

Screening Assessment for the Challenge

**Decamethylcyclopentasiloxane
(D5)**

**Chemical Abstracts Service Registry Number
541-02-6**

**Environment Canada
Health Canada**

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Synopsis

Pursuant to section 74 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999), the Ministers of the Environment and of Health have conducted a screening assessment on decamethylcyclopentasiloxane (D5), Chemical Abstracts Service Registry Number 541-02-6. During the categorization process, this substance was identified as a high priority for screening assessment and included in the Ministerial Challenge because it had been initially found to meet the ecological categorization criteria for persistence, bioaccumulation potential and inherent toxicity (PBiT) to non-human organisms and it is known to be in commerce in Canada.

Although the categorization exercise did not determine D5 to be a priority for assessment of potential risks to human health, a human health assessment of D5 was also conducted due to its structure and use pattern similarity to D4, octamethylcyclotetrasiloxane (also known as D4), a high priority for assessment for both human health and ecological risks under CEPA 1999.

D5 is an industrial chemical which was not manufactured in Canada in 2006 in a quantity above the reporting threshold of 100 kg, but which is imported into the country as an essentially pure substance, in mixtures with other cyclic siloxanes, as a residual in silicone polymers, and in finished consumer products. From responses to a notice published under section 71 of CEPA 1999, it was determined that between 1 000 000 and 10 000 000 kg of D5 were imported into Canada in 2006.

D5 may be released to the environment from industrial processes such as blending, formulation and packaging operations, and from its use as an industrial defoamer and degreaser. It is also released from the use and disposal of personal care products. Air, wastewater, and agriculture soil are the principal receiving environmental media for D5 based on its physical-chemical properties and its use patterns.

In air, D5 is persistent with calculated atmospheric half-lives of more than 3 days. D5 has the potential to be transported over long-distances in the atmosphere. However, it has a low potential to be deposited in water or soil in remote regions. The hydrolysis half-lives for D5 under Canadian water conditions ranged from 1 to 733 days, indicating the substance is persistent under certain Canadian water conditions, especially in cooler and neutral water (5-10°C). D5 is also judged to be persistent in sediment, with half-lives of 49 to 588 days estimated under realistic Canadian sediment conditions (temperature of 5-25°C) based on information to structurally similar analogue, D4, indicating the substance may be persistent in sediment. D5 is not considered persistent in soil, based on evidence of clay-catalysed degradation, with dimethylsilanediol being the stable hydrolysis product. Therefore, D5 has been determined to meet the persistence criterion as set out in the *Persistence and Bioaccumulation Regulations*.

The empirical bioconcentration factor and modelled bioaccumulation factor are both above 5000, indicating D5 may have a high potential to accumulate in aquatic organisms.

However, data from a biomagnification study in fish and a biota-sediment accumulation study in invertebrates suggest that the bioaccumulation potential of D5 may be lower, possibly due to reduced bioavailability. Therefore, while D5 has the potential to accumulate in biota, it is not possible to conclude at this time that D5 meets the criterion for bioaccumulation as set out in the *Persistence and Bioaccumulation Regulations* based on consideration of the conflicting evidence from laboratory studies and predictive models.

Adverse effects from exposure to D5 in sediment-dwelling organisms were observed at a concentration of 160 mg/kg. The experimental toxicity data showed no adverse effects to pelagic aquatic organisms at concentrations up to 0.015 mg/L, its approximate water solubility limit. However, it is possible that toxicity may manifest at the solubility limit if sufficient exposure and sensitive species were present. Risk quotients derived from exposure scenarios involving discharges of D5 from both consumer use and industrial operations show that a total of 65 sites (~6.8%) evaluated across Canada have predicted environmental concentrations in water higher than the 0.015 mg/L predicted no-effect concentration, without the use of an application factor, for aquatic organisms. Considering the persistence of D5 under colder Canadian water conditions and its potential to bioaccumulate in biota, long-term environmental exposure to D5 may potentially cause adverse effects to aquatic organisms in certain Canadian environments.

Based principally on the weight of evidence-based assessment of the Danish EPA, a potential effect for repeated-dose toxicity is carcinogenicity, as observed in a 2-yr rat study. It is noted that the uterine tumours in this study were observed at higher levels than the effects identified for the lung and liver in several other toxicity studies. The lung was identified as a target organ for inhalation exposures of D5 whereas the liver was identified as a target organ for oral and inhalation exposures. The critical effect level for repeated dose toxicity via the inhalation route was based on a significant increased incidence of pulmonary vascular mineralization as observed in a rat reproduction study. Comparison of the critical effect level for repeated dose effects via inhalation and the conservative upper-bounding exposure estimate via inhalation for decamethylcyclotetrasiloxane, results in adequate margins of exposure. The critical effect level for repeated-dose toxicity was based on increased liver weight in a 90-day rat study and using as support, the determinations of oral critical effect levels for the similar compounds, octamethylcyclotetrasiloxane and dodecamethylcyclohexasiloxane. Based on an independent review of a refined exposure assessment for personal care products, an adequate margin of exposure was derived by comparison of the critical effect level for repeated dose effects via the oral route and a conservative upper-bounding estimate of daily intake of D5 via use of personal care products.

Based on the available information on its potential to cause ecological harm, it is concluded that D5 is entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity.

Based on the available information on its potential to cause harm to human health, it is concluded that D5 is not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

This substance will be included in the upcoming *Domestic Substances List* inventory update initiative. In addition and where relevant, research and monitoring will support verification of assumptions used during the screening assessment and, where appropriate, the performance of potential control measures identified during the risk management phase.

Based on the information available, it is concluded that D5 meets one or more of the criteria set out in section 64 of the *Canadian Environmental Protection Act, 1999*.

Table of content

Introduction	1
Substance Identity	3
Physical and Chemical Properties	5
Sources	8
Uses	9
Releases to the Environment	11
Environmental Fate	13
Persistence and Bioaccumulation Potential	14
Environmental Persistence	14
Potential for Bioaccumulation	22
Potential to Cause Ecological Harm	28
Ecological Effects Assessment	28
A - In the Aquatic Compartment	28
B – In Other Environmental Compartments	31
Ecological Exposure Assessment	33
Characterization of Ecological Risk	38
Uncertainties in Evaluation of Ecological Risk	41
Potential to Cause Harm to Human Health	43
Exposure Assessment	43
Health Effects Assessment	46
Characterization of Risk to Human Health	49
Uncertainties in Evaluation of Risk to Human Health	50
Conclusion	51
References	52
Appendix 1	63
Appendix 2	65
Appendix 3	67
Appendix 4	71
Appendix 5	92

Introduction

The *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada 1999) requires the Minister of the Environment and the Minister of Health to conduct screening assessments of substances that have met the categorization criteria set out in the Act to determine whether these substances present or may present a risk to the environment or human health. Based on the results of a screening assessment, the Ministers can propose to take no further action with respect to the substance, to add the substance to the Priority Substances List (PSL) for further assessment, or to recommend that the substance be added to the List of Toxic Substances in Schedule 1 of the Act and, where applicable, the implementation of virtual elimination.

Based on the information obtained through the categorization process, the Ministers identified a number of substances as high priorities for action. These include substances that

- met all of the ecological categorization criteria, including persistence (P), bioaccumulation potential (B) and inherent toxicity to aquatic organisms (iT), and were believed to be in commerce in Canada; and/or
- met the categorization criteria for greatest potential for exposure (GPE) or presented an intermediate potential for exposure (IPE), and had been identified as posing a high hazard to human health based on classifications by other national or international agencies for carcinogenicity, genotoxicity, developmental toxicity or reproductive toxicity.

The Ministers therefore published a notice of intent in the *Canada Gazette*, Part I, on December 9, 2006 (Canada 2006), that challenged industry and other interested stakeholders to submit, within specified timelines, specific information that may be used to inform risk assessment, and to develop and benchmark best practices for the risk management and product stewardship of those substances identified as high priorities.

The substance decamethylcyclopentasiloxane, also known as D5, was identified as a high priority for assessment of ecological risk as it was found to be persistent, bioaccumulative and inherently toxic to aquatic organisms and is believed to be in commerce in Canada. The Challenge for this substance was issued in the *Canada Gazette* on May 12, 2007 (Canada 2007). A substance profile was released at the same time. The substance profile presented the technical ecological information available prior to December 2005 that formed the basis for categorization of this substance. As a result of the Challenge, more than 100 submissions of information were received for this substance pertaining to its physical and chemical properties, bioaccumulation potential, persistence, ecotoxicology, quantity in commerce and so on.

Although the categorization exercise did not determine D5 to be a priority for assessment with respect to risks to human health, it was recommended that a human health assessment also be conducted due to its structure and use pattern similarity to D4, also known as octamethylcyclotetrasiloxane, a high priority for assessment for both human health and

ecological risks under CEPA 1999. Therefore, this assessment focuses on information relevant to the evaluation of ecological risks and risks to human health.

Under CEPA 1999, screening assessments focus on information critical to determining whether a substance meets the criteria for defining a chemical as toxic as set out in section 64 of the Act, where

“64. [...] a substance is toxic if it is entering or may enter the environment in a quantity or concentration or under conditions that

- (a) have or may have an immediate or long-term harmful effect on the environment or its biological diversity;
- (b) constitute or may constitute a danger to the environment on which life depends; or
- (c) constitute or may constitute a danger in Canada to human life or health.”

Screening assessments examine scientific information and develop conclusions by incorporating a weight-of-evidence approach and precaution.

This screening assessment includes consideration of information on chemical properties, hazards, uses and exposure, including the additional information submitted under the Challenge. Data relevant to the screening assessment of this substance were identified in original literature, review and assessment documents, stakeholder research reports and from recent literature searches, up to August 2008 for both human health and ecological sections of the document. Key studies were critically evaluated; modelling results may have been used to reach conclusions. When available and relevant, information presented in hazard assessments from other jurisdictions was considered. The screening assessment does not represent an exhaustive or critical review of all available data. Rather, it presents the most critical studies and lines of evidence pertinent to the conclusion.

Evaluation of risk to human health involves consideration of data relevant to estimation of exposure (non-occupational) of the general population, as well as information on health hazards (based principally on the weight-of-evidence assessments of other agencies that were used for prioritization of the substance). Decisions for human health are based on the nature of the critical effect and/or margins between conservative effect levels and estimates of exposure, taking into account confidence in the completeness of the identified databases on both exposure and effects, within a screening context. The screening assessment does not represent an exhaustive or critical review of all available data. Rather, it presents a summary of the critical information upon which the conclusion is based.

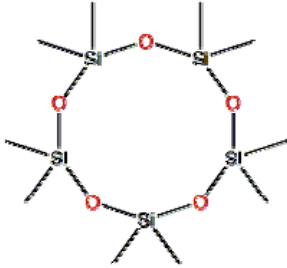
This screening assessment was prepared by staff in the Existing Substances Programs at Health Canada and Environment Canada and incorporates input from other programs within these departments. This assessment has undergone external written peer review/consultation. Comments on the technical portions relevant to human health were received from Toxicology Excellence for Risk Assessment (TERA). While external comments were taken into consideration, the final content and outcome of the screening risk assessment remain the responsibility of Health Canada and Environment Canada. Additionally, the draft of this screening assessment was subject to a 60-day public comment period. The critical information and considerations upon which the assessment is based are summarized below.

Substance Identity

For the purposes of this document, decamethylcyclopentasiloxane will be referred to as D5, an abbreviated name derived from the siloxane notation developed by General Electric (Hurd 1946).

D5 belongs to a group of cyclic volatile methyl siloxanes (cVMS) with relatively low molecular weight (< 600 g/mol) and high vapour pressure. These cVMS are volatile, low-viscosity silicone fluids consisting of $[-\text{Si}(\text{CH}_3)_2\text{O}-]_x$ structural units in a cyclic configuration. D5 consists of five of these $[-\text{Si}(\text{CH}_3)_2\text{O}-]$ structural units ($x = 5$) as shown in the chemical structure below (Table 1).

Table 1. Substance identity for D5

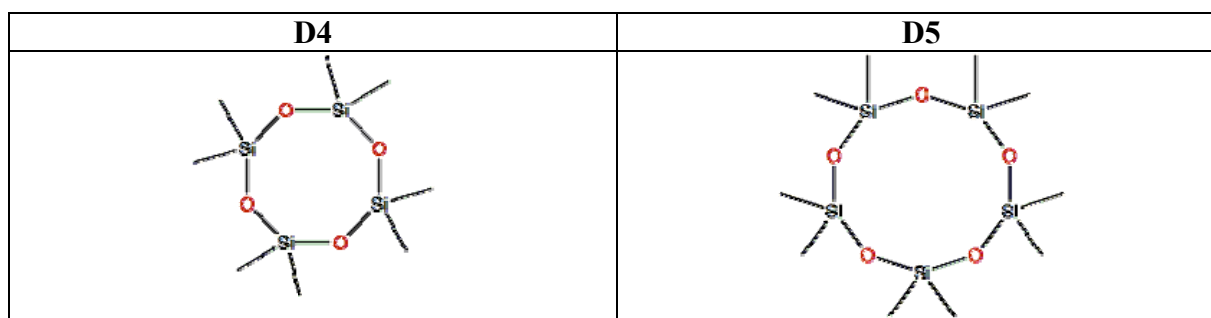
Chemical Abstracts Service Registry Number (CAS RN)	541-02-6
Name on Domestic Substances List (DSL)	Cyclopentasiloxane, decamethyl-
National Chemical Inventories (NCI) names¹	Cyclopentasiloxane, 2,2,4,4,6,6,8,8,10,10-decamethyl- (TSCA); Cyclopentasiloxane, decamethyl- (AICS, PICCS, ASIA-PAC, NZIoC); Decamethylcyclopentasiloxane (EINECS); Decamethyl cyclopentasiloxane (ECL)
Other names	Cyclic dimethylsiloxane pentamer; Cyclo-decamethylpentasiloxane; D5; Dimethylsiloxane pentamer; Pentacyclomethicone
Major chemical class or use	Organosilicon compounds
Major chemical sub-class	Cyclic volatile methyl-siloxanes (cVMS)
Chemical formula	$\text{C}_{10}\text{H}_{30}\text{O}_5\text{Si}_5$
Chemical structure	
Simplified Molecular Input Line Entry System (SMILES)	<chem>C[Si]1(C)O[Si](C)(C)O[Si](C)(C)O[Si](C)(C)O[Si](C)(C)O1</chem>
Molecular mass	370.78 g/mol

¹ National Chemical Inventories (NCI). 2006: AICS (Australian Inventory of Chemical Substances); ASIA-PAC (Asia-Pacific Substances Lists); ECL (Korean Existing Chemicals List); EINECS (European Inventory of Existing Commercial Chemical Substances); PICCS (Philippine Inventory of Chemicals and Chemical Substances); NZIoC (New Zealand Inventory of Chemicals); and TSCA (Toxic Substances Control Act Chemical Substance Inventory).

It should be noted that D5 is also contained under another Chemical Abstracts Service Registry Number. This registry number, CAS RN 69430-24-6, refers to a mixture of dimethyl-substituted cyclosiloxanes of the general structure $[-\text{Si}(\text{CH}_3)_2\text{O}-]_x$ in a cyclic configuration, where x is generally less than 8, and more commonly x is 3-7 (SEHSC 2007b). This CAS number is associated with the following names:

cyclopolydimethylsiloxane, cyclopolydimethylsiloxane (DX), cyclosiloxanes di-Me, dimethylcyclopolysiloxane, polydimethyl siloxy cyclics, polydimethylcyclosiloxane, cyclomethicone and mixed cyclosiloxane. In this report it will be referred to as cyclomethicone, a term commonly used for the mixture in the cosmetics industry.

For this assessment, data from analogue D4 has also been used based on structural similarity as shown in the table below:



Physical and Chemical Properties

Table 2 contains experimental and modelled physical and chemical properties of D5 that are relevant to its environmental fate.

Table 2. Physical and chemical properties of D5

Property	Type	Value ¹	Temperature (°C)	Reference
Melting point (°C)	Experimental	-38*		PhysProp 2006
	Modelled	-5.19		MPBPWIN 2000
Boiling point (°C)	Experimental	210*		PhysProp 2006
	Modelled	196.78		MPBPWIN 2000
Density (kg/m³)	Experimental	954		SEHSC 2005a
Vapour pressure (Pa)	Experimental	26.66 (0.20 mm Hg)	25	Flaningam 1986
		33.2* (0.249 mm Hg)	25	SEHSC 2005a
	Modelled	29.06 (0.22 mm Hg)	25	MPBPWIN 2000
Henry's Law constant (Pa·m³/mol)	Experimental	3 350 000* (33.1 atm·m ³ /mol)		Calculated from K _{aw} value of Xu and Kropscott 2007
		13 444 (0.133 atm·m ³ /mol)	23	David et al. 2000
		32 317 (0.319 atm·m ³ /mol)	26	Kochetkov et al. 2001
		29 831 (0.294 atm·m ³ /mol)	26	Kochetkov et al. 2001
	Modelled	12 159 (0.12 atm·m ³ /mol)	25	HENRY WIN 2000
Log K_{aw} (Air-water partition coefficient) (dimensionless)	Experimental	3.13*	24.6	Xu and Kropscott 2007
Log K_{ow} (Octanol-water partition coefficient) (dimensionless)	Experimental	5.2		Bruggeman et al. 1984
		8.03*	25.3	Kozerski 2007
		4.76	22.4	Sible 2006
	Modelled	5.71		KOWWIN 2000
Log K_{oc} (Organic carbon-water partition coefficient) (dimensionless)	Experimental	5.17		SEHSC 2005a
	Modelled	5.16		PCKOCWIN 2000
Water solubility (mg/L)	Experimental	0.017*	23	Varapath et al. 1996
	Modelled	0.05	25	WSKOWWIN 2000
Log K_{oa} (Octanol-air partition)	Experimental	5.06*	24	Xu 2006

coefficient) (dimensionless)				
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¹ If different, values and units in parentheses represent the original values as reported by the authors or as estimated by the models.

* Values used in modelling for this screening assessment.

Recently, empirical log K_{ow} values for D5 were received and were critically evaluated. A recent experimental log K_{ow} value of 8.03 for D5 (99.19% purity) at 25.3°C was determined by using the slow stir method following OECD Draft Guideline 123 (Kozerski 2007). The measurement of K_{ow} was carried out in triplicate with one blank control. Two-litre borosilicate glass vessels were used in performing the equilibrium and were charged with 1.6 L high-purity water followed by adding 0.10 L of 1-octanol carefully to minimize droplet formation. The test was initiated by adding ~5 mL of D5 spiking solution (119.68 mg D5/g) in 1-octanol to the test vessel. The temperature was maintained at between 25.2 and 25.6°C during the study. It was concluded that equilibrium was achieved 24 hours after test initiation. The weighted average K_{ow} was calculated to be 8.03. A headspace of ~0.3 L (15% total volume) was present in the test vessels, indicating that the D5 may have volatilized into the headspace from the water phase (high vapour pressure, low water solubility). However, a flask mass balance check suggested that the total vaporized D5 was less than 2%. Therefore, the study is considered acceptable and the log K_{ow} of 8.03 will be used for this screening assessment report.

The experimental log K_{ow} of 4.76 was determined by using the slow stir method following OECD Draft Guideline 123 (Sible 2006). Radio-labelled ¹⁴C-D5 (99.8% radiochemical purity) diluted with unlabelled D5 (99.77%) was used in the test. The measurement of K_{ow} was carried out in triplicate with one blank control. The temperature was maintained constant at 22.4 ± 0.5°C throughout the study. Equilibrium was achieved in 7 days and a log K_{ow} of 4.76 was obtained based on the average measured log K_{ow} from day 8 to day 11. The analysis of radio-labelled ¹⁴C-D5 in water and octanol was conducted by using liquid scintillation counting (LSC). LSC analysis does not distinguish between a parent compound and its potential hydrolysis products. Since D5 may undergo slow hydrolysis under the test condition (Durham 2006), the D5 concentration in water may have been overestimated, which may have resulted in underestimation of the log K_{ow} value.

A log K_{ow} value of 5.2 was determined by Bruggeman et al. (1984) using a high-performance liquid chromatography (HPLC)-retention time method. The measurement was performed on an octadecylsilyl-bonded silica column with 90:10 methanol:water as the mobile phase. Homologous series of n-alkylbenzenes with known log K_{ow} values were used as reference compounds to calibrate the method. The experimental details of this study are currently unavailable.

Recent experiments on the air-water partition coefficient for D4 and D5 were conducted by Xu (Xu and Kropscott 2007). The partitioning equilibrium among air, water and an organic phase (octanol) was simultaneously achieved during the experiment; the log K_{oa} was calculated to be 4.94 and the log K_{ow} was calculated to be 8.07 for D5. Both values are reasonably consistent with the measured experimental values reported by Xu (2006, Table 2) and Kozerski (Kozerski 2007, Table 2). The study is therefore considered acceptable and

the log K_{aw} of 3.13 for D5 from the study will be used for this screening assessment report. A simultaneous determination of the partitioning equilibrium among air, water, and organic phase (octanol) has been conducted by Xu (Xu and Kropscott 2007). A custom-made double syringe system was designed for measuring the partitioning equilibrium among the three phases. The system consists of two air-tight syringes with the left syringe containing ~5 mL octanol-saturated water, ^{14}C -labelled D5 in octanol on top of the water phase, and a gas phase of ~70–80 cm^3 . The right syringe contained a ~60–80 mL octanol-saturated water phase and a ~20–40 cm^3 air phase. The air phases between the two syringes were connected during the test. The equilibrium between the air and water phase was accelerated by the slow stirring of water and was reached after 20 hours. The average log K_{aw} of 3.13 for D5 at 24.6°C was thus determined by the total D5 radioactivity in air and water. This value is in good agreement with the equilibrium of $\log K_{ow} = \log K_{oa} + \log K_{aw}$. The experimental K_{aw} gives a Henry's Law constant of 3 350 000 $\text{Pa}\cdot\text{m}^3/\text{mol}$ at ~25°C.

For D5, other modelled physical and chemical properties are in good agreement with its measured experimental data. Except for the data discussed above, the most conservative experimental data, when applicable, are used in various model predictions in this screening assessment report.

Sources

There are no known natural sources of D5.

D5 is an industrial chemical which was not manufactured by any company in Canada in 2006 in a quantity above the reporting threshold of 100 kg, but which is imported into the country as an essentially pure substance, in mixtures with other cyclic siloxanes, as a residual in silicone polymers, and in finished consumer products. From responses to a notice published under section 71 of CEPA 1999, it was determined that between 1 000 000 and 10 000 000 kg of D5 were imported into Canada in 2006, as raw materials or in finished products (Environment Canada 2007). The quantities of D5 imported into Canada have increased significantly since the DSL nomination (Environment Canada 1988).

D5 is a constituent of CAS RN 69430-24-6, termed cyclomethicone in the cosmetics industry. Although cyclomethicone was not directly surveyed under CEPA section 71 by Environment Canada and Health Canada in 2007, it is evident that in some cases, responses to the notice published under section 71 of CEPA 1999 for the 2006 calendar year contained data on the quantity of D5 used or imported as CAS RN 69430-24-6 (Environment Canada 2007).

The quantity of CAS RN 69430-24-6 reported in commerce in Canada during the 1986 calendar year was 2 220 000 kg (Environment Canada 1988). In 2005, Canada was a net importer of 11 500 000 kg of all types of silicone polymers and siloxanes (Will et al. 2007).

D5 is an intermediate in the production of polydimethylsiloxanes (PDMS) silicone polymers, and all PDMS polymers contain residual amounts of volatile cyclosiloxanes, including D5. The lower molecular weight (and consequently lower viscosity) polymers may contain from < 0.1% to 0.5% volatile cyclosiloxanes, and higher molecular weight (and consequently higher viscosity) polymers may contain 1–3% volatile cyclosiloxanes. The proportion of the volatile cyclosiloxanes that consists of D5 is highly product-specific. Release of D5 from some applications of PDMS is expected to occur once the PDMS product is in use (SEHSC 2007b).

Detection of D5 at sewage treatment plants, landfills and near industrial plants, as well as in indoor and ambient air away from industrial activity, is evidence that both point sources and disperse sources contribute to the concentration of D5 in the environment (Norden 2005; Kaj et al. 2005; personal communication, Environment Canada, Canadian Centre for Inland Waters, 2007 [unreferenced]). The application of D5-containing pesticides on crops and the disposal of sewage sludge on agricultural lands, by incineration and by deposit in landfills will result in the release of D5 to environmental media. There is some evidence that D5 is a transient degradation product of PDMS in contact with soil, while the principal degradation products are silanols prior to complete mineralization (Herner et al. 2002). Thus, in addition to release of residual D5 from PDMS manufacture there may be *de novo* synthesis of D5 occurring in landfills and agricultural lands where sewage sludge containing PDMS is

spread, although the overall contribution of PDMS degradation is not considered significant under environmental conditions.

D5 has been identified as a high production volume (HPV) chemical by the Organisation for Economic Co-operation and Development (OECD 2007), the US Environmental Protection Agency (US EPA 2007), and the European Chemicals Bureau (ECB 2007).

In the United States, there is a trend toward the increased use of volatile methyl siloxanes, including D5, because of their exemption from volatile organic compound (VOC) legislation in 1994 (US EPA 1994). Volatile methyl siloxanes were used as an alternative to chlorofluorocarbons (CFCs) as a means of reducing the regulated VOC content in products (specifically, precision and electronic cleaning applications). According to information from the US EPA, the import/production of D5 in the United States was in the range of 4500–22 500 tonnes in 1986, 1990 and 1994. The import/production increased to 22 500–45 000 tonnes in 1998, and to 45 000–225 000 tonnes in 2002.

In Europe, four companies have been identified as producers/importers of D5: Bayer AG of Germany, Dow Corning Limited of the United Kingdom, Rhodia Chimie of France and Amway Europe of Belgium (ECB 2007). The quantity of D5 used in the European Union as a site-limited intermediate during 2003–2004 is confidential information.

Uses

The predominant use, worldwide and in Canada, of D5 is in blending and formulating consumer products (Environment Canada 2007). D5 is also used as an intermediate in the production of PDMS polymers and a small number of commercial dry cleaners in Canada use D5 as a dry-cleaning fluid (Green Earth 2008). Also, all silicone polymers contain trace residual amounts of volatile cyclosiloxanes, including D5. As indicated above, D5 is also a constituent of CAS RN 69430-24-6, termed cyclomethicone in the cosmetics industry.

Cyclomethicone is a mixture of low molecular weight volatile cyclic siloxanes, the principal ingredients of which are octamethylcyclotetrasiloxane (D4), decamethylcyclopentasiloxane (D5) and dodecamethylcyclohexasiloxane (D6), in varying proportions. In Canada, the most important uses of the mixtures of low molecular weight volatile cyclic siloxanes, which may contain a high percentage of D4 or of D5, are in the preparation of personal care products, including hair and skin care products and antiperspirants (Environment Canada 2007).

Silicone polymers that contain trace amounts of D5 can be grouped as fluids, gums and resins. Uses of such polymers are described below.

Important uses of silicone fluids include as a formulation component of personal care products for hair and skin care, antiperspirants and deodorants; pharmaceuticals; processing aids such as defoamers; surfactants and mould release agents; lubricants; polishes and coatings on a range of substrates including textiles, carpeting and paper; sealants;

architectural coatings; mechanical, heat transfer and dielectric fluids; and reprography (Will et al. 2007).

While it is anticipated that higher molecular weight polymers are used in most of these applications, D5 was reported for use as a defoamer (Environment Canada 2007). Defoamers are employed often at parts per million levels in a range of processing industries including pulp and paper, food, petrochemical, petroleum, and chemical manufacture as well as water treatment. Silicones are also used as defoamers in household products such as cleaners and detergents (Will et al. 2007).

The use of silicone formulants containing D5 in certain pesticide products is regulated in Canada under the *Pest Control Products Act* (PMRA 2007).

Silicone fluid/gel mixtures are used for several types of cosmetics and medical devices. Silicone fluids have been approved as active and non-active ingredients in pharmaceuticals in Canada (DPD 2007), the most common use being in anti-flatulence drugs. Other biomedical uses of silicone fluids in Canada are in blood-handling equipment, as a blood defoaming agent, as protective barriers and lubricants, and for surface treatment of wound dressings.

