Chemical Substances and Biological Agents

Studies and Research Projects

REPORT **R-522**

Risk of Multiple Myeloma and Cancers of the Respiratory System, Oesophagus, Stomach, Pancreas, Prostate, Testes and Skin in Firemen

Douglas McGregor

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Narrative reviews of risks of several cancers (non-Hodgkin lymphoma, leukaemia, brain, colorectal, kidney and urinary bladder) and occupation as a fireman have been prepared in this series. These cancers were reviewed separately because there appeared that there might be evidence for an association of increased risk with fighting fires. There remain many other cancers for which evidence for an association with this occupation could also exist. These cancers have received less attention in the literature (although they may have been mentioned in numerous studies) and are reviewed together in the same narrative style here.

Multiple Myeloma.

Multiple, or plasma cell myeloma is a bone marrow based neoplasm that is multifocal, characterised by a monoclonal protein (M-component) in serum and urine, and causes skeletal destruction with consequent bone pain and fractures. A number of variations exist and in addition to those consisting primarily of plasma cells others may contain both lymphocytes and plasma cells. The diseases may be localised or disseminated with plasma cell infiltration of various organs and deposition of abnormal immunoglobulin chains in tissues (primary amyloidosis and light chain disease) (Grogan et al., 2001). In the U.S.A., multiple myeloma is the most common lymphoid neoplasm in Blacks and the second most common in Whites. There was a net increase of about 45% in the disease between 1940 and 1970. The median age at diagnosis is about 70 years and there is little difference in incidence between men and women (Devesa et al., 1987). Cosmetologists, farmers and takers of laxatives have been described as being at 3 – 4-fold increased risk for multiple myeloma and specific exposures associated with these risks include pesticides, petroleum products, asbestos, rubber and wood products (Linet et al., 1987).

Cancers of the Respiratory System.

These include cancers of the trachea, bronchi and lungs, involving almost exclusively the epithelia. Primary carcinoma of the lung was uncommon until the 1930s, since when there was a dramatic increase that is now abating in men of developed countries, but not in women of these same countries and not in people of either sex in many developing countries. Currently, there are approximately 960,000 new cases each year in men (about 13.5% from North America) and approaching 390,000 new cases each year in women (about 24% from North America), worldwide (Ferlay et al., 2002). Geographical and temporal patterns demonstrate that tobacco consumption is the most important risk factor. Compared with non-smokers, the relative risk of lung cancer in tobacco smokers is about 8 – 15 in men and 2 – 10 in women. There are several histological types of lung cancer, the more common being squamous cell carcinoma, adenocarcinoma and small (oat) cell carcinoma. Clearly, in some populations, tobacco smoking is not the only risk factor for lung cancer. Absolute lung cancer rates are high among non-smoking women in certain parts of China, an observation that has been attributed to indoor cooking with poorly ventilated coal-fuelled stoves. The absolute death rate from lung cancer among women who smoke in these areas of China is substantially higher than the average death rates from lung cancer among women in the U.S.A. (reviewed in IARC, 2004). This difference may be interpreted as indicating causes of lung cancer other than tobacco smoke; however, the latter is so important that any study of lung cancer that does not account for tobacco smoking is subject to such very strong confounding that it cannot be relied upon to provide evidence for another risk factor. Based on analyses of the American

Cancer Society Cohort (CPS-II), it has been estimated that, if the impact of competing causes of death are excluded from the calculation, the lifetime probabilities of death from lung cancer in male and female smokers aged ≥ 85 years is 24.1% and 11.0% for men and women, respectively, who continue smoking and 1.6% and 1.1% in male and female non-smokers, respectively (Thun et al., 2002; IARC, 2004).

Cancers of the Oesophagus.

About 412,000 cases of oesophageal cancer occur each year, of which over 80% are in developing countries. Of the various cancers of the oesophagus, over 95% are either squamous cell carcinomas or adenocarcinomas. Squamous cell carcinoma occurs at high frequency in developing countries while adenocarcinoma is essentially a tumour of more developed and industrialised countries. The age-standardised annual incidence of squamous cell carcinoma in most Western countries does not exceed 5 per 100,000 in men and 1 per 100,000 in women, whereas incidence and mortality are very high in Iran, Central China, South Africa and Southern Brazil (Gabbert et al., 2000). The incidence of adenocarcinoma is steadily increasing in Europe and North America at a rate 5 – 10% per year and now accounts for more than 50% of all oesophageal tumours in the U.S.A. and some European countries (Parkin et al., 1997).

Oesophageal squamous cell carcinoma is a malignant epithelial tumour characterised by keratinocyte-like cells with intercellular bridges or keratinisation. Its occurrence is associated with tobacco smoking, the consumption of alcohol and with the low intake of fresh fruit, vegetables and meat. It has been estimated that in the more developed countries 90% of squamous cell carcinoma is attributable to tobacco and alcohol, while dual exposure has a multiplicative effect on risk (Launoy et al., 1997). High risk is associated with a moderate use of tobacco over a long period and a high use of alcohol over a short period. Nutritional factors may also be involved, including a deficiency of certain trace elements, the consumption of Chinese-style pickled vegetables or mouldy foods and the consumption of burning-hot beverages that cause thermal injury leading to chronic oesophagitis and then to precancerous lesions (Gabbert et al., 2000).

Oesophageal adenocarcinoma is a malignant epithelial tumour with glandular differentiation arising predominantly from Barrett mucosa in the lower one-third of the oesophagus. Barrett mucosa is a condition arising from damage to the lining of the lower oesophagus resulting from chronic gastric and duodenal (bile acids and pancreatic enzymes) reflux. The normal squamous cells undergoes metaplasia and are replaced by a columnar epithelium with cells resembling those of the intestine or the salmon-pink mucosa of the stomach. Intestinal metaplasia is found in more than 80% of patients with adenocarcinoma of the distal oesophagus. Other risk factors include tobacco smoking, which may account for as much as 40% of cases, and obesity, but there is no strong association between alcohol consumption and adenocarcinoma and it has been suggested that infection by *Helicobacter pylori* may even exert a protective effect (Werner et a., 2000).

Without proper control for tobacco smoking, alcohol use and reflux disease, epidemiological studies cannot be relied upon to identify other risk factors for squamous cell carcinoma or adenocarcinoma and if the histological type of oesophageal cancer is not mentioned this omission only adds to the difficulties in identifying risk factors for any of these cancers.

Cancers of the Stomach.

Worldwide, mortality from cancer of the stomach, the vast majority of which are gastric carcinomas, is second only to lung cancer. There are approximately 800,000 new cases and 650,000 deaths each year, but the incidence and mortality rate are declining, even in Japan where they are highest and about 10-fold higher than in low incidence areas such as North America, Northern Europe, Africa and Southeast Asia. In some Western countries rates have fallen to less than one-third within just one generation. Nevertheless, mainly because of the aging population, the absolute number of new cases each year is increasing. Analysis of time trends by histological type indicates that the incidence decline is mainly the result of a decline in the intestinal type of carcinoma (Muñoz, 1988).

Gastric carcinomas are malignant epithelial tumours of the gastric mucosa with glandular differentiation. They are extremely rare below the age of 30 years. Adenocarcinomas are principally of the intestinal type or diffuse type. The age – adjusted rates rise faster with age for the intestinal type, which is also more frequent in men than women. In addition, papillary, tubular and mucinous adenocarcinomas occur in the stomach, as well as a number of different carcinomas and non-epithelial tumours (Fenoglio-Preiser et al., 2000). Most gastric cancers are preceded by a prolonged pre-cancerous process that includes the following sequential steps: chronic gastritis, multifocal atrophy, intestinal metaplasia and intraepithelial neoplasia (Correa, 1992).

Diet is the most consistent association with gastric cancer, particularly intestinal type adenocarcinoma, in different populations. High salt content, smoked or cured meats or fish, pickled vegetables and chilli peppers are risk factors, while fresh fruits and vegetables tend to lower risk. Studies of possible association with alcohol, tobacco and occupational exposures to nitrosamines and inorganic dusts have been inconclusive. In contrast, there is strong epidemiological evidence for an association with *Helicobacter pylori* infection. However, this organism predominantly occurs in the mucus layer overlying normal gastric epithelium, but is absent in areas overlying intestinal metaplasia where neoplasia originates (Fenoglio-Preiser et al., 2000).

Cancers of the Pancreas.

The pancreas is a complex organ consisting of both endocrine and exocrine tissues. About 90% of pancreatic cancers are adenocarcinomas of the ductal epithelium of the exocrine organ. Cancers arising in the islets of Langerhans, the endocrine portion, are rare. Pancreatic cancer is moderately common, with approximately 216,000 new cases arising per year worldwide. In the developed world, incidence is high and rose about three-fold over the period 1920s to 1970s, when it stabilised. Incidence is particularly high in male black Americans, Maoris, Korean Americans and female native Hawaiians (Jorgensen & Imrie, 1998). Cigarette smokers have a 2- to 3-fold higher risk than non-smokers and about 30% of cases are attributable to the habit. A number of other, less well-established risk factors have been suggested. These include diets low in fibre, high in heterocyclic amines generated in cooking meat and fish and high in fat. Occupations such as working in mines, metal works, sawmills, chemical plants, coke plants and rubber factories have also been suggested risk factors. These occupations include exposures to various solvents and aromatic amines, while polychlorinated biphenyl mixtures have also been suggested as a risk factor (Gold & Goldin, 1998).

Cancers of the Prostate.

Prostate cancers are the third most common cancers in men, with an estimated 513,000 new cases annually worldwide in 2000. They account for approximately 200,000 deaths annually worldwide, particularly in older men in developed countries. Thus, many men with a diagnosis of prostate cancer do not succumb to the disease (Epstein et al., 2004). Rising incidence of these diseases in part reflects the adoption of screening procedures (particularly circulating prostate-specific antigen) and earlier diagnosis (Potosky et al., 1995). These procedures have undoubtedly affected the apparent time trends. In the U.S.A., the recorded "incidence" is now by far the most commonly diagnosed cancer in men. It is also very high in Australia, Canada and northern Europe, whereas it remains relatively rare in Asian populations. Migrants from either West Africa or the Caribbean to the U.K. have higher mortality rates than the locally born population, whereas migrants from East Africa (who are mostly of Indian ethnicity) do not have high mortality (Grulich et al., 1992). Survival as a proportion of cases is much higher in high incidence countries. Differences between U.S.A. and China are almost 90-fold for incidence, but 26-fold for mortality (Epstein et al., 2004). Risk of prostate cancer as a clinical disease is low until after the age of 50, after which it rises steeply at approximately the 9-10th power of age, compared with the 5-6th power for other epithelial cancers (Cook et al., 1969). A large number of different cancers of the prostate have been identified, but the most common are heterogeneous adenocarcinomas (acinar) developing in the peripheral zone.

