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LONG-RANGE TRANSBOUNDARY AIR POLLUTION**

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**HEALTH RISKS OF PERSISTENT ORGANIC POLLUTANTS
FROM LONG-RANGE TRANSBOUNDARY AIR POLLUTION**

Executive summary of the report prepared by the joint Task Force on the Health Aspects of Air Pollution
of the World Health Organization/European Centre for Environment and Health
and the Executive Body

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Acronyms and abbreviations

ADI	Acceptable daily intake
ATSDR	Agency for Toxic Substances and Disease Registry
BCF	Bioconcentration factor
bw	Body weight
CLRTAP	Convention for Long-range Transboundary Air Pollution
CPs	Chlorinated paraffins
DDD	1,1-dichloro-2,2-bis(4-chlorophenyl)ethane
DDE	1,1-dichloro-2,2-bis(4-chlorophenyl)ethylene
DDT	1,1,1-trichloro-2,2-bis (4-chlorophenyl) ethane
DI	Daily intake
EPA	Environmental Protection Agency (United States of America)
FAO	Food and Agriculture Organization of the United Nations
HCB	Hexachlorobenzene
HCH	Hexachlorocyclohexane
IARC	International Agency for Research on Cancer
JECFA	Joint FAO/WHO Expert Committee on Food Additives
JMPR	Joint FAO/WHO Meeting on Pesticide Residues
K _{oc}	Organic carbon partition coefficient
K _{ow}	Octanol/water partition coefficient
LOAEL	Lowest observed adverse effect level
LRT	Long-range transport
LRTAP	Long-range transboundary air pollution
MAC	Maximum allowable concentration
NOAEL	No observed adverse effects level
PAHs	Polycyclic aromatic hydrocarbons
PBDD/Fs	Polybrominated dibenzo- <i>p</i> -dioxins and dibenzofurans
PBDDs	Polybrominated dibenzo- <i>p</i> -dioxins
PBDE	Polybrominated diphenylethers
PBDFs	Polychlorinated dibenzofurans
PCBs	Polychlorinated biphenyls
PCDD/Fs	Polychlorinated dibenzo- <i>p</i> -dioxins and dibenzofurans
PCDDs	Polychlorinated dibenzo- <i>p</i> -dioxins
PCDFs	Polychlorinated dibenzofurans
PCN	Polychlorinated naphthalenes
PCP	Pentachlorophenol
PCTs	Polychlorinated terphenyls
POP(s)	Persistent organic pollutant(s)
PTDI	Provisional tolerable daily intake
SCCPs	Short-chain chlorinated paraffins
TCDD	2,3,7,8-tetrachlorodibenzo- <i>p</i> -dioxin
TDI	Tolerable daily intake
TEQ	Toxic equivalent(s)
TRI	Toxic Release Inventory
WHO	World Health Organization

Introduction

1. Persistent organic pollutants (POPs) are organic compounds of anthropogenic origin which resist photolytic, biological or chemical degradation, leading to bioaccumulation in the food chain. They can be transported over long distances in the atmosphere resulting in widespread distribution across the Earth, including regions where they have never been used. Due to their toxic characteristics they can pose a threat to humans and the environment. In recent years the international community has, therefore, called for urgent global action to identify their possible risk to human health and the environment, and to reduce and eliminate their release.
2. The Protocol on Persistent Organic Pollutants to the UNECE Convention on Long-range Transboundary Air Pollution addresses several of those compounds (aldrin, chlordane, chlordecone, DDT, dieldrin, endrin, heptachlor, hexabromobiphenyl, hexachlorobenzene (HCB), hexachlorocyclohexane (HCH), mirex, polyaromatic hydrocarbons (PAHs), PCBs, PCDDs, PCDFs and toxaphene). The Protocol describes the technical measures to eliminate or restrict the production or use of these substances, and it identifies the action required to achieve that goal.
3. It is the objective of the Protocol to prevent adverse effects of POPs on human health or the environment. To this end the Executive Body for the Convention at its seventeenth session (29 November – 3 December 1999, Gothenburg, Sweden) requested the joint Task Force on the Health Aspects of Air Pollution of the World Health Organization/European Centre for Environment and Health (WHO/ECEH) and the Executive Body (Task Force on the Health Aspects) to provide a preliminary selection of priority POPs based on the assessment of potential health effects and on the potential contribution of long-range transport to population exposure and risk. Following this request, at its third meeting (10 May 2000) the Task Force selected the following groups of substances for which the risk assessment would be conducted: pentachlorophenol (PCP), DDT, lindane (HCH), hexachlorobenzene (HCB), heptachlor, polychlorinated dibenzodioxins and dibenzofurans (PCDD/Fs), polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs).¹ Furthermore, a short hazard assessment was planned for polychlorinated terphenyls (PCTs), ugilec, polybrominated diphenylethers (PBDEs), polybrominated dibenzodioxins and furans (PBDD/Fs) and short-chain chlorinated paraffins (SCCPs).
4. Experts invited by WHO/ECEH (Bonn Office) prepared first drafts of the background papers, which were reviewed at the fourth meeting of the Task Force (3-4 December 2001, Bonn, Germany). Following the recommendations of the Meeting, the expert group designated by the Task Force prepared the second draft of the report on the health risks of POPs from long-range transboundary air pollution. Comments on this draft were received both from members of the expert group and from external reviewers. A small drafting group met on 26 April 2002 and proposed a uniform format for the review of each group of substances, as well as the outline of the report's executive summary to be presented to the Working Group on Effects at its twenty-first session.

1 See: <http://www.unece.org/env/documents/2000/eb/wg1/eb.air.wg.1.2000.12.e.pdf>

5. The fifth meeting of the Task Force was held from 13 to 14 May 2002 in Bonn. Twenty experts from 14 Parties to the Convention as well as WHO participated in the meeting (see annex I). Mr. D. Stone (Canada) chaired the meeting; Mr. L. van Bree was rapporteur. The Task Force reviewed the second draft of the report prepared by the group of designated experts, as well as the comments received from external reviewers. The Task Force also prepared the executive summary of the report on the "Health risks of heavy metals from long-range transboundary air pollution" and approved it for submission to the Working Group on Effects. As the information was incomplete, the Task Force could not evaluate the health risks of unileg related to long-range transboundary air pollution. The information on unileg, however, will be included in the full report on the "Health risks of heavy metals from long-range transboundary air pollution" to be finalized for publication by August 2002. The full report will contain background information and references to the material on which the executive summary presented in this document is based.