Silicone gums are used in the production of elastomers that are used as sealants and adhesives, and in moulded silicone rubber, coatings and encapsulation. Silicone elastomers are used in the manufacture of consumer products such as pacifiers. Silicone elastomers are also used in a large number of biomedical applications including short- and long-term implants and prostheses, catheters, contact lenses and dentures (Will et al. 2007).

Silicone resins are primarily used in specialty coatings applications, and in the production of silicone-modified polymers (Will et al. 2007). Consumers may be exposed to D5 through the use of these products and by occupying enclosed spaces where coatings, caulking, sealants and silicone rubber are used as building materials or are present in consumer products.

Releases to the Environment

D5 is not reported as part of Environment Canada's National Pollutant Release Inventory. This substance belongs to a chemical group used in various industry and consumer applications that are associated with the potential for widespread releases.

D5 may be emitted to the environment from industrial processes in which it is reacted to form silicone polymers and co-polymers and from blending, formulation and packaging operations. All of these operations take place in Canada (Environment Canada 2007). Industrial releases of D5 may also occur when silicone polymers are used in process industries as foam control agents, as mould release agents, as lubricants, and in other applications (Environment Canada 2007). The use of D5 as a commercial dry-cleaning fluid (even though its current scale of use indicates that its contribution is not significant) and as an industrial degreaser may lead to atmospheric releases. The releases from industrial processes are expected to be to the atmosphere and wastewater. D5 will be released during the use of personal care products such as hair and skin care products, antiperspirants and others, and these releases will be to air and wastewater. It is estimated that more than 90% of D5 used in personal care products enters the atmosphere (Allen et al. 1997). The releases of D5 to sewage treatment plants as wastewater can lead to its association with sludges that may then be sent to landfills, incinerated or be applied to agricultural soils as soil enrichment. Disposal of consumer and industrial products containing D5 can also lead to the transfer of D5 to landfills.

Detection of D5 at sewage treatment plants, landfills and near industrial plants as well as in indoor and ambient air away from industrial activity is evidence that both point sources and disperse sources contribute to the concentration of D5 in the environment (Norden 2005, Kaj et al. 2005). The application of D5-containing pesticides of crops and the disposal of sewage sludge on agricultural lands, by incineration and by deposit in landfills will result in the release of D5 to environmental media. There is some evidence that D5 is a transient degradation product of PDMS in contact with soil, while the principal degradation products are silanols (Xu et al. 1998). Thus, in addition to release of residual D5 from PDMS manufacture, there may be *de novo* synthesis of D5 occurring in landfills and agricultural lands where sewage sludge containing PDMS is spread, although the overall contribution of PDMS degradation is not considered significant under environmental conditions.

Mass Flow Tool

To estimate the potential release of D5 to the environment at different stages of its life cycle, a Mass Flow Tool was used. Empirical data concerning releases of specific substances to the environment are seldom available. Therefore, for each identified type of use of the substance, the proportion and quantity of release to the different environmental media are estimated, as are the proportions of the substance chemically transformed or sent for waste disposal. Assumptions and input parameters used in making these estimates are based on information obtained from a variety of sources including responses to regulatory surveys, Statistics Canada, manufacturers' websites and technical databases. Of particular relevance

are emission factors, which are generally expressed as the fraction of a substance released to the environment, particularly during its manufacture, transformation, and use associated with industrial processes. Sources of such information include emission scenario documents, often developed under the auspices of the Organisation for Economic Co-operation and Development (OECD), and default assumptions used by different international chemical regulatory agencies. It is noted that the level of uncertainty in the mass of substance and quantity released to the environment generally increases further down the life cycle.

Table 3. Estimated releases and losses of D5 to environmental media, transformation and distribution to management processes, based on the Mass Flow Tool¹

Fate	Proportion of the mass (%) ¹	Major life cycle stage involved ²
Releases to environment:		
To soil	0	-
To air	77.5	Industrial use and consumer use
To sewer*	12.2	Formulation, industrial use and consumer use
Chemically transformed	0.8	Industrial use
Transferred to waste disposal sites (e.g., landfill, incineration)	9.5	Waste disposal

* Wastewater before any form of treatment

¹ For D5, information from the following OECD emission scenario documents was used to estimate releases to the environment and distribution of the substance as summarized in this table: OECD 2004; OECD 2006. Values presented for releases to environmental media do not account for possible mitigation measures that may be in place in some locations (e.g., partial removal by sewage treatment plants). Specific assumptions used in the derivation of these estimates are summarized in Environment Canada 2008a.

² Applicable stage(s): production, formulation, industrial use, consumer use, service life of article/product, waste disposal.

Based on the information available, the substance is mainly released to air and wastewater (sewer). Air receives the highest proportion of releases, approximately 77.5%. This is a result of the use of consumer products such as skin creams, sun creams or polishes (SPIN 2007). Releases to wastewater are estimated to be approximately 12.2% from industry use during product formulation and from the use of personal care products. Another 9.5% of the substance is transferred for waste disposal.

Environmental Fate

Based on its physical and chemical properties (Table 2) and the results of Level III fugacity modelling (Table 4; model input parameters are listed in Appendix 5 of this screening assessment), D5 may partition in significant quantities to any environmental medium, depending on the compartment of release.

Table 4. Results of the Level III fugacity modelling for D5 (EQC 2003)

Substance released to	Fraction of substance partitioning into each compartment			
	Air	Water	Soil	Sediment
Air (100%)	100	0.0	0.0	0.0
Water (100%)	4.4	30.9	0.0	64.7
Soil (100%)	71.2	0.0	28.7	0.0

Based on the information available (Table 3), the environmental release of D5 is estimated to be mainly to air. A vapour pressure of 26.66–33.2 Pa and a Henry's Law constant of 3 350 000 Pa·m³/mol, as well as a long half-life in air, indicate that when D5 is released to air it will remain there (100%) (Table 4).

When D5 is released to water (Table 4), it is expected to adsorb to suspended solids, such as sewage sludge and sediments, based on its high empirical log K_{oc} values (>5). Results of the Level III fugacity simulation for release to water show that approximately 64.7% will reside in the solid phase (suspended sediment and bed sediments) and 30.9% will reside in the aqueous phase (water column). The log K_{oc} for the compound is in the high sorption range, and the moderate hydrolysis rate of D5 in water at ambient temperature reduces the fraction that is expected to be adsorbed to sediments. Volatilization from water surfaces is also expected based upon the air-water partition coefficient (K_{aw}); the mass fraction expected to partition to air from volatilization at 25°C is 4.4% (Table 4).

When D5 is released to moist soils through, for example, the application of sewage sludge on agricultural lands, ~71% of the mass fraction will partition to air. This estimate is consistent with the observation by Xu (1999) that volatilization is the major loss process of cyclic siloxanes from moist soils. Approximately 28.7% will remain in soil associated with solids, for the same reasons as described for sediment. In dry soil, D5 is expected to be quickly hydrolyzed by clay minerals to form dimethylsilanediol as the final breakdown substance (Xu 1999, Xu and Chandra 1999).

Persistence and Bioaccumulation Potential

Environmental Persistence

Atmospheric Degradation

The Level III fugacity model results indicate that D5, when released to air, will remain in air, where it is expected to be slowly oxidized by the gas-phase reaction with photochemically produced hydroxyl (OH) radicals. The empirically derived half-life for D5 in the gas-phase hydroxyl radical reaction is 6.9 days (Atkinson 1989; see Table 5a). This is based on an experimental reaction rate of $1.55 \times 10^{-12} \text{ cm}^3/\text{mol}\cdot\text{sec}$ (Atkinson 1989), which may be converted to an estimated half-life of 6.9 days, assuming first-order kinetics, a 12-hour day, and $1.5 \times 10^6 \text{ OH}/\text{cm}^3$. D5 is not expected to react, or react appreciably, with other photo-oxidative species in the atmosphere, such as O_3 , nor is it likely to degrade via direct photolysis (Atkinson 1991). Therefore, it is expected that reactions with hydroxyl radicals will be the most important fate process in the atmosphere for this substance.

Recent field measurements of the hydroxyl radical concentrations in an urban environment (Ren et al. 2003, Kramp and Volz-Thomas 1997, Rivett et al. 2003) suggested that there is a higher OH concentration in the urban atmosphere than that observed in the rural and marine atmosphere due to higher OH radical precursors in polluted urban areas (SEHSC 2008b). Ren et al. (2003) have measured the concentration of hydroxyl radical in the summer atmosphere in New York City, NY, USA. The measurement was conducted over a 34-day period. The average maximum hydroxyl radical concentration was reported to be $7 \times 10^6 \text{ OH}/\text{cm}^3$ and was comparable to those measured ($1\text{--}10 \times 10^6 \text{ OH}/\text{cm}^3$) in similar urban environments of the United States (Los Angeles, CA and Nashville, TN; SEHSC 2008b) and in European countries (Kramp and Volz-Thomas 1997, Rivett et al. 2003). However, most of these measurements were carried out during the summer, when the sunlight was strong and the atmospheric photochemistry was active. The OH radical concentration measured by Ren et al. (2006) in the winter in New York City was ~ 5 times lower than in the summer at the same site. The measurement was conducted over a 28-day period and the average maximum concentration was $1.4 \times 10^6 \text{ OH}/\text{cm}^3$. Therefore, half-lives of 3.0 to 14.8 days can be calculated assuming first-order kinetics, a 12-hour day, and a daily average hydroxyl concentration of $3.5 \times 10^6 \text{ OH}/\text{cm}^3$ and $0.7 \times 10^6 \text{ OH}/\text{cm}^3$ (daily average concentration = maximum concentration/2), in summer and winter respectively. It is therefore concluded that D5 could be degraded more rapidly in urban centers in summer seasons when the atmospheric hydroxyl radicals are most abundant. However, when a yearly average removal half-life is considered, it is consistent with the half-life of 6.9 days estimated with a hydroxyl radical concentration of $1.5 \times 10^6 \text{ OH}/\text{cm}^3$. The degradation half-life of 6.9 days is considered critical and will be used for D5 in environmental fate modelling.

Navea et al. (2007) have investigated the effects of ozone, aerosols and solar radiation on the fate of D4 and D5 in a simulated environment chamber. They concluded that mineral aerosols such as kaolinite and hematite can significantly accelerate the removal of D4 and

D5 from the gas phase of the atmosphere, especially under daytime conditions. The finding also indicated that ozone can further accelerate these removal processes for D4 and D5. Although obtained data suggested that mineral aerosols, combined with ozone, may have significant effects on the environmental fate of cVMS in the air, it is difficult to quantitatively extrapolate the results of the simulations to realistic environmental conditions. First, it should be noted that the study was conducted under unrealistically high concentrations of cVMS, mineral aerosols and ozone. Second, mineral and carbon black samples used in the study were high-purity (>99%) analytical samples that provided maximum surface area and thus maximally available sorption sites, i.e., ideal conditions for D4/D5 absorption. The degree to which these pure minerals are representative of the particulate matter in air is questionable. Third, it is reasonable to believe that minerals such as kaolinite and hematite can be found in atmospheric particulate matter (PM); however, they are unlikely to be the most common and abundant components in atmospheric dust. In addition, it should be mentioned that the study was conducted in a simulated environment chamber and involved reacting the mineral aerosols with only one cVMS (D4 or D5) at a time. Under actual environmental conditions, thousands of chemicals compete for aerosols' adsorption sites. Therefore, in such conditions, the "effectiveness" of D4/D5 removal from the ambient air could be significantly lower than that observed in the chamber's mono-component atmosphere.

Thus, it may be concluded that the degree to which aerosols and ozone accelerate the degradation of cVMS in air under realistic environmental conditions is uncertain.

The AOPWIN (2000) model (Table 5b) also provides evidence indicating the potential for persistence of this substance, with a predicted atmospheric oxidation half-life of 3–15 days. Thus, the empirical and model data demonstrate that this substance is persistent in air (half-life > 2 days) in accordance with the *Persistence and Bioaccumulation Regulations* (Canada 2000).

Table 5a. Empirical data for persistence of D5

Medium	Fate process	Degradation value	Degradation endpoint /Units	Reference
Air	OH reaction	6.9	Half-life (days)	Atkinson 1989
Air	OH reaction	3.0–14.8	Half-life (days)	Ren et al. 2003, 2006
Water	Biodegradation	0.14%	28 d degradation	Springborn Smithers Laboratories 2004
Water	Hydrolysis	0.9–733	Half-life (days) pH 6–9 5–25°C	Durham 2006 Kozerski 2008 Bidleman 2008
Water/sediments	Abiotic degradation	49–588	Read-across half-life (days) from D4 neutral pH; 5–25°C	Xu and Miller 2008
Soil (Wahiawa soils from Hawaii)	Clay-catalyzed hydrolysis	~2 hours (32% relative humidity)	Half-life (hours)	Xu 1999 Xu and Chandra 1999
Soil (Londo soils from Michigan)	Clay-catalyzed hydrolysis	3.54 days (32% relative humidity); 5.25 days (93% relative humidity)	Read-across half-life (days) from D4	Xu and Chandra 1999

Table 5b. Modelled data for persistence of D5

Medium	Fate process	Degradation value	Degradation endpoint / Units	Model
Air	Atmospheric oxidation	7.15	Half-life (days)	AOPWIN 2000
Air	Atmospheric oxidation	3.1–15.3 ²	Half-life (days)	AOPWIN 2000
Water	Biodegradation	37.5	Half-life (days)	BIOWIN 2000 Ultimate survey
Water	Biodegradation	0 (does not biodegrade fast)	Probability	BIOWIN 2000, MITI Linear probability
Water	Biodegradation	0.0003 (does not biodegrade fast)	Probability	BIOWIN 2000, MITI Non-linear probability
Water	Biodegradation	2.3	Percent biochemical oxygen demand (BOD) (MITI 301C) ¹	CATABOL c2004-2008
Soil	Biodegradation (anaerobic)	0.04 (does not biodegrade fast)	Probability	BIOWIN 2000

¹ Results from CATABOL biodegradation simulation show that D5 is in the global parameter domain and metabolic domain, but out of the structural domain. The most important of these domains is the metabolic domain, and CATABOL suggests that the substance will not be degraded, as the probability of stable methyl group and aromatic ring oxidation products is low.

² Atmospheric oxidation half-lives re-calculated with measured OH radical concentrations from New York City in summer and winter, respectively.

Degradation in Water and Sediment

The empirical hydrolysis data for D5 (Durham 2006) were critically reviewed by internal experts (Bidleman 2008); the results of these reviews are summarized below. The hydrolysis kinetics of D5 were determined by measuring the disappearance of radio-labelled ^{14}C -decamethylcyclotetrasiloxane (D5, substance purity 98.9%) and the formation of hydrolysis products as a function of time based on OECD Guideline 111. Reactions were investigated in flame-sealed borosilicate glass tubes at pH values of 4, 5.5, 7, 8 and 9 and temperatures of 10°C, 25°C and 35°C. The initial test concentration was targeted at 6 µg/L upon spiking, corresponding to 1/2–1/3 of the water solubility of D5. Tetrahydrofuran (THF) was used as a solubilizer at a concentration of less than 1% v/v. A similar hydrolysis kinetics study was also conducted for radio-labelled ^{14}C -octamethylcyclotetrasiloxane (D4) at the same laboratory (Durham and Kozerski 2005). The hydrolysis rates of D4 and D5 were reported to be pH-dependent and followed pseudo first-order kinetics. Both D4 and D5 were found to undergo rapid hydrolysis under acidic (pH 4) and basic (pH 9) conditions, with average half-lives ($t_{1/2}$) ranging from minutes to less than 6.5 hours for D4 and from hours to less than 6 days for D5 at 10–35°C. Two additional hydrolysis tests were performed for D5 at near-neutral (pH 5.5 and 8) conditions at 25°C and their $t_{1/2}$ were approximately 15 and 9 days, respectively. The half-lives at neutral pH 7 conditions increased significantly for D4 and D5. The $t_{1/2}$ for D4 at 10–25°C was calculated to be 3.3–23 days. The $t_{1/2}$ for D5 at 10–25°C was calculated to be approximately 74–416 days. The hydrolysis products were intermediates dimethylsiloxane- α , and omega-diol oligomers $\text{HO}(\text{Me}_2\text{SiO})_n\text{H}$ ($n=2-4$ or 5), while dimethylsilanediol (DMSD) was the final hydrolysis product. Although loss of parent compounds and poor reproducibility were reported at neutral pH, loss rates at neutral pH may be estimated using the second-order rate constants for the acid- and base-catalyzed reactions. The hydrolysis studies for D4 and D5 are thus considered reliable for this screening assessment. An error in the calculation of the hydroxide catalytic rate constant, k_{OH} , at temperatures other than 25°C has, however, been identified by Bidleman (2008). Table 6 lists revised second-order rate constants for hydronium and hydroxide-catalyzed hydrolysis of D5 (Kozerski 2008).

Table 6. Revised second-order hydrolysis rate constants (i.e., catalytic constants) for D5 (Kozerski 2008)

Rate constant ($\text{M}^{-1} \text{h}^{-1}$)	Temperature ($^{\circ}\text{C}$)			
	5	10	25	35
k_{H}	141	210	742	1600
k_{OH}	1360	1730	3170	5020

The pseudo first-order rate constants, k_{obs} , for the hydrolysis of D5 can be calculated using the following kinetic equation (assuming negligible contribution of uncatalyzed hydrolysis, as confirmed by the experiments at a pH of 7):

$$k_{\text{obs}} = k_{\text{H}^+}[\text{H}^+] + k_{\text{OH}^-}[\text{OH}^-]$$

The calculated half-lives for D5 (Table 7) under realistic Canadian environmental conditions (pH 6–9, temperature 5–25°C) (GEMStat 2008, NOAA 2008) are in the range of 0.9–733 days.

Table 7. Calculated D5 half-lives under realistic Canadian environmental conditions

Temperature (°C)	Water dissociation constant pK_w	pH	Rate constant k (h^{-1})	Half-life (days)
25	14	6	7.74E-04	37
		7	3.91E-04	74
		8	3.18E-03	9.1
		9	3.17E-02	0.9
10	14.52	6	2.15E-04	134
		7	7.32E-05	395
		8	5.24E-04	55
		9	5.22E-03	5.5
5	14.73	6	1.44E-04	201
		7	3.94E-05	733
		8	2.54E-04	114
		9	2.53E-03	11

New information received on microbial degradation indicates that D5 is not likely to be biodegraded in water. The 28-day ready-biodegradability test was performed in sealed vessels in accordance with OECD Draft Guideline 310 and data showed limited biodegradation (0.14%) of D5 over 28 days in a ready-biodegradation test (Springborn Smithers Laboratories 2004). These data are further supported by two of the models in Table 5b. These models indicate that the probability of biodegradation of D5 in water is effectively zero. Also, BIOWIN reported an overall weighted conclusion of “not readily biodegradable” based on the combined results of the BIOWIN3 and BIOWIN5 models.

Experimental and modelled biodegradation data indicate that D5 has little potential to biodegrade in aqueous environments. Therefore, hydrolysis is the major degradation process for D5 in water. The evidence suggests that D5 may be degraded relatively rapidly by hydrolysis when the water temperature is elevated. However, D5 is expected to persist for relatively long periods at low temperatures and neutral or slightly acidic water conditions in Canada (pH 6–7, temperature 5–10°C). It is therefore concluded that D5 meets the criterion of persistence in water ($t_{1/2} > 182$ days) under the *Persistence and Bioaccumulation Regulations* (Canada 2000).

No sediment degradation information was available for D5. However, a preliminary degradation study for D4 in a water/sediment system has been received recently (Xu and Miller 2008). A modified OECD 308 guideline was followed. The study was conducted at ambient temperature (22–25°C) with natural sediment (sandy silt, high OC content, ~70% water content and 11% organic matter, pH ~7) and water collected from deep under an uncontaminated lake. Radio-labelled D4 in diethylene glycol methyl ether was added via syringe at 10–15 spots on the surface sediment in each flask after the overlying water was carefully removed. Overlying water was again added onto the spiked sediment with minimum disturbance of sediment. Spiking of sediment instead of water ensured the substance's distribution in sediment. This properly addressed the substance's specific physical and chemical properties (high volatility and potential hydrolysis) and improved the reproducibility of the study. The concentrations of D4 measured from day 6 to day 22 (test termination) indicated that a steady state had been reached between water and sediment, with more than 95% D4 and radioactivity being detected in sediment. As demonstrated in the hydrolysis study of D4, the degradation products in sediment/water were intermediate diol oligomers $\text{HO}(\text{Me}_2\text{SiO})_n\text{H}$ ($n=2-4$), while dimethylsilanediol (DMSD) was the final degradation product. The calculated half-life for D4 degradation in sediment was 49 days at 22–25°C. The same degradation products observed in the study and in the hydrolysis study of D4 suggested that hydrolysis was the major degradation process in the sediment/water system. The major uncertainty in the study is the lack of test replicates. Since no data were available for degradation half-lives at lower temperatures, a read-across approach using the D4 hydrolysis data was applied. The hydrolysis half-lives of D4 in water were ~6–12 times longer when water temperatures were decreased to 5–10°C from 25°C. Assuming a similar trend of decreases in sediment, the estimated half-lives for D4 in sediment are 294 and 588 days, at 10°C and 5°C, respectively.

The sediment degradation half-lives of D4 are considered as read-across for D5 based on the similarity of the two substances. It is therefore concluded that the lack of fast degradation of D5 under some environmental conditions, especially in colder Canadian environments, will result in a half-life in sediment of $t_{1/2} > 365$ days. D5 is thus considered persistent in water and sediment in accordance with criteria defined under the *Persistence and Bioaccumulation Regulations* (Canada 2000). The extrapolation from the D4 sediment degradation at lower temperatures based on hydrolysis, however, is not without uncertainty. The assumption that the loss of D4 in sediments is solely a function of hydrolysis in the pore water does not take into account the fraction that may be sorbed to the solid phase—the degradation processes and rate of which are unknown.

Degradation in Soil

Although no empirical data on biodegradation in soils are available, biodegradation of D5 in water is negligible as noted above, based on the ready-biodegradation test (Springborn Smithers Laboratories 2004) and predictions of five of the six biodegradation models (Table 5b).

Xu (Xu 1999, Xu and Chandra 1999) has extensively investigated potential degradation pathways of cyclosiloxanes, including D4, D5 and D6, in Wahiawa soil from Hawaii at room temperature and 32% relative humidity. He concluded that the ring-opening polymerization reaction to form polydimethylsiloxane (PDMS) and the demethylation for cyclosiloxanes were insignificant in soils at concentrations of < 200 mg/kg dry weight. Clay-catalyzed hydrolysis of D5 was observed in highly weathered Wahiawa soils under dry soil conditions. It was suggested that the dryness of soil severely limits biological activity but promotes abiotic reactions such as surface-acid-catalyzed hydrolysis of PDMS, a polymer with the same dimethylsiloxane backbone as cyclosiloxanes (Xu 1999). The degradation rates of cyclosiloxanes were determined by soil moisture, clay type and clay content, as well as the size of the siloxane molecules that determine the rate of diffusion to the surface catalytic sites. The hydrolysis degradation half-life of D5 on Wahiawa soil (55% clay content, 2.1% water content) was approximately 2 hours at 22°C under dry soil conditions. The degradation rate of D5 was comparable to that of D4 under the same soil conditions (~1 hour half-life) and supported the statement that degradation rates among the three cyclosiloxanes were expected to decrease as the molecular weight increases: D4 > D5 >> D6. The hydrolysis rate of D5 in temperate Londo soils from Michigan (22% clay content) was not reported. The degradation half-lives of D4 in Londo soil were 3.54–5.25 days at relative humidity of less than 93%. Comparable degradation half-lives would be expected for D5 in the temperate Londo soil. The weight of evidence suggests that D5 will undergo clay-catalyzed hydrolysis and is not likely to be persistent under dry soil conditions. The lack of hydrolysis degradation of D4 in water-saturated Londo soil also suggests that degradation of D5 is negligible under the same conditions. Volatilization becomes the major loss mechanism for D5 under such soil conditions in an open system.

While investigating the influence of clay types on the degradation potential of polydimethylsiloxanes (PDMS), Xu et al. (1998) demonstrated that PDMS were degraded by clay minerals even though their catalytic activities varied. The widespread presence of these clay minerals suggests that D5 will undergo clay-catalyzed degradation in soil as long as critical soil conditions such as low moisture content are present, despite the tremendous diversity of Canadian soils.

Based on the available empirical studies that show the potential for rapid clay-catalyzed hydrolysis in surface soils, D5 is not considered persistent in soil since its degradation half-life is less than the criterion of $t_{1/2} > 182$ days stated in the *Persistence and Bioaccumulation Regulations* (Canada 2000).

The available empirical and modelled data indicate that D5 meets the persistence criteria for air (half-life ≥ 2 days), water (half life ≥ 182 days) and sediments (half-life ≥ 365 days) as

set out in the *Persistence and Bioaccumulation Regulations* (Canada 2000), but that it does not meet the half-life criterion for soil (i.e., its half-life is < 182 days).

Long-range Transport Potential

The Transport and Persistence Level III model (TaPL3 2000), a regional model, was used to estimate the characteristic travel distance (CTD) of D5. CTD is defined as the maximum distance travelled in air by 63% of the substance. Beyer et al. (2000) have proposed that CTDs of >2000 km represent high long-range atmospheric transport potential (LRATP), those of 700–2000 km represent moderate LRATP, and those of <700 km represent low LRATP. Based on the CTD estimate of 3447 km, the long-range atmospheric transport potential of D5 is judged to be high. This means that D5 is subject to atmospheric transport to remote regions such as the Arctic.

Table 8. Model predicted characteristic travel distance for D5

Characteristic travel distance	Model (reference)
3447 km	TaPL3 v. 2.10 (TaPL3 2000)
3438 km	OECD LRTP POPs Tool v.2.0 (Scheringer et al. 2006)

The OECD POPs Screening Model can be used to help identify chemicals with high persistence and long-range transport potential (Scheringer et al. 2006). The OECD model is a global model which compartmentalizes the earth into air, water and soil. This model is “transport-oriented” rather than “target-oriented,” as it simply identifies the CTD without indicating specifically where a substance may be transported (Fenner et al. 2005). Klasmeier et al. (2006) have suggested that a threshold of 5098 km, based on the model’s CTD estimate for 2,2',3',4,4',5,5'-Heptachlorobiphenyl (PCB-180), can be used to identify substances with high long-range transport potential. PCB-180 has been found in remote regions. The CTD calculated for D5 using the OECD model is 3438 km, indicating that D5 still has a significant potential for transport in air, but is below the boundary suggested for global pollutants by Klasmeier et al. (2006).

The OECD POPs Screening Model also calculates the transfer efficiency (TE), which is the percentage of emission flux to air that is deposited to the surface (water and soil) in a remote region ($TE = D/E \times 100$, where E is the emission flux to air and D is the deposition flux to surface media in a target region). The TE for D5 was calculated to be 1.9E-06%, which is well below the boundary of 4.65E-04% (2,4,4'-trichlorobiphenyl, or PCB-28) established for the model’s reference substances that are empirically known to be deposited from air to soil or water. The low TE means that D5 has the potential for long-range travel in the atmosphere without being deposited to Earth’s surface in any particular remote region. In addition, the log K_{oa} and log K_{aw} of D5 suggest that it will also have a low Arctic contamination potential (ACP) when examined using chemical partitioning space plots as described by Wania (2003, 2006).

A preliminary monitoring study of a remote ecosystem was conducted in Lake Opeongo, the largest lake in Algonquin Provincial Park, Ontario, Canada. The lake is relatively remote from potential sources of cVMS from sewage and runoff. Therefore, it was assumed that the

only significant source of cVMS to the lake would be from atmosphere deposition (Powell 2008). Preliminary analysis of sediments and zooplankton samples for cVMS found no D5, suggesting that atmospheric deposition is not a significant source of D5 to Lake Opeongo. Limits of detection were 15.9 ng (background corrected mass) for both sediments and zooplankton.

It is therefore concluded that D5 has the potential to be transported over long distances in the atmosphere. However, the modelled TE for D5 is low, which suggests that it lacks the potential to be deposited in water or soil in remote regions. The monitoring results of Lake Opeongo also supported the low atmospheric deposition potential for D5. It is expected that airborne D5 will be eventually degraded by hydroxyl radicals in air.

