

SKIN CANCER

IARC SUPPLEMENT 7

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Things you need to do to file your Cancer Presumptive Claim

1. Call your immediate supervisor, and your Local IAFF representative to let them know you're filing a claim. They will also assist you through this process. Fill out an Injury report. This form is a DWC-1, and available online. Fill it out according to the Power point attached.
2. Send in, to your Human resources or Workers Comp Carrier, the paperwork given. These should be the NAWCJ document, CHAPTER 607 , House Bill 1388," Lemaster's" Meta-Analysis Study, and also any IARC monographs that are relevant to prove your case.
3. Also send a pre-employment physical from your department or any physical that showed normal findings prior to your diagnosis. Preferable to supply the pre-employment physical.
4. Keep in mind that if you're denied, to follow the Chapter 607 Presumptive Power point and involve your association to assist with your claim.
5. All claims denied must be denied in writing, with that written denial sent to the Texas Dept. of Insurance Per HB 1388. If denial has PLN-1 at the bottom left corner of denial letter, there is no timetable limitation to file appeal, PLN-11 has 15 days.
6. Gather all receipts related to any out of pocket expense you may have incurred.
7. Download records or online calendars to show your leave usage, so it may be converted to Occupational from Sick time instead.
8. Remember Occupational time is Tax Free! You may need to amend a tax return to recover those funds.

Contact Robert Webb if you need any information or have any questions
817-999-0573

THE BIG 6

Six qualifying criteria are needed in order to qualify your cancer as a Workers Comp claim.

- (1) The necessity that the firefighter suffers from the type of cancer listed. This list is the IARC monographs. The cancer will need to be identified within the monographs and that information submitted.
- (2) The precise occupation of the public safety employee who has contracted cancer. Firefighters are universally covered, Texas legislatures have added EMTs and other similar employees.
- (3) The firefighter's pre-claim physical exam failed to reveal pre-existing cancer. This can be a pre-hire physical, or an earlier physical given by the fire department or city which showed no cancer or illness present.
- (4) The firefighter's current work status. Is he or she still working, laboring somewhere else, or even retired? As of 2016, there is no provision for retired firefighters unless the disease manifested during employment years.
- (5) Time of manifestation of the disease. Most cancer presumption statutes will require that, before the presumption is available, the employee have labored in his or her position for a certain period of time, and/or that the disease have manifested itself within a certain period of time. In Texas that period of time is five years.
- (6) Time of incurrence of the cancer.

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urinary bladder and the adrenal glands; however, because of the lack of matched controls, it could not be concluded whether tumour induction was due to a combined effect of the three chemicals or of any one of them⁴.

C. Other relevant data

Neither chromosomal aberrations (in two patients) nor sister chromatid exchanges (in three patients) were induced following administration of 5-fluorouracil⁵.

5-Fluorouracil induced micronuclei but not specific locus mutations in mice treated *in vivo*. It induced aneuploidy, chromosomal aberrations and sister chromatid exchanges in cultured Chinese hamster cells. It did not induce sex-linked recessive lethal mutations in *Drosophila*, but caused genetic crossing-over in fungi. Studies on mutation in bacteria were inconclusive⁵.

References

- ¹IARC Monographs, 26, 217-235, 1981
- ²Boice, J.D., Greene, M.H., Kechn, R.J., Higgins, G.A. & Fraumeni, J.F., Jr (1980) Late effects of low-dose adjuvant chemotherapy in colorectal cancer. *J. natl Cancer Inst.*, 64, 501-511
- ³Ferguson, T. (1980) Prevention and delay of spontaneous mammary and pituitary tumors by long- and short-term ingestion of 5-fluorouracil in Wistar-Furth rats. *Oncology*, 37, 353-356
- ⁴Habs, M., Schmähl, D. & Lin, P.Z. (1981) Carcinogenic activity in rats of combined treatment with cyclophosphamide, methotrexate and 5-fluorouracil. *Int. J. Cancer*, 28, 91-96
- ⁵IARC Monographs, Suppl. 6, 316-318, 1987

FORMALDEHYDE (Group 2A)

A. Evidence for carcinogenicity to humans (*limited*)

A number of epidemiological studies using different designs have been completed on persons in a variety of occupations with potential exposure to formaldehyde¹⁻²⁴. Cancers that occurred in excess in more than one study are: Hodgkin's disease, leukaemia, and cancers of the buccal cavity and pharynx (particularly nasopharynx), lung, nose, prostate, bladder, brain, colon, skin and kidney¹. The studies reported are not entirely independent; the plant studied by Liebling *et al.*² and Marsh^{1,3} is also included in the study by Blair *et al.*⁴; the case-control study of Fayerweather *et al.*⁵ includes some subjects who were later studied by Blair *et al.*⁴. Detailed estimates of formaldehyde exposure levels were made in the studies of British chemical workers⁶, US formaldehyde producers and users⁴, Finnish wood workers⁷ and US chemical workers⁵, and for the case-control studies of Vaughan *et al.*^{8,9} and Hayes *et al.*¹⁰.

