CCME

Canadian Council of Ministers of the Environment

Le Conseil canadien des ministres de l'environnement

SCIENTIFIC CRITERIA DOCUMENT FOR THE DEVELOPMENT OF THE CANADIAN SOIL QUALITY GUIDELINES FOR METHANOL

Protection of Environmental and Human Health

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EXECUTIVE SUMMARY

Canadian environmental quality guidelines are numerical concentrations or narrative statements recommended to provide a healthy, functioning ecosystem capable of sustaining the existing and likely future uses of the site by ecological receptors and humans. Canadian soil quality guidelines can be used as the basis for consistent assessment and remediation of contaminated sites in Canada.

The guidelines in this report were derived according to procedures described in A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines (CCME 2006). According to this protocol, both environmental and human health soil quality guidelines are developed and the lowest value generated from the two approaches for each of the four land uses is recommended by the Canadian Council of Ministers of the Environment (CCME) as the Canadian Soil Quality Guidelines (CCME 2006).

This scientific criteria document provides the background information and rationale for the derivation of environmental and human health soil quality guidelines for methanol. It contains a review of the chemical and physical properties of methanol, the sources and emissions in Canada, the distribution and behaviour of methanol in the environment and the behaviour and effects of methanol in humans and mammalian species. This information is used to derive soil quality guidelines for methanol to protect human and ecological receptors in four types of land uses: agricultural, residential/parkland, commercial, and industrial.

Sufficient data were available to develop soil quality guidelines for methanol protective of human health, in accordance with the soil protocol. The human health soil quality guidelines for methanol are 4.6 mg/kg for coarse soil and 5.6 mg/kg for fine soil for all four land uses. Human health soil quality guidelines were calculated for soil ingestion, inhalation of indoor air, and protection of groundwater for drinking water. The limiting pathway in the calculation of human health guidelines was drinking water.

Sufficient data were available to develop soil quality guidelines for methanol protective of environmental health, in accordance with the soil protocol. The environmental health soil quality guidelines for methanol are: 7.7 mg/kg for coarse soil and 190 mg/kg for fine soil for all four land uses. Environmental health soil quality guidelines were calculated for ecological direct contact and protection of groundwater for aquatic life. The limiting pathway in the calculation of environmental health guidelines was aquatic life.

Since it was possible to calculate both human health and environmental soil quality guidelines for methanol, the overall methanol soil quality guidelines are the lower of the two, which are 4.6 mg/kg for coarse soil and 5.6 mg/kg for fine soil for all four land uses.

RÉSUMÉ

Les recommandations canadiennes pour la qualité de l'environnement sont des limites quantitatives ou descriptives recommandées dans le but d'assurer un écosystème sain, capable de supporter les utilisations actuelles et probables du site par les récepteurs écologiques et humains. Les recommandations canadiennes pour la qualité des sols peuvent être utilisées comme base pour l'uniformisation des processus d'évaluation et d'assainissement des terrains contaminés au Canada.

Les recommandations dans ce rapport ont été élaborées selon les procédures décrites dans le *Protocole d'élaboration de recommandations pour la qualité des sols en fonction de l'environnement et de la santé humaine* (CCME 2006). Conformément à ce protocole, les recommandations pour la qualité des sols (RQSo) visant la protection de l'environnement et de la santé humaine sont développées, et la plus petite valeur obtenue de ces deux procédures, pour chacun des quatre types d'utilisations des terres, est recommandée par le CCME comme étant la RQSo (CCME 2006).

Le présent document scientifique présente les renseignements généraux et les justifications qui sous-tendent l'élaboration de RQSo visant à protéger l'environnement et la santé humaine contre le méthanol. Il contient une analyse des propriétés chimiques et physiques du méthanol, des sources et émissions au Canada, de la distribution et du comportement du méthanol dans l'environnement ainsi que du comportement et des effets du méthanol chez les humains et certaines espèces mammifères. Ces renseignements ont servi à l'élaboration de RQSo pour le méthanol visant à protéger les récepteurs humains et écologiques dans quatre types d'utilisations des terres, à savoir : utilisation agricole, utilisation résidentielle/parc, utilisation commerciale et utilisation industrielle.

Il y avait suffisamment de données pour élaborer des RQSo visant la protection de la santé humaine contre le méthanol, conformément au protocole applicable au sol. Les RQSo visant la protection de la santé humaine contre le méthanol sont 4,6 mg/kg pour le sol à texture grossière et 5,6 mg/kg pour le sol à texture fine, et ce, pour les quatre types d'utilisations des terres. Des RQSo relatives à la santé humaine ont été calculées pour l'ingestion de sol, l'inhalation d'air intérieur et la protection des eaux souterraines destinées à l'alimentation en eau potable. Le mécanisme limitant le calcul des recommandations relatives à la santé humaine était l'eau potable.

Il y avait également assez de données pour élaborer des RQSo visant la protection de l'environnement contre le méthanol, conformément au protocole applicable au sol. Les RQSo visant la protection de l'environnement contre le méthanol sont 7,7 mg/kg pour le sol à texture grossière et 190 mg/kg pour le sol à texture fine, et ce, pour les quatre types d'utilisations des terres. Des RQSo relatives à l'environnement ont été calculées pour le contact direct avec le sol et pour la protection des eaux souterraines (vie aquatique). Le mécanisme limitant le calcul des RQSo relatives à l'environnement était la vie aquatique.

Puisqu'il a été possible de calculer des RQSo pour le méthanol aussi bien aux fins de protection de la santé humaine qu'aux fins de protection de l'environnement, les recommandations générales pour le méthanol sont les plus basses des deux valeurs calculées, soit 4,6 mg/kg pour le sol à texture grossière et 5,6 mg/kg pour le sol à texture fine, et ce, pour les quatre types d'utilisations des terres.

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1. INTRODUCTION

Methanol is a naturally occurring substance as well as an industrial chemical with a wide range of uses as a chemical feedstock, solvent and fuel. It is also used in the upstream oil and gas industry for hydrate inhibition in natural gas production and transport, removal of acid gasses, as a dehydration agent, in the recovery of heavy hydrocarbons, and in the pressure testing of pipelines and pressure vessels in cold temperatures. Any of these uses may result in the release of methanol into the environment. Common synonyms and trade names for methanol are included in Table 1.

Table 1. Common Synonyms and Trade Names for Methanol

Methanol	Methyl alcohol
Carbinol	colonial spirit
columbian spirit	Methylol
methyl hydroxide	pyroxylic spirit
monohydroxymethane	wood naphtha
Wood alcohol	wood spirit

This document develops proposed soil and groundwater quality guidelines consistent with A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines (CCME 2006).

2. BACKGROUND INFORMATION

2.1 Chemical and Physical Properties

Chemical and physical properties of methanol are summarized in Table 2. Methanol is characterized as a colourless, polar organic solvent that is miscible with water.

2.2 Analytical Methods

One of the principal reference sources for analytical methods for water, soils, and other materials is the U.S. EPA Document SW-846: "*Test Methods for Evaluating Solid Wastes – Physical/Chemical Methods*" (U.S. EPA 2004b). U.S. EPA Methods referred to below are sourced from this document. Most techniques for the analysis of methanol in soil include the following three elements:

1. sample extraction
2. sample preparation and
3. separation, followed by detection and quantification of the volatile compounds.

Methanol is first extracted from soil samples using water or another appropriate solvent. This step is not necessary for water samples.

Table 2. Physical and Chemical Properties for Methanol

Property	Units	Methanol	Source
Formula	-----	CH_3OH	1
CAS number	-----	67-56-1	1
Molecular weight	g/mole	32.04	2
Melting point	°C	-97.8	2
Boiling point	°C	64.7	2
Specific gravity (at 20/4 °C)	g/cm^3	0.791	2
Vapour density (air = 1)	-----	1.11	3
Vapour pressure (at 5 °C)	Pa	5,320	3,7
Vapour pressure (at 25 °C)	Pa	1.7×10^4	12
Solubility (at 25 °C)	mg/L	miscible	2
	g/L	1,163	3
Henry's law constant	$atm \cdot m^3/mol$	4.6×10^{-6}	3
Dimensionless Henry's law constant	-----	2.0×10^{-4}	5
Organic carbon partition coefficient (K_{oc})	log	-0.57	4
n-Octanol-water partition coefficient (K_{ow})	log	-0.73	3
	log	-0.66	2
Diffusion coefficient in air	cm^2/s	0.15	11
Conversion factor: 1ppm =	mg/m^3	1.31	8
Odour threshold (unadapted panelists)	mg/m^3	2,660	9
Biodegradation half-life in soil	days	1 to 7	6
Biodegradation half-life in surface water	days	1 to 7	6
Biodegradation half-life in groundwater	days	245	10

Sources:
[1]CRC (1996)
[2]Werl Treatability Database (1993) as reported in GRI (1996)
[3]Montgomery (1991)
[4]Calculated from K_{ow} using Baker *et al.* (1997) equation provided in Boethling and Mackay (2000; Table 8.1)
[5]Recalculated using the ideal gas law
[6]Howard *et al.* (1991)
[7]Recalculated from Montgomery (1991; 40 mm Hg) using the conversion 1 mm Hg = 1 torr = 133 Pa
[8]Adapted from Clayton and Clayton (1982)
[9]Verschueren (2001)
[10]Derived From API (1994), see Section 3.4
[11]ORNL (2007)
[12]Mackay *et al.* (2006)

U.S. EPA Methods for Sample Preparation

U.S. EPA-recommended methods for introducing a methanol-containing sample into the Gas Chromatograph are summarized below.

- Direct Injection.
- U.S. EPA Method 5031 "Volatile, non-purgeable, water-soluble compounds by azeotropic distillation" involves using an azeotrope with water to introduce the sample into the GC, and is used for water-soluble compounds that are not amenable to purge-and-trap or headspace techniques.

U.S. EPA Methods for Separation and Detection/Quantification

U.S. EPA-recommended methods for methanol for separation and detection/quantification include the following:

- EPA Method 8015B "Non-halogenated organics using GC/FID" provides details of a methodology involving gas chromatographic separation and flame ionization detection (FID).
- EPA Method 8260B "Volatile organic compounds by gas chromatography/mass spectrometry" provides details of a methodology involving gas chromatographic separation and identification/quantitation using mass spectrometry.

2.3 Production and Uses

The vast majority of commercial methanol is made from synthesis gas. Syngas is produced by steam reforming of methane, liquefied petroleum gas or naphtha to produce a mixture of H_2, CO, CO_2, and water. In steam reforming of natural gas, methane and steam are combined in a reactor with a catalyst (nickel) at a temperature between 700 and 1,100°C and at 10 to 50 bar pressure. Methanol is made from purified syngas in tubular reactors packed with catalyst (typically Cu/ZnO on alumina). The overall reaction is $CO + 2H_2 \leftrightarrow CH_3OH$. Methanol synthesis reactors operate at temperatures between 250 and 350°C and at pressures of 30-100 bar (Kirk-Othmer 1999).

Global production capacity for methanol was 95 million tonnes in 2012 (MMSA 2013) and 470 thousand tonnes per year in Canada (Cheminfo Services 2014).

Methanol usage is summarized in Figure 1 (1985 data from a U.S. survey; data source WHO 1997). As shown in that figure, the majority of methanol production (71%) is used as a chemical feedstock in the synthesis of other industrial chemicals including formaldehyde, acetic acid, methyl halides, and methyl t-butyl ether (MTBE). Other uses of methanol can be categorized into solvent (10%), fuel (6%), and miscellaneous (13%).

Oilfield uses of methanol include hydrate inhibition in natural gas production and transport, removal of acid gasses, as a dehydration agent, in the recovery of heavy hydrocarbons (Esteban *et al.* 2001), and in the pressure testing of pipelines and pressure vessels in cold temperatures (CAPP 1996). All of these uses would fall under the "solvent" or "miscellaneous" categories in Figure 1.

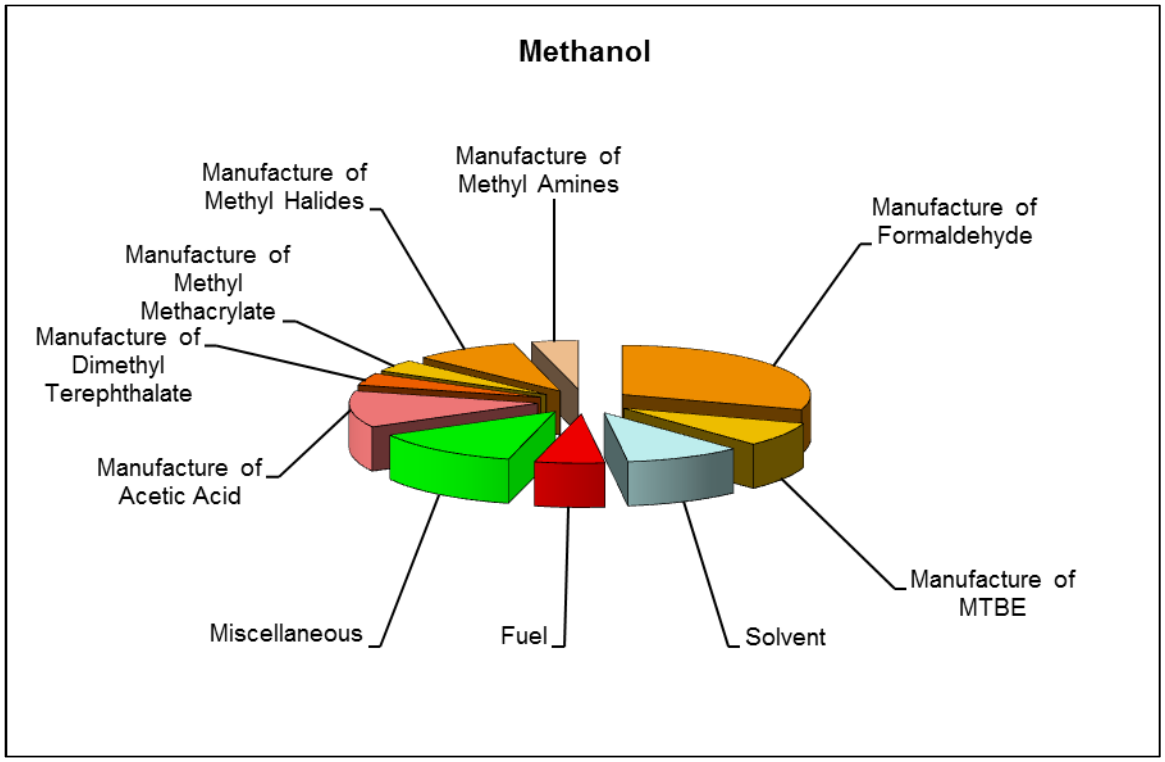

Figure 1: Major Uses of Methanol

2.4 Sources and Emissions

Methanol occurs naturally in humans, animals and plants. It is a natural constituent of blood, urine, saliva and expired air, and has also been found in mother's milk. Humans have a background body burden of 0.5 mg/kg body weight. Natural emission sources of methanol include volcanic gasses, vegetation, microbes, and insects (WHO 1997).

Given the high production volume, widespread use and physical and chemical properties of methanol, there is a very high potential for methanol to be released to the environment, principally to air (U.S. EPA 1976). Emissions of methanol primarily occur from miscellaneous solvent usage, methanol production, end-product manufacturing, and bulk storage and handling losses.

In an oilfield setting, emissions of methanol can occur through handling and storage of methanol, leakage from equipment that uses methanol (e.g., wellhead equipment for methanol injection for hydrate suppression), or through the failure of pipelines or pressure vessels undergoing hydrostatic testing with a methanol solution.

Methanol is included in the National Pollutant Release Inventory, with 13,000 tonnes released to air, 2300 tonnes to water and 85 tonnes to land in 2013. The main industry sectors reporting methanol releases are pulp and paper, chemical manufacture, oil & gas, and waste treatment. Approximately 28,000 tonnes were disposed, mostly via underground injection and mainly by the oil and gas sector (Environment Canada 2013)

2.5 Distribution in the Environment

Methanol can be present in air, water, and soil, both naturally and as a result of anthropogenic activities. In addition, methanol is present naturally in some foods. Methanol can also be present in consumer products.

Levels in Air

Levels of methanol in air well away from urban centers are generally low. Cavanaugh *et al.* (1969) reported the combined methanol/ethanol concentration in arctic air at Point Barrow, Alaska to be in the range 0.65-1.8 µg/m^3. The mean methanol concentration at two remote Arizona locations was 3 µg/m^3 (Snider and Dawson 1985). Concentrations in urban air are higher, and reported ranges include:

- 10.5-131 µg/m^3 (multiple locations, Graedel *et al*. 1986);
- 10 µg/m^3 (Tucson, Arizona, USA; Snider and Dawson 1985);
- 5-30 µg/m^3 (Stockholm, Sweden; Jonsson *et al.* 1985);
- 0.59-94 µg/m^3 (dense traffic sites in Stockholm, Sweden; Jonsson *et al.* 1985);
- 6-60 µg/m^3 (52 samples from Boston, Houston, and Lima, Ohio, USA; U.S. EPA 1993)

Methanol has been identified in exhausts from both gasoline and diesel engines and in tobacco smoke (WHO 1997).

Levels in Soil and Water

In Alberta, methanol spills and releases have been reported to Alberta Environment at concentrations up to 200 000 mg/kg in soil (G. Dinwoodie, personal communication).

In a 1982 assessment of urban and rural ambient concentrations in Arizona, methanol was detected at a mean level of 0.022 mg/L in rainwater collected during a thunderstorm (Snider and Dawson 1985). Methanol at levels of 17-80 mg/L (17-80 ppm) was detected in wastewater effluents from a specialty chemicals manufacturing facility in Massachusetts, USA, but none was detected in associated river water or sediments (Jungclaus et al. 1978). A concentration of 42.4 mg/L was found in a leachate from the Love Canal in Niagara Falls, New York (Venkataraman et al. 1984). Methanol at a level of 1,050 mg/L was detected in condensate waters discharged from a coal gasification plant in North Dakota, USA (Mohr and King 1985).

Levels in Food

Dietary methanol can arise in large part from fresh fruits and vegetables where it occurs as the free alcohol, methyl esters of fatty acids or the methoxy group on polysaccharides such as pectin. Reported values of the methanol content of fresh and canned fruit juices varies considerably and may range from 1-640 mg/L with an average of 140 mg/L (WHO 1997).

Methanol was found at levels of 6-27 mg/L in beer, 96-321 mg/L in wines, and 10-220 mg/L in distilled spirits (Greizerstein 1981). Fermented distilled beverages can contain high levels of methanol, with some spirits having as much as 1,500 mg/L (Francot and Geoffroy 1956). The methanol content in bourbon was reported to be 40-55 mg/L (Majchrowicz and Mendelson 1971). The presence of methanol in distilled spirits is directly linked to the pectin content of the raw

materials. During the process of making fruit spirits, pectic substances contained in different parts of the fruit undergo degradation by pectin methylases, which can lead to the formation of significant quantities of methanol (Bindler *et al.* 1988).

Humans can also ingest varying amounts of methanol in foods and/or drugs isolated or recrystallized from methanol. Methanol is used as an extraction solvent for spice oleoresins and hops (Lewis 1989). Additionally, certain foods and drugs, consumed or administered as their methyl ester, can release methanol during their metabolism and excretion. For example, 10% of the sweetening agent aspartame (L-aspartyl-L- phenylalanine methyl ester) hydrolyzes in the gastrointestinal tract to become free methanol. Artificially sweetened carbonated beverages contain about 555 mg aspartame/L (WHO 1997), equivalent to approximately 56 mg methanol per L. However, the amount of methanol present in an average serving of beverage sweetened by aspartame alone is considerably less than in the same volume of many fruit and vegetable juices. For instance, tomato juice will result in 6 times the amount of methanol exposure than consumption of an equivalent volume of aspartame sweetened beverage (Wucherpfennig *et al.* 1983).

Occurrence in Consumer Products

Methanol is a constituent of a large number of commercially available solvents and consumer products including paints, shellacs, varnishes, paint thinners, cleansing solutions, antifreeze solutions, automotive windshield washer fluids and deicers, duplicating fluids, denaturant for ethanol, and in hobby and craft adhesives. Potential uses of large quantities of methanol include direct use as a fuel, in gasoline blends or as a gasoline extender. Methanol has been identified in exhausts from both gasoline and diesel engines and in tobacco smoke.

2.6 Human Exposure

Methanol occurs naturally in humans, animals, and plants. It is a natural constituent in blood, urine, saliva, and expired air. Sedivec et al. (1981) reported a mean blood methanol level of 0.73 mg/L in unexposed individuals. The U.S. EPA (2013) combined the results of six studies to calculate a mean and standard deviation for the concentration of methanol in human blood of 1.36 mg/L and 0.77 mg/L, respectively. Eriksen and Kulkarni (1963) reported a range of 0.06 to 0.32 mg/m^3 in expired air.

