Director, Force Health Protection
Health Services Group Headquarters
1745 Alta Vista Drive
Ottawa, Ontario, K1A 0K6

HMCS CHICOUTIMI Fire Incident of 5 October 2004

Potential Chemical Exposures and Health Consequences

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Prepared by:

Stephen Tsekrekos, BSc, MD, MSc, FRCPC
Occupational Medicine Specialist

Janick Lalonde, PhD
Senior Advisor Toxicology

Executive Summary

The HMCS CHICOUTIMI fire of 5 Oct 2004 rapidly produced highly toxic, thick black smoke, that caused the death of Lt(N) Chris Saunders and smoke inhalation injuries in several crewmembers. After the fire was extinguished, crewmembers who remained on board had to live and work within the smoke damaged submarine during the 5-day journey back to Faslane, Scotland.

The crew of HMCS CHICOUTIMI experienced toxic chemical exposures as a result of the fire, and so it is important to a) understand the nature of those exposures, and b) consider the possible long-term health consequences of such exposures. The purpose of this report is to explore these issues, utilizing information that is specific to the HMCS CHICOUTIMI fire incident, as well as general information from the medical and scientific literature.

Hazardous Chemicals and Smoke Exposure

The potential for a hazardous chemical to cause harm is based on the fundamental principle of dose-response: the greater the amount, or dose, of exposure to a hazardous chemical, the greater the risk and/or severity of harm. We are all exposed to very small amounts of hazardous chemicals every day through the food we eat, water we drink, air we breathe, and products we use. However, whether or not harm will result from exposure to a hazardous chemical is dependent on the amount, frequency, and duration of that exposure.

Smoke from a fire represents a hazardous chemical exposure. The risk of harm and the severity of harm depend on the amount of smoke exposure. Mild exposures may result in no symptoms or only temporary minor symptoms, such as watery eyes or mild cough, whereas severe smoke exposures can be fatal or cause significant lung injury.

Smoke Exposures from the HMCS CHICOUTIMI Fire

It will never be possible to exactly re-create the exposure conditions that existed on-board HMCS CHICOUTIMI during the fire. However, it is possible to make reasonable conclusions, based on investigations related to the incident as well as general scientific knowledge regarding the nature of fires and combustion products.

It is known that a basic set of combustion products is associated with nearly all fires, and that these can be broadly classified into a small number of categories. For example, some combustion gases act as *simple* asphyxiants. These gases, such as carbon dioxide, are not toxic in their own right, but if produced in large enough amounts will displace the oxygen that is normally present in the air. It is this lowered level of oxygen that results in harm. Other gases, such as carbon monoxide, are *chemical* asphyxiants. Once inhaled, they interfere with the body's ability to transport and use oxygen, making the body internally "oxygen deficient" even if there is enough oxygen in the atmosphere to be inhaled. Another major category of combustion gases is irritants. These are highly reactive chemicals that can cause direct tissue irritation or damage, and can result in injury throughout the respiratory tract, from the nose and mouth to deep in the lungs.

As part of the investigation into the HMCS CHICOUTIMI fire, the National Research Council of Canada (NRC) conducted laboratory studies on several items damaged in the fire. This was done to determine the fire properties of these items, as well as to analyze the gases produced when those items were burnt. The tested items included the mattress, sleeping bags, briefcase, entrance curtain, and entrance door to the Commanding Officer's Cabin, insulation samples (vent trunk, deck head, and pressure hull), and electrical cables from the Commanding Officer's Cabin and Electrical Space.

These studies demonstrated that carbon dioxide was the most abundant combustion gas produced, at levels that were much higher than other gases. The next most abundantly produced gas was carbon monoxide. Both of these observations were consistent with what has been documented in the scientific study of fires in both laboratory and "real-world" situations. Irritant gases are also commonly produced in fires, and several irritant gases were detected in the NRC tests. Some of these gases, such as nitric oxide, were common to all tested materials, whereas other irritant gases were specific to the tested material. For example, hydrogen chloride and hydrogen fluoride were predominantly produced by the combustion of the electrical cables obtained from the Commanding Officer's Cabin and Electrical Space.

The NRC laboratory test results of individual HMCS CHICOUTIMI materials cannot be used to exactly re-create the smoke that existed on board on 5 Oct 2004. However, the results are suggestive that several minutes of unprotected exposure to the smoke could have resulted in serious smoke inhalation injury, which is consistent with what occurred on board HMCS CHICOUTIMI.

Long-Term Health Consequences of Smoke Inhalation

Although investigations such as those conducted by the NRC offer some insight into the nature of the possible exposures related to the HMCS CHICOUTIMI fire, there will always be uncertainty regarding the exact chemical composition of the smoke as well as the degree of smoke exposure for individual crewmembers. This uncertainty is not unusual; detailed exposure information is typically unknown for survivors of smoke inhalation.

Although the combustion products created by fires may be numerous, the body's response to combustion product exposure is a relatively narrow range of symptoms and injuries. These health effects have been well characterized from the medical and scientific study of smoke inhalation survivors. Once the acute injury has occurred, the body's natural healing and repair processes proceed. Full recovery from smoke inhalation injury, even in severe cases, is expected. However, some smoke inhalation survivors may not recover completely, and can develop persistent long-term health problems.

The risk of developing long-term health problems after smoke inhalation depends on the dose, or amount, of smoke inhaled. Clinically, the smoke inhalation dose can be judged by the severity of the symptoms experienced in the short term. If the initial symptoms were relatively mild, then it can be reasonably concluded that the amount of smoke exposure was minimal. Generally speaking, those who sustained a severe smoke inhalation injury are at greater risk of having long-

term health problems, whereas such problems are very rare among people whose initial injury was mild.

The most commonly observed long-term effect of smoke inhalation injury is changes in lung function, including the development of reactive airways, or asthma. Tests of lung function shortly after smoke inhalation injury are good predictors of long-term outcomes: if these tests are normal several months after smoke inhalation injury, then lung function abnormalities would not be expected to occur as a result of the smoke exposure.

Carbon monoxide poisoning is a common result of smoke inhalation. Long-term effects on the central nervous system have been observed in studies of survivors. These effects can include impaired memory and concentration, and changes in mood. Such effects, if they are to occur, are present within weeks to a few months after exposure. Fire survivors who do not have cognitive symptoms shortly after exposure would not be expected to develop cognitive difficulties years down the road as a result of the smoke exposure.

Medical experience indicates that, in general, individuals who go on to develop long-term health problems from smoke inhalation injury show signs of these problems within hours to weeks after smoke inhalation. Those who are clinically well several months after smoke inhalation are essentially "in the clear" and would not be expected to develop long-term health problems because of their smoke exposure.

Cancer

Fires can produce numerous chemical products, some of which are carcinogens. Carcinogens are chemicals that have the potential to cause cancer. This potential to cause cancer is dependent on the amount of exposure to the carcinogen: the greater the amount of exposure to a carcinogen, then the greater the risk of developing cancer.

Any degree of exposure to a chemical carcinogen will theoretically increase the risk of developing cancer. Practically speaking, however, a certain amount of exposure to a chemical carcinogen is required before this risk becomes meaningful. We are all exposed to very low levels of chemical carcinogens (many of them occurring in nature) through the food we eat, water we drink and air we breathe. These exposures are considered to be non-harmful, however, since the theoretical risk of developing cancer from such exposures is so low that it is essentially non-existent.

Several chemicals that are commonly produced by fires are recognized carcinogens, such as benzene, polycyclic aromatic hydrocarbons, and dioxins and furans. The exact types and quantities of these chemicals that may have been present in the fire smoke is unknown, but it is reasonable to conclude that the HMCS CHICOUTIMI smoke contained chemical carcinogens, and that the crew were exposed to them.

The actual risk of developing cancer will depend on the amount, or dose, of exposure. This risk can be reasonably qualified by comparing the HMCS CHICOUTIMI crew to occupational

groups who have also experienced exposure to the multiple chemical products contained within fire smoke, such as firefighters.

Several scientific studies suggest that the risk of developing cancer is increased among individuals who have worked as firefighters. However, this increased cancer risk is typically only evident after 10 years (and in some cases after more than 20 to 30 years) of employment as a firefighter. A certain "dose" of firefighter exposures are required before cancer risk is meaningfully increased, and current scientific knowledge suggests that this dose is in the neighbourhood of several hundred fires.

As stated above, any degree of exposure to a chemical carcinogen will theoretically increase the risk of developing cancer. However, the increased risk from just one fire, as experienced by the crew, would be much smaller than the risk associated with exposure to several hundred fires. The development of cancer in crewmembers, as a result of exposures from a single fire, is therefore highly unlikely.

Exposures after the Fire was Extinguished and Long-Term Health Consequences

Once the fire of 5 Oct 2004 was extinguished, crewmembers who remained on board had to live and work within the smoke damaged submarine during its 5-day journey back to Faslane, Scotland. Although the gases produced by the fire had dissipated, the aerosol particles (soot) produced by the fire remained behind as visible smoke damage. Crewmembers were potentially exposed to this leftover soot and dust, through inhalation, ingestion, and skin absorption.

In an effort to assess these potential exposures, sampling of the leftover soot and dust was performed on board HMCS CHICOUTIMI shortly after it returned to Faslane. These samples were analyzed for various chemical compounds, such as metals, polycyclic aromatic hydrocarbons, and dioxins and furans. Several hazardous chemical compounds were identified. Potential exposures to these chemicals were assessed using estimations of the amount of leftover soot/dust that was inhaled, ingested, or absorbed through the skin. Because of uncertainties regarding actual individual exposures, conservative "worst case" exposure estimates were used to minimize the chances of underestimating actual exposures.

Even with "worst-case" exposure estimates, the analysis indicated that exposure to soot and dust on board HMCS CHICOUTIMI during the 5-day journey back to Faslane, Scotland, would not have resulted in a significantly increased risk of long-term health problems, including cancer.

Conclusions

The HMCS CHICOUTIMI fire of 5 Oct 2004 was a dramatic event that produced highly toxic smoke. Smoke exposure has the potential to result in acute injury, and may also cause long-term health problems. The risk of harm and the severity of harm depend on the amount of smoke exposure.

Current medical knowledge regarding smoke inhalation indicates that, in general, individuals who go on to develop long-term health problems from smoke inhalation injury show signs of

these problems within hours to weeks after smoke inhalation. For crewmembers who fit this description, it is important that they receive appropriate medical care and follow-up.

Crewmembers who were clinically well several months after the fire would be expected to remain well; it is not anticipated that they would go on to develop long-term health problems because of their exposures.

It must be emphasized that this report is specific to health effects related to chemical exposures only. As in any life-threatening scenario, the HMCS CHICOUTIMI incident represented significant psychological trauma. The effects of such trauma are experienced variably by different individuals, possibly causing psychological, cognitive, and/or physical symptoms. This is an important area for consideration regarding the long-term health of the crew, but this topic was beyond the scope of this report.

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Acronyms and Abbreviations

2,3,7,8-TCDD	2,3,7,8-Tetrachlorodibenzo-p-dioxin
A35	Aft of bulkhead 35
ADI	Acceptable daily intake
AEGL	Acute Exposure Guideline Levels
AMS	Auxiliary Machinery Space
ATSDR	Agency for Toxic Substances and Disease Registry
BOI	Board of Inquiry
BOOP	Bronchiolitis obliterans with organizing pneumonia
BW	Body weight
CCME	Canadian Council of Ministers of the Environment
CCS/NCIC	Canadian Cancer Society/National Cancer Institute of Canada
CF	Canadian Forces
CH_4	Methane
CO	Carbon monoxide
COC	Contaminants of (potential) concern
CO Cabin	Commanding Officer's Cabin
CR	Control Room
CT	Computed tomography
D FHP	Director Forces Health Protection
DMEPM(SM)	Director Maritime Equipment Program Management (Submarines)
DMSS	Director Maritime Ship Support
DRDC-A	Defence Research and Development Canada - Atlantic
EBS	Emergency Breathing System
F35	Forward of bulkhead 35
FEV_1	Forced expiratory volume in 1 second
FVC	Forced vital capacity
GERD	Gastroesophageal reflux disease
HC	Health Canada
HCl	Hydrogen Chloride
HCN	Hydrogen Cyanide
HF	Hydrogen Fluoride
HMCS	Her Majesty's Canadian Submarine
HQ	Hazard quotient
IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System
JECFA	Joint Expert Committee on Food Additives and Contaminants
LOAEL	Lowest observable adverse effect level
MR	Motor Room
MRI	Magnetic Resonance Imaging
MRL	Minimal risk level
NFPA	National Fire Protection Association
NAS	National Academy of Science
NETE	National Engineering and Test Establishment
NO	Nitric oxide

NOAEL	No observable adverse effect level
NRC	National Research Council Of Canada
PAHs	Polycyclic aromatic hydrocarbons
PCDDs	Polychlorinated dibenzo-para-dioxins
PCDFs	Polychlorinated dibenzofurans
ppm	Parts per million
PVC	Polyvinyl chloride
RADS	Reactive Airway Dysfunction Syndrome
RfD	Reference dose
RUDS	Reactive Upper Airways Dysfunction Syndrome
SCBA	Self-contained breathing apparatus
SVOCs	Semi-volatile organic compounds
SO_2	Sulphur Dioxide
TEQ	Toxic equivalency
TDI	Tolerable daily intake
TRV	Toxicity Reference Values
US EPA	United States Environmental Protection Agency
VOCs	Volatile organic compounds
WHO	World Health Organization
WSC	Weapons Storage Compartment

1.0 Introduction

1.1 Background

On 5 October 2004, one day after departing Faslane, Scotland for Canada, a fire occurred on board HMCS CHICOUTIMI. The fire originated in the Commanding Officer's Cabin and spread one deck below to the Electrical Space before being extinguished. Large amounts of thick black smoke were rapidly produced by the fire, resulting in heavy smoke and soot damage to the mid-section of the submarine, between bulkheads 35 and 56, on both 1 Deck and 2 Deck. Nine crewmembers out of the 57 on board sustained smoke inhalation injuries as a result of the fire (Board of Inquiry, 2004). On 6 Oct 2004, three of the injured crewmembers were airlifted off of the HMCS CHICOUTIMI and transferred to hospital in Sligo, Ireland. Sadly, one of these crewmembers, Lt(N) Chris Saunders, died as a result of his smoke inhalation injury. The remaining crew stayed on board the smoke-damaged vessel while it was towed back to Faslane, Scotland, where it arrived on 10 Oct 2004.

A brief overview of the HMCS CHICOUTIMI fire incident is provided in Section 1.5. An abridged timeline of events related to the HMCS CHICOUTIMI fire is provided in Annex A. Annex B provides more detail regarding the extent of damage that resulted from the fire.

1.2 Purpose

The fire on board HMCS CHICOUTIMI was a significant occupational exposure event. The smoke was highly toxic, as evidenced by the fact that exposure resulted in death and smoke inhalation injuries in several crewmembers. As well, all surviving crewmembers who remained on-board were exposed to leftover soot from the fire as they lived and worked in the smoke-damaged submarine during the five-day transit back to Faslane, Scotland. The time period from the onset of the fire until the final docking in Faslane, will be referred to as the HMCS CHICOUTIMI fire "incident" in this report.

The crew of HMCS CHICOUTIMI experienced toxic chemical exposures as a result of the fire incident, and so it is important to a) understand the nature of those exposures, and b) consider the possible long-term health consequences of such exposures. The purpose of this report is to explore these issues, utilizing information that is specific to the HMCS CHICOUTIMI fire incident, as well as general information from the medical and scientific literature.

The intent of this report is to:

- Inform the crew of the HMCS CHICOUTIMI regarding what they were most likely exposed to and what those exposures could mean to their health; and

- To provide background information to medical staff so as to assist in their care of HMCS CHICOUTIMI crewmembers.

1.3 Scope

This report focuses on potential chemical exposures experienced by HMCS CHICOUTIMI crewmembers as a result of the fire that originated in the Commanding Officer's Cabin on 5 October 2004, and considers the time period from the onset of the fire until docking in Faslane, Scotland on 10 October 2004 (the HMCS CHICOUTIMI fire "incident"). The possible long-term health effects discussed in this report are therefore specific to this event. Consideration of any long-term health effects related to occupational and non-occupational chemical exposures that may have occurred outside this time frame is beyond the scope of this report.

An additional unrelated fire occurred in an oxygen generator in the weapons storage compartment several hours after the main fire in the Commanding Officer's Cabin. This fire was short-lived (the fire was reported out two minutes after onset) and resulted in no further injuries or damage to the submarine (Board of Inquiry, 2004). Therefore, the oxygen generator fire is not considered further in this report.

Due to privacy and medical confidentiality considerations, personal medical information of individual crewmembers and the results of medical investigations performed on the crew after the incident will not be discussed in this report. This report provides general information, and it is intended that crewmembers and their medical caregivers will apply this information to the context of their own specific individual circumstances.

This report only focuses on potential chemical exposures and possible long-term health effects related to such exposures. As in any life-threatening scenario, the HMCS CHICOUTIMI incident represented significant psychological trauma. The effects of such trauma are experienced variably by different individuals, possibly causing psychological, cognitive, and/or physical symptoms. Although this is an important area for consideration regarding the long-term health of the crew, such discussion was beyond the scope of this report.

1.4 Limitations

Extensive environmental monitoring was not performed during or immediately after the fire. Therefore, it is impossible to know with complete certainty the exact type and quantity of chemical compounds produced from the fire. As a result, it is impossible to know with complete certainty the exact type and quantity of chemical exposures experienced by individual crewmembers.

To address this uncertainty, several initiatives have been undertaken since the fire in an attempt to better characterize the potential exposures experienced by HMCS CHICOUTIMI crewmembers. These initiatives, such as laboratory combustion studies of materials consumed in the fire, provide some degree of insight into potential exposures experienced by the crew. However, there are limitations to these information sources and the conclusions that can be derived from them. The specific limitations of each information source are discussed in the body of the report.

In addition to specific initiatives related to the HMCS CHICOUTIMI fire, a body of scientific literature exists that has examined fire atmospheres, smoke inhalation injury, and long-term health effects from acute chemical exposures. This literature has been relied upon to make inferences regarding possible long-term health consequences for the HMCS CHICOUTIMI crew. This report is based on current medical and scientific knowledge, and health guidelines that are derived from this knowledge. It is recognized that such knowledge evolves over time; future scientific developments may, or may not, affect the conclusions reached in this report.

1.5 HMCS CHICOUTIMI Fire Incident - Exposure Scenario

The following discussion is intended to provide background general information regarding the fire and resultant exposure scenario, based on information extracted from the HMCS CHICOUTIMI fire Board of Inquiry report (Board of Inquiry, 2004). An abridged timeline of events related to the HMCS CHICOUTIMI fire is provided in Annex A. Annex B provides more detail regarding the extent of damage that resulted from the fire.

The fire was caused by an electrical arcing event that originated near the joints in the main power cables running from the forward battery switchboard to the main propulsion switchboard located in the Motor Room. The cables in question were located in the Commanding Officer's Cabin, under the bunk. The fire at the point of origin was extremely hot, perforating through the steel deck and spread to the Electrical Space on the deck below the Commanding Officer's Cabin.

The crew reacted quickly to fight the fire: electrical power was cut and the fire was brought under control with carbon dioxide and aqueous film forming foam extinguishers. The fire had rapidly produced thick black smoke and resulted in zero visibility in the midsection of the submarine, between bulkheads 35 and 56. The use of smoke curtains at the bulkhead 35 and 56 hatches limited the spread of smoke beyond the midsection of the submarine.

Crewmembers were protected from the smoke atmosphere through the use of the submarine's Emergency Breathing System (EBS). The EBS is a clean air supply drawn from the general use high-pressure air system that runs throughout the submarine. In the event of a fire, crew don full-face mask airline respirators that are then plugged into air system couplings that are distributed throughout the submarine. Movement through the submarine while wearing EBS masks requires that the airline be disconnected from a coupling and then reconnected to another open coupling.

The EBS was not completely protective, as evidenced by the smoke inhalation injuries experienced by the crew. Donning of EBS masks was delayed for some crewmembers in order to conduct fire-fighting activities. As well, limitations in the availability of EBS masks and open EBS couplings to plug the mask air-lines into also resulted in delays in accessing the EBS air supply, and in some cases, a need to rely on "buddy-breathing" (sharing of a single mask between crewmembers).

It is difficult to make generalizations regarding potential crew exposures to the fire atmosphere based on their location in the submarine at the time of the fire. Although smoke exposures would be expected to be greatest close to the source of the fire, being located some distance from

the fire does not necessarily imply minimal exposure. A submarine is an enclosed space, and any smoke that is produced will eventually spread to fill the compartment if it is not vented. As well, research indicates that toxic gases produced by a fire undergo little dilution as they spread from the point of origin (Lizhong et al., 2008). Smoke curtains limited the spread of soot to other compartments. However, if these curtains were not airtight, they may not have limited the spread of toxic gases produced by the fire.

The upper conning tower lid was opened after the fire to allow for passive smoke clearance, but active smoke clearance did not commence until the starboard engine was started about 90 minutes after the onset of the fire. This engine then continued to run for the entire 5-day transit back to Faslane, ventilating the submarine with air incoming through the Conning Tower. The passageways, hatches, and doorways acted as trunking to allow for airflow throughout the submarine, and the incoming air was exhausted through the diesel exhaust (DMEPM(SM), 2008).

With the fire out, starboard engine running for ventilation, and submarine atmosphere "in spec" (based on Draeger tube air monitoring), the crew came off of the EBS just over three hours after the fire started. From that time until the HMCS CHICOUTIMI docked in Faslane, Scotland, 5 days later, the crew worked and lived in the soot contaminated submarine environment.

The exposures potentially experienced by the crew due to the HMCS CHICOUTIMI fire incident can essentially be divided into two main categories:

- Exposure to the fire atmosphere that was produced when the fire was actively burning and smoldering. This atmosphere would have consisted of gases and aerosols (soot), and would have been present until "cleared" through active ventilation.

- Exposure to the settled soot left over from the fire. Exposure to the soot could have occurred during the entire 5-day period from the onset of the fire until docking in Faslane, Scotland on 10 October 2004.

1.6 Toxicological Principles

This report focuses on potential health effects related to chemical exposures. The potential for a chemical to result in harm to a person is based on certain fundamental principles.

Firstly, exposure to that chemical must occur. Exposure implies that the chemical entered into the body, such as through inhalation, ingestion, or absorption through the skin. In the absence of exposure to a chemical, that chemical cannot cause harm.

Secondly, the risk of harm is directly related to how much chemical exposure occurred. In other words, "the dose makes the poison": the greater the amount, or dose, of exposure to a hazardous chemical, the greater the risk and/or severity of harm. We are all exposed to very small amounts of hazardous chemicals every day through the food we eat, water we drink, air we breathe, and products we use. Whether or not harm will result from exposure to a hazardous chemical is dependent on the amount, frequency, and duration of that exposure.

2.0 HMCS CHICOUTIMI Fire Atmosphere

2.1 Overview of Fire Atmospheres

This section provides general background information on the combustion products that are produced from a fire (the fire atmosphere). Following this general discussion, information sources that provide insight into the specific fire atmosphere that existed on board HMCS CHICOUTIMI are discussed in Sections 2.2 to 2.5.

Fire is a complex chemical and physical process. When sufficient heat is applied to a material, the material starts to break down, referred to as thermal decomposition. Products released during the decomposition, or smoldering phase, include combustible liquids and gases. In the presence of sufficient oxygen and an ignition source, the combustible products will ignite, and flaming occurs. Flaming combustion will release heat and generate combustion products, consisting of gases, liquids, and smoke particles (Orzel, 1993).

Combustion products from fires are complex and are influenced by several factors, such as the chemical composition of the base material burned, the temperature of the fire, and the availability of oxygen (Hartzell, 1996; Orzel, 1993; Blanc et al., 2005). As well, the types and quantities of combustion products will vary with time over the life of the fire, due to factors such as changes in temperature, oxygen availability, and airflow (Jankovic et al., 1991; Miller et al., 2003; Hartzell, 1996). Fire atmospheres also evolve dynamically as they spread beyond the fire origin, partitioning into different vertical layers and moving between vertical layers over time (Lizhong et al., 2008). Fire atmospheres can contain hundreds, if not thousands, of different chemical products (Lees, 1995; Kimmel et al., 1997).

Because of the complexity of fire atmospheres, it is virtually impossible to accurately predict the composition of such atmospheres at any given location or time. However, it is possible to characterize the fire environment in general terms, since a basic set of combustion products is associated with nearly all fires (Lees, 1995). Although the combustion products created by fires may be numerous, the body's response to combustion product exposure is a relatively narrow range of symptoms and injuries. The vast array of chemical products produced by a fire can be broadly classified into a small number of categories, based on the manner in which they cause harm. Gases that are produced by fires can be categorized into simple asphyxiants, chemical asphyxiants, and irritants (Hartzell, 1996; Bizovi, et al., 1995; Lees, 1995; Orzel, 1993; Miller et al., 2003).

2.1.1 Simple Asphyxiants

Simple asphyxiants are gases that do not directly cause harm on their own. In other words, they are not toxic in their own right. If these gases are produced in large enough amounts, they will displace the oxygen that is normally present in the air. It is this lowered level of oxygen that results in harm, because oxygen is essential to life. Exposure to very low levels of oxygen in the air can rapidly lead to unconsciousness and death.

Fires commonly produce simple asphyxiant gases. These gases are typically small, very simple molecules. The combustion of any carbon-containing substance in the presence of oxygen will produce carbon dioxide. In fire scenarios, carbon dioxide is typically the gas produced in the greatest quantity, with measured levels exceeding all other combustion gases. Measured quantities of carbon dioxide from structural and experimental fires range from several hundred to several thousand parts per million (ppm), with the highest recorded levels in the 50,000 to 100,000 ppm range (Jankovic et al., 1991; Lees, 1995).

Examples of other simple asphyxiants gases produced in fire atmospheres include simple hydrocarbons (small molecules consisting solely of carbon and hydrogen atoms), such as methane, ethylene, acetylene, and propylene. These gases may also be produced in large quantities during fires, although not as much as carbon dioxide. Measured levels of total simple hydrocarbon asphyxiants can reach as high as a few hundred ppm (Jankovic et al., 1991).

2.1.2 Chemical Asphyxiants

Chemical asphyxiants are those gases that, when absorbed into the body through the lungs, interfere with the body's ability to transport and use oxygen. Chemical asphyxiants can cause the body to become internally "oxygen deficient", even if there is enough oxygen in the atmosphere to be inhaled. Examples of chemical asphyxiants that are typically observed in fire smoke include carbon monoxide and hydrogen cyanide.

Carbon monoxide is produced in virtually all fire environments. Quantities of carbon monoxide produced in fire atmospheres are relatively high, typically exceeded only by carbon dioxide. Measured quantities of carbon monoxide from structural and experimental fires can reach a few hundred ppm, and in some cases can reach a few thousand ppm (Jankovic et al., 1991; Lees, 1995; Austin et al., 2001a). Carbon monoxide poisoning is the most common immediate cause of death in fire victims (Wald et al., 1987; Lee et al., 2006).