6. The present assessment conducted by the Task Force complements the work of the Experts Group on POPs under the Executive Body for the Convention. This Group is reviewing the obligations on substances already in the 1998 Protocol on Persistent Organic Pollutants and is advising national experts preparing preliminary risk profiles on substances which may be candidates for inclusion in the Protocol.

ASSESSMENT OF THE HEALTH RISKS OF SELECTED PERSISTENT ORGANIC POLLUTANTS FROM LONG-RANGE TRANSBOUNDARY AIR POLLUTION

I. PENTACHLOROPHENOL

Introduction

7. Pentachlorophenol (PCP) can enter the environment as a by-product from various chemical-manufacturing processes, from its uses as a wood preservative, as a general-purpose herbicide, as a biocide in industrial water systems and as a result of incineration of chlorine-containing waste. Its sodium salt is used for similar purposes and readily degrades to PCP. Actions on PCP are not included in the 1998 Protocol on POPs. However, PCP may be identified through article 8 concerning research, development, monitoring and cooperation. Under this article priority is given to substances most likely to be considered to be submitted under the provisions which allow substances to be added.

A. Characteristics and a long-range transboundary air pollution potential

8. The physical and chemical properties of the compound suggest limited evaporation to the atmosphere and most of it will move with water and generally associate with soil particles. The mobility and availability of PCP in the environment depends on the acidity of the medium. The volatilization of PCP from treated wood increases with the temperature; similar results with temperature change were seen with all the numerous solvent systems used for the application of the compound.

9. In air, soil and surface water PCP is subjected to photolysis and hydroxyl degradation, with atmospheric half-lives ranging from hours to weeks.

10. PCP bioconcentrates in aquatic organisms and the BCF value increases with falling pH.

B. Human exposure and long-range transboundary air pollution

11. Human exposure will be derived from three main routes, emission from treated products, from drinking water and from food. The last two sources are relevant for long-range transboundary air pollution (LRTAP). The long-term average daily intake of PCP by the general population was estimated in 1989 to be 16 µg/day in the United States; in Canada the estimated daily intake is 0.05 µg/kg of body weight per day. It seems likely that food accounts for most of the intake, unless there is specific local chlorophenol contamination causing increased concentrations in drinking water or exposure from wooden homes treated with PCP.

12. Concerning measurements in biota as evidence of transport to remote regions, the situation is complicated by two main factors: on the one hand PCP is metabolized into other molecules and therefore the absence of it in animal tissues is not conclusive, on the other hand PCP is also a major product of the metabolism of hexachlorobenzene and other common pesticides in mammals and, therefore, if it is found it does not mean that it was taken up as PCP.

C. Health hazard characterization

13. PCP is rapidly absorbed by the digestive tract. The highest concentrations of PCP are in the liver, kidney and brain, but the tendency for bioaccumulation remains low. Regarding human health effects, the experimental data related to PCP are well documented for the oral low-dose chronic exposures and indicate:

- Impact on the liver characterized by biochemical, functional and histopathological changes;
 - Impact on the immune system;
 - Significant alteration on thyroid hormone levels at exposure of 1 or 2 mg/kg bw per day.
- Data on occupationally exposed workers confirm effects on the immune system and the liver.

14. The International Agency for Research on Cancer (IARC) (1991) has classified PCP in group 2B, the agents possibly carcinogenic to humans.

15. WHO has assessed PCP in order to establish water quality guidelines. In 1993 a tolerable daily intake (TDI) of 0.003 mg/kg bw was set, and although a subsequent risk assessment was conducted based on neoplastic effects in 1998, it is noted that the resultant water quality guideline was the same (9 µg/l).

D. Human health and long-range transboundary air pollution

16. The health characterization of PCP indicates a potential for a number of human health effects associated with low-level chronic exposure via the oral route. Some of these effects have been seen as a result of occupational exposure. It is also known that anthropogenically produced PCPs introduced into the environment have the potential for long-range atmospheric transport and may reach human foodstuffs and drinking water. However, more research is needed to assess the significance of LRTAP as a significant pathway leading to human exposure via the oral route.

II. DDT

Introduction

17. 1,1,1-trichloro-2,2-bis (4-chlorophenyl) ethane (DDT) was first synthesized in 1874. Technical grade DDT is composed of up to 14 chemical compounds, of which only 65-80% is the active ingredient p,p'-DDT. Worldwide production peaked in the sixties. DDT is still being released into the atmosphere from current production and use in some developing countries. Oceans and large bodies of fresh water may release significant amounts of DDT (residues from previous uses, and its breakdown products) into the atmosphere. The 1998 Protocol on POPs eliminates the production and use of DDT by its Parties except as an intermediate chemical to produce dicofol or for public health purposes (e.g. malaria control). The former is to be reassessed within two years of entry in force and the latter is accompanied by special conditions. In addition, the Parties are committed to eliminating all production once available and feasible alternatives have been identified and for this purpose consultation with health agencies including WHO is necessary. These provisions acknowledge that DDT remains an important and necessary substance for vector control in the developing countries.

A. Characteristics and a long-range transboundary air pollution potential

18. DDT and its breakdown products are semi-volatile and can be expected to partition into the atmosphere as a result, and precipitate at low temperatures. Therefore, in addition to being found close to known sources, they can also occur at significant levels in remote areas. They are insoluble in water and soluble in most organic solvents. Due to these physico-chemical properties, DDT and its metabolites are readily absorbed by organisms; the high lipid- and the low water-solubility lead to the retention of the compounds in fatty tissues. Consequently, there is significant potential for biomagnification. The breakdown products of DDT, DDD and DDE, are present virtually everywhere in the environment and are more persistent than the parent compound.

