in regional lymph nodes, as well as DNA adduct formation, protein adduct formation, and sister chromatid exchange. Particles or their extracts induce mutations and DNA damage in bacteria, and the gaseous phase is also mutagenic to bacteria (IARC, 1996).

In rats, a small fraction of ultrafine particles are translocated from the lungs into other organs (Kreyling *et al*., 2002). In humans, studies of ultrafine 99mTc-labelled carbon particles also support translocation of the particles from the lung into the systemic circulation (Nemmar *et al*., 2002). Translocation of ultrafine carbon particles from the olfactory mucosa to the brain has also been described *in vivo* (Oberdörster *et al*., 2004).

4.1.2 *Aldehydes*

Multiple aldehydes are found in smoke, including but not limited to formaldehyde, acetaldehyde and acrolein. For all of these aldehydes, exposure is predominantly to the respiratory tract due to local metabolism. For formaldehyde, this local exposure is consistent with the human cancer data linking exposure to nasopharyngeal and sinonasal cancer (IARC, 2006). Due to this local metabolism and significant endogenous production of formaldehyde, exposure of humans, monkeys or rats to formaldehyde by inhalation has not been found to alter endogenous concentrations. No information is available on relative absorption by site within the respiratory system in humans. In monkeys, formaldehyde is absorbed in the nasopharynx, trachea and proximal regions of the major bronchi whereas in rats absorption occurs almost entirely in the nasal passages (IARC, 2006). Dermal application of formaldehyde results in a relatively low extent of absorption, so in firefighters the predominant absorption route should be through inhalation.

Table 4.1. Toxicokinetics and metabolism for selected carcinogenic products of structural and wildfire smoke

Chemical	Absorption	Distribution	Metabolism	Excretion	Mechanism	Cancer	Note/Reference
Particles	Inhalation (variable depending on size)	Lungs	Dependent on solubility of adsorbed chemicals	Macrophage phagocytosis followed by migration to mucociliary escalator or transport to interstitium	Inflammation	For carbon black, lung, lymphatic cancer (in presence of PAHs). For diesel exhaust, lung and bladder cancer, possibly non-Hodgkin lymphoma, multiple myeloma, and prostate cancer	IARC (2010c); Oberdörster (1992); Boffetta & Silverman (2001); Lipsett & Campleman (1999); McDuffie *et al*. (2002); Boffetta *et al*. (1988); Lee *et al*. (2003); Hansen (1993); Seidler *et al*. (1998)
Acetaldehyde	Inhalation (45–70%)	Predominantly peripheral blood	Acetic acid	Blood half life 3.1 min (rat)	DNA damage including acetaldehyde–DNA adducts	Nasal cancer	IARC (1999); Egle (1970); Hobara *et al*. (1985); Hardman *et al*. (1996)
Acrolein	Inhalation (81–84%)	Predominantly local	Conjugated rapidly with thiols	Inadequate data in humans, urinary *S*-carboxyethyl-mercapturic acid following oral exposure in rats	DNA damage in cultured mammalian cells	IARC Group 3, urinary bladder papillomas in rats	IARC (1995); Egle (1972); ATSDR (2005)

Table 4.1 (contd)

Chemical	Absorption	Distribution	Metabolism	Excretion	Mechanism	Cancer	Note/Reference
Benzene	Inhalation (20–80%), dermal (<1%)	Preferably to fat, bone marrow and urine	Metabolism predominantly in liver and bone marrow. Reactive metabolites are considered carcinogenic.	Elimination half-life 42 min to 1.2 h	Benzene metabolites hydroquinone and 1,4-benzo-quinone inhibit topoisomerase II and micro-tubule function, induce oxidative stress, and damage DNA	Leukaemia	IARC (1987); Srbova *et al*. (1950); Franz (1984), Schrenk *et al*. (1941); Irons *et al*. (1980); Sherwood (1988)
1,3-Butadiene	Inhalation 43.4–45.6%	Widely distributed throughout the body	Urinary metabolites 1,2-dihydroxylbutyl mercapturic acid and monohydroxy-3-butenyl mercapturic acid	Elimination half life 2–10 h for elimination radiolabel of C^{14} 1,3-butadiene	Induces DNA adducts and damage and activates oncogenes	Lympho-haematopoietic	IARC (2008); Lin *et al*. (2002); Evelo *et al*. (1993); ATSDR (1992a)

Table 4.1 (contd)

Chemical	Absorption	Distribution	Metabolism	Excretion	Mechanism	Cancer	Note/Reference
Formaldehyde	Inhalation (100%) Dermal (3.4% in rats)	Predominantly local before metabolism	Metabolism in all tissues to carbon dioxide, formate, other one-carbon molecules	Plasma half-life 1 min (rat)	DNA–protein crosslinking, chromosomal abberations, and cell proliferation. Gene mutations Sister chromatid exchange	Nasopharyngeal and sinonasal cancer, leukaemia	IARC (2006); Egle 1972); Bartnik *et al*. (1985); Heck *et al*. (1982, 1983)
PAHs	Dermal (20% for pyrene) > inhalation	Following dermal exposure, highest concentrations in liver, kidney, fat, and lung	Metabolism in all tissues. 1-hydroxy-pyrene used as proxy for overall exposure	Elimination half-life (dermal exposure) 30 h for benzo[*a*]pyrene	Metabolites PAH oxides and diol epoxides form stable DNA adducts and induce mutations. Other mechanisms also postulated	Lung, bladder, skin, possibly prostate	IARC (2010a); Van Rooij *et al*. (1993); Withey *et al*. (1993); ATSDR (2007); Sanders *et al*. (1986); Rybicki *et al*. (2006); Seidler *et al*. (1998)

Table 4.1 (contd)

Chemical	Absorption	Distribution	Metabolism	Excretion	Mechanism	Cancer	Note/Reference
PCBs	Inhalation Dermal (variable depending on solvent)	Highest concentration in adipose tissue	Liver metabolism	Elimination half-life (occupational exposure) 1–24 yrs	Covalent modification of proteins and DNA, possible increased cell proliferation following injury caused by reactive oxygen species	Liver and biliary tract	IARC (1987); Fait *et al*. (1989); Fitzgerald *et al*. (1986); Wester *et al*. (1983, 1990); Jensen (1987); Wolff *et al*. (1992)
Styrene	Inhalation 60–70%	Wide distribution with highest concentration in adipose tissue	Liver metabolism	Blood elimination half-life biphasic with rapid phase (0.58 h) and slow phase (13.0 h). Predominant excretion as urinary metabolites, 0.7–4.4% exhaled unchanged	Protein and DNA adducts, other genotoxicity	Lympho-haematopoietic	IARC (2002); ATSDR (1992b)