Potential for Bioaccumulation

The empirical and modelled log K_{ow} values for D5 (Table 2) suggest that this substance has the potential to bioaccumulate in biota.

In the Aquatic Compartment

New empirical data indicate that D5 has the ability to bioconcentrate in aquatic organisms. A bioconcentration study for D5 was conducted on fathead minnows (*Pimephales promelas*) in a flow-through system (Drottar 2005). The uptake of radio-labelled [^{14}C] D5 in fish tissue was investigated at concentrations of 1.1 $\mu\text{g/L}$ and 15 $\mu\text{g/L}$ (measured) over a 35-day period and depuration was monitored over a 70-day period. The mean steady-state bioconcentration factor (BCF_{ss}) was calculated to be 7060 L/kg based on concentrations measured from day 14 to day 35. The kinetic bioconcentration factor (BCF_k) was calculated to be 13 300 L/kg based on the uptake and depuration rates (k_1/k_2) for the 1.1 $\mu\text{g/L}$ treatment groups (Table 7a). Fish tissue analysis also indicated that the depuration half-life for radio-labelled D5 was 24 days. There is uncertainty as to whether steady-state was reached on day 14 of the uptake phase as claimed. While no statistically significant differences were noted in the concentration of D5 in fish for days 14, 21, 28 and 35, the data was variable and the average concentrations continued to increase with time. This, plus the inconsistency noted between the kinetic and steady-state BCF (~2 times) and the long depuration half-life suggests that steady-state may not have been reached on day 14. Therefore, the kinetic BCF_k of 13 300 L/kg is considered more reliable in this screening assessment report.

A separate study investigating the metabolic rate of radio-labelled [^{14}C] D5 in rainbow trout (*Oncorhynchus mykiss*) was conducted through oral administration (Springer 2007). A nominal dose of 15 mg D5/kg (9.4–13 mg D5/kg measured) body weight of [^{14}C] D5 (radio purity 97.6%) in corn oil was fed to three mature trout over a 96-hour period. The majority of metabolites in fish were found in bile, with < 1% of radioactivity contributed by the parent compound, while 56%, 60% and 76% of radioactivity in egg sacs, liver and digestive tract was attributed to parent D5, respectively. Insignificant metabolism was found in fish fat and blood. The administered D5 was eliminated through urine and feces. The calculated metabolism in trout over the 96-hour period was estimated to be 14% based on the total

metabolism in μg (tissues, bile and urine) divided by the total μg equivalents recovered in carcass, tissues (including bile) and urine. The calculated metabolic rate constant, k_M , determined from this study was 0.166 day^{-1} (personal communication, Environment Canada, Dow Corning Corporation, Health and Environmental Sciences, 2008, unreferenced). However, it is not clear how the metabolic rate was calculated since the calculated metabolic rate is equivalent to a 4.2-day total metabolism, while the total metabolism observed in the study was 14% in 4 days.

Experimental data have also been received on a dietary bioaccumulation study in aquatic organisms (Drottar 2006; see Table 9a of this assessment). A dietary bioaccumulation study of ^{14}C -decamethylcyclopentasiloxane (radiochemical purity 98.7%, 6.09 mCi/g) in rainbow trout (*Oncorhynchus mykiss*) was carried out in a flow-through system (Drottar 2007) for 35 days, followed by 42 days of depuration. Fish were fed trout chow dosed with an average measured parent concentration of 458 $\mu\text{g/g}$. The higher feeding rate was modified to provide better instrument detection and is considered justified. Although the temperature dropped below 8.6°C (value recommended in protocol is $12 \pm 2^\circ\text{C}$) on day 15 of the test and several measurements of dissolved oxygen were below 60%, no adverse effects on the fish were observed throughout the study. The entire extracted radioactivity from fish tissue was identified as parent D5, while unknown metabolites were detected in fish liver and digestive tract. This result indicates that D5 had been metabolized as observed in the D5 fish BCF (Drottar 2005) and metabolic studies (Springer 2007), even though the metabolic rates of D5 are not considered significant (<17% D5 was metabolized). The calculated elimination rate constant indicated that a period of 57 days would be required to achieve 90% of steady state instead of the 35 day uptake in the test. Therefore, a fish residue at day 57 uptake was extrapolated and the corrected fish biomagnification factor, BMF (lipid-normalized), was 0.82. The kinetic BMF was calculated by a model accounting for fish growth rates during the uptake and the depuration phases of the study, the amount of D5 in the fish over time, the mass of the fish over time, as well as the food consumption rate. The metabolic rate constant, k_M , was assumed to be zero. Fish growth rates were calculated by using linear expression (Domoradzki 2008a, 2008b; SEHSC 2008c). The resultant kinetic BMF (lipid-normalized) was 0.91. The BMF values agreed reasonably well. It is therefore considered that D5 did not show biomagnification potential in the laboratory fish dietary study.

Table 9a. Empirical bioaccumulation data for D5

Test organism	Endpoint ¹	Value	Reference
<i>Pimephales promelas</i> (fathead minnow)	BCF _{ss}	7060 L/kg wet wt	Drottar 2005
<i>Pimephales promelas</i> (fathead minnow)	BCF _k	13 300 L/kg wet wt	Drottar 2005
<i>Oncorhynchus mykiss</i> (rainbow trout)	BMF	0.82–0.91 (lipid normalized)	Drottar 2006; Domoradzki 2008a, 2008b; SEHSC 2008c
<i>Chironomus riparius</i> (midge)	BSAF	0.46–1.2	Springborn Smithers Laboratories 2003b

¹BCF_{ss}: steady-state bioconcentration factor; BCF_k: kinetic bioconcentration factor; BMF: biomagnification factor; BSAF: biota-sediment accumulation factor

The Arnot-Gobas model (Arnot and Gobas 2003) can be used to predict the bioaccumulation factor (BAF) of this substance, while taking into account any potential metabolism using a metabolic rate constant (k_M). The available BCF and BMF *in vivo* tests data were used to derive an *in vivo*-based metabolic rate constant according to the method of Arnot et al. (2008a). In this method, k_M is derived according to the following equation:

$$k_M = (k_1\phi/BCF) - (k_2 + k_E + k_G) \quad (1)$$

where

k_M = the metabolic rate constant (1/days)

k_1 = the uptake rate constant (Arnot and Gobas 2003)

ϕ = fraction of freely dissolved chemical in water (Arnot and Gobas 2003)

BCF = the available empirical bioconcentration factor

k_2 = the elimination rate constant (Arnot and Gobas 2003)

k_E = fecal egestion rate constant (Arnot and Gobas 2003)

k_G = growth rate constant (Arnot and Gobas 2003)

The method of Arnot et al. (2008a) provides for the estimation of confidence factors (CF) for the k_M to account for error associated with the *in vivo* data (i.e., measurement variability, parameter estimation uncertainty and model error). A CF of ± 2.6 was calculated for the available BCF and BMF data.

Because metabolic potential can be related to body weight and temperature (e.g., Hu and Layton 2001, Nichols et al. 2007), the k_M was further normalized to 15°C and then corrected for the body weight of the middle trophic level fish in the Arnot-Gobas model (184 g) (Arnot et al. 2008b). The middle trophic level fish was used to represent overall model output as suggested by the model developer (Arnot, personal communication to Bonnell M. of Environment Canada, 2008, unreferenced) and is most representative of fish weight likely to be consumed by an avian or terrestrial piscivore. After normalization routines, the k_M ranges from ~0.001 to 0.01 with a median value of ~0.004.

Table 9b. Metabolism-corrected BCF and BAF estimates using Arnot-Gobas (2003)

k_M (middle trophic level normalized) day^{-1}	log K_{ow} used	Arnot-Gobas BCF	Arnot-Gobas BAF
1.30E-03 (2.5%)	8.0	4265	2 630 268
3.90E-03 (average)	8.0	1905	1 023 293
1.09E-02 (97.5%)	8.0	759	269 153

The calculated k_M values based on *in vivo* experiments suggest that the rate of metabolism of D5 is quite low ($\leq 0.01 \text{ day}^{-1}$ at best). The experimental BCF study on fathead minnows (Drottar 2005), the dietary bioaccumulation study on rainbow trout (Drottar 2006), and the metabolic study (Springer 2007) demonstrated metabolism, though limited, of D5. The calculated BCF of 4265 using the lower percentile rate constant (~ 0.001) is approximately 50% lower than the BCF values reported by Drottar (2005), but is within the acceptable range of the experimental values. A corresponding BAF of 2 630 268 was calculated for D5 for fish in Canadian waters using this metabolic rate constant.

In the Sediment Compartment

New information was received on D5 biota-sediment accumulation factors (BSAFs). The potential for bioaccumulation from sediment was investigated during a sediment toxicity test with midges (*Chironomus riparius*) (Springborn Smithers Laboratories 2003b; see Table 9a of this assessment). Midge larvae were exposed to measured concentrations of sediment-incorporated radio-labelled ^{14}C -D5 (99.37% radiochemical purity) ranging from 13 to 580 mg/kg (mean measured concentration from test days 0 and 10). Artificial sediment was used, composed of 79% sand, 4% silt and 17% clay. Tests were conducted with sediments of 2.0% organic carbon content. The measured pH of the sediment was 6.6–7.6 and the measured temperature was 19–22°C. The midge whole dry weight BSAF was calculated to be 1.2, 1.1, 0.83 and 0.46 at treatment levels of 13, 30, 73 and 180 mg/kg, respectively. The results indicate that D5 may have some potential to bioaccumulate through exposure to sediment. However, this study did not specify whether the gut contents of test organisms had been purged before calculation of BSAF values. The BSAF value may thus be over-estimated due to the presence of D5 on sediments within the gut of the invertebrates.

In the Soil Compartment

No bioaccumulation information for D5 was available for the soil compartment.

In the Terrestrial Compartment

The Gobas mass-balance bioaccumulation model for terrestrial organisms (Gobas et al. 2003) uses a chemical's octanol-air and octanol-water partition coefficient (K_{oa} and K_{ow}) to estimate the chemical's biomagnification (BMF) potential in terrestrial food chains. It was estimated that chemicals with a $\log K_{oa} > 5$ can biomagnify in terrestrial food chains if $\log K_{ow} > 2$ and the rate of chemical transformation or metabolism is low. A $\log K_{oa}$ of 5.06 indicates that D5 may have the potential to biomagnify in terrestrial food chains. However, the metabolism of cVMS demonstrated in laboratory mammals could reduce the biomagnification potential in the terrestrial food web.

Summary of the Bioaccumulation Potential of D5

Overall, the empirical steady-state and kinetic fish BCF study, although optimized for water-borne exposure, has shown that the bioconcentration potential for D5 from water is high (i.e., ≥ 5000). Although the $\log K_{ow}$ for D5 would suggest that dietary uptake will be significant and likely predominate, it is still at the limit of $\log K_{ow}$ values where bioconcentration in laboratory studies has been observed to be significant for many chemicals (e.g., Arnot and Gobas 2006). Empirical values exceed 5000, which suggests that there may be potential for high bioconcentration in other organisms at different trophic levels as well as fish, especially those with lower growth or metabolic rates (e.g., autotrophs).

Predicted BCF values, corrected for metabolism, are close to but less than 5000 and are lower than empirical BCF values. Predicted BAF values are also high and exceed 5000. As with other models, some uncertainty exists with predicted BCF and BAF values (e.g., uncertainty increases at higher $\log K_{ow}$ values, as few chemicals have been studied for bioaccumulation in this range). Higher confidence is attributed to the predicted BAF value with a corresponding predicted BCF that most closely compares with the empirical BCF data (i.e., $BAF = \sim 2.6E06$). The mass-balance kinetic model used is based on “first principles,” meaning that the most important domain of the model is that a chemical obeys the principal mechanism of the model, in this case passive diffusion. D5 meets this domain and is within the model’s $\log K_{ow}$ and molecular weight boundaries as well. Therefore, the predictions for bioaccumulation are considered to be applicable to D5. However, there is less agreement between metabolism-corrected BCF values and empirical BCF values (compared with D4), suggesting that BCF and BAF model results for D5 are more uncertain due to the relative absence of empirical BCF and BAF values at D5’s range of $\log K_{ow}$ used in the model (i.e. ≥ 8). Also, the possibility that super-hydrophobic chemicals do not reach steady state in the environment (which the model assumes), adds to the uncertainty (Arnot and Gobas 2006).

BSAF values for D5 would suggest a relatively low level of accumulation in sediment macroinvertebrates. As this is the only sediment bioaccumulation test available for cVMS and there are no predictive models for sediment organisms, testing or field evidence at more realistic environmental loadings would help verify these values. The BMF values generated for D5 are less than 1, which suggests that there may be low biomagnification potential in fish for D5, but there is currently no evidence to suggest that this may be the case for other trophic levels. Field mesocosm studies are currently under way to examine trophic transfer of cVMS in aquatic food webs, but these data are not yet available for full evaluation and were not considered for this assessment.

Finally, there is conflicting evidence on the bioaccumulation potential of D5 tested under laboratory conditions as well as that determined from predictive models. BMF studies in fish and BSAF studies in invertebrates suggest that the bioaccumulation potential of D5 is low, possibly due to reduced bioavailability. Available optimized BCF test data suggest there may be potential for significant bioconcentration of D5 in fish and potentially at lower levels of an aquatic food web. Predicted BCF values are much lower than measured values

for fish. Therefore, it is reasonable to conclude that D5 has bioaccumulation potential in biota. However, considering the conflicting evidence, it is not possible to conclude that D5 meets the criterion for bioaccumulation (BCF or BAF ≥ 5000) as set out in the *Persistence and Bioaccumulation Regulations* (Canada 2000).

Potential to Cause Ecological Harm

Ecological Effects Assessment

A - In the Aquatic Compartment

New empirical ecotoxicity data have been received for D5 and are discussed in detail below (Table 10a). These studies indicate that this substance does not exhibit adverse effects on fish and *Daphnia* exposed at concentrations up to its water solubility limit (0.017 mg/L).

The aquatic toxicity of D5 to rainbow trout (*Oncorhynchus mykiss*) was conducted under a flow-through system (Springborn Laboratories 2000). Juveniles of rainbow trout (mean wet weight 2.3 g and mean length 61mm) were exposed to concentrations of D5 (99.35% purity) of 2.1, 3.1, 5.0, 8.6 and 16 µg/L (measured) for 14 days. The test system was sealed and filled to capacity to minimize the loss of D5 from the test solution. Acetone was used as a solubilizer at a concentration of 0.1 mL/L. The pH was maintained between 6.3 and 6.9 and the temperature oscillated between 13 and 15°C. Fish were fed ~2% of the total biomass of the test fish once daily. The temperature during the test deviated slightly from the OECD protocol (12 ± 2°C) and the dissolved oxygen concentration fell below 60% of saturation for more than 24 hours, but these factors were not considered to interfere with the test. There was 5–10% mortality in the negative and solvent controls, as well 10% mortality in the 0.005 mg/L and 15% mortality in the 0.0086 mg/L treatment groups. However, the mortality observed was not considered test-substance-related since no mortality was found in the highest concentration treated (0.016 mg/L).

Table 10a. Empirical aquatic toxicity values for D5

Test organism	Type of test	Duration	Endpoint ¹	Value (mg/L)	Reference
Rainbow trout <i>Oncorhynchus mykiss</i>	Acute	14 d	LC ₅₀	> 0.016	Springborn Laboratories 2000
Rainbow trout <i>Oncorhynchus mykiss</i>	Acute	14 d	NOEC	0.016	Springborn Laboratories 2000
Water flea <i>Daphnia magna</i>	Acute	48 hr	LC ₅₀	> 0.003	Springborn Smithers Laboratories 2002
Water flea <i>Daphnia magna</i>	Chronic	21 d	NOEC	0.015	Springborn Smithers Laboratories 2003a
Freshwater algae <i>Pseudokirchneriella subcapitata</i>	Acute	96 hr	EC ₅₀	invalid	Springborn Laboratories 2001

¹ LC₅₀: the lowest concentration causing 50% mortality; EC₅₀: the lowest concentration causing sublethal effects in 50% of the population; NOEC: no-observed-effect concentration

Both acute and chronic toxicity studies of D5 were carried out for *Daphnia magna*. Juveniles of *Daphnia magna* were exposed to D5 (99.35% purity) concentrations of 1.6, 1.8, 2.1, 2.5 and 2.9 µg/L (measured) for 48 hours under flow-through conditions (Springborn Smithers Laboratories 2002). The exposure system was designed to have minimum

volatilization of the test substance. Acetone was used as a solubilizer at a concentration of 0.1 mg/L. The pH was maintained between 7.7 and 8.0, the temperature oscillated between 20 and 21°C and dissolved oxygen remained at > 60%. The test water had a hardness of 160–180 mg CaCO₃/L. A mortality of ≤10% was observed at the control and test concentrations, resulting in an EC₅₀ ≥ 2.9 µg/L.

A full life-cycle toxicity test of D5 with *Daphnia magna* was conducted under static-renewal conditions (Springborn Smithers Laboratories 2003a). Juveniles of *Daphnia magna* were exposed to radio-labelled ¹⁴C-D5 (100% radiochemical purity, 5.758 mCi/g specific activity) at concentrations of 1.0, 1.7, 3.5, 7.2 and 15 µg/L (measured) for 21 days under static-renewal conditions, and the test solutions were renewed every 24 hours. The test system was designed to minimize the loss of D5 from the test solution. Acetone was used as a solubilizer at a concentration of 0.1 mL/L. The pH was maintained between 6.6 and 8.7, the temperature oscillated between 20 and 23°C and dissolved oxygen remained at > 60%. The test water had a hardness of 160–170 mg CaCO₃/L. At the end of the 21-day exposure period, the mean offspring per female were 150, 148, 145, 138 and 139, respectively for treatment concentrations of 1.0, 1.7, 3.5, 7.2 and 15 µg/L. The offspring number decreased slightly at treatment concentrations of 7.2 and 15 µg/L compared to negative and solvent controls (mean offspring number 146), though it was not considered statistically significant. No mortality, growth or other biologically significant effects were observed at the highest test concentration of 15 µg/L (no-observed-effect concentration ≥ 15 µg/L). The temperature during the test deviated slightly from the OECD protocol (20 ± 1°C), but was not considered to interfere with the test. However, the feeding during the test was 0.41 mg C/daphnid/day—higher than the OECD guideline of 0.1–0.2 mg C/daphnid/day—which is considered favourable to the health of the test organisms in resistance to toxic effects. A higher carbon load to the test system may also act to reduce bioavailability of D5 by providing a sorption substrate. Nonetheless, the experimental data were considered acceptable for the purposes of the screening assessment, noting possible test limitations as mentioned.

New experimental data were received on an acute toxicity study on freshwater algae, *Pseudokirchneriella subcapitata* (formerly known as *Selenastrum capricornutum*) under a 96-hour static condition (Springborn Laboratories 2001). The toxicity test was performed following *Toxic Substances Control Act* (TSCA) and Organisation for Economic Co-operation and Development (OECD) test standards for algae. Algae were exposed to a saturated solution of D5 (99.35% active ingredient). The test system was sealed and filled to capacity to minimize the loss of D5 from the test solution. Acetone was used as a solubilizer at a concentration of 0.1 mL/L. However, the concentrations of the test solution decreased steadily, from 12 µg/L at test initiation to ~2 µg/L at the end of the 96-hour test. It was hypothesized that the loss of D5 may be attributed to volatilization, adsorption of D5 to algae biomass or to its consumption as a carbon source for algae. Hydrolysis under alkaline conditions may also contribute to the rapid loss of D5, as the pH increased from 7.5 at test initiation to 9.4 during the 96-hour test, which reflected photosynthesis and respiration of the algae. The temperature during the study was maintained at 24°C. It is concluded that no decrease of cell density or growth rate was observed during the test. An additional 500 mg/L sodium bicarbonate was added to provide sufficient dissolved bicarbonate to compensate for

cell growth in a closed system. Considering the significant reduction of D5 concentrations during the test and all the uncertainties associated with the study, the results of the test is considered invalid.

The acute and chronic toxicity of D5 was predicted using ECOSAR (2004) model. Predicted results are given in Table 10b.

Table 10b. Modelled aquatic toxicity values for D5

Organism	Type of test	Endpoint ¹	Duration	Concentration (mg/L)	Reference
Fish	Chronic	ChV	30 d	0.0002	ECOSAR 2004
<i>Daphnia</i>	Chronic	EC ₅₀	16 d	0.0007	ECOSAR 2004
Green algae	Chronic	ChV	96 h	0.003	ECOSAR 2004

¹ EC₅₀: the lowest concentration causing sublethal effects in 50% of the population; ChV is the geometric mean of the NOEC (no-observed-effect concentration) and LOEC (lowest-observed-effect concentration).

The modelled results for D5 using ECOSAR are below the water solubility limit of 0.017 mg/L, which would suggest acute or chronic effects at aqueous saturation. However, the reliability of the predicted toxicity values is considered to be low. The log K_{ow} for D5 (8.03) is narrowly higher than the recommended ECOSAR log K_{ow} cut off (8.0) for prediction of chronic toxicity using the neutral organic structure-activity relationships and the lack of empirical siloxane data in the model's training set limit their consideration in this assessment. Also, the reliability of acute and chronic toxicity predictions at and below the microgram per litre range becomes increasingly uncertain given that these predictions rely solely on log K_{ow} and the empirical ability to quantify effects in this range is highly variable. The model predicted aquatic toxicity data were, therefore, not considered further.

The empirical data suggest that D5 does not exhibit adverse effects on fish and *Daphnia* at concentrations at or below its solubility limit (0.017 mg/L). However, caution should be exercised in relation to these results. As mentioned in trout and *Daphnia* toxicity studies conducted with the analogue D4, mortality caused by D4 was not observed until the test organisms were exposed to the substance for 7–14 days, at which time the D4 concentration in fish tissue reached steady state and a body burden sufficient to cause toxicity (Sousa et al. 1995). With D5, there is uncertainty as to whether tissue concentrations achieved steady-state within 14 days during the BCF study with fathead minnows (Drottar 2005). This calls into question whether the 14-day toxicity test duration with D5 and trout (Springborn Laboratories 2000) was sufficiently long to achieve a toxic body burden in the fish tissue and, thus, sufficiently long to determine toxicity in the study. Another observation is that D4-related mortality was only observed in small rainbow trout (≤ 1 g) and not larger trout or other fish species, indicating that D4 could be more toxic to sensitive aquatic organisms and/or aquatic organisms at sensitive early-life stages. D5, however, was only tested with larger trout (~2.3 g), and no early-life stage tests were conducted. Due to the structural similarity between D4 and D5, it is possible that D5 toxicity may have been observed had smaller trout been used. Therefore, questions regarding the sufficiency of the test duration, as well as a lack of empirical data comparable to that in which D4 toxicity was noted (i.e.,

comparable fish size), as well as a lack of empirical data for early-life stage fish contributes to the uncertainty in the toxicity of D5 to aquatic organisms.

B – In Other Environmental Compartments

In the Sediment Compartment

New empirical sediment toxicity data were received for D5 and have been accepted for this screening assessment report.

A full life-cycle toxicity test with D5 in sediments was conducted using midge (*Chironomus riparius*) in a series of 10-day and 28-day exposures under static conditions (Springborn Smithers Laboratories 2003b). Midge larvae were exposed to measured concentrations of sediment-incorporated radio-labelled ¹⁴C-D5 (99.37% radiochemical purity) ranging from 13 to 580 mg/kg (mean measured concentration from test days 0 and 10) and 12 to 570 mg/kg (mean measured concentration from test days 0, 10 and 28). Artificial sediment was used, composed of 79% sand, 4% silt and 17% clay. Tests were conducted with sediments of 2.0% organic carbon content. The measured pH of the sediment was 6.6–7.6 and the measured temperature was 19–22°C. The midge survival rates determined after 10 days of exposure were 100%, 97%, 98%, 97% and 27%, respectively, for treatment levels of 13, 30, 73, 180 and 580 mg/kg. The average wet weights determined after 10 days of exposure were 5.59, 5.65, 4.77, 4.44 and 1.36 mg/midge, respectively for treatment levels of 13, 30, 73, 180 and 580 mg/kg. The 10-day median lethal concentration (LC₅₀) for midge survival was calculated to be 450 mg/kg dry weight and the median effects concentration (EC₅₀) for midge growth was calculated to be 410 mg/kg dry weight (Table 8c). The emergence and development rates of midge larvae were determined after 28 days of exposure. The mean percent emergence in the 470 mg/kg treatment level was significantly lower (18%) than that in the control (87%). The mean development rates of male midges at the 180 and 570 mg/kg treatment levels were significantly decreased compared to the control. The decrease in mean development rates among females and combined males/females were only significant at the 570 mg/kg treatment level. The 28-day no-observed-effect concentration (NOEC) for midge larvae development is 69 mg/kg and the lowest-observed-effect concentration (LOEC) for midge larvae development is 180 mg/kg (Table 10c).

Table 10c. Empirical sediment toxicity values for D5

Test organism	Type of test	Endpoint ¹	Value (mg/kg dry weight)	Reference
Midge <i>Chironomus riparius</i>	Acute	LC ₅₀	450	Springborn Smithers Laboratories 2003b
Midge <i>Chironomus riparius</i>	Acute	EC ₅₀	410	Springborn Smithers Laboratories 2003b
Midge <i>Chironomus riparius</i>	Chronic	Male development NOEC	69	Springborn Smithers Laboratories 2003b
Midge <i>Chironomus riparius</i>	Chronic	Male development LOEC	180	Springborn Smithers Laboratories 2003b
Midge <i>Chironomus riparius</i>	Chronic	Female development NOEC	180	Springborn Smithers Laboratories 2003b
Midge <i>Chironomus riparius</i>	Chronic	Male/female development NOEC	180	Springborn Smithers Laboratories 2003b
Midge <i>Chironomus riparius</i>	Chronic	Emergence NOEC	180	Springborn Smithers Laboratories 2003b
Oligochaete <i>Lumbriculus variegates</i>	Chronic	NOEC	1272	Krueger et al. 2007
Midge <i>Chironomus riparius</i>	Chronic	LC ₅₀	257	Krueger et al. 2008
Midge <i>Chironomus riparius</i>	Chronic	Development NOEC	70	Krueger et al. 2008
Midge <i>Chironomus riparius</i>	Chronic	Development LOEC	160	Krueger et al. 2008
Midge <i>Chironomus riparius</i>	Chronic	Emergence NOEC	160	Krueger et al. 2008
Midge <i>Chironomus riparius</i>	Chronic	Emergence LOEC	248	Krueger et al. 2008

¹ LC₅₀: the lowest concentration causing 50% mortality; EC₅₀: the lowest concentration causing sublethal effects in 50% of the population; NOEC: no-observed-effect concentration; LOEC: lowest-observed-effect concentration

A recent chronic toxicity study (Krueger et al. 2007) with the freshwater oligochaete *Lumbriculus variegates* (blackworm) was also critically reviewed and accepted for this screening assessment. Adult blackworms were exposed to measured concentrations of sediment-incorporated D5 (99.19% purity) ranging from 24 mg/kg to 1272 mg/kg (mean measured concentration from test days 0, 7 and 28) for 28 days under flow-through conditions. Formulated sediment was used, composed of 13% peat, 10% kaolin clay and 77% industrial quartz sand. The measured pH of the sediment was 7.9–8.3 and the measured temperature was 22.7–23.4°C (OECD protocol 20+/-2). The organic carbon content of the sediment was 3.7% based on peat as the sole source of organic carbon. Since blackworms reproduced during the 28-day test and it was hard to distinguish adults from young organisms, the total number of organisms present at test termination was used as the measurement endpoint. The mean number of worms was 33, 30, 31, 21, 19 and 26, respectively for treatment levels of 24, 46, 94, 226, 495 and 1272 mg/kg dry weight. No mortality was observed throughout the test. It was concluded that the mean numbers were not significantly different from that of control (26). No effects were observed for the growth of the surviving worms. The observed NOEC was thus determined to be 1272 mg/kg.

Another prolonged sediment toxicity study on midges, *Chironomus riparius*, was conducted using spiked sediment (Krueger et al. 2008). The midges were exposed to mean measured concentrations of D5 ranging from 35 mg/kg to 759 mg/kg for 28 days at 20°C. The organic carbon content of the formulated sediment was 3.2%. The overlaying water was renewed partially every week due to the high ammonia measured in the test chamber. The observed NOEC for percent survival and emergence ratio was determined to be 160 mg/kg (measured). The calculated LC₅₀ value for survival was 257 mg/kg. Midges exposed to 160 mg/kg of D5 showed statistically significant reduction in development. The NOEC and LOEC for midge development were determined to be 70 mg/kg and 160 mg/kg, respectively.

