APPENDIX I

Toxicological Profiles

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1.0 ACETALDEHYDE

Synonyms: acetaldehyde; acetic aldehyde; acetylaldehyde; aldehyde; ethanal; ethylaldehyde; methyl formaldehyde

1.1 PHYSICAL/CHEMICAL PROPERTIES

Acetaldehyde is a colourless liquid with a pungent, fruity odour at room temperature (AIHA, 1989). Reported odour threshold values range from approximately 2.7 to 1,800,000 $\mu g/m^3$ with a mean odour threshold of 90 $\mu g/m^3$ (van Gemert, 1999; Amoore and Hautala, 1983).

1.2 SOURCES

Acetaldehyde is used primarily as a chemical intermediate for the production of acetic acid and related chemicals. It is released via natural sources such as forest and brush fires, and can be formed by the irradiation of humic substances in water by sunlight (CEPA, 2000). Anthropogenic sources include incomplete combustion of gasoline, cigarette smoking, furnaces, power plants, waste incinerators, fireplaces and the cooking of food. Emissions of acetaldehyde have been detected from pulp and paper mills, tire and rubber plants, food-processing facilities, coal processing plants and petroleum refining (CEPA, 2000).

Acetaldehyde is a natural component of many foods and is generated during the ripening of fruit, cooking, baking and the storage and maturation of alcoholic beverages (CEPA, 2000). It is generally recognized as safe in the United States, and is used in Canada and the United States as a synthetic flavouring substance and adjuvant in various foods (*e.g.*, dairy and meat products, juices, baked goods, alcoholic and non-alcoholic beverages, gelatine desserts and candy) (CEPA, 2000).

1.3 ENVIRONMENTAL FATE

Acetaldehyde released into the air will react with photochemically produced hydroxyl radicals and may form chemicals such as peroxyacetyl nitrate, formaldehyde, peroxyacetic acid and acetic acid (CEPA, 2000). The half-life of acetaldehyde in the atmosphere is expected to be less

than 6 hours suggesting a limited potential for long-range transport. In water, acetaldehyde is expected to biodegrade within several days. Its half-life in surface water is estimated to be between 30 and 100 hours (CEPA, 2000). In addition, based on its organic carbon partition coefficient of less than two, acetaldehyde is not expected to adsorb on to soil and is therefore considered only moderately mobile.

1.4 SYMPTOMS OF OVEREXPOSURE

Acute (short-term) health effects for acetaldehyde include irritation of the skin, eyes and respiratory tract (US EPA, 1994). The acute toxicity of acetaldehyde is low, but at very high concentrations symptoms can include central nervous system depression, reduced respiratory rate, increased heart rate and blood pressure, pulmonary edema and albuminuria (CEPA, 2000).

Based on animal studies, the International Agency for Research on Cancer (IARC) has classified acetaldehyde as possibly carcinogenic to humans (IARC, 1999). Although it is a genotoxic chemical, tumours have been observed following inhalation only at concentrations that have produced significant cytotoxicity (CEPA, 2000). Thus, it is likely that both genotoxicity and irritancy of acetaldehyde play a role in its carcinogenicity.

1.5 EXPOSURE LIMITS

An acute inhalation exposure limit for acetaldehyde of 500 $\mu g/m^3$ was employed for the current assessment based on a short-term acetaldehyde exposure limit recommended by the Ontario Ministry of the Environment and Energy (OMOE). OMOE (2001) recommended an exposure limit of 500 $\mu g/m^3$ for both 24-hour average exposure and 30-minute exposure, presumably because the short-term toxicity of acetaldehyde is more dependent on concentration than duration of exposure (CEPA, 2000). This exposure limit was derived based on health considerations, but the specific basis was not provided (OMOE, 2001).

The Risk-specific Concentration (RsC) of 17.2 $\mu g/m^3$ used for this assessment was based on the tumorigenic concentration for inhalation exposure recommended by Health Canada (CEPA, 2000). The RsC was developed from a tumorigenic concentration (TC_{05}) of 86 mg/m^3, which is associated with a 5% increase in nasal cavity tumours in the most sensitive sex (males) of Wistar

rats (Woutersen *et al.*, 1986). The TC_{05} was adjusted by CEPA for intermittent to continuous exposure (*i.e.*, six hours a day for five days a week). This objective represents the daily dose *via* inhalation that is associated with an increased cancer risk of one in 100,000. The RsC of 17.2 $\mu g/m^3$ was converted to an exposure limit of 5.65 μg/kg bw/day assuming a typical adult breathing rate of 23 m^3/day and a body weight of 70 kg.

No specific data were identified in the literature regarding the amount of acetaldehyde that is absorbed via inhalation. It was therefore assumed that 100% of inhaled acetaldehyde is absorbed. Acetaldehyde was not assessed through the secondary pathways since most acetaldehyde will remain in the medium to which is discharged. Fugacity modelling predicts that when acetaldehyde is released into air, the distribution of mass is 97.1% in air, 2.6% in water, 0.3 % in soil and 0.0% in sediment (CEPA, 2000).

1.6 REFERENCES

AIHA (American Industrial Hygiene Association). 1989. Odour Thresholds for Chemicals with Established Occupational Health Standards. AIHA Press.

Amoore, J.E. and Hautala, E. 1983. Odour as an Aid to Chemical Safety: Odour Thresholds Compared with Threshold Limit Values and Volatilities for the 214 Industrial Chemicals in Air and Water Dilution. Journal of Applied Toxicology 3(6):272-290.

CEPA (Canadian Environmental Protection Act). 2000. Priority Substances List Assessment Report. Acetaldehyde. Government of Canada, May 2000.

IARC (International Agency for Research on Cancer). 1999. Acetaldehyde (Group 2B). Chapter 5: Summary of Data Reported and Evaluation. URL: http://www.iarc.fr/

OMOE (Ontario Ministry of the Environment and Energy). 2001. Summary of Point-of-Impingement Standards, Point-of-Impingement Guidelines, and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ministry of the Environment, Ontario, September 2001.

US EPA (United States Environmental Protection Agency). 1994. OPPT Chemical Fact Sheet. Chemicals in the Environment: Acetaldehyde. Prepared by: Office of Pollution Prevention and Toxics, Environmental Protection Agency, August, 1994.

Woutersen, R., Appelman, L., Van Garderen-Hoetmer, A., and Feron, V. 1986. Inhalation toxicity of acetaldehyde in rats. III. Carcinogenicity study. Toxicol 41:213-231.

van Gemert, L.J. 1999. Compilations of odour threshold values in air and water. TNO Nutrition and Food Research Institute, Boelens Aroma Chemical Information Service (BACIS), The Netherlands, 1999.

2.0 ACROLEIN

Synonyms: 2-propenal; 2-propen-1-one; acrylaldehyde; acroleine; acrylaldehyde; aquinte; propylene

2.1 PHYSICAL/CHEMICAL PROPERTIES

Acrolein is a colourless or yellowish liquid with a piercing, disagreeable odour that has been described as pungent and may cause tearing (AIHA, 1989). Reported odour threshold values range from approximately 50 to 4,100 $\mu g/m^3$ with a mean odour threshold of 368 $\mu g/m^3$ (van Gemert, 1999; Amoore and Hautala, 1983).

2.2 SOURCES

Acrolein is not commercially produced in Canada, but it is imported from the United States for use as an aquatic herbicide and as a microbiocide in produced water during oil explorations. It is released as a product of incomplete combustion from sources such as waste incinerators, furnaces, fireplaces, power plants, agricultural burns and the cooking of food. Forest product manufacturing also is known to release appreciable amounts of acrolein into the air. Cigarette smoking is another source of acrolein. Acrolein is believed to be a common component of foodstuffs (CEPA, 2000).

2.3 ENVIRONMENTAL FATE

Acrolein is extremely reactive in the atmosphere. It primarily reacts with photo-chemically produced hydroxyl radicals in the atmosphere to yield carbon dioxide, formaldehyde and glycoaldehyde. Less frequent reactions occur by direct phytolysis and reactions with nitrates and ozone. Its half-life in the atmosphere is from 10 to 33.7 hours, thus long-range transport is considered unlikely (Mackay *et al*., 1992). Removal of acrolein from the atmosphere may occur *via* wet deposition. In water, acrolein may undergo three key reactions: reversible hydration, biodegradation by microorganisms and volatilization. Long-range transport of acrolein in water is unlikely as the half-life of acrolein is between 30 and 100 hours. With a half-life of 14 to 56 days in groundwater, removal mechanisms for acrolein include anaerobic degradation and hydrolysis of acrolein. The primary removal mechanisms of acrolein in soil include biodegradation, hydrolysis and volatilization. Acrolein will adsorb to soil, thus modest leaching is expected. The half-life of acrolein in soil is estimated to be seven to 28 days (CEPA, 2000; ATSDR, 1990; Mackay *et al*., 1992).

2.4 SYMPTOMS OF OVEREXPOSURE

Acute (short-term) exposure to acrolein can cause eye, skin and upper respiratory tract irritation. At higher concentrations, central nervous system effects including dizziness, light-headedness, nausea, headache, diarrhoea and fainting have been observed. Very high concentrations may cause unconsciousness and death (CEPA, 2000).

Adverse health effects associated with acrolein overexposure occur primarily at the tissue of first contact (*i.e*., the respiratory and gastrointestinal tract after inhalation and ingestion, respectively) and are concentration related (CEPA, 2000). Acrolein may cause mutations and there is limited evidence that it is a teratogen in animals (New Jersey DHSS, 1998).

2.5 EXPOSURE LIMITS

The Ontario Ministry of the Environment (OMOE) one-hour point of impingement limit of 23.3 $\mu g/m^3$ was employed as the acute exposure limit for the current assessment (OMOE, 2001). This exposure limit is based on health considerations, but the specific basis was not provided.

The chronic inhalation exposure limit of 0.13 µg/kg bw/day was based on the lowest threshold concentration for inhalation exposure recommended by Health Canada of 0.4 µg/m^3 (CEPA, 2000). This exposure limit was based on the lower benchmark concentration of 0.14 mg/m^3 associated with a 5% increase in non-neoplastic lesions in the nasal respiratory epithelium of rats. A safety factor of 100 was incorporated to derive the human exposure limit accounting for interspecies variation (10) and intraspecies variation (10). The limit was further adjusted for continuous exposure (*i.e.*, rats were exposed intermittently for six hours per day so the limit was multiplied by six hours over 24 hours). The recommended threshold concentration of 0.4 µg/m^3 was converted to an exposure limit in µg/kg bw/day by assuming a typical breathing rate of 23 m^3/day and a body weight of 70 kg.

The chronic oral exposure limit of 7.5 µg/kg bw/day was based on a no-observable-adverse-effect level (NOAEL) of 0.15 mg/mL in rats exposed to acrolein *via* gavage for thirteen weeks (NTP, 1998; CEPA, 2000). At higher dose levels, effects on the gastrointestinal tract, such as hyperplasia, necrosis, inflammation and haemorrhage, were observed. A safety factor of 100 was applied by CEPA (2000) in the derivation of the human exposure limit to account for interspecies (10) and intraspecies variation (10).

In order to incorporate acrolein into the multi-media exposure model, bioavailability was assessed for the various exposure pathways (*i.e.*, inhalation, ingestion and dermal contact). No specific data were identified in the literature regarding the amount of acrolein that is absorbed via inhalation; therefore it was assumed that 100% of inhaled acrolein is absorbed. Oral bioavailability in humans was assumed to be 80% and dermal bioavailability was assumed to be 1% for this assessment (RAIS, 2004).

2.6 REFERENCES

AIHA (American Industrial Hygiene Association). 1989. Odour Thresholds for Chemicals with Established Occupational Health Standards. AIHA Press.

Amoore, J.E. and Hautala, E. 1983. Odour as an Aid to Chemical Safety: Odour Thresholds Compared with Threshold Limit Values and Volatilities for the 214 Industrial Chemicals in Air and Water Dilution. Journal of Applied Toxicology, 3(6):272-290.

ATSDR (Agency for Toxic Substances and Disease Registry). 1990. Toxicological Profile for Acrolein, December 1990. URL: http://www.atsdr.cdc.gov/toxprofiles/tp124.html

CEPA (Canadian Environmental Protection Act). 2000. Priority Substances List Assessment Report. Acrolein. Government of Canada, May 2000.

Feron, VJ; Kryusse, A; Til, HP; et al. 1978. Repeated exposure to acrolein vapour: subacute studies in hamsters, rats and rabbits. Toxicology 9:47-57. Cited In: US EPA IRIS 2004.

Mackay, D., W.C. Shiu, and K.C. Ma. 1992. Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals: Volumes I-IV. Chelsea, MI: Lewis Publishers.

New Jersey DHSS (Department of Health and Senior Services). 1998. Hazardous Substance Fact Sheet - Acrolein. URL: http://www.state.nj.us/health/eoh/rtkweb/rtkhsfs.htm

NTP (National Toxicology Program). 1998. 13-week gavage toxicity studies of allyl acetate, allyl alcohol and acrolein in Fischer 344 rats and B6C3F1 mice. October 1995. Abstract of study and Pathology Working Group Review received from Sharon Soward, National Institute of Environmental Health Sciences, U.S. Department of Health and Human Services, Research Triangle Park, North Carolina.

OMOE (Ontario Ministry of the Environment). 2001. Summary of Point of Impingement Standards, Ambient Air Quality Criteria (AAQCs) and Approvals Screening Levels (ASLs). Standards Development Branch.

RAIS (Risk Assessment Information System). 2004. URL: http://risk.lsd.ornl.gov/cgi-bin/tox/TOX_select?select=nrad

van Gemert, L.J. 1999. Compilations of odour threshold values in air and water. TNO Nutrition and Food Research Institute, Boelens Aroma Chemical Information Service (BACIS), The Netherlands, 1999.

3.0 ARSENIC

3.1 CHEMICAL/PHYSICAL PROPERTIES

Arsenic is an odourless silver-grey brittle metalloid having oxidation states of –3, 0, +3 and +5. It exhibits both metallic and non-metallic properties. Arsenic will form stable organic complexes in its trivalent and pentavalent states. For this assessment arsenic primarily exists in its inorganic oxidative forms, arsenic (III) oxide and/or arsenic (V) oxide; the former is translucent, while the latter is white in colour. Organic forms of arsenic occur when arsenic combines with carbon and hydrogen (HSDB, 2004; ATSDR, 2000).

3.2 SOURCES

Trace amounts of arsenic can be found in the earth's crust and it naturally enters the atmosphere *via* forest fires, volcanic emissions, weathering of rocks, and windblown soil particles. It is also found naturally in sulphidic ores. In Canada, anthropogenic emission sources include coal-fired power plants, industrial and domestic waste incineration, metal smelting, and chemical production and use. Currently, arsenic is most often used in metallurgical applications such as in the manufacturing of transistors, lasers and semi-conductors. Additional uses include pharmaceuticals, glass, pigments, textiles, paper, metal adhesives, wood preservatives, ammunition and explosives. Before 1975, it was used as a component of pesticides sprayed on fruits and vegetables (CEPA, 1993; HSDB, 2004; ATSDR, 2000).

3.3 ENVIRONMENTAL FATE

Arsenic is highly mobile and extremely reactive in the natural environment, with tremendous potential to cycle through abiotic and biotic components of both terrestrial and aquatic ecosystems. As arsenic cycles through these ecosystems various chemical transformations such as oxidation, reduction, methylation and demethylation may occur. Arsenic is primarily released into the atmosphere in its arsenic (III) form, which is then oxidized to its pentavalent state, though arsenic usually exists in the air as a mixture of both forms. Both arsenic forms are removed from the atmosphere *via* wet and dry deposition (CEPA, 1993).

Inorganic arsenic is soluble in water and most commonly found as pentavalent arsenic; however, aquatic organisms may reduce it to its trivalent or methylated forms. It is rarely found in its elemental form in water. Arsenic easily sorbs to soils; however, this is dependent on the pH, amount of clay, iron, aluminum, calcium and phosporous present. Leaching occurs when the arsenic capacity of the soil has been exceeded (CEPA, 1993; ATSDR, 2000).

Arsenic can bioaccumulate in the environment, biomagnification is unlikely. Bioconcentration factors (BCF) of several thousand have been reported for arsenic in aquatic organisms (CEPA, 1993; Health Canada, 1996).

3.4 SYMPTOMS OF OVEREXPOSURE

Acute (short-term) health effects after arsenic exposure include irritation to the eyes, nose and throat. Eyes may become red and watery, while skin may become itchy and a rash may develop. Higher exposures may cause poor appetite, nausea, vomiting and muscle cramps. At sufficiently high exposures, it can cause cardiac failure leading to death (New Jersey DHSS, 1998).

Chronic (long-term) health effects include, darkening of the skin, the appearance of small corns or warts on the palms, soles or torso and/or a hole in the cartilage dividing the inner nose. Nerve damage is also possible resulting in a “pins and needles” sensation in the hands and feet, and burning and weakness in the arms and legs. Repeated exposure may also damage the liver and stomach. Long-term (*i.e.*, years) ingestion of drinking water containing inorganic forms of arsenic has been found to increase the risk of skin cancer and tumours of the bladder, kidney, liver and lung (New Jersey DHSS, 1998; Health Canada, 1999).

3.5 EXPOSURE LIMITS

The 24-hour guideline of 0.3 $\mu g/m^3$ proposed by the Ontario Ministry of the Environment and Energy (OMOE) was applied as an acute exposure limit (OMOE, 2001). This exposure limit was derived based on health considerations, but the specific basis was not provided. The California Environmental Protection Agency (CalEPA) reports reproductive and developmental effects as the toxicological endpoints for acute exposure to arsenic (OEHHA, 2000).

Health Canada and the Canadian Environmental Protection Agency (CEPA) classifies arsenic as a human carcinogen *via* inhalation based on an observed increase in lung cancer mortality within multiple occupational populations exposed primarily through inhalation (Health Canada, 2003; CEPA, 1993). The Risk-specific Concentration (RsC) was developed from an inhalation unit risk of 6.4 $[mg/m^3]^{-1}$, which was derived from a tolerable concentration (TC_{05}) of 7 $\mu g/m^3$. The TC_{05} is associated with a 5% increase in respiratory cancer among smelter workers and represents the daily dose *via* inhalation that is associated with an increased cancer risk of one in 100,000. The RsC of 0.0016 $\mu g/m^3$ was converted to a Risk-specific Dose (RsD) of 0.00051 μg/kg bw/day assuming a typical adult breathing rate of 23 m^3/day and a body weight of 70 kg.