Racial differences in incidence suggest that genetics factors play a role in the disease, but evidence from migration studies also implicate environmental factors; however, these are not well understood. There is a strong positive association with intake of animal products, yet the evidence is not convincing for a protective role for fruit and vegetables (Epstein et al., 2004). While some occupations (farming, metal working and rubber manufacture) have been implicated in prostate cancer risk, these have been largely retrospective studies affected by recall bias and results have been either inconsistent or not satisfactorily confirmed in subsequent studies (Parent et al., 2001). A prospective study (described in more detail later) found no association or non-significant reductions in risks for metal workers and farmers, respectively, and a non-significant increase in risk for men who had ever worked in the rubber industry (Zeegers et al., 2004). On the other hand, this same study found a significant increase in risk for policemen (Relative Risk, 3.91; 99% CI = 1.14-13.42), especially those who reported being in that occupation for most of their working life (note that the CI was 99%, not the less conservative 95% that is normally used).

Cancers of the Testes.

Cancers at this site are relatively uncommon. Amongst whites in the USA, the age-adjusted incidence of testicular cancer is about 5 per 100,000 and is about seven-fold lower (0.7 per 100,000) amongst blacks in the USA (Ferlay et al., 2002). The incidence is also low amongst blacks living elsewhere. Rates are low amongst Japanese and Chinese living in Asia, but rates for Japanese living in California or Hawaii are increased. Thus, environmental or life-style factors, as well as genetic factors may be involved in testicular tumourigenesis. However, in contrast to the Japanese, Chinese Americans living in California have lower rates than Chinese living in Singapore or Hong Kong. Testicular cancers are most commonly diagnosed between the ages of 20 and 44 years (Buetow, 1995; Schottenfeld, 1996).

Tumours arising from germinal elements accounts for about 95% of all testicular cancers (Schottenfeld, 1996), of which there are 5 basic types: seminoma, teratoma, choriocarcinoma and yolk-sac tumour (endodermal sinus tumour of Teilum). In patients < 15 years of age, yolk-sac tumours and teratomas predominate, while in patients aged 20 – 40 years, seminomas are most common. The remaining 5% of testicular tumours are sex cord – stromal tumours and include Leydig cell, Sertoli cell and granulosa cell tumours.

International patterns show that rates are 4 – 9 times higher in Denmark, Norway, New Zealand and North American whites, compared with the lower rates registered in Asian, African Americans, Black populations in general and non-Jews in Israel. Professional and white-collar occupations have been associated with moderately elevated risks, but no specific risk factors have been identified, although low physical activity is one possible factor. Elevated odds ratios for testicular cancers have sometimes been described for certain other occupational groups, e.g., civilians engaged in aircraft repair, printing, leather finishing, skilled workers exposed to metals, metal dusts and cutting oils; however, testicular cancers have never been important in any evaluation conducted by IARC of these groups of workers or the substances to which they have been exposed. Few risk factors for testicular cancers have been consistently found, the stronger candidates being cryptorchidism and hormonal exposure *in utero*. Nevertheless, cryptorchidism accounts for only about 10% of the cases of human testicular cancers, the association being most consistently found for seminoma and, perhaps, for Leydig cell tumours. There may be an adverse role of endogenous maternal oestrogens on the development of male embryos (Henderson et al., 1979) and the hypothesis has been proposed that environmental exposure to oestrogens and anti-androgens could be possible risk factors (Sharpe & Skakkebæk, 1993).

Cancer of the Skin.

The classification used for skin cancers in most of the publications relating to firemen specifies malignant melanoma, so most of this discussion will be about this neoplasm. The skin is a complex organ, consisting of many different tissues derived from both ectoderm and mesoderm. The incidence of non-melanoma skin cancer is increasing rapidly and consists mainly of basal cell carcinoma and squamous cell carcinoma. Together, these account for approximately 90% of all skin cancers (Albright, 1982). More recent estimates are uncertain because many non-melanoma skin tumours are no longer recorded in cancer registries. Malignant melanoma was once an uncommon cancer, but its incidence has also risen rapidly throughout the world in recent years. It is believed that these changes in both melanomatous and non-melanomatous skin cancer incidence are largely due to changing patterns of exposure to sunlight and a depletion in the protective ozone layer in the stratosphere allowing more solar ultraviolet radiation to reach the earth's surface (Staehelin et al., 1990).

Melanoma is a malignant proliferation of melanocytes, the pigment-forming cells of the skin. These cells also occur in other organs. It is predominantly a cancer of white-skinned populations living in countries with high-intensity ultraviolet radiation, although all ethnic groups are affected to some degree. There are about 133,000 new cases worldwide each year, almost 80% of which are in Australia, Europe, New Zealand and North America (WHO, 2003). Despite the strong association between

exposure to sunlight and development of melanoma, the details of this relationship are not clear. While melanoma may occur anywhere on the skin, the majority of melanoma in men is on the back, while in women the majority is on the legs. This difference is not completely explained by differential exposure to sunlight. Melanoma can also occur in relatively unexposed areas of the skin, such as the palms and the soles. In contrast to basal cell and squamous carcinomas, risk of developing melanoma does not have a direct relationship to cumulative sun exposure; severe, acute skin damage caused as a result of intense exposure to sunlight appears to be important (Dubin et al., 1989; IARC, 1992). Established but rare risk factors include congenital naevi, immunosuppression and excessive use of solaria. Melanomas are much commoner in higher socio-economic groups and is commoner in *indoor* than in *outdoor* workers, even within the same socio-economic group (IARC, 1992).

Firemen: General Characteristics and Exposures

The task of fire fighting consists of two phases: (1) *knockdown*, during which the fire is brought under control, and (2) *overhaul*, when the fire is extinguished and clean up begins. Approximately 90% of structural fires are either extinguished or abandoned and fought from outside within 5 – 10 min., the average duration of heavy physical activity being 10 min. (Gilman & Davis, 1993). Although self-contained breathing apparatuses (SCBA)s are available, these are seldom worn from the time the firemen arrive at the scene until the time that they leave. In a study of exposures in the City of Montréal fire department, Austin et al., 2001a, found that SCBAs were worn about 50% of the time at structural fires, but they were worn for only 6% of the total time spent at all types of fires. Masks are generally put on when the firemen enter a fire or "see smoke," but it is difficult for them to judge when the mask can be safely removed and they are rarely worn during the smouldering phase of a fire or during mop-up operations. Furthermore, communication is essential and this is difficult with the mask in place. The largest category of non-fatal fire fighter injuries associated with fires has been reported to be contact with flames and smoke (39%) and the leading cause of non-fatal injury among younger firemen is related to smoke inhalation (FEMA, 1990).

In a study of municipal structural fires, Austin et al., 2001b found that just 14 different compounds accounted for about 75% of the total volatile organic materials measured (Table 1). These same compounds constituted approximately 65% of all volatile organic compounds in experimental fires burning various materials commonly found in structural fires (Austin et al., 2001c). The spectra of volatile organic compounds were dominated by benzene along with toluene and naphthalene. They also found that propylene and 1,3-butadiene were present in all of the fires and that styrene and other alkylated benzenes were frequently identified. Other materials that have quantified with some regularity include acrolein, carbon monoxide, formaldehyde, glutaraldehyde, hydrogen chloride, hydrogen cyanide and nitrogen dioxide (Bolstad-Johnson et al., 2000; Caux et al., 2003). There may also be exposure to asbestos and various metals, such as cadmium, chromium and lead. In addition, there is almost certainly exposure to diesel exhaust and fumes and to polycyclic aromatic hydrocarbons (PAHs).

During overhaul, recommended ceiling or short-term exposure levels can often be exceeded, e.g., for acrolein, benzene, carbon monoxide, formaldehyde, glutaraldehyde, nitrogen dioxide and sulphur dioxide (Bolstad-Johnson et al., 2000) and several of these, e.g., carbon monoxide and benzene, were found to be present at

appreciable concentrations in the atmosphere at real fire scenes when SCBAs were only used part of the time or not at all, owing to the impression that there was low smoke intensity (Brandt-Rauf et al., 1988, 1989).

The following discussion is focused on the substances mentioned here and listed in Table 1 that could be risk factors for the miscellaneous cancers being considered.

Certain exposures to possibly carcinogenic materials, such as polycyclic aromatic hydrocarbons (PAH), benzene, 1,3-butadiene and diesel exhaust, are associated with fighting fires. The evidence for their involvement will be briefly summarised, as will consideration of the effect of tobacco smoke, because it has components in common with those encountered by firemen in their work, and it could also act as a confounder.

1,3-Butadiene. Among four particularly informative studies of occupational cohorts with exposure to 1,3-butadiene evaluated by the IARC (IARC, 1999), the most prominent malignancy was leukaemia. Of the malignancies discussed here, stomach cancer was significantly higher in just one of the studies.

Benzene and other Petroleum Components. The occurrence of leukaemia in rubber hydrochloride workers was first described by Infante et al. (1977). Since then, there have been many studies in this and related industries from which it has emerged that there is clearly an increase in risk of acute myelogenous leukaemia associated with exposure to benzene (IARC, 1987). Risks of other malignancies have also been suggested, but no conclusive evidence found. Amongst these other malignancies is multiple myeloma. Nevertheless, a review of this topic (Bergsagel et al., 1999) found that while there was strong evidence that high exposure levels of benzene are causally related to the risk of developing acute myelogenous leukaemia, with a “threshold” most likely around 370 – 530 ppm-years, there was no scientific evidence linking exposure to benzene or other petroleum products and the risk of developing multiple myeloma. Data contributing to this latter conclusion include a large cohort of Pilofilm workers, two nested case-control studies of gasoline distribution workers (one in the U.S.A., the other in Canada) and a multinational cohort study of 250,000 petroleum workers in Australia, Canada, U.K. and U.S.A. In a study of exposure of firemen to benzene in Toronto, Canada, the main conclusion was that while there is exposure, even with modern protective equipment, the exposure was rather low, based on *t,t*-muconic acid measurements in urine (Caux et al., 2002).