In the Soil Compartment

No effects studies for soil organisms were found for D5 or its analogues.

In the Terrestrial Compartment

No ecological studies were identified for terrestrial wildlife. Laboratory studies on mammals are discussed under the “Potential to Cause Harm to Human Health” section in this screening assessment.

Ecological Exposure Assessment

In Air

In Canada, preliminary environmental measurement of volatile methyl siloxanes, including D5, were conducted in the Great Lakes region during February and March of 2006 (personal communication, Environment Canada, Canadian Centre for Inland Waters, 2007, unreferenced). Eighteen outdoor air samples were collected from rural and urban areas in Ontario and D5 was present in almost all the samples at concentration levels of < 1 µg/m³, with the exception of one relatively high D5 concentration, reported to be ~20.5 µg/m³, in the Toronto urban area. This result is in agreement with what has been reported in other jurisdictions (Table 11a).

It is, however, possible that the detection of D5 in ambient air is in part a result of sample contamination. Volatile cyclosiloxanes are present in a wide variety of commercial products, and both Canadian and Nordic monitoring programs have reported problems of high levels of cyclosiloxanes in sample blanks. The methodology for measuring and analyzing air concentrations at ng/m³ to the low µg/m³ level is still under development. Very few duplicate measurements are available for outdoor air monitoring and the few that are available exhibit poor reproducibility. It has been hypothesized that the particulate phase may be important to consider and that this could influence results for duplicates (personal communication, Environment Canada, Canadian Centre for Inland Waters, 2007, unreferenced).

Table 11a. Concentrations of D5 in air

Medium	Location; year	Concentration	Reference
Air	Great Lakes region, Canada; February and March 2006	< 1–20.5 µg/m ³	see footnote 2
Air	Nordic countries ¹ ; 2004–2005	0.05–19 µg/m ³	Norden 2005

¹ Outdoor samples (n=24) were collected in Nordic countries. The detection limit for D5 was 0.02 µg/m³.

² Personal communication, Environment Canada, Canadian Centre for Inland Waters, 2007, unreferenced

In Water

In Canada, water from a total of nine sewage treatment plants (STPs), including conventional secondary and tertiary water treatment plants and lagoons, in large urban centres in southwestern Ontario was sampled in October 2005 and winter 2005 (personal communication, Environment Canada, Canadian Centre for Inland Waters, 2007, unreferenced). D5 was detected at concentrations of 0.49–227.72 µg/L and 1.00–2.29 µg/L, respectively, in the influents and effluents. Seasonal differences in D5 concentrations in influents in STPs were also noted; most influent concentrations increased from 0.49–57.33 µg/L in the fall to 8.04–227.72 µg/L in the winter. Seasonal differences in D5 concentrations in effluents in STPs were also observed but these were not as significant as those for influents (from 1.00–1.30 in the fall to 1.65–2.29 in the winter).

Similar monitoring results have been reported in other jurisdictions (Table 11b). In the United States, D5 has been qualitatively detected in drinking water systems (Lucas 1984, as cited in US EPA 1992). In Europe, water samples were also collected from influents, effluents and downstream of sewage treatment plants including some associated with silicone manufacturing facilities (Boehmer and Gerhards 2003; see Table 11b of this assessment). Higher concentrations of D5 were detected in the influents and effluents of plants associated with silicone industries. Most water samples downstream of silicone industries contained non-detectable levels of D5 with one exception where the concentration reported was 0.4 µg/L.

Table 11b. Concentrations of D5 in water

Medium	Location; year	Concentration	Reference
STP influents	Southwestern Ontario, Canada; October 2005	0.49–227.72 µg/L	See footnote 9
STP effluents	Southwestern Ontario, Canada; October 2005	1.00–2.29 µg/L	See footnote 9
Drinking water	United States	Qualitatively detected	Lucas 1984, as cited in USEPA 1992
Water	Background and urban sites, ¹ Nordic countries	< 0.07 (d.l.)	Norden 2005, NILU 2007
Water	Germany ⁴ ; 2000–2001	< 0.04 µg/L (d.l.)	Boehmer and Gerhards 2003
STP influents	Nordic countries ²	0.33–26 µg/L	Norden 2005, NILU 2007
STP effluents	Nordic countries ³	< 0.063–1.0 µg/L	Norden 2005, NILU 2007
STP influents	Germany ⁵ ; 2000–2001	1.3–50.1 µg/L	Boehmer and Gerhards 2003
STP effluents	Germany ⁶ ; 2000–2001	0.1–1.0 µg/L	Boehmer and Gerhards 2003
STP influents	Wastewater treatment plants (WWTPs) associated with silicone industries in Germany, France and the United Kingdom (UK) ⁷ ; 2001	365–3694 µg/L	Boehmer and Gerhards 2003
STP effluents	WWTPs associated with silicone industries in Germany, France and the UK ⁸ ; 2001	< 0.02 (d.l.) – 26.7 µg/L	Boehmer and Gerhards 2003
Water	Downstream of WWTPs associated with silicone industries in Germany, France and the UK ⁸ ; 2001	< 0.02 (d.l.) – 0.4 µg/L	Boehmer and Gerhards 2003

¹ A total of 28 sampling sites excluding STP influents and effluents

² 7 STP influent sampling sites

³ 12 STP effluent sampling sites

⁴ A total of 12 marine water samples collected at 2 locations

⁵ 7 STP influent samples collected

⁶ 4 STP effluent samples collected

⁷ A total of 5 samples collected at 2 locations

⁸ A total of 8 samples collected at 3 locations

⁹ Personal communication, Environment Canada, Canadian Centre for Inland Waters, 2007, unreferenced

d.l. = detection limit

In Sediments

In Canada, surface sediments and sediment cores were collected from Lake Ontario in July 2006 and analyzed for D4, D5 and D6 (Powell and Kozerski 2007). Surface sediments consisting of the upper 5 cm of sediment were collected from Toronto Harbour and the Kingston Basin. Sediment cores, which were sectioned into strata 5 mm thick, were collected from the Rochester, Mississauga, and Niagara basins. The surface sediments from Toronto Harbour and Kingston Basin contain moderate total organic carbon (TOC = 2.1–2.4% dw), while sediment cores contain high TOC (4–5% dw). Loss-on-ignition analysis of sediments also demonstrated lower water contents in surface sediments (55–70% ww) than

in sediment cores (80–89%). Sediments in Toronto Harbour and the four sedimentary basins are known to be contaminated with a variety of organic compounds that enter the lake through direct discharges of treated wastewater, flow from the upper Great Lakes (Erie, Huron and Michigan) and the Niagara River, and atmospheric deposition. Surface sediments from Toronto Harbour contained the highest concentration of D5, at 0.78 µg/g dry weight. In contrast, concentrations of the cyclic siloxane materials in the surface sediments and sediment cores from the four sedimentary basins were all less than the analytical method detection limit, which was 0.017 µg/g for D5. Similar monitoring results have been reported in other jurisdictions, where D5 was detected in surface sediments from urban areas and point sources (Table 11c).

A preliminary monitoring study of a remote ecosystem was conducted in Lake Opeongo, the largest lake in Algonquin Provincial Park, Ontario, Canada. The lake is relatively remote from potential sources of cVMS from sewage and runoff (Powell 2008). Preliminary analysis of surface sediment and sediment core samples found no D5, with a limit of detection of 15.9 ng.

The sediment monitoring results from Lake Opeongo and the Lake Ontario area suggest that D5 contamination is more likely to be found near urban centres and point sources.

Table 11c. Concentrations of D5 in sediments

Medium	Location; year	Concentration	Reference
Surface sediments	Toronto Harbour, Canada; July 2006	0.78 µg/g dw	Powell and Kozerski 2007
Surface sediments	Kingston Basin, Canada; July 2006	<0.017 µg/g dw (d.l.)	Powell and Kozerski 2007
Sediment cores	Rochester, Mississauga, and Niagara basins, Canada; July 2006	<0.017 µg/g dw (d.l.)	Powell and Kozerski 2007
Surface sediments	Lake Opeongo, Algonquin Provincial Park, Ontario, Canada; October 2007	15.9 ng (d.l.) ⁴	Powell 2008
Sediment cores	Lake Opeongo, Algonquin Provincial Park, Ontario, Canada; October 2007	15.9 ng (d.l.) ⁴	Powell 2008
Sediments	Nordic countries ¹	< d.l. (varied from sample to sample) – 0.92 µg/g dw	Norden 2005, NILU 2007
River sediments	Germany ²	< 0.003 (q.l.) – 0.042 µg/g dw	Boehmer and Gerhards 2003
Marine sediments	Germany ³	0.033–0.28 µg/g dw	Boehmer and Gerhards 2003

¹ A total of 30 sediment sampling sites

² A total of 11 sampling matrix at 9 locations

³ A total of 12 marine sediment samples from 2 areas

⁴ Background corrected mass as reported in the preliminary study
dw = dry weight, d.l. = detection limit, q.l. = quantification limit

In Soil

D5 may enter soil from land application of sewage sludge. No monitoring data for D5 in sewage sludge are available for Canada. In Europe, D5 is present in sewage sludge at levels ranging from the low mg/kg dry weight level up to 130 mg/kg dry weight (Norden 2005, Kaj et al. 2005, NILU 2007).

No monitoring data are available for D5 in Canadian soil. D5 concentrations in two soil samples from the Faroe Islands were below detection limit (< 5 ng/g dw) (Norden 2005).

In Biota

A preliminary monitoring study of a remote ecosystem was conducted in Lake Opeongo, the largest lake in Algonquin Provincial Park, Ontario, Canada. The lake is relatively remote from potential sources of cVMS from sewage and runoff (Powell 2008). Preliminary analysis of zooplankton samples found no D5. Bulk zooplankton samples were pooled into a single sample for each of the two locations from the lake without being sorted into species. The limit of detection was 15.9 ng (background corrected mass).

In Europe, D5 was the predominant cyclosiloxane in fish livers and marine mammals in the Nordic screening program. The substance was detected in both freshwater and marine fish from sampling sites in urban areas and near STPs, at concentrations in the range of < 5–84 ng/g wet weight, except for one sample of cod liver (9 livers pooled) collected at a location near a city centre in Norway that had an extremely high concentration of D5 (2200 ng/g ww). The follow-up environmental monitoring program conducted by the Norwegian government confirmed the originally reported levels of D5 in cod livers (NILU 2007; see Table 11d of this assessment). D5 was also found in common mussels, flounder livers and fillets, and in cod stomach contents from Norway in the same monitoring program. The concentrations varied with species, gender, and age. D5 was also detected in the blubber of seals and pilot whales at concentrations ranging from <5 to 24 ng/g ww (Norden 2005). D5 was also detected in fish samples in Germany at concentrations ranging from 0.15 to 2.6 mg/kg (SEHSC 2005a). D5 was not detected in fish muscle samples in Sweden (Kaj et al. 2005).

The presence of D5 in European biota indicates that despite the low detected concentrations or even non-detection of the substance in or near fish habitats, D5 is available in the environment for biota to take up and accumulate.

Table 11d. Concentrations of D5 in biota

Organism	Location; year	Concentration	Reference
Zooplankton	Lake Opeongo, Algonquin Provincial Park, Ontario, Canada; October 2007	< 15.9 ng (d.l.)	Powell 2008
Marine fish liver	Nordic countries ¹ ; 2002–2004	< 5 (d.l.) – 2200 ng/g ww	Norden 2005
Freshwater fish liver	Nordic countries ² ; 2002	< 5 (d.l.) – 84 ng/g ww	Norden 2005
Marine mammals	Nordic countries ³ ; 2002	< 5 (d.l.) – 24 ng/g ww	Norden 2005
Seabird eggs	Nordic countries ⁴ ; 2000–2005	< 5 ng/g ww (d.l.)	Norden 2005
Common mussels	Norway ⁵ ; 2006	3.3–8.7 ng/g ww	NILU 2007
Flounder livers	Norway ⁶ ; 2006	27.1 ng/g ww	NILU 2007
Flounder fillets	Norway ⁶ ; 2006	3.4 ng/g ww	NILU 2007
Cod stomach contents	Norway ⁷ ; 2006	22.9–149.3 ng/g ww	NILU 2007
Cod livers	Norway ⁷ ; 2006	1490.8–1978.5 ng/g ww	NILU 2007
Fish	Rhine River, ⁸ Germany	150–2600 ng/g ww	SEHSC 2005b

¹ A total of 11 sampling matrices for marine fish

² A total of 10 sampling matrices for freshwater fish

³ A total of 7 sampling matrices for marine mammals

⁴ A total of 17 sampling matrices for seabird eggs

⁵ A total of 3 sampling matrices for mussel

⁶ A total of 2 sampling matrices for flounder

⁷ A total of 3 sampling matrices for cod

⁸ A total of 5 fish matrices were sampled from the Rhine River; a Danish salmon obtained from an unspecified location showed no detectable D5

ww = wet weight, d.l. = detection limit, q.l. = quantification limit

Characterization of Ecological Risk

The approach taken in this ecological screening assessment was to examine various supporting information and develop conclusions based on a weight-of-evidence approach and using precaution as required under subsection 76.1 of CEPA 1999. Particular consideration was given to risk quotient analysis, persistence, bioaccumulation potential, toxicity, sources and fate in the environment.

Based on the information available, D5 has been determined to be highly persistent in water and potentially bioaccumulative in aquatic organisms. A quantitative risk quotient evaluation of exposure and of ecological effects was therefore conducted as part of the weight-of-evidence evaluation of D5's potential to cause harm.

In the aquatic compartment, results of two acceptable experimental chronic toxicity studies with *Daphnia magna* and rainbow trout indicated that D5 caused no significant adverse effects at concentrations of up to 0.015 mg/L, a concentration that is very close to the water solubility limit of the substance (~0.017 mg/L). No application factor was applied to the

chronic NOEC of 0.015 mg/L. The calculated predicted no effect concentration (PNEC) is therefore 0.015 mg/L. It is considered that D5 exposure to aquatic organisms may reach the level above its laboratory-measured water solubility under realistic environmental conditions.

A risk quotient (RQ) analysis, integrating level of exposure with a toxicity threshold, was performed for D5. In order to address the potential risk of D5 on a national scale in Canada, a distribution profiling risk quotients in water at multiple release sites where the substance can be released by industry or by consumers (i.e., municipal sewage treatment plants) was determined. This type of analysis provides a line of evidence for the risk assessment of a substance when the full range of geographic locations of the industrial and consumer releases of the substance cannot be fully established.

Specifically, when a substance is used by a number of industry sectors but the actual facilities involved cannot be identified, the aquatic exposure can be estimated for all sites where facilities related to these sectors are located. In addition to this, information on potential releases from consumer use can be integrated into the calculations. A predicted environmental concentration (PEC) for the water compartment is determined based on the use quantities identified from the section 71 survey submissions and estimates of releases from individual industrial sites and from consumers. The receiving water is either a watercourse or a lake, and a dilution factor based on the size of the receiving water—up to a maximum of 10—is used to estimate the PEC. The risk quotient at each site is then determined for the water column. The distribution indicates not only the proportion or number of threshold-exceeding sites, but also the magnitude of the exceedence at each of these sites. Further details on the approach are provided in Environment Canada (2008b).

The consumer releases used a database of approximately 1000 municipal discharge sites accounting for about 2/3 of the Canadian population. The industrial release analysis was done for 64 sites relating to 87 industrial facilities identified by NAICS code as possible users of D5. Under these scenarios, a total of 65 (~6.8%) of all evaluated municipal discharge sites across Canada showed a risk to aquatic organisms, with RQs exceeding 1 (Figure 1). The equation and inputs used to calculate the PEC in the receiving watercourses are described in Environment Canada (2008c).

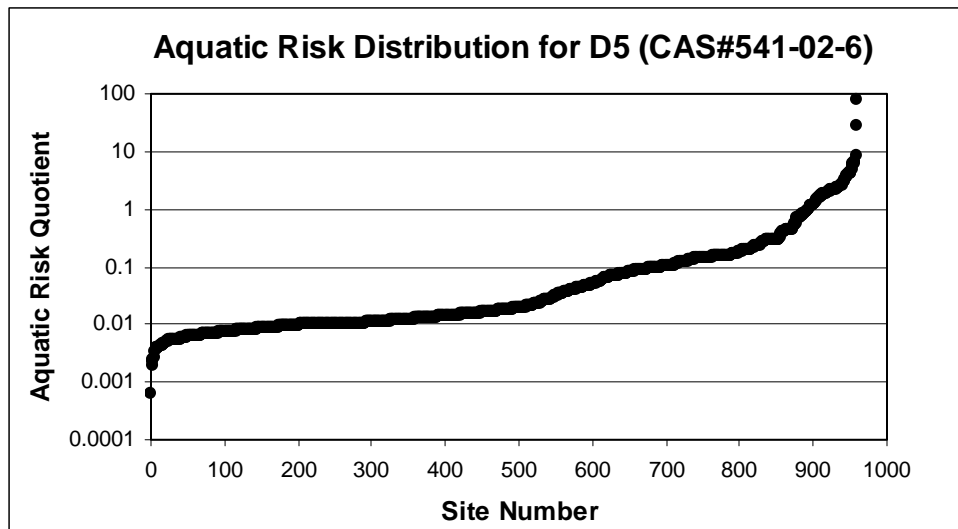


Figure 1. Aquatic risk distribution for D5 (Environment Canada 2008b, 2008c)

Although a risk quotient analysis was conducted for D5, the empirical ecotoxicity evidence suggests that the threshold at which adverse effects in pelagic biota is expected to occur has not been observed in available toxicity tests. Therefore, the RQs calculated in the above scenario are essentially “unbounded” and may not represent “real” observable effects expected at the above sites.

The high $\log K_{ow}$ for D5 would suggest that it is in the class of “super-hydrophobic” chemicals which are often associated with low bioavailability to pelagic and benthic biota. However, because the $\log K_{ow}$ for D5 is not higher than ~ 8.0 and a threshold for effects in benthic biota was observed, some degree of bioavailability is expected in the environment, especially for longer term exposures. Laboratory bioconcentration data, albeit optimized, as well as evidence of detection in biota sampled from the field suggest that the potential for body burdens reaching critical internal levels may not be mitigated by the expected bioavailability of D5, especially near STP outfalls.

The physical-chemical property, bioconcentration and ecotoxicity profiles for D5 do not provide a consensus basis for a weight of evidence. There are some uncertainties as to whether D5 poses the potential to cause ecological harm. D5 is imported into Canada in significant quantities and is considered persistent in the environment. Based on the information available, the quantities of D5 imported and used in Canada have increased significantly since the DSL nomination (Environment Canada 1988). Given the trend of increasing use, its environmental release and potential for environmental exposure may increase.

Therefore, considering the above, a reasonable level of precaution is required and as such it is concluded that D5 may have the potential to cause ecological harm when released to the Canadian environment, particularly for long term exposures near discharge zones.

Uncertainties in Evaluation of Ecological Risk

D5 is one of the major components in CAS RN 69430-24-6. It is also present in PDMS at up to 3%. The Challenge to industry and other stakeholders issued by the Government of Canada (Canada 2007) did not survey CAS RN 69430-24-6 (cyclomethicone, the mixture) or PDMS. Even though there is evidence that some companies did report individual cVMS in the mixture under the survey, the quantities of these substances imported into Canada and their uses in 2006 are not completely known, and their releases into the Canadian environment are not considered fully in this screening assessment report.

Several factors contribute to the uncertainty of the assessment of toxicity in the aquatic compartment. These include a lack of toxicity studies using fish of comparable size as those for which toxicity was demonstrated with the similar cVMS, D4; a lack of toxicity studies with fish early-life stages; a lack of testing with exposures somewhat above the solubility limit; and concerns that the exposure duration was insufficient for the achievement of steady-state tissue concentrations. For the risk distribution analysis, the NOEC was derived from a chronic *Daphnia magna* toxicity study of D5, a concentration that is very close to its water solubility limit (~ 0.017 mg/L). This NOEC was at the highest concentration tested (unbounded), and thus no application factor was applied to extrapolate from laboratory data to the field due to a lack of observed significant adverse effects at this concentration. Since there is likely variability in water solubility under realistic environmental conditions, and it is possible that D5 attached to suspended organic matter coming from STPs (which potentially contain higher concentration of D5 than its water solubility (see Table 9b)) may be ingested by aquatic organisms, it is possible that chronic adverse effects may occur. Since no release information from industrial operations was available for the risk distribution analysis, it is assumed that releases to wastewater were uniformly distributed among 64 industrial sites evaluated. In reality, certain industrial sites may use higher quantities of D5 than others, resulting in higher releases to the municipal discharging sites associated with these industrial sites and therefore a higher risk than predicted. The distribution concentrations in the analysis applied instantaneous dilution of the effluent from sewage treatment plants (STP) into the receiving water. However, under realistic environmental conditions, instantaneous dilution may not be achieved over a certain distance from the discharge point, and the area near the discharge point of an STP may present a higher risk than predicted.

Sediment is an important media of concern for D5. The D4 sediment degradation studies were used as read-across values for D5 and those studies are not without uncertainties. Extrapolation of half-lives at low temperature in sediment based on hydrolysis data may also contribute to the overall uncertainty in sediment persistence. Limited data for bioaccumulation potential in this compartment also contributes to the overall ecological assessment uncertainty.

The available bioconcentration data and biomagnification factor and sediment accumulation values for D5 are conflicting. There is a lack of field data on bioaccumulation potential via the food web and in non-aquatic organisms.

Model predictions were also included in this screening assessment as a line of evidence. There are some uncertainties associated with predicted values, as few siloxanes or chemicals with high $\log K_{ow}$ values (> 8) have been studied and included in the models. The predicted inherent toxicity for aquatic organisms may have an additional source of uncertainty when these concentrations exceed the solubility of the chemical in water. Given that concentrations for both the toxicity and water solubility under realistic environmental conditions are often uncertain (vary from optimal laboratory conditions), toxicity values that exceed solubility estimates by up to a factor of 1000 were accepted during categorization.

Environmental monitoring data in Canada and elsewhere are limited. Sample contamination is a potential problem in environmental monitoring due to D5's widespread uses. Data on environmental concentrations of D5 in biota and surface water in Canada are lacking and few environmental concentrations have been reported outside of urban areas in Canada. Consequently, monitoring data from European countries have been presented in this report. However, monitoring has been identified as a key component in the Chemicals Management Plan in Canada and D5 is being considered for environmental monitoring under the Plan. Environmental monitoring will contribute to a better understanding of the environmental presence and "true" environmental accumulation potential of the substance in the environment.

Potential to Cause Harm to Human Health

Exposure Assessment

The data on levels of D5 found in environmental media including ambient air near and away from point sources, surface waters, sediments, sewage sludge and biota are described in this report in the section entitled “Ecological Exposure Assessment.” Unpublished data from Canada include measurements taken of biogas at landfills (personal communication, Environment Canada, Environmental Technology Centre, 2008, unreferenced); air near and away from point sources (personal communication, Environment Canada, Canadian Centre for Inland Waters, 2007, unreferenced); air, influent and effluent water at wastewater treatment plants (personal communication, Environment Canada, Canadian Centre for Inland Waters, 2007, unreferenced); and Great Lakes sediment (Powell and Kozerski 2007). Many analyses of volatile siloxanes have been confounded by sample contamination during collection and analysis, resulting in siloxanes being detected in blanks at levels comparable in some cases to those in samples taken near point sources. A survey of volatile organic chemicals, including siloxanes, in residential air was conducted from 2002 to 2004 in homes in Syracuse, New York, U.S.A. (NYIEQ 2005). Results of extensive sampling and measurement of siloxanes in environmental media in Scandinavia have been published by the Nordic Council of Ministers and the Swedish Environmental Research Institute (Norden 2005, Kaj et al. 2005). Data from these reports were considered reliable and were used to produce the upper-bounding estimates of exposure to siloxanes in air, water and soil by the general population in Canada.

The upper-bounding estimates of daily intake of D5 for six age groups in the Canadian population are shown in Appendix 1. The estimates of intake from environmental media and diet range from 68.7 $\mu\text{g}/\text{kg}\text{-body weight}/\text{day}$ ($\mu\text{g}/\text{kg}\text{-bw}/\text{day}$) for adults aged 60 years and older to 208 $\mu\text{g}/\text{kg}\text{-bw}/\text{day}$ for children aged 6 months to 4 years. The most significant contribution to daily intake from environmental media is inhalation of indoor air, based on a study of approximately 130 homes in Syracuse, New York, in which D5 was detected in 85% of homes. The maximum concentration of D5 in indoor air measured in homes in this study was 1560 $\mu\text{g}/\text{m}^3$ (mean 136 $\mu\text{g}/\text{m}^3$) (NYIEQ 2005). To characterize a more representative upper-bounding estimate, the 90th percentile was estimated, based on available data. An approximate 90th percentile concentration of 393 $\mu\text{g}/\text{m}^3$ was derived (e-mail from Health Canada, Biostatistics Division, January 2008, unreferenced) and this value was used in the intake table (Appendix 1).

Confidence in the upper-bounding estimate of exposure to D5 through environmental media and diet is moderate. No Canadian data were used, but data from studies in Scandinavia and the United States were available for ambient and indoor air, water and soil. The derivation of the 90th percentile from the indoor air study was based on available data and an assumption of normal distribution, although the available data indicate the data may not be normally distributed. The use of a regulatory limit for dimethylpolysiloxane in one quarter of dairy products and one half of processed food may overestimate the dietary contribution

to total exposure, but the estimated contribution from all food to exposure of the general population from environmental media and diet is less than one percent of the contribution from air.

Using ConsExpo 4.1, software developed to estimate exposure to consumer products, the potential systemic dose of D5 through the use of personal care products was estimated for women that use skin care products, hair care products and antiperspirants (RIVM 2006). Manufacturers of personal care products are required to notify Health Canada of the concentration, within broad ranges, of siloxanes, including D5 and polydimethylcyclsiloxanes, termed cyclomethicone in personal care products.¹ Health Canada has been notified of approximately 3000 cosmetic products that contain D5 as the sole siloxane or in mixtures with other specified siloxanes, and that department has also been notified of approximately 6000 cosmetic products that contain cyclomethicone or cyclomethicone mixtures (CNS 2007). The data on the concentration of D5 in personal care products were obtained principally from the information provided by Canadian industry (Environment Canada 2007, CNS 2007) and were supplemented by information from other sources noted in Appendix 2. Market share data were not used to determine the concentration of D5 in the dominant products in each use category. In cases where the concentration for a product category reported in response to a notice published under section 71 of CEPA 1999 was higher than concentrations reported in the CNS database, a lower concentration of D5 for that product category, consistent with the range reported to the CNS, was used in estimating the systemic dose (Environment Canada 2007, CNS 2007).

Based on experimental observations that 80–91% of D5 evaporated from skin in 24 hours (Jovanovic et al. 2008), it was assumed that 80% of a product left on the skin evaporated during use and was therefore not available for dermal absorption. A distinction was made between products that are washed off and those that are left on the body.

For the purpose of modelling the absorption of personal care products, it was decided to use the same absorption rates for exposure by inhalation and ingestion as those established for D4, consistent with a conservative approach to estimating dose. For dermal absorption, the figure of 0.17% established by Jovanovic et al. (2008) experimentally for rat skin was used. Other assumptions are noted in Appendix 2.

The results of a sample calculation for the application of body lotion are shown in Appendix 2, and a summary of the estimated systemic dose arising from the use of personal care products by women is shown in Table 1. For adult women, the upper-bounding estimate of daily systemic dose from the modelled personal care products, aggregated over inhalation, dermal and oral exposure, is 0.17 mg/kg-bw/day. In the screening assessment of octamethylcyclotetrasiloxane (D4) it was shown that the systemic dose received by women from the use of personal care products was higher than the dose received by men and this is expected to be the case for D5.

¹ *Cosmetic Regulations C.R.C.*, c. 869.