Arsenic is also classified as a human carcinogen by Health Canada for exposure *via* ingestion (Health Canada, 2003). Health Canada reports an oral slope factor of 2.8 $[mg/kg\ bw/day]^{-1}$ (HEALTH CANADA, 2003). This exposure limit was derived based on health considerations, but the specific basis was not provided. The oral slope factor was converted to a risk-specific concentration (RsC) of 0.0036 μg/kg bw/day, which represents the daily dose *via* ingestion that is associated with an increased cancer risk of one in 100,000.

In order to be run through the multi-media exposure model, the bioavailability of arsenic was assessed for the various exposure pathways (*i.e.*, inhalation, ingestion and dermal contact). No specific data were identified in the literature regarding the amount of arsenic that is absorbed via inhalation. It was therefore assumed that 100% of inhaled arsenic is absorbed. The Risk Assessment Information System (RAIS) reports an oral bioavailability in humans of 41% and dermal bioavailability of 0.1% (RAIS, 2004).

3.6 REFERENCES

ATSDR (Agency for Toxic Substances and Disease Registry). 2000. Toxicological Profile for Arsenic. URL: http://www.atsdr.cdc.gov/toxpro2.html

CEPA (Canadian Environmental Protection Act). 1993. Priority Substances List Assessment Report. Arsenic. Government of Canada.

Health Canada. 1996. Health-Based Tolerable Daily Intakes/Concentrations and Tumorigenic Doses/Concentrations for Priority Substances. Environmental Health Directorate. Health Protection Branch. Ministry of Health: Ottawa.

Health Canada. 1999. It's Your Health – Arsenic and Human Health. April 20th, 1999. URL: www.hc-sc.gc.ca/ehp/ehd/catalogue/general/iyh/arshuman.htm

Health Canada. 2003. Federal Contaminated Site Risk Assessment in Canada. Part II: Health Canada Toxicological Reference Values (TRVs). Version 1.0. October 3, 2003.

HSDB (Hazardous Substances Data Bank). 2004. Database on-line search. Arsenic. URL: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

New Jersey DHSS (Department of Health and Senior Services). 1998. Hazardous Substance Fact Sheet - Arsenic.

OMOE (Ontario Ministry of the Environment and Energy). 2001. Summary of Impingement Standards, Point of Impingement Guidelines, and Ambient air Quality Criteria (AAQCs). September 2001.

OEHHA (Office of Environmental Health Hazard Assessment). 2000. Acute Reference Exposure Levels (RELs), Averaging Times, and Toxicologic Endpoints. Arsenic and Inorganic Arsenic Compounds. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. May 2000.

RAIS (Risk Assessment Information System). 2004. URL: http://risk.lsd.ornl.gov/cgi-bin/tox/TOX_select?select=nrad

4.0 BENZENE

Synonyms: phenyl hydride; coal naphtha; benzol; cyclohexatriene; benzine; benzolene; phene; annulene; bicarburet of hydrogen; carbon oil; mineral naphtha; motor benzol; nitration benzene

4.1 PHYSICAL/CHEMICAL PROPERTIES

Benzene is a clear, colourless to light yellow liquid with an aromatic, sweet odour (AIHA, 1989). It evaporates into the air very quickly and is highly flammable (ATSDR, 1997). Reported odour threshold values for benzene range from 1,500 to 1,000,000 $\mu g/m^3$ with a mean odour threshold of 38,400 $\mu g/m^3$ (Amoore and Hautala, 1983; van Gemert, 1999).

4.2 SOURCES

Benzene enters the environment from production, storage, transport, venting and combustion of both gasoline and benzene itself (HSDB, 2004). It is used as a solvent and as an intermediate in the production of other chemicals used in resins, nylon and synthetic fibres, rubbers, lubricants, dyes, detergents, drugs and pesticides (ATSDR, 1997). In addition, benzene is indirectly produced in coke ovens, during nonferrous metal manufacture, ore mining, wood processing, coal mining and textile manufacture (IARC, 1982; Graedel, 1978). Other anthropogenic sources of benzene include oil spills, cigarette smoke and the refuelling and operation of motor vehicles. Natural sources of benzene include volcanoes and forest fires. Indoor air generally contains higher levels of benzene than outdoor air due to benzene-containing products such as glues, paints, furniture wax and detergents (ATSDR, 1997).

4.3 ENVIRONMENTAL FATE

Benzene evaporates into the air very quickly and has a half-life of approximately 2.4 to 501 hours (Mackay *et al*., 1992; HSDB, 2004). In the atmosphere, benzene is expected to exist solely as a vapour, which is broken down via reaction with photochemically-produced hydroxyl radicals. The half-life of vapour-phase benzene in polluted atmospheres which contain nitrogen oxides or sulphur dioxide is expected to be considerably less (approximately 4 to 6 hrs) (HSDB, 2004). Direct photolysis of benzene is not considered an important degradation process since benzene has a maximum absorbance frequency of 253 nm (HSDB, 2004).

Benzene is expected to volatilize from any moist surfaces and potentially dry surfaces. The volatilization half-lives for a model river and model lake are one hour and 3.5 days, respectively.

Based on its physical-chemical properties, benzene is not expected to adsorb to sediment and suspended solids in water and is likely to be highly mobile in soil (HSDB, 2004). Mackay *et al*. (1992) reports a half-life of 5 to 365 days for benzene in soil. It can be found in air, soil and water; however it is unlikely to persist in soil or water because it biodegrades and volatilizes rapidly to the atmosphere (Health Canada, 1998).

The bioconcentration factor for benzene indicates that the potential for bioconcentration in aquatic organisms is low (HSDB, 2004).

4.4 SYMPTOMS OF OVEREXPOSURE

Acute (short-term) overexposure to benzene can cause such symptoms as dizziness, light-headedness, headache, vomiting and irritation of the eyes, skin, nose or throat (US EPA, 1997). At very high exposure concentrations it can cause convulsions, coma and sudden death from irregular heartbeat. Such exposures are generally confined to the workplace and follow improper handling, storage or use, without regard to proper safety precautions.

The main concern with breathing benzene at lower concentrations for long periods (*i.e*., chronic exposure) is damage to blood cells and bone marrow, which could lead to anaemia or leukemia (New Jersey DHSS, 2001). It also can affect the immune system, increasing the chance of infection (ATSDR, 1997). Benzene has been shown to cause chromosome damage in a variety of 'test tube' and animal studies, as well as in persons occupationally exposed to benzene over long periods of time (US EPA, 1997).

Benzene has been classified by a number of government agencies (IARC, US EPA, Health Canada, NTP, *etc*.) as carcinogenic to humans based on numerous epidemiological or case studies of benzene-exposed workers. The type of leukemia most commonly associated with benzene exposure is acute myelogenous leukemia (AML) (CEPA, 1993). Other blood disorders associated with benzene exposure include preleukemia and aplastic anaemia (US EPA, 1997). There is an inconclusive, weak association in the literature between benzene exposure and chronic lymphocytic leukemia (CLL). Evidence of benzene's carcinogenic potential also can be found in animal studies, many of which have shown that exposure results in an increased risk of the development of multiple cancers (US EPA, 1997).

In the worker population for which exposure has been most extensively characterized, a clear exposure-response relationship has been demonstrated between benzene exposure and the risk of developing AML (Rinsky *et al*., 1987). In other words, the risk is proportional to the concentration to which one is exposed and the length of exposure to that concentration. A study of over 200,000 workers in the petroleum industry found no increased risk of leukemia associated with benzene exposure, even for AML (Raabe and Wong, 1996). They attributed this to the low levels of benzene exposure in the petroleum industry, particularly in comparison to benzene exposure levels in previous studies of workers in other industries (*e.g*., rubber manufacturers) where an association with AML was found.

It may be that there is a true increase in risk of leukemia at low exposure levels, but the risk is so small that very large populations would be required to detect it. Although the study of petroleum workers was large, the number of deaths on which the assessment was based was small. Nevertheless, it is reassuring that no increase in leukemia risk has been detected in workers exposed continuously to levels of benzene which, although low relative to levels associated with leukemia in some occupations, are still likely much higher than typical environmental exposures.

The mechanism by which benzene causes leukemia remains unclear, despite substantial research. It has been hypothesized that the development of leukemia as a result of benzene exposure is a 'threshold' event, but the understanding of the mechanism of benzene toxicity is not sufficient to determine whether this hypothesis is correct. If it is a 'threshold' event, it means that a certain level of benzene exposure is required to increase the risk of developing leukemia; below that exposure level no increase in risk is present. In the absence of conclusive evidence for this, benzene is by default considered to be a 'non-threshold' toxicant – a substance for which there is some probability of harm at any level of exposure. Recent evidence suggests there may be multiple steps leading to leukemia resulting from benzene exposure (US EPA, 1997).

In a Health Canada fact sheet on chemical carcinogens and health risks, the following statements are relevant:

"Under the Canadian Environmental Protection Act (CEPA), carcinogens are considered toxic to human health. In theory, there is no 'safe' level of exposure to a carcinogen. It is generally assumed, but has never been proven, that there is some chance of developing cancer at any level

of exposure. In other words, contact between a single human cell and a carcinogen molecule may be enough to cause cancer. This does not mean that cancer is the inevitable or even the likely outcome of such an event."

Human cells have developed a range of defences to guard against all sorts of injuries, including those that lead to cancer. As a result, cancer usually occurs only after prolonged and continuous exposure to carcinogens. In addition, as the exposure levels decrease, so does the risk of developing cancer.

For the average Canadian, the risk of developing cancer as a result of exposure to any particular pollutant in the environment is extremely small. People today are exposed to trace amounts of various chemicals in their air, water and food. But the levels are generally far below those levels associated with harmful effects. In the workplace, however, exposure to much higher levels of chemicals can occur (Health Canada, 1999).

4.5 EXPOSURE LIMITS

The acute exposure limit of 160 $\mu g/m^3$ was recommended for benzene by the Agency for Toxic Substances and Disease Registry (ATSDR, 2004). The Minimal Risk Level (MRL) was derived a lowest-observable-adverse-effect-level (LOAEL) of 10 ppm (32 mg/m^3) for reduced lymphocyte proliferation following mitogen stimulation (*i.e.*, immunological effects) in mice exposed *via* inhalation. The LOAEL was adjusted for intermittent exposure by multiplying the LOAEL by 6/24 and then converted to a human equivalent concentration by multiplying the ratio of a mouse ventilation rate (2.21 L/hour) to body weight (0.0316 kg) and dividing by human ventilation rate (833 L/hour) to body weight (70 kg) ratio. An uncertainty factor of 300 was then applied to the resulting $LOAEL_{(HEC)}$ of 14.7 ppm (47 mg/m^3) to account the use of a LOAEL (10), sensitive individuals (10) and for interspecies variability (3).

To be conservative in the assessment of potential risks from chronic exposure, benzene was assumed to be a non-threshold carcinogen (*i.e.*, exposure at any level increases risk, if only very slightly). Based on the Health Canada assessment of benzene, a risk specific concentration (RsC) of 3 $\mu g/m^3$ was employed as the inhalation exposure limit and an RsD of 0.032 µg/kg bw/day was employed as the oral exposure limit (Health Canada, 2003). The inhalation and oral

exposure limits were based an inhalation unit risk of 0.0033 $(\mu g/m^3)^{-1}$ and an oral slope factor of 0.31 $(\mu g/kg/\ bwday)^{-1}$, respectively. These represent the daily dose *via* either inhalation or ingestion that is associated with an increased cancer risk of 1 in 100,000. Both the RsC and the RsD were based on health considerations, however the specific basis was not provided. The RsC was converted to an exposure limit of 1 µg/kg bw/day by assuming a typical breathing rate of 23 m^3/day and a body weight of 70 kg.

In order to be run through the multi-media exposure model, the bioavailability of benzene was assessed for the various exposure pathways (*i.e.*, inhalation, ingestion and dermal contact). Inhalation absorption of benzene is assumed to be about 50% by US EPA IRIS (2004) and 60 % by Srbova (1950). However, for the purposes of this assessment the absorption of benzene following inhalation exposure was conservatively assumed to be 100%. The Risk Assessment Information System (RAIS) reports an oral bioavailability in humans of 97% and dermal bioavailability of 1% (RAIS, 2004). Oral bioavailability in humans is commonly reported to be 97% (RAIS, 2004, Park and Williams, 1953, Nomiyama and Nomiyama, 1973ab) and dermal bioavailability is most frequently reported to be 1% (RAIS, 2004, Cooper and Snyder, 1988).

4.6 REFERENCES

AIHA (American Industrial Hygiene Association). 1989. Odour Thresholds for Chemicals with Established Occupational Health Standards. AIHA Press.

Amoore, J.E. and Hautala, E. 1983. Odour as an Aid to Chemical Safety: Odour Thresholds Compared with Threshold Limit Values and Volatilities for the 214 Industrial Chemicals in Air and Water Dilution. Journal of Applied Toxicology, 3(6):272-290.

ATSDR (Agency for Toxic Substances and Disease Registry). 1997. Toxicology Profile for Benzene. September 1997. URL: http://www.atsdr.cdc.gov/toxprofiles/tp3.html

ATSDR (Agency for Toxic Substances and Disease Registry). 2004. Minimal Risk Levels (MRLs) for Hazardous Substances. January 2004.

CEPA (Canadian Environmental Protection Act). 1993. Priority Substances List Assessment Benzene. Government of Canada. Ottawa.

Cooper, K.R., and Snyder, R. 1988. Benzene Metabolism (Toxicokinetics and the Molecular Aspects of Benzene Toxicity). Cited In: Aksoy, M. (Ed.) Benzene Carcinogenicity. CRC Press, Inc., Boca Raton, FL. pp. 33-58.

Graedel, T.E. 1978. Chemical Compounds in the Atmosphere. New York, NY: Academic Press. Cited In: HSDB 2004.

Health Canada. 1998. The Health and Environment Handbook for Health Professionals. Contaminant Profiles. Minister of Public Works and Government Services Canada.

Health Canada. 1999. It's Your Health. Chemical Carcinogens: Health Risks. URL: http://www.hc-sc.gc.ca/ehp/ehd/catalogue/general/iyh

HSDB (Hazardous Substances Data Bank). 2004. Database on-line search. Benzene. URL: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

IARC (International Agency for Research on Cancer). 1982. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: Some Industrial Chemicals and Dyestuffs. Volume 29. Lyon: IARC. pp. 95-148.

Mackay, D., W.C. Shiu, and K.C. Ma. 1992. Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals: Volumes I-IV. Chelsea, MI: Lewis Publishers.

New Jersey DHSS (Department of Health and Senior Services). 2001. Hazardous Substance Fact Sheet - Benzene.

Nomiyama, K., and Nomiyama, H. 1974a. Respiratory retention, uptake and excretion of organic solvents in man. Benzene, toluene, n-hexane, trichloroethylene, acetone, ethyl acetate and ethyl alcohol Int Arch Arbeitsmed 32:85-91.

Nomiyma, K., and Nomiyama, H. 1974b. Respiratory retention, uptake and excretion of organic solvents in man. Benzene, toluene, n-hexane, trichloroethylene, acetone, ethyl acetate and ethyl alcohol Int Arch Arbeitsmed 32:85-91

Park, D.V., and Williams, R.T. 1953. Studies in detoxication. The metabolism of benzene containing 14C benzene. Biochem J 54:231.

Raabe, G.K. and Wong, O. 1996. Leukemia Mortality by Cell Type in Petroleum Workers with Potential Exposure to Benzene. Environmental Health Perspectives, 104 (6).

RAIS (Risk Assessment Information System). 2004. URL: http://risk.lsd.ornl.gov/cgi-bin/tox/TOX_select?select=nrad

Rinsky, R.A., Smith, A.B., Horning, R. et al. 1987. Benzene and leukemia: An epidemiologic risk assessment. N. Engl. J. Med. 316: 1044-1050. Cited In: US EPA IRIS 2004.

Srbova, J., Teisinger, J., and Skramovsky, S. 1950. Absorption and elimination of inhaled benzene in man. Arch Ind Hyg Occup Med 2:1-8. Cited In: ATSDR, 1993.

US EPA IRIS (United States Environmental Protection Agency Integrated Risk Information System). 2004. IRIS Database On-line search. Benzene. United States Environmental Protection Agency, Cincinnati, OH.

US EPA (United States Environmental Protection Agency). 1997. Carcinogenic Effects of Benzene: An Update. National Center for Environmental Assessment. United States Environmental Protection Agency: Washington, D.C.

van Gemert, L.J. 1999. Compilations of odour threshold values in air and water. TNO Nutrition and Food Research Institute, Boelens Aroma Chemical Information Service (BACIS), The Netherlands, 1999.

5.0 BENZO(A)PYRENE

Synonyms: 3,4-benz(a)pyrene; B(a)P; benz(a)pyrene; BP

5.1 PHYSICAL/CHEMICAL PROPERTIES

Benzo(a)pyrene (B(a)P) is a five-ringed polycyclic aromatic hydrocarbon (PAH) with a faint aromatic odour (HSDB, 2004). An odour threshold for B(a)P was not available.

5.2 SOURCES

There is no commercial production or known use for B(a)P. However, it occurs ubiquitously as a product of incomplete combustion. For example, whenever gasoline, garbage or any animal/plant material burns, B(a)P is usually found in the smoke and soot (ATSDR, 1995). It has been identified in cigarette smoke, gasoline engine exhaust, diesel engine exhaust, various crude, fresh and used motor oils; gasoline; charcoal-broiled steaks; various processed foods; various oils, margarine, butter and fats; fruits, vegetables and cereals; roasted coffee; and tea (HSDB, 2004). B(a)P occurs in fossil fuels and is a product of incomplete combustion associated with volcanoes and forest fires (HSDB, 2004). There also is some evidence for biosynthesis by plants, bacteria and algae (HSDB, 2004).

