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Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade

Chemical Review Committee
Tenth meeting
Rome, 21–24 October 2014
Item 4(c)(ii) of the provisional agenda*

Technical work: Review of notifications of final regulatory actions Short-chained chlorinated paraffins

ADVANCE COPY

Short-chained chlorinated paraffins: Supporting documentation provided by Canada

Note by the Secretariat

The Secretariat has the honour to provide, in the annex to the present note, documentation received from Canada to support its notification of final regulatory action for Short-chained chlorinated paraffins as an industrial chemical. The documentation has not been formally edited.

* UNEP/FAO/RC/CRC.10/1.

Annex

Supporting documentation provided by Canada: Short-chained chlorinated paraffins

- *Prohibition of Certain Toxic Substances Regulations, 2012. Canada Gazette*, Part II, Vol. 147, No. 1 — January 2, 2013.

- Government of Canada. 1993. Priority Substances List assessment report. Chlorinated paraffins. Minister of Supply and Services, Ottawa, Ontario.

- Proposed Risk Management Approach for Chlorinated Paraffins (August 2008)

- Government of Canada. 2004. Follow-up Report on a PSL1 Substance for Which There Was Insufficient Information to Conclude Whether the Substance Constitutes a Danger to the Environment. Ottawa, Ontario.

- Government of Canada. 2003. Follow-up Report on a PSL1 Substance for Which Data Were Insufficient to Conclude Whether the Substance Was “Toxic” to Human Health Chlorinated Paraffins. Ottawa, Ontario.

- Government of Canada. 2008. 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. Ottawa, Ontario.

***Prohibition of Certain Toxic Substances Regulations, 2012. Canada Gazette*, Part II, Vol. 147, No. 1 — January 2, 2013.**

CANADA

CONSOLIDATION

CODIFICATION

Prohibition of Certain Toxic Substances Regulations, 2012

Règlement sur certaines substances toxiques interdites (2012)

SOR/2012-285

DORS/2012-285

Current to May 27, 2014

À jour au 27 mai 2014

Last amended on March 14, 2013

Dernière modification le 14 mars 2013

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OFFICIAL STATUS OF CONSOLIDATIONS

Subsections 31(1) and (3) of the *Legislation Revision and Consolidation Act*, in force on June 1, 2009, provide as follows:

Published consolidation is evidence

31. (1) Every copy of a consolidated statute or consolidated regulation published by the Minister under this Act in either print or electronic form is evidence of that statute or regulation and of its contents and every copy purporting to be published by the Minister is deemed to be so published, unless the contrary is shown.

...

Inconsistencies in regulations

(3) In the event of an inconsistency between a consolidated regulation published by the Minister under this Act and the original regulation or a subsequent amendment as registered by the Clerk of the Privy Council under the *Statutory Instruments Act*, the original regulation or amendment prevails to the extent of the inconsistency.

NOTE

This consolidation is current to May 27, 2014. The last amendments came into force on March 14, 2013. Any amendments that were not in force as of May 27, 2014 are set out at the end of this document under the heading "Amendments Not in Force".

CARACTÈRE OFFICIEL DES CODIFICATIONS

Les paragraphes 31(1) et (3) de la *Loi sur la révision et la codification des textes législatifs*, en vigueur le 1er juin 2009, prévoient ce qui suit :

Codifications comme élément de preuve

31. (1) Tout exemplaire d'une loi codifiée ou d'un règlement codifié, publié par le ministre en vertu de la présente loi sur support papier ou sur support électronique, fait foi de cette loi ou de ce règlement et de son contenu. Tout exemplaire donné comme publié par le ministre est réputé avoir été ainsi publié, sauf preuve contraire.

[...]

Incompatibilité — règlements

(3) Les dispositions du règlement d'origine avec ses modifications subséquentes enregistrées par le greffier du Conseil privé en vertu de la *Loi sur les textes réglementaires* l'emportent sur les dispositions incompatibles du règlement codifié publié par le ministre en vertu de la présente loi.

NOTE

Cette codification est à jour au 27 mai 2014. Les dernières modifications sont entrées en vigueur le 14 mars 2013. Toutes modifications qui n'étaient pas en vigueur au 27 mai 2014 sont énoncées à la fin de ce document sous le titre « Modifications non en vigueur ».

TABLE OF PROVISIONS

TABLE ANALYTIQUE

Registration
SOR/2012-285 December 14, 2012

CANADIAN ENVIRONMENTAL PROTECTION ACT, 1999

Prohibition of Certain Toxic Substances Regulations, 2012

P.C. 2012-1714 December 13, 2012

Whereas, pursuant to subsection 332(1)[a] of the *Canadian Environmental Protection Act, 1999*[b], the Minister of the Environment published in the *Canada Gazette*, Part I, on July 23, 2011, a copy of the proposed *Prohibition of Certain Toxic Substances Regulations, 2012*, substantially in the annexed form, and persons were given an opportunity to file comments with respect to the proposed Regulations or to file a notice of objection requesting that a board of review be established and stating the reasons for the objection;

Whereas, pursuant to subsection 93(3) of that Act, the National Advisory Committee has been given an opportunity to provide its advice under section 6[c] of that Act;

And whereas, in the opinion of the Governor in Council, pursuant to subsection 93(4) of that Act, the proposed Regulations do not regulate an aspect of a substance that is regulated by or under any other Act of Parliament in a manner that provides, in the opinion of the Governor in Council, sufficient protection to the environment and human health;

Therefore, His Excellency the Governor General in Council, on the recommendation of the Minister of the Environment and the Minister of Health, pursuant to subsection 93(1) of the *Canadian Environmental Protection Act, 1999*[b], makes the annexed *Prohibition of Certain Toxic Substances Regulations, 2012*.

[a] S.C. 2004, c. 15, s. 31
[b] S.C. 1999, c. 33
[c] S.C. 2002, c. 7, s. 124

Enregistrement
DORS/2012-285 Le 14 décembre 2012

LOI CANADIENNE SUR LA PROTECTION DE L'ENVIRONNEMENT (1999)

Règlement sur certaines substances toxiques interdites (2012)

C.P. 2012-1714 Le 13 décembre 2012

Attendu que, conformément au paragraphe 332(1)[a] de la *Loi canadienne sur la protection de l'environnement (1999)*[b], le ministre de l'Environnement a fait publier dans la *Gazette du Canada* Partie I, le 23 juillet 2011, le projet de règlement intitulé *Règlement sur certaines substances toxiques interdites (2012)*, conforme en substance au texte ci-après, et que les intéressés ont ainsi eu la possibilité de présenter leurs observations à cet égard ou un avis d'opposition motivé demandant la constitution d'une commission de révision;

Attendu que, conformément au paragraphe 93(3) de cette loi, le comité consultatif national s'est vu accorder la possibilité de formuler ses conseils dans le cadre de l'article 6[c] de celle-ci;

Attendu que le gouverneur en conseil est d'avis que, aux termes du paragraphe 93(4) de cette loi, le projet de règlement ne vise pas un point déjà réglementé sous le régime d'une autre loi fédérale de manière à offrir une protection suffisante pour l'environnement et la santé humaine,

À ces causes, sur recommandation du ministre de l'Environnement et de la ministre de la Santé et en vertu du paragraphe 93(1) de la *Loi canadienne sur la protection de l'environnement (1999)*[b], Son Excellence le Gouverneur général en conseil prend le *Règlement sur certaines substances toxiques interdites (2012)*, ci-après.

[a] L.C. 2004, ch. 15, art. 31
[b] L.C. 1999, ch. 33
[c] L.C. 2002, ch. 7, art. 124

PROHIBITION OF CERTAIN TOXIC SUBSTANCES REGULATIONS, 2012

APPLICATION

Application

1. Subject to sections 2 and 3, these Regulations apply to toxic substances that are both specified in the List of Toxic Substances in Schedule 1 to the *Canadian Environmental Protection Act, 1999* and set out in either Schedule 1 or 2 to these Regulations.

Non-application — substance

2. These Regulations do not apply to any toxic substance that

(*a*) is contained in a hazardous waste, hazardous recyclable material or non-hazardous waste to which Division 8 of Part 7 of the *Canadian Environmental Protection Act, 1999* applies;

(*b*) is contained in a pest control product as defined in subsection 2(1) of the *Pest Control Products Act*; or

(*c*) is present as a contaminant in a chemical feedstock that is used in a process from which there are no releases of the toxic substance and on the condition that the toxic substance is destroyed or completely converted in that process to a substance that is not a toxic substance set out in either Schedule 1 or 2.

Non-application — use

3. (1) These Regulations, except for subsections (2) to (4), do not apply to any toxic substance or to any product containing it that is to be used in a laboratory for analysis, in scientific research or as a laboratory analytical standard.

RÈGLEMENT SUR CERTAINES SUBSTANCES TOXIQUES INTERDITES (2012)

CHAMP D'APPLICATION

Application

1. Sous réserve des articles 2 et 3, le présent règlement s'applique aux substances toxiques qui sont à la fois mentionnées aux annexes 1 ou 2 et inscrites sur la liste des substances toxiques de l'annexe 1 de la *Loi canadienne sur la protection de l'environnement (1999)*.

Non-application — substance

2. Le présent règlement ne s'applique pas aux substances toxiques suivantes :

a) celles qui sont contenues dans des déchets dangereux, des matières recyclables dangereuses ou des déchets non dangereux auxquels s'applique la section 8 de la partie 7 de la *Loi canadienne sur la protection de l'environnement (1999)*;

b) celles qui sont contenues dans un produit antiparasitaire au sens du paragraphe 2(1) de la *Loi sur les produits antiparasitaires*;

c) celles qui sont présentes comme contaminants dans une matière première chimique utilisée au cours d'un processus n'occasionnant aucun rejet de telles substances toxiques, pourvu qu'elles soient, au cours de ce processus, détruites ou totalement converties en toute substance autre que celles mentionnées aux annexes 1 ou 2.

Non-application — utilisation

3. (1) Le présent règlement, sauf les paragraphes (2) à (4), ne s'applique pas aux substances toxiques ni aux produits qui en contiennent qui sont destinés à être utilisés pour des analyses en laboratoire, pour la recherche scientifique ou en tant qu'étalon analytique de laboratoire.

Information to Minister — more than 10 g

(2) Every person must submit to the Minister in any calendar year the information set out in Schedule 3 for each toxic substance or a product containing it that they intend to use for a purpose referred to in subsection (1) as soon as feasible before the use of more than 10 g of the substance in that calendar year. The information must be submitted only once in a calendar year in respect of each substance or product.

Renseignements au ministre — plus de 10 g

(2) Toute personne présente au ministre, au cours d'une année civile, les renseignements prévus à l'annexe 3 pour chaque substance toxique ou produit qui en contient qu'elle prévoit utiliser à l'une des fins visées au paragraphe (1) dès que possible avant d'utiliser plus de 10 g de la substance au cours de l'année civile. Ces renseignements sont présentés une seule fois pour chaque substance ou produit dans une année civile.

On-going use

(3) Any person that, on the day on which these Regulations come into force, is using a toxic substance or product referred to in subsection (1) for a purpose referred to in that subsection must, if the quantity of the toxic substance used, by itself or in a product, exceeded 10 g in the calendar year of the coming into force of these Regulations, submit to the Minister, within 60 days after the day on which these Regulations come into force, the information referred to in Schedule 3. The information must be submitted only once in a calendar year in respect of each substance or product.

Utilisations en cours

(3) Toute personne qui, à la date d'entrée en vigueur du présent règlement, utilise une substance toxique ou un produit qui en contient à l'une des fins visées au paragraphe (1) présente au ministre les renseignements prévus à l'annexe 3, dans les soixante jours suivant cette date, si la quantité de la substance toxique utilisée — seule ou dans un produit — depuis le début de l'année civile en cours au moment de l'entrée en vigueur a dépassé 10 g. Ces renseignements sont présentés une seule fois pour chaque substance ou produit dans une année civile.

Addition of substance

(4) If, after the coming into force of these Regulations, a toxic substance is added to Schedule 1 or 2, any person that, on the day on which the Regulations adding the toxic substance come into force, is using the toxic substance or a product containing it for a purpose referred to in subsection (1) must, if the quantity of the toxic substance used, by itself or in a product, exceeded 10 g in the calendar year of the coming into force of the Regulations adding the toxic substance, submit to the Minister, within 60 days after the day on which those Regulations come into force, the information referred to in Schedule 3. The information must be submitted only

Ajout d'une substance

(4) Si une substance toxique est ajoutée aux annexes 1 ou 2 après l'entrée en vigueur du présent règlement, toute personne qui, à la date d'entrée en vigueur du règlement visant à ajouter la substance, utilise la substance ou un produit qui en contient à l'une des fins visées au paragraphe (1) présente au ministre les renseignements prévus à l'annexe 3, dans les soixante jours suivant cette date, si la quantité de la substance toxique utilisée — seule ou dans un produit — depuis le début de l'année civile en cours au moment de l'entrée en vigueur du règlement visant à ajouter la substance a dépassé 10 g. Ces renseignements sont pré-

once in a calendar year in respect of each substance or product.

sentés une seule fois pour chaque substance ou produit dans une année civile.

PROHIBITIONS AND PERMITTED ACTIVITIES

INTERDICTIONS ET ACTIVITÉS PERMISES

Toxic substance — Schedule 1

4. (1) Subject to sections 5 and 9, a person must not manufacture, use, sell, offer for sale or import a toxic substance set out in Schedule 1 or a product containing it unless the toxic substance is incidentally present.

Substance toxique — annexe 1

4. (1) Sous réserve des articles 5 et 9, il est interdit de fabriquer, d'utiliser, de vendre, de mettre en vente ou d'importer toute substance toxique mentionnée à l'annexe 1 ou tout produit qui en contient, à moins que celle-ci n'y soit présente fortuitement.

Non application

(2) Subsection (1) does not apply to a product that is a manufactured item that is formed into a specific physical shape or design during its manufacture and that has, for its final use, a function or functions dependent in whole or in part on its shape or design, if a toxic substance set out in Part 2 of Schedule 1 is present in that product.

Non-application

(2) Le paragraphe (1) ne s'applique pas aux produits qui sont des articles manufacturés dotés d'une forme ou de caractéristiques matérielles précises pendant leur fabrication et ayant, pour leur utilisation finale, une ou plusieurs fonctions en dépendant en tout ou en partie si une substance toxique mentionnée à la partie 2 de l'annexe 1 est présente dans ces produits.

Exception — manufactured or imported before coming into force

5. A person may use, sell, or offer for sale a product containing a toxic substance set out in item 11 or 12 of Part 1 of Schedule 1 if the product is manufactured or imported before the day on which these Regulations come into force.

Exception — fabrication ou importation précédant l'entrée en vigueur

5. Il est permis d'utiliser, de vendre et de mettre en vente tout produit contenant la substance toxique mentionnée aux articles 11 ou 12 de la partie 1 de l'annexe 1 qui a été fabriqué ou importé avant l'entrée en vigueur du présent règlement.

Toxic substance — Schedule 2

6. (1) Subject to subsections (2) and (4) and sections 7 and 9, a person must not manufacture, use, sell, offer for sale or import a toxic substance set out in column 1 of Part 1, 2 or 3 of Schedule 2 or a product containing it unless the toxic substance is incidentally present.

Substance toxique — annexe 2

6. (1) Sous réserve des paragraphes (2) et (4) et des articles 7 et 9, il est interdit de fabriquer, d'utiliser, de vendre, de mettre en vente ou d'importer toute substance toxique mentionnée à la colonne 1 de la partie 1, 2 ou 3 de l'annexe 2 ou tout produit qui en contient, à moins que celle-ci n'y soit présente fortuitement.

Permitted activities — Schedule 2

(2) The prohibition to manufacture, use, sell, offer for sale or import a toxic substance set out in column 1 of Part 1, 2 or 3 of Schedule 2 or a product containing it does not apply if

Activités permises — annexe 2

(2) L'interdiction de fabriquer, d'utiliser, de vendre, de mettre en vente ou d'importer toute substance toxique mentionnée à la colonne 1 de la partie 1, 2 ou 3 de l'annexe 2 ou tout produit qui en contient ne s'applique pas dans les cas suivants :

(*a*) the toxic substance set out in column 1 of Part 1 of Schedule 2 or a product containing it, other than a substance or product set out in item 3 or 4, is designed for a use set out in column 2;

(*b*) the toxic substance set out in column 1 of Part 2 of Schedule 2 or a product containing it is designed for a use set out in column 2 and that activity occurs before the date set out in column 3; or

(*c*) a product set out in column 2 of Part 3 of Schedule 2 containing the toxic substance set out in column 1 in a concentration less than or equal to that set out in column 3 including any incidental presence of the substance.

a) la substance toxique mentionnée à la colonne 1 de la partie 1 de l'annexe 2 — sauf celles mentionnées aux articles 3 et 4 de cette partie — ou le produit qui en contient sont destinés à une utilisation prévue à la colonne 2;

b) la substance toxique mentionnée à la colonne 1 de la partie 2 de l'annexe 2 ou le produit qui en contient sont destinés à l'une des utilisations prévues à la colonne 2 et l'activité en cause se déroule avant la date prévue à la colonne 3;

c) le produit mentionné à la colonne 2 de la partie 3 de l'annexe 2 contient la substance toxique mentionnée à la colonne 1 en une concentration inférieure ou égale à celle prévue à la colonne 3, compte tenu de toute présence fortuite de la substance.

Exception — incidental presence

(3) For greater certainty, the exception of the incidental presence referred to in subsection (1) does not apply in the case of a product described in paragraph (2)(*c*).

Précisions

(3) Il est entendu que l'exception relative à la présence fortuite prévue au paragraphe (1) ne s'applique pas dans le cas d'un produit visé à l'alinéa (2)*c*).

Exception — permitted use

(4) The prohibition to use a product that contains a toxic substance set out in column 1 of Part 1 of Schedule 2 does not apply to a product set out in item 3 or 4 of column 2.

Exception — Utilisation permise

(4) L'interdiction d'utiliser un produit qui contient une substance toxique mentionnée à la colonne 1 de la partie 1 de l'annexe 2 ne s'applique pas aux produits visés aux articles 3 et 4 de la colonne 2 de cette partie.

Exception — personal use

(5) Subsection (1) does not apply to the use or import of a product containing a toxic substance set out in item 1 of Part 2 of Schedule 2, if the product is used or intended to be used for a personal use.

Exception — Usage personnel

(5) Le paragraphe (1) ne s'applique pas à l'importation ou à l'utilisation d'un produit contenant des substances toxiques mentionnées à l'article 1 de la partie 2 de l'annexe 2 qui est utilisé à des fins personnelles ou destiné à l'être.

Exception — temporary permitted uses

7. (1) A person may use, sell, or offer for sale a product set out in column 2 of Part 2 of Schedule 2 containing a toxic substance set out in column 1 of Part 2 of Schedule 2 if the product is manufactured

Exception — utilisations permises temporairement

7. (1) Il est permis d'utiliser, de vendre et de mettre en vente le produit visé à la colonne 2 de la partie 2 de l'annexe 2 contenant la substance toxique mentionnée à la colonne 1 de la partie 2 de l'annexe 2

or imported before the expiry date set out in column 3 of Schedule 2.

qui a été fabriqué ou importé avant la date d'expiration prévue à la colonne 3 de la partie 2.

Exception — Tributyltins

(2) A person may use, sell, or offer for sale a product containing a toxic substance set out in item 2 of Part 3 of Schedule 2 if it is manufactured or imported before the day on which these Regulations come into force.

(2) Il est permis d'utiliser, de vendre et de mettre en vente tout produit contenant la substance toxique mentionnée à l'article 2 de la partie 3 de l'annexe 2 qui a été fabriqué ou importé avant l'entrée en vigueur du présent règlement.

Exception — tributylétains

Exception — manufacture or import under permit

8. A person may use, sell or offer for sale a toxic substance or a product containing it, if the substance or the product is manufactured or imported in accordance with a permit that is issued under section 10.

8. Il est permis d'utiliser, de vendre et de mettre en vente les substances toxiques ou les produits en contenant qui ont été fabriqués ou importés conformément à un permis délivré aux termes de l'article 10.

Exception — fabrication ou importation conformément à un permis

PERMITS

PERMIS

Application

Demande

Requirement for permit

9. (1) Any person that is a manufacturer or importer of a toxic substance or a product containing it that is prohibited under section 4 or 6, on the day on which these Regulations come into force, may continue to manufacture or import the substance or product if they have been issued a permit under section 10.

9. (1) Toute personne qui, à la date d'entrée en vigueur du présent règlement, est un fabricant ou un importateur de substances toxiques visées par l'interdiction prévue aux articles 4 ou 6 ou de produits qui en contiennent peut continuer de fabriquer ou d'importer ces substances ou ces produits si un permis lui a été délivré aux termes de l'article 10.

Permis exigé

Addition of substance

(2) In the case of a toxic substance added either to Schedule 1 and prohibited under section 4, or added to Schedule 2 and prohibited under section 6, any person that is a manufacturer or importer of a toxic substance or a product containing it, on the day on which the Regulations adding the toxic substance come into force may continue to manufacture or import the substance or a product containing it if they have been issued a permit under section 10.

(2) Dans le cas d'une substance toxique qui est soit ajoutée à l'annexe 1 et visée par l'interdiction prévue à l'article 4, soit ajoutée à l'annexe 2 et visée par l'interdiction prévue à l'article 6, toute personne qui, à la date d'entrée en vigueur du règlement visant à ajouter la substance, est un fabricant ou un importateur d'une telle substance ou d'un produit qui en contient peut continuer de fabriquer ou d'importer cette substance ou ce produit si un permis lui a été délivré aux termes de l'article 10.

Ajout d'une substance

Temporary permitted uses

(3) Any person that manufactures or imports a toxic substance set out in Part 2 of Schedule 2 or a product containing it under paragraph 6(2)(*b*), on the day on which the period set out under that paragraph for which a temporary permitted use expires, may continue that activity if they have been issued a permit under section 10.

Utilisations permises temporairement

(3) Toute personne qui, à la date où expire la période pendant laquelle l'utilisation visée à l'alinéa 6(2)*b*) était permise temporairement, fabrique ou importe, aux termes de cet alinéa, une substance toxique mentionnée à la partie 2 de l'annexe 2 ou un produit qui en contient, peut poursuivre cette activité si un permis lui a été délivré aux termes de l'article 10.