Toluene and the xylenes have been evaluated by the IARC much more recently than benzene. There were eight epidemiological studies in which *toluene* was mentioned as an exposure (IARC, 1999). In two of these studies, one of rotogravure printers in Sweden and one of shoe-manufacturing workers in the U.S.A., it was believed that toluene was the predominant exposure. Cancers at most sites were not significantly associated with toluene exposure in any study. Mortality from cancers of the stomach, colon or rectum and respiratory tract were significantly elevated in the study of Swedish rotogravure printers, but only mortality from respiratory tract cancers was also significantly elevated in the study of shoe-manufacturing workers. Furthermore, cancer incidence was significantly elevated amongst the printers only in the case of respiratory tract cancer. This suggests that there could have been bias in the mortality data for cancers of the stomach, colon and rectum in the Swedish study. There was no dose-related increase in risk in either study for any of these cancers. Exposures to *xylenes* were mentioned in four epidemiological studies summarised by IARC (1999). Cancers at most sites were not significantly associated with exposure to xylenes in

any study. An exception was colorectal cancer in one case-control study, but in none of the others. There are no epidemiological studies in which *ethylbenzene* (IARC, 2000), *propylene* (IARC, 1994) or *naphthalene* (IARC, 2002) could be implicated in any of the cancers discussed here. In addition, *styrene*, which is produced mainly by the catalytic dehydrogenation of ethylbenzene, has not been implicated as a risk factor for any of these miscellaneous malignancies (IARC, 2002).

PAHs. A study involving 3730 cancer cases of the possible association between occupational exposure to PAHs and 14 different kinds of cancer was conducted in Montréal (Nadon et al., 1995). Among the PAH exposures considered were benzo(a)pyrene and five categories of PAHs defined on the basis of the source material, which were wood, petroleum, coal, other sources and any source. Each PAH source was subdivided into unexposed, low and high exposure sub-groups (based on duration, concentration and frequency of exposure). Significantly increased risks were found for cancers of the stomach, oesophagus, lung (squamous cell, but not lung overall or oat cell) and prostate. For oesophageal and gastric cancers, the evidence came from low dose exposures to wood sources, but not high dose wood source or to any other source of PAHs. For squamous cell cancer of the lung, the significant elevation was in the low dose, but not the high dose petroleum source of PAHs, and the evidence came from light smokers or non-smokers of tobacco. For cancer of the prostate, the evidence came from low sources of benzo(a)pyrene specifically, but not to high dose sources, and to low dose wood or "other" sources of PAHs, but not to high dose sources. This study suggests that occupational exposure to PAHs might be a risk factor for squamous cell cancer of the lung, but only if tobacco smoking is not also a significant factor.

Cancer risk from occupational exposure to PAHs has been reviewed (Boffetta et al., 1997). High occupational exposure to PAHs occurred in aluminium production, coal gasification, coke production, iron and steel foundries, tar distillation, shale oil extraction, wood impregnation, roofing, road paving, carbon black production, carbon electrode production, chimney sweeping and calcium carbide production. In addition, workers exposed to diesel engine exhaust are exposed to PAHs and nitro-PAHs. It was concluded that heavy exposure to PAHs entails a substantial risk of cancer in the lung, skin and urinary bladder. Of these three, the lung seemed to be the major target with increased risk observed in most of the named industries and occupations. Skin cancer follows high dermal exposure and increased risk of urinary bladder cancer is found mainly in industries with high exposure to PAHs from coal tars and pitches. In spite of these generalisations, it is clear that the increased risks frequently failed to reach a statistically significant level. That said, a substantial number of case control studies found increased lung cancer risks amongst drivers, particularly if they were driving diesel-powered vehicles. In a study of firemen to PAH exposure in Toronto, Canada, the main conclusion was that while there is exposure, even with modern protective equipment, the exposure appears low in comparison to that observed in many industrial workers (Caux et al., 2002). Additionally, unlike industrial workers, the exposure of firemen is not repeated 8 h per day, 5 days per week. Shortcomings of this study noted by the authors were the small number of fire types encountered during the study and the possibility that volunteer firemen may well have used their protective equipment more extensively than others.

Diesel exhausts and fumes. An authoritative evaluation of possible carcinogenic effects of diesel exhausts and fumes found that there was *limited evidence* for the carcinogenicity in humans of diesel engine exhaust, a conclusion that was based on

moderate increases in lung cancer risks. Increased risks for other malignancies were not identified (IARC, 1989). Similar findings were the outcome of a review of work with exposure to diesel engine exhaust in the transport industry (Boffetta et al., 1997).

Tobacco smoke. An important potential confounder in any study of cancer is the smoking of tobacco. An evaluation of the carcinogenic hazard due to direct and environmental exposure to tobacco smoke has been undertaken (IARC, 2004). As a result, the following conclusions were reached that are relevant to the cancers under consideration here.

In populations with prolonged cigarette use, the proportion of lung cancer cases attributable to cigarette smoking has reached 90%. Tobacco smoking increases the risk of all histological types of lung cancer including squamous-cell carcinoma, small-cell carcinoma, adenocarcinoma (including bronchiolar-alveolar carcinoma) and large-cell carcinoma. Duration of smoking is the strongest determinant of lung cancer in smokers and risk of lung cancer increases in proportion to the numbers of cigarettes smoked. Stopping smoking at any age avoids the further increase in risk of lung cancer incurred by continued smoking.

Tobacco smoking is causally associated with cancer of the oesophagus, particularly squamous-cell carcinoma, but also adenocarcinoma. In contrast to lung cancer, risk of oesophageal cancer remains elevated many years after cessation of smoking. There is a greatly increased risk for squamous-cell carcinoma if tobacco smoking and alcohol are co-exposures.

Pancreatic cancer is causally associated with cigarette smoking and the risk increases with duration of smoking and the number of cigarettes smoked. Risk decreases with time from stopping smoking.

There is now a consistent association of stomach cancer with cigarette smoking, as shown by many cohort and case-control studies in many parts of the world. Confounding by factors such as alcohol and *Helicobacter pylori* infection can be reasonably ruled out. Risk increases with duration of smoking and number of cigarettes smoked, and decreases with increasing duration after stopping smoking.

While there is some evidence from prospective cohort and case-control studies that colorectal cancer is increased amongst smokers, a causal connection has not been made. As for the other malignancies being considered – multiple myeloma, cancer of the prostate and cancer of the testes – the evidence for any association with cigarette smoking is either weaker or non-existent.

It is clear that any study of lung cancer that does not control adequately for cigarette smoking is fundamentally flawed and this factor must also be carefully considered when evaluating cancers of the oesophagus and pancreas.

Epidemiological Studies Reviewed.

Multiple myeloma (Table 2).

This cancer was mentioned in six of the studies: one case-control study (Demers et al., 1993) and five cohort studies, one of which was a study of incidence (Demers et al., 1994), the others of mortality (Aronson et al., 1994; Burnett et al., 1994; Ma et al., 1998; Baris et al., 2001). A significantly elevated risk was observed in the proportionality study of Burnett et al. (1994) involving firemen from in 27 states of the USA: PMR = 1.48 (CI 1.02-2.07), based on 34 cases, although this finding was

not confirmed in the overlapping, mortality odds ratio (MOR) study of Ma et al. (1998), which involved firemen from 24 states: MOR = 1.1 (CI 0.8-1.6), based on 28 cases. In none of the other studies was there a statistically significant increase in overall risk. The numbers of these cancers, however, were small. In the study of Baris et al. (2001) there were subgroup analyses of the total of 10 cases, using measures that could be surrogates for exposure. Duration of employment was one of these and there was an increase in risk with this parameter that reached significance amongst those with the longest employment (≥ 20 years): SMR = 2.31 (CI 1.04-5.16), based on 6 cases. A significantly increased risk was also observed in those firemen employed only in engine companies: SMR = 2.54 (CI 1.15-5.68), based on 6 cases, but not in those employed in both engine and ladder companies: SMR = 1.33 (CI 0.50-3.56), based on 4 cases. However, no significantly increased risks were observed according to either year of hiring (before 1935, 1935-1944 or after 1944) or the number of runs made during employment (low, < 3323; medium, ≥ 3323 - < 5099; high, ≥ 5099), although both of these estimates were made of a total of only 6 cases each. Also, the number of lifetime runs with diesel exposure had no significant effect on risk, although these estimates were also made on very small numbers (6 in total, 3 of which were in the non-exposed group). Thus, the evidence for fire fighting as a risk for multiple myeloma rests on an unconfirmed proportionality study and certain elements of the Baris et al. (2001) study. Although, in several respects, this is the best study of those available, it is not enough to allow a conclusion that the occurrence of multiple myeloma in a fireman is due to his employment. The number of cases is small, there are inconsistencies in the risks associated with various measures of exposure and it is not possible from the publication to see if the same or different 6 firemen had both the longest employment and only worked in engine companies. Based on these data it cannot be assumed that a fireman with multiple myeloma developed the disease as a result of his employment.

Cancers of the Respiratory System (Table 3).

This cancer was mentioned in 22 publications relating to firemen. These included a case-control study, 16 retrospective (historical) mortality studies, three incidence studies and two studies that included separate analyses of mortality and incidence. A significant increase in risk was observed in one mortality study amongst white men (Ma et al., 1998) in which mortality odds ratios cancers of the lung and bronchus were marginally, but significantly increased: MOR = 1.1 (CI 1.0 – 1.2), based on 633 cases. Statistically significant increases in risk were also observed in sub-sets of lung cancer data in two studies: (Hansen, 1990) for men aged 60 – 74 years, SMR = 3.17 (CI 1.17 – 6.91), number of cases not stated, and (Heyer et al., 1990) for men aged > 65 years, SMR = 1.77 (CI 1.05 – 2.79), based on 18 cases. No significantly increased risk for lung cancers was observed in any other study. Notably, these non-significant studies included those of Demers et al. (1992b) and Demers et al. (1994). The first of these analysed both death certificate and the more reliable cancer registry data. The second compared lung cancer incidences derived from cancer registries in firemen with men in the same counties and with police in the same cities. Based on these data it cannot be assumed that a fireman with cancer of the lung or elsewhere in the respiratory system developed the disease as a result of his employment.

Cancers of the Oesophagus (Table 4).

