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A Study of Smoke Inhalation and its Effects on Firefighters

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Introduction

One of the primary hazards to which firefighters are exposed is smoke. Evaluation or assessment of the risk factors associated with toxic properties of the variations of smoke produced in fires, which may range from vegetation, to wood, to petroleum products and the myriad of chemical compounds used in industry and commerce today, is a complex area. Smoke causes many problems for firefighters in their attempts to control fires and rescue people from fires. In addition to the problems of the heat generated and reduced visibility, firefighters have been concerned about the effects of repeated exposure to smoke, whether adverse effects can result from cumulative or repeated exposure.

The literature describes an environment of carbon monoxide (CO) and other toxic substances that firefighters throughout the world work in during firefighting. If BA does not protect firefighters from this environment, when their exposure to CO is greater than the allowable limit, they will suffer

adverse health effects. The health effects that have been reported include heart disease, acute and chronic lung disease, increased decline in Forced Expiratory Volume in one second, some forms of cancer and increased mortality rates. CO is a possible direct cause of some of the cumulative effects of smoke exposure. For example, CO is suspected to be a contributory cause of development of atherosclerosis, and exposure to CO has produced electrocardiogram changes and angina in subjects with known coronary artery disease.

Background:

Smoke has many other components as well as CO. Smoke also has an irritant effect. It is probable that continuing exposure to some of these components is responsible for a decline in respiratory function and other diseases that have been associated with firefighting. CO uptake is a useful indicator of smoke inhalation.

Not all firefighters suffer these health effects. Other factors that protect firefighters are the reduction in exposure by use of BA for protection, the individual firefighters fitness level and the general health of the firefighter.

The results of the situation found in the MFS are presented and discussed in the remainder of this paper.

LITERATURE REVIEW

The literature has been reviewed from a firefighter's health perspective. The review considered general health effects of CO, the effects of firefighters'

exposure to CO as a product of combustion and to other airborne toxic substances encountered by firefighters during their work. Studies of cardiovascular and respiratory disease were considered. Asphyxiation, measurement of carboxyhaemoglobin, the effects of fitness and stress on firefighters health was also reviewed. Finally cancer and mortality studies of firefighters were reviewed.

The Effects of CO Exposure

CO is an odourless, colourless and non-irritant substance and is a gas produced at fires. Carbon dioxide (CO2) is the end product of combustion of carbonaceous matter. It acts as an asphyxiant by displacement of oxygen in the air. CO is produced in smaller quantities but it is more toxic and can produce health effects at much lower concentrations than CO2. Nightingale (1980) stated in a review that the primary organs affected by exposure to CO are the heart and the brain, with the effects caused by impaired oxygen delivery. A study by Norman and Halton, (1990) found that CO has teratogenic and embryo toxic effects in humans. A Swedish review of CO by Rylander (1981) found that it affected the heart, central nervous system (CNS) and the foetus.

A prevailing view of CO toxicity is that it has a greater (200 times) affinity than oxygen for haemoglobin and that anaemic hypoxia is the cause of the poisoning. An atmosphere of 1000 ppm of CO when inhaled for a period of 2 hours can cause death (Zaren, 1973).

However, Runciman and Gorman (1993) have reported that oxygen delivery to the brain and heart increases, not decreases during CO insults. Runciman (1992) has shown, in anaesthetised rabbits that the effects of CO on the brain are different from the effects produced by equivalent levels of nitrogen. This effect cannot therefore be explained by either hypotension, hypoperfusion or by a reduction in oxygen delivery. Gorman et, al. (1993) suggest that the theory that best fits the evidence is the CO toxicity is due to direct tissue toxicity not an anaemic hypoxia mechanism.

Increased coronary blood flow was also found by Runciman (1992) when conscious sheep were exposed to an atmosphere with a 1-% CO concentration. However, Aronow and Isbell (1973) in a double blind study with angina patients found that when the subjects were breathing 50 ppm of CO for 2 hours, angina was induced faster and after less cardiac work than when breathing pure air. This study demonstrated the potential problems for a person with pre-existing coronary artery disease when exposed to CO before performing a physical task. The coronary reserve capacity of an individual exposed to CO with cardiovascular disease would be reduced. Further stress that demanded increased coronary flow would be above the maximum attainable and would cause ischaemia of the myocardium. This could lead to irreversible cardiac damage or transient damage that would reduce the overall functional ability of the heart to deliver blood to the rest of the body (Astrup, 1973).

Another common belief was that direct tissue toxicity occurred with CO exposure (Apthorp et al., 1958). Runciman and Gorman's (1993) view is that

uncertainty over the mechanism of toxicity of CO exists and that more research is needed to test the developing theories. Runciman (1992) suggests that it is possible that CO may cause abnormal activation of guanylyl cyclase, the enzyme that provides secondary messages to cyclic guanine monophosphate.

CO is believed to cause an atherogenic effect when inhaled either at a low exposure over a long time or at a high exposure over a short time (Hawkins, 1976). Wanstrup (1969) found atherosclerosis-related degeneration in rabbits that were exposed to 90 ppm of CO for 3 months. Scott (1978) has interpreted this by suggesting that CO plays a role in the development of focal vascular changes of the atherosclerotic type. Increased endothelial permeability leads to focal oedema and subsequent regenerative and reparative vascular changes eventually leading to plaque formation. Using monkeys, Thomsen (1974) found that after an exposure of 250 ppm of CO for two weeks, changes had occurred in the coronary arteries consistent with developing atherosclerotic disease.

Nightingale (1980) observed that the individual subject's susceptibility was also an important factor. Categories of subjects with increased susceptibility include those with a history of myocardial infarction, anaemia, or a tendency to arterial vasospasm, and pregnant women.

The hazard that CO produces at smouldering fires has been reviewed by Quintiere et al (1982). Quintiere et al concluded that the small amount of smoke in such cases could give a misleadingly low impression of the risk.

Barnard (1979c) found CO readings of up to 3000 ppm in a study of 25 fires in Los Angeles.

Even at exposure levels insufficient to cause impairment of consciousness, there are causes for concern at the possibility of adverse effects on persons with certain risk factors, and from the possibility of long term effects. In response to these concerns, occupational exposure standards have been set at levels well below that likely to cause impaired consciousness or impaired cerebral function.