Required information

(4) An application for a permit must be submitted to the Minister and contain the information referred to in Schedule 4.

Renseignements exigés

(4) La demande de permis est présentée au ministre et comporte les renseignements prévus à l'annexe 4.

Conditions of Issuance

Conditions de délivrance

Issuance

10. (1) Subject to subsection (2), the Minister must issue the permit if the following conditions are met:

(*a*) there is no technically or economically feasible alternative or substitute available to the applicant at the time of the application, other than a substance regulated under these Regulations, for the toxic substance;

(*b*) the applicant has taken the necessary measures to minimize or eliminate any harmful effect of the toxic substance on the environment and human health; and

(*c*) a plan has been prepared respecting the toxic substance identifying the measures that will be taken by the applicant to comply with these Regulations, and the period within which the plan is to be implemented does not exceed three years after the day on which a permit is first issued to the applicant.

Délivrance

10. (1) Sous réserve du paragraphe (2), le ministre délivre le permis si les conditions suivantes sont réunies :

a) au moment de la demande, le demandeur n'est pas en mesure, sur le plan technique ou économique, de remplacer la substance toxique par une substance non visée par le présent règlement ou d'utiliser une solution de rechange;

b) le demandeur a pris les mesures nécessaires pour éliminer ou réduire au minimum les effets nocifs de la substance toxique sur l'environnement et la santé humaine;

c) un plan a été élaboré à l'égard de la substance toxique comportant les mesures que le demandeur prendra pour se conformer au présent règlement et le délai prévu pour son exécution n'excède pas trois ans à compter de la date initiale de délivrance du permis.

Grounds for refusing permit

(2) The Minister must refuse to issue a permit if

(*a*) the Minister has reasonable grounds to believe that the applicant has provided

Refus

(2) Le ministre refuse de délivrer le permis dans les cas suivants :

a) il a des motifs raisonnables de croire que le demandeur a fourni des rensei-

false or misleading information in support of their application; or

(*b*) information required under subsection 9(4) has not been provided or is insufficient to enable the Minister to process the application.

gnements faux ou trompeurs à l'appui de sa demande;

b) les renseignements exigés aux termes du paragraphe 9(4) n'ont pas été fournis ou sont insuffisants pour lui permettre de traiter la demande.

Expiry and permit renewal

(3) A permit expires 12 months after the day on which it is issued unless, at least 30 days before the day on which the permit expires, the applicant submits an application for renewal to the Minister that contains the information referred to in Schedule 4. The validity of the first permit may only be extended twice, subject to the same conditions.

(3) Le permis expire douze mois après la date de sa délivrance, sauf si le demandeur présente au ministre une demande qui comporte les renseignements prévus à l'annexe 4 pour le renouvellement de celui-ci au moins trente jours avant son expiration. Le permis ne peut être renouvelé que deux fois aux mêmes conditions.

Expiration et demande de renouvellement

Revocation

Révocation

Revocation

11. (1) The Minister must revoke a permit if the conditions set out in paragraphs 10(1)(*a*) to (*c*) are no longer met or if the Minister has reasonable grounds to believe that the permit holder has provided false or misleading information to the Minister.

11. (1) Le ministre révoque le permis si les conditions prévues aux alinéas 10(1)*a*) à *c*) ne sont plus respectées ou s'il a des motifs raisonnables de croire que le titulaire du permis lui a fourni des renseignements faux ou trompeurs.

Révocation

Conditions for revocation

(2) The Minister must not revoke a permit unless the Minister has provided the permit holder with

(*a*) written reasons for the revocation; and

(*b*) an opportunity to be heard, by written representation, in respect of the revocation.

(2) Il ne peut révoquer le permis qu'après :

a) avoir avisé par écrit le titulaire des motifs de la révocation;

b) lui avoir donné la possibilité de présenter des observations écrites au sujet de la révocation.

Conditions de révocation

ANNUAL REPORTS

RAPPORT ANNUEL

Certain substances

12. Every person that manufactures or imports a toxic substance set out in column 1 of Part 4 of Schedule 2 or a product containing it, whether incidentally or not, must submit to the Minister a report that contains the information referred to in

12. Toute personne qui fabrique ou importe une substance toxique mentionnée à la colonne 1 de la partie 4 de l'annexe 2 ou un produit qui en contient, fortuitement ou non, présente au ministre un rapport contenant les renseignements prévus à l'an-

Certaines substances

Schedule 5 by March 31 following the end of the calendar year during which either the toxic substance or a product containing it was manufactured or imported if, in that year

(*a*) the total annual quantity of the toxic substance manufactured or imported was equal to or greater than that set out in column 2, if any;

(*b*) the product imported contained the toxic substance in an annual weighted average concentration equal to or greater than that set out in column 3, if any; or

(*c*) the total annual quantity of the toxic substance contained in a product manufactured or imported and its annual weighted average concentration in the product were equal to or greater than those set out in column 4, if any.

nexe 5 au plus tard le 31 mars suivant la fin de l'année civile durant laquelle la substance toxique ou le produit qui en contient a été fabriqué ou importé si, au cours de cette année :

a) la quantité totale annuelle de la substance toxique fabriquée ou importée était égale ou supérieure à celle prévue à la colonne 2, le cas échéant;

b) la concentration moyenne pondérée annuelle de la substance toxique dans le produit importé était égale ou supérieure à celle prévue à la colonne 3, le cas échéant;

c) la quantité totale annuelle de la substance toxique contenue dans un produit fabriqué ou importé et la concentration moyenne pondérée annuelle de la substance toxique dans le produit étaient toutes deux égales ou supérieures à celles prévues à la colonne 4, le cas échéant.

ACCREDITED LABORATORY

Accredited laboratory

13. Any concentration or quantity to be determined under these Regulations must be determined, in accordance with generally accepted standards of scientific practice, by a laboratory that is accredited under the International Organization for Standardization standard ISO/IEC 17025:2005, entitled *General requirements for the competence of testing and calibration laboratories*, as amended from time to time, or by a laboratory that meets an equivalent standard.

LABORATOIRE ACCRÉDITÉ

Laboratoire accrédité

13. Pour l'application du présent règlement, la concentration et la quantité sont déterminées conformément aux exigences de pratiques scientifiques généralement reconnues par un laboratoire qui est accrédité selon la norme de l'Organisation internationale de normalisation ISO/CEI 17025:2005, intitulée *Exigences générales concernant la compétence des laboratoires d'étalonnages et d'essais*, avec ses modifications successives, ou par un laboratoire qui répond à une norme équivalente.

PRESENTATION OF INFORMATION

Certification

14. (1) Any information or an application for a permit required to be submitted

PRÉSENTATION DES RENSEIGNEMENTS

Attestation

14. (1) Tout renseignement ou toute demande de permis devant être fourni au mi-

to the Minister under these Regulations must bear the signature of the interested person or their authorized representative and be accompanied by a certification dated and signed by the interested person or the person authorized to act on their behalf, stating that the information is accurate and complete.

nistre en application du présent règlement porte la signature de l'intéressé ou de la personne autorisée à agir en son nom et est accompagné d'une attestation, datée et signée par l'intéressé ou par la personne autorisée à agir en son nom, portant que les renseignements sont complets et exacts.

Writing or electronic format

(2) The information, application for a permit and certification may be submitted either in writing or in an electronic format that is compatible with the one that is used by the Minister.

Support papier ou électronique

(2) Les renseignements, la demande de permis et l'attestation peuvent être présentés sur un support papier ou sur un support électronique compatible avec celui utilisé par le ministre.

RECORD KEEPING

REGISTRES

Records

15. (1) Every person that submits information to the Minister under these Regulations must keep a record containing a copy of that information, a copy of the certification and any documents supporting the information, including test data if applicable, for a period of at least five years beginning on the date of the submission of the information.

Registres

15. (1) La personne qui présente au ministre des renseignements en application du présent règlement conserve copie de ceux-ci et de l'attestation, ainsi que tout document à l'appui, y compris, s'il y a lieu, des données d'analyse, dans un registre pendant au moins cinq ans à compter de la date de leur présentation.

Location

(2) The records must be kept at the person's principal place of business in Canada or, on notification to the Minister, at any other place in Canada where the records can be inspected.

Lieu de conservation

(2) Les registres sont conservés à l'établissement principal de la personne au Canada ou en tout autre lieu au Canada dont le ministre a été avisé et où ils peuvent être examinés.

TRANSITIONAL

DISPOSITIONS TRANSITOIRES

Activity referred to in *Prohibition of Certain Toxic Substances Regulations, 2005*

16. A permit must not be obtained under these Regulations for an activity prohibited under the *Prohibition of Certain Toxic Substances Regulations, 2005*.

Activités visées par le *Règlement sur certaines substances toxiques interdites (2005)*

16. Un permis ne peut être obtenu en vertu du présent règlement à l'égard d'une activité interdite aux termes du *Règlement sur certaines substances toxiques interdites (2005)*.

REPEAL

17. [Repeal]

ABROGATION

17. [Abrogation]

COMING INTO FORCE

Three months after registration

18. These Regulations come into force three months after the day on which they are registered.

ENTRÉE EN VIGUEUR

Trois mois après la date de l'enregistrement

18. Le présent règlement entre en vigueur trois mois après la date de son enregistrement.

SCHEDULE 1
(Sections 1 to 5 and 9 and Schedule 3)

PART 1

PROHIBITED TOXIC SUBSTANCES

Item	Toxic Substance
1.	Dodecachloropentacyclo [5.3.0.$0^{2,6}$.$0^{3,9}$.$0^{4,8}$] decane (Mirex)
2.	Polybrominated Biphenyls that have the molecular formula $C_{12}H_{(10-n)}Br_n$ in which "n" is greater than 2
3.	Polychlorinated Terphenyls that have the molecular formula $C_{18}H_{(14-n)}Cl_n$ in which "n" is greater than 2
4.	Bis(chloromethyl) ether that has the molecular formula $C_2H_4Cl_2O$
5.	Chloromethyl methyl ether that has the molecular formula C_2H_5ClO
6.	(4-Chlorophenyl) cyclopropylmethanone, O-[(4-nitrophenyl)methyl] oxime that has the molecular formula $C_{17}H_{15}ClN_2O_3$
7.	*N*-Nitrosodimethylamine, which has the molecular formula $C_2H_6N_2O$
8.	Hexachlorobutadiene, which has the molecular formula C_4Cl_6
9.	Dichlorodiphenyltrichloroethane (DDT), which has the molecular formula $C_{14}H_9Cl_5$
10.	Hexachlorobenzene
11.	Polychlorinated naphthalenes, which have the molecular formula $C_{10}H_{8-n}Cl_n$ in which "n" is greater than 1
12.	Chlorinated alkanes that have the molecular formula $C_nH_xCl_{(2n+2-x)}$ in which $10 \leq n \leq 13$

PART 2

PROHIBITED TOXIC SUBSTANCES UNLESS PRESENT IN MANUFACTURED ITEMS

Item	Toxic Substance
1.	Hexane, 1,6-diisocyanato-, homopolymer, reaction products with alpha-fluoro-omega-2-hydroxyethyl-poly(difluoromethylene), C16-20-branched alcohols and 1-octadecanol
2.	2-Propenoic acid, 2-methyl-, hexadecyl ester, polymers with 2-hydroxyethyl methacrylate, gamma-omega-perfluoro-C10-16-alkyl acrylate and stearyl methacrylate
3.	2-Propenoic acid, 2-methyl-, 2-methylpropyl ester, polymer with butyl 2-propenoate and 2,5 furandione, gamma-omega-perfluoro-C8-14-alkyl esters, tert-Bu benzenecarboperoxoate-initiated
4.	2-Propen-1-ol, reaction products with pentafluoroiodoethane tetrafluoroethylene telomer, dehydroiodinated, reaction products with epichlorohydrin and triethylenetetramine

ANNEXE 1
(articles 1 à 5 et 9 et annexe 3)

PARTIE 1

SUBSTANCES TOXIQUES INTERDITES

Article	Substance toxique
1.	Dodécachloropentacyclo [5.3.0.$0^{2,6}$.$0^{3,9}$.$0^{4,8}$] décane (mirex)
2.	Les biphényles polybromés dont la formule moléculaire est $C_{12}H_{(10-n)}Br_n$, où « n » est plus grand que 2
3.	Les triphényles polychlorés dont la formule moléculaire est $C_{18}H_{(14-n)}Cl_n$, où « n » est plus grand que 2
4.	Éther bis(chlorométhylique) dont la formule moléculaire est $C_2H_4Cl_2O$
5.	Oxyde de chlorométhyle et de méthyle dont la formule moléculaire est C_2H_5ClO
6.	Le (4-chlorophényle) cyclopropylméthanone, O-[(4-nitrophényle)méthyl]oxime dont la formule moléculaire est $C_{17}H_{15}ClN_2O_3$
7.	*N*-Nitrosodiméthylamine, dont la formule moléculaire est $C_2H_6N_2O$
8.	Hexachlorobutadiène, dont la formule moléculaire est C_4Cl_6
9.	Dichlorodiphényltrichloroéthane (DDT), dont la formule moléculaire est $C_{14}H_9Cl_5$
10.	Hexachlorobenzène
11.	Naphtalènes polychlorés, dont la formule moléculaire est $C_{10}H_{8-n}Cl_n$, où « n » est plus grand que 1
12.	Alcanes chlorés dont la formule moléculaire est $C_nH_xCl_{(2n+2-x)}$, où $10 \leq n \leq 13$

PARTIE 2

SUBSTANCES TOXIQUES INTERDITES SAUF SI ELLES SONT PRÉSENTES DANS UN ARTICLE MANUFACTURÉ

Article	Substance toxique
1.	1,6-Diisocyanatohexane, homopolymérisé, produits de réaction avec l'alpha fluoro oméga-(2-hydroxyéthyl)-poly(difluorométhylène), des alcools ramifiés en C16-20 et l'octadécan-1-ol
2.	Méthacrylate d'hexadécyle, polymères avec le méthacrylate de 2-hydroxyéthyle, l'acrylate de gamma-oméga-perfluoroalkyle en C10-16 et le méthacrylate de stéaryle
3.	Méthacrylate d'isobutyle, polymérisé avec l'acrylate de butyle, l'anhydride maléique, esters de gamma-oméga-perfluoroalkyle en C8-14, amorcé avec du benzènecarboperoxoate de tert-butyle

Article	Substance toxique
4.	Alcool allylique, produits de réaction avec du pentafluoroiodoéthane et de tétrafluoroéthylène télomérisés, déshydroiodés, produits de réaction avec de l’épichlorhydrine et la triéthylènetétramine

SCHEDULE 2
(Sections 1 to 3, 6, 7, 9 and 12 and Schedule 3)

PERMITTED USES, CONCENTRATION LIMITS AND REPORTING THRESHOLDS

PART 1

PERMITTED USES

Item	Column 1 Toxic Substance	Column 2 Permitted Uses
1.	Benzidine and benzidine dihydrochloride that have the molecular formulas $C_{12}H_{12}N_2$ and $C_{12}H_{12}N_2 \cdot 2HCl$, respectively	(*a*) Staining for microscopic examination, such as immunoperoxidase staining, histochemical staining or cytochemical staining; (*b*) Reagent for detecting blood in biological fluids; (*c*) Niacin test to detect some micro-organisms; and (*d*) Reagent for detecting chloralhydrate in biological fluids.
2.	2-Methoxyethanol, which has the molecular formula $C_3H_8O_2$	(*a*) Adhesives and coatings for aircraft refinishing; and (*b*) Semiconductor manufacturing process.
3.	Pentachlorobenzene, which has the molecular formula C_6HCl_5	Use with chlorobiphenyls contained in equipment or liquids in the service of such equipment in which their use is permitted under the *PCB Regulations*
4.	Tetrachlorobenzenes, which have the molecular formula $C_6H_2Cl_4$	Use with chlorobiphenyls contained in equipment or liquids in the service of such equipment in which their use is permitted under the *PCB Regulations*
5.	Benzenamine, *N*-phenyl-, reaction products with styrene and 2,4,4-trimethylpentene	Additive in rubber, except in tires

PART 2

TEMPORARY PERMITTED USES

Item	Column 1 Toxic Substance	Column 2 Permitted Uses	Column 3 Expiry date
1.	Benzenamine, *N*-phenyl-, reaction products with styrene and 2,4,4-trimethylpentene	Additive in lubricants	2 years after the coming into force of these Regulations

ANNEXE 2
(articles 1 à 3, 6, 7, 9 et 12 et annexe 3)

UTILISATIONS PERMISES, CONCENTRATIONS MAXIMALES ET SEUILS POUR LES RAPPORTS

PARTIE 1

UTILISATIONS PERMISES

Article	Colonne 1 Substance toxique	Colonne 2 Utilisations permises
1.	La benzidine et le dichlorhydrate de benzidine, dont les formules moléculaires sont respectivement $C_{12}H_{12}N_2$ et $C_{12}H_{12}N_2 \cdot 2HCl$	*a*) Coloration pour l'examen microscopique, telle que la coloration immunoperoxydase, la coloration histochimique et la coloration cytochimique *b*) réactif pour détecter le sang dans les liquides biologiques *c*) test à la niacine pour détecter certains micro-organismes *d*) réactif pour détecter l'hydrate de chloral dans les liquides biologiques
2.	2-Méthoxyéthanol, dont la formule moléculaire est $C_3H_8O_2$	*a*) Adhésif et revêtement pour la finition d'aéronefs *b*) procédé de fabrication de semi-conducteurs
3.	Pentachlorobenzène, dont la formule moléculaire est C_6HCl_5	Utilisation avec un biphényle chloré contenu dans des pièces d'équipement ou un liquide servant à l'entretien de celles-ci, dont l'utilisation est permise aux termes du *Règlement sur les BPC*
4.	Tétrachlorobenzènes, dont la formule moléculaire est $C_6H_2Cl_4$	Utilisation avec un biphényle chloré contenu dans des pièces d'équipement ou un liquide servant à l'entretien de celles-ci, dont l'utilisation est permise aux termes du *Règlement sur les BPC*
5.	*N*-Phénylaniline, produits de réaction avec le styrène et le 2,4, 4-triméthylpentène	Additif dans le caoutchouc, à l'exception des pneus

PARTIE 2

UTILISATIONS PERMISES TEMPORAIREMENT

Article	Colonne 1 Substance toxique	Colonne 2 Utilisations permises	Colonne 3 Date d'expiration
1.	*N*-Phénylaniline, produits de réaction avec le styrène et le 2,4, 4-triméthylpentène	Additif dans les lubrifiants	Deux ans après l'entrée en vigueur du présent règlement

PART 3

PERMITTED CONCENTRATION LIMITS

Item	Column 1 Toxic Substance	Column 2 Product Containing the Toxic Substance	Column 3 Concentration Limit of the Toxic Substance
1.	2-Methoxyethanol, which has the molecular formula $C_3H_8O_2$	Diethylene glycol methyl ether, which has the molecular formula $C_5H_{12}O_3$	0.5 % (w/w)
2.	Tributyltins, which contain the grouping $(C_4H_9)_3Sn$	Tetrabutyltin, which has the molecular formula $(C_4H_9)_4Sn$	30 % (w/w)

PARTIE 3

CONCENTRATIONS MAXIMALES PERMISES

Article	Colonne 1 Substance toxique	Colonne 2 Produit contenant la substance toxique	Colonne 3 Concentration maximale de la substance toxique
1.	2-Méthoxyéthanol, dont la formule moléculaire est $C_3H_8O_2$	Éther méthylique de diéthylèneglycol, dont la formule moléculaire est $C_5H_{12}O_3$	0,5 % (p/p)
2.	Tributylétains qui contiennent le groupement $(C_4H_9)_3Sn$	Tétrabutylétain, dont la formule moléculaire est $(C_4H_9)_4Sn$	30 % (p/p)

PART 4

REPORTING THRESHOLDS

Item	Column 1 Toxic Substance	Column 2 Annual Quantity	Column 3 Annual Weighted Average Concentration	Column 4 Annual Quantity and Annual Weighted Average Concentration
1.	Benzidine and benzidine dihydrochloride that have the molecular formulas $C_{12}H_{12}N_2$ and $C_{12}H_{12}N_2 \cdot 2HCl$, respectively	1 kg		
2.	Chlorinated alkanes that have the molecular formula $C_nH_xCl_{(2n+2-x)}$ in which $10 \leq n \leq 13$			1 kg and 0.5% (w/w)

PARTIE 4

SEUILS POUR LES RAPPORTS

Article	Colonne 1 Substance toxique	Colonne 2 Quantité annuelle	Colonne 3 Concentration moyenne pondérée annuelle	Colonne 4 Quantité et concentration moyenne pondérée annuelles
1.	La benzidine et le dichlorhydrate de benzidine, dont les formules moléculaires sont respectivement $C_{12}H_{12}N_2$ et $C_{12}H_{12}N_2 \cdot 2HCl$	1 kg		
2.	Alcanes chlorés dont la formule moléculaire est $C_nH_xCl_{(2n+2-x)}$, où $10 \leq n \leq 13$			1 kg et 0,5 % (p/p)

SCHEDULE 3
(Subsections 3(2) to (4))

INFORMATION RELATED TO THE USE OF CERTAIN TOXIC SUBSTANCES IN A LABORATORY FOR ANALYSIS, IN SCIENTIFIC RESEARCH OR AS A LABORATORY ANALYTICAL STANDARD

1. Information respecting the laboratory where a toxic substance or a product containing it is used or is to be used:

(*a*) the name, civic and postal addresses, telephone number and, if any, email address and fax number of the laboratory; and

(*b*) the name, title, civic and postal addresses, telephone number and, if any, email address and fax number of any person authorized to act on the laboratory's behalf.

2. Information respecting each toxic substance set out in Schedule 1 or 2, and each product containing it that is used or is to be used:

(*a*) the name of the toxic substance and the name of the product, if applicable;

(*b*) the anticipated period of its use;

(*c*) the estimated quantity of the toxic substance to be used in a calendar year and its unit of measurement;

(*d*) the identification of each proposed use and each actual use, as the case may be; and

(*e*) in the case of a product,

(i) the estimated quantity of the product to be used in a calendar year and its unit of measurement, and

(ii) the estimated concentration of the toxic substance in that product and its unit of measurement.

