Rotterdam Convention Operation of the Prior Informed Consent (PIC) procedure for banned or severely restricted chemicals

Decision Guidance Document

Tetraethyl lead and tetramethyl lead

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

Introduction

The objective of the Rotterdam Convention is to promote shared responsibility and co-operative efforts among Parties in the international trade of certain hazardous chemicals in order to protect human health and the environment from potential harm and to contribute to their environmentally sound use, by facilitating information exchange about their characteristics by providing for a national decision-making process on their import and export and by disseminating these decisions to Parties. The interim secretariat of the Convention is provided jointly by the United Nations Environment Programme (UNEP) and the Food and Agriculture Organisation of the United Nations (FAO).

Candidate chemicals[1] for the Rotterdam Convention include those that have been banned or severely restricted by national regulatory actions in two or more Parties[2] in two different regions. Inclusion of a chemical in the Convention is based on regulatory actions taken by Parties that have addressed the risks associated with the chemical by banning or severely restricting it. Other ways might be available to control/reduce such risks. However, inclusion does not imply that all Parties to the Convention have banned or severely restricted this chemical. For each chemical included in the Rotterdam Convention, Parties are requested to make an informed decision whether they consent or not to the future import of the chemical.

At its eleventh session, held in Geneva on 18 September the Intergovernmental Negotiating Committee adopted the decision guidance document for tetraethyl lead and tetramethyl lead with the effect that these chemicals became subject to the interim PIC procedure.

At its first meeting, held in Geneva 20 to 24 September 2004, the Conference of the Parties agreed to include tetraethyl lead and tetramethyl lead in Annex III of the Rotterdam Convention, with the effect that these chemicals became subject to the PIC procedure.

The present decision guidance document was communicated to the Designated National Authorities on 1 February 2005 in accordance with Articles 7 and 10 of the Rotterdam Convention.

Purpose of the Decision Guidance Document

For each chemical included in Annex III of the Rotterdam Convention a decision guidance document has been approved by the Conference of the Parties. Decision guidance documents are sent to all Parties with a request that they provide a decision regarding future import of the chemical.

The decision guidance document is prepared by the Chemical Review Committee (CRC). The CRC is a group of government designated experts established in line with Article 18 of the Convention, that evaluates candidate chemicals for possible inclusion in the Convention. The decision guidance document reflects the information provided by two or more Parties in support of the national regulatory actions to ban or severely restrict the chemical. It is not intended as the only source of information on a chemical nor is it updated or revised following its adoption by the Conference of the Parties.

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[1] "'Chemical' means a substance whether by itself of in a mixture or preparation and whether manufactured or obtained from nature, but does not include any living organism. It consists of the following categories: pesticide (including severely hazardous pesticide formulations) and industrial."

[2] "'Party' means a State or regional economic integration organization that has consented to be bound by this Convention and for which the Convention is in force."

There may be additional Parties that have taken regulatory actions to ban or severely restrict the chemical as well as others that have not banned or severely restricted it. Such risk evaluations or information on alternative risk mitigation measures submitted by Parties may be found on the Rotterdam Convention web-site (www.pic.int).

Under Article 14 of the Convention, Parties can exchange scientific, technical, economic and legal information concerning the chemicals under the scope of the Convention including toxicological, ecotoxicological and safety information. This information may be provided directly to other Parties or through the Secretariat. Information provided to the Secretariat will be posted on the Rotterdam Convention website.

Information on the chemical may also be available from other sources.

Disclaimer

The use of trade names in this document is primarily intended to facilitate the correct identification of the chemical. It is not intended to imply any approval or disapproval of any particular company. As it is not possible to include all trade names presently in use, only a number of commonly used and published trade names have been included in this document.

While the information provided is believed to be accurate according to data available at the time of preparation of this Decision Guidance Document, the Food and Agriculture Organization of the United Nations (FAO) and the United Nations Environment Programme (UNEP) disclaim any responsibility for omissions or any consequences that may flow there from. Neither FAO nor UNEP shall be liable for any injury, loss, damage or prejudice of any kind that may be suffered as a result of importing or prohibiting the import of this chemical.

The designations employed and the presentation of material in this publication do not imply the expression of any opinion whatsoever on the part of FAO or UNEP concerning the legal status of any country, territory, city or area or of its authorities or concerning the delimitation of its frontiers or boundaries.

ABBREVIATIONS WHICH MAY BE USED IN THIS DOCUMENT

(N.B. Chemical elements and pesticides are not included in this list)

<	less than
≤	less than or equal to
<<	much less than
>	greater than
≥	greater than or equal to
µ	micro- (÷ 1 000 000)
µg	microgram
µm	micrometre
ArfD	acute reference dose
a.i.	active ingredient
ADI	acceptable daily intake
ADP	adenosine diphosphate
ATP	adenosine triphosphate
b.p.	boiling point
bw	body weight
^{o}C	degree Celsius (centigrade)
CA	Chemicals Association
CAS	Chemical Abstracts Service
cc	cubic centimetre
CHO	Chinese hamster ovary
cm	centimetre
CNS	central nervous system
d	deci (÷ 10)
dl	decilitre
DNA	deoxyribose nucleic acid
E.C.	European Community
EC_{50}	effect concentration, 50%
ED_{50}	effect dose, 50%
EEC	European Economic Community
EHC	Environmental Health Criteria
EINECS	European Inventory of Existing Commercial Substances
FAO	Food and Agriculture Organization of the United Nations
g	gram
h	hour
ha	hectare
i.m.	intramuscular
i.p.	intraperitoneal
IARC	International Agency for Research on Cancer

ABBREVIATIONS WHICH MAY BE USED IN THIS DOCUMENT

(N.B. Chemical elements and pesticides are not included in this list)

IC_{50}	inhibition concentration, 50%
ILO	International Labour Organisation
IPCS	International Programme on Chemical Safety
IOMC	Inter-Organization Programme for the Sound Management of Chemicals
IUPAC	International Union of Pure and Applied Chemistry
JMPR	Joint FAO/WHO Meeting on Pesticide Residues (Joint Meeting of the FAO Panel of Experts on Pesticide Residues in Food and the Environment and a WHO Expert Group on Pesticide Residues)
k	kilo- (x 1000)
kg	kilogram
Koc	organic carbon-water partition coefficient
l	litre
LC_{50}	lethal concentration, 50%
LD_{50}	lethal dose, 50%
LD_{LO}	lowest lethal dose
LOAEL	lowest observed adverse effect level
LOEL	lowest observed effect level
m	metre
m.p.	melting point
mg	milligram
ml	millilitre
mPa	milliPascal
MMT	methyl cyclopentadienyl manganese tricarbonyl
MTBE	methyl tertiary butyl ether
MTD	maximum tolerated dose
ng	nanogram
NOAEL	no-observed-adverse-effect level
NOEL	no-observed-effect level
NTP	National Toxicology Program
OECD	Organisation for Economic Co-operation and Development
PbA	lead in air
PbB	lead in blood
PCM	phase contrast microscopy
Pow	octanol-water partition coefficient
ppm	parts per million (used only with reference to the concentration of a pesticide in an experimental diet. In all other contexts the terms mg/kg or mg/l are used).
RfD	reference dose for chronic oral exposure (comparable to ADI)
RTECS	Registry of Toxic Effects of Chemical Substances
SMR	standardized mortality ratio
STEL	short term exposure limit

ABBREVIATIONS WHICH MAY BE USED IN THIS DOCUMENT

(N.B. Chemical elements and pesticides are not included in this list)

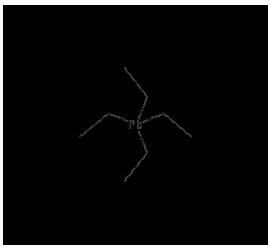
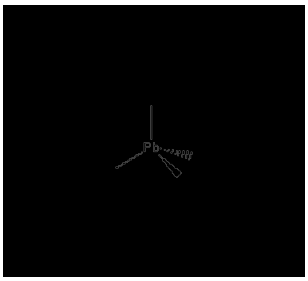
t	metric tonne
TEL	tetraethyl lead
TML	tetramethyl lead
TLV	threshold limit value
TWA	time weighted average
UNEP	United Nations Environment Programme
USEPA	United States Environmental Protection Agency
UV	ultraviolet
VOC	volatile organic compound
WHO	World Health Organization
wt	weight

PIC Decision guidance document for a banned or severely restricted chemical

TETRAETHYL LEAD AND TETRAMETHYL LEAD

Published:1 February 2005

1. Identification and uses (see Annex 1)

	Tetraethyl lead	Tetramethyl lead
Common name		
Chemical name	Plumbane, tetraethyl-	Plumbane, tetramethyl
Other names/ synonyms	TEL	TML
Structural formula		
CAS-No.(s)	78-00-2	75-74-1
Other CAS numbers that may be used		
Harmonized System Customs Code	3811.11	3811.11
Other numbers:	UN No: 1649 E.C. Customs No: 2931 00 95 EINECS No: 201-075-4 RTECS No: TP4550000	UN No: 1649 E.C. Customs No: 2935 00 95 EINECS No: 200-897-0 RTECS No: TP4725000
Category	Industrial	
Regulated Category	Industrial	
Use(s) in regulated category	TEL and TML are used in gasoline as a motor anti-knock additive. They are added to fuels to raise their octane rating to allow higher compression ratios without the damaging effects of knock, also called pre-detonation or pre-ignition.	
Trade names		
Formulation types	The pure grade of TEL and TML are rarely sold; it is more usual to find TEL and TML in mixtures such as antiknock preparations and leaded gasoline. A typical formulation to produce automotive gasoline consists of about 62% tetraethyl lead (TEL), 18% ethylene dibromide (scavenger for lead), 18% ethylene dichloride (scavenger for lead), and 2% of other ingredients, such as dye, petroleum solvent, and stability improver. A typical formulation to produce aviation gasoline includes about 61-62% TEL, 35-36% ethylene dibromide, and 3% of dye, solvent, inhibitor, etc. For overall best performance of aviation piston engines, the scavenger for lead consists entirely of ethylene dibromide. Another type of additive is made by mixing TEL with TML to produce physical mixtures containing 10-75% TML.	
Uses in other categories	No other applications have been reported by the notifying Parties (Canada and E.C.).	
Basic manufacturers	Octel Corporation, Ethyl Corporation *This is an indicative list of current and former manufacturers and is not intended to be exhaustive.*	

2. Reasons for inclusion in the PIC procedure

Tetraethyl lead and tetramethyl lead have been severely restricted as industrial chemicals by both notifying Parties.