Hydrogen cyanide is produced from the incomplete combustion of nitrogen containing materials such as nylons, synthetic fabrics, wool, and polyurethane (Kimmel et al., 1997; Lees, 1995). Measured quantities of hydrogen cyanide from structural and experimental fires can range from non-detectable to a few ppm, but can reach as high as a few hundred ppm in confined enclosed spaces (Kimmel et al., 1997; Jankovic, 1991; Lees, 1995). Concentrations of hydrogen cyanide in the few hundred ppm range can cause acute fatality (Kimmel et al., 1997). Acrylonitrile is a gas with irritant properties, but once absorbed into the body it is metabolized into hydrogen cyanide, thereby acting as a potential chemical asphyxiant as well.

2.1.3 Irritants

Irritants are gases that can cause direct tissue irritation or injury at the site of contact. Irritant gases are highly reactive, and their interaction with the body's cells can lead to cellular damage and/or death and subsequent inflammation. Irritant gases that are inhaled are capable of causing damage throughout the respiratory tract, from the nose and mouth to deep in the lungs (Haponik, 1993; Blanc, 2005; Rabinowitz et al., 2002).

The different types of irritant gases that may be produced in a fire are numerous. Examples of common irritants found in fire smoke include formaldehyde, acrolein, sulphur dioxide, nitrogen dioxide and other oxides of nitrogen, hydrogen chloride acid, ammonia, and acetaldehyde. Measured quantities of irritant gases from structural and experimental fires are typically in the few ppm to tens of ppm range (Jankovic et al., 1991; Lees, 1995; Austin et al., 2001a), but can reach much higher levels, depending on the source material burned (for example, burning plastics such as polyvinyl chloride (PVC) can produce several hundred ppm of hydrogen chloride; Kimmel et al., 1997). Some irritant gases are extremely reactive and can cause significant damage even at low concentrations. A classic example of this is acrolein, which can rapidly produce pulmonary edema (fluid in the lungs) at concentrations as low as 10 ppm (Delclos et al., 1996).

2.1.4 Other Toxic Effects

The acute symptoms related to smoke inhalation are largely due to the effects of the three categories of gases listed above: simple asphyxiants, chemical asphyxiants, and irritants. As well, virtually hundreds of volatile organic compounds (VOCs) may be produced by combustion. Exposure to sufficient quantities of VOCs can result in acute irritant effects, however VOCs produced by fires are typically present at concentrations far below levels at which acute effects would be observed (Lees, 1995). Many VOCs can also be absorbed through the lungs into the general circulation. Some VOCs can affect organ systems other than the lungs, such as the blood, immune system, nervous system, liver, or kidneys. Some VOCs that may be produced by fires, such as benzene, are chemical carcinogens. Sufficient exposure to chemical carcinogens may increase long-term cancer risk.

Of all the possible VOCs produced by fires, benzene is typically the most frequently observed VOC, and is often detected in greater quantities than other VOC's (Jankovic et al., 1991; Austin et al., 2001a, 2001b). Although the total number of different VOCs produced in a fire may be numerous, recent studies suggest that the bulk of the total VOCs produced in a fire are made up of only a small number of individual chemical compounds. Out of 144 sampled VOCs at both experimental and municipal structural fires, for example, 65% to 77% of the total VOC mass was made up of just 14 different chemical compounds. These 14 compounds were propene, benzene, xylenes, 1-butene/2-methylpropene, toluene, propane, 1,2-butadiene, 2-methylbutane, ethylbenzene, naphthalene, styrene, cyclopentene, 1-methylcyclopentene, isopropylbenzene (Austin et al., 2001a, 2001b).

2.1.5 Aerosols

In addition to the gaseous combustion products described above, fires also produce aerosol particles (soot), which may be solid or liquid droplets. Aerosols are derived from the burned material or can be newly formed from the combustion process (Lees, 1995). The chemical composition of fire-created aerosol particles is varied, from relatively inert elemental carbon to more complex carbon-based molecules such as polycyclic aromatic hydrocarbons (PAHs), and dioxins and furans. Metals and fibres may also be detected in fire-generated aerosols, depending on the base material that is consumed.

Most aerosol particles produced by a fire are small enough to penetrate deeply into the lungs if inhaled, and can cause adverse health effects through a number of different mechanisms (Miller et al., 2003; Lees et al., 1995):

- Chemically inert aerosols, such as elemental carbon, can cause lung damage if inhaled in sufficient quantities to overwhelm natural lung clearance mechanisms;
- Aerosols that are made up of harmful chemical compounds can cause local lung damage and/or be absorbed into the general circulation. Once absorbed, these compounds may affect other organ systems, similarly to VOCs, as discussed above. As well, some organic aerosols (for example: PAHs, dioxins and furans), heavy metals (for example: chromium (VI), cadmium, nickel), and fibres (for example: asbestos), are carcinogenic. Sufficient exposure to carcinogens can increase long-term cancer risk; and
- Toxic gases and vapours produced by the fire can adsorb onto aerosol particles; the aerosols can then act as a delivery vehicle, transporting these adsorbed gases and vapours deep into the lungs (Lee et al., 2006; Kimmel et al., 1997; Miller et al., 2003; Blanc et al., 2005).

2.2 Airborne Monitoring After the Fire

The only available direct measurements of the atmosphere on board HMCS CHICOUTIMI shortly after the fire were obtained from Draeger tubes. Draeger tubes are used to measure the instantaneous concentration of gases or vapours in air. Different tubes are used to measure different airborne contaminants. Further technical information regarding Draeger tubes is provided in Annex C.

The CF Fire Marshall collected the Draeger tubes used on board HMCS CHICOUTIMI as evidence for the Board of Inquiry. The six types of Draeger tubes collected had been used to measure oxygen, carbon dioxide, carbon monoxide, nitrous gases (nitric oxide and nitrogen dioxide), chlorine, and hydrochloric acid (Veinot et al., 2004).

Data transcribed from the original HMCS CHICOUTIMI Draeger tube measurement logs was provided by the Board of Inquiry (BOI President, 2004). Gases had been sampled from six locations on HMCS CHICOUTIMI after the fire: Motor Room (MR), Control Room (CR), aft of bulkhead 35 (A35), Auxiliary Machinery Space (AMS), forward of bulkhead 35 (F35), and Weapons Storage Compartment (WSC). Full Draeger tube measurement results are provided in Annex D.

As discussed in Section 2.1.1, carbon dioxide is usually the predominant gas produced in fires. However, carbon dioxide is also produced naturally by the body's metabolism and released into the air during exhalation. Carbon dioxide is normally present in a submarine atmosphere, and has been measured at concentrations up to a few thousand ppm under normal operating conditions on board HMCS WINDSOR, a sister submarine of HMCS CHICOUTIMI (Severs, 2005). Therefore, carbon dioxide would not necessarily be a good indicator of the gases that were specifically produced by the fire.

For illustrative purposes, the Draeger tube results for carbon monoxide are used in this report as an indicator gas to represent the atmosphere produced by the fire. Normal background concentrations on board HMCS WINDSOR, a sister submarine of HMCS CHICOUTIMI, were only in the range of a few ppm (Severs, 2005). Therefore, because carbon monoxide is produced in virtually all fires at relatively high concentrations compared to other combustion gases, levels of carbon monoxide that exceeded a few ppm could reasonably be assumed to be produced by the fire. As discussed below, with the exception of carbon dioxide, carbon monoxide was the only combustion gas that was measured in considerable quantities (several ppm). All of the other combustion gas measurements (nitrous gases, chlorine, and hydrochloric acid) did not exceed 2 ppm.

A summary of Draeger tube carbon monoxide measurements obtained on board HMCS CHICOUTIMI are provided in Table 1.

Table 1: Results of Carbon Monoxide (CO) Draeger Tube Measurements Obtained On Board HMCS CHICOUTIMI After the Fire

Date	Time of CO Measurement	Elapsed time from fire onset (1315 hrs)	Highest CO measurement (ppm)	Location of Highest CO measurement
5-Oct-04	1555 hrs	2 hrs, 40 min	70	F35 [a]
5-Oct-04	1851 hrs	5 hrs, 36 min	25	WSC [b]
5-Oct-04	1957 hrs	6 hrs, 42 min	40	F35 [c]
5-Oct-04	2036 hrs	7 hrs, 21 min	10	AMS
5-Oct-04	2230 hrs	9 hrs, 15 min	2	F35
5-Oct-04	2350 hrs	10 hrs, 35 min	0	-
6-Oct-04	0030 hrs	11 hrs, 15 min	0	-
6-Oct-04	0200 hrs	12 hrs, 45 min	0	-
6-Oct-04	0425 hrs	15 hrs, 10 min	0	-
6-Oct-04	0600 hrs	16 hrs, 45 min	0	-
6-Oct-04	0800 hrs	18 hrs, 45 min	0	-
6-Oct-04	1000 hrs	20 hrs, 45 min	0	-

Notes:

a. F35 = forward of bulkhead 35; WSC = Weapons Storage Compartment; AMS = Auxiliary Machinery Space; MR = Motor Room; A35 = aft of bulkhead 35
b. Measurements only recorded for WSC, A35, MR
c. Measurements only recorded for F35

There are several limitations to this Draeger tube data with respect to characterizing the fire atmosphere that existed on board HMCS CHICOUTIMI, considering the timing of when these samples were obtained. For example:

- The initial carbon monoxide measurement occurred at 1555 hrs, two hours and forty minutes *after* the fire in the Commanding Officer's Cabin had started;
- The initial carbon monoxide measurement was obtained *well after the fire had been extinguished*: two hours and 20 minutes after the fire was reported out in the Electrical Space, and one hour and 9 minutes after the fire was reported out in the Commanding Officer's cabin; and
- The initial carbon monoxide measurement was obtained one hour and twelve minutes *after the starboard engine was started for active smoke clearance* (passive smoke clearance had started shortly after the onset of the fire, with the opening of the conning tower lid).

It is clear from this timeline that the initial measurements of atmospheric quality were obtained well after the fires were out (and fire smoke was no longer actively being produced) and after smoke clearing measures had been undertaken. As well, several chemical compounds can cause interference with the Draeger tube measurement of carbon monoxide, such as high concentrations of hydrocarbons and halogenated hydrocarbons (Dräger Safety, Inc, 2004), which may have been present in the fire atmosphere. For all of these reasons, it can be assumed that the actual measured gas readings presented in Table 1 and Annex D underestimate the actual gas concentrations present in the submarine atmosphere while the fire was burning and shortly thereafter.

However, the Draeger tube results can be used in general terms to describe overall air quality in the hours after the fire. Of several samples taken at just over seven hours after the fire began, the highest recorded carbon monoxide concentration was only 10 ppm. By 10.5 hours after the fire, carbon monoxide was no longer detectable at any on board sampling location. If it is assumed that carbon monoxide is representative of gases produced by the fire, then it can be reasonably concluded that gases produced by the fire had been effectively cleared from the submarine and were no longer detectable 10.5 hours after the fire.

As described earlier, the crew donned EBS masks shortly after the fire started, and remained on EBS up until three hours and ten minutes after the start of the fire. Combustion gas concentrations on board HMCS CHICOUTIMI were likely at their highest levels during the fire (when combustion gases were actively produced) and shortly thereafter. Active smoke clearance measures, such as the use of the starboard engine, would have greatly reduced combustion gas concentrations. However, the Draeger tube results indicate that combustion gases were likely still present, to some degree, in the submarine atmosphere up until 10.5 hours after the fire, when carbon monoxide was no longer detectable. It can therefore be assumed that the entire crew was exposed to combustion gases, to some degree, after the instruction to remove EBS masks and before the atmosphere was completely cleared. These exposures would have been to combustion gas concentrations that were considerably lower than would have occurred when the fire was burning and before active smoke clearance measures were initiated.

As also described earlier, the EBS masks were not completely protective. Therefore, it can be assumed that at least some members of the crew were exposed to the fire atmosphere, to some degree, prior to the instruction to remove EBS masks. These exposures could potentially have been to the maximum combustion gas concentrations that were present as a result of the fire.

2.3 NRC cone calorimeter tests

2.3.1 Gas Analyses

2.3.1.1 Test Description

The National Research Council of Canada (NRC) conducted laboratory fire studies on several items that were exposed to the fire environment on HMCS CHICOUTIMI (Kanabus-Kaminska et al., 2007). These items are listed below, along with their National Engineering and Test Establishment (NETE) numerical designation:

- Mattress from the Commanding Officer's Cabin (NETE-13)
- Briefcase from the Commanding Officer's Cabin (NETE-14)
- Sleeping bags from the Commanding Officer's Cabin (NETE-15, NETE-16)
- Vent trunk insulation from the Electrical Space (NETE-31)
- Deckhead insulation from the Electrical Space (NETE-32)
- Pressure hull insulation from the Commanding Officer's Cabin (NETE-53)
- Entrance curtain to the Commanding Officer's Cabin (NETE-54)
- Entrance door to the Commanding Officer's Cabin (NETE-55)
- Deckhead insulation (NETE-56)
- Electrical cables from the Commanding Officer's Cabin (NETE-78)
- Electrical cables from the Electrical Space (NETE-79)

The fire studies were conducted using a cone calorimeter, a standard test procedure for determining fire properties of tested objects. Such properties included heat release rate, heat of combustion, ignition time, mass loss, and smoke production. As well, a multi-gas analyzer was utilized to investigate combustion gases produced by the test specimens. The specific gases that were analyzed included:

- Hydrocarbons (methane, ethylene, acetylene, formaldehyde, acrylonitrile, acrolein)
- Acid gases (hydrogen bromide, hydrogen chloride, hydrogen fluoride)
- Oxides of nitrogen (nitric oxide, nitrous oxide, nitrogen dioxide)
- Hydrogen cyanide
- Sulphur dioxide
- Phosgene
- Carbon monoxide
- Carbon dioxide, water vapour

According to the NRC, these gases were selected for analysis because they are common combustion gases. They are also representative of the categories of combustion chemicals produced by a fire, as discussed Section 2.1: simple asphyxiants (carbon dioxide, methane, ethylene, acetylene), chemical asphyxiants (carbon monoxide, hydrogen cyanide), and irritants (formaldehyde, acrylonitrile, acrolein, hydrogen bromide, hydrogen chloride, hydrogen fluoride, nitric oxide, nitrous oxide, nitrogen dioxide, sulphur dioxide, phosgene).

2.3.1.2 Limitations

The combustion gas analysis component of the cone calorimeter tests provides some insight into the types and quantities of combustion gases produced by the HMCS CHICOUTIMI fire. However, the cone calorimeter results cannot be used to "exactly" re-create the fire atmosphere on-board HMCS CHICOUTIMI. There are several reasons for this:

- Although the materials tested may be the same objects that were burned in the HMCS CHICOUTIMI fire, fire temperature and oxygen availability are important factors in determining the specific types and quantities of chemicals produced by a fire. Although the cone calorimeter tests provide a reasonable estimation of the gases produced during flaming combustion of the tested materials, it is impossible to exactly re-create the heat and oxygen characteristics of the HMCS CHICOUTIMI fire in a laboratory.

- Each tested item was analyzed individually, and the gas results are reflective of the gases produced by a specific item combusted on its own. During the HMCS CHICOUTIMI fire, however, multiple items were burning simultaneously. There is no reliable way to combine the individual item gas results to re-create the exact combustion atmosphere during the HMCS CHICOUTIMI fire.

- The test results are specific to standard-sized samples that were prepared from each item (for example, 10 cm x 10 cm sections were prepared from items such as the mattress, sleeping bag, and insulation, and the cable samples were 10 cm in length). The use of standard-sized samples allows for relative comparisons between tested items. However, the types and quantities of combustion products present in the HMCS CHICOUTIMI fire atmosphere would have been dependent upon the specific quantities of materials that were consumed in the fire.

- The gas concentration values provided by the cone calorimeter tests represent gas concentrations measured in the smoke plume of the test apparatus. In a non-laboratory setting, actual gas concentrations would be dependent not only on gas production by the fire, but also on other factors, such as airflow and the volume of air that the gases were dispersed into.

- It is recognized that fires may produce hundreds of different chemical compounds. The gases selected for analysis in the cone calorimeter tests were selected because they are common to fire atmospheres. It was not possible to measure "every" combustion gas produced during the cone calorimeter tests, and so it is unknown what other types and quantities of gases may have been produced by the combustion of the tested items.

- The tests were conducted on materials that were collected from the HMCS CHICOUTIMI after the fire. These provided materials may not have been completely representative of all of the on board materials that were damaged in the fire. As well, some of the tested materials had surface finishes that were almost completely burned off in the fire, and therefore there was little left of these surface finishes that could be tested in the lab.

For the above reasons, the results of the cone calorimeter gas analyses cannot be assumed to be exactly representative of the HMCS CHICOUTIMI fire atmosphere.

However, the cone calorimeter results do provide information about the relative differences between tested items in terms of types and quantities of combustion gases produced. General observations can also be made regarding the potential toxicity of the combustion gases produced by the individual tested items.

2.3.1.3 Results

The results of the cone calorimeter test combustion gas analyses were presented in the NRC report as figures depicting gas concentrations over time. Carbon dioxide concentrations were not included in the figures. According to the NRC, this is because carbon dioxide concentration values were much higher than the other gases produced and it would have been difficult to include carbon dioxide in the plotted figures (NRC, 2008).

Examples of gas analysis figures contained within the NRC report are provided in Annex I. These example figures illustrate several observations of the gas analysis results from the cone calorimeter tests:

- Gas concentrations fluctuated considerably with time (as would be expected in any fire atmosphere);
- The patterns of gas production (types and quantities) were unique to the items tested;
- Different gases reached their peak concentrations at different times during the test; and
- Some gases were mostly produced within a short span of time, whereas other gases were produced over a much longer period of time.

In order to discuss the health significance of the NRC combustion gas analysis results, it is first necessary to simplify the complex data provided in the figures from the NRC report.

Each figure was visually inspected and the peak concentration of each gas was identified. This is illustrated in the last figure of Annex I. In some cases, combustion gases were measured in more than one test on a particular HMCS CHICOUTIMI item. Where this occurred, the highest peak concentration of a particular gas out of all of the tests for a particular HMCS CHICOUTIMI item was selected as the representative peak gas concentration.

Given the dynamic nature of the measured combustion gases over time, the use of peak gas concentration to describe combustion gas production is an oversimplification. In most cases, the peak gas level was not sustained for more than a few seconds, and is not representative of gas production over the entire test. However, as a simple measure, the peak gas concentration indicates the maximum gas concentration value that existed at any particular time over the course of the test of an individual sample.

A summary of the observed peak combustion gas concentrations from the cone calorimeter tests is provided in Table 2.

Table 2: Summary of Peak Gas Concentrations Observed in Combustion Products Plots Contained Within the NRC Cone Calorimeter Report

Sample	NETE Number	CH_4[a]	Ethy	Acet	CO	NO	N_20	SO_2	HCN	Acry[b]	Acro	HCl	HF	Form
CO Cabin Mattress	NETE-13	20[c]	--[d]	--	380	16	--	26	8	15	[e]	--	--	14
CO Cabin Briefcase	NETE-14	12	4	10	220	9	--	--	--	--	--	--	--	--
CO Cabin Sleeping Bags	NETE-15 & NETE-16	6	4	8	240	35	--	--	4	--	--	1	--	--
Elect Space Vent Trunk Insulation	NETE-31	14	4	11	205	17	--	--	4	--	--	125	--	--
Elect Space Deck Head Insulation	NETE-32	24	7	14	330	27	--	--	5	10	--	--	--	--
CO Cabin Pressure Hull Insulation	NETE-53	26	2	4	85	7	--	--	6	52	[e]	--	--	--
CO Cabin curtain	NETE-54	12	--	--	175	7	--	--	3	--	[e]	--	--	5
CO Cabin Entrance Door	NETE-55	48	--	--	200	31	7	--	13	10	--	--	--	17
CO Cabin Cable - SS-23 (10 mm diam)	NETE-78 (Test 78-B)	5	3	--	70	5	--	--	--	--	--	250	--	--
CO Cabin Cable – DC-175 (10 mm diam)	NETE-78 (Test 78-C)	12	8	12	235	21	--	--	--	14	--	21	110	--
CO Cabin Cable – IC-44 (9 mm diam)	NETE-78 (Test 78-D)	13	8	11	235	9	--	--	--	14	--	7	33	--
CO Cabin Cable – IC-45 (19 mm diam)	NETE-78 (Test 78-E)	10	10	10	235	12	--	--	--	2	--	15	265	--
CO Cabin Cable - SS-1 (25 mm diam)	NETE-78 (Test 78-F)	13	10	--	60	5	--	--	--	--	--	36	4	--
CO Cabin Cable – DC-221 (6.5 mm diam)	NETE-78 (Test 78-G)	23	12	20	275	12	--	--	--	9	--	9	7	--
Elect Space Cable – PL-5 Pen-190 (23 mm diam)	NETE-79 (Test 79-A)	22	8	--	45	4	--	26	--	[f]	[e]	185	2	--
Elect Space Cable – PL-5 Pen-190 (23 mm diam)	NETE-79 (Test 79-B)	18	10	--	45	4	--	27	--	[f]	[e]	38	1	--
Elect Space Cable – PD-130 Pen-168 (33 mm diam)	NETE-79 (Test 79-C)	37	19	--	60	3	--	21	--	[f]	[e]	36	1	--
Elect Space Cable – PD-130 Pen-168 (33 mm diam)	NETE-79 (Test 79-D)	10	6	--	35	2	--	13	--	[f]	[e]	18	1	--
Elect Space Cable – PD-130 Pen-168 (33 mm diam)	NETE-79 (Test 79-E)	26	11	--	45	2	--	17	--	[f]	[e]	23	1	--

Notes:

a. CH_4 = methane, Ethy = ethylene, Acet = acetylene, CO = carbon monoxide, NO = nitric oxide, N_20 = nitrous oxide, SO_2 = sulphur dioxide, HCN = hydrogen cyanide, Acry = acrylonitrile, Acro = acrolein, HCl = hydrogen chloride, HF = hydrogen fluoride, Form = formaldehyde, diam = diameter, CO Cabin = Commanding Officer's Cabin; Elect Space = Electrical Space.

b. Problems with the measurement of acrylonitrile were noted in the NRC cone calorimeter report. Further discussions with NRC staff indicated that the reported values for acrylonitrile might have been underestimated by approximately 1 to 10 ppm.

c. All values are in parts per million (ppm)

d. "--" = not reported (plots were only provided for combustion products with peak concentrations of approximately 5 ppm or higher).

e. Problems with the measurement of acrolein were noted in the NRC cone calorimeter report. Acrolein was detected but was measured in negative concentrations. Further discussions with NRC staff indicated that acrolein was present at low levels, likely less than 1 to 10 ppm. Where "--" appears, acrolein was not detected.

f. Problems with the measurement of acrylonitrile were noted in the NRC cone calorimeter report. Acrylonitrile was detected but was measured in negative concentrations. Further discussions with NRC staff indicated that acrylonitrile was present at low levels, likely less than 1 to 10 ppm.

- Gas analyses of the Deckhead Insulation (NETE-56) were not performed, due to insufficient material

From Table 2 it can be seen that carbon monoxide was a commonly produced combustion gas and, with a few exceptions, carbon monoxide was measured at higher concentrations than other combustion gases for any particular sample. This is consistent with observations made in previous fire studies: carbon monoxide is produced in virtually all fire environments and typically produced in greater quantities than other combustion gases, excluding carbon dioxide (Lees et al., 1995; Bolstad-Johnson et al., 2000; Jankovic et al., 1991; Austin et al., 2001a, 2001b)

Gases that act as simple asphyxiants (methane, ethylene, acetylene) were observed in relatively low concentrations. However, as noted above, carbon dioxide (another simple asphyxiant) was produced in much larger quantities than any of the other combustion gases but was not included in the plotted figures.

Irritant gases (nitric oxide, sulphur dioxide, acrylonitrile, acrolein, hydrogen chloride, hydrogen fluoride, formaldehyde) were detected in varying amounts, with the type and quantity dependent on the tested material.

2.3.1.4 Comparison of Results to Health Reference Values

To place the reported values into the context of potential health effects, the peak gas concentrations were compared to health reference values relevant to short-term exposures. For this report, the United States Environmental Protection Association (US EPA) Acute Exposure Guideline Levels (AEGL's) were selected.

AEGL values are developed by a U.S. EPA advisory committee and submitted for review and comment to a Toxicology subcommittee that is part of the National Research Council of the National Academies of Science. Once proposed AEGL's have passed through this review stage plus a round of public comment, they are accepted for use by federal agencies in the U.S. (Woodall, 2005). There is no such equivalent acute exposure guideline system in Canada.

AEGL's are designed to be used in rare, accidental exposure scenarios. Three levels of AEGL's exist, based on the severity of potential health effects that may occur after acute chemical exposure (U.S. EPA, 2008):

- AEGL-1 – the threshold airborne concentration of a chemical, above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or other effects. However, the effects are not disabling and are transient and reversible once the exposure stops.

- AEGL-2 – the threshold airborne concentration of a chemical, above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape the contaminated area.

- AEGL-3 - the threshold airborne concentration of a chemical, above which it is predicted that the general population, including susceptible individuals, could experience life-threatening health effects or death.

For each AEGL severity level, airborne concentration values are derived for different durations of exposure: 10 minutes, 30 minutes, 1 hour, 4 hours, 8 hours. A 10-minute exposure to a chemical at an airborne concentration that exceeded the 10-minute AEGL-3 value, for example, could be potentially lethal.

Table 3 compares the peak gas concentrations obtained from the NRC tests with different AEGL values (U.S. EPA, 2008). For each specific gas, the peak concentration values provided in Table 2 were reviewed. The maximum peak value for each gas, out of all of the tested items, was then selected for comparison purposes with the AEGL values. Both AEGL-2 and AEGL-3 values were utilized, as these are most relevant to discussions of serious and potential long-term health effects from short-term exposures.