B. Human exposure and long-range transboundary air pollution

19. DDT and its metabolites have been found to be ubiquitous in foods, particularly in fatty food of animal origin (meat, fish, dairy products) and also in human milk. Globally, food is the main source of exposure for the general population. Estimated current intake varies, according to different diets and geographical areas, and in some countries the estimated daily intake may approach the acceptable daily intake. Since DDT and its metabolites are secreted through human milk, breastfed children have to be considered as a high-exposure group. Also prenatal exposure may take place, due to the capacity of DDT and its metabolites to cross the placenta and enter the amniotic fluid. Once absorbed, DDT is readily distributed to all body tissues, where the storage rate is proportional to the fat content of the organ.

C. Health hazard characterization

20. Regarding the human health implication of DDT, the critical issues associated with the levels of lifetime chronic exposure typically reported in the literature are:

(a) Carcinogenicity: There is inadequate evidence in humans and sufficient evidence in experimental animals of the carcinogenicity of DDT, therefore DDT is possibly carcinogenic to humans (Group 2B) (WHO, 1991);

(b) Endocrine disruption: p,p'-DDE exhibits anti-androgenic properties, and a DDT isomer, o,p'-DDT, is estrogenic. Even if it is not clear whether an endocrine effect may be observed also in man, the possibility of endocrine disruption has to be taken into account, mainly because exposure may involve fetuses and newborn infants in the critical period of their development;

(c) Neurobehavioural changes: although the topic is poorly explored by research, DDT neurotoxicity must be investigated, especially because neurobehavioural effects may be consequent to endocrine disruption.

(d) Fertility: the topic is scarcely explored, though some data are available suggesting DDT and its metabolites have the capacity to affect fertility;

(e) Immunotoxicity: even if no firm conclusions can be reached on this topic, some data are available suggesting that DDT and its metabolites act as immunosuppressive agents;

(f) Developmental toxicity: the provisional tolerable daily intake (PTDI) for humans is 0.01 mg/kg body weight. This limit has been established based on a no observed adverse effects level (NOAEL) of 1 mg/kg bw per day for developmental toxicity in rats, with a safety factor of 100 (Joint FAO/WHO Meeting on Pesticide Residue, 2000).

D. Human health and long-range transboundary air pollution

21. Human intake through the diet may approach or even exceed the PTDI, particularly in tropical and developing countries, where DDT is still used for public health purposes, or even illegally used. In these countries, local use represents the main source of exposure. On the other hand, high-level exposed groups also occur within the ECE region. These include the Inuit of Arctic regions, where DDT has not been used for decades, or has never been used. The main source of exposure in this case and the consequent health implications are mainly related to LRTAP.

III. HEXACHLOROCYCLOHEXANES

Introduction

22. γ -hexachlorocyclohexane (γ -HCH, or lindane) is used as an insecticide; it is used on fruit and vegetable crops (including greenhouse vegetables and tobacco), for seed treatment, in forestry (including Christmas tree treatment) and for animal treatment. Other HCH isomers are still found in environmental samples, due to the former use of technical HCH as insecticide.

23. Technical HCH is restricted under the 1998 Protocol on POPs to use as an intermediate in manufacturing other substances. γ -HCH (lindane) is restricted to the following uses: seed treatment, soil application directly followed by incorporation into the topsoil, professional remedial and industrial treatment of lumber, timber and logs, public health and veterinary topical insecticide, non aerial application to tree seedlings and indoor industrial and residential applications. All of these uses are to be reassessed under the Protocol no later than two years after entry into force.

24. HCH is not covered by the Stockholm Convention on POPs, and the use of technical HCH and γ -HCH in other parts of the world is continuing.

A. Characteristics and a long-range transboundary air pollution potential

25. α - and γ -HCH are water-soluble and have little bioconcentration potential. γ -HCH is very prevalent in the marine environment and soils, but low levels are found in biota. A minor constituent of lindane is β -HCH; this isomer has reduced water solubility and hence a more significant bioconcentration factor (BCF) than γ -HCH.

26. HCH residues are found in water and air samples all over the world. Often higher concentrations are found in northern waters than in major source regions in the mid-latitudes. The presence of HCH in the environment far away from the sources is considered to be due to LRTAP.

27. The presence of large quantities of γ -HCH in the oceans and lakes introduces a delay in the response of atmospheric concentrations to decreases in emissions.

B. Human exposure and long-range transboundary air pollution

28. More than 90% of human exposure to all HCH isomers originates from food, particularly those which are animal based (IPCS 1991). The food intake of lindane decreased by more than an order of magnitude in the 1970s, to levels at least two orders of magnitude below the acceptably daily intake (ADI) established by WHO in 1989. Intake from (indoor) air may be considerable for people living in houses treated for pest control.

29. β -HCH is the predominant HCH isomer accumulating in human tissues, which is indicated for example by the levels in human milk. Levels of β -HCH in human milk range from 0.1 to 0.69 mg/kg, and those of γ -HCH from < 0.001 to 0.1 mg/kg (on a fat basis). The intake of lindane derived from commercially produced food has decreased since the 1970s which is a response to decreasing emissions. However, this trend is not evident for populations using marine foods, particularly marine mammals. There is a relationship between the HCH concentration in breast milk and the consumption of meat products, animal fat and fatty fish. Levels in the milk of women living in rural areas appear to be higher than in urban areas.

C. Health hazard characterization

30. γ -HCH is rapidly absorbed by the oral route and undergoes extensive metabolism mainly in the liver. Animal studies showed neurotoxic effects, hepatic effects and reproductive effects as well as immunotoxicity in mice. In humans, poisoning incidents have generally been associated with significant misuse of the compound. The most common signs of toxicity following oral ingestion were seizures, convulsions, vomiting and dizziness. Human data suggest that γ -HCH has a potential to induce haematological effects (aplastic anaemia) but establishing a causal relationship has been difficult due to a lack of personal exposure data.

31. IARC (1987) has concluded that for the technical grade and α -HCH there is sufficient evidence of carcinogenicity to animals, whereas this evidence is limited for β - and γ - isomers. There is inadequate evidence of their carcinogenicity to human beings. Hexachlorocyclohexanes were classified in group 2B as possible carcinogenic to humans. However, it should be noted that the European Union and US EPA have not classified HCH as carcinogenic to humans.

