Ecosystem Health

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Canadian Water Quality Guidelines for the Protection of Aquatic Life: Inorganic Mercury and Methylmercury

Report No. 1-7

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NOTE TO READERS

The *Ecosystem Health: Science-based Solutions* series is dedicated to the dissemination of scientific knowledge, information and tools for monitoring, assessing, and reporting on ecosystem health to support Canadians in making sound decisions. Documents published in this series include the scientific basis, methods, approaches and frameworks for environmental guidelines and their implementation; monitoring, assessing, and rehabilitating environmental quality in Canada; and, indicator development, environmental reporting and data management. Issues in this series are published *ad libitum*.

This particular issue provides the scientific supporting information and rationale for the development of Canadian Water Quality Guidelines for the protection of aquatic life for inorganic and methylmercury. The information in this document is current as of 2002, when the document was originally prepared. Minor revisions and editorial changes have been made for publication in 2003. For additional information regarding these guidelines, please contact:

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Canadian Water Quality Guidelines are approved by the Water Quality Task Group of the Canadian Council of Ministers of the Environment (CCME). Environment Canada acts as the federal representative and serves as the technical secretariat to this Task Group. These guidelines are included as a 2003 update to the *Canadian Environmental Quality Guidelines*, which was published by the CCME in October of 1999. For CCME publications, please contact:

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This scientific supporting document is available in English only. A factsheet is also available in English under the title *Canadian Environmental Quality Guidelines* (CCME 1999). Ce document scientifique du soutien n'est disponible qu'en anglais avec un résumé en français. Un feuillet d'information est aussi disponible en français sous le titre *Recommandations canadiennes pour la qualité de l'environnement* (CCME 1999).

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ABSTRACT

Mercury (Hg) is one of the most toxic metals found in the environment. It occurs naturally, but significant amounts (approximately 50%) enter ecosystems through anthropogenic emissions and discharges. The monomethylated form of mercury (CH_3Hg^+, commonly referred to as methylmercury and abbreviated as MeHg) is often at least ten times more toxic to aquatic biota than inorganic forms (e.g., $HgCl_2$). Methylmercury is of special concern not only because of its greater toxicity, but also because it biomagnifies in upper trophic levels of aquatic food webs. Total mercury (THg) refers to the total concentration of all mercury species (i.e., both inorganic and organic forms).

This report is divided into two major parts. Part I summarises and provides a rationale for the recommended Canadian Water Quality Guidelines (CWQGs) for inorganic Hg and MeHg in fresh and marine waters. It addresses the link between the CWQGs and the recently developed Canadian Tissue Residue Guideline (TRG) for methylmercury. Gaps in the toxicological data set are identified, where applicable. Part I describes also existing water quality guidelines from other jurisdictions that are designed to protect aquatic biota or wildlife (Provincial, United States and Europe). Part II provides background information for inorganic Hg and MeHg including physical and chemical properties, sources and uses, environmental concentrations, forms and fate, bioaccumulation potential and toxicity to aquatic biota. Some chapters in Part II of this report are abbreviated and cross-referenced where there is overlap with information in the TRG for methylmercury (Environment Canada 2002).

The CWQGs tabulated below were derived according to the nationally approved protocol (CCME 1991). They are based on high quality laboratory studies that examined the toxicity of mercury to aquatic biota through direct exposure to water. The derivation and rationale for these guidelines are fully described in Part I, while a full description of the relevant toxicity studies may be found in Part II, Chapter 7. **To attain the highest degree of environmental protection, all Canadian Environmental Quality Guidelines for mercury (water, sediment, tissue, and soil) should be applied concurrently.**

Canadian Water Quality Guidelines for the Protection of Aquatic Life

Aquatic Life	Guideline (ng Hg/L)	Status
Freshwater		
Inorganic mercury	26	Full
Methylmercury	4*	Interim
Marine		
Inorganic mercury	16	Interim
Methylmercury	NRG	NA

NRG = no recommended guideline; NA = not applicable

*May not protect fully high trophic level aquatic organisms. May not prevent accumulation of MeHg in aquatic organisms; therefore, through this process the TRG for the protection of wildlife consumers of aquatic biota (33 µg/kg ww) may be exceeded. Please see text for application guidance.

The water quality guideline for inorganic Hg recommended to protect freshwater biota was derived by applying a safety factor of 0.1 to the most sensitive lowest-observable-adverse effect-level (LOAEL) of 0.26 µg Hg/L. This concentration of inorganic Hg reduced spawning and egg production for fathead minnows (*Pimephales promelas*) (Snarski and Olson 1982a). The resulting WQG is 0.026 µg Hg/L or 26 ng Hg/L.

The interim freshwater guideline for MeHg was derived by applying a safety factor of 0.1 to the LOAEL of 0.04 µg Hg/L which caused reduced offspring production in the invertebrate, *Daphnia magna* (Biesinger et al. 1982). The resulting interim WQG is 0.004 µg Hg/L or 4 ng Hg/L.

The interim marine guideline for inorganic Hg is based on a study in which exposure to 0.16 µg Hg/L (as $HgCl_2$) for 72-h reduced the growth of a population of the coccolithophore algae, *Emiliania huxleyi*, by 50% (Fisher et al. 1984). A safety factor of 0.1 was applied to the LOAEL of 0.16 µg Hg/L to give an interim WQG of 0.016 µg Hg/L or 16 ng Hg/L. Insufficient data are available to recommend a marine guideline for MeHg.

The protocol for the derivation of CWQGs does not address exposure through food or bioaccumulation to higher trophic levels. As such, aquatic life that are exposed to methylmercury primarily through food (e.g., piscivorous fish) may not be adequately protected. Moreover, these CWQGs for mercury may not prevent the accumulation of methylmercury in aquatic life; therefore, through this process the tissue residue guideline (TRG; 33 µg MeHg/kg ww) for the protection of wildlife that consume aquatic life may be exceeded. Thus, if the ultimate management objective for mercury is to protect high trophic level aquatic life and/or those wildlife that prey on aquatic life, more stringent site-specific application of these water quality guidelines may be necessary.

For example, the issue that faces many environmental managers is ensuring the protection of wildlife consumers of mercury-ladened fish. Calculations using reference concentrations[1] of MeHg for wildlife species and field-based bioaccumulation factors (BAFs) produced estimates of water concentrations that could protect wildlife that consume aquatic biota. These generic calculations are intended as a guide to determining site- and species-specific water quality objectives. From conservative assumptions, concentrations of MeHg below 0.007 ng Hg/L may be required to protect all wildlife species in Canada while concentrations above 0.2 ng Hg/L (0.0002 µg Hg/L) may pose a risk to wildlife species. MeHg concentrations in water between these limits may be hazardous to some wildlife depending on their feeding habits (preferred prey items, and the trophic level and BAFs of these prey items). More specific information is given in Part I, Section 4.2.

While mercury occurs naturally, humans effectively cause accelerated emissions of mercury through direct mining of Hg ore and use in a variety of products. Mercury is used in dental

[1] A reference concentration is the concentration of MeHg in tissues of aquatic biota below which adverse effects are not expected for a given species of wildlife that consume aquatic biota. Reference concentrations for MeHg were derived for a suite of Canadian wildlife species; the lowest of which, that for Wilson's storm petrel, *Oceanites oceanicus* (33 µg/kg diet ww), was selected as the Canadian Tissue Residue Guideline (Environment Canada 2002).

amalgams, exterior paints, thermometers, barometers, and electrical products such as dry-cell batteries, fluorescent lights, switches, and other control equipment. It is also used in substantial quantities in the electrolytic preparation of chlorine and caustic soda (chlor-alkali industry). Historically, tailings produced from amalgamating gold mines contained unknown and variable quantities of mercury, but none of the current gold mining operations in Canada practice mercury amalgamation.

Mercury can be carried in the atmosphere for thousands of kilometres and be deposited with wet and dry fallout directly on surface waters or watersheds. The major sources of anthropogenic Hg to the Canadian environment include metal smelting, municipal and medical waste, and sewage sludge incineration, fossil-fuel burning (including coal for electric power generation) and cement manufacturing (1995 estimates) (Pilgrim and Ecological Monitoring and Assessment Network 1998). Natural sources include forest fires and other wood burning, volcanoes and a portion of oceanic emissions[2]. Runoff and flow-through from terrestrial areas may transport the various forms of mercury to surface waters containing fish and other aquatic biota. While fallout consists normally of inorganic forms of Hg, the more toxic methylmercury may be produced in receiving environments under conditions where bacterial activity is enhanced such as highly organic surface sediments and in wetlands.

The net production of MeHg is a balance between methylation and demethylation; both may occur through abiotic and biotic (microbial) processes (Weber 1993). Several factors influence the rate of net MeHg production in water including the concentration and availability of Hg^{2+}, composition of the microbial population, nutrient and mineral substrate, pH, temperature, redox potential, dissolved and particulate organic matter (DOC/DOM and POM), iron and sulphate. Because bacterial activity increases with increasing temperature and available biodegradable organic carbon, methylation rates tend to be highest in surface sediments with freshly deposited organic matter, and in warm shallow sediments where abundant bacterial activity takes place (Ramlal et al. 1986; Winfrey and Rudd 1990). Methylmercury formation is reduced with increasing salinity in estuarine sediments (Blum and Bartha 1980).

The collection of environmental samples and subsequent analytical procedures have been refined in the past decade to include ultra-clean sampling and analysis techniques. Samples of rainwater taken in the early 1990s at the Experimental Lakes Area of north-western Ontario averaged from 3.8 to 5.3 ng THg/L and from 0.017 to 0.049 ng MeHg/L (<1% of THg). In snow samples, concentrations of THg averaged 0.95 to 3.58 ng Hg/L, while concentrations of MeHg ranged from 0.008 to 0.104 ng Hg/L, representing 0.8% to 2.9% of the THg (St.Louis et al. 1994; 1995a). The concentration of MeHg represents typically less than 10% of that for THg in surface waters, but it can exceed 30% in perturbed systems such as newly formed reservoirs. For, example, in an experimental reservoir, the average post-flood proportion of MeHg (0.05 - 3.2 µg Hg/L) was 32% of THg (0.98 - 6.95 µg Hg/L) which is about seven times higher than pre-flood. The maximum proportion of MeHg in water from the reservoir was 73% of THg (Kelly et al. 1997). A pristine lake at the Experimental Lakes Area had concentrations of 0.6 - 3.5 ng THg/L, about 6% of which was MeHg (0.004 - 0.22 ng Hg/L) (Kelly et al. 1997).

[2] The portion of oceanic emissions that are natural, not re-emitted anthropogenic Hg.

Concentrations of mercury in sediments, aquatic plants, invertebrates and fish are described in Part II and are tabulated in the TRG for MeHg (Environment Canada 2002). Very generally, the concentration of total Hg increases with trophic level and the proportion of that which is MeHg increases with trophic level as well. In aquatic plants, MeHg accounts usually for less than 50% of the THg, while in aquatic invertebrates MeHg is about 50% of THg although a greater variability in percent MeHg occurs in marine invertebrates than in most freshwater invertebrates. Total mercury concentrations in fish increase with age, and piscivorous fish have higher total mercury burdens than non-piscivorous fish. Essentially 100% of the mercury found in the muscle tissues of piscivorous fish is MeHg (Bloom 1992). Somewhat lower percentages of MeHg may be expected in young non-piscivorous fish.

Aquatic organisms take up mercury compounds directly from water or through their diet. Mercury dissolved in water is taken up from adsorption or absorption through the body surface and respiratory organs. Uptake via food is based on mercury absorption across the digestive tract. Dietary uptake accounts for the majority of MeHg found in organisms at higher trophic levels. Once taken up, MeHg accumulates in organic tissue because of its affinity for sulphydryl groups, the relative ease with which it passes through the digestive wall, and slower depuration rate relative to inorganic mercury (Saouter et al. 1993). MeHg is assimilated very efficiently but inorganic Hg is not.

Fish from reservoirs may retain elevated mercury concentrations anywhere from a few years to decades because the newly flooded organic matter and associated increased bacterial activity enhances MeHg production in the reservoirs themselves (Bodaly et al. 1984; Verdon et al. 1991). In addition, low pH (<6), low alkalinity (acid-neutralising capacity 50 µeq/L or less), and low calcium (<5 mg/L) lakes are associated with elevated mercury concentrations in fish (Grieb et al. 1990; Spry and Wiener 1991).

The sensitivity of aquatic organisms to either inorganic Hg or MeHg varies considerably between species, although MeHg is the more toxic form. In fresh waters, acute (24- to 96-h LC_{50}) toxicity concentrations for inorganic Hg range from 5 to 5600 µg Hg/L in invertebrates and from 150 to 900 µg Hg/L in fish (Biesinger and Christensen 1972; Rehwoldt et al. 1973; Call et al. 1983; Wobeser 1975). In contrast, acute toxicity concentrations for MeHg range from 24 to 125 µg Hg/L in fish (no invertebrate data available) (Wobeser 1975). From limited acute data, algae appear sensitive with 24-h LC_{50}s from 9 to 27 µg Hg/L for inorganic Hg and from 3.5 to 6.3 µg Hg/L for MeHg (Chen and Lin 1997; Thomas and Montes 1978).

In chronic tests, invertebrates are about as sensitive to mercury as fish. Effect concentrations (EC_{50}s) in invertebrates range from 1.28 to 12.0 µg Hg/L for inorganic Hg and from 0.04 to 1.14 µg Hg/L for MeHg (Biesinger et al. 1982; Spehar and Fiandt 1986). In fish, chronic values for inorganic Hg range from 0.26 to >64 µg Hg/L in 5- to 60-d tests (Niimi and Kissoon 1994h; Snarski and Olson 1982a); effects range from weight loss to death. Amphibians are sensitive also to inorganic Hg with 5- to 21-day LC_{50}s ranging from 1.3 to 67.2 µg Hg/L (Birge et al. 1979). A single chronic test with MeHg reported an EC_{50} for teratogenesis in leopard frogs (*Rana pipiens*) of 5 µg Hg/L (Dial 1976). Adverse effects reported commonly include growth, impaired reproduction and development, and death. Inorganic Hg (as $HgCl_2$) and MeHg (as MeHgCl) were described by Wobeser (1975) to cause flaring of the opercula and increased frequency and

force of respiratory movements of rainbow trout (*Oncorhynchus mykiss*) fry and fingerlings. Higher concentrations caused loss of equilibrium, sluggishness and a tendency to lie on the bottom of the tank. Prolonged exposure caused death. In side-by-side tests, MeHgCl is typically more than ten times as toxic as $HgCl_2$ to fish, invertebrates, and aquatic plants (Niimi and Kissoon 1994h; Biesinger et al. 1982; Thomas and Montes 1978).

Data for marine species are much more limited but trends are similar to those observed for freshwater species. A single acute (96-h) study on the effects of inorganic Hg on fish reported a LC_{50} of 68 µg Hg/L (Sharp and Neff 1982). For invertebrates, 24 to 96-h LC_{50} range from 3.5 to 161 µg Hg/L (Lussier et al. 1985; Nelson et al. 1988). For MeHg, acute (96-h LC_{50}) values range from 50 to 200 µg Hg/L for fish (mummichog [*Fundulus heterclitus*]) (Khan and Weis 1987; Sharp and Neff 1982). In addition, reduced fertilisation of mummichog eggs occurred when eggs were exposed to 500 µg Hg/L of MeHg for 20 minutes (Khan and Weis 1987). No studies regarding the acute effects of MeHg on marine invertebrates and plants were found.
Similar to freshwater studies, chronic studies on marine life report reduced growth and survival, and impaired development (e.g., increased incidence of deformities). EC_{50}s for inorganic Hg range from <5 to 55 µg Hg/L for fish, from 1.2 to 20 µg Hg/L for invertebrates, and from 0.16 to 1002 µg Hg/L for plants and algae (Brown and Parsons 1978; Sharp and Neff 1982; Lussier et al. 1985; Warnau et al. 1996; Fisher et al. 1984). No chronic studies on MeHg were found for marine species.

Toxicity of mercury is negatively correlated with salinity, selenium concentration, and oxygen content, and positively correlated with temperature (McKenney, Jr. and Costlow, Jr. 1981; reviewed by Cuvin-Aralar and Furness 1991; Snell et al. 1991; Heit and Fingerman 1977; MacLeod and Pessah 1973) (Slooff et al. 1991). Water hardness has a negligible effect on mercury toxicity unlike other toxic metals such as copper or aluminium, where toxicity significantly decreases with increasing hardness (Keller and Zam 1991).

RÉSUMÉ

Le mercure (Hg) est l'un des métaux les plus toxiques qui soient présents dans l'environnement. Il existe à l'état naturel, mais une proportion importante (environ 50 %) du mercure pénètre dans les écosystèmes du fait des émissions et des rejets d'origine anthropique. La forme monométhylée du mercure (CH_3Hg^+, communément appelée méthylmercure, avec l'abréviation MeHg) est souvent au moins dix fois plus toxique pour le biote aquatique que les formes inorganiques (p. ex., $HgCl_2$). Le méthylmercure est particulièrement préoccupant, non seulement à cause de sa toxicité plus grande, mais du fait de sa bioamplification aux niveaux supérieurs des réseaux trophiques aquatiques. Par mercure total (THg), on désigne la concentration totale de toutes les espèces de mercure (c.-à-d. les formes inorganiques et organiques).

Le rapport se divise en deux grandes parties. La partie I résume et justifie les seuils proposés pour les Recommandations pour la qualité des eaux au Canada (RQE) concernant le Hg inorganique et le MeHg dans les eaux douces et marines. Elle fait le lien entre les RQE et la Recommandation canadienne pour les résidus dans les tissus (RRT) récemment formulée pour le méthylmercure. On y relève, le cas échéant, les lacunes dans les séries de données toxicologiques. La partie I décrit aussi les autres directives concernant la qualité de l'eau qui existent ailleurs et qui visent à protéger le biote aquatique ou la faune et la flore sauvages (provinces, États-Unis et Europe). La partie II rassemble des données générales sur le Hg inorganique et le MeHg, notamment leurs propriétés physiques et chimiques, leurs sources et leurs utilisations, les concentrations dans l'environnement, les diverses formes et leur devenir, le potentiel de bioaccumulation et la toxicité pour le biote aquatique. Certains chapitres de la partie II sont résumés et signalent par des renvois les recoupements avec l'information présentée dans la RRT sur le méthylmercure (Environment Canada 2000).

Les RQE présentées ci-dessous ont été calculées à partir du protocole approuvé à l'échelle nationale (CCME, 1991). Elles se fondent sur des études de laboratoire très soignées qui ont examiné la toxicité du mercure pour le biote aquatique par exposition directe dans l'eau. Le mode de calcul et la justification de ces recommandations sont décrits en détail dans la partie I, et on trouvera dans la partie II, chapitre 7, une description des études toxicologiques pertinentes. **Pour arriver au degré le plus haut de protection de l'environnement, toutes les RQE sur le mercure (eau, sédiments, tissus et sol) doivent être appliquées concurremment.**

Recommandations pour la qualité de l'eau en vue de la protection de la vie aquatique

Vie aquatique	Recommandation (ng Hg/L)	Niveau
Vie dulcicole		
Mercure inorganique	26	Définitive
Méthylmercure	4*	Provisoire
Vie marine		
Mercure inorganique	16	Provisoire
Méthylmercure	AR	SO

AR = aucune recommandation; SO = sans objet

*Ne protège pas toujours entièrement les prédateurs aquatiques supérieurs. N'empêche pas toujours l'accumulation de MeHg chez les organismes aquatiques; donc, la RRT fixée pour la protection des espèces fauniques consommant le biote aquatique (33 µg/kg – poids humide) peut être dépassée. Voir le texte pour les instructions sur l'application.

Pour établir la concentration maximale de Hg inorganique recommandée pour protéger le biote aquatique, on a multiplié par un facteur de sécurité de 0,1 la valeur de la dose minimale avec effet nocif observé (DMENO), soit 0,26 µg Hg/L. Cette concentration de Hg inorganique réduisait la fraye et la production d'œufs chez la tête-de-boule (*Pimephales promelas*) (Snarski and Olson 1982a). La RQE qui en résulte est de 0,026 µg Hg/L, soit 26 ng Hg/L.

Pour établir la recommandation provisoire visant le MeHg en eau douce, on a multiplié par un facteur de sécurité de 0,1 la valeur de la DMENO, soit 0,04 µg Hg/L, qui causait une réduction de la progéniture chez l'invertébré *Daphnia magna* (Biesinger et al. 1982b). La RQE provisoire qui en découle est de 0,004 µg Hg/L, soit 4 ng Hg/L.

La RQE provisoire visant le Hg inorganique en eau de mer se base sur une étude dans laquelle l'exposition à 0,16 µg Hg/L (sous la forme $HgCl_2$) pendant 72 h a réduit de 50 % la croissance d'une population de l'algue coccolithophore *Emiliania huxleyi* (Fisher et al. 1984). On a multiplié par un facteur de sécurité de 0,1 la valeur de la DMENO, soit 0,16 µg Hg/L, pour établir une RQE provisoire de 0,016 µg Hg/L, soit 16 ng Hg/L. Les données sont insuffisantes pour permettre d'établir une recommandation visant le MeHg en eau de mer.

Le protocole d'élaboration des RQE ne vise pas l'exposition par l'intermédiaire de la nourriture ou par la bioaccumulation aux niveaux trophiques supérieurs. C'est dire que les organismes aquatiques qui sont d'abord exposés au méthylmercure par leur alimentation (p. ex. les poissons piscivores) ne sont pas toujours correctement protégés. De plus, ces RQE visant le mercure n'empêchent pas nécessairement l'accumulation du méthylmercure chez les formes de vie aquatiques, de sorte que la Recommandation pour les résidus dans les tissus (RRT) fixée pour la protection des espèces fauniques consommant le biote aquatique (33 µg/kg – poids humide) peut être dépassée. Aussi, si l'objectif ultime de gestion visant le mercure est de protéger les organismes supérieurs du réseau trophique aquatique et/ou les prédateurs des formes de vie aquatiques, il peut être nécessaire de formuler des modalités d'application plus strictes et spécifiques au site de ces RQE.

Par exemple, l'un des problèmes que doivent résoudre de nombreux gestionnaires de l'environnement est d'assurer la protection des consommateurs de poissons sauvages contaminés au mercure. Les calculs faisant appel à des concentrations de référence[3] de MeHg pour les espèces fauniques et des facteurs de bioaccumulation (FBA) obtenus sur le terrain ont donné des estimations des concentrations dans l'eau qui pourraient protéger les espèces fauniques consommant le biote aquatique. Ces calculs de base doivent guider l'établissement d'objectifs de qualité de l'eau spécifiques au site et à l'espèce. Si l'on adopte des hypothèses prudentes, il peut être nécessaire de fixer les concentrations de MeHg à moins de 0,007 ng Hg/L pour protéger toutes les espèces fauniques au Canada, alors que des concentrations supérieures à 0,2 ng Hg/L (0,0002 µg Hg/L) peuvent représenter un risque pour les espèces fauniques. Les concentrations de MeHg dans l'eau qui se situent entre ces limites peuvent présenter un danger pour certaines espèces, selon leurs habitudes alimentaires (proies favorites, ainsi que le niveau trophique et les FBA de ces proies). On trouvera des renseignements plus détaillés dans la partie I, section 4.2.

Si le mercure est présent à l'état naturel, l'activité humaine peut accélérer les émissions de ce métal par l'exploitation directe du minerai et par l'utilisation du mercure à diverses fins. Il sert en effet à fabriquer des amalgames dentaires, des peintures d'extérieur, des thermomètres, des baromètres et du matériel électrique comme les piles sèches, les lampes fluorescentes, les commutateurs et autres dispositifs de contrôle. On en emploie aussi des quantités substantielles dans la préparation électrolytique du chlore et de la soude caustique. Jusqu'ici, les stériles des mines extrayant l'or par amalgamation contenaient des quantités indéterminées et variables de mercure, mais aucune des mines d'or en exploitation au Canada ne pratique l'amalgamation au mercure.

Le mercure peut être transporté dans l'atmosphère sur des milliers de kilomètres et se déposer avec les retombées humides et sèches directement dans les eaux de surface ou les bassins hydrographiques. Les principales sources anthropiques de Hg dans l'environnement canadien sont les fonderies de métaux, les déchets municipaux et médicaux, ainsi que l'incinération des boues des stations de traitement des eaux usées, la combustion des combustibles fossiles (ce qui comprend le charbon brûlé pour produire de l'électricité) et la fabrication de ciment (estimations de 1995) (Pilgrim and Ecological Monitoring and Assessment Network 1998a). Parmi les sources

[3] Une concentration de référence (CR) est la concentration de MeHg dans les tissus du biote aquatique au-dessous de laquelle on peut s'attendre à une absence d'effets chez une espèce faunique donnée qui consomme le biote aquatique. Les CR pour le MeHg ont été calculées pour une série d'espèces fauniques canadiennes, et on a retenu la plus basse de ces CR, celle qui concerne le pétrel océanite, *Oceanites oceanicus* (33 µg/kg poids humide), comme Recommandation canadienne pour les résidus dans les tissus (Environnement Canada, 2002).

naturelles, on compte les incendies de forêt et autres formes de combustion du bois, les éruptions volcaniques et une portion des émissions des océans[4]. Le ruissellement et l'écoulement des eaux terrestres peut transporter les diverses formes de mercure vers les eaux de surface abritant des poissons et d'autres membres du biote aquatique. Les retombées sont généralement constituées de formes inorganiques de Hg, mais le méthylmercure, plus toxique, peut être produit dans les milieux récepteurs lorsque les conditions sont favorables à l'activité bactérienne, comme dans les sédiments superficiels à forte charge organique et dans les zones humides.

La production nette de MeHg est le résultat d'un équilibre entre la méthylation et la déméthylation, qui peuvent toutes deux résulter de processus abiotiques et biotiques (microbiens) (Weber 1993). Plusieurs facteurs influent sur le taux de production nette de MeHg dans l'eau : concentration et disponibilité du Hg^{2+}, composition de la population microbienne, matières nutritives et substrat minéral, pH, température, potentiel d'oxydoréduction, matière organique dissoute et particulaire (COD/MOD ET MOP), fer et sulfate. Étant donné que l'activité microbienne s'accélère avec la hausse de la température et de la disponibilité du carbone organique biodégradable, le taux de méthylation est généralement au plus haut dans les sédiments de surface composés de matière organique fraîchement déposée, et dans les sédiments peu profonds et chauds où l'activité bactérienne est intense (Ramlal et al. 1986; Winfrey and Rudd 1990). La formation du méthylmercure diminue avec l'augmentation de la salinité dans les sédiments estuariens (Blum and Bartha 1980).

Au cours de la dernière décennie, la collecte d'échantillons environnementaux et les méthodes d'analyse subséquentes se sont enrichies de techniques ultrapropres d'échantillonnage et d'analyse. Les échantillons d'eau de pluie recueillis au début des années 1990 dans la région des Lacs expérimentaux (nord-ouest de l'Ontario) présentaient des concentrations moyennes de 3,8 à 5,3 ng THg/L et de 0,017 à 0,049 ng MeHg/L (<1 % du THg). Dans les échantillons de neige, les concentrations de THg étaient en moyenne de 0,95 à 3,58 ng Hg/L, tandis que les concentrations de MeHg allaient de 0,008 à 0,104 ng Hg/L, ce qui représentait de 0,8 % à 2,9 % du THg (St.Louis et al. 1994; 1995a). La concentration de MeHg représente de façon typique moins de 10 % que celle de THg dans les eaux de surface, mais elle peut dépasser 30 % dans les systèmes perturbés comme les réservoirs nouvellement aménagés. Par exemple, dans un réservoir expérimental, la proportion moyenne de MeHg après la mise en eau (0,05 – 3,2 µg Hg/L) était de 32 % du THg (0,98 – 6,95 µg Hg/L), ce qui est environ sept fois supérieur à la proportion avant la mise en eau. La proportion maximale de MeHg dans l'eau du réservoir correspondait à 73 % du THg (Kelly et al. 1997). Dans un lac intact de la région des Lacs expérimentaux, on a noté des concentrations de 0,6 – 3,5 ng THg/L, dont environ 6 % était constitué de MeHg (0,004 – 0,22 ng Hg/L) (Kelly et al. 1997).

Les concentrations de mercure dans les sédiments, les végétaux aquatiques, les invertébrés et les poissons sont décrits dans la partie II et sont calculés dans la RRT pour le MeHg (Environment Canada 2000). De façon très générale, la concentration de Hg total augmente avec le niveau trophique, et la proportion constituée de MeHg augmente aussi avec le niveau trophique. Dans les végétaux aquatiques, le MeHg représente généralement moins de 50 % du THg, tandis que chez les invertébrés aquatiques le MeHg représente environ 50 % du THg; on note cependant une plus grande variabilité du pourcentage de MeHg chez les invertébrés marins que chez la plupart des invertébrés dulcicoles. Les concentrations totales de mercure chez les poissons augmentent avec l'âge, et les poissons piscivores présentent des charges totales en mercure supérieures à celles des poissons non piscivores. Dans l'ensemble, 100 % du mercure trouvé dans les tissus musculaires des poissons piscivores est sous la forme MeHg (Bloom 1992). On peut s'attendre à mesurer des pourcentages légèrement plus faibles chez les jeunes poissons non piscivores.

Les organismes aquatiques absorbent les composés du mercure directement dans l'eau ou dans leur nourriture. L'assimilation du mercure dissous dans l'eau résulte de l'adsorption ou de l'absorption par la surface du corps et les organes respiratoires. La voie alimentaire consiste en une absorption du mercure dans le tractus digestif, et correspond à la plus grande part du MeHg mesuré chez les organismes des niveaux trophiques supérieurs. Une fois absorbé, le MeHg s'accumule dans les tissus organiques à cause de son affinité pour les groupements sulfhydryles, de l'aisance relative avec laquelle il franchit la paroi digestive, et de son taux de dépuration plus lent que celui du mercure inorganique (Saouter et al. 1993c). Le MeHg est assimilé de façon très efficace, ce qui n'est pas le cas du Hg inorganique.

[4] La portion des émissions des océans considérée comme naturelle, c.-à-d. autre que la réémission de Hg d'origine anthropique.

Dans les réservoirs, les poissons peuvent présenter des concentrations élevées de mercure pendant longtemps (des années ou même des décennies) étant donné que la matière organique nouvellement inondée et l'accélération de l'activité bactérienne qui en résulte stimule la production de MeHg à l'intérieur des réservoirs (Bodaly et al. 1984; Verdon et al. 1991a). De plus, les lacs à faible pH (<6), à faible alcalinité (potentiel de neutralisation de l'acide de 50 µeq/L ou moins) et à faible concentration de calcium (<5 mg/L) sont associés à des concentrations élevés de mercure chez les poissons (Grieb et al. 1990; Spry and Wiener 1991c).

La sensibilité des organismes aquatiques au Hg inorganique ou au MeHg varie considérablement d'une espèce à l'autre, mais le MeHg reste la forme la plus toxique. En eau douce, les concentrations correspondant à la toxicité aiguë (CL_{50} sur 24 à 96 h) du Hg inorganique vont de 5 à 5600 µg Hg/L chez les invertébrés et de 150 à 900 µg Hg/L chez les poissons (Biesinger and Christensen 1972; Rehwoldt et al. 1973; Call et al. 1983; Wobeser 1975). Par contre, les concentrations correspondant à la toxicité aiguë du MeHg vont de 24 à 125 µg Hg/L chez des poissons (pas de données sur les invertébrés) (Wobeser 1975). À partir de données limitées sur la toxicité aiguë, les algues semblent sensibles au mercure, avec des CL_{50} sur 24 h allant de 9 à 27 µg Hg/L pour le Hg inorganique et de 3,5 à 6,3 µg Hg/L pour le MeHg (Chen and Lin 1997; Thomas and Montes 1978b).

Dans les essais de toxicité chronique, les invertébrés sont à peu près aussi sensibles au mercure que les poissons. Les concentrations effectives (CE_{50}) chez les invertébrés vont de 1,28 à 12,0 µg Hg/L pour le Hg inorganique et de 0,04 à 1,14 µg Hg/L pour le MeHg (Biesinger et al. 1982b; Spehar and Fiandt 1986; Biesinger et al. 1982b). Chez les poissons, les chiffres correspondant à la toxicité chronique du Hg inorganique vont de 0,26 à >64 µg Hg/L dans des tests de 5 à 60 jours (Niimi and Kissoon 1994h; Snarski and Olson 1982a); la gamme d'effets va de la perte de poids à la mort. Les amphibiens sont eux aussi sensibles au Hg inorganique; les CL_{50} sur 5 à 21 jours vont de 1,3 à 67,2 µg Hg/L (Birge et al. 1979c). Un seul essai de toxicité chronique du MeHg chez la grenouille léopard (*Rana pipiens*) a donné une CE_{50} pour la tératogénicité de 5 µg Hg/L (Dial 1976a). Les effets négatifs couramment rapportés sont des perturbations de la croissance, de la reproduction et du développement, et la mort. Selon Wobeser (1975), le Hg inorganique (sous la forme $HgCl_2$) et le MeHg (sous la forme MeHgCl) causaient une ouverture des opercules et une augmentation de la fréquence et de la force des mouvements respiratoires chez les alevins et les jeunes d'un an de la truite arc-en-ciel (*Oncorhynchus mykiss*). À des concentrations plus fortes, on notait une perte d'équilibre, l'atonie et une tendance à rester sur le fond de l'aquarium. La prolongation de l'exposition causait la mort. Dans des tests en parallèle, le MeHgCl se révèle typiquement au moins dix fois plus toxique que le $HgCl_2$ pour les poissons, les invertébrés et les végétaux aquatiques (Niimi and Kissoon 1994h; Biesinger et al. 1982b; Thomas and Montes 1978b).

Pour les espèces marines, les données sont beaucoup plus limitées, mais les tendances sont semblables à celles qu'on observe chez les espèces dulcicoles. Une seule étude (96 h) de toxicité aiguë concernant les effets du Hg inorganique sur les poissons signale une CL_{50} de 68 µg Hg/L (Sharp and Neff 1982). Pour les invertébrés, la CL_{50} sur 24 à 96 h va de 3,5 à 161 µg Hg/L (Lussier et al. 1985; Nelson et al. 1988). Pour le MeHg, les chiffres de toxicité aiguë (CL_{50} à 96 h) vont de 50 à 200 µg Hg/L pour les poissons (choquemort [*Fundulus heteroclitus*]) (Khan and Weis 1987; Sharp and Neff 1982). De plus, on a observé une baisse de la fécondation des œufs de choquemort quand ils étaient exposés à 500 µg Hg/L (MeHg) pendant 20 minutes (Khan and Weis 1987). Nous n'avons trouvé aucune étude concernant les effets aigus du MeHg sur les invertébrés et les végétaux marins.

Comme les travaux menés en eau douce, les études sur la toxicité chronique pour la vie marine signalent une réduction de la croissance et de la survie, et une perturbation du développement (incidence accrue de malformations). La CE_{50} pour le Hg inorganique va de <5 à 55 µg Hg/L pour les poissons, de 1,2 à 20 µg Hg/L pour les invertébrés, et de 0,16 à 1002 µg Hg/L pour les plantes vasculaires et les algues (Brown and Parsons 1978; Sharp and Neff 1982; Lussier et al. 1985; Warnau et al. 1996; Fisher et al. 1984). Nous n'avons trouvé aucune étude de toxicité chronique du MeHg pour les espèces marines.

La toxicité du mercure est corrélée négativement à la salinité, à la concentration de sélénium et à la teneur en oxygène, et positivement à la température (McKenney, Jr. and Costlow, Jr. 1981; reviewed by Cuvin-Aralar and Furness 1991; Snell et al. 1991; Heit and Fingerman 1977; MacLeod and Pessah 1973)(Slooff et al. 1991). La dureté de l'eau a un effet négligeable sur la toxicité du mercure, à la différence d'autres métaux toxiques comme le cuivre ou l'aluminium, pour lesquels la toxicité diminue de façon notable quand la dureté augmente (Keller and Zam 1991).

Part I: Canadian Water Quality Guidelines for Inorganic Mercury and Methylmercury

1.0 Guideline Summary

The following full and interim water quality guidelines (CWQGs) are recommended to protect aquatic biota from harmful exposure to mercury compounds in water. **To attain the highest degree of environmental protection, all Environmental Quality Guidelines (EQGs) for mercury (water, sediment, tissue, and soil) should be applied concurrently.**

Canadian Water Quality Guidelines for the Protection of Aquatic Life

Aquatic Life	Guideline (ng Hg/L)	Status
Freshwater		
Inorganic mercury	26	Full
Methylmercury	4*	Interim
Marine		
Inorganic mercury	120	Interim
Methylmercury	NRG	NA

NRG = no recommended guideline; NA = not applicable

*May not protect fully high trophic level aquatic organisms. May not prevent accumulation of MeHg in aquatic organisms; therefore, through this process the TRG for the protection of wildlife consumers of aquatic biota (33 µg/kg ww) may be exceeded. Please see text for application guidance.

2.0 Introduction

Environment Canada develops Canadian EQGs under the auspices on the Canadian Council of Ministers of the Environment (CCME) using formal protocols (CCME 1991; CCME 1995; CCME 1998) to provide a consistent, scientifically defensible approach for assessing and managing toxic substances in the environment. These guidelines are numerical concentrations in various media (water, sediment, aquatic biota, and soil) that are recommended to protect, enhance and restore designated uses of the environment. Guidelines derived from the full complement of required data are termed full guidelines, while those meeting lesser data requirements are termed interim; however, interim guidelines should be used in the same manner as full guidelines. These concentrations provide benchmarks for the interpretation of environmental monitoring data and serve as the scientific basis for determining elimination strategies. The degradation of the existing environmental quality should always be avoided, that is EQGs are not "pollute up to" levels. EQGs are intended for use by Canadian provincial, territorial, and federal agencies as well as private, corporate or public stakeholders to assess environmental quality problems and to manage competing uses of resources. These national numerical environmental quality guidelines are important tools in comprehensive ecosystem management but they are not intended to preclude the need for site-specific consideration and

approaches. Thus, the use of EQGs requires consideration of local conditions. For example, the natural background concentrations of parameters and their range should also be taken into account in the design of monitoring programs and the interpretation of the resultant data.

Canadian EQGs for mercury have been elaborated elsewhere. Sediment quality guidelines (SQGs) for the protection of aquatic life were published in 1997 while the tissue residue guideline (TRG) for the protection of wildlife consumers of aquatic life was published in 1999 (Environment Canada 1997, 1999). Air quality objectives, soil quality guidelines for environmental and human health, and water quality guidelines for agricultural uses exist also (CCME 1999). The CCME adopted a water quality guideline (WQG) for the protection of aquatic life in 1987 from a 1979 Canadian guideline (CCREM 1987). Since that time, concern over mercury contamination has grown with the increase in the number of commercial fisheries advisories and fish consumption restrictions at numerous locations across Canada. A wealth of new information on mercury toxicity, and environmental fate and behaviour in the aquatic environment has become available. For these reasons, the CCME Water Quality Guidelines Task Group agreed to update the WQG for mercury for the protection of aquatic life. This updated WQG is developed herein and supersedes the earlier value published in 1987 (CCREM 1987).

To develop CWQGs for mercury, a detailed technical review was carried out for inorganic mercury, methylmercury (MeHg), and total mercury (THg). Where material overlapped with the technical supporting documents for the SQGs and the TRG, descriptions in this report are abbreviated and cross-referenced with the previous reports. Sources, uses, and pathways for entering the environment are detailed; levels of mercury in abiotic (water, sediment, and soil) are documented in tables to facilitate comparisons among regions. Concentrations of inorganic mercury, methylmercury, and total mercury in this document are reported as Hg ion equivalents, unless otherwise indicated. Wherever possible, the chemical formula of the mercury compound in question is provided (e.g., $HgCl_2$ for mercuric chloride). Available data on the environmental fate and behaviour of mercuric species are evaluated and summarised. Most importantly, a comprehensive assessment of the toxicity of mercury to aquatic life (plants, invertebrates, amphibians, and fish) was undertaken to evaluate the environmental hazards posed by mercury.

In addition to detailing the CWQGs for mercury, guidance regarding the application of these guidelines is provided. First, implementation of Canadian EQGs is discussed in general, and then specific recommendations are given for applying the national CWQGs and developing site-specific water quality objectives (WQOs) for mercury. Factors discussed are: filtered versus unfiltered water samples; bioaccumulation of MeHg; and coherence with the Canadian TRG for MeHg.

3.0 Water Quality Guidelines for Inorganic Mercury and Methylmercury

3.1 Freshwater Life

3.1.1 Full and Interim Guidelines

A water quality guideline of 26 ng Hg/L is recommended for the protection of all stages of freshwater life against the adverse affects of inorganic mercury in water. An interim water quality guideline of 4 ng/L is recommended for the protection of low trophic level freshwater life (i.e., generally trophic levels 1-2) against the adverse affects of direct exposure to methylmercury through water. **This later guideline may not protect high trophic level aquatic life (i.e., generally trophic levels 3-4) which are exposed to methylmercury primarily through food.** Nor may it prevent the accumulation of methylmercury in aquatic life which could cause the tissue residue guideline (33 ng/kg diet ww) for the protection of wildlife consumers of aquatic biota to be exceeded. Both freshwater guidelines apply to unfiltered water samples.

3.1.2 Summary of Existing Water Quality Guidelines to Protect Aquatic Life

The Canadian water quality guideline for mercury to protect aquatic life has been 100 ng THg/L since 1979 (Reeder et al. 1979; Gaudet et al. 1995). The guideline was intended to protect biota from exposure not only to 100 ng/L THg, but also to protect them from exposure to more than 10 ng MeHg/L on the assumption that MeHg is less than 10% of THg in surface waters (Gaudet et al. 1995). While the basis of the original guideline was bioaccumulation related, the value was later supported by chronic toxicity studies. The goal was to derive a water concentration that would protect the marketability of freshwater fish, while protecting fish from toxic effects (CCREM 1987). Many provinces adopted this guideline for water.

British Columbia employs two criteria for total mercury. First, a value of 100 ng/L to protect freshwater aquatic life from acute exposure and second, a 30-day average (5 weekly samples) value of 20 g/L to protect aquatic life from chronic effects plus "prevent undesirable accumulation of mercury from water in fish" (Nagpal 1989). The chronic value was derived by dividing a recommended acceptable fish tissue concentration (100 ng/g)[5] by the bioconcentration factor[6] (BCF) for inorganic Hg (as $HgCl_2$) for fathead minnows (5000) (Snarski and Olson 1982).

[5]Concentration considered by British Columbia to be a level that would not result in excessive accumulation of fish-based mercury by human consumers for whom fish is the main source of meat.

[6] BCF = $[Hg]_{tissue}/[Hg]_{water}$; considers uptake from water only

Alberta developed draft acute and chronic guidelines for total Hg and MeHg. These guidelines were intended to protect all stages of aquatic life (Alberta Environmental Protection 1998). The values have not been approved or adopted formally, and Alberta presently uses the CCME value.

Manitoba adopted one of the most stringent guidelines for mercury to protect cold water aquatic life, cool water aquatic life, and wildlife. Their freshwater guideline for total mercury of 6 ng/L was adopted in 1988 (Williamson 1988) and is ten times higher than their guideline recommended in 1983 (0.57 ng/L)[7]. Manitoba's objectives for toxic substances in general are that the media should be "free from substances in concentrations or in combinations that injure, be toxic to, or produce adverse physiological or behavioural responses in humans, aquatic, semi-aquatic and terrestrial life" (Williamson 1983). The guideline may be adjusted for mixing zones and for low flow periods which are thoroughly defined by Williamson (1983).

Ontario's Provincial Water Quality Objective (PWQO) for total mercury to protect aquatic life is 200 ng/L in filtered water. The total amount of mercury in filtered water samples is arbitrarily considered to be methylmercury. The province recognises that this level of mercury in water "will result in accumulations of methylmercury in aquatic life in excess of 0.5 µg/g"; 0.5 µg/g was the NOAEL for wildlife consumption of fish as well as the guideline for human consumption of fish. Ontario recommends simultaneous application of both objectives. The PWQO was first published in 1976 by the International Joint Commission for the Great Lakes and adopted in 1979 by Ontario (MOEE 1979). Ontario re-published this objective in 1999 (MOEE 1999).