Table 3: Comparison of Maximum Peak Gas Concentration Values Observed in NRC Tests with Acute Exposure Guideline Levels (AEGLs)

			AEGL-2 [a]			AEGL-3 [b]		
Gas	**Sample with Maximum Peak Gas Concentration**	**Maximum Peak Gas Value**	**10-min**	**1 hour**	**8 hour**	**10-min**	**1 hour**	**8 hour**
CO [c]	Commanding Officer's Cabin Mattress	**380** [d]	420	83	27	1700	330	130
NO [e]	Commanding Officer's Cabin Sleeping Bags	**35**	20	12	6.7	34	20	11
SO_2	Electrical Space Cable – PL-5 Pen-190 (23 mm diam)	**27**	0.75	0.75	0.75	30	30	9.6
HCN	Commanding Officer's Cabin Entrance Door	**13**	17	7.1	2.5	27	15	6.6
Acry	Commanding Officer Cabin Pressure Hull Insulation	**52**	290	57	8.6	480	100	19
Acro	[f]	[f]	0.44	0.10	0.10	6.2	1.4	0.27
HCl	Commanding Officer Cabin Cable - SS-23 (10 mm diam)	**250**	100	22	11	620	100	26
HF	Commanding Officer Cabin Cable – IC-45 (19 mm diam)	**265**	95	24	12	170	44	22
Form	Commanding Officer's Cabin Entrance Door	**17**	14	14	14	100	56	35

Notes:

a. AEGL-2. Acute Exposure Guideline Level 2: the threshold airborne concentration of a chemical, above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape the contaminated area
b. AEGL-3. Acute Exposure Guideline Level 3: the threshold airborne concentration of a chemical, above which it is predicted that the general population, including susceptible individuals, could experience life-threatening health effects or death.
c. CO = carbon monoxide, NO = nitric oxide, SO_2 = sulphur dioxide, HCN = hydrogen cyanide, Acry = acrylonitrile, Acro = acrolein, HCl = hydrogen chloride, HF = hydrogen fluoride, Form = formaldehyde. Methane, ethylene, acetylene, and nitrous oxide are not included in the table as they are considered as simple asphyxiants; no AEGL's exist for these gases since their mechanism of action is dependent on oxygen displacement.
d. All values are in parts per million (ppm)
e. AEGL values are provided for nitrogen dioxide (NO_2); nitric oxide (NO) is oxidized in air to NO_2.
f. Problems with the measurement of acrolein were noted in the NRC cone calorimeter report. Acrolein was detected but was measured in negative concentrations. Further discussions with NRC staff indicated that acrolein was present at low levels, likely less than 1 to 10 ppm. Test items that produced acrolein are noted in Table 2.

The Table 3 comparisons are provided for contextual purposes. Because of the many limitations that were identified earlier, the results of the NRC cone calorimeter gas analyses cannot be assumed to be exactly representative of the HMCS CHICOUTIMI fire atmosphere. The actual gas concentrations that existed on board would have been dependent upon many factors that cannot be accounted for in the NRC tests, such as the quantity of materials burnt, the combined effect of gas production from multiple items burning simultaneously, and the volume of air inside the submarine that the combustion gases dispersed into, for example. The NRC tests only provide information on gas concentrations present in the smoke plume of individual tested items.

As well, it is important to note that AEGL values for specific chemicals only consider exposure to that single airborne chemical on its own. Exposure to a fire atmosphere produced from a fire will entail exposure to multiple chemical products at once. Many of the chemicals produced in fires have similar biological effects, and so their combined effects are additive (Hartzell, 1996; Halperin, 2008; Kimmel et al., 1997; Levin, 1996; Alarie, 2002).

For example, many chemicals produced in fires are respiratory irritants (examples include formaldehyde, acrolein, sulphur dioxide, nitrogen dioxide and other oxides of nitrogen, hydrochloric acid, ammonia, and acetaldehyde); if multiple respiratory irritant chemicals are inhaled at the same time, the effects of each one will essentially add together. Inhaling 50 ppm of hydrochloric acid while at the same time inhaling nitrogen dioxide, sulphur dioxide, acrolein, and formaldehyde would potentially be far more damaging than inhaling 50 ppm of hydrochloric acid on its own.

Therefore, because AEGL values only consider exposures to single chemicals, they likely *underestimate* the potential for health effects when those chemicals are present in a fire atmosphere with other similarly acting chemicals. More realistic AEGL values for a fire atmosphere scenario, when *multiple* gases were present at the same time, would likely be lower than the AEGL values for *single* gas exposures that are provided in Table 3.

Therefore, the comparisons in Table 3 likely *underestimate* the potential for health effects due to exposure to the peak gas concentrations measured in the NRC tests. Even without consideration of this likely underestimation, it is clear from Table 3 that the maximum peak gas concentration

observed in the cone calorimeter tests for several irritant exceeded their respective 10-minute AEGL-2 values, the threshold for potential irreversible or other serious, long-lasting adverse health effects after only 10 minutes of unprotected exposure. These gases included nitric oxide, sulphur dioxide, hydrogen chloride, hydrogen fluoride, and formaldehyde. Acrolein may also have exceeded its 10-minute AEGL-2 value, although this cannot be determined due to measurement difficulties encountered during the cone calorimeter tests. Two gases, nitric oxide and hydrogen fluoride, exceeded their respective 10-minute AEGL-3 values (threshold for potentially life-threatening effects), and sulphur dioxide came very close to its 10-minute AEGL-3 values.

2.3.1.5 Gas Analysis Conclusions

As discussed above, there are limitations to the conclusions that can be drawn from the results of the NRC cone calorimeter tests, and direct extrapolation of these results to the HMCS CHICOUTIMI fire smoke is not possible. However, the results of the NRC tests are suggestive that several minutes of unprotected exposure to the HMCS CHICOUTIMI fire smoke could have resulted in potentially lethal or serious health effects. This conclusion is consistent with what occurred on board HMCS CHICOUTIMI.

2.3.2 Smoke Density

In addition to the combustion gas analyses, the cone calorimeter tests also measured other smoke properties, such as the density, or thickness, of the smoke.

Smoke density is related to the concentration of airborne aerosols (soot) contained within the smoke: the denser the smoke, the higher the airborne aerosol concentration. According to the NRC, the smoke parameter values obtained from the cone calorimeter tests cannot be used directly to obtain a numerical estimate of airborne aerosol concentration. However, the smoke parameter values can be used to make relative comparisons between tested objects.

Table 4 summarizes the values for smoke production rate, total smoke, and specific extinction area for the HMCS CHICOUTIMI tested materials. An explanation of the smoke parameter measurements used in the NRC report is provided in Annex E. Essentially, the higher the numerical values for the smoke parameters reported in Table 4, the “worse” the smoke would be.

Table 4: Summary of Smoke Density Values Obtained From NRC Tests of HMCS CHICOUTIMI Materials

Sample	NETE Number	Peak Smoke Production Rate [a] (m^2/s) [b]	Total Smoke Value, Range [c] (m^2/m^2)	Specific Extinction Area Value, Range [c] (m^2/kg)
CO Cabin Mattress	NETE-13	0.045	22.6 - 76.0	0.27 - 75.8
CO Cabin Briefcase	NETE-14	0.110	1039.6 - 3014.9	186.1 - 401.6
CO Cabin Sleeping Bags	NETE-15 & NETE-16	0.090	158.1 - 394.3	230.9 - 419.3
Elect Space Vent Trunk Insulation	NETE-31	0.076	132.3 - 486.5	68.0 - 277.2
Elect Space Deck Head Insulation	NETE-32	0.125	69.4 - 450.9	144.1 - 688.3
CO Cabin Pressure Hull Insulation	NETE-53	0.015	21.9 - 125.3	39.0 - 171.6
CO Cabin curtain	NETE-54	0.020	2.0 - 15.9	11.3 - 34.3
CO Cabin Entrance Door	NETE-55	0.060	16.5 - 168.4	8.4 - 118.0
Deck Head Insulation [d]	NETE-56	0.018	53.7 - 57.2	17.9 - 216.7
CO Cabin Cable - SS-21 (10 mm diam) [e]	NETE-78 (Test 78-A)	0.070	1640.3	405.6
CO Cabin Cable - SS-23 (10 mm diam)	NETE-78 (Test 78-B)	0.035	1133.3	354.8
CO Cabin Cable – DC-175 (10 mm diam)	NETE-78 (Test 78-C)	0.110	1146.3	452.8
CO Cabin Cable – IC-44 (9 mm diam)	NETE-78 (Test 78-D)	0.065	1210.2	461.4
CO Cabin Cable – IC-45 (19 mm diam)	NETE-78 (Test 78-E)	0.065	1359.2	323.3
CO Cabin Cable - SS-1 (25 mm diam)	NETE-78 (Test 78-F)	0.035	884.7	96.3
CO Cabin Cable – DC-221 (6.5 mm diam)	NETE-78 (Test 78-G)	0.110	797.9	529.0
Elect Space Cable – PL-5 Pen-190 (23 mm diam)	NETE-79 (Test 79-A)	0.028	345.9	57.1
Elect Space Cable – PL-5 Pen-190 (23 mm diam)	NETE-79 (Test 79-B)	0.027	341.4	N/A
Elect Space Cable – PD-130 Pen-168 (33 mm diam)	NETE-79 (Test 79-C)	0.015	218.2	120.1
Elect Space Cable – PD-130 Pen-168 (33 mm diam)	NETE-79 (Test 79-D)	0.008	106.9	74.7
Elect Space Cable – PD-130 Pen-168 (33 mm diam)	NETE-79 (Test 79-E)	0.012	143.7	87.9

Notes:

a. Smoke production rates were plotted in figures in the NRC cone calorimeter report. Similarly to the identification of peak gas concentrations, the peak smoke production rate was identified from the figures. When multiple samples of the test item were tested, the table value represents the highest peak smoke production rate value of all of the sample for that item.
b. A more detailed discussion of smoke parameter units of measurement is provided in Annex A.
c. When multiple samples of the test item were tested, the range of reported values, from lowest to highest, is provided as "minimum - maximum". Only a single value is provided for items with only one sample tested.
d. Combustion gas analysis was not performed for NETE-56 due to a limited amount of provided material.
e. Combustion gas analysis was not performed for NETE 78-A

The highest peak smoke production rates (all greater than 0.1 m^2/s) were observed for the Commanding Officer's Cabin briefcase, the Electrical Space deck head insulation, and two electrical cables from the Commanding Officer's Cabin. The highest values for total smoke were observed for the Commanding Officer's Cabin briefcase and several electrical cables from the Commanding Officer's Cabin. The highest values for specific extinction area were observed for the Commanding Officer's Cabin briefcase, the Commanding Officer's Cabin sleeping bags, the Electrical Space deck head insulation, and the electrical cables from the Commanding Officer's Cabin.

Based on personal accounts of the HMCS CHICOUTIMI crew, the fire rapidly produced very thick black smoke, severely obscuring visibility. This testimony is supported by the Fire Investigator, who concluded that based on the degree of observed smoke damage, visibility would have been almost zero from approximately 1 metre above deck on 1 and 2 deck forward of bulkhead 56 and aft of bulkhead 35 for a short period of time.

The observed smoke parameters of the Commanding Officer's Cabin electrical cable samples, coupled with the fact that the cables were at the source of the very high-temperature electrical arcing event that started the fire, suggests that the electrical cables were significant initial contributors to the thick black smoke that was rapidly produced by the HMCS CHICOUTIJMI fire.

2.4 DRDC-Atlantic Pyrolysis Tests

Prior to the NRC cone calorimeter tests, testing was also conducted on HMCS CHICOUTIMI materials damaged in the fire by Defence Research and Development Canada - Atlantic (DRDC-A). The DRDC-A studies were pyrolysis experiments, and therefore quite different from the combustion studies conducted by NRC.

Combustion refers to the breakdown of a material in the presence of oxygen. Flaming combustion occurs in the presence of a flame, and is the process that is commonly associated with fires. Pyrolysis refers to the breakdown of a material in an atmosphere that does not contain oxygen, such as pure nitrogen, helium, or a vacuum (Orzel, 1993). Because the presence of oxygen is a major determinant of the chemical products released from a fire, there will be "substantial differences between the degradation products found in the pyrolysis experiment and those in an oxygen rich environment" (Hiltz et al., 2004). However, every fire is dynamic, and unless oxygen is actually injected into the fire source, the evolving fire will lack adequate oxygen for "pure" combustion. In a typical fire, such as that aboard HMCS CHICOUTIMI, the

material breakdown process lies somewhere between the two extremes of pure combustion and pure pyrolysis. It is therefore a reasonable assumption that the pyrolysis products identified in the DRDC-A tests could have existed in the atmosphere produced by the HMCS CHICOUTIMI fire (DRDC-A, 2008).

2.4.1 Results

In an interim report, samples of electrical cables from the Commanding Officer's cabin were used in pyrolysis experiments. Although quantities of breakdown products were not provided in the report, the identified chemical compounds that were produced included benzene, toluene, xylenes, styrene, ethenyl-3-methylbenzene, naphthalene, decene, undecene, dodecene, tridecene, tetradecene. These are all hydrocarbon molecules, consisting of carbon and hydrogen, and their presence was "consistent with the degradation of an elastomer containing chlorine" (Hiltz et al., 2004).

It was determined that the cable jacket material was a chlorosulfonated polyethylene. This was the major component of the electrical cable insulation and would have been the major contributor to smoke and gases in a fire that consumed the cable. The report noted that chlorosulfonated polyethylene releases hydrogen chloride when exposed to heat and/or flame. Thermal decomposition of the cable insulation released dense, "sooty" smoke and acid gases into the surrounding atmosphere (Hiltz et al., 2004). These results are consistent with the NRC combustion tests of electrical cable samples, which demonstrated dense smoke production as well as the production of hydrogen chloride, an acid gas.

Further pyrolysis analyses were also conducted. Tested items included the mattress, sleeping bag, curtain, and briefcase from the Commanding Officer's Cabin. The pyrolysis products were consistent with cellulose-based materials such as cotton, polyethylene terephthalate (a thermoplastic polymer), polypropylene, and nylon, and included simple hydrocarbons, benzene, benzoic acid, ethenyl benzoate, esters of 1,4-benzenedioic acid, caprolactam, and 1,6-anhydro-β-d-glucopyranose (levoglucosan) (Veinot et al., 2004).

If present in the HMCS CHICOUTIMI fire atmosphere, the chemicals identified in the DRDC-A pyrolysis studies would have added to the toxicity of the irritant gas mixture suggested by the results of the NRC tests.

2.5 Additional HMCS CHICOUTIMI Fire Atmosphere Considerations

The NRC cone calorimeter tests provide information on some of the combustion gases that may have existed in the HMCS CHICOUTIMI fire atmosphere. However, as previously noted, it was not possible to measure "every" combustion product that was produced in these tests. It is highly likely that many more chemical compounds were released during the fire on board HMCS CHICOUTIMI, which is not reflected in the NRC tests. For example, the DRDC-A pyrolysis studies suggest that several different chemicals that were not measured in the NRC tests could have been present in the fire atmosphere. Several other considerations regarding the HMCS CHICOUTIMI fire atmosphere are provided below.

The electrical arcing event that produced the HMCS CHICOUTIMI fire would likely have resulted in exposures that may not usually be observed in other fires. For example, ozone is product of electrical currents passing through the air, such as in welding (Rabinowitz et al., 2002). As well, exposure of chlorinated products to high heat electrical arc sources can result in the production of phosgene gas (Orzel, 1993). Phosgene gas was not detected in the NRC tests, however the ignition temperatures used in these tests were lower than the high temperatures associated with an electrical arc. Both ozone and phosgene are highly toxic irritant gases, and would have added to the toxicity of the irritant gas mixture suggested by the NRC tests.

As previously noted, some of the materials tested by the NRC had surface finishes that were almost completely burned off in the fire; these finish materials could therefore not be included in the tests. These surface finishes could have included adhesive materials used to secure insulation to the hull of the submarine. According to Director Maritime Ship Support (DMSS) 2-4, the epoxy adhesives used on board HMCS CHICOUTIMI included peridite and sigmacover pitakote. These products made up a very small part of the material burned in the fire, and potential combustion products would have included carbon monoxide, carbon dioxide, nitric oxide, nitrogen dioxide, hydrogen cyanide, and phenol (DMSS 2-4, 2008). Of these potential combustion products, phenol is the only one not previously discussed. Phenol would have added to the toxicity of the irritant gas mixture suggested by the NRC tests.

As noted in Section 2.1.5, fires produce aerosol particles (soot). Although the NRC tests assessed smoke density, which is an indirect measure of aerosol content, the actual quantity and chemical composition of those aerosol particles was not determined. It is reasonable to conclude that the HMCS CHICOUTIMI fire smoke would have contained aerosolized chemicals that are commonly produced in fires, such as elemental carbon, polycyclic aromatic hydrocarbons, dioxins and furans, and fibres (such as cellulose and fibrous glass). As well, the electrical arcing event on board HMCS CHICOUTIMI was hot enough to burn through a metal deck plate. High temperature vapourization of metals leads to the production of metal fumes, which are very small particles of condensed metal vapour. Small metal particles were likely present in the HMCS CHICOUTIMI fire atmosphere. The soot (aerosol particles) produced by the fire remained on interior submarine surfaces after the airborne smoke was cleared. An analysis of this leftover soot is provided in Section 4.0.

2.6 Conclusions

Fires are complex and dynamic, and it is virtually impossible to determine the exact chemical composition of a fire atmosphere at any given location or time. The HMCS CHICOUTIMI fire atmosphere is no different in this respect. Although investigations such as those conducted by the NRC provide some insight, the exact chemical composition of the HMCS CHICOUTIMI fire atmosphere will never be known with complete certainty. Despite this uncertainty, however, some general conclusions can be made about the HMCS CHICOUTIMI fire atmosphere, based on the information sources discussed above as well as general knowledge regarding fire atmospheres.

The concentrations of combustion products would have been greatest during the time that the fire was burning, when these chemical products were being produced by the fire. In an enclosed space such as a submarine, these high concentrations of combustion products would likely have diminished very little in the absence of active ventilation. However, once active ventilation commenced through the use of the starboard engine, the atmosphere on board HMCS CHICOUTIMI rapidly improved within hours. This is evident from the Draeger tube results, discussed in Section 2.2. With continuous ventilation on board throughout the transit back to Faslane, it is likely that possible exposure to any residual gases or vapours produced from the fire would have been extremely small.

The NRC cone calorimeter tests of HMCS CHICOUTIMI materials cannot be directly used to "re-create" the fire atmosphere on board. However, the results of the NRC tests are suggestive that several minutes of unprotected exposure to the HMCS CHICOUTIMI fire smoke could have resulted in potentially lethal or serious health effects. This conclusion is consistent with what occurred on board HMCS CHICOUTIMI.

Although the exact chemical composition of the HMCS CHICOUTIMI fire atmosphere will never be known, sufficient exposure to the gases identified in the NRC tests alone would likely have resulted in acute, and potentially severe, smoke inhalation injury.

3.0 Smoke Inhalation

The complex atmosphere produced by a fire is well recognized to be highly toxic. Smoke inhalation is the leading cause of death from fires (Rabinowitz et al., 2002; Miller et al., 2003; Kimmel et al., 1997; Wald et al., 1987; Schwartz, 2005).

As discussed in Section 2.6, the exact chemical composition of the HMCS CHICOUTIMI fire atmosphere will never be known with certainty. Each individual crewmember's exposure to the chemicals contained within the fire atmosphere will also never be known with absolute certainty. This situation is not unusual, since detailed exposure information is rarely, if ever, known for smoke inhalation survivors.

Although the combustion products created by fires are numerous, the body's response to combustion product exposure is a relatively narrow range of symptoms and injuries. The severity of smoke inhalation injury is dependent upon the degree of exposure to the smoke. Exposure will be dependent upon:

- the amount of combustion products in the smoke (airborne concentration); and
- the amount of smoke that was inhaled, which will be dependent upon breathing rate and the length of time over which exposure occurred.

These factors will determine the dose of smoke that an individual received: the greater the dose, the more severe the health effects. Therefore, the severity of smoke inhalation injury acts as a clinical index of exposure to known chemicals (such as those identified by the NRC tests) as well as the unknown chemicals present in the HMCS CHICOUTIMI fire atmosphere.

The following sections discuss the potential acute and long-term health consequences of smoke inhalation.

3.1 Acute Effects

The heat and chemical content of the smoke can result in immediate symptoms and injury. The hot dry air can cause direct thermal burns to the upper respiratory tract. Because the upper respiratory tract is so efficient at heat exchange and cooling the inhaled hot air, thermal injuries typically only affect the respiratory system below the level of the throat (Wald et al., 1987; Rabinowitz et al., 2002; Schwartz, 2005). Burns to the upper airway can lead to tissue swelling and potential blockage of the upper airway, which is a life-threatening complication.

Although thermal injury to the upper airway may occur among those in close proximity to the fire source, immediate symptoms related to smoke inhalation are more commonly due to the effects of inhaled asphyxiants (both simple and chemical) and irritants (Haponik, 1993; Schwartz, 2005).

Depending on the degree of exposure, the asphyxiants within smoke can result in headache, dizziness, light-headedness, nausea, and confusion. If exposure is great enough, asphyxiants can rapidly lead to unconsciousness followed by death within minutes if the individual is not rescued from the smoke environment. The greatest short-term threat to life in a smoke environment is carbon monoxide, a chemical asphyxiant, which is produced in virtually all fire environments at relatively high concentrations. Carbon monoxide poisoning is the most frequent immediate cause of death in fire victims (Wald et al., 1987; Lee, 2006).

Irritant gases that can be produced by a fire are numerous. These gases are highly reactive and undergo chemical reactions with protein and lipid components of the cells that they come into contact with, leading to cellular damage. For example, nitric oxide is readily oxidized to nitrogen dioxide, which generates free radicals that destroy the lipid components of cellular membranes. Gases such as hydrogen chloride and hydrogen fluoride react rapidly with moisture to form hydrochloric acid and hydrofluoric acid, respectively, which cause immediate damage to cellular components. If the cellular damage from irritant gas exposure is severe enough, cellular death will result.

For irritants, the location and timing of the injury will depend on the properties of the irritant as well as the degree of exposure. Irritant gases that are water-soluble react immediately when they some into contact with moist tissues, such as the eyes or respiratory tract. Examples of water-soluble irritant gases include sulphur dioxide, hydrogen chloride, hydrogen fluoride, acrolein, and formaldehyde. Exposure to these gases will result in immediate eye and upper respiratory tract (nose, mouth, throat) symptoms. Because these gases react immediately with moist tissues, the site of injury is usually restricted to the upper airway.

However, if the exposure is severe enough, water-soluble irritant gases can cause injury lower down in the respiratory system (Schwartz, 2005; Blanc et al., 2005). As well, water-soluble

irritant gases may adsorb onto smoke aerosol particles, allowing them to travel more deeply into the respiratory tract than they otherwise would (Wald et al., 1987).

Gases that are poorly soluble in water, such as oxides of nitrogen and phosgene, can pass through the upper respiratory tract without producing any symptoms, but instead cause damage primarily deep in the respiratory tract.

Although a fire may produce a large variety of irritant gases, the body's response to these exposures is a relatively limited range of symptoms and injuries (Haponik, 1993; Blanc et al., 2005; Miller et al., 2003).

Immediate symptoms related to irritant exposures can include a burning sensation in the eyes, nasal passages, and throat. Watery eyes, coughing, sneezing, and hoarseness may also result. If the irritant exposure is severe enough, direct injury to the cellular mucosa lining the upper airway can lead to sloughing of dead cells, increased mucous secretions, and swelling of the underlying tissues due to the body's inflammatory response to tissue injury. These changes, which may take place over several hours, can lead to obstruction of the upper airway, a potentially life-threatening complication.

Irritant injury to the conducting airways, farther down in the respiratory tract, can lead to constriction (narrowing) of these airways, resulting in symptoms such as wheezing, cough, and shortness of breath. This airway constriction may happen shortly after exposure, as a reflex in response to irritation of nerve endings in the airways, or may develop after several hours due to the body's inflammatory response to tissue injury.

Injury to the lower respiratory tract may damage the small airways and gas-exchange structures (alveoli and supporting tissues). This may manifest as cough, shortness of breath, and an impaired ability to absorb oxygen into the bloodstream. The damage, and the body's subsequent inflammatory response in the hours that follow, can lead to the release of fluid into the alveolar spaces of the lungs, where gas-exchange occurs. This condition, referred to as pulmonary edema, can be life-threatening since it greatly impairs the ability of the lungs to transfer oxygen to the body's tissues (Schwartz, 2005; Rabinowitz et al., 2002; Miller et al., 2003).

Toxic gases that may be present in the fire environment are the major inhalational toxins and account for virtually all of the clinically recognized respiratory effects of smoke inhalation (Haponik, 1993; Schwartz, 2005). The aerosol particles that are present in smoke will also have irritant properties. Aerosol particles that are present in smoke are generally cleared by the respiratory tract, such as through sputum production. Dense, black, carbonaceous secretions may be coughed up following smoke inhalation for a considerable period of time. However, the presence and duration of carbonaceous secretions does not correlate well with the presence or severity of inhalation injury (Haponik, 1993). In other words, coughing up large amounts of black sputum does not necessarily imply that a severe smoke inhalation injury has occurred.

As noted in Section 2.5, the electrical arcing event that started the HMCS CHICOUTIMI fire was hot enough to burn through a metal deck plate. High temperature vapourization of metals leads to the production of metal fumes, which are very small particles of condensed metal

vapour. Inhalation of some metal fumes, particularly zinc oxide, can lead to a condition known as metal fume fever. Metal fume fever is characterized by a dry throat and/or a metallic taste in the mouth, followed by flu-like symptoms such as fever, chills, nausea, headache, and muscle aches several hours later. Cough, chest tightness, and shortness of breath may also occur. These symptoms are only temporary, and resolve completely within one to two days (Schwartz, 2005). Some metal oxides, such as cadmium oxide, are capable of inducing a more severe lung injury if inhaled in sufficient quantities. This type of injury manifests as pulmonary edema (described above), which can be life-threatening.

Because the body's inflammatory response to injury may take several hours to develop, the full extent of the severity of smoke inhalation injury may not be known until 24 to 72 hours after exposure (Wald et al., 1987; Lee et al., 2006).

Exposure to smoke entails exposure to a complex mix of gases and aerosol particles. Although the exact composition of a fire atmosphere cannot be known with certainty, asphyxiant and irritant exposures are common to all fires and result in acute symptoms and effects. Generally speaking, the severity of the acute symptoms described above provides a good clinical index of the amount of exposure to the smoke produced by a fire. If the initial symptoms were relatively mild, it can be safely concluded that the amount of exposure to the smoke was also relatively mild.

3.2 Long-term Effects

The acute, short-term effects of smoke inhalation injury described in the preceding section may range from mild symptoms, such as cough, to life-threatening complications, such as pulmonary edema. Once the full severity of the initial smoke inhalation injury has become apparent, the body's natural healing and repair processes proceed. Although full recovery from smoke inhalation injury is expected, even in severe cases (Haponik, 1993; Alberts et al., 1996), some smoke inhalation survivors may not recover completely. Generally speaking, those who sustain a more severe acute inhalation injury are at greater risk of having long-term health problems. Persistent abnormalities are rarely observed among individuals with mild inhalation injury (Haponik, 1993, Blanc et al., 2005).

The discussion that follows explores the potential long-term health implications of smoke inhalation injury and exposure to chemicals produced in a fire, based on existing medical and scientific literature. As explained in Section 1.3, personal medical information is not discussed in this report. Subsequently, the sections that follow describe possibilities, not individual predictions. Guidance regarding the application of this information to specific individuals, based on their own individual clinical experience, is included in the discussion that follows.

Most of the long-term health problems identified in smoke inhalation survivors are related to the respiratory tract, and these are discussed below. This is not unexpected, considering that the respiratory tract is the main site of injury in smoke inhalation. Effects of smoke inhalation on other organ systems have also been observed, and are discussed below.