A summary of recommended exposure limits for occupational exposure to CO is provided in Table 1.

***Table 1*: A List of Recommended Exposure Limits for CO**

Reference	*ppm*	*Recommended Maximum Exposure Limits*
Worksafe, 1991	50	Time Weighted Average
Pervious standard	400	Short Term Exposure Level
ACIGH, 1991	35	Permissible Exposure Limit
	200	Short Term Exposure Level
Runciman, 1992	20	Time Weighted Average
	200	Short Term Exposure Level
NIOSH, 1973	35	Time Weighted Average
Worksafe, 1995	30	Time Weighted Average
Present standard	200	Short Term Exposure Level
English Standard	50	Time Weighted Average
German Standard	30	Time Weighted Average

Swedish Standard	35	Time Weighted Average

Firefighters' Exposure to CO and to Other Airborne Substances

Cotes and Steel (1987) listed some of the substances firefighters are exposed to during firefighting activities and discussed the potential health problems. CO exposure was considered the greatest hazard. They state that CO may cause hypoxia and irritant gases from the fire may cause pulmonary oedema (which can occur up to 24 hours after the fire). Firefighters can develop acute symptoms that include eye pain, chest pain, cough, breathlessness, headache, nausea, hypoxaemia and burns.

Brotherhood (1989) studied 27 bush firefighters and measured their exposure to CO at bush fires. He concluded that CO was unlikely to be a hazard to bushfire-firefighters. The maximum level of carboxyhaemoglobin that he measured in non-smoking firefighters was 7%. This finding could be interpreted differently if Runciman and Gorman's (1993) warning was accepted. They stated, "little or nothing is known with any confidence about CO poisoning." The COHb level of 7% could be enough to interact with other factors such as the workload of the firefighter and possible undiagnosed coronary heart disease.

In a study of firefighters and navy reserves Sammons and Colman (1974) measured carboxyhaemoglobin levels. Both the control and exposed groups

gave blood samples at 28-day intervals throughout the 5-month duration of the study. Sammons and Colman found that non-smoking firefighters had carboxyhaemoglobin levels of 5% compared to 2.3% for a control group of navy reserves. The findings in the exposed group of firefighters were considered to be most likely due to exposure while firefighting.

Levy et al (1976) found a consistent increase in mean carboxyhaemoglobin levels after exposure to smoke at fires in both smoking and non-smoking firefighters. COHb levels were 4.4% and 8.7% for non-smokers and smokers respectively. Nonsmokers were defined in this study as those who smoked less than ten. This value is 2.5 times higher the value for the Hathaway value. Thomas (1984) reported an approximate level of COHb of 5% for non-smokers and 11% for smokers after firefighters were exposed at fires.

Takano (1981) found the mean concentration of CO taken from expired breath of the non-smoker group of Tokyo firefighters was 3.5%. The unexposed firefighters on standby had a mean concentration of 1.5%. This is higher than British reports (Blanks et al, 1987) but similar to the American studies cited in this section. This could be due to the way the fire services in the different countries fight fires. For example, the use of BA in Japan follows the traditional American approach, whereas the British use an entry control procedure that results in the firefighters wearing the BA in more situations than in the traditional American approach. The traditional American approach to BA use differs from the British approach mainly because the Americans did not use an entry control procedure. This resulted in individual firefighters

making their own decision as to whether the smoke was heavy enough to require the use of BA.

Levine (1975) reported that exposures of firefighters to CO at major fires had sometimes produced levels of COHb of 15-20%. Levine reported on a study of civilian fatalities of fires that showed an additive effect between the level of COHb absorbed by the blood and the degree of narrowing of the coronary arteries. He concluded that the exposure of the firefighters measured was significant, and he stated that people who are compromised by coronary artery disease are significantly more sensitive to CO in the atmosphere than people without coronary artery bug.

Table 2 shows a summary of typical carboxyhaemoglobin levels that have been measured in a number of environments, including bushfire, structural fire and general environments.

Table 2: **A Summary of Typical Carboxyhaemoglobin Levels that have been Recorded in People that have been Exposed to Various Fires**

Reference	*Subject*	*COHb*	*Comments*
Brotherhood 1989	Bush firefighters	Maximum 7%	Non-smokers
Hathaway 1988	General public	2% to 10% 1%	Smoker Non-smoker
Takehito 1981	Tokyo firefighters	3.4% 0.9%	On-duty On-standby (Non-smokers)
Thomas 1984	Before f/f After f/f Before f/f After f/f	3% 5% 7% 11%	Non-smoker Non-smoker Smokers Smokers
Sammons 1974	Firefighters navy reserves	5.0% 2.3%	Non-smokers Smokers
Levy 1976	Before f/f After f/f Before f/f After f/f	2.5% 4.4% 5.5% 8.7%	Non-smokers Non-smokers Smokers Smokers

Blanks et al (1987) studied 100 firefighters in the UK and found raised COHb levels due mainly to cigarette smoking, and no elevation in COHb from

occupational exposure was found. This could be an indication that the British system of BA Procedure is effective, or more effective than the American systems. The Americans have traditionally not controlled the use of BA. They have had "compulsory mask rules" but these rules have usually been interpreted as a requirement to put on a mask only when in thick smoke.

Young et al. (1980) provided a description of three different groups of firefighters. The first group was called the old "smoke chewers". This group of firefighters did not usually use BA. The second was the group of firefighters with about ten or more years service whom usually use BA. The third group was the young firefighters who always use BA. They found that the prevalence of respiratory disease in NSW firefighters studied was less than in those reported in Boston firefighters. This was due to lower exposure as measured by the number of fires attended, an average of 10 per year in NSW compared to 40 in Boston and also a possible survivor effect in NSW firefighters.

These groups have different exposure levels and the levels of exposure reported in the literature do not describe the attitude of the individual firefighter. This creates a need for interpretation as to the relevance of the results found in the literature. I have observed, from 24 years experience as a firefighter, that most MFS firefighters are in group two.

