7 June 2012

INDEPENDENT FISKVILLE INVESTIGATION

Health Hazards of Fuels and Possible Combustion Products

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REPORT

Report Number. 117613201-011-R-Rev0

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Table of Contents

TABLES

FIGURES

APPENDICES

1.0 INTRODUCTION

Golder Associates Pty Ltd (Golder Associates) was engaged to assist Professor Rob Joy in his Independent Investigation into the CFA Facility at Fiskville (1971 – 1999 (the Independent Investigation).

This report was undertaken based on our proposal (P17613413-001-P-Rev0) dated 13 December 2011 and addendum letters (117613201-005-L-Rev0, 117613201-008-L-Rev0) dated 1 March 2012 and 18 April 2012 respectively.

The preliminary site assessment report (Golder 2012) provides a description of the site including both current and historical uses and activities at the site. This report is intended to provide contextual hazard toxicological information to the Fiskville Investigation team regarding substances potentially present at fire training facilities. It is not intended to provide specific Fiskville related advice regarding health outcomes nor exposures.

Your attention is drawn to the document - "Limitations", which is included in Appendix C of this report. The statements presented in this document are intended to advise you of what your realistic expectations of this report should be. The document is not intended to reduce the level of responsibility accepted by Golder Associates, but rather to ensure that all parties who may rely on this report are aware of the responsibilities each assumes in so doing.

2.0 OBJECTIVES

The objectives of this report are to provide:

- A summary of information on agents used as fuels and other materials used in training at the Fiskville facility.
- A summary of the various types of combustion that can occur and the types of combustion products that may arise.
- The chronic health hazards associated with exposure to fuels and combustion products such as particulate matter, and gaseous components such as volatile organics, nitrogen dioxide, and carbon oxides. Chronic adverse health outcomes reflect long-term exposure and in this report the focus has been on the hazard of cancer.
- A discussion of the typical activities likely to occur at a fire training facility and the associated exposure routes.

The present report is intended to provide contextual hazard information to support the current investigation. It does not seek to reconstruct exposures at Fiskville nor to consider the likelihood of health conditions arising as a consequence of activities at Fiskville.

3.0 GENERIC APPROACH

3.1 Identifying Substances Potentially Present at Fiskville

This report considers substances that plausibly may have been used or emitted during fire training activities at the site. The list of substances considered in this report is based on;

- Information provided by the Independent Fiskville Investigation (IFI 2012, Appendix 2) team regarding substances (chemicals and articles) potentially used as a fuel or as fire fighting agents during the period under investigation (i.e. 1970's to the present day).
- Information on substances identified as present[1] in soil, surface or groundwater during the Preliminary Site Investigation
- Information on possible products of incomplete combustion.

Details of the process for selecting Substances Potentially Present (SPP) are provided in Appendix A and the process is illustrated in Figure 1:

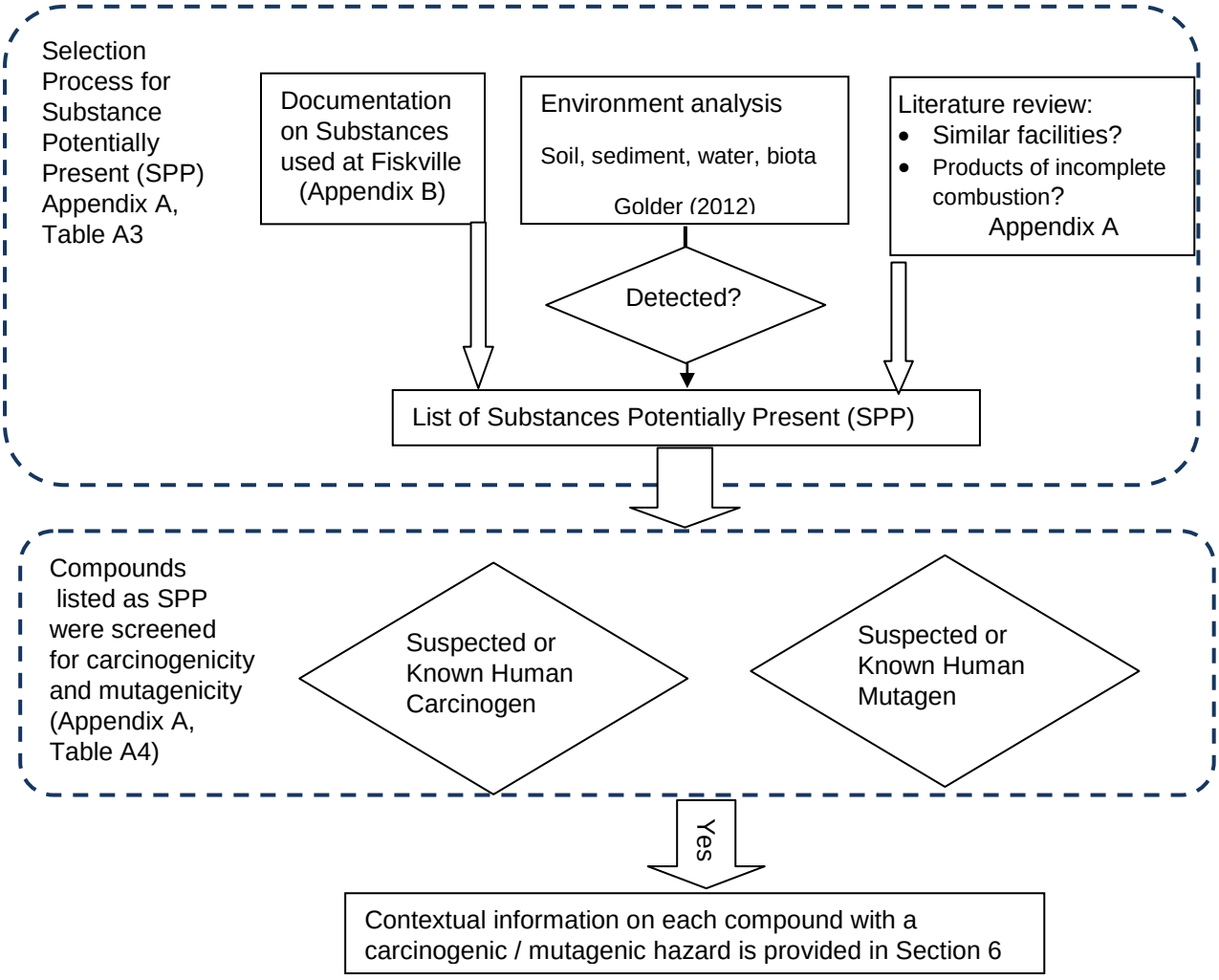

Figure 1: Decision logic for selection and screening Substances Potentially Present (SPP)

[1] Based on concentrations being above the limit of reporting

3.2 Health Hazard Information

The process for screening carcinogenic chemicals is described in Appendix A. The three sources of information as detailed in Section 3.1 were combined which included the Master List of Chemicals supplied by IFI (2012), chemicals identified from preliminary site investigations and information obtained from the literature regarding products of incomplete combustion.

The health hazard assessment was a qualitative appraisal of this list of SPP against the chronic health hazard data for cancer. The SPP reflect substances emitted from fuels and combustion products such as particulate matter, nitrogen dioxide, carbon oxides (carbon monoxide), benzene and polycyclic aromatic hydrocarbons and are commonly associated with emissions from incomplete combustion sources.

The SPP list of chemicals was screened to identify substances that are known or presumed human cancer-causing substances or carcinogens, and/or known or presumed human mutagenic chemicals. Mutagenic chemicals are also considered as these reflect a potential for the substance to act as a carcinogen

The screening was undertaken based on regulatory agency reviews relevant to the carcinogenicity and/or mutagenicity classification/categorisation of a chemical (or mixture of chemicals). Literature reviews and authority evaluations such as the following were used.

- IARC Monographs on the Evaluation of Carcinogenic Risks to Humans and Supplements.
- World Health Organization (WHO) reports such as those published by the International Programme on Chemical Safety (IPCS), relevant expert committees, WHO Drinking Water Guidelines and WHO Air Guidelines.
- Australian Hazardous Substances Information Service (HSIS) produced by Safework Australia.
- Reports from Australian federal authorities responsible for chemical assessment (Office of Chemical Safety, Department of Health and Aging (National Industrial Chemicals Notification and Assessment Scheme), and Australian Pesticides and Veterinary Medicines Authority (Agricultural and Veterinary Chemicals Notification and Assessment Scheme).
- European Chemicals Agency (ECHA) agreed classification according to the European Classification, Labelling and Packaging Regulations for Hazardous Substances.
- EU Directive Dangerous Substances and Preparations, Annex 1 26th Adaption European Commission (2000). Directive 67\548|EEC.
- Toxicological Profiles for Chemical Substances, Agency for Toxic substances and Disease Registry (ATSDR), US Department of Health and Human Services.
- US EPA Toxicology reports produced for various US EPA sections including those for the Integrated Risk Information System (IRIS)

Cancer is largely attributable to the impact of exogenous environmental factors, a wide spectrum of circumstances of exposure to particular carcinogens having been described. As a consequence of this complexity the presence of a chemical on a list of carcinogens often requires additional qualification to consider the circumstances under which a chemical may cause cancer. For example, exposure to benzene may occur in the workplace, or from inhalation as a consequence of atmospheric pollution by engine exhaust or from ingestion of benzene (a water soluble chemical) present in bottled water.

These various exposures may each warrant further consideration in a hazard assessment. Hazard identification, however, does not provide information relating to the exposure contribution a person may have from various exposure pathways such as ingestion, dermal contact and inhalation. This exposure apportionment will vary according to the differing contexts in which exposure to a carcinogen may be known to occur.