The two most important sources of background body burdens for methanol and formate (a metabolic product of methanol, see Section 6.1) are diet and metabolic processes. Methanol is available in the diet principally from fresh fruits and vegetables, fruit juices, fermented beverages, and diet foods (principally soft drinks). U.S. EPA, (1977) suggest that the average intake of methanol from natural sources would be considerably less than 10 mg methanol/day. However, consumption of a moderate amount of fruit juices and/or aspartame-containing beverages would significantly increase this amount. If aspartame were used to replace all sucrose in the diet, its average daily ingestion would be 7.5-8.5 mg/kg which would be the equivalent to 0.75-0.85 mg methanol/kg (WHO 1997).

The U.K. Food Standards Agency estimates that endogenous methanol production ranges from 300 to 600 mg/day (Lindinger *et al.* 1997) (4.3 to 8.6 mg/kg-day) and that diet can contribute up to an additional 1,000 mg/day (14.3 mg/kg-day), principally from fruits and vegetables (COT,

2011). Thus the upper bound of the combined endogenous and dietary exposures estimated in the U.K. is 23 mg/kg-day. This is significantly greater than the tolerable daily intake (TDI) (2 mg/kg-day, see Section 6.6.3).

Exposures to methanol can occur in occupational settings through inhalation or dermal contact. Many national occupational health exposure limits suggest that workers are protected from any adverse effects if exposures do not exceed a time-weighted average of 260 mg/m^3 (200 ppm) methanol for any 8-h day and for a 40-h working week. Current general population exposures through air are typically 10,000 times lower than occupational limits. The general population is exposed to methanol in air at concentrations ranging from less than 0.001 mg/m^3 in rural air to nearly 0.04 mg/m^3 in urban air (WHO 1997).

If the projected use of methanol as an alternate fuel or in admixture with fuels increases significantly, it can be expected that there will be a widespread increase in the exposure of the general population to methanol via inhalation of vapours from methanol-fuelled vehicles and/or siphoning or percutaneous absorption of methanol fuels or blends (WHO 1997).

Based on the above information, it is clear that for a member of the general population, the primary source of methanol intake is via food. It is also clear that the daily intake of methanol will vary significantly with dietary choices, and will depend strongly on the consumption of fruit and fruit juices, as well as on consumption of the sweetener aspartame. Replacing all sugar in the diet could potentially result in an exposure to methanol several times the TDI.

The guidelines in this document require a value for estimated daily intake (EDI) which is defined at the total dose of a chemical to which an average person is exposed in the absence of any sources of contaminant. The EDI for some individuals may exceed the TDI. Where the EDI exceeds the TDI it is not possible to calculate certain guideline values since the acceptable dose for the chemical is already exceeded by the background exposure. For the purposes of setting guidelines for methanol, the EDI was set at 80% of the TDI, or 1.6 mg/kg of body weight (bw) per day. The rationale for this is that for a person receiving a methanol exposure through food of 80% or more of the TDI an additional 20% of the TDI is unlikely to have a significant incremental effect. Thus, the EDI used in this report is 1.6 mg/kg bw per day.

Methanol is not reported to be present in uncontaminated soil and accordingly the background soil concentration (BSC) is assumed to be zero.

The concentration of methanol in ambient air is assumed to be 0.04 mg/m^3 based on the WHO value for urban air reported above.

2.7 Existing Criteria, Guidelines and Standards

Canadian National

No soil or water quality guidelines for methanol are included in CCME (1999 and updates). Health Canada (2007) does not include methanol in its "Guidelines for Canadian Drinking Water Quality", and does not publish a Tolerable Daily Intake or Tolerable Concentration for methanol (Health Canada 2004)

Canadian Provincial
Ontario (OMEE 1994) has set an Interim Provincial Water Quality Objective for methanol of 0.2 mg/L, protective of aquatic life and recreational uses. Alberta (AENV 2010) has established soil and groundwater quality guidelines for methanol.

U.S. Federal
The U.S. EPA (2002, 2004a) does not publish a water quality guideline for methanol protective of aquatic life, or a Maximum Contaminant Level (MCL) for methanol in drinking water. Methanol is not included in the list of chemicals for which the U.S. EPA publishes Ecological Soil Screening Levels (EcoSSLs).

U.S. State
No criteria, guidelines, or standards were found for methanol in a limited search of U.S. state information.

Europe
The Dutch Ministry of the Environment (VROM 2000) has published "Indicative Levels for Serious Contamination" for methanol of 24 mg/L for groundwater and 30 mg/kg for soil. No other European methanol guidelines for soil or groundwater were found.

Australia and New Zealand
Australia and New Zealand have a collaborative set of water quality guidelines protective of aquatic uses (ANZECC 2000). These guidelines do not include values for methanol. No Australian drinking water guideline has been set for methanol (NHMRC 1996).

Global
The World Health Organization (WHO 2004) does not include methanol in its "Guidelines for Drinking Water Quality, Third Edition".

Occupational Exposure Limit
Many jurisdictions have published occupational health exposure limits. WHO (1997) indicate that workers are unlikely to experience any adverse effects if exposures do not exceed a time-weighted average of 260 mg/m3 (200 ppm) methanol for any 8-h day and for a 40-h working week.

3. ENVIRONMENTAL FATE AND BEHAVIOUR

3.1 Adsorption and Mobility

Methanol has negative log octanol-water (log K_{ow}) and log organic carbon-water (log K_{oc}) partition coefficients (-0.73 and -0.57, respectively, Table 2). Accordingly, sorption of methanol to organic carbon in soil will be minor, and methanol will tend to remain in soil pore water. The mobility of methanol in the subsurface will not be significantly limited by adsorption.

3.2 Aqueous-Phase Solubility

Methanol is miscible with water (Table 2). Accordingly, its mobility in the subsurface will not be limited by solubility.

3.3 Leaching and Lateral Movement

As noted in the two Sections above, the movement of methanol in the subsurface will not be limited by either adsorption or solubility. Consequently, leaching and lateral movement will be potentially significant factors in the subsurface transport of methanol. The hydrogeological retardation factor is the ratio of the rate at which groundwater moves divided by the rate at which a given contaminant in groundwater can be expected to move. If standard (CCME 2006) properties for coarse and fine soils are assumed, then retardation factors of 1.006 and 1.004 can be calculated for coarse and fine soils, respectively, indicating that the movement of methanol will not be significantly retarded relative to groundwater movement.

API (1994), confirmed the lack of methanol retardation in an aquifer study where an introduced methanol plume was found to move at the same rate as a chloride plume.

3.4 Biodegradation

Methanol has been shown to degrade rapidly under favourable conditions by a number of researchers (Appendix 1). However, in real environmental settings, degradation can be much slower than in laboratory microcosms due to factors including limited supplies of oxygen and/or other terminal electron acceptors, limited availability of nutrients, and lower temperatures. Thus, degradation rates from field studies typically have more environmental relevance than many laboratory microcosm studies.

Definitive Groundwater Study

One field study was available which gave information relevant to determining a degradation rate for methanol in groundwater. API (1994) injected gasoline, methyl tertiary butyl ether (MTBE) and methanol into the shallow sand aquifer at Canadian Forces Base Borden in Ontario. Solute movement and remaining mass were monitored for a period of 500 days via an extensive series of multi-level samplers. Removal of methanol from the aquifer was complete after 400 days. The initial total mass of methanol measured in the aquifer was approximately 14 kg. The total mass was reduced to 7 kg after approximately 245 days, and therefore 245 days is taken as an approximation of the half-life of methanol in groundwater (Appendix 1). This degradation rate was adopted for guideline development in this document (Appendix 7). Aquifer conditions in the injection zone prior to the experiment indicated a low background dissolved oxygen of approximately 2 mg/L. Measurements taken during the experiment indicated that initial methanol biodegradation was aerobic. Once oxygen was depleted in the plume, degradation proceeded by anaerobic pathways.

Other Degradation Studies and Data
Howard *et al.* (1991) quote the half-life of methanol in soil, groundwater, and surface water as being in the range 1-7 days (Appendix 1).

Methanol has been shown to degrade relatively rapidly in aerobic and anaerobic sludge systems. Available data have been summarized by Verschueren (2001) and are reproduced in Appendix 1. Figure 2 offers a graphical representation of the methanol degradation data, and shows that in the majority of tests, 50-100% of the methanol in a test system is biodegraded within 5-20 days. However, biodegradation data from aerobic sludges may have little relevance in predicting the biodegradation of methanol in soil and groundwater.

No data were available for methanol biodegradation in soils at natural moisture contents, but low concentrations of methanol (0.1 mg/L) in a soil water suspension were shown to degrade by 53% in 5 days under aerobic conditions, and only slightly less (46%) under anaerobic conditions (Appendix 1).

A concentration of 800 mg/L methanol was found to halve the oxidation of ammonia by *Nitrosomas* bacteria (i.e., the 50% inhibition concentration, or IC_{50} was 800 mg/L). However, bacterial oxygen consumption was much more robust, with an IC_{50} of 72,000 – 80,000 mg/L (Appendix 1).

The above data demonstrate that methanol will degrade rapidly in the presence of appropriate bacterial cultures and excess oxygen or other electron acceptors. Thus it may reasonably be anticipated that methanol will degrade rapidly in aerobic surface water or surficial soils. However, groundwater conditions can be very different, and in particular electron acceptors may be limited.

3.5 Volatilization

Volatilization potential is commonly expressed using the vapor pressure and the Henry's law constant of a compound. The Henry's law constant is the equilibrium ratio of the partial pressure in the gas phase to the concentration in the aqueous phase. This value is closely related to the vapour pressure of the pure compound but is also dependent on its aqueous solubility and molecular weight and, therefore, can be used to make a more accurate prediction of volatility than one based on solely on vapour pressure.

Lyman *et al.* (1982) used Henry's law constants to classify volatilization potential as follows:

- values less than 10^{-7} atm.m^3/mol indicate that the substance is less volatile than water and can be considered essentially non-volatile;
- values between 10^{-7} and 10^{-5} atm.m^3/mol indicate that the substance may volatilize slowly but the compound will still tend to partition into the aqueous phase;
- values between 10^{-5} and 10^{-3} atm.m^3/mol indicate that volatilization is significant; and,
- values greater than 10^{-3} atm.m^3/mol indicate that the majority of the mass of the compound will tend to partition into the gas phase.

The Henry's law constant of methanol is $4.6x10^{-6}$ atm.m^3/mol (Table 2). Accordingly, by the above definition, methanol may volatilize slowly from an aqueous solution, but will still tend to partition into the aqueous phase.

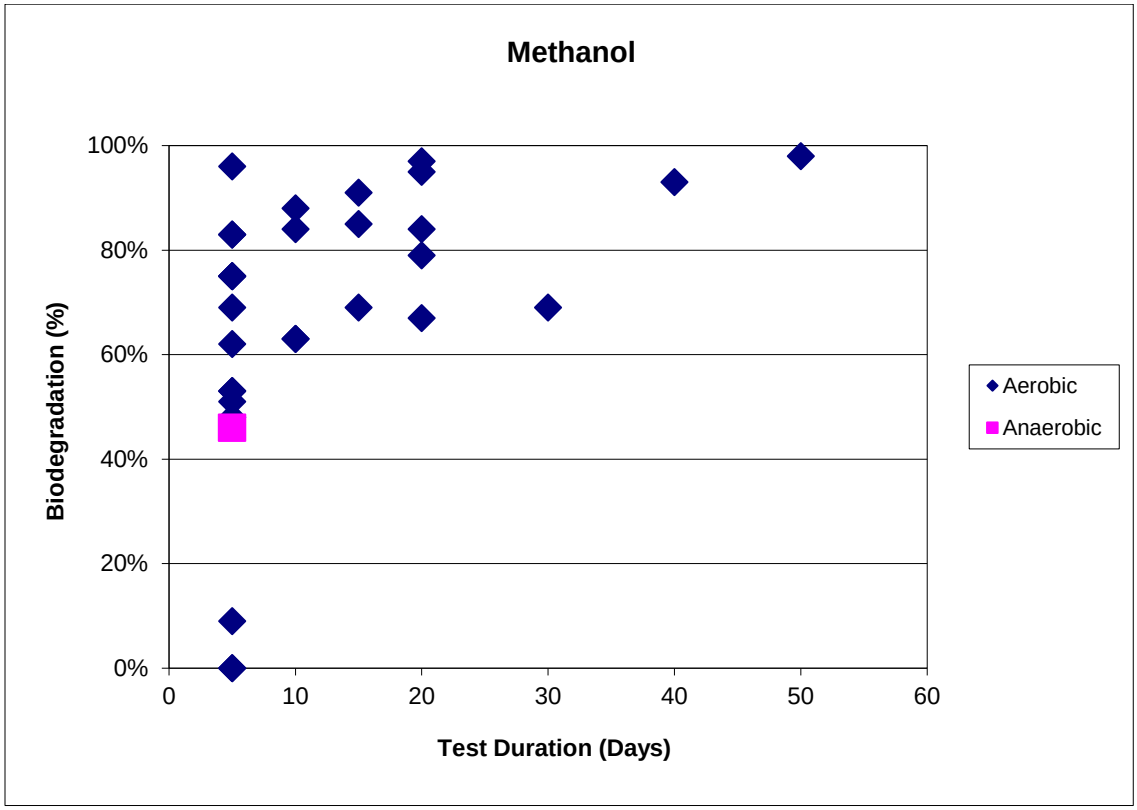

Figure 2: Methanol Biodegradation as a Function of Test Duration

3.6 Photolysis

Methanol degradation in the atmosphere can occur through reaction with photochemically-produced OH radicals (Kwok and Atkinson 1995; Grosjean 1997). The half-life of methanol in the atmosphere is estimated to be 17 to 18 days (OECD 2004; HSDB 2012).

4. BEHAVIOUR AND EFFECTS IN TERRESTRIAL BIOTA

4.1 Terrestrial Plants

Seven studies were found in the existing literature that investigated the toxicity of methanol to seven species of terrestrial plants: common onion *(Allium cepa),* lettuce *(Lactuca sativa),* common camellia *(Camellia japonica),* cotton *(Gossypium hirsutum),* potato *(Solanum tuberosum),* soybean *(Glycine max), and* wild carrot *(Daucus carota)* (Appendix 2). However, none of these studies were conducted using soil as a medium, but rather used plants grown in water or on agar plates, or

applied methanol directly to specific plant organs or cells. As such, none of these data are relevant for developing soil quality guidelines.

Accordingly, definitive (14 or 21 day) growth tests were commissioned (Stantec 2006) for three plant species, alfalfa (*Medicago sativ*a), barley (*Hordeum vulgare*), and northern wheatgrass (*Elymus lanceolatus*). Environment Canada toxicity test protocols (or the most recent available Environment Canada draft protocol, as appropriate) were used for this work with minor modifications to minimize the volatile losses of methanol (Stantec 2006). A full report on these tests is available at www.ptac.org, and the results are summarized in Appendix 2. EC_{25} values for various endpoints for these three species ranged from 1,808 mg/kg to 12,202 mg/kg.

4.2 Soil Invertebrates

No studies on the toxicity of methanol to terrestrial invertebrates in soil were found in the existing literature and therefore new tests were commissioned. One other study was found on the toxicity of methanol to soil invertebrates in other media. In a 48 hour filter paper test with methanol and *Eisenia fetida*, the LC_{50} was found to be >1,000 $\mu g/cm^2$ (Appendix 3). This study was not conducted in soil and is not relevant for developing soil quality guidelines.

Reproduction tests were commissioned (Stantec 2006) for two invertebrate species, the earthworm *Eisenia andrei*, and the springtail *Folsomia canadida*. Environment Canada toxicity test protocols (or the most recent available Environment Canada draft protocol, as appropriate) were used for this work with minor modifications to minimize the volatile losses of methanol (Stantec 2006). A full report on these tests is available at www.ptac.org, and the results are summarized in Appendix 3. EC_{25} values for reproduction endpoints for these two invertebrates ranged from 2842 mg/kg to 13,323 mg/kg.

4.3 Soil Microbial Processes

No information was available that directly considered the effect of methanol on soil microbial processes. However, information on the degradation of methanol presented in Section 2.3.4. indicates that bacterial ammonia oxidation in sludge by *Nitrosomas* bacteria is inhibited (IC_{50}) in sludge at 800 mg/L, and the IC_{50} for bacterial oxygen consumption in sludge has been reported to be in the range 72,000 to 80,000 mg/L.

5. BEHAVIOUR AND EFFECTS IN AQUATIC BIOTA

5.1 Freshwater Biota

5.1.1 Freshwater Aquatic Vertebrates

Aquatic toxicity data for freshwater vertebrates is provided in Appendix 4. Data points for 3 species [tilapia (*Orochromis mossambicus*), medaka *(Oryzias latipes)*, and Chum salmon (*Oncorhynchus keta*)] were retained. Effects endpoints ranged from 33.6 mg/L for tilapia growth to 5616 mg/L for medaka hatching success. One study had an unbounded no observable effects concentration (NOEC) of 7,910 for Chum salmon fertilization.

5.1.2 Freshwater Aquatic Invertebrates

Aquatic toxicity data for freshwater invertebrates is provided in Appendix 4 for 3 invertebrate species [Gekielte plate snail *(Planorbis carinatus)*, water flea *(Ceriodaphnia dubia)*, and midge *(Chironomus riparius)*]. All the studies generated unbounded NOECs, ranging from 79.1 for snail mortality to 10,253 mg/L for midge behaviour.

5.1.3 Freshwater Aquatic Plants and Algae

Toxicity data for aquatic plants and algae in Appendix 4 include data from 4 studies of green algae. Endpoints ranged from an IC10 of 369 mg/L to and IC10 of 1582 mg/L. The data included one study with an unbounded NOEC of 15 820 mg/L.

5.2 Marine Biota

The long-term dataset for marine taxa in Appendix 5 does not meet the data quantity requirements for either Type A, Type B-1, or Type B-2 guidelines, based on the lack of a temperate fish species. Both the fish species in Appendix 5 are classified as sub-tropical based on information in the database FishBase (FishBase 2014). Since the data requirements for developing a marine guideline are not met by the currently available long-term dataset for methanol, no long-term marine water quality guideline was calculated for methanol.

6. BEHAVIOUR AND EFFECTS IN HUMANS AND MAMMALIAN SPECIES

There is a large body of data concerning the mammalian toxicity of methanol. Drivers for research in recent years have included: i) the possibility of methanol being increasingly used as an automotive fuel, and the associated increase in inhalation exposure for the general population; and, ii) the observation that aspartame, a widely-used artificial sweetener, is hydrolyzed in the human gut to yield methanol.

The following reviews of the mammalian toxicology of methanol were consulted in the development of the summary that follows:

- *U.S. EPA IRIS database for Risk Assessment – Methanol. U.S. EPA (2014b).*
- *Toxicological Review of Methanol (Non-Cancer) in Support of Summary Information on the Integrated Risk Information System (IRIS) (U.S. EPA 2013).*
- *California EPA document developing maximum allowable dose levels (MADLs) for methanol. (CalEPA 2012).*
- *NTP-CERHR Expert Panel report on the reproductive and developmental toxicity of methanol. (CERHR 2004).*
- *Environmental Health Criteria 196—Methanol. World Health Organization. (WHO 1997).*
- *The toxicity of inhaled methanol vapors. In: Critical Reviews in Toxicology. (Kavet and Nauss 1990).*

No attempt is made here to include all the available toxicological data on methanol, but rather the main elements of methanol toxicity and the key studies are discussed below and summarized in Appendix 6.

6.1 Toxicokinetics

Methanol occurs naturally in the human body as a product of metabolism and through intake of fruits, vegetables, and alcoholic beverages (CERHR 2004). The absorption, excretion, and metabolism of methanol are well understood, and are summarized in U.S. EPA (2013) based on previous reviews including CERHR (2004), IPCS (1997), U.S. EPA (1996), Kavet and Nauss (1990), HEI (1987), and Tephly and McMartin (1984). The following summary is primarily based on information in U.S. EPA (2013).

Methanol is absorbed rapidly following oral, inhalation, or dermal exposure and distributes readily and uniformly to all organs and tissues in direct relation to their water content.

At doses that do not saturate metabolic pathways, a small percentage of methanol is excreted directly in urine. Because of the high blood:air partition coefficient for methanol and rapid metabolism in all species studied, the bulk of clearance occurs by metabolism, though exhalation and urinary clearance become more significant when doses or exposures are sufficiently high to saturate metabolism.