Table 4.1 (contd)

Chemical	Absorption	Distribution	Metabolism	Excretion	Mechanism	Cancer	Note/Reference
Sulfur dioxide	Inhalation (40–>90% in rabbits)	Large proportion to upper airways	Sulfite and bisulfite in airways	Excreted in urine as sulfate	Conflicting results seen in human studies	Lung cancer	IARC (1992); Strandberg (1964); Balchum *et al*. (1959); Gunnison *et al*. (1987); Yokoyama *et al*. (1971)
Sulfuric acid	Inhalation (50–87%)	Predominantly upper airways	Converted to sulfate before absorption into blood	Excess sulfate excreted in the urine	DNA damage	Laryngeal, nasal sinus, lung	IARC (1992); Amdur *et al*. (1952); Dahl *et al*. (1983); Vander *et al*. (1975)

h, hour; min, minute; PAH, polycyclic aromatic hydrocarbon; PCB, polychlorinated biphenyl; yrs, years

For acetaldehyde, inhalation exposure leads to degeneration of nasal epithelium followed by hyperplasia and proliferation in rats (IARC, 1999). For acrolein, repeated inhalation results in changes in bronchiolar epithelial cells and emphysema in dogs (IARC, 1995). Dermal absorption does not appear to be important for acetaldehyde and acrolein.

Formaldehyde exposure results in DNA–protein cross-links and chromosomal abberations. Cell proliferation, which appears to amplify the genotoxic effects of formaldehyde, is increased at concentrations of around 6 ppm. No clear mechanism has been identified for the induction of myeloid leukemia in humans (IARC, 2006). Acetaldehyde causes gene mutations in bacteria; gene mutations, sister chromatid exchanges, micronuclei and aneuploidy in cultured mammalian cells; DNA damage in cultured mammalian cells and in mice in vivo. Acetaldehyde–DNA adducts have been found in white blood cells from human alcohol abusers (IARC, 1999). Acrolein induces gene mutation, sister chromatid exchange and DNA damage in cultured mammalian cells, but reportedly does not induce DNA damage in rats or dominant lethal mutations in mice treated in vivo (IARC, 1995).

4.1.3 *Benzene*

Benzene is systemically absorbed following inhalation, and due to rapid evaporation, dermal exposure should not be a significant source of systemic dose for firefighters. Benzene is oxidized primarily by CYP2E1 to benzene oxide, which exists in equilibrium with its tautomer oxepin (Kim *et al*., 2006; 2007). Spontaneous rearrangement of benzene oxide produces phenol that is either excreted or oxidized by CYPs to hydroquinone, which is excreted or oxidized by myeloperoxidase in the bone marrow to 1,4-benzoquinone. Conversely, NAD(P)H quinone oxidoreductase 1 transforms 1,4-benzoquinone to hydroquinone. Hydroquinone and 1,4-benzoquinone are thought to influence the toxic effects of benzene through their ability to inhibit topoisomerase II and microtubule function, induce oxidative stress, and damage DNA. Other major metabolites include catechol, representing the pathway involving the hydrolysis of benzene oxide by epoxide hydrolases, and *trans,trans*-muconic acid, representing the pathway involving oxidation of oxepin and ring opening. Reaction between benzene oxide and glutathione, possibly mediated by glutathione-*S*-transferases (GSTM1, GSTT1), can produce the minor metabolite *S*-phenylmercapturic acid (Kim *et al*., 2006; 2007). Although it is widely accepted that benzene toxicity is dependent upon metabolism, no single benzene metabolite has been found to be the major source of benzene haematopoietic and leukemogenic effects (ATSDR 2005). At low exposure levels, benzene is rapidly metabolized and excreted predominantly as conjugated urinary metabolites. The metabolism of benzene in the bone marrow is consistent with the increase in haematopoietic cancers seen in humans (ATSDR, 2005).

Chromosomal aberrations in human peripheral lymphocytes have been associated with occupational exposure to benzene and include hypo- and hyperdiploidy, deletions, breaks, and gaps (ATSDR, 2005). Sister chromatid exchange was not found to be a significant effect of benzene exposure in humans. In-vivo animal studies provide convincing evidence of the genotoxicity of benzene. Benzene induced chromosomal aberrations, micronuclei and

sister chromatid exchanges in bone-marrow cells of mice, chromosomal aberrations in bone-marrow cells of rats and Chinese hamsters and sperm-head anomalies in mice treated *in vivo*. It induced chromosomal aberrations and mutation in human cells *in vitro* (IARC, 1987). In-vitro studies strongly imply that the genotoxicity of benzene is derived primarily from its metabolites hydroquinone and 1,4-benzoquinone through their ability to inhibit topoisomerase II and microtubule function, induce oxidative stress, and damage DNA (ATSDR 2005).

4.1.4 *1,3-Butadiene*

Butadiene is absorbed through inhalation and is systemically distributed. It is metabolized primarily by CYP2E1 and CYP2A6 (Evelo *et al*., 1993). The metabolic rate in lung is greater at lower doses, and in liver, at higher doses. The butadiene metabolite epoxy-1,2-butanediol is reportedly the major electrophile binding with DNA and haemoglobin (Swenberg *et al*., 2001). Adducts formed by reaction of the metabolites 1,2-epoxy-3-butene and 3,4-epoxy-1,2-butanediol with haemoglobin and urinary mercapturic acids derived from 1,2-epoxy-3-butene have been detected in workers exposed to 1,3-butadiene. There is considerable interindividual variability in the ability of human liver microsomes to metabolize 1,3-butadiene and 1,2-epoxy-3-butene *in vitro* (Swenberg *et al*., 2001).

There are conflicting results on whether 1,3-butadiene increases *HPRT* mutations in lymphocytes from humans exposed to 1,3-butadiene compared with unexposed controls (IARC, 1999). One study of workers exposed to 1,3-butadiene demonstrated an increase in *HPRT* variant frequency in lymphocytes with high (mean 1.48 ppm) as compared with low (mean 0.15 ppm) exposures (Ammenheuser *et al*., 2001). However, sister chromatid exchanges, micronuclei, chromosomal aberrations and DNA-strand breaks were not significantly elevated above control levels in peripheral blood lymphocytes of occupationally exposed workers. 1,3-Butadiene induces DNA adducts and damage in both mice and rats *in vivo* and is mutagenic in virtually all in-vitro and in-vivo test systems. Activated K-*ras* oncogenes have been detected in lymphomas and in liver and lung tumours induced in mice by 1,3-butadiene. Mutations in the *p53* tumour suppressor gene have been detected in mouse lymphomas (IARC, 1999).