5.3 ENVIRONMENTAL FATE

Based on the low vapour pressure for B(a)P, it typically exists in the particulate phase in the atmosphere. Removal occurs *via* wet and dry deposition. Based on Koc values ranging from 930 to 6,300, there will be low or no mobility in the soil. Volatilization from the soil or surface water is low based on low vapour pressure and Henry's Law values. Biodegradation of B(a)P in soil is slow due to adsorption. The half-life in soil ranges from 120 to 309 days. If released into water B(a)P will adsorb to suspended solids and sediments (HSDB, 2004).

Bioconcentration of B(a)P is expected to be low to high depending on whether an organism has metabolic detoxification enzyme mechanisms (HSDB, 2004).

5.4 SYMPTOMS OF OVEREXPOSURE

Acute (short-term) overexposure to B(a)P can cause skin irritation with rash and/or burning sensations and can irritate and/or burn the eyes on contact (New Jersey DHSS, 1998). Exposure to sunlight and the chemical together can increase these effects.

Chronic or repeated exposure also may cause skin changes and adverse effects on foetal development and the male reproductive system (New Jersey DHSS, 1998). Critical effects of long-term exposure include lung cancer and genitourinary tract cancer in humans (OMOE,

1997). B(a)P has been classified as a probable carcinogen in humans, primarily on the basis of animal studies and genetic toxicity testing (US EPA IRIS, 2004).

5.5 EXPOSURE LIMITS

The only regulatory agency that has recommended a public acute exposure limit for B(a)P is the Ontario Ministry of Environment and Energy (OMOE). OMOE (2001) has recommended a 24-hour air quality guideline for B(a)P of 0.0011 $\mu g/m^3$. This limit is based on the carcinogenic potential for B(a)P, but it was derived based on an annual exposure limit of 0.00022 $\mu g/m^3$ for protection against cancer using a simple extrapolation factor that was considered to be overly conservative. This limit was not employed in the current assessment because it did not account for the influence of duration of exposure on the carcinogenic action of a chemical.

As recommended in OMOE (1997), the assessment of carcinogenic PAHs can employ two approaches (i) the Whole Mixture Model (WMM) and ii) the Individual PAH Model (IPM). The WMM approach is based on the conservative assumption that the potency of the PAH fraction of any environmental mixture is proportional to the B(a)P content of the mixture (OMOE, 1997). The WMM was derived from the methodology of the OMOE (1997), using the concentration of B(a)P together with the toxic potency of the PAH-WMM group. The cancer slope factor of B(a)P was estimated by OMOE, based on an examination of the composition and toxic potency of PAH mixtures derived from many different sources (*e.g.*, coal tar, coke oven emissions, diesel emissions, wood burning, *etc.*). The unit risk was derived based on a weight-of-evidence review of numerous epidemiology and rodent toxicity studies of B(a)P. Critical effects included lung cancer and genitourinary tract cancer in humans. This approach, used in conjunction with the Individual PAH Model, ensures that potential risks are not underestimated in the current assessment.

The Individual PAH Model predicts risk of the PAH fraction based on the sum of the attributable risks for each individual PAH. The first step in the IPM requires the estimation of the inhalation potency of B[a]P and of other PAHs relative to B[a]P. This step involves the use of Toxic Equivalency Factors (TEFs) to denote the cancer potency of specific PAH compounds relative to the potency of B[a]P (Bostrom *et al.*, 2002). TEFs allow large groups of compounds with a

common mechanism of action such as PAHs to be assessed when there are limited data available for all but one of the compounds (*i.e.*, B[a]P). The TEFs employed in the current assessment of PAHs *via* the IPM approach are presented in Table 5-1.

Table 5-1: Relative Potency of Individual PAHs Compared with B[a]P (adapted from Larsen and Larsen, 1998)

COMPOUND [(1)]	TOXIC EQUIVALENCY FACTOR (TEF)
Acenaphthene	NA
Acenaphthylene	NA
Benz(a)anthracene	0.005
Benzo(a)pyrene	1
Benzo(b)fluoranthene	0.1
Benzo(k)fluoranthene	0.05
Benzo(g,h,i)perylene	0.01
Chrysene	0.03
Dibenz(a,h)anthracene	1.1
Fluoranthene	0.05
Fluorene	0.0005
Indeno(1,2,3-cd)pyrene	0.1
Naphthalene	NA
Phenanthrene	0.0005
Pyrene	0.001

1) Acenaphthene, acenaphthylene, fluoranthene, fluorene, phenanthrene, pyrene and naphthalene were also assessed separately in accordance with the Total Petroleum Hydrocarbon (TPH) approach.

Secondly, the levels of individual PAHs in air must be determined and the risk attributable to each of the PAHs at the environmental levels must be assessed. The final step in the model is the summation of the risks attributable to each individual PAH.

For the chronic assessment, B(a)P was evaluated along with all other carcinogenic polyaromatic hydrocarbons. The exposure limits used in this assessment are summarized in Table 5-2 and are recommended by OMOE (1997) and WHO (1999).

Table 5-2: Summary of Chronic Exposure Limits for Human Receptors Exposed to Benzo(a)pyrene

CHEMICAL	ROUTE	EXPOSURE LIMIT (µg/kg bw/day)		ENDPOINT	REGULATORY AGENCY
		TYPE	VALUE (1)		
Benzo(a)pyrene (WMM)	Inhalation	RsD	0.00014 (2)	Lung tumour	OMOE, 1997
	Oral	RsD	0.0034	Stomach tumour	OMOE, 1997
	Dermal	RsD	0.000071	Skin tumour	OMOE, 1997
Benzo(a)pyrene (IPM)	Inhalation	RsD	0.000038 (3)	Lung tumour	WHO, 1999
	Oral	RsD	0.055	Stomach tumour	OMOE, 1997
	Dermal	RsD	0.0012	Skin tumour	OMOE, 1997

1) The exposure limits are associated with an acceptable incremental lifetime cancer risk of 1 in 100,000.
2) The inhalation RsD of 0.00014 µg/kg bw/day is equivalent to 0.00043 µg/m^3, assuming the average adult has an inhalation volume of 23 m^3/day and weights 70 kg.
3) The inhalation RsD of 0.000038 µg/kg bw/day is equivalent to 0.00012 µg/m^3, assuming the average adult has an inhalation volume of 23 m^3/day and weights 70 kg.

In order to be run through the multi-media exposure model, the bioavailability of B(a)P was assessed for the various exposure pathways (*i.e.*, inhalation, ingestion and dermal contact). The bioavailability values used for the human assessment are presented in Table 5-3.

Table 5-3: Bioavailability Values for Benzo(a)pyrene

COMPOUND	ROUTE OF EXPOSURE	ABSORPTION FACTOR	REFERENCE
Benzo(a)pyrene	Inhalation	100%	Assumed
	Oral	31%	RAIS, 2004
	Dermal	1%	RAIS, 2004

5.6 REFERENCES

ATSDR (Agency for Toxic Substances and Disease Registry). 1995. Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAH), August 1995. URL: http://www.atsdr.cdc.gov/toxprofiles/tp124.html

Bostrom, C., Gerde, P., Hanberg, H., Jerstrom, B., Johansson, C., Kyrklund, T., Rannug, A., Tornqvist, M., Victorin, K., and Westerholm, R. 2002. Cancer Risk Assessment, Indicators, and Guidelines for Polycyclic Aromatic Hydrocarbons in the Ambient Air. Environ Health Perspect 110(Suppl 3):451-488.

HSDB (Hazardous Substances Data Bank). 2004. Database on-line search. Benzo(a)pyrene. URL: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

Larsen, J.C. and Larsen, P.B. 1998. Chemical carcinogens: In: Air Poluution and Health (Hester, R.E. and Harrison, R.M. eds). Cambridge, UK: The Royal Society of Chemistry, 33-56. Cited In: Bostrom *et al*., 2002.

New Jersey DHSS (Department of Health and Senior Services). 1998. Hazardous Substance Fact Sheet - Benzo(a)pyrene.

OMOE (Ontario Ministry of the Environment and Energy). 1997. Scientific Criteria Document for Multimedia Standards Development - Polycyclic Aromatic Hydrocarbons (PAH). Part 1: Hazard Identification and Dose-response Assessment. Ontario Ministry of Environment and Energy, Standards Development Branch. February 1997.

OMOE (Ontario Ministry of the Environment and Energy). 2001. Summary of Point-of-Impingement Standards, Point-of-Impingement Guidelines, and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ministry of the Environment, Ontario, September 2001.

RAIS (Risk Assessment Information System). 2004. URL: http://risk.lsd.ornl.gov/cgi-bin/tox/TOX_select?select=nrad

US EPA IRIS (United States Environmental Protection Agency Integrated Risk Information System). 2004. IRIS Database On-line search. Benzo(a)pyrene. United States Environmental Protection Agency, Cincinnati, OH.

WHO (World Health Organization). 1999. Air Quality Guidelines, WHO, 1999. Chapter 3. Health-based Guidelines.

6.0 1,3-BUTADIENE

Synonyms: butadiene; alpha-butadiene alpha-gamma-butadiene; buta-1,3-diene; bivinyl; divinyl; erythrene; vinylethylene; biethylene and pyrrolylene.

6.1 PHYSICAL/CHEMICAL PROPERTIES

1,3-Butadiene is a colourless gas with a slightly aromatic odour (ATSDR, 1993). It has a reported odour threshold range of 2200-3520 $\mu g/m^3$ based on odour recognition (Amoore and Hautula, 1983). Based on odour detection, 1,3-butadiene has a reported odour threshold of 55 $\mu g/m^3$ (Verschueren, 1983).

6.2 SOURCES

1,3-Butadiene is produced during the combustion of organic matter and thus it is ubiquitous in the urban environment because of widespread combustion sources (CEPA, 2000). It is also used in the chemical polymer industry to make synthetic rubber products such as tires, food wrap, car sealants, and adhesives (New Jersey DHSS, 1998). Combustion of biomass (*e.g.* forest fires) is a natural source of 1,3-butadiene (CEPA, 2000).

6.3 ENVIRONMENTAL FATE

1,3-Butadiene is a highly volatile gas that will primarily partition to the atmosphere (ATSDR, 1993). In the atmosphere, 1,3-butadiene is expected to undergo rapid degradation via photo-initiated reactions that produce hydroxyl radicals. The reported half-life of 1,3-butadiene in the atmosphere is 6 hours. 1,3-Butadiene is also broken down via nightly gas-phase reactions with ozone (ATSDR, 1993).

In soil and water, 1,3-butadiene is expected to rapidly volatise to the atmosphere, based on it physical and chemical properties. 1,3-Butadiene is not expected to adsorb to soil or sediment and biodegradation is not anticipated (ATSDR, 1993).

6.4 SYMPTOMS OF OVEREXPOSURE

Acute (short-term) overexposure to 1,3-butadiene can cause central nervous system effects including blurred vision, nausea, fatigue, headache, decreased blood pressure and pulse rate and unconsciousness or death (ATSDR, 1993). It can also irritate the eyes, nose, mouth and throat causing coughing and wheezing (New Jersey DHSS, 1998).

Chronic inhalation studies have shown 1,3-butadiene to induce tumours at multiple sites in mice and rats and occupational exposure has been associated with the induction of leukemia (CEPA, 2000). There is some limited evidence that it is genotoxic in the exposed workers. Given the weight of epidemiological and toxicological data, CEPA (2000) considered 1,3-butadiene likely to be carcinogenic in humans. IARC has classified it as probably carcinogenic to humans.

Studies on workers exposed to 1,3-butadiene over long periods have shown an increase in heart and lung damage, but because these workers were also exposed to a number of other chemicals definite conclusions regarding 1,3-butadiene cannot be made (ATSDR, 1993). Animal studies show that breathing 1,3-butadiene during pregnancy can increase the number of birth defects (ATSDR, 1993).

6.5 EXPOSURE LIMITS

No acute health-based exposure limits have been recommended for 1,3-butadiene as an ambient air quality guideline. Consequently, a one-hour exposure limit was derived from the Emergency Response Planning Guidelines recommended by the American Industrial Hygiene Association (AIHA). An ERPG-1 of 10 ppm (22,000 $\mu g/m^3$) was recommended by AIHA (2001). ERPG-1 guidelines represent the maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to one hour without experiencing other than mild transient adverse health effects or perceiving a clearly defined, objectionable odour (AIHA, 2001). The ERPG guidelines were developed to protect most individuals in the population, but not hypersensitive types (AIHA, 2001). Accordingly, a ten-fold safety factor was applied to the ERPG-1 for 1,3-butadiene in the derivation of an acute exposure limit for the current assessment (*i.e.,* 2,200 $\mu g/m^3$).

Health Canada classifies 1,3-butadiene as a human carcinogen *via* inhalation based on an observed increase in leukemia in both epidemiological studies and investigations in experimental animals (CEPA, 2000). The Risk-specific Concentration (RsC) was developed from a tumorigenic concentration (TC_{01}) of 0.1 mg/m^3, which is associated with a 1% increase in leukemia in humans. The RsC of 0.1 $\mu g/m^3$ represents the daily dose *via* inhalation that is associated with an increased cancer risk of one in 100,000. The RsC was converted to a Risk-specific Dose (RsD) of 0.033 µg/kg bw/day assuming a typical adult breathing rate of 23 m^3/day and a body weight of 70 kg.

As a result of limited oral toxicity data for 1,3-butadiene, the RsD of 0.033 µg/kg bw/day was employed as the oral exposure limit in the current assessment of 1,3-butadiene.

In order to be run through the multi-media exposure model, the bioavailability of 1,3-butadiene was assessed for the various exposure pathways (*i.e.*, inhalation, ingestion and dermal contact). No specific data were identified in the literature regarding the amount of 1,3-butadiene that is absorbed via inhalation. It was therefore assumed that 100% of inhaled 1,3-butadiene is absorbed. The Risk Assessment Information System (RAIS) reports an oral bioavailability in humans of 80% and dermal bioavailability of 1% (RAIS, 2004).

6.6 REFERENCES

ATSDR (Agency for Toxic Substances and Disease Registry. 1993. Toxicological profile for 1,3-butadiene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. URL: http://www.atsdr.cdc.gov/toxprofiles/tp28.html

AIHA (American Industrial Hygiene Association). 2001. Emergency Response Planning Guidelines. AIHA Press.

Amoore, J.E. and Hautula, E. 1983. Odor as an Aid to Chemical Safety: Odor Thresholds Compared with Threshold Limit Values and Volatilities for the 214 Industrial Chemicals in Air and Water Dilution. Journal of Applied Toxicology, 3(6):272-290.

CEPA (Canadian Environmental Protection Act). 2000. Priority Substances List Assessment 1,3-Butadiene. Government of Canada. Ottawa.

New Jersey DHSS (Department of Health and Senior Services). 1998. Hazardous Substance Fact Sheet – 1,3-butadiene.

RAIS (Risk Assessment Information System). 2004. URL: http://risk.lsd.ornl.gov/cgi-bin/tox/TOX_select?select=nrad

Verschueren, K. 1983. Handbook of environmental data on organic chemicals. 2nd ed. Van Nostrand Reinhold Co., New York, N.Y.

7.0 CADMIUM

7.1 PHYSICAL/CHEMICAL PROPERTIES

Cadmium is an odourless, silver-blue lustrous metal, which is insoluble in water. Cadmium in nature usually exists in the plus II oxidation state which includes freely soluble cadmium chloride and cadmium nitrate. In the current assessment, cadmium is primarily found combined with oxygen (cadmium oxide) (ATSDR, 1999).

7.2 SOURCES

Trace amounts of cadmium can be found in the earth's crust and naturally enters the atmosphere via forest fires, volcanic emissions, weathering of rocks, and windblown soil particles. In Canada, anthropogenic emission sources include base metal smelting and refining operations. Cadmium is a by-product of zinc refining, but may also be recovered from industrial scrap or recycled cadmium products. It is used in nickel-cadmium batteries, coatings and pigments, stabilizers in plastics and synthetic products, and in alloys. Small amounts of cadmium may be found in television picture tubes, telephone and trolley wires, automobile radiators, control rods and nuclear reactor shields, motor oils and curing agents for rubbers (CEPA, 1994; Health Canada, 1996).

7.3 ENVIRONMENTAL FATE

In air, cadmium exists as particulate matter and will generally be deposited close to its source. Wet and dry deposition is the primary removal mechanisms of cadmium from the atmosphere. Cadmium oxide deposited on soil will largely be adsorbed on to the soil surface layers, particularly under conditions of high pH and high organic soil content. Elevated soil acidity increases the availability of cadmium to plants and increases the likelihood of leaching (HSDB, 2004; CEPA, 1994).

Cadmium will bioconcentrate in organisms, with bioconcentration factors (BCF) reported to range from 1,000 to 17,600. It can build up in the body after many years of exposure (CEPA, 1994).

7.4 SYMPTOMS OF OVEREXPOSURE

Acute (short-term) overexposure to cadmium can cause flu-like symptoms (*i.e.*, chills, headache, aching and/or fever), nausea, salivation, vomiting, abdominal cramps and diarrhea. Pulmonary irritation (*i.e.*, shortness of breath and coughing), pulmonary edema and shortness of breath may result from cadmium inhalation at sufficient exposures (New Jersey DHSS, 1999).

Chronic (long-term) overexposure can cause pulmonary irritation and bronchitis. Kidney damage, kidney stones, anemia, loss of smell, fatigue and yellow coloured teeth may result after repeated exposures to sufficient concentrations. Kidney damage can occur due to the build-up of cadmium in the kidneys. An increased risk of lung cancer has been associated with chronic cadmium oxide dust and fume exposure (New Jersey DHSS, 1999).

7.5 EXPOSURE LIMIT

The Ontario Ministry of the Environment and Energy (OMOE) 24-hour guideline of 2 $\mu g/m^3$ was applied in this assessment as an acute exposure limit for cadmium (OMOE, 2001). This exposure limit was derived based on health considerations, but the specific basis was not provided.