On this basis, substances identified as carcinogens in the hazard assessment require brief additional contextual information and this is provided in Section 4. The information considers the circumstances under which the particular chemical may cause cancer but does not discuss potential risks due to exposure of chemicals at CFA, Fiskville

The information for each chemical is derived from international and overseas expert reviews including the International Agency for Research on Cancer, World Health Organisation and US Department of Health and Human Services.

4.0 EXPOSURE PATHWAYS

There are a number of theoretical exposure pathways, summarised in Table 1through which workers or members of the general public may be exposed to fuels, foams or products of combustion at fire training facilities. Table 1 considers the frequency of exposure as either; frequent, intermittent or infrequent. Frequent exposure is defined as daily exposure for a prolonged period of time (several months to years). Intermittent exposure relates to repeated yet infrequent exposure over a prolonged period of time. Infrequent exposure relates to one-off exposures or accidental exposure.

As described in Table 1 the theoretical exposure pathways associated with fire training facilities are considered to be either of an intermittent or infrequent frequency.

In general the following scenarios and exposures are considered of relevance to fire training facilities.

- The primary sources of exposure relate to occupational exposures including handling of flammable materials, foams used in fire training exercises, reticulated fire fighting water and smoke generated during fire fighting training.
- Key people of interest include fire training supervisors and site workers involved in maintaining fire training facilities. Such workers are likely to be involved in the receive, storage, and transfer of flammable fuels. Such activities are likely to occur on a regular (daily) basis however, contact with chemicals will generally only occur during transfer operations or spills and thus result in intermittent exposure.
- During fire training, supervisors and trainees are exposed to smoke although the degree is dependent on local practices, and conditions.

In addition to direct contact with the fuels, foams or products of combustion, people may be exposed to chemicals of interest via the deposition of windblown particulate matter from the combustion of fuels. Spills may contaminate soil and surface water and permeate to groundwater,

Deposition or spills may result in contamination of soil, vegetation used as food, surface waters, and surfaces used for collection of rain water. The deposition of contaminants on soil and other surfaces over a long period with environmental distribution may also result in incidental ingestion. It should be noted that this exposure pathway is likely to be more significant for young children due to their specific behaviours. Children come into intimate contact with soil compared with adults as they may be crawlers, wash their hands less frequently and exhibit more frequent hand-to-mouth activities.

Another potential exposure pathway of interest is ingestion of fuels or fire fighting chemicals present in aerosols of contaminated fire fighting water.

In considering the potential exposure pathways in Table 1, inhalation exposure is considered the most likely scenario to result in frequent exposures of workers and trainees.

The health effects of key components of smoke and the fuels or fire fighting chemicals are discussed in detail in Section 6.0.

Table 1: Potential exposure pathways for fire training facilities

Exposure medium Direct (Primary Pathways)	Cause	Exposure route	Receptor	Exposure activities	Frequency
Chemicals used as fuel for fire fighting operation	Handling and transfer operations during preparation of fuels, flammable liquids and other chemicals	Inhalation of vapour and skin contact with liquid during spills	Site workers.	Receiving, storage, handling of flammable liquids	Intermittent
Chemicals used for fire fighting	Fighting fires	Incidental ingestion, inhalation and dermal contact	Fire training instructors and Trainees	Fire fighting training	Intermittent
Air	Fire generated for training purposes	Inhalation	Site workers Fire Training Instructors Trainees Members of the general public	Being outside in close proximity to training area. Potential exposure to plumes of smoke or smoke fall out. Fire fighting. Exposed to smoke plume. Fire fighting. Exposed to smoke plume. People within the zone of plumes including nearby residents, playing sport at nearby sporting grounds, attending nearby schools ,	Intermittent Intermittent Infrequent Infrequent
Surface soil	Transfer operations Spills Particulate deposition	Incidental ingestion, dermal contact and inhalation of dust	General maintenance and landscaping staff General public (particularly children)	Gardening, maintenance work Playing in contaminated soil	Intermittent Infrequent
Sediment in waste water treatment systems.	Sedimentation of constituents in fire water	Ingestion Dermal Aerosol inhalation	General maintenance staff	Maintenance works	Infrequent
Water	Soluble or suspended components present in fire water	Dermal contact, inhalation /ingestion of inhalable aerosol droplets	Fire Training instructors and trainees. General maintenance staff	Fire fighting Maintenance works	Intermittent Infrequent

Exposure medium Direct (Primary Pathways)	Cause	Exposure route	Receptor	Exposure activities	Frequency
Exposure Medium Indirect (Secondary Pathways)					
Consumption of vegetables	Translocation from soil to plants and soil water to plants Particulate matter deposition onto plants	Ingestion	Nearby residents	Eating unwashed/washed vegetables grown in contaminated soil or exposed to contaminated dust	Infrequent
Surfaces	Transfer of particulate matter from surfaces to hands, dust generation	Incidental Ingestion, dermal uptake, dust inhalation of surface generated dusts	Children and adult residents and workers	Hand to mouth transfer of contaminated dust from outdoor equipment or indoor surfaces; dermal uptake through intact, occluded or abraded skin, dust inhalation	Infrequent

5.0 SELECTION OF SUBSTANCES POTENTIALLY PRESENT (SPP)

The process for selection of substances potentially present (SPP) has been described in Section 4 and presented in detail in Appendix A. Information to select SPP was drawn from three sources of information:

- Review of list of chemicals provided by IFI (2012). .
- Review of chemicals identified above the laboratory limit of reporting in the environmental preliminary site investigation undertaken by Golder Associates (2012). Although such chemicals were included as SPP it is unknown whether the presence of these substances is due to fire training activities, other site activities or are unrelated to site activities.
- Literature search considering previously reported chemical exposures from similar facilities, and potential products of incomplete combustion. The resulting compilation of compounds is entirely theoretical and does not consider the variables associated with the combustion process that may have occurred. Well ventilated fires (i.e. fires where there is an abundance of air and therefore combustion is most efficient) produce less products of incomplete combustion (by-products) compared to ventilation limited fires (i.e. indoor fires where air supply is limited).

6.0 CONTEXTUAL HEALTH HAZARD INFORMATION

6.1 Toxicity of smoke

Smoke consists of a mixture of gases, liquid droplets and solid particles representing the decomposition and combustion products from fires. Toxic products from fires can be considered to comprise three categories being asphyxiants, irritants and organic by-products (HPA 2002, 2012).

Asphyxiants present a major acute hazard where individuals are trapped within a plume of smoke. Simple asphyxiants displace oxygen causing hypoxia while chemical asphyxiants interfere with oxygen delivery and metabolism and include carbon monoxide (CO) and hydrogen cyanide (HCN) (HPA 2002, 2012).

Irritants may cause immediate reactions to the respiratory tract and/or eyes and skin. The severity of effect depends on the substance involved and their respective concentration. Irritant effects can be due to the cumulative action of a mixture of substances. Inorganic irritants include nitrogen or sulphur oxides, hydrogen chlorides and fluorides. Organic irritants include acrolein, formaldehyde and related aldehydes (HPA 2002, 2012).Organic by-products may include any chemical present in bulk or which is toxic at low concentrations and could be released in the fire plume with resultant critical short (acute) and long term (chronic) consequences. Creosote is a mixture of organic by-products that may be produced from incomplete combustion of wood (or coal) (HPA 2002, 2012).

The International Agency for Research on Cancer (IARC, 2010) concluded that occupational exposure as a firefighter is possibly carcinogenic to humans (Group 2B) based on limited evidence in humans from occupational epidemiological studies. IARC commented that aalthough increases in various cancers in fire-fighters compared with the general population have been noted in several studies, consistent patterns are difficult to discern due to the large variations in reported exposures. The strongest associations were identified with testicular cancer, prostate cancer, and non-Hodgkin's lymphoma (IARC, 2010). Some studies have correlated cumulative frequency of fires attended with the mean annual reduction in pulmonary function (IARC, 2010). Years of employment has not been found to correlate with exposure to combustion products or related adverse health effects such as decline in pulmonary function or airway responsiveness (IARC, 2010).

6.2 Particulate matter (PM)

Combustion-derived particulates are comprised of metals (and metal oxides), black or elemental carbon, primary and secondary organic compounds (e.g. PAHs) as well as sulphates, nitrates, ammonium and hydrogen ions (US EPA, 2004).

There is general agreement in the scientific literature that there is a dose-response relationship (with no indication of a threshold) between respirable particulate matter (expressed as PM_{10} or $PM_{2.5}$) and particular health endpoints. In particular, toxicological, clinical and epidemiological studies consistently show decrements in cardio-pulmonary outcomes following both short (acute) and long-term (chronic) exposure.

The strongest evidence comes from epidemiological studies conducted in large urban population areas where an association between fine PM (defined as material with a diameter of less than 2.5 microns.) and health effects has been reported. A recent US EPA review (2010) concluded that there was causal evidence linking $PM_{2.5}$ exposure to cardiovascular effects, and daily and long-term mortality. Furthermore, the US EPA concluded that there was a likely causal association with adverse respiratory effects following both short-term (24 hour) and long-term (annual average) exposure.