Expected levels of COHb in smokers and non-smokers were reported by Hathaway et al (1988) in a study that did not expose subjects to fires. They reported that smokers often had COHb levels between 2% and 10%, but some smokers have been reported to have a COHb level of 18%, whereas

the average level of non-smokers is 1%. These levels of COHb should be considered in the context of the other levels reported in Table 2. Non-smoking firefighters had levels after exposure at fires that are in the range that Hathaway et al (1988) reported in smokers. Cigarette smokers provide a model for studying the effects of chronic low-dose CO exposure in humans. Firefighter's exposure, which has been discussed in the literature review above, has similar exposures to that of people smoking. In both cases people are exposed to CO and a variety of other toxic gases. From the literature reviewed a reasonable case can be made that firefighting, like smoking, is hazardous to health.

Griggs (1977) discussed the use of BA and found that it provided full protection from CO exposure when it was used. The rate of ventilation in firefighters while working hard produced a significant increase in COHb uptake. This occurred even when firefighters were exposed to relatively low levels of CO (around 200 ppm). Field evaluation of BA has been conducted by NIOSH (Spandoni et al., 1976).

In summary, CO exposure has still been occurring over the last 25 years, sufficient to cause elevated COHb. The two main issues are firstly, firefighters attitude to the wearing of BA, and secondly the protection factor that is provided when BA is worn. BA when used provides the protection that is required from CO and other airborne contaminants. The literature demonstrates that over the last 25 years firefighters still have been exposed to CO, resulting in elevated COHb levels. This has occurred despite the worldwide trend for increased use of protective BA by firefighters in this

period. Significant exposure can occur in the absence of thick smoke. BA provides protection, but effective use is highly dependent on procedures and attitude to use.

. Measurement Of CO And Carboxyhaemoglobin (COHb)

Measurement of subjects' exposure to CO is usually undertaken by analysis of blood to determine the carboxyhaemoglobin level. An alternative method of measurement involves analysis of expired breath. The National Institute for Occupational Safety and Health, updated 16/8/1996, references 13 studies that use blood analysis, and six references that use expired breath.

Ringold et al (1962) studied the basis of using expired air to estimate recent exposures to CO and associated levels of carboxyhaemoglobin. He described the factors that affect CO uptake into the blood as being the ventilation rate, the concentration of carboxyhaemoglobin, blood volume and the diffusion capacity of the lung.

Coburn et al (1965) analysed the relationship between the rate of endogenous CO production, blood COHb percentage saturation and body CO stores, and CO exchange via the lungs. The principle physiological parameters were found to be: the rate of CO production, alveolar ventilation, diffusing capacity of the lungs, mean oxygen tension in the pulmonary capillaries and the concentration of CO in the inspired air.

Stewart and Stewart (1975) described the analysis of expired breath that provides a rapid estimate of carboxyhaemoglobin for firefighters. They showed the operational significance of carboxyhaemoglobin testing. Stewart et al (1976) described rapid estimation of carboxyhaemoglobin in firefighters and graphed CO in alveolar air in ppm versus carboxyhaemoglobin in blood. Mols et al (1988) also studied the relationship between blood carboxyhaemoglobin levels and measurement of CO in expired breath. Lauwerys (1985) explained the equilibrium relationship between carboxyhaemoglobin in blood, haemoglobin and CO levels in alveolar air. Jarvis and Russel (1980) provided a description of a non-invasive method testing for carboxyhaemoglobin levels using an Ecolyzer. The Ecolyzer instruction manual includes a ppm to percentage carboxyhaemoglobin graph.

The literature has shown that exposure of firefighters to CO at fires can be rapidly and accurately estimated by measuring firefighters' carboxyhaemoglobin levels. This method can be used at all fire service activities and allows a comparison to be made of the effectiveness of the protection factors eg. BA used at the activity.

Other Toxic Substances Encountered

In addition to CO firefighters encounter other airborne contaminants at the fire ground. Purser (1988a) described the toxic effect of exposure to other substances at structural fires. Generally CO and hydrogen cyanide (HCN) are produced. The combustion process, depending on a number of factors can

produces irritants such as toluene di-isocyanate (TDI), acrolein and hydrochloric acid (HCl).

Birky and Clarke (1981) also reviewed toxic products such as CO, HCN, heavy metals and irritants (HCl) inhaled during fires. The gas mixture was found to be dependent on the substances present, the temperature, and oxygen levels. The issue of whether or not "supertoxicants" exist has caused some concern because of the possibility that a highly toxic substance might kill in very low concentrations and make escape of the victims impossible (Clarke, 1983). No consensus on the existence of "supertoxicants" has been found.

Hurst and Jones (1985) reviewed the products that evolved from heated wood, coal and PVC and found that CO is produced by all three materials.

Experimental modelling of the development of fires in rooms has been conducted using materials that are commonly found in houses. Morikawa and Yanai (1986) conducted this kind of experiment and found that the toxic gases evolved from air-controlled fires in a model of a room included CO which was the principal toxicant. HCN and acrolein were also found.

Purser (1988b) reported that narcotic gases found at fires include CO, HCN and carbon dioxide. Products of combustion cause death and incapacitation in fires by two main processes - narcosis and irritancy. Using observations from both small and large-scale fires and the known effect on fire gases on human beings, primates and rodents he developed models to predict time to incapacitation in human fire victims. From the models he summarised that

after two minutes in a room on fire, smoke would make escape difficult because of visual and irritancy problems. By four minutes, skin burns would be expected with a room temperature in excess of 200 degrees Celsius. At five minutes, the victim is expected to lose consciousness. With a six minutes exposure, the models suggest that the victim would not be expected to survive. A person should leave a room on fire within the first 3 minutes to expect to escape safely.

Sampling of fire atmospheres by using personal sampling devices worn by firefighters has been conducted by Gold et al (1978) who identified and measured toxic combustion gases. CO was one of the gases monitored at levels that could cause acute toxicity. The other gases detected were oxygen, carbon dioxide, nitrogen dioxide, HCl and HCN.

An exposure assessment of Buffalo (USA) firefighters was carried out by Brandt-Rauf (1988) who found frequent exposure to significant concentrations of hazardous materials including CO, benzene, SO2, HCN, aldehydes, HCl, dichlorofluromethane and particulates. In many cases, it was reported that BA was not used due to low smoke intensity and therefore these levels represent a significant risk to firefighters.