The chronic inhalation exposure limit of 0.001 $\mu g/m^3$ was developed by Health Canada (2003). The risk-specific concentration (RsC) was calculated using an inhalation unit risk of 9.8 $[mg/m^3]^{-1}$. The inhalation unit risk was based on lung tumour incidences in rats at 2.9 $\mu g/m^3$, which was amortized to be constant over the lifetime of the rat, adjusted for the longer than standard lifetime duration of the experiment (*i.e.*, 130 weeks), and converted to an equivalent human concentration. An acceptable risk level of one in 100,000 was assumed in calculating the RsC, which was converted to a dose of 0.00034 µg/kg bw/day assuming the average adult weighs 70 kg and breaths 23 m^3/day.

The chronic oral exposure limit employed in the current assessment was developed by Health Canada as a tolerable daily intake of 0.8 μg/kg bw/day (Health Canada, 2003). This limit was derived from the Canadian Guideline for Drinking Water Quality reported by Health Canada (2002), which is based on health considerations but the specific basis was not provided.

In order to be run through the multi-media exposure model, the bioavailability of cadmium was assessed for the various exposure pathways (*i.e.*, inhalation, ingestion and dermal contact). No specific data were identified in the literature regarding the amount of cadmium that is absorbed via inhalation. It was therefore assumed that 100% of inhaled cadmium is absorbed. The Risk Assessment Information System (RAIS) reports an oral (food or drink) bioavailability in humans of 1% and dermal bioavailability of 1% (RAIS, 2004). No data were available on inhalation bioavailability, thus it was assumed to be 100%.

7.6 REFERENCES

ATSDR (Agency for Toxic Substances and Disease Registry). 1999. Toxicological Profile for Cadmium.

ATSDR (Agency for Toxic Substances and Disease Registry). 2004. Minimal Risk Levels (MRLs) for Hazardous Substances. January 2004.

CEPA (Canadian Environmental Protection Act). 1994. Priority Substances List Assessment Report. Cadmium. Government of Canada.

Health Canada. 2002. Guidelines for Canadian Drinking Water Quality – Supporting Documentation. Ottawa, ON. http://www.hc-sc.gc.ca/hecs-sesc/water/dwgsup.htm

Health Canada. 2003. Federal Contaminated Site Risk Assessment in Canada. Part II: Health Canada Toxicological Reference Values (TRVs). Version 1.0. October 3, 2003.

HSDB (Hazardous Substances Data Bank). 2004. Issue 01-1. Cadmium. Canadian Centre for Occupational Health and Safety.

OMOE (Ontario Ministry of the Environment and Energy). 2001. Ambient Air Quality Criteria (AAQCs). Standards Development Branch. Ontario Ministry of the Environment and Energy (OMEE).

New Jersey DHSS (Department of Health and Senior Services). 1999. Hazardous Substance Fact Sheet - Cadmium.

RAIS (Risk Assessment Information System). 2004. URL: http://risk.lsd.ornl.gov/cgi-bin/tox/TOX_select?select=nrad

Thun, M.J., Schnorr, T.M., Smith, A.B., Halperin, W.E., and Lemen, R.A. 1985. Mortality among a cohort of U.S. cadmium production workers - An update. JNCI 74:325-333.

US EPA IRIS. (United States Environmental Protection Agency's Integrated Risk Information System). 2004. IRIS Database On-line search. U.S. Environmental Protection Agency, Cincinnati, OH.

8.0 CARBON MONOXIDE

Synonyms: carbonic oxide; monoxide; carbon oxide

8.1 CHEMICAL/PHYSICAL PROPERTIES

Carbon monoxide is a colourless, odourless gas (HSDB, 2004). A mean odour threshold of 116,000,000 $\mu g/m^3$ has been reported for carbon monoxide (Amoore and Hautala, 1983).

8.2 SOURCES

Carbon monoxide is formed as a product of incomplete combustion, but it is produced primarily through natural sources: volcanic, marsh and natural gases, oceans, fires and electrical storms. The major manufactured source in Canada is from fuel combustion in vehicles and stationary engines, but other important sources include tobacco smoke, burning fuel to generate electricity or heat, wood or gas stoves, kerosene lamps and waste disposal. High levels of carbon monoxide in the home can be caused by "back drafting" of a furnace or fireplace, when gases from the flue

or chimney are drawn back into the home due to poor ventilation. The greatest exposure to carbon monoxide for most people occurs in the vehicle and indoor microenvironments associated with combustion sources (Health Canada, 1998; US EPA, 1991).

8.3 ENVIRONMENTAL FATE

Carbon monoxide is non-persistent and dissipates with air currents. Carbon monoxide in the body is either from inhalation or it has been endogenously produced through the breakdown of haemoglobin and other heme-containing pigments. The only route of exposure is inhalation (Health Canada, 1998).

8.4 SYMPTOMS OF OVEREXPOSURE

Acute (short-term) overexposure to carbon monoxide can cause headache, dizziness, light-headedness and fainting, as well as adverse effects on the cardiovascular system (New Jersey DHSS, 1998; Health and Welfare Canada, 1990). Extremely high exposure levels can decrease the ability of the blood to carry oxygen (New Jersey DHSS, 1998). Groups that may be more sensitive to carbon monoxide exposure include those with cardiovascular, cerebrovascular and peripheral vascular diseases, foetuses, the newborn, pregnant women and individuals living at high altitude (Health and Welfare Canada, 1990).

Chronic (long-term) health effects of exposure to low concentrations have not been well studied in humans. High concentrations, usually indoors, can result in headache, drowsiness, cardiac arrhythmias, and in sufficient levels coma and death (Health Canada, 1998). Chronic exposure studies of laboratory animals have revealed physiological and behavioural changes such as impairment in time discrimination and consistent trace metal loss (Health Canada, 1998).

The toxicity of carbon monoxide is caused by its ability to compete with oxygen in combining with the heme component of red blood cells to form carboxyhemoglobin, which reduces the capacity of the red blood cells to carry oxygen to body tissues (Health Canada, 1998). The heart, brain and exercising skeletal muscle are the most sensitive to the effects of carbon monoxide because they are the tissues with the greatest oxygen demand.

Cardio-respiratory effects in people with exercise-induced myocardial ischemia are the health effects of greatest concern with carbon monoxide exposure. CEPA/FPAC has identified studies showing a relationship between low carbon monoxide levels and mortality from exercise-induced arrhythmia in patients with coronary artery disease (CEPA/FPAC, 1994).

8.5 EXPOSURE LIMITS

The one-hour and eight-hour acute exposure limits used for the assessment of carbon monoxide concentrations in air are 14,300 $\mu g/m^3$ and 5,500 $\mu g/m^3$ and are based on B.C. Level A air quality objectives and standards (BC MWLAP, 1995). These objectives were developed to protect the subpopulation sensitive to cardio respiratory effects and thus are expected to provide an adequate margin of safety for the general population with respect to neurobehavioral and developmental toxicity effects (CEPA/FPAC, 1994).

No chronic exposure limits for carbon monoxide have been recommended by regulatory agencies. The critical effect of carbon monoxide exposure is considered to be the resulting carboxyhemoglobin (COHb) concentration in the blood. Because COHb concentrations reach a steady-state after six to eight hours of exposure, CO exposure for longer periods of time (*i.e.*, chronic exposure) is not expected to cause accumulation of COHb in the blood (WHO, 2000).

It might be noted that epidemiological studies have identified associations between ambient low-level CO concentrations and various health effects (Burnett *et al.*, 2000; Moolgavkar, 2000). However, the results across studies are inconsistent and it has been suggested that CO may represent only a surrogate compound for particulate emissions from mobile sources (Sarnat *et al.*, 2001; Schwartz, 1999).

8.6 REFERENCES

Amoore, J.E. and Hautala, E. 1983. Odour as an Aid to Chemical Safety: Odour Thresholds Compared with Threshold Limit Values and Volatilities for the 214 Industrial Chemicals in Air and Water Dilution. Journal of Applied Toxicology, 3(6):272-290.

BC MWLAP (British Columbia Ministry of Water, Land and Air Protection). 1995. Air Quality Objectives and Standards. URL: http://wlapwww.gov.bc.ca/air/airquality/airqual_1.pdf

Burnett, R.T., Brok, J., Dann, T., Delocla, C., Philips. O., Cakmak, S., Vincent, R., Goldberg, M.S., Krewski, D. 2000. Association between particulate and gas-phase components of urban air pollution and daily mortality in eight Canadian cities. Inhalation Toxicology. 12(suppl. 4):15-39.

CEPA/FPAC Working Group on Air Quality Objectives and Guidelines. 1994. National Ambient Air Quality Objectives for Carbon Monoxide. Desirable, Acceptable and Tolerable Levels. Canadian Environmental Protection Act: Ottawa.

Health and Welfare Canada. 1990. Exposure Guidelines for Residential Indoor Air Quality. A Report of the Federal-Provincial Advisory Committee on Environmental and Occupational Health.

Health Canada. 1998. The Health and Environment Handbook for Health Professionals. Minister of Public Works and Government Services Canada.

HSDB (Hazardous Substances Data Bank). 2004. Database on-line search. Carbon monoxide. URL: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

Moolgavkar, S.H. 2000. Air pollution and daily mortality in three US counties. Environmental Health Perspectives. 108(8): 777-784.

New Jersey DHSS (Department of Health and Senior Services). 1998. Hazardous Substance Fact Sheet - Carbon monoxide.

Sarnat, J.A., Schwartz, J., Catalano, P.J., Suh, H.H. 2001. Gaseous pollutants in particulate matter epidemiology: confounders or surrogates. Environmental Health Perspectives. 109(10):1053-1061

Schwartz, J. 1999. Air pollution and hospital admissions for heart disease in eight US cities. Epidemiology. 11:11-17.

US EPA (United States Environmental Protection Agency). 1991. Guidance for Risk Assessment. Environmental Protection Agency, US (EPA). Risk Assessment Council. Washington, DC.

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe. World Health Organization: Copenhagen. URL: http://www.who.int/peh/air/Airqualityg

9.0 CHROMIUM

9.1 CHEMICAL/PHYSICAL PROPERTIES

Chromium compounds have no taste or odour and exist primarily in three forms, chromium (0), chromium (III) and chromium (VI). Chromium (III) compounds (trivalent chromium) are stable and occur naturally in the environment. Metallic chromium (chromium (0)) does not occur naturally and chromium (VI) occurs only rarely (ATSDR, 2000).

9.2 SOURCES

Trace amounts of chromium, as chromium (III) can be found in the earth's crust where it naturally enters the atmosphere *via* forest fires, volcanic emissions, weathering of rocks, and windblown soil particles. In central Canada, wind blown prairie dust is thought to be a major source of chromium. Currently Canada does not mine chromium and consequently it must import chromium ores, approximately 74,000 tonnes in 1991. Anthropogenic sources of chromium (*i.e.*, Chromium (III) and (IV)) include combustion of natural gas, oil and coal, waste incineration, steel and chemical manufacturing and cement plant emissions. In Canada, chromium containing chemicals are primarily used in pigments, metal finishing, leather tanning, wood preservatives, textiles, drilling muds, toner for copy machines and water treatment (CEPA, 1994; HSDB, 2004).

9.3 ENVIRONMENTAL FATE

Chromium is released to the atmosphere primarily as a particulate. Little is known about speciation of chromium in the air, however, the predominant form released from the burning of

fossil fuels is chromium III. Based on air data collected in California, chromium (VI) has been reported to comprise 3 to 7% of ambient air. Chromium particles settle from air in less than 10 days via wet and dry deposition. They stick strongly to soil particles. In water, most chromium sticks to dirt particles that fall to the bottom; only a small amount dissolves.

Chromium (VI) can be removed from soils by uptake into plants and animals, leaching, and adsorption. However, the majority is converted to chromium (III), which is immobile in soil. Hexavalent chromium in soil is expected to be reduced to trivalent chromium by organic matter. Consequently, exposure to chromium in food is mostly trivalent (ATSDR, 2000; CEPA, 1994; HSDB, 2004; US EPA, 1998).

9.4 SYMPTOMS OF OVEREXPOSURE

At high enough exposure levels, all forms of chromium are toxic but chromium VI is more toxic than chromium III. Acute (short-term) overexposure to chromium VI can damage and irritate the nose, lungs, stomach and intestines. Breathing high levels of either chromium VI or chromium III may cause asthma attacks in sensitive individuals. An individual may become sensitized to chromium (VI) whereby future exposures may result in skin rashes, asthma-attacks or shortness of breath. Contact with the skin may cause burns and/or ulcers. Ingestion of large amounts of chromium can cause stomach upsets and ulcers, convulsions, kidney and liver damage and death (ATSDR, 2000; New Jersey DHSS, 1998).

Chronic (long-term) overexposure to chromium VI can cause damage to the nose (bleeding, itching, sores) and lungs, and can increase the risk of non-cancer lung disease and lung cancer. Based on increased lung cancer in some workers who were exposed to chromium, chromium VI has been classified as a known human carcinogen (ATSDR, 2000; New Jersey DHSS, 1998).

9.5 EXPOSURE LIMITS

The Ontario Ministry of the Environment and Energy (OMOE) 24-hour guideline of 1.5 $\mu g/m^3$ was employed in this assessment as an acute exposure limit for chromium (III) and Chromium (VI) (OMOE, 2001). This limit was based on health considerations but the specific basis was not provided.

The chronic inhalation exposure limit for chromium (III) was based on a chronic feeding study of rats (US EPA IRIS, 2004). The reference dose (RfD) of 1,500 μg/kg bw/day was derived from a no observable adverse effect level (NOAEL) of 1,800 g/kg bw average total dose of 5% Cr_2O_3 in diet for 5days/week and 600 feedings. The NOAEL was adjusted by the United States Environmental Protection Agency (US EPA) for continuous exposure to a dose of 1,468 mg/kg bw/day. In addition, the US EPA applied an uncertainty factor of 100 to the adjusted NOAEL to account for interspecies (10) and intraspecies (10) variability and an additional 10-fold modifying factor was applied to reflect database deficiencies including the lack of a study in a non-rodent mammal, lack of unequivocal data evaluating reproductive impacts, and the concern regarding potential reproductive effects raised by the study of Elbetieha and Al-Hamood (1997). The resulting RfD was converted to the inhalation exposure limit of 4,600 μg/m^3 assuming an average adult breathing rate of 23 m^3/day and average body weight of 70 kg.

The chronic oral exposure limit of 1,500 μg/kg bw/day for chromium (III) that was employed in the current assessment is discussed above in the derivation of the inhalation exposure limit (US EPA IRIS, 2004).

For chromium (VI), the chronic inhalation exposure limit of 0.00013 μg/m^3 was reported by Health Canada (2003). The risk-specific concentration (RsC) was calculated using an inhalation unit risk of 75.8 [mg/m^3]$^{-1}$, which was derived from a tolerable concentration (TC_{05}) of 0.66 μg/m^3 for hexavalent chromium based on mortality due to lung cancer in a cohort of 332 men employed at a chromate production plant between 1931 and 1937. The TC_{05} is associated with a 5% increase in lung cancer among plant workers. An acceptable risk level of one in 100,000 was assumed in calculating the RsC, which was converted to a dose of 0.000043 μg/kg bw/day assuming the average adult weighs 70 kg and breaths 23 m^3/day.

The chronic oral exposure limit for chromium (VI) that was employed in the current assessment was developed by Health Canada as a tolerable daily intake of 1 μg/kg bw/day (Health Canada, 2003). This limit was derived from the Canadian Guideline for Drinking Water Quality reported by Health Canada (2002), which is based on health considerations but the specific basis was not provided.

In order to be run through the multi-media exposure model, the bioavailability of chromium was assessed for the various exposure pathways (*i.e.*, inhalation, ingestion and dermal contact). No specific data were identified in the literature regarding the amount of chromium that is absorbed via inhalation. It was therefore assumed that 100% of inhaled chromium (III) and chromium (VI) is absorbed. The Risk Assessment Information System (RAIS) reports an oral bioavailability in humans of 0.5% and dermal bioavailability of 0.1% for chromium (III) (RAIS, 2004). For chromium (VI), RAIS reports an oral bioavailability in humans of 2% and dermal bioavailability of 0.1%, regardless of whether the chromium (VI) is present as a chromic acid mist or a particulate (RAIS, 2004).

9.6 REFERENCES

ATSDR (Agency for Toxic Substances and Disease Registry). 2000. Toxicological Profile for Chromium. September 2000. URL: www.atsdr.cdc.gov/tfacts7.html

CEPA (Canadian Environmental Protection Act). 1994. Priority Substances List Assessment Report. Chromium VI. Government of Canada.

Health Canada. 2002. Guidelines for Canadian Drinking Water Quality – Supporting Documentation. Ottawa, ON. http://www.hc-sc.gc.ca/hecs-sesc/water/dwgsup.htm

Health Canada. 2003. Federal Contaminated Site Risk Assessment in Canada. Part II: Health Canada Toxicological Reference Values (TRVs). Version 1.0. October 3, 2003.

HSDB (Hazardous Substances Data Bank). 2004. Issue 01-1. Chromium. Canadian Centre for Occupational Health and Safety.

New Jersey DHSS (Department of Health and Senior Services). 1998. Hazardous Substance Fact Sheet – Chromium.

OMOE (Ontario Ministry of the Environment and Energy). 2001. Summary of point impingement standards, point of impingement guidelines, and ambient air quality criteria (AAQCs). Standards Development Branch. Ontario Ministry of the Environment.

RAIS (Risk Assessment Information System). 2004. URL: http://risk.lsd.ornl.gov/cgi-bin/tox/TOX_select?select=nrad

US EPA (United States Environmental Protection Agency). 1998. Toxicological Review of Hexavalent Chromium. In support of summary information on the Integrated Risk Information System (IRIS). August, 1998.

US EPA IRIS. (United States Environmental Protection Agency's Integrated Risk Information System). 2004. IRIS Database On-line search. U.S. Environmental Protection Agency, Cincinnati, OH.

Zhang, J; Li, X. 1987. Chromium pollution of soil and water in Jinzhou. J of Chinese Preventive Med 21:262-264. Cited In: US EPA IRIS 2004.