Ten publications were identified that mentioned cancer of the oesophagus in relation to employment as a fireman, seven of which were mortality studies, two were incidence studies and one study included separate analyses of mortality and incidence. In none of them was there a statistically significant increase in risk. Based on these data it cannot be assumed that a fireman with testicular cancer developed the disease as a result of his employment.

Cancers of the Stomach (Table 5)

Fourteen publications were identified that mentioned cancer of the stomach in relation to employment as a fireman, 12 of which were mortality studies and two studies included separate analyses of mortality and incidence. Only in one of them was there a statistically significant increase in risk (Demers et al., 1992b). The significant finding was an increase in mortality: SMR = 2.04 (CI 1.05-3.56), based on 12 cases. This found some support from the incidence analysis of the more reliable cancer registry data, although a statistically significant increase in risk was not quite achieved: SIR = 1.75 (CI 0.98-2.89), based on 15 cases. It is clear from these data that there is in fact little difference between the results of the two analyses. Refinement of analysis (Demers et al., 1994) by comparing cancer registry data for firemen and police in the same cities did not show any increased risk for firemen, but the number of cases available was reduced: IDRp = 0.4 (CI 0.1-1.2), based on 8 cases. Numbers of cases were also very small in all other studies, except for Ma et al. (1998), which had 52 white cases: MOR = 1.2 (CI 0.9-1.6) and Beaumont et al. (1991), which had 22 cases: SMR = 1.31 (CI 0.82-1.99). In no study was there any attempt to control for significant confounders such as diet and *Helicobacter pylori* infection. Based on these publications, there is insufficient evidence to lead to an assumption that a fireman with stomach cancer developed the disease as a result of his employment.

Cancers of the Pancreas (Table 6).

There were publications in which these cancers were mentioned in relation to firemen, one of which was a case-control study, eight were mortality cohort studies, one an incidence study and one in which both incidence and mortality were studied. The case-control analysis of 6 white male firemen in Massachusetts with pancreatic cancer (Sama et al., 1990) did not reveal a significantly increased risk in comparison with either white men in general in the State or with police. The mortality study of Ma et al. (1998), based on death certificates, observed a significant increase in risk amongst white firemen and an increase in risk that almost reached significance in black firemen. Although this finding is interesting, it is from a study that is in the nature of a hypothesis generating study and, particularly for white firemen, the mortality odds ratio is small: MOR = 1.2 CI 1.0-1.5) based on 88 cases. None of the other studies observed any significant overall increase in risk. In the Baris et al. (2001) study, there was no overall increase in risk of pancreatic cancer: SMR = 0.96 (CI 0.64-1.44), based on 23 cases. Subgroup analysis showed an increased risk where the duration of employment was ≤ 9 years: SMR = 2.33 (CI 1.36-4.02), based on 13 cases, but risks were less than expected in groups with longer duration of employment. Furthermore, there was no increasing dose-response relationship according to the cumulative number of fire runs or runs during the first five years of employment and there was no relationship with either year of hiring or the type of company (engine, ladder or both)

in which the firemen were employed. Based on these data it cannot be assumed that a fireman with pancreatic cancer developed the disease as a result of his employment.

Cancers of the Prostate (Table 7).

Fifteen publications were identified that mentioned cancer of the prostate in relation to employment as a fireman, 9 of which were retrospective mortality studies, one was a prospective case-cohort study, three were incidence studies and two were studies of both incidence and mortality. Statistically significant increases in risk of prostate cancer were observed in two of the mortality studies (Grimes et al., 1991; Ma et al., 1998) and in two of the incidence studies (Demers et al., 1992b; 1994). A significant increase in incidence was observed in Demers et al. (1992b): SIR = 1.37 (CI 1.11-1.69), based on 94 cases, but not in mortality: SMR = 1.14 (CI 0.65-1.85), based on 16 cases. The incidence of prostate cancer in firemen, as reported in cancer registries (Demers et al., 1994), was also increased in comparison with men living in the same counties as the firemen: SIRm = 1.4 (CI 1.1-1.7), based on 66 cases. This study was a sub-set of the previous one, allowing these same 66 cases to be compared with police working in the same cities of Seattle and Tacoma, WA. In this comparison that benefited not only from the use of cancer registry data but also because the reference group was likely to be more appropriate and closer to the firemen in terms of socioeconomic factors, the risk was less and did not reach significance: SIRp = 1.1 (CI 0.7-1.8).

The other studies reporting increased risks of prostate cancer among firemen, included a proportionality study (Grimes et al., 1991): PMR = 2.61 (1.38-4.97) and a study of mortality odds ratios (Ma et al., 1998) in which increased risks of prostate cancer were found in both black and white firemen: MOR_{Black} = 1.9 (CI-1.2-3.2), based on 16 cases; MOR_{White} = 1.2 (CI1.0-1.3), based on 189 cases. Both of these studies are in the nature of hypothesis generating studies. In the study by Baris et al. (2001) that considered exposure more closely, there was no overall increase in risk of prostate cancer: SMR = 0.96 (CI 0.68-1.37), based on 31 cases. Subgroup analysis showed an increased risk where the duration of employment was ≤ 9 years: SMR = 2.36 (CI 1.42-3.91), based on 15 cases, but risks were less than expected in groups with longer duration of employment. Furthermore, there was no increasing dose-response relationship according to the cumulative number of fire runs or runs during the first 5 years of employment and there was no relationship with either year of hiring or the type of company (engine, ladder or both) in which the firemen were employed. In all other studies there were no statistically significant increases in risk of prostate cancer. Based on these data it cannot be assumed that a fireman with prostate cancer developed the disease as a result of his employment.

Cancers of the Testes (Table 8)

Six publications were identified that mentioned cancer of the testes in relation to employment as a fireman. These included two mortality cohort studies, three incidence cohort studies and a population-based case-control study. A study of firemen in Wellington, New Zealand found a cluster of 4 cases of testicular cancer between 1980 and 1991 that in comparison with men in the general population of New Zealand showed a statistically increased relative risk: RR = 8.2 (CI 2.2-21) (Bates & Lane, 1995). This study was extended as a historical cohort of all firemen in New Zealand between 1977 and 1995 (Bates et al., 2001). This time, 11 cases were found (excluding 2 of the 4 cases reported in the Wellington cluster). Risk of

testicular cancer was not significantly elevated in this larger study: SIR = 1.55 (CI0.8-2.8). The mortality studies and the third incidence study (Giles et al., 1993) did not find a significantly increased risk of testicular cancers in firemen; however, the numbers of cases were very small. The case-control study included 269 testicular cancer cases and 797 controls matching on age and region in north Germany (Stang et al., 2003). There were three controls and four cases (two seminomas and two embryonal carcinomas, all occurring within the age-range of maximum incidence) who ever worked as firemen, giving an odds ratio (OR) = 4.3 (CI 0.7-30.5). Adjustment for cryptorchidism or family history of testicular cancer did not alter the results. This study focused on occupation and was not originally designed to detect increased testicular cancer risks among firemen. Given that testicular cancers are rare, the opinion of the authors is justified that the numbers of exposed cases and controls were too low to obtain precise effect estimates. The results were, however, consistent with those of Bates et al. (2001). On the other hand, no specific risk factors have been identified for these tumours; therefore, the observed increase in risk in this study currently has no support from what is known of exposures among firemen (although such evidence is not strictly necessary for reaching a conclusion on causality). Furthermore, for two cases (one seminoma, one embryonal carcinoma), histological diagnosis was made after just five and three years of employment, respectively, and at ages (29 and 25 years) of close to maximum age-adjusted incidence, at least for Quebec. Based on these data it cannot be assumed that a fireman with testicular cancer developed the disease as a result of his employment.

Cancers of the Skin (Table 9).

Twelve publications were identified that mentioned cancer of the skin in relation to employment as a fireman. Eight publications were mortality studies and five publications were incidence studies (one of these studies involved both mortality and incidence analyses). Melanoma was specifically mentioned as the diagnosis in six studies, but the diagnosis was not clear in the remainder. In the latter, the ICD-9 codes would often include melanoma (172), but would not be restricted to this code (e.g., 173 was sometimes mentioned). Statistically significant increases in risk were recorded in a proportionality study (Burnett et al., 1994) that listed melanoma as the only skin cancer: PMR = 1.63 (CI 1.15-2.23), based on 38 deaths; a mortality study (Ma et al., 1998) that overlapped the previous one and listed melanoma: MOR = 1.4 (CI1.0-1.9), based on 35 cases; and in a case-control study (Sama et al., 1990) using a cancer registry data base for a diagnosis of melanoma and comparison with white men in Massachusetts: SMORm = 2.92 (CI 1.70-5.03), based on 18 cases. This last study did not find a significant increase in overall risk when the comparison was with police in Massachusetts: SMORp = 1.38 (CI 0.60-3.19), although it remained elevated among those aged 55-74 years: SMORp = 5.13 (CI 1.50-17.50), based on 11 cases.

The other publications did not report any statistically significant increase in risk for skin cancer amongst firemen and, although several recorded risks were greater than one, the elevations were small in the mortality studies and hardly elevated at all in the incidence studies. Demers et al. (1992b) obtained incident data from cancer registries and compared melanoma diagnoses made on firemen with those made on police from the same cities. This is probably the comparison that would introduce least bias and no significant increase in risk of melanoma was observed: SIR = 1.21 (CI 0.68-2.00) based on 15 cases. Based on these data it cannot be assumed that a fireman with melanoma developed the disease as a result of his employment.

Discussion

Consistency and the strength of the observed associations are important factors in the identification of causality. No single study can provide definitive evidence for a relationship and the strength of the association (the size of the relative risk) must be taken into consideration.

In many of the studies listed in the tables 2-9, there were some indications of increased risk, but risks were significantly increased in minorities of studies; some of which were not independent with regard to study populations (Burnett et al., 1994; Ma et al., 1998). Some general problems in the interpretation of epidemiological data will now be discussed.

a. Healthy worker effect.