Table 3 provides an overview of the toxic exposures that firefighters could encounter in their work environment.

T*able 3*: An overview of toxic exposures that firefighters could encounter in their work environment.

Author	*Source*	*Chemical*	*Comments and findings*
Esch 1977	Burning PVC	PVC	A discussion on toxicity and its effect on firefighters.
Birky & Clarke 1981	Fires	CO, HCN, heavy metals, irritants, HCl	CO was found to be the principal toxicant.
Clarke 1983	Fires	Super-toxicants	Not identified.
Hurst & Jones 1985	Heated wood, coal, PVC	CO	CO is a product of all 3 materials.
Lowry et al 1985	Fires	CO, HCN, HCl, Aldehydes Organic Compounds	Free radicals were found. The fire environment is complex. Firefighters need to use BA.
Alexeeff & Lee 1986	Smoke	PVC PI	A study in rats that demonstrated injury to the epithelial surface, trachea and bronchi.
Morikawa & Yanai 1986	Fires in a model room	CO HCN acrolein	CO found to be the principle toxicant.
Froines et al 1987	Diesel exhaust in fire stations	Diesel emissions	Found to be a hazard at fire stations.
Purser 1988b	Fires	CO, HCN, CO2 and low O2	Narcotic and irritancy effects found.
Cotes & Steel 1987	Fires	CO, CO2, HCl phosgene HF, NOx Acrolein NH4 aldehydes Isocynate HCN, SO2 lipid aerosol	The gas mixture is dependent on the substances present, the temperature and oxygen levels. The combustion products may react chemically or biologically and synergistic effects may occur.
Hughes & Taylor 1972	Fires	CO, HCN, HCl, HF, SO2, NOx, NH3, CL2, COCl2	UK and USA survey of toxic gasses. Combination of gasses more toxic than a single gas.

Burgess & Treitman 1979	Structural fires	CO and other gasses	A report on air contaminants in structural fires.

The exposure of firefighters to diesel emissions from internal combustion engines in fire stations has been studied. Froines et al (1987) found significant exposure that required preventive programs to be implemented. This type of exposure does not occur in a fire environment that is, by its nature, an uncontrolled environment, but at the fire station, which should be a safe environment. Lucas (1982) discussed poor ventilation in the fire station. CO and nitrogen oxides were found in excess. This is a recent, but significant concern for firefighters. Another recent British study (Socrates, 1998) discussed firefighters exposure to CO and other products of combustion that may occur at fire stations and in fire appliances while driving. The main chemical that caused concern was polycyclic aromatic hydrocarbons (PAH) due to its carcinogenic nature.

Thus the literature reports that CO is only one of the toxic substances to which a firefighter may be exposed, although CO is present at all fires. It is prudent to assume that if a firefighter has been shown to have been exposed to CO at a fire, then it is probable that exposure to other gases has occurred also. Therefore raised carboxyhaemoglobin levels in firefighters can be used as a useful predictor of exposure to other toxic substances produced in the fire gases. Although the most likely time when exposure can occur is during firefighting, exposure to toxic gases must be considered at other times, such as at the fire station.

Cardiovascular Disease

Ischaemic heart disease continues to be the leading cause of death among middle-aged people in industrialised countries. The rate has declined has been known for years to be due to cholesterol levels, smoking and hypertension (Elford and Yeo, 1988).

One possible explanation for smoking as a risk factor is the increased exposure in smokers to CO. There are two possible mechanisms that could be working here. One is that CO may be atherogenic (Levine, 1975). The other possibility is that CO in smoke directly impairs oxygenation. Although a CO challenge also causes increased perfusion of the heart muscle this compensatory mechanism is impaired by coronary artery disease (Runciman, 1992, Griggs, 1977).

Myocardial ischaemia may accelerate in working firefighters who have subclinical coronary heart disease (Griggs, 1977). The view expressed was that exertion levels and ventilation rates are so great during firefighting that even low levels of atmospheric CO can give rise to dangerous levels of carboxyhaemoglobin in minutes. If this is true then firefighting may be a risk factor in subjects with recognised or unrecognised ischaemic heart disease.

Barnard (1978) reported that the death rate from atherosclerotic heart disease in the 55-64 age group of firefighters is more than twice as high as in all other groups studied. Two factors, exposure to CO and high levels of adrenalin that are associated with atherosclerotic heart disease, were also found in the firefighters studied. He also found that electrocardiogram stress tests of firefighters show abnormally high incidence of heart disease.

Ferguson (1981) cautioned that asymptomatic firefighters after the age of 55 years who may be required to exert themselves maximally as part of their job are likely to have significant coronary artery disease, which may not be detected. They are able to do less work, they have a higher risk of incapacity and therefore it is argued should retire at 55 years of age. He supported his opinion with the death rate reported by the National Fire Protection Association statistics that showed 44% of firefighters' deaths were cause by heart attacks.

Dibbs et al (1982) carried out a normative ageing study in the USA and found no difference between firefighters and others. Peters (1982) argued that Dibbs used an unrepresentative group of firefighters (mostly officers) in his study. Levine (1982) also pointed out other problems with Dibbs' study: no base line was established at the start of the study and firefighters were too young to show the full effects of the exposure.

Blast furnace workers were found to be at no greater risk of arterial disease than other steelworkers, despite finding raised COHb levels (Jones and Sinclair, 1975). So besides the possibility that CO exposure in firefighters is a problem other occupational factors may also be part of the problem.

Reich (1953) provided an early description of heart disease in firefighters. This research covered heat, cold, trauma, shock (electric), burns, smoke, gases and aggravation of pre-existing conditions.

Krohn (1971) called for research and discussed high-resolution electrocardiogram and its use with Detroit firefighters. The "Heart Disease

Study of New York City Firefighters," published by the Uniformed Firefighters Association Local 94 and the Uniformed Fire Officers Association Local 854 (1978) described the firefighting environment, mortality, CO exposure and case studies from Los Angeles, New York and Toronto. This report was used to argue a case for providing workers' compensation benefits for firefighters with heart disease.

Since the firefighting environment contains smoke, CO and other toxic gases a preventive health program was required to reduce the rate of heart disease. Davolos (1972) argued that strategies for prevention of ischaemic heart disease should include the following areas: selection, re-examination, no smoking, weight control, fitness, diet, BA, safety officer, monitoring under operational conditions.