2.1 Final regulatory action: (see Annex 2 for details)

Canada

The final regulatory action restricts the use of leaded gasoline and limits the concentration of TEL and TML in leaded gasoline. Leaded gasoline can be used in farm equipment, boats or heavy trucks. The average lead concentration in leaded gasoline for these uses must not exceed 26 mg/l. The actual lead content must never exceed 30 mg/l at any time. The maximum lead concentration for [unleaded] gasoline used for any other purposes is 5 mg/l. The final regulatory action does not apply to aircraft. High performance competition vehicles (cars, boats, snowmobiles) are also exempted until January 1, 2008. The exemption means that there are no restrictions on the lead content of aviation gasoline and gasoline for high performance competition vehicles.

Reason: Human Health

European Community

The final regulatory action restricts the use of leaded gasoline and limits the concentration of TEL and TML in leaded gasoline. The final regulatory action bans the placing on the market of leaded gasoline for vehicles. The marketing of leaded gasoline containing not more than 150 mg/l lead remains allowed until January 1, 2005, provided that it can be demonstrated that a ban would result in severe socio-economic problems or would not lead to overall environmental or health benefits. There is also a derogation for small quantities of leaded gasoline containing not more than 150 mg/l, up to a maximum of 0.5% of total sales, for collectors' old cars. The lead content of gasoline for aircraft is not covered by the final regulatory action.

Reason: Human Health

2.2 Risk evaluation

Canada

The final regulatory action was taken to protect human health based on the toxicity of lead, not on the basis of the toxicological profile of TEL or TML. Signs and symptoms of chronic and subchronic lead poisoning include anorexia, constipation, abdominal cramp (colic), pallor (anemia), headache, irritability, fatigue, and peripheral neuropathy (wrist drop, ankle drop). In severe poisoning episodes, impaired kidney function, heart damage, mental retardation, convulsions, coma, encephalopathy and death are found.

In young children, relatively low blood lead levels cause neurological and behavioural changes: hyperactivity, lower learning capacity, reduced attention span, fearfulness, and general regression, among others. Lead also inhibits the activity of the brain enzyme dihydropteridine reductase, essential for the synthesis of several neurotransmitters.

Health Canada determined from studies that adverse health effects can occur at blood lead levels of 20-30µg/dl. Furthermore, these studies indicated that:

- significant numbers of Canadian children could have blood levels in this range and could therefore be at risk. Results from a study in Toronto in 1982 indicated that 1% of children aged 0-4 years had blood lead concentrations greater than 30µg/dl and 12% greater than 20µg/dl; and
- gasoline lead contributed an estimated 30-35% of the lead in the blood of urban adults. The proportion of lead in children's blood originating from gasoline may range from 30-40%. These estimates were based on a balance scheme that took into account several sources of lead intake by humans (air, water, food and dust) and the following data:

- average lead concentration in urban areas of 0.54 µg/m^3. It was assumed that gasoline lead accounted for 88% of all lead released to the atmosphere in Canada;
- average lead concentration in dust and dirt of 850 µg/g (50%-75% of this was assumed to originate from lead in gasoline);
- amount of fallout lead in adult daily diet of 13 µg/day and children daily diet of 7.1 µg/day (88% of this was assumed to originate from lead in gasoline).

European Community

The final regulatory action was taken to protect human health based on the toxicity of lead, not on the basis of the toxicological profile of TEL or TML. Humans are exposed directly through inhalation of lead emitted into the air, which also acts to transport lead into other human exposure media, including dust, soil, food and water. Since the beginning of the 1970s, it has been recognised that the combustion of alkyl lead additives in motor fuels accounts for the major part of all lead emissions and blood lead levels increase with traffic density. At the same time, it became evident that lead concentrations steadily increased in various environmental compartments such as air and soil in close connection with the increase of motor vehicle traffic.

3. Protective measures that have been applied concerning the chemical

3.1 Regulatory measures to reduce exposure

Canada	The only regulatory measure to reduce exposure is the one to restrict the use of TEL and TML in leaded gasoline.
European Community	The only regulatory measure to reduce exposure is the one to restrict the use of TEL and TML in leaded gasoline.

3.2 Other measures to reduce exposure

Canada

In 1994, the Federal Provincial Advisory Committee on Environmental and Occupational Health has recommended 10µg/dl as the national "action level" for blood lead levels in individual children or adults, that is the level at which intervention is appropriate.

European Community

In 1977, the E.C. introduced a directive on biological screening of the population for lead (77/312/EEC) designed to identify and control sources of unacceptable lead exposure. In each Member States, 50 or more blood lead samples had to be analysed per million inhabitants.

International Initiatives

Agenda 21 of the United Nations Conference on Environment and Development includes a commitment to reduce lead exposure, although this does not specifically call for actions with regard to lead in gasoline.
In December 1994, at the Summit of the Americas, Heads of State from a number of countries pledged to develop national action plans for the phase out of leaded gasoline in the Western Hemisphere.
At the April 1996 Transatlantic Automotive Industry Conference on International Regulatory Harmonization, automotive industry representatives from Europe and the Americas supplied joint recommendations for automotive standards to the United States government and the European Union. This group also noted as an "Area of Potential Cooperation with Respect to Other Countries (e.g., Emerging Markets)" implementation of a lead phase-out as a necessary step to regulatory harmonization.
In May 1996, the World Bank called for a global phase out of leaded gasoline, and offered to help countries design feasible phase out schedules and create incentive frameworks. This general initiative has been reflected in a series of specific projects, including support for several national governments in developing tax, price, and market liberalization policies to facilitate lead phase-out.

In June 1996, the second United Nations Conference on Human Settlements, Habitat II, included the elimination of lead from gasoline as a goal in its agenda.

3.3 Alternatives

It is essential that before a country considers substituting alternatives, it ensures that the use is relevant to its national needs, and the anticipated local conditions of use. The hazards of the substitute materials and the controls needed for safe use should also be evaluated.

Canada

Fuel octane can be matched to engine compression ratios in several ways or combination of ways:

(i) more severe refining of petroleum fuels, converting low octane hydrocarbon molecules into differently structured but chemically similar molecules with higher octane values by reforming, cracking, alkylation and isomerization;
(ii) replacing alkyl lead additives (TEL and TML) in the fuel blend with other additives; and
(iii) using different fuels having more satisfactory antiknock and emissions characteristics than gasoline.

It was recognized that the health and environmental impacts of such substitutions had to be evaluated to avoid substituting one set of problems for another set. In its final report, the Royal Society of Canada recommended that methyl cyclopentadienyl manganese tricarbonyl (MMT)[3] and methyl tertiary butyl ether (MTBE)[4] be regarded as environmentally acceptable substitutes for alkyl lead additives as octane improvers in gasoline. The same applied to methanol and ethanol used as additives or blending components, though there are technical problems to overcome. If more severe reforming and isomerization were to be adopted by refiners as octane improvers, an increase in aromatic hydrocarbons was considered likely in gasoline. The Royal Society of Canada therefore recommended that the health effects of benzene in gasoline be examined, and that limits on the allowable concentration be established.

European Community

No information provided by the E.C. on alternatives.

International

The IOMC (1998) also reviews alternatives to lead in gasoline.

3.4 Socio-economic effects

Countries should consider the results of this information in the context of their own national conditions.

Canada

Canada first adopted regulations in 1973 to limit the concentration of lead in gasoline. Due to health and environmental concerns, the regulations were amended from time to time, with the intent of further reducing the lead content and exposure. In the end, the lead concentration has been decreased according to the following schedule:

- Commencing July 1, 1974, the maximum concentration of elemental lead in gasoline represented as lead-free was limited to 13 mg/l.
- Commencing January 1, 1976, the maximum concentration of lead in gasoline to which lead was added during the production process (leaded gasoline) was limited to 770 mg/l. This restriction did not apply to gasoline for use in aircraft.
- As of January 1, 1987, the maximum concentration of lead in gasoline to which lead was added during the production process (leaded gasoline) was limited to 290 mg/l. Again, this restriction did not apply

[3] In 1997, the Canadian Parliament acted to ban [the import and inter-provincial transport] of the gasoline additive MMT. Ethyl Corporation, the manufacturer of MMT, contested the ban with a lawsuit brought against the government. This action was made possible when Canada signed the North American Free Trade Agreement (NAFTA). In settlement, the government agreed to withdraw the ban.