Fighting fires is a mentally and physically demanding occupation in which burns, falls and crush injuries are commonplace and exposure to smoke and other airborne organic and inorganic substances is part of the job (Gochfeld, 1995). Nevertheless, firemen tend to have a lower mortality rate than the general population, at least during the earlier years of employment. It may be, therefore, that front line firemen are a work population particularly prone to a healthy worker effect, since it appears that many firemen who develop symptoms early in their careers may be moved to other, non-fire-fighting roles (Guidotti & Clough, 1992). This suggestion is supported by the higher prevalence of non-specific respiratory disease amongst firemen who do not actually fight fires (Peters et al., 1974). The healthy worker effect is important during the earlier years of employment, due in large part to a reduction in cardiovascular disease, but the effect tends to disappear with longer employment (Guidotti, 1995). Any effect in late employment could be due to the removal of workers (because they become unfit) from exposure to risk factors predisposing them to early mortality. Bias of this kind, should it occur, is more likely to affect disease categories other than cancer. In the study of Baris et al. (2001) the SMR for all causes of death was significantly reduced during the first 9 years and after 20 years of employment, but not in the employment range 10 – 19 years. These changes seemed to be largely due to changes in SMRs for circulatory disease and ischaemic heart disease and so they are consistent with the suggestions made by Guidotti (1995).

b. Study size

Most of the studies reviewed here are based on small numbers of cases. While some studies included substantial case numbers, particularly for lung cancer, e.g., Ma et al., 1998 (633 white cases) and the overlapping Burnett et al., 1994 (562 cases), many of the remainder relied upon much smaller numbers of cases. Thus, most of the studies lacked sufficient statistical power to detect a possible moderate association (e.g., a 2-fold increase in risk).

c. Occupational classification

There have also been concerns in several studies based on cancer registry data that information about occupation was missing or that misclassification of the reported occupation could have occurred. The probability of such misclassification is even higher for studies based on death certificates. This is because firemen belong to an occupational group that routinely retires early and then pursues another occupation and it is the last occupation that is recorded; however, it was judged that the effect of

such misclassification would be to reduce the strength of the observed association, because it is expected that the misclassification would be non-differential, i.e., it is independent of the cancer diagnosis.

d. Exposure assessment

Generally few studies could address the issue of actual fire-fighting experience among workers employed as firemen, because a fireman might have been fighting fires, or he might have been assigned purely administrative tasks. Such a lack of sub-classification could influence risk ratios, biasing them towards the null. In addition, the small numbers of cases available in most studies precluded meaningful analysis according to a fireman's actual or likely exposure.

A general problem affecting studies with firemen is that reliance by the investigators upon the number of years employed as a surrogate for actual exposures at fires, which probably resulted in misclassification and might not form a sound basis for describing dose-response relationships. Austin et al. (2001a) provide such evidence, as only 66% of fire department personnel were in fact 1st line combat fire fighters and many of these combat firemen moved into non-exposed jobs before the end of their careers. In the same study there was a good correlation between the number of runs to fires and the time spent at fires, and they concluded that the number of runs would be a better surrogate for exposure than years worked as a fireman. However, it was also found that the number of runs may seriously over- or under-estimate the time spent at fires for individual crews, so that time spent at fires is the preferred surrogate. In one example, two crews had almost identical numbers of fire runs, but one spent 1.72 times longer at fires than the other. These results are contrary to the opinion of Guidotti & Goldsmith (2002) who specifically do not advise that job assignments or cumulative number of fire alarms to which firemen were assigned be used as measures of exposure (the only practical basis being, in their estimation, duration of employment). Within the studies reviewed here, Demers et al. (1994) used duration of active duty fighting fires and Baris et al. (2001) used not only duration of employment, but also estimated cumulative number of runs and number of runs during the first 5 years as a fireman. The last estimate of exposure was divided into two groups, low and high and the relative risk was greater in the high number of runs category. Year of first employment as a fireman, which was used in several of the studies, is more a measure of latency than exposure, since job categories are likely to have changed over the years. Interpretation of dose-response associations between fire fighting and a particular malignancy is, however, difficult for most malignancies evaluated here, taking into account the very small numbers of cases available in sub-group analyses, even when only two categories of exposure were compared.

e. Adjustment for confounders

Most of the studies relied on death certificates as the main source of information and consequently no information about possible confounders (e.g., tobacco smoking, diet, exercise and alcohol consumption) was available. The absence of adjustments for these factors is particularly important for cancers of the lung and elsewhere in the respiratory system, oesophagus, stomach, pancreas and prostate. An interesting observation is that in those studies (Sama et al., 1990 and Demers et al., 1994) where both policemen and general population males were used as comparison groups, the risk was either very similar (e.g., lung, pancreas) or weaker (e.g., stomach, prostate) when policemen were used as the reference population. Policemen would be

expected to share many socio-economic factors with firemen and this proximity could make for a more appropriate comparison. .

f. Design weaknesses

Proportional measures can be misleading because their denominator is the total number of cases or deaths from all causes within the same population. The PMR method of estimating risk, as used by Burnett et al. (1994), will overestimate risk if the overall death rate for the occupational group is low, as might be the case for firemen (DeCouflé et al., 1980). To obtain Mortality Odds Ratios (MOR), as used by Ma et al. (1998), the expected numbers were calculated using all causes of death except cancers from the same occupational mortality database from which the firemen's deaths were obtained. As noted for PMRs, this measure relies on death certificate information and is therefore prone to bias because of misclassification of both the cause of death and the exposure. These two studies are interesting, however, because their study populations are overlapping (which would detract from their significance) and have used different analytical methods - which would add to the significance of their common conclusion.

Generally speaking, incidence data obtained from cancer registries form a more reliable and more detailed source of information on which to base analyses (Demers et al., 1992b). In any of these analyses, the characteristics of the reference population is important; it being an advantage to select a reference population as close to the study population as possible. This advantage was maximised in those studies (Sama et al. (1990); Demers et al. 1992a; 1994) that selected police as a reference group in addition to a more general population.

Conclusion

The available epidemiological data do not suggest that any of the malignancies considered here can be assumed to arise in firemen as a result of their occupation. This conclusion is based on the lack of significantly increased risk in most studies and the small magnitude of any increased risk where they did occur. Many of the difficulties in interpreting the cancer data relating to fireman could be addressed in well designed prospective studies of incidence.

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Table 1. Volatile Organic Compounds Consistently found in 9 Municipal Structural Fires.

Chemical	**Concentration Range Found (ppm)**
Propene	0.22 – 21.64
Benzene	0.12 – 10.76
Xylenes (*o*-, *m*-, *p*-)	0.06 – 9.19
1-Butene/2-methyl propene	0.03 – 4.08
Toluene	0.05 – 5.52
Propane	0.03 – 3.63
1,3-Butadiene	0.03 – 4.84
2-Methylbutane	0.004 – 0.43
Ethylbenzene	0.01 – 5.97
Naphthalene	0.01 – 2.14
Styrene	0.003 – 2.01
Cyclopentene	0.002 – 3.29
1-Methylcyclopentene	0.001 – 1.79
Isopropylbenzene	0.0004 – 0.55

***Table 2. Summary characteristics and results of studies on the relation between occupational exposure as a fireman and multiple myeloma.**

Reference	Study base & type	Reference group	Numbers	Risk* (95% C.I. or significance level)	Adjustments & Comments
Aronson et al., 1994	Firemen in Toronto, Ontario, 1959-89	Men in Ontario	5414 firemen with 199 cancer deaths	Multiple myeloma, 1 case, SMR = 0.39 (0.01-2.15)	114008 person-years of follow-up. Dose response analysis non-significant. No information on smoking habits or diet
Demers et al., 1994	Firemen in Seattle & Tacoma, NW USA, 1974-89.	Police in the same cities (p) Men in the same counties (m)	2447 firemen with 244 cancer deaths	Multiple myeloma, 2 cases, SIRm = 0.7 (0.1-2.6)	Small number of cancers
Demers et al., 1993	Case-control study of 5 firemen from 4 states of the USA (WA, UT, GE, MI)	Men of the general population		Multiple myeloma, 5 cases, OR = 1.9 (0.5-9.4) With 10 years and more employment, OR = 2.9 (0.4-21.6)	Response rate 83-89%.
Burnett et al., 1994	Firemen in 27 states of the USA, 1984-90	Men who died in the same 27 states of the USA	Number of firemen not stated. 1636 cancer deaths	Multiple myeloma, 34 cases, PMR = 1.48 (1.02-2.07) <65 years age, 11 cases, PMR = 1.36 (0.68-2.43)	Proportionality study
Ma et al., 1998	Mortality odds ratio study of death certificates of firemen for race-specific cancer risk in 24 states of USA, 1984-93	Men who died from causes other than cancer	6607 deaths of firemen with 1817 white cancer deaths	**WHITE**: Multiple myeloma, 28 cases, MOR = 1.1 (0.8-1.6) **BLACK** Multiple myeloma, 1 case, MOR = 0.8	.
Baris et al., 2001	Historical cohort mortality study of Philadelphia firemen employed 1925-1986	Men in the general USA population	7789 firemen with 2220 deaths	Multiple myeloma, 10 cases, SMR = 1.68 (0.90-3.11) See text for SMRs according to exposure indices	204821 person-years of follow-up. Best estimates of expore because, in addition to duration of employment, the cohort was analysed according to job assignment and number of runs

*C.I. Confidence Interval

MOR = mortality odds ratio; OR = odds ratio; PMR = proportional mortality ratio; SIR = standardised incidence ratio; SMR = standardised mortality ratio; Subscripts: m = men

***Table 3. Summary characteristics and results of studies on the relation between occupational exposure as a fireman and cancers of the respiratory system.**