With regard to carcinogenicity the US EPA (2010) concluded that there was suggestive evidence for an association with fine particulate matter. Epidemiologic studies have shown a consistent positive association between $PM_{2.5}$ and lung cancer mortality but have not reported associations with lung cancer incidence. A number of studies have concluded that diesel exhaust particulates can be mutagenic and genotoxic. IARC (1998) have evaluated the evidence for the carcinogenicity of diesel exhausts as probably carcinogenic to humans (IARC Group 2A) based on:

- Limited evidence from epidemiology studies with workers (bus company and dock-side workers) and
- Sufficient evidence from animal studies (rats, mice Syrian hamsters and monkeys) which have shown exposure dependent increases in the frequency of benign and malignant lung tumours.

The IARC summary for diesel exhausts reports that the association is significant following prolonged exposure (multiple years).

6.3 Nitrogen dioxide (NO_2)

In recent years the health effects of ambient air exposure to nitrogen dioxide (NO_2) have been well studied and reviewed by national and international agencies (US EPA 2008, WHO 1997, 2000a, 2000b, 2006; NEPM, 1998, NEPC 2010; OEHHA, 1999). The critical health outcomes include respiratory disease and associated symptoms, and changes in lung function. Individuals with asthma and other chronic lung disease and cardiovascular diseases are recognised as being particularly vulnerable. Other susceptible populations include infants, children and the elderly (>65 years of age) (NEPC, 2010).

Only very high concentrations of NO_2 (approximately 2,000 µg/m^3 (~1,050 ppb)) affect breathing in healthy people[2]. However small changes in lung function (< 5%) and changes in airway responsiveness have been reported in several studies of sensitive asthmatics or the elderly exposed to concentrations as low as 375-575 µg/m^3 (~200-300 ppb) over 20 minutes to 4 hours (Bauer et al., 1986; Bylin et al., 1988; Roger et al., 1985a & b; Morrow et al., 1992; Strand et al., 1996, 1997, as cited in Streeton 1997 and US EPA 2008). These levels represent a clear low-observed-effect level (LOEL) for NO_2 based on increased responsiveness in mild asthmatics to broncho-constrictors or in subjects with chronic obstructive pulmonary disease (COPD).

[2] Conversions are performed at STP (0°C and 101.7kPa) consistent with data provided by PAE (2011). The conversion for NO_2 is: ppb = µg/m^3 x 0.49; µg/m^3 = ppb x 2.05. Many unit conversions in this section have been rounded.

6.4 Carbon monoxide (CO)

Carbon Monoxide binds with haemoglobin to form carboxyhaemoglobin (COHb), and when formation of this compound is high enough the oxygen carrying capacity of the blood decreases to such an extent that tissues highly dependent on oxygen are not able to function properly. Thus the toxic effects of CO become evident in organs and tissues with high oxygen consumption such as the brain, the heart, exercising skeletal muscle and the developing foetus. This is especially the case when tissue oxygen utilisation is already compromised such as in people with ischaemic heart disease (WHO 2000a).

As COHb stays longer in the foetus than in the pregnant mother the foetus is more vulnerable to the effects of CO than the mother (WHO 2000a).

Asthmatics are not more sensitive to the effects of CO than are healthy people and COHb levels in the latter are normally about 0.4–0.7%. The World Health Organisation recommends that a COHb level of 2.5% should not be exceeded and have accordingly set an ambient air quality guideline based on this endpoint. . The Australian NEPC adopted the WHO recommendation (Streeton 1997). The California EPA estimated the no observed effect level (NOEL) to be 1.1-1.3% COHb and have set their guideline on the amount of CO that does not lead to COHb blood levels greater than those associated with this NOEL (OEHHA 1999).

6.5 Benzene

Knowledge of the health effects of benzene come from studies of human populations exposed to the chemical in their workplace and from observations of laboratory animals exposed for defined periods to known levels of benzene. The most relevant information regarding effects of long-term exposure to benzene is from studies of workers employed in industries that made or used benzene some 30 to 60 years ago. In this period, exposure to benzene in workplace air was far greater than the current levels. Unfortunately estimates of past exposure of workers in these studies are only approximations of the actual exposures which may have occurred and in many cases the exposures are likely to have been underestimated, thus the likely risks to workers at any given benzene concentration is probably overestimated (NICNAS 2001, Goldstein & Witz 2009).

Very high levels of exposure (well over 16 mg/m^3) on repeated occasions have led to the development of severe and sometimes fatal damage to the blood forming elements of the bone marrow. This bone marrow toxicity disrupts normal blood production and can lead to a condition known as aplastic anaemia and cause a decrease in important cellular components of blood (a condition known as cytopaenia) (ATSDR 2007, NICNAS 2001, Goldstein & Witz 2009). .

The effect of long-term exposure to benzene of most concern is leukaemia, particularly those leukaemias known collectively as the non-lymphocytic leukaemias. A series of studies of groups of workers in the synthetic rubber and petroleum industries have confirmed the increased risk of leukaemia in some of these workers. Furthermore, animal studies involving exposures of rats and mice to inhaled concentrations of 32 mg/m^3 or more for most of their lifetimes have shown adverse benzene-related effects consistent with the observations following human exposures.

The mechanism by which benzene causes leukaemia has been investigated in special studies in both whole animals and cultured animal cells. These studies are designed to determine whether a chemical can interfere with the expression of genetic material. Benzene has been shown to adversely affect the genetic material of cells (i.e. it is genotoxic). The genotoxic action of a chemical is generally taken to indicate the possibility of it causing malignant disease, even with small exposures (ATSDR 2007, NICNAS 2001, Goldstein & Witz 2009).

International, national and overseas regulatory agencies (e.g. World Health Organization, US Department of Health and Human Services, American Conference of Governmental Industrial Hygienists and the National Industrial Chemical and Notification Scheme in Australia have reviewed the available evidence provided in published reports of studies in workers exposed to benzene. These regulatory agencies consider a study of workers from an American rubber manufacturing plant between 1940 and 1965 and a larger study of American chemical workers exposed to benzene between 1946 and 1975 as the most reliable. Both these studies showed an increased risk of non-lymphocytic leukaemias in workers with the highest benzene

exposures, estimated to have been greater than 200 ppm .years (that is equivalent to 10 ppm for 20 years or any other mathematical combination giving 200 ppm.years). Excess numbers of cases of leukaemia occurred with exposure to lower concentrations however the possibility that this was due to chance could not be ruled out. In examining the data from these two studies, regulatory agencies concluded the risk of leukaemia in workers was not detectable when average exposures over a working lifetime were around 0.5 to 1 ppm (1.6-3.2 mg/m^3). Note this is because studies of feasible size have low statistical power, i.e. an inability of the study to measure small increases in risk, even if the risk exists arising from insufficient study population size to observe an effect (WHO 2000a, ATSDR 2007, NICNAS 2001, Goldstein & Witz 2009).

6.6 Polycyclic Aromatic Hydrocarbons (PAHs)

There are more than 100 different PAHs which generally occur as part of complex mixtures, thus environmental exposures are not represented by single PAH compounds. Benzo[a]pyrene is the surrogate compound against which the risk assessment for PAH is conducted (see Section 3.0), for this substance there is an extensive toxicological database. In a total of 65 studies the most notable health effect in chronic studies performed on animals is cancer and this has occurred via every exposure route (n = 5) and every species tested (n = 7) (WHO, 1998). It is therefore a reasonable assumption that sufficient exposure of humans to PAHs by any exposure route is associated with risk of induction of cancer and cellular proliferation. Benzo(*a*)pyrene and a number of other PAHs are genotoxic in a large number and wide variety of *in vitro* and *in vivo* tests that have the capacity for metabolic activation of the substance (ATSDR 1995, WHO 1998). Animal studies demonstrate that PAHs including benzo[*a*]pyrene tend to affect proliferating tissues such as bone marrow, lymphoid organs, gonads, and intestinal epithelium (ATSDR 1995).

Benzo[*a*]pyrene and other PAHs are not construed as being acutely toxic to humans or animals. Only naphthalene, a lower molecular weight and volatile lPAH, has a known human lethal dose (> 5,000 mg for an adult) based on information from case reports of accidental poisonings following ingestion of moth repellent. Short term studies of high applications of benzo[*a*]pyrene in animals have resulted in hyperkeratosis and induction of contact hypersensitivity in guinea pigs and mice. Benzo[*a*]pyrene is embryotoxic, teratogenic and is a reproductive toxin in mice and rats but at doses higher than those associated with cancer induction. It is evident from high dose animal carcinogenicity feeding studies that PAHs mixtures cause dose-related increases in a wide variety of tumours including liver, lung, forestomach and small intestine whereas oral benzo(*a*)pyrene intake has resulted only in forestomach tumours (Culp et al. 1998). This suggests that tumours in locations other than the forestomach may be due to other components contained in the PAH mixture rather than benzo(*a*)pyrene.

Human exposure occurs to mixtures of PAHs, with benzo[*a*]pyrene recognised as one of the more active carcinogenic components. Epidemiological studies in coke oven workers, asphalt workers, aluminium smelter pot room workers have all shown dose related increased risks of lung cancer, with PAHs and benzo[*a*]pyrene being the putative carcinogens. Occupational exposure to PAHs in tars and paraffins is reported to induce skin cancer, but the incidence of this is now much lower due to better personal hygiene in occupational settings (WHO 1998).

Overall, the animal toxicity and human health information for PAHs indicates they have low acute toxicity and that cancer is the most sensitive health end point. Furthermore benzo(*a*)pyrene is a PAH considered to be amongst the most potent of the carcinogenic PAHs and is often used as an indicator of the carcinogenic potency of PAH mixtures.