[4] MTBE was subsequently placed on the First Priority Substances List. For this reason, a comprehensive assessment under the Canadian Environmental Protection Act was performed to determine the toxicity of the substance. The Assessment Report concluded that the predicted concentrations of MTBE in Canada do not constitute a danger to the environment or to the environment on which human life depends, or to human life or health.

to gasoline for use in aircraft.

- Since December 1, 1990, the average concentration of lead in leaded gasoline produced in Canada is specified at 26 mg/l, with a maximum concentration limit of 30 mg/l. The maximum concentration for leaded gasoline imported to Canada is limited to 26 mg/l. The utilization of leaded gasoline is restricted to specific uses. For all other purposes, the lead content in gasoline is limited to 5 mg/l. These restrictions do not apply to gasoline for use in aircraft. They also do not apply until December 31, 2002[5] to gasoline for use in high performance competition vehicles. These requirements were specified in the *Gasoline Regulations* (SOR/90-247) and subsequent modifications.

The reduction of lead in gasoline to 290 mg/l, effective January 1, 1987, was expected to have the following costs:

- increased refinery capital and operating costs due to increased processing requirements; and,
- decommissioning costs due to the closure and dismantling of lead-additives manufacturing plants.

Those costs were expected to range from $114 million to $452 million (in 1983 Canadian dollars), depending on the assumptions made.

Further, the reduced demand for lead additives was expected to cause a decline in the demand for primary (as opposed to secondary/recycled) refined lead. In 1981, lead additives manufacture accounted for about 6% of total Canadian primary refined lead production. It was forecasted that the production of primary refined lead accounted for by lead additives would decline to 1.5%. Closure of one of the two lead-additives manufacturing plant was therefore projected.

Impact upon employment was also anticipated. Loss of jobs in the lead-additives industry was expected to be greater than increased employment realized in the petroleum refining industry (158 VS 100).

The foreseen allocative benefits produced by the 290 mg/l limit consisted of a reduction of automotive lead emissions with a resulting reduction of the human lead burden. The emission reduction from 1987 to 2006 was estimated at 71 800 tonnes. No monetary value was assigned to human health.

The expected costs from the *Gasoline Regulations* (SOR/90-247) were capital and operating costs for refineries. The *Light Duty Vehicles* (LDV) *Regulations*, effective since 1987, made the use of unleaded fuel mandatory for light duty vehicles. This requirement had greatly accentuated the demand for unleaded gasoline. It was expected that the *Gasoline Regulations* (SOR/90-247) would further increase this demand. It was estimated that the petroleum refining industry had spent about $500 million (in 1984 Canadian dollars) to complete its modernization program to respond to the expected increase in demand for unleaded gasoline as a result of the LDV Regulations. To meet the increase from the *Gasoline Regulations*, it was estimated that the oil refining industry would incur incremental costs in the order of $100 million. Most of this amount was deemed to represent interest payments resulting from advancing the date of completion of refinery modernization programs by two to three years.

Industry estimated that accelerating the elimination of lead additives would increase refineries' cost of production by some $120 million annually as a result of using higher octane blending components and running octane generating processes at higher severity in refineries. The incremental operating costs were, however, expected to decrease as plant modernization and upgrading were completed, and more efficient state-of-the-art refining processes used.

It was estimated that after December 1, 1990, lead emissions from gasoline combustion would be reduced to about 12 tonnes per annum.

The amendment to exempt high performance competition vehicles until 2002 was expected to engender some costs. Sellers and importers would have to assume certain costs associated with keeping records. These were estimated to be $12,000 per annum (in 1994 Canadian dollars). It was also believed that vehicle owners had to assume costs associated with obtaining a letter of certification. This cost was determined at $8,000 total (in 1994 Canadian dollars). This requirement to obtain a letter of certification was later removed.

On the other hand, the exemption was also expected to generate benefits to the industry. Some anticipated benefits were in terms of local economic spin-offs resulting from continuance of significant racing events in Canada. It was estimated that racing events in Canada in 1996 generated about $44 million (in 1996 Canadian dollars) in direct sales of tickets and fuel. From these direct revenues, it was estimated that the racing sector would engender indirect economic activity between $88 million and $110 million (in 1996 Canadian dollars) each year. The extension of the time limit to 2002 was expected to remove regulatory

[5] The exemption for high performance competition vehicles has recently been extended until January 1, 2008. Reference: *Regulations Amending the Gasoline Regulations* (SOR/2003-106)

uncertainty. Overall, the economic impacts estimated by race-sanctioning bodies were an increase of about $2.5 million in direct revenues, $5 million in indirect revenues and 90 jobs.

The time limit for the applications of the amendment was expected to give producers of gasoline the time to develop acceptable unleaded gasoline for use in competition vehicles.

European Community

No detailed assessment of socio-economic effects was undertaken.

4. Hazards and Risks to human health and the environment

4.1 Hazard Classification

IARC	Not classifiable as a human carcinogen (Group 3)	
European Community (in accordance with Directive 67/548/EEC)	T+(very toxic)	
	N (dangerous for the environment)	
	R61	May cause harm to the unborn child
	R26/27/28	Very toxic by inhalation, in contact with skin and if swallowed
	R33	Danger of cumulative effects
	R50/53	Very toxic to aquatic organisms, may cause long-term adverse effect in the aquatic environment
	R62	Possible risk of impaired fertility

4.2 Exposure limits

WHO blood lead levels (since 1980): 20µg/dl

4.3 Packaging and labelling

The United Nations Committee of Experts on the Transportation of Dangerous Goods classifies the chemicals in:

Hazard Class and Packing Group for TEL:	T+ Symbol R: 26/27/28-33 S: 13-26-36/37-45 UN Hazard Class: 6.1 UN Pack Group: I
Hazard Class and Packing Group for TML:	T+ Symbol R: 61-26/27/28-33 S: 53-45 UN Hazard Class: 6.1 UN Pack Group: I
International Maritime Dangerous Goods (IMDG) Code	IMO 6.1
Transport Emergency Card	TEC (R)-157

4.4 First aid

The following advice is based on information available at the time of publication. This advice is provided for information only and is not intended to supersede any national first aid protocols.

In all cases consult a physician (a person licensed to practice medicine).
Inhalation: fresh air, rest. Artificial respiration if indicated.
Skin: remove contaminated clothes. Rinse and then wash skin with water and soap.
Eyes: first rinse with plenty of water for several minutes (remove contact lenses if easily possible).
Ingestion: rinse mouth. Induce vomiting (only in conscious persons). Give plenty of water to drink.
Further information may be found on the website of the IPCS (International Chemical Safety Cards) at www.inchem.org/pages/icsc.html

4.5 Waste management

Not applicable.

Annexes

Introductory text to Annex I

The information presented in this Annex reflects the conclusions of the notifying Parties, Canada and the European Community.

Information provided by these Parties on the hazards and risks are synthesised and presented together. This information is based on documents referenced in the notifications in support of their final regulatory actions, and includes international reviews.

The notification from Canada was first reported in PIC Circular XII of December 2000 and the notification from the European Community in PIC Circular XVI of December 2002.

TEL and TML are released into the environment primarily through evaporative emissions from unburned gasoline retained in carburettors or fuel tanks, losses during filling operations, accidental spillage or during production. However, due to the combustion process, lead emitted from the exhaust of vehicles is in the form of inorganic Pb (salts) or is readily transformed to inorganic lead.

As a consequence, restrictions and bans of leaded gasoline have been based on the harmful effects of inorganic lead rather than alkyl lead. To this end, much of the data contained in this annex focuses on properties and effects of lead rather than those of TEL and TML.

Annex 1 – Further information on tetraethyl lead and tetramethyl lead

1 Physico-Chemical properties

1.1	**Identity**	Tetraethyl lead	Tetramethyl lead
1.2	**Formula**	$Pb(C_2H_5)_4$	$Pb(CH_3)_4$
1.3	**Colour and Texture**	colourless viscous liquid, with characteristic odour. Commercial mixtures may be dyed red, orange or blue	colourless viscous liquid, with characteristic odour.
1.4	**Melting point**	-136.8 ^oC	-27.5 ^oC
1.5	**Boiling point**	200 ^oC (decomposes)	110 ^oC (at 1.33 kPa)
1.6	**Relative density (water = 1)**	1.7	2.0
1.7	**Vapour pressure**	0.027 kPa at 20 ^oC	3.0 kPa at 20 ^oC
1.8	**Flash point**	77 ^oC	38 ^oC (open cup)
1.9	**Explosive limits (vol% in air)**	1.8	

2 Toxicological properties

2.1	**General**	One significant source of human exposure to lead has been through inorganic lead compounds emitted from the combustion process as a direct result of the use of alkyl lead as an additive in gasoline. Lead emitted from the exhaust of vehicles is primarily in the form of inorganic particles (e.g. PbBrCl), with only small amounts (less than 10% of total emissions) in the form of organolead vapours (Royal Society of Canada, September 1986). The discussion on toxicological properties therefore focuses on the risks to human health associated with exposure to lead, tetraethyl lead and tetramethyl lead.
2.1.1	**Symptoms of poisoning**	The IPCS (1991) reports that poisoning by organic lead compounds presents mainly acute effects on the central nervous system. Poisoning may result from the absorption of a sufficient quantity of lead, whether briefly at a high rate or for prolonged periods at a lower rate. Mild manifestations are: insomnia and nervous excitation, nausea, vomiting, associated with tremor, hyperreflexia, muscular contractions, bradycardia, arterial hypertension, and hypothermia. Most severe cases present episodes of complete disorientation, mania, ataxia, hallucinations, exaggerated muscular activity, and violent convulsive seizures, which may terminate in coma and death.

