Canadian Environmental Protection Act, 1999

Follow-up Report on a PSL1 Assessment for Which Data Were Insufficient to Conclude Whether the Substances Were "Toxic" to the Environment and to the Human Health

Chlorinated Paraffins

August 2008

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LIST OF ACRONYMS AND ABBREVIATIONS

ACP	Arctic contamination potential
BAF	bioaccumulation factor
BCF	bioconcentration factor
BMF	biomagnification factor
BOD	biochemical oxygen demand
BSAF	biota–sediment accumulation factor
CB	chlorinated biphenyl
CEPA 1988	Canadian Environmental Protection Act
CEPA 1999	Canadian Environmental Protection Act, 1999
CoA	coenzyme A
CP	chlorinated paraffin
CPIA	Chlorinated Paraffins Industry Association
CTV	Critical Toxicity Value
CYP	cytochrome P-450
DDT	dichlorodiphenyltrichloroethane
DFO	Department of Fisheries and Oceans
DNA	deoxyribonucleic acid
EC50	median effective concentration
ECNI	electron capture negative ion
ECNI-HRMS	electron capture negative ion high-resolution mass spectrometry
EEV	Estimated Exposure Value
ENEV	Estimated No-Effects Value
EPA	Environmental Protection Agency
EQC	Equilibrium Criterion
EU	European Union
foc	fraction of organic carbon
GC	gas chromatography
GLP	Good Laboratory Practice
HCB	hexachlorobenzene
HCH	hexachlorocyclohexane
HLC	Henry's law constant
HPLC	high-pressure liquid chromatography
HR	high resolution
HRGC	high-resolution gas chromatography
HRMS	high-resolution mass spectrometry
IC50	median inhibitory concentration
KAW	air–water partition coefficient (or unitless Henry's law constant)
kg-bw	kilogram body weight
KOA	octanol–air partition coefficient
KOC	organic carbon sorption coefficient (or organic carbon–water partition coefficient)
Kow	octanol/water partition coefficient
LC50	median lethal concentration
LCCP	long-chain chlorinated paraffin

LCCPs	long-chain chlorinated paraffins
liquid LCCPs	C18–20 and C>20 liquid LCCPs
LOAEL	Lowest-Observed-Adverse-Effect Level
LOEC	Lowest-Observed-Effect Concentration
LOEL	Lowest-Observed-Effect Level
LR	low resolution
LRMS	low-resolution mass spectromety
LT50	time to 50% lethality
MCCP	medium-chain chlorinated paraffin
MCCPs	medium-chain chlorinated paraffins
MS	mass spectrometry
NCI	negative ion chemical ionization
NIMS	negative ion mass spectrometry
NOAEL	No-Observed-Adverse-Effect Level
NOEC	No-Observed-Effect Concentration
NOEL	No-Observed-Effect Level
NPRI	National Pollutant Release Inventory
NTP	National Toxicology Program (U.S.A.)
NWRI	National Water Research Institute
OCDD	octachlorodibenzodioxin
OECD	Organisation for Economic Co-operation and Development
PAH	polycyclic aromatic hydrocarbon
PCB	polychlorinated biphenyl
PCDD	polychlorinated dibenzodioxin
PCDF	polychlorinated dibenzofuran
PNEC	Predicted No-Effect Concentration
POC	particulate organic carbon
PPARα	peroxisome proliferator activated receptor, α isoform
PSL1	first Priority Substances List
PUF	polyurethane foam
PVC	polyvinyl chloride
QSAR	quantitative structure–activity relationship
SCCP	short-chain chlorinated paraffin
SCCPs	short-chain chlorinated paraffins
T3	triiodothyronine
T4	thyroxine
TCDD	tetrachlorodibenzodioxin
TD05	Tumorigenic Dose05, the dose associated with a 5% increase in tumour incidence
TDI	Tolerable Daily Intake
TGD	(European) Technical Guidance Document
TLC	thin-layer chromatography
TSH	thyroid stimulating hormone
TSMP	Toxic Substances Management Policy
UDP	uridine diphosphate
UDPG	uridine diphosphoglucose

UDPGGT	uridine diphosphoglucose glucuronosyl transferase
UDPGT	uridine diphosphate glucuronosyl transferase
UNECE	United Nations Economic Commission for Europe
VP	vapour pressure
wt.	weight
WWTP	wastewater treatment plant

PREFACE

Following the assessment of chlorinated paraffins conducted under the first Priority Substances List (PSL1), available data were considered inadequate to evaluate whether medium and long chain chlorinated paraffins were considered to be "toxic" as defined under section 11 of the 1988 *Canadian Environmental Protection Act* (CEPA 1988). While information on the environmental effects of short-chain chlorinated paraffins was considered insufficient to conclude whether they were "toxic" under Paragraph 11(a) of CEPA 1988, this group of substances was considered "toxic" to human health under Paragraph 11(c) of CEPA 1988. In updating the assessments of medium and long chain chlorinated paraffins, included herein, more recent data on the effects of short-chain chlorinated paraffins on human health and on the environment were also examined.

In this report, the impact of critical new data on the initial assessment under CEPA 1988 is considered. These data are presented separately for the environmental and health effects, but cross-referenced, where appropriate. Information relevant to assessment of effects on the environment is presented initially, followed by information relevant to assessment of effects on human health.

SYNOPSIS

Chlorinated paraffins (CPs) are chlorinated derivatives of n-alkanes with carbon chain lengths from 10 to 38 carbon atoms, and with varying chlorine contents. CPs include short chain chlorinated paraffins (SCCPs) (CPs with 10–13 carbon atoms), medium chain chlorinated paraffins (MCCPs) (CPs with 14–17 carbon atoms) and long chain chlorinated paraffins (LCCPs) (CPs with ≥18 carbon atoms).

CPs were included on the first Priority Substances List (PSL1) under the 1988 *Canadian Environmental Protection Act* (CEPA 1988) for assessment of potential risks to the environment and human health. With the data available at that time, it was concluded that SCCPs were "toxic" because they constitute or may constitute a danger in Canada to human life or health as defined under paragraph 11(c) of CEPA 1988. However, as outlined in the PSL1 assessment report released in 1993, relevant data identified before August 1992 were considered insufficient to conclude whether SCCPs, MCCPs or LCCPs could have immediate or long-term harmful effects on the environment as defined under paragraph 11(a) of CEPA 1988 and whether MCCPs or LCCPS could be considered "toxic" to human health as defined under paragraph 11(c) of CEPA 1988.

Research to address data gaps relevant to the assessment of impacts of CPs on the environment was funded; an industry survey on the Canadian manufacture, import and uses of CPs was conducted for the years 2000 and 2001 through a *Canada Gazette* Notice issued pursuant to section 71 of the *Canadian Environmental Protection Act* 1999 (CEPA 1999); and literature was also reviewed for new exposure and toxicological data on CPs on human and non-human organisms in Canada and elsewhere.

Total reported annual usage of CPs in Canada (production + imports – exports) was approximately 3,000 tonnes in 2000 and 2001. MCCPs accounted for a large majority of CP usage in Canada, followed by smaller proportions of SCCPs and LCCPs. The major uses of CPs in Canada are in plastics, in lubricating additives and in metalworking. There was only one manufacturer of CPs in Canada, and only MCCPs and LCCPs were produced at this facility. In 2000, their production capacity was reported to be 8.5 kilotonnes; however, there is no production in Canada at present.

There are no known natural sources of CPs. The major sources of release of CPs (SCCPs, MCCPs and LCCPs) into the Canadian environment are likely the formulation and manufacturing of products containing CPs and use in metalworking fluids. The possible sources of releases to water from manufacturing include spills, facility wash-down and drum rinsing/disposal. CPs in metalworking/metal cutting fluids may also be released to aquatic environments from drum disposal, carry-off and spent bath. These releases are collected in sewer systems and often ultimately end up in the effluents of sewage treatment plants. When released to the environment, CPs tend to partition primarily to sediment or soil.

Environmental Assessment

SCCPs have been detected in the following Canadian environmental media: Arctic air, sediments from remote northern lakes, sewage treatment plant effluents from southern Ontario, surface water, sediments, plankton, invertebrates and fish from Lake Ontario and marine mammals from the Canadian Arctic and the St. Lawrence River. SCCPs have also been detected in plankton, invertebrates and fish from Lake Michigan. MCCPs have been detected in effluent from a CPs manufacturing facility near Cornwall, Ontario, and also in sediments near this facility (which has since ceased operation), in fish from Lake Ontario and in beluga from the St. Lawrence River. Maximum Canadian concentrations of SCCPs and MCCPs were observed in aquatic biota and sediments from the St. Lawrence River and also in sediments and fish from southwestern Ontario. No data on environmental concentrations in Canada exist for LCCPs. They have been detected in marine sediments, crabs and mussels near a CPs manufacturing facility in Australia.

Atmospheric half-lives for many CPs are estimated to be greater than 2 days. In addition, SCCPs have been detected in Arctic biota and lake sediments in the absence of significant sources of SCCPs in this region, which suggests that long-range atmospheric transport of SCCPs is occurring. SCCP and MCCP residues have been detected in Canadian lake sediments dating back over 25 years at concentrations suggesting that the half-lives of SCCPs and MCCPs in sediment are greater than 1 year. There are no data available for LCCPs in Canadian lake sediments; however, based on their physical/chemical properties, which are similar to those of MCCPs, LCCPs are expected to be persistent in sediments. Several biodegradation studies have also found that biodegradation is hindered by increasing carbon chain length. It is, therefore, concluded that SCCPs, MCCPs and LCCPs are persistent as defined in the Persistence and Bioaccumulation Regulations of CEPA 1999.

Bioaccumulation factors (BAFs) of 9900–51200 wet weight in sculpin, smelt and trout from Lake Ontario indicate that SCCPs are bioaccumulating to a high degree in aquatic biota in Canada, this is supported by very high laboratory-derived bioconcentration factors (BCFs). Despite the lack of valid studies of BCFs, MCCPs have been found to have significant potential to bioaccumulate in aquatic food webs: field BAFs for MCCPs in some Lake Ontario fish were calculated to be approximately 5450 wet weight. In addition BAF values calculated using the Modified Gobas BAF Model are >5000 for all SCCP and MCCP congeners.

While biomagnification factors (BMFs) are not considered in the bioaccumulation Regulations, they are supporting evidence for bioaccumulation when substantially above 1. Both SCCPs and MCCPs were found to have biomagnification factors (BMFs) greater than one in various food webs. MCCPs also had BMFs greater than one. The liquid LCCP $C_{18}H_{30}Cl_7$ had BMF values greater than one in rainbow trout in laboratory studies, and its half-life in rainbow trout was found to be similar to those of recalcitrant compounds that are known to accumulate in organisms and magnify in food chains. In addition, SCCPs, MCCPs and LCCPs have octanol–water partition coefficient (log K_{OW}) values greater than five. Elevated concentrations of MCCPs have been measured in aquatic biota from the St. Lawrence estuary, the United States and Australia. While all of the available published BCF studies for LCCP have found values <5000, some elevated

concentrations of LCCPs have been found in marine benthic organisms in Australia. In addition the Gobas BAF Model predicts that 44% of liquid C_{18-20} LCCP congeners have BAF≥5000. On the other hand, none of the $C_{>20}$ LCCP congeners had modeled BAFs ≥5000. Therefore, based on the weight of evidence, it is concluded that SCCPs, MCCPs, and liquid C_{18-20} LCCPs are bioaccumulative as defined in the Persistence and Bioaccumulation Regulations of CEPA 1999. However based on the limited information available (particularly BAF estimates), $C_{>20}$ liquid and solid LCCPs are not bioaccumulative as defined under the Persistence and Bioaccumulation Regulations.

The available toxicity data indicate that SCCPs , MCCPs and C_{18-20} LCCPs may be harmful to certain aquatic species (e.g., *Daphnia magna*) at low concentrations (e.g., chronic NOECs < 100 μg/L).

SCCPs, MCCPs and C_{18-20} LCCPs are considered to be both highly persistent and bioaccumulative. In addition, there is evidence that SCCPs, MCCPs and C_{18-20} LCCPs are released into the Canadian environment and have the potential to cause harm to sensitive aquatic organisms at relatively low concentrations. Substances that are persistent remain in the environment for a long time, increasing the magnitude and duration of exposure. Releases of small amounts of bioaccumulative substances may lead to high internal concentrations in exposed organisms. Highly bioaccumulative and persistent substances are of special concern, since they may biomagnify in food webs, resulting in very high internal exposures, especially for top predators.

Human Health Assessment

For SCCPs, critical data relevant to both estimation of exposure of the general population in Canada and assessment of the weight of evidence for the mode of induction of specific tumours were identified following release of the PSL1 assessment and prior to February 2001, although most of this information has been reported in incomplete published summary accounts or abstracts. These data suggest that several tumours observed in carcinogenicity bioassays in rats and mice exposed to SCCPs are induced by modes of action either not relevant to humans (kidney tumours in male rats) or for which humans are likely less sensitive (in rats, liver tumours related to peroxisome proliferation and thyroid tumours related to thyroid–pituitary disruption). Complete documentation of available studies and consideration in additional investigations of the reversibility of precursor lesions in the absence of continued exposure is lacking. However, reported data on mode of induction of tumours in addition to the weight of evidence that SCCPs are not DNA reactive are at least sufficient as a basis for consideration of a Tolerable Daily Intake (TDI) for non-cancer effects as protective for carcinogenicity for observed tumours. Upper-bounding estimates of daily intake of SCCPs approach or exceed the TDI for these compounds, which, on the basis of available information, is likely also protective for potential carcinogenicity.

For MCCPs and LCCPs, critical data relevant to both estimation of exposure of the general population in Canada and assessment of effects were identified following release of the PSL1 assessment and prior to December 2000. Based upon these semi-quantitative

data, upper-bounding estimates of daily intake for MCCPs and LCCPs are within the same order of magnitude of, or exceed, the TDIs for these substances.

Conclusion

Based on the information available, it is concluded that CPs containing up to twenty carbon atoms are entering, or may enter, the environment in quantities or concentrations or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity and that all chlorinated paraffins constitute or may constitute a danger in Canada to human life or health. CPs containing up to twenty carbon atoms are persistent and bioaccumulative as defined in the Persistence and Bioaccumulation Regulations.

1. INTRODUCTION

Chlorinated paraffins (CPs) are chlorinated derivatives of n-alkanes with carbon chain lengths from 10 to 38 carbon atoms, and with varying chlorine contents. Commercial products, of which there are over 2000 (Serrone et al. 1987), are complex mixtures of homologues and isomers. CPs with carbon chains containing 10–13 carbon atoms ($C_{10–13}$) are termed "short", those with 14–17 carbon atoms ($C_{14–17}$) are called "medium" and those having 18 or more carbon atoms ($\geq C_{18}$) are called "long". This report addresses the short-chain chlorinated paraffins (SCCPs), the medium-chain chlorinated paraffins (MCCPs) and the long-chain chlorinated paraffins (LCCPs).

CP waxes appeared on the first Priority Substances List (PSL1) of the 1988 *Canadian Environmental Protection Act* (CEPA 1988), published in the *Canada Gazette*, Part I, on February 11, 1989. An assessment was performed to determine whether CPs should be considered "toxic" as defined under CEPA 1988 and was completed in 1993 (Government of Canada 1993a). As a result of this assessment, SCCPs were declared "toxic" under Paragraph 11(c) of CEPA 1988, because they were found to constitute a danger to human health. The conclusion of this assessment, published in the *Canada Gazette*, Part I, on January 22, 1994, also indicates that available data were considered insufficient to determine whether SCCPs, MCCPs or LCCPs could have immediate or long-term harmful effects on the environment as defined under paragraph 11(a) of CEPA 1988 and whether MCCPs or LCCPS could be considered "toxic" to human health as defined under paragraph 11(c) of CEPA 1988.

Subsequent to the completion of the PSL1 assessments, a revised CEPA, CEPA 1999, came into effect on March 31, 2000. Section 64 of CEPA 1999 has a definition of "toxic" that is similar to that in section 11 of CEPA 1988. CEPA 1999 places more emphasis on pollution prevention, and mandates the application of a weight of evidence approach and the precautionary principle when conducting and interpreting the results of risk assessments of existing substances. In addition, CEPA 1999 provides for special consideration of persistent and bioaccumulative substances. Substances that are shown to be both persistent and bioaccumulative, therefore, may be assessed using a more precautionary approach than is used for other substances.

In 1997, a Scientific Justification document recommending that SCCPs be candidate substances for management under Track 1 (virtual elimination) of the Toxic Substances Management Policy (TSMP) (Government of Canada 1995) was published (Environment Canada 1997). The overall conclusion of the document stated: "On the basis of the information reviewed, it is concluded that short chain chlorinated paraffins are predominantly anthropogenic, persistent, bioaccumulative, and CEPA-toxic. Short chain chlorinated paraffins satisfy all four criteria outlined in the Toxic Substances Management Policy to identify substances for management under Track 1. Therefore, short chain chlorinated paraffins are proposed for management under Track 1 of the Policy." During the public comment period on the Scientific Justification, the Chlorinated Paraffins Industry Association (CPIA) reviewed the information cited in the document proposing to list SCCPs as a Track 1 substance. They argued that the evidence did not

constitute a scientifically credible basis to determine CEPA toxicity. Additionally, it was stated that the Scientific Justification document offered no persuasive evidence that SCCPs met the TSMP's prescribed half-life criteria for persistence. In order to further examine the persistence of SCCPs and their potential to cause ecological harm, as well as to reassess MCCPs and LCCPs based on new information, scientists at the National Water Research Institute (NWRI) of Environment Canada and at the Freshwater Institute of the Department of Fisheries and Oceans (DFO) have generated new scientific information to address data gaps relevant to the assessment of impacts of CPs on the environment.

To set further context for the update of the CPs assessment, an industry survey on the Canadian manufacture, import and uses of CPs was conducted for the years 2000 and 2001 through a *Canada Gazette* Notice issued pursuant to section 71 of CEPA 1999 (Environment Canada 2003a). Recent literature was also reviewed for new exposure and toxicological data on CPs on human and non-human organisms in Canada and elsewhere.

This new information is considered in this assessment report. Data acquired prior to February 2001 and December 2000 were considered in the follow-up assessment of whether SCCPs and MCCPs/LCCPs, respectively, constitute or may constitute a danger in Canada to human life or health. Data obtained as of July 2007 were considered as part of the ecological follow-up assessment of SCCPs, MCCPs, and LCCPs.

This assessment report was prepared under the authority of Section 68 of CEPA 1999. It was written by the staff of the Existing Substances Division of Environment Canada and Health Canada, as well as the National Water Research Institute of Environment Canada. The content of this report has been subjected to external review by Canadian and international experts selected from government and academia, and also to a 60-day public comment period. However, the conclusions presented in this report are those of Environment Canada and Health Canada and do not necessarily reflect the opinions of the external reviewers.

This report represents a summary of more detailed information presented in a supporting document. For additional information the reader should consult this document. This assessment report and the associated environmental supporting document are available upon request by e-mail from existing.substances.existantes@ec.gc.ca. Information on assessments under CEPA 1999 is available at http:www.chemicalsubstanceschimiques.gc.ca.

2. IDENTITY AND PHYSICAL/CHEMICAL PROPERTIES

2.1. Identity

As was the case for the PSL1 assessment, SCCPs, MCCPs and LCCPs are assessed separately in this report.

2.1.1 Composition of CP mixtures

SCCPs ($C_{10–13}$), MCCPs ($C_{14–17}$) and the lower chlorinated LCCPs are mixtures that are viscous, colourless or yellowish dense oils. $C_{>20}$ highly chlorinated alkanes are waxy solids at ambient temperatures. The average chlorine content by weight is 30–52% for $C_{18–20}$ liquid products, 40–54% for $C_{>20}$ liquid products, and 70–72% for $C_{>20}$ solid products.