The primary route of methanol elimination in mammals is through a series of oxidation reactions that form formaldehyde, formate, and carbon dioxide. Methanol is converted to formaldehyde by alcohol dehydrogenase-1 (ADH1) in primates and by catalase (CAT) and ADH1 in rodents. Although the first step of metabolism occurs through different pathways in rodents and nonhuman primates, Kavet and Nauss (1990) report that the reaction proceeds at similar rates. In all species, formaldehyde is rapidly converted to formate, with the half-life for formaldehyde being ~1 minute. The mechanism and rate of the metabolism of formate to carbon dioxide differs significantly between rodents and primates. Rodents are able to metabolize formate both through a folate-dependent enzyme system and through a CAT-peroxide system. In primates, however, formate metabolism occurs solely through a folate-dependent pathway. Accordingly, primates do not have the same capacity as rodents to clear formate, and consequently are more sensitive to metabolic acidosis following methanol poisoning.

6.3 Acute Toxicity

6.2.1 Human Studies

There is an extensive library of case reports that have documented the consequences of acute accidental/intentional methanol poisoning, typically via oral exposure. Typical symptoms include blurred vision and bilateral or unilateral blindness, convulsions, tremors, coma, nausea, headache, dizziness, abdominal pain, diminished motor skills, acidosis, dyspnea, behavioural and/or emotional deficits, and speech impediments. Typically, the most severe symptoms and the poorest health outcomes were correlated to patients in a metabolic acidotic state (blood pH <7.0). In cases of human methanol poisoning, the minimum lethal dose is in the range 300 to 1,000 mg/kg bw (CERHR 2004).

Kavet and Nauss (1990) indicate that acute toxicity to humans from inhalation of methanol vapours follows a very similar clinical pattern to that observed for oral exposure. Two controlled studies have evaluated humans for neurobehavioral function following exposure to methanol vapours in a controlled setting. Chuwers et al. (1995) exposed 12 healthy men to 250 mg/m^3 methanol for 75 minutes while Cook et al. (1991) exposed 15 men and 11 women to 262 mg/m^3 methanol for 4 hours. These two studies were interpreted by U.S. EPA (2013) to correspond to exposures below the threshold for substantial neurological effects.

6.2.2 Animal Studies

Although there are few studies that have examined the short-term toxic effects of methanol via the oral route, a number of median lethal dose (LD_{50}) values have been published for the compound. As summarized in Lewis (1989), these include 5,628 mg/kg bw in rats, 7,300 mg/kg bw in mice, and 7,000 mg/kg bw in monkeys.

The database of acute effects from animal studies via inhalation exposure is less extensive, but includes a 4-hour median lethal concentration (LC_{50}) for methanol in rats of 64 000 ppm (84,000 mg/m^3) (Lewis, 1989). NEDO (1987) exposed monkeys (*M. fascicularis*), to methanol by inhalation for a range of exposure durations from 5 to 20 days. Details are limited, but an assessment by U.S. EPA (2013) suggests that clinical signs of toxicity were apparent in animals exposed to 5,000 ppm (6,500 mg/m^3) or higher concentrations of methanol.

6.3 Subchronic and Chronic Toxicity

6.3.1 Oral Studies

The U.S. EPA (1986) conducted a sub-chronic oral study on the toxicity of methanol to rats. Sprague-Dawley rats were gavaged daily with 0, 100, 500, or 2,500 mg/kg bw/day of methanol for 90 days. At the highest dose, effects were noted on liver function, as evidenced by elevated levels of SGPT, SAP, and increased, but not statistically significant, liver weights in both male and female rats. Elevated levels of the enzymes SGPT and SAP in blood are indicators of liver damage. These data suggest possible treatment-related effects in rats dosed with 2,500 mg methanol/kg bw/day despite the absence of supportive histopathologic lesions in the liver. Based on these findings, 500 mg/kg/day of methanol is considered to be the NOEL from this rat study.

The European Ramazzini Foundation (ERF) conducted a chronic duration rat study that was reported by Soffritti et al. (2002) and by Cruzan (2009). In this study, methanol was provided to 100 Sprague-Dawley rats/sex/group ad libitum in drinking water at concentrations of 0, 500, 5,000, and 20,000 ppm (v/v). The animals were 8 weeks old at the beginning of the study. Rats were exposed for up to 104 weeks, then maintained until they died naturally. Overall, there was no pattern of compound-related clinical signs of toxicity, and the available data did not provide any indication that the control group was not concurrent with the treated group (Cruzan 2009).

6.3.2 Inhalation Studies

A number of experimental studies have examined the effects of subchronic exposure to methanol via inhalation. Selected studies are summarized below.

Sayers *et al.* (1944) repeatedly exposed (8 times daily for 3 minutes/exposure) two male dogs to 10,000 ppm (13,000 mg/m^3) methanol for 100 days. There were no clinical signs of toxicity.

White *et al.* (1983) exposed 4 male Sprague-Dawley rats/group, 6 hours/day, 5 days/week to 0, 200, 2,000, or 10,000 ppm (0, 260, 2,600, or 13,000 mg/m^3) methanol for periods of 1, 2, 4, and 6 weeks. There were no clinical signs of toxicity among the groups.

Andrews *et al.* (1987) carried out a study of methanol inhalation in 5 Sprague-Dawley rats/sex/group and 3 *M. fascicularis* monkeys/sex/group, 6 hours/day, 5 days/week, to 0, 500, 2,000, or 5,000 ppm (0, 660, 2,600, or 6,600 mg/m^3) methanol for 4 weeks. All animals survived to term with no clinical signs of toxicity among the monkeys and only a few signs of irritation to the eyes and nose among the rats.

Poon *et al.* (1994), exposed 10 Sprague-Dawley rats/sex/group via inhalation, 6 hours/day, 5 days/week to 0, 300, or 3,000 ppm (0, 400, or 4,000 mg/m^3) methanol for 4 weeks. All animals survived to term, and there were no clinical signs of toxicity among the groups.

Poon *et al.* (1995) exposed 15 Sprague-Dawley rats/sex/group, 6 hours/day, 5 days/week for 4 weeks to 0 or 2,500 ppm (0 or 3,300 mg/m^3) methanol. Few if any of the monitored parameters showed any differences between controls and those animals exposed to methanol. However, two male rats had collapsed right eyes, and there was a reduction in relative spleen weight in females exposed to methanol.

One study (NEDO 1987) examined the effects on several species of chronic exposure to methanol via inhalation. NEDO (1987) included the results of experiments on i) monkeys exposed for up to 3 years, ii) rats and mice exposed for 12 months, iii) mice exposed for 18 months, and iv) rats exposed for 2 years. These are unpublished studies but were externally peer reviewed by EPA in 2009.

In the monkey experiment, 8 animals (sex unspecified) were exposed to 10, 100, or 1,000 ppm methanol (13, 130, 1,300 mg/m^3), 21 hours/day, for 7 months (2 animals), 19 months, (3 animals), or 29 months (3 animals). There was no indication in the NEDO (1987) report that this study employed a concurrent control group. The U.S. EPA (2013) interpretation of this study highlighted possible hepatic effects with a lowest-observed-adverse-effect level (LOAEL) of 1,000 ppm (1,300

mg/m^3), and possible dose-dependent renal effects with a LOAEL of 100 ppm (130 mg/m^3). However the confidence in both these findings is low based on the lack of a documented control and poor experiments detail available in the paper.

In the 12 month rat and mouse experiments, 20 F344 rats/sex/group or 30 B6C3F1 mice/sex/group were exposed to 0, 10, 100, or 1,000 ppm (0, 13, 130, or 1,300 mg/m^3) methanol, approximately 20 hours/day, for a year. There were no clinical findings that U.S. EPA (2013) was able to attribute unequivocally to the methanol exposure.

In the 18 month mouse experiments, 52 male and 53 female B6C3F1 mice/group were exposed to 0, 10, 100, or 1,000 ppm (0, 13, 130, or 1,300 mg/m^3) methanol, approximately 20 hours/day, for 18 months. A few animals showed clinical signs of toxicity, but the incidence of these responses was not related to dose. High-concentration males had lower testis weights compared to control males.

In the 24 month rat experiments, 52 F344 rats/sex/group were exposed to 0, 10, 100, or 1,000 ppm (0, 13, 130, or 1,300 mg/m^3) methanol, approximately 19.5 hours/day, for 733-736 days (males) or 740-743 days (females). The authors reported that variations observed in urinary, hematology, and clinical chemistry parameters were not related to chemical exposure.

6.4 Reproduction and Developmental Toxicity

Many studies have been conducted to investigate the reproductive and developmental toxicity of methanol. The purpose of these studies was principally to determine if methanol has a similar toxicology profile to another widely studied teratogen, ethanol. Key studies are summarized below.

6.4.1 Oral Studies

Rogers *et al.* (1993) conducted a developmental toxicity study in which methanol in water was administered to pregnant female CD-1 mice via gavage on gestation day 6 to 15 (GD6–GD15). Eight test animals received 4 g/kg-day methanol given in 2 daily doses of 2g/kg; four controls received distilled water. The primary toxicological findings in the exposed animals were cleft palate, exencephaly, an increase in totally resorbed litters and a decrease in the number of live fetuses per litter. U.S. EPA (2013) notes that it is possible that these effects may have been caused or exacerbated by the high bolus dosing regimen employed. U.S. EPA (2013) also notes that the small number of animals in the control group relative to the test group limits the power of this study to detect treatment-related responses.

Sakanashi *et al.* (1996) tested the influence of dietary folic acid intake on various reproductive and developmental effects observed in CD-1 mice exposed to methanol using groups of mice on low, marginal, and sufficient folic acid diets. On GD6–GD15, pregnant mice in each of the diet groups were given 4.0 or 5.0 g/kg-day methanol by gavage. On GD18, mice were weighed and sacrificed. Similar to Rogers et al. (1993), Sakanashi et al. (1996) observed that an oral dose of 4-5 g/kg-day methanol during GD6-GD15 resulted in an increase in cleft palate in mice fed sufficient folic acid diets, as well as an increase in resorptions and a decrease in live fetuses per litter.

Fu *et al.* (1996) also tested the influence of dietary folic acid intake on reproductive and developmental effects observed in CD-1 mice exposed to methanol. This study was performed by the same laboratory and used a similar study design and dosing regimen as Sakanashi et al. (1996), but exposed the pregnant mice to only the higher 5.0 g/kg-day dose on GD6-GD10. These authors found that methanol exposure during GD6-GD10 appeared to have similar fetotoxic effects, including cleft palate, exencephaly, resorptions, and decrease in live fetuses, as the same level of methanol exposure administered during GD6-GD15 (Rogers *et al*. 1993; Sakanashi *et al*. 1996). This is consistent with the hypothesis made by Rogers et al. (1993) that the critical period for methanol-induced cleft palate and exencephaly in CD-1 mice is within GD6-GD10.

6.4.2 Inhalation Studies

Nelson *et al.* (1985) exposed 15 pregnant Sprague-Dawley rats/group to 0, 5000, 10 000, or 20000 ppm (0, 6,600, 13,000, or 26,000 mg/m^3) methanol for 7 hours/day. Exposures were conducted on GD1–GD19 in the two lower concentration groups and GD7-GD15 in the highest concentration group. Two groups of 15 control rats were exposed to air only. The maternal no-observed-adverse-effect-level (NOAEL) for this study was identified as 10,000 ppm (13,000 mg/m^3) (unsteady gait in dams during first few days of test). The fetal NOAEL for this study was identified as 5,000 ppm (6,600 mg/m^3). Fetal effects included skeletal malformations, including rudimentary and extra cervical ribs and malformations in brain development including exencephaly and encephaloceles.

NEDO (1987) exposed 36 pregnant females/group to 0, 200, 1,000, or 5,000 ppm (0, 260, 1,300, or 6,600 mg/kg) methanol vapour on GD7–GD17 for 22.7 hours/day. Contrary to the Nelson et al. (1985) report of a 10,000 ppm (13,000 mg/m^3) maternal NOAEL for this rat strain, reduced body weight gain and food and water intake during the first 7 days of exposure were reported for dams in the 5,000 ppm (6,600 mg/m^3) group during the prenatal portion of the study (NEDO 1987). However, it was not specified if these results were statistically significant. On GD20, 19-24 dams/group were sacrificed to evaluate reproductive and developmental parameters. The remaining 12 litters per group were allowed to develop and assessed at 8 weeks post-natal. The fetal NOAEL and LOAEL in this study were 1,000 ppm (1,300 mg/m^3) and 5,000 ppm (6,600 mg/^{3}), respectively, based on a critical effect of reduced brain, pituitary, thyroid, thymus and testis weights at 8 weeks post-natal.

NEDO (1987) also contains an account of a two-generation reproductive study that evaluated the effects of pre- and postnatal methanol exposure (20 hours/day) on reproductive and other organ systems of Sprague-Dawley rats. The F0 generation (30 males and 30 females per exposure group) was exposed to 0, 10, 100, and 1,000 ppm (13, 130, 1,300 mg/m^3) from 8 weeks old to the end of mating (males) or to the end of lactation period (females). The F1 generation was exposed to the same concentrations from birth to the end of mating (males) or to weaning of F2 pups 21 days after delivery (females). Males and females of the F2 generation were exposed from birth to 21 days old (one animal/sex/litter was exposed to 8 weeks of age). The fetal NOAEL and LOAEL for exposure from F1 birth to end of mating or weaning, and F2 birth to 8 weeks, as interpreted by U.S. EPA (2013), were 100 ppm (130 mg/m^3) and 1,000 ppm (1,300 mg/m^3), based on a critical effect of reduced brain, pituitary and thymus weight. In a follow-up study of brain weights in the F1 generation, the fetal NOAEL and LOAEL for exposure from GD1 through the F1 generation, as interpreted by U.S. EPA (2013), were 500 ppm (660 mg/m^3) and 1,000 ppm (1,300 mg/m^3), respectively, based on a critical effect of reduced brain and cerebrum weight in males.

Rogers et al. (1993) evaluated development toxicity in pregnant female CD-1 mice exposed to air or 1,000, 2,000, 5,000, 7,500, 10,000, or 15,000 ppm (1,300, 2,600, 6,600, 9,900, 13,000, or 20,000 mg/m^3) methanol vapour in a chamber for 7 hours/day on GD6-GD15. The numbers of mice exposed at each dose were 114, 40, 80, 79, 30, 30, and 44, respectively. During chamber exposures to air or methanol, the mice had access to water but not food. In order to determine the effects of the chamber exposure conditions, an additional 88 control mice were not handled and remained in their cages; 30 control mice were not handled but were food deprived for 7 hours/day on GD6-GD15. No methanol-related maternal toxicity was noted. The NOAEL and LOAEL for fetal effects were 1,000 ppm (1,300 mg/m^3) and 2,000 ppm (2,600 mg/m^3), respectively, based on increased incidence of extra cervical ribs, cleft palate, exencephaly, reduced fetal weight, reduced pup survival, and delayed ossification.

6.5 Carcinogenicity and Genetic Toxicity

There have been no studies reported in the peer-reviewed literature on the potential carcinogenicity of methanol in either humans or laboratory animals (WHO 1997). However, unpublished reports from the New Energy Development Organization (NEDO 1987; Katoh 1989) in Japan included carcinogenicity studies on mice and rats exposed by inhalation to methanol vapours in chambers at up to 1,300 mg/m^3 for up to 24 months. No evidence of carcinogenicity was found in either species. It is unlikely that methanol is carcinogenic to mouse skin. In a dermal exposure study on mice with an exposure period of 50 weeks and observation for lifetime, no indication of methanol-related carcinogenicity was reported (Lijinsky *et al.* 1991). While the database on carcinogenicity is extremely limited, no evidence suggesting that methanol is carcinogenic to animals or humans was found.

A number of *in-vitro* and *in-vivo* studies have investigated the genetic toxicity of methanol.

Endpoints studied in *in-vitro* tests include:

- bacterial reverse mutation assays (Standard Ames assay)***;***
- DNA repair test in the bacterium *E. coli;*
- chromosomal malsegregation in the fungus *Aspergillus nidulans;*
- gene mutation in the yeast *Schizosaccharomyces pombe;*
- mutagenicity test in the fungus *Neurospora crassa;*
- sister chromatid exchanges in Chinese hamster cells;
- mutation frequency in mouse lymphoma cells;
- cell transformation in Syrian hamster embryo cells; and,
- cell transformation in rat embryo cell.

Results from the *in-vitro* tests were negative, with a the exception of two tests (WHO 1997). Methanol (6% v/v) induced 3.02% chromosomal malsegregation in *Aspergillus nidulans*. Mutation frequency in mouse lymphoma cells increased in the presence of methanol and S-9.

In-vivo tests have considered a range of genotoxicity endpoints in mice exposed to methanol via oral, inhalation, and intraperitoneal routes. As with the *in-vitro* tests, the majority of the results were negative, but some positive results were obtained (WHO 1997).

WHO (1997) considers that the structure of methanol (by analogy with ethanol) does not suggest that it would be genotoxic. Cruzan (2009) reviewed the available information on the carcinogenicity of methanol, and concluded that methanol was unlikely to be carcinogenic in humans. Overall, the weight of evidence appears to suggest that methanol is likely not genotoxic.

6.6 Dose-Response Assessments

Health Canada (2004) has not reviewed the toxicity of methanol, or developed a tolerable daily intake or tolerable concentration for methanol.

The United States Environmental Protection Agency (U.S. EPA 2014b) has developed an inhalation reference concentration and an oral reference dose for methanol. The U.S. EPA (2013) approach is discussed in Sections 6.6.1 and 6.6.2 below.

Additionally, the California Environmental Protection Agency (CalEPA 2012) has developed maximum allowable dose levels (MADLs) for methanol for inhalation and oral exposure. The findings of this document are also discussed in Sections 6.6.1 and 6.6.2 below.

6.6.1 Inhalation

The previous U.S. EPA dose response assessment for methanol (oral exposure only) involved applying uncertainty factors to the NOAEL for the critical effect. The approach taken in U.S. EPA (2013) to develop an inhalation reference concentration (RfC) is more sophisticated. The U.S. EPA (2013) calculated a total of four candidate RfC values based on the following study, endpoint, and benchmark response (BMR) combinations:

- Rogers et al. (1993), mouse cervical rib, 10% BMR.
- Rogers et al. (1993), mouse cervical rib, 5% BMR.
- NEDO (1987), rat fetal brain weight, 5% BMR.
- NEDO (1987), rat fetal brain weight, 1 standard deviation (1SD) change from mean.

Three main steps were involved for each of the four combinations noted above. Firstly the applied dose in the principal study was converted to an internal dose metric – the concentration of methanol in blood was selected - using a physiologically based pharmokinetic (PBPK) model for methanol developed by the U.S. EPA. The measure of dose used for the mouse cervical rib endpoint was the maximum blood concentration of methanol, since the gestational window of susceptibility for this effect is thought to be small. The measure of dose used for the fetal brain weight effect was the area under the curve (AUC) which represents the cumulative product of concentration and time for methanol in the blood. This measure was selected because data indicate that exposure duration is important for this effect. Next the lower bound confidence limit on the 5% or 10% benchmark dose (BMDL), or the measure of dose required to cause a 1SD change from the mean value was calculated from the critical study. The BMDL approach is preferred over using the NOAEL as the point of departure since it is independent of the arbitrary experimental exposure levels. Finally,

the BMDL values were converted to human equivalent concentrations (HECs) via the use of a PBPK model parameterized for humans.

A composite uncertainty factor of 100-fold (10-fold for inter-individual variation, 3-fold for residual toxicodynamic differences associated with animal-to-human extrapolation, and 3-fold for database uncertainty) was used in the calculation of each of the four candidate RfC values. The lowest of the candidate RfC values was 17.8 mg/m^3 from the rat brain weight endpoint at 1SD from the mean. This value was rounded to 1 significant figure to give an RfC of 20 mg/m^3.

The California EPA (CalEPA, 2012) developed an MADL for methanol for the inhalation route. They selected the inhalation experiments in Rogers et al. (1993) as the principal study, and identified increased incidence of cervical ribs as the critical effect with a NOEL of 1,000 ppm. They converted this concentration into units of mg/m^3 by using a conversion factor of 1.33 mg/m^3 per ppm, corrected for the 7 hour a day exposure by applying a factor of 7 h/24 h, calculated the NOEL mouse dose using an inhalation rate of 0.063 m^3/day and a body weight of 0.030 kg, to be 814.6 mg/kg/day. They then applied the body mass of a 58 kg woman and an uncertainty factor of 1,000 to calculate a MADL of 47,000 µg/day. Using CCME (2006) adult body weight and inhalation rate parameter values would allow an equivalent RfC/TC of 3.6 mg/m^3 to be calculated:

$$47\,000\,\frac{\mu g}{day} \times \frac{1\,mg}{1000\mu g} \times \frac{70.7\,kg}{58\,kg} \times \frac{1}{\frac{15.8 m^3}{day}} = 3.6 mg/m^3$$

6.6.2 Oral

The U.S. EPA (2013) noted limitations in the oral database for methanol, including limited reporting of non-cancer findings in the subchronic (U.S. EPA, 1986) and chronic studies (Soffritti *et al*. 2002) of rats and the high dose levels used in the two rodent developmental studies. Accordingly, U.S. EPA (2013) derived an RfD by using relevant inhalation data and route-to-route extrapolation with the aid of the EPA PBPK model. U.S. EPA (2013) commented that several factors supported the use of route-to-route extrapolation for methanol, including the following: the limited data for oral administration indicated similar effects as reported via inhalation exposure (e.g., the brain and fetal skeletal system are targets of toxicity); and methanol has been shown to be rapidly and well-absorbed by both the oral and inhalation routes of exposure (CERHR 2004; Kavet and Nauss 1990). Once absorbed, methanol distributes rapidly to all organs and tissues according to water content, regardless of route of exposure.