Québec has both acute and chronic criteria. The critère de vie aquatique aigu (CVAA) is the maximum concentration of a substance to which aquatic organisms may be exposed for a short period of time without being adversely affected. For inorganic mercury the CVAA is 1 694 ng/L (MEF 1998). MEF recognises that this criterion requires modification when methylmercury is present. The critère de vie aquatique chronique (CVAC) is the highest concentration of a substance which does not result in adverse effects in aquatic organisms exposed daily throughout their life cycle. The CVAC for inorganic mercury is 908.1 ng/L (MEF 1998). These values were adopted from the U.S. EPA Great Lakes Water Quality Initiative (U.S.EPA 1995a).

To protect aquatic life, the U.S. EPA (U.S.EPA 1999) currently recommends a national criteria maximum concentration (CMC) for total mercury of 1 400 ng/L. A CMC is defined as the highest concentration of a pollutant to which aquatic life can be exposed for a short period of time (1-h average) without deleterious effects. The national criteria continuous concentration (CCC) for total mercury is 770 ng/L. The CCC is the highest concentration of a pollutant to which aquatic life can be exposed for an extended period of time (4 days) without deleterious effects. These criteria are based on those for the derived for the Great Lakes System (below), corrected for the dissolved fraction of mercury in the water column. The criteria were developed with data for inorganic mercury but are applied to total mercury. The U.S. EPA recognises that

[7] The 1988 guideline was derived by dividing Health Canada's recommended consumption guideline for fish of 500 ng/g by the highest BCF for MeHg and fathead minnows (81670; Olson et al. 1975). The calculation is 500 ng/g ÷ 81670 X 1000 g/L to give 6 ng/L.

if a substantial portion of the mercury in the water column is methylmercury these criteria may be under-protective. These criteria do not account for bioaccumulation (U.S.EPA 1999).

In addition to national criteria, the U.S. EPA has criteria for the Great Lakes system. In contrast to the national values (above), the Great Lakes values are expressed as total recoverable inorganic mercury. The CMC and CCC to protect freshwater life in ambient water are 1 694 and 908.1 ng/L, respectively. The Great Lakes wildlife criterion for total mercury is 1.3 ng/L (U.S.EPA 1995b).

The European Economic Community (EEC) has water quality objectives to protect biota under EEC Directives. According to the Royal Society of Chemistry, EEC Directives 82/176/EEC and 84/156/EEC stipulate that inland surface waters should not exceed 1 000 ng/L total mercury (annual mean; Royal Society of Chemistry 1994). Higher levels of 50 000 ng/L (50 µg/L) are given for effluents from plants treating toxic wastes, for metal refineries, and several other industries (p. 412, Royal Society of Chemistry 1994).

Detailed compendia of environmental quality guidelines, criteria, and other benchmarks have been published elsewhere (Haines et al. 1994; MacDonald et al. 1999).

3.1.3 Rationale for Inorganic Mercury

According to the data requirements, sufficient information was available to derive a full guideline for inorganic mercury. Studies using inorganic mercury (as $HgCl_2$) that were ranked primary were available for six species of fish (fathead minnow [*Pimephales promelas*], channel catfish [*Ictalurus punctatus*], largemouth bass [*Micropterus salmoides*], rainbow trout [*Oncorhynchus mykiss*], coho salmon [*O. kisutch*] and white sucker [*Catostomas commerson*]), three types of invertebrates (daphnids, crayfish and larval midge), and two types of plants (a macrophyte and periphyton). The toxicity of inorganic Hg to freshwater organisms is described in Part II, Chapter 7, and the toxicity endpoints summarised in Table 4 and Figure 1.

The guideline value recommended for inorganic Hg is based on the most sensitive lowest-observable-adverse-effects level (LOAEL) of 0.26 µg Hg/L for fathead minnows reported by Snarski and Olson (1982a). In flow-through experiments with measured Hg levels, juvenile fathead minnows were exposed to inorganic Hg (as $HgCl_2$) in unfiltered water from Lake Superior for 60 days. Observed effects include reduced growth and survival, reproductive impairment, and increased curvature of the spine. The most sensitive endpoints were reduced growth (weight) of exposed offspring from exposed parents, and reproductive impairment evidenced as reduced spawning and egg production. Exposure at all concentrations tested (0.26 to 3.69 µg Hg/L) reduced the growth of second generation larvae relative to controls. No spawning occurred at or above 1.02 µg Hg/L and the number of spawning pairs was reduced at 0.26 and 0.50 µg Hg/L resulting in total egg production of about 50% of controls (Snarski and Olson 1982a). Fish exposed chronically to 0.26 µg Hg/L accumulated mean whole body total mercury residues of 1.36 µg Hg/g wet weight.

The critical endpoint for guideline derivation, defined as the most sensitive non-lethal LOAEL on a native Canadian species, is the LOAEL of 0.26 µg Hg/L for the fathead minnow. This value was multiplied by a safety factor of 0.1 to give a WQG to protect freshwater life of 0.026 µg Hg/L or 26 ng Hg/L. The next most sensitive organism showing a non-lethal response was *Daphnia magna* with an EC_{50} (reproduction) over 21 days, of 1.28 µg Hg/L (Biesinger et al. 1982). Only studies ranked primary were considered (see Part II, Chapter 7 and Table 4).

3.1.4 Rationale for Methylmercury

Far fewer studies were available for methylmercury than for inorganic Hg. Studies ranked primary and secondary were available for four species of fish (rainbow trout, brook trout [*Salvelinus fontinalis*], coho salmon, and lamprey [*Petromyzon* sp.]), one species of invertebrate (water flea [*Daphnid magna*]), an amphibian (leopard frog [*Rana pipiens*]) and three types of plants (macrophyte, blue-green and green algae). The toxicity of MeHg to freshwater organisms is described in the Part II, Chapter 7, and summarised in Table 4 and Figure 2.

There is a shortage of studies on invertebrates in that just one group, daphnids, is represented. The WQG protocol outlines a minimum recommended data requirement of two studies on two or more invertebrate species from different classes (CCME 1991). This factor might normally exclude a substance from eligibility for guidelines; however, the available studies are of high quality with ecologically significant endpoints (e.g., reproductive effects) and demonstrate that MeHg is at least ten times more toxic than inorganic Hg. An interim guideline is recommended for MeHg based on a study that employed flow-through experiments, unfiltered water from Lake Superior, and measured concentrations of MeHg ranging from 0.04 to 0.26 µg Hg/L. The LOAEL of 0.04 µg Hg/L caused a significant decrease in *D. magna* production of young (Biesinger et al. 1982). A safety factor of 0.1 is applied to the LOAEL (0.1 X 0.04 µg Hg/L) to give an interim freshwater guideline for methylmercury of 0.004 µg Hg/L or 4 ng Hg/L.

According to protocol, this guideline was derived based on exposure to water only. Methylmercury, however, is known to accumulate principally in higher trophic level aquatic life (e.g., piscivorous fish) and wildlife through food. Thus, we recommend that this WQG be restricted to low trophic level species (generally trophic levels 1-2) that are exposed to MeHg primarily through water. High trophic level species (generally trophic levels 3-4) that take up MeHg from food may not be adequately protected. Moreover, this WQG may not prevent the accumulation of MeHg in high trophic level freshwater fish; therefore, through this process the Canadian TRG (33 µg MeHg/kg ww) for the protection of wildlife consumers of aquatic biota may be exceeded. Please read section 4.2 for guidance.

3.1.5 Research Recommendations

At the very minimum, one study of chronic effects to a non-crustacean invertebrate would improve the database for inorganic Hg ($HgCl_2$). While all the assays that were ranked primary were of high quality, many of them are not recent studies. It would strengthen the database in general if some of the older studies were repeated in modern laboratory facilities for the same and/or different species and life stages.

For MeHg, a general shortage of studies exists. No suitable studies were found for non-daphnid invertebrate species. To optimise the development of a full freshwater MeHg guideline, the following studies on North American freshwater biota are required:

- chronic studies on a coldwater fish species;
- chronic and acute studies on warmwater fish species;
- chronic and acute studies on a non-planktonic invertebrate species; and
- studies on a freshwater vascular plant or algal species.

There is a strong need for a guideline that recognises the bioaccumulative nature of methylmercury, and that high trophic level aquatic organisms take up methylmercury primarily through food. It is recommended here that a protocol for the derivation of Tissue Residue Guidelines to protect aquatic life (as opposed to TRGs to protect wildlife consumers of aquatic life) be developed to address uptake and accumulation of substances such as methylmercury. A TRG to protect aquatic life could be a generic numerical concentration or narrative statement pertaining to a chemical or substance present in the tissues of aquatic biota such as fish, shellfish, invertebrates, and aquatic plants that is not expected to result in adverse effects in these organisms. By being tissue based, such a TRG could include toxicity elicited from contaminants in water, sediment, and food. Moreover, the bioavailability and bioaccumulation of these substances would be inherent in this type of guideline.

3.2 Marine Life

3.2.1 Interim Guidelines

An interim water quality guideline of 16 ng Hg/L is recommended for the protection of all stages of marine life against the adverse affects of inorganic mercury in water. This WQG applies to unfiltered water samples. Owing to the paucity of data, no guideline is recommended specifically for MeHg. The WQG for inorganic Hg will protect organisms from adverse affects associated with direct exposure to water, but may not prevent bioaccumulation of MeHg in aquatic biota; therefore, through this process, concentrations of MeHg in tissues of marine life could exceed the Canadian TRG to protect wildlife. The toxicity of inorganic Hg to marine organisms is described in the Part II, Chapter 7, and summarised in Table 5 and Figure 3.

3.2.2 Summary of Existing Water Quality Guidelines to Protect Aquatic Life

Few jurisdictions have distinct guidelines for mercury to protect marine biota. British Columbia has two types of guidelines. One, to protect marine and estuarine life from acute exposure to mercury, the maximum concentration of total mercury in water should not exceed 2.0 µg/L. Second, to prevent undesirable accumulation of mercury from water in fish, the average concentration of total mercury in water over a 30-day period (based on 5 weekly samples) should not exceed 0.02 µg/L (Nagpal 1989). Québec adopted an acute criterion of 2.1 µg/L to protect marine life from the U.S. EPA, although the U.S. EPA has since changed this value (MEF 1998; U.S.EPA 1985; U.S.EPA 1999). Québec applies this guideline to inorganic mercury while the

U.S. EPA applied it to total recoverable mercury. Currently, the U.S. EPA recommends a CMC and a CCC of 1.8 and 0.94 µg/L for total dissolved mercury (U.S.EPA 1999). Both of these criteria are based on U.S. EPA's 1984 assessment; modifications included correction from total recoverable to dissolved fraction and, in the case of the CCC, use of the final chronic value instead of the final residue value. No new data were included in the re-calculations (U.S.EPA 1985; U.S.EPA 1999). European guidelines for salt waters range from 0.005 µg/L to prevent ecotoxicological damage to estuaries in the Netherlands (Stortelder et al. 1989), to 0.5 µg/L as a level for elemental Hg and its compounds to protect aquatic life in estuary waters (CEC 1988).

3.2.3 Rationale for Inorganic Mercury

Studies using inorganic Hg (as $HgCl_2$) ranked primary or secondary were available for two species of fish (mummichog [*Fundulus heteroclitus*] and chum salmon [*Oncorhynchus keta*]), nine types of invertebrates (mysid [*Mysidopsis bahia*], blue crab [*Callinectes sapidus*], slipper limpet [*Crepidula fornicata*], American oyster [*Crassostrea virginica*], hard clam [*Mercenaria mercenaria*], amphipod [two species], sea urchin [*Paracentrotus lividus*], estuarine rotifer [*Brachionus plicatilis*], and blue mussel [*Mytilus edulis*]), and several types of unicellular algae and macrophytic brown algae.

The interim guideline is based on the most sensitive LOAEL inhibiting growth of a population of the marine coccolithophore, *Emiliania huxleyi* (Fisher et al. 1984). In nature, *E. huxleyi* occurs in all but the polar oceans. This species is ecologically important because of its influence on the ocean carbon cycle and global climate change. *E. huxleyi* takes up bicarbonate to produce calcium carbonate plates called coccoliths. Coccoliths settle to the ocean floor and with much time form chalk and limestone. Blooms of *E. huxleyi* (up to 100 000 km^2) reflect light and heat, cooling the ocean slightly. *E. huxleyi* affects climate also by reducing CO_2 to organic matter. In the critical study, a static test, exposure to 0.16 µg/L of inorganic mercury (as $HgCl_2$) for 72-h reduced the population growth of *E. huxleyi* by 50%; 0.32 µg/L halted growth completely. In the same study, a cyanophyte, *Oscillatoria woronichinii*, and a diatom, *Thalassiosira pseudonana*, were sensitive as well with EC_{50}s for growth of 0.40 and 0.63 µg/L, respectively while a chlorophyte, *Dunaliella tertiolecta* was quite tolerant with an EC_{50} for growth of 1005 µg/L (Fisher et al. 1984). The most sensitive animal species was an invertebrate, *Mysidopsis bahia*, with a LOAEL for reproductive impairment of 1.2 µg/L (Gentile et al. 1983). Test fish species were typically more than an order of magnitude less sensitive than the mysid.

The most sensitive LOAEL of 0.16 µg Hg/L was multiplied by a safety factor of 0.1 to give a water quality guideline for inorganic mercury of 0.016 µg/L or 16 ng/L.

3.2.4 Methylmercury

There was insufficient toxicological research available to recommend an interim guideline for MeHg for the protection of marine biota. The few studies available are described in Part II, Chapter 7. Although no WQG could be derived, MeHg that accumulates in marine biota may pose a threat to wildlife. Please read section 4.2 and the Canadian Tissue Residue Guideline for the protection of wildlife consumers of aquatic biota for more information (Environment Canada 2002).

3.2.5 Research Recommendations

To develop a full guideline for inorganic Hg, studies are needed for two other marine fish species besides the mummichog. While invertebrates and plant species are sufficiently represented for inorganic Hg, suitable studies for MeHg do not exist. The following studies are required for a interim marine guideline for MeHg:

- chronic studies on one more temperate marine fish species (besides mummichog);
- at least two chronic studies on two or more temperate marine invertebrate species from different classes;
- at least one study on a temperate marine vascular plant or marine algal species;
- and given that plant (algal) species appear to be the most sensitive species based on studies with inorganic mercury.

Please see section 3.1.5 for a recommendation regarding the development of a Tissue Residue Guideline to protect aquatic life.

4.0 Application of Canadian EQGs

Canadian EQGs are used by federal, provincial, and territorial governments to achieve the highest levels of environmental quality across Canadian jurisdictions. The legislative authority for implementation of Canadian EQGs and other environmental assessment tools lies primarily with each provincial or territorial jurisdiction, with the exception of federal lands. Provincial and territorial jurisdictions may have or may develop their own science-based environmental assessment tools (e.g., criteria, guidelines, objectives, and standards), which may be implemented within their respective jurisdictions. In many cases, the Canadian EQGs form the scientific basis upon which further site-specific criteria, guidelines, objectives, or standards are developed within the various jurisdictions. For example, provincial and territorial governments may use EQGs in developing point-source licenses and permits for discharges. Variation in environmental conditions across Canada will affect environmental quality in different ways. EQGs do not constitute values for uniform national environmental quality. Users of EQGs may need to consider local conditions and other supporting information during the implementation of EQGs. Science-based site-specific criteria, guidelines, objectives, or standards may, therefore, differ from the Canadian EQGs recommended in this document. For ecosystems of superior quality, impairment to guideline concentrations is not advocated.

4.1 Factors to Consider Before Applying for the Canadian WQG for Mercury

Until now, Canadian Water Quality Guidelines have been recommended for "mercury", meaning all mercury species (i.e., total Hg). This approach was used despite the fact that toxicity studies on which guidelines were based were completed on a specific chemical species, whether it was the inorganic form, usually mercuric chloride, or an organic form, methylmercury. These species are a mixture in natural environments, but are not mixed in controlled laboratory experiments. There may be trace amounts of MeHg in an experiment with inorganic Hg due to methylation in laboratory vessels[8].

Analytical techniques and instrumentation for mercury analyses have improved dramatically in the past decade, and detection limits as low as 1 pg Hg/L are now achievable using the U.S. EPA Method 1631 (R. Flett, Flett Research Ltd., pers. com. 2000). This permits the detection of the trace levels of MeHg that are found in natural environments, based on reports that MeHg is typically less than 10% of THg in water samples (Hurley et al. 1995; Kelly et al. 1997). It also permits the recommendation of MeHg guidelines, where sufficient data are available for this most toxic of the mercury species.

Total mercury is routinely measured in water samples, without separation of inorganic and organic species. Many Canadian laboratories report THg and are either unequipped to or wish to avoid the greater expense of separate inorganic and methylated mercury analyses. The recommended guideline for inorganic mercury serves as an "alert level" for water bodies with THg values that exceed 26 ng Hg/L. Where there is concern about an exceedance of the

[8] Methylation rates are extremely low in the absence of sediments, especially in flow through conditions where the water is continually replaced (Miskimmin et al. 1992).

guideline, stakeholders could proceed with separate inorganic and MeHg measurements for those water samples. Suspected contaminated systems, such as newly formed reservoirs, could be analysed for MeHg wherever THg measurements exceed the MeHg guideline (4 ng Hg/L) because of the greater potential for a high proportion of MeHg in those water samples. For example, the percent MeHg in an experimental reservoir at the Experimental Lakes Area reached as high as 73% compared with about 4% of THg prior to flooding (Kelly et al. 1997). This was unusual and would only be expected in very perturbed systems, not in most natural surface waters. It serves to highlight the necessity for the vigilant application of a MeHg guideline at some sites.

Canadian freshwater CWQGs for mercury were derived from studies that employed and measured mercury concentrations in unfiltered water while the marine WQG was derived from a study that used artificial media. Use of unfiltered water is consistent with the CCME WQG protocol which states that CWQGs apply to the concentration of a substance in an unfiltered water sample. This application estimates conservatively the bioavailable fraction in that all of the compound is assumed to be available to be taken up by organisms; however, mercury species in unfiltered water may be bound to particulates, depending on the species in question, organic content, and particulate concentration. As such, recent studies tend to measure the dissolved fraction of mercury in water samples because this fraction is believed to reflect more accurately the fraction that is bioavailable (see Part II, section 4.3). Even in filtered water samples, bioavailability of mercury may be affected by the formation of complexes with colloids like humic substances (dissolved organic carbon). Analytically, the bioavailable fraction is that which remains in the water sample following filtration through a 0.45 μm filter (U.S.EPA 1995c). Comparing a measurement from a filtered water sample to the WQG based on an unfiltered water sample may lead to an inaccurate conclusion about whether or not the WQG is exceeded. We recommend that concentrations of mercury in the dissolved fraction be converted to the total fraction equivalent (i.e., dissolved plus particulate) where the site-specific dissolved:total ratio is known. Otherwise, the WQG may be expressed in terms of the dissolved fraction by dividing the appropriate WQG by 0.7. This conversion factor is based on a literature review conducted by the U.S. EPA who found that for total mercury (inorganic Hg and MeHg), the dissolved fraction accounted for an average of 70% of the total (dissolved plus particulate) fraction (U.S.EPA 1997a). Separate estimates for inorganic mercury and methylmercury were not given.

4.2 Coherence between the CWQGs and the TRG for Mercury

Many environmental managers will be faced with the issue of ensuring the protection of wildlife consumers of Hg contaminated fish. While this issue was addressed in the TRG document, the CWQGs were derived to protect aquatic species, and may not prevent fish from exceeding the TRG of 33 μg MeHg/kg at all sites (Environment Canada 2002). In areas where the TRG is exceeded, wildlife that consume aquatic biota may be at risk of adverse health effects. Reference concentrations for selected wildlife species from the TRG technical supporting document were used in conjunction with bioaccumulation factors (BAFs) estimated by the U.S. EPA, to determine concentrations of methylmercury in water that would protect wildlife that consume aquatic biota (see Box1; Environment Canada 2002).

Box 1

[MeHg] in water = RC ÷ BAF (Equation 1)

where;

[MeHg] in water below	=	the concentration of MeHg dissolved in water which methylmercury is not expected to bioaccumulate in tissues of aquatic life above the RC;
RC	=	a reference concentration (units µg MeHg/kg diet ww) for a particular wildlife species; and
BAF	=	a bioaccumulation factor for the trophic level of aquatic life at which the wildlife species feeds; ratio of concentration of methylmercury in tissue to concentration of methylmercury in water.

A reference concentration is the maximum concentration of methylmercury (on a wet weight basis) in tissues of aquatic life recommended to protect a given wildlife species that consume aquatic biota. RCs are calculated for key indicator wildlife species (e.g., piscivores) using information on body weight and daily food intake for these wildlife species as well as the tolerable daily intake of the substance of concern. The lowest RC is adopted as the national tissue residue guideline (CCME 1998).

A bioaccumulation factor is the ratio of the concentration of methylmercury in the tissues of an organism to its concentration in water. BAFs account for accumulation from all routes of exposure (i.e., could include food, sediment, and water) unlike bioconcentration factors (BCFs) which account for accumulation from water only. Bioaccumulation of mercury in aquatic biota is discussed in detail in Part II, Chapter 6, while bioaccumulation in wildlife is discussed in the TRG for MeHg (Environment Canada 2002). In general, bioaccumulation factors for methylmercury vary greatly among aquatic biota depending on species, trophic level, and food chain structure. **As such the selection of a BAF should be as species- and site-specific as possible to ensure adequate representation of local conditions.**

In the absence of appropriate site-specific data, the U.S. EPA recommends a BAFs for methylmercury of 1.6×10^6 and 6.8×10^6 for trophic levels 3 and 4, respectively (U.S.EPA 1997a). The U.S. EPA estimated these BAFs directly from field data collected primarily from oligotrophic water bodies in the northern United States with some data from Manitoba (Jackson 1991; U.S.EPA 1997a). These BAFs were calculated from the concentrations of methylmercury in tissues and concentrations of methylmercury *dissolved* in water. The geometric mean BAF of each trophic level data set was taken as the final BAF estimate because it is considered to be the best (unbiased) estimate of central tendency for lognormal distributions. This direct field-based approach to calculating BAFs is preferred over other methods (e.g., modified Great Lakes Water Quality Initiative methodology, predator-prey factors) because it results in the least variability of the geometric mean estimate (U.S.EPA 1997a). In using these BAFs, the following assumptions apply: 1) the BAFs are appropriate to all Canadian aquatic life; and 2) the BAFs are appropriate

for all Canadian sites. As stated previously, we recommend use of species- and site-specific BAFs whenever possible.

Concentrations of MeHg in water may be adjusted to equivalent concentrations of Hg ion for comparison with the recommended Canadian WQG for methylmercury for the protection of aquatic life (see section 3.1.4). An additional correction is needed to express the amount of MeHg in unfiltered water because the BAFs from the U.S. EPA were based on the *dissolved* (i.e., filtered) fraction. No estimates, however, exist on the proportion of dissolved methylmercury in an unfiltered water sample. The U.S. EPA noted that on average total mercury dissolved in water accounted for approximately 70% of total mercury in unfiltered water (U.S.EPA 1997b). We recommend that in the absence of empirical data specific to methylmercury, assume that the dissolved portion of methylmercury accounts for 70% of methylmercury in an unfiltered water sample.

Methylmercury RCs for four species of wildlife were chosen to exemplify Equation 1 (Box 1): Wilson's storm petrel (*Oceanites oceanicus*), mink (*Mustela vision*), herring gull (*Larus argentatus*), and bald eagle (*Haliaeetus leucocephalus*) (Appendix A). These RCs were used in conjunction with BAFs for aquatic life to estimate concentrations of methylmercury in water. Concentrations were adjusted for expression as Hg^{2+} ion equivalents in unfiltered water samples. Below these values, methylmercury is not expected to bioaccumulate in tissues of aquatic life to an appreciable degree; therefore, adverse effects in wildlife that consume aquatic life are not expected to occur. The examples provide generic calculations intended as a guide to determine the relationship between the WQG and the TRG. They do not consider many site-specific factors including wildlife species of concern and their preferred diet, or site- or species-specific bioaccumulation factors and, therefore, should not be used as site-specific water quality objectives (WQOs).

The example calculations suggest that concentrations of MeHg in unfiltered water above 200 pg Hg/L could pose a risk to wildlife in that this level could result in the accumulation of MeHg in fish tissues to levels above all RCs. A water concentration of 7 pg Hg/L or less is thought to be protective of all wildlife species that consume aquatic biota. Concentrations in-between these two limits may be a hazard to some wildlife depending on their RC, their feeding preferences, and the BAF for their prey species. Because these estimates are based on national RCs and on field-derived BAFs, they are believed to reasonably represent average conditions. These upper (200 pg Hg/L) and lower (7 pg Hg/L) limits for MeHg concentrations in water to protect wildlife are 10- and 1000-fold, respectively, below the WQG of 4 ng Hg/L (or 4 000 pg Hg/L); see Chapter 3.

Site-specific WQOs are preferred wherever possible and they may be calculated using the basic approach presented above, modified with species- and site-specific data. For example, these calculations should employ the RC for the most appropriate wildlife species of concern at a given site. RCs themselves may be adjusted with site-specific food intake:body weight ratios or with a safety factor (note that none was used on the derivation of the national RCs). Site-specific WQOs may consider also the measured proportions of aquatic life at each trophic level in the diet of a particular wildlife species. In addition, BAFs specific to the aquatic species comprising the diet of the wildlife species should be used.

4.3 Summary of Existing Water Quality Guidelines to Protect Wildlife

In their congressional report, the U.S. EPA recommended a Water Criteria (WC) of 50 pg MeHg/L (0.05 ng MeHg/L) to protect wildlife. The WC is defined as the concentration of MeHg dissolved in water that, if not exceeded, protects both avian and mammalian wildlife that use the water as a drinking or foraging source (U.S.EPA 1997b). The value of 50 pg/L was the average of two WC values for mammals (mink and river otter), and was lower than the mean value of four avian species (kingfisher, loon, osprey, and bald eagle). Previous to this national criterion, the U.S. EPA calculated a criterion for total mercury specific to the Great Lakes system of 1 300 pg/L (U.S.EPA 1995b). The value of 1 300 pg/L was the average of two WC values for mammals (mink and river otter), and was lower than the mean value of three avian species (kingfisher, herring gull, and bald eagle). In order to compare these values, the U.S. EPA converted the national value for dissolved MeHg to a value for total mercury in unfiltered water (dissolved plus particulate). The corrected national value (910 pg/L) is approximately 70% of the Great Lakes value (1 300 pg/L) (U.S.EPA 1997b).

PART II: Background information – Mercury and Methylmercury

1.0 Physical and Chemical Properties

1.1 Properties

Elemental mercury (Hg^0, atomic No. 80, CAS[9] No. 7439-97-6) has a molecular weight of 200.59, belongs to group IIb of the periodic table of elements, and has often been called quicksilver because it is a silver-white liquid at room temperature. This form of Hg is quite volatile (vapour pressure 0.16 Pa at 20 ^oC), and is more likely to be found in the atmosphere than in water (Table 1).

Elemental Hg is chemically quite different from the other two members of group IIb metals, cadmium and zinc. Elemental Hg has a very low melting point (-37^oC) compared with cadmium (321^oC) and zinc (420^oC). Its high surface tension and uniform volume of expansion make mercury ideal for use in thermometers, barometers and other measuring devices (Smith and Rowan-West 1996). Of the three metals, only Hg may become methylated in the environment.

Mercury can exist theoretically in various valence states (0, I and II). Hg^0 is not readily soluble in water, though natural waters tend to be supersaturated with Hg^0 compared to air resulting in its volatisation (Morel et al. 1998). In water, the mercurous state [Hg(I)] of mercury exists as a doubly-charged binuclear ion (dimer), Hg_2^{2+}. Hg(I) combines most commonly with inorganic molecules, though Weber (Weber 1993) speculated that methyl iodide could act as a methyl donar. The chemical compounds of the mercuric ion [Hg(II) i.e., Hg^{2+}] are highly stable and much more numerous than those of Hg(I) (OECD 1994b).

The mercuric cation, Hg^{2+}, forms a covalent bond with chloride which is relatively weak compared to bonds formed with other inorganic anions, although mercuric chloride ($HgCl_2$) may dominate when chloride salts are abundant (Alberta Environmental Protection 1992). $HgCl_2$ is more likely to be associated with sediments, with log K_{sed} values of 3.4-4.1 reported (Hurley et al. 1994). Hydroxyl anion (log K_1=10.6) has a higher affinity for Hg in the absence of organic complexing agents and will dominate in most freshwaters unless the pH is low, or chloride is high. Under reducing conditions, Hg preferentially forms stable, largely covalent bonds with sulphide (including thiols) and selenides whenever these ligands are present (Jackson 1998).

Mercuric forms of Hg can be transformed through abiotic and biotic processes to form alkylmercury compounds such as monomethylmercury [CH_3Hg^+], dimethylmercury [$(CH_3)_2Hg$], and aryl compounds [e.g., phenyl-mercury] (Alberta Environmental Protection 1992). Monomethylmercury is commonly referred to as methylmercury and abbreviated as MeHg. It is very toxic and accumulates readily in aquatic biota (World Health Organization 1989; Beckvar et al. 1996). The dimethylmercuric form is volatile. Methylation, demethylation and the environmental fate and behaviour of the various forms of Hg are described later in this report.

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[9] The Chemical Abstract Service (CAS) Number asssociated with a pollutant. Used to uniquely identify a pollutant.

The most toxicologically relevant form of mercury is monomethylmercury. MeHg has a high chemical affinity for sulphydryl (-SH) groups in body tissue proteins compared to inorganic Hg, which facilitates its transport across cell membranes, resulting in toxicity to and bioaccumulation in biota (Saouter et al. 1993; Mason et al. 1995). Methylmercury has a log K_{ow} or octanol-water partition coefficient ($K_{ow} = [Hg]_{octanol}:[Hg]_{water}$) of 1.7-2.5, compared with up to 3.3 for neutral $HgCl_2$ (Table 1). These log K_{ow} values indicate that the mercury compounds have only a moderate affinity for body fats relative to many organochlorine substances (e.g., toxaphene 6.44; DDT compounds >5.5). For mercury, accumulation in lipid is insignificant.

1.2 Sample Collection and Analytical Methods

Until the late 1980s, many mercury measurements in water samples were artificially high because of sample contamination (Driscoll et al. 1994). For example, mercury concentrations in water from Vandercook Lake, Wisconsin, saw an apparent decrease from over 200 ng/L in 1983 to ~50 ng/L in 1985 to 0.5 ng/L in 1986 as investigators used progressively cleaner techniques for sample collection and handling (Fitzgerald and Watras 1989). Coincidentally, formerly undetectable levels of mercury and MeHg (such as in filtered water samples) are now commonly reported because of improvements in analytical techniques. Notably, the detection limits for MeHg are often in the pg/L range (Bloom 1989a; Lee 1987). At the Third and Fourth International Conferences on "Mercury as a Global Pollutant" in July 1994 and August 1996, respectively, entire sessions were devoted to mercury collection and measurement methods (see Water, Air, and Soil Pollution. 1995. 80[1-4] and Fresenus' Journal of Analytical Chemistry. 1997. 358[3]).

1.2.1 Water sample collection

It is now common practice to use trace-metal clean collection techniques for samples intended for mercury analyses.

Typically, *Teflon*® bottles are first washed with hot HNO_3, filled with a 1% ACS grade HCl solution, and stored in new sealable polyethylene bags (St.Louis et al. 1994). Bottles should be transported to the field within clean containers that may also be placed inside plastic bags. All handlers wear new polyethylene gloves. At the site, the HCl solution is drained and the bottle rinsed three times with sample water, filled, then placed back into the bags.[10] If a sample pump is used, acid-rinsed *Teflon*® tubing should be used throughout. Within 2 h of sample collection, a small quantity of concentrated *trace metal* grade HCl (250 µL in 250 mL sample; to make a 0.1% solution) is added as a preservative (St.Louis et al. 1994).

Unfiltered samples may then be used for mercury analysis. For dissolved mercury analyses, filtering should be done with either an acid-cleaned glass (Ramamoorthy 1982) or *Teflon*®

[10] For the 1 % acid solution which is stored in the Teflon bottles, Vincent St. Louis (pers. com.) uses ACS grade HCl acid. It is poured out of the bottle before sampling and the bottles are subsequently rinsed with sample at least 2 times before sample is put in the bottle. The acid which is used in reagents or for preservation is high quality and is analysed every bottle for total Hg content.

filtration apparatus using acid-cleaned polycarbonate filters (e.g., 0.4 μm nucleopore filters) (Gill and Bruland 1990; Hurley et al. 1995).

1.2.2 Analysis of Total Mercury and Methylmercury in Water

The methods of analysis used world-wide for mercury and its compounds are too numerous to discuss in this report. Descriptions of methods in use are tabulated from pages 248 to 255 in U.S. DOH and HS (1994a). Another method recently approved by the U.S. EPA is method 7473 which is suitable for detecting total mercury in soils, sediments, bottom deposits, and sludge-type materials as well as in aqueous wastes and ground waters. The typical working range for this AAS (atomic absorption spectrophotometric) method is 0.05 - 600 ng (Salvato and Pirola 1996[11]), making it less sensitive than Method 1631 described below.

Bloom (1989a) and Watras et al. (1995)[12] analyse for total and dissolved mercury and MeHg, plus Hg^0. They use a cryogenic gas chromatograph with a highly sensitive cold vapour atomic fluoresence detector (CVAFS). MeHg is determined by aqueous-phase ethylation followed by purging of volatile methyl ethyl mercury onto graphitized carbon black (Carbotrap). Hg^0 is determined by sparging 500 mL of unfiltered, untreated water for 20 min with Hg-free N_2 and trapping the volatile Hg on gold. MeHg and Hg^0 are both thermally desorbed from their respective "traps" prior to CVAFS detection. The detection limits for total mercury ranges from 0.001 to 0.06 ng/L, from 0.001-0.02 ng Hg/L for MeHg (R. Flett, Flett Research Ltd., pers. com. 2000), and from 0.004 to 0.02 ng Hg/L for Hg^0 (Watras et al. 1995). Hydro-Québec, the Université de Sherbrooke and the Université du Québec à Montréal use modified versions of two techniques to measure total mercury and methylmercury in water samples: cold vapour atomic absorption spectrophotometry (CVAAS) and atomic fluorescence spectrophotometry. Detection limits are 0.1 ng/L for total mercury and 1 pg/L for methylmercury (Pichet et al. 1999).

An excerpted summary of a standard technique developed by scientists for the U.S. EPA follows (1996a). This method is sensitive enough to detect low levels of MeHg in surface water. The excerpt is intended to provide the reader with an overview of the general steps involved in analysing mercury in water; it is not an endorsement for the methodology, though it is used by a few labs in Canada.

"2.0 Summary of EPA Method 1631
A 100–2000 mL sample is collected directly into cleaned, pretested, fluoropolymer bottle(s) using sample handling techniques specially designed for collection of mercury at trace levels (U.S.EPA 1995c).
For dissolved Hg, samples are filtered through a 0.45-μm capsule filter.
Samples are preserved by adding 5 mL/L of pretested 12 N HCl (to allow both total and methyl Hg determination) or 5 mL/L BrCl solution, if total mercury only is to be determined.

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[11] Method 7473 available on US EPA website: http://www.epa.gov/epaoswer/hazwaste/test/7473.pdf. GSC in Ottawa is the only practitioner of method 7473 in Canada (G. Groulx 2000, ATS Scientific, pers. com.)

[12] Both of these first authors were directly involved in developing U.S. EPA Method 1631 (U.S. EPA 1996).

Prior to analysis, a 100 mL sample aliquot is placed in a specially designed purge vessel, and 0.2 N BrCl solution is added to oxidize all Hg compounds to Hg(II).
After oxidation, the sample is sequentially prereduced with NH_2OH+HCl to destroy the free halogens, and then reduced with $SnCl_2$ to convert Hg(II) to volatile Hg(0).
The Hg(0) is separated from solution by purging with nitrogen onto a gold-coated sand trap.
The trapped Hg is thermally desorbed from the gold trap into an inert gas stream that carries the released Hg(0) into the cell of a cold-vapor atomic fluorescence spectrometer (CVAFS) for detection.
Quality is ensured through calibration and testing of the oxidation, purging, and detection systems." (U.S.EPA 1996a).

1.2.3 Analysis of Mercury in Sediment and Tissue Samples

Analysis of mercury in sediment and tissue samples was described in the Tissue Residue Guideline (TRG) (Environment Canada 2002). Briefly, the analysis is similar to water samples except the sample is either 1) digested with a mixture of hot (180^{o}C) nitric and sulphuric acids, or 2) treated with 60^{o}C nitric and sulphuric acids and oxidised with a solution of potassium permanganate, with excess permanganate titrated with 30% peroxide, or 3) microwave digested in the presence of nitric acid. In all cases the oxidised mercury is converted to the elemental state with a reducing solution containing stannous chloride (or stannous sulphate), hydroxylamine sulphate and sodium chloride (Hendzel and Jamieson 1976; Scruton et al. 1994). The mercury is partitioned into vapour and determined by GC/CVAA or GC/CVAFS as described above.

Table 1: Physical and chemical properties of mercury.

*Property**	**Mercuric Hg (II) ($HgCl_2$)**	**Methylmercury (CH_3Hg^+)**	**Elemental Mercury (Hg^0)**
Atomic number	--	--	80
Molecular Weight	271.5	215.63	200.59
Boiling point	--	$Hg(CH_3)_2$: 92°C/98.66 Kpa(4)	356.9°C/ 101.32 Kpa(1)
Chemical Abstract Services (CAS) No.	7487-94-7	22967-92-6	7439-97-6
Conductivity	--	--	0.022 $cal \cdot s^{-1} \cdot cm^{-3} \cdot {}^oC^{-1}$ (1)
Density (spec.gravity)	5.4(4)	$Hg(CH_3)_2$: 3.1874(4)	13.534 $g \cdot cm^{-3}$ at 25 °C(4)
Melting point	--	CH_3ClHg: 170 °C (4)	-38.87 °C
Bioconcentration factor (BCF) for fish (Fathead minnows)	5000(13)	81,670(14)	--
K_{0C} (organic carbon) **	Not avail.	DOC increases solubility (6)	Not avail.
Log K sediment	3.4-4.1(3)	2.9-3.2 (reduced by DOC in water; 6)	--
log K susp.particulates	5.35(2); 4.8-4.9(10)	5.73(2)	--
log K_{ow}**	3.3(12)	1.7(7)-2.54(5)	0.62(7)
Surface tension	--	--	475 dynes/cm(1)
Vapour pressure, Pa	1.87 X 10^{-2} @ 34 °C (4)	1.13 @ 20 °C MeHgCl (11)	0.16 @ 20°C (11) 2 X 10^{-3} at 25°C(10)
Water Solubility	69 g/L (4)	1 µg/L (8);	25-30 µg/L(9)

* With the exception of molecular weight, most of these properties are dependent on pH, temperature, carbon content and other characteristics, and are given as general values only.
** K refers to a partition coefficient of a substance between indicated phase:water (Hg concentrations in each medium). For example, K_{ow}=octanol/water partition coefficient; $K_{susp.part.}$=suspended particle/water; $K_{sed.}$=sediment/water; K_{oc}=organic carbon/water.

(1) (Alberta Environmental Protection 1992); (2) (Hurley et al. 1989); (3) (Hurley et al. 1994); (4) (Royal Society of Chemistry 1994); (5) (Halbach 1985); (6) (Miskimmin 1991); (7) (Major and Rosenblatt 1991); (8) (Eisler 1987); (9) (Krenkel 1974); (10) (Hurley et al. 1995); (11) (World Health Organization 1990); (12) (Mason et al. 1995) (13) (Snarski and Olson 1982a); (14) (Olson et al. 1975).

2.0 Production and Uses

The production and uses of mercury were described in the Canadian Tissue Residue Guideline for Methylmercury for the Protection of Wildlife Consumers of Aquatic Biota (Environment Canada 2002), and in the Canadian Sediment Quality Guidelines for Mercury (Environment Canada 1997). Because the production (mining) and use of mercury ultimately results in mercury deposition to watersheds (mainly through atmospheric transport), some of the basic information will be briefly described here.

2.1 Mining of Mercury

The mining of mercury introduces additional mercury into the global mercury cycle that would otherwise have remained buried. Mining essentially causes accelerated weathering, resulting in the release of much more mercury to the environment than would naturally occur.

Relatively few countries are involved in the production of mercury from mining cinnabar or other deposits. Important international mining locations include Algeria, Russia, China, Finland, Spain, and Mexico (OECD 1994b). Canada has no mercury mines that are currently active. Two mines were formerly active in central British Columbia along the Pinchi Fault zone. The Pinchi Mine was operational in the early 1940s and briefly in the early 1970s, and the Bralorne Takla Mine was operational for one year in the 1940s (Armstrong 1946; A. Plouffe 1999, GSC, pers. com.).

Mercury production from mining has declined steadily in the last 20 years (OECD 1994b). World-wide production decreased from 10,000 tonnes in 1970 to 5,500 tonnes in 1990. Significant secondary production of mercury results from recycling, recovery, and industrial reprocessing of mercury. In the United States, secondary industrial production ranged between 10 and 20 percent of total mercury consumption in 1985-89 (Minerals Yearbook 1989).

2.2 Uses of Mercury

Briefly, mercury is used in dental amalgams, exterior paints, thermometers, barometers, and electrical products such as dry-cell batteries, fluorescent lights, switches, and other control equipment. It is also used in the electrolytic preparation of chlorine and caustic soda (chlor-alkali industry) (Alberta Environmental Protection 1992), was formerly used as a seed and turf fungicide, and is an important chemical globally utilised by the gold mining industry to separate gold from other minerals into a gold-mercury amalgam.

Details of quantities of mercury used in specific Canadian products can be found in OECD (1994b), with information on current product use and registration given in Environment Canada (Environment Canada 1997) and Pilgrim and Ecological Monitoring and Assessment Network (1998).

There is only one operational chlor-alkali plant in Canada that uses mercury to produce chlorine and caustic soda (which is used by the pulp and paper industry) by passing a brine solution over mercury cells. This plant in northwestern New Brunswick meets all provincial and federal operational requirements in that mercury emissions do not exceed 1.68 kg of mercury per day. Atmospheric emissions from this plant are 15 times greater than emissions to water (Pilgrim and Ecological Monitoring and Assessment Network 1998). Emissions from abandoned chlor alkali sites may be significant, and the Commission for Environmental Cooperation (CEC) is developing guidelines for remediation of decommissioned mercury-cell chlor-alkali sites in the NAFTA countries (under Article 13 of the NAAEC[13]; Pilgrim et al. 2000).

By the early 1990s, there were only four remaining mercurial fungicides registered for use in Canada, all of which were for turf application (Sang and Lourie 1997). Since December 31, 1995, no further registration of mercury-containing fungicides was permitted although the remaining retail stocks could be used until depleted.

While tailings from amalgamating gold mines from the past contained unknown and variable quantities of mercury, none of the current gold mining operations in Canada practice mercury amalgamation (Pilgrim and Ecological Monitoring and Assessment Network 1998).

Mercury and mercury compounds are ranked as Level I persistent toxic substances by Environment Canada and the United States Environmental Protection Agency. In a bilateral strategy to eventually eliminate anthropogenic toxic substances in the Great Lakes Basin, goals have been set by each country concerning reductions in use of mercury compounds. The twelve Level I substances including mercury and its compounds are considered highly toxic, persistent and are likely to bioaccumulate in aquatic biota. The ranking considered a wide range of factors such as chemical and physical properties, potential to cause cancer, toxicity, risk to human health and wildlife, presence in the environment, as well as adverse impacts observed in the environment (Environment Canada and U.S.EPA 1997). Other international, national and regional policies and initiatives concerning mercury are summarised in the draft document "The Status of Cd, Pb and Hg in Canada: Natural Resources and Environmental Contaminants" (draft dated Mar/98, L. Trip, Transboundary Air Issues Branch, Environment Canada, pers. com.).