The carcinogenicity of benzo[*a*]pyrene, and other PAHs, is dependent upon it being metabolically transformed into reactive metabolites that interact with DNA. Within an individual (and thus population) the degree of carcinogenicity associated with benzo[*a*]pyrene at any given level of exposure is dependent on the individual's constitutive activity of the enzyme systems involved with activation/deactivation and the extent to which these systems can be induced by environmental factors such as diet, and cigarette smoke. .

6.7 Dioxins

During the investigations undertaken by Golder (2012), dioxins were measured at levels near to the limit of reporting. The tetrachlorinated dibenzo-p-dioxin with chlorine atoms attached in the 2, 3, 7 and 8 positions (2,3,7,8-TCDD usually simplified to TCDD) is known to possess the highest toxic potency and toxic effects of this congener have been the most studied. For this reason the toxicity of each dioxin congener is compared to the toxicity of TCDD using the International Toxic Equivalents (TEQ). The TEQ scheme assigns each isomer a specific Toxic Equivalency Factor (TEF) relative to the most toxic isomer (TCDD (2,3,7,8-TCDD) - which is given a value of one).

Dioxins (expressed as TEQ) were detected in soil and sediment. Although the levels detected were below the relevant health based screening value a brief description on the toxicity of dioxins has been included.

Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are collectively called ‘dioxins’. Co-planar polychlorinated biphenyls (co-planar PCBs) possess toxicity similar to that of dioxins and are called ‘dioxin-like’ compounds. Dioxin and furan molecules consist of two benzene rings joined together by oxygen atom(s) with various amounts of chlorine or hydrogen atoms attached in the numbered positions of Figure 10.1. There are 75 kinds of PCDDs, 135 PCDFs and more than 10 co-planar PCBs. The different types of dioxins are called congeners (OCS 2005).

Adverse effects reported in animals following administration of dioxins include immunotoxicity, endometriosis in Rhesus monkeys and developmental and behavioural effects in offspring of treated monkeys. Developmental effects have also been observed in treated rats. The most sensitive effect, i.e. the one occurring at the lowest dioxin exposure, was decreased sperm production and sexual feminisation in male off-spring of exposed rats. TCDD is carcinogenic in several species, but does not damage DNA (OCS 2005).

In humans the data, mostly from relatively highly exposed populations, report a variety of subtle biochemical responses following exposure. These include induction of hepatic enzymes, changes in hormonal levels and reduced glucose tolerance. However, these effects are of unknown clinical significance, and may or may not indicate a toxic response or potential for toxic response. Of the many health effects evaluated in exposed adult populations, many were transient and not observed when exposure ceased. Human studies have failed to provide compelling evidence for endometriosis. The most consistently observed effect following high dose exposure is chloracne and other skin conditions. There is also some evidence that high paternal exposure to TCDD may be associated with the birth of more girls than boys. From animal cancer experiments with TCDD and occupational studies, plus an understanding of the plausibility of a common mechanism of action for animals and humans IARC has concluded TCDD is carcinogenic to humans (OCS 2005).

Data from animal and human studies provides information that there is a common mechanism of action for biochemical and toxicological effects. This information is used to predict the possibility of health effects in humans that have not been observed in human studies.

The International Agency for Research on cancer (IARC) concluded that TCDD is carcinogenic to humans based on limited evidence of an overall increase risk from all cancers in occupational studies of herbicide manufacturing workers involving heavy exposure to herbicides (IARC 1997). The human studies were supported by strong evidence for the carcinogenicity in animal carcinogenicity as well as animal mechanistic studies (IARC 1997).

6.8 Contextual information on substances classified as probable or known human carcinogens

The carcinogenic classification method used in this assessment is based on IARC which is a WHO regulatory agency and classification details are presented in Appendix A. Where data are limited, US EPA classification methods have been presented.

For substances designated by international or national regulatory authorities as known human carcinogens or probable human carcinogens the type of tumour following inhalation and a concise contextual summary is provided in Table 2. Generally the table highlights that causal relationships between exposure and cancer outcomes are generally due to relatively high occupational exposure over a prolonged duration of exposure.

Table 2: Contextual information on carcinogens

SCC	Cancer Classification.	Tumour types[1] (inhalation)	Key evidence in support of classification
Arsenic & Arsenic compounds	IARC 1	Lung	Increased incidence of lung cancer has been observed following cumulative exposure to inorganic arsenic in an occupational setting, especially in mining and copper smelting.
Cadmium	IARC 1	Lung	Excess mortality from lung cancer has been reported following occupational exposure to cadmium (e.g. cadmium processing and recovery plants), which was correlated with the duration of employment and intensity of exposure.
Chromium VI	IARC 1	Lung	In occupational settings where the highest exposure to chromium VI may occur, such as chromate production, chromate pigment manufacture and chromium plating, epidemiological studies of workers inhaling chrome fumes and dust have consistently shown excess risks for lung cancer.
Nickel	IARC 1	Lung and Nasal	Increased risks for lung and nasal cancers have been reported for workers exposed to high levels of nickel compounds, for example during high temperature oxidation of nickel matte and nickel copper matte, electrolytic refining, leaching processes at copper plants and hydrometallurgy.
Inorganic Lead	IARC 2A	Lung, Stomach and Kidney	The IARC classification is based on sufficient information from animal studies and limited information from human studies. Epidemiological studies found small increases in lung, stomach, and kidney cancer in occupational settings (e.g. battery workers and primary smelter workers). Epidemiological studies of the general population have also found a positive dose–response relationship between blood lead concentrations and lung cancer. All these studies have potential confounding factors implicated.
Ethanol	IARC 1	Not Applicable	Not Applicable

SCC	Cancer Classification.	Tumour types[1] (inhalation)	Key evidence in support of classification
Creosote	IARC 2A	Lung	An increased risk of lung cancer among gas and electricity workers exposed to creosote and workers who used creosote for the treatment of wood. The risk of lung cancer was related to increased exposure to creosote.
Formaldehyde (irritant)	IARC 1	Upper respiratory system	Excess deaths from nasopharyngeal cancer have been observed in industrial workers exposed to formaldehyde. Higher probability, level, and duration of exposure to formaldehyde in industrial workers is associated with higher risks of nasopharyngeal cancer.
2,4,6-Trichlorophenol	EPA B2	Not Available	The US EPA classification is based on oral animal studies alone as no human evidence was identified. An inhalation slope factor is calculated from oral animal data.
Silica (crystalline)	IARC 1	Lung	Evidence supporting the carcinogenicity of crystalline silica in the lung comes from analysis of numerous studies in a range of industrial settings, including ceramics, diatomaceous earth, ore mining, quarries, and sand and gravel yards. Evidence of an exposure-response relationship has been clearly demonstrated.
Sulphuric acid	IARC 1	Upper respiratory system	IARC has classified “occupational exposure to strong-inorganic-acid mists containing sulfuric acid” as carcinogenic (Group 1). A number of studies have found excess laryngeal cancer in workers occupationally exposed to sulfuric acid, for example in isopropanol manufacture, pickling operations in the steel industry and petrochemical plants.
Tetrachloroethylene	IARC2A	Upper respiratory system, Haematolymphatic organs, Cervix	There is evidence for consistently positive associations between exposure to tetrachloroethylene and the risks for oesophageal and cervical cancer and non-Hodgkin's lymphoma. The greatest exposure occurs via inhalation, and workers in dry cleaning and degreasing are the most heavily exposed.
Trichloroethylene	IARC 2A	Kidney, Liver, Haematolymphatic organs	Several epidemiological studies showed elevated risks for cancer of the liver and biliary tract and for non-Hodgkin's lymphoma, including workers exposed to trichloroethylene during maintenance of military aircraft and missiles.
1,3 Butadiene	IARC 1	Haematolymphatic organs	The epidemiological evidence from the styrene-butadiene and the butadiene-monomer industries clearly indicates an increased risk for haematolymphatic malignancies (leukaemia and malignant lymphoma), and a dose–response relationship with cumulative exposure to

SCC	Cancer Classification.	Tumour types[1] (inhalation)	Key evidence in support of classification
			butadiene. This is supported by findings of an association between environmental levels of butadiene and risk for leukaemia in children.
Pentachlorophenol(s)	EPA – L[2]	Haematolymphatic organs Liver	A two- to four-fold increased risk was generally seen between occupational exposure (e.g. sawmill workers) to pentachlorophenol and non-Hodgkin's lymphoma, multiple myeloma, or soft tissue sarcoma. Increased risk of liver cancer in sawmill workers was also observed.
Soot	IARC 1	Lung	IARC has classified "soot as found in occupational exposure of chimney sweeps" as carcinogenic to humans (Group 1). There is evidence from human epidemiological studies that lung cancer is causally associated with occupational exposure during work as a chimney sweep.
Beryllium	IARC 1	Lung	A large body of evidence for elevated lung cancer mortality was observed in a number of studies of workers exposed occupationally to beryllium, including beryllium processing plants and the fluorescent tube industry. Higher risks were associated with higher exposures (prior to 1950).
Ethylene oxide	IARC 1	Haematolymphatic organs	A number of epidemiological studies of exposed workers in chemical plants where ethylene oxide was produced or converted into derivatives, or in facilities where it was used as a sterilant, found an association between ethylene oxide and lymphatic and haematopoietic cancers, and specifically lymphoid tumours.

Not applicable is used in the context of has the chemical been tested and associated with cancers following inhalation.

[1] Only tumour types associated with inhalation exposure are relevant for this assessment.

[2] It is noted that pentachlorophenol was classified as a possible carcinogen (2B) by IARC in 1999, however US EPA have classified pentachlorophenol as a "likely carcinogen" based on 2006 human epidemiological studies.