The approach taken by U.S. EPA (2013) to develop an RfD was to start from the same principal studies, critical effects, and BMDL values as were used in development of the four candidate RfC values. These BMDLs were then converted to equivalent human oral exposures using the EPA human PBPK model. The same overall uncertainty factor of 100 is used to calculate the candidate RfDs as was used in the RfC calculations. The lowest of the four candidate RfD values was 2 mg/kg-day for the mouse cervical rib endpoint at a BMR of 5%.

The California EPA (CalEPA 2012) developed an MADL for methanol for oral exposure. They selected the oral exposure experiments in Rogers et al. (1993) as the principal study, and identified decreased fetal weight, increased resorptions, decreased live fetuses, and an increased incidence of fetuses/litter with cleft palate or exencephaly as the critical effects. The unbounded LOEL for these effects was 4,000 mg/kg/day. They divided the unbounded LOEL by 10 to determine an NOEL of 400 mg/kg/day "for the purposes of assessment". They then applied the body mass of a 58 kg woman and an uncertainty factor of 1,000 to calculate an MADL of 23,000 µg/day. Applying the uncertainty factor of 1,000 directly to the NOEL of 400 mg/kg/day would allow an equivalent RfD/TDI of 0.4 mg/kg/day to be calculated.

6.6.3 Recommended TDI and TC for Guideline Calculation

Overall, the most appropriate values of TDI and TC for use in guideline derivation appear to be the values developed for an RfD and RfC by the U.S. EPA (2013), as follows:

- TDI = 2 mg/kg/day.
- TC = 20 mg/m^3.

The rationale for preferring the U.S. EPA values over the CalEPA values includes the following considerations:

- The U.S. EPA (2013) appears to have considered a wider range of studies.
- CalEPA (2012) appears not to have considered inhalation NOAELs from two aspects of the NEDO (1987) study that were lower than the NOEL selected from the Rogers et al. (1993) study.
- The CalEPA (2012) approach for oral exposure is based on an unbounded LOEL at a relatively high dose at which multiple adverse effects are seen. There is significant uncertainty in extrapolating from this high dose to estimate where the NOEL might occur.
- The U.S. EPA (2013) approach to using a PBPK model to estimate maximum blood methanol levels based on periodic exposure and adsorption and clearance rates appears superior to the CalEPA (2012) approach of a time-based amortization of the periodic exposure.
- The U.S. EPA (2013) BMDL approach to using a best fit to the whole dataset to estimate the threshold concentration for adverse effects appears superior to the CalEPA (2012) approach of using the NOEL directly, or estimating a NOEL from the LOEL.
- The U.S. EPA (2013) approach of calculating RfC values for multiple studies, critical effects and using different methodologies appears to be more robust than the CalEPA (2012) approach of selecting a single NOEL value as point of departure.

7. TOXICITY OF DEGRADATION PRODUCTS

In certain cases, organic compounds can have degradation products that are more toxic than the parent compound. Prudent management of such a parent compound should take into consideration the possibility of more toxic degradation products. A complete review of the toxicity of degradation products is outside the scope of the current study. However, it is worth noting that formaldehyde is a potential degradation product of methanol. Dutch environmental regulators (VROM 2000), provide "indicative levels for serious contamination" for formaldehyde in soil and

groundwater of 0.1 mg/kg and 0.05 mg/L, respectively. These values are 2-3 orders of magnitude lower than the corresponding values for methanol (30 mg/kg and 24 mg/L), indicating that the Dutch regulators consider formaldehyde significantly more toxic than methanol.

Environment Canada and Health Canada (EC and HC 2001) reviewed formaldehyde as a priority substance under the Canadian Environmental Protection Act (CEPA). Under the criteria provided by CEPA (Environment Canada 1999), it was found not likely to cause adverse effects to terrestrial or aquatic organisms. However, it was found to contribute to the photochemical formation of ground-level ozone and was considered to be a human carcinogen.

No attempt was made to incorporate possible formaldehyde toxicity in the guidelines for methanol. However, formaldehyde should always be analyzed at any site with a significant methanol release, and the results managed on a site-specific basis.

8. DATA ADEQUACY AND DATA GAPS

The available data for methanol were assessed against CCME (2006) requirements for developing soil and water quality guidelines.

8.1 Soil Quality Guidelines

Human Health Guidelines
Sufficient data are available to develop soil quality guidelines protective of human soil ingestion, indoor air inhalation, and potable groundwater, based on CCME (2006) requirements.

Ecological Guidelines
A battery of terrestrial toxicity tests was commissioned for this project and the results form an adequate database for guideline development for the soil eco-contact pathway based on CCME (2006) requirements.

None of the available data are suitable for calculating the nutrient and energy cycling check, and accordingly, this check was not calculated for methanol. A soil quality guideline can be calculated without this check.

Insufficient data exist to calculate the soil and food ingestion guideline. The CCME (2006) protocol for this guideline requires toxicity data from tests conducted on livestock species, and these data do not currently exist.

There are sufficient data to calculate the soil quality guideline protective of groundwater for freshwater aquatic life, based on CCME (2006) requirements.

8.2 Groundwater Quality Guidelines

Drinking Water
Sufficient data are available to develop a Source Guidance Value for Groundwater to use as a basis for the development of a soil quality guideline protective of potable groundwater.

Freshwater Aquatic Life
The freshwater aquatic life dataset for methanol is fairly detailed as far as acute toxicity is concerned. However, there were not sufficient chronic (long-term) toxicity data to meet the CCME (2007) requirements for a Canadian water quality guideline (CWQG). In fact, the lack of acceptable salmonid long-term data prevented the development of even an interim CWQG. In order to provide some guidance regarding soils at contaminated sites however, a non-standard salmonid test was considered. This test had a sensitive endpoint (fertilization), that would appear to be protected by the proposed water quality guideline. The guideline should be updated when long-term salmonid data become available.

Irrigation Water
Insufficient data are available to calculate a water quality guideline for irrigation.

Livestock Watering
Insufficient data are available to meet the CCME (2006) requirements for developing a livestock watering guideline.

9. PARAMETER VALUES

Parameter values required to calculate the Canadian soil quality guidelines for methanol fall into two main groups: i) parameters that relate to the chemical properties, toxicity, or background exposure to methanol, referred to as “chemical-specific parameters”; and, ii) parameters relating to receptor exposure and properties of the site, referred to as “non-chemical-specific parameters”. These two groups of parameters are discussed below.

9.1 Chemical-Specific Parameters

Chemical-specific parameters for methanol are summarized in Appendix 7, together with an indication of where to find a discussion of the rationale for the value selected. The soil allocation factor (SAF) and water allocation factor (WF) each take their default values of 0.2, since exposure to methanol is possible via all five potentially contaminated environmental media: soil, water, air, food, and consumer products.

9.2 Non Chemical-Specific Parameters

Non chemical-specific parameter values are taken without change from CCME (2006). Parameter values for human receptor characteristics, soil and hydrogeological parameters, site characteristics, and building parameters are provided in Appendices 8 to 11 respectively.

10. DERIVATION OF WATER GUIDELINES

CCME uses surface water quality guidelines as a basis from which to calculate corresponding groundwater and soil quality guidelines. Surface water quality guidelines calculated for methanol are provided and discussed below.

10.1 Human Drinking Water

No Guidelines for Canadian Drinking Water Quality (GCDWQ) currently exists for methanol. In such cases, CCME (2006) includes a protocol for calculating an allowable concentration in potable water (Source Guidance Value for Groundwater) from the tolerable daily intake using the following equation:

$$SGVG = \frac{TDI \times BW \times WF}{WIR}$$

where:

SGVG	=	Source Guidance Value for Groundwater (mg/L)
TDI	=	tolerable daily intake (mg/kg/d)
BW	=	body weight (kg)
WF	=	water allocation factor (unitless)
WIR	=	water ingestion rate (L/d)

The SGVG is calculated using adult parameters (CCME, 2006). Substituting appropriate adult parameter values from Appendix 7 and 8 gives a value of 19 mg/L which is the Source Guidance Value for Groundwater for methanol (Table 3).

Table 3. Surface Water Quality Guidelines for Methanol

Water Use	Guideline Value (mg/L)
Human drinking water ("Source Guidance Value for Groundwater")	19
Freshwater aquatic life	23
Irrigation [1]	n/c
Livestock watering [2]	n/c

Notes:

n/c = not calculated

1. guideline protective of irrigation not calculated;
   not expected to be an issue due to volatility and degradability of methanol.
2. guideline not calculated due to the lack of toxicity information for livestock species.

10.2 Freshwater Aquatic Life

The CCME (2007) Protocol for the Derivation of Water Quality Guidelines for the Protection of Aquatic Life ("the Protocol") was used to calculate a surface water guidance value for methanol for the protection of aquatic life. Although the dataset was not sufficient to meet the requirements for a Canadian water quality guideline, there were enough data to provide guidance for a soil quality guideline for the protection of aquatic life pathway.

The Protocol includes methodologies for guidelines protective of both long-term and short-term exposure. Long-term exposure guidelines identify benchmarks in the aquatic ecosystem that are intended to protect all forms of aquatic life for indefinite exposure periods, while short-term guidelines protect only a specified fraction of individuals from severe effects for a defined short-term exposure period. Soil quality guidelines protective of aquatic life are based on long-term water quality guidelines. Short term water quality guidelines are generally not relevant for developing soil quality guidelines and were not calculated.

Freshwater aquatic toxicity data for methanol were obtained from the U.S. EPA ECOTOX database (U.S. EPA 2014a) and other sources, discussed in Section 5.1. The data selected for the species sensitivity distribution (SSD) calculation are summarized in Appendix 4. Potential effects of methanol on biological oxygen demand were not considered in the development of the interim freshwater aquatic life water quality guideline.

10.2.1 Data Search and Screening

Candidate data were identified by conducting a search on the U.S. EPA ECOTOX database (U.S. EPA, 2014a). A search was made based on the Chemical Abstracts Service (CAS) number for methanol (67561), for both plants and animals, all effects, all publication years, aquatic biota only. The data identified by this search were first separated into freshwater and marine data. The following procedure was carried out separately on the freshwater and marine data sets.

The first step was to screen out the short-term data since only a long-term guideline is required to support soil quality guideline development. Based on guidance in the Protocol the following data were retained.

- Fish and amphibian data were retained if the exposure period was ≥21 days for adult or juvenile stages, or ≥7 days for eggs or larvae.
- Data for non-lethal endpoints for aquatic invertebrates were retained if the exposure period was ≥96 hours for short-lived invertebrates, or ≥7 days for longer-lived invertebrates.
- Data for lethal endpoints for aquatic invertebrates were retained if the exposure period was ≥21 days for longer-lived invertebrates, or retained for additional consideration ≥7 days for longer-lived invertebrates.
- Data for algae were retained if the exposure period was >1 day.
- There were no data for aquatic plants other than algae.

Any data for other biota not included in the above groups (e.g., cyanobacteria) were excluded.

The Craig et al. (1977) data for chum salmon (*Oncorhynchus keta*) were retained in the long-term dataset based on professional judgment. This experiment was run from fertilization of ova to hatching of alevins (approximately 58 days), with endpoints including survival rate to hatching, time to hatching (expressed as degree days), alevin length at hatching, and alevin deformities at hatching. However, the maximum time of exposure to methanol was only 30 minutes, timed to coincide with what the authors felt was the most sensitive stage of development, from fertilization of ova to "water hardening" of the eggs. Based on the above, this endpoint was considered meaningful in the long-term dataset. A conservative approach was taken, however, and the value included was the NOEC, rather than the MATC, since there was an order of magnitude between NOEC and low observable effects concentration (LOEC) concentrations in this study.

Once the set of long-term toxicity data was established, the data were classified as primary, secondary, or unacceptable based on the criteria provided in the Protocol. Upon assessment, the Altenburger (2004) study was classified as unacceptable based on limited experimental detail provided and control response not reported. The remainder of the long-term data were assessed as acceptable, based on having generally adequate methodology which was sufficiently well documented and included control survival. None of the data met all the required criteria to be classified as primary, and thus all of the long-term data were classified as secondary.

One further screening step was applied to the data. This was in relation to several aquatic toxicity studies on compounds other than methanol, which did, however, use methanol as a solvent for the test chemical(s), and then conducted a solvent control to ensure that the concentration of methanol used was not having a toxic effect on the test organisms. The level of methanol used is typically selected to be below the threshold where any adverse effect would be expected, and typically only a single concentration of methanol (plus negative control) is used. Accordingly, these tests typically yield an unbounded NOEC. An unbounded NOEC that is lower than any bounded low effects estimate does not contribute to the knowledge of where a threshold for a low effects level might lie, and could significantly skew the interpretation of the overall dataset, and accordingly such points were rejected. Studies that were rejected for this reason included, Koprivnikar et al. (2011), Lv et al. (2006), Morley et al. (2004), and Suedel et al. (1997) in the freshwater dataset, and Bengtsson et al. (1984), Linden et al. (1979), and Nice (2005), in the marine dataset. However, unbounded NOEC data points were retained if they fell within the range of bounded data points, since such points provide a conservative lower bound estimate of a low effect level for that species without unduly distorting the data distribution, and help to make up the minimum required number of data points.

Where possible, an MATC was calculated as the geometric mean of NOEC and LOEC for a study that presented both of these measures.
Where there were more than one data point for the same species from one or more studies, the longest exposure duration was selected. If there were still more than one data point, the most preferred endpoint was selected in the order EC/IC_x (representing a no effects threshold) > EC/IC_{10} > $EC/IC_{11\text{-}25}$ > MATC > NOEC/IC>LOEC/IC>$EC/IC_{26\text{-}49}$>non-lethal EC/IC_{50}.

Note that the nine data points from the Okumura (2001) study were erroneously included in the ECOTOX database as freshwater data points, whereas in fact these were marine tests.

The long-term freshwater and marine data points remaining after the screening process described above are summarized in Appendix 4 and 5.

The long-term freshwater dataset in Appendix 4 provided sufficient data to develop a surface water guidance value for the purposes of deriving soil guidelines for the protection of fresh water aquatic life pathway. There are studies on three fish species including at least one salmonid (*Oncorhynchus keta*) and at least one non-salmonid (*Oreochromis mossambicus* and *Oryzias latipes*). There are studies on three aquatic or semi-aquatic invertebrates (*Planorbis carinatus, Ceriodaphnia dubia*, and *Chironomus riparius*) with at least one planktonic crustacean (the *C. dubia*). There are four studies on algae, meeting the requirements for at least one plant study.

10.2.2 Guideline Development

The minimum data set appears to have been met, based on Appendix 4. There are three fish studies, albeit with a non-standard salmon study, and two with warm water species, medaka and tilapia. There are three invertebrate studies and four algal/plant studies.

The Protocol requirements for developing a Type A Guideline were followed. In general, this involved ranking the data in Appendix 4, and then fitting various statistical models to this species sensitivity distribution (SSD). The model yielding the best fit to the data was used to estimate the 5^{th} percentile of the distribution and this value was adopted as the long-term water quality guideline.

The process noted above was facilitated by the use of the software SSD Master Version 3.0 (CCME 2012) which was developed explicitly for this purpose. SSD Master was run using the input data in Appendix 4 and using Hazen plotting positions and a logarithmic scale. The goodness of fit of the four models is illustrated in Figure 3, and shows that the Extreme Value distribution (also known as the Gompertz Distribution) is the best fit to the data. This is confirmed by the statistics calculated for each model, where the mean square error (MSE) is lowest for the Extreme Value distribution both for the whole SSD and for the lower tail alone. The 5^{th} percentile of the Extreme Value model fit to this dataset is 23 mg/L, and this is adopted as the surface water quality guideline for the purposes of calculating methanol soil quality guidelines for the protection of freshwater aquatic life.

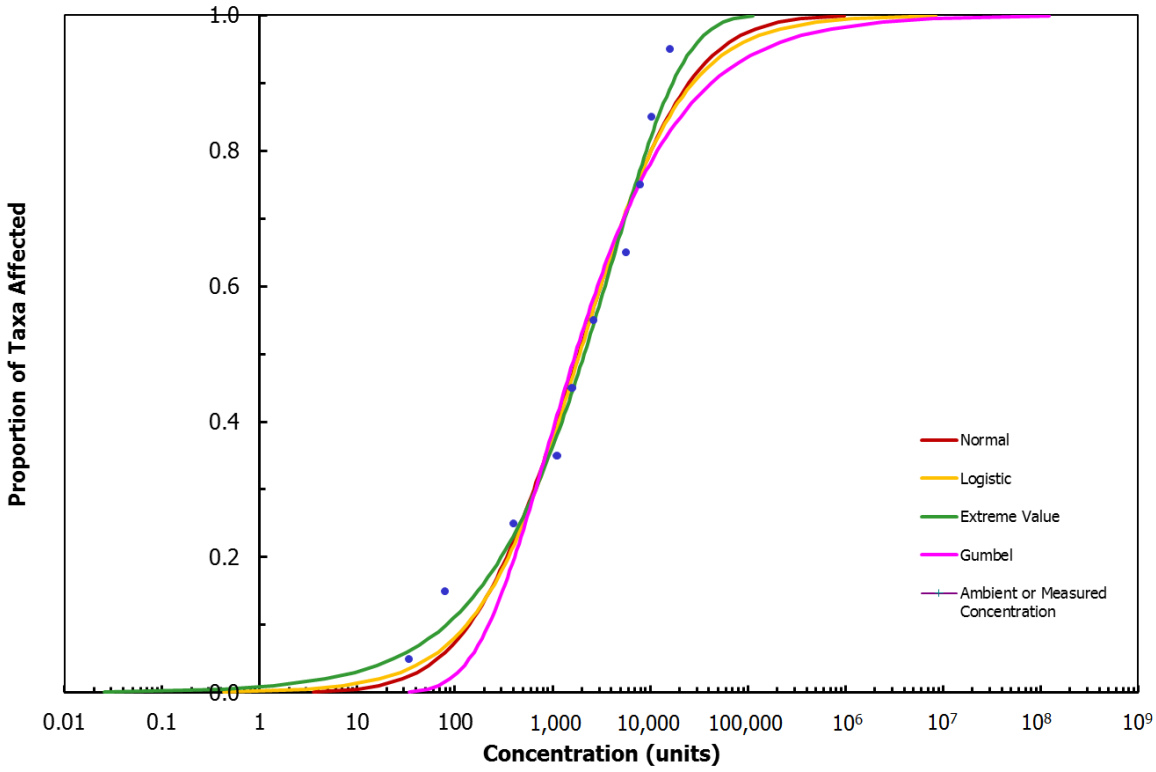

Figure 3. Freshwater SSD with Statistical Models Fit to the Data

10.3 Irrigation Water

No guideline was calculated for methanol in irrigation water, since the minimum data requirements were not met. Due to the volatility and ready degradability of methanol in surface water and shallow aerobic soil systems, this exposure pathway is not expected to be an issue at the majority of sites.

10.4 Livestock and Wildlife Watering

Methanol toxicity data were not available for livestock or wildlife species, and accordingly, these guidelines could not be calculated.