4.1.5 *Free radicals*

Smoke contains highly reactive oxygen- and carbon-centred radicals, which may initiate cancer through the oxidative activation of a procarcinogen and/or through binding of the radical to DNA. The major effect should therefore be on the epithelial layer of the respiratory tract. Cigarette smoke, which has been better studied than wood smoke, contains a quinone–hydroquinone–semiquinone system that can reduce oxygen to produce superoxide, and hence, hydrogen peroxide and the hydroxyl radical, as well as penetrate viable cells, bind to DNA, and cause nicks (Pryor, 1997; Church & Pryor, 1985).

4.1.6 *Furan*

Furan is rapidly and extensively absorbed after oral administration. Part of the absorbed dose becomes covalently bound to protein, mainly in the liver, although DNA binding has not been demonstrated in this organ. Repeated administration of furan to mice and rats leads to liver necrosis, liver-cell proliferation and bile-duct hyperplasia. In rats, prominent cholangiofibrosis develops. The induction of chromosomal aberrations but not sister chromatid exchange has been observed in rodents treated *in vivo*. Gene mutation, sister chromatid exchange and chromosomal aberrations are induced in rodent cells *in vitro* (IARC, 1995).

4.1.7 *PAHs*

PAHs are a diverse set of chemicals, and their toxicokinetics vary accordingly. PAHs generally occur as complex mixtures and not as single compounds. The percutaneous absorption of PAHs appears to be rapid for both humans and animals, but the extent of absorption is variable among the different compounds. Although distribution through the circulatory system is widespread, slow absorption through most epithelia results in higher levels of enzymes that activate PAH substrates at the site of entry. This uneven distribution of dose may contribute to the propensity of PAHs acting as carcinogens at the sites where they enter the body (IARC, 2010a). Metabolic activation of PAHs occurs primarily in the liver, but also in many other tissues, including the epithelial barriers. Metabolites include epoxide intermediates, dihydrodiols, phenols and quinones, which can be conjugated to glucuronides, sulfate esters and/or glutathione. Specific cytochrome P450 isozymes and epoxide hydrolase can form reactive diol epoxides. The major cytochrome P450s that are involved in the formation of diol epoxides are CYP1A1, CYP1A2, and CYP1B1, while CYP2C9 and CPY3A4 play a minor role in the activation of PAHs. Additional enzymes that may play a role in the further activation of some PAH diols include members of the aldo-keto reductase family. NAD(P)H quinone oxidoreductase 1 catalyses the reduction of PAH quinones to hydroquinones which may be re-oxidized and generate reactive oxygen species. The major phase II enzymes include the glutathione *S*-transferases (GSTs), uridine 5′-diphosphate glucuronosyltransferases, and sulfotransferases. The major GSTs involved in the conjugation of PAH metabolites are GSTM1, GSTP1, and GSTT1. Quantitative data on the excretion of PAHs in humans are lacking. In general, the major elimination route of PAHs in animals following inhalation exposure is through the faeces. PAHs are eliminated to a large extent within 2 days following low- and high-level oral exposure in rats. Following dermal exposure, the elimination of PAHs occurs rapidly in the urine and faeces of rodents (IARC, 2010a).

The current understanding of the carcinogenesis of PAHs in experimental animals is almost solely based on two complementary mechanisms: those of the diol epoxide, and the radical cation. The diol epoxide mechanism features a sequence of metabolic transformations of PAHs, each of which leads to potentially reactive genotoxic forms. In general, PAHs are converted to oxides and dihydrodiols, which are in turn oxidized to diol

epoxides. Both oxides and diol epoxides are ultimate DNA-reactive metabolites. PAH oxides and diol epoxides can form stable DNA adducts and induce mutations (e.g. in *ras* proto-oncogenes) that are strongly associated with the tumorigenic process (IARC, 2010a). Measured end-points in human populations include mutagenicity in urine and the presence of aromatic DNA adducts in the peripheral lymphocytes of exposed workers. Cytogenetic effects such as micronucleus formation have also been reported. Other mechanisms of carcinogenesis have been proposed for PAHs, but these are less well developed and include generation of reactive oxygen species, activation of the aryl hydrocarbon receptor with regulation of phase I and II metabolism, lipid peroxidation, production of arachidonic acid-reactive metabolites, decreased levels of serum thyroxine and vitamin A and persistent activation of the thyroid hormone receptor, as well as activation of mitogen-activated protein kinase pathways, suppression of immunity by p53-dependent and other pathways (IARC, 2010a).

4.1.8 *PCBs*

PCBs are absorbed by inhalation and dermal contact. Wester *et al*. (1990) found that PCBs penetrated skin in a time-dependent manner, but that 93±7% of PCBs were removed from the skin with five successive washes using soap and water, the removal efficiency decreasing with increasing time from initial contact. Washing 24 hours following skin contact removed only 25% of the initial PCBs. The highest concentrations are found in adipose tissue, with metabolism occurring in the liver. PCBs are metabolized by cytochrome P450 followed by conjugation with glutathione or glucuronic acid. The rate of metabolism depends on the extent of chlorination, the location of the chlorine atoms, and the levels of P450 isozymes and other enzymes. Metabolites of PCBs with low chlorine content are predominately eliminated in the urine. PCBs with high chlorine content and substitution patterns resistant to metabolism are either retained or excreted unchanged in the faeces. In one study, following exposure to an electrical transformer fire in New York, USA, serum PCBs were higher in firefighters initially when compared to levels 9 months later (Kelly *et al*., 2002). Orris *et al*. (1986) reported two cases of lesions consistent with chloracne in firefighters, although in both cases the blood PCB level was less than 10 μg/L.

A wide variety of cancers have been reported in association with PCB exposure. Exposure to PCB mixtures predominantly causes liver tumours in rats; although tumours in mouse lung and mouse skin have also been observed (IARC, 1987). Cancer mechanisms that are both dependent on and independent of the aryl hydrocarbon receptor may be involved. PCBs may be involved in tumour initiation and promotion. Metabolism of less chlorinated PCBs in rat microsomes can lead to covalently modified macromolecules including proteins and DNA, although PCB mixtures generally are inactive as mutagens, and are not potent genotoxicants. PCB promotion of liver tumours may involve increased cell proliferation following cell or tissue injury caused by reactive oxygen species, resulting from induction of CYP oxygenases and GSTs, decreased activity of glutathione

peroxidases, and/or disruption of calcium homeostatic processes and signal transduction pathways (ATSDR 2000).