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APPENDIX A

Methodology for Hazard Assessment

1.0 METHODOLOGY FOR HAZARD ASSESSMENT

1.1 Identification of Substances

A list of substances potentially present (SPP) was generated and is presented in Table A1 below. The SPP were selected using a combination of three different strategies:

- Review of list of chemicals provided by IFI (2012).
- Review of chemicals identified above the laboratory limit of reporting in the environmental preliminary site investigation undertaken by Golder Associates (2012).
- Literature search considering previously reported chemical exposures from similar facilities, and potential products of incomplete combustion.

1.2 Master List of Chemicals

IFI (2012) provided Golder Associates with an List of chemicals that include substances, mixtures and articles that may have been utilised in fire training. Chemicals included on the List have been included in the SPP and a copy of the List is provided in Appendix 2.

For chemical mixtures provided on the List, Golder Associates requested further information from CFA regarding any brand names, product names, documentation or photographs that may be available for the chemical mixtures that were burnt. No additional information was able to be provided.

Golder Associates reviewed material safety data sheets (MSDS) for the following foam products provided by IFI (2012):

- Angus Forexpan S Class A Foam Concentrate (ChemWatch 1997).
- FC-600 Light Water Brand ATC/AFFF. Reference 10-3869-4 (3m Australia, 1990).
- FC-3150 3M (TM) Fire Brake BFFF Concentrate (3M Australia, 1996a).
- FC-3045 3M Brand Superconcentrated Training Foam (3M Australia, 1996b).

Substances identified during this review were included as a SPP.

1.3 Chemicals identified from Preliminary Site Assessment

Golder Associates (2012) undertook an environmental preliminary site assessment at the Fiskville Fire Training facility which included targeted sampling from soil, sediment and surface water. Based on a review of the analytical data obtained during the preliminary site assessment any chemical that was above the laboratory limit of reporting (LOR) was included as a SPP.

It is important to note that the presence of substances in soil, sediment and surface water does not necessarily mean that these are present due to activities on the site. Also the presence of a compound in environmental media is not necessarily indicative of prior exposures of people attending the site. Regardless if a substance was identified in as present it was included as an SPP.

The laboratory undertook analysis of soil, sediment and surface water samples for tentatively identified compounds (TICs), a method of identifying chemicals that are not included as a target compounds in the standard analytical suite. Only discrete chemicals from the TICs analysis have been included as SPP. Compounds such as substituted alkenes have been excluded.

1.4 Products of Incomplete Combustion

A literature search was undertaken to identify previously reported chemical exposures from similar facilities, and potential products of incomplete combustion. Consistent search words were used during the literature search, “fire”, “training”, “burn pit” and “combustion”.

The following sources were reviewed in the literature search:

- US National Library of Medicine (NLM) Medline Database (Medline, 2012).
- US National Library of Medicine (NLM) Toxline Database (Toxline, 2011).
- Ebsco Publishing (Ebsco) Ebscohost Databases (Ebsco, 2012).
- United States Environmental Protection Agency (USEPA, 2012).
- Agency for Toxic Substances and Disease Registry (ATSDR , 2012).
- Health Protection Agency (HPA, 2012).
- Department of Health (DoH, 2012).
- World Health Organisation (WHO, 2012).
- A limited google internet search.

The E Journals, Environment Complete, Greenfile and Library Information Science and Technology Abstracts databases were searched when using the Ebsco (2012) website.

2.0 SUBSTANCES POTENTIALLY PRESENT (SPP)

On completion of Stage 1, a list of substances potentially present (SPP) was produced, provided in Table A1 below. It must be noted that the SPP are solely based on the references in Section **Error! Reference source not found.**,.

Table A1: Substances Potentially Present

Abbreviatons used in the table :
M : Master list of chemicals provided by CFA
MSDS: MSDS provided by CFA
E: Chemical identified above the laboratory detection limit in Golder Associates (2012) Environmental Investigation
C : Identified as an incomplete combustion by product through literature search

Substances Potentially Present (SPP)	Source	Reference	Substances Potentially Present (SPP)	Source	Reference
Mixture			**Combustion Products**		
Oil	M	a	Phosphoric acid	C	d
Sump oil	M	a	Arsenic Trioxide	C	d
Thinner(s) – e.g. paint thinner	M	a	Chromate Copper Arsenate	C	d
Solvents – e.g. S51	M	a	Bromine	C	d
Paint	M	a	Polytetrafluoroethylene (PTFE)	C	d
Waste fuel	M	a	1,3 Butadiene	C	e
Foam	M	a	Polychlorinated Biphenyl (PCB)	C	e
High expansion (foam)	M	a	Naphthalene	C	e
Training Foam	M	a	Acetaldehyde	C	e
Articles			Benzofuran	C	e
Tyre	M	a	Carbon Black	C	e
Crate	M	a	Dichloromethane (methylene chloride)	C	e
Wood	M	a	Furan	C	e
Pine	M	a	Isoprene	C	e
Treated timber	M	a	2-Nitroamisole	C	e
Hay	M	a	Polychlorophenols	C	e
Plastic	M	a	Pentachlorophenols	C	e
Cars	M	a	2,4,6-Trichlorophenol	C	e
Dry chem	M	a	Silica (crystalline)	C	e
Dry powder	M	a	Silica (amorphous)	C	e
Inorganics			Styrene	C	e
Sodium	M	a	Sulphuric acid	C	e
Magnesium	M	a	Tetrachloroethylene	C	e
Chlorine	M	a	Trichloroethylene	C	e

Substances Potentially Present (SPP)	Source	Reference	Substances Potentially Present (SPP)	Source	Reference
Sulphur	M	a	Chloroform	C	e
Aluminium	M	a	Triphenylene	C	e
Oxygen	M	a	Polychlorinated benzenes	C	e
Arsenic	E	b	1,2,4 Trichlorobenzene	C	n
Cadmium	E	b	1,3 Dichlorobenzene	C	n
Total Chromium	E	b	1,2 Dichlorobenzene	C	n
Chromium III	E	b	Methyl chloroform	C	f
Chromium VI	E	b	smog	C	f
Copper	E	b	Hexachlorobenzene	C	f
Mercury	E	b	Hydrochloric acid	C	g
Nickel	E	b	Polyvinyl Chloride	C	g
Lead	E	b	Carbon disulphide	C	h
Zinc	E	b	Chlorodifluoromethane	C	h
Benzene	M	a	Chloromethane (methyl chloride)	C	h
Toluene	M	a	Hexane	C	h
Xylene	M	a	Pentane	C	h
Acetylene	M	a	Propylene (propane)	C	h
TPH (C6-C9)	E	b	Ethylene oxide	C	o
TPH (C10-C14)	E	b	Hydrogen Peroxide	C	o
TPH (C15-C28)	E	b	Soot	C	p
TPH (C29-C36)	E	b	Ethylbenzene	C	i
Dioxins incl (TCDD, 2,3,7,8 tetrachlorodibenzodioxin)	E	b	Cyclopentanone	C	i
4- Methylphenol (p cresol)	E	b	Dimethyl Cyclohexene	C	i
3 - Methylphenol (m cresol)	E	b	Ethenyl Cyclohexene	C	i
1,2,4 trimethylbenzene	E	b	Hexanenitrile	C	i
Fluoranthene	E	b	Ethynyl Benzene	C	i
Pyrene	E	b	Nonane	C	i
1,3,5 trimethylbenzene	E	b	Propenyl Cyclohexane	C	i
Foam			Methylethyl Benzene	C	i
Halon	M	a	Propyl Benzene	C	i
B-class	M	a	Phenol	C	i
Aqueous Film Forming Foam (AFFF)	M	a	Cyanobenzene	C	i
Alcohol resistant AFFF	M	a	Propenyl Benzene	C	i

Substances Potentially Present (SPP)	Source	Reference	Substances Potentially Present (SPP)	Source	Reference
Fluoroprotein Foam (FP)	M	a	Methyl Ethenyl Benzene	C	i
Bromochlorodifluoromethane (CBF)	M	a	Methyl Methylethyl Benzene	C	i
Perfluorooctanoic acid (PFOA)	E	b	Limonene	C	i
6:2 fluorotelomer sulfonate (6:2 FtS)	E	b	Dihydro Indene	C	i
Perfluorooctanesulfonic acid (PFOS)	E	b	Hydroxy Benzaldehyde	C	i
Fuel			Indene	C	i
Methyl Ethyl Ketone (MEK)	M	a	Ethyl Dimethyl Benzene	C	i
Acetone	M	a	Methyl Phenol (o cresol - p& m cresol provided elsewhere)	C	i
Methanol	M	a	Methyl Benzaldehyde	C	i
Ethanol	M	a	Undecane	C	i
Ether	M	a	Dimethylpropyl Benzene	C	i
Petrol – leaded, unleaded, not specified	M	a	Butynyl Benzene	C	i
Diesel	M	a	Methyl Indene	C	i
Gas	M	a	Azulene	C	i
LPG	M	a	Benzo(b)thiophene	C	i
AvGas	M	a	Benzisothiazole	C	i
Creosote	M	a	Hexahydro Azepinone	C	i
Kerosene	M	a	Dihydro Methyl Naphthalene	C	i
Paraffin	M	a	Butyl Trimethyl Benzene	C	i
Tentitatively Identified Compounds (TICS)			Methyl Naphthalene	C	i
TICS in surface water			Biphenyl	C	i
Ethyl cyclohexane	E	b	Dimethyl Naphthalene	C	i
Bi cyclo (4,2,0) octa 1,3,5 triene	E	b	Dihydro Acenaphthylene	C	i
Benzaldehyde	E	b	Dimethyl Hexenyl Methyl Benzene	C	i
Docecanoic acid	E	b	Pentadecane	C	i
Tetradecanoic acid	E	b	1,1 Biphenyl Methyl	C	i
Octadecanoic acid	E	b	Isocyano Naphthalene	C	i
TICS in sediment			Naphthalene carboxaldehyde	C	i
Hexadecanoic acid	E	b	Propenyl Naphthalene	C	i