[13] NAAEC = North American Agreement on Environmental Cooperation, signed in 1994 between Canada, Mexico and United States.

3.0 Sources and Pathways into the Environment

Mercury is emitted from a variety of natural and anthropogenic sources, and can be carried in the atmosphere for thousands of kilometres and deposited in watersheds remote from the original sources. Although mercury usage continues to decline in Canada and the United States and many point sources of mercury have been reduced, mercury in the atmosphere remains elevated in North America in comparison with pre-industrial times because former discharges contribute to the current atmospheric loading and redistribution of mercury in the environment. (Rada et al. 1989; Engstrom et al. 1994; Pirrone et al. 1998). Atmospheric mercury may be deposited with wet and dry fallout directly on surface waters or on terrestrial areas where runoff and flow-through transport the various forms to surface waters containing fish and other aquatic biota (Mierle 1990; NESCAUM 1998; Sorensen et al. 1990).

The sources of mercury were described previously in the Canadian Tissue Residue Guideline for Methylmercury for the Protection of Wildlife Consumers of Aquatic Biota (Environment Canada 2002), and in the Canadian Sediment Quality Guidelines for Mercury (Environment Canada 1997). In addition, mercury sources, uses and recycling in the environment were reviewed by Nriagu (1979; 1989), International Joint Commission (1990a), Alberta Environmental Protection (1992), Watras et al. (1994), and Rudd (1995).

Natural sources of mercury include geological mercury deposits, rock weathering, forest fires and other wood burning, faults/volcanoes (land-based and oceanic), hotsprings, and a portion of the volatilisation from the oceans[14]. World-wide fossil fuel combustion is estimated to provide one-half of the global anthropogenic source, while the rest is released through volatilisation of fungicides, paints, manufacturing, mining, waste incineration, battery decay, fluorescent lamps and various other sources (Douglas 1991). The primary anthropogenic sources of Hg in Canada include: metal smelting, coal-burning power plants, municipal waste incineration, sewage and hospital waste incineration, coal and other fossil fuel combustion, cement manufacturing, and mercury waste in landfills or storage (Pilgrim and Ecological Monitoring and Assessment Network 1998). Other sources include removal and disposal of dental amalgams, and use and disposal of mercury-containing lamps (CCME 2000a; CCME 2000b).

There is uncertainty as to the contribution of natural compared to anthropogenic sources, as well as the relative importance of primary emissions compared to re-emitted mercury (Jackson 1997). For example, a large proportion of airborne anthropogenic Hg deposited in aquatic environments (notably the oceans) and on land is re-emitted to the atmosphere. These re-emissions might wrongly be considered as natural emissions, and result in an overestimation of natural emissions and underestimation of anthropogenic emissions (Jackson 1997).

The world-wide emissions of mercury, including natural and anthropogenic sources to the atmosphere, soil and water were 6060, 10 200, and 4600 tonne/a, respectively, in 1983 (Nriagu and Pacyna 1988; Nriagu 1989). The anthropogenic component has since decreased as use has declined, but there is general agreement that anthropogenic sources comprise at least half of the

[14] Another portion of oceanic emissions is re-emission of anthropogenic inputs.

global emissions (Fitzgerald 1986; Jernelov and Ramel 1994; Lindqvist et al. 1991; Fitzgerald 1986; Jernelov and Ramel 1994). A more recent review of world-wide Hg emissions to the atmosphere reported total anthropogenic emissions in the range of 2000 – 4000 tonne/a, and natural emissions in the range of 2200 – 4000 tonne/a (Pirrone et al. 1998). These recent global estimates are comparable to those shown in Table 2 (Nriagu and Pacyna 1988; Nriagu 1989). Precise estimates are difficult to make because it is currently impossible to accurately assess the recycled portion of mercury compounds re-emitted from the earth's surface including the oceans.

World-wide, major sources of Hg to water include coal combustion (41%), manufacturing processes (26%), and atmospheric fallout (20%) (Table 2). Mierle (Mierle 1990) found that more than half (57%) of the mercury supplied to a small lake in the Canadian Shield came directly from wet atmospheric deposition. Melting snow during spring runoff was estimated to contribute an additional 13% or more. Similarly, direct atmospheric deposition accounted for an average of 60% of the mercury found in the sediments of lakes in northern Minnesota (Sorensen et al. 1990); inputs from the watershed accounted for the balance (40%) of the mercury. In addition, newly created reservoirs are often responsible for temporarily increasing methylmercury concentrations in aquatic environments due to the accelerated microbial methylation of existing inorganic Hg forms caused by decomposing flooded vegetation (Abernathy and Cumbie 1977; Bodaly and Hecky 1979; Alberta Environmental Centre 1989; Hecky et al. 1991; Scruton et al. 1994; Anderson et al. 1995; Kelly et al. 1997; Bodaly and Fudge 1999; Schetagne and Verdon 1999a). The elevated MeHg in fish may decline in a few years or may remain elevated for decades (Bodaly et al. 1984).

Anthropogenic emissions estimates for various Canadian sectors were described for 1990 and 1995 by Pilgrim and Ecological Monitoring and Assessment Network (1998). North American emissions and deposition of mercury was recently reviewed by Pirrone et al. (1998), who estimated, based on the 1990 emissions estimate of 330 tonnes/a, that deposition on the continent ranged from 14.3 - 19.8 µg Hg m^2/a. Their estimates compared well with measured wet fluxes of 5 - 25 µg Hg·m^2/a in remote areas (Electric Power Research Institute 1994). In the Maritimes, mercury emissons from fuel combustion (wood, coal, and refined petroleum products) total 50 tonnes since 1800, the majority (>75%) of which is accounted for by coal combustion. Emissons peaked at 0.778 to 1.494 tonne/a in the 1940s, but there has been no consistent decline since this time (Sunderland and Chmura 2000).

The Canadian mining and smelting industry reduced the amount of mercury (elemental and inorganic) it releases into air and water from 28.2 tonne/a in the base year to 2.5 tonne/a in 1998 (i.e., 91% reduction) (MAC 2000). The Canada-wide Standard for base metal smelting set an environmental source performance (atmospheric) guideline of 2 g Hg/tonne of total production of finished metals for existing facilities. For new and expanding facilities, environmental source performance guidelines are: a) 0.2 g Hg/tonne production of finished zinc, nickel, and lead, and b) 1 g Hg/tonne finished copper (CCME 2000c). Existing facilities expect to meet their guideline by 2008 while all new facilities must comply upon full scale operations. These guidelines are expected to reduce mercury emissions by an additional 0.8 tonne/a (CCME 2000d).

Table 2: Estimates of world-wide emissions (tonne/a) of mercury to the atmosphere, water and soil in 1983.

Source category	**Atmosphere * min.**	**max.**	**Water min.**	**max.**	**Soil ** min.**	**max.**
Coal combustion	650	3500	0	3600	370	4800
Non-ferrous metal production	45	220	0	40	0	80
Refuse incineration Municipal Sewage sludge	 140 15	 2100 60	no estimate	--	no estimate	--
Wastewater (domestic)	no relevance	--	0	600	10	800
Wood combustion	60	300	no estimate	--	no estimate	--
Metal mining	Insignificant input	--	0	150	no estimate	--
Urban refuse	no estimate	--	no estimate	--	0	260
Wastage - commercial prod.	no estimate	--	no estimate	--	550	820
Manufacturing processes	no estimate	--	20	2300	no estimate	--
Atmospheric fall-out	no relevance	--	220	1800	630	4300
Phosphate fertilizer production and use	insignificant input	--	no estimate	--	no estimate	--
Agricultural Waste	no estimate	--	no estimate	--	0	1700
Logging and other wood wastes	no estimate	--	no estimate	--	0	2200
Mine tailings	no estimate	--	no estimate	--	550	2800
Smelter slags and wastes	no estimate	--	no estimate	--	50	280
Dumping of sewage sludge	no relevance	--	10	310	no relevance	--
Anthropogenic total inputs	910	6200	250	8800	2200	18100
Median	3560		4600		10200	
Natural source totals	100	4900	no estimate	--	no estimate	--
Median	2500		--		--	
Grand totals (min max)	**1010**	**11 100**	**250**	**8800**	**2200**	**18100**
Median)	**6060**		**4600**		**10200**	

(Anthropogenic values from Nriagu and Pacyna 1988; Natural values from Nriagu 1989)

* Insignificant contributions to the atmosphere from: oil combustion, zinc-cadmium production, secondary non-ferrous production, steel and iron manufacturing, cement production, and mobile sources.

** Landfills included

4.0 Forms and Fate in the Aquatic Environment

In aquatic ecosystems, the form of mercury and its transfer among phases (dissolved, adsorbed) is dependent on environmental characteristics. Site-specific factors that control the net production of MeHg (balance of methylation and demethylation), photodegradation, and distribution of MeHg between dissolved and solid phases are of particular importance because MeHg is much more toxic and more readily bioaccumulated than any other form of mercury.

The mercury cycle describes the speciation, fate, behaviour, and transport of mercury in the environment. For the purposes of this report, fate is defined as the quantification and movement of various mercury species among environmental phases (i.e., gaseous, water, colloids, suspended particulates, sediment or biota) in lakes and watersheds. Sorption, desorption, diffusion, volatilisation and transformation (reduction, methylation, demethylation) are among the processes that the various mercury species undergo in aquatic environments.

For inorganic mercury, the most toxicologically relevant process affecting natural environments is its conversion to MeHg. The relative abundance of MeHg in environmental samples is of particular concern because of its high toxicity to biota. MeHg is the major form of mercury accumulated in biological tissues due to the ease with which it permeates cell membranes and its strong affinity for sulphydryl groups in proteins (Faust 1992). It is not highly lipophilic, as is often reported, with moderate K_{ow} values of up to 2.5 found experimentally (Table 1). For MeHg, processes that define its fate as a species include demethylation and photodegradation. Diffusion of MeHg through biological surfaces and bioaccumulation are components of the mercury cycle that will be discussed more fully in the following chapter.

4.1 The Mercury Cycle

Chemical cycling is defined as inputs to and outputs from a system, along with transport and transformation of chemical species within the system (Zillioux et al. 1993). Transport of mercury, and especially MeHg, among various abiotic and biotic compartments is a concern when it threatens the health of aquatic biota or humans.

The mercury cycle in aquatic ecosystems is complicated because of the numerous possible mercury species that are present, and by variables affecting speciation. In the last decade, there was a marked increase in our knowledge of environmental concentrations of mercury species because of the development of strict clean protocols for the sampling and improvements to the analysis of mercury and MeHg (Driscoll et al. 1994; Gill and Fitzgerald 1985; 1987). Typical total mercury concentrations in 'uncontaminated' lake water (epilimnetic) in the north central United States, northern Ontario, and Quebec are about 0.5-4 ng/L , with MeHg usually making up a small proportion of the total (Table 3).

Generally, the aquatic mercury cycle may be characterised by high atmospheric fallout, removal by deposition to sediments, re-emission to the atmosphere as elemental Hg (Hg^0), methylation and demethylation transformation processes within the lake or watershed (Watras et al. 1994; Watras et al. 1995; Figure 4) and exchanges through tributaries and groundwater (runoff and

seepage; Hurley et al. 1994). Some portion of the total mercury pool is accumulated by biota, with biomagnification of MeHg in higher trophic levels of food webs.

Atmospheric fallout directly provides most of the mercury to watersheds that are uninfluenced by point source or high natural source contamination (Figure 4). Wet and dry fallout of mercury to lakes occurs largely as inorganic mercury (mainly Hg^{2+}; Winfrey and Rudd 1990), with insignificant inputs of MeHg (Bloom and Watras 1989). Sorensen et al. (Sorensen et al. 1990) found that direct wet deposition to the lake surface accounted for an average of 60% of the mercury in lake sediment. Long-range atmospheric transport of mercury results in mercury deposition to environments thousands of kilometres from emission sources, although about half of the fallout originates within 1000 km (EPMAP 1994).

In surface waters, mercuric ions (Hg^{2+}) may be reduced microbially to form elemental mercury (Hg^{o}; Ramamoorthy et al. 1983). Photochemical reduction may also occur with exposure to UV_B in sunlight (Amyot et al. 1994). Most surface waters are oversaturated with Hg° and because it is volatile, it is likely to be re-emitted to the atmosphere (Vandal et al. 1991; Driscoll et al. 1994). Small amounts of MeHg may also volatilise (especially dimethylmercury), but Hg^0 is by far the largest component of the total gaseous mercury concentration in air (Schroeder et al. 1991). Re-emissions are important sources of mercury to the global cycle and can result in oxidation to Hg^{2+} and re-entry to surface waters in association with precipitation or dry fallout (Figure 4).

Oceanic production and subsequent emission of Hg^0 is a particularly important facet of the global Hg cycle. This is especially true near ocean margins (Cossa et al. 1996). The oceans receive about 90% of their mercury as Hg^{2+} from wet and dry atmospheric deposition. Hg^0 forms about 5 to 30% of the total Hg in ocean waters (Mason et al. 1994). Biological production of Hg^0 has been reported in the open ocean, coastal mesocosms and in permanently anoxic fjords in Norway (Vandal et al. 1991). While re-emissions of the volatile Hg^0 result in a loss of reactive Hg species (Hg^{2+}) from a location where they might become methylated, the continual recycling of Hg back to the atmosphere prolongs the impact of anthropogenic Hg on aquatic systems. This is because while most of the Hg deposited to the ocean is recycled to the atmosphere, the terrestrial environment and freshwater ecosystems ultimately become the principal atmospheric Hg sinks (Mason et al. 1994).

Watersheds tend to be net sinks of mercury, with the amount of mercury retained often depending on the proportion of wetlands in the watershed (Mierle 1995). Seepage and runoff of mercury associated with particulates or dissolved organic matter (i.e., DOM; DOC; humic substances) from watersheds may introduce both inorganic and methylated forms of mercury to lakes and rivers. In seepage lakes (no surface inflow), essentially all MeHg is formed within the lake (Watras et al. 1994). In drainage lakes, significant inorganic mercury or MeHg may be transported from the drainage basin to the lake complexed with dissolved organic carbon (DOC)[15] or particulate matter (Lee and Hultberg 1990; Lee and Iverfeldt 1991), thereby supplementing direct atmospheric sources and in-lake net methylation.

[15] Often measured as colour.

Numerous studies have indicated the importance of organic carbon in binding and transporting mercury compounds (Ramamoorthy and Kushner 1975; Lee and Iverfeldt 1991; Mierle and Ingram 1991; Miskimmin 1991; St.Louis et al. 1994). The export of mercury from watersheds to lakes is closely tied to the export of humic material (DOC; Mierle and Ingram 1991). The quantity of Hg brought to a lake appears directly proportional to the amount of carbon leached from the surrounding soils in the drainage basin. More mercury and methylmercury are exported downstream from watersheds dominated by wetlands because of complexation with DOC (Mierle and Ingram 1991; Engstrom et al. 1994; Rudd 1995). Hurley et al. (1995) found a positive correlation between the percent wetland in the watershed and MeHg yield to rivers. In northern Ontario, even though upland watersheds without wetlands yielded more total mercury per area to lakes, high DOC wetland portions of watersheds yielded 26-79 fold more MeHg than uplands (St.Louis et al. 1994). Substantial increases in mercury and MeHg may occur when groundwater discharges through wetlands into streams or emerges to form rivulets that drain across the surface of wetlands (Krabbenhoft et al. 1995). In general, more mercury enters lakes that have a large drainage basin relative to the lake area. The same relationship to high $A_{Dr.}/A_{lake}$ has been associated with increasing mercury levels in some fish (Ontario; Suns and Hitchin 1990).

Once in surface waters, mercury may enter sediments, be transformed (e.g., methylated or demethylated) and/or be transferred to biota. MeHg has affinity for organic matter and biological tissue[16] because of its strong attraction to sulphydryl groups in proteins, thus it may become preferentially concentrated in fish and other aquatic biota. Most mercury binds to particulates and is quickly deposited to sediments, where it may be buried or transformed. Of all of the binding sites, particulates are considered to be most significant. Mercury binding to particulates may result in the efficient removal of mercury from the water column by sedimentation. Inorganic mercury (as $HgCl_2$) and to a lesser extent MeHg, bind quite strongly with suspended particulates (K_{sed}; Table 1). After deposition, mercury complexes with sulphide (as HgS), or adsorbs on organics, clays and Fe or Mn oxides (Miller 1975; Jackson 1989). As in humic horizons of soils, Hg is quite stable in lake sediments, being strongly bound to organic matter, with only minor diagenic remobilization after deposition (Schetagne et al. 1999). Some evidence suggest that by binding to dissolved organic material in the water column, mercury increases its persistence and resistance to sedimentation (Sorensen et al. 1990).

The deeper water near the sediment-water interface in lakes is often enriched in mercury compared with other zones in the lake. Because particulate-associated mercury concentrations were higher in the deeper layers than the shallow layers of stratified lakes, Watras et al. (1994) concluded that allochthonous (source outside the lake) Hg was scavenged by particles in the upper waters and transported down with settling particulates. The alternative was that deeper waters were enriched in particle-associated mercury because of resuspension from bottom sediments. Resuspension can occur due to turbulence from water currents, or bioturbation. In sediments, tubificids, clams, chironomids, other invertebrates and even fish have been known to reintroduce mercury to the water column by disturbing the sediments (Andersson et al. 1990). Porewater diffusion (movement through sediments) of mercury is undocumented for many types of lakes. Measurements of sediment porewater in a seepage lake indicated that diffusional

[16] MeHg has relatively low log K_{ow} values of 1.6 ± 0.2 (Major and Rosenblatt 1991) and 2.5 (Halbach 1985).

movement of total mercury averaged 1.4 X 10^{-8} ng/cm^2/s (concentration gradient of 15-35 ng/L; Hurley et al. 1994). Hurley et al. (1994) considered that the diffusional flux from sediments to the water column was minor compared to redissolution of recently fallen particulate material at the sediment surface. Compared with other metals like iron, mercury release from bottom sediments is not nearly as highly redox-dependent (Hurley et al. 1991).

Seasonally, mercury in the water column is lower in winter than in summer because frozen lakes are sealed off from atmospheric sources of mercury and most particulate settling has occurred (Watras et al. 1994). There is also a seasonal component to methylmercury cycling. MeHg concentrations in water were found to be higher in the late summer than at other times of the year in both rivers (Hurley et al. 1995) and lakes (Watras et al. 1995). Ramlal et al. (1993) observed the same seasonal trend for measured net methylation rates in sediment cores from northern Ontario. MeHg concentrations were highest by late summer because of the high bacterial activity associated with higher temperatures as the summer season progressed, causing MeHg to accumulate. Methylation but not demethylation was stimulated by warm summer temperatures; while the opposite was true in cold winter conditions (Ramlal et al. 1993). In general, net MeHg production in lakes is increased by factors that enhance bacterial activity, and total Hg is reduced when lakes are sealed off from atmospheric sources.

Reviews of the aquatic mercury cycle may be found in Driscoll et al. (1994), Zillioux et al. (1993), and Morel et al. (Morel et al. 1998). Also, a mathematical model of mercury cycling in lakes was developed by Hudson et al. (1994).

4.2 Mercury Methylation and Demethylation

Before the mid-1960s, inorganic mercury was believed to be relatively inert in the environment. Then it was demonstrated that inorganic mercury could be methylated by microorganisms to form MeHg, which was biomagnified in food webs (Wood et al. 1968; Jensen and Jernelov 1969). The production of methylmercury is a balance between methylation and demethylation. Wherever MeHg is detected in the environment, methylation has exceeded demethylation.

Because methylation and demethylation always co-occur, the balance between the two in the aquatic ecosystem is called net MeHg production. Several factors influence the rate of net MeHg production in water. These include the concentration of Hg^{2+}, composition of the microbial population, nutrient and mineral substrate, pH, temperature, redox potential, dissolved and particulate organic matter, iron and sulphate. Methylation and demethylation occur through both biotic and abiotic processes. Historically, microbial activity was thought to dominate the net MeHg production, however, there has been a lack of research on abiotic processes. Some recent work suggests that the significance of abiotic processes, particularly for methylation, has been underestimated.

Biotically, MeHg is formed through the microbial transfer of methyl groups (CH_3) to inorganic mercury (e.g., Hg2+ transformed to CH3Hg+; Robinson and Tuovinen 1984). Early studies reported that micro-organisms produced MeHg under anaerobic conditions (Jensen and Jernelov 1969). Subsequently, aerobic methylation was demonstrated (Rissanen et al. 1970; Matilainen and Verta 1995). It is now widely accepted that methylation occurs in both aerobic and

anaerobic environments (water column and sediments), but in most lakes MeHg at the surface originates from the anoxic environment below (Furutani and Rudd 1980; Ramlal et al. 1985; Xun et al. 1987; Morel et al. 1998).

Model and laboratory studies demonstrate clearly that anaerobic sulfate-reducing bacteria (sulfidogens) are primary methylators of mercury in marine and freshwater sediments (Compeau and Bartha 1985); however, some questions remain under natural, field conditions. For example, high concentrations of sulphate (>200-500 μM), such as those found in marine and estuarine waters, inhibit methylation by sulfidogens (Morel et al. 1998). Moreover, sulphate reducers make up only a small proportion of the microbial community in most freshwater lakes, where methanogens are the dominant anaerobic bacteria (Gilmour et al. 1992). Co-populations of sulfidogens and methanogens in low-sulphate anoxic freshwater sediments may produce conditions favorable to Hg^{2+} methylation (Pak and Bartha 1998a). The type or species of micro-organism responsible for methylation in aerobic water columns and surface sediments has not been identified (Gilmour and Henry 1991; Gilmour et al. 1992).

Many micro-organisms produce methylcobalamin, a non-enymatic methylation agent capable of transferring a methyl group to an inorganic mercuric ion. It can transfer groups as a carbanion (CH_3^-) and a methylradical ($CH_3^\bullet$) to produce mono- and di-methylmercury (D'Itri 1991), as follows:

$$Hg^{2+} \xrightarrow{CH_3B_{12}} \underset{\textit{Mono-MeHg}}{CH_3Hg^+} \xrightarrow{CH_3B_{12}} \underset{\textit{Di-MeHg}}{(CH_3)_2Hg}$$

The methylcobalamin-dependent methylation process is optimized at pH 4.7 (D'Itri 1991). Under anaerobic conditions, methylcobalamin is present, and the reaction requires ATP and hydrogen as the source of electrons (Wood et al. 1968; Robinson and Tuovinen 1984). Three major co-enzymes may participate in enzymatic methylation, but the process remains invalidated (D'Itri 1991). Methylation by naturally-occurring microorganisms is probably limited by processes that alter the availability of inorganic mercury (Ramamoorthy et al. 1982; Winfrey and Rudd 1990).

Abiotic methylation of mercury occurs potentially by three different types of methyl donors: methylcobalamin, methyltin compounds, and humic matters (Weber 1993). Of these, humic matter is the strongest candidate. Indeed, in laboratory studies methylation of Hg^{2+} occurs in the presence of high concentrations of humic matter (6380 mg/L; Nagase et al. 1982) along with metal ions (humics >170 mg/L; Lee et al. 1985). Abiotic methylation rates in freshwater sediments may be two to three times higher than biotic methylation rates (Zhang and Planas 1994)

Like methylation, demethylation of mercury occurs biotically and abiotically. Demethylation was first discovered by Furukawa et al. (1969), at about the same time as methylation was reported. Biotic demethylation is the degradation of MeHg involving the microbial cleavage of the carbon-mercury linkage followed by the reduction of Hg^{2+} to Hg^0 (Robinson and Tuovinen

1984). In general, demethylation of methylmercury in freshwater sediments occurs under anaerobic conditions; sulfidogens demethylate while methanogens catalyze demethylation of mercury (Oremland et al. 1991; Pak and Bartha 1998b). In marine sediments, demethylation occurs principally by sulfidogens under aerobic conditions (Oremland et al. 1991). In addition to metallic mercury, methane is a by-product of demethylation; in anoxic sediments methane is oxidized by methanotrophs to carbon dioxide (Pak and Bartha 1998c). The biochemistry and genetics of the plasmids and associated enzymes involved in mercury demethylation were thoroughly reviewed by Summers and Silver (1978), Robinson and Tuovinen (1984), Summers (1986) and Foster (1987).

Much less is reported on abiotic demethylation. In the presence of light, methylmercury underwent nitrate-induced photoxidation (Zepp 1987). More recently, Sellers et al. (1996b) reported that the photodegradation of MeHg in natural ecosystems was about 350 times greater than microbial degradation. It is not known whether photodegradation results in the production of the volatile Hg^0. In dim ambient light, Zhang and Planas (Zhang and Planas 1994) found no signficant demethylation in sterilized lake sediments. These results suggest that MeHg photodegradation may be an important process where light penetration is significant (such as in clear and/or shallow water).

The relative importance of a biotic vs. abiotic processes is a point of contention. Biotic processes are assumed often to be dominant, particularly in fresh waters; however, data on abiotic processes are limited and that which exists suggest that their significance has been underestimated (Weber 1993; Gilmour and Henry 1991).

A number of environmental variables affect the net production of methylmercury. Most factors that increase rates of bacterial activity will enhance MeHg production. Bacterial activity in freshwater and marine sediments increases with increasing temperature and available biodegradable organic carbon. Thus, methylation rates tend to be highest in surface sediments with freshly deposited organic matter, and in warm shallow sediments where abundant bacterial activity takes place (Callister and Winfrey 1986; Ramlal et al. 1986; Winfrey and Rudd 1990).

4.3 Factors Affecting Partitioning and Speciation

The partitioning of mercury between "dissolved" and "particulate" phases is directly related to aqueous speciation and to lake chemistry. Partitioning of a given species is the outcome of the competition for that species among ligands in solution (truly dissolved), small particles called colloids, and organic or inorganic material. Speciation describes whether the form of mercury is an organic form (e.g., MeHg), or is one of the many inorganic forms (e.g., $HgCl_2$, HgS, etc.). This document will only discuss MeHg and a few of the important inorganic forms. Full reviews and examples of mercury speciation research may be found in Gill and Bruland (1990), Aceto et al. (1995e), and in several papers within the book, "Mercury Pollution: Integration and Synthesis" (Watras and Huckabee 1994).

The effect of pH on mercury speciation is very complex. Higher rates of MeHg production are associated with decreasing pH, but pH does not directly affect rates of methylation and demethylation (Xun et al. 1987; Miskimmin et al. 1992; Pak and Bartha 1998b). The relative

concentration of all inorganic species as well as total solubility changes markedly with pH. A relationship between pH and DOC may be important in regulating the availability of inorganic mercury for methylation. Although organic matter in sediments enhances bacterial activity and, dissolved and particulate organic carbon increase the amount of mercury and methylmercury in an aquatic system by facilitating transportation (section 4.1), dissolved and particulate organic carbon in the water column can bind mecury, making it unavailable for methylation (Miskimmin et al. 1992; Matilainen and Verta 1995). Reduction in pH changes the character of DOC by increasing protonation of anions and thus desorbing metals (Davis et al. 1985). If binding of inorganic mercury to some forms of DOC is reduced by low pH, more mercury may be available to DOC-associated microorganisms for methylation (Miskimmin et al. 1992).

In natural environments, redox conditions and chloride ions influence Hg speciation and methylation (Gill and Bruland 1990). In well-aerated water (redox ≥0.5V), mercuric (+2) species will predominate (Alberta Environmental Protection 1992). Abundant MeHg may be produced by anaerobic (e.g., Ramlal et al. 1985), as well as aerobic bacteria. At low chloride concentrations ($<10^{-3.5}$ M), the speciation of inorganic Hg is dominated by three uncharged complexes, $HgCl_2$, HgOHCl, and $Hg(OH)_2$, with $Hg(OH)_2$ being the most abundant. As the chloride concentration increases to about $10^{-2.3}$ M, a value typical of high chloride lake water, the percent of $HgCl_2$ rises to its maximum while concentrations of HgOHCl and $Hg(OH)_2$ decrease to only a few percent of the total inorganic Hg (Mason et al. 1996; Alberta Environmental Protection 1992).

Sulphate levels are important to mercury speciation, as well. Mercury methylation rates are correlated positively with rates of bacterial sulphate reduction in marine sediments (King et al. 1999) as well as freshwater sediments (Gilmour et al. 1992; Gilmour and Riedel 1995). Yet, at concentrations of sulfate (SO_4^{2-}) greater than 200-500 µM, production of sulphide (H_2S) by sulphate reducers inhibits methylation of Hg^{2+} (Pak and Bartha 1998a). Within anoxic zones, inorganic mercury forms strong aqueous complexes with sulphide and precipitates as HgS, a form unavailable for (Craig and Bartlett 1978; Leermakers et al. 1993). Sulphide controls speciation of both Hg^{2+} and MeHg because it outcompetes humic acid (DOC) by factors of 10^4 for CH_3Hg^+ and 10^{18} for Hg^{2+} (Hudson et al. 1994). Hurley et al. (1994) completed extensive calculations of the equilibrium speciation of mercury and sulphur at a range of redox and pH levels.

Potential sulphide binding that reduces bioavailable mercury may be particularly important in the saline lakes (>3 g/L salt) of Alberta and Saskatchewan which have some of the highest sodium sulphate concentrations in the world (Na2SO4 >125 M; Waiser and Robarts 1995). HgS precipitation combined with high pH (associated with low methylation rates), decreased methylation with increasing salinity (Blum and Bartha 1980) and potential Hg^0 re-emissions (high from shallow, wind-mixed waters) makes significant MeHg bioaccumulation an unlikely problem in prairie saline lakes. Perhaps an analogous situation is in the high pH, high-alkalinity desert lakes in California, where very little or no MeHg was detected using highly sensitive analytical methods (0.5 pg/L detection limit; Gill and Bruland 1990).

Methylmercury formation is reduced with increasing salinity in estuarine sediments (Blum and Bartha 1980). The bicarbonate component of seawater is thought to slow mercury methylation

under both aerobic and anaerobic conditions (Compeau and Bartha 1983). Along a salinity gradient in the lower Hudson River, methylation rates decreased down river with increasing salinity because the H_2S formed in reducing sulphate-rich environments precipitates mercuric ions, which are unavailable for methylation (Gilmour and Capone 1987). In an experiment that removed the sulphide effect on Hg^{2+} availability, organic matter content and not salinity, was the major controling factor for methylation rates (Choi and Bartha 1994).

5.0 Bioaccumulation of Mercury and Methylmercury in Aquatic Organisms

Mercury compounds including MeHg may be taken up by organisms directly from water or through their diet. Uptake directly from water is the result of dissolved mercury adsorption or absorption through the body surface and respiratory organs. Uptake via food is based on mercury transfers through the digestive tract. The relative importance of either pathway depends on trophic level of the organisms, duration and intensity of exposure, Hg species, and environmental factors (Zillioux et al. 1993). A few studies demonstrate that mercury may be methylated, demethylated or reduced by intestinal contents of both fish and mammals (Ludwicki 1990; Rudd et al. 1980)

Organisms at lower trophic levels usually contain the lowest proportion of total mercury as MeHg and uptake is primarily a passive process occurring by adsorption to or absorption within the cell (Beckvar et al. 1996). Aquatic plants contain the lowest MeHg as a percentage of total mercury, typically less than 50%. Invertebrates often contain about 50% MeHg and 50% inorganic mercury (Hildebrand et al. 1980). Organisms higher in the food chain, like piscivorous fish (e.g., walleye, lake trout), aquatic birds (loons, herons), piscivorous mammals (mink, otters) and marine mammals contain a very high proportion of THg as MeHg in muscle tissue (90 – 100%).

Most piscivorous fish have essentially all MeHg in their muscle tissue (Bloom 1992). The main route of uptake for MeHg is consistent with a number of persistent organic chemicals like DDT, PCBs or dioxins. Once debated, it is now generally accepted that uptake through diet sources is the most important route of uptake for fish (Rodgers 1994) and, of course, for wildlife consumers of fish. MeHg accumulates in organic tissue because of its affinity with sulphydryl groups, the relative ease with which it passes through the digestive wall and slower depuration rate relative to inorganic mercury (Saouter et al. 1993).

5.1 Aquatic Plants

Direct passive adsorption of mercury is the critical pathway for phototrophic organisms like macrophytes and algae. Small algal cells may accumulate more mercury than large ones because of the higher surface area to volume ratio for small cells compared to large cells. MeHg is preferentially sequestered within algal cytoplasm while inorganic Hg is retained in the algal outer membrane (Mason et al. 1995).

Because phytoplankton cells continually divide, the partitioning of Hg into live cells is a balance of uptake, depuration and growth dilution (Davies 1974). This is fundamentally different from adsorption of Hg onto detrital or inorganic particles. Plankton may potentially accumulate more Hg than does detritus because the living cells have both passive and facilitated transport mechanisms, as well as intracellular ligands that sequester Hg (Hudson et al. 1994). The estimated bioconcentration factor for the phytoplankton species, *Isochrysis galbana*, is 100 000 (Davies 1974).

The reported data on mercury uptake by aquatic macrophytes are sometimes contradictory, with some studies indicating that mercury accumulation takes place primarily in roots while others indicating accumulation is primarily through rhizomes or shoots. Given that rooted macrophytes accumulate the majority of their nutrients through their roots and the fact that mercury concentrations are higher in sediments than in water, it may seem intuitive that more mercury would be accumulated through the roots from sediments. Jana (1988) found twice as much mercury in roots compared to shoots of a water hyacinth (*Eichhornia crassipes*) and Coquery and Welbourn (1995f) found seven times more total mercury in the roots than in shoots of a submergent perennial in Ontario. With MeHg, while high accumulation was measured in leaves, stems and roots of the waterweed, *Elodea densa*, root absorption was reported to be the dominant direct route of uptake, and the leaves were the principal storage organ (Maury et al. 1988).

In lakes of northern Quebec, Lucotte et al. (Lucotte et al. 1999) found that *Nuphar variegatum* accumulated less Hg than other aquatic plant species growing nearby and accumulates as much Hg in its leaves as in its roots. Moreover, Hg concentrations in any part of *N. variegatum* were always an order of magnitude lower than those of the sediments in which they grew, suggesting that the plant takes up Hg from the water column through its leaves and stems, with limited transfer from the sediments to the rhizosphere.

Environmental variables can affect the bioaccumulation of mercury by plants. Coquery and Welbourn (1995f) found that total mercury in macrophyte roots was related to mercury in sediments, but only after correction for sediment organic content. If organic content of the sediments was high, mercury concentrations of the sediments could also be elevated without significant uptake by the plant. In that study, concentrations of mercury in plants were greater than the concentration in sediments, and mercury in roots was consistently higher than in the shoots of the pipewort, *Eriocaulon septangulare* (0.22 μg/g and 0.03 μg/g dw, respectively). Increasing temperature and photoperiod exerted a strong positive influence of MeHg accumulation to macrophytes, with the effects amplified when both are increased concurrently (Elodea densa; Maury-Brachet et al. 1990). Differences in accumulation of mercury may also depend on the species of plant, type and amount of complexing ligands and seasonal growth rate changes (Czuba and Mortimer 1980).

Experimentally, the presence of DOC (in the form of humic acid or EDTA) significantly suppressed dissolved mercury uptake by duckweed (Mo et al. 1989). However, in a field survey of 34 Precambrian Shield lakes in Ontario and Quebec, algal mercury concentrations showed a significant positive correlation with DOC and with fish mercury concentrations even though mercury in the water was at low to undetectable concentrations (Stokes et al. 1985). The difference between experimental and field results is probably because while DOC may suppress uptake, high DOC in lakes often contain more mercury from terrestrial/wetland sources, as discussed elsewhere in this report.

Bioaccumulation is proportional to dissolved concentrations assuming that the rates of uptake by plants are similar for inorganic and organic forms (Mortimer 1985). Aquatic plants are known to bioconcentrate mercury and MeHg up to several thousand times the water concentration (per g dry wt.). Four genera of submergent macrophytes had bioconcentration factors (BCFs) for total mercury ranging from 900 - 3300 (concentration in plant : water concentration, where the

experimental water concentration was 0.28 mg/L) (Mortimer 1985). Calculations from Jana's (1988) data result in similar BCFs of 670 for an alga, and 800 for water hyacinth.

Chloride concentration and pH influenced uptake of inorganic mercury by a marine diatom. Uptake rates were low in seawater and low-chloride freshwater with neutral pH (Mason et al. 1996). MeHg uptake rates were high in high-chloride waters and were not influenced by pH. The uptake rate of MeHg only became limited when very low chloride concentrations decreased the concentration of CH_3HgCl. Accumulation of elemental and dimethylmercury were insignificant (Mason et al. 1996).

5.2 Invertebrates

Mercury is accumulated by aquatic invertebrates directly from water (including porewater), and through ingestion of contaminated food. Mercury may be bound on the outer membranes or chitinous exoskeleton, absorbed within gut contents, incorporated into body tissues and excreted. Mercury bound to outer membranes or exoskeletons may be lost with moulting (Zauke 1977), but it is not considered an important depuration pathway for some crustaceans (Fowler et al. 1978). Also, egg-carrying females have higher concentrations than other individuals because MeHg is readily transferred across lipid bilayers into eggs. MeHg is removed from females with their eggs. The rate of MeHg bioaccumulation from water has been shown to be related to metabolic rate in chironomids (Visman et al. 1995).

Depuration (Hg loss or excretion from organisms) is much slower for MeHg than for inorganic Hg. Asiatic clams (*Corbicula fluminea*) were exposed to inorganic and MeHg for 14 days, and depuration was followed for 30 days. The kinetics of uptake and depuration were tested at 12 and 24^{o}C and at pH levels of 6.0 and 8.0. In 30 days, 30% of Hg^{2+} and negligible MeHg was lost from the clam soft tissues. After a further 120 days, 40% depuration of MeHg occurred (Inza et al. 1998). Temperature and pH made no difference to depuration.

Invertebrates tend to have a lower percentage of MeHg than fish partly because the sources of mercury to which invertebrates are exposed usually comprise relatively low percentages of MeHg, and because any forms of mercury taken up are either associated with gut contents (diet uptake) or the exoskeleton/gills (water exposure) rather than absorbed into tissue. There may be a wide range of the proportion of Hg that is MeHg in invertebrates reflecting the range of feeding strategies (herbivory, carnivory) and short length of exposure (Beckvar et al. 1996). For example, Tremblay (Tremblay 1999) reported the mean proportion of MeHg in sixteen Quebec lakes increased from detrivores (35-45%; dipterans, ephemeropterans, trichopterans) to predacious species (70-85%; notonectids, coleopterans, odonates).

Sediment-associated invertebrates or 'benthos' (e.g., chironomids, amphipods, mussels) generally contain more mercury than those in the water column (e.g., *Daphnia*). Mercury uptake by sediment-dwelling organisms is likely favoured by the combination of higher mercury concentrations in their diet as well as in their physical environment (Parkman and Meili 1993). Gagnon and Fisher (1997) suggested that the most important source of uptake for mussels changes from water to particulates depending on the concentration and composition of

suspended particles. While laboratory experiments suggested water was the major route of mercury uptake for *Mytilus edulis*, under estuarine conditions, when inorganic Hg on suspended sediments was up to 80 times higher than dissolved Hg, the particulate pathway was dominant (King and Davies 1987). Malley et al. (1996c) found that uptake of MeHg by the freshwater mussel, *Pyganodon grandis,* was higher after reservoir flooding experiments when suspended particulate concentrations increased substantially.

The proportion of accumulated mercury taken up either from water or food depends on the degree of contamination of each compartment, as well as on the chemical forms of mercury present. Saouter et al. (1993) measured the distribution of mercury and MeHg in a common mayfly, *Hexagenia rigida,* after a nine-day exposure in laboratory microcosms containing both water and sediment. Mercury was added either to the overlying water or to the sediment. MeHg was taken up sixty times more readily than inorganic Hg (as $HgCl_2$) when it was added to sediments, and twice as readily when added to overlying water. Both forms of mercury tended to accumulate in the gut when taken up through ingestion of sediments, whereas they accumulated on the gills when taken up from water (Saouter et al. 1993).

Water quality can influence the uptake of mercury by aquatic invertebrates. For zooplankton, total mercury content varies positively with mercury concentration in water (Sorensen et al. 1990). For benthic invertebrates, mercury concentrations are not correlated with sediment mercury concentrations (Kristensen 1982; Parkman and Meili 1993). Higher concentrations of MeHg in zooplankton have been reported in acidified lakes (Bloom 1992), especially if they were also brown-water (high DOC) drainage lakes (Westcott and Kalff 1996). Laboratory experiments confirmed that increased uptake of inorganic Hg (as $HgCl_2$) and MeHgCl occurred with decreasing pH (Saouter et al. 1993) and that the diffusional fluxes of mercury over lipid bilayers were higher at pH 5.0 than at pH 8.5 (Boudou et al. 1991).

Some field evidence suggested that DOC supressed the uptake of both inorganic Hg and MeHg by invertebrates based on results that showed a negative correlation of all forms of Hg in zooplankton with DOC in seepage lakes (no terrestrial sources of Hg; Watras et al. 1994; Back and Watras 1995). Suppression of both forms of Hg was not supported experimentally, where aquatic humus (DOC) suppressed the uptake of MeHg but not inorganic Hg, to *Daphia magna*. Total Hg tissue concentrations in *D. magna* increased in proportion to DOC concentrations (3 - 10 mg C/L) typically found in low alkalinity lakes, but MeHg showed the opposite trend (Monson and Brezonik 1999). Monson and Brezonik (1999) separated food and water uptake, finding that inorganic Hg readily desorbed from both food particles and DOC resulting in transfer to the daphnid. MeHg bound to food was readily absorbed, but DOC-bound MeHg was not.

Higher mercury concentrations are found in invertebrates that reside in the deepwater compared to shallow sediments, particularly in anoxic conditions (Sarkka 1979). Mercury concentrations may be ten times higher in deepwater than nearshore taxa (Parkman and Meili 1993). The sediment-water interface is the zone of highest methylation rates, steep oxygen gradients, and high bacterial activity (thus methylation potential), therefore minor differences in feeding strategy may result in large differences in Hg bioaccumulation.

Because of the scarcity of MeHg measurements done on water, plants and invertebrates within individual studies, few bioconcentration factors and/or bioaccumulation factors for MeHg are available for invertebrates. Tremblay (Tremblay 1999) found a MeHg biomagnification (BMF = transfers from diet only) of about three between the two adjacent trophic levels of phytoplankton and zooplankton. BCFs (bioconcentration is transfer from water only) for zooplankton were 10 to 100 times higher for MeHg compared to inorganic Hg (based on wet weights; Watras and Bloom 1992). Concentrations of MeHg and THg were higher in all compartments in the experimentally acidified basin (pH 4.7) of Little Rock Lake, WI. The BCFs ([zooplankton]/[water]) were an order of magnitude higher for MeHg than for inorganic Hg. Bioaccumulation factors (BAFs = transfers from diet and water) were the same for inorganic Hg in both the acidified and reference basins (BAF=1.9); BAFs for MeHg were 3.6 and 5.0 in the acidified and reference basins, respectively (Watras and Bloom 1992). These data supported the hypotheses that the uptake of MeHg by zooplankton was more efficient than inorganic Hg, and that uptake of MeHg is proportional to supply regardless of lake acidification.

In marine invertebrates, percentages of MeHg relative to THg concentrations were less than 1% for the polychaete *Nereis succinea* (Luoma 1977), 10% in copepods, mussels and shrimp (Horvat 1991), 10 to 100% in the cockle (Mohlenberg and Riisgard 1988), 16% in urchin gonads (Eganhouse and Young 1978), 87% in crab muscle (Eganhouse and Young 1978), and 100% in red rock crab, Dungeness crab and spot shrimp (Bloom 1992). Moulting is not considered an important depuration pathway in shrimp and mussels (Fowler et al. 1978).

5.3 Fish

Fish accumulate Hg from both their diet and from the water column (Sorensen et al. 1990). The accumulation of mercury by fish from water occurs via the gill membranes. Diet is the most important route by which fish take up MeHg. Essentially all mercury found in muscle tissues of piscivorous freshwater, whether from water or diet sources, is MeHg (approximately 99%, Bloom 1992). Early studies that reported less than 95% MeHg of the total in large predatory fish may have been limited by their analytical methodology or problems with homogeneity of subsamples (Bloom 1992). Somewhat lower percentages may be expected in young non-piscivorous fish. For example, young finescale dace (*Phoxinus neogaeus*) that fed on benthic insects only contained 71 - 89% MeHg in muscle tissue (Bodaly and Fudge 1999).