ANNEXE 3
(paragraphes 3(2) à (4))

RENSEIGNEMENTS RELATIFS À L'UTILISATION DE CERTAINES SUBSTANCES TOXIQUES POUR DES ANALYSES EN LABORATOIRE, POUR LA RECHERCHE SCIENTIFIQUE OU EN TANT QU'ÉTALON ANALYTIQUE DE LABORATOIRE

1. Renseignements sur le laboratoire où la substance toxique ou le produit qui en contient est utilisé ou sera utilisé :

a) les nom, adresses municipale et postale, numéro de téléphone et, le cas échéant, numéro de télécopieur et adresse électronique du laboratoire;

b) les nom, titre, adresses municipale et postale, numéro de téléphone et, le cas échéant, numéro de télécopieur et adresse électronique de la personne autorisée à agir au nom du laboratoire, s'il y a lieu.

2. Renseignements sur chacune des substances toxiques mentionnées aux annexes 1 et 2 et sur chaque produit en contenant qui est ou sera utilisé :

a) le nom de la substance toxique et, le cas échéant, le nom du produit;

b) la période d'utilisation prévue;

c) la quantité de la substance toxique que l'on prévoit utiliser au cours d'une année civile ainsi que l'unité de mesure;

d) une description de chaque utilisation réelle ou projetée, selon le cas;

e) dans le cas d'un produit :

(i) la quantité du produit que l'on prévoit utiliser au cours d'une année civile ainsi que l'unité de mesure,

(ii) la concentration prévue de la substance toxique dans ce produit ainsi que l'unité de mesure de cette concentration.

SCHEDULE 4
(Subsection 9(4) and 10(3))

INFORMATION REQUIRED IN AN APPLICATION FOR A PERMIT OR AN APPLICATION FOR RENEWAL OF A PERMIT

1. Information respecting the applicant:

(*a*) their name, civic and postal addresses, telephone number and, if any, email address and fax number; and

(*b*) the name, title, civic and postal addresses, telephone number and, if any, email address and fax number of any person authorized to act on the applicant's behalf.

2. In the case of a toxic substance referred to in either section 4 or 6 of these Regulations or a product containing it, the following information:

(*a*) the name of the toxic substance and the name of the product, if applicable;

(*b*) the quantity of the toxic substance manufactured or imported during the 12 months before the day on which the application is submitted, and its unit of measurement;

(*c*) the estimated quantity of the toxic substance to be manufactured or imported during the period to which the permit will apply, and its unit of measurement;

(*d*) in the case of a product,

(i) the quantity of the product manufactured or imported during the 12 months before the day on which the application is submitted, and its unit of measurement,

(ii) the estimated quantity of the product to be manufactured or imported during the period to which the permit will apply, and its unit of measurement, and

(iii) the estimated concentration of the toxic substance in that product and its unit of measurement;

(*e*) the identification of each proposed use, if known; and

(*f*) if the applicant is a manufacturer or importer, the name, civic and postal addresses, telephone number and, if any, email address and fax number of each person in Canada to whom the applicant intends to sell a toxic substance or a product containing it and the name of each toxic substance or product.

3. Information that demonstrates that there is no technically or economically feasible alternative or substitute available to the applicant at the time of the application, other than a substance regulated under these Regulations, for the toxic substance.

4. Information that explains what measures have been taken to minimize or eliminate any harmful effect of the toxic substance on the environment and human health.

5. A description of the plan prepared respecting the toxic substance identifying the measures that will be taken by the applicant to comply with these Regulations and the period within which the plan

ANNEXE 4
(paragraphes 9(4) et 10(3))

RENSEIGNEMENTS À FOURNIR DANS LA DEMANDE DE PERMIS OU DE RENOUVELLEMENT DE PERMIS

1. Renseignements concernant le demandeur :

a) ses nom, adresses municipale et postale, numéro de téléphone et, le cas échéant, numéro de télécopieur et adresse électronique;

b) les nom, titre, adresses municipale et postale, numéro de téléphone et, le cas échéant, numéro de télécopieur et adresse électronique de la personne autorisée à agir au nom du demandeur, s'il y a lieu.

2. S'agissant d'une substance toxique visée aux articles 4 ou 6 du présent règlement, ou d'un produit qui en contient, les renseignements suivants :

a) le nom de la substance toxique et, le cas échéant, le nom du produit;

b) la quantité de substance toxique que le demandeur a fabriqué ou importé au cours des douze mois précédant la date de présentation de la demande, ainsi que l'unité de mesure;

c) la quantité de substance toxique que le demandeur prévoit fabriquer ou importer au cours de la période visée par le permis, ainsi que l'unité de mesure;

d) dans le cas d'un produit :

(i) la quantité du produit que le demandeur a fabriqué ou importé au cours des douze mois précédant la date de la présentation de la demande, ainsi que l'unité de mesure,

(ii) la quantité du produit que le demandeur prévoit fabriquer ou importer au cours de la période visée par le permis, ainsi que l'unité de mesure,

(iii) la concentration prévue de la substance toxique dans ce produit, ainsi que l'unité de mesure;

e) la mention de chaque utilisation projetée, si le demandeur dispose de cette information;

f) si le demandeur est un fabricant ou un importateur, les noms, adresses municipale et postale, numéro de téléphone et, le cas échéant, numéro de télécopieur et adresse électronique de chaque personne au Canada à qui il projette de vendre la substance toxique ou le produit qui en contient, ainsi que le nom de la substance ou du produit en cause.

3. Les renseignements qui établissent qu'au moment de la demande de permis le demandeur n'est pas en mesure, sur le plan technique ou économique, de remplacer la substance toxique par une substance non visée par le présent règlement ou d'utiliser une solution de rechange.

4. Une explication des mesures qui ont été prises pour éliminer ou atténuer les effets nocifs de la substance toxique sur l'environnement et la santé humaine.

5. Le détail du plan élaboré à l'égard de la substance toxique comportant les mesures que le demandeur prendra pour se conformer au présent règlement ainsi que le délai prévu pour son exécution, lequel

is to be implemented, which must not exceed three years after the day on which the permit is first issued.

ne peut excéder trois ans à compter de la date initiale de délivrance du permis.

SCHEDULE 5
(Section 12)

INFORMATION RELATING TO THE MANUFACTURE OR IMPORT OF A TOXIC SUBSTANCE OR THE IMPORT OF A PRODUCT CONTAINING IT

1. Information respecting the manufacturer or importer:

(*a*) their name, civic and postal addresses, telephone number of their principal place of business and, if any, email address and fax number; and

(*b*) the name, title, civic and postal addresses, telephone number and, if any, email address and fax number of any person authorized to act on behalf of the manufacturer or importer.

2. Information respecting each toxic substance referred to in column 1 of Part 4 of Schedule 2 that is imported or manufactured and each product containing it that is imported or manufactured during a calendar year:

(*a*) the name of the toxic substance and the name of the product, if applicable;

(*b*) the calendar year;

(*c*) the total quantity of the toxic substance manufactured, and its unit of measurement;

(*d*) the total quantity of the toxic substance sold in Canada, and its unit of measurement;

(*e*) the total quantity of the toxic substance imported, and its unit of measurement;

(*f*) the identification of each proposed use of the toxic substance and the product, if applicable;

(*g*) the annual weighted average concentration of the toxic substance in the product and its unit of measurement, if applicable;

(*h*) the analytical method used to determine the concentration of the toxic substance in the product, if applicable;

(*i*) the analytical method detection limit used to determine the concentration of the toxic substance in the product, if applicable; and

(*j*) the name, civic and postal addresses, telephone number and, if any, email address and fax number of each person in Canada to whom the manufacturer or importer sold the toxic substance or the product.

3. The name, civic and postal addresses, telephone number and, if any, email address and fax number of the laboratory that determined the concentration of the toxic substance in the product, if applicable.

ANNEXE 5
(article 12)

RENSEIGNEMENTS SUR LA FABRICATION ET L'IMPORTATION DE SUBSTANCES TOXIQUES ET SUR L'IMPORTATION DE PRODUITS QUI EN CONTIENNENT

1. Renseignements concernant le fabricant ou l'importateur :

a) leur nom, les adresses municipale et postale et le numéro de téléphone de leur établissement principal et, le cas échéant, leur numéro de télécopieur et leur adresse électronique;

b) les nom, titre, adresses municipale et postale, numéro de téléphone et, le cas échéant, numéro de télécopieur et adresse électronique de la personne autorisée à agir au nom du fabricant ou de l'importateur, s'il y a lieu.

2. Renseignements sur chacune des substances toxiques mentionnées à la colonne 1 de la partie 4 de l'annexe 2 qui est fabriquée ou importée au cours de l'année civile et sur chaque produit en contenant qui est fabriqué ou importé au cours de l'année civile :

a) le nom de la substance toxique et, le cas échéant, le nom du produit;

b) l'année civile visée;

c) la quantité totale de la substance toxique fabriquée, ainsi que l'unité de mesure;

d) la quantité totale de la substance toxique vendue au Canada, ainsi que l'unité de mesure;

e) la quantité totale de la substance toxique importée, ainsi que l'unité de mesure;

f) la mention de l'utilisation projetée de la substance toxique et, le cas échéant, du produit;

g) la concentration moyenne pondérée annuelle de la substance toxique dans le produit, ainsi que l'unité de mesure, le cas échéant;

h) la méthode analytique utilisée pour déterminer la concentration de la substance toxique dans le produit, le cas échéant;

i) la limite de détection de la méthode analytique utilisée pour déterminer la concentration de la substance toxique dans le produit, le cas échéant;

j) les nom, adresses municipale et postale, numéro de téléphone et, le cas échéant, numéro de télécopieur et adresse électronique de chaque personne au Canada à qui le fabricant ou l'importateur a vendu la substance toxique ou le produit.

3. Les nom, adresses municipale et postale, numéro de téléphone et, le cas échéant, numéro de télécopieur et adresse électronique du laboratoire qui a déterminé la concentration de la substance toxique dans le produit, le cas échéant.

Government of Canada. 1993. Priority Substances List assessment report. Chlorinated paraffins. Minister of Supply and Services, Ottawa, Ontario.

Canadian Environmental Protection Act

Priority Substances List
Assessment Report

Chlorinated Paraffins

Government of Canada / Gouvernement du Canada
Environment Canada / Environnement Canada
Health Canada / Santé Canada

Canada

CANADA'S GREEN PLAN

Canadian Environmental Protection Act

PRIORITY SUBSTANCES LIST
ASSESSMENT REPORT

CHLORINATED PARAFFINS

Government of Canada
Environment Canada
Health and Welfare Canada

Also available in French under the title:
Loi canadienne sur la protection de l'environnement
Liste des substances d'intérêt prioritaire
Rapport d'évaluation:
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Synopsis

The term "chlorinated paraffin waxes" is generally restricted to chlorinated paraffins having long carbon chains (i.e., $\geq C_{18}$). However, the scope of this assessment was broadened to include the short chain (i.e., $\leq C_{13}$) and medium chain (i.e., $C_{14\text{-}17}$) chlorinated paraffins which are also of concern because of their potential effects on the environment and human health.

Chlorinated paraffins (CPs) are produced in, and imported into, Canada for use as plasticizers and flame retardants as well as extreme-pressure additives in lubricating oils. They are persistent compounds and have the potential to bioaccumulate in aquatic organisms. No data were identified on the concentrations of these substances in any medium in the Canadian environment. However, data from other countries (including the United States) where these compounds are produced and used confirm their presence in the environment, particularly near production facilities.

Short chain chlorinated paraffins cause adverse effects in fish and aquatic invertebrates at concentrations below 1 µg/L in laboratory studies. However, owing to the lack of information on concentrations of short chain chlorinated paraffins in the Canadian environment, it is not possible to estimate exposure of Canadian biota or to compare this exposure with levels estimated to cause adverse effects.

Short chain chlorinated paraffins have caused cancer in experimental animals, although relevant data for humans are not available. Therefore, short chain chlorinated paraffins are considered to be "non-threshold toxicants", i.e., substances for which there is believed to be some chance of adverse effects at any level of exposure. For such substances, where data permit, estimated exposure is compared to quantitative estimates of cancer potency in order to characterize risk and provide guidance for further action, such as analysis of options to reduce exposure, under the *Canadian Environmental Protection Act* (CEPA). However, owing to the lack of information on concentrations of short chain chlorinated paraffins in environmental media to which humans are exposed, it is not possible to quantitatively estimate the total average daily intake of these compounds by the general population in Canada, or to subsequently compare these values to quantitative estimates of cancer potency.

There is also a lack of information on concentrations of **medium and long chain chlorinated paraffins** in environmental media to which humans and other biota are exposed. Therefore, it is not possible to estimate exposure of Canadian biota or to compare this exposure with levels estimated to cause adverse effects. Similarly, it is not possible to quantitatively estimate the total average daily intake of these compounds by the general population in Canada. The Tolerable Daily Intakes (i.e., the intake to which it is believed that a person can be exposed over a lifetime without deleterious effect) are derived on the basis of data from bioassays in animal species for these two groups of chlorinated paraffins and therefore cannot be compared with the estimated total daily intake in the general environment in Canada.

None of the chlorinated paraffins volatilizes readily to the atmosphere. Due to their predicted short tropospheric residence time (a few days), these compounds are not expected to contribute significantly to depletion of stratospheric ozone or global warming.

Based on these considerations, the Minister of the Environment and the Minister of National Health and Welfare have concluded that short chain chlorinated paraffins are considered to be "toxic" as defined under Paragraph 11(c) of the *Canadian Environmental Protection Act.* Available data are considered inadequate to evaluate whether medium and long chain chlorinated paraffins are considered to be "toxic" as defined under Paragraphs 11(a) or (c) of the *Canadian Environmental Protection Act.*

1.0 Introduction

The *Canadian Environmental Protection Act* (CEPA) requires the Minister of the Environment and the Minister of National Health and Welfare to prepare and publish a Priority Substances List that identifies substances, including chemicals, groups of chemicals, effluents, and wastes that may be harmful to the environment or constitute a danger to human health. The Act also requires both Ministers to assess those substances to determine whether they are "toxic" as defined under Section 11 of the Act which states:

> "...a substance is toxic if it is entering or may enter the environment in a quantity or concentration, or under conditions:
>
> (a) having or that may have an immediate or long-term harmful effect on the environment;
>
> (b) constituting or that may constitute a danger to the environment on which human life depends; or
>
> (c) constituting or that may constitute a danger in Canada to human life or health."

Substances that are assessed as "toxic" as defined under Section 11 may be placed on Schedule I of the Act. Consideration can then be given to developing regulations, guidelines, or codes of practice to control any aspect of these substances' life cycle, from the research and development stage through manufacture, use, storage, transport, and ultimate disposal.

The substance "chlorinated paraffin waxes" was included in Group 3 of the Priority Substances List. This term is generally restricted to chlorinated paraffins having long carbon chains. However, the scope of the assessment was broadened to include the short chain and medium chain chlorinated paraffins since they are also of concern due to their potential effects on the environment and human health. In this report, chlorinated paraffins having carbon chain lengths of 13 or less ($\leq C_{13}$) are termed "short", those having 14 to 17 carbon atoms ($C_{14\text{-}17}$) are considered to be "medium", and those having 18 or more ($\geq C_{18}$) are considered to be "long". To the extent possible, in each section of this report, these compounds are addressed in this order.

The assessment of whether chlorinated paraffins are "toxic", as defined under CEPA, was based on the determination of whether they **enter** or are likely to enter the Canadian environment in a concentration or quantities or under conditions that could lead to **exposure** of humans or other biota at levels that could cause adverse **effects.**

To identify data relevant to the assessment of effects on human health, literature searches of the following computerized databases were conducted: Medline (1966 to 1989), Hazardous Substances Data Bank (HSDB), Registry of Toxic Effects of Chemical Substances (RTECS), Integrated Risk Information System (IRIS), Chemical Carcinogenesis Research Information System (CCRIS) (all to January, 1992), Toxline

(1965 to 1992), Toxlit (1981 to 1992), and EMBASE (1985 to 1992). Data included in an unpublished background document prepared under contract (Mitchell, 1991) were also considered in the preparation of this report.

To identify data relevant to the estimation of exposure of the general population to chlorinated paraffins, literature searches were conducted in the following computerized databases: Environment Canada Departmental Library Catalogue (ELIAS) (1992), AQUAREF (1970 to 1992), Canadian Research Index (MICROLOG) (1979 to 1992), and Co-operative Documents Project (CODOC) (1992). Dr. G. Jenkins of the Ontario Ministry of the Environment, Mr. D. Spink of the Alberta Ministry of Environment, and Mr. H. St.-Martin of the Quebec Ministry of the Environment were also consulted in an attempt to identify relevant information on concentrations of chlorinated paraffins in environmental media to which humans are exposed, i.e., drinking water.

With respect to the approach adopted for identifying the data relevant to assessment of effects on the environment, literature searches of the following computerized databases were conducted: Chemical Abstracts (1967 to 1992), BIOS IS Previews (1969 to 1992), National Technical Information Service (NTIS) (1980 to 1992), and Pollution and Toxicology Database (POLTOX) (1982 to 1992). Other sources of information were identified through FATERATE (1989) and the Chemical Evaluation Search and Retrieval System (CESARS) (1988).

Information on both the environmental and health aspects was also sought from the following agencies:

- Umweltbundesamt, Berlin, Federal Republic of Germany;
- Norwegian State Pollution Control Authority, Oslo, Norway;
- Office fédéral de l’énvironnement, des fôrets et du paysage, Berne, Switzerland;
- National Chemicals Inspectorate, Solna, Sweden;
- National Environmental Protection Board, Solna, Sweden;
- National Board of Waters and Environment, Helsinki, Finland;
- British Industrial Biological Research Association, Surrey, England;
- World Health Organization, Geneva, Switzerland;
- Environmental Protection Agency, Copenhagen, Denmark;
- Environmental Agency, Japan; and
- International Agency for Research on Cancer, Lyon, France.

Every effort was also made to obtain all the detailed reports of an extensive series of studies conducted by the Working Party of the Chlorinated Paraffin Manufacturers Toxicology Testing Consortium which are briefly described in Serrone *et al.* (1987). Assistance in this regard was requested from Dr. D.M. Serrone of Ricerca Inc., Painesville, Ohio, Mr. R.J. Fensterheim of the Chlorinated Paraffins Industry Association, Dr. M.T. Richardson of Imperial Chemical Industries (ICI), U.K., and Mr. R. Zampini of ICI Canada, who were unable to provide the requested reports. However, full reports of the studies in this series, which were considered critical to this assessment, were obtained from the United States Environmental Protection Agency (U.S. EPA).

Data relevant to the assessment of whether chlorinated paraffins are "toxic" to human health obtained after completion of the peer review of human health-related sections of the report in August 1992 were not considered for inclusion. Similarly, data relevant to assessment of whether chlorinated paraffins are "toxic" to the environment obtained after completion of peer review of those sections of the report in June 1992 were not considered.

The results of recent investigations and all original studies relevant to the assessment of whether chlorinated paraffins are "toxic" as defined under Section 11 of CEPA have been critically evaluated by the following Health and Welfare Canada staff (exposure of the general population and effects on human health), Environment Canada staff (entry, environmental exposure and effects), and Fisheries and Oceans staff (environmental exposure and effects):

Environment Canada	Health and Welfare Canada	Fisheries and Oceans
L. Brownlee	P.K.L. Chan	V. Zitko
K.M. Lloyd	M.E. Meek	
	D. Riedel	

Following circulation and external peer review of the health-related sections by staff of British Industrial Biological Research Association (BIBRA) Toxicology International, U.K. and Dr. D.M. Serrone of Ricerca Inc., Painesville, Ohio (Supporting Document only), they were reviewed and approved by the Guidelines and Standards Rulings Committee of the Bureau of Chemical Hazards of Health and Welfare Canada. As part of the review and approval process established by Environment Canada, the environmental sections of the Assessment Report and Supporting Document were reviewed by Drs. J.A. Cotruvo, P. Miller, M. Zeeman, and W.S. Rabert of the U.S. EPA and Dr. D.C.G. Muir of Fisheries and Oceans. In addition, Mr. R. Zampini of ICI Canada and Dr. M.T. Richardson of ICI U.K. provided comments on Subsections 2.2 and 2.3 and Dr. N. Bunce of the University of Guelph provided comments on Subsection 2.3. The final Assessment Report was reviewed and approved by the Environment Canada/Health and Welfare Canada CEPA Management Committee.

In this report, a Synopsis is presented which will appear in the Canada Gazette. An extended summary of technical information that is critical to the assessment is presented in Section 2.0. This information is presented in greater detail in a Supporting

Document which is available upon request. The assessment of whether chlorinated paraffin waxes are "toxic" under CEPA is presented in Section 3.0.

Copies of this Assessment Report and the unpublished Supporting Document are available upon request from:

Commercial Chemicals Branch
Environment Canada
14th Floor, Place Vincent Massey
351 St. Joseph Boulevard
Hull, Quebec
K1A 0H3

Environmental Health Centre
Health and Welfare Canada
Room 104
Tunney's Pasture
Ottawa, Ontario
K1A 0L2

2.0 Summary of Information Critical to Assessment of "Toxic"

2.1 Identity, Properties, Production, and Use

Chlorinated paraffins (CPs) are chlorinated derivatives of n-alkanes, having carbon chain lengths ranging from 10 to 38, and a chlorine content ranging from about 30 to 70% (by weight). Commercial products, of which there are over 2000, (Serrone *et al.*, 1987) are complex mixtures of homologues and isomers. The products vary in the distribution, possibly type, and range of chain lengths, and in the degree of chlorination.

The melting point of CPs increases with increasing carbon chain length and with increasing chlorine content. Consequently, at room temperature, CPs range from colourless to yellowish liquids at about 40% chlorine, to white solids (softening point at about 90^0C) at 70% chlorine. Chlorinated paraffins have very low vapour pressures (e.g., 1.3×10^{-4} Pa for $C_{14\text{-}17}$, 52% C1 at 20^0C) and solubilities in water, the latter ranging from 95 to 470 µg/L for some of the short chain mixtures ($C_{10\text{-}13}$) to as low as 3.6 to 6.6 µg/L for some of the long chain mixtures ($C_{20\text{-}30}$) (Campbell and McConnell, 1980). Log octanol:water partition coefficients (i.e., log K_{ow}) values (as measured by high performance thin layer chromatography) are very high, ranging from about 5 to 12 (Renberg *et al.*, 1980).