2.1.2 Absorption, distribution, excretion and metabolism in humans

Absorption: Accidental or deliberate ingestion of alkyl lead compounds may occur, but is not frequent. Inhalation of vapours of alkyl lead compounds should be considered as a major route of entry. Dermal absorption is an efficient route of entry for organic compounds of lead (IPCS, 1991).

According to the Royal Society of Canada (September 1986), the absorption of lead by the human body depends on many factors. Children tend to absorb and retain more than adults, particularly through the intestinal tract. There is also evidence that the sexes may differ as regards absorption of lead. Finally, both nutritional status and diet play a role in the absorption and toxicity of lead.

The WHO (1995) further indicates that depending upon chemical speciation, particle size, and solubility in body fluids, up to 50% of the inhaled lead compound may be absorbed. Some inhaled particulate matter (larger than 7µm) is swallowed following mucociliary clearance from the respiratory tract. In experimental animals and humans, absorption of lead from the gastrointestinal tract is influenced by the physico-chemical nature of the ingested material, nutritional status, and type of diet consumed. In adult humans approximately 10% of the dietary lead is absorbed; the proportion is higher in fasting conditions. However, in infants and young children as much as 50% of dietary lead is absorbed, although absorption rates for lead from dust/soils and paint chips can be lower depending upon the bioavailability. Diets that are deficient in calcium, phosphate, selenium or zinc may result in increased lead absorption. Iron and vitamin D also affect absorption of lead.

Blood lead (PbB) levels are used as a measure of body burden and absorbed (internal) doses of lead. The relationship between blood lead and the concentration of lead in exposure sources is curvilinear (WHO, 1995).

Distribution: Lead is distributed in man according to a three-compartment pharmacokinetic model. Once it has been absorbed, lead is not distributed homogeneously throughout the body (IPCS, 1991). There is rapid uptake into blood and soft tissue, followed by a slower redistribution to bone (WHO, 1995). Blood and soft tissues represent the active pool and bones the storage pool. Lead is distributed to kidney tubular epithelium and to liver. There is redistribution by deposition in bone, teeth and hair. The long bones contain more lead and about 95% of the body load is stored in the skeleton. The largest part of circulating lead is bound to haemoglobin in erythrocytes, in which the concentration of lead is about 16 times greater than in plasma (IPCS, 1991).

In view of the extremely long half-life for lead in bone, this compartment can serve as an endogenous source of lead to other compartments long after exposure ceases (WHO, 1995). This lead can be mobilized from bone during pregnancy. Lead also transfers readily from the mother to the foetus via the placenta (umbilical cord blood lead levels are typically very close to maternal blood lead levels). Thus, lead exposure in a pregnant woman’s history, even in her own childhood, may affect her children (IOMC, 1998).

Excretion: The WHO (1977) reports that elimination of lead from the body is mainly by way of urine (about 76%) and the gastrointestinal tract (about 16%). The other 8% is excreted by miscellaneous routes (sweat, exfoliation of the skin, loss of hair) about which little is known. Losses per day are as follows:

- urine: 38µg
- gastrointestinal: 8µg
- hair, nails, sweat, other: 4 µg

The amount excreted through any route is affected by age and exposure characteristics and is species dependent (WHO, 1995).

Metabolism: Alkyl lead compounds are transformed to trialkyl derivatives by dealkylation in the liver. TEL and TML are not the primary toxic substances but they are converted to other lead compounds (IPCS, 1991). TEL is initially converted mainly to triethyl lead and partly to inorganic lead. The triethyl lead concentration in organs then falls only slowly. Even after several days, there is no significant reduction. The behaviour of TML is quite similar to the behaviour of TEL. TML is much less toxic probably because it is dealkylated to the trialkyl toxic form much more slowly than is the case with TEL. Details of alkyl lead metabolism have been learned from animal studies and have not been defined in man (WHO, 1977).

2.2 Toxicity

2.2.1 Health effects to humans of lead exposure

It is well established that lead is a highly toxic chemical with no known physiological benefit (IOMC, 1998). Lead adversely affects several organs and organ systems, with subcellular changes and neurodevelopmental effects appearing to be the most sensitive (OECD, 1993). Its effects depend on many factors, some genetic (such as individual or racial susceptibility), some acquired (nutritional status, socio-economic situation) and some depending on the chemical form of the lead (organic or inorganic) and the quantity and duration of exposure (Royal Society of Canada, September 1986).

The WHO (1995) reports that effects at the subcellular level, as well as effects on the overall functioning of the body, have been noted and range from inhibition of enzymes to the production of marked morphological changes and death. Such changes occur over a broad range of doses, the developing human generally being more sensitive than the adult.

Lead has been shown to have effects on many biochemical processes; in particular, effects on haem synthesis have been studied extensively in both adults and children. Increased levels of serum erythrocyte protoporphyrin and increased urinary excretion of coproporphyrin and delta-aminolaevulinic acid are observed when PbB concentrations are elevated. Inhibition of the enzymes delta-aminolaevulinic acid dehydratase and dihydrobiopterin reductase are observed at lower levels.
The effects of lead on the haemopoietic system result in decreased haemoglobin synthesis, and anaemia has been observed in children at PbB concentrations above 1.92 µmol/l (40 µg/dl).

For neurological, metabolic and behavioural reasons, children are more vulnerable to the effects of lead than adults. Both prospective and cross-sectional epidemiological studies have been conducted to assess the extent to which environmental lead exposure affects CNS-based psychological functions. Lead has been shown to be associated with impaired neurobehavioural functioning in children.

Impairment of psychological and neurobehavioural functions has been found after long-term lead exposure of workers. Electrophysiological parameters have been shown to be useful indicators of subclinical lead effects in the CNS.

Peripheral neuropathy has long been known to be caused by long-term high-level lead exposure at the workplace. Slowing of nerve conduction velocity has been found at lower levels. These effects have often been found to be reversible after cessation of exposure, depending on the age and duration of exposure.

The effect of lead on the heart is indirect and occurs via the autonomic nervous system; it has no direct effect on the myocardium. The collective evidence from population studies in adults indicates very weak associations between PbB concentration and systolic or diastolic blood pressure. Given the difficulties of allowing for relevant confounding factors, a causal relationship cannot be established from these studies.

Lead is known to cause proximal renal tubular damage, characterized by generalized aminoaciduria, hypophosphataemia with relative hyperphosphaturia and glycosuria accompanied by nuclear inclusion bodies, mitochondrial changes and cytomegaly of the proximal tubular epithelial cells. Tubular effects are noted after relatively short-term exposures and are generally reversible, whereas sclerotic changes and interstitial fibrosis, resulting in decreased kidney function and possible renal failure, require chronic exposure to high lead levels. Increased risk from nephropathy was noted in workers with a PbB level of over 3.0 µmol/l (about 60 µg/dl). Renal effects have recently been seen among the general population when more sensitive indicators of function were measured.

The reproductive effects of lead in the male are limited to sperm morphology and sperm count. In the female, some adverse pregnancy outcomes have been attributed to lead. Some but not all epidemiological studies show a dose-dependent association of pre-term delivery and some indices of fetal growth and maturation at PbB levels of 0.72 µmol/l (15 µg/dl) or more (OECD, 1993).

The evidence for carcinogenicity of lead and several inorganic lead compounds in humans is inadequate (OECD, 1993). In rats, renal tumours were seen at dietary levels of 500 mg lead/kg, which were associated with blood lead levels of 80µg/dl. No tumours were seen at dietary levels of 200 mg lead/kg or below (WHO, 1995).

According to the WHO (1995) lead does not appear to have deleterious effects on skin, muscle or the immune system.

2.2.2 Relationship between exposure and dose

The most widely used surrogate for the absorbed dose is whole PbB concentration.

The WHO (1995) reports that the relationship between PbB level and lead intake is curvilinear over a wide range of PbB values. On the basis of a single study of 17 infants, the relationship between PbB level and lead intake from food has been determined to be 0.0077 µmol lead/l (0.16 µg/dl) per µg lead intake per day for a median PbB level of approximately 0.48 µmol/l (10 µg/dl).

Most studies of the relationship between PbB level and lead exposure apply to a single environmental source, i.e. air, food, water or soil/dust. A summary of the relationship between PbB median level and lead intake from individual media is given below:

Medium	Children[a]	Adults[a]
Air[b]	0.09 µmol Pb/l per µg Pb/m^3 (1.92 µg Pb/dl)	0.079 µmol Pb/l per µg Pb/m^3 [c] (1.64 µg Pb/dl)
Water		0.003 µmol Pb/l per µg Pb/l (0.06 µg Pb/dl)
Food[b]	0.01 µmol Pb/l per µg Pb/day (0.16 µg Pb/dl)	0.002-0.003 µmol Pb/l per µg Pb/day (0.04-0.06 µg Pb/dl)
Dust[b]	0.09 µmol Pb/l per 1000 µg Pb/g (1.8 µg Pb/dl)	
Soil[b]	0.11 µmol Pb/l per 1000 µg Pb/g (2.2 µg Pb/dl)	

[a] These data are provided for illustrative purposes only recognizing that the relationships are curvilinear in nature and are broad guidelines which will not apply at lower or higher levels of exposure.
[b] A value between 0.144 to 0.24 µmol Pb/l or 3-5 µg Pb/dl per µg/m^3 is obtained when one considers indirect contribution through deposition on soil/dust.
[c] The air to blood lead relationship in occupational settings is best described by a curvilinear relationship having slopes between 0.02 and 0.08 µg/m^3 air. The slope is variable but lower than that found for humans in the general environment, which is between 1.6 and 1.9 µg/m^3.