Impurities in commercial CPs are likely to be related to those present in the n-alkane feedstocks, which consist of a mixture of homologues. Furthermore, the n-alkanes may contain branched alkanes (usually <1%) and aromatics (<0.1%), which could be chlorinated. Commercial mixtures also contain stabilizers, which include epoxidized esters and soya bean oils, erythritol, thymol, urea, glycidyl ethers, acetonitriles and organic phosphates (European Commission, 2000; Schenker 1979; Houghton 1993). Various stabilizers are often added to commercial CP products in order to improve their thermal stability or light stability.

2.2. Physical/chemical properties

The large difference in chlorine content is primarily responsible for the large differences that are evident in measurements and estimates of physical/chemical properties. The approximate range of molecular weights for SCCPs is 320–500 (European Commission, 2000), for MCCPs is 235–825 (U.K. Environment Agency 2003) and for LCCPs is 325–1355 (U.K. Environment Agency 2001).

Presented in Table 1 are ranges of physical properties for SCCPs, MCCPs and 3 subclasses of LCCPs.

Table 1. Range of physical properties of CPs congeners.

CP Class	Vapour pressure[a] (Pa)	Henry's Law Constant ($Pa \cdot m^3/mol$)	Water solubility (µg/L)	log K_{OW}	log K_{OA}	Log K_{OC}	Reference[b]
SCCPs	2.8×10^{-7} – 0.028 (48 – 71% Cl)	0.68 – 17.7 (48 – 56% Cl)	6.4 – 2370 (48 – 71% Cl)	4.39 – 8.69 (48 – 71% Cl)	8.2 – 9.8 (48 – 56% Cl)	4.1 – 5.44	1-7, 14, 15
MCCPs	4.5×10^{-8} – 2.27×10^{-3} (42 – 58% Cl)	0.014 – 51.3 (37 – 56% Cl)	9.6×10^{-2} – 50 (37 – 56% Cl)	5.47 – 8.21 (32 – 68% Cl)	8.81 – 12.96 (32 – 68% Cl)	5.0 – 6.23	4, 5, 6, 7, 8, 9, 10, 11

CP Class	Vapour pressure[a] (Pa)	Henry's Law Constant ($Pa \cdot m^3/mol$)	Water solubility (µg/L)	log K_{OW}	log K_{OA}	Log K_{OC}	Reference[b]
C_{18-20} liquid LCCPs	$2 \times 10^{-8} - 5 \times 10^{-4}$ (40 – 52% Cl)	0.021 – 54.8 (34 – 54% Cl)	0.017 – 6.1 (34 – 54% Cl)	7.34 – 7.57 (34 – 54% Cl)	9.21 – 12.12[c] (34 – 54% Cl)	-	4, 10, 11, 12,
$C_{>20}$ liquid LCCPs	3×10^{-15} - 2.7×10^{-3} (40 – 54% Cl)	0.003 (50% Cl)	$1.6 \times 10^{-6} - 6.6$ (41.9 – 50% Cl)	7.46 – 12.83 (42 – 49% Cl)	-	-	4, 5, 6, 9, 10, 12, 13
$C_{>20}$ solid LCCPs	$1 \times 10^{-23} - 3 \times 10^{-14}$ (70% Cl)	$3.6 \times 10^{-7} - 5.6 \times 10^{-6}$ (70 – 71.3% Cl)	$1.6 \times 10^{-11} -$ 5.9 (70 – 71.3% Cl)	-	-	-	4, 5, 12

[a] Vapour pressure values not given at a consistent temperature.

[b] References: 1. Drouillard et al. (1998a), measured data; 2. Drouillard et al. (1998b), estimated data; 3. Sijm and Sinnige (1995), measured data; 4. BUA (1992), estimated data; 5. Madeley et al. (1983a), measured data; 6. Renberg et al. (1980), thin-layer chromatography (TLC) – K_{OW} correlation; 7. Fisk et al. (1998a), measured data; 8. U.K. Environment Agency (2003), measured data; 9. Campbell and McConnell (1980a), measured data; 10. BUA (1989), measured data; 11. Sijm and Sinnige (1995), estimated data; 12. U.K. Environment Agency (2001), estimated data; 13. Howard et al. (1975), estimated data; 14. Drouillard (1996), measured and estimated; 15. Thompson et al. (1998), measured.

[c] Octanol–air partition coefficients, estimated from ratio of K_{OW}/HLC (unitless).

3. ENTRY INTO THE ENVIRONMENT

3.1. Production, importation and use pattern

Canadian production and usage data for CPs were collected by means of a Notice, issued pursuant to section 71 of CEPA 1999, that was published in the *Canada Gazette* (Environment Canada 2003a). CPs are no longer produced in Canada (Camford Information Services, 2001). Pioneer Chemicals Inc. (formerly ICI Canada), Cornwall, Ontario, was the only Canadian producer of CPs. However, this plant was recently sold to Dover Chemical Corporation and it is currently not producing chlorinated paraffins. This Cornwall plant previously produced MCCPs and LCCPs with a chlorine content of up to 56% under the trade name Cereclor (Camford Information Services 2001). The capacity for production was 5.0, 5.0, 8.5 and 8.5 kilotonnes in 1997, 1998, 1999 and 2000, respectively; the corresponding imports to Canada in these years were 2.0, 2.0, 1.7 and 1.8 kilotonnes, respectively.

Total reported annual usage of CPs in Canada (production + imports – exports) was approximately 3,000 tonnes in 2000 and 2001 (Environment Canada 2003a). Whether the amount in use is the same at present is not known. North American demand for CPs fluctuates depending on the strength of the economy (Camford Information Services 2001).

Canadian use pattern data were obtained in two ways in the Notice issued pursuant to section 71 of CEPA 1999 (Environment Canada 2003a); distributors of CPs reported their sales volumes and intended usages for their customers, and users of CPs also reported on

how they use CPs and the end uses for products that they formulate. There were some differences in reported usage volumes for CPs by distributors and users, but the uses generally were in agreement.

Nearly all usage of SCCPs was reported to be in metalworking applications. Minor uses included use as a flame retardant in plastics and rubber.

The majority of uses for MCCPs as reported by distributors were in plastics and as lubricating additives. Minor uses were as an additive in sealants and caulking, in rubber and paints, and as a flame retardant in plastics or rubber.

The major uses of LCCPs are in lubricating additives, metalworking fluids and paints. Minor uses were in plastics and as flame retardants, engine oil, fabric adhesive and rock drilling fluid. Additional information on uses is available in the supporting document (Environment Canada 2008).

3.2. Releases to the environment in Canada

There is currently no evidence of any significant natural source of CPs (U.K. Environment Agency 2003). Anthropogenic releases of CPs into the environment may occur during production, storage, transportation, industrial and consumer usage of CP-containing products, disposal and burning of waste, and land filling of products (Tomy et al. 1998a).

The two major sources of release of SCCPs, MCCPs and LCCPs into the Canadian environment are likely use in metalworking applications and manufacturing of products containing these CPs. The possible sources of releases to water from manufacturing include spills, facility wash-down and storm water runoff. CPs in metalworking/metal cutting fluids may also be released into aquatic environments from drum disposal, carry-off and spent bath use (Government of Canada 1993a). These releases are collected in sewer systems and ultimately end up in the effluents of sewage treatment plants.

Other releases could be associated with use of gear oil packages, fluids used in hard rock mining and equipment use in other types of mining, fluids and equipment used in oil and gas exploration, manufacture of seamless pipe, metalworking and operation of turbines on ships (CPIA 2002; Environment Canada 2003b).

Landfilling is a major disposal route for polymeric products in Canada. CPs would be expected to remain stabilized in these products, with minor losses to washoff from percolating water. Leaching from landfill sites is likely to be negligible owing to strong binding of CPs to soils. Minor emissions of these products, which are effectively dissolved in polymers, could occur for centuries after disposal (IPCS 1996).

Polymer-incorporated CPs could also be released during recycling of plastics, which may involve processes such as chopping, grinding and washing. If released as dust from these

operations, the CPs would be adsorbed to particles because of high sorption and octanol–air partition coefficients.

Another significant source of release of CPs to the environment is from losses during the service life of products containing CP polymers (PVC, other plastics, paints, sealants, etc.) (European Commission, 2000; U.K. Environment Agency 2003). These releases are predicted to be mainly to urban/industrial soil and to wastewater.

3.2.1 National Pollutant Release Inventory (NPRI) data

Since 1999, on-site environmental releases of CPs (alkanes, C10-13, chloro; alkanes, C6-18, chloro) in Canada must be reported to the National Pollutant Release Inventory (NPRI) by companies meeting the reporting criteria. Based on information collected by the NPRI, very small amounts of CPs are being released to the Canadian environment by companies that meet the NPRI reporting requirements. In 2002, small transfers of short-chain CPs for disposal to landfill (1.45 tonnes) and recycling by recovery of organics (1.94 tonnes) have been reported to the NPRI from only two companies, both located in Ontario. Less than 5 kg of releases and/or transfers of $C_{6–18}$ CPs have been reported by a third company in Ontario. In 2001, the same three companies mentioned above reported similar quantities of releases/transfers of CPs to the NPRI. It should be noted, however, that CPs are likely to be released from sources other than the industrial sectors included in the NPRI, and releases to the Canadian environment could thus be considerably higher than those reported to this inventory.

4. RISK ASSESSMENT OF ECOLOGICAL IMPACTS

4.1. Environmental fate

4.1.1 SCCPs

Level III fugacity modelling of SCCPs has shown that they would achieve their highest concentrations in sediment and soil (Muir et al. 2001).

4.1.2 MCCPs/LCCPs

The environmental distribution of three MCCPs (C_{14-17}) and a liquid C_{18} LCCPs was estimated using the Equilibrium Criterion (EQC) Level III fugacity model of Trent University's Canadian Environmental Modelling Centre (Mackay et al. 1996). Level III represents a steady-state, non-equilibrium system comprised of soil, sediment, air and water compartments, with the chemical undergoing reactions or inputs and removal processes (advection, volatilization, deposition, photolysis, hydrolysis and biodegradation). Inputs of 100 kg/hour to soil, 1.6–6.4 kg/hour to air and 2.2–8.8 kg/hour to surface water were designed to reflect potential emissions of CPs mainly associated with landfills, land application of sewage sludge and consumer uses. Results from the

Level III EQC model suggest that these CPs would achieve their highest concentrations in sediment and soil. Concentrations in water and air were extremely low for all compounds. The environmental residence time of the three $C_{16–18}$ CPs were estimated to be greater than 500 days compared with 250 days for the C_{14} CP. However, these results should be viewed with caution because the degradations rates, used as input parameters for the CPs, were highly uncertain. Similar results were obtained using a Level III fugacity calculation with a $C_{14–17}$ MCCPs (U.K. Environment Agency 2003).

4.2. Persistence and bioaccumulation potential

When evaluating persistence in this section, the focus is on sediment as results of fugacity modelling indicate that this is an important compartment for all CPs. Persistence in air is also evaluated, because of the potential for long-range transport in this medium. Although soil is potentially an important compartment for CPs, there are too few data available to permit meaningful evaluation of persistence in soil.

4.2.1 SCCPs

Table 2 summarizes persistence and bioaccumulation information for SCCPs in comparison with criteria of the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

Table 2. Summary of persistence and bioaccumulation information on SCCPs.

Medium or parameter	CEPA criteria [1]	SCCPs Information
Air	$t_{1/2} \geq 2$ days *or* it is subject to atmospheric transport from its source to a remote area	Estimated $t_{1/2}$ of many SCCPs are ≥ 2 days SCCPs have been detected in air, sediment and biota in the Arctic in the absence of significant sources, indicating long range transport
Sediment	$t_{1/2} \geq 1$ year	Back calculation using concentrations from sediment cores shows half-life >1 year. Biodegradation test following OECD standard methods indicates half-lives >1 year in aerobic and anaerobic freshwater and marine sediments.
Soil	$t_{1/2} \geq 6$ months	Limited evidence for rapid biodegradation or removal following sludge applications
BAF	≥5000	Field BAFs >5000 in sculpin, smelt and trout; BMF values approaching or >1; Modified Gobas Model predicts BAF >5000 for some SCCPs
BCF	≥5000	BCFs>5000 in trout and mussels.
Log K_{OW}	≥5	4.39 – 8.69 (measured and modeled)

[1] Government of Canada 2000

A- Persistence

Air and Long-Range Transport

Estimated atmospheric half-lives for SCCPs based on reaction with hydroxyl radicals range from 0.81 to 10.5 days, using the default atmospheric hydroxyl radical concentration of 1.5×10^6 molecules/cm^3 during sunlight hours in AOPWIN (v. 1.86) computer program (Meylan and Howard, 1993; Atkinson 1986, 1987). Using a lower hydroxyl radical concentration of 5×10^5 molecules/cm^3, which is generally used as a daily (24-hour) average in relatively unpolluted air in the EU, atmospheric half-lives ranged from 1.2 to 15.7 days. Tomy (1997) also estimated atmospheric half-lives of greater than 2 days for the major SCCPs detected in the Great Lakes and Arctic air and biota.

SCCPs have vapour pressures (VPs) (2.8×10^{-7} to 0.028 Pa) and Henry's Law Constants (HLCs) (0.68–17.7 Pa·m^3/mol for $C_{10–12}$ congeners) that are in the range of VPs and HLCs for some persistent organic pollutants that are known to undergo long-range atmospheric transport under the 1979 UNECE Convention on Long Range Transboundary Air Pollution (e.g., hexachlorocyclohexane [lindane], heptachlor, mirex).[1] The HLC values imply partitioning from water to air or from moist soils to air, depending on environmental conditions and prevailing concentrations in each compartment.

SCCPs were detected in four air samples collected at Alert at the northern tip of Ellesmere Island in the high Arctic. Concentrations ranged from <1 to 8.5 pg/m^3 in gas-phase samples. Borgen et al. (2000) measured SCCPs in Arctic air samples taken at Mt. Zeppelin, Svalbard, Norway, in 1999. Concentrations ranging from 9.0 to 57 pg/m^3 were detected. Borgen et al. (2002) found much higher SCCPs concentrations in air at Bear Island, a small isolated island between Svalbard and mainland Norway. Total SCCPs concentrations ranged from 1,800 to 10,600 pg/m^3. SCCPs residues were found in the surficial sediments in three remote Arctic lakes including Yaya Lake, Hazen Lake and Lake DV-09. Concentrations ranged from 0.0016 to 0.0176 mg/kg dry wt. (Tomy et al. 1998a; Stern and Evans 2003).

SCCPs have been found at concentrations ranging from 0.095 to 0.626 mg/kg wet wt. in the blubber of marine mammals, including beluga (*Delphinapterus leucas*), ringed seal (*Phoca hispida*) and walrus (*Odobenus rosmarus*) from several locations in the Arctic (Tomy et al. 1998b;2000). Tomy et al. (2000) observed that the concentration profiles for the Arctic marine mammals show a predominance of the shorter carbon chain length congeners, i.e., the C_{10} and C_{11} formula groups. Drouillard et al. (1998a) showed that these congeners are the more volatile components of SCCPs mixtures, which show a trend of decreasing VPs with increasing carbon chain length and degree of chlorination. Reth et al. (2006) measured SCCPs in liver and muscle from seabirds (little auk and kittiwake) collected at Bear Island (European Arctic). Concentrations ranged from 0.005 to 0.088 mg/kg wet weight.

Estimated atmospheric half-lives of many SCCPs are greater than 2 days for a large percentage (61% using hydroxyl radical concentration of 1.5×10^6 molecules/cm^3 and 83% using hydroxyl radical concentration of 5×10^5 molecules/cm^3) of example

[1] The VP of lindane is 4.3×10^{-3} Pa (IPCS 1991), the VP of heptachlor is 3.0×10^{-6} Pa (IPCS 1984a) and the VP of mirex is 2.3×10^{-9} Pa (IPCS 1984b). The HLCs of lindane and heptachlor are 0.13 and 0.02 Pa·m^3/mol, respectively.

structures. Therefore, SCCPs meet the CEPA 1999 half-life criterion for persistence in air specified in the Persistence and Bioaccumulation Regulations (Government of Canada 2000). The detection of the more volatile shorter carbon chain length congeners of SCCPs in Arctic biota and in Arctic lake sediments in the absence of significant sources of SCCPs in this region suggests that these residues are present due to long-range atmospheric transport.

On the basis of the available information, it is concluded that estimated atmospheric half-lives of SCCPs exceed the criterion of 2 days and SCCPs are subject to long-range atmospheric transport. Hence, SCCPs are persistent in air according to the criteria stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

Sediments and Soils

There is limited evidence for the biodegradation or removal of SCCPs from soil following sewage sludge application. Nicholls et al. (2001) did not detect SCCPs/MCCPs in farm soils amended with sludges containing mg/kg concentrations of CPs. However, worms living in these same soils did contain low mg/kg wet wt. levels of CPs.

Using 25-day biochemical oxygen demand (BOD) tests, Madeley and Birtley (1980) found that SCCPs composed of 49% chlorine appeared to be rapidly and completely degraded by acclimatized micro-organisms after 25 days. However, no significant oxygen uptake was observed in tests using the highly chlorinated CPs, which included two SCCPs (60% and 70% chlorine). On the other hand, Fisk et al. (1998a) found that two ^{14}C-labelled C_{12} chloro-n-alkanes (56% and 69% chlorine) were degraded at 12°C in aerobic sediments used for a study of the bioavailability of SCCPs to oligochaetes. Half-lives in sediment were 12 ± 3.6 days and 30 ± 2.6 days for the 56% and 69% chlorine products, respectively.

A study on the aerobic and anaerobic biodegradation of SCCPs in both freshwater and marine sediments was undertaken by Thompson and Noble (2007). Using ^{14}C-labelled n-decane and n-tridecane 65% chlorine by weight products and basing their experiments on the OECD 308 Test Guideline (aerobic and anaerobic transformation in aquatic sediment systems), mineralization (as measured by carbon dioxide or methane production) over 98 days was determined. The mean half-lives for mineralization for a C_{10}-$_{13}$, 65% chlorine by weight product, calculated as the average for the two individual products, were estimated to be 1630 days in freshwater sediments and 450 days in marine sediments under aerobic conditions. Little or no mineralization was noted in anaerobic sediments. It should be noted that these half-lives were calculated based on degradation observed after the 40-50 day lag phase, and that the half-lives were extrapolated beyond the available data.

SCCPs residues were found in the surficial sediments of the following remote Arctic lakes (reported in mg/kg dry wt.): Yaya Lake (0.0016), Hazen Lake (0.0045) and Lake

DV09 (0.0176). The profile from Lake DV09 generally follows the pattern of historical usage of SCCPs (Stern and Evans 2003). Concentration profiles of SCCPs in sediments from Lake Winnipeg (Manitoba), Fox Lake (Yukon Territory), the west basin of Lake Ontario (Ontario) and Lake DV09 (Devon Island, Nunavut) indicate that SCCPs residues were present in the 1940s (Muir et al. 1999a; Tomy et al. 1999). The highest concentration in Lake Ontario (800 ng/g dry wt.) was observed in the slice dated at 1971 (Muir et al. 1999a).

In the absence of information on loading for any of the years at any of these locations, it is not possible to calculate discrete half-life values from these data for comparison with the criteria for persistence in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000). However, the fact that SCCPs residues were detected in sediment cores dating back to the 1940s at these locations is evidence that SCCPs can persist for more than 50 years in subsurface anaerobic sediments. Environment Canada (2008) used first order decay equations in a back calculation method to determine that SCCPs have a half-life in sediments longer than 1 year. The equation used for these calculations are standard first order decay equations.