Chlorinated paraffins are obtained by direct chlorination of n-alkanes of high purity in the liquid phase, in the presence of hydrogen chloride. They are manufactured commercially by letting gaseous chlorine flow or bubble into straight chain $C_{9\text{-}30}$ petroleum fractions, such as normal paraffins, at least 98% linear, and wax fractions averaging as many as 24 carbon atoms. The process is catalyzed by ultraviolet light (Mukherjee, 1990; ICI, 1992a).

The high molecular weight, large number of isomers and congeners, low volatility, non-polar character, and loss of hydrochloric acid or chlorine at elevated temperatures make it very difficult to measure low concentrations of chlorinated paraffins. The current method of choice is gas chromatography with negative ions chemical ionization mass spectrometry. This method is described in Muller and Schmid (1984), Schmid and Muller (1985), and Jansson *et al.* (1991).

Imperial Chemical Industries Canada is the only producer of CPs in Canada, operating a plant with a production capacity of 5-kt/year in Cornwall, Ontario. This plant, however, has been operating well below capacity for several years, producing approximately 2.9 kt in 1990 (Camford Information Services, 1991). Specific information was not found on the amounts of each chain length (i.e., short, medium, and long) produced. Chlorinated paraffins produced in Canada are sold under the trade name Cereclor.

Estimated total imports from the United States, United Kingdom, and Germany were 2.3 kt for 1990 (Camford Information Services, 1991), although it is expected that total imports for 1992 will be between 1.0 and 1.5 kt (ICI, 1992a). Total exports from Canada are considerably lower at about 200 t/yr (Camford Information Services, 1991).

Again, specific information was not found on the amount of each chain length imported and exported.

In Canada, CPs are used mainly in plastics as a plasticizer and flame retardant (65% of use). The other major market (20%) for CPs is as an extreme-pressure additive in metal-working fluids to lower the heat and allow faster metal working. Smaller applications for chlorinated paraffins include flame-retardant additives in heavy-duty rubber (8%), paints (3%), and adhesives and sealants (2%) (ICI, 1992b). Total Canadian demand is about 3.5 to 4 kt/yr (ICI, 1992a). The short chain CPs ($\leq C_{13}$) are used primarily as lubricants, flame retardants, and sealants; the medium chain CPs ($C_{14\text{-}17}$) as plasticizers; and the long chain CPs ($\geq C_{18}$) are used in paints and as lubricants and flame retardants.

2.2 Entry into the Environment

Chlorinated paraffins are not known to occur naturally. There are no recorded releases of CPs into the Canadian environment and estimates of releases have not been identified. Although releases of CPs could occur during their manufacture, use, transport, and disposal, the major sources of release into the environment are likely manufacturing and lubricant applications. These two sources are discussed in this subsection, based on data prepared by the Chlorinated Paraffins Industry Association for the U.S. EPA (CPIA, 1992).

Waterborne releases from manufacturing can occur from spills, facility wash-down, and stormwater runoff. As CPs are insoluble in water, and releases from these sources are routinely collected and treated in the facilities' wastewater treatment system, the CPIA considers these releases to be negligible (CPIA, 1992).

The formulation and use of metal-working fluids, composed of short-chain, 50 to 60% CPs, are potential sources of release of CPs into aquatic environments. The release from process metal-working fluids results from disposal of used drums, carry-off from work pieces, and disposal of spent baths. Release to the environment from drum recycling is considered negligible, although relevant data have not been identified. No data were identified for Canada, but the U.S. EPA estimates that fluid releases in the United States due to carry-off from work pieces may range from 2.5 kg/site per year for a small user (100-L capacity) to 2500 kg/site per year for a large user (95 000-L capacity). Similarly, for small and large users, CPIA (1992) estimates that releases to water in the United States from disposal of spent chlorinated paraffin baths vary from 12 to 1500 kg/site per year, respectively, with 90% of the shops discharging near the lower end of the range.

These estimates are considerably lower than those from Sweden, where it is estimated that about 50% of the used oils may be directly discharged (KEMI, 1991). Minimal release is expected because of paint formulating or when CPs are used as flame retardants (a major use in Canada). According to Swedish estimates, less than 0.001% of CPs is released during use as a flame retardant (KEMI, 1991).

2.3 Exposure-related Information

2.3.1 *Fate*

Few data are available on the environmental fate of CPs because of the complex nature of the mixtures and difficulties in measuring low levels. Based on general patterns of behaviour of hydrophobic organics in the environment, it is likely that CPs are fairly immobile, remain adsorbed onto soil or sediment particles, and are slowly degraded. In the natural environment, CPs are generally stable, but degradation is possible by micro-organisms (Madeley and Birtley, 1980). The ability of aerobic micro-organisms to oxidize a range of CPs depends on the previous acclimatization of the microbes, the chain length, and the degree of chlorination of the CPs. Short and medium chain CPs (i.e., C_{10-20}) are degraded most rapidly. The longer the carbon chain and the higher the chlorine content, the less chlorine that is released (Omori *et al.,* 1987).

Few data have been identified on the mobility and transport of CP residues from sites of manufacturing, use, or disposal. However, some of the calculated Henry's Law constants for CPs are similar to those for chlorinated aliphatic pesticides, such as toxaphene, chlordane, and aldrin (Sunito *et al.,* 1988), which are known to be transported in the atmosphere. Airborne dispersion of CPs has been found in the United Kingdom and Sweden where monitoring data indicate widespread levels of low contamination in water, sediments, aquatic and terrestrial biota, and even commercial foods (Campbell and McConnell, 1980; Jansson *et al.,* 1993).

Chlorinated paraffins are generally considered to be persistent. Hydrolysis, oxidation, and photolysis with visible or near ultraviolet radiation are insignificant routes of transformation at ambient temperatures. No experimental data are available on the fate of any CPs that volatilize to the atmosphere. However, it may be assumed that any volatilized CPs would be subject to attack by hydroxyl radicals in the troposphere. Using the method of Atkinson (1986) for estimating the rate constant for reaction of chlorinated paraffins with hydroxyl radicals, the likely tropospheric half-life is a few days under summer conditions.

While data indicate a potential for bioaccumulation, few bioconcentration factors (BCFs) or biomagnification factors (BAFs) have been experimentally determined. The uptake and accumulation of CPs in fish from water and food appear to be inversely proportional to molecular weight, i.e., CPs with short chain length and a low chlorine content are taken up most rapidly. Similarly, depuration is slowest for highly chlorinated forms. Measurement of BCFs and BAFs is difficult due to the low water solubility of these substances, and subsequent slow uptake rates requiring long exposure periods to achieve steady-state equilibrium. In several of the reviewed tests, it was unclear whether steady-state had been achieved. Reported bioconcentration factors vary dramatically between different CPs and between species, ranging from 0.007 to 139 000 (Sundstrom and Renberg, 1985). The highest bioconcentration factor, which was observed for mussels (Renberg *et al.,* 1986), was reported at a much lower concentration of chlorinated paraffins in water than that in most other studies. Observations for dioxins

and furans have been similar, with Cook *et al.* (1991) reporting much higher BCFs when aquatic species were exposed to concentrations of pg/L rather than ng/L.

Based on log Kows of >6, accumulation of CPs via the food chain (i.e., biomagnification) could be significant (Thomann, 1989). In studies on uptake of various short chain (C_{10-13}) chlorinated paraffins from food using rainbow trout *(Oncorhynchus mykiss)* and bleaks *(Alburnus alburnus)*, BAFs ranged from 2 to 41 on a wet weight (w.w.) basis (Lombardo *et al.*, 1975; Bengtsson and Ofstad, 1982), indicating that biomagnification could occur in the environment.

2.3.2 *Concentrations*

No information was identified on levels of CPs in any environmental medium in Canada. In a study conducted in Atlantic Canada to monitor organic and inorganic contaminants in edible shellfish, CPs were not detected (detection limit = 0.4 µg/g w.w.) in any of the 30 assayed samples from various locations (Environment Canada, 1989). Environmental fate modelling (e.g., Fugacity model; Mackay *et al.*, 1985) was considered unsuitable for predicting levels in the Canadian environment, as CPs are mixtures of paraffins of varying chain lengths and chlorination, each with very high log K_{ow} values, making model predictions unreliable. In addition, there is little information on transformation and release rates for specific CPs.

Data on environmental levels of CPs in other countries are also sparse. Murray *et al.* (1988) found that short, medium, and long chain CPs were generally present at quantifiable concentrations in sediment, suspended solids, and biota in a creek downstream from the discharge of a chlorinated paraffin manufacturing plant in Ohio. Campbell and McConnell (1980) reported detectable concentrations of C_{10-20} and C_{20-30} CPs in marine and fresh waters, and sediment, as well as in birds, seals, fish, and mussels, both close to and far from manufacturing sites in the United Kingdom. Jansson *et al.* (1993) reported residues of CPs in all samples of various species from several terrestrial, freshwater, and marine ecosystems in Sweden. These monitoring studies demonstrate the potential for presence and transport in the environment.

Only one study was identified in which levels of chlorinated paraffins were determined in a limited range of foodstuffs and human tissues (Campbell and McConnell, 1980). Data available in the published account of this early study were insufficient, however, to permit evaluation of the validity of these results.

2.4 Effects-related Information

2.4.1 *Experimental Animals and* In Vitro

The acute toxicity of all chlorinated paraffins is considered to be low with oral LD_{50}s for rats and mice being greater than 4 g/kg b.w. (Dover Chemical Corp., 1975; Birtley *et al.*, 1980; Bucher *et al.*, 1987). Signs of toxicity in rats, which were most evident following oral administration of the shorter chain CPs (doses greater than

4 g/kg b.w.) included piloerection, muscular incoordination, and urinary and fecal incontinence (Birtley *et al.,* 1980).

Short Chain Chlorinated Paraffins ($\leq C_{13}$) - In a well documented study by the National Toxicology Program, enlarged livers (mice), decreased body weights (rats), and diarrhea (both species) were reported in F344/N rats and $B6C3F_1$ mice following administration of a short chain CP (C_{12}, 60% C1) by gavage in corn oil for 16 days (NTP, 1986a; Bucher *et al.,* 1987). The lowest-observed-effect-levels (LOEL) based on the compound-related hepatomegaly were 469 mg/(kg b.w.·day) and 938 mg/(kg b.w.·day) for rats and mice, respectively. In 14-day studies in F344 rats conducted by the Working Party of the Chlorinated Paraffin Manufacturers Toxicology Testing Consortium, the no-observed-effect-level (NOEL) for a short chain CP ($C_{10\text{-}13}$, 58% C1) administered by gavage in corn oil was considered to be 30 mg/(kg b.w.· day), based on enlarged livers and hepatocellular hypertrophy at dose levels of 100 mg/(kg b.w.· day) or above (IRDC, 1981a; Serrone *et al.,* 1987).

A NOEL was not established in a 13-week study in which a short chain CP (C_{12}, 60% C1) was administered by gavage in corn oil to $B6C3F_1$ mice and F344/N rats [LOELs were 313 and 125 mg/(kg b.w.· day) for mice and rats, respectively, based on dose-related increases in relative liver weights] (NTP, 1986a; Bucher *et al.,* 1987). For short chain CPs ($C_{10\text{-}13}$, 58% C1), a NOEL of 10 mg/(kg b.w.· day) in F344 rats was reported following administration for 90 days by gavage in corn oil or in the diet, on the basis that no treatment-related microscopic changes were found in any tissues at this dose (Serrone *et al.,* 1987). In this study, there were increases in liver and kidney weights, increases in the incidence of hepatocellular hypertrophy, increases in thyroid-parathyroid weights, and hypertrophy and hyperplasia of the thyroid. There were high incidences of trace-to-mild chronic nephritis in the kidneys of male rats and increased pigmentation of the renal tubules in female rats.

In a study conducted by the National Toxicology Program on a short chain CP (C_{12}, 58% C1) in which F344/N rats and $B6C3F_1$ mice were administered the compound by gavage in corn oil for two years (NTP, 1986a; Bucher *et al.,* 1987), mean body weights of high-dose male rats [625 mg/(kg b.w. · day)] were 8 to 12% lower than that in controls after week 20. The body weights of exposed female mice were about 10% lower than those of the controls during the second year. Survival of both low-dose [312 mg/(kg b.w · day)] and high-dose [625 mg/(kg b.w. · day)] male rats and low-dose female rats was significantly less than controls after week 90. Survival of high-dose [250 mg/(kg b.w.·day)] female mice was significantly less than that of controls after week 100. The incidence of hepatocellular neoplasms (primarily neoplastic nodules) and adenomas or adenocarcinomas (combined) of the liver were significantly increased at both dose levels in both species and sexes. The incidence of adenomas or hyperplasia of the renal tubular cells increased significantly in exposed male rats. The incidence of follicular cell adenomas or carcinomas (combined) of the thyroid gland was increased in exposed female rats and female mice. In addition, alveolar/bronchiolar adenomas or carcinomas (combined) were induced in male mice, and mononuclear cell leukemia was significantly increased in exposed male rats and in low-dose female rats.

Non-neoplastic lesions induced by the short chain CP in exposed rats included necrosis, hypertrophy, and angiectasis of the liver; erosion, inflammation, and ulceration of the glandular stomach and forestomach; and the formation of multiple cysts in the kidneys of males. The incidence of nephropathy was also increased in exposed female rats and mice but was decreased in male mice as compared with controls [LOAEL = 312 mg/(kg b.w.· day) for rats and 125 mg/(kg b.w. · day) for mice)]. It was concluded by the NTP that "under the conditions of these 2-year gavage studies, there was clear evidence of carcinogenicity of chlorinated paraffins (C_{12}, 60% C1) for F344/N rats and B6C3F_1 mice. However, the maximum tolerated dose may have been exceeded in male and female rats" (NTP, 1986a).

Available data are limited on the genotoxicity of the short chain CPs. Although not mutagenic in bacterial assays *in vitro* with or without metabolic activation (Birtley *et al.*, 1980; NTP, 1986a), short chain CPs have been clastogenic in *in vitro* bioassays in the absence of metabolic activation (Myhr *et al.*, 1990) and have also induced cell transformation in the majority of available *in vitro* assays of this endpoint (ICI, 1982a). In two identified *in vivo* studies, the complete reports of which were not available for this assessment, short chain CPs did not induce dominant lethal mutations in rats or increase the frequency of chromosomal aberrations in bone marrow cells in rats (Serrone *et al.*, 1987).

In a series of developmental studies conducted for the Chlorinated Paraffins Manufacturers Toxicology Testing Consortium, the number and location of viable and nonviable fetuses, early and late resorptions, the number of total implantations and corpora lutea, and the incidence of fetal malformations were examined following administration of a short chain CP (C_{10-13}, 58% C1) by gavage in corn oil to pregnant Charles River rats on days 6 to 19 of gestation and pregnant Dutch Belted rabbits on days 6 to 27 of gestation. An increase in the incidence of adactyly and/or shortened digits in the offspring of rats exposed to a maternally toxic dose [2000 mg/(kg b.w.· day) by gavage in corn oil] (IRDC, 1982) and embryo- or feto-toxic effects at doses less than those that were toxic to the mothers were observed in rabbits exposed to 30 and 100 mg/kg b.w. (IRDC, 1983a).

Available data are extremely limited on the potential neurotoxicity of the short chain chlorinated paraffins. Following oral administration of a single dose (1 mg/kg b.w.) of a short chain CP (polychlorohexadecane) to 10-day-old male and female mice, there was no effect on muscarinic receptors, though it was suggested on the basis of an observed decrease in the V_{max} for sodium-dependent choline uptake, that there was a presynaptic effect on the cholinergic system (Eriksson and Nordberg, 1986). There was a dose-related trend to decreased motor capacity in adult NMRI male mice administered a single dose of 30 to 300 mg/kg b.w. of a short chain CP (C_{10-13}, 49% C1) intraperitoneally , which was statistically significant at the highest dose (Eriksson and Kihlstrom, 1985).

Data were not found on the immunotoxicity of the short chain chlorinated paraffins.

Medium Chain Chlorinated Paraffins (C_{14-17}) - In 14-day studies conducted by the Working Party of the Chlorinated Paraffin Manufacturers Toxicology Testing Consortium as summarized by Serrone *et al.* (1987) in which F344 rats were administered a medium chain CP (C_{14-17}, 52% C1) in the diet, the no-observed-effect-level (NOEL) was considered to be 500 ppm [30 mg/(kg b.w.· day)], based on increases in liver weight and diffuse hepatocellular hypertrophy.

For a medium chain CP (C_{14-17}, 52% C1), a NOEL of 10 mg/(kg b.w. · day) (more appropriately a NOAEL since an increase in liver weight was observed at this dose) in F344 rats was reported following administration by gavage in corn oil or in the diet for 90 days, on the basis that no treatment-related microscopic changes were found in any tissues at this dose (Serrone *et al.*, 1987). There were increases in liver and kidney weights, increases in the incidence of hepatocellular hypertrophy, increases in thyroid-parathyroid weights, and hypertrophy and hyperplasia of the thyroid. There were also high incidences of trace-to-mild chronic nephritis in the kidneys of male rats and increased pigmentation of the renal tubules in female rats.

In another 90-day study in which a medium chain CP (C_{14-17}, 52% C1) was administered in the diet, Birtley *et al.* (1980) reported dose-related proliferation of the smooth endoplasmic reticulum in the hepatic cells of rats at 500 ppm and above {NOEL = 250 ppm [12.5 mg/(kg b.w. · day)], LOEL = 500 ppm [25 mg/(kg b.w. · day)] }. In beagle dogs exposed to the same compound in the diet, exposure-related effects were confined principally to male dogs receiving 100 mg/(kg b.w. · day) (significant increases in serum alkaline phosphatase activity and liver-weight-to-body-weight ratios). Electron microscopy also revealed an increase in the smooth endoplasmic reticulum of hepatocytes in all exposed animals [(NOEL = 10 mg/(kg b.w. · day), LOEL = 30 mg/(kg b.w. · day)].

Available limited data on the genotoxicity of medium chain CPs indicate that these compounds are not mutagenic in bacterial assays *in vitro* with or without metabolic activation (Birtley *et al.*, 1980). They have been negative in *in vitro* assays of cell transformation (Birtley *et al.*, 1980) and in the only identified *in vivo* study (the complete report of which was not available for this assessment). Oral administration of a medium chain CP did not increase the frequency of chromosomal aberrations in bone marrow cells in rats (Serrone *et al.*, 1987).

Only one reproductive study has been identified in which rats were exposed to a medium chain CP (C_{14-17}, 52% C1) (IRDC, 1985; Serrone *et al.*, 1987). In this investigation, although there were no dose-related differences in appearance, fertility, body weight gain, food consumption, or reproductive performance in the parental generation, there were adverse effects on body weight and condition, and possibly haematological parameters in the pups at all doses (100 to 6250 ppm) [LOEL = 100 ppm or 5.7 mg/(kg b.w. · day) for the males and 7.2 mg/(kg b.w. · day) for the females]. Observations in pups included bruised areas, decreased activity, laboured breathing, pale discolouration, and/or blood around the orifices. Pup survival was also decreased at doses $\geq$ 1000 ppm in the diet. Observations at necropsy in pups that died during the study

included pale liver, kidneys, and lungs, and blood in the cranial cavity, brain, stomach, and intestines. The authors suggested that these effects were more likely attributable to lactational rather than *in utero* exposure and added that, based on preliminary results from a cross-fostering study, mortality in pups exposed via milk was greater than that in pups exposed only *in utero* (Serrone *et al.,* 1987).

In a series of developmental studies conducted for the Chlorinated Paraffins Manufacturers Toxicology Testing Consortium, the number and location of viable and nonviable fetuses, early and late resorptions, the number of total implantations and corpora lutea, and the incidence of fetal malformations were examined following administration of a medium chain CP (C_{14-17}, 52% C1) by gavage in corn oil to pregnant Charles River rats on days 6 to 19 of gestation and pregnant Dutch Belted rabbits on days 6 to 27 of gestation. Teratogenic effects were not observed and embryo- or feto-toxic effects were observed only at doses greater than those that were toxic to the mothers [lowest NOAEL in mothers was 30 mg/(kg b.w. · day) in rabbits and in offspring, 100 mg/(kg b.w. · day) in rabbits] (IRDC, 1983b; 1984).

Data were not identified on the neurotoxicity or immunotoxicity of the medium chain chlorinated paraffins.

Long Chain Chlorinated Paraffins ($\geq C_{18}$) - Following administration of a long chain CP (C_{23}, 40% C1) by gavage in corn oil for 16 days, no compound-related clinical signs or gross pathological effects were observed in F344 rats or $B6C3F_1$ mice. The no-observed-effect-levels (NOELs) were considered to be the highest doses [3750 mg/(kg b.w. · day) for the rats and 7500 mg/(kg b.w.· day) for the mice (NTP, 1986b; Bucher *et al*., 1987)]. In 14-day studies in F344 rats conducted by the Working Party of the Chlorinated Paraffin Manufacturers Toxicology Testing Consortium, the no-observed-effect-levels (NOELs) were considered to be 3000 mg/(kg b.w. · day) for a long chain CP (C_{20-30}, 43% C1) administered by gavage in corn oil and 15 000 ppm [1715 mg/(kg b.w. · day)] for another long chain CP (C_{22-26}, 70% C1) administered in the diet, respectively, based on a lack of observed compound-related effects on clinical signs or organ weights or in the tissues examined microscopically (IRDC, 1981b; 1981c; Serrone *et al.,* 1987).

Based on the results of a well documented, 13-week study, a NOEL for a long chain CP (C_{23}, 43% C1) administered to mice by gavage was reported to be 7500 mg/(kg b.w. · day), based on no effects noted at any dose (Bucher *et al.,* 1987; NTP, 1986b). In rats, the same CP caused a dose-related granulomatous inflammation of the liver in all exposed females [LOEL = 235 mg/(kg b.w. · day)]. Serrone *et al.* (1987) reported similar hepatic lesions in female rats following administration by gavage of another long chain CP (C_{20-30}, 43% C1). 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 b.w. · day). [The authors considered the NOEL to be 3750 mg/(kg b.w. · day) for males, though this is more appropriately a NOAEL, based on observed effects in the kidneys.] A NOEL could not be established for the females [LOEL = 100 mg/(kg b.w. · day)]. In similar studies in which a long chain

CP (C_{22-26}, 70% C1) was administered in the diet, hepatocellular hypertrophy and cytoplasmic fat vacuolation in the liver and increases in serum hepatic enzymes of both sexes were observed at 3750 mg/(kg b.w. · day) [NOEL was 900 mg/(kg b.w. · day)].