32. The Joint FAO/WHO Meeting on Pesticide Residues (JMPR) established a temporary ADI of 0.001 mg/kg bw for lindane in 1997 based on a NOAEL of 0.5 mg/kg bw established in a two-year toxicity and carcinogenicity study in rats and using a safety factor of 500.

D. Human health and long-range transboundary air pollution

33. It is known that large reservoirs of HCH exist in the environment, which indicate a potentially long time for environmental levels to reflect control actions. The health hazard characterization has identified a range of health effects related to the γ -HCH isomer from the oral route. Some might be relevant to observed environmental exposures. The oral route is the most relevant for the LRTAP sources. Taking into account the uncertainties of the information and specifically the level of exposure at which human health effects can happen, HCH may be considered as a possible risk to health acting through LRTAP.

IV. HEXACHLOROBENZENE

Introduction

34. Hexachlorobenzene (HCB) is introduced into the environment as a seed fungicide, through industrial production (by-products) and due to unwanted emission during waste incineration. Another minor source of hexachlorobenzene releases to the air is the use of pyrotechnic mixtures that produce white obscurant screening smokes. HCB is banned as a chemical for production and use in many developed countries. HCB is today found in almost all compartments of the global ecosystem in at least trace amounts. This substance is already included in the 1998 Protocol on POPs (in its annexes I and III).

A. Characteristics and a long-range transboundary air pollution potential

35. Hexachlorobenzene is very persistent in the environment due to its chemical stability and resistance to biodegradation. Long-range transport plays a significant role as a means of redistribution of HCB throughout the environment via atmospheric or oceanic systems. If released to the atmosphere, hexachlorobenzene exists primarily in the vapour phase and degradation is extremely slow.

B. Human exposure and long-range transboundary air pollution

36. HCB has the ability to bioconcentrate and biomagnify under typical environmental conditions. It is estimated that more than 91% of the total exposure of the general population to HCB originates from common food items, both of animal origin (e.g. meat, certain fish and dairy products) and plant products. Intakes from ambient air (about 7%) and drinking water (about 1%)

of the total intake) are considerably lower. The total average daily intake of HCB from food, air and drinking water in the general population in Europe and North America is between 0.0004 and 0.003 µg/kg body weight per day. HCB has been detected in the milk of several species, including humans.

C. Health hazard characterization

37. The most sensitive target organ for HCB is the liver, where the effect resulting from low-level chronic exposure is a disturbance of porphyrin metabolism. In case of high-level exposure, the disturbance of porphyrin metabolism will also lead to skin lesions (erythema, bullae), hyperpigmentation and enlarged liver. In animal experiments a range of effects have been observed at levels close to that which causes liver effects, such as disturbance of the immune function, neurobehavioural development, changes in calcium metabolism and ovarian morphology. Regarding carcinogenicity, there is sufficient evidence in experimental animals, but insufficient evidence in humans, therefore HCB is possibly carcinogenic to humans (IARC 2B). For human risk assessment, WHO (1997) has derived tolerable daily intakes (TDI) of 0.17 µg/kg body weight per day for non-neoplastic effects and a guidance value of 0.16 µg/kg body weight per day for neoplastic effects. Since HCB crosses the placenta and is present in breast milk, there may be concern for effects resulting from prenatal and neonatal exposure.

D. Human health and long-range transboundary air pollution

38. HCB is still released to the environment in the region covered by the Convention on Long-range Transboundary Air Pollution, mainly as a result of unintentional emission from waste incineration and as a by-product from manufacturing. The health characterization of HCB has identified a number of human health effects of potential relevance for low-level chronic exposure via the oral route. Exposure through food is the most relevant to LRTAP-derived sources.

V. HEPTACHLOR

Introduction

39. Heptachlor is a non-systemic contact insecticide, used primarily against soil insects and termites. It has also been used against cotton insects, grasshoppers and some crop pests, and to combat malaria. Heptachlor is present as an impurity in the pesticide chlordane. The use of heptachlor has been banned or severely restricted in many countries around the globe since the late 1970s and therefore current environmental concentrations are principally the result of environmental recycling. Contemporary use of chlordane that is contaminated with heptachlor may be responsible for sporadic atmospheric inputs to the remote Arctic environment. This substance is already included in the 1998 Protocol on POPs in the list of substances scheduled for elimination.

A. Characteristics and a long-range transboundary air pollution potential

40. Heptachlor is characterized by its semi-volatility, resistance to degradation and low water solubility. These characteristics predispose it to high environmental persistence and to long-range transport. The persistence of heptachlor and its oxidation product, heptachlor epoxide, combined

with a high octanol to water partition coefficient provides the necessary conditions for it to bioconcentrate in terrestrial and aquatic food web chains. Air is probably the most significant compartment for global environmental distribution.

B. Human exposure and long-range transboundary air pollution

41. The general population is exposed to heptachlor and heptachlor epoxide mainly via food items, particularly fatty food of animal origin (e.g. meat, fish and dairy products). Heptachlor is generally not detectable in the human population, but its oxidation product, heptachlor epoxide, has been found in human fat, blood, organs and milk. Since the 1970s, dietary intakes of heptachlor and heptachlor epoxide have declined significantly in industrialized countries as the use of the compound has been reduced. Current values in the ECE region range from 0.02 to 1.2 µg/day. The exposure of infants to breast milk results in higher exposure levels in children than in adults. The presence of heptachlor and heptachlor epoxide in remote locations points to the significance of LRTAP as an important element of the exposure pathway in those areas.

C. Health hazard characterization

42. Heptachlor is readily absorbed via all routes of exposure and is readily metabolized to heptachlor epoxide by mammals. Heptachlor epoxide is metabolized slowly and is the most persistent metabolite; it is mainly stored in adipose tissue, but also in liver, kidney and muscle. Animal studies have reported effects on the liver, kidney and the immune and nervous systems from oral exposure to heptachlor. Heptachlor has been shown to cross the placenta to the developing foetus in humans. Based on consistent findings of neoplastic effects in experimental animals, IARC (1991) has classified heptachlor as a possible human carcinogen (2B). Inadequate information is available to determine whether heptachlor may cause developmental or reproductive effects in humans. JMPR (1991) has established an ADI of 0.1 µg/kg of body weight for heptachlor.