4.1.9 *Styrene*

Styrene is absorbed by inhalation and dermal contact. In humans, 60–70% of inhaled styrene is absorbed. It is rapidly distributed throughout the body in treated rats. A large percentage of absorbed styrene is excreted as urinary mandelic and phenylglyoxylic acids, with glutathione conjugates forming a minor fraction of the metabolites. The dominant first metabolite is styrene-7,8-oxide, the formation of which appears to be catalysed in humans principally by CYP2B6 but also by CYP2E1 and CYP1A2. Isolated erythrocytes are also capable of non-enzymatic conversion of styrene to styrene-7,8-oxide (IARC, 1994).

Exposure to styrene leads to the formation of both protein and DNA adducts in man, rat and mouse. The levels of the *N*-terminal valine adduct of haemoglobin, *N*-(1-hydroxy-2-phenylethyl)valine, have been found to be four times higher in styrene-exposed workers than in controls, and the levels of the DNA adduct, *O*6-(2-hydroxy-1-phenylethyl)-2′-deoxyguanosine-3′-monophosphate, have been found to be about five times higher than in controls. Some 25 studies on chromosomal aberrations, micronuclei and sister chromatid exchange have been performed in workers exposed to styrene in various countries and different industries. These have provided variable results with regard to the association between exposure to styrene and chromosomal damage. Chromosomal aberrations were observed in nine of 22, sister chromatid exchange in three of 12, and micronuclei in three of 11 studies. The frequency of single-strand DNA breakage/alkali-labile sites was increased in workers exposed to styrene at less than 20 ppm (85 mg/m3). Chromosomal aberrations have not been seen in most studies in rodents, while several studies indicate weak induction of sister chromatid exchange in various tissues of rats and mice. Contradictory results have been obtained with regard to the induction of micronuclei in mice. Significant increases have been observed consistently in the frequency of sister chromatid exchange and chromosomal aberrations in human lymphocytes *in vitro*. Most studies did not show mutation in bacteria, although mutation was seen in some studies in the presence of an exogenous metabolic activation system (IARC, 1994).

4.1.10 *Sulfur dioxide and sulfuric acid*

Sulfur dioxide and sulfuric acid are initially converted to sulfite and sulfate respectively, with excretion as sulfate in the urine. Their local effect on the respiratory system is consistent with the increase in respiratory system cancer in humans (sulfuric acid), and animals (sulfur dioxide and sulfuric acid). Conflicting results for the induction of chromosomal aberrations in lymphocytes have been obtained in studies of workers exposed to sulfur dioxide, among other agents. No increase has been reported in the frequency of sister chromatid exchange in lymphocytes of exposed workers. Sulfur dioxide and its aqueous forms do not induce sister chromatid exchange, chromosomal aberrations or micronucleus formation in bone marrow of mice or Chinese hamsters, although sister chromatid exchange and chromosomal aberrations have been induced in human

lymphocytes. Significant increases in the incidences of sister chromatid exchange, micronucleus formation, and chromosomal aberrations in peripheral lymphocytes have been observed in a single study of workers engaged in the manufacture of sulfuric acid. In cultured mammalian cells at pH 6.7 or below, cell transformation, gene mutation, and chromosomal aberrations were induced (IARC, 1992).

4.2 Genetic and related effects

4.2.1 *Humans*

(a) Direct genotoxicity

There are a limited number of studies evaluating genotoxic effects in firefighters (see Table 4.2). In general, these studies did not show evidence of direct genotoxicity, except in the case of atypical exposures.

In the Washington DC area, USA, a study of peripheral blood lymphocytes collected from 43 firefighters selected from two fire stations with frequent firefighting activities demonstrated an increase in sensitivity to mitomycin-C-induced sister chromatid exchange correlated with the number of fires fought in the previous 24 hours, although no difference was seen comparing the firefighters to 40 non-firefighter controls matched by age, gender, and smoking status. Firefighters as a group had a significantly lower level of unstimulated sister chromatid exchange (8.44 per cell) when compared to controls (9.23 per cell, P=0.02), although no significant differences were found when the comparison was limited to those who had not consumed charbroiled food in the last month (Liou *et al*., 1989). No significant association was found between recent firefighting history and the number of sister chromatid exchange. The firefighters did not have a significant increase in benzo[*a*]pyrene diol epoxide–DNA antigenicity when compared to controls (OR: 1.73, 95% CI: 0.60–4.99). However, following stratification by race, the subset of 37 caucasian firefighters had concentrations of detectable benzo[*a*]pyrene diol epoxide–DNA antigenicity that were 3.56-fold (95% CI: 1.04–12.12) higher than caucasian controls (Liou *et al*., 1989).

In a comparison of 53 male firefighters in the Republic of Korea exposed to fire within 5 days of the study compared to 25 that were not, no increase in urinary 8-hydroxy-2′-deoxyguanosine concentration was observed (Hong *et al*., 2000). [The extent of respiratory protection used by the firefighters was not described in the study and the amount of time elapsed between exposure and collection of urine may have limited the ability to discern a difference between groups.]

Ray *et al*., (2005) compared 47 non-smoking firefighters engaged in firefighting for ≥10 years to 40 age-matched non-smoking controls with a comparable alcohol consumption. Micronuclei frequency in firefighters (3.91 ± 0.19 was significantly higher than in controls (1.25 ± 0.12). In addition, the 27 firefighters with ≥ 20 years of service has more micronuclei than (4.43 ± 0.32) the 20 firefighters with < 20 years of service (3.21±0.24, P < 0.05). [The study group was concerned over potential use of smokeless tobacco or other

substances by the firefighters which may have confounded the study results, and for which information was not provided in the article.]

Data are available for atypical firefighter exposures, including a study of 16 German firefighters exposed to *O*-nitroanisole and other chemicals during clean-up activities following an industrial accidental release from a methoxylation plant. These firefighters were in training and, with the exception of one individual, were not yet involved in actual firefighting. The firefighters did not use respiratory protection or protective clothing while removing the contaminant with brushes and high power cleaning machines. The firefighters had a small but statistically significant elevation in DNA single-strand breaks measured by alkaline elution 19 days after the exposure (normalized elution rate 1.48 ± 95% CI: 0.21) in comparison to 19 unexposed firefighters (normalized elution rate 1.21 ± 95% CI: 0.21, $P < 0.05$) matched by age, alcohol consumption, town of residence, and, within smokers, numbers of cigarettes smoked. The controls had relatively little firefighting activity, responding to less than two fires per month (Hengstler *et al*., 1995).

In a small study of nine residents of the USA, aged 36–67, volunteering to fight oil fires in Kuwait between April and June, 1991, no increase in DNA adducts in lymphocytes (measured by nuclease PI modification of the ^{32}P-post-labelling assay) was observed after an average of 12 days after return from duty (Darcey *et al*., 1992). Of the subjects, five were current smokers and three were ex-smokers. All subjects were exposed to smoke from the oil fires both while working and at their residence adjacent to the oil fields. The only respiratory protection used was "particle masks" for 1–2 hours per day.