Liver and kidney in fish tend to have higher percentages of inorganic mercury than muscle tissue, with the percentages varying by organ and species (Riisgard and Hansen 1990). The liver and kidney are also the main depuration pathways for mercury, with MeHg half lives in fish ranging from one to greater than three years (McKim et al. 1976; Riisgard and Hansen 1990).

While inorganic mercury is always more abundant in natural ecosystems, it is absorbed by fish much less efficiently than MeHg, and if taken up, is eliminated more rapidly than is MeHg (Huckabee et al. 1979; Boudou and Ribeyre 1985; Trudel and Rasmussen 1997). This occurs because the digestive wall is much more permeable to MeHg than to inorganic mercury (Boudou and Ribeyre 1985), allowing MeHg to be readily transferred to other tissues. A recent finding is that waterborne inorganic Hg may reach specific areas of fish brains and the CNS via axonal

transport in neurons, allowing Hg to circumvent the blood-brain barrier (Rouleau et al. 1999). It is unknown whether MeHg may follow a similar route to reach the brain.

Harris and Snodgrass (1993) modelled the uptake of MeHg from water to yellow perch and walleye, as well as uptake through two types of diet. MeHg concentrations were modelled with realistic concentrations for water (0.05 ng/L), for zooplankton (0.033 ng/g ww) and for forage fish (0.30 ng/g ww). Ninety-nine percent of MeHg was accumulated from the diet over several years, taking about one year to reach equilibrium when the diet changed from zooplankton to forage fish. Even when the water concentrations were increased to 'unrealistically high' levels (0.3 ng/L MeHg), and allowing for uncertainty factors, uptake from water was always less than 20% of the total. These results were confirmed experimentally, in that no more than 15% of MeHg uptake by finescale dace was attributable to passive uptake from water through the gills (Hall et al. 1997).

Harris and Snodgrass' simulations indicated that excretion was more effective in young fish than from older fish, suggesting that remedial actions may be more effective in reducing mercury in younger fish (may take 10 years to excrete 50% for a 10-yr-old walleye; Harris and Snodgrass 1993). Harris and Snodgrass based their calculations on an assumed allometric exponent of -0.6 for Hg elimination. Trudel and Rasmussen (1997) recently calculated the exponent to be closer to -0.2, which would mean that smaller fish excrete MeHg more slowly than the Harris and Snodgrass model predicts. In general, MeHg is excreted very slowly relative to uptake for most fish. MeHg is not eliminated rapidly enough to prevent a net accumulation as long as exposure is continued.

Large, adult predatory fish tend to have the highest MeHg concentrations. While this lends support to the biomagnification through diet argument, longer exposure, and the slower excretion by larger fish described above, as well as other factors such as differences in food consumption rates, food conversion efficiencies, and fish metabolic rates may also be important (Canada-Manitoba Mercury Agreement 1987). The assimilation efficiency of MeHg from food to fish is often estimated at 80% (Norstrom et al. 1976; Harris and Snodgrass 1993; Trudel and Rasmussen 1997).

Reservoirs may have fish with elevated mercury concentrations because the flooded organic matter and associated increased bacterial activity enhances MeHg production (Canada-Manitoba Mercury Agreement 1987; Ramlal et al. 1987). Any bioavailable MeHg may then be bioaccumulated. Overviews of the mercury problem in specific reservoirs are found in Bodaly and Hecky (1979), Verta et al. (1986b), Jackson (1987), Canada-Manitoba Mercury Agreement (1987), Verdon et al. (1991), Anderson et al. (1995), Rodgers et al. (1995), Verdon and Tremblay(Verdon and Tremblay 1999), and Schetagne and Verdon (Schetagne and Verdon 1999a). A model of fish mercury levels as related to physical characteristics of Canadian reservoirs was developed by Johnston et al. (1991), and mercury accumulation by fish in an experimental reservoir was examined by Bodaly and Fudge (Bodaly and Fudge 1999).

Numerous chemical and physical variables of surface waters determine the potential for fish bioaccumulation of mercury (Environment Canada 2002). In particular, low pH (<6), low alkalinity (acid-neutralizing capacity 50 µeq/L or less), and low calcium (<5 mg/L) lakes are

associated with elevated mercury concentrations in fish (Grieb et al. 1990; Spry and Wiener 1991). Studies of the effect of pH on uptake by fish show conflicting results. In controlled laboratory studies, Rodgers et al. (1987) found no difference in accumulation of MeHg by walleye and trout among three pH treatments (5, 6 and 7); whereas Drummond et al. (1974) found greater MeHg accumulation by brook trout at pH 6 than 9.

Dissolved organic carbon (DOC) appears to prevent mercury uptake by organisms from water. In seepage lakes mercury concentrations in fish are negatively correlated with DOC concentration (Watras et al. 1994). In drainage lakes, however, significant inorganic mercury or MeHg may be transported from the drainage basin to the lake complexed with dissolved organic carbon (DOC) or particulate matter. High Hg concentrations in fish may occur in drainage lakes that have high DOC (Grieb et al. 1990; Schetagne and Verdon 1999a).

The efficiency of MeHg uptake across the gills of fish is inversely correlated with the calcium ion content of water (Rodgers and Beamish 1983). The water hardness relationship (calcium) has particular relevance in the prairie provinces, where many lakes and rivers are high in calcium and pH. This may explain why fish in many Alberta reservoirs do not have elevated mercury concentrations even though other water bodies in western Canada do. Uptake would be reduced in these high calcium, high pH water bodies compared with softwater lakes. Selenium may effectively block the accumulation of MeHg in soft, as well as harder, more alkaline waters (Southworth et al. 1994; see review by Pelletier 1985). Accumulation of MeHg by fish in temperate waters is greatest during summer when the feeding and metabolic rates of fish are most rapid and when the production of MeHg is greatest (Spry and Wiener 1991).

MeHg may be passed from female fish to eggs. THg, MeHg and inorganic Hg were measured in eggs stripped from yellow perch carcasses from several lakes, with the finding that MeHg averaged >80% of THg, while maternal carcasses contained an average of 95% MeHg. The conclusion was that the perch were not using their eggs as a mode of excreting Hg, because the burdens of mercury in eggs of forty-eight perch averaged only 2% of the whole-body burden (Hammerschmidt et al. 1999). In northern Québec, both dwarf and normal lake whitefish, gonads were less concentrated in Hg than muscle. For example, mean THg concentrations of 0.42 µg THg/g (ww) were found in the gonads of dwarf and normal specimens from the Caniapiscau Reservoir, compared to mean concentrations ranging from 0.11 to 0.17 µgTHg/g in muscle tissue (Doyon et al. 1998).

5.4 Field-based Bioaccumulation Factors for Trophic Level 3 and 4 Fish

The bioaccumulation factor (BAF) for any given trophic level is defined as the ratio of a contaminant concentration in fish tissue divided by the concentration of dissolved contaminant in the water column. The BAF differs from the bioconcentration factor (BCF) in that BAFs represent the accumulation of MeHg in fish of a given trophic level from both direct uptake from water and from ingestion of contaminated organisms, whereas BCFs represent accumulation from water only.

MeHg concentrations increase with the age of the fish, which equates to the duration of exposure to MeHg. Accumulation increases as young fish grow and change their diets from lower trophic levels (such as aquatic invertebrates) to fish. Foraging fish that feed on lower trophic levels are considered trophic level 3, and piscivorous fish are trophic level 4 (U.S.EPA 1997c).

Until relatively recently, BAF values were typically available only from laboratory studies. A number of studies have either calculated, or permitted the calculation of BAFs from their data. A BAF of 667,000 was estimated for gizzard shad from Onondaga Lake (NY) from data reported by Becker and Bigham (Becker and Bingham 1995). The value was derived from an average concentration of 0.2 µg/g MeHg in shad (age classes 3-4 years) and 0.3 ng/L dissolved MeHg in the water. There is uncertainty in this BAF estimate as water MeHg concentrations were determined for a single season (summer) only.

A BAF of 1,460,000 for yellow perch from Lake Iso Valkjarvi in Finland was calculated from data reported by Rask and Verta (Rask and Verta 1995). Concentrations of total mercury in perch averaged 0.15 µg/g over a 3-year period (1990-1993). The mean concentration of dissolved MeHg in the epilimnion was 0.103 ng/L. The water concentrations were determined on a single day (8/24/93). All measurements were taken from the control basin of Iso Valkjarvi as the lake was partitioned for experimental liming to control pH.

BAFs for silversides and juvenile bass in Clear Lake, California were estimated from data tables providing cross-seasonal measurements for two years (Suchanek et al. 1993). BAFs were estimated from matched fish (THg) and water (dissolved surface MeHg) concentrations for each measurement for this period across four lake areas and up to five sampling locations for each area. The average BAF for silversides was 1,130.000 and for juvenile bass was 1,930,000, with an overall mean of 1,530,000.

A BAF of 4,170,000 for bloater in Lake Michigan was calculated by US EPA (U.S.EPA 1997a) from data reported by Mason and Sullivan (Mason and Sullivan 1997). A two-seasonal average (August/October, 1994) of MeHg in the surface waters was calculated to be 0.0104 ng/L (all MeHg assumed to be monomethylmercury). The average concentration of MeHg in bloater was calculated to be 0.0434 µg/g.

In their Report to Congress, the US EPA completed probabilistic simulations including sensitivity analysis for both modeled BAFs and the above field-derived BAFs (U.S.EPA 1997a). They recommended those developed from field data at each of trophic level 3 and 4, partly because they showed less variability than the modeled BAFs (used in the Great Lakes Water Quality Initiative prior to availability of field data). Also GLWQI recommended that when high quality field data were available, BAFs should be developed from those data.

The US EPA recommended the geometric mean of field-derived BAFs for trophic level 3 of 1,600,000 and for trophic level 4 of 6,800,000 (U.S.EPA 1997a). These values were used in Part I-3.3 to calculate a water concentration for MeHg that would be protective of relevant piscivorous wildlife at specific sites. Also refer to the TRG document for guidance on protecting piscivorous wildlife (Environment Canada 2002).

6.0 Environmental Concentrations

The environmental concentrations section of the MeHg Tissue Residue Guideline document (Environment Canada 2002) described mercury concentrations in aquatic plants, aquatic invertebrates, fish, amphibians, reptiles, semi-aquatic mammals, marine mammals, aquatic birds and non-biological media in Canada. Where MeHg concentrations for Canada were unavailable or scarce, information was supplemented with total mercury (THg) concentrations or with MeHg values from bordering states. For fish, measurements were typically for total Hg, although the majority of Hg in fish muscle is assumed to be MeHg (Bloom 1992). Rather than repeat the extensively tabulated data of the TRG document, the present document will use examples where necessary and provide additional information including total Hg measurements that were not cited in the TRG document.

The information contained in this chapter, Table 3 and the TRG (Environment Canada 2002) are examples only and are not intended to be exhaustive. Instead, readers are referred to such databases as are being developed by Environment Canada (Kent et al. 1998), and to those that have been compiled for many years by some of the provinces (e.g., British Columbia, Ontario, and Quebec) or through special projects (e.g., Fraser River Action Plan [FRAP], Northern River Basins Study Project [NRBS], and Northeast States and Eastern Canadian Provinces [NES/ECP] Mercury Study) .

6.1 Water[17]

Wet deposition can be a major source of Hg to lakes and tributaries (see Chapter 3). In the Canadian Shield, where direct wet deposition contributed 57% of the Hg to a small lake, concentrations in precipitation ranged from 2 to 28 ng Hg/L in 1987; the deposition rate was 10.2 µg $Hg/m^2/a$ (Mierle 1990; Mierle and Ingram 1991). Samples of rainwater taken in the early 1990s at the Experimental Lakes Area (ELA) in north-western Ontario contained average concentrations from 3.8 to 5.3 ng Hg/L for total mercury and from 0.017 to 0.049 ng Hg/L for MeHg (i.e., <1% of total Hg). In snow samples, levels of total Hg averaged 0.95 to 3.58 ng/L, while levels of MeHg ranged from 0.008 to 0.104 ng Hg/L, representing 0.8% to 2.9% of total Hg (St.Louis et al. 1994; St.Louis et al. 1995a). In 1995-1996, rain at St. Anicet, QC[18] contained total mercury concentrations ranging from 0.81 to 21.29 ng/L, with a mean of 6.58 ng/L (NESCAUM 1998). In Atlantic Canada, mean concentrations of mercury in 1996-1997 were 7.37 and 7.51 ng/L for Kejimkujik National Park, NS and St. Andrews NB, respectively (NESCAUM 1998). Mean concentrations of total Hg in precipitation collected from the Brunette River watershed (Lower Fraser Valley, BC) in 1997 contained relatively low levels of Hg (1 n/L) (Environment Canada 1999). Samples collected earlier, in 1993, from nine locations in the Fraser River Estuary contained no detectable levels of total Hg (detection limit of 50 ng/L) (Morse 1994).

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[17] Most of the available information about MeHg in water samples in Canada and elsewhere has been published relatively recently because analytical techniques that permit detection of low levels were developed only in the late 1980s (Bloom 1989a).

[18] At this site, most of the mercury was in particulate form.

The six-year trend of wet Hg deposition was monitored weekly from 1990 to 1995 at several sites in the northern U.S. (Minnesota, North Dakota, Michigan). This comprehensive study found levels of total Hg and MeHg to be comparable with the northwestern Ontario results discussed above. Average annual concentrations in wet deposition ranged from 5.5 to 19.5 ng/L total Hg, with MeHg comprising about 1.5% of the total (Glass and Sorensen 1999). While there was significant variation among the sites and seasons, the annual precipitation had an average concentration of 10.9 ng/L and a deposition rate of 7.4 μg Hg/m^2 per year, values that could reasonably be applied to nearby Canadian provinces.

In lake water samples, MeHg is typically a smaller proportion of total mercury than is inorganic mercury. In low alkalinity lakes, MeHg has been reported to represent 10-20% (Adirondacks, Driscoll et al. 1994), and <1-13.3% (lake inflows, Precambrian Shield of NW Ontario; St.Louis et al. 1994) of total mercury in water samples. An unusual case was for two moderately alkaline (up to 3 meq/L or 150 mg/L $CaCO_3$), high pH (8-9) lakes in California, where up to 89% of total Hg was MeHg (Gill and Bruland 1990).

Water samples from a variety of surface waters at the Experimental Lakes Area had average MeHg concentrations ranging from 0.03 to 1.38 ng Hg/L. The higher concentration was from a pond after experimental flooding as part of an experimental reservoir project (Kelly et al. 1995). The lower concentration (0.03 ng Hg/L) was for water flowing from an upland area that lacked wetlands, while water from watersheds dominated by wetlands had average levels of 0.626 ± 0.126 ng Hg/L for MeHg (St.Louis et al. 1994). Porewater from peat in wetlands contained 0.6 ng Hg/L MeHg, which was 10% of THg (Moore et al. 1995).

Within six weeks of the creation of an experimental reservoir at the ELA, MeHg concentrations in unfiltered and filtered water samples increased from a pre-impoundment mean of 0.1 to greater than 1.0 ng/L. Mean concentrations of particulate-associated MeHg increased from a pre-impoundment mean of 0.032 to greater than 300 μg MeHg/g dw (Paterson et al. 1998). In contrast to MeHg, flooding had very little effect on THg concentrations where the average levels were approximately 2.5 to 3 ng THg/L before and after reservoir creation (Kelly et al. 1997). The average post-flood percent MeHg was 32% (maximum of 73%) of THg, about seven times higher than pre-flood.

Water samples taken in the late 1970s from the mercury-contaminated Wabigoon/English River area near Dryden, ON reportedly contained 0.22 to 1.3 ng/L MeHg (Parks et al. 1989). While these values seem quite possible considering the high levels of mercury found in biota in the area, rigorous clean techniques for sampling and modern analytical methods were not developed at that time.

Total mercury levels in brown water streams in Ontario varied seasonally and correlated to colour (i.e., humic material) (Mierle 1990; Mierle and Ingram 1991). The highest levels of Hg, sometimes exceeding 20 ng/L, occured during periods of low flow in the summer. In constrast, clear water streams exhibited little seasonal variablity with Hg levels typically less than 5 ng/L. Samples from the Ottawa River contained 2.3 ng/L MeHg (26%), and from the Gatineau River, 4.1 ng/L MeHg (36%). As much as 36% of total Hg in water samples was MeHg. In the same study, water from Black Lake contained a lower concentration of MeHg (1.3 ng/L), but the

proportion of MeHg remained high, at 37% (Schintu et al. 1989). No explanation was given for the relatively high levels of MeHg found in the Gatineau River, but the authors noted that the results were similar to those of an earlier study (Kudo et al. 1982).

The total Hg levels reported by Kirkwood et al. (1999b) for two dystrophic lakes in south-central Ontario were among the lowest THg measurements reported for water samples in Canada. Using the "Mercury Clean Lab" at Dorset Research Centre, the 1995 seasonal means ± standard error for total mercury were 0.855 ± 0.318 and 0.462 ± 0.123 ng/L in unfiltered water samples for Mouse and Ranger lakes, respectively.

In the Great Whale and Nottaway-Broadback-Rupert areas of northern Quebec, water/seston samples taken in the summer of 1989-1991 contained 0.45 and 0.94 ng/L MeHg, respectively. Samples taken in the fall/winter from these areas contained 0.02 and 0.46 ng/L MeHg, respectively. The MeHg levels were 9 to 31% of THg (Langlois et al. 1995). MeHg levels in water samples from natural lakes of northern Quebec from 1994 to 1996 averaged 0.049 ±0.004 SE ng/L ranging from 0.018 to 0.115 ng/L (N=30) (Lucotte et al. 1999). MeHg levels were approximately 3% of THg.

In the flooded systems of the Robert Bourassa and Laforge-1 Reservoirs of northern Quebec, MeHg levels in water samples averaged 0.28 ng/L ranging from 0.03 to 0.85 ng/L (Thérien and Morrison 1999). Over the same period, MeHg in water from neighbouring natural lakes was lower averaging 0.05 ng/L and ranging from 0.02 to 0.11 ng/L (N=30). The percent of THg that was MeHg averaged 12% in the reservoirs and 3% in the lakes.

In particulate organic matter filtered from marine water samples from Lancaster Sound, NWT, THg was undetectable (<0.02 µg/g dw) (Atwell et al. 1998).

6.2 Sediments

Information on total Hg in sediments in the Canadian environment is detailed in "Canadian Sediment Quality Guidelines for Mercury" (Environment Canada 1997), while the natural distribution of geological mercury as it relates to sediments in Canada is mapped and described in Friske and Coker (1995). Average background concentrations of THg in Canadian freshwater sediments ranged from 0.003 to 21.0 mg/kg in lakes, and from 0.005 to 15.0 mg/kg in streams. Twenty-fifth and 95th percentile THg values for lakes were 0.04 and 0.175 mg/kg, respectively; and for streams were 0.023 and 0.20 mg/kg, respectively.

Ranges in marine sediments were similarly variable, with the highest levels of up to 23 mg/kg (Halifax Harbour) found in contaminated marine and estuarine sediments (Environment Canada 1997). MeHg in marine sediments is typically a small percentage of THg, similar to water, because MeHg production is inhibited by high sulphate concentrations (Gilmour and Henry 1991). For soil THg information, refer to Environment Canada (1996).

6.3 Aquatic Plants

Levels of THg and MeHg in aquatic plants in the Canadian environment were tabulated and discussed in the TRG guideline document (Environment Canada 2002). Briefly, the available data indicate that methylmercury usually represents less than 50% of total mercury in freshwater aquatic plants.

The levels of THg in plants ranged from 6 µg/kg dw for certain macrophytes at the ELA (NW Ontario, Moore et al. 1995) to 1600 µg/kg dw for a macrophyte (*Myriophyllum* sp.) collected in 1976 from the St. Lawrence River near Cornwall, ON (Mortimer 1985). While there were very few MeHg measurements, levels ranged from 0.45 µg/kg dw for the macrophytes at ELA to 20 µg/kg dw for sphagnum moss at the ELA (Moore et al. 1995).

The percentage of MeHg may depend on the type of tissue measured. The lower stem, upper stem, and capitulum of *Sphagnum augustifolium* contained 0.3%, 1.4% and 2.7% MeHg, respectively (0.3, 1.6, 2.5 µg/kg dw; Moore *et al*. 1995). The type of habitat appeared to affect the levels of MeHg in that species from a pool/stream habitat contained about 20 µg/kg dw (19%) MeHg, while sphagnum species from hollow/lawn and hummock habitats contained about 1 and 0.5 µg/kg dw, respectively (2.5% and 1.4% MeHg). The macrophyte and sedge species, *Carex aquatilis, C. rostrata* and *Sparganium* sp. contained about 2 µg/kg dw MeHg, on average, representing 37% of THg (Moore et al. 1995)

Macrophytes from the Ottawa River contained lower percentages of MeHg in their roots than their shoots. The roots of *Sagittaria latifolia, Sparganium augustifolium*, and *S. eurycarpum* contained 9 to 14% MeHg compared with 30 to 48% MeHg in their shoots. The levels of THg were 48 to 550 µg/kg, while the specific MeHg levels were not reported (Mortimer 1985). Data on Hg in plants from northern Quebec are found in Grondin (1994).

The spatial and temporal trends in THg concentration in phytoplankton in two shield lakes in south-central Ontario indicated that Hg in phytoplankton varied little in the mixed-water zones of the lakes seasonally, but was highest in the deep waters of the lakes during stratification (Kirkwood et al. 1999b). The dry weight concentrations of Hg in phytoplankton from both lakes at all depths averaged 0.21 ± 0.3 ng/mg dw for the 1995 season. Both lakes had Hg concentrations greater than 0.3 ng/mg dw in the hypolymnetic (deep water) samples, compared to <0.05 ng/mg dw in the metalimnion (mixed zone). These levels are lower than those reported for macrophytes above.

No data were located on levels of methylmercury in estuarine or marine plants in the Canadian environment.

6.4 Aquatic Invertebrates

MeHg concentrations in invertebrates were tabulated and described in the TRG guideline document. The present document will summarize the important information and describe some new studies including THg concentrations and levels in marine invertebrates.

The levels of THg in freshwater invertebrates in the Canadian environment ranged from an average of 0.014 µg/g ww in chironomids from a Manitoba reservoir (1981, Jackson 1988) to 2.2 µg/g ww for crayfish from contaminated Clay Lake near Dryden, ON (1979, Parks et al. 1991). MeHg levels ranged from 0.014 µg/g dw for mayfly larvae from an unaffected lake in northern Quebec to 1.52 µg/g dw in larval corixids (*Sigara* spp.) in LaGrande 2 Reservoir during the early 1990s. In general, the proportion of MeHg in invertebrates is higher than in aquatic plants with percentages ranging from 6% in chironomids from LaGrande-1 Reservoir in Québec to 100% in the larval Odonate, *Somatochlora* spp. from LaGrande-2 (Tremblay et al. 1996).

In the first study to document changes in Hg concentration in zooplankton collected both before and after reservoir creation, Paterson et al. (1998) found that average THg increased five times, and MeHg increased 10 times within six weeks of flooding. Zooplankton collected with an 80-µm net prior to impoundment of the northwestern Ontario water body ranged from 0.060 to 0.191 µg THg/g dw (mean=0.122) and from 0.011 to 0.054 µg MeHg/g dw (mean=0.032). After flooding, average THg concentrations exceeded 0.5 µg/g and MeHg concentrations exceeded 0.3 µg/g.

Unpolluted and unimpounded lakes in the ELA had much lower Hg in zooplankton than found in the reservoir created there. In 1993 and 1996, the mean MeHg concentration in zooplankton from 31 ELA lakes was 0.0574 µg/g dw (range 0.004 - 0.126 µg/g dw; M. Paterson, Dept. of Fisheries and Oceans, Winnipeg, unpubl. data).

MeHg concentrations in benthic invertebrates did not increase as dramatically as in zooplankton in the experimental reservoir project described above. Prior to impoundment, concentrations of MeHg in benthos were similar to or higher than in zooplankton; predacious insects had a mean MeHg concentration of 0.189 µg/g dw, and collector/shredders had a mean concentration of 0.072 µg/g dw. After impoundment, concentrations of MeHg increased approximately three times in predators and up to 4 times in collector/shredders (much less than the 10X change exhibited by zooplankton). Extensive tabulation of MeHg changes in invertebrates before and after flooding is given in Hall et al. (1998d).

Malley et al. (1996c) found that while the kidney of the mussel, *Pyganodon grandis*[19] contained higher concentrations of MeHg than other tissues, the proportion of MeHg in the kidney was the lowest, at 15%. Of 1.99 µg THg/g dw in the kidney, 0.29 µg/g was MeHg. The whole organism contained 44% MeHg, while the mantle, viscera, gill and foot contained 45%, 47%, 58% and 62% MeHg, respectively. This represented a range of 0.11 to 0.25 µg MeHg/g (Malley et al.

[19] Formerly *Anodonta grandis grandis*

1996c). All mussels were from an unmanipulated source lake (Lake 104) at the ELA, northwestern Ontario.

Organisms from the contaminated Wabigoon River/Clay Lake area of NW Ontario showed among the highest levels of THg measured in freshwater invertebrates. The crayfish *Orconectes virilis* from the east basin of Clay Lake collected in 1979-80 contained 2.2 ± 0.66 µg/g ww THg, and from the Wabigoon River (inflow to Ball Lake) contained 1.7 ± 0.49 µg/g ww THg (Parks et al. 1991). These are high levels for invertebrates considering that the consumption guideline for human consumers of fish in Canada is 0.5 µg/g on a wet weight basis. If the levels for the crayfish were converted to a dry weight basis, they would be even higher. For example, if tissues were considered at least 70% water, THg concentrations would be more than twice as high as reported above. Allard and Stokes (1989) reported THg concentrations in thirteen unmanipulated lakes in Ontario.

Concentrations of THg in the estuarine mussel, *Mytilus edulis*, were reported in areas of the St. Lawrence estuary for the year of cessation of chlor-alkali plant activities and one year later (1976-1977). Concentrations at the mouth of the Saguenay Fjord declined from 0.63 µg/g dw to 0.17 µg/g dw during this period. Similarly, levels of THg declined in two locations on the south shore of the lower estuary and in the Gaspé Peninsula during the same period (Cossa and Rondeau 1985).

Marine invertebrates from an unimpacted site in Lancaster Sound, NWT, were reported to contain from 0.04 ± 0.01 µg/g dw THg for the starfish (*Crossaster papposus, Leptastarias* spp.) to 0.18 µg/g dw for sea urchins (two specimens of *Strongylocentrotus* spp.). The clam, *Hiatella arctica*, contained an average of 0.15 ± 0.2 µg/g THg and several other species averaged below 0.1 µg/g dw, although sample sizes were small (from one to 7 specimens; Atwell et al. 1998).

6.5 Fish

Mercury in fish muscle is comprised almost exclusively of MeHg regardless of the composition of diet sources and exposure water. As such, the data tabulated in the TRG guideline document (Environment Canada 2002) includes numerous measurements of THg only, which are assumed to be MeHg concentrations in muscle tissue unless specifically reported as measured MeHg. Levels are generally highest in piscivorous species such as lake trout, northern pike, walleye and bull trout, and levels increase with age and size (length of exposure) of the fish. The literature is dominated by reports on freshwater fish in comparison to marine fish. All levels are reported as µg/g **ww** and in muscle tissue unless otherwise stated. Results will be discussed by province or territory, in alphabetical order.

Some lakes are more prone to MeHg problems than others. These include hydroelectric reservoirs (Abernathy and Cumbie 1977; Lodenius et al. 1983; Stokes and Wren 1987; Jackson 1988; Hecky et al. 1991; Schetagne et al. 1999a), low pH lakes (Wren and MacCrimmon 1983; Verta et al. 1986b; McMurtry et al. 1989), and even some remote lakes with no apparent stressors associated with high mercury. In some cases, water bodies were either directly contaminated with mercury by mine tailings or industry or by high geologic levels of mercury.

The explanation for high MeHg in fish is different in each case, but usually relates to factors affecting net MeHg production and transfer to the food web. In the case of newly formed reservoirs, the flooding of fresh vegetation and subsequent high bacterial activity causes mercury methylation rates to increase substantially; the subsequent elevated MeHg in fish may decline in a few years or may remain elevated for decades. (Ramlal et al. 1987; Hecky et al. 1991; Bodaly et al. 1984). Prediction of mercury problems associated with reservoirs may be possible using mathematical modelling (Johnston et al. 1991).

In low pH as well as non-acidic remote lakes, the explanation for high MeHg in fish is more complex. Mercury methylation rates are higher in low pH lake water and surface sediments (Xun et al. 1987). In fact, the methylcobalamin-dependent methylation process is optimized at pH 4.7 (D'Itri 1991). It is also possible that the uptake of MeHg by aquatic organisms in acidic waters is increased by calcium mediated changes in gill permeability (Rodgers and Beamish 1983). Further, the importance of sulphate reducing bacteria is enhanced in culturally-acidified lakes; these bacteria are thought to be the most important methylators in lake sediments (Gilmour and Riedel 1995). In remote non-acidified lakes with no apparent point source of mercury, sometimes the 'source' is traced to wetlands which may have high methylation rates combined with high DOC-related export of MeHg, as discussed in The Mercury Cycle section (section 4.1).

6.5.1 Freshwater fish

In Alberta, high levels of mercury in fish were reported from lakes contaminated with mine tailings. Lake trout (*Salvelinus namaycush*), northern pike (*Esox lucius*), and round whitefish (*Prosopium cylindraceum*) collected in 1977 from Giauque Lake in northern Alberta contained averages of 3.79, 1.75 and 1.22 µg/g ww, respectively (Moore and Sutherland 1980). In another contaminated lake, Thompson Lake, northern pike collected in 1978 had an average of 1.7 µg/g THg in muscle tissue. Regardless of whether a lake was contaminated or not, lake whitefish (*Coregonus clupeaformis*) had low average mercury concentrations of 0.2 µg/g. In a number of control lakes, lake trout and northern pike muscle concentrations ranged from 0.11 to 1.0 and 0.05 to 1.91 µg/g, respectively (Moore and Sutherland 1980).

Ramamoorthy et al. (1985) reported mercury levels in seven fish species from the North Saskatchewan River in the vicinity of Edmonton. Average concentrations ranged from 0.25 µg/g for longnose sucker (*Catostomus catostomus*) to 0.74 µg/g for sauger (*Stizostedion canadense*). Two other species that exceeded the consumption guideline of 0.5 µg/g were goldeye (*Hiodon alosoides*; 0.59 µg/g) and walleye (*S. vitreum*; 0.65 µg/g). Ramamoorthy et al. (1985) also reported the proportion of MeHg ranged from 77 to 95% in longnose sucker and walleye, respectively.

In British Columbia, mercury in fish has been quantified in a number of lakes and rivers, as well as Williston Lake (reservoir associated with the Bennett Dam on the Peace River). The highest mercury concentrations were found in lake trout from Pinchi Lake, which is located in an area of high geological mercury and a former mercury mine. Mercury in these lake trout ranged from 1.06 to 5.78 µg/g (Watson 1992). Bull trout (*Salvelinus confluentus*) levels were relatively high

both in Pinchi Lake (0.49 to 0.75 µg/g) and in Williston Lake (0.25 to 1.62 µg/g). Kokanee (*Oncorhynchus nerka kennerlyi*) and rainbow trout (*Oncorhynchus mykiss*) from Pinchi Lake contained moderate to high levels at 0.32 to 0.76 µg/g and 0.20 to 0.40 µg/g, respectively. This geographical area has extensive bedrock mercury deposits and has supported mercury mining in the past (C. Gray, Environment Canada, pers. comm. 2000). In contrast, fish collected from a total of 54 uncontaminated lakes throughout BC had low concentrations of mercury. For example, mean Hg levels in muscle tissue ranged from 0.08 to 0.29 µg/g among six species of fish (Rieberger 1992).

In a number of rivers in northern BC, mercury concentrations ranged from 0.02 to 0.78 µg/g. On average, rainbow trout and lake whitefish had the lowest levels, while burbot (*Lota lota*) and bull trout had higher levels (Watson 1992).

From 1975 to 1982, extensive monitoring of mercury in fish was done in association with reservoirs in northern Manitoba. Average mercury concentrations associated with the Churchill River diversion and flooding (Notigi Reservoir) including Southern Indian Lake exceeded 0.5 µg/g in all species except lake whitefish[20] (Bodaly et al. 1984). Average mercury levels exceeded 2 µg/g in walleye and northern pike from the Rat Lake portion of the Notigi Reservoir, and exceeded 1 µg/g in most other areas of the Notigi Reservoir. Levels were also elevated (>0.75 µg/g) in these species in five lakes downstream of the Notigi Reservoir. In Southern Indian Lake, walleye and northern pike had average muscle concentrations of 0.57 µg/g and 0.68 µg/g, respectively, while lake whitefish averaged 0.13 µg/g (Bodaly et al. 1984).

The Atlantic provinces have been active in acquiring fish mercury data. In a multi-agency study in Kejimkujik National Park, NS, hundreds of measurements in fish tissue have been made (report publications pending, Mercury Team 1998). In the Smallwood Reservoir in Labrador during the 1970s, land-locked salmon reportedly contained 1.5 to 2.3 µg/g (c.f. Bailey, 1985; Bruce and Spencer 1978). Mercury measured in 1977 and 1978 in northern pike from Labrador lakes ranged from 0.01 to 3.08 µg/g (n=117), in lake trout from 0.12 to 3.37 µg/g (n=107) and in brook trout from 0.01-1.69 µg/g (n=96) (n=96 Bruce et al. 1979).

In Wreck Cove Reservoir, NS, brook trout weighing from 49-211 g had muscle mercury concentrations of 0.27-0.73 µg/g, averaging 0.50 ± 0.16 µg/g (ADI Nolan Davis Inc. 1994). Two land-locked salmon[21] from Sisson Branch Reservoir in the St. John River basin (New Brunswick), measuring 38 and 58 cm and weighing less than 1.5 kg, contained 0.84 and 1.5 µg/g THg (Bailey 1985). Twelve lake whitefish from the Green River Reservoir (NB) averaged 0.12 µg/g. A survey of 108 brook trout in 40 New Brunswick lakes found an average of 0.18 µg Hg/g (range of 0.03-0.84 µg Hg/g); a small sample (6 specimens) of smallmouth bass in New Brunswick found an average of 0.76 µg/g (range of 0.31-1.38 µg/g; NESCAUM 1998).

The anadromous striped bass (*Morone saxatilis*) in the Annapolis River had elevated Hg in muscle tissue ranging from 0.26 - 1.97 µg/g (mean 0.77, mean weight of 7.4 kg); lower for

[20] Others have reported on mercury in these systems; the summary of (Bodaly et al. 1984) is used here for brevity.

[21] Species not reported, Atlantic salmon is the likely species based on location.

smaller specimens (mean 1.8 kg) from the Shubenacadie River ranging from 0.16 - 1.44 µg/g (mean 0.40 µg/g) (Ray et al. 1984).

In Northwest Territories, mercury concentration in a variety of species was reported to be below 0.5 µg/g (on average), with some exceptions. Lake trout from Kaminak Lake, west of Hudson Bay, contained 0.57 to 2.0 µg/g THg (Shilts and Coker 1995). The Kaminak Lake area has high geological mercury. For unknown reasons, northern pike, lake trout and walleye have elevated mercury (means of 0.74, 1.34, 1.49 µg/g, respectively) in Lac Ste. Thérèse in the Johnny Hoe River system (Stephens 1995). The high levels have persisted over time, with measurements in walleye being made in 1975, 1980, 1992 and 1993. Walleye, but not pike or lake whitefish have average concentrations that exceed 0.5 µg/g in another lake in the system, Tseepantee Lake. Two additional lakes (Keller and Taché) have fish with relatively lower THg concentrations.

In the Hay River, NWT, lake whitefish, walleye and northern pike averaged 0.07, 0.22 and 0.32 µg/g, respectively (1989-1990). Similar levels were found for these species from 1988-1990 in the Slave River and in Leland Lake (Grey et al. 1995).

The Ontario Ministry of the Environment maintains a very large database of mercury concentrations in fish (over 126,000 as of Sept. 1999; G. Mierle, OME, pers. com.). The most highly mercury contaminated area in the country is likely the English-Wabigoon River system in northwestern Ontario. The source of mercury was a chlor-alkali plant located in the town of Dryden (Rudd et al. 1983). Very high levels of mercury in Clay Lake burbot and walleye were 21.9 and 15.7 µg/g on average, respectively (Fimreite and Reynolds 1973). In Ball Lake, downstream of Clay Lake, walleye, rock bass (*Ambloplites rupestris*) and northern pike averaged 4.5, 6.2 and 7.7 µg/g, respectively. White suckers (*Catostomus commersoni*) from Clay Lake and the Wabigoon River had mercury levels in excess of 3 and 4 µg/g, respectively. Fish from many other connected lakes and rivers also had elevated mercury concentrations (Fimreite and Reynolds 1973).

Cabana and Rasmussen (1994) found a positive correlation between lake trout mercury concentrations and length of the food chain in large number of Ontario lakes. Lake trout from lakes with pelagic forage fish and *Mysis* shrimp had an average of 0.65 µg/g compared with 0.18 µg/g in lake trout from lakes that contained *Mysis* but lacked forage fish. The relationship did not exist for smallmouth bass (*Micropterus dolomieui*), a species that does not exploit the pelagic food chain, for which THg averaged from 0.43 to 0.53 µg/g regardless of the length of the food chain.

Lake trout had high mercury concentrations relative to rainbow smelt (*Osmerus mordax*) in a series of Ontario lakes examined from 1977 to 1981. Lakes Muskoka, Mary and Vernon were rated high mercury because lake trout concentrations averaged 2.96 to 4.49 µg/g (0.34 to 0.49 for smelt). Lakes Koshlong, Rosseau and Tadenac were rated moderate because lake trout concentrations averaged 0.61 to 0.99 µg/g (0.18 to 0.26 for smelt), and Lakes Simcoe, Joseph and Bella were rated low Hg because of lake trout concentrations of 0.39 to 0.55 µg/g (0.08 to 0.14 for smelt; MacCrimmon et al. 1983).

Northern pike from 170 lakes and walleye from 255 lakes from across Ontario indicate that fish of a certain size from seemingly unpolluted systems overall average muscle concentration at or above the Health Canada human health guideline level of 0.5 µg/g. Northern pike of approximately 54 cm in length averaged 0.50 µg/g (0.07 to 1.28 µg/g), and walleye of approximately 41 cm in length averaged 0.58 µg/g (0.09 to 3.24 µg/g) over a wide variety of lakes (Wren et al. 1991).

Yearling yellow perch (*Perca flavescens*) collected both from the Ottawa River and a number of lakes in northwestern Ontario had consistently low levels of mercury. Perch collected in 1974 and 1975 from the Ottawa River contained 0.13 µg/g (Rodgers and Qadri 1982) and from six remote lakes from NW Ontario averaged 0.04 to 0.14 µg/g (Bodaly et al. 1993).

In Québec, environmental impact studies related to hydroelectric development projects have focused on the Great Whale, La Grande and Nottaway-Broadback-Rupert (NBR) drainage basins. In all, over 8 000 fish from more than 180 lakes were sampled. In natural lakes in these watersheds, mean mercury levels for 400 mm longnose sucker and lake whitefish ranged from 0.07 to 0.30 µg/g and from 0.05 to 0.36 µg/g respectively (Schetagne and Verdon 1999b). For 400 mm walleye and 700 mm northern pike, corresponding mean concentrations ranged from 0.30 to 1.41µg/g and from 0.30 to 1.81 µg/g, respectively. Although for each species, significant differences ($p<0.05$) in average concentrations were found between regions, no clear south to north gradient was observed between latitudes 49^{o}N and 54^{o}N. In the NBR region, where lakes show a wide range of physical and chemical properties, higher mercury levels in fish were found in lakes with high organic content.

Mercury levels in longnose sucker, lake whitefish, walleye and northern pike were monitored before and after the impoundment of reservoirs at the La Grande hydroelectric complex on the Eastern coast of James Bay. Concentrations in all species increased rapidly after impoundment, peaking after 5 to 9 years in non piscivorous fish, and after 10 to 13 years in piscivorous species, at levels 3 to 7 times those measured in surrounding natural lakes. Levels then gradually declined (Schetagne and Verdon 1999a). The following maximum mean levels were reported for the La Grande 2 reservoir: 0.53 µg/g for 400 mm lake whitefish. 0.63 µg/g for 400 mm longnose sucker, 3.33 µg/g for 700 mm northern pike and 2.76 µg/g for 400 mm walleye. Mercury levels in lake whitefish returned to levels measured in surrounding natural lakes after 10 to 19 years depending on the reservoir. The reduction in lake whitefish mercury probably occurred as a result of the depletion of decomposable flooded organic material (which stimulated mercury methylation) and extensive shoreline erosion which reduced the transfer of MeHg to fish by benthic invertebrates (Schetagne and Verdon 1999a; Doyon and Schetagne 1999). In the La Grande 2 reservoir, nineteen years after flooding, mercury levels in northern pike and walleye had decreased moderately (by 30 and 45%, respectively) compared to maximum levels. Studies in older reservoirs show a return to background levels after 20 to 30 years for piscivorous species (Schetagne and Verdon 1999a).

Measurements of Hg in northern pike and walleye from 235 lake and 214 river sites across Quebec indicated that Hg averages exceeded 0.5 µg/g in many cases. Northern pike between 40-50 cm, 55-70 cm and >70 cm measured 0.4 µg/g, 0.66 µg/g and 1.12 µg/g, respectively.

Walleye between 30-40 cm, 40-50 cm and >50 cm averaged 0.53 µg/g, 0.78 µg/g and 1.26 µg/g, respectively (Laliberte 1996).

In Saskatchewan, northern pike collected in 1979 from Lac LaRonge ranged from 0.07 to 0.19 µg Hg/g. These were low levels considering that the pike (piscivores) were over 50 cm in length and weighed over 1.2 kg. Northern pike collected from 1978 – 1980 from Pasqua Lake ranged from 0.2 to 2.3 µg Hg/g. The fish that measured over 2 µg/g weighed over 3 kg, and a few weighed over 6 kg, indicating long-term exposure to Hg in its diet of fish. Walleye from the Saskatchewan River collected in 1973 ranged from 0.17 to 1.45 µg Hg/g. All of these fish weighed less than 1 kg, and were less than 45 cm total length (DFO database cited from Kent et al. 1998). Thousands of other measurements on fish from Saskatchewan, and the other regions of Canada may be found in the GIS database project in preparation at Environment Canada (Kent et al. 1998).

6.5.2 Marine fish

Atlantic herring (*Clupea harengus harengus*) collected in 1981 from the southwestern Bay of Fundy had low levels of total mercury ranging from 0.005 to 0.015 µg/g in muscle and whole body. The fish were one to five years in age, and mercury in muscle and whole body was found to increase with with age (Braune 1987).

Greenland cod (*Gadus ogac,* 400 mm) caught in the coastal waters of James Bay between 1987 and 1994 contained from 0.14 to 0.42 µg/g of mercury, while the fourhorn sculpin (*Myoxocephalus quadricornis*, 250 mm) from the same regions contained from 0.1 to 0.55 µg/g (Hydro-Québec and Groupe-Conseil Genivar Inc. 1997).

Muscle tissue of arctic cod (*Boreogadus saida*) from Lancaster Sound averaged 0.19 ± 0.03 µg THg/g dw, and two-horn sculpin (*Icelus bicornis*) averaged 0.24 ± 0.04 µg THg/g dw (Atwell et al. 1998)

6.6 Amphibians

Levels of THg in the mudpuppy, *Necturus maculosus*, from various locations in the St. Lawrence and Ottawa Rivers averaged from 0.087 to 0.239 µg/g on a wet weight basis, with a maximum individual concentration of 0.445 µg/g ww (Bonin et al. 1995). The high concentration was an adult female from the Beauharnois area of the St. Lawrence River; whole body with gonads removed. The average whole body concentration (without gonads) was 0.239 ± 0.137, while the gonads of the same six individual females averaged 0.068 µg/g ww.