In the study conducted by the National Toxicology Program (NTP, 1986b; Bucher *et al.*, 1987), the carcinogenic response following exposure to the long chain CP (C_{23}, 43% C1), administered to rats and mice under identical conditions to those of the short chain CP, was not as clear as that for the short chain CP; however, there were some increases in tumor incidence in both species. Doses administered were 0, 1875, or 3750 mg/(kg b.w. · day) to male rats; 0, 100, 300, or 900 mg/(kg b.w. · day) to female rats; and 0, 2500, or 5000 mg/(kg b.w. · day) to male and female mice. There were no significant differences in survival and clinical signs of toxicity between exposed and control groups in both sexes and species. Mean body weights of rats were similar in exposed and control animals but both male and female mice in the low-dose group gained less weight than those in the control or high-dose groups. There was a statistically significant increase in the incidence of malignant lymphomas in male mice, a marginal (not statistically significant) increase of hepatocellular carcinomas in female mice, and adenomas or carcinomas (in both males and females). There was a positive trend for increased incidence of phaeochromocytomas of the adrenal medulla with increased dose in female rats.

The primary non-neoplastic lesion related to administration of this CP included a diffuse lymphohistiocytic inflammation in the liver and in the pancreatic and mesenteric lymph nodes of male and female rats. Splenic congestion was a secondary effect. These lesions occurred earlier in female rats and at lower doses than in male rats [LOAEL = 100 mg/(kg b.w. · day)]. No significant non-neoplastic lesions were attributed to exposure in mice; however, for female mice, 60 to 70% of the early deaths in each group were attributed to utero-ovarian infection and this may have decreased the sensitivity of the study to detect a carcinogenic effect. Under the conditions of these two-year gavage studies, the NTP concluded that there was no evidence of carcinogenicity for male F344/N rats, equivocal evidence of carcinogenicity for female F344/N rats and female $B6C3F_1$ mice, and clear evidence of carcinogenicity for male $B6C3F_1$ mice. Members of the NTP Peer Review Panel commented that, although the high viscosity of the vehicle may have prevented administration of maximum tolerated doses (as indicated by the lack of observed effects on survival or body weight gain), the linear increase in liver weight and increases in serum enzyme levels in concurrent six-month and one-year studies in rats indicated achievement of a biologically effective dose.

Available limited data on the genotoxicity of long chain CPs indicate that these compounds are not mutagenic in bacterial assays *in vitro* with or without metabolic activation (Birtley *et al.*, 1980; NTP, 1986b). They have been negative in an *in vitro* assay of cell transformation (ICI, 1982b) and, in the only identified *in vivo* study, the complete report of which was not available for this assessment, oral administration of the long chain CPs did not increase the frequency of chromosomal aberrations in bone marrow cells in rats (Serrone *et al.*, 1987).

In a series of developmental studies conducted for the Chlorinated Paraffins Manufacturers Toxicology Testing Consortium, the number and location of viable and nonviable fetuses, early and late resorptions, the number of total implantations and corpora lutea, and the incidence of fetal malformations were examined following administration of one long chain CP (C_{20-30}, 43% C1) by gavage in corn oil and another (C_{22-26}, 70% C1) in 1% carboxymethyl cellulose to pregnant Charles River rats on days 6 to 19 of gestation and pregnant Dutch Belted rabbits on days 6 to 27 of gestation. Teratogenic effects were not observed and embryo- or feto-toxic effects were observed only at doses greater than those that were toxic to the mothers [lowest LOEL in mothers = 100 mg/(kg b.w. · day) in rabbits exposed to the C_{22-26}, 70% C1 CP; lowest NOEL in offspring = 1000 mg/(kg b.w. · day) in rabbits exposed to the C_{22-26}, 70% C1 CP] (IRDC, 1983c; 1981d; 1983d; 1982).

Data have not been identified on the neurotoxicity and immunotoxicity of the long chain chlorinated paraffins.

2.4.2 *Humans*

Epidemiological studies of populations exposed to CPs are not available and data on effects in humans are restricted to poorly documented clinical studies of the potential to induce irritation or sensitization of the skin following dermal application (Dover Chemical Corp., 1975; Howard *et al.,* 1975; English *et al.,* 1986).

2.4.3 *Ecotoxicology*

No data were identified on the toxicity of any of the chlorinated paraffins to micro-organisms, amphibians, reptiles, plants, and terrestrial invertebrates. No field data were found for any terrestrial species and laboratory studies on the acute or chronic effects of chlorinated paraffins are sparse. The relevant studies are described here, with the exception of mammalian data; the effects on laboratory mammals are described in Subsection 2.4.1.

In 1983, the Chlorinated Paraffins Producers Testing Consortium (a consortium of international manufacturers) determined the aquatic toxicity of C_{10-13}, 58% C1; C_{14-19}, 52% C1; C_{20-30}, 42% C1; and C_{20-30}, 70% C1, to the common mussel *(Mytilus edulis)* and rainbow trout *(Oncorhynchus mykiss).* Further testing was conducted on the most toxic of the four substances, the short chain CP (C_{10-13}), in several species. The results of several of these studies are discussed here. The chlorinated paraffins were dissolved in acetone before dilution with water in most studies because of their low water solubility. Values for toxicity are based on measured, rather than nominal, concentrations.

Short Chain Chlorinated Paraffins ($\leq C_{13}$) - The species most acutely sensitive to the short chain CP (C_{10-13}, 58% C1) were daphnids *(Daphnia magna)* and mysid shrimp *(Mysidopsis bahia)* with 96-hour LC_{50} values of 18 µg/L and 14 µg/L, respectively, in flow-through tests (Thompson and Madeley, 1983a; 1983b). The value for daphnids is based on data presented in the 21-day, chronic study (as opposed to an acute study), where 70% mortality was seen at 25.5 µg/L after three days. In addition, in another

chronic, 14-day, static-renewal study using daphnids, 50% mortality was seen after five days at 10 µg/L.

The sensitivities of the two species of algae tested varied, with the marine diatom *Skeletonema costatum* being more sensitive, having a 96-hour EC_{50} of 42.3 µg/L for growth. For the freshwater green alga *Selenastrum capricornutum,* the lowest reported EC_{50} was 1310 µg/L after 10 days (Thompson and Madeley, 1983c; 1983d). Interpretation of the results in algae is complicated by the loss (50 to 80%) of residues from the water during the course of the studies due to sorption to algal cells. In addition, effects noted on the diatom were transient over a 10-day test period, and may have been caused by a decrease in nutrient levels.

Significant chronic adverse effects were noted in the range of 2.4 to 20 µg/L for the freshwater species, daphnids and rainbow trout, and the marine species, common mussels and mysid shrimp. In the 21-day chronic flow-through study on *Daphnia magna,* the percentage of dead offspring per female was significantly increased at 8.9 µg/L, the highest concentration at which adults survived. Although the number of offspring per female appeared to be reduced even at the lowest concentration (i.e., 2.7 µg/L), interpretation of results is complicated due to variability in control results (Thompson and Madeley, 1983a). The toxicity to rainbow trout was demonstrated in a bioconcentration study by Madeley and Maddock (1983a), in which trout were exposed to concentrations of 3.1 and 14.3 µg/L for 168 days. The fish were removed to fresh water for a depuration period of 105 days. Starting at day 63 of depuration, fish which previously had been exposed, began to exhibit behavioural symptoms associated with exposure to high concentrations. By day 69, all fish exposed to the higher concentration had died, as well as 50% of those from the group exposed to the lower concentration. Results from this study indicate that aquatic organisms may require a long exposure period for the toxicity of chlorinated paraffins to be demonstrated, and that based on results of other studies using short exposure periods, toxicity may be underestimated. In an 84-day flow-through test, reduction of mussel growth, as measured by shell length and weight of soft tissues, occurred at 9.3 µg/L, with no significant response at 2.3 µg/L (Thompson and Shillabeer, 1983). In a 28-day chronic flow-through study, mysids were exposed to measured concentrations of between 0.6 and 7.3 µg/L. Although no dose-response curve was established and mortalities in controls exceeded the commonly accepted value of 20%, mortality in mysids exceeded that of controls at all concentrations tested, significantly so at 1.2 and 2.4 µg/L (Thompson and Madeley, 1983b).

Based on the studies previously described, a no-observed-effect-level (NOEL) has not been determined for aquatic organisms, since the lowest level tested (i.e., 0.6 µg/L) did cause effects.

For the terrestrial environment, no acute studies were identified for any species. In a one-generation reproductive study in which mallard ducks *(Anas platyrynchos)* were fed 28, 166, and 1000 mg/kg-diet of a short chain CP (C_{10-13}), at the highest dose level

there was a slight decrease in eggshell thickness and 14-day embryo viability (Serrone *et al*, 1987). The NOEL for this study was, therefore, 166 mg/kg-diet.

Medium Chain Chlorinated Paraffins (C_{14-17}) - The toxicity over 60 days to common mussels and rainbow trout in a flow-through system was determined for a 52% chlorinated medium chain (C_{14-17}) chlorinated paraffin. The measured concentrations to which mussels were exposed were 220 and 3800 µg/L, and 1050 and 4800 µg/L for rainbow trout. In both studies, at the higher concentration, some of the chlorinated paraffins were lost from the dispersion due to their low solubility. This loss was not reduced significantly by dissolution in 1000 ppm of acetone. Although there was no mortality at either concentration for either species, reduced filtration activity of the mussels was consistently observed at the higher concentration (Madeley and Thompson, 1983a; Madeley and Maddock, 1983b).

Based on the limited studies available, the acute toxicity of medium chain CPs to birds is low. In a study of the medium chain CP, Cereclor S52, (C_{14-17}, 52% C1), the acute oral LD_{50}s were >24 606 mg/kg for ring-necked pheasants *(Phasianus colchicus)* and >10 280 mg/kg for mallard ducks. The acute dietary LC_{50} for the latter species was >24 063 mg/kg-diet (Madeley and Birtley, 1980).

Long Chain Chlorinated Paraffins ($\geq C_{18}$) - As with the medium chain length paraffins, the toxicity over 60 days of two long chain CPs (43% and 70% C1) to common mussels and rainbow trout in a flow-through system was determined. The measured concentrations to which mussels were exposed were 120 and 2200 µg/L for the 43% chlorinated CP, and 460 and 1330 µg/L for the 70% chlorinated CP. In the studies on rainbow trout, the measured concentrations tested were 970 and 4000 µg/L for the 43% chlorinated CP, and 840, 1900, and 3800 µg/L for the 70% chlorinated CP. For all studies, at the higher concentration, some of the chlorinated paraffins were lost from dispersion. Although there was no mortality at any concentration for either species, reduced filtration activity of the mussels was consistently observed at the higher concentration of both substances (Madeley and Thompson, 1983b; 1983c; Madeley and Maddock, 1983c; 1983d).

No relevant data were identified for any terrestrial species.

3.0 Assessment of "Toxic" Under CEPA

3.1 CEPA 11(a) Environment

Chlorinated paraffins are used in relatively large quantities in Canada, with demand being about 3.5 to 4 kt/yr. They are considered persistent as hydrolysis, oxidation, and photolysis are insignificant routes of transformation at ambient temperatures. Bioconcentration factors as high as 139 000 have been measured, and potential for biomagnification exists. Airborne dispersion of chlorinated paraffins has been reported in the United Kingdom and Sweden where monitoring data indicate widespread levels of low contamination in water, sediments, aquatic and terrestrial biota, and even commercial foods (Campbell and McConnell, 1980; Jansson *et al.,* 1993). Data on levels in any medium in the Canadian environment were not identified. Although data do exist for other countries, the relevance of these data could not be assessed due to the lack of information on comparability between Canadian production and that of other countries. As noted in Subsection 2.3.2, the use of modelling to predict environmental concentrations was considered unsuitable.

Short Chain Chlorinated Paraffins ($\leq C_{13}$) - Statistically significant effects were observed in aquatic invertebrate and fish species following chronic exposure to a range of concentrations from about 2.4 to 20 µg/L of a short chain CP (58% C1). Even at the lowest concentration tested, i.e., 0.6 µg/L, mortality of mysid shrimp exceeded that in controls. Results also indicate that toxicity may have been underestimated in available studies. Rainbow trout exposed to 14.3 µg/L for 168 days, and then removed to uncontaminated water, began on day 63 of depuration, to exhibit signs similar to those seen following exposure to acutely toxic concentrations. By day 69, all had died, suggesting delayed toxicity, as has been seen for other hydrophobic substances such as tetrachlorodibenzodioxin (TCDD).

Studies are lacking on the effects on terrestrial organisms. For short chain length paraffins, the NOEL for a one-generation reproductive study on mallard ducks was 166 mg/kg-diet.

Medium Chain Chlorinated Paraffins ($C_{14\text{-}17}$) - The toxicity of the medium chain chlorinated paraffins is lower than that of the short chain based on 60-day studies with mussels and rainbow trout. When exposed to a 52% chlorinated medium chain ($C_{14\text{-}19}$) CP, at concentrations of 3800 µg/L (mussels) or 4800 µg/L (rainbow trout), there was no mortality of either species, although reduced filtration activity of the mussels was consistently observed at the higher concentration (Madeley and Thompson, 1983a; Madeley and Maddock, 1983b).

Based on the limited studies available, the acute toxicity of medium chain chlorinated paraffins to birds is low. In a study of Cereclor S52, ($C_{14\text{-}17}$, 52% C1), the acute oral LD_{50}s were >24 606 mg/kg for ring-necked pheasants and >10 280 mg/kg for

mallard ducks. The acute dietary LC_{50} for mallard ducks was >24 063 mg/kg-diet (Madeley and Birtley, 1980).

Long Chain Chlorinated Paraffins (≥C is) - The toxicities of the long chain chlorinated paraffins are lower than those of the short chain CPs based on 60-day studies using mussels and rainbow trout. When mussels were exposed to concentrations of 2200 µg/L (C_{20-30}, 43% C1) or 1330 µg/L (C_{20-30}, 70% C1, there was no mortality, although reduced filtration activity was consistently observed. Similary, no mortality was observed in rainbow trout at concentrations of 4000 µg/L (C_{20-30}, 43% C1) or 3800 µg/L (C_{20-30}, 70% C1) (Madeley and Thompson, 1983b; 1983c; Madeley and Maddock, 1983c; 1983d).

No relevant data were identified on the terrestrial toxicity of long chain chlorinated paraffins.

Conclusion - Data were not identified on the concentrations of short, medium, or long chain chlorinated paraffins in the Canadian environment. As such, there are no data with which to compare levels reported as causing adverse effects in biota.

Therefore, it is not possible to assess whether these compounds are "toxic" as defined under Paragraph 11(a) of the *Canadian Environmental Protection Act.*

3.2 CEPA 11(b) Environment on Which Human Life Depends

None of the chlorinated paraffins is volatile. As such, only minor amounts of these compounds are expected to volatilize into the troposphere. Once in the troposphere, their estimated half-lives are short (few days in the summer) since they are subject to attack by hydroxyl radicals. Therefore, chlorinated paraffins are not expected to contribute significantly to depletion of stratospheric ozone or global warming.

On the basis of the available data, chlorinated paraffins are not considered to be "toxic" as defined under Paragraph 11(b) of the *Canadian Environmental Protection Act.*

3.3 CEPA 11(c) Human Life or Health

3.3.1 *Population Exposure*

Owing to their high octanol:water partition coefficients, it is likely that food is the principal source of exposure of the general population to chlorinated paraffins. However, because of the lack of adequate information on concentrations in environmental media to which humans are exposed and the lack of suitability of available models to estimate such levels (see Subsection 2.3.2), it is not possible to quantitatively estimate the total daily intake of chlorinated paraffins by the general population in Canada.

3.3.2 *Effects*

Available data on the toxicity of the chlorinated paraffins are limited. Epidemiological studies of exposed populations are not available and data on effects in humans are restricted to poorly documented clinical studies of the potential to induce irritation or sensitization of the skin following dermal application. Investigations of sub-chronic toxicity in experimental animals are available for the short, medium, and long chain CPs, although information on chronic toxicity or carcinogenicity in studies in experimental animals is available for only the short and long chain CPs. In general, results indicate that the target organs are the liver, kidneys, and the thyroid and parathyroid glands and that toxicity is inversely related to chain length and possibly increases with greater degrees of chlorination.

Information on developmental toxicity in experimental animals is available for the short, medium, and long chain CPs. Teratogenic effects have not been observed at dose levels below those that were toxic to the mothers. With the exception of one study using a short chain CP, embryo- and feto-toxic effects have not been observed at doses less than those that were toxic to the mothers. Identified studies of the reproductive toxicity of chlorinated paraffins are restricted to one for a medium chain CP only. The results indicated that suckling pups were more sensitive than those exposed *in utero.* Available data are inadequate to assess the neurotoxicity or immunotoxicity of the CPs.

Short Chain Chlorinated Paraffins ($\underline{\leq}C_{13}$) - Based on available data, carcinogenicity is potentially the most sensitive endpoint for the assessment of "toxic" for the short chain chlorinated paraffins under CEPA. The first step in evaluating whether short chain CPs are "toxic" as defined under Paragraph 11(c) of CEPA is, therefore, an assessment of the weight of evidence for genotoxic carcinogenicity, an effect for which it is believed there is no threshold.

Though the available information is inadequate to assess the carcinogenicity of short chain CPs in humans, in a well documented carcinogenesis bioassay, there was clear evidence of carcinogenicity of chlorinated paraffins (C_{12}, 60% C1) in $B6C3F_1$ mice and F344/N rats. It was further specified, however, that the maximum tolerated dose may have been exceeded in male and female rats (NTP, 1986a; Bucher *et al.,* 1987). It should be noted, however, that increases in tumor incidence were observed in rats in the absence of histopathological damage in at least one organ, i.e., the thyroid. Moreover, most of the mortality in exposed male rats occurred after 80 weeks, whereas overall survival in exposed female rats was reasonable compared with that in vehicle controls. The fact that the maximum tolerated dose may have been exceeded has, therefore, probably not significantly jeopardized the validity of the findings. Available data, though limited, also indicate that the short chain CPs are clastogenic and induce cell transformation in *in vitro* assays.

Based on these considerations, the short chain CPs have been classified in Group II ("Probably Carcinogenic to Humans") of the classification scheme developed by the

Bureau of Chemical Hazards for the assessment of "toxic" as defined under Paragraph 11(c) of CEPA (EHD, 1992).

The results of two studies (one for which the published account is only an abstract), indicate that short chain CPs may not be genotoxic but may act as peroxisome proliferators in the induction of liver adenomas in rats based on their lack of effect on unscheduled DNA synthesis but their positive response on cell proliferation following exposure of rats to single doses of a short chain CP up to 2000 mg/kg b.w. (Elcombe *et al.*, 1989; Ashby *et al.*, 1990). However, the pattern of tumor development in the NTP bioassay for short chain chlorinated paraffins is not entirely the same as that of identified epigenetic carcinogens. In addition, short chain chlorinated paraffins have been clastogenic and induced cell transformation in *in vitro* studies, though they have not been clastogenic or mutagenic in a limited number of *in vivo* assays. Therefore, available data are insufficient to conclude that short chain chlorinated paraffins induce any of the observed tumors in an epigenetic manner.

For substances classified in Group II, where data permit, estimated exposure is compared to quantitative estimates of cancer potency in order to characterize risk and provide guidance for further action, such as analysis of options to reduce exposure, under CEPA. However, because of the lack of adequate information on concentrations of short chain chlorinated paraffins in environmental media to which humans are exposed and the lack of suitability of available models to predict levels in the environment, it is not possible to quantitatively estimate the total average daily intake of these compounds by the general population in Canada, or to compare these estimates to quantitative estimates of cancer potency.

Substances classified in Groups I and II on the basis of the weight of evidence of carcinogenicity are considered non-threshold toxicants - substances for which there is some probability of harm for the critical effect at any level of exposure.

Short chain CPs are, therefore, considered to be "toxic" as defined under Paragraph 11(c) of the *Canadian Environmental Protection Act.*

This approach is consistent with the objective that exposure to non-threshold toxicants should be reduced wherever possible and obviates the need to establish an arbitrary "de minimis" level of risk for determination of "toxic" under the Act.

Medium Chain Chlorinated Paraffins (C_{14-17}) - Information has not been found on chronic toxicity or carcinogenicity of the medium chain chlorinated paraffins in studies in experimental animals. The weight of available limited data indicates that the medium chain CPs are not genotoxic.

Based on these considerations, medium chain CPs have been classified in Group VI ("Unclassifiable with respect to Carcinogenicity in Humans") of the classification scheme for carcinogenicity developed for the assessment of "toxic" under

Paragraph 11(c) of CEPA (EHD, 1992). For compounds classified in Group VI, a Tolerable Daily Intake (TDI) is derived on the basis of division of a no- or lowest-observed-(adverse)-effect-level [NO(A)EL or LO(A)EL] in animal species by an uncertainty factor.

The lowest effect level in the longer term studies of the effects of medium chain CPs was reported in a reproductive bioassay in which rats were exposed to one of three doses of a C_{14-17} (52% C1) CP in the diet for 28 days before mating, during mating, and for females, continuously up to postnatal day 21. Pups were also exposed from weaning to 70 days of age (IRDC, 1985). The lowest reported effect level in this study was in exposed pups - at 100 ppm in the diet [5.7 mg/(kg b.w. · day) in males and 7.2 mg/(kg b.w. · day) in females], there was a decrease in body weight gain by day 21 of lactation, an effect which continued after weaning but became less pronounced in males. (Histopathological effects were not observed at this concentration.) These effects appeared to be attributable to lactational rather than to *in utero* exposure.

The lowest reported effect levels in sub-chronic studies are similar to those observed in the reproductive study previously mentioned. In three sub-chronic studies, in which the medium chain CPs were administered in the diet to rats and dogs (Birtley *et al.*, 1980; Serrone *et al.*, 1987), the NO(A)ELs have ranged from 10 to 13 mg/(kg b.w. · day); effects observed at the next highest doses included increases in organ weights (liver and kidney), in serum hepatic enzymes, and in the smooth endoplasmic reticulum of the hepatocytes.