Substances Potentially Present (SPP)	Source	Reference	Substances Potentially Present (SPP)	Source	Reference
Pentatriacontene isomer	E	b	Trimethyl Naphthalene	C	i
1-octadecene	E	b	1 H fluorene	C	i
Nonadecane	E	b	Dimethyl Biphenyl	C	i
Coprostes-3-one	E	b	Dibenzothiophene	C	i
Stigmast-4-en-3 -one	E	b	9 H Fluorene Methylene	C	i
TICS in soil			Phenylnaphthalene	C	i
(3 beta) 3 methoxy -olean -1,2 ene	E	b	1,2 Dichloroethane	C	j
(beta) methoxy - d - friedoolean -14 -ene	E	b	Diethtyl Phthalate	C	j
tetratetracontane	E	b	2,4 Dimethylphenol	C	j
eicosane isomer	E	b	2 Methylnaphthalene	C	j
Ingredients in Foam			Barium	C	j
Diethylene glycol mono n butyl ether	MSDS	l	Beryllium	C	j
fluoroalkyl surfactants	MSDS	l	Calcium	C	j
Hexylene glycol	MSDS	l	Cobalt	C	j
Sodium chloride	MSDS	l	Iron	C	j
Zinc oxide	MSDS	l	Manganese	C	j
Bactericide	MSDS	l	Molybdenum	C	j
Fluorosurfactants	MSDS	l	Potassium	C	j
alkyl sulfate amine salt	MSDS	l	Strontium	C	j
sodium alykl sulfate	MSDS	l	Tin	C	j
Surfactants	MSDS	l	Titanium	C	j
Tolyl Triazole	MSDS	l	Vanadium	C	j
lauryl alcohol	MSDS	m	Yttrium	C	j
Sodium lauryl C10- C16 alkyl ether sulfate	MSDS	m	Acetophenone	C	k
Combustion By Products			2-butanol	C	k
Carbon Monoxide (CO)	C	c,d	n-butanol	C	k
Acrolein (irritant)	C	c,d	2-butoxyethanol (butyl cellosolve)	C	k
Formaldehyde (irritant)	C	c,d,e	2-butoxyethanol acetate	C	k
Crotonaldehyde (irritant)	C	c	n-butyl acetate	C	k
Polycyclic Aromatic Hydrocarbons (PAH)	C	c,d	2,6 di-t-butyl-p-cresol (BHT)	C	k

Substances Potentially Present (SPP)	Source	Reference	Substances Potentially Present (SPP)	Source	Reference
Benz(a)anthracene	C	e	C8-C12 alkanes	C	k
Benzo(b)fluoranthene	C	e	Chlorotoluene	C	k
Benzo(k)fluoranthene	C	e	Cyclohexanone	C	k
Benzo(a)pyrene	C	e	Diacetone alcohol	C	k
Chyrsene	C	e	2-ethoxyethanol (cellosolve)	C	k
Dibenz(a,h)anthracene	C	e	2-ethoxyethanol acetate	C	k
Indeno[1,2,3-cd]pyrene	C	e	ethyl amyl ketone (3 octanone)	C	k
Anthracene	C	e	Heptane	C	k
Benzo(e)pyrene	C	e	2-heptanol	C	k
Benzo(g,h,i)perylene	C	e	2-hexanol	C	k
Fluorene	C	e	indane	C	k
Phenanthrene	C	e	Isobutanol	C	k
Acenaphthylene	C	e	Isobutyl isobutyrate (IBIB)	C	k
Acenaphthene	C	e	Isopropanol	C	k
Particles (PM2.5)	C	c,d	Isopropyl acetate	C	k
Particles (PM10)	C	c,d	mesityl oxide	C	k
Ozone (O_3)	C	c	methyl amyl alcohol	C	k
Hydrogen Cyanide (HCN)	C	c,d	Methyl amyl ketone (MAK)	C	k
Hydrogen Chloride (HCl)	C	c,d	Methyl butyl ketone (MBK) (2 hexanone)	C	k
Phosphorous Pentoxide (P_2O_5)	C	c,d	Methyl cyclohexane	C	k
Isocyanate	C	c,d	Methyl Ethyl Ketone (MEK)	C	k
Hydrogen Flouride (HF)	C	c,d	Methyl hexanes	C	k
Hydrogen Bromide (HBr)	C	c,d	Methyl indane	C	k
Nitrogen Oxides	C	d	Methyl isobutyl ketone (MIBK)	C	k
Sulphur Dioxide (SO_2)	C	c,d	Methyl naphthalenes	C	k
Ammonia (NH_3)	C	c	n propyl acetate	C	k
Methane	C	c	Tetrahydronaphthalene	C	k
Nitrogen	C	c	1,1,1-trichloroethane	C	k
Carbon Dioxide (CO_2)	C	d	Bis-2-ethylhexylphthlate (DEHP)	C	k
Phosgene (inorganic irritant)	C	d	Cyanide	C	k
Polychlorinated dibenzodioxins (PCDD)	C	d,e	1,1 dichloroethane	C	k
Polychlorinated dibenzofurans (PCDF)	C	d,e	n-nitrosodiphenylamine	C	k
Perfluoroisobutene (PFIB)	C	d	Xirconium	C	k

Substances Potentially Present (SPP)	Source	Reference	Substances Potentially Present (SPP)	Source	Reference
Phosphorous	C	d	Thallium	C	k

References:

a: IFI (2012)
b: Golder Associates (2012)
c: HPA (2002)
d: Wakefield (2010)
e: IARC (2010)
f: US Air Force (unknown)
g: Lamden (1999)
h: USACHPPM (2008).
i: Ryan
j:USEPA (1992)
k: Salisbury (1987)
l: 3M Australia (1990)
m: ChemWatch (1997)
n: Instititute of Medicine (2011)
o : Al-Malki *et al* (2008)
p: Fuenekes *et al* (1997)

3.0 CANCER HAZARD IDENTIFICATION

The chemicals listed in Table A1 were investigated to identify if the chemical was a suspected or known carcinogen or a suspected or known human mutagen. For the purposes of screening, classifications by expert international organisations or Australian/overseas regulatory authorities were used. In particular classifications were sought from:

- International Agency for Research on Cancer (IARC, 2012).
- Australian Hazardous Substances Information System (HSIS, 2010).
- European Chemicals Agency (ECHA, 2012).
- United States National Toxicology Program (NTP, 2011).
- United States Environment Protection Authority (US EPA, 2012).
- American Conference of Industrial Hygienists (ACGIH, 2012).
- DFG Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area (German MAK, presented in ACGIH, 2012).

In the absence of a classification information on similar substances was considered. If no information was available the status of the substances was designated as unknown (U).

Since each of the organisations have different requirements for considering a chemical as a potential human carcinogen, a hierarchical approach was adopted during screening. The hierarchy is consistent with Australian science policy (enHealth 2004).

To assign a chemical into the carcinogenic group, the following classifications were used:

1) The chemical is classified by the International Agency for Research on Cancer (IARC) as Class 1 or 2A.
   - **IARC-1**: "Carcinogenic to Humans. The exposure circumstance entails exposures that are carcinogenic to humans. This category is used when there is *sufficient* evidence of carcinogenicity in humans."
   - **IARC-2A:** "Probably Carcinogenic to Humans. The exposure circumstance entails exposures that are probably carcinogenic to humans. This category is used when there is *limited* evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals."
2) The chemical is classified by the Australian Hazardous Substance Information System (HSIS) and/or the European Chemicals Agency (ECHA) as the following:
   - R45, H350, Carc Cat 1 or Carc Cat 2: May cause cancer.
   - R46, H351 or Muta 1 or Muta 2: May cause heritable genetic damage.
   - R49: May cause cancer by inhalation.
3) The chemical is classified by the United States National Toxicology Program (NTP) as NTP-K:
   - NTP-K: Known to be a human carcinogen.
4) Over the last few decades the United States Environment Protection Authority (US EPA) Integrated Risk Information System (IRIS) has changed its cancer classification scheme and descriptions of the evidence required for classification. These are summarised in USEPA (2011). Classifications may appear as some compounds classified in the 1980's or 1990's have not been reclassified using the upgraded system(s). The classifications used to determine whether a compound was considered carcinogenic were Class A, B, CaH, L or K, these are essentially equivalent to IARC groups 1 and 2A.

From the US EPA 1986 *Risk Assessment Guidelines* (US EPA 1986)

- **EPA - A:** "Human Carcinogen: Sufficient evidence from epidemiologic studies to support a causal association between exposure and cancer."
- **EPA - B:** "Probable Human Carcinogen: Weight of evidence of human carcinogenicity based on epidemiologic studies is limited; agents for which weight of evidence of carcinogenicity based on animal studies is sufficient."

From the 1996 *Proposed Guidelines for Carcinogen Risk Assessment* (US EPA 1996). This preceded the revised draft released in 1999.

- **EPA - K:** "Known Human Carcinogen: agents *known* to be carcinogenic in humans based on either epidemiologic evidence or a combination of epidemiologic and experimental evidence, demonstrating causality between human exposure and cancer, or agents that should be treated as *if* they were *known* carcinogens, based on a combination of epidemiologic data showing plausible causal association (not demonstrating it definitively) and strong experimental evidence."