An exposure assessment for use of D5, including personal care product uses, was submitted to the Government of Canada under the Challenge Program (SEHSC 2008a). The methodology is different from that shown in Appendix 2, Table 1, as a Monte Carlo probabilistic analysis was conducted and aggregate general population exposures from all sources (including personal care products) and routes (inhalation, dermal, ingestion) were derived. The contribution of the use of personal care products to total exposure via separate exposure routes (inhalation, dermal, oral) was characterized and then summed to arrive at an aggregated exposure estimate. Independent review of the submitted probabilistic assessment showed that this assessment evaluated exposure to both user and non-user groups (see Appendix 4). The data were re-analyzed based on just user groups to allow comparison with the deterministic exposure assessment in this screening assessment. Based on user groups only, the probabilistic exposure values for adult females (most highly exposed adult group) were 10–16 times lower than the deterministic values shown in Appendix 2, Table 1. Note that due to the requirement for detailed analysis and validation of probabilistic exposure assessments, such assessments are normally outside scope for conducting the exposure component of a screening assessment.

Based on user groups only, the probabilistic exposure values for children aged 0–6 months (most highly exposed children's group) were in the range of 0.016–0.032 mg/kg bw/day (see Appendix 4). A comparison with a deterministic exposure assessment for children was not possible due to the lack of sufficient product use data required for modelling children's exposure in a deterministic exposure assessment. When the children's probabilistic exposure values are compared with the adult female deterministic exposure values, they are 5–10 times lower.

Other types of consumer products such as surface coatings, caulking and cleaners were deemed to contribute significantly less to daily exposure through daily use and were not further considered in the modelling of daily dose through consumer exposure scenarios. Both personal care products and other consumer products such as surface coatings, caulking and cleaners contribute to the concentration of D5 in indoor and ambient air and thus exposure by inhalation. The contribution of all consumer products to total exposure of non-occupationally exposed individuals is estimated via indoor air in the multi-media environmental exposure model discussed in the preceding text of this section.

Confidence in the estimate of systemic dose of D5 through the use of personal care products is low. All estimates were made by the use of models and the use pattern data were not from Canadian studies. The extent of use of D5 in personal care products and the concentration of D5 in products on the market currently may be lower than used in estimating the systemic dose reported above and in Appendix 2 (Environment Canada 2007, SEHSC 2007a). Consequently, these values are expected to be overestimates of exposure to D5 from use of personal care products.

Health Effects Assessment

Appendix 3 contains a summary of the available health effects information for decamethylcyclpentasiloxane (D5).

No international agency has classified D5 for carcinogenicity, genotoxicity or reproductive/developmental toxicity. Only one national review on the health effects of cyclsiloxanes was identified to date, that of the Danish Environmental Protection Agency (EPA). They reviewed health effects for D4 and D5 (Lassen et al. 2005).

Based on the assessment of the Danish EPA, a potential effect for repeat-dose toxicity is carcinogenicity. This was based on uterine tumours observed in a two-year rat inhalation study (Lassen et al. 2005). The US EPA, in a fact sheet on D5 in dry-cleaning applications, also noted this endpoint and indicated that they will determine whether it is appropriate to conduct a risk assessment for D5 once a mode-of-action analysis is completed (US EPA 2005). No further information on D5 was identified from the US EPA.

In a chronic toxicity and carcinogenicity study, rats were exposed to vapour concentrations of 0, 10, 40 or 160 ppm D5 for 6 h/day, 5 days/week for 24 months. In the highest dose group, there was a statistically significant increase in uterine tumours (endometrial adenocarcinomas) observed in female rats in the highest exposure group and an increased incidence of hyaline inclusions in the nasal respiratory/olfactory epithelium of both sexes. Also, a subgroup exposed to the same concentrations for 12 months with a 12-month recovery period showed an increased incidence of hyaline inclusions in the nasal respiratory/olfactory epithelium of both sexes at 40 and 160 ppm (Dow Corning 2005). Decamethylcyclpentasiloxane (D5) was not genotoxic in several *in vitro* and *in vivo* assays (see Appendix 3). The limited genotoxicity results suggest that the tumours observed in the chronic toxicity/carcinogenicity study could be due to threshold effects.

Although a thorough analysis of the mode of action of decamethylcyclpentasiloxane is beyond the scope of this screening level assessment, it is recognized that D5 may possibly act as a dopamine agonist, thus contributing to the observed tumourigenic effects in female rats (SEHSC 2008a). Although the Silicones Environmental, Health and Safety Council (SEHSC 2008a) state that this mode of action is not relevant to humans, this position has not been adopted by other regulatory agencies due to lack of a thorough mode-of-action analysis by these agencies (such as the Danish EPA assessment).

In the Danish EPA assessment, the lung was identified as the primary target organ (Lassen et al. 2005). The lowest concentration at which lung effects were observed was 450 mg/m³ (30 ppm) based on a significant increased incidence of pulmonary vascular mineralization as observed in an inhalation reproduction study (Siddiqui et al. 2007; see below). However, other respiratory tract effects were observed at lower doses. The lowest-observed-effect concentration (LOEC) for repeated inhalation exposure to D5 was 10 ppm (150 mg/m³) based on nasal cavity effects (increased incidence and severity of goblet cell proliferation in level 1 of nasal cavity in both sexes) in a 28-day rat study (Burns-Naas et al. 1998a). At

higher concentrations ($\geq 380 \text{ mg/m}^3$, $\geq 25 \text{ ppm}$) in short- and long-term rat studies, additional effects included increased liver and lung weights, reversible changes in thymus weight, further effects in the nasal cavity, lung effects, and clinical chemistry effects (Burns-Naas et al. 1998a, 1998b; TNO 1984). A LOEC of 700 mg/m^3 (46 ppm) was determined in a 3-month rat study based on increased liver weight (TNO 1984) and a LOEC of 600 mg/m^3 (40 ppm) was determined in a 2-year rat study based on an increased incidence of hyaline inclusions in the nasal respiratory/olfactory epithelium (Dow Corning 2005).

For repeated-dose oral exposures, the lowest-observed-effect level (LOEL) was 5 mg/kg-bw/day based on increased liver enzyme activities (CYP2B1/2 and EROD) in a 4-day rat study (Zhang et al. 2000). At higher doses ($\geq 20 \text{ mg/kg-bw/day}$) in short-term oral rat studies, other effects included increased liver weights (Zhang et al. 2000, Jager and Hartmann 1991).

Liver effects were observed in both oral and inhalation studies using D5. For example, there was a significant increase in liver weight in a 3-month oral rat study (Jager and Hartmann 1991) and increased liver weight in a 3-month inhalation rat study (LOEC determined to be 700 mg/m^3 ; Burns-Naas 1998b), thus indicating a common effect based on both oral and inhalation exposures of the same duration. Although the Danish EPA (Lassen et al. 2005) did not identify liver effects as critical for D5, it did state that “D5 has an enzyme induction profile similar to that of D4” and that the liver was a target organ for D4 exposure. D4 was described as an inducer of hepatocellular enzymes, and thus based on similarity of the liver enzyme induction profile for D4 and D5, it is considered that the liver is a target organ for D5 oral and inhalation exposures. Zhang (2000) noted an increased induction of the liver enzyme, CYP3A1/2, by D5 that was greater than the induction caused by phenobarbital in a 4-day oral rat study, and concluded that, although similar to a phenobarbital type of induction caused by the CYP2B enzyme series, there may be important mechanistic differences in the induction caused by D5. However, for D4, Falany and Li (2005) also noted an increased induction of liver enzyme, CYP3A1/2, in 8-day rat studies but suggested that this was part of the phenobarbital-type induction due to related induction of the CYP2B and PROD enzymes. Consequently, it was not considered appropriate to determine adverse effect levels based on enzyme induction alone.

In a 2-generation study, Sprague-Dawley rats were exposed by whole-body vapour inhalation to 0, 450, 1050 or 2400 mg/m^3 (0, 30, 70, or 160 ppm) D5 for 6 h/day. Reproductive (number of days between pairing and mating, mating and fertility indices, gestation length, and parturition) and spermatogenic parameters, ovarian primordial follicle counts and number of corpora lutea in the F0 and F1 parental animals in all exposure groups were not significantly different from the control. The total number of F1 and F2 pups born, mean live litter size, sex ratio of litters, pup body weight and postnatal pup survival were not affected by exposure to D5. However, a significant increase in the incidence of pulmonary vascular mineralization was observed in all F0 and F1 animals at 450 mg/m^3 (30 ppm) and above. Also, significantly increased incidences of minimal alveolar histiocytosis were observed at the high concentration in F0 and F1 females (Siddiqui et al. 2007).

Estrogenic, androgenic and progestagenic activities were evaluated in several *in vitro* and *in vivo* bioassays. *In vivo* rat studies (16 hours/day for 3 days inhalation exposure to 160 ppm D5) showed no increase in uterine wet or blotted weights and no increase in male reproductive organ weights. In addition, D5 did not bind to human estrogen receptors α and β or progesterone receptors and was negative in ER α and PR β reporter gene assays (Quinn et al. 2007).

It is uncertain whether liver weight increases due to treatment with D5 are adaptive or adverse. As per a US EPA Health Effects Division guidance document (2002), observations of increased liver weight or hepatocellular hypertrophy should be associated with significantly increased or decreased serum levels of at least two of the liver enzymes—alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase or gamma-glutamyl transferase—before the changes are ascribed to liver toxicity. In the 1-month rat inhalation study, at 2400 mg/m³ (160 ppm), relative liver weight was significantly increased in both sexes and serum alkaline phosphatase was decreased in females, even after a 14-day recovery period (as well as increased lung weight and focal alveolar macrophage accumulation in the lung in both sexes) (Burns-Naas et al. 1998a). As observed in the 3-month rat inhalation study, there were significant increases in liver/brain weight ratios and in gamma glutamyl transferase (γ -GT) in female rats at 700 mg/m³ (46 ppm) and higher (Burns-Naas et al. 1998b). And as observed in a one-year rat inhalation study, absolute and relative liver (liver/body and liver/brain) weights and gamma glutamyl transferase (γ -GT) were significantly increased in females at 2400 mg/m³ (160 ppm) (Dow Corning 2005).

However, data on changes in levels of these liver enzymes following oral administration were not located. As shown in Appendix 3, liver weights were increased at 100, 330 and 1000 mg/kg-bw/day of D5 in a 3-month oral rat study (Jager and Hartmann 1991). Although no other effects were observed, 100 mg/kg-bw/day is considered to be the critical effect level for D5 based on the critical oral LOELs chosen for the similar compounds, D4 and D6. For D4, an oral LOEL of 100 mg/kg-bw/day was determined based on decreased serum estradiol in the 7-day mouse studies and decreased body weights and relative liver weights in fetuses in 8-day rat studies (D4 administered to pregnant females). For D6, an oral LOEL of 100 mg/kg-bw/day was determined based on increased liver weights and periportal lipidosis in the liver of females and follicular cell hypertrophy of the thyroid in both sexes of rats in a 28-day oral study.

Toxicokinetic studies in rats indicated that 0.17% of topically applied ¹⁴C-D5 was absorbed across the skin, approximately 30% of the absorbed D5 reached the systemic compartment (Jovanovic et al. 2008), and it was metabolized and eliminated in urine (Varaparth et al. 2003). In a 6-hr nose-only inhalation study in rats, 1-2% of ¹⁴C-D5 was retained in the body, 69-80% of the absorbed D5 was deposited in tissues, and the primary route of elimination was expired air (Tobin et al. 2008)

The confidence in the toxicity database is low to moderate as there was information to address effects that may be of concern and identify critical endpoints based on inhalation exposures, as well as some relevant supporting information. However, there was a lack of

dermal and/or oral studies for several endpoints (subchronic, chronic toxicity/carcinogenicity, reproductive and developmental studies).

Characterization of Risk to Human Health

Based principally on the weight-of-evidence assessment of the Danish EPA, a potential effect for repeated-dose toxicity is carcinogenicity, as observed in a 2-year rat study (Lassen et al. 2005). As stated above, the lack of genotoxic effects for D5 based on limited genotoxicity data suggests that the uterine tumours observed in the chronic toxicity/carcinogenicity study could be due to threshold effects. It is also noted that these tumours were observed at higher exposure levels than the effects identified for the lung and liver as described below.

The Danish EPA also identified the lung as a target organ for D5 exposures. Thus, the critical target organ is considered to be the lung and the critical effect level for repeated-dose toxicity is considered to be 450 mg/m³ (30 ppm) via the inhalation route based on a significant increased incidence of pulmonary vascular mineralization as observed in both generations in the rat reproduction study (Siddiqui et al. 2007). Although a lower effect level was determined in another repeated-dose study (150 mg/m³ in a 28-day rat study), the effect observed (increased incidence and severity of goblet cell proliferation in level 1 of the nasal cavity) was not considered to be critical because the 2-year rat inhalation study showed no effects at this same lower concentration, but at the next higher concentration (600 mg/m³), effects were observed in the nasal cavity (and the nasal cavity was not identified as a target organ by the Danish EPA).

Comparison of the critical effect level for repeated-dose effects via inhalation (450 mg/m³) and the upper-bounding exposure estimate (90th percentile from indoor air survey) via inhalation for decamethylcyclopentasiloxane (393 µg/m³) results in a margin of exposure of approximately 1150. Use of the 90th percentile rather than the maximum concentration from the indoor air survey (1560 µg/m³) to derive the margin of exposure was considered an appropriate refinement to the risk characterization as use of the 90th percentile is considered to result in upper-bounding exposure estimates. Thus, the margin of exposure for repeated-dose effects via inhalation and exposure via environmental air concentrations to the general population is adequate to account for uncertainties in the databases on exposure and effects. This margin of exposure also provides protection against potential carcinogenic effects as the endometrial tumours in the chronic/carcinogenicity study were observed at a much higher dose, 2400 mg/m³.

The Danish EPA assessment did not distinguish between exposure routes. Thus, it is considered prudent to establish a critical effect level for oral exposure, as a limited amount of oral toxicity data was available. The critical effect level for repeated-dose toxicity is considered to be 100 mg/kg-bw/day via the oral route. This is based on increased liver weights in a 90-day rat study, as well as the extrapolation of critical effects and levels from oral data on the similar compounds D4 and D6 to D5 (see the “Health Effects Assessment” section).

Due to similar effects in the liver via the oral and inhalation routes, it is considered appropriate to compare oral critical effects with exposure intake estimates which aggregate the three main routes of exposure (oral, inhalation, dermal). Comparison of the critical effect level for repeated dosing via the oral route (100 mg/kg-bw/day) and the upper-bounding estimate of daily intake of D5 by the general population in Canada results in a margin of exposure of approximately 17 200. This is based on adjusting the inhalation contribution to daily intake by an inhalation absorption value of 2%, resulting in a systemic exposure of 5.8 µg/kg-bw/day. This margin of exposure is considered adequate to account for uncertainties in the database on exposure and effects.

The systemic dose of 0.16 mg/kg-bw/day from use of personal care products incorporates absorption factors for the dermal, inhalation and oral routes of exposure (Table 1, Appendix 2). To calculate the equivalent oral dose, this systemic dose of 0.16 mg/kg-bw/day was corrected by applying the reciprocal of the oral factor, resulting in an equivalent systemic dose of 0.21 mg/kg-bw/day. Using this calculated upper-bounding estimate of 0.21 mg/kg-bw/day, a comparison with the critical effect level for repeated dosing via the oral route (100 mg/kg-bw/day) resulted in a margin of exposure of approximately 480 from use of personal care products. However, it is considered that the exposure estimates presented above are overestimates of actual exposure based on an independent review of the submitted probabilistic assessment and information indicating that the percentage of personal care products containing D5 on the Canadian market may be lower than assumed in deriving exposure estimates. Based on values derived from the independent review of the probabilistic exposure assessment, it appears that the margin of exposure from use of personal care products would be at least 10 times higher for adults and at least 5 times higher for children from that shown above (i.e. > 2000). On the basis of the above considerations, including consideration for the extent of its database, D5 is considered not to meet the criteria under paragraph 64(c) of CEPA 1999.

Uncertainties in Evaluation of Risk to Human Health

The scope of this screening assessment does not take into consideration a full analysis of the mechanism of action of decamethylcyclopentasiloxane and it does not take into account possible differences between humans and experimental species in sensitivity to effects induced by this substance. There is uncertainty surrounding the mechanism of carcinogenicity following exposure via the inhalation route. There is also uncertainty as to the mechanism of action resulting in liver effects following exposure via the inhalation or oral routes.

Potential adverse effects of D5 via the oral route were based on using D4 and D6 as analogues and there is uncertainty on the boundaries/limits for using D4 and D6 to extrapolate effects for D5.

Although physiologically based pharmacokinetic modelling of dermal absorption data have been published (Reddy et al. 2007), only experimental data on absorption were used in this assessment.

There is uncertainty regarding the estimation of exposure and systemic dose because of the use of modelling and a lack of Canadian data. There is uncertainty associated with the use of models and the choice of variables related to the use of consumer products including quantity and frequency of use, absorbed fraction and environmental parameters.

The cumulative exposures of the cyclosiloxanes in polydimethylsiloxanes (PDMS) are not considered in this assessment. However, D4 and D6 are considered in separate assessments.

Conclusion

Based on the information presented in this screening assessment on the potential of D5 to cause ecological harm, it is concluded that decamethylcyclopentasiloxane is entering or may be entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity.

Based on the available information on its potential to cause harm to human health, it is concluded that decamethylcyclopentasiloxane is not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

It is therefore concluded that decamethylcyclopentasiloxane meets the definition of toxic as set out in paragraph 64a of CEPA 1999. It is concluded that D5 meets the criteria for persistence as set out in the *Persistence and Bioaccumulation Regulations*. However, it is not possible to conclude that D5 meets the criterion for bioaccumulation, considering the conflicting evidence presented in this screening assessment report.

The conclusion in this screening assessment is based on the available information at this time and acknowledges that there are uncertainties associated with this assessment. Research on cVMS is currently being conducted to help address these uncertainties, but some of this research has not been completed at this time. In the context of the Challenge program, any new information provided after the final screening assessment may be considered during the risk management phase.

Monitoring has also been identified as a key component in the Chemicals Management Plan in Canada and D5 is being considered for environmental monitoring under the Plan. Field level data will contribute to a better understanding of the distribution of D5 in the environment and its bioaccumulation potential in relevant food webs.

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Appendices

Appendix 1

Table 1. Upper-bounding estimates of daily intake of D5 by the general population in Canada

Route of exposure	Estimated intake ($\mu\text{g}/\text{kg}\text{-bw}/\text{day}$) of D5 by various age groups							
	0–6 months ¹			0.5–4 years ⁵	5–11 years ⁶	12–19 years ⁷	20–59 years ⁸	60+ years ⁹
	Breast fed ²	Formula fed ³	Fed solid food ⁴					
Ambient air ¹⁰	0.09			0.19	0.15	0.08	0.07	0.07
Indoor air ¹¹	96.3			206.3	161	91.5	78.6	68.3
Drinking water ¹²	0.45	.01	.004	.004	.003	.002	.002	.002
Food and beverages ¹³			2.97	1.62	0.92	0.50	0.32	0.29
Soil ¹⁴	<.001			<.001	<.001	<.001	<.001	<.001
Total intake	96.9	96.4	99.3	208	162	92.0	79.0	68.7

- ¹ Assumed to weigh 7.5 kg, to breathe 2.1 m³ of air per day, to drink 0.8 L of water per day (formula fed) or 0.3 L/day (fed solid food) and to ingest 30 mg of soil per day (Health Canada 1998).
- ² The highest concentration of D5 detected in human breast milk was 4.5 $\mu\text{g}/\text{L}$ in Sweden (Kaj et al. 2005). Breast-fed children 0–6 months of age are assumed to have an intake rate of 0.75 kg of breast milk per day (Health Canada 1998).
- ³ For exclusively formula-fed infants, intake of water is only that required to reconstitute formula. No data on detectable concentrations of D5 in drinking water were located. No data on concentrations of D5 in formula or baby food were identified for Canada. Approximately 50% of infants are introduced to solid foods by 4 months of age and 90% by 6 months of age (NHW 1990).
- ⁴ The dietary intake is based on consumption of 0.3 litres of water and up to 1.18 kg of food daily. This intake pattern is presented as a hypothetical extreme case and does not reflect recommended infant feeding practice.
- ⁵ Assumed to weigh 15.5 kg, to breathe 9.3 m³ of air per day, to drink 0.7 L of water per day and to ingest 100 mg of soil per day (Health Canada 1998).
- ⁶ Assumed to weigh 31.0 kg, to breathe 14.5 m³ of air per day, to drink 1.1 L of water per day and to ingest 65 mg of soil per day (Health Canada 1998).
- ⁷ Assumed to weigh 59.4 kg, to breathe 15.8 m³ of air per day, to drink 1.2 L of water per day and to ingest 30 mg of soil per day (Health Canada 1998).
- ⁸ Assumed to weigh 70.9 kg, to breathe 16.2 m³ of air per day, to drink 1.5 L of water per day and to ingest 30 mg of soil per day (Health Canada 1998).
- ⁹ Assumed to weigh 72.0 kg, to breathe 14.3 m³ of air per day, to drink 1.6 L of water per

- day and to ingest 30 mg of soil per day (Health Canada 1998).
- ¹⁰ D5 has been measured in ambient air near point sources in Canada, the United States, Europe and Asia. The highest measured concentration not near a point source, 2.5 µg/m³ in Manglerud, Norway, was used for the level of D5 in ambient air (Norden 2005). Canadians are assumed to spend 3 h per day outside (Health Canada 1998).
 - ¹¹ D5 was detected in 85% of 130 air samples taken in homes in Syracuse, New York. The maximum value of D5 in indoor air was 1560 µg/m³ (NYIEQ 2005). The 90th percentile of the air concentrations (393 µg/m³) for D5 in this indoor air survey is considered appropriate to derive upper-bounding exposure estimates. The data set considered includes a survey of bedrooms in homes in Sweden in which D5 was detected in 63% of samples and at a maximum concentration of 79.4 µg/m³ (Kaj et al. 2005). Canadians are assumed to spend 21 h per day inside (Health Canada 1998).
 - ¹² No data on levels of D5 in Canadian drinking water were identified. D5 was not detected in two samples of surface water away from point sources in Norway. The higher limit of detection of 0.09 µg/L was used (Norden 2005).
 - ¹³ No data were identified for the concentration of D5 in foods in Canada. The concentration of D5 in flounder filets from Norwegian waters was reported to be 3.4 ng/g on a wet-weight basis (NILU 2007). A value of 0.1 ppm D5 was used for dairy and two groups of processed foods. The maximum concentration of DMPS (CAS RN 9006-65-9, dimethylpolysiloxane) in certain processed food is limited by regulation to 10 ppm*. A value of 1% D5 in DMPS was assumed and it was further assumed that one quarter of dairy and one half of processed food had been treated with antifoaming agents containing D5. The amounts of foods consumed on a daily basis by each age group are described by Health Canada (1998). The probable daily intake of D5 from food packaging for an adult was estimated to be 0.01 µg/kg-bw/day (as per email from Food Packaging and Incidental Additives Section, Health Products and Food Branch of Health Canada, dated Feb. 27, 2008, unreferenced).
 - ¹⁴ No Canadian data were available for D5 levels in soil. No D5 was detected in two soil samples from the Faroe Islands taken at an abandoned and an operating landfill. The higher limit of detection of 5 µg/kg was used (Norden 2005).

* Food and Drug Regulations, Division 16. C.R.C., c. 870.

Appendix 2

Consumer Exposure Modelling

Sample ConsExpo 4.1 Report

Product

D5 hair spray – women – partitioning 80/20

Compound

Compound name	Decamethylcyclopentasiloxane	
CAS RN	541-02-6	
molecular weight	371	g/mol
vapour pressure	33.2	Pascal
K _{ow}	8.03	10Log

General exposure data

exposure frequency	1.2	1/day
body weight	69	kilogram

Inhalation model: exposure to vapour – constant rate

weight fraction compound	0.3	fraction
exposure duration	8	hour
room volume	80	m ³
ventilation rate	1	1/hr
applied amount	5.4	gram
release duration	8	hour

Uptake model: fraction

uptake fraction	0.02	fraction
inhalation rate	22	m ³ /day

Dermal model: direct dermal contact with product – instant application

weight fraction compound	0.3	fraction
exposed area	1.44E3	cm ²
applied amount	1.4	gram

Uptake model: fraction

uptake fraction	0.0017	fraction
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Output

Inhalation (point estimates)

inhalation mean event concentration	2.21	mg/m ³
inhalation mean concentration on day of exposure	0.885	mg/m ³
inhalation air concentration year average	0.885	mg/m ³ /day
inhalation acute (internal) dose	0.0047	mg/kg
inhalation chronic (internal) dose	0.00564	mg/kg/day

Dermal: point estimates

dermal load	0.292	mg/cm ²
dermal external dose	6.09	mg/kg
dermal acute (internal) dose	0.0103	mg/kg
dermal chronic (internal) dose	0.0124	mg/kg/day

Integrated (point estimates)

total external dose	6.32	mg/kg
total acute dose (internal)	0.0151	mg/kg
total chronic dose (internal)	0.0181	mg/kg/day

Table 1	D5		Systemic Dose by Exposure to Personal Care Products – 69-kg Woman								
Product	Amount per application grams	Authority	Frequency per day	Authority	Weight fraction VMS	Authority	Retention factor	Inhalation mg/kg-bw/day at 2.0% abs	Dermal mg/kg-bw/day at 0.17% abs	Oral mg/kg-bw/day at 52% abs	Total mg/kg-bw/day
antiperspirant solid	0.8	RIVM	1	Maxim	0.58	NMI	1	0.00113	0.00229		
body lotion	8	RIVM	1.5	RIVM	0.63	Section 71	1	0.00184	0.0373		
sunscreen	6.1	RIVM	0.2	RIVM	0.347	NMI	1	0.00003	0.00205		
face moisturizer	2.5	RIVM	2	RIVM	0.63	Section 71	1	0.0204	0.0155		
face makeup	0.8	RIVM	1	RIVM	0.63	Section 71	1	0.00123	0.00248		
lipstick	0.01	Maxim	4	RIVM	0.25	Maxim	1	0	0.00005	0.0603	
hair spray	6.8	RIVM	1.2	RIVM	0.3	CNS	0.1	0.00564	0.00124		
hair shampoo	20	RIVM	0.7	RIVM	0.03	CNS	0.01	0	0.00002		
hair conditioner	20	RIVM	0.3	RIVM	0.63	Section 71	0.01	0	0.000186		
hair styling	4.7	Maxim	0.6	Maxim	0.63	Section 71	0.1	0.00437	0.00084		
Totals								0.035	0.062	0.0603	0.16

Consumer exposure modelling based on ConsExpo (RIVM 2006).

Basic assumptions:

Body weight 69 kg

Absorption by inhalation 2.0% (Tobin et al. 2008); dermal absorption 0.17% (Jovanovic et al. 2008); absorption by ingestion 52% (Dow Corning 1998).

For products left on skin except lipstick, 20% of applied amount is available to be dermally absorbed, 80% evaporates.

For lipstick, 20% is available for dermal absorption and 80% is available for absorption by ingestion.