11. DERIVATION OF HUMAN HEALTH SOIL QUALITY GUIDELINES

11.1 Direct Contact

The model used to calculate the soil quality guideline protective of the human direct soil contact (soil ingestion, dermal contact, and particulate inhalation) exposure pathway for methanol is taken without change from CCME (2006). Based on guidance in CCME (2006), exposure via particulate inhalation is not considered for volatile compounds such as methanol, since volatile chemicals are presumed to be lost from soil particles during wind transport. Excluding the particulate inhalation pathway was achieved by setting IR_S to 0 kg/day for volatile chemicals in the equations below. Parameter values are summarized in Appendix 7 and 8. The following equation was used.

$$PSQG_{HH} = \frac{(TDI - EDI) \times SAF \times BW}{\left[\left(AF_G \times SIR\right) + \left(AF_L \times IR_S \times ET_2\right) + \left(AF_S \times SR\right)\right] \times ET_1} + \left[BSC\right]$$

Where:

$PSQG_{HH}$	=	preliminary human health-based soil quality guideline (mg/kg)
TDI	=	tolerable daily intake (mg/kg bw per day)
EDI	=	estimated daily intake (mg/kg bw per day)
SAF	=	soil allocation factor (dimensionless)
BW	=	adult or toddler body weight (kg)
AF_G	=	absorption factor for gut (dimensionless)
AF_L	=	absorption factor for lung (dimensionless)
AF_S	=	absorption factor for skin (dimensionless)
SIR	=	adult or toddler soil ingestion rate (kg/day)
IR_S	=	inhalation of particulate matter re-suspended from soil (kg/day)
SR	=	adult or toddler soil dermal contact rate, see below (kg/day)
ET_1	=	exposure term 1 (dimensionless) (days/week ÷ 7 x weeks/year ÷ 52)
ET_2	=	exposure term 2 (dimensionless) (hours/day ÷ 24)
BSC	=	background soil concentration (mg/kg)

Substituting appropriate values from Tables 9 and 10 into this equation and rounding to 2 significant figures gives values of 8,900 mg/kg (agricultural and residential), 13,000 mg/kg (commercial), and 64,000 mg/kg (industrial) for the human direct contact guideline (Tables 4 and 5).

Soil Dermal Contact Rate

The soil dermal contact rate (SR) is the mass of contaminated soil which is assumed to contact the skin each day. This parameter is calculated as follows CCME (2006):

$$SR = \left\{\left(SA_H \times DL_H\right) + \left(SA_O \times DL_O\right)\right\} \times EF$$

Where:

SR	=	soil dermal contact rate (kg/day)
SA_H	=	exposed surface area of hands (m^2)
DL_H	=	dermal loading of soil to hands (kg/m^2 per event)
SA_O	=	area of exposed body surfaces other than hands (m^2)
DL_O	=	dermal loading of soil to other surfaces (kg/m^2 per event)
EF	=	exposure frequency (events/day)

The soil dermal contact rate is calculated separately for toddlers and adults using the parameters in Appendix 8.

11.2 Inhalation

Soil and groundwater guidelines protective of the indoor infiltration and inhalation pathway were calculated using the equations from CCME (2006) without change for soil and groundwater.

11.2.1 Model Assumptions

Assumptions implicit in the model include the following:

- contaminant vapour immediately above the groundwater table is assumed to be in equilibrium with contaminant concentrations in the groundwater based on Henry's Law
- the soil is physically and chemically homogeneous
- cracks in the building floor slab are filled with dry material of the underlying soil type
- the moisture content is uniform throughout the unsaturated zone
- decay of the contaminant source is not considered (i.e., infinite source mass)
- attenuation of the contaminant in the unsaturated zone is not considered and
- interactions of the contaminant with other chemicals or soil minerals are not considered.

11.2.2 Soil

The equation used was as follows CCME (2006).

$$SQG_I = \frac{(TC - C_a) \times [\theta_w + (K_{oc} \times f_{oc} \times \rho_b) + (H' \times \theta_a)] \times SAF \times DF_i \times 10^3}{H' \times \rho_b \times ET \times 10^6} + BSC$$

Where:

SQG_I	=	soil quality guideline for indoor infiltration (mg/kg)
TC	=	tolerable concentration (mg/m^3)
C_a	=	background air concentration (mg/m^3)
θ_w	=	moisture-filled porosity (dimensionless)
K_{oc}	=	organic carbon partition coefficient (L/kg)
f_{oc}	=	fraction of organic carbon (g/g)
ρ_b	=	dry soil bulk density (g/cm^3)
H'	=	dimensionless Henry's Law Constant (dimensionless)
θ_a	=	vapour-filled porosity (dimensionless)
SAF	=	soil allocation factor (dimensionless)

DF_i	=	dilution factor from soil gas to indoor air (calculated below)
10^3	=	conversion factor from kg to g
ET	=	exposure term (dimensionless)
10^6	=	conversion factor from m^3 to cm^3
BSC	=	background soil concentration (mg/kg)

Substituting appropriate values (found in Appendices 7, 8, 9, 11) into this equation gives values of 3,800 mg/kg (agricultural and residential, coarse soil), 40,000 mg/kg (commercial and industrial, coarse soil), Table 4, and 100,000 mg/kg (agricultural and residential, fine soil), 490,000 mg/kg (commercial and industrial, fine soil), Table 5.

11.2.3 Dilution Factor Calculation

This section presents the CCME (2006) equations that were used to calculate the dilution factor in the above equations. The dilution factor (DF_i) was calculated as follows:

$$DF_i = \frac{1}{\alpha}$$

Where:	DF_i	=	dilution factor from soil gas concentration to indoor air concentration (unitless)
	α	=	attenuation coefficient (unitless; see derivation below).

Calculation of α

The attenuation coefficient, α, was calculated using the following equation:

$$\alpha = \frac{\left(\frac{D_T^{eff} A_B}{Q_B L_T}\right) exp\left(\frac{Q_{soil} L_{crack}}{D_{crack} A_{crack}}\right)}{exp\left(\frac{Q_{soil} L_{crack}}{D_{crack} A_{crack}}\right) + \left(\frac{D_T^{eff} A_B}{Q_B L_T}\right) + \left(\frac{D_T^{eff} A_B}{Q_{soil} L_T}\right)\left[exp\left(\frac{Q_{soil} L_{crack}}{D_{crack} A_{crack}}\right) - 1\right]}$$

where:

α	=	attenuation coefficient (dimensionless)
D_T^{eff}	=	effective porous media diffusion coefficient (cm^2/s)
A_B	=	building area (cm^2)
Q_B	=	building ventilation rate (cm^3/s)
L_T	=	distance from contaminant source to foundation (cm)
Q_{soil}	=	volumetric flow rate of soil gas into the building (cm^3/s)
L_{crack}	=	thickness of the foundation (cm)
D_{crack}	=	effective vapour diffusion coefficient through the crack (cm^2/s)
A_{crack}	=	area of cracks through which contaminant vapours enter the building (cm^2)

Calculation of DT^{eff}:

$$D_T^{eff} \approx D_a \times \left(\frac{\theta_a^{10/3}}{\theta_t^2} \right)$$

Where:
- D_T^{eff} = overall effective porous media diffusion coefficient based on vapour-phase concentrations for the region between the source and foundation (cm^2/s)
- D_a = diffusion coefficient in air (cm^2/s)
- θ_a = soil vapour-filled porosity (dimensionless)
- θ_t = soil total porosity (dimensionless)

Calculation of D_{crack}:

D_{crack} is calculated in exactly the same way as DT^{eff}, with the exception that the assumption is made that the soil material in the cracks is dry CCME (2006), and accordingly, the air filled porosity is the same as the total porosity, and the equation becomes:

$$D_{crack} \approx D_a \times \left(\frac{\theta_t^{10/3}}{\theta_t^2} \right)$$

Where:
- D_{crack} = effective porous media diffusion coefficient in floor cracks (cm^2/s)
- D_a = diffusion coefficient in air (cm^2/s)
- θ_t = total porosity for coarse soil (dimensionless)

Calculation of Q_B:

$$Q_B = \frac{L_B W_B H_B ACH}{3{,}600}$$

Where:
- Q_B = building ventilation rate (cm^3/s)
- L_B = building length (cm)
- W_B = building width (cm)
- H_B = building height (cm)
- ACH = air exchanges per hour (h^{-1})
- 3,600 = conversion factor from hours to seconds

Calculation of Q_{soil}:

$$Q_{soil} = \frac{2\pi \Delta P k_v X_{crack}}{\mu \, ln\left[\frac{2Z_{crack}}{r_{crack}} \right]}$$

Where	Q_{soil}	=	volumetric flow rate of soil gas into the building (cm^3/s)
	ΔP	=	pressure differential ($g/cm \cdot s^2$)
	k_v	=	soil vapour permeability to vapour flow (cm^2)
	X_{crack}	=	length of idealized cylinder (cm)
	μ	=	vapour viscosity (0.000173 g/cm·s)
	Z_{crack}	=	distance below grade to idealized cylinder (cm)
	r_{crack}	=	radius of idealized cylinder (cm; calculated as A_{crack}/X_{crack})

11.3 Offsite Migration

Offsite migration guidelines are calculated to check that the guideline set for commercial and industrial land use will not result in adjacent more sensitive land being contaminated at levels above the applicable guideline for the sensitive land due to wind and/or water transport of contaminated soil from the commercial or industrial site. However, the guideline is not applicable to volatile or readily degradable compounds (CCME 2006) since significant contaminant mass loss is expected to occur during wind and/or water transport of contaminated soil.

Accordingly, the soil quality guideline protective of off-site migration is not calculated for methanol.

12. DERIVATION OF ENVIRONMENTAL SOIL QUALITY GUIDELINES

12.1 Soil Contact

The soil quality guideline for soil contact by soil dependent organisms (i.e., plants and invertebrates) is calculated based on a weight of evidence approach using an EC25 distribution following CCME (2006). Data relevant for guideline development are sourced from Stantec (2006) and are summarized in Appendix 2 and 3. The values provided in Appendices 2 and 3 are nominal values based on the known amount of chemical spiked into the test soils. Stantec (2006) included analytical data to confirm exposure concentrations. The regression for the analytical data was $y = 0.9714x - 401.66$ where x is the nominal concentration and y the measured concentration. The CCME (2006) protocol uses data standardized at the 25^{th} percentile effect level. EC_{25} data, corrected for analytical recovery, are summarized below.

Species	Endpoint	EC_{25} (mg/kg)
Alfalfa	Shoot Length	1,748
Alfalfa	Root Length	7,317
Alfalfa	Shoot Dry Mass	1,355
Alfalfa	Root Dry Mass	2,716
Barley	Shoot Length	4,344
Barley	Root Length	5,186
Barley	Shoot Dry Mass	2,064
Barley	Root Dry Mass	2,341
Northern Wheatgrass	Shoot Length	3,629
Northern Wheatgrass	Root Length	11,452
Northern Wheatgrass	Shoot Dry Mass	2,393
Northern Wheatgrass	Root Dry Mass	3,129
Eisenia andrei	Number of Progeny	12,540
Eisenia andrei	Dry Mass of Individual Progeny	9,076
Folsomia candida	Number of Progeny	2,359

The soil contact guideline for natural areas, agricultural and residential is based on the 25th percentile of ranked distribution these data. The soil contact guideline for commercial and industrial land use is based on the 50th percentile of the ranked distribution.

- 25th percentile: 2,341 mg/kg
- 50th percentile: 3,129 mg/kg

The soil guideline protocol (CCME 2006) states that plant and invertebrate data should come from a minimum of 3 studies, but data from fewer than 3 studies can be used if professional judgment is satisfied that the data is sufficient. In the case of methanol, plant and invertebrate data come from one study, but the study provides sufficient data and includes both plants and invertebrates. The invertebrate data are for reproduction endpoints, which are typically more sensitive than mortality endpoints. The protocol further recommends an uncertainty factor of 1 to 5 if only the minimum 3 studies is available. Based on a review of safety factors used in developing guidelines from similar datasets, the 25th and 50th percentile values were further adjusted with a safety factor of 2 and rounded to two significant figures. The resulting soil contact guidelines are summarized below:

- Agricultural and residential/parkland soil contact guideline: 1,200 mg/kg
- Commercial and industrial soil contact guideline: 1,600 mg/kg

12.2 Nutrient and Energy Cycling

Insufficient data were available and this guideline was not calculated for methanol.

12.3 Soil and Food Ingestion

Insufficient data were available (Section 8.1), and this guideline was not calculated for methanol. However, this exposure pathway was not expected to be a concern, since i) methanol is expected

to degrade rapidly in surficial soil (Appendix 1) and accordingly livestock and wildlife are unlikely to get significant exposure to methanol through incidental ingestion of surficial soil; and ii) based on its very low K_{ow} (Table 2) methanol is not expected to accumulate into plants to any significant extent, and thus the exposure of livestock or wildlife to methanol in soil is expected to be minimal.

12.4 Offsite Migration

Offsite migration guidelines are calculated to check that the guideline set for commercial and industrial land use will not result in adjacent more sensitive land being contaminated at levels above the applicable guideline for the sensitive land due to wind and/or water transport of contaminated soil from the commercial or industrial site. However, the guideline is not applicable to volatile or readily degradable compounds (CCME 2006) since significant contaminant mass loss is expected to occur during wind and/or water transport of contaminated soil.

Accordingly, the soil quality guideline protective of off-site migration is not calculated for methanol.

13. GROUNDWATER PATHWAYS

This section provides the protocols used to calculate soil quality guidelines protective of exposure pathways involving groundwater. The following receptors are considered:

- humans (potable drinking water sourced from groundwater), and
- aquatic life (via lateral groundwater transport and discharge into a surface water body).

In the first case, it is assumed that a water well could potentially be installed at any location, and hence it is assumed that there is no lateral offset between the location where the contaminated soil or groundwater is measured and the receptor.

In the second case, a minimum lateral separation of 10 m is assumed between the location where the contaminated soil or groundwater is measured and the location of the surface water body. In cases where contamination is present within 10 m of a surface water body, a site-specific approach will be required (see CCME 2006).

Surface water quality guidelines protective of the above water uses are provided in Table 3.

Soil quality guidelines for groundwater pathways were calculated using the model and equations from CCME (2006).

13.1 Model Assumptions

Assumptions implicit in the model include the following:

- the soil is physically and chemically homogeneous
- moisture content is uniform throughout the unsaturated zone
- infiltration rate is uniform throughout the unsaturated zone
- decay of the contaminant source is not considered (i.e., infinite source mass)
- contaminant is not present as a free phase product
- maximum possible concentration in the leachate is equivalent to the solubility limit of the chemical in water under the defined site conditions
- the groundwater aquifer is unconfined
- groundwater flow is uniform and steady
- co-solubility and oxidation/reduction effects are not considered
- attenuation of the contaminant in the saturated zone is assumed to be one dimensional with respect to sorption-desorption, dispersion, and biological degradation
- dispersion in groundwater is assumed to occur in the longitudinal and transverse directions only and diffusion is not considered
- mixing of the leachate with the groundwater is assumed to occur through mixing of leachate and groundwater mass fluxes and
- dilution of the plume by groundwater recharge down-gradient of the source is not considered.

13.2 Guideline Calculation

The soil quality guideline protective of groundwater uses is calculated in the same way for both groundwater uses noted at the start of this section, using the corresponding surface water quality guideline (Table 14) as the starting point for each. However, as noted above, the lateral offset between the point at which the contaminated soil is measured and the surface water body (parameter "x" in the equation for DF4 below) is assumed to be 10 m for aquatic life, and 0 m for human drinking water.

The model considers four processes:

1. partitioning from soil to leachate
2. transport of leachate from base of contamination to water table
3. mixing of leachate and groundwater and
4. groundwater transport down-gradient to a discharge point.

For each of these four processes, a dilution factor was calculated (DF1 through DF4, respectively). DF1 has units of (mg/kg)/(mg/L) or L/kg. The other three dilution factors are dimensionless [units of (mg/L)/(mg/L)]. The overall dilution factor is used to calculate the soil concentration that is protective of groundwater using the following equations:

$SQG_{GW} = SWQG \times DF$

$DF = DF1 \times DF2 \times DF3 \times DF4$

where: SQG_{GW} = soil quality guideline protective of groundwater pathways (mg/kg) (i.e., SQG_{PW}, SQG_{FL}, SQG_{IR}, SQG_{LW})

SWQG= corresponding surface water quality guideline (drinking water or aquatic life) (mg/L)

DF = overall dilution factor (L/kg)

DF1 = dilution factor for process 1 (L/kg)

DF2 = dilution factor for process 2 (dimensionless)

DF3 = dilution factor for process 3 (dimensionless)

DF4 = dilution factor for process 4 (dimensionless)

Dilution Factor 1

Dilution factor 1 (DF1) is the ratio of the concentration of a contaminant in soil to the concentration in leachate that is in contact with the soil. This "dilution factor" represents the three phase partitioning between contaminant sorbed to soil, contaminant dissolved in pore water (i.e., as leachate), and contaminant present as soil vapour. DF1 is calculated using the following equation:

$$DF1 = K_{oc} \times f_{oc} + \frac{(\theta_w + H' \times \theta_a)}{\rho_b}$$

where:

DF1 = dilution factor 1 (L/kg)

K_{oc} = organic carbon-water partition coefficient (L/kg)

f_{oc} = fraction organic carbon (g/g)

θ_w = water filled porosity (dimensionless)

H' = dimensionless Henry's Law constant (dimensionless)

θ_a = air filled porosity (dimensionless)

ρ_b = dry soil bulk density (g/cm^3)

Dilution Factor 2

Dilution factor 2 (DF2) is the ratio of the concentration of a contaminant in leachate that is in contact with the soil, to the concentration in pore water just above the groundwater table. DF2 takes the value 1.00 (i.e., no dilution) for generic guidelines because it is assumed at Tier 1 that the contaminated soil extends down to the water table. DF2 can be calculated on a site-specific basis at Tier 2.

Dilution Factor 3

Dilution factor 3 (DF3) is the ratio of the concentration of a chemical in pore water just above the groundwater table, to the concentration in groundwater beneath the source. This dilution factor reflects a decrease in concentration as leachate mixes with uncontaminated groundwater. DF3 is a function of groundwater velocity, infiltration rate, source length, and mixing zone thickness. The mixing zone thickness is calculated as being due to two processes: i) mixing due to dispersion, and ii) mixing due to infiltration rate. The equations used are as follows:

$$DF3 = 1 + \frac{Z_d \times V}{I \times X}$$

$$Z_d = r + s$$

$$r = 0.01 \times X$$

$$s = d_a \left\{ 1 - exp\left(\frac{-2.178 \times X \times I}{V \times d_a} \right) \right\}$$

$$V = K \times i$$

where:

DF3	=	dilution factor 3 (dimensionless)
Z_d	=	average thickness of mixing zone (m)
V	=	Darcy velocity in groundwater (m/year)
I	=	infiltration rate (m/year)
X	=	length of contaminated soil (m)
r	=	mixing depth due to dispersion (m)
s	=	mixing depth due to infiltration rate (m)
d_a	=	unconfined aquifer thickness (m)
K	=	aquifer hydraulic conductivity (m/year)
i	=	lateral hydraulic gradient in aquifer (dimensionless)

Dilution Factor 4

Dilution factor 4 (DF4) accounts for the processes of dispersion and biodegradation as groundwater travels downgradient from beneath the source of contamination, and is the ratio of the concentration of a chemical in groundwater beneath the source, to the concentration in groundwater at a distance of 10 m (at Tier 1 for aquatic life) downgradient of the source. Consistent with CCME (2006), the time dependent version of the equation to calculate DF4 was used:

$$DF4 = \frac{4}{exp(A) \times erfc(B) \times [erf(C) - erf(D)]}$$

$$A = \frac{x}{2D_x} \left\{ 1 - \left(1 + \frac{4L_s D_x}{v} \right)^{1/2} \right\}$$

$$B = \frac{x - vt\left(1 + \frac{4L_s D_x}{v} \right)^{1/2}}{2(d_x vt)^{1/2}}$$

$$C = \frac{y + Y/2}{2\left(D_y x\right)^{1/2}}$$

$$D = \frac{y - Y/2}{2\left(D_y x\right)^{1/2}}$$

$$L_s = \frac{0.6931}{t_{1/2s}} e^{-0.07d}$$

$$v = \frac{V}{\theta_t R_s}$$

$$R_s = 1 + \frac{\rho_b K_{oc} f_{oc}}{\theta_t}$$

$$D_x = 0.1x$$

$$D_y = 0.01x$$

where:

DF4	=	dilution factor 4 (dimensionless)
erf	=	the error function
A	=	dimensionless group A (dimensionless)
C	=	dimensionless group C (dimensionless)
D	=	dimensionless group D (dimensionless)
x	=	distance to source (10 m, aquatic life and wildlife watering, 0 m other water uses)
D_x	=	dispersivity in the direction of groundwater flow (m)
L_s	=	decay constant (1/year)
v	=	velocity of the contaminant (m/year)
y	=	distance to receptor perpendicular to groundwater flow (m)
Y	=	source width (m)
D_y	=	dispersivity perpendicular to the direction of groundwater flow (m)
$t_{1/2s}$	=	decay half-life of contaminant in saturated zone of aquifer (years)
d	=	water table depth (m)
V	=	Darcy velocity in groundwater (m/year)
θ_t	=	total soil porosity (dimensionless)
R_s	=	retardation factor in saturated zone (dimensionless)
ρ_b	=	dry soil bulk density (g/cm^3)
K_{oc}	=	organic carbon partition coefficient (mL/g)
f_{oc}	=	fraction organic carbon (g/g)
t	=	time since contaminant release (year)

Aquatic Life
Substituting appropriate values from Appendices 7, 8, 9 and 10 into this equation gives values of 7.7 mg/kg for coarse soil (Table 4) and 190 mg/kg for fine soil (Table 5).