10.0 DIESEL PARTICULATE MATTER

10.1 CHEMICAL/PHYSICAL PROPERTIES

Diesel exhaust is a complex mixture containing gases, vapours and fine particulates. The gaseous components include carbon dioxide, oxygen, nitrogen, water vapour; however, diesel exhaust particles are thought by most researchers to contribute the majority of the risk from diesel exhaust exposure (OEHHA, 1997). Diesel exhaust particles may carry harmful organics and metal in the exhaust. Diesel particles typically have a mass-medium aerodynamic diameter of approximately 0.1 to 0.25 μm, where more than 75% of the particles are smaller than 1 μm (OEHHA, 1997). Diesel particles have a large surface area that attracts compounds of low volatility to the inside of the particle or to the particle surface. Organic compounds that are commonly associated with diesel particles include aliphatic hydrocarbons, polycyclic aromatic hydrocarbons (PAHs), and PAH-derivatives (OEHHA, 1997)

10.2 SOURCES

Sources of diesel exhaust particulate matter include mobile sources (*i.e.*, on-road vehicles and off-road mobile sources), stationary area sources and stationary point sources. Stationary area

sources include shipyards, warehouses, heavy equipment repair yards, and oil and gas production operations (OEHHA, 1997). Stationary point sources include heavy construction with the exception of highway construction, electrical services, and crude petroleum and natural gas extraction (OEHHA, 1997).

10.3 ENVIRONMENTAL FATE

Diesel exhaust particles may be transported over long distances due to their non-reactive behaviour in the atmosphere. Diesel exhaust particles are expected to have a low overall rate of removal from the atmosphere, resulting in an atmospheric lifetime of several days. Fall-out rates may increase as particles coagulate over time (WHO, 1999). Physical removal of diesel exhaust particulate matter typically occurs via accretion of the particles, atmospheric fall-out (*i.e.,* dry deposition), and atmospheric removal by rain (*i.e.,* wet deposition) (OEHHA, 1997). The elemental carbon of the particulate matter can act as a catalyst in the formation of sulphuric acid by oxidizing sulphuric dioxide. The organic portions adsorbed to the elemental carbon may undergo a number of physical and chemical reactions with other atmospheric compounds and during exposure to sunlight (WHO, 1999)

10.4 SYMPTOMS OF OVEREXPOSURE

Short-term exposure to diesel exhaust particulates may result in irritation of the ocular and nasal mucous membranes and an increased frequency in respiratory symptoms.

Long-term non-cancer health effects are considered to be the result of the presence of particles in the exhaust. Airborne respirable particulate matter has recently been associated with increased morbidity and mortality. Observed health effects may include respiratory symptoms, changes in lung function, and increased hospitalization for respiratory and cardiovascular disease (OEHHA, 1999).

Sufficient information is not currently available to determine whether diesel exhaust exposure induces reproductive, developmental or teratogenic effects in humans.

Statistically significant increase in the incidence of lung cancer associated with diesel exhaust exposure has been documented in epidemiological studies of truck drivers, transport and equipment workers, dock workers, and railway workers (OEHHA, 1999). The International Agency for Research on Cancer (IARC) indicates that there is sufficient evidence that whole diesel engine exhaust probably causes cancer in humans and classifies diesel exhaust as a probable human carcinogen (IARC, 1989).

10.5 EXPOSURE LIMITS

No acute exposure limit for diesel PM has been reported. The chronic exposure limit for diesel PM was developed the United States Environmental Protection Agency (US EPA) based on pulmonary inflammation and histopathology in a rat chronic inhalation study (US EPA IRIS, 2004). A no observable adverse effect level of 0.46 mg/m^3 was converted to a human equivalent concentration (HEC) of 0.144 mg/m^3 using a mathematical model of diesel PM deposition and clearance. The US EPA applied and uncertainty factor of 30 to the HEC to account for residual interspecies (*i.e.*, rat to human) extrapolation uncertainties (3) and to account for inter-individual human variation in sensitivity (10). The result is a reference concentration (RfC) of 5 $\mu g/m^3$, which was employed as the chronic exposure limit for the current assessment of diesel PM in air.

10.6 REFERENCES

OEHHA (Office of Environmental Health Hazard Assessment). 1997. Diesel Exhaust. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Toxic Air Contaminant Identification, Chronic Reference Exposure Levels. September 1997.

US EPA IRIS (Integrated Risk Information System). 2004. IRIS Database On-line search. Formaldehyde. United States Environmental Protection Agency, Cincinnati, OH.

11.0 FLUORENE

Synonyms: 9H-Fluorene; o-Biphenylenemethane; diphenylenemethane; 2,2'-methylenebiphenyl; o-biphenylmethane; 2,3-benzindene; alpha-diphenylenemethane-9H-fluorene

11.1 PHYSICAL/CHEMICAL PROPERTIES

Fluorene is a white solid that is fluorescent when impure. It is insoluble in water. An odour threshold for fluorene was not available (HSDB, 2004).

11.2 SOURCES

Fluorene is a natural component of fossil fuels and coal tar. It is a product of incomplete combustion and may be found in mainstream cigarette smoke, gasoline exhaust and sewage sludge. Other fluorene emission sources include coal, polymer, duff (humus on forest floors) and biomass combustion, asphalt paving and municipal waste incinerators (HSDB, 2004; Irwin e*t al*., 1998).

11.3 ENVIRONMENTAL FATE

In the atmosphere, fluorene exists in the vapour phase. The primary removal mechanism of fluorene from air is by photo-oxidation, where hydroxyl radicals oxidize fluorene. The estimated half-life in air is 29 hours. Wet deposition and dry deposition are the primary removal mechanisms for particulate phase fluorene. Due to the stability of particulate phase fluorene, long-range transport is likely. Under aerobic conditions fluorene is readily biodegraded in soil or water by microbes, and has been reported to strongly adsorb to soil and water sediments. The half-life of fluorene in soil is two to 64 days. Volatilization of non-adsorbed fluorene from water is likely with an estimated half-life of 15 to 167 hours (HSDB, 2004; Irwin *et al*., 1998).

Bioconcentration factors of 2.62 to 3.67 have been reported in fluorenes with alkyl-groups attached. Alkyl-groups increase lipophilicity thereby increasing absorption. Fluorenes with no

alkyl groups, as is being assessed in the present risk assessment, will be less likely to bioaccumulate (Irwin *et al*., 1998).

11.4 SYMPTOMS OF OVEREXPOSURE

Acute (short-term) overexposure to fluorene may irritate and possibly burn the eyes and skin (New Jersey DHSS, 1999).

Fluorene has been shown to cause a variety of subchronic effects in mice when administered 250 or 500 mg fluorene/kg body weight/day by gavage for 13 weeks including increased hypo activity, labored respiration, increased liver weight, decreased blood cell count, decreased blood urea nitrogen and increased total serum bilirubin (US EPA, 1989). No significant effects were observed at 125 mg/kg body weight/day.

The limited animal carcinogenicity data for fluorene suggest that this compound is non-carcinogenic. No increase in tumor incidence was observed in rats treated orally with fluorene (Wilson *et al*., 1947, Morris *et al*., 1960), or mice treated by skin painting (Kennaway, 1924; Riegel *et al*., 1951) or subcutaneous injection (Shear, 1938) with fluorene.

No data were available concerning human overexposure to fluorene in the literature reviewed for the current exposure assessment.

11.5 EXPOSURE LIMITS

An acute exposure limit for fluorene of 1,217 $\mu g/m^3$ was derived based on the Minimal Risk Level (MRL) recommended for intermediate exposure to fluorene by the Agency for Toxic Substances and Disease Control (ATSDR, 2004). The MRL was derived from a minimal lowest observable adverse effect level (LOAEL) of 125 mg/kg bw/day based on increases in relative liver weight in mice exposed *via* gavage for 90 days (ATSDR, 1995). The ATSDR applied an uncertainty factor of 300 to the LOAEL to account for the use of a LOAEL instead of a no observable adverse effect level (NOAEL; 3), for extrapolation from animals to humans (10) and for human variability (10). The resulting MRL of 400 µg/kg bw/day was converted to an air concentration of 1,217 $\mu g/m^3$, assuming an average person breathes 23 m^3/day and weighs 70 kg.

This exposure limit is considered to be conservative since it is recommended for intermediate exposure and presumably a higher exposure over a shorter time-period (*i.e.*, acute exposure) could occur without risk of adverse effects.

The chronic exposure limit was based on the US EPA IRIS (2004) assessment of fluorene, which derived an oral reference dose (RfD) of 40 µg/kg bw/day. The US EPA RfD was based on a subchronic mouse study demonstrating an absence of adverse effects on the blood at a dose of 125 mg/kg bw/day. The US EPA applied an uncertainty factor of 3000 to account for use of a subchronic study for chronic RfD derivation (10), interspecies variability (10), intraspecies variability (10), and for lack of adequate toxicity data in a second species and reproductive/developmental data (3).

An inhalation limit for fluorene was unavailable, so the oral limit was employed for the assessment of inhalation exposures, with adjustments for chemical bioavailability (equivalent to 25.3 µg/kg bw/day or 77 µg/m^3). Based on airborne particle dynamics in the respiratory system (Brain and Mosier, 1980; Potts and Oberdorster, 1983) about 49.7 to 79.1% of airborne fluorene on particles would be bioavailable through the lung. Oral bioavailability in humans was assumed to be 50% and dermal bioavailability was assumed to be 1% for this assessment (RAIS, 2004).

11.6 REFERENCES

ATSDR (Agency for Toxic Substances and Disease Registry). 1995. Toxicological Profile for PAHs. August 1995. URL: www.atsdr.cdc.gov/tfacts7.html

ATSDR (Agency for Toxic Substances and Disease Registry). 2004. Minimal Risk Levels for Hazardous Substances (MRLs). U.S. Public Health Service. Agency for Toxic Substances and Disease Registry (ATSDR).

Brain, D.B., and Mosier, M.J. 1980. Deposition and Clearance of Grain Dusts in the Human Lung. In: Dossman, J.A., and Cotton, D.J. (Eds.) Occupational Pulmonary Disease: Focus on Grain Dust and Health. pp. 77-94.

HSDB (Hazardous Substances Data Bank). 2004. Fluorene. Database on-line search.

Irwin, R.J., M. VanMouwerik, L. Stevens, M.D. Seese, and W. Basham. 1998. Environmental Contaminants Encyclopaedia. National Park Service, Water Resources Division, Fort Collins, Colorado. Distributed on the Internet via the Nature Net portion of the Park Service Home Page (www.nps.gov).

Kennaway, E.L. 1924. On cancer-producing tars and tar-fractions. J Ind Hyg 5:462-90. Cited In: US EPA IRIS 2004.

Morris, H.P., Velat, C.A., Wagner, B.P., Dahlgard, M., and Ray, F.E. 1960. Studies of carcinogenicity in the rat of derivatives of aromatic amines related to N-2-fluorenylacetamide. JNCI 24:149-80. Cited In: US EPA IRIS 2004.

New Jersey DHSS (Department of Health and Senior Services). 1999. Hazardous Substance Fact Sheet - Fluorene.

Potts, F., and Oberdorster, G. 1983. Intake and Distribution of PAH. In: Environmental Carcinogens: Polycyclic Aromatic Hydrocarbons. Grimmer, G. (Ed.). CRC Press, pp. 130-136.

RAIS (Risk Assessment Information System). 2004. URL: http://risk.lsd.ornl.gov/cgi-bin/tox/TOX_select?select=nrad

Riegel, G., Warman, W.B., Hill, W.T., Reeb, B.B., Shubik, P., and Stranger, D.W. 1951. Delay of methylcholanthrene skin carcinogensis in mice by 1,2,5,6-dibenzofluorene. Cancer Res 11:301-6. Cited In: US EPA IRIS 2004.

Shear, M.J. 1938. Studies in carcinogensis. V. Methyl derivatives of 1,2- benzanthracene. Am J Cancer 33:499-537. Cited In: US EPA IRIS 2004.

US EPA IRIS (United States Environmental Protection Agency's Integrated Risk Information System). 2004. IRIS Database On-line search. U.S. Environmental Protection Agency, Cincinnati, OH.

US EPA (United States Environmental Protection Agency). 1989. Mouse Oral Subchronic Toxicity Study. Prepared by Toxicity Research Laboratories, Ltd., Muskegon, MI for the Office of Solid Waste, Washington, DC. Cited In: US EPA IRIS 2004.

Wilson, R.H., DeEds, F., and Cox, A.J. 1947. The carcinogenic activity of 2-acetominofluorene. IV. Action of related compounds. Cancer Res 7:453-458. Cited In: US EPA IRIS 2004.

12.0 FORMALDEHYDE

Synonyms: formalin; formic aldehyde; formol; HCHO; methanol; methyl aldehyde; methylene glycol; methylene oxide; morbicid; oxomethane; oxymethylene; Veracur

12.1 PHYSICAL/CHEMICAL PROPERTIES

Formaldehyde is a colourless liquid or gas with a pungent odour (AIHA, 1989). Its odour threshold ranges from 33 to 12,000,000 $\mu g/m^3$ with a mean odour threshold of 1,024 $\mu g/m^3$ (van Gemert, 1999; Amoore and Hautala, 1983).

12.2 SOURCES

Formaldehyde is produced industrially in many applications; in medical applications as a sterilant and as a preservative in consumer products such as food, cosmetics and household cleaning agents (WHO, 1989). It is formed by the oxidation of hydrocarbons and a considerable amount comes from the exhaust emissions of motor vehicles. Minor natural sources include the decomposition of plant residues and the transformation of various chemicals emitted by foliage (WHO, 1989).

12.3 ENVIRONMENTAL FATE

Formaldehyde is released into the environment primarily through combustion processes. It may be released directly into the atmosphere or it may be formed indirectly through the photochemical oxidation of hydrocarbons and/or other formaldehyde precursors by sunlight.

Removal of formaldehyde occurs by direct photolysis and/or oxidation by photo-chemically produced hydroxyl radicals. As a result, it has a short half-life in the troposphere (*i.e.*, few hours). Mackay *et al*. (1992) reported a half-life of 1.26 to 120 hours in air. Dry deposition, wet removal or dissolving in surface water also contributes to formaldehyde removal from the air (ATSDR, 1999).

Formaldehyde's half-life in soil has been reported as one to seven days (Howard, 1989). Formaldehyde has an organic carbon/water partition coefficient (Koc) less than 100, suggesting that it would be mobile in soil (CEPA, 2001). Dry deposition and wet removal have a half-life of 19 and 50 hours, respectively. Because it is highly soluble in water, transfer from rain to surface water occurs easily; however, it is expected to biodegrade in a few days (HSDB, 2004).

12.4 SYMPTOMS OF OVEREXPOSURE

Formaldehyde is a respiratory, skin and eye irritant at sufficient concentrations and there are large individual differences in sensitivity to its irritant potential (NTP, 1991; WHO, 1989). The available human evidence indicates that formaldehyde does not have any adverse effects on reproduction or development (WHO, 1989). Critical effects of chronic exposure include nasal lesions (ATSDR, 1999; CEPA, 2001).

The US EPA IRIS (2004) considers formaldehyde a probable human carcinogen based on evidence in humans and sufficient evidence in animals. Nine studies of the effects of formaldehyde on humans have demonstrated a statistically significant association between exposure to formaldehyde and site-specific respiratory neoplasms. Chronic (long-term) studies in mice and rats have shown an increase in incidence of nasal squamous cell carcinomas. In addition, in vitro genotoxicity data shows that formaldehyde can cause gene mutation, single strand DNA breaks, DNA-protein cross links, sister chromatid exchanges and chromosomal aberrations. The classification of formaldehyde as a possible carcinogen is further supported by formaldehyde's structural similarity to other carcinogenic aldehydes such as acetaldehyde (US EPA IRIS, 2004).

12.5 EXPOSURE LIMITS

The acute exposure limit for formaldehyde of 49.3 µg/m^3 is recommended by the Agency for Toxic Substances and Disease Registry and was employed in the current assessment (ATSDR, 2004). This acute inhalation exposure limit was developed based on respiratory system effects in volunteers exposed for 2 hours. A safety factor of 9 was incorporated by the ATSDR (1999) to account for the use of a minimal LOAEL (3) and to account for intraspecies variability (3).

The United States Environmental Agency (US EPA IRIS, 2004) classifies formaldehyde as a probable human carcinogen. The chronic exposure limit was based on the assessment of formaldehyde by CEPA (2001), in which a TC_{05} (tumorigenic concentration) or risk specific concentration (RsC) of 1.9 µg/m^3 was calculated. This TC_{05} represents the total intake associated with a 5% increase in incidence of nasal squamous tumours in rats exposed to formaldehyde. The TC_{05} correspond to an increased cancer risk of one in 100,000. The RsC of was converted to an exposure limit of 0.62 µg/kg bw/day by assuming a typical breathing rate of 23 m^3/day and a body weight of 70 kg.

No specific data were identified in the literature regarding the amount of formaldehyde that is absorbed via inhalation. It was therefore assumed that 100% of inhaled formaldehyde is absorbed. Formaldehyde was not assessed through the secondary pathways since most formaldehyde will remain in the medium to which is discharged (CEPA, 2001).

12.6 REFERENCES

AIHA (American Industrial Hygiene Association). 1989. Odour Thresholds for Chemicals with Established Occupational Health Standards. AIHA Press.

AENV (Alberta Environment). 2000. Alberta Ambient Air Quality Guideline. Alberta Environment Home Page. URL: http://www.gov.ab.ca/env/air/

Amoore, J.E. and Hautala, E. 1983. Odour as an Aid to Chemical Safety: Odour Thresholds Compared with Threshold Limit Values and Volatilities for the 214 Industrial Chemicals in Air and Water Dilution. Journal of Applied Toxicology, 3(6):272-290.

ATSDR (Agency for Toxic Substances and Disease Registry). 1999. Toxicological Profile for Formaldehyde. July 1999.

ATSDR (Agency for Toxic Substances and Disease Registry). 2004. Minimal Risk Levels (MRLs) for Hazardous Substances. January 2004.

CEPA (Canadian Environmental Protection Act). 2001. Priority Substances List Assessment Report. Formaldehyde. Government of Canada. February 2001.