On the basis of these results, a tolerable daily intake (TDI) is conservatively (owing to the shortage of available data) derived as follows:

$$\text{TDI} = \frac{5.7 \text{ mg/(kg b.w. · day)}}{1000}$$

$$= 0.006 \text{ mg/(kg b.w. · day)} \; [6 \text{ µg/(kg b.w. · day)}]$$

where:

5.7 mg/(kg b.w. · day)	=	the lowest effect level reported to date (reproductive study)
1000	=	uncertainty factor [x 10 for intraspecies variation; x 10 for interspecies variation; x 10 for lack of data on carcinogenicity and less than chronic study; no uncertainty factor incorporated for LOEL rather than a NO(A)EL owing to the minor nature of the effects observed at this concentration]

In developmental studies on rats and rabbits, the medium chain CPs have not induced adverse effects at concentrations below those upon which the TDI derived for medium chain CPs is based (IRDC, 1984; 1983b).

Since it is not possible to quantitatively estimate exposure of the general population in Canada to medium chain chlorinated paraffins, the calculated TDI cannot be compared with the estimated total daily intake of these compounds in the general environment in Canada.

Available data are, therefore, considered inadequate to evaluate whether current concentrations of medium chain chlorinated paraffins present in the environment constitute a danger in Canada to human life or health; as a result, it is not possible to assess whether these compounds are "toxic" as defined under Paragraph 11(c) of the *Canadian Environmental Protection Act.*

Long Chain Chlorinated Paraffins ($\geq C_{18}$) - Based on available data, carcinogenicity is potentially the most sensitive endpoint for the assessment of "toxic" for the long chain chlorinated paraffins under CEPA. Therefore, the first step in evaluating whether long chain CPs are "toxic" as defined under Paragraph 11(c) of CEPA is an assessment of the weight of evidence for genotoxic carcinogenicity, an effect for which it is believed there is no threshold.

Though the available information is inadequate to assess the carcinogenicity of long chain CPs in humans, in a well documented carcinogenesis bioassay in rats and mice, there was no evidence of carcinogenicity for male F344/N rats, there was equivocal evidence of carcinogenicity for female F344/N rats and female $B6C3F_1$ mice, and there was clear evidence of carcinogenicity for male $B6C3F_1$ mice (NTP, 1986b). (For female mice, 60 to 70% of the early deaths in each group were attributed to utero-ovarian infection and it was noted that this may have decreased the sensitivity of the study to detect a carcinogenic effect.) The weight of available limited data indicates that the long chain CPs are not genotoxic.

Based on these considerations, the long chain CPs have been classified in Group III ("Possibly Carcinogenic to Humans") of the classification scheme for carcinogenicity developed for the assessment of "toxic" under Paragraph 11(c) of CEPA (EHD, 1992). For compounds classified in Group III, a Tolerable Daily Intake (TDI) is derived on the basis of division of a no- or lowest-observed-(adverse)-effect- level [NO(A)EL or LO(A)EL] in animal species by an uncertainty factor, that takes into account where appropriate, the limited evidence of carcinogenicity.

The lowest dose at which non-neoplastic effects have been observed in the longest term bioassay conducted to date following exposure to the long chain chlorinated paraffins is 100 mg/(kg b.w. · day) (NTP, 1986b; Bucher *et al.*, 1987). At this dose, there was a diffuse lymphohistiocytic inflammation in the liver and in the pancreatic and mesenteric lymph nodes in female rats. Splenic congestion was a secondary effect. In sub-chronic studies, the lowest reported effect level was 100 mg/(kg b.w. · day), which induced increases in liver weight and multifocal granulomatous hepatitis (characterized by inflammatory changes) and necrosis in female rats (Serrone *et al.*, 1987).

On the basis of these data, the TDI for the long chain CPs is derived as follows:

$$\text{TDI} = \frac{[100 \text{ mg/(kg b.w.} \cdot \text{day)}] \times 5/7}{1000}$$

$$= 0.071 \text{ mg/(kg b.w.} \cdot \text{day)} \; [71 \text{ µg/(kg b.w.} \cdot \text{day)}]$$

where:

100 mg/(kg b.w. · day) = the lowest effect level reported to date (well documented, two-year carcinogenicity bioassay)

5/7 = conversion of 5 days/week administration to daily exposure

1000 = uncertainty factor (x 10 for intraspecies variation; x 10 for interspecies variation; x 10 for use of a LOAEL rather than a NOAEL; additional factor for limited evidence of carcinogenicity not incorporated since there was no increase in tumor incidence in female rats in the target organ for the non-neoplastic effect on which the LOAEL is based)

In developmental studies in rats and rabbits, the long chain CPs have not induced adverse effects at concentrations below those upon which the TDI derived for long chain CPs is based (IRDC, 1981d; 1982b; 1983c; 1983d).

Since it is not possible to quantitatively estimate exposure of the general population in Canada to long chain chlorinated paraffins, the calculated TDI cannot be compared with the estimated total daily intake of these compounds in the general environment in Canada.

Available data are, therefore, considered inadequate to evaluate whether current concentrations of long chain chlorinated paraffins present in the environment constitute a danger in Canada to human life or health; as a result, it is not possible to assess whether these compounds are "toxic" as defined under Paragraph 11(c) of the *Canadian Environmental Protection Act.*

3.4 Conclusion

Due to their carcinogenicity, short chain chlorinated paraffins are considered to be "toxic" as defined under Paragraph 11(c) of the *Canadian Environmental Protection Act.* Data are considered inadequate to determine whether they are "toxic" as defined under Paragraph 11(a).

Available data are considered inadequate to determine whether medium or long chain chlorinated paraffins are "toxic" as defined under Paragraphs 11(a) and 11(c) of the *Canadian Environmental Protection Act.*

On the basis of available data, short, medium, and long chain chlorinated paraffins are not considered to be "toxic" as defined under Paragraph 11(b) of the *Canadian Environmental Protection Act.*

4.0 Recommendations for Research and Evaluation

Due to the relative persistence of the chlorinated paraffins, their potential for bioaccumulation, observed toxicity of short chain compounds to environmental organisms in experimental studies at concentrations similar to those measured in other countries, and potential carcinogenicity to humans (particularly for the short chain compounds), the first three recommendations are considered to be of high priority. The last two recommendations are considered to be of medium priority.

1. To complete the assessment of whether short, medium, or long chain chlorinated paraffins are "toxic" as defined under Paragraph 11(a) of CEPA, data are required on levels in the aquatic environment (particularly in biota and sediments) around the manufacturing site.

2. The releases of chlorinated paraffins to the environment from industrial sources (particularly metal-working) are not well characterized, but such characterization is needed to guide more widespread monitoring of CPs in the environment. This additional monitoring should be undertaken if estimates of releases show it to be warranted.

3. To enable the assessment of whether the medium or long chain CPs are "toxic" under Paragraph 11(c) of CEPA, development of suitable analytical methods and monitoring of these compounds in environmental media to which humans are exposed (particularly in food and mothers' milk) is required. Such information is also required in order to compare quantitative estimates of cancer potency to estimated total daily intake of the short chain chlorinated paraffins to characterize risk and provide guidance in establishing priorities for further action under CEPA.

4. A carcinogenicity bioassay for the medium chain CPs and additional data on the neurotoxicity and immunotoxicity for all of the CPs are desirable.

5. Additional research is also recommended on the mechanisms by which the short chain CPs induce tumors.

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Proposed Risk Management Approach for Chlorinated Paraffins (August 2008)

Proposed Risk Management Approach for Chlorinated Paraffins

Environment Canada
Health Canada

August 2008

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1. Issue

1.1 Background

Ecological and human health follow-up assessments were recently undertaken on chlorinated paraffins (CPs) under the provisions of the *Canadian Environmental Protection Act, 1999* (CEPA 1999). The assessments were undertaken as a follow-up to the work done on chlorinated paraffins under the first Priority Substances List (PSL1) program.

CPs were included on the PSL1 program under the 1988 *Canadian Environmental Protection Act* (CEPA 1988) for assessment of potential risks to the environment and human health. In 1993, Environment Canada and Health Canada published a proposed assessment report that concluded short chain chlorinated paraffins constitute or may constitute a danger to human health or life as set out in the CEPA. However, data identified at that time were considered insufficient to conclude whether short, medium or long chain chlorinated paraffins were harmful to the environment or whether medium or long chain chlorinated paraffins were considered a danger to human health.

Following the publication of the proposed assessment report, research to address data gaps relevant to the assessment of impacts of CPs on the environment was undertaken and an industry survey on the Canadian manufacture, import and uses of chlorinated paraffins was conducted for the years 2000 and 2001 through a *Canada Gazette* Notice issued pursuant to section 71 of CEPA 1999. Literature was also reviewed for new exposure and toxicological data on chlorinated paraffins on human and non-human organisms in Canada and elsewhere.

On June 11, 2005, the Ministers of the Environment and of Health published, for a 60-day public comment period, in the *Canada Gazette* Part I, the summary of the scientific results of the follow-up assessment on CPs and a statement indicating the measures they propose to take on the basis of scientific considerations. Comments were received from industry and industry associations on the follow-up assessment. Stakeholder input was carefully reviewed by Environment Canada and Health Canada. The final follow-up assessment report was revised based on these comments.

1.2 Final Follow-Up Assessment Report Conclusion for Chlorinated Paraffins

A Notice summarizing the scientific considerations of a final follow-up assessment report was published by Environment Canada and Health Canada in the *Canada Gazette* Part I for chlorinated paraffins that have the molecular formula $C_nH_xCl_{(2n+2-x)}$ in which $10 \leq n \leq 38$, on August 30, 2008, under paragraph 68(*b*) of CEPA 1999. The final follow-up assessment report concluded that:

- All CPs meet the criteria under paragraph 64(*c*)[1] of CEPA 1999; and

- CPs containing up to 20 carbon atoms meet the criteria under paragraph 64(*a*)[2] of CEPA 1999.

Based on conclusions of the assessment, it is therefore recommended that all CPs be added to Schedule 1 of CEPA 1999.

Furthermore, CPs containing up to 20 carbon atoms are predominantly anthropogenic, and the available data regarding their persistence and bioaccumulation potential indicate that they satisfy the criteria outlined in the Persistence and Bioaccumulation Regulations, made under CEPA 1999. CPs containing up to 20 carbon atoms will be recommended for addition to the Virtual Elimination List.

The full assessment report may be obtained from the Chemical Substances website at www.chemicalsubstances.gc.ca or from the Existing Substances Division, Environment Canada, Gatineau QC K1A 0H3; 819-953-4936 (fax); or by email at Existing.Substances.Existantes@ec.gc.ca.

1.3 Proposed Measure

Following a screening assessment of a substance under section 74 of CEPA 1999, a substance may be found to meet the criteria under section 64 of CEPA 1999. The Ministers can propose to take no further action with respect to the substance, add the substance to the Priority Substances List for further assessment, or recommend the addition of the substance to the List of Toxic Substances in Schedule 1 of CEPA 1999. Under certain circumstances, the Ministers must make a specific proposal to add the substances to the Virtual Elimination List.

In this case, the Ministers proposed to recommend the addition of all CPs to the List of Toxic Substances in Schedule 1 of CEPA 1999 and recommend CPs containing up to 20 carbon atoms for virtual elimination. As a result, the Ministers will develop an instrument respecting preventive or control actions to protect the health of Canadians and the environment from the potential effects of exposure to these CPs.

The final follow-up assessment report concluded that CPs containing up to 20 carbon atoms meet the virtual elimination criteria set out in subsection 77(4) of CEPA 1999 because:

- CPs containing up to 20 carbon atoms meet the criteria under section 64 of CEPA 1999;
- CPs containing up to 20 carbon atoms meet the criteria for persistence and bioaccumulation as defined by the Persistence and Bioaccumulation Regulations made under CEPA 1999;
- CPs in the environment result primarily from human activity; and
- CPs are not a naturally occurring radionuclide or a naturally occurring inorganic substance.

As a result, for CPs containing up to 20 carbon atoms, the Government of Canada will follow the process specified in CEPA 1999 for substances that meet the criteria for virtual elimination.

2. Background

2.1 Substance Information

CPs are chlorinated hydrocarbons (n-alkanes) that can have carbon (C) chain lengths ranging from 10 to 38. They are grouped by chain length: 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). For the ecological assessment, the long group was divided into three sub-groups: liquid LCCPs with 18-20 carbon atoms (C_{18-20} - liquid), liquid LCCPs with carbon atoms greater than 20 ($C_{>20}$ - liquid) and solid LCCPs with carbon atoms greater than 20 ($C_{>20}$ - solid).

Molecular Formula: $C_nH_xCl_{(2n+2-x)}$, $10 \leq n \leq 38$

SCCPs, MCCPs and the lower chlorinated LCCPs are mixtures that are viscous, colourless or yellowish dense oils. Highly chlorinated alkanes, with carbon chain length greater than 20, 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. The difference in chlorine content results in differing physical/chemical properties. Various stabilizers are often added to commercial CPs products in order to improve their thermal stability.

3. Why We Need Action

Environmental Risks

SCCPs have been detected in sewage treatment plant effluents from southern Ontario, surface water, sediments, plankton, invertebrates and fish from Lake Ontario and marine mammals from the St. Lawrence River.

SCCPs have also been detected in Arctic air, sediments from remote northern lakes and marine mammals from the Canadian Arctic. SCCPs detected in Arctic biota and lake sediments, in the absence of significant sources of SCCPs in the region, suggest that long-range atmospheric transport of SCCPs is occurring.

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. The available toxicity data indicate that SCCPs and MCCPs, as well as C_{18-20} LCCPs, may be harmful to certain aquatic species (e.g., *Daphnia magna*) at low concentrations.

SCCPs and MCCPs, as well as C_{18-20} LCCPs, are also considered to be both highly persistent and bioaccumulative. 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. All CPs have been found to meet the criteria under paragraph 64(*c*) of CEPA 1999.

Human Health Risks

A comprehensive literature search was conducted (SCCP, up to February 2001; MCCP and LCCP, up to September 2000) to identify critical new data for the assessment of human health risk under paragraph 64(*c*) of CEPA 1999.

For all CPs (SCCP, MCCP and LCCP), there was insufficient information to characterize the effects of exposure upon humans. Reports of health effects were limited to studies with laboratory animals.

There is low confidence in the upper-bounding estimates of exposure to all CPs. 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 SCCP and MCCP, and, as a result, intake of the individual groups of chlorinated paraffins (SCCP, MCCP and LCCP) from these sources has been overestimated.

Lifetime exposure of SCCP to mice resulted in cancer of the liver and thyroid. In a similar study with rats, there were tumours in liver, thyroid and kidney. Although there are uncertainties, it could not be concluded that these effects were not relevant to humans, i.e., there is some probability of harm at any level of exposure.

In other studies, non-cancer effects (on liver, kidney and thyroid) were observed in rats. The World Health Organization has derived a tolerable daily intake for SCCP. A tolerable daily intake is the level of intake to which it is believed that a person may be exposed daily over a lifetime without deleterious effects.

SCCP has been identified in Canada in ambient air, river water and fish. Estimates of total daily intake by Canadians were calculated by supplementing these data with concentrations of SCCP reported in foods in an older British study. The highest intake of SCCP calculated for the population of Canada is within the range of the tolerable daily intake of SCCP derived by the World Health Organization.

No studies were identified which had investigated carcinogenicity of MCCP in laboratory animals. Non-cancer effects (decrease in body weight) were observed in the offspring of rats exposed to MCCP. Effects upon liver and thyroid were reported elsewhere. A tolerable daily intake was derived on the basis of non-cancer effects.

Concentrations of MCCP were identified in fish in Canada. However, the estimates of total daily intake of MCCP by Canadians were based almost entirely upon older data from Britain. The estimated intake of MCCP by several age groups in the Canadian population exceeded the value of the tolerable daily intake.

In a study with laboratory animals, LCCP was carcinogenic to male mice but not to male rats. Non-cancer effects were observed in the liver, pancreas and lymph nodes of female rats. Based upon these effects, a tolerable daily intake was derived.

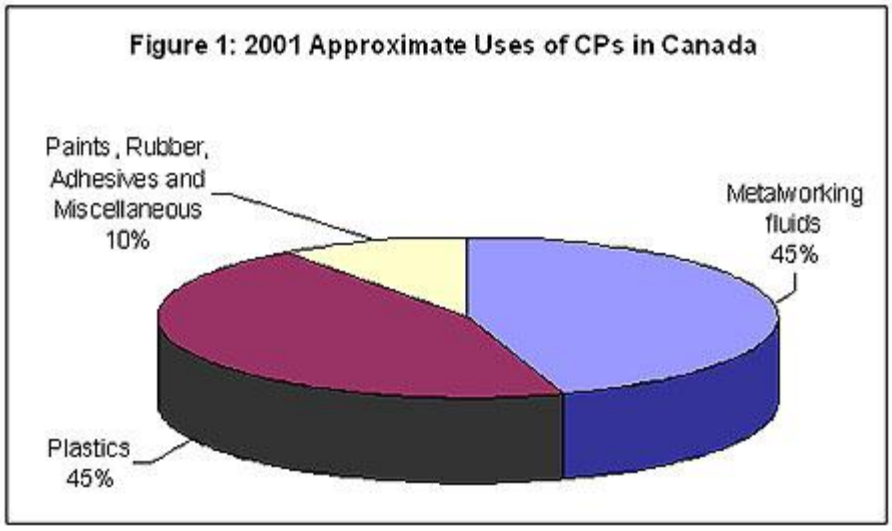
Concentrations of LCCP were not identified in any medium in Canada (food, drinking water, air, soil). The estimates of total daily intake for the Canadian population were based entirely upon older data from Britain. The highest estimate of total daily intake for Canadians was within the same order of magnitude as the tolerable daily intake.

4. Current Uses and Industrial Sectors

Total reported annual usage of chlorinated paraffins in Canada (production + imports - exports) was approximately 2.8 kilotonnes in 2000 and 2001. As production of CPs in Canada has stopped, CPs are now imported into Canada as chemical formulations from foreign producers or as formulations in products such as paints, sealants, plastics and metalworking fluids.

The vast majority of CP consumption in Canada is MCCPs, while much smaller quantities of SCCPs and LCCPs are also being consumed in specific applications. The two dominant end-use applications for CPs in Canada are in the formulation of metalworking fluids such as cutting oils

and high pressure lubricating oils used in the metalworking industry and as a plasticizer in polyvinyl chloride (PVC) applications. CPs are also used as flame retardants in various plastics or formulated chemical products (e.g., adhesives, paints, sealants). The use profile information provided in Figure 1 was developed based upon information collected from an Environment Canada survey of CP producers and end-users in Canada.



Metalworking Fluids

Approximately 15 metalworking fluid formulators in Canada consumed an estimated 1.2 kilotonnes of CPs in 2001. CPs are used as extreme pressure additives across a wide temperature range to enhance lubrication and surface finish in demanding metalworking and forming applications where hydrodynamic lubrication cannot be maintained.

Polyvinyl Chloride

Approximately 15-25 companies in the polyvinyl chloride (PVC) processing sector in Canada consumed an estimated 1.2 kilotonnes of CPs in 2001. CPs are used in the PVC processing industry as secondary plasticizers, and in some cases as flame retardants. While there are several different sub-sectors within the PVC processing industry, the use of CPs (on a volume basis) has historically been restricted to three specific sub-sectors where they have been used as secondary plasticizers and flame retardants, namely: (i) flooring manufacturing; (ii) wire and cable sheathing and insulation; and (iii) wall coverings and emulsions. MCCPs are the dominant CPs used in the PVC processing industry, with only minor amounts of SCCPs or LCCPs used.

Paints and Coatings, Adhesives and Sealants, and Rubber and Elastomers

Approximately 0.4 kilotonnes of CPs were used in the Canadian paints and coatings, adhesives and sealants, and rubber and elastomer sectors in 2001. CPs are used in other end-use segments in Canada, in addition to metalworking fluids and PVC, although in much smaller quantities. These specific applications are: (i) paints and coatings; (ii) adhesives and sealants; and (iii) rubber and elastomers. The majority of CPs consumed in these applications are MCCPs along with small amounts of LCCPs. CPs are used in these applications because of their ability to: (i) improve the flexibility (and therefore the durability) of the coating; and (ii) reduce the amount of time needed to achieve "tack-free" conditions.

5. Presence in the Canadian Environment and Exposure Sources

5.1 Releases to the Environment

There are no known natural sources of CPs. The major sources of release of CPs into the Canadian environment are likely the formulation and manufacturing of products containing CPs. 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 carry-off and spent bath. These releases are collected in sewer systems and often ultimately end up in the effluents of sewage treatment plants.

Landfilling is also a major disposal route for CP-containing 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. Outlined, in Table 1, below are estimates of CP releases in Canada, which are based upon the demand profile for CPs in Canada and the release factors for CPs that were contained in European risk assessment reports.

Table 1: 2001 Estimated Releases of CPs in Canada (kilotonnes)

Production/End-use Area	Estimated Annual Releases
Metalworking fluids formulation and end-use	0.3
Plastics production and end-use	<0.1
Rubber production and end-use	<<0.1
Sealants, adhesives and caulks formulation and end-use	<<0.1
Paint formulation and end-use	<<0.1
Other	<<0.1

5.2 Exposure Sources

As there are no known natural sources of CPs, the release of CPs to the environment is primarily from formulation and use. The release of CPs as a result of leaching or volatilization from products containing the substances is thought to be negligible, as was the leaching of CPs from products that are landfilled. The primary source of release of CPs is predicted to occur from the metalworking sector through the disposal of metalworking fluid. SCCPs have been found in the Arctic food web. The higher volatility of certain SCCPs suggests their presence resulted from long-range atmospheric transportation.

6. Overview of Existing Actions

6.1 Existing Canadian Risk Management

Though there are at present no restrictions on uses of CPs in Canada, there are two categories of CPs that are listed on the National Pollutant Release Inventory (NPRI). CPs are reported to the NPRI as "alkanes, C_{10-13}, chloro" (SCCPs) and "alkanes, C_{6-18}, chloro", a category that includes SCCPs, MCCPs and some LCCPs.

6.2 Existing International Risk Management

Existing European Initiatives

A Marketing and Use Directive in the European Union (EU), (Directive 2002/45/EC), restricts the concentration of short chain CPs in metalworking and leather fat liquoring preparations to 1% or less. These restrictions took effect on January 6, 2004.

Also, SCCPs are on the initial list of 16 substances identified as substances of very high concern under REACH[3] (Registration, Evaluation, Authorization and Restriction of Chemical substances).