2.2.3 Relationship between dose and effect

Toxic Effects in Adults (IOMC, 1998)	Blood lead level
Nervous system: overt clinical encephalopathy	100-120 µg/dl
Kidney: atrophy and interstitial nephritis	40-100 µg/dl
Gastrointestinal: colic	40-60 µg/dl
Anemia	50 µg/dl
Reproductive system: hypospermia, testicular atrophy	40-50 µg/dl
Nervous system: IQ/learning disruption, sensory system deficit	40 µg/dl
Heart and blood vessels: hypertension	< 7 µg/dl
Biochemical (enzyme changes)	3-30 µg/dl

Toxic Effects in Children (IOMC, 1998)	Blood lead level
Kidney: atrophy and interstitial nephritis	80-120 µg/dl
Nervous system: overt clinical encephalopathy	80-100 µg/dl
Gastrointenstinal: colic	60-100 µg/dl
Anemia	20-40 µg/dl
Biochemical (enzyme) changes	< 10 µg/dl
Nervous system: IQ/learning disruption	< 10 µg/dl

2.2.4 Summary of mammalian toxicity and overall evaluation

Lead toxicity to animals, including mammals, has been extensively studied. According to the WHO (1995) review, in all species of experimental animals studied, including non-human primates, lead has been shown to cause adverse effects in several organs and organ systems, including the haematopoietic, nervous, renal, cardiovascular, reproductive and immune systems. Lead also affects bone and has been shown to be carcinogenic in rats and mice.

2.2.5 Alkyl lead - Acute toxicity

2.2.5.1 Ingestion

Acute poisoning from ingestion is rare. In one case of massive ingestion of pure tetraethyl lead the initial signs and symptoms were related to increased intracranial pressure. The patient died 36 hours later with pulmonary oedema (IPCS, 1991).

2.2.5.2 Inhalation

Inhalation induces sneezing, irritation of the upper respiratory tract and mild to severe systemic responses: insomnia, lassitude, nervous excitation, anxiety states, associated with tremors, hyperreflexia, spasmodic muscular contractions, bradycardia, vascular hypotension, and hypothermia. The most severe responses include complete disorientation with hallucinations, and facial contortions. Such episodes may progress to maniacal and violent convulsive seizures which may terminate in coma and death (IPCS, 1991).

2.2.5.3 Skin exposure

In contact with the skin, alkyl lead compounds induce itching, burning and transient redness. In one case of massive skin exposure, a patient remained asymptomatic even though the urinary lead excretion was very high (IPCS, 1991).

2.2.5.4 Eye contact

In contact with the ocular membranes, organolead compounds induce itching, burning, and transient redness (IPCS, 1991).

2.2.6 Alkyl lead - Chronic toxicity

2.2.6.1 Inhalation

No chronic form has been observed in a population exposed occupationally. Chronic recreational sniffing of leaded gasoline as a drug of abuse has led to neurological damage: tremors, exaggerated tendon reflexes, severe encephalopathy, and death (IPCS, 1991).

2.2.7 Alkyl lead - Cause of death

Cause of death is direct damage to the brain (encephalopathy) involving capillary dysfunction, cerebral oedema, and interference with cerebral metabolism. In one case, pulmonary oedema was described as the terminal event. (IPCS, 1991)

2.2.8 TEL – Relevant animal data

Lowest toxic dose oral mouse: 11 mg/kg
LD_{50} oral rat: 1.2 mg/kg
LC_{50} inhalation rat: 850 mg/m^3
Lowest lethal dose skin dog: 547 mg/kg
(IPCS, 1991)

2.2.9 TML – Relevant animal data

Lowest toxic dose oral rat: 112 mg/kg
LD_{50} oral rat: 105 mg/kg
LC_{50} inhalation rat: 8,870 mg/m^3
Lowest lethal dose oral rabbit: 24 mg/kg
Lowest lethal dose skin rabbit: 3391 mg/kg
IPCS (1991)

3 Human exposure/Risk evaluation

3.1 Introduction

There are many possible sources of exposure to lead. Lead is ubiquitous in the global ecosystem, as well as occurring naturally (WHO, 1995). Lead has been widely used in industrialization. Some examples are its use in paints, in plumbing, in batteries and in consumer packaging. It is also often released into the environment through industrial operations involving lead, such as mining, smelting, and producing or recycling lead containing products. One significant source of lead exposure has been through its use as an additive in gasoline.

The particular uses of lead that are most significant from a toxicological, public health perspective can thus vary widely among countries and among populations within those countries. Leaded gasoline is clearly not the only exposure source in many countries (IOMC, 1998). Whereas in specific areas point sources may contribute significant amounts of lead to the environment, on a global scale, the combustion of alkyl lead in gasoline is the predominant source of increased lead in all compartments of the environment (WHO, 1995).

Exposure to lead from gasoline additives is unique in several ways. It is more widely dispersed geographically than industrial sources, which tend primarily to affect very local areas. Use of leaded gasoline contributes to airborne lead in respirable form (this is also true of industrial facilities). Unlike many other sources of soil and dust contamination, lead emitted from gasoline combustion occurs as extremely small particles, which stay suspended in the air for longer periods and inhaled more deeply into the lungs, and carry a greater contaminant load (on a mass basis) than do larger particles (IOMC, 1998). Primary vehicle exhaust lead comprises particles around 0.015 µm, and may be attached to particles of carbon of similar dimensions. Particles of this size very rapidly combine with other particles. When they have grown to around 0.1 to 1.0 µm, they cease to grow and can have an atmospheric lifetime of around seven to 24 days (OECD, 1993).

Over 70% of the lead in gasoline is likely to enter the environment immediately after combustion, the rest being trapped in the crank case oil and in the exhaust system of vehicles (WHO, 1977). The alkyl lead additive in motor gasoline is almost completely combusted in the engine cylinder. Therefore, automobile lead emissions are primarily in the form of lead bromochloride, with only small amounts (less than 10% of total emissions) in the form of organolead vapours (OECD, 1993; Royal Society of Canada, September 1986).

3.2 General population exposure

The WHO (1995)[6] reports that in the absence of specific stationary sources of lead, concentrations in ambient air are directly related to density of traffic and whether lead is still utilized as an additive in gasoline. Reduction or elimination of lead in gasoline in those countries which have instituted regulations has resulted in a decline by as much as eight-fold in ambient air concentrations of lead.

Levels of lead in indoor air are affected by the presence of cigarette smoke and dust from lead-painted surfaces. Without such sources, air lead levels indoors are about 60% of those in outdoor air.

For most adults, the total daily exposure to lead is via food, water and air. For infants aged up to 5 months, formula or breast milk and water are the main sources of lead. In children, an additional source of exposure is dust and soils. Absorption is dependent on the chemical form of lead, type of soil and particle size (bioavailability). Lead intake may be augmented from unusual sources such as folk remedies, cosmetics and hobby activity. Community contamination and workplace practices may contribute to lead exposure.

Food (including drinking-water and beverages) is the major source of lead exposure for the general population. Infants and children may receive an added lead burden from soil and dust. The most significant foodstuffs will vary from country to country. In areas still utilizing lead-soldered cans, levels of lead are substantially higher. Depending upon lifestyles, there may be significant oral intake of lead from some alcoholic beverages and due to the leaching of lead from low temperature-fired ceramic containers.

Most drinking-water supplies contain lead levels lower than 5 µg/l when they leave the treatment plant. However, where the water is known to be plumbo-solvent, up to 40% of the samples may exceed 100 µg/l in homes where lead solder, lead pipes or brass fixtures have been used.

Absorption of lead from the lung is a function of particle size and pulmonary deposition pattern. Small particles (< 0.5 µm in diameter) characteristic of ambient air will be deposited deeply in the lungs with absorption rates of 90%. Larger particles, such as those that may be encountered in occupational settings, exhibit high deposition rates in the upper airway. Absorption of such particles will be a function of both dissolution in the lung and particle clearance to the gastrointestinal tract.

Human dermal absorption of inorganic lead through unabraded skin is of limited significance.

[6] Entire section from the WHO 1995.

3.3 Food

The concentration of lead in various items of food is best described as highly variable. In fact, there seems to be about as much variation within specific items of food as between different categories of foods. The WHO (1977) reports that the range of lead was 0-1.5 mg/kg for condiments, 0.2-2.5 mg/kg for fish and seafood, 0-0.37 mg/kg for meat and eggs, 0-1.39 mg/kg for grains, and 0-1.3 mg/kg for vegetables.

Although plants do not take lead up from the soil readily, fruits and vegetables grown in areas exposed to smelter emissions may be appreciably contaminated. Kerin (1972) determined lead in the total diet of peasants near a smelter and found that the daily ingestion of lead with food was 670-2640 µg (WHO, 1977).

3.4 Air

Ambient air can be a major pathway of lead distribution in the environment (WHO, 1995). Processes that release lead into the air include mining and smelting, incineration, gasoline burning, battery manufacturing and sand-blasted or flaking paint. In addition, soils may be a source of airborne lead on a local scale, as suggested by the strong correlation between lead in the dust and lead in soil. The re-entrainment of deposited lead in dust is, however, unlikely to be an important net source to the atmosphere, because large particle size limits dispersal (Royal Society of Canada, September 1986).