Egle, J.L. 1972. Retention of inhaled formaldehyde, propionaldehyde and acrolein in the dog. Arch Environ Health 25:119-124.

Howard, P.H. 1989. Formaldehyde. In: Howard, P.H., ed. Handbook of Environmental Fate and Exposure Data for Organic Chemicals. Chelsea, MI: Lewis Publishers. pp. 342-350. Cited In: HSDB 2004.

HSDB (Hazardous Substances Data Bank). 2004. Database on-line search. Formaldehyde. URL: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

Mackay, D., W.C. Shiu, and K.C. Ma. 1992. Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals: Volumes I-IV. Chelsea, MI: Lewis Publishers.

NTP (National Toxicology Program). 1991. NTP Chemical Repository. Formaldehyde. Radian Corporation, August 29.

RAIS (Risk Assessment Information System). 2004. URL: http://risk.lsd.ornl.gov/cgi-bin/tox/TOX_select?select=nrad

US EPA IRIS (Integrated Risk Information System). 2004. IRIS Database On-line search. Formaldehyde. United States Environmental Protection Agency, Cincinnati, OH.

van Gemert, L.J. 1999. Compilations of odour threshold values in air and water. TNO Nutrition and Food Research Institute, Boelens Aroma Chemical Information Service (BACIS), The Netherlands, 1999.

WHO (World Health Organization). 1989. Environmental Health Criteria 89. Formaldehyde. World Health Organization: Geneva.

13.0 MANGANESE

13.1 CHEMICAL/PHYSICAL PROPERTIES

Manganese is a grey-white brittle metal. Elemental manganese is not found in nature, rather it is found in one of its seven-oxidation states in conjunction with various sulfides, oxides, carbonates, silicates, phosphates and borates. No data was available in regards to odour threshold (HSDB, 2004; ATSDR, 2000).

13.2 SOURCES

Manganese compounds make up 0.10% of the earth's crust. Manganese naturally occurs in pyrolusite (manganese dioxide), rhodochrosite (manganese carbonate) and rhodanate (manganese silicate) ores. Processes such as weathering of rocks and minerals naturally release manganese into the environment. Manganese compounds are commonly used in dry-cell batteries, matches, fireworks, porcelain and glass- bonding materials, amethyst glass, glazes and varnishes, in ceramics, fertilizers, as a fungicide, nutritional supplement, a disinfectant, an anti-algal agent, and for metal cleaning (ACGIH, 1997; ATSDR, 2000).

13.3 ENVIRONMENTAL FATE

Manganese in the atmosphere is most often found in the particulate form, and can thereby be removed *via* dry and to a lesser degree, wet deposition. Residence time in the atmosphere is dependent on the size of the particulate; the atmospheric half-life is approximately 2 days. Transport of manganese in water is dependent on the solubility of the manganese form present. Manganese may adsorb to sediments and soils depending on the organic concentration and cation exchange capacity of the sediments and soils (ATSDR, 2000).

Manganese may bioaccumulate in organisms, particularly at lower trophic levels. Bioconcentration factors for marine plants and marine mammals have been reported to be 10,000 to 20,000 and 100 to 600, respectively (ATSDR, 2000).

13.4 SYMPTOMS OF OVEREXPOSURE

Short-term (acute) overexposure to barium may cause nose and throat irritation resulting in coughing and shortness of breath. Flu-like symptoms often referred to as "metal fume fever" may result from high levels of manganese exposure (HSDB, 2004; New Jersey DHSS, 1999).

Long-term (chronic) overexposure to manganese affects mainly the central nervous system. Symptoms reported include weakness, lethargy, speech disturbances, twitching, tremors, poor muscle coordination, muscle cramps and loss of facial expression. Lung irritation may also occur and can be characterized by bronchitis and shortness of breath. Repeated high exposures to manganese may cause permanent lung damage and may damage the liver (HSDB, 2004; New Jersey DHSS, 1999).

13.5 EXPOSURE LIMITS

The 24-hour reference concentration (RfC) for manganese of 2.5 $\mu g/m^3$ proposed by the Ontario Ministry of the Environment and Energy (OMOE) was applied as an acute exposure limit (OMOE, 2001). This exposure limit was derived based on health considerations, but the specific basis was not provided.

The Reference Concentration (RfC) employed in the current assessment of manganese in air was based on the most sensitive and significant effects (*i.e.*, neurological deficits) caused by inhalation exposure to manganese dusts in the air. The neurological effects progressively increase in injury severity with prolonged exposures. The RfC was derived from an occupational study with manganese dioxide (MnO_2) exposed workers that experienced impairment of neuro-behavioural function at air concentrations ranging from 0.040 to 4.433 mg/m^3 x years (respirable dust), with a geometric mean of 0.793 mg/m^3 x years (US EPA IRIS, 2004). The US EPA derived a lowest observable adverse effect level (LOAEL) of 0.15 mg/m^3 by dividing the geometric mean by the average duration of exposure (5.3 years). The LOAEL was adjusted for

discontinuous exposure. An uncertainty factor of 1000 was applied to the adjusted LOAEL to account for intraspecies variability (10), for use of a LOAEL (10), and for database limitations reflecting both the less-than-chronic periods of exposure and the lack of developmental data, as well as potential but unquantified differences in the toxicity of different forms of Mn (10). The result is a reference concentration (RfC) of 0.05 $\mu g/m^3$, which is equivalent to a dose of 0.016 µg/kg bw/day assuming the average adult inhales 23 m^3 air/day and weights 70 kg (US EPA IRIS, 2004).

The current Reference Dose (RfD) of 140 µg/kg bw/day reported by the United States Environmental Protection Agency was used in this assessment as the chronic oral exposure limit for manganese (US EPA IRIS, 2004). The RfD was based on a chronic human ingestion study demonstrating an absence of effects on the central nervous system at a dose of 0.14 mg/kg bw/day. The US EPA applied no uncertainty factors to the no observable adverse effect level (NOAEL). The US EPA (1984) reported that, although some evidence exists that manganese has carcinogenic activity in laboratory animals, these studies are considered inadequate by current standards. Furthermore, no epidemiological evidence relating manganese exposure to cancer has been reported in humans.

In order to be run through the multi-media exposure model, the bioavailability of manganese was assessed for the various exposure pathways (*i.e.*, inhalation, ingestion and dermal contact). No specific data were identified in the literature regarding the amount of manganese that is absorbed via inhalation. It was therefore assumed that 100% of inhaled manganese is absorbed. The Risk Assessment Information System (RAIS) reports an oral bioavailability in humans of 4% and dermal bioavailability of 0.1% (RAIS, 2004).

13.6 REFERENCES

ACGIH. (American Conference of Governmental Hygienists). 1997. Documentation of the Threshold Limit Values (TLVs) and Biological Exposure Indices (BEIs). Sixth edition. American Conference of Governmental Industrial Hygienists.

ATSDR (Agency for Toxic Substances and Disease Registry). 2000. Toxicological Profile for Manganese. September 2000.

ATSDR (Agency for Toxic Substances and Disease Registry). 2004. Minimal Risk Levels (MRLs) for Hazardous Substances. January 2004.

HSDB (Hazardous Substances Data Bank). 2004. Issue 01-1. Manganese. Canadian Centre for Occupational Health and Safety.

OMOE (Ontario Ministry of the Environment and Energy). 2001. Summary of point impingement standards, point of impingement guidelines, and ambient air quality criteria (AAQCs). Standards Development Branch. Ontario Ministry of the Environment.

New Jersey DHSS (Department of Health and Senior Services). 1999. Hazardous Substance Fact Sheet – Manganese.

RAIS (Risk Assessment Information System). 2004. URL: http://risk.lsd.ornl.gov/cgi-bin/tox/TOX_select?select=nrad

US EPA (United States Environmental Protection Agency). 1984. Health Effects Assessment for Iron (and Compounds). U.S. Environmental Protection Agency. Cincinnati, OH. NTIS PB86-1346547.

US EPA IRIS (United States Environmental Protection Agency's Integrated Risk Information System). 2004. IRIS Database On-line search. (U.S.) Environmental Protection Agency, Cincinnati, OH.

14.0 NAPHTHALENE

Synonyms: albocarbon; dezodorator; mothballs; tar camphor; white tar

14.1 PHYSICAL/CHEMICAL PROPERTIES

Naphthalene is a two-ringed white crystalline flake or solid with a strong odour like mothballs (New Jersey DHSS, 2003). Reported odour thresholds range from 0.007 to 5.34 mg/m^3 with an average threshold of 1.34 mg/m^3 (van Gemert, 1999).

14.2 SOURCES

Naphthalene is a component of crude oil and is a natural combustion product of forest fires (HSDB, 2004). It also has been identified in cigarette smoke, condensate and motor vehicle emissions (HSDB, 2004). Naphthalene has been used in fungicides, cutting fluids, synthetic tanning, preservatives, textile chemicals, emulsion breakers, scintillation counters, raw materials and as an intermediate in the plastics industry, air refreshers and the manufacture of lacquers and varnishes (NTP, 1991). Emissions from its production in petroleum refining and coal tar distillation, in addition to oil spills and leaking underground petroleum storage tanks also represent sources of exposure to naphthalene (HSDB, 2004).

14.3 ENVIRONMENTAL FATE

Naphthalene primarily reacts with photochemically produced hydroxyl radicals in the atmosphere, with a half-life of three to eight hours. Absorption to particulates in the atmosphere followed by wet and dry deposition is an important removal mechanism. Volatilization is another removal mechanism from soil and surface water, though in aquatic systems it may partition out to organic particles and sediment where it can be more easily biodegraded; half-lives in soil and water may range from a few days to a couple months (HSDB, 2004; ATSDR, 1995).

Bioconcentration is thought to be moderate, however, in most cases naphthalene can be easily removed from an organism through depuration or metabolic detoxification enzyme mechanisms. Bioconcentration factors (BCFs) reported range from 1.6 to 3.0 (HSDB, 2004; ATSDR, 1995).

14.4 SYMPTOMS OF OVEREXPOSURE

Acute (short-term) overexposure to naphthalene can cause irritation of the skin, eyes, nose and throat (HSDB, 2004). Very high concentrations can cause headache, fatigue, confusion, nausea and vomiting (New Jersey DHSS, 2003).

Chronic (long-term) overexposure to naphthalene may cause clouding of the eye lens (cataracts), skin allergies, liver and kidney damage and anemia (low blood count) (New Jersey DHSS, 2003).

14.5 EXPOSURE LIMITS

OMOE (2001) air quality guidelines are generally developed for 24-hour and half-hour averaging periods, with the half-hour exposure limit typically three-fold higher than the 24-hour exposure limit. Moreover, the half exposure limit is typically 1.25-fold higher than the one-hour exposure limit. Consequently, a one-hour RfC of 28.8 $\mu g/m^3$ was derived by dividing the half-hour exposure limit (36 $\mu g/m^3$) by 1.25. This exposure limit was based on odour and health considerations, but the specific basis was not provided.

The US EPA inhalation reference dose for naphthalene of 1 µg/kg bw/day (3 $\mu g/m^3$) is based on nasal effects and was used as the chronic exposure limit (US EPA IRIS, 2004). This RfD chronic inhalation exposure limit was estimated from a chronic inhalation mouse study that reported the lowest observed effect on nasal epithelium at a dose of 9.3 mg/m^3 (NTP, 1991). US EPA incorporated a safety factor of 3,000 to account for interspecies differences (10), sensitive human individuals in the population (10), to extrapolate from a NOAEL to a LOAEL (10) and for database uncertainties (3) to derive its inhalation exposure limit.

The US EPA oral reference dose for naphthalene of 20 µg/kg bw/day was used as a chronic exposure limit and was based on decreased body weights in rats (US EPA IRIS, 2004). This RfD was estimated from a subchronic study in rats that reported no effect on body weight at a dose of 100 mg/kg bw/day (BCL, 1980). The NOAEL was initially adjusted to account for continuous exposure (i.e., multiplied by the study's five days of exposure per week), resulting in an adjusted NOAEL of 71 mg/kg bw/day. The US EPA incorporated a safety factor of 3,000 to account for interspecies differences (10), sensitive human individuals in the population (10), to extrapolate from a sub-chronic to a chronic study (10) and for database uncertainties (3) to derive its oral exposure limit.

Based on airborne particle dynamics in the respiratory system (Brain and Mosier, 1980; Potts and Oberdorster, 1983) about 50 to 80% of airborne naphthalene on particles would be bioavailable through the lung. Oral bioavailability in humans was assumed to be 80% and dermal bioavailability was assumed to be 1% for this assessment (RAIS, 2004).

14.6 REFERENCES

ATSDR (Agency for Toxic Substances and Disease Registry). 1995. Toxicological Profile for Naphthalene. December 1995. URL: http://www.atsdr.cdc.gov/toxprofiles/tp67.html

BCL (Battelle's Columbus Laboratories). 1980. Unpublished subchronic toxicity study: Naphthalene (C52904), Fischer 344 rats. Prepared by Battelle Laboratories under NTP Subcontract No. 76-34-106002. Cited in US EPA IRIS 2003.

Brain, D.B., and Mosier, M.J. 1980. Deposition and Clearance of Grain Dusts in the Human Lung. In: Dossman, J.A., and Cotton, D.J. (Eds.) Occupational Pulmonary Disease: Focus on Grain Dust and Health. pp. 77-94.

HSDB (Hazardous Substances Data Bank). 2004. Naphthalene. Database on-line search.

New Jersey DHSS (Department of Health and Senior Services). 2003. Hazardous Substance Fact Sheet - Naphthalene.

NTP (National Toxicology Program). 1991. Final report on the developmental toxicity of naphthalene (CAS no. 91-20-3) in Sprague Dawley (CD) rats. #TER91006. NTIS Technical Report (NTIS/PB92-135623). Cited in US EPA IRIS 2003.

OMOE (Ontario Ministry of the Environment and Energy). 2001. Summary of Point of Impingement Standards, Ambient Air Quality Criteria (AAQCs) and Approvals Screening Levels (ASLs). Standards Development Branch.

Potts, F., and Oberdorster, G. 1983. Intake and Distribution of PAH. In: Environmental Carcinogens: Polycyclic Aromatic Hydrocarbons. Grimmer, G. (Ed.). CRC Press, pp. 130-136.

RAIS. 2004. Risk Assessment Information System. URL: http://risk.lsd.ornl.gov/cgi-bin/tox/TOX_select?select=nrad

van Gemert, L.J. 1999. Compilations of odour threshold values in air and water. TNO Nutrition and Food Research Institute, Boelens Aroma Chemical Information Service (BACIS), The Netherlands, 1999.

US EPA IRIS (Integrated Risk Information System). 2004. IRIS Database On-line search. (U.S.) Environmental Protection Agency, Cincinnati, OH.

15.0 NICKEL

15.1 PHYSICAL/CHEMICAL PROPERTIES

Nickel is a hard lustrous grey metal, with no odour. In nature it is usually found in the +2 oxidation state, but can also be found in the 1, 0, +2, +3, and +4 oxidation states. As a naturally occurring element, it exists mainly in the form of sulphide ores found underground (ATSDR, 2003a; OMOE, 2001a).

15.2 SOURCES

Nickel is one of the five most abundant elements on the earth. Because it is found in the earth's crust and in trace amounts in many minerals, it is a natural component of the soil. Weathering of rocks, soil dust, sea salt, volcanoes and forest fires are common natural sources of nickel in the environment. All foods grown in Canada and almost all water supplies contain small amounts of nickel.

Primary base metal production is the main source of anthropogenic emissions of nickel in Canada. Canada is rich in sulfide ores containing nickel and as a result, it is the world's second highest producer of nickel (OMOE, 2001a). Other sources include fossil fuel combustion, alloy production, scrap reprocessing, incineration, cement manufacturing, coke ovens, asbestos mining/milling and cooling towers. Nickel alloys are used to make coins and jewelry. Nickel compounds can also be used in batteries, colour ceramics, nickel plating and used as catalysts in chemical reactions (OMOE, 2001a; CEPA, 1994; ATSDR, 2003a).

15.3 ENVIRONMENTAL FATE

Data is limited on the chemistry of nickel in the atmosphere. Nickel from anthropogenic sources is released mostly in the form of particulate matter (< $PM_{2.5}$). Larger particulates (*i.e.*, PM_{10}) primarily come from natural sources. Nickel particles in the air settle out in the soil via wet or

dry deposition. Nickel tends to be found in soil and sediments because it attaches to particles that contain iron or manganese, which are often present in soil and sediments. It does not have a strong tendency to collect in fish, plants or animals used for foods (ATSDR, 2003b).

15.4 SYMPTOMS OF OVEREXPOSURE

Acute (short-term) overexposure to nickel can cause eye, nose and skin irritation. Throat irritation may also occur, causing coughing and wheezing (New Jersey DHSS, 1998).

Chronic (long-term) overexposure may cause a hole to develop in the septum of the nose resulting in loss of smell. Nickel has also been known to cause skin allergies and asthma-like attacks. An individual may become sensitized to nickel whereby future exposures may result in skin rashes, asthma-attacks or shortness of breath. Some individuals are particularly sensitive to nickel and may develop a skin rash at the site of contact with a nickel containing object (*e.g.*, jewelry). However, contact with soil containing nickel will not normally cause a reaction (New Jersey DHSS, 1998; OMOE, 2001a).

15.5 EXPOSURE LIMITS

The Ontario Ministry of the Environment and Energy (OMOE) 24-hour reference concentration (RfC) of 2 $\mu g/m^3$ was used in the current assessment as an acute exposure limit for nickel (OMOE, 2001b). This limit was derived based on vegetation effects; however, it is more conservative then the one-hour reference exposure level (REL) of 6 $\mu g/m^3$ reported by the California Environmental Protection Agency (CalEPA) for respiratory system effects in human volunteers (OEHHA, 2000). Thus, the RfC of 2 $\mu g/m^3$ was employed as the acute exposure limit in the current assessment of nickel in air.