D. Human health and long-range transboundary air pollution

43. It appears that the general population is not at risk from LRTAP-derived heptachlor; however, highly exposed groups such as some breastfed infants and the Inuit in the Arctic may be at risk. Long-range transport represents the most important source of heptachlor found in the terrestrial and aquatic food chain in remote regions, although the environmental concentrations of heptachlor in those regions are likely to be very low as a result of limited contemporary use.

VI. DIOXINS AND DIOXIN-LIKE PCBs

Introduction

44. Polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans (PCDD/Fs) are today found in almost all compartments of the global ecosystem in at least trace amounts. PCDD/Fs are formed as unwanted by-products in many industrial and combustion processes. PCDD/Fs are also generated in forest fires and volcanoes. Polychlorinated biphenyls (PCBs) have been used commercially since 1929 as dielectric and heat exchange fluids and in a variety of other

applications. These three substance classes are found in human tissue in many parts of the world, including remote areas with no production or use. At present, the major source of exposure could be redistribution, but primary sources could also be of significant importance. PCDD/Fs and dioxin-like PCBs are considered to act via a common mechanism of toxicity. These substances are already included in the 1998 Protocol on POPs and PCBs were given "Elimination status" (annex A) in the Stockholm Convention on POPs, whereas PCDD/Fs are annex C substances (article 5: Measures to reduce or eliminate releases from unintentional production) in the same Convention.

A. Characteristics and a long-range transboundary air pollution potential

45. PCDD/Fs and dioxin-like PCBs are characterized by their semi-volatility and resistance to degradation. Their water solubility is low. These characteristics predispose them to environmental persistence and to long-range transport. They intensively adsorb onto particles in air, soil and sediment and accumulate in fat-containing tissues. The strong adsorption causes their mobility in soil and sediments to be negligible. Air is probably the most significant compartment for environmental distribution. They have the ability to bioconcentrate and biomagnify under typical environmental conditions, thereby potentially achieving toxicologically relevant concentrations.

B. Human exposure and long-range transboundary air pollution

46. The general population is mainly exposed to PCDD/Fs and dioxin-like PCBs via common food items, particularly fatty food of animal origin (e.g. meat, certain fish and dairy products). Estimated average current intake levels are in the range of 1-3 pg TEQ/kg bw per day. Data available from industrialized countries have shown a reduction in population exposure levels in the last decades, but there are indications that this decline has levelled off. Prenatal and neonatal exposure is considered particularly important, as breastfed infants exceed adult exposures to PCDD/Fs and PCBs by one to two orders of magnitude. As a result of LRTAP and dietary habits, human exposure to dioxin-like PCBs in many Arctic regions is considerably higher than in industrialized areas. This observation, as well as the presence of dioxins in remote locations, point to the significance of LRTAP as an important element of the exposure pathway in those areas.

C. Health hazard characterization

47. As 2,3,7,8-substituted PCDD/Fs and dioxin-like PCBs are believed to act through a common toxicological mechanism, a toxic equivalency factor (TEF) concept has been established, allowing calculation of the combined toxicity in a mixture of PCDD/Fs and dioxin-like PCBs. As it is likely that other substances, e.g. polychlorinated naphthalenes (PCNs), could act via the same mechanism, it has been proposed that they could also be included in the TEF scheme and add to the estimated toxicity. Critical health outcomes include cancer, immunosuppression, behavioural changes and reproductive effects. The developing foetus and the neonate are thought to represent a population potentially "at risk" population due to their increased susceptibility. WHO recommends a TDI with a range of 1-4 pg TEQ/kg bw, but stresses that the upper limit of the range should be considered a maximal tolerable intake, which should be reduced to below 1 pg TEQ/kg bw/day. The Scientific Committee for Food of the European Commission has proposed a temporary tolerable weekly intake of 14 pg TEQ /kg bw, while the Joint FAO/WHO Expert Committee on Food Additives (JECFA) has suggested a provisional tolerable monthly intake value of 70 pg

TEQ /kg bw. IARC has classified TCDD as a human carcinogen (class 1) (IARC, 1997). PCBs were classified as probably carcinogenic to humans (class 2A).

D. Human health and long-range transboundary air pollution

48. As human exposure levels often exceed the TDI, the weight of evidence suggests an increased risk of harmful health effects in the general population, especially for breastfed infants and populations with specific diets.

49. Since the chemical and physical properties of PCDD/Fs and dioxin-like PCBs make them susceptible to LRTAP, it is expected to contribute significantly to the exposure and health risks.

VII. POLYCHLORINATED BIPHENYLS

Introduction

50. Polychlorinated biphenyls (PCBs) are found in almost all compartments of the global ecosystem in at least trace amounts. PCBs have been used commercially since 1929 as dielectric and heat exchange fluids and in a variety of other applications. PCBs are found in human tissues in many parts of the world, including remote areas with no PCB production or use. At present, the major source of exposure could be redistribution, but primary sources could also be of significant importance. Based on biological activity, PCBs have been divided into non-dioxin-like and dioxin-like categories. Dioxin-like PCB congeners are considered to act via the same mechanism of toxicity as polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs). PCBs are scheduled for elimination under both the 1998 Protocol on POPs and the Stockholm Convention on POPs.

A. Characteristics and a long-range transboundary air pollution potential

51. PCBs are characterized by their semi-volatility and resistance to degradation. Their water solubility is low. These characteristics predispose them to environmental persistence and to long-range transport. They intensively adsorb onto particles in air, soil and sediment, and accumulate in fat-containing tissues. The strong adsorption causes their mobility in soil and sediments to be negligible. Air is probably the most significant compartment for environmental distribution. PCBs have the ability to bioconcentrate and biomagnify under typical environmental conditions, thereby potentially achieving toxicologically relevant concentrations.

B. Human exposure and long-range transboundary air pollution

52. The general population is exposed to PCBs mainly via common food items, particularly fatty food of animal origin (e.g. meat, certain fish and dairy products). Recent estimated intake levels for adults in the Western world are about 50 ng/kg bw/day. Data available from industrialized countries have shown a reduction in population exposure levels in the last decades, but there are indications that this decline has levelled off. Prenatal and neonatal exposure is considered particularly important, as breastfed infants will exceed adult exposures by one to two orders of magnitude. As a result of LRTAP and dietary habits, human exposure to PCB in many Arctic regions is considerably higher than in industrialized areas. This observation as well as the

presence of PCBs in remote locations, point to the significance of LRTAP as an important element of the exposure pathways in those areas.