3.2.1 Pulmonary Function

Impairment in pulmonary function and airway hyper-responsiveness are the most commonly identified persistent abnormalities after an irritant inhalation injury (Blanc et al., 2005). Acute changes in pulmonary function following smoke inhalation are well recognized (Haponik, 1993; Scannell et al., 1995). These changes are typically manifest as increased airways obstruction to expiratory airflow, and can result in symptoms such as wheeze or shortness of breath. Airway obstruction can also be measured clinically through pulmonary function tests, which may reveal decreases in forced expiratory flow and forced expiratory volume in 1 second (FEV_1). Pulmonary function changes suggestive of a restrictive impairment, such as a decline in forced vital capacity (FVC), have also been noted (Kimmel et al., 1997).

Pulmonary function typically returns to normal within a few weeks to months (Cha et al., 2007; Bourbeau et al., 1996). However, there are many reports of persistent pulmonary function decrements as well as greater than normal rates of decline in pulmonary function among smoke inhalation survivors (Weiden et al., 2007; Scannell et al., 1995; Blanc et al., 2005).

It has been suggested that any residual pulmonary function impairment that is evident at six months post-injury is unlikely to improve substantially (Kimmel et al., 1997). The severity of persistent pulmonary function impairments may range from mild and clinically insignificant to potentially disabling. The possibility of persistent pulmonary function problems is essentially restricted to individuals who had objective impairment on pulmonary function tests performed shortly after a fire. If these tests are normal several months after smoke inhalation, then lung function impairment would not be expected to occur as a result of the smoke inhalation.

3.2.2 Reactive Airways Dysfunction Syndrome (RADS)

One of the more commonly observed long-term complications of smoke inhalation injury is airway hyper-responsiveness (also referred to as airway hyper-reactivity or airway hypersensitivity), which is manifest as episodes of widespread airway narrowing in response to inhaled stimuli (Blanc et al., 2005; Haponik, 1993; Rabinowitz et al., 2002). This reactive airway narrowing can lead to symptoms of cough, wheeze, and shortness of breath. These symptoms, and airway hyper-responsiveness, are synonymous with asthma. Irritant-induced asthma and Reactive Airway Dysfunction Syndrome (RADS) are names that have been given to the development of airway hyper-responsiveness following an acute irritant inhalation injury, such as smoke inhalation. Individuals with RADS may have completely normal pulmonary function when they are not having an asthma “attack”.

The exact mechanism of RADS is unclear. It has been postulated that repair mechanisms in response to the irritant injury of the airways leads to increased reactivity: subsequent exposure to irritants or other stimuli leads to an over compensatory airway reaction (Schwartz, 2005)

RADS was first formally described in 1985, although the presence of persistent asthma following irritant inhalation injury had previously been identified (Brooks et al., 1985). The criteria required for a diagnosis of RADS included high exposure to a respiratory irritant (such as fire

smoke), new onset of symptoms within 24-hours after a single exposure event, persistence of symptoms beyond three months, symptoms consistent with asthma (such as cough, wheeze, or shortness of breath), and positive tests of airway hyper-responsiveness (such as methacholine challenge testing) (Brooks et al., 1985). In one of the cases described by Brooks et al. (1985), RADS developed after only a ten to fifteen minute smoke inhalation exposure.

It is difficult to predict the development of RADS following a high level irritant exposure (Alberts et al., 1996). In some cases, no cases of RADS have been observed among cohorts with significant irritant exposures, including smoke inhalation (Bourbeau et al., 1996; Hickmann et al., 2001). In other instances, however, the reported prevalence of RADS can be fairly high: 18% of firefighters had a diagnosis of asthma or bronchitis 22 months after a polyvinyl chloride (PVC) fire (Markowitz, 1989), and 28% of highly-exposed World Trade Center firefighters had persistent airway hyper-reactivity 6 months after exposure (Banauch et al., 2003).

Although irritant exposures severe enough to induce RADS typically result in the affected individual seeking immediate medical attention (Alberts et al., 1996), RADS has been reported following smoke exposure even though the affected individual did not recall any immediate symptoms (Brooks et al., 1985). A dose-response effect has been observed with RADS, in that the more intense the irritant exposure or initial symptoms, the greater the risk of development of RADS (Kern, 1991; Banauch, et al., 2003). However, this is not always evident; in a small case series of smoke inhalation patients, there was no clear relationship between airway reactivity and either the degree of smoke exposure or initial lung function impairment (Kinsella et al., 1991).

The natural history of RADS is quite variable. Some cases resolve within months to a few years, whereas some case series have observed the persistence of RADS beyond 12 years (Brooks et al., 1985; Demeter et al., 2001; Piirila et al., 1996; Schwartz et al., 1990). It has been suggested that if asthma symptoms do not clear within 6 months, then they are likely to persist for several years (Alberts et al., 1996). The majority of individuals with RADS improve over time, although a small minority of individuals may experience worsening of their symptoms with time (Demeter et al., 2001; Kinsella et al., 1991; Bardana, 1999).

If RADS has developed following an inhalation injury, then the signs and symptoms of this condition are apparent shortly after injury. Tests of airway hyper-responsiveness (such as methacholine challenge testing) that are performed several weeks to months after a fire have a very high negative predictive value: if such tests are normal, then RADS can be ruled out as a possible long-term health complication of smoke inhalation.

3.2.3 Upper Airway

The mucous membranes of the nose and throat are common sites of initial injury and subsequent inflammation following smoke inhalation. Although full recovery is expected in most cases, persistent problems have been reported following irritant inhalation injuries, such as chronic rhinitis, sinusitis, pharyngitis, and laryngitis (Rabinowitz et al., 2002; Haponik, 1993). Symptoms can include nasal or sinus congestion, facial tenderness, post-nasal drip, sore throat, hoarseness, and difficulty swallowing.

A syndrome of chronic rhinitis/sinusitis and/or throat irritation that is triggered by exposure to inhaled irritants may occur following an acute inhalation injury. This condition as been termed Reactive Upper Airways Dysfunction Syndrome (RUDS), and is considered to be the upper airway equivalent of RADS (Weiden et al., 2007; Rabinowitz et al., 2002). RUDS is often observed in conjunction with RADS following acute inhalation injury, and the prognosis and natural history of the two conditions may be similar (Meggs et al., 1996; Demeter et al., 2001).

Another condition that has been described following irritant exposure is vocal cord dysfunction (Rabinowitz et al., 2002; Shusterman, 1999). This condition is characterized by inappropriate vocal cord closure, leading to episodic symptoms of hoarseness, changes in voice pitch, wheezing, or stridor (a high-pitched sound resulting from turbulent airflow in the upper airway). Vocal cord dysfunction may co-exist with asthma, and is occasionally misdiagnosed as asthma (Shusterman, 1999).

Persistent upper airway symptoms may occur after smoke inhalation injury, but this possibility only exists if these symptoms are present shortly after smoke inhalation. If such symptoms are absent several weeks to months after smoke inhalation, then no long-term upper airway symptoms as a result of smoke inhalation would be expected to develop.

3.2.4 Pulmonary Infections

Damage to the lungs from smoke inhalation injury can cause impairment of natural defence mechanisms against infection. Survivors of smoke inhalation injury can be at greater risk of developing lung infections, such as pneumonia (Wald et al., 1987; Fogarty et al., 1991; Wolter et al., 2005). This risk is greatest during recovery of the acute injury; an increased risk of pulmonary infection would not be expected after recovery from smoke inhalation injury.

3.2.5 Structural abnormalities

In rare cases, survivors of smoke inhalation injury develop structural lung abnormalities. Such abnormalities include bronchiectasis (localized, irreversible dilatation of airways as a consequence of inflammation and destruction of the structural components of the bronchial wall), bronchiolitis obliterans (a lung disease characterized by fixed airway obstruction secondary to inflammation and scarring of small airways), bronchiolitis obliterans with organizing pneumonia (BOOP) (chronic inflammation of the small airways and surrounding tissue), and pulmonary fibrosis (the formation of fibrous scar tissue in the lungs) (Blanc et al., 2005; Schwartz, 2005; Rabinowitz et al., 2002; Haponik, 1993; Shusterman, 1999; Weiden et al., 2007).

These outcomes are typically observed only in individuals who experienced a significant lung injury as a result of irritant inhalation, often requiring prolonged hospitalization, and even among such cases these outcomes are rare (Loke et al., 1980; Slutzker et al., 1989; Tasaka et al., 1995; Blanc et al., 2005). Subsequently, it is unlikely that any HMCS CHICOUTIMI crewmembers would develop any of these outcomes.

3.2.6 Sarcoidosis

Sarcoidosis is a multisystem granulomatous disease that predominantly affects the lungs. Lung manifestations include lymph node enlargement and inflammatory infiltration of lung tissue. It has recently been reported that sarcoidosis is found more frequently in firefighters as compared to other emergency workers (Prezant et al., 1999). As well, among New York City firefighters who responded to the World Trade Center attacks, the incidence of sarcoidosis increased in the years following the attacks (Izbicki et al., 2007).

It is unclear from these two studies if survivors of single acute smoke inhalation exposures are at greater risk of developing sarcoidosis, given that a) professional firefighters would experience multiple smoke exposure episodes, and b) exposures at the World Trade Center were not limited to possible smoke inhalation, but also included respiratory exposure to large quantities of dust, including alkaline concrete dust, gypsum, and glass fibres (Blanc et al., 2005; Weiden et al., 2007). As well, there are no other published reports of sarcoidosis developing in smoke inhalation survivors.

3.2.7 Gastrointestinal

Gastrointestinal disorders arising as a result of acute smoke inhalation injury have only rarely been described. In a small cohort of RADS patients, gastrointestinal symptoms were noted in several individuals, often persisting beyond several months (Demeter et al., 2001). In a recent study of World Trade Centre firefighters, persistent gastroesophageal reflux disease (GERD) was reported by 45% of firefighters with persistent airway hyperreactivity 6 months after exposure, whereas only 29% of firefighters who did not have persistent airway hyperreactivity reported symptoms of GERD (Banauch et al., 2003). The predominant symptom of GERD is heartburn.

It is unclear if GERD can be directly related to smoke inhalation injury, since exposures at the World Trade Center were not limited to possible smoke inhalation, but also included exposure to large quantities of dust, including alkaline concrete dust, gypsum, and glass fibres (Blanc et al., 2005; Weiden et al., 2007). It has been suggested that GERD in this setting may be due to ingestion of airborne or expectorated dusts, possibly exacerbated by increased psychosocial stress and shift-work related dietary indiscretions (Weiden et al., 2007).

It is unknown if GERD is unique to the World Trade Center exposure or represents a previously unrecognized aspect of inhalation injury in general (Banauch et al., 2005).

3.2.8 Cardiac

Heart injury related to smoke inhalation has been shown to occur. For example, firefighters may be at greater risk of suffering an acute cardiac event, such as a heart attack, at the time of firefighting activities or shortly thereafter, potentially due to carbon monoxide poisoning and/or the acute physical stress of firefighting (Weiden et al., 2007; Kales et al., 2003).

The heart and brain are the two main target organs for damage due to carbon monoxide poisoning (the effects on the brain are discussed in Section 3.2.9). The main toxic effects of

carbon monoxide on the heart are decreased oxygen delivery to heart muscle tissue and impairment of enzymes necessary for heart muscle function. This can lead to heart rhythm abnormalities, heart muscle injury, impaired heart function, and in severe cases, heart muscle tissue death (also known as myocardial infarction, or "heart attack") (Gandini et al., 2001; Satran et al., 2005; Aslan et al., 2006; Rosenman, 2007; Kalay et al., 2007).

If any of these effects on the heart due to carbon monoxide poisoning were to occur, they would occur at the time of exposure. Alterations in heart muscle function related to carbon monoxide poisoning are usually short-lived, and full recovery in heart function may occur as quickly as 24 hours after exposure (Kalay et al., 2007). In one case report of an individual with smoke inhalation injury, transient changes in heart function were observed, but these completely reversed and heart function returned to normal within four days (Jan et al., 2007).

The risk of toxic effects on the heart due to carbon monoxide poisoning is related to the severity of poisoning. For example, a recent study examined 230 patients with carbon monoxide poisoning severe enough to require hospitalization (Satran et al., 2005). For 42 of these 230 patients, their source of carbon monoxide exposure was a fire (Henry et al., 2006a), while the remaining 188 patients had been exposed to carbon monoxide from other sources (such as leaking furnaces or domestic water heaters, for example). As a group, these 230 patients had experienced substantial carbon monoxide poisoning: 81% of the patients had lost consciousness as a result of their carbon monoxide exposure, and 50% had to be intubated (a tube placed down their throat as a life support measure) in hospital. In this patient population, 37% of individuals had evidence of heart muscle injury (Satran et al., 2005). The risk of heart muscle injury was higher in individuals with high blood pressure and those who had an altered level of consciousness due to carbon monoxide poisoning (Satran et al., 2005).

A different study included 83 carbon monoxide poisoned patients, 63% of whom had lost consciousness as a result of their exposure. Heart muscle damage was only observed in patients with significant levels of carbon monoxide exposure, and only in those patients who had experienced a loss of consciousness due to carbon monoxide poisoning (Aslan et al., 2006).

There have been few long-term studies that have examined the long-term cardiac effects of acute carbon monoxide poisoning. Although firefighters are at greater risk of acute cardiac events at the time of firefighting, currently there is no convincing scientific evidence that firefighters are at greater risk of developing chronic cardiovascular disease (Guidotti, 1995; Weiden et al., 2007). The 230 patients mentioned above (Satran et al., 2005) were included in the only study to examine long-term mortality outcomes in patients hospitalized with moderate to severe carbon monoxide poisoning. After a median follow-up period of 7.6 years, those patients who had experienced a heart injury at the time of carbon monoxide poisoning were twice as likely to die during the follow-up period than patients who had not experienced an acute heart injury at the time of carbon monoxide exposure (Henry et al., 2006b). Other significant risk factors for increased long-term mortality in this study were older age, and a history of diabetes, high blood pressure, coronary artery disease, and congestive heart failure.

In summary, acute carbon monoxide poisoning can lead to impaired heart muscle function and heart muscle damage. The risk of heart muscle damage is dependent on the degree of carbon

monoxide exposure; heart damage is most likely to occur in individuals with significant carbon monoxide poisoning (Satran et al., 2005; Aslan et al., 2006). In addition to exposure, individual susceptibility is also important, since those with pre-existing heart disease or older age are also at greater risk for heart damage from carbon monoxide exposure (Gandini et al., 2001; Prockop et al., 2007). Long-term heart problems appear to only occur in individuals who have sustained an acute heart injury shortly after moderate to severe carbon monoxide poisoning (Henry et al., 2006b).

Whether or not HMCS CHICOUTIMI crewmembers experienced temporary heart function abnormalities as a result of carbon monoxide exposure from the fire cannot be definitively assessed. Such abnormalities may only last for a few days, and detailed cardiac studies (such as blood tests, echocardiograms, electrocardiograms) were not performed on the crewmembers who remained on board during the 5-day transit to Faslane, Scotland, after the fire. However, in the absence of (i) pre-existing heart disease, (ii) symptoms of significant carbon monoxide poisoning (such as loss of consciousness), or (iii) signs and symptoms of a heart attack at the time of exposure, the possibility of an increased risk of long-term heart problems in crewmembers seems highly unlikely.

3.2.9 Central Nervous System

During a fire, production of simple asphyxiant gases such as carbon dioxide can displace the oxygen that is in the air. If oxygen levels drop too low, individuals in that atmosphere can rapidly lose consciousness. If an individual remains unconscious in this oxygen-deprived atmosphere for several minutes, permanent brain damage may result if the individual can be revived successfully.

Significant exposure to combustion product chemical asphyxiants, such as carbon monoxide and hydrogen cyanide, can also lead to neurologic injury in survivors (Shusterman, 1993). Potential neurologic outcomes following carbon monoxide poisoning are numerous and have recently been reviewed (Hopkins et al., 2006). Cognitive problems (such as impaired memory, impaired executive function, slow mental processing speed, and decreased intellectual function) lasting one month or more may occur in 25%-50% of patients with loss of consciousness due to carbon monoxide poisoning. Among reviewed studies of carbon monoxide-poisoned patients (a total of 979 patients, 30% of whom had loss of consciousness), cognitive impairments were reported in 25 to 70% of studies, with impaired memory being the most frequent type of cognitive impairment reported (Hopkins et al., 2006).

Cognitive impairments can occur after less severe carbon monoxide poisoning. In one of the few studies to explore this, students exposed to 17 to 100 ppm of carbon monoxide for 1.5 to 2.5 hours had lower scores on neuropsychological tests, such as test of memory, attention, concentration, visuomotor skills, and visuospatial planning (Amitai et al., 1998). In this study, the neuropsychological tests were administered during carbon monoxide exposure, so it is unclear how long the cognitive impairments lasted. As well, the methodology of this study and the interpretation of its results have been criticized (Bleeker, 1999).

In a separate study of patients with a higher level of carbon monoxide poisoning than the Amitai et al. (1998) study, but not high enough to result in immediate neurologic injury, there was no evidence of memory impairment on tests performed one month after the poisoning episode (Deschamps et al., 2003). In a study of 343 patients with mild carbon monoxide poisoning (defined as no loss of consciousness), self-reported cognitive problems at one month after the exposure event included feeling weak (26%), headaches (19%), impaired memory function (9%), sleep disorders (8%), difficulties concentrating (5%), behavioural impairment (4%), and visual disturbances (4%). Neurological examinations of these patients were normal, and 97% were back at work within one month, leading the authors to suggest that even if patients were experiencing some degree of cognitive problems, they "must be quite minor" (Annane et al., 2001).

Cognitive problems from carbon monoxide poisoning can occur immediately and persist, or develop after a period of time. Delayed cognitive problems occur in 0.06% to 40% of carbon monoxide poisoned individuals, with the onset occurring 2 to 40 days after poisoning (Hopkins et al., 2006). Cognitive problems that develop more than 6 weeks after exposure would not be expected to be due to carbon monoxide poisoning (Choi, 1983; Weaver et al., 2002, 2007).

Several other adverse neurological outcomes have been reported following carbon monoxide poisoning, including visual field defects, movement disorders (Parkinsonism, choreoathetosis), and Gilles de la Tourette's syndrome. These outcomes are rare and typically develop within days to weeks of significant carbon monoxide poisoning. They are associated with structural brain lesions that are observable with imaging modalities such as computed tomography (CT) and magnetic resonance imaging (MRI) scans (Hopkins et al., 2006).

Mood changes following carbon monoxide poisoning have also been reported, but findings across studies have been mixed, owing to differences in patient populations and assessment methods (Hopkins et al., 2006). A generally consistent finding in these studies has been high rates of depression and anxiety in carbon monoxide poisoned patients. In a recent prospective study, the prevalence of self-reported depression and anxiety at 6 weeks, 6 months, and 12 months after carbon monoxide poisoning was 45.2%, 44.4%, and 42.5%, respectively, out of all 127 patients studied (Jasper et al., 2005).

The prevalence of depression and anxiety differed across patient subgroups in this study: patients whose carbon monoxide poisoning was due to a suicide attempt were four times more likely to have depression and anxiety than those who were accidentally exposed; patients with cognitive problems due to carbon monoxide poisoning were three times more likely to report depression and anxiety than those with no cognitive problems (Jasper et al., 2005). The authors of this study noted that theirs as well as similar studies on affective changes related to carbon monoxide poisoning are limited by an inability to quantify pre-poisoning mood status, a lack of structured clinical interviews, and a lack of an appropriate control group.

The risk of developing persistent neurological problems after carbon monoxide exposure depends on the amount of carbon monoxide exposure. However, markers of high exposure, such as loss of consciousness, do not reliably predict later cognitive impairments (Hopkins et al., 2006). Some risk factors for the development of persistent cognitive problems that have been

identified include older age, exposure to carbon monoxide for 24 hours or longer, and initial memory complaints around the time of exposure (Weaver et al., 2007).

In general, smoke inhalation survivors who do not have cognitive symptoms within weeks of exposure would not be expected to develop cognitive difficulties years down the road as a result of the smoke exposure.

3.2.10 Summary

The above discussion summarizes some of the chronic health problems that have either been reported following smoke inhalation injury or exposure to chemicals that are commonly found in fire smoke, such as carbon monoxide. It is worth reiterating that even in severe cases, full recovery from smoke inhalation injury is expected and persistent abnormalities are rare among individuals with mild inhalation injury (Haponik, 1993; Alberts et al., 1996; Blanc et al., 2005).

As discussed above, some of the more common chronic health problems related to smoke inhalation injury include impairments in pulmonary function, RADS, and possible cognitive problems, such as memory loss, related to carbon monoxide exposure. In general, individuals who go on to develop long-term health problems from smoke inhalation show signs of these problems within hours to weeks after smoke inhalation. Those who are clinically well several months after smoke inhalation are essentially "in the clear" and would be expected to remain healthy.

For those with persistent signs and symptoms in the weeks following smoke inhalation injury, the prognosis is less clear. Although some will improve over time, signs and symptoms may be present for years and can potentially worsen. Therefore, those with persistent signs and symptoms in the weeks following smoke inhalation injury require appropriate medical care and follow-up.

3.3 Cancer

Cancer is a common disease. It is estimated that Canadian males have a 44.5% chance (1 in 2.2) of developing cancer in their lifetime, and a 28.5% chance (1 in 3.5) of dying of cancer (CCS/NCIC, 2008). Although the exact cause of cancer is often not known, some factors are known to increase cancer risk, such as smoking, dietary factors, genetics, and exposure to chemical carcinogens.

Some chemicals are recognized as being capable of causing cancer, and are referred to as carcinogens. Sufficient exposure to chemical carcinogens can lead to the development of cancer many years after exposure occurred, even though the exposed individual was clinically well in the years leading up to the diagnosis.

Simply being exposed to a chemical carcinogen does not mean that cancer will inevitably develop. A critical factor in assessing the risk of developing cancer after exposure to a carcinogen is the dose, or amount, of exposure: the greater the amount of exposure, then the greater the risk of developing cancer. We are all exposed to very low levels of chemical

carcinogens (many of them occurring in nature) through the food we eat, water we drink, and air we breathe. These low exposures are considered to be non-harmful, since the theoretical risk of developing cancer from such exposures is so low that it is essentially non-existent.

Several chemical compounds that are common produced in virtually all fires, such as benzene, polycyclic aromatic hydrocarbons (PAHs), and dioxins and furans, are also recognized chemical carcinogens. The exact types and quantities of these chemicals that may have been present in the fire atmosphere on board HMCS CHICOUTIMI is unknown, but it is reasonable to conclude that the fire atmosphere contained chemical carcinogens and that the crew were exposed to them.

An important question is: were these exposures large enough to result in a significantly increased risk of developing cancer? It is impossible to answer this question with complete certainty, simply because exact exposures for HMCS CHICOUTIMI crewmembers are unknown. However, this risk can be reasonably qualified by comparing the HMCS CHICOUTIMI crew to studies of individuals who have also experienced exposure to the multiple chemical products contained within fire smoke.

No studies exist that have examined long-term cancer risk among survivors of single, major fires. However, a relevant occupational group for comparison purposes is firefighters.

Studies of cancer risk in firefighters are numerous. Many of these studies examine populations of firefighters dating back to the 1920's, when the availability and use of modern protective equipment, such as self-contained breathing apparatus (SCBA), differed greatly from today (Golden et al., 1995; Baris et al., 2001; Youakim, 2006).

Even in modern times, firefighters are not completely protected from the chemicals present in fire atmospheres. According to the National Fire Protection Association, 10% of firefighter deaths in 2006 were due to asphyxiation (Fahy et al., 2007), and smoke inhalation accounted for 7% to 19% of all firefighter fire scene injuries from 1981 to 2006 (NFPA, 2008). In their study of 22 separate fires, Jankovic et al. (1991) noted that 70% of firefighters had removed their SCBA for at least some portion of the knockdown phase (when the main body of the fire is brought under control) and roughly one-third of firefighters only used their SCBA for 50% of the time during knockdown. In a one-year study of Montreal firefighters, it was concluded that firefighters used their SCBA for 50% of the time at structural fires, on average, and that SCBA was never worn during the overhaul stage (when firefighters are searching for and extinguishing hidden fire or hot embers) (Austin et al., 2001c).

Several cancer types have been associated with the occupation of firefighting. These include multiple myeloma, non-Hodgkin lymphoma, prostate cancer, testicular cancer, malignant melanoma, brain cancer, colorectal cancer, stomach cancer, leukemia, kidney cancer, bladder cancer, and cancer of the pharynx (Baris et al., 2001; Golden et al., 1995; LeMasters et al., 2006; Guidotti, 1995, 2007; Youakim, 2006). The patterns of increased cancer risk are not consistent across studies and as a result, there is some disagreement among researchers regarding which specific cancers are deemed to be plausibly related to firefighter exposures. A recent meta-analysis of published studies concluded that the likelihood of increased cancer risk due to firefighting was "probable" for multiple myeloma, non-Hodgkin lymphoma, and prostate cancer,

and “possible” for testicular cancer, skin cancer, malignant melanoma, brain cancer, colorectal cancer, stomach cancer, leukemia, and cancer of the buccal cavity and pharynx (LeMasters et al., 2006).

The International Agency for Research on Cancer (IARC) is part of the World Health Organization (WHO) and is one of the leading international bodies that examines the causes of cancer. In October 2007, scientists from 10 countries met at IARC to assess the carcinogenicity of thew occupation of firefighting. The details of their assessment will be provided in an upcoming IARC monograph (Volume 98), which as of June 2008 had not yet been published. On the basis of “limited evidence of carcinogenicity in humans”, IARC has classified occupational exposure as a firefighter as “possibly carcinogenic to humans” (Straif et al., 2007).

Assuming that exposures experienced while firefighting is a potential cause of cancer, a key consideration is the amount of exposure that is required before cancer risk is increased. The issue of amount of exposure was recently addressed in a review of published firefighter studies (Youakim, 2006). In general, increased cancer risks, if present at all, are typically only evident after 10 years (and in some cases after more than 20 to 30 years) of employment as a firefighter.

Very few studies have actually assessed the number of fires that firefighters respond to, relying on duration of employment as a surrogate exposure measure. A recent study of Montreal firefighters is one of the few to actually attempt to measure time spent at fires by firefighters, and the firefighters in this study responded to an average of 37 fires per year (Austin et al., 2001c). As a rough estimate, firefighters would likely respond to several hundred fires over the course of a 10-year period.

In summary, the available scientific literature indicates that firefighters may be at increased risk for certain cancers. A certain “dose” of firefighter exposures are required before that potentially increased cancer risk becomes apparent, however, and current knowledge suggests that this “dose” is in the neighbourhood of several hundred fires.

3.3.1 Summary

In theory, any degree of exposure to a chemical carcinogen will be associated with an increased risk of cancer as compared to zero exposure to a chemical carcinogen. For example, a person who has smoked one pack of cigarettes is theoretically at greater risk of developing lung cancer than the person who has never smoked. In practice, however, the increased risk from smoking one cigarette is extremely small and of no practical significance. For an individual who smokes one pack a day for many years however, the risk is considerably greater: approximately 90% of lung cancer cases are due to prolonged cigarette smoking (Hecht et al., 2007).