U.S. Initiatives

The U.S. EPA added the category of polychlorinated alkanes to its list of toxic chemicals subject to Toxics Release Inventory reporting under EPCRA section 313 (see 40 CFR 372.65) based on available carcinogenicity and ecotoxicity data for short chain species (59 Federal Register 61432, November 30, 1994).

International Agreements

In May 2006, the Parties to UNECELRTAP Convention's Protocol on Persistent Organic Pollutants agreed that SCCPs meet the UNECE criteria for long term transboundary transport, persistence and bioaccumulation. Discussions regarding addition of SCCPs to the POPs Protocol are ongoing under the Convention. The United Nations Environment Program is also considering adding SCCPs as a POP to the Stockholm Convention on Persistent Organic Pollutants.

In October 2001, Canada offered to prepare an information dossier for the ad hoc Expert Group on Persistent Organic Pollutants (POPs) under the United Nations Economic Commission for Europe (UNECE) Convention on Long-range Transboundary Air Pollution (LRTAP) relating to the possible addition of short chain CPs to the LRTAPPOPs Protocol. A draft dossier incorporating the new information generated by the National Water Research Institute (NWRI) was submitted to the LRTAP Convention in the spring of 2002.

7. Considerations

7.1 Alternative Chemicals, Substitutes, Technologies and/or Techniques

In determining risk management options, there is a need to consider the risks and costs of potential alternative substances and technologies.

Metalworking Fluids

There are two approaches to minimizing the releases of CPs within the metalworking industry, specifically to: (i) increase the adoption rate of substitutes to CPs among metalworking fluid formulators and end-users; and (ii) increase the adoption of best management practices by end-users of metalworking fluids.

Although substitutes to CPs are available to metalworking fluid formulators, several issues need to be considered in their implementation, as some potential alternatives are:

- not technically suitable for all applications;
- more costly; and
- may also pose environmental and health risks.

Polyvinyl Chloride

In PVC manufacturing, CPs are used primarily in applications where moderate plasticizing and flame retardant properties are required at low cost. Moreover, it is not anticipated that there would be many technical obstacles if CPs had to be replaced with alternative plasticizers and/or flame retardants. Analysis of CP alternatives suggests that, in many cases, the overall technical characteristics of the PVC product such as flexibility and stability would improve with the use of alternatives. Although technically feasible, the use of these alternatives would likely increase the raw material costs for manufacturers and they may also pose environmental and health risks.

Paints and Coatings, Adhesives and Sealants, and Rubber and Elastomers

Very small quantities of CPs are used annually in Canada in the formulation of paints and coatings, adhesives and sealants, and rubber and elastomers relative to metalworking fluids and PVC. Less than 100 tonnes of both MCCPs and LCCPs were reported to Environment Canada for the year 2001. The favorable characteristics provided by CPs include good compatibility with the resin systems where they are used; they are colourless; they are non-volatile and do not add to volatile organic compounds (VOC) content of a coating system; and they have low viscosity.

The use of CPs in the rubber industry has historically involved the utilization of SCCPs to manufacture rubberized conveyor belts for the underground mining industry as well as other technical products such as hoses and gaskets. They are used in these applications because of their superior flame retardant properties, which are often required in order to meet fire standard codes for products.

Technical barriers have been reported for adhesives and sealants substitutes as well; the primary technical issue is that they are more prone to bleeding from the sealant product, thus directly affecting the durability of the sealant and the substrate.

7.2 Socio-Economic Considerations

Socio-economic factors have been considered in the selection process for an instrument respecting preventive or control actions, and in the development of the risk management and human health objectives. Socio-economic factors will also be considered in the development of instrument(s) and/or tool(s) as identified in the *Cabinet Directive on Streamlining Regulation* (Treasury Board of Canada Secretariat, 2007) and the guidance provided in the Treasury Board document *Assessing, Selecting, and Implementing Instruments for Government Action*.

Socio-economic considerations for CPs included a qualitative analysis of costs to industry in terms of switching to alternatives and benefits to the public.

It was determined that the cost of using alternative substances would have a minimal economic effect for most uses. However, where the costs of alternatives are significant there would be an increase in the raw material costs for manufacturers that are currently using CPs in their products. This increase, along with other re-formulation costs, could hinder the competitiveness of these products in domestic and foreign markets.

8. Proposed Objectives

8.1 Environmental and Human Health Objective

The proposed human health objective for SCCPs, MCCPs and LCCPs is to minimize human exposure to the extent practicable.

The environmental objective for CPs with up to 20 carbon atoms is virtual elimination (VE) as specified under subsection 77(4) of CEPA 1999.

8.2 Risk Management Objectives

The proposed risk management objective is to reduce releases of CPs, to the lowest level possible, from all sources and prevent the re-introduction of their manufacture in Canada.

9. Proposed Risk Management

9.1 Proposed Risk Management Instrument

As recommended by the Government of Canada's *Cabinet Directive on Streamlining Regulation*[4] and criteria identified in the Treasury Board document entitled Assessing, Selecting, and Implementing Instruments for Government Action, the proposed risk management instrument was selected using a consistent approach taking into consideration information received from industry and other information available at the time.

In order to achieve the risk management objective and to work towards achieving the environmental or human health objective, the risk management being considered for CPs is a prohibition regulation. The prohibition regulation would prohibit the manufacture, use, import, sale and offer for sale of CPs.

Specific use exemptions for some CP uses may be allowed where it can be demonstrated that alternatives are not technically or economically feasible. If specific use exemptions are granted, complementary tools will be considered as a means to ensure that releases of CPs are reduced to the lowest level possible.

9.2 Implementation Plan

CPs will be recommended for addition to Schedule 1 of CEPA 1999. Furthermore, consistent with the provisions of CEPA 1999, it will be recommended that CPs containing up to 20 carbon atoms be subject to Virtual Elimination. A proposed instrument will be published in the *Canada Gazette* Part I following consultations with stakeholders.

Monitoring for CPs in the environment will be considered under a more comprehensive monitoring and surveillance strategy for all substances in the Chemicals Management Plan. Monitoring has been identified as a key pillar in the Chemicals Management Plan to: collect and generate human health and environmental data to inform decision-making; provide an adaptive management framework to support intervention; and measure the efficacy of preventive and mitigation actions. Monitoring may include the analysis of landfill leachate to confirm that it is not being released from landfill, and monitoring to measure the efficacy of preventive and mitigation actions.

10. Consultation Approach

The Risk Management Approach (RMA) for CPs will be posted on Environment Canada’s website for comments:

http://www.ec.gc.ca/CEPARegistry/participation/

http://www.ec.gc.ca/ceparegistry/documents/subs_list/ChlorinatedParaffins/RiskManagement.cfm

Environment and Health Canada will seek advice on the proposed risk management objectives and risk management instruments. The design of further stakeholder consultations will be assessed following the publication of the proposed RMA and receipt of comments. These consultations could cover the risk management process, the proposed instruments and alternatives to CPs.

Stakeholders in the consultation process will include associations representing formulators and manufacturers of CPs and products containing CPs. As one proposed instrument is the regulation of CPs, users of products containing CP would also be included in the consultation. Other stakeholders will include various levels of government and environmental non-government organizations (ENGOs).

11. Next Steps / Proposed Timeline

Actions	Date
Consult on Risk Management Approach	Fall 2008
Initiate Development of Proposed Instrument(s)	Fall 2008
Consult on Proposed Instrument(s)	Winter 2009
Publication on Proposed Instruments in *Canada Gazette* I	Summer 2010
Publication on Proposed Instruments in *Canada Gazette* II	Winter 2012

Industry and other interested stakeholders are invited to submit comments on the content of this proposed risk management approach or other information that would help to inform decision making. Please submit comments prior to October 29, 2008, since the Government of Canada will be moving forward with the risk management of CPs after this date. Pursuant to section 313 of CEPA 1999, any person who provides information to the Minister under CEPA 1999 may submit with the information a request that it be treated as confidential. During the development of the risk management instrument(s) and/or tool(s), there will be opportunity for consultation on the proposed instrument. Comments and information submissions on the proposed risk management approach should be submitted to the address provided below:

Chemicals Management Division
351 St. Joseph Blvd.
Gatineau QC
K1A 0H3
Tel.: 819-934-6449
Fax: 819-953-8963
Email: RiskManagementPrograms@ec.gc.ca

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Treasury Board of Canada Secretariat. 2007. Cabinet Directive on Streamlining Regulation, section 4.4. www.regulation.gc.ca/directive/directive01-eng.asp

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Canada Gazette Part I; Vol. 139, No. 24 - June 11, 2005; *Publication of results of investigations and recommendations for the substances short chain chlorinated paraffins, medium chain chlorinated paraffins and long chain chlorinated paraffins (paragraphs 68(b) and 68(c) of the* Canadian Environmental Protection Act, 1999)
http://www.gazette.gc.ca/archives/p1/2005/2005-06-11/html/ notice-avis-eng.html#i8

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; Environment Canada/Health Canada February 2008. http://www.ec.gc.ca/CEPARegistry/documents/subs_list/ChlorinatedParaffins/CPs_TOC.cfm

Analysis of Options to Mitigate Chlorinated Paraffin Releases in Canada; Environment Canada, January 2005

Survey on the Canadian manufacture, import and uses of chlorinated paraffins for the years 2000 and 2001 through a Canada Gazette Notice issued pursuant to section 71 of the Canadian Environmental Protection Act, 1999

Footnotes

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[1] Paragraph 64(*c*) of CEPA 1999 defines a substance as "toxic" if it is entering or may enter the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

[2] Paragraph 64(*a*) of CEPA 1999 defines a substance as "toxic" if it is 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.

[3] REACH is a European Community Regulation on chemicals and their safe use (EC 1907/2006).

[4] Section 4.4 of the *Cabinet Directive on Streamlining Regulation* states that "Departments and agencies are to: identify the appropriate instrument or mix of instruments, including regulatory and non-regulatory measures, and justify their application before submitting a regulatory proposal".

Government of Canada. 2004. Follow-up Report on a PSL1 Substance for Which There Was Insufficient Information to Conclude Whether the Substance Constitutes a Danger to the Environment. Ottawa, Ontario.

Canadian Environmental Protection Act, 1999

Follow-up Report on a PSL1 Substance for Which There Was Insufficient Information to Conclude Whether the Substance Constitutes a Danger to the Environment

Chlorinated Paraffins

April 2004

SYNOPSIS

Chlorinated paraffins (CPs) are chlorinated derivatives of n-alkanes, having carbon chain lengths ranging from 10 to 38 and a chlorine content ranging from 30 to 70% by weight. 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 that appeared on the first Priority Substances List (PSL1) were assessed to determine whether they should be considered "toxic" as defined under the *Canadian Environmental Protection Act* (CEPA). With the data available at that time, it was concluded in the PSL1 assessment that SCCPs were "toxic" because they were constituting or may constitute a danger in Canada to human life or health under paragraph 11(c) of CEPA 1988; however, there was insufficient information to conclude whether SCCPs, MCCPs or LCCPs could have immediate or long-term harmful effects on the environment under paragraph 11(a) or whether MCCPs or LCCPS could be considered "toxic" under paragraph 11(c). .

Subsequent to the completion of the PSL1 assessments, a revised CEPA, CEPA 1999, came into effect. Paragraph 64(a) of CEPA 1999 has a definition of "toxic" that is similar to that in paragraph 11(a) under the original CEPA, and addresses whether a substance has or may have an immediate or long-term harmful effect on the environment. However, in CEPA 1999 paragraph 64(a) has been expanded to include effects on biodiversity. Research to address data gaps relevant to the assessment of impacts on the environment was funded. Recent literature was reviewed for new data on concentrations in the environment, as well as for information on the effects on human and non-human organisms.

Total reported annual usage of CPs in Canada (production + imports – exports) was approximately 3000 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 is only one manufacturer of CPs in Canada, and only MCCPs and LCCPs are produced at this facility. In 2000, their production capacity was reported to be 8.5 kilotonnes.

There are no known natural sources of CPs. The major sources of release of CPs into the Canadian environment are likely the formulation and manufacturing of products containing CPs, such as polyvinyl chloride (PVC) plastics, 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.

In this assessment, the LCCPs were divided into two groups: (1) $C_{18–20}$ and $C_{>20}$ liquid LCCPs (together referred to as liquid LCCPs) and (2) $C_{>20}$ solid LCCPs. This division

was made based on their different physical/chemical properties, which are related to the much higher chlorine content of $C_{>20}$ solid LCCPs relative to liquid LCCPs.

SCCPs have been detected in the following environmental samples from Canada: in Arctic air, in sediments from remote northern lakes, in sewage treatment plant effluents from southern Ontario, in surface water, sediments and fish from Lake Ontario and in marine mammals from the Canadian Arctic and the St. Lawrence River. MCCPs have been detected in effluent from a CPs manufacturing facility near Cornwall, Ontario, and also in sediments near this facility, in fish from Lake Ontario and in beluga from the St. Lawrence River. Internationally, MCCPs have been detected in sewage sludge, surface water near a CPs manufacturing plant, sediments, fish, aquatic invertebrates and earthworms. 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, 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. 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 16 440–25 650 wet weight (wet wt.) in trout from Lake Ontario indicate that SCCPs are bioaccumulating to a high degree in aquatic biota in Canada. This is supported by very high bioconcentration factors (BCFs) for SCCPs measured in mussels (5785–138 000 wet wt.). Despite the lack of valid laboratory studies of BCFs and BAFs, MCCPs and liquid LCCPs have been found to have significant potential to bioaccumulate in aquatic food webs: field BAFs for MCCPs in Lake Ontario fish are estimated to range from 7.77×10^5 to 5.45×10^6 wet wt.

Furthermore, MCCPs were found to have biomagnification factors (BMFs) greater than 1 in the Lake Ontario food web and in laboratory studies with rainbow trout and oligochaetes. The LCCP $C_{18}H_{30}Cl_7$ had BMF values greater than 1 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, MCCPs and LCCPs have octanol–water partition coefficient (log K_{OW}) values greater than 7, elevated concentrations of MCCPs have been measured in aquatic biota from the St. Lawrence estuary, the United States and Australia, and elevated concentrations of LCCPs have been found in marine benthic organisms in Australia. Therefore, based on these data, as well as the physical/chemical similarities of CP chain

lengths, it is concluded that SCCPs, MCCPs and liquid LCCPs meet the bioaccumulation criteria as defined in the Persistence and Bioaccumulation Regulations of CEPA 1999.

In cases where appropriate Canadian environmental exposure data were not available, international concentration data were used for the risk quotients. Conservative risk quotients indicate that SCCPs, MCCPs and liquid LCCPs have the potential to harm pelagic and soil organisms, that SCCPs and MCCPs may harm benthic organisms and that SCCPs have the potential to harm fish-eating wildlife through food chain effects. Based on the limited toxicity data available and the use of environmental exposure data for liquid LCCPs, $C_{>20}$ solid LCCPs appear to have low potential to harm Canadian wildlife through food chain effects. However, no toxicity studies for $C_{>20}$ solid LCCPs were available with daphnids, which was the most sensitive organism for SCCPs, MCCPs and liquid LCCPs.

As CPs have been found to persist in the environment and to have the potential to bioaccumulate, risk assessments for these compounds were more conservative than for compounds not meeting the criteria defined in the Persistence and Bioaccumulation Regulations of CEPA 1999.

There are special concerns about persistent and bioaccumulative substances. Persistent substances can remain in the environment for long periods of time, increasing the probability and the duration of exposure. In addition, the long-range atmospheric transport of persistent substances may result in low-level, widespread contamination. Bioaccumulative substances have the potential to biomagnify; consequently, releases of extremely low concentrations of persistent and bioaccumulative substances may — either alone or in combination with similar substances — cause severe adverse effects.

Based on the information available, it is proposed that SCCPs, MCCPs and $C_{18–20}$ and $C_{>20}$ liquid LCCPs are entering 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 proposed that SCCPs, MCCPs and $C_{18–20}$ and $C_{>20}$ liquid LCCPs be considered “toxic” as defined in paragraph 64(a) of CEPA 1999. SCCPs, MCCPs and $C_{18–20}$ and $C_{>20}$ liquid LCCPs are persistent, bioaccumulative and predominantly anthropogenic and thus they also meet the criteria for Track 1 substances under the Government of Canada Toxic Substances Management Policy, making them candidates for virtual elimination.

Government of Canada. 2004. Follow-up Report on a PSL1 Substance for Which Data Were Insufficient to Conclude Whether the Substance Was "Toxic" to Human Health Chlorinated Paraffins. Ottawa, Ontario.

Canadian Environmental Protection Act, 1999

Follow-up Report on a PSL1 Substance for Which Data Were Insufficient to Conclude Whether the Substance Was "Toxic" to Human Health
Chlorinated Paraffins

October 2003

SYNOPSIS

Short-chain chlorinated paraffins (SCCP) are imported into Canada for use as additives in extreme-pressure lubricants, plasticizers and flame retardants. Medium- and long-chain chlorinated paraffins (MCCP and LCCP, respectively) are produced in, and imported into, Canada for similar uses.

Chlorinated paraffins 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. As outlined in the Assessment Report released in 1993, relevant data identified before August 1992 were considered insufficient to conclude whether MCCP and LCCP were "toxic" to human health as defined in Paragraph 11(c) of CEPA 1988. As also outlined in the Assessment Report released in 1993, SCCP were considered to be "toxic" as defined under Paragraph 11(c) of CEPA 1998. This conclusion was based principally on the observed carcinogenicity of these compounds, for which available information on mode of action could not preclude it being the result of direct interaction with genetic material. To set context for the update on MCCP and LCCP, more recent data on the effects of SCCP on human health have also been considered here, and the conclusion under Paragraph 11(a) of CEPA 1988 has been updated.

For SCCP, 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 SCCP 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). Additional documentation of available studies and consideration in additional investigations of the reversibility of precursor lesions in the absence of continued exposure is desirable. However, reported data on mode of induction of tumours in addition to the weight of evidence that SCCP 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 SCCP approach or exceed the TDI for these compounds, which, on the basis of available information, is likely also protective for carcinogenicity.

Therefore, the Ministers of the Environment and of Health confirm that short-chain chlorinated paraffins are "toxic" to human health as defined in Paragraph 64(c) of the *Canadian Environmental Protection Act, 1999* (CEPA 1999).

For MCCP and LCCP, 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, upperbounding estimates of daily intake for MCCP and LCCP are within the same order of magnitude of, or exceed, the TDIs for these compounds.

Therefore, it is proposed that there is reason to suspect that medium- and longchain chlorinated paraffins are "toxic" to human health as defined in Paragraph 64(c) of the *Canadian Environmental Protection Act, 1999* (CEPA 1999).

Acquisition of data on levels of these compounds (SCCP, MCCP and LCCP) within foodstuffs in Canada continues to be considered a high priority.

Government of Canada. 2008. 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. Ottawa, Ontario.

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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Canadian Environmental Protection Act, 1999

Follow-up Report on a PSL1 Substance for Which Data Were Insufficient to Conclude Whether the Substance Was "Toxic" to Human Health

Chlorinated Paraffins

October 2003

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List of Acronyms and Abbreviations

CEPA 1988	*Canadian Environmental Protection Act*
CEPA 1999	*Canadian Environmental Protection Act, 1999*
CoA	coenzyme A
CYP	cytochrome P-450
DNA	deoxyribonucleic acid
ECNI	electron capture negative ion
GC	gas chromatography
HRGC	high-resolution gas chromatography
HRMS	high-resolution mass spectrometry
kg-bw	kilogram body weight
K_{ow}	octanol/water partition coefficient
LCCP	long-chain chlorinated paraffins
LOAEL	Lowest-Observed-Adverse-Effect Level
LOEL	Lowest-Observed-Effect Level
LRMS	low-resolution mass spectromety
MCCP	medium-chain chlorinated paraffins
MS	mass spectrometry
NOAEL	No-Observed-Adverse-Effect Level
NOEL	No-Observed-Effect Level
NTP	National Toxicology Program (U.S.A.)
PPARα	peroxisome proliferator activated receptor, α isoform
PSL1	first Priority Substances List
SCCP	short-chain chlorinated paraffins
T_3	triiodothyronine
T_4	thyroxine
TD_{05}	Tumorigenic $Dose_{05}$, the dose associated with a 5% increase in tumour incidence
TDI	Tolerable Daily Intake
TSH	thyroid stimulating hormone
UDP	uridine diphosphate
UDPG	uridine diphosphoglucose
UDPGGT	uridine diphosphoglucose glucuronosyl transferase
UDPGT	uridine diphosphate glucuronosyl transferase

PREFACE

For very few of the substances on the first Priority Substances List (PSL1) for which data were considered insufficient to conclude whether they were "toxic" under Section 11 of the 1988 *Canadian Environmental Protection Act* (CEPA 1988) did this conclusion apply to both the environment (under Paragraphs 11(a) and 11(b) of CEPA 1988) and human health (under Paragraph 11(c) of CEPA 1988). Medium-chain and long-chain chlorinated paraffins are two of these substances. While information on the environmental effects of short-chain chlorinated paraffins was considered insufficient to conclude whether they were "toxic" under Paragraph 11(b) 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 were also examined.

In the documentation that follows, 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 (i.e., determination of "toxic" under Paragraphs 64(a) and 64(b) of the *Canadian Environmental Protection Act, 1999* [CEPA 1999]) is presented initially, followed by information relevant to assessment of effects on human health (determination of "toxic" under Paragraph 64(c) of CEPA 1999).

SYNOPSIS

Short-chain chlorinated paraffins (SCCP) are imported into Canada for use as additives in extreme-pressure lubricants, plasticizers and flame retardants. Medium- and long-chain chlorinated paraffins (MCCP and LCCP, respectively) are produced in, and imported into, Canada for similar uses.

Chlorinated paraffins 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. As outlined in the Assessment Report released in 1993, relevant data identified before August 1992 were considered insufficient to conclude whether MCCP and LCCP were "toxic" to human health as defined in Paragraph 11(c) of CEPA 1988. As also outlined in the Assessment Report released in 1993, SCCP were considered to be "toxic" as defined under Paragraph 11(c) of CEPA 1998. This conclusion was based principally on the observed carcinogenicity of these compounds, for which available information on mode of action could not preclude it being the result of direct interaction with genetic material. To set context for the update on MCCP and LCCP, more recent data on the effects of SCCP on human health have also been considered here.