Protection of Potable Groundwater
Substituting appropriate values from Appendices 7, 8, 9 and 10 into this equation and setting x to 0, gives values of 4.6 mg/kg for coarse soil (Table 4) and 5.6 mg/kg for fine soil (Table 5).

14. MANAGEMENT LIMIT

Management limits are soil guidelines values that take into consideration issues beyond direct human or ecological toxicity. This includes issues such as aesthetics (odour, soil appearance), flammability and risk of infrastructure damage. No information was available on methanol concentrations in soil that would lead to offensive odours or to infrastructure damage. However, data were available on the flammability of soils containing methanol and a management limit was calculated for methanol based on flammability.

A series of experiments were conducted by Methanex, a major worldwide producer of methanol, on the flammability of field soil samples contaminated with methanol (Terry Rowat, Methanex Corporation, *pers. comm.*). A trench was dug outward from an area of known high methanol contamination towards an area without methanol contamination. The soil in this area was a clay till. The trench provided access to soils with a range of methanol concentrations depending on the point along the trench from which the sample was taken. A series of samples was collected, and a sub-sample from each was preserved for analysis at the Methanex Kitimat lab. Then an attempt was made to ignite each sample, and an observation made as to whether the sample would burn. The results from these experiments are provided in Appendix 12, and indicate that the lowest concentration of methanol which would support combustion was 9,310 mg/kg. Samples at 7,460 mg/kg and lower did not support combustion.

A safety factor of 10 was used together with the concentration of 7,460 mg/kg noted above to set the value for the flammability check for methanol in soil to 750 mg/kg (Tables 4 and 5).

15. RECOMMENDED CANADIAN SOIL QUALITY GUIDELINES

According to the CCME soil protocol (CCME, 2006), both environmental and human health soil quality guidelines are developed for four land uses: agricultural, residential/parkland, commercial, and industrial. The lowest value generated by the two approaches for each of the four land uses is recommended by CCME as the final Canadian Soil Quality Guideline. Therefore, the recommended final Canadian Soil Quality Guidelines for the protection of ecological and human health are 4.6 mg/kg for coarse soil and 5.6 mg/kg for fine soil for all land uses. Tables 4 and 5 summarize the soil quality guideline values derived for all exposure pathways and land uses utilized in the determination of the Canadian Soil Quality Guidelines for methanol for coarse and fine soil, respectively.

Table 4. Canadian Soil Quality Guidelines for Methanol (mg·kg^{-1} dry wt.) - Coarse Soil

	Land Use			
Land Use:	**Agricultural**	**Residential**	**Commercial**	**Industrial**
Guideline	**4.6**	**4.6**	**4.6**	**4.6**
Human Exposure Pathways				
Direct contact	8900	8900	13 000	64 000
Vapour inhalation	3800	3800	40 000	40 000
Protection of potable water	4.6	4.6	4.6	4.6
Produce, milk and meat check [a]	n/c	n/c	n/c	n/c
Off-site migration [b]	n/a	n/a	n/c	n/c
Ecological Exposure Pathways				
Soil contact	1200	1200	1600	1600
Nutrient and Energy cycling check [c]	n/c	n/c	n/c	n/c
Livestock soil and food ingestion [d]	n/c	n/c	n/c	n/c
Protection of freshwater life	7.7	7.7	7.7	7.7
Off-site migration [b]	n/a	n/a	n/c	n/c
Management Limit	750	750	750	750

Notes:
n/a = exposure pathway not applicable in this scenario.

n/c = not calculated

a. Produce, meat and milk check not calculated - methanol not expected to accumulate in produce, milk, or meat.

b. Offsite migration not considered a concern given the volatility and degradability of methanol.

c. Nutrient and energy cycling check not calculated - insufficient data
d. Livestock soil and food ingestion not expected to be a concern, methanol expected to be lost rapidly from surface soil, and not accumulate into fodder.

Table 5. Canadian Soil Quality Guidelines for Methanol (mg·kg^{-1} dry wt.) - Fine Soil

	Land Use			
Land Use:	**Agricultural**	**Residential**	**Commercial**	**Industrial**
Guideline	**5.6**	**5.6**	**5.6**	**5.6**
Human Exposure Pathways				
Direct contact	8900	8900	13 000	64 000
Vapour inhalation	100 000	100 000	490 000	490 000
Protection of potable water	5.6	5.6	5.6	5.6
Produce, milk and meat check [a]	n/c	n/c	n/c	n/c
Off-site migration [b]	n/a	n/a	n/c	n/c
Ecological Exposure Pathways				
Soil contact	1200	1200	1600	1600
Nutrient and Energy cycling check [c]	n/c	n/c	n/c	n/c
Livestock soil and food ingestion [d]	n/c	n/c	n/c	n/c
Protection of freshwater life	190	190	190	190
Off-site migration [b]	n/a	n/a	n/c	n/c
Management Limit	750	750	750	750

Notes:
n/a = exposure pathway not applicable in this scenario.

n/c = not calculated

a. Produce, meat and milk check not calculated - methanol not expected to accumulate in produce, milk or meat.
b. Offsite migration not considered a concern given the volatility and degradability of methanol.

c. Nutrient and energy cycling check not calculated - insufficient data
d. Livestock soil and food ingestion not expected to be a concern, methanol expected to be lost rapidly from surface soil and not accumulate into fodder.

REFERENCES

Abou-Waly, H.F., 2000. Effect of organic solvents on growth of freshwater algae. Int. J. Environ. Stud.57(4): 411-418.

Alberta Environment, 2010. Soil and Groundwater Remediation Guidelines for Methanol.

Allen, M., Richardson, M. and Jones-Otazo, H. 2008. Probability density functions describing 24-hour inhalation rates for use in human health risk assessments: An update and comparison. Hum. Ecol. Risk Assess. 14(2): 372-391.

Altenburger, R., H. Walter, and M. Grote, 2004. What contributes to the combined effect of a complex mixture? Environ. Sci. Technol.38(23): 6353-6362.

Andrews, L.S., Clary, J.J., Terrill, J.B., & Bolte, H.F., 1987. Subchronic inhalation toxicity of methanol. Journal of Toxicology and Environmental Health, 20: 117-124.

ANZECC (Australian and New Zealand Environment and Conservation Council), 2000. Australian and New Zealand Guidelines for Fresh and Marine Water Quality. National Water Quality Management Strategy.

Apaja, M. (1980). Evaluation of toxicity and carcinogenicity of malonaldehyde: An experimental study in Swiss mice. Acta Universitatis Ouluensis, Series D, Medica 55 (Vol. 8). Finland: *Anat Pathol Microbiol*.

API (American Petroleum Institute), 1994. Transport and Fate of Dissolved Methanol, Methyl-Tertiary-Butyl Ether, and Monoaromatic Hydrocarbons in a Shallow Sand Aquifer. API Publication 4601. April, 1994.

Baker, J.R., Mihelcik, J.R., Leuhrs, D.C., and Hickey, J.P., 1997. Evaluation of estimation methods for organic carbon normalized sorption coefficients. *Water Environmental Research.* **69(2)**:136-144.

Bengtsson, B.E., L. Renberg, and M. Tarkpea, 1984. Molecular structure and aquatic toxicity - an example with C1-C13 aliphatic alcohols. *Chemosphere* **13(5-6)**:613-622.

Bindler F., Voges E., & Laugel P., 1988. The problem of methanol concentration admissible in distilled fruit spirits. *Food Additives and Contaminants*, **5**: 343-351.

Boethling, R.S., and Mackay, D., 2000. Handbook of Property Estimation Methods for Chemicals, Environmental and Health Sciences. Lewis Publishers, Boca Raton, FL, 481 p.

Burbacher, T. M., Grant, K., Shen, D., Damian, D., Ellis, S., & Liberato, N., 1999a. Reproductive and offspring developmental effects following maternal inhalation exposure to methanol in nonhuman primates Part I: methanol disposition and reproductive toxicity in adult females Cambridge, MA: Health Effects Institute.

Burbacher, T. M., Grant, K., Shen, D., Damian, D., Ellis, S., & Liberato, N., 1999b. Reproductive and offspring developmental effects following maternal inhalation exposure to methanol in nonhuman primates Part II: developmental effects in infants exposed prenatally to methanol. Cambridge, MA: Health Effects Institute.

Burbacher, T. M., Grant, K. S., Shen, D. D., Sheppard, L., Damian, D., Ellis, S., & Liberato, N., 2004a. Chronic maternal methanol inhalation in nonhuman primates (Macaca fascicularis): exposure and toxicokinetics prior to and during pregnancy. *Neurotoxicology and Teratology*, **26**, 201-221.

Burbacher, T. M., Grant, K. S., Shen, D. D., Sheppard, L., Damian, D., Ellis, S., & Liberato, N., 2004b. Chronic maternal methanol inhalation in nonhuman primates (Macaca fascicularis): reproductive performance and birth outcome. *Neurotoxicology and Teratology*, **26**, 639-650.

CalEPA (California Environmental Protection Agency), 2012. Safe Drinking Water and Toxic Enforcement Act of 1986. Proposition 65. Initial Statement of Reasons. Title 27, California Code of Regulations Proposed Amendment to Section 25805(B), Specific Regulatory Levels: Chemicals Causing Reproductive Toxicity. Methanol.

Cameron, A. M., Nilsen, O. G., Haug, E., & Eik-Nes, K. B., 1984. Circulating concentrations of testosterone, luteinizing hormone and follicle stimulating hormone in male rats after inhalation of methanol. *Archives of Toxicology*, **7**, 441-443.

Cameron, A. M., Zahlsen, K., Haug, E., Nilsen, O. G., & Eik-Nes, K. B., 1985. Circulating steroids in male rats following inhalation of n-alcohols. *Archives of Toxicology*, **8**, 422-424.

Canada. 1999. Canadian Environmental Protection Act, 1999. S.C., 1999, c33.

CAPP, 1996. Hydrostatic Test Water Management. Canadian Association of Petroleum Producers. CAPP Pub # 1996-0014. Available at: http://www.capp.ca/publications-and-statistics

Cavanaugh, L.A., Schadt, C.F., and Robinson, E., 1969. Atmospheric hydrocarbon and carbon monoxide measurements at Point Barrow, Alaska. *Environmental Science and Technology* **3**, 251-257.

CCME, 1999 (and updates). Canadian Environmental Quality Guidelines. Canadian Council of Ministers of the Environment, Winnipeg.

CCME, 2006. A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines. Canadian Council of Ministers of the Environment, Winnipeg. PN 1332.

CCME, 2007. A Protocol for the Derivation of Water Quality Guidelines for the Protection of Aquatic Life. Canadian Council of Ministers of the Environment, Winnipeg, Manitoba.

CCME, 2008. Canada-Wide Standards for Petroleum Hydrocarbons (PHC) in Soil: Scientific Rationale. Canadian Council of Ministers of the Environment, Winnipeg, Manitoba.

CCME, 2012. SSD Master. Determination of Hazardous Concentrations with Species Sensitivity Distributions. Version 3.0. August 2012.

CERHR (National Toxicology Program – Center for the Evaluation of Risks to Human Reproduction), 2004. NTP-CERHR Expert Panel report on the reproductive and developmental toxicity of methanol. *Reproductive Toxicology*, **18**, 303-390.

Cheminfo Services, 2014. Bio Based Chemical Import Replacement Initiative. Chemical Markets and Biochemical Options for Alberta, prepared for Enterprise and Advanced Education Alberta. http://www.albertacanada.com/business/industries/industry-reports.aspx

Chuwers, P., Osterloh, J., Kelly, T., D'Alessandro, A., Quinlan, P., & Becker, C., 1995. Neurobehavioral effects of low-level methanol vapor exposure in healthy human volunteers. *Environmental Research*, **71(2)**, 141-150.

Clayton G.D. & Clayton F.E. ed., 1982. Patty's industrial hygiene and toxicology - Volume 2C: Toxicology with cumulative index for Volume 2, 3rd ed. New York, Chichester, Brisbane, Toronto, John Wiley & Sons, pp 4527-4551.

Cook, M. R., Bergman, F. J., Cohen, H. D., Gerkovich, M. M., Graham, C., Harris, R. K., & Siemann, L. G., 1991. Effects of methanol vapor on human neurobehavioral measures. (Report No. Research Report Number 42). Boston, MA: Health Effects Institute.

COT (Committee on Toxicity). (2011). COT Statement on the effects of chronic dietary exposure to methanol. http://cot.food.gov.uk/cotstatements/cotstatementsyrs/cotstatements2011/cot201102.

Craig, P.C., F.C. Withler, and R.B. Morley, 1977. Effects of methanol on the fertilisation of chum salmon (Oncorhynchus keta) ova. Environ. Pollut.14(2): 85-92.

CRC (Chemical Rubber Company), 1996. CRC Handbook of Chemistry and Physics, Taylor and Francis, CRC Press.

Cruzan, G., 2009. Assessment of the cancer potential of methanol. *Critical Reviews in Toxicology*, **39(4)**, 347-363. doi: 10.1080/10408440802475199

Davis, D.G., Wergin, W.P. and Dusbabek, K.E. 1978. Effect of organic solvents used in herbicides on growth and ultrastructure of plant cell suspensions. *Pesticide Biochemistry and Physiology*, **8**: 84-97.

Ding, F.H., Z.Z. Xiao, and J. Li, 2007. Preliminary studies on the vitrification of Red Sea bream (Pagrus major) embryos. Theriogenology68(5): 702-708.

Eisenmenger, W.S., 1930. Toxicity of some aliphatic alcohols. *Plant Physiology*, **5**: 131-156.

Environment Canada. 2013. National Pollutant Release Inventory. On-Line Database available at http://www.ec.gc.ca/inrp-npri/

Environment Canada/Health Canada. 2001. Priority Substances List Assessment Report – Methanol. Ottawa, Ontario.

Eriksen, S.P, and Kulkarni, A.B., 1963. Methanol in normal human breath. *Science*, **141**, 639-640.

Esteban, A., Hernandez, V., and Lunsford, K., 2001. Exploit the benefits of methanol. Proc. 79th Annual Gas Processing Association Annual Convention, Atlanta, Georgia. Published by Bryan Research and Engineering Inc., Bryan, Texas, and available online at: http://www.bre.com/portals/0/technicalarticles/Exploit%20the%20Benefits%20of%20Methanol.pdf

FishBase. 2014. FishBase. R. Froese and D. Pauly (eds.). www.fishbase.org, version (11/2014).

Fiskesjo, G., 1985. The Allium Test as a standard in environmental monitoring. *Hereditas* **102**:99-112 (OECDG Data File).

Francot P & Geoffroy P, 1956. Le méthanol dans les jus de fruits, les boissons fermentées des alcools et spiriteux. *Revue des Fermentations et des Industries Alimentaires*, **11**, 279-287.

Fu, S. S., Sakanashi, T. M., Rogers, J. M., & Hong., 1996. Influence of dietary folic acid on the developmental toxicity of methanol and the frequency of chromosomal breakage in the CD-1 mouse. *Reproductive Toxicology*, **10**, 455-463.

Garrett, D.C., 2004. Effects of Methanol, Atrazine, and Copper on the Ultrastructure of Pseudokirchneriella subcapitata (Selenastrum capricornutum). Ph.D.Thesis, University of North Texas, Denton, TX:192 p.

Gonzalez-Doncel, M., M.S. Okihiro, C.F. Torija, J.V. Tarazona, and D.E. Hinton, 2008. An artificial fertilization method with the Japanese medaka: Implications in early life stage bioassays and solvent toxicity. Ecotoxicol. Environ. Saf.69(1): 95-103.

Graedel T.E., Hawkins D.T., & Claxton L.D. eds., 1986. Atmospheric chemical compounds: Sources, occurrence and bioassay. New York, London, Academic Press, pp 512-514, 557.

Greizerstein HB, 1981. Congener contents of alcoholic beverages. *Journal of Studies on Alcohol*, **42**, 1030-1037.

Grosjean, D. 1997. Atmospheric chemistry of alcohols. J. Braz. Chem. Soc. 8(4):433-442.

GRI (Gas Research Institute), 1996. Background Report on Subsurface Environmental Issues Relating to Natural Gas Sweetening and Dehydration Operations. GRI-95/0143, prepared by Sorensen, J.A., Fraley, R.H., Gallagher, J.R., and Schmit, C.R.

Guinn, G., 1977. Effects of some organic solvents on ethylene evolution from young cotton bolls. *Plant Physiology*, **60**: 446-448.

Hack, L.A., L.A. Tremblay, S.D. Wratten, G. Forrester, and V. Keesing, 2008. Zinc sulfate and atrazine toxicity to the marine harpacticoid copepod *Robertsonia propinqua*. N. Z. J. Mar. Freshw. Res.42(1): 93-98.

Han, T., Y.S. Han, C.Y. Park, Y.S. Jun, M.J. Kwon, S.H. Kang, and M.T. Brown, 2008. Spore release by the green alga *Ulva*: A quantitative assay to evaluate aquatic toxicants. Environ. Pollut.153(3): 699-705.

Health Canada, 2004. Federal Contaminated Site Risk Assessment in Canada. Part II: Health Canada Toxicological Reference Values (TRVs). Prepared by: Environmental Health Assessment Services Safe Environments Programme, September 2004. .

Health Canada, 2007. Summary of Guidelines for Canadian Drinking Water Quality. Federal–Provincial–Territorial Committee on Drinking Water, March 2007.

HEI. (Health Effects Institute)., 1987. Automotive Methanol Vapors and Human Health: An Evaluation of existing Scientific Information and Issues for Future Research. Boston, MA.

Hogan, A.C., J.L. Stauber, F. Pablo, M.S. Adams, and R.P. Lim, 2005. The development of marine Toxicity Identification Evaluation (TIE) procedures using the unicellular alga *Nitzschia closterium*. Arch. Environ. Contam. Toxicol.48(4): 433-443.

Howard, P.H., Boethling, R.S., Jarvis, W.F., Meylan, W.M., and Michalenko, E.M., 1991. Handbook of Environmental Degradation Rates. Lewis Publishers Inc. Michigan, USA.

HSDB Hazardous Substances Data Bank. 2012. Bethesda (MD): U.S. National Library of Medicine. Available from: http://toxnet.nlm.nih.gov/

Infurna, R., & Weiss, B., 1986. Neonatal behavioral toxicity in rats following prenatal exposure to methanol. Teratology, **33**, 259-265.

IPCS. (International Programme on Chemical Safety)., 1997. Methanol. Geneva, Switzerland: World Health Organization.

Jonsson A., Persson K.A., and Grigoriadis V., 1985. Measurements of some low molecular-weight oxygenated, aromatic and chlorinated hydrocarbons in ambient air and in vehicle emissions. *Environment International*, **11**, 383-392.

Jungclaus G.A., Lopez-Avila, V., and Hites, R.A., 1978. Organic compounds in an industrial wastewater: A case study of their environmental impact. *Environmental Science and Technology*, **12**, 88-96.

Katoh, M., 1989. New Energy Development Organization data. Presented at the Methanol Vapors and Health Effects Workshop: What we know and what we need to know - Summary Report. Washington, DC, ILSI Risk Science Institute/US Environmental Protection Agency/Health Effects Institute/American Petroleum Institute, p A-7.

Kavet, R., and Nauss K., 1990. The toxicity of inhaled methanol vapors. *Critical Reviews in Toxicology*. **21**:21–50.

Kaviraj, A., F. Bhunia, and N.C. Saha, 2004. Toxicity of methanol to fish, crustacean, oligochaete worm, and aquatic ecosystem. Int. J. Toxicol.23:55-63.

Kirk-Othmer, 1999. Encyclopedia of Chemical Technology. Fourth Edition, 1999. John Wiley & Sons.

Koprivnikar, J., and P.A. Walker, 2011. Effects of the herbicide atrazine's metabolites on host snail mortality and production of *Trematode cercariae*. J. Parasitol.97(5): 822-827.

Kwok, E.S.C, Atkinson, R. 1995. Estimation of hydroxyl radical reaction rate constants for gas-phase organic compounds using a structure-reactivity relationship: an update. Atmos. Environ. 29(14):1685-1695.

Lewis R.J., Sr, 1989. Food Additives Handbook. New York, Van Nostrand Reinhold Co., pp 291-292.

Lewis, R. J., Sr., 1992. Sax's Dangerous Properties of Industrial Materials: Vol. III (8th ed.). New York, NY: Van Nostrand Reinhold.