Several government assessments and published reviews have concluded that only slow biodegradation in sediment may be expected to occur, even in the presence of adapted micro-organisms (Government of Canada 1993a,b; Tomy et al. 1998a; European Commission, 2000). On the basis of the available information, it is thus concluded that SCCPs are persistent in sediments according to the criterion stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

B- Bioaccumulation

Bioaccumulation factors (BAFs) for SCCPs chain length groups in Lake Ontario plankton, alewife (*Alosa pseudoharengus*), slimy sculpin (*Cottus cognatus*), rainbow smelt (*Osmerus mordax*) and lake trout (*Salvelinus namaycush*) were determined based on a whole organism (wet weight) and filtered water concentrations using data from Houde et al. (2006). SCCPs were found in all components of the food chain and BAFs ranged from 9,900 to 51,200 (wet weight). SCCPs bioaccumulated to the greatest extent in fish, with the highest BAFs (51,200) in sculpin, smelt and trout. Assuming no metabolism, the Modified Gobas BAF model for fish estimated BAF values greater than 5000 for all possible SCCPs (Arnot and Gobas 2003).

Bioconcentration factors (BCFs) calculated from laboratory studies for SCCPs have been reviewed in Government of Canada (1993b) and were found to vary dramatically among different species. Relatively low BCF values have been determined in freshwater and marine algae (<1–7.6). BCF values of up to 7816 wet wt. have been measured in rainbow trout (*Oncorhynchus mykiss*) (Madeley and Maddock 1983a,b) and 5785–138 000 wet wt. in the common mussel (*Mytilus edulis*) (Madeley et al. 1983b, Madeley and Thompson 1983d, Renberg et al. 1986).

Other evidence that SCCPs are bioaccumulative is as follows:

- Reported log Kow values for SCCPs range from 4.39 – 8.69 (Table 1).
- Lipid normalized biomagnification factors (BMFs) were also determined by Houde et al. (2006) for pelagic food webs in both Lakes Ontario and Michigan. BMFs ranged from 0.3 to 3.2. While biomagnification factors (BMF) are not a parameter considered in the Persistence and Bioaccumulation Regulations (Government of Canada 2000), BMFs are important supplemental information. If a substance has a BMF greater than one, it is more likely to have high BCF/BAF values.
- Concentrations of SCCPs in fish collected around the Great Lakes between 1996 and 2001 ranged from 0.0046 to 2.63 mg/kg wet weight (Muir et al. 2001; and Houde et al. 2006). SCCPs have also been detected in the blubber of belugas from the St. Lawrence River at an average concentration of 0.785 mg/kg wet wt. (Tomy et al. 1998b; 2000) and blubber of ringed seal from several Arctic locations. Concentrations in these mammals from the Arctic and the St. Lawrence River ranged from 0.095 to 0.626 mg/kg wet wt. (Jansson et al. 1993; Tomy et al. 1998b; 2000). These relatively high concentrations suggest that SCCPs have the potential to bioaccumulate in aquatic organisms.
- Tomy (1997) found that SCCPs (around 60–70% chlorine by weight) were present at a concentration of 0.011–0.017 mg/kg lipid (mean concentration 0.013 mg/kg lipid) in human breast milk from Inuit women living on the Hudson Strait in northern Quebec, Canada. These findings are indicative of bioaccumulation through the food chain since food would be the major or only source of environmental exposure for the Inuit.

On the basis of the available information, it is concluded that SCCPs are bioaccumulative substances according to the criteria stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

4.2.2 MCCPs

Table 3 summarizes persistence and bioaccumulation information for MCCPs in comparison with criteria in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

Table 3. Summary of persistence and bioaccumulation information on MCCPs.

Medium or parameter	CEPA criteria [1]	MCCPs Information
Air	$t_{1/2} \geq 2$ days	Estimated to be 2.7–7.1 days for vapor phase, but it should be noted that the extent of partitioning for MCCPs to air is low Degradation rate on airborne particles likely to be much slower
Sediment	$t_{1/2} \geq 1$ year	Back calculation using concentrations from sediment cores shows half-life >1 year
Soil	$t_{1/2} \geq 6$ months	Limited evidence for rapid biodegradation or removal following sludge applications

Medium or parameter	CEPA criteria [1]	MCCPs Information
BAF	≥5000	Field BAFs for fish >5000 in Lake Ontario; high BMFs found in laboratory studies and a food web study in Lake Ontario and Lake Michigan; Modified Gobas Model predicts BAF>5000 for all congeners
BCF	≥5000	Laboratory BCFs <5000; however, the BCF was probably underestimated due to CP concentrations exceeding solubility
Log K_{OW}	≥5	5.47–8.21 (measured and modeled)

[1] Government of Canada 2000

A- Persistence

Air

Atmospheric half-lives for MCCPs were calculated using the Syracuse Research Corporation AOPWIN (v. 1.91) program using a hydroxyl radical concentration of 5×10^5 molecules/cm^3. Half-lives for vapour phase MCCPs ranged from 2.7 to 7.1 days, with the longest half-lives for MCCPs with the highest chlorine contents and also with the shorter chain lengths. However, MCCPs have very low partitioning to air.

MCCPs have estimated VP (4.5×10^{-8} to 2.27×10^{-3} to Pa at 20–25°C) and HLC (0.014 – 51.3 Pa·m^3/mol for $C_{14\text{-}17}$ congeners) values that are in the range of VPs and HLCs for some persistent organic pollutants that are known to undergo long-range atmospheric transport under the 1979 UNECE Convention on Long Range Transboundary Air Pollution, such as lindane, heptachlor and mirex.

On the basis of the available information, it is concluded that estimated atmospheric half-lives of MCCPs exceed the criterion of 2 days and hence are persistent in air according to the criteria stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

Sediment and Soil

There is limited evidence for the biodegradation or removal of MCCPs from soil following sewage sludge application. Nicholls et al. (2001) did not detect SCCPs/MCCPs in farm soils amended with sludges containing mg/kg concentrations of CPs. However, worms living in these same soils did contain low mg/kg wet wt. levels of CPs.

Concentrations of total MCCPs in a sediment core from Lake St. Francis, downstream of Cornwall, Ontario, ranged from 0.75 to 1.2 mg/kg dry wt, with the highest concentrations estimated to have been deposited in 1972 (Muir et al. 2002). Environment Canada (2008) used first order decay equations in a back calculation method to determine that MCCPs have a half-life in sediments longer than 1 year. The equation used for these calculations are standard first order decay equations. Moreover, the fact that MCCPs residues were detected in sediment cores dating back to the 1970s at these locations is evidence that

SCCPs can persist for more than 30 years in subsurface anaerobic sediments. Persistence in sediment is particularly important as Level III fugacity calculations show that MCCPs are expected to partition primarily to sediment and soil.

On the basis of the available information, it is concluded that MCCPs are persistent in sediments according to the criteria stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

B- Bioaccumulation

Bioaccumulation factors (BAFs) for MCCPs chain length groups in Lake Ontario alewife (*Alosa pseudoharengus*), slimy sculpin (*Cottus cognatus*), rainbow smelt (*Osmerus mordax*) and lake trout (*Salvelinus namaycush*) were determined based on a whole organism (wet weight) and filtered water concentrations collected in 2001 using data in Houde et al. (2006). $C_{14\text{-}15}$ MCCPs were found in all components of this food chain and BAFs ranged from 9.99×10^6 to 7.15×10^8. In addition, bioaccumulation factors (BAF) for 21 MCCPs congeners using the Modified Gobas BAF Model (assuming no metabolism) were all above the bioaccumulative criteria (≥5000 BAF) (Arnot and Gobas 2003).

Most of the laboratory-based BCF studies conducted on aquatic organisms may underestimate the true BCF, because the studies were performed at MCCPs concentrations above the water solubility limit, using acetone as the co-solvent in the test solutions, and hence are not in compliance with OECD guideline requirements. Estimated BCF values for common mussel, bleak and rainbow trout (32-2856) are all below the BCF criterion of 5000 (Madeley et al. 1983b; Madeley and Maddock 1983a; Bengtsson et al. 1979; Madeley and Thompson 1983a), except for one common mussel study which reported a BCF of 6920 (Renberg et al. 1986). The only BCF study that did not use acetone reported BCFs values of 349 to 1087 for rainbow trout following the OECD test method 305 (Thompson et al. 2000).

Other evidence that MCCPs are bioaccumulative is as follows:

- Reported log Kow values for MCCPs range from 5.47 – 8.21 (Table 1).
- Lipid normalized biomagnification factors (BMFs) were also determined by Houde et al. (2006) between *Diporeia* and sculpin in Lake Ontario and Lake Michigan. BMFs ranged from 1 to 15. Large BMFs were observed for these species for all chain lengths in Lake Ontario, and for C_{14} in Lake Michigan, indicating biomagnification. BMFs (2.4 – 7.7) were also above 1 for smelt and lake trout in Lake Michigan. In laboratory studies with rainbow trout and oligochaetes, lipid-normalized equilibrium BMFs estimated from a first-order bioaccumulation model for constant dietary exposure (Bruggeman et al. 1981) ranged from 0.4-5.0 (Fisk et al. 1996; 1998b;2000). While biomagnifications factors (BMF) are not a criterion considered in the Persistence and Bioaccumulation Regulations (Government of Canada 2000), BMFs are

important supplemental information. If a substance has a BMF greater than one, it is more likely to have high BCF/BAF values.

- Oligochaetes were found to have biota-sediment accumulation factors (BASFs) ranging from 0.6 to 4.4 (Fisk et al. 1998a). These BASFs, reflecting bioaccumulation from sediment at levels above that expected at equilibrium, imply significant food chain transfer.
- Elevated levels of MCCPs were found in catfish from the Detroit River (0.904 mg/kg wet wt.), and in crab and mussel (up to 38.7 mg/kg lipid wt.) located near a CPs manufacturing plant in Australia (Tomy and Stern 1999; Kemmlein et al. 2002). Kemmlein et al. (2002) stated: "Bioaccumulation is clearly evident, the mussel meat containing around double and crab meat around six times the amount of chloroparaffins found in the most contaminated sediment sample."
- MCCPs have been found in a breast milk sample (0.061 mg/kg lipid) from the United Kingdom (Thomas and Jones 2002), and $C_{10–20}$ CPs were detected in liver, adipose and kidney tissues from human cadavers at up to 1.5 mg/kg wet wt. (Campbell and McConnell 1980a). These findings qualitatively indicate potential for bioaccumulation of MCCPs through the human food chain.

On the basis of the available information, and in particular the field BAF estimates, it is concluded that MCCPs are bioaccumulative substances according to the criteria stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

4.2.3 LCCPs

4.2.3.1 C18–20 liquid LCCPs

Table 4 summarizes persistence and bioaccumulation information for $C_{18\text{-}20}$ liquid LCCPs in comparison with criteria in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

Table 4: Summary of persistence and bioaccumulation information on $C_{18\text{-}20}$ LCCPs.

Medium or parameter	CEPA criteria 1	$C_{18\text{-}20}$ LCCPs Information
Air	t1/2 ≥ 2 days	Estimated to be 2.4–10.5 days but it should be noted that the extent of partitioning to air for LCCPs is low
Sediment	t1/2 ≥ 1 year	Unknown, but half-life likely > 1 year
Soil	t1/2 ≥ 6 months	Unknown
BAF	≥5000	Laboratory diet studies suggest highly chlorinated C18 has high BMF from food; insufficient information on field BAFs; Modified Gobas Model finds nearly half of the C18-20 congeners examined have BAF≥5000 (see section 4.4.3.3.)
BCF	≥5000	Laboratory BCFs <5000; BCF probably underestimated due to CP concentrations exceeding solubility
Log K_{OW}	≥5	7.34 – 7.57 (modeled)

[1] Government of Canada 2000

A- Persistence

Air

Atmospheric half-lives for liquid LCCPs were calculated using the Syracuse Research Corporation AOPWIN (v. 1.91) program using a hydroxyl radical concentration of 5×10^5 molecules/cm^3. Half-lives for liquid LCCPs ranged from 2.4 to 10.5 days, with many example structures having half-lives greater than 2 days. However, $C_{18\text{-}20}$ liquid LCCPs have very low partitioning to air.

$C_{18\text{-}20}$ liquid LCCPs have estimated VP (5×10^{-4} to 2×10^{-8} Pa at 25 ˚C) values that are in the range of VPs for some persistent organic pollutants that are known to undergo long-range atmospheric transport under the 1979 UNECE Convention on Long Range Transboundary Air Pollution, such as lindane, heptachlor and mirex.

On the basis of the available information, it is concluded that estimated atmospheric half-lives of $C_{18\text{-}20}$ liquid LCCPs exceed the criterion of 2 days and hence are persistent in air according to the criteria stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

Sediment and Soil

There is no empirical information available on the fate (i.e., half-lives) of LCCPs in soil or sediment with which to compare with the CEPA criteria. However, given that both SCCPs and MCCPs are expected to be persistent in sediment (half lives > 1 year), and that resistance to microbial degradation has been observed to generally increase with increases in carbon chain length (Allpress and Gowland 1999; Omori et al. 1987), it is likely that LCCPs also have half lives of more than 1 year in sediment.

On the basis of the available information, it is concluded that $C_{18\text{–}20}$ liquid LCCPs are persistent in sediments according to the criteria stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

B- Bioaccumulation

Assuming no metabolism the Modified Gobas BAF Model predicts that 12 out of 27 (44%) $C_{18\text{-}20}$ congeners meet the bioaccumulation criteria of BAF ≥5000 (Arnot and Gobas 2003). As confirmed by personal communication with Frank Gobas (Simon Fraser University, Burnaby, BC), the model is applicable for LCCPs, as they are simple hydrophobic and persistent chemicals.

On the other hand, BCF values for $C_{18\text{-}26}$ liquid LCCPs were estimated by U.K. Environmental Agency (2001), using the data of Bengtsson et al. (1979) and were found to range from 8 to 16 in bleak; these values are below the BCF criterion of 5000

(Government of Canada 2000). However, this study may underestimate the true BCF values, because the study was performed at LCCPs concentrations above the solubility limit for water and hence was not in compliance with OECD guidelines. As well, the study did not indicate if steady state was reached during the uptake phase of the test.

Other evidence that LCCPs are bioaccumulative is as follows:

- Reported log Kow values for $C_{18–20}$ liquid LCCPs range from 7.34 – 7.57 (Table 1).
- Biomagnification factors (BMFs) were determined by Fisk et al (2000) in a dietary accumulation study involving juvenile rainbow trout exposed to $C_{18}H_{31}Cl_7$. Lipid normalized BMFs ranged from 2.1 to 2.8. While biomagnifications factors (BMF) are not a criterion considered in the Persistence and Bioaccumulation Regulations (Government of Canada 2000), BMFs are important supplemental information. If a substance has a BMF greater than one, it is more likely to have high BCF/BAF values.
- Fisk et al. (2000) also found that $C_{18}H_{31}Cl_7$ has similar biotransformation half-lives in rainbow trout compared to half-lives of recalcitrant organochlorines (Fisk et al. 1998c). This suggests limited biotransformation or metabolism.
- Limited biotransformation of LCCPs was also observed during an uptake/elimination study with bleak. Bengtsson and Baumann-Ofstad (1982) found that a $C_{18–26}$ LCCPs had a low uptake efficiency, but 50% of this compound remained in the fish tissues after a 316-day elimination period, which suggests that some of the LCCPs isomers in this formulation are bioaccumulative (Bengtsson and Baumann-Ofstad 1982).
- Elevated levels of $C_{18–29}$ LCCPs were found in crab and mussel (9.3 and 14.3 mg/kg lipid wt., respectively) located near a CPs manufacturing plant in Australia (Kemmlein et al. 2002). Kemmlein et al. (2002) stated: "Bioaccumulation is clearly evident, the mussel meat containing around double and crab meat around six times the amount of chloroparaffins found in the most contaminated sediment sample." However it is unclear if bioaccumulation of $C_{18\text{-}20}$ or $C_{>20}$ congeners was responsible for the elevated concentrations.

On the basis of the available information, and in particular the BAF model and empirical BMF estimates, it is concluded that $C_{18–20}$ liquid LCCPs are bioaccumulative substances according to the criteria stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

4.2.3.2 C>20 liquid LCCPs

Table 5 summarizes persistence and bioaccumulation information for $C_{>20}$ liquid LCCPs in comparison with criteria in the CEPA 1999 Persistence and Bioaccumulation Regulations (Government of Canada 2000).

Table 5. Summary of persistence and bioaccumulation information on $C_{>20}$ liquid LCCPs.

Medium or parameter	CEPA criteria [1]	$C_{>20}$ liquid LCCPs Information
Air	$t_{1/2} \geq 2$ days	Estimated to be 1.8–8.4 days but it should be noted that the extent of partitioning to air for LCCPs is low
Sediment	$t_{1/2} \geq 1$ year	Unknown, but half life likely > 1 year
Soil	$t_{1/2} \geq 6$ months	Unknown
BAF	≥5000	Insufficient information on field BAFs; Modified Gobas Model finds none of the $C_{>20}$ congeners examined have BAF≥5000
BCF	≥5000	Laboratory BCFs <5000; BCF probably underestimated due to CP concentrations exceeding solubility
Log K_{OW}	≥5	>7.46 – 12.83 (estimated)

[1] Government of Canada 2000

A- Persistence

Air

Atmospheric half-lives for liquid LCCPs were calculated using the Syracuse Research Corporation AOPWIN (v. 1.91) program using a hydroxyl radical concentration of 5×10^5 molecules/cm^3. Half-lives for liquid LCCPs ranged from 1.8 to 8.4 days, with many example structures having half-lives greater than 2 days. However, LCCPs have very low partitioning to air.

$C_{>20}$ liquid LCCPs have estimated VPs (5×10^{-5} to 3×10^{-15} Pa at 25 ˚C) that are in the range of VPs for some persistent organic pollutants that are known to undergo long-range atmospheric transport under the 1979 UNECE Convention on Long Range Transboundary Air Pollution, such as heptachlor and mirex.

On the basis of the available information, it is concluded that estimated atmospheric half-lives of $C_{>20}$ liquid LCCPs exceed the criterion of 2 days and hence are persistent in air according to the criteria stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

Sediment and Soil

There is no empirical information available on the fate (i.e., half-lives) of LCCPs in soil or sediment with which to compare with the CEPA criteria. However, given that both SCCPs and MCCPs are expected to be persistent in sediment (half lives > 1 year), and that resistance to microbial degradation has been observed to generally increase with increases in carbon chain length (Allpress and Gowland 1999; Omori et al. 1987), it is likely that LCCPs also have half lives of more than 1 year in sediment.

On the basis of the available information, it is concluded that $C_{>20}$ liquid LCCPs are persistent in sediments according to the criteria stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

B- Bioaccumulation

Although $C_{>20}$ liquid LCCPs may have some potential to bioaccumulate, the Modified Gobas BAF Model predicts that none of the $C_{>20}$ congeners meet the bioaccumulation criteria of BAF ≥5000. Thus, these very high molecular weight LCCPs are not expected to be bioaccumulative.

BCF values were found to range from 8-16 for $C_{18\text{-}26}$ liquid LCCPs in bleak, and 18-1158 for liquid $C_{>20}$ LCCPs in rainbow trout and common mussel (Madeley and Maddock 1983b; Bengtsson et al. 1979; Madeley and Thompson 1983b; U.K. Environment Agency 2001). However, these values may underestimate the true BCF values, because the studies were performed at LCCPs concentrations above the solubility limit for water and hence were not in compliance with OECD guidelines. As well, the studies did not indicate if steady state was reached during the uptake phase of the tests. BCF values for these species were below the BCF criterion of 5000.

