Douglas Consulting Australia Pty Ltd ACN 88 003 582 385
Office 1, 483 Adelaide St BRISBANE PO Box 10398, BRISBANE ADELAIDE STREET, QLD 4000

FIREFIGHTER CHEMICAL REVIEW – ARP1701

A report prepared for the Commonwealth of Australia

(as represented by the Department of Veterans' Affairs,
the Repatriation Commission and the Military Rehabilitation and
Compensation Commission)

Dr David DOUGLAS MBBS MSc DIH FAFOEM
Consultant Occupational Physician

9 April 2018

Contents

EXECUTIVE SUMMARY

This report is based on a medical literature search done in relation to the health effects of chemicals identified as contaminants at the former fire training area at Royal Australian Air Force (RAAF) Base, Williams, at Point Cook, Australia.

Australian Defence Force (ADF) firefighters carried out fire training and disposal at Point Cook in the 1970s. This required the lighting of large fires which would then be extinguished. The flammable materials used in the fires included a wide range of solid and liquid waste materials, leading to complex mixtures of pyrolysis products to which the ADF firefighters were potentially exposed during their duties. After some years, the setting of such fires was discontinued; but there is an ongoing concern by ex-serving ADF firefighters regarding the impact of the pyrolysis products, and the materials used to build the fires, on their health.

In 2016, the Australian Department of Defence provided a list of chemical contaminants identified by HLA ENSR (HLA – Envirosciences Pty Limited) following an environmental risk assessment of the relevant area. The list contained some 200 contaminants which include chemicals, chemical compounds, and metals.

The report includes toxicological profiles of each of the listed contaminants aimed at identifying probable adverse health outcomes, followed by cross-referencing the adverse health outcomes to the factors under the Repatriation Medical Authority (RMA) Statements of Principles. Additionally, the report includes a literature review which compares the current state of knowledge of the long-term health outcomes experienced by firefighters with the toxic effects of the contaminants identified at Point Cook.

The toxicological profile of each of the listed contaminants has been prepared utilizing many reference sources including the Agency for Toxic Substances and Disease Registry (ATSDR) and PubChem in the United States, and the International Agency for Research on Cancer (IARC) in Europe.

All contaminants present in smoke from the pyrolysis of multiple materials have the potential to cause ill-health by inhalation and skin contact. But whereas acute health effects are usually immediately obvious at the time of exposure, long-term health effects may occur years later, and this is the issue of current concern. Accordingly, the contaminants were separated into two categories:

lower concern contaminants – not considered to be the cause of long-term health problems (Appendix B), and

higher concern contaminants – considered to have the potential to cause long-term health problems including cancer (Appendix C).

The adverse health effects of most concern have been summarised and grouped into categories for easy reference: (a) volatile organic compounds (VOC), (b) dioxins and furans, (c) polycyclic aromatic hydrocarbons (PAH), (d) total petroleum hydrocarbons (TPH), (e) trihalomethanes (TPH), and (f) metals (See Table 1, Section 4).

*VOC*s: acute and chronic effects on the central nervous system; peripheral neuropathy; upper respiratory tract and eye irritation; damage to the heart, lungs, liver, kidneys, and thyroid; damage to the immune system; cancer of the bladder and liver; and haemato-lymphatic cancers including acute myeloid leukaemia, aplastic anaemia, multiple myeloma, and non-Hodgkin's lymphoma.

Dioxins and furans: skin damage including chloracne; damage to the liver; endocrine disruption and birth defects; increased cancer incidence overall; cancers of the liver and bladder; multiple myeloma and non-Hodgkin's lymphoma.

Polycyclic aromatic hydrocarbons (PAHs): skin damage and dermatitis; photosensitisation; bronchitis and pulmonary oedema; cancer of the skin and lung; and leukaemia.

Total petroleum hydrocarbons (TPH): acute and chronic effects on the central nervous system; peripheral neuropathy; and other effects as noted above for VOCs.

Trihalomethanes (THM): acute and chronic effects on the central nervous system; liver and kidney damage, and possibly carcinogenic to liver and kidneys.

Metals: irritant and allergic dermatitis; damage to the heart, lungs, liver, and kidneys; nasal sinus damage and asthma; chronic effects on the central nervous system including tremors; and cancers of the skin, nasal sinuses and lungs.

In relation to mixtures, the limited data indicate that probable additive effects exist between substances within mixtures, but the extent of such effects is not currently known or quantifiable. Therefore, the toxic effects of the most toxic compound in any mixture are assumed to be the minimum effects of the mixture.

All the Statements of Principles (SoPs) published by the Repatriation Medical Authority (RMA) were reviewed. Based on the assumption that the firefighters, whose chemical exposures are the subject of this review, experienced their exposures during non-operational service in peace time, the **balance of probabilities** standard has been applied.

The occupation of firefighter is listed as a factor in only one SOP – mesothelioma. Smoke from fires is listed as a factor for bronchiectasis and chronic obstructive pulmonary disease, and smoke in an enclosed space is a factor for fibrosing interstitial lung disease and malignant neoplasm of the lung. Inhalation of irritants is a factor for asthma, and exposure to irritants is a factor for irritant dermatitis.

Additionally, many of the contaminants listed in Appendix C are factors in a wide range of SoPs, but not in exposures experienced by firefighters. These contaminants include arsenic, benzene, carbon disulfide, cobalt, dioxins and furans, manganese, PAHs, vinyl chloride, and VOCs (Table 2 Section 5).

A literature review was undertaken to address the gaps in relation to the reported health effects arising from the contaminants that have not been listed specifically as factors in the SoPs (see References below).

Published reports on exposures added support to the assumption that the contaminants found at the site of ADF firefighter training at Point Cook could have been present in the smoke to which the firefighters were exposed.

A study of respiratory dysfunction six years after smoke exposure supported the SoPs for bronchiectasis, chronic obstructive pulmonary disease, fibrosing interstitial lung disease, and asthma which all list smoke, in various exposures, as a causal factor.

There were no publications providing probable causal links between firefighters and other non-cancer long-term health outcomes.

There were several well conducted large-scale studies that supported the view that, on the balance of probabilities, exposures experienced by firefighters contributed materially to the subsequent development of cancers in general, and to some specific malignancies: cancers of the bladder, brain, colon, kidney, lung, prostate, and testes; and leukaemia, multiple myeloma, and non-Hodgkin's lymphoma.

Overall, this Firefighter Chemical Review, combined with a review of the SoPs and a literature review, concludes that **"firefighting"**, as in the current SoP for mesothelioma, should be considered as a factor in other SoPs as indicated in Table 3, Section 7.

1 INTRODUCTION

1.1 Background

Douglas Consulting Australia was contracted by the Department of Veterans' Affairs (DVA) to supply medical literature research services in relation to the health effects of chemicals identified as contaminants at the former fire training area at Royal Australian Air Force (RAAF) Base, Williams, at Point Cook, Australia.

Australian Defence Force (ADF) firefighters carried out fire training and disposal at Point Cook in the 1970s. This required the lighting of large fires which would then be extinguished. The flammable materials used in the fires included a wide range of solid and liquid waste materials, leading to complex mixtures of pyrolysis products to which the ADF firefighters were potentially exposed during their duties. After some years, the setting of such fires was discontinued; but there is an ongoing concern by ex-serving ADF firefighters regarding the impact of the pyrolysis products, and the materials used to build the fires, on their health.

In 2016, the Australian Department of Defence provided a list of chemical contaminants identified by HLA ENSR (HLA – Envirosciences Pty Limited) following an environmental risk assessment of the relevant area[1]. The list contains some 200 contaminants which include chemicals, chemical compounds, and metals. The contaminants had been identified by analysis of five media: soil, groundwater, sediment, soil vapour, and DNAPL (dense non-aqueous phase liquid) – Appendix A.

1.2 Scope

DVA commissioned Douglas Consulting Australia to: (i) create a toxicological profile of the listed contaminants of greatest concern; (ii) to cross-reference these contaminants to the Factors under the Repatriation Medical Authority (RMA) Statements of Principles (SoPs); and (iii) to carry out a literature review. This work was commissioned to help identify the extent of coverage already established by the RMA, and the need for any additional coverage associated with these contaminants.

1.3 Pyrolysis toxicology

Pyrolysis is defined as the decomposition of materials due to high temperatures, and the process invariably generates toxic gases, vapours and particulates. The toxicity of the products of pyrolysis varies considerably and depends on the chemical nature and concentrations of the substances when released from the materials involved in a fire. These

[1] Human Health Risk Assessment Point Cook Foreshore, Former Fire Training Area RAAF Williams, Point Cook, Victoria 02 July 2007. Report by: HLA ENSR.
Prepared for: Property Disposal Task Force Department of Defence BP-2-A017 Canberra ACT 2600

toxic substances may produce acute effects, latent effects, and with repeated exposure, as for firefighters, cumulative and/or long-term effects.

Smoke is a complex mixture of airborne solid and liquid particulates, vapours, and gases which are produced when the materials in the fire undergo vapourisation or thermal decomposition. Therefore, the atmosphere in any fire is extremely complex. Because of the constantly changing conditions during the progress of a fire, the chemical composition, both nature and concentration of materials, varies markedly at different stages of the fire.[i]

1.4 Limitations and assumptions.

The data presented in Appendix A represent the list of contaminants that have been analysed within the former Fire Training Area (FTA) at Point Cook. The concentrations reported are stated to represent the highest recorded concentration identified for each contaminant in the five media of soil, groundwater, sediment, soil vapour and DNAPL at the time of sampling. The contaminants were selected for laboratory analysis based upon the site history and relevant background information. They include relevant environmental and broad screening contaminant categories, and specific compounds associated with the protocols of the analytical laboratory.

The concentrations of contaminants found in the detailed media analyses cannot be applied directly to the concentration of contaminants in the smoke to which the ADF firefighters were exposed. But it can be assumed that the contaminants found in the media analyses could have been present at various times, and in variable concentrations, in the smoke generated by the training fires at Point Cook.

2. METHODOLOGY

2.1 Reference sources

During the past thirty years, there has been such an exponential growth in information technology that freely available databases have been established by government agencies in North America and Europe. These include the Agency for Toxic Substances and Disease Registry (ATSDR) and PubChem in the United States, and the Health and Safety Executive in the United Kingdom. Other valuable reference sources include those of the World Health Organisation (WHO) and the International Labor Organisation (ILO).

The databases that have been most valuable in sourcing data on the contaminants listed in Appendix A are summarised in the following paragraphs. The summaries have been based on the material supplied on-line by each database.

2.2 The US Agency for Toxic Substances and Disease Registry (ATSDR)

ATSDR is a Federal public health agency of the U.S. Department of Health and Human Services (DHHS). ATSDR has been established to help protect communities from harmful health effects related to exposure to natural and man-made hazardous substances. It does this by responding to environmental health emergencies; investigating emerging environmental health threats; conducting research on the health impacts of hazardous waste sites; and building capabilities for providing actionable guidance to state and local health partners.

By Congressional mandate, the Agency for Toxic Substances and Disease Registry (ATSDR) produces "toxicological profiles" for hazardous substances found at National Priorities List (NPL) sites. These hazardous substances are ranked based on frequency of occurrence at NPL sites, toxicity, and potential for human exposure. Toxicological profiles are developed from a priority list of 275 substances. ATSDR also prepares toxicological profiles for the Department of Defense (DOD) and the Department of Energy (DOE) on substances related to Federal sites. Toxicological profiles are developed in two stages: the toxicological profiles are first produced as drafts. ATSDR announces in the Federal Register the release of these draft profiles for a 90-day public comment period. After the 90-day comment period, ATSDR considers incorporating all comments into the documents and then finalises the profiles.

The ATSDR toxicological profiles covered most of the substances in Appendix A and have been the major source of data for this review.

2.3 PubChem

PubChem is an open chemistry database at the US National Institutes of Health (NIH). *"Open"* means that contributors can submit scientific data to PubChem and that others may access it. PubChem collects information on chemical structures, identifiers, chemical and physical properties, biological activities, patents, health, safety, toxicity data, and many

others. Since its launch in 2004, PubChem has become a key chemical information resource for scientists, students, and the public. Each month the PubChem website and programmatic services provide data to several million users worldwide. PubChem records are contributed by hundreds of data sources, including: research results from universities, pharmaceutical companies, and others; government agencies; chemical vendors; journal publishers; and the efforts of curation of chemical biology.

The PubChem database has proved to be an important additional resource to that of ATSDR in this review.

2.4 The International Agency for Research on Cancer (IARC)

IARC is the specialised cancer agency of the World Health Organization. The objective of the IARC is to promote international collaboration in cancer research. The Agency is inter-disciplinary, bringing together skills in epidemiology, laboratory sciences and biostatistics to identify the causes of cancer so that preventive measures may be adopted, and the burden of disease and associated suffering reduced. A significant feature of the IARC is its expertise in coordinating research across countries and organizations; its independent role as an international organization facilitates this activity. Emphasis is placed on elucidating the role of environmental and lifestyle risk factors and studying their interplay with genetic background in population-based studies and appropriate experimental models. This emphasis reflects the understanding that most cancers are, directly or indirectly, linked to environmental factors and thus are preventable. The IARC Monographs Programme is a core element of the Agency's portfolio of activities, with international expert working groups evaluating the evidence of the carcinogenicity of specific exposures. Since commencing its work in 1970, the IARC has published 120 Monographs with the following evaluations:

Group 1	Carcinogenic to humans	120 agents
Group 2A	Probably carcinogenic to humans	81 agents
Group 2B	Possibly Carcinogenic to humans	299 agents
Group 3	Not classifiable as to its carcinogenicity to humans	502 agents
Group 4	Probably not carcinogenic to humans	1 agent

The IARC Monographs have been an essential source of data on the carcinogenicity of all substances in this review.

2.5 The US Occupational Safety and Health Administration (OSHA)

OSHA maintains an Occupational Chemical Database as a convenient reference for the occupational safety and health community. It compiles information from several government agencies and organisations, and information available in the database includes: physical properties; exposure guidelines; National Institute for Occupational Safety and Health

(NIOSH) pocket guides; and emergency response information. The database originally was developed by OSHA in cooperation with the US Environmental Protection Agency (EPA).

The OSHA Occupational Chemical Database lists 454 chemicals as carcinogens, and this list has been sourced in the compilation of this review.

2.6 The Australian National Industrial Chemicals Notification and Assessment Scheme (NICNAS)

NICNAS helps protect the Australian people and the environment by assessing the risks of industrial chemicals and providing information to promote their safe use. The focus is on the industrial use of chemicals and covers a broad range of chemicals used in inks, plastics, adhesives, paints, glues, solvents, cosmetics, soaps and many other products.

Data relevant to this review exist on assessments of Priority Existing Chemicals (PEC) for only the following substances listed in Appendix A: benzene, lead, dichlorobenzenes, tetrachloroethylene, and trichloroethylene.

2.7 The United States Environmental Protection Agency (US EPA)

US EPA is an agency of the Federal Government of the United States which was created in 1970 for protecting human health and the environment by writing and enforcing regulations based on laws passed by Congress. Its publications cover a wide range of environmental topics including chemicals and toxins. Priority substances are mercury, lead, hazardous/toxic pollutants, PCBs, and pesticides. Hazardous/toxic pollutants, also known as air pollutants or air toxics, are those pollutants that are known or suspected to cause cancer, or other serious health effects such as reproductive effects or birth defects, or adverse environmental effects. The list of 187 air toxics includes PAHs, benzene and benzene derivatives, bromoform, carbon disulphide, carbon tetrachloride, furans, ethylene oxide, trichloroethane, MIBK, ethylene oxide, PCBs, toluene, vinyl chloride, xylenes, and carcinogenic metals. The US EPA refers to the US Agency for Toxic Substances and Disease Registry (ATSDR) for detailed toxicity data.

The US EPA databases have also been utilized in this review, but most have referred to ATSDR as the authoritative source of toxicity data.

2.8 The International Programme on Chemical Safety (IPCS)

IPCS is a programme of the World Health Organisation (WHO) that works to establish the scientific basis for the sound management of chemicals and to strengthen national capabilities and capacities for chemical safety. Its publication in 2016 *Public Health Impact of Chemicals – Knowns and Unknowns* stated that air pollutants from ambient air and household sources are a mixture of many components, including carbon monoxide, sulphur dioxide, nitrogen oxide, and particulate matter. The particulate matter contains substances such as acids, organic chemicals, metals, soil, and dust particles. The largest sources of air pollution are combustion and other processes from energy generation, industry, and

transport. The IPCS has estimated that an overall reduction or elimination of exposure to chemicals from air pollution would have a significant impact in reducing morbidity and mortality from a wide range of conditions including: ischaemic heart disease, stroke, cancers, mental and neurological disorders, chronic obstructive pulmonary disease, bronchitis, and asthma.

The IPCS publications support the data from other reference sources that indicate both acute and cumulative effects due to toxic pyrolysis products.

2.9 The UK Health and Safety Executive (HSE)

HSE is an Executive Non-Departmental Public Body of the UK Government, and its core purpose is to reduce work-related injury and ill-health. The HSE is an international centre of expertise for occupational safety and health, and the effective management and control of risks. Control of Substances Hazardous to Health (COSHH) is the law that requires employers to control substances that are hazardous to health (except asbestos and lead which have specific regulations).

The HSE publications focus on implementation, but do not provide the basic toxicological data needed for this review.

3. EVALUATION OF CONTAMINANTS

3.1 List of contaminants

All the contaminants listed in Appendix A have been reviewed by utilizing the reference sources cited above. Based on the review, the contaminants have been separated into two categories:

Lower concern contaminants – not considered to be the cause of long-term health problems – listed in Appendix B, and

Higher concern contaminants – considered to have the potential to cause long-term health problems including cancer – listed in Appendix C.

All contaminants present in smoke from the pyrolysis of multiple materials have the potential to cause ill-health. But whereas acute health effects are usually immediately obvious at the time of exposure, long-term health effects may occur years later, and this is the issue of current concern.

3.2 Appendix B – Lower concern contaminants – not considered to be the cause of long-term health problems

Appendix B lists all the contaminants identified from Appendix A as those that are not considered to be the cause of long-term health problems. But all those listed in Appendix B are not innocuous, for many are volatile organic compounds **(VOCs)** with acute irritancy properties. That is, they can cause acute irritation to the upper respiratory tract (URT), lungs, eyes, skin, and central nervous system (CNS).

VOCs are a class of chemicals that are volatile, evaporate easily, and are organic compounds because they contain carbon atoms. The VOCs in Appendix B are:

1,1,1-trichloroethane
1,1,2-trichloroethane
1,2,4-trimethylbenzene
1,2-dichloroethene
1,3-dichloropropane
acetone
bromobenzene
bromomethane
chlorobenzene
ethene
ethyl acetate
heptane
hexaethylbenzene
isopropylbenzene
n-butylbenzene
n-propylbenzene
tetraethylbenzene
triethylbenzene

3.3 Appendix C - Higher concern contaminants - considered to have the potential to cause long-term health problems including cancer.

Appendix C lists all the contaminants identified from Appendix A as those considered to have the potential to cause long-term health problems including cancer. A summary of the toxic properties of each contaminant has been included in Appendix C. The contaminants have been grouped into categories: volatile organic compounds (VOC), dioxins and furans, polycyclic aromatic hydrocarbons (PAH), total petroleum hydrocarbons (TPH), trihalomethanes (TPH), and metals, as follows.

3.4 Volatile organic compounds (VOCs).

The **VOCs** of concern in Appendix C are:

1,1,2,2-tetrachloroethane
1,1-dichloroethane
1,1-dichloroethene
1,2,4-trichlorobenzene
1,2-dichlorobenzene
1,2-dichloroethane
1,2-dichloropropane
1,3,5-trimethylbenzene
1,3-acute4-methyl-2-pentanone
benzene
bromodichloromethane
bromoform
bromomethane
carbon disulfide
carbon tetrachloride
chloroethane
chloroform
chloromethane
dichloromethane
ethanol
ethylbenzene
hexachloroethane
hexane
styrene
tetrachloroethylene
toluene
trichloroethylene
trihalomethanes
vinyl chloride
xylenes

This list of VOCs includes **benzene, toluene, ethyl benzene, and xylene** which are referred to collectively as the **BTEX** mixture of volatile organic compounds.

Adverse health effects reported from VOCs include:

acute and chronic effects on the central nervous system
peripheral neuropathy
upper respiratory tract (URT) irritation
eye irritation
damage to the heart, lungs, liver, kidneys, and thyroid
damage to the immune system
cancer of the bladder and liver
haemato-lymphatic cancers including:
- acute myeloid leukaemia
- aplastic anaemia
- multiple myeloma
- non-Hodgkin's lymphoma

3.5 Dioxins and furans

Dioxins and furans of concern in Appendix C are:

1234678-HpCDD
1234678-HpCDF
123478-HxCDF
2378-TCDD
hepta-dioxins
hepta-furans
OCDD
OCDF
octa-dioxin
octa-furan
PCBs
tetra-dioxin
tetra-furans

Adverse health effects reported from dioxins and furans include:

skin damage including chloracne
damage to the liver
endocrine disruption
birth defects
increased cancer incidence overall
cancer of the liver
cancer of the bladder
multiple myeloma
non-Hodgkin's lymphoma

3.6 Polycyclic aromatic hydrocarbons (PAHs).

PAHs of concern in Appendix C are:

acenaphthalene
anthracene
benz(a)anthracene
benzo(a)pyrene
benzo(b)&(k)fluoranthene
benzo(b)fluoranthene
benzo(g,h,i)perylene
benzo(k)fluoranthene
chrysene
dibenz(a,h)anthracene
fluoranthene
fluorene
indeno(1,2,3-c,d)pyrene
phenanthrene
pyrene

Adverse health effects reported from PAHs include:

dermatitis
photosensitisation
bronchitis and pulmonary oedema
cancer of the skin
cancer of the lung
leukaemia

3.7 Total petroleum hydrocarbons (TPH).

TPH of concern, as listed in Appendix C, consist of a mixture of aliphatic and aromatic hydrocarbons ranging from C6 to C40.

Adverse health effects reported from TPH include:

acute and chronic effects on the central nervous system
peripheral neuropathy
other effects as noted above for VOCs

3.8 Trihalomethanes (THMs).

Trihalomethanes (THMs) of concern, as listed in Appendix C, are a group of organic chemicals that include:

trichloromethane (chloroform)
bromodichloromethane (BDCM)
dibromochloromethane (DBCM)
tribromomethane (bromoform)

Adverse health effects reported from THMs include:

acute and chronic effects on the central nervous system
liver and kidney damage
possibly carcinogenic to liver and kidneys

3.9 Metals

Metals of concern, as listed in Appendix C, are:

antimony
arsenic
barium
beryllium
cadmium
chromium (III) and (VI)
cobalt
lead
magnesium
manganese
mercury
nickel
vanadium

Adverse health effects reported from metals include:

irritant and allergic dermatitis
damage to the heart, lungs, liver, and kidneys
nasal sinus damage;
asthma
chronic effects on the central nervous system including tremors
cancer of the skin
cancer of the nasal sinuses
cancer of the lungs

4 HEALTH EFFECTS OF MIXTURES

4.1 Overview

The ATSDR has stated that the health assessment of hazardous substances is complicated by the reality that most toxicological testing has been performed on single chemicals, but human exposures are rarely limited to single chemicals. Exposures resulting from smoke inhalation, and from hazardous waste sites generally, involve more than one hazardous substance. At issue is whether a mixture of contaminants may be more hazardous due to additivity, interactions, or both. The available data of relevance for this review relate to mixtures of dioxins and furans, PAHs, TPH, THM, BTEX, and some metals.

4.2 Dioxins and furans

ATSDR has stated that dioxins, furans, and polychlorinated biphenyls (PCBs) are a class of similar chlorinated aromatic organic compounds. Dioxins have two phenyl rings connected by two oxygen atoms. Furans have one or two phenyl rings connected to a furan ring. PCBs have two phenyl rings attached at one point. One or more chlorine atoms can attach to any available carbon atom, allowing for 100 to 200 forms of each. Dioxins and dioxin-like furans have no known commercial or natural use. They are produced primarily during the incineration or burning of waste; the bleaching processes used in pulp and paper mills; and the chemical syntheses of trichlorophenoxyacetic acid, hexachlorophene, vinyl chloride, trichlorophenol, and pentachlorophenol. PCBs were once synthesized for use as heat-exchanger, transformer, and hydraulic fluids, and used as additives to paints, oils, window caulking, and floor tiles. Production of PCBs peaked in the early 1970s and was banned after 1979.

Dioxins and furans can cause many health effects. The most well-known member of the dioxins/furans family is 2,3,7,8 TCDD. The U.S. Environmental Protection Agency (EPA) has stated that it is likely to be a cancer-causing substance to humans. In addition, people exposed to dioxins and furans have experienced changes in hormone levels. High doses of dioxin have caused the skin disease chloracne. Animal studies show that animals exposed to dioxins and furans experienced changes in their hormone systems, changes in the development of the fetus, decreased ability to reproduce and suppressed immune system.

PubChem has stated that 2,3,7,8-tetrachlorodibenzofuran (TCDF) has a magnitude of toxicity like 2,3,7,8-tetrachloro-p-dioxin. TCDF has been shown to affect biochemical activity, suppress immune function, cause fetal abnormalities, and induce tumors in non-human test organisms. While not used commercially, TCDF is found as an impurity in polychlorinated biphenyl products, and 2,4,6-trichlorophenol. The major route of exposure to the general population results from municipal waste incineration, industrial boilers burning hazardous waste, and exhausts from leaded gasoline engines. Occupational exposure occurs through inhalation and dermal contact to fire fighters and cleanup workers associated with polychlorinated biphenyl transformer fires.

IARC has evaluated dioxins, furans, and PCBs and provided the following classifications: There is sufficient evidence in humans for the carcinogenicity of 2,3,7,8-tetrachlorodibenzo-para-dioxin. The strongest evidence in humans for the carcinogenicity of 2,3,7,8-tetrachlorodibenzo-para-dioxin is for all cancers combined. Also, a positive association has been observed between exposure to 2,3,7,8-tetrachlorodibenzo-para-dioxin and soft-tissue sarcoma, non-Hodgkin lymphoma and cancer of the lung.

There is sufficient evidence in experimental animals for the carcinogenicity of 2,3,7,8-tetrachlorodibenzo-para-dioxin.

There is sufficient evidence in experimental animals for the carcinogenicity of 2,3,4,7,8-pentachlorodibenzofuran.

There is sufficient evidence in experimental animals for the carcinogenicity of 3,3,4,4,5-pentachlorobiphenyl.

There is strong evidence to support a receptor mediated mechanism that operates in humans for carcinogenesis associated with 2,3,7,8-tetrachlorodibenzo-para-dioxin, where the primary mechanism is the promotion of tumour development through modification of cell replication and apoptosis, with a secondary mechanism related to increases of oxidative stress causing DNA damage. The conservation of the aryl hydrocarbon receptor and the related signalling pathways and responses across species, including humans, add additional strength to the notion that this mechanism is active in humans.

2,3,7,8-Tetrachlorodibenzo-para-dioxin is carcinogenic to humans (Group 1).

2,3,4,7,8-Pentachlorodibenzofuran is carcinogenic to humans (Group 1).

3,3,4,4,5-Pentachlorobiphenyl is carcinogenic to humans (Group 1).

In making the second and third overall evaluations, the Working Group considered the following mechanistic arguments: There is strong evidence to support a receptor-mediated mechanism for 2,3,4,7,8-pentachlorodibenzofuran- and 3,3,4,4,5-pentachlorobiphenyl-associated carcinogenesis in humans based upon evidence of carcinogenicity in experimental animals and upon extensive evidence showing activity identical to 2,3,7,8-tetrachlorodibenzo-paradioxin (TCDD) for every step of the mechanism described for TCDD-associated carcinogenesis in humans including receptor binding, gene expression, protein-activity changes, cellular replication, oxidative stress, promotion in initiation-promotion studies and complete carcinogenesis in laboratory animals.