Barnard (1975) suggested that a fit firefighter is less likely to suffer the health effects of the profession. The introduction of a no smoking policy, provision of support and health promotion for firefighters, improved systems of work and training, and enhanced use of protective BA would reduce the health effects of the profession. Kurt (1977) added to Barnards' suggestions. His program to lower risk factors of firefighters to heart disease included: screening (health monitoring), no smoking programs, extended use of BA, a fitness program, rehabilitation, and a prospective study of firefighters disease. Balanoff (1976) found the mean age of American firefighters killed by heart attack to be 51.3 years. They averaged 22 years of service and 60% of them smoked.

A review by Gouldman and Cook (1984) estimated that more than half the reduction in ischaemic heart disease mortality between 1968 and 1976 in the

USA was related to changes in lifestyle (less smoking and reduced cholesterol levels). Other factors, such as medical intervention, pre-hospital resuscitation and treatment of blood pressure, were other factors that also helped to reduce ischaemic heart disease mortality. Leon et al (1987) concluded from a study that questioned men on their leisure-time physical activity that leisure-time physical activity has a modest inverse relationship to coronary heart disease and overall mortality in middle aged men at high risk for coronary heart disease.

In the USA, firefighters as a group have been concerned with heart disease since the late 1950's. "The Heart Disease study of New York City Firefighters", (UFA, 1978), is a good example of the concern that was raised. In most states of the USA there is presently an assumption under Workers Compensation Law that heart disease, unless it can be proven otherwise is compensable. In Britain and Australia the legislation is different. In Britain, Blanks et al (1987) suggested that smoking and other life style factors (eg. obesity) were more important in the cause of heart disease than firefighting because exposure had been reduced with the enhanced use of BA. On the other hand Britain's extremely high rate of heart disease would make any occupational factor contributing to heart disease difficult to observe.

Goldstein and Niaura (1992) and Niaura and Goldstein (1992) conducted a literature review to better characterise the current state of knowledge regarding the relationship between psychological factors and cardiovascular disease. They summarised the review by making six points. First, epidemiological evidence suggests that the "type A behaviour pattern" is a

risk factor for the development of coronary artery disease. Second, people with a major depressive disorder are at increased risk of cardiovascular morbidity and mortality. Third, there is considerable evidence linking disturbing situations and life events with myocardial ischaemia, ventricular arrhythmia and sudden death. Fourth, occupational factors such as high job strain, low control, and few possibilities for promotion are significantly associated with excess risk of coronary artery disease. Fifth, low levels of social support appear to interact with life stress, job strain and type A behaviour pattern to increase risk for coronary artery disease events. Finally, there is evidence that increasing blood pressure reactivity is a risk factor for the development of hypertension. Additionally, personality or coping style is linked to increased blood pressure.

The stress factors should be considered in addition to firefighter exposure to smoke. Many firefighters would have some of the following risks, type A behaviour patterns, exposure to disturbing situations, high job strain, low job control and hypertension. These firefighters would be expected to be at increased risk of coronary artery disease.

Oklahoma City firefighters were studied by Abrams (1974) who found that cardiovascular and renal disease indicated a significant decrement in life span for firefighters. Over the period of the study cardiovascular and renal disease did not decrease in line with the American average.

Donnan et al (1982) conducted a cohort study of UK firefighters from 1965-1979 and found a slight increase in ischaemic heart disease. A second report (Donnan, 1987) that extended the study a further 7 years found a decline in

ischaemic heart disease rate. This was possibly due to health checks and the selection criteria. Effect of cigarette smoking and the decline in the smoking rate was thought to be an important factor affecting the change from the first to the second study.

These mortality studies reviewed can be divided into two types. One has a control group consisting of the general male population of the region or state. In this type of study there is usually no significant difference found. The other studies have controls consisting of a comparable group. This often in practise is the local police force. In these studies there have been some significant differences found; however, there needs to be more of these types of studies to establish if this significance exists beyond the regions of the studies and into other countries, cultures and operational systems.

In summary, there is some evidence of increased risk of cardiovascular disease in firefighters. Such a finding appears plausible, because of the likely atherogenic effect of CO, because of ischaemia induced by CO in subjects with coronary artery disease, and from the effects of stress and heat. However a conclusive finding is made difficult because of the confounding effects of conventional risk factors (eg. High cholesterol, high blood pressure and smoking behaviour).

Respiratory Disease

Impaired Respiratory Function

A decline in lung function, measured by forced vital capacity (FVC) and forced expired volume in one second (FEV1) normally occurs with age. The normal rate of decline has been estimated by Knudson et al (1983) in a prospective longitudinal study of 697 randomly selected non-smoking white subjects. They found an annual decrement of 27 ml/year in FVC and FEV1.

Several studies have examined whether firefighting is associated with acceleration of the age-related decline in respiratory function. Thomas (1971) described a study on firefighters where pulmonary function tests were carried out as soon as possible after exposure at fires. FVC and FEV1 showed a statistically significant decrease but returned to the pre-exposure levels within 24 hours.

When 30 firefighters were followed for 18 months after smoke exposure during firefighting, Unger (1980) found a decrease in lung function related to repeated insults over time rather than sudden decrease associated with specific exposures. After a particular fire in Houston, 160 out of the 175 firefighters who attended required medical treatment. Out of this group 30 were able to participate in a follow-up study of pulmonary function. The mean recorded decrement in FVC was 62 ml and FEV1 was 122 ml over an 18-month period.

A longitudinal study of 890 UK firefighters (Oakes, 1980) found that the decline in FEV1 (90 ml/ year) and FVC (110 ml/year) was larger than expected. FEV1 and FVC fell rapidly after 40 years of age, but for no obvious

reason according to the author. The length of service did not effect lung function except after 20 years service.

Sparrow et al (1982) undertook a longitudinal analysis of 168 firefighters and 1474 non-firefighters. The results confirmed earlier reports (Thomas, 1971) (Sidor and Peters, 1974) (Loke et al, 1980) (Peters et al, 1974) (Musk et al, 1982) of chronic effect on firefighters' pulmonary function and suggest an association between firefighting and increased respiratory symptoms and disease independent of smoking. Firefighters had greater reduction in pulmonary function than non-firefighters within each smoking status group. Firefighters averaged an 18-ml/year greater decrease in FVC and a 12-ml/year greater decrease in FEV1 than non-firefighters after adjustment for smoking status.