For the assessment of inhalation exposures, a Tolerable Concentration (TC) of 0.02 $\mu g/m^3$ for nickel oxide developed by the Canadian Environmental Protection Agency (CEPA, 1994; Health Canada, 2003) was employed. Assuming a typical breathing rate of 23 m^3/day and a body weight of 70 kg, this is equivalent to an inhalation Reference Dose (RfD) of 0.007 µg/kg bw/day.

This limit was derived based on non-carcinogenic health effects, but the specific basis could not be found.

For oral exposures, the only appropriate studies to derive a chronic exposure limit were those with soluble forms of nickel such as nickel soluble salts. The United States Environmental Protection Agency (US EPA) recommends an oral RfD of 20 µg/kg bw/day based on a study which showed no adverse effects in rats fed 100 ppm (5 mg/kg bw/day) nickel in their diet over a period of 2 years (US EPA IRIS, 2004). The US EPA applied an uncertainty factor of 300 to account for interspecies extrapolation (10), to protect sensitive populations (10) and to account for inadequacies in the reproductive studies. The resultant RfD was used in the current assessment.

In order to be run through the multi-media exposure model, the bioavailability of nickel was assessed for the various exposure pathways (*i.e.*, inhalation, ingestion and dermal contact). No specific data were identified in the literature regarding the amount of manganese that is absorbed via inhalation. It was therefore assumed that 100% of inhaled manganese is absorbed. Oral bioavailability in humans was assumed to be 27% and dermal bioavailability was assumed to be 0.1% for this assessment (RAIS, 2004).

15.6 REFERENCES

ATSDR (Agency for Toxic Substances and Disease Registry). 2003a. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Nickel. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

ATSDR (Agency for Toxic Substances and Disease Registry). 2003b. ToxFAQs – Nickel. Agency for Toxic Substances and Disease Registry. September, 2003. www.atsdr.cdc.gov/tfacts15.html

CEPA (Canadian Environmental Protection Act). 1994. Priority Substances List Assessment Report. Nickel. Government of Canada.

Health Canada. 2003. Federal Contaminated Site Risk Assessment in Canada. Part II: Health Canada Toxicological Reference Values (TRVs). Version 1.0. October 3, 2003.

New Jersey DHSS (Department of Health and Senior Services). 1998. Hazardous Substance Fact Sheet – Nickel. August 1998.

OEHHA (Office of Environmental Health Hazard Assessment). 2000. Acute Reference Exposure Levels (RELs), Averaging Times, and Toxicologic Endpoints. Arsenic and Inorganic Arsenic Compounds. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. May 2000.

OMOE (Ontario Ministry of the Environment and Energy). 2001a. Fact Sheet – Nickel in the Environment. March, 2001. Ontario Ministry of the Environment programs and initiatives.

OMOE (Ontario Ministry of the Environment and Energy). 2001b. Ambient Air Quality Criteria (AAQCs). Standards Development Branch. Ontario Ministry of the Environment and Energy (OMOE). September, 2001.

US EPA IRIS (United States Environmental Protection Agency's Integrated Risk Information System). 2004. IRIS database On-line search. U.S. Environmental Protection Agency, Cincinnati, OH.

16.0 NITROGEN DIOXIDE

Synonyms: NO_2; nitrogen peroxide

16.1 PHYSICAL/CHEMICAL PROPERTIES

Nitrogen dioxide (NO_2) is a reddish-brown gas with an odour described as "bleach" (AIHA, 1989). It is slightly soluble in water. Reported odour thresholds for NO_2 range from 110 to 1,000 $\mu g/m^3$ with a mean odour threshold of 728 $\mu g/m^3$ (van Gemert, 1999; Amoore and Hautala, 1983).

16.2 SOURCES

Nitrogen oxides are emitted by transportation and industrial sources (HSDB, 2004) and through combustion of fossil fuels. In addition, they are released in the reaction between nitric acid and

any organic material, in exhaust from metal cleaning, in electroplating, in dynamite blasting, automobile and diesel engine exhaust, burning of nitrocellulose and combustion of some shoe polishes (HSDB, 2004). Natural sources include forest fires, lightning and oxidation of nitrogen containing compounds in soil (HSDB, 2004).

16.3 ENVIRONMENTAL FATE

As an ubiquitous product of combustion, nitrogen dioxide can be transported great distances in regional air masses. Nitrogen dioxide gas is degraded in the atmosphere by hydroxyl radicals, leading to the formation of nitric acid, a primary component of acid rain. Photolysis of nitrogen oxides may contribute to the formation of smog and the production of ozone. Nitrogen oxides are extremely reactive in air, with an estimated atmospheric half-life of 35 hours. In soil, nitrogen dioxide may decompose to nitric acid or volatilize to the air. Wet deposition of nitrogen dioxide is another removal mechanism (Health Canada, 1998).

The most common route of exposure in humans is through inhalation or dermal contact (HSDB, 2004).

16.4 SYMPTOMS OF OVEREXPOSURE

In humans, the most common effects resulting from overexposure to NO_2 include the observation of increased airway resistance and altered lung function after short-term exposure under controlled conditions. Epidemiology studies have reported increased incidence of respiratory illness. Lung tissue damage and decreased immune functioning were observed in laboratory animals (Health Canada, 1998). NO_2 also may produce irritation of the eyes (HSDB, 2004).

NO_2 is considered to be the most toxic of the nitrogen oxides, therefore ambient air standards have been based primarily on data pertaining to this chemical species. NO_2 is a deep lung irritant that can produce pulmonary edema if it is inhaled at high concentrations. Epidemiological studies and animal studies suggest that peak concentrations contribute more to the toxicity of NO_2 than does an integrated dose (the product of concentration and time of exposure) (WHO, 1987). Nevertheless, animal studies indicate that repeated exposures can cause changes in lung structure and metabolism and may affect defences against bacterial infection (WHO, 1987).

In a review of clinical studies, Gong (1992) found that in healthy adolescents and adults, there were no acute pulmonary effects of NO_2 at concentrations from 0.1 to 4.0 ppm (188 to 7,480 μg/m^3); it was reported that short-term exposure to NO_2 at 1.5 to 2.0 ppm (2,822 to 3,740 μg/m^3) also enhanced cholinergic bronchial activity. However, Magnussen (1992) reported increased airway resistance during short-term exposure in healthy individuals at about 2.5 ppm (4,703 μg/m^3).

Gong (1992) estimated that adolescents and adults having either mild asthma or chronic obstructive pulmonary disease generally showed no effects at exposures of 0.12 to 4.0 ppm NO_2 (224 to 7,480 μg/m^3) for 20 to 120 minutes and 0.5 to 2.0 ppm NO_2 (935 to 3,740 μg/m^3) for one hour. This assessment is in general agreement with that of Magnussen (1992). Orehek *et al.* (1976) demonstrated an increased airway responsiveness in some asthmatics exposed to certain bronchoconstrictive agents following exposure to NO2 concentrations as low as 0.1 ppm (188 μg/m^3). Other asthmatics have demonstrated no or insignificant adverse bronchial effects at concentrations ranging from 0.1 to 0.2 ppm (188 to 374 μg/m^3) (Hazucha *et al*., 1983; Kleinman *et al*., 1983) or concentrations up to 4 ppm (7,526 μg/m^3) without exercise (Linn *et al*., 1985). Conversely, Bauer *et al.* (1986) demonstrated bronchospasm in exercising asthmatics exposed to 0.3 ppm NO_2 (560 μg/m^3), but not during rest, whereas exposure to 0.5 ppm NO_2 (935 μg/m^3) has been reported to increase airway reactivity in asthmatics, but not alter other lung functions (Mohsenin, 1987). Magnussen (1992) concluded that asthmatics were more sensitive to NO_2 than healthy subjects, but that their response depended on both the severity of their disease and the study protocol used.

Much variability exists among the NO_2 studies to date, with no particular study reporting a clearly defined concentration-response relationship (WHO, 2000).

16.5 EXPOSURE LIMITS

One-hour and 24-hour acute exposure limits have been reported by the Canadian Council of Ministers of the Environment (CCME, 1999). The one-hour acute guideline of 400 μg/m^3 and 24-hour acute guideline of 200 μg/m^3 were used for the assessment of nitrogen dioxide

concentrations in air based on Canadian Council of Ministers of the Environment (CCME) ambient air quality guidelines for NO_2 (CCME, 1999).

A chronic exposure limit of 60 $\mu g/m^3$ is reported by the CCME (1999). This objective was used for the assessment of nitrogen dioxide concentrations in air.

16.6 REFERENCES

AIHA (American Industrial Hygiene Association). 1989. Odour Thresholds for Chemicals with Established Occupational Health Standards. AIHA Press.

Amoore, J.E. and Hautala, E. 1983. Odour as an Aid to Chemical Safety: Odour Thresholds Compared with Threshold Limit Values and Volatilities for the 214 Industrial Chemicals in Air and Water Dilution. Journal of Applied Toxicology 3(6):272-290.

Bauer, M.A., Utell, M.J., Morrow, P.E., Speers, D.M., and Gibb, F.R. 1986. Inhalation of 0.03 ppm nitrogen dioxide potentiates exercise-induced bronchospasm in asthmatics. Am Rev Respir Dis 134:1203-1208.

CCME (Canadian Council of Ministers of the Environment). 1999. Canadian Environmental Quality Guidelines. URL: http://www.ccme.ca/assets/pdf/e1_062.pdf

Gong, H. 1992. Health effects of air pollution. A review of clinical studies. Clin Chest Med 13(2):210-214.

Hazucha, M.J., Ginsberg, J.F., McDonnell, W.F., Edward, D.H. Jr., Pimmel, R.L., Salaam, S.A., House, D.E., and Bromberg, P.A. 1983. Effects of 0.1 ppm nitrogen dioxide on airways of normal and asthmatic subjects. J Appl Physiol 54(3):730-739.

Health Canada. 1998. The Health and Environment Handbook for Health Professionals. Contaminant Profiles. Minister of Public Works and Government Services Canada.

HSDB (Hazardous Substances Data Bank). 2004. Nitrogen dioxide. Canadian Centre for Occupational Health and Safety. URL: http://toxnet.nlm.nih.gov/cgi-bin/sis/search

Kleinman, M.T., Bailey, R.M., Linn, W.S., Anderson, K.R., Whynot, J.D., Shamoo, D.A., and Hackney, J.D. 1983. Effects of 0.2 ppm nitrogen dioxide on pulmonary function and response to broncho provocation in asthmatics. J Toxicol Environ Health 12:815-826.

Linn, W.S., Solomon, J.C., Trim, S.C., Shamoo, D.A., Venet, T.G., Avol, E.L., and Hackney, J.D. 1985. Effects of exposure to 4 ppm nitrogen dioxide in healthy and asthmatic volunteers. Arch Environ Health 40(4):234-239.

Magnussen, H. 1992. Experimental exposure to nitrogen dioxide. Environ Respir J 5:1040-1042.

Mohsenin, V. 1987. Airway responses to nitrogen dioxide in asthmatic subjects. J Toxicol Environ Health 22:371-380.

Orehek, J., Massari, J.P., Gayrard, P.G., Grimaud, C., and Charpin, J. 1976. Effect of short-term, low-level nitrogen dioxide exposure on bronchial sensitivity of asthmatic patients. J Clin Invest 57:310-307.

van Gemert, L.J. 1999. Compilations of odour threshold values in air and water. TNO Nutrition and Food Research Institute, Boelens Aroma Chemical Information Service (BACIS), The Netherlands, 1999.

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe. World Health Organization: Copenhagen. URL: http://www.who.int/peh/air/Airquality

17.0 PARTICULATE MATTER

Synonyms: aerosols; particles; particulates; $PM_{2.5}$; PM_{10}

17.1 PHYSICAL/CHEMICAL PROPERTIES

Particulate matter consists of minute, solid or liquid particles that remain suspended in the air and that can be inhaled into the respiratory system. The particles vary in size, shape and chemical composition and are a vehicle for a number of compounds that adsorb onto the surface of particulates. The fine particulate fraction (less than 2.5 microns) is composed mainly of

particles such as sulphates that are formed from gases, while the coarse fraction (>10 microns) is formed from the break-up of larger particles originating from wind blown dust. Accordingly, coarse particles consist of mainly earth crust oxides (*i.e.*, iron, calcium, silicon and aluminum) and fine particulates consist of chemicals derived from anthropogenic sources (*i.e.*, sulphate, nitrate, ammonium, inorganic and organic compounds and heavy metals). Secondary particles form when gaseous chemicals react with atmospheric particles. Gaseous precursors may include NO_x, SO_2, VOCs and NH_3. Though the components of particulates can include a wide-range of chemicals, particle size is the primary parameter to characterize particulate physical behaviour (Health Canada, 1998).

17.2 SOURCES

PM originates from both natural and man-made sources. Natural sources include volcanoes, wind erosion of soil and rock, forest fires and plants. The main sources associated with human activity are industrial processes, fuel combustion, transportation and solid wastes. Industrial operations emitting very fine particles include power stations, smelters, mills, refineries, factories, solid waste incinerators and construction activities. Other sources include agriculture, landfills and exhaust from motor vehicles, aircraft and marine vessels. Particulates can contain a variety of compounds such as acid mist, arsenic, beryllium, cadmium, lime, mercury and silica (Health Canada, 1998).

17.3 ENVIRONMENTAL FATE

Particle size affects removal rate and residence times in the atmosphere (CEPA, 2000). Effective scattering of light by smaller particles can contribute to reduced visibility (CEPA, 2000). Resident time is dependent upon meteorological factors such as wind speed and temperature. The chemical constituents adsorbed on the surface of the particle determine its persistence (Health Canada, 1998). As particles settle out they may deposit on water, plants and soils. Contaminants adsorbed on the particles may then enter the food chain through direct absorption by plants, or via other pathways (Health Canada, 1998). However, with particulate matter specifically, the pathway of concern is exposure to inhalable particles via the air pathway, particularly the finer fraction (< 2.5 μm) that can penetrate deeper into the lungs.

17.4 SYMPTOMS OF OVEREXPOSURE

Fine particulate matter ($PM_{2.5}$) can enter deep into the airways where there is the greatest potential for health effects; however, the chemical composition of the particle will determine its effect on health. Some of the particles themselves may be toxic or they may have toxic elements adsorbed onto them.

The main health effects of concern with PM exposure are affected pulmonary function, increased respiratory symptoms and aggravation of existing heart and lung disease as measured by increased physician visits, hospitalization and mortality (Burnett *et al*., 1997; Delfino *et al*., 1997; Schwartz, 1994; Thurston *et al*., 1994). An increased risk of lung cancer mortality has been identified more recently (Pope *et al*., 2002). Particulates in the lung also may impede the natural ability of the respiratory system to clear itself of foreign matter and may affect other body defence mechanisms.

Studies indicate an association between premature mortality due to respiratory disease and airborne particulates (among other air pollutants). This is supported by 19 time-series epidemiological studies, where a significant relationship was found between relative risk and PM_{10} concentrations in 21 out of 23 cities across North and South America and Europe (CEPA, 2000). Schwartz *et al*. (1996; reanalyzed in HEI, 2003) studied the effects of PM2.5 in six US cities and found almost double the mortality rate as compared to PM10. Mortality rates have been observed to increase at even low ambient levels (*i.e*., PM10 concentrations range from below 80 to 100 $\mu g/m^3$) (Schwartz, 1993; Ostro *et al.*, 1996, Pope and Kalkstein, 1996).

The association between PM exposure and the range of health outcomes described above could not be explained by the influence of weather, season, yearly trends, day-to-day variations or variations due to holidays, epidemics or other non-pollutant factors (CEPA, 2000; US EPA, 2003ab). In addition, they could not be explained by co-exposure to other air pollutants since associations of a similar magnitude were observed across a large number of locations with differing air pollutant mixtures (CEPA, 2000; US EPA, 2003ab). The association with particulate matter remained when the analyses were adjusted for the effects of various other pollutants.

The most vulnerable segments of the population to particulate air pollution seem to be the elderly, those with chronic pulmonary or heart disease, and the very young. In addition, asthmatics, smokers and people with the flu or bronchitis were found to be vulnerable (Health Canada, 1998).

The results of controlled experimental studies with animals and humans provide limited support for the epidemiological findings described above. The health effects observed in controlled studies tend to occur at much higher particulate concentrations than those associated with health effects in the epidemiological studies examining ambient air exposures. This is thought to be a function of the constraints imposed on experimental work by the technological difficulties in reproducing environmentally relevant particulate matter. Additionally, the particles to which volunteers are exposed in human clinical studies do not adequately reflect the complexity of ambient particles (CEPA, 2000).

Chronic particulate exposure in animal toxicity studies resulted in a decrease in lung function in pigs and rats (Weister *et al*., 1980; Begin *et al*., 1989; Heinrich *et al*., 1989), impaired airway clearance in guinea pigs, donkeys and rabbits (Gearhart and Schlesinger, 1989; Nagi *et al*., 1991; Schlesinger *et al*., 1992; Samet and Cheng, 1994) and immune function reduction in the lungs of rats and mice (Spiegelberg *et al*., 1984; Gilmour *et al*., 1989ab; Kleinman *et al*., 1995). Examples of short-term particulate exposures causing cardiovascular effects and respiratory effects in animal studies have been demonstrated by Bouthillier *et al*., 1998; Vincent *et al*., 2001; Zeilifof *et al*., 2003; and Dye *et al*., 2001.

17.5 EXPOSURE LIMITS

The Canadian Council of Ministers of the Environment (CCME) has developed an acute Canada-Wide Standard (CWS) of 30 $\mu g/m^3$ for 24-hour exposure to $PM_{2.5}$ (CCME CWS, 2000). This exposure limit should be compared to a 98th percentile concentration value, averaged over three consecutive years. The CWS for $PM_{2.5}$ represents a balance between the desire to achieve the best health/environmental protection possible and the costs of pollution reduction. CCME (2000), however, cautioned that it may not be fully protective and may need to be re-visited at some future date, targeted as 2010.

Health Canada has established a short-term reference level of 15 μg/m^3 for 24-hour exposure to $PM_{2.5}$ (Health Canada/Environment Canada, 1999). This exposure limit was derived based on epidemiological observations of PM associations with mortality and hospitalizations. However, this is not currently being proposed as a regulatory guideline.