4.3 Polycyclic aromatic hydrocarbons (PAHs)

ATSDR has stated that polycyclic aromatic hydrocarbons (PAHs) are a class of more than 100 chemicals generally produced during the incomplete burning of organic materials, including coal, oil, gas, wood, garbage, and tobacco. PAHs are composed of up to six benzene rings fused together such that any two adjacent benzene rings share two carbon bonds. Examples include phenanthrenes, naphthalene, and pyrene. Important PAH sources include motor vehicle exhaust, residential and industrial heating sources, coal, crude oil and natural gas processing, waste incineration, and tobacco smoke. The emitted PAHs can form or bind to particles in the air, and particle size depends in part on the source of the PAHs.

Human exposure usually occurs to PAH mixtures rather than to individual chemicals, and PAH mixture composition varies with the combustion source and temperature. PAH exposure can occur in workplaces where petroleum products are burned or coked, such as coke production, coal gasification and gas refining, iron or steel production, roofing tar and asphalt application, waste incineration, and aluminium smelting. Coal tar ointments containing PAHs are used to treat several inflammatory skin conditions.

PAHs are lipid soluble and can be absorbed through the skin, respiratory tract, and gastrointestinal tract. PAH metabolism is complex and occurs primarily in the liver, and to a lesser extent, in other tissues. The metabolic pathways and enzyme-inducing effects of specific PAHs, such as benz[a]pyrene, have been actively studied to elucidate cancer potential and causal mechanisms. Although immunologic, kidney and brain toxicity have been seen in animals after high doses were administered, it is unclear if similar effects may occur in humans. Lung, bladder, and skin cancers have been reported in occupational settings following high PAH exposures. Exposure to fine particulates has been associated with foetal growth retardation, respiratory disorders, and cardiovascular disease.

IARC has evaluated PAHs and provided the following classifications: there is sufficient evidence for the carcinogenicity of benzo[a]pyrene in experimental animals. No epidemiological data on benzo[a]pyrene alone were available. Benzo[a]pyrene is carcinogenic to humans (Group 1). In making the overall evaluation, the Working Group took the following into consideration: The strong and extensive experimental evidence for the carcinogenicity of benzo[a] pyrene in many animal species, supported by the consistent and coherent mechanistic evidence from experimental and human studies provide biological plausibility to support the overall classification of benzo[a]pyrene as a human carcinogen (Group 1).

Additionally, IARC classifies many other PAHs as probable (Group 2A) or possible (Group 2B) human carcinogens. IARC and NTP have classified specific PAH-containing chemical mixtures (e.g., soot, coke oven emissions, coal tars and coal tar pitches) as human carcinogens.

4.4 Total petroleum hydrocarbons (TPH)

ATSDR has stated that total petroleum hydrocarbons (TPH) is a term used to describe a large family of several hundred chemical compounds that originally come from crude oil. Crude oil is used to make petroleum products, which can contaminate the environment. Because there are so many different chemicals in crude oil and in other petroleum products, it is not practical to measure each one separately. However, it is useful to measure the total amount of TPH at a site. TPH is a mixture of chemicals, but they are all made mainly from hydrogen and carbon, called hydrocarbons. TPH are divided into into groups of petroleum hydrocarbons that act alike in soil or water. These groups are called petroleum hydrocarbon fractions. Each fraction contains many individual chemicals. Some chemicals that may be found in TPH are hexane, jet fuels, mineral oils, benzene, toluene, xylenes, naphthalene, and fluorene, as well as other petroleum products and gasoline components. Samples of TPH usually contain only some, or a mixture, of these chemicals.

Some of the TPH compounds can affect the central nervous system resulting in headaches and dizziness at high levels in the air. Another compound, hexane, can cause peripheral neuropathy with numbness in the feet and legs. Other TPH compounds can cause effects on the blood, immune system, lungs, skin, and eyes. Animal studies have shown effects on the lungs, central nervous system, liver, and kidney from exposure to TPH compounds. Some TPH compounds have also been shown to affect reproduction and the developing foetus in animals.

IARC has evaluated TPH and provided the following classifications: one TPH compound (benzene) is carcinogenic to humans. IARC has determined that other TPH compounds (benzo[a]pyrene and gasoline) are probably and possibly carcinogenic to humans. Most of the other TPH compounds are considered not to be classifiable by IARC.

4.5 Trihalomethanes (THMs)

PubChem has stated that trihalomethanes (THMs) are a group of organic chemicals that often occur in drinking water because of chlorine treatment for disinfectant purposes and, therefore, are also known as "disinfection byproducts" or DBPs. THMs are formed when chlorine reacts with naturally occurring organic material found in water such as decaying vegetation. Typically, the following four THMs are found because of chlorination: trichloromethane (chloroform), bromodichloromethane (BDCM), dibromochloromethane (DBCM), tribromomethane (bromoform). Untreated or raw water rarely contains THMs in significant concentrations. Since chloroform is the THM found in highest concentrations and about which the most is known, the bulk of the information contained in this summary pertains to chloroform.

ATSDR has stated that chloroform is a colourless liquid with a pleasant, non-irritating odour and a slightly sweet taste. It will burn only when it reaches very high temperatures. In the past, chloroform was used as an inhaled anaesthetic during surgery. Today, chloroform is used to make other chemicals and can also be formed in small amounts when chlorine is

added to water. Other names for chloroform are trichloromethane and methyl trichloride. Breathing about 900 parts of chloroform per million parts of air (900 ppm) for a short time can cause dizziness, fatigue, and headache. Breathing air, eating food, or drinking water containing high levels of chloroform for long periods of time may damage the liver and kidneys. Large amounts of chloroform can cause sores when chloroform touches the skin.

The US Department of Health and Human Services (DHHS) has determined that chloroform may reasonably be anticipated to be a carcinogen. Rats and mice that ate food or drank water with chloroform developed cancer of the liver and kidneys.

IARC has evaluated chloroform and provided the following classification: there is inadequate evidence in humans for the carcinogenicity of chloroform. There is sufficient evidence in experimental animals for the carcinogenicity of chloroform. Chloroform is possibly carcinogenic to humans (Group 2B).

4.6 Benzene, toluene, ethyl benzene, and xylene (BTEX)

ATSDR has concluded that no studies were available that directly characterized health hazards and dose-response relationships for exposures to whole mixtures of benzene, toluene, ethylbenzene, and xylenes (BTEX). All four components can produce neurological impairment, and benzene can additionally cause hematological effects which may ultimately lead to aplastic anemia and development of acute myelogenous leukemia. Concern for the carcinogenicity of BTEX has been raised by evidence that ethylbenzene is carcinogenic. No studies were located that directly examined joint toxic actions of mixtures of benzene, toluene, ethylbenzene, and xylenes on the nervous system, but additive joint action is plausible. Results of model simulations and experimental exposures with BTEX, and ternary and quinary mixtures of its components, strongly suggest that joint neurotoxic action is expected to be additive at BTEX concentrations below approximately 20 ppm of each component. Neurotoxicity interaction studies of binary component mixtures support the plausibility of additive joint action at environmental levels of BTEX exposure. It is unclear whether the models are adequate for characterizing interactions from inhalation of BTEX mixtures above approximately 200 ppm of each component, or if the results are applicable to oral exposures. The possible hematotoxic and leukemogenic hazards of BTEX exposures should be evaluated on a benzene-specific basis because the other mixture components do not induce these effects. Considering the causal relationship between the non-cancer hematological effects of benzene and development of leukemia, as well as lack of a cancer risk value for ethylbenzene, ATSDR has recommended that the inhalation cancer unit risk value for benzene be used to assess the benzene/ethylbenzene-related hematological/carcinogenic hazards from exposures to BTEX. Exposure to relatively high concentrations of BTEX (above approximately 20 ppm of each chemical) is expected to increase the potential for neurotoxicity and decrease the potential for hematotoxicity/carcinogenicity due to competitive metabolic interactions among the mixture components.

IARC has evaluated BTEX compounds and provided the following classifications: benzene is carcinogenic to humans (Group 1), ethyl benzene is possibly carcinogenic to humans (Group 2B), and toluene and xylenes are not-classifiable as to their carcinogenicity (group 3).

4.7 Metals

ATSDR has stated that lead, arsenic, cadmium, and chromium, which have all been included in the review of higher priority contaminants, have been assessed because they constitute a frequently occurring quaternary mixture at hazardous waste sites. But, as the following summary indicates, there are difficulties in providing reliable data on the health effects of mixtures, even when there are extensive data on each metal.

The ATSDR toxicity profile focuses on inorganic forms of these metals, consistent with the sampling data, and on chromium(VI), the species of concern for chromium. No relevant health effects data or physiologically based pharmacokinetic models were located for the quaternary mixture, so assessment of health hazards depended on an evaluation of the toxicity data for the individual metals and on the joint toxic action and mechanistic data for various combinations of these metals found from animal studies.

Most of the information regarding joint toxic action for the metals in this mixture was for binary combinations of the metals. Experimental data were voluminous for the lead-cadmium mixture, and extensive for the lead-arsenic mixture, but all were from oral exposure studies and not from inhalation exposures. The data for the other binary mixtures were less extensive and less relevant in terms of sequence, duration, and route of exposure, as well as endpoints of toxicity.

For these reasons, the weight-of-evidence (WOE) approach for the assessment of interactions was used to prepare binary weight-of-evidence determinations (BINWOE) for the binary mixtures. The BINWOE determinations provided conclusions regarding the expected direction of interaction and considered the potential endpoint-specificity of joint toxic action.

Each of the four metals affects a wide range of target organs and endpoints, but they do not share the same critical effects.

Following oral exposures, the critical effect for lead is neurological, for arsenic it is dermal, for cadmium it is renal, and for chromium(VI) it is uncertain; though each is carcinogenic by various routes of administration. Sensitive effects in common across two or more of these metals include neurological, renal, cardiovascular, and hematological effects. Although less sensitive, testicular effects also are an endpoint of concern because a synergistic interaction has been noted for lead and cadmium, and because chromium(VI) also affects the testes.

The binary mixtures with the most extensive interaction databases were the lead-arsenic mixture and the lead-cadmium mixture; but the predicted direction of interaction for the effects of these mixtures was not consistent across endpoints. This observation was most striking for the effects of cadmium on the toxicity of lead. The predicted direction was greater than additive for the neurological effects and testicular effects, less than additive for renal and hematological effects, and additive for cardiovascular effects. Confidence in the BINWOE determinations ranged from relatively high for renal and testicular, to low for neurological. The observation of inconsistency in predicted direction of interaction underscored the uncertainty in extrapolating interactions from one endpoint to another. The recommendations for assessing the potential hazard to the public (and to firefighters) of the joint toxic action of lead, arsenic, cadmium, and chromium(VI) was to estimate endpoint-specific hazard indexes for neurological, renal, cardiovascular, hematological, and testicular toxicity of the mixture.

Neurological: The predicted direction of joint toxic action for neurological effects, an endpoint common to all four components, was greater than additive for the effect of lead on arsenic, arsenic on lead, cadmium on lead, and chromium(VI) on arsenic; and less than additive for the effect of arsenic on chromium(VI).

Renal: The potential health hazard regarding renal effects was found to be lower than the additive, though uncertainty regarding the impact of interactions on this endpoint was less than for neurological toxicity because more information was available.

Cardiovascular: The effects of cadmium on lead and vice versa were additive, but no clear effects were found for mixtures other than those predominated by lead and cadmium.

Hematological: There were no clear additive effects found from testing the various binary mixtures.

Testicular: The potential health hazard may be higher for mixtures of cadmium and lead because of significant additive effects; but arsenic effects on cadmium and chromium(VI) testicular toxicity were less than additive.

Dermal: Interactions of the other mixture components on the dermal toxicity of arsenic were indeterminate for lead and cadmium, and greater than additive for chromium(VI). Carcinogenic: Data regarding effects of the other mixture components on arsenic carcinogenicity were not available. Mechanistic considerations suggested that the effect of chromium(VI) on arsenic carcinogenicity may be greater than additive, but confidence in this assessment was low. Uncertainty regarding interactions was high due to the lack of pertinent information.

4.8 Summary of health effects of mixtures

The limited data indicate that probable additive effects exist between substances within mixtures. It is probable that mixtures of VOCs, PAHs, dioxins and furans, and metals act synergistically, but the extent of any synergism is not currently known or quantifiable. As noted above in the data from ATSDR and PubChem, the toxic effects of the most toxic compound in any mixture are assumed to be the minimum effects of the mixture.

Table 1: Summary of health effects of mixtures

Chemical groups	Health effects
Volatile organic compounds (VOC)	acute and chronic effects on the central nervous system peripheral neuropathy upper respiratory tract (URT) irritation eye irritation damage to the heart, lungs, liver, kidneys, and thyroid damage to the immune system cancer of the bladder and liver haemato-lymphatic cancers including: acute myeloid leukaemia aplastic anaemia multiple myeloma non-Hodgkin's lymphoma
Total petroleum hydrocarbons (TPH)	acute and chronic effects on the central nervous system peripheral neuropathy other effects as noted above for VOCs
BTEX – benzene; toluene; ethyl benzene; xylene	As noted above for VOCs
Trihalomethanes (THM)	acute and chronic effects on the central nervous system liver and kidney damage possibly carcinogenic to liver and kidneys
Polycyclic aromatic hydrocarbons (PAH)	dermatitis photosensitisation bronchitis and pulmonary oedema cancer of the skin cancer of the lung leukaemia
Dioxins and furans	skin damage including chloracne damage to the liver endocrine disruption birth defects; increased cancer incidence overall cancer of the liver cancer of the bladder multiple myeloma non-Hodgkin's lymphoma

Chemical groups	Health effects
Metals antimony arsenic barium beryllium cadmium chromium (III) and (VI) cobalt lead magnesium manganese mercury nickel vanadium	irritant and allergic dermatitis damage to the heart, lungs, liver, and kidneys nasal sinus damage asthma chronic effects on the central nervous system including tremors cancer of the skin cancer of the nasal sinuses cancer of the lungs

5. STATEMENTS OF PRINCIPLES (SoP)

5.1 Statements of Principles (SoPs) determined by the Repatriation Medical Authority (RMA)

SoPs are legislative instruments and have the same legal effect as any legislation passed by Parliament. SoPs exclusively state what factors must exist to establish a causal connection between specific diseases, injuries or death, and service. In 1994 the Australian Government requested the Repatriation Commission, in consultation with veterans' organisations, to prepare legislation to reform the process of decision making about disease causation. The aim was to create a more equitable and consistent system of dealing with claims for disability pensions received from Australian veterans and their dependents. One of the outcomes of the legislative reform was the formation of the Repatriation Medical Authority (RMA) which is an independent statutory authority responsible to the Minister for Veterans' Affairs.

The RMA consists of a panel of five practitioners eminent in fields of medical science. Their role is to determine Statements of Principles (SoPs) for any disease, injury or death that could be related to military service, based on sound medical-scientific evidence. The SoPs state the factors which "must" or "must as a minimum" exist to cause a specific disease, injury or death.

It is important to note that there are two SoPs for each condition as explained on the RMA website. The legislation provides that claims for pension, and the SoPs used to determine claims, should be assessed at two different standards of proof.

5.2 Reasonable hypothesis standard

The more generous (beneficial) standard, known as the reasonable hypothesis standard, applies to veterans and serving members who have operational, or equivalent, service. This includes peacekeeping, hazardous and British nuclear test defence service under the Veterans' Entitlements Act 1986 (the VEA), and warlike and non-warlike service under the Military Rehabilitation and Compensation Act 2004 (the MRCA).

5.3 Balance of probabilities standard

The balance of probabilities standard is for veterans and serving members with non-operational service.

Therefore, for any given condition there are two SoPs. In most cases there are at least slight differences, and in many cases the more generous reasonable hypothesis version of the SoP will contain more causal factors. The legislation requires that the same body of evidence be interpreted differently for the two standards of proof. For the reasonable hypothesis standard, the sound medical-scientific evidence must indicate or point to a causal association between a risk factor and the disease in question. For the balance of

probabilities standard, the sound medical-scientific evidence must show that it is more probable than not that there is a causal association between a risk factor and the disease.

Based on the assumption that the firefighters, whose presumed chemical exposures are the subject of this review, experienced their exposures during non-operational service in peace time, the **balance of probabilities** standard has been applied.

5.4 SoP with direct relevance to this firefighter chemical review

Only one SoP, that for Mesothelioma, lists **"firefighting"** as a causal factor. In this case, the causal link between firefighting and mesothelioma may be because of exposure to asbestos in smoke, rather than any of the substances in this review.

> ***MESOTHELIOMA No. 105 of 2015***
>
> ***Factors: 9*** *(6)* ***firefighting*** *for a cumulative period of at least 1 000 hours before the clinical onset of mesothelioma, where the first exposure occurred at least 15 years before the clinical onset of mesothelioma.*
>
> ***Firefighting*** *means being involved in the direct combat of fires, including activities to control, extinguish, mop-up or prevent fires, or participating in training activities involving fires.*

5.5 SoP with smoke from fires as a causal factor

Two SoPs, **Bronchiectasis** and **Chronic Obstructive Pulmonary Disease,** list "smoke from fires" as a causal factor:

> ***BRONCHIECTASIS No. 31 of 2017***
>
> ***Factors:*** *9. (6)* ***inhaling vapours, gases or fumes of a chemical agent*** *from the specified list of chemical agents: (a) resulting in signs and symptoms of severe acute lower respiratory damage requiring medical attention within 48 hours after exposure; and (b) the persistence of respiratory symptoms and signs for at least one week after exposure, within the five years before the clinical onset of bronchiectasis.*
>
> ***Specified list of chemical agents means:*** *(a) ammonia; (b) chlorine; (c) oxides of nitrogen; (d) oxides of sulphur; (e) paraquat; (f) phosgene; or (g)* ***smoke from fires****.*
>
> ***CHRONIC OBSTRUCTIVE PULMONARY DISEASE No. 38 of 2014 and Amendment No. 129 of 2015***
>
> ***Factors:*** *6. (b)* ***inhaling a respiratory tract irritant from the specified list:*** *(i) resulting in signs and symptoms of severe acute lower respiratory damage requiring medical attention within 48 hours after exposure; and (ii) the persistence of respiratory symptoms and signs for at least one week after exposure, within the ten years before the clinical onset of chronic obstructive pulmonary disease; or (c)* ***inhaling smoke from the combustion of wood, charcoal, coal or other biomass***

***or fossil fuel**, in an enclosed space: (i) for a cumulative period of at least 5 000 hours, before the clinical onset of chronic obstructive pulmonary disease; and (ii) where that exposure has ceased, the clinical onset of chronic obstructive pulmonary disease has occurred within 20 years of cessation.*

***A respiratory tract irritant from the specified list** means: (a) ammonia; (b) chlorine; (c) dust; (d) Lewisite; (e) oxides of nitrogen; (f) oxides of sulphur; (g) phosgene; (h) phthalic anhydride; **(i) smoke from fires**; (j) sulphur mustard (mustard gas); or (k) another respirable agent which causes comparable tissue damage.*

5.6 SoP with smoke as a causal factor

One SoP, that for **Fibrosing Interstitial Lung Disease**, lists "smoke" as a causal factor:

FIBROSING INTERSTITIAL LUNG DISEASE No. 54 of 2013

***Factors:** 6. (k) developing inflammation of the pulmonary interstitium due to inhalation of **toxic gases or fumes** within the 12 months before the clinical onset of fibrosing interstitial lung disease.*

***Toxic gases or fumes** means toxic agents, including anhydrous ammonia fumes, **smoke,** oxides of sulphur, oxides of nitrogen, chlorine or phosgene.*

5.7 SoP with smoke in an enclosed space as a causal factor

One SoP, that for Malignant neoplasm of the Lung, lists "smoke in an enclosed space" as a causal factor. Other factors listed include substances subject to this review – arsenic, cadmium, chromium (VI), nickel - but not in exposure conditions stated in the SoP:

MALIGNANT NEOPLASM OF THE LUNG No. 93 of 2014

***Factors:** 6 (n) inhaling fumes, vapours or dusts of a **metal or metalloid** from the specified list: (i) for a cumulative period of at least 3 500 hours before the clinical onset of malignant neoplasm of the lung; and (ii) where the first inhalation of fumes, vapours or dusts occurred at least ten years before the clinical onset of malignant neoplasm of the lung; (q) being exposed to **arsenic** as specified before the clinical onset of malignant neoplasm of the lung, where the first exposure to arsenic occurred at least ten years before the clinical onset of malignant neoplasm of the lung; or (r) **inhaling smoke** from the combustion of coal, wood, charcoal or another solid biomass fuel while in an enclosed space with a visible smoke haze: (i) for a cumulative period of at least 15 000 hours before the clinical onset of malignant neoplasm of the lung; and (ii) where the first inhalation of smoke commenced at least ten years before the clinical onset of malignant neoplasm of the lung.*

***A metal or metalloid from the specified list** means: (a) arsenic and inorganic arsenic compounds; (b) beryllium and beryllium compounds; (c) cadmium and cadmium compounds; (d) hexavalent chromium (chromium VI) compounds; or (e) mixtures that include nickel metal and nickel compounds.*

Being exposed to arsenic as specified" *means: (a) consuming drinking water with an average arsenic concentration of at drinking water resulting in a cumulative total arsenic exposure equivalent to having consumed drinking water containing at least 50 micrograms per litre for at least ten years; or (c) having clinical evidence of chronic arsenic toxicity.*

5.8 SoP with inhalation of irritants as a causal factor

The SoP for Asthma lists both 'immunological or non-immunological" stimuli from irritant metals, chemicals and gases, as well as "substances with irritant properties" as causal factors:

ASTHMA No. 61 of 2012

Factors: *6. (a) being* ***exposed to an immunologic or non-immunologic stimulus*** *within the 24 hours before the clinical onset of asthma; or (b) for reactive airways dysfunction syndrome only,* ***inhaling very high concentrations of a substance with irritant properties****, where such inhalation has resulted in acute toxic lower respiratory tract effects, within the 24 hours before the clinical onset of asthma; or (e) being* ***exposed to an immunologic or non-immunologic stimulus*** *within the 24 hours before the clinical worsening of asthma;*

An immunologic or non-immunologic stimulus *means a substance, activity or irritant which can cause inflammation of the airways and bronchial hyperresponsiveness. Examples include* ***metals****, drugs, cereal dusts, wood dusts,* ***chemical fumes****, moulds,* ***irritant gases*** *(including mustard gas), exercise, cold air, air pollutants, respiratory infections and proteins derived from animals, insects and fish.*

Reactive airways dysfunction syndrome *means an asthma-like condition satisfying the following criteria: (a) a documented absence of preceding asthma or other ongoing bronchial disorders; (b) onset of symptoms after a single exposure incident or accident; (c)* ***inhalation of very high concentrations of a substance with irritant properties****; (d) onset of symptoms within 24 hours after the acute exposure, with persistence of symptoms for at least three months; (e) symptoms simulate asthma; (f) presence of reversible airflow obstruction on pulmonary function tests, or the presence of nonspecific bronchial hyperresponsiveness; and (g) other pulmonary diseases have been ruled out.*

5.9 SoP with exposure to an irritant as a causal factor

The SoP for Irritant Contact Dermatitis lists "exposure to an irritant" as a causal factor, where an irritant is not specified, but includes a chemical which damages the skin:

IRRITANT CONTACT DERMATITIS No. 111 of 2011

Factors: *6. (a) having direct cutaneous* ***exposure of the affected area to an irritant*** *within the three days before the clinical onset of irritant contact dermatitis; or (b) having direct cutaneous exposure of the affected area to an* ***irritant*** *within the three days before the clinical worsening of irritant contact dermatitis.*

Irritant *means an agent or substance, for example a chemical, which damages the epidermis on contact and causes inflammation of the contacted skin. It does not include physical agents such as heat, cold, solar radiation or other forms of radiation.*

5.10 SoP with inhalation of polycyclic aromatic hydrocarbons as a causal factor

The SoP for Malignant Neoplasm of the Bladder lists "inhaling polycyclic aromatic hydrocarbons" as formed "during the combustion of organic material" as a causal factor:

MALIGNANT NEOPLASM OF THE BLADDER No. 97 of 2011

Factors: *6. (c) inhaling fumes containing high concentrations of* ***polycyclic aromatic hydrocarbons****, or ingesting polycyclic aromatic hydrocarbons, in the specified circumstances for a cumulative period of at least 5000 hours before the clinical onset of malignant neoplasm of the bladder, where the first exposure occurred at least ten years before the clinical onset of malignant neoplasm of the bladder.*

Polycyclic aromatic hydrocarbons *mean hydrocarbons with three or more condensed aromatic rings in which certain carbon atoms are common to two or three rings. Polycyclic aromatic hydrocarbons occur in crude oil, shale oil and coal tars, and can be* ***formed during the combustion of organic material*** *or during high temperature processing of crude oil, coal, coke or other industrial carbon compounds.*

5.11 SoP with chemical compounds as causal factors

Specific chemicals and metallic compounds have been listed as causal factors in a further twelve SoPs, though in each case the exposure conditions do not include firefighting or exposure to the substances in smoke.