From the revised classifications first published in the revised draft *Guidelines for Carcinogen Risk Assessment* (US EPA 1999). In 2005, the US EPA published the final version of the *Guidelines for Carcinogen Risk Assessment* (US EPA 2005), which contained the same descriptor for summarising weight of evidence for human carcinogenic potential.

- **EPA - CaH:** "Carcinogenic to Humans: this is appropriate when there is convincing epidemiologic evidence showing causality between human exposure and cancer. The descriptor is also appropriate when there is absence of conclusive epidemiologic evidence to clearly establish a cause and effect relationship between human exposure and cancer, but there is compelling evidence of carcinogenicity in animals and mechanistic information in animals and humans showing similar modes of carcinogenic action."
- **EPA - L:** "Likely to be Carcinogenic to Humans: this is appropriate when the available tumour effects and other key data are adequate to demonstrate carcinogenic potential to humans. Adequate data are within a spectrum: at one end is evidence for an association between human exposure to the agent and cancer and strong experimental evidence of carcinogenicity in animals. At the other, with no human data, the weight of experimental evidence shows animal carcinogenicity by a mode of action that is relevant or assumed to relevant to humans."

5) The chemical is classified by the American Conference of Industrial Hygienists (ACGIH) as a TLV-A1 or a TLV-A2.

- **TLV-A1:** "Confirmed Human Carcinogen: The agent is carcinogenic to humans based on the weight of evidence from epidemiologic studies."
- **TLV-A2:** "Suspected Human Carcinogen: Human data are accepted adequate in quality but conflicting or insufficient to classify in experimental animals at dose(s), by route(s) of exposure, at site(s), of histologic type(s), or by mechanism(s) considered relevant to worker exposure."

6) The chemical is classified by the German MAK Commission (ACGIH, 2012) as a MAK Class 1 or 2.

- **MAK-1:** "Substances that *cause cancer in man* and can be assumed to make a significant contribution to cancer risk. Epidemiological studies provide adequate evidence of a positive correlation between the exposure of humans and the occurrence of cancer."
- **MAK-2:** "Substances that are *considered to be carcinogenic to man* because sufficient data from long-term animal studies or limited evidence from animal studies substantiated by evidence from epidemiological studies indicate that they can make a significant contribution to cancer risk."

7) The chemical is classified by the U.S. National Institute for Occupational Safety and Health (NIOSH) presented in ACGIG (2012):

- **NIOSH-Ca:** "Potential occupational carcinogen, with no further categorization. Any substance, or combination or mixture of substances, which causes an increased incidence of benign and/or malignant neoplasms in humans or in one or more experimental mammalian species as the result of any oral, respiratory or dermal exposure, or any other exposure which results in the induction of tumours at a site other than the site of administration. Includes any substance which is metabolised into one or more potential occupational carcinogens by mammals."

8) Professional judgement using weight of evidence appraisals were used in the absence of a conclusion in a peer reviewed expert toxicology report.

The results of the carcinogen screen are presented in Table A2 below.

Table A2: Carcinogen Screen for Substances Potentially Present

Substances Potentially Present (SPP)	Genotoxic	Carcinogenic	Substances Potentially Present (SPP)	Genotoxic	Carcinogenic
Mixture			**Combustion Products**		
Oil	U	U	Phosphoric acid	N	N
Sump oil	U	U	Arsenic Trioxide	N	Y
Thinner(s) – e.g. paint thinner	U	U	Chromate Copper Arsenate	U	U
Solvents – e.g. S51	U	U	Bromine	N	N
Paint	U	U	Polytetrafluoroethylene (PTFE)	N	N
Waste fuel	U	U	1,3 Butadiene	Y	Y
Foam	U	U	Polychlorinated Biphenyl (PCB)	N	Y
High expansion (foam)	U	U	Naphthalene	N	Y
Training Foam	U	U	Acetaldehyde	Y	Y
Articles			Benzofuran	N	N
Tyre	U	U	Carbon Black	N	Y
Crate	U	U	Dichloromethane (methylene chloride)	Y	Y
Wood	U	U	Furan	N	Y
Pine	U	U	Isoprene	N	N
Treated timber	U	U	2-Nitroamisole	N	Y
Hay	U	U	Polychlorophenols	N	N
Plastic	U	U	Pentachlorophenols	N	Y
Cars	U	U	2,4,6-Trichlorophenol	N	Y
Dry chem	U	U	Silica (crystalline)	N	Y
Dry powder	U	U	Silica (amorphous)	N	N
Inorganics			Styrene	N	N

Substances Potentially Present (SPP)	Genotoxic	Carcinogenic	Substances Potentially Present (SPP)	Genotoxic	Carcinogenic
Sodium	N	N	Sulphuric acid	N	Y
Magnesium	N	N	Tetrachloroethylene	Y	Y
Chlorine	N	N	Trichloroethylene	Y	Y
Sulphur	N	N	Chloroform	N	Y
Aluminium	N	N	Triphenylene	N	N
Oxygen	N	N	Polychlorinated benzenes	U	U
Arsenic	Y	Y	1,2,4 Trichlorobenzene	N	N
Cadmium	Y	Y	1,3 Dichlorobenzene	N	N
Total Chromium	*	*	1,2 Dichlorobenzene	N	N
Chromium III	N	N	Methyl chloroform	Y	Y
Chromium VI	Y	Y	smog	U	U
Copper	N	N	Hexachlorobenzene	N	Y
Mercury	N	N	Hydrochloric acid	N	N
Nickel	Y	Y	Polyvinyl Chloride	N	N
Lead	N	Y	Carbon disulphide	N	N
Zinc	N	Y	Chlorodifluoromethane	N	N
Benzene	Y	Y	Chloromethane (methyl chloride)	Y	Y
Toluene	N	N	Hexane	N	N
Xylene	N	N	Pentane	N	N
Acetylene	N	N	Propylene (propane)	N	N
TPH (C6-C9)	Y	Y	Ethylene oxide	Y	Y
TPH (C10-C14)	Y	Y	Hydrogen Peroxide	N	N
TPH (C15-C28)	Y	Y	Soot	N	Y
TPH (C29-C36)	U	U	Ethylbenzene	N	N
Dioxins incl (TCDD, 2,3,7,8 tetrachlorodibenzodioxin)	N	Y	Cyclopentanone	N	N
4- Methylphenol (p cresol)	N	N	Dimethyl Cyclohexene	U	U
3 - Methylphenol (m cresol)	N	N	Ethenyl Cyclohexene	U	U
1,2,4 trimethylbenzene	N	N	Hexanenitrile	N	N

Substances Potentially Present (SPP)	Genotoxic	Carcinogenic	Substances Potentially Present (SPP)	Genotoxic	Carcinogenic
Fluoranthene	Y	Y	Ethynyl Benzene	U	Y
Pyrene	Y	Y	Nonane	N	N
1,3,5 trimethylbenzene	N	N	Propenyl Cyclohexane	U	U
Foam			Methylethyl Benzene	N	N
Halon	U	U	Propyl Benzene	N	N
B-class	U	U	Phenol	N	N
Aqueous Film Forming Foam (AFFF)	U	U	Cyanobenzene	U	U
Alcohol resistant AFFF	U	U	Propenyl Benzene	U	U
Fluoroprotein Foam (FP)	U	U	Methyl Ethenyl Benzene	U	N
Bromochlorodifluoromethane (CBF)	U	U	Methyl Methylethyl Benzene	U	U
Perfluorooctanoic acid (PFOA)	N	N	Limonene	N	N
6:2 fluorotelomer sulfonate (6:2 FtS)	U	U	Dihydro Indene	U	U
Perfluorooctanesulfonic acid (PFOS)	N	N	Hydroxy Benzaldehyde	U	U
Fuel			Indene	N	N
Methyl Ethyl Ketone (MEK)	N	N	Ethyl Dimethyl Benzene	U	U
Acetone	N	N	Methyl Phenol (o cresol - p& m cresol provided elsewhere)	N	N
Methanol	N	N	Methyl Benzaldehyde	N	N
Ethanol	N	Y	Undecane	N	N
Ether	N	N	Dimethylpropyl Benzene	U	U
Petrol – leaded, unleaded, not specified	Y	Y	Butynyl Benzene	U	U
Diesel	Y	Y	Methyl Indene	U	U
Gas	Y	Y	Azulene	U	N
LPG	Y	Y	Benzo(b)thiophene	U	N
AvGas	Y	Y	Benzisothiazole	U	U
Creosote	N	Y	Hexahydro Azepinone	U	U
Kerosene	N	N	Dihydro Methyl Naphthalene	U	U
Paraffin	N	N	Butyl Trimethyl Benzene	U	U
Tentitatively Identified Compounds (TICS)			Methyl Naphthalene	U	U