Lijinsky, W., Thomas, B.J., and Kovatch, R.M., 1991. Differences in skin carcinogenesis by methyl nitroso urea between mice of several strains. *Cancer Letters*, **61**: 1-5.

Linden, E., B.E. Bengtsson, O. Svanberg, and G. Sundstrom, 1979. The acute toxicity of 78 chemicals and pesticide formulations against two brackish water organisms, the Bleak (*Alburnus alburnus*) and the harpacticoid *Nitocra spinipes*. *Chemosphere* **8(11-12):**843-851.

Lindinger, W; Taucher, J; Jordan, A; Hansel, A; Vogel, W. (1997). Endogenous production of methanol after the consumption of fruit. *Alcoholism, Clinical and Experimental Research* 21: 939-943.

Lv, X., J. Shao, M. Song, Q. Zhou, and G. Jiang, 2006. Vitellogenic Effects of 17beta-estradiol in male chinese loach (*Misgurnus anguillicaudatus*). Comp. Biochem. Physiol. C Comp. Pharmacol. Toxicol.143(1): 127-133.

Lyman W.J., Reehl, W.F., and Rosenblatt, D.H., 1982. Handbook of Chemical Property Estimation Methods, Environmental Behavior of Organic Compounds. American Chemical Society, Washington, DC.

Mackay, D., Shiu, W.Y., Ma, K.-C. and Lee, S.C. 2006. Physical-Chemical Properties and Environmental Fate for Organic Chemicals, Volume 3. Oxygen Containing Compounds. CRC Press, Boca Raton.

Majchrowicz, E. and Mendelson. J.H., 1971. Blood methanol concentrations during experimentally induced ethanol intoxication in alcoholics. *J Pharmacol Exp Ther*, **179**: 293-300.

Mohr, D.H. and King, C.J., 1985. Identification of polar organic compounds in coal-gasification condensate water by gas-chromatography-mass spectrometry analysis of high-performance liquid chromatography. *Environmental Science and Technology*, **19**, 929-935.

Montgomery, J.H., 1991. Groundwater Chemicals Desk Reference. Lewis Publishers, Chelsea, MI, USA.

MMSA, 2013. Methanol Supply and Demand 2008 to 2013E. Methanol Market Services Asia. http://www.methanol.org/Methanol-Basics.aspx

Morley, N.J., K.M.Y. Leung, D. Morritt, and M. Crane, 2004. Toxicity of anti-fouling biocides to encysted metacercariae of *Echinoparyphium recurvatum* (Digenea: Echinostomatidae) and their snail hosts. Chemosphere56(4): 353-358.

NEDO (New Energy Development Organization), 1987. Toxicological research of methanol as a fuel for power station: Summary report on tests with monkeys, rats and mice. Tokyo, Japan, New Energy Development Organization, pp 1-296.

Nelson, B.K., Brightwell, W.S., and MacKenzie, D.R., 1985. Teratological assessment of methanol and ethanol at high inhalation levels in rats. *Fundamental and Applied Toxicology*, **5**:727–36.

NHMRC (National Health and Medical Research Council), 1996. Australian Drinking Water Guidelines. National Water Quality Management Strategy. Canberra. ISBN 0 642 24462 6

Nice, H.E., 2005. Sperm motility in the Pacific Oyster (*Crassostrea gigas*) is affected by nonylphenol. Mar. Pollut. Bull.50(12): 1668-1674.

OECD Organisation for Economic Cooperation and Development. 2004. SIDS Initial Assessment Report. Methanol. SIAM 19, 18-20 October 2004. Available from: http://webnet.oecd.org/Hpv/UI/SIDS_Details.aspx?id=39B5D34A-2F5D-4D53-B000-E497B3A3EE89

Okumura, Y., J. Koyama, H. Takaku, and H. Satoh, 2001. Influence of organic solvents on the growth of marine microalgae. Arch. Environ. Contam. Toxicol.41(2): 123-128.

OMEE (Ontario Ministry of the Environment and Energy). 1994. Water management Policies Guidelines Provincial Water Quality Objectives of the Ministry of Environment and Energy. PIBS 3303E. Queen's Printer for Ontario, Toronto, Ontario.

ORNL (Oak Ridge National Laboratory), 2007. Risk Assessment Information System. On-Line Database available at http://rais.ornl.gov/

Poon, R., Chu, I., Bjarnason, S., Potvin, M., Vincent, R., Miller, R. B., & Valli, V. E., 1994. Inhalation toxicity study of methanol, toluene, and methanol/toluene mixtures in rats: effects of 28-day exposure. *Toxicology and Industrial Health*, **10**, 231-245.

Poon, R., Chu, I., Bjarnason, S., Vincent, R., Potvin, M., Miller, R. B., & Valli, V. E., 1995. Short-term inhalation toxicity of methanol, gasoline, and methanol/gasoline in the rat. *Toxicology and Industrial Health*, **11**, 343-361.

Pounds, N., S. Maclean, M. Webley, D. Pascoe, and T. Hutchinson, 2008. Acute and chronic effects of Ibuprofen in the mollusc *Planorbis carinatus* (Gastropoda: Planorbidae). Ecotoxicol. Environ. Saf.70(1): 47-52.

Rahman, S.M., S.K. Majhi, T. Suzuki, S. Matsukawa, C.A. Strussmann, and R. Takai, 2008. Suitability of cryoprotectants and impregnation protocols for embryos of Japanese Whiting *Sillago japonica*. Cryobiology57(2): 170-174.

Reynolds, T., 1977. Comparative effects of aliphatic compounds on inhibition of lettuce fruit germination. *Annals of*

Botany, **41**: 637-648.
Roberts, B.L., and Dorough, H.W., 1984. Relative toxicities of chemicals to the earthworm *Eisenia foetida*. *Environmental Toxicology and Chemistry*, **3(1)**: 67-78.
Rogers, J. M., & Mole, M. L., 1997. Critical periods of sensitivity to the developmental toxicity of inhaled methanol in the CD-1 mouse. *Teratology*, **55**, 364-372.
Rogers, J.M., Mole, M.L., and Chernoff, N., 1993. The developmental toxicity of inhaled methanol in the CD-1 mouse, with quantitative dose–response modeling for estimation of benchmark doses. *Teratology*, **47**:175–88.
Sakanashi, T. M., Rogers, J. M., Fu, S. S., Connelly, L. E., & Keen, C. L., 1996. Influence of maternal folate status on the developmental toxicity of methanol in the CD-1 mouse. *Teratology*, **54**, 198-206.
Sayers, R.R., Yant, W.P., & Schrenk, H.H., 1942. Methanol poisoning exposure of dogs to 450-500 ppm methanol vapour in air. Washington, DC, US Bureau of Mines (Investigation Report No. 3619).
Sedivec, V., Mraz, M., and Flek, J., 1981. Biological monitoring of persons exposed to methanol vapors. *International Archives of Occupational and Environmental Health*, **48,** 257-271.
Snider, J.R. and Dawson, G.A., 1985. Tropospheric light alcohols, carbonyls and acetonitrile: Concentrations in the Southwestern United States and Henry's law data. *Journal of Geophysical Research*, **90,** 3797-3805.
Soffritti, M., Belpoggi, F., Cevolani, D., Guarino, M., Padovani, M., & Maltoni, C., 2002. Results of long-term experimental studies on the carcinogenicity of methyl alcohol and ethyl alcohol in rats. In M. A. Mehlman (Ed.), Carcinogenesis Bioassays and Protecting Public Health: Commemorating on the Lifework of Cesare Maltoni and Colleaques (pp. 46-69). Bologna, Italy: Ann. N. Y. Acad. Sci.
Stantec (Stantec Consulting Ltd.), 2006. Ecotoxicity Assessment of Amines, Glycols, and Methanol to Soil Organisms. Report prepared for Petroleum Technology Alliance Canada and available at www.ptac.org.
Stanton, M.E., Crofton, K.M., Gray, L.E., Gordon, C.J., Boyes, W.K., Mole, M.L., Peele, D.B. and Bushnell, P.J. 1995. Assessment of offspring development and behavior following gestational exposure to inhaled methanol in the rat. Fund. Appl. Toxicol. 28(1): 100-110
Stiles, W., and Stirk, M.L.L., 1931. Studies on toxic action. II. The toxicity of normal aliphatic alcohols towards potato tuber. III. The parallelism between surface activity and toxicity of normal monohydric alcohols. *Protoplasma*, **13**:1-20.
Suedel, B.C., T.M. Dillon, and W.H. Benson, 1997. Subchronic effects of five di-ortho PCB congeners on survival, growth and reproduction in the fathead minnow *Pimephales promelas*. Environ. Toxicol. Chem.16(7): 1526-1532.
Tephly, T.R., and McMartin, K.E., 1984. Methanol metabolism and toxicity. In: Stegink LD & Filer LJ Jr ed. Aspartame: Physiology and biochemistry. New York, Basel, Marcel Dekker, pp. 111-140.
Tien, C.J., and C.S. Chen, 2012. Assessing the toxicity of organophosphorous pesticides to indigenous algae with implication for their ecotoxicological impact to aquatic ecosystems. J. Environ. Sci. Health Part B: Pestic. Food Contam. Agric. Wastes47(9): 901-912.
U.S. EPA (United States Environmental Protection Agency), 1976. Assessment of Methyl Alcohol as a Potential Air Pollution Problem - Volume II. Research Triangle Park, North Carolina, US Environmental Protection Agency, (NTIS Publication No. PB-258354).
U.S. EPA, 1977. Multimedia Environmental Goals for Environmental Assessment - Volume II: MEG, Charts and Background Information. Washington, DC, US Environmental Protection Agency, pp E28-E29 (EPA 600/7-77-126b).
U.S. EPA, 1986. Rat Oral Subchronic Toxicity Study With Methanol. Office of Solid Waste, Washington, DC.
U.S. EPA (United States Environmental Protection Agency), 1993. Ambient Concentration Summaries for Clean Air Act, Title III: Hazardous Air Pollutants. Washington, DC, US Environmental Protection Agency, p B-17 (EPA 600/R-94-090).
U.S. EPA, 1996. Guidelines for Reproductive Toxicity Risk Assessment. (Report No. EPA/630/R-96/009). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum.
U.S. EPA, 2002. National Recommended Water Quality Criteria: 2002. Report # EPA-822-r-02-047.
U.S. EPA, 2004a. National Primary Drinking Water Standards. EPA 816-F-03-016 June 2003.
U.S. EPA, 2004b. Test Methods for Evaluating Solid Wastes – Physical/Chemical Methods. EPA Publication SW-846.
U.S. EPA, 2013. Toxicological Review of Methanol (Non-Cancer) In support of summary information on the integrated risk information system (IRIS). September 2013. EPA/635/R-11/001Fa.
U.S. EPA, 2014a. ECOTOX Online Database. Release 4.0. http://cfpub.epa.gov/ecotox.
U.S. EPA, 2014b. Integrated Risk Information System (IRIS) On-Line Database.

Van der Zandt, P.T.J., F. Heinis, and A. Kikkert, 1994b. Effects of narcotic industrial pollutants on behaviour of midge larvae (*Chironomus riparius* (Meigen), Diptera): A quantitative structure-activity relationship. Aquat. Toxicol.28(3-4): 209-221.

Venkataraman, E.S., Ahlert, R.C., and Corbo, P., 1984. Biological treatment of landfill leachates. CRC *Critical Reviews in Environmental Control*, **14,** 333-376.

Verschueren, K., 2001. Handbook of Environmental Data on Organic Chemicals. Fourth Edition. Wiley-Interscience.

VROM (Netherlands Ministry of Housing, Spatial Planning and the Environment), 2000. Circular on Target Values and Intervention Values for Soil Quality (plus annexes). Available online at http://www.esdat.net/Environmental%20Standards/Dutch/annexS_I2000Dutch%20Environmental%20Standards.pdf.

Vuthiphandchai, V., B. Pengpun, and S. Nimrat, 2005. Effects of cryoprotectant toxicity and temperature sensitivity on the embryos of Black Tiger Shrimp (*Penaeus monodon*). Aquaculture246(1-4): 275-284.

Weiss, B., Stern, S., Soderholm, S. C., Cox, C., Sharma, A., Inglis, G. B., Gelein, R., 1996. Developmental Neurotoxicity of Methanol Exposure by Inhalation in Rats. (Report No. HEI Research Report Number 73). Boston, MA: Health Effects Institute.

Werl Treatability Database, 1993. Environmental Protection Agency. Citation in GRI (1996) used for selected chemical/ physical properties for methanol.

Werner, I., L.A. Deanovic, V. Connor, V. De Vlaming, H.C. Bailey, and D.E. Hinton, 2000. Insecticide-caused toxicity to *Ceriodaphnia dubia* (Cladocera) in the Sacramento-San Joaquin River delta, California, USA. Environ. Toxicol. Chem.19(1): 215-227.

White LR, Martinsen ABL, & Nilsen OG (1983) Biochemical and cytological studies of rat lung after inhalation of methanol vapour. *Toxicology Letters*, **17**: 1-5.

White, K.L., B.E. Haggard, M.D. Matlock, and J.W. Kim, 2005. Periphytic chlorophyll -- A response to triclosan xxposure: Application of a passive diffusion periphytometer. Appl. Eng. Agric.21(2): 307-311.

WHO (World Health Organization), 1997. Environmental Health Criteria 196—Methanol. Geneva.

WHO (World Health Organization), 2004. WHO Guidelines for Drinking Water Quality, Third Edition. ISBN 92 4 154638 7. Available online at

Wucherpfennig, K., Dietrich, H., and Bechtel, J., 1983. Alcohol actual, total and potential methyl alcohol of fruit juices. *Flussiges Obst*, **8**: 348-354.

APPENDICES

Appendix 1. Summary of Available Information on Methanol Biodegradation

Test Method	Test Duration	Initial Compound Concentra-tion	% Re-moved	Inoculum or Medium	Rates / Comments	Reference
Definitive Groundwater Study						
Aquifer Study	500 days	7034	100%	groundwater	half-life = 245 days (see Section 3.4)	API (1994)
Other Data						
NV	NV	NV	NV	soil	half-life in soil: 1-7 days	Howard et al. (1991)
NV	NV	NV	NV	groundwater	half-life in groundwater: 1-7 days	Howard et al. (1991)
NV	NV	NV	NV	surface water	half-life in surface water: 1-7 days	Howard et al. (1991)
BOD_5	5 days	NV	48%	NV	ThOD	Verschueren (2001)
BOD_5	5 days	NV	53%	NV	ThOD	Verschueren (2001)
BOD_5	5 days	NV	75%	NV	ThOD	Verschueren (2001)
BOD_5	5 days	NV	69%	NV	ThOD	Verschueren (2001)
BOD_5	5 days	500-1500 mg/L	9%	10% sewage	ThOD	Verschueren (2001)
BOD_5	5 days	1-1000 mg/L	40-73%	NV	ThOD	Verschueren (2001)
BOD_5	5 days	NV	51-57%	NV	ThOD	Verschueren (2001)
BOD_5	5 days	NV	51%	sewage	ThOD	Verschueren (2001)
BOD_5	5 days	NV	75%	sewage	ThOD	Verschueren (2001)
BOD_5	5 days	NV	83%	sewage	ThOD	Verschueren (2001)
BOD_5	5 days	6000 mg/L	83%	NV	ThOD	Verschueren (2001)
BOD_5	5 days	6000 mg/L	96%	NV	ThOD	Verschueren (2001)
BOD_5	5 days	NV	62%	NV	ThOD; aclimated	Verschueren (2001)
BOD_5	5 days	10 mg/L	75%	unadapted sewage	ThOD; lag period = 1 day	Verschueren (2001)
BOD_{10}	10 days	2.5 mg/L	63%	sewage	ThOD	Verschueren (2001)
BOD_{10}	10 days	NV	63%	NV	ThOD	Verschueren (2001)
BOD_{10}	10 days	NV	88%	NV	ThOD	Verschueren (2001)
BOD_{10}	10 days	NV	84%	NV	ThOD	Verschueren (2001)
BOD_{15}	15 days	NV	69%	NV	ThOD	Verschueren (2001)
BOD_{15}	15 days	NV	91%	NV	ThOD	Verschueren (2001)

BOD_{15}	15 days	NV	85%	NV	ThOD	Verschueren (2001)
BOD_{20}	20 days	NV	67%	NV	ThOD	Verschueren (2001)
BOD_{20}	20 days	NV	95%	NV	ThOD	Verschueren (2001)
BOD_{20}	20 days	NV	97%	NV	ThOD	Verschueren (2001)
BOD_{20}	20 days	NV	84%	NV	ThOD	Verschueren (2001)
BOD_{20}	20 days	NV	79%	unadapted sewage	ThOD; lag period = 1 day	Verschueren (2001)
BOD_{30}	30 days	NV	69%	NV	ThOD	Verschueren (2001)
BOD_{40}	40 days	NV	93%	NV	ThOD	Verschueren (2001)
BOD_{50}	50 days	NV	98%	NV	ThOD	Verschueren (2001)
aerobic	5 days	0.1 mg/L	53%	soil-water suspension	mineralization to CO_2	Verschueren (2001)
anaerobic	5 days	0.1 mg/L	46%	soil-water suspension	mineralization to CO_2	Verschueren (2001)
ammonium oxidation inhibition test	NV	800 mg/L	NV	sludge digestion by *Nitrosomas*	IC_{50} for oxidation of NH_3	Verschueren (2001)
oxygen consumption inhibition test	NV	72,000 mg/L	NV	municipal sludge	IC_{50} for oxygen consumption	Verschueren (2001)
oxygen consumption inhibition test	NV	80,000 mg/L	NV	industrial sludge	IC_{50} for oxygen consumption	Verschueren (2001)
respiration inhibition test	3 hour	>1,000 mg/L	NV	activated sludge	IC_{50} for respiration	Verschueren (2001)
bacterial growth inhibition test	16 hour	>5,000 mg/L	NV	sludge digestion by *Nitrosomas*	IC_{50} for oxygen consumption	Verschueren (2001)

[a]Biochemical oxygen demand (BOD) is defined as parts of oxygen consumed per part of compound during degradation. This value is expressed as a percentage of the theoretical (ThOD) oxygen demand.