5.12 SoP for peripheral neuropathy

The SoP for **peripheral neuropathy** lists carbon disulphide, hexane, TCDD, PCBs, and cobalt as causal factors:

PERIPHERAL NEUROPATHY No. 75 of 2014

Factors: *6 (m) inhaling, ingesting or having cutaneous contact with a* ***volatile substance*** *from the specified list, in an unventilated and confined space: (i) on at*

*least 30 occasions within a continuous period of six months before the clinical onset of peripheral neuropathy; and (ii) where contact with a volatile substance from the specified list has ceased, the clinical onset of peripheral neuropathy has occurred within three months of cessation; (o) inhaling, ingesting or having cutaneous contact with a chemical agent contaminated by **2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD)** within the 30 days before the clinical onset of peripheral neuropathy; or (r) being **poisoned with an agent** as specified within the 30 days before the clinical onset of peripheral neuropathy*

***A volatile substance from the specified list** means: (a) allyl chloride; (b) **carbon disulphide**; (c) methyl n-butyl ketone (MNBK); or (d) **n-hexane**;*

***Being poisoned with an agent as specified** means having clinical, haematological or biochemical evidence of poisoning with one of the following agents: (a) **a polychlorinated biphenyl**; (b) aniline-denatured rapeseed oil; (c) brevetoxin; (d) ciguatera toxin; (e) **cobalt;** (f) fruit of the Buckthorn pyridyl methyl-N'-p-nitrophenyl urea (Vacor); (k) saxitoxin; (l) tetrodotoxin; (m) thallium salts; (n) tri-cresyl phosphate; or (o) tri-ortho-cresyl phosphate;*

***Inhaling, ingesting or having cutaneous contact with a chemical agent contaminated by 2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD)** means: (a) decanting or spraying; (b) cleaning or maintaining equipment used to apply; (c) being sprayed with; (d) handling or sawing timber treated with; (e) being in an environment shrouded in dust from timber treated with TCDD.*

5.13 SoP for Parkinson's Disease and secondary Parkinsonism

The SoP for **Parkinson's Disease and Secondary Parkinsonism** also lists multiple agents as causal factors – "carbon disulphide, manganese, and cyanide":

PARKINSON'S DISEASE AND SECONDARY PARKINSONISM No. 56 of 2016

***Factors:** 9 (n) inhaling **carbon disulphide** vapour in an enclosed space, or having cutaneous contact with carbon disulphide, for a cumulative period of at least 500 hours, within the ten years before the clinical onset of secondary parkinsonism; (p) being exposed to **manganese** as specified for a cumulative period of at least 500 hours, within the ten years before the clinical onset of secondary parkinsonism; (r) inhaling, ingesting or having cutaneous contact with cyanide, and having clinical, haematological or biochemical evidence of cyanide intoxication, within the six weeks before the clinical onset of secondary parkinsonism.*

***Being exposed to manganese as specified** means: (a) working in the mining or smelting of ores containing manganese; or (b) welding with rods containing manganese; or (c) inhaling dust containing manganese.*

5.14 SoP for chloracne

The SoP for **Chloracne** lists specific "dioxins and furans" as causal factors:

> ***CHLORACNE No. 18 of 2012***
>
> ***Factors:*** *6. (a) inhaling, ingesting or having cutaneous contact with a* ***polyhalogenated aromatic hydrocarbon*** *from the specified list, or a chemical mixture containing a polyhalogenated aromatic hydrocarbon from the specified list, within the three months before the clinical onset of chloracne; (c) inhaling, ingesting or having cutaneous contact with a* ***polychlorodibenzofuran (PCDF)*** *from the specified list, within the three months before the clinical onset of chloracne; (d) inhaling, ingesting or having cutaneous contact with a* ***polychlorodibenzo-para-dioxin (PCDD)*** *from the specified list, within the three months before the clinical onset of chloracne.*
>
> ***A polychlorodibenzofuran (PCDF) from the specified list*** *means: (a) 2,3,7,8-tetrachlorodibenzofuran (TCDF); (b) pentachlorodibenzofurans (PeCDFs); (c) hexachlorodibenzofurans (HxCDFs); (d) heptachlorodibenzofurans (HpCDFs); or (e) octachlorodibenzofurans (OCDFs).*
>
> ***A polychlorodibenzo-para-dioxin (PCDD) from the specified list*** *means: (a) 2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD); (b) 1,2,3,7,8-pentachlorodibenzo-para-dioxin (PeCDD); (c) hexachlorodibenzo-para-dioxin (HxCDD); (d) 1,2,3,4,6,7,8-heptachlorodibenzo-para-dioxin (HpCDD); or (e) octachlorodibenzo-para-dioxin (OCDD).*
>
> ***A polyhalogenated aromatic hydrocarbon from the specified list*** *means: (a) azobenzenes, including 3,3',4,4'-tetrachloroazobenzene; (b) azoxybenzenes, including 3,4,3',4'-tetrachloroazoxybenzene; (c) mono-ortho-substituted PCB congeners 105, 114, 118, 123, 156, 157, 167 or 189; (d) non-ortho-substituted PCB congeners 77, 81, 126, or 169; (e) o-dichlorobenzene; (f) polybrominated biphenyls (PBBs); (g) polybromodibenzofurans, including tetrabromodibenzofuran; (h) polychlorinated naphthalenes, including pentachloronaphthalene (Halowax 1013) and hexachloronaphthalene (HCN); (i) polychromonaphthalenes; or (j) triazoloquinoxalines.*

5.15 SoPs related to benzene, other than in smoke

Three SoPs, being those for **Acute Myeloid Leukaemia, Aplastic Anaemia, and Myelodysplastic Syndrome**, list "benzene", from various exposures other than in smoke, as a causal factor:

> ***ACUTE MYELOID LEUKAEMIA No 72 of 2015***
>
> ***Factor:*** *9. (7)* ***being exposed to benzene****: (a) for a cumulative total of at least 1 250 hours within a continuous period of ten years before the clinical onset of acute myeloid leukaemia; and (b) where the first exposure in that period occurred at least*

five years before the clinical onset of acute myeloid leukaemia; (8) receiving greater than five ppm-years of cumulative ***exposure to benzene*** *before the clinical onset of acute myeloid leukaemia, and where the first exposure occurred at least five years before the clinical onset of acute myeloid leukaemia;*

Being exposed to benzene means: *(b) inhaling benzene vapour where such exposure occurs at an ambient 8-hour time-weighted average benzene concentration exceeding five parts per million.*

APLASTIC ANAEMIA No. 51 of 2012 and Amendment No.32 of 2016

Factors: *6. (g) being exposed to* ***benzene*** *on at least 30 days within the six months before the clinical onset of aplastic anaemia;*

Being exposed to benzene *means: (c) inhaling benzene vapour where such exposure occurs at an ambient 8-hour time-weighted average benzene concentration exceeding five parts per million.*

MYELODYSPLASTIC SYNDROME No. 74 of 2015

Factors: *(7) receiving greater than five ppm-years of* ***cumulative exposure to benzene*** *before the clinical onset of myelodysplastic syndrome, and where the first exposure occurred at least five years before the clinical onset of myelodysplastic syndrome*

Being exposed to benzene *means: (c) inhaling benzene vapour where such exposure occurs at an ambient 8hour time-weighted average benzene concentration exceeding five parts per million.*

5.16 SoPs with exposure to TCDD, other than in smoke

Three SoPs, namely **Myeloma, Porphyria Cutanea Tarda,** and **Soft Tissue Sarcoma,** list "exposure to TCDD" as a causal factor, though not in the context of smoke exposure:

MYELOMA No. 70 of 2012 - Amendment Statement of Principles concerning MYELOMA No. 73 of 2014

Factors: *"(ca) having exposure to* ***2,3,7,8 tetrachlorodibenzo-para-dioxin (TCDD)*** *sufficient to produce an expected initial serum TCDD level of at least 1 500 parts per trillion before the clinical onset of myeloma;*

PORPHYRIA CUTANEA TARDA No. 44 of 2012

Factors: *6. (b) having exposure as specified to a chemical agent contaminated with* ***2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD)*** *within the two years before the clinical onset of porphyria cutanea tarda*

Having exposure as specified to a chemical agent contaminated with 2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD) *means being in an environment shrouded in dust from timber treated with a specified chemical agent; (b) being sprayed with a specified chemical agent; (c) cleaning or maintaining equipment used to apply a specified chemical agent; (d) decanting or spraying a specified chemical agent; (e) handling or sawing timber treated with a specified chemical agent; or (f) using cutting oils contaminated with a specified chemical agent.*

SOFT TISSUE SARCOMA No. 6 of 2015

Factors: *6. (b) inhaling, ingesting or having cutaneous contact with a chemical agent contaminated by* ***2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD)*** *for a cumulative period of at least 250 hours, at least two years before the clinical onset of soft tissue sarcoma.*

Inhaling, ingesting or having cutaneous contact with a chemical agent contaminated by 2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD) *means: (a) decanting or spraying; (b) cleaning or maintaining equipment used to apply; (c) being sprayed with; (d) handling or sawing timber treated with; (e) being in an environment shrouded in dust from timber treated with TCDD.*

5.17 SoP for vinyl chloride exposure, not in smoke

The SoP for **Malignant Neoplasm of the Liver** lists "inhaling gaseous vinyl chloride" as a causal factor, but not in the context of smoke exposure:

MALIGNANT NEOPLASM OF THE LIVER No. 22 of 2011

Factors: *6 (k) inhaling a cumulative dose of at least 2500 ppm-years of* ***gaseous vinyl chloride*** *at least five years before the clinical onset of malignant neoplasm of the liver.*

5.18 SoP for arsenic

Finally, the SoPs for **Malignant Neoplasm of the Renal Pelvis and Ureter,** and **Non-Melanotic Malignant Neoplasm of the Skin** list "being exposed to arsenic" as a causal factor, but not in the context of smoke exposure:

MALIGNANT NEOPLASM OF THE RENAL PELVIS AND URETER No. 99 of 2011

Factors: *6 (c) being exposed to* ***arsenic*** *as specified before the clinical onset of malignant neoplasm of the renal pelvis or ureter, where the first exposure to arsenic occurred at least ten years before the clinical onset of malignant neoplasm of the renal pelvis or ureter;*

Being exposed to arsenic as specified *means: (a) consuming drinking water with an average arsenic concentration of at least 50 micrograms per litre for a cumulative period of at least ten years; (b) consuming drinking water resulting in a cumulative*

total arsenic exposure equivalent to having consumed drinking water containing at least 50 micrograms per litre for at least ten years; or having clinical evidence of chronic arsenic toxicity.

NON-MELANOTIC MALIGNANT NEOPLASM OF THE SKIN No. 8 of 2016

Factors: *9 (11) being exposed to* ***arsenic*** *as specified before the clinical onset of non-melanotic malignant neoplasm of the skin, where the first exposure to arsenic occurred at least ten years before the clinical onset of non-melanotic malignant neoplasm of the skin;*

Being exposed to arsenic as specified *means: (a) consuming arsenic containing compounds (for example, Fowler's solution) for a cumulative period of at least three months; or (b) consuming drinking water with an average arsenic concentration of at least 50 micrograms per litre for a cumulative period of at least five years; or (c) inhaling, ingesting or having cutaneous contact with a pesticide containing arsenic, or arsenic in copper smelting operations, for a cumulative period of at least 1 000 hours; or (d) having clinical evidence of chronic arsenic toxicity.*

5.19 Summary of SoPs and the firefighter chemical review

The occupation of firefighter is listed as a factor in only one SoP – mesothelioma. Smoke from fires is listed as a factor for bronchiectasis and chronic obstructive pulmonary disease, and smoke in an enclosed space is a factor in the SoP for fibrosing interstitial lung disease and malignant neoplasm of the lung. Inhalation of irritants is a factor for asthma, and exposure to irritants is a factor for irritant dermatitis.

Additionally, many of the contaminants listed in Appendix C, are factors in a wide range of SoPs, but not in exposures experienced by firefighters. These contaminants include arsenic, benzene, carbon disulfide, cobalt, dioxins and furans, manganese, PAHs, vinyl chloride, and VOCs.

Table 2: Tabulated summary of contaminants listed as factors, correlated with SoPs:

Factor	Statement of Principles (SoP)
Firefighting for a cumulative period of at least 1 000 hours before the clinical onset of mesothelioma, where the first exposure occurred at least 15 years before the clinical onset of mesothelioma.	MESOTHELIOMA No. 105 of 2015
Inhaling vapours, gases or fumes of a chemical agent including **smoke from fires**.	BRONCHIECTASIS No. 31 of 2017
Inhaling a respiratory tract irritant from the specified list including smoke from fires; inhaling smoke from the combustion of wood, charcoal, coal or other biomass or fossil fuel	CHRONIC OBSTRUCTIVE PULMONARY DISEASE No. 38 of 2014 and Amendment No. 129 of 2015

Factor	Statement of Principles (SoP)
Inhalation of toxic gases or fumes including smoke	FIBROSING INTERSTITIAL LUNG DISEASE No. 54 of 2013
Inhaling smoke from the combustion of coal, wood, charcoal or another solid biomass fuel	MALIGNANT NEOPLASM OF THE LUNG No. 93 of 2014
Volatile organic compounds (VOC) as irritants in general	ASTHMA No. 61 of 2012 IRRITANT CONTACT DERMATITIS No. 111 of 2011
Volatile organic compounds (VOCs), in the form of benzene	ACUTE MYELOID LEUKAEMIA No 72 of 2015 APLASTIC ANAEMIA No. 51 of 2012 and Amendment No.32 of 2016 MYELODYSPLASTIC SYNDROME No. 74 of 2015
Volatile organic compounds (VOC) in the form of carbon disulfide and hexane	PERIPHERAL NEUROPATHY No. 75 of 2014
Volatile organic compounds (VOC) in the form of carbon disulfide	PARKINSON'S DISEASE AND SECONDARY PARKINSONISM No. 56 of 2016
Volatile organic compounds (VOC) in the form of vinyl chloride	MALIGNANT NEOPLASM OF THE LIVER No. 22 of 2011
Polycyclic aromatic hydrocarbons (PAH)	MALIGNANT NEOPLASM OF THE BLADDER No. 97 of 2011
Dioxins and furans	PERIPHERAL NEUROPATHY No. 75 of 2014 CHLORACNE No. 18 of 2012 MYELOMA No. 70 of 2012 – Amendment Statement of Principles concerning MYELOMA No. 73 of 2014 PORPHYRIA CUTANEA TARDA No. 44 of 2012 SOFT TISSUE SARCOMA No. 6 of 2015
Metals	
arsenic, beryllium, cadmium, chromium, nickel	MALIGNANT NEOPLASM OF THE LUNG No. 93 of 2014
arsenic	MALIGNANT NEOPLASM OF THE RENAL PELVIS AND URETER No. 99 of 2011
cobalt	NON-MELANOTIC MALIGNANT NEOPLASM OF THE SKIN No. 8 of 2016
manganese	PERIPHERAL NEUROPATHY No. 75 of 2014 PARKINSON'S DISEASE AND SECONDARY PARKINSONISM No. 56 of 2016

6 LITERATURE REVIEW – ADDRESSING THE GAPS

6.1 Introduction

A literature review has been undertaken to address the gaps in relation to the reported health effects arising from the contaminants identified in Appendix C that have not been listed specifically as factors in the SoPs. The resources of PubMed[2] were utilized in the literature search, and initially over 500 scientific publications were found by searching for "firefighters and exposures", "firefighters and health effects", and "firefighters and cancer". The publications of most relevance to this review were those demonstrating probable causal relationships, and these have been summarised in the following sections.

6.2 Firefighter exposures

Published research on exposures experienced by firefighters at training and operational fires first appeared in the 1980s as the necessary analytical methods became available.

One of the early reports was from 1985[ii] when a specially designed system was used to collect samples of aerosols and vapors directly from the smoke plumes at a fireperson training facility. The chemical analyses demonstrated the presence of polynuclear aromatic hydrocarbons and alkanes in both the vapour and particulate phases of the smoke plume. Though all constituents of the complex mixture of organics were not identified, GC/MS (gas chromatography/ mass spectrometry) analysis confirmed the presence of several known carcinogens including polynuclear aromatic hydrocarbons (PAHs).

Following concerns about health risks to firefighters, a 1988 study in New York State[iii] assessed the types and levels of exposure encountered by firefighters during their routine occupational duties. Twenty-six firefighters were monitored during firefighting activities with personal, portable, and ambient environmental sampling devices. The results indicated that firefighters were frequently exposed to significant concentrations of hazardous materials including carbon monoxide, benzene, sulphur dioxide, hydrogen cyanide, aldehydes, hydrogen chloride, dichlorofluoromethane, and particulates. Furthermore, in many cases of the worst exposure to these materials, respiratory protective equipment was not used. The authors concluded that many of the materials found through the monitoring study had been implicated in the production of cardiovascular, respiratory, or neoplastic diseases, thus providing an explanation for the alleged increased risk for these illnesses among firefighters.

In 2001, Austin and co-authors[iv] monitored volatile organic compounds (VOCs) in the smoke at nine municipal structural fires to identify potential sources of long-term health risks. They stated that they expected firefighters to be exposed to different substances at different fires;

[2] PubMed is an online service of the US National Library of Medicine, National Institutes of Health. PubMed comprises more than 28 million citations for biomedical literature from MEDLINE, life science journals, and online books. Citations may include links to full-text content from PubMed Central and publisher web sites.

but instead found that exposures to VOCs at the structural fires was like that found in smoke at training fires. At all monitoring sites, the VOCs found were typically benzene, toluene, 1,3-butadiene, naphthalene, and styrene.

More recent research in Queensland by Kirk and Logan[v] has found that cumulative exposures of firefighting instructors to toxic PAHs generated from fire training exercises had the potential to exceed exposures arising from operational fires. Most PAHs found during five fire training exercises were naphthalene, phenanthrene, and acenaphthalene, but benzo(a)pyrene was the most toxic of the PAH mixture. The study also found that concentrations of PAHs inside the firefighters' protective equipment were lower than outside, but still included the presence of carcinogenic PAHs. Exposures to PAHs from a single 40-minute training exercise were equivalent to the highest 8-hour time-weighted average exposure reported from occupational exposures in industry.

In summary, these reports on exposures add support to the assumption that the contaminants found at the site of ADF firefighter training at Point Cook could have been present in the smoke to which the firefighters were exposed.

6.3 Firefighters and respiratory dysfunction

Persistent respiratory dysfunction has been reported in a Danish study[vi] whose authors investigated respiratory effects among emergency services first responders and residents exposed to combustion products from a chemical waste depot fire. One hundred and thirty-eight people who were downwind of the fire were identified and followed-up six years after the fire. Detailed tests for bronchial hyperresponsiveness were performed, and those suspected of suffering from RADS (reactive airways dysfunction syndrome) were compared with healthy controls for exposure to combustion products, lung function, and bronchial hyperresponsiveness. It was concluded that persistent respiratory symptoms and bronchial hyperresponsiveness were associated with combustion products generated in the chemical waste depot fire which occurred more than six years earlier.

In summary, the Danish study supports the SoPs for bronchiectasis, chronic obstructive pulmonary disease, fibrosing interstitial lung disease, and asthma which all list smoke, in various exposures, as a causal factor. There were no publications providing probable causal links between firefighters and other non-cancer long-term health outcomes.

6.4 Firefighters and cancer

Two publications in 2006 have used meta-analysis techniques in reviewing numerous previous reports on cancer incidence and mortality:

Le Masters and co-authors[vii] reviewed 32 studies on firefighters to determine both quantitatively and qualitatively the cancer risk using a meta-analysis. A comprehensive search of computerized databases and bibliographies from identified articles was performed. Three criteria used to assess the probable, possible, or unlikely risk for 21 cancers included

pattern of meta-relative risks, study type, and heterogeneity testing. The findings indicated that firefighters had a probable cancer risk for: (i) multiple myeloma with a summary risk estimate (SRE) of 1.53 and 95% confidence interval (CI) of 1.21–1.94; (ii) non-Hodgkin's lymphoma (SRE 1.51, 95%CI 1.31–1.73); and (iii) prostate (SRE 1.28; 95% CI 1.15–1.43). Testicular cancer was upgraded to probable because it had the highest summary risk estimate (SRE 2.02; 95% CI 1.30–3.13). Eight additional cancers were listed as having a "possible" association with firefighting. The authors concluded that their results confirmed previous findings of an elevated relative risk for multiple myeloma among firefighters. In addition, a probable association with non-Hodgkin lymphoma, prostate, and testicular cancer was demonstrated.

Following a comprehensive computer-assisted search of databases for the years 1966 to 2005, Youakim[viii] selected sixteen studies in which researchers had focused on the risks of six specific cancers of interest among firefighters using a mortality outcome; and ten studies that were based on disease morbidity or occurrence. The studied populations were from five countries, being Australia, Canada, New Zealand, Sweden and the United States. The risks were estimated for cancers of the colon, bladder, kidneys, and brain, and for non-Hodgkin's lymphoma and leukaemia. The risk of these six cancers was not found to be markedly elevated when cohort mortality studies were considered, but mildly increased risks for kidney cancer and non-Hodgkin's lymphoma were found when all mortality studies were considered. A sub-cohort analysis, using years of service as a surrogate for exposure, revealed that firefighters with 30 or more years of employment had a significantly increased risk of colon cancer [RR 1.51, CI 1.05-2.11], kidney cancer [RR 6.25, CI 1.70-6.25], brain cancer [RR 36.12, CI 4.03-120.43], and bladder cancer [RR 5.7, CI 1.56-14.63]. There was a statistically non-significant raised RR of 1.72 for non-Hodgkin's lymphoma among firefighters with 20 or more years of employment, and kidney cancer risk was significantly elevated as early as the second decade of employment.

A study published by Daniels et al[ix] in 2015 is important because it enhanced the work of Youakim by addressing the limitations in previous research regarding exposure-response relationships. The authors conducted internal analyses of disease rates among career firefighters in a large pooled cohort. Eight cancer and four non-cancer outcomes were examined using conditional logistic regression. Incidence density sampling was used to match each case to 200 controls on attained age. Days accrued in firefighting assignments (exposed-days), run totals (fire-runs) and run times (fire-hours) were used as exposure surrogates. Piecewise constant models were used to examine risk differences by time since exposure, age at exposure, and calendar period. Among 19,309 male firefighters eligible for the study, there were 1,333 cancer deaths and 2609 cancer incidence cases. Significant positive associations between fire-hours and lung cancer mortality and incidence were evident. A similar relation between leukaemia mortality and fire-runs was also found. The lung cancer associations were nearly linear in cumulative exposure, while the association with leukaemia mortality was attenuated at higher exposure levels and greater for recent exposures. Significant negative associations were evident for the exposure surrogates and colorectal and prostate cancers, suggesting a healthy worker survivor effect possibly enhanced by medical screening. Lung cancer and leukaemia mortality risks were modestly

increasing with firefighter exposures. These findings add to evidence of a causal association between firefighting and cancer, though the authors cautioned that small effects merit careful interpretation.

Further to the research described above, a major study of Australian firefighters by Glass et al[x], published in 2016, has used a range of exposure metrics to address limitations in other studies. The authors investigated mortality and cancer incidence of paid male Australian firefighters, and of subgroups of firefighters, by era of first employment, duration of employment, and number and type of incidents attended. Participating fire agencies supplied records of individual firefighters including their job histories and incidents attended. The cohort was linked to the Australian National Death Index and Australian Cancer Database. Firefighters were grouped into tertiles by duration of employment and by number of incidents attended. Relative mortality ratios and relative incidence ratios were calculated. Analyses were carried out separately for full-time and part-time male firefighters. The study included 17,394 full-time and 12,663 part-time firefighters. Compared with the Australian population, there were significant increases in cancer risks: all cancers for all paid firefighters [RR 1.09, CI 1.03-1.14]; prostate cancer for full-time firefighters [RR 1.23, CI 1.10-1.37]; prostate cancer for part-time firefighters [RR 1.51, CI 1.28-1.77]; melanoma for full-time firefighters [RR 1.45, CI 1.26-1.66]; and melanoma for part-time firefighters [RR 1.43, CI 1.15-1.76]. Kidney cancer was associated with longer service in internal analyses for paid firefighters. Prostate cancer was associated with longer service and increased attendance at fires, particularly structural fires for full-time firefighters. The overall risk of mortality was significantly decreased and almost all major causes of death were significantly reduced for paid firefighters. The authors concluded that male paid firefighters have an increased risk of cancer.

In summary, the above publications support the view that, on the balance of probabilities, exposures experienced by firefighters contribute materially to the subsequent development of cancers in general and to some specific malignancies: cancers of the bladder, brain, colon, kidney, lung, prostate, and testes; and leukaemia, multiple myeloma, and non-Hodgkin's lymphoma.

7 CONCLUSIONS

Overall, this Firefighter Chemical Review, combined with a review of the SoPs and a literature review, concludes that **"firefighting"**, as in the current Sop for mesothelioma, should be considered as a factor in other Sops as indicated in the following Table 3:

Table 3: Firefighter exposures as a factor in future SoPs

Factor	Statement of Principles (SoP)
Firefighting for a cumulative period before the clinical onset of the condition, where the first exposure occurred before the clinical onset of the condition	Acute myeloid leukaemia Adenocarcinoma of the kidney Aplastic anaemia Asthma Bronchiectasis Chloracne Chronic obstructive pulmonary disease Fibrosing interstitial lung disease Irritant contact dermatitis Malignant neoplasm of the bladder Malignant neoplasm of the brain Malignant neoplasm of the colorectum Malignant neoplasm of the liver Malignant neoplasm of the lung Malignant neoplasm of the prostate Malignant neoplasm of the renal pelvis and ureter Malignant neoplasm of the testis Mesothelioma Myelodysplastic syndrome Myeloma Non-Hodgkin's lymphoma Non-melanotic malignant neoplasm of the skin Parkinson's disease and secondary parkinsonism Peripheral neuropathy Porphyria cutanea tarda Soft tissue sarcoma

8 APPENDICES

Appendix A: List of chemical contaminants provided to Department of Defence in support of HLA ENSR Human Health Risk Assessment, 2 July 2007

List of chemical contaminants provided to Department of Defence in support of HLA ENSR Human Health Risk Assessment, 2 July 2007.

Limitations of Data Presented

The data presented in the individual media tables represents the list of contaminants that were analysed for within the former Fire Training Area (FTA) at RAAF Williams Base, Point Cook. The concentrations reported represent the highest recorded concentration identified for each contaminant in the five-individual media of soil, groundwater, sediment, soil vapour and DNAPL at the time of sampling. The contaminants within the data tables were selected for laboratory analysis based upon the site history and relevant background information and include typically relevant environmental broad screening contaminant categories and the specific compounds associated with the laboratory analytical suite. Other contaminants may be present at the FTA Site but have not been listed as they were not included in the laboratory testing program as there was no historical basis to consider analysis. Contamination may also be present at levels above those reported in the data table within the FTA at locations not investigated or within samples collected and not analysed and laboratory techniques may have changed over time including limits of detection and analytical methods. In addition, site conditions may have altered following the time of sampling and concentrations detected may have altered over time.