The chronic exposure limit for $PM_{2.5}$ of 12 μg/m^3 was based on the draft California Air Resources Board's (CARB) annual average standard for $PM_{2.5}$. This draft standard was recommended based on long-term epidemiological studies associating PM2.5 with mortality at a mean concentration of 18 μg/m^3 (CARB, 2002). It also gives weight to the results of multiple daily exposure studies that examined relationships between $PM_{2.5}$ and adverse health outcomes where long-term means were in the range of 13 to 18 μg/m^3.

The acute exposure limit for PM_{10} of 50 μg/m^3 was based on the British Columbia Ministry of Water, Land and Air Protection (BC MWLAP) recommended 24-hour exposure limit (BC MWLAP, 1995). This air quality objective recognises the potential health effects from exposure to PM_{10} and is considered to be equivalent to a maximum acceptable level described by the federal Canadian Environmental Protection Act. No one-hour acute exposure limit for PM_{10} is reported by Canadian regulatory agencies.

A chronic exposure limit of 30 μg/m^3 was used for the assessment of PM_{10} concentrations in air (BC GVRD, 2002). The specific basis for the development of this guideline was not provided.

17.6 REFERENCES

BC GVRD (British Columbia, Greater Vancouver Regional District). 2002. Lower Fraser Valley Ambient Air Quality Report 2001. Greater Vancouver Regional District, Policy and Planning Department, October 2002.

BC MWLAP (British Columbia Ministry of Water, Land and Air Protection). 1995. Air Quality Objectives and Standards. URL: http://wlapwww.gov.bc.ca/air/airquality/airqual_1.pdf

Begin, R.A, Dufresne, A., Cantin, A., Possmayer, F., and Sebastien, P. 1989. Quartz exposure, retention, and early silicosis in sheep. Exp. Ling Res. 15: 409-428. Cited In: CEPA 2000.

Bouthillier, L.; Vincent, R.; Goegan, P.; Adamson, I. Y. R.; Bjarnason, S.; Stewart, M.; Guénette, J.; Potvin, M.; Kumarathasan, P. 1998. Acute effects of inhaled urban particles and ozone: lung morphology, macrophage activity, and plasma endothelin-1. Am. J. Pathol. 153: 1873-1884.

Burnett, R.T., Brook, J.R., Cakmak, S., and Krewski, D. 1997. The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiovascular diseases. Environ. Health Perspectives. 105: 614-620. Cited in CEPA 2000. Particulate Matter.

CARB (California Air Resources Board). 2002. Ambient Air Quality Standards. URL: http://www.arb.ca.gov/research/aaqs/caaqs/caaqs.htm

Health Canada/Environment Canada. 1999. National Ambient Air Quality Objectives for Particulate Matter. Part 1: Science Assessment Document. CEPA/Federal-Provincial Advisory Committee Working Group on Air Quality Objectives and Guidelines. Ottawa.

CCME (Canadian Council Ministry of the Environment). 2000. Canada-Wide Standards for Particulate Matter (PM) and Ozone. Accepted November 29, 1999, for Endorsement in May 2000.

CEPA (Canadian Environmental Protection Act). 2000. Priority Substances List Assessment Report. Respirable Particulate Matter Less Than or Equal To 10 Microns. Government of Canada. May 2000.

Delfino, R.J., Murphy-Moulton, A.M., Burnett, R.T., Brook, J.R., and Becklake, M.R. 1997. Effects of air pollution on emergency room visits for respiratory illnesses in Montreal, Quebec. Am. J. Respir. Crit. Care Med. 155:568-576. Cited In: CEPA 2000.

Dye, J. A.; Lehmann, J. R.; McGee, J. K.; Winsett, D. W.; Ledbetter, A. D.; Everitt, J. I.; Ghio, A. J.; Costa, D. L. 2001. Acute pulmonary toxicity of particulate matter filter extracts in rats: coherence with epidemiological studies in Utah Valley residents. Environ. Health Perspect. Suppl. 109(3): 395-403.

Gearheart, J.M., and Schlesinger, R.B. 1989. Sulphuric acid-induced changes in the physiology and structure of the tracheobronchial airways. Environ. Health Perspect. 79:127-137. Cited In: CEPA 2000.

Gilmour, M.I., Taylor, F.G.R., Baskerville, A., and Wathes, C.M. 1989a. The effect of titanium dioxide inhalation on the pulmonary clearance of Pasteurella haemolytica in the mouse. Environ. Res. 50:157-172. Cited In: CEPA 2000.

Gilmour, M.I., Taylor, F.G.R, and Wathes, C.M. 1989b. Pulmonary clearance of Pasteurella haemolytica and immune response in mice following exposure to titanium dioxide. Environ. Res. 50-184-194. Cited In: CEPA 2000.

Health Canada. 1998. The Health and Environment Handbook for Health Professionals. Contaminant Profiles. Minister of Public Works and Government Services Canada.

HEI (Health Effects Institute). 2003. Special Report. Revised analyses of time-series studies of air pollution and health. May 2003.

Heinrich, U., Muhle, H., Takenaka, S., Ernst, H., Fuhst, R., Mohr, U., Pitt, F., and Stober, W. 1989. Chronic effects on the respiratory tract of hamsters, mice and rats after long-term inhalation of high concentrations of filtered and unfiltered diesel engine emissions. J. Appl. Toxicol. 6(6):383-395. Cited In: CEPA 2000.

Kleinman, M.T., Bhalla, D.K., Mautz, W.J., and Phalen, R.F. 1995. Cellular and immunologic injury with PM10 inhalation. Inhal. Toxicol. 7:589-602. Cited In: CEPA 2000.

Nagi, A., Sakamoto, K., Takizawa, T., Morinibu, S., Yamano, Y., Nagao, N., Ishihara, Y., and Kagawa, J. 1991. Sulphuric acid submicron aerosols induce morphological changes in the central airways in the guinea pig. Jpn. J. Thorac. Dis. 29: 992-997. Cited In: CEPA 2000.

Ostro, B., Sanchez, J.M., Aranda, C. and Eskeland, G. 1996. Air pollution and mortality: results from a study of Santiago, Chile. J Expos Anal Environ Epidemiol 6(1): 97-114. Cited In: CEPA 2000.

Pope, C.A., and Kalkstein, L.S. 1996. Synoptic weather modeling and estimates of the exposure-response relationship between daily mortality and particulate air pollution. Environ. Health Perspect. 104(4): 414-420. Cited In: CEPA 2000.

Pope, C.A., Burnett, R.T., Thun, M.J., Calle, E.E., Krewski, D., Ito, K., and Thurston, G.D. 2002. Lung cancer, cardiopulmonary mortality and long-term exposure to fine particulate air pollution. JAMA. 287(9), 1132-1141.

Samet, J.M., and Cheng, P.W. 1994. The role of airway mucus in pulmonary toxicology. Environ. Health Perspect. 102(Suppl. 2): 89-103. Cited In: CEPA 2000.

Schlesinger, R.B., Gorczynski, J.E., Dennison, J., Richards, L., Kinney, P.L., and Bosland, M.C. 1992. Long-term intermittent exposure to sulphuric acid aerosol, ozone, and their combination: Alterations in tracheobronchial mucociliary clearance and epithelial secretory cells. Exp. Lung Res. 18: 505-534. Cited In: CEPA 2000.

Schwartz, J. 1993. Air pollution and daily mortality in Birmingham, Alabama. Am. J. Epidemiol. 137(10):1136-1147. Cited In: CEPA 2000.

Schwartz, J. 1994. Air pollution and hospital admissions for the elderly in Detroit, Michigan. Am. J. Respir. Care Med. 150: 648-655. Cited In: CEPA 2000.

Schwartz, J., Dockery, D.W., Neas, L.M. 1996. Is daily mortality associated specifically with fine particles? J. Air Waste Manage. Assoc. 46:927-939.

Spiegelberg, T., Kordel, W., and Hochrainer, D. 1984. Effects of NiO inhalation on alveolar macrophages and the humeral immune systems in rats. Ecotoxicol. Environ. Saf. 8:516-525. Cited In: CEPA 2000.

Thurston, G.D., Ito, K., Hayes, C.G., Bates, D.V., and Lippmann, M. 1994. Respiratory hospital admissions and summertime haze air pollution in Toronto, Ontario: consideration of the role of acid aerosols. Environ. Res. 65:271-290. Cited In: CEPA 2000.

US EPA (United States Environmental Protection Agency). 2003a. Air Quality Criteria for Particulate Matter. Fourth External Review Draft. June, 2003. United States Environmental Protection Agency. URL: http://www.epa.gov/ncea/partmatt.htm

US EPA (United States Environmental Protection Agency). 2003b. Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and technical Information. OAQPS Staff Paper – First Draft. August 2003

US EPA IRIS (Integrated Risk Information System). 2004. IRIS Database On-line search. Formaldehyde. United States Environmental Protection Agency, Cincinnati, OH.

Vincent, R.; Kumarathasan, P.; Goegan, P.; Bjarnason, S. G.; Guénette, J.; Bérubé, D.; Adamson, I. Y.; Desjardins, S.; Burnett, R. T.; Miller, F. J.; Battistini, B. 2001. Inhalation toxicology of urban ambient particulate matter: acute cardiovascular effect in rats. Boston, MA: Health Effects Institute; research report no. 104. URL: http://www.healtheffects.org/Pubs/Vincent.pdf [29 January 02].

Weister, M.J., Iltis, R. and Moore, W. 1980. Altered function and histology in guinea pigs after inhalation of diesel exhaust. Environ. Res. 22:285-297. Cited In: CEPA 2000.

Zelikoff, J. T.; Chen, L. C.; Cohen, M. D.; Fang, K.; Gordon, T.; Li, Y.; Nadziejko, C.; Schlesinger, R. B. 2003. Effects of inhaled ambient particulate matter on pulmonary antimicrobial immune defense. Inhalation Toxicol. 15: 131-150.

18.0 SULPHUR DIOXIDE

Synonyms: Sulfurous oxide; sulfur oxide; sulfurous anhydride; sulfur dioxide

18.1 PHYSICAL/CHEMICAL PROPERTIES

Sulphur dioxide is a highly soluble gas with a characteristic metallic taste and sharp irritating odour (AIHA, 1989). Reported odour thresholds range from 870 to 21,000 $\mu g/m^3$ with a mean odour threshold of 2,860 $\mu g/m^3$ (van Gemert, 1999; Amoore and Hautala, 1983).

18.2 SOURCES

The major sources of sulphur dioxide are industrial, including metal smelters, petroleum refining, pulp and paper mills, electrical utilities, incineration, and fuel combustion (Health Canada, 1998). Poorly vented kerosene space heaters are an indoor source of sulphur dioxide (SO_2) and volcanoes produce it naturally.

18.3 ENVIRONMENTAL FATE

Due to its high vapour pressure SO_2 usually exists as a gas. Depending upon climate and region, the resident time of SO_2 in the atmosphere is approximately one to ten days. SO_2 in the air may be photochemically oxidized to sulphur trioxide and/or sulphate. Hydroxyl radicals also will oxidize SO_2. As SO_2 is extremely soluble in water, it may combine with water molecules to produce sulphuric acid (H_2SO_4) in the form of fine droplets of aerosols. Adsorption of SO_2 by cloud cover or rainwater may be facilitated by the presence of iron and manganese ions, a high pH and high temperature. H_2SO_4 in the atmosphere then may be neutralized by ammonia to the neutral salt ammonium sulphate. Neutralization rates are much higher over cities and agricultural areas than forests and water bodies. SO_2 is effectively removed from the atmosphere via wet and dry deposition. Plants also may remove SO_2 from the atmosphere. Uptake of SO_2 in the soil is dependent upon soil pH and the moisture content (ATSDR, 1998; HSDB, 2004).

Data is lacking in regards to bioaccumulation following ingestion of soil, water, air and foods grown in SO_2 contaminated areas (ATSDR, 1998).

18.4 SYMPTOMS OF OVEREXPOSURE

The most common effects resulting from short-term overexposure to SO_2 involve the breathing passages and include throat or lung irritation or bronchospasm in asthmatics (WHO, 1999). Generally, levels of sulphur dioxide in ambient air will not produce adverse effects in healthy individuals (Health Canada, 1998).

Reported long-term health effects include reduction of lung function, chronic respiratory symptoms in asthmatics and increased hospital admissions to acute care hospitals (Health Canada, 1998).

Sulphur dioxide is known to be a potent bronchoconstrictor and has been shown to induce bronchoconstriction, hyper reactivity and airway inflammation in both human and animal studies. The response is rapid in onset, with a maximum effect in 5 to 10 minutes, and resolves within 30 minutes (UK Department of Health, 1995). The magnitude of the response is dependent on several factors, including: i) the concentration of SO_2 inhaled; ii) the breathing rate; iii) the breathing pattern (*i.e.*, nasal *vs*. oronasal *vs*. oral); and, the degree of underlying airway hyper reactivity (Bethel *et al., 19*83). Conditions that could pre-dispose toward increased sensitivity to SO_2 may include chronic bronchitis, cystic fibrosis and any respiratory disorder characterized by hyperactive airways (Bethel *et al., 19*83).

Asthmatics are known to be especially sensitive to the bronchoconstriction induced by SO_2 and will respond to lower concentrations than the normal population. Individual responses among asthmatics can be highly variable, presumably due to differences in individual sensitivities of the mechanism(s) responsible for the SO_2-induced bronchoconstriction. Symptoms most commonly reported by asthmatics following challenge with SO2 in controlled laboratory settings are shortness of breath, chest discomfort, wheezing, non-productive cough, substernal irritation, and chest tightness.

In clinical studies conducted to date, clear responses have not been observed in healthy subjects exposed for brief periods to concentrations of SO_2 less than 0.75 to 1.0 ppm (equivalent to approximately 2,000 to 2,600 $\mu g/m^3$) (ATSDR, 1998; Environment Canada, 1987; Linn *et al., 19*83; UK Department of Health, 1995). A similar lack of response has been reported for non-exercising asthmatics at a concentration of 0.5 ppm (1,300 $\mu g/m^3$) (Linn *et al., 19*83; Sheppard *et al., 19*81).

18.5 EXPOSURE LIMITS

A 10-minute acute guideline of 500 $\mu g/m^3$ recommended by the World Health Organisation was used for the assessment of SO_2 in air (WHO, 2000). This guideline is health-based and relies on

controlled studies of the most sensitive population (*i.e.,* asthmatics) to air pollutants such as SO_2 and thus is expected to provide an adequate margin of safety for the general population.

The acute exposure limits used for the assessment of SO_2 concentrations in air were based on the Canadian Council of Minister of the Environment (CCME) ambient air quality guidelines for sulphur dioxide (CCME, 1999). These include a one-hour guideline of 450 $\mu g/m^3$ and a 24-hour guideline of 150 $\mu g/m^3$. The one-hour guideline was based on taste and odour discomfort in sensitive individuals in conjunction with potential injury to vegetation. Development of the 24-hour guideline relied on a health-based study demonstrating an increased incidence of hospital admission rates reported when SO_2 concentrations exceeded 300-500 $\mu g/m^3$.

The chronic exposure limit of 25 $\mu g/m^3$ used for the assessment of sulphur dioxide concentrations in air is based on B.C. Level A air quality objectives and standards (BC MWLAP, 1995). The specific basis for the development of this guideline was not provided.

18.6 REFERENCES

AIHA (American Industrial Hygiene Association). 1989. Odour Thresholds for Chemicals with Established Occupational Health Standards. AIHA Press

Amoore, J.E. and Hautala, E. 1983. Odour as an Aid to Chemical Safety: Odour Thresholds Compared with Threshold Limit Values and Volatilities for the 214 Industrial Chemicals in Air and Water Dilution. Journal of Applied Toxicology, 3(6):272-290.

ATSDR (Agency for Toxic Substances and Disease Registry). 1998. Toxicological profile for Sulphur dioxide. URL: http://www.atsdr.cdc.gov/toxprofiles/tp116.html

BC MWLAP (British Columbia Ministry of Water, Land and Air Protection). 1995. Air Quality Objectives and Standards. URL: http://wlapwww.gov.bc.ca/air/airquality/airqual_1.pdf

Bethel, R.A., Epstein, J., Sheppard, D., Nadel, J.A., and Boushey, H.A. 1983. Sulfur dioxide-induced bronchoconstriction in freely breathing, exercising, asthmatic subjects. Am Rev Respir Dis 128: 987-980.

CCME (Canadian Council of Ministers of the Environment). 1999. Canadian Environmental Quality Guidelines. URL: http://www.ccme.ca/assets/pdf/e1_062.pdf

Environment Canada. 1987. Review of National Ambient Air Quality Objectives for Sulphur Dioxide. Desirable and acceptable levels. Federal-Provincial Advisory Committee on Air Quality. April, 1987.

Health Canada. 1998. The Health and Environment Handbook for Health Professionals. Minister of Public Works and Government Services Canada.

HSDB (Hazardous Substances Data Bank). 2004. Canadian Centre for Occupational Health and Safety. Sulphur dioxide. URL: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

Linn, W.S., Shamoo, D.A., Spier, C.E., Valencia, L.M., Anzar, U.T., Venet, T.G., Hackney, J.D. 1983. Respiratory effects of 0.75 ppm sulfur dioxide in exercising asthmatics: influence of upper-respiratory defences. Environmental Research. 30:340-348.

Sheppard, D., Saisho, A., Nadel, J.A., Boushey, H.A. 1981. Exercise increases sulfur dioxide-induced bronchoconstriction in asthmatic subjects. Am Rev Respir Dis 123:486-491.

UK Department of Health (United Kingdom Department of National Health). 1995. Asthma and Outdoor Air Pollution. Department of Health. Committee on the Medical Effects of Air Pollutants. London: HMSO.

van Gemert, L.J. 1999. Compilations of odour threshold values in air and water. TNO Nutrition and Food Research Institute, Boelens Aroma Chemical Information Service (BACIS), The Netherlands, 1999.

WHO (World Health Organization). 1999. Air Quality Guidelines for Europe. World Health Organization: Copenhagen. URL: http://www.who.int/peh/air/Airqualityg