Authorities:

- NMI: see in references NMI 2007
- RIVM: see in references RIVM 2006
- CNS: see in references CNS 2007
- Section 71: see in references Environment Canada 2007
- Maxim: see in references Maxim 1998

Appendix 3

Summary of Health Effects Information for Decamethylcyclopentasiloxane (D5)

Endpoint	Lowest effect levels ¹ /Results
Acute toxicity	<p>Lowest oral LD₅₀: > 4800 mg/kg-bw in Wistar rats (Löser 1984).</p> <p>Lowest inhalation LC₅₀: 8670 mg/m³ (560 ppm) in Fischer 344 (F344) rats for single 4-hour whole-body inhalation exposure (Dow Corning 1994).</p> <p>Other studies: Pauluhn (1984). No dermal studies identified.</p>
Short-term repeated-dose toxicity	<p>Lowest-observed-effect level (LOEL) – oral: 5 mg/kg based on significant increases in liver enzyme activities of CYP2B1/2 and EROD in male and female Sprague-Dawley (SD) rats treated by gavage with 1, 5, 20 or 100 mg/kg-bw/day D5 in corn oil daily for 4 days. At 20 mg/kg-bw/day and higher, significant increases in relative liver weight (33% increase at 100 mg/kg-bw/day) was observed in female rats (Zhang et al. 2000).</p> <p>Other oral studies: Dow Corning 1990a, 1990b.</p> <p>Lowest-observed-effect concentration (LOEC) – inhalation 150 mg/m³ (10 ppm) based on increased incidence and severity of goblet cell proliferation in level 1 of the nasal cavity in both sexes. Male and female Fischer 344 rats were exposed by whole-body inhalation to 0, 150, 380, 1100 or 2400 mg/m³ (0, 10, 25, 75 or 160 ppm) D5 for 6 h/day for 28 days. At 380 mg/m³ (25 ppm), relative spleen weight was increased in both sexes only after the 14-day recovery period. At 1100 mg/m³ (75 ppm), incidence and severity of submucosal inflammation was increased in the nasal cavity in male rats, and at 2400 mg/m³ (160 ppm), there was increased lung weight and focal alveolar macrophage accumulation in the lung in both sexes, relative liver weight was significantly increased in both sexes (13% in females, 5% in males), relative thymus weight was increased in males (14%) and serum alkaline phosphatase was decreased in females (even after a 14-day recovery period) (Burns-Naas et al. 1998a).</p> <p>Other inhalation studies: RCC Group 1995, TNO 1984.</p> <p>Highest no-observed-adverse-effect level (NOAEL) – dermal: 1600 mg/kg-bw/day based on no systemic adverse effects in SD rats exposed dermally under occlusive conditions for 6 h/day, 7 days/wk for 28 days (no LOEL at 1600 mg/kg-bw/day). Evidence of dermal absorption and metabolism based on urinalysis (Dow Corning 1990c).</p> <p>Other dermal studies: Huntingdon Research Center 1979, Krötlinger 1988.</p>

Endpoint	Lowest effect levels ¹ /Results
Subchronic toxicity	<p>Lowest oral LOEL: 100 mg/kg-bw/day for significant increases in liver weight (percent increase not stated) in both sexes of Wistar rats, administered 100, 330 or 1000 mg/kg-bw/day for 13 weeks by oral gavage (Jager and Hartmann 1991). No other oral studies identified.</p> <p>Lowest inhalation LOEC: 700 mg/m³ (46 ppm) based on a dose-related increase in absolute and relative lung weights in both sexes (still increased in females after a 1-month recovery period), significant increases in liver weight / brain weight ratios in females (13, 9 and 16% increases at 700, 1300 and 3700 mg/m³, respectively) and in gamma glutamyl transferase (γ-GT) in female rats (Fischer 344 rats exposed to D5 6 hours/day 5 days/week for 3 months (nose-only exposure) at concentrations of 0, 400, 700, 1300 or 3700 mg/m³ (0, 26, 46, 86 or 244 ppm)). At 1300 mg/m³ (86 ppm), serum lactate dehydrogenase was decreased in females (even after a 1-month recovery period). At 3700 mg/m³ (224 ppm), there was increased focal macrophage accumulation and interstitial inflammation in the lungs of both sexes (Burns-Naas et al. 1998b). Other inhalation studies: Dow Corning 1990d.</p> <p>No dermal studies identified.</p>
Chronic toxicity / Carcinogenicity	<p>Groups of Fischer 344 rats were exposed to vapour concentrations of 0, 150, 600 or 2400 mg/m³ (0, 10, 40 or 160 ppm) D5 for 6 hours/day, 5 days/week for 6 (6 males and 6 females per group), 12 (10 males and 10 females per group), 12 plus 12 months recovery (20 males and 20 females per group) and 24 months (60 male and 60 females per group). Female rats exposed to the highest concentration of D5 exhibited a significant increase of uterine tumours, specifically endometrial adenocarcinomas (Dow Corning 2005, US EPA 2005, Lassen et al. 2005).</p> <p>Lowest inhalation non-neoplastic LOEC: 600 mg/m³ (40 ppm) for increased incidence of hyaline inclusions in the nasal respiratory/olfactory epithelium of both sexes (Dow Corning 2005).</p> <p>In the 6-month subgroup, there was an increased incidence of hyaline inclusions in the nasal respiratory/olfactory epithelium of both sexes, relative liver weights were increased in females, serum calcium concentration was increased in both sexes and mean corpuscular volume (MCV) was increased in females at 2400 mg/m³ (160 ppm). In the 12-month subgroup, urea concentration was decreased and cholesterol concentration was increased in females at 600 and 2400 mg/m³; and at 2400 mg/m³, absolute liver weight, liver weight / body weight ratio and liver weight / brain weight ratio in females were increased (11, 10 and 10%, respectively), serum gamma glutamyl-transferase (γ-GT), triglycerides, calcium and MCV were increased in females, and aspartate aminotransferase (AST) was decreased in males. In the 12-month exposure plus 12-month recovery subgroup, there was an increased incidence of hyaline inclusions in the nasal respiratory/olfactory epithelium of both sexes at 600 and 2400 mg/m³, the brain weight / body weight ratio in females was decreased (8%) at 2400 mg/m³, and the combined incidence of endometrial adenomatous polyps and adenocarcinomas was 1, 1, 0 and 3, in the 0, 150, 600 and 2400 mg/m³ groups respectively, but the trend or difference was not statistically significant. In the 24-month group, there was an increased incidence of hyaline inclusions in the nasal respiratory/olfactory epithelium of both sexes and the liver weight / brain weight ratio in males was increased (9%) at 2400 mg/m³ (160 ppm). In this same subgroup the incidence of endometrial adenocarcinomas was 0, 1, 0 and 5 in the 0, 150, 600 and 2400 mg/m³ groups respectively, which was statistically significant, and the incidence of endometrial adenomatous polyps was 1, 0, 1 and 0, respectively, which was not statistically significant.</p> <p>No oral or dermal studies identified.</p>

Endpoint	Lowest effect levels ¹ /Results
Developmental toxicity	No developmental toxicity studies were identified.
Reproductive toxicity	<p>Inhalation NOAEC for reproductive toxicity: 2400 mg/m³ (160 ppm) based on a 2-generation study in which SD rats were exposed by whole-body inhalation to 0, 450, 1050 or 2400 mg/m³ (0, 30, 70, or 160 ppm) D5 for 6 h/day from 70 days prior to mating, throughout mating and to gestation day 20, and again on lactation days 5 to sacrifice of F1 animals (Siddiqui et al. 2007).</p> <p>Inhalation LOEC for systemic toxicity: 450 mg/m³ (30 ppm) based on a significant increased incidence of pulmonary vascular mineralization in all F0 and F1 animals (Siddiqui et al. 2007).</p> <p>Other inhalation studies: WIL Research Laboratories 1996.</p> <p>Negative in estrogenic activity: D5 showed no increase in uterine wet or blotted weights or in uterine epithelial cell height following 16 hours/day inhalation exposure for 3 days in SD and F344 rats at 2400 mg/m³ (160 ppm) (Quinn et al. 2007).</p> <p>Negative in androgenic activity: In a Hershberger assay, D5 did not result in any increase in reproductive organ weights following 16 hours/day inhalation exposure for 10 days in male F344 rats at 2400 mg/m³ (160 ppm) (Quinn et al. 2007).</p>
Endocrine disruption <i>in vitro</i>	D5 did not bind to human estrogen receptor α (ER α) or estrogen receptor β (ER β), and was negative in ER α reporter gene assay in a human epithelial cell line MCF-7. D5 showed no binding affinity to progesterone receptors (PRs) and was negative in PR β reporter gene assay (Quinn et al. 2007).
Genotoxicity and related endpoints: <i>in vivo</i>	<p>DNA damage:</p> <p>Negative: No significant increase of unscheduled DNA synthesis (UDS) in primary hepatocytes from F344 rats exposed to D5 by whole-body inhalation to 2400 mg/m³ (160 ppm) 6 hours/day for 7 days (Dow Corning 2004a).</p> <p>Micronuclei formation:</p> <p>Negative: No significant increase of micronucleus frequency in bone marrow cells of F344 rats exposed to D5 by whole-body inhalation to 2400 mg/m³ (160 ppm) 6 hours/day for 7 days (Dow Corning 2004a).</p>
Genotoxicity and related endpoints: <i>in vitro</i>	<p>Mutagenicity:</p> <p>Negative in bacterial reverse mutation: <i>Salmonella typhimurium</i> TA98, TA100, TA1535, TA1537, TA1538 and <i>Escherichia coli</i> strains W3110/polA⁺, P3748/polA⁻ with and without activation (Isquith et al. 1988, Dow Corning 2004c, Litton Bionetics 1978).</p> <p>Negative in bacterial reverse mutation: <i>Escherichia coli</i> strain WP2 uvrA with and without activation (Dow Corning 2004c).</p> <p>Negative in gene mutation at the TK locus: mouse lymphoma L5178Y cells with and without activation (Isquith et al. 1988).</p> <p>Negative in mitotic gene conversion: <i>Saccharomyces cerevisiae</i> strain D4 (Isquith et al. 1988).</p> <p>Chromosome aberrations:</p> <p>Negative: mouse lymphoma cells; with and without activation (Isquith et al. 1988).</p> <p>Negative: Chinese hamster V79 cells; with and without activation (Dow Corning 2004b).</p> <p>Sister chromatid exchange:</p> <p>Negative: mouse lymphoma cells; with and without activation (Isquith et al. 1988).</p> <p>DNA damage:</p> <p>Negative: alkaline elution assay in mouse lymphoma cells; with and without activation (Isquith et al. 1988).</p> <p>Negative: DNA repair assay in <i>E. Coli</i> pol A^{+/+}; with and without activation (Isquith et al. 1988).</p>

- ¹ LD₅₀ = median lethal dose
- LC₅₀ = median lethal concentration
- LOEL = lowest-observed-effect level
- LOEC = lowest-observed-effect concentration
- NOAEL = no-observed-adverse-effect level
- NOAEC = no-observed-adverse-effect concentration

Appendix 4: Review of D5 Probabilistic Exposure Assessment

PROJECT	D5 Probabilistic Exposure Assessment
TASK	Review D5 Probabilistic Exposure Assessment conducted by Silicones Environmental, Health and Safety Council (SEHSC)
FOR	Health Canada (Healthy Environments and Consumer Safety Branch)
BY	infoscientific, Henderson, Nevada, USA
PERIOD	August–September, 2008

EXECUTIVE SUMMARY

A review of the D5 probabilistic exposure assessment submitted by SEHSC was done and comments to assist in preparing the screening assessment report for D5 is provided in this report.

“User Only” daily exposures were estimated based on a Monte Carlo analysis using Crystal Ball. Mean exposure and 90th percentile exposure summaries were generated for different subpopulations (children and adults).

For dermal and inhalation exposure routes, the current assessment resulted in higher exposures than the assessment done by SEHSC. The primary reason was the consideration of “user only” subpopulation in the current assessment compared to the “user” and “non-user” subpopulations considered in the SEHSC assessment. The dermal exposure route had higher exposures for both children and adults. Diaper cream, body lotion and sunscreen contributed to higher exposures in the dermal route; soothing vapour in the inhalation route and antifoam and fish in the ingestion route.

INTRODUCTION

As a part of Canada's Chemicals Management Program, Decamethylcyclopentasiloxane (D5) has been identified by Health Canada as a material to be reviewed and considered in a screening level assessment.

SEHSC submitted information on D5 to assist Health Canada in preparing the screening assessment report for D5. The information provided included toxicity information not readily available in the literature and a comprehensive exposure assessment utilizing Monte Carlo analysis. The exposure assessment included information on the levels of D5 in different environmental media and on consumer product use patterns.

Health Canada contracted with infoscientific, USA to review the D5 probabilistic exposure assessment submitted by SEHSC and to provide comments to assist in preparing the screening assessment report for D5.

DESCRIPTION

SEHSC's Monte Carlo-based probabilistic assessment for D5 included the following age-dependent and exposure-route-dependent scenarios:

- Children – dermal route: body lotion, conditioner (leave in), conditioner (rinse off), diaper cream, shampoo (2-in-1) soothing vapour, spray detangler, sunscreen
- Children – ingestion route: antifoam, baby bottle nipple, fish (general population), fish (subsistence population), human milk, leafy vegetables (greens), meat, milk, pacifier, root vegetables, sipper tube, soil, straws, water
- Children – ingestion route: OTC (over-the-counter) drugs
- Children – inhalation route: indoor air, outdoor air, soothing vapour
- Adults – dermal route: after shave, body lotion, conditioner (leave in), conditioner (rinse off), foundation, hair spray, mascara, moisturizer, nail care, roll-on antiperspirant, shampoo, solid antiperspirant, soothing vapour, sunscreen, under-eye cream
- Adults – ingestion route: antifoam, fish (general population), fish (subsistence population), leafy vegetables (greens), lipstick, meat, milk, root crops, soil, water
- Adults – ingestion route: OTC (over-the counter) drugs
- Adults – inhalation route: indoor air, outdoor air, soothing vapour

Separate route-specific and total exposure estimates were made for the following subpopulations:

- Children: ages 0 to 6 months, breastfed
- Children: ages 0 to 6 months, non-breastfed
- Children: ages 0 to 6 months, males
- Children: ages 0 to 6 months, females
- Children: ages 7 to 11 months, breastfed
- Children: ages 7 to 11 months, non-breastfed
- Children: ages 1 to 2 years, breastfed
- Children: ages 1 to 2 years, non-breastfed
- Children: ages 2 to 4 years
- Children: ages 6 months to 4 years, males
- Children: ages 6 months to 4 years, females
- Children: ages 4 to 11 years, males
- Children: ages 4 to 11 years, females

- Adults: ages 12 to 19 years, males
- Adults: ages 12 to 19 years, females
- Adults: ages 20 to 59 years, males
- Adults: ages 20 to 59 years, females
- Adults: ages 60+ years, males
- Adults: ages 60+ years, females

The following documents and data files were provided to assist with the review process:

- D5_Kids, an Excel file, compatible with Crystal Ball, that contained all the exposure calculations for children
- D5_Adults, an Excel file, compatible with Crystal Ball, that contained all the exposure calculations for adults
- UPDATED Final Submission for Health Canada – D5, a Word file that contains information related to toxicity and exposure for D5
- Attachment 1 – Exposure Assessment for D5, a Word file that is a report explaining the probabilistic exposure assessment, including inputs used and outputs generated

The following steps were taken during the process of reviewing the D5 probabilistic assessment submitted by SEHSC:

- reviewed documents provided by Health Canada
- identified product-based exposure scenarios, exposure pathways and exposure subpopulations
- used the Excel files provided by Health Canada (D5_Kids.xls and D5_Adults.xls; files created by SEHSC) as starting points
- assured the quality of Crystal Ball-based probabilistic calculations
- generated Crystal Ball-based probabilistic Monte Carlo outputs and compared them with those listed in documents submitted by SEHSC
- commented on the robustness of industry's (SEHSC) probabilistic assessment and recommended whether it should be considered further in the screening assessment for D5

It must be mentioned that the review process did not

- validate the list of scenarios that cover all D5 exposures to children and adults
- validate the input values used in the SEHSC assessment
- validate the sources of the input values

However, the review process did

- check cells designated as Crystal Ball Assumptions (check the assignment of distributional parameters for inputs)
- check cells designated as Crystal Ball Forecasts (check the assignment of results)
- check formulas for the different calculations

A few errors were detected in the calculations. All these errors, which appeared in the formula cells, were incorrect references to formula inputs (incorrect cell references were provided).

Each exposure scenario—dermal, ingestion or inhalation—has two use-related parameters associated with it: frequency of use/occurrence (number of times per day) and percentage of population engaged in scenario. For the parameter “frequency of use/occurrence,” the values used in the SEHSC assessment were also used in the current assessment, except when the value was less than 1.0; in this case, a value of 1.0 was assigned.

The SEHSC assessment relies heavily on the parameter “percentage of population engaged in scenario” to estimate exposures for the general population, which includes users and non-users. The current assessment ignores this parameter completely. For example, in the case of a scenario where 20% of the population is engaged, in a probabilistic Crystal Ball run with 200 000 simulations, the SEHSC assessment will have 160 000 estimates with zero values and 40 000 estimates with values greater than zero. On the other hand, the current assessment will have 200 000 estimates with values greater than zero. As a result, both mean exposure estimates and 90th percentile exposure estimates generated by the current assessment will be greater than those generated by the SEHSC assessment. Conclusion: “user-only” exposures make a significant difference when comparing results generated by SEHSC and by infoscientific.

Exposure summary results were generated for 1) individual scenarios by specific exposure routes, 2) multiple scenarios by specific exposure routes (total exposure by specific exposure route) and 3) multiple scenarios aggregated over multiple routes (total exposure).

Total exposure within an exposure route is estimated by summing exposures for each scenario. Then total exposure across multiple exposure routes is estimated by summing exposures for each exposure route. Let us consider single Monte Carlo simulations within two separate probabilistic assessments: (1) a “user only” assessment (similar to the current assessment) and (2) a “user/non-user” assessment (similar to the SEHSC assessment).

In case (1), for each scenario, there is a finite probability that the individual represented in the simulation engages in that scenario. Thus, for multiple scenarios, the individual is involved only in a fraction of the scenarios and not all the scenarios considered. For those scenarios in which the individual engages, exposure estimates are generated. Total exposure is the sum of individual scenario exposures. This case can be extended to represent individuals in a general population.

By contrast, in case (2), for all scenarios, the probabilities for the individual represented in the simulation engaging in each equal 1.0 (100%). And, in this case, for multiple scenarios, the individual is involved in all the scenarios. Total exposure, which is the sum of individual scenario exposures, represents all the scenarios. The probability of an individual in a general population engaging in all the scenarios is unlikely.

Based on the above explanations for the two cases, in the current assessment, the “user only” summaries generated for individual scenarios are valid results. However, the summaries generated for total exposures, either within individual exposure routes or across exposure routes, are improbable and should be interpreted with caution. For total exposures, the estimates generated by SEHSC would be more applicable than the ones generated by the current assessment.

Adding exposures across exposure routes should be done after consideration of route-specific toxicological endpoints. If the route-specific toxicological endpoints are unequal, route-specific total exposures cannot be added without applying appropriate absorption/penetration factors and/or potency factors.

EXPOSURE RESULTS AND DISCUSSION: CHILDREN

Table 1. Children's mean exposures (based on 200 000 Crystal Ball simulations)

Mean exposures		Ages 0–6 months		6 m – 4 yrs		4 yrs – 11 yrs	
		M	F	M	F	M	F
DERMAL	2-in-1 shampoo	3.636E-07	2.178E-07	1.760E-07	1.038E-07	2.358E-07	1.417E-07
	Body lotion					3.625E-03	3.517E-03
	Conditioner leave in					2.498E-04	1.492E-04
	Conditioner rinse off					7.513E-06	4.497E-06
	Soothing vapour			1.447E-05	1.495E-05	6.662E-06	6.464E-06
	Diaper cream	5.665E-03	5.944E-03	2.742E-03	2.832E-03		
	Spray detangler			1.654E-07	9.776E-08	7.617E-08	4.228E-08
	Sunscreen	2.409E-03	2.529E-03	2.915E-03	3.011E-03	2.684E-03	2.605E-03
	Total Dermal	8.075E-03	8.473E-03	5.672E-03	5.858E-03	6.573E-03	6.282E-03
	INHALATION	Indoor air	2.403E-04	2.523E-04	2.251E-04	2.326E-04	1.515E-04
Outdoor air		4.775E-06	5.006E-06	4.472E-06	4.619E-06	3.012E-06	2.707E-06
Soothing vapour				2.921E-03	3.017E-03	1.965E-03	1.767E-03
Total Inhalation		2.451E-04	2.573E-04	3.151E-03	3.255E-03	2.119E-03	1.905E-03
INGESTION	Antifoam					9.252E-04	8.539E-04
	Baby bottle nipple						
	Fish, general					4.499E-04	3.883E-04
	Greens					2.069E-08	2.126E-08
	Human milk						
	Meat					7.121E-08	6.347E-08
	Milk					1.027E-07	8.833E-08
	Pacifier						
	Root vegetable					1.129E-05	1.051E-05
	Sipper tube					5.176E-05	5.176E-05
	Soil					2.181E-06	2.116E-06
	Straws					2.133E-05	2.069E-05
	Fish, subsistence					3.770E-04	3.657E-04
	Water					1.461E-07	1.418E-07
	OTC drugs	5.434E-03	5.701E-03	1.316E-03	1.359E-03	2.213E-06	2.146E-06
	Total Ingestion, Subsistence	4.372E-03	4.372E-03	2.794E-03	2.794E-03	1.389E-03	1.305E-03
	Total Ingestion, General	2.654E-03	2.654E-03	2.428E-03	2.428E-03	1.462E-03	1.328E-03
TOTAL	Total, Subsistence Population	1.813E-02	1.880E-02	1.293E-02	1.327E-02	1.008E-02	9.494E-03
	Total, General Population	1.641E-02	1.709E-02	1.257E-02	1.290E-02	1.016E-02	9.517E-03

Table 1 summarizes all the exposure results for children as mean values. When compared with similar results generated by the SEHSC assessment, all the values are higher in the current assessment. Compared to the SEHSC assessment, the current assessment results in a difference of 1.79 to 2.32 times for total dermal exposures, in a difference of 1.00 to 13.56 times for total inhalation exposures, and in a difference of 0.97 to 1.56 times for total ingestion (general population) exposures. The primary reason for the differences is that the SEHSC assessed users and non-users whereas the current assessment considered users only.

Wherever exposures are estimated for multiple age groups, the estimates for lower age groups are usually greater than the estimates for higher age groups. Within dermal exposure scenarios, diaper cream, sunscreen and body lotion are the highest contributions; within inhalation, soothing vapour is the highest contributor; and within ingestion, the highest contributor is over-the-counter drugs for lower age groups and fish (subsistence) for higher age groups.

Table 2. Mean ingestion exposures for breastfed (BF) and non-breastfed (nBF) infants

Mean exposures		Ages 0–6 months		7–11 months		1–2 years		2–4 years
		BF	nBF	BF	nBF	BF	nBF	all
INGESTION	Antifoam	1.060E-03	2.904E-03	1.145E-03	2.260E-03	1.259E-03	1.821E-03	1.390E-03
	Baby bottle nipple		4.871E-04		3.484E-04		2.900E-04	2.092E-04
	Fish, general	3.341E-06	3.980E-04	1.700E-04	4.794E-04	4.800E-04	4.828E-04	5.023E-04
	Greens	4.889E-08	3.968E-08	1.749E-08	3.329E-08	6.773E-08	2.030E-08	2.718E-08
	Human milk	3.425E-04		2.009E-04		1.124E-04		
	Meat	5.972E-08	4.442E-08	5.182E-08	7.268E-08	5.008E-08	9.237E-08	9.152E-08
	Milk	1.355E-07	1.343E-07	6.381E-08	1.957E-07	1.778E-07	2.985E-07	1.639E-07
	Pacifier	4.873E-04	4.873E-04	3.485E-04	3.485E-04	2.902E-04	2.902E-04	2.093E-04
	Root vegetable	1.961E-05	2.103E-05	2.126E-05	2.799E-05	1.702E-05	1.998E-05	1.571E-05
	Sipper tube	2.436E-04	2.436E-04	1.743E-04	1.743E-04	1.451E-04	1.451E-04	1.046E-04
	Soil	9.959E-06	9.959E-06	7.123E-06	7.123E-06	5.929E-06	5.929E-06	4.277E-06
	Straws	4.873E-04	9.740E-05	6.967E-05	6.967E-05	5.799E-05	5.799E-05	4.184E-05
	Fish, subsistence	1.721E-03	1.721E-03	1.231E-03	1.231E-03	1.025E-03	1.025E-03	7.393E-04
	Water	6.673E-07	6.673E-07	4.773E-07	4.773E-07	3.973E-07	3.973E-07	2.866E-07

Table 2 summarizes all ingestion-related mean exposures specific to breastfed and non-breastfed infants. There are no significant differences in the results generated by the current assessment (shown in Table 2) and the SEHSC assessment. The two highest contributors to ingestion exposure for this subpopulation are antifoam and fish (subsistence).

Table 3. Children's 90th percentile exposures (based on 200 000 Crystal Ball simulations)

90 th percentile exposures		Ages 0–6 months		6 m – 4 yrs		4 yrs – 11 yrs		
		M	F	M	F	M	F	
DERMAL	2-in-1 shampoo	7.952E-07	4.747E-07	3.847E-07	2.260E-07	5.177E-07	3.132E-07	
	Body lotion					7.894E-03	7.662E-03	
	Conditioner leave in					5.265E-04	3.203E-04	
	Conditioner rinse off					1.608E-05	9.733E-06	
	Soothing vapour			2.019E-05	2.085E-05	9.294E-06	9.019E-06	
	Diaper cream	1.463E-02	1.535E-02	7.073E-03	7.312E-03			
	Spray detangler			3.192E-07	1.882E-07	1.468E-07	8.138E-08	
	Sunscreen	7.264E-03	7.613E-03	8.766E-03	9.062E-03	8.081E-03	7.860E-03	
	Total Dermal	1.849E-02	1.942E-02	1.244E-02	1.286E-02	1.391E-02	1.338E-02	
	INHALATION	Indoor air	5.001E-04	5.262E-04	4.700E-04	4.859E-04	3.185E-04	2.853E-04
		Outdoor air	1.137E-05	1.200E-05	1.071E-05	1.100E-05	7.186E-06	6.495E-06
Soothing vapour				4.379E-03	4.522E-03	2.964E-03	2.640E-03	
Total Inhalation		5.038E-04	5.298E-04	4.663E-03	4.813E-03	3.155E-03	2.808E-03	
INGESTION	Antifoam					1.680E-03	1.567E-03	
	Baby bottle nipple							
	Fish, general					1.008E-03	8.716E-04	
	Greens					3.873E-09	4.007E-09	
	Human milk							
	Meat					1.425E-07	1.276E-07	
	Milk					1.991E-07	1.747E-07	
	Pacifier							
	Root vegetable					2.966E-05	2.903E-05	
	Sipper tube					6.587E-05	6.587E-05	
	Soil					4.180E-06	4.056E-06	
	Straws					2.715E-05	2.634E-05	
	Fish, subsistence					8.404E-04	8.146E-04	
	Water					2.901E-07	2.813E-07	
	OTC drugs	1.304E-02	1.366E-02	3.627E-03	3.746E-03	5.306E-06	5.150E-06	
	Total Ingestion, Subsistence	7.195E-03	7.195E-03	4.406E-03	4.406E-03	2.333E-03	2.203E-03	
	Total Ingestion, General	4.326E-03	4.326E-03	3.804E-03	3.804E-03	2.488E-03	2.266E-03	
TOTAL	Total, Subsistence Population	3.317E-02	3.456E-02	2.201E-02	2.260E-02	1.756E-02	1.673E-02	
	Total, General Population	3.102E-02	3.242E-02	2.155E-02	2.215E-02	1.773E-02	1.677E-02	

Table 3 summarizes all the exposure results for children as 90th percentile values. When compared with similar results generated by the SEHSC assessment, all the values are higher in the current assessment. Compared to the SEHSC assessment, the current assessment results in a difference of 1.33 to 1.69 times for total dermal exposures, in a difference of 1.00 to 9.83 times for total inhalation exposures, and in a difference of 0.97 to 1.36 times for total ingestion (general population) exposures. The primary reason for the differences is that the SEHSC assessed users and non-users whereas the current assessment considered users only.