Chemical Name	Media	Units	Maximum Detected Concentration
1,1,1-trichloroethane	Groundwater	µg/L	447
	Soil Vapour	µg/m3	11
1,1,2,2-Tetrachloroethane	DNAPL	%	3.6
	Groundwater	µg/L	106,000
	Soil	mg/kg	321,000
	Soil Vapour	µg/m3	23,600
1,1,2-Trichloroethane	DNAPL	%	10
	Groundwater	µg/L	876,000
	Soil	mg/kg	1,260,000
	Soil Vapour	µg/m3	1,380,000
1,1'-Biphenyl	DNAPL	%	0.3
1,1-bis-p-Tolylethane	DNAPL	%	2.2
1,1-Dichloroethane	DNAPL	%	0.7
	Groundwater	µg/L	170,000
	Soil	mg/kg	136,000
	Soil Vapour	µg/m3	747,000
1,1-Dichloroethene	DNAPL	%	0.2
	Groundwater	µg/L	61,200
	Soil	mg/kg	26,500
	Soil Vapour	µg/m3	272,000
1,1-Diphenylethane	DNAPL	%	3.7
1,2,4-trichlorobenzene	Groundwater	µg/L	14
	Soil	mg/kg	8.9
	Soil Vapour	µg/m3	200
1,2,4-trimethylbenzene	Groundwater	µg/L	228
	Soil	mg/kg	11
	Soil Vapour	µg/m3	1,340
1,2-dichlorobenzene	Groundwater	µg/L	6
1,2-Dichloroethane	DNAPL	%	3.5
	Groundwater	µg/L	2,150,000
	Soil	mg/kg	1,140,000
	Soil Vapour	µg/m3	45,900
1,2-Dichloroethene	Groundwater	µg/L	56,600
	Soil	mg/kg	969
1,2-dichloropropane	Groundwater	µg/L	2
1,2-Diethylbenzene	DNAPL	%	0.7
1,2-Dihydrophenanthrene	DNAPL	%	0.6
1,2-Diphenylcyclobutane	DNAPL	%	0.4
1,2-Diphenylethane	DNAPL	%	0.6
1,2-Diphenylpropane	DNAPL	%	1.2
1,3,5-trimethylbenzene	Groundwater	µg/L	154
	Soil Vapour	µg/m3	550
1,3-Butadiene	Soil Vapour	µg/m3	400
1,3-dichlorobenzene	Groundwater	µg/L	56,300
	Soil	mg/kg	7
	Soil Vapour	µg/m3	1,180
1,3-dichloropropane	Groundwater	µg/L	1270
	Soil	mg/kg	30
1,4-Dichloro-2-butene	DNAPL	%	1.2
1,4-dichlorobenzene	Groundwater	µg/L	43,500
	Soil	mg/kg	6.8
	Soil Vapour	µg/m3	450
1,4-Diethylbenzene	DNAPL	%	0.5
1,4-Dihydroxy-9,10-anthracenedione	DNAPL	%	0.7
1,4-Diphenylbutadiene	DNAPL	%	0.7
1,4-Diphenylbutane	DNAPL	%	0.4
1234678-HpCDD	Groundwater	pg/L	24
	Sediment	pg/g	30.6
1234678-HpCDD I-TEF	Groundwater	pg/L	0.01
1234678-HpCDD I-TEQ3 (LOR)	Groundwater	pg/L	0.9
1234678-HpCDD WHO-TEF	Groundwater	pg/L	0.01
1234678-HpCDD WHO-TEQ3 (LOR)	Groundwater	pg/L	0.9
1234678-HpCDF	Sediment	pg/g	4.1
123478-HxCDF	Sediment	pg/g	2.6

Chemical Name	Media	Units	Maximum Detected Concentration
1-Ethyl-4-(1-phenylethyl)benzene	DNAPL	%	3.4
1-methyl-4 ethyl benzene	Soil Vapour	µg/m3	770
1-Phenylnaphthalene	DNAPL	%	1.4
2,3-Diphenylbutane	DNAPL	%	0.7
2378-TCDD	Sediment	pg/g	0.8
2-chlorotoluene	Groundwater	µg/L	9
2-methylnaphthalene	Soil	mg/kg	35
2-Phenylnaphthalene	DNAPL	%	2.1
4-Methyl-2-pentanone	Groundwater	µg/L	160
Acenaphthylene	Soil	mg/kg	1.2
Acetone	Groundwater	mg/L	180
	Soil Vapour	µg/m3	51
Alkalinity (Bicarbonate as CaCO3)	Groundwater	mg/L	569
Alkalinity (Carbonate as CaCO3)	Groundwater	mg/L	1,500
Alkalinity (Hydroxide) as CaCO3	Groundwater	µg/L	3,630,000
Alkalinity (total) as CaCO3	Groundwater	mg/L	3,900
Aluminium	Soil	mg/kg	286,000
a-Methylstyrene	DNAPL	%	0.4
Ammonia as N	Groundwater	µg/L	10,800
Anions Total	Groundwater	meq/L	2,060
Anthracene	DNAPL	%	0.1
	Soil	mg/kg	2
Antimony	Groundwater	mg/L	0.004
	Sediment	mg/kg	0.2
	Soil	mg/kg	1
Arsenic	Groundwater	mg/L	0.164
	Sediment	mg/kg	6.9
	Soil	mg/kg	28
Barium	Groundwater	mg/L	0.344
	Sediment	mg/kg	26.1
	Soil	mg/kg	8
Benz(a)anthracene	Soil	mg/kg	4.7
Benzene	DNAPL	%	0.7
	Groundwater	µg/L	89,700
	Soil	mg/kg	34,100
	Soil Vapour	µg/m3	34,700
Benzo(a) pyrene	Soil	mg/kg	4.7
Benzo(b)&(k)fluoranthene	Soil	mg/kg	3.1
Benzo(b)fluoranthene	Soil	mg/kg	3.7
Benzo(g,h,i)perylene	Soil	mg/kg	2.6
Benzo(k)fluoranthene	Soil	mg/kg	3.8
Beryllium	Groundwater	mg/L	0.001
	Sediment	mg/kg	0.2
Bicarbonate	Groundwater	mg/L	693
Bicarbonate as CaCO3	Groundwater	mg/L	790
Bis(2-chloroethyl)ether	Soil	mg/kg	0.5
Bromobenzene	Groundwater	µg/L	1
Bromodichloromethane	Groundwater	µg/L	9
Bromoform	Groundwater	µg/L	1
Bromomethane	Groundwater	µg/L	70
Butylated hydroxy toluene	DNAPL	%	0.6
Cadmium	Groundwater	mg/L	0.0016
	Sediment	mg/kg	0.2
	Soil	mg/kg	23
Calcium	Groundwater	mg/L	2,010
	Soil	mg/kg	79
Carbon disulfide	Groundwater	µg/L	84
	Soil Vapour	µg/m3	22
Carbon tetrachloride	Groundwater	µg/L	2,660
	Soil	mg/kg	69.5
	Soil Vapour	µg/m3	430
Carbonate	Groundwater	mg/L	117
Cations Total	Groundwater	meq/L	1,860

Chemical Name	Media	Units	Maximum Detected Concentration
Chloride	Groundwater	mg/L	66,500
	Sediment	mg/kg	7560
	Soil	mg/kg	3,500
Chlorobenzene	DNAPL	%	0.3
	Groundwater	µg/L	9,510
	Soil	mg/kg	17,400
	Soil Vapour	µg/m3	9,140
Chloroethane	Groundwater	µg/L	2,870
	Soil Vapour	µg/m3	170
Chloroform	DNAPL	%	0.4
	Groundwater	µg/L	56,500
	Soil	mg/kg	52,700
	Soil Vapour	µg/m3	159,000
Chloromethane	Groundwater	µg/L	20
Chromium (III+VI)	Groundwater	mg/L	0.024
	Sediment	mg/kg	8.7
	Soil	mg/kg	34
Chrysene	Soil	mg/kg	4.2
cis-1,2-Dichloroethene	DNAPL	%	0.3
	Groundwater	µg/L	163,000
	Soil	mg/kg	34,700
	Soil Vapour	µg/m3	251,000
cis-1,4-Dichloro-2-butene	Groundwater	µg/L	24
Cobalt	Sediment	mg/kg	2.5
	Groundwater	mg/L	0.0055
Copper	Groundwater	mg/L	0.028
	Sediment	mg/kg	3.9
	Soil	mg/kg	15,000
Cyanide Total	Soil	mg/kg	2
Dibenz(a,h)anthracene	Soil	mg/kg	1.7
Dichloromethane	Groundwater	µg/L	14,500
	Soil Vapour	µg/m3	210
Diphenylmethane	DNAPL	%	0.5
Diphenylpentane	DNAPL	%	1
Diphenylthiophenes	DNAPL	%	3.1
Dissolved Organic Carbon	Groundwater	mg/L	1,740
Ethanol	Soil Vapour	µg/m3	27
Ethene	Groundwater	µg/L	27,600
Ethyl acetate	Soil	mg/kg	2.2
Ethylbenzene	DNAPL	%	0.4
	Groundwater	µg/L	12,200
	Soil	mg/kg	18,400
	Soil Vapour	µg/m3	7,940
Ferrous Iron	Groundwater	mg/L	42
Fluoranthene	Soil	mg/kg	9.8
Fluorene	Soil	mg/kg	9.4
Fluoride	Groundwater	mg/L	0.9
	Soil	mg/kg	290
Hepta-Dioxins	Groundwater	pg/L	48.1
	Sediment	pg/g	51.1
Hepta-Furans	Sediment	pg/g	11
Heptane	Soil Vapour	µg/m3	6,180
Hexachloroethane	DNAPL	%	0.1
	Groundwater	µg/L	260,000
Hexaethylbenzene	DNAPL	%	0.7
Hexane	Soil Vapour	µg/m3	4,370
Indeno(1,2,3-c,d)pyrene	Soil	mg/kg	2.3
Iodomethane	Groundwater	µg/L	1
Iron	Groundwater	mg/L	66
	Soil	mg/kg	990
Isopropylbenzene	Groundwater	µg/L	78
	Soil	mg/kg	8.5
Kjeldahl Nitrogen Total	Groundwater	mg/L	11
Lead	Groundwater	mg/L	0.011
	Sediment	mg/kg	5.8
	Soil	mg/kg	881

Chemical Name	Media	Units	Maximum Detected Concentration
Magnesium	Soil	mg/kg	1020
	Groundwater	mg/L	5,370
Manganese	Sediment	mg/kg	261
	Soil	mg/kg	1,930
	Groundwater	mg/L	9.25
Mercury	Groundwater	mg/L	0.001
	Soil	mg/kg	0.3
Meta- & Para- Xylene	Groundwater	mg/L	1.99
Methane	Groundwater	mg/L	2.63
Naphthalene	DNAPL	%	0.3
	Groundwater	µg/L	198
	Soil	mg/kg	48
	Soil Vapour	µg/m3	76
n-butylbenzene	Soil	mg/kg	30
Nickel	Groundwater	mg/L	0.092
	Sediment	mg/kg	5.2
	Soil	mg/kg	102
Nitrate (as N)	Groundwater	mg/L	25.2
Nitrate (as NO3-)	Groundwater	mg/L	9.37
Nitrite (as N)	Groundwater	mg/L	59
Nitrite (as NO2-)	Groundwater	mg/L	0.019
Nitrite + Nitrate as N	Groundwater	mg/L	0.374
Nitrogen (Total Oxidised)	Groundwater	mg/L	25.3
n-propylbenzene	Groundwater	µg/L	36
	Soil	mg/kg	8.7
OCDD	Groundwater	pg/L	96.2
	Sediment	pg/g	488
OCDD I-TEF	Groundwater	pg/L	0.001
OCDD I-TEQ3 (LOR)	Groundwater	pg/L	1.19
OCDD WHO-TEF	Groundwater	pg/L	0.0003
OCDD WHO-TEQ3 (LOR)	Groundwater	pg/L	0.36
OCDF	Sediment	pg/g	25.2
Octa-Dioxin	Groundwater	pg/L	96.2
	Sediment	pg/g	488
Octa-Furan	Sediment	pg/g	25.2
PCBs (Sum of total)	Soil	mg/kg	0.4
Pentachloroethane	Groundwater	µg/L	1,360
	Soil	mg/kg	90.5
pH	Groundwater	pH_Units	12.9
	Sediment	pH_Units	8.3
Phenanthrene	DNAPL	%	0.9
	Soil	mg/kg	26.8
Phenyl ether	DNAPL	%	0.4
Phosphate total (P)	Groundwater	mg/L	2.6
Podocephalol	DNAPL	%	1.6
Potassium	Groundwater	mg/L	1,260
	Soil	mg/kg	27
Propenylbenzene	DNAPL	%	0.2
Pyrene	Soil	mg/kg	8.9
Reactive Phosphorus as P	Groundwater	mg/L	1.51
Redox Potential	Sediment	mV	133
sec-butylbenzene	Groundwater	µg/L	38
	Soil	mg/kg	16
Selenium	Groundwater	mg/L	0.02
Silver	Groundwater	mg/L	0.0086
	Soil	mg/kg	0.4
Sodium	Groundwater	mg/L	26,200
	Soil	mg/kg	1,330
Stilbene	DNAPL	%	2.2
Styrene	DNAPL	%	1.1
	Groundwater	µg/L	2,340
	Soil	mg/kg	257

Chemical Name	Media	Units	Maximum Detected Concentration
Sulfate as SO4 - Turbidimetric	Groundwater	mg/L	2,620
Sulphate	Groundwater	mg/L	28,900
	Soil	mg/kg	30
Sulphate as S	Groundwater	mg/L	990
Sulphide	Groundwater	mg/L	7
Sulphur as S	Soil	mg/kg	1,400
Tetrachloroethylene	DNAPL	%	3.3
	Groundwater	µg/L	20,000
	Soil	mg/kg	148,000
	Soil Vapour	µg/m3	429,000
Tetradecane	DNAPL	%	0.3
Tetra-Dioxins	Sediment	pg/g	3.7
Tetraethylbenzene	DNAPL	%	0.4
Tetra-Furans	Groundwater	pg/L	9.5
	Sediment	pg/g	3.5
Tin	Sediment	mg/kg	0.4
	Soil	mg/kg	30
	Groundwater	mg/L	0.002
Toluene	Groundwater	µg/L	2,940
	Soil	mg/kg	23
	Soil Vapour	µg/m3	18,900
TPH >C10 - C12 (Aliphatic)	Soil Vapour	µg/m3	37,800
TPH >C6 - C8 (Aliphatic)	Soil Vapour	µg/m3	157,000
TPH >C8 - C10 (Aliphatic)	Soil Vapour	µg/m3	46,900
TPH >C8 - C10 (Aromatic)	Soil Vapour	µg/m3	17,200
TPH C10 - C14	Groundwater	µg/L	27,000
	Soil	mg/kg	920
TPH C10 - C16	Soil	mg/kg	150
TPH C10 - C36 (Sum of total)	Groundwater	µg/L	62,100
	Soil	mg/kg	6,270
TPH C10 - C40 (Sum of total)	Soil	mg/kg	7,130
TPH C15 - C28	Groundwater	µg/L	99,500
	Soil	mg/kg	4,720
TPH C15 - C36 (Sum of total)	Soil	mg/kg	5,880
TPH C16 - C34	Soil	mg/kg	4,640
TPH C29 - C36	Groundwater	µg/L	41,200
	Soil	mg/kg	2,200
TPH C34 - C40	Soil	mg/kg	310
TPH C6 - C10	Soil	mg/kg	190
TPH C6 - C8 (Aromatic)	Soil Vapour	µg/m3	59,800
TPH C6 - C9	Groundwater	µg/L	963,000
	Soil	mg/kg	1,650
TPH C6 - C9 ALIPHATIC	Soil	mg/kg	340
trans-1,2-Dichloroethene	DNAPL	%	0.3
	Groundwater	µg/L	88,400
	Soil	mg/kg	29,200
	Soil Vapour	µg/m3	189,000
trans-1,3-dichloropropene	Groundwater	µg/L	12
trans-1,4-Dichloro-2-butene	Groundwater	µg/L	56
	Soil	mg/kg	4.9
Trichloroethylene	DNAPL	%	2.3
	Groundwater	µg/L	92,500
	Soil	mg/kg	135,000
	Soil Vapour	µg/m3	888,000
Triethylbenzenes	DNAPL	%	0.6
Trihalomethanes	Groundwater	mg/L	11.2

Chemical Name	Media	Units	Maximum Detected Concentration
Vanadium	Sediment	mg/kg	9.3
	Groundwater	mg/L	0.01
Vinyl chloride	Groundwater	µg/L	226,000
	Soil Vapour	µg/m3	34,700
Xylene (m & p)	Groundwater	µg/L	3,060
	Soil	mg/kg	23
	Soil Vapour	µg/m3	3,910
Xylene (o)	Groundwater	µg/L	750
	Soil	mg/kg	6.7
	Soil Vapour	µg/m3	1,740
Xylene Total	Groundwater	µg/L	280
	Soil	mg/kg	29.7
Zinc	Groundwater	mg/L	0.084
	Sediment	mg/kg	18.5
	Soil	mg/kg	397

Appendix B: Lower concern contaminants – not considered to be the cause of long-term health problems

Chemical Name	Comments
1,1,1-trichloroethane	ATSDR: A synthetic volatile organic compound (VOC) with acute irritant properties, but not known or classified as causing long-term health problems.
1,1,2-Trichloroethane	ATSDR: A synthetic volatile organic compound (VOC) with acute irritant properties, but not known or classified as causing long-term health problems.
1,1'-Biphenyl	PubChem: Whilst listed on the Australian National Pollutant Inventory, this chemical is also used as a food preservative and is not known or classified as causing long-term health problems.
1,1-bis-p-Tolylethane	PubChem: A chemical not known or classified as causing long-term health problems.
1,1-Diphenylethane	PubChem: A chemical not known or classified as causing long-term health problems.
1,2,4-trimethylbenzene	PubChem: A flammable aromatic hydrocarbon occurring naturally in coal tar and petroleum with acute irritant properties, but not known or classified as causing long-term health problems.
1,2-Dichloroethene	PubChem: A highly flammable liquid with pungent odour used in manufacture of solvents and chemicals. It has acute irritant properties, but not known or classified as causing long-term health problems.
1,2-Diethylbenzene	PubChem: A toxic and flammable liquid used in manufacture of solvents and chemicals. It has acute irritant properties, but not known or classified as causing long-term health problems.
1,2-Dihydrophenanthrene	PubChem: One of the phenanthroids which are chemical compounds naturally occurring in plants but can be synthesized. May be markers of smoking, or exposure to polycyclic aromatic hydrocarbons (PAH).
1,2-Diphenylcyclobutane	PubChem: Found as an impurity in polystyrene food containers and is not known or classified as causing long-term health problems.
1,2-Diphenylethane	PubChem: This chemical is a derivative of ethane and is the core of some natural products. When synthetised, it is a crystalline powder with irritant properties, but is not known or classified as causing long-term health problems.
1,2-Diphenylpropane	PubChem: A chemical used in research and development only and is not known or classified as causing long-term health problems.

Chemical Name	Comments
1,3-dichloropropane	PubChem: A colourless liquid used as an intermediate in chemical manufacture. It has irritant properties but is not known or classified as causing long-term health problems.
1,4-dichloro-2-butene	PubChem: An irritant and corrosive liquid chemical used as an intermediate, but not known or classified as causing long-term health problems.
1,4-Diethylbenzene	PubChem: Flammable liquid used in manufacture of solvents and as chemical intermediate. It has irritant properties, but is not known or classified as causing long-term health problems.
1,4-Dihydroxy-9,10-anthracenedione	Toxnet: A chemical irritant, but not known or classified as causing long-term health problems.
1,4-Diphenylbutadiene	PubChem: A chemical irritant, but not known or classified as causing long-term health problems.
1,4-diphenylbutane	PubChem: A chemical irritant, but not known or classified as causing long-term health problems.
1-Ethyl-4-(1-phenylethyl) benzene	PubChem: A chemical not known or classified as causing long-term health problems.
1-methyl-4 ethyl benzene	PubChem: A chemical not known or classified as causing long-term health problems.
1-Phenylnaphthalene	PubChem: A chemical not known or classified as causing long-term health problems.
2,3-Diphenylbutane	PubChem: A chemical not known or classified as causing long-term health problems.
2-chlorotoluene	PubChem: A flammable liquid with irritant properties, but not known or classified as causing long-term health problems.
2-methylnaphthalene	PubChem: A crystalline powder with irritant properties, but not known or classified as causing long-term health problems.
2-Phenylnaphthalene	PubChem: A chemical not known or classified as causing long-term health problems.
Acetone	PubChem: A solvent with irritant properties, but not known or classified as causing long-term health problems.
Alkalinity (Bicarbonate as CaCO3)	Property of test material, but not toxic per se.
Alkalinity (Carbonate as CaCO3)	Property of test material, but not toxic per se.
Alkalinity (Hydroxide) as CaCO3	Property of test material, but not toxic per se.

Chemical Name	Comments
Alkalinity (total) as CaCO3	Property of test material, but not toxic per se.
Aluminium	Multiple forms of Aluminium (Al) occur naturally in soil.
Ammonia as N	Ammoniacal nitrogen is a measure of the amount of nitrogen found in landfill leachate, but not toxic per se.
Anions Total	Anion analysis is part of standard environmental sampling for inorganic compounds, and as a generic term does not indicate toxicity per se.
Bicarbonate	Property of test material and not toxic per se.
Bicarbonate as CaCO3	Property of test material and not toxic per se.
Bromobenzene	PubChem: A colourless liquid with irritant properties, but not known or classified as causing long-term health problems.
Butylated hydroxy toluene	PubChem: A high volume industrial chemical not known or classified as causing long-term health problems.
Calcium	A naturally occurring element found routinely in environmental analyses and not toxic per se.
Carbonate	A generic term indicating the presence of salts or esters of carbonic acid, but not toxic per se.
Cations Total	Cation analysis is part of standard environmental sampling for inorganic compounds, and as a generic term does not indicate toxicity per se.
Chloride	Chlorides are widely distributed in nature as salts of sodium, potassium, and calcium. Chloride analysis is part of standard environmental sampling, and as a generic term does not indicate toxicity per se.
Chlorobenzene	ATSDR: A colourless flammable liquid with irritant properties, but not known or classified as causing long-term health problems.
Copper	A naturally occurring essential element not classified as causing long-term health problems.
Cyanide Total	Trace amount only detected of this naturally occurring element.
Diphenylmethane	PubChem: A chemical compound with irritant properties, but not known or classified as causing long-term health problems.
Diphenylpentane	PubChem: A chemical compound with irritant properties, but not known or classified as causing long-term health problems.
Diphenylthiophenes	PubChem: Chemical compounds with irritant properties, but not known or classified as causing long-term health problems.
Dissolved Organic Carbon	A generic term and not toxic per se.

Chemical Name	Comments
Ethene (ethylene)	PubChem: A chemical compound with irritant properties, but not known or classified as causing long-term health problems.
Ethyl acetate	PubChem: A chemical compound with irritant properties, but not known or classified as causing long-term health problems.
Ferrous Iron	An abundant naturally occurring element and not toxic per se.
Heptane	PubChem: A chemical found in cardamom with irritant properties, but not known or classified as causing long-term health problems.
Hexaethylbenzene	PubChem: A chemical compound with irritant properties, but not known or classified as causing long-term health problems.
Iodomethane (methyl iodide)	PubChem: An intermediate in chemical manufacture but not known or classified as causing long-term health problems.
Iron	An abundant naturally occurring element and not toxic per se.
Kjeldahl Nitrogen Total	A generic term and not toxic per se.
Methane	PubChem: A colourless, odourless, and acutely toxic gas, but not known or classified as causing long-term health problems.
n-butylbenzene	PubChem: A chemical, used as a solvent and intermediate in plastics, with irritant properties but not known or classified as causing long-term health problems.
Nitrate (as N)	A generic term and not toxic per se.
Nitrate (as NO3-)	A generic term for inorganic or organic salts or esters of nitric acid, but not toxic per se in environmental context.
Nitrite (as N)	A generic term and not toxic per se.
Nitrite (as NO2-)	A generic term for salts or esters of nitrous acid, but not toxic per se in environmental context.
Nitrite + Nitrate as N	A generic term and not toxic per se.
Nitrogen (Total Oxidised)	A generic term and not toxic per se. Nitrogen (N) occurs in many organic and inorganic forms in the environment, with air being 78% N. Nitrogen in soil, not air, is used by plants.
n-propylbenzene	PubChem: A clear colourless liquid used as a chemical intermediate. It has irritant properties but not known or classified as causing long-term health problems.

Chemical Name	Comments
pH	Measurement of acidity/alkalinity in samples.
Phenyl ether (diphenyl ether)	PubChem: A colourless liquid or in crystalline form and used in flavourings and fragrances. It has irritant properties but not known or classified as causing long-term health problems.
Phosphate total (P)	A generic term and not toxic per se. Phosphates in groundwater arise from many natural and waste sources.
Podocephalol	PubChem: A naturally occurring chemical used as drug intermediate. It is not known or classified as causing long-term health problems.
Potassium	An essential element in human cells and not toxic per se.
Propenylbenzene	PubChem: A chemical not known or classified as causing long-term health problems.
Reactive Phosphorus as P	An essential element for human and plant growth and one of the most common substances found in nature. Not toxic per se.
Redox Potential	A common measurement of water quality and not toxic per se.
sec-butylbenzene	PubChem: A colourless liquid used as solvent and in plastics manufacture. It has irritant properties but not known or classified as causing long-term health problems.
Selenium	An essential non-metallic chemical element and only toxic in high concentrations.
Silver	Trace amount found and not toxic per se.
Sodium	An essential element and not toxic per se.
Stilbene	A chemical not known or classified as causing long-term health problems.
Sulfate as SO4 - Turbidimetric	A salt or ester of sulphuric acid and not toxic per se.
Sulphate	A salt or ester of sulphuric acid and not toxic per se.
Sulphate as S	A salt or ester of sulphuric acid and not toxic per se.
Sulphide	A generic term for compounds of sulphur but not toxic per se.
Sulphur as S	Naturally occurring non-metal element and not toxic per se.
Tetradecane	PubChem: A colourless liquid not known or classified as causing long-term health problems.
Tetraethylbenzene	PubChem: A chemical not known or classified as

Chemical Name	Comments
	causing long-term health problems.
Tin	A naturally occurring metal not known or classified as causing long-term health problems.
Trans-1,2-dichloroethene	See 1,2-dichloroethene above.
Trans-1,4-dichloro-2-butene	See 1,4-dichloro-2-butene above.
Triethylbenzenes	PubChem: Chemicals with irritant properties but not known or classified as causing long-term health problems.
Zinc	An essential element not known or classified as causing long-term health problems.

Appendix C: Higher concern contaminants – considered to have the potential to cause long-term health problems including cancer

Chemical Name	Comments
1,1,2,2-Tetrachloroethane **acute and chronic CNS irritant** **irritant to eyes and URT** **toxic to liver**	ATSDR: 1,1,2,2-Tetrachloroethane is a manufactured, colourless, dense liquid that does not burn easily. It is volatile and has a sweet odour. In the past, it was used in large amounts to produce other chemicals, as an industrial solvent to clean and degrease metals, and as an ingredient in paints and pesticides. Commercial production of 1,1,2,2-tetrachloroethane for these uses has stopped. It presently is used only as a chemical intermediate in the production of other chemicals. Breathing very high concentrations of 1,1,2,2 -tetrachloroethane can rapidly cause drowsiness, dizziness, nausea, and vomiting. Most people recover from these effects once they are in fresh air. Breathing high levels of 1,1,2,2-tetrachloroethane for a long time can cause liver damage. It is not known whether 1,1,2,2-tetrachloroethane causes cancer in humans. In a long-term study, 1,1,2,2-tetrachloroethane caused an increase in liver tumors in mice, but not in rats. EPA has determined that it is a possible human carcinogen. OSHA: 1,1,2,2-tetrachloroethane is not listed as carcinogenic. IARC: 1,1,2,2-tetrachloroethane cannot be classified as to its ability to cause cancer in humans (Group 3).
1,1-Dichloroethane **toxic to CNS, heart and kidneys**	ATSDR: Exposure to 1,1-dichloroethane occurs mainly from eating contaminated food, but may also occur from skin contact, breathing contaminated air, or drinking contaminated water. 1,1-Dichloroethane affects the function of the nervous system. 1,1-Dichloroethane has been found in at least 673 of the 1,699 National Priorities List sites identified by the Environmental Protection Agency (EPA). High levels of 1,1-dichloroethane that cause anesthesia can cause irregular heartbeats, which is why its use as a surgical anesthetic was discontinued. Kidney effects have been observed in cats exposed to 1,1 dichloroethane in air for long periods. However, kidney effects have not been observed in other animal species following long-term inhalation or oral exposure. A study in rats and mice found suggestive evidence that 1,1-dichloroethane may cause cancer. However, the study had several flaws and the results are not conclusive. Another long-term study in mice drinking water containing 1,1-dichloroethane did not find cancer. The US Department of Health and Human Services (DHHS has not evaluated the carcinogenic potential of 1,1-dichloroethane. The EPA has determined that 1,1-dichloroethane is a possible human carcinogen.

Chemical Name	Comments
	OSHA: 1, 1-dichloroethane is listed as a carcinogen. IARC: Not classified as a carcinogen.
1,1-Dichloroethene **toxic to CNS, lungs, liver**	ATSDR: Exposure to 1,1-dichloroethene occurs mainly in the workplace. Breathing high levels of 1,1-dichloroethene can affect the liver, kidney, and central nervous system. This chemical has been found in at least 515 of 1,416 National Priorities List sites identified by the Environmental Protection Agency. The main effect from breathing high levels of 1,1-dichloroethene is on the central nervous system. Some people lost their breath and fainted after breathing high levels of the chemical. Breathing lower levels of 1,1-dichloroethene in air for a long time may damage the nervous system, liver, and lungs. Workers exposed to 1,1-dichloroethene have reported a loss in liver function, but other chemicals were present. Animals that breathed high levels of 1,1-dichloroethene had damaged livers, kidneys, and lungs. The offspring of some of the animals had a higher number of birth defects. It is not known if birth defects occur when people are exposed to 1,1-dichloroethene. Animals that ingested high levels of 1,1-dichloroethene had damaged livers, kidneys, and lungs. There were no birth defects in animals that ingested the chemical. Spilling 1,1-dichloroethene on the skin or in the eyes can cause irritation. The Environmental Protection Agency (EPA) has determined that 1,1-dichloroethene is a possible human carcinogen. Animal studies have shown mixed results. Several studies reported an increase in tumors in rats and mice, and other studies reported no such effects. OSHA: Not listed as a carcinogen. IARC: Group 3, not classifiable as a human carcinogen.
1,2,4-trichlorobenzene **irritant to CNS, skin, URT** **toxic to liver and kidneys**	ATSDR: Trichlorobenzenes have been used as solvents. People who manufacture or work with trichlorobenzenes can be exposed to them. It is unlikely that the public will be exposed to high amounts of trichlorobenzenes. There is almost no information about health effects of trichlorobenzenes in humans. 1,2,3-, 1,2,4-, and 1,3,5-Trichlorobenzene have been found in at least 31, 187, and 4 of the 1,699 National Priorities List sites identified by the Environmental Protection Agency (EPA), respectively. There is virtually no information regarding health effects of trichlorobenzenes in humans. However, based on results from studies in animals, it is reasonable to predict that humans exposed to high amounts of trichlorobenzenes may develop liver problems.