6.7 Reptiles

Snapping turtle eggs (*Chelydra serpentina*) collected in 1989 and 1990 from the St. Lawrence River and tributaries contained relatively low concentrations of THg (0.09 to 0.15 µg/g ww) (Bonin et al. 1995). No data were located on levels of methylmercury or proportion of MeHg to THg in reptiles in the Canadian environment.

Table 3: Concentrations of total Hg and MeHg in selected abiotic compartments in the Canadian environment

Location	Year	Media	N	Total Hg ng Hg/L or µg Hg/g dw[22]	MeHg ng Hg/L or µg Hg/g dw	% MeHg	Reference
ON/QC: Black L.	1987	Water, lake	5	3.5	1.3	37%	(Schintu et al. 1989)
ON/QC: Gatineau R.	1987	Water, river	5	11.4	4.1	36%	(Schintu et al. 1989)
ON/QC: Ottawa R.	1987	Water, river	5	9.0	2.3	26%	(Schintu et al. 1989)
ON: Experimental Lakes Area	1992	Rain, August & Sept. samples	4	4.62	0.017	0.4%	(St.Louis et al. 1995a)
ON: Experimental Lakes Area	1993	Rain, May - August	6	5.33	0.049	0.9%	(St.Louis et al. 1995a)
ON: Experimental Lakes Area	1995	Peat porewater	NR[23]	6	0.6	10%	(Moore et al. 1995)
ON: Experimental Lakes Area	1993	Snow on lake surface	2	0.95	0.008	0.8%	(St.Louis et al. 1995a)
ON: Experimental Lakes Area	1994	Snow on lake surface	2	1.96	0.032	1.7%	(St.Louis et al. 1995a)
ON: Experimental Lakes Area	1992	Snow, Oct. & Nov. samples	2	3.58	0.104	2.9%	(St.Louis et al. 1995a)
ON: Experimental Lakes Area	1991	Water from upland terrestrial area	9	13.11 ± 0.67	0.03 ± 0.006	0.2%	(St.Louis et al. 1994)
ON: Experimental Lakes Area	1991	Water from wetland dominated area	19	5.02 ± 0.36	0.626 ± 0.126	13.3%	(St.Louis et al. 1994)
ON: Experimental Lakes Area	1991	Water from wetland/upland area#1	15	11.77 ± 0.73	0.176 ± 0.012	1.6%	(St.Louis et al. 1994)
ON: Experimental Lakes Area	1991	Water from wetland/upland area#2	9	11.40 ± 0.87	0.228 ± 0.033	2.0%	(St.Louis et al. 1994)
ON: Experimental Lakes Area	1991-2	Water, Pond 979 before flooding	NR	2.6 ± 1.5	0.09 ± 0.04	4.4%	(Kelly et al. 1995)
ON: Experimental Lakes Area	1993	Water, Pond 979 after flooding	NR	4.5 ± 1.7	1.38 ± 0.51	31%	(Kelly et al. 1995)
ON: Experimental Lakes Area	1991-2	Water, Pond 979 before flooding	NR	0.40 - 7.20	0.05 - 0.28	4%	(Kelly et al. 1997)

[22] ng/L is unit for water-based media; µg/g is unit for solid media.

[23] NR = not reported

Location	Year	Media	N	Total Hg ng Hg/L or µg Hg/g dw[22]	MeHg ng Hg/L or µg Hg/g dw	% MeHg	Reference
ON: Experimental Lakes Area	1993-4	Water, Pond 979 after flooding	NR	0.98 - 6.95	0.05 - 3.2	32% (max 73%)	(Kelly et al. 1997)
ON: Experimental Lakes Area	1991-4	Water, Lake 240 (clear lake control)	NR	0.62-3.52	0.004-0.22	~6%	(Kelly et al. 1997)
ON: Experimental Lakes Area	1991-4	Water, Lake 632 (brown water wetland pond control)	NR	1.46 - 6.73	0.13 - 0.99	~9%	(Kelly et al. 1997)
ON: Experimental Lakes Area	1991	Water, upland/wetland & lake area	16	1.69 ± 0.27	0.040 ± 0.003	4.4%	(St.Louis et al. 1994)
ON: Experimental Lakes Area	1991	Wet deposition	5	3.81 ± 0.85	0.019 ± 0.004	0.6%	(St.Louis et al. 1994)
ON: Wabigoon/English R.	1978/9[24]	Water, Clay L.	26	NR	1.3	6%[25]	(Parks et al. 1989)
ON: Wabigoon/English R.	1978/9	Water, English R., Ball L. outflow	12	NR	0.22	NR	(Parks et al. 1989)
ON: Wabigoon/English R.	1978/9	Water, Wabigoon L.	25	NR	0.27	NR	(Parks et al. 1989)
ON: Wabigoon/English R.	1978/9	Water, Wabigoon R., Clay L. outflow	50	NR	1.3	NR	(Parks et al. 1989)
ON: Wabigoon/English R.	1978/9	Water, Wabigoon R., inflow to Wainwright Dam	25	NR	0.43	NR	(Parks et al. 1989)
QC: Great Whale area, Northern Quebec	1989-1990	Sediments	12	0.07 ± 0.05	NR	NR	(Langlois et al. 1995)
QC: Great Whale area, Northern Quebec	1989-1990	Seston - fall samples	7	0.11 ± 0.07	0.02 ± 0.02	18.2%	(Langlois et al. 1995)
QC: Great Whale area, Northern Quebec	1989-1990	Water - summer samples	22	4.8 ± 6.62	0.453 ± 0.37	9.4%	(Langlois et al. 1995)

[24] Samples taken at a time prior to the development of "ultra-clean" techniques.

[25] Cited as %MeHg in English-Wabigoon system, from Kelly et al. (Kelly et al. 1997).

Location	Year	Media	N	Total Hg ng Hg/L or µg Hg/g dw[22]	MeHg ng Hg/L or µg Hg/g dw	% MeHg	Reference
QC: LaGrande 2 Reservoir	1992	Soils, flooded, 0-4 cm depth	NR	0.129	0.0085	6.6%	(Mucci et al. 1995)
QC: LaGrande 2 Reservoir	1992	Soils, flooded, 11-19 cm depth	NR	0.032	0.0019	5.9%	(Mucci et al. 1995)
QC: LaGrande 2 Reservoir	1992	Soils, flooded, 4-11 cm depth	NR	0.142	0.043	30%	(Mucci et al. 1995)
QC: Northern reservoirs/lake	1992	Sediments, surface	NR	0.036 to 0.059	0.0001 to 0.0006	1%	(Tremblay et al. 1996)
QC: Nottaway-Broadback-Rupert area, SE coast James Bay	1990-1991	Water - summer samples	25	3.07 ± 1.71	0.94 ± 0.54	31%	(Langlois et al. 1995)
QC: Nottaway-Broadback-Rupert area, SE coast James Bay	1990-1991	Water - winter samples	20	4.45 ± 1.73	0.46 ± 0.83	10.3%	(Langlois et al. 1995)
QC: Natural lakes	1994-1996	Water	30	--	0.049 ± 0.004 0.018 to 0.115	3%	(Lucotte et al. 1999)
SK: Pasqua L.	1982[26]	Water	4	51.6	0.081	0.16%	(Jackson 1993)

[26] Samples taken at a time prior to realization that "ultra-clean" techniques were necessary.

7.0 Toxicity of Mercury and Methylmercury to Aquatic Organisms

Mercury is considered to be one of the most toxic metals found in the environment, although is it relatively less toxic to aquatic organisms than to piscivorous birds and mammals (Environment Canada 2002). Inorganic Hg (mercuric chloride) was more toxic than arsenic and cadmium to bluegills and crayfish (Abdelghani et al. 1995) and more toxic than chromium and zinc to arthropods and protozoans (reviewed by Abbasi et al. 1988). Mercuric chloride had the highest acute toxicity and second highest chronic toxicity (next to cadmium) to *Daphnia magna* in a comprehensive study comparing eighteen metal ions (Biesinger and Christensen 1972).

While the majority of toxicity tests have been conducted with inorganic Hg (as $HgCl_2$), the methylated form of mercury (CH_3HgCl or MeHg) is the most toxic of the mercuric compounds. Concurrent tests indicated that MeHg is at least ten times more toxic than inorganic Hg to a variety of aquatic species. Lock (1981) reported a lethal concentration (96-h LC_{50}) of 24 µg Hg/L (as MeHg) compared to 275 µg Hg/L (as $HgCl_2$) for rainbow trout; Niimi and Kissoon (1994h) reported reduced survival (60-day EC_{50}) at 4 µg Hg/L MeHg and 64 µg Hg/L inorganic Hg (as $HgCl_2$) for rainbow trout; Biesinger et al. (1982) reported that survival of *D. magna* was significantly affected at 0.26 µg Hg/L for MeHg compared to 2.70 µg Hg/L for inorganic Hg; and Thomas and Montes (1978) reported 24-h LC_{50} values of 3.5 µg Hg/L MeHg and 72 µg Hg/L inorganic Hg for the blue-green alga, *Anabaena flos-aquae*. For perspective, it is important to note that inorganic Hg may have ten or more times the abundance of MeHg in aquatic environments (see environmental concentrations section).

The sensitivity of aquatic organisms to either inorganic or MeHg varies considerably between species. For freshwater fish alone, the concentration of inorganic Hg causing acute toxicity ranged from 74 µg Hg/L (7-day LC_{50}, fathead minnows) to more than 800 µg Hg/L (48-h LC_{50}, young white suckers). With fewer studies completed on MeHg, the range in acute toxicity for all freshwater species varied from 24 µg Hg/L (rainbow trout fry) to 88 µg Hg/L for larval lamprey (only studies ranked primary[27]; see Table 4). For saltwater species, 96-h LC_{50} values ranged from 3.5 µg Hg/L (as $HgCl_2$) for an estuarine mysid to over 1000 µg Hg/L for winter flounder (all ranks of studies included; see Table 5). Two studies for MeHg indicated that toxicity ranged from 50 µg Hg/L (96-h test) to 500 µg Hg/L (20-min test) for mummichog embryos. All concentrations are for Hg regardless of whether the compound is organic or inorganic.

The toxic concentration of Hg compounds may be affected by water quality parameters such as temperature, oxygen, salinity and selenium. Toxicity is increased at elevated temperatures (Snell et al. 1991; Heit and Fingerman 1977; MacLeod and Pessah 1973), lower oxygen content (Slooff et al. 1991), lower salinity (McKenney, Jr. and Costlow, Jr. 1981), and lower selenium (e.g. Heisinger et al. 1979). The importance of selenium in reducing the toxic effects of mercury has long been known (reviewed by Cuvin-Aralar and Furness 1991). Water hardness has a negligible effect on mercury toxicity (Keller and Zam 1991) unlike other toxic metals such as copper or aluminum, where toxicity significantly decreases with increasing hardness.

[27] Primary, secondary and unacceptable data as defined by CCME (CCME 1991).

Prior exposure to mercury may produce populations that acclimated to mercury contamination. Offspring from fish previously exposed to mercury contamination were more tolerant and accumulated less mercury than offspring from clean environments (Weis and Weis 1989).

7.1 Toxicity to Fish

7.1.1 Acute Toxicity to Freshwater Fish

Mercuric chloride and MeHg were described by Wobeser (1975) to cause flaring of the opercula and increased frequency and force of respiratory movements of rainbow trout fry and fingerlings. Increasing concentrations caused loss of equilibrium, sluggishness and a tendency to lie on the bottom of the tank. Prolonged exposure caused death. Mercuric chloride tended to induce mucus production more so than MeHg.

Studies ranked primary indicate that 96-h LC_{50} values for MeHg ranged from 24 - 88 µg Hg/L for juvenile and older fish, and inorganic Hg concentrations of 150 - 687 µg Hg/L were lethal to similar-aged fish. Considering studies ranked secondary, the single study for MeHg indicated 24 µg Hg/L was lethal to rainbow trout fry (Wobeser 1975), while inorganic Hg 96-h LC_{50} values ranged from 124 to 400 µg Hg/L (as $HgCl_2$; Table 4).

Rainbow trout (31-44 g) were the most sensitive species to MeHg in flow-through tests, with a 96-h LC_{50} value of 24 µg Hg/L (Lock and Van Overbeeke 1981). Slightly larger brook trout (*Salvelinus fontinalis* weighing 50-70 g) were somewhat less sensitive with a 96-h LC_{50} value of 75 µg Hg/L in a similarly designed test (McKim et al. 1976). Mallatt et al. (1986c) found that larval lamprey (*Petromyzon* sp.) were less sensitive to acute MeHg toxicity at 4^{o}C than at 12 ^{o}C, with 88 and 48 µg Hg/L 96-h LC_{50} values, respectively.

Fathead minnows (*Pimephales promelas)* were more sensitive than coho salmon (*Oncorhynchus kisutch)*, rainbow trout or white sucker in 96-h LC_{50} tests with $HgCl_2$. All tests were performed under flow-through conditions, with measured and reported Hg concentrations. The lowest fathead minnow lethal concentration was 150 µg Hg/L (Call et al. 1983), the salmonids ranged from 200 - 300 µg Hg/L (Lorz et al. 1978; Lock and Van Overbeeke 1981) and the white sucker (*Catostomas commersoni*) was 687 µg Hg/L (Call et al. 1983; Snarski and Olson 1982a; Lorz et al. 1978; Lock and Van Overbeeke 1981; Duncan and Klaverkamp 1983, respectively).

7.1.2 Chronic Toxicity to Freshwater Fish

Indicators of chronic mercury poisoning in fish include weight loss (lost appetite), reproductive failure, avoidance or inability to capture food, brain lesions, cataracts, various erratic behaviours, deformities, increased metallothionein production, and various biochemical changes (Eisler 1987). Similar to acute tests, the earlier life stages are more susceptible than later ones.

A study ranked primary (flow-through conditions, measured Hg, etc.) that was completed by Snarski and Olson (1982a) lasting two generations of fathead minnows, indicated that inorganic

Hg concentrations as low as 0.26 µg Hg/L caused reduced growth of second generation larvae. Some fish failed to mature and spawn. A concentration of 0.5 µg Hg/L was the 60-day EC_{50} (reproductive impairment), and 4.5 µg Hg/L was the 60-day EC_{50} (survival and spinal malformation). They recommended a Maximum Acceptable Toxicant Concentration (MATC) and LOAEL of the lowest level of Hg they tested, <0.26 µg Hg/L.

A study of MeHg toxicity to three generations of brook trout indicated that at 0.93 µg Hg/L, second generation trout developed deformities, and all but one female died during spawning (McKim et al. 1976). They found that the MeHg was relatively rapidly accumulated by the fish, and that virtually none was eliminated. A NOAEL of 0.29 µg Hg/L (as MeHgCl) was found, and at water concentrations as low as 0.03 µg Hg/L the fish quickly and irreversibly (monitored for up to two years) accumulated >0.5 µg Hg/g (as MeHg) in muscle tissue.

Very early life stages of some fish species were sensitive to inorganic Hg toxicity. Birge et al. (1979) conducted flow-through tests on spawned eggs through four days post-hatch on rainbow trout, channel catfish and largemouth bass. A measured concentration of 0.1 µg Hg/L was lethal to all rainbow trout eggs in 8 days (none hatched), although they did not use a range of Hg concentrations for this species. The 10-d EC_{50} (survival) to embryo-larval catfish was 0.3 µg Hg/L. Largemouth bass embryo-larval stages proved most resistant, with an 8-d EC_{50} (survival) of 5.3 µg Hg/L (Birge et al. 1979).

In static tests, the mosquitofish (*Gambusia* sp.) displayed reduced survival over 60 days at <42 µg Hg/L (as $HgCl_2$) with impaired reproduction over 111 days at a concentration of 18 µg Hg/L (Mulvey et al. 1995). In a follow-up experiment, four generations of the mosquitofish were exposed over two years to residual mercury from sediments in large mesocosms. No significant differences were found in standard length, weight, sex ratio, or age class ratio between the control and mercury-exposed populations, but genetic changes (allele frequency changes at the *Gpi-2* locus) suggested a population-level response to chronic mercury exposure (Tatara et al. 1999). While the Hg concentration in the exposure water was below 0.1 µg Hg/L (detection limit), uptake and exposure to Hg in the diet undoubtedly contributed to any effects. The environmental relevance of the genetic change is unknown.

In static assays comparing MeHg and inorganic Hg (as $HgCl_2$) toxicity over a 60 day period, Niimi and Kissoon (1994h) found MeHg to be about 16 times more toxic than inorganic Hg (EC_{50} survival) to rainbow trout (4 and 64 µg Hg/L, respectively). Similarly, Lock (1981) reported a lethal concentration (96-h LC_{50}) of 24 µg Hg/L (as MeHg) compared to 275 µg Hg/L (as $HgCl_2$) for rainbow trout.

The review paper of Spry and Wiener (1991) outlines mercury and other metal bioavailability and toxicity to fish, with emphasis on low-alkalinity lakes.

7.1.3 Toxicity to Marine Fish

Mercury toxicity studies on marine fish are scarce, but those available indicate that marine/estuarine fish are less susceptible to Hg toxicity from direct water exposure than most freshwater fish (Table 5).

One exception was a study indicating that a salmonid (chum salmon, *Oncorhynchus keta*), may be at least as sensitive as freshwater salmonids, despite exposure in saline water. Chum salmon fry were introduced to large mesocosms (1350 m^3) located in Saanich Inlet off Vancouver Island, BC. Three mesocosms were used with a control, 1 µg Hg/L and 5 µg Hg/L (as $HgCl_2$) . It was estimated that 2% of added Hg was lost each day of the 72-day experiment. On day 72, fry exposed to 5 µg Hg/L weighed about half that of fry in either the control or 1 µg Hg/L mesocosm (Brown and Parsons 1978). This study was ranked as secondary.

The most commonly studied marine fish is the mummichog (sometimes called killifish; *Fundulus heteroclitus*) which is one of the most productive and abundant small fish in the tidal marshes on the east coast of NA up to the Gulf of St. Lawrence. They are an important component in the benthic food web of estuaries, and are a major prey item for shorebirds and fishes (Abraham 1985).

The acute lethal (96-h LC_{50}) value for the embryo stage of mummichog in a study ranked primary was 68 µg Hg/L inorganic Hg (Sharp and Neff 1980). Spinal deformities and reduced hatching success resulted from 32 day exposures (EC_{50}) of embryo-larval stages to 32 - 37 µg Hg/L (as $HgCl_2$). The authors measured Hg concentrations finding that 25-30% of added Hg was lost despite daily replacement of the media (Sharp and Neff 1980), while in a subsequent experiment, only 10% of inorganic Hg and MeHg was lost (Sharp and Neff 1982). MeHg was moderately more acutely toxic than inorganic Hg to embryonic stages of the mummichog, with 96-h LC_{50} values of 50 µg Hg/L (as MeHg), and 67 µg Hg/L inorganic Hg (Sharp and Neff 1982). Results were based on nominal concentrations and may thus be considered conservative.

Too few studies are available using MeHg to make definitive statements on MeHg toxicity to marine fish. In static, nominal 96-h LC_{50} assays, MeHg was lethal to juvenile mummichog at approximately 200 µg Hg/L, which was higher than the concentration for embryos reported above. The test containers were covered, but the actual MeHg concentrations were unmeasured. In short duration exposures of 20 minutes, fertilisation success but not embryonic development was reduced at 500 µg Hg/L. The authors found that eggs stripped from fish collected from a polluted region were more tolerant to the brief exposure than those from an unpolluted region (Khan and Weis 1987). MeHg resistance was not transferred to juvenile fish that developed from pre-exposed egg stages.

Six other species were tested in U.S. EPA laboratories although the results were not published, and were reported only in internal memoranda. These tests indicated that unknown-duration LC_{5o} values ranged from 36 µg Hg/L for the spot (*Leiostomus xanthurus*) to 1810 µg Hg/L inorganic Hg for winter flounder (Pseudopleuronectes americanus; see Table 5; cited from U.S. EPA 1980).

7.2 Toxicity to Invertebrates

7.2.1 Acute Toxicity to Freshwater Invertebrates

Including studies ranked secondary, the most sensitive invertebrates to Hg were *Daphnia* spp., moderately sensitive were the larval insect *Chironomus*, the pulmonate snail *Lymnaea*, and the amphipod *Gammarus*. The most resistant were the freshwater mussel *Anodonta imbecilis*, Copepoda, the gastropod *Amnicola* sp., as well as a caddisfly (Trichoptera), and a mosquito larvae (*Aedes* sp.).

MeHg was acutely lethal in 96-h tests to *Daphnia pulex* at a level of 1.8 µg Hg/L (Chen and McNaught 1992), compared to the lowest acute level for inorganic Hg of 5 µg Hg/L in 48-h tests with *D. magna* (Biesinger and Christensen 1972). The common amphipod, *Gammarus* sp., had a 96-h LC_{50} value of 10 µg Hg/L (Rehwoldt et al. 1973). All of these assays were conducted under static conditions with nominal Hg concentrations.

One species of adult male crayfish (*Faxonella clypeata*) showed a lethal response to inorganic Hg at 20 µg Hg/L, and was 10 times more sensitive than adult males of the crayfish, *Procambarus clarki,* with a 3-day LC_{50} of 200 µg Hg/L (Heit and Fingerman 1977). These tests were completed in natural pond water, with daily water renewal and measurement of Hg.

Copepods, oligochaetes, caddisflies and a gastropod snail appear to be among the least sensitive to inorganic Hg of the freshwater invertebrates with LC_{50} values of 850 (48-h), 1000 (96-h), 1200 (96-h), and 2100 µg Hg/L (96-h), respectively. All of these studies are from the early 1970s, and were conducted with $HgCl_2$ under static conditions, with nominal Hg concentrations (see Table 4).

7.2.2 Chronic Toxicity to Freshwater Invertebrates

Compared with the abundant research into acute affects, few primary or other studies have determined the chronic effect of mercury to invertebrates. Considering primary studies only, MeHg and inorganic Hg reduced the survival (EC_{50} survival) of *Daphnia magna* over a period of 21 days at 0.26 µg Hg/L (as MeHg) and 2.7 µg Hg/L (as $HgCl_2$), respectively (Biesinger et al. 1982).

In flow-through studies where toxicant concentrations were monitored, a MeHg concentration of 0.04 µg Hg/L and an inorganic Hg concentration of 1.3 µg Hg/L impaired reproduction of the water flea, *D. magna* in that significantly fewer young were produced compared with the control (Biesinger et al. 1982). The same researchers conducted static-renewal tests that resulted in significant loss of mercury compounds (unlike flow-through tests), and reported effects at 1-2 µg Hg/L for both MeHg and $HgCl_2$. For $Hg(NO_3)_2$, a MATC of 0.73 µg Hg/L was recommended for the cladoceran *Ceriodaphnia dubia* (Spehar and Fiandt 1986).

Two studies of the effect of MeHg on invertebrates were ranked as unacceptable studies because they were static bioassays with unmeasured mercury concentrations. Static bioassays typically

err on the high side because of contaminant losses (volatilisation and adherence to container walls). In this case, the nominal concentrations of 0.1µg Hg/L (as MeHg) caused a decrease in the the reproductive rate of *D. pulex* over 30 days (Chen and McNaught 1992), and 0.03 µg Hg/L caused malformations and decreased fissions in the planarian *Dugesia* sp. (Best et al. 1981). These values are among the lowest MeHg concentrations found to cause toxic effects to invertebrates (see freshwater toxicity table, and review by Wren and Stephenson 1991), and perhaps lend some support to the decreased young production in daphnids found by Biesinger (1982) above, at 0.04 µg Hg/L (as MeHg).

7.2.3 Toxicity to Marine Invertebrates

The beneficial effects of salinity on the toxicity of mercury were demonstrated in a study with the megalopae of the blue crab, *Callinectes sapidus* (McKenney, Jr. and Costlow, Jr. 1981). As salinity was reduced below 20‰, less inorganic Hg (as $HgCl_2$) was required to produce equivalent toxicity among megalopae. This effect is significant for blue crab and other estuarine species that inhabit, migrate through, and use areas of lower salinity for foraging, spawning, and nursery grounds (Beckvar et al. 1996).

The estuarine mysid, *Mysidopsis bahia,* was the most sensitive of the saltwater invertebrates. Over 35 days in flow-through tanks, reproduction and survival were reduced (EC_{50}) at 1.2 µg Hg/L, and 3.5 µg Hg/L (as $HgCl_2$) was acutely toxic in 96-h tests (Gentile et al. 1983; Lussier et al. 1985).

Atlantic slipper limpet (*Crepidula fornicata*) larvae ceased swimming at 10 µg/L and ceased feeding at 6 µg/L, in 96-hr EC tests. Results were based on measured Hg concentrations in flow-through containers. The same author found a 96-h LC_{50} for larval and adult slipper limpets of 60 and 330 µg Hg/L (as $HgCl_2$), respectively (Thain 1984).

The amphipods, *Corophium insidiosum* and *Elasmopus bampo* had 96-h LC_{50} values of twice that reported for the freshwater amphipod, at 20 µg Hg/L (Reish 1993). The same level was found to be both spermiotoxic and embryotoxic to Mediterranean sea urchins, *Paracentrotus lividus* (Warnau et al. 1996).

No studies were located on the toxicity of MeHg to marine invertebrates.

7.3 Toxicity to Amphibians

Since most amphibians have obligate aquatic larval stages, they are susceptible to adverse effects from mercury contamination. The most sensitive of the amphibians were the embryo/larval stage of the narrow-mouthed toad (*Gastrophryne carolinensis*), the spring peeper (*Hyla crucifer*), the leopard frog (*Rana pipiens*) and three other treefrogs, with negative effects indicated at 1.3 - 7.3 µg Hg/L (see Table 4). Acute toxicities ranged from 10 - 68 µg Hg/L for other embryo/larval frogs, and from 44 - 53 µg Hg/L for toad tadpoles (*Bufo melanosticctus*) in 4 to 7 day tests. The marbled salamander (*Ambystoma opacum*) had an 8-d EC_{50} (survival) of

110 µg Hg/L (Birge et al. 1979). All amphibian studies were ranked as secondary because the mercury concentrations in the treatment vessels were nominal (not measured).

While most of the chronic bioassays were long-term survival tests, one study examined sublethal effects of MeHg to various early life stages of the leopard frog. In a study by Dial (1976), concentrations of 5-10 µg Hg/L (as MeHg) over five days caused poor tail growth and overall impaired development compared with controls in embryos at the blastula, gastrula and neuralplate stages. Death was caused in a matter of hours by moderately higher MeHg concentrations (Dial 1976).

The effects of mercury and other metals on amphibians were reviewed by Freda (1991).

7.4 Toxicity to Macrophytes and Algae

Mercury and methylmercury are not considered to be particularly acutely toxic to macrophytes and algae, although data are limited. Common effects observed following chronic exposure include decreased photosynthesis, and increased incidence of abnormalites and mortality.

7.4.1 Freshwater Macrophytes and Algae

Except for the periphyton (algae attached to substratum) community studies by Sigmon et al. (1977) and Kania et al. (1976), the chronic effects of inorganic mercury have been examined rarely on North American aquatic plants. In an 11-month study of periphyton colonization of artificial streams, Sigmon et al. (1977) found that 1 µg Hg/L caused decreased algal numbers, standing stock and diversity. Similarly, Kania et al. (1976) reported decreased colonization and diversity of periphyton at 5 µg Hg/L and decreased macrophyte biomass (*Juncus* sp.) at 1 µg Hg/L. Both of these reports resulted from high quality research efforts involving flow-through streams, use of $HgCl_2$, measured mercury concentrations, and replicate treatments. The macrophyte, *Elodea densa*, exposed for 25 d to a radioactive MeHg concentration of 0.15 µg Hg/L had structurally damaged chloroplasts (Mortimer and Czuba 1982). Although a flow-through study, the authors did not report measured concentrations of merucry or information on water quality.

Early studies often compared the toxicity of various forms of merucry. At a concentration of 1 µg/L, each of four organomercurial fungicides reduced photosynthesis in natural populations of phytoplankton by 40-60%; diphenyl mercury was the least toxic while phenyl mercuric acetate (PMA) was the most toxic (Harriss et al. 1970). PMA was shown also to be more toxic than $HgCl_2$ in a test that examined growth inhibition in *Chlamydommonas* sp., *Chlorella* sp., and *Phaeodactylum tricornutum* (Nuzzi 1972). At PMA concentrations of 3 to 9 µg Hg/L, *P. tricornutum* was highly vaculated and ovid in shape compared to the controls. *Chlorella* sp. formed giant, morphologically aberrant cells at a PMA concentration of 0.9 µg Hg/L.

Toxicity of mercury may be modified by water chemistry. For example, Chen (1994i) found that inorganic Hg toxicity to *Selenastrum* decreased with increasing orthophosphate concentrations.

In that case, growth in a chemostat was affected at 5 µg Hg/L with a PO_4^{3-} level of 1.2 µg/L, but was unaffected by up to 44 µg Hg/L with a PO_4 level of 167 µg/L. Rai and Dey (Rai and Dey 1980) found that orthophosphate and calcuim each reduce toxicity of methylmercury on *Chlorella vulgaris*, but that alkaline pH enhances toxicity. The toxicity of $HgCl_2$ and $Hg(NO_3)_2$ to diatoms *Fragilaria crotonensis* and *Asterionella formosa* was reduced by the presence of undefined soil extracts in the test solution compared to controls (Tompkins and Blinn 1976). *F. crotonensis* was most sensitive, with complete inhibition of growth ocurring at a mercury concentrations of 0.5 µg/L regardless of compound and, whether or not soil extracts were present.

7.4.2 Marine Macrophytes and Algae

With one exception, unicellular algae were more sensitive to inorganic Hg than macrophytic algae. In 72-hour tests that measured growth (EC_{50}), the coccolithophore (*Emiliania huxleyi*) was the most sensitive of four unicellular species tested at 0.16 µg Hg/L. A cyanophyte (*Oscillatoria woronichinii*) and a diatom (*Thalassiosira pseudonana*) were somewhat less sensitive with EC_{50}s for growth of 0.40 and 0.63 µg Hg/L, respectively. The chlorophyte, *Dunaliella teriolecta*, was very resistant with an EC_{50} for growth of 1005 µg Hg/L (Fisher et al. 1984). These tests were conducted under static conditions of moderately high salinity (Mediterranean Sea water; 36‰), at 17°C, and nominal Hg concentrations. Complimentary uptake experiments using radiolabelled ^{203}Hg found that up to 17% of the Hg adhered to the glass walls of the test vessel (Fisher et al. 1984).

Five macrophytic brown algae (at least 3 of which occur in North America), had EC_{50} (growth) values of 100 to 200 µg Hg/L. The tests were carried out under flow-through conditions over ten days, at relatively cold temperatures, of 4-8 °C (Stromgren 1980). Hg concentrations were nominal (see Table 5).

Chronic toxicity (defined as reduced population growth) in a marine diatom exposed to inorganic Hg, MeHg, di-MeHg, and elemental Hg, was related to the aqueous concentration of a single mercury species (the chloride species $HgCl_2$ or CH_3HgCl). Toxicity was not related to the total mercury or free mercury ion concentration (Mason et al. 1996). Approximately the same concentration of MeHg and inorganic Hg (16-20 µg Hg/L) reduced diatom growth by 50% because the neutral chloride complexes cross cellular barriers with the same efficiency. The toxic concentrations were two or more orders of magnitude higher than are typically reported in aquatic environments (Bloom et al. 1991).

Since the form of MeHg in experiments in low salinity water is in the form of a neutral permeable complex, while inorganic Hg is not, MeHg is often found to be about 30 times more toxic than its inorganic counterpart. The proportion of $HgCl_2$, like toxicity, decreases from 25% to 7.5% between salinities of 10‰ and 20‰, hence growth rate inhibition in this marine diatom was directly related to the concentration of $HgCl_2$, independent of total Hg (Mason et al. 1996). Elemental and di-MeHg were neither accumulated nor toxic to the diatom.

7.5 Toxicity to Micro-organisms

Microbial communitites collected from Chesapeake Bay and Baltimore Habor were inhibited at concentrations of 0.5 and <1 µg Hg/L for MeHg and $HgCl_2$, respectively (Jonas et al. 1984). Endpoints in this study included thymidine incorporation, glutamate incorporation, glutamate respiration, and viability.

Table 4: Summary of freshwater toxicity studies - mercury and methylmercury

Species (common name)	Species (scientific name)	Endpoint	Effective Hg Concentration[28] (µg Hg/L)	Duration of Exposure	Life Stage	Test Type * See endnote	Test Conditions					Comments	Reference
							Temp. (°C)	DO (mg/L)	Hardness (mg/L)	Alkalinity (mg/L)	pH		
ACUTE STUDIES													
Fish - Acute studies ranked primary:													
Brook trout	*Salvelinus fontinalis*	LC_{50}	75 MeHgCl	96 h	Yearlings 50-70g	F,M, 2 reps[29]	12	7.7	45	42	8	Acute/chronic ratio (A/C) = 81; Maximum Acceptable Toxicant Concentration (MATC) = 29-93	(McKim et al. 1976)
Coho salmon	*Oncorhynchus kisutch*	LC_{50}	240	96-h	Yearlings	R,M	10	9.8	93	72	7.3	No Hg loss detected	(Lorz et al. 1978)
Fathead minnow	*Pimephales promelas*	LC_{50}	150	96-h	30 d	F,M, 2 reps	26	93%	42.8	41.2	7.5	Lake Superior water used	(Call et al. 1983)
Fathead minnow	*Pimephales promelas*	LC_{50}	168 74	96-h 7-day	Juvenile	F,M 2 reps	23	86±5%	46	43	7	--	(Snarski and Olson 1982a)
Fathead minnow	*Pimephales promelas*	LC_{50}	172	96-h	30 d	F,M, 2 reps	25	70+%	44	42	7	Lake Superior water used	(Spehar and Fiandt 1986)
Lamprey	*Petromyzon sp.*	LC_{50} LC_{50} LC_{50}	88 @ 4°C MeHg 48 @ 12 °C MeHg 44 @ 20 °C MeHg	96-h 96-h 29-h	Larval	F,M	4,12 & 20	>70%	145	150	8	TP-35 µg/L; No replicates. MeHgCl used. Temperature effect.	(Mallatt et al. 1986c)
Rainbow trout	*Oncorhynchus mykiss*	LC_{50}	24 MeHgCl	96-h	31-44 g	F,M						--	(Lock and Van Overbeeke 1981)
Rainbow trout	*Oncorhynchus mykiss*	LC_{50}	275	96-h	31-44 g	F,M						--	(Lock and Van Overbeeke 1981)
White sucker	*Catostomas commerson*	LC_{50} LC_{50}	687 830	96-h 48-h	20 g fish	F,M 2 reps	12	7.9	18	6.4 mg $CaCO_3$	6	7 ng/L background Hg	(Duncan and Klaverkamp 1983)
Invertebrates - Acute studies ranked primary:													
Crayfish	*Faxonella clypeata*	LC_{50}	20	4-d	Adult males	R,M	20	--	--	--	--	"pond water" used. Units reported incorrectly as µg/L.	(Heit and Fingerman 1977)
Crayfish	*Procambarus clarki*	LC_{50}	200	3-d	Adult males	R,M	20	--	--	--	--	"pond water" used. Units reported incorrectly as µg/L.	(Heit and Fingerman 1977)

[28] Compound tested is $HgCl_2$ unless noted otherwise.

[29] Reps = replicates, where 0 reps is no replication (single vessel). Sometimes reps were indicated in the paper but the number of reps was not given. Two or more replicates are preferable as a component of good quality assays.

Species (common name)	Species (scientific name)	Endpoint	Effective Hg Concentration[28] (µg Hg/L)	Duration of Exposure	Life Stage	Test Type * See endnote	Test Conditions					Comments	Reference
							Temp. (°C)	DO (mg/L)	Hardness (mg/L)	Alkalinity (mg/L)	pH		
Midge larvae	*Chironomus riparius*	LC_{50} LC_{50}	100 316	96-h 48-h	4th instar	F,M 0 reps	20	--	50	--	7	BCF = 12600	(Rossaro et al. 1986)
Water flea	*Ceriodaphnia dubia*	LC_{50}	8.8 $Hg(NO_3)_2$	96-h	<24 h	F,M 10 reps	25	70+%	100	97	8	- see chronic test	(Spehar and Fiandt 1986)
Fish - Acute studies ranked secondary													
Arctic Grayling	*Thymallus arcticus*	LC_{50} LC_{50}	124 218	96-h 96-h	Alevin Juvenile	S,N, 0 reps	12	40+%	41	31	8	Calculated NOEC for juveniles as 2.2 µg Hg/L	(Buhl and Hamilton 1991)
Coho salmon	*Oncorhynchus kisutch*	LC_{50} LC_{50}	282 238	96-h 96-h	Alevin Juvenile	S,N, 0 reps	12	40+%	41	31	8	Calculated NOEC for juveniles as 2.4 µg Hg/L	(Buhl and Hamilton 1991)
Fathead minnow	*Pimephales promelas*	LC_{50} LC_{50}	190 Hg^{2+} acetate 150 Hg-hiocyanate	96-h	3.2-4.2 cm	S,M, 2 reps	22	Aerated	48	35	8	Conductivity 140 µmhos/cm. Nominal Hg.	(Curtis et al. 1979)
Rainbow trout	*O. mykiss*	LC_{50} LC_{50}	217 193	96-h 96-h	Alevin Juvenile	S,N, 0 reps	12	40+%	41	31	8	Calculated NOEC for juveniles as 1.9 µg Hg/L	(Buhl and Hamilton 1991)
Rainbow trout	*O. mykiss*	LC_{50}	220	96-h	--	F,N, 0 reps	15	--	27.2 Ca	90	8	TDS 130 mg/L. Examined temperature effect.	(MacLeod and Pessah 1973)
Rainbow trout	*O. mykiss*	LC_{50} LC_{50}	24 MeHg 42 MeHg	96-h 96-h	Fry Fingerlg	R,N, 3 reps	10	8	101	70	9	TDS 190 mg/L.	(Wobeser 1975)
Rainbow trout	*O. mykiss*	LC_{50}	280	96-h	--	F,N, 0 reps	10	--	27.2 Ca	90	8	TDS 130 mg/L. Examined temperature effect.	(MacLeod and Pessah 1973)
Rainbow trout	*O. mykiss*	LC_{50}	400	96-h	--	F,N, 0 reps	5	--	27.2 Ca	90	8	TDS 130 mg/L. Examined temperature effect.	(MacLeod and Pessah 1973)
Rainbow trout	*O. mykiss*	LC_{50} LC_{50}	84 MeHg 125 MeHg	24-h 24-h	Fry Fingerlg	R,N, 3 reps	10	8	101	70	9	TDS 190 mg/L. Short exposure.	(Wobeser 1975)
Rainbow trout	*O. mykiss*	LC_{50}	900	24-h	Fingerlg	R,N, 3 reps	10	8	101	70	9	TDS 190 mg/L. Short exposure.	(Wobeser 1975)
Invertebrates - Acute studies ranked secondary													
Amphipod	*Gammarus sp.*	LC_{50}	10 90	96-h 24-h	--	S,N	17	6	50	--	8	Some Hg measured, not reported. Few details.	(Rehwoldt et al. 1973)
Caddis fly	Trichopteran species (unnamed)	LC_{50}	1200 5600	96-h 24-h	--	S,N	17	6	50	--	8	Some Hg measured, not reported. Few details. Older study.	(Rehwoldt et al. 1973)
Copepod	*Cyclops sp.*	LC_{50}	2200	48-h	--	S,N, reps	10	--	--	0.58 meq/L	7	Conductivity = 75 mg/L	(Baudouin and Scoppa 1974)
Copepod	*Cyclops sp.*	LC_{50}	600	48-h	NR	S,M	--	--	"soft"	"low"	6	Measured [Hg] not reported.	(Abbasi et al. 1988)
Copepod	*Eudiaptomus sp.*	LC_{50}	850	48-h	--	S,N, reps	10	--	Cond. 75	0.58 meq/L	7	--	(Baudouin and Scoppa 1974)

Species (common name)	Species (scientific name)	Endpoint	Effective Hg Concentration[28] (µg Hg/L)	Duration of Exposure	Life Stage	Test Type * See endnote	Test Conditions					Comments	Reference
							Temp. (°C)	DO (mg/L)	Hardness (mg/L)	Alkalinity (mg/L)	pH		
Midge	*Chironomus sp.*	LC_{50}	20 60	96-h 24-h	Larvae	S,N	17	6	50	--	8	Some Hg measured, not reported. Few details. Older study.	(Rehwoldt et al. 1973)
Midge	*Chironomus tentans*	LC_{50}	29	48-h	Larvae	S,N 2 reps	14	7	25	25	6	--	(Khangarot and Ray 1989a)
Mosquito	*Aedes sp.*	LC_{50}	290	48-h	Larvae	S,M	--	--	"soft"	"low"	6	Measured [Hg] not reported.	(Abbasi et al. 1988)
Mussel	*Anodonta imbecilis*	LC_{50}	147 216 88 (mix 148 Cr)	96-h 48-h 96-h	2 d old Juvenile	S,N, 2 reps	23±3	--	"soft" (see also "hard")	--	--	No chemical measurements. Cr+Hg as a mixture in test #3.	(Keller and Zam 1991)
Mussel	*Anodonta imbecilis*	LC_{50}	171 233	96-h 48-h	2 d old Juvenile	S,N, 2 reps	23±3	--	"hard" (see also "soft")	--	--	No chemical measurements.	(Keller and Zam 1991)
Oligochaete	*Nais sp.*	LC_{50}	1000 1900	96-h 24-h	--	S,N	17	6	50	--	8	Some Hg measured, not reported. Few details. Older study.	(Rehwoldt et al. 1973)
Snail	*Lymnaea sp.*	LC_{50}	19 26	96-h 48-h	Adults	S,N, reps	32	6	195	160	7	--	(Khangarot and Ray 1988)
Snail	*Amnicola* sp.	LC_{50}	2100 6300	96-h 24-h	Eggs (see adults)	S,N	17	6	50	--	8	Some Hg measured, not reported. Few details. Older study.	(Rehwoldt et al. 1973)
Snail	*Amnicola* sp.	LC_{50}	80 1100	96-h 24-h	Adults (see eggs)	S,N	17	6	50	--	8	Some Hg measured, not reported. Few details. Older study.	(Rehwoldt et al. 1973)
Water flea	*D. magna*	LC_{50}	5	48-h	Adult	S,N, 2 reps	18	9	48	45	8	--	(Biesinger and Christensen 1972)
Water flea	*D. magna*	LC_{50}	5.2 8.1	48-h 24-h	--	S,N 2 reps	13	6	240	400	8	Ca = 152 mg/L, Mg = 92 mg/L.	(Khangarot and Ray 1987a)
Water flea	*D. magna*	LC_{50}	5.2	48-h	Adults	S,N, 3 reps	13	6	240	400	8	Hard water.	(Khangarot and Ray 1989b)
Water flea	*D. hyalina*	LC_{50}	5.5	48-h	--	S,N, reps	10	--	Cond. 75	0.58 meq/L	7	--	(Baudouin and Scoppa 1974)
Amphibians - Acute studies ranked secondary													
Black-spined toad	*Bufo melanostictus*	LC_{50}	43.6 52.8	96-h 24-h	~2 cm tadpoles	S,N, 3 reps	31	6.4	185	135	7.4	Cond. 950.	(Khangarot and Ray 1987b)
Leopard frog	*Rana pipiens*	LC_{50}	20 MeHg 12 calculated	5-d	Blastula gastrula Embryo	R,N, reps	21	--	--	--	--	--	(Dial 1976)