On the other hand there is some evidence to suggest that $C_{>20}$ LCCPs may be bioaccumulative:

- Reported log Kow values for $C_{>20}$ liquid LCCPs range from 7.46 – 12.83 (Table 1).
- Limited biotransformation of LCCPs was also observed during an uptake/elimination study with bleak. Bengtsson and Baumann-Ofstad (1982) found that a $C_{18–26}$ LCCPs had a low uptake efficiency, but 50% of this compound remained in the fish tissues after a 316-day elimination period, which suggests that some of the LCCPs isomers in this formulation are bioaccumulative (Bengtsson and Baumann-Ofstad 1982).
- Elevated levels of $C_{18–29}$ LCCPs were found in crab and mussel (9.3 and 14.3 mg/kg lipid wt., respectively) located near a CPs manufacturing plant in Australia (Kemmlein et al. 2002). Kemmlein et al. (2002) stated: "Bioaccumulation is clearly evident, the mussel meat containing around double and crab meat around six times the amount of chloroparaffins found in the most contaminated sediment sample." However it is unclear if bioaccumulation of $C_{18\text{-}20}$ or $C_{>20}$ congeners was responsible for the elevated concentrations.
- $C_{20–30}$ CPs were detected in fat and liver of some postmortem human tissues from the United Kingdom at concentrations between 0.080 and 3.5 mg/kg wet wt. These findings qualitatively indicate the potential for bioaccumulation of LCCPs in the human food chain.

Although there are noteable uncertainties, based mainly on the available BAF information, it is concluded that $C_{>20}$ liquid LCCPs are not bioaccumulative substances according to the criteria stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

4.2.3.3 C>20 solid LCCPs

Table 6 summarizes persistence and bioaccumulation information for $C_{>20}$ solid LCCPs in comparison with criteria in the CEPA 1999 Persistence and Bioaccumulation Regulations (Government of Canada 2000).

Table 6. Summary of persistence and bioaccumulation information on $C_{>20}$ solid LCCPs.

Medium or parameter	CEPA criteria [1]	$C_{>20}$ solid LCCPs Information
Air	$t_{1/2} \geq 2$ days	Estimated to be ≥7.8 days but it should be noted that the extent of partitioning to air for LCCPs is low
Sediment	$t_{1/2} \geq 1$ year	Unknown, but half life likely > 1 year
Soil	$t_{1/2} \geq 6$ months	Unknown
BAF	≥5000	Low accumulation by salmon; poor absorption and high excretion via feces by rats; Modified Gobas Model predicts BAF <5000
BCF	≥5000	Laboratory BCFs <5000; BCF probably underestimated due to CP concentrations exceeding solubility
Log K_{OW}	≥5	Unknown

[1] Government of Canada 2000

A- Persistence

Air

Atmospheric half-lives for solid LCCPs were calculated using the Syracuse Research Corporation AOPWIN (v. 1.91) program using a hydroxyl radical concentration of 5×10^5 molecules/cm^3. Half-lives for solid $C_{18\text{-}25}$ LCCPs ranged from 7.8 to 15.5 days. However, LCCPs have very low partitioning to air.

On the basis of the available information, it is concluded that estimated atmospheric half-lives of $C_{>20}$ solid exceed the criterion of 2 days and hence are persistent in air according to the criteria stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

Sediment and Soil

No soil or sediment half-life data exist for the $C_{>20}$ solid LCCPs. However, given that both SCCPs and MCCPs are expected to be persistent in sediment (half lives > 1 year), and that resistance to microbial degradation has been observed to generally increase with

increases in carbon chain length (Allpress and Gowland 1999; Omori et al. 1987), it is likely that LCCPs also have half lives of more than 1 year in sediment.

On the basis of the available information, it is concluded that $C_{>20}$ solid LCCPs are persistent in sediments according to the criteria stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

B- Bioaccumulation

Although $C_{>20}$ LCCPs may have some potential to bioaccumulate, the Modified Gobas BAF Model predicts that none of the $C_{>20}$ congeners meet the bioaccumulation criterion of BAF ≥5000. Thus, these very high molecular weight LCCPs are not expected to be bioaccumulative.

Measured BCF values for solid LCCPs were found to range from 5.7 to 341 in fish and common mussels (Madeley and Maddock 1983c, Madeley and Thompson 1983c). However, these studies may underestimate the true BCF values, because the studies were performed at LCCPs concentrations above the solubility limit for water and hence were not in compliance with OECD guidelines. As well, the studies did not indicate if steady state was reached during the uptake phase of the tests. Estimated BCF values for these species were below the BCF criterion of 5000 (Madeley and Maddock 1983b,c; Bengtsson et al. 1979; Madeley and Thompson 1983b,c).

Log Kow values are not available for $C_{>20}$ solid LCCPs.

Other evidence that $C_{>20}$ LCCPs may not be bioaccumulative is as follows:

- One aquatic BAF study was identified for $C_{>20}$ solid LCCPs. Zitko (1974) observed very low accumulation of a 70% chlorine LCCPs by juvenile Atlantic salmon fed a diet that had high CP concentrations (10 and 100 µg/g) during a 181-day exposure period.
- Two rat bioaccumulation studies with LCCPs, including $C_{>20}$ solid LCCPs, showed high rates of excretion via feces and poor absorption of the LCCPs, Section 4.4.3.2, supporting document (Environment Canada, 2008).. No BMF data exist for $C_{>20}$ solid LCCPs.

Although there are noteable uncertainties, based on the available information it is concluded that $C_{>20}$ solid LCCPs are not bioaccumulative substances according to the criteria stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

4.3. Environmental Concentrations

This section describes the results of recent monitoring of CPs in environmental samples using analytical techniques having higher specificity for SCCPsS. Data on environmental levels of MCCPs and LCCPs are very limited. Due to the non-volatile and hydrophobic characteristics of these CP groups, the majority of results are for sediments and sewage sludges.

Data presented in this section focus on Canadian concentrations. In situations where Canadian data are lacking or are few, concentrations measured in other countries are presented. Additional information on ambient concentrations may be found in the Supporting Document (Environment Canada, 2008).

4.3.1 Atmospheric concentrations

SCCPs were detected in air in Canada, United Kingdon and Norway. They have also been detected in arctic air and in air of other remote areas (Section 4.2.1). Concentrations of SCCPs in air samples collected at Egbert, Ontario, Canada, in 1990 ranged from 65 to 924 pg/m^3 (Tomy 1997; Tomy et. al. 1998a). Concentrations of SCCPs over Lake Ontario in 1999 and 2000 ranged from 120 to 1,510 pg/m^3 (Muir et al. 2001).

No atmospheric concentration data are available for MCCPs and LCCPs, either in Canada or elsewhere.

4.3.2 Wastewater treatment effluents, sewage sludge and soils

SCCPs were detected in wastewater effluents in Canada, the United States and Germany. SCCPs were detected in all eight sewage treatment plant final effluents from southern Ontario, Canada, sampled in 1996. Total SCCPs (dissolved and particulate C_{10-13}) ranged from 59 to 448 ng/L. The highest concentrations were found in samples from treatment plants in industrialized areas, including Hamilton, St. Catharine's and Galt. No wastewater treatment effluent concentration data are available for MCCPs and LCCPs, either in Canada or elsewhere.

Concentrations of CPs have also been detected in sewage sludge in several European countries and the United States. Nicholls et al. (2001) found total CPs (SCCPs + MCCPs) concentrations in digested sewage sludge ranging from 1.8 to 93.1 mg/kg dry wt. in England and Wales. Similarly, Stevens et al. (2002) found SCCPs concentrations ranging from 6.9 to 200 mg/kg dry wt. in sewage sludge from 14 WWTPs in the United Kingdom. Highest concentrations of SCCPs were in sludge from industrial catchments. However, a rural catchment with zero industrial effluent had significant levels (590 mg/kg) of total SCCPs/MCCPs in sludge (Stevens et al. 2002). Total concentrations of MCCPs in sewage sludges from 15 WWTPs in the United Kingdom ranged from 30 to 9,700 mg/kg dry wt. (Stevens et al. 2002). Agricultural soils may also be a potentially major reservoir of CPs due to sewage sludge application (Stevens et al. 2002; Nicholls et al. 2001). No values in sewage sludge or soil were identified for LCCPs. Concentrations of CPs in sewage sludge in Canada are not available.

4.3.3 Surface waters

SCCPs were detected in surface waters in Canada and the United Kingdom. Low levels of dissolved total (C_{10-13}) SCCPs were measured in western Lake Ontario between 1999 and 2004 (Muir et al. 2001, Houde et al. 2006). The concentration of total SCCPs was 1.75 ng/L in 1999. Concentrations of total SCCPs ranged from 0.606 – 1.935 ng/L over the 2000 – 2004 sampling period. Concentrations were generally greater in western Lake Ontario, likely due to the proximity of large urban areas (Houde et al. 2006). SCCPs concentrations of 30 ± 14 ng/L were measured in the Red River in Selkirk, Manitoba, over a 6-month period in 1995 (Tomy 1997).

MCCPs were detected in surface waters in Canada, the United States, the United Kingdom and Germany. Metcalfe-Smith et al. (1995) reported $C_{14–17}$ MCCPs concentrations in a 24-hour composite sample of effluent from the only manufacturing plant in Canada, ICI Canada (now PCI Canada), on the St. Lawrence River at Cornwall, Ontario, to be 12,700 ng/L. This plant is not currently manufacturing CPs. Houde et al. (2006) collected water samples from various sites in Lake Ontario in 2002 and 2004. Total MCCPs concentrations ranged from <0.0005 to 0.0026 ng/L in filtered samples. Concentrations of MCCPs in an impoundment ditch that received effluent from a CP production plant in Dover, Ohio, were <150 – 3,800 ng/L (Murray et al. 1988). MCCPs were found in all the samples taken in 16 rivers, canals and reservoirs in the United Kingdom (ICI 1992). Concentrations ranged from 620 to 3,750 ng/L. The majority of the samples appear to have been collected in urban/industrial areas. Levels of MCCPs have been measured at several sites in Germany (Hoechst AG 1987; Ballschmiter 1994). The levels measured in 1987 ranged from 4,000 to 20,000 ng/L while those of 1994 were substantially lower and ranged from < 50 to 185 ng/L.

There are no Canadian measured water concentrations of LCCPs and very few measurements of LCCPs in surface waters from other countries. Nicholls et al. (2001) reported <100 ng/L of any CP group in all sites near sewage treatment works in the United Kingdom except for one (Darwen, U.K.). Only one study was identified measuring surface water concentrations of LCCPs. Murray et al. (1988) conducted a study near a CPs production plant in Dover, Ohio, reporting total concentrations of $C_{20–30}$, 40–50% chlorine LCCPs of 8,300 ng/L in the middle of the impoundment lagoon at this site. In a drainage ditch leading from the impoundment lagoon, a concentration of 4,200 ng/L total LCCPs (3,700 ng/L particulate, 500 ng/L dissolved) was measured just above its discharge to Sugar Creek. A concentration of 620 ng/L particulates (<50 ng/L dissolved) was found in water from Sugar Creek, just downstream of the outlet of the drainage ditch.

4.3.4 Sediments

SCCPs were detected in sediments around the Great Lakes, St. Lawrence River, and other lakes in Canada, as well as in Germany, Czech Republic and the United Kingdom. They have also been detected in arctic sediment (Section 4.2.1). Concentrations of SCCPs in Lake Winnipeg and Lake Nippigon ranged from 0.008 to 0.176 mg/kg dry wt. (Tomy et al. 1999; Stern and Evans 2003). Tomy et al. (1997) measured SCCPs at concentrations

around 0.245 mg/kg dry wt. in sediment from the mouth of the Detroit River at Lake Erie and Middle Sister Island in western Lake Erie, in 1995. SCCPs were also detected in all surface sediment samples from harbour areas along Lake Ontario at concentrations ranging from 0.0059 to 0.290 mg/kg dry wt. in 1996 (Muir et al. 2001). The highest concentrations were found at the most industrialized site (Windermere Basin, Hamilton Harbour), which has well-documented heavy metal, PAH and PCB contamination. Similarly, Marvin et al. (2003) reported a SCCPs concentration of 0.410 mg/kg dry wt. in Lake Ontario sediments near an industrialized area. SCCPs were detected in all 26 samples from Lake Ontario, and the average SCCPs concentration was 0.049 mg/kg dry wt., which is much higher than sediment concentrations reported for lakes (Yaya, DV09, Hazen, Nipigon) influenced primarily by atmospheric sources (Tomy et al. 1999; Stern and Evans 2003).

MCCPs were detected in sediments around the Great Lakes in Canada, as well as in the United States, Germany, Wales, Switzerland, Australia and the United Kingdom. Metcalfe-Smith et al. (1995) were unable to detect (<3.5 mg/kg dry wt.) SCCPs + MCCPs in sediments from the St. Lawrence River downstream of a CP manufacturing plant. Tomy and Stern (1999) reported concentrations of $C_{14–17}$ MCCPs of 0.068 mg/kg dry wt. in sediment samples collected near the mouth of the Detroit River in western Lake Erie. Muir et al. (2002) reported concentrations of total MCCPs in a sediment core from Lake St. Francis downstream of Cornwall, Ontario, of 0.75 –1.2 mg/kg dry wt. The highest concentrations of MCCPs detected in sediments were found downstream from sewage treatment works in the United Kingdom. Concentrations of MCCPs ranged from <0.2 to 65.1 mg/kg dry wt. (Nicholls et al. 2001). Similar concentrations were found at several other locations downstream from sewage treatment plants in the United Kingdom (Nicholls et al. 2001).

No LCCPs were measured in sediments in Canada, but they have been detected in the United States, Australia and Germany near CP manufacturing plants. Concentrations of LCCPs in these countries ranged from 0.0081 to 170 mg/kg dry wt. (Rotard et al. 1998; Murray et al. 1988; Kemmlein et al. 2002).

4.3.5 Biota

A- Aquatic Biota

SCCPs were detected in biota in Canada, England, Norway, Chile, Greece, Germany, Iceland, France, the United States, and the North and Baltic Seas. Muir et al. (2001) and Houde et al. (2006) measured SCCPs in fish collected from Lake Ontario and Lake Michigan, between 1996 and 2001. Concentrations of total SCCPs ranged from 0.0046 to 2.63 mg/kg wet wt. The highest concentration was measured in carp collected at Hamilton harbour (Muir et al. 2001). Houde et al. (2006) determined the concentration of SCCPs in plankton, *Diporeia* sp. and *Mysis* sp. from Lakes Ontario and Michigan. In Lake Ontario, total SCCPs concentrations in plankton, *Diporeia* and *Mysis* were 0.0055, 0.0063, and 0.0028 mg/kg wet wt., respectively, and in Lake Michigan they were 0.023, 0.024, and 0.0075 mg/kg wet wt., respectively.

MCCPs have been measured in fish in Canada, the United Kingdon, Norway, Chile, Greece and Germany amongst others. Houde et al. (2006) also measured the concentrations of MCCPs in fish in Lake Ontario and Lake Michigan in 1999 and 2001. Concentrations of total MCCPs ranged from 0.0028 to 0.109 mg/kg weight wt. MCCPs were also detected in *Diporeia* at concentrations ranging from 0.0024 to 0.0041 mg/kg (Houde et al. 2006). The highest concentration in fish measured in Canada was 0.904 mg/kg weight wt. for catfish in the Detroit River (Tomy and Stern 1999).

Murray et al. (1988) measured concentrations of $C_{20–30}$, 42% chlorine LCCPs in zebra mussels from Sugar Creek, Ohio, near a CPs manufacturing plant. Concentrations ranged from <0.007 upstream to 0.18 mg/kg downstream of where the drainage ditch from the plant emptied into Sugar Creek. Kemmlein et al. (2002) found high levels of $C_{18–29}$ LCCPs in marine mussels and crabs (9.3 and 14.3 mg/kg lipid wt., respectively) near a CPs manufacturing plant in Australia.

B- Marine Mammals

SCCPs have been detected in the blubber of belugas from the St. Lawrence River at an average concentration of 0.785 mg/kg weight wt. SCCPs have also been detected in the blubber of ringed seal from southwest Ellesmere Island (Eureka), Pangnirtung (Cumberland Sound) and Svalbard; in beluga whales from northwest Greenland, Sanikiluaq (Hudson Bay), Pangnirtung (Cumberland Sound), Kimmirut and the Mackenzie Delta; and in walrus from northwest Greenland. Concentrations of SCCPs from these areas ranged from 0.095 to 0.626 mg/kg weight wt. (Jansson et al. 1993; Tomy et al. 1998b; 2000).

Concentrations of MCCPs in beluga blubber in the St-Lawrence ranged from 1.8 – 80.0 mg/kg weight wt. (Bennie et al. 2000). However, results obtained by Bennie et al. (2000) may not be reliable due to interferences in the analytical method.

C- Terrestrial and Avian Wildlife

Very limited information is available on SCCPs concentrations in tissues of terrestrial wildlife. In Sweden, Jansson et al. (1993) reported CP concentrations (unspecified chain length) in rabbit (Revingeshed, Skåne), moose (Grismsö, Västmanland), reindeer (Ottsjö, Jaämtland) and osprey (from various regions in Sweden) to be 2.9, 4.4, 0.14 and 0.53 mg/kg lipid wt., respectively. Nicholls et al. (2001) reported the concentrations of SCCPs and MCCPs in earthworms residing in fields on which sludge had been applied ranging from <0.1 to 0.7 mg/kg dry wt. in the United Kingdom in the summer of 1998. Campbell and McConnell (1980a) determined levels of $C_{10–20}$ CPs in birds in the United Kingdom. The $C_{10–20}$ levels were likely to be dominated by contributions from the SCCPs and MCCPs. Concentrations of $C_{10–20}$ CPs ranged from 0.1 to 1.2 mg/kg weight wt. in liver of birds and from <0.05 to >6 mg/kg in seabird eggs. Concentrations of $C_{20–30}$ CPs ranged from not detected to 1.5 mg/kg weight wt. in liver of birds and from <0.05 to 1 mg/kg in seabird eggs. Reth et al. (2006) quantified SCCPs in liver and muscle from

the seabirds, little auk (*Alle alle*) and kittiwake (*Rissa tridactyla*) collected at Bear Island (European Arctic). Concentrations between 0.005 and 0.088 mg/kg wet weight were measured. Reth et al. (2006) determined the concentration of C_{14-15} MCCPs in seabirds from the European Arctic. Concentrations ranged from 0.005 to 0.370 mg/kg wet wt.

4.4. Environmental effects

Overall, toxicity studies are few for effects of SCCPs to pelagic biota and mammals. LOECs (i.e., survival, reproduction and growth) ranged from 8,900 to 10,000 ng/L for pelagic biota. Effects of SCCPs to benthic and soil-dwelling organisms are not available. More toxicological data are available for MCCPs. In particular, the acute and chronic toxicity of MCCPs has been studied in algae, invertebrates and several species of fish. The range of acute effects is 5,900 ng/L to > 10g/L (10 000 000 000 ng/L). LOECs for pelagic biota ranged from 18,000 to 31,000 ng/L. Contrary to SCCPs, toxicity studies, albeit few, are available for benthic and soil-dwelling organisms. LOECs for sediment-dwelling biota ranged from 270 to 410 mg/kg dry weight. A reproductive LOEC for earthworm was reported to be 383 mg/kg dry weight. Few studies are available for effects of MCCPs to mammals; LOAELs ranged from 4.2 to 5.7 mg/kg bw/day for effects to rats. Similarly, limited number of studies is available for effects to pelagic biota. Acute effects were observed at greater than 3 800 000 ng/L. Very few toxicological data are available for the three types of LCCPs. These data are presented below.

This section will focus on the most sensitive toxicological information used to derive the critical toxicity values (CTV) only. Additional toxicity information is available in the supporting document.

4.4.1 SCCPs

A- Pelagic aquatic organisms

The lowest toxic effect level identified for a pelagic freshwater aquatic species is 8,900 ng/L, which is the 21-day chronic LOEC for *Daphnia magna* (Thompson and Madeley 1983a). The effect was for mortality of the offspring. The NOEC is 5,000 ng/L.

B- Benthic organisms

A valid measurement endpoint was not available for a sediment-dwelling invertebrate. As a result, an equilibrium partitioning approach (Di Toro et al. 1991) using the most sensitive chronic measurement endpoint identified for a pelagic freshwater invertebrate aquatic species (8,900 ng/L) was used to estimate the toxicity to benthic organisms. The $LOEC_{benthic}$ was estimated to be 35.5 mg/kg dry wt. for sediment containing 2% organic carbon (Environment Canada, 2008).