In a study by Sheppard et al (1986) changes in FEV1 and FVC in firefighters after attending fires were compared with changes across shifts without fires. Following attendance at fires, 24% of firefighters showed a fall in FEV1 and/or FVC of greater than 2 standard deviations, compared to only 1% after shifts without fires. In 13 firefighters with significant falls who were retested 3 to 18.5 hours later, 4 were still more than 2 standard deviations below baseline. The decrement showed no association with baseline airway responsiveness (measured by testing with methacholine challenge); however the airway responsiveness was found to be increased following attendance at fires in two cases. The authors concluded that the decrements in lung function were not due merely to irritant bronchoconstriction.

The first year of a prospective study of Boston firefighters' pulmonary function was reported by Peters et al (1974). He reported that frequency of exposure to combustion products as measured by the number of fires fought is associated with an accelerated loss of lung function. The rate of loss in pulmonary function observed was more than twice the expected rate. The mean FEV1 decrease found by Peters (1974) was 68 mls/year and the mean FVC decrease was 77 ml/year that was described as resembling the rate seen in patients with chronic obstructive lung disease.

In a follow-up report the observed annual rate of decrements for a three-year period was 30ml/yr for FEV1 and 40 ml/yr for FVC (Musk et al, 1977a). The authors suggested that the lower rate of decline could have been due to "selection factors", that is, those who exhibited symptoms may have resigned, retired prematurely, arranged transfers to less active units, or were promoted away from busy units.

A further follow-up study of Boston firefighters aimed to test the effect of the "selection factor" by measuring impairment of lung function at the time of retirement. (Musk et al, 1977b) The study population was reduced from a possible 211 firefighters who retired between 1970 and 1975 to 109 subjects. This cohort did not show that retired firefighters have severely impaired respiratory function.

After a six year follow-up study (Musk et al, 1982), decrement of lung function was observed in firefighters at a rate of 36 ml/year (FEV1) and 29 ml/year (FVC). The use of BA and the selection out of those with impaired lung

function from active firefighting appeared to reduce the large decrement that was found by Peters et al (1974).

Acute changes in lung function were studied when thirty-nine firefighters' FEV1 and FVC were measured at the start of each shift and upon return from a fire call (Musk et al, 1979). A decline in FEV1 in excess of 100 ml was recorded in 30% of observations. COHb levels and samples of smoke were analysed for HCl, HCN, NO2 and CO. Repeated episodes of irritation of the bronchial tree may explain the origin of the chronic effect of firefighting on the respiratory system and pulmonary function that have been mentioned above.

Cotes and Steel (1987) reviewed a range of studies and concluded that a greater than expected decline in FEV1 per year had been recorded in some studies, but other studies found the decline in FEV1 per year no greater than expected.

In summary, studies that have measured FEV1 and FVC decline have shown that if measurements are taken within a few hours of the exposure significant decrement can be observed; however recovery often takes place. The literature suggests that firefighters' exposure is associated with acute decrement post exposure and a probable acceleration of age-related decrement in spirometry values. These studies also add weight to the view that firefighters have traditionally been exposed to smoke and the methods of protection from smoke exposure have not been effective.

Chronic Obstructive Airways Disease

Sidor and Peters (1974a) studied Boston firefighters and reported that the occupational effects of firefighting are underestimated in cross-sectional studies because of selection factors. In their cross-sectional study Sidor and Peters, (1974b) determined the prevalence of chronic non-specific respiratory disease in firefighters, and found that experienced firefighters had a higher prevalence than new firefighters of same age. Smoking history partly obscured the occupational effect.

Table 4: **Prevalence Rate of Chronic Non-specific Respiratory Diseases in the Boston Fire Department, 1970 (Sidor and Peters, 1974b)**

Disease Classification	*Crude rate (n)*
No disease	76.5% (1352)
Chronic non-productive cough	2.5% (44)
Chronic Bronchitis	11.9% (211)
Chronic Obstructive Airway Disease	5.0% (88)
Chronic Bronchitis + Chronic Obstructive Airway Disease	3.5% (62)
Other	0.6% (11)

Young et al (1980) reported on the prevalence of respiratory disease in 193 NSW firefighters. Twenty-one per cent of firefighters in the study had chronic bronchitis and six per cent had chronic obstructive airway disease. The prevalence of chronic respiratory disease was mostly associated with smoking.

Fifty-four firefighters were investigated by Loke et al (1980) for signs of small airway disease. They found both smoking and service for more than 25 years to be associated with small airways disease. One episode of acute exposure to smoke was found to produce irreversible pulmonary injury in one firefighter.

This is similar to a recent (1997) incident in Adelaide where a firefighter who was not wearing BA at a car fire inhaled smoke, required admission to an intensive care unit of a local hospital suffering from pneumonia. His exposure at the fire did not immediately effect him but within 20 hours he required hospitalisation.

Tashkin et al (1977) performed lung function tests on 21 Los Angeles firefighters who had been exposed to a fire containing PVC, one month after the exposure no chronic symptoms or impairment was found in a subsequent test.

Cotes and Steel (1987) reviewed a range of studies and concluded that that some firefighters developed chronic bronchitis, chronic airflow limitations or disease of the lung parenchyma. They suggested that in some cases the cause would have been toxic gas exposure, while in others it could have been due to smoking.

A long term follow up study was conducted on 96 firefighters using the British Medical Council questionnaire for respiratory symptoms (Horshfield et al, 1988a). No evidence of chronic lung damage was found. This was attributed to the selection of fit firefighters for the service, continued physical training and the regular use of BA. In a subsequent clinical study Horshfield found no evidence of chronic lung damage in firefighters. This study measured FEV1, FVC, peak expiratory flow, flow at 50% and 25% remaining vital capacity, airways resistance and alveolar mixing efficiency (a measure of small airways function). (Horshfield et al, 1988b)

In a study of British Naval firefighters Minty et al (1985) performed both lung function test and a measurement of alveolar-capillary permeability to determine the effects of chronic exposure to smoke. The measurement of permeability was determined from the rate of transfer from the lung into the blood of a hydrophilic substance. It was found that non-smoking naval firefighting instructors who had chronic exposure to smoke had changes in the permeability of the alveolar-capillary barrier and showed a significant difference to other non-smokers. All other lung function tests had values within the normal range.