It is reasonable to conclude that the HMCS CHICOUTIMI fire atmosphere contained chemical carcinogens, and that the crew were exposed to these carcinogens. It is therefore also reasonable to conclude that HMCS CHICOUTIMI crewmembers are at greater theoretical risk of developing cancer than if they had never been exposed to the smoke from a submarine fire.

However, a certain dose of exposure to a chemical carcinogen is required before the theoretically increased cancer risk is of any practical significance. We are all exposed to very low levels of chemical carcinogens (many of them occurring in nature) through the food we eat, water we drink, and air we breathe. These low exposures are considered to be non-harmful, since the theoretical risk of developing cancer from such exposures is so low that it is essentially non-existent.

Although the exact risk related to HMCS CHICOUTIMI crew exposures cannot be quantified with certainty, it is possible to qualify this risk by comparing the experience of the crew with studies of individuals who have had similar exposures, such as firefighters. These studies indicate that a certain "dose" of firefighter exposures are required before the theoretical risk of developing cancer becomes clinically significant.

Although the exposures experienced by the HMCS CHICOUTIMI crew may not be directly comparable to the exposures experienced by a firefighter crew responding to a single individual fire, it is reasonable to conclude that HMCS CHICOUTIMI crew exposures were substantially less than what a firefighter may accumulate in responding to hundreds of fires over a career. Subsequently, the cancer risk related to exposures from the HMCS CHICOUTIMI fire would also be much smaller than the potential risk associated with exposures to several hundred fires. The development of cancer in crewmembers, as a result of exposures from a single fire, is therefore highly unlikely.

4.0 Exposures to Soot and Dust Left Over From the Fire

As described in Section 1.5, the HMCS CHICOUTIMI fire incident can be divided into two main categories. The preceding sections addressed the potential exposures and possible long-term health effects related to the fire atmosphere that was produced when the fire was actively burning and smoldering. This fire atmosphere would have been present until "cleared" through ventilation. Even though the fire atmosphere was cleared within hours after the fire, exposure to the leftover aerosols (soot, dust) that were produced by the fire could have occurred during the 5-day period from the time of the fire until docking in Faslane, Scotland.

The following discussion provides a detailed screening level risk assessment of the possible long-term health effects related to soot and dust exposure on board HMCS CHICOUTIMI during the 5-day transit back to Faslane, Scotland. In order to simplify the discussion that follows, the terms "dust" and "settled dust" are used to describe the leftover soot, dust, and debris that was sampled on board HMCS CHICOUTIMI.

4.1 Background

On 13th October 2004, Capt(N) McFadden (head of the HMCS CHICOUTIMI mission in the United Kingdom) and Capt(N) Orf Thamer (MARLANT N3) verbally requested to DCOS FHP (now DFHP) that a health hazard assessment be conducted on board HMCS CHICOUTIMI. This assessment was intended to provide health protection guidance for Canadian Forces (CF) Personnel required to board the damaged submarine (e.g. Duty watch, investigators). D FHP

staff developed a sampling strategy, and a PMed Tech, WO Newman, was deployed to Faslane, Scotland from 15 Oct to 27 Oct 2004 to conduct the sampling. Details of the sampling activities performed on board HMCS CHICOUTIMI can be found in a previous report (D FHP, 2005). An extensive series of data points were produced from the survey. Air, wipe, settled dust, and filter samples were taken with the intent of using them to conduct a risk assessment of potential adverse health effects.

4.2 Settled Dust Samples

On board air, wipe, settled dust, and air filter samples were obtained during the sampling period of 19 Oct to 21 Oct 2004, roughly two weeks after the fire. Only the settled dust samples were used for the present risk assessment. Annex F describes the air, wipe, and filter samples and discuss the reasons why these were not be used in the current risk assessment.

Settled dust samples were analysed for one or more of the following analytes: volatile organic compounds (VOCs) and semi-volatile organic compounds (SVOCs), polycyclic aromatic hydrocarbons (PAHs), dioxins and furans, metals, and asbestos.

Settled dust samples were obtained from three different locations on board the submarine (Commanding Officer's Cabin, Firing Control Area, and the Electrical Space) and these were incorporated in the present exposure assessment. For the purposes of this risk assessment, these three settled dust samples were regarded as being soil samples; the approaches, assumptions and considerations used in the risk assessment are those that would be applied in the evaluation of potentially contaminated soil samples.

4.3 Screening and Identification of Contaminants of Concern

Contaminants of (potential) concern (COC) were identified by comparing measured concentrations in settled dust to published Canadian (CCME, 1996) or American human health-based soil quality guidelines (see Annex G) since no settled dust guidelines exist. When available, the Canadian industrial Canadian Council of Ministers of the Environment (CCME) soil guidelines for the protection of human health from inadvertent soil ingestion were used to screen COCs in the settled dust samples as they are the most relevant for the current assessment.

The industrial CCME soil guideline for the protection of human health assumes that adults are exposed to the soil for their working life (40 hours a week for 48 weeks per year). It is recognized that settled dust is a different medium then soil and in this regard, using soil guidelines as a screening tool is not ideal. However, soil quality guidelines for protection of human health are established very conservatively, generally being derived such that exposure to contaminants in the soil at the guideline concentration will deliver only 20% ($1/5^{th}$) of the acceptable or tolerable daily intake of the contaminant (CCME, 1996). Therefore, as an initial tool to identify potential COCs, these guidelines were considered a reasonable and conservative screening method with the understanding that contamination in the settled dust samples in excess of the CCME guidelines does not necessarily constitute a health risk.

Among the close to 200 compounds measured in the three settled dust samples collected in HMCS CHICOUTIMI, most had concentrations that were either below their detection limit and/or human health-based soil criteria. The only exceptions were aluminum, copper, antimony, and dioxins and furans. Risk characterization related to these substances is discussed in more detail in Section 4.4.5.

Eight VOCs/SVOCs compounds merit discussion since even though they were undetected in the floor dust samples, their analytical detection limits was slightly higher than their health-based soil guidelines (Annex G). The probability of four out of eight compounds being on board the HMCS CHICOUTIMI is very low since their utility profiles are not compatible with the HMCS CHICOUTIMI, i.e. explosives residues (2,4-DNT and 2,6-DNT) and compounds used in the manufacturing of pesticides (2,4,5-Trichlorophenol and Bis(2-chloroethyl)ether). The assumption was made that since these materials are volatile, hypothetical low ppm levels of VOCs in settled dust should not be used to quantify potential exposure.

A detailed analytical characterization of PAHs in floor dust from HMCS CHICOUTIMI revealed that the PAHs measured in floor dust of the HMCS CHICOUTIMI originated largely from the fire (i.e. were of pyrogenic origin) as opposed to being present on board prior to the fire (Wang, 2004; Wang 2005). However, a small proportion of the detected PAHs came from oil and heavy diesel fuel (i.e. were of petrogenic origin) and were therefore not attributable to the fire (Wang, 2004; Wang 2005). Regardless, the PAHs in the floor dust were present only in very low concentrations of ppb, well below any benchmarks for PAH contamination in soils (Annex G).

During firefighting efforts, a dry chemical fire extinguisher was accidentally discharged in the Electronic Warfare Office. A sample was obtained of the residual dry chemical powder and analyzed for PAHs, dioxins and furans, and metals. The main parameters detected were calcium and sodium (likely as a result of assumed main ingredients of the dry chemical powder: calcium carbonate and sodium bicarbonate). The detected levels of calcium and sodium (140,000 ppm and 160,000 ppm, respectively) would not have represented a health concern. All other measured parameters results (metals, PAHs, dioxins and furans) were much lower than in the samples used for the risk assessment. It was noted that the dry chemical extinguisher powder sample had a slightly basic pH (alkaline). It is likely that the pH was more alkaline shortly after discharge of the extinguisher (roughly 2 weeks prior to when the sample was obtained), and this alkalinity may have caused irritation of mucous membranes (throat, nose, eyes). This irritation would have been only temporary and of no long-term health significance.

In the days following the fire, DMSS 2-4 staff confirmed that while there was asbestos on board Victoria-class submarines, such as the HMCS CHICOUTIMI, it was present in very modest amounts and mostly associated with machinery and gaskets, especially in the Motor Room and Engine Room (DMSS 2-4, 2004). As these areas were not involved in the fire, asbestos was not presumed to be a hazard on board HMCS CHICOUTIMI after the fire. Air sampling conducted at multiple locations on board HMCS CHICOUTIMI two weeks after the fire did not detect airborne asbestos in any form. As well, a sample of settled dust from the Commanding Officer's Cabin also did not detect any asbestos. Subsequently, it is concluded that asbestos was not a hazard on board HMCS CHICOUTIMI as a result of the fire.

In summary, based on the above considerations, four compounds - aluminum, copper, antimony, and dioxins and furans - were identified as contaminants of potential concern (COC).

4.4 Assessment of Potential Human Health Risks

4.4.1 Methodology and Organization

By necessity, due to the fact that individual exposures were directly measured during the time period from 5 Oct to 10 Oct 2004, this risk assessment employs a variety of assumptions concerning the frequency, duration and intensity of potential exposures. These assumptions have been selected such that potential exposures and risks have not been under-estimated. If negligible risks are indicated by these conservative assumptions, then actual exposure patterns would also likely present negligible risks.

The risk assessment methodology employed herein is consistent with the approach and framework provided by the Contaminated Sites Program of Health Canada (HC, 2004a), and the United States Environmental Protection Agency (US EPA, 1989). In the context of the estimation of health risks associated with the presence of environmental contamination, this methodology involves the following steps:

- hazard identification;
- exposure assessment;
- toxicity assessment;
- risk characterization.

The following sections are organized in parallel with the above methodology.

4.4.1.1 Non-Carcinogenic Contaminants

Risk assessment methods differ for carcinogens versus non-carcinogens. For non-carcinogens, it is anticipated that a threshold dose exists below which detrimental effects will not occur in the vast majority of the population (HC, 2004a). The tolerable daily intake (TDI) or reference dose (RfD) is a conservative estimate of that threshold (HC, 2004a).

Since a threshold dose is believed to exist for non-carcinogenic agents, it is recommended that total exposure (from the source in question combined with background exposure that arises through day-to-day activities unrelated to the settled dust) be quantified for risk characterization (HC, 2004a). Most inorganic elements are ubiquitous in the environment. As a result, exposure of CF members to these substances involves background sources, such as food, and not just the settled dust that they may have come into contact with. By considering background exposures in addition to exposure from the settled dust within the submarine, it is possible to determine if total exposure from all sources exceeds the acceptable or tolerable daily intake.

4.4.1.2 Carcinogenic Contaminants

Carcinogenic risk assessment is not wholly consistent with risk assessment for non-carcinogens. Continuous chronic exposure to most carcinogens is assumed to present some risk of cancer at any level of exposure (HC, 2004a). Despite this generalization, some carcinogens such as dioxins and furans discussed in the risk assessment presented herein, have been shown to be a threshold carcinogen (WHO, 1998; Popp et al., 2006). Based on available evidence from human, animal and mechanistic studies, most regulatory agencies consider dioxin a threshold carcinogen; i.e. it is believed there is a level of exposure below which cancer risk is not increased or is only negligibly increased above background incidence (WHO, 1998).

4.4.1.3 Body Burden Assessment

In the case of chemicals that resist metabolic elimination and accumulate, such as dioxins and furans, concerns related to chronic effects can occur following an acute exposure. This concern results when a compound has low acute toxicity but is known to persist for long periods of time within the body. Following an acute exposure to such chemicals, the absorbed dose may be stored within the body, and then only slowly released into the blood stream. The consequence of this response to an acute exposure event is a low-level release that effectively acts like a long-term dose.

The exposure to the on board fire and the subsequent five-day voyage back to Faslane could have resulted in an elevated dioxins and furans body burden. This elevated body burden, if present, would have peaked within a short time of exposure and then declined steadily thereafter. Dioxins and furans body burden was estimated for crewmembers and compared with dioxins and furans body burden indicative of adverse health effects. The body burden approach is an accepted and well-documented approach that has been utilized for the evaluation of chronic health risks following acute duration exposures to dioxins and furans (WHO, 1998; US EPA, 2003).

4.4.1.4 Consideration of Chemical Mixtures

Concurrent exposure to more than one chemical can cause interactions among toxicological effects; this may result in a combined toxicity, which is equal to the sum of toxicities of the individual chemicals (additivity or independence), greater than the sum (synergism or potentiation) or less than the sum (antagonism). In general, toxicological interactions depend on the chemicals present, the levels of exposure to each, their mode of action and their concentrations. The likelihood of a biologically significant interaction occurring is a function of at least the physical, chemical and biological properties of the chemicals involved, their modes of action, and their concentrations.

Most non-additive interactions can only be demonstrated at relatively high exposures, where clear adverse effects are observed. Such interactions have not been observed or quantified at the relatively low rates of exposure typical of those associated with most environmental or occupational situations (NAS, 1983; Krewski et al., 1992), and are therefore not typically considered in risk assessments. Additivity is generally recognized as the most plausible type of

interaction that may occur in situations of chemical exposure in the ambient environment. However, it requires that the chemicals act through the same or similar mechanisms of action and/or affect the same target tissue(s). In risk assessment where the COC act *via* different mechanisms of toxic action, and affect different target tissues, it is typically assumed that no potential toxicological interactions warrant consideration, and the estimated exposures and risks for the COCs are considered separately. Because aluminum, copper, antimony, and dioxins and furans act via unique mechanisms of action and/or do not affect the same target tissue, these individual substances were evaluated separately.

4.4.1.5 Risk Assessment for Short-Term Exposure

From a risk assessment perspective, the exposure period for the crew was a relatively short duration (5 days). Estimates for cancer and non-cancer risk from short-term exposure to chemicals generally rely on chemical toxicity values derived from chronic, lifetime exposure studies. Exposures of limited duration are associated with proportional reduction in risk as compared to long duration exposures. Although Health Canada currently has no *formal* guidance on the conduct of risk assessment for short-term exposures, their interim advice regarding averaging of short-term exposure time periods has been utilized in this risk assessment (HC, 2008).

4.4.2 Hazard Identification

Hazard identification involves the characterization of the nature of the contaminants present at the site and their concentrations in the relevant environmental media (settled dust). For the four COCs being investigated, the highest measured concentrations out of the three samples are presented in Annex G.

The three settled dust samples were collected at different sampling locations within HMCS CHICOUTIMI (Commanding Officer's Cabin, Firing Control Area, and the Electrical Space). Due to the limited nature of the sampling at individual sites (one composite sample per site), the data are insufficient to permit a valid, separate analysis of potential risks posed at each different location. Rather, the samples collected were considered reflective of the average settled dust contamination conditions that were likely encountered by crewmembers.

4.4.3 Exposure Assessment

The purpose of the exposure assessment is to estimate the type and magnitude of human exposure to the contaminants of concern. The exposure assessment typically consists of the following steps: characterization of the exposure setting, identification of the critical receptor, determination of routes of exposure, and estimation of the frequency, duration and amount of chemical exposure. These steps are discussed below.

4.4.3.1 Exposure Setting

The exposure setting considered within this risk assessment is CF members living and working within HMCS Chicoutimi with limited or no access to washing facilities.

4.4.3.2 Critical Receptors

The receptors of concern are the crew of HMCS CHICOUTIMI. Since military-specific exposure factors (such as skin surface area, inhalation rate, etc.) are unavailable, data relating to the Canadian general male adult population were employed for the purpose of quantifying receptor characteristics, which was required to estimate potential exposures (Richardson, 1997).

4.4.3.3 Exposure Duration

The duration of exposure to crewmembers was five days or from the time of the on board fire on 5 Oct 2004 to the time HMCS Chicoutimi was docked in Faslane, Scotland, on 10 Oct 2004. During these five days, 24 hours of exposure per day was assumed.

4.4.3.4 Identification of Exposure Pathways

Crewmembers may have been exposed to settled dust and soot by three pathways: inhalation, skin absorption and ingestion. Living and working on board HMCS CHICOUTIMI after the fire would have disturbed the settled dust and re-suspended it into the air, where it could have been inhaled. Interaction with the soot-covered areas would have resulted in transfer of the settled soot onto exposed skin surfaces. Ingestion of settled soot and dust could have occurred through a number of ways: direct transfer of soot from soot-covered hands to the mouth; inadvertent swallowing of soot or dust particles that are inhaled, deposited in the upper respiratory tract, and then cleared back up into the mouth; mouthing of objects or consumption of food that has been touched by soot-covered hands; and consumption of food or beverages that suspended dust may have settled on to prior to consumption.

The contaminants of concern discussed here are not volatile, have relatively low solubility, and tend to adsorb (attach) to particulate matter. As a result, these COCs will generally remain adsorbed to soil particulate matter.

4.4.3.5 Exposure Pathway Analysis

A pathway consists of a source, a transport medium, a point of human exposure and an intake route at the point of contact. The purpose of the exposure assessment is to develop a relationship between the contaminant source concentration and the projected intake of the contaminant. This relationship is then used in conjunction with the applicable toxicological information to estimate the health risk.

Annex H presents the general equations used to calculate doses of COCs *via* each pathway. Pathway-specific considerations are discussed below. Two sets of input parameters were required to conduct the quantitative exposure assessment: receptor characteristics and contaminant-specific factors. The receptor characteristics govern the degree of exposure of an individual to the contaminant and are used in determining pathway-specific intakes based on predicted point-of-exposure concentrations. These characteristics include parameters such as soil ingestion and dermal contact rates, as well as the duration of exposure. These characteristics

are discussed further in the following sections and summarized in Table 5. Table 5 also contains chemical-specific factors required for the assessment of exposures to crewmembers, such as dermal and inhalation absorption rates.

Table 5: Summary of Assumptions Defining Critical Receptors

PARAMETER	Default receptor values	Crewmember receptor values
Body weight (kg)	70	70
Inhalation rate (moderate) (m^3/hr)	0.64	1.6
Dust ingestion rate (mg/day)	20	1000
Concentration of respirable particulate in air (mg/m^3)	0.00076	1
SURFACE AREA OF SKIN (cm^2)[c]		
Hands	880	880
Area other than hands (head, upper and lower arms and legs)	9220	9220
Dust loading to hands (mg/cm^2-event)	0.1	1
Dust loading to body surfaces other than hands (mg/cm^2-event)	0.01	0.1
Skin contact event with dust per day (events/day)	1	1
EXPOSURE DURATION		
Hours/day		24
Days/week		5
ABSORPTION FACTORS		
Absorption factor for inhalation exposure (unitless)	1	1
Absorption factor for gastrointestinal tract (unitless)	1	1
Absorption factor for contact of skin with dust (unitless)	0.01	0.01

i. Methodology and Assumptions for Direct Ingestion of Settled Dust

Unintentional ingestion of dust occurs in all age groups of the population (Sedman et al., 1994). This results from the mouthing of unwashed hands and other surfaces, from transfer from unwashed hands to food, and from ingestion of inhaled dust particles deposited in the mouth and upper respiratory tract which are then transferred to the mouth and swallowed. Quantitative data respecting the inadvertent ingestion of soil or dust are extremely uncertain (US EPA, 1997). No data on soot or dust ingestion rates relevant to post-fire exposure are available. Therefore, other data must be used as a basis for assumed dust ingestion rates.

It is generally assumed that a typical adult ingests soil at a rate of between 20 mg/day (default value for Health Canada; HC, 2004a) and 50 mg/day (default value for the US EPA; US EPA, 1997) while 100 mg/day is assumed for construction workers in dusty environment (HC, 2004a). These inadvertent ingestion rates do not likely reflect the level of incidental ingestion that would have occurred on board HMCS Chicoutimi. The on board fire would have created much more dust than the default environmental assumptions. In addition to living in a dustier environment, it was assumed that the crewmembers had limited access to washing facilities. The levels of

suspended dust and the intensity of direct contact with the soil would likely have resulted in soil ingestion exceeding 50 mg/day.

The US EPA recommends using 400 mg/day for assessing potential risks posed to children involved in outdoor activities based on upper percentile measurements reported in Calabrese et al. (1989). Hawley (1985) suggested a similar value (480 mg/day) for adults engaged in outdoor activities. However, Hawley's estimate was derived from assumptions about soil/dust levels on hands and mouthing behavior with no supporting measurements. The estimate of 480 mg/day was rounded to one significant figure and doubled to account for the fact that HMCS crewmembers did not benefit from washing facilities. For this assessment, it was therefore assumed that the dust ingestion rate for crewmembers of HMCS Chicoutimi was 1 g/day. This assumption is clearly uncertain but considered reasonable and conservative.

ii. Methodology and Assumptions for Inhalation of Settled Dust

The fire itself plus working and walking through areas that sustained smoke damage would have resulted in the presence of airborne soot and dust particles. No data on suspended dust and soot air concentrations are available specific to post-fire activities within HMCS CHICOUTIMI during the 5-day transit back to Faslane, Scotland.

Lacking data specific to HMCS CHICOUTIMI, assumptions concerning air-borne dust levels were drawn from outdoor data relating to vehicular travel on unpaved roads and agricultural practices as well as from indoor cooking fires and from environmental data taken shortly after the World Trade Center attacks of September 11, 2001.

Research has demonstrated that farming activities generate considerable levels of suspended particulate matter. Much of this suspended particulate matter is in the respirable size range (aerodynamic diameter < 10 μm) (Nieuwenhuijsen et al., 1998). Likewise, dust caused by vehicle traffic on unpaved roads can contribute significantly to levels of suspended particulate matter (Claiborn et al., 1995; Evans et al., 1981). Similarly, particulate matter generated during indoor cooking fires is generally within the respirable fraction.

The average dust level created by vehicle traffic on unpaved roads is about 0.25 mg/m^3 (down-wind side of road; Claiborn et al., 1995). Agricultural activities such as land planning generates on average 10 mg/m^3 of airborne dust (Clausnitzer et al., 1996 and 1997; Nieuwenhuijsen et al., 1998). Albalak et al. (2001) reported average respirable particulate matter generated from indoor open cooking fires of 1 mg/m^3 in poorly ventilated houses. Several mg/m^3 were assumed to have been experienced following the World Trade Center attacks (US EPA, 2002). Although no direct measurements of particulate matter concentrations are available for nearby lower Manhattan areas during the collapse of the World Trade Center, estimations were made based on the optically dense cloud seen from the airborne images (US EPA, 2002). Under such conditions, sunlight does not reach the surface, and visibilities are greatly restricted. Conditions such as these have been encountered in dust storms and in the London smog episodes of 1952 and 1962 (Elsom, 1992 cited in US EPA, 2002). During such conditions, particulate matter concentrations could have been several mg/m^3 (US EPA, 2002).

To be conservative, it was assumed in this assessment that submariners experienced average airborne dust levels of 1 mg/m^3 (similar to particulate matter levels generated during indoor open cooking fires and within the range of the World Trade Center estimates) throughout the 5 days spent within HMCS CHICOUTIMI following the fire. This is a conservative estimate of possible exposure and it is highly unlikely that crewmembers would have inhaled 1 mg/m^3 of particulate matter for the entire 5 days of exposure, especially with continued use of the starboard engine for active ventilation during this time period. Levels within the mg/m^3 range would translate to reduced visibility.

The default inhalation rate for adult men and construction workers is 15.3 m^3/day or 0.64 m^3/hr (HC, 2004a). For purposes of this risk assessment, it was assumed that the crewmembers would have had an elevated inhalation rate of 1.6 m^3/hour, 24 hours a day for the five-day exposure period. This inhalation rate corresponds to the US EPA recommended inhalation rate for adult males conducting moderate physical activities (US EPA, 1997).

iii. Methodology and Assumptions for Skin Absorption of Settled Dust

Contaminants may be absorbed into the body from contaminants deposited on the skin. The amount absorbed is dependent on the amount of exposed skin surface, the amount of contaminants deposited on that exposed skin, and the rate of absorption (penetration) of contaminant through the skin.

Direct data relating to rates of dust loading to skin of HMCS CHICOUTIMI crewmembers are not available, nor are data available for activities following an indoor fire. Therefore, quantitative assumptions were derived from alternate sources.

Research on soil loading onto skin from both field and controlled trials has been published by Kissel et al. (1996 and 1998). Loadings are consistently greatest on the hands, with lower loadings to face, arms and legs. Loadings are generally greater for activities involving direct contact with soil (gardening, farming activities, pipe laying, for example) and are greater to exposed skin than to skin covered by clothing. Duration of activity has little or no significant influence on total loading to the hands. Loadings of moist soil are about an order of magnitude greater than loadings of dry soil.

HMSC CHICOUTIMI crewmembers are considered likely to have relatively high rates of dust loading to skin, similar to persons engaged in activities such as farming, gardening and pipe laying. Based on the studies of Kissel et al. (1996, 1998), loadings of dust to hands of crewmembers were assumed to be 1.0 mg/cm^2 of exposed skin surface area per exposure event, with deposition to other exposed body surfaces (arms, legs, face and neck) of 0.1 mg/cm^2 per event. These values are the default values recommended by HC for construction workers (HC, 2004a).

Furthermore, it was assumed that the hands, arms, legs, and head were always exposed for potential dust deposition during the 5-day exposure of concern. Health Canada's default surface areas of hands, arms, legs and head for adult men were used for the exposure estimation (HC, 2004a).

4.4.3.6 Background Exposures to Contaminants of Concern

Within this risk assessment, consideration was given to background exposure to COCs, unrelated to exposures within HMCS CHICOUTIMI. It is recognized that people are exposed to a myriad of metals and environmental contaminants through their day-to-day activities, unrelated to any specific site of contamination. Trace amounts of metals and persistent organics can be found in airborne dust and particulate matter, house dust, backyard soils, and drinking water. Metals are naturally occurring elements found in all media in which people come in contact (air, water, food, soil). Chlorinated organics, due to their resistance to degradation, are now ubiquitous and found at low levels in all environmental media. Metals and persistent organics are found in most foods and food consumption is generally the single largest source of background exposure.

Because people are exposed to contaminants from sources other than soils, risk assessments for non-carcinogenic endpoints should take this background exposure into account (HC, 2004a). To ensure human safety, total exposure from the contaminated site and background exposure, combined, should not exceed the reference (safe) dose prescribed for the particular contaminant (HC, 2004a).

Background exposures to the COCs cannot be directly quantified for HMCS CHICOUTIMI crewmembers. However, since crewmembers dietary habits and foods would be similar to those for Canadians in general, it is reasonable to assume that background exposures would be similar to those experienced by average Canadians. For purposes of this assessment, it was assumed that background intakes of the COCs were equivalent to those experienced in Canada. Average or typical Canadian adult daily intakes for the COCs have been reported as follows:

Aluminum = 0.12 mg/kg/day (HC, 1998);
Copper = 0.03 mg/kg/day (WHO, 1996);
Antimony = 0.0001 mg/kg/day (HC, 1997);
Dioxins and Furans = 0.6 pg/kg/day or 0.0000000006 mg/kg/day (HC, 2005).

4.4.4 Toxicity Assessment

Regulatory agencies routinely evaluate quantitative relationships between the dose of each contaminant and the likelihood of adverse health effects in order to prescribe allowable levels of chemical exposure. These allowable levels are termed reference doses (RfD), tolerable daily intakes (TDI), Toxicity Reference Values (TRV) or acceptable daily intakes (ADI). These reference doses represent the best estimate of the human threshold for health effects, and provide a benchmark exposure level against which estimated exposures arising from the site in question can be compared. Where estimated exposures do not exceed those benchmarks, potential risks are considered negligible, acceptable or tolerable.