C- Soil-dwelling organisms

Bezchlebová et al. (2007) investigated the effects of SCCPs on the survival and reproduction of five species of soil organisms (*Fosomia candida, Eisenia fetida, Enchytraeus albidus, Enchytraeus crypticus,* and *Caenorhabditis elegans*). All tests were preformed following international methods, using an OECD artificial soil (70% sand, 20% clay, 10% peat) with an organic carbon content of approximately 2.7%. *Folsomia candida* (collembola) was identified as the most sensitive organism, with an LC_{50} value for adult survival and EC_{50} and EC_{10} values for reproduction of 5733, 1230, and 660 mg/kg dry wt. (nominal), respectively. The soil CTV for SCCPs is 660 mg/kg dry wt.

D- Mammals

In a 13-week oral (gavage) rat study by IRDC (1984), increases in liver and kidney weight and hypertrophy of the liver and thyroid occurred at doses of 100 mg/kg-bw per day. This value was the most sensitive LOAEL for mammals. Interspecies scaling using data for a typical adult otter was used to extrapolate to a food concentration for this species. This resulted in a CTV of 1,000 mg/kg food. See Table 7 of this report and Appendix 2 of supporting document for additional information (Environment Canada, 2008).

4.4.2 MCCPs

A- Pelagic aquatic organisms

In a 21-day chronic study with *Daphnia*, Thompson et al. (1997) reported a LOEC of 18,000 ng/L and a NOEC of 10,000 ng/L for a decrease in the number of live offspring and the length of the parent organisms. This LOEC is the most sensitive toxicity value for aquatic organisms.

B- Benthic organisms

The most sensitive value for sediment toxicity of MCCPs is the LOEC for growth from a 28-day study with the amphipod *Hyalella azteca* using sediment that contained 5% organic carbon (Thompson et al. 2003). A statistically significant ($p = 0.05$) reduction in the mean dry weights of survivors in the treatment groups was seen at exposure concentrations of 270 mg/kg dry wt. and above when compared with the solvent control.

C- Soil-dwelling organisms

The most sensitive toxicity value for terrestrial organisms is the chronic (28-day) LOEC

of 383 mg/kg dry wt. in soil with an organic carbon content of 2%, for reproduction in earthworms (Thompson et al. 2001a).

D- Mammals

The lowest effect level observed for mammals is the LOAEL of 4.2 mg/kg-bw per day for mild effects on the kidney and thyroid of female rats during a 13-week feeding study (Poon et al. 1995). Interspecies scaling using data for a typical adult otter was used to extrapolate to a food concentration for this species. This resulted in a CTV of 42 mg/kg food. See Table 7 of this report and Appendix 2 of supporting document for additional information.

4.4.3 LCCPs

4.4.3.1 LCCPs (C18-20 liquid)

A- Pelagic aquatic organisms

A chronic 21-day *Daphnia magna* study was carried out by Frank (1993) and Frank and Steinhäuser (1994). The most sensitive aquatic toxicity value for liquid $C_{18\text{-}20}$ LCCPs is the 21-day (chronic) LOEC of 68,000 ng/L.

B- Soil-dwelling organisms

There are no studies available on the toxicity of either liquid or solid LCCPs to terrestrial plants, earthworms or other soil-dwelling organisms. Therefore, an equilibrium partitioning approach (Di Toro et al. 1991) using the most sensitive measurement endpoint identified for a pelagic freshwater species (68,000 ng/L) was used to estimate the toxicity of liquid $C_{18\text{-}20}$ LCCPs to soil-dwelling organisms . The $LOEC_{soil}$ for $C_{18\text{-}20}$ LCCPs was estimated to be 2,035 mg/kg dry wt. for a soil containg 2% organic carbon (Environment Canada, 2008).

4.4.3.2 LCCPs (C>20 liquid)

There is no relevant exposure or toxicity data available for $C_{>20}$ liquid LCCPs in pelagic organisms, benthic organisms, or soil dwelling organisms.

A- Mammals

In 90-day and 2-year feeding studies with rats with $C_{>20}$ (43% chlorine by weight)

LCCPs, the lowest LOAEL in the studies was 100 mg/kg-bw per day (Serrone et al. 1987; Bucher et al. 1987; NTP 1986). This LOAEL was the most sensitive toxicity value. The main effects were seen on the liver, and in both studies effects were seen at the lowest concentrations. Interspecies scaling using data for a typical adult otter will be used to extrapolate to a food concentration for this species. This resulted in a CTV of 1,000 mg/kg food. See Table 7 of this report and Appendix 2 of supporting document for additional information.

4.4.3.3 LCCPs (C>20 solid)

There is no relevant exposure or toxicity data available for $C_{>20}$ solid LCCPs in pelagic organisms, benthic organisms, or soil dwelling organisms.

A- Mammals

Serrone et al. (1987) reported a LOAEL for hepatic lesions in female rats following administration by gavage of another long-chain CP ($C_{20–30}$, 43% chlorine) during a 90-day study. In addition, mild nephrosis was observed in the kidneys of male rats, as was mineralization in the kidneys of female rats administered 3750 mg/kg-bw per day. A NOEL could not be established for the females (LOEL = 100 mg/kg- food). Interspecies scaling using data for a typical adult otter will be used to extrapolate to a food concentration for this species. This resulted in a CTV of 100 mg/kg food. See Table 7 of this report and Appendix 2 of supporting document for additional information.

4.5. Potential to cause ecological harm

Potential to cause environmental harm may be estimated quantitatively using risk quotients (RQs). When RQs exceed 1 (i.e., in this case when Estimated Exposure Values (EEVs) exceed Estimated No-Effect Values (ENEVs)) this is an indication of potential for risk.

It is acknowledged, however, that when risks for persistent and bioaccumulative substances - such as SCCPs, MCCPs, and C_{18-20} LCCPs - are determined using standard methods, the risks may be underestimated. For example, since it can take decades for persistent substances to achieve maximum steady state concentrations in sediment and soil, EEVs based on monitoring data will be too low if steady state concentrations have not been achieved in these media. Similarly, since it can take a long time for persistent and bioaccumulative substances to reach maximum steady state concentrations in the tissues of laboratory organisms, ENEVs based on standard toxicity tests may underestimate effect thresholds if test durations are insufficient to achieve maximum internal organism concentrations. Furthermore, since food consumption is usually the primary route of exposure to persistent and bioaccumulative substances in the field – especially for top predators - ENEVs may underestimate effect thresholds if the food pathway is not considered in key toxicity studies. These factors are exacerbated when

available effects and exposure data are limited, as is the case for the chlorinated paraffins.

Risk quotients were calculated for SCCPs, MCCPs, C_{18-20} LCCPs and $C_{>20}$ LCCPs (Table 7). For each identified class of risk receptors (e.g., pelagic organisms, benthic organisms), an EEV was selected based on empirical data. The maximum reported field concentration which is relevant to the Canadian environment was used as the EEV. Chemical concentrations from the Canadian environment were preferably used for EEVs; however, data from other regions in the world were used in the absence of suitable Canadian data. Section 8.2 of the supporting document (Environment Canada, 2008) further discusses this point. An ENEV was determined by dividing a Critical Toxicity Value (CTV) by an assessment factor. CTVs, a detailed description is provide in Section 8.0 of the supporting document (Environment Canada, 2008), typically represent the lowest chronic ecotoxicity value from an available and acceptable data set. Assessment factors were used to reduce the CTV to account for extrapolation from a sometimes limited set of effects data for laboratory organisms, to estimates of effect thresholds for sensitive species in the field. Note that an extra assessment factor was not used to account for the tendency for conventional RQs to underestimate potential for harm for persistent and bioaccumulative substances. Results are summarized in Table 7.

Concentrations of C_{18-20} liquid LCCPs in sediments representative of Canadian environments are not available. In addition, no toxicity data were available for the effects of C_{18-20} liquid LCCPs on secondary consumers. Therefore, risk quotients could not be calculated for exposure of benthic organisms and secondary consumers to C_{18-20} liquid LCCPs. Furthermore there are no relevant exposure and toxicity data available for $C_{>20}$ liquid and $C_{>20}$ solid LCCPs in pelagic organisms, benthic organisms, or soil dwelling organisms. As such, risk quotients were not calculated for these groups.

Table 7. List of Estimated Exposure Values (EEV), Critical Toxicity Values (CTV), Assessment Factors (AF), and Estimated No Exposure Values (ENEV) used in the calculation of Risk Quotients (RQ) for SCCPs, MCCPs, C18-20 liquid LCCPs, C>20 liquid LCCPs and C>20 solid LCCPs.

Organism	EEV	CTV	AF	ENEV	RQ (EEV/ENEV)
SCCPs					
Pelagic	44.8[a] ng/L	8,900[b] ng/L	10 (lab/field)	890 ng/L	0.05
Benthic	0.41[c] mg/kg	35.5[d] mg/kg	10 (lab/field)	3.55 mg/kg	0.12
Soil-dwelling	0.64[e]mg/kg	660[d] mg/kg	10 (lab/field)	66.0 mg/kg	0.01
Secondary Consumer	2.63[f] mg/kg	1,000[g] mg/kg food	100 (lab/field & species variations)	10 mg/kg	0.26
MCCPs					
Pelagic	0.0026[h] ng/L	18,000[i] ng/L	10 (lab/field)	1,800 ng/L	0.0000014
Benthic	65.1[j] mg/kg	270[k] mg/kg	10 (lab/field)	27 mg/kg	2.40
Soil-dwelling	31.0[l] mg/kg	383[m] mg/kg	10 (lab/field)	38.3 mg/kg	0.81
Secondary Consumer	0.904[n] mg/kg	42[o] mg/kg food	100 (lab/field & species variations)	0.42 mg/kg	2.15
C_{18-20} liquid LCCPs					
Pelagic	100[p] ng/L	68,000[q] ng/L	10 (lab/field)	6,800 ng/L	0.02

Soil-dwelling	3.1[r] mg/kg	2,035[s] mg/kg	10 (lab/field)	203.5 mg/kg	0.02
$C_{>20}$ liquid LCCPs					
Secondary Consumer	0.0465[t] mg/kg	1,000[u] mg/kg	10 (lab/field)	100 mg/kg	0.0005
$C_{>20}$ solid LCCPs					
Secondary Consumer	0.0465[v] mg/kg	100[w] mg/kg	10 (lab/field)	100 mg/kg	0.000465

[a] The highest concentration of SCCPs observed in final effluent of sewage treatment plants in southern Ontario was 448 ng/L at the Woodward Avenue sewage treatment plant in Hamilton, Ontario. A dilution factor of 10 was used to calculate the EEV which results in an EEV of 44.8 ng/L.

[b] 21-day LOEC for *Daphnia magna*.

[c] Highest concentration in surface sediments observed from Lake Ontario, Niagara (or west) basin, in 1998.

[d] EC_{10} for *F. candida* reproduction.

[e] The maximum allowable rate for sewage biosolid application to agricultural lands is 8 tonnes of solids per hectare per 5 years (MOE 1998). The soil mass is 5,000 tonnes/ha (assuming that the biosolids are incorporated into the top 20 cm of the soil having a dry soil bulk density of 2500 kg/m^3 (EU 2003)). Using a SCCPs concentration in sewage sludge of 200 mg/kg dry wt. and assuming that SCCPs-containing sludge is applied to the land for 10 years and that no or little biodegradation of the SCCPs occurs, a soil concentration of 0.64 mg/kg dry wt is estimated.

[f] Concentration of total SCCPs found in carp from Hamilton Harbour in Lake Ontario.

[g] The LOAEL for the 13-week oral (gavage) rat study is 100 mg/kg bw/day (IRDC 1984). Interspecies scaling using this LOAEL for a typical adult otter (*lutra canadensis*) (adult body weight of 8 kg and average daily food ingestion rate of 0.8 kg wet wt. per day) was used to extrapolate to a food concentration for this species (CCME 1998). The resulting CTV was 1,000 mg/kg food wet wt.

[h] Concentration measured in Lake Ontario.

[i] 21-day LOEC for *Daphnia magna.*

[j] Concentration found downstream from sewage treatment works in the United Kingdom.

[k] 28-day LOEC for growth for the amphipod *Hyalella azteca.*

[l] The maximum allowable rate for sewage biosolid application to agricultural lands is 8 tonnes of solids per hectare per 5 years (MOE 1998). The soil mass is 5,000 tonnes/ha (assuming that the biosolids are incorporated into the top 20 cm of the soil having a dry soil bulk density of 2500 kg/m^3 (EU 2003)). Using a MCCPs concentration in sewage sludge of 9,700 mg/kg dry wt. and assuming that SCCPs-containing sludge is applied to the land for 10 years and that no or little biodegradation of the SCCPs occurs, a soil concentration of 31 mg/kg dry wt is estimated.

[m] 28-day LOEC in soil with an organic carbon content of 2% for reproduction in earthworms.

[n] Concentration of MCCPs in catfish collected from the Detroit River, Michigan, and southern Ontario.

[o] The LOAEL for the 13-week oral (gavage) rat study is 4.2 mg/kg bw/day (Poon et al. 1995). Interspecies scaling using this LOAEL for a typical adult otter (*lutra canadensis*) (adult body weight of 8 kg and average daily food ingestion rate of 0.8 kg wet wt. per day) was used to extrapolate to a food concentration for this species (CCME 1998). The resulting CTV was 42 mg/kg food wet wt.

[p] Detection limit for sewage treatment works in the United Kingdom.

[q] 21-day LOEC for *Daphnia magna.*

[r] The maximum allowable rate for sewage biosolid application to agricultural lands is 8 tonnes of solids per hectare per 5 years (MOE 1998). The soil mass is 5,000 tonnes/ha (assuming that the biosolids are incorporated into the top 20 cm of the soil having a dry soil bulk density of 2500 kg/m^3 (EU 2003)). Using a MCCPs concentration in sewage sludge of 9,700 mg/kg dry wt. (worst-case concentration in the absence of exposure data for $C_{18\text{-}20}$ liquid LCCPs) and assuming that SCCPs-containing sludge is applied to the land for 10 years and that no or little biodegradation of the SCCPs occurs, a soil concentration of 31 mg/kg dry wt is estimated. Since LCCPs usage is about 10% of MCCPs usage, this would result in a $C_{18\text{-}20}$ liquid LCCPs soil concentration of 3.1 mg/kg dry wt.

[s] Value calculated using an equilibrium partitioning approach using the *Daphnia magna* LOEC.

[t] $C_{18–29}$ CPs in mussel near a manufacturing plant in Australia had a lipid wt. concentration of 9.3 mg/kg (Kemmlein et al. 2002). Using an average lipid content of zebra mussels and other North American exotic mussel species in the Great Lakes of 0.5% wet wt. (Marvin 2003), and assuming that the Australian mussel had a similar lipid content to zebra mussels and that all of the LCCPs measured in the Australian mussel were of the $C_{>20}$ liquid type, the concentration of LCCPs in mussels was estimated to be 0.0465 mg/kg on a wet wt.

[u] The LOAEL for a 90-day (Serrone et al. 1987) and 2-year feeding (Bucher et al. 1987) studies with rats with $C_{>20}$ (43% chlorine by weight) LCCPs is 100 mg/kg bw/day. Interspecies scaling using this LOAEL for a typical adult otter (*lutra canadensis*) (adult body weight of 8 kg and average daily food ingestion rate of 0.8 kg wet wt. per day) was used to extrapolate to a food concentration for this species (CCME 1998). The resulting CTV was 1,000 mg/kg food wet wt.

[v] $C_{18–29}$ CPs in mussel near a manufacturing plant in Australia had a lipid wt. concentration of 9.3 mg/kg (Kemmlein et al. 2002). Using an average lipid content of zebra mussels and other North American exotic mussel species in the Great Lakes of 0.5% wet wt. (Marvin 2003), and assuming that the Australian mussel had a similar lipid content to zebra mussels and that all of the LCCPs measured in the Australian mussel were of the $C_{>20}$ solid type, the

concentration of LCCPs in mussels was estimated to be 0.0465 mg/kg on a wet wt.

[w] Effects were seen in the liver and kidney of rats at a concentration of 3750 mg/kg-bw per day in a 90-day dietary study (Serrone et al. 1987). The LOAEL for a 90-day dietary study with rats was 3,750 mg/kg bw/day (Serrone et al. 1987). Interspecies scaling using this LOAEL for a typical adult otter (*lutra canadensis*) (adult body weight of 8 kg and average daily food ingestion rate of 0.8 kg wet wt. per day) was used to extrapolate to a food concentration for this species (CCME 1998). The resulting CTV was 37,500 mg/kg food wet wt.

Only two of the 12 calculated risk quotients are larger than 1. The MCCPs risk quotient for benthic organisms (RQ=2.40) and the MCCPs risk quotient for secondary consumers (RQ=2.15) both suggest that MCCPs pose a risk to these receptors. However, because of limitations in available exposure and effects data mentioned above and explained in more detail in Section 8.2 of the supporting document, the absence of RQs above 1 for SCCPs and C_{18-20} LCCPs cannot be considered proof that these persistent and bioaccumulative substances do not cause ecological harm.

Because data available for $C_{>20}$ LCCPs are very limited, only one RQ could be calculated for each of the solid and liquid subgroups.. Although the resulting RQs are very low, this too is likely an underestimate of possible high-end risks, in part because of limitations in information on environmental concentrations close to relevant point sources (Section 8.2 of the supporting document).

Evidence that a substance is very persistent and bioaccumulative as defined in the Persistence and Bioaccumulation Regulations of CEPA 1999, when taken together with potential for environmental release and potential for toxicity to organisms, provides a significant indication of its potential to cause harmful long term ecological effects. Substances that are persistent remain in the environment for a long time, increasing the magnitude and duration of exposure. Releases of small amounts of bioaccumulative substances may lead to high internal concentrations in exposed organisms. Highly bioaccumulative and persistent substances are of special concern, since they may biomagnify in food webs, resulting in very high internal exposures, especially for top predators.

SCCPs, MCCPs and C_{18-20} LCCPs are considered to be both highly persistent and bioaccumulative. The limited available evidence suggests that although $C_{>20}$ LCCPs are persistent, they are not bioaccumulative.

In addition, there is evidence (including some monitoring data), that SCCPs, MCCPs and C_{18-20} LCCPs are released into the Canadian environment and have the potential to cause harm to sensitive aquatic organisms at relatively low concentrations (i.e., chronic NOECs for pelagic organisms < 100 ng/L).

In light of this evidence, it is concluded that SCCPs, MCCPs and LCCPs up to C_{20} may be causing long term ecological harm in Canada.

4.6. Uncertainties on the ecological risk assessment

This risk assessment contains several sources of uncertainty. Uncertainties in the

exposure and effects assessment can influence the characterization of risks. Below is a brief discussion of these uncertainties. Additonal details can be found in Section 8.2 of the supporting document.

4.6.1 Exposure, Effects and Risk Quotient Calculations

When Canadian exposure data were lacking, data from other countries were used as EEVs and assumed to be representative of Canadian conditions. Concentrations of CPs in various media were often only available for certain areas, and were only representative of a short time period, in Canada and other countries. As a result, it is unkown how concentrations of CPs vary temporally and spatially. Moreover, concentrations were often not available near potential point sources such as metalworking operations (primary source of CPs) and other formulating/manufacturing sites that use CPs.

Uncertainties with the toxicity information used to drive ENEVs in this assessment include:

- The use of an equilibrium partitioning approach to estimate toxicity to benthic and soil organisms for SCCPs and LCCPs.
- The lack of aquatic toxicity tests for $C_{>20}$ solid LCCPs, particularly with daphnids, a species that was found to be the most sensitive to SCCPs, MCCPs and liquid LCCPs.
- The use of test substance concentrations in excess of their water solubility for all fish toxicity tests.

Additional assessment factors were not used to account for these limitations when deriving ENEVs from CTVs.