Reference	Study base & type	Reference group	Numbers	Risk* (95% C.I. or significance level)	Adjustments & Comments
Musk, et al., 1978	Firemen in Boston, MA Cohort 1915-75	Massachusetts men	5655 firemen with 367 cancer deaths	Respiratory, SMR = 0.88, n.s.	No dose-response analysis. Based on death certificates. 104561 person-years actively working; 38414 person-years retirees
Eliopulos et al., 1984	Firemen in Western Australia, 1939-78	Western Australia men	990 firemen with 30 cancer deaths	Respiratory, 7 cases, SPMR = 1.04 (0.42-2.13)	No dose response analysis. Based on death certificates. 16876 person-years
Feuer & Rosenman, 1986	Firemen in New Jersey, 1974-80	NJ police NJ men USA men	No. of firemen not clear. 67 cancer deaths,	Respiratory, 23 cases, PMRp = 1.02, Respiratory, PMR_{NJm} = 0.92 Respiratory, PMR_{USAm} = 0.98 Not significant	Proportional analysis. No indication of a dose-response
Vena & Fiedler, 1987	Firemen in Buffalo, NY, 1950-1979	White USA men	102 cancer deaths	Respiratory, 28 cases, SMR = 0.94 (0.62-1.36)	Not corrected for age. 32858 person-years
Hansen, 1990	Firemen in Denmark, 1970-80	Danish civil servants (military officers, police, prison guards, postmen, etc.)	886 firemen, 21 cancer deaths	Lung cancers, SMR = 1.63 (0.75-3.10) Last 5 years of follow-up: 30-74 years, SMR = 2.20 (0.95-4.34) 60-74 years, SMR = 3.17 (1.17-6.91)	Cohort incomplete. Workers who were active on the day of entry were followed for 10 years. No dose-response analysis. 8625 person-years
Heyer et al., 1990	Firemen in Seattle, WA, 1945-83.	White USA men	2289 firemen, 92 cancer deaths	Lung cancers, 29 cases SMR = 0.97 (0.65-1.39) >65 years, 18 cases, SMR = 1.77 (1.05-2.79) <65 years, 11 cases, SMR = 0.56 (0.28-1.00)	Small numbers of cancer cases, resulting in instability of the SMRs. Dose-response analysis inconclusive. 52914 person-years.
Sama et al., 1990	Firemen in Massachusetts, 1982-86	MA police (p) White MA men (m)	315 cancer deaths	Lung, 71 cases, SMORp = 1.30 (0.84-2.03) SMORm = 1.22 (0.87-1.69)	Case-control study of selected cancers from a cancer registry.
Beaumont et al., 1991	Firemen in San Francisco, California, 1940-82.	White USA men	3066 firemen with 236 cancer deaths	Trachea, bronchi, lung, 60 cases, SMR = 0.84 (0.64-1.08)	Analysis of dose-response inconclusive. Number of person years not stated.
Grimes et al.,	Firemen in Honolulu,	Hawaii men	205 deaths,	All cancers, PMR = 1.19 (0.96-1.49)	Proportionality study.

1991	Hawaii, 1969-88.		including 58 cancer deaths.	Lung, PMR = 1.28 (0.82-2.00)	Small numbers of individual cancers No analysis of dose-reponse
Demers et al., 1992a	Firemen in Seattle, Tacoma & Portland, NW USA, 1945-89	Police in the same cities (p). White USA men (m)	4401 firemen with 291 cancer deaths	Lung, 95 cases, IDRp = 0.95 (0.67-1.33) SMRm = 0.96 (0.76-1.17)	122852 person-years for the 3 cities
Demers et al., 1992b	Firemen and policemen in Seattle & Tacoma WA, U.S.A. 1974-1979	For SIR, male rates for the urban counties of region For SMR, white WA, U.S.A. men	4528 firemen & policemen with 338 cancer cases	Lung, 65 cases, SIR = 0.92 (0.71-1.17) 64 cases, SMR = 1.01 (0.77-1.29)	SIR from cancer registry; SMR from death certificates
Demers et al., 1994	Firemen in Seattle & Tacoma, NW USA, 1974-89.	Police in the same cities (p) Men in the same counties (m)	2447 firemen with 244 cancer deaths	Lung, 45 cases, SIRp = 1.1 (0.6-1.9) SIRm = 1.0 (0.7-1.3)	Sub-group of the preceding study
Giles etal.,1993	Firemen in Melbourne, Australia, 1980-89	Men in Victoria	2865 firemen, 50 cancer cases	Lung, SIR = 0.77 (0.28-1.68)	20853 person-years of observation. Dose-response analysis non-significant
Guidotti, 1993	Firemen in Edmonton & Calgary, Alberta, 1927-87	Men in Alberta	3328 firemen with 92 cancer deaths	Trachea, bronchi, lung, 24 cases, SMR = 1.42 (0.91-2.11)	Follow-up of 96% of the cohort for 64983 person-years. Dose-response analysis inconclusive
Aronson et al., 1994	Firemen in Toronto, Ontario, 1959-89	Men in Ontario	5414 firemen with 199 cancer deaths	Trachea, bronchi, lung, 54 cases, SMR = 0.95 (0.71-1.24)	114008 person-years of follow-up. Dose response analysis non-significant. No information on smoking habits or diet.
Tornling et al., 1994	Firemen in Stockholm, Sweden, 1951-86	Men in the Stockholm region	1116 firemen with 93 cancer deaths	Lung, 18 cases, SMR = 0.90 (0.53-1.42)	
Burnett et al., 1994	Firemen in 27 states of the USA, 1984-90	Men who died in the same 27 states of USA	Number of firemen not stated. 1636 cancer deaths	Lung, 562 cases, PMR = 1.02 (0.94-1.11) <65 years age, 236 cases,PMR = 0.98 (0.86-1.12)	Proportionality study
Deschamps et al., 1995	Firemen in Paris, France, 1977-91	Men in the general population of France; age and cause-specific cancer rates	830 firemen with 18 cancer deaths	Respiratory, 7 cases, SMR = 1.12 (0.45-2.30)	
Finkelstein, 1995	Case-control analysis of death certificates in Hamilton and Sault Ste.	Men who died from causes other than lung cancer	6 firemen cases and 8 firemen	Lung, RR = 1.94 (0.56-6.25)	Small numbers

	Marie, Ontario		controls		
Ma et al., 1998	Mortality odds ratio study of death certificates of firemen for race-specific cancer risk in 24 states of USA, 1984-93	Men who died from causes other than cancer	6607 deaths of firemen with 1883 cancer deaths (1817 white, 66 black)	**WHITE**: Lung + bronchus, 633 cases, MOR = 1.1 (1.0-1.2) **BLACK** Lung + bronchus, 15 cases, MOR = 0.8 (0.5-1.3)	Small numbers for cancers in blacks, leading to instability of the MORs.
Bates et al., 2001	Historical cohort study of all firemen in New Zealand, 1977-95	Men who died from cancer in the same period throughout New Zealand	4221 firemen	Lung, 17 cases, SIR = 1.14 (0.7-1.8)	58709 person-years. Results not changed by analysis for 1990-96 period (Cancer Registry database more complete from about 1990)
Baris et al., 2001	Historical cohort mortality study of Philadelphia firemen employed 1925-86	Men in the general USA population	7789 firemen with 2220 deaths	Lung, 162 cases, SMR = 1.13 (0.97-1.32)	204821 person-years of follow-up. Thus, the largest study available to date. Best estimates of exposure because, in addition to duration of employment, the cohort was analysed according to job assignment and – most importantly – number of runs.

*C.I. Confidence Interval

IDR = incidence density ratio; MOR = mortality odds ratio; PMR = proportional mortality ratio; RR = relative risk; SIR = standardised incidence ratio; SMR = standardised mortality ratio; SMOR = standardised morbidity odds ratio; SPMR = standardised proportional mortality; Subscripts: m = men; p = police

***Table 4. Summary characteristics and results of studies on the relation between occupational exposure as a fireman and oesophageal cancer.**

Reference	Study base & type	Reference group	Numbers	Risk* (95% C.I. or significance level)	Adjustments & Comments
Vena & Fiedler, 1987	Firemen in Buffalo, NY, 1950-1979	White USA men	102 cancer deaths	Oesophagus, 3 cases, SMR = 1.34 (0.27-3.91)	Not corrected for age. 32858 person-years
Heyer et al., 1990	Firemen in Seattle, WA, 1945-83.	White USA men	2289 firemen, 92 cancer deaths	Oesophagus, 1 case, SMR = 0.44 (0.01-2.50)	Small numbers of cancer cases, resulting in instability of the SMRs. Dose-response analysis inconclusive. 52914 person-years.
Beaumont et al., 1991	Firemen in San Francisco, California, 1940-82	White USA men	3066 firemen with 236 cancer deaths	Oesophagus, 12 cases, SMR = 2.04 (1.05-3.57)	Analysis of dose-response inconclusive. Number of person years not stated
Demers et al., 1992a	Firemen in Seattle, Tacoma & Portland, NW USA, 1945-89	Police in the same cities (p). White USA men (m)	4401 firemen with 291 cancer deaths	Oesophagus, 6 cases, SMR = 0.83 (0.30-1.80)	122852 person-years for the 3 cities Data from death certificates
Demers et al., 1992b	Firemen and policemen in Seattle & Tacoma WA, U.S.A. 1974-1979	For SIR, male rates for the urban counties of region For SMR, white WA, U.S.A. men	4528 firemen & policemen with 338 cancer cases	Oesophagus, 5 cases, SIR = 1.06 (0.34-2.47) 5 cases, SMR = 1.13 (0.37-2.63)	SIR from cancer registry; SMR from death certificates
Demers et al., 1994	Firemen in Seattle & Tacoma, NW USA, 1974-89.	Police in the same cities (p) Men in the same counties (m)	2447 firemen with 244 cancer deaths	Oesophagus, 4 cases, SIRm = 1.3 (0.4-3.3)	Sub-group of the preceding study Small numbers for some cancers
Aronson et al., 1994	Firemen in Toronto, Ontario, 1959-89	Men in Ontario	5414 firemen with 199 cancer deaths	Oesophagus, 2 cases, SMR = 0.40 (0.05-1.43)	114008 person-years of follow-up. Dose response analysis non-significant. No information on smoking habits or diet.
Ma et al., 1998	Mortality odds ratio study of death certificates of firemen for race-specific cancer risk in 24 states of USA, 1984-93	Men who died from causes other than cancer	6607 deaths of firemen with 1817 white cancer deaths	**WHITE** Oesophagus, 37 cases, MOR = 0.9 (0.7-1.3) **BLACK** 4 cases, MOR = 1.4	

Bates et al., 2001	Historical cohort study of all firemen in New Zealand, 1977-95	Men who died from cancer in the same period throughout New Zealand	4221 firemen	Oesophagus, 3 cases, SIR = 1.67 (0.3-4.9)	58709 person-years. Results not changed by analysis for 1990-96 period (Cancer Registry database more complete from about 1990)
Baris et al., 2001	Historical cohort mortality study of Philadelphia firemen employed 1925-86	Men in the general USA population	7789 firemen with 2220 deaths	Oesophagus, 6 cases, SMR = 0.56 (0.25-1.24)	204821 person-years of follow-up. Thus, the largest study available to date. Best estimates of exposure because, in addition to duration of employment, the cohort was analysed according to job assignment and – most importantly – number of runs.