In the study of US producers and users of formaldehyde, 11% of the subjects were not exposed, 12% had an estimated time-weighted average (TWA) exposure of <0.1 ppm (<0.12 mg/m³), 34% a TWA of 0.1-<0.5 ppm (0.12-<0.6 mg/m³), 40% a TWA of 0.5-<2 ppm

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In further analyses of the Exxon refineries and chemical plants in Baton Rouge, LA, Baytown, TX, and Bayway/Bayonne, NJ, mortality was examined by occupation and work site (Hanis *et al.*, 1985b). Directly adjusted death rates for each subgroup of interest and for the total US population were calculated using the age, sex, race and calendar year distribution of the total cohort as a standard; thus, direct comparisons could be made between mortality rates in cohort subgroups and in the US population by calculating ratios of the directly adjusted rates. Workers were classified as having been 'potentially exposed' or 'unexposed' on the basis of their longest-held job. The 'exposed' category included those who had worked as process operators, mechanical workers and labourers (75% of the study population); while the 'unexposed' category included primarily white-collar office workers (22% of the population). Cause-specific cancer rates were higher among potentially exposed workers than among the unexposed for every cancer site except brain, but none of the site-specific rate ratios was significantly different from 1.0. Directly adjusted death rates were consistently greater than those for the total US population only for renal cancer in each of the three plants. The death rates for pancreatic cancer were higher than the US rates among employees at the Baton Rouge and Baytown plants only, and elevated rates of large intestinal cancer occurred at the Baytown and Bayway/Bayonne plants.

A series of investigations of mortality has been performed among members of the Oil, Chemical and Atomic Workers international union (OCAW) in Texas (Thomas *et al.*, 1980, 1982a,b, 1984). In all of these reports, proportionate mortality among male members of the OCAW was compared with that among US men, adjusting for age, race and calendar period.

The first report concerned 3105 Union members in Texas whose deaths in 1947-77 while actively employed were reported to OCAW and whose death certificates could be located (90%; Thomas *et al.*, 1980). Of the white OCAW members, 1722 had held blue-collar jobs in petroleum refineries and petrochemical plants, primarily in maintenance and production (Thomas *et al.*, 1982a), and had significant excess frequencies of deaths from cancers of the digestive and respiratory systems, skin and brain (ICD8 191, 192).

Subsequent analyses were limited to three petroleum refineries located in the Beaumont/Port Arthur area of the Texas Gulf Coast (Thomas *et al.*, 1982a,b, 1984) and included 1194 retired workers as well as those who had died while actively employed between 1943 and 1979. Among 2509 deceased men who had been employed by the three refineries combined (Thomas *et al.*, 1982a,b), the adjusted PMRs using national rates for all causes of death were significantly elevated for all cancers as well as for cancers of the stomach, pancreas, skin (ICD8 172, 173), prostate and brain (ICD8 191, 192) and for leukaemia. Nine deaths from multiple myeloma were observed and 4.6 were expected, but the PMR was not significant. When national cancer rates were used to calculate proportionate cancer mortality ratios (PCMRs), these ratios were also elevated but significantly so only for brain and leukaemia in whites. When county cancer mortality rates were used, none of the PCMRs was significantly raised. A detailed examination of brain tumour mortality in whites indicated that OCAW members had had elevated frequencies of mortality from benign and unspecified tumours of the brain as well as those specified on death certificates as malignant. [The Working Group noted that, of the 2509 deaths studied,

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1.2 Composition of fire smoke

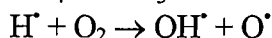
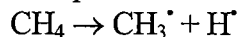
1.2.1 Fire chemistry

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



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

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



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

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

The burning of plastics typically produces voluminous amounts of soot, together with higher levels of hydrogen cyanide (HCN), hydrochloric acid (HCl) and acrolein ($\text{CH}_2=\text{CHCHO}$) than the burning of materials such as wood, and fossil fuels. More smoke evolves from fires involving aromatic polymers, such as polystyrene, compared to aliphatic polymers, such as polyethylene.

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

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

1.2.2 *Modern versus pre-modern fires*

All types of fire release toxic and carcinogenic substances, including benzene, 1,3-butadiene, and formaldehyde. The focus has generally been on substances having short-term acute effects: carbon monoxide (CO), carbon dioxide, hydrogen cyanide, nitrogen oxides (NO_x), sulfur dioxide (SO₂) and hydrogen chloride. With the increasing use of polymers in building construction and furnishings, there is concern that the burning of these new materials might release large quantities of other highly toxic substances (Austin *et al.*, 2001b).

Combustion and pyrolysis products from newer building materials and furnishings were believed to be more toxic than smoke from fires in buildings built before these materials became commonplace, and more toxic than smoke from wildland fires (Betol *et al.*, 1983; Alarie, 1985). However, many of the carcinogenic products of combustion identified are volatile organic compounds and are common to most burning materials. In a more recent study, no new or unusual non-polar volatile organic compounds (VOCs) were observed in current structural fires compared to the combustion of wood (Austin *et al.*, 2001b, 2001c). Adding polyvinyl chloride (PVC) to the fire load at simulated apartment fires was observed to significantly increase levels of polychlorinated phenols (IARC Group 2B), while polycyclic aromatic hydrocarbon (PAH) levels remained essentially unchanged (Ruokojärvi *et al.*, 2000). The increases in levels of polychlorinated biphenyls (PCBs, 0.021 to 0.031 mg/m³), polychlorinated benzenes (0.002 to 0.010 mg/m³) and I-TEQs [or PCDD/F] (3.5 to 5.4 ng/m³) as products of combustion were not significant [possibly due to the small sample size]. In another study, proportionately higher levels of ethyl benzene (IARC Group 2B) were found at an electronics factory fire when compared to levels at residential and mixed occupancy fires (Austin *et al.*, 2001b).

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

1.2.3 *Carcinogens found in smoke at fires*

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

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

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

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

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

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

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

(c) *Thyroid cancer*

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

(d) *Melanoma*

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

2.3 Descriptive studies

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

2.3.1 *Cohort and linkage studies of firefighters*

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

2.4 Case reports

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

2.5 Meta-analyses

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

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

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

3. Studies of Cancer in Experimental Animals

No data were available to the Working Group.

4. Mechanistic and Other Relevant Data

4.1 Absorption, distribution, metabolism and excretion

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

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

4.1.1 Particles

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

Table 4.1 (contd)

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