In a study of 47 currently non-smoking (for at least 6 months) California wildland firefighters aged 18–49, Rothman *et al*., (1993) found that white blood cell PAH–DNA adduct concentrations were associated with the frequency of charbroiled food intake and not with occupational exposure. In this analysis, the mean number of hours of firefighting during the previous week was 0.11 for the early season period, and 22.36 for the late season period.

There are several studies associating exposure to wood smoke from cooking and heating with lung cancer (Delgado *et al*., 2005) as described in the IARC Monograph on Indoor Air Pollution (IARC, 2010b). While analysis of these studies is not within the scope of this section, some studies have evaluated biomarkers which help shed light on potential carcinogenic mechanisms of wood smoke. Given that the extent of most firefighters' exposure to smoke is limited to discreet episodes, and is of a much shorter duration than typical exposures for residential exposure to wood smoke, it is difficult to determine the extent to which the results of the wood smoke studies can be applied to firefighters.

Table 4.2. Genotoxicity studies of firefighters

Effect	Exposure	Country	Note	Reference
Increase in susceptibility to mitomycin C induced sister chromatid exchange correlated with the number of fires fought in the previous 24 hours	43 Firefighters and 40 matched controls	USA (Washington DC)	Municipal firefighters as a group did not have an increased level of SCE as compared with matched controls	Liou *et al.* (1989)
No increase in urinary 8-OHdG	53 Firefighters exposed to fire within 5 days of the study	Republic of Korea	Municipal firefighters	Hong *et al.* (2000)
Increased micronuclei in buccal cells	47 Firefighters compared with 40 controls	India	Municipal firefighters	Ray *et al.* (2005)
DNA single strand breaks 19 days after the fire in comparison to control groups	16 Firefighters without respiratory protection exposed to *o*-nitroanisole and other chemicals	Germany	Incident did not involve a fire	Hengstler *et al.* (1995)
No increase in white blood cell DNA adducts	9 Firefighters an average of 12 days after return from duty	Exposures in Kuwait, firefighters from USA	Oil well fires	Darcey *et al.* (1992)
White blood cell PAH–DNA adducts only from diet, not from occupational exposure	47 Firefighters assayed early and late in the 1988 forest fire season	USA (California)	Wildland firefighters, used rabbit antibody 33 capable of recognizing multiple PAH diol epoxides linked with DNA	Rothman *et al*. (1993)

SCE, sister chromatid exchange; 8-OHdG, 8-hydroxy-2′-deoxyguanosine

(b) Indirect effects potentially related to genotoxicity

(i) *Chronic effects in municipal firefighters*

Studies describing the chronic and acute effects from firefighting that could indirectly lead to genotoxicity are reported in Tables 4.3 and 4.4. These effects are mostly inflammatory in nature. For studies of chronic effects of firefighting, more recent studies have failed to demonstrate the previously described accelerated rate of decline in lung function seen in older studies. This difference is likely due to a more assiduous use of respiratory protection, specifically self-contained breathing apparatus.

Markowitz *et al*. (1991) evaluated 212 New York City firefighters (mean age, 57 years) for evidence of asbestos-related pulmonary disease. A total of 42 (20%) had pleural thickening and/or parenchymal abnormalities, including 20 (13%) of the firefighters without a reported previous exposure to asbestos. Only 15 (7%) of the study participants had worked fewer than 20 years as a firefighter. [No data were available for firefighter asbestos-related pulmonary disease in other locales, so it is difficult to determine the generalizability of these findings.] In a sub-analysis of this larger group, Ford *et al*. (1992) evaluated 33 New York City firefighters for serum biomarkers associated with carcinogenic pathways including transforming growth factor β, plate-derived growth factor, and seven oncogene proteins, ras, fos, myc, myb, mos, src, and int in comparison with 16 healthy matched controls from a medical centre. Fourteen of the firefighters and none of the controls were positive for transforming growth factor β ($P < 0.01$). None of the other tumour genes was found in any subject.

In Seattle, a study of 812 firefighters demonstrated a longitudinal decline in the percent-predicted diffusion capacity of carbon monoxide from 1989–1996, suggesting lung tissue loss from inflammation (Burgess *et al*., 1999), although no longitudinal changes in spirometry were noted. Loke *et al*., (1980) evaluated the pulmonary function in 54 firefighters from Connecticut, including 32 smokers and 22 non-smokers. In non-smokers, small airway obstruction was present only in firefighters with at least 25 years of firefighting, again consistent with a lung inflammatory response.

Studies of municipal firefighters published between 1974–1991 reported variable rates of longitudinal decline in spirometry. In Boston, Peters *et al*., (1974) reported an average annual decline in the Forced Expiratory Volume in 1 Second (FEV1) of 68 mL in a group of 1430 firefighters. In another study in the same city, Sparrow *et al*., (1982) reported an increased annual average rate of decline in FEV1 of 12 mL in 168 firefighters when compared to 1474 non-firefighters. Tepper *et al*. (1991) reported a 2.5-fold increased rate of decline in FEV1 in 632 Baltimore firefighters when compared with retired or firefighters that had resigned during the follow-up period. However, Musk *et al*. (1977) evaluated the pulmonary function in 1146 Boston firefighters for whom the average annual decline in FEV1 was in the normal range of 0.03 L/year, and this rate of decline was not associated with number of fires fought. The authors reported a more frequent use of respiratory protection over the period 1970–1974.

Table 4.3. Chronic effects of firefighting exposure with indirect relevance to cancer

Effect	Exposure	Country	Notes	Ref
Asbestos-related pulmonary disease	42 (20%) Firefighters engaged in routine firefighting had pleural thickening and/or parenchymal abnormalities	USA (New York City)	20 (13%) of these firefighters did not report previous exposure to asbestos	Markowitz *et al.* (1991)
Beta-transforming growth factor related proteins	33 Firefighters engaged in routine firefighting	USA (New York City)	Subset of above study	Ford *et al.* (1992)
Longitudinal decline in percent predicted diffusion capacity of carbon monoxide (DL_{CO}) from 1989 to 1996.	812 Firefighters engaged in routine firefighting	USA (Seattle)	Suggests lung tissue loss from inflammation. No change in percent predicted FEV_1 and FVC	Burgess *et al.* (1999)
Small airway obstruction	54 Firefighters engaged in routine firefighting	USA (Connecticut)		Loke *et al.* (1980)
Accelerated decline in FEV_1	Routine firefighting	USA (Boston, Baltimore)	Multiple studies	Peters *et al.* (1974); Sparrow *et al.* (1982); Tepper *et al.* (1991)
No accelerated decline in FEV_1	Routine firefighting	USA and United Kingdom	Multiple studies	Musk *et al.* (1977); Musk *et al.* (1982); Horsfield *et al.* (1988)
Increased lymphocytes, native fibronectin and hyaluronic acid in bronchoalveolar lavage fluid	13 non-smoking municipal firefighters compared with 112 controls	Sweden	Suggests cell activation and inflammation	Bergström *et al.* (1997)