*C.I. Confidence Interval
MOR = mortality odds ratio; SIR = standardised incidence ratio; SMR = standardised mortality ratio

***Table 5. Summary characteristics and results of studies on the relation between occupational exposure as a fireman and cancer of the stomach.**

Reference	Study base & type	Reference group	Numbers	Risk* (95% C.I. or significance level)	Adjustments & Comments
Williams et al., 1977	3rd. U.S.A. National Cancer Survey, workers in protection services (firemen, police, etc.). Interview de 58% of 13179 cancer cases in 8 regions of the USA	Men with cancers at other sites		Stomach, OR = 1.04, n.s.	No dose-response analysis
Eliopulos et al., 1984	Firemen in Western Australia, 1939-78	Western Australia men	990 firemen with 30 cancer deaths	Stomach, 5 cases, SPMR = 2.02 (0.65-4.70)	No dose response analysis. Based on death certificates. 16876 person-years
Vena & Fiedler, 1987	Firemen in Buffalo, NY, 1950-1979	White USA men	102 cancer deaths	Stomach, 7 cases, SMR = 1.19 (0.48-2.46)	Not corrected for age. 32858 person-years
Heyer et al., 1990	Firemen in Seattle, WA, 1945-83.	White USA men	2289 firemen, 92 cancer deaths	Stomach, 6 cases, SMR = 1.13 (0.41-2.47)	Small numbers of cancer cases, resulting in instability of the SMRs. Dose-response analysis inconclusive. 52914 person-years.
Beaumont et al., 1991	Firemen in SanFrancisco, California, 1940-82.	White USA men	3066 firemen with 236 cancer deaths	Stomach, 22 cases, SMR = 1.31 (0.82-1.99)	Analysis of dose-response inconclusive. Number of person years not stated.
Grimes et al., 1991	Firemen in Honolulu, Hawaii, 1969-88.	Hawaii men	205 deaths, including 58 cancer deaths.	Stomach, PMR = 0.79 (0.30-2.09)	Proportionality study. Small numbers of individual cancers No analysis of dose-reponse
Demers et al., 1992a	Firemen in Seattle, Tacoma & Portland, NW USA, 1945-89	Police in the same cities (p). White USA men (m)	4401 firemen with 291 cancer deaths	Stomach, 16 cases, SMR = 1.02)0.61-1.73)	122852 person-years for the 3 cities Data from death certificates
Demers et al., 1992b	Firemen and policemen in Seattle & Tacoma WA, U.S.A. 1974-1979	For SIR, male rates for the urban counties of region For SMR, white WA, U.S.A. men	4528 firemen & policemen with 338 cancer cases	Stomach, 15 cases, SIR = 1.75 (0.98-2.89) 12 cases, SMR = 2.04 (1.05-3.56)	SIR from cancer registry; SMR from death certificates
Demers et al.,	Firemen in Seattle &	Police in the same	2447 firemen	Stomach, 8 cases, IDRp = 0.4 (0.1-1.2)	Sub-group of the preceding study

1994	Tacoma, NW USA, 1974-89.	cities (p) Men in the same counties (m)	with 244 cancer deaths	8 cases, SIRm = 1.4 (0.6-2.7)	.
Guidotti, 1993	Firemen in Edmonton & Calgary, Alberta, 1927-87	Men in Alberta	3328 firemen with 92 cancer deaths	Stomach, 3 cases, SMR = 0.81 (0.30-1.76)	Follow-up of 96% of the cohort for 64983 person-years. Dose-response analysis inconclusive
Aronson et al., 1994	Firemen in Toronto, Ontario, 1959-89	Men in Ontario	5414 firemen with 199 cancer deaths	Stomach, 7 cases, SMR = 0.51 (0.20-1.05)	114008 person-years of follow-up. Dose response analysis non-significant. No information on smoking habits or diet.
Tornling et al., 1994	Firemen in Stockholm, Sweden, 1951-86	Men in the Stockholm region	1116 firemen with 93 cancer deaths	Stomach, 12 cases, SMR = 1.21 (0.62-2.11)	Tendency for a dose-response relationship between duration of employment and number of fires attended for cancers of the stomach.
Ma et al., 1998	Mortality odds ratio study of death certificates of firemen for race-specific cancer risk in 24 states of USA, 1984-93	Men who died from causes other than cancer	6607 deaths of firemen with 1817 white cancer deaths	**WHITE** Stomach, 52 cases, MOR = 1.2 (0.9-1.6) **BLACK** 3 cases, MOR = 1.2	
Baris et al., 2001	Historical cohort mortality study of Philadelphia firemen employed 1925-1986	Men in general USA population	7789 firemen with 2220 deaths	Stomach, 24 cases, SMR = 0.90 (0.61-1.35)	

*C.I. Confidence Interval

OR = odds ratio; MOR = mortality odds ratio; PMR = proportional mortality ratio; SIR = standardised incidence ratio; SMR = standardised mortality ratio; SMOR = standardised morbidity odds ratio; SPMR = standardised proportional mortality ratio

***Table 6. Summary characteristics and results of studies on the relation between occupational exposure as a fireman and cancer of the pancreas.**

Reference	Study base & type	Reference group	Numbers	Risk* (95% C.I. or significance level)	Adjustments & Comments
Vena & Fiedler, 1987	Firemen in Buffalo, NY, 1950-1979	White USA men	102 cancer deaths	Pancreas, 2 cases, SMR = 0.94 (0.62-1.36)	Not corrected for age. 32858 person-years
Sama et al., 1990	Firemen in Massachusetts, 1982-1986	MA police (p) White MA men (m)	315 cancer deaths	Pancreas, 6 cases, SMORp = 3.19 (0.72-14.15) SMORm = 0.98 (0.42-2.26)	Case-control stud of selected cancers from a cancer registry.
Beaumont et al., 1991	Firemen in San Francisco, California, 1940-82	White USA men	3066 firemen with 236 cancer deaths	Pancreas, 17 cases, SMR = 1.25 (0.73-2.00)	Analysis of dose-response inconclusive. Number of person years not stated
Demers et al., 1992a	Firemen in Seattle, Tacoma & Portland, NW USA, 1945-89	Police in the same cities (p). White USA men (m)	4401 firemen with 291 cancer deaths	Pancreas, 14 cases, SMR = 0.89 (0.49-1.49)	122852 person-years for the 3 cities Data from death certificates
Demers et al., 1992b	Firemen and policemen in Seattle & Tacoma WA, U.S.A. 1974-1979	For SIR, male rates for the urban counties of region For SMR, white WA, U.S.A. men	4528 firemen & policemen with 338 cancer cases	Pancreas, 9 cases, SIR = 1.06 (0.49-2.01) 10 cases, SMR = 1.11 (0.53-2.04)	SIR from cancer registry; SMR from death certificates
Guidotti, 1993	Firemen in Edmonton & Calgary, Alberta, 1927-87	Men in Alberta	3328 firemen with 92 cancer deaths	Pancreas, 5 cases, SMR = 1.55 (0.50-3.62)	Follow-up of 96% of the cohort for 64983 person-years. Dose-response analysis inconclusive
Aronson et al., 1994	Firemen in Toronto, Ontario, 1959-89	Men in Ontario	5414 firemen with 199 cancer deaths	Pancreas, 14 cases, SMR = 1.40 (0.77-2.35)	114008 person-years of follow-up Dose-response analysis non-significant No information on smoking habits or diet
Demers et al., 1994	Firemen in Seattle & Tacoma, NW USA, 1974-89	Police in the same cities (p) Men in the same counties (m)	2447 firemen with 244 cancer deaths	Pancreas, 6 cases, SIRm = 1.1 (0.4-2.3) SIRp = 1.1 (0.3-5.5)	Sub-group of Demers et al., 1992b
Tornling et al., 1994	Firemen in Stockholm, Sweden, 1951-86	Men in the Stockholm region	1116 firemen with 93 cancer deaths	Pancreas, 5 cases, SMR = 0.84 (0.27-1.96)	
Ma et al., 1998	Mortality odds ratio study of death certificates of firemen for race-specific	Men who died from causes other than cancer	6607 deaths of firemen with 1883	**WHITE**: Pancreas, 88 cases, MOR = 1.2 (1.0-1.5)	Small numbers for some cancers in whites, small numbers for most cancers in blacks, leading to instability of the MORs.

	cancer risk in 24 states of USA, 1984-93		cancer deaths (1817 white, 66 black)	**BLACK** Pancreas, 5 cases, MOR = 2.0 (0.9-4.6)	
Baris et al., 2001	Historical cohort mortality study of Philadelphia firemen employed 1925-1986	Men in the general USA population	7789 firemen with 2220 deaths	Pancreas, 23 cases, SMR = 0.96 (0.64-1.44) See text for SMRs according to exposure indices	

*C.I. Confidence Interval

MOR = mortality odds ratio; SIR = standardised incidence ratio; SMR = standardised mortality ratio; SMOR = standardised morbidity odds ratio;

***Table 7. Summary characteristics and results of studies on the relation between occupational exposure as a fireman and cancer of the prostate.**

Reference	Study base & type	Reference group	Numbers	Risk* (95% C.I. or significance level)	Adjustments & Comments
Williams et al., 1977	3rd. U.S.A. National Cancer Survey, workers in protection services (firemen, police, etc.). Interview de 58% of 13179 cancer cases in 8 regions of the USA	Men with cancers at other sites		Prostate, OR = 0.90, n.s.	No dose-response analysis
Vena & Fiedler, 1987	Firemen in Buffalo, NY, 1950-1979	White USA men	102 cancer deaths	Prostate, 5 cases, SMR = 0.71 (0.23-1.65)	Not corrected for age. 32858 person-years
Beaumont et al., 1991	Firemen in SanFrancisco, California, 1940-82.	White USA men	3066 firemen with 236 cancer deaths	Prostate, 8 cases, SMR – 0.38 (0.16-0.75)	Analysis of dose-response inconclusive. Number of person years not stated.
Grimes et al., 1991	Firemen in Honolulu, Hawaii, 1969-88.	Hawaii men	205 deaths, including 58 cancer deaths.	Prostate, PMR = 2.61 (1.38-4.97)	Proportionality study. Small numbers of individual cancers No analysis of dose-reponse
Demers et al., 1992a	Firemen in Seattle, Tacoma & Portland, NW USA, 1945-89	Police in the same cities (p). White USA men (m)	4401 firemen with 291 cancer deaths	Prostate, 30 cases, IDRp = 1.43 (0.71-2.85) SMRm = 1.34 (0.90-1.91)	122852 person-years for the 3 cities
Demers et al., 1992b	Firemen and policemen in Seattle & Tacoma WA, U.S.A. 1974-1979	For SIR, male rates for the urban counties of region For SMR, white WA, U.S.A. men	4528 firemen & policemen with 338 cancer cases	Prostate, 94 cases, SIR = 1.37 (1.11-1.69) 16 cases, SMR = 1.14 (0.65-1.85)	Comparison of cancer registries and death certificate data for reliability.
Demers et al., 1994	Firemen in Seattle & Tacoma, NW USA, 1974-89.	Police in the same cities (p) Men in the same counties (m)	2447 firemen with 244 cancer deaths	Prostate, 66 cases, SIRp = 1.1 (0.7-1.8) SIRm = 1.4 (1.1-1.7)	Sub-group of the preceding study Small numbers for some cancers
Giles etal.,1993	Firemen in Melbourne, Australia, 1980-89	Men in Victoria	2865 firemen, 50 cancer cases	Prostate, SIR = 2.09 (0.67-4.88)	20853 person-years of observation. Dose-response analysis non-significant
Guidotti, 1993	Firemen in Edmonton & Calgary, Alberta, 1927-87	Men in Alberta	3328 firemen with 92	Prostate, 8 cases, SMR = 1.46 (0.63-2.88)	Follow-up of 96% of the cohort for 64983 person-years.