Substances Potentially Present (SPP)	Genotoxic	Carcinogenic	Substances Potentially Present (SPP)	Genotoxic	Carcinogenic
TICS in surface water			Biphenyl	N	N
Ethyl cyclohexane	U	U	Dimethyl Naphthalene	U	U
Bi cyclo (4,2,0) octa 1,3,5 triene	U	U	Dihydro Acenaphthylene	U	U
Benzaldehyde	N	N	Dimethyl Hexenyl Methyl Benzene	U	U
Docecanoic acid	N	N	Pentadecane	N	N
Tetradecanoic acid	N	N	1,1 Biphenyl Methyl	N	N
Octadecanoic acid	N	N	Isocyano Naphthalene	U	U
TICS in sediment			Naphthalene carboxaldehyde	U	U
Hexadecanoic acid	N	N	Propenyl Naphthalene	U	U
Pentatriacontene isomer	U	U	Trimethyl Naphthalene	U	U
1-octadecene	U	N	1 H fluorene	U	U
Nonadecane	N	N	Dimethyl Biphenyl	U	N
Coprostes-3-one	U	U	Dibenzothiophene	U	N
Stigmast-4-en-3 -one	U	U	9 H Fluorene Methylene	U	U
TICS in soil			Phenylnaphthalene	U	U
(3 beta) 3 methoxy -olean -1,2 ene	U	U	1,2 Dichloroethane	Y	Y
(beta) methoxy - d - friedoolean - 14 -ene	U	U	Diethtyl Phthalate	N	N
tetratetracontane	U	U	2,4 Dimethylphenol	N	N
eicosane isomer	N	N	2 Methylnaphthalene	N	N
Ingredients in Foam N/A			Barium	N	N
Diethylene glycol mono n butyl ether	N	N	Beryllium	Y	Y
fluoroalkyl surfactants	U	U	Calcium	N	N
Hexylene glycol	N	N	Cobalt	N	Y
Sodium chloride	N	N	Iron	N	N
Zinc oxide	N	N	Manganese	N	N
Bactericide	U	U	Molybdenum	N	N
Fluorosurfactants	U	U	Potassium	N	N
alkyl sulfate amine salt	U	U	Strontium	N	N
sodium alykl sulfate	U	U	Tin	N	N

Substances Potentially Present (SPP)	Genotoxic	Carcinogenic	Substances Potentially Present (SPP)	Genotoxic	Carcinogenic
Surfactants	U	U	Titanium	N	N
Tolyl Triazole	U	U	Vanadium	N	N
lauryl alcohol	N	N	Yttrium	N	N
Sodium lauryl C10- C16 alkyl ether sulfate	N	N	Acetophenone	N	N
Combustion By Products			2-butanol	N	N
Carbon Monoxide (CO)	N	N	n-butanol	N	N
Acrolein (irritant)	N	N	2-butoxyethanol (butyl cellosolve)	N	N
Formaldehyde (irritant)	Y	Y	2-butoxyethanol acetate	N	N
Crotonaldehyde (irritant)	Y	Y	n-butyl acetate	N	N
Polycyclic Aromatic Hydrocarbons (PAH)	Y	Y	2,6 di-t-butyl-p-cresol (BHT)	U	U
Benz(a)anthracene	Y	Y	C8-C12 alkanes	U	U
Benzo(b)fluoranthene	Y	Y	Chlorotoluene	N	N
Benzo(k)fluoranthene	Y	Y	Cyclohexanone	N	N
Benzo(a)pyrene	Y	Y	Diacetone alcohol	N	N
Chyrsene	Y	Y	2-ethoxyethanol (cellosolve)	N	N
Dibenz(a,h)anthracene	Y	Y	2-ethoxyethanol acetate	U	U
Indeno[1,2,3-cd]pyrene	Y	Y	ethyl amyl ketone (3 octanone)	N	N
Anthracene	Y	Y	Heptane	N	N
Benzo(e)pyrene	Y	Y	2-heptanol	U	U
Benzo(g,h,i)perylene	Y	Y	2-hexanol	N	N
Fluorene	N	N	indane	U	U
Phenanthrene	Y	Y	Isobutanol	N	N
Acenaphthylene	Y	Y	Isobutyl isobutyrate (IBIB)	U	U
Acenaphthene	Y	Y	Isopropanol	N	N
Particles (PM2.5)	N	N	Isopropyl acetate	N	N
Particles (PM10)	N	N	mesityl oxide	N	N
Ozone (O_3)	N	N	methyl amyl alcohol	U	U
Hydrogen Cyanide (HCN)	N	N	Methyl amyl ketone (MAK)	N	N
Hydrogen Chloride (HCl)	N	N	Methyl butyl ketone (MBK) (2 hexanone)	N	N
Phosphorous Pentoxide (P_2O_5)	N	N	Methyl cyclohexane	N	N
Isocyanate	N	U	Methyl Ethyl Ketone (MEK)	N	N
Hydrogen Flouride (HF)	N	N	Methyl hexanes	U	U
Hydrogen Bromide (HBr)	N	N	Methyl indane	U	U

Substances Potentially Present (SPP)	Genotoxic	Carcinogenic	Substances Potentially Present (SPP)	Genotoxic	Carcinogenic
Nitrogen Oxides	N	N	Methyl isobutyl ketone (MIBK)	N	N
Sulphur Dioxide (SO_2)	N	N	Methyl naphthalenes	U	U
Ammonia (NH_3)	N	N	n propyl acetate	N	N
Methane	N	N	Tetrahydronaphthalene	N	U
Nitrogen	N	N	1,1,1-trichloroethane	N	N
Carbon Dioxide (CO_2)	N	N	Bis-2-ethylhexylphthlate (DEHP)	N	Y
Phosgene (inorganic irritant)	N	N	Cyanide	Y	Y
Polychlorinated dibenzodioxins (PCDD)	N	N	1,1 dichloroethane	N	N
Polychlorinated dibenzofurans (PCDF)	N	N	n-nitrosodiphenylamine	Y	Y
Perfluoroisobutene (PFIB)	N	N	Xirconium	U	N
Phosphorous	N	N	Thallium	N	N

Y: Chemical considered to be genotoxic and / or a mutagen.

N: Inadequate information to consider the chemical to be genotoxic and / or a carcinogen.

U: Unable to find information on the genotoxic or carcinogenic status of the chemical.

*Total chromium assessed as chromium (III) and chromium (VI).

4.0 CONTEXTUAL INFORMATION OF SUBSTANCES CLASSIFIED AS CARCINOGENS

If a chemical was classified as a 'suspected or known human carcinogen' or 'suspected or known mutagen' in the literature search outlined in the section above then it was considered to be a substance classified as a carcinogen(SCC). A list of SCC is provided in Table A3 below. It should be noted that the fact that a chemical has been placed into a 'carcinogen' categorisation in this document should not be taken as any indicator of potential risk to people given a set of circumstances. There are 64 SCC listed below.

Table A3: Substances classified as carcinogens

SCC	Genotoxic	Carcinogenic	SCC	Genotoxic	Carcinogenic
Arsenic & Arsenic compounds	Y	Y	Dioxins including (TCDD, 2,3,7,8 tetrachlorodibenzodioxin)	N	Y
Cadmium	Y	Y	Polychlorinated Biphenyl (PCB)	N	Y
Chromium VI	Y	Y	PAH Compounds	Y	Y
Nickel	Y	Y	Naphthalene	N	Y
Lead	N	Y	1,3 Butadiene	Y	Y
Benzene (when present in petroleum hydrocarbons or other solvents, or fuels such as Petrol – leaded, unleaded, Diesel, Gas, LPG, AvGas)	Y	Y	Dichloromethane (methylene chloride)	Y	Y
Ethanol[1]	N	Y (in alcoholic beverages)	Furan	N	Y
Creosote	N	Y	2-Nitroamisole	N	Y
Formaldehyde (irritant)	Y	Y	Pentachlorophenol(s)	N	Y
Crotonaldehyde (irritant)	Y	Y	Soot	N	Y
2,4,6-Trichlorophenol	N	Y	Ethynyl Benzene	U	Y
Silica (crystalline)	N	Y	1,2 Dichloroethane	Y	Y
Sulphuric acid	N	Y	Beryllium	Y	Y
Tetrachloroethylene	Y	Y	Cobalt	N	Y
Trichloroethylene	Y	Y	Cyanide	Y	Y
Chloroform	N	Y	n-nitrosodiphenylamine	Y	Y
Methyl chloroform	Y	Y	Chloromethane (methyl chloride)	Y	Y
Hexachlorobenzene	N	Y	Ethylene oxide	Y	Y

1. Excluded as the route of exposure is not relevant to this report.

Y: Chemical considered to be genotoxic and / or a mutagen.
N: Chemical is not considered to be genotoxic and / or a carcinogen.
U: Unable to find information on the genotoxic or carcinogenic status of the chemical.

APPENDIX B

List of Chemicals Supplied by Independent Fiskville Investigation Team

1.0 LIST OF CHEMICALS SUPPLIED BY INDEPENDENT FISKVILLE INVESTIGATION TEAM

A list of chemicals/materials was supplied to Golder from the Independent Fiskville Investigation Team on the 2nd April 2012 as an email attachment (word document). The list received is reproduced below.

"MASTER CHEMICALS/MATERIALS LIST"

Sodium

Magnesium

Chlorine

Sulphur

Aluminium

Oxygen

Oil

Sump oil

Petrol – leaded, unleaded, not specified

Diesel

Gas

LPG

AvGas

Aviation Fuel or Av Gas

Creosote

Waste fuel

Kerosene

Paraffin

Thinner(s) – e.g. paint thinner

Solvents – e.g. S51

Benzene

Toluene

Toluol

Xylene

Acetylene

Ether

EMK (?)

Acetone

Methanol

Ethanol

Paint

Tyre

Crate

Wood

Pine

Treated timber

Hay

Plastic

Cars

Asbestos

Foam

CBF

BCF – (Bromocholorodifluoromethane) – Halon 2011

Halon

B-class

AFFF – (Aqueous Film Forming Foam)

AR AFFF – Alcohol resistant AFFF

Dry powder

Dry chem

High expansion

FP – Fluoroprotein Foam

Training Foam

PFOS

APPENDIX C

Limitations

LIMITATIONS

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