Chemical Name	Comments
	Studies in animals indicate that oral administration of trichlorobenzenes for short or long periods produces mainly alterations in the liver and kidneys. Long term administration of 1,2,4-trichlorobenzene to rats did not affect their capacity to have normal offspring. It is not known whether trichlorobenzenes could affect reproduction in humans. There are no studies of cancer in people exposed to trichlorobenzenes. Mice given 1,2,4-trichlorobenzene in the food for 2 years developed cancer of the liver. The EPA has stated that 1,2,4-trichlorobenzene is not classifiable as to human carcinogenicity. However, this was based on studies conducted prior to 1990; newer information has not been evaluated. NICNAS: High levels of trichlorobenzene may damage the liver, kidney, and thyroid. Trichlorobenzene is absorbed via oral, inhalation, and dermal routes. It is believed to be metabolized via cytochrome p-450 enzymes into metabolites that include phenols, mercapturic acid, and catechols. OSHA: Not listed as carcinogenic. IARC: Not classifiable.
1,2-dichlorobenzene **irritant to CNS, skin, URT** **liver cancer in experimental animals**	ATSDR: There are three dichlorobenzene isomers- 1,2-dichlorobenzene, 1,3-dichlorobenzene, and 1,4-dichlorobenzene. Dichlorobenzenes do not occur naturally. 1,2-Dichlorobenzene is a colorless to pale yellow liquid used to make herbicides. 1,3-Dichlorobenzene is a colorless liquid used to make herbicides, insecticides, medicine, and dyes. 1,4-Dichlorobenzene, the most important of the three chemicals, is a colorless to white solid with a strong, pungent odor. When exposed to air, it slowly changes from a solid to a vapor. Most people can smell 1,4-dichlorobenzene in the air at very low levels. Exposure to dichlorobenzenes mostly occurs from breathing indoor air or workplace air. Exposure to high levels of 1,2- or 1,4-dichlorobenzene may be very irritating to the eyes and nose and cause difficult breathing, and an upset stomach. Extremely high exposures to 1,4- dichlorobenzene can result in dizziness, headaches, and liver problems. 1,2-, 1,3-, and 1,4-Dichlorobenzenes have been identified in at least 281, 175, and 330, respectively, of the 1,662 National Priorities List sites identified by the Environmental Protection Agency (EPA). The US Department of Health and Human Services (DHHS) has determined that 1,4-dichlorobenzene may reasonably be anticipated to be a carcinogen. There is no direct evidence that 1,4-dichlorobenzene can cause cancer in humans. However, animals given very high levels in water developed liver tumors. 1,2-Dichlorobenzene was not carcinogenic in laboratory animals and 1,3-dichlorobenzene has not been tested for its potential to cause cancer. The EPA concluded that 1,2- and 1,3-dichlorobenzene are not classifiable as to human carcinogenicity.

Chemical Name	Comments
	NICNAS: Assessed as a Priority Existing Chemical (PEC) OSHA: Not listed as carcinogenic. IARC: There is inadequate evidence in humans for the carcinogenicity of dichlorobenzenes. There is evidence suggesting lack of carcinogenicity in experimental animals of ortho-dichlorobenzene. There is inadequate evidence in experimental animals for the carcinogenicity of meta-dichlorobenzene. There is sufficient evidence in experimental animals for the carcinogenicity of para-dichlorobenzene. ortho-Dichlorobenzene is not classifiable as to its carcinogenicity to humans (Group 3). meta-Dichlorobenzene is not classifiable as to its carcinogenicity to humans (Group 3). para-Dichlorobenzene is possibly carcinogenic to humans (Group 2B).
1,2-Dichloroethane **toxic to CNS, liver, kidneys** **cancer at multiple sites in experimental animals**	ATSDR: 1,2-Dichloroethane, also called ethylene dichloride, is a manufactured chemical that is not found naturally in the environment. It is a clear liquid and has a pleasant smell and sweet taste. The most common use of 1,2-dichloroethane is in the production of vinyl chloride which is used to make a variety of plastic and vinyl products including polyvinyl chloride (PVC) pipes, furniture and automobile upholstery, wall coverings, housewares, and automobile parts. It is also used as a solvent and is added to leaded gasoline to remove lead. Nervous system disorders, liver and kidney diseases, and lung effects have been reported in humans ingesting or inhaling large amounts of 1,2-dichloroethane. In laboratory animals, breathing or ingesting large amounts of 1,2-dichloroethane have also caused nervous system disorders and liver, kidney, and lung effects. Animal studies also suggest that 1,2-dichloroethane may damage the immune system. Kidney disease has also been seen in animals ingesting low doses of 1,2-dichloroethane for a long time. Studies in animals indicate that 1,2-dichloroethane does not affect reproduction. Human studies examining whether 1,2-dichloroethane can cause cancer have been considered inadequate. In animals, increases in the occurrence of stomach, mammary gland, liver, lung, and endometrium cancers have been seen following: inhalation, oral, and dermal exposure. The Department of Health and Human Services (DHHS) has determined that 1,2-dichloroethane may reasonably be expected to cause cancer. The EPA has determined that 1,2-dichloroethane is a probable human carcinogen. NICNAS: Australian use information for this chemical is limited. Based on the available exposure data, the majority of industrial

Chemical Name	Comments
	environmental emissions in Australia are expected to result from use as an industrial solvent in closed-system manufacturing plants or small scale uses in specialty fuels. Any emissions resulting from these uses are expected to partition to the air compartment. Based on concentrations of the chemical in air determined internationally, industrial use of the chemical is not expected to pose an unreasonable risk to the environment. OSHA: Not listed as carcinogenic. IARC: There is *inadequate evidence* in humans for the carcinogenicity of 1,2-dichloroethane. There is *sufficient evidence* in experimental animals for the carcinogenicity of 1,2-dichloroethane. 1,2-Dichloroethane is *possibly carcinogenic to humans (Group 2B)*.
1,2-dichloropropane **toxic to lungs, CNS, liver, kidneys** **causes liver cancer** **Group 1 carcinogen**	ATSDR: 1,2-Dichloropropane is primarily used to make other chlorinated chemicals. Exposure to high levels of 1,2-dichloropropane can damage the liver, kidneys, blood, and lungs, and affect the brain. It has been found at 26 of the 1,177 National Priorities List sites identified by the Environmental Protection Agency (EPA). People who intentionally or accidentally breathe high levels of 1,2-dichloropropane have experienced difficulty breathing, coughing, vomiting, nosebleed, fatigue, and damage to blood cells, liver, and kidneys. People who accidentally drank cleaning solutions containing 1,2-dichloropropane experienced headaches, dizziness, nausea, liver and kidney damage, anemia, coma, and death. Animal studies indicate that breathing low levels of 1,2-dichloropropane over short- or long-term periods causes damage to the liver, kidney, and respiratory system. Breathing high levels causes death. Similar effects have been reported when animals were given 1,2-dichloropropane by mouth. Some studies indicate that ingesting 1,2-dichloropropane may cause reproductive effects. One study reported a delay in bone formation of the skull in fetal rats following exposure of the mother rats to 1,2-dichloropropane. It is not known whether 1,2-dichloropropane causes cancer in people. The carcinogenicity of 1,2-dichloropropane has been evaluated in animal studies with rats and mice. Liver tumors have been observed in mice, and mammary gland tumors have been found in rats. OSHA: Listed as carcinogenic. IARC: There is sufficient evidence in humans for the carcinogenicity of 1,2-dichloropropane. 1,2-Dichloropropane causes cancer of the biliary tract (confirmed as cholangiocarcinoma). There is sufficient evidence in experimental animals for the carcinogenicity of 1,2-dichloropropane.

Chemical Name	Comments
	1,2-Dichloropropane is carcinogenic to humans (Group 1)
1,3,5-trimethylbenzene (TMB) **Irritant to CNS, skin, eyes**	PubChem: Mesitylene or 1, 3, 5-trimethylbenzene is a derivative of benzene with three methyl substituents symmetrically placed on the ring. Isomeric trimethylbenzenes include hemimellitene (1,2,3-trimethylbenzene) and pseudodocumene (1,2,4-trimethylbenzene). All three compounds have the formula C6H3(CH3)3, which is commonly abbreviated C6H3Me3. Mesitylene is a colourless liquid with sweet aromatic odor. It is a component of coal tar, which is its traditional source. It is a major urban volatile organic compound (VOC) resulting from combustion and vehicle emissions are a major source. It is a precursor to diverse fine chemicals. May cause toxic effects if inhaled or absorbed through skin. Inhalation or contact with material may irritate or burn skin and eyes. Fire will produce irritating, corrosive and/or toxic gases. Vapors may cause dizziness or suffocation. Runoff from fire control or dilution water may cause pollution. IARC, EPA, OSHA: Not classifiable as human carcinogen.
1,3-Butadiene **Irritant to URT, eyes and CNS** **causes leukaemia** **Group 1 carcinogen**	ATSDR: 1,3-Butadiene is a chemical made from the processing of petroleum. It is a colorless gas with a mild gasoline-like odor. About 60% of the manufactured 1,3-butadiene is used to make synthetic rubber. Synthetic rubber is widely used for tires on cars and trucks. 1,3-Butadiene is also used to make plastics including acrylics. Small amounts are found in gasoline. Breathing high levels of 1,3-butadiene for a short time may cause nausea, dry mouth and nose, headache, and decreased blood pressure and pulse rate. In laboratory animals, 1,3-butadiene causes inflammation of nasal tissues, changes to lung, heart, and reproductive tissues, neurological effects, and blood changes. The Department of Health and Human Services (DHHS), and EPA have determined that 1,3-butadiene is a human carcinogen. Studies have shown that workers exposed to 1,3-butadiene may have an increased risk of cancers of the stomach, blood, and lymphatic system. Animal studies found increases in a variety of tumor types from exposure to 1,3-butadiene. IPCS: While 1,3-butadiene is not persistent, it is ubiquitous in the urban environment because of its widespread combustion sources. The highest atmospheric concentrations have been measured in air in cities and close to industrial sources. The general population is exposed to 1,3-butadiene primarily through ambient and indoor air. In comparison, other media, including food and drinking-water, contribute negligibly to exposure to 1,3-butadiene. Tobacco smoke may contribute significant amounts of 1,3-buta diene.

Chemical Name	Comments
	An association between exposure to 1,3-butadiene in the occupational environment and leukaemia fulfils several of the traditional criteria for causality. In the largest and most comprehensive study conducted to date, involving a cohort of workers from multiple plants, mortality due to leukaemia increased with estimated cumulative exposure to 1,3-butadiene in the styrene- butadiene rubber industry; this association remained after controlling for exposure to styrene and benzene and was strongest in those subgroups with highest potential exposure. Similarly, an association between exposure to 1,3-butadiene and leukaemia was observed in an independently conducted case–control study of largely the same population of workers. However, there was no increase in mortality due to leukaemia in butadiene monomer production workers who were not concomitantly exposed to some of the other substances present in the styrene-butadiene rubber industry, although there was some limited evidence of an association with mortality due to lymphosarcoma and reticulosarcoma in some subgroups. The available epidemiological and toxicological data provide evidence that 1,3-butadiene is carcinogenic in humans and may also be genotoxic in humans. The carcinogenic potency (the concentration associated with a 1% increase in mortality due to leukaemia) was determined to be 1.7 mg/m^3, based on the results of the largest well conducted epidemiological investigation in exposed workers. This value is like the lower end of the range of tumorigenic concentrations determined on the basis of studies in rodents. 1,3-Butadiene also induced reproductive toxicity in experimental animals. As a measure of its potency to induce reproductive effects, a benchmark concentration of 0.57 mg/m^3 was derived for ovarian toxicity in mice. OSHA: Listed as carcinogenic. IARC: There is sufficient evidence in humans for the carcinogenicity of 1,3-butadiene. 1,3-Butadiene causes cancer of the haemato-lymphatic organs. There is sufficient evidence for the carcinogenicity of 1,3-butadiene in experimental animals. There is sufficient evidence for the carcinogenicity of diepoxy-butane in experimental animals. There is strong evidence that the carcinogenicity of 1,3-butadiene in humans operates by a genotoxic mechanism that involves formation of reactive epoxides, interaction of these direct-acting mutagenic epoxides with DNA, and resultant mutagenicity. The metabolic pathways for 1,3-butadiene in experimental animals have also been demonstrated in humans. 1,3-Butadiene is carcinogenic to humans (Group 1).
1,3-dichlorobenzene	See 1,2-dichlorobenzene above.
1,4-dichlorobenzene	See 1,2 -dichlorobenzene above.
1234678-HpCDD and all	ATSDR: Exposure to chlorinated dibenzo-p-dioxins (CDDs) (75

Chemical Name	Comments
dioxins identified. **toxic to liver and causes birth defects** **causes skin damage and chloracne** **causes cancer of lung, soft tissue sarcoma, non-Hodgkin's lymphoma**	chemicals) occurs mainly from eating food that contains the chemicals. One chemical in this group, 2,3,7,8-tetrachlorodibenzo-p-dioxin or 2,3,7,8-TCDD, has been shown to be very toxic in animal studies. It causes effects on the skin and may cause cancer in people. This chemical has been found in at least 91 of 1,467 National Priorities List sites identified by the Environmental Protection Agency (EPA). CDDs are a family of 75 chemically related compounds commonly known as chlorinated dioxins. One of these compounds is called 2,3,7,8-TCDD. It is one of the most toxic of the CDDs and is the one most studied. In the pure form, CDDs are crystals or colorless solids. CDDs enter the environment as mixtures containing a number of individual components. 2,3,7,8-TCDD is odorless and the odors of the other CDDs are not known. CDDs are not intentionally manufactured by industry except for research purposes. They (mainly 2,3,7,8-TCDD) may be formed during the chlorine bleaching process at pulp and paper mills. CDDs are also formed during chlorination by waste and drinking water treatment plants. They can occur as contaminants in the manufacture of certain organic chemicals. CDDs are released into the air in emissions from municipal solid waste and industrial incinerators. The most noted health effect in people exposed to large amounts of 2,3,7,8-TCDD is chloracne. Chloracne is a severe skin disease with acne-like lesions that occur mainly on the face and upper body. Other skin effects noted in people exposed to high doses of 2,3,7,8-TCDD include skin rashes, discoloration, and excessive body hair. Changes in blood and urine that may indicate liver damage also are seen in people. Exposure to high concentrations of CDDs may induce long-term alterations in glucose metabolism and subtle changes in hormonal levels. In certain animal species, 2,3,7,8-TCDD is especially harmful and can cause death after a single exposure. Exposure to lower levels can cause a variety of effects in animals, such as weight loss, liver damage, and disruption of the endocrine system. In many species of animals, 2,3,7,8-TCDD weakens the immune system and causes a decrease in the system's ability to fight bacteria and viruses. In other animal studies, exposure to 2,3,7,8-TCDD has caused reproductive damage and birth defects. Some animal species exposed to CDDs during pregnancy had miscarriages and the offspring of animals exposed to 2,3,7,8-TCDD during pregnancy often had severe birth defects including skeletal deformities, kidney defects, and weakened immune responses. Several studies suggest that exposure to 2,3,7,8-TCDD increases the risk of several types of cancer in people. Animal studies have also shown an increased risk of cancer from exposure to 2,3,7,8-TCDD.

Chemical Name	Comments
	The US Department of Health and Human Services (DHHS) has determined that 2,3,7,8-TCDD may reasonably be anticipated to cause cancer. OSHA: Not listed as carcinogenic. IARC: There is sufficient evidence in humans for the carcinogenicity of 2,3,7,8-tetrachlorodibenzo-para-dioxin. The strongest evidence in humans for the carcinogenicity of 2,3,7,8-tetrachlorodibenzo-para-dioxin is for all cancers combined. Also, a positive association has been observed between exposure to 2,3,7,8-tetrachlorodibenzo-para-dioxin and soft-tissue sarcoma, non-Hodgkin lymphoma and cancer of the lung. There is sufficient evidence in experimental animals for the carcinogenicity of 2,3,7,8-tetrachlorodibenzo-para-dioxin. 2,3,7,8-Tetrachlorodibenzo-para-dioxin is carcinogenic to humans (Group 1). There is sufficient evidence in experimental animals for the carcinogenicity of 2,3,4,7,8-pentachlorodibenzofuran. There is sufficient evidence in experimental animals for the carcinogenicity of 3,3′,4,4′,5-pentachlorobiphenyl. There is strong evidence to support a receptor mediated mechanism that operates in humans for carcinogenesis associated with 2,3,7,8-tetrachlorodibenzo-para-dioxin, where the primary mechanism is the promotion of tumour development through modification of cell replication and apoptosis, with a secondary mechanism related to increases of oxidative stress causing DNA damage. The conservation of the aryl hydrocarbon receptor and the related signaling pathways and responses across species, including humans, add additional strength to the notion that this mechanism is active in humans. 2,3,4,7,8-Pentachlorodibenzofuran is carcinogenic to humans (Group 1). 3,3′,4,4′,5-Pentachlorobiphenyl is carcinogenic to humans (Group 1).
1234678-HpCDD I-TEF	See above for all dioxins identified.
1234678-HpCDD I-TEQ3 (LOR)	See above for all dioxins identified.
1234678-HpCDD WHO-TEF	See above for all dioxins identified.
1234678-HpCDD WHO-TEQ3 (LOR)	See above for all dioxins identified.
1234678-HpCDF and all furans identified.	ATSDR: Dioxins, furans, and polychlorinated biphenyls (PCBs) are a class of similar chlorinated aromatic organic compounds. Dioxins have two phenyl rings connected by two oxygen atoms. Furans have one or two phenyl rings connected to a furan ring. PCBs have two phenyl rings attached at one point. One or more chlorine atoms can attach to any available carbon atom, allowing

Chemical Name	Comments
	for 100 - 200 forms of each. Dioxins and dioxin-like furans have no known commercial or natural use. They are produced primarily during the incineration or burning of waste; the bleaching processes used in pulp and paper mills; and the chemical syntheses of trichlorophenoxyacetic acid, hexachlorophene, vinyl chloride, trichlorophenol, and pentachlorophenol. PCBs were once synthesized for use as heat-exchanger, transformer, and hydraulic fluids, and also used as additives to paints, oils, window caulking, and floor tiles. Production of PCBs peaked in the early 1970s and was banned in the United States after 1979. PubChem: The major hazards encountered in contacting 2,3,7,8-tetrachlorodibenzofuran (TCDF) stem from its toxicologic properties. Having a magnitude of toxicity similar to 2,3,7,8-tetrachloro-p-dioxin. TCDF has been shown to affect biochemical activity, suppress immune function, cause fetal abnormalities, and induce tumors in non-human test organisms. While not used commercially, TCDF is found as an impurity in polychlorinated biphenyl products, and 2,4,6-trichlorophenol. The major route of exposure to the general population results from municipal waste incineration, industrial boilers burning hazardous waste, and exhausts from leaded gasoline engines. Food is a secondary source of exposure (eg, contaminated fish). Occupational exposure occurs through inhalation and dermal contact to fire fighters and cleanup workers associated with polychlorinated biphenyl transformer fires.
123478-HxCDF	See above for all furans identified.
2378-TCDD	PubChem: 2,3,7,8-Tetrachlorodibenzo-p-dioxin which is often referred to simply as dioxin and is the reference for a number of compounds which are similar structurally and have dioxin-like toxicity. A substance extremely toxic to mammals, with a wide variation in sensitivity among species. Longer-term exposure of test mammals to lesser amounts can affect reproduction, cause birth defects, damage the liver and suppress the immune system. Several studies suggest that exposure to TCDD increases the risk of several types of cancer in people. Animal studies have also shown an increased risk of cancer from exposure to TCDD. The IARC and the USA DHHS have determined that TCDD is a human carcinogen. 2,3,7,8-Tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) is formed as an unintentional by-product of incomplete combustion. It may be released to the environment during the combustion of fossil fuels and wood, and during the incineration of municipal and industrial wastes. It causes chloracne in humans, a severe acne-like condition. It is known to be a developmental toxicant in animals, causing skeletal deformities, kidney defects, and weakened immune responses in the offspring of animals exposed to 2,3,7,8-TCDD during pregnancy. Human studies have shown an association between 2,3,7,8-TCDD and soft-tissue sarcomas,

Chemical Name	Comments
	lymphomas, and stomach carcinomas. EPA has classified 2,3,7,8- TCDD as a probable human carcinogen (Group B2). Exposure Routes: inhalation, skin absorption, ingestion, skin and/or eye contact Symptoms: Irritation eyes; allergic dermatitis, chloracne; porphyria; gastrointestinal disturbance; possible reproductive, teratogenic effects Target Organs: Eyes, skin, liver, kidneys, reproductive system. IARC has determined that 2,3,7,8-TCDD is a human carcinogen (Group 1).
4-Methyl-2-pentanone, also known as methyl isobutyl ketone (MIBK) **acute and chronic irritant to CNS, eyes, URT** **Toxic to liver.**	PubChem: Methyl isobutyl ketone (MIBK) is an organic solvent. MIBK is among the top ten most popular organic solvents used in industry. Methyl isobutyl ketone is used as a solvent for gums, resins, paints, varnishes, lacquers, and nitrocellulose. Acute (short-term) exposure to methyl isobutyl ketone may irritate the eyes and mucous membranes, and cause weakness, headache, nausea, lightheadedness, vomiting, dizziness, incoordination, narcosis in humans. Chronic (long-term) occupational exposure to methyl isobutyl ketone has been observed to cause nausea, headache, burning in the eyes, weakness, insomnia, intestinal pain, and slight enlargement of the liver in humans. Lethargy and kidney and liver effects have been observed in rats and mice chronically exposed by gavage (experimentally placing the chemical in the stomach), ingestion, and inhalation. EPA has classified methyl isobutyl ketone as a Group D, not classifiable as to human carcinogenicity. OSHA: MIBK is listed as a carcinogen. IARC: There is sufficient evidence for the carcinogenicity of MIBK in experimental animals – possibly carcinogenic to humans (Group 2B).
Acenaphthylene **toxic to skin, lungs, immune system** **causes cancer of lung, skin, and stomach**	PubChem: Acenaphthylene is a polycyclic hydrocarbon (PAH). It occurs as a crystalline powder. May irritate skin and mucous membranes. Emits acrid smoke and irritating fumes when heated to decomposition. Derived from coal tar and used to make dyes, pharmaceuticals, insecticides, fungicides, and plastics. ATSDR: PAHs are a group of chemicals that are formed during the incomplete burning of coal, oil, gas, wood, garbage, or other organic substances, such as tobacco and charbroiled meat. There are more than 100 different PAHs. PAHs generally occur as complex mixtures (for example, as part of combustion products such as soot), not as single compounds. PAHs occur naturally, but they can be manufactured as individual compounds for research purposes, but not as the mixtures found in combustion products. As pure chemicals, PAHs generally exist as colorless, white, or pale yellow-green solids. They can have a faint, pleasant odor. A few PAHs are used in medicines and to make dyes, plastics, and pesticides.

Chemical Name	Comments
	Others are contained in asphalt used in road construction. They can also be found in substances such as crude oil, coal, coal tar pitch, creosote, and roofing tar. They are found throughout the environment in the air, water, and soil. They can occur in the air, either attached to dust particles or as solids in soil or sediment. Although the health effects of individual PAHs are not exactly alike, the following 17 PAHs are considered as a group: Acenaphthene; acenaphthylene; anthracene; benz[a]anthracene; benzo[a]pyrene; benzo[e]pyrene; benzo[b]fluoranthene; benzo [g,h,i] perylene; benzo[j]fluoranthene; benzo[k]fluoranthene; chrysene; dibenz[a,h]anthracene; fluoranthene; fluorene; indeno [1,2,3-c,d] pyrene; phenanthrene; and pyrene. These 17 PAHs were chosen to be reviewed by ATSDR because (1) more information is available on these than on the others; (2) they are suspected to be more harmful than some of the others, and they exhibit harmful effects that are representative of the PAHs; (3) there is a greater chance that people will be exposed to these PAHs than to the others; and (4) of all the PAHs analyzed, these were the PAHs identified at the highest concentrations at hazardous waste sites. Animal studies have also shown that PAHs can cause harmful effects on the skin, body fluids, and ability to fight disease after both short- and long-term exposure. The US Department of Health and Human Services (DHHS) has determined that some PAHs may reasonably be expected to be carcinogens. Some people who have breathed or touched mixtures of PAHs and other chemicals for long periods of time have developed cancer. Some PAHs have caused cancer in laboratory animals when they breathed air containing them (lung cancer), ingested them in food (stomach cancer), or had them applied to their skin (skin cancer). OSHA: Listed with coal-tar pitch volatiles as carcinogenic. IARC: Exposures to mixtures of PAHs in a wide range of occupations have been classified as carcinogenic to humans (Group 1).
a-methylstyrene (isopropenylbenzene) **acute and chronic irritant to eyes, skin, URT, and CNS**	PubChem: a-methylstyrene (isopropenylbenzene) is a colorless liquid. Insoluble in water and less dense than water. May be mildly toxic by ingestion, inhalation and skin absorption. Vapors may be narcotic by inhalation. Used as a solvent and to make other chemicals. Inhalation causes irritation of respiratory tract, headache, dizziness, light-headedness, and breathlessness. Ingestion causes irritation of mouth and stomach. Contact with liquid irritates eyes. Prolonged skin contact can cause severe rashes,

Chemical Name	Comments
	swelling, and blistering. Repeated or prolonged contact with skin may cause dermatitis. The substance defats the skin, which may cause dryness or cracking. The substance may have effects on the liver and kidneys. This may result in tissue lesions. OSHA: Isopropenylbenzene is listed as carcinogenic. IARC: Evaluated as possibly carcinogenic to humans (Group 2B).
Anthracene	ATSDR: Anthracene is a polycyclic aromatic hydrocarbon (PAH) – see detailed review above for acenaphthylene.
Antimony **chronic irritant to CNS, heart, GIT, skin, and lungs** **causes lung cancer in experimental animals**	ATSDR: Antimony is a silvery-white metal that is found in the earth's crust. Antimony ores are mined and then mixed with other metals to form antimony alloys or combined with oxygen to form antimony oxide. Antimony may be produced as a by-product of smelting lead and other metals. Antimony isn't used alone because it breaks easily, but when mixed into alloys, it is used in lead storage batteries, solder, sheet and pipe metal, bearings, castings, and pewter. Antimony oxide is added to textiles and plastics to prevent them from catching fire. It is also used in paints, ceramics, and fireworks, and as enamels for plastics, metal, and glass. Exposure to antimony at high levels can result in a variety of adverse health effects. Breathing high levels for a long time can irritate the eyes and lungs and can cause heart and lung problems, stomach pain, diarrhea, vomiting, and stomach ulcers. In short-term studies, animals that breathed very high levels of antimony died. Animals that breathed high levels had lung, heart, liver, and kidney damage. In long-term studies, animals that breathed very low levels of antimony had eye irritation, hair loss, lung damage, and heart problems. Problems with fertility were also noted. In animal studies, problems with fertility have been seen when rats breathed very high levels of antimony for a few months. Long-term animal studies have reported liver damage and blood changes when animals ingested antimony. Antimony can irritate the skin if it is left on it. Antimony can have beneficial effects when used for medical reasons. It has been used as a medicine to treat people infected with parasites. Lung cancer has been observed in some studies of rats that breathed high levels of antimony. OSHA: Antimony trioxide as antimony has been listed as carcinogenic.