Because of the above-mentioned limitations - and the fact that in general risks of persistent and bioaccumulative substances are likely to be underestimated using standard assessment approaches – ecological risks from exposure to SCCPs, MCCPs, $C_{18\text{-}20}$ LCCPs in Canada have likely been underestimated by risk quotient calculations, especially close to industrial sources. In the case of $C_{>20}$ LCCPs, limitations in the available exposure and effects data mean that risks to secondary consumers have likely been underestimated, and that risks to other types of organisms cannot be estimated at all.

4.6.2 Persistence and Bioaccumulation Status and Risk Implications

Information on physical properties of MCCPs, and especially LCCPs, is limited. Values used in this assessment are based on extrapolations mainly from SCCPs or QSARs. The analysis of SCCPs and MCCPs in sediment cores and associated calculations provide strong evidence for the persistence of these substances in the environment. Even though there are no data for persistence of LCCPs in sediment, based on biodegradation data which indicate increasing stability with increasing carbon chain length, it is reasonable to conclude that LCCPs are persistent in sediment.

The empirical and modelled bioaccumulation data for SCCPs and MCCPs are very robust and indicate the substances are bioaccumulative. While there is a lack of empirical

bioaccumulation data for LCCPs, the modelling results provided by the Modified Gobas BAF Model - which suggest that of all the LCCPs congeners only liquid C_{18-20} LCCPs have significant bioaccumulation potential – are considered credible.

Lastly, there are uncertainties associated with extrapolating from evidence that a substance is both persistent and bioaccumulative to a conclusion that it may be causing ecological harm. However, given that persistent and bioaccumulative substances have the potential to cause widespread harm that is difficult to reverse, a precautionary assessment approach is justified.

5. HUMAN HEALTH RISK ASSESSMENT

5.1. Population exposure

The following presentation is limited to identified recent data considered critical to quantitative estimation of exposure of the general population in Canada to chlorinated paraffins and, hence, to assessment of "toxic" under Paragraph 64(c) of CEPA 1999. Other sources of data that were also identified but were not directly relevant to estimation of exposure in Canada include Peters *et al*. (2000), Borgen *et al*. (2000, 2002) and Lahaniatis *et al*. (2000).

The degree of confidence in data on the concentrations of chlorinated paraffins in various media varies considerably, depending upon the nature of the analysis. To the extent possible, estimates of intake have been based on higher-confidence analyses by high-resolution gas chromatography (HRGC)/electron capture negative ion high-resolution mass spectrometry (ECNI-HRMS), due to its higher mass resolving power and selectivity. However, such information is limited solely to determination of SCCPs in human breast milk (Tomy, 1997), fish (Muir *et al*., 1999) and media that contribute less to human exposure, including ambient air (Tomy, 1997), surface water (Tomy, 1997) and sediment (Muir *et al*., 2001). For all chlorinated paraffins, either concentrations in surface water and sediment, or the limits of detection for these media, were used as surrogates for concentrations in drinking water and soil, respectively, in estimating intake.

Indeed, data on concentrations of chlorinated paraffins in foodstuffs are extremely limited. While additional data on the concentrations of SCCPs, MCCPs and LCCPs in foods in the United Kingdom (Campbell and McConnell, 1980b) reported in an early investigation reviewed in the PSL1 assessment (Campbell and McConnell, 1980a) were acquired and are presented in Table 8, they are considered, at best, to be semi-quantitative, owing to limitations of the methodology available at that time. Analysis was based on liquid–solid adsorption chromatography, which has now largely been replaced by micro-analytical techniques and quantification by visual reference to spots appearing on thin-layer chromatographic plates.

Table 8. Concentrations of short-chain, medium-chain and long-chain chlorinated paraffins in foodstuffs

Food group	Concentration used to represent food group	
	Short- and medium-chain chlorinated paraffins	**Long-chain chlorinated paraffins**
Dairy	0.3 µg/g mean of 13 samples of dairy products in U.K. $C_{10–20}$ (SCCPs and MCCPs) (Campbell and McConnell, 1980a)	0.19 µg/g 1 sample of cheese in U.K. $C_{20–30}$ (Campbell and McConnell, 1980a)
Fats	0.15 µg/g mean of 6 samples of vegetable oils and derivatives $C_{10–20}$ (SCCPs and MCCPs) (Campbell and McConnell, 1980a)	0.05 µg/g detection limit in analysis of 1 sample of lard in U.K. $C_{20–30}$ (Campbell and McConnell, 1980b)
Fruits	0.025 µg/g mean of 16 samples of fruits and vegetables in U.K. $C_{10–20}$ (SCCPs and MCCPs) (Campbell and McConnell, 1980a)	0.025 µg/g 1 sample of peach fruit in U.K. $C_{20–30}$ (Campbell and McConnell, 1980a)
Vegetables	0.025 µg/g mean of 16 samples of fruits and vegetables in U.K. $C_{10–20}$ (SCCPs and MCCPs) (Campbell and McConnell, 1980a)	0.025 µg/g 1 sample of potato crisps in U.K. $C_{20–30}$ (Campbell and McConnell, 1980a)
Cereal products	SCCPs 0.13 µg/g one reported concentration for "Chlorowax 500C" in enriched white bread in market basket survey carried out by U.S. Food and Drug Administration (KAN-DO Office and Pesticides Team, 1995); average molecular formula is $C_{12}H_{19}Cl_7$, with 60–65% chlorine content (w/w) (IPCS, 1996)	0.05 µg/g detection limit in analyses of corn flakes in U.K. $C_{20–30}$ (Campbell and McConnell, 1980b)
	SCCPs/MCCPs 0.05 µg/g detection limit in analysis of 1 sample of corn flakes in U.K. $C_{10–20}$ (SCCPs and MCCPs) (Campbell and McConnell, 1980b)	
Meat and poultry	0.099 µg/g 1 sample of bacon in U.K. $C_{10–20}$ (SCCPs and MCCPs) (Campbell and McConnell, 1980b)	0.05 µg/g detection limit in analysis of 1 sample each of ox liver and beef in U.K. $C_{20–30}$ (Campbell and McConnell, 1980b)

Food group	Concentration used to represent food group	
	Short- and medium-chain chlorinated paraffins	**Long-chain chlorinated paraffins**
Fish	Note: Campbell and McConnell (1980b) presented data for combined SCCPs and MCCPs. Data for fish identified in Bennie *et al*. (2000), Muir *et al*. (1999) and Tomy and Stern (1999) were presented as separate analyses.	no data identified
	SCCPs 2.630 µg/g (wet weight); analysis of whole samples of carp from Hamilton Harbour; C_{10}–C_{13} (Muir *et al*., 1999) 0.0588 µg/g; lake trout, Niagara-on-the-Lake (Muir *et al*., 1999) 0.0726 µg/g; lake trout, Port Credit (Muir *et al*., 1999) 0.502 µg/g; carp (n = 3) (Bennie *et al*., 2000) 1.47 µg/g; trout (n = 10) (Bennie *et al*., 2000) 1.8 µg/g (estimated); perch, Detroit River (Tomy and Stern, 2000)	
	MCCPs 1.23 µg/g; mean of 10 samples of whole trout from western Lake Ontario (Bennie *et al*., 2000) 0.393 µg/g; carp (n = 3) (Bennie *et al*., 2000) 82 ng/g in perch; 904 ng/g in catfish (Tomy and Stern, 1999) 0.008 µg/g (estimated); perch, Detroit River (Tomy and Stern, 2000)	
Eggs	no data identified	no data identified
Foods primarily sugar	0.025 µg/g 1 sample of strawberry jam in U.K. $C_{10–20}$ (SCCPs and MCCPs) (Campbell and McConnell, 1980b)	0.05 µg/g detection limit in 1 sample of strawberry jam in U.K. $C_{20–30}$ (Campbell and McConnell, 1980b)
Mixed dishes	no data identified	no data identified
Nuts and seeds	no data identified	no data identified
Soft drinks, alcohol, coffee, tea	0.05 µg/g detection limit in analyses of beverages in U.K. (Campbell and McConnell, 1980a)	0.05 µg/g detection limit in analysis of 1 sample each of beer and tea in U.K. $C_{20–30}$

Food group	Concentration used to represent food group	
	Short- and medium-chain chlorinated paraffins	Long-chain chlorinated paraffins
		(Campbell and McConnell, 1980b)

5.1.1 SCCPs

Tomy (1997) determined SCCPs ($C_{10–13}$, 60–70% chlorine) in 24-hour air samples collected daily during a 4-month period in the summer of 1990 in Egbert, Ontario, a "rural site northwest of Toronto," by HRGC/ECNI-HRMS (Muir *et al*., 1999). Concentrations ranged from 65 to 924 pg/m^3. Although a summary statistic of 543 pg/m^3 was reported, it was not specified whether this was a mean or median value. Egbert has also been reported to be near an "industrialized area" (Muir *et al*., 2000). Lower concentrations of SCCPs have been identified at other sites in Canada (Halsall *et al*., 1998; Stern *et al*., 1998; Bidleman *et al*., 1999, 2000, 2001; Muir *et al*., 2001).

Concentrations of SCCPs ($C_{10–13}$, 52% chlorine) ranged from 11 to 17 µg/kg in human breast milk in Canada (Tomy, 1997). Analyses were carried out by HRGC/ECNI-HRMS. No additional details were reported.[2]

Muir *et al*. (1999) analysed whole fish samples for SCCPs ($C_{10–13}$) and detected 2630 ng/g (wet weight) in carp from Hamilton Harbour, 58.8 ng/g (wet weight) in lake trout from Niagara-on-the-Lake and 72.6 ng/g (wet weight) in lake trout from Port Credit. The quantification was by GC/ECNI-HRMS. Lower concentrations were reported in an earlier study (Muir *et al*., 1996).

In a market basket survey (KAN-DO Office and Pesticides Team, 1995)[3] of 234 ready-to-eat foods, which represented approximately 5000 food types in American diets, "Chlorowax 500C"[4] was detected once, in enriched white bread, at a concentration of 0.13 µg/g. Food items were screened by gas or liquid chromatography using ion-selective detectors. Findings were confirmed by unspecified analysis.

Concentrations of SCCPs have been identified in blubber of aquatic mammals such as ringed seal, beluga and walrus (Tomy *et al*., 2000[5]; Bennie *et al*., 2000[6]). The samples were from animals in Greenland, the Canadian Arctic and the St. Lawrence River. A mean concentration of 46 100 ng/g (n = 15) was reported for beluga from the St. Lawrence River/Gulf of St. Lawrence. Concentrations in ringed seals from Ellesmere Island ranged from 370 to 770 ng/g. Jansson *et al*. (1993) detected SCCPs in biota in

[2] These data were identified in a secondary source and were originally reported in a Ph.D. thesis.

[3] Reported as a summary of results from 1982 to 1991.

[4] The average molecular formula for Chlorowax 500C is $C_{12}H_{19}Cl_7$, with 60–65% chlorine content (w/w) (IPCS, 1996).

[5] Analysis by HRGC/ECNI-HRMS.

[6] Analysis by GC/low-resolution negative chemical ionization mass spectrometry.

Sweden, including fish and both terrestrial and marine mammals. Analysis was by GC/MS.

Data on concentrations of SCCPs in drinking water in Canada or elsewhere were not identified. The maximum concentration of SCCPs ($C_{10–13}$, 50–70% chlorine) in the Red River, at a site remote from industrialized areas, was 0.05 µg/L (Tomy, 1997).[7] Analyses were by HRGC/ECNI-HRMS. A lower concentration was reported in surface water from Lake Ontario (Muir *et al*., 2001).

Concentrations of SCCPs in soil in Canada or elsewhere were not identified. The concentrations in surface sediment in harbours in Lake Ontario ranged from 5.9 to 290 ng/g dry weight (Muir *et al*., 2001). Analyses were by HRGC/ECNI-HRMS.

Upper-bound estimates of intake of SCCPs for the general Canadian population and the assumptions upon which they are based are presented in Table 9. For each age group in the Canadian population, virtually all of the estimated intake is from food. The upper-bound estimated intake of breast-fed infants was 1.7 µg/kg-bw per day, and that of formula-fed infants was 0.01 µg/kg-bw per day. For the remaining age groups, intakes ranged from 5.1 µg/kg bw per day for adults over 60 years of age to 26.0 µg/kg-bw per day for infants who were not formula fed (i.e., those being introduced to solid foods[8]).

Table 9. Upper-bounding estimated average daily intake of short-chain chlorinated paraffins by the population of Canada

Route of exposure	**Estimated intake (µg/kg-bw per day) of short-chain chlorinated paraffins 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]**	**not formula fed[4]**					
Ambient air[10]	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Indoor air[11]	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Drinking water[12]	1.7	0.005	0.001	0.001	0.001	<0.001	<0.001	<0.001
Food[13]			25.96	24.26	16.44	9.02	7.18	5.14
Soil[14]	0.001	0.001	0.001	0.002	0.001	<0.001	<0.001	<0.001
Total intake	**1.7**	**0.01**	**25.97**	**24.26**	**16.44**	**9.02**	**7.18**	**5.14**

1 Assumed to weigh 7.5 kg and to breathe 2.1 m^3 per day (21 hours indoors, 3 hours outdoors) (EHD, 1998).

2 Concentrations of SCCP ($C_{10–13}$, 52% chlorine) ranged from 11 to 17 µg/kg in human breast milk in Canada (Tomy, 1997). No additional details were reported. These data were identified in a secondary source and were originally reported in a Ph.D. thesis. Assumed to consume 0.75 kg breast milk per day (EHD, 1998).

3 For formula-fed infants, intake from water is synonymous with intake from food. Assumed to consume 0.8 L

[7] These data were identified in a secondary source and were originally reported in a Ph.D. thesis.

[8] Solid foods are introduced to approximately 50% of infants by 4 months of age and to 90% by 6 months of age (NHW, 1990).

reconstituted formula daily (EHD, 1998).

[4] Assumed to drink 0.2 L of water per day. Consumption of food groups reported by Health Canada (EHD, 1998).

[5] Assumed to weigh 15.5 kg, to breathe 9.3 m^3 per day (21 hours indoors, 3 hours outdoors) and to drink 0.2 L of water per day. Consumption of food groups reported by Health Canada (EHD, 1998).

[6] Assumed to weigh 31.0 kg, to breathe 14.5 m^3 per day (21 hours indoors, 3 hours outdoors) and to drink 0.4 L of water per day. Consumption of food groups reported by Health Canada (EHD, 1998).

[7] Assumed to weigh 59.4 kg, to breathe 15.8 m^3 per day (21 hours indoors, 3 hours outdoors) and to drink 0.4 L of water per day. Consumption of food groups reported by Health Canada (EHD, 1998).

[8] Assumed to weigh 70.9 kg, to breathe 16.2 m^3 per day (21 hours indoors, 3 hours outdoors) and to drink 0.4 L of water per day. Consumption of food groups reported by Health Canada (EHD, 1998).

[9] Assumed to weigh 72.0 kg, to breathe 14.3 m^3 per day (21 hours indoors, 3 hours outdoors) and to drink 0.4 L of water per day. Consumption of food groups reported by Health Canada (EHD, 1998).

[10] The maximum concentration of C_{10}–C_{13} (60–70% chlorine) in gas-phase air samples collected every day over a 4-month period in the summer of 1990 at Egbert, a rural site northwest of Toronto, was 924 pg/m^3 (Muir *et al*., 1999).

[11] Concentrations of SCCP in indoor air in Canada or elsewhere were not identified. The value used for calculating intake here is the above concentration identified for ambient air (Muir *et al*., 1999).

[12] Concentrations of SCCP in drinking water were not identified. The maximum concentration of SCCP ($C_{10–13}$, 50–70% chlorine) identified in the Red River, at a site remote from industrialized areas, was 0.05 µg/L (Tomy, 1997).

[13] Estimates of intake from food are based upon concentrations in foods that are selected to represent the food groups addressed in calculating exposure to Priority Substances (EHD, 1998):

Dairy: 0.3 µg/g; mean of 13 samples of dairy products in U.K.; $C_{10–20}$ (SCCP and MCCP) (Campbell and McConnell, 1980a)
Fats: 0.15 µg/g; mean of 6 samples of vegetable oils and derivatives; $C_{10–20}$ (SCCP and MCCP) (Campbell and McConnell, 1980a)
Fruits: 0.025 µg/g; mean of 16 samples of fruits and vegetables in U.K.; $C_{10–20}$ (SCCP and MCCP) (Campbell and McConnell, 1980a)
Vegetables: 0.025 µg/g; mean of 16 samples of fruits and vegetables in U.K.; $C_{10–20}$ (SCCP and MCCP) (Campbell and McConnell, 1980a)
Cereal products: 0.13 µg/g; one reported concentration for "Chlorowax 500C" in enriched white bread in market basket survey carried out by U.S. Food and Drug Administration (KAN-DO Office and Pesticides Team, 1995); average molecular formula is $C_{12}H_{19}Cl_7$, with 60–65% chlorine content (w/w) (IPCS, 1996)
Meat and poultry: 0.099 µg/g; 1 sample of bacon in U.K.; $C_{10–20}$ (SCCP and MCCP) (Campbell and McConnell, 1980b)
Fish: 2.630 µg/g (wet weight); analysis of whole samples of carp from Hamilton Harbour; C_{10}–C_{13} (Muir *et al*., 1999)
Eggs: no data identified
Foods primarily sugar: 0.025 µg/g; 1 sample of strawberry jam in U.K.; $C_{10–20}$ (SCCP and MCCP) (Campbell and McConnell, 1980b)
Mixed dishes: no data identified
Nuts and seeds: no data identified
Soft drinks, alcohol, coffee, tea: 0.05 µg/g; detection limit in analyses of beverages in U.K. (Campbell and McConnell, 1980a)

Amounts of foods consumed on a daily basis by each age group are described by Health Canada (EHD, 1998).

[14] No data were identified on concentrations of SCCP in soil in Canada. The maximum concentration in surface sediment in harbours in Lake Ontario was 290 ng/g dry weight (Muir *et al*., 2001).

Canadian data incorporated within this estimate include high-confidence values in fish (whole carp determined by GC/ECNI-HRMS) and data on breast milk, for which details of sampling and analysis were not reported. Estimated intake of SCCPs in fish represents up to 58% of the total daily intake. The intake from dairy products, which accounts for 89.9% of the intake of infants not formula fed, is based upon limited sampling and analysis — considered semi-quantitative only — of dairy products in the United Kingdom, reported in 1980. Probably the most representative estimates of intake are those from cereals, which are based upon data reported in an American market basket survey, carried out from 1982 to 1991; however, intake from this foodstuff constitutes

<0.1% of total estimated intake, and analytical methods were not specified.

Intake of SCCPs by a potentially higher-exposure subgroup of Inuit for whom the primary source of food is subsistence hunting and fishing (Kuhnlein, 1989; Kinloch *et al*., 1992) was also estimated, based on data on concentrations of SCCPs in blubber from marine mammals in Canada (Tomy *et al*. 2000) and less specific data (including both SCCPs and MCCPs) for terrestrial and marine mammals from Sweden (Jansson *et al*., 1993). On the basis of these data, the estimated intake of an Inuit adult, namely 1.47 µg/kg-bw per day, is well within the range of values estimated above for the general population.

5.1.2 MCCPs

MCCPs were detected by HRGC/low-resolution mass spectrometry (LRMS) in effluent (13 µg/L) from a chlorinated paraffin manufacturing plant in Canada in 1993, but not in surface water or sediment (Metcalfe-Smith *et al*., 1995). MCCPs were detected in three samples of carp from Hamilton Harbour in 1996 by low-resolution GC/MS (mean 0.393 µg/g; range 0.276–0.563 µg/g) (Bennie *et al*., 2000). Similarly, MCCPs were detected in the homogenized (whole) samples of 10 trout collected from western Lake Ontario in 1996 (mean 1.23 µg/g; range 0.257–4.39 µg/g) (Bennie *et al*., 2000).