C. Health hazard characterization

53. Typical effects of PCBs exposure, including the critical effects of carcinogenicity, immunotoxicity and neurodevelopmental alterations, are caused both by the dioxin-like and the non-dioxin-like congeners. However, the underlying mechanisms involved are probably different. The developing foetus and the neonate are thought to represent a population potentially “at risk” due to increased susceptibility. For further information about the toxicology profile and the human health implications of dioxin-like PCB congeners relative to LRTAP, see chapter VI above. To date, toxic effects specific to non-dioxin-like PCB congeners have not been identified; however, endocrine disturbances and developmental toxicity are of major concern. Toxicity data on PCB hydroxy- and methyl-sulphonyl metabolites indicate that these compounds have their own toxicity profiles, which could include endocrine disturbances and respiratory tract toxicity.

54. The lowest observed effect level (LOEL) for subtle neurotoxic effects in infants following perinatal exposure is in the range of 0.014 - 0.9 µg PCB/kg bw/day. This exposure is in the same order of magnitude as the present PCB exposure of the general population in many countries.

55. It has not yet been possible, based on the available database, to reach a scientifically justified agreement on a TDI of either PCB mixtures or of any individual non-dioxin-like PCB congener.

56. IARC (1987) classified PCBs as probably carcinogenic to humans (class 2A).

D. Human health and long-range transboundary air pollution

57. As human PCB exposure, including both dioxin-like and non-dioxin-like congeners, may reach estimated LOAELs for neurodevelopmental effects in infants, the weight of evidence suggests an increased health risk due to current exposures. The lack of congener-specific exposure and toxicity data limits the possibilities for indicating which congeners are responsible for the effects.

58. Since the chemical and physical properties of PCBs make them susceptible to LRTAP, such pollution is expected to contribute significantly to the exposure and health risks, especially in remote areas.

VIII. POLYCYCLIC AROMATIC HYDROCARBONS

Introduction

59. Polycyclic aromatic hydrocarbons (PAHs) are a large group of compounds that consist of two or more fused aromatic rings made entirely from carbon and hydrogen. Most direct releases of PAHs to the environment from both natural and anthropogenic sources are to the atmosphere, with predominant emissions from human activities. The primary natural sources of airborne PAHs are

forest fires and volcanoes. The residential burning of wood is the largest source of atmospheric PAHs. Other important stationary anthropogenic sources include industrial power generation, incineration, the production of asphalt, coal tar, coke, petroleum catalytic cracking and primary aluminium production (Sodeberg technology in particular). Stationary sources account for about 80% of total annual PAHs emissions; the rest are from mobile sources. The most important mobile sources are vehicular exhausts from petrol and diesel-powered engines. PAHs are subject to emission controls specified in annex III to the 1998 Protocol on POPs.

A. Characteristics and a long-range transboundary air pollution potential

60. PAHs are present in the atmosphere in the gaseous phase or as adsorbed to particulates with relatively low degradation rates. Fine particles can remain airborne for a few days or longer and can be transported over long distances, therefore some PAHs have LRTAP potential. Air is probably the most significant compartment for environmental distribution.

61. The accumulation of PAHs in the soil is not significant. Bioaccumulation is limited and biomagnification has not been observed because most organisms have a high biotransformation potential for PAHs.

B. Human exposure and long-range transboundary air pollution

62. In the average American diet the intake of carcinogenic PAHs was estimated to be 1-5 µg/day, mostly from ingestion of unprocessed grains and cooked meats. This dietary intake estimate was 6-9 µg/day for those whose diets had a large meat content and resulted from the additional contribution of charcoal-cooked or smoked meats and fish. Exposure via inhalation of ambient air was estimated to be 0.16 µg/day (median) with a range of 0.02-3 µg/day, assuming an inhalation rate of 20 m³. Exposure via drinking water was estimated to be 0.006 µg/day (median), with a range of 0.0002-0.12 µg/day (2 litres of water daily).

63. Available data for Europe reported by EMEP in 2001 suggest that the mean annual air concentration of benzo-[a]-pyrene (BaP), one of the substances belonging to PAH group, which could be attributed to the long-range transport in 1998 ranged from 0.1 to 0.5 ng/m³. Available data also suggest that during the last 20 years both the emission and concentrations of PAHs in the air have decreased due to the modifications in heating systems and in the kind of heating fuel used, except for a few countries with increasing numbers of mobile PAH sources.

C. Health hazard characterization

64. The toxic effect of most concern from exposure to PAHs is cancer. IARC considers several purified PAHs and PAH derivatives to be probable (Group 2A) or possible (Group 2B) human carcinogens. Some mixtures containing PAHs are known human carcinogens (Group 1). Data obtained as a result of epidemiological studies in occupational settings suggest that there is an association between lung cancer and exposure to PAHs. The most important exposure route for lung cancer appears to be via inhalation. WHO (2000) considered BaP concentration in the air as an accurate index of the carcinogenic potential of the total fraction. A unit risk for BaP (lifetime exposure to a mixture represented by 1 ng/m³ BaP) is estimated to be 8.7×10^{-5} , roughly 90 cases

per million people exposed. Consequently, the excess lifetime risk of cancer corresponding to the mean BaP levels from LRTAP would amount to $8.7 \times 10^{-6} - 4.3 \times 10^{-5}$, approximately 9 to 50 cases per million people exposed.

65. Food constitutes the main route of PAH intake. Nevertheless, according to the FAO/WHO, the large difference between estimated human intake of BaP and the doses that induce tumours in animals suggests that any effects on human health as a result of oral intake are likely to be small or insignificant. The WHO guidelines for BaP in drinking water, corresponding to an excess lifetime risk for gastric cancer of 10^{-5} and 10^{-6} are 0.7 and 0.07 µg/l. The present concentrations of BaP in drinking water are below 0.002 µg/l.