The primary sources of reference doses used to conduct this assessment were Health Canada (HC, 2004b) and the US EPA's Integrated Risk Information System (IRIS) database.

4.4.5 Risk Characterization

4.4.5.1 Estimated Exposures and Hazard Quotients for Chemicals of Concern

Exposure estimates and subsequent hazard quotients for HMCS CHICOUTIMI crewmembers are presented in Table 6. The hazard quotient (HQ) represents the ratio of the estimated exposure divided by the reference or tolerable dose. Hazard quotients which are less than or equal to 1.0, when rounded to one significant digit, suggest that the estimated exposure is tolerable, acceptable, or of minimal risk. This is particularly true when background exposures, unrelated to the exposure event under investigation, are included in the calculation, as was done herein.

Table 6: Exposure Estimates and Hazard Quotients for Contaminants of Potential Concern

	Aluminum	Copper	Antimony	Dioxins and Furans TEQ*
A. Maximum Concentration (mg/kg)**	41000	54000	2300	915 pgTEQ/g
B. Inhalation Exposure (mg/kg bw/day)	0.016	0.021	0.00094	0.0069*
C. Ingestion Exposure (mg/kg bw/day)	0.42	0.55	0.023	0.18*
D. Skin Absorption (mg/kg bw/day)	0.0075	0.0099	0.00042	0.0032*
E. Total On board Dust Exposure (B + C + D) (mg/kg bw/day)	0.44	0.58	0.024	0.19*
F. Background Exposure (mg/kg bw/day)	0.12	0.03	0.0001	0.6*
G. Total Exposure (E + F) (mg/kg bw/day)	0.56	0.61	0.024	0.79*
H. Health Reference Value (TRV or TDI) (mg/kg bw/day)	1	0.14	0.0004	2.3*
Hazard Quotient (HQ) (G ÷ H)	**0.6**	**4**	**60**	**0.3**

* Note that units of measurement in the table for aluminum, copper and antimony are in mg/kg bw/day while pg/kg bw/day are used to describe dioxins and furans. The concept of TEQ is discussed in Section 4.4.5.2.
** From Annex G

Because of the inherent conservatism built in screening level risk assessment such as the current one, hazard quotients exceeding 1.0 do not necessarily imply that adverse health outcomes will occur. However, Hazard Quotients that exceed 1.0 warrant a more in depth look at the exposure estimation and risk characterization.

Of the four contaminants of concern, and considering the various scenarios and assumptions discussed herein, only antimony and copper appear to present any potential risk. The hazard quotients of aluminum, and dioxins/furans are equal to or less than 1.0 while estimated exposures to antimony and copper exceeded the published reference dose slightly.

The following sections describe the risk characterization for each of the four chemicals of concern separately.

4.4.5.2 Risk Characterization of Dioxins and Furans

Dioxins and furans are generic terms for polychlorinated dibenzo-para-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs), respectively. These are triple carbon ring structures with two or more attached chlorine atoms. In theory, 75 different PCDDs and 135 different PCDFs are possible, depending on the number and orientation of attached chlorine atoms. Only 17 of these possible structures (congeners) are considered to be toxic. The most toxic of these is 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD). To assess the hazard posed by mixtures of these 17 species, each is assigned a toxicity equivalency factor, based on its potency in relation to 2,3,7,8-TCDD. The factor for 2,3,7,8-TCDD is equal to 1, and all other PCDDs and PCDFs have factors that are some fraction of 1. The total amount of PCDDs and PCDFs in a mixture is expressed as a toxic equivalency (TEQ), which is the sum of the products of the concentration and toxic equivalency factor for each congener in the mixture. The toxicity of a mixture of dioxins and furans, expressed as an amount of TEQ, is comparable to the toxicity of the same amount of pure 2,3,7,8-TCDD. For simplicity, PCDD's and PCDFs have simply been referred to as dioxins and furans, respectively, in this report.

The International Agency for Research on Cancer (IARC) has classified 2,3,7,8-Tetrachlorodibenzo-para-dioxin as carcinogenic to humans (IARC, 1997). Dioxins are also associated with adverse dermatologic, immunologic, endocrine, cardiovascular, reproductive and neurological effects. The World Health Organization (WHO) established a tolerable daily dioxin intake equivalent to 1-4 pg TEQ /kg bw/day (WHO, 1998), which is similar to other international health assessments (Pohl et al., 2002). A picogram (pg) is equivalent to 1 trillionth of a gram: 1 picogram = 0.000000000001 grams.

Although the current official Health Canada TDI is still 10 pg/kg bw/day (HC, 2004b), it is suggested to opt for the more conservative and revised WHO/FAO Joint Expert Committee on Food Additives and Contaminants (JECFA) TDI of 70 pg/kg bw/month, or 2.3 pg/kg bw/day (WHO, 1999). As a comparison, a recent meta-analysis of occupationally exposed cohorts found a 1% increase in lifetime risk of cancer mortality for lifetime average daily TEQ intakes of 45 pg TEQ/kg bw/day, and no increase in total cancer at a lifetime dose of 6 pg TEQ/kg bw/day (Crump et al., 2003).

The highest value for total TEQ was obtained in a settled dust sample from the Electrical Space: 915 pg TEQ/g (Annex G).

The risk characterization of dioxins and furans was conducted in two ways; firstly, by comparing the estimated intake resulting from the on board fire to the established tolerable daily intake and evaluating the resulting hazard quotient, and secondly, by predicting a body burden of dioxins and furans that would have resulted from the on board fire exposure and then comparing this with a body burden indicative of adverse health effects.

i. Evaluation of the Hazard Quotient for Dioxins and Furans

Annex H summarizes the calculations used to assess the potential dose received by crewmembers from exposure to dioxins/furans (expressed as total TEQ). A summary of critical receptor characteristics used in the assessment is found in Table 5. It was deemed appropriate to average short-term exposure for dioxins and furans because of their long half-lives and the fact that the TRV is based on developmental effects and an averaging period of 30 days. Moreover, the JECFA's dioxins/furans TRV is based on development effects in the offspring of exposed females, the most sensitive endpoint in the most sensitive subpopulation. Since the male population is expected to be less sensitive to the effects of dioxins/furans, the JECFA's TRV is expected to be protective to the HMCS CHICOUTIMI crewmembers.

Table 6 contains the exposure pathway dose estimates based on conservative assumptions that likely overestimate exposure (see Annex H for the chemical-specific detailed exposure calculations).

The calculated dioxins/furans exposure from on board dust was 0.19 pg TEQ/kg bw/day. When combined with the assumed background exposure to dioxins and furans of 0.6 pg TEQ/kg bw/day (HC, 2005), the overall total dioxins/furans exposure was 0.8 pg TEQ/kg bw/day. This total is lower than the JECFA's TDI of 2.3 pg TEQ/kg bw/day. It should also be noted that the exposure to the on board submarine contributed only slightly to the background exposure. Considering that the conservatively estimated daily TEQ is below the range of tolerable daily intakes, the risk of adverse health effects for crewmembers from exposure to dioxins/furans in settled dust is considered to be negligible.

ii. Evaluation of Predicted Dioxins and Furans Body Burden

Dioxins, like many chemicals, accumulate in various body tissues through a variety of exposure routes (inhalation, ingestion, or skin absorption). A common approach for evaluating chemical loading and associated effects in humans is a body burden approach. The term body burden refers to the concentration of chemical in the body at a specific point in time. For chemicals like dioxin, that are persistent within the body, there is a potential for high short-term exposures to remain in the body and result in long-term health effects.

It is possible to relate doses at which effects occur to body burdens. JECFA's TRV of 2.3 pg TEQ/kg bw/day can be expressed on the basis of a body burden. Using a simple steady-state pharmacokinetic model provided by the US EPA Dioxin Reassessment Manual (Equation 1; US EPA, 2003), and the assumption that the TRV represents a lifetime average dose, the Health Canada TRV can be expressed as a body burden of 28 pg TEQ/g lipid. This value is referred to as the body burden TRV.

Equation 1

$$\text{Body burden TRV} = \frac{D \times A \times BW}{(\ln 2/t_{1/2}) \times V \times CF_1}$$

D = Daily dioxin TEQ intake (2.3 pg/kg bw/day; HC 2004b)
$t_{1/2}$ = Body half-life of dioxin (7.1 years; US EPA, 2003)
V = Volume of lipid in the body 17500 g (default assumption; US EPA, 2003)
A = Absorption factor (0.80; US EPA, 2003)
BW = Body weight of 70 kg (default assumption; HC, 2004a)
CF_1 = 0.00274 years/day

Similarly, the dioxin tissue concentration of crewmembers following the on board exposure during the 5-day voyage back to Falsane can be predicted from the estimated absorbed daily dose calculated in the above section (Equation 2; US EPA, 2003). Equation 2, as opposed to Equation 1, represents the body burden event (in this case, the 5-day exposure period) and assumes that none of the accumulated dioxin was eliminated during the five-day exposure.

Equation 2

$$BB_{event} = AD \times ND \times BW / V$$

Where:

BB_{event} = Body burden resulting from the on board exposure (pg TEQ/g lipid)
AD = Absorbed daily dose from the on board dust exposure (0.19 pg TEQ/kg bw/day; see Section 4.4.5.2.i)
ND = Number of days of exposure (5 days)
BW = Body weight of 70 kg (default assumption; HC, 2004a)
V = Volume of lipid in the body 17500 g (default assumption; US EPA, 2003)

The predicted dioxins and furans body burden of crewmembers due to the 5 days of dust exposure is 0.004 pg TEQ/g lipid. This body burden is very low compared to predicted Canadian background body burden level of 7 pg TEQ/g lipid (calculated using Equation 1 and the Health Canada estimated daily intake of dioxins and furans by the general Canadian population of 0.6 pg TEQ/kg bw/day) or to the body burden TRV of 28 pg TEQ/g lipid. It is therefore concluded that the fire exposure contributed minimally to the background body burden of crewmembers and no risk of adverse chronic health effects from this exposure would be expected.

4.4.5.3 Risk Characterization of Aluminum

Aluminum is the most abundant metal, comprising 8.1% of the earth's crust. It is found in a variety of minerals, such as feldspars and micas, which ultimately weather to clays. Aluminum is mined primarily as bauxite, a family of mixed Aluminum oxides and hydroxides. Aluminum

is found as a normal constituent of soil, plants and animal tissues (HC, 1998). Aluminum metal and its alloys are widely used in aircraft and shipbuilding. They are light and durable.

Salts of Aluminum are used by the pharmaceutical industry as ingredients in antacids and antidiarrhoeals. Aluminum (as aluminum chlorhydrate) is a common component of anti-perspirants. Aluminum is also used extensively as a food additive and as a component of food packaging materials. In addition, substantial amounts of aluminum salts (alum) are commonly added as flocculating agents during the treatment of drinking water (HC, 1998). A background exposure to aluminum was estimated for Canadians at 0.12 mg/kg bw/day (HC, 1998).

Aluminum in workplaces is generally only considered harmful when it exists in the form of a dust or fume. As a dust it can irritate the eyes and skin. As a fume produced from welding, it can cause metal fume fever. The clinical manifestations of metal fume fever are discussed in Section 3.1.

Long-term inhalation exposure to aluminum dust for many months or years can cause scarring of the lungs with cough and shortness of breath.

A TDI for aluminum has not been established by either Health Canada or the US EPA. However, the US Agency for Toxic Substances and Disease Registry (ATSDR) has derived an intermediate-duration oral minimal risk level (MRL) of 1 mg aluminum/kg/day for aluminum (ATSDR, 2006). This MRL is based on a No Observable Adverse Effect Level (NOAEL) of 26 mg aluminum/kg/day and a Lowest Observable Adverse Effect Level (LOAEL) of 130 mg aluminum/kg/day for neurodevelopmental effects in the offspring of female mice exposed to aluminum lactate in the diet on gestation day 1 through lactation day 21 followed by pup exposure until postnatal day 35 (Golub et al., 2001). The MRL was derived by dividing the NOAEL by an uncertainty factor of 100 (10 for animal to human extrapolation and 10 for human variability) and a modifying factor of 0.3 to account for the higher bioavailability of the aluminum lactate used in the principal study, as compared to the bioavailability of aluminum in the human diet and drinking water. MRLs derived for intermediate duration are considered to adequately protect the health of humans continuously exposed to the substance for duration of up to one year.

Table 6 contains the exposure pathway dose estimates based on conservative assumptions that likely overestimate exposure (see Annex H for the chemical-specific detailed exposure calculations).

The highest aluminum concentration measured in settled dust on HMCS CHICOUTIMI was 41000 mg/kg, from the Commanding Officer's Cabin (Annex G). The calculated aluminum exposure from on board dust was 0.44 mg/kg bw/day. When combined with the estimated background exposure of 0.12 mg/kg bw/day, the overall aluminum exposure was ~0.6 mg/kg bw/day. This is lower than the recommended TDI of 1 mg/kg bw/day, even though the exposure that occurred over 5-days is not averaged over a year. Moreover, the range of aluminum levels measured in settled dust samples was 2400-41000 mg/kg, which is less than the range of levels expected in uncontaminated soils (45000–100000 mg/kg; Pais et al., 1997). Therefore, the risk

of adverse health effects for crewmembers from exposure to aluminum in settled dust is considered to be negligible.

4.4.5.4 Risk Characterization of Copper

Copper occurs naturally in many minerals such as cuprite, malachite and azurite. It is obtained from its ores by smelting or extraction and floatation methods. Industrially it is an important material because of its durability, malleability and electrical and thermal conductivity. Copper and its alloys have many uses in the electrical and construction industries, as parts for machinery, water pipes and structural components.

Copper is an essential nutrient. The background exposure value for copper was assumed to be 0.03 mg/kg bw/day. This is the estimated average daily intake from the WHO range for the minimum daily nutritional requirement (0.02 to 0.08 mg/kg bw/day; WHO, 1996).

In workplace situations copper is hazardous as a dust or fume. As a dust it can irritate the eyes, nose or throat. It can also cause a skin allergy. Repeated exposures can cause a greenish colouration of the skin, teeth and hair. Copper fume from welding operations can cause flu like symptoms known as metal fume fever. The clinical manifestations of metal fume fever are discussed in Section 3.1.

Long-term exposure over many months or years can cause nasal ulcers and perforation of the nasal septum. Repeated exposure to high levels of copper dust or fume can damage the liver. Repeated ingestion of some copper salts may cause gastrointestinal irritation.

No oral reference dose for copper has been established. However, ATSDR has set a minimal risk level (MRL) of 0.01mg/kg bw/day based on acute gastrointestinal effects of women consuming 0.02 mg /kg bw/day (Pizarro et al., 1999). This seems unusual, because the minimal risk level is less than the range required for copper as an essential nutrient. The ambiguity is probably related to the particular copper salt in the Pizarro et al. (1999) investigation (copper sulphate). Copper sulphate is known to be innately more toxic than copper in many other forms, which is why it is employed as an active ingredient in several fungicides and algicides. For this reason the MRL was not used as a substitute for the Reference Dose in the risk characterization.

The value used for the risk characterization calculation is the TDI quoted by the Dutch Organization, RIVM, in which the TDI of 0.14 mg/kg bw/day was derived from the data of Sloof et al. (1989). The RIVM TDI is an estimate of the amount of a non-cancer causing chemical to which people can be exposed every day for their whole life without threatening their health.

Table 6 contains the exposure pathway dose estimates based on conservative assumptions that likely overestimate exposure (see Annex H for the chemical-specific detailed exposure calculations).

The highest concentration of copper in settled dust on HMCS CHICOUTIMI was recorded at 54000 mg/kg, from the Commanding Officer's Cabin (Annex G). The calculated copper exposure from on board dust was 0.58 mg/kg bw/day. When combined with the assumed

background exposure to copper of 0.03 mg/kg bw/day, the overall total copper exposure was ~0.6 mg/kg bw/day. This copper daily exposure is slightly higher then the copper TDI of 0.14 mg/kg bw/day, resulting in a Hazard Quotient that is greater than 1.0. However, it should be noted that the estimated copper exposure for crewmembers was not averaged out to account for the short exposure period while the RIVM TDI assumes a lifetime of exposure. Considering the short period of exposure for HMCS CHICOUTIMI crewmembers, the risk of adverse health effects for crewmembers from exposure to copper in settled dust is considered to be negligible.

4.4.5.5 Risk Characterization for Antimony

Antimony is a silvery white metal, but is rarely found as the pure element. It occurs naturally in the earth's crust in more than 100 minerals such as sulphides and oxides. There is some mining of Antimony in the US and Canada, but large amounts are imported or retrieved from scrap metal refining and as a by-product of lead smelting. Antimony is a brittle metal and is largely used in alloys with solder, sheet and pipe metal, bearings, castings, pewter and lead storage batteries. As a pure metal it is used in the production of semiconductors, infrared detectors and diodes. Non-metallic Antimony oxides are added to textiles and plastics as a fire retardant. It is also used in paints, enamels, ceramics, glass and fireworks.

Antimony can irritate the nose, throat, and lungs, and can cause wheezing and shortness of breath. In addition, short-term exposure can lead to headaches, nausea, vomiting, and abdominal pain. Long-term exposure over many months or years can cause ulcers in the nose and damage to the kidneys, heart and liver.

The oral reference dose for antimony is derived principally from studies with rats fed drinking water contaminated with antimony, which established a LOAEL of 0.35 mg/kg bw/day (Schroeder et al 1970). An uncertainty factor of 1000 was applied to this figure (10 for interspecies conversion, 10 for protection of sensitive individuals and 10 because a LOAEL rather than a NOAEL was used). The oral TDI was therefore established at 0.0004 mg/kg bw/day (IRIS, 1991).

Table 6 contains the exposure pathway dose estimates based on conservative assumptions that likely overestimate exposure (see Annex H for the chemical-specific detailed exposure calculations).

The highest concentration of antimony in settled dust on HMCS CHICOUTIMI was recorded at 2300 mg/kg (Annex G), from the Firing Control Area. The calculated antimony exposure from on board dust was 0.024 mg/kg bw/day. When combined with the assumed background exposure to antimony of 0.0001 mg/kg bw/day, the overall antimony exposure was 0.024 mg/kg bw/day. This value is 60 times greater than the recommended TDI for antimony (Table 6).

Despite this elevated hazard quotient, the following considerations indicate that potential health effects related to exposure to antimony in settled dust would be unlikely. Firstly, the exposure period for crewmembers is very short (five days *versus* a lifetime), whereas the TDI assumes a lifetime of exposure. Secondly, the estimate assumes that 100% of the antimony bound to dust would be absorbed by the gut, but this is surely not the case. ATSDR (1992) cites a 10%

gastrointestinal absorption in humans for antimony tartrate and 1% for all other forms of antimony as reference values. Lastly, the TDI for antimony was derived by assuming a considerable uncertainty factor (1000 fold) including a 10-fold uncertainty factor for protection of sensitive individuals, which does not necessarily apply to HMCS CHICOUTIMI crewmembers.

Therefore, with the above considerations in mind, it is concluded that exposure to antimony in settled dust on board HMCS CHICOUTIMI would not have resulted in adverse health effects after a 5-day exposure period.

4.5 Uncertainty

Risk assessments are always associated with uncertainty. This current risk assessment is no exception, with uncertainty arising from dust sampling, use of soil quality guideline to assess dust, and from the method used to characterize risk from short-term exposure. While efforts were made to reduce the level of uncertainty to the greatest extent possible, it is recognized that not all uncertainty can be realistically addressed. Health risk therefore needs to be interpreted within this context.

Generating a list of the various sources of uncertainties that affect a human health risk assessment is an important step in understanding the results of the assessment.

4.5.1 Uncertainties due to Timing of Dust Sampling

On board dust sampling took place more than one week after the HMCS CHICOUTIMI arrived in Faslane, Scotland. Subsequently, the dust samples may not be exactly representative of the conditions that existed on board during the 5-day post-fire period. Potential uncertainties related to this are discussed below.

The contaminants of (potential) concern (COCs) examined in detail in this risk assessment (aluminum, copper, antimony, dioxins and furans) are not volatile, have relatively low solubility, and tend to stay attached to particulate matter, such as settled dust. Therefore, although there was a delay between the time of exposure and the sampling of the settled dust, this delay alone was unlikely to have significantly affected the concentrations of these contaminants in the samples.

During the 5-day period after the fire, crewmembers had performed cleaning activities in various spaces, notably the accommodations space, messes, galley and other principal living areas, including passageways. This was done in an effort to make the areas more habitable since it was necessary for most of the crew to remain and live on-board (D FHP, 2005). Dust samples for this risk assessment were obtained from areas that were heavily smoke-damaged and minimally affected by cleaning procedures (e.g. Commanding Officer's Cabin, Electrical Space). The possible effects of cleaning efforts on the samples cannot be determined with certainty. However, this source of uncertainty is likely not relevant to the present risk assessment, since the risk assessment is based on *concentrations* of chemical contaminants in the settled dust, not the *amounts* of dust. Although cleaning efforts may have affected the *amounts* of dust available for

sampling, it is unlikely that cleaning efforts would have greatly affected the chemical *composition* of the dust itself.

Prior to dust sampling in the Commanding Officer's Cabin and Electrical Space, evidence collection activities occurred, such as the use of a hacksaw to cut out large gauge electrical cables (D FHP, 2005). These activities may have resulted in the production of non fire-related dust, such as metal dust from the cutting of electrical cables. As a result, the obtained samples may not have been exactly representative of the soot and dust found in other areas of the submarine.

The influence of the above uncertainties on the present risk assessment is estimated to be small, and likely accounted for by the use of conservative exposure assessment assumptions, as discussed in Section 4.4.3.5 and 4.5.3.

4.5.2 Uncertainty in the Screening of Contaminants of Concern

The COCs were identified by comparing measured concentrations in settled dust to published Canadian (CCME, 1996) or American human health-based soil quality guidelines (see Annex G) since no settled dust guidelines exist. It is recognized that settled dust is a different medium than soil and in this regard, using soil guidelines as a screening tool is not ideal. However, soil quality guidelines for protection of human health are established very conservatively, generally being derived such that exposure to contaminants in the soil at the guideline concentration will deliver only 20% ($1/5^{th}$) of the acceptable or tolerable daily intake of the contaminant (CCME, 1996). Therefore, as an initial tool to identify potential COCs, these guidelines were considered a reasonable and conservative screening method, with the understanding that contamination in the settled dust samples in excess of the CCME guidelines does not necessarily constitute a health risk.

4.5.3 Uncertainty in Exposure Assessment

The current exposure assessment has provided a worst-case potential exposure to settled dust for crewmembers on board the HMCS CHICOUTIMI during the five-day voyage back to Faslane. The following represents a list of assumptions that were used during the development of exposure estimates:

- The highest contaminant concentration value out of the three settled dust samples was used for risk assessment purposes. Although this is a conservative approach, it is noted that only three samples were available and it is possible that higher levels of some compounds might have been present.

- Airborne levels of contaminants were extrapolated from settled dust samples since no airborne dust samples were obtained during the 5-day exposure period. The airborne dust levels were estimated by assuming worst-case scenario particulate matter levels (1 mg/m^3 or optically dense levels), that all of the particulate matter would have been respirable (able to reach deep into the lungs), and 100% of the contaminants absorbed to the particulate matter would be absorbed into the body.

- Exposure to dust through inadvertent ingestion was estimated assuming a 1 g/day ingestion rate and that 100% of the contaminants bound to the dust was absorbed into the body.

- Skin absorption of contaminants bound to dust was estimated using assumptions that were either similar to or more conservative then default assumptions utilized for construction workers.

4.5.4 Uncertainty in the Risk Characterization

The following conservative assumptions were used in development of toxicological criteria for the COCs in order to increase the confidence that potential risks would not be underestimated:

- TRVs utilized in the risk assessment are inherently conservative, as they are set at levels several fold lower than the lowest known effect level. This ensures that they are set at levels where there is a reasonably high degree of certainty that no effect would occur.

- The chronic TRV is based on an endpoint of questionable relevance to HMCS CHICOUTIMI crewmembers. For example, the dioxin TRV is based on effects in the off-spring of the exposed females. However, it was assumed that a TRV that was protective of more sensitive subpopulations then crewmembers would be a conservative approach.

4.6 Summary

The current risk assessment is based on a very limited number of soot samples from which inhalation, skin absorption and inadvertent ingestion were estimated. However, in an effort to overestimate potential exposures (and therefore minimize the chance of *under*estimating health risk), worst-case scenario assumptions were assumed. Despite these conservative assumptions, only minor deviations from the chronic TRVs were observed. It is therefore assumed that no adverse health effects to crewmembers of the HMCS CHICOUTIMI are expected from their 5-day exposure to settled soot and dust that existed on board after the fire.

5.0 Conclusions

The HMCS CHICOUTIMI fire of 5 Oct 2004 was a dramatic event that produced highly toxic smoke, as evidenced by the fact that unprotected exposure resulted in death and smoke inhalation injuries in several crewmembers. Exposure to the chemicals contained within smoke has the potential to result in acute injury, and may also cause long-term health problems. Uncertainties exist regarding the exact chemical exposures experienced by the crew as a result of the HMCS CHICOUTIMI fire incident. To address these uncertainties, this report has relied on available data, fundamental toxicological principles, accepted risk assessment approaches, conservative assumptions, and current medical and scientific knowledge in order to derive reasonable conclusions regarding potential long-term health risks.

In general, individuals who go on to develop long-term health problems from smoke inhalation injury show signs of these problems within hours to weeks after smoke inhalation. The crew of the HMCS CHICOUTIMI underwent close medical monitoring following the fire in order to ensure that individuals who may have been adversely affected by the incident were properly identified. For those crewmembers who may have persistent medical problems as a result of the HMCS CHICOUTIMI fire incident, it is important that they receive appropriate medical care and follow-up.

Crewmembers who were clinically well several months after the fire would be expected to remain well; it is not anticipated that they would go on to develop long-term health problems as a result of chemical exposures related to the HMCS CHICOUTIMI fire incident.

It must be emphasized that this report is specific to health effects related to chemical exposures only. As in any life-threatening scenario, the HMCS CHICOUTIMI incident represented significant psychological trauma. The effects of such trauma are experienced variably by different individuals, possibly causing psychological, cognitive, and/or physical symptoms. This is an important area for consideration regarding the long-term health of the crew, but this topic was beyond the scope of this report.

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Annex A

Time-line of Events Related to the HMCS CHICOUTIMI Fire

The following abridged time-line of events was extracted from the HMCS CHICOUTIMI fire Board of Inquiry report (Board of Inquiry, 2004).

5 October 2004

1315 hrs: onset of fire in Commanding Officer's Cabin with rapid spread through deck to Electrical Space below. Fire fighting efforts initiated. Upper conning tower lid opened. Emergency breathing system masks donned. Smoke boundaries established. Power shut down.

1325 hrs: Lt(N) Saunders found in Control Room

1330 hrs: Commanding Officer orders auxiliary power breaker remade to clear smoke using low pressure blower

1331 hrs: Subsequent report of fire. Laying off of all power.