Concentrations of lead in air range from 7.6×10^{-5} µg/m^3 in remote areas such as Antartica to > 10 µg/m^3 near lead smelters (WHO, 1995). The highest concentrations of lead in ambient air are found in dense population centres. The larger the city, the higher the ambient air lead concentration. As one moves away from the centre of the city, the concentration falls progressively. For urban stations, an average concentration of 1.1 µg/m^3 has been reported; for non-urban stations (near the city) the average was 0.21 µg/m^3; for stations somewhat farther removed it was 0.10 µg/m^3, and for remote areas, 0.02 µg/m^3. Air over streets with heavy traffic contained more lead than air over streets with light traffic, and considerably more than the ambient air over rural areas.

There is a clear pattern in this picture, the non-urban sites showing less than 0.5 µg/m^3, while the urban sites have values ranging from 1 to 5-10 µg/m^3. The highest levels have been recorded on highways during rush hours, 14-25 µg/m^3 (WHO 1977).

Concentrations of lead in urban air in various OECD countries in the 1970s ranged from 0.5 µg/m^3 up to 10 µg/m^3 in densely trafficked inner cities. The air lead levels found in European and North American cities (circa 1993) are in the range of 0.2-0.8µg/m^3, and in rural areas usually in the range of 0.05-0.3µg/m^3 (OECD, 1993).

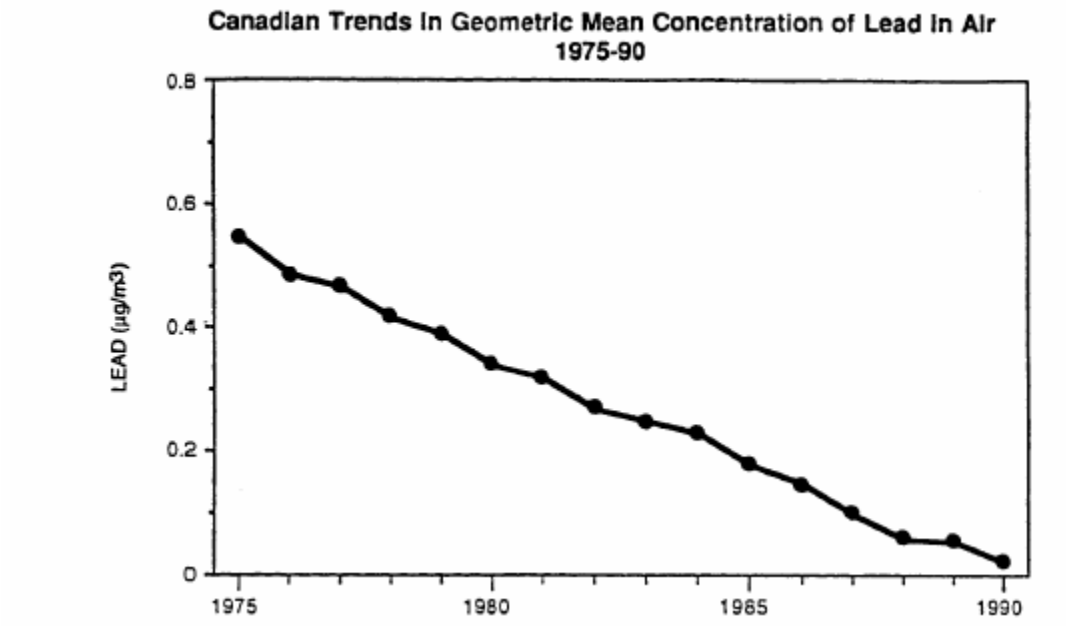
The OECD (1993) reports that in Canada, the average levels of lead in air have steadily declined over the last few decades (Figure 1). The decline has been largely attributed to reductions in the use of lead in gasoline, as lead emissions from other sources have remained relatively constant (Figure 2). It should be noted that lead emissions from the lead industry were relatively constant during the 1980s, as the majority of reductions occurred when federal and provincial controls were introduced prior to 1978. The reduction of lead in air since 1987 is, in part, attributed to a decline in lead emissions from gasoline combustion and copper-nickel production.

The average PbB levels for the Canadian population at greatest risk from exposure (i.e. children) also declined to about 6 µg/dl in 1988, below the 10µg/dl level of concern (Figure 3). Although there is a strong correlation between PbB levels in children and PbA levels, it has been suggested that the decline may also be related to other factors such as a voluntary industry phase-out of lead soldered food cans and a decline in the use of lead in indoor paints.

The OECD (1993) reports that in several Member States of the E.C. where blood analyses were carried out in the 70s and the 80s, it was shown that the decreases in air concentration due to restrictions on leaded fuel was related to the diminution in blood lead levels. For example:

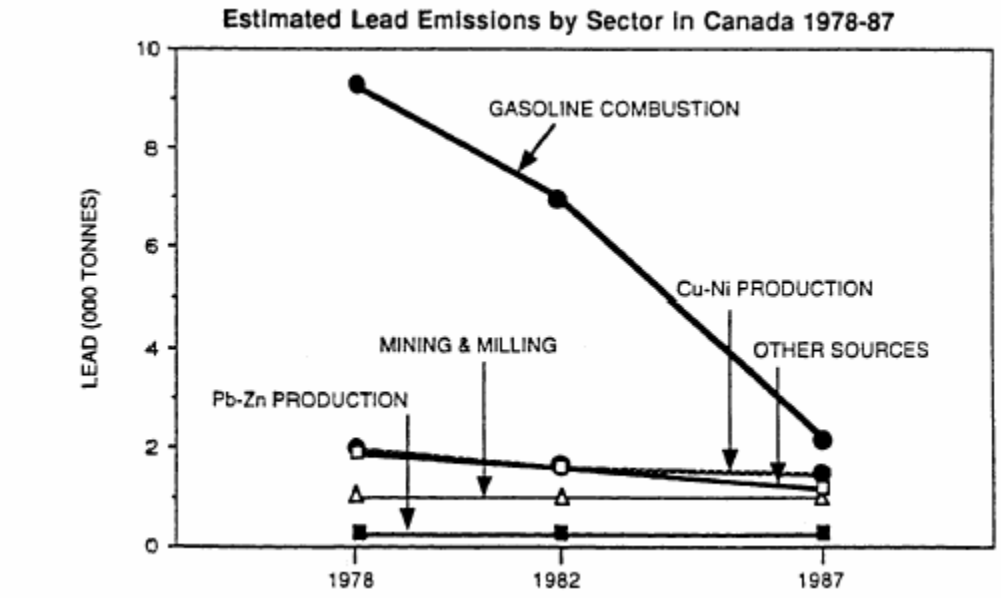
- In Belgium, the average PbB levels for various segments of the population steadily declined during the 1980s. The decline has been, in part, attributed to the reduction in the permissible level of lead in gasoline.
- In Finland, average PbB levels decreased from 11 µg/dl to 2.8 µg/dl in the period 1975 to 1992. In Helsinki, the average PbB level of children decreased from 4.6 µg/dl to 3.0 µg/dl between 1983 and 1988; in the same period, car exhaust emissions of lead decreased by 75% in Helsinki.
- In Germany, various studies indicate that average PbB levels in school children and adults declined since 1975. The reductions are believed to be a result of lead in air, attributed to the phasing out of lead in gasoline.

Figure 1



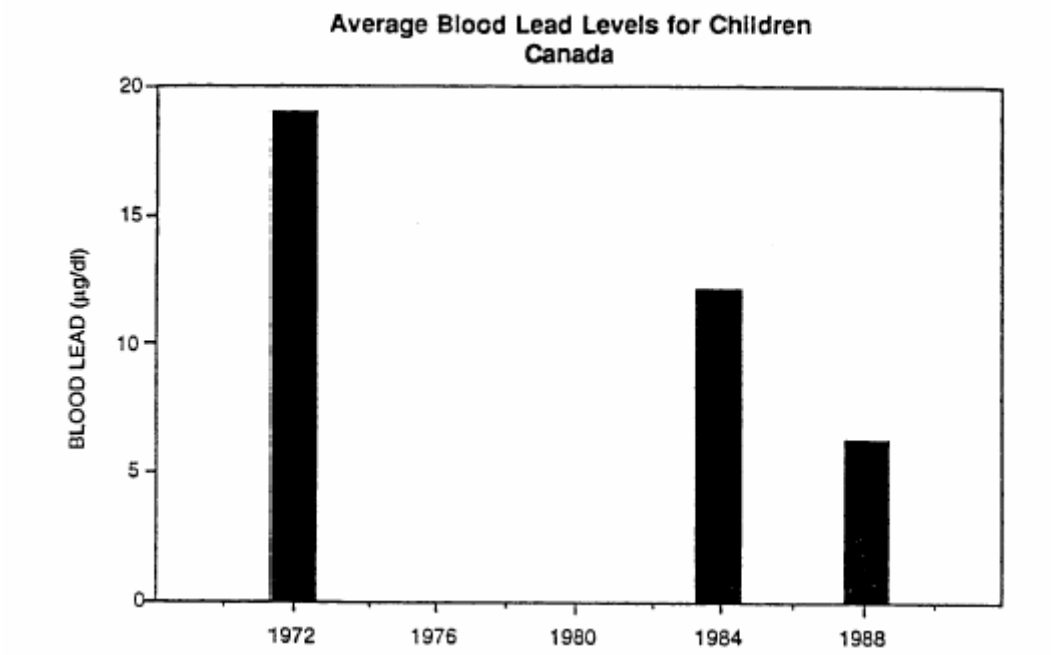
Source: OECD, 1993

Figure 2



Source: OECD, 1993

Figure 3



Source: OECD, 1993

- In Sweden, average PbB levels in children living near smelters or in rural or urban environments have been declining since 1978, to below 5µg/dl in 1988. Average PbB levels for the general population in Stockholm also fell to below 6µg/dl by 1984. The reduction in the permissible level of lead in gasoline was considered as one of the important factors contributing to the decline in PbB levels.
- In the United Kingdom, the average PbB levels for children, as well as adult females and males, also declined steadily during the mid-1980s, with the average levels being well below the 25 µg/dl level of concern. According to the UK Department of the Environment, the reduction in the permissible level of lead in gasoline during 1985 appeared to contribute slightly to the decrease of lead in the body burden of children.