Table 4. 90th percentile ingestion exposures for breastfed (BF) and non-breastfed (nBF) infants

90 th percentile exposures		Ages 0–6 months		7–11 months		1–2 years		2–4 years
		BF	nBF	BF	nBF	BF	nBF	all
INGESTION	Antifoam	2.706E-03	5.188E-03	2.490E-03	3.981E-03	2.423E-03	3.240E-03	2.487E-03
	Baby bottle nipple		6.202E-04		4.434E-04		3.901E-04	2.663E-04
	Fish, general	6.719E-06	9.746E-04	3.701E-04	1.082E-03	1.146E-03	1.063E-03	1.126E-03
	Greens	1.028E-08	1.011E-08	2.465E-09	6.328E-09	1.852E-08	3.972E-09	4.953E-09
	Human milk	4.587E-04		3.428E-04		1.952E-04		
	Meat	1.471E-07	9.750E-08	1.133E-07	1.476E-07	9.887E-08	1.841E-07	1.825E-07
	Milk	3.244E-07	3.045E-07	1.349E-07	4.601E-07	3.777E-07	5.904E-07	3.267E-07
	Pacifier	6.201E-04	6.201E-04	4.432E-04	4.432E-04	3.907E-04	3.907E-04	2.662E-04
	Root vegetable	5.571E-05	6.142E-05	5.768E-05	7.632E-05	4.353E-05	5.262E-05	4.168E-05
	Sipper tube	3.100E-04	3.100E-04	2.216E-04	2.216E-04	1.953E-04	1.953E-04	1.331E-04
	Soil	1.909E-05	1.909E-05	1.365E-05	1.365E-05	1.146E-05	1.146E-05	8.200E-06
	Straws	6.201E-04	1.240E-04	8.864E-05	8.864E-05	7.805E-05	7.805E-05	5.323E-05
	Fish, subsistence	3.837E-03	3.837E-03	2.744E-03	2.744E-03	2.284E-03	2.284E-03	1.648E-03
	Water	1.325E-06	1.325E-06	9.479E-07	9.479E-07	7.950E-07	7.950E-07	5.691E-07

Table 4 summarizes all ingestion-related 90th percentile exposures specific to breastfed and non-breastfed infants. There are no significant differences in the results generated by the current assessment (shown above) and the SEHSC assessment. The two highest contributors to ingestion exposure for this subpopulation are antifoam and fish (subsistence).

Figure 1. Contribution of scenarios to children’s mean and 90th percentile dermal exposures

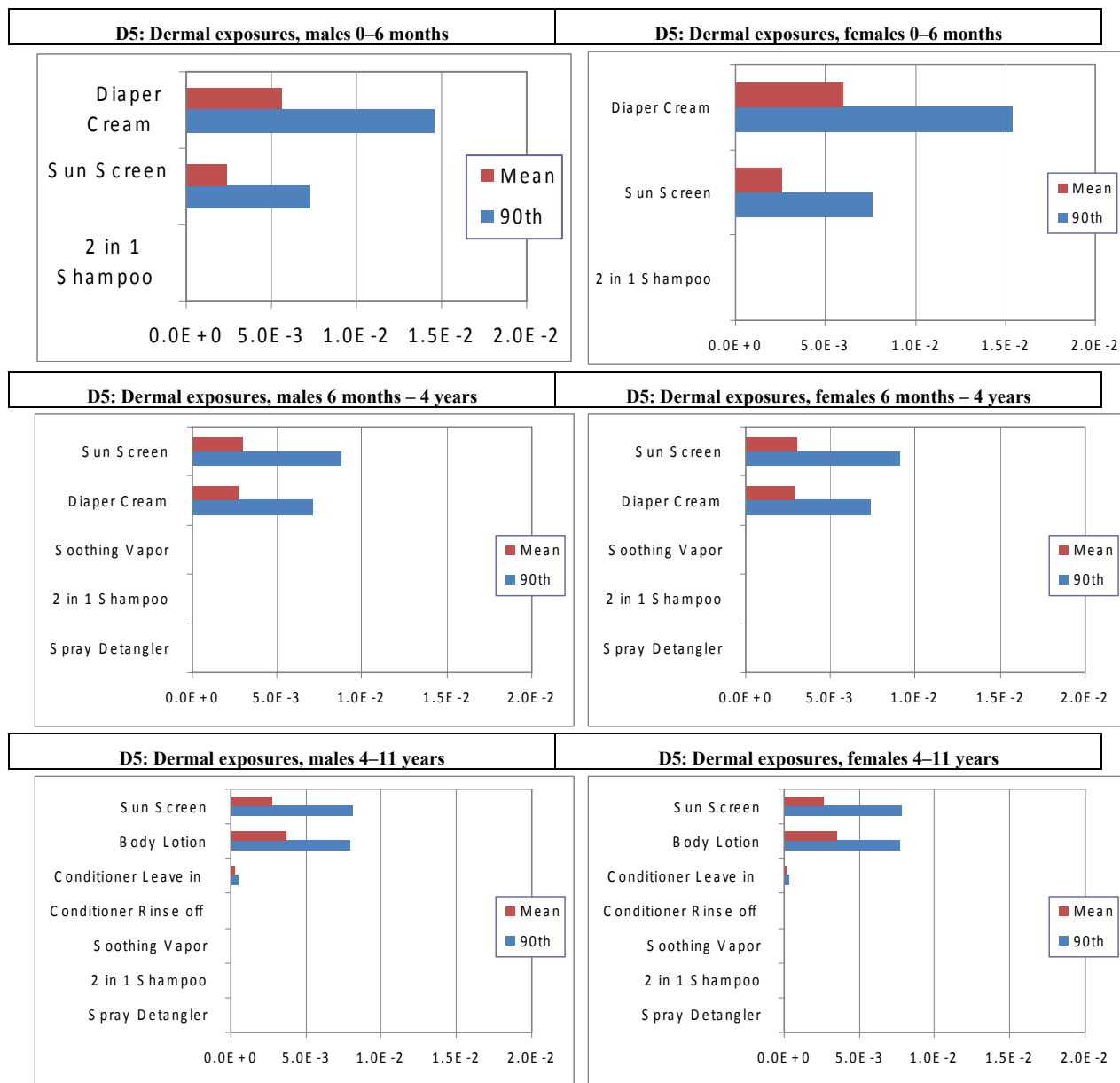


Figure 1 shows the contribution of scenarios to dermal exposures for children’s mean and 90th percentile exposures. As seen in the bar charts, diaper cream, sunscreen, and body lotion are the highest contributors to dermal exposures.

Figure 2. Contribution of scenarios to children’s mean and 90th percentile inhalation exposures

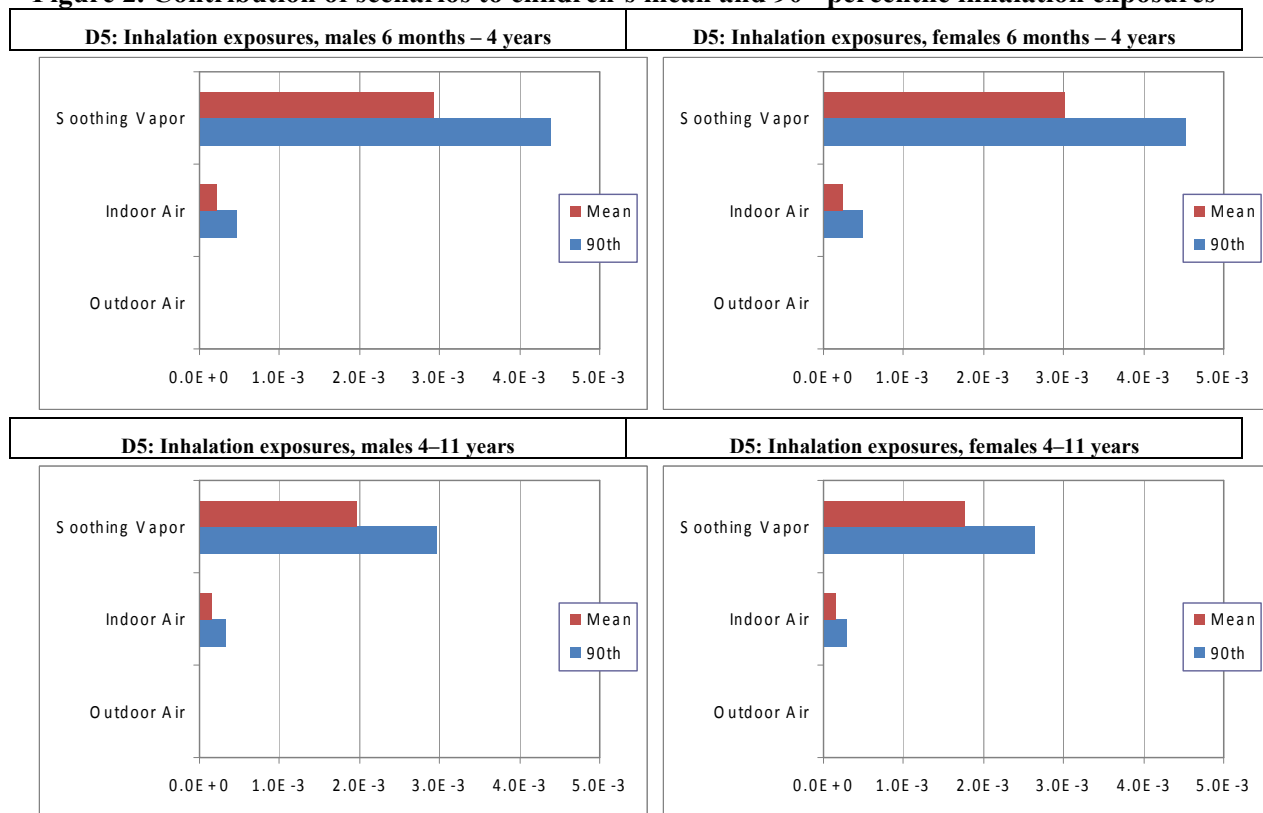


Figure 2 shows the contribution of scenarios to inhalation exposures for children’s mean and 90th percentile exposures. As seen in the bar charts, soothing vapour is the highest contributor to inhalation exposures.

Figure 3. Contribution of scenarios to children’s (breastfed vs. non-breastfed) mean and 90th percentile ingestion exposures

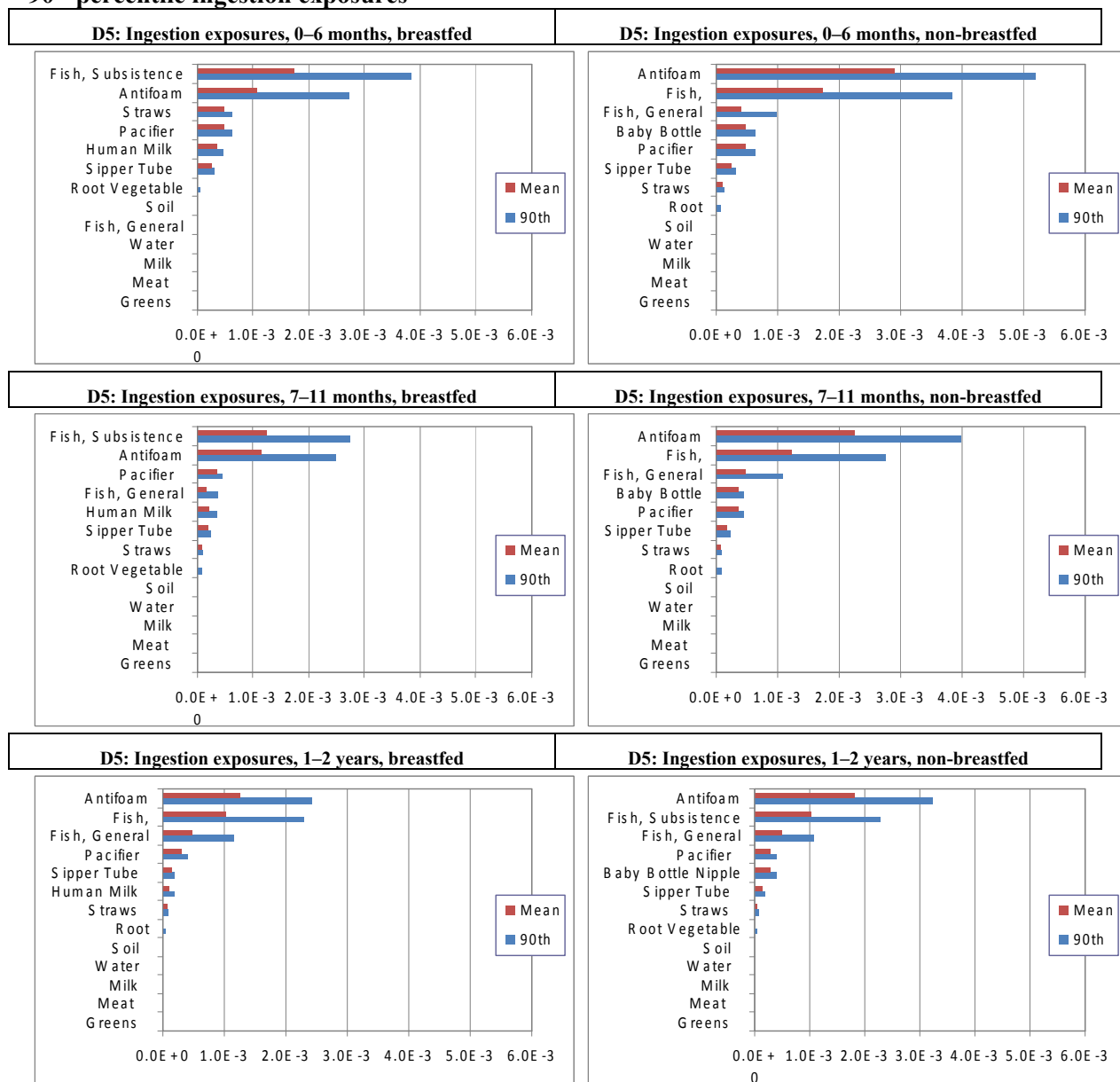


Figure 3 shows the contribution of scenarios to ingestion exposures for children’s (breastfed and non-breastfed) mean and 90th percentile exposures. As seen in the bar charts, the highest contributors to ingestion exposures for this subpopulation are antifoam and fish (for the general and subsistence population).

Figure 4. Contribution of scenarios to children’s (2–4 and 4–11 years) mean and 90th percentile ingestion exposures

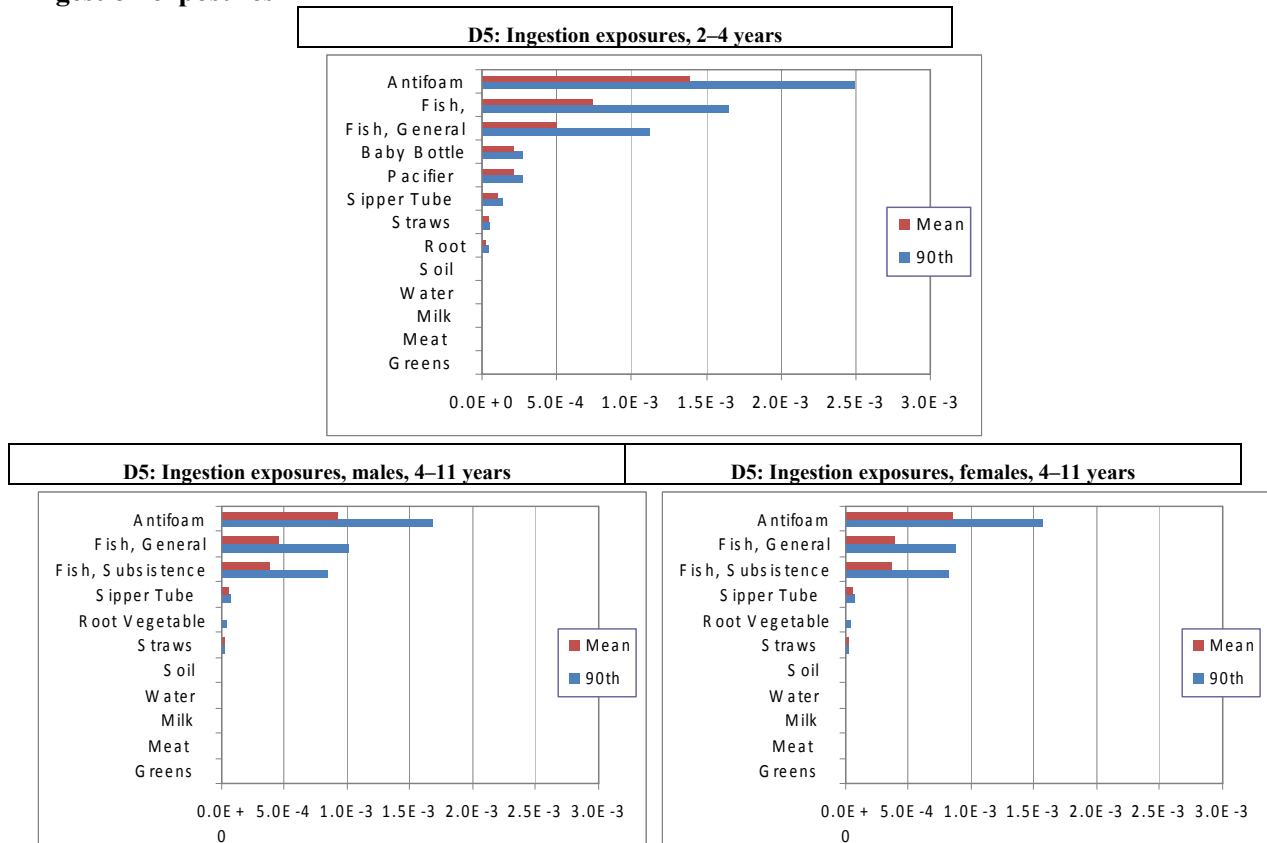


Figure 4 shows the contribution of scenarios to ingestion exposures for children’s (2–4 and 4–11 years) mean and 90th percentile exposures. Exposure estimates are based on 200 000 Crystal Ball simulations. As seen in the bar charts, the highest contributors to ingestion exposures for this subpopulation are antifoam and fish (for the general and subsistence population).

Figure 5. Contribution of exposure route to children’s mean and 90th percentile total exposure (general population)

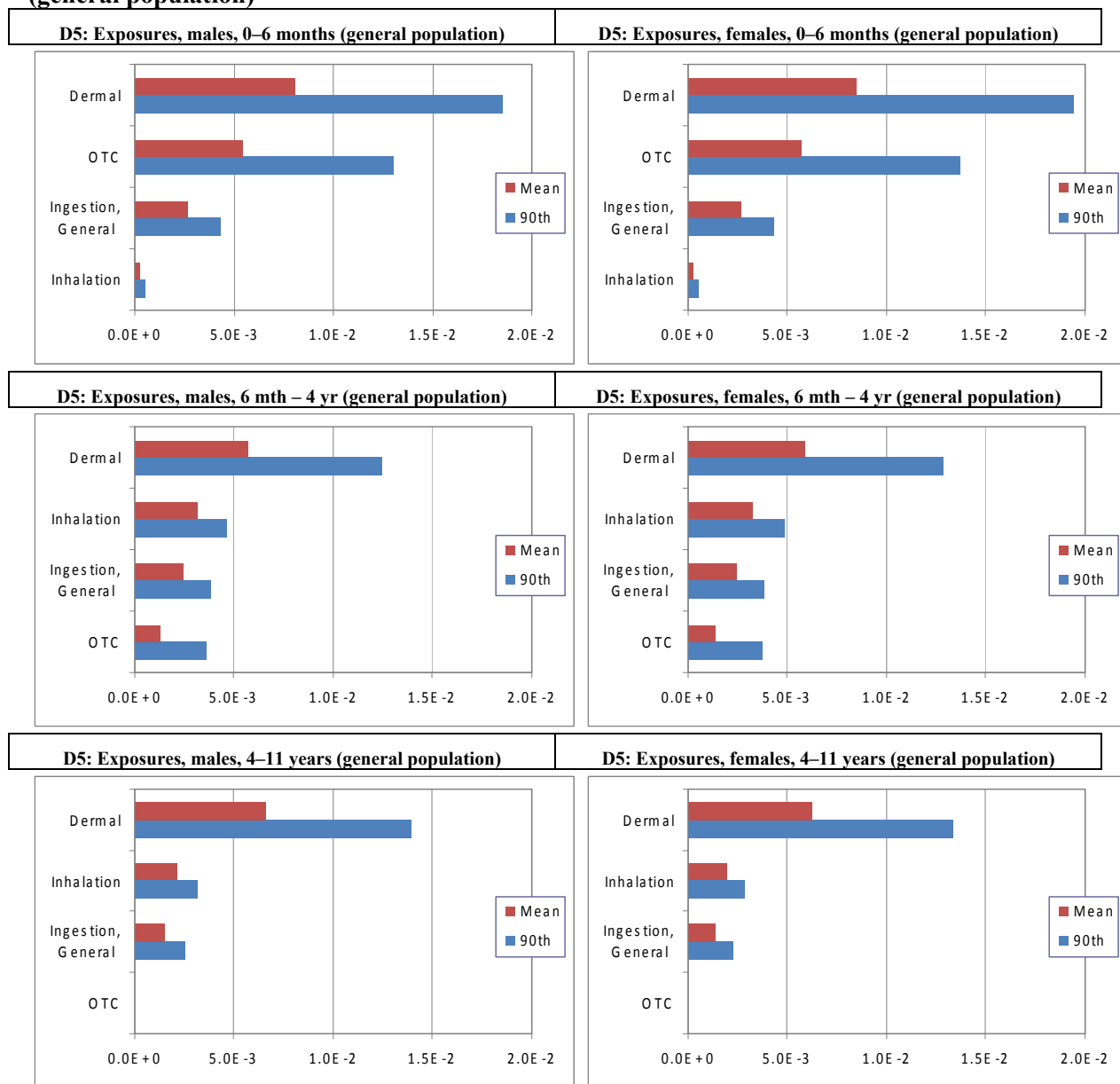


Figure 5 shows the contribution of exposure route to children’s mean and 90th percentile total exposures for the general population. The highest exposure route is dermal. In the case of children 0–6 months old, dermal exposure is followed by over-the-counter drugs and then by ingestion; however, in the case of children 6 months – 4 years old and children 4–11 years old, dermal exposure is followed by inhalation and then by ingestion.

Figure 6. Contribution of exposure route to children’s mean and 90th percentile total exposure (subsistence population)

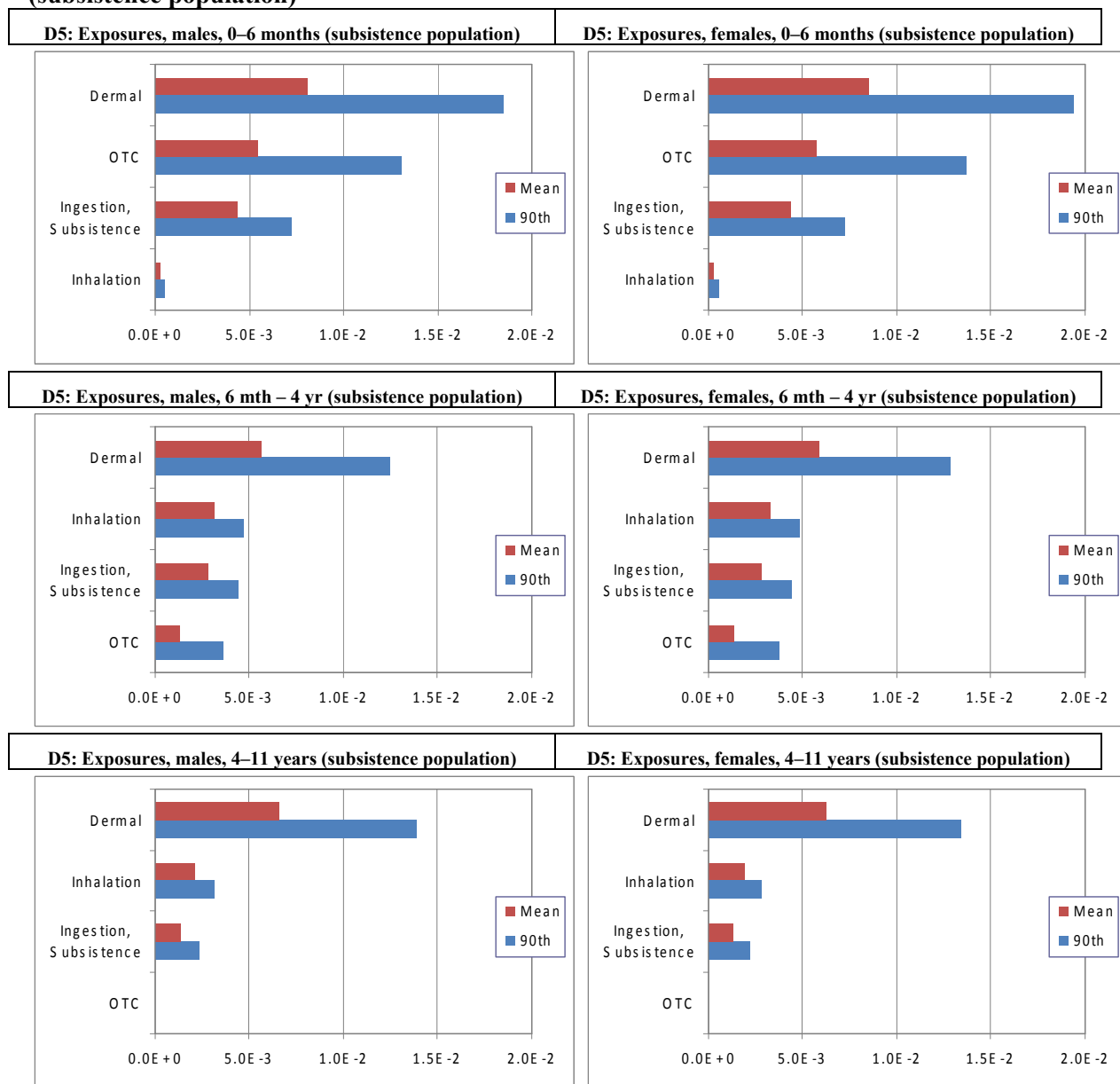


Figure 6 shows the contribution of exposure route to children’s mean and 90th percentile total exposures for the subsistence population. The highest exposure route is dermal. In the case of children 0–6 months old, dermal exposure is followed by over-the-counter drugs and then by ingestion; however, in the case of children 6 months – 4 years old and children 4–11 years old, dermal exposure is followed by inhalation and then by ingestion.

EXPOSURE RESULTS AND DISCUSSION: ADULTS

Table 5. Adult's mean exposures (based on 200 000 Crystal Ball simulations)

Mean exposures		12–19 years		20–59 years		60+ years		
		Males	Females	Males	Females	Males	Females	
DERMAL	After shave	1.07E-04		8.25E-05		8.45E-05		
	Body lotion	3.11E-03	3.46E-03	2.40E-03	2.83E-03	2.45E-03	2.93E-03	
	Soothing vapour	3.07E-06	3.41E-06	2.37E-06	2.80E-06	2.42E-06	2.90E-06	
	Foundation		1.47E-04		1.21E-04		1.25E-04	
	Hair spray	1.09E-04	6.44E-05	8.36E-05	5.28E-05	8.56E-05	5.46E-05	
	Leave-in conditioner	6.21E-05	3.70E-05	4.78E-05	3.04E-05	4.89E-05	3.14E-05	
	Rinse-off Cnditioner	1.86E-06	1.11E-06	1.43E-06	9.08E-07	1.47E-06	9.40E-07	
	Mascara		3.45E-05		2.83E-05		2.93E-05	
	Moisturizer		1.18E-03		9.70E-04		1.00E-03	
	Nail care		1.14E-04		9.31E-05		9.65E-05	
	Roll-on antiperspirant	5.58E-04	4.13E-04	4.29E-04	3.38E-04	4.40E-04	3.51E-04	
	Shampoo	1.17E-07	6.95E-08	9.04E-08	5.70E-08	9.26E-08	5.90E-08	
	Solid antiperspirant	6.54E-04	4.47E-04	5.03E-04	3.66E-04	5.15E-04	3.79E-04	
	Sunscreen	1.88E-03	2.09E-03	1.45E-03	1.71E-03	1.48E-03	1.77E-03	
	Under-eye cream				4.24E-05		4.39E-05	
	<i>Total Dermal</i>	6.49E-03	7.99E-03	5.00E-03	6.59E-03	5.11E-03	6.83E-03	
	INHALATION	Indoor air	8.48E-05	6.94E-05	6.13E-05	5.28E-05	5.36E-05	4.91E-05
		Outdoor air	1.69E-06	1.38E-06	1.22E-06	1.05E-06	1.07E-06	9.77E-07
		Soothing vapour	1.10E-03	9.01E-04	7.96E-04	6.86E-04	6.95E-04	6.37E-04
		<i>Total inhalation</i>	1.19E-03	9.72E-04	8.59E-04	7.40E-04	7.50E-04	6.87E-04
INGESTION	Fish, general population	3.08E-04	2.24E-04	2.39E-04	2.36E-04	2.35E-04	2.68E-04	
	Leafy vegetables	1.90E-08	1.75E-08	2.21E-08	2.51E-08	2.45E-08	2.71E-08	
	Root crops	8.39E-06	6.83E-06	7.49E-06	6.30E-06	7.33E-06	6.59E-06	
	Lipstick		1.26E-05		1.03E-05		1.07E-05	
	Meat	5.47E-08	3.76E-08	4.68E-08	3.32E-08	3.28E-08	2.86E-08	
	Milk	4.63E-08	3.20E-08	1.97E-08	1.89E-08	2.05E-08	1.97E-08	
	Soil	5.90E-07	6.55E-07	4.54E-07	5.37E-07	4.65E-07	5.56E-07	
	Fish, subsistence population	4.07E-04	4.51E-04	3.13E-04	3.70E-04	3.20E-04	3.83E-04	
	Water	1.01E-07	1.13E-07	1.02E-07	1.20E-07	1.04E-07	1.24E-07	
	Antifoam	8.09E-04	7.98E-04	7.07E-04	6.38E-04	5.89E-04	5.63E-04	
	OTC drugs	1.02E-06	1.13E-06	7.84E-07	9.26E-07	8.02E-07	9.59E-07	
	<i>Total Ingestion, General</i>	1.13E-03	1.04E-03	9.55E-04	8.92E-04	8.32E-04	8.49E-04	
	<i>Total Ingestion, Subsistence</i>	1.23E-03	1.27E-03	1.03E-03	1.03E-03	9.17E-04	9.65E-04	
TOTAL	<i>General Population</i>	8.80E-03	1.00E-02	6.81E-03	8.22E-03	6.70E-03	8.36E-03	
	<i>Subsistence Population</i>	8.90E-03	1.02E-02	6.88E-03	8.36E-03	6.78E-03	8.48E-03	

Table 5 summarizes all the exposure results for adults as mean values. When compared with similar results generated by the SEHSC assessment, almost all the values are higher in the current assessment. Compared to the SEHSC assessment, the current assessment results in a difference of about 2.16 times for total dermal exposures, in a difference of about 13.55 times for total inhalation exposures, and in a difference of about 1.24 times for total ingestion (general population) exposures. The primary reason for the differences is that the SEHSC assessed users and non-users whereas the current assessment considered users only.