D. Human health and long-range transboundary air pollution

66. The weight of evidence arising from epidemiological studies based on inhalation and occupational exposure to PAHs suggests an increased risk of harmful health effects, mainly lung cancer. The excess lifetime risk of lung cancer which can be attributed to LRTAP is low compared to the risk due to the exposure to PAHs from local sources.

IX. POLYCHLORINATED TERPHENYLS

Introduction

67. Polychlorinated terphenyls (PCTs) are chlorinated aromatic compounds that are structurally and chemically similar to polychlorinated biphenyls (PCBs). PCTs were produced in the United States (from 1929 to 1972), France, Italy, Germany and Japan, but their production was discontinued due to environmental concerns. Commercial PCT mixtures frequently contain PCBs. PCTs have been used since 1929 for a variety of uses including as a heat exchange fluid due to their chemical and thermal stability. Approximately 60 million tons of PCTs in total were produced between 1959 and the mid-1970s. Based on their chemical and physical similarity, PCTs may be expected to behave in a similar manner as PCBs in the environment. The production and use of PCTs will be reassessed under the 1998 Protocol on POPs by 31 December 2004 (see its annex I).

A. Characteristics and a long-range transboundary air pollution potential

68. Based on their persistence and their potential to bioaccumulate, PCTs satisfy the criteria of a POP with LRTAP potential. PCTs are characterized by their chemical and thermal stability; they have low volatility and water solubility. Generally, PCTs are assumed to be similar to PCBs with respect to environmental fate and transport processes and distribution. However, the long-range transport of PCTs has not been as extensively studied as that of PCBs. Atmospheric transport has been found to be a major pathway for PCT deposition into the Great Lakes.

69. The limited environmental data available indicate that PCTs are resistant to both biodegradation and photodegradation processes, which in combination with their lipophilicity and stability may indicate their ability to persist, bioconcentrate and biomagnify within the food chain. However, the database of literature for PCTs is both limited and dated.

B. Human exposure and long-range transboundary air pollution

70. PCTs have been detected in the environment; however, usually at levels lower than PCBs within the same samples. Potential exposure to PCTs by the general population would primarily occur via the consumption of meat, fish and dairy products. Young infants may be exposed to PCTs *in utero* prior to birth or by consumption of breast milk. Although there are limited data on the toxicokinetics of PCTs, they are absorbed and readily distributed to all body compartments, with the highest concentration in the liver.

C. Health hazard characterization

71. The toxicity of PCTs is considered to be very similar to that of PCBs, which suggests long-term toxicity might be critical, although chronic toxicity information is lacking. A general difficulty in toxicological studies of PCTs is the contamination of the PCT mixtures with PCBs. It is difficult to determine whether observed effects are caused by the PCTs or by the PCB contamination in the PCT mixtures. PCTs seem to be less acutely toxic than most PCBs. Effects in animals include dose-dependent increase in relative liver weights, reduced growth and proliferation of the endoplasmic reticulum. High doses of PCTs have been reported to stimulate hepatic microsomal enzymes in *in vivo* and *in vitro* test systems. Due to the limitations of the available data, the characterization of health hazards of PCTs is concluded to be limited.

72. The available data are inadequate to determine whether PCTs are causing the same health effects as PCBs. However, since the production and use of PCTs are banned in the UNECE region, the likelihood of obtaining adequate toxicity data to meet LRTAP criteria is low.

D. Human health and long-range transboundary air pollution

73. There is insufficient information to evaluate the health implications from long-term exposures to PCTs. Further studies are needed in order to be able to evaluate the health impact of PCTs and their potential link to LRTAP.

X. POLYBROMINATED DIPHENYL ETHERS

Introduction

74. Polybrominated diphenyl ethers (PBDEs) belong to a family of diverse chemicals employed in various industrial/consumer product applications as flame-retardants. Commercial production and use of PBDEs as additive flame retardants began in the 1960s with the majority of uses confined to the plastic (resins, polymers, substrates), textile, electronic, furniture and, to a lesser extent, paint industries. Annual worldwide production of all PBDEs in 1990 was estimated at 40 000 metric tons, with a continued market demand in 1999 of 42 000 metric tons for the Americas and Europe. Based on evidence of long-range atmospheric transport, environmental persistence and bioaccumulation in various species, including humans, PBDE congeners, mainly specific to the commercial penta-brominated diphenyl ether mixtures, appear to satisfy the criteria under which new chemicals can be considered for addition to the 1998 Protocol on POPs.

A. Characteristics and a long-range transboundary air pollution potential

75. The PBDE congeners which are typical for commercial penta-brominated diphenyl ether mixtures have certain physico-chemical and structural properties similar to polychlorinated biphenyls (hydrophobic, lipophilic, low vapour pressure, high log K_{ow}), which make them generally resistant to environmental degradation, susceptible to long-range transport processes and able to bioaccumulate. These PBDEs have been detected in both abiotic and biotic samples collected from remote locations with some evidence that concentrations have been increasing over the last two decades. From 1981 to 2000 the concentration of PBDEs in ringed seals collected from the Canadian Arctic increased by almost an order of magnitude (0.6 vs. 4.6 ng/g) suggesting efficient atmospheric transport. This is in contrast to PCBs levels, which over the same time period either stabilized or declined.

B. Human exposure and long-range transboundary air pollution

76. The vast majority of the population is exposed to PBDEs through the consumption of foods. Although market-basket survey data are limited, preliminary indications estimate daily intakes of approximately 1 ng/kg bw/day. Persons consuming large quantities of fish have been shown to accumulate high levels of PBDEs. As with other POPs, breastfed infants ingest quantities almost one or two orders of magnitude higher during the lactation period. Intake estimations have been attempted from locations near industrial uses of PBDEs and range up to 1 µg/kg bw/day. Based on the dietary practices of certain indigenous populations, it could be assumed that LRTAP is responsible for most of their exposure.

C. Health hazard characterization

77. Initial results from experimental animals indicate that certain PBDEs are efficiently absorbed from the gastrointestinal tract; they can induce various liver enzymes, cause organ changes and endocrine-related effects. While there is limited evidence to suggest PBDEs are reproductive toxicants, individual congeners found in the commercial penta-brominated diphenyl ether mixtures can induce neurodevelopmental alterations (in learning, memory, spontaneous behaviour) in neonatal mice. While uncertainties in the current exposure and toxicological database prevent an accurate risk characterization, there are indications that margin-of-safety estimates may be unacceptably low, especially considering the environmental persistence and bioaccumulative nature of PBDEs.