Rosenstock et al (1990) examined mortality rates from non-malignant respiratory disease. Compared to police officers, firefighters had a significantly decreased overall death rate from all causes (SMR = 82). However an excess of deaths among firefighters from chronic non-malignant respiratory disease was found (SMR = 141). The reasons given for their conclusions were that in the past increased risk to firefighters have been masked because suitable control groups were not used. Police were seen as a better control group than the general population because smoking rates was similar between firefighters and police, the absence of excess in other smoking related outcomes and biological plausibility.

In summary, some of these studies have found, not surprisingly, that smoking was associated with chronic obstructive airway disease. Studies of chronic obstructive airway disease have found some evidence of disease and changes to the lungs but overall the evidence is not conclusive. These studies are mostly subject to the limitations of cross sectional design. This

and the confounding influence of smoking make firm conclusions not possible. The British Studies should be more similar to the Adelaide experience than the American studies since procedures for BA use in Adelaide were similar to the British but different to the Americans. It could be concluded from the British studies that normal range responses add support to the theory that Adelaide firefighters would not be exposed to sufficient excess exposure to smoke.

The likelihood of occupationally caused respiratory disease in firefighters depends on the effectiveness of their BA and the suitability of the BA procedure. However, the most important protection factor is whether or not the firefighter uses the BA for the complete period of potential exposure.

Cancer

Firefighters are by the nature of their work exposed to a large range of chemical carcinogens. Although most chemicals have not been tested for their toxic effects there are a number of chemicals that arise as the products of combustion that have been shown to be carcinogenic. These chemicals include benzene, formaldehyde, PAH and asbestos.

Chemicals that are present at a fire can cause harm to a firefighter. The factors that influence the amount of harm include the concentration of the exposure, duration of exposure, respiratory rate, type of breathing apparatus and type of personal protective equipment used. There is additionally the

possibility of a synergistic effect among the chemicals. Firefighters are usually exposed through inhalation and occasionally skin absorption.

Mastromatteo in 1959 studied Toronto City firefighters (n=271) and found a highly significant excess of cardiovascular and renal deaths, along with a statistically significant increase for all causes of deaths combined. Later in the same year he reviewed the then existing studies on firefighters mortality. He found reduced mortality from respiratory disease and tuberculosis, no difference for cancer, and increased mortality for cardiovascular and renal disease. In 1966 he gave a general description of health aspects of firefighting and included the problem of asthma. This study found no increase in overall cancer deaths. It is historically important because these firefighters' years of service occurred before the widespread use of synthetic chemicals.

Musk et al. (1978) found no strong association (SMR=91) between firefighting and death from cardiovascular, respiratory or malignant disease in Boston Firefighters (n=2470). Excess mortality from cancer of the rectum was found (SMR=153).

Schulte and Ehrenberg (1983) were called in by the local Fire Service to investigate a chemical fire at Chester Pennsylvania. They found a statically significant excess incidence of all cancer. Although carcinogens were found at the fire, the excess incidence in cancer was not due to this fire.

Lewis et al (1983) reported on LA firefighters where they found increased rates of brain, prostate, lung and colon cancer. Overall the cancer rate was found to increased over time.

Eliopulos et al (1984) studied Western Australian firefighters and found that mortality from all causes was less than expected (SMR=80) when compared to the Western Australian male population (n=990). The proportion of deaths was higher than expected for cancer of the stomach (PMR=202), large intestine (PMR=159), lymphatic and haematopoietic systems (PMR=188) and other cancers (PMR=297) of which 3 out of 4 were brain tumors. Analysis in this study found indications of the healthy worker effect. Overall this study does not provide strong evidence for an occupational association to cancer.

Feuer and Rosenman (1986) used proportionate mortality ratios instead of standardised mortality ratios to compare police mortality to firefighter mortality. This method was used to obtain a better comparison (since the two groups are similar except for their exposures), than firefighters with the general population. The proportion of death was found to be higher for skin cancer (PMR=270) and leukaemia (PMR=276).

Vena and Fiedler (1987) concluded that Buffalo firefighters (n=1867) cancer mortality was significantly raised in long-term firefighters and the risk of mortality from all malignant neoplasms (SMR=220 after 40 + years) tends to increase with latency. The proportion of death was statistically significant for cancer of the colon (overall SMR= 183, 40+ years SMR=471) and bladder (overall SMR=284, 40+years SMR = 571). The proportion of death was higher for cancer of the rectum (SMR = 208), oesophagus (SMR = 134), and kidney (SMR = 130). Brain cancer's SMR overall was 236, however it was statistically significant for firefighters with between 20 and 29 years of service (SMR = 375).

Rosenstock et al (1988) carried out a mortality study among three American (Pacific Northwest) cities. The control group was the police from the same cities. They described excess cancer in firefighters. The types of cancers that "deserve special attention" were brain, lymphatic and haematopoietic tissue.

Zahm (1989) reported on the findings of the Missouri cancer registry where lung cancer incidence in firefighters was 1.6 times the average rate of all other occupations studied. The shortcomings of this study are that only 60 cases were in the group that contained firefighters and that the listed occupation was that which the individual had at time of diagnosis.

Sama et al (1990) reported on Cancer Incidence among Massachusetts firefighters (n=315) between 1982-86. They used the police and the state male population as their controls. They argued that incidence data have several advantages over mortality data. Incidence data are able to provide better diagnostic information than death certificates; and are more useful for the study of non-fatal cancers. A disadvantage of this study was possible under-reporting of occupational category. Grouping all firefighters together can cause a dilution effect. Age-adjusted standardised mortality odds ratios (SMOR) compared the observed odds of cancer in exposed group to expected odds from each of the two comparison groups. When the state population was used as the control, statistically significant SMORs were observed for melanoma (SMOR = 292) and bladder cancer (SMOR = 159). Non-Hodgkin's lymphoma was elevated but not statistically significant (SMOR = 159). When the police were used as a control, statistically significant results were found for bladder cancer (SMOR = 211) and non-Hodgkin's lymphoma

(SMOR = 327). Elevated non-significant results were seen for pancreatic cancer (SMOR = 319), leukaemia (SMOR = 267) and brain cancer (SMOR = 152).