Table 6. Adults' 90th percentile exposures (based on 200 000 Crystal Ball simulations)

90 th percentile exposures		12–19 years		20–59 years		60+ years		
		Males	Females	Males	Females	Males	Females	
DERMAL	After shave	1.62E-04		1.24E-04		1.27E-04		
	Body lotion	6.77E-03	7.51E-03	5.21E-03	6.16E-03	5.33E-03	6.38E-03	
	Soothing vapour rub	4.29E-06	4.76E-06	3.30E-06	3.90E-06	3.38E-06	4.04E-06	
	Foundation		3.69E-04		3.02E-04		3.13E-04	
	Hair spray	2.70E-04	1.54E-04	2.08E-04	1.26E-04	2.13E-04	1.31E-04	
	Leave-in conditioner	1.92E-04	1.07E-04	1.48E-04	8.79E-05	1.51E-04	9.10E-05	
	Rinse-off conditioner	5.74E-06	3.18E-06	4.42E-06	2.61E-06	4.52E-06	2.70E-06	
	Mascara		6.19E-05		5.08E-05		5.26E-05	
	Moisturizer		2.79E-03		2.29E-03		2.37E-03	
	Nail care		1.16E-04		9.44E-05		9.75E-05	
	Roll-on antiperspirant	1.07E-03	7.92E-04	8.26E-04	6.48E-04	8.45E-04	6.72E-04	
	Shampoo	2.50E-07	1.59E-07	1.92E-07	1.30E-07	1.97E-07	1.35E-07	
	Solid antiperspirant	8.73E-04	9.63E-04	6.72E-04	7.90E-04	6.87E-04	8.18E-04	
	Sunscreen	3.38E-03	3.75E-03	2.60E-03	3.07E-03	2.66E-03	3.18E-03	
	Under-eye cream				7.33E-05		7.59E-05	
		Total Dermal	1.04E-02	1.29E-02	8.01E-03	1.06E-02	8.20E-03	1.10E-02
	INHALATION	Indoor	1.76E-04	1.43E-04	1.27E-04	1.09E-04	1.11E-04	1.01E-04
Outdoor		4.02E-06	3.31E-06	2.93E-06	2.52E-06	2.56E-06	2.33E-06	
Soothing vapour		1.62E-03	1.29E-03	1.13E-03	9.69E-04	9.93E-04	9.03E-04	
		Total inhalation	1.72E-03	1.37E-03	1.20E-03	1.03E-03	1.05E-03	9.58E-04
INGESTION	Fish, general population	6.91E-04	5.20E-04	5.39E-04	5.25E-04	5.20E-04	5.92E-04	
	Leafy vegetables	3.75E-09	3.39E-09	4.00E-09	4.47E-09	4.60E-09	4.78E-09	
	Root crops	2.24E-05	1.81E-05	2.04E-05	1.75E-05	2.03E-05	1.85E-05	
	Lipstick		3.13E-05		2.57E-05		2.66E-05	
	Meat	1.08E-07	7.57E-08	9.41E-08	6.64E-08	6.51E-08	5.70E-08	
	Milk	9.47E-08	6.81E-08	4.11E-08	4.00E-08	4.14E-08	4.14E-08	
	Soil	7.96E-07	8.83E-07	6.12E-07	7.23E-07	6.27E-07	7.49E-07	
	Fish, subsistence population	9.09E-04	1.01E-03	6.99E-04	8.27E-04	7.17E-04	8.56E-04	
	Water	1.91E-07	2.12E-07	1.74E-07	2.06E-07	1.78E-07	2.13E-07	
	Antifoam	1.46E-03	1.24E-03	1.26E-03	1.15E-03	1.03E-03	1.00E-03	
	OTC drugs	2.45E-06	2.72E-06	1.88E-06	2.23E-06	1.93E-06	2.31E-06	
		Total Ingestion, General	1.94E-03	1.56E-03	1.65E-03	1.54E-03	1.40E-03	1.44E-03
		Total Ingestion, Subsistence	2.09E-03	1.95E-03	1.76E-03	1.76E-03	1.54E-03	1.64E-03
TOTAL	General Population	1.28E-02	1.50E-02	9.95E-03	1.23E-02	9.87E-03	1.26E-02	
	Subsistence Population	1.30E-02	1.52E-02	1.00E-02	1.25E-02	9.96E-03	1.27E-02	

Table 6 summarizes all the exposure results for adults as 90th percentile values. When compared with similar results generated by the SEHSC assessment, almost all the values are higher in the current assessment. Compared to the SEHSC assessment, the current assessment results in a difference of about 1.55 times for total dermal exposures, in a difference of about 9.44 times for total inhalation exposures, and in a difference of about 1.17 times for total ingestion (general population) exposures. The primary reason for the differences is that the SEHSC assessed users and non-users whereas the current assessment considered users only.

Figure 7. Contribution of scenarios to adults' mean and 90th percentile dermal exposures

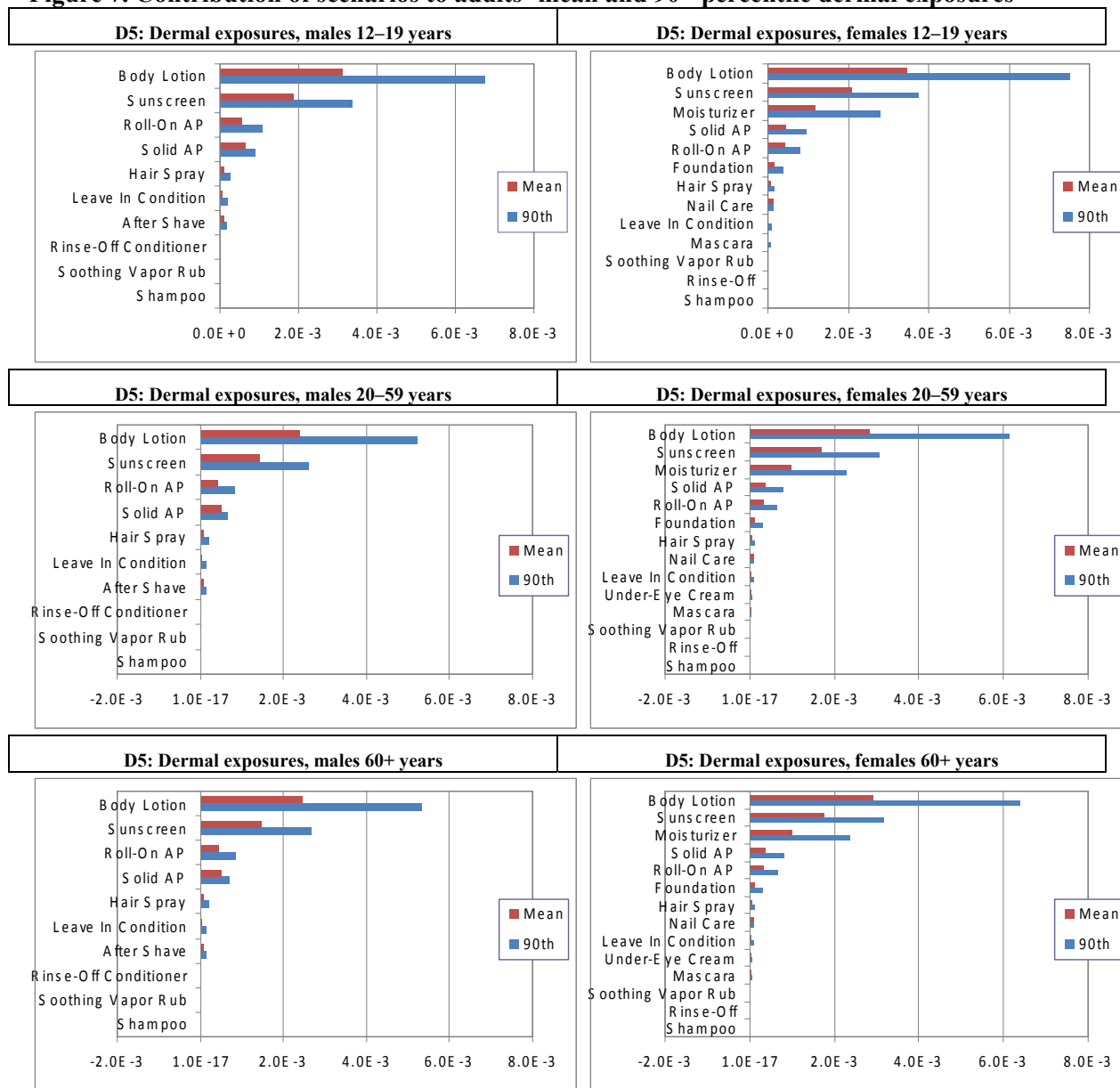


Figure 7 shows the contribution of scenarios to dermal exposures for adults' mean and 90th percentile exposures. As seen in the bar charts, body lotion, sunscreen and moisturizer are the highest contributors to dermal exposures.

Figure 8. Contribution of scenarios to adults’ mean and 90th percentile inhalation exposures

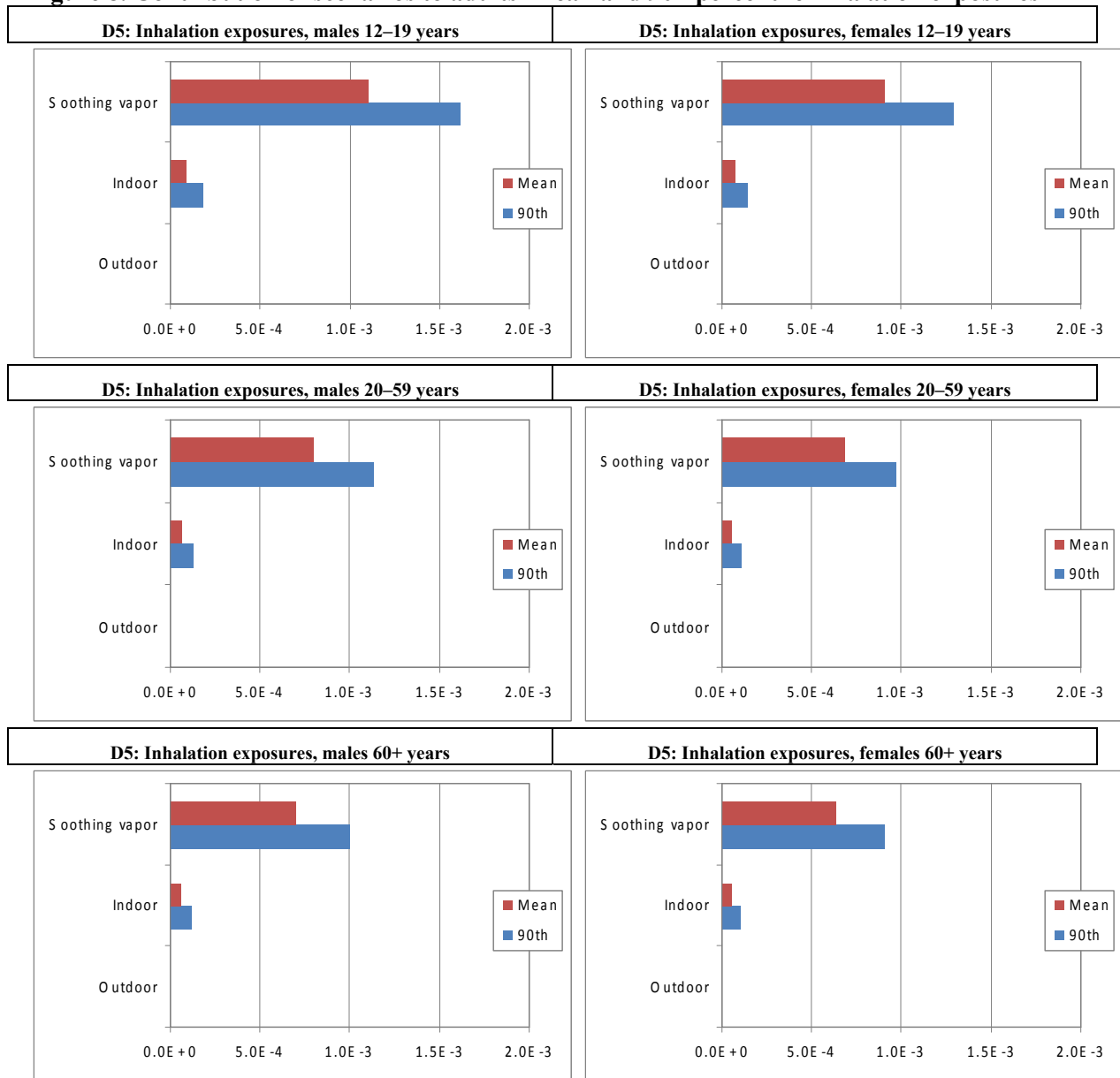


Figure 8 shows the contribution of scenarios to inhalation exposures for adults’ mean and 90th percentile exposures. As seen in the bar charts, soothing vapour is the highest contributor to inhalation exposures.

Figure 9. Contribution of scenarios to adults' mean and 90th percentile ingestion exposures

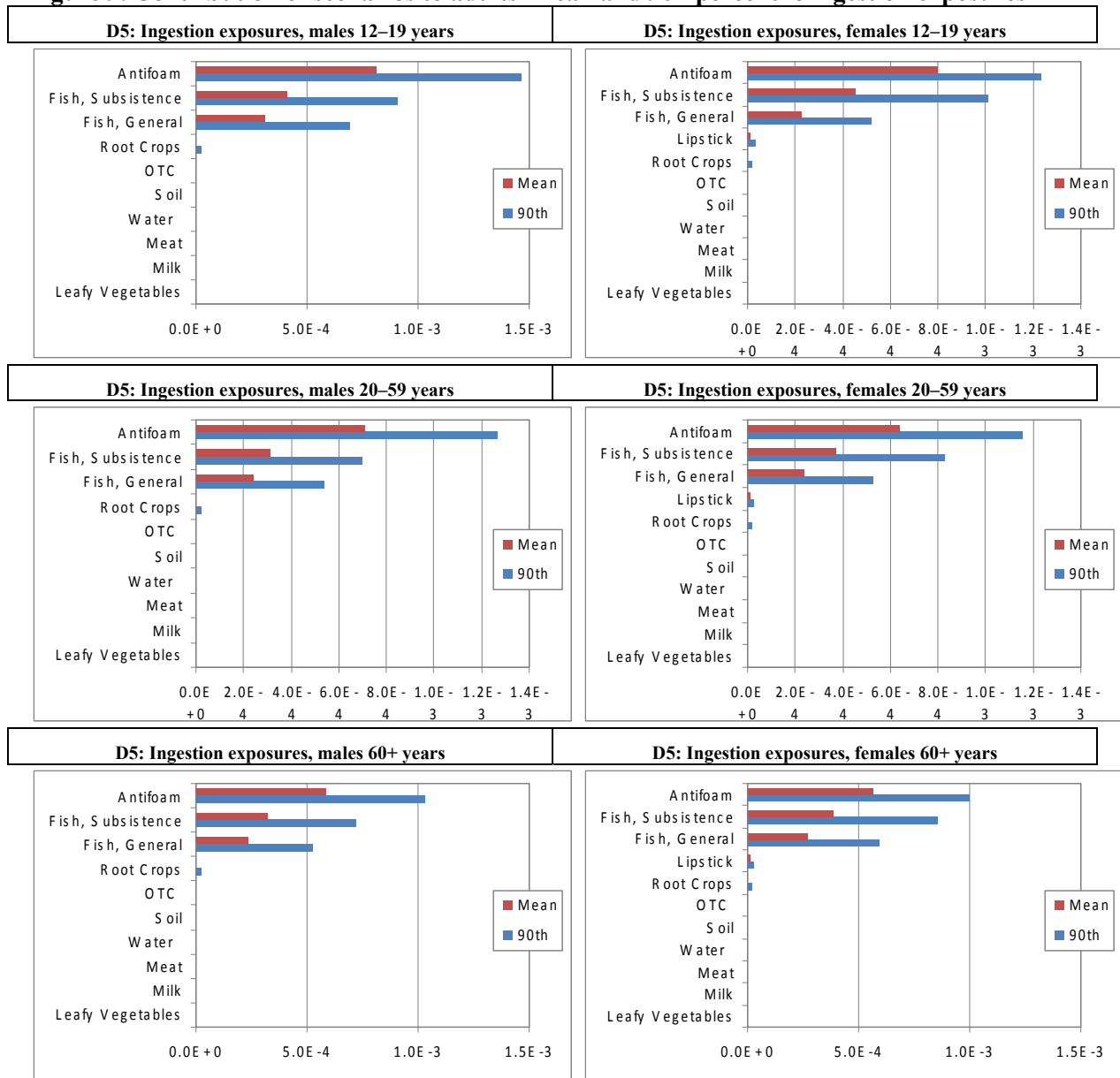


Figure 9 shows the contribution of scenarios to ingestion exposures (general population) for adults' mean and 90th percentile exposures. As seen in the bar charts, antifoam and fish are the highest contributors to ingestion exposures.

Figure 10. Contribution of exposure route to adults' mean and 90th percentile total exposure (general population)

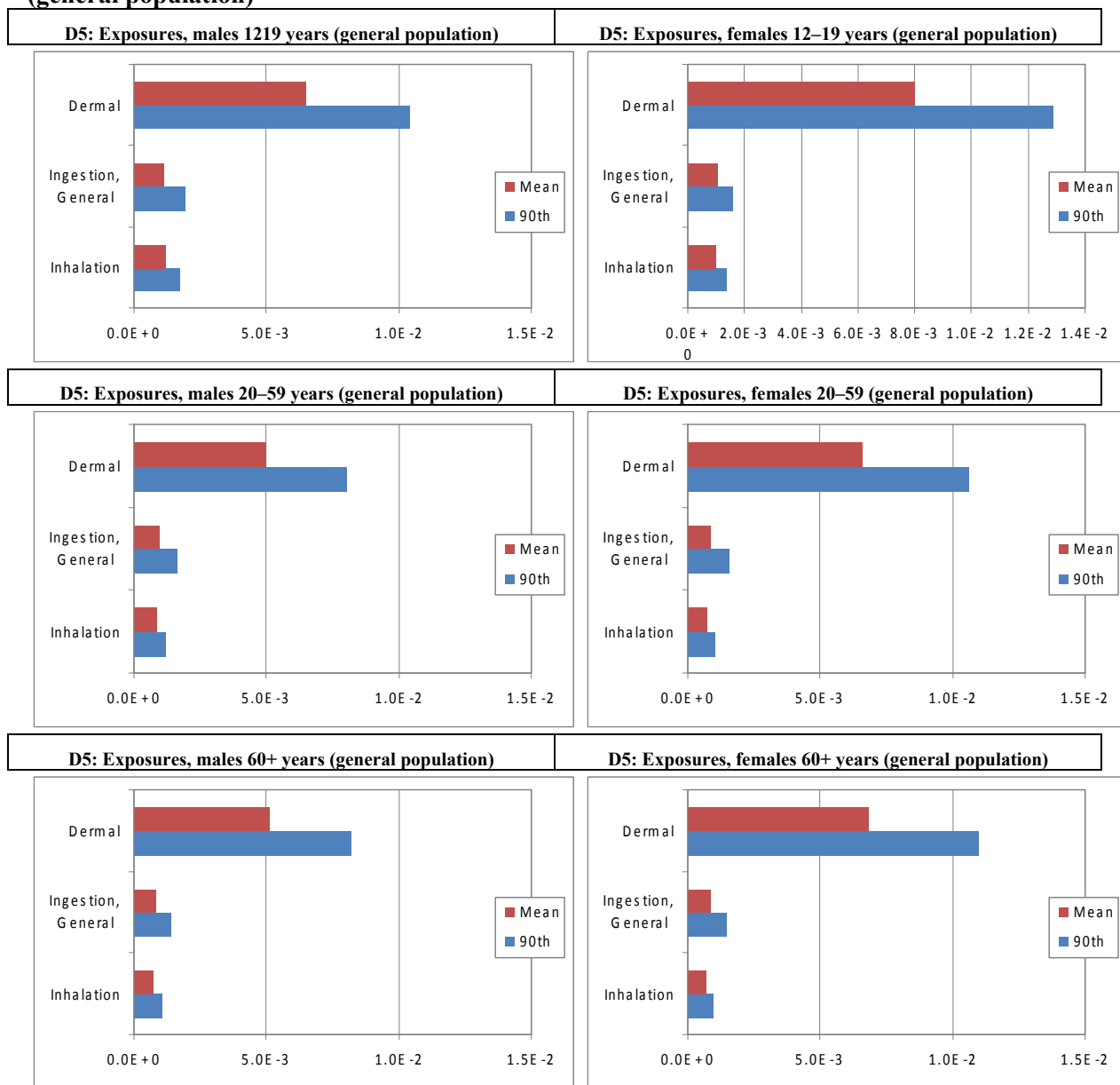


Figure 10 shows the contribution of exposure route to adults' mean and 90th percentile total exposures for the general population. The highest exposure route is dermal. Dermal is followed by ingestion and inhalation.

Figure 11. Contribution of exposure route to adults' mean and 90th percentile total exposure (subsistence population)

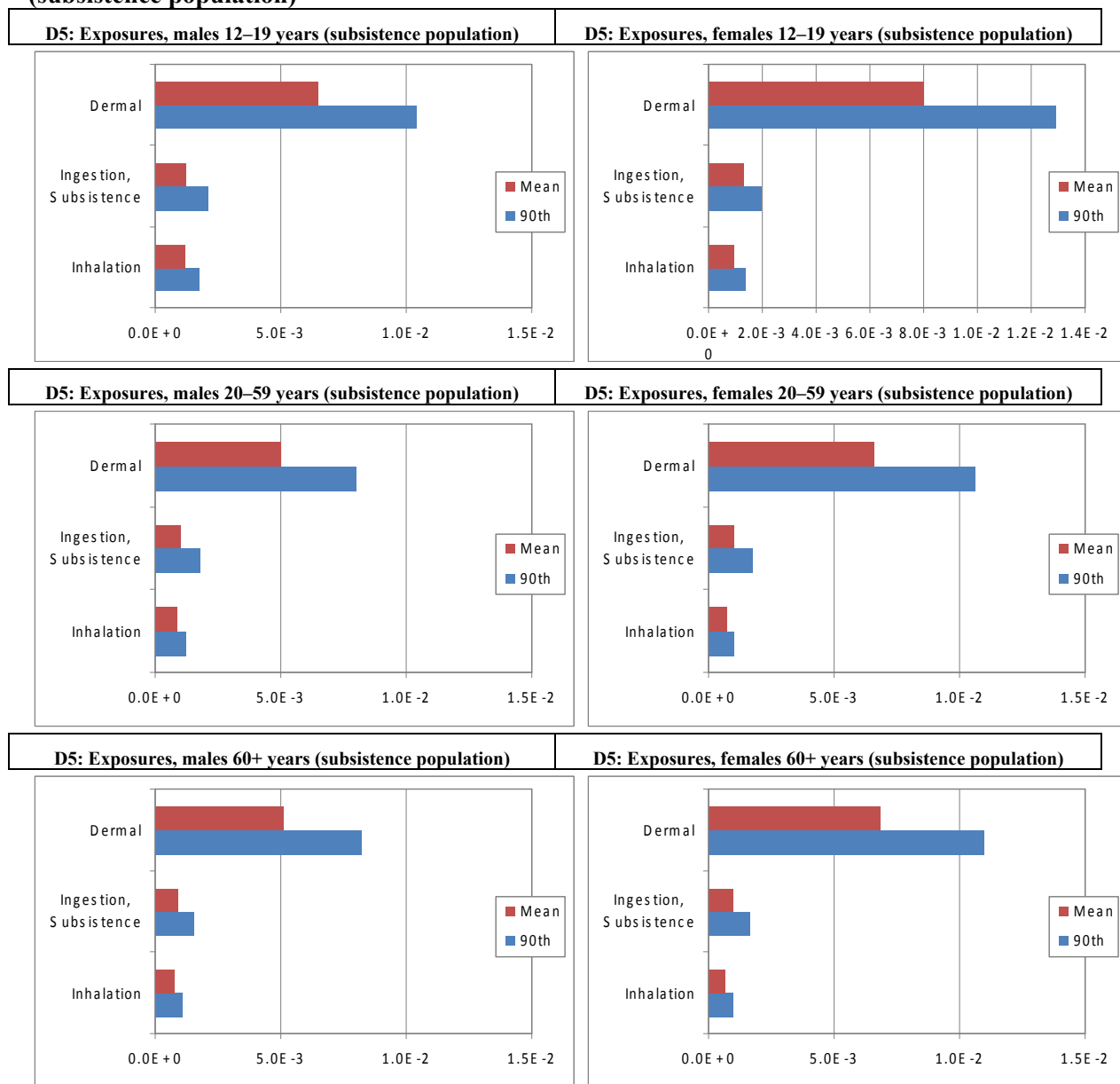


Figure 11 shows the contribution of exposure route to adults' mean and 90th percentile total exposures for the subsistence population. The highest exposure route is dermal. Dermal is followed by ingestion and inhalation.

Appendix 5: Multimedia modelling input parameters for D5 in the ecological screening assessment

Model input parameter	Value
Molecular weight (g/mol)	371
Melting point (°C)	-38
Boiling point (°C)	210
Data temperature (°C)	25
Density (kg/m³)	954
Vapour pressure (Pa)	33.2 (0.249 mm Hg)
Henry's Law constant (Pa·m³/mol)	3 350 000 (33.1 atm·m ³ /mol)
Log K_{aw} (Air-water partition coefficient; dimensionless)	3.13
Log K_{ow} (Octanol-water partition coefficient; dimensionless)	8.03
Log K_{oc} (Organic carbon-water partition coefficient – L/kg)	5.17
Water solubility (mg/L)	0.017
Log K_{oa} (Octanol-air partition coefficient; dimensionless)	5.06
Soil-water partition coefficient (L/kg)	2958
Sediment-water partition coefficient (L/kg)	5916
Suspended particles-water partition coefficient (L/kg)	29 580
Fish-water partition coefficient (L/kg)	13 300
Aerosol-water partition coefficient; dimensionless	100
Vegetation-water partition coefficient; dimensionless	1479
Half-life in air (days)	6.9
Half-life in water (days)	74
Half-life in sediment (days)	49
Half-life in soil (days)	5.25
Half-life in vegetation (days)	74