1335 hrs: Fire out in Electrical Space

1443 hrs: Starboard engine started for smoke clearance

1446 hrs: Commanding Officer's Cabin fire reported out by Commanding Officer, overhauled

1625 hrs: Atmosphere in spec throughout submarine, all personnel off Emergency Breathing System

1730 hrs: Commanding Officer's Cabin and Electrical Space have been overhauled a second time

1957 hrs: atmosphere in spec throughout submarine

6 October 2004

1730 hrs: start of evacuation of three casualties via helicopter. Casualty transfer complete by 1801 hrs

1909: Lt(N) Chris Saunders pronounced dead in hospital in Sligo, Ireland

8 October 2004

1406 hrs: 21 people transferred to RFA Argus for rest and hot showers. Includes six of the original casualties

10 October 2004

1635 hrs: HMCS CHICOUTIMI alongside in Faslane, Scotland

Annex B

Extent of Damage From the HMCS CHICOUTIMI Fire

The following details were extracted from the HMCS CHICOUTIMI fire Board of Inquiry report (Board of Inquiry, 2004).

- The Commanding Officer's Cabin and Electrical Space both sustained severe damage. Limited heat damage was observed outside these two areas.

- The following extracts from the Fire Investigator's Report detail the extent of damage in the Commanding Officer's Cabin: bulkheads and deckhead were heavily damaged by fire and smoke/soot; the false bulkhead and false deckhead between the bunk and the pressure hull had very clear burn patterns and the finishing veneer was completely consumed; all light fixtures on the deckhead were melted with the exception of one heavy-duty light bulb; all thermoplastic items (light fixture, LCD screen, DVD, portable computer, headset, etc) were damaged almost beyond recognition; approximately one half of the mattress (forward to aft) was burnt through and one of the two sleeping bags found was almost completely consumed; the forward part of the Commanding Officer's bunk was completely destroyed; three electrical cables of approximately 2 inches in diameter were completely severed; a forth large cable (inboard) was damaged but not completely severed; a total of 10 cables (8 from Main Direct Current (DC) power and 2 from alternate supplies) were found running close to the deck. All cables had a certain amount of damage.

- The following extracts from the Fire Investigator's Report detail the extent of damage in the Electrical Space: a melted laptop computer; melted plastic and the remains of a burnt power cord were found on the deck; surface burning of the deckhead, bulkhead, ventilation ducts and power cables was observed.

- Based on burn patterns in the Commanding Officer's Cabin, the Fire Investigator concluded that the fire was rapidly brought under control. However, a large amount of smoke was produced during this short period of time. Based on the observed smoke damage, it was judged that visibility would have been almost zero from approximately 1 metre above deck on 1 and 2 deck forward of bulkhead 56 and aft of bulkhead 35 for a short period of time.

- Smoke and soot damage was limited to the midsection of the submarine: forward of bulkhead 56 and aft of bulkhead 35, and on both 1 Deck and 2 Deck. According to the Repair and Damage Assessment Team report the following compartments appeared to be unaffected by the fire: Weapon Stowage Compartment, Bunk Space, Sonar Cabinet Space, Mastwell Space, Auxiliary Machinery Space, Buoyant Wire Arial Compartment, Radio Office and all compartments aft of 56 bulkhead.

Annex C

Draeger Tube Background Technical Information

Draeger tube measurements rely on a chemical reaction. The tube contains inert silica that is impregnated with chemicals that react specifically with the target gas. These glass tubes are sealed at both ends prior to use. In order to take a measurement, the glass ends of the tube are broken off. One end of the tube is attached to a hand pump, and a known volume of air is drawn through the tube. The resultant chemical reaction with the target gas results in a colour change in the silica medium contained within the tube. The degree of colour change is then measured on the tube to determine an airborne concentration of the target gas, based on the volume of air that is drawn into the tube. According to manufacturer information, Draeger tube airborne concentration measurements have a standard deviation of +/- 5-15%.

If the tube is left "open" and the ends not sealed, diffusion of the target gas either into or out of the tube may occur. Therefore, it is difficult to interpret Draeger tube readings when the tube ends have been left open for prolonged periods of time. The reading of the tube must be done shortly after the measurement is obtained in order to obtain accurate results.

Because the silica matrix contained within the tube has no specific adsorptive capability (such as there would be with activated carbon or silica gel), the tubes would be unlikely to capture and retain other chemicals (Dräger Safety, Inc, 2004). For this reason, the HMCS CHICOUTIMI Draeger tubes only provide information on the specific target gases that they were designed to measure.

Annex D

Results of Draeger Tube Measurements Obtained On Board HMCS CHICOUTIMI Shortly After the Fire

The tables below provide the Draeger Tube gas measurement results as provided by the HMCS CHICOUTIMI Board of Inquiry. It should be noted that not all of the 6 measured gases were assessed at each location and at each time period. The symbol "-" indicates that no measurement result was available from the provided records.

Abbreviations:

ppm = parts per million
MR = Motor Room
CR = Control Room
A35 = aft of bulkhead 35
AMS = Auxiliary Machinery Space
F35 = forward of bulkhead 35
WSC = Weapons Storage Compartment
JR = Junior Ratings Mess

1. OXYGEN Draeger Tube Measurements (%)

Date	Time of Measurement	Elapsed time from fire onset (1315 hrs)	MR	CR	A35	AMS	F35	WSC	JR
5-Oct-04	1555 hrs	2 hrs, 40 min	18.5	18.5	20	19	14	18.5	-
5-Oct-04	1851 hrs	5 hrs, 36 min	19.5	-	20	-	-	18.2	-
5-Oct-04	1957 hrs	6 hrs, 42 min	-	-	-	-	19	-	-
5-Oct-04	2036 hrs	7 hrs, 21 min	20	20	20	19	19.5	19.5	-
5-Oct-04	2230 hrs	9 hrs, 15 min	19.5	20.7	20	18.5	19.5	-	-
5-Oct-04	2350 hrs	10 hrs, 35 min	19.5	-	-	-	19	19.2	-
6-Oct-04	0030 hrs	11 hrs, 15 min	-	18.5	-	20	-	-	-
6-Oct-04	0200 hrs	12 hrs, 45 min	19.75	20.5	-	19.5	19.5	19	20
6-Oct-04	0425 hrs	15 hrs, 10 min	19	19	-	20	19.5	19.5	19.5
6-Oct-04	0600 hrs	16 hrs, 45 min	19.1	18.5	19	20	19	19	-
6-Oct-04	0800 hrs	18 hrs, 45 min	19.5	20.5	20	19.5	20	19.5	-
6-Oct-04	1000 hrs	20 hrs, 45 min	19.5	20	20	19	19	19.5	-
6-Oct-04	1200 hrs	22 hrs, 45 min	19.5	20.5	20	19.5	19.5	20.5	-
6-Oct-04	1350 hrs	24 hrs, 35 min	19	19.25	20	20.5	20	20	-
6-Oct-04	1640 hrs	27 hrs, 25 min	19.5	18.75	-	19	19.25	19.75	-
6-Oct-04	1803 hrs	28 hrs, 48 min	19.5	20	19.5	20	19	20	-

2. CARBON DIOXIDE Draeger Tube Measurements (%)

Date	Time of Measurement	Elapsed time from fire onset (1315 hrs)	MR	CR	A35	AMS	F35	WSC	JR
5-Oct-04	1555 hrs	2 hrs, 40 min	0	0.5	0.5	0.5	0.5	0.5	-
5-Oct-04	1851 hrs	5 hrs, 36 min	0.2	-	0.3	-	-	0.5	-
5-Oct-04	1957 hrs	6 hrs, 42 min	-	-	-	-	0.5	-	-
5-Oct-04	2036 hrs	7 hrs, 21 min	0.5	0.5	0.5	0.5	0.4	0.4	-
5-Oct-04	2230 hrs	9 hrs, 15 min	1	0.1	0.3	0.1	0.5	-	-
5-Oct-04	2350 hrs	10 hrs, 35 min	0.1	-	-	-	0.5	0.2	-
6-Oct-04	0030 hrs	11 hrs, 15 min	-	0	-	0.1	-	-	-
6-Oct-04	0200 hrs	12 hrs, 45 min	0.1	0	-	0	4	0.1	0.1
6-Oct-04	0425 hrs	15 hrs, 10 min	0.1	0.1	-	0	0.3	0.3	0.1
6-Oct-04	0600 hrs	16 hrs, 45 min	0.1	0	0	0	0.3	0.3	-
6-Oct-04	0800 hrs	18 hrs, 45 min	0.1	0.1	0	0	0.1	0.2	-
6-Oct-04	1000 hrs	20 hrs, 45 min	0.1	0.1	0	0.1	0.4	0.2	-
6-Oct-04	1200 hrs	22 hrs, 45 min	0.1	0.1	0.1	0.1	0.2	0.2	-
6-Oct-04	1350 hrs	24 hrs, 35 min	0.1	0.1	0.1	0	0.2	0.2	-
6-Oct-04	1640 hrs	27 hrs, 25 min	0.1	0	-	0	0.1	0.25	-
6-Oct-04	1803 hrs	28 hrs, 48 min	0.2	0	0.2	0	0.1	0.1	-

3. CARBON MONOXIDE Draeger Tube Measurements (ppm)

Date	Time of Measurement	Elapsed time from fire onset (1315 hrs)	MR	CR	A35	AMS	F35	WSC	JR
5-Oct-04	1555 hrs	2 hrs, 40 min	15	0	10	10	70	50	-
5-Oct-04	1851 hrs	5 hrs, 36 min	0	-	0	-	-	25	-
5-Oct-04	1957 hrs	6 hrs, 42 min	-	-	-	-	40	-	-
5-Oct-04	2036 hrs	7 hrs, 21 min	0	0	0	10	0	0	-
5-Oct-04	2230 hrs	9 hrs, 15 min	0	0	0	0	2	-	-
5-Oct-04	2350 hrs	10 hrs, 35 min	0	-	-	-	0	0	-
6-Oct-04	0030 hrs	11 hrs, 15 min	-	0	-	0	-	-	-
6-Oct-04	0200 hrs	12 hrs, 45 min	0	0	-	0	0	0	0
6-Oct-04	0425 hrs	15 hrs, 10 min	0	0	-	0	0	0	0
6-Oct-04	0600 hrs	16 hrs, 45 min	0	0	0	0	0	0	-
6-Oct-04	0800 hrs	18 hrs, 45 min	0	0	0	0	0	0	-
6-Oct-04	1000 hrs	20 hrs, 45 min	0	0	0	0	0	0	-
6-Oct-04	1200 hrs	22 hrs, 45 min	-	-	-	-	-	-	-
6-Oct-04	1350 hrs	24 hrs, 35 min	-	-	-	-	-	-	-
6-Oct-04	1640 hrs	27 hrs, 25 min	-	-	-	-	-	-	-
6-Oct-04	1803 hrs	28 hrs, 48 min	-	-	-	-	-	-	-

4. NITROUS GASES Draeger Tube Measurements (ppm)

Date	Time of Measurement	Elapsed time from fire onset (1315 hrs)	MR	CR	A35	AMS	F35	WSC	JR
5-Oct-04	1555 hrs	2 hrs, 40 min	0.5	2	0.5	0.5	0.5	0.5	-
5-Oct-04	1851 hrs	5 hrs, 36 min	0.1	-	0.3	-	-	0.5	-
5-Oct-04	1957 hrs	6 hrs, 42 min	-	-	-	-	-	-	-
5-Oct-04	2036 hrs	7 hrs, 21 min	0	0.5	0.2	0.5	0	0	-
5-Oct-04	2230 hrs	9 hrs, 15 min	0	0	0.1	0	0	-	-
5-Oct-04	2350 hrs	10 hrs, 35 min	0	-	-	-	0	0	-
6-Oct-04	0030 hrs	11 hrs, 15 min	-	0	-	0	-	-	-
6-Oct-04	0200 hrs	12 hrs, 45 min	0	0	-	0	0	0	0
6-Oct-04	0425 hrs	15 hrs, 10 min	0	0	-	0	0	0	0
6-Oct-04	0600 hrs	16 hrs, 45 min	0	0	0	0	0	0	-
6-Oct-04	0800 hrs	18 hrs, 45 min	0	0	0	0	0	0	-
6-Oct-04	1000 hrs	20 hrs, 45 min	0	0	0	0	0	0	-
6-Oct-04	1200 hrs	22 hrs, 45 min	-	-	-	-	-	-	-
6-Oct-04	1350 hrs	24 hrs, 35 min	-	-	-	-	-	-	-
6-Oct-04	1640 hrs	27 hrs, 25 min	-	-	-	-	-	-	-
6-Oct-04	1803 hrs	28 hrs, 48 min	-	-	-	-	-	-	-

5. HYDROCHLORIC ACID Draeger Tube Measurements (ppm)

Date	Time of Measurement	Elapsed time from fire onset (1315 hrs)	MR	CR	A35	AMS	F35	WSC	JR
5-Oct-04	1555 hrs	2 hrs, 40 min	0	0	0	0	0.3	0	-
5-Oct-04	1851 hrs	5 hrs, 36 min	0	-	0	-	-	0.5	-
5-Oct-04	1957 hrs	6 hrs, 42 min	-	-	-	-	-	-	-
5-Oct-04	2036 hrs	7 hrs, 21 min	0	0	0	0	0	0	-
5-Oct-04	2230 hrs	9 hrs, 15 min	0	0	0	0	0	-	-
5-Oct-04	2350 hrs	10 hrs, 35 min	0	-	-	-	0	0	-
6-Oct-04	0030 hrs	11 hrs, 15 min	-	0	-	0	-	-	-
6-Oct-04	0200 hrs	12 hrs, 45 min	0	0	-	0	0	0	0
6-Oct-04	0425 hrs	15 hrs, 10 min	0	0	-	0	0	0	0
6-Oct-04	0600 hrs	16 hrs, 45 min	0	0	0	0	0	0	-
6-Oct-04	0800 hrs	18 hrs, 45 min	0	0	0	0	0	0	-
6-Oct-04	1000 hrs	20 hrs, 45 min	0	0	0	0	0	0	-
6-Oct-04	1200 hrs	22 hrs, 45 min	-	-	-	-	-	-	-
6-Oct-04	1350 hrs	24 hrs, 35 min	-	-	-	-	-	-	-
6-Oct-04	1640 hrs	27 hrs, 25 min	-	-	-	-	-	-	-
6-Oct-04	1803 hrs	28 hrs, 48 min	-	-	-	-	-	-	-

6. CHLORINE Draeger Tube Measurements (ppm)

Date	Time of Measurement	Elapsed time from fire onset (1315 hrs)	MR	CR	A35	AMS	F35	WSC	JR
5-Oct-04	1555 hrs	2 hrs, 40 min	-	-	-	-	-	-	-
5-Oct-04	1851 hrs	5 hrs, 36 min	-	-	-	-	-	-	-
5-Oct-04	1957 hrs	6 hrs, 42 min	-	-	-	-	0	-	-
5-Oct-04	2036 hrs	7 hrs, 21 min	0	0.5	0	0	0	0	-
5-Oct-04	2230 hrs	9 hrs, 15 min	0	0	0	0	0	-	-
5-Oct-04	2350 hrs	10 hrs, 35 min	-	-	-	-	-	-	-
6-Oct-04	0030 hrs	11 hrs, 15 min	-	-	-	-	-	-	-
6-Oct-04	0200 hrs	12 hrs, 45 min	-	-	-	-	-	-	-
6-Oct-04	0425 hrs	15 hrs, 10 min	-	-	-	-	-	-	-
6-Oct-04	0600 hrs	16 hrs, 45 min	-	-	-	-	-	-	-
6-Oct-04	0800 hrs	18 hrs, 45 min	-	-	-	-	-	-	-
6-Oct-04	1000 hrs	20 hrs, 45 min	-	-	-	-	-	-	-
6-Oct-04	1200 hrs	22 hrs, 45 min	-	-	-	-	-	-	-
6-Oct-04	1350 hrs	24 hrs, 35 min	-	-	-	-	-	-	-
6-Oct-04	1640 hrs	27 hrs, 25 min	-	-	-	-	-	-	-
6-Oct-04	1803 hrs	28 hrs, 48 min	-	-	-	-	-	-	-

Annex E

Smoke Density Values Used in NRC Cone Calorimeter Tests

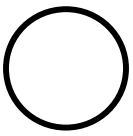
Smoke is a dynamic and complex substance that can be characterized in many different ways. In a laboratory setting, a key method for evaluating different physical characteristics of smoke involves the use of optical instruments that project and receive beams of light. A light beam of known intensity ("incident" light beam) is directed at a smoke cloud, travels through the smoke cloud, and then the light intensity of the beam is measured after it has passed through the smoke cloud. The light beam that has passed through the smoke is referred to as the "transmitted" light beam.

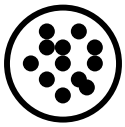
By comparing the intensity of the incident light beam to the intensity of the transmitted light beam, the amount of light blocked by the smoke can be determined. Essentially, the intensity of the incident light beam, minus the light that is blocked by the smoke, will be equal to the intensity of the transmitted light beam. If the intensity of the transmitted light beam is much less than the intensity of the incident light beam, then this is indicates that the smoke is relatively dense, or "thick".

Mathematically, the "light-blocking" aspect of smoke is described in terms of an area measurement, using units of meters squared (m^2). This unit can be visualized by imagining the light beam as a 3-dimensional tube, through which the light travels (i):

i) 

If the 3-dimensional tube is observed end-on, it has the appearance of a 2-dimensional circle (ii). If the area of this circle is partially filled by smoke particles that block light (iii), then there is less area available for light to travel through:

ii) 

iii) 

Therefore, the decrease in light intensity as it travels through the tube will be proportional to the area of the light path that is blocked by the smoke particles. A higher value of this light-blocking area, measured in units of meters squared (m^2), indicates denser smoke.

Units of smoke measurement used in the NRC Study

Smoke Production Rate:

- The smoke production rate describes the volume of smoke with a given density that is released per second. It is a function of the density of the smoke (in units of m^2, as discussed above) produced per unit of time. The unit of smoke production rate is meters squared per second (m^2/s). The higher the smoke production rate, the denser the smoke that is released per second.

Total Smoke:

- If the smoke production rate is known (smoke produced per unit of time), then combining this value with the total time that an object burns will indicate the total density of smoke that the burning object produced. Total smoke is dependent on how much of an object is consumed by fire. The smoke density (in units of m^2, as discussed above) is expressed in terms of the area of the object that has burned (also in units of m^2). Subsequently, the unit of total smoke is m^2/m^2 (smoke produced per area of burning material). The greater the total smoke value, then the greater the amount of dense smoke that is produced from a burning object.

Specific Extinction Area:

- Specific extinction area provides a measure of smoke density (in units of m^2, as discussed above) per mass of the object that is burned. The unit of specific extinction area is meters squared per kilogram of burned material (m^2/kg). Objects with high specific extinction area values will produce denser smoke than objects with lower values.

Annex F

Description of Air, Wipe, and Filter Samples

Air Samples

The air samples were reviewed in a previous toxicological report that focused on the situation on board prior to clean-up while the vessel was in port, and relevant to the CF Personnel and visitors who may have been exposed during that time (Halton et al., 2005). The toxicological assessment of the air samples revealed that none of the compounds exceeded their respective health threshold limit value and that no adverse health effects would have been expected at that particular time. However, these samples were considered to be of limited use for the present risk assessment since they were taken too long after HMCS CHICOUTIMI incident and would not have been representative of air quality during the 5-day transit to Faslane. The air samples were therefore not used for the current risk assessment.

Wipe Samples

Several wipe samples were analyzed for heavy metals content only. Wipe sample results are not ideal to estimate potential exposures since they are expressed as a mass of metal per surface area that was wiped (mg of metal/cm^2) rather than being expressed as a concentration (mg of metal/mg of total dust). A high mass of metal could be wiped from a given surface simply because a lot of dust had accumulated in that area and not necessarily because that area was particularly contaminated. Therefore, it was concluded that the results of settled dust samples (which determined chemical concentrations) would provide a better exposure parameter.

Filter Samples

Filter samples were analyzed for various chemicals of potential concern. The air filter results would not have captured the airborne contaminants released during the fire or shortly after since the ventilation system was shut down at the time of the fire (DMEPM(SM) 4-4-6, 2008). Passive deposition of particulates released from the fire could have occurred but would not necessarily have been representative of the atmospheric fire conditions. It was also suggested that the particulates detected on the ventilation filters more likely represented pre-fire conditions (DMEPM(SM) 4-4-6, 2008). The results of filter analyses were therefore not used for the exposure estimation.

Annex G
Identification of Chemicals of Potential Concern for Human Health in Settled Dust

Identification of chemicals of potential concern for human health in settled dust

Exceeds the human health guideline

Detection limit exceeds the guideline

Maximum measured concentration in settled dust versus risk-based soil guidelines

Analyte	Sampling location	[Max] mg/kg	DL mg/kg	CCME mg/kg[a]	EPA Reg9 mg/kg[b]
Ag (Silver)	Firing control area	29	20	-	100000
Al (Aluminum)	**CO Cabin**	**41000**	**100**	**40**	**1020**
As (Arsenic)	-	<DL	100	12	1.6
B (Boron)	Electrical space	260	100	-	100000
Ba (Barium)	CO Cabin	2100	100	-	13400
Be (Berylium)	-	<DL	40	-	1900
Ca (Calcium)	Firing control area	140000	1000	-	-
Cd (Cadmium)	-	<DL	50	2090	90
Co (Cobalt)	CO Cabin	550	100	300	1900
Cr (Chromium)	Electrical space	160	100	2300	450
Cu (Copper)	**CO Cabin**	**54000**	**100**	**20000**	**8200**
Fe (Iron)	CO Cabin	20000	100	-	100000
Hg (Mercury)	Firing control area	8.2	0.1	690	62
K (Potassium)	Firing control area	43000		-	-
Li (Lithium)	-	<DL	100	-	-
Mg (Magnesium)	CO Cabin	42000	1000	-	-
Mn (Manganese)	Electrical space	250	100	-	3800
Mo (Molibdenum)	-	<DL	100	-	1020
Na (Sodium)	Firing control area	15000	1000	-	-
Ni (Nickel)	Electrical space	450	100	-	4000
P (Phospherus)	CO Cabin	1500	100	-	-
Pb (Lead)	Electrical space	1120	100	8200	150
Sb (Antimony)	**Firing control area**	**2300**	**100**	**40**	**82**
Se (Selenium)	-	<DL	100	4700	1020
Sn (Tin)	Firing control area	150	100	300	100000
Si (Silicon)	Electrical space	1200	100	-	-
Sr (Strontium)	CO Cabin	230	100	-	100000
Ti (Titanium)	CO Cabin	1300	100	-	-
Tl (Thallium)	-	<DL	100	1	13
U (Uranium)	-	<DL	50	-	40
V (Vanadium)	-	<DL	100	130	1440
Zn (Zinc)	Firing control area	22000	100	-	100000

Analyte	Sampling location	[Max] pg/g	[Max] pgTEQ/g	DL pg/g	CCME pgTEQ/g
Total Dioxins & Furans	**Electrical space**	**170892**	**915**	**20 pg/g**	**175**

Analyte	Sampling location	[Max] pg/g
Asbestos content	CO Cabin	None detected

CAS	Analyte	Sampling location	[Max] mg/kg	DL mg/kg	CCME mg/kg	EPA Reg9 mg/kg
83-32-9	Acenaphthene	Electrical space	0.04	0.0005	-	-
208-96-8	Acenaphthylene	Electrical space	0.71	0.0005	-	-
120-12-7	Anthracene	Electrical space	0.25	0.0005	-	100000
56-55-3	Benzo(a)anthracene	Electrical space	0.13	0.0005	-	2.1
50-32-8	Benzo(a)pyrene	Electrical space	0.09	0.0005	1.5	0.21
205-99-2	Benzo(b)fluoranthene	Electrical space	0.09	0.0005	10	2.1
192-17-2	Benzo(e)pyrene	Electrical space	0.08	0.0005	-	-
191-24-2	Benzo(ghi)perylene	Electrical space	0.07	0.0005	-	-
207-08-9	Benzo(k)fluoranthene	Electrical space	0.08	0.0005	10	21
92-52-4	Biphenyl	Electrical space	0.32	0.0005	-	70
218-01-9	Chrysene	Electrical space	0.17	0.0005	-	2100
53-70-3	Dibenzo(a,h)anthracene	Electrical space	0.02	0.0005	10	0.21
206-44-0	Fluoranthene	Electrical space	0.48	0.0005	-	4400

86-73-7	Fluorene	Electrical space	0.35	0.0005	-	5200
193-39-5	Indeno(1,2,3-cd)pyrene	Electrical space	0.06	0.0005	10	2.1
91-20-3	Naphthalene	Electrical space	0.76	0.0005	22	38
198-55-0	Perylene	Electrical space	0.02	0.0005	-	-
85-01-8	Phenanthrene	Electrical space	1.18	0.0005	50	-
129-00-0	Pyrene	Electrical space	0.42	0.0005	100	5800