Table 4.3 (contd)

Effect	Exposure	Country	Notes	Ref
Cross-seasonal decline in FEV_1	Wildland firefighters	USA (California, Montana, Oregon, Washington)		Rothman *et al.* (1991); Liu *et al.* (1992); Betchley *et al.* (1997)
Increased percentage of neutrophils and MMP-9 concentrations in sputum	39 New York City Firefighters exposed to World Trade Center dust compared to 12 firefighters and 8 non-firefighters from Tel Aviv	USA (New York City) and Israel (Tel Aviv)	Sputum collected 10 months after the collapse	Fireman *et al.* (2004)
Decline in FEV_1	323 Firefighters exposed to World Trade Center dust compared with a historical referent group (*n*=687) and unexposed firefighters (*n*=34)	USA (New York)	Mean decline of 264 mL compared with 147 mL in the historical referent group and 85 mL in unexposed	Feldman *et al.* (2004)

FEV_1, forced expiratory volume; FVC, forced ventilatory capacity; MMP-9, matrix metallopeptidase 9

A follow-up study of these firefighters (Musk *et al*., 1982) demonstrated an average annual decline in FEV1 of 36 mL in 951 subjects. Horsfield *et al*., (1988) failed to demonstrate an increased rate of decline in spirometry in 96 British firefighters when compared to a control group of 69 non-smoking men from other occupations. [The variable use of self-contained breathing apparatus may help to explain the differences in the rate of decline in lung function.]

Bergström *et al*. (1997) collected bronchoalveolar lavage fluid from 13 non-smoking firefighters and the results were compared to a reference group of 112 non-smoking healthy volunteers (the ethical committee who approved this study was from the Karolinska Hospital, Stockholm, Sweden). Nine of the 13 firefighters had one occasion of firefighting during the previous 3 months and four had no exposure during this time period. Firefighters had an increased percentage of lymphocytes and a decreased percentage of activated macrophages when compared to controls (median 8.2 versus 5.7% and 90.5 versus 92.5%, respectively, $P < 0.05$ for both). Native fibronectin and hyaluronidase were higher in firefighters (median 34.6 versus 22.0 μg/L, and 27.7 versus 10.0 μg/L, respectively, $P < 0.05$).

Table 4.4. Acute effects of firefighting exposure with indirect relevance to cancer

Acute effect	Exposure	Country	Note	Reference
Increased lung permeability, measured by serum concentrations of Clara Cell protein and surfactant-associated protein A	51 Firefighters engaged in structural overhaul	USA (Arizona)	Consistent with lung inflammation caused by smoke	Burgess *et al.* (2001)
Decrease in sputum IL-10 concentration	19 Firefighters engaged in structural overhaul (subset of previous study)	USA (Arizona)	IL-10 may play a role in preventing cancer	Burgess *et al.* (2007)
Transient change in lung permeability	6 Volunteer firefighters exposed to polypropylene combustion in a chemical plant	Not described	Change in serum Clara Cell protein	Bernard & Van Houte (1996)
Reduction in FEV_1	Structural firefighters	USA	Mixture of studies with variable use of SCBA	Musk *et al.* (1979); Sheppard *et al.* (1986); Brandt-Rauf *et al.* (1989); Large *et al.* (1990)
Reduction in FEV_1 and FVC	Cross-shift study of 76 wildland firefighters	USA (Washington and Oregon)		Betchley *et al.* (1997)

FEV_1, forced expiratory volume; FVC, forced ventilatory capacity; IL-10, interleukin-10; SCBA, self-contained breathing apparatus

(ii) *Chronic effects in wildland firefighters*

In wildland firefighters, a cross-seasonal decline in FEV_1 has been reported in several studies. Betchley *et al*. (1997) evaluated spirometry tests in 53 Oregon and Washington wildfire firefighters taken before and after the fire season. On average, participants worked on 9.5 prescribed burns (range 1–25), 5.8 wildfires (range 0–24), and 0.3 urban fires (range 0–5). Testing was performed 1–211 (average 78) days after the last occupational smoke exposure. The mean decline in FEV_1 over this time period was 0.104 litres (P = 0.032) without a significant decline in Force Vital

Capacity (FVC). In 52 California wildland firefighters, a small (–1.2%) but significant cross-season decline in FEV_1 was noted, with firefighters with greater firefighting activity within the previous week having the greatest extent of decline (Rothman *et al*., 1991). In 63 western USA wildland firefighters, cross-season declines in spirometry (FVC 0.09 L, $P < 0.001$, FEV_1 0.15 L), and increased airway responsiveness were observed (Liu *et al*., 1992).

(iii) *Chronic effects of unique exposures*

Fireman *et al*. (2004) analysed sputum samples from 39 New York firefighters highly exposed to the World Trade Center dust and those from firefighters and health care workers (non-firefighters) from Tel Aviv. All 39 New York firefighters were exposed to the World Trade Center dust cloud on the day of collapse. The sputum was collected 10 months after the collapse. The authors stated that it was necessary to recruit control populations from outside New York because of the high percentage of individuals exposed to World Trade Center dust to at least some extent. [However, they did not describe the potential bias from breathing New York City air as compared with Tel Aviv air contaminants other than those produced by the World Trade Center, and no comparisons were provided of ambient air quality in each city.] The percentage of neutrophils in the New York firefighters (mean 50.7 ± 17%) was significantly higher than in the health care worker controls (29.1 ± 8.9%, $P < 0.05$), but not in Tel Aviv firefighters (44.1 ± 22.9%). Sputum matrix metalloproteinase 9 concentrations in the New York firefighters (mean 2.2 ± 2.6 ng/mL) were also significantly higher than in the controls (0.3 ± 0.1 ng/mL, $P < 0.05$), but not in Tel Aviv firefighters (1.2 ± 1.0 ng/mL, $P = 0.057$). Within the New York firefighters, those with ≥ 10 cumulative workdays at the World Trade Center ($n = 16$) had a greater percentage of sputum neutrophils than those with <10 days ($n = 23$) (55.7±15.2% v. 44.2±16.5%, $P = 0.05$). Chemical analysis of the sputum samples from New York firefighters revealed multiple elements including zinc, mercury, gold, tin, and nickel, which were not present in the sputum samples of Tel Aviv firefighters. Feldman *et al*. (2004) noted an increased decline in FEV_1 in New York City firefighters when compared to historical controls and to an unexposed group.