NV = not reported in the abstract and not verified in this literature search

Appendix 2. Toxicity of Methanol to Terrestrial Plants

Scientific Name	Common Name	Effect Measurement	Concentration	Endpoint/ Response	Response Site	Test Duration	Media Type	Application Method	Chemical Analysis	Reference
						days				
Data Relevant for Guideline Development										
Medicago sativa	Alfalfa	Length	2,213	EC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Medicago sativa	Alfalfa	Length	7,945	EC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Medicago sativa	Alfalfa	Dry Mass	1,808	EC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Medicago sativa	Alfalfa	Dry Mass	3,209	EC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Length	4,886	EC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Length	5,752	EC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Dry Mass	2,538	EC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Dry Mass	2,823	EC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Length	4,149	EC25	shoot	21	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Length	12,202	EC25	root	21	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Dry Mass	2,877	EC25	shoot	21	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Dry Mass	3,635	EC25	root	21	artificial soil	spiked	Y	Stantec (2006)

Scientific Name	Common Name	Effect Measurement	Concentration	Endpoint/ Response	Response Site	Test Duration	Media Type	Application Method	Chemical Analysis	Reference
						days				
Data Not Relevant for Guideline Development										
Allium cepa	Common onion	Growth	19,300 mg/L	EC50	NV	6	aqueous	NV	NV	Fiskesjo (1985)
Lactuca sativa	Lettuce	Germination	40,850 mg/L	EC50	NV	3	agar	NV	NV	Reynolds (1977)
Gossypium hirsutum	Cotton	Damage	25 uL	no change compared to control	fruit	0.21	culture medium	injection	NV	Guinn (1977)
Gossypium hirsutum	Cotton	ethylene production	26 uL	50% of control	fruit	0.21	culture medium	injection	NV	Guinn (1977)
Solanum tuberosum	Potato	Damage	32,040 mg/L	no change compared to control	cell	0.01	NV	soaked	NV	Stiles and Stirk (1931)
Solanum tuberosum	Potato	Damage	64,080 mg/L	no change compared to control	cell	0.01	NV	soaked	NV	Stiles and Stirk (1931)
Glycine max	Soybean	Biomass	1,922 mg/L	50% of control	shoot	4.08	NV	soaked	NV	Eisenmenger (1930)
Glycine max	Soybean	Biomass	10,000 mg/L	49% of control	cell	11	culture medium	soaked	NV	Davis et al. (1978)
Glycine max	Soybean	Biomass	20,000 mg/L	82% of control	cell	11	culture medium	soaked	NV	Davis et al. (1978)
Glycine max	Soybean	Biomass	5,000 mg/L	13% of control	cell	11	culture medium	soaked	NV	Davis et al. (1978)
Glycine max	Soybean	Size	1,922 mg/L	18% of control	root	4.08	NV	soaked	NV	Eisenmenger (1930)
Daucus carota	Wild carrot	Biomass	20,000 mg/L	20% of control	cell	14	culture medium	soaked	NV	Davis et al. (1978)
Daucus carota	Wild carrot	Biomass	10,000 mg/L	27% of control	cell	7	culture medium	soaked	NV	Davis et al. (1978)

Scientific Name	Common Name	Effect Measurement	Concentration	Endpoint/ Response	Response Site	Test Duration	Media Type	Application Method	Chemical Analysis	Reference
						days				
Daucus carota	Wild carrot	Biomass	20,000 mg/L	13% of control	cell	7	culture medium	soaked	NV	Davis et al. (1978)

Notes: NV = not reported in the abstract and not verified in this literature search

Appendix 3. Toxicity of Methanol to Terrestrial Invertebrates

Scientific Name	Common Name	Effect Measurement	Concentration	Endopint/Response	Test Duration	Media Type	Application Method	Chemical Analysis	Reference
					days				
Data Relevant for Guideline Development									
Eisenia andrei	Earthworm	adult survival	17,199	EC50	35	artificial soil	spiked	Y	Stantec (2006)
Eisenia andrei	Earthworm	# progeny	13,323	EC25	63	artificial soil	spiked	Y	Stantec (2006)
Eisenia andrei	Earthworm	progeny mass	9,756	EC25	63	artificial soil	spiked	Y	Stantec (2006)
Folsomia candida	Springtail	# progeny	2,842	EC25	28	artificial soil	spiked	Y	Stantec (2006)
Data Not Relevant for Guideline Development									
Eisenia fetida	earthworm	Mortality	>1,000 ug/cm^2	LC50	2	filter paper	direct application	NV	Roberts and Dorough (1984)

Notes:

NV = not reported in the abstract and not verified in this literature search

Appendix 4 . Toxicity of Methanol to Freshwater Aquatic Life

Rank	Scientific Name	Common Name	Endpoint	Effective Concentration (mg/L)	Data Quality	Reference	Comment
1	*Oreochromis mossambicus*	Mozambique Tilapia	90 day MATC (growth)	33.6	S[c]	Kaviraj et al., (2004)	MATC calculated from NOEC and LOEC in report.
2	*Planorbis carinatus*	Gekielte Plate Snail	21 day NOEC (mortality)	79.1[b]	S	Pounds (2008)	MeOH used as a solvent control in this study on ibuprofen toxicity. This value is an unbounded NOEC.
3	*Algae* [a]	Algae	4 day IC10 (abundance)	396	S	Tien and Chen (2012)	Algae cultured, isolated and grown from Taiwan river water.
4	*Scenedesmus quadricauda*	Alga	10 day MATC (abundance)	1,110	S	Abou-Waly (2000)	MATC calculated from NOEC and LOEC in report.
5	*Pseudokirchneriella subcapitata*	Alga	4 day IC10 (abundance)	1,582	S	Garrett (2004)	Mean of 6 replicate tests
6	*Ceriodaphnia dubia*	Water Flea	7 day NOEC (mortality)	2,610[b]	S	Werner et al. (2000)	MeOH used as a solvent control in this study on insecticide toxicity. This value is an unbounded NOEC.
7	*Oryzias latipes*	Japanese Medaka	200 hour MATC (hatching success)	5,616	S	Gonzales-Doncel et al. (2008)	Hatching success was the most sensitive of several endpoints investigated.

Rank	Scientific Name	Common Name	Endpoint	Effective Concentration (mg/L)	Data Quality	Reference	Comment
8	*Oncorhynchus keta*	Chum Salmon	NOEC (fertilization to hatching)	7,910	S	Craig et al. (1977)	Eggs exposed over the sensitive period from fertilization until "water hardening", development observed until hatching.
9	*Chironomus riparius*	Midge	4 day NOEC (behaviour)	10,253	S	Van der Zandt (1994)	Study looked at changes in patterns of feeding and ventilating behaviour.
10	*Alga*	Alga	7 day NOEC (abundance)	15,820[b]	S	White (2005)	MeOH used as a solvent control in this study on triclosan toxicity. This value is an unbounded NOEC.

Notes:
(a) equal mixture of Nitzschia sp., Oscillatoria sp. and Chlorella sp.
(b) data from single concentration study; no LOEC available
(c) S – secondary data, as per CCME (2007)

Appendix 5. Toxicity of Methanol to Marine Aquatic Life

Rank	Scientific Name	Common Name	Endpoint	Effective Concentration (mg/L)	Data Quality	Reference
1	*Ulva pertusa*	Alga	4 day NOEC (spore production)	10	S	Han et al. (2008)
2	*Eutreptiella sp.*	Alga	4 day NOEC (abundance)	24	S	Okumura (2001)
3	*Heterosigma akashiwo*	Alga	4 day NOEC (abundance)	71	S	Okumura (2001)
4	*Prorocentrum minimum*	Alga	4 day NOEC (abundance)	410	S	Okumura (2001)
5	*Skeletonema costatum*	Alga	4 day NOEC (abundance)	1,400	S	Okumura (2001)
6	*Robertsonia propinqua*	Marine Harpacticoid Copepod	4 day NOEC (mortality)	1,978	S	Hack et al. (2008)
7	*Chaetoceros calcitrans*	Alga	4 day NOEC (abundance)	5,600	S	Okumura (2001)
8	*Pavlova lutheri*	Alga	4 day NOEC (abundance)	5,700	S	Okumura (2001)
9	*Isochrysis galbana*	Alga	4 day NOEC (abundance)	8,100	S	Okumura (2001)
10	*Dunaliella tertiolecta*	Alga	4 day NOEC (abundance)	10,000	S	Okumura (2001)
11	*Tetraselmis tetrathele*	Alga	4 day NOEC (abundance)	14,000	S	Okumura (2001)
12	*Nitzschia closterium*	Diatom	2 day MATC (abundance)	27,400	S	Hogan et al. (2005)
13	*Penaeus monodon*	Jumbo Tiger Prawn	Until hatch LOEC (mortality)	39,550	S	Vuthiphandchai et al. (2005)
14	*Pagrus major*	Red Sea Bream	Until hatch LOEC (mortality)	79,100	S	Ding et al. (2007)
15	*Sillago japonica*	Japanese Whiting	Until hatch LOEC (mortality)	79,100	S	Rahman et al. (2008)

Appendix 6. Toxicity of Methanol to Mammalian Experimental Animals

Species, strain, number/sex	Dose/duration	NOAEL (mg/kg/day)	LOAEL (mg/kg/day)	Effect	Reference
Chronic and Subchronic Toxicity Studies - Oral					
Rat: Sprague-Dawley; 30/sex/group	0, 100, 500, and 2500 mg/kg-day for 13 wk	500	2500	Reduction of brain weights, increase in the serum activity of ALT and AP. Increased liver weights	U.S. EPA (1986b)
Rat: Sprague-Dawley; 100/sex/group	0, 500, 5000, or 20 000 ppm (v/v) in drinking water, for 104 wk. Doses were approx. 0, 46.6, 466, and 1872 mg/kg-day (male) and 0, 52.9, 529, and 2101 mg/kg- day (female)	ND	ND	No noncancer effects were reported	Soffritti et al. (2002)
Mouse: Swiss	560, 1000 and 2100 mg/kg/d (female) and 550, 970, and 1800 mg/kg/d (male), 6 days/wk for life	ND	1800-2100	Increased incidence of liver parenchymal cell necrosis	Apaja (1980)
Reproductive/Developmental Toxicity Studies - Oral					
Rat: Long-Evans; 10 pregnant females/group	0 and 2500 mg/kg-day on either GD15-GD17 or GD17-GD19.	NA	2500	Neurobehavioral deficits (such as homing behavior, suckling ability	Infurna and Weiss (1986)
Mouse CD-1; 8 pregnant females and 4 controls	4 g/kg-day in 2 daily doses on GD6-GD15	NA	4000	Increased incidence of totally resorbed litters, cleft palate and exencephaly. A decrease in the number of live fetuses/litter	Rogers et al. (1993)

Species, strain, number/sex	Dose/duration	NOAEL (mg/kg/day)	LOAEL (mg/kg/day)	Effect	Reference
Chronic and Subchronic Toxicity Studies - Inhalation					
Monkey; *M. fascicularis*; 1 or 2 animals/group	0, 3000, 5000, 7000, or 10 000 ppm, 21 hr/day, for up to 14 days	ND	ND	Clinical signs of toxicity, CNS changes, including degeneration of the bilateral putamen, caudate nucleus, and claustrum. Edema of cerebral white matter.	NEDO (1987)
Dog (2)	10 000 ppm for 3 min, 8 times/day for 100 days	NA	NA	None	Sayers et al. (1944)
Rat; Sprague-Dawley; 5 males/ group	0, 200, 2000, or 10 000 ppm, 8 hr/day, 5 days/wk for up to 6 wk	NA	200	Transient reduction in plasma testosterone levels	Cameron et al. (1984)
Rat; Sprague-Dawley; 5 males/ group	0, or 200 ppm, 6 hr/day, for either 1 or 7 days	NA	200	Transient reduction in plasma testosterone levels	Cameron et al. (1985)
Rat: Sprague-Dawley; 5/sex/group	0, 500, 2000, or 5000 ppm, 5 days/wk for 4 wk	5000	NA	No compound-related effects	Andrews et al. (1987)
Monkey: *M. fascicularis;* 3/sex/group	0, 500, 2000, or 5000 ppm, 5 days/wk for 4 wk	5000	NA	No compound-related effects	
Rat: Sprague-Dawley; 10/sex/group	0, 300, or 3 000 ppm, 6 hr/day, 5 days/wk for 4 wk	NA	300	Reduction in size of thyroid follicles	Poon et al. (1994)
Rat: Sprague-Dawley; 15/sex/group	0 or 2500 ppm, 6 hr/day, 5 days/wk for 4 wk	NA	2500	Reduction of relative spleen weight in females, histopathologic changes to the liver, irritation of the upper respiratory tract	Poon et al. (1995)
Monkey: *M. fascicularis;* 2 or 3 animals/ group/time point	0, 10, 100, or 1000 ppm, 21 hr/day for either 7, 19, or 29 mo	ND ND	ND ND	Limited fibrosis of the liver. Possible myocardial and renal effects	NEDO (1987)

Species, strain, number/sex	Dose/duration	NOAEL (mg/kg/day)	LOAEL (mg/kg/day)	Effect	Reference
Rat F344; 20/sex/group	0, 10, 100, or 1000 ppm, 20 hr/day, for 12 mo	NA	NA	No compound-related effects	
Mouse: B6C3F1; 30/sex/group	0, 10, 100, or 1000 ppm, 20 hr/day, for 12 mo	NA	NA	No clear-cut compound-related effects	
Mouse: B6C3F1; 52-53/sex/group	0, 10, 100, or 1000 ppm, 20 hr/day, for 12 mo	100	1000	Increase in kidney weight, decrease in testis and spleen weights	
Rat: F344; 52/sex/group	0, 10, 100, or 1000 ppm, ~20 hr/day for 2 yr	100	1000	Fluctuations in a number of urinalysis, hematology, and clinical chemistry parameters.	
Reproductive/Developmental Toxicity Studies - Inhalation					
Rat: Sprague-Dawley; 15/pregnant females/group	0, 5000, 10 000, or 20 000 ppm, 7 hr/day on either GD1-GD19 or GD7-GD15.	5000	10 000	Reduced fetal body weight, increased incidence of visceral and skeletal abnormalities, including rudimentary and extra cervical ribs	Nelson et al. (1985)
Rat: Sprague-Dawley; 36/pregnant females/group	0, 200, 1000, or 5000 ppm, 22.7 hr/day, on GD7-GD17	1000	5000	Late-term resorptions, reduced fetal viability, increased frequency of fetal malformations, variations and delayed ossifications.	NEDO (1987)
Rat: Sprague-Dawley F1 and F2 generations of a two-generation study	0, 10, 100, or 1000 ppm, 20 hr/day. F1: birth to end of mating (M) or weaning (F); F2- birth to 8 wks	100	1000	Reduced weight of brain, pituitary, and thymus at 8, 16 and 24 wk postnatal in F1 and at 8 wk in F2	
Rat: Sprague-Dawley Follow-up study of brain weights in F1 generation of 10-14/sex/group in F1 generation	0, 500, 1000, and 2000 ppm; GD0 through F1 generation	500	1000	Reduced brain weight at 3 wk and 6 wk (males only). Reduced brain and cerebrum weight at 8 wk (males only)	

Species, strain, number/sex	Dose/duration	NOAEL (mg/kg/day)	LOAEL (mg/kg/day)	Effect	Reference
Mouse: CD-1; 30-114 pregnant females/group	0, 1000, 2000, 5000, 7500, 10 000, or15 000 ppm, 7 hr/day on GD6-GD15.	1000	2000	Increased incidence of extra cervical ribs, cleft palate, exencephaly; reduced fetal weight and pup survival, Delayed ossification	Rogers et al. (1993)
Mouse: CD-1; 12-17 pregnant females/group	0 and 10 000 ppm on two consecutive days during GD6-GD13 or on a single day during GD5-GD9	NA	10 000	Cleft palate, exencephaly, skeletal malformations	Rogers and Mole (1997)
Rat: Long-Evans; 6-7 pregnant females/group	0 or 15 000 ppm, 7 hr/day on GD7-GD19	NA	15 000	Reduced pup weight	Stanton et al. (1995)
Rat: Long-Evans; 10-12 pregnant females/group	0 or 4500 ppm from GD10 to PND21.	NA	4500	Subtle cognitive deficits	Weiss et al. (1996)
Monkey: *M. fascicularis;* 12 monkeys/group	0, 200, 600, or 1800 ppm, 2.5 hr/day, 7 days/wk, during premating, mating and gestation	ND	NDa	Shortened period of gestation; may be related to exposure (no dose-response), neurotoxicological deficits, including reduced performance in the VDR test; may be related to premature births.	Burbacher et al. (1999a; 1999b; 2004a; 2004b)

Source: US EPA (2013);
ND = Not determined;
NA = Not applicable

Appendix 7. Chemical-Specific Parameter Values for Methanol

Parameter	Unit	Value	Rationale
Human Toxicity			
Tolerable Daily Intake (oral exposure)	mg/kg-bw/day	2	see Section 6.6
Tolerable Concentration (inhalation exposure)	mg/m^3	20	see Section 6.6
Human Background Exposure			
Estimated daily intake	mg/kg-bw/day	1.6	see Section 2.6
Ambient air concentration	mg/m^3	0.04	see Section 2.6
Background soil concentration	mg/kg	0	see Section 2.6
Soil allocation factor	-	0.2	see Section 9.1
Water allocation factor	-	0.2	see Section 9.1
Human Adsorption			
Absorption factor - gut	-	1.0	assumed
Absorption factor - skin	-	1.0	assumed
Absorption factor - lung	-	1.0	assumed
Chemical and Physical Properties			
Soil Organic Carbon/Water Partition Coefficient (Koc)	L/kg	0.27	see Table 2
Dimensionless Henry's law coeffcient	(mg/L)/(mg/L)	0.0002	see Table 2
Dynamic viscosity of vapour	g/cm.s	0.000173	CCME (2008)
Diffusion coefficient in air	cm^2/s	0.15	ORNL (2007)
Degradation			
Degradation half-life (saturated)	days	245	see Section 3.4

Appendix 8. Human Receptor Characteristics

Parameter	Symbol	Unit	Toddler	Adult
Body Weight	BW	kg	16.5	70.7
Air Inhalation Rate	IR	m^3/d	8.3	16.6
Soil Inhalation Rate	IR_S	kg/d	7.1×10^{-9}	1.2×10^{-8}
Water Ingestion Rate	WIR	L/d	0.6	1.5
Soil Ingestion Rate	SIR	kg/d	0.00008	0.00002
Skin Surface Area				
- Hands	SA_H	m^2	0.043	0.089
- Other	SA_O	m^2	0.258	0.25
Dermal Loading to Skin				
- Hands	DL_H	kg/m^2-event	0.001	0.001
- Other	DL_O	kg/m^2-event	0.0001	0.0001
Dermal Exposure Frequency	EF	events/d	1	1
Exposure Term, agricultural and residential/parkland	ET	-	1	1
Exposure Term, commercial and industrial	ET	-	0.2747	0.2747
Exposure Term, agricultural and residential/parkland	ET_1	-	1	1
Exposure Term, commercial and industrial	ET_1	-	0.6593	0.6593
Exposure Term, agricultural and residential/parkland	ET_2	-	1	1
Exposure Term, commercial and industrial	ET_2	-	0.4167	0.4167

Notes:

All parameter values from CCME (2006), except IR from Allen et al. (2008)

Appendix 9. Soil and Hydrogeological Parameters

Parameter	Symbol	Unit	Fine Soil	Coarse Soil
Soil Bulk Density	ρ_B	*kg/L*	1.4	1.7
Soil Total Porosity	θ_t	*cm^3/cm^3*	0.47	0.36
Soil Moisture-Filled Porosity	θ_w	*cm^3/cm^3*	0.168	0.119
Soil Vapour-Filled Porosity	θ_a	*cm^3/cm^3*	0.302	0.241
Soil Vapour-Filled Porosity in Floor Cracks	θ_a	*cm^3/cm^3*	0.47	0.36
Gravimetric Water Content	MC	*g/g*	0.12	0.07
Fraction of Organic Carbon	f_{oc}	*mass/mass*	0.005	0.005
Saturated Hydraulic Conductivity	K	*m/y*	32	320
Hydraulic Gradient	i	*m/m*	0.028	0.028
Recharge (Infiltration) Rate	I	*m/y*	0.2	0.28
Soil Permeability to Vapour Flow	k_v	*cm^2*	10^{-10}	$6x10^{-8}$

Notes:
All parameter values from CCME (2006)

Appendix 10. Site Characteristics

Parameter	Symbol	Unit	Value
Contaminant Source Width	Y	m	10
Contaminant Source Length	X	m	10
Contaminant Source Depth	Z	m	3
Distance to Surface Water	x	m	10
Distance to Potable Water User	x	m	0
Distance to Agricultural Water User	x	m	0
Distance from Contamination to Building Slab	L_T	cm	30
Depth to Groundwater (water table)	d	m	3
Depth of unconfined aquifer	d_a	m	5
Time since contaminant release	t	year	100

Notes:

All parameter values from CCME (2006)

Appendix 11. Building Parameters

Parameter	Symbol	Unit	Residential Basement	Residential Slab-on-Grade	Commercial Slab-on-Grade
Building Length	L_B	*cm*	1,225	1,225	2,000
Building Width	W_B	*cm*	1,225	1,225	1,500
Building Height (including basement)	H_B	*cm*	488	488	300
Area of Substructure	A_B	cm^2	$2.7x10^6$	$1.5x10^6$	$3.0x10^6$
Thickness of Floor Slab	L_{crack}	*cm*	11.25	11.25	11.25
Depth of Floor Slab Below Ground	Z_{crack}	*cm*	244	11.25	11.25
Distance from Source to Slab:	L_T	*cm*			
surface soil			30	30	30
subsoil			30	139	139
Crack Area	A_{crack}	cm^2	994.5	994.5	1,846
Crack Length	X_{crack}	*cm*	4,900	4,900	7,000
Air Exchange Rate	ACH	*exch/hr*	1	1	2
Pressure Differential	ΔP	$g/cm.s^2$	40	40	20

Notes:
All parameter values from CCME (2006)

Appendix 12. Flammable and Non-Flammable Methanol Concentrations in Soil

Sample #	Location	Distance Along Trench (m)	Date Collected	Flammability	Methanol (mg/kg)
19	Area 3 Trench	1m	14-Oct-05	flame	12,700
20	Area 3 Trench	2m	14-Oct-05	flame	15,900
21	Area 3 Trench	3m	14-Oct-05	flame	14,900
22	Area 3 Trench	3.5m	14-Oct-05	flame	9,310
23	Area 3 Trench	3.75m	14-Oct-05	flame	10,700
24	Area 3 Trench	4.0m	14-Oct-05	no flame	7,460
25	Area 3 Trench	4.5m	14-Oct-05	no flame	13,700
26	Area 3 Trench	5.0m	14-Oct-05	no flame	6,390
27	Area 3 Trench	6.0m	14-Oct-05	no flame	3,990
28	Area 3 Trench	7.0m	14-Oct-05	no flame	80
29	Area 3 Trench	8.0m	14-Oct-05	no flame	48
30	Area 3 Trench	9.0m	14-Oct-05	no flame	53