According to the IOMC (1998), numerous studies document a relationship between the use of lead in gasoline and the concentration of lead in air. Airborne lead concentrations were generally higher in urban areas, in countries as diverse as Hungary, Mexico and Thailand. Further, strong linear correlations have been found with traffic density within urban areas, whether in Saudi Arabia or India. Finally nations that have curtailed the lead content of their gasoline have been able to track corresponding declines in the concentration of lead in urban air.

The IOMC (1998) reports that more than one quarter of newborns in Mexico City had blood lead levels high enough to impair neurological development while in Bangkok, between 30,000 and 70,000 children risk the loss of four or more IQ points due to high lead levels. Blood lead of children in Budapest were three times higher downtown than in suburbs. In an analysis of available data worldwide, the percentage of children with PbB above 10µg/dl was found to be much higher in urban than in non-urban areas. The highest proportion in non-urban children (under age 12) was 19 percent above the standard. For urban areas, the lowest exceedance was 55 percent.

3.5 Water

Water may be contaminated following the deposition of atmospheric lead, the concentration of which is increased by the combustion of leaded fuels. However, according to the WHO (1977), man's exposure to lead through water is generally low in comparison with exposure through air and food. The lead concentration in the water supplies of most of the 100 largest American cities, as determined in 1962, ranged from a trace to 62 µg/l. Since 1962, continuous monitoring of American water supplies has indicated that the US Public Health Service prescribed limit of 50 µg/l has not been exceeded. In another study, only 41 out of 2595 samples of tap water contained more than 50 µg/l, and 25% contained no measurable amount of lead.

Lead levels in surface and ground waters were reviewed by a WHO Working Group (1973). Natural surface waters have been reported to contain usually less than 0.1 mg/l. In unpolluted areas the concentrations are of the order of 1 µg/l or less. Some rivers in France were analyzed and it was found that, in the Midi-Pyrenees region, the mean concentration of dissolved lead varied from 6.7 to 10.4 µg/l.

3.6 Dust

According to the WHO (1995), dust is a significant source of exposure to lead, particularly for young children, as has been demonstrated in several studies correlating children's blood lead concentrations with dust lead levels.

The major contributions to lead levels in soil and outdoor dust are from the combustion of fossil fuels (principally leaded gasoline), stationary sources such as smelters, and peeling and flaking of lead-based paint. Typical lead levels in road dust in the USA are 800-1300 mg/kg in rural areas to 100-5000 mg/kg in urban areas.

Concentrations of lead in household dust vary greatly between different dwellings and areas of the world. Mean concentrations of 300-2500 mg/kg have been found in the United Kingdom and USA, but individual samples may be in the range of 10 000 to 30 000 mg/kg.

3.7 Soil

According to the WHO (1995), in rural and remote areas, lead in soil is derived mainly from natural geological sources. These natural sources account for 1-30 mg lead/kg, but where soils are derived from lead mineralized rocks, natural concentrations may range from several hundred to several thousand mg/kg.

Concentrations of lead in urban soil vary greatly. In the USA, a study of city parks recorded concentrations of 200 to 3300 mg/kg. Concentrations of up to 10 960 mg/kg have been reported for urban garden soils in the USA, and up to 14 100 mg/kg in the United Kingdom. Concentrations can exceed 20 000 mg/kg around lead mining and processing operations. In areas where lead-based paint has been used, soil samples taken near building foundations have been reported to be as high as 20 000 mg/kg.

In general, lead concentrations in soils near roads are high where road traffic density is high. Concentrations decrease exponentially with distance from the road.

4 Environmental fate and effects

4.1 Transportation and distribution between media

4.1.1 Atmospheric deposition

From the mass balance point of view the transport and distribution of lead from major emission sources, both fixed and mobile, is mainly atmospheric. Most of the lead discharged to the atmosphere is deposited near the source. However, approximately 20% is widely dispersed and contaminates areas as remote as glacial strata in Greenland. The extent of long-range transport of lead particles is dependent upon particle size, particles > 2 µm in diameter being deposited close to the source of emission. Between 20 and 60% of emissions from vehicles has been reported to remain within 25 m of the roadway. However, in view of the marked decrease in the concentration of lead in cores of ice from Greenland since the decreased use of leaded gasoline, it is apparent that vehicle emissions can contribute to the levels of lead in air far from the source. Long-range transport of lead particles was also observed.

Lead can be removed from the atmosphere and transferred to environmental surfaces and compartments by wet or dry deposition. Wet deposition appears to be more important than dry deposition for the removal of atmospheric lead. Depending upon geographical location and the level of emissions in the area, between 40 and 70% of atmospheric lead is removed by wet deposition. In most cases it is poorly soluble and either precipitates out in soils and sediments or is bound to organic matter in these compartments. For these reasons lead is not readily removed and tends to accumulate in those ecosystems where it is deposited. The ratio of wet to dry deposition was calculated to be 1.63, 1.99 and 2.50 for sites in south, central, and northern Canada, respectively, while wet deposition accounted for 80% of the total lead deposited in a semi-remote site in the USA.

Making several assumptions regarding global atmospheric lead concentrations, wind speed, surface area and texture, a global deposition of approximately 410 000 tonnes/year (combined wet and dry) was calculated by the US EPA (1986).

4.1.2 Transport to water and soil

According to the WHO (1995), when deposited in water, whether from air or through run-off from soil, lead partitions rapidly between the sediment and aqueous phase, depending upon the salt content of the water as well as the presence of organic complexing agents. For example, at pH > 5.4 the total solubility of lead is about 30 µg/l in hard water and 500 µg/l in soft water. In addition, the presence of sulfate and carbonate ions can limit lead solubility.

Water-borne lead has been found to exist as soluble lead or undissolved colloidal particles, either suspended in the aqueous phase or carried as surface coatings on other suspended solids. The ratio of lead in suspended solids to lead in the dissolved form has been found to vary from 4:1 in rural areas to 27:1 in urban streams.

Both natural organic compounds (humic and fulvic acids) as well as those of anthropogenic origin (e.g., ethylenediamino-tetraacetic and nitrilotriacetic acids) may complex lead found in surface waters. The presence in water of such chelators can increase the rate of solution of lead compounds (e.g., lead sulfide) 10 to 60 times over that of water at the same pH without fulvates.

Lead accumulation in soils is primarily a function of the rate of wet and dry deposition from the atmosphere. Transport within soil and the bioavailability of lead from soil are dependent upon many factors, including pH, mineral composition of the soil, and amount and type of organic material, with most of the lead being bound within the upper 5 cm of soil. This limits the amount which can be leached into water or be available for uptake into plants. It has been shown that only 0.2% of the total lead in soil can be released into solution by shaking. However, the release of lead from organic complexes to the soluble, and thus bioavailable, form is highly pH dependent. Within the usual pH range for soils (4 to 6), the organic-lead complexes become more soluble and the lead more available for plant uptake and leaching into water.

4.2 Environmental transformation

4.2.1 Abiotic transformation

The WHO (1995) reports that, once released into the environment, lead may be transformed from one inorganic species or particle size to another. However, as an element it is not subject to degradation. For example, lead-containing particles in automobile exhaust are usually lead halides or double salts with ammonium halides. Within 24 h, over 75% of lead particulate matter is transformed to lead carbonates and sulfates.

4.2.2 Biotransformation

The WHO (1995) reports that the transformation of inorganic lead to tetramethyl lead (TML) has been observed in aquatic systems, particularly in sediments, and biomethylation was postulated by Wong et al. (1975) and Schmidt & Huber (1976). However, no biological methylation of inorganic lead was noted by Reisinger et al. (1981) in studies under many conditions using several bacterial species known to alkylate mercury and other heavy metals. The authors did find chemical methylation in the presence of methylcobalamin and sulfide or aluminium ions and it was independent of the presence of bacteria. The evidence for microbial methylation of various compounds of lead in aquatic systems has been reviewed by Beijer & Jernelöv (1984). It is still unclear whether the TML formed is produced abiotically or by biotransformation.

4.3 Ecotoxicity

4.3.1 Toxicity to Microorganisms

In general, according to the WHO (1989), inorganic lead compounds are of lower toxicity to microorganisms than are trialkyl- and tetraalkyl lead compounds. Tetra- alkyl lead becomes toxic by decomposition into the ionic trialkyl lead. One of the most important factors which influence the aquatic toxicity of lead is the free ionic concentration, which affects the availability of lead to organisms. The toxicity of inorganic lead salts is strongly dependent on environmental conditions such as water hardness, pH, and salinity, a fact which has not been adequately considered in most toxicity studies. There is evidence that tolerant strains exist and that tolerance may develop in others.