CAS	Analyte	Sampling location	[Max] mg/kg	DL mg/kg	CCME mg/kg	EPA Reg9 mg/kg
71-55-6	1,1,1-Trichloroethane	-	<DL	0.01	50	340
79-34-5	1,1,2,2-Tetrachloroethane	-	<DL	0.01	50	0.9
79-00-5	1,1,2-Trichloroethane	-	<DL	0.01	50	1.6
75-34-3	1,1-Dichloroethane	-	<DL	0.01	50	340
75-35-4	1,1-Dichloroethene	-	<DL	0.01	50	82
96-18-4	1,2,3-Trichloropropane	-	<DL	0.01	-	0.01
120-82-1	1,2,4-Trichlorobenzene	-	<DL	0.01	10	600
95-63-6	1,2,4-Trimethylbenzene	CO Cabin	0.034	0.01	-	34
106-93-4	1,2-Dibromoethane	-	<DL	0.01	-	0.03
95-50-1	1,2-Dichlorobenzene	-	<DL	0.01	10	74
107-06-2	1,2-Dichloroethane	-	<DL	0.01	50	0.6
78-87-5	1,2-Dichloropropane	-	<DL	0.01	50	0.74
108-67-8	1,3,5-Trimethylbenzene	CO Cabin	0.017	0.01	-	1.4
541-73-1	1,3-Dichlorobenzene	-	<DL	0.01	10	13
106-46-7	1,4-Dichlorobenzene	-	<DL	0.01	10	7.9
78-93-3	2-Butanone	CO Cabin	0.16	0.01	-	-
110-75-8	2-Chloroethyl vinyl ether	-	<DL	0.1	-	-
591-78-6	2-Hexanone	-	<DL	0.1	-	-
108-10-1	4-Methyl-2-Pentanone	-	<DL	0.1	-	-
67-64-1	Acetone	CO Cabin	0.28	0.1	-	-
107-13-1	Acrylonitrile	-	<DL	0.1	-	0.21
71-43-2	Benzene	CO Cabin	0.015	0.01	5	1.3
75-27-4	Bromodichloromethane	-	<DL	0.01	-	1.8
75-25-2	Bromoform	-	<DL	0.01	-	220
74-83-9	Bromomethane	CO Cabin	0.02	0.01	-	2.6
75-15-0	Carbon Disulfide	-	<DL	0.1	-	72
56-23-5	Carbon Tetrachloride	-	<DL	0.01	50	0.55
108-90-7	Chlorobenzene	-	<DL	0.01	10	106
75-00-3	Chloroethane	-	<DL	0.01	-	6.5
67-66-3	Chloroform	-	<DL	0.01	50	12
74-87-3	Chloromethane	CO Cabin	0.073	0.01	-	2.6
156-59-2	cis-1,2-Dichloroethene	-	<DL	0.01	50	30
10061-01-5	cis-1,3-Dichloropropene	-	<DL	0.01	-	-
124-48-1	Dibromochloromethane	-	<DL	0.01	-	2.6
74-95-3	Dibromomethane	-	<DL	0.01	-	46
75-71-8	Dichlorodifluoromethane	-	<DL	0.01	-	62
100-41-4	Ethylbenzene	-	<DL	0.01	20	20
76-13-1	Freon-113	-	<DL	0.01	-	1120
98-82-8	Isopropylbenzene	-	<DL	0.01	-	400
08-38-3 + 106-42-3	m+p-Xylene	-	<DL	0.02	20	84
1634-04-4	Methyl tert-butyl ether	-	<DL	0.01	-	62
75-09-2	Methylene chloride	-	<DL	0.01	50	21
142-82-5	n-Heptane	-	<DL	0.01	-	-
110-54-3	n-Hexane	-	<DL	0.01	-	22
95-47-6	o-Xylene	-	<DL	0.01	20	84
100-42-5	Styrene	CO Cabin	0.011	0.01	50	340
127-18-4	Tetrachloroethene	-	<DL	0.01	0.6	3.4
109-99-9	Tetrahydrofuran	-	<DL	0.1	-	9.4
108-88-3	Toluene	CO Cabin	0.019	0.01	0.8	104
156-60-5	trans-1,2-Dichloroethene	-	<DL	0.1	50	46
10061-02-6	trans-1,3-Dichloropropene	-	<DL	0.1	-	-
79-01-6	Trichloroethene	-	<DL	0.1	31	0.11

75-69-4	Trichlorofluoromethane	-	<DL	0.1	-	400
108-05-4	Vinyl acetate	-	<DL	0.1	-	86
75-01-4	Vinyl chloride	-	<DL	0.1	-	0.75
122-66-7	1,2-Diphenylhydrazine	-	<DL	5	-	2.2
88-06-2	2,4,6-Trichlorophenol	-	<DL	5	5	12
95-95-4	2,4,5-Trichlorophenol	-	<DL	30	5	124000
120-83-2	2,4-Dichlorophenol	-	<DL	5	5	360
105-67-9	2,4-Dimethylphenol	-	<DL	5	10	2400
51-28-5	2,4-Dinitrophenol	-	<DL	26	10	240
121-14-2	2,4-Dinitrotoluene	-	<DL	5	-	2.5
606-20-2	2,6 Dinitrotoluene	-	<DL	5	-	2.5
91-58-7	2-Chloronaphthalene	-	<DL	5	-	4600
95-57-8	2-Chlorophenol	-	<DL	5	5	48
91-57-6	2-Methylnaphthalene	-	<DL	5	-	-
95-48-7	2-Methylphenol	-	<DL	5	-	6200
88-75-5	2-Nitrophenol	-	<DL	26	10	-
91-94-1	3,3'-Dichlorobenzidine	-	<DL	30	-	0.76
99-09-2	3-Nitroaniline	-	<DL	26	-	-
534-52-1	4,6-Dinitro-2-methylphenol	-	<DL	26	-	-
101-55-3	4-Bromophenylphenylether	-	<DL	5	-	-
59-50-7	4-Chloro-3-methylphenol	-	<DL	5	-	-
106-47-8	4-Chloroaniline	-	<DL	20	-	48
7005-72-3	4-Chlorophenylphenylether	-	<DL	5	-	-
100-01-6	4-Nitroaniline	-	<DL	26	-	-
100-02-7	4-Nitrophenol	-	<DL	5	10	-
92-87-5	Benzidine	-	<DL	5	-	0.0021
65-85-0	Benzoic acid	-	<DL	50	-	100000
100-51-6	Benzyl alcohol	-	<DL	5	-	3600
111-91-1	Bis(2-chloroethoxy)methane	-	<DL	5	-	-
111-44-4	Bis(2-chloroethyl)ether	-	<DL	5	-	0.55
108-60-1	Bis(2-chloroisopropyl)ether	-	<DL	5	-	7.4
117-81-7	Bis(2-ethylhexyl)phthalate	Firing control area	270	5	-	1200
85-68-7	Butylbenzylphthalate	-	<DL	5	-	100000
84-66-2	Diethylphthalate	-	<DL	5	-	100000
131-11-3	Dimethylphthalate	-	<DL	5	-	100000
84-74-2	Dibutylphthalate	Firing control area	14	5	-	12400
117-84-0	Di-n-octylphthalate	-	<DL	5	-	5000
118-74-1	Hexachlorobenzene	-	<DL	5	10	1.1
87-68-3	Hexachlorobutadiene	-	<DL	5	-	22
77-47-4	Hexachlorocyclopentadiene	-	<DL	5	-	740
67-72-1	Hexachloroethane	-	<DL	5	-	120
78-59-1	Isophorone	-	<DL	5	-	1800
98-95-3	Nitrobenzene	-	<DL	5	-	20
62-75-9	N-nitrosodimethylamine	-	<DL	5	-	0.034
621-64-7	N-nitrosodi-n-propylamine	-	<DL	5	-	0.25
86-30-6	N-nitrosodiphenylamine	-	<DL	5	-	3500
87-86-5	Pentachlorophenol	-	<DL	26	7500	9
108-95-2	Phenol	Firing control area	12	5	150000	100000

Notes

[a]CCME industrial soil for the protection of human health was used when available. When such guideline was not published, the 1991 CCME interim remediation criterion was used.

[b]20% of the EPA Reg9 industrial soil guidelines for non-carcinogenic compounds and 100% of the carcinogenic compounds.

DL = Analytical dection limit

[Max] = Maximum concentration of compound measured from the three availble soot sample results

CAS = Chemical Abstracts Service Number

Annex H

Settled Dust Calculation for Contaminants of Potential Concern

General Equations Used to Estimate Doses*

INADVERTENT INGESTION OF DUST

The predicted intake of each element via dust ingestion is calculated as:

$$Dose\ \ (mg/kgbw\ \ / day) = \frac{C_S \times IR_S \times AF_{GIT} \times D_1 \times D_2}{BW}$$

Where:

C_S = concentration of element in floor dust (mg/kg)
IR_S = adult human floor dust ingestion rate (kg/d)
AF_{GIT} = absorption factor from the gastrointestinal tract (unitless)
D_1 = days per week exposed/7 days
D_2 = weeks per year exposed/52 weeks
BW = body weight (kg)

SKIN ABSORPTION OF DUST

The predicted intake of each element via skin absorption of dust is calculated as:

$$Dose\,(mg/kgbw/day) = \frac{[(C_S \times SA_H \times SL_H) + (C_S \times SA_{Other} \times SL_{Other})] \times AF_{Skin} \times EF \times D_1 \times D_2}{BW}$$

Where

C_S = concentration of element in floor dust (mg/kg)
SA_H = surface area of hands (cm^2)
SL_H = floor dust loading to hands (kg/cm^2-event)
SA_{Other} = surface area of exposed body surfaces other than hands (head, legs and arms) (cm^2)
SL_{Other} = floor dust loading to exposed body surfaces other than hands (kg/cm^2-event)
AF_{Skin} = dermal absorption factor (unitless)
EF = exposure frequency (events/d)
D_1 = days per week exposed/7 days
D_2 = weeks per year exposed/52 weeks
BW = body weight (kg)

INHALATION OF DUST

The predicted intake of each element via inhalation of dust is calculated as:

$$Dose\ (mg/kg\ bw\ /day) = C_S \times P_{air} \times IR_a \times AF_{inh} \times D_1 \times D_2 \times D_3 / BW$$

Where:

C_S = concentration of element in floor dust (mg/kg)
P_{air} = particulate concentration in air (mg/m^3)
AF_{inh} = inhalation absorption factor (unitless)
IR_a = air intake rate (m^3/h)
D_1 = hours per day exposed
D_2 = days per week exposed/7 days
D_3 = weeks per year exposed/52 weeks
BW = body weight (kg)

***Note: These are generalized equations only. The chemical-specific detailed exposure estimations follow.**

Inhalation of dust containing dioxins/furans

Dose (pg TEQ/kg bw/day) = C_d x P_{air} x IR_a x AF_{inh} x D_1 x D_2 x D_3 / BW

Where:

C_d = 915 pg TEQ/g floor dust or 915000 pg TEQ/kg
P_{air} = 1 mg/m^3 or 0.000001 kg/m^3
IR_a = 1.6 m^3/hour
AF_{inh} = 1 (unitless) (assumes complete absorption)
D_1 = 24 hour/day
D_2 = 5 days/7 days = 0.71
D_3 = 1 week/52 weeks = 0.019
BW = 70 kg

Dose = (915000 x 0.000001 x 1.6 x 1 x 24 x 0.71 x 0.019) / 70 = 0.0069 pg TEQ/kg bw/day

Inadvertent ingestion of dust containing dioxins/furans

Dose (pg TEQ/kg bw/day) = (C_d x IR_d x AF_{git} x D_1 x D_2) / BW

Where:

C_d = 915 pg TEQ/g floor dust or 915000 pg TEQ/kg
IR_d = 1 g floor dust ingested/day or 0.001 kg/day
AF_{git} = 1 (unitless) (assumes complete absorption)
D_1 = 5 days/7 days = 0.71
D_2 = 1 week/52 weeks = 0.019
BW = 70 kg

Dose = (915000 x 0.001 x 1 x 0.71 x 0.019) / 70 = 0.18 pg TEQ/kg bw/day

Absorption of dioxins/furans through skin contact with dust

Dose (pg TEQ/kg bw/day) = [[(C_d x SA_H x SL_H) +(C_d x SA_{other} x SL_{other})] x AF_{skin} x EF x D_1 x D_2] / BW

Where:

C_d = 915 pg TEQ/g floor dust or 915000 pg TEQ/kg
SA_H = 880 cm^2 (hands)
SA_{other} = 9220 cm^2 (head, arms, legs)
SL_H = 1 mg/cm^2 per floor dust contact event (hands) or 0.000001 kg/cm^2
SL_{other} = 0.1 mg/cm^2 per floor dust contact event (head, arms, legs) or 0.0000001 kg/cm^2
AF_{skin} = 0.01 (i.e. one percent of dioxins/furans in contact with skin is absorbed)
EF = 1 event/day
D_1 = 5 days/7 days = 0.71
D_2 = 1 week/52 weeks = 0.019
BW = 70 kg

Dose = [[(915000 x 880 x 0.000001)+(0.000915 x 9220 x 0.0000001)] x 0.01 x 1 x 0.71 x 0.019] / 70
Dose = 0.0032 pg TEQ/kg bw/day

Inhalation of dust containing aluminium

Dose (mg/kg bw/day) = C_d x P_{air} x IR_a x AF_{inh} x D_1 X D_2 / BW

Where:

C_d = 41000 mg/kg
P_{air} = 1 mg/m^3 or 0.000001 kg/m^3
IR_a = 1.6 m^3/hour
AF_{inh} = 1 (unitless) (assumes complete absorption)
D_1 = 24 hour/day
D_2 = 5 days/7 days = 0.71
BW = 70 kg

Dose = (41000 x 0.000001 x 1.6 x 1 x 24 x 0.71) / 70 = 0.016 mg/kg bw/day

Inadvertent ingestion of dust containing aluminium

Dose (mg/kg bw/day) = (C_d x IR_d x AF_{git} x D_1) / BW

Where:

C_d = 41000 mg/kg
IR_d = 1 g floor dust ingested/day or 0.001 kg/day
AF_{git} = 1 (unitless) (assumes complete absorption)
D_1 = 5 days/7 days = 0.71
BW = 70 kg

Dose = (41000 x 0.001 x 1 x 0.71) / 70 = 0.42 mg/kg bw/day

Absorption of aluminum through skin contact with dust

Dose (mg/kg bw/day) = [[(C_d x SA_H x SL_H) +(C_d x SA_{other} x SL_{other})] x AF_{skin} x EF x D_1] / BW

Where:

C_d = 41000 mg/kg
SA_H = 880 cm^2 (hands)
SA_{other} = 9220 cm^2 (head, arms, legs)
SL_H = 1 mg/cm^2 per floor dust contact event (hands) or 0.000001 kg/cm^2
SL_{other} = 0.1 mg/cm^2 per floor dust contact event (head, arms, legs) or 0.0000001 kg/cm^2
AF_{skin} = 0.01 (i.e. one aluminum in contact with skin is absorbed)
EF = 1 event/day
D_1 = 5 days/7 days = 0.71
BW = 70 kg

Dose = [[(41000 x 880 x 0.000001)+(41000 x 9220 x 0.0000001)] x 0.01 x 1 x 0.71] / 70
Dose = 0.0075 mg/kg bw/day

Inhalation of dust containing copper

Dose (mg/kg bw/day) = C_d x P_{air} x IR_a x AF_{inh} x D_1 X D_2 / BW

Where:

C_d = 54000 mg/kg
P_{air} = 1 mg/m^3 or 0.000001 kg/m^3
IR_a = 1.6 m^3/hour
AF_{inh} = 1 (unitless) (assumes complete absorption)
D_1 = 24 hour/day
D_2 = 5 days/7 days = 0.71
BW = 70 kg

Dose = (54000 x 0.000001 x 1.6 x 1 x 24 x 0.71) / 70 = 0.021 mg/kg bw/day

Inadvertent ingestion of dust containing copper

Dose (mg/kg bw/day) = (C_d x IR_d x AF_{git} x D_1) / BW

Where:

C_d = 54000 mg/kg
IR_d = 1 g floor dust ingested/day or 0.001 kg/day
AF_{git} = 1 (unitless) (assumes complete absorption)
D_1 = 5 days/7 days = 0.71
BW = 70 kg

Dose = (54000 x 0.001 x 1 x 0.71) / 70 = 0.55 mg/kg bw/day

Absorption of copper through skin contact with dust

Dose (mg/kg bw/day) = [[(C_d x SA_H x SL_H) +(C_d x SA_{other} x SL_{other})] x AF_{skin} x EF x D_1] / BW

Where:

C_d = 54000 mg/kg
SA_H = 880 cm^2 (hands)
SA_{other} = 9220 cm^2 (head, arms, legs)
SL_H = 1 mg/cm^2 per floor dust contact event (hands) or 0.000001 kg/cm^2
SL_{other} = 0.1 mg/cm^2 per floor dust contact event (head, arms, legs) or 0.0000001 kg/cm^2
AF_{skin} = 0.01 (i.e. one percent of copper in contact with skin is absorbed)
EF = 1 event/day
D_1 = 5 days/7 days = 0.71
BW = 70 kg

Dose = [[(54000 x 880 x 0.000001)+(54000 x 9220 x 0.0000001)] x 0.01 x 1 x 0.71] / 70
Dose = 0.0099 mg/kg bw/day

Inhalation of dust containing antimony

Dose (mg/kg bw/day) = C_d x P_{air} x IR_a x AF_{inh} x D_1 X D_2 / BW

Where:

C_d = 2300 mg/kg
P_{air} = 1 mg/m^3 or 0.000001 kg/m^3
IR_a = 1.6 m^3/hour
AF_{inh} = 1 (unitless) (assumes complete absorption)
D_1 = 24 hour/day
D_2 = 5 days/7 days = 0.71
BW = 70 kg

Dose = (2300 x 0.000001 x 1.6 x 1 x 24 x 0.71) / 70 = 0.00094 mg/kg bw/day

Inadvertent ingestion of dust containing antimony

Dose (mg/kg bw/day) = (C_d x IR_d x AF_{git} x D_1) / BW

Where:

C_d = 2300 mg/kg
IR_d = 1 g floor dust ingested/day or 0.001 kg/day
AF_{git} = 1 (unitless) (assumes complete absorption)
D_1 = 5 days/7 days = 0.71
BW = 70 kg

Dose = (2300 x 0.001 x 1 x 0.71) / 70 = 0.023 mg/kg bw/day

Absorption of antimony through skin contact with dust

Dose (mg/kg bw/day) = [[(C_d x SA_H x SL_H) +(C_d x SA_{other} x SL_{other})] x AF_{skin} x EF x D_1] / BW

Where:

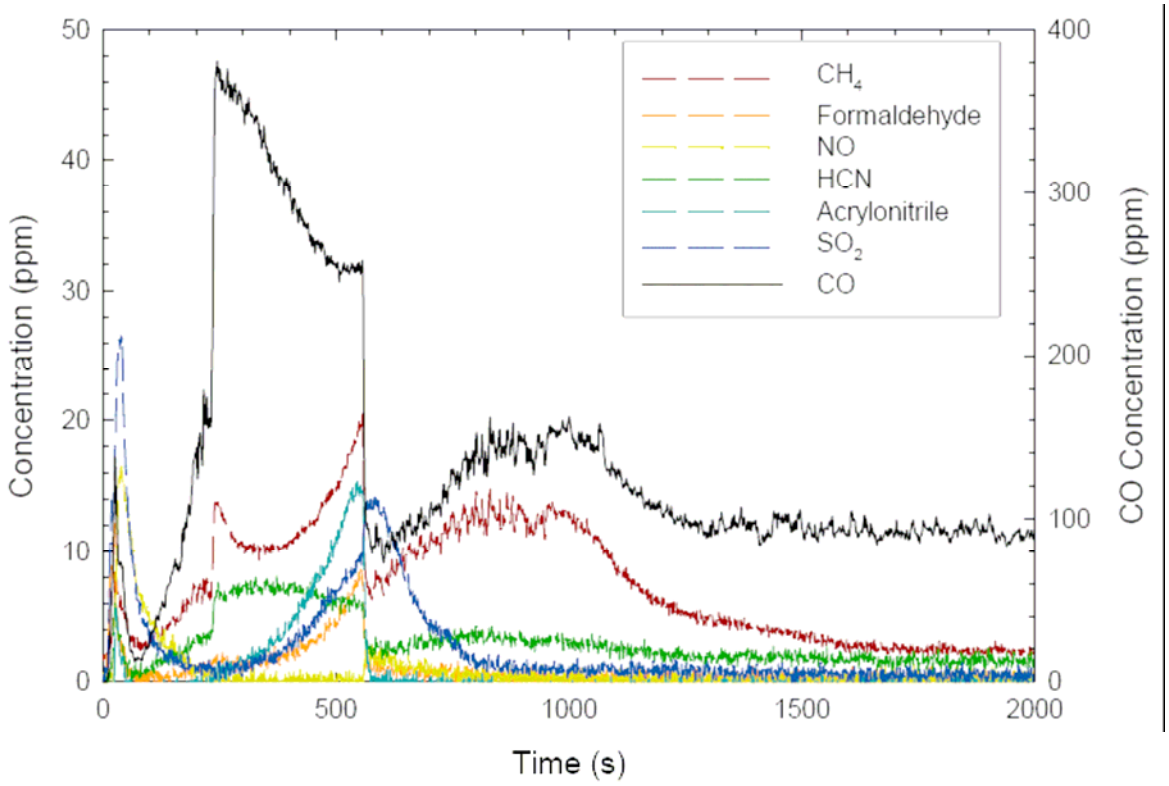
C_d = 2300 mg/kg
SA_H = 880 cm^2 (hands)
SA_{other} = 9220 cm^2 (head, arms, legs)
SL_H = 1 mg/cm^2 per floor dust contact event (hands) or 0.000001 kg/cm^2
SL_{other} = 0.1 mg/cm^2 per floor dust contact event (head, arms, legs) or 0.0000001 kg/cm^2
AF_{skin} = 0.01 (i.e. one percent of antimony in contact with skin is absorbed)
EF = 1 event/day
D_1 = 5 days/7 days = 0.71
BW = 70 kg

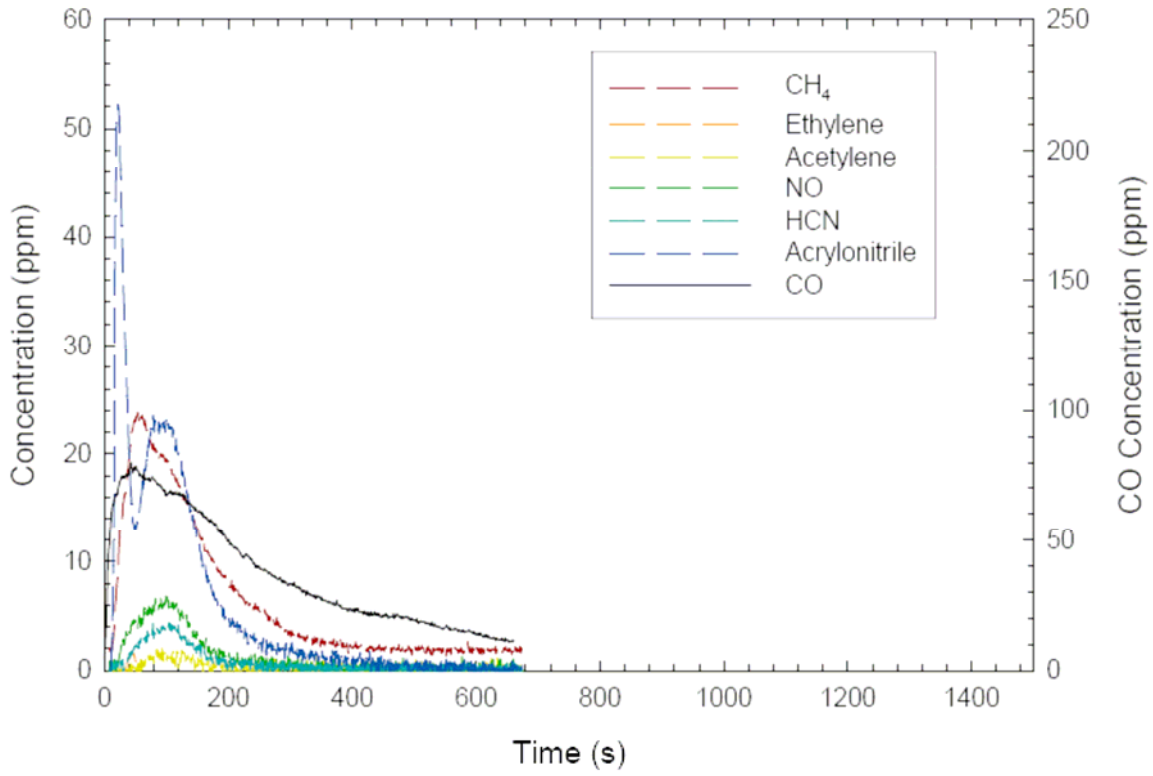
Dose = [[(2300 x 880 x 0.000001)+(2300 x 9220 x 0.0000001)] x 0.01 x 1 x 0.71] / 70
Dose = 0.00042 mg/kg bw/day

Annex I

Examples of gas analysis figures contained within the National Research Council of Canada Cone Calorimeter Report

Note that the CO concentration scale is provided on the right vertical axis. CH_4 = methane, NO = nitric oxide, HCN = hydrogen cyanide, SO_2 = sulphur dioxide, CO = carbon monoxide, HCl = hydrogen chloride, HF = hydrogen fluoride ppm = parts per million.

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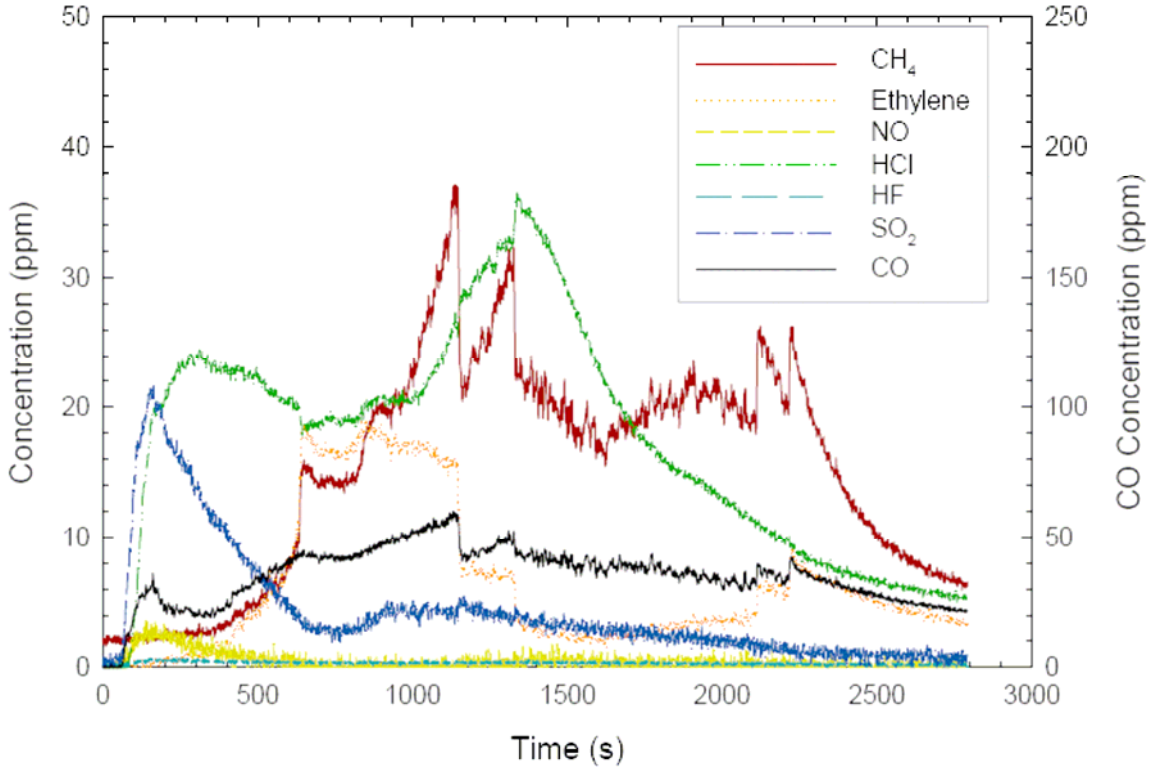
CO Concentration (ppm)
Time (s)
CH4
Ethylene
Acetylene
NO
HCN
Acrylonitrile
CO
0
10
20
30
40
50
60
0
50
100
150
200
250
0
200
400
600
800
1000
1200
1400


Concentration (ppm)
CO Concentration (ppm)
Time (s)
CH4
Ethylene
NO
HCl
HF
SO2
CO
0
10
20
30
40
50
0
50
100
150
200
250
0
500
1000
1500
2000
2500
3000

Example illustration of identification of peak gas concentrations, as discussed in Section 2.3.1.3. (Note that not all gas peaks are labeled in the figure)