(iv) *Acute effects*

Smoke exposure has also been associated with various acute health outcomes potentially related to cancer (Table 4.4). In a study of 51 Arizona firefighters during overhaul, a phase of firefighting during which fire fighters historically did not and may presently not wear respiratory protection, evidence of increased lung permeability was observed, consistent with lung inflammation caused by smoke. This was assessed by measuring the serum concentrations of Clara Cell protein and surfactant-associated protein A (Burgess *et al*., 2001). Changes in surfactant-associated protein A were correlated with atmospheric exposure to acetaldehyde and formaldehyde, as well as carboxyhaemoglogin levels. In a subset of 19 of these firefighters, smoke exposure also caused a decrease in sputum interleukin-10 (IL-10)

concentrations (Burgess *et al*., 2007), and IL-10 may play a role in cancer prevention (Giordani *et al*., 2003). Bernard and Van Houte, (1996) also found increased serum Clara Cell protein in six volunteer firefighters exposed to polypropylene combustion products in a chemical plant for approximately 20 minutes, as compared to six age-matched controls. When re-tested 10 days after the exposure, the serum Clara Cell protein levels had returned to values similar to that of controls.

Several studies have demonstrated an acute decline in spirometry following smoke exposure in firefighters. Musk *et al*. (1979) evaluated 39 firefighters during routine firefighting duty. Over 137 observations, the average decrease in FEV1 following smoke exposure was 0.05 L. The decline in FEV1 was related to the measured particulate concentration of the smoke, and also to the severity of smoke exposure as estimated by the firefighter. Sheppard *et al*. (1986) evaluated 29 firefighters from a single fire station over an 8-week period, measuring FEV1 and FVC in each firefighter before and after each 24-hour workshift and after every fire. Eighteen of 76 measurements obtained within 2 hours after a fire (24%) showed a greater than 2 standard deviations fall in FEV1 and/or FVC compared to two of 199 measurements obtained after routine shifts without fires (1%; $P < 0.001$). Brandt-Rauf *et al*. (1989) evaluated 37 firefighters with baseline and immediate post-firefighting spirometry tests. In 14 firefighters not wearing a self-contained breathing apparatus, there was an average 0.19 L decline in FEV1 ($P = 0.014$) and a 0.22 L decline in FEV1 ($P = 0.007$), whereas there was no significant decline in spirometry when including all firefighters (those wearing and not wearing a self-contained breathing apparatus). Large *et al*. (1990) evaluated 60 Pittsburgh firefighters before and after exposure to house fires with use of a self-contained breathing apparatus for variable periods of time, finding a 3–11% decline in FEV_1.

Acute decline in spirometry has also been reported in wildland firefighters. Betchley *et al*. (1997) evaluated cross-shift spirometry tests in 76 Oregon and Washington firefighters. The mean decline in FEV1 was 0.190 L ($P < 0.001$), and in FVC, 0.089 L ($P = 0.009$).

4.2.2 *Experimental systems*

Wood smoke has been shown to cause oxidative stress and DNA damage *in vivo* (see Table 4.5). Extensive additional information on the genotoxic effects of wood smoke is summarized in the IARC Monograph on Indoor Air Pollution (IARC, 2010b). Specifically, there are data supporting lipid peroxidation and free radical generation as mechanisms of injury. Demling & LaLonde (1990) exposed sheep to smoke from strips of dyed cotton towelling, 20 breaths at 5 and 10 mL/kg. The 5 mL/kg exposure resulted in an increase in plasma malondialdehyde level without significant lung or systemic physiological changes. Lipid peroxidation has been shown to be associated with cancer (Otamiri & Sjödahl, 1989). At higher exposure levels causing severe respiratory failure, this oxidant activity was felt to be due to smoke-induced systemic inflammation (Demling *et al*., 1994). Leonard *et al*. (2000) also demonstrated DNA strand breakage by single-cell gel electrophoresis in RAW

264.7 cell cultures incubated with 50 µL smoke collected in saline solution for 24 hours. This damage was felt to be due to the generation of hydroxyl radicals produced from the interaction of H_2O_2 with carbon-centred radicals in wood smoke. In a later study, Leonard *et al*. (2007) also demonstrated an increase in lipid peroxidation following exposure of RAW 264.7 cells to wildfire smoke suspensions, with the ultrafine and fine size particles inducing a significant increase in malonaldehyde levels. Forest fire smoke also induced dose-dependent increases in sister chromatid exchange in cultured human lymphocytes (Viau *et al*., 1982).

Ultrafine particles, similar in size to those produced during combustion, appear to have greater effects and a significant potential to injure the lung compared to an equivalent dose by weight of larger particles (Oberdörster *et al*., 1992). Atlas *et al*. (1985) collected particulate and vapour phase contaminants from diesel oil fires at a firefighter training facility and exposed the *S. typhimurium* strains TA98 and TA100 with and without enzyme (rat liver S-9 mix) activation. They found an approximately 5-fold greater mutagenicity from the contaminant mixture than would be expected from the concentration of benzo[*a*]pyrene alone. Woodsmoke has also been shown to be mutagenic in cellular assays, as reported in the IARC Monograph on Indoor Air Pollution (IARC, 2010b).

Table 4.5. Genetic and related effects of woodsmoke

Effect	Exposure	System	Reference
Lipid peroxidation (plasma malondialdehyde)	Cotton towelling smoke 5–10 mL/kg tidal volume x 20 breaths	Sheep	Demling & LaLonde (1990)
DNA lipid peroxidation and single strand breakage	Pine and fir bark smoke bubbled through saline	Mouse peritoneal macrophage cell line (RAW 264.7)	Leonard *et al.* (2000, 2007)
Sister chromatid exchange	Forest fire smoke (in Kentucky, USA)	Cultured human lymphocytes	Viau *et al.* (1982)
Mutagenicity	Particulate and vapour phase contaminants from oil fire plumes at a firefighter training facility	Ames test bioassay	Atlas *et al.* (1985)

RAW 264.7, mouse peritoneal macrophage cell line

4.3 Susceptible populations

A limited number of studies are available evaluating the effects of genetic polymorphisms on metabolites or clinical end-points likely related to cancer. In a study of 78 firefighters in the Republic of Korea, including 53 that had been exposed to smoke within 5 days of the study (Hong *et al*., 2000), polymorphisms in the