4.3.2 Toxicity to Aquatic Organisms

The WHO (1989) reports that lead is unlikely to affect aquatic plants at levels that might be found in the general environment.

In the form of simple salts, lead is acutely toxic to aquatic invertebrates at concentrations above 0.1 and >40 mg/l for fresh- water organisms and above 2.5 and >500 mg/l for marine organisms. For the same species, the 96-h LC_{50}s for fish vary between 1 and 27 mg/l in soft water, and between 440 and 540 mg/l in hard water. The higher values for hard water represent nominal concentrations. Available lead measurements suggest that little of the total lead is in solution in hard water. Lead salts are poorly soluble in water, and the presence of other salts reduces the availability of lead to organisms because of precipitation. Results of toxicity tests should be treated with caution unless dissolved lead is measured.

In communities of aquatic invertebrates, some populations are more sensitive than others and community structure may be adversely affected by lead contamination. However, populations of invertebrates from polluted areas can show more tolerance to lead than those from non- polluted areas. In other aquatic invertebrates, adaptation to hypoxic conditions can be hindered by high lead concentrations.

Young stages of fish are more susceptible to lead than adults or eggs. Typical symptoms of lead toxicity include spinal deformity and blackening of the caudal region. The maximum acceptable toxicant limit (MATC) for inorganic lead has been determined for several species under different conditions and results range from 0.04 mg/l to 0.198 mg/l. The acute toxicity of lead is highly dependent on the presence of other ions in solution, and the measurement of dissolved lead in toxicity tests is essential for a realistic result. Organic compounds are more toxic to fish than inorganic lead salts.

There is evidence that frog and toad eggs are sensitive to nominal lead concentrations of less than 1.0 mg/l in standing water and 0.04 mg/l in flow-through systems; arrested development and delayed hatching have been observed. For adult frogs, there are no significant effects below 5 mg/l in aqueous solution, but lead in the diet at 10 mg/kg food has some biochemical effects.

4.3.3 Toxicity to Terrestrial Organisms

According to the WHO (1989), the tendency of inorganic lead to form highly insoluble salts and complexes with various anions, together with its tight binding to soils, drastically reduces its availability to terrestrial plants via the roots. Translocation of the ion in plants is limited and most bound lead stays at root or leaf surfaces. As a result, in most experimental studies on lead toxicity, high lead concentrations in the range of 100 to 1000 mg/kg soil are needed to cause visible toxic effects on photosynthesis, growth, or other parameters. Thus, lead is only likely to affect plants at sites of very high environmental concentrations.

Ingestion of lead-contaminated bacteria and fungi by nematodes leads to impaired reproduction. Woodlice seem unusually tolerant to lead, since prolonged exposure to soil or grass litter containing externally added lead salts had no effect. Caterpillars maintained on a diet containing lead salts show symptoms of toxicity leading to impaired development and reproduction.

The information available is too meagre to quantify the risks to invertebrates during the decomposition of lead-contaminated litter.

Lead salts are only toxic to birds at a high dietary dosage (100 mg/kg or more). Almost all of the experimental work is on chickens and other gallinaceous birds. Exposure of quail from hatching and up to reproductive age resulted in effects on egg production at dietary lead levels of 10 mg/kg. Although a variety of effects at high dosage have been reported, most can be explained as a primary effect on food consumption. Diarrhoea and lack of appetite, leading to anorexia and weight loss, are the primary effects of lead salts. Since there is no experimental evidence to assess effects on other bird species, it is necessary to assume a comparable sensitivity. If this is so, then it is highly improbable that environmental exposure would cause adverse effects.

Metallic lead is not toxic to birds except at very high dosage when administered in the form of powder. It is highly toxic to birds when given as lead shot; ingestion of a single pellet of lead shot can be fatal for some birds. The sensitivity varies between species and is dependent on diet. Since birds have been found in the wild with large numbers of lead shot in the gizzard (20 shot is not unusual), this poses a major hazard to those species feeding on river margins and in fields where many shot have accumulated.

There is little information on the effects of organolead compounds. Trialkyl lead compounds produced effects on starlings dosed at 0.2 mg/day; 2 mg/day was invariably fatal.

There are too few reports to draw conclusions about the effects of lead on non-laboratory mammals. Wild rats showed similar effects to their laboratory counterparts.

5 Environmental Exposure/Risk Evaluation

Environmental effects were not relevant to the risk evaluations used by the notifying Parties to support their regulatory actions.

Annex 2 – Details on final regulatory actions reported

Country Name: Canada

1	**Effective date(s) of entry into force of actions**	April 26, 1990
	Reference to the regulatory document	*Regulations respecting concentrations of lead and phosphorus in gasoline* (also cited as the *Gasoline Regulations*)
2	**Succinct details of the final regulatory action(s)**	The Gasoline Regulations regulate the concentration of phosphorus and lead permitted in leaded and unleaded fuels manufactured or imported into Canada, and offered for sale or sold. The Regulations apply to gasoline producers and importers. The Regulations set maximum lead concentration limits for leaded gasoline to be used in farm equipment, boats or heavy trucks. The Regulations do not apply to aircraft. Amendments were made in 1994, 1997, 1998 and 2003 to exempt high performance competition vehicles. The latest amendment extended the exemption until January 1, 2008.
3	**Reasons for action**	Human health
4	**Basis for inclusion into Annex III**	
4.1	**Risk evaluation**	Health Canada determined from studies that adverse health effects can occur at blood lead levels of 20-30μg/dl. Furthermore, these studies indicated that: • significant numbers of Canadian children could have blood lead levels in this range and could therefore be at risk. Results from a study in Toronto in 1982, indicated that 1% of children aged 0-4 years have blood lead concentrations greater than 30 μg/L and 12% greater than 20 μg/L; and • gasoline lead contributed an estimated 30-35% of the lead in the blood of urban adults. The proportion of gasoline lead in children's blood may range from 30-40%.
4.2	**Criteria used**	Unacceptable risk to human health.
	Relevance to other States and Region	No information provided.
5	**Alternatives**	The octane rating of gasoline can be maintained either by redesigning refinery processes or with alternative octane-enhancing agents.
6	**Waste management**	No information provided.
7	**Other**	

Country Name: European Community

1	**Effective date(s) of entry into force of actions**	1 January 2000, for the most recent regulatory action, which was the latest in a series of actions progressively extending restriction on the use of leaded petrol.
	Reference to the regulatory document	Council Directive 98/70/EC of the European Parliament and of the Council of 12 October 1998 relating to the quality of petrol and diesel fuels and amending Council Directive 93/12/EEC
2	**Succinct details of the final regulatory action(s)**	As of 1 January 2000, the placing on the market of leaded petrol for vehicles was banned. However, Member States could be allowed to continue to permit the marketing of leaded petrol (< 150 mg/L) within their territory until January 1, 2005, provided that it could be demonstrated that a ban would result in severe socio-economic problems or would not lead to overall environmental or health benefits. Member States may also allow a derogation for small quantities of leaded petrol containing not more than 0.15g lead/L, up to a maximum of 0.5% of total sales, for collectors' old cars. The lead content of petrol for aircraft (piston engine) is not covered by the regulatory action. Other relevant regulatory actions: Council Directive 78/611/EEC of 29 June 1978, Council Directive 85/210/EEC of 20 March 1985, Council Directive 87/416/EEC of 21 July 1987
3	**Reasons for action**	Human health
4	**Basis for inclusion into Annex III**	
4.1	**Risk evaluation**	Blood lead sampling is one of the methods most widely used to assess human risk from exposure to lead. In 1977, the European Community adopted Council Directive 77/312/EEC of 29 March 1977 on biological screening of the population for lead. In each Member State, 50 or more blood lead samples had to be analysed per million inhabitants. In 1982 (Council Directive 82/884/EEC of 3 December 1982), a limit value was set at 2 $\mu g/m^3$ of lead in the ambient air in terms of annual average. In several Member States where blood analyses were carried out in the 70s and the 80s,it was shown that the decreases in air concentration due to restrictions on leaded fuel was related to the diminution in blood lead levels.
4.2	**Criteria used**	Unacceptable risk to human health.
	Relevance to other States and Region	General health problems in all states where the substance is used. Protection of workers and the general public.
5	**Alternatives**	No information provided.
6	**Waste management**	No information provided.
7	**Other**	No information provided.

Annex 3 – Addresses of designated national authorities

Canada

C Chief Chemicals Control Environment Canada Place Vincent Massey, 12th floor 351 St. Joseph Boulevard Hull, Quebec K1A 0H3 *Bernard Madé*	***Phone*** +819 994-3648 ***Fax*** +819 994-0007 ***Telex*** ***e-mail*** bernard.made@ec.gc.ca

European Community

CP DG Environment European Commission Rue de la Loi 200 B-1049 Brussels Belgium *Klaus Berend*	***Phone*** +322 299 48 60 ***Fax*** +322 296 85 58 ***Telex*** ***e-mail*** klaus.berend@cec.eu.int

C Industrial chemicals
CP Pesticides and industrial chemicals
P Pesticides

Annex 4 – References

Regulatory actions

Canada

Gasoline Regulations (SOR/90-247) under the *Canadian Environmental Protection Act*, *Canada Gazette, Part II*, Vol. 124, No.10, May 9, 1990.
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European Community

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Other relevant regulatory actions:

Council Directive 78/611/EEC of 29 June 1978 on the approximation of the laws of the Member States concerning the lead content of petrol (Official Journal of the European Communities L197 of 22/07/1978, p. 19).
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Canada

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Alternatives

Canada

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General

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Canada

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