A cohort mortality study (n=2289) of Seattle firefighters by Heyer et al (1990), 1945-1983 found overall elevated SMRs for brain cancer (SMR = 218), leukaemia (SMR = 173) and multiple myeloma (SMR = 225). Statistically significant results were seen among firefighters with 30 + years of service (SMR = 503), multiple myeloma (SMR = 989) and in firefighters older than 65 years (SMR = 177).

Hansen (1990) studied Danish firefighters (n=886); he found an overall statistically significant excess of firefighter deaths from cancer (SMR = 173). Excess in lung cancer (SMR=220) was found; the increase was statistically significant in age group 60-74 (SMR = 317).

Beaumont et al (1991) used a historical cohort to study San Francisco firefighters n=(3066), using the United States Death rate as a control. They found significant elevation of three causes of death: oesophageal cancer (12 deaths observed v 5.9 expected), cirrhosis and other liver diseases (59 observed v 26 expected) and accidental falls.

A Victorian study conducted by Staples et al. (1993) when compared to the general population found standardised incidence ratios elevated for cancer of the prostate (SIR = 209), Upper digestive tract (SIR = 146), and colon and rectum (SIR = 136) and for non-Hodgkin's lymphoma (SIR = 185) For

firefighters age greater than 65 years statistically significant results were found for all cancers (SIR = 214) and for colorectal cancer (SIR = 365).

A retrospective cohort study of firefighters (Demers et al., 1992) found a statically significant excess of brain cancer tumours (SMR = 209). Lymphatic/haematopoietic (SMR = 131) and prostate cancers were found in excess. Firefighters less than 40 years old had an increase of brain cancer (SMR = 375) and firefighters with over 30 years employment had an increased rate of leukaemia (SMR = 260).

Burnett et al. (1994) studied firefighters from 27 American states. The cause of death was determined for 5,744 firefighters who died between 1984-1990. The study found elevated Proportionate Mortality Ratios (PMR). The strengths of this study include its size and the geographic range of the firefighters studied. (The PMR is found by calculating the proportion of all deaths due to a specific cause in the study population, and comparing it with the expected proportion, which is usually derived from total population data.)

Guidotti (1995) reviewed the occupational mortality literature regarding firefighters. He concluded that there was some association between firefighting and

- lung cancer,
- sudden death, myocardial infarction, fatal arrhythmia occurring on or soon after near maximal stress on the job,
- aortic aneurysm, (incomplete evidence)

- cancer of the genitourinary tract (kidney, ureter and bladder), (strong evidence)
- cancer of the colon and rectum
- acute lung disease

Landrigan et al (1995) reviewed the literature and concluded that firefighters have been shown to be at increased risk of leukaemia, lymphoma, multiple myeloma, melanoma and cancer of the urinary tract, stomach, colon, rectum, prostate and brain.

One limitation of the some studies is evaluating a changing population over an extended period of time. Not only are different procedures used in different locations, but over time procedures change and therefore exposure has varied.

One recent study (Guidotti, 1993) has found that there is a possible increased risk of lung cancer among firefighters. This could be interpreted as a weak occupational effect with a strong confounding effect of smoking. A possible reason for this is the "healthy worker effect" (Eliopullos, 1984, McMichael, 1988), since firefighters are healthier than the general population to whom they have been usually compared.

It could be expected that morbidity and mortality rates would be lower among firefighters than in the general population because they are selected at time of employment to befit and healthy. A study may show no difference in mortality or morbidity rates between firefighters and the general population, when in

reality, the firefighters may be sustaining higher mortality and morbidity rates than would be expected in a similar healthy group.

Additionally, only healthy firefighters stay in the job. Those who become ill may leave the fire service without the disability being recorded. Others may leave seemingly healthy, only to suffer the long term effects later when the relationship to fire service exposure is less obvious.

These studies and reviews have been used in North America as the medical evidence that has supported presumptive cancer legislation. In 1982 workers' compensation was amended to include cancer as a compensable disease for firefighters in California and in 1984 The Pension Code in Illinois was also amended to include cancer as an occupational disease.

In summary, the cancer literature has developed over the years to the state where an association between firefighting and increase risk of cancer can be argued. The types of cancer where this statement is strongest include leukaemia, non-Hodgkin's lymphoma, multiple myeloma, brain and bladder cancer (Golden et al, 1995). The evidence of an association between firefighting and rectal, colon, stomach, prostate cancers and melanoma is weaker but still plausible. Overall the cancer evidence adds support to the view that exposure to smoke has occurred.

Conclusion of Literature Review

CO exposure at fires is sufficient to cause increased COHb levels in firefighters, although smoking is, in most circumstances, a more important consideration. Firefighting and the associated exposure to smoke, although not usually enough to cause impaired consciousness is of concern for the following reasons:

- CO is atherogenic
- CO exposure will cause angina in persons with coronary artery disease
- Firefighting sufficient to cause increased COHb levels is demonstrated to be accompanied often by exposure to other contaminants, which may have irritant or narcotic effects
- The empirical evidence suggests that exposure to smoke causes
  - a reduction in FEV1 and FVC
  - possible but not strong support for increased occurrence of respiratory disease (although the studies are inconclusive, due to their cross-sectional design and the confounding effects of smoking)
  - possible cardiovascular disease
  - cancer

The evidence that cancer has been caused by exposure during firefighting has been not strong but recent reviews suggest that firefighters may be at increased risk of leukaemia, lymphoma, multiple myeloma, melanoma and cancer of the urinary tract, stomach, colon, rectum, prostate and brain.

Although studies do not specifically address the protection effect of BA, some inferences can be drawn. For example, a comparison between the experience in the UK and North America suggest that BA is critical, particularly its manner and likelihood of use. In the US use is a matter of individual discretion. It is shown that CO levels can be high even when the smoke level is low. Thus it tends to be under-utilised unless there are regulatory requirements for its use and high compliance.

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