Chemical Name	Comments
	IARC: There is *sufficient evidence* for the carcinogenicity of antimony trioxide in experimental animals. Antimony trioxide is possibly carcinogenic to humans (Group 2B).
Arsenic **toxic to GIT, and skin** **causes skin and lung cancer**	ATSDR: Arsenic is a naturally occurring element that is found in combination with either inorganic or organic substances to form many different compounds. Inorganic arsenic compounds are found in soils, sediments, and groundwater. These compounds occur either naturally or as a result of mining, ore smelting, and industrial use of arsenic. Organic arsenic compounds are found mainly in fish and shellfish. In the past, inorganic forms of arsenic were used in pesticides and paint pigment. They were also used as wood preservatives and as a treatment for a variety of ailments. Today, usage of arsenic-containing pesticides and wood preservatives is restricted. People are most likely to be exposed to inorganic arsenic through drinking water and to a lesser extent through various foods. Water sources in some areas have higher naturally occurring levels of inorganic arsenic than other areas. Other sources of inorganic arsenic exposure include contact with contaminated soil or with wood preserved with arsenic. People are exposed to organic arsenic by consuming seafood. Unusually large doses of inorganic arsenic can cause symptoms ranging from nausea, vomiting, and diarrhea to dehydration and shock. Long-term exposure to high levels of inorganic arsenic in drinking water has been associated with skin disorders and increased risks for diabetes, high blood pressure, and several types of cancer. Inorganic arsenic and arsenic compounds are considered to be cancer-causing chemicals. Forms of organic arsenic (for example, arsenobetaine) found in seafood are not known to be toxic to humans. Copper and lead ores contain small amounts of arsenic. Arsenic has been found in at least 781 of 1.300 National Priorities List sites identified by the Environmental Protection Agency. OSHA: Arsenic metal and inorganic and organic compounds of arsenic have been listed as carcinogenic. IARC: Arsenic and arsenic compounds are *carcinogenic to humans (Group 1)*.
Barium **toxic to GIT and kidneys**	ATSDR: Barium is a silvery-white metal which exists in nature only in ores containing mixtures of elements. It combines with other chemicals such as sulphur or carbon and oxygen to form barium compounds. Barium compounds are used by the oil and gas industries to make drilling muds. Drilling muds make it easier to drill through rock by keeping the drill bit lubricated. They are also used to make paint, bricks, ceramics, glass, and rubber.

Chemical Name	Comments
	Barium sulphate is sometimes used by doctors to perform medical tests and to take x-rays of the gastrointestinal tract. Exposure to barium occurs mostly in the workplace or from drinking contaminated water. Ingesting drinking water containing levels of barium above drinking water guidelines for relatively short periods of time can cause gastrointestinal disturbances and muscle weakness. Ingesting high levels for a long time can damage the kidneys. Barium and barium compounds have been found in at least 798 of the 1,684 National Priority List sites identified by the Environmental Protection Agency (EPA). Barium in air can result from burning coal and oil. The health effects of the different barium compounds depend on how well the compound dissolves in water or in the stomach contents. Barium compounds that do not dissolve well, such as barium sulphate, are not generally harmful, but all kits acid soluble salts are toxic by all exposure routes. The US Department of Health and Human Services (DHHS) has not classified barium as to its carcinogenicity. The US EPA has determined that barium is not likely to be carcinogenic to humans following ingestion and that there is insufficient information to determine whether it will be carcinogenic to humans following inhalation exposure. OSHA: Soluble barium compounds except barium sulphate have been listed as carcinogenic. IARC: Barium has not been evaluated for carcinogenicity.
Benz(a)anthracene **toxic to skin, lungs, immune system** **causes cancer of lung, skin, and stomach**	PubChem: Benz(a)anthracene is a crystalline, aromatic hydrocarbon consisting of four fused benzene rings, produced by incomplete combustion of organic matter. Benz(a)anthracene is primarily found in gasoline and diesel exhaust, tobacco and cigarette smoke, coal tar and coal tar pitch, coal combustion emissions, charcoal-broiled foods, amino acids, fatty acids and carbohydrate pyrolysis products, wood and soot smoke, and creosote, asphalt and mineral oils. This substance is used only for research purposes. Benz(a)anthracene is reasonably anticipated to be a human carcinogen. ATSDR: Benz-a-anthracene is a polycyclic aromatic hydrocarbon (PAH) – see detailed review above for acenaphthylene.
Benzene **acute and chronic irritant to CNS, eyes and skin** **toxic to immune system, bone marrow, and blood** **causes leukaemia – acute myeloid leukaemia (AML)**	ATSDR: Benzene is a colourless liquid with a sweet odour. It evaporates into the air very quickly and dissolves slightly in water. It is highly flammable and is formed from both natural processes and human activities. Benzene is widely used in the United States; it ranks in the top 20 chemicals for production volume. Some industries use benzene to make other chemicals which are used to make plastics, resins, and nylon and other synthetic fibres. Benzene is also used to make some types of rubbers, lubricants, dyes, detergents, drugs,

Chemical Name	Comments
Group 1 carcinogen	and pesticides. Natural sources of benzene include emissions from volcanoes and forest fires. Benzene is also a natural part of crude oil, gasoline, and cigarette smoke. Outdoor air contains low levels of benzene from tobacco smoke, automobile service stations, exhaust from motor vehicles, and industrial emissions. Vapours (or gases) from products that contain benzene, such as glues, paints, furniture wax, and detergents, can also be a source of exposure. Air around hazardous waste sites or fuel stations will contain higher levels of benzene. Breathing very high levels of benzene can result in death, while high levels can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness. Eating or drinking foods containing high levels of benzene can cause vomiting, irritation of the stomach, dizziness, sleepiness, convulsions, rapid heart rate, and death. The major effect of benzene from long-term exposure is on the blood. Benzene causes harmful effects on the bone marrow and can cause a decrease in red blood cells leading to anaemia. It can also cause excessive bleeding and can affect the immune system, increasing the chance for infection. Long-term exposure to high levels of benzene in the air can cause leukemia, particularly acute myelogenous leukemia, often referred to as AML. This is a cancer of the blood-forming organs. The US Department of Health and Human Services (DHHS), and the US EPA have determined that benzene is a known carcinogen. IARC: OSHA: Benzene has been listed as carcinogenic. NICNAS: Benzene has been assessed as a Priority Existing Chemical (PEC). IARC: IARC: Benzene has been evaluated as carcinogenic to humans (Group 1).
Benzo(a) pyrene (3,4-benzpyrene) **acute and chronic irritant skin, URT, lungs, eyes, CNS** **causes cancer of lung, skin, aplastic anaemia** **Group 1 carcinogen**	PubChem: Benzo[a]pyrene (3,4-benzpyrene) is a potent mutagen and carcinogen. It is a public health concern because of its possible effects on industrial workers, as an environmental pollutant, and as a component of tobacco smoke. 3,4-Benzpyrene is a crystalline, aromatic hydrocarbon consisting of five fused benzene rings and formed during the incomplete combustion of organic matter. 3,4-Benzpyrene is primarily found in gasoline and diesel exhaust, cigarette smoke, coal tar and coal tar pitch, charcoal-broiled foods and certain other foods, amino acids, fatty acids and carbohydrate pyrolysis products, soot smoke, creosote oil, petroleum asphalt and shale oils. This substance is used only for research purposes. 3,4-Benzpyrene is reasonably anticipated to be a human carcinogen.

Chemical Name	Comments
	Symptoms of exposure to this compound include mucous membrane irritation, dermatitis, bronchitis, cough, dyspnea, conjunctivitis, photosensitization, pulmonary edema, reproductive effects and leukemia. Contact with the skin may result in erythema, pigmentation, desquamation, formation of verrucae and infiltration. It may also cause keratoses which are relatively small, heaped-up, scaling, brown plaques on the skin, some of which may be fissured and may itch. Exposure to this type of compound may cause reddening and squamous eczema of the lid margins with only small erosion of the corneal epithelium and superficial changes in the stroma which disappear a month following exposure. Repeated exposure may cause sunlight to have a more severe effect on a person's skin and also an allergic skin rash. Aplastic anemia may also occur. Chronic exposure to the fumes and dust of this type of compound can cause discoloration of the cornea and epithelioma of the lid margin. This compound may be harmful by ingestion or inhalation. It may cause irritation. When heated to decomposition it emits acrid smoke and toxic fumes of carbon monoxide and carbon dioxide. IARC: Benz(a)pyrene has been evaluated as carcinogenic to humans (Group 1). OSHA: Benz(a)pyrene has been listed as carcinogenic. ATSDR: Benz(a)pyrene is a polycyclic aromatic hydrocarbon (PAH) – see detailed review above for acenaphthylene.
Benzo(b)&(k)fluor-anthene	ATSDR: This compound is a polycyclic aromatic hydrocarbon (PAH) – see detailed review above for acenaphthylene.
Benzo(b)fluoranthene	ATSDR: This compound is a polycyclic aromatic hydrocarbon (PAH) – see detailed review above for acenaphthylene.
Benzo(g,h,i)perylene	ATSDR: This compound is a polycyclic aromatic hydrocarbon (PAH) – see detailed review above for acenaphthylene.
Benzo(k)fluoranthene	ATSDR: This compound is a polycyclic aromatic hydrocarbon (PAH) – see detailed review above for acenaphthylene.
Beryllium **toxic to skin, eyes, and lungs** **causes lung cancer** **Group 1 carcinogen**	PubChem: Beryllium is a hard, grayish metal naturally found in mineral rocks, coal, soil, and volcanic dust. Beryllium compounds are commercially mined, and the Beryllium is purified for use in nuclear weapons and reactors, aircraft and space vehicle structures, instruments, x-ray machines, and mirrors. Beryllium ores are used to make specialty ceramics for electrical and high-technology applications. Beryllium alloys are used in automobiles, computers, sports equipment (golf clubs and bicycle frames), and dental bridges. Any dramatic, unexplained weight loss should be considered as possible first indication of beryllium disease. Dust is extremely toxic when inhaled; symptoms include coughing, shortness of breath, and acute or chronic lung disease. There is no record of

Chemical Name	Comments
	illness from ingestion of beryllium. Contact with dust causes conjunctival inflammation of eyes and dermatitis. NTP: Beryllium and beryllium compounds are known to be human carcinogens based on sufficient evidence of carcinogenicity from studies in humans. Beryllium and beryllium compounds were first listed in the Second Annual Report on Carcinogens as reasonably anticipated to be human carcinogens based on sufficient evidence of carcinogenicity from studies in experimental animals. The listing was revised to 'known to be human carcinogens' in the Tenth Report on Carcinogens in 2002. IARC: There is sufficient evidence in humans for the carcinogenicity of beryllium and beryllium compounds. Beryllium and beryllium compounds cause cancer of the lung. There is sufficient evidence in experimental animals for the carcinogenicity of beryllium and beryllium compounds. Beryllium and beryllium compounds are carcinogenic to humans (Group 1). OSHA: Beryllium metal and compounds are listed as carcinogenic.
Bis(2-chloroethyl) ether **acute and chronic irritant skin, eyes, URT, lungs**	ATSDR: Bis(2-chloroethyl) ether is a colourless, non-flammable liquid with a strong unpleasant odour. It dissolves easily in water, and some of it will slowly evaporate to the air. It does not occur naturally. Bis(2-chloroethyl) ether is made in factories, and most of it is used to make pesticides. Some of it is used as a solvent, cleaner, component of paint and varnish, rust inhibitor, or as a chemical intermediate to make other chemicals. Bis(2-chloroethyl) ether causes irritation to the skin, eyes, throat, and lungs. In some cases, damage to the lungs can be severe enough to cause death. Breathing low concentrations will cause coughing and nose, and throat irritation. Animal studies show effects like those observed in people. These effects include irritation to the skin, nose, and lungs; lung damage; and a decrease in growth rate. Animals that survived the exposures recovered fully in 4 to 8 days. Some animal studies indicate that bis(2-chloroethyl) ether can affect the nervous system resulting in sluggish and slow movement, staggering, unconsciousness, and death. The ability of bis(2-chloroethyl) ether to cause cancer in humans has not been established. There is some evidence that bis(2-chloroethyl) ether causes cancer in mice. IARC: Bis(2-chloroethyl) ether is *not classifiable as to its carcinogenicity to humans (Group 3)*.

Chemical Name	Comments
	OSHA: Bis (2-chloroethyl) ether is not listed as carcinogenic.
Bromodichloromethane **similar to chloroform**	ATSDR: Bromodichloromethane is a colourless, non-flammable liquid. Small amounts are formed naturally by algae in the oceans. Some of it will dissolve in water, but it readily evaporates into air. Only small quantities of bromodichloromethane are produced. The small quantities that are produced are used in laboratories or to make other chemicals. However, most bromodichloromethane is formed as a by-product when chlorine is added to drinking water to kill bacteria. No studies are available regarding health effects in people exposed to bromodichloromethane. Animal studies indicate that the liver, kidney, and central nervous system are affected by exposure to bromodichloromethane. The effects of high doses on the central nervous system include sleepiness and incoordination. Longer exposure to lower doses causes damage to the liver and kidneys. There is some evidence from animal studies that bromodichloromethane may cause birth defects at doses high enough to make the mother sick. It is not known if lower doses would cause birth defects. There is evidence that eating or drinking bromodichloromethane causes liver, kidney, and intestinal cancer in rats and mice. The US Department of Health and Human Services (DHHS) has determined that bromodichloromethane is reasonably anticipated to be a human carcinogen. IARC: There is sufficient evidence in experimental animals for the carcinogenicity of bromodichloromethane. Bromodichloromethane is possibly carcinogenic to humans (Group 2B). OSHA: Bromodichloromethane is not listed as carcinogenic.
Bromoform (tribromomethane) **similar to chloroform**	ATSDR: Bromoform and dibromochloromethane are colourless to yellow, heavy, non-flammable, liquids with a sweet odour. Small amounts are formed naturally by plants in the ocean. They are somewhat soluble in water and readily evaporate into the air. Most of the bromoform and dibromochloromethane that enters the environment is formed as by-products when chlorine is added to drinking water to kill bacteria. Only small quantities of bromoform and dibromochloromethane currently are produced. These chemicals were used in the past as solvents and flame retardants, or to make other chemicals, but now they are used mainly as laboratory reagents. Eating or breathing a large amount of bromoform slows down the normal brain activities and causes sleepiness; this tends to go away within a day. Exposure to very high amounts may cause unconsciousness and even death. No studies are available about health effects in people exposed to dibromochloromethane.

Chemical Name	Comments
	Animals exposed to high amounts of bromoform or dibromochloromethane developed liver and kidney injuries. Exposure to low levels of bromoform or dibromochloromethane do not appear to seriously affect the brain, liver, or kidneys. There is no conclusive evidence that bromoform or dibromochloromethane cause cancer in humans because no cancer studies of humans exposed exclusively to these chemicals are available. Studies in animals indicate that long-term intake of either bromoform or dibromochloromethane can cause liver and kidney cancer. The EPA classified bromoform as a probable human carcinogen and dibromochloromethane as a possible human carcinogen. IARC: There is *limited evidence* in experimental animals for the carcinogenicity of bromoform. Bromoform is not classifiable as carcinogenic to humans (Group 3). OSHA: Bromoform is listed as carcinogenic.
Bromomethane **irritant to CNS, lungs** **toxic to liver, CNS and kidneys**	ATSDR: Bromomethane is a manufactured chemical. It also occurs naturally in small amounts in the ocean where it is formed, probably by algae and kelp. It is a colorless, non-flammable gas with no distinct smell. Other names for bromomethane are methyl bromide, mono-bromomethane, and methyl fume. Bromomethane is used to kill a variety of pests including rats, insects, and fungi. It is also used to make other chemicals or as a solvent to get oil out of nuts, seeds, and wool. Inhalation of bromomethane causes headaches, lethargy and nausea after several hours. Inhalation of large amounts causes severe respiratory irritation, dyspnoea, and fluid may build up in the lungs. Bromomethane may also cause muscle tremors, seizures, kidney damage, nerve damage, and even death. Exposure levels leading to death vary from 1,600 to 60,000 parts of bromomethane in 1 million parts of air (1,600-60,000 ppm), depending on the length of the exposure. The respiratory, kidney, and neurologic effects are of the greatest concern to people. No cases of severe effects on the nervous system from long-term exposure to low levels have been noted in people, but studies in rabbits and monkeys have shown moderate to severe injury. Bromomethane on the skin can cause itching, redness, and blisters. Studies in animals suggest that bromomethane does not cause birth defects and does not interfere with reproduction, except at high exposure levels. The US Environmental Protection Agency (EPA) has determined that bromomethane is not classifiable as to its human carcinogenicity. There are no studies available to indicate that

Chemical Name	Comments
	bromomethane is carcinogenic to people. Animal studies do not provide conclusive evidence. OSHA: Bromomethane is listed as carcinogenic. IARC: There is inadequate evidence in humans for the carcinogenicity of methyl bromide (bromomethane). There is limited evidence in experimental animals for the carcinogenicity of methyl bromide. Methyl bromide (bromomethane) is not classifiable as to its carcinogenicity to humans (Group 3).
Cadmium **toxic to lungs and kidneys** **causes cancer of lung, prostate, kidney** **Group 1 carcinogen**	ATSDR: Cadmium is a metal found in the earth's crust, associated with zinc, lead, and copper ores. Pure cadmium is a soft, silver-white metal. Cadmium chloride and cadmium sulphate are soluble in water. Cadmium is used in the manufacture of a wide range of products, including batteries, pigments, coatings and platings, stabilizers for plastics, nonferrous alloys, and photovoltaic devices. Cadmium is emitted to soil, water, and air by non-ferrous metal mining and refining, manufacture and application of phosphate fertilizers, fossil fuel combustion, and waste incineration and disposal. Cadmium can accumulate in aquatic organisms and agricultural crops. Cadmium (as oxide, chloride, and sulphate) will exist in air as particles or vapours from high temperature processes. It can be transported long distances in the atmosphere, where it will deposit, wet or dry, onto soils and water surfaces. Cadmium and its compounds may travel through soil, but its mobility depends on several factors such as pH and amount of organic matter, which will vary depending on the local environment. Generally, cadmium binds strongly to organic matter where it will be immobile in soil and be taken up by plant life, eventually, entering the food supply. Cadmium exists as the hydrated ion or as ionic complexes with other inorganic or organic substances. Soluble forms migrate in water. Insoluble forms of cadmium are immobile and will deposit and absorb to sediments. Breathing air with very high levels of cadmium can severely damage the lungs and may cause death. Breathing air with lower levels of cadmium over long periods of time (for years) results in a build-up of cadmium in the kidney, and if sufficiently high, may result in kidney disease. Damage to the lungs and nasal cavity has been observed in animals exposed to cadmium.

Chemical Name	Comments
	Lung cancer has been found in some studies of workers exposed to cadmium in the air and studies of rats that breathed in cadmium. The US Department of Health and Human Services (DHHS) has determined that cadmium and cadmium compounds are known human carcinogens. The EPA has determined that cadmium is a probable human carcinogen. IARC: There is *sufficient evidence* in humans for the carcinogenicity of cadmium and cadmium compounds. Cadmium and cadmium compounds cause cancer of the lung. Also, positive associations have been observed between exposure to cadmium and cadmium compounds and cancer of the kidney and cancer of the prostate. There is *sufficient evidence* in experimental animals for the carcinogenicity of cadmium compounds. There is *limited evidence* in experimental animals for the carcinogenicity of cadmium metal. Cadmium and cadmium compounds are *carcinogenic to humans (Group 1).* OSHA: Cadmium metal and compounds, and cadmium oxide fume are listed as carcinogenic.
Carbon disulfide **toxic to CNS, heart, liver, and skin**	ATSDR: Pure carbon disulphide is a colourless liquid with a pleasant odour that is like the smell of chloroform. The impure carbon disulphide that is usually used in most industrial processes is a yellowish liquid with an unpleasant odour, like that of rotting radishes. Carbon disulphide evaporates at room temperature, and the vapor is more than twice as heavy as air. It easily explodes in air and catches fire very easily. In nature, small amounts of carbon disulphide are found in gases released to the earth's surface as, for example, in volcanic eruptions or over marshes. Commercial carbon disulphide is made by combining carbon and sulphur at very high temperatures. At very high levels, carbon disulphide may be life-threatening because of its effects on the nervous system. People who breathed carbon disulphide near an accident involving a railroad car showed changes in breathing and some chest pains. Some workers who breathed high levels during working hours for at least 6 months had headaches, tiredness, and trouble sleeping. However, these workers may have been exposed to other chemicals besides carbon disulphide. Studies in animals indicate that carbon disulphide can affect the normal functions of the brain, liver, and heart. After pregnant rats

Chemical Name	Comments
	breathed carbon disulphide in the air, some of the newborn rats died or had birth defects. High concentrations of carbon disulphide have caused skin burns when the chemical accidentally touched people's skin. The US Department of Health and Human Services (DHHS), and the EPA have not classified carbon disulphide for carcinogenicity. IARC: There is inadequate evidence in humans for the carcinogenicity of carbon disulphide. There is sufficient evidence in experimental animals for the carcinogenicity of carbon disulphide. Carbon disulphide is possibly carcinogenic to humans (Group 2B). OSHA: Carbon disulphide is listed as carcinogenic.
Carbon tetrachloride **toxic to liver, kidneys, and CNS**	ATSDR: Carbon tetrachloride is a manufactured chemical that does not occur naturally. It is a clear liquid with a sweet smell that can be detected at low levels. It is also called carbon chloride, methane tetrachloride, perchloromethane, tetrachloroethane, or benziform. Carbon tetrachloride is most often found in the air as a colourless gas. It is not flammable and does not dissolve in water very easily. It was used in the production of refrigeration fluid and propellants for aerosol cans, as a pesticide, as a cleaning fluid and degreasing agent, in fire extinguishers, and in spot removers. Because of its harmful effects, these uses are now banned, and it is only used in some industrial applications. High exposure to carbon tetrachloride can cause liver, kidney, and central nervous system damage. These effects can occur after ingestion or breathing carbon tetrachloride, and possibly from exposure to the skin. The liver is especially sensitive to carbon tetrachloride because cells are damaged or destroyed. Kidneys also are damaged, causing a build-up of wastes in the blood. If exposure is low and brief, the liver and kidneys can repair the damaged cells and function normally again. Effects of carbon tetrachloride are more severe in persons who drink large amounts of alcohol. If exposure is very high, the nervous system, including the brain, is affected. People may feel intoxicated and experience headaches, dizziness, sleepiness, and nausea and vomiting. These effects may subside if exposure is stopped, but in severe cases, coma and even death may occur. There have been no studies of the effects of carbon tetrachloride on reproduction in humans, but studies in rats showed that long-term inhalation may cause decreased fertility. Studies in humans have not been able to determine whether carbon tetrachloride can cause cancer because usually there has

Chemical Name	Comments
	been exposure to other chemicals at the same time. Swallowing or breathing carbon tetrachloride for years caused liver tumours in animals. Mice that breathed carbon tetrachloride also developed tumours of the adrenal gland. The US Department of Health and Human Services (DHHS) has determined that carbon tetrachloride may reasonably be anticipated to be a carcinogen. The EPA has determined that carbon tetrachloride is a probable human carcinogen. IARC: There is *inadequate evidence* in humans for the carcinogenicity of carbon tetrachloride. There is *sufficient evidence* in experimental animals for the carcinogenicity of carbon tetrachloride. Carbon tetrachloride is possibly carcinogenic to humans (Group 2B). OSHA: Carbon tetrachloride is listed as carcinogenic.
Chloroethane (ethyl chloride) **irritant to skin, eyes, URT, and CNS**	ATSDR: Chloroethane is a colourless gas at room temperature and pressure. It has a characteristically sharp smell. It is a liquid when stored in pressurized containers; however, the liquid evaporates quickly when exposed to room air. Chloroethane catches fire easily. It was used in leaded gasoline. It is used in the production of cellulose, dyes, medicinal drugs, and other commercial products, and as a solvent and refrigerant. It is also used to numb the skin before medical procedures such as ear piercing and skin biopsies and as a treatment in sports injuries. Brief exposure to high levels can produce temporary feelings of drunkenness. At higher levels, it can cause lack of muscle coordination and unconsciousness. It can also cause stomach cramps, nausea, vomiting, and eye irritation. Chloroethane is sometimes applied to the skin as a numbing agent before surgery. If it is applied for too long, frostbite can result. Some people had allergic reactions to it, and others experienced mild pain after being sprayed for 10 seconds. Laboratory tests in animals have shown that long-term exposure can cause cancer in mice. It is not known whether it causes cancer in humans. IARC: Chloroethane is not classifiable as to its carcinogenicity in humans (Group 3). OSHA: Chloroethane is listed as carcinogenic.
Chloroform **toxic to skin, liver, kidneys, and CNS**	ATSDR: Chloroform is a colourless liquid with a pleasant, non-irritating odour and a slightly sweet taste. It will burn only when it reaches very high temperatures.

Chemical Name	Comments
	In the past, chloroform was used as an inhaled anaesthetic during surgery. Chloroform is used to make other chemicals and can also be formed in small amounts when chlorine is added to water. Other names for chloroform are trichloromethane and methyl trichloride. Breathing about 900 parts of chloroform per million parts of air (900 ppm) for a short time can cause dizziness, fatigue, and headache. Breathing air, eating food, or drinking water containing high levels of chloroform for long periods of time may damage the liver and kidneys. Large amounts of chloroform can cause sores when chloroform touches the skin. Animal studies have shown that miscarriages occurred in rats and mice that breathed air containing 30 to 300 ppm chloroform during pregnancy and also in rats that ate chloroform during pregnancy. Offspring of rats and mice that breathed chloroform during pregnancy had birth defects. Abnormal sperm were found in mice that breathed air containing 400 ppm chloroform for a few days. The US Department of Health and Human Services (DHHS) has determined that chloroform may reasonably be anticipated to be a carcinogen. Rats and mice that ate food or drank water with chloroform developed cancer of the liver and kidneys. IARC: There is *inadequate evidence* in humans for the carcinogenicity of chloroform. There is *sufficient evidence* in experimental animals for the carcinogenicity of chloroform. Chloroform is possibly carcinogenic to humans (Group 2B). OSHA: Chloroform is listed as carcinogenic.

Chemical Name	Comments
Chloromethane (methyl chloride) **toxic to heart, liver, kidneys, and CNS**	ATSDR: Chloromethane is also known as methyl chloride. It is a clear, colorless gas. It has a faint, sweet odor that is noticeable only at levels that may be toxic. It is heavier than air, and it is extremely flammable. Breathing high levels of chloromethane can cause serious problems to the nervous system, including convulsions and coma. It can also affect the liver, kidneys, and heart. Lower exposures can also cause staggering, blurred or double vision, dizziness, fatigue, personality changes, confusion, tremors, nausea, or vomiting. These symptoms can last for several months or years. It could also affect the heart rate and blood pressure. Some animal studies showed that animals that breathed low levels of chloromethane experienced slower growth and had brain damage. In other animal studies, males that were exposed to chloromethane were less fertile, or even sterile, or produced damaged sperm. Females that became pregnant by these males lost their developing young. There is no evidence that chloromethane causes cancer in people. In animal studies, male mice that breathed contaminated air for 2 years developed tumours in their kidneys, but female mice, and male and female rats did not. The EPA has determined that chloromethane is a possible human carcinogen. IARC: There is inadequate evidence for the carcinogenicity of methyl chloride (chloromethane) to humans. There is inadequate evidence for the carcinogenicity of methyl chloride (chloromethane) in experimental animals. Methyl chloride (chloromethane) is not classifiable as to its carcinogenicity to humans (Group 3). OSHA: Chloromethane is listed as carcinogenic.
Chromium (III+VI) **toxic to skin and respiratory tract** **causes nasal sinus and lung cancer** **Group 1 carcinogen**	ATSDR: Chromium is a naturally occurring element found in rocks, animals, plants, and soil. It can exist in several different forms. Depending on the form it takes, it can be a liquid, solid, or gas. The most common forms are chromium (0), chromium (III), and chromium (VI). No taste or odour is associated with chromium compounds. The metal chromium, which is the chromium (0) form, is used for making steel. Chromium (VI) and chromium (III) are used for chrome plating, dyes and pigments, leather tanning, and wood preserving. Chromium (III) is an essential nutrient that helps the body use sugar, protein, and fat. Breathing high levels of chromium (VI) can cause irritation to the lining of the nose, nose ulcers, runny nose, and breathing problems, such as asthma, cough, shortness of breath, or

Chemical Name	Comments
	wheezing. The concentrations of chromium in air that can cause these effects may be different for different types of chromium compounds, with effects occurring at much lower concentrations for chromium (VI) compared with chromium (III). The main health problems seen in animals following ingestion of chromium (VI) compounds are irritation and ulcers in the stomach and small intestine and anaemia. Chromium (III) compounds are much less toxic and do not appear to cause these problems. Sperm damage and damage to the male reproductive system have also been seen in laboratory animals exposed to chromium (VI). Skin contact with certain chromium (VI) compounds can cause skin ulcers. Some people are extremely sensitive to chromium (VI) or chromium (III). Allergic reactions consisting of severe redness and swelling of the skin have been noted. The US Department of Health and Human Services (DHHS), and the EPA have determined that chromium (VI) compounds are known human carcinogens. In workers, inhalation of chromium (VI) has been shown to cause lung cancer. Chromium (VI) also causes lung cancer in animals. An increase in stomach tumours was observed in humans and animals exposed to chromium(VI) in drinking water. IARC: There is sufficient evidence in humans for the carcinogenicity of chromium (VI) compounds. Chromium (VI) compounds cause cancer of the lungs, and positive associations have been observed between exposure to chromium (VI) compounds and cancer of the nose and nasal sinuses. There is sufficient evidence in experimental animals for the carcinogenicity of chromium (VI) compounds. Chromium (VI) compounds are carcinogenic to humans (Group 1). OSHA: Chromium (VI) compounds, chromium metal, and chromium (III) compounds as Cr are listed as carcinogenic.
Chrysene	ATSDR: Chrysene is a polycyclic aromatic hydrocarbon (PAH) – see detailed review above for acenaphthylene.
cis-1,2-Dichloroethene **irritant to skin, eyes, URT, and CNS**	ATSDR: 1,2-Dichloroethene, also called 1,2-dichloroethylene, is a highly flammable, colourless liquid with a sharp, harsh odour. It is used to produce solvents and in chemical mixtures. Very small amounts of 1,2-dichloroethene can be detected by odour in air (about 17 parts of 1,2-dichloroethene per million parts of air [17 ppm]). There are two forms of 1,2-dichloroethene; one is called *cis*-1,2-dichloroethene and the other is called *trans*-1,2-di-chloroethene. Sometimes both forms are present as a mixture.