For SCCP, 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 SCCP 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). Additional documentation of available studies and consideration in additional investigations of the reversibility of precursor lesions in the absence of continued exposure is desirable. However, reported data on mode of induction of tumours in addition to the weight of evidence that SCCP 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 SCCP approach or exceed the TDI for these compounds, which, on the basis of available information, is likely also protective for potential carcinogenicity.

Therefore, it is proposed that there is no reason to revise the conclusion for PSL1 that short-chain chlorinated paraffins are "toxic" as defined under Paragraph 64(c) of the *Canadian Environmental Protection Act, 1999*.

For MCCP and LCCP, 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 MCCP and LCCP are within the same order of magnitude of, or exceed, the TDIs for these substances.

Therefore, it is proposed that there is reason to suspect that medium- and long-chain chlorinated paraffins are "toxic" to human health as defined in Paragraph 64(c) of the *Canadian Environmental Protection Act, 1999* (CEPA 1999).

Acquisition of data on levels of these compounds (SCCP, MCCP and LCCP) within foodstuffs in Canada continues to be considered a high priority.

1.0 INTRODUCTION

A common Introduction, which describes the process for the preparation of the updates of the Assessment Reports for the seven substances (including medium-chain and long-chain chlorinated paraffins [MCCP and LCCP, respectively]) on the first Priority Substances List (PSL1) for which data were considered insufficient to conclude whether the substances were "toxic" to human health under the 1988 *Canadian Environmental Protection Act* (CEPA 1988) is posted on all web sites where the Assessment Reports appear.[1]

The strategy for the literature search to identify critical new data (including commercial activity in Canada, human exposure and effects) on short-chain chlorinated paraffins (SCCP), MCCP and LCCP is presented in Appendix A of this report. Only relevant data acquired prior to February 2001 and December 2000 were considered in the re-examination of whether SCCP and MCCP/LCCP, respectively, are "toxic" to human health under Paragraph 64(c) of the *Canadian Environmental Protection Act, 1999* (CEPA 1999).

2.0 SUMMARY OF THE HUMAN HEALTH RISK ASSESSMENT FOR CHLORINATED PARAFFINS CONDUCTED UNDER CEPA 1988 (BASED UPON INFORMATION IDENTIFIED UP TO AUGUST 1992 (GOVERNMENT OF CANADA, 1993)

The PSL1 assessment addressed the short-chain ($C_{10–13}$), medium-chain ($C_{14–17}$) and long-chain ($C_{18–28}$) chlorinated paraffins. At the time of release of the PSL1 assessment, SCCP, MCCP and LCCP were produced in, and imported into, Canada for use as plasticizers and flame retardants, as well as extreme-pressure additives in lubrication oils. Quantitative data on amounts produced or used were not reported.

Relevant data were insufficient to derive quantitative estimates of exposure to chlorinated paraffins for the general population in Canada. Identified information was limited principally to the lack of detection of chlorinated paraffins in edible shellfish in a survey in Atlantic Canada (Environment Canada, 1989) and levels in a limited range of foodstuffs in the United Kingdom for which identified information was insufficient to permit evaluation of the validity of the results (Campbell and McConnell, 1980a). Environmental fate modelling (e.g., fugacity model;

[1] See "Introduction to Assessment Reports for Reconsideration of PSL1 Substances for Which Data Were Insufficient to Conclude Whether the Substances Were 'Toxic' to Human Health (Paragraph 11(c), CEPA 1988; Paragraph 64(c), CEPA 1999)" at the following web site: <http://www.hc-sc.gc.ca/hecs-sesc/exsd/psl1.htm>.

Mackay *et al*., 1985) was considered unsuitable for predicting levels in the Canadian environment, due to the paucity of information on transformation and release rates, complexity of composition (chlorinated paraffins are mixtures of compounds of varying chain lengths) and very high octanol/water partition coefficients (log K_{ow}).

Epidemiological studies of populations exposed to chlorinated paraffins were not available, and data on effects in humans were restricted to poorly documented clinical studies of the potential to induce irritation or sensitization of the skin following dermal application (Dover Chemical Corporation, 1975; Howard *et al*., 1975; English *et al*., 1986).

In a well-documented carcinogenesis bioassay for which there were some, but not critical, limitations, there was clear evidence of the carcinogenicity of SCCP (C_{12}, 60% chlorine content) in B6C3F1 mice and F344/N rats. Based on these considerations, the SCCP were considered to be probably carcinogenic to humans. Moreover, available data were insufficient to support a mode of induction of tumours other than through direct interaction with genetic material. As a result, **short-chain chlorinated paraffins were considered to be "toxic" under Paragraph 11(c) of CEPA 1988.** It was not possible to estimate exposure of the general population in Canada to SCCP. Hence, it was not possible to compare quantitative estimates of cancer potency with estimated exposure, as a basis for providing guidance on the priority for investigating options to reduce exposure, as part of the risk management strategy.

Data were adequate to derive a Tolerable Daily Intake (TDI) for MCCP and LCCP (see below). However, since it was not possible to quantitatively estimate exposure of the general population in Canada to either group of substances, the calculated TDIs could not be compared with estimated daily intake of these compounds. Based primarily on limitations of information to serve as a basis for estimation of exposure, therefore, **available data were considered inadequate to determine whether medium-chain or long-chain chlorinated paraffins were "toxic" to human health as defined in Paragraph 11(c) of CEPA 1988.**

2.1 Short-chain chlorinated paraffins

In the well-documented bioassay in which there was clear evidence of the carcinogenicity of SCCP (C_{12}, 60% chlorine) in B6C3F1 mice and F344/N rats, it was further specified that the maximum tolerated dose may have been exceeded in male and female rats (NTP, 1986a; Bucher *et al*., 1987). In the PSL1 Assessment Report, it was noted, however, that increases in tumour incidence were observed in rats in the absence of histopathological damage in at least one organ (i.e., the thyroid). Moreover, most of the mortality in exposed male rats occurred after 80 weeks, whereas overall survival in exposed female rats was reasonable compared with that in vehicle controls.

At the time of release of the PSL1 assessment, data available on the mode of induction of tumours were restricted to the results of two studies (one for which the published account was an abstract), which indicated that SCCP may act as peroxisome proliferators in the induction of liver adenomas in rats, based upon their lack of effect on unscheduled DNA synthesis but their

positive response on cell proliferation following exposure of rats to single doses of a SCCP up to 2000 mg/kg-bw (Elcombe *et al*., 1989; Ashby *et al*., 1990). In addition to the very limited data available on mode of induction of the observed tumours, the pattern of tumour development in the National Toxicology Program (NTP) bioassay for SCCP was dissimilar to that of identified epigenetic carcinogens, in that tumours were observed at multiple sites and sometimes in the absence of tissue damage. In addition, SCCP were clastogenic and induced cell transformation in *in vitro* studies, although they had not been clastogenic or mutagenic in a limited number of *in vivo* assays.

2.2 Medium-chain chlorinated paraffins

At the time of release of the PSL1 assessment, information was not identified on the chronic toxicity or carcinogenicity of MCCP in studies in experimental animals. MCCP were not mutagenic in *in vitro* assays with or without metabolic activation and were negative in an *in vitro* assay for cell transformation (Birtley *et al*., 1980). In an inadequately reported *in vivo* study, oral administration of MCCP to rats did not increase the frequency of chromosomal aberrations in bone marrow (Serrone *et al*., 1987).

The lowest effect level in the longer-term studies of the effects of MCCP identified in the PSL1 report was that in a reproductive bioassay in which rats were exposed to one of three doses of a $C_{14–17}$ (52% chlorine) chlorinated paraffin in the diet for 28 days before mating, during mating and, for females, continuously up to postnatal day 21. Pups were also exposed from weaning to 70 days of age (IRDC, 1985). At 100 ppm in the diet (5.7 mg/kg-bw per day in males and 7.2 mg/kg-bw per day in females), there was a decrease in body weight gain in exposed pups by day 21 of lactation, an effect that continued after weaning but became less pronounced in males. Histopathological effects were not observed at this concentration. These effects appeared to be attributable to lactational rather than to *in utero* exposure.

The lowest reported effect levels in subchronic studies identified in the PSL1 report were similar to those observed in the reproductive study. In three subchronic studies in which MCCP were administered in the diet to rats and dogs (Birtley *et al*., 1980; Serrone *et al*., 1987), the No-Observed-(Adverse-)Effect Levels (NO(A)ELs) ranged from 10 to 13 mg/kg-bw per day; effects observed at the next highest doses included increases in organ weights (liver and kidney), in serum hepatic enzymes and in the smooth endoplasmic reticulum of the hepatocytes.

A TDI (i.e., the level of intake to which it is believed that a person may be exposed daily over a lifetime without deleterious effects) of 6 µg/kg-bw per day was derived, therefore, for non-neoplastic effects. This value was based on the lowest dose of MCCP (Lowest-Observed-Effect Level [LOEL] = 5.7 mg/kg-bw per day) at which adverse effects (decrease in body weight gain in male rats by day 21 of lactation, which continued after weaning) were observed, in a reproductive animal study in which an adequate range of endpoints had been examined (IRDC, 1985), divided by an uncertainty factor of 1000 (×10 for intraspecies variation; ×10 for interspecies variation; ×10 for lack of data on carcinogenicity and less than chronic study). No uncertainty factor was incorporated for use of a LOEL rather than a NO(A)EL owing to the

minor nature of the effects observed at this concentration.

2.3 Long-chain chlorinated paraffins

Although the available information at the time of release of the PSL1 report was inadequate to assess the carcinogenicity of LCCP in humans, in a well-documented carcinogenesis bioassay in rats and mice, there was no evidence of carcinogenicity for male F344/N rats, equivocal evidence of carcinogenicity for female F344/N rats and female B6C3F1 mice and clear evidence of carcinogenicity for male B6C3F1 mice (NTP, 1986b). For female mice, 60–70% of the early deaths in each group were attributed to utero-ovarian infection, and it was noted that this may have decreased the sensitivity of the study to detect a carcinogenic effect. LCCP were not mutagenic in bacterial assays with or without metabolic activation (Birtley *et al*., 1980; NTP, 1986b). They were negative in an *in vitro* assay for cell transformation (ICI, 1982). In an *in vivo* study, the complete report of which was not available, LCCP did not increase the frequency of chromosomal aberrations in bone marrow cells of rats (Serrone *et al*., 1987).

The lowest dose at which non-neoplastic effects were observed in the longest-term bioassay following exposure to LCCP identified in the PSL1 report was 100 mg/kg-bw per day (NTP, 1986b; Bucher *et al*., 1987). At this dose, there was a diffuse lymphohistiocytic inflammation in the liver and in the pancreatic and mesenteric lymph nodes in female rats. Splenic congestion was a secondary effect. In subchronic studies, the lowest reported effect level was 100 mg/kg-bw per day, which induced increases in liver weight and multifocal granulomatous hepatitis (characterized by inflammatory changes) and necrosis in female rats (Serrone *et al*., 1987).

A TDI (i.e., the level of intake to which it is believed that a person may be exposed daily over a lifetime without deleterious effects) of 71 µg/kg-bw per day was derived, therefore, for non-neoplastic effects. This value was based on the lowest dose of LCCP (Lowest-Observed-Adverse-Effect Level [LOAEL] = 100 mg/kg-bw per day) at which adverse effects (diffuse lymphohistiocytic inflammation in the liver and in the pancreatic and mesenteric lymph nodes in female rats) were observed, in a carcinogenicity bioassay in which an adequate range of endpoints had been examined (NTP, 1986b; Bucher *et al*., 1987), divided by an uncertainty factor of 1000 (×10 for intraspecies variation; ×10 for interspecies variation; ×10 for use of a LOAEL rather than a NOAEL) and multiplied by 5/7 (for conversion of 5 days/week administration to daily exposure). An additional factor for limited evidence of carcinogenicity was not incorporated, since there was no increase in tumour incidence in female rats in the target organ for the non-neoplastic effect upon which the LOAEL is based.

3.0 POST-PSL1 ANALYSIS (BASED UPON INFORMATION IDENTIFIED BETWEEN AUGUST 1992 AND DECEMBER 2000 (MCCP/LCCP) OR FEBRUARY 2001 (SCCP)

3.1 Production, importation, use and release

Canadian producers of SCCP have not been identified. Most SCCP used in Canada are imported from the United States (Camford Information Services, 2001).

PCI Canada (formerly ICI Canada), Cornwall, Ontario, produces MCCP and LCCP with a chlorine content of up to 56% (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 were 2.0, 2.0, 1.7 and 1.8 kilotonnes. Chlorinated paraffins are used primarily as plasticizers and high-pressure lubricant additives.

3.2 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 SCCP 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 SCCP, MCCP and LCCP 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 1, 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.

3.2.1 Short-chain chlorinated paraffins

Tomy (1997) determined SCCP ($C_{10–13}$, 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 SCCP 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 SCCP ($C_{10–13}$, 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 SCCP ($C_{10–13}$) 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 SCCP 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 SCCP in biota in Sweden, including fish and both terrestrial and marine mammals. Analysis was by GC/MS.

Data on concentrations of SCCP in drinking water in Canada or elsewhere were not identified. The maximum concentration of SCCP ($C_{10–13}$, 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

[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.

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

HRGC/ECNI-HRMS. A lower concentration was reported in surface water from Lake Ontario (Muir *et al*., 2001).

Concentrations of SCCP 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 SCCP for the general Canadian population and the assumptions upon which they are based are presented in Table 2. 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]).

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 SCCP 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 SCCP 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 SCCP in blubber from marine mammals in Canada (Tomy *et al*. 2000) and less specific data (including both SCCP and MCCP) 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 (see supporting documentation).

[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).

3.2.2 Medium-chain chlorinated paraffins

MCCP 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). MCCP 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, MCCP 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 MCCP and the assumptions on which they are based are presented in Table 3. 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.

3.2.3 Long-chain chlorinated paraffins

Upper-bounding estimates of total intake of LCCP and associated assumptions are presented in Table 4. As for SCCP and MCCP, 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).

3.3 Hazard characterization and dose–response analyses

A limited number of studies on the toxicity of SCCP 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 MCCP or LCCP 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 MCCP and LCCP presented here reflect primarily those developed in the PSL1 Assessment Report released under CEPA 1988.

3.3.1 Short-chain chlorinated paraffins

3.3.1.1 Liver

Increased liver weight, hepatocellular hypertrophy, peroxisomal proliferation and increased S-phase activity in hepatocytes were reported in Fischer 344 rats administered SCCP 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 SCCP (Chlorowax 500C: $C_{10–13}$, 58% chlorine; or Cereclor 56L, $C_{10–13}$: 56% chlorine). For the 58% chlorine SCCP, 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 SCCP, 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 SCCP, 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 SCCP, 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 SCCP inhibited gap junction intercellular communication in rat liver cells (Kato and Kenne, 1996; Warngard *et al*., 1996).

3.3.1.2 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 SCCP/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 SCCP/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 SCCP (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.

3.3.1.3 Thyroid

Elcombe *et al*. (1994) reported that exposure of rats to SCCP 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 SCCP (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 SCCP. 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.

3.3.2 Medium-chain chlorinated paraffins

A subchronic dietary study with MCCP 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, 1993). 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.

3.3.3 Long-chain chlorinated paraffins

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

3.4 Human health risk characterization and conclusions

3.4.1 Short-chain chlorinated paraffins

3.4.1.1 Hazard characterization

Genotoxicity

Requisite criteria for assessing the weight of evidence for hypothesized modes of induction of tumours addressed below include the criterion that SCCP 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 SCCP 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, SCCP are not mutagenic" (European Commission, 2000).

Liver

It has been hypothesized that SCCP 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, 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 SCCP-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

[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.

considerably more sensitive. This pattern of induction of liver tumours by SCCP 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 SCCP 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 SCCP, 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 SCCP 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 SCCP. 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 SCCP. 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 SCCP 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 SCCP 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 SCCP 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.

3.4.1.2 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 SCCP, 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 SCCP 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, 1984). 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 SCCP, 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 SCCP (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 SCCP and the Tumorigenic Dose (TD_{05}) (i.e., 440) is also considered inadequate in view of the uncertainty concerning mode of induction of tumours.

Therefore, it is proposed that there is no reason to revise the conclusion for PSL1 that short-chain chlorinated paraffins are "toxic" as defined previously under Paragraph 11© of the *Canadian Environmental Protection Act, 1988* and currently under Paragraph 64(c) of the *Canadian Environmental Protection Act, 1999*.

3.4.2 Medium-chain chlorinated paraffins

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 MCCP 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 MCCP (i.e., 25.5 µg/kg-bw per day) exceeds the TDI by up to 4-fold.

Based on the limited available data, therefore, there is reason to suspect that medium-chain chlorinated paraffins are "toxic" to human health, as defined in Paragraph 64(c) of the *Canadian Environmental Protection Act, 1999*.

3.4.3 Long-chain chlorinated paraffins

None of the highly uncertain bounding estimates of total daily intake of LCCP 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 LCCP (16.8 µg/kg-bw per day) is within the same order of magnitude as the TDI.

Based on the limited available data, therefore, there is reason to suspect that long-chain chlorinated paraffins are "toxic" to human health, as defined in Paragraph 64(c) of the *Canadian Environmental Protection Act, 1999.*

3.5 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 SCCP and MCCP, and, as a result, intake of the individual groups of chlorinated paraffins (SCCP, MCCP and LCCP) from these sources has been overestimated.

The estimates of intake for SCCP 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 SCCP 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 SCCP, 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 SCCP 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 MCCP. 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 LCCP. 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 SCCP, 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 MCCP is based, for which studies on chronic toxicity or carcinogenicity are lacking. The database for LCCP is more complete, including a well-documented carcinogenicity bioassay in rats and mice.

3.6 Considerations for follow-up

Acquisition of higher-confidence data on levels of, particularly, MCCP and LCCP in environmental media to which the general population is exposed, particularly foodstuffs, is desirable. Since, on the basis of limited available data, there is reason to suspect that MCCP and LCCP are toxic, additional information is being requested as a basis for concluding whether the compounds can be considered to be "toxic" under CEPA 1999. If no relevant information is received, it is proposed that the Ministers of the Environment and of Health consider the substances to be "toxic" under CEPA 1999.

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Table 1. 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. $C_{10–20}$ (SCCP and MCCP) (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 $C_{10–20}$ (SCCP and MCCP) (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. $C_{10–20}$ (SCCP and MCCP) (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. $C_{10–20}$ (SCCP and MCCP) (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	SCCP 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)
	SCCP/MCCP 0.05 µg/g detection limit in analysis of 1 sample of corn flakes in U.K. $C_{10–20}$ (SCCP and MCCP) (Campbell and McConnell, 1980b)	
Meat and poultry	0.099 µg/g 1 sample of bacon in U.K. $C_{10–20}$ (SCCP and MCCP)	0.05 µg/g detection limit in analysis of 1 sample each of ox liver and beef in U.K.

Food group	Concentration used to represent food group	
	Short- and medium-chain chlorinated paraffins	**Long-chain chlorinated paraffins**
	(Campbell and McConnell, 1980b)	$C_{20–30}$ (Campbell and McConnell, 1980b)
Fish	Note: Campbell and McConnell (1980b) presented data for combined SCCP and MCCP. 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
	SCCP 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)	
	MCCP 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. $C_{10–20}$ (SCCP and MCCP) (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

Food group	Concentration used to represent food group	
	Short- and medium-chain chlorinated paraffins	**Long-chain chlorinated paraffins**
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. $C_{20–30}$ (Campbell and McConnell, 1980b)

Table 2. 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[15]	**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 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).

[15] Medium-specific and total intakes were calculated on a Microsoft Excel spreadsheet. Only significant numbers have been presented, which accounts for seemingly inaccurate totals.

Table 3. 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[14]	**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)

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

[13] 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).

[14] Medium-specific and total intakes were calculated on a Microsoft Excel spreadsheet. Only significant numbers have been presented, which accounts for seemingly inaccurate totals.

Table 4. 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–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]		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[14]	**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)

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

13 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).

[14] Medium-specific and total intakes were calculated on a Microsoft Excel spreadsheet. Only significant numbers have been presented, which accounts for seemingly inaccurate totals.

APPENDIX A: SEARCH STRATEGY — NEW INFORMATION FOR THE ASSESSMENT OF "TOXIC" TO HUMAN HEALTH UNDER PARAGRAPH 64(C) OF CEPA 1999

A comprehensive literature search was conducted (SCCP, up to February 2001; MCCP and LCCP, up to September 2000) of monitoring data in Canada (or elsewhere) and toxicological studies in animals and humans to identify critical new data for the assessment of human health risk under Paragraph 64(c) of CEPA 1999. To identify critical new exposure and toxicological data, a search was conducted in the following databases: CCRIS (Chemical Carcinogenesis Research Information System, U.S. National Library of Medicine), Current Contents (Institute for Scientific Information), DART (Development and Reproductive Toxicology, Environmental Teratology Information Centre), GENE-TOX (Genetic Toxicology, Office of Toxic Substances, U.S. Environmental Protection Agency), HSDB (Hazardous Substances Data Bank, U.S. National Library of Medicine), IRIS (Integrated Risk Information System, U.S. Environmental Protection Agency), Medline (U.S. National Library of Medicine; 1994–2000), Toxline (U.S. National Library of Medicine; 1994–2000) and Toxline Plus — including BIOSIS (Biological Abstracts), CA (Chemical Abstracts, Chemical Abstracts Service), CIS (CIS Abstracts, International Labour Office), CRISP (Computer Retrieval of Information on Scientific Projects, National Institutes of Health), DART, EPIDEM (Epidemiology Information System, Toxicology Information Response Centre), FEDRIP (Federal Research in Progress, National Technical Information Service), HMTC (HMTC Abstracts Bulletin, Hazardous Material Technical Centre), IPA (International Pharmaceutical Abstracts, American Society of Hospital Pharmacists), NTIS (Government Reports Announcements and Index, National Technical Information Service), PESTAB (Pesticide Abstracts, U.S. Environmental Protection Agency), PPBIB (Poisonous Plants Bibliography), RISKLINE (Swedish National Chemicals Inspectorate), TOXBIB (Medline, National Library of Medicine) and TSCATS (Toxic Substances Control Act Test Submissions to U.S. Environmental Protection Agency). A search of the following web sites was also conducted (up to December 2000): Centers for Disease Control and Prevention (U.S. Department of Health and Human Services), Health Canada, National Pollutant Release Inventory, U.S. Consumer Product Safety Commission, U.S. Environmental Protection Agency and U.S. Food and Drug Administration.