D. Human health and long-range transboundary air pollution

78. The developing foetus and breastfed infants are considered to be the main groups “at risk” from potential adverse effects due to exposure to PBDE congeners found in commercial penta-brominated diphenyl ether mixtures. The overall contribution of LRTAP to daily PBDE exposure depends on the region but would be substantial for more remote locations.

XI. POLYBROMINATED DIBENZO-*P*-DIOXINS AND POLYBROMINATED DIBENZOFURANS

Introduction

79. Polybrominated dibenzo-*p*-dioxins and polybrominated dibenzofurans (PBDD/Fs) consist of two groups of tricyclic aromatic compounds. PBDD/Fs exist as unintentional by-products in chemical processes but can also be formed during various combustion processes and photolytic degradation of polybrominated diphenylethers (PBDEs) and bromophenols. Among the possible 210 congeners, 17 have bromine atoms at least in the positions 2, 3, 7 and 8 of the parent molecule and these are very toxic compared to molecules lacking this configuration. All the 2,3,7,8-substituted PBDD/Fs show the same type of biological and toxic response as the corresponding PCDD/Fs. It is also known that mixed Cl-Br dioxins/furans can be formed. Thus 1550 mixed dioxins and 3050 mixed furans are theoretically possible. Due to the paucity of analytical reference standards, a very limited number of these congeners have been studied and analysed so far. These substances are not included in the 1998 Protocol on POPs.

A. Characteristics and a long-range transboundary air pollution potential

80. There are very few data available on environmental transport and distribution. PBDD/Fs are more readily degraded photochemically than PCDD/Fs. Generally, the physico-chemical properties of PBDD/Fs suggest similarities to PCDD/Fs. Therefore, they would be expected to accumulate in carbon- and/or fat-rich compartments.

B. Human exposure and long-range transboundary air pollution

81. There are no quantitative data on levels of the current substances in food or wildlife. Lower brominated congeners (mono - tetra) have been found close to motorways on pine needles and grass.

C. Health hazard characterization

82. The kinetics and metabolism of PBDD/Fs have been investigated in a limited number of studies. PBDD/Fs congeners show obvious similarities with their chlorinated analogues concerning metabolism, elimination and biological half-lives. Also in the limited number of effect studies performed, PBDD/Fs congeners show similarities with their PCDD/Fs analogues. They are believed to share a common mechanism of action with PCDD/Fs and other related hydrocarbons. Binding to the Ah receptor has been confirmed for several PBDD/Fs and the mixed Cl-Br compounds. Also the receptor-binding capacity has been reported to be similar to that of the chlorinated analogues. There are no data on effects in humans.

D. Human health and long-range transboundary air pollution

83. Based on the physical and chemical similarities with PCDD/Fs, it is possible that PBDD/Fs could resist degradation, bioaccumulate and be transported through air across international

boundaries. However, there is still a lack of data to confirm the presence of PBDD/Fs in biota. The human health implications relative to LRTAP cannot be judged on present data.

XII. SHORT-CHAIN CHLORINATED PARAFFINS

Introduction

84. Chlorinated paraffins (CPs) are straight-chain alkanes with varying degrees of chlorination. They have been produced since the 1930s to an extent of approximately 300 kilotons per year estimated for the Western world. CPs have been used as high-temperature and pressure lubricants as well as secondary plasticizers and flame-retardants in plastics and paints.

85. CPs are divided into three main categories, short- (C10-C13), medium- (C14-C17) and long-chain (C18-C30), and further by their degree of chlorination, low (<50%) and high (>50%). Because of their relatively high assimilation and accumulation potential, the short-chain chlorinated paraffins (SCCPs) have been the most widely studied.

86. The complexity of SCCP mixtures makes it difficult to provide an analytical method for their precise and specific quantitative determination. Technical SCCP mixture consists of several thousand components and, due to the large number of isomers, complete chromatographic separation seems impossible at this point. This analytical challenge has resulted in different approaches to SCCP analysis; however, the number of relevant monitoring results is still limited.

87. These substances are not included in the 1998 Protocol on POPs.

A. Characteristics and a long-range transboundary air pollution potential

88. SCCPs are complex mixtures, which vary in chain lengths and in the degree of chlorination. The vapour pressure values, Henry's law constants and atmospheric half-life values are in the same range as those of other persistent organic pollutants and imply a significant potential for long-range atmospheric transport. SCCPs have been detected in Arctic air, biota and lake sediments, and in the water column around the Bermuda Islands despite the absence of significant sources of SCCPs in these regions, which suggests that these residues are due to long-range atmospheric transport.

SCCPs clearly fulfil the criteria for bioconcentration and there is some evidence of biomagnification.

B. Human exposure and long-range transboundary air pollution

89. The main environmental source of human exposure is food and, to a lesser extent, drinking water. The risk of human exposure related to long-range transboundary atmospheric transport is difficult to quantify but should not be neglected. The lack of monitoring data hampers reliable exposure estimation. Levels in food in the range of 30 to several thousands µg/kg have been measured. The European Union's risk assessment report considers a human uptake value of about 20 µg/kg bw/day as a reasonable worst-case value.

C. Health hazard characterization

90. Compared to PCBs or chlorinated pesticides, SCCPs appear to exhibit fewer toxic effects. SCCPs show lower reproductive and embryo toxicity in mammals and birds. The main target organs for repeated doses of SCCPs seem to be the liver, kidney and thyroid. SCCPs show neoplastic effects in the liver of mice and rats, however the relevance of this evidence for humans is uncertain.

91. In 1996, WHO recommended that daily doses of SCCPs for the general population should not exceed 11 µg/kg bw for neoplastic effects.

D. Human health and long-range transboundary air pollution

92. Long-range transboundary atmospheric transport is an important aspect of the global distribution of SCCPs and is responsible for their occurrence in remote areas.

93. The EU risk assessment report concludes that there is no significant risk to man exposed to SCCPs via the environment. However, the EU worst-case human uptake estimate is greater than the guideline value established by WHO.

Annex I

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Annex II

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