CYP1A1, *CYP2E1*, *GSTM1*, and *GSTT1* genes were not associated with an increase in urinary 8-hydroxy-2′-deoxyguanosine concentration [although the sample size was small for a genetic study]. Recent firefighting activity was not a predictor of urinary 8-hydroxy-2′-deoxyguanosine. In studies of decline in lung function in firefighters, a single nucleotide polymorphism at position 1668 in the IL-10 gene, was related to the rate of decline in lung function in 379 firefighters (Burgess *et al*., 2004). In a smaller study of 67 firefighters, TT genotypes at IL-10 (–819) (P = 0.009) and CT or TT genotypes at IL-1RA (2018) (P = 0.050) as well as increased sputum IL-1RA were associated with a slower rate of FEV_1 decline (P = 0.025) (Josyula *et al*., 2007). These genes and protein are all associated with inflammation and the rate of decline in lung function may potentially be associated with cancer end-points. This possible association is supported by research linking IL-10 single nucleotide polymorphisms to lung cancer (Tseng *et al*., 2006) and IL-1RA gene polymorphisms to bulky hydrophobic DNA adducts in the lung (Lind *et al*., 2005). Data on genetic polymorphisms in metabolic genes and DNA repair genes in relation to lung cancer are available in individuals with exposure to smoke (IARC, 2010b) but not firefighters. As described elsewhere in this monograph, shiftwork may increase firefighter susceptibility to certain cancers.

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5. Summary of Data Reported

5.1 Exposure data

Several types of firefighters exist, including municipal, wildland, industrial, aviation, and military firefighters. Municipal firefighters may be assigned to combat firefighting units only or to unexposed activities such as fire prevention or technical support. Firefighters may also be fire-scene investigators who are exposed during fires or shortly following a fire. Many firefighters work in shifts (see the monograph in this Volume).

Both municipal and wildland firefighting involve two phases: in an initial phase (knockdown and attack, respectively), the fire is extinguished; in a second phase (overhaul and mop-up, respectively), small fires and hot-spots are extinguished.

All fires generate an enormous number of toxic combustion products, including known and possible carcinogens, long-lived free radicals, and particulate matter. Smoke particles may serve as vehicles for adsorbed volatile organic compounds. Peak exposures to some carcinogens may be very high, notably for benzene, 1,3-butadiene, and formaldehyde. The concentrations of respirable particulate matter to which firefighters may be exposed during overhaul can reach 50 mg/m^3, or up to 1000 mg/m^3, and above in the case of coarser particles. Exposures of firefighters to volatile organic vapours have generally been in the low parts-per-million range.

Firefighters may be exposed at different levels depending on crew assignment, tasks and/or the time spent at fires. Wildland firefighters appear to spend more time at fires during a fire season than municipal firefighters spend during an entire year. In municipal firefighting, overhaul also involves pulling down ceilings and walls, which may entail exposures to substances other than combustion products. Both municipal and wildland firefighters engage in heavy work levels when combating fires, and the increased respiration rate results in an increase in absorbed dose. In recent decades, very effective respiratory protection equipment has been made available to municipal firefighters. In most jurisdictions, wildland firefighters generally do not use respiratory protection.

5.2 Human carcinogenicity data

The Working Group reviewed 42 studies of cancer in firefighters that included 19 cohorts, 11 case–control studies, and 14 studies that used other designs. The studies that were most relevant to the assessment of the risk for cancer among firefighters were the larger historical cohort studies.

Elevated relative risks for cancer at many different sites were identified by one or more studies, but few were observed consistently. A recent meta-analysis evaluated 32 studies and found that the risk for cancer in firefighters was significantly elevated for ten sites, four of which showed the strongest evidence of an association. Since that analysis, two more large epidemiological studies of cancer in firefighters have been

reported. Therefore, another meta-analysis that included these two studies was performed by the Working Group for the four primary cancer sites. Three types of cancer showed significant summary risk estimates: the incidence of testicular cancer was ~50% in excess based on six studies and approximately 150 cases, that of prostatic cancer was ~30% in excess based on 17 studies and approximately 1800 cases, and that of non-Hodgkin lymphoma was ~20% in excess based on seven studies and more than 300 cases.

Four cohort studies that investigated testicular cancer in firefighters yielded risk estimates that ranged from 1.2 to 2.5 and one case–control study gave odds ratios that ranged from 1.5 to 4.3. One of three studies found a positive trend between duration of exposure and the increased risk for testicular cancer.

Of 20 studies of prostatic cancer, 17 reported elevated risk estimates that ranged from 1.1 to 3.3; however, only two reached statistical significance and only one study showed a trend with duration of employment.

The studies that investigated non-Hodgkin lymphoma in firefighters used different definitions of this tumour. Five cohort and one case–control studies that evaluated non-Hodgkin lymphoma reported risk estimates that ranged from 0.9 to 2.0. Only one study evaluated exposure–response with duration and did not find a positive relationship.

Although firefighters are exposed concurrently to a multitude of chemical compounds that include numerous carcinogens, human epidemiological studies at best used indirect (poor) measurements of exposure to such agents. Also, exposures of firefighters vary considerably depending on their job activities, and only crude measures of exposure, such as duration of employment and number of runs, have been used in these studies. Despite these limitations, increased risks for some cancers were found for firefighters in the meta-analysis.

5.3 Animal carcinogenicity data

No data were available to the Working Group.

5.4 Other relevant data

Smoke is a complex mixture of suspended particulate matter, gas, and vapour. The lack of data on toxicokinetics and toxicity of the adsorption of chemical components onto particles prevents a full understanding of the effects of smoke on firefighters. The toxicokinetics of chemical mixtures are not well understood but are probably of significant importance because of the multiplicity of chemicals in smoke. For individual smoke components, inhalation was considered to be the major source of exposure; however, dermal absorption is also an important route of exposure for polycyclic aromatic hydrocarbons and polychlorinated biphenyls.

There are insufficient studies to evaluate genotoxic effects in firefighters.

There is clear evidence of chronic and acute inflammatory respiratory effects in firefighters, which provides a potential mechanism for carcinogenesis, although the major effect would be expected in the respiratory system.

No genotoxicity studies in animals were found that involved exposure to smoke from the combustion of structural materials. Smoke causes lipid peroxidation, which may be associated with cancer. Wood smoke suspensions has been shown to cause DNA strand breakage and lipid peroxidation in cell cultures.

6. Evaluation and Rationale

6.1 Cancer in humans

There is *limited evidence* in humans for the carcinogenicity of occupational exposure as a firefighter.

6.2 Cancer in experimental animals

There is *inadequate evidence* in experimental animals for the carcinogenicity of occupational exposure as a firefighter, since no data were available to the Working Group.

6.3 Overall evaluation

Occupational exposure as a firefighter is *possibly carcinogenic to humans (Group 2B)*.