Species (common name)	Species (scientific name)	Endpoint	Effective Hg Concentration[28] (µg Hg/L)	Duration of Exposure	Life Stage	Test Type * See endnote	Test Conditions					Comments	Reference
							Temp. (°C)	DO (mg/L)	Hardness (mg/L)	Alkalinity (mg/L)	pH		
Plants and Algae - Acute studies ranked secondary													
Blue green	*Anacystis nidulans*	LC_{50}	145	24-h	--	S,N, reps	24	Saturated	--	--	7	Cell death/bleaching.	(Thomas and Montes 1978)
Blue green	*Anabaena flos-aquae*	LC_{50}	3.5 MeHg	24-h	--	S,N, reps	24	Saturated	--	--	7	Cell death/bleaching.	(Thomas and Montes 1978)
Blue green	*Anacystis nidulans*	LC_{50}	6.3 MeHg	24-h	--	S,N, reps	24	Saturated	--	--	7	Cell death/bleaching.	(Thomas and Montes 1978)
Blue green	*Anabaena flos-aquae*	LC_{50}	72	24-h	--	S,N, reps	24	Saturated	--	--	7	Cell death/bleaching.	(Thomas and Montes 1978)
Green alga	*Selenastrum captricornutum*	LC_{50}	9 batch test 27 chemostat	24-h	--	S,N, 3rep F,N, 0rep	24	--	--	--	--	Batch test more sensitive for Hg unlike other metals.	(Chen and Lin 1997)
Fish - Acute studies ranked unacceptable													
Goldfish	--	LC_{50} LC_{50}	350 440 with equivalent Se	48-h	Adults	S,N, 0 reps	25	--	100 mg/L $CaCO_3$ add	--	--	Selenium effect	(Heisinger et al. 1979)
Invertebrates - Acute studies ranked unacceptable													
Planarian	*Dugesia sp.*	LC_{50}	>200 MeHg	5-d (120-h)	16 mm	S,N, No reps	22	--	--	--	--	--	(Best et al. 1981)
Water flea	*D. pulex*	LC_{50}	1.8 MeHg 5.7 MeHg 31.2 MeHg	96-h 48-h 24-h	<24 h	S,N, 5 reps	20	--	--	--	--	--	(Chen and McNaught 1992)
Water flea	*D. magna & pulex*	LC_{50}	3.2	48-h	<24 h	S,N, reps	--	--	--	--	--	--	(Canton and Adema 1978)
Water flea	*D. magna*	NOAEL LT_{50}	7.5 --	Varied 24-120 h	Adults	S,N, reps	20 - 25	--	#1: 46 #2: 119	--	--	Time to 50% lethality. Hardness increased LT. Poor description.	(Brkovic-Popovic 1990)
CHRONIC STUDIES **Fish - Chronic studies ranked primary:**													
Brook trout	*Salvelinus fontinalis*	NOAEL deformities & survival EC_{50} deformities & survival	0.29 MeHgCl 0.93 MeHgCl	39 weeks	Yearlings 50-70g	F,M, 2 reps	9	7.7	45	42	8	2nd gen deformities; death in <108 weeks to females.	(McKim et al. 1976)
Brook trout	*Salvelinus fontinalis*	EC_{50} weight loss	0.93 MeHgCl	21-days	Alevin	F,M 2 reps	18	9	48	45	8	- other effects were biochemical.	(Christensen 1975)

Species (common name)	Species (scientific name)	Endpoint	Effective Hg Concentration[28] (µg Hg/L)	Duration of Exposure	Life Stage	Test Type * See endnote	Test Conditions					Comments	Reference
							Temp. (°C)	DO (mg/L)	Hardness (mg/L)	Alkalinity (mg/L)	pH		
Channel catfish	*Ictalurus punctatus*	EC_{50} survival	0.3	10-days	Embryo-larval	F,M, 3 reps	29	86-98%	101	65	8	Embryo-larval stages + teratogenic effect	(Birge et al. 1979)
Fathead minnow	*Pimephales promelas*	LOAEL growth & weight loss EC_{50} reproductive impairment EC_{50} survival EC_{50} spinal curve	0.26 0.5 4.5 4.5	60-days	Juvenile	F,M 2 reps	25	86±5%	46	43	7	Acute/chronic ratio = 280; MATC[30] considered to be <0.26 µg Hg/L **CRITICAL STUDY**	(Snarski and Olson 1982a)
Fathead minnow	*Pimephales promelas*	NOAEL hatch & survival	<0.23	5 - 6 days	Egg-larval	F,M 2 rep	25	81%	45.6	41.3	7.5	Lake Superior water -	(Call et al. 1983)
Fathead minnow	*Pimephales promelas*	EC_{50} growth	0.89 $Hg(NO_3)_2$	32-days	30 days old	F,M 2 reps	25	70+%	44	42	7	A/C = 193	(Spehar and Fiandt 1986)
Largemouth Bass	*Micropterus salmoides*	EC_{50} survival	5.3	8-days	Embryo-larval	F,M, 3 reps	18	86-98%	101	65	8	TDS 172 mg/L, Cond 176 µmhos/cm	(Birge et al. 1979)
Invertebrates - Chronic studies ranked primary:													
Water flea	*Daphnia magna*	EC_{50} reproduction	<0.04 MeHg	21-d	<24 h	F,M, 4 reps	18	--	45	42	8	Detection Limit = 0.01. "<" =lowest level tested caused significantly fewer young than control were produced.	(Biesinger et al. 1982)
Water flea	*Daphnia magna*	EC_{50} survival	>0.26 MeHg	21-d	<24 h	F,M, 4 reps	18	--	45	42	8	--	(Biesinger et al. 1982)
Water flea	*Daphnia magna*	EC_{50} reproduction	1.28	21-d	<24 h	F,M, 4 reps	18	--	45	42	8	--	(Biesinger et al. 1982)
Water flea	*Daphnia magna*	EC_{50} survival	2.7	21-d	<24 h	F,M, 4 reps	18	--	45	42	8	--	(Biesinger et al. 1982)
Water flea	*Ceriodaphnia dubia*	EC_{50} reproduction	12	7-d	<24 h	R,M 10 reps	25	70+%	100	97	8	Higher value than for acute study may be food effect.	(Spehar and Fiandt 1986)
Plants and Algae - Chronic studies ranked primary:													
Macrophyte	*Juncus sp.*	EC decreased biomass	1	11 mos.	--	F,M 2 reps	~23	Saturated	--	--	5-6	DOC < 0.5 mg/L. Inverts & fish present.	(Kania et al. 1976)

[30] MATC=maximum acceptable toxicant concentration

Species (common name)	Species (scientific name)	Endpoint	Effective Hg Concentration[28] (µg Hg/L)	Duration of Exposure	Life Stage	Test Type * See endnote	Test Conditions					Comments	Reference
							Temp. (°C)	DO (mg/L)	Hardness (mg/L)	Alkalinity (mg/L)	pH		
Periphyton community	--	EC decreased algal #s, St. stock & diversity	1	11 mos.	--	F,M 2 reps	22	Saturated	--	--	6	DOC < 0.5 mg/L. cond. 26 µmhos/cm. See Kania study.	(Sigmon et al. 1977)
Periphyton community	--	EC decreased colonization & diversity	5	11 mos.	--	F,M 2 reps	~23	Saturated	--	--	5-6	DOC < 0.5 mg/L. Inverts & fish present.	(Kania et al. 1976)
Fish - Chronic studies ranked secondary:													
Brook trout	*Salvelinus fontinalis*	EC_{50} survival, deformity	1.0 MeHgCl	2-years	Egg - Adult	F,U 2 reps	18	9	48	45	8	Rigour of study details for the full duration not clear.	(Christensen 1975)
Brook trout	*Salvelinus fontinalis*	EC_{50} biochemical changes	2.9 MeHgCl	14-d	Yearlings	F,U 2 reps	9	7.7	45	42	8	Ecological relevance? Rigour of study details for the full duration not clear.	(Christensen 1975)
Coho salmon	*O. kisutch*	EC_{50} survival	63 MeHg OH	48-d	Embryo	R,N, 3 reps	10	--	--	--	--	Hg measured in stock. No chemistry	(Devlin and Mottet 1992)
Mosquitofish	*Gambusia sp.*	EC survival, reproduction	<42 18	60-d 111-d	Fry to adult	S,M, 2 reps	18	9.1	13 mg Ca	--	7	DOC: 4 mg/L, other chemistry given.	(Mulvey et al. 1995)
Rainbow trout	*O. mykiss*	EC_{50} biochemical changes	2 - 10	42-d	85-250 g	S,M 0 reps	11	7.5	106	--	--	100 v 6 mg Cl/L (as NaCl) tested. Reduced blood protein, albumin, etc.	(Walczak et al. 1986)
Rainbow trout	*O. mykiss*	EC_{100} survival	<0.1 Test repeated 3 times.	8-days	Egg-embryo	F,M, 3 reps	14	10	101	65	8	83-88% survival in controls - complete mortality in tests. One concentration used (no range) - no LOAEL.	(Birge et al. 1979)
Rainbow trout	*O. mykiss*	EC_{50} food avoidance	0.2	20-min	30-50 mm	F,M, 2 reps	17	90%	112	63	8	Cond. 136 µmhos/cm. Short duration.	(Birge et al. 1993)
Rainbow trout	*O. mykiss*	EC_{50} survival	>4 MeHg	~60-d	Sub-adult	S,N, 0 reps	15	8-9	135	--	8	Charcoal filtered water. MeHg ~15X more toxic than inorganic Hg	(Niimi and Kissoon 1994h)
Rainbow trout	*O. mykiss*	EC_{50} biochemical changes	5	4 & 14-d	20-175 g	F,M	15	9.8	133	--	8	Metallothionein increased, other biochemical changes. Cond. 280, Cl 32 mg/L.	(Angelow and Nicholls 1991)
Rainbow trout	*O. mykiss*	EC_{50} survival	>64	~60-d	Sub-adult	S,N, 0 reps	15	8-9	135	--	8	Charcoal filtered water	(Niimi and Kissoon 1994h)
Invertebrates - Chronic studies ranked secondary:													
Water flea	*D. magna*	EC_{50} survival	0.87 MeHg	21-d	<24 h	R,M 4 reps	18	--	45	42	8	--	(Biesinger et al. 1982)
Water flea	*D. magna*	EC_{50} repro-duction	1.14 MeHg	21-d	< 24 h	R,M 4 reps	18	--	45	42	8	--	(Biesinger et al. 1982)
Water flea	*D. magna*	EC_{50} repro-duction	1.82	21-d	< 24 h	R,M 4 reps	18	--	45	42	8	--	(Biesinger et al. 1982)

Species (common name)	Species (scientific name)	Endpoint	Effective Hg Concentration[28] (µg Hg/L)	Duration of Exposure	Life Stage	Test Type * See endnote	Test Conditions					Comments	Reference
							Temp. (°C)	DO (mg/L)	Hardness (mg/L)	Alkalinity (mg/L)	pH		
Water flea	*D. magna*	EC_{50} reproduction	3.4	21-d	Adults	S,N 2 reps	18	9	48	45	8	Na 1.1 mg/L. Impaired reprod. Older study.	(Biesinger and Christensen 1972)
Water flea	*D. magna*	EC_{50} survival	3.5	21-d	<24 h	R,M 4 reps	18	--	45	42	8	--	(Biesinger et al. 1982)
Amphibians - Chronic studies ranked secondary (no primary studies):													
Barking treefrog	*Hyla gratiosa*	EC_{50} survival	2.5	7-d	Embryo-larval	R,N, reps	21	Saturated	100	--	7.5	Unmeasured.	(Birge et al. 1979)
Fowler's toad	*Bufo fowleri*	EC_{50} survival	66	7-d	Embryo-larval	R,N, reps	21	Saturated	100	--	7.5	Unmeasured.	(Birge et al. 1979)
Green toad	*Bufo debilis d.*	EC_{50} survival	40	7-d	Embryo-larval	R,N, reps	21	Saturated	100	--	7.5	Unmeasured.	(Birge et al. 1979)
Grey treefrog	*Hyla versicolor*	EC_{50} survival	2.6	7-d	Embryo-larval	R,N, reps	21	Saturated	100	--	7.5	Unmeasured.	(Birge et al. 1979)
Leopard frog	*Rana pipiens*	EC_{50} teratogenesis	5 MeHg 8 calculated	5-d	Blastula gastrula Embryo	R,N, reps	21	--	--	--	--	--	(Dial 1976)
Leopard frog	*Rana pipiens*	EC_{50} survival	7.3	7-d	Embryo-larval	R,N, reps	21	Saturated	100	--	7.5	Unmeasured.	(Birge et al. 1979)
Marbled salamander	*Ambystoma opacum*	EC_{50} survival	110	8-d	Embryo-larval	R,N, reps	20	Saturated	100	--	7.5	Unmeasured.	(Birge et al. 1979)
Narrow mouthed toad	*Gastrophryne carolinensis*	EC_{50} survival	1.3	7-d	Embryo-larval	R,N, reps	21	Saturated	100	--	7.5	Unmeasured.	(Birge et al. 1979)
Northern cricket frog	*Acris crepitans*	EC_{50} survival	10.4	7-d	Embryo-larval	R,N, reps	21	Saturated	100	--	7.5	Unmeasured.	(Birge et al. 1979)
Pig frog	*Rana grylio*	EC_{50} survival	67.2	7-d	Embryo-larval	R,N, reps	21	Saturated	100	--	7.5	Unmeasured.	(Birge et al. 1979)
Red-spotted toad	*Bufo punctatus*	EC_{50} survival	36.8	7-d	Embryo-larval	R,N, reps	21	Saturated	100	--	7.5	Unmeasured.	(Birge et al. 1979)
River frog	*Rana heckscheri*	EC_{50} survival	60	7-d	Embryo-larval	R,N, reps	21	Saturated	100	--	7.5	Unmeasured.	(Birge et al. 1979)
Southern gray treefrog	*Hyla chrysoscelis*	EC_{50} survival	2.4	7-d	Embryo-larval	R,N, reps	21	Saturated	100	--	7.5	Unmeasured.	(Birge et al. 1979)
Spring peeper (treefrog)	*Hyla crucifer*	EC_{50} survival	2.8	7-d	Embryo-larval	R,N, reps	21	Saturated	100	--	7.5	Unmeasured.	(Birge et al. 1979)
Squirrel treefrog	*Hyla squirella*	EC_{50} survival	2.4	7-d	Embryo-larval	R,N, reps	21	Saturated	100	--	7.5	Unmeasured.	(Birge et al. 1979)
Plants and Algae - Chronic studies ranked secondary:													
Green alga	*Chlorella vulgaris*	EC_{50} survival	10 MeHg	15-d	--	S,N, reps	24	--	--	--	Var.	P, Ca, Mg varied. Decreased tox at high pH.	(Rai et al. 1981)

Species (common name)	Species (scientific name)	Endpoint	Effective Hg Concentration[28] (µg Hg/L)	Duration of Exposure	Life Stage	Test Type * See endnote	Test Conditions					Comments	Reference
							Temp. (°C)	DO (mg/L)	Hardness (mg/L)	Alkalinity (mg/L)	pH		
Green alga	*Chlorella vulgaris*	EC_{50} survival	400	15-d	--	S,N, reps	24	--	--	--	Var.	P=10-40, Ca, Mg varied. Decreased tox at high pH.	(Rai et al. 1981)
Green alga	*Selenastrum captricornutum*	EC_{50} reduced growth	5 (1.2 PO_4) 28 (16.2 PO_4) 44 (164 PO_4)	24-h	--	F,N, reps	24	--	--	--	--	Varied orthophosphate in chemostat.	(Chen 1994i)
Diatom	*Fragilaria crotonenis*	2-4 fold reduction in population growth; abnormal colony formation	50 as $HgCl_2$ 50 as $Hg(NO_3)_2$	12-d	--	S,N, 3 reps	14 stock culture	--	-	--	7.5-7.7	*Asterionella formosa* tested also but less sensetive; presence of soil extract decreased toxicity for both species	(Tompkins and Blinn 1976)
Fish - Chronic studies ranked unacceptable:													
Catfish	*Clarias batrachus*	EC ovary maturity	40 MeHg 50	180-d 90-d	Adults	S,N, 0 reps	16-22	8	23.2	--	7	Ovaries did not mature	(Kirubagaran and Joy 1988)
Guppies	*Poecilia sp.*	EC reprod-impairment	1.8 MeHg	3-mos	4-weeks	R,M	--	--	--	--	--	Male reproductive impairment. Hg measured/ chemistry not reported.	(Wester 1991)
Knifefish	*Notopterus notopterus*	EC blood chemistry	88	15-60 days	--	R,N	--	--	--	--	--	Asian species; Little info given. Blood glc, lactate, etc. changes.	(Verma et al. 1986)
Invertebrates - Chronic studies ranked unacceptable:													
Planarian	*Dugesia sp.*	EC_{50} malform-ations	0.03 MeHg	14-d	16 mm	S,N, No reps	22	--	--	--	--	Malformations and decreased fissions.	(Best et al. 1981)
Water flea	*D. pulex*	EC_{50} Birth rate	>0.1 MeHg	30-d	<24 h	S,N, reps	20	--	--	--	--	--	(Chen and McNaught 1992)
Plants and Algae - Chronic studies ranked unacceptable:													
Green alga	*Ankistrodesmus* sp.	EC reduced growth	1000	12-d	--	S,N, 2 reps	20	--	--	--	5-7	--	(Baker et al. 1983)
Green alga	*Chlamydomonas variabilis*	EC delayed growth	2 (10^{-8} M) PMA, phenyl Hg acetate	14-d	--	S,N, 3 reps	23	--	--	--	--	Unusual Hg form.	(Delcourt and Mestre 1978)
Macrophyte	*Elodea densa*	EC chloroplast disruption	0.15 MeHg	25-d	--	F,?M/N, 0 reps	--	--	--	--	--	Used Ottawa municipal water.	(Mortimer and Czuba 1982)
Macrophyte	*Elodea densa*	EC reduced growth	0.19-6.17 or 0.16-12.51 MeHg	17-d	--	F,M, pseudo reps	19	--	--	--	--	Controls not mentioned; MeHg more toxic than $HgCl_2$ Used Ottawa municipal water.	(Mortimer and Kudo 1975)

Species (common name)	Species (scientific name)	Endpoint	Effective Hg Concentration[28] (µg Hg/L)	Duration of Exposure	Life Stage	Test Type * See endnote	Test Conditions					Comments	Reference
							Temp. (°C)	DO (mg/L)	Hardness (mg/L)	Alkalinity (mg/L)	pH		
Phyto-plankton	natural population	photo-synthesis (40-60% reduction)	1 four mercurials	5-d	--	S,N, ? reps	25	--	--	--	--	diphenylmercury least toxic	(Harriss et al. 1970)
Phyto-plankton	*Chlorella vulgaris*	53.8% suvival (no. of colonies)	0.0001	15-d	--	agar plates,N, ? reps	25	--	--	--	--	--	(Rai and Dey 1980)
Phyto-plankton	*Chlorella* sp.	morpho-logical abnormalities	0.9 as PMA	16-d	--	S,N ? reps	18	--	--	--	--	--	(Nuzzi 1972)
Phyto-plankton	*Phaeodactylum tricornutum,*	growth inhibition	0.06 as PMA	16-d	--	S,N ? reps	18	--	--	--	--	$HgCl_2$ tested also, but few details given; less toxic than PMA	(Nuzzi 1972)
Phyto-plankton	*Phaeodactylum tricornutum,*	morpho-logical abnormalities	3.0-9.0 as PMA	16-d	--	S,N ? reps	18	--	--	--	--	--	(Nuzzi 1972)
Phyto-plankton	*Synura, Nitzschia, Chlamydomonas*	EC reduced growth	0.05 per 10^4 cells/L - only 1 Hg conc. used.[31]	14-d	--	S,N, 3 reps	20	--	--	--	--	Temperature stress increased Hg effect using 15, 25,30. Hg measured at conclusion, sign.loss.	(Knowles and Zingmark 1978)

*Indicates: static (S), flow-through (F), daily renewal ®, nominal/unmeasured (N), measured conc.(M), reps (replicates). Endpoint abbreviations: chronic or sub-chronic effect concentration (EC), acute or lethal endpoint concentration (LC).

Note: The data are organised alphabetically by common name within each group. This table provides a summary of the available toxicological information for the compound. It is not intended to detail every study, but rather highlight the most important studies. It includes the studies with the lowest endpoints (even if the data are unacceptable); endpoint values spans approximately two orders of magnitude above the lowest end-point.

[31] Value reported as given, relation to Effective Concentration not clear.

Table 5: Summary of marine toxicity studies - mercury and methylmercury

Species (common name)	Species (scientific name)	Endpoint	Effective Hg Concentration[32] (µg Hg/L)	Duration of Exposure	Life Stage	Test Type * See endnote	Test Conditions					Comments	Reference
							Temp. (°C)	DO (mg/L)	Salinity	Nitrogen Content	Other Variables		
ACUTE STUDIES													
Fish - Acute studies ranked primary:													
Mummichog or killifish	*Fundulus heteroclitus*	LC_{50} embryo survival LC_{50} embryo survival	50 MeHg 67	96-h	4-8 cell cleavage eggs	R,M	25	--	20 ‰	--	--	Only 10% of Hg lost in 24 hrs; media replaced daily. Results are nominal therefore within 10% of actual.	(Sharp and Neff 1982)
Mummichog or killifish	*Fundulus heteroclitus*	LC_{50} embryo survival	73 MeHg	96-h	1-d old embryos	R,M	25	--	20 ‰	--	--	Only 10% of Hg lost in 24 hrs; media replaced daily. Results are nominal therefore within 10% of actual.	(Sharp and Neff 1982)
Mummichog or killifish	*Fundulus heteroclitus*	LC_{50}	68	96-h	Embryo	R,M	25	--	20 ‰	--	--	25-30% of Hg lost in 24 hrs; media replaced daily. Results based on nominal & are conservative.	(Sharp and Neff 1980)
Invertebrates - Acute studies ranked primary:													
Mysid	*Mysidopsis bahia*	LC_{50}	3.5	96-h	24-h post-larvae	F,M	21±1	--	30 ‰	--	pH 8	Control survival reported; Hg measured 2X weekly	(Lussier et al. 1985)
Slipper limpet	*Crepidula fornicata*	LC_{50}	60 330	96-h 96-h	Larvae Adults	F,M	21±0.6	aerated	29-33 ‰	--	--	Pre-exposed test chambers to Hg. Hg measured daily. Effects calc'd from measured values. Fed algae.	(Thain 1984)
Fish - Acute studies ranked secondary:													
Mummichog or killifish	*Fundulus heteroclitus*	LC_{50}	200 MeHg	96-h	Juvenile	S,N	24	--	20 ‰	--	--	Fish not fed; 2L test beakers covered. Resistance did not carry through a generation.	(Khan and Weis 1987)
Invertebrates - Acute studies ranked secondary:													
Amphipod	*Corophium insidiosum*	LC_{50}	20	96-h	Adults	S,N	19.1	--	--	--	--	Test vessels = petri dishes; Sea water not defined.	(Reish 1993)
Amphipod	*Elasmopus bampo*	LC_{50}	20	96-h	Adults	S,N	19.1	--	--	--	--	Test vessels = petri dishes; Sea water not defined.	(Reish 1993)
Blue mussels	*Mytilus edulis*	LC_{50}	161	96-h	Juvenile	R,N 2 reps	20±2	Not aerated	25±2 ‰	--	pH 6.9-7.5	Controls used. Tests repeated 3-4X.	(Nelson et al. 1988)
Rotifer	*Brachionus plicatilis*	LC_{50}	61	24-h	Neonate	S,N	25	--	15 ‰	--	dark	Hg most toxic of 21 substances. No WQ or Hg measurements. <10% control mortality.	(Snell et al. 1991)
Sea Urchin	*Paracentrotus lividus*	LOEC Embryo-toxicity	20	To 72 hrs	Larval	S,N	17.8 ± 0.5°C	--	38 ‰	--	--	-frequency of development defects on 72-h-old larvae; eggs fertilised with Hg contaminated sperm	(Warnau et al. 1996)

[32] Compound tested is mercuric chloride ($HgCl_2$) unless noted otherwise.

Species (common name)	Species (scientific name)	Endpoint	Effective Hg Concentration[32] (µg Hg/L)	Duration of Exposure	Life Stage	Test Type * See endnote	Test Conditions					Comments	Reference
							Temp. (°C)	DO (mg/L)	Salinity	Nitrogen Content	Other Variables		
Plants and Algae - Acute studies ranked secondary:													
Chlorophyte	*Dunaliella tertiolecta*	LC_{50}	4002	72-h	--	S,N	17±1	--	~36 ‰	--	14:10 light cycle	GFF[33] Mediterranean surface water; Used controls, quantified Hg on glass walls	(Fisher et al. 1984)
Coccolithophore	*Emiliania huxleyi*	LC_{50}	0.32	72-h	--	S,N	17±1	--	~36 ‰	--	14:10 light cycle	GFF Mediterranean surface water; Used controls, quantified Hg on glass walls	(Fisher et al. 1984)
Cyanophyte	*Oscillatoria woronichinii*	LC_{50}	1.0	72-h	--	S,N	17±1	--	~36 ‰	--	14:10 light cycle	GFF Mediterranean surface water; Used controls, quantified Hg on glass walls	(Fisher et al. 1984)
Diatom	*Thalassiosira pseudonana*	LC_{50}	1.0	72-h	--	S,N	17±1	--	~36 ‰	--	14:10 light cycle	GFF Mediterranean surface water; Used controls, quantified Hg on glass walls	(Fisher et al. 1984)
Fish - Acute studies ranked unacceptable:													
Spot (saltwater drum)	*Leiostomus xanthurus*	LC_{50}	36	Unknown	Juvenile	S,N	--	--	--	--	--	Unpublished laboratory data.	(U.S.EPA 1980)
Tidewater silverside	*Menidia peninsulae*	LC_{50}	71	Unknown	Juvenile	S,N	--	--	--	--	--	Unpublished laboratory data.	(U.S.EPA 1980)
Atlantic silverside	*Menidia menidia*	LC_{50}	86	Unknown	Juvenile	S,N	--	--	--	--	--	Unpublished laboratory data.	(U.S.EPA 1980)
Haddock	*Melanogrammus aeglefinus*	LC_{50}	98	Unknown	Larva	S,N	--	--	--	--	--	Unpublished laboratory data.	(U.S.EPA 1980)
Atlantic silverside	*Menidia menidia*	LC_{50}	125	Unknown	Larva	S,N	--	--	--	--	--	Unpublished laboratory data.	(U.S.EPA 1980)
Winter flounder	*Pseudopleuron-ectes americanus*	LC_{50}	1320 - 1810	Unknown	Larva	S,N	--	--	--	--	--	Species mean=1680 µg Hg/L; Unpublished laboratory data.	(U.S.EPA 1980)
Invertebrates - Acute studies ranked unacceptable:													
Soft-shell clam	*Mya arenaria*	LC_{50}	400	Unknown	Adults	S,N	--	--	--	--	--	Cited from (U.S.EPA 1980)	(Eisler and Hennekey 1977)
Plants - Acute studies ranked unacceptable:													
Diatom	*Nitzschia delicatissima*	photosynthesis (<50% of controls)	0.5 two organo-mercurial fungicies	24-h	--	S,N ? reps	25	--	--	--	--	--	(Harriss et al. 1970)

[33] GFF = glass fibre filter

Species (common name)	Species (scientific name)	Endpoint	Effective Hg Concentration[32] (µg Hg/L)	Duration of Exposure	Life Stage	Test Type * See endnote	Test Conditions					Comments	Reference
							Temp. (°C)	DO (mg/L)	Salinity	Nitrogen Content	Other Variables		
CHRONIC STUDIES													
Fish - Chronic studies ranked primary:													
Mummichog or killifish	*Fundulus heteroclitus*	EC_{50} hatching success EC_{50} hatching success EC_{50} hatching success	37 49 55	32-d 5-d 48-h	Embryo	R,M	25	--	20 ‰	--	--	25-30% of Hg lost in 24 hrs; media replaced daily. Results are nominal therefore conservative.	(Sharp and Neff 1980)
Mummichog or killifish	*Fundulus heteroclitus*	EC_{50} spinal deformity EC_{50} spinal deformity EC_{50} spinal deformity	32 47 48	32-d 5-d 48-h	Embryo	R,M	25	--	20 ‰	--	--	25-30% of Hg lost in 24 hrs; media replaced daily. Results are nominal therefore conservative.	(Sharp and Neff 1980)
Invertebrates - Chronic studies ranked primary:													
Blue Crab	*Callinectes sapidus*	NOEC (survival)	10	11-d	Mega-lopae	R,M	25±0.5	--	26-31 ‰	--	12L:12D cycle	Effects more prominent both above & below salinity range. 50 percentiles not given.	(McKenney, Jr. and Costlow, Jr. 1981)
Blue Crab	*Callinectes sapidus*	EC duration of early life stage extended	16 measured (20 nominal)	Control=8d Test=11d	Mega-lopae	R,M	25±0.5	--	20, 30, 40 ‰	--	12L:12D cycle	20-33% Hg lost in 24 hr - measured 5 times/24 hrs.	(McKenney, Jr. and Costlow, Jr. 1981)
Mysid	*Mysidopsis bahia*	EC_{50} reproduction, survival	1.2	Time to maturity, 35 d	Post-larval to mature	F,M	21±1	--	30 ‰	--	pH 8	Control survival reported; Hg measured 2X weekly	(Lussier et al. 1985)
Slipper limpet	*Crepidula fornicata*	EC_{50} cessation of swim/feeding	10 (-swim) 6 (-feeding)	96-h	Larvae	F,M	21±0.6	aerated	29-33 ‰	--	--	Pre-exposed test chambers to Hg. Hg measured daily. Effects calc'd from measured values. Fed algae.	(Thain 1984)
Fish - Chronic studies ranked secondary:													
Chum salmon	*Oncorhynchus keta*	EC_{50} weight growth NOAEL	<5 1	72-d	Fry	F,N	Varied	--	--	--	Meso-cosms	1350 m^3 mesocosms in Saanich Inlet, BC. Hg losses estimated (2%/d); nominal values reported.	(Brown and Parsons 1978)
Mummichog or killifish	*Fundulus heteroclitus*	EC Fertilisation, embryo development	500 MeHg	20 min	Eggs & embryos	S,N	24	--	15 ‰	--	--	Reduced fertilisation, but not embryonic development. Eggs from fish from unpolluted region less tolerant than from a polluted one.	(Khan and Weis 1987)
Invertebrates - Chronic studies ranked secondary:													
American Oyster	*Crassostrea virginica*	EC_{50} survival	12	12-d	Larvae	R,N	25±1	--	24±2 ‰	--	pH 7-8.5	Test seawater measured 0.9 µg/L Hg; no measurements of test media	(Calabrese et al. 1977)
Hard Clam	*Mercenaria mercenaria*	EC_{50} survival	15	10-d	Larvae	R,N	25±1	--	24±2 ‰	--	pH 7-8.5	Test seawater measured 0.9 µg/L Hg; no measurements of test media	(Calabrese et al. 1977)
Sea Urchin	*Paracentrotus lividus*	EC_{50} Spermio-toxic	20	75 min	Sperm	S,N	18	--	38 ‰	--	--	Mediterranean species. Does not occur in N. America, close relative.	(Warnau et al. 1996)

Species (common name)	Species (scientific name)	Endpoint	Effective Hg Concentration[32] (µg Hg/L)	Duration of Exposure	Life Stage	Test Type * See endnote	Test Conditions					Comments	Reference
							Temp. (°C)	DO (mg/L)	Salinity	Nitrogen Content	Other Variables		
Plants and Algae - Chronic studies ranked secondary:													
Brown alga (macrophyte)	*Ascophyllum nodosum*	EC_{50} growth LOAEL	100 to 200 5	10-days	--	F,N	4-8	--	33 ‰	6 µg/L NO_3-N	0.6 µg/L PO_4-P	Found in North America.	(Stromgren 1980)
Brown alga (macrophyte)	*Fucus serratus*	EC_{50} growth LOAEL	100 to 200 5	10-days	--	F,N	4-8	--	33 ‰	6 µg/L NO_3-N	0.6 µg/L PO_4-P	European specie.	(Stromgren 1980)
Brown alga (macrophyte)	*Fucus spiralis*	EC_{50} growth LOAEL	100 to 200 5	10-days	--	F,N	4-8	--	33 ‰	6 µg/L NO_3-N	0.6 µg/L PO_4-P	Found in North America.	(Stromgren 1980)
Brown alga (macrophyte)	*Fucus vesiculosus*	EC_{50} growth LOAEL	100 to 200 5	10-days	--	F,N	4-8	--	33 ‰	6 µg/L NO_3-N	0.6 µg/L PO_4-P	Found in North America.	(Stromgren 1980)
Brown alga (macrophyte)	*Pelvetia canaliculata*	EC_{50} growth LOAEL	100 to 200 5	10-days	--	F,N	4-8	--	33 ‰	6 µg/L NO_3-N	0.6 µg/L PO_4-P	European specie.	(Stromgren 1980)
Chlorophyte	*Dunaliella tertiolecta*	EC_{50} Growth	1005	72-h	--	S,N	17±1	--	~36 ‰	--	14:10 light cycle	GFF Mediterranean surface water; Used controls, quantified Hg on glass walls	(Fisher et al. 1984)
Coccolithophore	*Emiliania huxleyi*	EC_{50} Growth	0.16	72-h	--	S,N	17±1	--	~36 ‰	--	14:10 light cycle	GFF Mediterranean surface water; Used controls, quantified Hg on glass walls	(Fisher et al. 1984)
Cyanophyte	*Oscillatoria woronichinii*	EC_{50} Growth	0.40	72-h	--	S,N	17±1	--	~36 ‰	--	14:10 light cycle	GFF Mediterranean surface water; Used controls, quantified Hg on glass walls	(Fisher et al. 1984)
Diatom	*Thalassiosira pseudonana*	EC_{50} Growth	0.63	72-h	--	S,N	17±1	--	~36 ‰	--	14:10 light cycle	GFF Mediterranean surface water; Used controls, quantified Hg on glass walls	(Fisher et al. 1984)
Invertebrates - Chronic studies ranked unacceptable:													
American Oyster	*Crassostrea virginica*	EC_{50} growth	12	12-d	Larvae	R,N	25±1	--	24±2 ‰	--	pH 7-8.5	Growth not measured at a range of concentrations.	(Calabrese et al. 1977)
Brine Shrimp	*Artemia franciscana*	EC delayed hatching	200	72-h	Early stages	S,N	28	--	--	--	--	Control hatch=60%, 1µM (200 µg Hg/L) hatch=33% (~50% of C); NOEC=2 µg Hg/L	(Go et al. 1990)
Hard Clam	*Mercenaria mercenaria*	EC growth	15 resulted in growth of 67% compared to control	10-d	Larvae	R,N	25±1	--	24±2 ‰	--	pH 7-8.5	Growth not measured at a range of concentrations.	(Calabrese et al. 1977)
Plants - Chronic studies ranked unacceptable:													
Phytoplankton	*Isochrysis galbana*	reduced population growth	18.6	28-d		S,M ? reps	14.5	--	--	--	--	Hg concentration in solution decrease steadily; not detected after day 12; at conc. <18.6 cultures recovered	(Davies 1974)

*Indicates: static (S), flow-through (F), daily renewal ®, nominal/unmeasured (N), measured conc.(M), reps (replicates). Endpoint abbreviations, chronic or sub-chronic effect concentration (EC), acute or lethal endpoint concentration (LC).

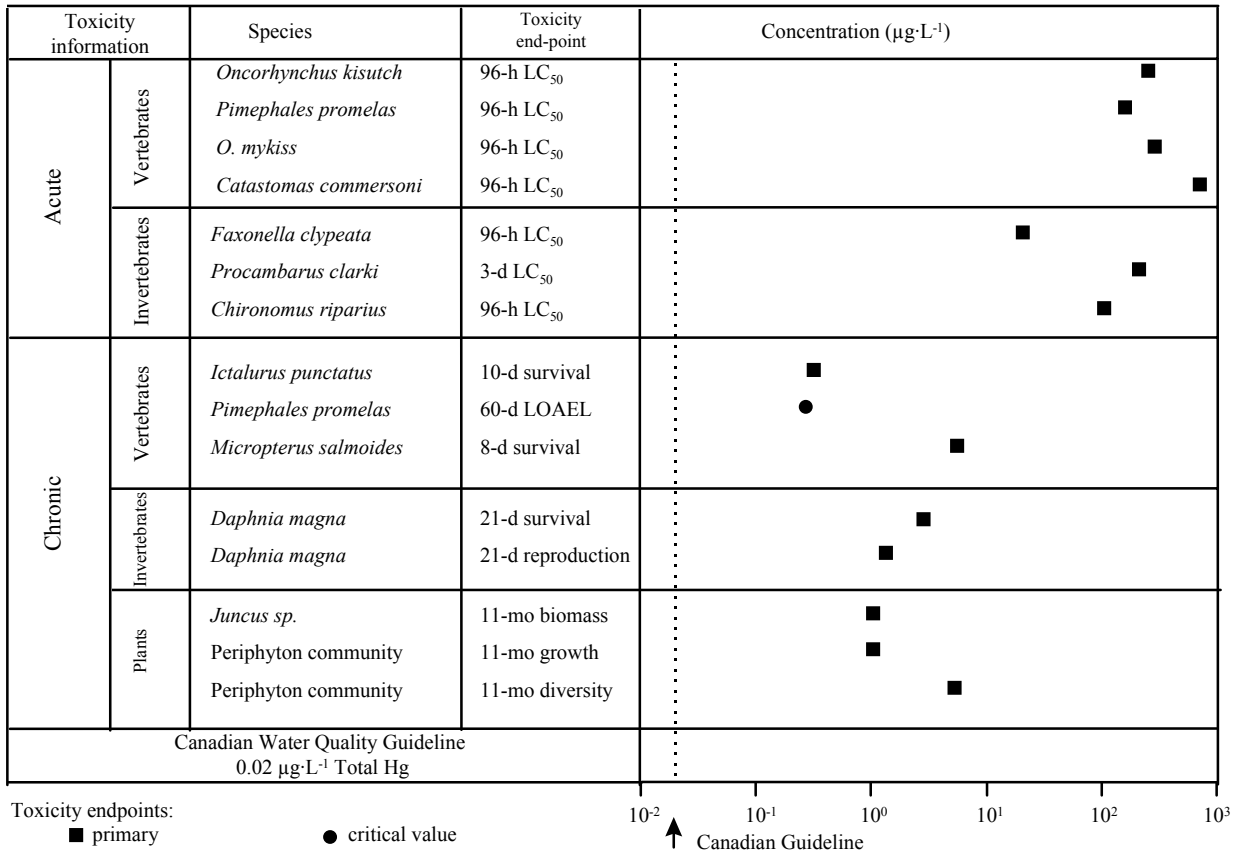

Figure 1. Derivation graph for mercuric chloride ($HgCl_2$) and freshwater organisms

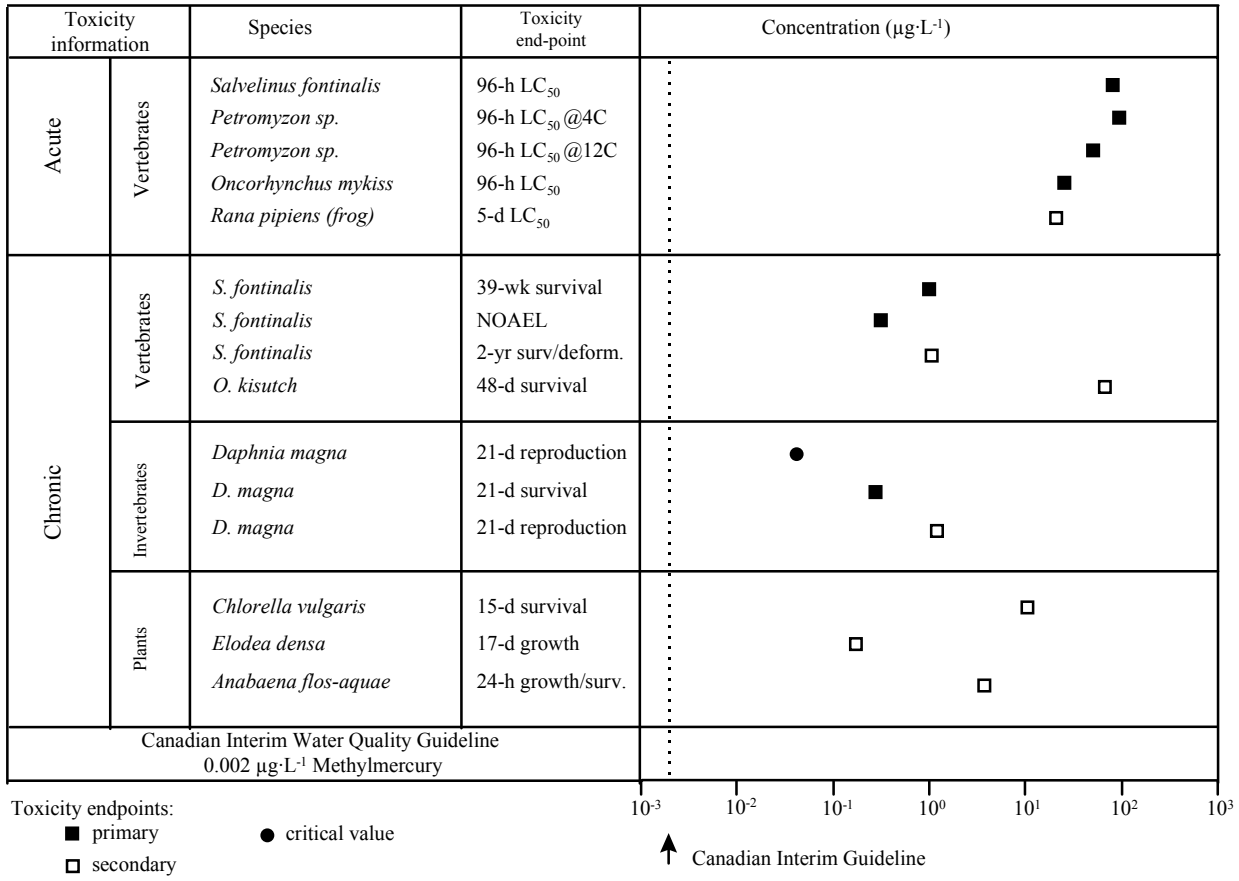

Figure 2. Derivation graph for methylmercury and freshwater organisms

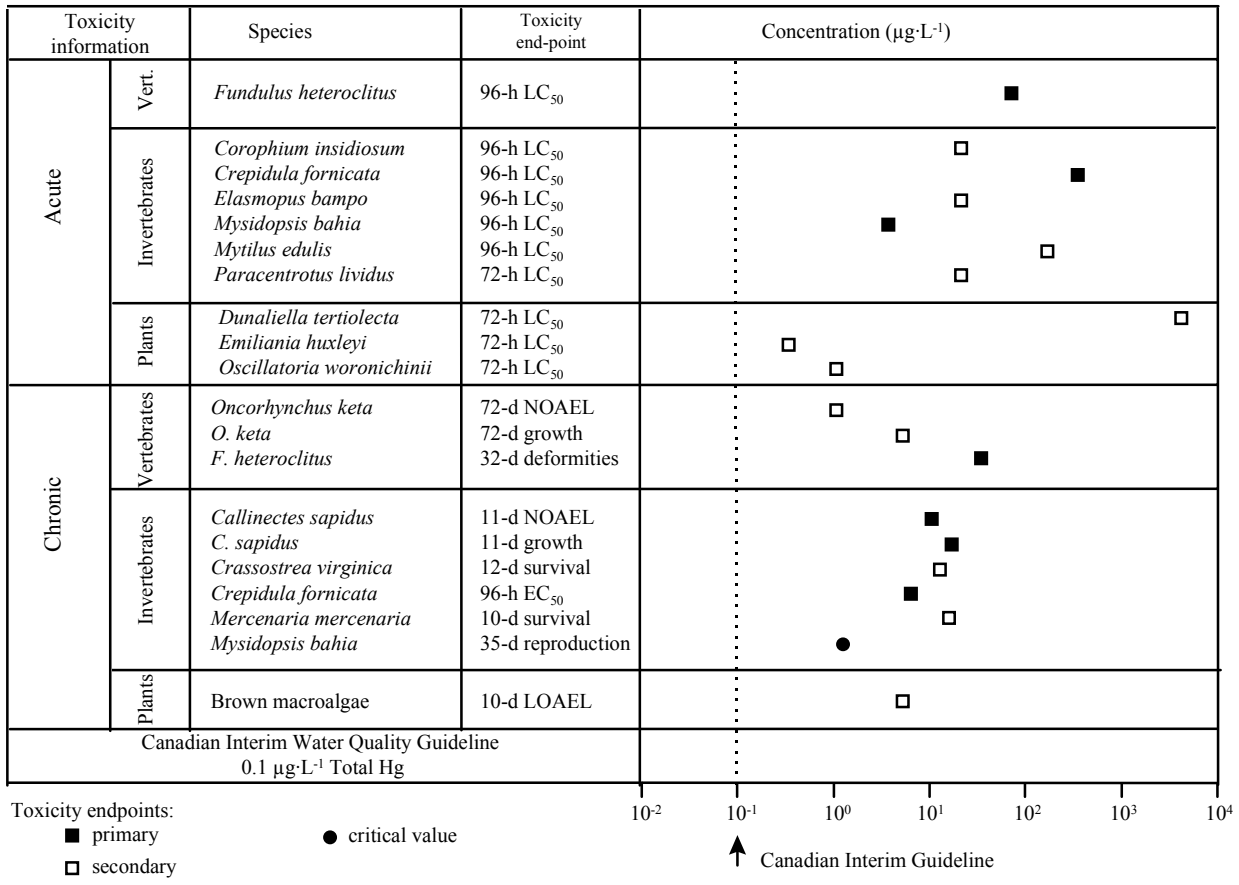

Figure 3. Derivation graph for mercuric chloride ($HgCl_2$) and saltwater organisms

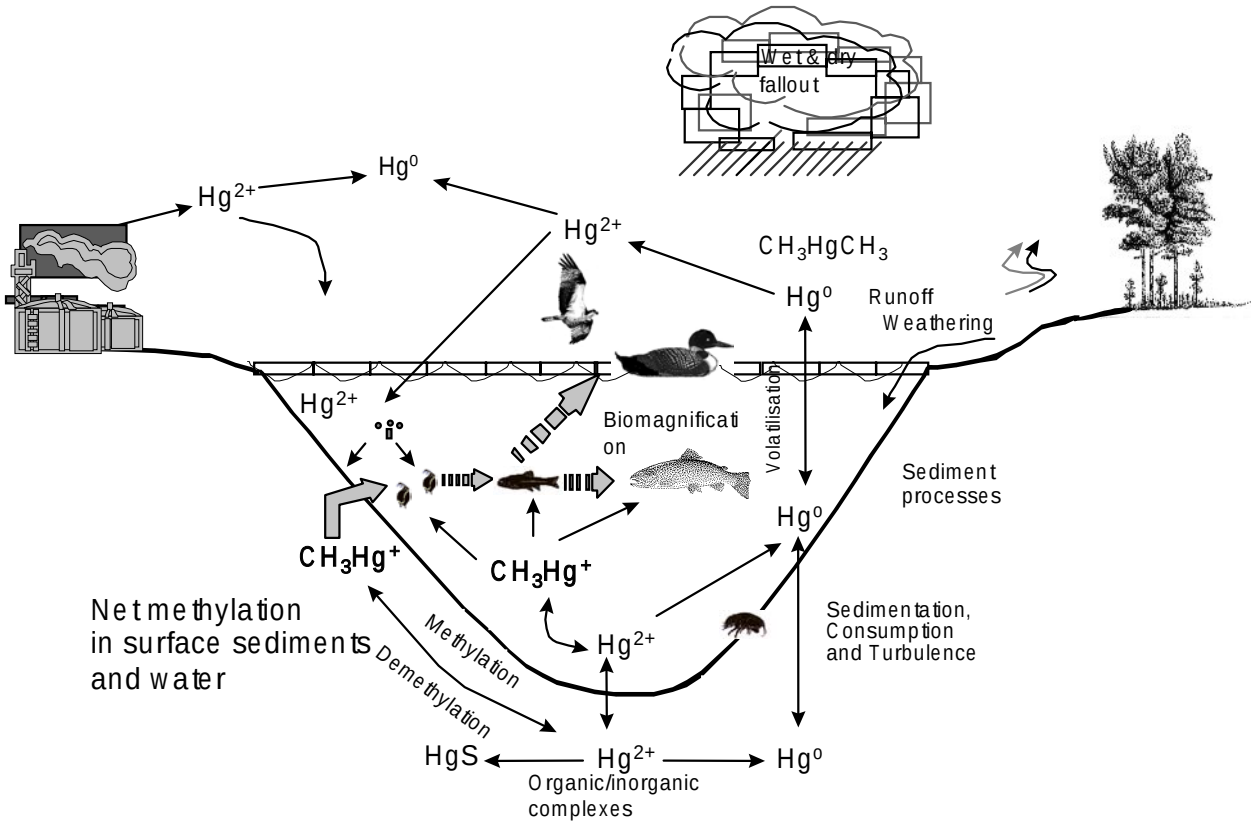

Figure 4. Schematic of the Mercury Cycle

8.0 References

Abbasi, S. A., P. C. Nipaney, and R. Soni. 1988. Studies on environmental management of mercury (II), chromium (VI) and zinc (II) with respect to the impact on some arthropods and protozoans--toxicity of zinc(II). Intern. J. Environ. Studies 32:181-187.