Upper-bounding estimates of intake for MCCPs and the assumptions on which they are based are presented in Table 10. For each age group, virtually all of the estimated intake is from food, which, in turn, is based almost entirely upon the limited data reported by Campbell and McConnell (1980a,b). The highest intake estimated (25.5 µg/kg-bw per day) was for infants not formula fed.

Table 10. Upper-bounding estimated average daily intake of medium-chain chlorinated paraffins by the population of Canada

Route of exposure	**Estimated intake (µg/kg-bw per day) of medium-chain chlorinated paraffins by various age groups**						
	0–6 months[1]		**6 months–4 years[4]**	**5–11 years[5]**	**12–19 years[6]**	**20–59 years[7]**	**60+ years[8]**
	formula fed[2]	**not formula fed[3]**					
Ambient air[9]	–	–	–	–	–	–	–
Indoor air[10]	–	–	–	–	–	–	–
Drinking water[11]	0.05	0.01	0.01	0.01	<0.01	<0.01	<0.01
Food[12]		25.48	18.48	11.64	6.3	4.69	3.47
Soil[13]	0.01	0.01	0.02	0.01	<0.01	<0.01	<0.01
Total intake	**0.07**	**25.51**	**18.51**	**11.65**	**6.3**	**4.69**	**3.47**

1 Assumed to weigh 7.5 kg and to breathe 2.1 m^3 per day (21 hours indoors, 3 hours outdoors) (EHD, 1998).

2 For formula-fed infants, intake from water is synonymous with intake from food. Assumed to consume 0.8 L reconstituted formula daily. No data on concentrations of MCCP in formula were identified for Canada.

3 Assumed to drink 0.2 L of water per day. Consumption of food groups reported by Health Canada (EHD, 1998).

4 Assumed to weigh 15.5 kg, to breathe 9.3 m^3 per day (21 hours indoors, 3 hours outdoors), to ingest 100 mg of

soil per day and to drink 0.2 L of water per day. Consumption of food groups reported in EHD (1998).

[5] Assumed to weigh 31.0 kg, to breathe 14.5 m^3 per day (21 hours indoors, 3 hours outdoors), to ingest 65 mg of soil per day and to drink 0.4 L of water per day. Consumption of food groups reported in EHD (1998).

[6] Assumed to weigh 59.4 kg, to breathe 15.8 m^3 per day (21 hours indoors, 3 hours outdoors), to ingest 30 mg of soil per day and to drink 0.4 L of water per day. Consumption of food groups reported in EHD (1998).

[7] Assumed to weigh 70.9 kg, to breathe 16.2 m^3 per day (21 hours indoors, 3 hours outdoors), to ingest 30 mg of soil per day and to drink 0.4 L of water per day. Consumption of food groups reported in EHD (1998).

[8] Assumed to weigh 72.0 kg, to breathe 14.3 m^3 per day (21 hours indoors, 3 hours outdoors), to ingest 30 mg of soil per day and to drink 0.4 L of water per day. Consumption of food groups reported in EHD (1998).

[9] Concentrations of MCCP in ambient air in Canada or elsewhere were not identified.

[10] Concentrations of MCCP in indoor air in Canada or elsewhere were not identified.

[11] Concentrations of MCCP in Canadian drinking water were not identified. Intakes are based upon the limit of detection (0.5 µg/L) in a survey of drinking water in reservoirs in the U.K. (Campbell and McConnell, 1980a).

[12] Estimates of intake from food are based upon concentrations in foods that are selected to represent the food groups addressed in calculating exposure to Priority Substances (EHD, 1998):

Dairy: 0.3 µg/g; mean of 13 samples of dairy products in U.K.; $C_{10–20}$ (SCCP and MCCP) (Campbell and McConnell, 1980a)
Fats: 0.15 µg/g; mean of 6 samples of vegetable oils and derivatives; $C_{10–20}$ (SCCP and MCCP) (Campbell and McConnell, 1980a)
Fruits: 0.025 µg/g; mean of 16 samples of fruits and vegetables in U.K.; $C_{10–20}$ (SCCP and MCCP) (Campbell and McConnell, 1980a)
Vegetables: 0.025 µg/g; mean of 16 samples of fruits and vegetables in U.K.; $C_{10–20}$ (SCCP and MCCP) (Campbell and McConnell, 1980a)
Cereal products: 0.05 µg/g, detection limit in analyses of corn flakes in U.K. (Campbell and McConnell, 1980b)
Meat and poultry: 0.099 µg/g; 1 sample of bacon in U.K.; $C_{10–20}$ (SCCP and MCCP) (Campbell and McConnell, 1980b)
Fish: 1.23 µg/g (wet weight); mean of 10 samples of whole trout from western Lake Ontario (Bennie *et al*., 2000)
Eggs: no data identified
Foods primarily sugar: 0.025 µg/g; 1 sample of strawberry jam in U.K.; $C_{10–20}$ (SCCP and MCCP) (Campbell and McConnell, 1980b)
Mixed dishes: no data identified
Nuts and seeds: no data identified
Soft drinks, alcohol, coffee, tea: 0.05 µg/g; detection limit in analyses of beverages in U.K. (Campbell and McConnell, 1980a)

[13] Amounts of foods consumed on a daily basis by each age group are described by Health Canada (EHD, 1998). The value used for calculating intake from soil is the limit of quantification (3.5 µg/g) in a survey of sediment from the St. Lawrence River (Metcalfe-Smith *et al*., 1995).

5.1.3 LCCPs

Upper-bounding estimates of total intake of LCCPs and associated assumptions are presented in Table 11. As for SCCPs and MCCPs, for each age group, virtually all of the estimated intake is from food. The highest intake estimated (16.8 µg/kg-bw per day) was for infants not formula fed. In addition to the limitations of the analytical methodology noted previously, these estimates are further limited in that estimates for five of the eight food groups are based upon the limit of detection in that survey (Campbell and McConnell, 1980a,b).

Table 11. Upper-bounding estimated average daily intake of long-chain chlorinated paraffins by the population of Canada

Route of exposure	Estimated intake (µg/kg-bw per day) of long-chain chlorinated paraffins by various age groups					
	0–6 months[1]	6 months–	5–11	12–19	20–59	60+ years[8]

	formula fed[2]	not formula fed[3]	4years[4]	years[5]	years[6]	years[7]	
Ambient air[9]	–	–	–	–	–	–	–
Indoor air[10]	–	–	–	–	–	–	–
Drinking water[11]	0.05	0.01	0.01	0.01	<0.01	<0.01	<0.01
Food[12]		16.81	9.66	5.61	3.04	2.12	1.73
Soil[13]	0.01	0.01	0.02	0.01	<0.01	<0.01	<0.01
Total intake	**0.07**	**16.83**	**9.69**	**5.63**	**3.04**	**2.12**	**1.73**

[1] Assumed to weigh 7.5 kg and to breathe 2.1 m^3 per day (21 hours indoors, 3 hours outdoors) (EHD, 1998).

[2] For formula-fed infants, intake from water is synonymous with intake from food. Assumed to consume 0.8 L reconstituted formula daily. No data on concentrations of LCCP in formula were identified for Canada.

[3] Assumed to drink 0.2 L of water per day. Consumption of food groups reported in EHD (1998).

[4] Assumed to weigh 15.5 kg, to breathe 9.3 m^3 per day (21 hours indoors, 3 hours outdoors), to ingest 100 mg of soil per day and to drink 0.2 L of water per day. Consumption of food groups reported in EHD (1998).

[5] Assumed to weigh 31.0 kg, to breathe 14.5 m^3 per day (21 hours indoors, 3 hours outdoors), to ingest 65 mg of soil per day and to drink 0.4 L of water per day. Consumption of food groups reported in EHD (1998).

[6] Assumed to weigh 59.4 kg, to breathe 15.8 m^3 per day (21 hours indoors, 3 hours outdoors), to ingest 30 mg of soil per day and to drink 0.4 L of water per day. Consumption of food groups reported in EHD (1998).

[7] Assumed to weigh 70.9 kg, to breathe 16.2 m^3 per day (21 hours indoors, 3 hours outdoors), to ingest 30 mg of soil per day and to drink 0.4 L of water per day. Consumption of food groups reported in EHD (1998).

[8] Assumed to weigh 72.0 kg, to breathe 14.3 m^3 per day (21 hours indoors, 3 hours outdoors), to ingest 30 mg of soil per day and to drink 0.4 L of water per day. Consumption of food groups reported in EHD (1998).

[9] Concentrations of LCCP in ambient air in Canada or elsewhere were not identified.

[10] Concentrations of LCCP in indoor air in Canada or elsewhere were not identified.

[11] Concentrations of LCCP in Canadian drinking water were not identified. Intakes are based upon the limit of detection (0.5 µg/L) in a survey of drinking water in reservoirs in U.K. (Campbell and McConnell, 1980a).

[12] Estimates of intake from food are based upon concentrations in foods that are selected to represent the food groups addressed in calculating exposure to Priority Substances (EHD, 1998):

Dairy: 0.19 µg/g; 1 sample of cheese in U.K.; $C_{20–30}$ (Campbell and McConnell, 1980a)
Fats: 0.05 µg/g; detection limit in analysis of 1 sample of lard in U.K.; $C_{20–30}$ (Campbell and McConnell, 1980a)
Fruits: 0.025 µg/g; 1 sample of peach fruit in U.K.; $C_{20–30}$ (Campbell and McConnell, 1980a)
Vegetables: 0.025 µg/g; 1 sample of potato crisps in U.K.; $C_{20–30}$ (Campbell and McConnell, 1980a)
Cereal products: 0.05 µg/g, detection limit in analysis of corn flakes in U.K. (Campbell and McConnell, 1980b)
Meat and poultry: 0.05 µg/g; detection limit in analysis of 1 sample each of ox liver and beef in U.K.; $C_{20–30}$ (Campbell and McConnell, 1980b)
Fish: no data identified
Eggs: no data identified
Foods primarily sugar: 0.05 µg/g; detection limit in analysis of 1 sample of strawberry jam in U.K.; $C_{20–30}$ (Campbell and McConnell, 1980b)
Mixed dishes: no data identified
Nuts and seeds: no data identified
Soft drinks, alcohol, coffee, tea: 0.05 µg/g; detection limit in analysis of 1 sample each of beer and tea in U.K.; $C_{20–30}$ (Campbell and McConnell, 1980b)

[13] Amounts of foods consumed on a daily basis by each age group are described by Health Canada (EHD, 1998). The value used for calculating intake from soil is the maximum concentration (3.2 µg/g) reported in a survey of sediment in the U.K. (Campbell and McConnell, 1980a).

5.2. Hazard characterization and dose–response analyses

A limited number of studies on the toxicity of SCCPs have been reported in the period following release of the PSL1 assessment. Most of these studies were conducted to

investigate the mode of action of carcinogenicity for the tumours observed in the NTP (1986a) bioassay, which were liver tumours in both sexes of rats and mice, kidney tumours in male, but not female, rats and thyroid tumours in rats and mice (females only). For several of these more recent studies, results have been reported in abstracts or summaries only: Elcombe *et al*. (1994) (abstract), Elcombe *et al*. (2000) (summary) and Warnasuriya *et al*. (2000) (abstract). For only one of the relevant investigations has a full published account been identified (Wyatt *et al*., 1993). While secondary accounts of (possibly) other studies investigating mode of action of tumour induction in assessments have been reported by the European Commission (2000), the U.S. National Research Council (U.S. NRC, 2000) and the Australian National Industrial Chemicals Notification and Assessment Scheme (NICNAS, 2001), they are not further considered here, owing to lack of availability or confirmation of subsequent publication (Jackson, 2001).

Few data relevant to the assessment of the toxicity of either MCCPs or LCCPs were identified for the period to the release of the PSL1 assessment report. The following presentation is limited to those considered critical to hazard characterization or dose–response analyses for effects in the general population and, hence, to assessment of "toxic" under Paragraph 64(c) of CEPA 1999. Other sources of non-critical data identified but not included were DuPont (1995), Kato and Kenne (1996) and Warngard (1996).

In view of the absence of recent toxicological data that impact on critical aspects, the dose–response analyses for MCCPs and LCCPs presented here reflect primarily those developed in the PSL1 Assessment Report released under CEPA 1988.

5.2.1 SCCPs

A- Liver

Increased liver weight, hepatocellular hypertrophy, peroxisomal proliferation and increased S-phase activity in hepatocytes were reported in Fischer 344 rats administered SCCPs for up to 90 days (presumably by gavage) at dose levels up to 1000 mg/kg-bw per day (Elcombe *et al*., 1994; abstract). Lower doses administered were not specified, and quantitative dose- or sex-specific data and analyses were not presented.

Elcombe *et al*. (2000) administered Chlorowax 500C ($C_{10–13}$; 58% chlorine) to male and female Fischer 344 rats by gavage in corn oil for up to 90 days, at dose levels of 0, 312 or 625 mg/kg-bw per day. In both sexes, liver weight was increased, accompanied by peroxisomal proliferation (as indicated by an increase in cyanide-insensitive palmitoyl coenzyme A [CoA] oxidation) and increased thyroxine (T_4)–uridine diphosphoglucose glucuronosyl transferase (UDPGGT). (The effects were, presumably, observed at both dose levels.) These effects were not observed in male Dunkin Hartley guinea pigs similarly administered 0, 500 or 1000 mg/kg-bw per day for 14 consecutive days. The numbers of animals exposed were not specified, and quantitative dose- or sex-specific data and analyses were not presented in this summary account.

Wyatt *et al*. (1993) exposed groups of five male rats (Alpk:APfSD strain) each by gavage for 14 days to 0, 10, 50, 100, 250, 500 or 1000 mg/kg-bw per day to two SCCPs (Chlorowax 500C: $C_{10–13}$, 58% chlorine; or Cereclor 56L, $C_{10–13}$: 56% chlorine). For the 58% chlorine SCCPs, both absolute and relative liver weights were significantly increased in a dose-related manner, at doses of 100 mg/kg-bw per day or greater. Peroxisomal fatty acid β-oxidation activity (indicated by palmitoyl CoA oxidation) was significantly increased at 250 mg/kg-bw per day and greater (irregular dose–response). For the 56% chlorine SCCPs, the pattern of response for absolute liver weight was irregular; however, relative liver weight was increased in a dose-related manner, significantly at 50 mg/kg-bw per day and greater. Palmitoyl CoA oxidation was significantly increased only at the highest dose.

In similarly exposed male mice (Alpk:APfCD-1 strain), for the 58% chlorine SCCPs, there was a dose-related increase in relative liver weight and palmitoyl CoA oxidation, both significant at 250 mg/kg-bw per day and greater (Wyatt *et al*., 1993). For the 56% chlorine SCCPs, both absolute and relative liver weights were significantly increased in a dose-related manner at doses of 100 mg/kg-bw per day or greater. Palmitoyl CoA oxidation was significantly increased in a dose-related manner at 250 mg/kg-bw per day and greater.

The only other relevant investigation identified was an *in vitro* study in which SCCPs inhibited gap junction intercellular communication in rat liver cells (Kato and Kenne, 1996; Warngard *et al*., 1996).

B-Kidney

Increased proximal tubular cell eosinophilia (suggestive of a protein overload, but not necessarily α_{2u} globulin) and regenerative focal basophilic tubules, as well as increased S-phase activity in the proximal tubular cells, were reported in male, but not female, rats administered up to 1000 mg SCCPs/kg-bw per day for up to 90 days (other dose levels were not specified) (Elcombe *et al*., 1994). These observations were reported in an abstract; neither quantitative data nor statistical analyses were presented.

Elcombe *et al*. (2000) also investigated renal effects in F344 rats and guinea pigs administered 0, 312 or 625 mg SCCPs/kg-bw per day for up to 90 days. In the male rats only, there was chronic protein nephropathy, associated with regenerative hyperplasia and increased DNA synthesis (S-phase activity), presumably at both dose levels. There was "some limited evidence" for an involvement of α_{2u} globulin. These changes were not observed in the guinea pigs. Again, neither quantitative data nor statistical analyses were presented in this summary account.

Warnasuriya *et al*. (2000) exposed male and female rats by gavage for 28 days to 625 mg SCCPs (C_{12}; 60% chlorine)/kg-bw per day. There was an increase in α_{2u} globulin and cell proliferation in the kidney of males only. Data from individual rats indicated that increased cell proliferation was directly correlated with the increase in α_{2u} globulin. Five different isoelectric isoforms of α_{2u} globulin were identified by Western blotting in the

control male kidney, and all five were increased in the treated males. These observations were reported in an abstract; neither quantitative data nor statistical analyses were presented.

C- Thyroid

Elcombe *et al*. (1994) reported that exposure of rats to SCCPs for up to 90 days resulted in induction of T_4–glucuronosyl transferase activity, accompanied by a decrease in plasma T_4 and an increase in thyroid stimulating hormone (TSH). Thyroid follicular cell hypertrophy and hyperplasia were also observed. Increased S-phase activity in the thyroid follicular cells was also reported. The maximum dose was 1000 mg/kg-bw per day; other dose levels were not specified. This study was reported as an abstract; neither quantitative data nor statistical analyses were presented.

In male and female Fischer 344 rats exposed by gavage in corn oil to 0, 312 or 625 mg/kg-bw per day for up to 90 days, there were decreases in plasma T_4, increases in plasma TSH and thyroid follicular cell hypertrophy and hyperplasia in both sexes, changes that were not observed in male guinea pigs (Elcombe *et al*., 2000). Quantitative data and statistical analyses were not presented in this summary account.

Gavage administration of 6.8 mg/kg-bw per day commercial $C_{10–13}$ (71% chlorine) to female Sprague-Dawley rats for 14 days had no effect upon thyroid hormonal T_4 levels or microsomal enzyme activity (Hallgren and Darnerud, 1998).

In male rats (Alpk:APfSD strain) exposed by gavage for 14 days to two SCCPs (Chlorowax 500C: $C_{10–13}$, 58% chlorine; or Cereclor 56L, $C_{10–13}$: 56% chlorine), for which examination of thyroid function was restricted to the control and high-dose groups (1000 mg/kg-bw per day), both free and total T_4 were significantly reduced, TSH was significantly increased and the capability of liver microsomes to glucuronidate T_4 was significantly increased in exposed animals (Wyatt *et al*., 1993). No differences in levels of free or total triiodothyronine (T_3) were observed for either SCCPs. A significant increase in glucuronosyl transferase activity with p-nitrophenol was observed only from microsomes from rats exposed to the $C_{10–13}$ (58% chlorine) compound.

5.2.2 MCCPs

A subchronic dietary study with MCCPs in rats (Poon *et al*., 1995) was initiated by Health Canada in response to the research needs identified in the PSL1 assessment of chlorinated paraffins (Government of Canada, 1993a). Sprague-Dawley rats (10 per sex per group) were fed diets containing 0, 5, 50, 500 or 5000 ppm for 13 weeks. The dose levels calculated by the authors on the basis of weekly food consumption were 0, 0.4, 3.6, 36 and 363 mg/kg-bw per day for males and 0, 0.4, 4.2, 42 and 419 mg/kg-bw per day for females. The protocol included serum biochemistry, hematology, hepatic enzyme activities, urinary enzyme activity, organ weights and histopathology. Mild, adaptive histological changes were detected in the liver of rats of both sexes at the two highest doses (LOEL = 36 mg/kg-bw per day) and in the thyroid of males at 36 mg/kg-bw per

day and greater and of females at 4.2 mg/kg-bw per day and greater (NOAEL = 0.4 mg/kg-bw per day). Minimal changes were observed in the renal proximal tubules of males at the highest dose and in the inner medulla of females at the two highest doses.

5.2.3 LCCPs

No critical data relevant to the assessment of the toxicity of LCCPs were identified for the period since the PSL1 assessment was released.