			cancer deaths		Dose-response analysis inconclusive
Aronson et al., 1994	Firemen in Toronto, Ontario, 1959-89	Men in Ontario	5414 firemen with 199 cancer deaths	Prostate, 16 cases, SMR = 1.32 (0.76-2.15)	114008 person-years of follow-up. Dose response analysis non-significant. No information on smoking habits or diet.
Tornling et al., 1994	Firemen in Stockholm, Sweden, 1951-86	Men in the Stockholm region	1116 firemen with 93 cancer deaths	Prostate, SMR = 1.21 (0.66-2.02)	
Ma et al., 1998	Mortality odds ratio study of death certificates of firemen for race-specific cancer risk in 24 states of USA, 1984-93	Men who died from causes other than cancer	6607 deaths of firemen with 1883 cancer deaths (1817 white, 66 black)	**WHITE**: Prostate, 189, MOR = 1.2 (1.0-1.3) **BLACK** Prostate, 16, MOR = 1.9 (1.2-3.2)	Small numbers for some cancers in whites, small numbers for most cancers in blacks, leading to instability of the MORs.
Baris et al., 2001	Historical cohort mortality study of Philadelphia firemen employed 1925-1986	Men in the general USA population	7789 firemen with 2220 deaths	Prostate, 31 cases, SMR = 0.96 (0.68-1.37) See text for SMRs according to exposure indices	
Bates et al., 2001	Historical cohort study of all firemen in New Zealand, 1977-95	Men who died from cancer in the same period throughout New Zealand	4221 firemen	Prostate, 11, SIR = 1.08 (0.5-1.9)	58709 person-years. Results not changed by analysis for 1990-96 period (Cancer Registry database more complete from about 1990)
Zeegers et al., 2004	Prospective case-cohort study of prostate incidence in the Netherlands		763 prostate cancer cases, but only 2 were firemen	Prostate, 2, RR = 0.59 (0.05-6.33) after adjustments	8690 person-years in the sub-cohort. Adjustments made for age, consumption of fruit, vegetables, dairy products, meat, alcohol, cigarette smoking, family history of prostate cancer, education and level of physical activity.

*C.I. Confidence Interval

IDR = incidence density ratio; MOR = mortality odds ratio; PMR = proportional mortality ratio; RR = relative risk; SIR = standardised incidence ratio; SMR = standardised mortality ratio; Subscripts: m = men; p = police

***Table 8. Summary characteristics and results of studies on the relation between occupational exposure as a fireman and cancer of the testes.**

Reference	Study base & type	Reference group	Numbers	Risk* (95% C.I. or significance level)	Adjustments & Comments
Giles etal.,1993	Firemen in Melbourne, Australia, 1980-89	Men in Victoria	2865 firemen, 50 cancer cases	Testes, SIR = 1.15 (0.13-4.17)	20853 person-years of observation. Dose-response analysis non-significant
Aronson et al., 1994	Firemen in Toronto, Ontario, 1959-89	Men in Ontario	5414 firemen with 199 cancer deaths	Testes, 3 cases, SMR = 2.52 (0.52-7.37)	114008 person-years of follow-up. Dose response analysis non-significant. No information on smoking habits or diet.
Bates & Lane, 1995	Firemen in Wellington, New Zealand, with testicular cancer, 1980-91	Men in the general population of New Zealand	4 cases	Testes, RR = 8.2 (2.2-21)	
Ma et al., 1998	Mortality odds ratio study of death certificates of firemen for race-specific cancer risk in 24 states of USA, 1984-93	Men who died from causes other than cancer	6607 deaths of firemen with 1817 white cancer deaths	**WHITE** Testes, 1 case, MOR = 0.6	
Bates et al., 2001	Historical cohort study of all firemen in New Zealand, 1977-95	Men who died from cancer in the same period throughout New Zealand	4221 firemen	Testes, 11 cases, SIR = 1.55 (0.8-2.8)	58709 person-years. Data do <u>not</u> include 2/4 testicular cancer cases occurring in the Wellington cluster (Bates & Lane, 1995) Results (notably for testes) not changed by analysis for 1990-96 period (Cancer Registry database more complete from about 1990)
Stang et al., 2003	Population-based case-control study in north Germany. Testicular cancers diagnosed between 01 July 1995 and 31 December 1997	Men matched for age and place of residence	4 firemen with testicular cancer among 269 cases and 3 firemen among 797 controls	Testes, 4 cases, OR = 4.3 (0.7-30.5) Duration of work as a fireman: ≥ 10 years ever. 2 cases, OR = 3.0 (0.2-45.5); ≥ 5 years before reference date, 3 cases, or = 3.1 (0.4-24.4).	Statistical power too low for the specific study of testicular cancers in firemen (not the primary objective).

*C.I. Confidence Interval

MOR = mortality odds ratio; OR = odds ratio; RR = relative risk; SIR = standardised incidence ratio; SMR = standardised mortality ratio.

***Table 9. Summary characteristics and results of studies on the relation between occupational exposure as a fireman and cancer of the skin.**

Reference	Study base & type	Reference group	Numbers	Risk* (95% C.I. or significance level)	Adjustments & Comments
Feuer & Rosenman, 1986	Firemen in New Jersey, 1974-80	NJ police NJ men USA men	No. of firemen not clear. 67 cancer deaths,	Skin, PMR = 1.35, n.s	Proportional analysis. No indication of a dose-response
Sama et al., 1990	Firemen in Massachusetts, 1982-86	MA police (p) White MA men (m)	315 cancer deaths	Melanoma, 18 cases, SMORp = 1.38 (0.60-3.19) SMORm = 2.92 (1.70-5.03)	Case-control study of selected cancers from a cancer registry.
Beaumont et al., 1991	Firemen in SanFrancisco, California, 1940-82.	White USA men	3066 firemen with 236 cancer deaths	Skin, 7 cases, SMR = 1.69 (0.68-3.49)	Analysis of dose-response inconclusive. Number of person years not stated.
Demers et al., 1992a	Firemen in Seattle, Tacoma & Portland, NW USA, 1945-89	Police in the same cities (p). White USA men (m)	4401 firemen with 291 cancer deaths	Skin, 6 cases, IDRp = 1.12 (0.27-4.76) SMRm = 0.98 (0.36-2.13)	122852 person-years for the 3 cities Data from death certificates
Demers et al., 1992b	Firemen in Seattle & Tacoma, NW USA, 1944-79, followed 1974-1989	Police in the same cities	4528 firemen with 362 incident cancers	Melanoma, 5 cases, SMR = 1.64 (0.53-3.83) Melanoma, 15 cases, SIR = 1.21 (0.68-2.00)	Mortality data from death certificates Incidence data from cancer registry
Demers et al., 1994	Firemen in Seattle & Tacoma, NW USA, 1974-89.	Police in the same cities (p) Men in the same counties (m)	2447 firemen with 244 cancer deaths	Melanoma, 9 cases, SIRp = 1.0 (0.4-1.8) SIRm = 1.2 (0.6-2.3)	Sub-group of Demers et al., 1992a . Small numbers for some cancers
Giles etal.,1993	Firemen in Melbourne, Australia, 1980-89	Men in Victoria	2865 firemen, 50 cancer cases	Melanoma, SIR = 1.08 (0.35-2.53)	20853 person-years of observation. Dose-response analysis non-significant
Aronson et al., 1994	Firemen in Toronto, Ontario, 1959-89	Men in Ontario	5414 firemen with 199 cancer deaths	Melanoma, 2 cases, SMR = 0.73 (0.09-2.63)	114008 person-years of follow-up. Dose response analysis non-significant. No information on smoking habits or diet.
Burnett et al., 1994	Firemen in 27 states of the USA, 1984-90	Men who died in the same 27 states of USA	Number of firemen not stated. 1636 cancer deaths	Skin, 38 cases,PMR = 1.63 (1.15-2.23) <65 years age, 24 cases, PMR = 1.67 (1.07-2.48)	Proportionality study

Ma et al., 1998	Mortality odds ratio study of death certificates of firemen for race-specific cancer risk in 24 states of USA, 1984-93	Men who died from causes other than cancer	6607 deaths of firemen with 1883 cancer deaths (1817 white, 66 black)	**WHITE**: Melanoma, 35, MOR = 1.4 (1.0-1.9)	Small numbers for some cancers in whites, small numbers for most cancers in blacks, leading to instability of the MORs Overlaps with Burnett et al., 1994.
Baris et al., 2001	Historical cohort mortality study of Philadelphia firemen employed 1925-1986	Men in the general USA population	7789 firemen with 2220 deaths	Skin, 10 cases, SMR = 1.16 (0.64-2.20)	
Bates et al., 2001	Historical cohort study of all firemen in New Zealand, 1977-95	Men who died from cancer in the same period throughout New Zealand	4221 firemen	Melanoma, 23, SIR = 1.26 (0.8-1.9)	58709 person-years. Results not changed by analysis for 1990-96 period (Cancer Registry database more complete from about 1990)

*C.I. Confidence Interval

IDR = incidence density ratio; MOR = mortality odds ratio; PMR = proportional mortality ratio; SIR = standardised incidence ratio; SMR = standardised mortality ratio; SMOR = standardised morbidity odds ratio; Subscripts: m = men; p = police