Abdelghani, A. A., Y. V. Pramar, T. K. Mandal, P. B. Tchounwou, and L. Heyer. 1995. Levels and toxicities of selected inorganic and organic contaminants in a swamp environment. J. Environ. Sci. Health B30:717-731.

Abernathy, A. R. and P. M. Cumbie. 1977. Mercury accumulation by largemouth bass (*Micropterus salmoides*) in recently impounded reservoirs. Bull. Environ. Contam. Toxicol. 17:595-602.

Abraham, B. J. 1985. Species profiles: life histories and environmental requirements of coastal fishes and invertebrates (mid-Atlantic). Mummichog and striped killifish. U.S. Fish and Wildlife Service, 82(11.40). Washington, DC.

Aceto, M., A. M. Foglizzo, E. Mentasti, G. Sacchero, and C. Sarzanini. 1995e. Mercury speciation in biological samples. Intern. J. Environ. Anal. Chem. 60:1-13.

ADI Nolan Davis Inc. 1994. Limnology and fisheries studies at Wreck Cove Reservoir. Nova Scotia Power Inc., 24-3117-023-1. Sydney, NS.

Alberta Environmental Centre. 1989. A five-year study of mercury in fish from a newly formed reservoir (Gleniffer Lake, Alberta). Alberta Environmental Centre, AECV89-R4. Vegreville, AB.

Alberta Environmental Protection. 1992. Mercury and its compounds in the environment: their chemistry, toxicology and human health hazards with special reference to Alberta. Standards Research and Development Branch. Environmental Assessment Division Edmonton, AB.

Alberta Environmental Protection. 1998. Draft Alberta water quality guideline for the protection of aquatic life: mercury and methylmercury. Alberta Environmental Protection Edmonton, AB.

Amyot, M., G. Mierle, D. R. S. Lean, and D. J. McQueen. 1994. Sunlight-induced formation of dissolved gaseous mercury in lake waters. Environ. Sci. Technol. 28:2366-2371.

Anderson, M. R., D. A. Scruton, U. P. Williams, and J. F. Payne. 1995. Mercury in fish in the Smallwood Reservoir, Labrador, twenty-one years after impoundment. Water Air Soil Pollut. 80:927-930.

Andersson, I., H. Parkman, and A. Jernelov. 1990. The role of sediments as sink or source for environmental contaminants- a case study of mercury and chlorinated organic compounds. Limnologica 20:347-359.

Angelow, R. V. and D. M. Nicholls. 1991. The effect of mercury exposure on liver mRNA translatability and metallothionein in rainbow trout. Comp. Biochem. Physiol. 100:439-444.

Armstrong, J. E. 1946. Geology and mineral deposits of northern British Columbia west of the Rocky Mountains. Dept. of Mines and Resources.

Atwell, L., K. A. Hobson, and H. E. Welch. 1998. Biomagnification and bioaccumulation of mercury in an arctic marine food web: insights from stable nitrogen isotope analysis. Can. J. Fish. Aquat. Sci. 55:1114-1121.

Back, R. C. and C. J. Watras. 1995. Mercury in zooplankton of northern Wisconsin lakes: taxonomic and site-specific trends. Water Air Soil Pollut. 80:931-938.

Bailey, H. S. 1985. 1983 toxic chemical survey: a survey of nine impoundments in the Saint John River Basin. Environment Canada Moncton,N.B.

Baker, M. D., C. I. Mayfield, W. E. Inniss, and P. T. S. Wong. 1983. Toxicity of pH, heavy metals and bisulfite to a freshwater green alga. Chemosphere.12:35-44.
Baudouin, M. F. and P. Scoppa. 1974. Acute toxicity of various metals to freshwater zooplankton. Bull. Environ. Contam. Toxicol. 12:745-751.
Becker, D. S. and G. N. Bingham. 1995. Distribution of mercury in the aquatic food web of Onondaga Lake, New York. Water Air Soil Pollut. 80:563-571.
Beckvar, N., J. Field, S. Salazar, and D. J. Hoffman. 1996. Contaminants in aquatic habitats at hazardous waste sites: mercury. National Oceanic and Atmospheric Administration,Hazardous Materials Response and Assessment Division, NOAA Technical Memorandum NOS ORCA 100. Seattle, WA.
Best, J. B., M. Morita, J. Ragin, and J. Best. 1981. Acute toxic responses of the freshwater planarian, *Dugesia dorotocephala* to methyl mercury. Bull. Environ. Contam. Toxicol. 18:572-580.
Biesinger, K. E., L. E. Anderson, and J. G. Eaton. 1982. Chronic effects of inorganic and organic mercury on *Daphnia magna*: toxicity, accumulation, and loss. Arch. Environ. Contam. Toxicol. 11:769-774.
Biesinger, K. E. and G. M. Christensen. 1972. Effects of various metals on survival, growth, reproduction, and metabolism of *Daphnia magna*. J. Fish. Res. Board Can. 29:1691-1700.
Birge, W. J., J. A. Black, A. G. Westerman, J. E. Hudson. 1979. The effects of mercury on reproduction of fish and amphibians. In: The biogeochemistry of mercury in the environment, ed. J. O. Nriagu, pp. 629-655. Elsevier/North-Holland Biomedical Press New York.
Birge, W. J., R. D. Hoyt, J. A. Black, M. D. Kercher, and W. A. Robison. 1993. Effects of chemical stresses on behavior of larval and juvenile fishes and amphibians. Amer. Fish. Soc. Symposium. 14:55-65.
Bloom, N. S. 1989a. Determination of picogram levels of methylmercury by aqueous phase ethylation, followed by cryogenic gas chromatography with cold vapour atomic fluorescence detection. Can. J. Fish. Aquat. Sci. 46:1131-1140.
Bloom, N. S. 1992. On the chemical form of mercury in edible fish and marine invertebrate tissue. Can. J. Fish. Aquat. Sci. 49:1010-1017.
Bloom, N. S. and C. J. Watras. 1989. Observations of methylmercury in precipitation. Sci. Total Environ. 87/88:199-207.
Bloom, N. S., C. J. Watras, and J. P. Hurley. 1991. Impact of acidification on the methylmercury cycle of remote seepage lakes. Water Air Soil Pollut. 56:477-491.
Blum, J. E. and R. Bartha. 1980. Effect of salinity on methylation of mercury. Bull. Environ. Contam. Toxicol. 25:404-408.
Bodaly, R. A. and R. J. P. Fudge. 1999. Uptake of mercury by fish in an experimental boreal reservoir. Arch. Environ. Contam. Toxicol. 37:103-109.
Bodaly, R. A. and R. E. Hecky. 1979. Post-impoundment increases in fish mercury levels in the Southern Indian Lake reservoir, Manitoba. Can. Fish. Mar. Serv., MS Rep. 1531. Manitoba.
Bodaly, R. A., R. E. Hecky, and R. J. P. Fudge. 1984. Increases in fish mercury levels in lakes flooded by the Churchill River Diversion, Northern Manitoba. Can. J. Fish. Aquat. Sci. 41:682-691.
Bodaly, R. A., J. W. M. Rudd, R. J. P. Fudge, and C. A. Kelly. 1993. Mercury concentrations in fish related to size of remote Canadian Shield lakes. Can. J. Fish. Aquat. Sci. 50:980-987.
Bonin, J., J.-L. DesGranges, C. A. Bishop, J. Rodrigue, A. Gendron, and J. E. Elliott. 1995. Comparative study of contaminants in the mudpuppy (Amphibia) and the common snapping turtle (Reptilia), St. Lawrence River, Canada. Arch. Environ. Contam. Toxicol. 28:184-194.

Boudou, A., M. Delnomdedieu, D. Georgescauld, F. Ribeyre, and E. Saouter. 1991. Fundamental roles of biological barriers in mercury accumulation and transfer in freshwater ecosystems (analysis at organism, organ, cell and molecular levels). Water Air Soil Pollut. 56:807-822.

Boudou, A. and F. Ribeyre. 1985. Experimental study of trophic contamination of *Salmo gairdneri* by two mercury compounds - HgCl2 and CH3HgCl - analysis at the organism and organ levels. Water Air Soil Pollut. 26:137-148.

Braune, B. M. 1987. Mercury accumulation in relation to size and age of Atlantic Herring (*Clupea harengus harengus*) from the southwestern Bay of Fundy, Canada. Arch. Environ. Contam. Toxicol. 16:311-320.

Brkovic-Popovic, I. 1990. Effect of mercury on the survival of *Daphnia magna*. Water Sci. Tech. 22:241-246.

Brown, D. A. and T. R. Parsons. 1978. Relationship between cytoplasmic distribution of mercury and toxic effects to zooplankton and chum salmon (*Oncorhynchus keta*) exposed to mercury in a controlled ecosystem. J. Fish. Res. Board Can. 35:880-884.

Bruce, W. J. and K. D. Spencer. 1978. Mercury levels in Labrador fish,. 1977-78. Department of Fisheries and Oceans St. John's, Nfld.

Bruce, W. J., K. D. Spencer, and E. Arsenault. 1979. Mercury content data for Labrador fishes,. 1977-78. Fisheries and Marine Service Data Report, 142.

Buhl, K. J. and S. J. Hamilton. 1991. Relative sensitivity of early life stages of Arctic grayling, coho salmon, and rainbow trout to nine inorganics. Ecotoxicol. Environ. Safe. 22:184-197.

Cabana, G. and J. B. Rasmussen. 1994. Modelling food chain structure and contaminant bioaccumulation using stable nitrogen isotopes. Nature 372:255-257.

Calabrese, A., J. R. MacInnes, D. A. Nelson, and J. E. Miller. 1977. Survival and growth of bivalve larvae under heavy metal stress. Mar. Biol. 41:179-184.

Call, D. J., L. T. Brooke, N. Ahmad, and J. E. Richter. 1983. Toxicity and metabolism studies with EPA priority pollutants and related chemicals in freshwater organisms. US Environmental Protection Agency, NTIS, EPA-600/3-83-095. Springfield, VA.

Callister, S. M. and M. R. Winfrey. 1986. Microbial methylation of mercury in upper Wisconsin River sediments. Water, Air, and Soil Pollution 29:453-465.

Canada-Manitoba Mercury Agreement. 1987. Summary Report. Canada-Manitoba agreement on the study and monitoring of mercury in the Churchill River Diversion. Joint Report. Environment Can. and Environ. and Workplace Safety and Health of Manitoba Winnipeg, MB.

Canton, J. H. and D. M. M. Adema. 1978. Reproducibility of short-term and reproduction toxicity experiments with *Daphnia magna* and comparison of the sensitivity of *D. magna* with *D. pulex* and *D. cucullata* in short-term experiments. Hydrobiol. 59:135-140.

CCME. 1991. A protocol for the derivation of water quality guidelines for the protection of aquatic life. In: Canadian environmental quality guidelines pp. 1-9. Canadian Council of Ministers of the Environment Winnipeg.

CCME. 1995. Protocol for the derivation of Canadian sediment quality guidelines for the protection of aquatic life. In: Canadian environmental quality guidelines pp. 1-35. Canadian Ministers of the Environment Winnipeg.

CCME. 1998. Protocol for the derivation of Canadian tissue residue guidelines for the protection of wildlife that consume aquatic biota. In: Canadian Environmental Quality Guidelines pp. 1-18. Canadian Council of Ministers of the Environment Winnipeg.

CCME. 1999. Canadian environmental quality guidelines. Canadian Council of Ministers of the Environment. Winnipeg.

CCME 2000b. Canada-wide Standard for mercury-containing lamps.
CCME 2000a. Canada-wide Standard for mercury in dental amalgams. Canadian Council of Ministers of the Environment Winnipeg, MB.
CCME 2000c. Canada-wide standards for mercury emissions. Canadian Council of Ministers of the Environment Winnipeg, MB.
CCME 2000d. Mercury emissions - Canada-wide Standards. Canadian Council of Ministers of the Environment Winnipeg, MB.
CCREM. 1987. Canadian Water Quality Guidelines. Task Force on Water Quality Guidelines Ottawa, ON.
CEC. 1988. European Community Environmental Legislation:. 1967 -. 1987. Directorate-General for Environment. Consumer Protection and Nuclear Safety, XI/989/87. Brussels, Belgium.
Chen, C.-Y. 1994i. Theoretical evaluation of the inhibitory effects of mercury on algal growth at various orthophosphate levels. Water Res. 28:931-937.
Chen, C.-Y. and K.-C. Lin. 1997. Optimization and performance evaluation of the continuous algal toxicity test. Environ. Toxicol. Chem. 16:1337-1344.
Chen, T.-Y. and D. C. McNaught. 1992. Toxicity of methylmercury to *Daphnia pulex*. Bull. Environ. Contam. Toxicol. 49:606-612.
Choi, S.-C. and R. Bartha. 1994. Environmental factors affecting mercury methylation in estuarine sediments. Bull. Environ. Contam. Toxicol. 53:805-812.
Christensen, G. M. 1975. Biochemical effects of methylmercuric chloride, cadmium chloride, and lead nitrate on embryos and alevins of the brook trout *Salvelinus fontinalis*. Toxicol. Appl. Pharmacol. 32:121-197.
Compeau, G. and R. Bartha. 1983. Effects of sea salt anions on the formation and stability of methylmercury. Bull. Environ. Contam. Toxicol. 31:486-493.
Compeau, G. C. and R. Bartha. 1985. Sulfate-reducing bacteria: principal methylators of mercury in anoxic estuarine sediment. Appl. Environ. Microbiol. 50:498-502.
Coquery, M. and P. M. Welbourn. 1995f. The relationship between metal concentration and organic matter in sediments and metal concentration in the aquatic macrophyte *Eriocaulon septangulare*. Water Res. 29:2094-2102.
Cossa, C. and J.-G. Rondeau. 1985. Seasonal, geographical and site-induced variability in mercury content of *Mytilus edulis* in an estuarine environment: a re-assessment of mercury pollution level in the estuary and Gulf of St.Lawrence. Mar. Biol. 88:43-49.
Cossa, D., M. Coquery, C. Gobeil, J. M. Martin. 1996. Mercury fluxes at the ocean margins. In: Regional and Global Mercury Cycles: Sources, Fluxes and Mass Balances, ed. W. Baeyens, pp. 229-229. Kluwer Publ. Netherlands.
Craig, P. J. and P. D. Bartlett. 1978. The role of hydrogen sulphide in environmental transport of mercury. Nature 275:635-637.
Curtis, M. W., T. L. Copeland, and C. H. Ward. 1979. Acute toxicities of 12 industrial chemicals to freshwater and saltwater organisms. Water Res. 13:137-137.
Cuvin-Aralar, M. L. A. and R. W. Furness. 1991. Mercury and selenium interaction: a review. Ecotoxicol. Environ. Safe. 21:348-364.
Czuba, M. and D. C. Mortimer. 1980. Stability of methyl-mercury and inorganic mercury in aquatic plants (*Elodea densa*). Can. J. Bot. 58:316-320.
D'Itri, F. M. 1991. Mercury Contamination - what we have learned since Minamata. Environ. Monitor. Assess. 19:165-182.

Davies, A. G. 1974. The growth kinetics of *Isochrysis galbana* in cultures containing sublethal concentrations of mercuric chloride. Journal of the Marine Biological Association of the United Kindom 54:157-169.
Davis, R. B., D. S. Anderson, and F. Berge. 1985. Palaeolimnological evidence that lake acidification is accompanied by loss of organic matter. Nature 316:436-438.
Delcourt, A. and J. C. Mestre. 1978. The effects of phenylmercuric acetate on the growth of Chlamydomonas variabilis Dang. Bull. Environ. Contam. Toxicol. 20:145-148.
Devlin, E. W. and N. K. Mottet. 1992. Embryotoxic action of methyl mercury on Coho salmon embryos. Bull. Environ. Contam. Toxicol. 49:449-454.
Dial, N. A. 1976. Methylmercury: teratogenic and lethal effects in frog embryos. Teratology 13:327-334.
Douglas, J. 1991. Mercury in the environment. EPRI Journal December:4-11.
Doyon, J.-F. and R. Schetagne. 1999. Réseau de suivi environnemental du complexe LaGrande, Phase I (1997-1998). Évolution des teneurs en mercure des poissons et études complémentaires. Rapport conjoint Groupe-conseil Génivar inc. et Hydro-Québec, Direction expertise et support technique de production.
Doyon, J.-F., R. Schetagne, and R. Verdon. 1998. Different mercury bioaccumulation rates between sympatric populations of dwarf and normal lake whitefish (*Coregonus clupeaformis*) in the La Grande complex watershed, James Bay, Quebec. Biogeochemistry 40:203-216.
Driscoll, C. T., C. Yan, C. L. Schofield, R. K. Munson, and J. G. Holsapple. 1994. The mercury cycle and fish in the Adirondack lakes. Environ. Sci. Technol. 28:136A-143A.
Drummond, R. A., G. F. Olson, and A. R. Batterman. 1974. Cough response and uptake of mercury by brook trout, Salvelinus fontinalis, exposed to mercuric compounds at difference hydrogen- ion concentrations. Trans. Amer. Fish. Soc. 103:244-249.
Duncan, D. A. and J. F. Klaverkamp. 1983. Tolerance and resistance to Cd in white suckers (Catostomus commersoni) previously exposed to Cd, Hg, Zn or Se. Can. J. Fish. Aquat. Sci. 40:128-138.
Eganhouse, R. P. and D. R. Young. 1978. Total and organic mercury in benthic organisms near a major submarine wastewater outfall system. Bull. Environ. Contam. Toxicol. 19:758-766.
Eisler, R. 1987. Mercury hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Dept. of Interior, Fish & Wildlife Service Washington DC.
Eisler, R. and R. J. Hennekey. 1977. Acute toxicities of Cd(II), Cr(VI), Hg(II), Ni(II), and Zn(II) to estuarine macrofauna. Arch. Environ. Contam. Toxicol. 6:315.
Electric Power Research Institute. 1994. EPRI Electric utilities trace substances sythesis report. EPRI unknown.
Engstrom, D. R., E. B. Swain, T. A. Henning, M. E. Brigham, P. L. Brezonik. 1994. Atmospheric mercury deposition to lakes and watersheds. A quantitative reconstruction from multiple sediment cores. In: Environmental Chemistry of Lakes and Reservoirs, ed. L. A. Baker, pp. 33-66.
Environment Canada. 1996. Canadian soil quality guidelines for inorganic mercury: environmental supporting document- final draft. Ottawa, ON.
Environment Canada. 1997. Canadian sediment quality guidelines for mercury: supporting document. Environmental Conservation Service, Ecosystem Science Directorate, Science Policy and Environmental Quality Branch, Guidelines and Standards Division Ottawa, ON.
Environment Canada. 1999. Health of the Fraser River Aquatic Ecosystem Vol. II. Environment Canada, DOE FRAP. 1998-11. Vancouver.

Environment Canada 2002. Canadian tissue residue guideline for the protection of wildlife consumers of aquatic biota: methylmercury. Ecosystem Health: Science-based Solutions Report No. 1-4. National Guidelines and Standards Office, Water Policy and Coordination Directorate, Environment Canada. Ottawa. 188 pp.
Environment Canada and U.S.EPA. 1997. Canada -- United States Strategy For The Virtual Elimination of Persistent Toxic Substances In The Great Lakes. The Great Lakes Binational Toxics Strategy. www.epa.gov/glnpo/p2/bns.html From website.
EPMAP. 1994. Mercury atmospheric processes: A synthesis report. Tampa FL.
Faust, B. C. 1992. The octanol/water distribution coefficients of methylmercuric species: the role of aqueous-phase chemical speciation. Environ. Toxicol. Chem. 11:1373-1376.
Fimreite, N. and L. M. Reynolds. 1973. Mercury contamination of fish in northwestern Ontario. J. Wildl. Manage. 37:62-68.
Fisher, N. S., M. Bohe, and J.-L. Teyssie. 1984. Accumulation and toxicity of Cd, Zn, Ag, and Hg in four marine phytoplankters. Mar. Ecol. Prog. Ser. 18:201-213.
Fitzgerald, W. F. 1986. The role of air-sea exchange in geochemical cycling. Reidel. Dordrecht.
Fitzgerald, W. F. and C. J. Watras. 1989. Mercury in the surficial waters of rural Wisconsin lakes. Sci. Total Environ. 87/88:223-232.
Foster, T. J. 1987. The genetics and biochemistry of mercury resistance. Crit. Rev. Microbiol. 15:117-140.
Fowler, S. W., M. Heyraud, and J. LaRosa. 1978. Factors affecting methyl and inorganic mercury dynamics in mussels and shrimp. Mar. Biol. 46:267-276.
Freda, J. 1991. The effects of aluminum and other metals on amphibians. Environ. Pollut. 71:305-328.
Furukawa, K., T. Suzuki, and K. Tonomura. 1969. Decomposition of organic mercurial compounds by mercury-resistant bacteria. Agric. Biol. Chem. 33:128-130.
Furutani, A. and J. W. M. Rudd. 1980. Measurement of mercury methylation in lake water and sediment samples. Appl. Environ. Microbiol. 40:770-776.
Gaudet, C., S. Lingard, P. Cureton, K. Keenleyside, S. Smith, and G. Raju. 1995. Canadian environmental quality guidelines for mercury. Water Air Soil Pollut. 80:1149-1159.
Gentile, J. H., S. M. Gentile, G. Hoffman, J. F. Heltsche, and N. Hailstone, Jr. 1983. The effects of a chronic mercury exposure on survival, reproduction and population dynamics of *Mysidopsis bahia*. Environ. Toxicol. Chem. 2:61-68.
Gill, G. A. and K. W. Bruland. 1990. Mercury speciation in surface freshwater systems in California and other areas. Environ. Sci. Technol. 24:1392-1400.
Gill, G. A. and W. F. Fitzgerald. 1985. Mercury sampling of open ocean waters at the picomolar level. Deep-Sea Res. 32:287-297.
Gill, G. A. and W. F. Fitzgerald. 1987. Picomolar mercury measurements in seawater and other materials using stannous chloride reduction and two-stage gold amalgamation with gas phase detection. Mar. Chem. 20:227-243.
Gilmour, C. C. and D. G. Capone. 1987. Relationship between Hg methylation and the sulfur cycle in estuarine sediments. EOS Trans. Am. Geophys. Union 68:1718-1725.
Gilmour, C. C. and E. A. Henry. 1991. Mercury methylation in aquatic systems affected by acid deposition. Environ. Pollut. 71:131-169.
Gilmour, C. C., E. A. Henry, and R. Mitchell. 1992. Sulfate stimulation of mercury methylation in freshwater sediments. Environ. Sci. Technol. 26:2281-2286.

Gilmour, C. C. and G. S. Riedel. 1995. Measurement of Hg methylation in sediments using high specific-activity Hg and ambient incubation. Water Air Soil Pollut. 80:747-756.
Glass, G. E. and J. A. Sorensen. 1999. Six-year trend (1990-1995) of wet mercury deposition in the upper midwest, U.S.A. Environ. Sci. Technol. 33:3303-3312.
Go, E. C., A. S. Pandey, and T. H. MacRae. 1990. Effect of inorganic mercury on the emergence and hatching of the brine shrimp *Artemia franciscana*. Mar. Biol. 107:93-102.
Grey, B. J., S. M. Harbicht, and G. R. Stephens. 1995. Mercury in fish from rivers and lakes in southwestern Northwest Territories. Department of Indian Affairs and Northern Development Ottawa, Canada.
Grieb, T. M., C. T. Driscoll, S. P. Gloss, C. L. Schofield, G. L. Bowie, and D. B. Porcella. 1990. Factors affecting mercury accumulation in fish in the Upper Michigan Peninsula. Environ. Toxicol. Chem. 9:919-930.
Haines, M. L., K. Brydges, M. J. MacDonald, S. L. Smith, and D. D. MacDonald. 1994. A Review of Environmental Quality Criteria and Guidelines for Priority Substances in the Fraser River Basin: Supporting Documentation. Environment Canada North Vancouver, BC.
Halbach, S. 1985. The octanol/water distribution of mercury compounds. Arch. Toxicol. 57:139-141.
Hall, B. D., R. A. Bodaly, R. J. P. Fudge, J. W. M. Rudd, and D. M. Rosenberg. 1997. Food as the dominant pathway of methylmercury uptake by fish. Water Air Soil Pollut. 100:13-24.
Hall, B. D., D. M. Rosenberg, and A. P. Wiens. 1998d. Methyl mercury in aquatic insects from an experimental reservoir. Can. J. Fish. Aquat. Sci. 55:2036-2047.
Hammerschmidt, C. R., J. G. Wiener, B. E. Frazier, and R. G. Rada. 1999. Methylmercury content of eggs in yellow perch related to maternal exposure in four Wisconsin lakes. Environ. Sci. Technol. 33:999-1003.
Harris, R. C. and W. J. Snodgrass. 1993. Bioenergetic simulations of mercury uptake and retention in walleye (*Stizostedion vitreum*) and yellow perch (*Perca flavescens*). Water Poll.Res.J.Canada 28:217-236.
Harris, R. C., D. B. White, and R. B. Macfarlane. 1970. Mercury compounds reduce photosynthesis by plankton. Science 170, 736-737. 1970.
Hecky, R. E., D. J. Ramsey, R. A. Bodaly, N. E. Strange. 1991. Increased methylmercury contamination in fish in newly formed freshwater reservoirs. In: Advances in Mercury Toxicology, eds. T. F. W. Clarkson, T. Suzuki, and A. Imura, pp. 33-52. Plenum Press New York.
Heisinger, J. F., C. D. Hansen, and J. H. Kim. 1979. Effect of selenium dioxide on the accumulation and acute toxicity of mercuric chloride in goldfish. Arch. Environ. Contam. Toxicol. 8:279-283.
Heit, M. and M. Fingerman. 1977. The influence of size, sex and temperature on the toxicity of mercury to two species of crayfishes. Bull. Environ. Contam. Toxicol. 18:572-580.
Hendzel, M. R. and D. M. Jamieson. 1976. Determination of mercury in fish. Anal. Chem. 48:926-928.
Hildebrand, S. G., R. H. Strand, and J. W. Huckabee. 1980. Mercury accumulation in fish and invertebrates of the North Fork Holston River, Virginia and Tennessee. J. Environ. Qual. 9:393-400.
Horvat, M. 1991. Determination of methylmercury in biological certified reference materials. Water Air Soil Pollut. 56:95-102.

Huckabee, J. W., J. W. Elwood, S. G. Hildebrand. 1979. Accumulation of mercury in freshwater biota. In: The biogeochemistry of mercury in the environment, ed. J. O. Nriagu, pp. 277-302. Elsevier/North-Holland Biomedical Press New York.

Hudson, R. J. M., S. A. Gherini, C. J. Watras, D. B. Porcella. 1994. A mechanistic model of the biogeochemical cycle of mercury in lakes. In: Mercury pollution: Integration and synthesis, eds. C. J. Watras and J. W. Huckabee, pp. 473-526. Lewis Publishers Ann Arbor, MI.

Hurley, J. P., J. M. Benoit, C. L. Babiarz, M. M. Shafer, A. W. Andren, J. R. Sullivan, R. Hammond, and D. A. Webb. 1995. Influences of watershed characteristics on mercury levels in Wisconsin rivers. Environ. Sci. Technol. 29:1867-1875.

Hurley, J. P., D. P. Krabbenhoft, C. L. Babiarz, A. W. Andren. 1994. Cycling of mercury across the sediment-water interface in seepage lakes. In: Environmental Chemistry of Lakes and Reservoirs, ed. L. Baker, pp. 425-449. American Chemical Society Washington DC.

Hurley, J. P., C. J. Watras, and N. S. Bloom. 1989. Biogeochemistry of mercury in aquatic ecosystems. Aqueous speciation of mercury: seston component. Semiannual Report to EPRI Madison WI.

Hurley, J. P., C. J. Watras, and N. S. Bloom. 1991. Mercury cycling in a northern Wisconsin seepage lake: the role of particulate matter in vertical transport. Water Air Soil Pollut. 56:543-551.

Hydro-Quebec and Groupe-Conseil Genivar Inc. 1997. Summary Report - Evolution of Fish Mercury Levels at the La Grande complex, Quebec (1978-1994). Hydro-Quebec Quebec.

International Joint Commission. 1990a. Production, usage and atmosphere emissions of 14 priority toxic chemicals. Appendix 2 to the Proceedings of the Workshop on Great Lakes Atmospheric Deposition.

Inza, B., F. Ribeyre, and A. Boudou. 1998. Dynamics of cadmium and mercury compounds (inorganic mercury or methylmercury): uptake and depuration in *Corbicula fluminea* - effects of temperature and pH. Aquat. Toxicol. 43:273-285.

Jackson, T. A. 1987. Methylation, demethylation, and bio-accumulation of mercury in lakes and reservoirs of Northern Manitoba, with particular reference to effects of environmental changes caused by the Churchill-Nelson River diversion. Can. Data Rep. Fish. Aquat.Sci. 628.

Jackson, T. A. 1988. The mercury problem in recently formed reservoirs of northern Manitoba (Canada): effects of impoundment and other factors on the production of methyl mercury by microorganisms in sediments. Can. J. Fish. Aquat. Sci. 45:97-121.

Jackson, T. A. 1989. The influence of clay minerals, oxides, and humic matter on the methylation and demethylation of mercury by micro-organisms in freshwater sediments. Appl.Organometal.Chem. 3:1-30.

Jackson, T. A. 1991. Biological and environmental control of mercury accumulation by fish in lakes and reservoirs of northern Manitoba, Canada. Can. J. Fish. Aquat. Sci. 48:2249-2470.

Jackson, T. A. 1993. Effects of environmental factors and primary production on the distribution and methylation of mercury in a chain of highly eutrophic riverine lakes. Water Poll. Res. J. Can. 28:177-216.

Jackson, T. A. 1997. Long-range atmospheric transport of mercury to ecosystems, and the importance of anthropogenic emissions - a critical review and evaluation of the published evidence. Environ. Rev. 5:99-120.

Jackson, T. A. 1998. Mercury in aquatic systems. In: Metal Metabolism in Aquatic Environments, eds. W. J. Langston and M. J. Bebianno, pp. 77-158. Chapman & Hall London.

Jana, S. 1988. Accumulation of Hg and Cr by three aquatic species and subsequent changes in several physiological and biochemical plant parameters. Water Air Soil Pollut. 38:105-109.
Jensen, S. and A. Jernelov. 1969. Biological methylation of mercury in aquatic organisms. Nature 223:753-754.
Jernelov, A. and C. Ramel. 1994. Mercury in the environment. Ambio 23:166.
Johnston, T. A., R. A. Bodaly, and J. A. Mathias. 1991. Predicting fish mercury levels from physical characteristics of boreal reservoirs. Can. J. Fish. Aquat. Sci. 48:1468-1475.
Jonas, R. B., C. C. Gilmour, D. L. Stoner, M. M. Weir, and J. H. Tuttle. 1984. Comparison of methods to measure acute metal and organometal toxicity to natural aquatic microbial communities. Appl. Environ. Microbiol. 47:1005-1011.
Kania, H. J., R. L. Knight, and R. J. Beyers. 1976. Fate and biological effects of mercury introduced into artificial streams. Environmental Protection Agency Springfield VA.
Keller, A. E. and S. G. Zam. 1991. The acute toxicity of selected metals to the freshwater mussel, *Anodonta imbecelis*. Environ. Toxicol. Chem. 10:539-546.
Kelly, C. A., J. W. M. Rudd, R. A. Bodaly, N. P. Roulet, V. L. St.Louis, A. Heyes, T. R. Moore, S. L. Schiff, R. Aravena, K. J. Scott, B. Dyck, R. Harris, B. Warner, and G. Edwards. 1997. Increases in fluxes of greenhouse gases and methyl mercury following flooding of an experimental reservoir. Environ. Sci. Technol. 31:1334-1344.
Kelly, C. A., J. W. M. Rudd, V. L. St.Louis, and A. Heyes. 1995. Is total mercury concentration a good predictor of methyl mercury concentration in aquatic systems. Water Air Soil Pollut. 80:715-724.
Kent, R. A., P. Y. Caux, R. Allan, R. Post, and J. Parks. 1998. A Canada-wide GIS analysis of methylmercury in fish - exploring relative risks to wildlife and human health. Ottawa, ON.
Khan, A. T. and J. S. Weis. 1987. Effects of methylmercury on sperm and egg viability of two populations of killifish (*Fundulus heteroclitus*). Arch. Environ. Contam. Toxicol. 16:499-505.
Khangarot, B. S. and P. K. Ray. 1987a. Relation between heavy metal acute toxicity values in Daphnia magna and fish. Bull. Environ. Contam. Toxicol. 38:722-726.
Khangarot, B. S. and P. K. Ray. 1987b. Sensitivity of toad tadpoles, Bufo melanostictus (Schneider), to heavy metals. Bull. Environ. Contam. Toxicol. 38:523-527.
Khangarot, B. S. and P. K. Ray. 1988. Sensitivity of freshwater pulmonate snails, Lymnaea lutiola L. to heavy metals. Bull. Environ. Contam. Toxicol. 41:208-213.
Khangarot, B. S. and P. K. Ray. 1989a. Sensitivity of midge larvae of *Chironomus tentans* Fabricius (Diptera Chironomidae) to heavy metals. Bull. Environ. Contam. Toxicol. 42:325-330.
Khangarot, B. S. and P. K. Ray. 1989b. Investigation of correlation between physicochemical properties of metals and their toxicity to the water flea *Daphnia magna* Straus. Ecotoxicol. Environ. Safety 18:109-120.
King, D. G. and I. M. Davies. 1987. Laboratory and field studies of the accumulation of inorganic mercury by the mussel *Mytilus edulis* (L.). Mar. Pollut. Bull. 18:40-45.
King, J. K., F. M. Saunders, R. F. Lee, and R. A. Jahnke. 1999. Coupling mercury methylation rates to sulfate reduction rates in marine sediments. Environ. Toxicol. Chem. 18:1362-1369.
Kirkwood, A. E., P. Chow-Fraser, and G. Mierle. 1999b. Seasonal mercury levels in phytoplankton and their relationship with algal biomass in two dystrophic shield lakes. Environ. Toxicol. Chem. 18:523-32.
Kirubagaran, R. and K. P. Joy. 1988. Toxic effects of mercuric chloride, methylmercuric chloride, and Emisan 6 (an organic mercurial fungicide) on ovarian recrudescence in the catfish *Clarias batrachus* (L.). Bull. Environ. Contam. Toxicol. 41:902-909.

Knowles, S. C. and R. G. Zingmark. 1978. Mercury and temperature interactions on the growth rates of three species of freshwater phytoplankton. J. Phycol. 14:104-109.
Krabbenhoft, D. P., J. M. Benoit, C. L. Babiarz, J. P. Hurley, and A. W. Andren. 1995. Mercury cycling in the Allequash Creek watershed, northern Wisconsin. Water Air Soil Pollut. 80:425-433.
Krenkel, P. A. 1974. Mercury: environmental considerations, Part II. Crit.Rev.Environ.Control. 4:251-339.
Kristensen, P. 1982. Time-dependent variation of mercury in a stream sediment and the effect upon mercury content in *Gammarus pulex* (L.). Water Res. 16:759-764.
Kudo, A., H. Nagase, and Y. Ose. 1982. Proportion of methylmercury to the total amount of mercury in river waters in Canada and Japan. Water Res. 16:1015.
Laliberte, D. 1996. Teneurs en mercure et en composes organochlores dans les poissons captures au lac Aylmer en. 1995. Ministere de l'Environnement et de la Faune du Quebec Quebec.
Langlois, C., R. Langis, and M. Perusse. 1995. Mercury Contamination in Northern Quebec Environment and Wildlife. Water Air Soil Pollut. 80:1021-1024.
Lee, Y.-H. 1987. Determination of methyl- and ethylmercury in natural waters at sub-nanogram per liter using SCF-adsorbent preconcentration procedure. Intern. J. Environ. Anal. Chem. 29:263-276.
Lee, Y.-H. and H. Hultberg. 1990. Methylmercury in some Swedish surface waters. Environ. Toxicol. Chem. 9:833-841.
Lee, Y.-H., H. Hultberg, and I. Andersson. 1985. Catalytic effect of various metal ions on the methylation of mercury in the presence of humic substances. Water Air Soil Pollut. 25:391-400.
Lee, Y.-H. and A. Iverfeldt. 1991. Measurement of methylmercury and mercury in run-off, lake and rain waters. Water Air Soil Pollut. 56:309-321.
Leermakers, M., M. Elskens, S. Panutrakul, F. Monteny, W. Baeyens. 1993. Geochemistry of mercury in an intertidal flat of the Scheldt Estuary. In: Marine and Estuarine Gradients pp. 267-277. ECSA Symposium held in Belgium.
Lindqvist, O., K. Johansson, M. Aastrup, A. Andersson, L. Bringmark, G. Hovsenius, L. Håkanson, Å. Iverfeldt, M. Meili, and B. Timm. 1991. Emissions of mercury to the atmosphere. Water, Air and Soil Pollution 55[1/2], 23-32.
Lock, R. A. and A. P. Van Overbeeke. 1981. Effects of mercuric chloride and methylmercuric chloride on mucus secretion in rainbow trout. Comp. Biochem. Physiol. 69C:67-73.
Lodenius, M., A. Seppanen, and M. Herranen. 1983. Accumulation of mercury in fish and man from reservoirs in northern Finland. Water Air Soil Pollut. 19:237-246.
Lorz, H. W., R. H. Williams, and C. A. Fustish. 1978. Effects of several metals on smolting of coho salmon. Environmental Protection Agency, EPA-600/3-78-090, NTIS Technical report PB 290168. Springfield, VA.
Lucotte, M., S. Montgomery, B. Caron, M. Kainz. 1999. Mercury in natural lakes and unperturbed terrestrial ecosystems of northern Québec. In: Mercury in the biogeochemical cycle: mercury in natural environments and hydroelectric reservoirs of northern Quebéc, eds. M. Lucotte, R. Schetagne, N. Thérien, C. Langlois, and A. Tremblay, pp. 55-87. Springer-Verlag Berlin.
Ludwicki, J. K. 1990. *In vitro* methylation and demethylation of mercury compounds by the intestinal contents. Bull. Environ. Contam. Toxicol. 44:357-362.
Luoma, S. N. 1977. The dynamics of biologically available mercury in a small estuary. Est. Coast. Mar. Sci. 5:643-652.