5.3. Human health risk characterization

5.3.1 SCCPs

A- Hazard characterization

Genotoxicity

Requisite criteria for assessing the weight of evidence for hypothesized modes of induction of tumours addressed below include the criterion that SCCPs are not DNA-reactive. Recent data on genotoxicity reported since the PSL1 assessment was released have not been identified. Limited available data reviewed within the PSL1 assessment indicated that SCCPs were clastogenic in *in vitro* assays, although they had not been clastogenic or mutagenic in a limited number of *in vivo* assays.

Based on review of the available data, including two additional unpublished studies in which no increases in revertant colonies in five strains of *Salmonella*[9] and no increases in mutant colonies in Chinese hamster V79 cells[10] were reported in the secondary account, it was concluded that "as a group, SCCPs are not mutagenic" (European Commission, 2000).

Liver

It has been hypothesized that SCCPs cause liver tumours in rodents secondary to peroxisome proliferation. Peroxisome proliferation involves activation of a nuclear receptor in rodent liver, the peroxisome proliferator activated receptor, α isoform (PPARα). The activated PPARα interacts with regulatory elements of the DNA to initiate transcription of genes for increased peroxisomal enzyme activity and cell proliferation characterized by morphological and biochemical changes in the liver. These changes include increased liver weight through both hepatocyte hypertrophy and hyperplasia,

[9] Cited by the European Commission (2000) as: Unpublished Report 86, Hoechst AG, Unpublished study, 88.0099, 1988.

[10] Cited by the European Commission (2000) as: Unpublished Report 92, Hoechst AG, Unpublished study, 87.1719, 1987.

increased number and size of peroxisomes, increased activity (up to 40-fold) of peroxisomal enzymes (especially those involved in peroxisomal fatty acid oxidation) and induction of microsomal fatty acid oxidation through the CYP4A subfamily of cytochrome P-450 isozymes. Minimum criteria for characterizing peroxisome proliferation are considered to include hepatomegaly, enhanced cell proliferation and an increase in hepatic acyl-CoA oxidase and/or palmitoyl-CoA oxidation levels.

In the NTP bioassay (NTP, 1986a; Bucher *et al*., 1987) reported in the PSL1 assessment, increases in benign liver tumours were observed in both SCCPs-exposed rats (312 and 625 mg/kg-bw per day) and mice (125 and 250 mg/kg-bw per day), with males of both species being considerably more sensitive. This pattern of induction of liver tumours by SCCPs is consistent with that for other peroxisome proliferating hepatocarcinogens, such as di(2-ethylhexyl)phthalate.

Available data on the role of peroxisome proliferation in the etiology of hepatic effects and liver tumours induced by SCCPs are restricted to one study for which there is a published manuscript (Wyatt *et al*., 1993) and two investigations reported only in summary (Elcombe *et al*., 2000) or abstract form (Elcombe *et al*., 1994). Significant, dose-related increases in both absolute and relative liver weights accompanied at higher doses by increases in palmitoyl CoA oxidation in male Alpk:APfSD rats and Alpk:APfCD-1 mice exposed to two SCCPs, reported by Wyatt *et al*. (1993), are consistent with the observations in rats of Elcombe *et al*. (1994, 2000). Also, to the extent to which the more recent and better-documented study of Wyatt *et al*. (1993), with more extensive characterization of dose–response, can be compared with the earlier investigations of Elcombe *et al*. (1994, 2000), for which only summary reports are available, observations on dose–response for increases in liver weight and palmitoyl CoA oxidation in rats in these investigations are also consistent (increases in relative liver weight in rats were significant at ≥50 mg/kg-bw per day and palmitoyl CoA oxidation at ≥250 mg/kg-bw per day; comparable values for mice were 100 mg/kg-bw per day and 250 mg/kg-bw per day).

Therefore, although characterization of exposure–response was limited in the NTP bioassay to only two dose levels, evidence to date indicates that tumours in both rats and mice occur only at doses at which peroxisome proliferation and associated morphological and biochemical effects have been observed in shorter-term studies (Wyatt *et al*., 1993; Elcombe *et al*., 1994, 2000).

Additional weight of evidence for concordance might have been afforded through consideration of sex-related differences in peroxisome proliferation in shorter-term mechanistic studies. Unfortunately, this aspect was not investigated in the well-reported study by Wyatt *et al*. (1993) in which only male rats and mice were exposed; moreover, the limited extent of reporting in Elcombe *et al*. (1994, 2000) precludes consideration of relevant data in this context, if such data were, indeed, collected. Recovery studies would also have been informative, since peroxisome proliferation is initiated rapidly after treatment with a proliferator begins, attains a maximal response in a few weeks and is maintained only in the continued presence of the proliferator. Consistent with a receptor-

mediated response, the process is reversible.

While there have been no carcinogenesis bioassays for SCCPs in species other than rats and mice, the variation in species sensitivity to peroxisome proliferation reported by Elcombe *et al*. (2000) is consistent with that observed for other peroxisome proliferators. Rats and mice are uniquely responsive to the morphological and biochemical effects of peroxisome proliferators, while Syrian hamsters exhibit intermediate responsiveness. This is consistent with marked interspecies variations in the expression of PPARα.

Additional published documentation of existing relevant studies is desirable. Also, investigation of additional aspects of concordance would strengthen the weight of evidence for causality for the purported association between peroxisome proliferation and liver tumours induced by SCCPs. However, although there are limitations of the identified information, data are strongly suggestive that peroxisome proliferation plays a role in the etiology of liver damage and hepatic tumours associated with exposure to SCCPs. Although additional evidence for the weight of causality for liver tumours is desirable, a TDI based on hepatic effects in experimental animals is considered to be protective for carcinogenicity.

Kidney

It has been hypothesized that the kidney tumours observed following exposure of male rats to SCCPs are a species- and sex-specific response attributable to α_{2u} globulin nephropathy and hence not relevant to humans. This mode of induction of renal tumours, which is relatively well characterized, involves binding to α_{2u} globulin, a protein specific to male rats. This binding renders the protein more resistant to proteolytic degradation, which causes its accumulation in renal proximal tubule cells (manifested as hyaline droplets on histopathological examination), resulting in cell death and regenerative proliferation. Sustained cell proliferation leads to a low but significant incidence of renal tubular tumours.

Minimum criteria for establishment of α_{2u} globulin nephropathy as a basis for tumour development include lack of genotoxicity and observation of requisite precursor lesions and tumours in male rats only. Confirmation of requisite precursor lesions is based not only on histopathological observations such as excessive accumulation of hyaline droplets in renal proximal tubule cells, subsequent cytotoxicity and single-cell necrosis of the tubular epithelium and sustained regenerative tubular cell proliferation in the presence of continued exposure, but also on explicit identification of the protein accumulating in tubule cells as α_{2u} globulin, along with demonstrated reversible binding of the relevant chemical or metabolite to α_{2u} globulin (U.S. EPA, 1991; IARC, 1999).

In the NTP bioassay (NTP, 1986a; Bucher *et al*., 1987) reported in the PSL1 assessment, renal tubular cell adenomas were observed in male rats at both doses (312 and 625 mg/kg-bw per day), although the increase was significant ($p < 0.05$) only at the lower dose. Characterization of exposure–response was limited, therefore, in the NTP bioassay to only two dose levels.

Available data on the mode of induction of kidney tumours in male rats by SCCPs are restricted to three investigations reported only in summary or abstract format (Elcombe *et al*., 1994, 2000; Warnasuriya *et al*., 2000). In Elcombe *et al*. (1994, 2000), regenerative focal basophilic tubules and increased S-phase activity in the proximal tubular renal cells were observed in male, but not female rats and considered by the authors to constitute "limited evidence" of the role of α_{2u} globulin. More recently, the presence of α_{2u} globulin was confirmed using immunohistochemical techniques, although no details of methodology were provided (Warnasuriya *et al*., 2000).

Owing to the inadequate characterization in abstracts of even administered doses, in some cases with quantitative data on effects and analyses not being reported, there is very limited documentation to serve as a basis for conclusion that renal tumours occur only at doses at which either chronic protein nephropathy associated with regenerative hyperplasia and increased DNA synthesis (Elcombe *et al*., 2000) or α_{2u} globulin is observed (Warnasuriya *et al*., 2000).

While information is strongly suggestive that the kidney tumours observed in male rats are attributable to hyaline droplet formation, a male rat-specific phenomenon not relevant to humans, additional published documentation of available studies is clearly desirable as a basis for consideration of the weight of evidence of mode of induction of kidney tumours. Although additional confirmation is desirable, a TDI based on renal effects in experimental animals is considered to be protective for carcinogenicity.

Thyroid

There are a variety of non-DNA-reactive compounds that cause thyroid tumours in rats associated with decreased circulating thyroid hormone levels due to increased hepatic metabolism (particularly Phase II conjugating enzymes such as uridine diphosphate (UDP) glucuronosyl transferases [UDPGTs] and glutathione S-transferases) and clearance. These compounds induce hepatic glucuronidation of thyroid hormones and increase biliary excretion of the conjugated hormones, resulting in decreased circulating T_3 and T_4 levels. As a result of the hypothyroid state, TSH levels increase and cause sustained thyroid follicular cell hyperplasia, leading to tumour formation.

While the basic physiology and feedback mechanisms of the hypothalamic–pituitary–thyroid axis are qualitatively similar across species, quantitative differences make rodents more sensitive than humans to development of thyroid cancer for which the sole mode of action is thyroid–pituitary disruption (U.S. EPA, 1998). These include the lack of a high-affinity thyroid binding globulin in rats relative to humans (Dohler *et al*., 1979), which likely affects the turnover of the hormone. With a more rapid turnover of T_4, there is a generalized increased activity of the pituitary–thyroid axis in rats compared with humans, which correlates with increased susceptibility to thyroid gland neoplasia.

Minimum criteria for establishment of this mode of action as a basis for tumour development include evidence of increases in thyroid growth and hormonal changes (the

latter including reduction in circulating serum T_4 and T_3 and an increase in TSH levels within days or a few weeks of exposure). Evidence of increases in thyroid growth is provided by measured increases in absolute or relative thyroid weight, histological indication of cellular hypertrophy and hyperplasia, morphometric determination of alteration in thyroid cellular components and changes in proliferation of follicular cells detected by DNA labelling or mitotic indices (U.S. EPA, 1998).

In the NTP bioassay (NTP, 1986a; Bucher *et al*., 1987) reported in the PSL1 assessment, increases in follicular cell adenomas and carcinomas (combined) were observed in female rats only, at 312 and 625 mg/kg-bw per day, and in female mice only, at 250 mg/kg-bw per day.

Available data relevant to assessment of the weight of evidence of induction of thyroid tumours in rats by SCCPs are limited to one study for which there is a published manuscript (Wyatt *et al*., 1993) and two investigations for which only a published summary report (Elcombe *et al*., 2000) or abstract (Elcombe *et al*., 1994) is available. In the study for which a complete account was published, effects on the thyroid were considered only in the control and highest dose groups; the administered dose for the latter was considerably greater than those in the NTP bioassay associated with thyroid tumours (i.e., 1000 mg/kg-bw per day versus 312 and 625 mg/kg-bw per day). In addition, in the abstract and summary accounts, quantitative data on effects or analyses were not presented. For example, Elcombe *et al*. (2000) reported only that male and female Fischer 344 rats were exposed by gavage in corn oil for up to 90 days at dose levels of 0, 312 or 625 mg/kg-bw per day and that "there were decreases in plasma thyroxine, increases in plasma TSH concentration and thyroid follicular cell hypertrophy and hyperplasia in both sexes." There are extremely limited data, therefore, to serve as a basis for consideration of concordance of dose–response between thyroid tumour induction and precursor effects in shorter-term studies, such as thyroid growth and hormonal changes. In a single additional study for which a full account is available (Hallgren and Darnerud, 1998), the dose level at which effects on thyroid hormonal T_4 levels or microsomal enzyme activity were not observed were much less than those administered in the NTP bioassay; as a result, these are not additionally meaningful in this context.

As a result, although data from the studies reported by Elcombe *et al*. (1994, 2000) and Wyatt *et al*. (1993) fulfil the criteria for tumour induction by thyroid disruption in part, it should be noted that these data are insufficient as a basis for analysis of dose–response for concordance with that for thyroid tumours. Also, recovery in the absence of continued exposure has not been investigated. In view of the limitations of both reporting and dose–response analyses, therefore, there is considerable uncertainty in attributing observed thyroid tumours to thyroid–pituitary disruption, to which rodents are more sensitive than humans.

B- Risk characterization

Available data relevant to consideration of the weight of evidence for proposed modes of

induction of liver, kidney and thyroid tumours associated with exposure to SCCPs, although limited, are suggestive that tolerable intakes that protect for non-neoplastic precursor effects will likely also be protective for cancer. However, owing principally to limited investigation of aspects such as recovery and inadequate documentation of relevant studies, there is considerable uncertainty in drawing this conclusion, particularly for the thyroid tumours. In recognition of this uncertainty, both neoplastic and non-neoplastic effects are considered here.

IPCS (1996) derived a TDI of 100 µg/kg-bw per day for non-neoplastic effects of SCCPs on the basis of the lowest reported No-Observed-Effect Level (NOEL) of 10 mg/kg-bw per day in a 13-week study in rats (IRDC, 1984a). At the next higher dose in the critical study (100 mg/kg-bw per day), there were increases in liver and kidney weight and hypertrophy of the liver and thyroid. In IPCS (1996), an uncertainty factor of 100 was applied in the development of the TDI to account for interspecies variation (×10) and intraspecies variation (×10). The potential for progression of lesions following longer-term exposure was not explicitly addressed in the development of the TDI. This is balanced to some degree by the relatively large margin between the NOEL and the LOEL (10-fold) in the critical study and the minimal severity of the effects at the next higher concentration; however, there is some justification for considering a somewhat lower value for the TDI.

On the basis of multistage modelling of the tumours with highest incidence (hepatocellular adenomas or carcinomas [combined] in male mice) in the carcinogenesis bioassay with SCCPs, IPCS (1996) also estimated the dose associated with a 5% increase in tumour incidence (Tumorigenic $Dose_{05}$ [TD_{05}]) to be 11 mg/kg-bw per day (amortized for period of administration).

The upper-bound estimate of exposure for the age group with greatest exposure to SCCPs (i.e., 26 µg/kg-bw per day) is within the range of the IPCS (1996) TDI, for which there is some justification for considering a somewhat lower value, to take into account potential progression of the lesions in longer-term studies.

The margin between the upper-bound estimate of exposure for the age group with greatest exposure to SCCPs and the Tumorigenic Dose (TD_{05}) (i.e., 440) is also considered inadequate in view of the uncertainty concerning mode of induction of tumours.

5.3.2 MCCPs

A TDI developed on the basis of the NOAEL (0.4 mg/kg-bw per day) in the more recent subchronic study conducted by Health Canada (Poon *et al*., 1995) would be similar to that derived for the PSL1 assessment (i.e., 6 µg/kg-bw per day).

Several of the highly uncertain bounding estimates of total daily intake of MCCPs from drinking water, food and soil for the general population of Canada exceed the TDI (6 µg/kg-bw per day) for non-neoplastic effects. Indeed, for infants not formula fed, the

total daily intake of MCCPs (i.e., 25.5 µg/kg-bw per day) exceeds the TDI by up to 4-fold.

5.3.3 LCCPs

None of the highly uncertain bounding estimates of total daily intake of LCCPs from drinking water, food and soil for the general population of Canada exceeds the TDI (71 µg/kg-bw per day) for non-neoplastic effects. However, for infants not formula fed, the total daily intake of LCCPs (16.8 µg/kg-bw per day) is within the same order of magnitude as the TDI.

5.4. Uncertainties and degree of confidence in human health risk characterization

There is low confidence in the upper-bounding estimates of exposure to all chlorinated paraffins. The estimates of intake for most age groups in the general Canadian population are based almost entirely upon limited sampling of foodstuffs in the United Kingdom, which were published in 1980. Methodology for analysis in this study is considered inadequate by present-day standards, and, as such, the data can be regarded at best as semi-quantitative. Reported concentrations represented both SCCPs and MCCPs, and, as a result, intake of the individual groups of chlorinated paraffins (SCCPs, MCCPs and LCCPs) from these sources has been overestimated.

The estimates of intake for SCCPs are based in part upon the results of more recent surveys, for which methods of analysis were more reliable (i.e., quantification by GC/ECNI-HRMS). Concentrations of SCCPs determined by HRMS were available for ambient air, water and samples of carp from Hamilton Harbour (intake from fish represented 38–58% of estimated total intake of SCCPs, although fish accounts for, at most, 4% of the total daily intake of food across the six age groups).

However, it is not possible to quantify the extent of overestimation of exposure based on the earlier, likely less selective analytical methodology, owing to lack of comparable data. Moreover, results based on analysis of the same samples by LRMS versus HRMS have been inconsistent, with levels of SCCPs being 1–2 orders of magnitude less for the latter in samples of whale blubber (Bennie *et al*., 2000; Tomy *et al*., 2000) and trout (Muir *et al*., 1999; Bennie *et al*., 2000) but slightly greater for the high-resolution analysis in carp (Muir *et al*., 1999; Bennie *et al*., 2000).

There is minimal confidence in the upper-bounding estimates of exposure to MCCPs. These estimates are based in large part upon concentrations reported in a limited number of foodstuffs in the United Kingdom, which were published in 1980. More recent, although limited, data on concentrations in trout analysed by LRMS were included in the calculation of upper-bounding estimates.

There is minimal confidence in the upper-bounding estimates of exposure to LCCPs. These estimates are based entirely upon concentrations reported in a limited number of

foodstuffs in the United Kingdom, which were published in 1980. Furthermore, concentrations in foods were represented by the limits of detection for five of eight food groups in the calculations of daily intake.

There is a low degree of confidence in the database of toxicological studies that serves as the basis for the assessment of the weight of evidence for mode of induction of tumours by SCCPs, for which only one published complete report (Wyatt *et al*., 1993) is available and for which it has not been possible to identify published accounts for reported pre-publication manuscripts reviewed in previous assessments. Results in the only fully documented study provide most meaningful support for the purported role of peroxisome proliferation in induction of liver tumours in rats and mice.

There is a moderate degree of confidence in the database of toxicological studies upon which the TDI for MCCPs is based, for which studies on chronic toxicity or carcinogenicity are lacking. The database for LCCPs is more complete, including a well-documented carcinogenicity bioassay in rats and mice.

6. CONCLUSIONS

6.1. SCCPs

On the basis of the available information, it is concluded that short-chain chlorinated paraffins (SCCPs) are entering, or may enter, 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; and,
- constitute or may constitute a danger in Canada to human life or health.

Therefore, it is concluded that short-chain chlorinated paraffins are “toxic” as defined under paragraphs 64 (a) and (c) of the *Canadian Environmental Protection Act, 1999*.

6.2. MCCPs

On the basis of the available information, it is concluded that medium-chain chlorinated paraffins (MCCPs) are entering, or may enter, the environment in quantities or concentrations or under conditions that:

- have or may have an immediate or long-term harmful effect on the environment or its biological diversity;and,
- constitute or may constitute a danger in Canada to human life or health.

Therefore, it is concluded that medium-chain chlorinated paraffins are “toxic” as defined in paragraphs 64 (a) and (c) of the *Canadian Environmental Protection Act, 1999.*

6.3. LCCPs

On the basis of the limited available data, it is concluded that long-chain chlorinated paraffins up to C_{20} are entering, or may enter, the environment in quantities or concentrations or under conditions that:

- have or may have an immediate or long-term harmful effect on the environment or its biological diversity.

Therefore, it is concluded that long-chain chlorinated paraffins up to C_{20} are "toxic" as defined in paragraphs 64 (a) of the *Canadian Environmental Protection Act, 1999.*

In addition, on the basis of the limited available data, it is concluded that long-chain chlorinated paraffins are entering, or may enter, the environment in quantities or concentrations or under conditions that:

- constitute or may constitute a danger in Canada to human life or health.

Therefore, it is concluded that long-chain chlorinated paraffins are "toxic" as defined in paragraph 64 (c) of the *Canadian Environmental Protection Act, 1999.*

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