Chemical Name	Comments
	Breathing high levels of 1,2-dichloroethene can cause nausea, drowsiness, and lethargy. Breathing very high levels can be fatal. When animals breathed high levels of *trans*-1,2-dichloroethene for short or longer periods of time, their livers and lungs were damaged, and the effects were more severe with longer exposure times. Animals that breathed very high levels of *trans*-1,2-dichloroethene had damaged hearts. Animals that ingested extremely high doses of *cis*- or *trans*-1,2-dichloroethene died. Lower doses of *cis*-1,2-dichloroethene caused effects on the blood, such as decreased numbers of red blood cells, and effects on the liver. The long-term (365 days or longer) human health effects after exposure to low concentrations of 1,2-dichloroethene aren't known. The EPA has determined that *cis*-1,2-dichloroethene is not classifiable as to its human carcinogenicity. No EPA cancer classification is available for *trans*-1,2-dichloroethene. IARC: Not evaluated for carcinogenicity. OSHA: Not listed as carcinogenic.
cis-1,4-Dichloro-2-butene **irritant to skin, eyes, URT, and CNS**	PubChem: 1,4-dichloro-2-butene is a clear colorless liquid. It is flammable, though may be difficult to ignite. Corrosive to tissue. Denser than water and insoluble in water. Vapors heavier than air. Used to make other chemicals. Inhalation of vapor irritates nose and throat. Contact with eyes causes intense irritation and tears. Contact of liquid with skin causes severe blistering and dermatitis. Ingestion causes severe irritation of mouth and stomach. IARC: 1,4-dichloro-2-butene is not classifiable as to its carcinogenicity to humans (Group 3). OSHA: 1,4-dichloro-2-butene is listed as carcinogenic.
Cobalt **toxic to heart, liver, kidneys, and skin**	ATSDR: Cobalt is a naturally occurring element found in rocks, soil, water, plants, and animals. Cobalt is used to produce alloys used in the manufacture of aircraft engines, magnets, grinding and cutting tools, artificial hip and knee joints. Cobalt compounds are also used to colour glass, ceramics and paints, and used as a drier for porcelain enamel and paints. Radioactive cobalt is used for commercial and medical purposes. ^{60}Co (read as cobalt sixty) is used for sterilizing medical equipment and consumer products, radiation therapy for treating cancer patients, manufacturing plastics, and irradiating food. ^{57}Co is used in medical and scientific research. It takes about 5.27

Chemical Name	Comments
	years for half of ^{60}Co to give off its radiation and about 272 days for ^{57}Co; this is called the half-life. Cobalt can benefit or harm human health. Cobalt is beneficial for humans because it is part of vitamin B12. Exposure to high levels of cobalt can result in lung and heart effects and dermatitis. Liver and kidney effects have also been observed in animals exposed to high levels of cobalt. Exposure to large amounts of radiation from radioactive cobalt can damage cells in the body from the radiation. Nonradioactive cobalt has not been found to cause cancer in humans or animals following exposure in food or water. Cancer has been shown, however, in animals that breathed cobalt or when cobalt was placed directly into the muscle or under the skin. IARC: Cobalt and cobalt compounds are *possibly carcinogenic to humans (Group 2B)*. OSHA: Cobalt and cobalt compounds are listed as carcinogenic.
Dibenzo (a, h) anthracene **toxic to skin, lungs, immune system** **causes cancer of lung, skin, and stomach**	ATSDR: Dibenzo (a, h) anthracene is a polycyclic aromatic hydrocarbon (PAH) – see detailed review above for acenaphthylene. IARC: There is *sufficient evidence* that dibenzo (*a, h*) anthracene is carcinogenic to experimental animals. Dibenzo (a, h) anthracene is probably carcinogenic to humans (Group 2A). OSHA: Included with coal tar pitch volatiles and listed as carcinogenic.
Dichloromethane (methylene chloride) **irritant to skin and CNS** **causes liver cancer and non-Hodgkin's lymphoma** **Group 2A probable carcinogen**	ATSDR: Dichloromethane is a colourless liquid with a mild, sweet odour. Another name for it is methylene chloride. Dichloromethane does not occur naturally in the environment. It is used as an industrial solvent and as a paint stripper. It may also be found in some aerosol and pesticide products and is used in the manufacture of photographic film. Inhalation of large amounts of dichloromethane causes dizziness, nausea and paresthesia of the fingers and toes. A person breathing smaller amounts may become less attentive and less accurate in tasks requiring hand-eye coordination. Skin contact with dichloromethane causes burning and redness of the skin. An increased cancer risk was seen in mice breathing large amounts of dichloromethane for a long time. The US Department of Health and Human Services (DHHS) has determined that dichloromethane can be reasonably anticipated to be a cancer-causing chemical. The EPA has determined that dichloromethane is a probable cancer-causing agent in humans.

Chemical Name	Comments
	IARC: There is limited evidence in humans for the carcinogenicity of dichloromethane. Positive associations have been observed between exposure to dichloromethane and cancer of the biliary tract and non-Hodgkin lymphoma. There is sufficient evidence for the carcinogenicity of dichloromethane in experimental animals. Dichloromethane is probably carcinogenic to humans (Group 2A). OSHA: Dichloromethane is listed as carcinogenic.
Ethanol **toxic to CNS and gastro-intestinal tract** **excessive consumption causes GIT cancers**	PubChem: Ethanol is a clear, colorless liquid rapidly absorbed from the gastrointestinal tract and distributed throughout the body. It has bactericidal activity and is used often as a topical disinfectant. It is widely used as a solvent and preservative in pharmaceutical preparations as well as serving as the primary ingredient in alcoholic beverages. Ethanol has widespread use as a solvent of substances intended for human contact or consumption, including scents, flavorings, colorings, and medicines. Ethanol has a depressive effect on the central nervous system and because of its psychoactive effects, it is considered a drug. Ethanol has a complex mode of action and affects multiple systems in the brain, most notably it acts as an agonist to the GABA receptors. Death from ethanol consumption is possible when blood alcohol level reaches 0. 4%. A blood level of 0. 5% or more is commonly fatal. Levels of even less than 0. 1% can cause intoxication, with unconsciousness often occurring at 0. 3-0. 4 %. Ethanol is metabolized by the body as an energy-providing carbohydrate nutrient, as it metabolizes into acetyl CoA, an intermediate common with glucose metabolism, that can be used for energy in the citric acid cycle or for biosynthesis. Ethanol within the human body is converted into acetaldehyde by alcohol dehydrogenase and then into acetic acid by acetaldehyde dehydrogenase. The product of the first step of this breakdown, acetaldehyde, is more toxic than ethanol. Acetaldehyde is linked to most of the clinical effects of alcohol. It has been shown to increase the risk of developing cirrhosis of the liver, multiple forms of cancer, and alcoholism. Industrially, ethanol is produced both as a petrochemical, through the hydration of ethylene, and biologically, by fermenting sugars with yeast. Small amounts of ethanol are endogenously produced by gut microflora through anaerobic fermentation. However, most ethanol detected in biofluids and tissues likely comes from consumption of alcoholic beverages. Absolute ethanol or anhydrous alcohol generally refers to purified ethanol, containing no more than one percent water. Absolute alcohol is not intended for human consumption. It often contains trace amounts of toxic benzene, used to remove water by azeotropic distillation. Consumption of this form of ethanol can be fatal over a short time period. Generally absolute or pure ethanol is used as a solvent for

Chemical Name	Comments
	lab and industrial settings where water will disrupt a desired reaction. Pure ethanol is classed as 200 proof in the USA and Canada, equivalent to 175 degrees proof in the UK system. Ethanol is a general biomarker for the consumption of alcohol IARC: There is sufficient evidence in humans for the carcinogenicity of alcohol consumption. Alcohol consumption causes cancers of the oral cavity, pharynx, larynx, oesophagus, colorectum, liver (hepatocellular carcinoma) and female breast. Also, an association has been observed between alcohol consumption and cancer of the pancreas. For cancer of the kidney and non-Hodgkin lymphoma, there is evidence suggesting lack of carcinogenicity. There is sufficient evidence in humans for the carcinogenicity of acetaldehyde associated with the consumption of alcoholic beverages. Acetaldehyde associated with the consumption of alcoholic beverages causes cancers of the oesophagus and of upper aerodigestive tract combined. There is sufficient evidence in experimental animals for the carcinogenicity of ethanol. There is sufficient evidence in experimental animals for the carcinogenicity of acetaldehyde. Alcohol consumption is carcinogenic to humans (Group 1). Ethanol in alcoholic beverages is carcinogenic to humans (Group 1). Acetaldehyde associated with the consumption of alcoholic beverages is carcinogenic to humans (Group 1). OSHA: Ethanol is listed as carcinogenic.
Ethylbenzene **irritant to skin, eyes, URT, and CNS**	ATSDR: Ethylbenzene is a colourless, flammable liquid that smells like gasoline. It is naturally found in coal tar and petroleum and is also found in manufactured products such as inks, pesticides, and paints. Ethylbenzene is used primarily to make another chemical, styrene. Other uses include as a solvent, in fuels, and to make other chemicals. Exposure to high levels of ethylbenzene in air for short periods can cause eye and throat irritation. Exposure to higher levels can result in dizziness. Irreversible damage to the inner ear and hearing has been observed in animals exposed to relatively low concentrations of ethylbenzene for several days to weeks. Exposure to relatively low concentrations of ethylbenzene in air for several months to years causes kidney damage in animals. IARC: There is *inadequate evidence* in humans for the carcinogenicity of ethylbenzene. There is *sufficient evidence* in experimental animals for the carcinogenicity of ethylbenzene.

Chemical Name	Comments
	Ethylbenzene is *possibly carcinogenic to humans (Group 2B)*. OSHA: Ethylbenzene is listed as carcinogenic.
Fluoranthene	ATSDR: Fluoranthene is a polycyclic aromatic hydrocarbon (PAH) – see detailed review above for acenaphthylene.
Fluorene	ATSDR: Fluorene is a polycyclic aromatic hydrocarbon (PAH) – see detailed review above for acenaphthylene.
Fluoride **irritant to skin, eyes, and URT**	ATSDR: Fluorides, hydrogen fluoride, and fluorine are chemically related. Fluorine is a naturally-occurring, pale yellow-green gas with a sharp odour. It combines with metals to make fluorides such as sodium fluoride and calcium fluoride, both white solids. Sodium fluoride dissolves easily in water, but calcium fluoride does not. Fluorine also combines with hydrogen to make hydrogen fluoride, a colourless gas. Hydrogen fluoride dissolves in water to form hydrofluoric acid. Fluorine and hydrogen fluoride are used to make certain chemical compounds. Hydrofluoric acid is used for etching glass. Other fluoride compounds are used in making steel, chemicals, ceramics, lubricants, dyes, plastics, and pesticides. Fluorides are often added to drinking water supplies and to a variety of dental products, including toothpaste and mouth rinses, to prevent dental cavities. Small amounts of fluoride help prevent tooth cavities, but high levels can harm human health. In adults, exposure to high levels of fluoride can result in denser bones. However, if exposure is high enough, these bones may be more fragile and brittle and there may be a greater risk of breaking the bone. In animals, exposure to extremely high doses of fluoride can result in decreased fertility and sperm and testes damage. Fluorine and hydrogen fluoride are very irritating to the skin, eyes, and respiratory tract. At high levels, such as may occur through exposure from an industrial accident, hydrogen fluoride may also damage the heart. Most of the studies of people living in areas with fluoridated water or naturally high levels of fluoride in drinking water did not find an association between fluoride and cancer risk. Two animal cancer studies were inconclusive. IARC: Fluorides (inorganic, used in drinking-water) *are not classifiable as to their carcinogenicity to humans (Group 3)*. OSHA: Fluorides are not listed as carcinogenic.
Hepta-Dioxins	See above for all dioxins identified.
Hepta-Furans	See above for all furans identified.

Chemical Name	Comments
Hexachloroethane **irritant to skin, eyes, URT**	ATSDR: Hexachloroethane is a colourless solid that gradually evaporates when it is exposed to air. It is also called HCE, perchloroethane, and carbon hexachloride. Its vapours smell like camphor. In the United States, about half of the hexachloroethane is used by the military for smoke-producing devices. It is also used to remove air bubbles in melted aluminium. Hexachloroethane may be present as an ingredient in some fungicides, insecticides, lubricants, and plastics. Hexachloroethane does not occur naturally in the environment. It is no longer made but is formed as a by-product in the production of some chemicals. Some hexachloroethane can be formed by incinerators when materials containing chlorinated hydrocarbons are burned. Hexachloroethane itself does not catch fire easily. Some hexachloroethane can also be formed when chlorine reacts with carbon compounds in drinking water. Based on animal studies, hexachloroethane in air can irritate the nose and lungs and cause some build-up of mucus in the nose, much like an allergy. It can also irritate the eyes and make them tear. Liver tumours developed in mice that were orally exposed to hexachloroethane for their whole lifetime. Male rats that were exposed to hexachloroethane for their lifetime developed kidney tumours. The US Department of Health and Human Services (DHHS) has determined that hexachloroethane may reasonably be anticipated to be a carcinogen. IARC: There is *inadequate evidence* in humans for the carcinogenicity of hexachloroethane. There is *sufficient evidence* in experimental animals for the carcinogenicity of hexachloroethane. Hexachloroethane is *possibly carcinogenic to humans (Group 2B)*. OSHA: Hexachloroethane is listed as carcinogenic.
Hexane **toxic to central and peripheral nervous system**	ATSDR: *n*-Hexane is a chemical made from crude oil. Pure *n*-hexane is a colorless liquid with a slightly disagreeable odour. It is highly flammable, and its vapours can be explosive. Pure *n*-hexane is used in laboratories. Most of the *n*-hexane used in industry is mixed with similar chemicals called solvents. The major use for solvents containing *n*-hexane is to extract vegetable oils from crops such as soybeans. These solvents are also used as cleaning agents in the printing, textile, furniture, and shoemaking industries. Certain kinds of special glues used in the roofing and shoe and leather industries also contain *n*-hexane. Several consumer products contain *n*-hexane, such as gasoline, quick-drying glues used in various hobbies, and rubber cement.

Chemical Name	Comments
	The only people known to have been affected by exposure to *n*-hexane used it at work. Breathing large amounts caused numbness in the feet and hands, followed by muscle weakness in the feet and lower legs. Continued exposure led to paralysis of the arms and legs. If removed from the exposure, the workers recovered in 6 months to a year. In laboratory studies, animals exposed to high levels of *n*-hexane in air had signs of nerve damage. Some animals also had lung damage. In other studies, rats exposed to very high levels of *n*-hexane had damage to sperm-forming cells. There is no evidence that *n*-hexane causes cancer in people or animals. The Department of Health and Human Services (DHHS), and the EPA have not classified *n*-hexane for carcinogenicity. IARC: Hexane has not been classified. OSHA: Hexane is listed as carcinogenic.
Indeno (1,2,3-c, d) pyrene	ATSDR: Indeno (1,2,3-c, d) pyrene is a polycyclic aromatic hydrocarbon (PAH) – see detailed review above for acenaphthylene.
Isopropylbenzene (Cumene) **irritant to skin and eyes, and chronic irritant to CNS**	PubChem: Isopropylbenzene is found in Ceylon cinnamon and is a trace constituent of ginger oil. Cumene is the common name for isopropylbenzene, an organic compound that is an aromatic hydrocarbon. It is a constituent of crude oil and refined fuels. It is a flammable colorless liquid. Acute short-term inhalation exposure to cumene may cause headaches, dizziness, drowsiness, slight incoordination, and unconsciousness in humans. Cumene has a potent central nervous system (CNS) depressant action characterized by a slow induction period and long duration of narcotic effects in animals. Cumene is a skin and eye irritant. No information is available on the chronic long-term, reproductive, developmental, or carcinogenic effects of cumene in humans. Animal studies have reported increased liver, kidney, and adrenal weights from inhalation exposure to cumene. The US EPA has classified cumene as a Group D, not classifiable as to human carcinogenicity. OSHA: Cumene is listed as carcinogenic. IARC: Isopropylbenzene has not been classified.
Lead **toxic to multiple organs, especially CNS and kidneys** **Group 2A probably carcinogenic**	ATSDR: Lead is a naturally occurring bluish-grey metal found in small amounts in the earth's crust. Lead can be found in all parts of the environment. Much of it comes from human activities including burning fossil fuels, mining, and manufacturing. Lead has many different uses. It is used in the production of batteries, ammunition, metal products (solder and pipes), and devices to shield X-rays. Because of health concerns, lead from paints and ceramic products, caulking, and pipe solder has been

Chemical Name	Comments
	dramatically reduced in recent years. The use of lead as an additive to gasoline has been banned. The effects of lead are the same whether it enters the body through breathing or swallowing. Lead can affect almost every organ and system in the human body. The main target for lead toxicity is the nervous system, both in adults and children. Long-term exposure of adults can result in decreased performance in some tests that measure functions of the nervous system. It may also cause weakness in fingers, wrists, or ankles. Lead exposure also causes small increases in blood pressure, particularly in middle-aged and older people and can cause anemia. Exposure to high lead levels can severely damage the brain and kidneys in adults or children and ultimately cause death. In pregnant women, high levels of exposure to lead may cause miscarriage. High level exposure in men can damage the organs responsible for sperm production. There is no conclusive proof that lead causes cancer in humans. Kidney tumors have developed in rats and mice that had been given large doses of some lead compounds. The US Department of Health and Human Services (DHHS) has determined that lead and lead compounds are reasonably anticipated to be human carcinogens; and the EPA has determined that lead is a probable human carcinogen. IARC: There is limited evidence in humans for the carcinogenicity of inorganic lead compounds. There is inadequate evidence in humans for the carcinogenicity of organic lead compounds. There is sufficient evidence in experimental animals for the carcinogenicity of inorganic lead compounds. There is sufficient evidence in experimental animals for the carcinogenicity of lead acetate, lead subacetate, lead chromate, and lead phosphate. There is inadequate evidence in experimental animals for the carcinogenicity of lead oxide and lead arsenate. There is inadequate evidence in experimental animals for the carcinogenicity of organic lead compounds. There is inadequate evidence in experimental animals for the carcinogenicity of tetraethyl lead. There is inadequate evidence in experimental animals for the carcinogenicity of lead powder. Inorganic lead compounds are probably carcinogenic to humans (Group 2A). Organic lead compounds are not classifiable as to their carcinogenicity to humans (Group 3). OSHA: Lead inorganic dusts and fumes are listed as carcinogenic. NICNAS: lead compounds have been assessed as Priority Existing Chemicals (PEC).

Chemical Name	Comments
Magnesium **irritant to skin and eyes**	PubChem: Magnesium is a light silvery metal. The more finely divided material reacts with water to liberate hydrogen, a flammable gas. In finely divided forms it is easily ignited. Burns with an intense white flame. Dust irritates eyes in same way as any foreign material. Penetration of skin by fragments of metal is likely to produce local irritation, blisters, and ulcers which may become infected. IARC: Magnesium has not been evaluated for carcinogenicity. OSHA: Magnesium oxide fume is listed as carcinogenic.
Manganese **toxic to CNS**	ATSDR: Manganese is a naturally occurring metal that is found in many types of rocks. Pure manganese is silver-coloured but does not occur naturally. It combines with other substances such as oxygen, sulphur, or chlorine. Manganese occurs naturally in most foods and may be added to some foods. Manganese is used principally in steel production to improve hardness, stiffness, and strength. It may also be used as an additive in gasoline to improve the octane rating of the gas. Manganese is an essential nutrient and eating a small amount of it each day is important to stay healthy. The most common health problems in workers exposed to high levels of manganese involve the nervous system. These health effects include behavioural changes and other nervous system effects, which include movements that may become slow and clumsy. Other less severe nervous system effects such as slowed hand movements have been observed in some workers exposed to lower concentrations in the work place. Exposure to high levels of manganese in air can cause lung irritation and reproductive effects. Nervous system and reproductive effects have been observed in animals after high oral doses of manganese. The EPA concluded that existing scientific information cannot determine whether manganese can cause cancer. IARC: Manganese has not been evaluated for carcinogenicity. OSHA: Manganese compounds and dust are listed as carcinogenic.
Mercury **irritant to skin, eyes, and URT** **toxic to heart, lungs, kidneys, and CNS**	ATSDR: Mercury is a naturally occurring metal which has several forms. The metallic mercury is a shiny, silver-white, odourless liquid. If heated, it is a colourless, odourless gas. Mercury combines with other elements, such as chlorine, sulphur, or oxygen, to form inorganic mercury compounds or "salts," which are usually white powders or crystals. Mercury also combines with carbon to make organic mercury compounds. The most common one, methylmercury, is produced mainly by microscopic

Chemical Name	Comments
	organisms in the water and soil. More mercury in the environment can increase the amounts of methylmercury that these small organisms make. Metallic mercury is used to produce chlorine gas and caustic soda, and is also used in thermometers, dental fillings, and batteries. Mercury salts are sometimes used in skin lightening creams and as antiseptic creams and ointments. Mercury vaporises at room temperature and the vapour is toxic. The nervous system is very sensitive to all forms of mercury. Methylmercury and metallic mercury vapours are more harmful than other forms, because more mercury in these forms reaches the brain. Exposure to high levels of metallic, inorganic, or organic mercury can permanently damage the brain, kidneys, and developing foetus. Effects on brain functioning may result in irritability, shyness, tremors, changes in vision or hearing, and memory problems. Short-term exposure to high levels of metallic mercury vapours may cause effects including lung damage, nausea, vomiting, diarrhea, increases in blood pressure or heart rate, skin rashes, and eye irritation. There are inadequate human cancer data available for all forms of mercury. Mercuric chloride has caused increases in several types of tumors in rats and mice, and methylmercury has caused kidney tumors in male mice. The EPA has determined that mercuric chloride and methylmercury are possible human carcinogens. IARC: There is *inadequate evidence* in humans for the carcinogenicity of mercury and mercury compounds. There is *inadequate evidence* in experimental animals for the carcinogenicity of metallic mercury. There is *limited evidence* in experimental animals for the carcinogenicity of mercuric chloride. There is *sufficient evidence* in experimental animals for the carcinogenicity of methylmercury chloride. Methylmercury compounds are *possibly carcinogenic to humans (Group 2B)*. Metallic mercury and inorganic mercury compounds are *not classifiable as to their carcinogenicity to humans (Group 3)*. OSHA: Mercury, elemental and inorganic compounds as Hg, and methyl mercury are listed as carcinogenic.
Meta- & Para- Xylene **acute and chronic irritant to skin, eyes, URT, lungs, and CNS**	ATSDR: There are three forms of xylene in which the methyl groups vary on the benzene ring: *meta*-xylene, *ortho*-xylene, and *para*-xylene (*m*-, *o*-, and *p*-xylene). These different forms are referred to as isomers. Xylene is a colourless, sweet-smelling liquid that catches on fire easily. It occurs naturally in petroleum and coal tar. Chemical industries produce xylene from petroleum. It is one of the top 30 chemicals produced in the United States in terms of volume.

Chemical Name	Comments
	Xylene is used as a solvent and in the printing, rubber, and leather industries. It is also used as a cleaning agent, a thinner for paint, and in paints and varnishes. It is found in small amounts in aircraft fuel and gasoline. High levels of exposure for short or long periods can cause headaches, lack of muscle coordination, dizziness, confusion, and changes in balance. Exposure of people to high levels of xylene for short periods can also cause irritation of the skin, eyes, nose, and throat; difficulty in breathing; problems with the lungs; delayed reaction time; memory difficulties; stomach discomfort; and possibly changes in the liver and kidneys. It can cause unconsciousness and even death at very high levels. US EPA has found that there is insufficient information to determine whether xylene is carcinogenic. IARC: There is *inadequate evidence* in humans for the carcinogenicity of xylenes. There is *inadequate evidence* in experimental animals for the carcinogenicity of xylenes. Xylenes are *not classifiable as to their carcinogenicity to humans (Group 3)*. OSHA: All isomers of xylene are listed as carcinogenic.
Naphthalene **toxic to GIT and red blood cells**	ATSDR: Naphthalene is a white solid that evaporates easily. Fuels such as petroleum and coal contain naphthalene. It is also called white tar, and tar camphor, and has been used in mothballs and moth flakes. Burning tobacco or wood produces naphthalene. It has a strong, but not unpleasant smell. The major commercial use of naphthalene is in the manufacture of polyvinyl chloride (PVC) plastics. Its major consumer use is in moth repellents and toilet deodorant blocks. 1-Methylnaphthalene and 2-methylnaphthalene are naphthalene-related compounds. 1-Methylnaphthalene is a clear liquid and 2-methylnaphthalene is a solid; both can be smelled in air and in water at very low concentrations. 1-Methylnaphthalene and 2-methylnaphthalene are used to make other chemicals such as dyes and resins. 2-Methylnaphthalene is also used to make vitamin K. Exposure to large amounts of naphthalene may damage or destroy red blood cells, resulting in haemolytic anaemia. Some symptoms of haemolytic anaemia are fatigue, lack of appetite, restlessness, and pale skin. Exposure to large amounts of naphthalene may also cause nausea, vomiting, diarrhoea, haematuria, and a yellow colour to the skin. Rats and mice that breathed naphthalene vapours daily for a lifetime developed irritation and inflammation of their nose and lungs. There are no studies of humans exposed to 1-methylnaphthalene or 2-methylnaphthalene.