Lussier, S. M., J. H. Gentile, and J. Walker. 1985. Acute and chronic effects of heavy metals and cyanide on *Mysidopsis bahia* (Crustacea: Mysidacea). Aquat. Toxicol. 7:25-35.
MAC 2000. Environmental Progress Report. 1999. The Mining Association of Canada.
MacCrimmon, H. R., C. D. Wren, and B. L. Gots. 1983. Mercury uptake by lake trout, *Salvelinus namaycush*, relative to age, growth, and diet in Tadenac Lake with comparative data from other PreCambrian Shield lakes. Can. J. Fish. Aquat. Sci. 40:114-120.
MacDonald, D. D., T. Berger, K. Wood, J. Brown, T. Johnsen, M. L. Haines, K. Brydges, M. J. MacDonald, S. L. Smith, and P. Shaw. A Compendium of Environmental Quality Benchmarchs for Priority Substances in the Georgia Basin. [Volumes I-IV]. 1999. Environment Canada. North Vancouver, BC.
Ref Type: Serial (Book,Monograph)
MacLeod, J. C. and E. Pessah. 1973. Temperature effects on mercury accumulation, toxicity and metabolic rate in rainbow trout (*Salmo gairdneri*). J. Fish. Res. Board Can. 30:485-492.
Major, M. A. and D. H. Rosenblatt. 1991. The octanol/water partition coefficient of methylmercuric chloride and methylmercuric hydroxide in pure water and salt solutions. Environ. Toxicol. Chem. 10:5-8.
Mallatt, J., M. G. Barron, and C. McDonough. 1986c. Acute toxicity of methyl mercury to the larval lamprey, *Petromyzon marinus*. Bull. Environ. Contam. Toxicol. 37:281-288.
Malley, D. F., A. R. Stewart, and B. D. Hall. 1996c. Uptake of methylmercury by the floater mussel, *Pyganodon grandis* (Bivalvia, Unionidae), caged in a flooded wetland. Environ. Toxicol. Chem. 15:928-936.
Mason, R. P., W. F. Fitzgerald, and F. M. M. Morel. 1994. The biogeochemical cycling of elemental mercury: anthropogenic influences. Geochim. Cosmochim. Acta 58:3191-3198.
Mason, R. P., J. R. Reinfelder, and F. M. M. Morel. 1995. Bioaccumulation of mercury and methylmercury. Water Air Soil Pollut. 80:915-921.
Mason, R. P., J. R. Reinfelder, and F. M. M. Morel. 1996. Uptake, toxicity, and trophic transfer of mercury in a coastal diatom. Environ. Sci. Technol. 30:1835-1845.
Mason, R. P. and K. A. Sullivan. 1997. Mercury in Lake Michigan. Environ. Sci. Technol. 31:942-947.
Matilainen, T. and M. Verta. 1995. Mercury methylation and demethylation in aerobic surface waters. Can. J. Fish. Aquat. Sci. 52:1597-1608.
Maury-Brachet, R., F. Ribeyre, and A. Boudou. 1990. Actions and interactions of temperature and photoperiod on mercury accumulation by *Elodea densa* from sediment source. Ecotoxicol. Environ. Safe. 20:141-155.
Maury, R., A. Boudou, F. Ribeyre, and P. Engrand. 1988. Experimental study of mercury transfer between artificially contaminated sediment (CH_3HgCl) and macrophytes (*Elodea densa*). Aquat. Toxicol. 12:213-228.
McKenney, C. L., Jr., J. D. Costlow, Jr. 1981. The effects of salinity and mercury on developing megalopae and early crab stages of the blue crab *Callinectes sapidus* Rathbun. In: Biological Monitoring of Marine Pollutants, eds. F. J. Vernberg, A. Calabrese, F. P. Thurberg, and W. B. Vernberg, pp. 241-262. Academic Publishers New York.
McKim, J. M., G. F. Olson, G. W. Holcombe, and E. P. Hunt. 1976. Long-term effects of methylmercuric chloride on three generations of brook trout (*Salvelinus fontinalis*): toxicity, accumulation, distribution, and elimination. J. Fish. Res. Board Can. 33:2726-2739.
McMurtry, M. J., D. L. Wales, W. A. Scheider, G. L. Beggs, and P. E. Dimond. 1989. Relationship of mercury concentrations in lake trout (*Salvelinus namaycush*) and

smallmouth bass (*Micropterus dolomieui*) to the physical and chemical characteristics of Ontario lakes. Can. J. Fish. Aquat. Sci. 46:426-434.
MEF. Critères de qualité de l'eau de surface au Québec. Web site . 1998. Direction des écosytèmes aquatiques, Ministère de l'Environnement et de la Faune.
Ref Type: Electronic Citation
Mierle, G. 1990. Aqueous inputs of mercury to Precambrian Shield lakes in Ontario. Environ. Toxicol. Chem. 9:843-851.
Mierle, G. 1995. Sources of mercury to lakes on the Precambrian Shield of Ontario. Proceedings of the. 1995 Canadian Mercury Network Workshop, Sept. 29-30.
Mierle, G. and R. Ingram. 1991. The role of humic substances in the mobilization of mercury from watersheds. Water Air Soil Pollut. 56:349-357.
Miller, R. W. 1975. The role of humic acids in the uptake and release of mercury by freshwater sediments. Verh.Internat.Verein.Limnol. 19:2082-2086.
Minerals Yearbook. 1989. Mercury. United States Bureau of Mines, 1. Washington, DC.
Miskimmin, B. M. 1991. Effect of natural levels of dissolved organic carbon (DOC) on methyl mercury formation and sediment-water partitioning. Bull. Environ. Contam. Toxicol. 47:743-750.
Miskimmin, B. M., J. W. M. Rudd, and C. A. Kelly. 1992. Influence of dissolved organic carbon, pH, and microbial respiration rates on mercury methylation and demethylation in lake water. Can. J. Fish. Aquat. Sci. 49:17-22.
Mo, S. C., D. S. Choi, and J. W. Robinson. 1989. Uptake of mercury from aqueous solution by duckweed: the effects of pH, copper and humic acid. J. Environ. Sci. Health 24A:135-146.
MOEE. 1979. Rational for the establishment of Ontario's provincial water quality objectives. Ministry of Environment and Energy, Government of Ontario, September. 1979.
MOEE. 1999. Appendix A: Provincial water aquality objectives. In: Water management policies, guidelines, provincial water quality objectives of the Ministry of Environment and Energy p. -31. Ministry of Environment and Energy, Government of Ontario Toronto.
Mohlenberg, F. and H. U. Riisgard. 1988. Partitioning of inorganic and organic mercury in cockles *Cardium edule* (L.) and *C. glaucum* (Bruguiere) from a chronically polluted area: influence of size and age. Environ. Pollut. 55:137-148.
Monson, B. A. and P. L. Brezonik. 1999. Influence of food, aquatic humus, and alkalinity on methylmercury uptake by *Daphnia magna*. Environ. Toxicol. Chem. 18:560-566.
Moore, J. W. and D. J. Sutherland. 1980. Mercury concentrations in fish inhabiting two polluted lakes in northern Canada. Water Res. 14:903-907.
Moore, T. R., J. L. Bubier, A. Heyes, and R. J. Flett. 1995. Methyl and total mercury in boreal wetland plants, Experimental Lakes Area, northwestern Ontario. J. Environ. Qual. 24:845-850.
Morel, F. M. M., A. M. L. Kraepiel, and M. Amyot. 1998. The chemical cycle and bioaccumulation of mercury. Ann. Rev. Ecol. Syst. 29:543-566.
Morse, D. 1994. Water quality plan monitoring data report January to March. 1993. Environment Canada, FREMP WQQM-93-02; DOE FRAP. 1993-31. New Westminister, BC.
Mortimer, D. C. 1985. Freshwater aquatic macrophytes as heavy metal monitors - the Ottawa River experience. Environ. Monitor. Assess. 5:311-323.
Mortimer, D. C. and M. Czuba. 1982. Structural damage to leaf chloroplasts of *Elodea densa* caused by methylmercury accumulated from water. Ecotoxicol. Environ. Safe. 6:193-195.
Mortimer, D. C. and A. Kudo. 1975. Interaction between aquatic plants and bed sediments in mercury uptake from flowing water. J. Environ. Qual. 4:491-495.

Mucci, A., M. Lucotte, S. Montgomery, Y. Plourde, P. Pichet, and H. V. Tra. 1995. Mercury remobilization from flooded soils in a hydroelectric reservoir of Northern Quebec, La Grande-2: results of a soil resuspension experiment. Can. J. Fish. Aquat. Sci. 52:2507-2517.
Mulvey, M., M. Newman, A. Chazal, M. M. Keklak, M. G. Heagler, and L. S. Hales, Jr. 1995. Genetic and demographic responses of mosquitofish (*Gambusia holbrooki* Girard 1859) populations stressed by mercury. Environ. Toxicol. Chem. 14:1411-1418.
Nagase, H., Y. Ose, T. Sato, and T. Ishikawa. 1982. Methylation of mercury by humic substances in an aquatic environment. Sci. Total Environ. 24:133-142.
Nagpal, N. K. 1989. Ambient water quality criteria for mercury. Province of British Columbia. Water Management Branch, Ministry of Environment Victoria, BC.
Nelson, D. A., J. E. Miller, and A. Calabrese. 1988. Effect of heavy metals on bay scallops, surf clams, and blue mussels in acute and long-term exposures. Arch. Environ. Contam. Toxicol. 17:595-600.
NESCAUM. 1998. Mercury study: a framework for action. February,. 1998. Northeast States and Eastern Canadian Provinces.
Niimi, A. J. and G. P. Kissoon. 1994h. Evaluation of the critical body burden concept based on inorganic and organic mercury toxicity to rainbow trout (*Oncorhynchus mykiss*). Arch. Environ. Contam. Toxicol. 26:169-178.
Norstrom, R. J., A. E. McKinnon, and A. S. W. DeFreitas. 1976. A bioenergetics-based model for pollutant accumulation by fish-- simulation of PCB and methylmercury residue levels in Ottawa River yellow perch. J. Fish. Res. Board Can. 33:248-267.
Nriago, J.O. (ed). 1979. The biogeochemistry of mercury in the environment. Elsevier/North-Holland Biomedical Press. New York.
Nriagu, J. O. 1989. A global assessment of natural sources of atmospheric trace metals. Nature 338:47-49.
Nriagu, J. O. and J. M. Pacyna. 1988. Quantitative assessment of worldwide contamination of air, water and soils by trace metals. Nature 333:134-139.
Nuzzi, R. 1972. Toxicity of mercury to phytoplankton. Nature 237, 38-40. 1972.
OECD. 1994b. Risk Reduction Monograph Number 4: Mercury. Organization for Economic Co-Operation and Development, Vol.II, 92. Paris, France.
Olson, G. F., D. I. Mount, V. M. Snarski, and T. W. Thorslund. 1975. Mercury residues in fathead minnows, *Pimephales promelas* Rafinesque, chronically exposed to methylmercury in water. Bull. Environ. Contam. Toxicol. 14:129-134.
Oremland, R. S., C. W. Culbertson, and M. R. Winfrey. 1991. Methylmercury decomposition in sediments and bacterial cultures: involvement of methanogens and sulfate reducers in oxidative demethylation. Appl. Environ. Microbiol. 57:130-137.
Pak, K.-R. and R. Bartha. 1998a. Mercury methylation by interspecies hydrogen and acetate transfer between sulfidogens and methanogens. Appl. Environ. Microbiol. 64:1987-1990.
Pak, K.-R. and R. Bartha. 1998b. Mercury methylation and demethylation in anoxic lake sediments and by strictly anaerabic bacteria. Appl. Environ. Microbiol. 64:1013-1017.
Pak, K.-R. and R. Bartha. 1998c. Products of mercury demethylation by sulfidognes and methanogens. Bull. Environ. Contam. Toxicol. 61:690-694.
Parkman, H. and M. Meili. 1993. Mercury in macroinvertebrates from Swedish forest lakes: influence of lake type, habitat, life cycle, and food quality. Can. J. Fish. Aquat. Sci. 50:521-534.
Parks, J. W., C. Curry, D. Romani, and D. D. Russell. 1991. Young northern pike, yellow perch and crayfish as bioindicators in a mercury contaminated watercourse. Environ. Monitor. Assess. 16:39-73.

Parks, J. W., A. Lutz, and J. A. Sutton. 1989. Water column methylmercury in the Wabigoon/English River-Lake system: factors controlling concentrations, speciation, and net production. Can. J. Fish. Aquat. Sci. 46:2184-2202.

Paterson, M. J., J. W. M. Rudd, and V. St.Louis. 1998. Increases in total and methylmercury in zooplankton following flooding of a peatland reservoir. Environ. Sci. Technol. 32:3868-3874.

Pelletier, E. 1985. Mercury-selenium interactions in aquatic organisms: a review. Mar. Environ. Res. 18:111-132.

Pichet, P., K. A. Morrison, I. Rheault, A. Tremblay. 1999. Analysis of total and methylmercury in environmental samples. In: Mercury in the biogeochemical cycle: mercury in natural environments and hydroelectric reservoirs of northern Québec, eds. M. Lucotte, R. Schetagne, N. Thérien, C. Langlois, and A. Tremblay, pp. 41-52. Springer-Verlag Berlin.

Pilgrim, W., Ecological Monitoring and Assessment Network. 1998. Mercury activities and measurements in the eastern Canadian provinces, Ch VIII in. In: Northeast states and eastern Canadian provinces - Mercury Study - a framework for action, ed. M. Tatsutani, pp. 1-64.

Pirrone, N., I. Allegrini, G. J. Keeler, J. O. Nriagu, R. Rossmann, and J. A. Robbins. 1998. Historical atmospheric mercury emissions and depositions in North America compared to mercury accumulations in sedimentary records. Atmos. Envrion. 32:929-940.

Rada, R. G., J. G. Wiener, M. R. Winfrey, and D. E. Powell. 1989. Recent increases in atmospheric deposition of mercury to north- central Wisconsin lakes inferred from sediment analyses. Arch. Environ. Contam. Toxicol. 18:175-181.

Rai, L. C. and R. Dey. 1980 Environmental effects on the toxicity on methyl mercuric chloride to *Chlorella vulgaris*. Acta Hydrochim. Hydrobiol. 8[4], 319-327.

Rai, L. C., J. P. Gaur, and H. D. Kumar. 1981. Protective effects of certain environmental factors on the toxicity of zinc, mercury, and methylmercury to *Chlorella vulgaris*. Environ. Res. 25:250-259.

Ramamoorthy, S. 1982. Mercury analysis in environmental samples. Alberta Environmental Centre, AECV82-M2. Vegreville AB.

Ramamoorthy, S., T. C. Cheng, and D. J. Kushner. 1982. Effect of microbial life stages on the fate of methylmercury in natural waters. Bull. Environ. Contam. Toxicol. 29:167-173.

Ramamoorthy, S., T. C. Cheng, and D. J. Kushner. 1983. Mercury speciation in water. Can. J. Fish. Aquat. Sci. 40:85-89.

Ramamoorthy, S. and D. J. Kushner. 1975. Heavy metal binding sites in river water. Nature 256:399-401.

Ramamoorthy, S., J. W. Moore, and L. George. 1985. Partitioning of mercury in the North Saskatchewan River. Chemosphere.14:1455-1468.

Ramlal, P. S., C. Anema, A. Furutani, R. E. Hecky, and J. W. M. Rudd. 1987. Mercury methylation and demethylation studies at Southern Indian Lake, Manitoba:. 1981 -. 1983. Dept. of Fisheries and Oceans, 1490. Winnipeg, MB.

Ramlal, P. S., C. A. Kelly, J. W. M. Rudd, and A. Furutani. 1993. Sites of methyl mercury production in remote Canadian Shield lakes. Can. J. Fish. Aquat. Sci. 50:972-979.

Ramlal, P. S., J. W. M. Rudd, A. Furutani, and L. Xun. 1985. The effect of pH on methyl mercury production and decomposition in lake sediments. Can. J. Fish. Aquat. Sci. 43:685-692.

Ramlal, P. S., J. W. M. Rudd, and R. E. Hecky. 1986. Methods for measuring specific rates of mercury methylation and degradation and their use in determining factors controlling net rates of mercury methylation. Appl. Environ. Microbiol. 51:110-114.
Rask, M. and M. Verta. 1995. Concentrations and amounts of methylmercury in water and fish in the limed and acid basins of a small lake. Water Air Soil Pollut. 80:577-580.
Ray, S., B. M. Jessop, J. Coffin, and D. A. Swetnam. 1984. Mercury and polychlorinated biphenyls in striped bass (*Morone saxatilis*) from two Nova Scotia rivers. Water Air Soil Pollut. 21:15-23.
Reeder, S. W., A. Demayo, and M. C. Taylor. 1979. Mercury. Water Quality Branch, Inland Waters Directorate, Environment Canada Ottawa, ON.
Rehwoldt, R., L. Lasko, C. Shaw, and E. Wirhowski. 1973. The acute toxicity of some heavy metal ions toward benthic organisms. Bull. Environ. Contam. Toxicol. 10:291-294.
Reish, D. J. 1993. Effects of metals and organic compounds on survival and bioaccumulation in two species of marine gammaridean amphipod, together with a summary of toxicological research on this group. J. Nat. Hist. 27:781-794.
Rieberger, K. 1992. Metal concentrations in fish tissue from uncontaminated B.C. lakes. Water Management Division, Ministry of the Environment, Lands and Parks, Province of British Columbia.
Riisgard, H. U. and S. Hansen. 1990. Biomagnification of mercury in a marine grazing food-chain: algal cells, *Phaeodactylum tricornutum*, mussels, *Mytilus edulis*, and flounders, *Platichthys flesus*, studied by means of a stepwise-reduction-CVAA method. Mar. Ecol. Prog. Ser. 62:259-270.
Rissanen, K., J. Erkama, and J. K. Miettinen. 1970. Experiments on microbiological methylation of mercury (2+) ion by mud and sludge under aerobic and anaerobic conditions. FAO Technical Conference on Marine Pollution and Its Effects on Living Resources and Fishing Rome, Italy.
Robinson, J. B. and O. H. Tuovinen. 1984. Mechanisms of microbial resistance and detoxification of mercury and organomercury compounds: physiological, biochemical, and genetic analyses. Microbiol. Rev. 48:95-124.
Rodgers, D. W. 1994. You are what you eat and a little bit more: bioenergetics-based models of methylmercury accumulation in fish revisited. In: Mercury pollution: integration and synthesis, eds. C. J. Watras and J. W. Huckabee, pp. 427-439. Lewis Publishers Ann Arbor, MI.
Rodgers, D. W. and F. W. H. Beamish. 1983. Water quality modifies uptake of waterborne methylmercury by rainbow trout, *Salmo gairdneri*. Can. J. Fish. Aquat. Sci. 40:824-828.
Rodgers, D. W., M. Dickman, and X. Han. 1995. Stories from old reservoirs: sediment Hg and Hg methylation in Ontario hydroelectric developments. Water Air Soil Pollut. 80:829-839.
Rodgers, D. W. and S. U. Qadri. 1982. Growth and mercury accumulation in yearling yellow perch, *Perca flavescens*, in the Ottawa River, Ontario. Env. Biol. Fish. 7:377-383.
Rodgers, D. W., T. A. Watson, J. S. Langan, and T. J. Wheaton. 1987. Effects of pH and feeding regime on methylmercury accumulation with aquatic microcosms. Environ. Pollut.(A) 45:261-274.
Rossaro, B., G. F. Gaggino, and R. Marchetti. 1986. Accumulation of mercury in larvae and adults, *Chironomus riparius* (Meigen). Bull. Environ. Contam. Toxicol. 37:402-406.
Rouleau, C., K. Borg-Neczak, J. Gottogrey, and H. Tjalve. 1999. Accumulation of waterborne mercury (II) in specific areas of fish brain. Environ. Sci. Technol. 33:3384-3389.
Royal Society of Chemistry. 1994. D.O.S.E. Thomas Graham House, The Science Park. Cambridge.

Rudd, J. W. M. 1995. Sources of methyl mercury to freshwater ecosystems: a review. Water Air Soil Pollut. 80:697-713.
Rudd, J. W. M., A. Furutani, and M. Turner. 1980. Mercury methylation by fish intestinal contents. Appl. Environ. Microbiol. 40:777-782.
Rudd, J. W. M., M. A. Turner, A. Furutani, A. Swick, and B. E. Townsend. 1983. The English-Wabigoon River system: I. A synthesis of recent research with a view towards mercury amelioration. Can. J. Fish. Aquat. Sci. 40:2206-2217.
Sang, S. and B. Lourie. 1997. Mercury emission inventory in Ontario. Environ. Monitor. Assess. 47:79-88.
Saouter, E., L. Hare, P. G. C. Campbell, A. Boudou, and F. Ribeyre. 1993. Mercury accumulation in the burrowing mayfly *Hexagenia rigida* (Ephemeroptera) exposed to CH_3HgCl or Hg Cl_2 in water and sediment. Water Res. 27:1041-1048.
Sarkka, J. 1979. Mercury and chlorinated hydrocarbons in zoobenthos of Lake Paijanne, Finland. Arch. Environ. Contam. Toxicol. 8:161-173.
Schetagne, R., M. Lucotte, N. Thérien, C. Langlois, A. Tremblay. 1999. Synthesis. In: Mercury in the biogeochemical cycle: mercury in natural environments and hydroelectric reservoirs of northern Québec, eds. M. Lucotte, R. Schetagne, N. Thérien, C. Langlois, and A. Tremblay, pp. 295-311. Springer-Verlag Berlin.
Schetagne, R. and R. Verdon. 1999a. Post-impoundment evolution of fish mercury levels at the La Grande complex, Québec, Canada (from. 1978 to. 1996). In: Mercury in the biogeochemical cycle: mercury in natural Environments and hydroelectric reservoirs of northern Québec, eds. M. Lucotte, R. Schetagne, N. Thérien, C. Langlois, and A. Tremblay, pp. 235-258. Springer-Verlag Berlin.
Schetagne, R. and R. Verdon. 1999b. Mercury in fish of natural lakes of northern Québec. In: mercury in the biogeochemical cycle: mercury in natural environments and hydroelectric reservoirs of northern Québec, eds. M. Lucotte, R. Schetagne, N. Thérien, C. Langlois, and A. Tremblay, pp. 115-130. Springer-Verlag Berlin.
Schintu, M., T. Kauri, and A. Kudo. 1989. Inorganic and methyl mercury in inland waters. Water Res. 23:699-704.
Schroeder, W. H., G. Yarwood, and H. Niki. 1991. Transformation processes involving mercury species in the atmosphere - results from a literature survey. Water Air Soil Pollut. 56:653-666.
Scruton, D. A., E. L. Pettigrew, L. J. Ledrew, M. R. Anderson, U. P. Williams, B. A. Bennett, E. L. Hill. 1994. Methylmercury levels in fish tissue from three reservoir systems in insular Newfoundland, Canada. In: Mercury pollution: integration and synthesis, eds. C. J. Watras and J. W. Huckabee, pp. 441-445. Lewis Publishers Boca Raton, FL.
Sellers, P., C. A. Kelly, J. W. M. Rudd, and A. R. MacHutchon. 1996b. Photodegradation of methylmercury in lakes. Nature 380:694-696.
Sharp, J. R. and J. M. Neff. 1980. Effects of the duration of exposure to mercuric chloride on the embryogenesis of the estuarine teleost, *Fundulus heteroclitus*. Mar. Environ. Res. 3:195-213.
Sharp, J. R. and J. M. Neff. 1982. The toxicity of mercuric chloride and methylmercuric chloride to *Fundulus heteroclitus* embryos in relation to exposure conditions. Env. Biol. Fish. 7:277-284.
Shilts, W. W. and W. B. Coker. 1995. Mercury anomalies in lake water and in commercially harvested fish, Kaminak Lake area, District of Keewatin, Canada. Water Air Soil Pollut. 80:881-884.

Sigmon, C. F., H. J. Kania, and P. J. Beyers. 1977. Reductions in biomass and diversity resulting from exposure to mercury in artificial streams. J. Fish. Res. Board Can. 34:493-500.
Slooff, W., P. F. H. Bont, M. van Ewijk, and J. A. Janus. 1991. Exploratory report mercury. National Institute of Public Health and Environmental Protection, 710401006. Bilthoven, The Netherlands.
Smith, C. M. and C. Rowan-West. 1996. Mercury in Massachusetts: An evaluation of sources, emissions, impacts and controls. Mass. Dept. of Environ. Protection Massachusetts.
Snarski, V. M. and G. F. Olson. 1982a. Chronic toxicity and bioaccumulation of mercuric chloride in the fathead minnow (*Pimephales promelas*). Aquat. Toxicol. 2:143-156.
Snell, T. W., B. D. Moffat, C. Janssen, and G. Persoone. 1991. Acute toxicity tests using rotifers. III. Effects of temperature, strain and exposure time on the sensitivity of *Brachionus plicatilis*. Environ. Toxicol. Water Qual. An Inter. J. 6:63-75.
Sorensen, J. A., G. E. Glass, K. W. Schmidt, J. K. Huber, and G. R. Rapp Jr. 1990. Airborne mercury deposition and watershed characteristics in relation to mercury concentrations in water, sediments, plankton, and fish of eighty northern Minnesota lakes. Environ. Sci. Technol. 24:1716-1727.
Southworth, G. R., M. J. Peterson, and R. R. Turner. 1994. Changes in concentrations of selenium and mercury in largemouth bass following elimination of fly ash discharge to a quarry. Chemosphere.29:71-79.
Spehar, R. L. and J. T. Fiandt. 1986. Acute and chronic effects of water quality criteria-based metal mixtures on three aquatic species. Environ. Toxicol. Chem. 5:917-931.
Spry, D. J. and J. G. Wiener. 1991. Metal bioavailability and toxicity to fish in low-alkalinity lakes: a critical review. Environmental Pollution 71:243-304.
St.Louis, V. L., J. W. M. Rudd, C. A. Kelly, and L. A. Barrie. 1995a. Wet deposition of methyl mercury in northwestern Ontario compared to other geographic locations. Water Air Soil Pollut. 80:405-414.
St.Louis, V. L., J. W. M. Rudd, C. A. Kelly, K. G. Beaty, N. S. Bloom, and R. J. Flett. 1994. Importance of wetlands as sources of methyl mercury to boreal forest ecosystems. Can. J. Fish. Aquat. Sci. 51:1065-1076.
Stephens, G. R. 1995. Mercury concentrations in fish in a remote Canadian arctic lake. Water Air Soil Pollut. 80:633-636.
Stokes, P. M., R. C. Bailey, and G. R. Groulx. 1985. Effects of acidification on metal availability to aquatic biota, with special reference to filamentous algae. Environ.Health Perspectives 63:79-87.
Stokes, P. M., and C. D. Wren. 1987. Bioaccumulation of mercury by aquatic biota in hydroelectric reservoirs: a review and consideration of mechanisms. In: Lead, Mercury, Cadmium and Arsenic in the Environment, eds. T. C. Hutchinson and K. M. Meema, pp. 255-277. John Wiley and Sons Ltd. Toronto, ON.
Stortelder, P. B., M. A. van der Gaag, and L. A. van der Kooij. 1989. An ecotoxicological basis for quality objectives for water and sediment. Part 1. Results and calculations. Perspectives for water organisms. Institute for Inland Water Management and Waste Water Treatment, DBW/RIZA Memorandum No. 89.016a (English Version August. 1991). The Netherlands.
Stromgren, T. 1980. The effect of lead, cadmium, and mercury on the increase in length of five intertidal fucales. J. Exper. Mar. Biol. Ecol. 43:107-119.
Suchanek, T. H., P. J. Richerson, L. A. Woodward, D. G. Slotton, L. J. Holts, and C. E. Woodmansee. 1993. A survey and evaluation of mercury. In: Sediment, water, plankton, periphyton, benthic invertebrates and fishes within the aquatic ecosystem of Clear

Lake, California. Preliminary lake study. U.S. Environmental Protection Agency, Region 9, Superfund Program.
Summers, A. O. 1986. Organization, expression, and evolution of genes for mercury resistance. Ann.Rev.Microbiol. 40:607-634.
Summers, A. O. and S. Silvers. 1978. Microbial transformations of metals. Ann.Rev.Microbiol. 32:637-672.
Sunderland, E. M. and G. L. Chmura 2000. The history of mercury emissions from fuel combustion in Maritime Canada. Environ. Pollut. 110:297-306.
Suns, K. and G. Hitchin. 1990. Interrelationships between mercury levels in yearling yellow perch, fish condition and water quality. Water Air Soil Pollut. 650:255-265.
Tatara, C. P., M. Mulvey, and M. Newman. 1999. Genetic and demographic responses of mosquitofish (*Gambusia holbrooki*) populations exposed to mercury for multiple generations. Environ. Toxicol. Chem. 18:2840-2845.
Thain, J. E. 1984. Effects of mercury on the prosobranch mollusc *Crepidula fornicata*: acute lethal toxicity and effects on growth and reproduction of chronic exposure. Mar. Environ. Res. 12:285-309.
Thérien, N., K. A. Morrison. 1999. *In vitro* release of mercury from flooded organic matter. In: Mercury in the biochemical cycle: mercury in natural environments and hydroelectric reservoirs of northern Québec, eds. M. Lucotte, R. Schetagne, N. Thérien, C. Langlois, and A. Tremblay, pp. 147-164. Springer-Verlag Berlin.
Thomas, D. L. and J. G. Montes. 1978. Spectrophotometrically assayed inhibitory effects of mercuric compounds on *Anabaena flos-aquae* and *Anacystis nidulans* (Cyanophyceae). J. Phycol. 14:494-499.
Tompkins, T. and D. W. Blinn. 1976. The effect of mercury on the growth rate of *Fragilaria crotonensis* Kitton and *Asterionella formosa* Hass. Hydrobiologia 49[2], 111-116.
Tremblay, A. 1999. Bioaccumulation of mercury and methylmercury in invertebrates from natural boreal lakes. In: Mercury in the biochemical cycle: mercury in natural environments and hydroelectric reservoirs of northern Québec, eds. M. Lucotte, R. Schetagne, N. Thérien, C. Langlois, and A. Tremblay, pp. 89-113. Springer-Verlag Berlin.
Tremblay, A., M. Lucotte, and I. Rheault. 1996. Methylmercury in a benthic food web of two hydroelectric reservoirs and a natural lake of northern Québec (Canada). Water Air Soil Pollut. 91:255-269.
Trudel, M. and J. B. Rasmussen. 1997. Modeling the elimination of mercury by fish. Environ. Sci. Technol. 31:1716-1722.
U.S.Dept.of Health and Human Services. 1994a. Toxicological profile for mercury. Clement International Corp. for Public Health Service, TP-93/10.
U.S.EPA. 1980. Unpublished laboratory data. U.S. EPA Narragansett,Rhode Island.
U.S.EPA. 1985. Ambient water quality criteria for mercury --. 1984. Criteria and Standards Division, U.S. EPA, EPA-440/5-84-026. Washington DC.
U.S.EPA. 1995a. Great Lakes Water Quality Initiative Criteria Documents for the Protection of Aquatic Life in Ambient Water. EPA-820-B-95-004. U.S. EPA, Office of Water. Washington, D.C.
U.S.EPA. 1995b. Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife: DDT, mercury, 2,3,7,8-TCDD, PCBs. [EPA-820-B-95-008]. U.S. EPA, Office of Water. Washington, DC.
U.S.EPA. 1995c. Method 1669 - Method for sampling ambient water for determination of metals at EPA ambient criteria levels. Office of Science and Technology, Office of Water, Engineering and Analysis Division Washington, DC.

U.S.EPA. 1996a. Method 1631 - Mercury in water by oxidation, purge and trap, and CVFAS. Office of Science and Technology, Office of Water, Engineering and Analysis Division Washington, DC.
U.S.EPA. 1997a. Mercury study report to congress. Vol. III: Fate and transport of mercury in the environment. Office of Research and Development Washington, DC.
U.S.EPA. 1997b. Mercury study report to Congress. Vol VI: An ecological assessment for anthropogenic mercury emissions in the United States. Office of Research and Development, 8 chapters. Washington, DC.
U.S.EPA. 1997c. Mercury study report to Congress. Vol VII: Characterization of human health and wildlife risks from mercury exposure in the United States. Office of Research and Development Washington, DC.
U.S.EPA. 1999. National recommended water quality criteria - correction. Office of Water, U.S. EPA, EPA 822-Z-99-001.
Vandal, G., R. P. Mason, and W. F. Fitzgerald. 1991. Cycling of volatile mercury in temperate lakes. Water Air Soil Pollut. 56:791-803.
Verdon, R., D. Brouard, C. Demers, R. Lalumiere, M. Laperle, and R. Schetagne. 1991. Mercury evolution (1978-1988) in fishes of the La Grande Hydroelectric Complex, Quebec, Canada. Water Air Soil Pollut. 56:405-417.
Verdon, R., A. Tremblay. 1999. Mercury accumulation in fishes from the La Grande Complex: influence of feeding habits and concentrations of mercury in ingested prey. In: Mercury in the biogeochemical cycle: mercury in natural environments and hydroelectric reservoirs of northern Québec, eds. M. Lucotte, R. Schetagne, N. Thérien, C. Langlois, and A. Tremblay, pp. 215-233. Springer-Verlag Berlin.
Verma, S. R., R. Chand, and I. P. Tonk. 1986. HgCl-induced physiological dysfunction in *Notopterus notopterus*. Environ. Res. 39:442-447.
Verta, M., S. Rekolainen, and K. Kinnunen. 1986b. Causes of increased fish mercury levels in Finnish reservoirs. Water Research Institute, National Board of Waters, Finland. No. 65.
Visman, V., G. Mierle, and D. J. McQueen. 1995. Uptake of aqueous methylmercury by larval *Chaoborus americanus*. Water Air Soil Pollut. 80:1007-1010.
Waiser, M. J. and R. D. Robarts. 1995. Microbial nutrient limitation in prairie saline lakes with high sulfate concentration. Limnol. Oceanogr. 40:566-574.
Walczak, B. Z., U. T. Hammer, and P. M. Huang. 1986. Ecophysiology and mercury accumulation of rainbow trout (*Salmo gairdneri*) when exposed to mercury in various concentrations of chloride. Can. J. Fish. Aquat. Sci. 43:710-714.
Warnau, M., M. Iaccarino, A. De Biase, A. Temara, M. Jangoux, P. Dubois, and G. Pagano. 1996. Spermiotoxicity and embryotoxicity of heavy metals in the echinoid *Paracentrotus lividus*. Environ. Toxicol. Chem. 15:1931-1936.
Watras, C. J. and N. S. Bloom. 1992. Mercury and methylmercury in individual zooplankton: implications for bioaccumulation. Limnol.Oceanogr. 37:1313-1318.
Watras, C. J., N. S. Bloom, R. J. M. Hudson, S. Gherini, R. K. Munson, S. A. Claas. 1994. Sources and fates of mercury and methylmercury in Wisconsin lakes. In: Mercury pollution: integration and synthesis pp. 153-177. Lewis Publishers Boca Raton, Florida.
Watras, C. J. and J. W. Huckabee. 1994. Mercury pollution: integration and synthesis. Lewis Publishers. Boca Raton, FL.
Watras, C. J., K. A. Morrison, and J. S. Host. 1995. Concentration of mercury species in relationship to other site- specific factors in the surface waters of northern Wisconsin lakes. Limnol.Oceanogr. 40:556-565.

Watson, T. 1992. Evaluation of mercury concentration in selected environmental receptors in the Williston Lake and Peace River areas of British Columbia. Triton Environmental Consultants Ltd. Vancouver, BC.
Weber, J. H. 1993. Review of possible paths for abiotic methylation of mercury (II) in the aquatic environment. Chemosphere. 26:2063-2077.
Weis, J. S. and P. Weis. 1989. Effects of environmental pollutants on early fish development. Rev. Aquat. Sci. 1:45-73.
Westcott, K. and J. Kalff. 1996. Environmental factors affecting methyl mercury accumulation in zooplankton. Can. J. Fish. Aquat. Sci. 53:2221-2228.
Wester, P. W. 1991. Histopathological effects of environmental pollutants beta-HCH and methyl mercury on reproductive organs in freshwater fish. Comp. Biochem. Physiol. 100C:237-239.
Williamson, D. A. 1983. Surface water quality management proposal. Manitoba Dept. of Environment and Workplace Safety and Health Winnipeg, MB.
Williamson, D. A. 1988. Manitoba surface water quality objectives and rationale document supporting revisions to Manitoba surface water quality objectives. Water Standards Section Report No. 88-5 Winnipeg, MB.
Winfrey, M. R. and J. W. M. Rudd. 1990. Environmental factors affecting the formation of methylmercury in low pH lakes. Environ. Toxicol. Chem. 9:853-869.
Wobeser, G. 1975. Acute toxicity of methyl mercury chloride and mercuric chloride for rainbow trout (*Salmo gairdneri*) fry and fingerlings. J. Fish. Res. Board Can. 32:2005-2013.
Wood, J. M., F. S. Kennedy, and C. G. Rosen. 1968. Synthesis of methylmercury compounds by extracts of a methanogenic bacterium. Nature 220:173-174.
World Health Organization. 1989. Mercury- Environmental Aspects. World Health Organization, 86. Geneva, Switz.
World Health Organization. 1990. Methyl mercury. Vol. 101. Geneva, Switz.
Wren, C. D. and H. R. MacCrimmon. 1983. Mercury levels in the sunfish, *Lepomis gibbosus*, relative to pH and other environmental variables of Precambrian Shield lakes. Can. J. Fish. Aquat. Sci. 40:1737-1744.
Wren, C. D., W. A. Scheider, D. L. Wales, B. W. Muncaster, and I. M. Gray. 1991. Relation between mercury concentrations in walleye (*Stizostedion vitreum vitreum*) and northern pike (*Esox lucius*) in Ontario lakes and influence of environmental factors. Can. J. Fish. Aquat. Sci. 48:132-139.
Wren, C. D. and G. L. Stephenson. 1991. The effect of acidification on the accumulation and toxicity of metals to freshwater invertebrates. Environ. Pollut. 71:204-241.
Xun, L., N. E. R. Campbell, and J. W. M. Rudd. 1987. Measurements of specific rates of net methyl mercury production in the water column and surface sediments of acidified and circumneutral lakes. Can. J. Fish. Aquat. Sci. 44:750-757.
Zauke, G. P. 1977. Mercury in benthic invertebrates of the Elbe Estuary. Helgolander wissenschaftliche Meeresuntersuchungen 29:357-374.
Zepp, R. G. 1987. Nitrate-induced photooxidation of trace organic chemicals in water. Environ. Sci. Technol. 21:443-450.
Zhang, L. and D. Planas. 1994. Biotic and abiotic mercury methylation and demethylation in sediments. Bulletin of Environmental Contamination and Toxicology 52, 691-698.
Zillioux, E. J., D. B. Porcella, and J. M. Benoit. 1993. Mercury cycling and effects in freshwater wetland ecosystems. Environ. Toxicol. Chem. 12:2245-2264.

Appendix A

The following four examples provide generic calculations intended as a guide to determine the relationship between the WQG and the TRG. They do not consider many site-specific factors including wildlife species of concern and their preferred diet, or site- or species-specific bioaccumulation factors and, therefore, should not be used as site-specific water quality objectives (WQOs). In these examples, reference concentrations for selected wildlife species from the TRG technical supporting document were used in conjunction with bioaccumulation factors (BAFs) estimated by the U.S. EPA, to determine concentrations of methylmercury in water that would protect wildlife that consume aquatic biota (Environment Canada 2002).

RCs for four species of wildlife were chosen for use in examples: Wilson's storm petrel (*Oceanites oceanicus*), mink (*Mustela vision*), herring gull (*Larus argentatus*), and bald eagle (*Haliaeetus leucocephalus*). Wilson's storm petrel was chosen because its RC is the lower limit of the range given in the TRG report, and therefore, is the national TRG for methylmercury. Mink and herring gull were chosen because both species are used commonly in monitoring programs, but feed at different trophic levels; 3 and 4, respectively. Bald eagle was chosen as an example of a species that feeds at multiple trophic levels, and because its RC value is the upper limit of the range given in the TRG report (Environment Canada 2002).

Example 1: Wilson's Storm Petrel

[MeHg] in water = $RC \div BAF_4$

where;

[MeHg] in water = the concentration of MeHg in water below which methylmercury would not bioaccumulate in tissues of aquatic life above the TRG. Adverse effects are, therefore, not expected to occur in any species of Canadian wildlife that consume aquatic life;

RC = reference concentration for Wilson's storm petrel,
= Canadian TRG of 33 µg MeHg/kg ww; and

BAF_4 = bioaccumulation factor for trophic level 4 fish 6.8×10^6 as determined by the U.S. EPA (1997). The trophic level 4 BAF is a conservative assumption to capture all wildlife species.

[MeHg in water] = $33 \div 6.8 \times 10^6$
= 0.000005 µg MeHg/L
= 5 pg MeHg/L
= 4.7 pg Hg/L; adjusted for the MW of Hg
= 6.7 pg Hg/L in an unfiltered water sample

Therefore, concentrations of MeHg in unfiltered water less than 7 pg Hg/L would not be expected to result in adverse health effects in Canadian wildlife.

Example 2: Mink

The reference concentration for MeHg in aquatic life that protects mink is 92 µg MeHg/kg ww (Environment Canada 2002). Because mink feed at trophic level 3, the BAF for trophic level 3 fish (BAF_3) of 1.6×10^6 is used in this example (U.S. EPA. 1997).

[MeHg] in water = $RC \div BAF_3$
= $92 \div 1.6 \times 10^6$
= 0.00005754 µg MeHg/L
= 57.5 pg MeHg/L
= 53.5 pg Hg/L; adjusted for the MW of Hg
= 76.4 pg Hg/L in an unfiltered water sample

Therefore, concentrations of MeHg in unfiltered water less than 76 pg Hg/L would not be expected to result in adverse health effects in mink.

Example 3: Herring Gull

The reference concentration for MeHg in aquatic life that protects herring gulls is 111 µg MeHg/kg ww (Environment Canada 2002). Because herring gulls feed at trophic level 4, the BAF for trophic level 4 fish of 6.8×10^6 is used in this example (U.S. EPA. 1997).

[MeHg] in water = $RC \div BAF_4$
= $111 \div 6.8 \times 10^6$
= 0.0000163 µg MeHg/L
= 16.3 pg MeHg/L
= 15.2 pg Hg/L; adjusted for the MW of Hg
= 21.7 pg Hg/L in an unfiltered water sample

Therefore, concentrations of MeHg in unfiltered water less than 22 pg Hg/L would not be expected to result in adverse health effects in herring gulls.

Example 4: Bald Eagle

This is an example of how to calculate a concentration of MeHg in water that would be protective of a species that feeds at more than one trophic level. In this case the bald eagle feeds 80% at trophic level 3 and 20% at trophic level 4. The reference concentration for MeHg in aquatic life that protects bald eagles is 282 µg MeHg/kg ww (Environment Canada 2002).

[MeHg] in water = $0.8(RC \div BAF_3) + 0.2(RC \div BAF_4)$
= $0.8(282 \div 1.6 \times 10^6) + 0.2(282 \div 6.8 \times 10^6)$
= 0.000141 + 0.0000083
= 0.000149 µg MeHg/L
= 149 pg MeHg/L
= 138.6 pg Hg/L; adjusted for the MW of Hg[34]
= 198.0 pg Hg/L in an unfiltered water sample[35]

Concentrations of MeHg in unfiltered water above 200 pg Hg/L could, therefore, pose a risk to wildlife in that this level could result in the accumulation of MeHg in fish tissues to levels

[34] We used molecular weights (MW) of 200.6 and 215.6 g/mol for Hg^{2+} and MeHg, respectively.

[35] We assumed that the dissolved portion of methylmercury accounts for 70% of methylmercury in an unfiltered water sample (see Section 4.2).

above all RCs. A water concentration of 7 pg Hg/L or less is thought to be protective of all wildlife species that consume aquatic biota. Concentrations in-between these two limits may be a hazard to some wildlife depending on their RC, their feeding preferences, and the BAF for their prey species. Because these estimates are based on national RCs and on field-derived BAFs, they are believed to reasonably represent average conditions. These upper (200 pg Hg/L) and lower (7 pg Hg/L) limits for MeHg concentrations in water to protect wildlife are 10- and 1000-fold, respectively, below the WQG of 4 ng Hg/L (or 4 000 pg Hg/L); see Chapter 3.

Site-specific WQOs are preferred wherever possible and they may be calculated using the basic approach presented above, modified with species- and site-specific data. For example, these calculations should employ the RC for the most appropriate wildlife species of concern at a given site. RCs themselves may be adjusted with site-specific food intake:body weight ratios or with a safety factor (note that none was used on the derivation of the national RCs). Site-specific WQOs may consider also the measured proportions of aquatic life at each trophic level in the diet of a particular wildlife species. In addition, BAFs specific to the aquatic species comprising the diet of the wildlife species should be used.