Chemical Name	Comments
	Mice fed food containing 1-methylnaphthalene and 2-methylnaphthalene for most of their lives had part of their lungs filled with an abnormal material. There is no direct evidence in humans that naphthalene, 1-methylnaphthalene, or 2-methylnaphthalene cause cancer. However, cancer from naphthalene exposure has been seen in animal studies. Some female mice that breathed naphthalene vapours daily for a lifetime developed lung tumours. Some male and female rats exposed to naphthalene in a similar manner also developed nose tumours. Based on the results from animal studies, the US Department of Health and Human Services (DHHS) concluded that naphthalene is reasonably anticipated to be a human carcinogen. The EPA determined that naphthalene is a possible human carcinogen (Group C) and that the data are inadequate to assess the human carcinogenic potential of 2-methylnaphthalene. IARC: There is *inadequate evidence* in humans for the carcinogenicity of naphthalene. There is *sufficient evidence* in experimental animals for the carcinogenicity of naphthalene. Naphthalene is *possibly carcinogenic to humans (Group 2B)*. OSHA: Naphthalene is listed as carcinogenic.
Nickel **toxic to lungs and nasal sinuses** **allergen causing skin and lung reactions** **causes cancer of lung and para-nasal sinuses** **Group 1 carcinogen**	ATSDR: Nickel is a very abundant natural element. Pure nickel is a hard, silvery-white metal. Nickel can be combined with other metals, such as iron, copper, chromium, and zinc, to form alloys. These alloys are used to make coins, jewellery, and items such as valves and heat exchangers. Most nickel is used to make stainless steel. Nickel can combine with other elements such as chlorine, sulphur, and oxygen to form nickel compounds. Many nickel compounds dissolve in water and have a green colour. Nickel compounds are used for nickel plating, to colour ceramics, to make some batteries, and as substances known as catalysts that increase the rate of chemical reactions. Nickel is found in all soil and is emitted from volcanoes. Nickel is also found in meteorites and on the ocean floor. Nickel and its compounds have no characteristic odour or taste. The most common harmful health effect of nickel in humans is an allergic reaction. Approximately 10-20% of the population is sensitive to nickel. People can become sensitive to nickel when jewellery or other items containing it are in direct contact with the skin for a long time. Once a person is sensitized to nickel, further contact with the metal may produce a reaction. The most common reaction is a skin rash at the site of contact. The skin rash may also occur at a site away from the site of contact. Less frequently, some people who are sensitive to nickel have asthma attacks following exposure to nickel. Some sensitized people react when

Chemical Name	Comments
	they consume food or water containing nickel or breathe dust containing it. People working in nickel refineries or nickel-processing plants have experienced chronic bronchitis and reduced lung function. Workers who drank water containing high amounts of nickel had stomach ache and suffered adverse effects to their blood and kidneys. Damage to the lung and nasal cavity has been observed in rats and mice breathing nickel compounds. Eating or drinking large amounts of nickel has caused lung disease in dogs and rats and has affected the stomach, blood, liver, kidneys, and immune system in rats and mice, as well as their reproduction and development. Cancers of the lung and nasal sinus have resulted when workers breathed dust containing high levels of nickel compounds while working in nickel refineries or nickel processing plants. The US Department of Health and Human Services (DHHS) has determined that nickel metal may reasonably be anticipated to be a carcinogen and that nickel compounds are known human carcinogens. The EPA has determined that nickel refinery dust and nickel subsulfide are human carcinogens. IARC: There is sufficient evidence in humans for the carcinogenicity of mixtures that include nickel compounds and nickel metal. These agents cause cancers of the lung and of the nasal cavity and paranasal sinuses. There is sufficient evidence in experimental animals for the carcinogenicity of nickel monoxides, nickel hydroxides, nickel sulfides (including nickel subsulfide), nickel acetate, and nickel metal. There is limited evidence in experimental animals for the carcinogenicity of nickelocene, nickel carbonyl, nickel sulfate, nickel chloride, nickel arsenides, nickel antimonide, nickel selenides, nickel sulfarsenide, and nickel telluride. There is inadequate evidence in experimental animals for the carcinogenicity of nickel titanate, nickel trioxide, and amorphous nickel sulfide. In view of the overall findings in animals, there is sufficient evidence in experimental animals for the carcinogenicity of nickel compounds and nickel metal. Nickel compounds are carcinogenic to humans (Group 1). OSHA: Nickel and nickel compounds are listed as carcinogenic.
OCDD	See above for all dioxins identified.
OCDD I-TEF	See above for all dioxins identified.
OCDD I-TEQ3 (LOR)	See above for all dioxins identified.
OCDD WHO-TEF	See above for all dioxins identified.

Chemical Name	Comments
OCDD WHO-TEQ3 (LOR)	See above for all dioxins identified.
OCDF	See above for all furans identified.
Octa-Dioxin	See above for all dioxins identified.
Octa-Furan	See above for all furans identified.
PCBs (Sum of total) **toxic to skin and liver** **causes liver cancer** **Group 1 carcinogen**	ATSDR: Polychlorinated biphenyls (PCBs) are mixtures of up to 209 individual chlorinated compounds (known as congeners). There are no known natural sources of PCBs. PCBs are either oily liquids or solids that are colourless to light yellow. Some PCBs can exist as a vapor in air. PCBs have no known smell or taste. PCBs have been used as coolants and lubricants in transformers, capacitors, and other electrical equipment because they don't burn easily and are good insulators. The manufacture of PCBs was stopped in the U.S. in 1977 because of evidence they build up in the environment and can cause harmful health effects. Products made before 1977 that may contain PCBs include old fluorescent lighting fixtures and electrical devices containing PCB capacitors, and old microscope and hydraulic oils. The most commonly observed health effects in people exposed to large amounts of PCBs are skin conditions such as acne and rashes. Studies in exposed workers have shown changes in blood and urine that may indicate liver damage. PCB exposures in the general population are not likely to result in skin and liver effects. Most of the studies of health effects of PCBs in the general population examined children of mothers who were exposed to PCBs. Animals that ate food containing large amounts of PCBs for short periods of time had mild liver damage and some died. Animals that ate smaller amounts of PCBs in food over several weeks or months developed various kinds of health effects, including anaemia; acne-like skin conditions; and liver, stomach, and thyroid gland injuries. Other effects of PCBs in animals include changes in the immune system, behavioural alterations, and impaired reproduction. PCBs are not known to cause birth defects. Studies of workers indicate that PCBs were associated with certain kinds of cancer in humans, such as cancer of the liver and biliary tract. Rats that ate food containing high levels of PCBs for two years developed liver cancer. The US Department of Health and Human Services (DHHS) has concluded that PCBs may reasonably be anticipated to be carcinogens. PCBs have been classified as probably carcinogenic by the Environmental Protection Agency (EPA). IARC: There is sufficient evidence in experimental animals for the carcinogenicity of PCBs. There is sufficient evidence in

Chemical Name	Comments
	experimental animals for the carcinogenicity of PCB-126, PCB-118, Aroclor 1260, Aroclor 1254, and Kanechlor 500. There is limited evidence in experimental animals for the carcinogenicity of PCB-153, 4′-OH-PCB-30, 4′OH-PCB-61, Aroclor 1242, Aroclor 1016, Clophen A30, and Clophen A60. There is inadequate evidence in experimental animals for the carcinogenicity of PCB-138, Kanechlor 300, and Kanechlor 400. Congeners for which there is sufficient evidence in experimental animals for carcinogenicity (PCB-126 and PCB-118) are agonists of the aryl hydrocarbon receptor and exhibit dioxin-like properties. Commercial mixtures for which there is sufficient evidence in experimental animals for carcinogenicity are highly chlorinated and are known to include aryl-hydrocarbon receptor agonists that exhibit dioxin-like properties, as well as agonists of the constitutive androstane receptor. The commercial mixtures for which there is limited evidence in experimental animals generally have a low degree of chlorination but are also known to contain congeners that are agonists of the aryl hydrocarbon and/or constitutive androstane receptors. The relative contributions of the different congeners (dioxin-like and non-dioxin-like) to the carcinogenicity of the commercial mixtures is not known. PCBs are carcinogenic to humans (Group 1). “Dioxin-like” PCBs, with a toxicity equivalency factor (TEF) according to WHO (PCB-77, PCB-81, PCB-105, PCB-114, PCB-118, PCB-123, PCB-126, PCB-169, PCB-156, PCB-157, PCB-167, PCB-189), are carcinogenic to humans (Group 1). OSHA: PCBs are not listed as carcinogenic.
Pentachloroethane **irritant to skin, eyes, URT**	PubChem: Pentachloroethane is a colorless liquid with a chloroform-like odor. Insoluble in water and denser than water. It is toxic by inhalation and ingestion and irritating to the skin and eyes. It is used as a solvent. OSHA: Pentachloroethane is listed as carcinogenic. IARC: No epidemiological data relevant to the carcinogenicity of pentachloroethane were available. There is *limited evidence* in experimental animals for the carcinogenicity of pentachloroethane. Pentachloroethane is *not classifiable as to its carcinogenicity to humans (Group 3)*.
Phenanthrene	ATSDR: Phenanthrene is a polycyclic aromatic hydrocarbon (PAH) – see detailed review above for acenaphthylene.
Pyrene	ATSDR: Pyrene is a polycyclic aromatic hydrocarbon (PAH) – see detailed review above for acenaphthylene.

Chemical Name	Comments
Styrene **irritant to skin, eyes, URT, and CNS** **toxic to CNS, URT and liver**	ATSDR: Styrene is a colourless liquid that evaporates easily and has a sweet smell. It often contains other chemicals that give it a sharp, unpleasant smell. Styrene is widely used to make plastics and rubber. Products containing styrene include insulation, fiberglass, plastic pipes, automobile parts, shoes, drinking cups and other food containers, and carpet backing. Most of these products contain styrene linked together in a long chain (polystyrene) as well as unlinked styrene. Inhalation of high levels of styrene may cause nervous system effects such as changes in colour vision, tiredness, feeling drunk, slowed reaction time, concentration problems, or balance problems. Hearing loss has been observed in animals exposed to very high concentrations of styrene. Changes in the lining of the nose and damage to the liver has also been observed in animals exposed to high concentrations of styrene. The US Department of Health and Human Services (DHHS), National Toxicology Program (NTP) listed styrene as reasonably anticipated to be a human carcinogen. IARC: There is *limited evidence* in humans for the carcinogenicity of styrene. There is *limited evidence* in experimental animals for the carcinogenicity of styrene. Styrene is *possibly carcinogenic to humans (Group 2B)*. OSHA: Styrene is listed as carcinogenic.
Tetrachloroethylene **irritant to skin, eyes, URT, and CNS** **toxic to CNS** **associated with bladder cancer, multiple myeloma, and non-Hodgkin's lymphoma** **Group 2A probably carcinogenic**	ATSDR: Tetrachloroethylene is a non-flammable colourless liquid. Other names for tetrachloroethylene include perchloroethylene, PCE, perc, tetrachloroethene, and perchlor. Most people can smell tetrachloroethylene when it is present in the air at a level of 1 part in 1 million parts of air (1 ppm) or more. Tetrachloroethylene is used as a dry-cleaning agent and metal degreasing solvent. It is also used as a starting material (building block) for making other chemicals and is used in some consumer products. Breathing high levels of tetrachloroethylene for a brief period may cause dizziness or drowsiness, headache, and incoordination; higher levels may cause unconsciousness and even death. Exposure for longer periods to low levels of tetrachloroethylene may cause changes in mood, memory, attention, reaction time, and vision. Studies in animals exposed to tetrachloroethylene have shown liver and kidney effects, and changes in brain chemistry.

Chemical Name	Comments
	Studies in humans suggest that exposure to tetrachloroethylene might lead to a higher risk of getting bladder cancer, multiple myeloma, or non-Hodgkin's lymphoma. In animals, tetrachloroethylene has been shown to cause cancers of the liver, kidney, and blood system. US EPA considers tetrachloroethylene likely to be carcinogenic to humans by all routes of exposure. The Department of Health and Human Services (DHHS) considers tetrachloroethylene to be reasonable anticipated to be a human carcinogen. IARC: There is limited evidence in humans for the carcinogenicity of tetrachloroethylene. Positive associations have been observed for cancer of the bladder. There is sufficient evidence in experimental animals for the carcinogenicity of tetrachloroethylene. Tetrachloroethylene is probably carcinogenic to humans (Group 2A). OSHA: Tetrachloroethylene is not listed as carcinogenic. NICNAS: Tetrachloroethylene has been assessed as a Priority Existing Chemical (PEC).
Tetra-Dioxins	See above for all dioxins identified.
Tetra-Furans	See above for all furans identified.
Toluene (methyl benzene) **irritant to skin, eyes, URT, and CNS** **toxic to CNS**	ATSDR: Toluene is a clear, colourless liquid with a distinctive smell. It is used extensively as a solvent. Toluene occurs naturally in crude oil. It is produced in the process of making gasoline and other fuels from crude oil and in making coke from coal. Toluene is used in making paints, paint thinners, fingernail polish, lacquers, adhesives, and rubber and in some printing and leather tanning processes. Toluene is also used in the manufacture of other chemicals, nylon, and plastics. It is also added to gasoline along with benzene and xylene to improve octane ratings. Toluene may affect the nervous system. Low to moderate levels can cause tiredness, confusion, weakness, drunken-type actions, memory loss, nausea, and loss of appetite. These symptoms usually disappear when exposure stops. Long-term daily inhalation exposure to toluene in the workplace may cause some hearing and colour vision loss. Repeatedly breathing toluene from glue or paint thinners may permanently damage the brain. The effects of toluene in animals are like those seen in humans. Studies in workers and animals exposed to toluene generally indicate that toluene is not carcinogenic. The EPA determined there is inadequate information to assess the carcinogenic

Chemical Name	Comments
	potential of toluene. The National Toxicology Program (NTP) has not considered the carcinogenic potential of toluene. IARC: There is *inadequate evidence* in humans for the carcinogenicity of toluene. There is *evidence suggesting lack of carcinogenicity* of toluene in experimental animals. Toluene is *not classifiable as to its carcinogenicity to humans (Group 3)*. OSHA: Toluene is listed as carcinogenic.
TPH >C10 - C12 (Aliphatic) **multiple acute and chronic health effects as for VOCs** **most severe toxicity due to benzene**	ATSDR: Total petroleum hydrocarbons (TPH) is a term used to describe a large family of several hundred chemical compounds that originally come from crude oil. Crude oil is used to make petroleum products, which can contaminate the environment. Because there are so many different chemicals in crude oil and in other petroleum products, it is not practical to measure each one separately. However, it is useful to measure the total amount of TPH at a site. TPH is a mixture of chemicals, but they are all made mainly from hydrogen and carbon, called hydrocarbons. Scientists divide TPH into groups of petroleum hydrocarbons that act alike in soil or water. These groups are called petroleum hydrocarbon fractions. Each fraction contains many individual chemicals. Some chemicals that may be found in TPH are hexane, jet fuels, mineral oils, benzene, toluene, xylenes, naphthalene, and fluorene, as well as other petroleum products and gasoline components. However, it is likely that samples of TPH will contain only some, or a mixture, of these chemicals. Some of the TPH compounds can affect the central nervous system. One compound can cause headaches and dizziness at high levels in the air. Another compound can cause peripheral neuropathy consisting of numbness in the feet and legs. Other TPH compounds can cause effects on the blood, immune system, lungs, skin, and eyes. Animal studies have shown effects on the lungs, central nervous system, liver, and kidney from exposure to TPH compounds. Some TPH compounds have also been shown to affect reproduction and the developing foetus in animals. IARC: The International Agency for Research on Cancer (IARC) has determined that one TPH compound (benzene) is carcinogenic to humans. IARC has determined that other TPH compounds (benzo[a]pyrene and gasoline) are probably and possibly carcinogenic to humans. Most of the other TPH compounds are considered not to be classifiable by IARC.
TPH >C6 - C8 (Aliphatic)	See above for all TPH.

Chemical Name	Comments
TPH >C8 - C10 (Aliphatic)	See above for all TPH.
TPH >C8 - C10 (Aromatic)	See above for all TPH.
TPH C10 - C14	See above for all TPH.
TPH C10 - C16	See above for all TPH.
TPH C10 - C36 (Sum of total)	See above for all TPH.
TPH C10 - C40 (Sum of total)	See above for all TPH.
TPH C15 - C28	See above for all TPH.
TPH C15 - C36 (Sum of total)	See above for all TPH.
TPH C16 - C34	See above for all TPH.
TPH C29 - C36	See above for all TPH.
TPH C34 - C40	See above for all TPH.
TPH C6 - C10	See above for all TPH.
TPH C6 - C8 (Aromatic)	See above for all TPH.
TPH C6 - C9	See above for all TPH.
TPH C6 - C9 ALIPHATIC	See above for all TPH.
Trans-1,3-dichloropropene **irritant to skin, eyes, URT, and CNS**	ATSDR: There are five different types (or isomers) of dichloropropene molecules: 1,1-dichloropropene; 1,2-dichloropropene; 1,3-dichloropropene; 2,3-dichloropropene; and 3,3-dichloropropene. 1,3-Dichloropropene is a colourless liquid with a sweet smell. It is used mainly in farming as a pesticide. Much less is known about the other dichloropropenes. 2,3-Dichloropropene is used in industry to make other chemicals. No uses were found for 1,1-, 1,2-, or 3,3-dichloropropene. Because 1,3-dichloropropene is produced and used in much higher amounts than the other isomers and because it is released to the environment as a pesticide, most of the data available are for 1,3-dichloropropene. Therefore, the focus of this summary is the 1,3-dichloropropene isomer.

Chemical Name	Comments
	Most of the 1,3- and 2,3-dichloropropene that is inhaled or ingested will rapidly enter the bloodstream. Rats and mice that inhaled 1,3-dichloropropene or 2,3-dichloropropene repeatedly had damage to the lining of the nose. Damage to the urinary bladder and anaemia were also seen in animals inhaling 1,3-dichloropropene for a long time. Damage to the stomach lining and anaemia were seen in animals orally exposed to 1,3-dichloropropene. Skin and eye irritation are seen in animals after 1,3-dichloropropene gets on their skin or in their eyes. A few workers who had skin contact with pesticides containing 1,3-dichloropropene developed blisters and an allergic reaction on their skin. The US Department of Health and Human Services (DHHS) has determined that 1,3-dichloropropene may reasonably be anticipated to be a carcinogen. The EPA has classified 1,3-dichloropropene as a probable human carcinogen. IARC: No epidemiological data relevant to the carcinogenicity of 1,3-dichloropropene were available. There is sufficient evidence in experimental animals for the carcinogenicity of mixed isomers of 1,3-dichloropropene (technical grade). 1,3-Dichloropropene (technical-grade) is possibly carcinogenic to humans (Group 2B). OSHA: 1,3-Dichloropropene and dichloropropenes are not listed as carcinogenic.
Trichloroethylene **toxic to skin, heart, liver, kidneys, and CNS** **causes kidney and other cancers in experimental animals** **Group 2A probably carcinogenic**	ATSDR: Trichloroethylene is a colourless, volatile liquid. Liquid trichloroethylene evaporates quickly into the air. It is non-flammable and has a sweet odour. The two major uses of trichloroethylene are as a solvent to remove grease from metal parts and as a chemical that is used to make other chemicals, especially the refrigerant, HFC-134a. Trichloroethylene was once used as an anaesthetic for surgery. Exposure to moderate amounts of trichloroethylene may cause headaches, dizziness, and sleepiness; large amounts may cause coma and death. Eating or breathing high levels of trichloroethylene may damage some of the nerves in the face. Exposure to high levels can also result in changes in the rhythm of the heartbeat, liver damage, and evidence of kidney damage. Skin contact with concentrated solutions of trichloroethylene can cause skin rashes. There is strong evidence that trichloroethylene can cause kidney cancer in people and some evidence for trichloroethylene-induced liver cancer and malignant lymphoma. Lifetime exposure to trichloroethylene resulted in increased liver cancer in mice and increased kidney cancer and testicular cancer in rats.

Chemical Name	Comments
	The US National Toxicology Program (NTP) has determined that trichloroethylene is a known human carcinogen. The EPA has determined that trichloroethylene is carcinogenic to humans. IARC: There is *limited evidence* in humans for the carcinogenicity of trichloroethylene. There is *sufficient evidence* in experimental animals for the carcinogenicity of trichloroethylene. Trichloroethylene is *probably carcinogenic to humans (Group 2A)*. OSHA: Trichloroethylene is listed as carcinogenic. NICNAS: Trichloroethylene has been assessed as a Priority Existing Chemical (PEC).
Trihalomethanes **toxicity as for chloroform**	PubChem: Trihalomethanes (THMs) are a group of organic chemicals that often occur in drinking water as a result of chlorine treatment for disinfectant purposes and, therefore, are also known as "disinfection byproducts" or DBPs. THMs are formed when chlorine reacts with naturally occurring organic material found in water such as decaying vegetation. Typically, the following four THMs are found as a result of chlorination: trichloromethane (chloroform), bromodichloromethane (BDCM), dibromochloromethane (DBCM), tribromomethane (bromoform). Untreated or raw water rarely contains THMs in significant concentrations. Since chloroform is the THM found in highest concentrations and about which the most is known, the bulk of the information contained in this summary will pertain to chloroform. ATSDR: Chloroform is a colourless liquid with a pleasant, non-irritating odour and a slightly sweet taste. It will burn only when it reaches very high temperatures. In the past, chloroform was used as an inhaled anaesthetic during surgery. Today, chloroform is used to make other chemicals and can also be formed in small amounts when chlorine is added to water. Other names for chloroform are trichloromethane and methyl trichloride. Breathing about 900 parts of chloroform per million parts of air (900 ppm) for a short time can cause dizziness, fatigue, and headache. Breathing air, eating food, or drinking water containing high levels of chloroform for long periods of time may damage the liver and kidneys. Large amounts of chloroform can cause sores when chloroform touches the skin. Animal studies have shown that miscarriages occurred in rats and mice that breathed air containing 30 to 300 ppm chloroform during pregnancy and also in rats that ate chloroform during pregnancy. Offspring of rats and mice that breathed chloroform during pregnancy had birth defects. Abnormal sperm were found in mice that breathed air containing 400 ppm chloroform for a few days.

Chemical Name	Comments
	The US Department of Health and Human Services (DHHS) has determined that chloroform may reasonably be anticipated to be a carcinogen. Rats and mice that ate food or drank water with chloroform developed cancer of the liver and kidneys. IARC: There is inadequate evidence in humans for the carcinogenicity of chloroform. There is sufficient evidence in experimental animals for the carcinogenicity of chloroform. Chloroform is possibly carcinogenic to humans (Group 2B). OSHA: Chloroform is listed as carcinogenic.
Vanadium **toxic to lungs and GIT**	ATSDR: Vanadium is an element that occurs in nature as white-to-gray metal compounds and is often found as crystals. Pure vanadium has no smell. It usually combines with other elements such as oxygen, sodium, sulphur, or chloride. Vanadium and vanadium compounds can be found in the earth's crust and in rocks, some iron ores, and crude petroleum deposits. Vanadium is used in producing rust-resistant, spring, and high-speed tool steels. Vanadium pentoxide is used in ceramics, as a catalyst, and in the production of superconductive magnets. Vanadyl sulphate and sodium metavanadate have been used as dietary supplements. Exposure to high levels of vanadium pentoxide in air can result in lung damage. Nausea, mild diarrhoea, and stomach cramps have been reported in people who have been exposed to some vanadium compounds. A number of effects have been found in animals ingesting vanadium compounds including decreases in the number of red blood cells, increased blood pressure, and mild neurological effects. The US Department of Health and Human Services (DHHS) and EPA have not classified vanadium as to its human carcinogenicity. IARC: There is inadequate evidence in humans for the carcinogenicity of vanadium pentoxide. There is sufficient evidence in experimental animals for the carcinogenicity of vanadium pentoxide. Vanadium pentoxide is possibly carcinogenic to humans (Group 2B). OSHA: Vanadium, metal and carbide are listed as carcinogenic.
Vinyl chloride **toxic to liver, CNS, and peripheral circulation**	ATSDR: Vinyl chloride is a colourless gas. It burns easily, and it is not stable at high temperatures. It has a mild, sweet odour. It is a manufactured substance that does not occur naturally. It can be formed when other substances such as trichloroethane, trichloroethylene, and tetrachloroethylene are broken down. Vinyl chloride is used to make polyvinyl chloride (PVC). PVC is used to

Chemical Name	Comments
causes liver cancer **Group 1 carcinogen**	make a variety of plastic products, including pipes, wire and cable coatings, and packaging materials. Vinyl chloride is also known as chloroethene, chloroethylene, and ethylene monochloride. Inhalation of high levels of vinyl chloride can cause dizziness and lethargy, with coma and death at very high concentrations. Some people who have breathed vinyl chloride for several years have changes in the structure of their livers. People are more likely to develop these changes if they breathe high levels of vinyl chloride. Some people who work with vinyl chloride have nerve damage and develop immune reactions. The lowest levels that produce liver changes, nerve damage, and immune reaction in people are not known. Some workers exposed to very high levels of vinyl chloride have problems with the blood flow in their hands. Their fingers turn white and hurt when they go into the cold – Raynaud's phenomenon. The effects of drinking high levels of vinyl chloride are unknown. If spilt on the skin, vinyl chloride will cause numbness, redness, and blisters. Animal studies have shown that long-term exposure to vinyl chloride can damage the sperm and testes. The US Department of Health and Human Services (DHHS) has determined that vinyl chloride is a known carcinogen. Studies in workers who have breathed vinyl chloride over many years showed an increased risk of liver, brain, lung cancer, and some cancers of the blood have also been observed in workers. IARC: There is sufficient evidence in humans for the carcinogenicity of vinyl chloride. Vinyl chloride causes angiosarcoma of the liver, and hepatocellular carcinoma. There is sufficient evidence in experimental animals for the carcinogenicity of vinyl chloride. There is sufficient evidence in experimental animals for the carcinogenicity of chloroethylene oxide. There is strong evidence that the carcinogenicity of vinyl chloride operates by a genotoxic mechanism that involves metabolic activation to reactive metabolites, binding of the metabolites to DNA, promutagenic action of these adducts leading to mutations in proto-oncogenes and tumour-suppressor genes. Many of these key events identified in experimental animals have also been demonstrated in humans. Vinyl chloride is carcinogenic to humans (Group 1). OSHA: Vinyl chloride is listed as carcinogenic.
Xylene (m & p)	See meta & para xylene above.
Xylene (o)	See meta & para xylene above.
Xylene Total	See meta & para xylene above.

9 REFERENCES

[i] Norris JC, Ballantyne B. (1993). Combustion toxicology. In General and Applied Toxicology, eds B Ballantyne, T Mars, P Turner, Vol 3, pp 1309-1327. London; MacMillan.

[ii] Atlas EL, Donnelly KL, Giam CS et al (1985). Chemical and Biological Characterization of Emissions from a Fireperson Training Facility. *Am Ind Hyg Assoc J,* **46:** 532-540.

[iii] Brandt-Rauf PW, Fallon LF, Tarantini T et al (1988). Health hazards of fire fighters: exposure assessment. *Br J Ind Med*, **45:** 606-612.

[iv] Austin CC, Wang D, Ecobichon DJ et al (2001). Characterization of volatile organic compounds in smoke at municipal structural fires. *J Toxicol Environ Health,* **63:** 437-438.

[v] Kirk KM & Logan MB (2015). Firefighting instructors' exposures to polycyclic aromatic hydrocarbons during live fire training scenarios. *J Occup Environ Hyg,* **12:** 227-234.

[G]Greven F, Kerstjens HAM, Duijn F et al (2009). Respiratory effects in the aftermath of a major fire in a chemical waste depot. *Scand J Work Environ Health,* **35:** 368-375.

[vii] Le Masters GK, Genaidy AM, Succop P et al (2006). Cancer Risk Among Firefighters: A Review and Meta-analysis of 32 Studies, *J Occup Environ Med,* **48:** 1189-1202.

[viii] Youakim S (2006). Risk of cancer among firefighters: a quantitative review of selected malignancies. *Arch Environ Occup Health,* **61:** 223-231.

[ix] Daniels RD, Bertke S, Dahm MM, et al (2015). Exposure–response relationships for select cancer and non-cancer health outcomes in a cohort of US firefighters from San Francisco, Chicago and Philadelphia (1950–2009). *Occup Environ Med,* **72:** 699-706.

[x]Glass DC, Pircher S, Del Monaco A et al (2016). Mortality and cancer incidence in a cohort of male paid Australian firefighters. *Occup Environ Med,* **73:** 761-771.